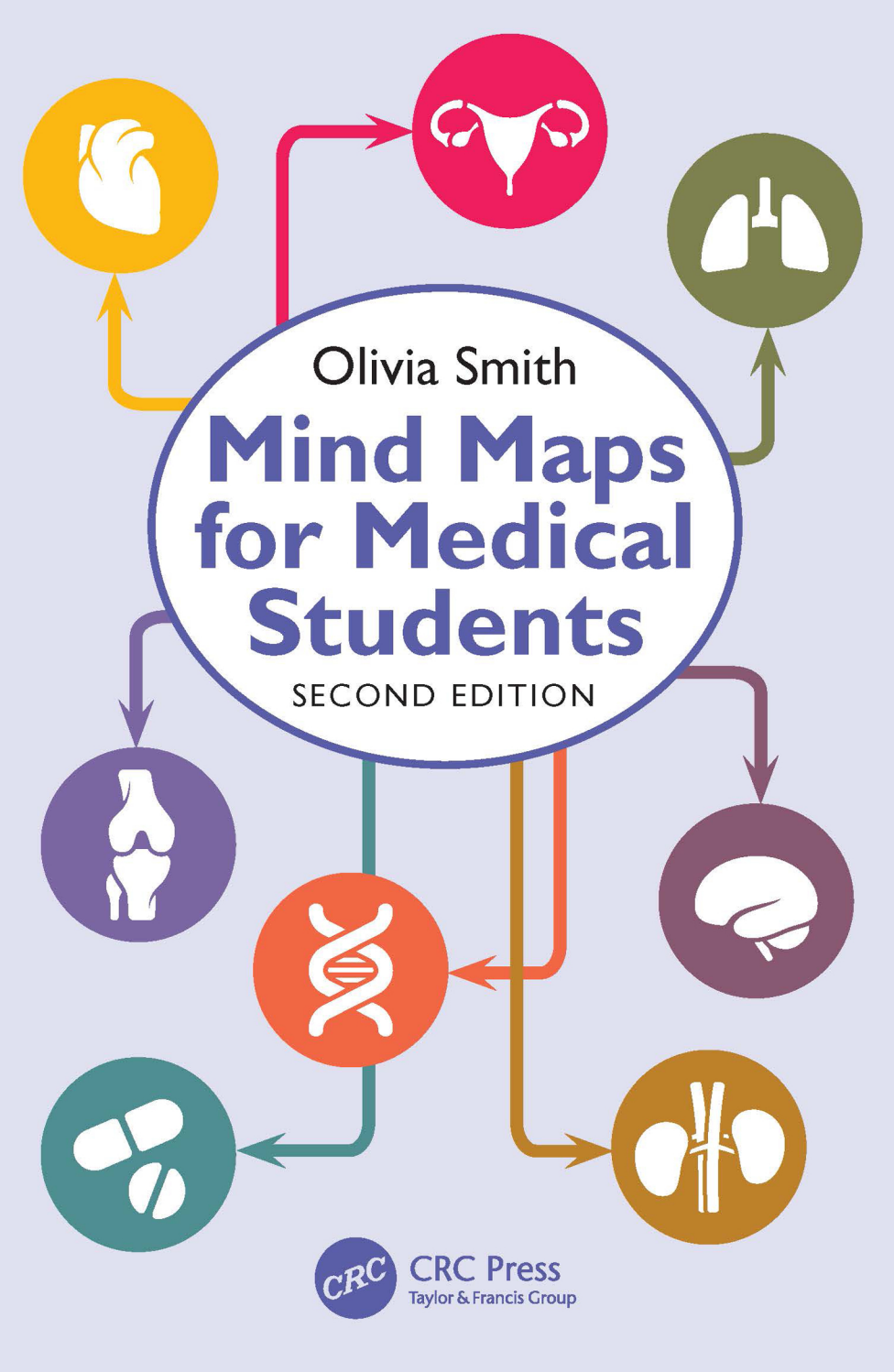
Olivia Smith
Mind Maps for Medical Students
SECOND EDITION
CRC
CRC Press
Taylor & Francis Group

