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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/

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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.

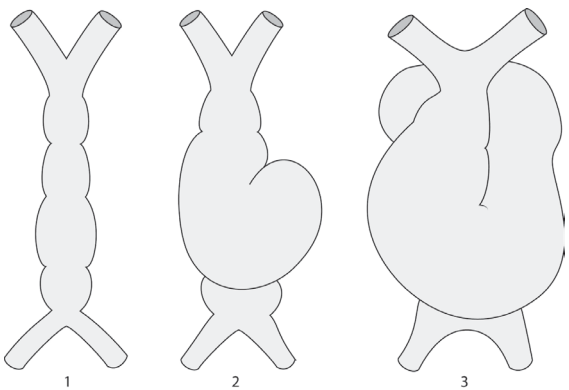


Fig. 2.1 Embryological development of the heart.

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).

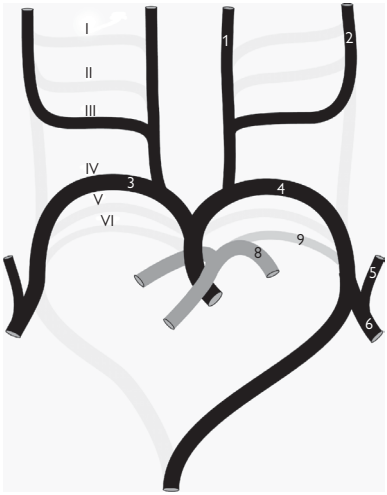
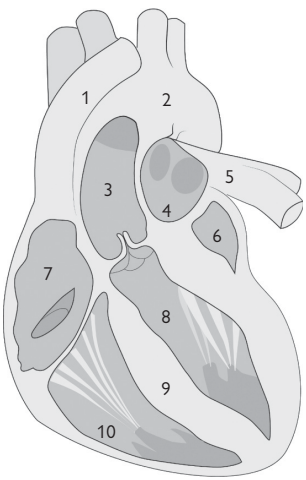


Fig. 2.2 Aortic arches. In early development there are paired dorsal aortae joined to the aortic sac by six arches. The dorsal aortae become the descending aorta.

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.

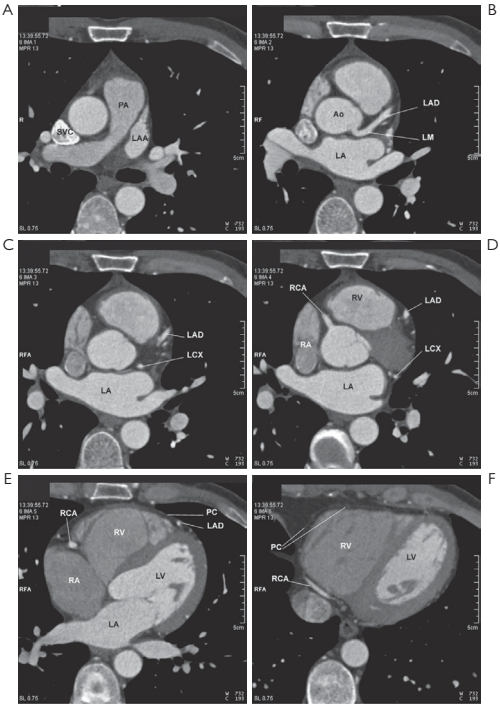


Fig. 2.4 Cross-sectional CT anatomy of the heart. CT images are displayed as if looking from below. The right side is indicated (R), the sternum is in the top of the image. Ao, ascending aorta; CS, coronary sinus; LA, left atrium; LAA, left atrial appendage; LAD, left anterior descending coronary artery; 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).

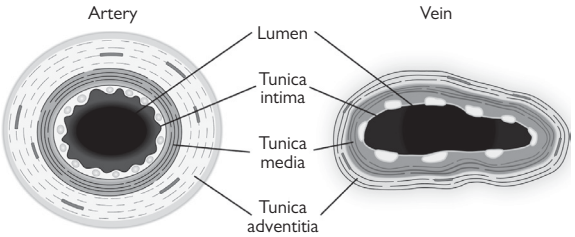


Fig. 2.5 Composition of vessel walls. The tunica adventitia is a dense network of collagen fibres and fibroblasts, which contains nerve fibres and acts to anchor vessels to surrounding structures. The tunica media is composed of circularly arranged smooth muscle cells arranged in bundles, with collagen 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.

