MRCS A ESSENTIAL REVISION NOTES

BOOK 2

Edited by
Claire Ritchie Chalmers
BA PhD FRCS
Catherine Parchment Smith
BSc MBChB FRCS

Pastest

Egerton Court
Parkgate Estate
Knutsford
Cheshire
WA16 8DX

Telephone: 01565 752000

First published 2012, reprinted 2015, 2016

ISBN: 978 1 905 63583 2
eISBN: 978 1 909 49115 1 MobiPocket
978 1 908 18571 6 ePUB

A catalogue record for this book is available from the British Library.

Text prepared in the UK by Carnegie Book Production, Lancaster
Printed and bound in the UK by Bell & Bain Limited, Glasgow

Contents

CHAPTER 1

Abdominal Surgery

Catherine Parchment Smith, Arin K. Saha and Ravinder S. Vohra

SECTION 1

Abdominal wall and hernias

CHAPTER 1

1.1 Anterior abdominal wall

Layers of the abdominal wall

Learning point

When you make an incision in the anterior abdominal wall you will go through several layers:

- Skin
- Subcutaneous fat
- Superficial fascia
- Deep fascia (vestigial)
- Muscles (depending on incision)
- Transversalis fascia
- Extraperitoneal fat
- Peritoneum

Skin

The skin has horizontal Langer's lines over the abdomen. Dermatomes are also arranged in transverse bands.

Deep fascia

This is a vestigial thin layer of areolar tissue over muscles.

Superficial fascia (Scarpa's fascia)

- Absent above and laterally
- Fuses with deep fascia of leg inferior to inguinal ligament
- Very prominent in children (can even be mistaken for external oblique!)
- Continuous with Colles' fascia over perineum (forms tubular sheath for penis/clitoris and sac-like covering for scrotum/labia)

Muscles

Learning point

The muscles you'll pass through depend on the incision site:

- External oblique
- Internal oblique
- Rectus abdominis
- Transversus abdominis
- Pyramidalis
- Rectus sheath

- **External oblique** is a large sheet of muscle fibres running downwards from lateral to medial like a 'hand in your pocket'. Medially, the external oblique becomes a fibrous aponeurosis which lies over the rectus abdominis muscle (see below), forming part of the anterior rectus sheath
- **Internal oblique** is a second large sheet of muscle fibres lying deep to the external oblique and at right angles to it. Medially, it forms a fibrous aponeurosis which splits to enclose the middle portion of rectus abdominis as part of the anterior and posterior rectus sheath
- **Transversus abdominis** is the third large sheet of muscle lying deep to the internal oblique and running transversely. Medially, it forms a fibrous aponeurosis which contributes to the posterior rectus sheath lying behind rectus abdominis
- **Rectus abdominis** and its pair join at the linea alba in the midline to form a wide strap that runs longitudinally down the anterior abdominal wall. It lies within the rectus sheath formed by the aponeuroses of the three muscles described above. It is attached to the anterior rectus sheath, but not to the posterior rectus sheath, by three tendinous insertions. These insertions are at the level of the xiphisternum, umbilicus and halfway between (giving the 'six-pack' appearance in well-developed individuals!). The blood supply of rectus abdominis is through the superior epigastric artery (a terminal branch of the internal thoracic artery) and the inferior epigastric artery (a branch of the external iliac artery) which anastomose to form a connection between the subclavian and external iliac systems (Fig. 1.1). The superior epigastric artery is the pedicle on which a TRAM flap is raised for breast reconstruction. The nerve supply to the recti is segmental from T6 to T12 and the nerves enter the sheath laterally and run towards the midline (so are disrupted in Battle's incision – see Figure 1.3)
- **Linea alba** is a fibrotendinous raphe running vertically in the midline between the left and right rectus abdominis muscles. It is formed by the fusion of the external oblique, internal oblique and transversus abdominis aponeuroses. They fuse in an interlocking/interdigitating structure through which epigastric hernias may protrude. The linea alba provides an avascular and relatively bloodless plane through which midline laparotomy incisions are made. It is easier to begin a laparotomy incision above the umbilicus, where the linea alba is wider, thicker and better defined than below the umbilicus
- **Pyramidalis** is a small (4 cm long) unimportant muscle arising from the pubic crest and inserting into the linea alba. It lies behind the anterior rectus sheath in front of rectus abdominis. This is the only muscle you go through in your lower midline laparotomy incision and it is not as bloodless as the linea alba which it underlies