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Olivia Smith

Core Surgical Trainee
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CRC Press
Taylor & Francis Group
Boca Raton London New York

CRC Press is an imprint of the
Taylor & Francis Group, an **informa** business

Second edition published 2023
by CRC Press
6000 Broken Sound Parkway NW, Suite 300, Boca Raton, FL 33487-2742

and by CRC Press
4 Park Square, Milton Park, Abingdon, Oxon, OX14 4RN

CRC Press is an imprint of Taylor & Francis Group, LLC

ISBN: 9781032201146 (hbk)
ISBN: 9781032201122 (pbk)
ISBN: 9781003262275 (ebk)

DOI: 10.1201/9781003262275

Typeset in Linotype Frutiger LT Pro
by Evolution Design & Digital

Dedication

In loving memory of my father D. J. W. Smith

Contents

How does one learn? This is the fundamental question for those who teach. To the student, however, 'How does one learn?' is the problem! This second edition of the successful *Mind Maps for Medical Students* is uniquely useful for both the teacher and student.

There seem to be a myriad of facts that fill the pages of textbooks – a myriad in each chapter! How does a student remember them, how does a student string them together to understand them and then finally recall them? In this era of booming knowledge and webs of digital connections, the volume of facts has become onerous.

In her time as an outstanding medical student at Hull York Medical School, Olivia used mind maps to create connections of knowledge and then taught this method to others. Her contributions led her to be runner up in 2016 for the 'Zeshan Qureshi Award for Outstanding Achievement in Medical Education'. Her books have been published in many languages internationally, including English, Chinese and Taiwanese. She has used this art in her research on quality of life in patients with complex abdominal wall hernias and complex abdominal wall reconstruction leading to an MD, as well as in revising and demonstrating surgical knowledge leading to successful completion of the MRCS examination.

Here, Olivia has gone back to the traditions of making the acquisition of facts simple, the connections organised and the recollection possible. Olivia's notes use mind maps – a powerful tool to aid understanding and recall while exploring myriad facts. These mind maps foster a skeleton upon which the reader can build a large body of knowledge.

I hope the readers of *Mind Maps for Medical Students* enjoy the layout and benefit from it. And that this book helps them explore the wonderful oceans of knowledge ahead. Happy surfing!

Chinta

Mr. Srinivas Chintapatla
Care Group Director – Cancer and Support Services
Consultant Surgeon (York Abdominal Wall Unit)
York Teaching Hospital NHS Foundation Trust

Preface

I am immensely grateful and humbled by the success of this book and the chance to provide a second edition. Acutely empathetic to the melancholies of medical training, it is my hope that this distillation of knowledge will help you in the intense preparation for your exams.

The book is an attempt to cover the main topics faced by medical students and presents these facts in a clear manner sufficient for final-year viva level. The work is not a substitute for larger, in-depth texts but it serves to complement them and act as an aide memoire to assist your understanding and revision.

It is not a definitive information source for students encountering a topic for the first time. However, it is a set of rapid revision notes covering a broad range of medical topics, ideally suited to students and early postgraduates revising for exams. This distillation of knowledge will save many hours of note-taking for other students. The format will appeal to those who construct their knowledge in logical sequences and the layout will allow the reader to add notes and annotations as information changes or to add a local context.

I hope that you enjoy this book and wish you all the very best of luck in your examinations and career.

Olivia Smith
MBBS(Hons), BSc(Hons), MSc(Dist), MD, PGcert, MRCS(Eng)
Core Surgical Trainee, Severn School of Surgery, UK

