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VASCULAR ANAESTHESIA

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ANAESTHESIA

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Vascular Surgery

Oxford Specialist Handbooks in Anaesthesia

Vascular Anaesthesia

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Foreword

Anaesthesia for vascular surgery is possibly one of the most challenging fields of our clinical practice. By virtue of their primary surgical pathology and its associated risk factors, patients have a very high incidence of co-morbidities, particularly hypertension, diabetes, cardiac, cerebrovascular, renal and respiratory disease. Advances in anaesthesia must also keep abreast of surgical and graft developments which have led to a dramatic increase in the use of endovascular stents such that they now constitute the majority of surgery. In turn, however, this has resulted in the presentation of patients previously regarded as medically unsuitable for surgery since the perturbations of surgical trauma are markedly reduced. At the same time, open repair of aneurysmal and stenotic disease is still an option for some and may be unavoidable for anatomical reasons in others. Patients with complex pathology may require hybrid procedures which entail an open surgical component in addition to graft placement. Further still, correction of graft complications and leaks may necessitate more complicated open surgery. Hence the traditional skills for handling clamping and unclamping of the aorta and major haemodynamic disturbance must be preserved. Perioperative organ protective is pivotal to optimising patient outcome. There is also a need to protect the kidney from ischaemia and contrast induced nephropathy, the spinal cord during open or endovascular aortic surgery, the brain in carotid surgery and the heart in all surgery. An understanding of appropriate monitoring of these organs, techniques for protection both pharmacologic and mechanical, appropriate use of anaesthetic and other drugs and amelioration of ischaemia-reperfusion injury is essential. Knowledge of regional anaesthesia, peripheral nerve blocks and sedation techniques is also important for anaesthesia and analgesia in many of these patients. A successful outcome is only as "strong as the weakest link" and both preoperative preparation and postoperative care are integral facets. In negotiating all these aspects of patient care, vascular anaesthetists can truly be considered model perioperative physicians.

Recent developments in both surgery and perioperative care make this an apposite time to produce this handbook and the editors, all very experienced and authoritative in this field, have collected an eclectic collection of clinicians to present a clear and easy to read overview of this challenging specialty. True to its status as a handbook, chapters are succinct, easy to read and search with appropriate use of bullet points and avoidance of verbosity. An excellent companion for the busy clinician both in training and those seasoned in practice.

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Preface

The management of the patient with vascular disease is evolving rapidly. Population screening for vascular disease has been introduced and its effects on practice are being evaluated. The management of associated medical conditions is also changing. New guidelines have been published for the management of acute coronary syndromes, stable coronary heart disease, diabetes, implantable pacemakers, and defibrillators. Decisions regarding surgical intervention and anaesthetic management remain complicated due to the co-existence of vascular disease at other sites or other co-morbid conditions, but have been aided by improved approaches to preoperative risk assessment, perioperative monitoring and postoperative care. Radiological expertise and procedures are increasingly used to treat conditions previously treated by open surgery, such as aortic aneurysms, occlusive lower limb arterial disease, or carotid disease. Priorities have evolved so that carotid endarterectomy is now performed as an urgent procedure after TIA or minor stroke. In addition, the pace of change over the last two decades has made it difficult for those involved in the care of the vascular surgical patient to remain updated with recent progress in perioperative management. The literature describes an increasing array of techniques for preoperative investigation and monitoring during anaesthesia, with disparate evidence and recommendations. When faced in clinical practice with a patient with vascular disease, the anaesthetist needs to know what to do and when to do it, as well as what not to do and why.

In this new addition to the Oxford Specialist Handbooks series we have tried to produce a concise volume that will enable the vascular practitioner (in its broadest sense) to adopt a practical, current, evidence-based approach to all aspects of perioperative care for the patient with vascular disease, particularly those undergoing interventional radiological, diagnostic, and surgical procedures. Although primarily aimed at anaesthetists, we hope the book will be relevant to vascular nurses, theatre practitioners, trainees in Intensive Care Medicine and possibly even vascular surgeons. Our approach has been to combine essential background knowledge with useful, clinically relevant sections on management, so the book can be used to enhance awareness of potential problems, as an aid to revision, but also as a practical 'How to do it' guide for patient management.

The book is divided into sections and starts with what we consider essential details on the epidemiology of vascular disease, followed by relevant anatomy and pathophysiology. Good preoperative evaluation is vital to a successful outcome, but the vascular anaesthetist must also be aware of the processes involved in complex surgical decision-making; these are detailed together. Current approaches to risk assessment and risk reduction are emphasized, including advice on how to set up and run a preoperative assessment clinic. In the sections on medical management of common co-existing diseases, monitoring, practical procedures and common regional anaesthetic techniques, we have tried to summarize what the practitioner needs to know for everyday practice, based on the most recent data.

Radiologists are taking an ever greater role in the care of the vascular patient; this can be a difficult and complex area of practice and we have deliberately included a section dedicated to radiological management. The final three sections are intended to be a convenient guide to the management of different vascular procedures and complications during and after surgery. They are intended to be used as an 'aide-memoire' for perioperative care and include guidance on the management of common postoperative problems.

The contributors are all experienced clinicians actively caring for patients with vascular diseases. They are predominantly consultant anaesthetists with a special interest in vascular anaesthesia, but include vascular surgeons, radiologists, cardiologists and other physicians. We hope this book will fulfil its aims and be useful, relevant and helpful in the day-to-day management of the vascular surgical patient.

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Contents

Symbols and Abbreviations

1°	primary
2°	secondary
<	less than
>	more than
+ve	positive
–ve	negative
AAA	abdominal aortic aneurysms
AbCS	abdominal compartment syndrome
ABG	arterial blood gases
ABPI	ankle brachial pressure index
ACC	American College of Cardiology
ACE	angiotensin-converting enzyme
AcLI	acute limb ischaemia
ACS	acute coronary syndrome
ACST	Asymptomatic Carotid Surgery Trial
ACT	activated clotting times
ADH	anti-diuretic hormone
ADP	adenosine di-phosphate
AF	atrial fibrillation
AHA	American Heart Association
AKA	above knee amputations
AKI	acute kidney injury
ALI	acute lung injury
ANH	acute normovolaemic haemodilution
ANP	atrial natriuretic peptide
AP	anteroposterior
APC	abnormalities of protein C
APL	adjustable pressure limiting
APTT	activated partial thromboplastin time
APTTR	activated partial thromboplastin time ratio
ARB	angiotensin II receptor blocker
ARDS	adult respiratory distress syndrome
ARR	absolute risk reduction
AT	angiotensin

ATD	adult therapeutic dose
ATh	anaerobic threshold
AU	aggregation units
AUC	area under the concentration curve
AUI	aorto-uniiliac
AV	atrioventricular
AVM	arteriovenous malformations
AXC	aortic cross-clamp
BAE	bronchial artery embolization
BAEP	brainstem auditory-evoked responses
bd	twice daily
BioMS	biomedical scientist
BIS	bispectral index
Biv-CRT	biventricular cardiac resynchronization therapy
BKA	below knee amputations
BMI	body mass index
BMS	bare metal stents
BNP	brain natriuretic peptide
BP	blood pressure
BPEG	British Pacing and Electrophysiology Group
CABG	coronary artery bypass grafting
CAD	coronary artery disease
CAS	carotid angioplasty with stenting
CBF	cerebral blood flow
CCB	calcium channel blocker
CCU	critical care unit
CEA	carotid endarterectomy
CePB	cervical plexus block
CFAM	cerebral function analysing monitor
CGRP	calcitonin gene-related peptide
CH	cerebral hyperperfusion
CHS	cerebral hyperperfusion syndrome
CIN	contrast-induced nephropathy
CKD	chronic kidney disease
CLI	critical limb ischaemia
CMI	chronic mesenteric ischaemia
CMV	cytomegalovirus
CNS	central nervous system

CO	cardiac output
CoNS	coagulase –ve staphylococci
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airways pressure
CPB	cardiopulmonary bypass
CPD	continuous peritoneal dialysis
CPET	cardiopulmonary exercise testing
CPI	Customized Probability Index
CPP	cerebral perfusion pressure
CPX	cardiopulmonary exercise testing
CSA	cross-sectional area
CSE	combined-spinal epidural
CSF	cerebrospinal fluid
CSpA	continuous spinal anaesthesia
CsT	closure time
CT	computed tomogram
CTA	CT angiography
cTN	cardiac troponin
CVA	central venous access
CVC	central venous catheter
CVD	cerebrovascular disease
CVI	chronic venous insufficiency
CVP	central venous pressure
CVR	cerebral vascular resistance
CVVH	continuous veno-venous haemofiltration
CXR	chest X-ray
DBP	diastolic blood pressure
DCT	distal convoluted tubule
DES	drug-eluting stents
DHCA	deep hypothermic cardiac arrest
DLT	double lumen tube
DM	diabetes mellitus
DNIC	diffuse noxious inhibitory control
DPG	diphosphoglycerate
DRG	dorsal root ganglia
DSE	dobutamine stress echocardiography
DVT	deep vein thrombosis
ECG	electrocardiogram
EEG	electroencephalogram

EET	eicosatrienoic acids
EF	ejection fraction
eGFR	estimated glomerular filtration rate
EMG	electromyogram
eNOS	endothelial NO synthase
EPO	erythropoietin
ESRD	end-stage renal disease
ESRF	end stage renal failure
ETS	endoscopic thoracic sympathectomy
EVAR	endovascular aneurysm repair
EVLA	endovenous laser ablation
EVLT	endovenous laser treatment
EWS	Early Warning Score
FAST	focused assessment with sonography in trauma
FBC	full blood count
FDP	fibrin degradation products
FEV	forced expiratory volume
FFP	fresh frozen plasma
FMR	functional mitral regurgitation
FPG	fasting plasma glucose
FRC	functional residual capacity
FTc	flow time corrected for heart rate
G&S	group & save
GA	general anaesthesia/anaesthetic
GCS	Glasgow Coma Score
GDT	goal-directed therapy
GFR	glomerular filtration rate
GI	gastrointestinal
GIB	gastrointestinal bleeding
GP	general practitioner
GRACE	Global Registry of Acute Coronary Events
GTN	glyceryl trinitrate
Hct	haematocrit
Hcy	homocysteine
HDU	high dependency unit
HF	heart failure
HHcy	hyperhomocysteinaemia
HIT	heparin-induced thrombocytopenia
HITT	heparin-induced thrombocytopenia and thrombosis

HME	heat and moisture exchanger
hsCRP	high sensitivity C-reactive protein
IABP	invasive arterial blood pressure
IAH	intra- abdominal hypertension
IAP	intra-abdominal pressure
IBP	intra-arterial blood pressure
IC	intermittent claudication
ICD	implantable cardiac defibrillators
ICP	intracranial pressure
ICS	intracoronary stents
ICU	intensive care unit
IFG	impaired fasting glycaemia
IGT	impaired glucose tolerance
IHD	ischaemic heart disease
IIA	internal iliac artery
IJV	internal jugular vein
iNOS	inducible nitric oxide synthase
INR	international normalized ratio
IOCS	intraoperative cell salvage
IPPV	intermittent positive pressure ventilation
IR	interventional radiology
ISWT	incremental shuttle walking test
ITU	intensive therapy unit
IV	intravenous
IVC	inferior vena cava
JVP	jugular venous pressure
KATP	adenosine triphosphate (ATP)-sensitive potassium (K+) channel
KDIGO	Kidney Disease: Improving Global Outcomes
LA	local anaesthesia/anaesthetic
LBBB	left bundle branch block
LCA	left coronary artery
LDL	low density lipoprotein
LFT	liver function test
LiDCO	lithium indicator dilution
LIJ	left internal jugular
LLA	lower limb amputation
LLL	left lower lobe
LMA	laryngeal mask airway

LMWH	low molecular weight heparin
LUL	left upper lobe
LV	left ventricle/ventricular
LVEDP	left ventricular end-diastolic pressure
LVEF	left ventricular ejection fraction
LVF	left ventricular failure
MA	mean acceleration
MAC	minimum alveolar concentration
MAP	mean arterial pressure
MASS	Multicentre Aneurysm Screening Study
MCA	middle cerebral artery
MCF	maximum clot firmness
MDRD	modification of diet in renal disease
MDT	multidisciplinary team
MEP	motor-evoked potential
MET	metabolic equivalent
MEWS	Modified Early Warning Score
MI	myocardial infarction
MR	modified release
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
MRSA	meticillin-resistant *Staphylococcus aureus*
MTPP	mitochondrial transition permeability pore
NANC	non-adrenergic non-cholinergic
NASSE	North American Society of Pacing and Electrophysiology
NGAL	Neutrophil gelatinase-associated lipocalin
NGT	nasogastric tube
NHANES	National Health and Nutrition Examination Survey
NIBP	non-invasive blood pressure
NICE	National Institute of Health and Care Excellence
NIRS	near infrared spectroscopy
NMB	neuromuscular blockers
NMDA	N-methyl-d-aspartate
nNOS	neural nitric oxide synthase
NNT	numbers needed to treat
NO	nitric oxide
NPV	negative predictive value
NSAID	non-steroidal anti-inflammatory drug
NSTEMI	non-ST segment elevation myocardial infarction

NT-proBNP	N-terminal pro-BNP
NVD	national vascular database
NYHA	New York Heart Association
ODM	oesophageal Doppler monitoring
ODP	operating department practitioner
OGTT	oral glucose tolerance test
OIH	opioid-induced hyperalgesia
OLV	one-lung ventilation
PA	pulmonary artery
PAC	pulmonary artery catheter
PACU	post-anaesthesia care unit
PAD	peripheral arterial disease
PAOP	pulmonary artery occlusion pressure
$PbrO_2$	brain tissue O_2 partial pressure
PCA	patient-controlled analgesia
PCC	prothrombin complex concentrate
PCEA	patient-controlled epidural analgesia
PCI	percutaneous coronary intervention
PCR	polymerase chain reaction
PCT	proximal convoluted tubule
PCV	packed cell volume
PCWP	pulmonary capillary wedge pressure
PE	pulmonary embolism
PEEP	positive end expiratory pressure
PES	post-embolization syndrome
PICC	peripherally-inserted central catheters
PIP	peak inspiratory pressure
PKCε	proteinkinase C espsilon
PLP	phantom limb pain
po	by mouth
POC	point of care
POCD	post-operative cognitive dysfunction
POISE	perioperative ischaemia evaluation
PONV	post-operative nausea and vomiting
PPC	post-operative pulmonary complication
PPV	positive predictive value
PreAD	preoperative autologous donation
PSIS	posterior superior iliac spine
PT	prothrombin time

PTFE	polytetrafluoroethylene
PTH	parathyroid hormone
PTT	partial thromboplastin time
PuPV	pulse pressure variation
PV	peak velocity
PVC	premature ventricular contractions
PVD	peripheral vascular disease
PVGI	prosthetic vascular graft infection
PVR	peripheral vascular reconstruction
RA	regional anaesthesia/anaesthetic
RAAA	ruptured open abdominal aortic aneurysm repair
RAAS	renin-angiotensin-aldosterone system
RBC	red blood cells
RBF	renal blood flow
RCA	right coronary artery
RCRI	revised cardiac risk index
RCT	randomized controlled trial
RFA	radiofrequency ablation
RIJ	right internal jugular
RLL	right lower lobe
RMB	right main bronchus
RML	right middle lobe
RMP	resting membrane potential
ROC	receiver operating characteristic
RPF	renal plasma flow
RR	respiratory rate
RRR	relative risk reduction
RRT	renal replacement therapy
r-TEG	rapid-TEG
rtPA	recombinant tissue plasminogen activator
RUL	right upper lobe
RV	right ventricle
SA	sino-atrial
SACU	surgical acute care unit
SAG-M	saline, adenine, glucose, and mannitol
SAM	S-adenosylmethionine
SBP	systolic blood pressure
sc	subcutaneous
SCM	sternocleidomastoid

ScO_2	calculated O_2 saturation
SCPP	spinal cord perfusion pressure
SCV	subclavian vein
SFA	superficial femoral artery
SHO	senior house officer
SIRS	systemic inflammatory response
SPECT	single photon emission computed tomographic
SpO_2	oxygen saturation
SpR	specialist registrar
SPV	systolic pressure variation
SR	sarcoplasmic reticulum
SSEP	somatosensory-evoked potentials
STEMI	ST elevation myocardial infarction
SV	stroke volume
SVC	superior vena cava
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
SVV	stroke volume variation
SWMA	systolic wall motion abnormalities
TAA	thoracic aortic aneurysm
TAAA	thoraco-abdominal aortic aneurysm
TAI	thoracic aorta injury
TAP	transversus abdominis plane
TASC	TransAtlantic Inter-society Consensus
TAVI	transcatheter aortic valve implantation
TCD	transcranial Doppler
TCI	target-controlled infusion
TEA	thoracic epidural analgesia
TEDS	thrombo-embolic stockings
TEVAR	thoracic endovascular aneurysm repair
TIA	transient ischaemic attacks
TIPPS	trans-hepatic porto-systemic shunt
TIPS	trans-jugular porto-systemic shunts
TIVA	total intravenous anaesthesia
TIVAD	totally in-dwelling venous access devices
TOE	transoesophageal echocardiography
TOS	thoracic outlet syndrome
TP	threshold potential
TPN	total parenteral nutrition

TRP	transient receptor potential
TT	thrombin time
TTE	transthoracic echocardiography
TV	tidal volume
U&E	urea & electrolytes
UFH	unfractionated heparin
UGRA	ultrasound-guided regional anaesthesia
UKSAT	UK Small Aneurysm Trial
UO	urine output
VASGBI	Vascular Anaesthesia Society of Great Britain and Ireland
vCJD	variant Creutzfeldt–Jacob disease
VF	ventricular fibrillation
VIB	vertical infraclavicular block
VRIII	variable rate intravenous insulin infusion
VT	ventricular tachycardia
VTI	velocity time index
VV	varicose veins
vWF	von Willebrand factor
WCC	white cell count
WMSI	wall motion score index
XM	cross-match

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Chapter 1

Epidemiology of vascular disease

Incidence, prevalence, and risk factors of vascular disease

Occlusive disease

Occlusive disease of the lower limbs and carotid artery disease are aspects of the atherosclerotic disease that is widespread in the populations of developed countries. The true incidence of atherosclerosis itself is difficult to determine because it is an asymptomatic disease in the majority of patients. Post-mortem studies in humans who died from non-cardiac disease revealed early morphological signs of aortic and coronary atherosclerosis that ranged widely from 50 to 100% of young people aged below 35yrs.

Risk factors for occlusive atherosclerotic disease include:

- Smoking.
- Increasing age.
- Hypertension.
- Diabetes.
- Previous cardiovascular disease.
- Hypercholesterolaemia.
- Hypertriglyceridaemia.
- Physical inactivity.

Diabetes is a particularly important risk factor for atherosclerosis. Post-mortem studies reveal that nearly 75% of diabetic individuals who did not have clinical coronary artery disease (CAD) had high grade coronary atherosclerosis.

It should be noted that atherosclerosis is a global disease of the circulation.

- In patients (without previously recognized extracranial cerebrovascular disease) who undergo elective peripheral vascular reconstruction, approximately 13% will have incidental asymptomatic carotid stenosis of more than 50%.
- Severe, potentially surgically-correctable CAD will be present in 24–29% of patients who undergo elective peripheral vascular reconstruction.
- Patients with asymptomatic peripheral arterial disease (PAD) have a much higher risk of systemic cardiovascular events than the general population.
- The Edinburgh Artery Study revealed that the incidence and mortality from acute myocardial infarction was increased in the presence of PAD, and was the same whether PAD patients were symptomatic or not.

This highlights the high incidence of significant atherosclerotic disease in territories other than that for which surgery is required in vascular surgery patients. These synchronous atherosclerotic diseases may need prior optimization to achieve best possible perioperative outcomes.

Peripheral arterial disease

PAD (atherosclerotic disease affecting the lower legs) is very common.

- It is asymptomatic in the majority of patients.
- If symptomatic, the first presentation is usually with intermittent claudication (pain in the leg on walking).
- In claudicants, over a 5-yr period:
  - 50% will remain relatively stable.
  - 25% will get worse, 5% will undergo revascularization (angioplasty or surgery), and 1% will undergo amputation.
- Therefore, a minority will progress to critical limb ischaemia (CLI) when the blood supply to the legs becomes further reduced with progression of atherosclerosis or failure of collateralization of new blood vessels.
- A European Consensus document published in 1990 defined CLI as rest pain for >2 weeks, or ulceration/gangrene, and an ankle pressure of <50mmHg, or toe pressure of <30mmHg.

Survey-based assessment of PAD epidemiology

Accurate assessment of the epidemiology of PAD is not straightforward and is dependent on the sample group. Previous epidemiological studies, which have focused on referrals to hospitals or workplace settings, are not truly representative of the wider population.

- The Edinburgh Artery Study, which was a random cross-sectional survey conducted on an age-stratified sample of men and women aged 55–74yrs selected from age–sex registers in ten general practices in the city, is one of the largest and most reliable source of information on the prevalence of PAD. In this study the prevalence of intermittent claudication was 4.5%.
- The Scottish Heart Study reported a prevalence of intermittent claudication of 1.1% in subjects aged 40–59yrs.
- In the Limburg Study, the reported prevalence in their subjects aged 40–79yrs was between 1.4 and 6.1%.
- In these population-based questionnaire studies, the prevalence of intermittent claudication increased with age.

Ankle brachial pressure index or ultrasound based assessment of PAD epidemiology

The technique used to establish the presence or absence of PAD is also an important consideration when assessing the epidemiology of PAD.

- The prevalence of asymptomatic PAD as defined by ankle brachial pressure index (ABPI) measurements less than 0.9 in the middle aged to elderly population is about 7–15%.
- The PERART study of a Spanish primary care population that defined peripheral vascular disease as ABPI <0.9 reported a PAD prevalence of 7.6% (6.7–8.4%). The prevalence in males was 10.2% that in females 5.3%. In this study, regular walking or a BMI >25kg/m^2 were protective.
- The National Health and Nutrition Examination Survey (NHANES, 1999–2000) from the USA analysed data from 2174 participants and reported that the prevalence of PAD (defined as ABPI <0.9 in either leg) was 4.3% in adults aged over 40yrs old. In those over 70yrs old, the prevalence was 14.5%.

- However, if direct assessment of the femoral artery using ultrasound was used, as in the British Regional Heart Study, 64% of subjects aged 56–77yrs had significant femoral atherosclerosis. Of these, only 10% of these were symptomatic.

Approximately 70% of patients with PAD diagnosed on ABPI are asymptomatic. The anaesthetist must remember that the patients who present for surgical or radiological intervention are those with most severe disease—they are a subset of a larger population whose condition could be managed medically or which goes undetected. Many patients are managed in the vascular clinic or in general practice with measures including blood pressure (BP) control, glycaemic control, smoking cessation, and lipid lowering therapy (in particular the use of statins).

The Vascular Society of Great Britain and Ireland carried out a prospective national survey of patients with critical lower limb ischaemia to estimate the prevalence of critical lower limb ischaemia. The report revealed that the extrapolated incidence of critical lower limb ischaemia in Great Britain and Ireland was a total of 21,450 limbs in 20,000 patients in the population as a whole, equating to a prevalence of 1 in 2500 of the population annually. Of these 25% will undergo major amputations

Amputation

Amputation of lower limbs in patients with severe lower limb ischaemia is indicated to achieve relief of pain or removal of gangrenous or necrotic or severely ischaemic tissue. It can restore function and quality of life.

- About 15,000 lower limb amputations are carried out in the UK every year, of which 48% are for amputation of toes and 7% for foot amputations.
- Vascular causes account for >80% of all amputations in the UK.
- Diabetes is involved for 20–30% of these cases.
- Insulin-dependent diabetics are at a higher risk (6-fold) than non-insulin-dependent diabetics.
- In patients referred to prosthetic centres in the UK, 52% were transtibial (i.e. below knee) amputations and 38% were transfemoral (i.e. above knee) amputations.
- 30% of vascular amputees will undergo amputation of the other leg within 2yrs.
- The mortality of amputee patients is about 50% in 5yrs.
- The survival of amputees is lower in diabetics than non-diabetics.
- The incidence of amputation fell by about 27% from 1980 to 1990 due to aggressive reconstruction policy and increased use of infra-inguinal bypass operations.

Carotid artery disease

Stroke is a loss of cerebral function from a vascular cause lasting more than 24h. About 80% of strokes are ischaemic and about 80% of ischaemic strokes originate from the carotid territory.

- Annually about 120,000 people in the United Kingdom develop a stroke.
- 20–30% of these patients will die within a month.

- The incidence of a first ever stroke is 2.4/1000, increasing with age.
- Stroke is responsible for 12% of UK deaths, and is the third commonest cause of mortality after heart disease and cancer.
- It is also the single largest cause of severe disability in adults. There are nearly 1 million people living with the consequences of stroke, and a third of these patients have long-term disabilities. As a result, the economic costs of stroke are enormous (~£7 billion/yr).

Transient ischaemic attacks (TIA) cause symptoms and signs lasting less than 24h

- The annual incidence of TIA is 0.5/1000.
- Each year approximately 21,000 patients in England and Wales (about half of whom are greater than 70yrs old) consult a doctor for the first time with a TIA.
- The incidence of TIAs increases sharply with age, from 0.9/1000 in those aged 55–64yrs to 2.6/1000 for those aged 75–84yrs.

Carotid endarterectomy

In the UK, around 4500 carotid endarterectomies (CEA) are performed each year to reduce the risk of stroke.

- CEA is indicated in symptomatic >50% carotid stenosis.
- CEA does not confer any benefit if the carotid artery is occluded or nearly occluded (string sign).
- For asymptomatic stenosis, the Asymptomatic Carotid Surgery Trial (ACST) reported that CEA for patients younger than 75yrs of age with >60% stenosis reduced 10-yr stroke risks.
- Numbers needed to treat (NNT) to prevent any stroke at 5yrs are:
  - Six for symptomatic 70–99% stenosis.
  - Thirteen for symptomatic 50–69% stenosis.
  - Nineteen for asymptomatic >60% stenosis.
- Asymptomatic carotid disease is a common condition that is usually detected from incidental finding of a carotid bruit or as an investigation of the contralateral side.
- About 4% of people over 45yrs old will have a carotid bruit and this increases to 12% in people over 60yrs old.
- However, the presence or absence of a bruit or the quality of bruit correlates poorly with the degree of carotid stenosis.
- In the population over 65yrs, the prevalence of 50–99% stenosis is about 5–10%. This prevalence is increased in the presence of PAD (12%) and hypertension (25%).

Chronic mesenteric ischaemia

- Chronic mesenteric ischaemia (CMI) is much more uncommon than acute mesenteric ischaemia.
- CMI constitutes only 5% of all mesenteric ischaemia.
- The incidence of atherosclerotic lesions affecting the mesenteric arteries in people more than 65yrs old is approximately 18%

Aneurysmal disease

Aneurysm is defined as an abnormal focal dilatation of a vessel, of greater than 50% in diameter. Aneurysms that are considered within the remit of vascular surgery are either aortic aneurysms or peripheral aneurysms (iliac, popliteal, and femoral aneurysms). Aneurysms can be fusiform (cylindrical dilatation of the whole vessel) or saccular (focal bulge arising from the side of the vessel).

Abdominal aortic aneurysm

The normal diameter of the abdominal aorta is up to 2cm and dilatation above 3cm is, therefore, generally considered to be aneurysmal.

Classification and risk factors

- 90% of abdominal aortic aneurysms (AAA) are infrarenal with the remaining 10% being juxtarenal or suprarenal.
- Inflammatory AAAs, defined as thickened aneurysm wall with marked peri-aneurysmal or retroperitoneal fibrosis and dense adhesions to adjacent organs, represent 3–10% of all AAAs.
- AAAs are four times more common in men than women.
- The mean age for presentation is 65–70yrs.
- Risk factors for AAAs include smoking, male sex, increasing age, hypertension, and presence of chronic obstructive pulmonary disease (independent of smoking).
- AAAs are less common in diabetics. The reason for this is unclear.

Incidence and mortality

- Historically, the prevalence in men over the age of 60 is approximately 2–6% and its incidence was considered to be rising in the developed world.
- However, recent evidence suggests that the AAA epidemic has stopped, and its incidence and mortality is on the decline. This is attributed to the decline in smoking and perhaps better control of BP and uptake in statin therapy.
- Ruptured AAA accounts for 1.4% of deaths in men and 0.5% of deaths in women over the age of 65yrs in England and Wales.
- In men, a peak in mortality rate due to ruptured AAA occurs between 65 and 85yrs of age
- In England, AAA accounts for over 11,000 hospital admissions and 5000 deaths a year.
- In USA, abdominal aortic aneurysm (AAA) ranks as the 13th leading cause of death and is responsible for 0.8% of all deaths.
- Currently, there is no pharmacological treatment available to effectively inhibit aneurysm expansion or rupture; and the only treatment option remains surgical repair by conventional or endovascular means.
- A mortality rate of less than 5% is expected for a high quality service undertaking elective surgical repair (open or endovascular) of the asymptomatic lesion.
- In contrast, the reported in-hospital mortality rate of ruptured AAA varies between 47 and 83%. When including the patients with rupture who do not reach hospital alive, the overall mortality rate of rupture is much higher (between 78 and 94%).

Natural history (expansion and rupture)

The natural history of AAAs is continued expansion and eventual rupture.

- The annual expansion is approximately about 10% of the sac diameter but there is a wide variation in growth rates.
- There is equivocal evidence for the role of statins in retarding AAA expansion.
- Current clinical trials are investigating the effects of angiotensin-converting enzyme (ACE) inhibitors and mast cell inhibitors on AAA growth.
- The classic triad of rupture is abdominal pain (frequently radiating to the back), shock and pulsatile mass. Any two of these three symptoms should alert the attending physician to the possibility of a ruptured AAA.
- Free intra-peritoneal rupture of an AAA is rapidly fatal.
- The risk of AAA rupture increases exponentially with the maximum diameter of the aneurysm (Table 1.1).
- The UK Small Aneurysm Trial (UK SAT) determined that it is appropriate to observe small AAAs until they reach a size of 5.5cm. At this threshold diameter, surgically intervention is recommended because the risk of death from rupture outweighs the risk of death from surgery.
- Other risk factors for aneurysm rupture include higher than expected rate of aneurysm expansion, female sex, hypertension, smoking, and chronic obstructive pulmonary disease.

AAA screening

Most AAAs are asymptomatic until rupture, when the mortality exceeds 80%. These asymptomatic AAAs are detected either during routine physical examination or during imaging investigation for other non-related conditions.

- Population screening for AAAs using ultrasound scan for 65-yr-old men is expected to be fully implemented in England by 2013 to detect asymptomatic AAAs.

Table 1.1 Annual rupture rates of abdominal aortic aneurysms according to size (based on pooled available data)

Initial aneurysm diameter (cm)	Annual risk of rupture
3.0	0.2–0.4%
4.0	0.8–1.1%
4.0–5.5	0.6–1.0%
5.5–5.9	5.0–9.4%
6.0–6.9	10.2%
>7.0	30.5–32.5%

- The Multicentre Aneurysm Screening Study (MASS) trial showed that aneurysm-related mortality was significantly reduced in screened male population between 65 and 74yrs old, with about 53% reduction in those who attended for screening.
- Over 4yrs, the MASS trial showed that the incremental cost effective ratio for screening was £28,400 per life-year gained (£36,000 per quality-adjusted life year. This falls to approximately £8000 per life year gained at 8yrs.
- This compares favourably with other existing screening programmes, such as breast and cervical cancer.

Thoracic aortic aneurysm

The most common location for thoracic aortic aneurysms (TAAs) is the descending thoracic aorta.

- The majority are degenerative and a minority are caused by Marfan's syndrome, Ehler–Danlos syndrome, syphilis, and connective tissue disorders.
- 25% are related to chronic dissections.
- Other causes include mycotic aneurysms, trauma-related (false aneurysms) and previous coarctation repair.
- They are classified according to their location in relation to the 6th intercostal space; above (type A), below (type B), and the entire descending aorta (type C).
- Risk factors include increasing age and male sex (3:1 ratio). Interestingly, the gender difference is less than that for infrarenal AAAs, where the ratio is 7:1, suggesting a difference in aetiology.
- The incidence is approximately 10/100,000 patient years.
- At 5yrs, only 13–39% of untreated TAAs survive.
- The risk of rupture increases with size. The rupture risk for a 6cm TAA is about 3.6% per year.
- Most clinicians would repair TAAs 6cm in size or more.

Thoraco-abdominal aortic aneurysm

Thoraco-abdominal aortic aneurysm (TAAA) formation affects various segments of the thoracic and abdominal aorta beginning from the left subclavian artery to variable components of the abdominal aorta. By definition, TAAA involves one or more of the origins of the coeliac, superior mesenteric and renal arteries.

- Crawford's classification (with Safi's modification), described five types of TAAAs:
  - *Type I*—from the level of the left subclavian artery extending into the proximal abdominal aorta just above the level of the renal arteries.
  - *Type II*—from the level of the left subclavian artery extending all the way down to the aortic bifurcation.
  - *Type III*—begins in the lower part of the descending thoracic aorta, classically at the sixth intercostal space to below the level of the renal arteries.
  - *Type IV*—begins at the diaphragm to the aortic bifurcation.
  - *Type V*—from the level of the 6th intercostal space of the descending thoracic aorta to just above the renal arteries.

- The aetiology of TAAAs are degenerative (80%), dissection (15%), or connective tissue disorders, arteritis, or trauma (5%).
- Prospective follow-up studies have demonstrated that TAAA rupture was more likely to occur in aneurysms with larger diameters and higher rates of expansion.
- The median size for TAAA rupture was estimated at 7.2cm.
- In aneurysms exceeding 6.0cm in size, the annual rate of rupture or dissection was at least 6.9% and the death rate was 11.8%. The rate of rupture rises exponentially, such that aneurysms equal to or >8cm have an 80% risk of rupture within a year of diagnosis.

Peripheral aneurysm

Popliteal artery aneurysm is the most common peripheral aneurysm, accounting for more than 80%:

- 40% are associated with AAAs.
- 50% are bilateral.
- Unlike AAAs, the majority (70%) are symptomatic.
- The ratio of popliteal aneurysm to AAA is about 1:15.
- 5–10% of patients with AAAs also have popliteal aneurysm.
- The prevalence is about 1% for people in their 8th decade.
- 50% will present as peripheral limb-threatening ischaemia.
- Laminated thrombus within the popliteal aneurysm is an indication for elective surgical intervention to prevent limb loss from embolization as a result of flexion and extension of the knee.
- In the absence of thrombus, 2cm is generally regarded as the threshold diameter for surgical repair.

Femoral arterial aneurysms are the second commonest peripheral aneurysms:

- They occur in 2–3% of patients with AAAs.
- They are also more common with increasing age and show a 30:1 preponderance for men.
- Surgical treatment is usually indicated for size of more than 3 cm.

Isolated iliac aneurysm is unusual and they are usually present in association with aortic aneurysms.

- They involve either common or internal iliac arteries, with involvement of the external iliac artery being an extremely rare event.
- Surgical intervention is generally recommended for asymptomatic iliac aneurysms greater than 3–4cm.

Aortic dissection

Aortic dissection is considered acute if less than 2 weeks since symptoms and chronic if more than 2 weeks.

- The incidence is approximately 3/100,000 per year, but accurate data are difficult to obtain as there is a high out-of-hospital mortality and autopsy rates are low.
- Ratio of men to women is 2:1.
- The risk increases with age.
- Using the Stanford classification, Type A dissection involves the ascending aorta and Type B dissection does not.

- Type A dissection has a 1% mortality per hour and needs emergency surgical repair.
- Type B dissection is mainly treated medically with pharmacological control of BP, but surgical intervention may be needed if complications arise
- Overall mortality for uncomplicated medically treated type B dissection is only 1.2%, but this rises to 18% in complicated dissections (rupture, malperfusion, persistent pain, refractory hypertension, and false aneurysm formation).

Venous disease

Varicose veins have a slightly higher prevalence in males compared with females (4:3.2):

- Age-adjusted prevalence of truncal varices (dilation of veins of the superficial venous system) from the Edinburgh Vein Study was 40% in men and 32% in women.
- The prevalence of smaller thread-like non-truncal varices (reticular or hyphenweb varices) is about 80%.
- 35% of the population between 18 and 64yrs is estimated to have significant venous reflux ≥0.5s.
- The prevalence of varicose veins increases with age.
- Risk factors for primary varicose veins include age, parity (female sex hormones), obesity, standing occupation, diet, and genetics.
- 2° varicosities are caused by previous deep vein thrombosis, pelvic obstruction, or deep venous reflux.

Chronic venous insufficiency (CVI) is the result of impaired venous return and leads to increased ambulatory venous pressure within the lower limbs. This, in turn, leads to skin changes, such as eczema, pigmentation, lipodermatosclerosis, and ulceration. Causes of CVI include venous reflux, venous obstruction, or failure of calf muscle pump.

- The prevalence of CVI is slightly greater in men than women (9% versus 7% in population aged 18–64yrs).
- The prevalence increases with age.

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Screening for vascular disease

Screening programme criteria

Screening is a method to detect disease in a population, in individuals with no signs or symptoms of the disease. The aim of screening is the early identification of disease in an at-risk population, in order to reduce the morbidity and mortality associated with the condition. In order for a screening programme to be of clinical use several criteria must be met:

- The condition should be an important health problem.
- There should be an accepted treatment for patients with the disease.
- There should be adequate facilities for screening.
- There should a recognizable latent phase of the condition.
- There should be a suitable test or examination for the disease.
- The test should be acceptable to the population.
- The natural history of the condition should be understood.
- There should be consensus over which patients to treat.
- The screening programme should be economically viable in terms of overall costs of medical care.
- Case finding should be a continuous process.

UK abdominal aortic aneurysm screening

- In the UK, AAA screening is being gradually introduced, with the timescale for national coverage to be completed by the end of 2013.
- The NHS AAA screening programme invites men for screening in the year they reach the age of 65yrs.
- Men aged over 65yr can request a scan from their local programme.
- Attendees undergo abdominal ultrasound scan to detect an AAA.
- Patients with an aortic diameter of less than 30mm will be discharged; those with an aortic diameter of 30mm or more will enter a local AAA surveillance programme.
- Local programmes will continue to monitor AAA diameter until it reaches the threshold for surgical intervention, at which time patients will be referred to a vascular surgeon for a discussion regarding the risks and benefits of AAA repair.

Sensitivity and specificity

The efficiency of a screening test is of utmost importance. For an effective screening test the disease should be easily detectable, and both those with and without the disease should be identified correctly. This is described by the sensitivity and specificity of the test.

- *Sensitivity* is the ability of the test to correctly identify the disease in patients who have the disease.
- *Specificity* is the ability of the test to correctly identify patients who do not have the disease.
- The positive predictive value (PPV) is the proportion of positive test results that are truly positive.
- The negative predictive value (NPV) is the proportion of patients with a negative test who do not have the disease.

See Table 1.2 for a summary.

The AAA screening programme achieves a sensitivity and specificity of almost 100%. Ultrasound aneurysm screening has a PPV and NPV approaching 100%; therefore, patients without the condition are rarely misclassified as having an AAA, and patients with the condition are rarely misdiagnosed. However, in up to 3% of patients ultrasound screening is not possible when the subject is seen because the abdominal aorta is obstructed by bowel gas, inhibiting visualization.

Population benefits

The aim of any screening programme is to decrease the morbidity and mortality associated with the condition. Mortality from elective AAA surgery is much lower than from aortic rupture (whether or not emergency surgery is undertaken. Therefore, AAA screening leading to elective repair expected to prevent significant numbers of AAA ruptures and deaths.

- The accepted diameter at which AAA's are deemed suitable for repair is 55mm: over this size, the yearly risk of rupture becomes greater than the risk of surgical intervention in a healthy individual.

Table 1.2 The calculation of sensitivity, specificity, and predictive values for clinical tests

Screening result	True disease classification of apparently well population	
	Diseased persons	Persons without disease
Positive	True +ve (with disease and +ve test)	False +ve (without disease but +ve test)
Negative	False –ve (with disease but –ve test)	True –ve (without disease and –ve test)
Total	Total unknown cases of disease	Total persons without the disease

Sensitivity = true +ve/total unknown cases of disease.

Specificity = true –ve/total persons without disease.

Positive predictive value = true +ves/(true +ves + false +ves).

Negative predictive value = true –ves/ (true –ves + false –ves).

- As more patients are identified to have an (asymptomatic) AAA by the screening programme, overall mortality rates from AAA should decrease, although it will inevitably lead still to death in a small number of patients.
- Data from the MASS trial suggested that one life will be saved per 240 men invited to the screening programme (number needed to treat), and one extra death will occur for every 2080 men invited to the screening programme (number needed to harm).
- The MASS trial showed that aneurysm-related mortality was significantly reduced in a screened male population aged between 65 and 74yrs, with a 53% reduction in those who attended for screening
- Over 4yrs, the MASS trial showed that the incremental cost effective ratio for screening was £28,400 per life-year gained (£36,000 per quality-adjusted life year). This falls to approximately £8000 per life year gained at 8yrs.
- This compares favourably with other existing screening programmes, such as breast and cervical cancer.

Limitations of aneurysm screening

As screening targets asymptomatic individuals it has important ethical differences from clinical practice. AAA screening has the potential to save lives. Patients whose aneurysm is 55mm or greater in diameter when screened will be offered intervention (open repair or endovascular abdominal aortic aneurysm repair (EVAR) to prevent rupture), whilst those will small aneurysms can be entered into a surveillance programme to monitor aneurysm expansion with the intention that surgical intervention will be undertaken when warranted. However, screening has limitations:

- False positive results cause increased stress and anxiety to patients, and further unnecessary investigations.
- False negative results give the patient a false sense of security, which may delay final diagnosis in later life
- Aneurysm screening occurs in the 65th year of life. As the age of the population increases, and cardiovascular risk factor modification improves, some patients may develop AAA's after this age, and may therefore not be detected.
- Recent papers have highlighted that the epidemiology of AAA may be changing. The incidence at age 65 may be lower than previously reported, which suggests that screening should be performed at an older age.
- The incidence of AAA differs between different populations and ethnic groups. Extrapolations regarding the incidence of AAA in different populations (and, hence, the benefits of screening) should be made with caution.
- Interventions performed to treat AAA only prevent potential rupture of the AAA. Future morbidity or mortality from other causes is unaffected. Hence some patients will undergo treatment which carries costs and potential risks yet does not extend their lifespan.

Additional screening

In the UK the only vascular NHS national screening programme is for abdominal aortic aneurysms. However, there are several other systems designed to reduce vascular risk factors, as well as commercial services offering vascular screening.

- The NHS Health Check programme aims to help decrease vascular risk through preventing heart disease, stroke, diabetes, and renal impairment through 5-yearly checks between the ages of 40–74yrs.
- Smoking cessation advice is mandatory during any consultation.
- Many private hospitals and clinics advertise vascular screening, including aortic and carotid ultrasonography, blood testing for vascular risk.

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Primary and secondary prevention of vascular disease

Background

- By definition, all patients with occlusive vascular disease have atherosclerosis.
- Individuals with PAD have a 2–6-fold increased risk of death due to coronary heart disease. They are ~4 times more likely to suffer a stroke or transient ischaemic attack than those without PAD.
- Secondary cardiovascular prevention, which may include both lifestyle modification and medical treatment, should be an integral part of the management of all patients with atherosclerotic disease.
- The aim of such treatment is to slow the progression of PAD and to reduce the incidence of cardiovascular events, such as myocardial infarction and stroke.
- The need for effective medical management of patients with PAD is enshrined in guidelines such as that issued by the American College of Cardiology and American Heart Association (ACC/AHA) in 2005 on the management of PAD. Whilst aneurysmal and occlusive arterial vascular disease are distinct entities, many patients with aneurysmal

disease also have significant atherosclerotic disease. The ACC/AHA guidelines recommend that atherosclerotic risk factors should also be actively managed in patients with abdominal aortic aneurysms.
- Other guidelines with recommendations for cardiovascular 2° prevention in patients with known or suspected atherosclerotic disease include the Joint British Societies' Guidelines on the Prevention of Cardiovascular Disease in Clinical Practice (2005), the SIGN Guidelines on Risk Estimation and the Prevention of Cardiovascular Disease (2007), the WHO Guidelines on the prevention of Cardiovascular Disease (2007), and the AHA/ACC guidelines for 2° prevention for patients with coronary and other atherosclerotic vascular disease (Smith *et al.* 2011). These all make similar, but not identical recommendations. The following recommendations are based on the Joint British Societies' Recommendations (JBS2).

Smoking cessation

- All patients should be advised to stop smoking, not only because on the impact on perioperative risk, but also because death, myocardial infarction, and amputation are significantly more frequent in patients with PAD who continue smoke.
- Medical advice and regular follow-up achieves cessation rates of approximately 5%. With nicotine replacement therapy this increases to approximately 16%.
- A smoking cessation programme is best managed in conjunction with the primary care physician or the support of a specialist clinic.

Diet

As with smoking cessation, dietary change is more likely to be achieved with professional support from a dietician or the primary care physician. The JBS2 guidelines recommend the following:
- Aim to maintain body mass index at between 20 and 25kg/m^2.
- Avoid central obesity (defined as a waist circumference in white Caucasians > 102cm in men and 88cm in women, or a waist circumference in Asians of > 90cm in men and 80cm in women).
- Maintain fat intake at ≤30% of total energy intake.
- Keep saturated fat intake at ≤10% of total fat intake and cholesterol intake at <300mg/day.
- Eat at least five portions of fresh fruit and vegetables a day.
- Consume ≤100mmol/day of salt, i.e. <6g of sodium chloride or <2.4g sodium/day.
- Limit alcohol consumption to 21 units a week for men and 14 units a week for women.

Exercise
- Regular physical activity (e.g. fast walking) of at least 30min/day should be taken.
- It has to be recognized that exercising to this intensity may not be feasible in patients with intermittent claudication or ischaemic rest pain. However, exercise should form part of the management of intermittent claudication. A regular supervised exercise programme can increase the speed, distance, and duration of walking.

Blood pressure

Raised BP should be controlled.

- Goals are:
  - <140mmHg systolic and <85mmHg diastolic in non-diabetics.
  - <130mmHg systolic and <80mmHg diastolic people with established atherosclerotic disease, diabetes, and chronic renal disease.
- Concerns that antihypertensive therapy may worsen claudication or critical limb ischaemia (by reducing perfusion pressure) are usually unfounded. Most patients are able to tolerate anti-hypertensive treatment without a worsening of symptoms.

Lipids and statins

Blood cholesterol concentrations should be controlled in people with atherosclerotic disease. This may require changes to diet with or without drug treatment.

Optimal targets are:

- Cholesterol <4.0mmol/L with a low density lipoprotein (LDL) cholesterol of <2.0mmol/L.
- A 25% reduction in total cholesterol and a 30% reduction in LDL cholesterol.
- The target depends on whichever gets the person to the lowest absolute value.

All patients should receive dietary and lifestyle advice directed towards cholesterol reduction. The prescription of a statin is also indicated in most patients presenting for vascular surgery.

Statins

- Studies in acute coronary syndrome suggest that early treatment with a statin after an acute coronary event reduces the incidence of subsequent cardiovascular events, when subsequent compliance is good.
- All patients should be treated with a statin from the time of presentation with an acute cardiovascular event. They should be followed up regularly to ensure that treatment is continued regardless of the initial cholesterol level.
- Most patients presenting for elective vascular surgery will already be on treatment when seen by the anaesthetist. However, patients with an indication for statin therapy may present in pre-assessment. It is inappropriate to initiate lifelong therapy without appropriate follow-up and, therefore, the patient should be referred to their general practitioner with a request to start treatment with a statin if appropriate.
- Before starting treatment it is important to confirm that the patient is statin-naive and that they have not previously discontinued statin treatment because of adverse effects.
- *Important considerations:*
  - Untreated hypothyroidism should be excluded.
  - Dose reduction may be required in severe renal impairment.
  - Specialist advice should be sought in patients with hepatic impairment.

  - Several statins are metabolized by the cytochrome P450 system and, therefore, may interact with drugs including amiodarone, diltiazem, verapamil, and macrolides.
- Five statins are currently available in the UK
  - Atorvastatin.
  - Fluvastatin.
  - Pravastatin.
  - Rosuvastatin.
  - Simvastatin.
- Financial considerations may inform the choice of first-line treatment and local guidance should be consulted.
- Treatment is generally begun with intermediate doses, e.g. simvastatin 40mg. The dose may be reduced or the drug changed in patients who cannot tolerate this.
- High dose treatment, e.g. with atorvastatin 40–80mg or simvastatin 80mg is reserved or those with very high initial cholesterol, progressive disease despite lipid lowering strategies, or following recent acute coronary syndrome.

Adverse effects of statins

- *Myositis (uncommon):*
  - Clinical manifestations range from muscle cramps and myalgia, through to life-threatening rhabdomyolysis. Adverse effects of statins on skeletal muscle are infrequent.
  - Pooled data from randomized controlled trials suggest an incidence of myositis and of rhabdomyolyis of 170 and 20, respectively, per 100,000 patients treated for 5yrs. The pooled incidences of these complications in the placebo group were 150 and 14 per 100,000 patients per 5yrs.
  - Rhabdomyolysis is primarily associated with cerivastatin, which is no longer available.
  - A meta-analysis reported that in the perioperative period a greater than 10 fold increase in serum CK was seen only slightly more frequently in patients who received statin than those who received placebo (0.17% versus 0.13%).
  - The adverse effects of statins on muscle are generally reversible if the drug is discontinued. If myositis is suspected the statin should be discontinued at once.
- Disordered *liver function (infrequent):*
  - Usually manifest as increased hepatic transaminases.
  - Usually occurs in the first year after starting treatment.
  - Incidence is <1% of patients.
  - Routine monitoring of liver function is no longer required in patients starting standard doses of simvastatin or pravastatin, but is still recommended in the drug information for higher doses and other statins.
  - If transaminases increase to >3 times the upper limit of normal, the statin should be discontinued. Specialist advice should be sought in the case of lesser elevations.

Perioperative statin therapy

Statins should be continued throughout the perioperative period.

- Preoperative statin therapy is associated with 59% reduction in the risk of mortality after vascular surgery (1.7% compared with 6.1%).
- Acute statin withdrawal is associated with worse outcome in patients with acute coronary syndrome. There is evidence of a similar effect in vascular surgery patients with one study showing a 2-fold increase in troponin release when statins were discontinued in the perioperative period.

Other drugs

Other lipid-lowering drugs should be considered if the total cholesterol and LDL cholesterol targets are not achieved. These include fibrates, bile acid sequestrants, cholesterol absorption inhibitors, nicotinic acid, and omega-3 fatty acids.

Blood glucose and diabetes

The optimal fasting glucose in people with cardiovascular disease, including vascular surgical patients, is ≤6.0mmol/L.

- If the non-fasting glucose is <6.1mmol/L the fasting glucose does not need to be checked.
- If the fasting glucose is ≥ 6.1mmol/L then the fasting glucose should be measured.
- If the fasting glucose is 6.1 – 6.9mmol/L, but not diagnostic of diabetes (≥ 7.0mmol/L) then it should be repeated or an oral glucose tolerance test (OGTT) should be performed.
- A fasting glucose between 6.1–6.9mmol/L on second testing indicates impaired fasting glycaemia (IFG).
- Repeated fasting glucose concentrations ≥7.0mmol/L indicate diabetes.
- If symptoms of diabetes are present (such as thirst, polyuria, and weight loss), a single fasting glucose of ≥7.0mmol/L indicates diabetes.
- Impaired glucose tolerance (IGT) can only be diagnosed by an oral glucose tolerance test with a 2-h glucose concentration between 7.8 and 11.0mmol/L. A 2-h glucose concentration of ≥11.1mmol/L is diagnostic of diabetes.
- For people with either IFG or IGT the aim is to prevent progress to diabetes through lifestyle intervention. If the person has diabetes tight glycaemic control is recommended with target of a fasting or pre-prandial glucose of 4.0–6.0mmol/L and an HbA1c <6.6%

Cardioprotective therapies

Antithrombotic therapy

Aspirin

- Aspirin 75mg od is recommended for all people with atherosclerotic disease. If aspirin is contraindicated clopidogrel 75mg od may be prescribed.
- Aspirin withdrawal is associated with:
  - A worse outcome in patients with acute coronary syndrome.
  - A significant increase in the risk of cardiac events after non-cardiac surgery.

- No increase in significant surgical bleeding.
- Therefore, aspirin should not be withdrawn from vascular surgery patients in the perioperative period.

Warfarin

Anticoagulation should be considered in cases of:

- Atrial fibrillation with moderate risk of embolic events (aged 60–75yrs without additional risk factors).
- High risk of embolic events (aged >75yrs with other risk factors, such as hypertension, diabetes, or left ventricular dysfunction).
- Target INR is 2–3.
- Patients at risk of systemic embolization, e.g. significant LV dysfunction, LV aneurysm, or a history of paroxysmal tachyarrhythmia.

Beta-blockers

- The POISE study of perioperative high dose metoprolol did not support prophylactic beta-blockade for all patients with established cardiac disease undergoing non-cardiac surgery.
- However, some patients with cardiovascular disease have a primary indication for beta-blockade. These include:
  - Heart failure.
  - Previous myocardial infarction (MI), especially large infarction or MI complicated by heart failure or ventricular arrhythmias.
- The POISE trial raised concerns about hypotension associated with perioperative beta-blockade.
- The withdrawal of beta-blockers in the perioperative period is associated with a significantly increased risk of cardiac events.
- Therefore, beta-blockers should be continued through the perioperative period (with intravenous (IV) substitution if necessary) unless there is significant hypotension.
- If it is deemed appropriate to reduce or discontinue beta-blockade the patient should be regularly monitored and any tachycardia managed appropriately.

ACE inhibitors

- ACE inhibitor or an angiotensin II receptor antagonist therapy are first line therapy in hypertension for people aged <55yrs except for Black people of African or Caribbean origin.
- ACE inhibitors reduce cardiovascular risk by lowering BP.
- They may also have a direct effect on atherosclerosis, by effects on endothelial function, decreasing plasma concentrations of type 1 tissue plasminogen activator, increasing the release of tissue-type plasminogen activator and changing the fibrinolytic balance.
- The importance of these effects in reducing cardiovascular risk is uncertain.

Perioperative management

- Discontinuation of aspirin or statin therapy after MI increases the risk of re-infarction. Similar considerations probably apply in the perioperative period.
- Discontinuation of aspirin therapy for cardiovascular prevention before surgery increases the risk of perioperative MI.

- In the context of vascular surgery, the risks of perioperative infarction outweigh any risk of bleeding associated with continuing aspirin, and the drug should therefore be continued up to the day of surgery and restarted as soon as possible after surgery.
- Some evidence suggests that, in patients on statin therapy, the risks of perioperative MI are increased if the statin is not restarted on the first or second postoperative day. Therefore, statins should be restarted as soon as possible after surgery. As statins have to be given by the enteral route, this may require the administration of the drug in liquid form via a nasogastric tube (NGT).
- Some evidence shows that intraoperative hypotension is more common in patients given an ACE inhibitor or angiotensin II receptor blockers within 12–24h before anaesthesia and surgery. Therefore, many practitioners omit the immediate preoperative dose of these drugs. Because of the risk of post-operative hypotension, the timing for restarting these drugs after surgery is a matter for clinical judgement.

Further reading

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World Health Organization. *Prevention of cardiovascular disease; guidelines for assessment and management of cardiovascular disease*. Geneva: WHO 2007 World Health Organization.

Vascular databases

Outcome data for vascular surgery are collected in a number of national and international databases. These are used for two main purposes:

- To generate epidemiological data.
- To monitor the performance of vascular units.

Both of these functions are relevant to the vascular anaesthetist:

- The epidemiological data from large databases informs clinical practice.
- There is an increasing recognition that outcome from vascular surgery depends on all aspects of care including anaesthetic care.
- It has been shown in at least one vascular unit that anaesthetists with a specialist interest in vascular anaesthesia achieved better outcomes.
- Some vascular databases now include data on anaesthetic care and anaesthetists are recognized as equal partners in initiatives to improve vascular care, such as the UK Quality Improvement Programmes for aortic surgery and amputation.

General databases

Data pertinent to vascular practice have been obtained from large national databases that include, but are not limited to vascular surgery.

American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP (USA))

- A United States national voluntary programme that collects data on all types of surgery, including vascular surgery.
- It generates risk-adjusted data allowing comparison of the performance of different surgical units.
- The ACS NSQIP risk model is complex and requires considerable data collection. Because of this, at least one simpler risk adjustment model has been proposed (the Surgical Mortality Probability Model).
- Analyses of vascular surgery data from ASC NSQIP suggest that general anaesthesia for carotid endarterectomy is an independent risk factor for perioperative MI and that general anaesthesia for EVAR is associated with increased length of stay and an increased incidence of pulmonary complications compared with spinal anaesthesia or local anaesthesia with monitored anaesthesia care.

United Kingdom Hospital Episode Statistics (HES) database

- Contains centrally collected data on activity in National Health Services (NHS) hospitals.
- Analyses of these data for vascular surgery demonstrated volume-outcome relationships for aortic and other vascular surgeries. Units that undertake more procedures tend to have better outcomes.
- In the UK this has led to a national initiative to centralize vascular services so that they are based around a limited number of larger centres.

Specific vascular databases

These have yielded valuable epidemiological data to inform clinical practice.

The EUROpean Collaborators on Stent/graft Techniques for aortic Aneurysm Repair (EUROSTAR) registry

This a database of endovascular procedures undertaken at hospitals in Europe for the repair of aortic aneurysms.

It currently includes data from over 130 centres and over 17,000 across Europe and a MEDLINE search identified 60 publications relating to the registry. Data from the EUROSTAR registry indicate that:

- Endoleak and a requirement from 2° interventions remains a problem with EVAR.
- After 8yrs approximately half of patients who underwent repair with first generation stent grafts remained alive and had not required conversion to open aneurysm repair.

The M2S Medical Imaging Repository

- M2S is a commercial company that provides medical imaging services to hospitals around the world and has provided the core imaging laboratory services for some endovascular device trials.
- Data from large M2S database have been used to study compliance with EVAR device guidelines and post-EVAR aneurysm sac enlargement.

VASCUNET

An international registry of vascular surgery conducted under the auspices of the European Society of Vascular Surgery.

- The first VASCUNET report was published in 2007 and included data on over 30,000 patients who underwent surgery over a 10-year period. This report included data from six countries; Denmark, the UK, New Zealand, Australia, Sweden, and Switzerland.
  - Amongst the findings of this report were the observation that the median age of patients undergoing aortic surgery was 72yrs and that the age of patients undergoing aortic surgery had tended to increase between 1997 and 2006.
  - It was also noted that there were fewer emergency operations, and by inference, ruptures in more recent years.
- The second VASCUNET audit was published in 2008 and included data from 10 countries reported marked differences in outcome from AAA repair between countries. Mortality for open elective open aortic aneurysm repair was higher in the UK than in any of the other nations that submitted data and led to establishment of the national Abdominal Aortic Aneurysm Quality Improvement Programme. (AAA QiP)

National vascular database

- The national vascular database (NVD) was established in 1997 under the auspices of the United Kingdom Vascular Society whose core membership is vascular surgeons.
- Data are entered on a number of 'index procedures', including elective AAA repair, carotid endarterectomy, and lower limb amputation.
- Currently used widely, but not universally, in the UK.
- Recent NHS guidance on commissioning vascular surgery requires that both NHS Trusts and surgeons undertaking AAA repair submit data to the NVD. This is likely to further expand data entry into the NVD.
- The recording of data into the NVD is also one of the key components of vascular surgery quality improvement in the United Kingdom.
- The NVD is explicitly a collaborative venture and includes data fields for preoperative data that may be collected by the anaesthetist and also data fields for intra- and post-operative anaesthetic management. These were developed in collaboration with the Vascular Anaesthesia Society of Great Britain and Ireland (VASGBI) and all vascular anaesthetists are encouraged to register for NVD access (https://nww.nvdonline.nhs.uk), to enter data for cases into the database.

Abdominal Aortic Aneurysm Quality Improvement Programme

- Not a national database itself, but arose from 2nd VASCUNET report and links with other databases.
- Its aims are to improve outcome from AAA repair through several initiatives, including:
  - Expansion of data collection into the NVD—analysis of both these data and data on vascular procedures from the UK HES.
  - The development of best practice protocols for pre-, intra- and post-operative care.

- Support for regional meetings involving all key stakeholders to develop Regional Action Plans.
- Conducting patient focus groups to inform the development of the AAA Quality Improvement Programme.

Useful information

ACS NSQIP. Available at: http://site.acsnsqip.org/

United Kingdom Hospital Episode Statistics. Available at: http://www.hesonline.nhs.uk

European Society of Vascular Surgery VASCUNET. Available at: http://www.esvs.org/social/vascunet

United Kingdom National Vascular Database. Available at: http://www.vascularsociety.org.uk/national-vascular-database.html

United Kingdom Abdominal Aortic Aneurysm Quality Improvement Programme. Available at: http://www.aaaqip.com/

Further reading

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Chapter 2

Anatomy physiology and responses to vascular surgery

Cardiovascular

Embryological development

The cardiovascular system is the first organ system to develop and function in the human embryo starting within 3 weeks of gestation (Fig. 2.1). By week 7 the heart resembles the adult heart, except for the patent foramen ovale. Blood vessels originate from angiogenic cells differentiated from the mesoderm (Fig. 2.2). They cluster and join together to form plexuses within which vacuoles develop into lumens, bordered by endothelial cells, which contain haemangioblasts containing haemoglobin.

Anatomy

The heart

See Figs 2.3 and 2.4.

- The adult heart is situated in the anterior mediastinum and weighs 200–400g.
- Two-thirds of its volume lies to the left of the midline.
- It is divided into left and right sides along its longitudinal axis by atrial and ventricular septa.
- It is divided horizontally by a fibrous septum containing the 4 cardiac valves, which separate the atria from the ventricles.
- Largely composed of muscular tissue (myocardium), attached to the fibrous rings of the atrioventricular (AV) and arterial orifices.
- The heart musculature forms a syncitium of interconnecting cells. This ensures contraction is coordinated to provide maximum efficiency.
- The myocardial conducting system is formed of specialized cardiac myocytes which exhibit an increased length of action potential from the sino-atrial (SA) node to the ventricular myocyte.
- Covered externally by pericardium and internally by endocardium.

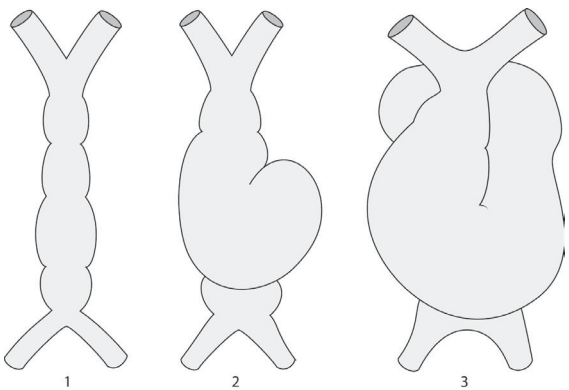


Fig. 2.1 Embryological development of the heart.

Clusters of angiogenic cells join to form endocardial tubes, blood vessels and blood cell precursors (1). The heart tube begins beating, grows, and loops into the adult shape (2, 3).

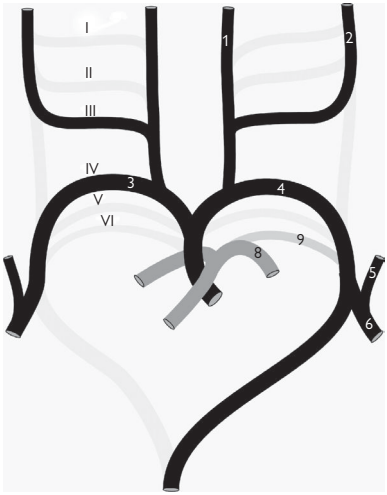
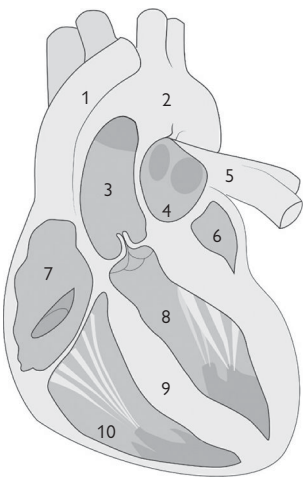


Fig. 2.2 Aortic arches. In early development there are paired dorsal aortae joined to the aortic sac by six arches. The dorsal aortae become the descending aorta.

1. External carotid artery. 2. Internal carotid artery. 3. Right subclavian artery. 4. Aortic arch. 5. Vertebral artery. 6. Left subclavian artery. 7. Right pulmonary artery. 8. Left pulmonary artery. 9. Ductus arteriosus



1. Superior vena cava
2. Aortic arch
3. Aortic root
4. Pulmonary trunk
5. Left pulmonary artery
6. Left atrium
7. Right atrium
8. Left ventricle
9. Interventricular septum
10. Right ventricle

Fig. 2.3 The heart and great vessels viewed from the front.

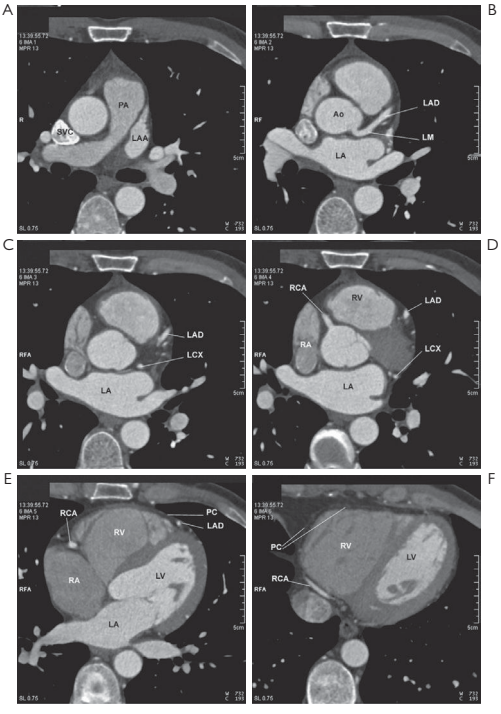


Fig. 2.4 Cross-sectional CT anatomy of the heart. CT images are displayed as if looking from below. The right side is indicated (R), the sternum is in the top of the image. Ao, ascending aorta; CS, coronary sinus; LA, left atrium; LAA, left atrial appendage; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; LM, left main coronary artery; LV, left ventricle; PA, pulmonary artery; PC, pericardium; RA, right atrium; RCA, right coronary artery; RV, right ventricle.

Reproduced from Camm, *et al. The ESC Textbook of Cardiovascular Medicine*, 2nd edn, 2009, figure 6.10, p. 194, with permission from OUP.

The right atrium

- Is thin walled (~2mm thick).
- Normal end-diastolic volume is around 60ml.
- Receives venous blood from the systemic circulation via the superior and inferior vena cavae, and the coronary sinus.
- Is separated from the right ventricle by the AV orifice.
- The AV orifice is an oval shaped fibrous ring of ~4cm diameter. It contains the tricuspid valve, which consists of anterior, posterior, and medial leaflets. These leaflets are attached to papillary muscles whose bases are continuous with the wall and septum of the right ventricle.

The right ventricle

- Semi-lunar in cross-section.
- Has a thin anterior wall (~5mm thick) is rounded and forms most of the anterior surface of the heart.
- Posterior wall is formed by the inter-ventricular septum.
- At the upper left aspect of the ventricle is the conus arteriosus, contiguous with the circular opening of the pulmonary valve.

The pulmonary valve

- Tricuspid in nature.
- Comprises two anterior and one posterior cusp.
- Attached to the wall of the pulmonary artery at its junction with the right ventricle.

The left atrium

- Thicker walled than the right atrium (~3mm).
- Smaller in volume than the right atrium.
- Blood enters from the four valveless pulmonary veins on its posterior aspect.
- Left AV orifice is smaller than right and contains the mitral valve.
- The mitral valve is formed from two unequally-sized leaflets, which are thicker and stronger than those of the tricuspid valve.

The left ventricle

- Has walls three times thicker than the right.
- Internal shape is conical with an almost circular cross-section.
- Makes up most of the inferior and lateral surfaces of heart, and apex.

Vascular system

The vascular system has several functions in addition to blood and fluid transport.

- *Arteries:* conserve the energy from each systolic contraction via intrinsic elastic recoil, returning that energy during diastole to ensure continued flow.
- *Arterioles:* are a major contributor to systemic vascular resistance and controlling organ specific blood flow in response to local or systemic mediators.
- *Capillaries:* large surface area facilitates exchange of oxygen, carbon dioxide, and other substrates.
- *Venules:* low pressure collecting system receives blood from capillaries.
- *Veins:* capacitance vessels able to vary their volume. A reservoir for approximately 60% of the total blood volume.
- *Lymphatics:* are in continuum with the extracellular space. Return fat, fluid and proteins to the circulation.

Arteries and veins share the same basic cellular structure comprising three tunica layers—the adventitia, media, and intima. The proportion of these three component layers varies dependent upon the pressure loading the vessel has to sustain (Fig. 2.5).

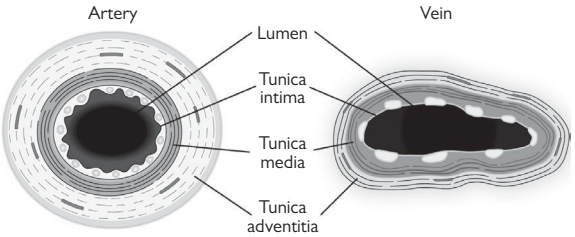


Fig. 2.5 Composition of vessel walls. The tunica adventitia is a dense network of collagen fibres and fibroblasts, which contains nerve fibres and acts to anchor vessels to surrounding structures. The tunica media is composed of circularly arranged smooth muscle cells arranged in bundles, with collagen and elastic fibres. It controls vascular tone in response to local and systemic factors. The tunica intima comprises the endothelium, a single continuous cellular layer with an internal elastic lamina. It is responsible for production of vasoactive mediators, e.g. endothelin.

Systemic circulation

The aorta is the main artery in the body, and of major interest to the vascular surgeon. It is divided anatomically into distinct regions (Fig. 2.6):

- *Ascending aorta:* starts from the aortic valve and is the anterior vertical portion of the aorta. Contains the three aortic sinuses and the ostial origins of the left and right coronary arteries.
- *Aortic arch:* starts from level of right sternoclavicular joint anterior to trachea, and then downwards and backwards to become descending aorta at the level of T4. Major branches include brachiocephalic artery, left common carotid, and left subclavian arteries.
- *Descending thoracic:* from the left side of the T4 medially to directly anterior to T12. Gives off several paired branches:
  - Bronchial arteries.
  - Mediastinal arteries.
  - Oesophageal arteries.
  - Pericardial arteries.
  - The superior phrenic arteries and the main arterial supply to the spinal cord.
- *Descending abdominal:* traverses the diaphragm at the aortic hiatus. Diminishes in size from 25 to 19mm, giving off large branches:
  - Coeliac.
  - Superior mesenteric.
  - Suprarenals.
  - Renals.
  - Gonadals.
  - Lumbars.
  - Inferior mesenteric.
  - Median sacral arteries before dividing into the common iliac arteries at the level of L4.

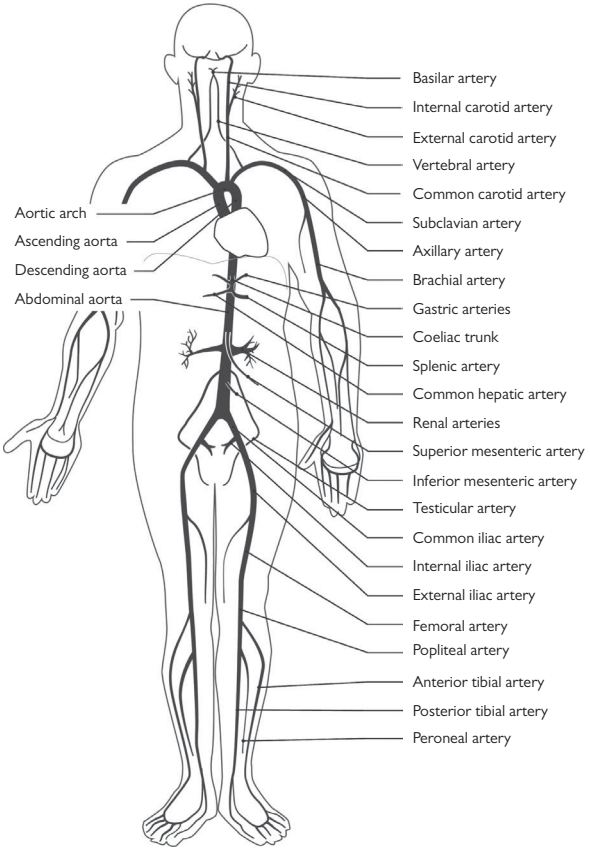
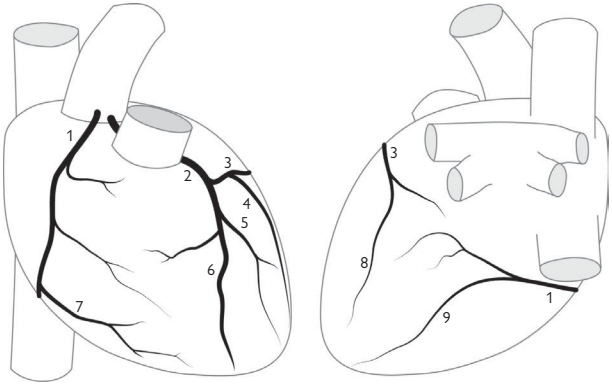


Fig. 2.6 The arterial system.

Coronary circulation

- The left coronary artery (LCA) and right coronary artery (RCA) arise from the aorta just distal to the aortic valve (Fig. 2.7).
- LCA supplies the majority of left ventricle and interventricular septum.
- The RCA supplies the right ventricle and SA node.
- In ~70% of people the origin of the posterior descending artery is the RCA; in others it is the LCA or both.
- Occasionally, there is a third, posterior coronary artery.



1. Right coronary artery
2. Left coronary artery (main stem)
3. Circumflex branch
4. Left marginal branch
5. Diagonal branch
6. Left anterior descending branch
7. Right marginal branch
8. posterior left ventricular branch
9. Right posterior descending branch

Fig. 2.7 The coronary circulation.

- A single coronary artery can occur.
- Anastomoses usually exist:
  - Posteriorly between the left circumflex and right coronary arteries.
  - Inferiorly between the anterior (LCA) and posterior (RCA) interventricular arteries.
- Most coronary venous blood returns to the right atrium via the coronary sinus, with a small percentage draining via Thebesian veins into the left heart and contributing to physiological shunt.

Cerebral circulation and the Circle of Willis

See Fig. 2.8.

- At rest the brain accounts for 20% of total oxygen consumption and receives 14% of cardiac output.
- The 'circle of Willis' is an anastomotic ring arising from the basilar and internal carotid arteries.
- It protects the brain by maintaining cerebral blood flow if flow through one of the tributaries decreases

Spinal cord blood flow

The spinal cord blood supply is depicted in Fig. 2.9.

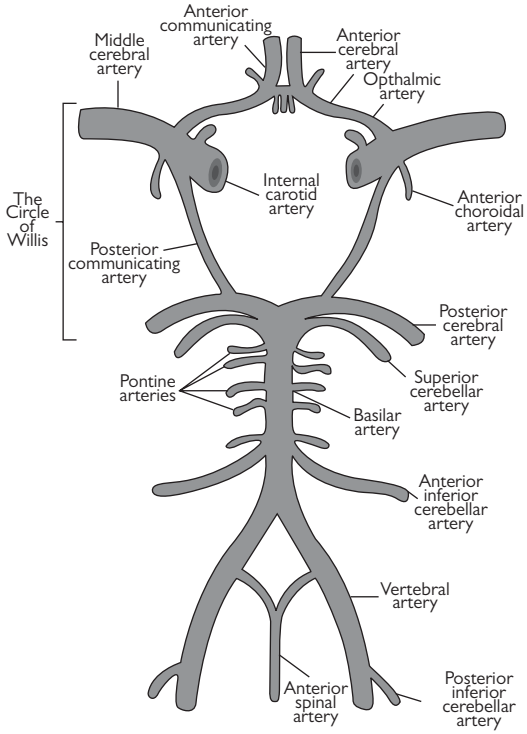


Fig. 2.8 The cerebral circulation and Circle of Willis.

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Major central veins

See Figs 2.10 and 2.11.

- *Internal jugular vein:* begins at the jugular foramen as a continuation of the sigmoid sinus. It receives blood from the brain, face, and neck, and travels down in the carotid sheath with the carotid artery to join the subclavian vein behind the medial end of the clavicle and become the brachiocephalic vein.
- *External jugular vein:* begins behind the angle of the mandible, travels obliquely across the neck in front of the sternomastoid muscle, and pierces deep fascia just above the clavicle to join the subclavian vein.
- *Subclavian vein:* a continuation of the axillary vein. It lies in front of the subclavian artery and anterior to the first rib, and runs behind the clavicle to join the internal jugular vein.

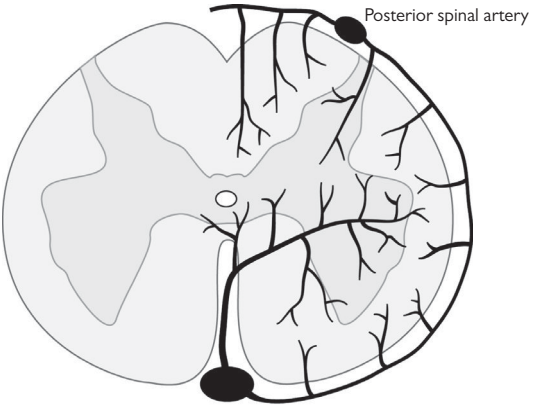


Fig. 2.9 Blood supply to the spinal cord. The spinal cord is supplied by the anterior spinal artery and posterior spinal arteries. These run longitudinally along its length. They are reinforced by the segmental medullary arteries of the cord, which arise from vertebral arteries in the cervical segment, the aorta in the thoracic and lumbar segments, and iliolumbar and sacral arteries in the sacral segment. The artery of Adamkiewicz—the largest segmental artery—arises from the aorta at T9–11 on the left in the majority of individuals and supplies the lower two-thirds of the cord via the anterior spinal artery. Obstruction of the artery of Adamkiewicz can lead to anterior spinal artery syndrome.

Physiology

The cardiac cycle

The cardiac cycle (see Fig. 2.12) comprises four phases that describe the activity of the chambers and valves of the heart. Each cycle is one complete 'heart beat' and, thus, lasts 1s at a heart rate of 60 beats/min.

Phase I: filling

- *Duration:* 0.55s (at 60 beats/min).
- The ventricle is in diastole, with its inlets (mitral and tricuspid) valves open and its outlets (aortic and pulmonary) valves closed. Filling initially rapid due to the 'sucking' effect of the relaxing ventricle; it then slows until augmented by atrial contraction during the final third of the phase.

Phase II: isovolumetric contraction

- *Duration:* 0.06s (at 60 beats/min).
- Systole begins and inlet valves close as soon as ventricular pressure rises above atrial pressure. The ventricle is now closed and pressure rises rapidly without any change in volume.

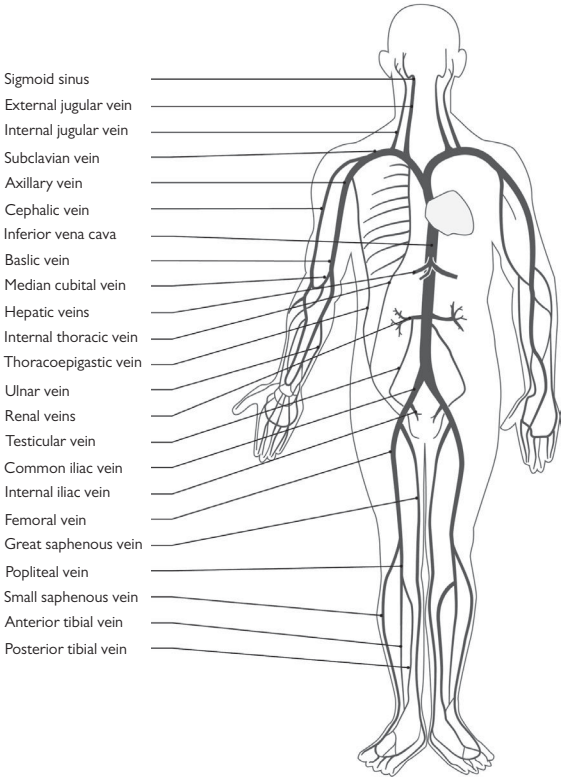


Fig. 2.10 The venous system.

Phase III: ejection

- *Duration:* 0.33s (at 60 beats/min).
- Ejection begins as soon as ventricular pressure exceeds arterial pressure and outlet valves open. The majority of intraventricular volume is ejected in the first half of this phase when the pressure gradient is at its highest. Once ventricular pressure drops to below arterial there will be a momentary backflow resulting in closure of the outflow valve.

Phase IV: isovolumetric relaxation

- *Duration:* 0.09s (at 60 beats/min).
- Diastole begins with all valves closed. Relaxation of the closed ventricle results in a rapid fall in pressure to below that of the atrial pressure at which point the inlet valves open and filling begins again.

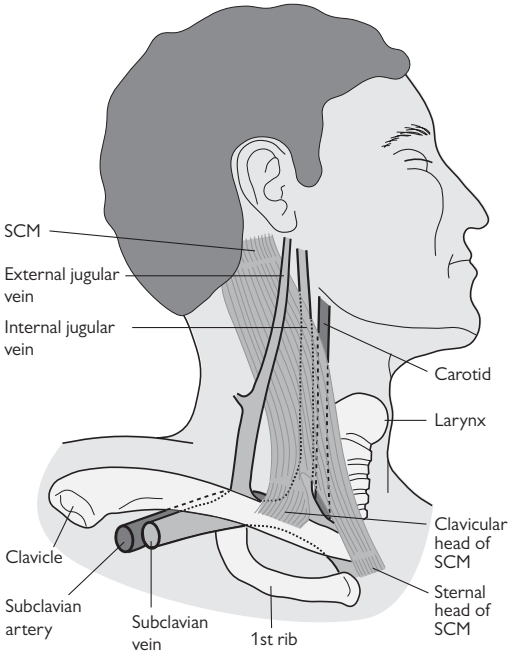


Fig. 2.11 Major veins in the neck.

Reproduced from Raine, *et al*., *Oxford Handbook for the Foundation Programme*, 3rd edn, 2011, figure 16.12, p. 525, with permission from Oxford University Press.

Cardiac electrophysiology

Each heart beat is initiated by modified cardiac muscle cells in the myocardium known as pacemaker cells. This electrical signal is conducted from one cell to the next along a conducting system (Fig. 2.13) until it reaches contractile myocytes, where it initiates an action potential that increases intracellular calcium ions and triggers a contraction. It must be noted that all cardiac myocytes have the ability to act as pacemaker cells.

In normal sinus rhythm, the heartbeat is initiated from pacemaker cells in the SA node. This strip of myocytes is about 2cm long and 0.4cm wide, and located in the posterior wall of the right atrium.

Pacemaker cells uniquely have an unstable plateau phase of their action potential, due to a constant influx of Na^+ ions. A potential of around –60mV is created by the efflux of K^+ ions through voltage-dependant rectifier K^+ channels. This potential then gradually decays until it reaches a threshold potential (TP) at about –40mV causing influx of Ca^{2+} ions through T-type

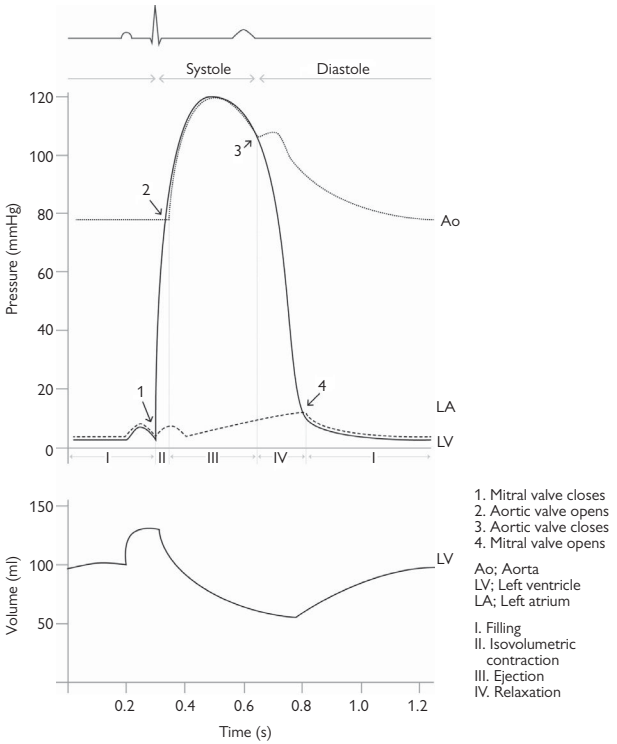
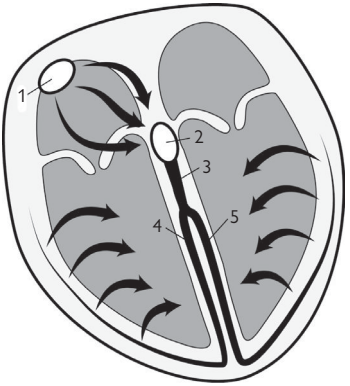


Fig 2.12 The cardiac cycle showing left ventricular pressures and volumes.

Ca^{2+} channels depolarizing the cell and initiating an action potential. The rate of decay (upslope) of the resting membrane potential (RMP) determines heart rate (Fig. 2.14).

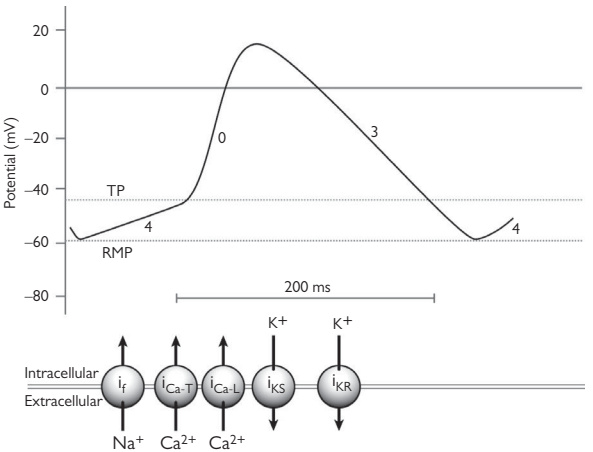
Non-pacemaker cardiac myocytes have five-phase action potentials (Fig. 2.15):

- *Phase 0:*
  - *Rapid depolarization*—the resting potential of –90mV is reduced by a nearby action potential to the threshold potential of –65mV.
  - *Fast Na^+ channels open*—rapid Na^+ ion influx causes complete depolarization and overshoot to about +20mV.
- *Phase 1:* early rapid repolarization—incomplete repolarization to a potential of 0 to –20mV caused by K^+ ion efflux through transiently open K^+ channels.



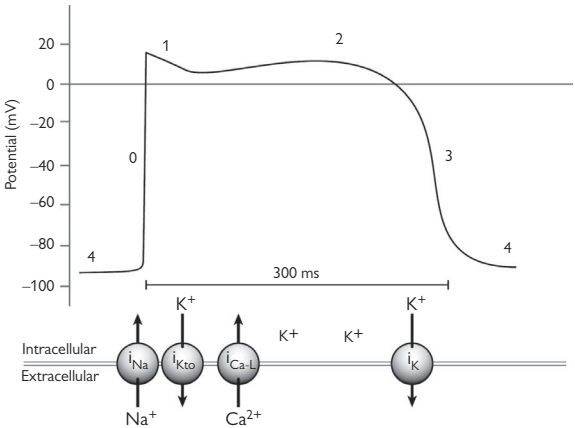
1. Sinoatrial (SA) node
2. Atrioventricular (AV) node
3. Bundle of His
4. Right bundle branch
5. Left bundle branch

Fig. 2.13 Electrical conduction in the heart.



RMP: resting membrane potential, TP: threshold potential, if; 'funny' sodium channel, iCa-T: transient calcium channel, iCa-L: long-lasting calcium channel iKS: slow rectifier potassium channel, iKR: rapid rectifier potassium channel

Fig. 2.14 Sinoatrial node action potential.



iNa: fast sodium channel, iKto: transient outward potassium channel
iCa-L: long-lasting calcium channel, iK: potassium channel

Fig. 2.15 Ventricular myocyte action potential showing movements of ions.

- *Phase 2:* plateau phase—relatively stable period of about 200–400ms. Due to Ca^{2+} ion influx through slow L-type Ca^{2+} channels.
- *Phase 3:*
  - *Final rapid repolarization*—increased K^+ permeability through delayed rectifier or slow K^+ channels.
  - *Facilitates complete repolarization*—whilst termed 'rapid' this occurs at 1/1000th of the rate of depolarization in phase 0.
- *Phase 4:* resting potential—ion gradients restored by ATPase ion pumps.

Excitation-contraction coupling

When an action potential arrives at a cardiac myocyte, it triggers a rise in intracellular Ca^{2+} ions. 10–25% of this rise is accounted for by the influx of Ca^{2+} ions through L-type Ca^{2+} channels during phase 2 of the action potential, whilst the remaining 75–90% is released from the sarcoplasmic reticulum (SR) following ryanodine receptor activation by free $Ca2^+$, termed 'Calcium-induced calcium release'. At the end of systole, the $Ca2^+$ ions are actively returned into the SR and from the myocytes by ATPase dependent pumps. (Fig. 2.16)

Sarcomere structure and function

- Sarcomeres are the basic contractile units and are bundled together to form myofibrils, each about 1μm in diameter.
- These myofibrils are packaged together to form myocytes.
- They have overlapping filamentous proteins between α-actinin end-plates called Z-lines.

1. Actin filament
2. Myosin head
3. Myosin-binding site
4. Tropomyosin

ATP: adenosine triphosphate
ADP: adenosine diphosphate
Pi: inorganic phosphate

a) Resting state. Myosin-binding site is covered by tropomyosin
b) Calcium ion influx causes conformational change of tropomyosin, revealing myosin-binding site
c) Myosin head flexion, using energy from ATP

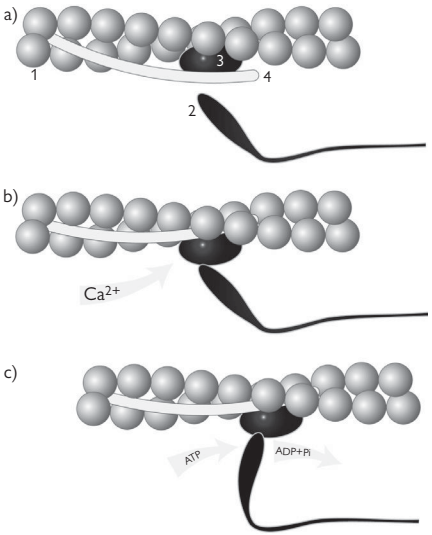


Fig. 2.16 Excitation-contraction coupling.

- Comprise two protein components—thick myosin filaments and thin actin filaments. The myosin filaments have a bulbous head protruding from the side.
- Myosin binding sites on the actin filaments are protected by troponin-tropomyosin complexes, which prevent binding in a resting state.
- An action potential is initiated by influx of Ca^{2+} ions into the sarcomere. This results in displacement of the troponin-tropomyosin complex by Ca^{2+} thus exposing the binding site.
- The myosin heads bind to the actin molecules and flex, resulting in shortening of the sarcomere.
- This occurs in a co-ordinated fashion throughout sarcomeres in many myocytes, resulting in cardiac muscle contraction.

Regulation of blood pressure and cardiac output

Blood pressure

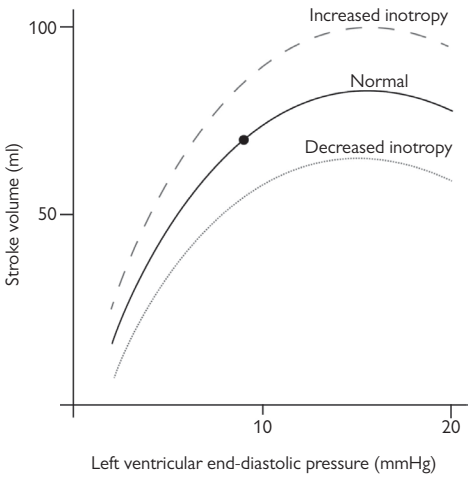
During cardiac cycle, continuous forward flow of blood is maintained in the arterial system despite intermittent ventricular ejection. In systole, the aorta dilates, capturing some of the energy of ventricular contraction and storing it as potential energy. Elastic recoil of the arteries during diastole releases this energy to maintain pressure and forward flow.

- The maximum pressure generated in the aorta during ventricular ejection is the systolic pressure.

- The minimum pressure is the diastolic pressure.
- Difference between systolic and diastolic pressures is pulse pressure.
- Mean arterial pressure (MAP) is the average pressure throughout cardiac cycle, calculated directly by monitoring equipment or can be estimated by adding one-third of the pulse pressure to the diastolic pressure.
- Characteristics of BP change as blood flows through arterial tree. Mean pressure remains relatively constant, pulse pressure widens due to alterations in the relative relationship of systolic and diastolic pressures.
- MAP is a function of cardiac output and systemic vascular resistance and is generally a poor indicator of actual blood flow, which is determined by the components of the Hagen–Poiseuille law (see 🕮 Vascular physiology, p. 42).

Cardiac output

- Defined as the volume of blood ejected by left ventricle per unit time (L/min) and is the product of ventricular stoke volume and heart rate.
- Stroke volume determined by preload, afterload, and contractility (Fig. 2.17).
- Preload is defined as the degree of stretch of cardiac myocytes just before systole. Preload is not measurable in vivo and is usually



Left ventricular end-diastolic pressure (LVEDP) is assumed to reflect left ventricular end-diastolic volume (LVEDV) and therefore myocyte stretch. In clinical settings, pulmonary artery wedge pressure (PAWP) may be approximated to LVEDP as a marker of preload.

Fig. 2.17 Relationship between preload and stroke volume. Frank–Starling's Law of the heart.

Table 2.1 Factors influencing preload

Factors that increase preload	Factors that decrease preload
↑Intrathoracic blood volume	↓Circulating volume
↓Venous compliance	Impaired atrial contraction
↑Ventricular compliance	Arrhythmia/↑heart rate
↑Atrial contractility	↓Ventricular compliance (diastolic dysfunction)
↓Heart rate	Sympathetic blockade
↑Aortic pressure	Anaesthesia

represented by a surrogate, e.g. left ventricular end-diastolic pressure (LVEDP) or volume (Table 2.1).

- Contractility (or inotropy) is defined as the ability of the ventricles to generate force, independent of preload.
- Increased inotropy results in an increased rate of force development and an increased maximal rate of pressure change (dP/dtmax) within the ventricle.
- In clinical practice, ejection fraction (EF, stroke volume as a fraction of end-diastolic volume) is used as an index for contractility.
- Increasing inotropy will increase the percentage i.e. proportion of ventricular volume ejected.
- Afterload is defined as ventricular wall stress and represents the load/force against which the ventricle ejects blood.
- Afterload is proportional to the intraventricular pressure and radius of, and inversely proportional to the wall thickness.
- Aortic pressure is often used as a surrogate for afterload.
- Increased afterload occurs 2° to increases in aortic pressure and systemic vascular resistance, aortic valve stenosis, and ventricular dilatation.
- The consequences of increased afterload are increased end-systolic volume and decreased stroke volume are

Pressure-volume loops

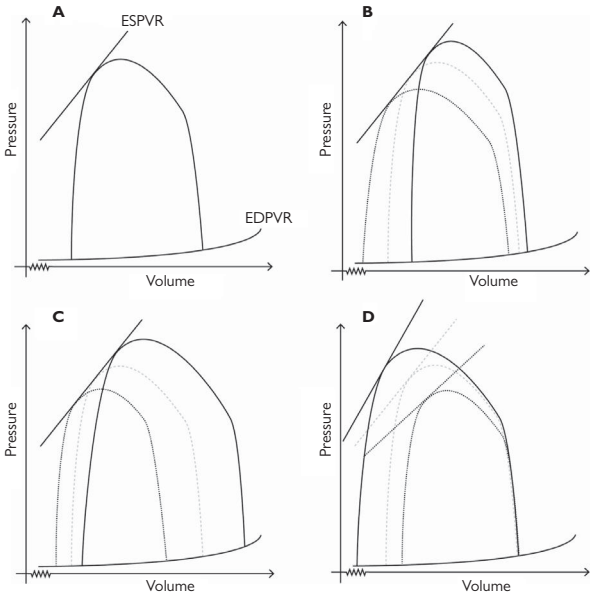
Plotting ventricular pressure against volume during a cardiac cycle gives a useful visual representation of the effect of physiological changes. Stroke volume is depicted by the width of the loop and stroke work by the area within the loop (Fig. 2.18).

Vascular physiology

Systemic vascular resistance is the resistance to blood flow throughout the entire vascular system and crudely calculated by the pressure difference across the system, divided by the flow i.e.

$$SVR = (MAP - CVP) \div CO$$

- SVR is not dependant on BP or flow.
- At a constant cardiac output; increased SVR results in increased MAP.



A. Normal. ESPVR; end-systolic pressure-volume relationship, EDPVR; end-diastolic pressure-volume relationship
B. Effects of increased (solid line) and decreased (dotted line) afterload
C. Effects of increased (solid line) and decreased (dotted line) preload
D. Effects of increased (solid line) and decreased (dotted line) inotropy

Fig. 2.18 Pressure-volume loops.

- Vascular resistance is determined by the components of the Hagen–Poiseuille law:

$$\Delta P = \frac{8\mu LQ}{\pi r^4}$$

where ΔP = pressure change, μ = viscosity, L = length, Q = flow, r = radius.

Vascular tone is regulated by systemic (extrinsic and hormonal) and local (intrinsic) mechanisms, which may be fast feedback responses or chronic adaptations in response to altered metabolic need. The main determinants of vascular resistance are:

- *The radius of the vessels:* resistance to flow is inversely proportional to the fourth power of the radius, so small changes in radius can result in large changes in resistance, if other factors remain constant.

- *Sympathetic tone:* determines vessel radius.
- *Adrenaline and noradrenaline:* cause vasoconstriction via α1-receptors in vascular smooth muscle.
- Blood viscosity and osmolarity.

Coronary circulation

Normal resting coronary blood flow is 70–80mL/min/100g, but can rise to 300–400mL/min/100g during maximal cardiac work. The myocardium has a high basal metabolic requirement for oxygen. Oxygen delivery and extraction must increase to match cardiac work, which can increase more than five times during exercise. These requirements are met through several mechanisms:

- Coronary blood flow and oxygen requirement are coupled in a linear relationship within normal ranges.
- Capillary density is higher than in skeletal muscle, and diffusion distance lower.
- The primary regulator of the coronary circulation is metabolic hyperaemia. Probable vasodilators are adenosine, H_2O_2, K^+, and H^+ ions.
- Metabolic hyperaemia overcomes sympathetic vasoconstriction.
- Autoregulation maintains myocardial perfusion during systemic pressure fluctuations.
- Myocardial oxygen extraction increases at high work rates.
- Adrenaline causes coronary vasodilatation via β2-adrenoceptors.
- Ischaemia is a potent coronary vasodilator.

Cerebral and spinal circulations

The nervous system has a high rate of oxygen metabolism, which varies according to regional activity and is very intolerant of ischaemia, with irreversible damage occurring in less than 5min. The cerebral circulation has special features, which aim to ensure oxygen supply:

- The Circle of Willis consists of anastomoses between the carotid and basilar arteries, which should maintain continuity of flow if one is obstructed. The spinal arterial system is similarly anastomotic.
- High capillary density, similar to that seen in myocardium.
- Brainstem regulation of systemic circulation can divert blood to the brain from other systems.
- Autoregulation maintains relatively constant cerebral blood flow during fluctuations of systemic MAP. Cerebral vasculature dilates in response to increased arterial CO_2 or hypoxia (Fig. 2.19).
- Regional activity stimulation metabolic hyperaemia via K^+, adenosine and eicosatrienoic acids (EETs).

Extrinsic control of the circulation

The central nervous system co-ordinates afferents from pressure receptors in the aortic arch and carotid sinus, and cardiopulmonary receptors in the atria, ventricles, vena cava, and pulmonary arteries.

- Arterial baroreceptors are mechanoreceptors that respond to stretch.
- Cardiopulmonary receptors include ventricular chemoreceptors, myelinated veno-atrial mechanoreceptors, and non-myelinated cardiac mechanoreceptors.

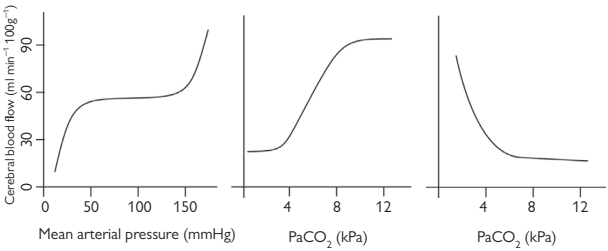


Fig. 2.19 Autoregulation in the cerebral circulation.

Cerebral blood flow is maintained constant when systemic pressure is between 50 and 150mmHg. Cerebral vasodilatation in response to increased $PaCO_2$ increases almost linearly within the normal physiological range. Critical arterial hypoxaemia, below about 6.5kPa causes a profound increase in cerebral blood flow.

Table 2.2 Adrenoceptors—mechanisms and vasomotor actions

Receptor	Mechanism	Location	Effect
α_1	G_q protein; ↑IP3 and DAG	Most vessels	Vasoconstriction
α_2	G_i protein; ↓cAMP	Skin vessels and muscle arterioles	Vasoconstriction
β_1	G_s protein; ↑cAMP	Coronary and cerebral arteries	Vasodilatation
β_2	G_s protein; ↑cAMP	Heart, skeletal muscle, liver	Vasodilatation

IP3, inositol triphosphate; DAG, diacylglycerol; cAMP, cyclic adenosine monophosphate.

- These afferents are integrated in the medulla oblongata with pain and temperature receptors from the reticular system.
- This system responds to peripheral and central input via sympathetic and parasympathetic efferent activity.

There are three types of autonomic vasomotor efferents (Table 2.2):
- Sympathetic vasoconstrictor.
- Sympathetic vasodilator.
- Parasympathetic vasodilator.

These terms refer to the result of increased neuronal activity and a reduction in activity promotes the opposite effect.

Parasympathetic nerves promote vasodilatation via M_3 sub-type muscarinic acetylcholine receptors, which raise intracellular Ca^{2+} via the Gq protein–phospholipase-3 mechanism. Their action is only partially attenuated by muscarinic receptor antagonists; some parasympathetic nerves release non-adrenergic non-cholinergic (NANC) transmitters such as substance P and nitric oxide.

Hormonal control of the circulation

A variety of hormones can affect vascular tone in a number of ways (Table 2.3).

Intrinsic control of the circulation

Autoregulation of blood vessel tone maintains blood flow to protect organs from short-term variations in BP. Main mechanism is the Bayliss myogenic response, arising when vessels subjected to increased pressure are initially distended, causing partial depolarization of myocytes, activating L-type Ca^{2+} channels, and increasing intracellular Ca^{2+} to cause sustained contraction. Other mechanisms also regulate vascular tone.

Endothelial secretions

- Shear stress, stimulates the vascular endothelium to produce nitric oxide (NO)—a local vasodilator—via increased endothelial NO synthase (eNOS).
- Vasodilatation also occurs in response to inflammatory mediators including bradykinin, substance P, and thrombin via activation of eNOS.
- The vasodilatation seen in septic shock is mediated by cytokines that promote the synthesis of inducible nitric oxide synthase (iNOS) and

Table 2.3 Hormones affecting vascular tone

Hormone	Source	Action
Adrenaline Noradrenaline	Adrenal medulla	Vasoconstriction in tissues with α1-adrenoceptors; skin, intestine. Vasodilatation in tissues with β2-adrenoceptors; myocardium, skeletal muscle, liver
Antidiuretic hormone (ADH)	Hypothalamus, via posterior pituitary	Peripheral vasoconstriction Increased water reabsorption in the kidney
Angiotensin II	Renin-angiotensin from kidneys, cleaved in lungs.	Generalized vasoconstriction. Increased aldosterone secretion—retention of salt and water. Stimulation of thirst
Insulin	Pancreas	Stimulates endothelium to produce nitric oxide (NO)—vasodilatation
Thyroxine	Thyroid	Increased basal metabolic rate leads to generalized vasodilatation. Increased density of cardiac β1-adrenoceptors
Oestrogen	Ovaries	Vasodilatation in kidneys, uterus, vagina, breasts, heart, and skin mediated by 17β-oestradiol activation of nitric oxide synthase
Relaxin	Ovaries	Vasodilatation in the uterus, breast and heart by attenuating endothelin-mediated vasoconstriction

the vasodilatation of penile erection by neural nitric oxide synthase (nNOS).
- Endothelin is a peptide vasoconstrictor continuously produced and released by endothelial cells. Endothelin release is stimulated by hypoxia, also increased in heart failure and pre-eclamptic toxaemia.

Vasoactive metabolic factors
- Increased metabolism in skeletal muscle, the myocardium, or in neuronal tissue; results in a rapid and significant increase in local blood flow termed functional hyperaemia. This response is mediated by several vasodilators released from active cells.
- Increased interstitial K^+ ions hyperpolarize myocytes via pH sensitive adenosine triphosphate (ATP)-sensitive potassium (K+) (KATP) channels. Hyperpolarization closes voltage-dependant Ca^{2+} channels and results in vasodilatation.
- Increased CO_2 and lactic acid cause acidosis leading to vasodilatation via myocyte hyperpolarization and endothelial NO release.
- Local hypoxia induces vasodilatation via several complex mechanisms that are independent of endothelium produced mediators.
- Adenosine is a vasodilator in the sustained response of skeletal muscle to exercise.

Vasoactive paracrine secretions
- Histamine is a chemical mediator of inflammation. It is formed by decarboxylation of histidine, stored in leukocytes and mast cells, and released in response to trauma or allergy. H_2 receptors are Gs coupled and cause vasodilatation via the adenyl cyclase pathway. H_1 receptor stimulation causes increased vascular permeability.
- Serotonin (5-hydroxytryptamine) is produced by enterochromaffin cells, endothelium, and neurons. It is also stored in platelets. Serotonin causes vasoconstriction, increased permeability and pain.
- These responses are responsible for vasoconstriction after endothelial damage and platelet activation.
- Prostaglandins and thromboxane are products of arachidonic acid metabolism by cyclo-oxygenase enzymes, and leukotrienes by 5-lipoxygenase. These contribute to reactive hyperaemia, vasodilatation, and the oedema of inflammation.

Temperature
- Relevant mainly to the cutaneous circulation.
- Heat-induced vasodilatation is mediated by increased eNOS activity.
- Cold-induced vasoconstriction mediated largely by α_2-adrenoceptors.

Pathophysiological responses
- *Metabolic hyperaemia:* there is an almost linear increase of blood flow with increased metabolic demand in skeletal muscle, myocardium, and brain. This response is mediated entirely by intrinsic factors with no central nervous input.
- *Post-ischaemic hyperaemia:* blood flow to tissues increases after removal of an obstruction to blood flow. The blood flow subsequently declines at an exponential rate back to its normal value. After short arterial

occlusions, the mechanism is largely a myogenic response—reduced pressures in vessels distal to obstruction result in vasodilatation. After longer occlusion, this response is augmented by accumulation of vasoactive metabolites and the release of sensory nerve vasodilator neuropeptides.

- *Ischaemia-reperfusion injury:* restoration of blood flow after prolonged periods of ischaemia can result in tissue damage:
  - Damage is mediated by leucocytes, oxygen, and increased Ca^{2+} concentration.
  - Leucocytes adhere to ischaemic endothelial cells and physically obstruct the microcirculation.
  - Oxygen is converted into free radicals by ischaemic tissue.
  - Free radicals are highly reactive and react with lipids and amino acids damaging cell membranes and enzymes.
  - Ischaemic myocardial cells can become overloaded with Ca^{2+} resulting in severe and sustained contracture with reperfusion related cell damage and necrosis.

Further reading

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Pathophysiology of aortic clamping and unclamping

Aortic cross-clamping and unclamping are key events in open aortic surgery. The vascular anaesthetist must know the physiological changes likely to occur, and be able to predict their potential impact on patients who are usually elderly and may have significant cardiovascular disease. Clinical experience and the ability to modify these changes are also essential to prevent adverse effects of aortic clamping and unclamping.

Aortic cross-clamping

Haemodynamic changes

The 1° cardiovascular disturbances caused by aortic cross-clamping are:

- Sudden increase in the resistance to forward flow in the aorta.
- Decrease in perfusion to organs and tissues distal to site of clamp.

These result in:

- Acutely increased arterial pressure.
- Decreased cardiac output, largely due to a decrease in left ventricular ejection fraction.
- Little early change in heart rate.
- Variable changes in indices of cardiac function and circulatory volume. Central venous pressure may increase, decrease, or remain the same.
- Increased sympatho-adrenal activity resulting in increases in plasma catecholamine concentrations.

Factors affecting the extent of the haemodynamic response:

- *The site of the cross-clamp on the aorta:* the more proximal the clamp, the greater the increase in arterial pressure above the clamp and the greater the decrease in ejection fraction.
- Intravascular volume status before clamping.
- *The extent of blood volume redistribution after clamping:* aortic clamps placed above the renal and visceral vessels (often called the 'supracoeliac' aorta) are often associated with an increase in venous return as passive recoil in the high-capacity splanchnic vessels moves blood volume proximal to the clamp. Infra-renal clamps may be associated with a decrease in venous return as blood pools in the splanchnic reservoir. The extent of the changes in venous return 2° to changes in the splanchnic circulation depend upon the depth of anaesthesia, anaesthetic technique used, use of vasodilator or vasoconstrictor drugs, and circulating volume.
- Pre-operative coronary circulation and myocardial function.
- Anaesthetic techniques used to modify the responses.
- *Surgical pathology:* haemodynamic changes are greater during surgery for aortic aneurysm compared with surgery for occlusive aortic disease. In the latter situation, numerous arterial collaterals have usually developed and the impact of clamping is usually less.

Other changes at aortic cross-clamping

These include:

- Decreased oxygen consumption in tissues distal to the clamp.
- Decreased venous return from hypoperfused regions of the body. Combined with decreases in overall oxygen consumption, this leads to increased mixed venous oxygen saturation.
- *Metabolic acidosis and increased lactate concentrations:* these are proportional to the level of the clamp—supra-coeliac clamps can be associated with marked and rapid changes.
- Increased renin activity and angiotensin concentrations.
- *Accumulation of harmful mediators in ischaemic regions*: including cytokines, endotoxins, tumour necrosis factor, free radicals, prostaglandins and other substances. These cause vasodilatation and myocardial depression, and will worsen both hypotension after clamp release and reperfusion injury.

Aortic unclamping

In principle, the physiological changes resulting from the removal of an aortic clamp are the reverse of those associated with its placement. However, the extent of the changes depends on:

- The level of the clamp.
- The duration of aortic clamping.
- Cardiac function and intravascular volume status at clamp release.

Physiological effects

- Vasodilation in all vascular beds distal to the clamp results in:
  - A dramatic decrease in aortic impedance.
  - A marked decrease in systemic vascular resistance.
  - A marked decrease in venous return.
  - Redistribution of blood volume.

- At the same time, blood returning from ischaemic areas will be acidaemic, with high concentrations of lactate, potassium, and other products of anaerobic metabolism. Arterial and venous oxygen saturations often decrease acutely, but return to normal as circulation normalizes.
- An initial acute decrease in arterial pressure is frequently followed by a 2° decrease 10–15min after unclamping. This may coincide with a maximal reactive hyperaemia response.
- The extent and timescale of the changes associated with unclamping can be modified by gradual or intermittent release of the clamp, and communication with the surgeon. Aortic unclamping is much easier to manage if the anaesthetist has been informed as to when it is going to be performed. This allows time for preparation and administration of fluids, vasoactive drugs and other measures to attenuate the physiological disturbances (see 📖 Open aortic aneurysm repair, p. 350).

Re-clamping the aorta

- Re-application of an aortic clamp is often required during aortic surgery. The main indication is that bleeding revealed by the re-establishment of aortic blood flow on clamp removal cannot be safely stemmed without re-applying the clamp.
- A particularly difficult challenge to the anaesthetist because clamp is being applied when pathophysiological effects of unclamping (vasodilatation, metabolic acidosis, ischaemia reperfusion injury, myocardial depression) are at their peak. Often compounded by concurrent acute brisk haemorrhage, hypovolaemia, and sometimes coagulopathy.
- The repeated application and removal of aortic clamps during periods of brisk haemorrhage and huge changes in cardiovascular physiology are one of the greatest challenges to the vascular anaesthetist. Good communication with the surgeon is essential

Clinical management of aortic clamping and unclamping

See 📖 Open aortic aneurysm repair, p. 350.

Further reading

Gelman S. The pathophysiology of aortic cross-clamping and unclamping. *Anesthesiology* 1995; **82**: 1026–60.

Respiratory

Anatomy of the respiratory system

Trachea

- The trachea extends in the midline from the lower border of the cricoid cartilage (C6) to the carina (T4), where it bifurcates into the two main bronchi. The carina is at T6 on deep inspiration.
- Normal length 10–15cm.
- Often elliptical in cross-section (transverse dimension > anteroposterior (AP).

- *Normal dimensions in adult male:* 20–23mm transverse, 15–20mm AP. Slightly smaller in females
- Tracheal wall is reinforced by 15–20 cartilaginous rings. These are incomplete posteriorly, approximately 4mm wide and 1mm thick. Tracheal wall and cartilages are vulnerable to ulceration if a tracheal tube cuff is inflated to a high pressure.
- Trachea is crossed anteriorly in the upper thorax by the brachiocephalic artery and vein.
- The common carotid, subclavian, and (inferiorly) the aortic arch lie to the left of the trachea. The trachea may be deviated to the right by the aorta, especially in the presence of a thoracic aortic aneurysm

Bronchi

See Fig. 2.20.

Right main bronchus and divisions

Right main bronchus is wider and less steeply angled from the midline (30°) compared with the left main bronchus (45°), although there is wide individual variation in these angles. Divides into three lobar bronchi (upper, middle, and lower), which give rise to ten bronchopulmonary segments (Fig. 2.20).

Left main bronchus and divisions

Left main bronchus slightly narrower than the right and divides into two lobar bronchi (upper and lower), which give rise to nine bronchopulmonary segments, each supplied by a 3rd order bronchus (Fig. 2.20).

Surface anatomy

- Lungs and dome of the pleura extend 2.5cm above the junction between the middle and inner thirds of the clavicle.
- Lung borders descend behind the medial border of the clavicles to meet in the midline behind the manubrium at the 2nd costal cartilage.

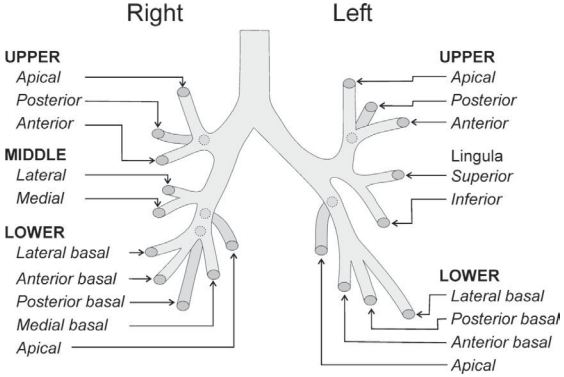

Fig. 2.20 Bronchi and bronchopulmonary segments.

- Anteriorly the two lung lines deviate at 4th costal cartilage (T7).
- Medial border of left lung is displaced laterally at T7 to left of the sternal edge as far as 6th costal cartilage (cardiac notch).
- Medial border of right lung continues vertically to 6th costal cartilage.
- Both lines of lung reflection then deviate laterally and inferiorly through 8th rib at mid-clavicular line, 10th rib at the mid-axillary line, and 12th rib at the paravertebral line.
- Diaphragmatic reflection of the pleura extends below the lung to the lower border of T12.

Bronchoscopic anatomy

See Figs 2.21, 2.22, and 2.23.

- Knowledge of bronchoscopic anatomy is essential when double lumen endobronchial tubes (DLTs) are used during some vascular procedures e.g. thoracoscopic sympathectomy (📖 Thoracoscopic sympathectomy, p. 266) or thoracic AAA repair (📖 Open aortic aneurysm repair, p. 350). The correct positioning of a DLT should ideally be confirmed by bronchoscopy.
- Contains 16–20 cartilaginous tracheal rings, which are incomplete posteriorly, but are visible anteriorly.

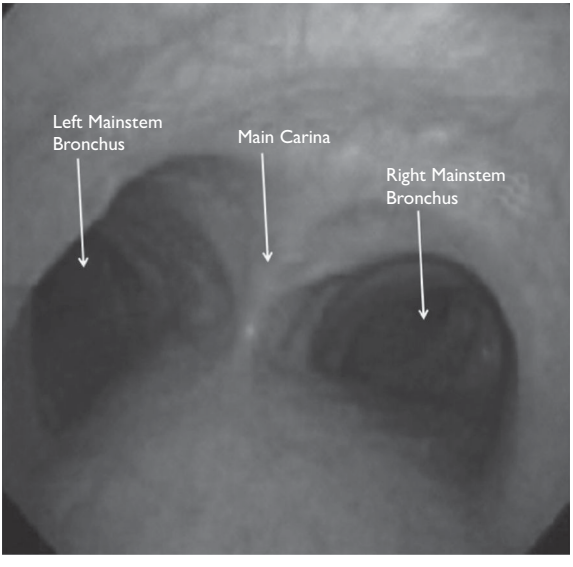

Fig. 2.21 Bronchoscopic view of the carina, right and left main bronchi.

Reproduced from Wilkinson, *et al*. *Oxford Specialist Handbook of Thoracic Anaesthesia*, 2011, figure. 2.2, p. 41, with permission from OUP.

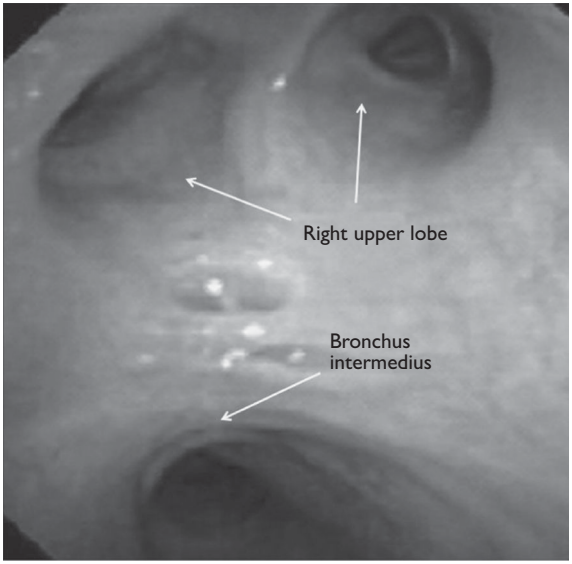

Fig. 2.22 Bronchoscopic view of the right main bronchus showing its divisions.

Reproduced from Wilkinson, *et al*. *Oxford Specialist Handbook of Thoracic Anaesthesia*, 2011, figure 2.4, p. 42, with permission from OUP.

- Posteriorly, the trachea consists of connective tissue and the trachealis muscle, the longitudinal fibres of which are visible extending into the two main bronchi. Cardiac pulsations are visible. The muscle stripes are not present in the right and left main bronchi.
- Average tracheal length is 10–15cm.
- Average distance from incisors to carina is 29cm for a patient of 170cm height. This increases by 1cm for every 10cm increase in patient height.

Right main bronchus

- Angled at 30° to carina; usual diameter ~16mm.
- Gives off right upper lobe (RUL) bronchus after 2.5cm usually at an acute angle .
- Note that anatomy can vary and RUL bronchus can arise at the level of right middle lobe (RML) and right lower lobe (RLL) bronchi, or rarely arise directly from the trachea.
- Right main bronchus (RMB) continues as bronchus intermedius which gives off RML bronchus anteriorly and then RLL bronchus.

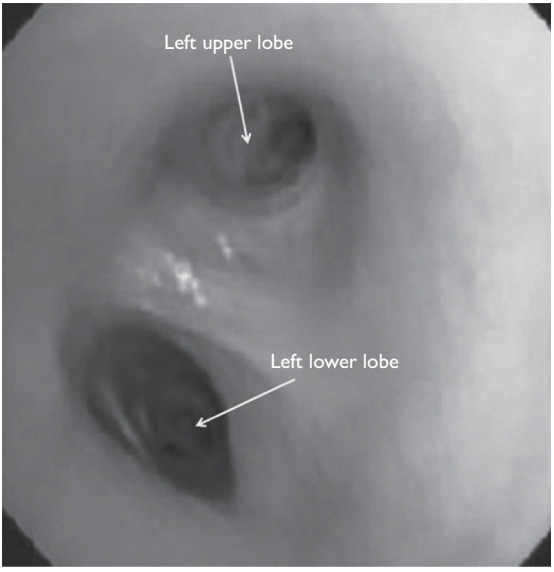

Fig. 2.23 Bronchoscopic view of the left main bronchus showing its divisions into upper and lower.
Reproduced from Wilkinson, *et al*. *Oxford Specialist Handbook of Thoracic Anaesthesia*, 2011, figure 2.3, p. 42, with permission from OUP.

Left main bronchus

- Angled at 45°; usual diameter ~13 mm.
- Divides into left upper lobe (LUL) bronchus laterally and left lower lobe (LLL) bronchus posteriorly after ~6–7 cm in men and 5–6cm in women.

Respiratory effects of general anaesthesia

Both general anaesthesia (GA) and regional anaesthesia (RA) can impair the normal functioning of the respiratory system:

- Normally minimal compared with the adverse effects of the surgical procedure, particularly in the post-operative phase.
- Effect depends on the size and proximity of the incision to the diaphragm, and the duration of surgery.
- Effects are greatest with open abdominal aortic surgery > endovascular repair > peripheral vascular surgery (normally little effect).

Respiratory control

- All commonly used anaesthetic agents cause respiratory depression.
- Resting alveolar ventilation is reduced.
- The responses to hypercapnia and hypoxia are reduced.
- Significant hypercapnia inevitably develops a few minutes after induction of anaesthesia with spontaneous ventilation, but $PaCO_2$ does not rise further with prolonged anaesthesia.

- Hypercapnia and its associated acidosis have adverse effects, so controlled ventilation is usually used for vascular surgery.
- Impaired ventilatory responses to hypoxia can be dangerous in the post-operative period.
- The administration of small amounts of supplemental oxygen delays the onset of hypoxaemia if alveolar ventilation is reduced.
- This risks the patient developing hypercapnia, although this is probably preferable to hypoxaemia.
- In patients at risk of respiratory depression, both SpO2 and respiratory rate should be monitored regularly.

Respiratory mechanics and atelectasis

- GA, with or without neuromuscular blockade, immediately alters respiratory muscle function, chest wall dynamics, and the position and shape of the diaphragm.
- Thoracic volume and functional residual capacity (FRC) reduce by ~10% within a few minutes of induction, but do not progress further.
- When ventilation-perfusion ($\dot{V}/\dot{Q}$) relationships within the lung deteriorate:
  - The spread of $\dot{V}/\dot{Q}$ ratios increases.
  - In some areas $\dot{V}/\dot{Q}$ increases, so increasing alveolar dead space and decreasing CO_2 elimination.
  - In other areas $\dot{V}/\dot{Q}$ decreases, causing venous admixture and impaired oxygenation.
  - In lung regions with very low $\dot{V}/\dot{Q}$ ratio (commonly in dependent lung regions), airway closure will occur, causing lung collapse, and atelectasis.
  - Atelectasis is believed to occur in over 75% of patients (Fig. 2.24).

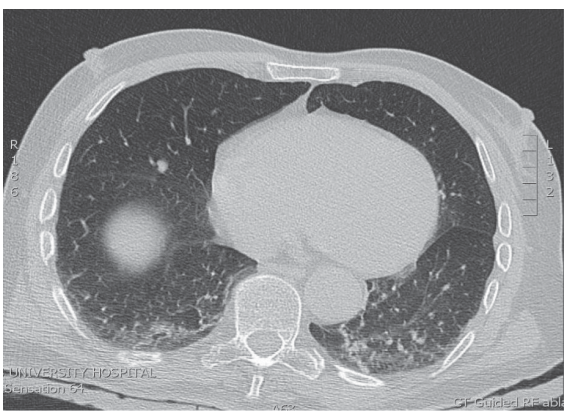

Fig. 2.24 Computered tomography scan of a patient with healthy lungs during general anaesthesia. The slice shown is immediately cephalad to the right diaphragm. Bilateral atelectasis is seen, which becomes progressively more dense on moving from the non-dependent (anterior) to dependent (posterior) regions.

- Atelectasis is worse in the obese and after administration of 100% oxygen (e.g. preoxygenation before induction of anaesthesia, for recruitment manoeuvre during GA or before extubation; Box 2.1).
- The use of 100% oxygen before extubation may cause post-operative atelectasis.

Avoidance and re-expansion of atelectasis.

- If only small areas of lung have a low $\dot{V}/\dot{Q}$ ratio, a small increase in Fi_{O2} prevents hypoxaemia.
- Increasing Fi_{O2} is less effective with larger areas of low $\dot{V}/\dot{Q}$ or atelectasis and a recruitment manoeuvre should be performed.
- In susceptible patients or after a recruitment manoeuvre, moderate levels (5–10cmH_2O) of positive end expiratory pressure (PEEP) should be used.

Respiratory mechanics

- Reduced lung volume will affect airway calibre and so increase respiratory system resistance.
- However, during anaesthesia with either inhaled or IV agents airway dilation also occurs as a result of inhibition of airway bronchoconstrictor reflexes.
- These effects balance out so there is little change in airway resistance.
- Reduced smaller lung volumes also reduce lung compliance, particularly if atelectasis is present.

Respiratory effects of regional anaesthesia

- Neuraxial blocks involving only lumbar nerve roots or below have no clinically relevant effects on respiration.
- Thoracic epidural anaesthesia or analgesia may reduce the ribcage contribution to ventilation, and at high minute volumes, stimulated, e.g. by hypercapnia, may reduce tidal volume.
- High thoracic epidural anaesthesia increases FRC as a result of caudad movement of the diaphragm and reduced thoracic blood volume.
- The muscles required for expulsive respiratory efforts, such as coughing, are inhibited by thoracic epidural or spinal anaesthesia.

Box 2.1 Lung recruitment manoeuvres for patients with healthy lungs during GA

- *Vital capacity manoeuvre:* increase inflation pressure to 40cmH_2O for 8–10s, using the reservoir bag and expiratory valve. Some ventilators can do this automatically. During the manoeuvre, temporary cardiovascular depression occurs. An inflation pressure of 30cmH_2O is partially effective and has less cardiovascular effects.
- *PEEP and large tidal volumes:* increase PEEP by 5cmH_2O every five breaths up to 15cmH_2O. Then increase tidal volume until peak inspiratory pressure (PIP) is 40cmH_2O, maintain for ten breaths then reduce tidal volume and PEEP in the same stepwise fashion. This manoeuvre has less cardiovascular side effects than a vital capacity manoeuvre

- However, these effects of RA have almost no clinical impact in non-pregnant patients.
- The beneficial effects of epidural analgesia are:
  - Improved analgesia, which improves the ability to cough and comply with chest physiotherapy.
  - Reduction of diaphragmatic dysfunction (reflex inhibition of diaphragm contraction by trauma following major abdominal surgery).
  - These beneficial effects of RA on respiratory function, therefore, easily outweigh detrimental effects.

Reperfusion and the lung

Pulmonary responses to arterial declamping

- The release of an arterial clamp placed during any major arterial reconstruction surgery allows reperfusion of those areas of the body supplied by the relevant artery.
- This blood containing low PO_2, high PCO_2, and other products of anaerobic metabolism re-enters the circulation.
- ∴ arterial declamping normally causes temporary increase in arterial PCO_2, end-tidal PCO_2 ($P_E'CO_2$) and decreases in PaO_2, SpO_2, and pH.
  - This should be predicted and actively observed.
  - The increased PCO_2 and decreased pH can usually be managed by increasing minute ventilation temporarily.
  - If $P_E'CO_2$ does not increase, anaesthetist should inform the surgeon: reperfusion may be inadequate because of a problem with the graft.
  - The decrease in PaO_2 may be marked when reperfusion of large areas of body tissue occurs, such as following aortic declamping.
  - This demonstrates the pulmonary effects of ischaemia-reperfusion injury, and is more common after clamping of the thoracic aorta compared with an infra-renal clamp.
- Two main causes of impaired lung function on aortic de-clamping:
  - Pulmonary hypervolaemia 2° to impaired myocardial function
  - Pulmonary hypertension as a result of acidosis, catecholamine release, and activation of the renin-angiotensin system.
- Consequently $\dot{V}/\dot{Q}$ relationships in the lungs (already abnormal as a result of GA) become further disturbed and hypoxaemia results.
- Increasing FiO_2 will attenuate arterial desaturation by improving oxygenation of blood in areas of low $\dot{V}/\dot{Q}$ ratio.
- Careful cardiovascular management is also required to reduce the effects of declamping on the pulmonary circulation.

Aortic surgery and acute lung injury

Acute lung injury (ALI) is common after aortic surgery is common, particularly following thoracic aortic procedures and emergency surgery for ruptured aneurysm. Many factors contribute, including:

- The normal systemic inflammatory response to major trauma.
- Transfusion-related ALI.
- Iatrogenic fluid overload.

- The substantial systemic inflammatory response to open aortic surgery that makes this particular procedure high risk. When compared with other abdominal or thoracic procedures, aortic surgery causes:
  - Production of reactive oxygen species on reperfusion of the tissue distal to the clamp.
  - Prostaglandin release, possibly from mesenteric traction.
  - Complement activation.
  - Endotoxin release from bowel in response to intestinal ischaemia during clamping.
  - Release of numerous pro-inflammatory cytokines, e.g. IL-6, IL-10, TNF-α.
- The systemic inflammatory response causes neutrophil sequestration and activation in the lung, increased permeability of pulmonary capillaries with consequent oedema.
  - There are no specific interventions that will prevent this sequence of events, but careful fluid management can ameliorate the adverse effects on gas exchange.

Further reading

Lumb AB. *Nunn's Applied Respiratory Physiology*, 7th edn. Edinburgh: Elsevier, 2010.

Posner M, Gelman S. Pathophysiology of aortic cross-clamping and unclamping. *Baillieres Clinical Anaesthesiology* 2000; **14**(1): 143–60.

Anatomy relevant to regional anaesthesia

The spinal cord and epidural space

- The spinal cord is enveloped by the dura, arachnoid and pia maters, which divide the vertebral canal into three compartments.
- The e*pidural space*, containing adipose tissue and blood vessels, lies between the dura mater and the bony structure of the spinal canal; the *subdural space* is a potential space between the dura and the arachnoid mater; and the subarachnoid space contains cerebrospinal fluid (CSF), spinal cord and nerve roots. (Fig. 2.25).
- The epidural space is described as discontinuous and segmented. Segmentation is attributed to thin meningovertebral ligaments, opposition of the dura with laminae and discontinuous epidural fat. The space becomes more continuous in the thoracic region.
- Epidural veins are large, valveless and communicate with the basivertebral and abdominal veins. Pressure changes within the chest and abdomen are reflected in the size of epidural veins.
- Subarachnoid space lies deep to the arachnoid mater. It contains the spinal cord, dorsal and ventral nerve roots, and CSF. The spinal cord extends to the lower border of the first lumbar vertebra The pia mater continues caudally as a thread-like filum terminale, ultimately attaching to the coccyx. There are 31 pairs of spinal nerves (8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal), each formed from a dorsal and a ventral root. (Fig. 2.26)

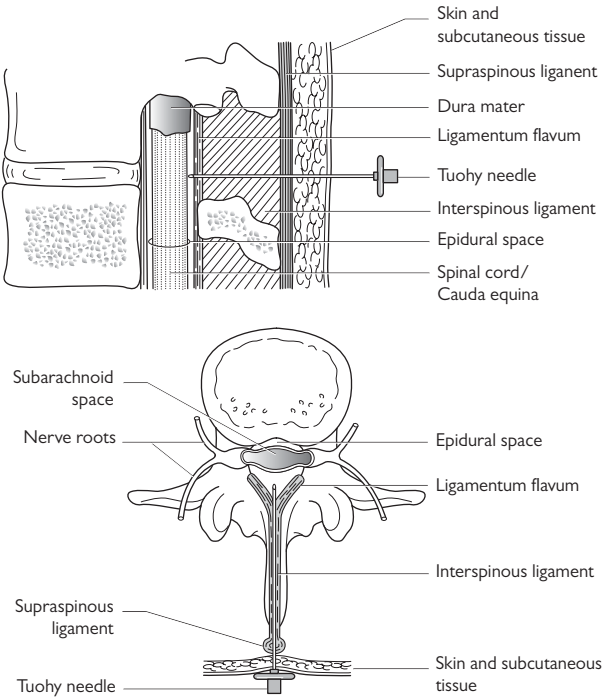

Fig. 2.25 Epidural and subarachnoid spaces.

Reproduced from Brook et al., Oxford Handbook of Pain Management, 2011, Figure 3.1, p. 45, with permission from Oxford University Press.