Rectus sheath

Learning point

Any incision over rectus abdominis will go through the anterior rectus sheath. Arrangement of the rectus sheath is best considered in three sections:

- Above the level of the costal margin
- From the costal margin to just below the umbilicus
- Below the line of Douglas

- **Above the level of the costal margin:** the anterior rectus sheath is formed by the external oblique aponeurosis only. There is no internal oblique or transversus abdominis aponeurosis at this level. Therefore there is no posterior rectus sheath and rectus abdominis lies directly on the fifth to seventh costal cartilages
- **From the costal margin to just below the umbilicus:** the anterior rectus sheath is formed by the external oblique aponeurosis and the anterior leaf of the split internal oblique aponeurosis. It is attached to rectus abdominis by tendinous intersections. The posterior rectus sheath is formed by the posterior leaf of the internal oblique aponeurosis and the transversus abdominis aponeurosis
- **Below the line of Douglas:** about 2.5 cm below the umbilicus lies a line called the 'arcuate line of Douglas' (Fig. 1.1). At this level, the posterior rectus sheath (ie the posterior leaf of the internal oblique aponeurosis along with the transversus abdominis aponeuroses) passes anterior to rectus abdominis. Therefore, below the arcuate line of Douglas there is no posterior rectus sheath. Rectus abdominis lies directly on transversalis fascia, which is thickened here, and called the 'iliopubic tract'. The anterior rectus sheath is now formed by all the combined aponeuroses of the external oblique, internal oblique and transversus abdominis muscles