5-ASA	5-aminosalicylic acid
ABG	arterial blood gas
ACE	angiotensin converting enzyme
ACE-i	angiotensin converting enzyme inhibitor
ACE-III	Addenbrooke's Cognitive Examination
ACTH	adrenocorticotrophic hormone
ADH	antidiuretic hormone
ADL	activity of daily living
ADP	adenosine diphosphate
ADPKD	autosomal dominant polycystic kidney disease
AF	atrial fibrillation
Ag	antigen
AIDS	acquired immunodeficiency syndrome
AKI	acute kidney injury
ALL	acute lymphoblastic leukaemia
ALP	alkaline phosphatase
AML	acute myeloid leukaemia
ANA	antinuclear antibody
ANCA	antineutrophil cytoplasmic antibody
APLA	antiphospholipid antibody
APML	acute promyelocytic leukaemia
Apo	apolipoprotein
APP	amyloid precursor protein
APTT	activated partial thromboplastin time
ARB	angiotensin receptor blocker
ARDS	acute respiratory distress syndrome
ARPKD	autosomal recessive polycystic kidney disease
ARR	aldosterone:renin ratio
ART	antiretroviral therapy
ASD	atrial septal defect
ATP	adenosine triphosphate
ATRA	all-trans retinoic acid
AV	atrioventricular
BBB	blood–brain barrier
BMI	body mass index
BNP	brain natriuretic peptide
BP	blood pressure
BPH	benign prostatic hypertrophy
CABG	coronary artery bypass graft
CADASIL	cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy
CAPS	catastrophic antiphospholipid syndrome
CCP	cyclic citrullinated peptide
CD	cluster of differentiation
CEA	carcinoembryonic antigen
CHF	congestive heart failure
CJD	Creutzfeldt–Jakob disease
CKI	chronic kidney injury
CLL	chronic lymphocytic leukaemia
CML	chronic myeloid leukaemia
CMV	cytomegalovirus
CNS	central nervous system
COPD	chronic obstructive pulmonary disease
COX	cyclooxygenase
CRC	colorectal cancer
CREST	Calcinosis, Raynaud's disease, oEsophageal dysmotility, Sclerodactyly and Telangiectasia
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computed tomography
CTA	computed tomography angiogram

CTS	carpal tunnel syndrome
CVS	chorionic villus sampling
CXR	chest X-ray
DaTSCAN	ioflupane ^{123}I for injection
DCIS	ductal carcinoma in situ
DEXA	dual-energy X-ray scan
DFA	direct fluorescent antibody test
DHT	dihydrotestosterone
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DIP	distal interphalangeal (joint)
DLCO	diffusing capacity of the lung for carbon monoxide
DM	diabetes mellitus
DMARD	disease modifying antirheumatic drug
DNA	deoxyribonucleic acid
DPP	dipeptidyl peptidase
DVLA	Driver and Vehicle Licensing Agency
DVT	deep vein thrombosis
DWI	diffusion-weighted MRI
EBV	Epstein–Barr virus
ECG	electrocardiography
ECHO	echocardiography
EEG	electroencephalography
EIA	enzyme immunoassay
ELISA	enzyme linked immunosorbent assay
EMA	eosin-5′-maleimide
EMB	eosin methylene blue
EMG	electromyography
ENA	extractable nuclear antigen
EPEC	enteropathogenic *E. coli*
EPO	erythropoietin
ERCP	endoscopic retrograde cholangiopancreatography
ERV	expiratory reserve volume
ESKD	end-stage kidney disease
ESR	erythrocyte sedimentation rate
ESWL	extracorporeal shock wave lithotripsy
FAP	familial adenomatous polyposis
FBC	full blood count
FEV_1	forced expiratory volume
FFP	fresh frozen plasma
FISH	fluorescence in situ hybridisation
FNA	fine needle aspiration
FRC	functional residual capacity
FSH	follicle-stimulating hormone
FTA	fluorescent treponemal antibody absorption
FVC	forced vital capacity
GABA	gamma-aminobutyric acid
GBM	glomerular basement membrane
(c)GFR	(calculated) glomerular filtration rate
GH	growth hormone
GHRH	growth hormone-releasing hormone
GI	gastrointestinal
GIT	gastrointestinal tract
GLP	glucagon-like peptide
GnRH	gonadotrophin-releasing hormone
GORD	gastro-oesophageal reflux disease
Gp	glycoprotein
G6PD	glucose-6-phosphate dehydrogenase
GTN	glyceryl trinitrate
HAV	hepatitis A virus
Hb	haemoglobin
HbA1c	glycated haemoglobin