- Lumbar and sacral roots lie freely within CSF below the level of the conus medullaris to form the cauda equina.
- The spinal cord is supplied by an anterior spinal artery formed from each vertebral artery and two posterior spinal arteries, branches of the posterior inferior cerebellar artery. Spinal branches of the vertebral, cervical, intercostal, and lumbar arteries augment blood supply (Fig. 2.27) Venous drainage is through a plexus of anterior and posterior veins, which drain along the nerve roots into epidural and segmental veins.
- CSF is normally clear and colourless with a density of 1.006 at 37°C, although this increases to 1.010 with age
- CSF is produced in the choroid plexuses of the ventricles. Total CSF volume is approximately 130mL, one-quarter as spinal CSF. Pressure is negative in the upper and middle thoracic regions because

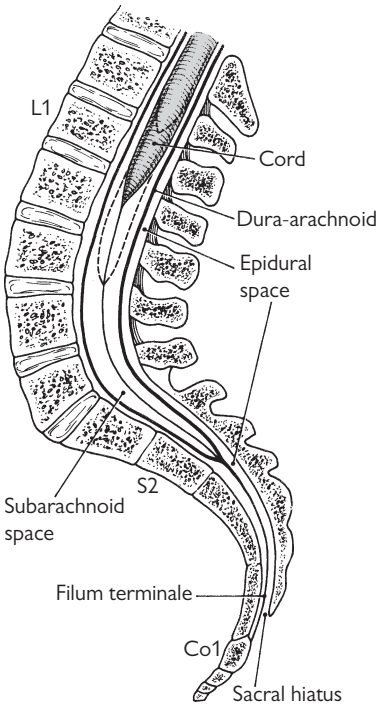

Fig. 2.26 Brain and spinal cord.

Reproduced from GA McLeod et al., Principles and Practice of Regional Anaesthesia, fourth edition, 2012, Figure 12.6, page 118, with permission from Oxford University Press.

the negative intrathoracic pressure is transmitted to the CSF through the intervertebral foraminae. CSF pressure may be positive in the lumbar regions, especially in those with chronic obstructive pulmonary disease.

Cervical plexus

- The cervical plexus (C1–C4) lies between the scalenus medius and sternocleidomastoid muscles and consists of deep motor and superficial sensory fibres.

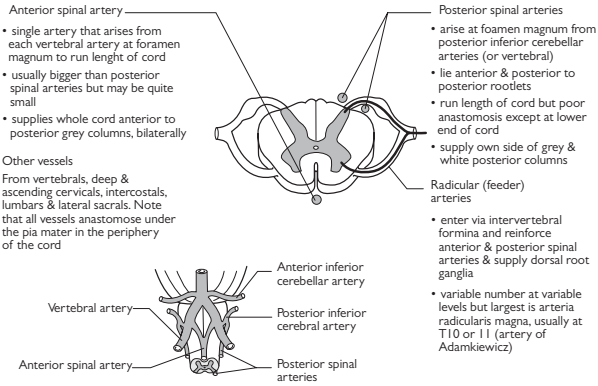

Fig. 2.27 Blood supply of the spinal cord.

Reproduced from Smith, *et al. Oxford Desk Reference of Major Trauma*, 2010, figure 11.6, p. 169, with permission from OUP.

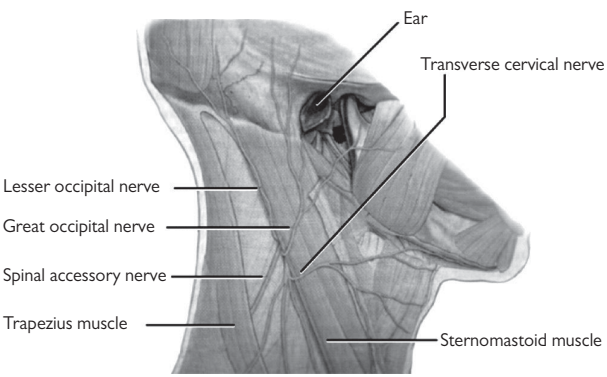

Fig. 2.28 Cervical plexus.

Reproduced from Ramachandran S K et al., Comparison of intermediate vs subcutaneous cervical plexus block for carotid endarterectomy, *British Journal of Anaesthesia*, 2011, 107, pp. 157–163, by permission of Oxford University Press and the British Journal of Anaesthesia.

- The superficial sensory fibres emerge from behind the midpoint of the posterior border of the sternocleidomastoid muscle and supply the neck and posterior parts of the head (Fig. 2.28).
- The angle of the jaw has a sensory supply from the trigeminal nerve, and the platysma muscle receives a contribution from cervical branches of the facial nerve.

The brachial plexus

- The brachial plexus is derived from the ventral primary rami of C5–T1 and consists of roots, trunks, divisions, cords and nerves (Fig. 2.29).
- The roots of the brachial plexus pass within the interscalene groove between the anterior and middle scalene muscles behind the sternocleidomastoid muscle.
- Using ultrasound, at the level of the cricoid cartilage (C6), the interscalene groove is seen angled antero-laterally towards scalenus medius and nerve roots are visualised as round and hypoechoic.
- Lateral to medial from the interscalene groove lie scalenus anterior, the internal jugular vein, carotid artery, and thyroid gland.
- In the posterior triangle, nerve roots unite to form three trunks.
- The C5 and C6 roots unite to form the superior trunk, C7 becomes the middle trunk and C8 and T1 join to form the inferior trunk.

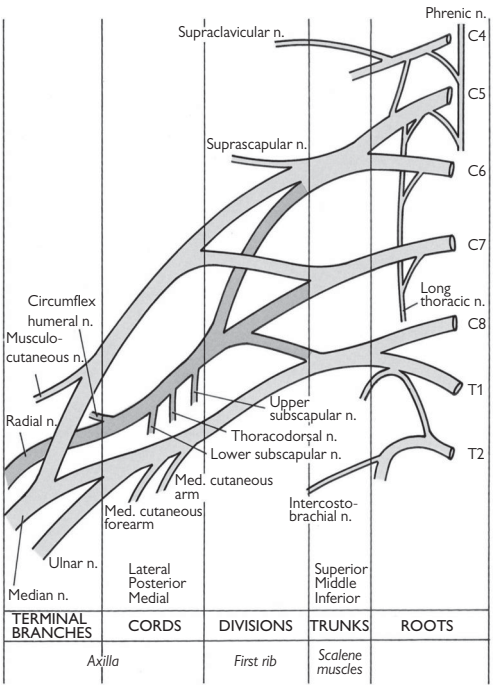

Fig. 2.29 Formation and components of the brachial plexus.

Reproduced from GA McLeod *et al.*, *Principles and Practice of Regional Anaesthesia*, fourth edition, 2012, Figure 17.1, page 171, with permission from Oxford University Press.

- On top of the first rib lie (from lateral to medial) the brachial plexus, subclavian artery, anterior scalene muscle, and subclavian vein. The subclavian artery serves as an easily identifiable reference point to locate the brachial plexus, lying behind the mid-point of the clavicle. (Fig. 2.30)The appearance of the brachial plexus on ultrasound is that of a 'bunch of grapes', superolateral to the artery.
- Below the clavicle, the anterior and posterior divisions of the three trunks form the medial, lateral, and posterior cords surrounding the axillary artery.
- The medial cord is derived from the anterior division of the lower trunk (C8–T1); the lateral cord from the anterior divisions of the upper and middle trunks (C5–C7); and the posterior cord from the posterior divisions of the upper, middle and lower trunks (C5–T1).
- With ultrasound, nerves in the infraclavicular region often appear hyperechoic. Anterior to the brachial plexus are the pectoralis major and minor muscles. The axillary vein is located caudal and medial to the axillary artery.
- The branches of the brachial plexus include the median, ulnar, radial, and musculocutaneous nerves. Using ultrasound, nerves in the axilla have a mixture of hypo-echoic nerve fasiscles and hyper-echoic connective tissue (see Table 2.4).
- Musculocutaneous nerves lies in the plane between short-head of biceps and coracobrachialis muscles. The median nerve is found at about 9 o'clock to the axillary artery and the ulnar nerve at about 3 o'clock. Radial nerve lies between 4 and 6 o'clock beneath ulnar nerve.

The lumboscral plexus

The nerve supply of the lower limb is from the lumbosacral plexus, which is formed from the anterior primary rami of the second lumbar to the third sacral roots (Fig. 2.31).

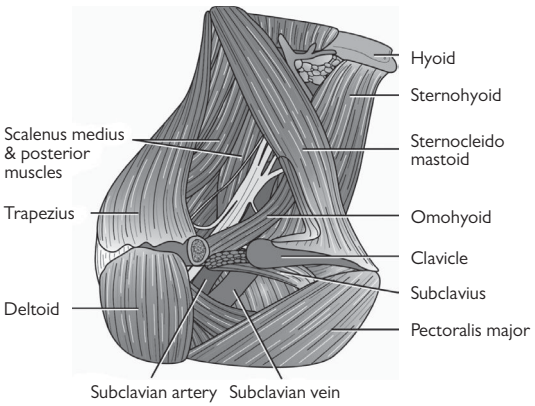

Fig. 2.30 Anatomical relationships of the brachial plexus.

Reproduced from Collier et al., Oxford Handbook of Clinical Specialties, Eighth edition, Figure 2, p. 765, with permission from Oxford University Press.

Table 2.4 Sensory and motor distribution of nerves supplying the upper limb

Nerve	Sensory distribution	Motor distribution
Radial	Lower lateral cutaneous nerve of arm Posterior cutaneous nerve of forearm Cutaneous nerves to dorsum of hand	Triceps Forearm extensors
Ulnar	Cutaneous nerves to palm and dorsum of hand	Flexor carpi ulnaris Flexor digitorum profundus (4th & 5th fingers) Lumbricals (3rd & 4th)
Median	Cutaneous nerves to palmar aspect of hand	Flexor digitorum profundus (2nd & 3rd fingers) Thenar muscles Lumbricals (1st & 2nd)
Musculo-cutaneous	Lateral cutaneous nerve of forearm	Biceps

- After emerging from the lateral foramina of the vertebral column, the nerves run within the psoas major muscle. This muscle is enclosed in a fascial sheath limited medially by the bodies of the lumbar vertebrae, and posteriorly by the lumbar transverse processes and quadratus lumborum.
- The femoral and lateral cutaneous nerves emerge from the lateral, and the obturator nerve from the medial aspects of psoas, respectively. The lumbar plexus is difficult to see using ultrasound.
- The femoral nerve(L2–L4) in the infra-inguinal region is visualized as a hyperechoic triangle lying just lateral to the pulsatile femoral artery, deep to the fascia iliaca and superficial to the iliopsoas muscle.
- The femoral vein is medial to the artery.
- The femoral nerve may be quite thin and flat in this region
- Just below inguinal ligament the nerve divides into several branches.
- Saphenous nerve, which is the terminal branch of the femoral nerve, can be blocked anywhere along the medial aspect of the lower leg
- The lateral cutaneous nerve of thigh (L2–L3) passes behind the inguinal ligament to enter the thigh deep to the fascia lata, 1–2cm medial to the anterior superior iliac spine and supplies the lateral aspect of the thigh.
- The obturator nerve (L2–L4) passes through the obturator foramen and divides into anterior and posterior branches. The obturator nerve supplies the adductor muscles and sensory fibres to the knee joint and inside of the thigh.

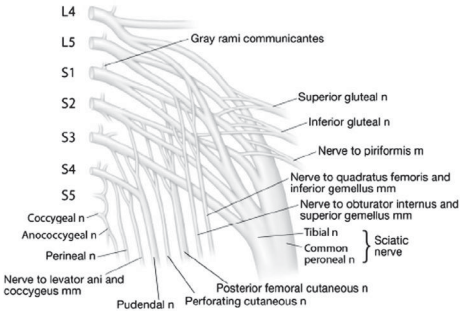

Fig 2.31 Lumbosacral plexus.

Reproduced from J Hebl and R Lennon, Mayo Clinic Atlas of Regional Anesthesia and Ultrasound-Guided Nerve Blockade, 2010, Figure 24, with permission from Oxford University Press.

- The sacral plexus is formed from L4 to S3 and passes from the pelvis into the thigh under the pirifomis muscle. It is located midway between the greater trochanter laterally and the ischial tuberosity medially.
- The sciatic nerve is bordered superolaterally by the long head of the biceps femoris muscle and superomedially by the semimembranosus and semitendinosus muscles.
- The sciatic nerve branches into the common peroneal nerve and the tibial nerve at variable location along its course in the thigh.
- Using ultrasound, the hyperechoic sciatic nerve in this location is superficial to the popliteal artery.
- The tibial nerve supplies motor fibres to the ankle flexors and sensory fibres to the heel and sole of the foot. The common peroneal nerve supplies motor fibres to the ankle extensors and sensory fibres to the upper lateral thigh and dorsum of the foot.
- The sural nerve formed from the tibial and common peroneal branches nerves supplies the lateral border of the foot.

Further reading

Whitaker R (ed.) *Instant Anatomy.* Available at: http://www.instantanatomy.net/

Wildsmith JAW, Armitage EN, McClure JH (eds). *Principles and Practice of Regional Anaesthesia*, 3rd edn. Philadelphia: Churchill Livingstone 2002.

Physiology of cerebral blood flow

The brain is a highly metabolically active organ. It has a high absolute (c. 700mL/min) and relative blood flow (*c.* 20% of cardiac output) and is poorly tolerant of acute interruption of blood flow. A complex system exists to maintain adequate, but not excessive blood flow in the face of varying systemic and cerebral demands.

Cerebral blood flow (CBF) is not homogeneous throughout the brain and is closely linked to cerebral metabolism.

Mean CBF is approximately 50mL/100g brain tissue/min.

- *Grey matter:* 70mL/100g/min.
- *White matter:* 20mL/100g/min.

It is important to understand that what matters is the balance between CBF and cerebral metabolism. Measuring CBF on its own is not a direct assessment of adequacy of nutrient delivery.

Unlike most vascular beds elsewhere, the cerebral circulation lies within a relatively rigid container—the cranium. Therefore there is a link between intracranial volume and the external pressure (intracranial pressure). Cerebral veins are collapsible structures and so blood flow through the brain is a function of not only the transvascular pressure gradient (MAP)-central venous pressure (CVP)), but also the transmural pressure gradient (MAP–ICP). The relationship is complex, but to a first approximation cerebral perfusion pressure (CPP) is often estimated as:

$$CPP = MAP - ICP, \text{ or}$$

$$CPP = MAP - CVP$$

if CVP > intracranial pressure (ICP).

CBF is affected by a variety of incompletely understood mechanisms. Of most relevance to the anaesthetist are:

- Pressure/flow regulation.
- CSF $[H^+]$ ($\approx PaCO_2$).
- CaO_2.
- Cerebral metabolic rate.

Autoregulation

Autoregulation is the ability of a vascular bed to maintain near constant blood flow over a range of perfusion pressures. This is largely achieved through active processes that alter cerebral vascular resistance (CVR). CVR is largely a function of arteriolar resistance. Several interacting mechanisms exist:

- Myogenic responses of arteriolar smooth muscles to changes in diameter associated with variations in transmural pressure gradients.
- Response to shear stress which is a function of blood velocity
- Local control in response to neuroglial metabolism, as well as autonomic input

Autoregulation is neither complete nor instantaneous. Between CPP of around 50–150mmHg CBF is kept relatively constant—the autoregulatory plateau. Beyond these upper and lower limits, CBF becomes much more pressure dependent (see Fig. 2.32). At low CPP the brain is therefore at risk of hypoperfusion. At high CPP, which occurs relatively commonly around the time of carotid endarterectomy, the brain is at risk of hyperaemia and possibly vasogenic oedema and haemorrhage. It is important to remember that these limits are average estimates and vary between individuals. Chronic hypertension 'resets' these thresholds such that the plateau is shifted towards higher CPP.

Depending slightly on the methods used to test it, autoregulation takes about 10–20s to be complete.

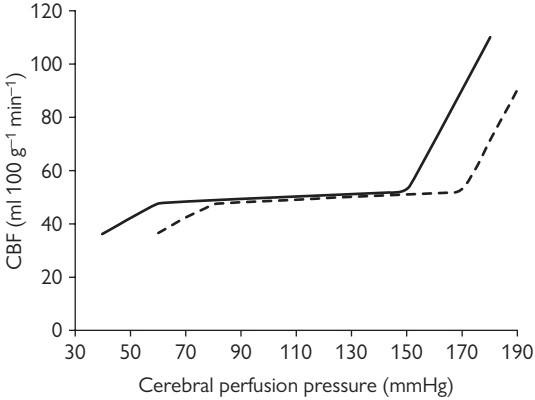

Fig. 2.32 The effect of cerebral perfusion pressure on cerebral blood flow. The solid line is the relationship in a normotensive patient, the dashed line is the relationship in a patient with chronic hypertension. (mmHg)

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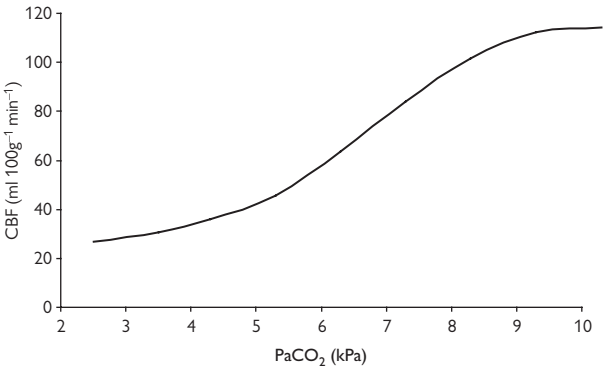

Fig. 2.33 The effect of $PaCO_2$ on cerebral blood flow.

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Biochemical responses

Arterial carbon dioxide

Cerebral arterioles vasodilate in response to reductions in CSF pH, which is closely linked to $PaCO_2$ and they vasoconstrict in response to CSF alkalosis/hypocapnia. This response is approximately linear in the region of normal $PaCO_2$, but has a ceiling/floor effect with maximal vasodilation/vasoconstriction (see Fig. 2.33).

- Hypercapnia causes ~25–35% increase in CBF per kPa.
- Hypocapnia causes ~20% decrease in CBF per kPa.
- The response is relatively prompt with a half-time of ~20s.
- The setpoint for this effect is adjusted over a matter of hours to the individual's $PaCO_2$ so resting $PaCO_2$ should be taken into account.

There is some evidence from traumatic brain injury research that even moderate hypocapnia can increase the volume of ischaemic brain tissue.

Blood oxygen content

Regulatory mechanisms allow cerebral oxygen delivery to remain relatively constant. CBF is ∴ responsive to arterial oxygen content. Hypoxia causes cerebral vasodilation. The relationship between PaO_2 and CBF is non-linear, with a large increase in CBF below approximately 8kPa. This non-linearity is largely due to the shape of the oxygen haemoglobin dissociation curve. Hyperoxia causes small degrees of vasoconstriction.

The effects of reductions in haemoglobin are slightly more complex. There is an effect on oxygen content that promotes vasodilation. In addition, reduction in [Hb] causes a decrease in blood viscosity with a resultant increase in blood velocity. This tends to cause a degree of vasoconstriction.

Flow metabolism coupling

The matching of flow to metabolism is a very rapid response, taking ~1s to complete. Under normal circumstances this coupling is very tight and changes in local CBF match changes in local glucose metabolism. Local increases in metabolism are usually matched by decreases in metabolism elsewhere, so global CBF tends to remain relatively constant. Global increases in metabolism occur with seizures and pyrexia. Flow-metabolism coupling is mediated by several mechanisms:

- Local K^+ and adenosine concentrations related to neuronal depolarization.
- Local neural innervation of feeding arterioles.

Pharmacology and cerebral blood flow

Anaesthetic agents

Nitrous oxide

- Given to awake patients increases cerebral metabolism and cerebral blood flow.
- Given to anaesthetized patients may decrease cerebral metabolism.
- Impairs autoregulation.

Volatiles

- Are all cerebral vasodilators.
- All depress cerebral metabolism.
- Maintain flow-metabolism coupling at clinically relevant doses.
- Have variable effects on autoregulation.
- Sevoflurane has no effect on autoregulation <1.5 minimum alveolar concentration (MAC).
- Carbon dioxide reactivity is relatively unaffected so vasodilation can be reversed with hypocapnia.

Propofol

- Reduces cerebral metabolism.

- Maintains flow-metabolism coupling at clinically relevant doses.
- Minimal effect on autoregulation.

Vasoactive drugs

Sympathomimetics

- Ephedrine has no clinically significant effect on CBF or regulatory mechanisms in awake, healthy subjects, but may increase CBF in patients with CPP below the lower limit of autoregulation through effects on mean arterial pressure (MAP).
- Phenylephrine and norepinephrine do not affect cerebral blood flow velocity or regulatory mechanisms. They may cause a reduction in cerebral oxygenation, possibly mediated via an indirect activation of sympathetic innervation of cerebral resistance vessels.

GTN

- Acts as a cerebral vasodilator.

Pathology

- Cerebral blood flow is reduced in dementia.
- Stroke has variable effects on CBF and autoregulation, and there is no clear consensus on the effects of anti-hypertensives in the acute post-stroke period.
- Seizures increase cerebral metabolism and cerebral blood flow markedly.

Renal

Anatomy

- Each kidney has an outer cortex and an inner medulla (Fig. 2.34)
- The kidneys receive 20% of cardiac output; renal blood flow (RBF) is 500–600mL/min to each kidney. This is calculated as renal plasma flow (RPF) adjusted for haematocrit (Hct).
- RBF = RPF/(1-Hct). RPF is usually 600mL/min.
- The cortex receives the majority (75%) of RBF.
- RBF flow is autoregulated between systolic arterial pressures of 90–180mmHg.
- Only a proportion of RPF is filtered at the glomerulus.
- Normal glomerular filtration (GFR) rate is 120mL/min.
- GFR ceases at systolic arterial pressure <60mmHg
- The filtration fraction is derived as GFR/RPF and is normally 20%.

Physiology

- The nephron is the specialized physiological unit of the kidney (Figs 2.35, 2.36).
- There are approximately 1 million nephrons in each kidney.
- 85% of nephrons are in the main cortex; the others are juxtamedullary.

A nephron is comprised of several elements.

Cortical elements

- *Glomerulus:* this is the filtering unit of the kidney. Electrolytes and free ions (but not large proteins or cells) are filtered passively into the

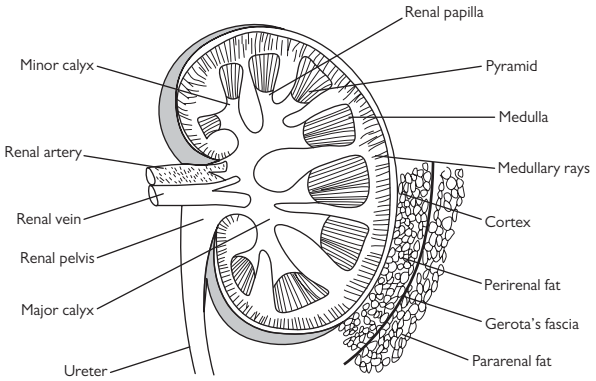

Fig. 2.34 Basic renal anatomy.

Reproduced from Reynard et al., Oxford Handbook of Urology, 2009, Figure 18.3, p. 761, with permission from Oxford University Press.

proximal convoluted tubule. Filtrate is produced at ~120mL/min and 99% of the filtrate is subsequently reabsorbed in the renal tubules.

- *Proximal convoluted tubule (PCT):* up to 75% of filtrate is reabsorbed here. Mechanisms of reabsorption are passive diffusion, facilitated diffusion (requiring interaction between ion and membrane bound carrier protein), specific pore diffusion and active transport.
- *Distal convoluted tubule (DCT):* further regulates ion concentrations (especially Na^+ reabsorption) and acid base balance.

Medullary elements

- *Loop of Henle:* comprising the descending and ascending (thick and thin) limbs, and the vasa recta. These are specialized capillaries adjacent to Loop of Henle. The loop of Henle and vasa recta have an important role in the generation of a hypertonic concentration gradient in the medulla. This is created by a countercurrent mechanism employing multiplier and exchange mechanisms. The concentration gradient is supplemented by the reabsorption of urea through the collecting ducts, under the influence of anti-diuretic hormone (ADH). Although some urea diffuses back into the filtrate, the majority remains in the medullary interstitium.
- *Collecting ducts:* these enable urine concentration by reabsorption of water under the direct control of ADH, depending on the maintenance of the medullary concentration gradient.

Renal function

The kidney maintains body homeostasis by regulating extracellular fluid. This, in turn, influences intracellular fluid volume, composition, osmolarity, and acid base status. The main processes involved are filtration/excretion of waste

products of metabolism, reabsorption (water and ions), and secretion (toxins and hormones).

- Glomerular filtration is affected by local alterations in permeability and pressure gradients across the glomerular membrane.
- Blood flow into the glomerulus is autoregulated by interaction between afferent and efferent arterioles and the macula densa—a group of cells situated in the ascending loop of Henle, but in close proximity to the arterioles.
- A reduction in arterial pressure leads to a decrease in RBF and filtration pressure, decreasing the volume of filtrate to the macula densa.
- The reduction in Na^+ and Cl^- ions is sensed at the macula densa and causes an increase in renin secretion from the juxta glomerular apparatus in the afferent arteriole. Renin is also secreted in response to increased renal sympathetic nerve stimulation, catecholamines, and prostaglandins.
- Increased renin concentration converts angiotensin (AT) I to ATII, a potent vasoconstrictor, which causes:
  - Generalized vasoconstriction.
  - Differential balanced constriction of the afferent and efferent arterioles (increasing GFR).
  - Increased aldosterone secretion by the adrenal, which stimulates Na^+ reabsorption.
  - Stimulation of thirst mechanisms at the hypothalamus.
- GFR is also regulated by the autonomic nervous system and other substances, including endothelin, prostaglandins, and nitric oxide. Increased sympathetic stimulation (in response to haemorrhage or other stimuli) causes afferent arteriolar vasoconstriction and reduces GFR

Water homeostasis

- Changes in plasma osmolality (principally Na^+ concentration) are sensed by the hypothalamic osmoreceptors.
- Increased plasma osmolality stimulates the production of ADH (vasopressin), which is released from the posterior pituitary.
- ADH acts on aquaporins to increase water reabsorption in the collecting ducts.
- Control of osmolality and ECF volume are closely linked.

Sodium homeostasis

Sodium reabsorption occurs predominantly in the PCT (65%), loop of Henle (20%), and DCT (5%). Na^+ reabsorption is determined by:

- *Aldosterone production:* aldosterone is produced by the zona glomerulosa of the adrenal cortex in response to pituitary ACTH, decreased plasma Na^+, increased plasma K^+, and ATII. Aldosterone acts via intranuclear receptors to increase Na^+/K^+ ATPase activity in the DCT, and promote Na^+ (and water) reabsorption and K^+ excretion.
- *Atrial natriuretic peptide (ANP):* ANP is secreted by atrial cells in response to stretch (water overload). ANP promotes Na^+ excretion by inhibiting renin and aldosterone release and by direct inhibition of Na^+ reabsorption in the collecting ducts. It also increases GFR.

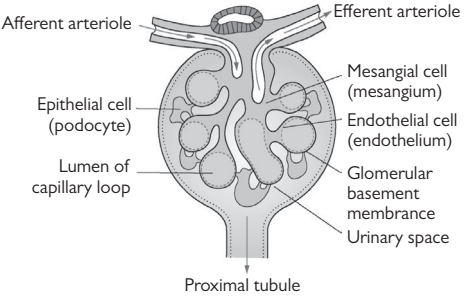

Fig. 2.35 The nephron.

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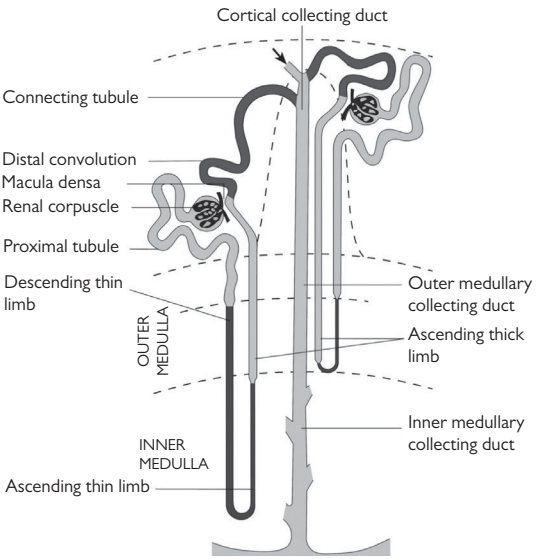

Fig. 2.36 Diagram of a short- (cortical) and long-looped (juxtamedullary) nephron to show their basic organization.

Reproduced with permission from Pocock G, Richards CD (2006). Human Physiology: The Basis of Medicine, 3rd edn, p. 349. Oxford: Oxford University Press.

Potassium homeostasis

- Potassium is freely filtered by the glomerulus and then reabsorbed by the PCT. There is no regulation at this point.
- Fine control of K^+ balance occurs in the DCT. Hypokalaemia stimulates K^+ reabsorption and H^+ excretion via the K^+/H^+ pump exchange mechanism.

Calcium and phosphate homeostasis

The kidney's main role in Ca^{2+} homeostasis is to activate vitamin D leading to reabsorption of Ca^{2+} from the gastrointestinal (GI) tract. Tubular reabsorption of Ca^{2+} is under the influence of parathyroid hormone (PTH).

Acid base balance

- Acid base balance is shared by mechanisms in the kidney and the liver.
- In the PCT, most filtered bicarbonate is reabsorbed. The DCT fine regulates acid base balance by absorption of any bicarbonate left in the filtrate.
- H^+ is secreted into the PCT in exchange for Na^+, but most H^+ is secreted in the DCT.
- When the filtrate reaches the collecting duct it is acidic, mostly bicarbonate reabsorption, rather than the excretion of acid.
- Hydrogen ions are generated from the dissociation of carbonic acid, and combined with ammonia (66%) as NH_4^+, which passes into the tubule, or with filtered phosphate ions in the tubular lumen (33%).

Eyrothropoeisis

Erythropoeitin is synthesized by the kidney in response to hypoxia, anaemia, or renal ischaemia.

Toxin removal

Toxins are removed by filtration and active secretion. This is a main mechanism for excretion of drugs.

Renal responses to vascular surgery

- The physiological stress responses to surgery involve neuroendocrine and cytokine responses.
- Both responses include effects on the kidney and are related to the amount of tissue damage.
- The neuroendocrine and sympathetic nervous system responses include increased production of ACTH, renin, ATII, and aldosterone.
- Together, these cause Na^+ and water retention, and K^+ excretion.
- In patients undergoing major vascular surgery, these may be compounded by disruption in RBF caused by hypovolaemia, aortic clamping, ischaemia-reperfusion injury, renal artery atherosclerosis, and other factors (see 🕮 Perioperative renal protection, p. 236; 🕮 Open aortic aneurysm repair, p. 350).
- Renal dysfunction may occur after vascular surgery and cause acute kidney injury (AKI).
- The time course and severity of AKI following major surgery, including vascular surgery, tends to follow three main patterns:
  - An abrupt transient decline in GFR, which promptly recovers; this is usually associated with only minor changes in serum creatinine.

  - Longer-term injury associated with sustained post-operative haemodynamic instability, often 2° to cardiac dysfunction. GFR reduces and serum creatinine increases concurrently; both recover when cardiac function improves.
  - Protracted renal dysfunction may develop as a consequence of prolonged haemodynamic compromise and is associated with multiple organ dysfunction.
- Causes of renal dysfunction that are not specific to vascular surgery, but which occur commonly due to the nature of the surgery include:
  - *Prerenal dysfunction*—episodes of hypotension and/or hypovolaemia may reduce RBF and GFR, leading to Na^+ and water retention. Effects are greater when pre-existing renal hypoperfusion exists.
  - *Non–oliguric AKI*—this is a milder form of AKI than oliguric AKI, with better maintenance of both glomerular and tubular function. The outcome of non-oliguric AKI is better than that of oliguric AKI. However, the use of high-dose diuretics in an attempt to convert oliguric AKI to non-oliguric AKI does not improve outcome.

Causes of renal dysfunction more specific to vascular procedures include:

Aortic clamping

- Infrarenal and suprarenal aortic clamping both promote alterations in post-operative renal function, which may persist in the most severe cases until 6 months after surgery.
- Infrarenal clamping creates a non-direct injury on the kidney. It is associated with a reduction in renal blood flow 2° to an increase in vascular resistance.
- Supra-renal clamping creates a more direct ischaemia-reperfusion injury associated with severe reductions in renal blood flow, GFR, and urine output.
- The mechanism of renal injury following aortic clamping is not fully understood. Blood flow redistribution within the renal microvasculature causes a preferential reduction in cortical flow and a resultant temporary reduction in GFR. In animal experiments, angiotensin inhibition (by ACE inhibitors) maintains a higher BP in the renal vasculature, implicating the renin-angiotensin system in the increase in renal vascular resistance and flow redistribution. However, other local factors including endothelin and prostaglandins may also be important.
- The autonomic system does not play an important role in reducing GFR after aortic cross clamping. The reduction in GFR does not correlate with changes in arterial BP, and cardiac or urine output. Indeed, reduction in GFR may be related to an adaptive response to renal hypoperfusion, thereby reducing the aerobic requirement of the kidney.

Myoglobin-induced renal dysfunction

- Myoglobin is an oxygen and iron-binding protein released from muscle after muscle injury or ischaemia.
- Myoglobin is normally loosely bound to $\alpha-2$ globulin and is filtered and reabsorbed by the PCT.

- Excess circulating myoglobin may be converted to ferrihaemate in the kidney in acidic urine. Ferrihaemate is a produces toxic swelling of glomerular and epithelial cells. This can cause tubular obstruction leading to further renal injury.
- Cortical vasoconstriction, secondary to hormonal release is also an important component of this form of injury.
- The mainstay of treatment is to maintain extracellular fluid volumes, and promote a diuresis with mannitol. Forced alkaline diuresis to maintain urine pH>6 using bicarbonate and furosemide may be helpful.

Contrast-induced nephropathy

See 🕮 Contrast-induced nephropathy and renal protection, p. 338.

An increasing number of vascular surgical and procedures are either preceded by, or include the administration of large doses of contrast media for imaging purposes.

The mechanisms involved in CIN are:

- Direct renal tubular toxicity.
- Tubular obstruction—by agglutination and aggregation of crenated red cells.
- Intra-renal vasoconstriction.

Risk factors for development of CIN are synergistic and include:

- Pre-existing renal insufficiency.
- Low cardiac output states.
- *Comorbidities:* diabetes, congestive heart failure, myeloma, hepatic disease.

Prevention of CIN is discussed in 🕮 Contrast-induced nephropathy and renal protection, p. 338.

Coagulation and the response to major haemorrhage

Introduction

- Blood loss during elective vascular surgery is usually minimized by the surgeon. By using controlled and careful dissection techniques most procedures can be performed with minimal or no blood component therapy. When emergency or unexpected bleeding occurs the challenges are similar to those presented by traumatic haemorrhage.
- Patients with abdominal trauma or suspected rupture of an abdominal aortic aneurysm (AAA) require urgent surgical intervention. The urgency with which this is undertaken will depend on the presenting features.
- A FAST scan (focused assessment with sonography in trauma) can be undertaken very quickly in the emergency department and only when the diagnosis is unclear is a computed tomogram (CT) required.
- FAST scanning allows rapid but limited assessment predicting the presence of free abdominal or pericardial fluid, which in the trauma patient usually indicates haemorrhage.

- Such scanning enables aneurysmal sections of the abdominal aorta to be identified by the experienced user.
- In cases of leaking or ruptured AAA, the priority is urgent surgical control of bleeding. Prolonged resuscitation with i.v. fluids is not indicated as it adds to surgical difficulty and causes dilutional coagulopathy. A technique of permissive hypotension (📖 Emergency abdominal aortic aneurysm repair: open and endovascular repair, p. 448) is indicated.

Principles of coagulation

- In health, the vascular endothelium provides a smooth surface for blood flow. Vascular endothelial cells express proteins and secrete substances that prevent clot formation, and stop platelet and coagulation proteins from coming into contact with the sub-endothelial proteins that stimulate haemostatic function.
- However, the whole haemostatic system allows for a rapid response to vessel injury. Clots form quickly to stem bleeding. This is followed by the process of fibrinolysis, which allows breakdown of the obstructive lesion during vessel wall healing and repair.
- Blood rheology in health ensures that erythrocytes tend to displace platelets to the periphery of the blood vessel. Margination of platelets occurs because red cell flow is maximal at the centre of the vessel; platelets are pushed to periphery where they are present in sufficient numbers to promote haemostasis rapidly in response to vessel injury.
- The concentration of platelets near the vessel wall is up to seven times the average concentration within the vessel. This explains why many patients can manage to initiate clot formation with abnormally low platelets concentrations, providing that haematocrit and blood volume are maintained.

Response to blood loss

- The natural physiological response to major haemorrhage is vasoconstriction in vessels with a muscular component to their walls, to minimize blood flow and further blood loss. The aim is to conserve blood volume and maintain BP to maintain perfusion of the vital organs.
- If bleeding becomes more severe, with a decrease in circulating volume and systemic hypotension, widespread peripheral vasoconstriction and tachycardia occur, mediated by the sympathetic nervous system.
- This response can vary. It may be a late sign in young fit adults. Unless traumatic injury is the underlying problem most vascular surgical patients are elderly and have other co-morbidities including diabetes and widespread arteriosclerosis. Therefore cardiovascular reserve and the ability to tolerate hypotension is usually decreased in the vascular surgical patients.
- A second natural response to bleeding is for the blood to become hypercoagulable.
- The extent of compensation by these mechanisms depends on the on the cause and amount of haemorrhage. In situations of large blood vessel disruption or major organ trauma, some form of surgical intervention will be required.

- Unfortunately the administration of anaesthesia can often diminish or even abolish these natural responses, as during anaesthesia may cause vasodilatation, and worsen hypotension and hypoperfusion.

Coagulopathy of sudden blood loss

- Many patients with severe acute trauma or haemorrhage are already coagulopathic on arrival in hospital. This may be worsened by interventions to correct blood volume before surgical control of bleeding.
- The initial management of major haemorrhage usually involves the infusion of IV fluids, usually crystalloids (as these are readily available) or a colloid solution. The administration of either crystalloids or colloids dilutes the remaining coagulation factors fibrinogen and platelets, and is detrimental to coagulation processes. Dilution of coagulation factors and platelets may not be clinically relevant at the time, because a clot will form usually at a normal rate. However, if dilution is excessive, the clot strength may be inadequate and further bleeding may occur on restoration of normal BP.
- Restoration of a normal arterial BP also disrupts the compensatory response to haemorrhage and may cause disruption of clots at an early stage of consolidation.
- This can also occur during elective surgery when the over-use of crystalloid and colloid can dilute clotting factor concentrations before the onset of haemorrhage.

Adequacy of coagulation

- Clots form at very low concentrations of coagulation factors, but clot firmness may not be sufficient for sustained haemostasis.
- Clot formation is initiated in response to tissue factor present on cells exposed by endothelial injury. Platelets adhere to tissue factor and become activated; this stimulates an amplification phase that greatly increases the production large amounts of thrombin, termed the 'thrombin burst'. (Fig. 2.37)
- When sufficient thrombin is present, it converts fibrinogen to fibrin. This helps strengthen the forming clot which forms a secure plug to stem the bleeding.
- The presence of tissue factor, factor VIIa (endogenous), calcium, active platelets and sufficient fibrinogen are vital to form a reliable clot.
- When deficiency of any of these components results in the formation of a weak clot, then excessive BP will displace the forming clot Constant clot removal by frequent swabbing or suction on the bleeding area will produce the same effect. A prolonged period of applied pressure may be required until the clot is established. If heparin has been administered during vascular surgery, this period may be prolonged significantly, even if platelet activity is normal.

Alteration by anaesthesia

Anaesthesia may affect coagulation in several ways:

- Alteration of body temperature.
- Decreased peripheral vascular resistance with volume loading.
- Respiratory depression and hypercarbia.
- Acidaemia.

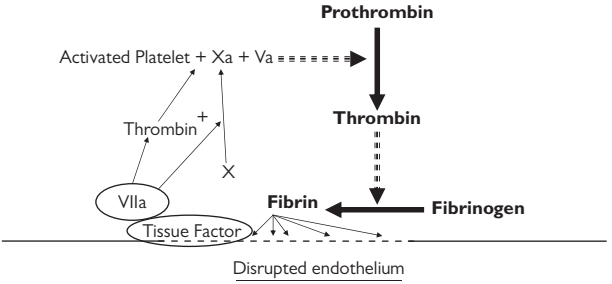

Fig. 2.37 A simplified summary of the essential tissue factor basis of cell-based coagulation. Initial activation of factor VII to VIIa creates a small amount of thrombin to activate adjacent platelets. Subsequent amplification increases thrombin concentration and stimulates the common coagulation pathway.

Alteration of body temperature

- Hypothermia impairs platelet function and coagulation factor activity, and has been shown to increase blood loss during surgery.
- Under normal conditions humans are poikilothermic and adapt to the environmental temperature. Homeostatic temperature control mechanisms are obtunded under general anaesthesia and patients easily become hypothermic. It is accepted that normothermia should be maintained during surgery using all available means

Decreased vascular resistance

- Both general and regional anaesthesia tend to decrease systemic vascular resistance and cause hypotension. This is often corrected in practice by the infusion of crystalloid or colloid solutions, which dilute clotting factors, fibrinogen, and platelets.
- Dilutional effects can also occur when infusing red cell concentrates or red cells salvaged via an autotransfusion device. Neither red cell concentrates nor salvaged red cells have many clotting factors or platelets and whilst they both restore red cells and correct haemoglobin level and haematocrit, the need for platelet and fibrinogen replacement needs to be kept in mind.

Respiratory depression and acidaemia

Maintenance of normocapnoea is important (regardless of anaesthetic technique used) as increased pCO_2 causes a reduction in pH; this decreases endogenous factor VIIa levels and impairs coagulation. The effect of increased CO_2 is minimal compared with the effects of metabolic acidaemia caused by poor perfusion and the development of tissue hypoxia.

Targets during resuscitation of the bleeding patient

- Normovolaemia.
- Normothermia.

- Normocapnoea.
- Normal pH.

Coagulation tests

Activated partial thromboplastin time (APTT)

- This test is a measure of the intrinsic coagulation pathway (Fig. 2.38).
- It is performed by adding Kaolin, a surface activator, phospholipids, and calcium ions to plasma.
- The normal range is 30–40s.
- APTT is prolonged by the presence of heparinoids, fibrin degradation products (FDP's) and in patients with a lupus anticoagulant.
- Prolonged APTT may indicate deficiencies in factors II, V, VIII, IX, X, XI, and XII.

Prothrombin time (PT)

- PT is a measure of extrinsic and common coagulation pathways, evaluating adequacy of factor VII and factors common to both pathways.
- PT is performed by adding thromboplastin and calcium ions to plasma.
- The normal range is 10–14s.
- PT is prolonged in deficiencies of II,V, VII, and X

Thrombin time (TT)

- TT detects a deficiency in fibrinogen or a dysfibrinogenic state.
- TT is measured by thrombin to plasma and observing time to fibrin formation.
- TT can be prolonged in the presence of heparin or FDP's.

Fibrinogen level

- Fibrinogen concentrations in health are usually >1g/dL.
- Levels should be maintained at or above this level during haemorrhage.
- In severe haemorrhage some advocate keeping fibrinogen >1.5 or 2g/dL.

Platelet count

- Maintain platelet count $>50 \times 10^9$/L when there is active bleeding
- If there is bleeding and traumatic brain injury platelet count should be $>100 \times 10^9$/L.

Point of care testing devices

- The tests listed above were designed to measure certain parts of the coagulation cascade, in particular the effect of certain haemostatic antagonists. They are all performed by laboratory analysis and even in the most efficient institutions there can be at least 45min delay in obtaining the results from initial sample collection.
- This does not allow dynamic and responsive management of coagulopathy problems. ∴ Point of care testing or near patient testing of coagulation in the clinical environment is gaining in popularity, to ensure timely and efficient use of blood and blood component therapy, and minimize patient morbidity and mortality.
- Two main thromboelastogram devices are available for the near patient testing of haemostasis—the TEG® and the ROTEM®.

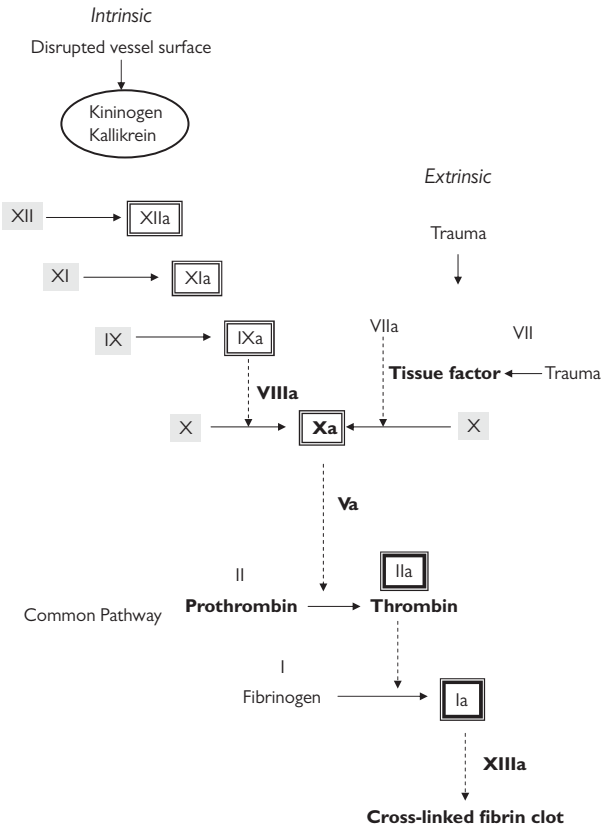

Fig. 2.38 The classical coagulation pathway.

- Both operate with the principle that the measurement of clot formation, structure, and lysis depends on the viscoelastic properties of whole blood as it clots in a small cuvette, which creates resistance to a measuring pin.
- In the TEG® device, the cuvette rotates around a fixed measuring pin. With the ROTEM®, the cuvette is fixed and the pin rotates.
- The claimed advantages using either form of thromboelastography is a rapid result on which to base further resuscitation.
- The *r* time or clotting time can be obtained in 5–7min if normal and then the strength of the clot can help determine the need for platelet or fibrinogen therapy.

- Early clot lysis can also be diagnosed. This indicates excessive fibrinolytic activity and suggests the need for treatment with tranexamic acid.
- Point of care testing devices are discussed further in 🕮 Monitoring the circulation.

Further reading

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Physiology of pain

Introduction

- *Pain is common after major vascular surgery:* approximately 1 in 5 patients experience moderate to severe pain on movement after abdominal surgery.
- *Pain-induced sympathetic stimulation has profound cardiovascular effects:* these are potentially harmful, especially in vascular patients in whom there is a high incidence of CAD. Increases in heart rate, BP and myocardial contractility stimulate oxygen demand, whereas coronary vasoconstriction and hypercoagulability reduce oxygen supply. These can cause myocardial ischaemia.
- *Many vascular patients have long standing hypertension:* they also may exhibit an exaggerated hypertensive response to pain. Hypertension after vascular surgery is associated with myocardial ischaemia, bleeding, and stroke.
- *Effective pain therapy in the vascular patient* has additional benefits by attenuating the sympathetic nervous responses to surgery, including improved myocardial oxygen supply, reduced myocardial oxygen demand, and attenuation of fluctuations in arterial pressure.

Mechanisms of pain transduction

- Pain is a sensory and emotional experience influenced by psychological and environmental factors, but correlates poorly with the extent of tissue damage.
- Surgical tissue damage caused by mechanical trauma, heat, and local release of inflammatory mediators sensitizes the peripheral nociceptors of myelinated A-δ and unmyelinated C-pain fibres. The hypersensitivity of skin overlying a surgical wound is termed *peripheral sensitization* and clinically discernible as 1° hyperalgesia.
- Impulses pass via A-δ & C fibres to the dorsal horn of the spinal cord. (Fig. 2.39).
- Pain signals carried by A-δ & C fibres terminate within laminae I and II of the dorsal horn, whereas light touch is transmitted to laminae III and IV A-beta fibres
- The relay of nocicieptive information via dorsal root ganglia (DRG), the cell bodies of nociceptive afferents, to the central nervous system is termed *transduction*.

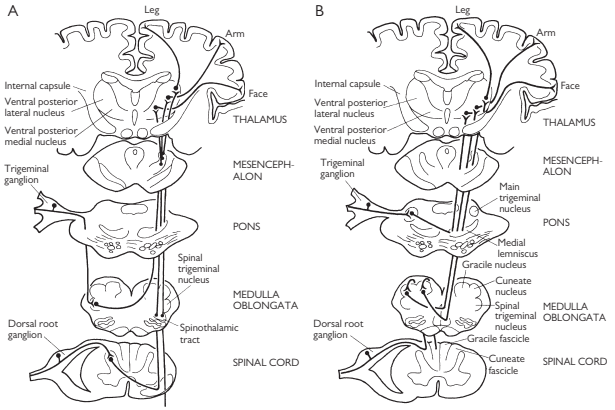

Fig. 2.39 Spinal cord sensory pathways. (A) The spinothalamic tract. This is the main pathway for transmission of signals from nociceptors and thermoreceptors. (B) The dorsal column-medial lemniscus pathway. This is the main pathway for transmission of signals from low-threshold mechanoreceptors. Fibres transmitting impulses from mechanoreceptors in the face join the medial lemniscus in the brainstem.

Reproduced from Donaghy, *Brain's Diseases of the Nervous System*, 12th edition, 2009, figure 2.5.1, p. 54, with permission from Oxford University Press

Pain transmission in the spinal cord

- *1° afferents* in the dorsal horn release neurotransmitters in response to pain signals. These include excitatory amino acids, such as glutamate, and peptides, such as substance P and calcitonin gene-related peptide (CGRP). This *central sensitization* manifests as increased sensitivity to both painful stimuli and non-painful stimuli ('*allodynia*').
- *2° hyperalgesia* describes hypersensitivity of uninjured skin to mechanical stimulation (*mechanoallodynia* or *punctate hyperalgesia*) adjacent to a surgical wound and reflects central sensitization of dorsal horn neurons.
- Sensitization within the central nervous system (CNS) stimulates excitatory pathways and depresses inhibitory pathways, amplifying responses to noxious stimuli, and altering gene expression in the spinal cord and DRG.

Central pain pathways

- Pain signals arise from the dorsal horn via two principal tracts:
  - The spinothalamic pathway, arising from laminae I and II. Passes via the thalamus to the somatosensory cortex.
  - The spinoreticular pathway, arising from lamina I neurons and projects to the ventromedial hypothalamus (Fig. 2.40).
- Structural reorganization in the somatosensory cortex is associated with the development of persistent pain.

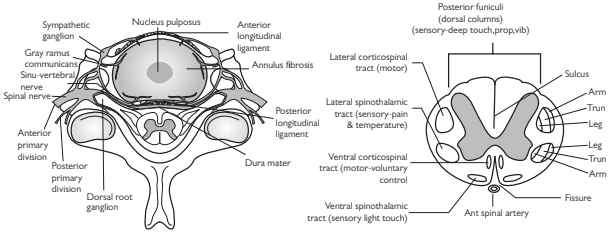

Fig. 2.40 The gross and microscopic structure of the spinal cord.

Reproduced from Smith *et al*., *Oxford Desk Reference: Major Trauma*, 2010, figure 11.6, p. 169, with permission from Oxford University Press.

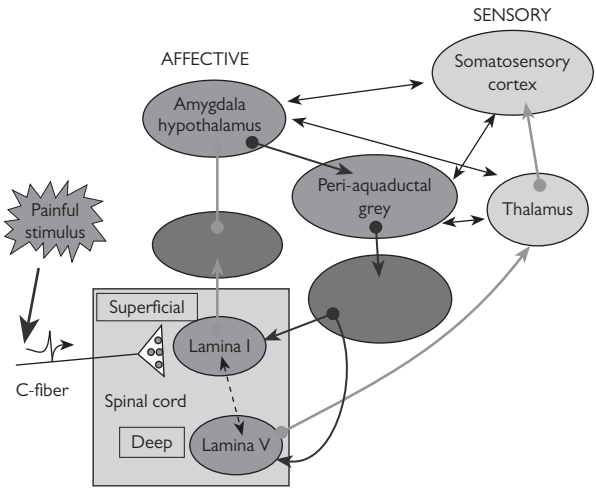

Fig. 2.41 Summary of some of the interactions between the peripheral neurons, dorsal horn, and higher centres in the brain.

Reproduced from Bennett, Oxford Pain Management Library: Neuropathic Pain, 2010, Figure 2.3, p. 15 with permission from Oxford University Press.

- Descending pathways contribute to the modulation of pain transmission in the spinal cord via presynaptic effects on 1° afferent fibres (Fig. 2.41).

Chronic pain after surgery

- Chronic pain is common after surgery and has a neuropathic element.
- The clinical pain states most likely to be encountered after vascular surgery are phantom limb pain, chronic pain, and opioid-induced hyperalgesia.

- Psychological factors are important. Preoperative anxiety, catastrophising, neuroticism, and depression are associated with higher post-operative pain intensity.
- Risk factors for chronic post-surgical pain include moderate to severe acute post-operative pain, poor diffuse noxious inhibitory control (DNIC), repeated surgery and nerve damage during surgery.
- Descending pathways of pain control play a crucial role in preventing post-surgical pain

Phantom limb pain

- Nerve injury induces structural and functional changes at several points in nociceptive pathways. These are associated with plastic changes in the 1° sensory and motor cortex. The size of change correlates with severity of phantom limb pain (PLP).
- Reorganizational changes occur in the thalamus and dorsal horn of the spinal cord.
- Peripheral nerve changes include the release of local pro-inflammatory cytokines and neuroma formation.
- Phantom limb pain should be differentiated from phantom limb sensation, which is related to posterior parietal and 2° somatosensory cortex changes (see 🕮 Pain Management, p. 303)
- Once established, PLP is difficult to treat (see 🕮 Pain Management, p. 303). Behavioural and pharmacological interventions should be used. Sensory discrimination training and motor imagery reduce chronic PLP.

Opioid -induced hyperalgesia

- Opioid-induced hyperalgesia (OIH) refers to increased pain sensitivity at baseline in response to a noxious stimulus, after the administration of opioids. In contrast, tolerance describes an increasing dose of opioid to get the same degree of pain relief.
- The mechanism is thought to be that high doses of opioids, or sudden changes in opioid concentrations, may sensitize the CNS. When the CNS is sensitized, pain is increased, despite analgesic rescue medication, and the size of mechanoallodynia on normal skin is increased.
- Both central and peripheral changes are involved in OIH. In the dorsal horn, the N-methyl-D-aspartate (NMDA) receptor is implicated
- Reduction in descending inhibitory control acts via 5-HT and cholecystokinin receptors.
- Peripheral changes include transient receptor potential (TRP)-V1 stimulation.
- Changes in endogenous opioid receptors (desensitization or internalization) may also be involved.

Further reading

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Wilder-Smith OHG, Arendt-Nielsen L. Postoperative hyperalgesia: its clinical importance and relevance. *Anesthesiology* 2006; **104**: 601–7.

Chapter 3

Evaluation of the vascular surgical patient

Preoperative evaluation

Preoperative evaluation is an important part of vascular surgical care. The anaesthetic components of preoperative assessment for elective surgery are best achieved within a dedicated, resourced preoperative assessment clinic with established pathways for investigation and referral, and a multidisciplinary approach to decision making.

Aims of preoperative evaluation

- To allow assessment of the potential risks and benefits of possible surgery, using local hospital audit data for morbidity and mortality.
- To facilitate specialized investigations and referral where necessary.
- To improve a patient's fitness for surgery by lifestyle changes and medical interventions when appropriate.
- To allow time for detailed discussion of the available options.
- To plan for surgery, post-operative care facilities and hospital discharge.
- To consider the 2° prevention of cardiovascular disease.

Practical aspects

- Patients should be assessed at least 2 weeks before elective surgery.
- The following have been recommended before AAA repair:
  - Patients should undergo standard preoperative assessment and risk scoring, including cardiac, respiratory, renal, diabetes.
  - Each hospital should have defined pathways for the correction of significant medical risks before intervention.
  - All patients should be seen by an experienced vascular anaesthetist.
  - Medication should be reviewed and optimized.
- Psychological and social aspects must be considered, especially before life-changing procedures, such as amputation.

Outcomes of preoperative evaluation

- Deferral for further investigations or other specialist opinion.
- The recommendation to proceed to surgery at a variable interval.
- To recommend a less-invasive surgical procedure.
- Advice against possible surgery where the risks outweigh the potential benefits (e.g. limited life expectancy because of co-existing disease).
- *Deferral for review:* the relative risks of surgery or some co-existing medical conditions (e.g. recent MI or coronary intervention) change over time. Deferral for review (e.g. to re-assess AAA size over several months and reconsider surgery if it increases, or to allow an appropriate interval after percutaneous coronary intervention (PCI)) can be the most appropriate course of action.

Multidisciplinary teams (MDTs)

- There must be an effective mechanism for the discussion of individual cases. As a minimum the consultant surgeon, anaesthetist and, if appropriate the radiologist, should discuss complex and high risk cases before the day of surgery; this discussion should be recorded in the patient's notes.

- The MDT provides a forum in which all aspects of individual patient care can be discussed. Procedures are planned in the context of clinical status and imaging results, and decision should take into account individually assessed perioperative risk.
- An anaesthetist should attend the vascular MDT meeting, where possible, in order to inform this discussion.
- Anaesthetists' attendance at vascular MDTs should be included in the job planning of several individuals, to allow a group of anaesthetists to develop expertise in this area and ensure adequate cover for leave.
- An MDT database and administrative support are required so that all parties know which patients are to be discussed and can collate the necessary information in advance.
- Details of the patient's clinical history should be available at the MDT meeting, ideally including the notes from a pre-assessment clinic assessment by a consultant anaesthetist and the results of baseline investigations.
- Pathways should be established to refer patients for additional investigations or specialist medical opinion with minimal delays. This may include regular review of patient's records or database entry at each MDT meeting, whilst the results of referral are awaited.
- Whatever the arrangements for the discussion of patients facing vascular surgery, a record should be kept of the matters considered and the conclusions reached. This may be in the form of minutes of the MDT meeting or a note in the medical records.

Potential benefits of good preoperative preparation

- Reduction of late cancellations because of co-existing disease or administrative errors.
- Standardization of preoperative testing and perioperative care.
- Improved patient satisfaction.
- Reduction of post-operative complications.

Further reading

Association of Anaesthetists of Great Britain and Ireland. *Preoperative assessment and patient preparation*, AAGBI Safety guideline. London: AAGBI 2010 Available at: http://www.aagbi.org/publications/guidelines/docs/preop2010.pdf

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Indications for vascular surgery

Aneurysmal disease

Infrarenal abdominal aortic aneurysm

- Infrarenal aneurysms account for 95% of all AAA. The remaining 5% extend superiorly to involve renal and possibly superior mesenteric vessels.
- The risk of aneurysm rupture increases as diameter increases; the aim of surgery is to prevent death from aneurysm rupture.
- For aneurysms less than 5cm in diameter, the risk of rupture is less than 1% per year, but increases markedly as diameter increases.

- When considering treatment, the risk of rupture must be balanced against the risk of intervention for the individual patient.
- For conventional open repair of infrarenal AAA, the UK Small Aneurysm Trial showed there was no overall benefit in early surgical repair of AAA with diameters 4–5.5cm. On the basis of this and other studies, patients with abdominal aneurysms 5.5 cm diameter should be assessed with a view to possible repair.
- The decision to intervene is based on a balanced assessment of risks. Patients deemed at higher operative risk (advanced age, associated co-morbidities) may be observed in surveillance programmes until AAA size increases to a diameter >5.5cm. In some patients at particularly high risk, observation may be appropriate at greater AAA diameters.

Open or endovascular aneurysm repair (EVAR)

- In most centres, surgical repair of AAA may be performed by 'conventional' open repair or by excluding the aneurysm with an endovascular stent graft (EVAR). Some specialist centres also offer a conventional repair performed laparoscopically.
- Infrarenal AAA morphology (neck length, shape, and angulation) precludes EVAR in a declining proportion of patients as stent graft design and operator experience improve.
- Conventional repair inflicts a major physiological insult on the patient and randomized controlled trials have shown an early survival advantage for EVAR. Thirty-day mortality after EVAR is approximately one-third that of conventional repairs (1.8% compared with 5.1% in the EVAR 1 study).
- However, open repair is a 'definitive' procedure with little or no need for further intervention. Hence, continued intense surveillance is not required after open repair.
- In contrast, long-term surveillance (combination of regular CT, ultrasound, and plain radiography) is required after EVAR to ensure graft function.
- Further interventions are required in ~20% of patients after EVAR. These are usually aimed at controlling endoleaks (persistent blood flow between the aneurysm sac and stent graft) and can often be performed percutaneously, although operative femoral artery exposure may be required.
- Although relatively minor, these interventions do result in morbidity and occasional mortality; this may counteract the early survival benefit.

EVAR for high risk patients

- The physiological consequences of EVAR are much less than conventional open surgery. Hence, EVAR was originally considered an ideal treatment for patients considered unfit for conventional surgery.
- The EVAR 2 trial compared stent grafting versus observation in patients deemed unfit for open repair. It found that the 30-day mortality rate for the stent graft arm was very high (9%) and many patients in the control group died of non-aneurysm-related causes. The trial concluded that there was no benefit to EVAR in this group of patients.

- However, in clinical practice, there are a significant number of patients who might be considered at higher-than-average risk from open surgery. Examples include previous abdominal surgery or stoma, or cardiorespiratory disease not severe enough to preclude open surgery. EVAR is often recommended for this group of patients if the AAA morphology is suitable.
- Each patient should be assessed individually to determine the relative risks of rupture and the risks of possible interventions. It is good practice to discuss all options with patients and help them decide which is best for them.

Rupture of AAA

- Free rupture of an AAA into the peritoneal cavity results in rapid exsanguination. This occurs in about 50% of AAA ruptures.
- Rupture into the retroperitoneum results in a large haematoma where tamponade and hypotension may slow or temporarily arrest blood loss enough for the patient to reach hospital. Death is usually inevitable without intervention, but emergency open repair is associated with a mortality rate of 40%.
- The decision not to operate is difficult. In general, end-of-life care should be considered in those with negligible chance of survival due to advanced age and associated co-morbidities.
- Stent graft repair of ruptured AAA is possible and case series suggest a lower mortality than open repair. However, this may reflect a selection bias as perhaps the more stable patients with more favourable anatomy are selected for emergency EVAR. An ongoing randomized controlled trial (RCT) within the UK aims to clarify the role of emergency EVAR.

Carotid disease

Symptomatic atherosclerotic carotid disease

- Carotid atherosclerosis usually causes symptoms through intracerebral embolization of atherothrombotic material resulting in a TIA or stroke.
- Only rarely are symptoms the result of reduced cerebral perfusion due to carotid artery stenoses.
- It is very difficult to predict which carotid plaque will cause an initial or further event.
- In current clinical practice, 'severity' of disease is judged by the degree of carotid artery stenosis, usually measured by ultrasound scanning.
- An internal carotid artery stenosis of 70–99% is most likely to cause new or recurrent embolic symptoms.
- The risk of a TIA or stroke in a patient with asymptomatic carotid disease is low (about 2% per year). However, once a TIA has occurred, the risk of subsequent stroke increases dramatically and is maximal within the first few days of the event. Clinical scoring systems, e.g. ABCD2 score for TIA (📖 Evaluation of the vascular surgical patient); transient ischaemic attack are used to assess the degree of risk.

Carotid endarterectomy

- The aim of CEA is to remove the unstable atheromatous plaque and leave a relatively smooth surface of arterial media.
- Large randomized clinical trials (ECST, NASCET and Veterans Affairs trials) including 35 000 patient years of follow-up, confirm that in patients after TIA or minor (non-disabling) stroke who have significant internal carotid stenosis, CEA combined with appropriate medical therapy reduces the risk of stroke.
- The benefit is marginal for patients with 50–69% stenosis and highly beneficial in those with 70–99% stenosis.
- Patients within these early trials were considered to be symptomatic if they had suffered an event in the preceding 6 months. Re-analysis of this data revealed that the benefit from endarterectomy was greatest when performed within 2 weeks of the index event for patients with both 70–99% and 50–69% stenosis.
- Delay in CEA after TIA will result in a number of patients suffering potentially preventable strokes. Realization of this fact has changed practice, and TIA and minor stroke or 'brain attacks' are now considered to be emergency conditions akin to a 'heart attack' that require urgent assessment and treatment:
  - UK Department of Health National Stroke Strategy recommends that carotid intervention should occur within 48h of index event.
  - National Institute for Health and Clinical Excellence recommends intervention within 2 weeks.
- When CEA endarterectomy is performed in acute cases where there is likely to be unstable atherosclerotic plaques, risk of stroke complicating procedure is likely to be slightly greater than for 'delayed' surgery. However, this is significantly outweighed by the considerably greater number of strokes prevented from early (within 2 weeks) intervention.

Asymptomatic atherosclerotic carotid disease

- The annual risk of a TIA or stroke in a patient with significant, but asymptomatic carotid disease on medical therapy is approximately 2%.
- Randomized control trials show that the risk can be approximately halved if CEA is performed in patients with severe carotid stenosis.
- The benefits of surgery are much less in women.
- Recent European guidelines state that:
  - CEA can be recommended for asymptomatic men with a stenosis of 70–99%, as long as the operative risk is <3%.
  - CEA in women should only be considered in younger and fitter women expected to live long enough to gain any benefit of surgery.

Carotid artery stenting

- The indications for the stenting of internal carotid artery stenoses continue to be an area of considerable controversy.
- The aim of treatment is to insert a metal mesh stent percutaneously, usually via the femoral artery, across the stenosis, and so prevent recurrent or future embolization.
- Stenting less invasive than CEA, but requires passage of wires and catheters through carotid stenosis. This may be a particular problem

when treating recently symptomatic disease when manipulations across the plaque would seem most likely to trigger further embolization.
- To counter this, equipment manufacturers have designed a variety of net-like cerebral protection devices to capture any dislodged atherothrombotic material.
- Recent studies comparing carotid endarterectomy and stenting of symptomatic internal carotid stenoses have found a greater risk of periprocedural stroke (and/or death) following stent treatment.
- The 2009 European Society of Vascular Surgery Guidelines on carotid intervention recommend surgery as the best option for symptomatic patients.

Miscellaneous carotid disease

Extracranial carotid artery aneurysms
- Extracranial carotid aneurysms are rare.
- They usually present through embolization of thrombus that forms within them, or as a lump in the neck.
- Their scarcity limits published evidence to case series, but the principal aim of treatment is the prevention of (further) neurological events.
- Intervention may involve surgical resection and reconstruction (direct anastomosis, interposition vein, or synthetic graft) or aneurysmorrhaphy (resection of redundant aneurysm wall).
- More recently, the interventional radiological techniques of stent graft insertion or embolization have been successful.

Carotid artery dissection
- Carotid dissection is rare.
- It causes symptoms through thromboembolism to the cerebral circulation (stroke or TIA).
- The principal treatment is systemic anticoagulation.
- Surgical intervention is extremely challenging due to the poor quality of the arterial tissues in these patients; surgery may be indicated for arterial rupture or pseudo-aneurysm formation.

Lower limb arterial disease
- The underlying pathological process in the vast majority of patients with lower limb ischaemia is atherosclerosis.
- Lower limb atherosclerosis results in a spectrum of disease depending on the extent of arterial stenosis or occlusion and the functional demands of the patient.
- Symptoms range through intermittent claudication to rest pain and tissue loss (ulceration and/or gangrene), although many patients are asymptomatic.
- If atheroma is present in the arteries in the legs it is also likely to be found in the coronary and cerebral circulation.

Intermittent claudication
- Intermittent claudication (IC) is exercise-induced pain that is rapidly relieved with rest.
- It is commonly felt within muscle groups (calf, thigh, buttock).
- Usually described as aching or cramp-like in nature.

- Prevalence is approximately 2% of people in their 6th decade, increasing to 7% in those in their 8th.
- Contrary to the expectations of many patients (who fear impending gangrene when told that the symptoms are caused by arterial disease), the prognosis for the limb is favourable.
- In the 5yrs after diagnosis:
  - 70–80% of patients will experience the same or an improvement in symptoms, through collateral development and changes in behaviour.
  - 10–20% of patients will experience worsening of symptoms.
  - 5–10% will develop limb-threatening critical leg ischaemia.
- IC is a marker for the presence of generalized atherosclerosis and this is reflected by the high cardiovascular morbidity and mortality suffered by these patients. Over the same 5-yr period after diagnosis of IC:
  - 20% will suffer a nonfatal myocardial infarction or stroke.
  - 10–15% will die (cardiovascular death in 75%; cancer in 25%).
- This highlights that patients with IC should receive optimal 2° prevention interventions, including life style modification, statins, and antiplatelet therapy.
- The indication for invasive intervention in patients with IC depends principally on the impact of symptoms on their quality of life, the pattern of their disease and the therapeutic options available. For example, there would usually be a lower threshold for proceeding with a balloon angioplasty of an isolated iliac stenosis than embarking on femoro-popliteal bypass.
- IC can occur in younger patients (ages 20–40). Some of these patients will have early and aggressive atherosclerosis; the remainder will have other causes of arterial stenosis or occlusion. These are rare, but include popliteal artery entrapment, cystic adventitial disease, fibromuscular dysplasia, and arteritis.

Critical limb ischaemia

- CLI is usually the result of progression of lower limb atherosclerosis, rendering limb perfusion inadequate even at rest.
- This results in ischaemic rest pain in the toes or forefoot, and the development of tissue necrosis.
- The TransAtlantic Inter-society Consensus (TASC) definition of CLI requires the presence of:
  - Persistent, recurring ischaemic rest pain requiring opiate analgesia for at least 2 weeks.
  - Ulceration or gangrene of the foot or toes.
  - An ankle systolic pressure <50mmHg or toe systolic pressure <30mmHg.
- Risk factors for disease progression in patients with IC are:
  - Continued smoking.
  - Diabetes.
  - An ankle-brachial pressure index of <0.5.
- More rarely, the arterial occlusions are the result of embolization from a proximal source, e.g. aortic or popliteal aneurysm, arterial entrapment, cystic adventitial disease, fibromuscular dysplasia, or arteritis.

- In patients with CLI, the limb is at risk and, even with intervention, there is a significant chance of limb loss.
- Coronary and cerebral atherosclerosis, chronic obstructive pulmonary disease (COPD), and other comorbidities are common, requiring appropriate medical therapy in their own right to optimize fitness prior to intervention.
- These are challenging patients and the development of CLI has serious implications for the patient and their leg.
- 1yr after diagnosis of CLI, 30% of patients will be living with an amputation and 25% will be dead.

Acute limb ischaemia

- Acute ischaemia denotes a quickly developing or sudden decrease in perfusion and may affect the upper, as well as lower limbs.
- Embolic acute limb ischaemia has decreased in recent years as rheumatic heart disease has declined and the anticoagulation management of atrial fibrillation has improved.
- As a result, an increasing proportion of acutely ischaemic lower limbs are due to thrombosis of pre-existing atherosclerotic disease and usually require more complex intervention.
- Another notable cause is the sudden thrombosis of a popliteal artery aneurysm. Chronic embolization from the aneurysm has often led to occlusion of one or more calf arteries and when it eventually occludes results in profound distal ischaemia.
- The severity of ischaemia may vary and dictates the urgency of intervention required:
  - A clearly viable, although ischaemic, limb will have no neurological compromise and audible distal Doppler signals. There is more time for arterial imaging and patient optimization prior to intervention.
  - A threatened limb is more ischaemic, and displays reduced sensation and motor function, and inaudible Doppler signals. Urgent intervention within 12h may be required to try to save the limb.
  - Occasionally, ischaemia is severe, presentation delayed, and assessment reveals an insensate, paralysed limb with fixed mottling and inaudible signals. Here, salvage is not possible; attempts at reperfusion harmful and likely to result in life-threatening reperfusion syndrome. 1° amputation or end-of-life care are the only options.

Venous disease

Varicose veins

- Varicose veins (VVs) are common.
- They affect 40% of men and 30% of women aged 18–64.
- Many patients express dissatisfaction with the appearance of VVs, although not all will seek intervention.
- Symptoms attributable to uncomplicated VVs and include:
  - Aching.
  - Heaviness or ‘tension’.
  - A feeling of swelling.
  - Restlessness.
  - Cramps.

  - Itching.
  - Tingling.
- A minority of patients develop complicated varicose veins where venous hypertension results in a spectrum of tissue injury through incompletely understood mechanisms.
- Complications include:
  - Venous/varicose eczema.
  - Lipodermatosclerosis (patches of inflamed and brown stained skin due to haemosiderin deposition).
  - Ulceration.
  - Usually occur on lower leg, more commonly on the medial aspect.
  - Bleeding following minor trauma to a particularly superficial varicosity may occur and is occasionally severe.
- It is generally accepted that varicose veins causing only cosmetic upset should not be treated within the UK National Health Service.
- Treatment of symptomatic varicose veins improves quality of life and is usually performed if symptoms are significant.
- Treatment of patients with complicated varicose veins is recommended and has been shown to reduce the risk of venous ulcer recurrence.

Treatment options

- A variety of minimally invasive therapeutic interventions are now available and have begun to replace conventional surgery.
- The great saphenous or small saphenous veins up to their respective saphenofemoral and saphenopopliteal junctions may be ablated by laser fibre, radiofrequency probe, or sclerosant injection. The visible varicosities may be left to regress or be treated at the same time with phlebectomy or sclerosant injection.
- These minimally invasive techniques cause less pain and allow quicker recovery than conventional surgery with comparable mid-term results.

Vascular access

- Within the United Kingdom, the number of patients commencing renal replacement therapy continues to grow. Peritoneal dialysis will be chosen in approximately 25%, while around 70% start haemodialysis.
- It is difficult to achieve durable vascular access for haemodialysis.
- Patients should be referred for permanent vascular access at least 16 weeks and preferably 6 months before the anticipated start of dialysis. This gives time for the access (fistula) to mature and allows 2° procedures in the event of fistula thrombosis or delayed maturation.
- Haemodialysis via autogenous arteriovenous access is preferred option, minimizing infective episodes and preserving central vein patency.
- The prognosis of patients who start haemodialysis with a catheter is worse than those commencing with an autogenous access for several reasons:
  - Inherent complications from the anastomosis.
  - Vascular injury from repeated needling predisposes to stenosis, aneurysm formation and thrombosis.
  - The need for repeated pressure to obtain haemostasis.

- Typically:
  - Early fistula failure occurs in 15%.
  - 1-yr 1° patency is 63%.
  - 1-yr 2° patency rates are 66%.
- Many patients require repeated procedures to maintain autogenous access. The non-dominant arm is initially used and should veins of both arms become exhausted then autogenous access can be created within the legs.
- Should no autogenous access be possible then insertion of a prosthetic graft between an artery and a more central vein will be required. Unfortunately, the patency of synthetic grafts used for access are poorer and infection risk greater than autogenous access.

Decision making in vascular surgery

Vascular clinical assessment

As with most areas of medicine, the diagnosis of a particular vascular disease can usually be made after taking a thorough history and performing a detailed clinical examination. Determination of the impact of symptoms on quality of life is particularly important. Currently prescribed medication needs to be established as this may require the addition of 2° prevention medication, such as antiplatelet agents, statins, ACE inhibitors, and beta-blockers. Cardiovascular risk factors (smoking, hypertension, diabetes, and hyperlipidaemia) should be documented and later addressed. Many patients have associated cardiorespiratory and renal disease, and the severity and functional significance of these requires consideration when planning patient care.

Vascular imaging

Ultrasound

Modern medical ultrasound (US) scanners produce high resolution, real time grey scale images of arteries and veins. This anatomical information may be all that is required, such as when measuring the diameter of an AAA (Fig. 3.1). However, US may also provide important functional information. Colour flow imaging demonstrates flow within vessels and arbitrarily attributes a red or blue colour depending on whether flow is towards or away from the probe. The relative velocity of blood flow is also depicted by the shade of the red or blue colour. Specific uses of colour flow include the detection of reflux (reversed flow) within veins during the assessment of varicose veins and detecting stenoses within arteries when the velocity of blood must increase to pass through the narrowing. Spectral analysis can then be utilized to measure the velocity of flow through a stenosis to give an indication of its haemodynamic significance.

Common specific indications for US in vascular surgery:

- Screening for or surveillance of AAA.
- Detecting sites of reflux when assessing venous disease including varicose veins.

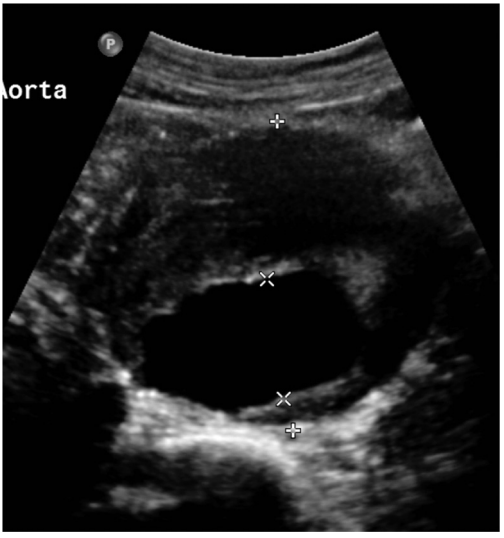

Fig. 3.1 Abdominal US image revealing a 5.6cm aneurysm of the aorta. Note how the thick layer of intramural thrombus is differentiated from the echo free vessel lumen.

Reproduced from Darby *et al*., *Oxford Handbook of Medical Imaging*, 2011, figure 7.15, p. 123, with permission from Oxford University Press.

- Detecting arterial stenoses/occlusions in peripheral and carotid arterial disease.
- Detecting acquired stenoses within the graft, as well as the inflow or outflow of arteries following bypass surgery.
- Preoperative assessment (size and patency) of vessels prior to dialysis access surgery.
- Preoperative planning, e.g. vein mapping prior to use for bypass surgery, on table marking of skin incisions.
- Detection of endoleaks following EVAR.

Advantages of US

- Non-invasive.
- Provides anatomical and functional information.
- Relatively cheap.
- Increasingly mobile.
- No ionizing radiation.
- No contrast usually required.
- Can be used in the presence of mobile ferrous implants (in contrast to MRI).

Disadvantages of US

- Operator dependent.
- Body habitus dependent with poor views of deep structures.
- No 'road map' images produced.
- Detailed scans may be time-consuming, especially with extensive disease.

Computed tomography

Following the injection of iodinated contrast, modern CT scanners can acquire high resolution scans, e.g. thoracic and abdominal aorta, within seconds. The data collected can be presented in transverse, sagittal or oblique planes. It may also be reformatted to provide 3D reconstructions. As a result, it is the preferred imaging modality to assess complex large vessel disease, e.g. aortic aneurysm or dissection, vena caval compression, or infiltration. Although CT can produce peripheral angiograms, evaluation of small arteries (e.g. within the calf) can be hampered by extensive calcification. It also allows the evaluation of the other organs within the body.

Common specific indications for CT angiography in vascular surgery

- Diagnosis of ruptured AAA.
- Diagnosis of thoracic acute aortic syndromes, e.g. aneurysm, dissection, intramural haematoma, penetrating ulceration.
- Assessment of AAA prior to EVAR.
- Stent graft surveillance following EVAR.
- Detection of synthetic graft infection.
- Assessment of blunt or penetrating trauma.
- Assessment of gastrointestinal bleeding.

Advantages of CT angiography

- Provides rapid, high resolution anatomical information.
- 'Road map' images produced.
- Less user and body habitus dependent.
- Can be used in the presence of mobile ferrous implants.

Disadvantages of CT angiography

- Ionizing radiation.
- Nephrotoxic iodinated contrast required for angiography.
- Not mobile.
- Poorer image quality of small calcified vessels compared with MRA or conventional intra-arterial angiography.

Magnetic resonance angiography (MRA)

The application and response of strong magnetic fields to tissues, and use of gadolinium contrast agents allows the production of high resolution images. Similarly to CT, the data can be manipulated to produce 3D reconstructions to aid treatment planning. In many centres, it has nearly replaced conventional diagnostic intra-arterial angiography to assess peripheral arterial disease. Although it can demonstrate aortic disease, CT angiography is generally preferred to provide the measurements required for the planning of endovascular treatment (Fig. 3.2). Patients must be screened for the presence of mobile ferrous material, e.g. metal heart valves, intracranial clips, pacemakers, intra-ocular metal fragments, etc.

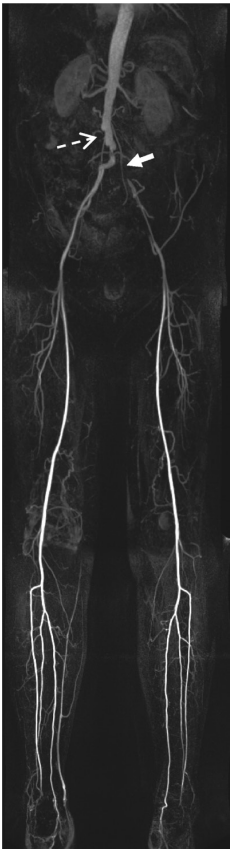

Fig. 3.2 Coronal view of a maximum intensity projection (MIP) composed of three multi-station MRA data sets. Note the atherosclerotic changes of the infrarenal aorta (dashed arrow) with occlusion of the left common iliac artery (thick arrow). The more distal leg vessels have no significant stenoses.

Reproduced from Myerson *et al.*, *Oxford Specialist Handbook: Cardiovascular Magnetic Resonance*, 2010, figure 14.5, p. 395, with permission from Oxford University Press.

Common specific indications for MRA in vascular surgery

- Assessment of peripheral arterial disease.
- Assessment of carotid artery disease often combined with brain imaging.
- Assessment of mesenteric arterial disease.
- Assessment of central vein patency.

Advantages of MRA
- No ionizing radiation.
- Provides rapid high resolution anatomical information.
- 'Road map' images produced.
- Less user and body habitus dependent.
- Fewer artifacts from calcification allowing improved imaging of small calcified vessels.

Disadvantages of MRA
- Contraindicated if mobile ferrous implants.
- Signal drop out limits assessment of vessel patency within vascular stents.
- Rare, but severe systemic fibrotic reaction associated with gadolinium and renal impairment.
- Not mobile.
- Equipment and monitoring difficulties when scanning critically ill patients.
- Significant proportion of patients refuse scan due to claustrophobia.

Direct intra-arterial angiography

In this technique, access to the arterial circulation made by direct puncture most commonly via the common femoral artery. The injection of iodinated contrast injection and the use of digital subtraction allows high resolution images to be produced. In many centres, its use as a purely diagnostic test has been superseded by less invasive imaging modalities (US, CT, MRA; see Fig. 3.3); its use being reserved to guide endovascular interventions, e.g. balloon angioplasty, stent insertion, or therapeutic embolization.

Common specific indications for intra-arterial angiography in vascular surgery include:
- Assessment and treatment of peripheral arterial disease.
- Assessment and treatment of carotid artery disease.
- Assessment and treatment of mesenteric arterial disease.
- Assessment and treatment following blunt and penetrating trauma.
- Assessment and treatment of gastrointestinal and pulmonary bleeding.

Advantages of intra-arterial angiography:
- Provides rapid, high resolution, anatomical information, even of small calcified vessels and within stents.
- 'Road map' images produced.
- Less user and body habitus dependent than other imaging modalities.
- Pressure measurements across stenoses allows determination of the haemodynamic significance of lesions.
- Speed of contrast flow allows subjective assessment of disease severity or improvement after intervention.
- Can be used in the presence of mobile ferrous implants.
- Able to proceed from imaging to therapeutic intervention as part of the same procedure if appropriate.

Disadvantages of intra-arterial angiography:
- Hemorrhage following arterial puncture.
- Distal embolization or local arterial injury e.g. dissection complicating puncture or intra-arterial manipulations.

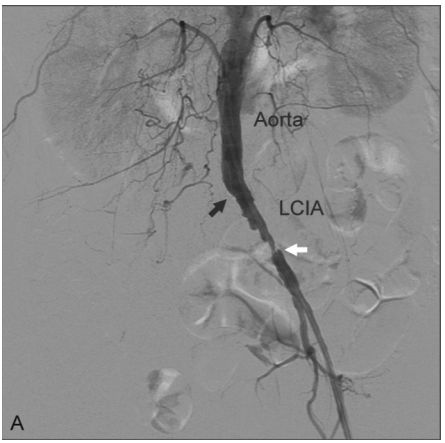

Fig. 3.3 Digital subtraction angiogram of chronic total iliac occlusion (upper arrow). Note also the severe stenosis in the left common iliac artery (lower arrow).

Reproduced from Camm et al., The ESC Textbook of Cardiovascular Medicine, second edition, 2009, Figure 36.5A, page 1314, with permission from Oxford University Press.

- Ionizing radiation
- Nephrotoxic iodinated contrast required for angiography

Vascular conditions

Infrarenal abdominal aortic aneurysm

AAAs cause few symptoms before rupture. Most are detected through examinations or investigation for other medical problems. Occasionally, they are considered to be the cause of chronic back ache, or a patient reports a pulsatile appearance or sensation within the abdomen. Although acute presentation of an AAA is usually due to rupture, they may rarely present through distal embolization or fistulization into the bowel (duodenum or colon) or inferior vena cava.

Elective presentation

These are usually an elderly group of patients. Significant co-morbidities are usual, and cardiac, respiratory, and renal disease are particularly common. Three interdependent factors need to be addressed to allow formulation of a patient's management:

- Size (and anatomy) of AAA.
- Fitness for potential intervention.
- Patient's ideas, concerns, and expectations regarding their AAA. What treatment do they wish?

Table 3.1 Annual risk of rupture with increasing abdominal aortic aneurysm diameter

Abdominal aortic aneurysm diameter (mm)	Risk of rupture per year (%/year)
<40	Very rare
40–49	1
50–59	1–11
60–69	10–20
70–79	20–40
>80	30–50

All interventions to repair AAA may cause complications. Therefore, before invasive treatment can be recommended, the risk of intervention must be balanced against the risk of rupture for each individual patient. The risk of AAA rupture increases with aneurysm diameter (see Table 3.1). In general, the balance tends to tip towards intervention when an aneurysm reaches 5.5–6cm.

Small AAA

The risk of rupture of an AAA less than in diameter 5.5cm is relatively low, so patients should be entered into surveillance programme, consisting of regular US scans, e.g. annually until 4.5cm diameter, then every 3 months.

Despite low AAA rupture risk, these patients have a significant risk of atherosclerotic cardiovascular events, e.g. myocardial infarction and stroke. This should be reduced as much as possible through risk factor modification (healthy life style advice, smoking cessation, optimization of BP, and diabetic control), plus the prescription of appropriate 2° prevention medication, e.g. antiplatelet agents (aspirin/clopidogrel), statins, and ACE inhibitors.

Large AAA

Patients with an AAA that is 5.5cm in diameter or is increasing in size rapidly, e.g. >0.5cm over 6 months will be considered for intervention. The usual methods for repair are conventional open surgery or EVAR using a stent graft. Some specialist centres offer laparoscopic repair.

Although stent graft designs continue to improve, not all AAAs are anatomically suitable for repair with a standard infrarenal stent graft. This may be due a short or angulated neck (a relatively normal aorta below the renal arteries required to form the proximal sealing zone), or due to narrow or occluded iliac arteries. Although techniques are evolving to deal with these anatomical challenges, e.g. fenestrated and branched grafts, and iliac conduits, these do increase the complexity, potential complications and cost of these procedures.

Patient assessment

Once the size (determined by US) of an AAA has reached the threshold to consider intervention, more anatomical detail is required which is usually provided by a CT angiogram (📖 Decision making in vascular surgery, p. 95, Computed tomography, p. 97).

A patient's 'fitness for surgery' is established on the basis of clinical assessment and investigations. These include:

- History of cardiorespiratory and other diseases and the impact that it has on functional status. Previous abdominal surgery or radiotherapy increases risk of laparotomy.
- *'Base line' investigations:*
  - Blood assays, e.g. full blood count (FBC), urea & electrolytes (U&E), liver function tests (LFTs), clotting.
  - Electrocardiogram (ECG).
  - Lung function tests.
- *Dynamic tests of physiological function:*
  - Echocardiogram (stress).
  - Cardiopulmonary exercising test.

The predictive value of these tests is imperfect, but may reveal under treated co-morbid disease, e.g. ischaemic heart disease, COPD. Optimization of medical therapy for these should be performed prior to surgical intervention if time allows.

Following these investigations, a patient may be classified into one of three groups:

- Usual/normal operative risk.
- Greater than normal/usual risk.
- Excessive risk.

This information then informs the discussion with the patient regarding the risks and benefits of treatment. At all times, the risk of intervention must be balanced against the risk of rupture. In general,

- *Patients with usual/normal risk:* proceed with intervention once medically optimized.
- Patients with greater than usual/normal risk may be considered to have an acceptable risk for intervention once medically optimized. If the risk of intervention is considered too great, treatment is deferred until AAA size (and rupture risk) increases.
- Patients with excessive risk who are never likely to be fit for any intervention are not offered invasive treatment.

Treatment options

In most centres these consist of open surgical repair or EVAR. Infrarenal AAA morphology (neck length, shape, and angulation) precludes EVAR in a decreasing proportion of patients, as stent graft design and operator experience improve. Certain factors may increase the desirability of EVAR, e.g. known dense intra-abdominal adhesions, or dual pathology of AAA and malignant neoplasm (e.g. colonic, renal, lung) that also requires treatment.

Thirty-day mortality following EVAR is approximately one-third that of conventional repair (2% versus 5%). However, once the patient has

recovered from open repair, intense surveillance is not required as the need for further intervention is very low. In contrast, following EVAR, long-term surveillance (combination of regular CT, US, and plain radiography) is required to ensure graft function and further procedures are required in approximately 20% of patients. Although relatively minor, these interventions do result in morbidity and occasional mortality, and this may counteract early survival benefit. On long-term follow-up there is no difference in survival between the two interventions. ∴ the treatment selected should be tailored to the individual patient, taking account of their fitness, their anatomy and their wishes.

Carotid disease

Carotid atherosclerosis usually causes symptoms through intracerebral embolization of atherothrombotic material resulting in a TIA or ischaemic stroke. It is responsible for up to 50% of carotid territory brain infarction (other causes include small cerebral vessel disease (25%) and cardiogenic embolism (15%)). Only rarely are symptoms the result of reduced cerebral perfusion due to carotid artery stenoses.

Predicting which carotid plaque will cause an initial or further event is difficult and in current clinical practice is limited to measuring the 'severity' of the carotid disease expressed as the degree of stenosis. This is usually measured by US scanning and internal carotid stenoses of 70–99% are most likely to cause new or recurrent embolic symptoms.

Transient ischaemic attack

Following a TIA, the risk of subsequent stroke increases dramatically and is maximal within the first few days of the event. Clinical scoring systems (e.g. ABCD2 score for TIA) have been devised to assess the degree of risk to help guide management (see Table 3.2).

After a TIA, patients require rapid assessment, e.g. by a stroke physician or neurologist:

- To confirm the diagnosis and exclude other significant conditions, e.g. epilepsy or migraine.
- To commence medical treatment including antiplatelet therapy (aspirin or clopidogrel) and statin therapy.
- To arrange imaging, generally US scan of carotid arteries and a head CT scan in many cases.

Patients with an internal carotid stenosis greater than 50% may benefit from of carotid endarterectomy in addition to ongoing medical therapy. The aim of the surgery is to lower the chance of a future ischaemic stroke. This is most likely to occur within the first few days or weeks following the index event so delay in endarterectomy following TIA will result in a number of patients suffering potentially preventable strokes. When carotid endarterectomy is performed in acute cases where there is likely to be an unstable atherosclerotic plaque, the risk of stroke complicating the procedure is likely to be slightly greater than for 'delayed' surgery. However, this is significantly outweighed by the considerably greater number of strokes prevented from early (within 2 weeks) intervention.

The risk of stroke falls with time after TIA. If a patient has not suffered a stroke after a certain period of time, they may no longer benefit from

Table 3.2 The components of the abcd2 score are age, blood pressure, clinical features, duration of TIA symptoms and diabetes.

abcd2 score	48 Hour risk of stroke
1	0%
2	2%
3	3%
4	5%
5	7%
6	14%
7	Up to 50%

They are scored as follows: age ≥ 60 years = 1, systolic blood pressure ≥ 140 mm Hg or diastolic pressure ≥ 90 mm Hg = 1; unilateral weakness = 2 or speech impairment without weakness =1; duration of TIA ≥ 60 min = 2 or 10 – 59 min = 1; diabetes = 1.

endarterectomy. This depends on the patient's gender, the degree of carotid stenosis and the time since the initial TIA. Such that:

- Maximal risk reduction is achieved for men and women when CEA performed within 2 weeks of TIA with 50–69% and 70–99% stenoses.
- Maintained benefit from CEA beyond 12 weeks after TIA in men with 70–99% stenosis and also marginal benefit with 50–69% stenosis.
- There is no benefit from CEA beyond 2 weeks after TIA in women with 50–69% stenosis or beyond 4 weeks with 70–99% stenosis.

Ischaemic stroke

Patients who suffer an ischaemic stroke and present immediately (within 4–6h) may be candidates for systemic thrombolysis once a cerebral haemorrhage has been excluded. These and other patients who are not eligible for thrombolysis should have urgent carotid imaging to exclude significant internal carotid disease. Those patients with a stenosis of >50% may benefit from the addition of carotid endarterectomy to ongoing antiplatelet therapy. A particular complication following endarterectomy in patients with cerebral infarction is the risk of provoking intracerebral haemorrhage. This risk appears to be greater in patients with large infarcts, dense deficits, and poor residual function. Deferring surgery for several weeks probably lowers the procedural risk, but significant numbers of patients will suffer further ischaemic events while awaiting surgery. Therefore, patients must be individually assessed with early endarterectomy being appropriate in those with:

- Relatively rapid recovery of deficit.
- Good functional status (Rankin score 0–2).
- Infarction less than a third of the carotid territory.
- No intracerebral haemorrhage.
- Lucid and able to consent.
- No carotid occlusion.

Rankin score

0 No symptoms at all.
1 No significant disability despite symptoms; able to carry out all usual duties and activities.
2 Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance.
3 Moderate disability; requiring some help, but able to walk without assistance.
4 Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance.
5 Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6 Dead

Asymptomatic carotid disease

The risk of a TIA or stroke in a patient with significant, but asymptomatic carotid disease on medical therapy is low at just over 2% per year. Randomized control trials show that the overall risk can be approximately halved with the addition of carotid endarterectomy. However, the effects are sex dependent with women gaining much less from surgery than men. Recent European guidelines find that endarterectomy can be recommended for asymptomatic men with a stenosis of 70–99% (as long as the operative risk is <3%). Conversely, endarterectomy in women should only be considered in younger and fitter women who are expected to live long enough to gain any benefit of surgery.

Peripheral arterial disease

In the vast majority of patients with lower limb ischaemia the cause is atherosclerosis. Lower limb atherosclerosis may result in a spectrum of disease depending on the extent of arterial stenosis or occlusion and the functional demands of the patient. It ranges from asymptomatic through intermittent claudication through to rest pain and tissue loss (ulceration and/or gangrene).

Intermittent claudication

The diagnosis of intermittent claudication can usually be made from the patient's symptom description (aching pain within calf, thigh, or buttock; precipitated by walking; relieved by rest). This is supported by demonstration of reduced or absent lower limb pulses and reduced ankle-brachial pressure index. A sense of the 'severity' of symptoms should be achieved by enquiring about walking distance and the impact this has on quality of life. Assessment of cardiovascular risk factors (diabetes mellitus, hypertension, hyperlipidaemia, smoking, family history) and co-morbidities (especially cardiac, respiratory, and renal) is also required.

Management of a patient with intermittent claudication can be considered as three interdependent parts:

- *Patient education and explanation:* what treatment do they wish?
- Modification of cardiovascular risk factors (smoking cessation, control of hypertension, diabetes optimization, etc.) and prescription of 2° prevention medication (antiplatelet agents, statins, ACE inhibitors). Optimization of cardiac or respiratory co-morbidity.
- Specific treatment of claudication symptoms.

Treatment of intermittent claudication

- *Conservative*: following explanation many patients require or desire no specific treatment of claudication symptoms. Over next 5yrs, 70–80% of patients can expect their symptoms to remain the same or even improve through collateral development and changes in behaviour.
- *Exercise training*: exercise programmes, particularly those that are supervised, result in a significant improvement in symptoms, although enthusiasm for attending these programmes wanes with time!
- *Drug therapy:* naftidrofuryl and cilostazol may improve walking distance in a proportion of patients. Little to be gained in continuing therapy for more than a few months if there has been no response.
- *Invasive intervention:* if symptoms are severe and are significantly reducing quality of life or have failed to respond to a period of conservative/exercise treatment then invasive intervention may be appropriate.
  - Arterial imaging is required to plan treatment. US, MRA, or CT-angiogram (CTA) may be used to demonstrate the pattern of disease and determine the need for balloon angioplasty, open surgery or combination of both to improve leg perfusion.
  - Only 5–10% of patients with intermittent claudication will develop critical leg ischaemia and so require treatment to preserve their leg.
  - Treatment for claudication is therefore aimed at improving quality of life through improved mobility and, therefore, a frank discussion about the risks (that include amputation and death) and benefits of invasive intervention is required to help patients make an informed choice.
  - Treatment needs to be tailored to the individual patient, but in general there would usually be a lower threshold for proceeding with a balloon angioplasty of an isolated iliac stenosis than proceeding with a femoro-popliteal bypass.

Critical lower limb ischaemia

Patients with ischaemic rest pain or ulceration and gangrene require a similar approach to those with intermittent claudication:

- *Patient education and explanation:* what treatment do they wish?
- *Modification of cardiovascular risk factors* (smoking cessation, control of hypertension, diabetes optimization, etc.) and prescription of secondary prevention medication (antiplatelet agents, statins, ACE inhibitor). Optimization of cardiac or respiratory co-morbidity.
- Specific treatment to improve limb perfusion or major amputation.

Treatment of critical limb ischaemia

Without treatment, the limb is at risk and so the aim of treatment is to preserve quality of life through limb salvage. Although end of life/palliative care is chosen by a few patients most wish to proceed with intervention if feasible. Arterial imaging is required and treatment again determined from the pattern of disease. Balloon angioplasty, open surgery or a combination of both may be required. As the stakes are higher, more invasive intervention is often recommended, e.g. distal bypass surgery that would not normally be considered for patients with claudication.

Acute lower limb ischaemia

Acute upper or lower limb ischaemia follows a sudden decrease in perfusion. Embolic acute limb ischaemia has decreased in recent years as rheumatic heart disease has declined and anticoagulation management of atrial fibrillation has improved. An increasing proportion of acutely ischaemic lower limbs is due to thrombosis of pre-existing atherosclerotic disease and often requires more complex intervention. Acute thrombosis of a popliteal artery aneurysm often results in a particularly ischaemic limb.

The diagnosis is usually clear with the findings of a painful, pale, pulseless and perishingly cold limb, possibly with a degree of paraesthesia and paralysis. See Table 3.3 for a classification scheme.

Significant patient co-morbidity is usual, e.g. recent myocardial infarction, uncontrolled atrial fibrillation, etc. It is important to define the degree of ischaemia (see Table 3.3) as this predicts clinical outcome and dictates the speed at which treatment must be arranged. A viable, although ischaemic, limb will have no neurological compromise. There is more time for arterial imaging and patient optimization prior to intervention. A threatened limb is more ischaemic and displays reduced sensation and motor function and inaudible Doppler signals. Urgent intervention that night may be required to attempt limb salvage. Occasionally, the ischaemia is severe and the presentation delayed and assessment reveals an insensate, paralysed limb with fixed mottling and inaudible signals. Limb salvage is not possible and reperfusion harmful as likely to result in life threatening reperfusion syndrome due to the 'washing' of ischaemic muscle metabolites (potassium, myoglobin, lactate, cytokines) into the systemic circulation. 1° amputation or end-of-life care are the only options.

Systemic anticoagulation is commenced to prevent propagation of thrombosis beyond the precipitating embolism or thrombosis. This will usually preclude neuroaxial anaesthesia and may be deferred if this mode of anaesthetic is considered important and delay in intervention minimal.

The need for preoperative imaging will depend on the surgeon's preference, skills, and local resources at the hour of treatment. Viable limbs can await imaging on the next working day. Patients with threatened limbs may be taken to theatre and on table angiography performed to determine the precise procedure required. This will depend on the cause and distribution of occluded arteries. Embolic occlusion of the femoral arteries in the groin will usually only require balloon thromboembolectomy. This can usually be performed through a relatively short groin incision under local anaesthesia with or without sedation. Acute on chronic superficial femoral or popliteal occlusion is likely to require femoro-popliteal or femoro-distal (to calf arteries) bypass, preferably using adjacent great saphenous vein or synthetic graft. In some complex cases, on table thrombolysis (tissue plasminogen activator) and balloon angioplasty of iliac and/or crural arteries are required in addition to bypass surgery. Following revascularization, compartment syndrome is a common and potentially devastating complication, and so prophylactic calf fasciotomies will often be performed.

Upper limb ischaemia

Limb-threatening chronic upper limb ischaemia is far less common than lower limb disease in part due to the good collateral blood supply around

Table 3.3 Classification scheme for acute limb ischaemia

Category	Description	Capillary return	Muscle paralysis	Sensory loss	Arterial Doppler signal	Venous Doppler signal
I Viable	Not immediately threatened	Intact	None	None	Audible	Audible
IIa Threatened	Salvageable if promptly treated	Intact/slow	None	Partial	Inaudible	Audible
IIb Threatened	Salvageable if immediately treated	Slow/absent	Partial	Partial/complete	Inaudible	Audible
III Irreversible	Primary amputation	Absent	Complete Tense compartments	Complete	Inaudible	Inaudible

the shoulder. Acute upper limb ischaemia is more common and usually the result of embolic occlusion. The principals of management are the same as those for lower limb disease. The most commonly required procedure is brachial embolectomy, which can usually be performed under local anaesthetic. Bypass surgery to salvage an ischaemic upper limb is rarely required.

Vascular access surgery

Haemodialysis via an autogenous arteriovenous access is preferred to the use of a central venous catheter, and minimizes infective episodes and preserves central vein patency. US assessment of upper limb vessels is increasingly used to guide intervention and has been shown to improve autogenous access maturation and use. The radial and brachial artery must be relatively free other atherosclerotic disease and contain multiphasic flow. Likewise, the cephalic and basilic veins must be patent and free from stenoses. The diameter of vessels is also measured and a threshold of 2 mm for radial artery and cephalic vein diameters at the wrist is recommended if these vessels are to be used. Using vessels with diameters below this threshold is significantly less likely to produce a fistula that matures to give functional access. Although the sequence of procedures will alter according to institutional or surgeon preference, the first access is usually created in the non-dominant arm to keep the dominant arm free during haemodialysis sessions. In addition, the access is created as distally as the quality of vessels allows preservation of more proximal options for future use. The likely sequence of procedures may be:

- Radial artery to cephalic vein—in anatomical snuff box.
- Radial artery to cephalic vein—at wrist.
- Radial artery to cephalic vein—mid-forearm.
- Brachial artery to cephalic vein—in arm.
- Brachial artery to basilic vein or brachial vein (vein requires transposition to bring it to a superficial position)—in arm
- The above sequence in the dominant arm.
- Synthetic graft—brachial artery to cephalic/basilic vein as loop in forearm or brachial artery to axially vein in arm

If the upper limbs are exhausted, then an access can be created within the lower limbs. As with the upper limb, an autogenous access is associated with a lower risk of infection and can be performed between the femoral arteries and the great saphenous or superficial femoral vein in a variety of configurations. Synthetic grafts are also used, but pose the risk of increased infection and thrombosis.

Rarely, the more common upper and lower limb options are exhausted. More creative, but invasive use of synthetic grafts is then required, e.g. axillary artery to axillary vein/jugular vein, axillary artery to IVC/iliac/femoral/popliteal vein, femoral artery cross over to femoral vein, or femoral or axillary artery to SVC.

Radial and brachial artery to cephalic vein accesses can be easily performed with local infiltration. Although transposition of the basilic or large brachial vein may be performed under local infiltration, a regional or general anaesthetic is usually more comfortable for both patient and surgeon.

Many patients with chronic kidney disease generally have significant cardiorespiratory disease and so the decision of which access and anaesthetic should be tailored to each patient. Close co-operation between surgeon and anaesthetist is required for optimal patient outcome.

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The vascular preoperative assessment clinic

Introduction

Patients presenting for vascular surgical procedures have a high incidence of co-morbidity, particularly in relation to cardiorespiratory disease. Co-morbid disease should be assessed and optimized in advance of surgery to minimize the risk of adverse outcome. A multidisciplinary approach has been identified as the most appropriate way to achieve this, with the vascular anaesthetist playing a major role. The ideal scenario is for patients to be assessed in advance of planned surgery allowing sufficient time for optimization, counselling, and specialist referral if required. A preoperative assessment clinic with senior anaesthetic involvement provides the ideal platform to achieve this. It is vital that appropriate infrastructure, education, resources, and facilities are made available locally to facilitate effective preoperative assessment. Once in place the preoperative assessment clinic should function as the hub of a well set-up multidisciplinary team.

Clinic organization

Consultant input

Preparation for vascular surgery involves the preoperative assessment of complex high-risk patients. Therefore clinics should be consultant-led with appropriate nursing support.

- Regular sessional time should be made available to adequately fulfill this requirement.
- The time required will depend on clinic structure and patient throughput.
- Consultants leading preoperative assessment should have an appropriate knowledge of likely co-morbid disease processes and risk assessment tools.

Nursing support

Medical provision should be supported by an appropriate nursing infrastructure. Not all tasks in the preassessment clinic have to be undertaken by medical staff.

- Basic history taking and, in some cases, basic medical examination and investigation, can be undertaken by non-medical staff.
- Ideally, the clinic team should include specialist nurses, as well as other grades of staff to facilitate all required tasks and streamlined patient throughput.
- Clerical, secretarial and information technology support are also required for patient booking, audit and governance.

Clinic structure

Effective vascular preoperative assessment can be delivered as part of a wider ranging preoperative facility or as a stand-alone clinic. The clinic processes and support are most important in this respect, but adequate office space is required. It is also important to minimize patient inconvenience during clinic visits. This can be achieved by:

- *Provision of a 'one-stop' facility where possible*—ideally medical and nursing review and investigations should be performed at one clinic visit.
- *Provision of appropriate transport facilities*—particularly where vascular services are centralized and patients may be required to travel long distances. Close liaison with local ambulance services helps facilitate this.
- *Allocation of appropriate length consultations to patients*—to allow for accurate risk assessment and informed discussion of complex issues relating to care.

Investigations

Ideally patients should be seen by nursing and medical staff with appropriate baseline investigations undertaken. This requires ready access to ECG, chest X-rays and phlebotomy, and rapid turnaround of blood tests. It is useful to have the facility to undertake arterial blood gas analysis.

Equipment

Funding should be made available for the purchase of simple clinical equipment that may influence risk assessment, e.g. spirometry and pulse oximetry. Such upfront capital outlay is negated in the longer term by reduced patient referrals and waiting times.

Multi-disciplinary links

In order to enable effective risk assessment clinicians require ready access to senior colleagues in other specialties, e.g. cardiology, respiratory, and renal medicine. This enables specialized, more invasive investigation of a minority of patients where indicated.

- These referrals should be prioritized so that undue delays to surgery are avoided.
- Locally drawn up referral guidelines will facilitate this process.

Streamlined referral pathways from the vascular surgical clinics are also important to minimize waiting times to surgery. This aspect is becoming

increasingly important in most settings and clinical services. For example, the newly introduced UK AAA screening programme recommends a maximum period of 8 weeks from referral to surgery.

Functions of preoperative assessment

Accurate risk assessment

This is the cornerstone of a well delivered service.

- Effective risk assessment should be delivered at the individual patient level with the aid of: history, examination, investigations, functional status and surgery specific risk.
- A validated, specific scoring system is useful to then bring all components together (see 🕮 The vascular preoperative assessment clinic, p. 110).

Patient counselling

Individuals should leave clinic fully informed of the implications and risks and benefits of their planned surgical procedure. Patient information leaflets are useful as part of this process. In certain circumstances the patient and the clinician may jointly reach the decision that surgery is inappropriate. Evidence is available to demonstrate that well conducted preoperative assessment also increases overall patient satisfaction levels.

Preoperative risk minimization

The preoperative assessment clinic provides the ideal platform to allow medical optimization in advance of surgery. This should be based on current best available evidence, and achieved in close liaison with primary care physicians and vascular surgical colleagues.

On occasion specialist referral, e.g. cardiology may be required to demarcate risk further. Guided medical optimization can then be achieved in conjunction with the appropriate specialty. In rare circumstances, interventional therapy may be required prior to planned vascular surgery. This might include preoperative coronary bypass grafting or percutaneous intervention for example. This is discussed in more detail in 🕮 Management of the vascular surgery patient after coronary revascularization, p. 173.

Planning of perioperative management

Early assessment allows identification of patients who may require more intensive intraoperative monitoring and planning of appropriate postoperative care, at an early stage. This improves utilization of critical care resources in particular.

Minimization of late cancellation

Last-minute surgical cancellations due to poor medical status should be avoided as a result of well delivered preoperative assessment. Appropriate advice in relation to preoperative medication, e.g. warfarin, clopidogrel should also be given at the clinic visit, thereby avoiding late cancellations for therapy, which has inappropriately been continued due to poor patient advice.

Lifestyle modification

Many patients with vascular disease are smokers, overweight, unfit, and sedentary in association with a high incidence of comorbid disease. As

part of any holistic package of care it is appropriate to empower individuals to make certain adjustments to behaviour and lifestyle. In addition to pharmacological optimization, such changes are aimed at reducing risk both perioperatively and in the longer term. This is covered in detail in 📖 Primary and secondary prevention of vascular disease, p. 14. In brief, this care may include:

- Weight and dietary advice.
- *Smoking cessation:* ideally this should be achieved in conjunction with an approved smoking cessation service. Such an arrangement can be effectively delivered with primary care assistance.
- *Optimization of co-morbidity:* education of patients at preoperative assessment is possible in relation to effective management of co-morbid disease. Simple advice on control of diabetes or COPD, for example, is likely to pay dividends in both the perioperative period and longer term.
- *Exercise:* evidence exists that significant improvements in aerobic fitness can be made within a 4–6-week timeframe with the aid of an exercise programme. Although the benefits of such fitness improvements on outcome with non-cardiac surgery are yet to be elucidated, compelling supporting data is available from cardio-respiratory rehabilitation programmes:
  - Interval-based vigorous exercise regimens have demonstrated greater improvements in aerobic fitness in the short-term as compared to more prolonged lower intensity sessions. This has been demonstrated in elderly individuals with cardiac disease.
  - It is important to consider the risks of such exercise prescription in previously sedentary individuals with a high incidence of covert coronary artery disease and potentially large aortic aneurysms. Familiar interventions, e.g. brisk walking, incorporated into weekly routines may have a greater degree of patient compliance than unfamiliar interventions.
  - In the face of a lack of definitive evidence, a pragmatic approach using a 4–6-week intervention is likely to prove beneficial. This can be achieved by suggesting a familiar form of exercise, which can be undertaken at a moderate intensity level, 3 or 4 times per week for perhaps 30–40min. Advice on monitoring intensity level (e.g. using Borg perceived exertion scale) and forms of exercise can be provided via patient information leaflets.

Summary

A preoperative assessment clinic provides the ideal foundation to improve many aspects of patient management in both the perioperative period and longer term. To achieve these aims it is important that appropriately experienced personnel are involved with clinic delivery. Finally, recognition of infrastructure requirements, education, and links with the multidisciplinary team are critical components to achieving success.

The risks of vascular surgery

What are the risks?

Background

Vascular surgery carries major risks due to the combination of frequent comorbidity in this patient population and the high-risk nature of the surgical procedures. The magnitude of risk associated with emergent vascular surgery is significantly higher than in the elective situation.

Comorbid disease

The incidence of significant cardiorespiratory and metabolic disease in vascular patients is higher than in the general population as highlighted in Table 3.4. The impact of such disease processes on surgical outcome is discussed in more detail in subsequent chapters. In summary, however, optimization of such comorbidities prior to elective surgery improves outcome. This may be difficult to achieve in the emergency setting.

Surgery specific risk

Outcome following vascular surgery is dependent on several factors including:

- Urgency of surgery.
- Magnitude of surgical intervention.
- Experience of surgeon.
- Fitness and age of patient.

There is also increasing evidence that outcome may be dependent on caseload, with 'higher' volume centres achieving better results. It is appropriate to consider risks of elective and emergency surgery separately.

Elective vascular surgical procedures

This covers a diverse number of operations including arterial and venous interventions. It is not within the scope of this chapter to cover the risks associated with all such procedures. It is useful to consider risk associated with "indexed" operations for which results are reported by the majority of vascular units in the United Kingdom (UK).

Table 3.4 Comorbidity in vascular surgical patients

Comorbidity	Prevalence in vascular surgical population (%)
Cardiovascular	
Hypertension	45–85
Cardiac disease	25–55
Respiratory	
Smoking history	35–85
Respiratory disease	10–50
Renal disease	4–13

Data from 2008 VASCUNET report in individuals with AAA. Prevalence varies depending on country studied.

Open infra-renal AAA repair

Considered high-risk non-cardiac surgery. Thirty-day mortality for AAA repair varies from 2 to 8% depending on country and centre. However, national reported figures for the UK has previously been reported as approaching 8%. This has resulted in a national drive to improve outcome in the UK, through an AAA quality improvement programme.

Organ specific morbidity

This is a cause for concern and can result in prolonged hospital stay and long-term reduction in quality of life. Morbidity rates are outlined below:

- *Cardiovascular*: 5–20%.
- *Respiratory*: 10–50%.
- *Renal*: 5–30%.
- *Gastrointestinal*: 10–30%.

CEA

This is considered an intermediate risk procedure. Thirty-day mortality rates for surgery are generally <1%, but significant life-threatening morbidity can be associated with surgery:

- *Major disabling stroke*: 1–4%.
- *Cardiac event*: 1–5%.
- *Neck haematoma*: <1%.
- *Cerebral hyperperfusion syndrome*: <1%.

EVAR

Introduced as a surgical option into the UK in late 1990s. Large randomized controlled trials have confirmed that 30-day mortality associated with EVAR is significantly less than AAA at 0.5–1.5%. This mortality benefit over AAA appears to be maintained for up to 2yrs. Organ-specific morbidity associated with EVAR is also significantly lower than AAA. Newer fenestrated stent procedures, bridging the renal arteries, may carry higher risk than standard EVAR.

Emergent vascular surgical procedures

Virtually all emergency vascular arterial procedures are considered high-risk. Major blood loss, physiological derangement, and limited patient cardiorespiratory reserve all greatly contribute to this situation. Again, due to the diversity of procedures undertaken, only the risks of indexed emergent operations are considered:

- *Ruptured open abdominal aortic aneurysm repair (RAAA)*: acknowledged as the highest risk procedure with out-of-hospital mortality approaching 90%. Individuals reaching theatre and undergoing surgery have 30-day mortality rates of 25–60% depending on country and centre. The majority of deaths result from major blood loss, cardiorespiratory adverse events and multi-organ failure. Survivors have a very high incidence of morbidity across all organ systems, generally causing prolonged hospital admission. It is unusual to return to pre-morbid quality of life following RAAA intervention.
- *Emergency EVAR:* undertaken as an alternative to open repair in patients with a ruptured AAA. Physiological and surgical stresses are generally considered lower than with RAAA. Very few outcome

studies presently exist, but some series have reported 30-day mortality rates of 15–40%.
- *Lower limb amputation:* generally involves surgery on patients with end-stage peripheral vascular disease as a consequence of smoking and often diabetes. Thirty-day mortality rates of 8–20% are reported across the UK depending on centre and nature of surgery. Above knee amputation generally carries higher risk than below knee amputation. A UK quality improvement framework is currently under development to improve outcome in this patient population.

Risk assessment tools in the vascular surgery patient

As highlighted in the preceding section, vascular surgery is often associated with adverse outcome. ∴ Accurate risk assessment prior to surgery is of paramount importance in order to reduce risk at the individual patient level and improve outcome following surgery. Many risk assessment tools have been investigated and proposed, each carrying particular strengths and weaknesses. The current accepted approach to risk assessment includes the following:
- *History:* including clinical risk predictors.
- *Physical examination:* particularly in relation to the cardiorespiratory system.
- Assessment of functional capacity/aerobic fitness.
- Targeted investigations (covered elsewhere).
- The use of scoring systems to bring the above points together and predict risk at the individual patient level.

Assessment should be performed by an appropriately skilled clinician with knowledge of comorbid disease and the stresses of the planned surgical procedure. Ideally, risk assessment should be undertaken at an appropriate time in advance of surgery to allow risk reduction strategies to be implemented. This is not always possible in the emergent setting. Elective and emergency risk assessment present somewhat different challenges and it is appropriate to discuss them separately.

Elective risk assessment

In the elective setting, risk assessment can be performed at an appropriate time in advance of surgery. Ideally, this should be undertaken in an appropriately resourced preoperative assessment clinic setting with established links for rapid specialist referral and targeted investigations. There are several benefits to such an arrangement:
- Accurate assessment.
- Patient informed consent as to risks of planned surgery.
- Appropriate time interval for introduction of best medical therapy and medical optimization strategies.
- Rapid referral for specialist assessment (where appropriate) without undue delay in planned surgery.

- Appropriate time interval to allow targeted further investigations to be undertaken without undue delay in planned surgery.
- Planning appropriate level of post-operative care at an early stage.

A broad range of elective vascular surgical procedures must be accommodated when considering preoperative risk, presenting diverse and unique challenges to the vascular team. Procedures include:
- Open AAA.
- EVAR.
- Carotid endarterectomy.
- Infra-inguinal arterial bypass surgery.

Components of preoperative assessment identified at the beginning of this chapter are paramount in achieving accurate risk assessment in elective setting. Validated scoring system should be utilized, where appropriate, to bring together the available data and more accurately quantify risk.

Scoring systems

The ideal elective scoring system should:
- Be validated against outcome in the desired surgical population.
- Be simple to use.
- Provide an accurate assessment of risk at the individual patient level.
- Be inclusive of overall risk, rather than risk specific to individual organ systems, e.g. cardiac, respiratory.
- Incorporate clinical risk factors, functional assessment, investigations, and surgical specific risk as part of a multi-faceted model.

Many scoring systems have been proposed prior to elective surgery. An overview of the more validated systems for vascular surgery highlights the strengths and weaknesses.

Lee risk score/revised cardiac risk index (RCRI)

Introduced in 1999 as a modification of the Goldman index. Vascular surgical procedures accounted for 21% of the original cohort of patients studied (Table 3.5).

The RCRI assesses cardiac risk in non-cardiac surgical procedures. Six independent, but evenly-weighted risk factors (1 point each) are incorporated into the risk assessment model. The risk factors are:
- *CAD:* angina or previous myocardial infarction
- *Cerebrovascular disease (CVD):* history of stroke or transient ischaemic attack.

Table 3.5 The Revised Cardiac Risk Index for the prediction of cardiac risk associated with major non-cardiac surgery

Number of risk factors	Risk of adverse cardiac event (%)
0	0.4
1	0.9
2	7
≥3	11

Reproduced from Lee TH et al., 'Derivation and Prospective Validation of a Simple Index for Prediction of Cardiac Risk of Major Noncardiac Surgery', Circulation, 100, 10, pp. 1043–1049, Copyright 1999, with permission from American Heart Association and Wolters Kluwer.

- Heart failure (HF).
- Insulin-dependent diabetes mellitus
- *Impaired renal function:* creatinine >177mmol/L.
- *High risk surgical procedure:* intraperitoneal, intrathoracic, suprainguinal vascular.

Strengths
- Validated in large population including significant proportion of vascular patients.
- High capability to discriminate between patients with and without cardiac events (area under receiver operating characteristic (ROC) curve 0.81).
- Incorporates clinical risk predictors and broad type of surgery into model.

Weaknesses
- External validation studies suggest scoring system to be suboptimal for identifying patients with multiple risk factors.
- No integration of functional capacity into risk prediction model.
- Assessment of cardiac risk only.

Customized Probability Index (CPI)

Introduced in 2005 as modification of the RCRI. CPI is designed to estimate risk of all causes of mortality in vascular surgical patient population.

The CPI is a modification of the RCRI incorporating; clinical risk factors (cardiorespiratory), type of vascular surgical procedure, and concomitant medication use. A validated scoring system for different weighted variables is utilized (Fig. 3.4) to estimate the probability of all-cause mortality. A higher positive weighting of variable equates to increased risk of adverse outcome associated with that variable. For each individual a total risk score is then calculated based on the variables shown. The all-cause mortality risk (individualized percentage risk) is then obtained by utilizing a probability nomogram constructed from the study population. (see 🕮 Further reading, Kertai MD *et al*., p. 122).

Strengths
- Provides individualized quantitative all-cause mortality risk.
- Validated for vascular surgery alone, thereby homogenous population.
- Validated for elective and emergency surgery.

Weaknesses
- No external validation studies to date.
- No integration of functional capacity into risk prediction model.

AHA/ACC 2007 consensus guideline update

Whilst not strictly a scoring system the American Heart Association/American College of Cardiology consensus guidance offers a structured approach to perioperative cardiac risk and will be considered here. The Guidelines were initially introduced in 1996 with subsequent modifications published in 2002, 2007, and 2009. The guidance encompasses cardiac risk for all non-cardiac surgical procedures.

The 2007 guidance update is a very comprehensive guidance document based on best available evidence at the time of writing. It utilizes three

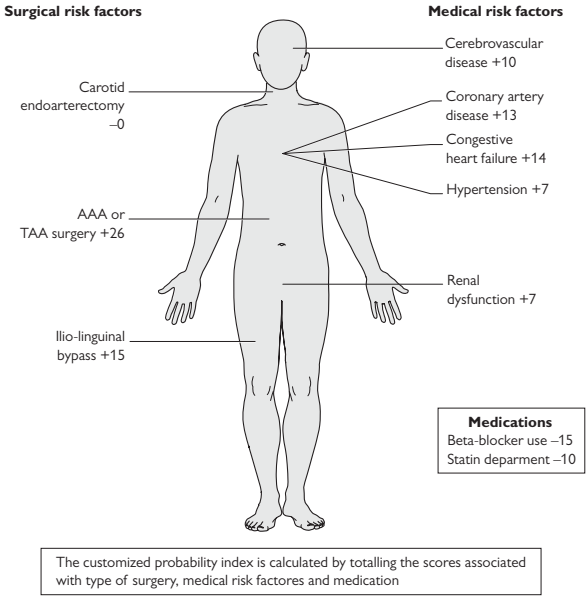

Fig. 3.4 Scores associated with customized probability index.

Data from Kertai MD et al. 'Optimizing the Prediction of Perioperative Mortality in Vascular Surgery by Using a Customized Probability Model', Archives of Internal Medicine, 2005; 165: 898–904.

variables (patient risk factors, functional capacity and severity of surgery) in assessment of cardiac risk prior to surgery (Table 3.6).

An algorithm based on three variables in Table 3.7 helps direct the user to best further management of individual patients in relation to cardiac risk management prior to surgery (see 📖 Further reading, ACC/AHA Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery, p. 122). The guidance document also provides very useful information on levels of evidence for various cardiac investigations and risk reduction strategies.

Strengths

- Comprehensive risk assessment tool based on best available international evidence.
- Multi-faceted model incorporating risk factors, nature of surgery, and functional assessment.
- Easy to follow algorithm with unequivocal pathway of management.

Weaknesses

- Assesses cardiac risk only.

Table 3.6 The structured approach to the assessment of perioperative cardiac risk described in the 2007 AHA/ACC consensus guideline update

(a) Patient risk factors

Active cardiac conditions	Clinical risk factors (RCRI factors)
Unstable coronary syndromes	History of CAD
Decompensated HF	History of HF
Significant arrhythmias	History of CVD
Severe valvular disease	Diabetes mellitus
	Renal insufficiency

(b) Functional capacity, estimated in metabolic equivalents (METs). Four METs is the ability to climb a flight of stairs without stopping. This level (≈14mL/kg/min O_2 consumption) represents the critical functional activity threshold below which cardiac risk is independently raised during non-cardiac surgery. Generally subjectively assessed.

(c) Surgery specific risk, separated into low, intermediate, and high risk. Examples for vascular surgery

Intermediate risk (cardiac risk 1–5%)	High risk (cardiac risk >5%)
Carotid endarterectomy	Aortic and major vascular surgery
(EVAR)*	Peripheral vascular surgery

*Not incorporated into 2009 AHA guidance document. However, similar European guidelines produced in 2009 by the European Society of Cardiology/European Society of Anaesthesiologists classifies EVAR as intermediate risk. ∴ Included here for complete set of vascular surgical interventions.

Data from LA Fleisher et al., 'ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery', Circulation, 116, pp. 418–499. doi: 10.1161/CIRCULATIONAHA.107.185699

- No actual quantitative assessment for individuals thereby only allowing for identification of low, intermediate or high risk
- Subjective assessment of functional capacity (METs) has been demonstrated as being inaccurate. Potentially leads to false –ve assessments with high-risk individuals being missed in the algorithm.

POSSUM score

- The POSSUM score was introduced in 1991 primarily for use in surgical audit.
- It predicts morbidity and mortality for both elective and emergency non-cardiac surgical procedures.
- The POSSUM Scoring system includes both preoperative physiological variables and intra-operative data to accurately predict outcome following surgery. Independent studies have validated use of preoperative physiological variables alone as being as predictive of adverse outcome as the total dataset. This cohort of patients included those undergoing surgery for RAAA and lower limb leg ischaemia.
- Following data entry into a computerized model, an all-cause morbidity and mortality estimate is produced.

Strengths

Preoperative scoring system validated for emergency vascular surgery.

Weaknesses

- Multiple preoperative variables required.
- Computerized data entry system required to calculate risk.
- In head-to-head studies, inferior to Hardmann Index in prediction of outcome following RAAA repair.

In summary several scoring systems have been investigated, and proposed for both elective and emergency vascular surgery. Individual scoring tools all have unique strengths and weaknesses; however, the 'ideal' risk scoring tool is yet to be established.

Other scoring systems exist, but are not within the scope of this chapter.

Emergency risk assessment

Risk assessment in the emergency setting presents different challenges for the perioperative team with often limited time available for full assessment and optimization. In circumstances where urgent surgery is required, e.g. lower limb amputation the AHA/ACC document provides very useful guidance and is recommended in this setting. It does, however, only inform the user in relation to cardiac risk.

Ruptured AAA surgery presents the most severe test in relation to risk assessment. Patients often present in hypovolaemic shock and are moribund requiring immediate surgical intervention. The challenge under these circumstances is to rapidly identify patients likely not only to survive the stress of surgery, but also be discharged from hospital with a reasonable quality of life. Any scoring system utilized for such purposes should ideally satisfy the following:

- Risk calculated on preoperative factors only.
- Factors utilized in risk assessment should be immediately available and not depend on lengthy laboratory delays.
- Easy to remember and calculate risk.
- Validated in external cohort to original study.

Validated scoring systems available to aid this process include.

Glasgow Aneurysm score

- First described in 1994. Independently validated on further occasions.
- Predicts all-cause 30-day mortality following ruptured AAA repair.

Scoring depends on weighted variables being added to formulate a total score to rapidly calculate risk:

- Age (in years).
- CVD = + 10.
- Shock = +17.
- Renal disease = +14.
- CAD = +10.

ROC analysis suggests a total score of 84 gives the best discriminatory power in predicting death postoperatively. Using this cut-off threshold the largest independent study showed mortality to be 28 and 65%, with a total score of <84 and >84, respectively.

Strengths

- Rapid and easy to formulate.
- Depends on preoperative variables only.
- Validated in several studies.

Weaknesses

- All-or-nothing cut-off threshold employed.
- Doesn't reliably identify individual high-risk patients due to its low positive predictive value.

Hardman Index

Introduced in 1996 following a study undertaken by group of Australian researchers the Hardman Index predicts all-cause mortality following surgery for repair of RAAA.

The Index was based on logistic regression analysis of 154 patients undergoing open repair of RAAA. This identified 5 factors associated with mortality:

- Age >76yrs.
- Creatinine >190mmol/L.
- Loss of consciousness.
- Haemoglobin < 9g/dL.
- ECG signs of ischaemia.

The presence of ≥ 3 risk factors was associated with 100% mortality. Zero, 1 and 2 risk factors were associated with mortality rates of 16, 37, and 72%, respectively.

Strengths

- Rapid assessment possible using preoperative variables only.
- Validated in early subsequent external cohort populations.
- Simple to apply and remember.
- Dedicated vascular model.

Weaknesses

More recent validation study has cast doubt over the claim of 100% mortality in the group with three or more risk factors, i.e. several patients survived.

Further reading

Kertai MD, Boersma E, Klein J, *et al*. Optimizing the prediction of perioperative mortality in vascular surgery by using a Customized Probability Model. *Arch Intern Med* 2005; **165**: 898–904.

ACC/AHA Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery. *Circulation* 2007; **116**: e418-e499.

Patterson BO, Holt PJE, Hinchcliffe R, *et al*. Predicting risk in elective abdominal aortic aneurysm repair: a systematic review of current evidence. *Eur J Vasc Endovasc Surg* 2008; **36**: 637–45.

Minimizing perioperative risk

For reasons outlined in the preceding sections it is imperative that, where possible, appropriate measures should be undertaken prior to surgery to minimize perioperative risk. This is a cornerstone in delivering good surgical outcomes at both institutional and national level. Early risk assessment and stratification are required in the elective setting to implement risk minimization strategies. In addition, close collaboration between relevant specialties, clear pathways of patient management and evidence-based practice will greatly enhance implementation of risk reduction strategies.

This chapter will focus on risk minimization strategies in the elective setting. The following elements of preoperative risk minimization are discussed in more detail below:

- Robust risk assessment and stratification.
- Lifestyle modification.
- Medical/pharmacological optimization.
- Interventional optimization.
- Reduction in surgical waiting time.
- Non-operative management.

Risk assessment and stratification

Accurate demarcation of individual patient risk is essential prior to major vascular surgery, and allows for subsequent general and targeted risk reduction strategies to be implemented as appropriate. The ideal environment for such assessment to take place is in the preoperative assessment clinic, at an appropriate time interval in advance of surgery. This element of patient management is discussed in detail in the preceding section.

Lifestyle modification

It is important to recommend appropriate lifestyle interventions to patients as part of any package of care aimed at reducing perioperative risk. Generally, a 4-week time window in advance of surgery is sufficient for such interventions to have clinically significant benefits. However, such advice should also be aimed at patient behaviour in the longer-term as part of 2° risk factor modification. This is discussed in more detail in 📖 Primary and secondary prevention of vascular disease, p. 14. Appropriate lifestyle recommendations to individuals preoperatively include:

- *Weight reduction in overweight individuals:* dietary advice for both overweight and underweight patients.
- *Smoking cessation:* minimum effective period 4–6 weeks prior to surgery.
- *Regular exercise:* should be within a patient's capabilities. Minimum effective period 4 weeks.

Such lifestyle interventions are an integral part of the management of cardiovascular disease and are generally suggested at first contact with individual patients, e.g. by the general practitioner (GP) or in the vascular surgical clinic. Ideally, the advice should be given several weeks before operation to allow time for maximum impact in advance of surgery. This does not remove the necessity of ensuring in the pre-assessment clinic

that the patient has received appropriate advice and checking if they acting on it.

Medical/pharmacological optimization

The management of all comorbid medical diseases should be optimized in advance of surgery to reduce perioperative risks. This constitutes 'medical optimization' and encompasses disease processes such as diabetes, chronic respiratory disease, and renal dysfunction. Specialist referral or advice may be required preoperatively to achieve this goal. Cardiac protection is discussed in detail in 📖 Dynamic testing and risk assessment, p. 136. Key aspects include:

- *Statins:* several studies have demonstrated the benefits of statins in patients at risk of and with established vascular disease.
  - In general all patients presenting for vascular surgery should be considered for statin therapy if no contraindication exists.
  - Statins should be commenced in high-risk surgery patients 7–30 days preoperatively Statins should be continued perioperatively
- *Anti-platelet agents:* commonly used agents are aspirin and clopidogrel. It is essential to balance the bleeding risks of surgery against the risk of cardiac adverse events, in deciding on management of established antiplatelet therapy perioperatively.
- *Aspirin:* it is generally recommended that low-dose aspirin therapy is continued during the perioperative period.
  - Central neuraxial blockade is considered safe in patients taking aspirin.
  - Consideration should be given to actively commencing low-dose aspirin preoperatively in individuals who have an indication for this drug and have not previously been established on treatment.
- *Clopidogrel:* the anti-platelet action of clopidogrel is irreversible and more profound than that of aspirin. Evidence from large RCTs in relation to clopidogrel in the perioperative period is limited. Consensus opinion recommends cessation 7 days prior to surgery; however, if there is a strong 1° indication for clopidogrel, in particular the presence of coronary stents, this may need to be modified.
- *β blockers:* several studies have demonstrated cardioprotective effects of recently introduced β blocker therapy in perioperative period. However, more recently, this strategy has caused international concern in relation to increase in all-cause mortality. A more targeted patient approach is now recommended in perioperative period:
  - Continuation in patients presently treated with β blockers for ischaemic heart disease (IHD), arrhythmias, HF, or hypertension.
  - Introduction of titrated β blocker therapy in patients undergoing vascular surgery who are at high cardiac risk due to underlying IHD or the presence of cardiac ischaemia on preoperative testing.
  - Introduction of titrated β blocker therapy in patients undergoing vascular surgery who are identified as being at high cardiac risk preoperatively due to the presence of >1 revised cardiac risk factor is recommended by the ESC, but not the ACC/AHA guidelines.