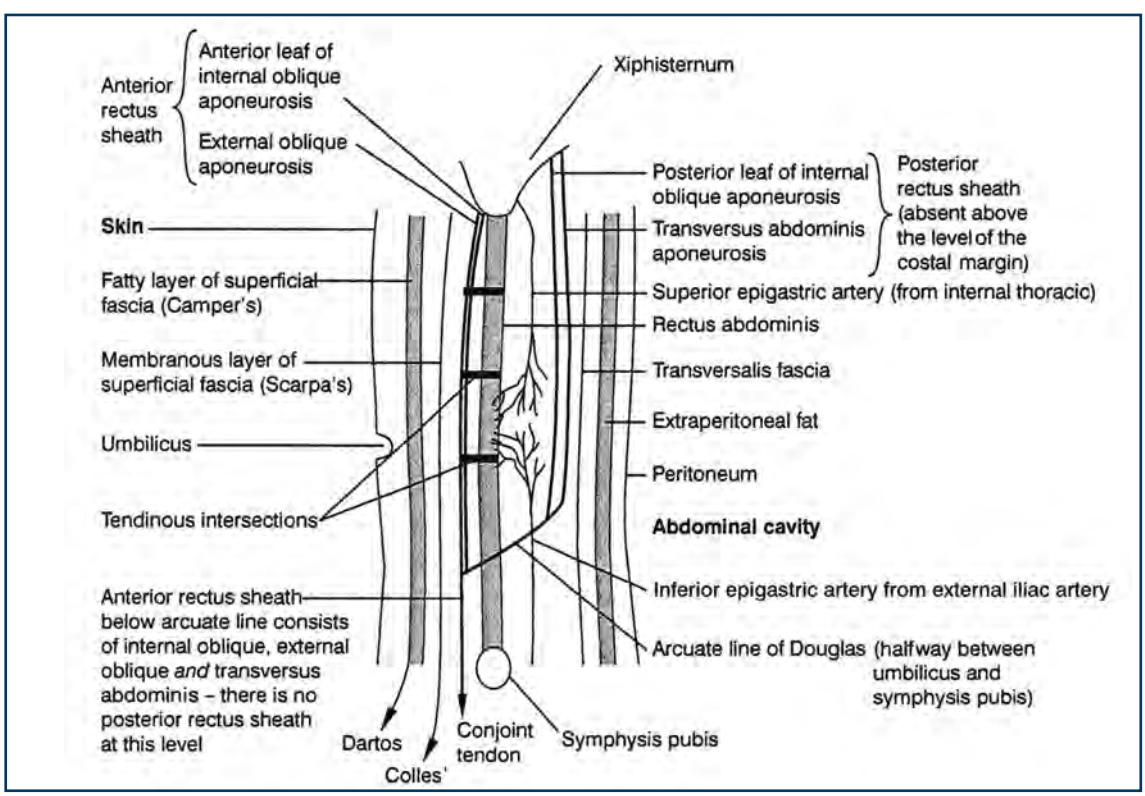

Figure 1.1 Sagittal section of the abdominal wall

CHAPTER 1

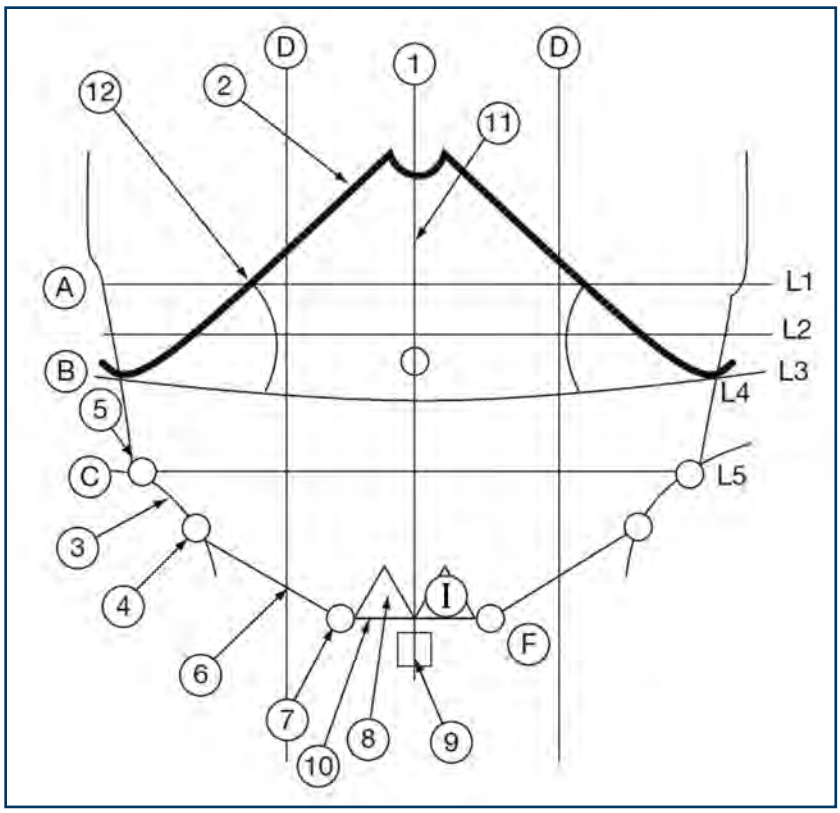

Figure 1.2 Surface landmarks of the anterior abdominal wall

(A) **Transpyloric line:** halfway between jugular notch and pubic symphysis at L1; this plane passes through pylorus, pancreatic neck, duodenojejunal flexure, fundus of gallbladder, tip of ninth costal cartilage, hila of kidneys; also it is the level of termination of the spinal cord.

(B) **Subcostal line:** under lowest rib (rib 10 at L3).

(C) **Intertubercular/transtubercular line:** between two tubercles of iliac crest (L5); note that plane of iliac crests (supracristal plane) is higher (at L4).

(D) **Midclavicular line:** through midinguinal point, halfway between ASIS and symphysis pubis.

(1) **Xiphoid process:** xiphisternal junction is at T9.

(2) **Costal margins:** ribs 7–10 in front; ribs 11 and 12 behind; tenth costal cartilage is lowest at L3.

(3) **Iliac crest:** anterior superior iliac spine (ASIS) to posterior superior iliac spine (PSIS); highest point L4.

(4) **ASIS**.

(5) **Tubercle of iliac crest:** 5 cm behind ASIS at L5.

(6) **Inguinal ligament:** running from ASIS to pubic tubercle.

(7) **Pubic tubercle:** tubercle on superior surface of pubis; inguinal ligament attaches to it, as lateral end of the superficial inguinal ring.