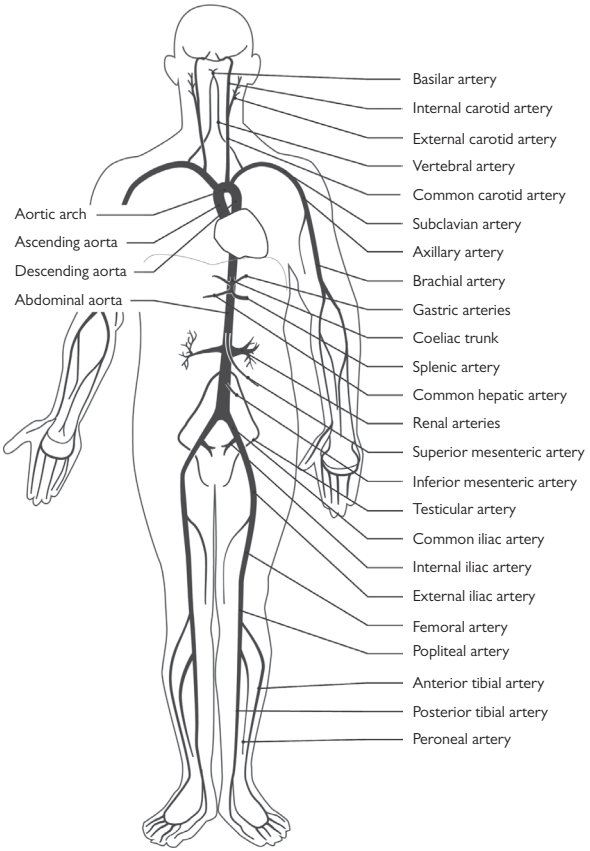
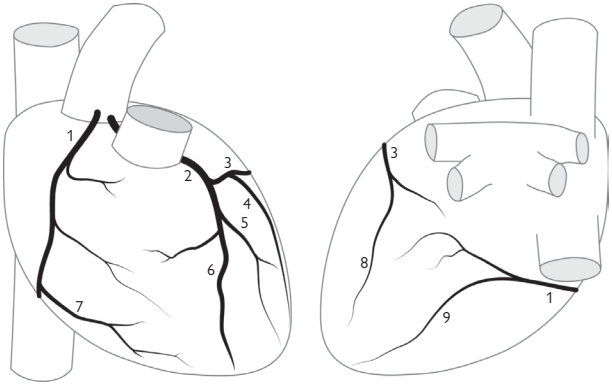


Fig. 2.6 The arterial system.

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.

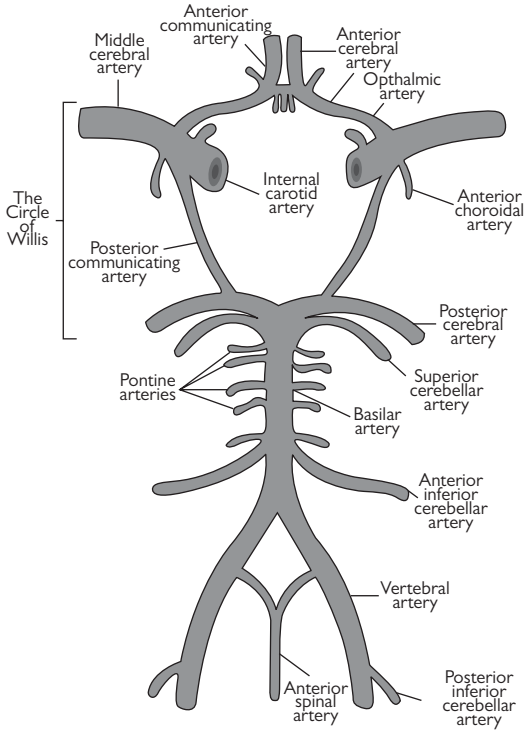


Fig. 2.8 The cerebral circulation and Circle of Willis.

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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.

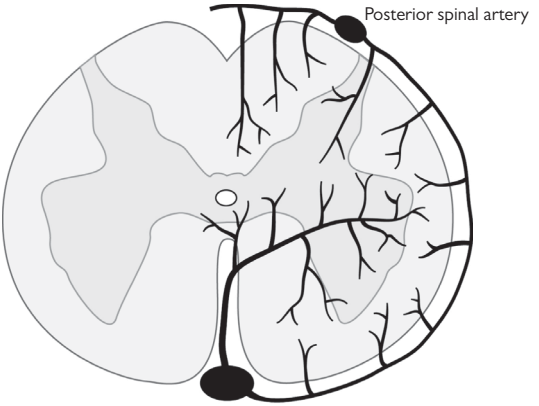

Fig. 2.9 Blood supply to the spinal cord. The spinal cord is supplied by the anterior spinal artery and posterior spinal arteries. These run longitudinally along its length. They are reinforced by the segmental medullary arteries of the cord, which arise from vertebral arteries in the cervical segment, 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.

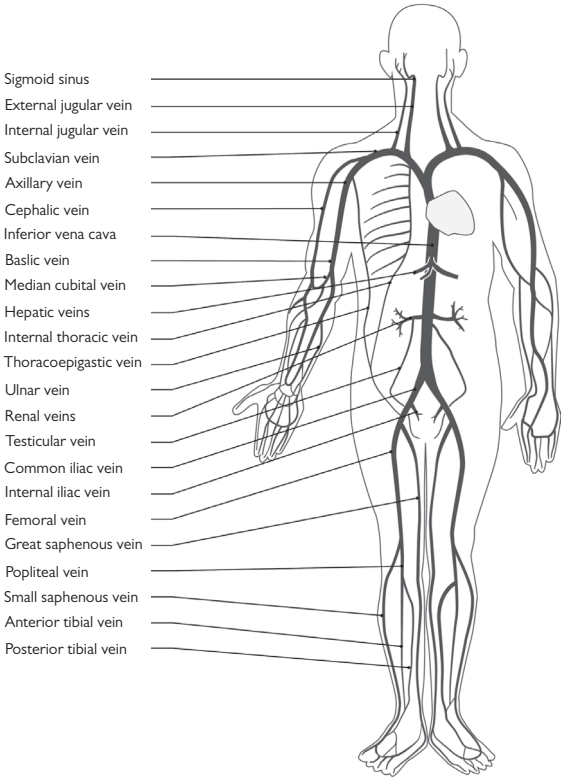

Fig. 2.10 The venous system.

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.