  - It is important to avoid prolonged periods of perioperative hypotension as this may predispose patients to an increased risk of stroke.
- *Other medication:* in general, all other established medication should be continued in the perioperative period. Exceptions to this rule exist, including drugs such as warfarin, where continuation may predispose patients to undue risk of adverse events.

Interventional optimization

Cardiac revascularization prior to vascular surgery is now rarely performed. Large prospective studies have demonstrated no outcome benefit in cardiac revascularization prior to vascular surgery, in patients with chronic stable angina. It is therefore not recommended to seek a specialist cardiology opinion in such individuals prior to non-cardiac surgery.

Despite this, circumstances exist where cardiology referral is required in advance of vascular surgery. Patients who have a 1° indication for cardiac intervention or who have poorly-controlled cardiac disease fall into this category. These situations include:

- Symptoms of unstable angina (Canadian classification III and IV).
- Deterioration in symptoms in previously well controlled angina.
- Clinical evidence of decompensated HF.
- Preoperative diagnosis of critical or symptomatic valvular heart disease.
- High grade arrhythmia, e.g. complete heart block, poorly-controlled tachyarrhythmia.

Under these circumstances, individuals may require an interventional cardiac procedure in advance of vascular surgery in order to afford risk reduction. Such procedures include balloon angioplasty, percutaneous stent insertion (PCI), coronary artery bypass grafting (CABG), cardiac valve replacement, or cardiac conduction pathway ablation. Following PCI patients are placed on single or dual anti-platelet therapy to reduce risk of graft or stent thrombosis. Recommendations regarding management of antiplatelet therapy following coronary intervention are given in 📖 Dynamic testing and risk assessment, p. 136 and 📖 Coronary artery disease, p. 164.

Reduction in surgical waiting time

Where possible the timeframe from referral to surgery should be minimized in order to reduce risk from vascular co-morbidity. This situation particular pertains to carotid and aortic aneurysm surgery, in an effort to avoid risks of stroke and aneurysm rupture, respectively. National targets for these two indexed procedures have recently been introduced:

- *Carotid surgery:* ideally endarterectomy should be performed within 2 weeks of symptoms. Generally surgery >12 weeks following symptoms is no longer recommended.
- *Aortic surgery:* recently introduced national screening programme recommends a target of 8 weeks from diagnosis to surgery. Similar time frames are recommended for non-screened patients by the national aortic aneurysm quality improvement framework.

Clearly both situations represent the ideal standard, which may be difficult to achieve for all individuals presenting for vascular surgery.

Non-operative management

Situations may exist where the risks of the proposed vascular surgical procedure may exceed risks of conservative management. Under such circumstances it may be in the patient's best interests to be managed conservatively or with best medical therapy. Such decisions should ideally be made following both multi-disciplinary and fully-informed discussion with the individual involved.

Further reading

Fleisher LA, Beckman JA, Brown KA, *et al*. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). *Anesth Analg* 2008; **106**: 685–712.

Poldermans D, Bax JJ, Boersma E, *et al*. Guidelines for pre-operative cardiac risk assessment and perioperative cardiac management in non-cardiac surgery. *Eur Heart J* 2009; **30**: 2769–812.

Clinical cardiological evaluation of the vascular surgery patient

The purposes of the cardiovascular history and examination are to:

- Identify previously undetected cardiovascular disease and direct further clinical investigations.
- To determine if any cardiovascular disease that is present is optimally treated.
- To inform preoperative risk assessment.

Preoperative assessment of cardiovascular risk takes into account:

- The extent and severity of cardiovascular co-morbities.
- The functional capacity of the patient.
- The magnitude of the planned surgery and, by implication, the severity of the physiological insult associated with the surgery.

The history and examination contribute to the assessment of all of these.

History

The cardiovascular history should include:

- *Detailed history of any cardiovascular events suffered by the patient:* including, acute coronary syndromes, cerebrovascular events, and HF.
- *Details of when the patient suffered any cardiovascular events:* acute coronary syndrome or cerebrovascular event in the past few weeks or months may be associated with greater risk than one that happened several years ago.
- *Details of current cardiovascular symptoms:* including angina, breathlessness, claudication, and transient ischaemic events.
- *Details of current and past treatments:* judgement must be made as to whether any cardiovascular disease is optimally treated.
- *Risk factors* that may influence the likelihood of underlying cardiovascular disease as well as indicating its presence, such as:
  - Smoking.
  - Diabetes mellitus.

  - Renal impairment.
  - Hypertension.
  - Dyslipidaemia.
  - Family history of cardiovascular disease.
- *Presence of a pacemakers or implantable defibrillator:* knowledge of type of device, pacing dependency, and ability to use electrocautery can affect operative procedure and suggest requirement for peri-operative alterations in pacemaker settings (see 📖 Arrhythmias, pacemakers and implantable cardioverter/defibrillators, p. 178).

It is important to identify unstable cardiovascular conditions as these greatly increase perioperative risk. Perioperative risk associated with unstable cardiac disease generally outweighs benefits of elective surgery, which should be deferred to allow treatment of the cardiovascular condition. It may also be appropriate in many cases to defer or modifiy urgent surgery. It is important to exclude:

- Decompensated heart failure (NYHA class IV—dyspnoea at rest, new onset or worsening heart failure).
- Haemodynamically significant arrhythmias (high grade AV blocks, ventricular arrhythmias, supra-ventricular arrhythmia with uncontrolled heart rate >100 beats/min).
- Cardiac ischaemia (unstable angina or patients with poor functional status and ischaemic heart disease, who may have critical ischaemia).
- Recent (<30 days) acute coronary syndrome (ACS).
- *Haemodynamically significant valve disease:* including:
  - Aortic valve (AV) stenosis with symptoms or a mean gradient >40mmHG or AV area <1 square cm.
  - Symptomatic mitral stenosis.

The clinical circumstances influence the extent to which patient specific information can be obtained or acted upon. For example, in urgent surgery, whilst the patient's history may have little impact upon the timing, it may alter the choice of intervention. In more elective cases, life expectancy suggested by co-existent cardiovascular disease may suggest that certain types of surgery (i.e. aortic aneurysm repair) may not be expected to prolong survival.

Taken together the patient specific and surgery specific factors allow perioperative risk to be estimated and considered in the decision making processes proceeding surgery.

Whilst baseline demographics, especially age, will influence all risk calculations, the increasing number of urgent operations and prevalence of co-morbidities limit their predictive ability. In the USA, the number of operations undertaken in patients over 75yrs of age increased by 25% between 1995 and 2005. Primary care data from the UK suggests that the prevalence of cardiovascular disease is 19% for men and 12% for women aged between 75 and 84yrs, suggesting that a large proportion of surgical patients will have underlying CAD.

Functional capacity

Functional capacity can be estimated from patient's ability to perform activities of daily living, e.g. being able to walk up two flights of stairs or walk up-hill would suggest a functional capacity greater than 4 METs. Whilst this

alone is a poor discriminator of post-operative cardiac outcome, if the patient has good functional capacity, perioperative management is unlikely to be influenced by further cardiac investigations. However, if the patient's functional capacity is poor or uncertain, functional testing (discussed in 📖 Decision making in vascular surgery, p. 95) may be used to gain objective information on physiological reserve, especially if other aspects of history suggest cardiovascular disease may be present.

Type of surgery

It is important to achieve clarity regarding what operation is planned or what surgical options are being considered. To do this the anaesthetist may need to glean information from the notes and the patient. If there is uncertainty then it is important to speak to the surgical team. The magnitude of the planned surgery determines the severity of associated physiological insult and, in particular, the systemic inflammatory response (SIRS) to surgery. Table 3.7 place the risks associated with vascular operations in the context of other types of surgery. It will be seen that both open aortic surgery and peripheral vascular surgery are considered high risk. In the case of aortic surgery, this is due to haemodynamic stress of surgery and severity of SIRS response. Patients who require surgery for peripheral vascular disease generally have extensive atheromatous disease and this places them at high risk of perioperative cardiovascular complications. CEA carries risk of stroke and haemodynamic complications, but is a superficial operation not associated with major SIRS response. As such, it is classified as an intermediate risk operation. Urgency of surgery is an important consideration; urgent or emergency surgery is associated with much greater risks than elective surgery.

Examination

A directed clinical examination may offer additional evidence of cardiovascular disease whose presence is suggested by the history. It is important to identify or exclude:

Table 3.7 Peri-operative risk and type of surgery (MI and cardiac death within 30 days after surgery)

Low risk <1%	Intermediate risk (1–5%)	High risk >5%
Breast	Abdominal	Aortic/vascular
Dental	Carotid	Peripheral vascular
Endocrine	Peripheral PCI	
Eye	Endovascular repair	
Gynaecology	Head and neck	
Reconstructive	Orthopaedic major	
Orthopaedic minor	Neurological	
Urological minor	Urological major	Pulmonary/renal/liver transplant

- Heart rate and rhythm abnormalities, in particular poorly-controlled atrial fibrillation with a significant apex/radial deficit or a significant increase in heart rate on mild or moderate exertion.
- Pulse or BP differences between the right and left arms. (Cardiovascular disease in the arm vessels may lead BP readings on both direct and indirect BP monitoring that are significantly below the true systemic pressure.)
- Cardiac murmurs.
- Evidence of pulmonary oedema on auscultation.
- Peripheral oedema or an elevated jugular venous pressure.
- Previously unidentified carotid bruits.
- Residual neurological deficit in patients with a history of CVD.

If patient has been noted to have raised BP, it may be appropriate to repeat BP reading, ensuring patient is sitting quietly in a chair with arm supported and is not engaged in conversation. Ideally, BP should be taken by auscultation, especially if the patient is in atrial fibrillation.

The discriminatory ability of clinical signs is poor, even to more experienced examiners. Evidence of cardiovascular disease detected on clinical examination should be confirmed by further investigations. For example, presence of a clinically suspicious murmur should be investigated by echocardiography.

Preoperative risk assessment

A number of tools are available to assess the perioperative risk of cardiovascular complications, cardiac death, or all-cause mortality. These are dealt with in detail in 📖 The vascular preoperative assessment clinic, p. 110, and include the Lee Revised Cardiac Risk Index and, in the case of emergency surgery, the Glasgow Aneurysm Score and Hardman Index. It is important to note that all of these tools give considerable weight to information gleaned directly from the patient, rather than from investigations. An accurate and comprehensive history and examination are essential for reliable preoperative risk assessment.

Cardiological investigations in the vascular surgery patient

The selection of preoperative cardiological investigations should be informed by the history and examination. Preoperative investigations are performed to:

- Test differential diagnoses suggested by the history and examination.
- Quantify the severity of disease, e.g. the severity of impairment of left ventricular function.
- Inform preoperative risk stratification, e.g. by demonstrating impaired glucose tolerance or renal impairment.
- To screen for medical conditions.

Preoperative cardiological investigations are directed towards the identification of myocardial ischaemia, LV dysfunction, and significant valvular

disease. Preoperative screening in people who are healthy (apart from their indication for surgery) is generally of limited value. The low prevalence of intercurrent disease means that many +ve test results are, in fact, false +ves. However, people who require arterial surgery, by the nature of their complaint, have identified themselves as having arterial disease, and routine preoperative screening tests are more likely to yield true +ve results.

Blood tests

Appropriate preoperative blood tests in patients presenting for vascular surgery include:

- *Full blood count:* knowledge of the preoperative haemoglobin is important in patients who have active cardiovascular disease and are to undergo surgery that may be associated with significant blood loss. The detection of anaemia is important given that an haematocrit of less than 28% is associated with increased peri-operative risk.
- *Urea (blood urea nitrogen), creatinine and serum electrolytes:* patients with vascular disease may have renal impairment or HF, and are frequently taking drugs (e.g. ACE inhibitors and diuretics) that can produce electrolyte abnormalities.
- *Platelet count, activated partial thromboplastin time (APPT) and international normalized ratio (INR):* – Whilst preoperative coagulation studies are not essential in all vascular surgery patients, a significant proportion will be receiving treatment with warfarin (most frequently for atrial fibrillation) or heparin (often for the same thromboembolic disease that has brought them to surgery.
- *Blood transfusion:* dealt with in 📖 Intravenous fluid therapy and blood product management, p. 228. However, it is worth noting here that patients undergoing arterial surgery with a significant chance of bleeding require a blood sample to be taken for, at the very least, group and antibody screen.

The value of testing for cardiac biomarkers in the preoperative period is limited, unless there is a specific indication for testing prior to surgery their value.

- *Cardiac troponins and C-reactive protein:* it may be anticipated that, in keeping with the general population, surgical patients with an elevated baseline high sensitivity C-reactive protein (hsCRP) or elevated cardiac troponin I, detected using the ultra-sensitive assays that are now available will be at increased risk of peri-operative cardiac complications. Studies to confirm this are awaited.
- *Brain natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP):* BNP and NT-proBNP are released from myocytes in response to increased wall stress and have been demonstrated to be prognostic markers in heart failure, ACS, and stable IHD. Small studies have suggested benefit in predicting cardiac events and post-operative mortality following non-cardiac vascular surgery. Their routine use has yet to be established and is not widely advocated at present.

Electrocardiography

The routine use of electrocardiograms is a matter of debate, with studies suggesting the ability of any ECG abnormality to discriminate peri-operative risk for patients undergoing low to intermediate risk surgery being small (absolute difference only 0.5%). The ECG may be of help in identifying those patients in whom history and examination suggested were at increased cardiovascular risk. Less than 7% of patients with impaired left ventricular systolic function to have a normal ECG, although the ECG may be normal even with severe CAD.

The current recommendation is to review a pre-operative ECG in the following patient groups:

- Cardiac risk factors and intermediate or high risk surgery.
- No risk factors and high/intermediate risk surgery.
- Consider if risk factors and low risk surgery.

It will be appreciated that almost all patients presenting for arterial surgery will have one or more indications for a preoperative ECG. The need for vascular surgery marks them out as having cardiovascular disease, and many will require intermediate or high risk surgery.

Echocardiography

Transthoracic echocardiography (TTE) can be used to:

- Detect structural abnormalities.
- Detect valvular disease.
- Assess baseline resting left ventricular systolic and diastolic function.
- Detect wall motion abnormalities.

If valve disease is suspected or known an echocardiogram should be performed to assess severity and haemodynamic effects. In patients undergoing high risk surgery it may influence the peri- and post-operative level of monitoring, especially if significant fluid volume shifts are expected. It is important not to overestimate the predictive value of resting echocardiography. A meta-analysis demonstrated that a reduced ejection fraction (<35%) achieved a sensitivity of only 50% in predicting peri-operative non-fatal cardiac events or death. A possible reason for this is that baseline resting scans give no information as to the presence or extent of CAD.

Current recommendations are to conduct preoperative echocardiography in the following patient groups:

- Patients with known or suspected significant valve disease who have not undergone a recent echocardiogram.
- Patients with dyspnoea of unknown origin.
- Echocardiography may be considered in patients undergoing high risk surgery. In this case, a significant abnormality on echocardiography identifies the patient as being at increased peroperative risk. A normal echocardiogram does not preclude significant cardiac disease.

Non-invasive testing for myocardial ischaemia

As has already been noted for echocardiography, the fact that a cardiac investigation is normal at rest does not preclude the presence of significant cardiac disease. In patients who have significant CAD, coronary blood flow may be sufficient meet myocardial oxygen demand at rest, but not as

cardiac work increases in response to the stress of surgery. A number of non-invasive tests of cardiac function are available. These examine cardiac performance in the face of an increasing workload allowing ischaemia that becomes manifest with increasing cardiac work to be identified. These tests are considered below. (Cardiopulmonary exercise testing (CPX), which is an integrated test of cardiac and respiratory function is considered in 📖 Dynamic testing and risk assessment, p. 136, and is contrasted with dobutamine stress echocardiography.)

Exercise ECG testing

The exercise ECG test is very widely used in cardiology as a tool for the risk stratification of patients who present with a history or symptoms consistent with myocardial ischaemia. The patient exercises on treadmill, whilst undergoing 12-lead ECG monitoring with ST-segment analysis of the ECG. A standard protocol is used to increase the workload steadily over time and the test is terminated at exhaustion, or when the patient develops symptoms or evidence of significant myocardial ischaemia. Exercise testing gives objective information on functional capacity and enables the detection of presence of stress-induced myocardial ischaemia with ST segment changes. It also allows the assessment of the heart rate and BP responses to stress and may allow the identification of a heart rate above which myocardial ischaemia becomes evident, the so called 'ischaemic threshold'.

A meta-analysis using treadmill testing in the setting of vascular surgery demonstrated a sensitivity of 74% and specificity of 69% for the detection of perioperative cardiovascular complications. Whilst the positive predictive value was low (10%) the –ve predictive value was useful at 98%, i.e. many patients with abnormal tests will not suffer perioperative complications, but a normal test is reassuring to both clinician and patient. The functional level at which ischaemia was precipitated was predictive of the level of risk. Some vascular surgery patients are unable to exercise to a sufficient level to perform a useful test, most frequently due to claudication.

Myocardial perfusion scanning

This technique uses a gamma emitting radiolabelled tracer drug, such as Technetium (99mTc) tetrofosmin, to map myocardial perfusion.

- Cardiac stress may be induced by exercise.
- Pharmacological stressor agents such an adenosine or dobutamine are often used as an alternative to exercise making the test particularly useful in patients with diminished exercise capacity.
- By comparing the distribution of the isotope in the myocardial at rest and during stress defects in myocardial perfusion are identified and classified as fixed (indicative of prior myocardial damage) or reversible (inducible ischaemia).
- Reversible defects can be semi-quantified to give an indication as to amount of myocardium at risk either in terms of segments or volume affected.
- A meta-analysis of studies in vascular patients found that small areas of ischaemia (<20% of myocardial volume) did not affect outcome compared with normal scans.

- Larger defects (>50% reversibility) were associated with higher peri-operative risks (11% increased risk).
- Further studies suggested a sensitivity of 83% and specificity of 47% for the prediction of perioperative cardiac complications.
- As with the exercise ECG, the test had a low positive predictive value (11%), but a reassuring negative predictive value of 97%.

Stress echocardiography

In stress echocardiography, the heart is imaged whilst being stressed with exercise or more commonly with dobutamine. Whilst providing the same baseline data on left ventricular and valve function as resting transthoracic echocardiography, stress echocardiography enables the identification of inducible ischaemia, and an examination of the haemodynamic effects of valve stenoses with increasing heart rates. (The quantification of inducible myocardial ischemia using echocardiography is described in 📖 Dynamic testing and risk assessment, p. 136, where the investigation is contrasted with cardiopulmonary exercise testing.)

The presence of myocardial ischaemia (manifested as new wall motion abnormality) at a heart rate of <60% of the age-predicted maximal heart rate is associated with an increased risk of perioperative cardiac complications. Patients with an ischaemic threshold <60% maximum predicted heart rate for age are reported to have a post-operative cardiac event rate of 43% compared with 9% if the ischaemic threshold is less than 60%> and 0% if stress echo is normal. The sensitivity of stress echocardiography for the prediction of perioperative cardiac complications is estimated to be 85% with specificity of 70%. Again, the test has a high –ve predictive value of between 90 and 100%.

MRI stress perfusion imaging

The MRI investigation of myocardial ischaemia is a growing area, although no pre-operative studies have reported to date. Wall motion abnormalities used as a surrogate for ischaemia had a sensitivity of 83% and specificity of 86%. The use of perfusion data increased the sensitivity to 91%, although specificity fell to 81%.

CT coronary calcium scoring and CT coronary angiography

CT scanning can be used to detect the presence of coronary artery calcification and can be used to assess the patency of coronary arteries with a specificity of 74% and sensitivity of 96%. At present CT coronary angiography is used for the exclusion of significant coronary disease in low risk individuals. At the time of writing no data are available on the use of this test for pre-operative assessment.

The utilization of static tests

The principle remains that testing should only be undertaken if it results in changed peri-operative management. It may be considered in patients undergoing higher risk surgery with low number of clinical risk factors.

A number of guidelines suggest functional testing based on the magnitude of the planned surgery and the presence of cardiac disease or cardiac risk

Box 3.1 Cardiac risk factors. The presence of two or three of the factors listed may support preoperative dynamic cardiac testing

- History of angina pectoris
- History of myocardial infarction
- History of heart failure
- History of stroke or transient ischaemic attack
- Diabetes mellitus requiring treatment with insulin
- Renal dysfunction (serum creatinine >170μmol/L)

factors (Box 3.1). The following groups of patients are candidates for pre-operative functional testing:

- High risk surgery with >3 clinical risk factors.
- Consider in high risk surgery <3 clinical risk factors.
- Active cardiac conditions in elective surgical patients, e.g. new onset or poorly-controlled angina.

Testing may be considered in intermediate risk surgery if cardiac risk factors are present. There is no evidence to support preoperative dynamic cardiac testing in low risk patients. Indeed, there is some evidence that it may be associated with harm.

Further reading

Priebe H-J. Preoperative cardiac management of the patient for non-cardiac surgery: an individualized and evidence-based approach. *Br J Anaes* 2011; **107**: 83–96.

Wijeysundera DN, Beattie WS, Karkouti K, *et al*. Association of echocardiography before major elective non-cardiac surgery with postoperative survival and length of hospital stay: population based cohort study. *Br Med J* 2011; **342**: d3695.

Preoperative coronary angiography and revascularization

Whilst the majority of patients who require arterial surgery will have coronary atheroma, preoperative coronary angiography is indicated in only a small minority of patients. Invasive coronary angiography should be considered in accordance with the algorithms for investigation of CAD, as opposed to specifically for pre-operative investigation. Perioperative cardiac events may be due to myocardial oxygen supply demand imbalance or perioperative coronary artery atheromatous plaque rupture with coronary thrombosis or embolism. Coronary angiography, whilst seen as the gold standard way of assessing the presence of coronary atheroma is poor at predicting the presence of ischaemia or the likelihood of a vulnerable plaque. Thus, coronary angiography does not have especial predictive value in surgical patient and the indications for pre-operative angiography are the same as for angiography in the non-surgical setting.

Recommendations for preoperative angiography

- Recommended in patients with ACS (ST elevation myocardial infarction (STEMI), non-ST segment elevation myocardial infarction (NSTEMI), unstable angina).
- Recommended in patients with angina not controlled with medical therapy.
- Presence of large areas of reversible ischaemia, which may benefit from stabilization pre-operatively.

Coronary angiography may be appropriate in patients with IHD presenting for high risk or intermediate risk surgery. It is not recommended in patients with IHD about to undergo low risk surgery.

Coronary revascularization

The objective of revascularization is the reduction of ischaemic symptoms and improvement of prognosis in patients with high-grade stenoses subtending large areas of ischaemic myocardium. However, it is estimated only 50% of peri-operative myocardial events are related to pre-existing high grade lesions the rest arising from vulnerable plaque rupture frequently occurring on lesions that would not induce functional ischaemia prior to plaque rupture. This pathophysiological mechanism accounts for the poor specificity of pre-operative functional imaging techniques.

Currently, revascularization should be only considered in accordance with the usual practice for patients with valve or coronary disease. The only exception is the consideration of earlier valve intervention in patients who would require intervention in the foreseeable future, but in whom the presence of a non-cardiac indication for surgery exists, which could be safely delayed until following the valve intervention.

Trials that have evaluated use of prophylactic revascularization have done so in the setting of non-cardiac vascular surgery. Irrespective of whether the patients randomized to revascularization were high risk (three-vessel disease, with many having reduced LV function <35%) or a lower risk population, there was no difference in peri-operative MI rates or mortality. It should be noted, however, that on follow-up those not revascularized had a higher rate of subsequent revascularization. ∴ Myocardial revascularization may be considered in accordance to the current guidelines on coronary revascularization in patients with the requirement for non-cardiac surgery that could be safely deferred.

Recommendations for preoperative revascularization

- May be considered prior high risk surgery in patients with proven IHD who are candidates for revascularization under current guidelines irrespective of the planned non-cardiac surgery. The following groups of patients with stable IHD may be candidates for revascularization:
  - Stable angina and left main stem disease.
  - Stable angina and 3-vessel disease (especially if left ventricular ejection fraction (LVEF) <50%).
  - Stable angina and 2-vessel disease (with proximal left anterior descending (LAD) (coronary artery) and LVEF <50%).
- Not recommended prior to intermediate or low risk surgery.

- Usefulness not well established for patients with documented ischaemia on non-invasive testing prior to non-cardiac surgery.

The urgency of surgery is an important consideration.

- Coronary angiography is performed in patients who may benefit from coronary revascularization.
- The option of preoperative revascularization is only possible where surgery can safely be delayed, either to allow recovery from cardiac surgery or until the patient no longer requires dual antiplatelet therapy after coronary stenting.
- The type of revascularization (PCI or CABG) is dependent on the anatomical features of stenoses within the coronary arteries and patient specific factors, such as co-morbidities (re-stenosis rates are higher with stents in diabetics).

Unstable ischaemic heart disease requiring prophylactic revascularization

In the case of elective surgery and many types of urgent surgery, presentation with an acute coronary syndrome would take precedence over the surgical complaint, in terms of risk and treatment, in accordance with national guidelines regarding coronary revascularization. In these cases, as the majority of patients will be undergo coronary revascularization. Preference should be given to percutaneous coronary intervention (PCI) using bare metal stents (BMS), rather than CABG to minimize any delay to non-cardiac surgery. Should the patient present with an ACS complicating an urgent, life-threatening situation, then surgery should take precedence with coronary revascularization and further risk stratification considered in the post-operative setting.

Further reading

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Dynamic testing and risk assessment

Introduction

In agreeing to surgery, the patient accepts a risk of harm from the procedure in order to gain a longer-term benefit in survival or quality of life. The vascular anaesthetist has an important role to play in risk assessment prior to surgery. This includes not only assessment of the risk of intraoperative complications, but also consideration of the risk of complications after surgery, long-term survival, and quality of life.

This section reviews the use of dynamic cardiac tests in the evaluation of high risk vascular surgical patients. Dynamic tests of cardiovascular function involve either physical exercise or pharmacological stimulation to increase heart rate and cardiac contractility.

Both in the perioperative setting and, more generally, there are four reasons for performing a clinical test:

- To obtain information to diagnose a disease or illness.
- To obtain information to give a prognosis on the outcome of a disease or illness.
- To evaluate the effects of a treatment on a disease or illness.
- To evaluate the side effects of a treatment.

It is important to be clear about the purpose of any test. Is the aim to quantify perioperative risk, or to evaluate cardiac disease and respiratory disease?

Tests performed to evaluate cardiac or respiratory disease should be interpreted in the context of the literature and guidelines for the management of such disease.

In many cases, the purpose of investigations will be to evaluate cardiac disease and quantify perioperative risk. A number of guidelines for the use of cardiac tests to evaluate non-cardiac surgery patients have been published. It is important to understand that these guidelines are not about recommending which tests to use to assess vascular or cardiac disease. They offer guidance on which patients may have their perioperative management changed by cardiac investigations, what tests can be used, and on interventions to modify perioperative cardiac risk. The results of preoperative risk assessment are also used to advise patients on the relative risks of accepting or declining surgery.

Walking and activity tests

Taking a history of exercise tolerance is a standard question in preoperative assessment. This is often a description of distance walked or stair climbing. The evidence base for its value in prognosis is limited outside thoracic surgery. Reasons for this include:

- Subjective assessment.
- Submaximal exercise.
- No assessment of power.
- Patient bias depending on their desire to have or decline surgery.

Structured questionnaires, such as the Duke Activity Status Index, can be used to quantify fitness based on an individual's functional activity. These assign a level of fitness based on METs. One MET is 3.5mL O_2/kg/min). There is a correlation between the results of structured questionnaires and formal measurement of oxygen consumption upon exercise. Tools such as the Duke Activity Status Index are good at indicating extemes of fitness and unfitness, but less good at providing evidence for prognosis in surgical patients.

Direct assessments of exercise capacity can be made with tests based on walking. The simplest of these is the 6-min walk test in which a patient walks up and down a measured line for 6min and total distance walked is recorded. This is a submaximal test and of limited value in vascular anaesthesia.

The incremental shuttle walking test (ISWT) is of greater value. Two cones are placed 10m apart and the patient walks around the cones at an increasing speed. The speed is dictated by a headphone or timer emiting a beep at the point when the patient is meant to reach a cone. This becomes progressively faster as the test goes on. This is a good test of aerobic fitness. Its limitation is in the patient who finds turning around at the cones safely difficult at increasing speeds and may stop early because of the risk of falling.

Dobutamine stress echocardiography (DSE)

In this test the patient is monitored with a 12-lead ECG, non-invasive BP monitor, and a 2D transthoracic echocardiography probe. An infusion of dobutamine is administered via a peripheral IV line starting at 5μg/kg/min. This is increased every 5min by 5μg/kg/min to reach a maximum of 40μg/kg/min. The test endpoints are:

- Reaching the maximum dose of dobutamine.
- A heart rate >120beats/min for patients >65yr of age and >135beats/min for patients <65yr.
- Limiting chest pain.
- Headaches, severe nausea, vomiting.
- 2mm of ST elevation or depression compared to baseline in 2 leads.
- Hypotension (systolic BP <90 mmHg).
- Hypertension (systolic BP >240 mmHg).
- Ventricular tachycardia or sustained supraventricular tachyarrhythmias.
- If the maximum heart rate is not reached a 0.2-mg bolus of atropine can be given up to a maximum of 2g.

The echocardographic images of the left ventricle are recorded after each increase in the dobutamine infusion. Six echocardiographic views (parasternal long and short axis, and apical 4-chamber, 2-chamber, and long and short axis) are videotaped at rest, and each dose of dobutamine and atropine. To analyse the test the left ventricle is divided into 16 segments. Each segment is evaluated using the following scoring system (1, normal; 2, mild hypokinesis; 3, severe hypokinesis; 4, akinesis; and 5, dyskinesis).

- *Normal* is defined as normal wall thickening.
- *Hypokinesis* is defined as reduced wall thickening.
- *Akinesis* is defined as the absence of wall thickening.
- *Dyskinesis* is defined as paradoxical excursion away from the lumen and systolic wall thinning.

A wall motion score index is calculated at each stage of the test. An increase in abnormal wall motion is associated with IHD or HF.

Cardiopulmonary exercise testing (CPET)

In this test, the patient exercises on a bike or treadmill, and is monitored with a 12-lead ECG, a non-invasive BP monitor, and a mouthpiece or face mask to sample inspired and expired air to measure breath by breath O_2 consumption and CO_2 excretion. In addition, power, heart rate, tidal volume, and respiratory rate are measured.

There are many different exercise protocols used in respiratory and sports medicine, but the most commonly used protocol in the UK for perioperative risk assessment uses:

- An electronically braked cycle ergometer.
- 1–2min of data collection with no exercise.
- 3min cycling against no resistance.
- A gradual or ramped increase in work to turn the pedals set at 10W/min. For some patients with increased levels of fitness, ramp may be set higher.
- Test is terminated by patient when they feel they can do no more exercise (symptom-limited test) or stopped if risks of test outweigh benefits of data obtained from the test.

Measurements that can be made from the CPET

- Peak O_2 consumption and peak power (the highest values recorded during the test).
- Anaerobic threshold (the oxygen consumption when the patient moves from predominantly aerobic metabolism into a mixture of aerobic and anaerobic metabolism).
- Oxygen pulse (a measurement that reflects stroke volume of the heart).
- *Ventilatory equivalents for carbon dioxide and oxygen:* these indicate how hard the patient has to breath to clear carbon dioxide or take in oxygen (these increase in pulmonary vascular disease and HF).

Integrating dynamic testing into prognosis: an empirical approach

The emphasis of preoperative evaluation is shifting from diagnosis of co-morbidity to prediction of perioperative adverse events to a broader view that includes consideration of both short- and long-term outcome. CPET and DSE contribute useful information in addition to that gained from clinical assessment.

- CPET quantifies physical fitness.
- DSE quantifies myocardial wall-motion abnormality (WMA).
- CPET discriminates survival in any population, both those with and without WMA, whereas DSE does not discriminate survival differences in patients without WMA.

Both patients and clinicians find it useful to be given a quantitative estimate of perioperative risk. The author and his colleagues have developed an empirical approach to furnishing such estimates based on actuarial data and insights from studies of the impact of fitness, cardiovascular disease, and surgery on survival. This approach is outlined below. Whilst not yet validated in a large prospective study the author has found the following approach, based on life tables and epidemiological data, to yield clinically useful results

The starting point for the estimation of both short- and long-term risk is the fact that survival depends upon age, sex, and physical fitness. For any population, actuarial life tables can provide information on the life-expectancy of a person of a given age. For the UK population, these data are available at www.gad.gov.uk.

Having established the patient's average risk of death, the next stage is to determine how average the patient is, i.e. the extent to which their risk of death is modified by their physical fitness, co-morbidity, and planned surgery.

The impact of fitness on survival*

The average peak METs that an individual of a given age and sex can achieve can be predicted from established formulae:

- 18.4 – (0.16 × age) for men.
- 14.7 – (0.13 × age) for women.

Large series have demonstrated that age-specific mortality risk increases as fitness falls. Each MET shortfall increases mortality risk by 15% compared with average. A 2-MET fitness shortfall increases risk by 1.15 × 1.15 = 1.32, equal to the average risk of someone 3.5yrs older.

Brain, heart, kidney, and PADs further increase mortality risk. Empirically, death rates calculated on the basis of age, sex, and fitness are multiplied ×1.5 each for peripheral arterial disease, old myocardial infarction and old strokes (>2yrs ago). More recent acute coronary syndromes or strokes increase risk, three-fold if 1yr ago, six-fold at 6 months and 13-fold at 3 months.

Renal dysfunction is reliably and independently correlated with increased long-term mortality. Mortality risk increases 0.85% for each 1mL fall in estimated glomerular filtration rate (eGFR). eGFR can be calculated from creatinine concentration as follows:

- $32788 \times [\text{creatinine } (\mu\text{mol/L})]^{-1.154} \times \text{age}^{-0.203}$ for men.
- $32788 \times [\text{creatinine } (\mu\text{mol/L})]^{-1.154} \times \text{age}^{-0.203} \times 0.742$ for women.

On average a 67-yr-old man would be expected to have an eGFR of 73mL. An eGFR of 56mL in such an individual would be associated with and increased mortality risk of $1.0085^{(73-56)} = 1.15$.

The calculations described above are combined in an online risk calculator which is available at https://sites.google.com/site/informrisk/.

Estimating the baseline risk of death regardless of surgical risk as precisely as possible is important before AAA repair. Mortality risk increases 14-fold in the month after open repair and six-fold after EVAR. People with a baseline risk of death greater than 1 in 60/month do not reap any survival benefit from repair of a 6.4-cm AAA. The risk of surgery is likely to exceed benefit in people with a 6.4-cm AAAs if their baseline risk of death is greater than 1 per 150/month as survival is usually accompanied by morbidity.

CPET and mortality risk assessment

Large observational studies have demonstrated a reliable association between exercise tolerance and life expectancy. While an estimate of the patient's peak oxygen consumption to determine peak MET can be made on the basis of self-reported exercise tolerance or from a shuttle walk test a CPET can directly measure peak oxygen consumption.

Peak MET or O_2 consumption (peak VO_2) incompletely captures risk associated with unfitness. Using at least one other value derived from CPET improves prognostic precision in most populations. The ventilatory equivalent for CO_2 (V_E/VCO_2), which is the amount one has to breathe to achieve CO_2 exchange, is useful in this regard. Age-adjusted risk calculated with METs increases as V_E/VCO_2 increases. MET-risk should be increased by

* The epidemiological model described below was supplied by Dr. John Carlisle.

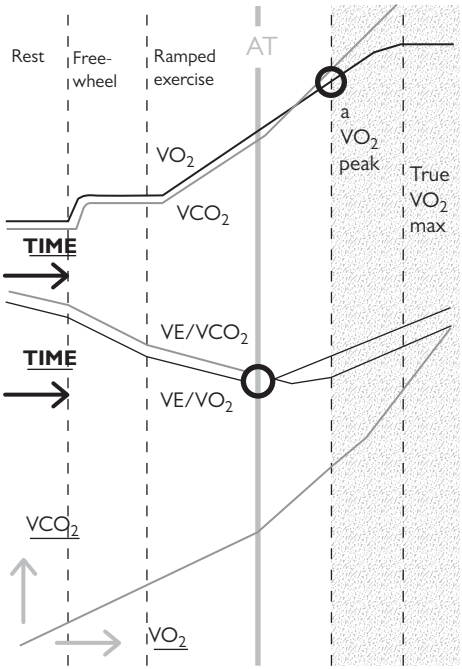

Fig. 3.5 Schematic diagram showing the changes in oxygen consumption, carbon dioxide production and ventilatory equivalents for oxygen and carbon dioxide during a cardiopulmonary exercise test.

1.06 times for each unit V_E/VCO_2 is above predicted. Predicted V_E/VCO_2 values at the anaerobic threshold are:

- 21.09 + (age × 0.123) for men.
- 22.09 + (age × 0.123) for women.

Fig. 3.5 depicts changes in VO_2, VCO_2, VE/VO_2 and VE/VCO_2 during a CPET. The peak VO_2 and VE/VCO_2 at anaerobic threshold (ATh) are circled. We have found that, taken together, these two variables are consistently useful for quantifying the contribution of fitness to mortality estimation in most populations. In our experience other derived variables, including AT, independently estimate survival but do not contribute additional information above and beyond that given by VO_2 and VE/VCO_2 have been quantified.

Anaerobic threshold has theoretical attractions as a prognostic variable in surgical patients. It is associated with the metabolic changes associated

Table 3.8 The association between anaerobic threshold, ECG evidence of myocardial ischaemia and perioperative mortality identified by Older and colleagues

Anaerobic threshold	Mortality rate		
	Test ECG: no ischaemia	Test ECG: ischaemia	Total
>11mL O_2/kg/min	0/107 (0%)	1/25 (4%)	1/132 (0.8%)
<11mL O_2/kg/min	2/36 (5.5%)	8/19 (43%)	10/55 (18%)
	2/143 (1.4%)	9/44 (20%)	11/187 (6%)

Data from Older P *et al*., 'Preoperative evaluation of cardiac failure and ischemia in elderly patients by cardiopulmonary exercise testing', Chest, 1993, **104**, pp. 701–704'.

with cardiovascular stress such as exercise. Whilst the author's preference is to use VO_{2peak} and VE/VCO_2 as the 1° prognostic variables derived from CPET anaerobic threshold is also widely used. Indeed, anaerobic threshold and inducible myocardial ischaemia were the 1° variables used in the work of Older and colleagues, which established the widespread use of CPET in preoperative assessment. Table 3.8 details the results of Older *et al*.'s initial research.

CPET adds prognostic value to other clinical information, allowing discrimination of AAA repair subpopulations with good or very poor long-term survival prospects. In a single-centre study, historical and CPET data individually estimated prognosis, but a more precise estimation resulted from their combination. Using combined data populations with a 2-year post-operative survival 97 and 55%, respectively, were identified.

DSE and mortality risk assessment

DSE has not been integrated into general survival estimation models. Considered in isolation a unit increase in wall motion score index (WMSI) during DSE is associated with a two fold increase in mortality in men and a fourfold increase in women. The WMSI is the sum of DSE segment scores (each 1 to 5) divided by the number of segments scored (0 to 5).

Further reading

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Protecting the heart

Cardiovascular complications of anaesthesia and surgery are frequent in patients with heart disease. The Perioperative Ischaemia Evaluation (POISE) trial showed that in patients with coronary artery disease or risk factors for that condition the risks of a major cardiovascular complication within 30 days of surgery are:

- 1.6% for cardiac death.
- 4.9% for myocardial infarction.
- 7.2% for other cardiovascular complications.

This means that one in seven patients suffer a major cardiovascular event. Clearly, cardiac protection is needed, especially in patients presenting for vascular surgery. There are three strands to cardiac protection:

- Coronary revascularization, which is appropriate only in a selected group of very high risk patients.
- Pharmacological protection.
- Optimization of cardiac function.

Protecting the heart

Coronary revascularization and pharmacological protection are the main strategies for perioperative cardiac protection, as they are for the management of cardiac disease outwith of the context of surgery.

Coronary revascularization

Where coronary revascularization is needed in its own right (i.e. irrespective of impending surgery), it should be performed before major vascular surgery if this is feasible. Revascularization may be by CABG or by PCI with insertion of BMS or drug-eluting stents (DES). Observational evidence shows that for 5yrs after successful CABG, providing there is no recurrence of symptoms and cardiac function is good, the risk of perioperative major cardiac events after non-cardiac surgery is small. By contrast, revascularization using coronary stents does not appear to offer much protection. Indeed, if non-cardiac surgery is performed within 6 weeks of insertion of bare metal or drug-eluting stents, the risk of perioperative cardiac events is very high. The risk declines with the passage of time.

There are two reasons for the high risk shortly after insertion of coronary stents:

- Surgery induces a state of hypercoagulability that facilitates stent thrombosis, even where dual antiplatelet therapy is maintained.
- Untimely interruption of dual antiplatelet therapy to reduce the risk of perioperative bleeding carries a high risk of stent thrombosis.

Guidelines indicate that, if at all possible, surgery should be delayed after PCI and take place only when dual antiplatelet therapy is no longer needed. The usual duration of dual antiplatelet therapy is 30–42 days after insertion of BMS and 1yr after insertion of drug eluting stents. Thus, it is recommended to wait for at least:

- 42 days and preferably 3 months, after BMS insertion.
- 1yr after DES insertion.

This is not always feasible.

Surgery that cannot be delayed until dual antiplatelet therapy is no longer necessary

Where non-cardiac surgery cannot be delayed until dual antiplatelet therapy is no longer needed (up to 3 months in the case of a BMS and 12 months in the case of a DES), it is important for surgeon, cardiologist, anaesthetist, and the patient to come to a joint decision as to the balance of risks of:

- Excessive bleeding if dual antiplatelet therapy is continued.
- Stent thrombosis if treatment is stopped.

The surgeon will be able to evaluate the risk of bleeding and the *consequences* of excessive bleeding as the benefits of surgery could be negated. The cardiologist will be able to evaluate the risk of stent thrombosis as a function of the type, number, and location of stents. Co-morbidities, such as diabetes and poor cardiac function increase the risk of perioperative stent thrombosis. The final decision may be that surgery should not be carried out at all, should be carried out under dual anti-platelet therapy, under aspirin alone, or under bridging therapy. The latter involves switching from aspirin and clopidogrel to aspirin and a short-acting antiplatelet agent, such as the glycoprotein IIb/IIIa inhibitor tirofiban.

Ideally, these patients should be monitored on a high dependency unit (HDU) as there is always risk of stent thrombosis. Immediate intervention would be needed to minimize the risk of extensive myocardial damage, thus availability of PCI at any time of day or night is essential.

Emergency surgery

- Aspirin and clopidogrel irreversibly block platelet function.
- Clotting only returns to normal as new platelets are released into the circulation so it may take up to a week for the effects of these drugs to be reversed after discontinuation.
- Short delays (hours) do not allow sufficient active platelets to be released into the circulation to reduce the risk of bleeding. Where emergency surgery is needed, delays of a few hours are only valuable to better evaluate the risk of surgery and prepare the patient.
- Excessive bleeding may require the administration of platelets as this is the only effective way of restoring normal haemostasis.
- Emergency surgery is associated with a three-fold increase in the risk of stent thrombosis. Thus, patients should be monitored on a high dependency unit. Involvement of an interventional cardiologist is essential, as a timely percutaneous cardiac intervention may reduce myocardial damage in the event of stent thrombosis.

Summary

Where coronary revascularization is needed, irrespective of impending surgery, available evidence suggests that surgical revascularization is likely to offer more protection than coronary stent insertion even using BMSs.

- If PCI is performed DES should be avoided because of need for protracted dual antiplatelet therapy.
- In patients with coronary stents, surgery should be delayed until dual antiplatelet therapy is no longer needed.

- Where surgery cannot be delayed, surgeon, cardiologist, anaesthetist, and the patient should come to a joint decision as to the best management of dual antiplatelet therapy. Patients should have their operation in a hospital where PCI is available and should be monitored in a HDU.

Pharmacological protection

Many classes of drugs have anti-ischaemic effects because they modify coronary vascular tone (thereby improving coronary blood flow or redistributing it to compromised areas), systemic vascular resistance (reducing afterload and myocardial oxygen demand), or cardiac dynamics (reducing heart rate and contractility). Unfortunately, many desirable effects of cardiovascular drugs translate into reduced myocardial ischaemia, but *not necessarily* into reduced perioperative myocardial damage or cardiac complications:

- Nitrates and ACE inhibitors are ineffective in preventing major cardiac events.
- Of the Ca^{2+} channel blockers, only verapamil and diltiazem have shown some efficacy, but they are little used for perioperative cardiac protection, and there are few randomized controlled trials to support their use.
- Alpha$_2$-adrenoceptor agonists (clonidine, dexmedetomidine) exert their effect centrally and reduce sympathetic outflow. They have been shown to reduce the risk of cardiac events but the number of patients included in RCTs is small.

Beta-blockers

Beta-adrenoceptor blockers were regarded as the ideal agents for perioperative cardiac protection from the mid-1970s onwards. They were increasingly recommended for the perioperative management of patients with risk factors for, or with coronary disease. The POISE trial enrolled over 8000 patients and cast serious doubts on the value of perioperative beta-blockade. Patients treated for a month with slow-release metoprolol, starting the day of surgery, showed reduced risk of cardiac events. However, there was an increase in all-cause mortality and strokes in the active arm of the study. The results of POISE forced a reappraisal of the use of perioperative beta-blockers and new European (ESC/ESA) and American (ACCF/AHA) guidelines were published.

The guidelines indicate that:

- Long-term treatment with beta-blockers should be continued throughout the perioperative period.
- Patients with reversible ischaemia on a stress test should be treated with beta-blockers preoperatively.
- Treatment should be started at least a week, preferably a month before surgery.
- The dose of beta-blocker should be titrated to effect namely heart rate 60–70 in the ESC guideline and 60–80 ACCF/AHA guideline with BP above 100mmHg (ESC guideline) or 'no hypotension' (ACCF/AHA guideline). The latter recommendation is more appropriate as 100mmHg can be regarded as too low in patients with preoperative

arterial hypertension and would be regarded by most clinicians as 'hypotension' in such patients.

The guidelines differ in their recommendations for perioperative beta-blockade: the ACCF/AHA guideline is more restrictive than the ESC guideline as the latter includes relatively low risk patients (one risk factor) and low risk surgery. This is not supported by the literature as several studies show benefits only in high risk patients.

The need for an early start to beta-blockade is logical, but the evidence is limited to a very small number of patients in two randomized controlled trials. Similarly, the benefits of titration to specified heart rate and BP is logical, and supported by a small number of patients in RCTs. By contrast a very large observational study published after the above guidelines were issued, showed that in order of protection, initiating beta-blockade is best, followed by continuing on-going beta-blockade, while no beta-blockade provides poorer outcome, and withdrawal of beta-blockade poorest outcome. This study suggests that in high risk patients initiating beta-blockers at the time of surgery does provide benefits.

The POISE trial and the guidelines did not address the issue of targeted perioperative beta-blockade where there is a need to control sympathetically-mediated tachycardia, hypertension, and myocardial ischaemia. This remains an entirely legitimate indication for perioperative beta-blockade.

Statins

Half of post-operative ischaemic cardiac complications are caused by the acute disruption of unstable coronary plaques. Plaques can be made more unstable by the release of inflammatory mediators during the perioperative period; conversely they are made more stable by statins.

The efficacy of statins in the prevention of perioperative complications following non-cardiac surgery is based essentially on observational studies in patients on chronic statin medication. The largest study, with over 780 000 patients, including over 77 000 taking statins showed a lower all-cause mortality in those on statins (2.18 versus 3.05%, a 29% reduction). By contrast, only four randomized controlled trials have examined the deliberate introduction of statins prior to surgery. Benefits were observed mostly in *high risk* patients. While the results are encouraging, there is need for more randomized controlled trials.

There is agreement that statins should not be discontinued during the perioperative period as withdrawal causes a large increase in the risk of acute coronary events while rhabdomyolysis is extremely rare.

In respect of statins, the ACCF/AHA guideline recommends continuing therapy in patients currently taking statins. The introduction of statins is reasonable in patients undergoing vascular surgery with or without clinical risk factors; statins may be considered in patients with at least one clinical risk factor who are undergoing intermediate-risk procedures. By contrast the ESC guideline considers that statins should be started only in high-risk surgery patients, optimally between 30 days and at least 1 week before surgery. It also supports the view that statins should be continued perioperatively.

Summary

- Beta-blockers should be used in high risk patients; whenever possible they should be started early, and be titrated carefully to avoid hypotension. They cannot be regarded as protective in patients at low risk of cardiac events as previously thought.
- A large observational study published after the guidelines suggest that in the presence of a clear indication beta-blockers can be introduced successfully on the day of surgery. Withdrawal of beta-blockers increases the risk of perioperative cardiac events and should be avoided.
- The evidence from observational studies of long-term statin therapy indicates that they confer cardiac protection. Statins, like beta-blockers, seem to be more protective in high risk than in low risk patients.
- Statin therapy should be maintained perioperatively in those on chronic treatment.

Further reading

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Optimizing cardiac function

Goal-directed therapy in now widely advocated and practiced in patients undergoing major surgery. This involves administering fluids and, in some cases, inotropic drugs to optimize haemodynamic performance. This approach grew out of the targeting of supranormal oxygen delivery in critically ill patients. The increasing sophistication of haemodynamic monitoring has made available a wide range of haemodynamic variables that may be targeted by goal-directed therapy. These include cardiac output, stroke volume, stroke volume variability, and pulse pressure variability. The reader is directed to 📖 General principles of monitoring, p. 312, on cardiovascular monitoring. While increasing oxygen delivery to the tissues to supranormal levels is no longer recommended, a considerable number of studies suggest that goal-directed therapy can improve outcome in patients undergoing

major surgery, a conclusion supported by a meta-analysis by Poeze and colleagues. However, a meta-analysis by Giglio and colleagues, which examined patients undergoing only cardiac or vascular surgery found no benefit of goal-directed therapy in vascular surgery patients. Therefore at the time of writing, the benefits of goal-directed therapy in vascular surgery patients remain uncertain.

If goal-directed therapy is to be employed, improvements in haemodynamic performance can usually be achieved with administration of fluids, rather than use of inotropic drugs unless there is evidence of cardiac dysfunction. Where cardiac failure is present, the addition of inotropic agents may be necessary. Catecholamines and their derivatives are generally used. For most inotropic agents, increased Ca^{2+} entry and transfer of calcium within the cell are essential to their effect. It follows that energy is expended in reversing calcium exchanges during the diastolic phase of each cardiac cycle. Recently, levosimendan, a calcium sensitizer has been found to be effective perioperatively after cardiac surgery. Its major benefit is that, unlike catecholamines and their derivatives, it does not cause an increase in metabolic demand over and above that due to increase cardiac work.

Further reading

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Optimizing renal function

Chronic kidney disease (CKD) increases the risk of perioperative acute kidney injury (AKI). AKI is an independent predictor of mortality after major vascular surgery and can lead to CKD.

Preoperative diagnosis of renal dysfunction

Renal function is assessed by laboratory tests which reflect GFR (urea, creatinine, creatinine clearance, proteinuria) or tubular function (urine osmolality or fractional sodium excretion). Many of these tests are insensitive or affected by other factors:

- *Urea:* (normal 2.5–6mmol/L). Reflects GFR. Also increased by dehydration, GI bleeding, high protein diet, catabolism.
- *Creatinine:* (normal 60–120μmol/L). Reflects GFR. Depends on metabolism of creatine in muscle. Depends on muscle mass, state of hydration, diet, certain drugs, age, gender, race, and laboratory methods used. Responds slowly (48–72h) after an episode of AKI.
- *Creatinine clearance:* (normal 110–150mL/min). Reflects, but tends to overestimate GFR by a variable amount. Can be used to determine need to start dialysis, or to estimate GFR, where there are extremes of body size (amputees, malnutrition, very high muscle mass) or exceptional dietary circumstances (e.g. vegetarians).

Table 3.9 Diagnosis of chronic kidney disease

CKD stage	GFR (mL/min/1.73m^2)	Notes
1	>90	Diagnosis of CKD requires other evidence of kidney disease*
2	60–89	Other evidence of kidney disease*
3	30–59	
4	15–29	
5	<15	

* Diagnosis of Stage 1 or 2 chronic kidney failure requires other evidence of CKD e.g. proteinuria, haematuria, a genetic diagnosis (e.g. polycystic kidney disease) or structural abnormalities (e.g. reflux nephropathy).

- *Proteinuria (albumin):* (normal <150mg/day). Transient proteinuria is associated with acute illness, e.g. fever, heart failure, pancreatitis, or exercise. Repeated proteinuria suggests renal disease.
- *Urine osmolality:* (normal 40–1400mOsm/L).
- *Fractional urinary sodium excretion:* (normal <40mEq.L.) Values above normal suggest tubular damage or diuretics.
- *Other specialized tests:* e.g. urinary neutrophil gelatinase-associated lipocalin (NGAL), interleukins, retinol binding protein, albumin/creatinine ratio are not used in routine practice.

The best index of kidney function is estimated GFR (normal values >90mL/min/1.73m^2).

- eGFR is calculated from serum creatinine and accounts for age, weight, sex.
  - CKD is diagnosed and classified on the basis of estimated GFR using the modification of diet in renal disease (MDRD) or Cockroft–Gault equations in adults (see Table 3.9).
  - eGFR has been shown to be a more sensitive predictor of preoperative renal dysfunction than serum creatinine (in patients undergoing AAA repair) and correlates with post-operative morbidity and long-term survival after AAA repair.
  - Those with GFR < 60mL/min/1.73m^2 are classified as having CKD.

Preoperative investigations (all vascular patients)

U&Es, glucose, GFR, FBC, ECG, chest X-ray (CXR), urinalysis. If renal dysfunction suspected:

- ABG, urine microscopy, platelet function tests, clotting studies.
- Urine/plasma osmolality ratio, urinary electrolytes.
- Renal tract US.
- *Consider:* echocardiography, further cardiological investigations (see 📖 Cardiological investigations in the vascular surgery patient, p. 129).

Refer to nephrologist before vascular surgery if:

- Currently requiring dialysis.

- eGFR <30 (CKD stages 4–5) for planning of anaesthesia, surgery, and post-operative care.
- New diagnosis of CKD stages 3–5 for further investigation before elective vascular surgery.

Optimizing renal function before surgery

- Liaise with nephrologist if patient is receiving renal replacement therapy (RRT), i.e. dialysis or haemofiltration. Surgery should be planned for 1–2 days after recent RRT to optimize fluid balance and allow the effects of anticoagulation to subside.
- *Identify and treat:*
  - Hyperkalaemia.
  - *Fluid overload or heart failure:* consult nephrologist regarding RRT.
  - *Uraemia:* urea >35mmol/L). Consult nephrologist regarding RRT.
  - *Hypertension:* see 🕮 Hypertension, p. 160.
  - *Renal artery stenosis:* consider preoperative renal artery angioplasty, or a combined procedure (in patients undergoing aortic surgery).
  - Pericardial effusion.
  - Coagulopathy (consider transfusion of platelets, fresh frozen plasma (FFP), cryoprecipitate). Uraemic coagulopathy may require treatment with Desmopressin (1-deamino-8-D-arginine vasopressin) (DDAVP) (see 🕮 Chronic kidney disease, p. 202).
- Avoid or omit any drug or condition which might worsen renal function:
  - *Nephrotoxins*—e.g. non-steroidal anti-inflammatory drugs (NSAIDs), ACE Inhibitors, angiotensin II receptor blocker (ARBs) (omit before surgery), aminoglycosides, amphotericin (lipid forms less toxic), cyclosporins.
  - *Radiocontrast*—delay surgery for at least 24h after administration of radiocontrast
- Optimize cardiovascular function throughout the perioperative period:
  - *Maintain circulating volume pre-operatively*—avoid dehydration and hypovolaemia, especially if bowel preparation is used.
  - *Encourage clear oral fluids up to 2h before elective surgery*—IV hydration using balanced electrolyte solutions (e.g. Hartmanns, Ringer's lactate) 1.5mL/kg/h may be infused for 8–12h preoperatively. Avoid excessive Na administration or K-containing fluids if renal function impaired. In these cases, K-free crystalloid solutions, such as 0.45% saline or 0.18% saline / 4.0% glucose solution should be used.
- Consider invasive cardiovascular monitoring in HDU/intensive care unit (ICU) before surgery using:
  - Direct arterial pressure monitoring.
  - CVP and/or flow-guided monitors (see 🕮 General principles of monitoring, p. 312).
  - Institute vasoactive drugs early if arterial pressure and cardiac output do not respond to IV fluids.
  - Targets are CVP 8–12mmHg, MAP >70mmHg, urine output >0.5mL/kg/h, central venous SvO_2 >70%.

Anaesthesia and post-operative care are detailed in 📖 Perioperative renal protection, p. 236 and 📖 Chronic kidney disease, p. 202.

Further reading

Brienza N, Giglio MT, Marucci M, Fiore T. Does perioperative hemodynamic optimization protect renal function in surgical patients? A meta-analytic study. *Crit Care Med* 2009; **37**: 2079–90.

Useful websites

http://www.kidney.org—definitions, investigations for CKD.
http://www.renal.org—definitions, investigations.

Optimizing respiratory function

Acute chest problems

For patients having elective vascular interventions, acute chest pathology must be excluded before proceeding. The decline in respiratory function seen with anaesthesia and the normal systemic stress response to surgery are both likely to exacerbate any pre-existing acute respiratory illness. Although there are no clinical outcome studies to back up this statement, considering the detrimental effects of anaesthesia, it is inadvisable to anaesthetize patients who already have an acute respiratory illness.

Acute chest infections or exacerbations of COPD are the most common preoperative respiratory problems encountered in vascular patients. Detection of these is most effectively achieved by a standard history and examination. A patient with symptoms of acute chest infection or a change in their normal respiratory symptoms requires further investigation with a CXR and white blood cell count. In an asymptomatic patient, the use of blood tests or CXRs as a screening tool for acute chest problems is ineffective, costly, and possibly harmful to the patient.

If an acute respiratory illness is discovered preoperatively then standard treatment should be initiated and a multidisciplinary discussion must take place to decide on the optimal time for surgery to be performed. This is a decision to be made on an individual patient basis, taking into consideration the risks of postponing surgery and the likely time required to treat the respiratory illness.

Existing respiratory disease

In patients with existing respiratory pathology and a stable clinical picture the anaesthetist must decide how much of a perioperative risk this presents. The anaesthetist should also assess whether the patient's current treatment is optimal, and therefore should have a good knowledge of appropriate management guidelines for common respiratory diseases such as asthma and COPD. Lack of compliance with therapy is a common cause for poor symptom control, and the anaesthetist should discuss with the patient how inhaled therapy may be administered in the perioperative period. For example, the use of a nebulizer, rather than inhalers may be required when a patient is unable to adequately co-ordinate their breathing and inhaler activation due to post-operative sedation.

For a patient with stable, but severe COPD it may be beneficial to increase their therapy preoperatively, e.g. by prescribing steroids, nebulizers, or

adding extra bronchodilator drugs. However, in patients with COPD of such severity that this requires consideration it is sensible to only do so in consultation with a respiratory specialist.

Identifying patients at risk of respiratory complications

Risk factors known to be associated with the development of a post-operative pulmonary complications (PPC) have been reviewed by Smetna. The relative risks (RR) of PPC associated with patient related risk factors are smoking (RR 1.4–4.3), ASA>II (RR 1.5–3.2), age greater than 70 years (RR 0.9–1.9), obesity (RR 0.8–1.7) and COPD (RR 2.7–3.6). For this purpose, PPCs are complications that prolong hospital stay, or cause additional morbidity or mortality, and include pneumonia, a requirement for artificial ventilation, atelectasis, or an exacerbation of existing lung disease. In addition to these factors, the site of surgery has an impact on the likelihood of PPCs, and the old adage remains true that the closer the incision is to the diaphragm, the worse the respiratory outcome. Laparoscopic or endovascular interventions are always associated with less PPCs than the equivalent procedure done by an open route.

The majority of risk factors can be determined by taking a history from the patient, with little further predictive power being gained with preoperative investigations. Knowledge of these factors allows the anaesthetist to tailor their proposed anaesthetic technique for each patient, effectively strengthening the case for regional anaesthesia if at all possible when more factors are present. The intervention most likely to reduce the chances of PPC is avoidance of general anaesthesia, and minimizing the risk of PPC remains the most evidence-based benefit of regional anaesthesia.

The place of spirometry for predicting PPCs remains controversial. Using absolute values for readings obtained is unhelpful—these should always be expressed as a percentage of predicted for that patient. These results may then be used as part of the clinical grading of the respiratory disease into mild, moderate, or severe, but there are no magic threshold values below which PPCs will occur.

Once an assessment has been made of the likelihood of PPCs, this information can be used as part of the preoperative discussion regarding the advisability of surgery or the most appropriate procedure to perform, the anaesthetic strategy for that patient, and the likely level of care required post-operatively.

Perioperative tobacco smoking

Effects of smoking in the perioperative period

Patients who smoke have an increased incidence of a host of minor intraoperative complications, such as cough, breath-holding, laryngospasm, and desaturation in recovery. More importantly, PPCs are several times more common in smokers, the size of this effect depending on the type of surgery and the definition of a PPC. Preoperative cessation of smoking is by far the most important single intervention that can reduce the risk of perioperative respiratory complications.

Smoking cessation

Physiological responses to smoking cessation

Within a few hours of stopping smoking a host of changes occur:

- Carboxyhaemoglobin levels fall with a half-life of 4h, so after 12h without a cigarette levels are the same as for a non-smoker.
- Nicotine is eliminated with a half-life of only 30min, so levels will also reach those of a non-smoker within hours.
- Pulmonary ciliary function recovers a few hours after the last cigarette, and the mucociliary escalator begins again, normally leading to a productive cough.

The first two of these changes are vital for patients having vascular surgery, as the cardiostimulant properties of nicotine and the reduced oxygen carrying capacity with carboxyhaemoglobinaemia both impair myocardial function. For example, patients with ischaemic heart disease who undergo exercise testing have more prolonged chest pain and more arrhythmias if they smoked immediately before the test. Considering the parallels commonly drawn between the stress of exercise and the perioperative period, the effects of recent smoking may be harmful in the first hours of a surgical intervention, in particular during induction of anaesthesia. Any patient having major vascular surgery is at risk of ischaemic heart disease and should abstain from smoking for 12h preoperatively.

Timing of perioperative smoking cessation

Several days of smoking cessation are required before the airway irritability and mucous hypersecretion responsible for the common intraoperative complications subside. Several weeks are required before the airway epithelial remodelling is reversed, e.g. the number of goblet cells returns to normal and smooth muscle cell hyperplasia reduces. The duration of preoperative smoking cessation required to reduce PPCs remains uncertain.

Two descriptive studies of patients having open heart surgery showed that the incidence of PPCs was *greater* in those who stopped smoking for less than 8 weeks preoperatively compared with those who continued smoking until the day before surgery. Several more recent randomized studies failed to reproduce these observations in a variety of other patient groups, and confirmed the benefit of pre-operative smoking cessation.

Current advice is that patients should be encouraged and supported to stop smoking preoperatively at any time. Anaesthetists should be actively involved in this as the impending major surgery can be used as a 'teachable moment' for the patient, i.e. a point in time when their motivation to stop smoking is particularly heightened.

Interventions to reduce post-operative complications

For patients at risk of PPCs, for example those with a significant number of risk factors for PPC having open abdominal or thoracic surgery, preoperative interventions may be instituted to reduce the risk. Evidence suggests that using any of these interventions reduces the incidence of PPCs by around 50%, but no single one is better than the rest.

Physiotherapy

Involving physiotherapists as part of the perioperative vascular team is important. A variety of techniques may be used to improve respiratory function, such as chest percussion, intermittent positive pressure breathing or deep breathing exercises. These are all likely to be more beneficial in the early post-operative period, but there is evidence that the interventions are more effective if taught to the patient preoperatively

Incentive spirometry

This technique attempts to mimic the deep breathing exercises that a physiotherapist would teach. By using brightly coloured balls in calibrated flow chambers an incentive spirometer aims to make the patient take a slow and prolonged vital capacity inhalation to facilitate re-expansion of collapsed lung regions. A patient can use the device frequently, but using the device on their own reduces motivation in comparison with when the physiotherapist is present and encouraging the patient throughout the manoeuvre. Once again, for the best results, the incentive spirometer technique should be learned and practiced preoperatively.

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Vascular disease and non-cardiovascular surgery

Introduction

Preoperative assessment of the cardiovascular system prior to major vascular surgery is covered in detail in chapters elsewhere. This chapter will focus on how extra-cardiac vascular disease impacts on patient management and outcome in the perioperative setting. This will have particular relevance to patients undergoing non-vascular surgery.

Surgical and anaesthetic teams may encounter several co-morbid vascular pathologies impacting on patient management for non-vascular surgery. In addition co-morbidities including renal dysfunction and diabetes (in isolation or together) should foster a high index of suspicion for the presence of vascular disease. Specific vascular conditions are considered below.

Cerebrovascular disease

Small and large vessel CVD is more common in elderly patients. Screening studies in the general population have demonstrated an incidence of

significant (>50%) internal carotid artery stenosis of 0.5 and 10% in those in their 50s and >80yrs, respectively. As the population ages, an increasing number of elderly individuals present for surgery, many of whom will have significant cerebrovascular disease.

Perioperative complications arising as a consequence of CVD include; TIA, acute stroke, post-operative cognitive dysfunction (POCD), delirium, and memory loss. Several factors have been implicated in the genesis of perioperative stroke, highlighting its complex nature.

Perioperative factors

- *Type of surgery:*
  - General surgery <1%.
  - Peripheral arterial/carotid 1–2%.
  - Cardiac 1–2%.
  - Risk is enhanced in the emergency versus elective setting.
- *Duration of surgery:* risk increased with surgical duration.
- Intraoperative hypotension.
- *Type of anaesthesia:* there is no real evidence available to implicate any specific 'type' of anaesthesia in contributing to post-operative cerebrovascular dysfunction. Intraoperative hypothermia and abnormal blood glucose have been implicated to contribute to incidental stroke. Normothermia and tight blood glucose control are recommended (consensus opinion) during prolonged or major surgical procedures.

Patient factors

- *Recent stroke/TIA:* a history of recent stroke or TIA is the strongest predictor for perioperative stroke. Symptomatic carotid disease may trigger surgical correction with carotid endarterectomy prior to consideration of other non-cardiac surgery. Where this is not the case, there is limited evidence to recommend a 'safe' time period between stroke and surgery to minimize further risks. In the face of a lack of evidence it is recommended that the anaesthetist ensures that medical treatment of cerebrovascular disease has been optimized before surgery. Ideally, resolution of any previous stroke should also have occurred. Close liaison between stroke physicians and surgical teams is essential, where surgery is deemed necessary, to reduce further risk.
- *Increasing age:* predisposes to increased risk of perioperative stroke. This is primarily as a result in an increased incidence of CVD as outlined above.
- *Medication:* discontinuation of warfarin or antiplatelet agents prior to surgery exposes patients to an increased risk of perioperative stroke. Acute beta blockade for perioperative cardiac protection has also been implicated in predisposing patients to increased stroke risk (POISE trial). This has been attributed to hypotension in the perioperative period in the patients receiving relatively high doses of beta-blockers.
- *Co-morbid disease:* perioperative stroke is more common in patients with diabetes and renal dysfunction. This is consistent with the increased risk of other vascular complications associated with these conditions, e.g. adverse cardiac events. Again, every effort should be made to ensure that these conditions are optimally managed.

- *Uncontrolled grade 3 hypertension* (systolic BP >180mmHg, diastolic BP > 110mmHg) has also been strongly implicated in predisposing patients to increased risk. Where possible it is recommended that surgery be delayed until hypertension has been treated. There is no evidence that grade 1 or 2 hypertension predisposes to similar increased risk.
- Although difficult to define, the risk of stroke is almost certainly increased in patients with critical illness or sepsis.

When considering the above factors, the complex nature of perioperative cerebrovascular dysfunction becomes apparent. Close collaboration, appropriately timed surgery and attention to detail should all contribute to minimizing risk.

Peripheral vascular disease (PVD)

Patients with infra-inguinal occlusive arterial disease have the highest incidence of significant coronary disease of any patient group presenting for surgery. Covert or overt coronary disease will be present in 60–70% of individuals. It is for this reason that infra-inguinal arterial bypass surgery is classified as high risk for cardiac complications by both American and European guidelines on cardiac risk for non-cardiac surgery.

Identifying which individuals presenting for surgery have significant coronary disease may be prove challenging. Conventional assessment relying on functional capacity to identify coronary disease is of limited use in this setting, as a consequence of claudication. Therefore preoperative assessment of individuals with PVD for non-cardiac or non-vascular surgery should be rigorous. Where there is concern it may be appropriate to employ a lower threshold for specialist cardiac assessment in this patient group.

There is a high prevalence of diabetes and renal disease in patients presenting for surgery with PVD. It is essential to ensure optimal treatment of these conditions prior to consideration elective of non-cardiac surgery.

Abdominal aortic aneurysm

Patients may present with a co-incidental finding of an AAA, whilst being considered for other forms of surgery. Where the AAA is >5.5cm, a balanced decision may need to be made as to which is the most appropriate pathology to address in the first instance. Under most circumstances, the most immediately life-threatening condition is addressed first, but such decisions require close multi-disciplinary collaboration.

Where a large AAA is coincidentally present with an intra-abdominal malignancy, it may be most appropriate to consider endovascular repair of the aneurysm in the first instance if possible. This staged approach allows a speedier recovery prior to consideration of a laparotomy for resection of intra-abdominal malignancy. Risk is also minimized, as combined procedures for correction of >1 pathology carry a high surgical risk of mortality, even in relatively healthy individuals. Infection of the surgical graft is also a major concern when considering combined procedures for differing pathologies.

Individuals presenting for non-vascular surgery with co-incidental small AAA (<5.5cm) require no special provisions to be made. There are,

however, anecdotal reports of increased risk of subsequent AAA rupture following surgical laporotomy for other intra-abdominal pathology where an incidental AAA is present.

Summary

Patients presenting for non-vascular surgery with co-incidental vascular disease present a significant risk of perioperative morbidity. There is a particularly high risk of cardiac and cerebrovascular complications, which can be minimized through identification and optimization of associated risk factors in the perioperative period.

Chapter 4

Management of specific medical conditions and medications

Hypertension

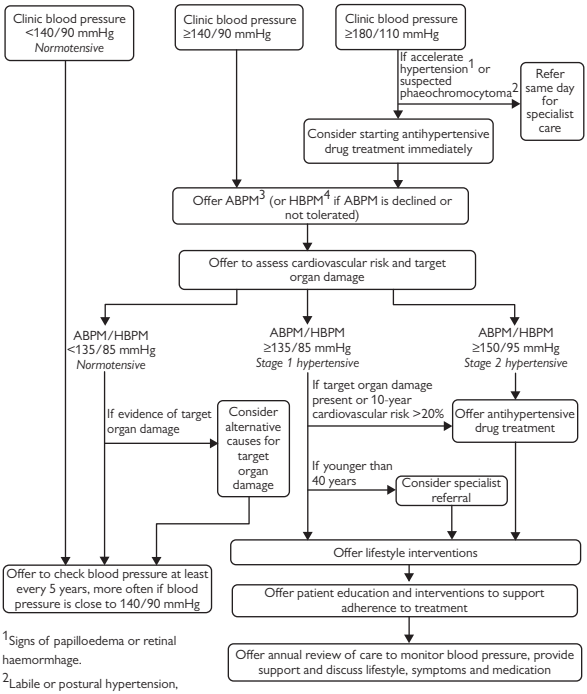
Diagnosis and treatment

Hypertension cannot be diagnosed on a single BP measurement. The 2011 United Kingdom National Institute for Health and Care Excellence (NICE) guidelines recommend ambulatory monitoring or home BP monitoring, if BP measured in the clinic is 140/90mmHg or greater. This is not currently feasible in all cases, but is of value where the diagnosis is uncertain.

The patient with suspected or confirmed hypertension should be investigated for target organ damage (including left ventricular hypertrophy, chronic kidney disease, and hypertensive retinopathy) and assessed for cardiovascular risk using a tool such as the QRISK2 Calculator (http://www.qrisk.org).

Several guidelines for the management of hypertension are available. In brief, the United Kingdom NICE guidelines (Fig. 4.1) recommend:

- If BP is ≥180/110mmHg consider starting antihypertensive medication immediately.
- Refer the patient for specialist care the same day if they have:
  - Accelerated hypertension, i.e. a BP of >180/110mmHg with signs of papilloedema or retinal haemorrhage.
  - Suspected phaeochromocytoma, i.e. labile or postural hypotension, headache, palpitations, pallor, and diaphoresis.
- Offer antihypertensive drug treatment to:
  - People aged under 80yrs with an average ambulatory or home BP of ≥135/85mmHg and <150/95mmHg.
  - A clinic BP ≥140/90mmHg and <160/100mmHg who have one or more of the following: target organ damage, established cardiovascular disease, renal disease, diabetes, or a10-yr cardiovascular risk equivalent to ≥20%.
- Offer antihypertensive drug treatment to people of any age with an average ambulatory, or home BP≥150/95mmHg or a clinic BP 160/100mmHg, irrespective of presence of target organ damage, cardiovascular disease, renal disease, or 10-yr risk of cardiovascular disease.
- For people under 40yrs with a clinic BP of ≥140/90mmHg, and average BP on home or ambulatory monitoring of >135/85mmHg (stage 1 hypertension) who have no evidence of target organ damage, cardiovascular disease, renal disease, or diabetes, consider seeking specialist evaluation for 2° causes of hypertension and a more detailed assessment of potential target organ damage.
- The drug treatment of hypertension escalates through a series of steps until BP control is achieved. In brief the recommendations in the UK 2011 NICE guidelines (Fig. 4.2) are:
  - *Step 1*—patients aged under 55yrs should be offered ACE inhibitor or ARB. For people aged over 55yrs and black people of African or Caribbean family origin of any age, offer a calcium channel blocker.
  - *Step 2*—offer a calcium channel blocker (CCB) in combination with an ACE inhibitor or ARB.



[1]Signs of papilloedema or retinal haemormhage.

[2]Labile or postural hypertension, headache, palpitations, pallor and diaphoresis

[3]Ambulatory blood pressure monitoring.

[4]Home blood pressure monitoring

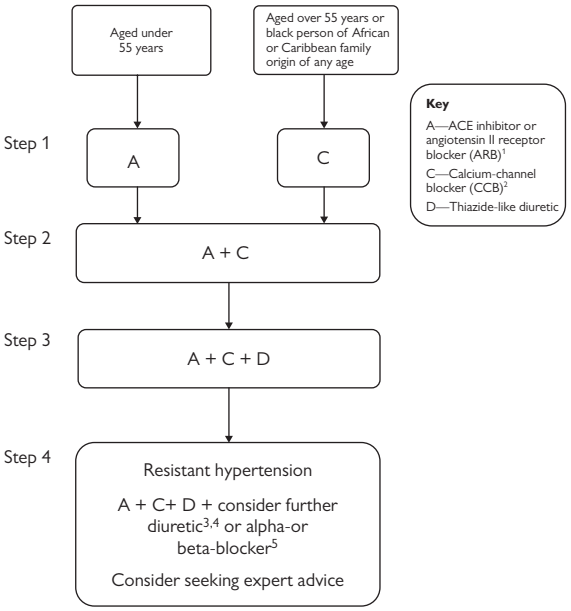
Fig. 4.1 The care pathway for hypertension.

National Institute for Health and Clinical Excellence (2011) Adapted from 'CG 127 Hypertension: clinical management of primary hypertension in adults'. London: NICE. Available from http://guidance.nice.org.uk/CG127 Reproduced with permission.

- *Step 3*—if treatment with three drugs is needed, offer an ACE inhibitor or ARB, combined with a CCB and a thiazide-like diuretic.
- *Step 4*—if clinic BP remains >140/90mmHg after treatment with the optimal or best tolerated doses of the drug combination mentioned in step 3, regard this as resistant hypertension, and consider adding a fourth antihypertensive drug and/or seeking expert advice.

Perioperative management

- The target organ damage associated with hypertension (IHD, HF, CVD, renal impairment) is associated with an increased risk of perioperative



[1]Choose a low-cost ARB.
[2]A CCB is preferred but consider a thiazide-like diuretic if a CCB is not tolerated or the person has oedema, evidence of heart failure or a high risk of heart failure.
[3]Consider a low dose of spironolactone or higher doses of a thiazide-like diuretic.
[4]At the time of publication (August 2011), spironolactone did not have a UK marketing authorisation for this indication. Informed consent should be obtained and documented.
[5]Consider an alpha-or beta-blocker if further diuretic therapy is not tolerated, or is constraindicated or ineffective.

Fig. 4.2 Summary of antihypertensive drug treatment.

National Institute for Health and Clinical Excellence (2011) Adapted from 'CG 127 Hypertension: clinical management of primary hypertension in adults'. London: NICE. Available from http://guidance.nice.org.uk/CG127 Reproduced with permission.

cardiovascular complications. Patients with a diagnosis of hypertension display increased cardiovascular instability during anaesthesia and surgery.

- Patients with severe uncontrolled hypertension should have their BP controlled before anaesthesia and surgery if possible. The ACC/AHA Guidelines recommend that if the systolic BP is 180mmHg and diastolic

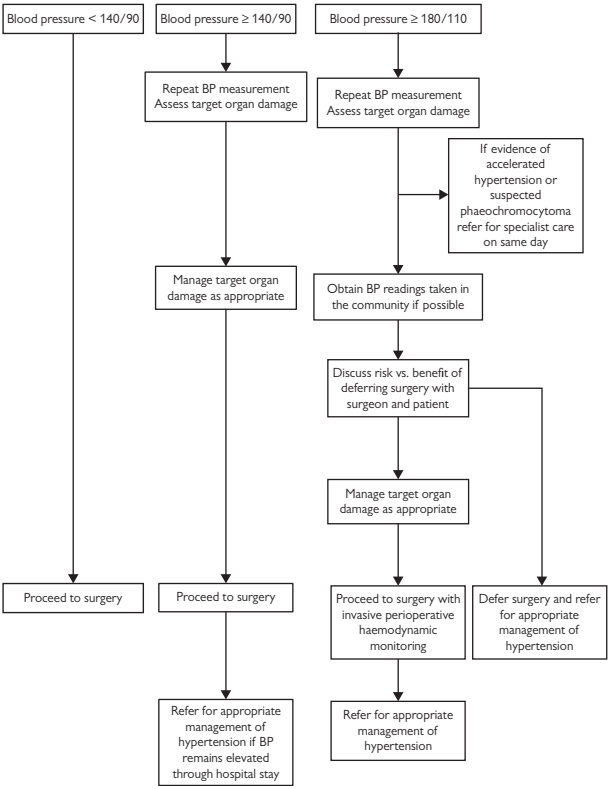


Fig. 4.3 Flowchart for preoperative blood pressure assessment

BP is 110mmHg, the potential benefits of delaying surgery to optimize the effects of antihypertensive medications should be weighed against the risk of delaying the surgical procedure.

- BP measurements taken in the pre-assessment clinic or on the ward just before surgery are unlikely to be the patient's 'true' BP. ∴ Anaesthesia

and surgery should not be deferred because of moderately high BP or a single BP reading >180/110mmHg. If the BP measured in hospital is consistently >180/110mmHg, contact the patient's GP for details of the patient's BP measured in the community. If the patient's BP is well controlled in the community setting, there is no benefit in deferring surgery to allow antihypertensive treatment to be modified.

- If the patient's BP is acceptable for surgery, but persistently elevated to >140/90mmHg whilst in hospital this information should be included in the discharge letter sent to the GP when the patient is discharged from hospital, so that the patient's BP can be rechecked in the community.
- In general, antihypertensive medications should be continued up to and including the morning of surgery. The exception to this is ACE inhibitors and ARBs, which may increase the risk of hypotension associated with anaesthesia if given within 12h before surgery. ∴ Some practitioners omit the dose of these drugs before surgery.
- The anaesthetist should estimate patient's true BP from the readings taken before surgery and try to maintain the MAP in the intraoperative period within 20% of the estimated true value.
- Restarting antihypertensive medications after surgery is a matter of clinical judgement. Whilst it is appropriate to restart treatment as soon as possible after minor surgery, this may not be possible after more major surgery, especially if the patient is nil by mouth. Beta-blockers are no longer a first line treatment for hypertension, but should be restarted as soon as possible in those patients who are taking them. IV beta-blockade may be required for heart rate control if the patient is nil by mouth. This is discussed in more detail in the section on coronary artery disease.

Further reading

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Coronary artery disease

Diagnosis and treatment

Almost all patients who have atherosclerotic disease requiring vascular surgery will also have coronary artery atherosclerosis. Some will be receiving medical treatment for CAD and others will have undergone PCI or coronary artery bypass grafting (CABG). In some cases, preoperative investigations will identify previously unrecognized CAD that requires appropriate management. The objectives of perioperative management are:

- To ensure that CAD is optimally treated before surgery.
- To avoid ischaemic cardiac events during and after surgery.

- To ensure that the patient is discharged on appropriate treatment for CAD with appropriate follow-up.

All patients with atherosclerotic vascular disease in any site (coronary, cerebral major arteries of the viscera, upper or lower limbs), should receive appropriate secondary cardiovascular prevention. This includes lifestyle advice, BP control, treatment for hyperlipidaemia (generally with a statin) and antiplatelet therapy (📖 Primary and secondary prevention of vascular disease, p. 14). Discontinuing anti-platelet therapy and statins in the perioperative period is associated with an increased risk of ischaemic injury to the heart and perioperative myocardial infarction. Both aspirin and statin therapy should be continued up to the day of surgery and restarted as soon as possible after surgery (📖 Primary and secondary prevention of vascular disease, p. 14).

Diabetic patients have a particularly high risk of recurrent cardiovascular events and every effort should be made to achieve good glycaemic control (HbA1c < 70mmol/mol).

Perioperative management

Many patients will have been investigated and treated for CAD before their presentation for vascular surgery. The aims of treatment are to relieve symptoms such as angina and to prevent future cardiac events including unstable angina, myocardial infarction, and cardiac death.

Therapeutic options for patients with CAD are medical management, PCI, or CABG. Decisions regarding treatment are informed by:

- The clinical history, for example the onset and frequency of chest pain and its response to treatment (see 📖 Clinical cardiological evaluation of the vascular surgery patient, p. 126).
- The clinical examination including both evidence of heart failure and evidence of other causes of chest pain such as musculoskeletal causes (see 📖 Clinical cardiological evaluation of the vascular surgery patient, p. 126).
- The ECG (see 📖 Cardiological investigations in the vascular surgery patient, p. 126).
- Echocardiography, in particular for evidence of left ventricular dysfunction (see 📖 Cardiological investigations in the vascular surgery patient, p. 126).
- Laboratory studies of cardiac biomarkers, in particular cardiac troponin, brain natriuretic peptide (BNP) and N-terminal pro-BNP (NT-pro BNP) (See 📖 Decision making in vascular surgery, p. 95, Vascular imaging, p. 95).
- Exercise ECG testing (See 📖 Cardiological investigations in the vascular surgery patient, p. 129).
- Other cardiac stress testing; tests may use exercise or pharmacological stress induced with dobutamine, dipyridamole, or adenosine. Imaging modalities include echocardiography; nuclear cardiac imaging, and cardiac magnetic resonance imaging (see 📖 Cardiological investigations in the vascular surgery patient, p. 129).
- Coronary angiography (see 📖 Cardiological investigations in the vascular surgery patient, p. 129, Preoperative coronary angiography and revascularization, p. 134)

Pharmacological therapy

- *Antiplatelet therapy:* as noted above, all patients should receive antiplatelet therapy with aspirin unless they have a contraindication to this drug. Clopidogrel or dipyridamole are alternatives in patients who cannot take aspirin. Drugs such as prasugrel and ticagrelor are also now increasingly being used.
- *Anti-anginal therapy:*
  - *Nitrates*—patients with a diagnosis of angina should carry a glyceryl trinitrate spray. Longer acting nitrates such as isosorbide mononitrate improve exercise tolerance and time to onset of angina. They may be given by the oral or transdermal routes. These agents should be continued through the intraoperative period.
  - *Calcium channel blockers*—also used as first line therapy for angina and are particularly indicated in patients with evidence of vasospastic angina. These drugs should be continued through the intraoperative period.
  - *Beta-blockers*—reduce heart rate and contractility, reduce myocardial oxygen demand and delay or prevent the onset of angina.
- *Perioperative beta blockade (BB):* beta blockers relieve the symptoms of angina and long-term BB improves survival following myocardial infarction and in patients with HF. Discontinuing BB in the perioperative period is associated with adverse outcome and beta-blockers prescribed for any of these indications should be continued through the perioperative period. The POISE study showed no survival benefit and some evidence of harm from de novo BB in surgical patients. However, patients taking BB did have a reduced incidence of cardiac events and there is evidence that heart rate control is associated with a reduced risk of myocardial injury in some high risk groups. Thus, initiating treatment with BB before surgery in patients without an explicit indication for this treatment does not appear to reduce perioperative risk. However, it may be appropriate to use BB to control intraoperative heart rate and prevent myocardial ischaemia. This requires real-time heart rate monitoring and ideally ST-segment monitoring.

Percutaneous coronary intervention

PCI involves the angioplasty of coronary stenosis and the placement of either BMS or DES. The perioperative management of patients who have undergone PCI is discussed in 📖 Management of the vascular surgery patient after coronary revascularization, p. 173. PCI may be performed acutely for ST-segment or non-ST segment myocardial infarction. In patients with stable angina, PCI provides symptom relief with better control of ischaemic symptoms than with medical therapy. However, there is no clear evidence of a survival benefit and some evidence of a greater incidence of ischaemic cardiac events in follow-up after PCI compared with patients receiving medical treatment. Overall, medical therapy is the preferred option except if:

- There is a change in symptom severity.
- Medical therapy has not achieved adequate symptom control.
- There is high risk coronary anatomy.
- There is worsening left ventricular function.

Coronary artery bypass grafting

In patients with multi-vessel disease CABG offers comparable outcomes with PCI in terms of survival and freedom from MI. However, a number of studies indicate that patients who have undergone PCI are more likely to require further interventions than those undergoing CABG. For diabetic patients and those aged over 65yrs CABG offers lower mortality and is the preferred option.

There is no evidence to support prophylactic coronary revascularization (see 📖 Management of the vascular surgery patient, p. 173) before non-cardiac surgery. PCI or CABG before non-cardiac surgery should only be undertaken if there is a 1° indication for revascularization.

Further reading

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Heart failure

Diagnosis and treatment

- HF is a major predictor of cardiovascular complications following non-cardiac surgery and is associated with a significantly greater risk of perioperative cardiovascular complications than is the presence of CAD.
- HF can be diagnosed using the Framingham criteria and classified using the New York Heart Association (NYHA) or ACC/AHA criteria (Tables 4.1a and 4.1b).

Framingham criteria

- The Framingham criteria for the clinical diagnosis of heart failure were published by McKee and colleagues in 1971. The symptoms and signs of heart failure are divided into major criteria and minor criteria. The diagnosis of heart failure requires the presence of either two major criteria or one major and two minor criteria.
- The major criteria include symptoms and signs that are relatively (but not absolutely) specific to heart failure. They include paroxysmal nocturnal

Table 4.1(a) NYHA classification of HF is in widespread use and is based on the functional limitation of the patient

Class	Functional capacity
I	Patients without limitation of physical activity
II	Patients with slight limitation of physical activity, in which ordinary physical activity leads to fatigue, palpitation, dyspnoea, or anginal pain; they are comfortable at rest
III	Patients with marked limitation of physical activity, in which less than ordinary activity results in fatigue, palpitation, dyspnoea, or anginal pain; they are comfortable at rest
IV	Patients who are not only unable to carry on any physical activity without discomfort, but who also have symptoms of HF or the anginal syndrome even at rest; the patient's discomfort increases if any physical activity is undertaken

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Table 4.1(b) The American College of Cardiology and the American Heart Association (ACC/AHA) classification of HF. This describes the development and progression of HF and leads the clinician to consider an individual patient's natural disease progression.

Stage	Description	
A	At high risk for HF, but without structural heart disease or symptoms of HF	Equivalent to NYHA class I HF
B	Structural heart disease, but without sign or symptoms of HF	
C	Structural heart disease with prior or current symptoms of HF	Equivalent to NYHA class II or II HF. Most patients presenting for elective surgery who have a diagnosis of HF will be in at this stage.
D	Refractory HF requiring specialized interventions	Interventions for patients at this stage include mechanical circulatory support, transplantation, and end of life care. These patients are unlikely to be candidates for non-cardiac surgery

Reproduced from SA Hunt et al., 'ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult', Circulation, 112, 12, pp. 154–235, Copyright 2005, with permission from American Heart Association and Wolters Kluwer.

dyspnoea, a history of acute pulmonary oedema and the presence of rales. These are a cause for concern in any patient presenting for anaesthesia and surgery. Similarly, an elevated systemic venous pressure (including neck vein distension, a central venous pressure of greater than 16 cm of water, or the presence of a hepato-jugular reflex) are major criteria. Also included as major criteria are radiographic cardiomegaly, a "gallop" cardiac rhythm, and an increased circulation time.

- The minor criteria for heart failure include symptoms and signs that may be attributed to heart failure but are also commonly caused by other conditions. These are nocturnal cough, dyspnoea on exertion, a one third decrease in vital capacity, persistent tachycardia, hepatomegaly and peripheral oedema.

Perioperative management

- The aims of perioperative management are:
  - To ensure that HF is optimally treated before surgery.
  - To avoid exacerbations of HF and in particular an episode of acute HF during and after surgery.
  - To ensure that the patient is discharged on appropriate treatment for HF with appropriate follow-up.
- A number of co-morbidities associated with HF have a direct impact on prognosis and care should be taken to ensure that these are adequately treated before surgery.
  - *Coronary artery disease and valvular heart disease:* patients presenting for surgery with diagnosis of HF will have generally been investigated appropriately for these conditions. However, the anaesthetist must be aware of the role of both in the aetiology of HF.
  - *Sleep apnoea* has an increased prevalence in patients with HF and is associated with increased long-term mortality. Questions regarding sleep apnoea should be included in the preoperative assessment and if the condition is suspected the patient should be managed appropriately.
  - *Anaemia is common in chronic HF:* there is limited evidence on the optimal perioperative management, but there is some evidence to support the use of iron supplements. These should be given before elective surgery if time allows. Ideally, the patient should come to surgery with a haematocrit >28%.
  - *Renal impairment is associated with HF and is described as cardio-renal syndrome:* a number of therapies have been proposed for this condition, but there is little evidence specific to the perioperative management of HF patients with renal impairment. The perioperative care for patients with renal impairment described in 📖 Perioperative renal protection, p. 236. Renal dysfunction is appropriate, with the proviso that perioperative fluid management in patients with both ACC/AHA stage 'C' HF and renal impairment can be very challenging. Excessive administration of IV fluids may lead to pulmonary oedema whilst hypovolaemia or hypotension may worsen renal function. Monitoring of central venous pressure and cardiac output is therefore recommended for major vascular procedures.
  - Many patients with HF have atrial fibrillation and this should be assessed and treated as described in 📖 Arrhythmias, pacemakers and implantable cardiac defibrillators, p. 178.
  - Patients with advanced HF may have cardiac cachexia. If elective surgery is deemed appropriate in such a patient a period of calorie supplementation before operation may be of value.

- The management of HF may include dietary restriction of Na to 2–3g/day. Patients with hyponatraemia may be fluid restricted to 2L/day. Perioperative fluid and electrolyte losses should be replaced, but every effort should be made to avoid the administration of more Na or water than is required. Fluid balance should be closely monitored, blood electrolytes measured at least daily and invasive cardiovascular monitoring continued into the postoperative period if appropriate (📖 Cardiac output monitoring, p. 259).

Pharmacological therapy

- The main therapies in HF are loop diuretics, ACE inhibitors, beta blockers. Vasodilators are also used in some patients.
- *Loop diuretics:* in general loop diuretics should be continued before and after surgery, although some practitioners omit the dose on the morning of surgery because of preoperative starvation and to spare the patient the inconvenience of a drug induced diuresis. Loop-diuretics should be restarted when clinically appropriate after surgery, dictated by the magnitude of the surgery and the patient's fluid balance.
- *ACE inhibitors*—improve survival and reduce symptoms in HF and treatment should be maintained before surgery. Alternatively, ARBs may be used to block the renin-angiotensin-aldosterone system. Patients receiving ACE inhibitors for HF are less likely to be hypovolaemic than those treated with ACE inhibitors for hypertension; hence, hypotension at induction of anaesthesia may be less of a concern. For this reason, many practitioners continue treatment with ACE inhibitors up to and including the morning of surgery in patients with HF.
- Cardioelective beta-blockers such as bisoprolol and metoprolol suppress autonomic sympathetic over activity and reduce heart rate. These drugs should be continued until the day of surgery and restarted as soon as clinically appropriate after surgery.
- Vasodilators, including nitrates and hydralazine, may be used in patients who are unable to tolerate ACE inhibitors or angiotensin receptor blockers. They may also be used as add-on therapy in more severe HF and, as such, identify the patient as being at high risk of post-operative complications. Such patients require extra caution and will benefit from high dependency care following surgery. Similar considerations apply to the use of thiazide and thiazide-like diuretics, and aldosterone antagonists. These are used to increase diuresis in patients with a poor response to loop diuretics.
- Sudden cardiac death is 5–10 times more common in patients with HF than in the general population. Patients with an ejection fraction of <35% may require an implantable cardioverter-defibrillator. Guidance on the management of patients who have these devices *in situ* is given in 📖 Arrhythmias, pacemakers, and implantable cardiac defibrillators, p. 178.
- Patients with HF and intraventricular conduction delay (QRS>120ms) may benefit from cardiac resynchronization therapy with a biventricular pacemaker. Anaesthetist should seek specialist cardiological advice

regarding management of these devices during intraoperative period. However, they can produce substantial improvements in cardiac performance and should not, in general, be disabled for surgery.

Further reading

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Valvular heart disease

Diagnosis and management

- Echocardiography should be performed before surgery in patients suspected on the basis of clinical history or symptoms of having valvular heart disease. Echocardiography is also indicated in patients with known valvular disease in whom no recent assessment of valve and ventricular function has been made or in whom there has been a recent change of symptoms.
- In patients who have undergone cardiac valve repair or replacement details of the procedure performed postoperative valve and ventricular function should be obtained where possible.
- A number of guidelines have been published on the perioperative management of patients with valvular heart disease including those from the ESC and the ACC/AHA.

Key points

Aortic stenosis

- Aortic stenosis is noted in the European Society of Cardiology guidelines for Perioperative Cardiac Care to be the most common form of valvular heart disease in Europe. Severe aortic stenosis is defined as a valve area of less than 1cm^2 or less than 0.6cm^2/m^2 of body surface area.
- If the patient is symptomatic with angina, syncope, or dyspnoea consideration should be given to aortic valve replacement before elective vascular surgery.

- If surgery is to be performed without valve replacement the patient should undergo invasive haemodynamic monitoring during and after surgery. Tachycardia limits the time for ventricular ejection and should be avoided. Hypotension is a particular hazard and an episode of hypotension may precipitate irretrievable cardiovascular collapse.
- It has previously been considered that neuro-axial blockade is contraindicated in patients with aortic stenosis. Many practitioners now take the view that epidural anaesthesia may be used, but only with close invasive cardiovascular monitoring and gradual incremental top-up of the block.
- The combination of severe aortic stenosis and left ventricular failure is particularly pernicious. Contractile reserve may be assessed with dobutamine and patients with limited or no reserve may not be considered candidates for aortic valve replacement. Latterly, transcatheter aortic valve implantation (TAVI) has offered a route for aortic valve repair in these patients and this may be performed before non-cardiac surgery. However, it must be assumed these patients are still high-risk candidates for non-cardiac surgery even after TAVI because of the underlying ventricular impairment.

Mitral stenosis

- Non-cardiac surgery, including vascular surgery, generally carries an acceptable risk in patients with mild to moderate mitral stenosis (valve area >1.5cm^2) and in those patients with significant mitral stenosis who do not have pulmonary hypertension (systolic pulmonary artery pressure <50mmHg). Heart rate control is important and tachycardia should be avoided. Particular concerns are fluid overload and the atrial fibrillation, both of which may precipitate sudden deterioration.
- Patients who have significant mitral stenosis and pulmonary hypertension should be considered for valve repair or replacement before surgery where possible.

Aortic and mitral regurgitation

- The risk of non-cardiac surgery is not increased in patients with non-significant aortic or mitral regurgitation. Similarly, severe asymptomatic aortic or mitral regurgitation in patient who are asymptomatic and have preserved LV function may be undertaken with consideration being given to preoperative cardiac valve repair or replacement. Perioperative bradycardia may increase aortic regurgitation by prolonging diastole and should be avoided if possible.
- Symptomatic patients and asymptomatic patients with left ventricular impairment (ejection fraction (EF) < 30%) are at increased risk of perioperative complications. Elective surgery should only be undertaken if essential.

Patients with prosthetic heart valves

- Patients who have undergone cardiac valve repair or replacement and who do not have left ventricular impairment are not at increased risk of perioperative complications.
- Patients with a non-biological valve may be on long-term anti-coagulation. This should be replaced by an unfractionated heparin infusion or therapeutic doses of low molecular weight heparin prior and may be discontinued before surgery and restarted as soon as deemed surgically acceptable (see 📖 The anti-coagulated patient, p. 215). The risks of a period without anticoagulation are greater for patients with mitral valve replacement than for those with aortic valve replacement as the lower blood flow velocity over the mitral valve increases the risk of thromboembolism.

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Management of the vascular surgery patient after coronary revascularization

- Unstable CAD is a major risk factor for perioperative cardiovascular morbidity and mortality after non-cardiac surgery.
- Perioperative cardiovascular risks are reduced in patients who have already undergone corrective coronary revascularization.
- However, there is no benefit from *prophylactic* coronary revascularization before elective major vascular surgery.
- Indications for coronary revascularization are based on severity of any cardiac disease. Revascularization is not performed solely to improve the outcome of subsequent vascular surgery.

Coronary artery bypass grafting

- Main indications for CABG are left main stem disease or triple vessel disease with left ventricular dysfunction.
- The cardiovascular risks of vascular surgery are reduced after prior CABG, especially if CABG has been performed within the previous five years and the patient has remained symptomatically stable.
- However, 1-yr mortality is increased in patients who undergo CABG compared with those treated medically. Some data suggest that morbidity and mortality are actually increased in patients who undergo non-cardiac surgery early (within 1–2 months) after CABG.

- There is no consensus on the optimum timing of non-cardiac surgery in patients who have undergone CABG. Where possible, major vascular surgery should be delayed for at least 6 weeks, and preferably 3 months, depending on the urgency for vascular surgery.
- If vascular surgery is required within 3 months of CABG, close attention to functional status, symptoms, investigations, and management are required. Invasive perioperative monitoring and post-operative critical care may be warranted for all, but the most minor procedures.

Percutaneous coronary interventions

- The clinical indications for PCI are:
  - Acute STEMI.
  - NSTEMI.
  - Stable angina.
  - Symptoms equivalent to angina (e.g. dyspnoea, arrhythmia, dizziness/syncope).
  - Evidence of a moderate to large area of viable myocardium or moderate to severe ischaemia on non-invasive testing.
- The angiographic indication for PCI is a haemodynamically significant lesion in a vessel with a diameter greater than 1.5mm serving viable myocardium.
- Angiographic contraindications to PCI are:
  - Left main stenosis in a patient who is a candidate for cardiac surgery.
  - Diffusely diseased small-calibre artery or vein graft.
  - Other coronary lesions not amenable to percutaneous intervention and which require surgery.
- PCI includes coronary angioplasty alone, but now most PCI procedures involve the deployment of coronary artery stents, which reduce the incidence of restenosis compared to angioplasty alone.
- Coronary artery stents may be BMS or DES. All stents carry a risk of in-stent thrombosis, especially early after intervention before re-endothelialization of the stent has occurred and can be catastrophic.
- DES are increasingly preferred as they have a lower rate of in-stent restenosis. They have polymers on the stent struts which contain anti-mitotic agents (e.g. sirolimus, tacrolimus, paclitaxel), which inhibit early endothelial overgrowth, but leave the stent struts uncovered for a longer period. Hence more prolonged antiplatelet therapy is required in patients who receive DES as compared with those who have BMS placed or angioplasty alone.
- As with CABG, there is no benefit in performing PCI before non-cardiac surgery in patients with coronary disease with the sole aim of preventing perioperative cardiac events. Preoperative PCI should only be considered in those patients in whom coronary intervention is indicated regardless of the need for non-cardiac surgery. For an acute coronary syndrome. If anything the benefits in decreasing perioperative cardiac morbidity following non-cardiac surgery are less with PCI as compared with CABG.

- Management of patients who have undergone PCI differs from those after CABG in several respects, the most important being the requirement for prolonged antiplatelet therapy to prevent stent thrombosis.
- Up to 5% of patients receiving intracoronary stents (ICS) will require non-cardiac surgery within 12 months. These patients are at particular risk of increased mortality and morbidity; they face the increased risk of perioperative bleeding if antiplatelet therapy is continued or acute thrombotic events if it is interrupted.
- The management of patients needing subsequent vascular surgery depends on the type of PCI, the required antiplatelet therapy, and the interval since PCI was performed.

Antiplatelet therapy after PCI

Low dose aspirin increases perioperative bleeding by 2–20%, but without increasing morbidity or mortality, and the effects depend on the procedure. Dual aspirin and clopidogrel therapy increases surgical bleeding and the transfusion rate by about 50%, but with the exception of neurosurgery, this probably does not increase morbidity or mortality. Current recommendations for antiplatelet therapy after PCI are:

- Aspirin 325mg for 1 months after BMS, 3–6 months after DES, then 75–162mg indefinitely.
- In addition, clopidogrel 75mg for 1 month after BMS, 3 months after sirolimus DES, and 6 months after paclitaxel DES, and ideally up to 12 months if the patient is not at increased risk of bleeding.
- If patients are considering undergoing surgery within 12 months of a planned PCI, consideration should be given to balloon angioplasty with a provisional stent or a BMS, rather than a DES.

Bare metal stents

- BMS become endothelialized within the first 6 weeks. Hence, dual antiplatelet therapy is obligatory for 6 weeks after the cardiac intervention to prevent sub-acute stent thrombosis.
- Elective surgery is best deferred for a minimum of 6 weeks and optimally 3 months after BMS.
- If surgery is required within this period then ideally, from a cardiological perspective, dual anti-platelet therapy should be continued through the perioperative period.
- The clinical dilemma in this case is between the risk of coronary stent thrombosis if dual anti-platelet therapy is discontinued and the risk of bleeding if dual anti-platelet therapy is continued.
- In practice, decisions regarding perioperative anti-platelet therapy should be made on a case by case basis and should involve discussion between the cardiologist, the surgeon, and the anaesthetist.
- Three months or more after PCI with deployment of a BMS surgery can be conducted with continuation of aspirin therapy alone.

Drug eluting stents

- The use of DES increased rapidly after their introduction because of their ability to reduce the endothelialization process leading to significantly reduced rates of re-stenosis.
- However, the same processes that reduce the risk of re-stenosis also slow re-endothelization so that the metal stent struts of the stent can remain exposed to the circulating blood for up to 1yr after stent deployment. Rarely the stent can remain exposed for even longer and this is responsible for late stent thrombosis.
- If dual anti-platelet therapy is interrupted before the stent is covered by endothelium there is an increased risk of thrombotic stent occlusion. Therefore surgery should be deferred for at least 12 months after DES if at all possible. After 12 months, surgery can be undertaken with continuation of aspirin alone.
- Decisions regarding antiplatelet therapy in patients with a DES in situ who require operation within 12 months of PCI should be made on a case by case basis. The cardiologist, anaesthetist, and surgeon should reach a consensus on the timing and use of anti-platelet agents. Vascular surgical patients who are taking aspirin should generally remain on this drug through the perioperative period regardless of the presence of a coronary stent.
- If the anti-platelet effects of clopidogrel require reversal it should be stopped 10 days before surgery to achieve a full return of normal platelet activity. Stopping clopidogrel within 5 days will reduce anti-platelet effects by >60%. In cases of urgent surgery where bleeding occurs supportive transfusions of platelets may be given, but no specific reversal agents exists for clopidogrel as it remains irreversibly bound to the platelets.

Urgent PCI

- When PCI is performed urgently (unstable angina or MI), unfractionated heparin is used and other agents such as 2B/3A antagonists or bivalirudin may be required.
- Pre-loading of patients with high doses of aspirin (300mg), clopidogrel (300–600mg) or the newer agents (prasugrel 60mg, tricagrelor 180mg) are used to induce an immediate anti-platelet effect.
- The need for high dose anti-platelet agents following PCI limits the use of this intervention in patients who have suffered a perioperative myocardial infarction. In many cases the risks of bleeding from the site of surgery are considered to outweigh the potential benefits of PCI.

Recommendations on the timing of non-cardiac surgery in patients with prior cardiac revascularization

Current recommendations (Table 4.2) are to defer all non-cardiac surgery for at least 2 weeks and, ideally, at least 4–6 weeks after the placement of an intracoronary stent. Non-urgent surgery should be deferred for 3 months after CABG where possible.

Table 4.2 Current recommendations for elective surgery after coronary intervention

Procedure	Timing of subsequent vascular surgery	Management of medication
CABG within 3 months	Defer for at least 6 weeks and, ideally, for 3 months, depending on urgency	Continue current medications
CABG within 3 months–5yrs	Proceed with surgery if the cardiac disease is stable	Continue current medications
PCI (angioplasty alone)	Defer for 2 weeks unless emergency	Continue dual antiplatelet therapy for 2–4 weeks after the coronary intervention and aspirin indefinitely.
PCI and BMS	Postpone urgent surgery for 6 weeks or more if possible Postpone elective surgery for ≥3 months	Continue dual antiplatelet therapy for 4–6 weeks and aspirin indefinitely.
PCI and DES	Defer elective surgery for ≥12 months if possible	Continue dual antiplatelet therapy for 12 months after PCI and aspirin indefinitely.

All patients should continue aspirin throughout the perioperative period.

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Arrhythmias, pacemakers, and implantable cardiac defibrillators

Arrhythmias

Cardiac arrhythmia is a generic term for a large number of conditions of disordered electrical activity of the heart. Arrhythmias range from the normal phenomenon of sinus arrhythmia through to immediately life-threatening ventricular fibrillation. Arrhythmias are common in patients presenting for, or undergoing vascular surgery.

- *Arrhythmias* can be classified according to:
  - *Rate*—brady- or tachyarrhythmias.
  - Mechanism of generation.
  - *Site of origin*—atria, pulmonary veins, nodes, ventricles.
- *Atrial:*
  - Sinus arrhythmia.
  - Atrial fibrillation (AF).
  - Atrial flutter.
- *Junctional:*
  - Supraventricular tachycardia (SVT).
  - AV nodal re-entrant tachycardia.
  - Junctional tachycardia.
- *Atrio-ventricular:*
  - Heart block.
  - Wolff–Parkinson–White syndrome.
- *Ventricular:*
  - Ventricular tachycardia (VT).
  - Ventricular fibrillation (VF).
  - Premature ventricular contractions (PVC).
- Arrhythmias may be associated with structural heart disease, but there are many other precipitants and often no other detectable cardiac disease. See Table 4.3.

  Patients with cardiac arrhythmias may be asymptomatic, but common symptoms include:
- Palpitations.
- Heart failure: acute or chronic.
- Myocardial ischaemia with dyspnoea or chest pain.
- Cardiovascular collapse and cardiac arrest.

Common arrhythmias

Sinus arrhythmia

- This is a normal physiological change in heart rate in response to respiration.
- Increased vagal parasympathetic activity during expiration causes a fall in heart rate.
- Decreased vagal activity during inspiration leads to an increase in heart rate.
- The response compensates in part for reduced stroke volume as a consequence of reduced left ventricular filling during inspiration.

Table 4.3 Precipitants and causes of arrhythmias

Cardiac conditions	Metabolic	Endocrine	Pharmacological
Myocardial ischaemia	↑K^+ or ↓K^+	Thyrotoxicosis	Anti-arrhythmics
Myocardial infarction	↑Ca^{2+} or ↓Ca^{2+}	Phaeochromocytoma	Sympathomimetics
Valvular heart disease	↓Mg^{2+}		Antidepressants
LV aneurysm	↓PaO_2		Aminophylline
Congenital	↑$PaCO_2$		Caffeine
Post-cardiac surgery	Acidosis		Alcohol
Pre-excitation syndromes			
Long QT			

- Changes in vagal activity have been demonstrated to continue during paralysis of respiration, indicating a central reflex mechanism.

Heart block

- Consequence of failure of transmission of electrical excitation from the atria to the ventricles.
- Usually caused by ischaemia or fibrosis in or around the AV node or bundle of His.

  Heart block is classified by degree
- *First degree heart block:* slowed conduction between the AV node and ventricles manifest as a PR interval of >0.2s
- *Second degree heart block:*
  - Intermittent failure of transmission of excitation from the atria to the ventricles.
  - Type1 (Mobitz I or Wenckebach).
  - Progressive PR prolongation followed by non-conducted P-wave.
  - Type 2 (Mobitz II).
  - No prolongation or shortening of PR interval.
- *Third degree (complete) heart block:*
  - Complete failure of transmission.
  - Atria and ventricles beat at independent rates. Often the ventricular rate is bradycardic.
  - Requires assessment for perioperative pacing or long-term permanent pacemaker.

Atrial fibrillation

- This is the commonest cardiac arrhythmia in the UK.
- The incidence increases with age:
  - 5% above 65yrs.
  - 10% above 75yrs.
- It is defined by the absence of organized atrial activity.
- AF is diagnosed by the complete absence of p-waves on the electrocardiogram.
- The ventricular response is irregular and often rapid.

- AF commonly co-exists with other conduction abnormalities, e.g. bundle branch block or accessory conduction pathways.
- Classification of AF is based on patterns of occurrence:
  - *First Onset*—new presentation of an ongoing episode of AF within 48h.
  - *Paroxysmal*—recurrent self-terminating episodes of AF usually lasting minutes to hours.
  - *Persistent*—not self-terminating AF requiring pharmacological or electrical cardio-version.
  - *Permanent*—failure of all attempts to restore sinus rhythm.

See Table 4.4 for a list of causes of AF and Table 4.5 for investigations.

Consequences of atrial fibrillation, especially with a rapid ventricular rate include:

- Significant impairment of cardiovascular function.
- HF.
- Exacerbation of IHD.

Management of arrhythmias

The management of arrhythmias should be focused on urgent assessment and management of the patient in accordance with appropriate guidelines (℔ www.resus.org.uk)

Specific management falls into four main areas:

- Restoration of sinus rhythm.
- Control of ventricular rate.

Table 4.4 Causes of atrial fibrillation

Acute	Cardiovascular	Neurogenic
Surgery	Myocardial infarction	High sympathetic tone
Hyperthyroidism	Hypertension High vagal tone	
Alcohol excess	Valvular disease	
Pneumonia	Myocarditis/Pericarditis	
Pulmonary embolism	Congenital (atrial septal defect)	
Pulmonary hypertension	Sick sinus syndrome	
Diabetes		

Table 4.5 Investigation of AF

Essential	Further investigations
History and physical examination	Exercise testing
Electrocardiogram	24h ECG event monitoring
Chest X-ray	Transoesophageal echocardiogram
Trans thoracic echocardiogram	Electrophysiological studies
Thyroid function tests.	

- Prevention of recurrence of AF.
- Prevention of thromboembolic complications.

Perioperative management of the patient with an arrhythmia

- *If AF is identified in the perioperative period:* identify, investigate, and treat any precipitating factors.
- *It is reasonable to proceed with anaesthesia if:* ventricular rate is well controlled (<100beats/min) with no evidence of:
  - Haemodynamic instability.
  - Ischaemia.
  - HF.
  - Pulmonary embolism.
  - Pericarditis.
  - Thyrotoxicosis.
- A normal echocardiogram rules out structural heart disease.

Investigation and management of other common arrhythmias

Newly-presenting arrhythmias require rapid assessment of clinical and electrocardiographic (ECG) findings:

- Open airway, apply oxygen.
- Establish IV access.
- Is there a pulse?
- Is it a brady- or tachyarrhythmia?
- Are there broad or narrow QRS complexes?
- Is the ventricular response regular or irregular?

Long-term management of arrhythmias can comprise life style changes, drug treatment, cardiological electrophysiological measures, and cardiac surgery.

Drugs to treat arrhythmias

Drugs are used for the treatment and prevention of the arrhythmia, or for risk modification via heart rate control, coupled with appropriate anticoagulation. Antiarrhythmic drugs were classified by Vaughan Williams in 1970 (Table 4.6), based upon their mechanism of action. This classification has limitations in clinical practice, because it does not predict which drug to use for which arrhythmia, some drugs have more than one action, and some arrhythmias can be treated by drugs from more than one class.

Direct nodal inhibition: management of peri-arrest arrhythmias

Peri-arrest arrhythmias should be managed according to the current Resuscitation Guidelines available at: http://www.resus.org.uk/pages/periarst.pdf (Figs 4.4 and 4.5). Intraoperative bradycardia is usually treated with atropine 0.6mg IV (dose can be repeated up to 3mg) and stopping any vagal stimuli (e.g. surgical traction). Ephedrine 3–6mg increments is an alternative. SVTs are usually treated with adenosine, beta blockers, amiodarone, or digoxin. VTs are usually treated with lidocaine, amiodarone, or magnesium.

Table 4.6 Vaughan Williams classification of anti-arrhythmic drugs

Class	Examples	Mechanism
Ia	Quinidine	Na^+ channel blockers (intermediate)
	Procainamide	
	Disopyramide	
Ib	Lidocaine	Na^+ channel blockers (fast)
	Phenytoin	
	Mexilitine	
Ic	Flecainide	Na^+ channel blockers (intermediate)
	Propafenone	
	Moricizine	
II	Propranolol	β-adrenergic receptor blockers
	Metoprolol	
	Bisoprolol	
III	Amiodarone	K^+ channel blockers
	Sotalol	
	Ibutilide	
IV	Verapamil	Ca^{2+} channel blocker
	Diltiazem	
V	Adenosine	Other mechanisms
	Digoxin	

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Indications for permanent pacemakers

There are many indications for the implantation of permanent pacemakers and cardioverter-defibrillators.

Class I indications for permanent pacemakers

The following are recommended (by ACC/AHA/NASPE):

- Third degree or advanced second-degree heart block with any of:
  - Symptomatic bradycardia (including HF).
  - Arrhythmia.
  - Documented periods of asystole.
- After catheter ablation of AV junction.
- Post-operative AV block.
- Neuromuscular disease with AV block.
- Intermittent 3rd-degree AV block.
- Type II 2nd-degree AV block.
- Alternating bundle-branch block.
- Post-myocardial infarction infranodal AV block with bundle branch block or persistent symptomatic second or third-degree heart block.
- Sinus node dysfunction with symptomatic bradycardia and symptomatic pauses or symptomatic chronotropic incompetence.

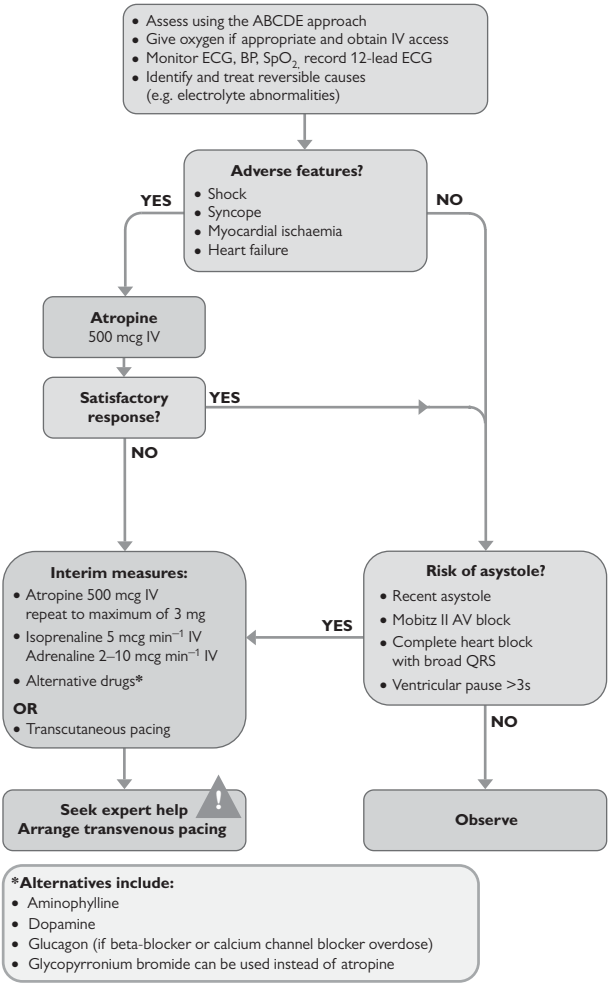

Fig. 4.4 Adult bradycardia algorithm.

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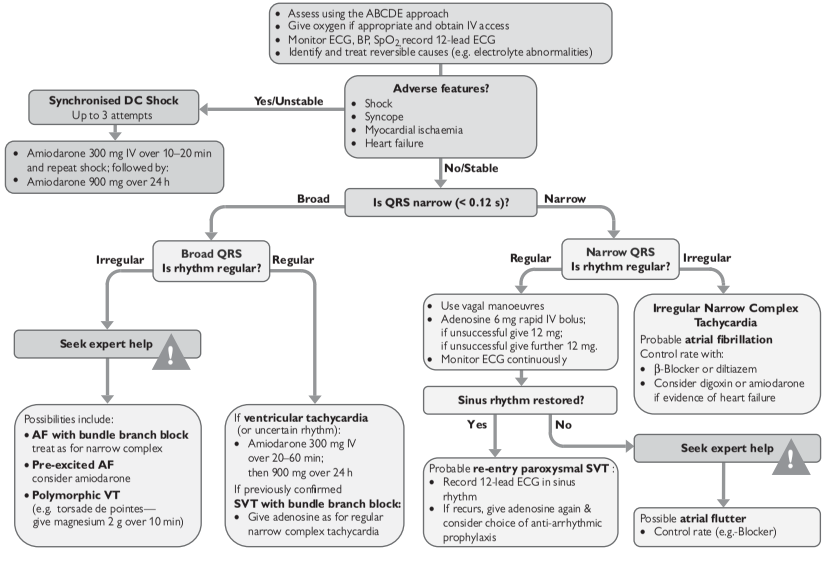

Fig. 4.5 Adult tachycardia (with pulse) algorithm.

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- Sustained VT, in which pacing efficacy is thoroughly documented.
- Recurrent syncope caused by carotid sinus stimulation.
- Symptomatic brady-arrhythmias following cardiac transplantation.

Classification of pacemakers

Pacemakers are classified according to a five-letter code produced by the North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG); see Table 4.7. The first three letters indicate anti-bradycardia functions; the fourth refers to programmability, and the fifth to anti-tachycardia functions. Modern pacemakers can usually be programmed to operate in different modes.

(Automatic) implantable cardiac defibrillators (ICDs)

Class I indications for ICD implantation

The following are recommended by NASPE:

- Cardiac arrest due to VF or VT not due to a transient or reversible cause.
- Spontaneous sustained VT with structural heart disease.
- Syncope with inducible sustained VT or VF, and failed drug therapy.
- Non-sustained VT with coronary disease, prior MI, LV dysfunction, and inducible VF or sustained VT not suppressible by a Class I antiarrhythmic drug.

Table 4.7 NASPE/BPEG generic (NBG) Pacemaker code

Position	Category	Letters
1	Chamber paced	O = None A = Atrium V = Ventricle D = Dual
2	Chamber sensed	O = None A = Atrium V = Ventricle D = Dual
3	Response to sensing	O = None T = Triggered I = Inhibited D = Dual
4	Programmability	O = None P = Simple programmable M = Multiprogrammable C = Communicating R = Rate modulation
5	Anti-tachyarrhythmia functions	O = None P = Pacing S = Shock D = Dual (P+S)

- Spontaneous sustained VT without structural heart disease, but not amenable to other treatments.

A further sub-classification has been recommended by the UK NICE in 2006.

- *Primary prevention:*
  - Previous MI and left ventricular ejection fraction (LVEF) <35% and demonstrated inducible VT.
  - Previous MI with LVEF <35% and QRS duration ≥120ms.
  - Familial condition with high risk of sudden death.
- *Secondary prevention:*
  - Survivors of cardiac arrest due to VT or VF.
  - Spontaneous sustained symptomatic VT.
  - Sustained asymptomatic VT with LVEF <35%.

The NASPE/BPEG Defibrillator (NBD) Code is shown in Table 4.8.

Biventricular cardiac resynchronization therapy (BiV-CRT)

HF is frequently complicated by cardiac conduction defects and impaired cardiac performance, including:

- QRS prolongation.
- Left bundle branch block (LBBB).
- Inefficient ventricular systolic function due to asynchronous contractions of the septal and lateral walls.

In a failing dilated left ventricle:

- The mitral valve annulus and papillary muscles are stretched.
- Apposition of the valve leaflets is impaired.

Table 4.8 The NASPE/BPEG Defibrillator (NBD) Code

Position	Category	Letters
1	Shock chamber	0 = None
		A = Atrium
		V = Ventricle
		D = Dual (A&V)
2	Anti-tachycardia pacing chamber	0 = None
		A = Atrium
		V = Ventricle
		D = Dual (A&V)
3	Tachycardia detection	E = Electrocardiogram
		H = Haemodynamic
4	Anti-bradycardia pacing chamber	0 = None
		A = Atrium
		V = Ventricle
		D = Dual (A&V)

- These lead to functional mitral regurgitation (FMR).
- Asynchronous papillary muscle contraction exacerbates functional mitral regurgitation.

Biventricular cardiac resynchronization therapy has been shown to improve cardiac output and efficiency, and to reduce functional mitral regurgitation. Modern BiV-CRT devices or 'triple chamber pacemakers' allow precise control of atrioventricular and interventricular timing delays and often include an ICD.

Indications for BiV-CRT (all must be present)
- Drug refractory, symptomatic NYHA class III-IV HF.
- Either ischaemic or non-ischaemic origin.
- Prolonged QRS complex (≥130ms).
- LV end-diastolic diameter of 55mm or greater.
- LV EF of 35% or less.

Causes for decompensation in BiV-CRT
- New onset AF.
- Lead dislodgement.
- Suboptimal atrioventricular or interventricular delay settings.

Implications for anaesthesia and surgery of pacemakers and similar devices

Preoperative assessment

Particular attention should be paid to the indication for treatment and date of insertion and outcome of subsequent follow-up.

Investigations
- ECG to establish pacemaker activity and/or underlying rhythm.
- CXR will confirm location of device and leads, and may aid diagnosis of HF.
- Serum electrolytes should be checked and corrected to minimize risk of loss of capture.

Monitoring
- Should be established before induction of anaesthesia.
- Most modern monitors will identify pacing activity.
- Invasive arterial BP monitoring gives real time confirmation of mechanical capture.
- Caution with central venous or pulmonary artery catheters and newly inserted pacemakers because of potential to dislodge wires.

Use of magnets

Magnets are no longer recommended for the routine management of a patient with a pacemaker or ICD because:
- Older non-programmable pacemakers could be switched by the magnet into a fixed rate mode.
- New pacemakers are almost always programmable and magnets may have an unpredictable effect.

Diathermy

The effects of electromagnetic interference on pacemakers have been diminished by the use of bipolar electrodes and shielding cases. Interference from diathermy can still cause problems:

- Inhibition of pacing.
- *Change in pacemaker program:* often to a fixed rate back-up mode.

The risks of diathermy can be attenuated by:

- Avoidance of diathermy if surgically possible.
- Use of bipolar (rather than monopolar) diathermy.
- Unipolar diathermy ground plate should be positioned as far away as possible from the pacemaker.
- Ensuring the diathermy current pathway runs at a 90° to pacemaker leads.

Perioperative pacemaker failure

Be prepared for the potential for complete failure of pacing. Emergency pacing may be required. Management includes:

- Precordial percussion (thumps).
- Transthoracic pacing via electrode pads is quick to set up and often available on hospital defibrillators.
- Transoesophageal pacing.
- Drugs (e.g. dobutamine) may help support an underlying rhythm until pacing can be commenced.

Post-operative care

Pacemakers should have a programme check after any operative procedure, especially if problems are suspected.

Further reading

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Recent stroke or transient ischaemic attack

Incidence and natural history

Stroke is the third commonest cause of death in the Western world.

- The incidence of stroke is >110 000 per year in England and Wales, including 87 000 first strokes and 54 000 recurrent strokes.
- 25% of these occur in people aged <65yrs.
- The incidence of TIA is 20 000 per year.
- There are approximately 60 000 deaths from stroke per year in England and Wales.
- Approximately 1/3 of acute strokes are fatal within 10 days, 1/3 lead to significant prolonged disability, and 1/3 of patients make a good recovery within 1 month.
- The risk of further disabling stroke after a TIA or minor stroke is 5% within 7 days and 10–20% within 1 month. The highest absolute risk (up to 5%) is within the first 24h.
- For these reasons it is now recommended that patients who have had a TIA or minor stroke should be treated urgently. They should ideally be assessed for CEA within 24h and it has been recommended that CEA performed within 48h if indicated (see 🕮 Anaesthesia for carotid surgery, p. 381).
- In some patients with previous stroke, CEA is not indicated, i.e. the stroke was not related to carotid atherosclerosis, or the degree of carotid stenosis at the time of assessment was <50%.

Whether or not CEA has been performed, patients with previous stroke frequently have widespread vascular disease and may present for other vascular or non-vascular surgery. The decision to undergo non-carotid surgery depends on the relative risks of the procedure, the extent of the stroke, other medical conditions, and the urgency of other surgery.

Pathophysiology of stroke or TIA

The causes of strokes and TIAs are:

- Ischaemia (70%).
- Primary intracerebral haemorrhage (13%).
- Subarachnoid haemorrhage (6%).
- Other uncertain aetiology (12%).

Acute ischaemic stroke is classified aetiologically by the TOAST criteria:

- *Large-artery atherosclerosis:* these account for <15% of all strokes, but carry the highest risk of early and medium term recurrent stroke. These patients benefit most from early CEA when significant carotid stenosis is present.
- *Small-vessel occlusion:* this carries the lowest risk of recurrent stroke, but a high risk of vascular cognitive impairment.
- *Cardio-embolic:* emboli from other sources, mainly cardiac, e.g. due to atrial fibrillation, aortic or mitral valve disease, prosthetic valves, infective endocarditis, left atrial myxoma, dilated cardiomyopathy or mural thrombus, e.g. after myocardial infarction.
- Other determined cause, e.g. vasculopathies, hypercoagulable states.
- Undetermined cause.

Diagnosis of stroke and TIA

15% of patients with a stroke have had a preceding TIA. Diagnosis depends on the symptoms and their duration. The risk of stroke after TIA can be assessed using the ABCD2 score (Table 4.9).

Symptoms and signs of stroke or TIA

These vary according to the vascular territory affected:

- *Middle cerebral artery or internal carotid artery:* contralateral hemiplegia and sensory loss of the face, arm, and leg. Dysphasia may occur especially if the dominant hemisphere is involved.
- *Anterior cerebral artery:* contralateral hemiplegia affecting leg > arm. Apraxia, motor dysphasia, micturition disturbances.
- *Posterior cerebral artery:* contralateral homonymous hemianopia with macular sparing. Contralateral diffuse burning pain if thalamus involved.
- *Vertebral and basilar arteries:* diplopia, divergent gaze, oculomotor palsy, hemiplegia or quadiplegegia, vertigo, loss of consciousness. Pinpoint pupils occur in pontine stroke.
- *Amaurosis fugax:* with or without contralateral hemiparesis suggests carotid territory involved.
- The long-term risks of stroke are lower after isolated monocular TIAs compared with after carotid artery territory TIAs.

The Oxford community stroke classification (Table 4.10) is widely used and relates to outcome.

Medical management after TIA or ischaemic stroke

Patients should be admitted to a dedicated acute stroke unit.

- Immediate assessment includes GCS and blood glucose. Hyperglycaemia is frequent: target blood glucose of 4–11mmol/L has been recommended, although this is controversial; hypoglycaemia is an important mimic of acute stroke and should be treated immediately.

Table 4.9 ABCD2 score

		Points
A	Age >60yrs	1
B	BP >140/90	1
C	Clinical features	
	Unilateral weakness	2
	Speech impairment without weakness	1
D	Duration >60mins	2
	Duration 10–59min	1
D	Diabetes mellitus	1

The ABCD2 score can predict the incidence and severity of major strokes or other events in the first few days after TIA. A low score (<4) is associated with 2 and 7 day risks of major stroke of 1% and 1.2% respectively but also a higher 7 day risk of recurrent TIA. A high score (>5) is associated with 2 and 7 day risks of major stroke of 8.1% and 11.7% respectively.

Table 4.10 Oxford system of stroke classification:

Total anterior circulation stroke (TACS)	All three of:	Contralateral motor or sensory deficit Homonymous hemianopia Higher cortical dysfunction*
Partial anterior circulation stroke (PACS)	Two of:	Contralateral motor or sensory deficit Homonymous hemianopia Higher cortical dysfunction
Posterior circulation stroke (POCS)	Any one of:	Isolated homonymous hemianopia Brain stem signs Cerebellar ataxia
Lacunar stroke (LACS)	Any one of:	Pure motor deficit Pure sensory deficit Sensorimotor deficit

* Includes dysphasia/visiospatial disturbance.

TACS involve both cortical and subcortical infarction and have a poor outcome; PACS are predominantly cortical infarcts with a high risk of recurrent stroke. POCS involve vertebrobasilar territory and although the risk of recurrent stroke is high overall outcome is better. LACS arise from deep perforating arteries (anterior or posterior circulation), and may be small in size, but functional outcome is often poor.

- Urgent CT or diffusion weighted MRI scan should be performed within 24h.
- IV Thrombolysis with alteplase (recombinant tissue-type plasminogen activator, r-tPA) (0.9mg/kg, maximum dose 90mg), within 4.5h where there is documented ischaemic stroke and no contraindications (Table 4.11). Note angioedema occurs in approximately 5% of patients after rt-PA. It is usually mild, can be delayed in onset and typically affects the ipsilateral half of the tongue initially. Incidence is increased in patients receiving ACE inhibitors.
- Hemicraniectomy is sometimes performed to alleviate the effects of massive cerebral oedema in cases of malignant stroke affecting the middle cerebral artery territory (e.g. superficial lobar haemorrhage >3cm diameter with Glasgow Coma Score (GCS) <13).
- Posterior fossa or superficial lobar haemorrhage or haematoma >3cm, or hydrocephalus with associated neurological deterioration (GCS <13) are indications for surgical decompression.
- Clot retrieval or arterial thrombolysis should only be considered within the context of a clinical trial.
- Documented intracranial haemorrhage associated with coagulopathy should be treated with Prothrombin complex. Vitamin K 10mg IV may be indicated if the international normalized ratio (INR) is >2.5 related to warfarin therapy.
- Swallowing, nutrition, and hydration should be assessed early.
- Common infections (e.g. urinary tract, pneumonia) should be actively sought and treated.
- Deep vein thrombosis (DVT) prophylaxis (e.g. dalteparin 2500 sc) should be considered after the acute phase.

Early secondary prevention of stroke after TIA or minor stroke

- Aspirin 300mg od po (or via NGT, rectally if unable to swallow) as soon as possible & certainly within 24h. Continue aspirin 300mg od for 2 weeks. Withhold aspirin for 24h after thrombolysis.
- Alternatives, e.g. oral clopidogrel (75mg od), should be considered in patients unable to take aspirin.
- In patients with TIA, minor stroke, or early spontaneous recovery, immediate diagnostic work-up, including urgent vascular imaging (US, CT angiography, or MRA), is recommended.

BP management

- Hypertension occurs in 80% of patients in the first few days after TIA or minor stroke, but decreases spontaneously thereafter. Both hypotension and hypertension are associated with a poor outcome after stroke, but optimum BP management in terms of drug therapy or BP targets is controversial.
- Cerebral autoregulation is impaired; hypotension could worsen cerebral ischaemia and hypertension could cause cerebral oedema or haemorrhage. This may be less likely after minor stroke or TIA than after major stroke.
- Routine antihypertensive treatment in the acute phase after TIA or stroke is not currently recommended. However, a target BP of <180/110 has been suggested, particularly in the context of intracerebral haemorrhage or patients undergoing thrombolysis. Further trials of more aggressive BP reduction are ongoing.

Later secondary prevention

The following are indicated after the acute phase (2 weeks):

- Clopidogrel 75mg od after minor stroke.
- Aspirin 75mg combined with dipyridamole 200mg modified release bd after TIA.
- Hypertension should be treated (target <140/90). Diuretics, Ca^{2+} channel blockers, and ACE inhibitors are often preferred.
- Statins are recommended for life to maintain LDL cholesterol <100mg/dL.
- Warfarin is only indicated in patients with AF. Target INR is 2.0–3.0.
- Newer alternatives include Factor Xa inhibitors (rivaroxaban) or direct thrombin inhibitors (dabigatran), but long-term data are not available.
- CEA is indicated in patients with a CEA stenosis >50% (see 🕮 Carotid artery surgery and stenting, p. 377).

Consideration for surgery- medical factors

Patients presenting with a history of stroke have an increased incidence of the following conditions, which may have separate implications for perioperative management:

- Previous stroke or TIA (30%).
- Old age.
- AF (15–20%).

- Congestive HF.
- CAD, peripheral occlusive vascular disease.
- Hypertension.
- Cigarette smoking.
- COPD.
- Male gender.
- Hypercholesterolaemia.
- Diabetes.
- Obesity.
- High alcohol intake.
- Sedentary lifestyle/physical inactivity.