HBV	hepatitis B virus
HCC	hepatocellular carcinoma
Hct	haematocrit
HCV	hepatitis C virus
HDV	hepatitis D virus
HELLP	Haemolysis, Elevated Liver enzymes and Low Platelets
HEV	hepatitis E virus
HGPRT	hypoxanthine–guanine phosphoribosyltransferase
HHV	human herpes virus
HIV	human immunodeficiency virus
HNPCC	hereditary nonpolyposis colorectal cancer
HPV	human papilloma virus
HRT	hormone replacement therapy
HS	hereditary spherocytosis
HTLV-1	human T-lymphotrophic virus-1
HUS	haemolytic–uraemic syndrome
IBD	inflammatory bowel disease
IBS	irritable bowel syndrome
ICS	inhaled corticosteroid
ICU	intensive care unit
IFA	immunofluorescence assay
IFN	interferon
Ig	immunoglobulin
IGF	insulin-like growth factor
IL	interleukin
INR	international normalised ratio
IPSS	International Prostate Symptom Score
IRV	inspiratory reserve volume
IV	intravenous
IVU	intravenous urogram
JVP	jugular venous pressure
KUB	kidney, ureter, bladder
LABA	long-acting beta-agonist
LAMA	long-acting muscarinic antagonist
LBBB	left bundle branch block
LDH	lactate dehydrogenase
LFTs	liver function tests
LH	luteinising hormone
LHRH	luteinising hormone-releasing hormone
LMN	lower motor neuron
LMWH	low molecular weight heparin
LP	lumbar puncture
LTRA	leukotriene receptor antagonists
LVF	left ventricular failure
MALT	mucosa-associated lymphoid tissue (lymphoma)
MAO	monoamine oxidase
MCH	mean corpuscular haemoglobin
MCPJ	metacarpophalangeal joint
MCV	mean corpuscular volume
MDT	multidisciplinary team
MDS	myelodysplastic syndromes
MEN	multiple endocrine neoplasia (syndrome)
MI	myocardial infarction
MLCK	myosin light chain kinase
MMR	mumps, measles, rubella
MND	motor neuron disease
MRA	magnetic resonance angiogram
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
MS	multiple sclerosis
MSK	musculoskeletal
MTPJ	metatarsophalangeal joint
NAAT	nucleic acid amplification test
NBM	nil by mouth

NICE	National Institute for Health and Care Excellence
NMDA	N-methyl-D-aspartate
NPI	Nottingham Prognostic Index
NSAID	nonsteroidal anti-inflammatory drug
NSCC	non-small cell carcinoma
NSTEMI	non-ST elevation myocardial infarction
OA	osteoarthritis
OCP	oral contraceptive pill
OGD	oesophago-gastro-duodenoscopy
OPSI	overwhelming post-splenectomy infection
$PaCO_2$	arterial partial pressure of carbon dioxide
PaO_2	arterial partial pressure of oxygen
PAH	phenylalanine hydroxylase
PAS	periodic acid stain
PCI	percutaneous coronary intervention
PCNL	percutaneous nephrolithotomy
PCR	polymerase chain reaction
PE	pulmonary embolus
PEP	post-exposure prophylaxis
PET	positron emission tomography
PG	prostaglandin
PI	protease inhibitor
PIP	proximal interphalangeal
PL	phospholipid
PMR	polymyalgia rheumatica
PPAR	peroxisome proliferator-activated receptor
PPI	proton pump inhibitor
PR	per rectum
PSA	prostate specific antigen
PT	prothrombin time
PTH	parathyroid hormone
PTT	partial thromboplastin time
RA	rheumatoid arthritis
RAAS	renin–angiotensin–aldosterone system
RBC	red blood cell
RCC	renal cell carcinoma
RCS	Raynaud Condition Score
RDS	respiratory distress syndrome
RNA	ribonucleic acid
RPR	rapid plasma regain
RV	residual volume
RVF	right ventricular failure
SABA	short-acting beta-agonist
SAMA	short-acting muscarinic antagonist
SCC	small cell carcinoma
SERM	selective oestrogen receptor modulator
SIADH	syndrome of inappropriate antidiuretic hormone secretion
SLE	systemic lupus erythematosus
SPECT	single photon emission computed tomography
SSRI	selective serotonin reuptake inhibitor
STEMI	ST elevation myocardial infarction
STI	sexually transmitted infection
SUDEP	sudden unexplained death in epilepsy
SWL	shockwave lithotripsy
T_3	triiodothyronine
T_4	thyroxine
TB	tuberculosis
TdT	terminal deoxynucleotidyltransferase