(8) **Superficial inguinal ring:** inguinal hernia comes out above and medial to pubic tubercle at point marked (I); femoral hernia below and lateral to pubic tubercle at point marked (F).

(9) **Symphysis pubis:** midline cartilaginous joint between pubic bones.

(10) **Pubic crest:** ridge on superior surface of pubic bone medial to pubic tubercle.

(11) **Linea alba:** symphysis pubis to xiphoid process midline.

(12) **Linea semilunaris:** lateral edge of rectus crosses costal margin at ninth costal cartilage (tip of gall bladder palpable here).

Contents of the rectus sheath

- Rectus abdominis
- Pyramidalis
- Segmental nerves
- Segmental vessels from T7 to T12
- Superior and inferior epigastric vessels (see Figure 1.1)

Layers of the abdominal wall divided in three common incisions

Midline laparotomy	Kocher's incision	Gridiron appendicectomy incision
Skin	Skin	Skin
Subcutaneous fat	Subcutaneous fat	Subcutaneous fat
Scarpa's fascia	Scarpa's fascia	Scarpa's fascia
Linea alba	Medially: Anterior rectus sheath Rectus abdominis Posterior rectus sheath	
	Laterally: External oblique Internal oblique Transversus abdominis	External oblique Internal oblique Transversus abdominis
Fascia transversalis	Fascia transversalis	Fascia transversalis
Preperitoneal fat	Preperitoneal fat	Preperitoneal fat
Parietal peritoneum	Parietal peritoneum	Parietal peritoneum

Diseases of the umbilicus

Congenital

- Cord hernias
- Gastroschisis
- Exomphalos

Tumours

- Primary
  - Benign (papilloma, lipoma)
  - Malignant (squamous cell carcinoma [SCC], melanoma)
- Secondary
  - Breast
  - Ovarian
  - Colon (via lymphatic, transcoloemic and direct spread along falciform ligament)

Endometriosis

Hernias

- Childhood (umbilical)
- Adult (paraumbilical)

Fistula

- Urinary tract (via urachal remnant)
- Gastrointestinal tract (via vitelloin-testinal duct)

Suppurations

- Primary
  - Obesity
  - Pilonidal
  - Fungal infections
- Secondary
  - From intra-abdominal abscess