The annual stroke rate in patients with AF is 4–5%, causing death or permanent disability in 50% of cases. The risks of stroke in patients with AF are increased when other risk factors are present; control of BP has additional benefits in patients with AF.

Preoperative assessment in patients with a history of stroke

In the early period after stroke, baroreceptor function is impaired leading to cardiovascular instability. Other problems may include impaired conscious level, increased sensitivity to sedative or anaesthetic drugs, impaired swallowing or airway reflexes. Therefore non-urgent surgery should be deferred for approximately 3 months (Table 4.12).

In addition to the high incidence of co-existing medical conditions, there are some specific risks of anaesthesia and surgery after recent stroke or TIA:

- Cerebral blood flow may be impaired by pre-existing carotid atherosclerosis.
- Cerebral blood flow autoregulation is impaired for several weeks or months after stroke or TIA.
- Risks related to the physical consequences of the stroke (immobility, poor nutrition coughing, impaired respiratory function).

Table 4.11 Absolute and relative contraindications to thrombolysis after ischaemic stroke

Absolute	Relative
Rapidly improving neurological deficit	Intracranial surgery within 3 months
Subarachnoid haemorrhage	Very severe stroke (NIHSS score >25)
History of intracranial haemorrhage	Major haemorrhage within 3 months
Arterial pressure >185/110 after attempts at reduction	Prior stroke within 3 months
Coincident major surgery or trauma	
Active internal bleeding	
Coagulopathy (INR>1.4)	
Pregnancy	

Urgent direct intra-arterial thrombolysis or clot retrieval should be considered if the patient is pregnant, coagulopathic or there is no response to intravenous rtPA

Pre-anaesthetic evaluation

The following questions should be addressed specifically:

- What is the cause of the stroke? If unclear, consider further investigations (e.g. CT or magnetic resonance imaging (MRI) of brain, duplex scan of carotid arteries, ECG, echocardiography).
- What is likely outcome from the stroke? Consider immobility, respiratory impairment, and functional assessment including ability to swallow. Around 50% lose the ability to swallow immediately after a stroke and 11% have problems after 6 months. Assess nutritional status.
- What is the nature and urgency of planned surgery? Defer non-urgent surgery (Table 4.12). In most cases non-urgent surgery should be deferred for 6 weeks after CEA or 3 months after disabling stroke, although few specific data are available.

Table 4.12 Suggested algorithm for decision making in patients after stroke or TIA

Scenario	Management
Recent non-disabling stroke or TIA not considered for CEA because mild or no carotid atherosclerosis or stroke not related to carotid disease.	Evaluate and proceed to non- CEA surgery. Probably defer for 3 months to allow cerebral autoregulation to normalize, and correction of other medical risk factors, though there are few data to guide this.
Recent disabling stroke, not considered for CEA, but presenting for other surgery	Postpone non-carotid surgery for at least 3 months and re-evaluate extent of stroke and urgency of surgery. If reasonable recovery and life expectancy, evaluate. If benefits of surgery outweight the risks, proceed with increased care and monitoring.
Distant non-disabling stroke or TIA and not considered for CEA at the time.	Reassess for CEA (? degree of carotid stenosis) and perform CEA first if indicated. If CEA not indicated, evaluate for non-carotid surgery.
Recent stroke or TIA and candidate for CEA, no major cardiac risk factors, but other disease process requiring urgent surgery, e.g. coexisting AAA or CLI	Correct other medical problems and perform CEA first.
Recent stroke or TIA and candidate for CEA with major cardiac risk factors (urgent treatment of cardiac risk factors eg anti-arryhthmic drugs, pacemaker, untreatedheart failure.	If cardiac revascularization indicated, consider PCI or combined CEA/ CABG). Correct major cardiac risk factors first; proceed to CEA.
Recent non-disabling stroke or TIA and has undergone successful CEA.	Defer non-urgent surgery for at least 6 weeks.

- What perioperative risk factors are present?
  - Major cardiac risk factors are: significant aortic stenosis, significant arrhythmias, uncompensated heart failure, and unstable angina (see 📖 Risk assessment tools in the vascular surgery patient, p. 116). In patients with these risk factors, any non-emergency surgery should be delayed to assess and optimize these conditions.
  - Most patients will have intermediate risk factors for cardiac complications. Aim to optimize these as far as possible, but without undue delay to surgery. Some patients with significant coronary disease may not be judged suitable for surgical coronary revascularization, especially if they remain physically inactive or have few symptoms. The benefits of investigation and intervention will depend on the urgency and nature of the surgery (see 📖 The vascular preoperative assessment clinic, p. 110).
  - Consider other likely co-existing risk factors: especially COPD, diabetes, CKD.
  - Be aware that assessment of functional capacity or cardiorespiratory symptoms may be difficult if mobility is impaired after a stroke.
  - The possibility of postoperative cognitive dysfunction should be considered.
- Do the potential benefits of the planned surgery outweigh the risks?
- Consider other concurrent therapy after TIA or non-disabling stroke: antiplatelet or anticoagulant therapy (aspirin, dipyridamole, clopidogrel, dabigatran or warfarin), statins, antihypertensive or antidiabetic drugs if indicated. Current warfarin therapy is a contra-indication to regional anaesthesia.

Investigations

- The patient will usually have had a CT or MRI scan of the brain.
- Duplex carotid artery scan may be indicated to evaluate a 2D image of the carotid arteries and carotid blood flow. Flow velocity and turbulence are used to estimate the degree of stenosis.
- ECG, FBC, U&E, LFT, and blood glucose will have been performed.
- Transthoracic echocardiography may have been performed.
- Further investigations depend on the planned surgery.

Principles of anaesthesia for non-carotid surgery in patients with a recent stroke or TIA

The main specific considerations of anaesthesia in patients with a recent TIA or stroke are to maintain cerebral perfusion, avoid hypotension and hypertension, and use a technique that permits early cognitive recovery. All general anaesthetic agents may provide some neuroprotection, but there is no good evidence that any particular anaesthetic technique is best.

- Consider a regional/local anaesthetic technique where possible, but remember the patient may have been receiving anticoagulant drugs.
- Antiplatelet therapy or warfarin should be withheld before surgery if the perioperative risk of stroke is low, and restarted >24h after surgery. If the stroke risk is high (e.g. recent TIA and urgent surgery), bridging therapy is advised (see 📖 The anti-coagulated patient, p. 215).

- Avoid hypertension (e.g. at laryngoscopy or aortic cross clamping).
- Have a lower threshold for intra-arterial or other cardiovascular monitoring.
- Avoid excessive coughing. Consider spraying the vocal cords or tracheal tube cuff with lidocaine 1%.
- Aim to maintain arterial pressure within 20% of preoperative values (though there are few data on which to base this). Avoid hypotension by the judicious use of vasopressors and IV fluids.
- Volatile anaesthetics impair cerebral autoregulation at concentrations >1 MAC. Sevoflurane has theoretical beneficial effects. Nitrous oxide is often avoided.
- Total IV anaesthesia has theoretical advantages. Both propofol and thiopental have neuroprotective effects.
- Maintain normocapnia as unpredictable changes in CBF may occur with increases or decreases in $PaCO_2$.

Further reading

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Diabetes

Incidence

- The incidence of diabetes mellitus (DM) is 20% in the population aged >60yrs.
- DM is an independent risk factor for morbidity and mortality in non-surgical patients.
- The incidence of atherosclerotic coronary artery disease and peripheral arterial disease is 2–3-fold higher in diabetics.
- In 2005 the WHO reconfirmed the classification of glucose metabolism in terms of venous plasma glucose measured (Table 4.13):
  - *After overnight fasting*—fasting plasma glucose (FPG) and
  - Two hours after an oral glucose load of 75g—oral glucose tolerance test (OGTT)
- The incidence of diagnosed DM in patients presenting for vascular surgery is 10–20%.
- However, when vascular surgical patients are screened for dysglycaemia (i.e. including those with undiagnosed DM, impaired glucose tolerance, or impaired fasting glucose) the incidence is >50%.

Table 4.13 WHO classification of glucose metabolism

Normal glucose metabolism	Fasting plasma glucose <6.1 2h plasma glucose <7.8
Impaired fasting glucose	Fasting plasma glucose 6.1–6.9 and 2h plasma glucose <7.8
Impaired glucose tolerance	Fasting plasma glucose <7.0 and 2h plasma glucose 7.8–11.0
Diabetes mellitus	Fasting plasma glucose ≥7.0 or 2h plasma glucose ≥11.1

Plasma glucose concentrations in mmol/L.

Reproduced from World Health Organization, 'Definition and Diagnosis of Diabetes Mellitus and Intermediate Hyperglycemia', Report of a WHO/IDF Consultation, Geneva, World Health Organization, 2006, p. 3, with permission. Available at http://whqlibdoc.who.int/publications/2006/9241594934_eng.pdf. Accessed 28th May 2013.

Table 4.14 Effects of insulin

Stimulatory	Inhibitory
Glucose uptake	Lipolysis
Lipogenesis	Proteolysis and gluconeogenesis
	Glycogenolysis

Pathophysiology

- Glucose homeostasis is regulated by a variety of hormones, including insulin, cortisol, glucagon, growth hormone, and catecholamines.
- Insulin has a variety of effects, both stimulatory and inhibitory, and the inhibitory effects may be more important in the regulation of plasma glucose (Table 4.14).

Type 1 diabetes

Patients with type 1 diabetes lack endogenous insulin due to destruction of the pancreatic islet β cells. This may be secondary to autoimmune or inflammatory processes.

Type 2 diabetes

Type 2 diabetes comprises a more heterogeneous group of hyperglycaemic disorders, involving a combination of insulin resistance and decreased insulin secretion. There is no single cause; both genetic and environmental factors are implicated.

Insulin is also involved in the vasodilatory and antithrombotic functions of the vascular endothelium. Abnormal insulin metabolism results in accelerated atherosclerosis, and in patients with insulin resistance exogenous insulin therapy may accelerate this process further.

Implications

- In the non-surgical population, impaired fasting glucose and glucose intolerance are associated with increased cardiovascular risk.

Table 4.15 Lee's RCRI. Major cardiac complication risk relates to the number of risk factors present

Risk factor	Criteria
High risk surgery	AAA/Thoracic/abdominal
Ischaemic heart disease	MI/Q waves on ECG/angina/nitrate therapy/stress test positive
Congestive cardiac failure	History/examination/CXR
Cerebrovascular disease	
IDDM	
Creatinine >177mcmol/L	

0 = 0.4%, 1 = 0.9%, 2 = 7.0%, ≥3 = 11% (high risk).

Reproduced from Lee TH et al., 'Derivation and Prospective Validation of a Simple Index for Prediction of Cardiac Risk of Major Noncardiac Surgery', Circulation, 100, 10, pp. 1043–1049, Copyright 1999, with permission from American Heart Association and Wolters Kluwer.

- DM is a known independent risk factor for perioperative cardiovascular morbidity and mortality, and features in risk stratification systems such as Lee's revised cardiac risk index (see Table 4.15).
- There is some evidence that abnormal glucose regulation in the vascular surgical patients, regardless of a diagnosis of DM, is also associated with an increased risk of perioperative myocardial ischaemia, troponin release, cardiac events and all cause 30-day mortality.
- Although recent WHO guidelines do not recognize HbA1C as a suitable diagnostic test, there is evidence that increased preoperative HbA1C concentrations are associated with increased cardiovascular morbidity and 30-day mortality after vascular surgery.
- Patients with DM undergoing arterial surgery are also at increased risk of non-cardiac complications, such as acute kidney injury, post-operative infections, and graft occlusion.

Preoperative screening

- Preoperative OGTT or FPG is not currently recommended as a routine before surgery: a recent European Society of Cardiology/European Association for the Study of Diabetes guideline only recommending testing where there are 2 or more cardiac risk factors as described in the ACC/AHA guidelines.
- However, there is a high prevalence of undiagnosed IGT and DM in vascular patients, and most of these newly-diagnosed diabetics would be missed if only a FPG test was performed without OGTT.
- The addition of a diagnosis of DM in a patient with other cardiac risk factors is important in that it informs the need for further preoperative cardiological testing.
- There is also a need to monitor post-operative blood glucose levels more closely in patients with DM or IGT, and IGT without DM may itself indicate a greater risk of cardiac morbidity/mortality.
- Additional preoperative assessment should focus on identifying the presence and severity of diabetes related end organ disease, and of other co-morbidities associated with increased risk.

- DM is associated with both large vessel atherosclerotic disease and microvascular disease.
- Complications of DM are wide ranging and include:
  - Microvascular complications (renal impairment, peripheral and autonomic neuropathy, retinopathy).
  - Macrovascular complications (IHD), CVD, peripheral vascular disease).

Preoperative assessment

- *History:*
  - Level of glycaemic control.
  - IHD.
  - Hypertension.
  - Evidence of autonomic neuropathy, e.g. orthostatic hypotension.
  - Renal impairment.
- *Medications:*
  - Cardiovascular.
  - Antidiabetic.
  - Insulin.
  - Oral hypoglycaemic agents.
- *Investigations:*
  - U&E.
  - HbA1C.
  - Urinalysis (proteinuria).
  - Resting ECG.

Cardiac investigations in DM

Recent European and American guidelines have sought to clarify the need for further cardiological investigations, such as echocardiography or stress testing. The current guidelines recommend testing for high risk surgery where there are three risk factors for cardiovascular disease. As DM itself counts as one of these factors the presence of two others would justify further investigation. In patients without a diagnosis of DM, but with two other risk factors, it may be justified to undertake formal screening for DM/dysglycaemia, i.e. FPG and OGTT.

Cardiovascular risk factors

- MI.
- Angina.
- DM (insulin).
- Renal insufficiency.
- Stroke/TIA.
- HF.

Perioperative management of diabetes

- There is a significant incidence of hyperglycaemia caused by critical illness or surgical stress even in those patients without known DM, especially in the older patient and the vascular surgical setting.
- There are few data on outcome specific to vascular surgery, but hyperglycaemia is associated with increased morbidity and mortality in the critically ill.

- Blood glucose levels should be monitored routinely during the perioperative period and a threshold set for escalating therapy.
- The glucose management of vascular surgical patients with known DM depends on the type of diabetes, overall level of control, the duration of surgery, and the anticipated stress response.

Diabetic control

- For diabetic patients undergoing shorter procedures only requiring the omission of one meal it is possible to adapt their usual management and avoid a variable rate intravenous insulin infusion (VRIII).
  - Prioritize diabetic patients to minimize starvation period.
  - For type 2 diabetics who are well controlled on oral agents omit their diabetic medication for the duration of starvation.
  - For type 1 diabetic management of their insulin depends on the specific agent's duration of action and frequency of injection. The dose may need to be reduced, but basal levels need to be maintained to prevent proteolysis and ketoacidosis.
  - For both groups blood glucose should be monitored hourly from admission to detect hyper/hypoglycaemia.

Management of perioperative hyperglycaemia

See Fig. 4.6.

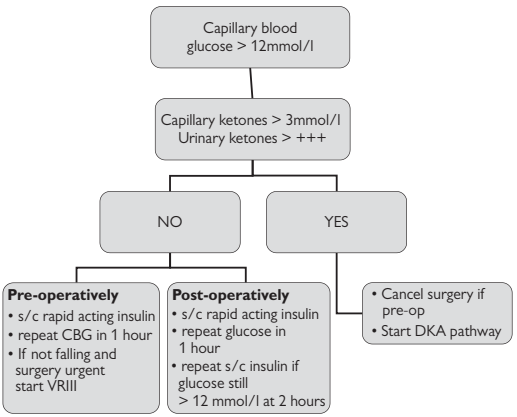

Fig. 4.6 Algorithm for the management of perioperative hyperglycaemia in surgical patients.

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Table 4.16 Management of VRIII

Rate of insulin infusion	• Set according to initial CBG. • Check CBG hourly. • Increase rate if: • levels remain above 12mmol/L and. • level is not falling by 3mmol/L. • Reduce rate to 0.5mmol/L if CBG <4.0, and treat hypoglycaemia.
Fluid regimen	• Initially 0.45% saline with 5% dextrose and 0.15% KCl. • Set rate to patients hourly fluid requirements. • Monitor U&E daily and alter fluid composition as appropriate. • Additional fluids may be required.
Administration	• Insulin and fluids should be infused via the same dedicated cannula. • One way valves are required to prevent backflow of insulin into the fluid line.

VRIII

For diabetics undergoing longer procedures or those associated with greater surgical stress a continuous VRIII titrated to regular blood glucose monitoring is indicated. There has been much debate over recent years regarding the necessary target for blood glucose levels. Initial enthusiasm for tight glucose control, aiming for a blood glucose of 4.4–6.1mmol/L, has been somewhat moderated, with the latest NHS guidelines suggesting a target of 6–10mmol/L (see Table 4.16).

Summary

- DM and dysglycaemia are highly prevalent among vascular surgery patients, and significantly increase the perioperative risk.
- Patients with DM should be identified before surgery operatively. Those without known DM, but with 2 other significant risk factors should be screened for dysglycaemia or undiagnosed DM.
- Blood glucose concentrations should be monitored in all patients in the perioperative period, and a threshold for insulin treatment set.
- A target level of <11mmol/L is recommended for patients with peripheral vascular disease. Moderately tight control (glucose maintained between 6.0–8.3mmol/L) may have greater benefit, but further research is needed.

Further reading

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Chronic kidney disease

Mild to moderate CKD (stages 1–3) occurs in up to 10% of the population, and severe CKD in 0.4%. CKD is more common in patients with vascular disease. The incidence increases with age. CKD is classified according to eGFR (see 📖 Optimizing renal function, p. 148).

Causes of chronic renal dysfunction in vascular surgical patients

- Hypertension.
- Diabetes.
- Previous aortic or renal surgery.
- HF.
- Renal artery emboli, atheroma, or renal artery stenosis.
- Drugs—NSAIDs, ACE inhibitors, ARBs, recent radiocontrast.

Implications of CKD in vascular surgical patients

- Severe CKD is an independent risk factor for cardiovascular disease.
- Cardiovascular disease is the commonest cause of death in non-diabetic patients with CKD.
- The majority of patients with CKD have impaired left ventricular function as assessed by echocardiography.
- CKD is an independent risk factor for adverse perioperative cardiovascular outcome.
- CKD is a risk factor for perioperative AKI or permanent worsening of renal function after vascular surgery (see 📖 Perioperative renal protection, p. 236).

Clinical consequences/signs symptoms of CKD

- *Metabolic:* metabolic acidosis, hyperkalaemia, hypermagnesaemia, hypocalcaemia, hyperphophataemia, hyponatraemia, glycosuria.
- *Cardiovascular:* circulating volume unpredictable- hypovolaemia or fluid overload, hypertension, (hyperaldosteronism or other causes), accelerated atherosclerosis and ischaemic heart disease, cardiac output (may be increased if anaemic), pulmonary or peripheral oedema, pericarditis, pericardial effusion.
- *Haematological:* chronic normochromic anaemia, coagulopathy with platelet dysfunction.
- *Neurological:* peripheral neuropathy, pruritis.
- *Endocrine:* hyperparathyroidism and renal osteodystrophy, hyperlipidaemia.
- *Other:* malnutrition, increased susceptibility to infection, delayed gastric emptying.

Clinical features and assessment

- *General examination:* thirst, dry tongue, reduced skin turgor, nutritional state, body weight (pre- and post-dialysis and target weight), fatigue, decreased exercise tolerance.
- *Cardiovascular:* blood volume status (tachycardia, orthostatic hypotension if recent dialysis), peripheral or pulmonary oedema, JVP, pericardial rub, pericardial effusion, cardiac tamponade.

- *Respiratory*—hyperventilation (compensatory to metabolic acidaemia or pulmonary oedema).
- *Abdominal*—ascites, renal transplant. Delayed gastric emptying if uraemic.
- *Neurological:* peripheral distal polyneuropathy, diabetic neuropathy, restless legs, confusion, deceased conscious level, seizures. Some acute symptoms may improve after dialysis.
- *Method of RRT*: identify and protect AV fistulae or continuous peritoneal dialysis (CPD) catheters.
- Timing of last RRT.
- *Haematological:* anaemia, bleeding tendency (bruising, epistaxis, GI bleeding).

Investigations

- U&Es, glucose, GFR, FBC, Hct, LFT, ECG, CXR, urinalysis.
- In selected cases.
- ABG (venous gases bleeding time, platelet function tests, clotting studies, HbA_{1C} (diabetics).
- Echocardiography, invasive cardiological investigations (see 📖 Cardiological investigations in the vascular surgery patient, p. 129).

Anaesthetic considerations

- Increased cardiovascular risk.
- Blood volume and hydration status variable. Difficulties with elimination of fluids and Na^+. Potential for hyperkalaemia, hyponatraemia (dilutional), fluid overload.
- Perioperative worsening of renal function because of:
  - *Pre-renal ischaemia*—2° to hypovolaemia, hypotension, hypoxia, relative hypovolaemia (e.g. HF, cirrhosis, sepsis), renal vascular disease (e.g. renal atherosclerosis, renal artery stenosis), surgical occlusion (e.g. aortic cross-clamping, endovascular occlusion), redistribution of renal blood flow (e.g. NSAIDs, ACE inhibitors, ARBs, direct vasoconstrictors), intra-abdominal hypertension.
  - *Direct renal damage*—caused by ischaemia reperfusion injury (surgical revascularization), athero-embolism at surgery, exogenous nephrotoxins (e.g. radio-contrast, gentamicin).
  - *Post-renal causes*—bladder outlet obstruction, intra-abdominal hypertension, bilateral ureteric damage or obstruction.
- *Volatile anaesthetic agents:* cause dose-related decreases in RBF, GFR, and urine output (UO). This is usually insignificant, but may contribute to renal ischaemia if hypotension occurs. Sevoflurane has the potential for fluoride-induced nephrotoxicity. Although not demonstrated in practice it is perhaps best avoided.
- *Pharmacological considerations:* many patients with CKD are receiving antihypertensive and other drugs. Elimination of drugs and drug metabolites excreted by the kidney is impaired (e.g. neuromuscular blockers (NMBs), opioids). Risk of drug accumulation and toxicity so plasma monitoring of some (e.g. digoxin, aminoglycosides) essential. Increased sensitivity to CNS depressant drugs (e.g. benzodiazepines,

IV anaesthetics). Decreased protein binding can result in increased free drug concentrations.

- Uraemic coagulopathy. CKD is associated with increased bleeding tendency due to platelet dysfunction. Conventional tests of coagulation (platelet count, partial thromboplastin time (PTT), APPT) are normal. Bleeding time is prolonged and correlates with bleeding tendency. Treatment is with:
  - Cryoprecipitate (provides Factor VIII-von Willebrand factor, vWF) 10iu IV over 30min. Duration of effect 12–18h.
  - Desmopressin 0.3μg/kg IV or sc (increases activity of circulating Factor VIII-vWF complex). Duration of effect 6–8h. Less effective with repeated doses.

Perioperative management

CKD stages 4 and 5

Refer to nephrologist before vascular surgery if:

- eGFR<30 (CKD stages 4–5), for planning of anaesthesia, surgery, and post-operative care.
- There is a new diagnosis of CKD stages 3–5, for further investigation before elective vascular surgery.

Preoperative preparation

- In dialysis dependent patients, RRT should be performed 24–48h before elective surgery to correct fluid overload acid-base and electrolyte disturbances. Many of the symptoms of uraemia are also corrected by RRT. Uraemic coagulopathy should be corrected before surgery or invasive monitoring procedures.
- Ensure K^+ is <5.5mmol/L before induction of anaesthesia.
- Fluid management can be difficult in dialysis-dependent patients as fluid overload can occur easily—CVP monitoring is required. Insensible fluid losses can be replaced with glucose 4%/saline 0.18% at 1–1.5mL/kg/h, with further balanced salt solutions (Hartmann's) titrated to estimated '3rd space' losses. Colloid solutions and/or packed red blood cells should be used to replace blood losses.

General anaesthesia

See 🕮 General anaesthesia, p. 205. Use drugs in minimum doses and titrated to effect. Arrangements should be made before surgery for early post-operative re-institution of RRT.

Consider special surgical measures, e.g. renal revascularization techniques in those with CKD stage 4–5 undergoing aortic surgery.

CKD stages 1–3

Patients with mild CKD (stages 1–2) or stable known CKD stage 3 can be managed as follows:

Pre-operative preparation

- *Optimize treatment of co-existing conditions:* e.g. hypertension, diabetes, IHD.
- *Avoid dehydration:* encourage clear oral fluids up to 2h before elective surgery. IV hydration using 0.9% saline 1.5mL/kg/h is often used for

8–12h pre-operatively Pre-medication. If required, light pre-medication (temazepam 10mg po) is adequate.
- Consider regional techniques alone (e.g. for carotid surgery, vascular access procedures, lower limb vascular surgery) or in conjunction with general anaesthesia. Check for presence of peripheral neuropathy and treat coagulopathy before regional anaesthesia.

General anaesthesia
- Cardiovascular responses to IV anaesthetic drugs may be marked, even when patient is not hypovolaemic. Increased sensitivity to IV anaesthetics. Induce anaesthesia with slow injection of IV anaesthetic. Reduce doses of propofol or thiopental (reduced protein binding).
- Total IV anaesthesia or inhalational anaesthesia using volatile agents with nitrous oxide or air in oxygen can be used.
- When using volatile agents, isoflurane or desflurane are preferred. Both are minimally metabolized and have little effect on RBF or GFR at doses <1.5 MAC. Sevoflurane can theoretically cause fluoride nephrotoxicity. Although there is little evidence that this is important in clinical practice.
- *NMBs:* hyperkalaemic response to suxamethonium is not exaggerated in patients with CKD, but hyperkalaemia could occur if serum K^+ is >5.5mmol/L before administration of suxamethonium. Atracurium, cisatracurium, or mivacurium are the NMBs of choice as their elimination is completely independent of renal function. Laudanosine, a metabolite of atracurium could potentially accumulate in severe renal failure and cause CNS toxicity so infusions of atracurium are best avoided. Other NMBs (vecuronium, rocuronium, pancuronium) are eliminated to some degree by the kidney so are best avoided. Elimination of neostigmine is prolonged.
- *Analgesics:* morphine, fentanyl, alfentanil, remifentanil can all be used. Reduce doses and titrate to effect; morphine has active metabolites (morphine-6 glucuronide) can accumulate. Pethidine is best avoided owing to accumulation of neurotoxic metabolites.

Fluids and monitoring

Perioperative optimization of haemodynamics reduces the risk of AKI. Maintain cardiac output and renal perfusion pressure throughout the perioperative period, using fluids and inotropic drugs as required, with appropriate monitoring (see 🕮 Cardiac output monitoring, p. 259). Balanced salt solutions (e.g. Hartmann's solution) are appropriate unless there is hyperkalaemia.
- *Targets are:* CVP 8–12mmHg, MAP >70mmHg, central venous SvO_2 >70%.
- Though intraoperative urine output does not relate to the incidence or severity of post-operative AKI, a spontaneous urine output of >0.5mL/kg/h during and after surgery (without diuretic drugs) suggests that renal perfusion is adequate.
- Institute vasopressors or vasodilators early if arterial pressure and cardiac output do not respond to IV fluids.

- *Avoid:*
  - Prolonged intra-abdominal hypertension.
  - High intra-thoracic pressures during and after surgery.
  - Nephrotoxic drugs (e.g. gentamicin, amphotericin, cyclosporin, repeated or high doses of radiocontrast).
  - Acute perioperative anaemia. This probably reflects adequacy of resuscitation and intravascular volume status.

Post-operative pain management

- Regional techniques are useful and should be used where appropriate.
- Avoid NSAIDs except paracetamol.
- Morphine can be used via a PCA device, but beware increased sensitivity to opioids and potential accumulation of morphine-6-glucuronide; a bolus dose of 0.5mg and a lockout time of 10min may be used. Other opioids should be administered with caution and reduced dose; beware increased and prolonged effects.

Post-operative monitoring

Patients with CKD stage 1–3 should be monitored in a HDU or ICU environment after major vascular surgery. Specific potential problems include:

- Increased risk of AKI.
- Hyperkalaemia.
- Oliguria, related to hypovolaemia, inadequate IV therapy, development of AKI. Fluid management is often difficult and invasive monitoring is required. Avoid hypotension and maintain CO at normal levels and MAP >70mmHg using IV fluids and inotropes. Dopamine, mannitol and other diuretics do not prevent AKI. Furosemide 20–40mg IV may be used to initiate a diuresis, but only after circulating volume and CO have been optimized. Patients with CKD stage 2–3 are often resistant to diuretic drugs; response to furosemide is usually a marker of the severity of AKI.
- *Hypertension:* may relate to pre-operative hypertension. May require treatment with vasodilators (e.g. glyceryl trinitrate (GTN), labetalol, hydralazine, nifedipine) or may require RRT if severe and fluid overload present.
- *Analgesia:* increased sensitivity to opioids; increased risk of respiratory depression.
- Anaemia.

Plans for post-operative RRT should be made before surgery. The indications for RRT are discussed in 📖 Perioperative myocardial infarction, p. 474.

Further reading

BNF. Drugs in renal impairment. Appendix 3.

Chronic obstructive pulmonary disease

Clinical features

- COPD is characterized by symptoms of wheeze, dyspnoea, and cough. Unlike asthma, in which the same symptoms occur intermittently, patients with COPD have constant respiratory disability, often interspersed with acute exacerbations secondary to otherwise innocuous respiratory pathogens.
- Exacerbations cause further lung damage, leading to disability and respiratory failure.
- Almost 5% of deaths worldwide are now believed to be caused by COPD.
- COPD may be regarded as a normal part of the ageing process in that lung function in everyone declines inexorably from a peak reached in middle age (Fig. 4.7). The rate of this decline is genetically determined, but most individuals will eventually experience some degree of respiratory impairment with symptoms of COPD, if they live long enough.
- However, the likelihood of ever achieving poor enough lung function to become symptomatic is mostly determined by tobacco smoking, which damages lung development from early adulthood and accelerates the rate at which lung function declines. As a result, a patient who smokes will develop respiratory disability decades before a non-smoking patient with the same genetic makeup.
- Once COPD develops, the rate of decline in lung function accelerates further—the forced expiratory volume in 1s (FEV_1) declines at a rate of around 60mL/year. One in five smokers will develop COPD; the more surprising aspect of this statistic is that four in five do not, and the search is on for a genetic explanation of why some individuals can smoke without apparently causing lung damage.

Pathophysiology

- COPD results from a combination of irreversible airway obstruction and emphysema, with the proportion of these varying widely between patients.
- Airway narrowing in large airways results from goblet cell hyperplasia and excessive mucus production. In smaller airways there is mucosal thickening and hypertrophy of airway smooth muscle; this airway remodelling soon becomes irreversible.
- Emphysema is the destruction of alveolar structure to form larger airspaces, eventually forming bullae. Emphysematous lung regions have a high V/Q ratio so contribute to alveolar dead space, but the more important detrimental effect of emphysema is loss of elastic tissue resulting in further narrowing of small airways.
- Expiratory airflow limitation in COPD commonly leads to hyperinflation of the chest, significantly impairing the function of the respiratory muscles, particularly the diaphragm.

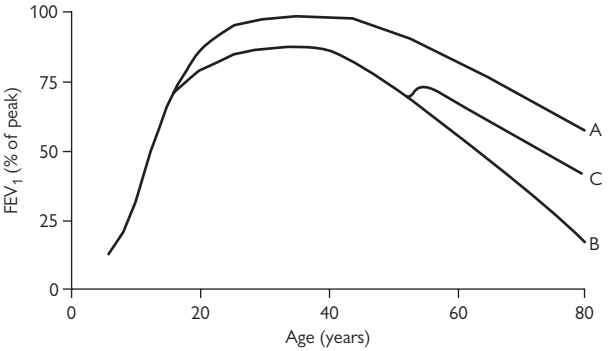

Fig. 4.7. Pattern of lung function during a lifetime. In a non-smoker (A) FEV_1 rises to a peak between 20–40yrs of age then declines linearly, the rate of decline being genetically determined. When FEV_1 reaches 50% of the peak, respiratory symptoms are likely and at 25% the patient is severely disabled. A smoker (B) reaches these levels at a much younger age, and a patient who quits smoking (C) achieves the same rate of further decline as a non-smoker from that point onwards.

- The fibre type of respiratory muscles is also abnormal in COPD, with a greater number of fatigue-resistant fibres being present making muscle function inefficient for breathing. These changes may result either from hyperinflation or from the frequent systemic inflammatory responses associated with COPD.

Optimization before surgery

- The severity of COPD in an individual patient is best assessed by measurement of FEV_1.
- Although there are slight differences between guidelines, the following is often used (FEV_1 as a % of predicted for that patient).
  - *FEV_1 50–70% of predicted*—mild disease.
  - *FEV_1 30–50% of predicted*—moderate disease.
  - *FEV_1 <30% of predicted*—severe disease.
- This grading should allow the anaesthetist to determine the degree of preoperative intervention required and guide the anaesthetic technique (📖 The risks of vascular surgery, p. 114).
- It is crucial to identify before surgery patients who are developing or currently have an acute exacerbation of their COPD. A change in symptoms is the most reliable approach, but a sudden decline in lung function tests should also lead to further evaluation. Non-emergency surgery should be deferred to allow treatment with bronchodilators, steroids, and antibiotics. Referral to a respiratory physician may be required.

Preoperative management

In a patient with stable COPD, a number of interventions may be useful before surgery.

Smoking cessation

In general, stopping smoking before surgery helps to reduce post-operative pulmonary complications (📖 Optimizing respiratory function, p. 151). However, in patients with established COPD who are still smoking, the challenge of preoperative cessation is even greater because these patients are now dedicated smokers. Furthermore, because the pathology in established COPD is essentially irreversible, there is less to gain from smoking cessation in terms of improving pulmonary function than in smokers who have not yet developed COPD. It is also not known how long the COPD patient would need to stop smoking before surgery to gain any outcome benefit. For the reasons described in 📖 Optimizing respiratory function, p. 151, all patients should abstain from smoking for as long as possible and at least 12h preoperatively.

Escalation of treatment

Potential options include increasing bronchodilator therapy in patients known to have a reversible component to their airflow limitation, addition of oral bronchodilators, or perioperative steroids. In patients with moderate or severe disease it is advisable to only institute these measures in discussion with their respiratory physician.

Preoperative pulmonary physiotherapy

The techniques and benefits of preoperative physiotherapy are discussed in 📖 The risks of vascular surgery, p. 114. These are all likely to be more beneficial in the early post-operative period, but there is evidence that the interventions are more effective if a physiotherapist is involved early and the techniques taught to the patient before surgery.

Perioperative management of COPD patients

A diagnosis of COPD is a risk factor for developing post-operative pulmonary complications (see 📖 Optimizing respiratory function, p. 151). ∴ RA is preferred technique for these patients if possible.

Management of RA in patients with COPD

Upper limb vascular surgery

- Brachial plexus block is an ideal technique for COPD patients having surgery involving the arm.
- Particular care should be taken to minimize the risk of pneumothorax as this will cause significant morbidity in a patient with COPD.
- US guidance is advisable and infraclavicular approaches to the brachial plexus should not be used.

Lower abdominal and lower limb surgery

- Surgery to the distal external iliac artery and below and endovascular aortic surgery are suitable for RA.
- There is no ideal RA technique—any combination of spinal, epidural, or spinal catheter approaches are suitable. The choice of technique depends on the expertise of the anaesthetist.

- Thoracic epidural anaesthesia has minor effects on respiratory muscle function. Effects include impaired responses to stimulated respiration and reductions in effort-dependent lung volumes such as peak expiratory flow rate. Although normally minor, these may impair the ability of a patient with COPD to cough effectively, particularly considering the already abnormal respiratory muscle function described above.

The following considerations apply when providing RA for patients with COPD:

- Neuraxial opioids should be used with care as patients with severe COPD are at risk of developing hypercapnia with what would in other patients be only a minor degree of central respiratory depression.
- Sedative drugs administered systemically may result in more respiratory depression compared with healthy patients.
- Oxygen should only be administered if required, and ideally titrated to a target SpO_2 of 88–92% as advised by British Thoracic Society guidelines.
- *Position and patient comfort:* if the patient has a long-term cough or inability to lie flat this must be carefully considered when assessing the feasibility of RA.

Management of GA in patients with COPD

Intra-operative care

- The greatest challenge during general anaesthesia in patients with COPD is achieving adequate gas exchange without risking lung damage from high airway pressures. Pressure controlled ventilation is preferable, though it can be challenging to achieve a minute volume adequate to maintain normocapnia.
- End-expiratory CO_2 is a very unreliable guide to arterial P_{CO2} in patients with COPD, and intraoperative blood gas monitoring is useful. If repeated blood gas measurements or post-operative intermittent positive pressure ventilation (IPPV) are anticipated, an arterial catheter should be placed.
- The strategy used in critical care patients of using permissive hypercapnia to minimize ventilator inflation pressures is not useful in the operating theatre because of the risks of post-operative hypercapnia.
- A strategy to provide effective ventilation, but avoid pulmonary damage during surgery should include:
  - Effective humidification.
  - Modest amounts of PEEP (5–10cmH_2O).
  - Avoidance of 100% oxygen except when indicated by hypoxaemia.
  - Recruitment manouvres to re-expand areas of atelectasis.
- Specific measures should be taken to allow a rapid recovery after surgery. This includes maintaining normothermia, the use of short acting anaesthetic agents, providing good analgesia, and avoiding excessive volumes of intravenous fluids.

Post-operative care

- Immediately after surgery, the combination of sedation, weakness, and reduced respiratory drive from residual anaesthesia, neuromuscular blockade and opioids will quickly enter the vicious circle of rising PCO_2 causing sedation and further hypoventilation. Therefore these conditions should be avoided at all costs.
- Close attention must be paid to adequate post-operative analgesia, avoiding excessive sedative drugs or opioids.
- All patients must be monitored closely for signs of impaired respiratory function, and a period of artificial ventilation instituted if an increasing PCO_2 or worsening sedation occurs. The potential need for post-operative care in a HDU or Critical Care Unit should be anticipated and planned.
- In patients with pre-existing hypercapnia from severe COPD, or intra-operative hypercapnia, a period of non-invasive ventilation immediately after tracheal extubation may be useful.

Further reading

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Uncommon conditions associated with vascular disease

Patients presenting for vascular surgery have an increased risk of a variety of pro-thombotic conditions. These diseases may have contributed to the development of the patient's arterial disease, and may also make the patient more at risk of perioperative thrombotic complications, in particular failure of revascularization in interventions for PVD.

Thrombophilias

Thrombophilia is a broad term describing any inherited or acquired defect of haemostasis that predisposes the patient to venous or arterial thrombosis. Clinical conditions that should raise suspicion of thrombophilia include:

- Recurrent venous thrombosis.
- Venous thrombosis in a patient aged <40yrs.
- A non-haemorrhagic stroke in a patient aged <60yrs.
- Arterial thrombosis in a patient aged <30yrs.

Almost one-third of patients with PVD have some form of thrombophilia, and these patients are more likely to develop critical ischaemia. There are a variety of types of thrombophilia.

Abnormalities of protein C (APC)

Protein C is activated when thrombin binds to the endothelium, and its activated form APC inhibits the actions of activated coagulation factors V and VIII to reduce thrombin production.

- The incidence of protein C deficiency is approximately 1 in 500.
- The incidence of venous thromboembolism is increased 10–15-fold in patients with protein C deficiency.

APC resistance

- APC resistance is a form of thrombophilia in which the normal interaction between APC and activated factor V is impaired.
- APC is structurally normal, but an inherited substitution mutation of one of the genes for factor V causes the activated factor V to be resistant to cleavage by APC.
- This is known as the factor V Leiden mutation and is the most common inheritable thrombophilia. The estimated incidence of factor V Leiden mutation is 2–15% in the general population.
- In patients with PVD, the prevalence is almost doubled (26 vs. 12% in one study).

Protein S deficiency

Protein S is a cofactor for the reaction between APC and the coagulation factors that it inhibits (activated V and VIII).

Although the prevalence of protein S deficiency in the population is uncertain, it is the most common cause of thrombophilia in patients with PVD.

Anti-thrombin deficiency

- Anti-thrombin is an important naturally occurring anticoagulant, which acts via several different mechanisms.
- Anti-thrombin, as its name implies, has a powerful direct inhibitory effect on thrombin activity, but also reduces thrombin production via inactivation of activated clotting factors V and VIII, and further retards coagulation by inhibition of activated factors IX, X, XI, and XII.
- Deficient antithrombin activity may result from inadequate production of a normal anti-thrombin protein or adequate amounts of a functionally abnormal protein (functional anti-thrombin deficiency).
- Anti-thrombin deficiency is rare (1 in 5000 individuals), but results in a severe predisposition to venous thrombosis (25–50 times normal).
- Functional anti-thrombin deficiency increases the risk of venous thrombosis approximately 4-fold.

Acquired thrombophilias

- The most common cause of an avoidable increase in thrombotic tendency is the progesterone analogues used in the oral contraceptive pill. This acquired thrombophilia will become a much more significant clinical problem in patients who also have one or more of the inherited thrombophilic tendencies already described.
- A more rare form of acquired thrombophilia is the presence of anti-phospholipid antibodies such as the lupus anticoagulant or anticardiolipin antibodies that can occur in a variety of autoimmune diseases.
- Prevalence of lupus anticoagulant is increased in patients with PVD.

Clinical implications of thrombophilias in vascular surgery

- The prevalence of both hereditary and acquired forms of thrombophilia is increased in patients with PVD, but this does not indicate a causative relationship between the two. Unfortunately, suitably designed and powered trials have not been performed to confirm this suspicion.
- Similarly, the effect of thrombophilia on the outcomes of peripheral revascularization procedures is not entirely clear.
- An early study, including only 60 patients, demonstrated a 3-fold increase in risk of early graft failure, but a further study of 775 patients found only a small, and non-significant, increase in risk of graft occlusion.
- From these studies, the factor V Leiden abnormality seems to be the form of thrombophilia most commonly associated with graft failure, but the lack of evidence for this means that routine preoperative screening for thrombophilia is not currently recommended.
- In the perioperative period, all patients should have meticulous attention paid to minimizing the chances of a graft failure or a venous thromboembolic event, following the usual guidelines.
- For patients with a clinical suspicion of thrombophilia (as described above) then further preoperative investigations are probably justified.
- If inherited form of thrombophilia is identified then consultation with haematologist is required to plan optimal management of the patient.
- Therapeutic options include the administration of anti-thrombin/protein C complex.

Homocysteine and vascular disease

The association between homocysteine (Hcy) and vascular disease was first reported in 1962. Since then, much research concluded that hyperhomocysteinaemia (HHcy) is an independent risk factor for the development of CHD, CVD, and PVD. However, it remains unclear as to whether HHcy is simply a marker of advanced vascular disease or actually responsible for vascular disease developing.

Homocysteine metabolism

- Hcy is a sulphur-containing amino acid that is not involved in protein structure. It is a fundamental part of two different metabolic pathways:
  - Formation and destruction of methionine, and its activated form, S-adenosylmethionine (SAM), which acts as a donor of methyl groups in a variety of cellular metabolic pathways, during which it is converted back to Hcy. The formation of methionine and SAM are dependent on both folate and vitamin B12.
  - Trans-sulphuration, in which Hcy is converted by a two-stage reaction into cysteine, which is required for the production of glutathione and plays a crucial role in protein structure, being the only amino acid that can form the disulphide bonds required for protein folding. This pathway is dependent on vitamin B6 and is important because it is the only mechanism for removing excessive Hcy from the body.

- Hcy metabolism therefore involves a variety of enzymes, many of which may be affected by single nucleotide polymorphisms that affect their function, so hereditary factors have a strong influence on Hcy levels.
- The requirement for adequate dietary methionine, folate, B6, and B12 means that diet and vitamin absorption also influence Hcy. Because Hcy is the starting point for both metabolic pathways, either a genetic deficiency in the activity of an enzyme, or a deficiency of a vitamin cofactor will cause HHcy.

Role of HHcy in vascular disease

- HHcy could cause arterial disease by many mechanisms:
  - *Increased platelet adhesion:* Hcy increases the adhesion of platelets to vascular endothelial cells, probably by inducing oxidant stress in the endothelium, which adversely affects the nitric oxide-mediated interaction with platelets.
  - *Pro-coagulant effects:* Hcy has pro-coagulant effects, in vitro evidence suggesting that these are mediated by a profound inhibition of protein C activity.
  - *Impaired fibrinolysis:* Hcy impairs fibrinolysis by a direct interaction with the receptor region of tissue plasminogen activator.
- This triad of interference with the vascular endothelial, coagulation and fibrinolytic systems is very likely to impact on vascular disease progression, particularly when HHcy goes undetected for many decades.
- For a majority of patients the major determinant of blood Hcy levels is their diet, i.e. the availability of folate and vitamins B_6 and B_{12}. This has led to the alluring prospect of being able to modify vascular disease progression by dietary manipulation. Unfortunately, this hypothesis is difficult to test due to the challenge of controlling dietary intake of these vitamins and the long term study required to assess vascular disease. Early results suggest that dietary manipulation can reduce Hcy levels, but without any demonstrable improvement in a patient's vascular disease.

Systemic vasculitis

Most systemic vasculitides cause widespread inflammation of blood vessel walls. Two types of vasculitis may be associated with PVD requiring surgery.

Thromboangiitis obliterans (Buerger's disease)

- Inflammatory vasculitis affecting small- to medium-sized arteries and veins.
- Occurs almost exclusively in men aged <45yrs.
- Associated with heavy smoking.
- Causes premature intermittent claudication, critical ischaemia, and arterial, and (less commonly) venous thrombosis.
- Smoking cessation is essential.
- Patients may present for revascularization surgery, although this is often ineffective because distal vessels are involved. 1° amputation may be the best option.
- Antiplatelet agents, anticoagulants or vasodilators may be prescribed, but are not of proven benefit.

Takayasu's arteritis

- Rare, progressive, occlusive vasculitis affecting large arteries of the systemic and pulmonary circulations.
- Causes obliteration of the aortic lumen and main branches.
- Associated with ankylosing spondylitis and rheumatoid arthritis.
- May present with CNS symptoms (including stroke), cardiac ischaemia, renal dysfunction, or acute arterial occlusion requiring urgent surgery.
- The mainstay of treatment is corticosteroids. Antiplatelet or anticoagulant drugs may also be used.
- Anaesthetic considerations include potential airway problems, perioperative steroid supplementation and the need to avoid hypoperfusion of areas supplied by stenosed arteries. Regional techniques may be technically difficult and the patient may be receiving anticoagulant drugs. Arterial pressure measured in the arm is usually lower than aortic pressure, and may be difficult to record using a non-invasive cuff. Intra-arterial pressure monitoring via the radial or femoral artery is an alternative.

Further reading

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The anti-coagulated patient

Management of the anti-coagulated patient undergoing vascular surgery

Patients presenting for vascular surgery are usually taking drugs that inhibit haemostasis. The indications for these are either the presenting vascular condition or co-existing diseases. The decision to continue or withhold anti-coagulant or antiplatelet medication must balance the risk of perioperative bleeding with an increased risk of thromboembolism if these drugs are stopped.

Anti-coagulant drugs

Warfarin

- Warfarin is the recommended oral anti-coagulant in the UK.
- It acts by inhibiting hepatic vitamin K synthesis and so reduces levels of the vitamin-K dependent coagulation factors II, VII, IX, and X.
- The effect of warfarin is monitored by measuring the prothrombin time, which is usually expressed as a ratio between the patient's result and a control reading, the international normalized ratio (INR).
- The target INR depends on the therapeutic indication (Table 4.17).

Table 4.17. Indications for warfarin therapy and the target INR.

Indication	Suggested INR
Prophylaxis after DVT	2.0–2.5
Pulmonary embolus; atrial fibrillation; cardioversion; dilated cardiomyopathy; rheumatic mitral valve disease	2.5–3.0
Recurrent DVT or pulmonary embolism	3.5

- In most patients receiving warfarin it is advisable to continue anti-coagulation during the perioperative period.
- Because the effect of warfarin takes several days to diminish, warfarin should be stopped before surgery. Current guidelines suggest stopping warfarin 5 days before planned major surgery.
- The offset of warfarin effect should be monitored using the INR.
- In some patients at high risk of arterial or venous thromboembolism, short acting heparin therapy is given when warfarin is stopped, either as unfractionated IV heparin or subcutaneous low molecular weight heparin (LMWH). This is termed bridging therapy and is continued for 10–12 days during interruption of warfarin and when the INR is outside the therapeutic range.
- The decision to institute bridging therapy depends on the risks of thromboembolism and the risks of haemorrhage related to surgery. Carotid and aortic surgery may be considered high risk procedures for perioperative bleeding so interruption of warfarin without bridging therapy may be considered. This decision should be taken in conjunction with a haematologist.
- Although an INR <1.5 is generally accepted as a safe level for surgery and anaesthestic interventions including neuraxial blockade, the acceptable value for an individual patient should account for other factors, such as the urgency of surgery, the concomitant use of antiplatelet drugs, and the potential consequences of delaying surgery.
- In an emergency situation vitamin K (2–4mg orally or IV) may be given to accelerate the rate at which new coagulation factors are produced, and in dire emergencies coagulation factors (prothrombin complex concentrates) should be given. These are now preferred over FFP.
- Warfarin should be restarted a minimum of 12–24h after surgery, but only when haemostasis has been restored and the risks of surgical re-bleeding subsided.

Heparin

Patients presenting for emergency vascular surgery or those who have been temporarily converted from warfarin therapy may be receiving therapeutic doses of heparin. Other patients may be receiving low dose heparin for DVT prophylaxis. In both cases the anaesthetist must be aware of the implications of the heparin therapy.

Unfractionated heparin

- Heparin binds to antithrombin III, enhancing its ability to inhibit the activity of coagulation factors IX, X, XI, and XII. Unfractionated heparin (UFH) also inhibits the activation of platelets by fibrin.
- Therapeutic anti-coagulation is achieved by IV infusion of UFH at a rate of approximately 1000U/h, monitoring the APTT to maintain a patient: control ratio of 1.5–2.5.
- The elimination half-life of heparin is dose-dependent. At these doses UFH has a half-life of approximately 90min, so for patients receiving therapeutic UFH, stopping the infusion 4–6h before surgery will reverse its effects.
- Normal coagulation usually returns in 3–4h, but this should be confirmed by re-checking APTT before major surgery or the use of neuraxial blockade. The risk of bleeding is low if the APTT ratio is <1.5.
- In emergency situations, protamine may be used to reverse heparin. The dose of protamine depends on the dose of heparin, and interval since stopping the heparin infusion. Near-patient testing of heparin activity is advisable to titrate the protamine dose accurately.
- Bridging doses of UFH should only be re-started a minimum of 48–72h after surgery, and only when haemostasis is adequate.

Low molecular weight heparin

- The main action of LMWH is to inhibit Factor Xa.
- LMWHs have a more predictable and longer duration of action compared with UFH so they can be administered od or bd as sc injection basis, rather than by continuous IV infusion.
- The LMWHs dalteparin, enoxaparin, and tinzaparin have now largely replaced UFH for DVT prophylaxis in surgical patients.
- LMWHs also have less effect on platelets and so a reduced tendency to cause bleeding. The peak effect of LMWH occurs 4–6h after its administration.
- The APTT does not reflect the activity of LMWH and is not used for monitoring therapy.
- It is generally accepted that 12h should elapse between administration of LMWH and performing high-risk procedures, such as insertion or removal of epidural catheters or major surgery. This advice is complicated by two different regimens for using LMWH for DVT prophylaxis in Europe (e.g. enoxoparin 20–40mg od commencing 12h preoperatively) and North America (e.g. 30mg bd commencing 1h post-operatively).
- When LMWH are used in higher doses as bridging therapy, a longer interval between last dose and surgery is required—24h when the surgical bleeding risk is low and 48–72h when the bleeding risk is high.
- Careful timing of the administration of LMWH in the perioperative period is therefore required.
- Protamine does not completely reverse the effects of LMWH heparin.

Other anti-coagulants

Dabigatran is a direct thrombin inhibitor. Rivaroxaban inhibits activated Factor Xa. Both these drugs are used for thromboprophylaxis after major orthopaedic surgery. Few data are available, but if a patient presents for vascular surgery (including neuraxial anaesthesia) whilst taking these drugs, the risk of bleeding may be increased. Expert advice should be sought.

Antiplatelet agents

Aspirin

Aspirin inhibits platelet aggregation by acetylation of the catalytic site of the enzyme prostaglandin synthetase to irreversibly inactivate it. This prevents the conversion of arachidonic acid to prostaglandin G_2 and, hence, the cycloxygenase pathway. The production of thromboxane A_2, a potent stimulant of platelet aggregation, is stopped.

- Aspirin is well absorbed orally. The effects on thromboxane A2 production are dose dependent, but complete inhibition of thromboxane A2 production occurs at doses >100mg.
- Since platelets cannot regenerate prostaglandin synthetase, recovery of function needs the production of new platelets, which takes 7–10 days.
- Aspirin is indicated for the prevention of thrombosis in:
  - Patients with symptomatic vascular disease (angina, stroke, atrial fibrillation) (see 🕮 Protecting the heart, p. 143, 🕮 Coronary artery disease, p. 164, Recent stroke or transient ischaemic attacks, p. 189).
  - Acute coronary syndrome NSTEMI.
  - STEMI.
  - After PCI or intracoronary stent (See 🕮 Management of the vascular surgery patient after coronary revascularization, p. 173).
- The major risk of aspirin is bleeding, particularly from the GI tract. There is also an increased risk of perioperative bleeding and in some situations, e.g. neurosurgery, spinal, or ophthalmic surgery, aspirin is commonly stopped before operation.
- There is little evidence that aspirin therapy alone increases the risk of complications from neuraxial procedures.
- Current guidelines suggest that aspirin should be continued throughout the perioperative period in patients undergoing vascular surgery as the benefits outweigh the potential risk from bleeding.

In recent years the management of coronary artery disease has shifted rapidly away from surgery towards PCIs. These require profound inhibition of platelet function for a prolonged period to prevent stent thrombosis and new drugs have been introduced to achieve this.

Clopidogrel

Clopidogrel irreversibly binds to the platelet P2Y12 ADP receptor to block activation of the glycoprotein IIb/IIIa pathway and so inhibit platelet aggregation and fibrin cross-linking. This is the final common pathway for platelet aggregation and so the effect of clopidogrel on platelets is more profound than that seen with aspirin.

- Clopidogrel is rapidly absorbed with a bioavailability of ~50%. It is a prodrug, which is metabolized to a potent and active metabolite by the CYP2C19 isoform of cytochrome P450 enzymes. The duration of action of the metabolite is ~8h.
- Genetically determined low CYP2C19 activity occurs in 14% of patients, and they have a reduced response to clopidogrel.
- Absorption also varies between individuals. The effect of clopidogrel on platelet function varies widely—some patients have almost no response, and therefore no beneficial effect and some have a profound response with a greater risk of adverse effects.
- The indications for clopidogrel are similar to those for aspirin:
  - Patients with symptomatic atherosclerosis (angina, stroke, AF) (see 🕮 Coronary artery disease, p. 164, Recent stroke or transient ischaemic attacks, p. 189).
  - Acute coronary syndrome without ST-segment elevation (NSTEMI).
  - STEMI.
  - After PCI or intracoronary stent (see 🕮 Valvular heart disease, p. 171).
- The adverse effects of clopidogrel are also similar to aspirin. However, by virtue of their different mechanisms of action, aspirin and clopidogrel may also be used together as dual anti-platelet therapy after some types of PCI.
- Clopidogrel increases the risk of perioperative bleeding. This can be a significant problem in some types of surgery (cardiac, spinal or neurosurgery). Anecdotal reports of life-threatening surgical haemorrhage exist, and almost certainly represent patients who are genetically sensitive to the effects of clopidogrel.
- It is not recommended that neuraxial blocks are performed in patients taking clopidogrel because of the potential for spinal haematoma formation.
- Stopping clopidogrel after PCI or intracoronary stenting increases risk of thrombosis. Risks are highest within the first 6 weeks after a bare metal intracoronary stent, or within 6 weeks after a drug-eluting stent.
- Perioperative management of patients taking clopidogrel is therefore complex, and depends on the indication for the clopidogrel and the potential for, and consequences of, bleeding.
- Current guidelines suggest that all elective surgery should be deferred for >6 weeks after BMS, and for >6 months after a drug-eluting stent. If urgent vascular surgery must be performed within these times, dual antiplatelet therapy (aspirin and clopidogrel) should be continued.
- In all cases, it is advisable to discuss the management of these patients with both surgeon and cardiologist.
- If clopidogrel is stopped, it should only be restarted when the risks of surgical bleeding have subsided. The therapeutic effects of maintenance dose (75mg) clopidogrel take several days, so a loading dose (300mg) is often required.

Other antiplatelet drugs

The phosphodiesterase inhibitors dipyridamole and cilostazol are used in patients with vascular disease for their vasodilator properties, but they also inhibit platelet aggregation. Used alone, these drugs are unlikely to cause significant perioperative bleeding, but they are often used in combination with aspirin and in this case consideration should be given to stopping one of the drugs.

Future developments in antiplatelet therapy

- The two main antiplatelet drugs currently in clinical use are irreversible, have widely variable effects between individuals and there is no readily available way to monitor their effects.
- Thromboelastography and the platelet function analyzing monitor are new ways of testing platelet function either in the laboratory or in a near-patient form (see 📖 Point of care coagulation monitoring, p. 319). They can discriminate between aspirin and other effects on platelets and should be used when available.
- Drugs with short-acting and reversible binding to the P2Y12 receptor are close to licensing. These include prasugrel (which has less pharmacokinetic variability than clopidogrel), ticagrelor (which has reversible effects with half-life of 7h), and cangrelor (an IV short-acting platelet ADP receptor antagonist).

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Chapter 5

Principles of perioperative care

Principles of general anaesthesia

Introduction

General anaesthesia provides a reversible state of unconsciousness in response to a combination of drugs that ensure hypnosis, amnesia, analgesia, and, where appropriate relaxation of skeletal muscle. Recent evidence suggests that general anesthesia is a reversible drug-induced coma. For many vascular operations GA, is combined with regional and/or local anaesthesia. RA or local anaesthetic (LA) techniques are sometimes used on their own.

The type of anaesthetic chosen is a combination of clinician judgement and patient choice.

Regional anaesthesia

- Is claimed to be safer than general anaesthesia.
- Was shown to reduce mortality by over 30% in one systematic review but not in others.
- Reduces the risk of pulmonary complications and deep vein thrombosis.
- Is often provided by a combination of local anaesthetic and opioid.

General anaesthesia

Provided by:

- IV or inhalation anaesthetics.
- Supplemented by the administration of opioids.

Since patients undergoing vascular surgery generally have a high incidence of coronary and/or hypertensive heart disease, cardiovascular stability is essential. However, GA agents have profound effects on the heart and the circulation.

Volatile and IV anaesthetics

Exert effects on:

- The heart (–ve inotropy).
- The peripheral vessels (systemic vasodilatation).
- The autonomic nervous system (depression or stimulation).
- The coronary circulation.

Negative inotropy

Volatile agents (isoflurane, desflurane, sevoflurane) and IV agents (thiopentone and propofol) decrease myocardial contractility (–ve inotropy) in a dose–dependent fashion. This results from a reduction of Ca^{2+} fluxes across both the membranes of the cardiac cells and the sarcoplasmic reticulum. Reduced sensitivity of the contractile proteins to calcium also plays a role.

Peripheral vasodilatation

With the exception of ketamine, induction of anaesthesia usually decreases peripheral vascular resistance. In addition, isoflurane, desflurane, and sevoflurane cause a dose–dependent reduction of peripheral vascular resistance. Peripheral vasodilatation, venodilatation, and depression of contractility all contribute to hypotension.

Effect on the autonomic nervous system

Surgery increases sympathetic nervous activity. Anaesthetic agents may decrease sympathetic activity (halothane, enflurane) or increase sympathetic activity causing tachycardia (ketamine, diethyl ether, step increases in desflurane concentration, nitrous oxide).

Effect on cardiac output

Negative inotropy and venodilatation might be expected to decrease cardiac output. The reduction may be minimized by anaesthesia-induced peripheral vasodilatation (as with isoflurane and sevoflurane). Propofol does not cause peripheral vasodilatation and, typically, decreases cardiac output in a dose–dependent fashion.

Effect on the coronary circulation

The effects of inhalation anaesthetics on the coronary circulation depend upon its local regulation and direct effects on coronary vascular tone:

- Reduced BP and contractility decrease myocardial O_2 consumption.
- Local regulation decreases coronary blood flow in response to decreased demand.
- Isoflurane, desflurane, and sevoflurane cause coronary vasodilatation so that coronary blood flow is maintained even though oxygen demand is reduced (luxury perfusion). In patients with CAD, luxury perfusion may be at the expense of poorer perfusion of compromised myocardium. It can cause myocardial ischaemia (coronary steal).

Gaseous anaesthetics

Nitrous oxide

Used extensively. Its myocardial depressant effect is offset by increased sympathetic activity resulting in beta-adrenoceptor activation (positive inotropy) and alpha-adrenoceptor stimulation (vasoconstriction). Nitrous oxide thus has little effect on BP. It is a potent greenhouse gas; environmental considerations militate in favour of a reduction of its use.

Xenon

An inert or 'noble' gas. In experimental studies xenon administration does not alter MAP and cardiac output. Xenon is not a greenhouse gas but it is very expensive.

High dose opioid anaesthesia

Remifentanil confers cardiovascular stability, but a high incidence of shivering (60%) has been reported.

- The rapid offset of action and intense analgesia are valuable characteristics, but the increased O_2 consumption caused by shivering may be detrimental in patients with coronary heart disease who have limited coronary reserve.
- Remifentanil is associated with clinical signs of deeper analgesia and anaesthesia, such as fewer responses to noxious stimuli, less hypertension, but more frequent episodes of bradycardia and hypotension.
- Attenuation of haemodynamic, autonomic, and somatic intraoperative responses is beneficial.
- The rapid offset of action and intense analgesia are valuable characteristics.

Total intravenous anaesthesia (TIVA)

Propofol and remifentanil are used the most frequently. Microprocessor-controlled infusion devices make it possible to adjust the infusion rate as a function of patients' characteristics and required depth of anaesthesia (target-controlled infusion). Pharmacokinetic models make it possible to reach an effective concentration of the agents very rapidly and to reach steady state promptly.

Anaesthetic agents and the ischaemic myocardium

- *In models of acute coronary occlusion:* most anaesthetic agents decrease the size of MI.
- *In models of myocardial stunning* (brief ischaemia followed by reperfusion), volatile anaesthetics, and opioids facilitate recovery of function

Pharmacological myocardial pre- and post-conditioning

Ischaemic pre- and post-conditioning

Result from the application of very short periods of ischaemia preceding (preconditioning), or following (post-conditioning), a more prolonged episode of ischaemia. This is known to reduce infarct size.

Pharmacological pre-and post-conditioning

Result from the administration of a drug before or after the ischaemic insult and also decrease infarct size. Experimentally, many anaesthetic agents protect the myocardium by pharmacological pre- or post-conditioning through proteinkinase C epsilon (PKCε) acting on the mitochondrial transition permeability pore (MTPP).

Large human studies (>10 000 patients), have shown sevoflurane and propofol to precondition the myocardium in cardiac surgery. A meta-analysis of 22 RCTs (approximately 1900 patients) found significant reductions in myocardial infarction and mortality when volatile anaesthetics (desflurane, sevoflurane) are used for anaesthetic maintenance, rather than propofol.

The clinical relevance of myocardial pre- or post-conditioning is still unclear and may remain so as, other than in experimental models, in vivo studies necessarily involve a combination of agents. Hence, protective effects may be obscured. However, recent evidence suggests that volatile anaesthetics protect other organ systems as well as the heart.

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Infection control and antimicrobial prophylaxis

Infection in vascular surgery

- Prosthetic vascular graft infection (PVGI) is a serious and sometimes life-threatening complication.
- Rates of PVGI vary between 1 and 6%, with higher rates for lower extremity reconstructions, which have a higher mortality (33–58% of infected aortic grafts, c.f. 22% of infra-inguinal PVGI).
- Most infections present within 30 days, the more pathogenic organisms (meticillin-resistant *Staphylococcus aureus* (MRSA), beta-haemolytic streptococci and *Pseudomonas aeruginosa*) presenting with obvious signs of sepsis. They may cause anastomotic breakdown and consequent haemorrhage
- The major causes of PVGI are Gram +ve organisms (staphylococci and streptococci) from the patients' skin. Infection occurs at the time of surgery or by direct spread in the early post-operative period. In 2000, MRSA was the leading cause of graft infection. Such infections are frequently lethal, but if successfully treated, are associated with prolonged hospital stays and a higher rate of amputation
- Other sources of sepsis include thrombus already seeded with bacteria, and haematogenous or lymphatic spread from infected leg ulcers or gangrenous tissue.

- Biofilm formation on prosthetic graft material may allow bacteria of relatively low pathogenicity such as coagulase –ve staphylococci (CoNS) to cause infections. These may be more indolent and present later.
- Anaerobic PVGI is rare and may be caused by translocation of gut organisms during intestinal handling. Anaerobic infections following amputation arise in necrotic tissue. The organisms are derived from rectal/perineal flora, which is predominantly a faecal veneer of enterococci, anaerobes and Gram –ve bacteria, with some skin staphylococci.

Factors predisposing to infection

- MRSA colonization (especially nursing home residents).
- Re-operation/re-exploration/tissue loss.
- Emergency operations.
- Long length of stay (>7 days).
- ICU admission.
- DM, obesity, cancer, and immunosuppression.
- Delayed wound healing.
- Lymphoedema.

Infection control

- Universal precautions, hand hygiene, and strict aseptic technique are essential.
- Identification and segregation of MRSA patients in isolation wards can result in dramatic decreases in MRSA acquisition.
- In the MRSA-colonized patient, nasal mupirocin and topical decolonization significantly lessens the bio-burden of bacteria on the skin, but until skin lesions and leg ulcers heal and urinary catheters are removed, MRSA eradication is unlikely to be successful.
- The value of pre-operative antiseptic baths (e.g. triclosan or chlorhexidine) is debatable.

Pre-operative MRSA screening

- Routine culture from pre-op clinic necessitates 24–48h incubation before the results are available,
- The site(s) of swabbing vary: nose swab, throat swab +/– perineal swab pooled for culture on selective agar for maximal detection rates.
- Molecular techniques (polymerase chain reaction (PCR)) may be used with nasal swabs to provide results within hours. They are expensive.
- Hospital acquisition of MRSA usually occurs in ICU or on vascular wards. When non-surgical patients (outliers) are present on surgical wards, there may be increased risk of unrecognized exposure to MRSA carriers.
- Glycopeptide antibiotic prophylaxis (vancomycin and teicoplanin) ensures cover against MRSA and meticillin-resistant CoNS.

There is little point screening staff for MRSA carriage unless there is evidence of an outbreak. The carrier/shedder is usually apparent and frequently has a history of skin problems.

Anti-microbial prophylaxis

Each vascular unit should create its own protocol, based on the nature of surgery and local prevailing sensitivities.

Therapeutic options

The spectrum of antimicrobial cover to be expected is illustrated in Table 5.1.

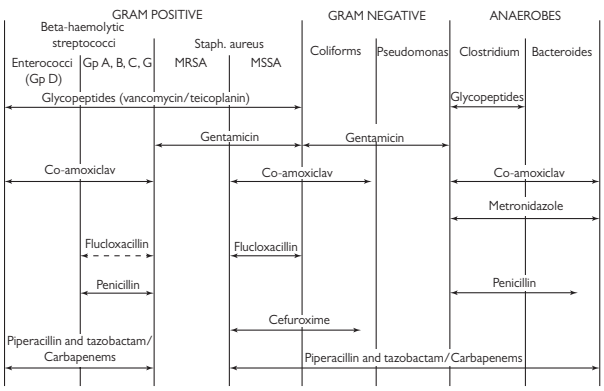
Gram +ve cocci are the major cause of PVGI. Sensitivity patterns of staphylococci vary widely between hospitals. Older prophylactic regimens involved varying combinations of cephalosporins, co-amoxiclav, and latterly glycopeptides, with varying results. As the antibiotic susceptibility of *S. aureus* has decreased, so has the sensitivity of CoNS, with most now resistant to flucloxacillin

- Penicillin-allergic patients may be given cephalosporins if a minor allergy such as skin rash is described, but cephalosporins and carbapenems (imipenem/meropenem) are contraindicated if there is a history of anaphylaxis to penicillin.
- The glycopeptides (vancomycin and teicoplanin) provide the best cover for Gram +ve organisms, with almost 100% of staphylococci being sensitive.
- Antimicrobial prophylaxis should be ideally be given at least 30min prior to incision.

Vancomycin

- Optimal timing of drug administration is difficult to achieve in practice.
- Vancomycin should be started 1h before 'knife to skin' for optimal prophylaxis.
- The drug should be infused over 2h to avoid the 'red man' syndrome caused by histamine release.

Table 5.1 Antibacterial spectra of major groups of antimicrobials used for prophylaxis in vascular surgery



Teicoplanin

- Quick and easy to administer (400mg slow IV injection at induction).
- The dose may be increased to 800mg if the patient is known to be MRSA +ve.
- A second 400-mg dose recommended if operation is >4h duration or major haemodilution/haemorrhage occurs.

Gentamicin

- Quick and easy to administer.
- Covers most staphylococci, and almost all Gram –ve organisms.
- 3mg/kg IV gives excellent levels in tissue and serum.

Rifampicin

- Graft impregnation or soaking has been used, but trial data do not show a demonstrable reduction in PVGI. This may be because the drug is diluted when the graft is irrigated or reperfused, or the initial concentration used is too low.
- In theory rifampicin should remain active locally in effective concentrations for several days.
- Rifampicin can penetrate biofilm, which forms on the prosthesis.

Treatment of an established graft infection

- Antimicrobial choice should be guided by prevailing organisms and their antibiotics sensitivities. Consultation with microbiologist advisable.
- A glycopeptide, gentamicin, and carbapenem (imipenem/meropenem) regime covers most pathogens.
- For fungal infections, consult a microbiologist.

Antibiotic prophylaxis for amputation

- The old 'triple therapy' of flucloxacillin, penicillin, and metronidazole does not cover coliforms, *Pseudomonas*, or MRSA.
- Three doses of co-amoxiclav with a single dose of vancomycin/teicoplanin is the best option if there is concern about MRSA.

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Intravenous fluid therapy and blood product management

During vascular surgery the anaesthetist plays a pivotal role in fluid management, and the transfusion of blood and blood components. Major vascular surgical procedures may be associated with marked fluid shifts and large volume blood loss. The vascular anaesthetist must be able to assess the patient's physiological reserve and use appropriate therapeutic interventions to maintain oxygen delivery to minimize the likelihood of end organ damage. The development of minimally invasive techniques which permit

the assessment of stroke volume and cardiac output (oesophageal Doppler or pulse contour analysis) coupled with an improved understanding of the physiology of anaemia has led to improved fluid administration regimes and more appropriate use of blood and blood products, tailoring requirements to the individual.

Physiological response to surgery

- The stress response to surgery promotes anti- diuresis and oliguria promoted by increased levels of vasopressin, catecholamines and activation of the renin-angiotensin-aldosterone system (RAAS).
- The ability of the body to excrete free water after a surgical insult is reduced because the kidney's ability to dilute, as well as concentrate urine is reduced. Thus, hypotonic fluid administration risks dilutional hyponatraemia.
- If normal saline is infused Cl^- and Na^+ overload may occur. Hyperchloraemia may cause renal vasoconstriction and reduce GFR, further compromising the ability to excrete sodium and water.
- K^+ depletion, due to RAAS activity, and the cellular loss of K^+, which accompanies protein catabolism in the fasted patient, reduces the ability to excrete a Na^+ load.
- AKI may occur in vascular surgery due to abdominal compartment syndrome and increased renal capsular pressure due to oedematous renal tissue.
- Increased capillary permeability may allow albumin and attendant fluid to leak into the interstitial space, worsening interstitial oedema. This may cause intravascular hypovolaemia, stimulating vasopressin release and activating the RAAS, which further increases sodium and water retention.
- They key question when confronted with an oliguric patient is whether there is significant intravascular hypovolaemia. This requires careful assessment of clinical parameters of intravascular volume (pulse rate, BP, capillary refill, and CVP measurement) supplemented perioperatively by minimally invasive cardiovascular monitoring.
- Balanced salt solutions (Hartmanns solution) should be used when crystalloid resuscitation or replacement is required to reduce the risk of inducing hyperchloraemic acidosis. Likewise any colloids administered should be formulated in a balanced electrolyte solution, rather than normal saline.
- Hypotonic solutions (5% glucose or glucose saline), although important sources of free water should be used with caution, as dangerous hyponatraemia may occur, particularly in the elderly.
- To meet maintenance requirements a 70 adult should receive 1.5–2.5L water/day with additional amounts to replace deficits or excess losses (Table 5.2). Careful monitoring should be undertaken using clinical examination, fluid balance charts and regular weighing where possible.

Table 5.2 Typical daily requirements of common electrolytes

	Plasma concentration	Daily requirement
Na^+	135–145mmol/L	1–1.5mmol/kg/day
K^+	3.5–5.0mmol/L	1–1.5mmol/kg/day
Mg^{2+}	0.75–1.05mmol/L	0.1–0.2mmol/kg/day
Ca^{2+}	2.12–2.65mmol/L (total) 1.0mmol/L (ionized)	0.1–0.2mmol/kg/day
Cl^-	95–105mmol/L	0.07–0.22mmol/kg/day
phosphate	0.8–1.45mmol/L	20–40mmol/kg/day

Perioperative fluid management

- Patients for elective vascular procedures with normal gastric emptying should receive clear non-particulate fluids until 2h prior to surgery.
- In the absence of diabetes or disorders of gastric emptying, the administration of carbohydrate rich beverages 2–3h prior to surgery may improve well-being and facilitate post-operative recovery.
- Open operations, such as AAA repair are associated with evaporative losses of up to 10mL/kh/h. This must be added to the normal maintenance requirements for sodium and water (Table 5.3).
- Patients undergoing major vascular surgery are a high risk group due to the high incidence of comorbidity. Where possible, minimally invasive cardiovascular monitoring techniques should be used to enable treatment with fluids and inotropes to achieve predetermined goals for stroke volume, cardiac output, and O_2 delivery, as this may improve outcome.
- Not all vascular surgical procedures permit the use of minimally invasive monitoring. In such cases, fluid management decisions have to be based on careful assessment of clinical parameters—pulse rate, BP, capillary refill, urine output, and CVP measurement.
- Intraoperative fluid management should be individually titrated to achieve an optimal value of stroke volume where possible. This may help to improve outcomes and reduce hospital length of stay. Where possible, this targeted volume management should be continued into the early post-operative period, supplemented in some patients by low dose infusions of an inotrope, such as dopexamine.
- The post-operative fluid regime should be titrated to individual patient's needs. The aim should be to restore patients to their normal weight and minimize the development of interstitial oedema.

Table 5.3 Hourly water Na^+ and K^+ losses during preoperative starvation (70kg/man)

Water loss (mL)	Na^+ loss (mmol)	K^+ loss (mmol)
105	3	3

- If patients become oedematous post-operatively, this should be treated with gradual persistent –ve Na^+ and water balance based on urine Na^+ concentration or excretion. Diuretics should be used with caution because of the risk of hypovolaemia. Plasma K^+ concentration should be monitored and maintained greater than 4mmol/L. Hypovolaeamia must be treated.
- Early enteral nutrition should be encouraged and IV fluids discontinued as soon as possible.

See Table 5.4 for the composition of commonly used IV fluids.

Blood products

Over 2 million units of blood and blood products were issued by the blood transfusion services of England, Scotland, Ireland and Wales per annum. There has been a steady fall in the number of units of packed red cells issued since 1999, which reflects the adoption of restrictive blood transfusion practices by clinicians. All donated blood is tested for HIV-1, HIV-2, hepatitis B, hepatitis C, and syphilis. Cytomegalovirus (CMV) antibody –ve blood components are available for immunosuppressed patients and neonates. In the UK, white cells are routinely removed from blood components as a precaution against new variant Creutzfeldt–Jacob disease (vCJD), leaving a residual leucocyte count of $<5 \times 10^6$/U. This also reduces the risk of CMV transmission.

Table 5.4 Composition of commonly used IV fluids

	Na	K	Cl	Osmolarity	Average Mol Wt	Plasma expansion (h)
Plasma	136–145	3.5–5	98–105	280–300		
Glucose 5%	0	0	0	278		
Glucose 4%, saline 0.18%	30	0	30	283		
Saline 0.9%	154	0	154	308		0.2
Hartmanns	131	5	111	275		0.2
Gelatine 4%	145	0	145	290	30 000	1–2
Gelatine 4% (Isoplex)	155	4	105	284	30 000	1–2

Whole blood is processed into blood components (Table 5.5):

- *Packed red cells:* most of the plasma is removed, then packed red cells are re-suspended in an optimal additive solution—saline, adenine, glucose, and mannitol (SAG-M). Total volume is 80–350mL and haematocrit is 50–70%. 4mL/kg packed red cells (280mL for a 70-kg man) typically raises the haemoglobin by 1g/dL.
- *Platelets:* one adult therapeutic dose (ATD) may be prepared from four to six donations of whole blood, by centrifugation, or from a single donor by platelet phoresis. One ATD contains $2.5–3 \times 10^{11}$ platelets and typically increases the platelet count by $20–40 \times 10^{9}$.
- *FFP and cryoprecipitate:*
  - FFP is produced either by centrifugation of whole blood or by apheresis. Plasma is rapidly frozen to maintain activity of labile clotting factors, this produces an average 'unit' of FFP of about 275mL.
  - Cryoprecipitate is obtained from controlled thawing of a single donation of FFP, which precipitates high molecular weight proteins including factor VIII, vWF, factor XIII, and fibrinogen. It is supplied as pooled units—each pooled unit contains cryoprecipitate from five donors. Two pooled units will typically raise the fibrinogen level by 1g/dL.

Table 5.5 Composition of blood and blood products

	Red cells	Platelets	FFP	Cryoprecipitate
Storage temperature	2–6°C	20–24°C on an agitator rack	–30°C	–30°C
Shelf life	35 days	5 days	1-yr (frozen)	1-yr (frozen)
Longest time from leaving controlled storage to completing infusion	Transfuse within 30min of removal from blood fridge. Transfuse unit over maximum of 4h	Start transfusion as soon as received from blood bank. Transfuse unit within 30min	Once thawed, should be infused ASAP	4h
Compatibility testing requirement	Must be compatible with recipient ABO and Rh D type	Preferably ABO identical with patient. Rhesus –ve females under the age of 45yr should be given Rh D –ve platelets	FFP and cryoprecipitate should be ABO compatible to avoid risk of haemolysis caused by donor anti-A or anti-B	
Administration	Infuse through a blood administration set—platelet concentrates should not be infused through giving sets that have been used for blood. Record details of each blood component infusion in the patient's case record. 100% traceability is a legal requirement			

Blood conservation techniques

Blood conservation techniques rely upon:

- Increasing the patient's red blood cell mass.
- Decreasing perioperative blood loss.
- Optimizing blood transfusion practices, including both allogeneic (from another human) and autologous (re-infusion of the patient's own blood) transfusion.

Preoperative management

- Perioperative anaemia is frequent and is associated with increased morbidity and mortality, especially in patients with cardiovascular disease. Anaemia should be investigated and treated. Iron deficiency should be treated with oral or IV iron
- Rarely red cell mass may be increased using recombinant human erythropoietin (EPO) to stimulate erythropoesis. This needs to be started 3–4 weeks prior to surgery for maximum effect. It's use has been described to permit major vascular surgery in Jehovah Witnesses without exposure to allogeneic blood
- Rarely preoperative autologous donation (PreAD) may be used. Patients donate a unit of blood per week in the month prior to their operation. PreAD is labour intensive and depends on good organization both of collection and storage of blood, and co-ordination of operating lists with guaranteed operating dates. Cost-effectiveness is low, mainly because of a high proportion of discarded units. It is not widely used in the UK. Blood transfusion may be more common in patients undergoing PreAD, possibly due to more liberal use of autologous blood. Disadvantages include:
  - Transfusion of wrong blood, due to clerical and laboratory errors.
  - Wastage of collected blood.
  - Circulatory overload due to transfusion of whole blood.

Intra-operative blood conservation techniques

- *Surgical methods to minimize blood loss include:* meticulous surgical technique, minimally invasive surgery (e.g. endoluminal grafts for abdominal aortic aneurysms), local vasoconstriction with adrenaline, topical haemostatic agents (fibrin glues), tourniquets, and surgical devices, e.g. ultrasonic/laser scalpels.
- *Anaesthetic techniques include:* avoidance of venous congestion (patient positioning), avoidance of high intra-thoracic pressures, hypercapnia and hypothermia. Central neuraxial anaesthesia aids blood conservation by reducing both arterial and venous pressures.
- *Pharmacological techniques may rarely be employed:* excessive fibrinolysis if identified may be treated with antifibrinolyic agents—tranexamic acid is the drug of choice.
- Autologous transfusion.

Acute normovolaemic haemodilution (ANH)

- This involves the immediate preoperative collection of whole blood from the patient with simultaneous infusion of crystalloid or colloid to maintain normovolaemia. It is usually performed in the anaesthetic

room. Venesection can be undertaken using a large bore IV cannula into citrated blood bags (available from Blood Transfusion).
- The volume of blood to be removed to achieve the desired haematocrit can be calculated using the following formula:

$$V = EBV \times (H_I - H_F)H_{av}$$

where: EBV is the estimated blood volume (70mL/kg); $H_I$is the initial haematocrit; H_F is the final haematocrit; and H_{AV} is the average haematocrit (mean of H_I and H_F).
- Once collected, bags should be labelled and stored at room temperature for reinfusion once surgical blood loss has ceased. They must be reinfused to the patient within 6h.
- Mathematical modelling has suggested that severe haemodilution (perioperative haematocrit less than 20%) accompanied by substantial blood losses would be required before the red cell volume saved becomes clinically important.
- Current UK guidelines state that acute normovolaemic haemodilution should be considered when the potential surgical blood loss is likely to exceed 20% of the blood volume. Patients should have preoperative haemoglobin of more than 10g/dL and not have severe myocardial disease, such as moderate to severe left ventricular impairment, unstable angina, severe aortic stenosis or critical left main stem coronary artery disease.
- ANH has several advantages over autologous blood donation. The blood procured by haemodilution requires no testing. The units are not removed from the operating theatre so that the possibility of an administrative error resulting in an ABO incompatible blood transfusion is virtually eliminated, as is the risk of bacterial contamination. Finally, blood obtained by haemodilution does not require substantial investment of time by the patient as it is obtained at the time of surgery. Despite this ANH is rarely used for major vascular procedures in the UK

Intraoperative cell salvage (IOCS)

- Most IOCS machines depend on a centrifugal principle using a collection bowl that spins and separates the red cells from the plasma, white cells, and platelets.
- Shed blood is aspirated into a collection reservoir via heparinized or citrated tubing.
- The cells are separated by haemoconcentration and differential centrifugation and finally washed in 1–2L normal saline. This removes circulating fibrin, debris, plasma, leucocytes, microaggregates, complement, platelets, free haemoglobin, circulating pro-coagulants, and most of the heparin.
- The end product of the process is packed red cells resuspended in saline with a haematocrit of 50–80%.
- Salvaged red cells are superior to or at least equal to banked homologous blood in terms of red cell survival, pH, 2,3 diphosphoglycerate (2,3 DPG) and K^+ levels.

- ICS devices can provide the equivalent of 10U bank blood/h in cases of massive bleeding. The technique is ideally suited to open aortic surgery.
- Some Jehovah's Witnesses may accept ICS provided the equipment is set up in continuity with the circulation. Specific consent must be obtained.
- Topical clotting agents, such as collagen, cellulose, gelatine, and thrombin and topical antibiotics or cleansing agents used in the operative field should not be aspirated into a cell salvage machine.

Transfusion triggers in patients presenting for vascular surgery

- Although the coronary vascular bed receives only 5% of cardiac output (250mL/min at rest), it accounts for 11% of the total body oxygen uptake.
- Oxygen extraction is high at rest (65%) consequently coronary venous blood oxygen content is very low (5mL O_2/100mL blood).
- Increased myocardial O_2 requirements during exercise or stress are met by an increase in coronary blood flow, which requires coronary arterial dilatation. Patients with fixed coronary stenoses may have problems matching myocardial O_2 supply to demand during exercise or stress, which may precipitate myocardial ischaemia in areas distal to the narrowed coronary vessels.
- Myocardial oxygen supply and demand are even more finely balanced in the anaemic patient. Normal myocardial O_2 delivery (assuming a Hb of 13.5g/dL) is 40mL/min. Resting global myocardial oxygen consumption is 25mL/min. A reduction in Hb to 8g/dL reduces global myocardial oxygen delivery to 25mL/min, removing this margin of safety.
- Since vascular surgical patients have a high incidence of coronary artery disease a higher transfusion trigger of 8.5–9g/dL is suggested to minimize the likelihood of myocardial ischaemia secondary to anaemia.

Further reading

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Perioperative renal protection

Acute renal failure is defined as an abrupt and sustained decrease in renal function resulting in retention of nitrogenous and non-nitrogenous waste products. The term has been replaced by the term acute kidney injury (AKI) and is now defined by the International Kidney Disease: Improving Global Outcomes (KDIGO) definition of acute kidney injury, which classifies acute kidney injury into three grades of damage and two of loss of function, according to biochemical markers and urine output. The new criteria recognize that acute renal dysfunction is a spectrum and that even small changes in serum creatinine have important prognostic implications (Tables 5.6 and 5.7). Perioperative AKI significantly increases the risk of morbidity and mortality after major vascular surgery so risk assessment and prevention are paramount.

Incidence

- 2–3% after infrarenal aortic surgery.
- 3–4% after cardiac surgery.
- 2–3% after radiocontrast injection.
- 3–14% after thoracic aortic surgery.
- 5–8% after suprarenal or supracoeliac aortic surgery.
- 25–40% after emergency abdominal aortic surgery.

Table 5.6 Modified RIFLE criteria for diagnosis of AKI. Acute kidney injury should be both abrupt (within 1–7 days) and sustained (more than 24h). Classification depends on changes in either serum creatinine or urine output (worst of either). Classifies AKI according to severity and allows progression from one category to another

Stage	GFR criteria	Urine output criteria
Risk	Serum creatinine increased 1.5-fold	<0.5mL/kg/h for >6h
Injury	Serum creatinine increased 2-fold	<0.5mL/kg/h for >12h
Failure	Serum creatinine increased 3-fold or creatinine >355μmol/L with an acute increase of >44μmol/L	<0.3mL/kg/h for 24h or anuria for 12h
Loss	Persistent acute renal failure = loss of function for >4 weeks	
End stage renal disease	End-stage renal disease for >3 months	

All occurrences of AKI are associated with increased hospital morbidity, mortality and worse long-term outcome. Adjusted hazards ratios for 10-yr mortality after major surgery are 1.2 (RIFLE stage: Risk) 1.6 (RIFLE stage: Failure). Risk is increased (AHR = 1.2) even in patients whose renal function returns to normal at hospital discharge.

Adapted from Bellomo R, et al., 'Acute renal failure—definition, outcome measures, animal models, fluid therapy and information technology needs: the second international consensus conference of the Acute Dialysis Quality Initiative (ADQI) group', Critical Care, 8, pp. R204–R212, http://ccforum.com/content/8/4/R204, under the creative commons licence.

Aetiology

Most cases of perioperative AKI result from acute tubular necrosis whether resulting from pre-renal hypoperfusion, hypoxia, or ischaemia-reperfusion injury, sepsis or the direct effects of nephrotoxins. Potential causes are:

- Pre-existing CKD.
- *Pre-renal ischaemia:*
  - Hypovolaemia.
  - Hypotension.
  - Hypoxia.
  - Relative hypovolaemia, e.g. heart failure, cirrhosis, sepsis.
  - Renal vascular disease, e.g. renal atherosclerosis, renal artery stenosis.
  - Surgical occlusion, e.g. aortic cross clamp (AXC), endovascular occlusion.
  - Redistribution of renal blood flow, e.g. NSAIDs, ACE inhibitors, ARBs, direct vasoconstrictors.
  - Intra-abdominal hypertension.
- *Direct renal damage:*
  - Ischaemia reperfusion injury (surgical revascularization).
  - Athero-embolism at surgery.
  - Exogenous nephrotoxins, e.g. radio-contrast, gentamicin.
- *Post-renal:*
  - Bladder outlet obstruction.
  - Intra-abdominal hypertension.
  - Bilateral ureteric damage or obstruction.

These may occur in the pre-, per-, or post-operative periods.

Pathogenesis

Renal ischaemia, hypoxia, sepsis, and nephrotoxins all lead to activation of several interacting inflammatory pathways involving immune cells, cytokines and other vasoactive mediators (e.g. TNF-α, IL-6, nitric oxide, reactive oxygen species, caspases). These mediate intra-renal microcirculatory dysfunction (vasoconstriction, endothelial dysfunction, impaired autoregulation) and direct tubular epithelial damage (tubular obstruction, back-leak, interstitial inflammation, and cell apoptosis), which manifest as oliguria and AKI, and also lead to multisystem organ dysfunction.

Table 5.7 AKIN criteria

Stage	GFR criteria	Urine output criteria
1	Serum creatinine increased 1.5-fold or acute increase of >25μmol/L	<0.5mL/kg/h for >6h
2	Serum creatinine increased 2-fold	<0.5mL/kg/h for >12h
3	Serum creatinine increased 3-fold or creatinine >355μmol/L with an acute increase of > 44μmol/L	<0.3mL/kg/h for 24h or anuria for 12h

Patients who receive RRT are considered to have met the criteria for Stage 3 irrespective of the stage they are in at the time of starting RRT.

Medical risk factors

Old age, poor LV function, liver disease, obesity, occlusive peripheral vascular disease, COPD.

Intraoperative risk factors

- Pre-existing renal dysfunction (see 📖 The risks of vascular surgery, p. 114).
- Aortic surgery.
- Suprarenal aortic occlusion or AXC.
- Prolonged duration of AXC.
- Emergency or revision aortic surgery.
- High vasopressor requirements or vasopressor infusion.
- Diuretic infusion.

Of these, pre-existing renal dysfunction is probably the best overall predictor of post-operative AKI. In aortic surgery, renal hypoperfusion occurs even with infrarenal AXC; the extent of AKI depends on the site and duration of the cross clamp. The risk of AKI increases markedly with cross clamp duration >30min. The risk of mortality in vascular patients who develop perioperative AKI is up to 30% and up to 50% if renal replacement therapy is required.

General measures

The principles of perioperative renal protection are to identify patients at risk, maintain renal perfusion pressure, to intervene early where appropriate and to avoid nephrotoxins.

- *Avoid dehydration:* encourage clear oral fluids up to 2h before elective surgery. IV hydration using 0.9% saline 1.5mL/kg/h is often used for 8–12 h preoperatively
- Perioperative optimization of the cardiovascular system reduces the risk of AKI. Maintain cardiac output and renal perfusion pressure throughout the perioperative period, using fluids and inotropic drugs as required, guided by appropriate monitoring (see 📖 The risks of vascular surgery, p. 114, 📖 Central venous catheterization, p. 252, 📖 General principles of monitoring, p. 312).
- Targets are: CVP 8–12mmHg, MAP >70mmHg, central venous SvO_2 > 70%.
- Although intraoperative urine output does not relate to the incidence or severity of post-operative AKI, a spontaneous urine output of >0.5mL/kg/h during and after surgery (without diuretic drugs) suggests that renal perfusion is adequate.
- Institute vasopressors or vasodilators early if arterial pressure and cardiac output do not respond to IV fluids.
- *Avoid:*
  - Prolonged intra-abdominal hypertension.
  - High intra-thoracic pressures during and after surgery.
  - *Nephrotoxic drugs*—e.g. gentamicin, amphotericin, ciclosporin, repeated, or high doses of radiocontrast.
  - *Acute perioperative anaemia*—this probably reflects adequacy of resuscitation and intravascular volume status.

Specific measures

No specific drug or measure has been convincingly shown to provide perioperative renal protection (Table 5.8). Some studies have suggested a benefit of natriuretic peptides or prolonged (>48h) treatment with the selective DA_1 agonist fenoldopam to reduce AKI after major surgery, cardiac or vascular surgery, but this is not established. Diuretics (furosemide, mannitol) have been used widely to maintain urine output during vascular surgery in the belief that non-oliguric AKI has a better prognosis than oliguric AKI. However, use of diuretics can lead to:

- Adverse effects on distribution of intrarenal blood flow and inhibition of important feedback mechanisms.
- Reduced circulating volume and perfusion pressure with activation of the renin angiotensin aldosterone system.
- Delays in correcting hypovolaemia or optimizing cardiac output.

Post-operative use of diuretics is discussed in 📖 Perioperative myocardial infarction, p. 474.

Post-operatively, high intra-abdominal or intrathoracic pressures reduce renal perfusion despite normal or raised MAP, and surgical laparostomy should be considered in patients with intra-abdominal hypertension and renal dysfunction.

Table 5.8 Specific measures to maintain perioperative renal function

Medical	Surgical
No benefit	*Benefit*
Dopamine	Minimize the duration of AXC or endovascular aortic occlusion during aortic surgery to <30min
Loop diuretics (furosemide)	
Osmotic diuretics (mannitol)	
N-acetyl cysteine	*Probable benefit*
Theophyllines	Selective cold perfusion of the kidneys or specific renal revascularization during aortic surgery
Ca^{2+} channel blockers, $NaCO_3$, clonidine, statins	
Possible benefit	Delayed surgical closure of the abdomen for 48h where post-operative intra- abdominal hypertension (IAH) is anticipated (e.g. ruptured AAA)
Fenoldopam infusion 0.1mcg/kg/min for 48h	
Natriuretic peptides, e.g. nesiritide [BNP analogue], anaritide [ANP analogue] 0.5mcg/kg/min for 48h	Post-operative surgical decompression of the abdomen when IAH has occurred
Statins (suprarenal EVAR)	

Further reading

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Temperature control and monitoring

Definitions

A distinction must be made between core and peripheral temperature. In non-anaesthetized individuals, a large core-to-peripheral temperature gradient is maintained by adaptive thermoregulatory mechanisms. The definition of abnormal temperature states are defined by alterations in blood temperature. The definitions are;

- *Normothermia:* 36.5–37.5°C.
- *Hyperthermia:* >38°C.
- *Hypothermia:* <36°C.

Measurement of temperature in perioperative setting

Direct blood temperature measurement requires the most invasive techniques. In most situations, indirect core temperature measurement is adequate. Peripheral skin temperature monitoring is the least accurate due to external influences.

Sites of measurement

Listed from most efficient to least efficient:

- *Pulmonary artery:* invasive, but best monitor of blood/core temperature.
- *Oesophagus:* measured at lower oesophagus to avoid artefact caused by respiratory gases.
- *Tympanic membrane:* representative of core temperature as membrane close to carotid artery.
- *Naso/oropharyngeal:* altered by respiratory gases.
- *Intracavity:* bladder, rectum.
- *Axillary:* close to axillary artery.
- Skin surface.

Physiological temperature changes during anaesthesia and surgery

Environmental exposure (radiation (40%), convection (30%), conduction (5%), evaporation (15%), respiratory (10%)) promotes inadvertent hypothermia. The physiology of temperature changes during anaesthesia and surgery are well established, and related more to alterations in the distribution of body heat from core to periphery than to systemic heat imbalance.

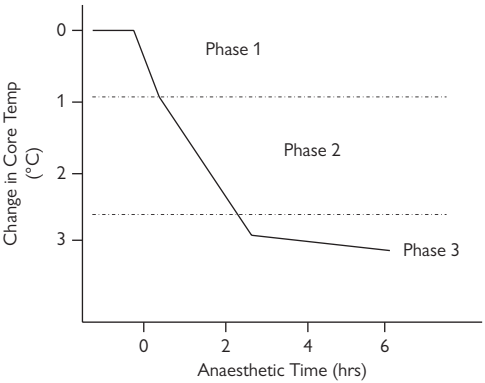

Fig. 5.1 Reduction in core temperature following induction of general anaesthesia.

A reduction in temperature following induction of general anaesthesia occurs in three phases (Fig. 5.1);

- *Phase 1:* redistribution of heat from core to periphery due to inhibition of tonic vasoconstrictive mechanisms. Body heat content remains unchanged, but core temperature is reduced in relation to peripheral temperature.
- *Phase 2:* linear phase. A more gradual reduction in core temperature by a further 1–2°C occurs over the next 2–3h. This is related to environmental exposure, supplemented by hypothalamic influences of anaesthesia preventing thermoregulatory vasoconstriction and shivering
- *Phase 3:* plateau phase. At around 34°C, shivering and vasoconstrictive response returns and balance heat losses. When core heat production equals heat loss to the periphery, core temperature reaches a plateau (despite continued systemic heat loss) and the normal core-to-peripheral temperature gradient re-established.

Temperature changes during central neuraxial blockade

Redistribution is the major initial cause of hypothermia in patients receiving central neuraxial blockade. Core temperature decreases at about half the rate when compared with general anaesthesia. Subsequently, patient core temperature decreases at a rate determined by the difference between heat losses and gains, but since sympathetic blockade prevents thermoregulatory vasoconstriction, prolonged procedures under neuraxial anaesthesia may lead to severe hypothermia. This is especially true when combined with general anaesthesia.

Temperature abnormalities

Hyperthermia

Hyperthermia occurring under anaesthesia (in contrast to fever-induced hyperthermia) is rare, even with active rewarming methods. This is due to preserved mechanisms for heat loss. The major abnormality seen during surgical procedures is hypothermia.

Hypothermia

NICE guidelines have defined the requirement for temperature maintenance in anaesthesia. In most situations, significant hypothermia is detrimental to recovery from anaesthesia and has adverse effects on multiple physiological systems (Table 5.9).

General clinical consequences of hypothermia

- *Postoperative shivering:* core hypothermia leads to adaptive peripheral vasoconstriction. This may be treated by temperature restoration and opiate drugs (e.g. pethidine). Increased O_2 consumption, related to shivering, may lead to deleterious effects on oxygen balance. The increase in O_2 consumption is in the order of 50%.
- *Infection risk:* reduction in macrophage function and innate immune response increases the risk of infection.
- *Coagulopathy:* where IV heparin is administered during vascular procedures, prolonged hypothermia may lead to more significant coagulation abnormalities.
- *Pharmacokinetics:* increased blood/gas solubility leads to a reduction in MAC (5% per 1°C temperature reduction). Delayed recovery from non-depolarizing muscle relaxants may also be evident

Table 5.9 Physiological effects of hypothermia

System	Effect
Metabolism	Post-operative shivering increases total body O_2 consumption
Respiratory	Blunted ventilatory response to CO_2 5% decrease in tissue oxygen requirements for each 3°C of cooling Increased O_2 solubility in blood Increased affinity of Hb for O_2(left shift in HbO_2 curve)
Adrenergic	Activation of sympathetic nervous system Minimal adrenomedullary or adrenocortical response
Cardiovascular	Systemic and pulmonary vasoconstriction. Increased arterial BP
Coagulation	Impaired platelet function, impaired coagulation cascade, enhanced fibrinolysis
Immune	Impaired neutrophil and macrophage function. Decreased tissue partial pressure for oxygen.
Pharmacokinetics	Increased effect of neuromuscular blockers, prolonged duration of neuromuscular blockade, decreased MAC for inhaled anaesthetics

Management of hypothermia

Prevention of initial temperature loss

Active prewarming prior to general anaesthesia and after induction (before surgical incision) may restrict reduction in temperature due to redistribution. Equally prevention of temperature reduction between the end of the procedure and recovery room is important.

Reduction of temperature loss

Measures to reduce intraoperative temperature loss include:

- Increase in ambient air temperature.
- Warming of inspired gases (e.g. heat and moisture exchanger
- (HME) filters).
- Warming of IV fluids
- Passive warming: blankets, etc.

Maintenance of temperature

Active warming is the only means of transferring heat to the patient and is therefore required to maintain normothermia. Active warming can be provided by;

- *Disposable air mattresses:* this is the commonest method of maintaining temperature. Holes in the mattress on the patient side, allow warm air at various temperatures, to be released against skin.
- *Circulating water mattresses:* usually underneath the patient.
- *Radiant warming with overhead heaters:* impractical in adult vascular surgery, but used in paediatrics.

Temperature considerations in vascular anaesthesia

Warming peripheries in ischaemic tissue

Warming extremities and tissues that have been rendered ischaemic and, therefore lack thermoregulatory mechanisms, may lead to thermal injury. This may occur in peripheral procedures on acutely ischaemic limbs or during aortic cross-clamping.

Intentional hypothermia (IAH)

Moderate systemic hypothermia (34°C) may be beneficial where spinal cord or central neurological protection is required. In suprarenal aortic aneurysm repair, where the spinal arteries are at risk of interruption, even moderate hypothermia between 2 and 3°C, reduces the risk of spinal injury and paraplegia, and may be an important protective modality. Hypothermia in juxtarenal and infrarenal aneurysm repair has not been demonstrated to have beneficial effects on postoperative renal dysfunction.

Localized cooling of regional organs by ice (e.g. kidneys, brain) and infusion of cold solutions into the epidural space have been used for the protection of organ function during periods of ischemia, although the efficacy of such intervention is unproven.

Inadvertent hypothermia

Many of the risk factors for the development of IAH are particularly relevant apply to patients undergoing vascular surgery:

- Co-existing comorbidity.
- Increased frequency of combined general and regional anaesthesia.

- Major surgical procedures.
- Prolonged anaesthetic times.
- High preoperative cardiovascular risk.

The development of IAH in vascular patients may lead to:

Increased incidence of post-operative cardiac morbidity

IAH is associated with an increase in postoperative cardiac events. This is especially relevant in vascular patients. A low core temperature (<35°C) is associated with a two- to three-fold increase in the incidence of early post-operative myocardial ischaemia, independent of anaesthetic technique. In one prospective randomized trial, there was a 55% reduction of the relative risk for early post-operative cardiac morbidity in patients who were warmed to normothermia during and after surgery. The incidence of post-operative ventricular tachycardia and morbid cardiac events were reduced in the normothermic group (core temperature 36.7°C) compared with the hypothermic group (core 35.4°C). The mechanism of this increase in cardiac events is unclear, but is likely due to adrenergic mechanisms.

Increased requirement for blood products

Even mild hypothermia (reduction of 1°C) may increase the transfusion of blood products. In emergency vascular procedures, where blood loss may be severe, this is an important consideration

Further reading

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Management of major haemorrhage

- Anaesthetists caring for vascular patients need to be well versed in the management of major blood loss. 7% of elective infrarenal aortic repairs in the 2005 NCEPOD report lost more than 5L of blood.
- Efficient management of major haemorrhage is an organizational challenge, as well as a clinical response supported by laboratory assessments of coagulation. Effective communication between clinical and laboratory staff is pivotal.
- All hospitals should have a massive haemorrhage protocol in place to ensure a timely supply of blood components (Fig. 5.2). There will be subtle differences in these between institutions; anaesthetists should be familiar with their local protocol. The priorities are to arrest the bleeding and treat any acquired anaemia or coagulopathy promptly.
- Rehearsal of the clinical scenario ensures that staff are familiar with the protocol and have a clear understanding of their roles in the emergency situation.

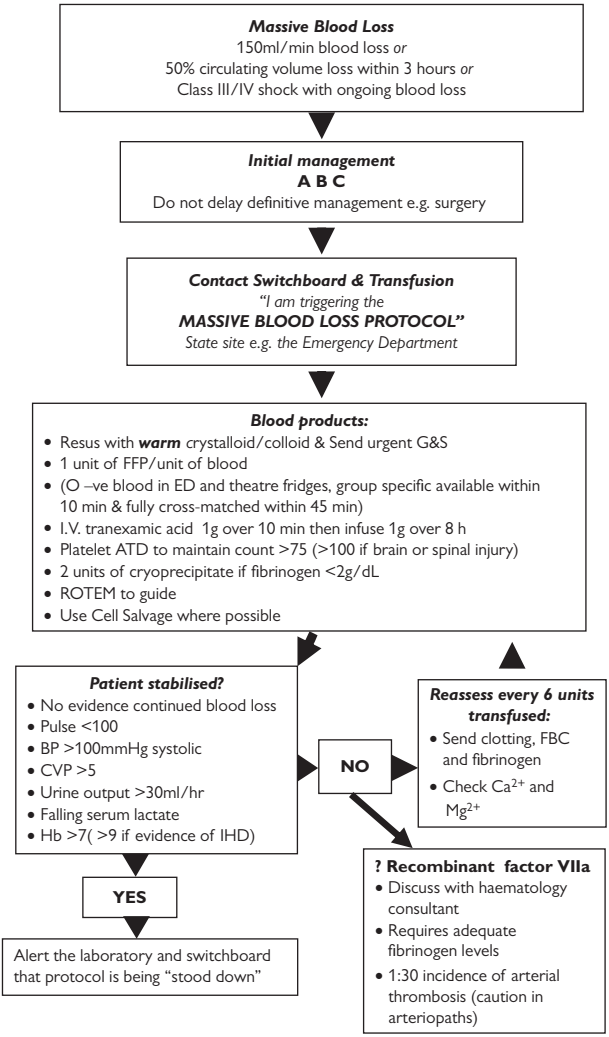

Fig. 5.2 Template for the management of massive haemorrhage. Adapted from the Royal Devon and Exeter Foundation NHS Trust Massive Haemorrhage protocol. This is intended as a guide to treatment - individual institutions will have their own protocols.

Initial actions

- Alert switchboard that you wish to activate the massive bleeding protocol.
- Switchboard contacts key personnel.
- A single communicator with blood bank.
- Prioritize and mobilize the clinical team:
  - Alert the hospital switchboard that a massive haemorrhage is occurring. A clear phrase must be used to ensure the massive haemorrhage protocol is activated.
  - Switchboard must contact key people listed in the protocol. This should include the blood bank biomedical scientist (BioMS), the haematologist on call and the lead porter who may need to allocate a specific person to the collection of blood samples and blood components.
  - A single communicator needs to be chosen for the duration of the resuscitation ensuring that the laboratory only has to deal with one request for components.
  - Prioritize and mobilize the team. When unexpected massive bleeding occurs there is usually a large number of staff in the immediate vicinity that can be mobilized to help. A team leader should be identified to ensure available staff are used effectively.

Diagnosis of massive bleeding

- *Bleeding:* if it occurs during the primary surgery, is frequently obvious. The source may be leaking anastomoses, or malpositioned clamps. Occasionally, it may be difficult to identify the bleeding point, particularly if it is located within a poorly visualized area of the abdomen, e.g. within the pelvis.
- *Post-operative bleeding:* may not be immediately obvious. Have a high index of clinical suspicion if there is ongoing hypotension despite adequate volume replacement, or an unexplained decrease in haematocrit. Seek senior advice. The risks of re-operation should be weighed against the risks of continued haemorrhage.
- Lack of bleeding into drains should not provide a false sense of security. A poorly positioned drain may not provide much useful information.
- A coagulopathy must be excluded. This can be diagnosed by conventional laboratory tests or by point of care (POC) testing using rotational thromboelastography (TEG or ROTEM).

Resuscitation targets: the 4 M's

- Maintain blood volume.
- Maintain an adequate mean arterial BP.
- Maintain O_2 carrying capacity.
- Monitor coagulation.

Maintain blood volume to maintain adequate tissue perfusion

- In the initial resuscitation phase, maintenance of blood volume may be with crystalloid or colloid, as blood may not be immediately available. This may contribute to a dilutional coagulopathy.
- In the majority of cases surgical control can be achieved. Rarely, it may be necessary to pack and apply pressure until blood and blood components become available to avoid unnecessary haemodilution.

Maintain an adequate mean arterial blood pressure

- This prevents end organ damage as profound and prolonged hypotension will lead to cerebral, cardiac and renal ischaemia. In certain situations, e.g ruptured AAA hypotensive resuscitation should be employed (see 🕮 Emergency abdominal aortic aneurysm repair: open and endovascular repair, p. 448).
- A low hourly urine output may be a warning sign of inadequate renal perfusion.
- ECG changes should alert the anaesthetist to the possibility of myocardial ischaemia. This can be improved by transfusion to increase myocardial O_2 delivery, maintaining adequate coronary perfusion pressure, and attempting to avoid a tachycardia, which preferentially shortens diastole and reduces the time for perfusion of the left ventricular muscle.
- Tissue hypoperfusion will produce a metabolic acidosis, which may further impair organ function and coagulation. This may be exacerbated by the development of AKI. Restoration of circulating volume to improve cardiac output and tissue oxygen delivery is vital.
- Resuscitation goals should be:
  - A falling heart rate (<100/min).
  - Restoration of BP (>100mmHg systolic).
  - An adequate central venous pressure (>5mmHg).
  - Satisfactory urine output (>30mL/h or 0.5mg/kg/h).
  - A falling serum lactate on blood gas analysis.

Maintain oxygen carrying capacity

- Administer 100% oxygen.
- Ventilate to normal or low $PaCO_2$ to counteract the any metabolic acidosis and reverse the vasodilatation associated with a high $PaCO_2$.
- Aim for a haemoglobin of 90–100g/L to optimize oxygen carriage. A haemoglobin at this level may aid coagulation, particularly when other coagulation factors may be below normal. The Hemocue is helpful to monitor haemoglobin concentration.
- Administer group O non-cross-matched red blood cells (RBC) if the blood group is unknown in an extreme emergency. Premenopausal females should be given O Rh(D) –ve RBC in order to avoid sensitization and the risk of haemolytic disease of the newborn in subsequent pregnancy.
- Group-specific RBC should be given at the earliest possible opportunity as group O blood is a scarce resource. These will be available within 10min of an urgent group and save sample being received in the laboratory in the vast majority of patients

Monitor coagulation

- Keep INR <1.5, PT <15s.
- Consider administration of prothrombin complex concentrate (PCC) if the prolonged INR is due to warfarin.
- Normalize the activated partial thromboplastin time ratio (APTTR) with protamine if heparin has been administered.
- Keep platelet count >75 000. In some hospitals it may be necessary to check availability. Transport times must be considered as it may be necessary to order platelets from the regional blood transfusion centre.
- Keep fibrinogen >2g/L. Early use of FFP may avoid the need for cryoprecipitate. (5U FFP contains the same quantity of fibrinogen as 2 pooled units of cryoprecipitate). If fibrinogen levels are <2g/L, 2 packs of pooled cryoprecipitate should be given. (1 pack contains pooled donations from 5 donors).
- Maintain normothermia, hypothermia is an important contributor to continued bleeding and adverse patient outcomes. It causes:
  - Platelet dysfunction.
  - Alteration of coagulation enzyme kinetics.
  - Enhanced fibrinolysis.
  - Increased affinity of haemoglobin for O_2.
  - Increased release of red cell K^+.
  - Decreased breakdown of lactate.
- Normalize Mg^{2+} and Ca^{2+}. Infuse 4g magnesium sulphate in 100ml normal saline and give a slow push of 10% $CaCl_2$.
- Administer tranexamic acid (1g loading dose over 10min followed by an infusion of 1g over 8h) if accelerated fibrinolysis can be demonstrated by laboratory assay of D-dimers or fibrin degradation products or by rotational thromboelastometry. The CRASH-2 study supports the empirical use of tranexamic acid in massive haemorrhage associated with trauma, but there is insufficient evidence from randomized clinical trials in other areas to support or refute a clinically significant effect.
- There is no current role for routine use of recombinant factor VIIa (rVIIa). It has been advocated in major haemorrhage, but recent data review has highlighted the risk of arterial thrombotic complications. Rarely, it's use may be considered when major blood loss persists in spite of standard attempts to control bleeding in a normothermic patient who has received adequate replacement of coagulation factors with FFP, cryoprecipitate, and platelets, and correction of acidosis. Its efficacy depends on the presence of active platelets. The decision to administer recombinant factor VIIa should be made at consultant level on a named patient basis. An initial dose of 200μg/kg is recommended followed by two doses of 100μg/kg administered at 1 and 3h following the first dose if bleeding continues.
- The use of cell salvage is mandatory to minimize homologous blood use. Approximately one-third of the RBC required can be supplied by cell salvage in a major haemorrhage. Washed red cells suspended in saline are reinfused: coagulation support will be required.

- Formulaic (1:1:1 blood:FFP:platelets) blood component replacement, as used by the military, is rarely required during vascular surgery. Platelets, which are stored at room temperature, have a shelf life of 5 days. Most hospitals rely on rapid resupply of platelets from the National Blood Service, rather than holding stocks to minimize costly wastage. Anaesthetists need to be aware of local arrangements and the normal time arrangements for obtaining platelets in an emergency.

Further reading

Association of Anaesthetists of Great Britain and Ireland. Blood transfusion and the anaesthetist: management of massive haemorrhage. *Anaesthesia* 2010; **65**: 1153–61. Available at: http://www.aagbi.org/sites/default/files/massive_haemorrhage_2010_0.pdf

Chapter 6

Practical procedures, regional anaesthesia, and pain management in vascular surgery

Central venous catheterization

Introduction

Indications

Many vascular surgical patients will require a CVC. Main indications:

- CVP monitoring.
- Drug administration.
- Large volume fluid resuscitation.
- Insertion of cardiac pacing wires.
- Insertion of pulmonary artery catheters (PACs).
- Renal dialysis/haemofiltration.
- Difficulties with peripheral venous access.
- Parenteral nutrition.

Contraindications

These are relative, but include:

- Limited sites for access.
- Previous difficulties or complications.
- Severe coagulopathy.
- Local sepsis.
- Previous vascular surgery including prosthetic graft in close proximity.

Choice of device

Several types of devices are available and the choice depends on the indications, available sites, likely duration of use, size needed, and presence or absence of an indwelling CVC. Options are:

- Standard 3 or 4 lumen CVC inserted via veins in the neck, subclavian area or groin.
- Long line inserted via arm veins (good for isolated vasopressor infusions).
- Wide bore (7.5Fr) introducer sheath.
- Double lumen dialysis catheters.

Considerations

- Short length large bore peripheral cannulae for large volume pressurised fluid infusions can be considered, but there is the risk of possible extravasation and production of a compartment syndrome. The use of these should be balanced against the risks of inserting a wide-bore CVC (e.g. introducer sheath or dialysis catheter).
- Large-bore dialysis-type catheters may be preferred when massive blood losses are predicted or there may be a need for post-operative dialysis/haemofiltration.
- Some patients already have a long-term CVC in situ, e.g. dialysis-Tesio-type. Consider using these during anaesthesia rather than inserting a new catheter—the patient's central veins may already be damaged and the risks of further cannulation may be increased (see 🕮 Long-term vascular access, p. 432).
- Stock a suitable range of catheter lengths for each route of insertion. A minimal range of lengths for use in adults would be 12.5cm (right internal jugular (RIJ)), 20cm (left internal jugular (LIJ)), 20cm right

axillary/subclavian vein, 24cm for left axillary/subclavian and femoral veins.
- Vascular patients may have prosthetic material (typically radiolucent) at proposed site of cannulation, e.g. carotid/femoral/axillary patches. These are a strong relative contraindication to use of this site due to the risks of vessel damage and/or infection.
- Surgical cut-down is a feasible option in vascular theatres, either at the site of surgery or elsewhere.
- Large bore catheters and dilators need straight route of access, i.e. via the femoral or right IJ route. The left internal jugular vein is best avoided. These dilators do not pass around tight bends easily and can easily damage or perforate vessels.

Choice of site

Internal jugular vein

The right internal jugular vein (IJV) is associated with the lowest risk of complications and central misplacement. Weigh up risks in patients with carotid artery disease (presence of a patch, unstable plaque, scarring) where inadvertent arterial puncture may be dangerous, even with a 21g seeker needle.

External jugular vein

Use in acute situations when a simple large-bore cannula can be inserted under direct vision. CVCs can be sited via external jugular, but angle of entry into subclavian vein leads to problems with central passage.

Subclavian vein

Associated with a higher risk of complications, particularly pneumothorax and incorrect tip placement. However, more comfortable for the patient long-term and a cleaner site. US can be used to guide puncture of the vein using a more lateral infraclavicular axillary approach. Avoid the side of potential or actual AV fistulae with arterialized vein with risk of bleeding or thrombus.

Femoral vein

Use US to identify vessels and ensure that the vein is punctured near the inguinal ligament where the artery and common femoral vein lie side by side. The superficial femoral artery (SFA) overlaps femoral vein just below this site.

TIP Ask vascular surgeons to show you open applied anatomy at various sites of cannulation, e.g. IJ (carotid), axillary (axillo-femoral bypass), basilic vein (vein transposition, note cutaneous nerves of the forearm).

Ultrasound guidance

US is recommended to guide all central venous access from deeper vein sites. US allows:
- Direct visualization of the vessels (artery and vein) and their associated structures. Veins show respiratory fluctuation (if patent and directly connected centrally) and are easily compressible.
- Identification of thrombosis, valve, or anatomical abnormalities.
- Identification of the optimal target vessel.

- First pass cannulation in the midline of a vessel directly avoiding other vital structures.
- Visualization of guidewire and cannulae entering vein.
- Reduction of puncture related complications.

Catheter tip positioning

Correct catheter tip positioning is important for accurate monitoring and to reduce the incidence of complications, particularly thrombosis (linked to infection), embolism, arrhythmias perforation into the pericardium, pleura or mediastinum, and the risk of extravasation (Fig. 6.1).

- Optimal positioning is most reliable and quickest with an image intensifier. ECG guidance is also increasingly used.
- The CVC tip should ideally lie in the long axis of the SVC, i.e. not abutting the vein wall at acute angle.
- The CVC tip should ideally lie above the pericardial reflection (to avoid perforation and tamponade). The carina is used as an X-ray landmark to define the upper border of pericardium.
- It may be impossible to get an adequate tip position above the carina using left-sided catheters (left IJ or subclavian vein (SCV)), or the right SCV due to the angulation of the distal catheter. Aim to have the last

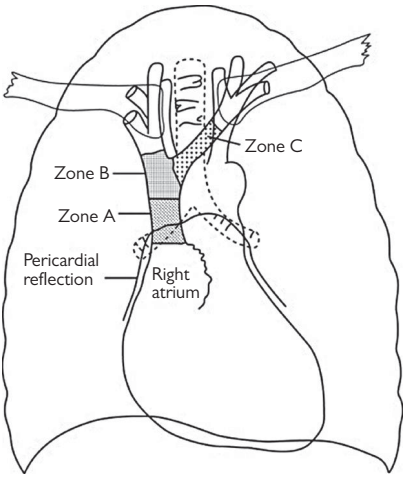

Fig. 6.1 Stylized anatomical figure dividing the great veins and upper RA into three zones (A–C), representing different areas for placement of CVC tips. (A) Junction of RA and SVC suitable for all catheters, but below pericardial reflection. (B) SVC above pericardial reflection, suitable for RIJ and sometimes right subclavian access. (C) Left inominate vein, suitable only for short-term fluids and CVP monitoring from left side access.

3–6cm of catheter tip in the long axis of the SVC (often at the junction SVC/RA or upper RA).
- CXR guidance can confirm a central position, but does not ensure optimal positioning of the tip in the SVC (it may be angled against the vein wall), particularly for left-sided catheters.
- Catheter tips move between lying and sitting/standing. Assess on inspiration/expiration with the patient flat. It will appear further centrally on supine/head down imaging vs. erect PA film + deep inspiration.

Complications

These can be considered early or late (Table 6.1). Vascular surgeons, radiologists, or anaesthetists may be called to rectify major complications, e.g. major bleeding or inadvertent arterial cannulation.

Major bleeding
- This can cause life-threatening haemothorax or haemoperitoneum.
- Minor tears of great veins are probably common during dilator/catheter insertion.
- Low venous pressure allows tamponade and haemostasis if the tear is surrounded by connective tissue/muscle.
- Major bleeding occurs when the tear connects directly to the low pressure pleural space.
- Veins at risk in chest, adjacent to pleura, include right side of SVC, azygos and hemi-azygos system, and internal mammary veins.
- Damage to the arterial tree can cause similar problems, when a needle hole alone is sufficient to cause major bleeding.
- The subclavian arteries protrude into the apex of the pleural space.
- An enlarging arterial haemotoma, from damage nearby, may burst into the pleural space. Similar mechanisms apply in the peritoneal space from damaged iliac vessels.
- Management relies on drainage, leaving catheters/dilators in place to reduce bleeding, and urgent repair or interventional radiology.

Arterial damage in the limbs or neck
- If punctured by a needle only, then remove then needle and apply firm pressure for 5–10min. (Watch the clock!)

Table 6.1 Common complications of CVC insertion

Early	Late
Arrhythmias	Infection
Vascular injury	Thrombosis
Pneumothorax	Embolization
Haemothorax	Erosion/perforation of vessels
Cardiac tamponade	Cardiac tamponade
Nerve injury	
Embolization (including guidewire)	

- Local haematoma or false aneurysm may cause skin or tissue loss, nerve damage, and upper airway compression (requiring tracheal intubation).
- Surgery may be required to decompress haematomas urgently, irrespective of how the leak is eventually closed (surgery or radiology).
- Correct any severe coagulopathy.
- Arterial dissection, thrombosis, embolus, or inadvertent arterial cannulation may cause distal ischaemic damage. This is particularly relevant to damage to, or placement in the carotid artery.
- Inadvertent arterial catheter placement may be missed by staff, who simply avoid using a 'malfunctioning' catheter.
- Removal of large bore catheters situated in carotid artery, needs careful consideration, to avoid emboli to brain, and to seal artery.
- Removing such devices and applying pressure for 20min to prevent carotid haemorrhage is very likely to produce a further risk of brain ischaemia from emboli and lack of blood flow.
- The preferred options for carotid catheterization are:
  - Either systemic heparinization and removal of the device with surgery (similar to carotid endarterectomy, venting the clot and suturing the defect).
  - Or a vascular stenting procedure performed by an interventional radiologist.

Further reading

Hughes P., Bodenham A. Ultrasonography of the femoral vessels in the groin: implications for vascular access. *Anaesthesia* 2000; **55**: 1198–202.

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Arterial access

Direct arterial access is required for many major arterial procedures, e.g. aortic, carotid surgery, or other revascularization procedures (Fig. 6.2).

Indications

- Aortic or carotid surgery.
- Haemodynamic monitoring when changes in arterial pressure are likely to be sudden or profound.
- Pulse contour analysis for cardiac output monitoring.
- Patients with severe cardiorespiratory disease.
- Need for repeated arterial blood gas (ABG) sampling.
- Anticipated need for inotropic or vasoconstrictor therapy.
- A second pressure monitoring line during surgery, e.g. stump pressure for carotid surgery or distal aortic pressure to assess spinal cord perfusion.

Arterial cannulation sites

- Typical sites for arterial cannulation include the radial, ulnar, brachial, dorsalis pedis, posterior tibial, and femoral arteries.

- Many vascular patients have poor distal arteries so vessels in the foot or ankle (dorsalis pedis, posterior tibial) are not often used.
- Others will have a poor arterial circulation, which is dependent on collaterals; a careful assessment of regional perfusion is required.
- Surgical considerations may apply, e.g. during high endovascular aortic surgery, the left subclavian artery may need to be occluded or, indeed, accessed by vascular radiology. Similar considerations apply for carotid to subclavian bypass surgery.
- The presence of, or future need for, an AV fistula requires careful consideration.
- The Allen test (compression of the radial/ulnar artery and assessing hand blood flow) is unlikely to be entirely reliable in such patients.
- Arteries may be calcified, making cannulation difficult. This may make vessel closure with pressure impossible after cannula removal. Other vessels may have aneurysmal changes or dissection.
- Many vascular patients will have prosthetic material in the groins or elsewhere. Prosthetic grafts are a relative contraindication to both venous and arterial cannulation at such sites because of the potential for graft damage or thrombosis, and the increased risk of infection.

Tips

- Multiple attempts at cannulating a difficult distal artery should be avoided.
- Consider asking a colleague to assist or assess the finite risk of cannulating a more proximal end artery.
- Procedures are technically easiest in awake patients before general anaesthesia, as the pulse is usually more palpable.
- In some cases, multiple attempts can lead to significant pain and distress. In these circumstances, cannulation after sedation or GA may be preferable. Ephedrine 3mg increments IV may be useful to augment arterial pressure if required.
- Multiple attempts at peripheral arterial cannulation in patients who are hypotensive or hypovolaemic are futile. The femoral and brachial arteries are useful during resuscitation of shocked patients.
- If difficulties ensue, consider a vascular surgical cut-down. A catheter can be passed higher up beyond the operative site (e.g. femoral) or as a separate cut-down procedure, e.g. radial.
- Care should be taken with the length and size of catheters, particularly for deeper situated vessels (femoral or brachial arteries) where catheters can be dislodged with movement.
- The success rate with guidewire-based cannulae is likely to be higher than for catheter-over-needle devices in the patient with peripheral arterial disease.
- *Catheter-over-needle technique:* if you have hit the artery, but failed to pass the catheter, advance the device inwards, remove needle, attach a syringe and slowly withdraw cannula with gentle aspiration. When blood can be easily aspirated attempt to re-advance with a twisting action. Many cannulations can be salvaged with practice in this way.

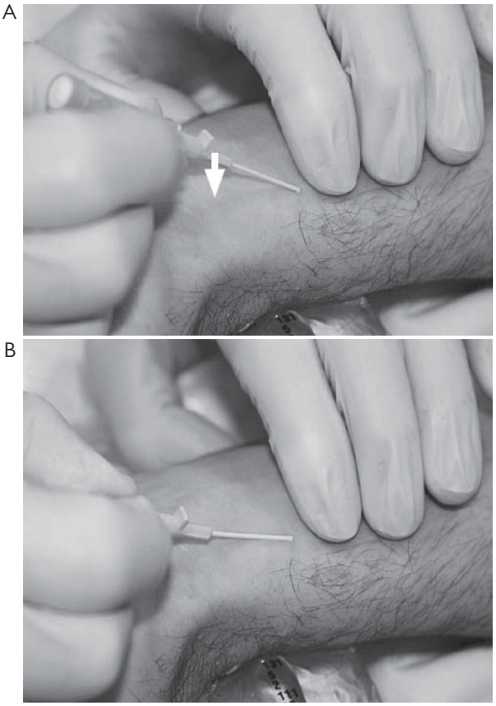

Fig. 6.2 Intra-arterial catheterisation. After observing blood at the cannula hub, the catheter should be angulated (arrow) to facilitate advancement within the arterial lumen.

Reproduced from Allman et al., Emergencies in Anaesthesia, 2009, Figure 14.22, p. 480, with permission from Oxford University Press.

- If using sutures to fix the cannula, do not place stitches too deeply. It is possible to inadvertently damage peripheral arteries.
- Stenoses or collateral flow may affect the validity of arterial pressure readings may be altered and the difference between central aortic and peripheral pressures. MAP may be a more realistic figure than the systolic or diastolic. It may be necessary to measure non-invasive pressures in different limbs to choose the optimal cannulation site.

Ultrasound guidance

- US is useful to assess the extent of atheroma, narrowing of vessels, potential dissection, presence of thrombus, pulsatility, and flow in the vessel. It demonstrates that arteries are relatively incompressible and pulsatile even in the shocked patient.

Table 6.2 Complications of arterial cannulation

Immediate	Early	Late
Bleeding	Arterial embolism	Infection
Haematoma	Vasospasm	Ulceration
Arterial damage	Nerve injury	Thrombosis Arteriovenous fistulae

- US is very useful when using deeper sites. When cannulating the femoral artery it allows visualization and cannulation of the common femoral as opposed to the lower superficial femoral artery. Look for and avoid vena commitantes and nerves.
- US facilitates allows cannulation at other deeper sites, e.g. the impalpable radial and ulnar arteries in the mid-forearm, so avoiding the flexures. Ideally, real time needle guidance should be used.

Cannula removal

After removal of the cannula, press firmly on the site for at least 5min. Occasionally, persistent bleeding may require a fine suture to close the skin wound and stabilize clot. Then apply further pressure.

Complications

- Vascular compromise may occur at any stage.
- Inadvertent injection of drugs into an arterial catheter is an important avoidable cause of morbidity. All cannulae and lines must be clearly labelled.
- The risks of infection increase with duration of catheterization. Arterial catheters have been implicated with MRSA and other septicaemias.

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Cardiac output monitoring

Cardiac output (CO) monitoring is potentially useful in major vascular surgery because:

- The risks of cardiovascular morbidity and mortality are high.
- The surgery is often invasive.
- The incidence of co-existing cardiovascular disease is high.

CO monitoring allows:

- Guided IV fluid therapy.
- Guided inotrope and vasoconstrictor drug therapy.

- Assessment of the effects of perioperative cardiovascular responses to major surgical insults, such as aortic cross-clamping or unclamping.
- Guided post-operative monitoring and management.

In recent years numerous CO monitoring devices have been developed and introduced. All use different techniques to measure or calculate cardiac output so have different sources of error. Some techniques are not well validated, practical, or commonly available, and are ∴ currently not used in vascular anaesthesia. These include transthoracic Doppler, partial CO_2 rebreathing, thoracic bio-impedance, and transcutaneous indocyanin green concentration measurements; these are not considered further.

Pulmonary artery catheter

Technique

- The pulmonary artery catheter (PAC) is a multi-lumen balloon tipped catheter inserted through a large bore sheath.
- Catheter is floated through CVC and right heart to sit in one of the proximal branches of the pulmonary artery.
- The PAC allows direct measurement of:
  - Mixed venous oxygen saturation (SvO_2).
  - Pulmonary artery pressure.
  - Right heart chamber pressure.
  - Pulmonary artery occlusion pressure (reflects left atrial pressure).
- It allows indirect measurement or calculation of:
  - Cardiac output.
  - Stroke volume.
  - Systemic and pulmonary vascular resistances.
  - Oxygen delivery and oxygen consumption.
- CO is calculated by intermittent thermodilution using older catheters or by repeated CO calculation via thermal filaments and distal thermistors in newer, 'continuous' catheters.
- PAC monitoring is best used in patients undergoing major vascular surgery who have HF, poor cardio-respiratory reserve evidenced on CPX testing or contraindications to other CO monitoring techniques.

Advantages

- The PAC has been used for >40yrs and is still considered the clinical 'gold standard' for CO measurement.
- It allows a great deal of information to be measured or calculated (CO, right heart pressures, SvO_2).
- It allows blood sampling and inotrope administration.
- Many hospitals have the equipment already.

Disadvantages

- Large scale clinical trials have shown no reduction in mortality in patients in whom it has been used.
- The PAC requires considerable operator experience for insertion and interpretation.
- Interpretation is difficult in certain situations (especially mitral and tricuspid valve disease).
- Overall use of the PAC has dwindled; trainees and nursing staff now have less experience of its use.

- It cannot measure 'beat-to-beat' CO continuously.
- It is invasive, with serious complications associated (infection, arrhythmias, knotting of catheter, thromboembolism, damage to heart, valves, pulmonary artery).

Oesophageal Doppler monitor

Technique

- Oesophageal Doppler monitor (ODM) comprises a probe containing angled piezoelectric crystal that emits US waves at a fixed frequency.
- Probe is inserted orally or nasally into oesophagus and positioned according to waveform and probe depth to pick up descending aortic flow.
- Frequency shift of sound reflected off aortic blood is proportional to velocity of blood.
- Velocity time waveform, in combination with entered patient data (height, weight, age) is used to calculate several variables according to an algorithm.
- Variables include:
  - Stroke volume.
  - Peak velocity.
  - Mean acceleration (related to left ventricular contractility).
  - Flow time corrected for heart rate (FTc) (relates to the circulating volume and systemic vascular resistance).

Advantages

- It is relatively non-invasive.
- It is relatively easy to insert.
- ODM does not require invasive vascular catheters (arterial or CVP).
- Several studies suggest reductions in post-operative complications and hospital length of stay when it is used to guide fluid therapy, mostly in trauma and general surgical patients.
- Demonstrates changes in CO over time with reasonable accuracy.

Disadvantages

- It can only be used in sedated or anaesthetized patients.
- There are potential inaccuracies from assumptions/algorithms (e.g. aortic cross-sectional area, blood leaving aorta before probe measurement).
- Use is limited during aortic surgery because aortic cross-clamping directly affects aortic cross-sectional area.
- It requires some operator skill.
- It requires specialist equipment.
- It is less able to give accurate absolute values compared with a PAC, ∴ is possibly unsuitable for high-risk patients.

Pulse power analysis: LiDCO

Technique

- The LiDCO system comprises a lithium dilution CO measurement to calibrate an arterial pressure waveform analysis (PulseCO).

- The lithium dilution calibration involves injecting a small dose of IV lithium chloride. This is measured by sampling arterial blood through a lithium electrode attached to the arterial catheter. CO is calculated from the lithium concentration/time curve.
- The PulsoCO system uses an algorithm to convert the whole pulse waveform from the arterial line trace into a standardized volume-time waveform and calculates the pulse power to derive a nominal stroke volume. This can then be converted to an actual stroke volume using the information from the lithium dilution.

Advantages

- Does not require a CVP line or a specialized arterial line, ∴ less invasive than other methods.
- It uses whole waveform in the algorithm not just systolic area, ∴ is theoretically less affected by damping issues than other pulse waveform analysis techniques.
- Both initial calibration and subsequent pulse power analysis have reasonable agreement with PA catheter.
- It allows display of additional information, such as pulse pressure variation to guide fluid management.
- Reasonably low learning curve and the equipment includes step-by-step guide for lithium dilution.