TCC	transitional cell carcinoma
TFTs	thyroid function tests
TIA	transient ischaemic attack
TIBC	total iron binding capacity
TLC	total lung capacity
TNF	tumour necrosis factor
ToF	tetralogy of Fallot
TPHA	*Treponema pallidum* haemagglutination test
TPPA	*Treponema pallidum* particle agglutination test
TSH	thyroid-stimulating hormone
TURP	transurethral resection of the prostate
TV	tidal volume
U&Es	urine and electrolytes
UMN	upper motor neuron
UPEC	uropathogenic *E. coli*
USS	(abdominal) ultrasound scan
URS	ureteroscopy
UTI	urinary tract infection
VC	vital capacity
VEP	visual evoked potential
VDRL	Venereal Disease Research Laboratory
V/Q	ventilation/perfusion
VSD	ventricular septal defect
VWF	von Willebrand factor
VZV	varicella-zoster virus
WCC	white cell count

What is heart failure?

This may be defined as the inability of cardiac output to meet the physiological demands of the body. It can be classified in several ways:

- Left ventricular failure (LVF): symptoms of LVF: paroxysmal nocturnal dyspnoea, wheeze, nocturnal cough with pink sputum caused by pulmonary oedema.
- Right ventricular failure (RVF): symptoms of RVF, which is usually caused by LVF or lung disease, peripheral oedema and ascites.
- Low output and high output heart failure. This is due to excessive afterload, excessive preload or pump failure.

Pathophysiology

See page 4.

Causes

Anything that causes myocardial damage may lead to heart failure.

Examples include:

- Coronary artery disease.
- Hypertension.
- Atrial fibrillation.
- Valve disease.
- Cardiomyopathies.
- Infective endocarditis.
- Anaemia.
- Endocrine disorders.
- Cor pulmonale: this is right ventricular failure secondary to pulmonary disease.

Classification

Framingham Criteria for Congestive Heart Failure: 2 major criteria *or* 1 major criteria and 2 minor criteria:

- Major criteria: **PAINS**
  - **P**aroxysmal nocturnal dyspnoea.
  - **A**cute pulmonary oedema.
  - **I**ncreased heart size, Increased central venous pressure.
  - **N**eck vein dilation.
  - **S**3 gallop.
- Minor criteria: **PAIN**
  - **P**leural effusion.
  - **A**nkle oedema (bilateral).
  - **I**ncreased heart rate >120 beats/min.
  - **N**octurnal cough.

New York Heart Association Classification for Heart Failure

I: No limitation of physical activity.
II: Slight limitation of physical activity.
III: Marked limitation of physical activity.
IV: Inability to carry out physical activity.