CHAPTER 2

Breast Surgery

Jenny McIlhenny and Ritchie Chalmers

CHAPTER 2

SECTION 1

The breast

CHAPTER 2

1.1 Anatomy of the breast and axilla

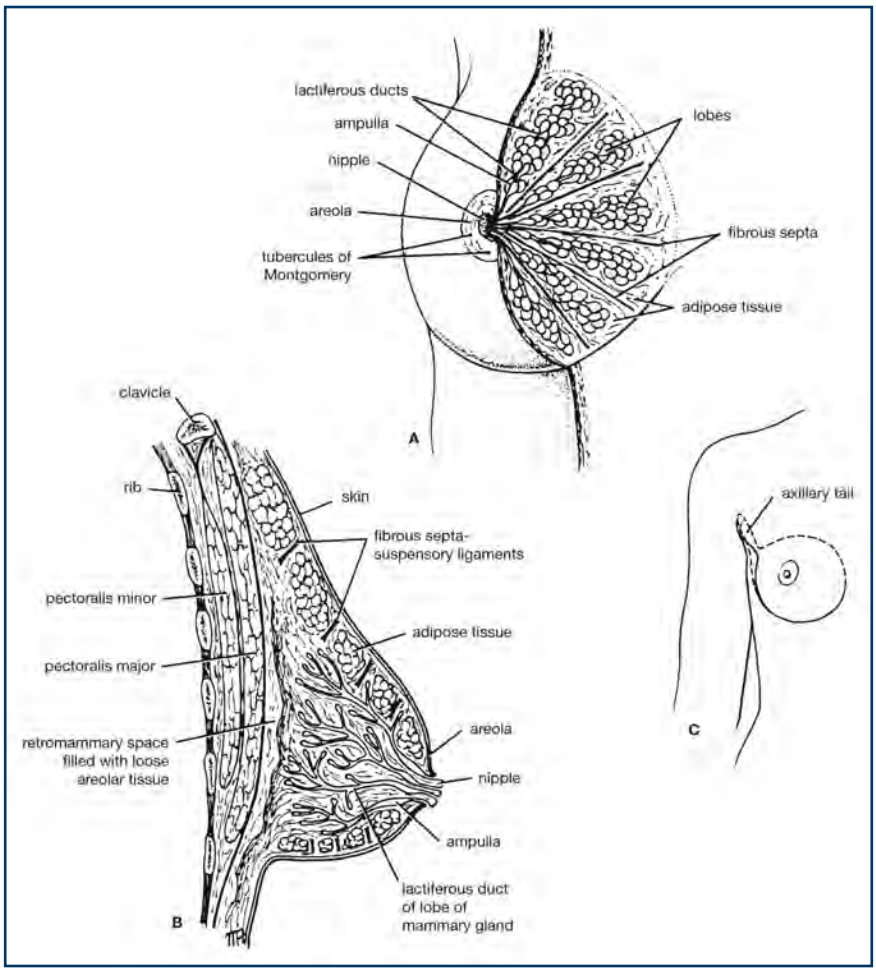

Figure 2.1 Anatomy of the breast. (A) Anterior view with skin partially removed to show internal structure. (B) Sagittal section. (C) The axillary tail, which pierces the deep fascia and extends into the axilla.

Embryology of the breast

- The breast is a modified apocrine sweat gland
- Ectoderm gives rise to the epithelial lining of the ducts and acini
- Mesenchyme gives rise to the supporting tissue called the stroma
- There is downward growth of ectoderm into underlying mesenchyme at 4 weeks' gestation
- The breast develops from the mammary ridge (first seen at 5 weeks' gestation) and this ridge develops from ectoderm at 5 weeks' gestation, extending from the axilla to the groin (milk line). In humans it regresses to leave a nipple with specialised epithelial cords which forms 15–20 lactiferous ducts
- At puberty the lactiferous ducts proliferate and the breast bud is formed (around age 10)
- Nipple development occurs at around 12 years

Anatomy of the breast

Learning point

- **Extent:** the breast extends from the second to the sixth rib, and from the midline to the midaxillary line. The axillary tail of Spence is a projection of the upper outer quadrant of the breast into the axilla.
- **Position:** the breast lies anterior to the muscles of the chest wall: medial two-thirds on pectoralis major and lateral third on serratus anterior; the inferior aspect of the breast base also rests on the upper aspects of the external oblique abdominis and rectus sheath. It is separated from these muscles by the deep fascia.
- **Structure:** the breast is composed of 15–20 lobules which open, via ducts, on to the nipple. Lobules drain into lactiferous ducts, which drain into lactiferous sinuses, the function of which is to store milk during lactation. The ducts of each lobule are lined with columnar or cuboidal epithelium, and the short excretory ducts (just beneath the nipple/areolar complex) are lined with squamous epithelium.
- **Suspensory ligaments:** anterior projections of the deep fascia form the suspensory ligaments of Astley Cooper which divide the breast into lobules and connect the deep fascia to the skin
- **Arterial supply:** lateral thoracic and thoracoacromial branches of the axillary artery, the internal thoracic (mammary) artery and the lateral perforating branches of the intercostal arteries
- **Venous drainage:** via the internal thoracic, axillary and posterior intercostal veins
- **Nerve supply:** intercostal nerves. Nipple sensation is from the fourth intercostal nerve
- **Lymphatic drainage:** >75% to axillary nodes, <25% to internal mammary nodes
- **The nipple** is anatomically in line with the fourth rib interspace (at the inframammary fold) but its position varies with increasing age as a result of glandular descent or ptosis.

Arterial supply of the breast

There is considerable crossover of arterial supply and venous and lymphatic drainage across the breast.