Disadvantages

- Lithium dilution technique is influenced by haematocrit, electrolytes, and certain medications (lithium, neuromuscular blockers, such as atracurium).
- Cannot be used in patients with weight <40kg, or women in the first trimester of pregnancy.
- Requires specialist equipment for lithium sensor and blood aspirator, monitor.
- Prone to inaccuracies if arterial waveform is suboptimal.
- Recalibration is required every 4–12h based upon instability of the patient (causing changes in arterial compliance).
- Use is limited in certain clinical situations, e.g. arrhythmias, balloon pumps, aortic regurgitation.

Pulse contour analysis: PiCCO

Technique

- Pulse contour cardiac output measurement use a thermistor tipped catheter in a proximal artery to analyse area of systolic part of arterial pressure waveform. Algorithm then determines stroke volume, systemic vascular resistance, and CO, which is displayed continuously.
- It is calibrated against transpulmonary thermodilution in a similar manner to the PAC, performed using a CVC.
- Pulse contour devices can also calculate stroke volume variability (SVV), and indicator of a patient's fluid responsiveness.

Advantages

- Less invasive than a PA catheter.
- Reasonable agreement with PA catheter measurements.

- Allows calculation and display of additional information, e.g. SVV, extravascular lung water, and global end-diastolic volume.
- Reasonable learning curve. Insertion technique similar to other Seldinger-type arterial catheters.

Disadvantages

- More invasive than LiDCO or ODM because it needs a specific proximal arterial catheter in a central artery (femoral or axillary) and a CVC.
- Prone to inaccuracies if arterial waveform is not optimal (over- or under-damped).
- Use is limited in certain clinical situations, e.g. arrhythmias, balloon pumps, aortic regurgitation. The presence of AAA affects accuracy of certain figures, when a femoral arterial line is used.
- Requires specialist equipment (specific arterial lines, monitoring equipment).
- Recalibration is required at least every 8h.

Pulse contour analysis-FloTrac/Vigileo

Technique

- Analyses the pulse waveform from a standard arterial catheter. The FloTrac algorithm calculates vascular compliance from the arterial waveform, in combination with inputted patient data (height, weight, age, sex). The vascular compliance and arterial pulsatility are used to calculate SV and CO.
- Does not require external calibration as the algorithm continuously corrects for vascular tone.
- Displays continuous values on the Vigileo monitor.

Advantages

- Minimally invasive requiring only a standard arterial line.
- No requirements for initial or repeat calibration.
- Allows calculation and display of additional information, e.g. SVV.
- Minimal learning curve, very easy to use.

Disadvantages

- Comparison with other methods of CO monitoring show conflicting results ∴ its accuracy is not yet confirmed.
- Prone to inaccuracies if arterial waveform is not optimal (over- or under-damped).
- Use is limited in certain clinical situations, e.g. arrhythmias, balloon pumps, aortic regurgitation. Presence of AAA affects accuracy of certain figures, when a femoral arterial line is used.
- Specialist equipment required (FloTrac sensors, Vigileo monitor).

Further reading

Hofer CK, Ganter MT, Zollinger A. What technique should I use to measure cardiac output? *Curr Opin Crit Care* 2007; **13**: 308–17.

Compton F, Schäfer J. Noninvasive cardiac output determination: Broadening the applicability of hemodynamic monitoring. *Semin Cardiothorac Vasc Anesth* 2009; **13**(1): 44–55.

Cerebrospinal fluid drainage

There are many strategies to preserve spinal cord blood flow during aortic surgery and thereby minimize the incidence of ischaemic cord damage and paraplegia. The best supporting evidence from both animal and human research is for CSF drainage.

Rationale for CSF drainage

- Spinal cord perfusion pressure (SCPP) is estimated as: SCPP = MAP—[CSF pressure or CVP] (whichever is the greater).
- In theory, spinal cord ischaemia is unlikely if SCPP is normal.
- Spinal cord ischaemia is thought to cause spinal cord oedema.
- The spinal cord and CSF are held within a relatively rigid bony canal—the spinal column—so an increase in spinal cord volume readily increases CSF pressure.
- Increased CSF pressure decreases SCPP, which will worsen ischaemia and create a 'vicious cycle' that may end in spinal cord injury.
- Hence, CSF drainage should maintain or increase SCPP and help prevent ischaemia.
- This is supported by studies in animals where CSF drainage reduced the incidence of paraplegia after high aortic clamping, and case reports in humans in which the sudden occurrence of early post-operative paraplegia has been successfully treated with CSF drainage.
- Clinical studies have shown that CSF drainage, when combined with other measures, minimizes the incidence of spinal cord injury.

Patient selection

- CSF drainage should be considered in patients at high risk of spinal cord injury (📖 Open aortic aneurysm repair, p. 350) and after discussion with surgeons.
- Patients at high risk include those undergoing open or endovascular procedures for Crawford Types I, 2, and 3 TAAAs.

CSF drain insertion

- CSF drain insertion should be performed with the same precautions and meticulous technique as for epidural or subarachnoid catheter insertion.
- Full aseptic technique is mandatory, and the patient should be awake or only lightly sedated.
- The same cautions regarding neuraxial procedures in patients with abnormalities of coagulation or receiving anticoagulant drugs should be followed (📖 The anti-coagulated patient, p. 215).
- A standard 18G epidural catheter is inserted through the dura into the subarachnoid space via a 16G Tuohy needle.
- Insertion at a lumbar interspace is favoured in order to decrease the chance of direct needle damage to the cord, which usually ends at the level of the first lumbar vertebra, continuing as the cauda equina.
- 4–6cm of catheter should be threaded into the subarachnoid space.

- There is no evidence that leaving a longer length of catheter in the subarachnoid space decreases the incidence of dislodgement or improves efficacy.

Management of CSF drains

- Purpose-made CSF drainage systems should be used, setting a drainage level at about 10–14cm above the heart, to produce a theoretical CSF pressure of about 10mmHg.
- Large drainage volumes should be avoided because of the risk of intracranial haemorrhage (see 📖 Complications of CSF drains, p. 265)—consider clamping the drain if drainage exceeds 10–15mL/h.
- If there is no CSF drainage, the catheter should be aspirated gently to confirm catheter patency. This can be repeated hourly.
- Drains are usually left in for 48h after surgery, or longer if there are concerns about spinal cord function.
- The coagulation profile should be normal at the time of drain removal.
- If there are any clinical signs suggesting intracranial or spinal cord pathology, an urgent MRI scan should be performed to exclude spinal canal or intracranial haematoma.

Complications of CSF drains

- These include:
  - All the complications of lumbar puncture with a large-bore needle, including nerve damage and spinal haematoma.
  - Excessive CSF leakage, which can lead to intracranial hypotension and subsequent haemorrhage.
  - Intracranial haemorrhage, usually subdural. This has an incidence of about 5% and is symptomatic in 1%. It carries a high mortality.
- Factors associated with the occurrence of intracranial haemorrhage:
  - High drainage volume.
  - High central CVP.
  - Cerebral atrophy, e.g. in the elderly.
  - Arteriovenous malformations.
  - Cerebral aneurysms.
  - Previous subdural haematoma.
  - Infection and meningitis.
  - Persistent CSF leak.
- The overall mortality of CSF drainage is up to 1%.

Further reading

McCullough JL, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: influence of cerebrospinal fluid drainage: experimental and early clinical results. *J Vasc Surg* 1988; 7: 153–60.

Lung isolation techniques and one lung ventilation

Indications

- Lung isolation techniques were originally developed to allow differential spirometry, but are now used regularly to facilitate surgery on intrathoracic structures.
- Either lung can be ventilated, and the other lung collapsed. This enables surgery either to the lung itself, or to intrathoracic structures.
- The main indications for lung isolation in vascular surgery are surgical repair of a thoraco-abdominal aortic aneurysm, for which the right lung is ventilated, whilst the left is collapsed giving surgical access to the descending aorta.
- Thoracoscopic sympathectomy (either lung is collapsed depending on the site of surgery).

Techniques for lung isolation

- The simplest way to ventilate a single lung is to advance an uncut single lumen endotracheal tube into the right or left main bronchus and inflate the cuff.
- However, this does not allow any access to other lung during procedure without re-positioning the tube, and is ∴ of limited value.
- A double-lumen tube or an endobronchial blocker is used.

Double lumen tubes

The most commonly used method of lung isolation is the endobronchial DLT. Earlier tubes were re-usable, made of red rubber, with various designs available. Today, they have largely been replaced by plastic tubes based on the Robertshaw DLT, although there is also a disposable rubber Robertshaw tube available.

Tube design

- Double lumen tubes consist of a tracheal lumen and a longer bronchial lumen fused together, each with their own cuff and pilot tube (Fig. 6.3).
- The bronchial cuff and pilot tube are generally colour-coded blue to identify them once the tube is positioned.
- Rubber tubes are more rigid and are manufactured with two curves, one at the level of the pharynx and the other, at 90° to the first, at the carina.
- Plastic tubes are more flexible and a stylet must be inserted into the tube for intubation.
- Right- and left-sided tubes are available, with the main difference being that right sided tubes have the addition of a 'Murphy's eye' on the bronchial tube.
- The Murphy's eye allows ventilation of the right upper lobe bronchus that arises shortly after the carina, which would otherwise be obstructed by the bronchial tube (Fig. 6.4).
- However, a right-sided DLT can be difficult to position correctly and many anaesthetists insert a left-sided tube even for left-sided surgery.

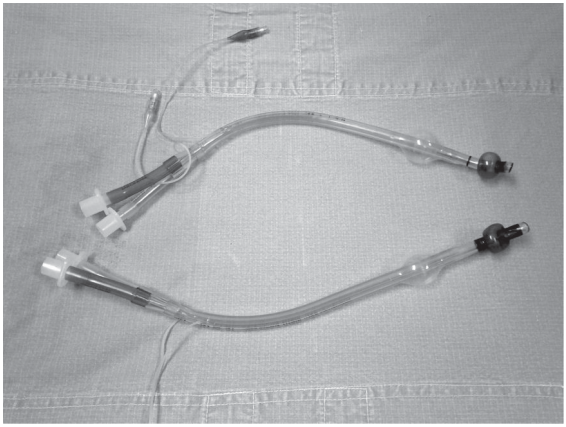

Fig. 6.3 Left (above) and right double lumen endobronchial tubes. Note the modified bronchial cuff and "Murphy's eye" on the right sided tube.

- It is important to communicate with the surgeon regarding tube placement, especially during lung resections to prevent inadvertent stapling of a tube inside a bronchus.

Correct size of DLT

- It is important to select the correct size of DLT for each patient to secure effective isolation and avoid airway trauma.
  - An oversized tube, which isolates the lung without any air in the cuff may cause mucosal compression and ischaemia.
  - An undersized tube may migrate too far into the bronchus, obstructing lobar bronchi, or may need excessive inflation of the cuff to achieve a seal, potentially causing mucosal damage.
- Plastic tubes are available in various sizes: 35Fr, 37Fr, 39Fr, and 41Fr (either right- or left-sided tubes); a left 32Fr is available for small adults.
- Sizes 35 or 37 are usually suitable for women.
- Sizes 37 or 39 are usually suitable for men.
- Disposable rubber tubes are also available in extra small, small, medium, and large for both right- and left-sided tubes.
- Various techniques for predicting the correct size of tube have been suggested. Many rely on the radiological measurement of tracheal and/or main bronchus diameter (see Table 6.3). In very short individuals this may lead to an oversized DLT being used.
- There is a relationship between patient height and airway diameter, and many anaesthetists base their choice of tube on height alone; however, this is also not entirely reliable.
- Common practice has been to use the largest size tube that allows a small leak with the cuff deflated.

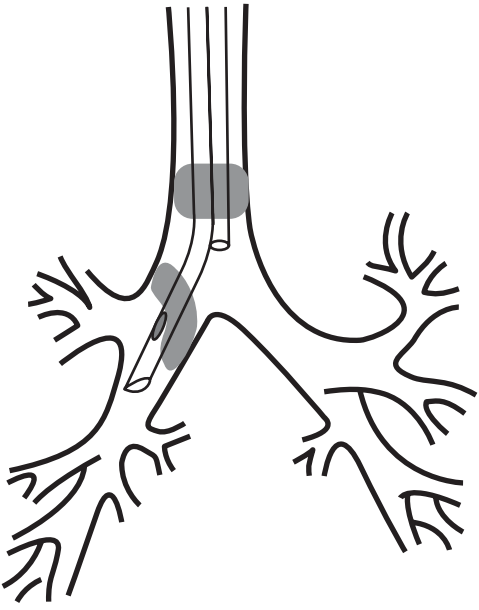

Fig. 6.4 Diagram of right-sided double lumen tube in position. The Murphy's eye and modified bronchial cuff allow ventilation of the proximally positioned upper lobe bronchus.

Table 6.3 Guidelines for left bronchocath double-lumen tubes

Measured tracheal width (mm)	Predicted left bronchus width (mm)	Recommended DLT size (F)
≥18	≥12.2	41
≥16	≥10.9	39
≥15	≥10.2	37
≥14	≥9.5	35
≥12.5	≥8.5	32
≥11	≥7.5	28

Adapted from: Brosdky *et al Anesth Analg* 1999; **88**(2): 466.

- However, the routine availability of fibre optic bronchoscopes to check tube position may allow more accurate sizing.

Tube positioning and checking

- Tracheal intubation is initially performed as for a standard endotracheal tube.
- Once the bronchial section of the tube is in the upper trachea, the stylet is removed, the tube rotated 90° left or right, depending on the tube in use, and advanced until slight resistance is met or the bite guard is at the level of the teeth.
- The tracheal cuff is inflated until there is no leak and air entry checked in both lungs.
- The airflow to the tracheal lumen is then clamped at the connector, and the lumen opened to air distal to the clamp (Fig. 6.5).
- Gentle inflation of the bronchial cuff should stop any leak from the open tracheal lumen, whilst the bronchial side is ventilated. If there is no leak without any air in the bronchial cuff the tube is either too large or inserted too far.
- Once the cuff is inflated it is important to auscultate the upper lobe on the bronchial side to ensure the cuff has not occluded the upper lobe bronchus. This is important because both right- and left-sided DLTs, which are too small can be advanced too far.
- Clinical methods of checking tube position include:
  - Auscultation.
  - Feeling/listening for a leak on the open lumen.
  - Noting a reduction in tidal volume when one lumen is clamped (if pressure controlled ventilation is in use).

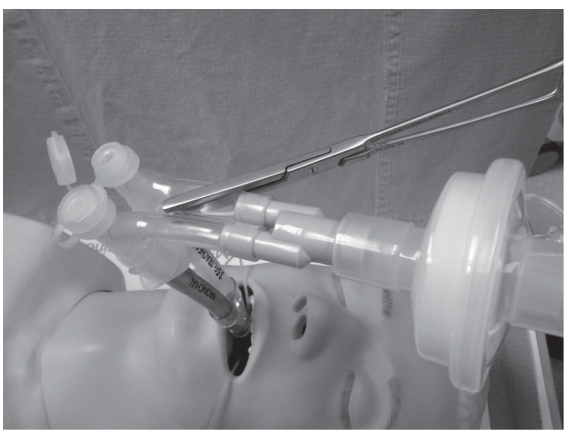

Fig. 6.5 A left-sided double lumen tube in position in a manikin. The proximal lumen on the right side is clamped to prevent ventilation of the right lung, and the lumen opened to air distal to the clamp to facilitate right lung collapse.

- A fibre optic bronchoscope is then used to check placement, especially in right-sided tubes to confirm alignment of the Murphy's eye with the upper lobe bronchus.
- Correct position of tube should be re-checked once patient has been positioned for surgery. Tube movement can occur and once surgery has started it is much more difficult to address any problems.

Bronchial blockers

- Bronchial blockers consist of balloon tipped catheters that can be advanced via a single lumen tracheal tube into the main or more distal bronchi to prevent ventilation of the relevant lung or lobe.
- They have become more popular over recent years with the development of improved catheters and greater access to fibre optic bronchoscopy for accurate placement.

Insertion

- Arndt type bronchial blocker catheters are flexible with a guidewire loop at the end allowing them to be guided into place under direct vision over a bronchoscope. Once in position the distal balloon is inflated, and isolation checked by auscultation.
- More rigid catheters are also available. These could theoretically be placed blindly, but it is still advisable to check the final position with a bronchoscope.

Limitations of bronchial blockers

- The main problem is the very narrow central lumen. It can take some time for the lung to collapse, delaying surgery, and increasing the risk of retraction trauma to the lung.
- There is also no access to collapsed lung whilst balloon is inflated.
- Techniques to improve speed of lung collapse include directly applied suction, and ventilation with a high concentration of inspired oxygen prior to balloon inflation. Oxygen is rapidly absorbed and as there is little nitrogen present to splint the alveoli, the lung collapses once chest is opened.

Physiology of one lung ventilation

- In the lateral position, perfusion of dependent lung is greater due to effects of gravity. The dependent lung is also more compliant and ventilation is greater than in the non-dependent lung. Overall, this results in reasonable matching of ventilation and perfusion (V/Q).
- Under GA, the loss of muscle tone causes a reduction in volume of the lower hemithorax, and therefore the FRC of the dependent lung decreases.
- This effect is further magnified by flexing the patient to improve surgical access.
- The net result is that the dependent lung moves down to a less favourable position on the lung compliance curve, whereas the non-dependent lung also moves down from the top of the curve to a more favourable position.
- Ventilation shifts from the more perfused, dependent lung to the less perfused, non-dependent lung, and V/Q mismatch increases.

- On clamping the non-dependent lung, ventilation is diverted back to the dependent lung. However, the upper lung is still perfused and this produces a significant shunt. This shunt is reduced in the lateral position compared to supine position (because of the effects of gravity) and is further reduced once chest has been opened and lung collapsed.
- Further factor in returning V/Q matching towards normal is development of hypoxic pulmonary vascoconstriction, reducing shunt through unventilated lung by approximately 40% and so improving perfusion of the ventilated lung. Factors that may interfere with HPV include:
  - Volatile anaesthetic agents.
  - Vasoactive drugs.
  - Disturbances of acid base balance (respiratory or metabolic).
  - Hypothermia.

Intra-operative management of OLV

- It can be difficult to ventilate the dependent lung during one-lung ventilation (OLV), especially in patients with pre-existing lung disease.
- It is not realistic to maintaining the same tidal volume (TV) as during two-lung ventilation, and attempts to do so will lead to excessive airway pressures (Paw).
- Excessive Paw can increase vascular resistance and divert blood away from the ventilated lung. This increases shunt and can lead to barotrauma of the ventilated lung.
- Excessive use of PEEP has the same effects, but low levels of PEEP may be useful to minimize atelectasis.
- Increasing respiratory rate (RR) to maintain minute ventilation helps to control PCO_2, but at high respiratory rates intrinsic PEEP can be a problem.
- The aim is to achieve a balance between TV, RR, and Paw that maintains SpO_2 at least above 90% (preferably ≥94%), whilst tolerating small increases in PCO_2.

Initiation of OLV

Before commencing OLV

- Increase FiO_2 to >0.6.
- Set PEEP at 5cmH_2O.
- Set ventilator (PC or VC) to achieve TV 6–8mL/kg.
- Increase RR slightly to maintain minute ventilation (MV).

Hypoxia during OLV

Certain situations may predict the development of hypoxia during OLV.

- Right-sided surgery.
- Poor oxygenation prior to lung isolation.
- High A-a gradient for CO_2
- Normal or good pulmonary function before surgery.

This last factor is thought to be due to the lack of auto PEEP resulting in secondary de-recruitment.

Management of hypoxia during OLV

First ensure that DLT is correctly positioned, and that there is no non-respiratory cause for a worsening of V/Q mismatch, such as hypotension. If hypoxia continues then the following is a logical sequence of actions:

- Increase FiO_2.
- Ensure the patient is fully paralysed.
- Modify ventilator settings to optimize ventilation, e.g. change I:E ratio, keeping peak Paw <30cmH_2O.
- Apply 100% O_2 via a CPAP circuit to the non-ventilated lung.
- Perform a recruitment manouevre (📖 Box 2.1, p. 56) on the ventilated lung.

If these steps are not successful then it will be necessary to go back to two lung ventilation and re-try, once normal values of SpO_2 have been achieved. If hypoxia occurs again it may be possible to continue surgery with intermittent OLV.

Further reading

Slinger P. *Principles and Practice of Anesthesia for Thoracic Surgery*. Berlin: Springer 2011.

Intra-abdominal pressure monitoring

The term abdominal compartment syndrome (ACS) and the technique for measuring intra-abdominal pressure (IAP) via the bladder were first proposed in relation to ruptured AAA. International definitions and recommendations are now established.

Definition and risk factors

Intra-abdominal hypertension (IAH) is defined as a sustained increase in IAP >11mmHg. Risk factors include:

- Reduced abdominal wall compliance.
- Increased bowel luminal contents.
- Increased abdominal contents.
- Increased capillary leak or positive fluid balance.

Several of these (abdominal surgery, ileus, haemoperitoneum, massive transfusion, or fluid resuscitation, coagulopathy) are likely to be present in patients after open aortic surgery, especially emergency AAA rupture.

Measurement

- IAP should be measured in all patients with risk factors for IAH (e.g. abdominal vascular surgery) on admission to ICU, and if any clinical deterioration occurs during ICU stay.
- If IAP is consistently <12mmHg, measurement can be discontinued.
- If IAP is sustained >11mmHg, measurements should be performed 4-hourly.
- IAP >20mmHg with signs of organ dysfunction, defines ACS. This needs active treatment (see 📖 Critical care management, p. 504).

Measurement technique

Several techniques have been proposed for measuring IAP including intravesical, gastric, rectal, vascular, and uterine routes; each has specific advantages and disadvantages. The intravesical technique is the best compromise between ease of use, cost and reproducibility, and represents direct intra-abdominal pressure with reasonable accuracy.

Technique

- The urinary bladder catheter is allowed to drain continuously until measurement, to ensure the bladder is empty.
- Using a sterile bladder syringe, 25mL of sterile saline is injected into the catheter, either after disconnecting the catheter tubing or via a three-way stopcock inserted at the proximal lumen. The latter is recommended as it does not interrupt the sterile circuit
- The catheter is clamped distally to the syringe and a pressure transducer is attached either to the stopcock or via a fluid filled 16G needle inserted into the culture aspiration port. The transducer pressure is calibrated to zero at atmospheric pressure.
- The system should be allowed to stabilize for a couple of minutes and a brief flush of the circuit will ensure any air bubbles are removed.
- Before measurement, the transducer circuit can be verified by tapping on the abdomen and observing transient pressure rises on the monitor.
- Measurements should be performed in an absolutely supine position, with the transducer at the mid-axillary line at the level of the thigh.
- Coughing or straining by the patient interferes with measurement accuracy. Adequate sedation is needed, including neuromuscular blockade in some cases.
- After measurement, the volume of added fluid should be removed or factored into urine output values.
- The fluid volume used for measurement should be constant each time and should be no more than 50mL. Higher volumes may reduce accuracy in sick patients with a relatively non-compliant bladder.
- If multiple readings are required over several days a modified technique, using a three stopcock ramp, a bag of saline and a 60mL bladder syringe allows repeated accurate readings without disconnection of the sterile system.
- In patients without a bladder or with bladder damage from trauma, an intra-gastric device can be used.

Treatment

Treatment of IAH or ACS is targeted at the individual risk factors and maintenance of abdominal perfusion. Abdominal perfusion pressure (APP) is defined as (MAP–IAP). The aim is to keep APP>60mmHg

- Improve abdominal wall compliance with sedation, neuromuscular blockade, and body positioning.
- Reduce bowel luminal volume by NGT aspiration and enemas.
- Percutaneous decompression of abdominal fluid collections.
- Correct positive fluid balance with diuretics, fluid restriction, and dialysis if necessary.

- Increase MAP using vasopressors if necessary to keep APP >60mmHg.
- If these measures fail, consider surgical abdominal decompression.

Further reading:

Cheatham ML, Malbrain ML, Kirkpatrick A. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome: II. Recommendations. *Intens Care Med* 2007; **33**: 951–62.

Iberti TJ, Kelly KM, Gentill DR, *et al*. A simple technique to accurately determine intra-abdominal pressure. *Crit Care Med* 1987; **15**: 1140–2.

Malbrain ML, Cheatham ML, Kirkpatrick A, *et al*. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome: I. Definitions. *Intens Care Med* 2006; **32**: 1722–32.

Malbrain ML. Different techniques to measure intra-abdominal pressure (IAP): time for a critical re-appraisal. *Intens Care Med* 2004; 30: 357–71.

Regional anaesthesia in vascular surgery

Many vascular surgical patients are elderly, with IHD, renal disease, and diabetes, and are at increased risk for perioperative complications. Many would benefit from RA blocks. Central neuraxial techniques (spinal or epidural) are suitable for lower limb vascular surgery; epidural techniques are highly suited for post-operative analgesia after abdominal or thoracic surgery. All major peripheral nerves of the upper and lower limb may be blocked with LA; either a single injection or continuous catheter technique may be used.

Benefits of regional anaesthesia

- *Pain relief:* this is the most consistent and apparent benefit from regional anaesthetic techniques in almost all surgical specialties. Many studies of both central and peripheral techniques have shown improved analgesia, either in terms of reduced pain scores, reduced opioid consumption, or other surrogate markers of pain.
- *Pre-emptive analgesia:* animal studies have shown that RA can reduce spinal cord excitation and, thus, reduce analgesia requirements. However, establishing RA before surgical incision has not conclusively shown a reduction in the occurrence of phantom limb pain after amputation or chronic pain syndromes in vascular surgery patients.
- *Intraoperative analgesia:* RA anaesthesia reduces the requirements for intraoperative opioids or other analgesics, but few other specific benefits have been found. In high-risk vascular patients undergoing carotid endarterectomy evidence has failed to show loco-regional over GA.
- *Surgical stress response:* neuraxial anaesthesia established before surgery minimizes the extent and duration of the surgical stress response during major abdominal surgery, including the reduction in serum cortisol and catecholamine levels. There is also less disturbance of blood glucose concentrations in diabetic patients. These effects are best achieved with epidural anaesthesia to above the level of T10.

- *Blood loss:* RA has been associated with reduced blood loss in a variety of lower limb surgical techniques.
- *Post-operative nausea and vomiting:* RA allows a reduction in both volatile anaesthetic and opioid requirements; both of these contribute significantly to post-operative nausea and vomiting. This benefit is particularly useful for carotid surgery, where coughing and vomiting increases the risk of bleeding and haematoma formation.
- *Reduction of opioid use:* minimizing opioid consumption reduces incidence of opioid-related adverse effects, such as pruritis, reduced gut motility, respiratory depression, confusion, drowsiness, and urinary retention.
- *Respiratory function:* neuraxial RA techniques preserve pulmonary function, functional residual capacity, oxygenation, and reduce infective respiratory complications for a variety of procedures, mostly thoracotomy and major upper gastrointestinal surgery. Post-operative respiratory morbidity is reduced.
- *Cardiac mortality/morbidity:* thoracic epidural analgesia (T1–T4) reduces cardiac sympathetic activity and reduces myocardial oxygen demand. It also improves oxygen supply, by increasing the diameter of stenotic epicardial coronary arteries in patients with coronary artery disease, and improves ischaemia-induced left ventricular dysfunction Thoracic epidural analgesia reduces mortality and the incidence of myocardial infarction after open AAA repair compared to lumbar epidural analgesia.
- *Early mobilization/physiotherapy:* good quality post-operative analgesia allows early mobilization and physiotherapy.
- *Bowel function:* epidural analgesia above the level of T10 increases gut motility and reduces post-operative ileus.
- *Cognitive function:* this may return to normal faster with the use of regional techniques. The reduction in opioid consumption is a significant factor in the elderly.
- *Avoidance of GA:* RA may remove the need for GA completely, e.g. neuraxial blocks for EVAR or lower limb revascularization. This avoids the 'minor' complications of GA (headache, sore throat, fatigue).
- *Time to discharge:* the combination of the benefits listed above can allow earlier discharge from hospital, best achieved where a multifaceted approach is adopted. This includes optimal care using regional techniques, which requires a consistent approach to the use of RA and training of surgical and nursing staff. Shorter ICU stays for high-risk patients have been reported.
- *Deep venous thrombosis:* a hypercoaguable state is part of the normal stress response to surgery. RA has been associated with a reduction in deep venous thrombosis following lower limb surgery.
- *Graft patency:* some studies in 1990s suggested that the incidence of early graft thrombosis after lower limb revascularization may be reduced with the use of RA. Subsequent studies have not confirmed this.

Adverse effects of regional anaesthesia

RA is commonly used, and direct complications are rare. This makes precise prediction of risks difficult, but nevertheless a careful assessment of the potential risks and benefits of RA should be performed and discussed with the patient. The generic complications of RA include: infection, vascular puncture, haematoma, LA toxicity, and nerve injury.

Nerve injury

- Nerve damage presents as sensory paraesthesia, motor block, or a mixture of both.
- Intra-fascicular injection is associated with neurological damage.
- Signs include pain, paraesthesia, resistance to injection, and intra-neural swelling on ultrasound using only 1–2mL.

Local anaesthetic toxicity

- Early signs of local anaesthetic toxicity are: lingual or circumoral tingling, anxiety, light headedness, tinnitus, drowsiness.
- In severe cases there may be:
  - Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions.
  - *Cardiovascular collapse*—sinus bradycardia, conduction blocks, asystole, and ventricular tachyarrhythmias
- The most important factor in the prevention of toxicity is to avoid intravascular injection.
- Repeated aspiration tests should be performed during injection of LA.
- Maximum safe doses of LA drugs should be followed, though these should account for the site of injection, the vascularity of the injection site, the general condition of the patient and the addition of vasoconstrictor drugs.

Intravascular injection

Systemic toxicity due to inadvertent intravascular local anaesthetic injection should be treated with conventional resuscitative measures and lipid emulsion.

- Give an initial intravenous bolus injection of 20% lipid emulsion 1.5mL/kg over 1min and start an IV infusion of 20% lipid emulsion at 15mL/kg/h.
- After 5min give a maximum of two repeat boluses (same dose) and double the rate to 30mL/kg/h at any time after 5min.

Complications of neuraxial anaesthetic techniques

The incidence of complications from neuraxial blockade are listed in Table 6.4. The Royal College of Anaesthetists Third National Audit Project found the incidence of injury due to epidural block was between 8 and 17 per 100 000. Two-thirds of injuries judged initially as severe resolved fully. Perioperative epidural anaesthesia represent approximately 1 in 7 of all central neuraxial procedures performed, but accounted for more than half of complications leading to harm.

Epidural abscess

- The classical signs of epidural abscess are back pain, fever, catheter insertion, site infection, and leukocytosis occurring 3 or 4 days after the block.

Table 6.4 Incidence of various complications after central neuraxial blockade.

Complication	Risk
Death	1:50 000 (all techniques) 1:125 000 (neuraxial)
Permanent disability (>6/12)	1:24 000 (neuraxial)
Permanent nerve damage (>6/12)	1:7000 (peripheral) 1:25 000–100 000 (neuraxial)
Temporary nerve damage(<6/12)	1:1000–10 000 (peripheral) 1:8700 (neuraxial)
Systemic local anaesthetic toxicity	1:9500 (peripheral) 1:35 000 (epidural)
Failure	1:20 (epidural) 1:50 (spinal) <1:20 (peripheral, nerve stimulator) <1:50 (peripheral, US)
Haematoma	1:20 000 (perioperative epidural) 1:4105 (epidural)
Abscess	1:24 000 (perioperative neuraxial) 1:1368 (epidural)

Data from Cook TM, Counsell D, Wildsmith JAW Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists Br J Anaesth (2009) 102: 179–190 and from Auroy Y, Benhamou D, Barguee L et al Major Complications of Regional Anesthesia in France. Anesthesiology 2002; 97: 1274–80.

- Neurological changes are often not present. If there is clinical suspicion of an epidural abscess, an MRI scan and immediate decompression should be performed urgently in order to prevent long term nerve damage.

Epidural haematoma

- If an unexpected neurological deficit develops during an epidural infusion, epidural haematoma should be suspected, the infusion stopped and the patient's neurological status assessed.
- Early diagnosis, MRI scanning and immediate decompression (less than 8h after the onset of neurological signs) increases the likelihood of neurological recovery.
- Measures to minimize the occurrence of epidural haematoma are discussed in 📖 Pain management, p. 303.

Respiratory depression

- Respiratory depression may occur after neuraxially administered opioids, though the incidence is perhaps less than with opioid-based analgesia via other routes. The mean (95% CI) incidence of respiratory depression (defined as a respiratory rate <10/min), is 0.8 (0.2–2.5%) for intramuscular opioids, 1.2 (0.7–1.9%) for PCA and 1.1 (0.6–1.9%) after epidural analgesia.

- The risk of respiratory depression is increased in patients with other risks (e.g. unstable medical condition, obesity, obstructive sleep apnoea, renal dysfunction, concomitant administration of opioid analgesics or hypnotics by other routes, or extremes of age.
- The risks are increased when morphine or diamorphine are used compared to fentanyl.
- Patients receiving spinal or epidural opioids should be monitored closely.

Block failure and withdrawal

The benefits of extradural analgesia are limited by technical failure in up to 1 in 7 patients. The commonest reasons are:

- Epidural never worked.
- *Technical problem during epidural infusion*—leaking from puncture site, unilateral block, missed segment, catheter fell out.
- Epidural analgesia failed despite a functioning catheter.

The sudden withdrawal of epidural analgesia may precipitate severe pain, anxiety, and myocardial ischaemia. Alternative analgesics should be introduced before discontinuing the epidural.

General strategies to reduce risks associated with regional anaesthesia

The risk of neurological damage is increased with use of a tourniquet, adrenaline, diabetes, and hypotension. Strategies to avoid risks include:

- Strict aseptic technique.
- Appropriate equipment, to include where relevant:
  - Short bevel needle (unlikely to penetrate the epineurium of nerves and are more likely to slide away).
  - Safe needle length for the intended block.
  - Use of nerve stimulation and/or US.
- Slow advancement of needle towards the target nerve.
- Ensuring –ve aspiration prior to injection.
- Slow incremental injection avoiding high pressures.
- Trained assistant to recognize high injection pressure.
- Use of an injection pressure monitor.
- STOP injection if patient reports pain.
- Use appropriate local anaesthetic considering all factors.
- Do not repeat a failed block in the same location.
- Use of US-guided techniques.
- If it feels wrong, or all is not going as it should, stop!

Drugs for neuraxial anaesthesia

The quality of epidural or spinal analgesia with LA is improved with addition of opioids. However, opioids commonly cause pruritis, nausea, and vomiting, and occasionally respiratory depression.

Local anaesthetics

- There is little practical difference between bupivacaine, levobupivacaine, and ropivacaine for surgical anaesthesia, but motor block occurs in the rank order ropivacaine< levobupivacaine< bupivacaine.

- The incidence of motor blockade after epidural administration is reduced by the use of lower concentrations (0.1 or 0.125%).
- All local anaesthetics may be infused without opioid, but increased infusion rates, early rescue analgesia and hypotension are common.

Opioids

- The addition of opioid spares LA and improves analgesia. Morphine has poor lipid solubility and a long CSF half-life, accounting for its propensity for late respiratory depression in doses >300micrograms. Fentanyl is lipid soluble with a short CSF half-life, whereas diamorphine has intermediate properties between the two.
- Liposomal morphine has been introduced for epidural use without concomitant LA. It provides pain relief for up to 48h. Dose should be restricted to 5–15mg depending on patient characteristics.

Others

Alpha-2 agonists. Both adrenaline (2.5μg/ml) and clonidine improve analgesia when added to post-operative thoracic epidural infusions containing fentanyl and LA.

Drugs for spinal anaesthesia

- Spinal anaesthesia with opioid supplementation is commonly used for lower limb revascularization surgery.
- The duration of anaesthesia is proportional to the mass of local anaesthetic, but large doses increase the risk of hypotension.
- A combined spinal epidural technique (CSE) is useful to maintain haemodynamic stability for long procedures.
- Hyperbaric bupivacaine 0.5% is most commonly used, typically in a dose of 2–4mL. Plain bupivacaine and levobupivacaine are also available for spinal administration.
- The most commonly used opioids for spinal administration are morphine 0.1–0.3mg and diamorphine 0.2–0.4mg. These provide 18–24h of analgesia and smooth out the transition between the profound block produced by the LA, and the establishment of adequate systemic analgesia.

Drugs for epidural anaesthesia

- The spread of epidural LA increases with age, obesity, dose, and volume of LA and higher injection site.
- Lumbar epidural block with LA is associated with more motor block, extensive sympathetic block, and proximal baroreceptor mediated reflex vasoconstriction. In the presence of blood loss, bradycardia, vasodilation, myocardial ischaemia, and hypotension are more common. Thoracic epidural anaesthesia is usually preferred.
- Levobupivacaine or bupivacaine are usually used for epidural anaesthesia (see 📖 Pain Management, p. 303).
- Three modes of epidural administration are available for pain relief after surgery—continuous infusion; patient-controlled epidural analgesia (PCEA); and Intermittent bolus.

Further reading

Auroy Y, Benhamou D, Barguee L, *et al*. Major complications of regional anesthesia in France. *Anesthesiology* 2002; **97**: 1274–80.

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Marhofer P, Harrop-Griffiths W, Willschke H, Kirchmair L. Fifteen years of ultrasound guidance in regional anaesthesia: Part 2—Recent developments in block techniques. *Br J Anaesth* 2010; **104**(6): 673–83.

Ultrasound and regional anaesthesia for vascular surgery

Introduction

The introduction of NICE guidelines for the placement of central venous lines has established the presence of ultrasound technology in most anaesthetic rooms. Effective ultrasound-guided regional anaesthesia (UGRA) holds particular advantages for high risk vascular surgical patients (see 📖 Pain management, regional techniques, p. 305) because perioperative complications can have disastrous consequences. The risks of regional anaesthesia (Table 6.2) can potentially further be reduced by the use of UGRA. US-guided blocks are more likely to be successful and have a faster onset than those performed using a peripheral nerve stimulator.

Basics of ultrasound

- Medical US frequencies range from 2 to 20MHz and the US transducers (probes) act as both a transmitter and receiver of sound waves.
- High frequency (10–18MHz) probes provide more detailed image quality, but lack tissue penetration. These linear probes are mostly used for superficial nerves and structures (about 5cm).
- Lower frequency (2–5MHz) curved probes would be more appropriate for deeper structures (6–13cm), e.g. neuraxial or lumbar plexus blocks.
- Needles inserted along the plane of the ultrasound beam (in-plane) are easier to see at low angulations to the horizontal (30°). In-plane needling techniques should produce an US image of the whole length of the needle and 6 it should be easier to identify the tip of the needle.
- Needles inserted out-of-plane to the US beam tend to be easier to see at high angulations (75°). Nerves should be visualized in cross section or 'short-axis' and not along the 'long axis'.
- Regional Anaesthesia of United Kingdom (RAUK) recommends an in-plane approach for the majority of blocks for less experienced clinicians. An out-of-plane approach may be more appropriate once advanced needling techniques and experience is acquired.

Ultrasound-guided vascular access

The advantages of US for central venous access are discussed in 📖 Central venous catheterization, p. 252. US is also useful for difficult venous access (e.g. obese patients) or difficult arterial access.

Ultrasound-guided regional anaesthesia

Advantages of UGRA

Advantages of UGRA are that it can be used to:

- Identify the relevant anatomy, including anatomical variations.
- Observe the needle throughout the technique.
- Visualize the spread of LA.
- Identify and avoid anatomical structures that can cause complications such as blood vessels and lungs. Avoiding vascular structures will reduce the incidence of haematomas that may distort the surgical field for example in AV fistula and carotid surgery.
- The ability to visualize the needle and its tip theoretically prevents direct axonal trauma or unintended intra-neural injection.
- Ultrasound allows the deposit of LA directly next to the intended nerve, the use of 'hydro-dissection' to manipulate the spread of LA and the ability to detect intravascular injection (See Fig. 6.6).
- There is some evidence to suggest a reduction in procedure time, and a more rapid onset of block with a longer duration.
- Much lower volumes of local anaesthetic can be used. This:
  - Reduces anatomical distortion of the surgical field.
  - Reduces the risks of local anaesthetic toxicity.
  - Reduces some adverse effects (incidence of phrenic nerve block and Horners syndrome with interscalene block <50% with UGRA).
- Presents the option to repeat nerve block in case of failure.
- The success rates of most blocks are higher than without ultrasound (>97%). A successful block avoids the need to convert to GA in the case of block failure.
- Using US avoids a physical interaction with nerves like the need to obtain paraesthesia with nerve stimulation techniques and avoids muscle twitches, thus making this technique less painful for patients.
- In addition, US can be used to teach anatomy relevant to the intended block.

Drawbacks of UGRA

- Users need education in how US works, and limitations of its technology.
- Each block needs to be 'relearnt' to accommodate a difference in technique—there is a definite learning curve with each block, as well as with the in-plane and out-of-plane needling techniques.
- Despite its advantages, there is no evidence to date that UGRA reduces the rate of rare complications, and it is unlikely that all complications will be prevented completely.

Ultrasound-assisted neuraxial blocks

Pre-procedural ultrasound

US can be used to scan the spine before central neuraxial blockade techniques are performed and provide helpful information on anatomical

structures. This can reduce the number of needling attempts, time taken, and potentially reduce the risk of spinal haematoma formation.

- A low frequency curved probe should be used to provide better images of the spine.
- Appropriate spinal level can accurately be determined by scanning from base of spine and counting spinous processes as the probe is moved cranially. Midline of the spine will become apparent as the tips of spinous processes are marked with a skin marker.
- The optimal interspinous space can be identified by locating the best acoustic window that will show the minimum depth from the skin to the ligament flavum and dural structures.
- The depth may vary depending on the pressure that the operator applies to the probe on the skin of the patient.
- The direction of the probe that provides the best view of the acoustic window should be noted and used as a guide for needle direction.
- Armed with the above information a clinician should find neuraxial techniques less challenging.

Ultrasound-guided neuraxial blocks

- Central neuraxial blocks are possible to perform using US guidance, but are more difficult in practical terms, especially in adults.
- They require a low frequency curved probe to be positioned in a longitudinal, paramedian approach to the spine. The operator requires an assistant or an automated springloaded loss of resistance syringe to locate the epidural space.
- The needle is introduced in an in-plane paramedian fashion. This is an advanced techique that will increase in use as ultrasound technology and experienced needling skills develop. Only a few clinicians worldwide are currently using this technique on a regular basis.

General tips for success with ultrasound-guided nerve blocks

- Use the highest possible frequency probe and the lowest possible depth setting on the US.
- Keep the patient awake or conscious sedation as much as possible.
- Make sure the patient and operator is comfortable.
- Keep your eyes, hands, and US image along the same axis.
- Check probe orientation. Medial on US screen should be medial on patient.
- Use an in-plane approach as a beginner. Possible exceptions to this are interscalene and femoral nerve blocks.
- Dim the lights or reduce ambient light.
- Use a nerve stimulator in conjunction if difficult US images are obtained.
- Prime the needle to eliminate any air.
- Do not advance the needle unless you can observe the tip.
- Before injecting, release the pressure on the US probe and check for compressed veins.
- Aim for around the nerve, not directly to the nerve.

- Identify anatomy you want to avoid, e.g. arteries, lungs, and anticipate needle passage through connective tissue planes.
- First inject local below the nerve if possible.
- Observe LA spread in real time, to avoid intravascular injection.
- Create a safe space with the LA in which you can move your needle to completely surround the nerve (hydro dissection).
- Do not inject if high resistance is encountered.
- Use bigger volumes of LA if poor US images are obtained.

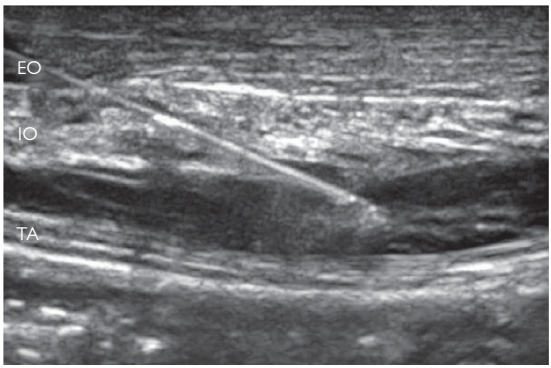

Fig. 6.6 US image of TAP block showing needle and transversus abdominis plane distended by 20mL of LA. EO = external oblique, IO = internal oblique, TA = transversus abdominis.

Reproduced from Allman et al., Oxford Handbook of Anaesthesia, 2011, Figure 41.17, p. 1156, with permission from Oxford University Press.

Further reading

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Useful websites

- www.usra.ca
- www.nysora.com
- www.neuraxiom.com

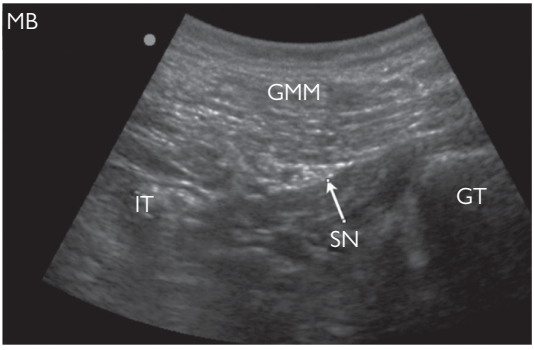

Fig. 6.7 US image of sciatic nerve (arrows) as it passes underneath gluteus maximus muscle (GMM) and between greater trochanter (GT) and ischial tuberosity (IT).

Reproduced from Allman *et al. Oxford Handbook of Anaesthesia*, 2011, fig. 41.21, p. 1161, with permission from Oxford University Press.

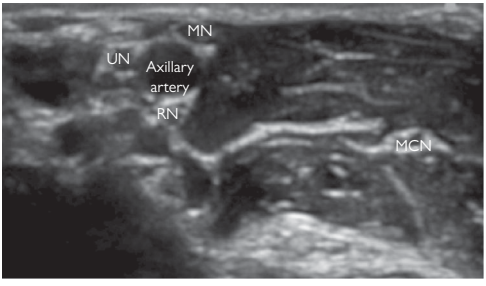

Fig. 6.8 US image showing the median nerve (MN), ulnar nerve (UN), and radial nerve (RN) lying around the axillary artery; the musculocutaneous nerve (MCN) lies between the biceps and coracobrachialis muscles.

Reproduced from Allman et al., Oxford Handbook of Anaesthesia, 2011, Figure 41.7, p. 1142, with permission from Oxford University Press.

Specific regional blocks

Cervical plexus blocks

Indication

Carotid endarterectomy (CEA)

- CEA is indicated for the 1° and 2° prevention of stroke in high risk patients. It can be performed under LA/RA or GA (with or without a regional block).

- Regional techniques include cervical plexus block (superficial, deep or combined superficial and deep), local infiltration and cervical epidural anaesthesia. Cervical plexus block is currently used most commonly.
- RA confers some advantages over GA for CEA surgery, mainly by monitoring neurological function (and, hence, adequacy of cerebral perfusion) during carotid artery cross-clamping.
- The deep cervical plexus block may be complicated by intravascular injection, subarachnoid injection and block of the phrenic, recurrent laryngeal and vagus nerves. Compared with superficial blocks, deep blocks are associated with a higher rate of conversion to GA (2.0 versus 0.4%) and a higher complication rate (0.25 versus 0%).
- Epidural anaesthesia is associated with higher rates of serious complications including subarachnoid injection and increased conversion rates to GA. It is not recommended for routine CEA.
- Local infiltration using a 'layer by layer' technique or a four-step method where individual cervical nerves are infiltrated, are both slow, uncomfortable and are not routinely used.
- Adjunctive sedative drugs are required in up to 66% of cases. Sedation must be carefully titrated to maintain verbal contact with the patient.
- Supplementary LA infiltration by the surgeon is needed in over 50% of patients. LA supplementation does not affect patient satisfaction.
- The complication rate is higher with deep or combined blocks compared to the superficial block alone (0.25 versus 0% (deep versus superficial).

Superficial cervical plexus block (CePB)

Landmarks

- The cervical plexus is formed from the ventral rami of the first, second, third, and fourth cervical spinal nerves. The superficial branches of the cervical plexus (from C2–4) are all sensory, supplying skin and sc tissue of the anterolateral neck and posterior aspect of the head.
- The branches of the plexus pierce the cervical fascia to emerge posterior to the mid-point of sternocleidomastoid muscle.
- The branches of the superficial cervical plexus are the lesser occipital, greater auricular, transverse cervical, and supraclavicular nerves.

Technique

- Turn the patient's head slightly to face the contralateral side. Insert a 22G or smaller needle perpendicular to the skin, and into the midpoint of the posterior border of the sternocleidomastoid muscle. The superficial nerves emerge at this point and the needle must puncture the first fascial layer.
- Infiltrate approximately 20mL of LA solution subcutaneously in a 'fan' cranially and caudally from the posterior border of sternocleidomastoid to the midline. Take caution to avoid the external jugular vein.
- Supplemental LA is most often needed at skin or carotid sheath incision.
- It is recommended that 1.4mg/kg of levobupivacaine or ropivacaine is used to allow for supplementation by the surgeon during surgery

Deep cervical plexus block

Landmarks

- This is mainly used in combination with the superficial CePB as the deep cervical plexus branches are purely motor, supplying muscles of the neck.
- The block is performed where cervical nerves exit through intervertebral foramina at the level of the transverse processes. Identify the posterior border of sternomastoid muscle at the thyroid cartilage (C4).

Technique

- Position the patient as for the superficial block.
- This block can be performed as a single-injection technique at C4 using a peripheral nerve stimulator or at three levels (C2, C3, and C4).
- For the latter, landmarks include a line drawn between the tip of the mastoid process and Chassaignac's tubercle (the prominent transverse process of C6, palpable at the level of the cricoid cartilage). The transverse process of C2, C3, and C4 lie along this line. C2 is approximately one finger's breadth below the mastoid process and C3, C4 follow at equidistant intervals caudad along this line.
- Insert a 22g or smaller needle perpendicular to the skin at each level, aiming slightly caudad, medial and posteriorly (towards the contralateral elbow) until either the transverse process is met or paraesthesia is elicited, indicating encounter with nerve root. The angulation aims to avoid injection into the vertebral artery or into the subarachnoid space.
- After careful aspiration, inject approximately 5ml LA solution, accounting for a third of the total volume; the rest of the dose is used for the superficial block. Single-injection is effective as there is spread of LA solution through the paravertebral plane with little resistance.
- Complications of the deep CPB include intrathecal or epidural injection, intravascular injection into the vertebral artery leading to loss of consciousness or convulsions, phrenic nerve palsy leading to potential respiratory embarrassment, hoarseness from recurrent laryngeal palsy and dysphagia.

Intermediate cervical plexus block

This is described as the injection of at least 30mL of LA solution between the superficial (sc) and deep (prevertebral) cervical fascial layers, producing spread of LA to cervical nerve roots. The injection is through the investing fascia of the neck, posterior to sternocleidomastoid (Fig. 6.9).

Little clinical data is available on this variation of the CePB. This volume of LA may distort the anatomy of the surgical field and vascular surgeons may prefer to infiltrate the LA themselves.

Cervical plexus block: tips for success

- Ask the patient to lift the head before siting the block, to help identify the posterior border of the sternocleidomastoid muscle.
- Use the superficial CePB approach, because it is as effective as the deep CePB, but with less complications.
- Do not use adrenaline containing LA solutions for this block. Patients may develop a tachycardia from the adrenaline.
- Carotid surgery also requires blockade of the glossopharyngeal nerve branches. This is easily accomplished by the surgeon injecting the LA inside the carotid artery sheath during surgery.
- Do not perform bilateral deep cervical plexus blocks because of the potential danger of bilateral phrenic nerve palsy.

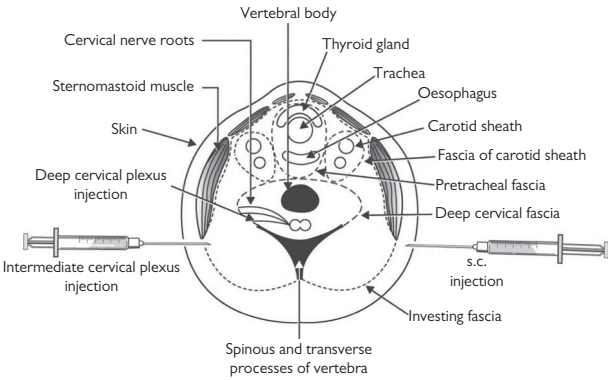

Fig. 6.9 Diagrammatic cross section of the neck at C4 level showing the site of injection of the cervical plexus blocks: deep, intermediate, and subcutaneous. Note the investing layer of cervical fascia that putatively acts as a barrier to deep spread of s.c. injection. It is this anatomical layer whose existence is questioned by the studies of Nash and colleagues.

Reproduced from Ramachandran S K et al., 'Comparison of intermediate vs subcutaneous cervical plexus block for carotid endarterectomy', British Journal of Anaesthesia, 2011, 107, pp. 157–163, by permission of Oxford University Press and the British Journal of Anaesthesia.

Ultrasound-guided cervical plexus block

The US-guided technique is a modification of the intermediate block described above. The significant differences are that the operator can identify the fascial plane deep to the sternocleidomastoid muscle, avoid puncture of the vascular structures and reduce the volume of LA required.

This technique reduces the need for supplementary LA inflitration compared with the traditional landmark techniques

Preliminary scan

- From the level of cricoid cartilage, identify the interscalene grove and brachial plexus. Scan cranially to view the C4 nerve root deep to the sternocleidomastoid (SCM) muscle and anterior to scalenus medius. The superficial cervical plexus can be seen as a line of hyperechoic

beads in the fascial plane deep to the SCM muscle. It may be difficult to visualize the individual nerves, but this is a fascial plane block, so do not waste time trying to identify them.
- Less experienced US users may struggle to identify the nerve root of C4. An easier approach would be to scan the carotid artery in a cross-sectional view. Follow the carotid artery cranially until the carotid bifurcation can be identified. At this level the SCM muscle will be lateral and superficial to the carotid artery on the ultrasound image and the fascial plane underneath the SCM muscle can be identified.
- It is important to limit the pressure applied to the carotid artery during scanning, to avoid atheromatous plaque rupture and a potential embolic event. Excessive pressure will easily collapse the internal jugular vein, making it difficult to identify and leaving it prone to needle puncture. This may lead to haematoma formation and spoiling the surgical field.

Technique
- Insert the needle posterior to the US probe using an in-plane approach (from posterior to anterior). Injection of about 10–15mL of LA should fill the fascial plane deep to the sternocleidomastoid muscle and be seen to spread freely towards the internal jugular vein. When performing a block for carotid surgery, the needle can be advanced slowly, under direct vision, towards the vascular structures and 3–5mL of LA deposited around the carotid sheath, not within it.
- Perform the US-guided block at the level of the carotid bifurcation. The level of the bifurcation varies and a high bifurcation may require surgical supplementation near the angle of the jaw because of skin retraction. Conversely, a low bifurcation may require supplementation for the skin incision at the base of the neck near the clavicle.
- US-guided cervical plexus block combined with US-guided infiltration around the carotid sheath of 15–20mL at the level of the carotid bifurcation provides adequate anaesthesia and reduces the need for surgical LA supplementation.

Brachial plexus blocks

RA is a useful technique for vascular access surgery (see 📖 Anaesthesia for renal vascular access and fistula formation, p. 426) and improves surgical outcome compared with GA.

Interscalene block

Indications

Upper limb amputation, vascular access surgery, and axillo-femoral bypass
- The interscalene approach to the brachial plexus is not often required for vascular surgery procedures. The more distal approaches to the brachial plexus are more appropriate for AV fistula surgery. The skin in the axilla is innervated by dermatome of T2, which is not within the brachial plexus. This area often needs surgical LA supplementation in procedures like high AV fistula formation involving transposition of the artery or graft.
- Interscalene block is also inappropriate for surgeries involving the medial aspect of the upper extremity due to inconsistent blockade of the lower trunk (C8 and T1).

- Interscalene block with the nerve stimulation technique requires large volumes of LA almost always produces ipsilateral phrenic nerve paralysis due to blockade of the 3rd, 4th, and 5th cervical nerve roots. This may decrease the patient's FRC by 25% and may not be suitable for patients with emphysema and other chronic lung diseases with decreased pulmonary reserve.
- Patients should be warned about phrenic nerve palsy (100%), heavy arm (motor block) (100%), Horner's syndrome due to blockade of the sympathetic chain (miosis, ptosis, and anhydrosis) (20%), hoarse voice (recurrent laryngeal nerve block) (10%).

Landmarks

With the patient lying supine, mark the posterior border of the strenocleidomastoid muscle at the level of the thyroid cartilage (C4). Palpate the scalenus anterior muscle underneath the lateral aspect of the sternomastoid muscle, then move slightly lateral to feel the interscalene groove between scalenus anterior and medius.

Technique: Meier's approach

Introduce a 50mm, 22G needle at the level of C4 along the axis of the interscalene groove, aiming caudally towards the subclavian artery pulsation as it passes over the first rib. Deltoid or biceps muscle stimulation is accepted. Aspirate and inject 20–30mL of LA.

Interscalene block: tips for success

- If the diaphragm is stimulated (phrenic nerve), aim more posteriorly.
- If the scapula is stimulated (dorsal scapular nerve) aim more anteriorly.
- Let the patient lift the head off the pillow or a short sharp sniff will help identify the interscalene groove.
- Avoid puncture of the external jugular vein.
- Keep patients awake or use conscious sedation.
- Do not inject LA if high resistance is encountered.

Ultrasound-guided interscalene block:

- US imaging improves the interscalene approach primarily by allowing visualization of the spread of LA within the fascia surrounding the trunks. This permits the use of lower amounts of LA.
- Reducing the volume of LA to 10mL significantly decreases the incidence of phrenic nerve palsy and other common adverse effects associated with the nerve stimulation technique.
- US guidance improves both the quality of the nerve block and shortens the time of onset of sensory blockade.

Preliminary scan

- Place a high frequency linear probe in a coronal oblique plane in the suprascapular fossa, exactly the same as for supraclavicular approach. This will provide a cross-sectional view of the subclavian artery, with the brachial plexus lateral and superficial.
- Now scan in a cranial direction along the interscalene groove changing the probe direction to an axial oblique plane (cross-sectional to the groove).

- At the level of the cricoid cartilage the brachial plexus trunks appear as three distinct oval hypo-echoic areas between the anterior and middle scalene muscles. The carotid artery and internal jugular vein are located on the medial side of the image. This method of tracing the nerve roots proximally in the interscalene groove provides direct visualization of the nerves and makes identification of the plexus at this level much easier than medial to lateral scanning at the level of the cricoid cartilage(C4).

Technique

- The ***in plane*** approach requires a 50mm 22G needle entry point on the lateral end of the ultrasound probe after skin LA infiltration. Advance the needle along the long axis of the transducer in the same plane as the ultrasound beam. With the in plane approach, the needle shaft and tip within the scalenus medius muscle can be visualized in real time as the needle is advanced towards the nerves.
- Aim to deposit LA between the nerve roots from the posterior side of the plexus, usually between C5 and C6, depending on spread. The major advantage of US is that the needle position can be moved to manipulate the spread of LA around the plexus, if the operator can always visualize the needle tip.
- The **out of plane** approach may feel more familiar, because it is similar needling as the nerve stimulation technique. Align the target nerves with the midpoint of the transducer and then insert a 50-mm needle at the level of C4 along the axis of the interscalene groove, aiming caudally.
- It can be technically difficult to identify the needle tip. Injection of a small volume of LA during needle advancement may help tracking of the needle tip. Aim just anterior or posterior to the plexus (do not aim for the middle).
- Once the correct needle position is obtained, inject a volume of 10–20mL LA depending on appropriate spread. The pattern of LA spread around the target nerves can be observed in real time during injection. The local will appear as a hypo-echoic collection around the nerves and between the interscalene muscles.

Ultrasound-guided interscalene block: tips for success

- Use an in-plane approach from posterior to anterior.
- Adjust the needle position during injection to manipulate and optimize LA spread if required. Most of the volume of LA should be deposited on the posterior side of the plexus and try to avoid the injectate moving over the front of the scalenus anterior muscle, because this is where the phrenic nerve is located.
- Scan proximal and distal to assess the extent of LA spread.
- This is a surprisingly superficial block, usually 2–3cm. C5 superficial to C6.
- Nerve root identification is possible by examining the shape of the corresponding transverse processes. The transverse process of C6 has large anterior (Chassaignac's tubercle) and posterior tubercles and casts a 'U'-shaped shadow. The vertebral artery is normally seen below C6.

Supraclavicular block

Indication

Upper limb vascular access surgery

- The supraclavicular approach has several advantages over more distal approaches to the brachial plexus.
- Due to the compact arrangement of the trunks of the brachial plexus at this level, there is a more rapid onset of the block and more complete blockade of the nerves supplying the upper extremity, including the axillary nerve and the musculocutanous nerve, but excluding the intercostobrachial nerve.
- Before the use of US, the supraclavicular approach was frequently avoided because of the increased risk of pneumothorax and puncture of the subclavian, superficial (transverse) cervical, suprascapular, or dorsal scapular arteries.

Landmark

Place a finger on the skin in the suprascapular fossa along the axis of the interscalene groove. Feel for the pulsation of the subclavian artery over the first rib.

Technique

Introduce a 50-mm needle posterior to your finger in the groove, aiming posterior to the artery. Flexion or extension of the wrist indicates the correct stimulation. Inject 30–40mL of LA.

Supraclavicular block: tips for success

- Do not angulate the needle medially.
- Contact with bone indicates the first rib, then walk the needle along the rib towards the plexus.
- The ulnar nerve is missed in about 5% of patients.
- Use US.

Ultrasound-guided supraclavicular block

- US guidance has increased the safety of the supraclavicular approach so that in experienced hands it is now the block of choice for most surgical procedures below the shoulder.
- It can be performed faster with US guidance compared with nerve stimulation (5 versus 10min). The anaesthetist can visualize and avoid the subclavian artery, the first rib, as well as the dome of the lung, and visualize needle placement and spread of LA.

Preliminary scan

- Position the patient supine with the head turned to the contralateral side.
- Place a linear probe in a coronal oblique plane in the supraclavicular fossa. Manipulate the probe to observe a cross-sectional view of the pulsatile subclavian artery, above the hyperechoic first rib and dome of the pleura.
- The nerves (trunks or divisions) of the brachial plexus appear as hypoechoic nodules in clusters like a bunch of grapes, lateral and superficial to the subclavian artery.

Technique

- Use an in line approach to the nerves from posterior to anterior.
- Insert a 50-mm 22G needle in plane from the lateral end of the transducer aiming for the inferior part of the plexus, just above the first rib.
- Pierce the brachial plexus sheath and inject a 2–5mL that will create a safe space to slowly advance the needle towards ('hydrodissection') the corner formed by the first rib and subclavian artery.
- Inject 10–15mL and observe the LA spread. Inadequate spread to the superior part of the plexus will require repositioning of the needle and administering the remaining LA.
- A volume of 20–30mL is often required.

Ultrasound-guided supraclavicular block: tips for success

- Use an in line approach.
- The transverse cervical artery is often (40%) seen dividing the nerve plexus, which may prevent LA spread to the whole plexus.
- The suprascapular fossa of petite or thin patients may require a smaller (25mm) footprint probe.

Infraclavicular block

Indication

Upper limb vascular access surgery

- The infraclavicular approach is a very useful alternative to the supraclavicular approach, because many patients with an AV fistula have a dialysis catheter placed in the supraclavicular region.
- The infraclavicular approach is recommended if US is not available, because the risk of pneumothorax is significantly lower than with the supraclavicular approach. The phrenic nerve is not blocked, but the infraclavicular block targets the brachial plexus at the level of the cords, which surround the second part of the axillary artery. This point is proximal to the origin of the musculocutaneous, axillary, and medial brachial cutaneous nerves. This may result in a higher success rate of complete blockade with a single injection technique.
- Similar to the supraclavicular and axillary approaches, the intercostobrachial nerve must be blocked separately in the axilla to anaesthetize the inner aspect of the upper arm.
- There are several landmark techniques such as subcoracoid and vertical infraclavicular block (VIB). The VIB is the favoured technique.

Vertical infraclavicular block

Landmark

Position the patient supine and mark the mid-point between the anterior process of the acromion and the jugular notch, just below the clavicle. This point indicates the position of the cords, which is still situated lateral to the subclavian artery, which may be palpable.

Technique

- Introduce a 50-mm needle in an absolutely vertical direction, with no medial angulation. Look for extension or flexion of the hand
- Inject 20–30mL of LA.

Infraclavicular block: tips for success

- Pectoralis muscle stimulation indicates the needle is too superficial.
- Elbow flexion indicates stimulation of the lateral cord and the musculocutanous nerve. The needle is too lateral and superficial.
- The needle should be vertical to the floor, not perpendicular to the skin, which would result in pleural puncture.
- The needle entry point should be as close the inferior border of the clavicle.
- There must be no medial angulation of the needle.
- The plexus depth is usually 3–4 cm.
- Use US to locate the position of the subclavian artery and introduce your needle lateral to it.

Ultrasound-guided infraclavicular block

- The infraclavicular anatomy may be harder to visualize under US guidance, particularly in obese patients, because the brachial plexus is situated more deeply compared to other approaches.
- The pectoralis major and minor muscles lie anterior to the brachial plexus. The lateral, posterior, and medial cords are seen in close proximity to the artery, usually on the lateral side when scanning closer to the clavicle, but at the level of the coracoid process the cords start to entwine themselves around the artery. The medial cord moves first to the posterior side of the artery, followed by the posterior cord.

Preliminary scan

- Position the patient supine with their arms by their sides
- Place a medium frequency curvilinear transducer (6–9MHz) immediately medial to the coracoid process underneath the clavicle in a parasagittal plane (delto-pectoral groove) to obtain a cross-sectional view of the axillary vessels and cords.
- Nerves in the infraclavicular region often appear hyperechoic and the axillary artery and vein are anechoic. The artery is pulsatile and the vein is compressible. The pleura can often be visualized on the inferior medial side.

Technique

- Insert a 5–7cm 22G needle below the clavicle depending on the depth. An US-specific needle makes visualization easier when the nerves are deep.
- Advance the needle at a 45–50° angle from the cephalad end of the probe along its long axis in the caudad direction. Target the posterior cord at the posterior edge of the artery.
- A nerve stimulator may be used to correctly position the needle and inject 20–30mL of LA. LA spread around the artery when injected posterior to the axillary artery results in complete arm blockade. A second injection (10mL) more superficial to the artery will target the lateral cord, including the musculocutanous nerve.

Tips for success

- Use a lower frequency probe for obese patients.
- The musculocutanous nerve originates from the lateral cord, which can be seen as the most superficial cord for this technique.
- Perform the block as close as possible to the clavicle, while the whole plexus is still on the lateral side of the artery. More laterally, the cords move around the artery, requiring multiple injections; the nerves are also located more deeply.
- Arm abduction to 90° will stretch the brachial plexus, make the nerves more visible and more superficial.

Axillary block

Indications

Procedures to the elbow and distal lower limb

- Axillary nerve blocks have a high success rate and are not associated with phrenic nerve involvement or pneumothorax. Hence, axillary block is advantageous in patients with pulmonary disease.
- *Contraindications:* include patients unable to abduct the arm to the position necessary to perform the block, localized infection in the axilla, or enlarged axillary lymph nodes.
- In the axilla, the terminal branches of the brachial plexus (median (C5–C8, T1), ulnar (C7–C8,T1) and radial (C5–C8, T1) nerves) are situated in close relation to the axillary artery, though with some anatomical variation. These structures are within a perivascular sheath which also contains the axillary vein medial to the artery, medial cutaneous nerve of the arm (C8,T1) and the medial cutaneous nerve of the forearm (C8,T1).
- The musculocutaneous nerve (see Fig 6.8) (C5–C7) and the intercostobrachial nerve (T2) are found outside the perivascular sheath.

Landmark

- Position the patient supine with the arm abducted to 90° and the elbow flexed to 90°.
- The axillary artery can be palpated in the axilla, directly inferior to the insertion of the pectoralis major muscle. Excessive abduction in the shoulder joint should be avoided because it makes palpation of the axillary artery pulse difficult.
- Once the pulse is felt, it should be straddled between the index and the middle finger and firmly pressed against the humerus to prevent the axillary artery rolling away during needle insertion.

Technique

- Insert a 50-mm, 22G nerve stimulator needle just in front of the palpating fingers and advance it slowly at 45° cephalad until stimulation of the brachial plexus, arterial blood or paraesthesia is obtained
- The usual depth is 1–2cm.
- The median and musculocutaneous nerves are usually above the artery, the ulnar nerve usually below and the radial nerve further behind and below the artery.
- Once the required response is obtained, inject 30–40mL LA slowly with intermittent aspiration to rule out an intravascular injection.

- The median nerve response is flexion of the index and middle finger. The ulnar nerve response is thumb adduction and little finger flexion. Stimulation of the radial nerve causes thumb extension and of musculocutaneous nerve causes elbow flexion. The musculocutaneous nerve often departs from the lateral cord in the proximal axilla and is commonly spared with the single shot axillary approach.

Axillary block: tips for success

- Infiltrate LA infiltrated tangentially, rather than at a single insertion point. The neurovascular bundle is very superficial and may be reached with the local infiltration needle. This also allows for needle repositioning during block performance if required.
- If the artery is punctured inadvertently with the block needle then a transarterial technique of transfixing artery may be used. Two-thirds of the LA should be deposited behind artery and one-third in front.
- Anatomical variation is very common in the axilla and a multiple injection technique, stimulating the appropriate nerves around the artery will increase success.

Ultrasound-guided axillary block

- A major advantage of using US is that musculocutaneous nerve blockade can be confirmed.
- Three of the four main nerves are easily visualized with US, but the radial nerve may be difficult to detect, because of post-acoustic enhancement, which distorts the image.
- US-guided axillary block decreases block failure rate and time of onset of sensory blockade.
- The success of US-guided axillary blocks depends on the multiple needle approach in which each nerve is identified individually and spread of LA is observed around the median, ulnar, radial, and musculocutaneous nerves.

Preliminary scan

- Position a linear, high frequency transducer in the transverse plane to obtain the best possible transverse view of the axillary artery. Axillary artery is hypo-echoic and pulsatile. Axillary veins are compressible.
- Relieve transducer pressure intermittently to confirm the compressible axillary vein and to identify non-compressible structures around the artery. Non-compressible structures are often the nerves, which may appear as round or oval shaped hypo-echoic nodules with hyperechoic fascicles. Often superficial, even in obese patients.
- Identify the biceps and coracobrachialis muscles lateral to the axillary artery, and the triceps muscle and conjoint tendon medial and posterior to the artery.
- Identify the humerus deep to the muscles. The median (between the artery and skin), ulnar (between artery and medial vein) and radial (posterior and medial to the artery) nerves are situated around the axillary artery and outside of the muscle layers. They often have a honeycomb appearance and are heterogeneous in echogenicity.

- Move the transducer proximally towards the axilla and distally towards the elbow to evaluate the course of each nerve. The median nerve has a close relationship with the artery all the way to the antecubital fossa where it is located on the medial side of the brachial artery. The ulnar nerve becomes more superficial and moves away from the artery further distal in the upper arm as you trace it towards the medial epicondyle of the elbow. The radial nerve is often most difficult to locate and it frequently lies deep to the ulnar nerve in the proximal axilla.
- When transducer is moved more distally, radial nerve descends and disappears under triceps muscle. The radial nerve can be visualized again as it travels posteriorly around the humeral shaft in the humeral groove. Identify the musculocutaneous nerve, which lies most commonly in the plane between the biceps and coracobrachialis muscles, but there is considerable variations in its distance from the artery.

Technique

- Insert a 50-mm 22G needle parallel to the long axis of the transducer in plane from the biceps side of the probe. In cross-section, nerves appear as round or oval shaped hypo-echoic nodules with hyperechoic rims. Axillary veins are often not seen because they may be compressed by US transducer. In-plane approach allows needle advancement to be visualized in real-time as needle approaches target nerves.
- Nerve stimulation can easily be used in conjunction to help identify the individual nerves.
- Identify and block the musculocutaneous nerve separately as it branches in the coracobrachialis muscle.
- Inject 5–10mL LA at each nerve location and ensure spread around each individual nerve at the time of injection.

Ultrasound-guided axillary block: tips for success

- Expect anatomical variation of the nerve locations if there is more than one vein or artery on the preliminary scan.
- The radial nerve may be difficult to visualize. Do not waste time looking for it, but deposit LA to the posterior medial side of the artery, superficial to the conjoint tendon. Injection of LA often makes the radial nerve easier to visualize with US.
- Locate and occlude the axillary vein(s) with transducer applied pressure to avoid unintentional intravascular injection.
- The musculocutanous nerve location may vary and be found anywhere between the artery and the fascial plane between biceps and the coracobrachialis muscle. Scan distal and proximal in the axilla to help identify the musculocutaneous nerve.

Femoral nerve block

Indications

Above knee amputation, femoral endarterectomy, femoro-popliteal bypass surgery and endovenous laser ablation (EVLA). A neuraxial technique may be more appropriate to facilitate bilateral surgery.

Landmark

- The femoral nerve is consistantly lateral to the femoral artery, deep to the fascia iliaca and superficial to the iliopsoas muscle.
- Identify the needle entry point 1cm below the inguinal ligament and 1cm lateral to the femoral artery.

Technique

- Position the patient supine with the leg in the neutral position
- Introduce a 50-mm 22G needle at a 45° angle in a proximal direction. Once through the skin two further distinct 'pops' should be felt. The first is the fascia lata and the second the fascia iliaca.
- Nerve stimulation should elicit movement of the patella.
- Inject 20–30mL of LA. A higher volume usually blocks the lateral cutaneous nerve of the thigh.

Femoral nerve block: tips for success

- The obturator nerve is not reliably blocked.
- The femoral nerve starts dividing as soon as it passes under the inguinal ligament, so keep close to inguinal ligament.
- Sartorius stimulation indicates needle is too lateral and superficial.
- Inject 15–20mL of lidocaine for EVLA surgery. This will produce a block for up to 2h and patients can mobilize soon afterwards. Care must be taken when these patients stand up for the first time to evaluate motor function.
- The incision for femoral artery access often extends above the inguinal ligament, so let the surgeon infiltrate LA to this area

Ultrasound-guided femoral nerve blockade

Preliminary scan

- Position the patient supine with the leg in the neutral position.
- Identify the femoral artery, with the femoral vein on the medial side, which may be compressed by the transducer pressure.
- If two arteries are seen, scan more proximally (cephalad) to visualize the artery before the profunda artery branches off.
- Deep to the femoral vessels is the iliopsoas (iliacus) muscle covered by the fascia iliaca. This fascial layer (hyperechoic line) covers the femoral nerve and the iliopsoas muscle, but not the femoral vessels.
- The femoral nerve appears within a triangular hyperechoic region, which is found lateral to the femoral artery and superficial to the iliopsoas muscle.
- The fascia lata (hypo-erechoic line) is superficial to all these structures; it is more dense laterally and covers the sartorius muscle.

Technique

- The in-plane approach allows the needle shaft and tip can be visualized distinctly, but some clinicians may still prefer the out of plane approach, because it the technique is similar to the nerve stimulation approach. The in plane technique requires the needle to be introduced on the lateral side of the probe aiming medially towards the lateral aspect of the femoral triangle, advancing over the top of the iliopsoas muscle. The needle must penetrate the fascia iliaca at this point.

- Injection of a small amount of LA will expand the femoral triangle and often enhances the view of nerve and the fascia iliaca.
- Observe the spread of LA and reposition the needle if required to aid the spread around the nerve.

Ultrasound-guided femoral nerve block: tips for success

- Enlarged inguinal lymph nodes may resemble the femoral nerve in cross-section on a single level scan. Scan proximally and distally in the inguinal region and trace the path of the femoral nerve.
- The anterior circumflex femoral artery may dissect the femoral nerve area and limit the spread of LA to all parts of the nerve.
- The posterior division of the femoral nerve, which innervates the quadriceps muscles is most commonly located on the posterior lateral aspect of the femoral triangle. Make this your first point of injection.

Sciatic nerve block

Indications

Above and below knee amputations, in combination with femoral nerve block. Anterior, posterior, inferior, and lateral approaches have been described for sciatic nerve blockade, but the most commonly used is the posterior (Labat) approach.

Landmarks

- Position the patient in the lateral decubitus position with a slight forward tilt.
- Position the foot on the side to be blocked over the dependent leg so that twitches of the foot or toes can be noted.
- Palpate and mark the posterior superior iliac spine (PSIS) and the greater trochanter. Connect these two points with a straight line and drop a 5-cm perpendicular line from the midpoint. This point should intersect a line drawn between the greater trochanter and the sacral hiatus.

Technique

- Press firmly on the gluteus muscle to decrease the skin-nerve distance with the palpating hand.
- Introduce a 100-mm, 22G needle perpendicular to the skin.
- The nerve stimulator should be initially set to deliver 1.5mA current (2Hz, 100μs). On initial needle advancement, twitches of the gluteal muscles are observed and indicate that the needle position is still too shallow.
- As the needle is advanced, the gluteal twitches disappear and a sciatic nerve response of plantar flexion (tibial nerve) of the foot should be sought. This is normally found at a needle depth of 5–8cm.
- Gradually decrease the stimulating current is gradually decreased until twitches are still seen or felt at 0.5mA.
- Slowly inject 15–20mL LA.

Sciatic nerve block: Tips for success

- Intraneural needle placement is relatively common with sciatic nerve blocks, so pay particular attention to avoid excessive injection pressure.

- Take care not to exceed the maximum dose of LA when performing combination blocks.
- Avoid adrenaline containing injectate near the sciatic nerve
- If no stimulus is elicited, then move the needle further along the perpendicular line.
- If dorsiflextion or eversion (common peroneal nerve) of the foot is elicited, move the needle slightly medially.

Ultrasound-guided sciatic nerve blockade

Sciatic nerve blockade in the subgluteal region is convenient and easily accessible. The nerve lies within a palpable groove in this location and is more superficial than in the gluteal region (See Fig 6.7).

Preliminary scan

- Place the patient in the lateral position with the block limb uppermost. The midpoint on the line between the greater trochanter lateral and the ischial tuberosity medial indicates the sciatic nerve location.
- Scan at this point along the line, preferably with a curved transducer to obtain a transverse image of the sciatic nerve. Identify the gluteus maximus muscle immediately underneath the layer of adipose tissue of varying thickness. The ischial tuberosity appears medial and the greater trochanter lateral on the ultrasound image.
- The nerve appears as a hyperechoic structure between the muscle planes of gluteus maximus (superficial) and quadratus femorus (deep) at a depth of about 5cm.
- The shape of the nerve changes from circular in the mid thigh region to triangular in the subgluteal region

Technique

- An in-plane approach is usually used for thinner patients when sciatic nerve is not too deep and needling angle to the nerve is not to steep.
- If nerve is deeper than 6cm, then often clinicians prefer an out of plane approach. Introduce a 100-mm 22G needle perpendicular to US beam and confirm correct needle placement with nerve stimulation.
- Inject 20–30mL LA while observing the spread around the nerve.
- Reposition your needle if required to surround the nerve with LA.

Ultrasound-guided sciatic nerve block: tips for success

- Use a nerve stimulator to help with the location of a sciatic nerve if in doubt about the US image.
- If you struggle to locate the sciatic nerve, turn the patient prone and scan longitudinal along the nerve axis. Often the nerve will appear as a hyperechoic white band across the US image, then turn the probe 90° to view the nerve in cross-section.
- For above knee amputations the sciatic nerve should be blocked at this level or more proximal to ensure that posterior cutaneous nerve of the thigh (a branch of the sciatic nerve) is incorporated.

Popliteal block

Indications

Below knee, forefoot, and toe amputations.

Landmark

- The sciatic nerve in the popliteal fossa is bordered superolaterally by the long head of the biceps femoris muscle and superomedially by the semimembranosus and semitendinosus muscles.
- The sciatic nerve branches into the common peroneal nerve and the tibial nerve at variable location along its course in the thigh. There are posterior, lateral, and supine approaches.

Technique (posterior approach)

- Position the patient prone and mark the popliteal triangle, which is bounded by the popliteal crease at the base, biceps femoris laterally and semimembranosus medially.
- Draw a line from the apex of the triangle to the middle of the popliteal crease.
- The needle entry point is 6–8cm along this line above the crease and 1cm laterally.
- Insert a 50-mm, 22G needle in a slight proximal direction and identify the tibial component by stimulation, which produces with plantar flexion of the foot. Slightly more laterally stimulation of the peroneal (fibular) component produces dorsiflexion and or eversion of the foot.
- Inject 10–15mL at each of nerve locations or a single injection of 20–30mL if both nerves can be stimulated with only small needle movements.

Popliteal block: tips for success

The sciatic nerve at this point is surrounded by a sheath, similar to the brachial plexus sheath. If the LA is deposited outside the sheath, the onset time for the block may be prolonged or a partial block may develop.

Ultrasound-guided popliteal blockade:

The success rate dramatically improves with the use of US for this block, because of the variable division of the sciatic nerve. The popliteal fossa easily allows a bolus of LA to spread along various fascial planes away from the nerves. The use of an US helps to manipulate the spread around the nerves under direct vision.

Preliminary scan

- The patient can be positioned prone, lateral or stay supine with leg raised with assistance for this **in plane** lateral approach.
- Place a 6–9MHz mid-frequency US probe transversely in the popliteal crease, above the popliteal crease, to obtain a cross-sectional view of the popliteal artery. The femur is deep to the artery and the popliteal vein is superficial and slightly lateral to the artery. The vein is easily compressed with the pressure from the transducer. Medial is the

semitedinosus and semimembranosus muscles and lateral the biceps femoris muscle .
- The tibial branch of the sciatic nerve is usually located superficial and lateral to the popliteal vein and often appears as a hyperechoic structure.
- Scan proximally and look for the peroneal branch joining the tibial branch from the lateral aspect. It is often necessary to angle the transducer caudally to enhance nerve visibility. Scan the nerve proximally and distally to assess the point of bifurcation.

Technique
- Note the depth of the nerves on the ultrasound image and at the same depth introduce a 100-mm 22G needle perpendicular to the probe from the lateral aspect of the leg.
- Take care not to go through the tendons of biceps femoris and vastus lateralis muscles. This approach will allow the needle to be introduced into the ultrasound image at 90° and maximize needle visibility. Advance the needle towards the nerves from lateral to medial.
- Deposit and manipulate the LA (20–30mL) to spread around both nerves; scan along the nerve proximally and distally to check longitudinal LA spread.
- Multiple needle movements and 'hydrodissection' with LA may be required to completely surround the nerves.

Ultrasound-guided popliteal block: tips for success
- Block the nerves below the sciatic bifurcation for a quicker onset time.
- Angle the transducer caudally to enhance nerve visibility.
- The popliteal vein lies in very close proximity deep to the tibial component of the nerve. Reduce probe pressure to identify the vein and aspirate to avoid unintentional intravascular injection.
- Plantar flexion and dorsiflexion of the foot will move the two nerve components in opposite directions (seesaw sign) and can aid in nerve identification.
- Avoid needling through the tendons of biceps femoris and vastus lateralis muscles, because it will anchor the needle shaft and limit needle tip movement, thus limiting your ability manipulate the spread of LA. It is also very uncomfortable for the patient.

Lumbar plexus block

Indications

Above knee amputation, femoral endartrectomy, femoral-popliteal bypass surgery and EVLA. The lumbar plexus block is a useful alternative for all unilateral vascular procedures and may be used in conjunction with a sciatic nerve block for postoperative analgesia. This is a deep block and the same cautions regarding anticoagulation for neuraxial blocks must be applied.

Landmarks

- Position the patient in the lateral decubitus position with a slight forward tilt. The foot on the side to be blocked should be positioned over the dependent leg so that twitches of the patella can be seen easily.
- Identify the PSIS and the line joining the iliac crests (Tuffier's line).
- Draw a line parallel to the spinous processes passing through the PSIS and mark the point where it crosses Tuffier's line. This is the needle entry point and should be about 4cm away from the midline.

Technique

- Fingers of the palpating hand should firmly press against the paravertebral muscles to stabilize the landmark and decrease the block distance.
- Insert a 100–150-mm needle at 90° to the skin. As the needle is advanced, local twitches of the paravertebral muscles are obtained for the first few centimetres.
- Contact with bone normally identifies transverse process of L4 and slight caudal or cranial needle redirection required. The needle is then advanced further until twitches of the quadriceps muscle are obtained (usually at the depth of 6–10cm).
- Inject 20–30mL of LA slowly with frequent aspiration to exclude inadvertent intravascular injection.

Ultrasound-guided lumbar plexus block:

US can be extremely useful to help locate the correct landmark (transverse process of L4) for a lumbar plexus block. The midline can be correctly identified by marking the spinous processes and the level of L3/L4 can be determined by scanning the spine. A curved array probe also helps to identify the muscles around the lumbar plexus. Most superficially lie the erector spinae muscles, then the quadratus lumborum muscles, deep and lateral to the L3 transverse process. The psoas muscle lies deep to the transverse process and the lumbar plexus is located one third depth into the psoas muscle.

Lumbar plexus block: tips for success

- Always use spinous processes to accurately determine the midline.
- Position the patient carefully to avoid spinal rotation.
- If hamstrings are stimulated then needle is too medial or caudad.
- Absolutely avoid medial needle angulation, which may lead to intrathecal or epidural injection.
- Care must be taken not to exceed LA maximum dosages when combining with a sciatic nerve block.
- Lumbar plexus block carries a higher risk of LA toxicity because of its deep location and the vicinity of well perfused muscles. Avoid high concentrations of long-acting LA.
- Do not seek signs of stimulation at currents <0.5mA. Dural sleeves envelop the roots of the lumbar plexus and motor stimulation at a low current may indicate placement of the needle inside a dural sleeve, with consequent unwanted epidural or spinal anaesthesia.

Further reading

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Pain management

Introduction

- Pain is common after vascular surgery, especially after open aortic procedures, thoracotomy, lower limb revascularization, or amputation.
- Specific considerations for pain management in the vascular patient include:
  - Age.
  - Common co-existing diseases especially IHD, hypertension, cerebrovascular disease, renal disease, respiratory disease, and diabetes.
  - The administration of antiplatelet or anticoagulant drugs in the perioperative period may dictate whether neuroaxial analgesic techniques can be performed safely.
  - Patients with critical limb ischaemia usually have longstanding pain and be receiving moderate to high doses of opioids before surgery.
  - Severe pain after vascular surgery stimulates the sympathetic nervous system. This increases myocardial oxygen demand and predisposes to myocardial ischaemia; it also increases blood coagulability and the propensity to arterial or venous thrombosis. Peripheral vasoconstriction associated with sympathetic stimulation can decrease distal perfusion after lower limb arterial reconstruction.
  - Hypertension or hypotension associated with administration of anaesthesia and pain relief may result in bleeding, myocardial ischaemia or stroke.
  - Early neurological assessment is desirable after carotid surgery so drugs, which cause excessive sedation should be avoided.
- Effective pain therapy in the vascular patient should also aim to attenuate the sympathetic nervous responses to surgery.

Clinical assessment of pain

- Measures of pain, drugs, and CNS sensitization provide complementary information on acute and chronic pain.
- Visual analogue, numerical rating, or other pain scores are widely used in clinical practice. However, they are subjective, and show inter- and intra-individual variation.
- Analgesic drug consumption is a surrogate marker of pain. However, the response to analgesics varies widely between patients because of

pharmacokinetic, pharmacodynamic physiological, and psychological factors.
- Better, more objective measures of pain include quantitative sensory testing of specific fibres. Examples include heat-induced pain threshold and supra-threshold testing of skin to predict the intensity of post-operative pain.

Acute pain services

- Pain management should be performed in conjunction with an acute pain service responsible for staff education, pain management protocols, prescribing, monitoring, and safety.
- The aim of pain therapy is to provide sufficient pain relief to allow movement, deep breathing, and effective coughing, in order to reduce perioperative complications. The achievement of pain relief at rest is not sufficient.
- Treatment of post-operative pain has traditionally relied on the use of morphine.
- Awareness of serious side effects such as opioid induced hyperalgesia (see 🕮 Physiology of pain, p. 81) and chronic persistent pain has led to the increased use of other classes of drugs.

Systemic analgesia

- Multimodal analgesia is the combination of different classes of drugs, often administered at a lower dose than normal, and having additive or synergistic effects. It is useful for all types of acute pain.
- The advantages of multimodal analgesia are:
  - Improved analgesia.
  - Improved recovery and function.
  - Reduced incidence of adverse effects.
- Analgesic therapy consists of simple oral medication, morphine-based regimens, RA, and anti-hyperalgesic modulators.

Non-opioid analgesics

- Paracetamol is an effective analgesic for acute pain, given po or IV.
- Analgesia is improved when paracetamol is combined with NSAIDs, tramadol or codeine.
- NSAIDs inhibit the synthesis of prostaglandins in wounds and spinal cord.
- Paracetamol and NSAIDs have an opioid-sparing effect after major surgery. The incidence of adverse effects is also reduced.
- Adverse effects of NSAIDs are significant and limit their use, especially in the presence of renal impairment, hypovolaemia, and hypotension.
- NSAIDS should be used sparingly in vascular anaesthesia.

Opioid analgesics

- Morphine is the main analgesic used for severe post-operative pain.
- Morphine may be administered as intermittent intramuscular/IV injection or as PCA.
- Intermittent sc morphine injections are as effective as intramuscular injections and are better tolerated by patients.

- The titration of opioids for severe acute pain is best achieved using intermittent IV injection because:
  - The onset of analgesia is quicker.
  - It allows rapid titration of dose to effect.
  - It avoids the uncertainties of intramuscular drug absorption in postoperative patients in whom peripheral blood flow may vary because of hypovolaemia or vasoconstriction.
- There is a high risk of respiratory depression if continuous intravenous infusions of morphine are used outside a HDU environment.
- Intravenous opioid PCA systems provide better analgesia than conventional intramuscular opioid regimens.
- The use of background infusion during PCA does not improve pain relief.
- PCA infusion systems must incorporate antisyphon valves; an antireflux valve should also be incorporated if a non-dedicated intravenous line is used.
- Drug concentrations within a hospital should be standardized.

Neuraxial administration of opioids

- Opioids can be administered via the intrathecal and epidural routes.
- Intrathecal morphine (0.1–0.2 mg) improves analgesia and has an opioid-sparing for up to 24h after surgery.
- Neuraxial administration of bolus doses of hydrophilic opioids, such as morphine carries an increased risk of delayed sedation and respiratory depression compared with lipophilic opioids such as fentanyl.
- Intrathecal morphine at doses ≥300micrograms increases the risk of delayed respiratory depression.
- Extended release epidural morphine uses a liposomal carrier to provide prolonged analgesia without the need for an in-dwelling catheter.
- Extended release epidural morphine provides analgesia for up to 48h.
- Doses of extended release epidural morphine should be limited to 5, 10, or 15mg depending on age, and ASA status to minimize the risk of respiratory depression.

Regional techniques

Neuraxial analgesia

The benefits of thoracic epidural analgesia (TEA) are greatest in patients with decreased physiological reserve or who are undergoing higher risk procedures.

Advantages of TEA

Effective TEA has the following advantages after major surgery:

- Better post-operative pain relief compared with parenteral opioid.
- Improved ischaemia-induced left ventricular dysfunction when TEA includes the cardiac sympathetic outflow (T1–T4).
- Better preservation of post-operative respiratory function. This improves oxygenation and reduces the incidence of respiratory complications.
- Reduced duration of tracheal intubation and mechanical ventilation.

- Improved recovery of GI function allowing early feeding after abdominal surgery.
- Nutritional support in the presence of TEA preserves total body protein content after upper abdominal surgery.

Disadvantages of TEA

- The adverse effects of TEA include pruritus, urinary retention, and motor block.
- Hypotension is common because of blockade of sympathetic vasoconstrictor fibres.
- Neurological complications (see 📖 Complications of neuraxial analgesia in relation to vascular surgery, p. 307) are rare, but serious.
- If TEA is not sited at a thoracic interspace at the mid-point or above in relation to the surgical incision, the benefits are negated. The adverse effect profile changes with an increased incidence of motor block.
- In patients undergoing peripheral vascular reconstruction, there is no difference in cardiac morbidity or operative mortality from epidural anaesthesia compared with PCA morphine.
- TEA is safe to use on general hospital wards, as long as it is supervised by an anaesthesia-based pain service with 24-h medical staff cover and patients are monitored by well-trained nursing staff.
- TEA is best administered as a combination of LA and opioid to maximize pain relief and minimize the adverse effects of each.
- The use of PCEA allows self-titration of pain relief (see Table 6.5).
- Maximum infusion rate of levobupivacaine 0.125% should not exceed 20mL/h; maximum dose of ropivacaine should not exceed 18.75mg/h.
- If high doses are required, consider that the epidural may be ineffective and other analgesics may be needed.
- Adrenaline (2.5μg/mL) added to the epidural mixture improves pain relief and reduces drug requirement by causing local vasoconstriction or through binding to α2-adrenergic receptors in the spinal cord.
- Clonidine (intermittent epidural bolus dose of 1–1.5μg/kg) is an α-2 adrenergic agonist acting on receptors in the substantia gelatinosa of the dorsal horn and may reduced mechanical hyperalgesia. It is best administered via the spinal or epidural route to minimize side effects such as hypotension, bradycardia, and sedation.

Table 6.5 Typical drugs, concentrations, delivery rates, and PCEA bolus doses for thoracic epidural analgesia. Addition of an opioid to the local anaesthetic allows lower bolus doses and infusion rates

Epidural drug	Concentration (% or micrograms/mL)	Rate (mL/h)	Bolus (mL)
Levobupivacaine	0.1–0.125%	3–20	3–6
Ropivacaine	0.15–0.20%	6–14	2 – 5
Fentanyl	2–5micrograms/mL	As above	n/a
Diamorphine	25–50 micrograms/mL	As above	n/a

Complications of neuraxial analgesia in relation to vascular surgery

- Neurological complications of neuroaxial analgesic techniques are discussed in 📖 Regional anaesthesia in vascular surgery, p. 274.
- Paraplegia may occur after thoracic aortic surgery due to interruption of the spinal arterial supply (📖 Open aortic aneurysm repair, p. 350).
- The risks of epidural haematoma in the vascular patient may be minimized by:
  - Inserting epidural catheter and testing block in an awake patient.
  - Avoiding heparin for at least 1h after insertion of an epidural.
  - Using low-concentration LA infusion to preserve motor function.
  - Maintaining a high index of suspicion and providing regular neurological assessment during the post-operative period.
  - Early referral for specialist opinion if pain or exaggerated motor block occur.

Perineural blockade techniques

- Perineural block for the upper limb include interscalene, supraclavicular, infraclavicular, and axillary blockade. For the lower limb, femoral and sciatic blockade are useful.
- A perineural catheter infusion can be used to provide analgesia for 3–7 days.
- Blocks may be performed by the anaesthetist using US or the surgeon under direct vision (e.g. sciatic nerve sheath catheter during lower limb amputation).
- Blocks performed by the anaesthetist using US guidance are more likely to be more successful compared with localization using a peripheral nerve stimulator.
- Nerve blocks may be combined with sedation, general or spinal anaesthesia where appropriate.
- Blocks must be performed before GA or spinal anaesthesia.
- Continuous peripheral nerve blockade provides better post-operative analgesia compared with opioid-based analgesia. It reduces opioid use as well as nausea, vomiting, pruritus and sedation.
- Continuous perineural analgesia provides equivalent analgesia to epidural analgesia and the incidence of side effects, such as urinary retention, motor block and immobility is lower.
- Either an electronic or elastomeric pump can be used to infuse LA.
- Typical dosages are levobupivacaine 0.125% 8mL/h or ropivacaine 0.2% 5mL/h.

Treatment strategies for chronic post-surgical pain

Pre-emptive analgesia

- In the 1980s, Wall proposed that interruption of the transmission of noxious inputs into the spinal cord before surgical incision could prevent the establishment of central sensitization, resulting in reduced pain intensity and lower analgesic requirements even after the analgesic effects had worn off. This was termed pre-emptive analgesia.
- Preventive analgesia was defined by Kissin as: 'an antinociceptive treatment that prevents establishment of altered central processing of afferent input from injuries'. Although successful in experimental

animals, it has been difficult to demonstrate that preventive analgesia strategies with LA alone are effective in clinical practice.
- A meta-analysis of 93 studies comparing interventions given either before or after surgical incision also found no difference in pain scores over 24h when drugs were administered by the epidural route.
- This may be because wound inflammation lasts longer than the pharmacological effect of drugs given either before or after incision, initiating re-sensitization of the spinal cord.
- For pre-emptive analgesia to be effective, it is necessary to reduce CNS plasticity with a combination of regional anaesthetic techniques and anti-hyperalgesic modulators (antagonists such as ketamine and spinal alpha-2-adrenoceptor agonists).

Specific early interventions

There is some evidence that specific early analgesic interventions may reduce the incidence of chronic pain after surgery. Analgesia may be achieved by reducing excitatory transmission (e.g. LA, ketamine) or by either enhancing inhibition (e.g. opioids, clonidine, antidepressants). The data available are not specific to vascular surgery.

Local anaesthetics

Epidural analgesia initiated before thoracotomy, and pre-incisional paravertebral block before breast surgery have been shown to reduce pain for several months.

Ketamine

The addition of IV ketamine to continuous perioperative epidural analgesia has been shown to reduce persistent pain up to 1yr after colonic surgery.

Gabapentin

The perioperative use of gabapentin or mexiletine have been shown to reduce the incidence of neuropathic pain at 6 months after mastectomy. Gabapentin and its analogues improve opioid analgesia, reduce opioid requirements and tolerance, and relieve anxiety and have a better side effects profile than ketamine.

Phantom limb pain

- Pain after amputation may be directly from the surgical amputation site itself (stump pain) or phantom pain. Phantom limb pain (PLP) is a chronic, neuropathic pain often described as burning, shooting or cramping, felt to be arising from the limb which has been amputated. It is often resistant to opioids. Phantom sensations refer to persistent non-painful feelings that the limb is still present; analgesic drugs are not usually required and sensations usually resolve over several months. Stump pain, phantom pain and phantom sensations should be actively differentiated because the aetiologies, assessment and treatments differ (Table 6.6).
- Some studies suggested that pre- and post-operative epidural analgesia reduced the incidence of PLP, but the largest and most recent data have shown no effect.

Table 6.6 Assessment of pain or sensation after amputation

	Assessment terms	Comments
Stump pain	Intensity Site Duration Type (tender, cramping, shooting)	Usually early post-operative period Exacerbated by movement Often responds to opioids
Phantom sensation	Includes sensations of movement, itching, something touching, abnormal shape or position, heat or cold, and 'electric' sensations, such as paraesthesia	Usually resolves spontaneously over months
Phantom pain	Intensity Site Frequency Duration Description (shooting, stabbing, cramping pain)	Often intermittent Difficult to treat once established

- Continuous regional blockade via nerve sheath catheters provides effective post-operative analgesia after amputation, but may not prevent PLP.
- Once established, PLP is difficult to treat. Behavioural and pharmacological interventions should be used. Sensory discrimination training and motor imagery reduce chronic PLP
- First line pharmacological treatment is with conventional analgesics (NSAIDs, weak opioids or combinations).
- Gabapentin in increasing doses 300–900mg tds is useful. Pregabalin is an alternative.
- Calcitonin, morphine, ketamine, amitriptyline, and tramadol are sometimes used for PLP, although there is little evidence for these drugs.
- Examples of behavioural treatment include prosthesis use, application of electrical stimuli to the stump, and use of a mirror to perceive the intact, moving hand.

The opioid-tolerant patient

- Patients with critical limb ischaemia may have been taking high doses of opioids and be opioid-tolerant, so that increased doses are required to provide effective analgesia.
- Opioid-tolerant patients report higher pain scores, and have a lower incidence of opioid induced nausea and vomiting.
- Ketamine improves pain relief after surgery in opioid-tolerant patients (see 🕮 Anti-hyperalgesic drugs, p. 310).
- PCA settings may need to include a background infusion to replace the usual opioid dose and a higher bolus dose.

- In patients with escalating opioid requirements and increasing pain the possibility of the development of both tolerance and opioid-induced hyperalgesia should be considered.

Anti-hyperalgesic drugs

- Anti-hyperalgesic drugs may not necessarily reduce post-operative pain but reduce the extent of 2° hyperalgesia weeks and months after surgery by reducing perioperative spinal cord sensitization.
- Anti-hyperalgesics should be considered in patients deemed to be at risk of chronic pain.
- Ketamine is an NMDA antagonist associated with psychomimetic side effects such as confusion and hallucinations in anaesthetic doses (2mg/kg).
- Ketamine reduces post-operative pain in opioid-tolerant patients.
- Intravenous ketamine (0.5mg/kg) as an adjunct to epidural block, peripheral nerve block, morphine or NSAIDs may marginally improve postoperative pain, but does not reduce side effects.
- One study showed that when combined with epidural bupivacaine and morphine for abdominal surgery, an IV bolus of ketamine (0.5mg/kg), then infusion of ketamine for 48h was associated with less PCA morphine use, improved pain relief, and reduced mechanoallodynia 6 months later.
- Pregabalin and gabapentin bind to the $\alpha 2$ subunit of voltage-gated calcium channels in the spinal cord and brain Both drugs are used for seizures and neuropathic pain.
- Pregabalin has higher bioavailability than gabapentin; side effects include dizziness, blurred vision, headache, and sedation.

Further reading

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Chapter 7

Monitoring of the vascular patient

General principles of monitoring

> You are what you repeatedly do; then excellence not an art but a habit
>
> Aristotle (384–322 BC)

Many vascular surgical procedures are complex. Patients requiring vascular surgery are frequently elderly with significant co morbidity and markedly diminished physiological reserve. Successful outcome depends on close collaboration between the surgical, anaesthetic, and intensive care teams. Meticulous attention to detail is crucial to minimize morbidity and mortality.

Aims of monitoring

The broad aims of monitoring are to detect and record physiological changes during and after surgery, to inform and guide medical, surgical, and anaesthetic management.

Types of monitoring devices

- A wide range of bedside monitoring devices are available. The devices most relevant to vascular anaesthesia are used to monitor the cardiovascular, haematological and neurological systems.
- Different monitors are required for specific procedures, depending on surgical techniques used, and patient comorbidities.

Use of monitoring

- It is vital that the vascular anaesthetist has a detailed understanding of the working principles, indications and limitations of the monitoring equipment they use.
- The anaesthetist must be able to process, interpret, and promptly correct the subtle physiological perturbations detected by a range of monitors.
- This is considered a core skill for the vascular anaesthetist and allows correct clinical decision-making.
- The advantages, disadvantages, and pitfalls of monitoring systems commonly used during vascular surgery are detailed in this section.

Monitoring the circulation

The selection of intraoperative monitoring techniques necessitates balancing the risks and invasiveness of a device with its ability to detect subtle changes indicating significant physiological events. In modern anaesthesia the traditional approach of monitoring parameters and reacting to changes is being supplemented by evidence for use of monitoring to assist in manipulating physiological systems to optimize cardiac function and tissue perfusion. The ideal monitor should:

- Be accurate under all conditions of use.
- Give reproducible results.
- Be operator independent

- Provide a fast and continuous display of a parameter.
- Be of benefit to the patient

Electrocardiography

Electrocardiography is the measurement and display of the electrical depolarization of myocardial cells detected as potential differences between paired skin electrodes known as leads. Electrodes are placed in predetermined positions around the thorax giving combinations of leads. A 12-lead configuration is used as a preoperative diagnostic test whilst 3- or 5-lead configurations are used for continuous intraoperative monitoring. The ECG demonstrates heart rate and rhythm, and the morphology of the waveform can be assessed to look for evidence of ischaemia, infarction, drug toxicity, or electrolyte disturbances. The ECG gives no information about the functional performance of the heart; it is possible to have normal ECG morphology with no cardiac output.

Advantages

- Non-invasive.
- Technically simple.
- Continuous 'real-time' monitoring.

Disadvantages

- Dependent on correct electrode placement.
- Subject to interference.
- Changes can be subtle and difficult to interpret.
- ECG changes appear late in the 'ischaemic cascade'.

Non-invasive blood pressure (NIBP)

Current automated systems utilize one cuff that inflates above systolic pressure, and deflates either continuously or in a step-wise manner. The cuff pressure and arterial pressure wave are detected by transducers in the cuff. The point where the rate of increase in the size of measured oscillation is maximal is systolic pressure; the point of maximal rate of decrease in size of oscillation, diastolic pressure. MAP is when the oscillations are maximal.

Advantages

- Technically easy.
- Reasonably accurate.
- Low risk.

Disadvantages

- Intermittent.
- Decreased accuracy in the presence of haemodynamic instability.
- Efficacy reliant on regular heart rate and rhythm.
- A correctly sized cuff both in length and circumference is required for an accurate measurement (20% larger than the circumference of the extremity and covering two thirds of the upper arm).
- *Can be uncomfortable for awake patients:* initial reading requires high inflation pressures.
- Complications include bruising, oedema, or nerve injury by cuff compression

Invasive arterial blood pressure (IABP)

This is the 'gold standard' of BP measurement and is especially useful where rapid changes in BP are anticipated due to cardiovascular morbidity, large fluid shifts, or the effects of drugs:

- Pressure is determined by the measurement of fluctuations in a continuous fluid column between arterial blood and an electronic pressure transducer.
- A short, parallel-sided cannula is placed into a non-end artery. This is connected to the pressure transducer by stiff, bubble-free tubing primed with pressurized plain or heparinized 0.9% saline.
- The fluctuations of column of fluid are converted to an electrical signal by a transducer which is most commonly a strain gauge.
- Transducer must be placed level with the patient's right atrium.
- Output data from gauge is represented on a monitor, both numerically and as a waveform giving beat-to-beat measurement of BP. The shape of the waveform can analysed clinically (e.g. the shape of the waveform, position of the dichrotic notch and 'swing' can give information about filling status and cardiac output) or electronically.
- Cardiac output devices, such as the pulse contour cardiac output (PICCO) and lithium dilution cardiac output (LiDCO) rely on such information to derive cardiac output. Damping is used in the measurement to counteract resonance of natural frequencies within the system. An under-damped system will show a rapid response but will overshoot true values, whilst an over-damped system will result in a slower response but no overshoot.

Advantages

- Accurate beat-to-beat measurement.
- Further information gained from waveform assessment.
- Arterial blood gas sampling.

Disadvantages

- Haemorrhage.
- Infection.
- Arterial damage.
- Distal ischaemia.
- Air embolus.
- Inadvertent injection of drugs.
- Accuracy may be dependent upon site of arterial cannula.

Central venous cannulation (CVC)

Pressure measurement in a central vein (CVP) represents the filling pressure of the right ventricle and allows assessment of intravascular volume status and right heart performance. Measurements are affected by many factors including heart function, pulmonary disease, ventilation pressures, and posture. Using CVP alone, it can be difficult to differentiate changes in volume or compliance within the cardiovascular system, but in combination with clinical information, such as response to fluid challenge it is possible to better estimate filling status. Assessment of the waveform morphology can give further information about pathological conditions, such as tricuspid regurgitation or atrial arrhythmia.

Advantages
- Assess volume status of the right side of the heart.
- Facilitates administration of drugs centrally.
- Allows central venous blood sampling.

Complications
- Haemorrhage.
- Pneumothorax.
- Haemothorax.
- Chylothorax.
- Infection.
- Air embolism.
- Guide wire embolism.
- Arrhythmias.
- Thrombosis.

Cardiac output monitoring

Goal-directed therapy (GDT) is a term used to describe the assessment of parameters, such as cardiac output, O_2 delivery, and indicators of volume status to guide administration of fluids, vasopressors, and inotropic drugs. There is evidence that GDT improves outcomes when used before, during, and after major surgery. As novel methods of measuring cardiac output become more reliable and accessible, they will play an important role in the intraoperative and post-operative management of patients undergoing vascular surgery.

Clinical

Standard monitoring parameters, such as heart rate and BP correlate poorly with changes in cardiac output. One useful sign is skin perfusion, as assessed by capillary refill time or 'warmth' of limbs. Reduced cardiac output can be detected by evidence of reduced end organ perfusion, and may manifest itself as confusion or reduced conscious level, poor urine output, and a rising serum lactate. There may be evidence of cardiac ischaemia.

Pulmonary artery catheter (PAC)

Traditionally, the gold-standard of cardiac output (CO) monitoring, the PAC utilizes Fick's principle to measure CO. In the original description of Fick's principle, arterial and mixed venous samples were obtained to calculate arteriovenous O_2 difference and oxygen consumption assessed by spirometry.

Modern PACs incorporate a thermistor at the tip and measure temperature change in response to proximally injected cold saline, or to blood heated by passage over a heating coil in the proximal part of the catheter.

Measurable PAC data
- Right atrial pressure.
- Right ventricular pressure.
- Pulmonary artery pressure.
- Pulmonary artery wedge pressure.
- Cardiac output.
- Mixed venous blood gases, PaO_2, SvO_2, and lactate.

The value of PAC's remains controversial, and there is an ever increasing choice of devices to measure CO offering a compromise between invasiveness and accuracy. Many different devices have been produced utilizing a variety of different technologies, but comparative data is difficult to assess especially in a critically ill or haemodynamically unstable population. Assessment of these varying devices is also hampered by the fact that the reference technique of thermodilution itself is also subject to variation when compared with laboratory techniques, such as aortic electromagnetic transit time flowmetry.

Fick partial CO_2 rebreathing method

The Fick principle can be applied to intubated and ventilated patients by using physiological CO_2 as the marker:

$$Q = VCO_2/(CaCO_2 - CvCO_2)$$

where Q is cardiac output, VCO_2 is amount of CO_2 eliminated, $CaCO_2$ and $CvCO_2$ are arterial and venous CO_2 content. There are systems that integrate with ventilator tubing and consist of valves, reservoir tubing, and detectors of flow and CO_2 content. By comparing periods of rebreathing with baseline periods, the cardiac output can be estimated. Readings are intermittent and not instantaneous, and are affected by haemodynamic instability or pulmonary shunting. There is evidence of correlation with thermodilution techniques to within limits comparable with other techniques.

Pulse contour analysis

There are several devices available that calculate CO based on analysis of the arterial pressure waveform. They use a variety of methods including:

- Diastolic pulse contour analysis.
- Systolic pulse contour analysis .
- Model flow pulse contour analysis.
- Pulse power analysis.

These rely on complex algorithms. The stroke volume is derived from the area under the systolic part of the pressure curve. Some of these devices are periodically calibrated using dye dilution or thermodilution whilst others rely upon mathematical modelling. They give estimations of volume status and myocardial contractility. Studies comparing pulse contour analysis techniques with thermodilution techniques have shown reasonable agreement between values, even in critically ill patients. Results should be interpreted with caution in patients on high doses of vasopressors and those whose arterial pressure trace is poor. Monitors utilizing this technology include the $PiCCO_2$ monitor (Pulsion Medical Systems AG) and Flo Trac/Vigileo system (Edwards Life Sciences)

Lithium indicator dilution (LiDCO)

Dye dilution techniques for measuring cardiac output were first described in 1932, originally using indocyanine green, but were technically demanding, and required frequent blood sampling and analysis. Lithium was first described as a useful indicator in 1993 and has been extensively validated. A bolus dose of isotonic lithium chloride (0.002–0.004mmol/kg) is injected

via a central or peripheral venous catheter, whilst a pump attached to an arterial line draws blood over a lithium ion electrode at 4mL/min. Cardiac output can be calculated from the integrated curve and the dose of lithium injected using the formula:

$$\text{Cardiac output} = (\text{Lithium dose} \times 60)/[AUC\ (1 - PCV)]$$

where *AUC* is the area under the lithium concentration curve and *PCV* is packed cell volume, to allow for the fact that lithium is distributed in the plasma. Clinically, the lithium dilution method is used to calibrate the system periodically (every 8h) with continuous reading provided by arterial BP trace analysis. The lithium dilution system has been studied in animals, children, and adults, and performs comparably with thermodilution using either central or peripheral lithium injection.

Limitations of LiDCO

- Not suitable for patients receiving therapeutic lithium as increased baseline lithium levels will cause overestimation of cardiac output.
- Some muscle relaxant drugs may cause lithium electrode accuracy to drift.
- Cardiac shunts distort the concentration curve.

Oesophageal Doppler monitoring (ODM)

A Doppler US probe placed in the oesophagus can measure blood flow velocity in the descending thoracic aorta. The cross-sectional area of the aorta is estimated based on the sex, age, and height of the patient. Velocity multiplied by area gives flow, which is then mathematically adjusted to allow for the 30% of cardiac output, which has already been distributed to supply the coronary, cerebral, and arm circulations. Further information is gained from analysis of the shape of the waveform. The systolic flow time corrected for heart rate (FTc) gives an indication of preload, and the peak velocity (PV) and mean acceleration (MA) of the systolic upstroke represent myocardial contractility.

- A 4MHz continuous wave Doppler transducer is inserted 35–40cm into the oesophagus.
- Characteristic blood flow signal is seen in descending thoracic aorta.
- Then:

$$V = (Df \times C)/(2ft \times \cos\theta)$$

where *V* is velocity of blood; D*f*, Doppler frequency; C, sound velocity in tissue; *ft*, transmitted frequency; θ, angle between US beam and flow direction (45°).

ODM interpretation

- Stroke distance equates to LV stroke volume in the thoracic aorta.
- LV stroke volume over time equates to CO.
- FTc is inversely proportional to systemic vascular resistance (SVR).
- Peak velocity is proportional to myocardial contractility.

See Fig. 7.1.

Advantages

- Technically simple relative to other methods.
- Non-invasive.
- Good indicator of cardiac output and volume status.

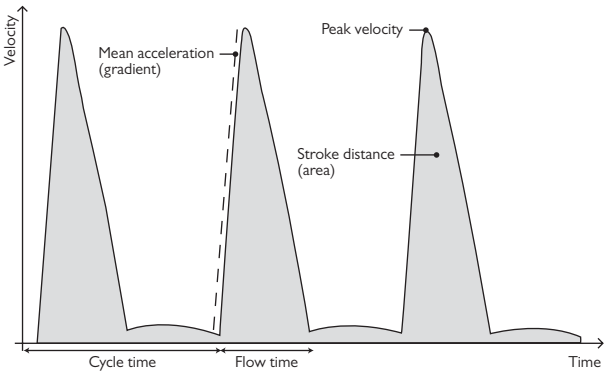

Fig. 7.1 The oesophageal Doppler waveform.

Disadvantages

- Only suitable for sedated or anaesthetized patients.
- Volume and flow readings are derived rather than measured.
- Assumes that a fixed percentage (30%) of CO is distributed to the coronary and cerebral circulations in all circumstances.
- Subject to probe movement errors.
- Inaccurate in certain circumstances:
  - Patients with aortic coarctation.
  - In the presence of an intra-aortic balloon pump.
  - Patients with thoracic aortic aneurysms.
  - When the aorta is cross-clamped.

The Cardio-Q ODM+ can also use an arterial line to record the pulse pressure wave. The monitor displays Pulse Pressure Variation enabling the clinician to assess fluid responsiveness and administer fluid to minimise respiratory swing.

Other recently introduced non invasive monitors which assess the state of the circulation such as the LiDCO Rapid or NICCOM Cheetah and may be considered but there is as yet little experience of their use in vascular surgical patients.

Transoesophageal echocardiography (TOE)

- A multi-mode endoscopic US probe is placed in the patient's oesophagus and is manipulated to provide views of the heart and surrounding structures. TOE is widely used in cardiac surgery
- TOE is contraindicated in patients with significant oesophageal pathology.
- It permits real-time assessment of volume status, structural defects, ventricular performance and valve lesions. To measure cardiac output, a Doppler assessment at a specific conduit (e.g. the aortic valve) will determine velocity of flow (velocity time index, VTI). Cross-sectional area (CSA) of the conduit can also be measured; stroke volume is the product of VTI × CSA.

- Detection of myocardial ischaemia is also possible using TOE. Systolic function is assessed by observing thickening and movement of ventricular walls during systole. Systolic wall motion abnormalities (SWMA) can indicate endocardial ischaemia, but can be difficult to interpret.
- There is growing evidence to support use of TOE and with published guidelines for training, intraoperative use, and indications. It is likely to play an increasing role in the perioperative care of high risk vascular surgery patients.

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Point of care coagulation monitoring

Point of care (POC) testing is the testing of blood at or near the patient's bedside. It provides timely information that facilitates real time decision making. The inevitable delay associated with conventional laboratory-based coagulation tests may mean that the results obtained are not suitable to guide therapy on a real time basis, particularly when the clinical scenario is rapidly changing, e.g. major haemorrhage. Consequently many blood products are administered on an empirical basis. In 2007 the Society of Cardiovascular Anesthesiologists Special Task Force on Blood Transfusion recommended that POC coagulation tests should be available to guide transfusion decisions. POC testing of coagulation facilitates targeted blood product administration and reduces the total number of transfusions given. Early treatment of evolving haemostatic defects is far more effective than late and/or empirical therapy. There is increasing evidence that access to POC testing of coagulation improves decision making and transfusion practice; this translates into improved patient outcomes and significant cost savings.

POC testing may have a role to play in the assessment of the perioperative bleeding risk in patients taking anti-platelet medication. POC monitoring of platelet function is likely to become crucial to the management of antiplatelet drugs in the perioperative period.

Intraoperative coagulation monitoring

Conventional coagulation tests

- Transfusion guidelines use the prothrombin time (PT), the international normalized ratio (INR), the activated partial thromboplastin time (APTT) the fibrinogen level and the platelet count as trigger thresholds.
- The APTT was developed to monitor heparin therapy and to investigate inherited coagulation defects such as haemophilia.
  The PT/INR was developed to monitor anticoagulant therapy with coumarins. These tests are performed at 37°C–'normal' test results run at this temperature may not be an accurate reflection of coagulation status if the patient is hypothermic.
- *APTT:* looks at activation of the 'intrinsic' coagulation cascade. It is performed by incubating plasma with partial thromboplastins, calcium and kaolin powder at a standardized pH. The endpoint is the formation of fibrin strands. APTT is prolonged by deficiencies of factors I, II, V, VIII, IX, XI, and XII. Multiple factor deficiencies tend to show a greater prolongation for a given factor level than single factor deficiencies.
- *PT:* studies activation of the 'extrinsic' coagulation cascade. It is performed by incubating plasma with tissue thromboplastin and calcium at 37°C at a standardized pH. The endpoint is the formation of fibrin strands. This test is sensitive to factors I, II, VII, IX, and X. Standardization of the PT for laboratory control of oral anticoagulant therapy is based on the responsiveness of one type of thromboplastin, measured by its international sensitivity index and conversion into the INR. Direct INR determination is performed by local calibration using plasma of certified levels of PT.
- FBC will give a platelet count, but give no information on platelet function.
- Fibrinogen levels are derived from the PT or as the Clauss fibrinogen and measured in g/L.

Limitations of routine coagulation tests to guide coagulation management in massive bleeding

Turn around time

- When there is active and ongoing blood loss, real time information is essential to direct replacement therapy in a timely manner. Laboratory-based test results are inevitably available only after a delay due to transportation, sample analysis, and result reporting. This may be at least 30min for a platelet count and up to 90min for a PT/INR and fibrinogen.
- POC monitors of haemostasis are increasingly used. Cuvette technology, such as the Coaguchek® and the Hemochron Signature® are available for POC testing of PT/INR, activated clotting time (ACT), and APTT. Although POCT of the ACT is routinely used in cardiac surgery to monitor heparin therapy, POC testing of PT and APTT for perioperative use is still undergoing validation. Preliminary results indicate that POC PT/INR results are reliable and produce similar results to central laboratory tests, but POC results of APTT still show marked variability.

Identification of the nature of the coagulopathy

- Perioperative bleeding is associated with multiple coagulation defects caused by haemodilution, consumption of clotting factors, fibrinolysis, hypothermia, and other metabolic derangements. These all may manifest as prolongation of the PT and APTT
- Conventional laboratory-based coagulation tests do not provide any information on the in vivo interaction of platelets and coagulation factors.
- The PT and APTT may be prolonged, even if thrombin generation is normal because reduced levels of procoagulant activity may be balanced by low levels of natural anticoagulants (e.g. antithrombin and protein C) for example in chronic liver disease.
- Laboratory-based tests do not measure the stability of the thrombus
- Conventional tests give no information about fibrinogen levels or the degree of fibrinolysis. Fibrinogen levels fall early with dilution and in massive haemorrhage. Excessive fibrinolysis is an important cause of bleeding in major trauma and complex cardiac, hepatic, and vascular surgery

Viscoelastic haemostatic tests: TEG® and ROTEM®

The traditional description of the intrinsic and extrinsic pathways of coagulation is obsolete. The current cell-based model of haemostasis emphasizes the role of platelets in intact thrombin generation and highlights the importance of the dynamics of thrombin generation influencing the quality and stability of thrombus formed. For effective haemostasis there must be sufficient thrombin generation (coagulation factors and platelets), adequate substrate (fibrinogen), and clot stability. Viscoelastic haemostatic tests measure changes in clot tensile strength over time and give information on the dynamics of clot formation (coagulation factor and anticoagulant activity), clot strength (platelets and fibrinogen), and clot stability (fibrinolysis and factor XIII)

General principles

- TEG and ROTEM measure clot tensile strength in a similar manner. In the TEG, whole blood (0.36mL) is placed into an oscillating cup into which a pin on a torsion wire is suspended. Once fibrin strands start to form, the motion of the cup is transmitted through the pin to a recording device to give the characteristic TEG trace (Fig. 7.2). In the ROTEM, the cup is stationary and the pin rotates and a similar trace is produced graphing the impedance of pin rotational movement that reflects clot characteristics (Fig. 7.3).
- The main parameters measured by both techniques are essentially the same (Fig. 7.4), but algorithms developed on one machine are not directly interchangeable to the other.

The cell-based model of coagulation describes haemostasis in three stages: initiation, amplification, and propagation. These three different phases of cell-based haemostasis are reflected by viscoelastic tests (Fig. 7.4):

- The R/CT corresponds to the initiation phase.
- K reflects the amplification phase.

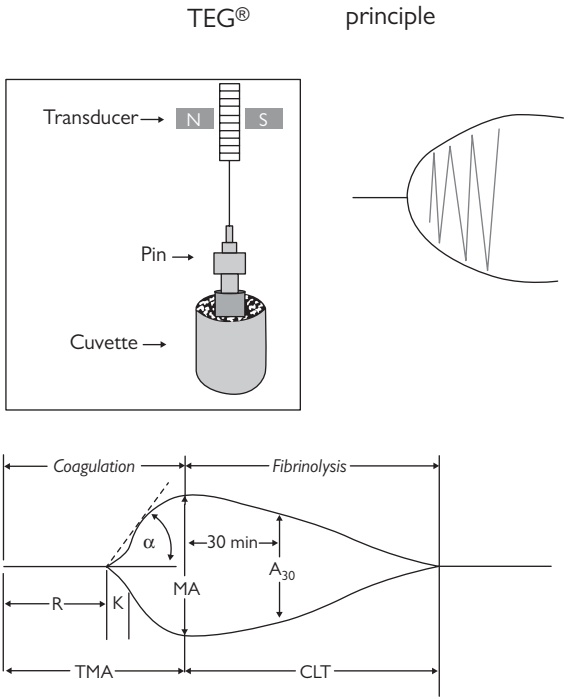

Fig. 7.2 TEG principles and trace.
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- The thrombin burst is reflected by the α angle. This is valuable information as the rate and peak of thrombin generation influences the final clot structure and stability.
- Reduced maximum aplitude/maximum clot firmness (MA/MCF) may indicate low platelet or low fibrinogen values.
- Low fibrinogen levels may cause bleeding. Both FIBTEM and TEG functional fibrinogen can be used to determine if levels of fibrinogen require treatment with fibrinogen concentrate or cryoprecipitate.
- Fibrinolysis is also readily identifiable on both assays by the clot lysis index, demonstrating rapid dissolution of the clot.

Thromboelastography

TEG®; Hemonetics corporation, Braintree, MA.

- The TEG originally used fresh native whole blood with no additional activator. Subsequent modifications have been developed to increase

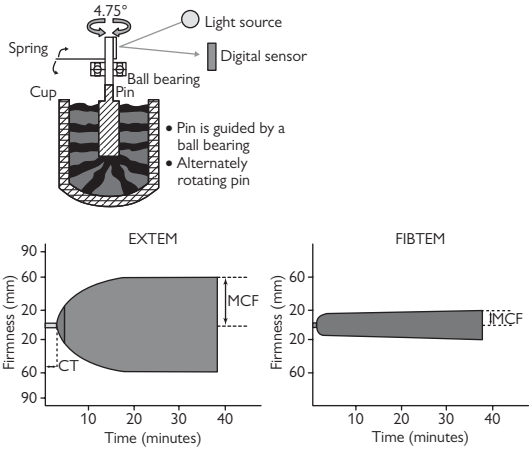
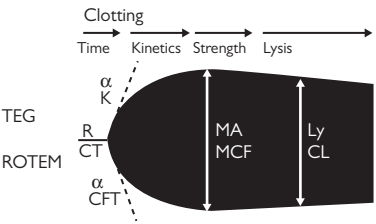


Fig. 7.3 ROTEM: Working principles and examples of traces.
With kind permission from Tem International GmbH http://www.rotem.de/site/index.php?option=com_content&view=article&id=1&Itemid=7&lang=en



	TEG	ROTEM	
Clotting time	R (reaction time)	CT (clotting time)	Period of initial fibrin formation
Clot Kinetics	K (K value)	CFT (clot formation time)	A measure of the speed to reach a specific level of clot strength
	α (angle in degrees)	α	Measures the rate of clot formation, reflects rate of fibrin build up and cross linking
Clot strength	MA (maximum amplitude)	MCF (maximum clot firmness)	Represents the ultimate strength of the clot (platelets & fibrin) function of maximum dynamic properties of fibrin & platelet bonding via GPIIb/IIIa receptors
Clot stability	Ly30 (Lysis at 30 minutes as ratio of MA)	CLI (Clot lysis index)	Measures rate of amplitude reduction from MA at 30 minutes, detects fibrinolysis

Fig. 7.4 Schematic and comparison of TEG/ROTEM traces and variables.

the rate at which the trace is generated, to standardize the test, and to provide additional information. Kaolin TEG is now more commonly used and is faster and more reproducible than native TEG. Rapid-TEG (r-TEG) is produced using tissue factor as an activator, information on *R* value is largely lost as this is so short, but the MA is generated within 10min and can be useful in situations where information on platelets, fibrinogen, and fibrinolysis is needed quickly.

- *Heparin:* TEG R time is markedly prolonged in the presence of heparin, but this effect can be neutralized using heparinase-coated cups. Simultaneous use of kaolin and heparinase-coated TEGs allows identification of the presence of heparin in the sample and also enables assessment of the underlying coagulation profile. This is very useful in any situation where heparin has been administered to the patient.

Thromboelastometry

ROTEM®; TEM International, Munich, Germany.

- The ROTEM has a shorter result time (10–15min) due to more intense activation. It uses an automated pipetting system and is less susceptible than the TEG to vibration and knocks due to the more stable platform.
- The ROTEM has four channels, compared with two on the TEG, enabling more analyses to be performed.
- Activators of the intrinsic pathway of coagulation (INTEM; phospholipid and ellagic acid) and extrinsic pathway (EXTEM: tissue factor) are used to provide similar information to APTT and PT.
- Three additional modifications include HEPTEM, which contains lyophilized heparinase for neutralizing heparin, APTEM which contains aprotinin to inhibit fibrinolysis and FIBTEM, which utilizes cytochalasin D, a platelet inhibitor that blocks the platelet contribution to clot formation, allowing quantitative analysis of the functional fibrinogen component. Current transfusion protocols advocate maintaining fibrinogen levels >1.5g/dL, but there is accumulating evidence that in active bleeding much higher levels of fibrinogen are required for sufficient clot strength with target levels of 2g/dL being recommended. FIBTEM has been used to guide administration of fibrinogen concentrate for the treatment of massive bleeding during thoraco-abdominal surgery.

Hypercoagulability

Surgical patients are at risk of hypercoagulability and thromboembolic events, especially in the immediate post-operative period. This risk is increased in some vascular patients who may have evidence of platelet hyper-reactivity and in patients with malignancies who characteristically have high fibrinogen levels. Both TEG and ROTEM allow identification of a hypercoagulable state. This is evidenced by a short R or CT or/and an increased MA/MCF. A number of studies have correlated high MA/MCF values with an increased likelihood of suffering post-operative thrombotic events including myocardial infarction. The sensitivity is very high (>90%) but the specificity is low.

POCT: platelet function monitoring

There is no consensus on the appropriate perioperative management of patients taking antiplatelet drugs. These drugs increase the bleeding risk but

abrupt discontinuation may predispose the patient to an increased risk of thrombosis.

Antiplatelet therapy

Has a key role in preventing atherothrombotic events in patients with vascular disease. Aspirin, which irreversibly inhibits platelet cyclo-oxygenase-1 (COX-1) and clopidogrel, which binds irreversibly to the platelet ADP $P2Y_{12}$ receptor are the antiplatelet drugs most commonly used. Dual therapy provides more effective platelet inhibition, as the drug effects are synergistic. Dual therapy is routine in patients who have received coronary stents to protect against in-stent thrombosis. Whilst antiplatelet drugs provide protection from thrombosis, they increase the risk of perioperative bleeding. Anaesthetists are increasingly confronted with patients taking dual antiplatelet therapy. Careful consideration must be given to the best management of these patients.

Stratifying the bleeding risk

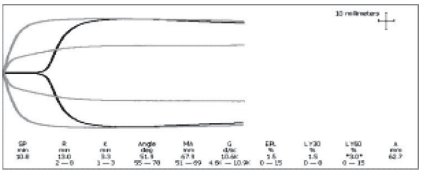
Because of the considerable inter-individual variability in response to both aspirin and especially clopidogrel, some patients may be at greater risk than others for adverse bleeding. Platelet inhibition in patients treated with the same anti-platelet regime is highly variable. Up to 30% of patients may show no demonstrable platelet inhibition on standard therapy. This has implications for the risks of recurrent ischaemic events in 'hypo-responders', conversely 'hyper-responders' with >60% platelet inhibition may be at increased risk of bleeding during surgery. Evidence suggests that bleeding risk increases as the degree of irreversible platelet inhibition increases and there is interest in attempting to quantify this risk. At present evidence suggests that POC platelet function tests could be used to identify those patients with minimal platelet inhibition (<30%) in whom surgery can proceed safely without the need for platelet transfusion. The association between higher degrees of platelet inhibition and the likelihood of problematic surgical bleeding is much more difficult to quantify. It is hoped that further research will provide cut off values which can be used as the basis for platelet transfusion algorithms (Fig. 7.5).

POC testing of platelet function

A variety of devices are available for platelet function POC testing.

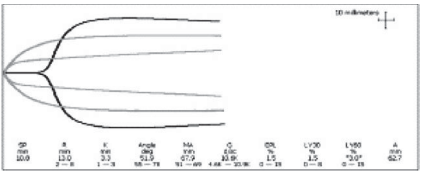
PFA -100®

Developed to mimic the template bleeding time and measures the closure time (CsT), i.e. time to cessation of blood flow of citrated blood aspirated at high shear rate through a central aperture of a membrane impregnated with collagen, and either adrenaline or adenosine di-phosphate (ADP). CsT may be prolonged when the platelet count is less than $100\,000 \times 10^9$, even if platelet function is normal and if the haematocrit is less than 30%, which limits its utility as a perioperative POC device. It is also affected by levels of vWF. A normal ADP CsT has been found to be a good negative predictor of bleeding, but an elevated CsT has a poor predictive value. The PFA-100 is relatively insensitive to the effects of clopiodgrel and it is not currently recommended as a POC test to quantify the response to clopidogrel or similar therapy.



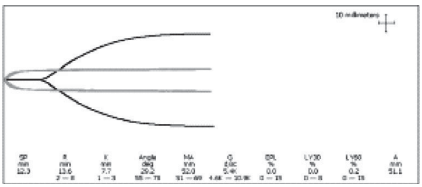
No platelet inhibition

MA on ADP stimulation (paler trace) same MA as Kaolin (dark) trace



Partial platelet inhibition: 40%

MA ADP less than MA Kaolin



Complete platelet inhibition

MA ADP same as fibrin trace, no demonstrable platelet activation with ADP

Fig. 7.5 TEG platelet mapping.

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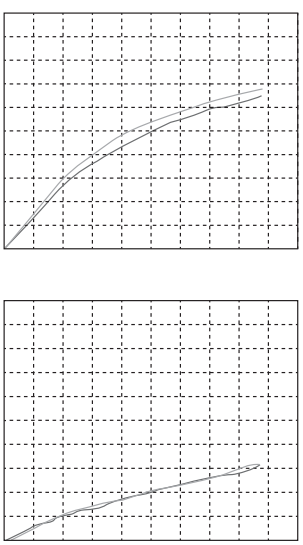
mTEG: Thromboelastography platelet mapping system™

Conventional TEG is not able to detect the effect of antiplatelet agents as their effects are overcome by thrombin that is generated in the sample. In TEG platelet mapping, thrombin generation is inhibited by heparin and a weak clot is generated by the addition of reptilase and factor XIIIa. This is equivalent to a functional fibrinogen trace. By adding a platelet agonist (arachidonic acid or ADP) any functional platelets will respond to activation by the agonist by increasing the amplitude of MA. If there is no platelet inhibition in the presence of the agonist this trace will have the same MA as the corresponding kaolin heparinase (KH) trace. Where inhibition is present, the MA will be reduced compared with the KH trace allowing estimation of the degree of inhibition.