MAP 1.1 **Heart Failure**

Treatment

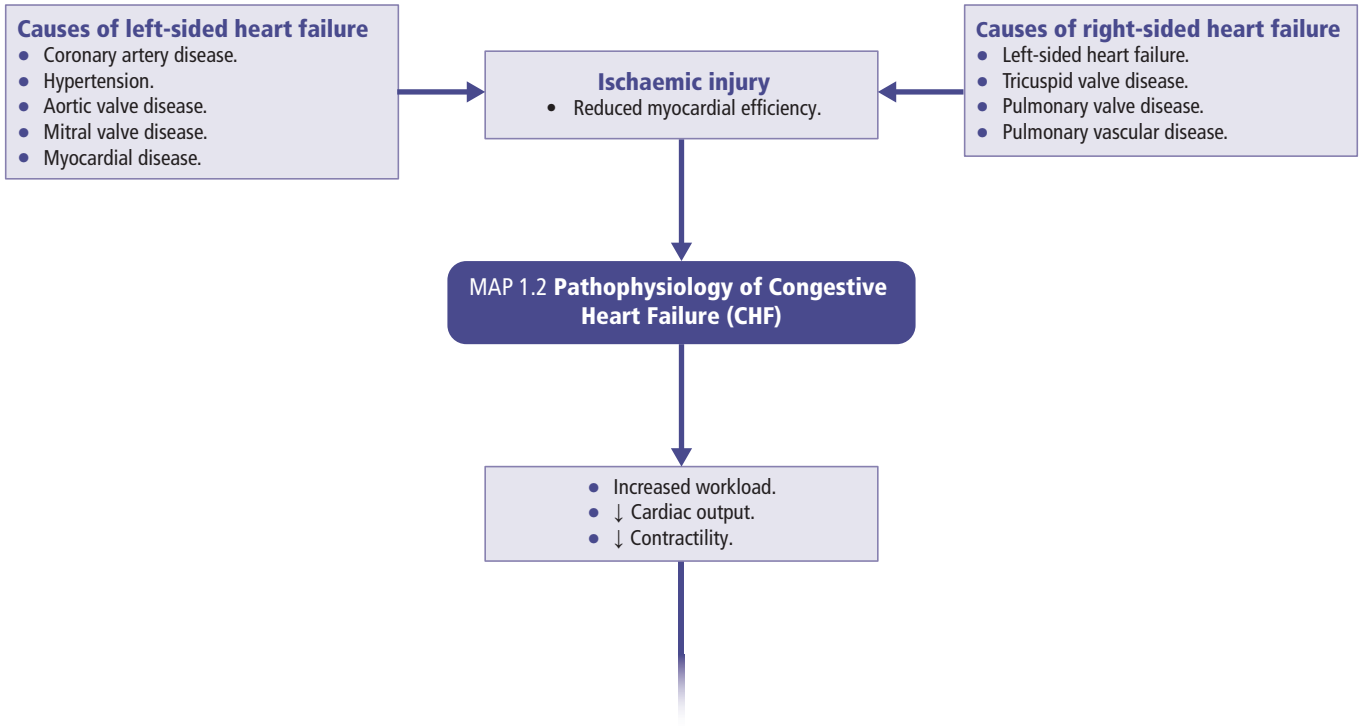
- Conservative: smoking cessation advice, weight loss, promotion of healthy diet and exercise.
- Medical: ACE-i (can be offered first line) at low dose and titrate upwards as tolerated. Do not give to those who have valvular disease until they have been assessed by a specialist. ARB is an alternative to ACE-i if ACE-i is not tolerated. Diuretics, e.g. furosemide/spironolactone, may be given to provide relief from overload.
  - Consider prescribing an antiplatelet to people with atherosclerotic arterial disease (including coronary heart disease).
    - Consider statin therapy after CVD risk assessment.
    - Refer for supervised exercise-based group rehabilitation programme for people with heart failure.
    - Offer annual flu vaccine and once-only pneumococcal vaccine.
  - T he following specialist treatments may be initiated after specialist assessment:
    - Combination of loop and thiazide diuretic.
    - Aldosterone antagonist (spironolactone or eplerenone).
    - Sacubitril valsartan.
    - Hydralazine in combination with a nitrate (especially if the person is of African–Caribbean origin).
    - Digoxin.
    - Ivabradine (slows the heart rate in sinus rhythm).
    - Anticoagulation may be indicated for people with heart failure who are in sinus rhythm and have a history of thromboembolism, left ventricular aneurysm or intracardiac thrombus.
    - Intravenous iron.
- Surgical intervention:
  - Cardiac resynchronisation therapy.
  - Insertion of an implantable cardioverter defibrillator (ICD).
  - Coronary revascularisation.
  - Cardiac transplantation.

Complications

- Arrhythmia.
- Valve dysfunction.
- Renal failure.
- Liver failure.
- Stroke.