Lateral breast

Subclavian artery → Axillary artery → **Lateral thoracic artery**
→ **Thoracoacromial artery**

(Also the **superior thoracic** and **subscapular** branches of the axillary artery to a lesser extent.)
Lateral perforating branches of the **intercostal arteries**

Medial breast

Subclavian artery → **Internal thoracic (mammary) artery** → Perforating branches (first to fourth intercostal spaces)

Venous drainage of the breast

Lateral breast → **Thoracoacromial vein**
→ **Lateral thoracic vein** → Axillary vein → Subclavian vein

Medial breast → **Internal thoracic vein** → Subclavian vein

Posterior intercostal vein: the posterior intercostal veins also receive tributaries from the ribs and communicate with the vertebral venous plexus via a valveless system; hence metastatic cells from breast cancer can spread to the ribs and thoracic vertebrae with relative ease.

Nerve supply of the breast

- Sensory supply via cutaneous branches of intercostal nerves of T4–6
- Also a sympathetic supply

Lymphatic drainage of the breast

- Studies using radiolabelled tracers demonstrate that 75–97% of the lymphatic drainage of the breast drains to the axillary nodes
- Up to 25% drains to the internal mammary nodes through the second to fourth intercostal spaces
- There is free communication of lymphatic channels across the breast (lateral to medial and vice versa)
- There is an anastomosis of the lymphatics across the midline to the contralateral breast, and down the abdominal wall; therefore lymphatic spread of breast cancer can be to the opposite axilla, to the peritoneal cavity and liver and, rarely, to the inguinal nodes
- The sentinel lymph node or nodes are believed to be the first node(s) in the axilla to receive drainage from the breast (see Sentinel node biopsy)

Anatomy of the axilla

The axilla is the space between the upper arm and the thorax. The surface markings are the anterior and posterior axillary folds, formed by pectoralis major and latissimus dorsi respectively. It is pyramidal in shape, with the base of the pyramid (the floor of the axilla) the most superficial, and the apex deep, towards the root of the neck (Figure 2.2). The axillary vessels and nerves pass through a space at the apex formed by the posterior clavicle, superior border of the scapula and lateral border of the first rib.

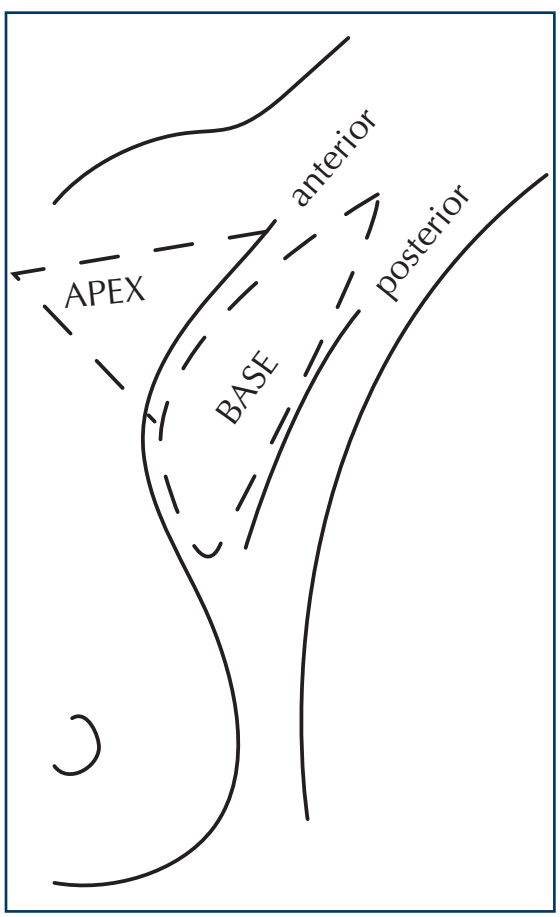

Figure 2.2 The shape of the axilla

Learning point

Contents of the axilla

- Brachial plexus
- Axillary artery
- Axillary vein
- Axillary fat pad, which contains 20–30 axillary lymph nodes
- Intercostal brachial nerves (sensory nerves to skin of axilla)
- Long thoracic nerve of Bell (motor nerve to serratus anterior)
- Thoracodorsal trunk (the artery, vein and motor nerve supplying latissimus dorsi)