Multiplate®

Impedance aggregometry is based on the principle that activated platelets expose receptors on their surface that allow them to attach to artificial surfaces. The multiplate provides two independent sensitive units, each consisting of two silver coated highly conductive copper wires. The instrument detects the increase in impedance caused by the intensity by which platelets attach to each of the sensors and transforms it into arbitrary aggregation units (AU) that are plotted against time (Fig 7.6). The 5-channel device uses various agonists, such as ADP, and arachidonic acid to assess



Examples of Multiplate Traces
A: Normal response to agonist: no platelet inhibition
B: Reduced response to agonist: significant platelet inhibition

Fig. 7.6 Multiple electrode platelet aggregrometry: Multiplate®.
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platelet function. Results are available in 3–6min. Although some interesting data is emerging especially in cardiac surgical patients, larger prospective trials are required to define the role of multiple electrode aggregometry in perioperative platelet testing and to define cut off values that can be used to predict and guide transfusion management.

Other POC analysers are available, such as Rapid Platelet Function Analyser (Ultigra, Accumetrics) with VerifyNow aspirin, and clopidogrel assays, PlateletWorks (ICHOR Helena BiiopScience), and the Cone and Plate Analyser (CPA 'Impat' Diamed). These have not been widely adopted in the perioperative setting.

Further reading

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Monitoring the brain

Vascular surgery and anaesthesia carries an inherent risk of injury to the CNS. Ideally, there would be a monitor which warned the anaesthetist and surgeon in time to prevent such injuries. The perfect monitor would have the following characteristics:

- *Highly sensitive:* absence of warning would indicate very low risk of injury.
- *Highly specific:* warnings would only occur when injury was likely to occur.
- *Diagnostic:* abnormalities would provide information on cause of injury.
- Continuous and rapid response.
- Response of monitor sufficiently early to avoid permanent injury.
- Unaffected by anaesthetic/surgical technique.
- Non-invasive.
- Inexpensive.
- Easy to set up.
- Low inter-user variability.

At the present time there is no such technology, each monitor has its own strengths and weaknesses.

There are various methods to categorize the monitoring available:

- Global or regional.
- Electrophysiological.
- Metabolic.
- Vascular.
- Pharmacological.
- Anatomical/imaging.

Global assessment

In the awake patient, it is theoretically possible to assess every aspect of function of the CNS. In practice, this is time-consuming and not achievable during surgery. However, focused examination of the relevant functions can be performed providing the patient is conscious and able to follow commands. It is important to remember that early deficits may be relatively subtle, so simply asking patients whether they feel alright is insufficient. Clearly, the person monitoring the patient needs to monitor the at risk region, e.g. right arm and speech for left-sided carotid surgery.

Advantages

- Non-invasive.
- Global.
- Specific.

Disadvantages

- Requires patient concentration/compliance.
- Only available when awake/unaffected by nervous system dysfunction (e.g. previous cerebrovascular accident (CVA), regional anaesthesia).
- Deficits may be detected too late.

Electrophysiological monitoring

Electroencephalography

The electroencephalogram (EEG) is the surface recording of the electrical activity of the brain, analogous to the ECG for the heart. Unlike the ECG, the amplitude is in the μV range, and there is no clear repetitive pattern.

The electrical activity originates from the excitatory and inhibitory postsynaptic potentials of the cortical pyramidal cells in layers II–V.

Although the continuous EEG appears chaotic, there are underlying characteristics that can be used to describe the EEG relating to amplitude, frequency and anatomy.

The frequency bands are:
- *Delta (δ), 0–4Hz:*
  - Stage 4 sleep.
  - Coma, deep anaesthesia, hypoxia, cerebral ischaemia, or infarction.
- *Theta (θ), 4–8Hz:*
  - Drowsiness.
  - GA, focal subcortical lesions.
- *Alpha (α), 9–12Hz:* awake—relaxed/eyes closed.
- *Beta (β), 13–30Hz:* awake—alert/working.

The EEG is affected by pathology and by drugs used during anaesthesia, so requires careful interpretation.
- Acute cortical ischaemia causes a reduction in amplitude of the EEG with reductions in amplitude of >75% viewed as major changes.
- Seizures may results in focal or generalized spikes, sharp waves or spike and wave activity.

Inhalational anaesthetic agents have dose and agent dependent effects on the EEG.
- Nitrous oxide causes activation of the EEG even in the presence of burst suppressing concentrations of isoflurane.
- *Isoflurane:*
  - Limited β activity at 1.0 MAC.
  - Burst suppression at 1.5 MAC.
  - Electrical silence at 2.0 MAC.
- *Sevoflurane:*
  - Limited β activity at 1.0 MAC.
  - Limited α activity at 1.5 MAC.
  - δ activity and burst suppression at 2.0 MAC.
- *Desflurane:*
  - Limited β activity at 1.0 MAC.
  - Limited α activity at 1.5 MAC.
  - Burst suppression or electrical silence at 2.0 MAC.

IV drugs used during anaesthesia also have specific effects:
- *Thiopental:* similar effects to isoflurane—increasing doses associated with β activity followed by burst suppression and electrical silence.
- *Propofol:* initial increase in α amplitude, followed by increased δ and θ activity and then burst suppression.
- *Benzodiazepines:* initial reduction in α activity and increase in β followed by increasing δ and θ activity.

Due to the difficulty of interpretation of the raw EEG, automated processing is commonly used.

Cerebral function analysing monitor (CFAM)

The CFAM applies a fast Fourier transform to the raw EEG obtained from two pairs of symmetrical scalp electrodes and displays the relative

proportion of the different EEG frequency bands and periods of burst suppression. It is easier for non-experts to interpret than the raw EEG.

Bispectral Index (BIS®)

The Bispectral Index monitor uses a proprietary algorithm to calculate a dimensionless number from a variety of EEG parameters from the time domain, frequency domain, and phase relationships between the individual component waveforms. The weighting of the individual parameters is varies with depth of anaesthesia as particular parameters are better at lighter versus deeper levels. The BIS ranges from 0 (electrical silence) to 100 (fully awake) and is statistically associated with the depth of anaesthesia and probability of awareness.

Although it may have utility as an adjunct to minimize the risk of unintentional awareness during general anaesthesia, it does not appear to be of benefit in predicting neurological dysfunction during carotid endarterectomy.

Entropy

Entropy is the degree of disorder in a system. In the awake state, the EEG has a high degree of disorder and hence high entropy. As depth of anaesthesia increases, the waveforms become more uniform and, hence, entropy decreases. The entropy monitor calculates the degree of entropy in both the frontalis electromyogram (EMG) and the frontal EEG. The response time of entropy (particularly 'response entropy', which includes the EMG component) is very rapid.

As with BIS it is unlikely that entropy will have specific value during vascular surgery.

Evoked potentials

Evoked potentials monitor the response of part of the nervous system to a repeated stimulus at another point. They therefore assess the integrity of the whole pathway between the two points. Repeated signals are averaged to remove noise.

Evoked potentials can either be motor or sensory, the latter using various senses— somatosensory, visual, audio.

Visual-evoked responses are not generally used during vascular surgery and brainstem auditory-evoked responses (BAEP) are used a depth of anaesthesia monitor, rather than assessing CNS integrity.

Somatosensory-evoked potentials (SSEP)

SSEP are averaged recordings of the brain electrical activity in response to repetitive peripheral sensory stimulation. They have the theoretical advantage over the global EEG that they monitor deeper structures, not just the cortex, as the neural signal has to pass through brain stem structure before eliciting a response in the somatosensory cortical areas. However, in practice, they are sensitive to the effects of volatile anaesthetic agents, and they have not been shown to demonstrate superior sensitivity or specificity over raw EEG during CEA. This may be in part because the deeper (subcortical) structures responsible for SSEP are less affected by ischaemia than cortical structures.

Cerebrovascular monitoring

The ideal monitor of cerebral blood flow would be non-invasive, easy to learn/use, continuous, accurate, able to detect regional differences, and be able to assess adequacy of perfusion at the same time. Such a monitor does not currently exist. There are many research tools available, but in clinical, non-neurosurgical practice there are only a few methods in use.

Transcranial Doppler (TCD)

The basal cerebral arteries are accessible to Doppler US through several acoustic windows including temporal (most common), the orbit, and foramen magnum. Using standard Doppler US principles, blood flow velocity in the basal arteries can be measured. The middle cerebral artery carries 50–60% of blood flow from its feeding carotid artery and is therefore the major source of CBF. It can be insonated through the temporal acoustic window, which is usually found in the area above a line drawn between the tragus and lateral canthus. The MCA usually lies 35–60mm below the skin. It is properly identified by its flow characteristics, following it up and down its course, and the effects of ipsilateral carotid artery compression.

- TCD gives an indirect measure of CBF (flow velocity is flow divided by vessel area).
- TCD can be used to detect gaseous and particulate emboli as these have characteristic acoustic patterns.
- TCD provides no direct information about the match between flow and metabolism.
- TCD is not possible in approximately 10% of patients due to absence of adequate bony 'windows.'
- Various measures of adequacy of TCD estimated cerebral blood flow have been described including:
  - Proportional reduction in MCA flow velocity on clamping during carotid endarterectomy (clamp/preclamp ratio <0.6).
  - Absolute MCA flow velocity (<30cm/s).
- Reported sensitivities and specificities for predicting the need for shunting vary but are around 90 and 75%, respectively.

Stump pressure

When the internal and external arteries are clamped during CEA, the pressure in the distal carotid (stump pressure) is the pressure within the Circle of Willis. If there is inadequate Circle of Willis flow, then stump pressure will be insufficient for cerebral perfusion. A variety of threshold values have been suggested for stump pressure but values of 40–50mmHg are generally used to indicate the need for shunting during CEA. Much of the data comes from patients undergoing CEA under local anaesthesia, so caution is needed in applying these thresholds when general anaesthesia is used.

Metabolic monitoring

The CNS requires an adequate supply of nutrients to meet metabolic demand. Under normal circumstances, supply and demand are tightly coupled, but under pathological conditions there may be mismatch. If this occurs then various monitors may be able to detect imbalances.

Cerebral oximetry

Cerebral oximetry uses similar principles to pulse oximetry to calculate the proportion of oxygenated haemoglobin in the light path. Unlike pulse oximetry the light detected by cerebral oximetry is reflected rather than transmitted light. The calculated O_2 saturation (ScO_2) is a weighted average of venous (70–75%) and arterial blood in an area of around 1cm^3 in the frontal lobes. Newer technologies claim to have reduced the impact of changes in non-cerebral (mainly skin) tissues in the light path.

The resulting ScO_2 reflects the balance between O_2 supply and demand. Reduced ScO_2 may be a consequence of reduced flow, reduced arterial oxygen content or increased cerebral metabolism.

- The evidence supporting specific near infrared spectroscopy thresholds as an indication of the need for shunting or the risk of cerebral hyperperfusion is modest.
- Changes in ScO_2 only reflect changes in the underlying brain. Other areas of the brain may be subject to undetected changes.
- Different technologies may give differing results.

Jugular venous oxygen saturation

The oxygen content of blood draining the brain is determined by the balance between delivery and demand. Catheters can be placed at the level of the jugular bulb for intermittent blood sampling or continuous oximetry. In theory, inadequate oxygen delivery in relation to cerebral demand should lead to reduction in jugular venous oxygen saturation (SjO_2) below its normal value of around 70–75%. Although reduction SjO_2 is relatively specific for supply–demand imbalance, SjO_2 is a relatively insensitive monitor. SjO_2 is a global average, so regional ischaemia may not be apparent. The relative flow of venous drainage from cortical and deeper structures to each jugular vein is inconstant between individuals; if the jugular vein sampled is not draining the area of interest, changes may be missed. Although experienced operators take great care to avoid sampling blood, which includes non-cerebral venous blood (notably the facial vein) there may be unavoidable contamination of the sample.

Invasive cerebral monitoring

It is relatively commonplace within neuro-intensive care to insert monitoring devices direct into the brain parenchyma. These probes can measure brain tissue O_2 partial pressure ($PbrO_2$) and/or metabolite concentrations from microdialysis. There is some evidence that these monitors can detect abnormal changes before other monitors, including neurological deficits in awake individuals, which may allow early intervention.

Pharmacological monitoring

Patients undergoing general anaesthesia routinely have end-tidal volatile agent monitoring. This has some value in predicting the pharmacological effects of volatile agents on the brain, particularly if care is taken to adjust for age. However, there is significant inter-individual variability, which is compounded by the interaction between other drugs, such as opioids and benzodiazepines, and the volatile agents.

Chapter 8

Anaesthesia for vascular radiology

General considerations regarding imaging and working in the radiology suite

Vascular interventional radiologists (interventionists) treat a very wide spectrum of conditions and have clinical interactions with almost every specialty. Procedures include:

- *Balloon angioplasty or stenting for non-cardiac vascular occlusive disease:* e.g.
  - Iliac, mesenteric, renal, and carotid arteries.
  - Central, upper limb, and iliocaval veins.
  - Renal dialysis fistula and grafts.
- *Embolization of:*
  - Active haemorrhage (gastrointestinal, traumatic, post-partum, haemoptysis).
  - *Tumours*—hepatic, renal, and osseous metastases.
  - Vascular malformations.
  - Chylothorax (thoracic duct).
  - Varicoceles.
- *Stent-grafting for:*
  - Thoracic, abdominal, iliac, and popliteal saccular, and fusiform true aneurysms.
  - Pseudoaneurysms.
  - Dissections.
  - Traumatic haemorrhage.
- *Thrombolysis:* arterial and venous.
- Trans-jugular porto-systemic shunts (TIPSS).
- US-guided thrombin injection of false aneurysms.
- Foreign body retrieval/central venous catheter repositioning.

The vast majority of these procedures are performed under local anaesthesia with variable use of radiology administered and monitored supplemental sedation, opioids, and Entonox®. Planned RA or GA for radiological interventions is becoming more common as technical advances increase the complexity of diseases that can be treated by an endovascular approach. In elective patients anaesthetic support is indicated when:

- Adjunctive open surgical procedures are required.
- The treatment is very painful (e.g. alcohol injection of a vascular malformation, TIPS).
- A long procedure time is anticipated.
- Patients are young or unable to tolerate/co-operate with procedure.
- Anaesthesia is requested by the patient.

Radiation safety/protection

Lead protective clothing

- Always wear a full lead (one piece apron or 2-piece jacket and skirt) and thyroid shield. To fit you it should be closed at the sides of the body and reach at least to mid-thigh level. Radiation is an invisible hazard, which should not be underestimated.

- If you suffer from back problems ask the radiographer for assistance.
- It is not good practice to sit in the lead glass protected radiology control area not wearing lead protection. This compromises the radiology procedure if the patient urgently requires attention.

Pregnancy

Inform the radiographic staff if you are or may be pregnant. They will discuss the options and the risks with you.

Reducing your radiation exposure

- Follow the advice and practice of the radiology staff. They know how to reduce their exposure and yours.
- Only the patient should be exposed to the primary X-ray beam, which passes from tube to image intensifier. If you need to adjust monitoring or support equipment close to area being imaged always check with radiologist when it can be performed safely.
- Staff exposure is due to scattered radiation from the primary beam.
- Scatter exposure rapidly drops the further you are from the primary beam. It follows the *inverse square law*. 1m from primary beam, the dose is 1/1000th. Taking one small step away from the primary beam has a big impact on your exposure.
- The scatter produced during fluoroscopy (screening) is much lower than during acquisitions (runs). During runs a single or usually a series of several images is acquired and a larger dose is given so that the images acquired are of better, diagnostic quality. Always step further away during runs.
- The radiologist will often ask for a 'breath-hold' during runs to reduce and movement artifact from breathing.

Radiology environment

The radiology angiographic suite is commonly within the radiology department and is, therefore, an isolated environment. Increasingly dedicated hybrid theatres for combined radiology and open surgical procedures are being developed. Some of these will be in the theatre complex and some within radiology.

If you do not normally work in the radiology department, but would be expected to out of hours you should know where all emergency drugs and equipment is kept. A standard operating procedure should define those patients who should be treated in radiology and the pathway for their acute and subsequent care.

Interactive working between vascular interventional radiology teams, vascular surgeons, anaesthetic teams, neuro-radiologist, and cardiologists is critical to performing many complex interventional procedures. As with all interventions case mix adjusted complications are lower in high volume centres. A radiology specific WHO safer procedure (surgery) check should be performed with all team members present. Its value is much greater when a team contains members who do not routinely work together. It should be performed without exception.

Contrast agents

Iodinated contrast agents are the most frequently used and are derivates of benzoic acid. For intra-vascular use, non-ionic agents have largely replaced ionic agents in the UK. Contrast reactions may be divided in to direct (dose related) and idiosyncratic. The most common direct reaction is contrast-induced nephropathy. It is more likely in those with pre-existing renal impairment, particularly if the patient is diabetic or volume depleted. The only proven renal-protective strategy is pre-hydration.

Serious allergic reactions are rare with severe reactions (circulatory collapse, arrhythmia, or bronchospasm) in 1:3000 and death in 1:40 000. Skin reactions may occur in up to 1:100 patients. The risk of an anaphylactoid reaction is increased 10-fold in those with a previous iodinated contrast allergy, 5 times increased in asthmatics and 3 times increased with a history of general allergic reactions. Reactions to topical iodine or shellfish does not represent an increased risk. Use of a different agent to the one a patient has previously reacted to is routine management and requires a detailed history. Steroid prophylaxis commenced orally 24h before contrast administration is unproven, but has some evidence to support its use. In urgent examinations hydrocortisone 200mg IV is commonly prescribed, but its use is empirical. The management of patients with possible increased risk of allergic reactions must be discussed with a radiologist at the earliest opportunity.

Carbon dioxide is a negative contrast agent which can be used in contrast allergic patients or to decrease the dose of iodinated contrast in those with poor renal function. It can only be used below the diaphragm or in the venous system. Intra-cerebral CO_2 causes convulsions.

Gadolinium is an MRI contrast agent. It is a poor X-ray contrast agent, but may be used in those with severe contrast allergy. It is expensive and doses of greater than 40mL are nephrotoxic, which limits its utility.

Sedation and anaesthesia in the radiology suite

Environment

Radiology suites are generally designed to optimize conditions for the radiologist and the very costly imaging equipment. As such the working environment for the anaesthetist is often compromised, in addition to already incurring the problems associated with working in any distant site. To avoid patient compromise it is important to anticipate potential problems related to the site. Factors to consider include:

Organizational

- Familiarity of anaesthetic staff with the department layout and available anaesthetic equipment.
- Access to blood products.
- Access to drugs, including resuscitation and controlled drugs.
- Rapid access to IV fluids.

- Access to additional equipment such as rapid flow IV warming or blood gas analyser.
- Availability of skilled recovery.

Patient comfort

- Narrow table with limited padding or support.
- Cool ambient temperatures require warming even of awake patients.
- Potential for prolonged procedures.

Procedural problems

- Interference with anaesthetic lines/equipment by moving imaging equipment.
- Difficult communication if there is a need for additional anaesthetic support.
- Limited access to the patient during the procedure.
- Additional surgical equipment and staff contributing to room crowding.
- Potential for sudden rapid blood loss or the need to convert to a surgical procedure.

As the radiology staff are less familiar with anaesthesia and the running of a surgical theatre it is vital that the anaesthetist and operating department practitioner (ODP) plan carefully. Once a procedure is underway the ODP will not be easily able to fetch forgotten or unanticipated items, especially if they need to come from the main theatre suite.

Procedures

Most vascular radiological procedures, such as angioplasty and more distal stents are conducted under LA without the need for anaesthetic assistance. The main procedures requiring an anaesthetist include endovascular aortic aneurysm stenting (EVAR), either electively or increasingly as an acute procedure, and combined surgical and endovascular procedures such as iliac stenting in combination with distal surgical bypass or endarterectomy. The variety of procedures will depend on the conditions in the radiology suite, e.g. whether it complies with requirements for theatre conditions, and if capacity allows time for non-radiological procedures to be completed.

Anaesthetic management

It is possible for an EVAR to be performed under local anaesthesia of the groin access sites only, and in an emergency with an unstable patient this may be the only option. Unfortunately, the femoral arteries are effectively clamped for much of the procedure, and ischaemic leg pain can become a significant problem during purely LA. As a result most anaesthetists opt for a regional procedure, either spinal, epidural, or CSE. The advantage of a catheter technique is that the duration of the procedure can be difficult to anticipate and a top-up may well be required. Heparinization of the patient during the procedure is necessary as with an open repair, and it is generally accepted that an hour between central neuraxial block and administration of heparin is adequate. Insertion of additional lines, patient positioning, and preparation, and surgical cut down usually provides the time needed.

As long as good wide bore IV access is secured there is generally no need for a central line; however, an arterial line is very helpful for monitoring any rapid changes in BP and blood sampling in longer procedures. Blood loss from the access sites can be significant and is not easily seen by the anaesthetist as it tends to collect in the drapes, so vigilance and regular haematocrit checks will help detect any occult loss.

Sedation can be added for patient comfort; however, there is a continual need for patient co-operation during screening, so it is important to maintain good communication throughout.

General anaesthesia

- A small number of patients may be unsuitable for or refuse RA.
- Choice of anaesthetic will depend on the patient's condition, the procedure and any environmental constraints.
- Induction is often conducted with patient on a trolley, as radiology table is narrow, and even if it is capable of tipping head down, this is limited, relatively slow, and may need assistance of a radiographer.
- Intubation and ventilation are generally preferred as procedure may be prolonged and it will be necessary to temporarily stop ventilation during screening runs.
- Central venous access may be considered for additional monitoring, or drug and fluid administration in unstable or compromised patients, but is generally not necessary in elective cases.
- Pay careful attention to patient positioning, and secure lines, ventilator tubing, and monitoring to prevent problems during imaging.
- Active warming and temperature monitoring are also important, and antibiotic prophylaxis and heparinization should be discussed with the surgeon and radiologist before starting procedure.
- If no dedicated recovery area is available, patient should be transferred to a central recovery area, or a critical care unit (CCU) if needed.

Contrast-induced nephropathy and renal protection

AKI occurring after IV iodinated radiocontrast administration is termed contrast-induced nephropathy (CIN).

Procedures associated with CIN

- Coronary angiography or PCI.
- Peripheral arterial angiography or angioplasty.
- EVAR.
- Contrast-enhanced CT.
- IV urography.

Incidence

- 2–3% after IV radiocontrast investigations.
- Up to 25% in those with multiple risk factors.

Aetiology

The is no single proven aetiology, however apoptosis of renal tubular cells and hypoxia in the renal medulla occur with the production of reactive oxygen species. These have been attributed to a direct toxic effect on tubular cells, ischaemia-reperfusion, injury and/or reduced renal blood flow.

Risk factors

- Pre-existing CKD.
- Diabetes.
- Dehydration.
- HF.
- Age >70yrs.
- Concurrent administration of nephrotoxic drugs (e.g. NSAIDs, aminoglycosides).
- High doses of IV contrast (>150mL).
- Repeated administration of IV contrast within 72h.
- Hyper-osmolar contrast.

Diagnosis

An increase in serum creatinine concentrations of >44μmol/l 48–72h after administration of IV radiocontrast, in the absence of other causes.

Clinical features

- Usually asymptomatic.
- Usually transient (serum creatinine usually returns to normal within 5 days).
- May increase the risk of acute kidney injury in response to other nephrotoxins.
- Rarely needs renal replacement therapy.
- May lead to permanent kidney damage particularly if several risk factors present.
- However, severe long-term kidney damage or death associated with CIN is very rare.

Prevention

- Careful assessment of volume status.
- Allow clear fluids orally up to 2h pre-procedure and encourage to drink post-procedure.
- IV hydration with isotonic or hypotonic crystalloid (0.9% saline) at 1.5mL/kg/h for 4–12h pre-procedure and up to 48h post-procedure.
- IV isotonic (1.26%) $NaCO_3$ may be used as an alternative to saline.
- Use iso-osmolar (290mOsm) contrast agents, e.g. iopamidol, ioxaglate, or iohexol.
- Always use minimum dose of contrast; do not exceed 150mL.
- Delay vascular surgery or other procedures where contrast is to be administered for >48h after contrast studies.
- Avoid other nephrotoxic drugs for 24h pre- and post-procedure.
- Monitor urine output for 4h post-procedure.

In high risk groups

- Consider N-acetylcysteine (600mg PO bd or 500mg IV bd for 24h pre- and post-procedure).
- Atrial natriuretic peptide (data limited).
- Monitor urine output and renal function for at least 48h post-procedure.
- Continuous veno-venous haemofiltration (CVVH) may be required in established severe CIN.

The following are not beneficial:

- Furosemide, dopamine, fenoldopam, Ca_{2+} channel blockers, theophylline (data conflicting).
- Prophylactic CVVH for high risk cases.

Further reading

Hoste EA, De Waele JJ, Gevaert SA, *et al*. Sodium bicarbonate for prevention of contrast-induced acute kidney injury: a systematic review and meta-analysis. *Nephrol Dialysis Transplant* 2010; 25(3): 747–58.

Useful website

http://www.renal.org

Radiological management of haemorrhage and other embolization procedures

Embolization is the intentional occlusion of vessels. It was first described by Charles Dotter in 1966. Since then Dotter's vision that 'the angiographic catheter, used with imagination, can become an important surgical instrument' has come to fruition. The first clinical acceptance of its clinical utility was in the early 1970s, when it was used to control gastric haemorrhage (initially using autologous blood clot) and traumatic pelvic bleeding. Since then, the vision and ingenuity of medical device companies and the enthusiasm of interventional radiologists have expanded the endovascular options beyond internal haemorrhage. Embolization has now become the treatment of choice for many clinical conditions and an important part of the therapeutic armamentarium in many more. Embolization procedures are often more technically challenging than other endovascular procedures. Inexpertly performed embolization results in failed haemorrhage control, restricts future treatment options, or results in significant morbidity or mortality.

Stent-grafts (metal meshwork tubes covered with an impervious fabric such as expanded PTFE or Dacron) have extended the range of conditions, which can be managed with the lesser physiological insult of an endovascular approach as opposed to open surgery. This includes the endovascular options in haemorrhage with increasing use in trauma and aneurysm rupture. Endovascular stents with diameters ranging from 3 to 46mm are available off-the-shelf. The larger devices are used for treating abdominal

and thoracic aortic disease (see 📖 Open aortic aneurysm repair, p. 350 and 📖 Emergency abdominal aortic aneurysm repair: open and endovascular repair, p. 448), for endovascular management of aortic disease.

In haemorrhage, interventional radiology (IR) may

- Be one of a number of treatment options.
- Be the only treatment option.
- Be a 'holding strategy' prior to definitive intervention (e.g. surgery).
- Be complementary to other therapies (e.g. embolization of a hypervascular renal metastasis prior to surgical spinal fixation).
- Not an option (not technically possible, suitable device not available or speed of haemorrhage control too slow).

Optimum treatment

- The optimum treatment for the individual patient with bleeding should be a consensus decision made by those with the skills to control bleeding (usually open surgical versus endovascular), those supporting the patient and whenever possible the patient.
- Speed of access to intervention, likelihood of success, co-morbidities, and adverse features for surgery or endovascular intervention will all inform the decision-making process.
- The decision-making process in those with internal bleeding tends to be slower than in those with similar rates of externally visible bleeding despite the risks being equivalent.
- If the patient has had a diagnostic CT to identify site or sites of bleeding the severity may be more immediately evident to the radiologist.
- It is important that this is appreciated particularly in young patients in whom there is a delay in cardiovascular decompensation, and a definitive treatment plan is quickly formed and followed through.

Basic principles

Embolization procedures vary from relatively simple to the most challenging cases faced by the angiographer. In line with the principle of first do no harm, the aim of embolization is to occlude a bleeding vessel or territory as selectively as possible to minimize collateral damage to adjacent non-target structures. This requires detailed knowledge of normal vascular anatomy, anatomical variants, and collateral pathways. Once occluded, it is usually impossible to recanalize vessels. There is no such thing as a standard embolization procedure and techniques have to be individualized to the particular patient's anatomy and clinical condition. The role of alternative therapies, their likely outcomes, the chances of success, and recurrence must all be considered when discussing the therapeutic options with a patient.

Embolization often requires selective catheterization of small branch vessels. A high level of technical catheter and guide wire skill is required and needs continuous practice to maintain. It also requires a co-operative patient. A wriggling patient or one who cannot breath-hold during acquisition runs increases the risk of an adverse event or failure to control

bleeding. Movement during image-guided procedures has similar consequence to movement on slow shutter speed photography—the images are blurred and the vessels cannot be identified. Such patients often require anaesthesia or sedation.

In stent-grafting the aim is to occlude the site of bleeding, whilst maintaining flow through the culprit vessel. This is most commonly applied to large vessels such as the aorta or other large arteries, such as the iliac, carotid, and subclavian arteries, but also applies smaller vital arteries such as the vertebral, renal, and hepatic arteries.

Embolic agents

There is an increasing choice of embolic materials. They may block by physical obstruction, stimulation of the clotting cascade, or endothelial damage.

The first question is 'Will a temporary agent work?' The commonest temporary agent is gelatin foam sponge (Spongostan™ or Gelfoam®). Occlusion usually lasts for a few days to weeks, but recanalization does not always occur. It is most often used in trauma.

The choice of permanent embolic agents depends on the size of the target vessel:

- Large vessels can be occluded with screw detachable plugs delivered via long sheaths or catheters for smaller vessels.
- Smaller vessels can be occluded by coils, which come in many configurations. Coils are commonly used in GI bleeding and trauma. If there is an ongoing angiogenic stimulus (e.g. tumours, chronic inflammation, or vascular malformations) proximal occlusion will simply allow rapid collateral development, which can then be difficult or impossible to treat.
- *Particulate agents:* such as poly-vinyl alcohol particles and acrylic co-polymer microspheres come in range of sizes, which allow a predictable level of vessel occlusion down to capillary level. The more distal the occlusion the greater the chance of tissue necrosis.
- *Liquid embolic agents* include:
  - Adhesive glue mixtures of variable polymerization times.
  - Non-adhesive Onyx™ (ethylene vinyl alcohol copolymer dissolved in dimethyl sulphoxide and suspended micronized tantalum powder to provide contrast for visualization under fluoroscopy).
  - Sclerosants including absolute ethanol (which is very painful on injection and requires GA) and the detergent sodium tetradecyl sulphate as liquid or foam when mixed with air.

Liquid agents occlude very distally, are the most difficult to use and the least forgiving. They are most commonly used in the treatment of vascular malformations.

Consent

The potential adverse consequences of occluding a vessel must always be considered, including damage to healthy tissues supplied by the target vessel, non-target embolization of adjacent vessels (e.g. due to reflux of particles or coil displacement) and post-embolization syndrome, and discussed with

the patient. In haemorrhage the risk of failure to control bleeding or, of late, recurrence should be estimated. For example, in patients with haemoptysis due to chronic inflammatory lung disease bronchial artery embolization may have to be repeated every few years due to recruitment of non-bronchial systemic artery collaterals from the chest wall and subclavian arteries.

When informed consent cannot be obtained due to patient's condition, it is appropriate to perform medical treatments necessary to save life or prevent serious deterioration in the patient's condition. This scenario commonly applies in patients with life-threatening haemorrhage. The decision process should be clearly documented by two doctors. Relatives should be consulted and may be able to give an indication of the patient's wishes, but are unable to provide consent on behalf of the patient.

Coagulation status

Patients for elective procedures should continue on aspirin and clopidogrel. Clopidogrel does not significantly increase risk of bleeding in angiographic procedures. If warfarin can be safely stopped for a few days, it should be discontinued for 3–5 days pre-procedure. The INR should be less than 1.5 at the time of the procedure. If warfarin cannot be safely stopped the patient should be converted to IV or low molecular weight heparin, according to the angiography department protocol.

In active haemorrhage the local massive transfusion protocol should be adopted if appropriate. Otherwise, the patient should be regularly assessed for transfusion or laboratory factors that indicate coagulopathy. It is important that this should be aggressively corrected as embolization procedures depend on activation of the patient's clotting cascade to completely occlude the bleeding site. Embolization may fail if the patient is coagulopathic.

Access vessels

The anaesthetist should have an appreciation of the vascular access routes that the radiologist may need to use; the shortest straight route is usually best. Common femoral vessels are most commonly used. For systemic arteries the femoral artery is used. Alternative access, such as the brachial artery is occasionally required. Venous lesions and pulmonary artery lesions are rarer and usually require common femoral or internal jugular vein access.

The risk of access site complications (bleeding, dissection, thrombosis, and distal embolization) increases as the sheath size increases. A 4 French sheath has a luminal diameter of 1mm (1 French = 0.333mm). A 12 French sheath has a luminal diameter of 4mm. The risk of post procedure arterial bleeding has a linear relationship to the area of the puncture. From school mathematics (area = r^2) it will be appreciated that the area of a 12 French tube is 15 times greater than a 4 French tube. Arterial punctures larger than 12 French cannot be controlled with manual compression, and require surgical or percutaneous closure devices.

Clinical applications of embolization

The clinical applications of embolization are listed below. Those in bold commonly require anaesthetic support, usually with GA.

- *Aneurysms:*
  - Renal.
  - Visceral.
- *CNS (by interventional neuron-radiologists):*
  - Coiling for sub-arachnoid haemorrhage.
  - Closure of AV fistulas.
  - AV malformation occlusion/control.
- Epistaxis.
- *Endo-leaks:* following EVAR.
- *Gastrointestinal (GI) bleeding:*
  - Upper GI (proximal to ligament of Treitz) bleeding.
  - Lower GI bleeding.
- Variceal bleeding—uncontrolled by endoscopy. Trans-hepatic porto-systemic shunt (TIPSS). Always requires GA.
- *Iatrogenic haemorrhage:*
  - Central line trauma or misplacement.
  - *Post-operative*—most commonly upper gastro-intestinal surgery.
- Internal iliac artery (IIA).
- Embolization.
- Pre-EVAR when the common-iliac artery is too dilated to get a seal; the stent-graft has to seal in the external iliac artery. The IIA is occluded to prevent back-flow into the aneurysm sac.
- *Liver:*
- ***TIPSS***—see GI bleeding.
  - *Tumour (hepatocellular carcinoma and metastases embolization)*—embolic beads tagged with chemotherapy agents (chemoembolization) or selective internal radiation therapy (microscopic spheres of around one-third the diameter of a strand of hair are bonded to Yttrium-90, a pure β emitter with a half-life of about two-and-a-half days are lodged in the vascular tumour bed).
  - *Portal vein embolization*—to devascularize part of the liver causing the remainder to hypertrophy before aggressive resective tumour surgery.
- *Lung:*
  - *Haemoptysis*—bronchial artery embolization (BAE) successfully treats >95% of cases. Acute life-threatening cases often require isolated lung ventilation. Risk is 'drowning' not exsanguination. Requires breath-holding for good quality imaging to prevent non-target occlusion of the anterior spinal artery (paraplegia risk of 1% in BAE). Children and poorly co-operative patients may require GA.
  - *Pulmonary artery pseudo-aneurysms*—trauma including iatrogenic (e.g. Swan–Ganz catheters), right-sided endocarditis, and TB (Rasmussen aneurysms).
  - *AV malformations*—treatment is to diminish the risk of stroke and reduce hypoxia from shunting. Haemoptysis is rare.
- *Ovarian vein:* ovarian vein incompetence.
- *Pancreatitis* complications.

- *Penis:* traumatic high-flow priapism.
- *Preoperative:* embolization:
  - Meningiomas and paraganglionomas.
  - Renal tumours.
  - Metastases.
- *Prostate:* hyperplasia (experimental).
- *Testicular vein:* varicocele treatment.
- *Thoracic duct*: in high output chylothorax, which does not respond to medical therapy percutaneous embolization of the cisterna chylii is an effective treatment.
- *Thyroid:* multinodular hyperplasia unfit for surgery (experimental).
- *Trauma:* need for GA dependent on the patient's condition and co-operation, rather than needed to perform procedure. The exception is traumatic aortic injury, which usually requires general anaesthesia.
- *Catastrophic haemorrhage:* a distal aortic occlusion balloon or mid-thoracic aortic occlusion balloon can be placed in A&E as a temporizing manoeuvre in the emergency department. This can be a life-saving measure, but definitive intervention (open surgery or IR) must follow immediately. It only buys a brief period of time.
- *Thoracic aortic injury:* treated by stent-grafting (see 📖 Open aortic aneurysm repair, p. 350).
- *Large vessel injuries:* treated by stent-grafting to maintain flow to vital structures.
- *Solid organs (spleen, liver, and kidneys most commonly and in descending order of frequency):*
  - Active bleeding treated acutely.
  - Pseudo-aneurysms may be treated the next working day.
- *Pelvic fractures:* arterial bleeding (not controlled by pelvic binding). External fixators rarely control arterial bleeding which does not respond to a binder. In this case their use delays bleeding control and makes embolization considerably more difficult.
- *Extremities:*
  - *Acute ischaemia*—stent-grafting or stenting is an alternative to open surgery and may avoid a thoracotomy in the case of upper limb ischaemia.
  - *Haemorrhage*—options are embolization of non-vital branches, stent-grafting of vital branches or endovascular balloon control and then surgical repair.
  - Pseudo-aneurysms without active haemorrhage and problems with AV fistulas can usually be treated on planned lists.
- *Tumours:*
  - *Renal angiomyolipomas*—these are vascular tumours. Radiological treatment is undertaken if known to have bled or >4cm (high risk of bleeding).
  - *Renal cell carcinoma*—to reduce blood loss at nephrectomy or for symptomatic relief or haemorrhage control in inoperable lesions.
  - *Renal and thyroid metastases* (hypervascular metastases)—vertebral and long bone metastases may be treated radiologically before stabilizing surgery or treatment of fracture, respectively.

- *Uterus:*
  - Control of post-partum haemorrhage.
  - Treatment of fibroids and adenomyosis (less effective for the later). Painful procedure requiring PCA for up to first 24h post-procedure.
- *Vascular malformations:*
  - Venous and lymphatic-sclerotherapy with limited embolization to control outflow veins. GA for children only.
  - *AV malformations*—GA for children, prolonged cases, and when 100% ethanol is planned to obliterate the nidus. Ethanol is extremely painful.
  - *Varicose veins*—atypical patterns of varicose veins may require radiological treatment.

Emergency treatment of haemorrhage

Emergency embolization is performed to control active bleeding or treat patients at high risk of active bleeding.

In trauma and post-partum haemorrhage bleeding is characteristically continuous until controlled. These patients require rapid intervention.

Gastrointestinal bleeding (GIB), haemoptysis, epistaxis, and iatrogenic (post-surgery, biopsy, or central line misplacement) bleeding tend to be intermittent with temporary thrombosis at the bleeding site, which is then dislodged by rising BP, endogenous clot lysis, or reversal of vasospasm of the culprit vessel. For GIB this is a particular problem as the secondary signs of bleeding (pseudoaneurysms and vessel spasm and cut-off) are not reliably seen and a there are multiple potential feeding vessels.

Embolization is useful in acute upper GIB, which has caused haemodynamic instability, particularly where the shock index is >1 (heart rate greater than systolic BP). Two different scenarios may occur:

- In the first upper GIB scenario:
  - The endoscopist can see the culprit lesion, but cannot control it even with the wide range of endoscopic techniques available or the patient rebleeds after it is considered that maximal endoscopic treatment has been applied.
  - Patient should be transferred to the angiography suite during resuscitation, whilst actively bleeding to maximize the chance of treating the culprit lesion at the first attempt. Failure to be able to identify and treat the site of bleeding at the first angiography results in greater transfusion requirements if the patient rebleeds, longer hospital stay, increased risk of multi-organ failure, and cardiac and cerebrovascular events, increased radiation burden, and higher risk of contrast nephrotoxicity.
  - The endoscopist can assist the radiologist by placing endoscopic clips at the site of bleeding. Targeted embolization can then be performed even if bleeding has temporarily stopped and there are no angiographic clues to direct therapy.
- In the second upper GIB scenario:
  - The endoscopy is normal or there is too much blood and clot to allow endoscopic diagnosis.

- CT angiography (CTA) is highly sensitive and specific in these patients with a very low false positive rate provided the CTA is performed during resuscitation to maximize the chance of a diagnostic study.
- If a bleeding site is identified the patient should be transferred for immediate embolization.
- If the CTA is negative, it is almost always because the bleeding has stopped.
- Supportive treatment should be continued with a clear management pathway should the patient rebleed (usually either to repeat the endoscopy or the CTA).

Lower GIB is 4 times rarer than upper GIB, often less severe and more likely to be self-limiting. If haemodynamic compromise occurs CTA is the emergency investigation of choice after anorectal causes have been excluded by procto/sigmoidoscopy in patients presenting with bright red rectal bleeding. When active bleeding is seen embolization is usually the preferred to surgery in these patients who are often elderly with multiple co-morbidities.

Early trauma deaths

Around 60% of early trauma deaths are due to unrecognized or undiagnosed bleeding. Early CT (within 30min of arrival) has been shown to improve the survival of the severely injured patient (level 2 evidence). This covers the patient from head to mid-thighs or more distally if clinically indicated. Units that do not have this facility should not routinely receive trauma patients. Patients with haemodynamic evidence of active bleeding appear to derive the greatest benefit, but require senior clinicians to manage their care and transfer. Indecision in these patients can be lethal. Major trauma units must have standard operating procedures and management protocols for all clinical scenarios. In addition they should routinely practice transfer procedures. The only imaging that is indicated prior to a polytrauma CT scan is a CXR to check tube positions. Earlier CT diagnosis often identifies life-threatening bleeding before there has been haemodynamic compromise (particularly in young patients) reduces transfusion requirements, facilitates the use of treatment options with lower physiological impact than laporotomy and thoracotomy to control bleeding, and reduces the late sequelae of blood product resuscitation.

Thoracic aorta injury

Thoracic aorta injury (TAI) is the second most common cause of pre-hospital death in high impact blunt trauma after traumatic brain injury. Patients surviving to hospitalization are much rarer, but the aorta is the most commonly injured large vessel in the chest. It may seem counter-intuitive, but even severe injuries do not necessarily require immediate treatment. It is very rare for TAI to be actively bleeding on the CT scan; when seen it is usually rapidly fatal before any intervention can be performed. The purpose of intervention in TAI is to prevent rupture and late aneurysm formation. Minor intimal tears do not require early intervention and can be monitored for change by CT. If a patient is haemodynamically unstable other actively bleeding injuries (e.g. liver laceration and pelvic fractures) should be treated

before the aorta. There is compelling surgical literature showing that blood pressure control with a mean arterial pressure of 60–70mmHg reduces the rupture rate to close to zero, particularly if β-blockade is used to diminish the shear force. This means that intervention can be safely delayed to a routine daytime list. Patients who cannot have delayed repair are those who have an associated brain injury who require higher cerebral perfusion pressures and those in whom BP cannot be controlled. Meta-analysis of cohort studies show that morbidity and mortality are considerably lower if stent-grafting is used to repair the aorta and this has become the treatment of choice. Patients do not require routine spinal drainage due to the short length of aorta covered.

Post-embolization syndrome

Post-embolization syndrome (PES) is common after *solid organ* embolization; predominantly liver, spleen, kidney, and uterus. It should not occur when a stent graft has been used to control bleeding unless the stent-graft occludes.

The precise aetiology of PES is unproven. It is related to the release of inflammatory mediators and vasoactive substances after cell death. It comprises pain, fever (typically continuous <39°C) and nausea, and vomiting, which manifests as a 'flu-like' illness. Leukocytosis occurs in 20% in the first 24h and is usually <24 000/μL. It cannot be predicted in whom it will occur. Toxic mediators can aggravate contrast media induced nephrotoxicity. Management is supportive with hydration, analgesia, sedation, antiemetic therapy, and reassurance (remind patients it is an anticipated and self-limiting side effect). When the planned procedure has a high risk of PES patient-controlled analgesia should be prescribed at the outset along with non-steroidal analgesics and paracetamol for their analgesic, anti-pyretic, and anti-inflammatory (for the latter) properties.

It is important to distinguish it from infection, which should be suspected when high or spiking fever occurs or a high or late rise in leukocytes.

Complications of embolization

The complications of embolization depend on the organ or other territory treated, the quantity of tissue devascularized and the type of embolic agent used. Where there are concerns these should be discussed with a vascular radiologist.

Further reading

Kessel D, Robertson I. *Interventional Radiology. A Survival Guide*, 3rd edn. London: Churchill Livingstone 2011.

Uberoi R. *Oxford Specialist Handbooks in Radiology: Interventional Radiology*. Oxford: Oxford University Press 2009.

Chapter 9

Management of patients undergoing specific elective vascular procedures

Open aortic aneurysm repair

- *Procedure:* Open AAA repair is performed via a midline or transverse abdominal incision.
  - The aorta is exposed in the retroperitoneal space and the upper and lower ends of the aneurysm identified.
  - The upper limit of the aneurysm may be proximal to the renal, superior mesenteric arteries or coeliac axis; the lower limit may be distal to the aortic bifurcation and involve the iliac arteries.
  - The aorta is cross-clamped proximal and distal to the aneurysm to exclude it from the circulation.
  - Major collateral vessels (principally lumbar arteries) are identified and ligated to stop retrograde bleeding into the aneurysm sac.
  - A graft is sewn into the proximal aorta, tested for leaks by temporary unclamping, then sewn to the distal aorta.
  - A tube graft (AAA ends above bifurcation) or bifurcated (iliac) graft is used.
- *Time:* 2–5h.
- *Pain:* Severe. Thoracic epidural analgesia recommended. Alternatively, IV morphine infusion (in HDU or ICU) followed by patient-controlled analgesia (PCA) until adequate oral intake established. Thereafter, oral opioids for 3–7 days.
- *Position:* supine, arms positioned on arm boards to allow access to arterial and venous catheters.
- *Blood loss:* variable, typically 500–3000mL. May be sudden and massive. Intraoperative cell salvage should be used. XM 2–6U.
- *Hospital stay:* 5–10 days
- *Practical aspects:* routine interventions/monitors are:
  - Arterial and central venous catheters, temperature, urine output, acid-base status. Epidural catheter mostly used for post-operative analgesia.
  - Balanced GA technique with tracheal intubation and IPPV.
  - Maintain normothermia; active warming needed.
  - Consider NGT.
  - Take measures to attenuate cardiovascular responses to aortic clamping and unclamping, renal protection, and myocardial ischaemia.
  - Anticipate need for vasoactive drugs.
  - Extubate immediately post-operatively if patient is stable; otherwise post-operative IPPV may be required. Transfer to HDU or ICU.

Introduction

- The aorta is termed aneurysmal when its diameter is >3cm.
- Prevalence of aortic aneurysm increases with age. Around 10% of men and 3% of women aged >65yrs have an aortic aneurysm. As the population ages and with the introduction of the NHS Abdominal Aortic Aneurysm Screening Programme, it is likely that more patients with aortic aneurysms will present for surgery.

- Aortic aneurysm surgery poses significant challenges to both anaesthetist and surgeon, because of the nature and extent of the surgery and the prevalence of co-existing disease.
- Although open aneurysm repair using synthetic tube or bifurcated grafts is the established method for treating AAA, the use of endovascular techniques is increasing rapidly.
- Many units reserve EVAR for patients with significant comorbidity, but it is likely that as endovascular techniques improve, patient fitness for surgery will become less of a consideration in choice of technique.
- This section considers only open surgical repair. EVAR is considered in 📖 Endovascular abdominal and thoracic aortic aneurysm repair, p. 363.

Natural history of aortic aneurysms

- An aneurysm develops when the wall of a blood vessel weakens. Most aneurysms occur in the abdominal aorta, 90% are infra-renal. The rest are juxta-renal or supra-renal.
- *The underlying cause of AAA is:*
  - Atherosclerosis (90%).
  - Infection.
  - Trauma.
  - Medial degeneration (e.g. Marfan's Syndrome, syphilis).
- *Other risk factors include:*
  - *Smoking*—the prevalence of AAA in smokers is 4 times greater than lifelong non-smokers.
  - Hypertension.
  - Genetic predisposition.
- The risk of aortic rupture is related to the size of the aneurysm.
- Small aneurysms <5cm in diameter rarely rupture (<2%). There is no survival benefit from early surgical intervention. Patients with small aneurysms >3cm and <5.4cm in diameter should undergo regular screening by US scanning to monitor the aneurysm size.
- The estimated annual rupture rate of aneurysms >6cm in diameter is 9%, rising to >25% for aneurysms >8cm in diameter.
- Current guidelines are to offer surgery when the aneurysm is >5.5cm in diameter, provided the patient is deemed fit for surgery.
- 30-day mortality after open AAA surgery in the UK is currently 6–8%.

Pre-operative evaluation and investigation

- Patients presenting for AAA repairs have a high incidence of arterial and other co-existing diseases, in particular:
  - *Hypertension*—60%.
  - *Myocardial infarction*—50%.
  - *Chronic obstructive pulmonary disease (COPD)*—50%.
  - *Renal dysfunction*—25%.
  - *Angina*—20%.
  - *Congestive cardiac failure*—15%.
  - *Diabetes*—12%.
- In addition to a detailed history and physical examination, the following investigations should be performed routinely:
  - ECG.
  - CXR.

  - FBC and clotting studies.
  - Biochemistry.
  - Lung function tests and arterial blood gases (ABGs) on room air.
- Further investigations should be informed by the results of the history, examination and routine investigations (see 📖 Evaluation of the vascular surgical patient, p. 85). Tests performed depend on local policies and guidelines. They include:
  - Carotid artery flow studies.
  - Exercise ECG.
  - Echocardiography, including stress echocardiography.
  - Dipyridamole-thallium scanning.
  - Coronary angiography.
  - Renal function scanning ('Mag3' or similar).
  - Cardiopulmonary exercise testing (CPEX)—this is becoming increasingly popular, and the results can be used to estimate the risk of open surgery and inform decision-making processes about whether open surgery should be performed (see 📖 Dynamic testing and risk assessment, p. 136).
  - Patients with active signs or symptoms of IHD should be referred to a cardiologist.
- Medical therapy should be optimized:
  - All patients should receive antiplatelet medication (aspirin or clopidogrel) and a statin.
  - Some very high risk patients who demonstrate inducible ischemia on pharmacological stress testing have improved outcomes if they receive carefully titrated beta blockade (e.g. low dose bisoprolol titrated to a resting heart fate of 50–80beats/min).
- Percutaneous coronary intervention is very rarely indicated before AAA surgery:
  - CABG should only be performed prior to open AAA surgery only if indicated on conventional grounds for coronary revascularization, i.e. significant (>50%) left main stem stenosis, severe (>70%) two or three vessel disease and/or left ventricular systolic dysfunction.
  - Open AAA repair should be postponed for 3 months post-CABG if possible (see 📖 Coronary artery disease, p. 164).

Surgical techniques

- The majority of infrarenal AAA repairs are performed via a long midline or a sub-umbilical transverse incision. Midline incisions are quicker to perform, but may be associated with an increased incidence of incisional hernia.
- A left retroperitoneal is approach sometimes used if the patients have severe respiratory disease. The peritoneum is not opened; there is less post-operative ileus and reduced post-operative atelectasis. The main disadvantage of this approach is that access to the right iliac artery can be difficult, particularly if there is a large iliac aneurysm.
- The inferior mesenteric artery, which takes its origin from the front of the aneurysm, is usually sacrificed. Rarely, it may be reimplanted into the front of the aortic prosthesis using a Carroll patch if there are concerns about the adequacy of the circulation to the descending colon, sigmoid colon, and rectum.

- The aortic graft is made of woven Dacron.
  - The upper anastomosis is end to end.
  - The distal anastomosis may be to the aortic bifurcation (a 'tube' graft) or to the iliac or femoral arteries (a 'trouser' graft).
  - The site of the distal anastomosis(es) is dependent on whether the iliac arteries are involved in the aneurysm.
  - Wherever possible groin anastomoses should be avoided because of the increased risk of infection associated with groin incisions.

Pre-operative preparation

- Lifestyle changes should be encouraged, e.g. smoking cessation and weight loss as far as time allows before surgery. A structured exercise programme may help improve fitness.
- Patients should be provided with detailed information about the likely post-operative course including the necessity for HDU or ICU. If an epidural is planned to provide post-operative analgesia, risks and benefits should be explained and verbal consent obtained.
- Regular medications should be continued up to the day of surgery with the possible exception of ACE inhibitors or ARBs. Continuation of these drugs may be associated with refractory intra-operative hypotension.
- Most UK vascular surgeons are happy to perform open AAA repair in patients taking aspirin or clopidogrel. It is safe to insert an epidural catheter in a patient taking aspirin. The situation with clopidogrel is less clear. If an epidural catheter is inserted in a patient taking clopidogrel, careful documentation of the risk: benefit ratio is essential. Patients should be monitored closely for symptoms and signs of an epidural haematoma (hack pain, bladder dysfunction, leg weakness). The patient taking dual antiplatelet therapy following PCI is more problematic. Ideally elective surgery should be deferred until dual antiplatelet therapy is no longer required (3 weeks after balloon angioplasty, 6 weeks after BMS insertion, 1 year after DES insertion). If surgery for AAA is indicated within these time frames it has been suggested that clopidogrel should be stopped 7 days before surgery and bridging therapy instituted with a short acting platelet glycoprotein IIa/IIIb inhibitor (tirofiban, eptifibatide) stopping 6 hours prior to surgery (see 🕮 The anti-coagulated patient, p. 215).
- Warfarin should be stopped 5 days before elective aortic surgery and bridging therapy with heparin considered (see 🕮 The anti-coagulated patient, p. 215). However, the risks of perioperative haemorrhage are high and it may be appropriate to withhold both warfarin and heparin. This decision should be taken in conjunction with the surgeon and haematologist.
- Optimal glycaemic control is important to minimise the risk of diabetes related complications. Consider referral to the diabetic team if the HbA1C level suggests poor diabetic control (HbA1C > 70 mmol/l). Locally agreed guidelines for the peri-operative management of diabetes should be followed (🕮 Diabetes, p. 196).
- Blood should be cross-matched and be available before the start of surgery. Blood products, such as FFP, platelet concentrates, and cryoprecipitate should be available in accordance with local protocols.

In the anaesthetic room

The following should be placed:

- Mid-thoracic epidural catheter (if patient agrees and no contra-indications) placed before induction of anaesthesia.
- At least one large-gauge peripheral IV line (no smaller than 14G).
- Intra-arterial catheter.
- Temperature probe.
- Urinary catheter.
- Triple or quad lumen central venous line.
- The use of cardiac output monitoring and the computation of derived haemodynamic variables should be considered, especially in high-risk cases, to permit rational use of inotropes and vasopressors. There is some evidence from other forms of major abdominal surgery that haemodynamic management to achieve an optimal value of stroke volume may reduce post-operative complication rates and shorten hospital stay.

In the operating theatre

Monitoring

In addition to standard monitoring, direct measurement of arterial and central venous pressure, temperature, and urine output is mandatory:

- Continuous 5-lead ECG will improve detection of ST segment changes.
- Intermittent blood gas analysis.
- Rotational thrombo-elastometry to guide appropriate administration of blood products.

In addition, the following may be used:

- Transoesophageal echocardiography is used in some centres to detect systolic wall motion abnormalities, which are a sensitive indicator of myocardial ischaemia. Left ventricular volume status may be directly assessed, which may aid cardiovascular management in the high risk patient.
- *Non-invasive cardiac output monitoring devices:* there is no published evidence to support the use of the oesophageal Doppler in aortic surgery. The cardiovascular variables obtained before aortic cross-clamping and after clamp release may provide useful information to help optimize the circulation but the readings obtained are of no value when the aorta is cross-clamped. The LiDCO Rapid may have a role. It uses a patented PulseCO algorithm to derive haemodynamic variables from a radial arterial line trace without the need for prior calibration.
- Pulmonary artery pressure monitoring with thermodilution cardiac output measurement remains the gold standard and is particularly useful in patients with severely impaired ventricular function.

Equipment, drugs and fluids

- Forced-air patient warming device.
- IV fluid warmer.
- Rapid infusion device (Level 1/Belmont).
- Infusion pumps.
- Intraoperative cell salvage device.
- A range of vasoconstrictor, vasodilator and inotropic drugs should be available. A glyceryl trinitrate (GTN) infusion may be used to

manage acute hypertension associated with aortic cross-clamping and to promote venodilatation whilst the cross-clamp is applied to allow judicious vascular filling. This is switched off when cross-clamp release is imminent in anticipation of the sudden reduction in afterload.
- Appropriate antibiotics should be administered according to local protocols to minimize the chances of graft infection.

Key steps in anaesthetic management
- Individual cases should be discussed in advance and with the operating surgeon. The vascular MDT meeting is an ideal forum: images can be discussed with the vascular radiologist to help identify any unusual anatomical features and plan the surgical approach
- Aortic cross-clamping and unclamping can cause sudden, marked cardiovascular changes (see 📖 Pathophysiology of aortic clamping and unclamping, p. 48). These should be anticipated and steps taken to attenuate them.
- There is no evidence that any particular anaesthetic technique improves outcome. There is some evidence that volatile anaesthetics and opioids improve tolerance of myocardial ischaemia by their effects on mitochondrial and sarcolemmal ATP-regulated potassium channels. The mechanism may be similar to ischaemic preconditioning.
- The aim of anaesthesia is to have a normovolaemic, haemodynamically stable, normothermic, pain-free patient on completion of surgery.
- Most UK anaesthetists use a thoracic epidural to provide high quality post-operative analgesia and to help ameliorate the stress response to surgery. The decision whether to use the epidural intraopereratively is an individual one – some argue that an extensive intraoperative sympathectomy makes manipulation of the cardiovascular system more difficult
- Remifentanil administered as a target-controlled infusion is an increasingly popular technique. It provides cardiovascular stability and the capacity for rapid changes in plasma concentration in response to haemodynamic changes caused by alterations in surgical stimulation. Epidural analgesia can be established towards the end of the procedure once surgical haemostasis is complete.
- Baseline ABGs and clotting measurements should be taken shortly after the start of surgery.
- Heparin 70IU/kg. is given before the aortic cross-clamp is applied.
- Arterial pressure may be deliberately decreased before application of the aortic clamp in anticipation of post-clamping hypertension, which may be dangerous for patients with ischaemic heart disease or cerebrovascular disease. This is frequently achieved by a GTN infusion.
- The opening of the aneurysmal sac after aortic clamping can be associated with brisk haemorrhage due to 'backbleeding' from lumbar arteries, which arise from the back of the aorta. These should be promptly undersewn. The native aorta may be very friable and surgical haemostasis may be difficult to achieve. Further blood loss may arise from malpositioned clamps. Blood loss is variable—in the 2005 NCEPOD report, 7% of elective AAA repairs had a measured blood loss >5L. Blood and blood products should be immediately available.

The vascular anaesthetist should be familiar with local protocols for the management of massive blood loss (see 📖 Management of major haemorrhage, p. 244).

- Forced air warming should not be used on the legs when the aorta is clamped as these will have been rendered ischaemic.
- Moderate fluid loading during aortic clamping is popular to maintain normovolaemia (or slight hypervolaemia) in preparation for cross-clamp release.
- Once the upper anastomosis is completed the surgeon will move the cross-clamp from the native aorta to the aortic graft, exposing the suture line to aortic pressure. This manoeuvre requires close collaboration between surgeon and anaesthetist. A leaking anastomosis can cause brisk haemorrhage and may require reapplication of the aortic clamp onto the native aorta to control the bleeding.
- Once the upper anastomosis is 'dry' the surgeon will anastomose the distal end of the graft to the aortic bifurcation or to each iliac artery
- ABGs should be measured regularly during and after aortic clamping. A moderate metabolic acidosis may develop on cross-clamp release as the products of anaerobic metabolism in the ischaemic lower limbs are released into the circulation, This is managed by increasing minute ventilation and maintaining cardiac output.
- Some anaesthetists give additional intravascular volume or small doses of vasoconstrictors prior to cross-clamp release
- The surgeon should warn the anaesthetist when the lower anastomosis(es) are nearly complete and clamp release is imminent.
- The cross-clamp should be released slowly paying careful attention to the BP and cardiac output. Marked falls may be managed by partial reapplication of the clamp. If a ' trouser graft' is used the legs should be reperfused sequentially to minimize the haemodynamic perturbation
- The administration of blood and blood products should be guided by near patient testing using the Hemocue, rotational thrombo-elastometry and laboratory measurement of fibrinogen levels. Appropriate goals are Hb >90g/L, INR <1.5, platelet count >50 × 10^9/L and fibrinogen >2g/dL.

Post-operative management

- Traditionally, patients were admitted to ICU after AAA surgery to allow for a short period of post-operative ventilation. Careful patient selection following meticulous preassessment, coupled with improvements in anaesthetic and surgical techniques allows early extubation and immediate transfer to a HDU in the majority of cases.
- Early enteral nutrition is encouraged to maintain gut mucosal integrity and reduce bacterial translocation. Oral medications should be restarted as soon as possible.
- Appropriate antacid, thromboembolic and antibiotic prophylaxis should be prescribed according to local protocols.
- AAA patients should be closely monitored for cardiac events with serial ECG's +/– troponin measurements.
- The possibility of concealed post-operative bleeding should be borne in mind—drains are not routinely used. Patients must be closely monitored for signs of increasing abdominal distension or intra-abdominal hypertension.

- Arterial pressure should be carefully controlled after surgery. Marked hypertension may increase the bleeding risk from recently performed aortic anastomoses. Conversely, prolonged hypotension may lead to acute kidney injury or organ dysfunction.

Juxta- and supra-renal AAA surgery

The general principles described above are applicable to patients undergoing surgery on the more proximal aorta. The risks of surgery increase progressively with more proximal aortic surgery.

Types of aneurysm

- *Juxta-renal aneurysms:* aneurysm starts a short distance distal to the renal arteries, but it is usually possible to place a clamp between neck of aneurysm and renal arteries. As aneurysmal sacs often contain friable, but solid matter, it is possible to dislodge some of this material into the renal arteries, which may produce renal failure ('trash kidney').
- *Supra-renal aneurysms:* originate at or just below the renal arteries and require a supra-renal clamp to allow a graft to be inserted. One or both renal arteries may need to be re-implanted into proximal graft.
- *Thoraco-abdominal aortic aneurysms (TAAAs)* (Fig. 9.1): classified by Crawford and modified by Safi as*:
  - *Type I TAAA*—extends from just distal to the left subclavian artery into the proximal abdominal aorta above the level of the coeliac axis. Since this aneurysm ends before origin of visceral and renal arteries, there is usually no need for re-implantation of these arteries.
  - *Type II TAAA*—extends from just distal to the left subclavian artery to the aortic bifurcation and carry greatest risk of complications. Re-implantation of renal and visceral vessels necessary.
  - *Type III TAAA*—begins in the lower part of the descending thoracic aorta, classically at the 6th intercostal space to the aortic bifurcation. Visceral and renal vessels will need to be re-implanted. It is sometimes possible

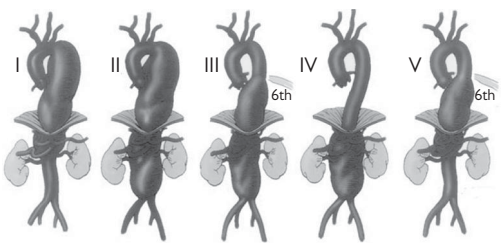

Fig. 9.1 Thoracoabdominal aortic aneurysm classification.

Reprinted from The Annals of Thoracic Surgery, 66, 4, HJ Safi et al., 'Effect of extended cross-clamp time during thoracoabdominal aortic aneurysm', pp. 1204–1208, Copyright 1998, with permission from The Society of Thoracic Surgeons and Elsevier.

*Reprinted from Cardiovascular Surgery, 7, 6, HJ Safi, 'How I do it: thoracoabdominal aortic aneurysm graft replacement', pp. 607–613, Copyright 1999, with permission from Elsevier.

to do this by leaving the vessels attached to a 'patch' of native aorta, but often the vessels will need to be re-implanted separately or in pairs.
- *Type IV TAAA*—begins at the diaphragm (12th intercostal space) to the aortic bifurcation. This is an abdominal aortic aneurysm that needs placement of the aortic clamp on the intra-thoracic aorta. It is often possible to leave the visceral and renal arteries attached to the native aorta, but re-implantation is sometimes necessary. A thoracotomy is not usually needed, but the diaphragm may need to be opened to allow access for the clamp.
- *Type V TAAA*—from the level of the 6th intercostal space of the descending thoracic aorta to just above the renal arteries. Reimplantation of the some visceral vessels may be required.

Thoraco-abdominal aortic aneurysm (TAAA) repair

- *Procedure:* thoracotomy and laparotomy incisions. Approach and procedure variable depending on the anatomy of the aneurysm.
- *Time:* 4–8h.
- *Pain:* severe.
- *Position:* thoracic epidural analgesia usual. Alternatively, high dose opioids (e.g. IV morphine infusion (in HDU or ICU) followed by PCA until adequate oral intake established (4–7 days). Oral opioids thereafter for 3–7 days.
- *Blood loss:* high, can be catastrophic. Intraoperative cell salvage mandatory. XM 12–16U; other blood products invariably needed.
- *Hospital stay:* 10–20 days.
- *Practical aspects:* very high risk surgery; should be performed in specialized centres
  - Considerations similar to AAA, but cardiovascular changes more marked. Anticipate massive blood loss.
  - Multiple large bore cannulae required.
  - Specific measures for organ protection (abdominal organs, spinal cord) needed including bypass procedures and CSF drainage.
  - Spinal cord monitoring during and after surgery.
  - Continue artificial ventilation and transfer to ICU for post-operative care.

Introduction

- The majority TAAAs are caused by degenerative aortic disease:
  - 5–10% are 2° to Marfan's syndrome.
- Other causes include:
  - Trauma.
  - Takayasu's aortitis.
  - Previous repair of aortic coarctation.
  - Ehlers–Danlos syndrome.
- Most patients with TAAAs are asymptomatic. Almost all are hypertensive. The first symptoms are often those associated with rupture, which has a very high mortality.

- Common presentations include:
  - Incidental diagnosis following CXR or other imaging.
  - Chest pain, caused by enlargement of the aneurysm or compression of adjoining structures.
  - Breathlessness and dysphagia if the lungs or oesophagus are compressed.
  - GI bleeding or haemoptysis caused by erosion into adjacent structures.
  - Thrombotic or embolic phenomena if atheromatous material is released from the aneurysmal sac.

Indications for surgery

- The threshold for treatment of TAAA is an aneurysm diameter of 5–6cm.
- Open surgical repair is a massive physiological insult. 30-day mortality rates are ~ 19% with 1-yr mortality rates as high as 31%. The 5-yr mortality rate is approximately 40% for all elective cases and >80% for ruptured TAAA.
- Despite high complication rates, the definitive treatment for TAAA remains surgical repair. The role of medical management is limited because TAAAs continue to expand despite adequate control of hypertension.
- Because of the risks of open surgery, patients with TAAAs are increasingly considered for thoracic endovascular aneurysm repair (TEVAR) or a combination of open and endovascular repair ('hybrid' procedures 📖 Endovascular abdominal and thoracic AA repair, p. 363, or Endovascular AAA and TAAA repair, p. 358)
- Not all TAAAs are anatomically suitable for TEVAR. Continued improvements in stent technology and the introduction of fenestrated and branched stent grafts have increased the number of TAAAs that are suitable for endovascular repair.
- Predictors of adverse outcome associated with TAAA repair are:
  - Pre-operative renal insufficiency.
  - Increasing age.
  - Symptomatic aneurysms.
  - Rupture at presentation.
  - Post-operative renal failure.
- Although there are no absolute contra-indications to open TAAA surgery, a considered approach to the possible risks and benefits of surgery is vital.

Preoperative evaluation and preparation

- Preoperative evaluation and preparation are as for AAA repair, but with a lower threshold for invasive testing. Morbidity and mortality are higher after TAAA repair because:
  - Surgical approach almost always requires a thoraco-laparotomy (except for juxtarenal, suprarenal or some Type 4 TAAAs). One-lung ventilation is often needed.
  - Cardiovascular effects of aortic cross-clamping are greater with more proximal clamping.
  - Risks of severe haemorrhage and coagulopathy are much greater.
  - Surgery is more prolonged.

  - Visceral ischaemia is common during and after surgery. This requires additional measures to maintain visceral and spinal cord blood flow during and after surgery (see 📖 Spinal cord protection, p. 369) and to protect abdominal organs from ischaemia-reperfusion injury.
  - Specific bypass techniques used to maintain organ perfusion carry their own risks and complications
- Because the risks are higher, meticulous preoperative assessment and discussion of the potential risks and benefits are particularly important. Co-existing medical conditions must be optimized fully. The patient should attempt to improve their physical fitness, if possible with a graded exercise programme.

Anaesthetic management

- The general considerations relating to management of anaesthesia and intraoperative management are similar to AAA repair.
- All patients require post-operative ICU admission.
- The likely need for ongoing organ support (post-operative ventilation, cardiovascular support, renal replacement therapy, spinal cord protection) is higher than for infrarenal AAA surgery.

Key steps in anaesthetic management

Discuss each case in advance and in detail with the surgeons in order to identify any unusual anatomy, or unexpected steps, or procedures.

Airway and ventilation

- Formal thoracotomies are performed, as the proximal anastomoses in Type I and II TAAAs (and some Type III TAAAs) are often difficult to perform with the left lung being ventilated. A double lumen tube or bronchial blocker is required to facilitate surgical access.
- One-lung ventilation may be associated with hypoxaemia and hypercapnia particularly in patients with co-existing COPD. Continuous positive airway pressure and/or insufflation of low flow oxygen into the collapsed lung may be required (see 📖 Intra-operative management of OLV, p. 271). Rarely, intermittent reinflation of the collapsed lung may be required. Such manoeuvres require close collaboration with the surgeon to ensure that inflation of the lung does not interfere with surgery.

Cardiovascular management

- The maintenance of haemodynamic stability is paramount during TAAA surgery due to the increased ischaemic risks to the spinal cord and viscera.
- Arterial hypertension is more severe after aortic cross-clamping during TAAA repair because of the proximal placement of the clamp. Vasodilators should be used in advance of aortic clamping to minimize the overshoot in BP. GTN is popular, but very proximal clamps may require the use of sodium nitroprusside and/or esmolol to produce acceptable cardiovascular stability.
- Arterial hypotension after de-clamping is also more marked than after distal AAA repair. Infusions of adrenaline and/or noradrenaline are commonly used to prevent dangerous degrees of hypotension. Staged or intermittent off-clamping can be particularly useful, with surgeon

maintaining some degree of manual restriction of blood flow through the graft while the anaesthetist strives to normalize the circulation.
- Although left thoracotomies allow direct visualization of myocardial movement, trans-oesophageal echocardiography should be seriously considered for all patients, particularly for those with valvular disease or poor ventricular function.
- Heparin may be requested by the surgeon before aortic clamping. However, those surgeons who use a 'clamp and sew' technique without bypass, rarely request heparinization.
- The greater extent and duration of visceral and body ischaemia means that the administration of sodium bicarbonate is almost always necessary. Regular arterial blood gas and acid-base measurements should be performed, and bicarbonate given as necessary.

Blood loss and management of coagulation
- Blood loss during open TAAA surgery can be substantial, and a short, wide-bore peripheral or central line that allows rapid transfusion should always be placed. The use of a double-lumen haemodialysis catheter is useful- this can be used for rapid infusion and the management of post-operative AKI.
- Intraoperative cell salvage should be used routinely.
- Rapid infusion systems are required.
- Large volumes of blood and blood products will often be needed. Adequate supplies of packed red cells, fresh frozen plasma, platelet concentrate, and cryoprecipitate should be available.
- Although formulaic administration of blood and blood products has been recommended, the use of rotational thromboelastometry and platelet function analysers are strongly recommended to guide rational administration of blood products and antifibrinolyic agents.
- The involvement of a specialist haematologist is highly desirable, as failure to maintain an acceptable coagulation profile is a common source of severe problems.
- Some units advocate the prophylactic administration of aprotinin before open TAAA surgery.

Temperature management
- Moderate hypothermia to 34–35°C may help protect spinal cord function by decreasing oxygen demand. It may also decrease the adverse metabolic results of aortic clamping.
- This can be achieved by passive heat loss: temperature management can be more accurately manipulated if bypass circuits are used. Forced air warming and fluid warmers should still be used as rewarming can be very difficult when body cavities are so widely exposed.

Spinal cord protection
- The risk of paraplegia with extensive TAAAs is up to 25%. Main factors predisposing to paraplegia are the type of aneurysm (highest incidence with Type II aneurysms) and duration of aortic cross-clamping.
- Specific bypass procedures are often required (see 🕮 Bypass procedures, p. 362) and have been shown to reduce the incidence of paraplegia.
- To reduce the incidence of paraplegia, spinal drainage (see 🕮 Spinal cord protection, p. 369) should be used for all thoracic aneurysms, with

the possible exception of Type IV TAAAs (after discussion with the surgeon). Paraplegia is possible after surgery for supra-renal abdominal aortic aneurysms but is rare.

- Monitoring of somatosensory or motor-evoked potentials (MEPs) may allow the identification of spinal cord ischaemia and the re-implantation of important nutrient vessels that have been occluded during surgery. Motor-evoked potentials have the advantage that they monitor the function of the anterior part of the spinal cord that is most vulnerable to ischaemia. Although some units use these routinely, their use is not widespread.
- Clamp time should be minimized. Most Type I and Type IV aneurysms can be managed with two aortic anastomoses. However, in Type II and Type III aneurysms, additional visceral and renal anastomoses inevitably increase ischaemic clamp time and therefore the increase risk of spinal cord injury.
- Maintenance of adequate arterial pressure, particularly during aortic clamping, will improve spinal cord perfusion pressure (qv). Moderate hypothermia will decrease spinal cord oxygen demand. Some anaesthetists place epidural catheters in preparation for post-operative use, but fewer inject epidural local anaesthetic lest immobility in the legs caused by neuraxial blockade be confused with paralysis resulting from spinal cord ischaemia.

Bypass procedures

- A variety of different bypass procedures have been used to maintain blood flow to the distal aorta in an attempt to decrease the incidence of spinal cord ischaemia and to minimize the adverse cardiovascular and metabolic effects of high aortic clamping.
- Techniques that draw blood from the left atrium may also provide adjustable mechanical unloading of the left ventricle, which may be of assistance to patients with poor left ventricular function.
  - Blood drawn from above the proximal aortic clamp and returned to the femoral artery can perfuse the aorta retrogradely, up to the level of a second aortic clamp placed just below the site of the upper aortic anastomosis.
  - This allows perfusion of the viscera, kidneys, some arteries that may supply the spinal cord and the legs during the proximal anastomosis.
  - As the lower clamp is moved down the aorta during the re-implantation of visceral and renal arteries, such a bypass can still provide blood flow to some nutrient arteries and the legs.
  - The use of bypass tubing and a heat exchanger/oxygenator with an anti-thombotic coating enables a lesser degree of anticoagulation to be used than for full cardiopulmonary bypass

Bypass strategies include:

- *Passive surgical shunts*—from an artery proximal to the aneurysm to a femoral artery.
- *Partial left heart bypass*—the left atrium is cannulated and oxygenated blood is pumped, often with a centrifugal, non-occlusive pump, into a femoral artery.
- *Left heart bypass*—with insertion of separate pump outputs into the origins of the visceral and renal vessels.

- *Full cardiopulmonary bypass*—usually reserved for ascending or arch aortic aneurysms. This requires full anticoagulation, and can associated with marked intra-operative and post-operative haemorrhage.

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Endovascular abdominal and thoracic aortic aneurysm repair

- *Procedure:* a stent-graft is introduced retrogradely into the lumen of the aorta via one or both femoral arteries to occlude the aneurysm from with the aortic lumen. The position of the graft is determined radiologically and angiographically. The stent-graft expands when the delivery device is withdrawn to fix itself to the arterial wall; an intraluminal balloon often used to mould the graft.
- *Time:* 1–3h.
- *Pain:* minimal. Local anaesthetic infiltration, +/– low dose morphine or simple analgesics (weak opioids/NSAIDs) during surgery usually sufficient for post-operative analgesia
- *Position:* supine.
- *Blood loss:* usually minimal, but often insidious. XM 2 units.
- *Hospital stay:* 2–5 days.
- *Practical aspects:* balanced GA, spinal, combined-spinal epidural (CSE), epidural, or local infiltration +/– sedative adjuncts. If using an awake technique, the patient must be able to breath-hold during stent positioning and deployment

Introduction

- *Endovascular aneurysm repair (EVAR):* increasingly used to treat aortic aneurysms and dissections. EVAR involves the placement of a self-expanding stent graft inside the aneurysm to exclude it from the circulation. Access to the aorta is usually via the femoral or brachial arteries, obviating the need to perform a laparotomy or thoracotomy.
- Many vascular surgery units have reserved EVAR for anatomically suitable aneurysms in patients thought to be at high risk from open aortic surgery. However, as techniques and technologies develop, the use of EVAR is likely to increase and become the first choice treatment for many patients with aortic aneurysm.
- Perioperative morbidity and mortality are lower after EVAR compared with open AAA surgery.

- However, current evidence suggests that long-term survival rates after EVAR or open surgery are similar, because the early survival benefit with EVAR is balanced by an increase in late mortality from co-existing cardiovascular and respiratory disease.
- Some patients will still require open surgery because the morphology (size, shape, and position) of the aneurysm makes it unsuitable for EVAR, either because of potential leaks or difficulties positioning the graft. All patients with an AAA undergo CT angiography to determine potential suitability for EVAR.

Aneurysm morphology

- Successful EVAR requires that the aneurysm have certain anatomical characteristics. These have altered with time as device manufacturers have produced more sophisticated stent grafts.
- Diameter of the aneurysm neck <32mm.
- Conventionally, the aneurysm neck length should be >15mm below the renal arteries. However, modern stent devices enable aneurysms with shorter necks to be offered EVAR.
- Originally, the neck angulation needed to be <45°, but newer devices allow aneurysms with neck angulations up to 90° to be stented.
- Modern low profile devices allow the deployment device to be inserted into iliac arteries as small as 6mm in diameter. There should be minimal ectasia or tortuosity of the iliac vessels.
- An occluded inferior mesenteric artery and small or absent lumbar arteries are beneficial as they reduce the risk of type II endoleak (retrograde filling of the aneurysm sack via branch vessels).

Overall, ~ 80% of infrarenal AAAs fulfill the anatomical criteria for EVAR. Earlier indications included that the AAA did not involve the renal arteries but advances in technology have led to the successful use of grafts with a bare suprarenal component that can be deployed without affecting renal artery flow and renal perfusion. See Fig. 9.2.

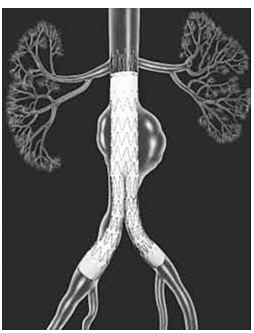

Fig. 9.2 Infrarenal abdominal aortic aneurysm with endovascular aortic stent-graft in place. Reproduced from Hands *et al.*, *Vascular Surgery*, 2007, Figure 13.3, p. 245, with permission from Oxford University Press

Devices used for EVAR

- Current stent grafts are typically made from a self-expanding metal skeleton, often a nickel-titanium alloy, over which is placed a woven fabric that is impervious to fluid. They are compressed within delivery tubes placed into the aorta, usually via the femoral artery. They are delivered precisely into position within the aorta under radiographic and angiographic control.
- The stent grafts expand as they emerge from the delivery systems and can be further expanded by balloons inflated within their lumens to ensure that they finally reach the correct degree of expansion. The proximal ends of stents often have hooks or anchors that allow them to attach themselves to the wall of the aorta and avoid distal migration.
- Long sections of aorta can be stented by placing the proximal end of a second or subsequent graft into the distal end of grafts already positioned in the aorta.
- Fenestrated grafts allow the creation of bifurcated grafts to be placed into the iliac arteries, and other fenestrations can allow selective stenting of renal or visceral vessels.
- Stents are usually placed via the common femoral artery. Access usually obtained by vascular surgeon, normally with formal dissection.
- A single tube stent may require exposure of only one femoral artery, but bifurcated and other more complex grafts will require surgery to both femoral arteries. Occasionally, stent placement may be facilitated by placing guide wires via a brachial artery.

Comparing endovascular and open aneurysm repair

Advantages of EVAR

- General anaesthesia can often be avoided.
- Procedures are usually of shorter duration than open procedures.
- Pain is usually minimal and the surgical trauma of laparotomy and thoracotomy is avoided.
- Minimal haemodynamic and metabolic disturbances.
- Fewer adverse effects on respiratory and renal function.
- Decreased blood and blood product transfusion requirements.
- Reduced requirement for Level 2 and 3 care, and reduced hospital stay.
- Lower 30-day mortality than open surgery.
- EVAR can be performed on patients unfit for open AAA repair.
- Several of these advantages are even greater in patients with thoracic aortic aneurysms (see 📖 Disadvantages of EVAR, p. 365).

Disadvantages of EVAR

Higher need for re-intervention compared with open repair to treat complications, including:

- *Endoleak:* where blood leaks around the stent into the aneurysm sac. The blood is at arterial pressure and hence the aneurysm can continue to expand with the risk of rupture. This occurs in up to 20% of patients
- *Distal migration of the device:* up to 2.5% of patients.
- Graft thrombosis.
- Graft kinking and ∴ narrowing or obstruction.
- High costs of the device (but lower hospital costs).

- *Some complications:* e.g. buttock claudication or impotence if the internal iliac arteries are occluded, are more common after EVAR.
- *Annual computerized tomography surveillance:* required after EVAR. The costs of surveillance and re-intervention mean that overall costs are similar between open repair and EVAR.
- Currently evidence suggests there is little long-term survival benefit between EVAR and open repair, but further studies are ongoing and endovascular techniques are improving. ∴ this situation may change.

Anaesthesia for EVAR

- Pre-operative assessment, investigation and preparation are the same as for any patient with an aortic aneurysm 📖 Evaluation of the vascular surgical patient, p. 85).
- Patients are often selected for EVAR because they are in a high-risk group for open repair and will ∴ have a higher incidence of comorbidities, such as significant cardiovascular and respiratory disease.
- Aneurysm or iliac artery rupture may occur during the procedure, although this is very rare. The anaesthetist should be prepared for the emergency management of these problems should they occur. It is advisable to insert at least one large-bore peripheral IV catheter and an arterial catheter. The need for other monitoring or access devices, e.g. central venous lines, depends on the patient's comorbidities and surgical complexity.
- EVAR procedures often require only infra-inguinal incisions in the groin. In this case, EVAR can be performed under local anaesthetic infiltration by the surgeon. However, the size of the delivery systems and snares placed around the arteries mean that leg ischaemia is common, and the subsequent pain can be difficult to manage; use of a low dose remifentanil infusion during surgery has been described. For this reason, spinal, CSE, epidural and continuous spinal anaesthesia (CSpA) are often used if they are not contra-indicated.
- Alternatively, EVAR can be performed under GA. Delivery system placement through a brachial artery will almost always cause painful ischaemia that is difficult to manage without brachial plexus anaesthesia or general anaesthesia. Another advantage of GA is that it avoids patient movement during imaging and allows apnoea when static imaging is required.
- There is no good evidence that the choice of anaesthetic technique has a substantial impact on overall outcome. The type of anaesthesia should be determined by anaesthetic considerations, e.g. the presence and severity of co-existing diseases, surgical considerations (type site and duration of EVAR), local facilities, and expertise.

Specific problems

- *Blood loss is rarely rapid during successful EVAR:* it is usually insidious, but sometimes substantial. Leakage of blood from around arterial access sites is common and can go unnoticed if the operators are concentrating on the radiological images.

- *Repeated angiography during EVAR:* may require the administration of large volumes of radio-contrast. This can potentially cause contrast-induced nephropathy and appropriate precautions should be taken, principally maintenance of adequate circulating volume and arterial pressure (📖 Contrast-induced nephropathy and renal protection, p. 338).
- Short periods of apnoea are usually needed during stent placement and, if EVAR is performed under local or regional anaesthesia, the patient must be able to co-operate. The anaesthetist provides apnoea during EVAR under GA with controlled ventilation; *Note:* remember to restart ventilation when the imaging is completed as radiologists will often forget to remind you.
- *IV heparin:* 3–5000IU may be requested.
- Some surgeons and radiologists request brief periods of relative hypotension immediately before stent deployment. This is rarely necessary except for very proximal stents, when it may help to prevent early distal stent migration.
- Aortic occlusion occurs briefly during stent deployment and inflation of angioplasty balloons. Although these periods are short, cardiovascular changes can sometimes be observed.

Location

- EVAR can be performed in operating theatres with mobile imaging devices or in radiology/imaging departments or angiography suites. Wherever EVAR is performed, the standards of anaesthetic and surgical equipment, monitoring and infection control should be equivalent to those in the vascular operating theatres.
- These include adequate space, facilities, and equipment to provide GA and invasive pressure monitoring. When the EVAR suite is separate from the radiology suite, anaesthesia should only be administered by an appropriately qualified anaesthetist competent in providing anaesthesia in an isolated location.
- A dedicated and fully trained anaesthetic assistant with specific training and experience in vascular anaesthetic practice should assist the anaesthetist.
- Post-operative recovery facilities should be identical to those provided after major surgery in the operating theatre suite, with access to HDU/ICU care and renal replacement therapy on-site.

Post-operative care

Post-operative admission to the ICU is rarely necessary, unless indicated by co-existing disease. Post-operative monitoring on the vascular ward should include specific attention to BP, SpO_2, and urine output.

TEVAR and hybrid procedures

- Aneurysms of the descending thoracic aorta have traditionally been treated using a thoraco-abdominal surgical approach.
- These have a very high morbidity and mortality (up to 25–30% for some extensive aneurysms).
- Endovascular approaches are very appealing, but purely endovascular techniques can only be used for isolated thoracic aneurysms when stent placement will not occlude renal or visceral vessels.

- A combined or 'hybrid' approach to a thoraco-abdominal aneurysm is to stent the aneurysmal aorta after performing a laparotomy, during which the visceral and renal vessels are anastomosed to large arteries distal to the lowest placement of the stent (the distal aorta or more commonly one of the iliac vessels).

Anaesthesia for TEVAR

- If performed solely through groin incisions, the anaesthetic considerations for TEVAR are identical to those for EVAR. LA, RA, or GA may be used. Monitoring depends on the patient's co-existing morbidity and complexity of surgery. Interventions to reduce arterial pressure during proximal stent deployment may be required.
- Pain and post-operative morbidity are relatively low. There are however reports of patients experiencing significant pain when intercostal arteries are occluded by thoracic aortic stents.
- The incidence of spinal cord injury is lower in TEVAR than in open surgery, ~4% compared with 6–12%. However, many anaesthetists will consider the insertion of a spinal drain if extensive coverage of the thoracic aorta by stents is planned, as intercostal vessels supplying the spinal cord will be occluded by the graft.

Anaesthesia for hybrid TEVARs with laparatomy

- For hybrid procedures, a formal laparotomy is performed in what may be a very long procedure; general anaesthesia is usually given with or without supplemental epidural analgesia.
- All lines, monitoring, blood, blood products, cell savers and infusion equipment appropriate for an open TAAA should be used in hybrid TEVARs. Large bore venous access, vascular monitoring lines, and infusion equipment should be in place in case the aneurysm ruptures. One-lung ventilation is not usually required and a single-lumen tracheal tube will suffice.
- During the phase of the operation when visceral and renal vessels are re-attached to the aorta or iliac vessels, there will be periods of major vessel clamping and ischaemia. The anaesthetist should be prepared to manipulate the circulation to prevent significant changes in cardiac output and BP, and to manage the consequent metabolic derangements.
- Blood loss can be marked during both phases of the surgical procedure—during the laparotomy and re-attachment of the visceral and renal vessels, and during the stenting phase. The thoracic and abdominal aortic stents are often placed through a side branch of the trifurcated or quadrifurcated graft used to re-anastomose the visceral and renal vessels. The anaesthetist should be prepared for large volumes of blood loss as the delivery systems pass through this side branch.

Location

The entirety of a hybrid TEVAR procedure is best performed in an operating theatre. Rarely, the higher quality radiological equipment sited in Imaging Departments will be needed. Some large units are investing in purpose built 'endo theatres' that combine the radiological facilities of a dedicated angiography suite with the facilities of an operating theatre.

Post-operative care

Hybrid procedures carry significantly higher risks than TEVAR or EVARs. They are major abdominal procedures in high-risk patients that carry the possibility of significant cardiovascular, haematological, and metabolic disturbances, together with a risk of paraplegia.

Early post-operative care after hybrid TEVAR should be in a HDU or ICU.

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Spinal cord protection in aortic surgery

Paraplegia resulting from interruption to the blood flow to the spinal cord is a significant complication of aortic surgery. The reported incidence is up to 40% in some historical series of extensive open thoracic aneurysm procedures. It has an enormous impact on the patient: those who become paraplegic have a high perioperative mortality. Although the factors associated with its occurrence are known and a number of strategies for minimizing its occurrence have been devised, it remains a feared and relatively common complication of aortic surgery. The advent of endovascular aortic surgery has decreased the incidence of ischaemic spinal cord injury, but has not eliminated it.

Blood supply to the spinal cord

- Blood supply to the spinal cord derives from three longitudinal arteries—two posterior and one anterior spinal artery (Fig. 9.3).
- Anterior spinal artery forms from vertebral arteries, runs length of spinal cord and supplies anterior two-thirds of the spinal cord.
- There is little overlap between the territories supplied by anterior and posterior arteries, and if blood flow in anterior artery is significantly decreased, this territory is rendered ischaemic, resulting in anterior spinal artery syndrome.
- Anterior spinal artery syndrome causes paralysis with loss of pain and temperature sensation below the level of the lesion. Touch, vibration, and position sensation are preserved as they are mediated by tracts in the posterior spinal cord (Fig. 9.4).

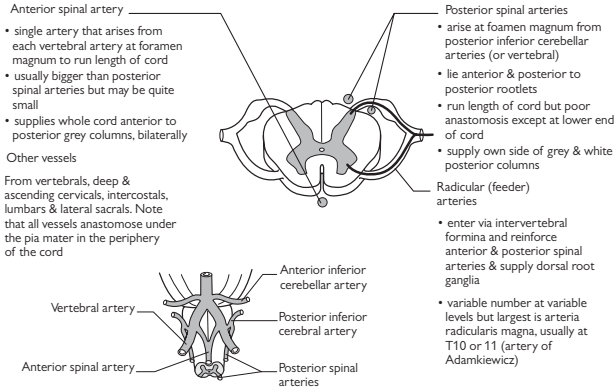

Fig. 9.3 The blood supply of the spinal cord.

Reproduced from Smith *et al. Oxford Desk Reference: Major Trauma* 2010, fig. 11.7, p. 169, with permission from Oxford University Press.

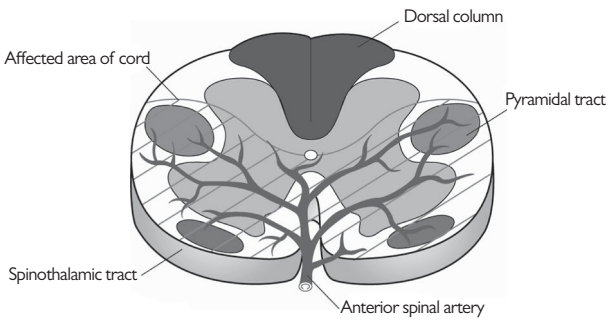

Fig. 9.4 Anterior cord syndrome. There is infarction of the spinal cord in the distribution of the anterior spinal artery, causing complete loss of motor function, and pain and temperature sensation below the lesion. Vibration and joint position sense are retained. This injury pattern has the worst prognosis of the incomplete injuries.

Reproduced from Collier *et al.*, *Oxford Handbook of Clinical Specialties* 2009, fig. 4, p. 769, with permission from Oxford University Press.

- The spinal arteries depend upon segmental arterial contributions along the length of the spinal cord and it is the interruption of these arteries that is responsible for spinal cord ischaemia. Although the cervical portion of the cord has a relatively reliable blood supply from vertebral arteries, thoracic, and lumbar regions are dependent upon a series of variable intercostal and lumbar arteries, supplemented by lumbar, iliac, and sacral branches, and segmental medullary arteries.

- The largest of these segmental arteries is the artery of Adamkiewicz, or the *arteria radicularis magna*. This has a variable origin between T5 and L3; in most patients it arises between T9 and T12.
- Aortic cross-clamping causes an increase in aortic pressure proximal to the clamp and a marked decrease in aortic pressure distal to the clamp. Distal aortic pressure is the major determinant of spinal cord blood flow during the period of aortic clamping (see 🕮 Methods used for spinal cord protection, p. 372) during thoracic aneurysm repair.
- If the area of aorta being replaced includes the major collateral supply to the anterior spinal cord (more likely during descending thoracic or supracoeliac aneurysm repair), this may be occluded by the aortic graft itself. When interruption to the arterial supply to the spinal cord occurs because of vascular occlusion, whether surgical or non-surgical, the lesions most often occur in 'watershed' areas in the mid-thoracic or high lumbar areas.

Factors associated with spinal cord injury in open aortic surgery

- Extensive aneurysm (thoracic>supracoeliac>infrarenal).
- Long aortic clamp times (incidence ↑ after >30min and ↑↑↑ after 60min).
- Hypotension.
- Emergency procedures.
- Spinal collateral vessel ligation.
- Previous aortic aneurysm surgery.
- Severe atherosclerosis.
- Diabetes.
- Old age.

Spinal cord protection

Monitoring

- Spinal cord function can be monitored during surgery using somatosensory or MEPs.
- Both require the assistance of specialist neurophysiological technicians, but SSEPs are slightly easier to monitor and are more reliable.
- Both SSEPs and MEPs are affected by factors such as the presence of inhalational anaesthetic agents, temperature, and other drugs.
- SSEPs monitor conduction in (and reflect blood flow through) the posterior part of spinal cord, i.e. the portion that is at least risk during aortic cross-clamping. They may detect ischaemia in the 'watershed' areas of the cord between areas supplied by the anterior and posterior spinal arteries. However, several incidences of paraplegia have occurred despite normal SSEP values and they are probably not useful in preventing neurological injury during aortic surgery.
- MEPs monitor conduction in the anterior and lateral parts of the spinal cord supplied by the anterior spinal artery. They are readily affected by neuromuscular blocking drugs, epidural local anaesthesia, and distal muscle ischaemia. However, some units have reported excellent results when they have re-implanted nutrient blood vessels in response to decreases in neurological activity by MEPs.

Methods used for spinal cord protection

Surgical

- *Preserving the anatomical blood supply to the spinal cord:* involves meticulous identification and preservation of nutrient arteries, or re-implantation during surgery.
- Minimizing the duration of aortic occlusion time.
- Minimizing blood loss and intraoperative hypotension.
- *Distal circulatory support:* to provide blood supply to the distal portion of the cord during proximal clamping and anastomosis. Various methods are available including:
  - Insertion of a shunt between proximal aorta above the clamp and distal aorta below the clamp.
  - Left atrio-femoral artery bypass.
  - Femoral vein to femoral artery bypass with an oxygenator.
- The effects of distal perfusion techniques are limited because they do not affect perfusion between the proximal and distal aortic clamps.

Non-surgical

- *Maximizing spinal cord perfusion pressure (SCPP):*

  SCPP = MAP – (CSF pressure or CVP, whichever is greater)

  SCPP is best achieved by maintaining arterial pressure distal to the clamp, with concurrent drainage of CSF.
- *Minimizing spinal cord oxygen demand:* using passive or active local or global cooling. Selective cooling of the spinal cord by infusing cold fluids into the epidural space was reported to produce excellent results, but these have not been confirmed in subsequent studies.
- *Avoiding hyperglycaemia:* may be associated with poorer neurological outcome.
- Avoiding post-operative hypotension.

Pharmacological methods

A number of pharmacological strategies have been tried but there is little conclusive proof that any is superior or effective in clinical practice.

- *Barbiturates ('spinal coma'):* decrease neural tissue oxygen demand, and also have membrane-stabilizing and free radical scavenging effects that may be useful in minimizing reperfusion injury.
- *Corticosteroids:* may reduce spinal cord oedema and stabilize cell membranes, but may cause hyperglycaemia.
- Naloxone, nimodipine, mannitol, allopurinol, Ca^{2+} channel blockers, papaverine, and magnesium have also been studied.

A pragmatic approach to spinal cord protection

Most of the methods described above are likely to have synergistic effects, and individual vascular surgical units will have favoured combinations. As a reasonable minimum during extensive aortic surgery, the following are recommended:

- Close attention to the maintenance of MAP.
- CSF drainage (📖 Cerebrospinal fluid drainage, p. 264).
- Passive global hypothermia to around 34–35°C.
- Avoidance of hyperglycaemia.

Further reading

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Aorto-iliac occlusive disease

- *Procedure:* surgical incision and procedure similar to bifurcated aortic graft for AAA. However, the graft is often placed on the aorta, rather than the aorta being opened and the graft placed within.
- Time: 2–5h.
- *Pain:* moderate/severe. Thoracic epidural analgesia recommended. Alternatively, IV morphine infusion (in HDU or ICU) followed by PCA until adequate oral intake established. Oral opioids thereafter for 3–7 days
- Position: Supine, arms positioned on arm boards to allow access to arterial or venous catheters
- Blood loss: Variable, typically 500–3000mL; may be sudden and massive. Intraoperative cell salvage should be used. XM 2–6 units.
- Hospital stay: 5–10 days
- *Practical aspects:* anaesthesia similar to open AAA repair. Cardiovascular changes at cross clamping and unclamping less than open AAA repair due to development of collateral circulation. Incidence of diabetes, IHD, and smoking higher.

- Patients with occlusive disease of the lower aorta and iliac arteries constitute a distinct sub-group within vascular surgery patients.
- The main risk factors for aorto-iliac occlusive disease are cigarette smoking and hypercholesterolaemia, which lead to the formation of extensive atherosclerotic plaques.
- These patients are less likely to have diabetes and tend to be younger than those with more peripheral occlusive disease.
- Aorto-iliac occlusive disease is rarely a cause of mortality, but it is an indicator of a significant level of generalized atherosclerosis throughout the body. The usual presentation of occlusive disease is intermittent claudication
- Patients with intermittent claudication have a 2–4-fold increased mortality compared with patients who do not claudicate.
- The 10-yr mortality for patients with claudication is 45%; most patients die from CAD or CVD.

Presentation

- The commonest presentation of aorto-iliac occlusive disease in proximal blood vessels is intermittent claudication. Muscle cramps and pain that occur on exercise and are relieved by resting.
- Limb-threatening ischaemia is rare. Collateral vessels develop over time that can provide a baseline vascular supply to the legs. If plaques are friable or break away, embolic events can occur, e.g. ‘trash foot’.

- The triad of buttock claudication, erectile dysfunction, and absent femoral pulses caused by infra-renal aortic atherosclerosis is termed 'Leriche syndrome'.

Therapeutic options

- Medical therapy is feasible for some patients, comprising smoking cessation, control of diabetes, statin, and antiplatelet drug therapy, weight loss, and exercise. These can be effective and can slow the progression of occlusive disease.
- Minimally invasive therapy with angioplasty and the placement of stents can also be effective if the anatomy allows.
- *Surgical options:*
  - *Aortic, iliac and aorto-iliac endarterectomy*—these have the advantage that no prosthetic materials are used and the risks of infectious complications are reduced.
  - Aorto-bi-iliac and aorto-bifemoral bypass grafts.
  - *Axillo-bifemoral grafts*—these tend to be used for high-risk patients who would tolerate abdominal procedures poorly or for patients whose infected aortic grafts need to be removed.
- Although most aortic procedures are performed through a longitudinal or transverse abdominal incision, some aorto-iliac and many iliac procedures can be performed with retroperitoneal approach, reducing duration of paralytic ileus. May reduce incidence of pulmonary complications. Axillo-bifemoral grafts always extraperitoneal.

Assessment, investigation, and preparation

- Pre-operative assessment, investigation, and preparation are similar to that for patients scheduled to undergo aortic aneurysm or peripheral revascularization surgery (📖 Evaluation of the vascular surgical patient, p. 85).
- There is a high incidence of cardiovascular and respiratory comorbidities that should be identified, quantified, and optimized before surgery. Careful assessment of cardiorespiratory function is required in the patient whose physical activity is limited by claudication as they may have few overt symptoms of cardiac or respiratory disease.
- Blood should be cross-matched for procedures on the aorta and major lower limb vessels, and blood products should be available in line with local protocols.

General or regional anaesthesia?

- Surgery for occlusive disease may be performed under spinal or epidural anaesthesia, and patient should be prepared and consented for these techniques if appropriate.
- There is no good evidence to show the superiority of either RA or GA, despite several large-scale published studies that hoped to prove the superiority of either technique.
- ∴ Choice of anaesthesia is at discretion of anaesthetist, accounting for available facilities, surgical, and patient factors.

Perceived advantages of regional anaesthesia

- Minimizes the stress response to surgery.
- Sympathetic blockade causing vasodilatation and thereby possibly improving graft blood flow and 'run-off'.

- Better post-operative analgesia after abdominal procedures and thereby improved respiratory function.
- The ability of the awake patient to report chest pain and breathlessness should these occur

Perceived advantages of general anaesthesia

- Control of cardiovascular function is arguably controlled more easily.
- There are no problems of patient discomfort during prolonged procedures.
- Post-operative graft failure is caused more by technical surgical issues, rather than peripheral vasoconstriction.
- There are no concerns about intraoperative administration of heparin if a 'bloody tap' occurs with epidural catheter insertion.

Anaesthetic management

- In practice, transabdominal procedures are better performed under a combination of GA and epidural anaesthesia; GA or RA alone may be suitable for lower retroperitoneal procedures
- The prolonged duration length of these procedures means that single-shot spinals are rarely appropriate, so CSEs or CSAs should be used if RA alone is planned.
- Axillo-bifemoral grafts are considered separately (📖 Axillo-bifemoral grafts, p. 376).

In the anaesthetic room

The following should be placed:

- Lumbar spinal catheter or thoracic epidural catheter, according to anaesthetic and analgesic techniques chosen.
- At least one large-gauge peripheral IV line (no smaller than 14G).
- Arterial line for all surgery on the aorta, iliac, and femoral arteries.
- If GA is chosen, balanced GA with tracheal intubation, neuromuscular blockade, and artificial ventilation is appropriate
- NGT if transabdominal approach to the aorta being used. If retroperitoneal approach, discuss with surgeon.
- Temperature probe if GA used.
- Urinary catheter.

In addition, the following may be considered:

- A multi-lumen central venous line in high-risk patients or in those undergoing aortic surgery. It is recommended that anaesthetists should have a low threshold for insertion of a central venous lines in all vascular surgical patients.
- Oesophageal Doppler probe or other non-invasive monitor of cardiac output.

In the operating theatre

Standard monitoring for aorto-iliac occlusive surgery should include:

- Continuous ECG.
- Pulse oximetry.
- Invasive arterial pressure.
- CVP (recommended).
- Temperature if general anaesthesia.

- Urine output.
- Peripheral nerve stimulator if neuromuscular blocking drugs used.

In addition, the following may be used:
- Oesophageal Doppler cardiac output measurement or other non-invasive device for estimating changes in cardiac output.
- Point of care coagulation testing.

The following equipment, drugs, and fluids should also be available:
- Forced-air warmer.
- IV fluid warmer.
- IV fluid warming and infusion device capable of transfusing large volumes of fluid at body temperature.
- Infusion pumps for drugs.
- A selection of vasoconstrictor, vasodilator, inotropic and chronotropic drugs and infusions.
- Antibiotics and heparin.
- All drugs necessary for resuscitation.

Anaesthetic management: key steps
- Discuss each case in advance and in detail with surgeons in order to identify any different or unexpected steps, anatomy, or additional procedures likely to occur in the case.
- *Aortic procedures:* the general approach taken to clamping and unclamping the aorta, described in 📖 Open aortic aneurysm repair, p. 350, should be followed. However, the existence of collateral vessels means that the cardiovascular and metabolic impact of clamping and de-clamping are much less.
- Anaesthetists should advise the surgeon if there are no cardiovascular changes after de-clamping. Although this may result from extensive collateral vessel formation, it may also be caused by distal thrombotic occlusion. Adequacy of circulation to the legs should be assessed a short time after de-clamping.
- Aorto-bi-iliac and aorto-bifemoral grafts can be unclamped one limb at a time to minimize cardiovascular and metabolic disturbances. If the surgical procedure involves only one limb, there will be little cardiovascular impact from clamping or de-clamping.
- It is sometimes suggested that haemorrhage associated with surgery for occlusive disease will be less than during surgery for aneurysmal disease. **This is incorrect**—any procedure on the aorta or major arteries can be associated with rapid and life-threatening bleeding, and it should be assumed that blood will be lost in large volumes at some stage—blood warmers should be used routinely and rapid infusers should be available. The use of a cell saver should be considered for complex or re-do cases.
- Heparin ~5000IU is usually given IV before arterial clamping.

Axillo-bifemoral grafts
- Usually reserved for high-risk patients, deemed unfit to undergo abdominal procedures, or in patients who have infected aortic grafts removed. These patients present particular challenges to anaesthetist.
- The operative mortality of axillo-bifemoral grafts is up to 10%.

- Axillo-bifemoral graft surgery has several steps, the exact order of which may vary:
  - Dissection of axillary artery on one side, usually below clavicle.
  - Creation of a sc tunnel from axillary area to groin.
  - Dissection of femoral arteries.
  - Creation of sc tunnel between two femoral arteries.
  - Passing a long bifurcated graft through sc tunnels.
  - Anastomosis of proximal end of graft to axillary artery.
  - Anastomosis of distal ends of grafts to femoral arteries.
- Axillary artery and proximal graft anastomosis can be performed under LA and sedation, and the femoral elements of the procedure can be performed under local or spinal/epidural anaesthesia
- However, balanced GA is preferred because of profound surgical stimulation associated with formation of sc tunnel from clavicle to groin.
- The use of a combination of LA and spinal anesthesia, with a short period of GA to cover the sc tunnelling has been reported.

Post-operative care

- Some patients will require early post-operative care in HDU or ICU, depending on their pre-existing condition, their physiological status during and after surgery, and the need for post-operative lung ventilation.
- Patients undergoing aortic occlusive surgery should be managed in a similar way to those who have undergone aortic aneurysm surgery.
- High-risk patients who have undergone axillo-bifemoral grafts should receive particularly close attention.

Further reading

McIntyre KE, Rowe VL.. Aortoiliac occlusive disease. Medscape 2011. Available at: http://emedicine.medscape.com/article/461285-overview#a0104 (accessed 27 November 2011).

Carotid artery surgery and stenting

Surgery to remove carotid atheroma was first described in 1954. CEA is a preventative procedure performed to prevent disabling or fatal embolic strokes in patients with significant carotid stenosis. Patients have usually suffered a TIA or minor stroke, but the carotid stenosis may be asymptomatic. Carotid angioplasty was first described in 1980; the first multicentre prospective data on carotid angioplasty with stenting (CAS) were published in 1993 and CAS has been performed increasingly over the last two decades. The rationale and therapeutic aims of CEA and CAS are similar: namely, to remove or dilate areas of atherosclerosis in the internal carotid arteries, which usually occur close to the carotid bifurcation. There is continued debate over the relative indications for and benefits of the two procedures. CEA can be performed under general or local/regional anaesthesia; CAS is performed under local anaesthesia.

Carotid endarterectomy

Surgical technique

- CEA is performed by clamping the internal, common, and external carotid arteries, opening internal carotid artery and directly removing atheromatous plaque.

- Adequate CBF (during carotid clamping) relies on collateral flow around the circle of Willis.
- The longitudinal arteriotomy can be closed directly (1° closure), but many surgeons prefer to insert a diamond-shaped patch (vein, prosthetic). Routine patching is associated with reduced rates of peri-operative stroke and late restenosis when compared with 1° closure.
- The carotid clamps are then released sequentially and carotid blood flow is restored.
- Many surgeons insert a prosthetic vascular shunt routinely in order to ensure adequate cerebral blood flow during clamping. Since shunt usage may be associated with complications, such as vessel damage or emboli, other surgeons shunt selectively when cerebral perfusion is deemed inadequate during cross clamping (e.g. loss of consciousness in the awake patient, low carotid stump pressures).
- The shunts used most commonly are the Javid and Pruit-Inahara shunts. The Pruit-Inahara shunt is perhaps more complex to insert and remove, but has cuffs at either end to ensure a good seal. It allows flow in the internal and external carotid arteries, completely bypasses the arteriotomy site and allows stump pressure to be measured. The Javid is inserted through the operative site and the surgeon needs to work around it.
- Eversion CEA is an alternative surgical technique, where the internal carotid artery is transected at its origin, the adventitial layers everted and atheroma removed before re-implantation onto the common carotid artery. Eversion CEA may be preferred when the carotid is tortuous or elongated. It is usually quicker to perform, restenosis rates may be lower and patching unnecessary. A shunt cannot be inserted until the plaque has been removed. A period of trial cross-clamping is recommended before the internal carotid artery is transected.
- An assessment (Duplex US, angioscopy, angiography) is often made towards the end of surgery to identify and correct any technical problems (luminal thrombus, intimal flaps), which will increase the risk of peri-operative stroke.
- A wound drain is usually inserted before skin closure to reduce risk of neck haematoma formation which, if severe, may the compromise airway.

Indications for CEA in symptomatic patients

CEA reduces incidence of major disabling stroke after minor stroke or TIA, in patients with stable neurological symptoms. The ~5-yr risks of stroke with medical therapy alone are:

- *>70% carotid stenosis:* 33%.
- *50–69% stenosis:* 28%.
- *<50% stenosis:* 25%.
- These risks are likely to improve with continuing advances in medical therapy (dual antiplatelet therapy, high dose statin therapy, aggressive multi-agent antihypertensive therapy).
- The benefits of CEA are greatest in patients with more severe carotid stenosis, and rely on low perioperative morbidity (30-day stroke or death rate <6%):
  - *>70% Carotid stenosis*—absolute risk reduction (ARR) of 16% for stroke or death within 5yrs. 5-yr relative risk reduction (RRR) 48%.

Numbers needed to treat (NNT) to prevent one major stroke or death within 5yrs = 6.
  - *50–69% stenosis*—ARR 8%, RRR 28%. NNT = 13.
  - *30–49% stenosis*—ARR 2.6%, RRR 10%, NNT = 38.
  - *<30% stenosis*—risks of CEA outweigh the benefits.
- In general, if CEA is indicated, this should be performed before other non-carotid surgery. A possible exception is where both CEA and coronary revascularization (bypass graft or percutaneous coronary intervention) are both indicated urgently (see 🕮 Combined carotid endarterectomy and coronary procedures, p. 409). Anaesthesia for CEA is detailed in 🕮 Endovascular abdominal and thoracic aortic aneurysm repair, p. 363.
- In some patients who have had a stroke, CEA is not indicated, i.e. the stroke was not related to carotid atherosclerosis, or the degree of carotid stenosis at the time of assessment was <50%.

Urgency for intervention

The risks of major stroke are highest in the first few hours or days after TIA or minor stroke. If indicated CEA should be performed as soon as possible. Current guidelines suggest:

- CEA is effective in reducing the risk of future stroke when performed within 2wks of minor stroke (NNT = 5) or TIA.
- The perioperative risks are similar when CEA performed within 1wk of minor stroke or TIA compared with delaying surgery.
- However, despite recent guidelines the benefits of performing urgent surgery (within 48h) are unclear.

Urgent or emergency surgery decreases the time available for optimizing co-existing medical conditions (see 🕮 Evaluation of the vascular surgical patient, p. 85).

Indications for CEA in asymptomatic patients

- The 5-yr risk of stroke in patients with carotid stenosis is lower in asymptomatic patients (6%).
- The risks and benefits from CEA in asymptomatic patients also depend on the degree of carotid stenosis.
- *Carotid stenosis>60%:* ARR 5%, RRR 46%. NNT = 19.
- *Carotid stenosis<60%:* risks of CEA outweigh the benefits.
- The benefits are greater in men.
- The risks of stroke with medical therapy alone are likely to decrease, and there are calls for new trials in asymptomatic patients comparing CEA, CAS, and medical therapy alone.

Carotid artery stenting

Technique

- CAS involves the endovascular placement of a stent across the stenosis within the carotid artery.
- A catheter is passed usually via femoral artery around aortic arch and into carotid artery under fluoroscopic guidance
- The stent is inflated using an intraluminal balloon, which is then withdrawn.

- Early stents were associated with a high risk of distal embolization during stent placement or balloon inflation, and emboli protection devices are now used. These include a mesh or filter positioned distal to the stent during stent placement or a technique whereby carotid blood flow is interrupted or reversed.
- Newer stents may be mesh or membrane covered to reduce late embolization.
- CAS is usually performed under LA in radiology or endovascular suite.

Indications for CAS

Current data suggest:

- CAS may be performed in 'average risk' symptomatic patients, provided the risks are low (30-day stroke or death rate <6%).
- Patients should undergo intervention as soon as possible after onset of symptoms.
- CAS should not be performed in 'average risk' asymptomatic patients outwith clinical trials.
- CAS may be indicated for correction of re-stenosis after CEA.
- CAS may be preferred depending on co-existing medical or surgical conditions. Patients at high medical risk for CEA including:
  - Class III/IV congestive heart failure, class III/IV angina, left main CAD, >2-vessel CAD, left ventricular ejection fraction (LVEF) <30%, recent MI.
  - Severe lung or severe renal disease.
- Adverse surgical or anatomical factors including:
  - Previous neck surgery (e.g. radical neck dissection or in the presence of a tracheostomy) or previous neck irradiation.
  - Restenosis after previous CEA.
  - Surgically inaccessible lesions (i.e. above C2).
  - Contralateral carotid occlusion, contralateral vocal cord palsy.

CEA or CAS in an individual patient?

- The decision to recommend CEA or CAS should be made by MDT including a stroke physician, vascular surgeon, and interventional radiologist.
- The decision to recommend CEA or CAS depends on several factors, including individual expertise, facilities, patient comorbidity, and patient preference.

Most randomized trials have consistently shown that:

- The incidence of periprocedural stroke is twice as high after CAS compared to CEA.
- There is a trend toward higher rates of death or death, and disabling stroke after CAS.
- The risk of cranial nerve injury is lower after CAS.
- The risk of minor MI is lower after CAS.
- Restenosis rates are higher following CAS, but this is not associated with an increase in late risk of stroke.

Antiplatelet therapy and CEA or CAS

- Combination therapy with aspirin (75–300mg) and clopidogrel 75mg should be given before, and for at least 30 days after CAS.
- Aspirin (75–300mg) should be given before CEA and continued indefinitely.
- From 30 days after CEA, clopidogrel 75mg daily or the combination of aspirin 75mg and modified release dipyridamole 200mg bd can be given for cardiovascular prophylaxis.

Further reading

Brott TG, Halperin JL, Abbara S *et al.* ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: executive summary: *Stroke* 2011; **42**: e464–540.

Furie KL, Kasner SE, Adams RJ, *et al*. Guidelines for the prevention of stroke in patients with stroke or transient ischaemic attack. *Stroke* 2011; **42**: 227–76.

Rerkasem K, Rothwell PM. Systematic review of the operative risks of carotid endarterectomy for recently symptomatic stenosis in relation to the timing of surgery. *Stroke* 2009; **40**: e564–72.

Rothwell PM Algra A, Amarenco P. Medical intervention in acute and long-term secondary prevention after transient ischaemic attack and ischaemic stroke. *Lancet* 2011; **377**: 1681–92.

Anaesthesia for carotid surgery

- *Procedure:* internal, external, and common carotid arteries clamped and atheromatous plaque removed, followed by direct arterial closure or insertion of a prosthetic or autogenous vein patch. Prosthetic shunt often inserted to maintain brain blood flow during carotid clamping.
- *Time:* 1–2h.
- *Pain:* minimal. Local anaesthetic infiltration or cervical plexus blocks +/– simple oral analgesics usually sufficient during and after surgery. Low dose morphine sometimes required.
- *Position:* supine, 10–20° head up on head ring, support behind shoulders.
- *Blood loss:* minimal; <500mL. Group and save only.
- *Hospital stay:* 1–3 days unless complications.
- *Practical aspects:* CEA can be performed under regional or general anaesthesia, or even local infiltration alone. Technique should permit rapid post-operative recovery. Some form of cerebral monitoring should be used. Arterial pressure can be very labile during and after surgery; start direct arterial pressure monitoring before induction of anaesthesia and continue for several hours post-operatively. Anticipate and attenuate arterial pressure instability using vasoactive drugs. Minimize IV fluid administration. Avoid bladder catheterization.

CAS includes CEA and carotid body tumour removal. CEA is a prophylactic operation designed to prevent 1° or 2° embolic stroke, and in UK is performed mainly by vascular surgeons. Current NICE guidelines are to offer surgery to all patients with a carotid stenosis >70% within 2wks of an acute embolic stroke or TIA. The principles of anaesthesia for carotid surgery combine those of cardiovascular and neurosurgical anaesthesia.

Anaesthesia for carotid endarterectomy

- The major risks of CEA are stroke (3–4%), and MI (0.5%) and death.
- Since the risks of stroke can be reduced by lifestyle changes and medical therapy and CEA is a preventative procedure, the perioperative risks of CEA must be low for surgery to be beneficial.
- CEA may be performed as an elective procedure for asymptomatic stenosis, or within 2wks of an acute stroke or TIA.

The principle aims of anaesthesia are to:

- Maximize cerebral perfusion and oxygen delivery.
- Minimize myocardial stress.
- Maintain cardiovascular stability before, during, and after surgery.
- Prevent post-operative complications (including cerebral hyperperfusion, hypertension, haematoma).
- These aims can be achieved using GA, LA, or RA.

Preoperative assessment

- Several factors have been shown to predict poor outcome (death, stroke, or MI) after CEA (Box 9.1)
- As CEA is a prophylactic procedure intended to reduce the risk of a future disabling major stroke, the risks of surgery must be low to achieve an overall benefit. The incidence of CAD is lower in patients undergoing CEA compared with aortic or peripheral vascular surgery, but angiographically significant coronary stenosis is still present in 26% of patients.
- Perioperative risk should be assessed carefully and appropriate investigations performed (📖 Evaluation of the vascular surgical patient, p. 85).

Box 9.1 Risk factors associated with CEA

- Ipsilateral ischaemic lesion on CT.
- Symptom status: ipsilateral > asymptomatic.
- Urgent surgery when neurological deficit.
- Contralateral occlusion.
- Intraluminal thrombus.
- Ulcerated or irregular plaque.
- Age >75yrs.
- Stenosis near the carotid syphon.
- Surgery prior to CABG.
- Angina.
- Hypertension with diastolic >115mmHg and/ or systolic>180mmHg.
- Chronic renal failure.

- Co-existing medical conditions should be optimized before elective CEA.
- Current recommendations to perform CEA within 2 weeks of a neurological event (with the aim of reducing this to 2 days by 2017), may limit the time for detailed risk stratification. However, potentially correctable risk factors (IHD, hypertension, renal dysfunction) should be addressed wherever possible.

Preoperative preparation

Medical complications occur in about 10% of patients undergoing CEA.

- Arterial pressure control is important to reduce the risk of perioperative stroke, but the threshold at which risks increase substantially is not well defined. (see 🕮 Hypertension).
- Poorly-controlled hypertension also increases the risks of perioperative myocardial ischaemia and arrhythmias.
- Current consensus is that arterial pressure should be <160 systolic and <110 diastolic before non-urgent CEA. Elective surgery should be delayed to treat values above these thresholds.
- Since baroreceptor function and cerebral autoregulation are impaired after an acute neurological event aggressive lowering of BP may precipitate a further neurological event. Antihypertensive therapy should be carefully titrated. If surgery is urgent (after TIA or minor stroke), acute aggressive arterial pressure control is not advisable and a stroke physician should be consulted (see 🕮 Recent stroke or TIA, p. 189).
- Established antihypertensive medication should be continued through the perioperative period. Diuretics may be omitted on the day if patient is having surgery under local anaesthesia to avoid potential for a full bladder during surgery. Some anaesthetists omit ACE inhibitors or ARBs on morning of surgery if CEA is planned under GA.
- *Current national recommendations for antiplatelet drug management of recent TIA or stroke:* aspirin and slow release dipyridamole, or clopidogrel as a single agent if the patient is aspirin intolerant. These drugs should be continued through the perioperative period. Some centres administer clopidogrel, in addition to aspirin, particularly for patients with drug-eluting coronary stents or crescendo TIAs.
- Hyperglycaemia is known to worsen ischaemic damage to the brain. A titrated insulin regimen may be required to prevent hyper- or hypoglycaemia in diabetic patients.
- There is a high incidence of COPD in patients undergoing CEA. The patient's ability to tolerate the semi-supine position for the anticipated duration of surgery must be assessed if surgery under local anaesthesia is proposed.
- A CT/single photon emission computed tomographic (SPECT) brain scan can help identify high risk patients with ipsilateral ischaemic infarcts and evolving infarcts with watershed ischaemia.
- If the patient has significant co morbidities, e.g. severe cardiac, renal, or respiratory disease, post-operative care in a HDU environment is recommended.
- The management of patients undergoing a combined CEA/CABG is covered in 🕮 Combined CEA and coronary procedures.

Surgical procedure

The details of the surgical procedure are described in 🕮 Carotid artery surgery and stenting.

Intraoperative considerations

- The patient is positioned with the neck and head extended slightly and turned away from the operative side. A gel support or 1L fluid bag may be placed between the scapulae to further extend the neck and aid surgical access. The head is supported on a gel ring.
- The duration of surgery is usually 60–90min.
- Patients with orthopnoea, severe respiratory disease, arthritis, or degenerative spine disease may find this position difficult to maintain for the duration of surgery, if surgery is to be performed under LA. A soft pillow placed behind the knees aids comfort.
- Minimal amounts of IV fluids should be administered if the CEA is performed under local anaesthesia to prevent discomfort from a full bladder. A urinary catheter may be inserted before surgery to alleviate this, although catheter can also cause discomfort.
- Blood loss is usually minimal, but a group and save should be performed. Rarely, surgeon may lose control of carotid artery.
- Patient temperature should be maintained, but active warming measures are not usually required.

Goals of anaesthesia

The combined incidence of death, stroke, or MI should be <5% in centres performing carotid surgery. Two-thirds of intraoperative strokes result from inadvertent surgical technical error, but the anaesthetic technique must aim to maintain cerebral and myocardial oxygen supply, and minimize demand.

The goals of anaesthesia are to:

- *Maintain oxygen delivery to the brain:* CEA includes a period of potential focal or global cerebral ischaemia during carotid cross-clamping. CBF should be maintained by ensuring an adequate arterial pressure.
- *Maintain airway control:* access to airway during surgery may be limited and sedative techniques, which potentially compromise the airway, should be avoided.
- *Avoid excessive fluctuations in arterial pressure:* cerebral autoregulation is impaired after a stroke and changes in arterial pressure can cause cerebral ischaemia, haemorrhage, or vasogenic oedema. Reduction of cerebral metabolic rate (using GA) may offer some protection against relative cerebral hypoperfusion.
- *Allow rapid and smooth emergence from anaesthesia:* this allows early post-operative neurological assessment, which may prevent secondary damage to the brain should there be post-operative neurological deterioration.
- *Minimize myocardial stress:* 25% of patients undergoing CEA have significant CAD. It is vital to maintain the balance between myocardial O_2 supply and demand by control of heart rate, preload, and afterload.
- *Maintain cardiovascular stability before, during and after surgery:* arterial pressure should be maintained within 20% of baseline values and any

fluctuations controlled quickly. Heart rate should ideally be maintained within a range of 50–70 beats/min in patients vulnerable to coronary ischaemia.

- *Prevent hypo-or hyperglycaemia:* hyperglycaemia can exacerbate neuronal damage during a period of ischaemia. The intraoperative target glucose concentration should be 4.4–7mmol/L, but in poorly controlled diabetes the range should be widened to 5–10mmol/L.

Selection of anaesthetic technique

CEA may be performed under general or local anaesthesia.

- GA offers the theoretical advantage of reducing cerebral metabolic requirements for oxygen and providing cerebral protection. Performing the operation awake under LA provides the 'gold standard' monitor of adequacy of cerebral perfusion, and so permits more selective use of shunts. In the GALA study, the combined endpoint of stroke, myocardial infarction or death occurred in 4.5% of patients receiving local anaesthesia and 4.8% under GA.
- No particular anaesthetic regimen has been shown to be best. Since both the GALA study and systematic reviews of studies found no difference in perioperative complications between local or general anaesthesia, the choice of anaesthetic should be determined by the preferences of the patient, anaesthetist, and surgeon. A possible exception to this is those patients with a contralateral carotid stenosis ≥90%; in these cases a local anaesthetic technique may reduce the risk of cerebral hypoperfusion.
- GA allows the possibility of reducing cerebral metabolic rate and providing cerebral protection. However, performing the procedure in the awake patient is the gold standard for the assessment of neurological well-being.
- The haemodynamic profile during surgery differs between LA and GA. There is a tendency to intraoperative hypertension and post-operative hypotension with LA. With GA, intraoperative hypotension and postoperative hypertension are more common. Interventions to maintain arterial pressures are required using both techniques.

Local anaesthesia

- Anaesthesia of the neck can be provided by superficial, intermediate or deep cervical plexus block, local infiltration by the surgeon or a cervical epidural. These techniques are detailed in 📖 Specific regional blocks, p. 284.
- Superficial cervical plexus block is performed by infiltration of local anaesthetic along the entire length of the posterior border of the sternomastoid muscle. A volume of 10–15mL LA(e.g. levobupivacaine 0.25%) is usually required.
- Intermediate cervical plexus block requires a needle to be inserted at right angles to the skin at the midpoint of the posterior border of sternomastoid until a loss of resistance or 'pop' is felt as the needle penetrates the investing layer of cervical fascia at a depth of 1–2cm.
- Deep cervical plexus block is described in 📖 Specific regional blocks, p. 284.

- Cervical epidural blocks at C6–C7 or C7–T1 have been described for CEA. They are rarely used because the carry significant risks (100% incidence of altered respiratory function, 1% incidence of respiratory failure requiring ventilation and an 11% incidence of hypotension. Epidural haematoma is a theoretical risk as the patient is taking anti-platelet drugs and receives an IV dose of heparin intraoperatively.
- A sc wheal of local anaesthetic should be placed along the angle of mandible as there may be some additional innervation from the mandibular branch of the trigeminal nerve. Intra-oral inferior alveolar nerve block has been described

Practical considerations

- The patient's head is supported on a gel ring and oxygen administered at 4L/min via a face mask or nasal cannulae
- Patient comfort can be increased by breaking the table into a 'deckchair' position. A pillow can be placed behind the knees.
- Encourage the patient to void their bladder before surgery and avoid excessive intraoperative IV fluids.
- The contralateral arm should be placed on an arm board to enable motor function (grip strength) to be assessed either subjectively or quantitatively via a 500mL bag of fluid attached to a pressure transducer. Verbal contact must be maintained. Dysphasia or aphasia implies cerebral ischaemia.
- There are numerous different ways of draping the patient. Whichever method is chosen it is essential that patient does not feel claustrophobic and that anaesthetist is confident they are able to easily access the airway should conversion to GA be required. A transparent plastic drape is useful, which may be either draped over an L bar or suspended from two drip poles.
- Should a patient complain of feeling claustrophobic this may be helped by placing cold wet swabs on forehead and blowing unheated air from a forced air warmer across the patient.
- Supplementary LA is often required. It is unusual to anaesthetize the carotid sheath completely with percutaneous injections, possibly because additional pain fibres travel with the sympathetic innervation of the carotid artery. There also sensory contributions from the trigeminal nerve over the angle of the jaw, facial nerve to platysma, and cranial nerves to the carotid sheath The carotid sheath is usually anaesthetized under direct vision by the surgeon. Care must be taken not to inadvertently inject LA directly into the carotid artery. If the carotid bifurcation is high in the neck there may be discomfort from parotid fascia, which is innervated by the mandibular branch of the trigeminal nerve. Additional LA infiltration of this area may be required.
- Supplementary analgesia may be provided using IV paracetamol 1g, non-steroidal analgesics (if not contraindicated by renal dysfunction).
- Remifentanil at a rate of 0.05–0.1μg/kg/min or a target plasma concentration <2ng/mL may be used to facilitate placement of the block.

Advantages of regional anaesthesia

- It allows definitive (real-time, awake) monitoring of cerebral function.
- Systemic and cerebral autoregulation is maintained.
- It avoids the cardiovascular effects of general anaesthesia and mechanical ventilation.
- Hypertension is much more common when CEA is performed under LA, particularly when carotid is cross-clamped. Intraoperative hypotension is less common and vasopressor requirements are less compared with GA.
- The use of awake cerebral monitoring allows selective use of shunts, although this depends on individual surgical technique.
- Allows assessment of effects of hypoglycaemia on cerebral function.
- Avoids 'minor' complications of GA.

Disadvantages of regional anaesthesia

- Earache and toothache (caused by submandibular retractors) common.
- Great care must be taken not to sedate the patient excessively.
- Perioperative hypertension can be severe and difficult to control. The choice of drug to treat intraoperative hypertension depends partly the preoperative drug regimen (see 📖 Evaluation of the vascular surgical patient, p. 85).
- Surgery can be technically difficult, particularly if carotid bifurcation is high and plaque extends towards carotid siphon. The morbidly obese patient with a thick neck needs particular care. The surgeon must remain calm and surgical technique must be gentle.
- The advantages of operating under LA may be negated if there is a language barrier or if patient is severely dysphasic or aphasic due to a preoperative neurological event
- Deep cervical plexus block is associated with unilateral impairment of diaphragmatic function in 60% of cases. Deep cervical plexus block should not be performed in patients with severe COPD or those known to have contralateral phrenic nerve palsy.

General anaesthesia

CEA is performed under balanced general anaesthesia. A variety of agents may be used, but ventilation should be controlled.

Advantages of general anaesthesia

The major advantages of GA are:

- Patient comfort.
- Easier patient positioning.
- Control of the airway.
- Artificial ventilation can be used to maintain oxygenation and manipulate arterial pCO_2.
- Cerebral metabolic rate of oxygen is reduced and cerebral autoregulation maintained using low concentrations of volatile agents.
- Better tolerance of some monitoring modalities (such as transcranial Doppler).

Practical considerations

- All commonly used agents for induction and maintenance of anaesthesia are acceptable, with the exception of ketamine. All will reduce cerebral metabolism and provide neuroprotection to ischaemic cells.
- IV induction is usual, with judicious doses of propofol or thiopental titrated to effect.
- **Beware**: time to onset of effect of IV anaesthetics increased in elderly.
- Definitive airway control using a tracheal tube is preferable, but a second generation supraglottic airway device may be used. IPPV is necessary to maintain normal values of $PaCO_2$.
- Hypotension is common after induction, caused by the cardiovascular effects of anaesthesia and artificial ventilation. This should be anticipated: drugs to treat hypotension should be prepared before induction of anaesthesia and be immediately available (Table 9.1).
- Rapid swings in arterial pressure are common. Arterial pressure management during CEA is detailed in 🕮 Blood pressure management for CEA, p. 391. In general, arterial pressure should be maintained to ~10–20% above preoperative values, although there is little evidence to support this figure.
- Reflex bradycardia may occur because of vagal traction during carotid sheath manipulation. Atropine should be available.
- Anaesthesia is maintained with a volatile agent in an oxygen/air mixture. Nitrous oxide should be avoided. It increases cerebral metabolic rate and produces a concomitant increase in middle cerebral artery blood flow velocity in the presence of both volatile agents and propofol.
- Volatile anaesthetic agents with low blood/gas solubility allow rapid emergence from anaesthesia. They do not affect CBF at doses <1 MAC. Desflurane may give better haemodynamic stability and faster emergence than the other agents.
- TIVA using propofol is an alternative to volatile agents. It may increase requirements for vasopressor drugs.
- An infusion of remifentanil (0.1–0.2microgram/kg/min, Cp <5ng/mL) is a very useful adjunct during surgery. It allows lower concentrations of volatile agents to be used and permits rapid emergence from anaesthesia. Haemodynamic control is good though bradycardia and hypotension may occur with excessive dosing.
- Bispectral index (BIS) monitoring may be used to help titrate the depth of anaesthesia and minimize hypotension
- Analgesic requirements for CEA are modest. Moderate doses of opioids, e.g. fentanyl 1.5–3μg/kg are required supplemented by paracetamol and a NSAID, (if not contraindicated), especially if combined with a superficial cervical plexus block or local anaesthetic wound infiltration (using 10–20ml levobupivacaine 0.25–0.5%)

Cerebral monitoring during carotid surgery

Patients presenting for CEA usually have widespread vascular disease and cerebrovascular autoregulation is impaired. Despite the normally copious cerebral blood flow via four main arteries and a generous collateral circulation, it is difficult to predict whether regional blood flow is at a critically low level when a carotid artery Is cross clamped. Cerebral monitoring may be used during CEA, to determine whether a shunt needs to be inserted after carotid cross-clamping, and may help guide decisions to increase arterial pressure and CBF using drugs.

The 'gold standard' monitor is the awake patient; it can reveal clinically significant ischaemia when the EEG remains unchanged. All other monitors (Box 9.2) have imperfect sensitivity and specificity and their use varies between centres. Cerebral monitoring is detailed in 📖 Monitoring of the brain, p. 327.

Post-operative considerations

- Most complications after CEA occur within the first 4h after surgery. Close neurological and cardiovascular monitoring is vital in the immediate post-operative period.
- Arterial pressure is often labile. Hypertension is commonly seen, which may predispose to serious complications including wound haematoma, myocardial ischaemia, haemorrhagic stroke, and cerebral hyperperfusion syndrome. Invasive arterial pressure monitoring is indicated and hypertension controlled aggressively in the immediate postoperative period (see 📖 Blood pressure management, p. 391).
- Practices differ but a minimum of 1–3h in recovery/post-anaesthesia care unit (PACU) is suggested. If patients have remained stable they can then be transferred to the vascular ward.
- Requirement for infusions to correct blood pressure to target levels is an indication for post-operative monitored in a Level 2 facility.

Neurological dysfunction

- Neurological function is typically assessed using a GCS.
- Pain may occur after CEA and responds to small increments of morphine IV (2–5mg).

Box 9.2 Summary of cerebral monitoring modalities used during CEA

- EEG has good correlation with neurological changes, but produces false +ves.
- Stump pressure has poor sensitivity and specificity, but is popular.
- Transcranial Doppler is invaluable to assess embolic phenomena, haemodynamic ischaemia, shunt function, and cerebral hyperperfusion syndrome. It can also be monitored in the post-operative period.
- Cerebral microdialysis has good predictive values for cerebral ischaemia, but is invasive.

- Early signs of neurological dysfunction may be non-specific (confusion, agitation, somnolence). Gross hemispheric neurological defects are more apparent. If in doubt, senior medical input should be sought urgently as these symptoms may indicate carotid occlusion, athero-embolism, or cerebral hypo- or hyperperfusion. If a gross neurological deficit occurs, an urgent bedside carotid Doppler examination should be performed to look for carotid occlusion. If bedside scanning is unavailable or the scan reveals an occluded carotid artery urgent surgical re-exploration is mandatory.
- Transient neurological dysfunction of the hypoglossal, superior laryngeal, recurrent laryngeal, glossopharyngeal nerve, and rarely the vagus nerve can occur.
- Carotid body dysfunction commonly affects the ventilatory response to mild hypoxaemia. Where bilateral carotid disease is present it can result in hypercarbia in addition to hypoxaemia. This effect can be compounded by hemi-diaphragmatic paralysis in patients who have had undergone deep cervical plexus blockade.

Airway problems

- Upper airway obstruction is a rare, but potentially fatal complication in early post-operative period. Most common cause is neck haematoma, compounded by tissue oedema 2° to venous and lymphatic obstruction.
- Treatment of neck haematoma is urgent surgical evacuation. This is best achieved in theatre using local anaesthetic infiltration if required. Senior help should be obtained immediately. Induction of GA can lead to catastrophic loss of the airway.

Carotid body tumours

The carotid body tumour (chemodectoma) is a type of paraganglionoma, which arises from chemoreceptor cells. Paraganglionomas arise from chromaffin cells in ganglia and also occur at the aortic body, vagus, jugular vein, and tympanic membrane. They are rare, highly vascular, slow- growing tumours, which usually present as an asymptomatic mass in the anterior triangle of the neck. Dysphonia or dysphagia and shoulder weakness have been reported due to their close anatomical relationship to cranial nerves X–XII). Around 10% of cases present with symptoms indistinguishable from phaeochromocytoma, characterized by hypertension, flushing and palpitations.

- A carotid body tumour occurs at the carotid bifurcation outside the carotid artery, but may encircle it.
- Most are benign, but 3% metastasize. Benign and malignant tumours can cause local damage including damage to cranial nerves.
- Various imaging modalities can be used to confirm the diagnosis (colour Doppler, CT, MRI, MRA). MRA is useful to delineate the vascularity of the tumour and the anatomy of the feeder vessels.
- Indirect laryngoscopy may be needed before surgery to assess vocal cord function as the recurrent laryngeal nerve may be affected.

- Surgical resection of the carotid body is the only curative treatment. It may be technically difficult because of the close relationship of the tumour to major vessels and cranial nerves. Torrential haemorrhage, cranial nerve damage and a 5% incidence of stroke have been reported.

Anaesthesia

- Anaesthetic considerations are similar to those for CEA, although patients are often younger and incidence of cardiovascular disease is lower.
- Although successful resection has been reported under cervical plexus block, GA is most frequently used. The incision is usually more extensive than for CEA. Occasionally, the surgeon may need to access the skull base to gain access to carotid canal
- Nerve stimulation may be used to test the location and integrity of cranial nerves—a short-acting neuromuscular blocking agent should be used.
- A nasotracheal tube may aid surgical access if the tumour is high.
- Continuous arterial pressure monitoring is required.
- Large bore IV access is mandatory as blood loss can be brisk.
- A shunt may be used.
- Transcranial Doppler is useful to monitor for emboli and to assess shunt flow during cross-clamping.

Further reading

Howell SJ. Carotid endarterectomy. *Br J Anaesth* 2007; **99**: 119–31.

Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. *Br J Anaesth* 2009; **102**: 442–52.

Blood pressure management for CEA

BP instability is very common in patients undergoing CEA, during and after surgery. Several factors are involved including impaired arterial baroreflex function and other aspects specific to carotid surgery.

Factors affecting arterial blood pressure

Altered baroreflex function

Arterial baroreflexes control autonomic responses to acute changes in arterial pressure. Impairment of baroreflexes leads to increased variability in arterial pressure during and after CEA. Baroreflex sensitivity is inevitably reduced in patients undergoing CEA owing to:

- Recent or chronic hypertension.
- Antihypertensive drugs.
- Old age.
- Diabetes mellitus
- Carotid atheroma or stenosis, especially bilateral stenosis

- CVD, especially in the first few weeks after TIA or minor stroke when hypertension is common.
- Previous contralateral CEA or radical neck surgery.
- *Surgical factors:* surgical removal of carotid plaque causes immediate partial disruption of baroreflex activity, lasting for hours or days and predisposing to BP instability. This is worse in patients with bilateral carotid disease or who have had previous contralateral carotid surgery. Deliberate or inadvertent surgical section of the carotid sinus nerve or LA blockade directly interferes with baroreceptor function
- *Anaesthesia:* IV and inhalational anaesthetic drugs reduce baroreflex activity in a dose-dependent manner.

Impaired baroreflex function contributes to cardiovascular lability during surgery, and is probably the main factor involved in post-operative hypertension after CEA.

Intraoperative factors

- Carotid cross-clamping during CEA reduces CBF with a compensatory increase in sympathetic nervous activity and arterial pressure. This is reversed on restoration of flow and transient hypotension may occur.
- The magnitude of these changes depends on collateral CBF, the degree of stenosis and the duration of cross-clamping. They may be less apparent under deep GA.

Anaesthetic factors

- Independent of their effects on baroreflex function, IV and volatile anaesthetic drugs can cause hypotension by several mechanisms, including reduction of central sympathetic tone, direct effects on the heart and vascular smooth muscle.
- Positive pressure ventilation may impair venous return.
- During CEA under GA, hypotension often occurs during carotid cross-clamping, followed by hypertension after flow restoration and post-operatively.
- During CEA under RA, a pattern of relative hypertension during and hypotension after cross-clamping is more common.

Post-operative factors

Wound pain or bladder distension can cause or exacerbate post-operative hypertension

Effect of hypertension on outcome

Some studies have shown that pre-existing hypertension (systolic pressure >180mmHg) increases the risk of post-operative stroke or death, but the data is conflicting. However:

- Preoperative hypertension increases the risk of perioperative hypo- and hypertension.
- Perioperative haemodynamic instability predisposes to cardiac arrhythmias and myocardial ischaemia.
- Cardiovascular complications are responsible for up to half the long-term mortality after CEA

- Post-operative hypertension increases the risk of wound haematoma, cerebral hyperperfusion syndrome (CHS) (📖 Cerebral hyperperfusion syndrome, p. 402) and haemorrhagic stroke.

Before surgery, a high arterial pressure may be required to maintain cerebral perfusion distal to a carotid stenosis, depending on the adequacy of collateral circulation around the Circle of Willis. However, restoration of flow at surgery will acutely increase perfusion pressure to these distal cerebral vessels. Thus hypertension should be detected and controlled before elective CEA.

Preoperative assessment and treatment goals

- Arterial pressure should be measured in both arms and the higher reading noted.
- When there is a significant disparity, direct arterial pressure monitoring should be from the arm with the higher reading.
- In hypertensive patients, look for end-organ damage (renal function, ECG, echocardiography)
- Continue usual antihypertensive medication to include the day of surgery. Some anaesthetists choose to omit ACE inhibitors and ARBs for 24–48h before surgery because of the risk of refractory hypotension after induction of GA.
- Current guidance suggests CEA should be performed within 2 weeks (and ideally within 48h) after minor stroke or TIA, because the risk of recurrent major stroke is highest during this time. In symptomatic patients, the risks of further stroke usually outweigh the potential benefits of delaying surgery to normalize BP.
- Impaired baroreflexes and impaired cerebral pressure autoregulation are common early after TIA or minor stroke. Aggressive lowering of BP may cause ischaemic stroke, especially in the presence of a carotid stenosis. Any new preoperative antihypertensive therapy should be introduced gradually.
- Although perioperative hypertension adversely affects outcome, optimal BP targets before or during CEA are not established. Most vascular anaesthetists would aim for a systolic BP <180mmHg) before urgent CEA, and <160mmHg before elective CEA.
- There is no good evidence for any specific antihypertensive drug, although β-blockers have theoretical advantages in patients with IHD.

Intraoperative management

- Haemodynamic instability is very common during CEA. In addition to hypo-or hypertension, bradycardia may be provoked by vagal stimulation.
- Pattern of BP changes is often different between GA or RA techniques.
- The anaesthetist should anticipate and take measures to prevent haemodynamic instability. The most important phases are induction of GA, laryngoscopy and tracheal intubation (exaggerated responses), carotid cross-clamping and restoration of flow, towards the end of surgery and anaesthesia (maintain normotension) and the early post-operative period (prevent hypertension).

- Hypotension may precipitate myocardial ischaemia and potentially cerebral ischaemia, although it must be emphasized that most intraoperative strokes are caused by thromboembolism or technical surgical error (including shunt malfunction). Hypotension causing watershed cerebral infarction, or hypertension causing intracranial haemorrhage, are rare causes of intraoperative stroke.
- However, it is probably beneficial to avoid hypotension especially during carotid cross-clamping. Relative hypotension during closure of the artery towards the end of surgery may conceal a potential site of haemorrhage should arterial pressure increase later.
- There is no evidence that any drug or technique is best to augment arterial pressure. Suitable drugs are incremental doses of ephedrine, phenylephrine, or metaraminol together with IV fluids, administered promptly and titrated to effect (Table 9.1). Choice of drug also depends on comorbidities, heart rate, and other medications.
- Intraoperative hypertension may also precipitate myocardial ischaemia and augmentation of BP has been associated with an increased risk of MI. Hypertension also makes shunt placement more difficult.
- Hypertension after restoration of cerebral blood flow should be avoided. Large increases in CBF at this point may indicate CHS.
- Overall, it is reasonable to maintain arterial pressure within 20% of baseline values throughout, although there is no definitive evidence to justify this figure.
- If reliable monitoring (including the awake patient) shows that cerebral perfusion is adequate, relative hypotension can be accepted.
- Arterial pressure must be maintained at normal values during closure of the artery. This can help prevent early post-operative bleeding and wound haematoma.
- Hypertension can be avoided or treated by increasing the depth of anaesthesia, judicious administration of opioids, beta blockers (atenolol, esmolol labetalol or metoprolol) or other drugs (Table 9.1). In a patient undergoing surgery under RA, carefully titrated conscious sedation with small doses of propofol or midazolam may be used, taking care to avoid excessive sedation. A low dose target controlled infusion of remifentanil may be useful in the awake patient.
- If post-operative hypertension is anticipated, based on preoperative risk factors (Box 9.3) or intraoperative values, preventative measures should be taken. IV β-blockers are suitable agents.
- Tracheal extubation should be performed smoothly and measures taken to minimize coughing.

Post-operative management

See 🕮 Anaesthesia for carotid surgery, p. 381 and Hypertension after early carotid surgery, p. 398, for management of post-operative hypertension

Table 9.1 Drugs for arterial pressure management during CEA

Hypertension	Hypotension
Clonidine 300μg IV at induction of anaesthesia may attenuate the autonomic responses to surgery.	IV fluids if hypotension is caused by hypovolaemia or dehydration
Deepen anaesthesia using volatile agent, propofol, or remifentanil. Ensure that analgesia is adequate In many cases, these measures alone are sufficient, but intraoperative hypertension may indicate that post-operative hypertension is more likely and preventative measures should be taken	If accompanied by bradycardia (HR<50): Atropine 0.6–1.2mg IV Glycopyrronium bromide 0.2–0.4mg IV Cyclizine 50mg IV is an alternative, especially if nausea is also present Ephedrine 3–6mg IV
If HR >70, try β-blockade Esmolol (bolus of 0.5mg/kg then infusion at 12.5–200μg/kg/min) or Atenolol 2.5mg IV repeated after 5min if needed up to 10mg Labetalol can be administered in increments of 0.15–0.2mg/kg up to 1mg/kg Metoprolol 5mg IV then 2.5mg every 5min up to a maximum of 15mg These drugs are often useful to prevent anticipated hypertension at emergence or recovery from anaesthesia	If HR 50–80beats/min Ephedrine 3–6mg increments IV up to 30mg
If hypertension is associated with chest pain or ECG signs of myocardial ischaemia: GTN 800μg by sublingual spray or 2–20mg/h IV infusion β-blockade is an alternative if HR >70	If HR >80, vasoconstrictor drugs are preferable. Phenylephrine 15–100μg/min or metaraminol 0.5mg are suitable

Box 9.3 Major risk factors for perioperative BP instability

- Recent TIA or minor stroke
- Bilateral carotid artery stenosis
- Previous contralateral CEA or radical neck surgery
- Pre-existing hypertension which is poorly controlled

Further reading

Howell SJ. Carotid endarterectomy. *Br J Anaesth* 2007; **99**: 119–31.

Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. *Br J Anaesth* 2009; **102**: 442–52.

Management of neck haematoma after carotid endarterectomy

- Post-operative wound haematoma is potentially one of the most devastating complications after carotid endarterectomy (CEA).
- Wound haematoma usually develops slowly and, if unrecognized or underestimated, may be fatal. Airway management for surgical re-exploration can be technically very difficult.

Aetiology and predisposing factors

- Haematoma formation after CEA is usually caused by faulty surgical technique. Other predisposing factors include the use of antiplatelet or anticoagulant drugs and post-operative hypertension.
- Bleeding may be arterial (from the suture line at the site of the surgery or from another artery or arteries exposed during the surgical dissection) or venous. In some patients a discrete bleeding point cannot be identified.
- Patients scheduled for CEA are invariably taking antiplatelet therapy (aspirin, dipyridamole modified release (MR), or clopidogrel) for 2° prevention of thromboembolic complications of their carotid disease. Platelet dysfunction may contribute to post-operative bleeding, especially in patients taking clopidogrel.
- Heparin is administered routinely before cross-clamping the carotid artery to minimize the risk of thrombo-embolic complications during carotid artery manipulation, shunt insertion, or early after patch placement. Some centres use a fixed dose of heparin for all patients which may be associated with a wide variation in activated clotting times (ACT) and anticoagulant effect. Individualized patient dosing of heparin should be considered. Reversal of the effect of heparin with protamine is unusual after CEA and, although there is no definite evidence of an association, residual effects of heparin may contribute to post-operative haematoma formation.
- Post-operative hypertension related to transient baroreceptor dysfunction may exacerbate arterial bleeding and cause a neck haematoma. BP must be rigorously controlled in the immediate post-operative period.
- Excessive coughing or vomiting may lead to venous congestion and bleeding.
- Some surgeons employ wound drainage routinely and there is some evidence this reduces the incidence of re-exploration for haematoma.
- The volume of the upper airway is reduced post operatively in all patients undergoing head and neck surgery. CT scanning after CEA demonstrates soft tissue oedema in all patients not only adjacent to the operative site, but also spreading posteriorly to the pharynx and sometimes encompassing the contralateral side. Evidence of a degree of tracheal deviation is frequently seen. This swelling reduces antero-posterior and transverse airway dimensions. If the additional insult of a haematoma is superimposed on this oedema this can further compress the airway and cause respiratory embarrassment.
- The mechanism of oedema after head and neck surgery is lymphatic and venous congestion. The swelling involves the tracheal mucosa, the

supraglottic airway, glottic aperture, and the cricothyroid membrane. Marked oedema of the supraglottic folds makes laryngeal visualization and tracheal intubation difficult.

Assessment

- Vascular units should use appropriate strategies to prevent the development of neck haematomas after CEA, and have established plans for their prompt detection and management. Particular care should be taken to ensure surgical haemostasis before wound closure. Surgical re- exploration should necessary in <5% of cases.
- Early detection and management are essential because airway compromise can occur quickly, tracheal intubation is often difficult and even a short period of hypoxaemia can cause myocardial ischaemia in patients with underlying coronary disease.
- Clinical assessment may underestimate the problem. The degree of external neck swelling may not be helpful, since it is the oedema and internal airway compression that dictates the degree of respiratory compromise.
- The development of stridor on exertion or at rest is an ominous sign. This implies a reduction in airway diameter of at least 50%. The development of stridor is thought to imply an airway diameter of 4mm or less.

Management

- As soon as a neck haematoma causing potential airway compromise is suspected:
  - Administer oxygen by face mask.
  - Call for a senior anaesthetist and vascular surgeon.
  - Sit the patient up.
  - Arrange urgent return to the operating theatre for surgical re-exploration.
  - Blood may need to be cross-matched, but this should not delay surgery.
  - Institute appropriate monitoring before theatre.
- The management technique will depend on the skill, and experience of the surgeon and the anaesthetist. However, the only treatment is prompt surgical evacuation of the haematoma. This removes the cause of the lymphatic and venous obstruction. Airway oedema rapidly resolves and airway obstruction is relieved.
- Evacuation of the neck haematoma under LA is the safest option. If the operation has been performed under cervical plexus blockade and the haematoma develops early in the post-operative period then it is likely the blocks will still be effective.
- If the CEA has been performed under GA or if the LA blocks have worn off, evacuation of the haematoma can be performed after simple infiltration of the wound edges with local anaesthetic.
- GA is fraught with difficulties in these patients. There may be cardiovascular compromise in addition to hypoxaemia and induction of anaesthesia can cause cardiovascular collapse. Laryngoscopy and tracheal intubation are often difficult. A full range of difficult intubation equipment should be available if GA and tracheal intubation is the chosen technique.

- Awake fibre optic intubation is not always successful. A recent publication reported a failure rate of 25%.
- Intubation following direct laryngoscopy may be performed in the awake or anaesthetized patient. A recent publication reported success rates of 71 and 87% when performed either before or after the induction of GA.
- Successful evacuation of the haematoma using a laryngeal mask airway (LMA) for airway maintenance has been reported.
- After successful drainage of the haematoma, the airway should be evaluated carefully for residual oedema before tracheal extubation. In some cases, a period of post-operative artificial ventilation in ICU may be required to allow residual oedema to subside. In all cases, close observation is required in a monitored environment for several hours after tracheal extubation.

Further reading

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Munro FJ, *et al*. Airway problems after carotid endarterectomy. *Br J Anaesth* 1996: **76:** 156–9.

Shakespeare WA, *et al*. Airway management in patients who develop neck hematomas after carotid endarterectomy. *Anesthes Analg* 2010; **110**(2): 588–93.

Hypertension early after carotid surgery

Acute hypertension commonly occurs after carotid surgery. It is usually transient and peaks in the first few hours after surgery, and is related to arterial baroreceptor dysfunction. Carotid atheroma *per se* interferes with baroreceptor reflexes and cerebrovascular reactivity, increasing the likelihood of perioperative hypo- or hypertension even in asymptomatic patients. This is exacerbated by surgery, where removal of a carotid plaque causes immediate partial disruption of baroreceptor activity leading to hypertension and increased arterial pressure instability for several hours or days. Hypertension should be assessed and treated promptly because it predisposes to wound haematoma, myocardial ischaemia and in some cases may indicate cerebral hyperperfusion syndrome (see 📖 Cerebral hyperperfusion syndrome, p. 403).

Predisposing causes

- Pre-existing chronic hypertension, especially if poorly controlled.
- *Recent TIA or stroke:* these cause short-term baroreceptor dysfunction independently of any surgery or intervention. Perioperative hypertension is more common when CEA is performed within 2 weeks of a TIA or stroke.
- Autonomic dysfunction, e.g. in diabetes.
- Omission of regular antihypertensive medication.
- Post-operative hypertension is more common in patients with bilateral carotid disease; previous contralateral CEA or radical neck surgery.
- Eversion endarterectomy or carotid sinus nerve blockade.

- Post-operative hypertension may be more common after CEA performed under GA compared with RA.

Other causes
- Post-operative pain.
- Urinary retention.
- Anxiety.
- Hypercapnia.

Definitions

Precise definitions vary, but a systolic pressure >170mmHg (or >30% of preoperative values) baseline should be monitored closely and treated promptly if it persists for more than 10–15min. If there are any neurological symptoms (headache, visual disturbance), any systolic pressure > 140mmHg should be treated immediately.

Presentation assessment and differential diagnosis

- Patients are usually asymptomatic.
- Headache, visual disturbances, photophobia, nausea, and seizures suggest CHS (see 🕮 Cerebral hyperperfusion syndrome). Look for factors associated with CHS (Box 9.4) and adopt a more aggressive approach to BP monitoring and control in these patients.

Investigations and monitoring

The following should be monitored:
- Invasive arterial pressure monitoring in all patients for 2–4h after surgery.
- ECG.
- Oxygen saturation (SpO_2).
- Respiratory rate.
- Conscious level and neurological symptoms.
- ABG (check for hypercarbia).
- Neck wound and drainage (check for haematoma).

Immediate management

Sit the patient up in bed. In mild cases, this is often sufficient. Check whether the patient has received their usual antihypertensive medication.
- Administer oxygen 4L/min by face mask or nasal cannulae.
- Titrate oxygen to maintain SpO_2 >95%.
- Institute monitoring as above.

Box 9.4 Risk factors for cerebral hyperperfusion syndrome
- Recent ipsilateral ischaemic stroke.
- Severe ipsilateral or contralateral carotid disease.
- Markedly increased cerebral perfusion (middle cerebral artery (MCA) flow velocity or pulsatility) after flow restoration.
- Severe post-operative hypertension.

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Look for and treat
- Post-operative pain.
- Urinary retention.
- Anxiety.
- Chest pain.
- Nausea and vomiting.

Further treatment depends on whether the patient:
- Can tolerate oral medication.
- Is receiving long-term antihypertensive medication.
- Has any neurological symptoms or signs (see 📖 Endovascular abdominal and thoracic aortic aneurysm repair, p. 363).

See Box 9.5 for drugs, routes and doses for use in the early post-operative patient (in PACU/HDU). Box 9.6 lists treatment options for patients who become hypertensive on the general ward after surgery.

Further management

- All patients who require IV anti-hypertensive medication should undergo invasive arterial pressure monitoring, continued for 2h after last dose to observe for rebound effects. Patients in whom CHS is suspected should be managed in a HDU environment and invasive arterial pressure monitoring continued for at least 6h after IV medication is discontinued (see 📖 Cerebral hyperperfusion syndrome, p. 403)
- When BP is stabilized, the patient may be transferred to a ward, but BP should be recorded hourly for a further 24h.
- The patients usual antihypertensive medication should be restarted as soon as possible, unless systolic BP is <120mmHg.
- Seek advice from a hypertension specialist regarding continuing further medication.

Pitfalls/difficult situations

Neck haematoma

See 📖 Management of neck haematoma after carotid endarterectomy, p. 396. This can cause airway compression and life-threatening airway obstruction. Emergency evacuation of the haematoma in theatre is required. In addition to the above:
- Contact the responsible vascular surgeon.
- Seek senior help.
- Check FBC, blood coagulation, group and save (G&S).
- Contact the theatre team with a view to returning to theatre for surgical exploration.
- Evacuation of the haematoma is best undertaken surgically using local anaesthetic infiltration.

Box 9.5 Management of early post-operative hypertension (PACU/HDU)

1. Systolic pressure remains >170mmHg despite simple measures. Patient unable to swallow tablets (early post-operative period)

First line labetalol 10mg slow IV boluses every 2min, up to 100mg.

- If BP remains elevated after 20min, move to second line agent.
- If BP decreases and does not rebound, continue regular BP observations.
- If BP decreases initially, but increases again, start labetalol infusion at 50–100mg/h, titrating dose to BP.

Second line drugs hydralazine (2–10mg slow IV injection) or GTN infusion (0–50mg/h).

- Invasive arterial pressure monitoring should be continued for 2h after administration of parenteral agents to observe for rebound hypertension.
- Restart any usual antihypertensive medication when feasible. Consider administration of oral antihypertensive medication before return to the ward

2. Systolic pressure remains >170mmHg despite simple measures. Patient is able to swallow tablets.

Patient NOT normally on antihypertensive therapy

First line nifedipine LA (10mg), repeat after 1h if no change in BP.

Do not use crushed nifedipine capsules. If no reduction in BP, move to second line agent

Second line bisoprolol 5mg.

Third line ramipril 5mg, repeated at 3h if necessary.

Patient is normally on antihypertensive therapy

First line Administer patient's usual medication.

Second line If patient is on ACE inhibitor and/or diuretic, nifedipine LA 10mg. If patient is on Ca^{2+} channel blocker, ramipril 5mg.

Observe closely for neurological signs or symptoms, and treat immediately if these occur.

Monitor and treat hypertension more aggressively in patients with risk factors for CHS (Box 9.2). Aim to maintain systolic BP in these patients below 140mmHg.

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Box 9.6 Management of hypertension presenting after discharge to the ward

Systolic BP >170mmHg, but NO headache/neurological symptoms suggestive of CHS

There are three scenarios:

- Patient is not normally on antihypertensive therapy.
- Patient is normally on antihypertensive therapy.
- Patient cannot swallow tablets.

Patient is NOT normally on antihypertensive therapy

First line nifedipine LA (10mg), repeated after 1h if no change in BP.

- **Do not use crushed nifedipine capsules.**
- If no reduction in BP, move to second line agent.

Second line bisoprolol 5mg.

If either contra-indicated or no effect, move to third line agent.

Third line ramipril 5mg, repeated at 3h if necessary.

Contact hypertension specialists for clinical review

Patient IS normally on antihypertensive therapy

First line Check the patient has received normal anti-hypertensive medication. If not, administer this.

Second line A, ACE inhibitor; B, β-blocker; C, Ca^{2+} channel blocker; D, diuretic.

- If patient is on A, add in C (nifedipine LA 10mg).
- If patient is on C, add in A (ramipril 5mg).
- If patient is on D, add in A (ramipril 5mg).
- If patient is on A + C, add in D (bendroflumethiazide 2.5mg).
- If patient is on A + D, add in C (nifedipine LA 10mg).
- If patient is on A + C + D, add in B (bisoprolol 5mg).

Contact Hypertension Specialists for clinical review

Patient cannot swallow tablets

Pass NGT and administer appropriate medicines in liquid form as prescribed above.

Contact hypertension specialists for clinical review

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Cerebral hyperperfusion syndrome.

See 🕮 Cerebral hyperperfusion syndrome, p. 403.

Chest pain

Initial management as above.

- Administer oxygen by face mask as above.
- Check 12-lead ECG for myocardial ischaemia, acute MI; treat accordingly.
- Seek senior help.

Further reading

Howell SJ. Carotid endarterectomy. *Br J Anaesth* 2007; **99**: 119–31.
Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. *Br J Anaesth* 2009; **102**: 442–52.

Cerebral hyperperfusion syndrome (CHS)

Restoration of CBF during CEA, carotid angioplasty with stenting, or certain other cerebral vascular procedures almost always results in a 20–40% increase in blood flow to the brain. This usually subsides over minutes or hours without problems. However, in some patients, the increase in CBF is much larger, 100–200% of baseline, peaking at days 3–4 after surgery and resolving within 7 days, but occasionally persisting for up to 2 weeks. Cerebral hyperperfusion (CH) is usually defined as an increase in CBF to >100% of preoperative values. When CH persists for several days associated with decreased cerebrovascular reserve it can lead to CHS, which occurs in 1 or 2% of patients after CEA, and is usually associated with postoperative hypertension, which may be severe. Conversely, patients may present with clinical features of CHS, but arterial pressures and CBF are normal.

Definition and clinical features

CHS is usually defined as severe ipsilateral migraine-like headache with transient focal neurological deficits and seizures, associated with CH. It typically occurs 0–7 days after CEA (although it can be later). Clinical and pathological features are similar to hypertensive encephalopathy.

Clinical features include:

- Throbbing frontal, temporal, periorbital, or sometimes diffuse headache.
- Ocular or facial pain.
- Nausea and vomiting.
- Visual disturbances with macular oedema.
- Focal neurological deficits.
- Seizures (focal or generalized): these can precede other neurological symptoms.
- Intracerebral or subarachnoid haemorrhage.
- Systemic hypertension (usually).

Predisposing factors

CHS typically occurs in a patient with diminished cerebrovascular reserve, increased post-operative cerebral perfusion and post-operative hypertension lasting more than a few hours.

Established predisposing factors include:

- Diminished cerebrovascular reserve.
- Preoperative hypoperfusion.
- Markedly increased cerebral perfusion (increase in MCA flow velocity or pulsatility >100% of baseline) after flow restoration.

- Severe ipsilateral or contralateral carotid disease (occlusion or stenosis >90%).
- Recent ipsilateral ischaemic stroke.

Other suggested risk factors include poor collateral brain blood flow, long standing or poorly-controlled systemic hypertension, diabetes, old age, intraoperative cerebral ischaemia, severe vertebral stenosis, and recent (within 3 months) contralateral CEA.

Prevention

There is some evidence that the risk of CHS is increased if CEA is performed within 4 weeks after an ischaemic stroke. However, current guidance is that the risk of major stroke is increased within the first few days after a TIA or minor stroke. Current NICE recommendations are that CEA should be performed within 2 weeks and preferably within 48h of a TIA. It is unclear whether this practice will increase the risk of CHS in susceptible patients.

Contralateral CEA within 3 months is thought to be a risk factor for CHS so subsequent CEA surgery should be delayed in neurologically stable patients

Pathophysiology

Thought to be similar to hypertensive encephalopathy. Severely impaired/absent cerebral autoregulation leads to vasogenic ipsilateral cerebral oedema formation after carotid surgery in previously underperfused areas of the brain. This is compounded by impaired baroreflex function after surgery. Cerebral oedema predominantly affects the white matter in areas of the posterior cerebral circulation, in particular, the occipital lobe. It can progress to endothelial damage causing further hyperperfusion with petechial haemorrhage, fibrinoid necrosis, focal ischaemic cerebral infarction and marked intracranial hypertension. The initial oedema is reversible with aggressive treatment of arterial pressure.

Immediate management

Sit the patient up in bed.

- Administer oxygen 4L/min by face mask or nasal cannulae.
- Institute monitoring as below.
- Treat hypertension as per Box 9.7.
- Arrange for transfer to HDU/ICU for invasive arterial pressure monitoring.
- Consider investigations as Box 9.7.

Monitoring

Invasive arterial pressure

- ECG.
- SpO_2.
- Respiratory rate.
- Conscious level and neurological symptoms.

Box 9.7 Management of patient with suspected CHS.

Systolic BP >160mmHg with headache/other neurological symptoms/deficit

- Treatment should start immediately on the ward using non-invasive monitoring
- On-call surgical specialist registrar (SpR)/ senior house officer (SHO) must:
  - Inform on-call consultant vascular surgeon of increase in BP associated with seizure/headache or onset of neurological deficit.
  - Contact on-call ICU team to arrange urgent transfer to surgical acute care unit (SACU), HDU, or PACU for invasive arterial BP monitoring.
  - Administer 8mg dexamethasone IV.
  - Check for and treat pain, and urinary retention.
  - Aim to reduce BP gradually to <140mmHg systolic, and lower if symptoms persist.

Seek expert advice early

First line labetalol 10mg slow IV boluses every 2min, up to 100mg.

- If BP remains elevated after 20min, move to second line agent.
- If BP decreases and does not rebound, continue regular BP observations.
- If BP decreases initially, but increases again, start labetalol infusion at 50–100mg/h, titrating dose to BP.

Second line drugs hydralazine (2–10mg slow IV injection) or GTN infusion (0–50mg/h):

- If BP remains elevated after 25min, move to third line agent.
- If BP reduces and does not rebound, continue regular BP observations.
- If BP reduces, but increases again, move to third line agent

Third line GTN 50mg in 50mL 0.9% NaCl (i.e. 1mg/mL) start infusion at 5mL/h (5mg/h), increasing rate to 12mL/h (12mg/h), titrated to BP.

When BP stable (in HDU/ICU), commence oral medication

Patient not normally on antihypertensive therapy

First line nifedipine LA (10mg), repeat after 1h if no change in BP.

Do not use crushed nifedipine capsules. If no reduction in BP, move to second line agent

Second line bisoprolol 5mg.

Third line ramipril 5mg, repeated at 3h if necessary.

Patient is normally on antihypertensive therapy

First line Administer patient's usual medication.

Second line If patient is on ACE inhibitor and/or diuretic, nifedipine LA 10mg. If patient is on Ca^{2+} channel blocker, ramipril 5 mg.

- Following transfer, patient should remain in SACU, PACU, or HDU while anti-hypertensive treatment ongoing.
- Following cessation of treatment, the patient should remain in SACU, PACU, or HDU for a minimum of further 6h to minimize rebound hypertension.

Adapted with permission from Stoneham M.D. Thompson JP, 'Blood Pressure Management and Carotid Endarterectomy', British Journal of Anaesthesia, 2009, 102, pp. 442–452, by permission of Oxford University Press and the Board of Management and Trustees of the British Journal of Anaesthesia.

Investigations

Management of BP must not be delayed to perform investigations.

- *TCD:* may show marked increase in middle cerebral artery flow velocity or pulsatility. Provides real time information on MCA perfusion, cerebral vasoreactivity and emboli. MCA flow velocity typically increased by 100–300% in CHS.
- *CT scan:* typical features of CHS are diffuse white matter oedema predominantly affecting posterior parietal or occipital lobes, ispilateral petechial or overt haemorrhages, or mass effects. However, CT may be normal particularly in the first hours after the onset of symptoms.
- *MRI scan:* more sensitive than CT, but can also be normal. In addition to demonstrating white matter oedema, haemorrhage, or mass effects, diffusion-weighted MRI (available in specialist centres) can be used to exclude cerebral ischaemia. Perfusion-weighted MRI can show subtle changes in cerebral blood flow.

Further management

- Aim to keep systolic BP <140mmHg, lower if symptoms persist.
- Treat fits with lorazepam 2mg IV initially (dose may be repeated).
- Second line treatment is phenytoin IV 20mg/kg IV loading dose followed by 100mg 8-hourly.

Further reading

Stoneham MD, Thompson JP. Blood pressure management and carotid endarterectomy. *Br J Anaesth* 2009; **102**: 442–52.

Van Mook MNKA, Rennenberg RJMW, Schurink GQ, *et al* Cerebral hyperperfusion syndrome. *Lancet Neurol* 2005; **4**: 8.

Subclavian steal syndrome

- *Procedure:* carotico–subclavian bypass involves insertion of a tunnelled prosthetic graft. Subclavian transposition requires detachment of the subclavian artery from is origin and re-anastomosis to the ipsilateral carotid artery.
- *Time:* 2–3h.
- *Pain:* moderate/severe. PCA frequently required.
- *Position:* supine. Operative arm abducted on an arm board.
- *Blood loss:* minimal. Group and save required.
- *Hospital stay:* 5–7 days.
- *Practical aspects:*
  - Balanced GA required. Invasive arterial monitoring used if significant cardiovascular co-morbidity.
  - Ipsilateral carotid artery clamped temporarily.
  - Systemic heparinization required. Incidence of stroke is very low.
  - Carotid shunting not usually required.

The term subclavian steal syndrome was first used by Fisher in 1961 to describe neurological symptoms during or immediately following exercise of the ipsilateral arm. The aetiology was proposed to be retrograde flow in the vertebral artery associated with ipsilateral proximal subclavian artery stenosis or occlusion.

In 1974, Harjola and Valle described a patient with proximal subclavian stenosis who reported angina after arm exercise following coronary artery bypass surgery, using an ipsilateral internal mammary artery conduit. They used the term coronary subclavian steal to differentiate it from the previously recognized vertebral subclavian steal. Angina is caused by retrograde flow in the internal mammary conduit 'stealing' blood from the heart.

Presentation

- Muscle cramping due to ischaemia may occur in manual workers who perform vigorous exercise of the arms, often with their arm elevated above their head.
- Neurological symptoms are due to ischaemia in the posterior cerebral circulation. Fortunately, these rarely progress to cerebral infarction.
  - Dizziness or vertigo occur commonly (>50%).
  - Visual symptoms may include transient visual loss, double vision, or the sensation that objects are moving.
  - Transient loss of consciousness may occur.
- Stenosis or occlusion of the proximal vertebral artery may produce similar neurological symptoms. Occlusive disease of the vertebral artery should be considered if posterior circulation symptoms occur with normal BP's in the ipsilateral arm.
- The most commonly reported cardiac symptom is angina. Coronary subclavian steal has been implicated as a cause of congestive cardiac failure, ischaemic cardiomyopathy and myocardial infarction.

Investigations

- A marked reduction in BP on the symptomatic side is almost invariably found in patients with subclavian steal.
- A CXR should be performed to look for unusual causes of subclavian stenosis, e.g. cervical rib.
- Doppler ultrasonography is most commonly used to diagnose vertebral subclavian steal. It is important to insonate the ipsilateral carotid artery to look for isolated stenoses if surgical intervention is planned. Ultrasonography is not useful to assess flow in the proximal subclavian artery.
- CT angiography with contrast gives excellent information of the nature of arterial lesion. Advantages include:
  - Does not require direct arterial puncture.
  - Can be used to plan surgical repair in detail.
- If endovascular treatment is being considered it may be more appropriate to proceed to conventional angiography, as treatment can be performed at the same time
- Magnetic resonance angiography can be used especially if there is pre-existing renal dysfunction; contrast is not required. It tends to overestimate the degree of arterial obstruction.

- A valid group and save should be performed before intervention is planned. Large bore IV access is mandatory because of the risk of bleeding.
- Other investigations depend on patient fitness and comorbidity.

Management

- Isolated subclavian stenosis can be treated by endovascular balloon angioplasty and stenting. Success rates are high, but restenosis rates of 6–20% have been reported.
- If the subclavian artery is occluded or endovascular repair is unsuccessful an extrathoracic carotico-subclavian bypass may be used. Surgical exposure is through a transverse incision at the base of the neck extending laterally from the sternal notch parallel to the clavicle. A prosthetic graft is tunnelled and anastomosed end to side to the subclavian artery.
- Alternatively subclavian transposition may be performed. The subclavian artery is anastomosed end to side onto the common carotid artery. This operation is performed through a transverse incision at the base of the neck and has the advantage of not requiring prosthetic material. The dissection is more extensive—care must be taken to avoid injury to the thoracic duct on the left side. Long-term results of subclavian transposition procedures are similar to those of carotid-subclavian bypass.
- A balanced general anaesthetic is required. The patient is usually placed in the supine position.
- The carotid artery must be cross clamped temporarily during open surgery. 5000IU of heparin is administered IV prior to cross-clamping. The short period of cortical ischaemia is generally well tolerated; the incidence or perioperative stroke is very low. Carotid shunting is not usually required.
- Post-operatively the patient must be observed closely for signs of bleeding.
- The peripheral circulation can be monitored using Doppler US. It is difficult to measure BP in the operative arm, particularly if a carotico-subclavian bypass has been preformed.

Acute upper limb ischaemia

Upper limb ischaemia is much less common than lower limb ischaemia. In part, this is due to the presence of a good collateral circulation around the shoulder. Acute upper limb ischaemia is a surgical emergency since the ischaemic tolerance of the upper limb is much less than the lower limb; tissue loss can occur rapidly.

Presentation

The patent may complain of an acutely painful arm, which may be weak and paraesthetic. Pulses are absent.

Aetiology

- Usually embolic.
- The source is most frequently the heart (AF or post-MI).
- Rarely, there may be a paradoxical venous embolus if there is a patent foramen ovale or a septal defect.
- Emboli can arise in the aorta following a thoracic dissection.
- Emboli may arise in a subclavian artery aneurysm which may arise as a result of a cervical rib or arterial thoracic outlet syndrome (see 📖 Thoracic outlet syndrome, p. 422).
- Iatrogenic damage to the subclavian artery during attempted subclavian venous cannulation may result in emboli to the upper limb.

Management

- Emergency brachial embolectomy to restore blood flow is usually preformed under LA. Monitored anaesthesia care is recommended; patients frequently have significant co-morbidity, and may be restless and in pain.
- Rarely, GA may be required, particularly if the upper limb has been ischaemic for some time and the patient is uncooperative. Occasionally, a compartment syndrome may occur in the forearm muscles; fasciotomies may be required.

Further reading

Takach TJ, Reul GJ, Cooley DA, *et al*. Myocardial thievery: the coronary-subclavian steal syndrome. *Ann Thor Surg* 2006: **81**: 386–92.

Combined carotid endarterectomy and coronary procedures

- *Procedure:* CEA is performed after induction of anaesthesia, with all the monitoring necessary for CABG, including neurological monitoring, before proceeding to CABG. Draping of the patient will be for the combined operation. Vein harvesting (for CABG) will usually be undertaken during the CEA. After CEA, the skin is left open (packed with a swab) and closed at end of operation to ensure that no haematoma collects when the patient is heparinized for cardiopulmonary bypass (CPB).
- *Time:* 4–5h (an additional 1–2h more than CABG).
- *Pain:* minimal beyond that expected for CABG.
- *Position:* supine, with 10° head up.
- *Practical technique:* anaesthesia as for CABG, but additional cerebral monitoring may be indicated. Maintaining BP at the preoperative level is important before completion of CEA. This is often significantly higher than would be usual for patients with CAD.

General considerations

- Patients presenting for combined operations (usually CABG, rather than valve surgery) may have been referred for CEA and found to have CAD, or may have been referred for CABG and found to have carotid artery disease.
- For a combined procedure to be considered, patients usually have unstable angina **and** a recent TIA, both of which are amenable to surgery.
- The decision to undertake combined CEA and CABG is taken after discussion between the cardiac and vascular surgeons.
- Anaesthetists involved in these cases should be competent in both vascular and cardiac anaesthesia.
- Patients often have other manifestations of vascular disease, e.g. PVD.
- There is no clear evidence that combined procedures carry a greater risk than sequential procedures (CEA followed by CABG).
- The risk of mortality after CABG is determined using the EuroSCORE II calculator. This includes presence or absence of carotid stenosis >50% in its risk prediction score. The mortality risk (EuroSCORE II) is greater in a combined procedure for two reasons:
  - The presence of a carotid stenosis increases mortality from CABG.
  - The additional procedure adds risk.
- In practice, the EuroSCORE II risk cannot be used to accurately predict differences in mortality between the two techniques.
- Estimates of risk of stroke after CABG depend on the presence/absence of previous stroke and known carotid artery stenosis. Age is also a significant predictor of stroke with the risk increasing significantly (>10%) in patients aged > 80yrs. There is no evidence that this risk is different between combined or sequential procedures.

Pre-operative assessment

- Pre-assessment of these patients often occurs on the ward, rather than the pre-assessment clinic, because most patients present for urgent surgery after an acute admission to hospital.
- Assessment should focus particularly on the cardiovascular and neurological systems.
- Ascertain the following:
  - Is there a history of previous myocardial infarction?
  - What is the frequency and severity of angina?
  - Is angina stable or unstable?
  - Is HF present?
  - What is the severity of HF?
  - Is drug treatment optimized? (β-blocker, ACE inhibitor, statin, aspirin)?
  - Is there a history of previous stroke or TIA? Are any residual symptoms or deficits present? What is the time interval since resolution of symptoms?
- The carotid artery is usually assessed by Doppler ultrasound or MRI
- The extent of the coronary artery disease is assessed by coronary angiography and echocardiography.

- Detailed neurological assessment should be performed to allow comparison post-operatively.
- Hypertension is common and the baseline BP should be noted.
- Dual anti-platelet therapy (aspirin and clopidogrel) is commonly started when patients have a TIA, and one or both drugs may still be being taken at the time of surgery. Stopping these drugs in the presence of critical CAD or carotid artery disease can be dangerous.

Risk scoring

- Risk scoring is routine for all patients undergoing cardiac surgery. The EuroScore II is the scoring system used and additional risk is recognized for extra-cardiac arteriopathy. This includes carotid stenosis >50%. Most combined procedures will be done urgently (during an acute admission), which also carries additional risk.
- The presence of a 50% carotid artery stenosis more than doubles the risk of death in a 70-yr-old patient undergoing CABG.

Sequential CEA and CABG

- Stable CAD (requiring surgery) in people who have carotid artery disease (requiring surgery) is usually treated after CEA is complete.
- Approximately 1 month should pass after CEA before CABG to ensure that no complications have occurred and that any period of hypercoaguability has ended.

Reverse sequential CEA and on CABG

CABG before CEA is only performed when CABG is performed as a salvage procedure because of the known additional risk of stroke using this technique.

Combined CEA and CABG

With a combined procedure CEA is always performed under general anaesthesia before cardiac revascularization. Vein harvesting will often be performed concurrently.

Monitoring

- *ECG:* 5-lead ECG monitoring will allow easier detection of myocardial ischaemia.
- *Arterial pressure:* monitoring is essential and the arterial cannula should be sited before induction of anaesthesia.
- *Central venous pressure:* should be monitored to allow easy titration of vasoconstrictors/vasodilators as required.
- *Cardiac output monitoring:* should be used if required for the cardiac surgery. The addition of CEA should not influence this.
- *Transoesophageal echocardiography:* can be used successfully during combined procedures, but manipulation of the probe may be difficult during CEA, when it may interfere with the surgery.
- *Intraoperative neurological monitoring:* should be as for any CEA being performed under GA.
- *Cerebral oximetry:* is now used widely during complex aortic surgery, is available in many cardiac centres and may be used in some centres to help assess the need for shunting during carotid cross-clamping.

- *Stump pressures:* may also be measured and used for same reason.
- *Jugular bulb catheters and measurement of jugular venous saturation:* less common since cerebral oximetry now more widely available.
- *Blood glucose:* should be monitored and controlled.
- *Temperature:* if the patient is going to have CABG using CPB, the temperature can be allowed to drift down during CEA (cerebral protection) knowing that the patient will be re-warmed before discontinuation of CPB.

Practical considerations and techniques

- Premedication is common for cardiac surgery, although when used for combined CEA and CABG it may make a 'final' pre-operative neurological assessment difficult.
- Supplementary oxygen should be prescribed if any pre-medication is given.
- Arterial lines should always be placed before induction of anaesthesia. Central venous lines are commonly placed before induction of anaesthesia. Central venous catheters should be placed on the opposite side to the CEA.
- Use of US to guide central line placement minimizes risk of carotid artery puncture in a patient with a compromised cerebral circulation.
- Hypotension during induction of anaesthesia should be prevented by the use of vasoconstrictors (ephedrine, phenylephrine, or metaraminol).
- An infusion of noradrenaline should be available before induction of anaesthesia and commenced as soon as required after intubation.
- Arterial pressure should be kept higher than would be the case if there was no carotid artery disease (within 20% of the baseline pressure) before completion of CEA.
- Prophylactic antibiotics are given routinely according to local protocol.
- Positioning is preference of surgeon, but usually includes some head-up position to increase venous drainage and head turned to the contra-lateral side. Extension of neck is also common. A head ring, rather than a pillow helps to stabilize the head.
- Whereas LA is commonly used in addition to general anaesthesia for CEA, this is often omitted in the combined procedure given fact that patient will be asleep for a number of hours after the infiltration.
- LA is sometimes injected by surgeon around carotid body in an attempt to minimize haemodynamic disturbance during dissection and manipulation.
- Heparin is administered before carotid cross-clamping and a baseline ACT should be performed before this.
- Low-dose heparin (compared with dose to anticoagulate before CPB) is used, often 5000IU.
- Decision to use a shunt depends on local preferences as for CEA performed as a sole procedure under GA, and may be guided by appropriate cerebral monitoring.
- Maintaining normotension is important during cross-clamping of the carotid artery.

- The surgeon should pay meticulous attention to haemostasis during CEA, especially as systemic heparinization is usual for CPB. After completion of the CEA, the wound is left open, packed with a swab so that final haemostasis can be completed as the chest is being closed. A small drain is placed in the wound.

Post-operative

- Early assessment of neurological function is desirable after CEA, but this is impractical after combined CEA and CABG. A period of postoperative artificial ventilation and sedation is common after cardiac surgery, and local protocols should be followed regardless of fact that CEA has been performed.
- Vasoconstrictors or vasodilators should be used as needed after any CABG operation. The need to maintain the same BP as pre-operative values is less important after completion of CEA
- Cerebral hyperperfusion syndrome may occur as after CEA as a single procedure, but it can be difficult to distinguish whether swings in BP are related to cardiac instability or hyperperfusion.
- The CEA wound must be monitored for evidence of haematoma or bleeding.
- Dual anti-platelet therapy is often re-introduced the day after surgery.

Further reading

Naylor AR, Cuffe RL, Rothwell PM, Bell PRF. A systematic review of outcomes following staged and synchronous carotid endarterectomy and coronary artery bypass. *Eur J Vasc Endovasc Surg* 2003; **25**: 380–9.

Venkatachalama S, Shishehborb MH. Management of carotid disease in patients undergoing coronary artery bypass surgery: is it time to change our approach? *Curr Opin Cardiol* 2011; **26**: 480–7.

Anaesthesia for lower limb vascular bypass surgery

- *Procedure:* bypass of diseased arterial segment with autologous vein or prosthetic graft.
- *Time:* 2–6h.
- *Pain:* moderate; epidural analgesia, lower limb nerve blocks, IV opioids or PCA may be required.
- *Position:* supine.
- *Blood loss:* variable, usually slow and insidious.
- *Hospital stay:* 5–10 days.
- *Practical aspects:* balanced GA or regional (epidural or CSE) are both appropriate. Spinal anaesthesia alone may be adequate for brief simple proximal procedures. Surgery may be prolonged. Temperature control and active warming essential. Lumbar plexus or femoral nerve blocks useful for post-operative analgesia.

PVD affects 5–7% of the middle aged and elderly population. The annual mortality rate is 4–6% and is highest in those with more severe disease.

- Risk factors for PVD include hypertension, tobacco use, diabetes, and old age. Progressive symptoms are cold extremities, paraesthesiae, intermittent claudication (IC), rest pain, and gangrene.
- Over 90% of patients have CAD; many have COPD, but because exercise capacity is limited, patients may not complain of any cardio-respiratory symptoms.
- Symptoms of PVD may respond to lifestyle changes (cessation of smoking, weight loss, graded exercise programme). The majority of patients can be treated by radiologically by percutaneous angioplasty (see 🕮 Chapter 8 Anaesthesia for vascular radiology, p. 333). Those presenting for surgery often have extensive, diffuse, or advanced disease.
- The overall risks of surgery (MI or cardiac death >5%) are similar to open aortic surgery. Anaesthesia for aorto-iliac occlusive disease is dealt with in 🕮 Emergency abdominal aortic aneurysm repair: open and endovascular repair, p. 448.

Indications for surgery

- Ischaemic rest pain.
- Tissue loss (ulceration or gangrene).
- Severe claudication with distal disease.
- Failure of non-surgical treatment.

Preoperative assessment

The principles of basic vascular preoperative assessment apply (🕮 Chapter 3 Evaluation of the vascular surgical patient). The risks of peripheral vascular reconstruction (PVR) are high because:

- Co-existing CAD is almost inevitable.
- The incidence of diabetes is increased.
- Symptoms of CAD are masked by immobility related to IC, old age, and arthritis.
- Surgery is often prolonged with the risk of hypothermia, blood loss, and cardiovascular instability
- Myocardial ischaemia is common and frequently silent.
- Patients are often elderly, frail, and immobile.
- Surgery for critical limb ischaemia is often urgent with limited time for investigations.
- Ability to exercise is limited by PVD so assessment of functional capacity is difficult
- Preoperative assessment should focus on detecting major cardiac risk factors:
  - Unstable coronary syndromes.
  - Decompensated heart failure.
  - Significant arrhythmias.
  - Significant valve disease.

These should be investigated (see 🕮 Evaluation of the vascular surgical patient, p. 85) and treated before surgery.

- Other important risk factors are a history of:
  - IHD.
  - HF.
  - CVD.
  - Diabetes.
  - Renal insufficiency.
  - COPD or asthma.
  - Current cigarette smoking.
  - Hyperlipidaemia.
  - Polycythaemia.
  - Homocysteinuria.

Surgery is urgent, but not usually an emergency. Therefore these conditions should be detected and optimised as far as possible before surgery.

Investigations and preoperative preparation

Routine investigations should be performed. Invasive cardiological investigations should only be undertaken if they would change management. If surgery is urgent (e.g. to salvage a limb) or the patient is not a candidate for coronary intervention, further investigation is not appropriate. Patients should be commenced on antiplatelet medication (usually aspirin) and statin therapy if they are not already receiving these drugs and there are no contraindications. There is some evidence that long-term cardiovascular outcome is improved with ACE inhibitors and these may be considered (see 🕮 Management of specific medical conditions and medications, p. 159).

- Spirometry and ABG are advised if there are symptoms of respiratory disease.
- Blood should be grouped and saved, and preparations made for postoperative management (pain control, HDU bed if required).
- Routine medications should generally be continued up to the day of surgery. Some patients will be receiving long-term opioids and may have developed tolerance. It may be useful to involve pain management specialists in their perioperative care.
- IV rehydration is advised for 12–24h before surgery, especially if radiological contrast has been administered.
- A sliding scale of insulin and glucose is required for diabetic patients.
- If patients are receiving warfarin, the indications for this should be reviewed. In some patients (e.g. atrial fibrillation with no other risk factors) warfarin may safely be omitted. Other patients (e.g. those with metallic mitral or aortic valve replacements may require bridging therapy with therapeutic doses of low molecular weight heparin. This must be stopped 24h before surgery (see 🕮 The anti-coagulated patient, p. 215).

Anaesthetic technique

RA, balanced GA or a combination are suitable techniques. RA (spinal or epidural blockade) has theoretical advantages in terms of peripheral blood flow, but does not affect overall outcome. Regional techniques may be contra-indicated in patients receiving therapeutic doses of anticoagulants. Close attention should be paid to maintaining body temperature,

normovolaemia, normotension, and providing good analgesia. Heart rate control using β-blockade should be considered. Surgery may be prolonged (several hours)

- Institute monitoring and IV lines before induction of anaesthesia. A wide bore cannula is required as rapid blood loss may occur.
- Minimal monitoring includes multi-lead ST segment ECG, temperature, urine output, invasive arterial pressure (procedures >2h). CVP and/or cardiac output monitoring should be considered in patients identified as likely to be haemodynamically unstable.
- Induce anaesthesia as smoothly as possible.
- If GA is used, balanced anaesthesia (O_2, air, or nitrous oxide and a volatile agent) using positive pressure ventilation is preferable. Neuromuscular blockade is usually used to facilitate intubation and IPPV.
- The airway can be managed using a tracheal tube or supraglottic airway device.
- Cardiovascular stability should be maintained throughout using fluids, vasopressors and/or positive inotropic drugs. IV heparin 5000IU is usually administered before interruption of blood flow to the operative limb and graft placement. Restoration of blood flow to an ischaemic limb following surgical reconstruction may cause transient myocardial depression and hypotension. This can be treated using IV fluids, ephedrine (increments of 3–6mg IV) or phenylephrine (increments of 50–100micrograms IV).
- Maintenance of normothermia is vital. Perioperative hypothermia causes vasoconstriction that may impair post-operative graft perfusion. It also predisposes to myocardial ischaemia and dysrhythmias, may contribute to coagulopathy and increases incidence of wound infections. Shivering can increase O_2 consumption up to 6-fold, placing excessive demands on the cardiovascular system. Active warming devices (forced air warming, thermal mattress) and insulation of exposed areas are mandatory. Fluids should be warmed. Potential pressure areas should be protected.
- PVR can be painful and post-operative pain can cause vasoconstriction. Combination of morphine 0.15–0.25mg/kg with a regional block or LA infiltration and NSAIDs (if not contraindicated) is usually satisfactory. Higher doses of opioids may be required in opioid-tolerant patients.

Post-operative care

- Appropriate post-operative plans must be made. At the end of surgery, patients should be normothermic (core temperature >36.5°C), normovolaemic, have a haematocrit >27% (Hb>90g/L).
- Stable patients may be returned to vascular surgical ward after a period of observation in the recovery ward. Others with co-existing medical conditions (e.g. HF) may require a period of Level II (HDU) care. If patients have undergone prolonged surgery under GA or cardiovascular instability has occurred, a period of post-operative ventilation in a Level III (ICU) facility may be required.

- All patients should be pain-free; parenteral opioids (morphine 5–10mg IV) may be required in the early post-operative period. PCA morphine may be useful, especially in opioid-tolerant patients. Ileus is not prolonged and conversion to oral medication can occur within 24h. Oxygen should be administered for at least 24h after surgery, and 'high risk' patients should be monitored and supplementary oxygen administered for a further 2–3 days.

Further reading

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Tovey G, Thompson JP. Anaesthesia for peripheral vascular surgery. *Cont Educ Anaesth Crit Care Pain*, 2005; **5**(3): 89–92.

Anaesthesia for endoscopic thoracic sympathectomy

- *Procedure:* sympathetic chain accessed via thoracoscope; single or double ports used. Lung on operated side collapsed using OLV via double lumen tube (DLT), or by slow insufflation of CO_2 through the thoracoscope. Sympathetic chain divided by surgical resection, laser or electrocautery, or application of clips.
- *Time:* 10–15min per side.
- *Pain:* moderate; retrosternal chest pain common. IV opioids required during and after surgery, supplemented with LA infiltration and NSAIDs.
- *Position:* supine head up with arms abducted, or lateral position with a pillow under ipsilateral shoulder.
- *Blood loss:* usually minimal, but large vessel damage may rarely occur. Large cannula and G&S required.
- *Hospital stay:* 1–3 days.
- *Practical aspects:* one lung ventilation with a DLT or conventional ventilation using standard tracheal tube. Minimize pressures with CO_2 insufflation as tension capnothorax may occur. Residual pulmonary atelectasis may cause short-lived post-operative hypoxaemia even in healthy young patients. Lungs must be fully expanded under direct vision before trocar and thoracoscope withdrawn.

Open cervical or thoracic cord ganglionectomy was originally performed for Raynaud's disease and acrocyanosis. Early surgical approaches (transthoracic, transaxillary, supraclavicular, and dorsal) were gross and mutilating and associated with a stormy convalescence. Endoscopic thoracic sympathectomy (ETS) was first described in 1942 in a series of 4 cases (2 cases for the treatment of hypertension, one for the relief of pain in the stump of an amputated arm and one for Raynaud's disease).

Indications for ETS

- The main indication for ETS is idiopathic palmar hyperhidrosis, which affects 0.6–1.0% of the population and causes excessive sweating,

disproportionate for thermoregulation. It typically affects the palms, axillae, and feet.
- Other indications for ETS include craniofacial hyperhidrosis, facial blushing, chronic regional pain syndromes, ischaemic upper limb syndromes, angina pectoris, and congenital long QT syndrome. It is rarely performed now for Raynaud's disease, because relief is temporary

Sympathetic nervous system anatomy

- Two paravertebral sympathetic chains arise from efferent fibres leaving the ventrolateral grey matter of the spinal cord between T1 and L2.
- The system comprises short pre-ganglionic cholinergic fibres to ganglia in the sympathetic trunks and longer post-ganglionic adrenergic fibres to effector sites (cholinergic to sweat glands). Special fibres pass through the chain without synapsing to supply the adrenal glands.
- Above T1, trunk ascends to supply cervical ganglia. The lower cervical and upper thoracic ganglia usually fuse to form the stellate ganglion.
- Anatomy of the chains is extremely variable and cross-connections are frequently found (e.g. the accessory fibres of Kuntz).
- Sympathetic innervation to the head and neck is mainly derived from segments T1–2, the upper limb is supplied by T2–5, and the thoracic viscera are supplied by T1–4.
- In the upper thorax, the sympathetic chain runs beneath the parietal pleura, over the neck of the ribs, close to the costo-vertebral junctions. The first rib is usually crossed by the subclavian artery, but is frequently invisible from inside the chest. The second rib is often the uppermost rib seen on thoracoscopy.

Surgical techniques for ETS

- Successful ETS depends on selective interruption of sympathetic outflow.
- Techniques used include resection, electrical coagulation, laser coagulation, and the application of removable clips. Clipping may offer the possibility of reversibility.
- The extent of sympathectomy depends on the indications for surgery and varies widely between centres. Typically the sympathetic chain is divided at T2–3 for palmar hyperhidrosis, T2–4 for axillary hyperhidrosis and T2–4/5 for treatment of angina and long QT syndrome. Some operators dissect the pleura up to 5cm lateral to the chain to ensure all accessory sympathetic fibres (nerve of Kuntz) are destroyed.
- Modern surgical techniques limit the extent of the sympathectomy in an attempt to minimize the occurrence of compensatory hyperhydrosis (excessive sweating in non sympathectomized areas).
- Most ETS procedures are performed with the patient lying supine in the reverse Trendelenberg position, with the arms abducted. A support is sometimes placed under the shoulder on the operated side. The prone and lateral positions have also been described.
- Both single port (using an operative thoracoscope) and multiple port techniques have been described. The Trocar needle may be placed in the 2nd, 3rd 4th or 5th intercostal spaces in the anterior axillary line

Anaesthesia for ETS

The surgeon needs to be able to visualize the upper thoracic sympathetic chain as it runs over the neck of the ribs. This usually requires GA, although successful ETS has been performed using thoracic epidural anaesthesia or intercostal blockade.

Preoperative assessment

- Most patients are young and healthy with minimal or no co-morbidity.
- Patients should be warned about post-operative pain and the rare. possibility of conversion to an open procedure.
- Blood group should be ascertained and a sample saved.

Anaesthetic technique

One lung ventilation using a double lumen tube

- The first detailed description of anaesthesia for ETS recommended the use of a balanced general anaesthetic and a DLT.
- If a DLT is used, it is imperative to check the position with a fibre optic bronchoscope. (see 🕮 Lung isolation techniques and one lung ventilation, p. 266).
- Some surgeons use an insufflating needle (Verres needle) to create a pneumothorax in order visualize the thoracic sympathetic chain. If a gas insufflation technique is used, the operative side of the DLT must be opened to air before needle insertion.
- However, with good lung isolation, gas insufflation is not usually necessary.
- Many surgeons now consider the use of a Verres needle unsafe because of the risk of needle trauma to the underlying lung. The alternative is to open the pleura with dissecting scissors to allow the lung to fall away from the chest wall reducing the danger of lung trauma.
- Small volumes of carbon dioxide (<1L) may be introduced via the bronchoscope, if required, to help collapse the lung.

Two lung ventilation

- In many centres DLT has been replaced by standard endotracheal intubation and two-lung ventilation.
- When using two-lung ventilation, continuous insufflation of CO_2 into the thorax is necessary to visualize the sympathetic chain. Insufflation pressure limits should be set at 5–10mmHg and flow rates minimized to prevent excess CO_2 administration causing a tension capnothorax, which may be associated with a catastrophic fall in cardiac output.
- Alternative techniques include controlled ventilation via a ProSeal™ laryngeal mask airway (LMA) or single-lumen tubes and a bronchial blocker.
- A large bore IV cannula is mandatory as catastrophic bleeding has been reported.

Intraoperative period

- Prepare and drape the patient so that an open thoracotomy can be performed if necessary.
- Take meticulous care with patient positioning. Brachial plexus injuries have been reported so shoulder abduction should be limited to <90°.

- The procedure typically takes 10–15min per side: it may be performed unilaterally as two separate procedures, but the majority of patients now undergo simultaneous bilateral ETS. The right side is usually operated on first as the proximity of the sympathetic chain to the hemiazygos vein can make the operation difficult, particularly if an extensive sympathectomy to T4 is planned.
- One-lung ventilation via a DLT often results in a pulmonary shunt of up to 35%. The combination of a large shunt and impaired hypoxic pulmonary vasoconstriction in patients with healthy lungs can cause marked hypoxia. This is more marked during bilateral ETS because of residual atelectasis, particularly when operating on the second side.
- Measures to attenuate hypoxia are:
  - Increasing the FiO_2.
  - Applying continuous positive airways pressure (CPAP) of 5–10cmH_2O to the collapsed lung via a simple reservoir bag/adjustable pressure limiting (APL) valve arrangement (CPAP system, Mallinckrodt) supplied with oxygen from an auxiliary flowmeter or cylinder.
  - Maintain normotension using increments of ephedrine 3–6mg
  - If hypoxia persists, it may be necessary to inflate the collapsed lung intermittently, in co-ordination with the surgeon.
- Hypoxia is less common with the use of a tracheal tube. Shunts of 10–15% have been reported during partial collapse of the operated lung. FiO_2 >0.4 should be used to prevent hypoxia.
- At the end of surgery, collapsed lung should be re-inflated under direct vision by manual ventilation of the lungs with the pressure relief valve set at 30cmH_2O. Once full lung re-inflation is confirmed visually, the trocar and telescope are removed, while positive pressure is applied. This both expels most of the intra-thoracic gas and recruits collapsed lung units.
- The surgeon should instil LA under direct vision onto the cut edges of the pleura, the electrical coagulation sites, the port sites, and skin. The patient may be placed head-down for a short period to ensure the LA reaches the intrathoracic sites. Absorption from the pleura is rapid (peak plasma concentrations occur less than an hour after boluses), and attention should be paid to maximum recommended doses. 20mL levobupivacaine 0.5% is usually sufficient.
- ETS is painful and opioids (morphine 10mg IV) and NSAIDs are useful adjunctive analgesics.
- Be aware of the risk of major vascular trauma from port and instrument insertion. The equipment to perform emergency thoracotomy should be immediately available.

Post-operative period

- Patients frequently require additional increments of IV opioid analgesia in recovery. Patients often complain of central crushing chest pain for 2–4h after operation.
- Once this has been controlled, simple regular analgesia with paracetamol and a NSAID agent will usually suffice.

- A CXR is often obtained after ETS to exclude residual pneumothorax. However, a small pneumothorax (requiring no intervention) is seen so frequently that many workers have abandoned this practice and rely on clinical signs to detect a large pneumothorax.
- Some centres discharge patients who have undergone straightforward procedures on the day of surgery

Complications

- Hypoxia is the main concern and deaths have been reported from hypoxia during ETS. Of note, all the reported deaths were associated with bilateral ETS and the use of DLTs.
- Sudden cardiovascular collapse on carbon dioxide insufflation has been reported. This responded to prompt removal of gas from chest.
- Such reports highlight the importance of meticulous positioning of airway devices and vigilant monitoring of haemodynamics and oxygenation. If problems occur, prompt removal of intrathoracic gas, lung re-inflation, and resumption of two-lung ventilation may be required.

Immediate surgical complications

- Deaths from haemorrhage have been reported. This may be due to major vessel laceration with the trocar (usually the subclavian artery) or from damage to intercostal veins.
- Rarely, CPB may be repaired for repair of major vessel injury.
- Non-fatal cardiac arrest has been described during surgery in previously healthy patients; moderate cardiovascular compromise occurs frequently. Possible causes include tension effects of rapid intrathoracic gas insufflation and electrocautery-related dysrhythmias.

Post-operative complications

- The majority of patients undergoing ETS for palmar hyperhidrosis are satisfied with the outcome.
- The most frequently reported long-term problem is compensatory sweating of other body areas.

Further reading

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Thoracic outlet syndrome

- *Procedure:* varies from excision of fibrous bands to complete excision of cervical or first rib using supraclavicular or trans-axillary approach. Arterial reconstruction may be required.
- *Time:* <1h for simple excisions, 1–3h if arterial reconstruction required.
- *Pain:* moderate to severe. Strong opioids needed intra- and post-operative PCA useful. LA infiltration should be used; consider cervical plexus block and supplementary NSAID's
- *Position:* supine for supraclavicular approach; lateral with arm elevated for transaxillary approach.
- *Blood loss:* variable; can be brisk and catastrophic especially if subclavian artery is damaged during transaxillary approach. G&S required
- *Hospital stay:* 2–3 days.
- *Practical aspects:* surgical approach depends on pathology. Balanced GA required. Superficial cervical plexus block provides cutaneous analgesia if supraclavicular approach used. Apical pleura frequently breached. Post-operative CXR required to rule out significant pneumothorax.

The thoracic outlet consists of two spaces. In the anterior space, the subclavian vein runs between the anterior scalene and subclavius muscles, and the medial end of the first rib and clavicle (Fig. 9.5). The posterior space contains the brachial plexus and subclavian artery running between the anterior and middle scalene muscles. Thoracic outlet syndrome (TOS) is characterized by symptoms relating to dynamic compression of the neurovascular bundle of the upper limb as it passes between the uppermost rib and the clavicle. It is most common in females aged between 20 and 40 (female: male ratio of 4:1).

Clinical signs and symptoms may be neurological or vascular, depending on which component of neurovascular bundle is involved.

Aetiology

Skeletal and bony abnormalities

- Cervical rib, elongated C7 transverse process.
- Exostosis or tumor of the first rib or clavicle.
- Excess callus formation the first rib or clavicle following a fracture.

Soft-tissue abnormalities

- Fibrous bands.
- Congenital variations in scalene muscle anatomy:
  - Insertion variations.
  - Supernumerary muscles.

Acquired soft-tissue abnormalities

- Post-traumatic fibrous scarring.
- Post-operative scarring.
- Poor posture and weak muscular support.

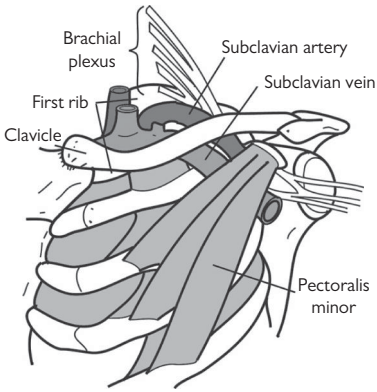

Fig. 9.5 Anatomy of the brachial plexus and subclavian vessels relevant to TOS.

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Types of thoracic outlet syndrome

Neurogenic

- Accounts for the majority of TOS cases.
- 90% of cases involve the C8 and T1 nerve roots:
  - Patients complain of pain and parasthesiae in the ulnar nerve distribution.
  - May be loss of manual dexterity with wasting of abductor pollucis brevis and to a lesser extent the muscles of the hypothenar eminence and the interosseii.
- Less frequently, the C5, C6, and C7 nerve roots are compressed causing pain referred to the neck, ear, upper chest, and outer arm.
- Diagnosis can be made by nerve conduction tests, but these may be normal.
- It is important to rule out median or ulnar nerve entrapment or radiculopathy.

Arterial

- Compression of the subclavian artery as it passes between a cervical rib or band, and scalenus anterior causes subclavian artery stenosis. Post-stenotic dilatation and aneurysm formation may be seen. There may be occlusion of the subclavian artery. Patients often have an occupational or recreational history of arm over use (e.g. painters, mechanics, weightlifters, rowers, throwing athletes).
- Presenting symptoms are those of brachial ischaemia or a pulastile mass in the supraclavicular fossa.
- Diagnosis is best made by arteriography, Duplex scan, or CT scan.

Venous (Paget–Schroetter syndrome)

- 'Effort thrombosis' of the axillo-subclavian vein causing swelling, aching, and venous congestion of the arm, classically after strenuous upper body exertion. Approximately 10% of patients may develop pulmonary emboli.
- Individuals may notice the development of prominent veins over the chest and shoulder area on the symptomatic side.
- Repetitive compression of the subclavian vein causes venous stasis and intimal damage, which further stimulates the coagulation system, eventually leading to thrombosis.
- Diagnosis is made by venography often with the extremity placed in the 'surrender' position

Treatment of thoracic outlet syndrome

Neurogenic

- Vigorous attempts are made to treat neurogenic TOS conservatively. Physiotherapy is used to improve posture and relax the scalene muscles.
- Multimodal analgesic techniques are useful.
- Surgical exploration of the brachial plexus is indicated if conservative measures fail and if abnormal anatomical features are identified.

Arterial

- Mild postural ischaemia is treated with physiotherapy.
- Initial treatment of complicated arterial TOS is focused on revascularisation to treat acute brachial ischaemia.
- Thrombolyis or thromboembolectomy may be indicated.
- Arterial reconstruction may be required to treat lesions that are often a combination of occlusive and aneurysmal in nature.
- Some patients are candidates for surgical decompression.

Venous

- The treatment of venous TOS depends on age and future aspirations of the patient.
- Younger sportsmen and women, and those who are manual workers, are offered a package of thrombolysis and surgery.
- Following successful lysis, patient is placed on heparin until transaxillary first rib resection can be performed on the next available list.
- Transaxillary rib resection is followed by venography and balloon venoplasty 2–3 weeks post-operatively to maintain vein patency.

Surgical procedure

- Surgical procedures for TOS are varied. They vary from excision of bony and soft tissue abnormalities to complex vascular reconstructions.
- Some patients require excision of the first rib to relieve neurovascular compression. There are two surgical approaches to the first rib supraclavicular and transaxillary. Uncomplicated arterial and venous TOS cases are approached via the transaxillary route.
- It is possible to remove a cervical rib using a transaxillary route, but the first rib must be removed initially to gain safe access.

- Complicated arterial cases (usually associated with aneurysm formation) and neurological TOS with cervical rib/band merit a supraclavicular approach.

Anaesthetic considerations

Preoperative assessment

The majority of patients are young and have minimal co-morbidity. A group and save should be performed.

Anaesthetic technique

- The patient is placed supine for a supraclavicular approach. A roll may be placed between the scapulae.
- If a transaxillary approach is planned the patient is positioned in the lateral position, operative side uppermost. Surgical access is more challenging with this approach the arm has to be elevated upwards to open up enough space in the axilla to access the first rib.
- Large bore venous access is mandatory. Surgical dissection is around the subclavian vessels. Catastrophic haemorrhage has been reported. Air embolus is a possibility.
- A high dose opioid, low dose hypnotic balanced GA required.
- A nerve stimulator may be used in complex neurological cases. Muscle relaxants should be avoided.
- Invasive monitoring is rarely required
- Superficial cervical plexus block provides useful cutaneous analgesia if the supraclavicular approach is used.
- Although the apical pleura is frequently breached with first rib resection perioperative hypoxia is rarely a problem.
- Avoidance of nitrous oxide is sensible.
- Local anaesthetic wound infiltration should be used.

Post-operative period

- Patients frequently require additional increments of IV opioid analgesia in recovery. Patient-controlled analgesia and simple regular analgesia with paracetamol and a NSAID agent should be prescribed.
- Prescribe supplementary oxygen if required.
- An erect CXR should be obtained before the patient leaves the recovery ward to exclude significant residual pneumothorax. Formal underwater seal drainage is rarely required. Pay particular attention to the costophrenic angle on the operative side. Blunting may indicate the possibility of intrathoracic blood accumulation, which is easy to underestimate.
- Pay close attention to the vital signs post-operatively. Although a drain is frequently employed post-operative blood loss may not be revealed. A significant haemothorax may insidiously accumulate as shed blood tends to pass through the breach in the apical pleura, rather accumulating in the drain.
- Occasionally, patients may have to return to theatre for video-assisted removal of a large intrathoracic blood clot.
- Formal thoracotomy to control severe bleeding has been reported, usually when the subclavian artery has been damaged during surgical dissection.

- Most patients are able to go home 2–3 days post-operatively.
- Patients are advised to maintain shoulder and cervical spine mobility, but to avoid aggressive physiotherapy or repetitive movement until seen for follow-up.

Anaesthesia for renal vascular access and fistula formation

- *Procedure:* surgical creation of arteriovenous fistula on the forearm.
- *Time:* 1–2h.
- *Pain:* minimal; oral analgesia required.
- *Position:* supine; operative arm abducted on an arm board.
- *Blood loss:* minimal.
- *Hospital stay:* 1–2 days.
- *Practical aspects:* patients usually have multiple co-morbidities, and have incipient or established renal failure. May have undergone recent dialysis; check post-dialysis electrolytes. Operation commonly performed under LA or RA. US-guided supraclavicular or axillary block frequently used.

- It is estimated that there are 150 000 patients in the UK with end-stage renal disease (ESRD) and 22000 requiring renal support.
- Currently almost 7000 new patients are commencing renal dialysis each year.
- The most common causes of ESRD are diabetes (30%), hypertension (24%), and glomerulonephritis (17%).
- These patients require creation of an A–V fistula/graft or the insertion of a peritoneal dialysis catheter. 77% of dialysis dependent patients in the UK receive haemodialysis.

Anaesthesia for patients with ESRD

- Patients with ESRD have a mortality 6.5–7.5 times greater than the general population. 56% of patients with ESRD have associated co-morbidity. Diabetes mellitus and ischaemic heart disease are the most common co-morbidities, observed in 33% and 21% of patients respectively. 11% of patients have cerebrovascular disease. 13% of patients hae an associated malignancy. 13% are smokers and 7.5% have COPD. The 5 year survival for patients on dialysis is ~ 40%.

Pre-operative assessment

- ESRD is a multisystem disorder with microvascular perfusion abnormalities leading to reduced capillary density and resulting in widespread organ dysfunction. A careful and systematic organ-based assessment is mandatory.
- The majority of temporary central venous haemodialysis catheters are inserted by renal physicians under local anaesthesia, but in 5–10% general anaesthesia is requested.

- Fistula formation may be performed as an elective procedure for patients with an impending need for dialysis in the following 3–6 months or urgently in patients admitted with an acute deterioration on a background of chronic renal disease.

The principle aims of anaesthesia for fistula formation

- Maximize limb perfusion by sympathetic blockade.
- Minimize myocardial stress.
- Maintain cerebral perfusion, during and after surgery.
- Ensure intraoperative patient comfort.

Pre-operative assessment

A number of features are common in patients with ESRD (Box 9.8).

Preoperative assessment focus

- *Assessment of metabolic status:* urea, creatinine, and electrolyte, and acid-base status should be checked. In addition, some patients will already be receiving dialysis by other routes (temporary dialysis catheter or peritoneal dialysis). The effectiveness of dialysis in clearing other toxins is quantified by dialysis dose. This is calculated as the volume of blood cleared during a treatment (Kt/V) [where Kt is dialyser clearance in mL/min multiplied by time on dialysis and V is patients total body water (i.e. body weight multiplied by 0.6)]. The minimum is 1.2 with a target of 1.4. The time of last dialysis should be checked.
- *Optimizing blood pressure and heart failure treatment:* medication often needs to be added or omitting depending on the fluid status on the day of surgery. Look to avoid diastolic hypotension, which is a significant predictor of poor cardiovascular outcome and for BHS/NICE BP targets, with minimum diastolic pressure of 60mmHg
- *Intravascular volume assessment:* this is best done by comparing the patient's dry weight (euvolaemic state) with their current weight and asking about the patients daily fluid allowance.
- *Blood glucose control:* 33% are diabetic and others will benefit from control of hyperglycaemia induced by surgical stress. A target blood

Box 9.8 Principle clinical features of patients with ESRD

- Chronic anaemia.
- Platelet dysfunction.
- Immune suppression.
- Hypertension.
- Accelerated vascular and cardiac calcification.
- Impaired systolic and diastolic ventricular function.
- Sudden death caused by arrhythmias.
- Unpredictable intravascular volume.
- GI dysfunction delayed gastric emptying and increased acid production.
- Metabolic acidosis.
- Hypocalcaemia, hyperkalaemia, hypomagnesaemia.
- Autonomic and peripheral neuropathy.
- Cognitive impairment.

glucose concentration of 6–10mmol/L has been suggested by American Diabetic Association. Control of blood glucose is important to reduce the incidence of infection (6–10%); this is a significant cause of fistula failure.

- *Cardiac risk should be assessed carefully:* including the patients functional capacity, and appropriate investigations performed (📖 Evaluation of the vascular surgical patient).
- *Serum K+ should ideally below normal:* patients with ESRD have large fluctuations in the plasma potassium, but not all tolerate a high level so assess for signs of cardiac toxicity on the ECG.
- *Patients with ESRD usually have 2° hyperparathyroidism:* serum calcium and phosphate should be checked before surgery to ensure muscle strength is not compromised. If well outside normal range, PTH levels should be measured to assess the possible need for parathyroidectomy before fistula formation.
- *Anaemia:* most patients with ESRD are chronically anaemic. A target Hb should be within a range to maintain blood viscosity at a level where NO is released to preserve capillary density, but keeping the advantages of anaemia in terms of improved flow. The value of this is unknown, but a Hb concentration of 100g/L has been suggested as a minimum if the patient is undergoing surgery. For A–V fistula formation a Hb concentration of >80g/L is probably sufficient.

Preoperative preparation

- Arterial pressure control is important to reduce the risk of perioperative stroke but the threshold at which risks increase substantially is not well defined. (see 📖 Blood pressure management, p. 391).
- Hypertension also increases the risks of perioperative myocardial ischaemia and arrhythmias.
- A reasonable consensus is that arterial pressure should be <150mmHg systolic and <85mmHg diastolic before non-urgent fistula formation.
- Established antihypertensive medication should be continued through the perioperative period; except if the patient's BP is below their target BP. In this case β-blockade should probably be maintained and other classes omitted to achieve the target pressure.
- ACE inhibitors or ARBs may also be omitted on the morning of surgery, although the evidence for this is conflicting.

Surgical procedure

The primary success rate of fistulae is variable with several series reporting patency rates as low as 40%. Hence, many patients need repeat procedures. Most common reasons for fistula failure are: intimal hyperplasia at the outlet of the fistula, vascular steal, aneurysm formation and infection.

The British Renal Association recommendations for renal access procedures in order of preference are:

- Formation of an A–V fistula in the upper limb using radial (first choice) or brachial artery.
- Transposition of the basilic vein and brachial basilic graft (2-stage procedure).
- Formation of a polytetrafluoroethylene (PTFE) straight or loop arteriovenous graft

- Tunnelled internal jugular CVC (📖 Central venous catheterization).
- Tunnelled subclavian CVC. These have a much greater incidence of thrombosis which jeopardizes future fistula formation in that limb by restricting the flow achieved and so are used as a last resort.

Goals of anaesthesia

The major risks of anaesthesia for patients with ESRD are cardiovascular complications (myocardial ischaemia or MI, arrhythmias, left ventricular failure, and stroke). The anaesthetist must maintain both cerebral and myocardial O_2 supply and minimize demand; GA, LA or RA techniques can be used. The goals of anaesthesia are to:

- *Maintain oxygen delivery to the brain:* CBF should be maintained by assuring an adequate arterial pressure.
- *Maintain airway control:* slow gastric emptying and hyperacidity are common in end stage renal failure (ESRF) patients so a careful history is necessary to choose between endotracheal tube and supraglottic airway.
- *Avoid tachycardia:* patients with autonomic neuropathy (including diabetic-induced nephropathy) are prone to tachycardia and potential myocardial ischaemia. Care must be used when antimuscarinic drugs are used at reversal of neuromuscular blockade.
- *Maintain body temperature:* the patient should be kept normothermic to maintain venodilatation and maximize limb blood flow.
- *Control pain:* the sympathetic response to pain causes peripheral vasoconstriction.

Selection of anaesthetic technique

Insertion of a haemodialysis catheter

Usually performed under LA. Indications for GA are:

- If the anatomy of the thoracic inlet is abnormal and/or there is known thrombosis of the central veins.
- Patients refuse further attempts at insertion of access under LA.

Insertion of a peritoneal dialysis catheter

These are also usually inserted under LA. Indications for GA are:

- Patient had a laparotomy or an infected peritoneal dialysis (PD) line.
- Presence of a known AAA.
- Patient refuses LA.

Fistula formation

Fistula formation may be performed using GA, LA, or RA (with or without adjunctive sedative drugs).

- No studies have demonstrated any difference in mortality or morbidity between these techniques. RA using US has been shown to be safe and effective, and potentially is the optimum technique, but no well-powered comparative studies have been published.
- Technique depends on local preference. Some centres perform all primary fistulas with LA only and no involvement of the anaesthetic team.
- Approximately 25% of fistula procedures overall will require the involvement of an anaesthetist.

Local anaesthesia

- LA can be performed by surgical infiltration of the site of surgery. This is easily achieved for radial A–V fistulas and is possible for brachiocephalic fistulas. It is not easily achieved for transposition grafts or PTFE loop A–V anastomosis grafts, which require a more extensive surgical exposure.
- LA infiltration of the surgical field avoids the complications of GA and causes least disturbance to patient physiology. May be preferred when patient has severe co-morbidities. However, risks of intravascular injection and haematoma are increased, and analgesia can be insufficient for some patients. May be associated severe vasospasm and provide no motor block, which may be advantageous to the surgeon. Many patients refuse a second procedure under LA.

Regional anaesthesia

Advantages of regional anaesthesia

- It avoids the cardiovascular effects of GA and mechanical ventilation patients with autonomic neuropathy.
- It allows early verbal reporting of angina symptoms and detection of myocardial ischaemia.
- It allows assessment of the effects of hypoglycaemia on cerebral function and disruption to the patient's usual drug regime.
- Regional sympathetic blockade produces arterial and venous dilatation, increasing intra- and post-operative blood flow. It gives excellent surgical conditions.
- Provides good post-operative analgesia, especially in patients with chronic pain syndromes.

Disadvantages of regional anaesthesia

- Uraemia increases the stimulation threshold of peripheral nerves making accurate location of plexus more difficult without the use of US.
- Onset of an adequate block may be delayed in patients with metabolic acidaemia and low serum bicarbonate concentrations. The duration of the block is also reduced due to lower plasma protein binding.
- Top up blocks are often required.
- Patients have the expectation they will receive GA for their surgery so may refuse RA if given a choice.
- The general complications of brachial plexus block (see 🕮 Specific regional blocks, p. 284) are more common in renal access patients

General anaesthesia

Fistula formation may be performed under a balanced GA. The choice of agents is informed by consideration of the pharmacokinetic profile of the agents available.

Advantages of general anaesthesia

The major advantages of GA are:

- Patient comfort.
- Artificial ventilation can be used to maintain oxygenation and control arterial pCO_2.

- The sympathetic nervous system response induced by patient anxiety can be obtunded.

Practical considerations

- The duration of surgery is usually 45–90min, but in repeat salvage procedures and transposition can be 180min.
- The choice of anaesthetic agents are determined largely by the by the degree of renal metabolism and excretion. Unless there are other anaesthetic considerations, induction of anaesthesia is usually achieved using judicious doses of propofol or etomidate titrated to effect.
- Beware that the time to onset of effect of IV anaesthetics is increased in patients with ESRD.
- Hypotension is common after induction of anaesthesia, regardless of the apparent intravascular volume. This should be anticipated: drugs to treat hypotension should be prepared before induction of anaesthesia and be immediately available (Table 9.1).
- The airway should be secured using a tracheal tube or Proseal laryngeal mask airway. IPPV is necessary to maintain normal values of $PaCO_2$
- Patients with ESRF have reduced plasma cholinesterase so prolonged block with mivacurium is possible.
- Suxamethonium is safe to use as long as the plasma potassium is within normal range.
- Atracurium or cis-atracurium are neuromuscular blocking drugs of choice as they are not significantly excreted by kidneys. Rocuronium is an alternative, but it is recommended that sugammadex is available.
- Anaesthesia is maintained with a volatile agent in an oxygen/air mixture, or TIVA with propofol; this may increase need for vasopressors.
- BIS monitoring may be used to help titrate the depth of anaesthesia and minimize hypotension.
- Volatile anaesthetic agents with low blood/gas solubility (isoflurane, sevoflurane, desflurane) should be used. These allow rapid emergence from anaesthesia. Desflurane may give better haemodynamic stability and faster emergence than the other agents.
- Analgesic requirements for fistula formation are modest. Moderate doses of opioids, e.g. fentanyl 1.5–3μg/kg are often used during surgery. IV paracetamol 1g with or without oral morphine 5–7.5mg 4-hourly as required are usually sufficient for postoperative analgesia, especially if combined with local anaesthetic wound infiltration (using 10–20mL levobupivacaine 0.25–0.5%).
- Tramadol, pethidine, and codeine should be avoided because active or toxic metabolites normally excreted by the kidneys may accumulate.
- Morphine-6 glucuronide will also accumulate so doses of morphine should be reduced and the dosing interval increased if it is used.
- NSAIDs are best avoided especially if there is any residual renal function; they may be useful for their opioid-sparing effect in selected patients **but only** after discussion with the renal physicians. Patients with renal disease have accelerated atherosclerosis. Many patients will be

taking aspirin as protection against the thromboembolic complications of cardiovascular disease.
- The volume of IV fluids administered should usually be limited as patients are oliguric or anuric, and there is a risk of fluid overload. Fluid replacement depends on preoperative circulating volume and cardiovascular responses to anaesthesia.
- Blood loss is usually minimal, but replacement of blood loss may occasionally be required. Rarely, significant intravascular deficits may need to be replaced with several litres of fluid. The facilities and schedule for postoperative dialysis should be checked.
- Isotonic low molecular weight colloids or Hartmann's solution can be used.
- Patient temperature should be maintained at >36.5°C to minimize peripheral vasoconstriction.
- Patients are immunosuppressed so prophylactic antibiotics should be given at induction of anaesthesia.

Post-operative considerations

- Patients need close observation; the best place is a specialist renal ward under the care of renal physicians. Careful fluid balance is required to prevent pulmonary oedema and hypotension.
- Ideally, the patients usual medication should be recommenced as soon as possible after anaesthesia; this is facilitated by regional anaesthesia.
- Pain control can be difficult especially in patients with neuropathy and regional pain syndromes secondary to diabetes and uraemia. A PCA using fentanyl or reduced dose morphine is suitable in such cases.

Further reading

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Long-term vascular access

Cannulae in peripheral veins last only a few days and there is increasing usage of long-term venous access devices in and out of hospitals. These are inserted by anaesthetists, vascular surgeons, radiologists, and specialist nurses. Definition of long-term access is not standard, but includes 'predicted use >6 weeks or the presence of internal anchoring devices'.

Choice of device

Consider:
- Indication and duration of the proposed therapy needing venous access.
- The proposed location for administration of therapy (hospital, GP/clinic, home).

- The risks of contamination.
- The patient's clinical status (coagulation, platelets, sepsis, cardiovascular stability).

Indications

These include:
- Cancer chemotherapy.
- Long-term antibiotics.
- Total parenteral nutrition (TPN; home or hospital).
- Haemodialysis or haemofiltration.
- Repeated blood transfusions.
- Repeated venesection.

Available devices

The original design of Hickman type catheters have been copied and adapted over the years. Totally in-dwelling venous access devices (TIVAD) have injection ports with a silicone membrane surrounded by a titanium or plastic case. These are surgically implanted beneath the patients' skin, usually on the chest wall or arm. These port systems are more expensive and more difficult to insert, but they do allow patients to undertake greater physical activity and have a lower risk of infection.

The ideal catheter is:
- *Flexible and soft:* stiff catheters increases the potential to damage vessel walls.
- Chemically inert.
- Non-thrombogenic.
- Radio opaque.

Available catheters

Short-term
- Peripheral cannulae.
- Midlines (10–20cm soft catheter) inserted via the antecubital fossa, with the tip in the upper 1/3rd of the basilic/cephalic vein, short of great veins.
- Non-cuffed, non-tunnelled CVCs are used for resuscitation/CVP monitoring. They are rarely used for >10–14 days, due to the risks of infection. Antimicrobial coated CVC's are available. Avoid insertion of multiple lumen catheters if only a single lumen is required.

Long-term
- *Peripherally-inserted central catheters (PICCs):* advanced centrally from an upper arm vein. They can last several months if managed correctly. Limitations of PICCs include:
  - Catheter tip may be difficult to manipulate and optimal tip positioning may be difficult. Malpositioning of the catheter may lead to complications, such as thrombosis, which increases from 21% if placed within the superior vena cava (SVC), to 60% if placed in the axillary, subclavian, or innominate veins.

  - Arm movement may cause movement of the catheter tip, increasing the risk of cardiac arrhythmias and increasing the risk of thrombosis due to endothelial damage.
  - The narrow lumen of PICCs limits the maximum flow rate.
  - A small percentage of these catheters are removed because of early failure, phlebitis, or occlusion.
- *Dialysis catheters:* there are a number of long-term double lumen dialysis catheters available, with cuffs to anchor the catheter. It is common to avoid the use of the subclavian vein to prevent subsequent subclavian thrombosis or narrowing which might then complicate future arteriovenous fistula formation.
- *Cuffed, 'Hickman type' catheters:* tunnelled from insertion site to chest/abdominal wall. They are open-ended or contain a two-way valve (e.g. Groshong catheter). A Dacron cuff develops tissue ingrowth for anchorage and acts as a barrier to infection (takes 3–4 weeks).
- *Subcutaneous ports (titanium/plastic, single/double lumen):* placed in a surgical cavity on the chest/abdominal wall. Ideal for intermittent therapy, e.g. antibiotics for cystic fibrosis, and popular for children. These are a sealed system between needle access episodes so the risk of infection is lower.

See Table 9.2 for a summary.

Site of access

- Anecdotal evidence suggests that right sided catheters have lower risk of thrombosis. They have a shorter straighter route to the SVC, with easier tip positioning.
- Choose the site considering patient factors, previous venous access, and clinician experience.
- Look for evidence of vein thrombosis, previous scars, venous collaterals (may suggest great vein stenosis). Use ultrasound to assess patency at access sites. Formal central vein imaging (venogram, CT, MRI) is helpful in difficult cases.
- Choose puncture sites and tunnel tract to avoid tight bends, if necessary use multiple punctures to avoid >90° bends and kinks.

Table 9.2 Long-term catheters and typical duration of use

Device	Normal Duration
Peripheral cannulae	48–72 h
Midlines	14–21 d
Non-cuffed, non-tunnelled CVC	5–14 d
Tunnelled non cuffed CVC	5–21 d
Peripherally Inserted CVC (PICC)	Several months
Tunnelled, cuffed, CVC (Hickman)	Months/yrs
Subcutaneous ports	Months/yrs

See Table 9.3 for a summary of the advantages and disadvantages of different access sites.

Tips for insertion

- Ask the patient to inspire on insertion of the guidewire and catheter to help central passage.
- Measure catheter length required from positioned guidewire (using image intensifier).
- Take care with rigid sheaths/dilators to avoid central vein damage. Do not insert sheaths/dilators too deeply (generally longer than required).
- Pinch sheath (or use valve) on removal of obturator to avoid bleeding and air embolism
- Sheaths readily kink; draw back until catheter passes.
- Pass long thin Terumo guidewire (70cm +) via an initial or final catheter to help central placement in difficult cases.
- Screen the guidewire, dilators, and/or sheath and catheter insertion if any resistance is encountered.
- Use venography through needle, sheath, or catheter if uncertain as to device position relative to vein path (check contrast allergy).
- For fixed length catheters (e.g. Groschong/dialysis) choose correct length for site of access and adjust length of tunnel to give correct tip position. Move the anchor cuff along tract to adjust length.

Ports

- Insert under LA +/– sedation or GA. Smaller low profile versions can be sited in the arm.
- Minimize incision and pocket size by placing anchor sutures in the pocket first, then slide port in and tie off.
- Access port with specific 'non-coring' needle via thick silicone membrane; with a distinct clunk as needle hits the back wall.

Positioning of a CVC

- Correct catheter tip positioning is important to reduce the incidence of complications, particularly thrombosis (linked to infection), embolism, arrhythmias perforation into the pericardium, pleura or mediastinum, and the risk of extravasation. This is described in 🕮 Central venous catheterization.

Complications of long-term CVCs

In addition to the complications of short-term CVC insertion (🕮 Central venous catheterization), long-term CVC increases the risks of:

- *Pinch off*: entrapment of subclavian catheters between the clavicle and first rib, this may lead to the catheter breakage.
- *Thrombosis:* this is a recurrent problem associated with long-term venous access.
- Venous clot/air embolism.
- Lumen blockage.
- Infection.
- Catheter movement leading to cardiac arrhythmias or extravasation injury.

Table 9.3 Advantages and disadvantages of different access site

Site	Advantages	Disadvantages
Upper arm (cephalic/basilic)	Simple to access: veins usually visible and palpable at elbow Use US for more proximal puncture sites Less vital structures nearby Patient comfort	Failure to achieve central position Higher incidence of thrombosis Low maximum infusion rates
Right internal jugular	Simple to insert. Direct route to central veins. High flow rate: low risk of thrombosis Lower risk of pneumothorax Ideal for larger stiff catheters, e.g. dialysis	Patient discomfort Possible higher risk of infection? Tunnelling more difficult to chest wall Cosmetic considerations
Axillary/ subclavian	Less patient discomfort. Lower risk of infection?	Curved insertion route Central catheter misplacement Difficult to access Increased risk of pneumothorax, haemothorax, nerve damage Catheter may be damaged between clavicle and first rib (pinch off)
Femoral	Easy insertion Tunnel to mid-abdomen	Higher rate of infection/ thrombosis More difficult in obese patients More discomfort Less mobility

Aftercare

- All staff using catheters must have adequate training and use maximum sterile precautions.
- Do not remove external anchoring sutures/devices for at least 3 weeks to allow a Dacron cuff to adhere.
- PICCs have no anchoring cuff; need continued external anchor device.
- Adhesive external anchor devices (e.g. Statlock) are thought to have lower risk of infection than sutured devices.
- If there is a high risk of thromboembolism, therapeutic dose anticoagulation may be indicated.
- Some units still lock dialysis catheters with heparin 1000U/mL; always aspirate the catheter deadspace if in doubt before use.

- Thrombosed/blocked catheters may be unblocked with urokinase (5000U). Either flush with deadspace volume if flushing is possible. If blocked repeatedly suction all air through 3-way tap to collapse catheter and create vacuum. Then instill urokinase diluted in 2mL saline.

Removing a Hickman line

- Anchored catheters need a cut down to free the cuff and avoid line snapping on traction.
- Cuffed catheters usually pull out if in <3 weeks, before adhesions anchor cuff.
- Heavily infected catheters may pull out as infection breaks down adhesions.
- Push and pull catheter to palpate cuff, it may be difficult to feel cuff if just inside exit site.
- Inject generous LA around cuff site and tunnel tract.
- Cut down (1.5cm incision) on venous side of cuff. Use forceps to feel catheter rolling beneath (incision too small for finger).
- Free up and remove the venous section first, a thin fibrin sheath will need to be incised.
- Then dissect around cuff to free adhesions and remove completely.
- Avoid sharp dissection until the venous section is removed to avoid catheter embolization (catheters pass to right ventricle (RV)/ pulmonary artery (PA)).
- Similar principles apply to removing ports, which lie in a fibrous sleeve with non-absorbable anchor sutures.

Further reading

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Venous surgery including endovenous laser treatment

- *Procedure:* unilateral/bilateral saphenofemoral/popliteal ligation +/– multiple avulsions.
- *Time:* 1–2h.
- *Pain:* mild to moderate. Simple analgesics plus local anaesthetic infiltration of wounds.
- *Position:* supine with head down tilt for saphenofemoral ligation. Prone if saphenopoliteal ligation required.
- *Blood loss:* minimal.
- *Hospital stay:* day case/overnight stay.
- *Practical aspects:* spontaneous respiration via LMA if supine. Tracheal intubation and artificial ventilation required if prone position necessary.

Varicose veins

Although a small number of venous surgical procedures are for reconstructive surgery, e.g. venous aneurysm repair, the vast majority are for the treatment of varicose veins caused by chronic venous insufficiency.

Indications for varicose vein surgery

- Venous ulceration.
- Oedema.
- Bleeding.
- Skin changes.
- Symptoms such as itching or pain.
- Cosmesis.

Surgery for varicose veins

Surgery for varicose veins generally involves exposure of saphenofemoral junction in the groin, or the sapheno-popliteal junction in the popliteal fossa. The junction is tied off and the long or short saphenous vein is stripped. Small superficial varicosities are then treated by multiple stab avulsions. This is usually as a day case procedure. The majority of patients are relatively young and fit, although a high body mass index (BMI) and smoking are common associations. Most patients require GA, although spinal anaesthesia is occasionally indicated or requested by the patient.

Conduct of anaesthesia

Early discharge is facilitated by providing rapid recovery, good pain control and avoiding post-operative nausea and vomiting (PONV).

- *Induction with a short-acting strong opioid:* such as fentanyl at 1–2micrograms/kg and propofol is suitable. Alfentanil may not provide adequate early post-operative analgesia. Spontaneous ventilation via LMA is usually appropriate, although necessity of the Trendelenberg position to reduce venous bleeding increases the risk of aspiration of gastric contents and exacerbates poor respiratory function in some (e.g. the obese). In this situation, intubation and positive pressure ventilation using a short-acting neuromuscular blocking agent is advised. Some surgeons prefer patient to be prone when exposing saphenopoliteal junction. Intubation and ventilation is mandatory in these cases.
- *Maintenance:* provided by a volatile agent or continuous infusion of propofol. Most single leg procedures last less than 1h. The addition of N_2O provides additional analgesia and promotes recovery due to its hypnotic sparing properties. The modest increase in PONV with N_2O is largely offset by the anti-emetic effects of propofol. In the authors' experience, the incidence of PONV with this technique is low, particularly when combined with an anti-emetic such as a 5-HT_3 receptor antagonist. Many of the patients in this group have multiple risk factors for PONV; routine prophylaxis is justified.
- *Multimodal post-operative analgesia:* is used, avoiding strong opioids. Post-operative pain is generally mild; a combination of paracetamol with a NSAID agent and a weak opioid is effective. The surgeon should infiltrate the groin or popliteal wound with LA. The common peroneal nerve is quite superficial behind the knee. Patients should be warned of the rare possibility of a temporary foot drop if undergoing short saphenous vein surgery.

Non-surgical management of varicose veins

Although surgery remains main treatment for large varicose veins, there are other treatment modalities, which avoid the need for GA. These include:

- *Injection sclerotherapy:* chemical sclerosants are injected directly into smaller varicosities causing scarring and occlusion of the vessels.
- *Foam injection sclerotherapy:* larger varicosities of the long or short saphenous veins require foaming of the sclerosant with CO_2 or O_2.
- *Endovenous laser treatment (EVLT):* a laser probe is manipulated into the varicose vein and, when activated, causes local heating and thrombosis.
- *Radiofrequency ablation (RFA):* a radiofrequency probe used, rather than a laser fibre.
- These procedures are generally performed as an outpatient under LA.
- Some use sedation or a light GA, particularly if multiple avulsions of distal varicosities are planned.
- During EVLT and RFA procedures a laser or radiofrequency probe is placed in the lumen of the varicose vein distally using US and its tip manipulated close to the saphenofemoral junction or saphenopopliteal junction under US guidance.
- Local heating of subcutaneous tissues along the length of the vein is painful and can potentially cause thermal damage to overlying skin. Tumescent anaesthesia with large volumes of very dilute LA is used. The fluid injected acts as heat sink to minimize the chance of thermal damage to skin and the LA provides post-operative analgesia.
- A high degree of skill in the use of US is required to ensure correct positioning of the laser fibre, RFA probe, or sclerosant within the varicose vein.
- Side effects of EVLT and RFA include minor local burns and nerve damage. Proximal spread of sclerosant is a concern, especially with foam sclerotherapy, potentially causing DVT, pulmonary embolism (PE), and rarely stroke.

Further reading

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Lower limb amputation

- *Procedure:* above (AKA) or below knee (BKA) amputations. BKA associated with better long-term function.
- *Time:* 45min to 2h.
- *Pain:* moderate/severe. Many patients receiving long term opioids for rest pain and may be opioid tolerant. PCA is useful if patient capable. Larger bolus dose and shortened lockout interval may be required if opiate tolerant.
- *Position:* supine.
- *Blood loss:* usually <500mL. G&S.
- *Hospital stay:* variable—depends on post-operative mobility, social, and other factors.
- *Practical aspects:* regional (spinal, CSE, or epidural) or balanced GA. RA contraindicated if systemic anticoagulation. Patients usually elderly,

frail with multiple co-morbidities. Femoral (AKA) lumbar plexus (AKA) and/or sciatic nerve blocks (AKA and BKA) as single shot or catheter techniques useful for post-operative analgesia.

- The rate of lower limb amputation (LLA) has remained relatively constant over the past decade. Approximately 5000 patients per annum are referred to UK prosthetics services.
- In approximately 80% of patients, the indication for LLA is vascular occlusive disease. Diabetes is present concurrently in up to 50% of patients; the incidence of amputation is eight times higher in the diabetic population compared with non-diabetics.
- Patients are usually elderly with a high incidence of associated co-morbidity in particular:
  - IHD—symptoms may be masked due to limited mobility secondary to claudication or rest pain.
  - Cerebrovascular disease.
  - Renal impairment.
  - Smoking-related lung disease (COPD)—if severe associated with a 3-fold increase in pulmonary complications.
- Morbidity and mortality following LLA is high—the 30-day mortality rate is as high as 17%. AKA has a higher mortality rate than BKA— 30-day mortality rates are 10–17% and 5–10%, respectively.
- LLA has significant associated morbidity, e.g. chest infection, poor wound healing, wound infection, poor mobility, and persistent post-operative pain. The higher the level of amputation, the harder it is to use a prosthesis. Mobilizing using a prosthesis requires considerable additional energy expenditure (~60% for BKA, ~ 120% for AKA). The high incidence of co-existing disease and the inactive lifestyle associated with the progression of PAD mean that a large number of elderly amputees never manage a prosthesis.
- LLA's are frequently given low clinical priority; many operations are performed on emergency lists out of hours. Patients presenting for LLA should be recognized as high risk. Care of patients undergoing LLA requires experience, care and attention to detail, and should not be delegated to the junior member of the team.
- Close communication is essential between the anaesthetic and vascular surgical teams with senior surgical and anaesthetic involvement to ensure appropriate preoperative investigations and to plan the timing of surgery and post-operative care.

Pre-operative investigations

Routine

- *Full blood count:* anaemia may require correction. The Hb concentration should be maintained >9g/dL. An elevated white cell count (WCC) should mandate a septic screen. Infection in the ischaemic limb may be the cause, but the possibility of a chest or urinary infection should be excluded.
- *U&E:* electrolyte abnormalities should be corrected. Renal impairment is common. Appropriate IV fluids should be administered to

avoid dehydration and to correct electrolyte abnormalities (see 📖 Evaluation of the vascular surgical patient, p. 85, 📖 Perioperative renal protection, p. 236).
- *Coagulation screen:* coagulopathy related to anticoagulant therapy or sepsis must be corrected. The INR should be <1.5 and the platelet count >50 000 prior to surgery.
- *Blood sugar levels and glycosylated haemoglobin level:* patients for amputation frequently have evidence of poor glycaemic control. Avoidance of persistent hyperglycaemia is important. Ideally, blood sugar should be maintained <10mmol/L. Involvement of the hospital diabetic team may be prudent
- *ECG:* to provide a baseline and identify a recent acute coronary event or a significant arrhythmia that may require intervention.
- *CXR:* indicated in patients with abnormal chest signs on physical examination. Chest infection should be treated with oxygen, physiotherapy, and antibiotics.

Further investigations

- A murmur or signs of ventricular failure should prompt consideration of an echocardiogram to look for significant valvular heart disease or significant ventricular dysfunction.
- In the presence of significant pulmonary disease arterial blood gases and LFTs may be helpful. An FEV_1 < 70% of the predicted value or an FEV_1/FVC ratio of < 65% indicates a high risk of pulmonary complications.

Pharmacological optimization

Statins

Long-term statin administration reduces cardiovascular events in the vascular surgical population by improving endothelial function, reducing vascular inflammation, and stabilizing atherosclerotic plaques (see 📖 Minimizing perioperative risk, p. 123). Acute statin cessation in the perioperative period is associated with an increased risk of adverse cardiac events; statin therapy must be continued. Although there is no evidence to support acute statin administration to reduce vascular risk prior to urgent surgery, most vascular surgical patients benefit from stain therapy as secondary prevention against the thromboembolic complications of cardiovascular disease. Commencing statin therapy would seem sensible.

β-blockade

- Acute β-blockade is associated with increased perioperative mortality and increased risk of stroke. Acute β-blockade may occasionally be indicated in extremely high-risk patients presenting for LLA who demonstrate inducible ischaemia on pharmacological stress testing. If instituted β-blockade should be very carefully titrated against the heart rate (e.g. low dose bisoprolol titrated to a resting hart rate of 50–80beats/min).
- Chronic β-blockade should be continued. β blockers have anti-inflammatory properties, which stabilize coronary plaques and contribute to their cardioprotective properties when taken long term.
- All patients presenting for LLA should receive antiplatelet medication (aspirin or clopidogrel if aspirin intolerant).

Pain control

- Many patients presenting for LLA are taking large doses of opiates for severe ischaemic pain. Preoperative pain intensity has been identified as a significant predictor of phantom limb pain (PLP) post-operatively. Good pain control, as well as modulating pain pathways, may help ameliorate the sympathetic stress response and aid perioperative cardiovascular stability. Involvement of the pain management service is recommended.
- Use a multimodal analgesic regime, combining simple analgesics with opioids. Include agents effective against neuropathic pain (e.g. amitryptiline or gabapentin). PCA may be required.

Perioperative management

- Aim to maintain cardiovascular stability, normovolaemia, normothermia, and avoid anaemia.
- Consider the use of a tourniquet to reduce intraoperative blood loss.
- Aim to provide good analgesia into the post-operative period.
- Consider invasive monitoring in those with severe cardiovascular disease or those who are acutely unstable due to sepsis.
- Administer appropriate antibiotics according to local guidelines, prior to the start of surgery.
- Consider RA, which offers the theoretical benefits of attenuation of the stress response to surgery, improved respiratory function post-operatively and a reduction in early post-operative cognitive dysfunction. Reduction in early post-operative delirium may have important advantages in compliance with medical therapy, functional recovery, and length of hospital stay. There is no evidence that RA, even if utilized pre-emptively, reduces the incidence of PLP.
- Central neuraxial anaesthesia is contraindicated when there are systemic manifestations of sepsis, or if the patient is anti-coagulated.
- GA may be employed. Controlled or spontaneous ventilation is appropriate. Cardiovascular stability is important. GA may be supplemented by peripheral nerve blocks (Sciatic nerve block for BKA, combined sciatic and femoral block for AKA). Catheter techniques may be employed using dilute infusions of local anaesthetic to prolong the block duration. LA infusions via surgically placed sciatic nerve catheters have been shown to improve pain relief and reduce opiate requirements in the immediate post-operative period.

Post-operative management

- Prescribe venous thromboembolism prophylaxis according to local guidelines.
- Administer supplemental oxygen for 72h post-operatively.
- Utilize high-dependency facilities for very high risk patients.
- Refer promptly to the local amputation rehabilitation team for early mobilization and physiotherapy.
- Aggressively institute secondary prevention measures to reduce the likelihood of loss of the second limb.

Further reading

Melsom H, Danjoux G. Perioperative care for lower limb amputation in vascular disease. *Cont Educ Anaesth Crit Care Pain* 2011; **11**: 162–6.

Uncommon vascular procedures

- *Procedure:* Removal of retroperitoneal sarcoma.
- *Time:* variable 3–5h.
- *Pain:* severe. Tumours frequently large necessitating large incisions. Thoracic epidural recommended.
- *Position:* dependent on location of tumour. Upper abdominal sarcomas may require the lateral position. Lower abdominal sarcomas usually supine.
- *Blood loss:* variable. Consider intraoperative cell salvage. G&S required.
- *Hospital stay:* 7–10 days.
- *Practical aspects:* Tumours can be massive and very vascular. Careful preoperative planning required. Large bore peripheral and central venous access essential. Invasive arterial pressure monitoring mandatory. Non-invasive cardiac output monitoring provides useful information. Post-operative HDU/ICU care may be required.

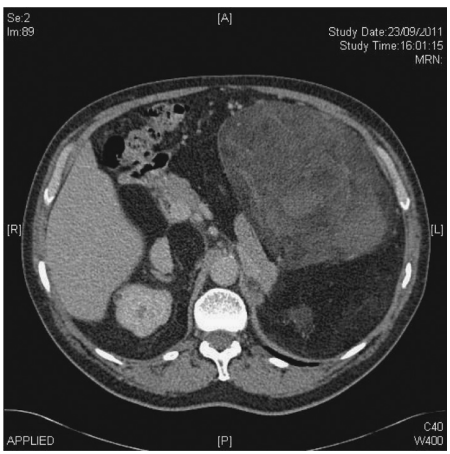

Fig. 9.6 Large L sided sarcoma displacing the abdominal contents to the right

Retroperitoneal sarcomas

- Sarcomas are rare tumours; they account for around 1% of all cancer cases.
- ~10–20% of all sarcomas occur in the retroperitoneum. Retroperitoneal tumours may occur anywhere in the retroperitoneum and can be difficult to access surgically.
- They can occur at any age, but the peak incidence is in the 5th decade.
- The most common histological types are liposarcomas and leiomyosarcomas.
- Patients present late; the retroperitoneal space is a large potential space and the tumours are slow growing. Symptoms such as abdominal pain

and fullness are non-specific and easily dismissed as being caused by other less sinister disease processes. Hence, retroperitoneal sarcomas are frequently very large at presentation.

- The definitive treatment of primary retroperitoneal sarcomas is surgical resection. Local recurrence and disease specific survival are dependent on complete resection and the histological grade of the tumour. 5-yr disease specific survival rates of 50–60% have been reported.
- A multidisciplinary team meeting is an ideal forum to plan definitive surgery.
- Primary resection may involve removal of adjacent organs in over 50% of cases. The kidney, colon, spleen, and distal pancreas were the organs most commonly removed in one large UK series.

Surgical technique

- Surgery may be challenging because of the close relationship of the tumour to multiple vital structures within the anatomically complex retroperitoneal space.
- Detailed imaging is required to define the location and size of the tumour, its relationship to adjacent organs and vascular structures, as well as normal anatomic variants and anomalies of the major abdominal arteries and veins. CT scanning is most commonly used (Fig. 9.6).
- The type of surgical incision is dictated by the site of the tumour. A thoraco-abdominal incision with the patient in the lateral position may be required for sarcomas in the upper quadrants of the abdomen. Midline incisions usually provide adequate surgical access for sarcomas arising in the lower abdomen and pelvis.
- Meticulous surgical technique is required. It is vital to operate in avascular planes and ligate major feeding vessels pre-emptively.
- Laser/ultrasonic scalpels and water jet dissectors are invaluable aids to minimize blood loss.

Anaesthetic technique

- A balanced GA is required. Large tumour removal requires large surgical incisions. Unless contraindicated, an epidural should be used to provide high quality post-operative analgesia.
- A rapid sequence induction may be required if gastric emptying is impaired by the presence of an upper abdominal sarcoma.
- NGT is often needed, especially if stomach or small bowel resection is planned.
- Massive sarcomas may cause compression of the vena cava. Caput medusae may be seen on clinical examination in extreme cases. A lateral tilt may be advisable if a midline surgical approach is planned.
- Large bore venous access and invasive arterial pressure monitoring are mandatory. Most anaesthetists will insert a central venous line.
- Non-invasive cardiac output measurement devices provide useful information about the status of the cardiovascular system.
- Surgery may be lengthy and blood loss may be significant. Vigorous attempts must be made to prevent hypothermia. Intraoperative cell salvage should be used. Near patient testing with the Hemocue and rotational thromboelastometry will provide useful information to guide blood and blood product replacement.

- Patients who are warm, and cardiovascularly stable may be extubated and managed in an HDU environment. In more unstable patients, a short period of post-operative ventilation may be indicated.
- The possibility of concealed post-operative haemorrhage into the retroperitoneum must be considered in unstable patients who have an unexplained fall in post-operative haemoglobin concentrations.
- Re-exploration for bleeding may be necessary, particularly following massive sarcoma removal due to the presence of very large raw areas following massive tumour removal.

Vascular malformations

- Vascular malformations are composed of abnormal vascular channels lined with a single layer of dysplastic endothelium. Although vascular malformations are present at birth, they may not become clinically evident until later in life. Unlike haemangiomas they do not regress spontaneously. They may occur in the liver, spleen, GI tract, brain, and lungs.
- Bleeding is the most common presentation.
- CT and MRI are the best modalities to image vascular malformations.
- Radio-labelled red cell nuclear scintography can be used to localize the site of active bleeding in patients with gastrointestinal haemorrhage.
- Definitive treatment is most commonly performed by interventional radiologists using vessel occluders, coils, and liquid embolic systems (e.g. Onyx™).
- GA frequently required as patients are frequently restless and in pain.

Arteriovenous malformations

- Arteriovenous malformations (AVM) occur when there is an abnormal communication between the arterial system and the venous system without an intervening capillary bed. The site at which abnormally dilated feeding arteries and veins communicate is called the nidus. Such lesions typically are associated with increased blood flow and may result in a steal phenomenon.
- An aortocaval fistula is the most common AVM encountered by the vascular anaesthetist. It commonly arises from an enlarging atherosclerotic aorta. It may be caused by penetrating abdominal trauma and iatrogenic trauma at lumbar disc surgery. Rare causes include mycotic aneurysm, syphilis and connective tissue disorders (e.g. Ehlers–Danlos or Marfan's syndrome). It is said to occur in approximately 1% of all operations for AAA and in 4% of operations for ruptured AAA.
- The presence of low back pain, a palpable AAA, an abdominal 'machinery' murmur and high-output cardiac failure unresponsive to medical treatment should raise clinical suspicion. Hepatomegaly, ascites, haematuria, haematochezia, scrotal oedema, priapism, pulsating varicose veins, and lower extremity oedema have been reported in long-standing cases
- The diagnosis is made on CT scan by early detection of contrast in a dilated inferior vena cava (IVC) adjacent to an aortic aneurysm and loss of the normal anatomic space between the aorta and the IVC.

- Surgery in a patient with an aortocaval fistula requires careful planning. Successful endovascular repair has been reported when time permits and the anatomy is favourable. Open repair is more common and is usually associated with massive blood loss. Arterial control is with aortic clamps. Venous bleeding may be controlled with balloon-tipped Fogarty catheters, inserted through the fistula into the proximal and distal IVC.
- Great care must be taken not to dislodge atheromatous material from within the aneurysm sac across the fistula; atheromatous PE has been reported.
- The incidence of DVT may be as high as 20%. Thromboprophylaxis is mandatory.

Chapter 10

Emergencies in vascular surgery

Emergency abdominal aortic aneurysm repair: open and endovascular repair

- *Procedure:* open ruptured AAA repair performed via midline or transverse abdominal incision. The spectrum of presentation is variable, ranging from a contained leak with relative haemodynamic stability to free intraperitoneal rupture when the patient may be in extremis. In the latter scenario the life-saving manoeuvre is application of an aortic cross-clamp-**time is of the essence**. Once bleeding is controlled and haemodynamic stability restored, surgical technique is analogous to open AAA repair.
- *Time:* variable 3–6h.
- *Pain:* severe. Most patients will be ventilated for several days on ICU. On opiate infusion will be part of the sedative regime. This may be replaced by PCA once the patient is extubated.
- *Position:* supine, arms positioned on arm boards to allow access to arterial and venous catheters.
- *Blood loss:* variable may be massive, typically several litres. Intraoperative cell salvage should be used. Cross-match (XM) 10U. Activate massive haemorrhage protocol—large volumes of blood and blood products will be required.
- *Hospital stay:* 7 days—several weeks.
- *Practical aspects:* at least 2 senior anaesthetists required. When haemodynamic stability permits insert arterial and central venous catheters, an NGT, urinary catheter, and temperature probe. Balanced GA technique with tracheal intubation and IPPV. Attempt to maintain normothermia; active warming needed. Anticipate need for vasoactive drugs. Transfer to ICU for post-operative IPPV.

Background

Ruptured abdominal aortic aneurysm (RAAA) is responsible for ~8000 deaths per annum in the UK. Just over half of patients do not reach hospital alive and in-hospital mortality of remainder is in region of 60% (overall mortality >80%). Of those who survive, many will go on to enjoy a good quality of life, but some are left with residual organ dysfunction (e.g. cardiac failure, renal impairment/failure, limb loss). The aim of the UK National Health Service AAA Screening Programme is to reduce the number of deaths from aneurysm rupture by detecting asymptomatic AAA's and permitting elective repair, which is associated with lower mortality.

Rupture of an abdominal aortic aneurysm is a true surgical emergency leading to certain death without operative intervention. Once a diagnosis of ruptured aortic aneurysm is made the patient should be transferred to the nearest hospital with emergency vascular surgery services unless a senior clinician makes the decision to offer palliative care. Mortality following rupture has changed little over the past few decades. The risk of rupture increases with increasing size of aneurysm. The estimated annual rupture rate of aneurysms larger than 6cm in diameter is 9%, rising to more than 25% for aneurysms larger than 8cm in diameter. The 5-yr survival of patients managed conservatively with aneurysms greater than 5.5cm is approximately 20%.

Presentation

Aortic rupture is most common in males aged over 65 and the clinical presentation usually includes:

- Lower abdominal pain (can be sudden onset or worsening over a few days).
- Lumbar back pain.
- Presence of a tender pulsatile mass in abdomen.
- Varying degrees of haemorrhagic shock and organ dysfunction.

The diagnosis may be made clinically and with portable US imaging. If any doubt exists and patient is haemodynamically stable then a contrast enhanced CT scan should be performed without delay. 3-D reconstruction will allow an assessment of the anatomical suitability for emergency EVAR (eEVAR).

Aneurysms may rupture either into the retroperitoneal space or into the peritoneal cavity. Free intraperitoneal rupture is associated with a poorer outcome because of the rapid development of haemorrhagic shock. Retroperitoneal bleeds often tamponade and stabilize for a short period. Occasionally, an aneurysm may rupture into duodenum (aorto-enteric fistula), due to adhesion of the duodenum to the inflammatory aneurysm sack. Erosion of an aneurysm into the inferior vena cava (aorto-caval fistula) is unusual and has varying presentations including a continuous abdominal murmur.

Preoperative evaluation

A brief targeted assessment should be made to permit an informed decision on patient suitability for emergency surgical repair. The advent of EVAR has perhaps allowed more patients with severe cardio-respiratory disease to be considered for emergency intervention.

History should include an assessment of functional capacity and documentation of the presence of co-morbidities which may include;

- Cardiac disease and/or respiratory disease.
- Chronic kidney disease.
- Cerebrovascular disease (history of CVA, TIA, dementia).
- DM.

Allergies and current medications should be noted.
Examination should focus on:

- Airway assessment.
- Conscious level (GCS, AVPU).
- Signs of haemorrhagic shock.
- BP in both arms (take the higher one).
- Evidence of cardiac ischaemia.

Decision to operate

This should be made by a senior surgeon and anaesthetist. Survival depends upon the extent and duration of shock, the clinical sequelae of sustained tissue hypoxia and the pre-morbid state. A common sense approach should be taken when surgery is futile with no realistic chance of a successful outcome. The 5-point Hardman index (Table 10.1) may be used to aid identification of those patients with the least likelihood of survival. It was originally

Table 10.1 Hardman Index

	Points
Age >76	1
Serum creatinine >190mmol/L	1
Hb >9g/dL	1
Myocardial ischaemia on ECG	1
A history of LOC after arrival to hospital	1

A score of > 2 is associated with a mortality of >80%.

Reprinted from Journal of Vascular Surgery, 23, 1, Hardman DTA, et al., 'Ruptured abdominal aortic aneurysms: Who should be offered surgery?', pp. 123–129, Copyright 1996, with permission from Society for Vascular Surgery and Elsevier.

stated that the presence of three Hardman variables was associated with 100% mortality. It is important to remember that this scoring system was developed 15 years ago after analysis of outcomes in patients undergoing open surgery. More recent studies have predicted a mortality of 80% with a Hardman index of 2 or more. Scoring systems have their limitations and should only be used to supplement clinical judgement. Patients who have previously been deemed unsuitable for elective surgery should not automatically be declined emergency surgery. The risk-benefit balance will now have swung towards intervention, and if the patient is not moribund it is often thought appropriate to attempt an emergency repair preferably using an endovascular stent graft (eEVAR). With the advent of endovascular surgery, the range of patients suitable for urgent vascular intervention has increased.

Preoperative investigation and preparation

Transfer to theatre should not be delayed except when there is clinical doubt over diagnosis that could considerably alter the management. If haemodynamically stable, the patient should undergo contrast enhanced CT scan to assess the suitability for eEVAR or if the diagnosis is in doubt. A ruptured AAA is best managed by 2 experienced anaesthetists.

The anaesthetist assessing the patient should also ensure:

- IV access with at least 2 large bore (14G) cannulae.
- Send blood samples for:
  - FBC, renal profile, PT, APTT, fibrinogen, Ca^{2+}, lactate and cross match.
  - Order:
  - 10U packed red cells.
  - 4–8U FFP.
  - 1–2 adult therapeutic doses of platelets (may need more if on clopidogrel/aspirin).
- Consider need for cryoprecipitate (if fibrinogen <2g/L).
- 12-lead ECG to exclude cardiac ischaemia.

Institute invasive arterial monitoring where possible (radial or brachial) and measure an ABG. If the patient is anti-coagulated with warfarin, correct INR with either prothrombin complex concentrate (PCC) or IV vitamin K (depending upon local policy) as soon as possible.

A second anaesthetist should prepare theatre and ensure that key personnel are informed of imminent arrival of the patient. They should warn

the blood transfusion laboratory of the potential activation of the massive transfusion protocol to ensure adequate supplies of blood and coagulation products.

Essential equipment required in theatre

- O –ve blood (while waiting for group specific/cross-matched blood).
- Cell salvage machine and operator.
- Rapid infusion device.
- Fluid warming devices.
- Forced warm air devices/patient warming mattress. The lower body should not be warmed, whilst the cross-clamp is in place as limb ischaemia may be exacerbated.
- Cardiac output monitoring device(s).
- POC testing devices, e.g. arterial blood gas analysis, Hemocue, rotational thromboelastometry.

Fluid management prior to surgery and hypotensive haemostasis

A restrictive resuscitation policy prior to surgical control (cross-clamp or balloon on a guidewire) is associated with a better outcome. It is important to maintain organ function, but not to restore a normal BP. Administration of large volumes of fluid is thought to dislodge thrombus and dilute already depleted clotting factors, causing further bleeding. Red cell transfusion should be avoided unless the patient is unconscious or there is clinically significant myocardial ischaemia. This is more common in scenarios when systolic BP is less than 70mmHg.

Analgesia

Analgesia prior to induction should be carefully titrated. The elderly patient with haemorrhagic shock and organ dysfunction is in a very precarious situation, and even small doses of opiates may precipitate cardiovascular collapse (loss of sympathetic drive). However, severe pain will cause hyperventilation and increased O_2 consumption and is inhumane. Thoracic epidural catheters have little place in the management of the emergency RAAA, even those with a contained retroperitoneal haematoma often develop significant coagulopathy and insertion of an epidural may delay definitive surgical treatment. Many patients remain on mechanical ventilation for a number of days after surgery and may not benefit from epidural analgesia. However, a patient with a tender aneurysm with no evidence of a leak on imaging, who undergoes urgent surgery can be managed in the same way as an elective case.

Anaesthesia for open repair

Induction

- Place the patient on the operating table with warming mattress/forced air warmer.
- Ensure the surgeon is in theatre, scrubbed, and the abdomen prepared before induction.
- Establish large bore IV access and connect to a rapid infusion device.
- Ensure packed red cells in theatre and checked (cross-matched, group specific or O –ve).

- Ideally, establish invasive arterial BP monitoring (and blood gas monitoring).
- Prepare the intraoperative cell salvage device.

There is no specific anaesthetic induction agent or technique that has demonstrated improvement in outcome. Many avoid N_2O due to its deleterious effects upon the sympathetic nervous system, myocardial contractility and its association with bowel distension.

- Rapid sequence induction: modified to allow careful titration of the dose of induction agent. (Large proportion of cardiac output is diverted to CNS during haemorrhagic shock.)
- Co-induction with opioid/benzodiazepine will reduce required dose of induction agent.

Be prepared to manage profound hypotension, hypertension, tachycardia, bradycardia, and cardiac arrest.

Prior to aortic clamping

- Heparin is seldom given.
- Be prepared to start a rapid infusion of fluid (balanced crystalloid and colloid) and to administer vasocontrictors to maintain BP to near pre-induction value until aortic cross-clamp is applied.
- Insert CVC (measure $ScVO_2$).
- Insert NGT and temperature probe.
- Commence cardiac output monitoring. Many have moved away from the pulmonary artery floatation catheter favouring devices that use the principle of pulse waveform analysis or the oDM. The oDM does not provide reliable information when the aorta is cross-clamped, but may provide useful haemodynamic information before and after cross-clamp release.

Aortic cross-clamp

Infra-renal clamp usually leads to temporary increase in afterload +/– hypertension.

Physiological effects of the cross-clamp depend upon:

- Level of clamp.
- Myocardial contractility.
- Circulating volume status at the time the clamp is applied.
- Duration of cross-clamping.
- Hypertension is rare during RAAA repair but can be managed by:
  - Increasing the depth of anaesthesia.
  - High dose opioid, e.g. fentanyl up to 20microgram/kg.
  - Vasodilators (e.g. GTN infusion; 1–5mg/h).

Once cross-clamp is applied and control of bleeding confirmed, aggressive resuscitation of the patient is appropriate.

General haematological/biochemical goals after cross-clamp include:

- Hb 80–100g/L.
- Platelets $>100 \times 10^9$/L.
- PT and APTT ratio <1.5.
- Fibrinogen > 2g/L.

- Calcium > 1mmol/L.
- Magnesium > 0.7mmol/L.
- pH > 7.35.

Increase minute ventilation and reduce $PaCO_2$ to 4.5kPa in anticipation of profound acidosis after clamp release.

Cross-clamp release

May lead to a significant fall in BP.

There should be close liaison between surgeon and anaesthetist when the cross-clamp is released. The anaesthetist should be confident that patient is well filled (e.g. CVP >10, FTc >350). The iliac clamps should be removed slowly, one at a time. Reclamping may be necessary if hypotension is profound.

Common causes of hypotension include;

- Sudden reduction in afterload.
- Relative hypovolaemia.
- Ischaemia-reperfusion injury.
- Myocardial dysfunction secondary to the negative inotropic effects of lactic acidosis and hyperkalaemia, secondary to anaerobic metabolism in the ischaemic lower limbs.
- Pulmonary hypertension and right ventricular dysfunction secondary to pulmonary microemboli.
- Further bleeding from a leaking anastomosis or generalized ooze caused by a dilutional coagulopathy.
- The degree of hypotension is related to:
  - Severity of shock prior to clamping.
  - Duration of cross-clamp time.
  - Cardiovascular co-morbidity.
  - Hypovolaemia.
  - Speed of clamp release.

If hypotension is profound, ask surgeon to reapply iliac clamps and attempt to further resuscitate patient with fluids, inotropic agents, or vasoconstrictors as appropriate. It may be necessary to increase minute ventilation to aid correction of the metabolic acidosis associated with cross-clamp release. IV $NaCO_3$ may occasionally be used.

Transfusion and coagulopathy

The lethal triad of coagulopathy, acidosis and hypothermia is an important cause of mortality in RAAA. Management should consist of:

1. Rapid control of haemorrhage
2. Maintenance of normothermia
3. Prompt and aggressive treatment of coagulopathy

Point of care (POC) testing if available is very helpful as the haemostatic picture is changing rapidly and laboratory based results may lag behind the clinical picture. Development of a dilutional coagulopathy is common. Early proactive administration of FFP and platelets may improve outcome. Packed red cells, FFP and platelets should be infused in the ratio 1:1:1. One adult therapeutic dose of platelets contains platelets pooled from 6 donor units of blood. Maintain fibrinogen levels > 2 g/dl. 5 units of FFP contains

the same quantity of fibrinogen as 2 pooled units of cryoprecipitate, thus early use of FFP may avoid the need for cryoprecipitate. If the fibrinogen level is <2 g/dl 2 packs of pooled cryoprecipitate should be given. 1 pack contains pooled donations from 6 donors.

Excessive fibrinolysis may occur and be identified by rotational thromboelastometry. Studies in trauma patients suggest improved outcomes with empirical administration of tranexamic acid -1gram intravenously administered over 10 minutes followed by an infusion of 1gram IV over 8 hours.

Post-operative care

- All patients should be transferred to ICU post operatively for close monitoring.
- Re-warming will be necessary and respiratory and cardiovascular support will be required, frequently for several days.
- Renal function, haemoglobin, coagulation and acid base derangements all require close monitoring. Renal replacement therapy is required in a significant proportion of patients.
- Coagulopathy, if present will require further blood product support ideally guided by POC testing.
- Post-operative ileus may be prolonged and parenteral nutrition is frequently required.
- Ischaemic colitis may occur after open RAAA repair. Persistent post-operative metabolic acidosis with a rising lactate concentration, particularly if associated with bloody diarrhoea should precipitate immediate surgical re-exploration.
- Spinal cord ischaemia is a rare but devastating complication with an increased incidence in open RAAA repair.
- Intrabdominal pressure (IAP) should be measured as patients are prone to developing abdominal hypertension (IAP >12mmHg) or abdominal compartment syndrome (IAP >20mmHg) particularly if they have required aggressive fluid resuscitation. Some patients may benefit from laparostomy and delayed abdominal wall closure.

Anaesthetic considerations for emergency EVAR

There is a move towards relaxing the strict anatomical criteria to allow greater numbers of patients to be offered eEVAR, currently between 55 and 80% of RAAA's are anatomically suitable. There are no published randomized controlled trials comparing EVAR with open repair for RAAA. Since patients require a spiral CT to assess aneurysm morphology and confirm anatomical suitability they are not usually in such a perilous state as those whose clinical picture necessitates immediate surgery. This may explain the results from small case series that suggest improved outcomes in patients undergoing eEVAR. It is important to remember that contrast-enhanced spiral CT angiography delays time from presentation to definitive treatment. Some patients become unstable in the interim—an endovascular supra-coeliac aortic balloon may be used to control haemorrhage prior to stent-graft deployment ('radiological cross-clamp'). As with open aortic cross-clamping, this is the point at which fluid resuscitation can commence more aggressively.