Boundaries of the axilla

- **Anteriorly:** pectoralis major and minor; subclavius at apex
- **Medial:** serratus anterior on the chest wall (and first four ribs)
- **Lateral:** coracobrachialis; short biceps tendon. These lie medial to the bicipital groove of the humerus (sometimes called the intertubercular groove, as it lies between the greater and lesser tubercles), where the anterior and posterior walls of the axilla converge
- **Posterior:** subscapularis superiorly; teres major; latissimus dorsi
- **Superior:** axillary vein
- **Apex:** clavicle; first rib; superior border of scapula
- **Floor (or base):** axillary fascia covers the axillary floor between serratus anterior, pectoralis major and latissimus dorsi, converging at a point to meet the deep fascia of the arm

CHAPTER 3

Cardiothoracic Surgery

George Tse and Sai Prasad

SECTION 1

Surgery for ischaemic heart disease

CHAPTER 3

Ischaemic heart disease is one of the major causes of morbidity and mortality in the western world. Survival is slowly improving due to medical advances; however, worldwide prevalence is rapidly rising due to the westernisation of the developing world, with increased rates of diabetes, obesity and smoking. In the UK, 30% of patients who have a myocardial infarction (MI) never get to hospital.

1.1 Pathology of ischaemic heart disease

Learning point

The majority of ischaemic heart disease is caused by atherosclerosis, with the steps of:

- Endothelial dysfunction
- Lipid accumulation (in subendothelial macrophages)
- Smooth muscle migration and fibroblast proliferation, resulting in intimal thickening
- Chronic inflammatory response, leading to calcification of the plaque

The vast majority of patients with coronary heart disease (CHD) have advancing occlusive coronary stenoses which, when sufficiently severe, erode the 'coronary reserve' and cause flow limitation on exercise, leading to exertional angina. Most myocardial infarctions (MIs) occur in the context of chronic atherosclerosis.

Atherosclerosis affects the epicardial vessels but not the intramyocardial coronary vessels (the proximal left anterior descending, circumflex and right coronary arteries are the worst affected). The internal thoracic (mammary) arteries (IMAs) are relatively spared from the disease and hence can be useful in revascularisation of the heart.

The pathological basis of atherosclerosis is still an area of research, but involves the following key steps:

- Endothelial cell injury and dysfunction due to several risk factors, most importantly hypertension, nicotine, immune mechanisms and hyperlipidaemia
- Low-density lipoprotein (LDL) accumulation form the plasma – plasma proteins carrying triglycerides and cholesterol. Leading to intimal thickening and atheroma formation
- Platelet adhesion to the surface of endothelial cells leading to microthrombi

formation and release of platelet-derived growth factor, which attracts monocytes to the area
- Low density lipoprotein (LDL) accumulation form the plasma – plasma proteins carrying triglycerides and cholesterol. Leading to intimal thickening and atheroma formation
- Monocytes/macrophages engulf cholesterol, becoming foam cells, and also secrete free radicals, which causes oxidation of circulating LDL
- Further oxidised LDL is taken up by macrophages via their scavenger receptors, leading to release of interleukin-1 (IL-1) and tumour necrosis factor (TNF), stimulating smooth muscle cell and fibroblast proliferation
- Fibroblasts lay down collagen and elastin which helps to form the mature athero-sclerotic plaque
- Smooth muscle cells migrate from the media to the intima and transform into secretory cells producing collagen, elastin and chemokines, which serve as important mediators of the process
- This chronic inflammatory response leads to calcification of the plaque

Initially, atherosclerotic plaques tend to bulge outwards and so the vascular lumen is maintained.
- Luminal loss of <50% can usually be tolerated without causing symptoms
- Obstruction of 75% is associated with exertional angina
- 90% stenosis causes angina at rest

In the progression of atherosclerotic plaques, there are two potential outcomes:
1. **Gradual progression of the plaque** causing increasing luminal stenosis and decreasing exercise tolerance
2. **Acute plaque rupture**, in which the endothelium becomes fissured, exposing the thrombogenic plaque contents (lipids, collagen and necrotic debris), and a clot forms on top of the plaque causing acute coronary obstruction, resulting in an 'acute coronary syndrome' (unstable angina, MI or acute coronary death)

The table opposite summarises the pathological and clinical features together with treatment options for stable and unstable angina and MI.

1.2 Indications for coronary artery revascularisation

Learning point

When medical therapies are insufficient to adequately control angina symptoms, revascularisation may be attempted. This field is subject to rapid evolution as both percutaneous and surgical techniques improve.