Endovascular interventions can be undertaken with bifurcated stent-grafts or with aorto-uni-iliac (AUI) graft deployment. AUI stent graft deployment requires subsequent open femoro-femoral cross over grafting and contralateral iliac artery occlusion necessitating general anaesthesia.. One option is to start deploy the AUI under LA (i.e. achieve interventional control of haemorrhage) and start aggressive fluid resuscitation and convert to GA for the cross over graft. GA should follow the same principles as those described for open repair. Interventional control under local anaesthesia may be challenging in an agitated hypoxic patient who may be experiencing severe abdominal pain.

Relative contra-indications to using LA as the sole technique are often present;

- CVS stability of patient may lead to agitation/hypoxia.
- High analgesia requirements/severe abdominal pain.
- Agitation/confusion/reduction in GCS causing imaging artifact.
- 2° procedures, such as embolectomy or femoral–femoral cross-over grafting may be necessary (e.g. after AUI device deployment).

Additional complications specific to EVAR include:

- Iliac artery rupture by the stent graft deployment device. Currently, available deployment devices require the iliac vessels to be at least 6mm in diameter.
- Inability to successfully isolate the aneurysm—primary endoleak.
- Contrast-induced nephropathy and AKI.

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Aortic dissection

Occurs when blood penetrates a defect in the tunica intima and enters the tunica media of the aorta. Blood at systolic pressure dissects the intima from the media, creating a false lumen, and can propagate in antero- or retrograde direction along the length of aorta from the aortic valve to the iliac bifurcation. Complete rupture of aorta can lead to rapid exsanguination and carries very high mortality rate.

Classification

Originally, there were two different anatomical classifications for aortic dissection: DeBakey and Stanford. Stanford classification is the most commonly used (Table 10.2; Fig. 10.1). Dissections are classified as acute if onset is thought to be less than 2 weeks.

More recently, a new classifiaction has been proposed by the European Society of Cardiology. This classification takes into account the aetiology of dissection or the signs of evolving dissection (Box 10.1).

Table 10.2 Classification of aortic dissection

DeBakey	Stanford
Type I: originates in ascending aorta, propagates at least to aortic arch and often beyond it distally	*Type A:* involves ascending aorta and/or aortic arch, and possibly descending aorta (DeBakey Type 1 and II)
Type II: originates in and is confined to ascending aorta	*Type B:* involves descending aorta (distal to origin of left subclavian artery)
Type III: originates in descending aorta, rarely extends proximally, but will extend distally	

Box 10.1 European Society of Cardiology Classification of Aortic Dissection

- *Class 1:* classical aortic dissection with an intimal flap between true and false lumen.
- *Class 2:* medial disruption with formation of intramural haematoma/haemorrhage.
- *Class 3:* discrete/subtle dissection without haematoma, eccentric bulge at tear site.
- *Class 4:* plaque rupture leading to aortic ulceration, penetrating aortic atherosclerotic ulcer with surrounding haematoma, usually subadventitial.
- *Class 5:* iatrogenic and traumatic dissection.

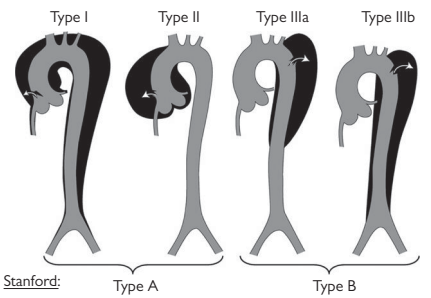

Fig. 10.1 The most common classification systems of thoracic aortic dissection: Stanford and DeBakey.

Box 10.2 Causes of aortic dissection

- Hypertension.
- *Connective tissue disorders:* Marfans Syndrome, Ehler–Danlos Syndrome, Turners' Syndrome.
- *Vasculitis:* Giant cell arteritis, Takayashu's, tertiary syphilis.
- *Congenital:* bicuspid aortic valve, aortic coarctation.
- Deceleration trauma.
- Iatrogenic (cardiac surgery or catheterization).
- Pregnancy.

Epidemiology

- Peak incidence between 50 and 70yrs.
- 65% of patients are male.
- Commonest predisposing factor is hypertension.
- Other potential causes and associations (Box 10.2).

Presentation

Chest pain is the most common presenting symptom. This is usually:

- Sudden in onset.
- Severe.
- Tearing or ripping in nature.
- In the posterior chest, back, or abdomen.

Some patients report migration of the pain, possibly due to propagation of the dissection. BP at presentation can be high, low, or normal.

- Hypertension more common with type B dissections and occurs in ~70% of cases.
- Hypotension or hypovolaemic shock more common with type A dissections and occurs in ~25% of cases.

Other presenting symptoms include:

- Syncope.
- Cerebrovascular accident.
- Congestive cardiac failure.

Initial clinical examination may reveal:

- Tachycardia and anxiety from pain and sympathetic stimulation.
- Cardiac murmur (aortic regurgitation).
- Differential or absent peripheral pulses.
- *Neurological deficit:* stroke, numbness, and weakness in lower limbs (secondary to spinal cord ischaemia).

Diagnosis

Initial presentation can be varied; a high index of clinical suspicion is required. Investigations must be used to confirm the diagnosis and exclude similarly presenting conditions.

- *Electrocardiogram:*
  - 20% of Type A dissections have ECG changes due to extension of the dissection to involve the coronary ostia.

Table 10.3 Frequency of findings on initial investigations

Electrocardiogram	Chest Radiography
Ischaemia/infarction (20%)	Widened mediastinum (62%)
Left ventricular hypertrophy (26%)	Abnormal aortic contour (50%)
Non-specific changes (41%)	Abnormal cardiac contour (26%)
No abnormalities (31%)	Pleural effusion (19%)
Displaced/calcified aorta (14%)	No abnormalities (12%)

  - It is important to rule out a primary coronary event—the treatment is very different. Further imaging is required to confirm diagnosis.
  - Bundle branch block may be due to stretching of aortic annulus.
- *Chest radiography:* the classical widened mediastinum may be absent in around one-third patients (Table 10.3).
- Aortography is established gold standard investigation, but is unsuitable in the unstable patient.
- Spiral CT scanning with contrast is relatively quick and can delineate extent of dissection and allow identification of true and false lumens.
- MRI gives high resolution images in absence of contrast, but is more time-consuming and less suitable for the unstable patient.
- TTE visualizes the ascending aorta and aortic arch well in thin individuals. TOE provides good visualization of most of the thoracic aorta. Its use is gaining in popularity.

Initial resuscitation and management

Anaesthetists are often involved in the initial management of patients with aortic dissection who may be:

- Haemodynamically unstable.
- Neurologically impaired.
- Might not yet have a confirmed diagnosis.

Early priorities are those of resuscitation and stabilization (Box 10.3) to facilitate further investigation and stabilize for transfer to a regional cardiothoracic centre. The major objective is to control BP and reduce the force of left ventricular contraction (dP/dt) to minimize shear forces on the aortic wall to reduce the risk of further propagation of the dissection or prevent complete rupture.

Ongoing management: Type A

Dissection of the ascending aorta is a surgical emergency and carries a high mortality associated with:

- Rupture and exsanguination.
- Cerebrovascular accident due to dissection of the innominate and left common carotid artery.

Type A dissections are repaired by cardiothoracic surgeons via a median sternotomy with CPB and frequently require deep hypothermic cardiac arrest (DHCA).

Box 10.3 Initial management priorities in aortic dissection

ABC assessment
- Give oxygen.
- Intubation if GCS <8 or profoundly unstable.
- Obtain wide-bore peripheral venous access.
- Send blood for cross-match, FBC, clotting, U&E, cardiac enzymes.
- Cautious fluid infusion.

Analgesia

Titrated IV morphine.

Blood pressure control
- Titrated β-blockers (esmolol or metoprolol): target systolic pressure 110–120mmHg
- Further control with sodium nitroprusside, GTN, or Ca^{2+}channel blockers.

Final management
- Confirm diagnosis with imaging studies as soon as possible.
- Transfer for definitive management.

Aims of surgery
- Prevention of rupture.
- Prevention of further propagation of the dissection.
- Repair of aortic regurgitation, either through valve replacement or repair.
- Coronary artery re-implantation or bypass grafting.
- Reperfusion of branching vessels of the aortic arch.

Principles of anaesthetic management are as for any cardiac surgery and beyond the scope of this chapter. Monitoring should include invasive arterial pressure monitoring, the left radial is preferred because of possibility of involvement of innominate artery. Continuous TOE monitoring is frequently employed.

Ongoing management: Type B

Patients with chronic or uncomplicated type B aortic dissections are usually treated medically. This involves:
- Aggressive antihypertensive therapy.
- Analgesia to attenuate autonomic tachycardia and hypertension.
- Long-term surveillance with imaging and clinical assessment to monitor any progression of the dissection.

Surgery is indicated for those with complications of type B dissection (Box 10.4). Surgical access to the descending thoracic aorta is usually via a left lateral thoracotomy. The standard anaesthetic procedure involves:
- Intubation with a double lumen tube, lung isolation, and one-lung ventilation.

Box 10.4 Indications for surgery in type B aortic dissection

- Aortic rupture.
- Ischemia of limbs and organ systems.
- Persistent or recurrent intractable pain.
- Progression of dissection.
- Aneurysm expansion.
- Uncontrolled hypertension.

- Invasive pressure monitoring is essential. A right radial arterial line is preferred if there is involvement of the left subclavian artery. A femoral arterial line is used for monitoring perfusion distal to the lesion.
- Spinal cord protection through the placement of a lumbar CSF drain should be considered. Anterior spinal artery syndrome is a devastating complication.

Endovascular intervention

Percutaneous endovascular interventions are gaining popularity, especially in stable type B dissections or in unfit patients, for whom open repair via a thoracotomy is considered inappropriate.

Principles of therapy

- Reconstruction of aortic segment containing entry tear, preventing further propagation of the dissection.
- Induction of thrombosis in the false lumen.
- Restoration and maintenance of flow in the true lumen and branching arteries.

The principles of anaesthetic management are broadly comparable with those for TEVAR; see 🕮 Open aortic aneurysm repair, p. 350.

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Acute limb ischaemia

- *Procedure:* variable, depends on the aetiology of the ischaemia. Acute limb ischaemia (AcLI) 2° to an embolus is treated by embolectomy. Thrombotic AcLI is more frequently treated with a lower limb bypass
- *Time:* variable 2–6h.
- *Pain:* variable, AcLI may be very painful, necessitating large doses of opiates
- *Position:* supine, one arm abducted on an arm board to permit access to arterial and venous catheters.
- *Blood loss:* variable, often insidious. Group and save mandatory, transfusion rarely required.
- *Hospital stay:* 7–14 days.
- *Practical aspects:* embolectomy usually performed under LA plus judicious sedation, if patient is restless or in pain. Target-controlled infusion (TCI) of remifentanil +/– TCI propofol popular. Lower limb bypass performed under central neuraxial blockade or balanced GA. Full systemic heparinization may preclude the use of central neuraxial blockade in some patients. Invasive arterial pressure monitoring may be indicated, particularly if patient has severe cardiorespiratory comorbidity. Unstable patients may benefit from a period of close monitoring in HDU.

Definition

The 2007 Trans-Atlantic Inter-Society Consensus (TASC II) guideline defines AcLI as 'a sudden decrease in limb perfusion that can threaten limb viability'.

Clinical significance

The prevalence of AcLI is around 1 per 6000 of the population per year. Approximately 5000 patients present each year with acute limb ischaemia in England and Wales. Associated mortality at 1yr is significant (~20%) as is subsequent limb loss (~35%) regardless of treatment modality. AcLI should be assessed and treated by an experienced specialist team.

Aetiology

Main causes of acute limb ischaemia

Thrombosis (60%)

Thrombosis usually secondary to pre-existing atherosclerosis compounded by acute plaque rupture or reduced flow secondary to cardiac failure or hypovolaemia. Collateral vessels are often present and flow is sluggish throughout the vascular tree.

Predisposing factors

- Hypotension.
- Unusual prolonged posture (e.g. prolonged kneeling).
- Malignancy.
- *Hypercoagulability:*
  - Protein C or S deficiency.
  - Activated Protein C resistance.

  - Antiphosholipid syndrome.
  - Hyperprothrombinaemia.
  - Factor V Leiden.
  - Antithrombin III deficiency.
- Dehydration.
- Hyperviscosity syndromes (e.g. polycythaemia).

Embolus (30%): the majority of emboli arise in the heart. Emboli are most commonly associated with atrial fibrillation or mural thrombus, complicating acute MI. They may be secondary to mechanical heart valves (particularly mitral), endocarditis, and rarely, atrial myxoma. Most emboli lodge at arterial bifurcations; the commonest site of embolic occlusion is the femoral bifurcation. Other sites include brachial, popliteal, and aortic bifurcations (saddle embolus). Embolic occlusion of brachial artery is not usually limb threatening, whereas femoral emboli usually result in significant ischaemia of the affected lower limb.

Aortic dissection: outflow to limbs is disrupted by the false lumen.

Thrombosed aneurysm (~5%): e.g. popliteal, aortoiliac, subclavian, femoral.

Trauma (~3%): limb fractures and dislocations.

Inadvertent intra-arterial drug administration.

Iatrogenic causes: include arterial cannulation, extended tourniquet use during surgery and major pelvic surgery.

Clinical presentation

AcLI presents acutely as a cold white extremity. Clinical findings are classically described by the following (six 'p's'):

- *Pain:* changes in nature and intensity over time.
- *Paraesthesia:* occurs in about 50% of patients. Varies between light touch and proprioceptive dysfunction through to reduction in deep pain and pressure sensation.
- *Paralysis:* significant feature and is often associated with severe ischaemia.
- *Pallor:* initial skin pallor may progress to mottled and cyanotic appearance over time.
- *Pulselessness:* confirmation with formal Doppler flow measurements
- *Perishingly cold:* although a common feature, temperature is the most unreliable diagnostic criteria, due to the influence of environmental ambient temperature.

Distinction between the two main aetiologies (thrombosis versus embolus) is important, since clinical management is different.

- *Clinical features suggestive of thrombosis:* include previous history of intermittent claudication, onset over hours or days, and reduced or absent peripheral pulses in the contralateral limb indicating widespread peripheral arterial atherosclerosis.
- *Clinical features suggestive of an embolus:* include acute onset of symptoms associated with an identified embolic source. Contralateral limb pulses are usually present, although emboli may affect multiple sites (~15%). Embolic sources are usually related to cardiac irregularities, including new onset atrial fibrillation or a ventricular mural thrombus.

Table 10.4 Clinical classification of acute limb ischaemia

Category	Sensation	Paralysis	Suggested treatment
I	No loss of sensation	None	Not immediately threatened. Time to investigate
IIa	Minimal loss (e.g. toe)	None	Urgent treatment need for salvage
IIb	More than toes and associated with rest pain	Partial	Immediate treatment needed for salvage
III	Profound, anaesthetic	Profound/ rigor	Irreversible: 1° amputation

Reprinted from Journal of Vascular Surgery, 26, 3, RB Rutherford et al., 'Recommended standards for reports dealing with lower extremity ischemia: Revised version', pp. 517–538, Copyright 1997, with permission from Society of Vascular Surgery and Elsevier.

Clinical classification

AcLI severity dictates the initial treatment of the patient. Determined by presence of pain, paraesthesia, or paralysis (Society for Vascular Surgery/ International Society for Cardiovascular Surgery 1997; Table 10.4).

Clinical management

- Category I patients need IV heparin and analgesia. Usually adequate time for patients to be fully investigated, both surgically and medically, and optimized prior to any surgical intervention.
- Category II patients also require heparinization and analgesia. In contrast to category I, complete acute ischaemia (category II) is medico-surgical emergency as irreversible tissue necrosis results if perfusion cannot be restored within 6h of onset. There is minimal time for investigation. Although resuscitation and preoperative optimization are important, these should not delay urgent surgical intervention. Initially, affected tissue appears marble white secondary to arterial spasm. As this spasm is unsustainable, involved arteries subsequently fill with deoxygenated blood, and skin becomes mottled and cyanosed. Blanching on pressure indicates potential for revascularization, whereas dark fixed staining of the skin indicates irreversibility, as does muscle tenderness or pain on passive movement.
- *Treatment:* embolectomy is usually the first line surgical management of complete acute ischaemia. May be followed by on-table arteriography with subsequent thrombolysis, angioplasty, stenting, or arterial bypass. Compartment syndrome secondary to reperfusion injury within the calf muscle necessitates fasciotomy in about 5% of patients who receive successful treatment for AcLI.
- Category III patients presenting with irreversible ischaemia should not be offered revascularization, but may be offered urgent amputation. This procedure should not be unduly delayed for medical optimization in order to ensure to minimize of the life-threatening systemic effects of extensive muscle necrosis in the affected limb. Entry onto a terminal care pathway is sometimes the most appropriate option in patients with extensive tissue involvement and significant co-morbidities.

Prognosis of the ischaemic limb relates to

- The extent of pre-existing collateral circulation (e.g. acute on chronic ischaemia).
- Duration and extent of ischaemia.
- Aetiology of the occlusion (thrombosis or embolus).
- Anatomical site of the occlusion.
- Patient co-morbidities (e.g. diabetes, renal failure, cardiac dysrhythmias).

Anaesthetic assessment and planning

Early communication with the vascular team is essential to ascertain whether limb salvage or amputation is planned. This decision will guide subsequent patient assessment and management. Anaesthetic considerations for limb amputation are dealt with in 📖 Lower limb amputation, p. 439.

Patient assessment for limb salvage

- Includes clinical history and examination paying particular attention to the rapidity of onset, duration of symptoms and pre-existing features of chronic arterial disease:
  - Suspicion of an embolic cause of AcLI should prompt a search for source of the embolus (e.g. a transthoracic echocardiogram to look for left atrial thrombus or an ECG to look for an acute cardiac event).
  - AcLI secondary to a thrombotic cause should prompt a search for thrombotic risk factors (e.g. inherited/acquired hypercoagulability or malignancy). Thrombotic events more common in patients with existing vascular disease; general assessment of vascular comorbidity is important.
- An estimate of the potential severity of the reperfusion injury should be made whenever revascularization is proposed.

Baseline investigations

- *Haematological:* FBC, clotting screen, group and save.
- *Biochemistry:* U&E, lipid profile, blood glucose, creatine kinase.
- *ABG:* look for metabolic acidosis.
- *Electrocardiogram:* exclude acute a myocardial event.
- An urgent echocardiogram may be indicated to look for atrial thrombus, assess ventricular performance and valvular function
- CXR.

Pre-operative optimization

- Normal resuscitative measures including fluid rehydration and oxygen therapy.
- *Intravenous heparinization:* a full anticoagulation dose is usually administered IV to avoid further deterioration of the ischaemic limb. Cessation of heparin to permit regional anaesthetic techniques must be given careful consideration.
- *Urinary catheterization:* appropriate due to lack of mobility and to monitor urine output.
- *Analgesia:* IV opioids usually required.

- Ventricular rate should be controlled if patient is in fast AF. β-blockers are the first line treatment.
- Blood sugar should be tightly controlled.

Anaesthetic techniques

Local anaesthesia

- Minimally-invasive surgical and radiological procedures, including embolectomy and endovascular stenting may be performed under LA.
- Monitored anaesthesia care recommended; patients frequently have severe comorbidity, may be restless and in pain.
- Conscious sedation using a TCI of propofol +/– TCI remifentanil can be very helpful.

Regional anaesthesia

- Use of central neuroaxial techniques in patients with acute lower limb ischaemia often contraindicated by full systemic anticoagulation.
- In contrast, peripheral nerve blocks, when performed under US guidance in experienced hands, may be appropriate for upper limb revascularization in patients with severe co-existing morbidity, even when the patient is fully heparinized.

General anaesthesia

- GA is technique of choice for the majority of limb salvage procedures.
- Drugs for induction and anaesthetic maintenance are dictated by urgency of the intervention, as well as the haemodynamic and fasting status of the patient.
- Maintenance of anaesthesia can be volatile- or propofol TCI-based, +/– supplementary opioid infusion.
- Large bore IV access should be established prior to induction.
- Direct monitoring of invasive arterial pressure is recommended.
- Cardiac output monitors (e.g. oDM, pulse contour analysis) can help guide IV fluid management.
- Serial measurement of arterial blood gases and serum K^+ is recommended as generalized reperfusion injury can result in profound acidosis and hyperkalaemia.
- Reperfusion of the ischaemic limb may produce profound vasodilation with significant hypotension. This should be treated with the judicious use of fluids and vasoactive drugs.

Post-operative care

The significant 1yr mortality rate (~20%) of patients with AcLI is predominantly related to burden of pre-existing vascular disease, especially IHD. Hospital morbidity and early mortality is dependent on combination of an acute illness and likelihood of a severe systemic reaction to limb reperfusion in patients with diminished physiological reserve.

The level of post-operative care and monitoring is determined by:

- Pre-operative status.
- Clinical events during the interventional procedure.
- Severity of reperfusion injury.

- Many patients can be managed successfully with a period of extended recovery followed by direct discharge to the ward.
- Unstable patients and those with significant comorbidity should be managed in a high dependency environment. Supplemental O_2 is required for 48–72h.
- Successful limb reperfusion following significant period of ischaemia is often associated with severe pain. Effective pain management will help prevent further catecholamine surges, which may predispose to haemodynamic and myocardial stress. Systemic opioids are usually required given the difficulty in using regional techniques. Multi-modal analgesia should be introduced as soon as possible.

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Chapter 11

Post-operative management

Post-operative monitoring of the vascular surgical patient

Introduction

High-risk patients account for 15% of inpatient surgical procedures, but over 80% of surgical deaths. Patients undergoing vascular procedures have an increased risk of perioperative complications, particularly cardiac events and are ∴ often considered high-risk. The perioperative outcome of vascular surgery may be improved by early identification of cardiac complications through appropriate monitoring, and the effective use of critical care resources.

Which patients require post-operative monitoring?

Post-operative monitoring should be targeted primarily at detecting cardiac ischaemia and ventricular impairment. 'At risk' patients will be defined by:

- Type and severity of the vascular procedure.
- Identification of significant preoperative comorbidity.
- Early recognition of acute intraoperative events.

Other monitoring will be more specific to patient requirements (e.g. renal, respiratory, or neurological monitoring in patients with relevant comorbidities; see Table 11.1).

Which post-operative cardiac monitors should be used?

An integrated approach to monitoring combines individual monitoring modalities to allow for early detection of ischaemia, ventricular function, and tissue injury. The extent of post-operative monitoring also depends on the incidence and severity of pre- and intraoperative factors identified above.

Post-operative surveillance for myocardial ischaemia

- Post-operative myocardial ischaemia is usually characterized by asymptomatic, rate-related ST depression.
- Up to 48% of episodes of perioperative ischaemia occur in the first 48–72h after surgery.
- Prevention of tachycardia is crucial to preventing myocardial ischaemia.
- Early detection of tachyarrhythmias allows aggressive treatment of other factors, which cause or worsen myocardial ischaemia including hypertension, hypotension, anaemia, and pain.
- If myocardial ischaemia is detected early, prompt intervention is indicated, including the timely use of beta blockers and the optimization of myocardial O_2 supply-demand balance.
- Prompt treatment of ischaemia reduces post-operative increases in cardiac troponin and 6-month mortality.

Options for post-operative surveillance of myocardial ischaemia include:

Computerized ECG (ST segment) monitoring

- Changes in ST segments are the most sensitive method of detecting ischaemia.

- Computerized ST segment trend analysis is better than visual interpretation of ST changes for detection of post-operative ischaemia.
- The CM5 and CB5 configurations give an early and sensitive detection of myocardial ischaemia, and should be the standard ECG configuration choice post-operatively.
- Accepted ST segment changes used to detect myocardial ischemia are: >=1mm (0.1mV) of horizontal or down sloping ST segment depression (measured 60ms from the J point). Up-sloping ST segment elevation of >=1.5mm (0.15mV) measured 80ms from the J point.
- Post-operative ST segment changes with a prolonged duration (>30min per episode or >2h cumulative length) are an independent predictor of post-operative cardiac events.

Transoesophageal echocardiography

- Transoesophageal echocardiography (TOE) provides direct visual assessment of changes in ventricular wall motion, which may be related to ischaemia.
- The transgastric, mid papillary short-axis and multiple mid-oesophageal views at TOE are best for detecting abnormal ventricular wall motion and thickening due to myocardial ischaemia.
- Changes in endocardial wall motion occur quickly in the setting of ischaemia and may occur even before ischaemic changes are seen on the ECG.

Pulmonary artery pressure monitoring

- Changes in the pulmonary arterial waveform and pulmonary arterial occlusion pressure can be sensitive indicators of ischaemia.
- Indicators of myocardial ischaemia include:
  - Prominent A and C waves.
  - Increased LVEDP.
  - Pulmonary artery occlusion pressure (PAOP).

Post-operative surveillance for ventricular function

Haemodynamic monitoring of ventricular function may be integrated within protocols to optimize O_2 delivery during and after surgery, using fluids and inotropes. This may identify patients who would benefit from additional pharmacological interventions. There are several options for post-operative monitoring of ventricular function (see 📖 Cardiac output monitoring, p. 259).

Invasive assessment of cardiac output

- The 'gold standard' of cardiac output measurement is the thermodilution method using the PAC (Stewart Hamilton algorithm).
- Pulse contour analysis and intermittent transpulmonary thermodilution gives a beat to beat measurement of cardiac output.

LiDCO is a variant of pulse contour analysis where calibration relies on an injection of lithium.

Table 11.1 Indications for post-operative monitoring

Factors	Notes
1. Preoperative factors	
Cardiac risk indices	E.g. Revised Cardiac Risk Index (Lee) >=2 factors
Poor ventricular function	Impaired resting left ventricular ejection fraction <30%
Functional assessment: • Simple functional assessment • Cardiopulmonary exercise testing • Dobutamine stress echocardiography	 MET score <4 AT <11mL/min/kg: VE/VCO_2 >43 Significant reversible wall motion abnormalities
Biomarkers BNP/NT-BNP	Elevated BNP or N-terminal pro-brain Natriuretic peptide (NT-proBNP) values ('cut-off' values for high-risk status are still debated)
Other preoperative scoring systems	(e.g. p-POSSUM; 📖 The vascular preoperative assessment clinic, p. 110)
2. Intraoperative factors	
New onset ECG changes • Ischaemic	Ischaemic ECG changes lasting more than 20min—high risk of intraoperative myocardial 'stunning' and post-operative myocardial ischaemia
• Arrhythmias	Significant arrhythmias include multiple ventricular ectopics, atrioventricular block, AF
Surgical complications	Unexpected complexity of surgery, reoperation for surgical bleeding, massive transfusion
3. Other specific indications	
Respiratory monitoring • Arterial blood gas • Oxygen saturations • Peak cough flow rate • Incentive spirometry	Patients at risk of respiratory failure: • Severe respiratory comorbidity • Suprarenal/TAAA repair (thoracic opening) • Emergency surgery • Albumin <30g/L, urea >10.7mmol/L • Poor functional status preoperatively
Monitoring renal function • Serum creatinine • Urine volumes	Risk factors for renal failure • Preoperative hypotension • Supra renal cross-clamp • Prolonged clamp time • High embolic load • Contrast nephropathy • Post-operative blood loss

(Continued)

Table 11.1 *(Continued)*

Factors	Notes
Intra-abdominal pressure	Risk factors for intra-abdominal hypertension: • Prolonged hypotension • Cardiopulmonary resuscitation • Base excess >14mEq • Fluid resuscitation >4L/h
Mesenteric perfusion: • Arterial lactate • Liver enzymes	Risk factors for post-operative mesenteric ischaemia include: • Supra-coeliac cross clamp • Intra-abdominal hypertension • Atheromatous disease superior mesenteric artery • Massive vasopressor requirements
Neurological monitoring	GCS status after CEA:Transcranial Dopplers after CEA
CSF pressure and volumes	Risk factors for spinal cord ischaemia: • Open TAAA repair • Post-TEVAR: patients with previous AAA repair, extensive stent coverage below T9, occlusion of left subclavian artery or hypogastric artery

Non-invasive assessment of cardiac output

- The oesophageal Doppler (CardioQ™) calculates the aortic cross-sectional area from a nomogram and non-invasively measures peak velocity, stroke volume, cardiac output, and FTc.
- The appearance of the velocity–time curve may be diagnostic of differences in circulating volume, including hypovolaemia, increased afterload or ventricular failure.

Functional haemodynamic monitoring (fluid optimization)

- Static markers of preload, such as CVP and PAOP measurements are poor predictors of fluid responsiveness.
- During positive pressure ventilation, cyclical changes occur in the right and left ventricular stroke volumes. The magnitude of these changes are predictive of the response of the circulation to intravenous fluids.
- 'Fluid responsiveness' can be measured using:
  - Stroke volume variation (SVV).
  - Systolic pressure variation (SPV).
  - Pulse pressure variation (PuPV).

Post-operative surveillance for biomarkers of tissue injury

- There is a close relationship between the magnitude of changes in post-operative cardiac troponin (cTn) concentrations, the duration of ischaemia on continuous ECG monitoring and post-operative mortality.

- Increases in both cardiac troponin I (cTnI) and cardiac troponin T (cTnT) are associated with equivalent risk.
- Plasma troponin concentrations should be measured in all patients with post-operative ECG changes or chest pain typical of acute coronary syndrome.
- The release of troponin following myocardial injury is biphasic. Up to 3% of patients may have a delayed peak in cTnI after 24h, preceded by a period when cTnI measurements are abnormal.
- Serial measurements are necessary to identify these patients with ongoing myocardial injury and guide aggressive intervention to prevent post-operative MI.
- Increased post-operative BNP concentrations are associated with cardiac death, non-fatal MI and major cardiac events.
- The optimal threshold values for post-operative plasma troponin and BNP concentrations have not been identified, and false positives may occur with several conditions including ventricular failure, diastolic dysfunction, PE, pulmonary hypertension, arrhythmias, and sepsis.

Post-operative critical care

Pre-emptive decisions regarding the site of post-operative care, and monitoring should be made based on individual preoperative risk assessment and the expected risk of the surgical procedure. There are also practical issues concerning the adequate provision of resources (multidisciplinary staff and medical equipment).

The duration of post-operative monitoring is also an important consideration, as the majority of myocardial ischaemic events occur between 48 and 72h after surgery. These decisions may be subject to change depending on surgical procedure complexity.

Utilization of critical care resources: admission criteria for HDU/ICU

- The NCEPOD report (2005) found that 56% of patients were admitted to intensive care following elective open AAA.
- 42% of these patients underwent post-operative artificial ventilation and in 34% overall the duration of ventilation was <24h, suggesting an inappropriate use of level 3 resources.
- Subsequently, the review 'Comprehensive Critical Care' recommended an increase in level 2 capacity and that level 2 and level 3 beds should be used interchangeably.
- Extubation immediately at the end of surgery should be considered in patients who are normothermic, have no signs of cardiovascular instability and have adequate analgesia.

Predicted length of stay in critical care

- Presence of a cardiovascular complication is a main determinant of an increased length of stay.
- Other ICNARC-validated predictors for increased length of stay are:
  - Increasing age.
  - Poor nutritional status.
  - Renal failure.
  - Poor pre-existing cardiovascular function.

Discharge from critical care

- Mortality is increased significantly (up to 37%) in patients discharged prematurely from critical care and subsequently needing readmission.
- Units will have their own discharge policies but suggested criteria include:
  - Haemodynamic stability without the need for vasoactive drugs.
  - Satisfactory respiratory function (assessed by RR, SpO_2, ABG, minimal respiratory secretions).
  - Adequate analgesia to allow mobilization and coughing.
  - Hb >8g/dL and stable with no indication for further surgical intervention.
  - Urine output >0.5mL/kg/h with normal or improving renal biochemistry and stable, predictable fluid requirements
- Patients should be monitored using standardized physiological scores, such as the Early Warning Score (EWS) or Modified EWS (MEWS).
- A high score is associated with an increased risk of clinical deterioration and should prompt early intervention to prevent readmission to critical care.

Major vascular surgery in the absence of ITU or HDU facilities

- Major elective surgery should *not* proceed unless all elements of care are available, including the provision for post-operative monitoring.
- The routine use of the recovery room (PACU) for prolonged care is **not recommended** (NCEPOD guidelines).
- Post-operative acute care units may provide level 2 care for patients who are likely to be able to return to the ward within 24h of surgery.
- Patients undergoing emergency surgery may present a difficult ethical dilemma over the rationing of critical care resources.
- Discharge of other patients from critical care to accommodate an emergency admission may be inevitable, but efforts should be taken to avoid this if at all possible.

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Perioperative myocardial infarction

Incidence

- Perioperative MI is the most common serious cardiovascular complication in patients undergoing major non-cardiac surgery. The POISE study reported a 30-day mortality rate of 11.6% in patients who had suffered a perioperative MI compared with 2.2% in those who had not.
- The reported incidence of perioperative MI varies depending on the diagnostic criteria and sensitivity of the biomarker used to detect myocardial injury. The incidence is set to rise with the widespread and systematic use of more sensitive biomarkers. The current literature suggests perioperative MI rates range from 1.4% in unselected patients to 27% in vascular patients with prior IHD.
- Most events occur within 48h of surgery.
- 65% of patients with a peri-operative myocardial event do not have typical symptoms and only 14% have typical 'cardiac' chest pain.
- The risk factors and validated risk indices for perioperative MI events are discussed 📖 Evaluation of the vascular surgical patient, p. 85. Patients with multiple cardiovascular morbidities including IHD, heart failure, and CVD are at increased risk of complications. DM and renal impairment further increase the burden of risk.
- Strategies to optimize the management of cardiovascular disease prior to surgery in order to reduce the risk of perioperative acute coronary syndromes and MI are discussed in 📖 Protecting the heart, p. 143.

Diagnosis

- MI is characterized by myocyte necrosis resulting from ischaemia (as distinct from myocyte necrosis 2° to trauma or myocarditis).
- The diagnosis of MI depends on two of the following three findings:
  - Symptoms typical of myocardial ischaemia.
  - Changes in biomarkers.
  - ECG changes of ischaemia, new or pathological Q waves, or new bundle branch block.
- In the clinical setting the patient may present with ischaemic myocardial injury ranging in severity from unstable angina to an ST-segment myocardial infarction. This range of presentations is encompassed by the term acute coronary syndrome (ACS) which is discussed in detail below under investigations.
- Only 50% of patients have symptoms suggesting myocardial ischaemia. ECG and troponin measurements should be taken in all patients with symptoms or signs suggestive of myocardial ischaemia.
- Routine post-operative screening (with ECGs or biomarkers, such as troponin I or troponin T) is advocated to detect myocardial ischaemia in high-risk patients, but is currently not recommended in lower risk patients.
- The routine use of intraoperative 5-lead ECG monitoring in high risk patients should be considered. This should be continued into the

post-operative period if there is any suspicion of cardiac complications as few patients with a peri-operative MIs will have typical chest pain.

- Troponins are the preferred cardiac biomarkers for the detection of myocardial necrosis. Cardiac troponin I or T are detectable within 12h of the cardiovascular event
- However, it is not yet clear as to whether systematic measurement of troponins will lead to earlier interventions, which will reduce morbidity or mortality.
- Some authorities suggest that troponin measurement should be routine in high risk patients. However, this is not widespread practice because:
  - The diagnostic cut-off level for high sensitivity troponin assays is unclear.
  - Other causes of increased troponin concentrations (including pulmonary emboli, arrhythmias and acute kidney injury) can interfere with diagnosis.

Investigations

Both biomarkers (cardiac troponin I or T) and ECG assessment are required.

Perioperative ischaemic myocardial injury should be considered within the same framework as used in the non-operative setting, that of ACS. ACSs encompass a range of acute ischaemic myocardial conditions.

- *ST-segment elevation ACS:* these patients have persistent (greater than 20min) ST-segment elevation on the ECG and, in the non-operative setting, frequently have chest pain. Most of these patients develop and ST-segment elevation myocardial infarction (STEMI)
- *Non ST-segment elevation ACS:* in the non-operative setting these patients present with chest pain, but do not have ST-segment elevation. Ischaemic ECG changes may be seen. These may include ST-segment depression, T-wave inversion or T-wave flattening. Non-ST-segment ACS is divided into:
  - *Unstable angina*—in which the patient has normal troponin levels.
  - *NSTEMI*—in which a rise in troponin levels is seen.

If it is likely or probable that the patient has an ACS advice from a cardiologist should be sought.

The treatment of patients with ACS is based on risk stratification to predict the likelihood of death or further myocardial infarction. Risk stratification tools such as the Global Registry of Acute Coronary Events (GRACE) calculator take into account factors including, age, haemodynamic status, ECG changes, and biomarker elevation. Details of treatment pathways based on the outcome of risk stratification can be found in the UK National Institute of Health and Care Excellence (NICE) Guidelines. In the surgical setting a management plan for the patient who has suffered an ACS should be decided in discussion the surgeon and with a cardiologist. Possible components of treatment are outlined in the section on management below.

Echocardiography for patients with suspected or confirmed ACS will help risk stratify and confirm extent of LV systolic dysfunction. It can aid

diagnosis of ACS in confirming the presence of presumed new regional wall motion abnormalities, in addition to excluding other significant abnormalities seen with acute PE or valve lesions.

It should be remembered that perioperative MI is not the only possible cause of haemodynamic instability and ECG changes. Differential diagnoses should be considered, including:

- Hypovolaemia.
- Pulmonary embolism.
- Pneumothorax.
- Anaphylaxis.
- Sepsis.
- Cardiac tamponade.

Management

- Maintain a high index of suspicion for ACS in any patient with symptoms that could potentially be cardiovascular in origin especially in higher risk patients. Continuous ECG monitoring and invasive peri-operative monitoring, such as arterial BP are indicated in these groups (Fig. 11.1) .
- If a peri-operative ACS is suspected or confirmed then establish close cardiovascular monitoring. A 12-lead ECG should be obtained as soon as practical, even during the operation if feasible as a decision whether to continue or abandon surgery may need to be made.
- The MAP should be maintained at or close to the preoperative value. Careful attention to intravascular volume status is required:
  - Invasive arterial pressure monitoring should be instituted if at all possible.
  - Modern techniques allow the interpretation of the pressure trace to allow calculation of pulse and stroke volume variation with positive pressure ventilation. These give an indication of circulating volume. If such technology if available and the patient is undergoing positive pressure ventilation it should be used to direct fluid therapy with a view to achieving a pulse pressure or cardiac output variability of less than 12%.
  - If such technology cannot be deployed the oesophageal Doppler may be used to direct fluid therapy in the operative setting.
  - If cardiac output or pulse pressure variability monitoring is not available the response of the central venous pressure or MAP to one or more 250-mL fluid boluses may give an indication of volume status.
  - If the patient is hypotensive despite adequate filling or volume overload inotropic therapy should be considered. In this circumstance, haemodynamic monitoring should be used to direct therapy if at all possible. Drugs such as dobutamine may be used to support cardiac output. On occasion, a vasopressor such as noradrenaline is required to maintain MAP at levels that provide adequate tissue perfusion. If local expertise in transoesphageal echocardiography is available this may be used to direct the management of the anaesthetized patient.
  - In the patient with cardiogenic shock the use of an intra-aortic balloon pump should be considered if available.

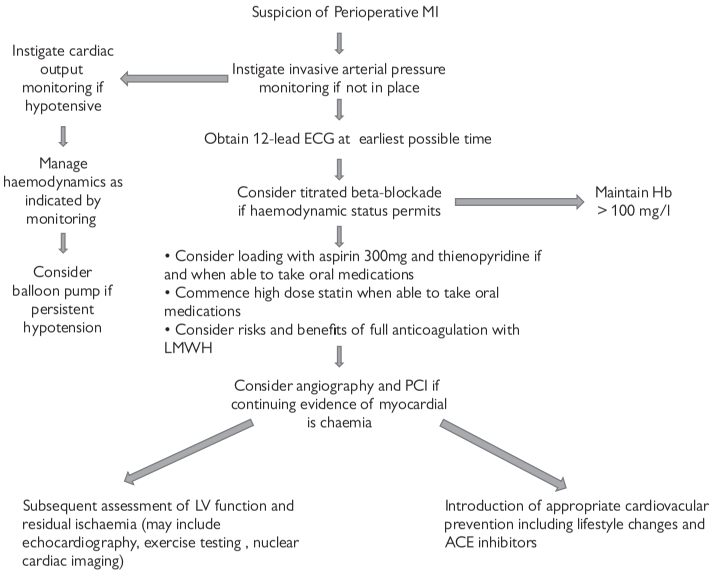

Fig. 11.1 Management of suspected perioperative MI.

- If there is evidence of acute pulmonary oedema with hypoxia the patient should be considered for positive pressure ventilation if this is not already in progress as part of intraoperative management.
- Anti-platelet agents, aspirin 300mg and clopidogrel (300–600mg) should be administered (via NGT if required) as soon as practical, following discussion with surgical team and cardiologists. Choice of agents depends on local protocols and the need for emergency revascularization, balanced against the bleeding risk imposed by the recent surgery. More recent agents (prasugrel and ticagrelor) offer a more rapid onset of platelet inhibition compared with a standard dose of clopidogrel. The bleeding risk is especially important when considering more potent anti-platelet agents such as glycoprotein 2b/3a receptor inhibitors and full heparinization with IV unfractionated or LMWH and, again, the decision to use these drugs requires discussion between cardiologist, anaesthetist, and surgeon.
- Statins should be commenced or continued, with consideration given to high intensity doses, such as atorvastatin 80mg, which may offer additional morbidity benefits over standard doses (simvastatin 40mg). Choice will depend on likelihood of drug interactions and myopathy risk.
- β-blockers should be considered to lower O_2 demand, reduce arrhythmias and potentially infarct size. IV β-blockers carry an increased risk of hypotension and bradycardia, but can be used carefully if oral intake is not possible.
- The haemoglobin concentration should be monitored regularly, both during and after surgery as levels below 100g/L increase cardiac risk. Other drugs, such as IV nitrates, can be used and titrated according to the haemodynamic responses to lower BP and reduce any ischaemic burden, but have not been shown to have mortality benefit.
- In the non-operative setting the current NICE Guidelines recommend that patients who have a 6-month risk of death of 3% or greater are offered angiography, and possible acute coronary revascularization. An example of such a patient would be a haemodynamically stable 65-yr-old man with ST-segment elevation and biomarker evidence of myocardial injury. There are no similar guidelines to direct management in the operative setting and clinical management decisions are made on a case by case basis. PCI requires the use of antiplatelet drugs and heparin that my precipitate life-threatening bleeding after major surgery. If there is evidence of ongoing myocardial ischaemia despite medical treatment it may be considered that the patient requires transfer to cardiology centre providing primary PCI (in the case of ST elevation events) or urgent angiography for non-ST elevation MI. The choice between interventional or conservative strategies will depend on both patient factors and perceived bleeding risk (PCI requiring heparinization and dual anti-platelet agents).
- Assessment of LV function with echocardiography can detect any early complications and provide prognostic information. Patients should also have subsequent assessment for residual ischaemia, especially if conservatively managed without early angiography. The imaging choice

dependent on local availability and includes options of nuclear perfusion scans, cardiac MRI stress perfusion, stress echocardiography, or exercise tolerance tests.

Secondary prevention

- Refer to cardiologist for 2° prevention and cardiac rehabilitation.
- 2° prevention includes:
  - Up-titration of β-blockers.
  - ACE-inhibitors and/or aldosterone antagonists (eplerenone) for patients with LV dysfunction.
  - Dual anti-platelet therapy for 1yr after ACS.
  - Statins.
- Patient education to improve compliance.

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Post-operative hypertension

Incidence and treatment thresholds

- Post-operative hypertension occurs in approximately 3% of all patients after surgery.
- The incidence is higher after some types of surgery (~35% after coronary revascularization and up to 66% after carotid endarterectomy) with the majority of these requiring therapeutic interventions (📖 Blood pressure management for CEA, p. 391).
- Treatment thresholds in the general population are based on reducing long-term cardiovascular risk of hypertension. These risks start to increase at pressures >115/75mmHg and double with each increment of 20/10mmHg. Although increased diastolic pressure has traditionally been the main indicator for therapy, systolic hypertension (>140mmHg) is now considered a more important risk factor for cardiovascular disease than diastolic pressure in those aged >50yrs. Current guidelines suggest a target BP <140/90, with lower goals for those with diabetes or CKD.
- However, there are no nationally-agreed guidelines for the investigation or treatment of post-operative hypertension.
- Post-operative hypertension can be defined as a systolic pressure >180mmHg or diastolic pressure >110mmHg; or an increase of >20% from the patient's baseline BP. Vascular surgical patients have a high incidence of CAD and so lower thresholds for treatment should

be considered: a systolic pressure >160mmHg or diastolic pressure >90mmHg are suggested.
- Post-operative systolic hypertension is associated with haemorrhage or haematoma formation, haemorrhagic stroke, arrhythmias, and myocardial ischaemia.
- Hence, lower treatment thresholds may be indicated in specific circumstances, where the risk of post-operative bleeding is high or after carotid surgery when there is a risk of cerebral hyperperfusion.
- In most cases hypertension occurs in the first 30min after surgery, but the onset may be delayed for up to 2h post-operatively. Episodes are frequently self-limiting or have a clearly defined cause; as a result most episodes have abated after 2h, but they may continue for up to 4h or longer if there is an ongoing underlying cause.
- Late (>24h) post-operative hypertension may also occur and is frequently caused by omission of anti-hypertensive drugs.

Causes and risk factors

- Post-operative hypertension is usually caused by increased systemic vascular resistance as consequence of increased sympathetic nervous system activity and activation of the renin-angiotensin system. This is part of the stress response to surgery and may be exaggerated by pain or other factors.
- Baroreceptor dysfunction predisposes to arterial pressure lability. Baroreceptor function is temporarily disturbed by general anaesthetic agents. There is an increased incidence of baroreceptor dysfunction in vascular surgical patients because of pre-existing hypertension, diabetes, recent stroke, or TIA. Baroreceptor dysfunction is most common after carotid surgery.

Risk factors

- Pre-operative hypertension (greater if poorly controlled or in the presence of end organ dysfunction; notably increased risk if pre-operative diastolic pressure >110mmHg).
- Autonomic neuropathy (e.g. in DM).
- Type of surgery (carotid>abdominal aortic, peripheral vascular, intra-abdominal).
- Bilateral carotid atheroma.

Common post-operative causes

- Hypoxia.
- Hypercarbia.
- Hypothermia.
- Pain.
- Anxiety.
- Fluid overload.
- Urinary retention.
- Drug related confusion and agitation (emergence phenomenon).
- Anti-hypertensive drug withdrawal.

Uncommon post-operative causes

- Drug and alcohol withdrawal.
- Renovascular hypertension (despite being more common in vascular patients it is relatively rare to diagnose this post-operatively).
- Thyrotoxicosis.
- Phaeochromocytoma.

Complications of post-operative hypertension

- Myocardial ischaemia and MI.
- Left ventricular failure (LVF).
- Arrhythmias.
- Haemorrhagic stroke.
- Bleeding and haematoma formation (this may cause particular problems after carotid endarterectomy when a neck haematoma can lead to airway obstruction).
- Cerebral hyperperfusion syndrome.
- Hypertensive encephalopathy (rare).
- Acute aortic dissection (rare).

Immediate assessment and management

- Follow an ABCDE approach (Table 11.2).
- Exclude airway problems and hypoxia immediately. This will also ensure that hypercarbia is assessed and detected.
- Assess for complications associated with acute hypertension (LVF, bleeding, etc.).
- During initial assessment, quickly consider the common post-operative causes listed above.

Monitoring

Institute appropriate monitoring

- *Invasive arterial pressure:* many vascular patients will already have an in-dwelling arterial catheter. It is important to be aware that these can under- and over-read the 'true' arterial pressure. This occurs as a result of either under- or over-damping, but it should be remembered that the MAP will still be accurate despite the error in systolic and diastolic pressures. Under damping leads to an over-estimate of the systolic and an under-estimate of the diastolic and may occur due to soft tubing being placed to extend the line. Over-damping causes the opposite and may be due to kinking of the line, or due to thrombus or air in the line. The main advantage of invasive monitoring is beat-to-beat measurement when there is likely to be rapid changes in pressure (e.g. when treating with IV agents).
- *NIBP:* if there is no direct arterial catheter in place or, if in doubt, about the reading, check with NIBP. Use correctly-sized cuff (cuffs that are two small will lead to an over-estimate of 'true' BP and vice versa). NIBP gives an accurate systolic and mean figure with the diastolic being a calculated figure and ∴ more prone to error.
- *ECG:* single lead ECG monitoring should be started to detect arrhythmias. If there is any concern about myocardial ischaemia then multi-lead monitoring and a formal 12-lead ECG should be performed.

Table 11.2 Suggested algorithm for immediate assessment

A	*Assess the airway:* neck haematoma can lead to rapid loss of airway; if this is suspected call for help immediately. Administer O_2 4L/min by facemask or nasal cannulae to all hypertensive patients
B	*Check O_2 saturations:* hypoxia is a potent cause of hypertension, give oxygen if not done so already. Titrate oxygen to maintain SpO_2 >95% *Assess respiratory rate and pattern:* poor respiratory function leading to hypercarbia will cause hypertension
C	*Recheck arterial pressure:* if invasive pressure monitoring is being used, re-zero and re-check (is the transducer at the level of the heart? Is the waveform appropriate or under-damped?). If in doubt, double check against NIBP *Assess the cardiac rhythm:* look for the presence of haematoma formation
D	*Assess and treat pain:* pain activates the sympathetic nervous system and is one of the most common causes of post-operative hypertension *Assess conscious level, confusion or anxiety:* a decreased conscious level may give diagnostic clues (e.g. CO_2 narcosis) or be a marker of severity of hypertension (e.g. hyperperfusion syndrome). Confusion or anxiety after carotid surgery may be an early sign of cerebral ischaemia Assess and treat post-operative nausea and vomiting
E	*Exclude urinary retention:* conversely the presence of a urinary catheter can cause pain or discomfort after carotid surgery. Check patient temperature and institute active warming if <36°C

Causes identified immediately should be treated before resorting to antihypertensive drugs, unless hypertension is severe or complications are present. In many cases treatment of these causes will be sufficient to reduce blood pressure to acceptable levels.

- SpO_2 and respiratory rate.
- Core temperature.

Specific treatment of hypertension

Treatment will depend on whether it is a hypertensive emergency or urgency. Management of hypertension after blood pressure after carotid surgery is discussed in 📖 Blood pressure management for CEA, p. 391 and Hypertension early after carotid surgery, p. 398.

- *Hypertensive emergencies*— systolic blood pressure (SBP) >180mmHg, diastolic blood pressure (DBP) >110mmHg or hypertension associated with complications.
- *Hypertensive urgency*—hypertension SBP >160mmHg, DBP >90mmHg and at high risk of complications.

Hypertensive emergencies

- Arterial pressure should be reduced using an IV agent; the aim should be to decrease the BP to approximately 160/100mmHg over a period of 15–20min, with any further reduction over the few hours following this.

- Hypertensive emergencies usually occur immediately after surgery and the patient is likely to be in a monitored environment. If, however, the patient is on a post-operative ward they should be transferred to an area with appropriate staffing and facilities
- The ideal antihypertensive would have an immediate onset of action, a short duration of action, be easy to titrate and have a wide therapeutic margin. Suitable drugs are labetalol, atenolol, metoprolol, hydralazine and GTN (see Box 11.1). Other agents, such as esmolol and sodium nitroprusside are best avoided outside a critical care environment.
- β-blockers are appropriate if heart rate is >70.

Hypertensive urgencies

- These include both the less severe immediate hypertensive episodes and the later or delayed cases (Box 11.2).
- Blood pressure can usually be controlled over several hours using oral medication unless there is a contraindication. Occasionally, IV agents may be required as described in Box 11.1.

Box 11.1 Suggested treatments for post-operative hypertensive emergencies, after treatment of immediately identified causes

First line

- *Labetalol (100mg in 20mL):* 10mg bolus, repeat at 2–5-min intervals until desired response. Consider other agent if no response after 50mg or poor response after 100mg. If BP decreases initially, but increases again, start labetolol infusion at 50–100mg/h, titrating dose to BP.
- *Atenolol:* 2.5mg IV repeated after 5min if needed up to 10 mg.
- *Metoprolol:* 5 mg IV then 2.5mg every 5min up to maximum of 15mg

If hypertension is associated with chest pain or ECG signs of myocardial ischaemia

- GTN 800μg by sublingual spray or 2–20mg/h by IV infusion (Box 11.2)
- β-blockade is an alternative if HR >70.

Second line, or if HR<65

- *Hydalazine (10mg in 10mL):* 2mg slow IV injection, repeat at 5–10-min intervals until desired response. Use another agent if no response after 10mg or rebound occurs.
- *GTN (50mg in 50mL, i.e. 1mg/mL):* continuous infusion, start at 5mL/h, increase up to 20mL/h for desired effect. If hypertension is associated with chest pain or ECG signs of myocardial ischaemia.

Box 11.2 Suggested treatments for post-operative hypertensive urgencies, after treatment of previously identified causes

If the patient is not normally on antihypertensive therapy

First line

Nifedipine LA (10mg), repeat after 1h if no change in BP. (**Do not use crushed nifedipine capsules**) If no reduction in BP, move to second line agent.

Second line

Bisoprolol 2.5–5mg. Maximum dose 10mg.

Third line

Ramipril 5mg, repeated after 3h if necessary.

If the patient is normally on antihypertensive therapy

First line

Administer patient's usual medication. Consider dividing doses if multiple antihypertensive medications.

Second line

- If patient is on ACE inhibitor and/or diuretic, nifedipine LA 10mg.
- If patient is on Ca^{2+} channel blocker, ramipril 5mg.

Further reading

Marik PE, Varon J. Hypertensive crises: challenges and management. *Chest* 2007; **131**: 1949–62.

Post-operative hypotension

- Post-operative hypotension is common after all types of surgery. The incidence is high after vascular surgery because of pre-existing cardiovascular disease, the effects of anaesthetic and antihypertensive drugs, disturbed baroreceptor function, autonomic blockade, and the effects of major surgery.
- Hypotension is a predictor of cardiovascular complications, specifically myocardial infarction and stroke; vascular surgical patients are at particular risk of these and ∴ post-operative hypotension is of greater importance.
- Post-operative hypotension caused by residual anaesthetic agents may be short-lived and benign, but in all cases hypotension should be assessed promptly and treated if necessary.

Definitions and aetiology

- Post-operative hypotension can be defined a reduction of BP of >20% from the patients normal BP. Alternative definitions are a systolic pressure <100mmHg or MAP <65mmHg.

- Note that hypotension in itself may not cause any physiological abnormality and systolic pressures of 80–90mmHg may be well tolerated in patients without cardiovascular disease.
- Only when hypotension causes a decrease in perfusion (either globally or locally) is homeostasis disturbed.
- Hypotension can lead to shock, which is defined as the presence of end organ dysfunction caused by hypoperfusion.

- Arterial pressure is directly related to cardiac output and systemic vascular resistance (SVR). (BP ∝ CO × SVR).
- Cardiac output is the volume of blood ejected from the left ventricle per unit time. It is the mathematical product of heart rate × stroke volume (SV).

- SV is determined by preload, contractility, and afterload (these are assumed theoretically to be independent of each other, but in practice are interdependent).
- Hypotension can be considered as a problem of reduced preload, decreased contractility, reduced SVR, either alone or in combination. This approach helps in diagnosis and management (Table 11.3).
- Preload is the end-diastolic stretch of the cardiac fibres, this is affected the blood volume, venous tone, and cardiac compliance.
- Afterload is the tension that cardiac cells must create to cause ejection, this is affected by AV valve diameter, arterial pressure (and hence SVR) and presence of cardiac dilatation or hypertrophy.

Types of shock

There are four main types of shock:

- *Cardiogenic:* 1° failure of cardiac contractility leading to reduced SV and CO.
- *Hypovolaemic:* 1° reduction intravascular volume with reduced filling of the heart leading to reduced SV and CO.
- *Distributive:* 1° problem of vasodilatation causing decreased pre- and afterload:
  - Septic.
  - Anaphylactic.
  - Neurogenic.
- *Obstructive:* prevention of either outflow (PE) or inflow (tamponade) of the heart.

Note: presence of hypotension without any end organ dysfunction does not equal shock.

Immediate assessment

The main priority is to assess the severity and cause of hypotension, and exclude any immediate life-threatening causes using an 'ABC' approach. Early post-operative hypotension is often caused by the residual effects of anaesthetic or analgesic drugs and is self-limiting. If mild and producing no physiological disturbance, no treatment is required; alternatively, simple measures, such as elevation of the foot of the bed or IV colloid infusion 3–4mL/kg.

Table 11.3 Common causes of post-operative hypotension

Reduced preload	Absolute	Hypovolaemia due to inadequate intraoperative fluid or blood replacement
		Haemorrhage
		Major pulmonary thromboembolism (reduces LV preload)
	Relative	Sympathetic blockade (e.g. regional anaesthesia)
		Drug-induced vasodilation (e.g. anaesthetic agents)
		Rewarming
		Sepsis
		Positive pressure ventilation
		Raised intra-abdominal pressure
		Tension pneumothorax
		Cardiac tamponade
Decreased contractility	1°	Myocardial infarction or ischaemia
		Arryhythmias (atrial fibrillation, tachycardias or bradycardia)
	2°	Hypoxia
		Acidaemia
		Drug-induced
		Sepsis
Reduced afterload		Sympathetic blockade (e.g. regional anaesthesia)
		Anaphylaxis
		Sepsis
		Drug-induced (e.g. anaesthetic agents, antihypertensives)
		Rewarming

Airway/breathing

Assess respiratory function (RR, depth, pattern and SpO_2) to exclude hypoxia and its major causes; as well as tension pneumothorax or major thrombo-embolic event) Give oxygen 4L/min by face mask, titrating flow rate to maintain SpO_2 >95%.

Circulation

- Recheck BP and other vital signs.
- *Assess the heart rhythm:*
  - Profound tachycardias cause hypotension because the time for cardiac filling during diastole is decreased.
  - Bradycardias may lead to hypotension due to decreased cardiac output.
  - Atrial fibrillation leads to a loss of the atrial kick in filling of the ventricles and, hence, a reduced stroke volume for given atrial preload.

- *Assess peripheral perfusion:*
  - *Capillary refill time*—less than 2s is normal. If peripheral capillary refill is abnormal check central (trunk) capillary refill also.
  - *Cool peripheries*—generally indicate peripheral vasoconstriction. This may be due to either hypovolaemia or reduced contractility.
  - *Warm peripheries*—suggest normovolaemia or vasodilatation.
  - *Check peripheral pulses*—the hypotensive episode may be caused by or lead to thrombotic events in at risk limbs.
- *Assess perfusion of vital organs:*
  - *Assess conscious level*—a decreased conscious level may indicate poor perfusion and is a sign of shock. Unless there is clear evidence to the contrary (e.g. anaesthetic agent or drug-induced) assume a reduced conscious level is a sign of shock until proven otherwise. If conscious level impaired, check pupils, peripheral neurological examination (GCS) and blood glucose.
  - *Check urine output*—a urine output <0.5mL/kg/h within the first few hours after surgery is often part of the physiological stress response to major surgery and is a non-specific sign. Ongoing oliguria should be identified as it may indicate poor renal perfusion (see 📖 Postoperative oliguria, p. 490.
  - Enquire about the presence of chest pain and check for signs of ischaemia on the ECG.
- *Assess for other causes:*
  - *Look for signs of dehydration*—dry mucous membranes and thirst.
  - Confirm the presence or absence of haemorrhage.
  - *Measure or estimate the jugular venous pressure (JVP) or CVP*—a raised JVP suggests tamponade, tension pneumothorax, or cardiac failure as a cause.

Further assessment

- Check core temperature as a sign of sepsis or hypothermia.
- Examine for haemorrhage including the abdomen, wound sites, and drains.

Monitoring

- There is little substitute for the presence of an experienced clinician. If in doubt get senior help.
- *Basic vital signs:* pulse, RR, BP, and temperature. Consideration should be given to the need for a urinary catheter.
- *SpO_2:* indicates hypoxaemia, but can also give an indication of peripheral perfusion.
- *ECG:* 3-lead continuous monitoring is useful to assess arrhythmias; 12 lead formal ECGs should be obtained if myocardial ischaemia is suspected.
- *ABG:* provide data both on respiratory function and metabolic state; the metabolic component may demonstrate poor tissue perfusion in the form of metabolic acidosis or raised lactate.
- *Invasive monitoring:* usually after failure of initial treatment, but if present allows accurate measurement of CVP.

Initial treatment

In almost all cases, barring obvious cardiac causes, the initial treatment should be:

- Give oxygen 4L/min by face mask, titrating flow rate to maintain SpO_2 >95%.
- Lay the patient flat.
- Give a bolus of fluid 250mL of crystalloid or colloid rapidly.

This should be done in parallel with the initial assessment allowing for a reassessment of vital signs at the end of the initial assessment.

Further assessment/treatment

This depends on the presumed diagnosis, severity of hypotension, and response to initial treatment. Patients may fall into one of several categories.

Hypotension, but not shocked (no signs of end organ dysfunction)

- Note this is a diagnosis of exclusion.
- No specific treatment is necessary. Increase the frequency of vital signs observations.

Hypotension with decreased preload

- Carefully assess to exclude haemorrhage. Taking a FBC or checking the Hb using an ABG may be prudent in cases where there is doubt, as there is a notable risk of post-operative haemorrhage and coagulopathy in the vascular population. If there is any concern re haemorrhage (overt or hidden) then senior help should be sought.
- If ongoing haemorrhage is excluded then most cases respond to the judicious infusion of IV fluids (e.g. a 250-mL bolus of crystalloid or colloid, repeated if necessary after further assessment).
- If no response after two fluid boluses, then further advice should be sought and a full reassessment should occur, for missed or occult diagnoses.

Hypotensive with decreased afterload

- This is probably the most common cause of hypotension post-operatively caused by the residual effects of anaesthetic agents and analgesics in most cases, it is probably a combination of a reduced preload with a reduced SVR. In this circumstance, the majority of cases respond to IV fluids alone.
- If there is no response after two fluid boluses, further assessment is needed. Ephedrine (increments of 3–6 mg IV) or phenylephrine (increments of 50–100micrograms IV) may be given. If the patient remains shocked, then seek senior help: invasive monitoring, ICU admission and vasoconstrictor drugs are often required.

Hypotensive with decreased contractility

- This is the least common cause of post-operative hypotension.
- Ensure adequate oxygenation, treat arrhythmias; in the absence of signs of LVF a cautious fluid challenge may be appropriate. If LVF is present then first line treatment should be instigated: diuretics (furosemide 40mg IV stat), vasodilators (GTN spray initially) and possibly analgesia/

anxiolysis (diamorphine 2.5mg IV stat). CPAP should be considered early.

- Treatment of post-operative cardiac dysfunction is difficult and senior help should be requested early. In the absence of tachycardia, ephedrine (increments of 3–6mg IV) may be given whilst awaiting advice (Table 11.4).
- *If no response to these simple measures then seek expert help:* invasive monitoring, ICU admission and other positive inotropic agents may be required.

Further monitoring and treatment

Regardless of the cause of hypotension or shock, continued assessment is needed after instigating initial treatment. If the patient responded to simple measures this may only be a repeat of vital signs and examination, but if multiple treatments have been necessary then a more in depth assessment is needed including arterial blood gas analysis to look for metabolic acidosis and hyperlactataemia (lactate >2mmol/L). Patients that fail to respond to first line measures should be assessed for admission to ICU.

Table 11.4 Treatment of hypotension

Reduced preload	Absolute	Fluid replacement Control haemorrhage	250-mL bolus of IV crystalloid or colloid. Repeat if required Apply pressure to bleeding sites and contact surgeon for intervention
	Relative	Fluid replacement Occasionally, vasopressors or positive inotropes Treatment of tamponade or tension pneumothorax	250-mL aliquots of fluid. Repeat if required Seek senior help. ICU admission may be required Emergency needle decompression and/or chest drain insertion
Decreased contractility	1°	Oxygen Control arrhythmias Vasoactive drugs	Maintain SpO_2 >95% Treatment depends on rhythm Seek senior help. ICU admission may be required
	2°	Oxygen Vasoactive drugs	Maintain SpO_2 >95% Seek senior help. ICU admission may be required
Reduced afterload		Fluid resuscitation Vasoactive drugs Treat the cause	250-mL aliquots of fluid. Repeat if required Seek senior help. ICU admission may be required Antibiotics/source control for sepsis

Further reading

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Post-operative oliguria

The stress response to surgery includes activation of the renin-angiotensin-aldosterone system with increases in concentrations of ADH. Together, these cause sodium and water retention (aldosterone) and inhibit water reabsorption (ADH) for a variable duration (up to 24–36h) depending on the degree of surgery and tissue damage. This tendency for fluid retention and oliguria after major surgery may be exacerbated by perioperative hypotension, hypovolaemia, positive pressure ventilation, and the effects of some drugs (e.g. opioids). In vascular patients it is most common after aortic surgery, particularly ruptured/emergency AAA.

Important predisposing factors for perioperative oliguria and AKI

- CKD.
- Hypotension, hypovolaemia, haemorrhage, major surgery with large blood loss, or fluid shifts
- Sepsis, HF, liver failure.
- Aortic surgery, especially when urgent.
- Prolonged AXC times.
- Suprarenal AXC.
- Renal artery stenosis.
- Old age.
- Vasoconstrictor drugs.
- Nephrotoxins (NSAIDs, ACE inhibitors, radiocontrast dyes).
- Rhabdomyolysis (severe surgical trauma, ischaemia reperfusion injury, crush injury).

Diagnosis

- Defined as UO < 0.5mL.kg/h. Early detection and intervention important as it is often easily reversible, but can progress to AKI if prolonged >6h. AKI has a high morbidity and is an independent marker of post-operative mortality. Conversely, AKI can occur in the absence of oliguria.
- Causes are pre-renal, renal, or post-renal obstruction (see 🕮 Perioperative renal protection, p. 236).
- Acute oliguria or anuria is most likely to be due to post-renal obstruction (e.g. clot retention, catheter obstruction). Catheterize or flush catheter if already catheterized.

Precipitating factors after vascular surgery

- Ongoing haemorrhage, coagulopathy.
- Sepsis.
- Nephrotoxins (e.g. radio contrast, NSAIDs, aminoglycosides).

- Ischaemia-reperfusion injury.
- Intra-abdominal hypertension/abdominal compartment syndrome.
- Rhabdomyolysis.

Management of oliguria (Box 11.3)

Box 11.3 Management of oliguria

- Exclude bladder outflow obstruction, which is often reversible. Can be confirmed by clinical examination and/or bladder US. Place urinary catheter if not already catheterized. Flush catheter/bladder washout with 50–100mL sterile water.
- If ureteric obstruction or hydronephrosis is a clinical possibility, consider early abdominal US/CT and urology referral. If severe bladder outflow obstruction (e.g. urethral stricture) is present, a suprapubic catheter may be required.
- Evaluate clinically for evidence of reduced circulating volume, i.e. presence of tachycardia, hypotension, low or absent JVP, thirst, cold peripheries (capillary refill >2s).
- Other signs include: CVP <8mmHg (or <12 if positive pressure ventilation), increase in CVP and MAP on passive leg raising, increased pulse pressure variability.
- Check fluid balance and anaesthetic charts for recent fluids in/out.
- Look for and discontinue any nephrotoxins (e.g. radiocontrast, NSAIDs).

If clinical suspicion of hypovolaemia

Give fluid challenge i.e. 250mL colloid over 15–30min. Can be repeated once or twice, but invasive monitoring should be instituted at this point.

- Increase maintenance fluids IV.
- Check U&E and ABG. Look for and treat hyperkalaemia.
- *Cardiovascular goals are:*
  - CVP >8 (breathing spontaneously, 12mmHg if IPPV) or pulmonary capillary wedge pressure (PCWP) > 12–16mmHg,
  - MAP >70mmHg (consider higher MAP if longstanding hypertensive).
  - CI >3.1L/min/m^2.
  - DO_2 >1000mL/min/m^2.
  - Hct >25%.
- If UO restored within 1–2h, continue monitoring. Look for and treat precipitating factors.
- *Once circulating volume restored:* if UO remains low for >2h, and/or MAP <70mmHg, start positive inotropes (e.g. dobutamine 2–10micrograms/kg/min) and/or vasoconstrictors (e.g. noradrenaline (0–2mg/h) to maintain renal perfusion.
- *If no response after further 2h:* and circulating volume and renal perfusion have been maintained, cardiovascular goals met and outflow obstruction excluded, consider diuretics (e.g. furosemide 10–20mg IV or infusion of 1–5mg/h). There is no benefit from doses of furosemide >50mg IV and higher bolus doses are associated with ototoxicity.

Fluid overload

If clinical suspicion of fluid overload (high JVP, oliguria, tachypnoea, pulmonary venous congestion, fine basal crepitations, peripheral oedema) administer diuretics and vasodilators. Maintain MAP <70mmHg and renal perfusion (e.g. with dobutamine 2–10micrograms/kg/min).

- Check urine and plasma U&E, osmolality (See Table 11.5). Does this confirm clinical suspicions?
- If renal cause for oliguria, maintain renal perfusion as above, stop nephrotoxins, consider renal imaging (US/CT). Consult nephrologists. Early CVVH may be indicated.
- If contrast-induced nephropathy suspected, consider N-acetyl cysteine (600mg po bd or 500mg IV bd for 24h post-procedure).
- If rhabdomyolysis suspected (clinical diagnosis, myoglobinuria, very high plasma CK) forced alkaline diuresis may be useful: administer $NaCO_3$ to maintain urine pH>8, mannitol 0.25–0.5mg/kg or furosemide infusion. Maintain MAP and renal perfusion as above.
- If prerenal aetiology for oliguria, look for cause and treat as above

If no improvement after 6h of oliguria:

- Recheck ABG, repeat U&E.
- Have cardiovascular goals been met?
- Have underlying causes been identified and treated?
- Maintain circulating volume and renal perfusion.
- Look for signs of metabolic acidaemia, hyperkalaemia, fluid overload.
- Be aware that increases in plasma urea and creatinine after an episode of AKI take 24–72h to develop.
- Consider diuretics (e.g. furosemide 10–20mg IV or infusion of 1–5mg/h). Diuretics have not been shown to improve outcome, but may help avoid fluid overload. Dopamine does not improve outcome
- **If no improvement after >24h**, refer to nephrology or ICU for consideration of renal replacement therapy (RRT).

Indications for RRT are:

- Severe salt and water overload (pulmonary oedema) unresponsive to diuretics.
- Hyperkalaemia (serum K^+ > 7.0mmol/L).
- Metabolic acidaemia (pH <7.15).
- Uraemia (urea >35mmol/L) where renal tract obstruction has been excluded.

Table 11.5 Urine biochemistry

Measure	Prerenal oliguria	Renal oliguria
Urinary [sodium] (mEq/L)	<20	>40
Fractional sodium excretion (%)	<1	>1
Urine osmolality (mOsm/L)	>400	250–300
Urine:plasma creatinine ratio	>40	<20
Urine:plasma osmolality ratio	>1.5	<1.1

Ensure urine and plasma biochemistry checked before administration of diuretics.

RRT should be instituted earlier in patients with known CKD who are already dialysis-dependent.

Further reading

Singer M, Webb A. *Oxford Handbook of Critical Care*, pp. 272–9. Oxford: Oxford University Press.

Post-operative bleeding

- Troublesome bleeding in the immediate period after vascular surgery is rare, but can be either overt or covert. It may follow elective as well as emergency surgery.
- There are only two causes of post-operative bleeding: a lack of surgical haemostasis or a coagulopathy.

Surgical causes of bleeding

Early

- Disruption of vascular integrity due to elevated BP.
- Stitch cut-out or unravelling of continuous suture.
- Vessel trauma that may have gone unwitnessed during surgery.
- Arterial or venous bleeding not obvious during surgical closure.

Late

- Graft infection.
- Aneurysmal progression in adjacent vessel.
- Pseudo-aneurysm formation at anastomosis.

Considerations

- If bleeding is suspected to arise within a body cavity the decision can be less easy. There may be a reluctance to re-operate on bleeding patient as re-operation is associated with a worse outcome; often associated with underlying problem requiring re-exploration, rather than actual procedure itself.
- Re-exploration in itself may increase risk of infection due to prolonged period of surgery and access to structures not usually open to the atmosphere, but all attempts need to be made to avoid morbidity and mortality from uncontrolled bleeding.
- Even in presence of wound drains, recorded blood loss via these devices may not truly reflect blood loss occurring within abdomen.

Lack of surgical haemostasis

- *Major bleeding* is uncommon unless a catastrophic event has occurred, such as disruption of a vascular anastomosis.
- *Wound bleeding:* overt bleeding may manifest itself as bleeding emerging through wound in case of peripheral vascular grafts, vascular access procedures, or carotid endarterectomy. If superficial, this is not usually associated with significant swelling and bleeding vessel can usually be seen.

- *Intra-abdominal bleeding:* covert bleeding usually presents with cardiovascular signs, suggesting ongoing blood loss, associated with a distended abdomen or respiratory distress. In general, failure to stabilize after infusion of 2U packed red blood cells over 2–3h suggests significant bleeding.
- If there is a surgically correctable cause, either due to vessel injury that has not been successfully corrected or a problem resulting from the surgical intervention, i.e. a direct breach in the vascular architecture that was caused by the surgical procedure. Successful management strategies rely upon prompt and timely intervention that will be guided by clinical assessment of most likely cause supported by confirmatory investigation if the clinical condition of the patient allows.

The clinical signs of bleeding that usually alert the medical and nursing staff when there is no obvious sign of blood loss include:
- Tachycardia.
- Hypotension.
- Low JVP or CVP.
- Fluctuating arterial wave form with respiration.
- Decreased urine output.
- Cold peripheries, poor or slow capillary refill time.
- An increasing temperature difference between core and peripheral temperature (Core – Toe temperature).
- Thirst, decreased conscious level.

Control of bleeding

Obvious bleeding
- Press on the wound.
- Take care taken to avoid excessive pressure; thrombosis may occur if excessive pressure is applied to the graft of a repaired vessel.

Life-threatening haemorrhage
- If haemorrhage is life-threatening, measures to prevent exsanguination are essential.
- For peripheral bleeding, use of tourniquet **may** be justified. More likely in severely traumatized patient.
- Brisk, large volume, concealed haemorrhage suggests that bleeding is present within abdominal cavity. Immediate return to theatre for surgical intervention is required.
- If the surgical cause cannot be identified at laparotomy and prolonged resuscitation and surgery has led to hypothermia and acidosis, packing of the wound and return to intensive therapy unit (ITU) for period of coagulation support and correction of abnormalities allows survival until a re-laparotomy can be considered.

Less dramatic bleeding
- If bleeding is not life-threatening, its source can be diagnosed by CT and/or CT angiography.
- US can be difficult to interpret in the post-operative period. A small amount of intra-abdominal fluid is often to be expected and its significance difficult to interpret.

- Girth measurements are notoriously difficult to interpret and even when measured at same anatomical point can be grossly misleading and often a late sign.

Contrast CT with real time angiographic screening

- Often helpful if patient is stable and able to lie still. A single vessel bleed of a small artery may be dealt with using an interventional radiological technique.
- Interventional techniques performed by a radiologist or competent vascular surgeon are a quicker and safer way of dealing with isolated vessel bleeding than surgical re-exploration.
- Emergency endovascular repair is becoming possible in some centres where the interventional skills and equipment are available.

Coagulation assessment including thromboelastography

- If there is uncertainty about site of bleeding and microvascular haemorrhage is considered, it may be appropriate to allow accumulation of blood, thereby increasing intra-abdominal pressure in an attempt to tamponade the bleeding.
- In these situations, the use of POC testing can be helpful. A rapid test, such as the TEG or ROTEM, can be invaluable, particularly if tests show normal trace. This indicates that a surgical cause of bleeding is more likely.
- Equally, an abnormal test will diagnose a coagulation problem and also offer information on the best use of blood component replacement.

Coagulopathy

- Unstable patients who have lost a significant amount of blood will have decreasing circulating clotting factors, platelets and fibrinogen (Box 11.4). They may be anaemic with decreased oxygen-carrying capacity.
- Major haemorrhage is accompanied by an acquired coagulopathy. Bleeding can occur from raw surfaces and even suture holes that do not seal off normally.
- Acquired coagulopathy is initiated by massive tissue destruction and endothelial injury in trauma or extensive surgery that, among other mechanisms, lead to release of tissue factor into circulation.
- Laboratory tests will reveal presence of D-dimers; thrombocytopenia; often a prolonged PT, PTT, and TT; and decreased fibrinogen levels (Box 11.4).

Diagnosis and treatment of coagulopathy

General approach

- Diagnosis of cause is often a process of elimination.
- Low concentrations of clotting factors and abnormal coagulation tests need correction.
- Normal values may not indicate normal coagulation.
- Clinical context is important. Response to the administration of clotting components should be monitored and recorded.
- Laboratory tests are usually hard to obtain quickly and, by the time they are available, may not be very helpful.

Steps in diagnosis

Ask the following questions:

- Has there been sufficient blood component replacement after excessive blood loss resulting in a hypo-coagulable state? Is there an ongoing acquired coagulopathy?
- Is the patient acidotic and/or hypothermic? These may occur during surgery and predispose the patient to bleeding. Hypothermia slows the enzymatic reactions of the coagulation cascade, decreases platelet counts and function, and stimulates fibrinolysis. Acidosis impedes fibrin polymerization and ∴ clot strengthening.
- Is the patient anaemic? Anaemia (haematocrit <30%) further aggravates a bleeding diathesis, as RBCs are thought to assist in platelet margination against an injured vessel wall. Conversely, massive RBC transfusions lower plasma Ca^{2+} concentration, inhibiting enzymatic reactions of the coagulation cascade.
- *Is it a drug-induced coagulation problem?*
  - Anticoagulants and antiplatelet agents are often used in vascular surgery to balance the haemostatic system, when pathology is present, or when necessitated by a procedure.
  - They can also introduce complications when trying to evaluate and diagnose a coagulopathic patient. A complete medication history is essential when evaluating a post-operative coagulopathy.
  - Heparin overdose, re-warfarinization or platelet dysfunction 2° to anti-platelet therapy may be present. If overdose of heparin is identified, protamine may be used to reverse, but is rarely necessary due to the short half-life of unfractionated heparin. Excessive warfarin can be reversed with PCC or, if unavailable, FFP transfusion.
- *Is there an auto-immune acquired coagulopathy?*
  - Although uncommon, autoantibodies directed against a specific coagulation factor may lead to severe bleeding disorders. The most common coagulation factors involved are factors V and VIII.

Box 11.4 Targets in the patient bleeding after vascular surgery

Therapeutic targets in the bleeding patient

- *Haemoglobin:* >90g/L
- *Platelets:* >75 000
- *Fibrinogen:* >2g/L

Calcium and magnesium

- Maintain within normal range
- Normothermia
- Normocapnea
- Normal pH
- Permissive hypotension (systolic pressure 80–90mmHg)

Table 11.6 The 4T score for heparin-induced thrombocytopenia (HIT). HIT is unlikely if the total score is <3, highly likely if the score is 6–8, and of intermediate probability if the score is 4–5. If a patient has a score of 4, then heparin should be stopped and an alternative drug substituted, pending further investigations for HIT

4Ts category	2 points	1 point	0 points
Thrombocytopenia	Platelet count fall >50% and platelet nadir 20	Platelet count 30%–50% or platelet nadir 10–19	Platelet count fall <30% or platelet nadir <10
Timing of platelet count fall	Clear onset days 5–10 or platelet fall -1 day (prior heparin exposure within 30 days)	Consistent with days 5–10 fall, but not clear (eg, missing platelet counts); onset after day 10; or fall -1 day (prior heparin exposure 30–100 days ago)	Platelet count -4 days without recent exposure
Thrombosis or other sequelae	New thrombosis (confirmed); skin necrosis; acute systemic reaction postintravenous unfractionated heparin bolus	Progressive or recurrent thrombosis; non-necrotizing (erythematous) skin lesions; suspected thrombosis (not proven)	None
Other causes of thrombocytopenia	None apparent	Possible	Definite

- The mechanism by which autoantibodies are generated is still unclear, but current data suggest that immune exposure to non-human coagulation proteins is a key step in the process. Repeated vascular surgery and exposure to animal products contained in bio-glues may predispose to auto-immune acquired coagulopathy.

Transfusion management

- Transfuse packed red blood cells to maintain Hb > 90g/L.
- *Correct hypofibrinogenaemia:* Normal fibrinogen concentrations are usually reassuring, although low values should be corrected. In emergency bleeding concentrations should be maintained at >2g/L by transfusion of either FFP or cryoprecipitate. Cryoprecipitate corrects fibrinogen more quickly than FFP and may also provide other useful factor replacement of factor IX and XII. Fibrinogen concentrate is more rapid and effective than both FFP or cryoprecipitate, but is not licensed for such use in the UK.
- *Maintain the platelet count >75 000:* consider platelet function, which may be impaired even when platelet count is normal.
  - Platelet action can be altered greatly by antiplatelet drugs, aspirin, and heparin. Platelet transfusion will aid clotting, but large doses may be required. Discuss with haematologist when platelet dysfunction is suspected or suggested by POC testing.
  - Consider the (rare) possibility of heparin-induced thrombocytopenia (HIT) (see Table 11.6)
  - Laboratory testing for HIT is not available quickly and likelihood of HIT can be assessed using 4 'T' pre-test assessment.
- Give tranexamic acid (1g given by slow IV bolus followed by a further 1g by infusion over 8h). This is indicated in all cases of massive haemorrhage to prevent excessive fibrinolysis.
- The use of Factor VIIa not justified (📖 Management of major haemorrhage, p. 244) and efforts need to be focused on the correction of low fibrinogen and platelet concentrations.

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Distal limb ischaemia

Acute limb ischaemia

Acute limb ischaemia is defined as the sudden reduction (<2 weeks duration) in the perfusion of a limb, resulting in a threat to limb viability. Acute ischaemia is often severe, because in contrast to chronic ischaemia, no collateral vessels have developed.

Aetiology

- *Acute limb ischaemia after vascular surgery most commonly caused by:*
  - Acute rupture of a pre-existing atherosclerotic lesion within the limb vasculature and thrombosis in situ.
  - Emboli from the heart (AF, cardiac valvular vegetations, HF). Incidence of cardiac emboli has decreased as incidence of rheumatic fever has declined and anticoagulation for AF has become common.
  - Embolization of thrombus from a more proximal source, usually proximal arterial atheroma. This can occur after more proximal vascular surgery (AAA repair or lower limb revascularization), especially if there is a prolonged period of low cardiac output after surgery.
- *Less common causes are:*
  - Vascular trauma or dissecting aneurysm.
  - Thrombosis of peripheral arterial aneurysms (especially popliteal).
  - Iatrogenic arterial damage (puncture for vascular access or vascular surgery) causing dissection or embolization from the site of arterial injury and resulting in distal ischaemia.

Clinical assessment

- The majority of patients with acute limb ischaemia have co-existing generalized cardiovascular disease.
- Clinical assessment of the patient with suspected acute limb ischaemia is the most important aspect of their subsequent management.
- The classical hexad of pain, pulselessness, paralysis, pallor, perishingly cold, and paraesthesia is useful as an *aide memoir*, but all signs are variable and subject to clinical interpretation.
- It is vital to distinguish whether the patient needs immediate intervention to save the limb, or whether investigation and management can be safely performed on an urgent basis.
- The most useful discriminators to aid this decision are sensory and motor loss: **immediate action is needed if sensory and motor loss are present in a patient presenting acutely with a pulseless limb**.
- In those patients where immediate intervention is required it is important to distinguish between salvageable and non-salvageable limbs:
  - Signs of irreversible ischaemia are fixed skin mottling and tense muscle compartments. This is usually associated with delayed presentation (>12h).
  - In the absence of definitive signs of irreversible ischaemia, painful passive muscle movements or tenderness over the muscle groups in

the affected limb suggest muscular inflammation or possible necrosis. In these indeterminate cases, possibility of limb salvage can often only be assessed at the time of surgery by visual inspection of the muscles through fasciotomy incisions.

- Revascularization of unsalvageable ischaemic limbs should not be attempted, because of the dangers of reperfusion syndrome. When limb is not salvageable (because ischaemia is irreversible) or because patient is unfit for prolonged attempts at revascularization, the only treatment option is amputation.
- In some cases of acute lower limb ischaemia, especially in patients with severe or terminal co-existing medical conditions, palliative care may be appropriate

Immediate management and investigation

- The immediate management of patients with acute limb ischaemia is resuscitation, often performed concurrently with assessment, and surgical or radiological intervention.
- When indicated, surgical, or radiological intervention should performed without delay. Appropriate laboratory or other investigations should be sent, but intervention should not be deferred, whilst awaiting the results of these. Specific pre-operative investigations in patients with acute limb ischaemia should focus on those needed in order to plan surgery. Any acute physiological derangements should be addressed at the same time as the planned intervention.
- Most surgeons will not give systemic heparin before emergency revascularization procedures, but will ask for it to be given during surgery. However, this practice can vary between surgeons.
- When the cause is clearly an embolism, urgent embolectomy should be performed without delaying for pre-operative investigations.
- However, the cause is often less clear and the surgical team may wish to obtain further imaging before intervention. In trauma, imaging is essential to determine whether vascular insufficiency is due to arterial damage or compression from bony injuries.
- Depending upon local resources, imaging may include percutaneous arteriography, cross-sectional angiography (CT or MRI) or duplex imaging, all of which can be performed with minimal delay. Intra-operative angiography is another option.
- Angiography in the operating theatre (often used to assess the end result of vascular surgical interventions) is the imaging modality of choice for many vascular surgeons and radiologists in acute ischaemia.

Advantages of angiography

- Delays resulting from transfer to the radiology department for investigations are minimized.
- Imaging is available directly in the operating suite theatre and can be used to plan interventions and assess their results.

Principle disadvantage is lower quality images produced by mobile image intensifiers compared with those available in radiology department.

Interventions for acute limb ischaemia

Interventional radiology

Interventional radiological procedures are rarely used in isolation for acute limb ischaemia, but are often used as adjuncts to surgery. Aspiration of thrombus that has formed *in situ* is feasible, followed by angioplasty of the culprit lesion. They are often performed in the radiology department with involvement of the anaesthetist.

Embolectomy

- Embolectomy will usually be attempted in all cases of acute limb ischaemia except for those due to thrombosed peripheral aneurysms.
- Embolectomy is a good procedure for patients without pre-existing peripheral vascular disease, in whom the retrieval of large emboli and associated thrombus is usually successful.
- The usual sites of embolectomy are femoral, popliteal and brachial.
- Embolectomy has become less common as atrial fibrillation has become better recognized and treated.
- Embolectomy can be performed under local or regional anaesthesia in compliant patients. In these cases, continued clinical monitoring of the patient during the procedure is essential.

Bypass

- Bypass surgery is usually required if:
  - Embolectomy is unsuccessful.
  - There is inadequate arterial inflow.
  - There is arterial trauma or thrombosed aneurysms (usually popliteal).
- The required bypass surgery depends on the affected vessel, but most commonly will consist of restoration of inflow to a femoral artery (ilio-femoral bypass/cross-over or femoro-femoral cross-over) or femoro-popliteal/distal graft.
- Limb bypasses will often require the harvest of a suitable venous conduit, usually the long-saphenous vein.
- General or regional techniques may be used, though the prior administration of heparin or thrombolytic drugs often precludes regional anaesthesia. For these reasons GA is usually preferred.

Thrombolysis

Thrombolysis is usually only used as an adjunct to surgical and radiological attempts at revascularization.

Fasciotomy

- Tissues maintain metabolic function during a period of ischaemia via anaerobic metabolism, resulting in the accumulation of xanthine. Upon reperfusion the addition of oxygen results in the conversion of xanthine to hypoxanthine and the production of free radicals. If this free radical production overwhelms natural anti-oxidant mechanisms tissue damage may occur (reperfusion injury). This damage will be in addition to the direct tissue damage caused by ischaemia.
- Resulting inflammation within the restricted osteo-fascial compartments of the limbs can cause compartment syndrome.

- The treatment compartment syndrome is prophylactic fasciotomy.
- This is frequently performed after embolectomy and is a common reason for requiring a general anaesthetic at the end of an embolectomy under local anaesthesia.
- Radiological contrast agents will often be used during interventions for acute ischaemia, often in larger doses than those used for elective bypass surgery. Contrast-induced nephropathy is unusual, but renal function should be monitored closely during and after surgery.

Anaesthesia for interventions for acute lower limb ischaemia

- Acute limb ischaemia should be treated as an emergency. Patients commonly have co-existing medical conditions in particular cardiovascular disease. These should be assessed, appropriate laboratory investigations performed (including recent ECG and CXR if indicated). These can be expedited quickly and not allowed to delay definite treatment.
- Acute limb ischaemia is painful and IV opioids are often required.
- The anaesthetist should be prepared to treat ongoing conditions urgently (AF, HF, acute electrolyte disturbances), whilst surgical treatment is ongoing.
- Embolectomy can be performed under LA or GA.
- If acute lower limb ischaemia occurs in a post-operative patient (e.g. after AAA surgery), where a working epidural is in place, RA is appropriate

Local anaesthesia

- Even when a LA technique is performed (usually local infiltration by the surgeon), the anaesthetist should be present in theatre to monitor the patient, and give any additional analgesics or sedative drugs (e.g. midazolam 0.5mg increments or low-dose propofol infusion) as required
- Oxygen 4L/min should be given by mask or nasal cannulae.
- Monitoring should include ECG, NIBP, and SpO_2.
- In some cases conversion to GA may be needed. This may occur if the patient becomes intolerant or a more extensive surgical procedure is required.

General anaesthesia

- GA may be required for fasciotomy, bypass, or prolonged procedures, or if the patient cannot tolerate the procedure under LA.
- A balanced technique is suitable, paying attention to maintaining patients temperature, cardiovascular stability and fluid requirements.
- Anaesthetic technique and monitoring are dictated by the patient's condition and procedure performed.
- Fasciotomy and bypass procedures can be painful; LA infiltration or nerve blockade with or without morphine may be useful during and after surgery.

Reperfusion syndrome

- With all revascularization procedures for there is the potential for the development of post-procedural systemic reperfusion syndrome. The highest risk is in those patients undergoing delayed revascularization.
- Systemic reperfusion syndrome is a result of the same mechanisms that cause local reperfusion injury. Moderate or severe tissue damage caused by ischaemia or localized reperfusion injury can result in the release of toxic by-products into the systemic circulation. This can cause hyperkalaemia and lactic acidaemia, which may lead to myocardial depression, cardiac arrhythmias, or cardiac arrest.
- Reperfusion syndrome is a cause of SIRS and multiple organ failure. In addition to renal impairment caused by generalized systemic inflammation renal tubular damage can occur due to the release of myoglobin into the systemic circulation as a result of muscle damage. Preventative treatments for myoglobin-induced renal injury, such as forced alkaline diuresis are often used, although there is limited evidence for their benefit. Maintenance of circulating volume, and monitoring of urine output and renal biochemistry are important (see 🕮 Perioperative renal protection, p. 236). In some cases, post-operative renal replacement therapy may be needed for AKI.
- When significant reperfusion injury is anticipated or occurs, the anaesthetist should be prepared to treat life-threatening hyperkalaemia or cardiac arrhythmias. Post-operative monitoring in a critical care environment should also be arranged.

Further reading

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Critical care management

Infrastructure, organization, and planning

A high quality service requires public health preventive strategies, surveillance, assessment, multidisciplinary decision making, optimization and excellent surgery, anaesthesia, Critical Care, ward care and follow-up. The infrastructure needed for Critical Care includes:

- Level 2 and Level 3 beds, ideally situated together and having close liaison with PACU and step-down areas
- Sufficient funding and staffing to enable appropriate bed occupancy which can accommodate elective and emergency admissions, without compromising the care of other patients. Discharges from ICU at night carry significant risks to the patient and should be avoided
- 24/7 availability of skilled doctor(s)
- High quality clinical, teaching environment
- Routine participation in (national) audit
- Close liaison between anaesthetic, surgical and Critical Care services. This is essential for patient selection, information, consent, management of patient's expectations and planning
- Pre-emptive Critical Care surveillance systems (Critical Care Outreach)
- Information for patients and their relatives (verbal and written) on surgical procedures, anaesthesia, post-operative care (pain control, RA techniques, potential complications, respiratory support, sedation, potential postponement of surgery and capacity limits, step down arrangements, the role of families in decision-making).
- Appropriate facilities for relatives including distinct areas for private discussions or communications.

Indications for admission to critical care after vascular surgery

Indications for admission relate to the type, complexity, and duration of surgery, co-existing medical conditions, or complications from surgery or anaesthesia, or (commonly) a combination of the above. Patients may be admitted for different levels of care:

- *Level 2 care:* preoperative optimization, extended post-operative care or single organ support. Examples:
  - Elective AAA
  - Abdominal or thoracic EVAR (if co-existing medical conditions).
  - Carotid surgery (if medical problems including difficulty with blood pressure control).
  - Peripheral vascular reconstruction (if co-existing medical conditions).
  - Major amputation (if co-existing medical conditions).
- *Level 3 care:* advanced respiratory support or two organ support (respiratory or cardiovascular plus another system). Examples:
  - Elective AAA.
  - Emergency AAA.
  - Thoracic AAA.

- Patients are very occasionally admitted for planned palliative care (if already unconscious and receiving organ support including artificial ventilation).

Main considerations

- AAA and TAA are major abdominal surgical procedures requiring conventional post-operative care. Particular risks are: coronary, cerebrovascular and renal complications.
- Problems mostly relate to complicated, emergency, or TAAA.
- The main issues are:
  - Cardiovascular function.
  - Respiratory function.
  - Maintenance of circulating volume and coagulation.
  - Renal function.
  - Maintenance of body temperature.
  - Detection and prevention of stroke/ spinal cord injury.
  - Pain control.
  - Nutrition.

Referral to critical care

May be from the Emergency Department, ward or theatre. Early, comprehensive liaison is crucial. On referral, patients may be:

- Extubated after adequate recovery.
- Intubated and ventilated:
  - If there is recent, current or potential instability.
  - To expedite theatre throughput.
- The patient is usually accompanied by anaesthetist, nurse, nurse/ operating department practitioner (ODP).

Handover

Handover should address:

- *Co-morbidities, functional capacity:* medications—β-blockers, statins, antiplatelet agents, and allergies.
- *Operative & anaesthetic history:* surgical difficulties, bleeding, re-explorations, redo of anastomoses, clamp position, and times.
- *Anaesthesia techniques:* regional techniques & effectiveness; agents, analgesia.
- Haemodynamics, myocardial ischaemia, vasoactive agent boluses or infusions, cardiac output data.
- Ventilation, urine output, diuretics.
- Bleeding, Hb, coagulation history (thromboelastography), blood loss, cell salvage, transfusion, fluid balance.
- Acid-base, electrolyte, and glucose status.
- Antibiotic prophylaxis.
- Analgesia/sedation plan.

Assessment on admission

Read the notes (current and old). Specifically assess:

- *Airway competence or tube size, length, fixation:*
  - NGT presence, location, function.
  - *Adequate sedation/analgesia*—ensure endotracheal tube is secure and correctly positioned.

- *Breathing:* ventilation, oxygenation, impact of pain, cough.
- *Circulation:*
  - Heart rate, rhythm, BP, perfusion, skin temperature, capillary refill time, pulse oximeter signal.
  - Limb perfusion, pulses, muscle compartments.
  - Cardiac output data and device.
  - *ECG:* on monitor and 12-lead. Look specifically for arrhythmias or evidence of ischaemia.
- *Disability:*
  - Cerebration and mentation or quality of sedation, analgesia.
  - Focal or lateralizing neurology.
  - Limb motor, sensory function.
- *Exposure:*
  - Type of incision.
  - Skin perfusion, rashes.
  - Line sites, integrity, length, and position on CXR.
  - Temperature.
- *Other:*
  - ABGs, glucose, Hb, urea, electrolytes (calcium, magnesium), albumin, LFTs, ECG, CXR
  - Drugs, microbiology, nutrition
  - Sociological features/relatives' review

Analgesia and sedation

- Ensure adequate immediate post-operative analgesia.
- Adequate analgesia is crucial. Pain is distressing and has several potential consequences:
  - Prevents good coughing, expectoration (risk of hospital acquired pneumonia).
  - Prevents mobilization, increasing length of stay.
  - Increases the risk of delirium.
  - Induces tachycardia (ACS risk).
  - Reduces appetite, nutritional intake.
  - Hence tends to increase mortality risk.
- Options for analgesia are nerve blocks, regional blocks, parenteral opioids and oral analgesics.
- Paravertebral blocks (retroperitoneal, lateral incisions), transversus abdominis plane (TAP) blocks.

Epidural analgesia: gold standard and almost routine for AAA (?consider inserting post-operatively in TAAA)

- *Advantages:* potentially perfect analgesia, avoids above complications, reduce peri-operative blood loss, DVT risk, nausea, constipation, confusion, and delirium.
- *Aim is adequate analgesia:*
  - Sympathetic block, BP support may be needed.
  - Epidural block assessed, monitored, optimize flow rate, leave unaltered until discontinued to oral analgesics (compare morphine needs).

Potential epidural-related problems

- *Epidural-related hypotension:* adequate vascular filling (without overloading patients), vasopressor infusion, e.g. metaraminol 2–10mg/h or noradrenaline (0.05–0.5mg/h).
- Plan epidural removal around the timing of DVT chemoprophylaxis.
- Difficulty assessing neurological signs in the legs if there is a risk of spinal cord injury:
  - Profound motor block occasionally occurs even with 0.125% levobupivacaine.
  - This mimics epidural haematoma and cord ischaemia.
- *If motor blockade occurs:*
  - Stop epidural infusion.
  - Normalize coagulation.
  - If no recovery within 2h, consider MRI scan (exclude haematoma, consider evacuation).
- Consent issues for epidurals inserted in critical care:
  - Normal coagulation.
  - Awake or 'asleep/sedated'.
  - Recognizing patient preferences, only with consent.
- *PCA considerations:*
  - Not all patients cope with PCAs (frail elderly, cognitively impaired, confused patients).
  - Simple (nurse supervised) morphine infusions can be used with appropriate monitoring.
  - IV paracetamol supplementation is useful.
- *Oral analgesics (Fig. 11.2):*
  - Paracetamol, tramadol, codeine.
  - *NSAIDs:* use with great caution or avoid completely.

Respiratory support

- Continue artificial ventilation until patient is stable.
- Extubate when 1° problems resolved and FiO_2 <0.4, PEEP <5, adequate 'huffing & puffing', good mentation, cardiovascular stability, nutrition, metabolism/glucose, analgesia.
- *Beware actual or potential ALI:* use 'Adult Respiratory Distress Syndrome' ventilator strategy:
  - Higher frequency, low tidal volume (6–8mL/kg lean body weight), higher PEEP.
  - Minimizes inflammation, frictional, torsional forces causing interstitial air spaces, pneumothoraces (barotrauma, volutrauma) generating ALI/ARDS.

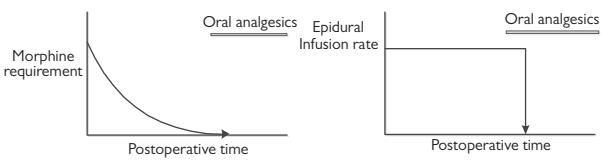

Fig. 11.2 Conversion to oral analgesia after epidural or IV morphine.

- Injured lungs (ALI/ARDS) function as 'baby lungs' (small).
- Consider oscillation in ALI/ARDS.

Cardiovascular function

Risk of acute coronary syndrome

- Avoid pain, haemodynamic stressors.
- *Minimize omission of cardiac drugs:* continue β-blockers if critically dependent, statins.

Leg perfusion, pulses and compartment syndromes

- Risk factors are long clamp times, emboli of clot and cholesterol, pre-existing PVD, excess leg oedema.
- There may be arterial clot and a white ischaemic foot (see 📖 Distal limb ischaemia, p. 499) or 'trash foot', which has variable blotchy areas, coloured, white and purple, and signifies small vessel occlusion.
- Compartment syndrome occurs when a closed (leg or arm or abdomen) compartment pressure rises, compromising blood flow. The presence of a pulse does not exclude this—the artery may be in another compartment, and ischaemia occurs before complete flow cessation. Avoid leg elevation, which reduces compartment arterial pressure.
- *Leg assessment:* pulses, temperature, perfusion, capillary refill time (normal <2s), Doppler pulses, limb oximetry; fullness, firmness of leg muscles.
- Transducer measurement of compartment pressure may confirm diagnosis if >25–30mmHg.
- *Diagnosis is clinical:* if there is a clinical suspicion decompression with fasciotomy should be performed immediately (within the hour).
- Rhabdomyolysis may occur, but malperfusion curtails this and delays any increases in creatine kinase.
- Myoglobin tests are commonly misleading and unreliable.
- Rhabdomyolysis causes renal injury: theoretically large fluid volumes (10–12L/day) help flush the kidneys and dilute myoglobin/Tamm-Horsfall protein precipitants. However, AKI is usually multifactorial. The following considerations apply:
  - Massive fluid volumes are often detrimental to the vascular patient.
  - Mannitol and/or, bicarbonate are not effective treatments.
  - The diagnosis is often made late so 'conventional' treatments to avoid renal failure may be futile and/or risky.
  - Muscle sequesters fluid, worsening compartment pressure and the circulation needs (some) volume support.
- Hence, a pragmatic approach to rhabdomyolysis is often best. Start haemofiltration, avoid excess IV fluids, facilitate early decompressive surgery for compartment syndrome.

Gut function and nutrition

- Episodes of diarrhoea early after surgery are common.
- *Gastric paresis is common, related to:*
  - The effects of abdominal surgery.
  - Duodenal handling and oedema (especially with ruptured AAA)
  - Effects of opioid medications

- Monitor NG volumes 4-hourly; if <250mL/4h, hypocaloric feeding may be useful, if patient is likely to remain ventilated for some time.
- Enteral feed is generally preferred as it promotes gut activity, motility, and microbiological GI barrier function. Increase volumes gradually according to 4-hourly gastric residual volumes.
- However, if enteral feed is unsuccessful by 3–5 days, start TPN. Build up *slowly*, avoiding hyperglycaemia.
- Immediate and late diarrhoea may occur.
- *Bowel ischaemia:* this is a severe complication with high mortality. Risk factors are AAA surgery, (especially ruptured AAA), prolonged shock or hypotension. Maintain a high level of suspicion if abdomen is distended, persistent ileus, unexplained lactic acidaemia (or persistent acidaemia despite CVVH). Consider early CT contrast-enhanced scanning or colonoscopy.
- *Abdominal compartment syndrome (AbCS):* Risk factors are: massive bleeding, retroperitoneal haematoma, bowel handling/displacement, excessive fluid loading. All these raise intra-abdominal pressure and compromise splanchnic and renal blood flow. Girth measurements are worthless in AbCS and intra-abdominal pressure (IAP) should be monitored (see 📖 Intra-abdominal pressure monitoring, p. 272).
- Intra-abdominal hypertension (IAH) is defined as IAP >12mmHg (Table 11.7).
- Abdominal compartment syndrome (ACS) is defined as IAP >20mmHg or abdominal perfusion pressure (MAP-IAP) <50mmHg with > one organ dysfunction, not previously present.
- ACS compromises renal, hepatic, gut blood flow, impairs respiratory function, reduces venous return and cardiac output, increases intracranial pressure.
- Management should aim to keep APP >60mmHg. This involves regular monitoring of IAP, optimizing cardiovascular function, correction of +ve fluid balance, percutaneous drainage of abdominal fluid collections, decompression of bowel contents, pain control, neuromuscular blockade in sedated patients. If these fail, decompressive surgery with laparostomy is needed.
- *Laparostomy usually reversed when IAP settles:* long-term laparostomy carries considerable morbidity

Renal perfusion and urine output

- Physiological oliguria is to be expected as part of the surgical stress response.
- Maintenance of renal function and urine output requires adequate perfusion pressure, volume, and flow (cardiac output), and the absence of renal tract obstruction or nephrotoxic drugs.
- A temporary decline in renal function is very common after aortic surgery, especially emergency AAA or TAAA. Diuretics, mannitol, dopamine have no overall benefit on renal function.
- Disadvantages of diuretics include production of an unnecessary polyuria, potential haemodynamic instability because of hypovolaemia, hyponatraemia (impacts on brain oedema, cognitive function), hypokalaemia (potential for arrhythmias).
- The management of post-operative oliguria is detailed in 📖 Postoperative oliguria, p. 490.

Table 11.7 Grading of Intra-abdominal hypertension

Grade	IAP mmHg
I	12–15
II	15–20
III	21–25
IV	>25

Reprinted from Surgical Clinics, 76, 4, Jon M Burch et al., 'The Abdominal Compartment Syndrome', pp. 833–842, Copyright 1996, with permission from Elsevier.

Neurology

- Monitor neurology for stroke, TIAs, epidural haematoma, cord ischaemia, paraplegia, nerve injury, meningitis (if CSF drains).
- Beware sympathetic block after epidural anaesthesia, prevent and/or treat hypotension.
- CSF drains (📖 Cerebrospinal fluid drainage, p. 264) should be levelled to 10mmHg: ensure slow drainage to minimize stress on stretched dural veins.

Infection control and ICU bundles

Routine policies include:

- Unless known to be –ve, take MRSA swabs, commence chlorhexidine wash, nasal mupirocin.
- Isolate MRSA-positive patients.
- *ICU bundles:*
  - Nurse all patients head-up 30–45° to minimize regurgitation, aspiration, pneumonia risk.
  - *Gastric protection:* H_2 antagonist or PPI whilst receiving artificial ventilation.
  - DVT prophylaxis.
  - Sedation break daily (unless contraindicated).
  - Chlorhexidine oral gel.
- Adherence to local antibiotic policies.
- Antibiotic prophylaxis is usually one intraoperative dose, although local microbiological guidelines should be followed. Strict in-dwelling catheter discipline (insertion, handling) required.
- Enteral feeding at 24h (unless contraindicated/imminent extubation): titrated to increase feeding rates slowly.
- Mobilization as early as is feasible.

Temperature

- Fever is a *physiological* response and sign, has a survival value, abolished only with good reason, e.g. brain/cord injury.
- Post-operative early hypothermia is common and usually avoidable.

Fluids, blood and acid base status

- Re-infuse any cell saver blood if not done so in theatre.
- *Beware the potential for bleeding:*
  - Coagulation studies (📖 Point of care coagulation monitoring, p. 319), especially POC testing (including thromboelastography, TEG) allows titration of coagulation support.

  - Residual effects of intraoperative heparin wane rapidly and reveral with protamine is rarely needed (beware hypotension).
  - Consider massive haemorrhage guideline if appropriate (📖 Management of major haemorrhage).
  - Use blood, FFP, and platelets together, and early.
  - Cryoprecipitate may be required with each 8–10U.
  - If there is a persistent volume requirement, falling Hb or Hct, and/or increasing inotrope/vasopressor usage after reasonable coagulation support or correction of coagulopathy, the patient is likely to be bleeding. Call senior colleagues and vascular surgeon and expedite and early return to theatre.
- *Consider thresholds for blood transfusion:*
  - Bleeding or arteriopathic: usually 90–100g/L.
  - Not bleeding, not arteriopathic: usually 70g/L.
- Ensure adequate vascular volume and cardiac output by:
  - Clinical evaluation.
  - CVP, urine output, bicarbonate/base excess are often unreliable indices of the adequacy of volume resuscitation; arterial pressure should be interpreted with care. Passive leg raising is a useful sign.
  - Consider cardiac output monitors with indices of preload (most need sinus rhythm, full IPPV).
- If the patient is thought to be hypovolaemic, give fluid boluses (Hartmann's solution or colloid) 200–250mL until:
  - There is no further increase in cardiac output, stroke volume, corrected flow time (FTc, oesophageal Doppler), global end diastolic volume index (GEDVI with PiCCO).
  - Systolic pressure variation (SPV), pulse pressure variation (PPV), stroke volume variation (SVV) (preload indices) approach <10%.
  - There is no increased blood pressure with passive leg raising.
- Beware that excessive fluid administration is detrimental and associated with increased mortality because it causes peripheral oedema, gut atonia, ileus, nausea, impaired wound healing, increased pulmonary water, may impair gas exchange and predispose to chest infection.
- *Perioperative fluid guidelines exist (GIFTASUP):*
  - Normal hydration requirements 60, 80, 100mL/h in small, medium, large patients (temperate climates).
  - Humans drink water-based, not salty (or normal saline) solutions.
- The normal response to surgical stress includes a surge in concentrations of cortisol, ADH, aldosterone. These result in:
  - Sodium, water retention, appropriate antidiuretic state, concentrated, high osmolality (dark), low volume urine (hence, normal fluid needs reduced in stressed, perioperative state).
  - This is physiological oliguria, not (*per se*) an indication of hypovolaemia or malperfusion.
  - It is not an indication to 'chase the urine output' to produce 'normal' volumes.
  - Protons are generated by the dissociation of water (Fig. 11.3). [H_2O] normally = 55.6mol/L [H+] = 40nmol/L. The equilibrium constant (k1) is influenced by various factors

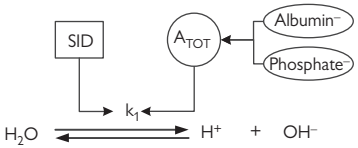

Fig. 11.3 Dissociation of water. SID = strong ion difference. A_{TOT} = total weak acids.

- Metabolic acidosis (falling bicarbonate, a *process* tending to cause a metabolic acidaemia) must be generated by either:
- A rising chloride concentrations, hyperchloraemia (i.e. a smaller difference between sodium and chloride, approximates strong ion difference). This commonly caused by saline administration, which has an unphysiological chloride load
- A high anion gap (AG) = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$The main causes are:
  - lactic acidosis
  - ketoacidosis (diabetic, starvation, alcoholic)
  - renal failure (residual acids normally eliminated by the kidney)
  - poisons – methanol, ethanol, ethylene glycol, aspirin
- The bicarbonate in Fig. 11.4 is the numerator of the Henderson Hasselbalch equation (Fig. 11.5).
  - Since total [+ves] = total [–ves], then $[HCO_3^-]$ can be replaced by the cations$^-$ – anions$^+$ in the twin towers, left in Fig. 11.4.
  - Simplify, by ignoring K^+, Ca^{2+}, Mg^{2+} (small compared with Na^+), derives an equation showing pH is related to the four subcomponents 1, 2, 3, 4.
  - For complex details of chemistry, the dissociation of water is influenced by the difference in the strong (**fully dissociated**) ions (positives – negatives) (SID)

    $SIDa = Na^+ + K^+ + Ca^{2+} + Mg^{2+} - Cl^- - Lact^-$

    From Figs 11.4 and 11.5, this is equivalent to:

    $HCO_3^- + Alb^- + Phos^{2-} = SIDe$

    (components normally easily measured), thus SIDa = SIDe for small XA^- otherwise:

    $SIDa = SIDe + XA^-$

Metabolic component (HCO_3^-) is mirrored by base excess. Total base excess is sub-quantified by the 4 components (Fig. 11.5)

1 $(Na^+ - Cl^- - 38)$
2 (Lactate) × –1
3 (42 – albumin) × 0.25 (low albumin means positive value, a metabolic alkalosis)
4 XA^- effect = total base excess –1, –2, –3 above (titratable acids and poisons)

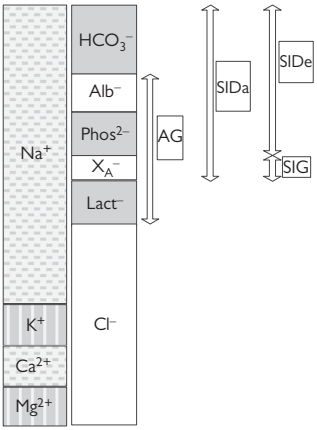

Fig. 11.4 Total anions and cations.

$$pH = pK + Log \left[\frac{HCO_3^-}{P_{CO2}} \right]$$

$$pH = pK + Log \left[\frac{Na^+ - Cl^- - Lact^- - Alb^- - Phos^{2-} - XA^-}{P_{CO2}} \right]$$

Fig. 11.5 The Henderson Hasselbalch equation can be substituted with anions and cations, illustrating how strong ion difference, (including both chloride and lactate), albumin, other (renal) acids form part of Henderson Hasselbalch equation and directly influence. See text for further explanation.

- Blood or plasma loss may be related to: bleeding, ooze (e.g. retroperitoneal, tissue, and bowel oedema), global capillary leak from surgical trauma and infection, NG losses. Fluid is sequestered outside the circulation (inaccurately called third spacing)—produced from blood/plasma (high Na^+ content). Eventually all is reabsorbed leading to polyuria. This is a common sign of healing/recovery, not diabetes insipidus.
- *Fluid management:*
  - *Titrate fluid boluses*—ideally to dynamic cardiovascular targets/end points.

  - Most high-Na^+-containing fluids (especially saline and colloids) also contain disproportionate amounts of chloride. Large volumes generate a hyperchloraemic state and metabolic acidosis (reduces bicarbonate, base excess becomes more negative).
  - This metabolic acidosis indicates chloride overload, not hypovolaemia.
  - Calculating ($Na^+ - Cl^- - 38$) gives an estimate of the contribution of the sodium-chloride difference to the base excess: it is often a negative value. If this is a major fraction of total base excess, no specific treatment is needed, apart from minimising further chloride administration. Further infusion of high volumes of high chloride containing solution to treat the low bicarbonate (misinterpreted as hypovolaemia), simply worsens the process, with increasing hyperchloraemia.
- Albumin concentrations commonly decrease after major vascular surgery. This is multifactorial and relates to:
  - Dilution by infused fluids.
  - Blood losses, increased vascular permeability (major surgery, cross clamping, sepsis).
- Albumin may fall by 25–50% in 24–48h. This has major effects on:
  - Pharmacodynamics and pharmacokinetics of many drugs.
  - Production of metabolic alkalosis, tending to raise bicarbonate and base excess. It may disguise the magnitude of a metabolic acidosis.
  - (42 – albumin) × 0.25 defines the magnitude of a hypoalbuminaemic metabolic alkalosis and equate to the change in base excess.
- Ischaemia of organs or limbs or global inadequate cardiac output causes cellular energetic failure, mitochondrial dysfunction, rise in lactate (lactic acidosis) contributing to lactic acidaemia.
- Treatment of metabolic acidosis should be directed to treat the cause. In AKI, there is a rise in acids, normally excreted in the urine. These can be eliminated by haemodialysis or haemofiltration, but although renal replacement therapy can normalize renal acids, minerals, and Cl^-, it does not solve cellular energetic failure (lactic acidosis).

Thromboprophylaxis

- There is a high risk of venous thrombo-embolism after major vascular surgery.
- The risks may be decreased by the use of thrombo-embolic stockings (TEDS), mechanical calf or foot arch pumps, and chemical prophylaxis. TEDS or mechanical calf pumps should not be used if there is severe peripheral arterial disease or low perfusion.
- *Chemical prophylaxis:*
  - Low-molecular weight heparins or unfractionated heparin; the latter may be preferred in renal impairment (creat >200, eGFR <30mL/min).
  - Beware risk of heparin-induced thrombocytopenia and thrombosis (HITT). Occurs in 2–3% patients exposed to heparin >4 days and 0.8% patients on low-molecular weight heparins.
  - Type 2 HITT is rapid thrombocytopenia (platelet count reduces to 50–80 within 24–48h and normalizes when heparin stopped. Typically occurs 5–10 days after starting heparin and causes major

arterial or venous white (platelet-rich) clot due to antibodies to platelet factor 4 (PF4).
- Management is to exclude other causes of thrombocytopenia, confirm diagnosis (send for antibodies), but in the meantime stop heparins, and use alternative mechanical or chemical prophylaxis

Drug management
- Continue normal medication if possible, including statins.
- Continue beta-blockers where critical, oral, or IV.
- Re-write drug chart.

Follow-up clinic
- Follow-up arrangements for patients with long critical care admission (perhaps > 3 days) may be offered.
- Prolonged critical illness may cause major morbidity: changes in sight, hearing, skin, weight loss, anorexia, malaise (prolonged), breathlessness, pain, insomnia, loss of libido, impotence, cognitive change, relationship difficulties, psychological problems.
- Convalescence and recovery may take 12–24 months after prolonged admissions.

Thoracic aortic surgery

Special considerations apply to the patient after TAAA repair.

Study the surgical and anaesthetic technique
- Clamp site, sequence, duration.
- *Spinal cord/visceral oxygenated perfusion:* available methods include:
  - Shunting of aorta to distal aorta/iliac/femoral artery, or axillo-femoral bypass.
  - Partial left heart bypass—pulmonary vein to pump to distal aorta/iliac/fem.
- *Cardiopulmonary bypass:* +/– deep hypothermic arrest +/– anterograde/retrograde brain and visceral perfusion.
- Re-implantation sequence/timing for intercostals, lumbar radicular, renal/visceral aortic branches.
- CSF drain use, CSF pressures during surgery.
- Cardiovascular measurements during surgery.
- Use of one lung anaesthesia, potential for acute lung injury and infection.
- Patient positioning during surgery.

Post-operative care

Coagulation and haemorrhage

There is significant potential for post-operative bleeding related to:
- Massive, prolonged surgery with raw areas, multiple arterial anastomoses, hypothermia, coagulopathy, and possible thrombocytopenia.
- Consider coagulation support, use of thromboelastogram. Follow massive haemorrhage guidelines if needed.

Positioning

The supine (median sternotomy) position during surgery reduces, and the right lateral posture increases the risks of:

- Nerve injuries, damage to pressure areas, shoulder/arm injury.
- Venous obstruction, facial/brain oedema.
- Prolonged deflation of the left lung may produce residual collapse and ALI.

Respiratory support

- Left lung ALI:
  - The left lung is traumatized by prolonged deflation, retraction, atelectasis and collapse, bleeding, ALI, oedema.
  - The risks of infection/pneumonia are increased.
  - ALI should be managed using an ARDSnet ventilator strategy.

SIRS

- SIRS is common after TAAA repair. It is defined as:
  - Temperature (>38, <36).
  - White count (>12, <4).
  - Hypotension <90 (or inotropes requirement).
  - Respiratory rate >20 (or need for ventilation).
- Inflammatory markers are increased, albumin falls with the trauma of surgery, bypass, hypothermia, and massive bleeding.
- Potential for multiple organ failure.
- Diagnosis/management of infection are made more difficult.
- Prophylactic antibiotics are indicated for intraoperative use only.

Cardiovascular function

- LV function important and TOE is useful guide to this. Patients often have modest requirements for +ve inotropes or vasopressors.
- *Multiple anastomosis are involved in TAAA surgery:* coeliac, mesenteric, renal, intercostals/lumbar re-implantation (artery of Adamkiewicz)
- It is important to measure and maintain cardiac output and MAP, hence, maintain spinal cord perfusion.
- Spinal cord perfusion pressure (MAP – CSF pressure or CVP, whichever is higher) should be maintained at >60mmHg

CNS: brain and spinal cord injury

- Strategies for brain protection and ischaemia/reperfusion injury are much debated. Practice varies and data are lacking.
- Spinal cord injury/paraplegia is more frequent after thoracic aneurysm surgery because the spinal cord blood supply is potentially compromised by division of crucial arteries and intraoperative hypotension. Spinal cord blood supply is from:
  - The anterior spinal artery, formed from both vertebral arteries.
  - Segmental arteries at each spinal level, from intercostals, lumbar arteries, which network longitudinally, anteriorly and posteriorly.
  - One branch forms radicular arteries, which form the posterior spinal arteries (running longitudinally, bilaterally, along the posterior aspect of the cord).
  - The radicular arteries also augment the anterior spinal artery, at most levels.
  - One critical, large radicular artery, (artery of Adamkiewicz), major supplier to anterior spinal artery flow. Arises variably, classically at T9–L1, enters the spinal canal, U-turns cephalad to anastomose with anterior spinal artery at T4–6.

- Spinal cord ischaemia is related to the number of segmental arteries interrupted and the degree of hypotension. Intraoperative and post-operative measures to decrease spinal cord injury include:
  - 'Slick' surgery to minimize clamp times, re-implanting of key vessels, preserving CNS and CSF perfusion pressures, shunting/bypass to perfuse distal aorta/cord and viscera, hypothermic arrest, CO_2 control, the use of generalized hypothermia, or epidural/spinal hypothermia.
  - Preserve cord perfusion pressure (MAP – CSF pressure).
  - CSF drainage to control CSF pressure (📖 Cerebrospinal fluid drainage, p. 517). Typically, aim for slow drainage (maximum 20mL/h or 120mL/6h), aiming to limit CSF pressure to 10mmHg.
- Cerebral/cord protection drugs have been trialled (but with little positive data) including: barbiturates, steroids, magnesium, mannitol, lidocaine; vitamin C, allopurinol, NAC, intrathecal papaverine, desferrioxamine, Ca^{2+} channel blockers.
- Prevention of hyperthermia is important in potential brain/cord injury.
- Hypothermia impacts on immune function, inflammation/healing, diuresis, haemodynamics.
- Intraoperative spinal cord monitoring may continue post-operatively. Options are:
  - Somatosenory-evoked potentials, motor-evoked potentials, F wave polysynaptic-respiratory complex.
  - These mostly examine motor function and are not 100% reliable in excluding leg/cord injury.

Renal function

The risk of AKI is much higher after thoracic aneurysm surgery compared with other vascular procedures. Perception that intraoperative polyuria indicates that renal blood flow is adequate and that polyuria may make AKI easier to manage, but there are no data to confirm this. Management of oliguria is discussed in 📖 Postoperative oliguria, p. 490.

Long-term outcome after vascular surgery

Aortic aneurysm

The aim of surgery for aneurysmal disease of the aorta is to prevent aortic rupture. Commonest form of aortic disease treated by vascular surgeons is AAA. Application of stent graft technology (developed to treat AAA) to thoracic aorta means that many vascular surgeons will perform endovascular procedures on thoracic aorta, including thoracic dissections. These procedures will also be considered here.

Survival after elective AAA repair

- 5-yr survival after AAA repair is approximately 75%.
- The majority of deaths after AAA repair are not related to the presence or repair of the aneurysm
- Mortality directly related to AAA is only responsible for ~10% of all late deaths. The majority (~75%) of patients die from cardiovascular or respiratory diseases, including lung cancer.

- Several randomized controlled trials have been performed comparing open AAA repair and endovascular AAA repair (EVAR1, DREAM, OVER); the EVAR1 trial has the longest follow-up period to date.
- Whilst all of the trials demonstrated a benefit in peri-operative mortality for EVAR over open repair, the EVAR 1 trial has shown that there is *no difference* in all-cause or aneurysm-related death rates at 8yrs between open AAA repair and EVAR.
- The commonest counter-argument against this data (by surgeons favouring EVAR) is that the rapid evolution of stent graft technology since the recruitment phase of this trial was completed means that these results are now outdated

Complications of EVAR

Endoleaks

- The aim of EVAR is the exclusion of the AAA from the circulation. The principle long-term complication of EVAR is movement or dislocation of the stent components, resulting in blood leaking around the stent into aneurysm sac (termed an endoleak). Other causes of endoleak include:
  - Changes in the aneurysm or local aortic morphology (further aneurysm expansion) so that the stent becomes ineffective.
  - Blood leaking around stent, irrespective of the cause, results in the re-pressurization of the aneurysm sac and places patient at risk of post-operative aneurysm rupture.
- Endoleaks are classified as:
  - *Type 1*—arising around the top or bottom ends of the graft.
  - *Type 2*—resulting from retrograde flow into the sac from branch vessels (lumbar arteries or inferior mesenteric artery).
  - *Type 3*—arising from gaps between graft components or holes in the graft material.
  - *Type 4*—resulting from graft porosity (direct leak through graft wall).
- Type 1 and type 3 endoleaks result in high pressure flow within the aortic sac and result in the risk of post-operative aneurysm rupture. These complicate up to 10% of EVARs during follow-up.
- Type 2 and 4 endoleaks only result in low intra-sac pressure.
- A less common complication of EVAR is graft thrombosis, affecting less than 5% of patients after EVAR. This usually affects just one limb of the stent-graft but can affect the entire graft.

Graft surveillance

- All patients are followed-up after EVAR with long-term serial imaging, to reduce the chance of late aneurysm rupture after EVAR, and to check for complications, such as limb stenosis or kinking that may predispose to graft thrombosis.
- No single imaging modality is better than any other.
- Local protocols for post-EVAR follow-up ∴ vary, but will usually consist of regular clinical review, together with either US and selective CT/MRI to clarify abnormal findings on US, or CT/MRI alone.

- Serial plain radiographs can be used to detect stent fractures, and are often used in conjunction with US and cross-sectional imaging.
- The length of follow-up varies between centres, but is usually for at least 5yrs and, in many cases, is life-long.

Interventions

- Most interventions after EVAR are performed to prevent future complications.
- Graft stenoses or kinking are usually treated by percutaneous angioplasty and stent placement if necessary.
- Type 1 and type 3 endoleaks are usually treated with extra stent-grafts placed to cover the endoleak. When a type 1 endoleak arises from or around the top end of the stent-graft, there may not be an adequate length of normal aorta above the graft to enable placement of an extra piece of stent graft. In this case, the options are either:
  - Placement of a fenestrated stent-graft extension cuff (i.e. with holes to allow renal/mesenteric perfusion).
  - An open stent-graft excision and traditional open AAA repair; this has a peri-operative mortality rate of at least 25%.

Complications after open AAA repair

- Open AAA repair is a durable procedure.
- The most serious complication is graft infection, often associated with aorto-enteric fistula.
- Graft infection and aorto-enteric fistula are rare, with incidences of <2% after open AAA repair. However they are both life-threatening; each with an overall mortality rate >75%.
- The treatment of both conditions is revision surgery: the excised aortic graft is usually replaced with a composite graft constructed from the deep veins of the lower limbs. Alternatively, the aorta may be ligated and an axillo-bifemoral bypass graft performed.
- Aorto-enteric fistulae may be temporarily treated using an endovascular technique. However, the remaining communication between the intestinal lumen and the aortic sac/graft invariably results in a graft infection, so graft excision is needed later.
- Incisional hernia is a minor, but significant long-term complication and affects approximately 10% of patients following open AAA repair.

Ruptured AAA

- Patients who survive repair of ruptured AAA are generally perceived to be at high risk of significant long-term sequelae related to prolonged hospitalization.
- However, there are limited good quality long-term data on survival after ruptured AAA repair.
- The available data suggest that:
  - Long-term survival in those who leave hospital appears similar to that after elective open AAA repair.
  - Quality of life in patients after ruptured AAA repair is similar to that after elective open AAA repair.

Thoracic aortic aneurysms

- Anatomically suitable TAA can be treated using endovascular stenting. These techniques also have a role in the treatment of aortic dissections and traumatic aortic arch transections.
- Outcomes for TAA treated with endovascular techniques are similar to those seen for AAA: overall 5-yr survival rates are 60–70%.
- The majority (60–80%) of deaths are unrelated to the aneurysm or the repair.
- Some non-randomized trial evidence shows that there is a potential long-term survival benefit for endovascular TAA repair compared with open repair, but this has not been confirmed in independent studies.
- Currently, there are inadequate data on long-term outcomes after endovascular treatment for aortic dissection or transection to define survival or complication rates accurately.

Peripheral arterial disease

- All patients with PAD are at >2-fold higher risk of cardiovascular deaths compared with patients without PAD.
- 5-yr survival in this patient group, irrespective of whether intervention is required, is approximately 80% (compared with approximate 90% 5-yr survival in age-matched healthy controls).
- ∴ All patients with PAD should receive treatment with anti-platelet agents and HMG-CoA reductase inhibitors unless intolerant of these medications, and be referred for smoking cessation therapy.
- Patients presenting with critical limb ischaemia have poorer long-term survival than those with uncomplicated peripheral arterial disease, irrespective of any treatment received. Five-year survival in this group is 50–60%.

Aortic occlusive disease

- The procedure of choice to revascularize patients with aortic occlusive disease is an aorto-bifemoral bypass.
- Aorto-bifemoral bypass has 5-yr patency rates of ~ 90%, and 5-yr survival rates of 80–90%. These are similar to or greater than those for patients with PAD in general.
- In patients unfit for abdominal surgery, axillo-bifemoral bypass is an alternative. The principle disadvantage of an axillo-bifemoral bypass is the poorer long-term patency rate (~70% at 5yrs).
- Axillo-unifemoral grafts can be performed, avoiding a graft crossing the midline, but at the expense of even poorer patency (~50% at 5yrs).

Iliac disease

- The majority of patients with short iliac stenoses or occlusions will be treated by percutaneous angioplasty with, or without, adjunctive stenting. This has a good long-term patency rate (~70% at 5yrs).
- In patients with long iliac occlusions or those in whom attempts at angioplasty have failed, surgery is usually offered if the patient is fit.
- Patients with bilateral disease are usually treated by aorto-bifemoral grafting, but if the disease is unilateral or if one side can be treated percutaneously then ipsilateral ilio-femoral bypass or ilio-femoral/femoro-femoral cross-over grafting can be performed. Surgical bypasses have similar long-term patency rates to angioplasty.

Infra-inguinal disease

Graft patency

- Many cases of infra-inguinal peripheral arterial disease are treated by percutaneous angioplasty. This has 5-yr primary patency rates (i.e. patency without re-intervention) of 50% – 60% for femoro-popliteal disease and slightly less than this for more distal procedures. Overall patency (secondary patency) rates are somewhat higher because re-interventions may be performed. Limb salvage rates following angioplasty are higher than patency rates.
- Surgery is usually reserved for those patients with critical limb ischaemia; surgery is generally avoided in patients with intermittent claudication only. An exception to this is where infra-inguinal bypass is also required in patients with large popliteal aneurysms to prevent thrombosis.
- The long-term outcomes after infra-inguinal bypass are partly dependent upon the conduit used—prosthetic grafts have poorer long-term patency rates, particularly when the distal end of the graft is below the knee.
- Graft patency also depends on the location of the distal anastomosis; more distal grafts have poorer long-term patency.
- 5-yr patency ranges from 80% for above-knee femoro politeal bypasses using vein as a graft to less than 50% for femoro-tibial bypasses using prosthetic grafts.

Survival

- Survival after infra-inguinal bypass grafting can be considered in terms of overall survival or amputation-free survival.
- In patients with critical limb ischaemia, lower limb bypass has a 5-yr survival rate of 50–70%. Amputation-free survival at 5yrs is approximately 50% (of all patients operated upon, not just those surviving to 5yrs).
- Survival after treatment of popliteal aneurysms has a 5-yr survival of ~80% and limb-salvage rates are >90%, despite 1° patency rates similar to those seen in bypasses performed for occlusive disease (~60–70%).

Surveillance

- When autologous vein has been used as a bypass conduit, patients are usually entered into vein-graft surveillance programmes, involving serial duplex imaging of the bypass to identify graft stenoses. The rationale behind this is to identify graft stenoses before they become severe enough to cause graft thrombosis. When identified, stenoses are often treated by percutaneous angioplasty rather than surgery.
- Graft surveillance has been shown to improve long-term graft patency from ~50 to 80% with resulting improvement in limb-salvage rates.
- Prosthetic grafts are not amenable to angioplasty and graft surveillance is not indicated in this group of patients.

Lower limb amputation

- Long-term outcomes after major lower-limb amputation for peripheral vascular disease are poor, both in terms of survival and mobility. This is in contrast to those performed for trauma, which have a better prognosis overall, with survival rates similar to those seen in the overall population.

- Survival after amputation for limb ischaemia is poor: 5-yr survival is 30–50%. The majority of deaths are due to cardiovascular disease.
- There is some evidence that above-knee amputations are associated with worse 5-yr survival rates (up to 70%).
- The chances of being to mobilize independently after lower limb amputation are significantly higher in younger patients.
  - *In patients aged <50yrs:* almost all are mobile with a limb prosthesis by 12 months after amputation.
  - *In those aged >50yrs:* only 50–75% of those undergoing below-knee amputation and 25–50% of those undergoing above-knee amputation will be mobilizing with a prosthesis at 12 months
  - The reasons for poor long-term mobility after amputation in elderly patients may relate to phantom limb pain, stump pain, psychological or social factors, infirmity and other disease, e.g. arthritis.
- Poor mobility after amputation results in significant social care and rehabilitation costs. The cost of amputation at 1yr (including inpatient costs) is approximately twice that of infra-inguinal bypass surgery.

Carotid endarterectomy

- The aim of CEA is to prevent future stroke and death from stroke. Patients with CAD have the same risks as all patients with cardiovascular disease and in trials of surgery for asymptomatic carotid stenosis the most common cause of death in all participants was non-stroke cardiovascular death. The same measures for 2° cardiovascular prevention should be followed.
- CEA reduces the long-term risk of stroke, but does not remove this risk completely.
- When CEA is performed for symptomatic disease (transient ischaemic attack, stroke or amaurosis fugax) the risk of stroke at 5yrs is reduced by 21% in patients with stenoses of 70–99%, allowing for 6.8% peri-operative risk of death or stroke associated with surgery.
- For symptomatic carotid stenoses between 50% and 69%, the absolute risk reduction from CEA is just under 6%.
- In patients who have asymptomatic carotid stenoses of >60%, the benefit from CEA is reduced, but still present: 5-yr combined risk of stroke and peri-operative death is 5.9% compared with a 5-yr risk of stroke without surgery of 10.9%.
- The most significant late complications of CEA are patch infection and re-stenosis.
- Patch infection occurs <1% of cases, but requires re-operation. Patch excision and arterial repair using autologous vein has a low mortality and morbidity rate.
- Re-stenosis occurs in ~ 5% of patients within 5yrs of surgery. The management of symptomatic re-stenosis involves either re-operation or angioplasty. Re-intervention for asymptomatic re-stenosis is controversial; some clinicians recommend re-operation or stenting in all cases and some recommend surveillance.

Further reading

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