As coronary vascular disease is primarily managed by cardiologists and **percutaneous coronary intervention** (PCI) has a lower risk of mortality and morbidity, surgery is largely offered when PCI options are exhausted. Hence the population undergoing **coronary artery bypass grafting** (CABG) is an older population, with more severe disease.

ISCHAEMIC HEART DISEASE: PATHOLOGY, CLINICAL FEATURES AND TREATMENT OF STABLE AND UNSTABLE ANGINA AND MYOCARDIAL INFARCTION

Coronary syndrome	Stable angina	Unstable angina (crescendo angina, subendocardial infarction)	Myocardial infarction
Pathology			
Coronary	'Fixed stenosis' Stable plaque	Acute plaque rupture or thrombosis	Acute plaque rupture or thrombosis
Myocardial	Reversible ischaemia	Reversible ischaemia or minor infarction in watershed areas, eg subendocardium	Full-thickness infarction
Clinical features			
Symptoms	Chest pain and shortness of breath on exertion Predictable exercise tolerance Symptoms relieved by nitrates and rest	Chest pain and shortness of breath on mild exertion Decreasing exercise tolerance Symptoms may or may not be relieved by rest or nitrates	Sudden death, 30 minutes of severe central chest pain and shortness of breath (except silent ischaemia, eg in diabetic patients)
ECG changes	ST depression in affected territory	ST depression in affected territory. May have deep T-wave inversion	ST elevation in affected territory, leading to Q waves
Enzyme rises	Nil	Rise of troponin I carries prognostic significance	Troponin I CK, LDH
Arrhythmias	Unlikely	VF or VT likely	VF or VT likely
Management			
Acute	GTN spray Rest	MONA (**m**orphine, **o**xygen, **n**itrates, **a**spirin)	MONA + clopidogrel
Medical	Aspirin Anti-angina agents: β Blockers Calcium channel blockers Long-acting nitrates Nicorandil Risk factor modification: Statin Stop smoking Control hypertension Lose weight Modify diet	Anticoagulation Enoxaparin Heparin infusion Abciximab GP IIb IIIa inhibitors Nitrates Nitrocine™ infusion GTN infusion Statin	PCI first line Thrombolysis if PCI not available within 90 minutes (streptokinase, rTPA)
PCI	If symptoms difficult to control medically	If anticoagulation and nitrates unsuccessful	First-line therapy
CABG	If anatomy unsuitable for PCI	If procedure unsafe for PCI	Rarely

CABG, coronary artery bypass graft; GP, glycoprotein; GTN, glyceryl trinitrate; PCI, percutaneous coronary intervention; rTPA, recombinant tissue plasminogen activator; VF, ventricular fibrillation; VT, ventricular tachycardia.

Pros and cons of PCI and CABG

	PCI	CABG
Advantages	Minimally invasive Low morbidity Acceptable to patients Low immediate complication rates	Reliable revascularisation Suitable for wide range of coronary lesions Ability to perform simultaneous procedures, eg valve replacement
Disadvantages	Unsuitable for some coronary lesions (eg left main stem – LM) Early re-occlusion of angioplasty sites and in-stent thrombosis High rate of recurrence of symptoms Poorer 'freedom from medication' Requires cardiac surgical back-up in case of complications	Major procedure Morbidity from sternotomy and conduit harvesting sites Late graft failure (especially saphenous vein)
Burgeoning technologies	Drug-eluting stents Wider range of suitable targets (LM)	Minimally invasive techniques 'Off-pump' techniques Total arterial revascularisation

1.3 Preoperative considerations and cardiac surgery

Learning point

Usually performed in a pre-assessment clinic, preoperative work-up before cardiac surgery for ischaemic heart disease includes:

- History
- Examination
- Investigations
- Adjustment of medications
- Discussion of morbidity and mortality
- Discussion of treatment options

History taking before cardiac surgery

Cardiac surgical procedures are major operations performed on patients who have high levels of comorbidity. Preoperative investigations allow physiological optimisation to reduce perioperative morbidity and mortality, either through alteration of medication or exclusion of other pathology. Therefore it is wise to see the patient in a pre-assessment clinic about 1 week before surgery.