Investigations

- Bloods:
  - FBC, U&Es, LFTs, TFTs, lipid profile.
  - BNP (brain natriuretic peptide). It suggests how much the myocytes are stretched. BNP is arguably cardioprotective as it causes Na^+ ion and H_2O excretion in addition to vasodilation. A concentration >400 pg/mL (>116 pmol/L) is suggestive of heart failure.
- CXR: **ABCDE**
  - **A**lveolar oedema.
  - K erley **B** lines.
  - **C**ardiomegaly.
  - **D**ilated upper lobe vessels.
  - pleural **E**ffusion.
- ECHO: look for ejection fraction, valve disease and regional wall motion abnormalities.
- ECG.


Causes of left-sided heart failure
Coronary artery disease.
Hypertension.
Aortic valve disease.
Mitral valve disease.
Myocardial disease.
Ischaemic injury
Reduced myocardial efficiency.
Causes of right-sided heart failure
Left-sided heart failure.
Tricuspid valve disease.
Pulmonary valve disease.
Pulmonary vascular disease.
MAP 1.2 Pathophysiology of Congestive Heart Failure (CHF)
Increased workload.
↓ Cardiac output.
↓ Contractility.

Activates compensatory mechanisms

- Activation of the renin–angiotensin–aldosterone system (RAAS) causes Na^+ ion and H_2O retention, and peripheral vasoconstriction. This increases preload.
- Activation of the sympathetic nervous system increases heart rate and causes peripheral vasoconstriction. This increases afterload.
- ↑ Myocyte size.

Chronic activation of these compensatory mechanisms worsens heart failure and leads to increased cardiac damage.

Remember that:

- The cause of cardiac dilation is increased end-diastolic volume.
- The raised jugular venous pressure (JVP) is related to right-sided heart failure and fluid overload.
- Hepatomegaly is caused by congestion of the hepatic portal circulation.

What is MI?

Also known as a heart attack. It occurs when there is myocardial necrosis following atherosclerotic plaque rupture, which occludes one or more of the coronary arteries. MI is part of the acute coronary syndromes. The acute coronary syndromes comprise:

- ST elevation MI (STEMI).
- Non-ST elevation MI (NSTEMI).
- Unstable angina.

Causes

- Type 1 MI – a primary coronary arterial event due to atherosclerosis.
- Type 2 MI – secondary to an imbalance in myocardial oxygen supply and demand without atherothrombosis, e.g. severe anaemia, coronary artery spasm.

Symptoms

- Nausea, sweating, palpitations.
- Crushing chest pain for more than 20 minutes.
- Pain radiating down left arm or into the jaw.
- Epigastric pain that is severe and may be mistaken for reflux or another upper gastrointestinal problem.
- N.B. Can be silent in diabetics.

Signs

Remember these as **RIP**:

- **R**aised jugular venous pressure (JVP).
- **I**ncreased pulse, blood pressure changes.
- **P**allor, anxiety.

Type of infarct

- Transmural:
  - Affects all of the myocardial wall.
  - ST elevation and Q waves.
- Subendocardial:
  - Necrosis of <50% of the myocardial wall.
  - ST depression.

Investigations

- ECG: this may show:
  - ST elevation, ST depression, inverted T waves.
  - New left bundle branch block (LBBB).
  - Pathological Q waves.
- CXR: this may show:
  - Cardiomegaly.
  - Pulmonary oedema.
  - Widening of the mediastinum.
- Bloods: look for cardiac biomarkers:
  - Troponin I.
  - Troponin T.
- Angiography with the view to performing percutaneous coronary intervention (PCI).

MAP 1.3
Myocardial Infarction (MI)

Pathophysiology

See page 9 for the pathophysiology of atherosclerosis.

Treatment

- Conservative: lifestyle measures such as smoking cessation and increased excercise.
- Medical – **MONA B** for immediate management:
  - **M**orphine.
  - **O**xygen (if hypoxic).
  - **N**itrates (glyceryl trinitrate [GTN]).
  - **A**nticoagulants, e.g. aspirin and an antiemetic.
  - **B**eta-blockers if no contraindication.

On discharge all patients should be prescribed: aspirin, an angiotensin converting enzyme (ACE) inhibitor, a beta-blocker (if no contraindication; calcium channel blockers are good alternatives) and a statin.

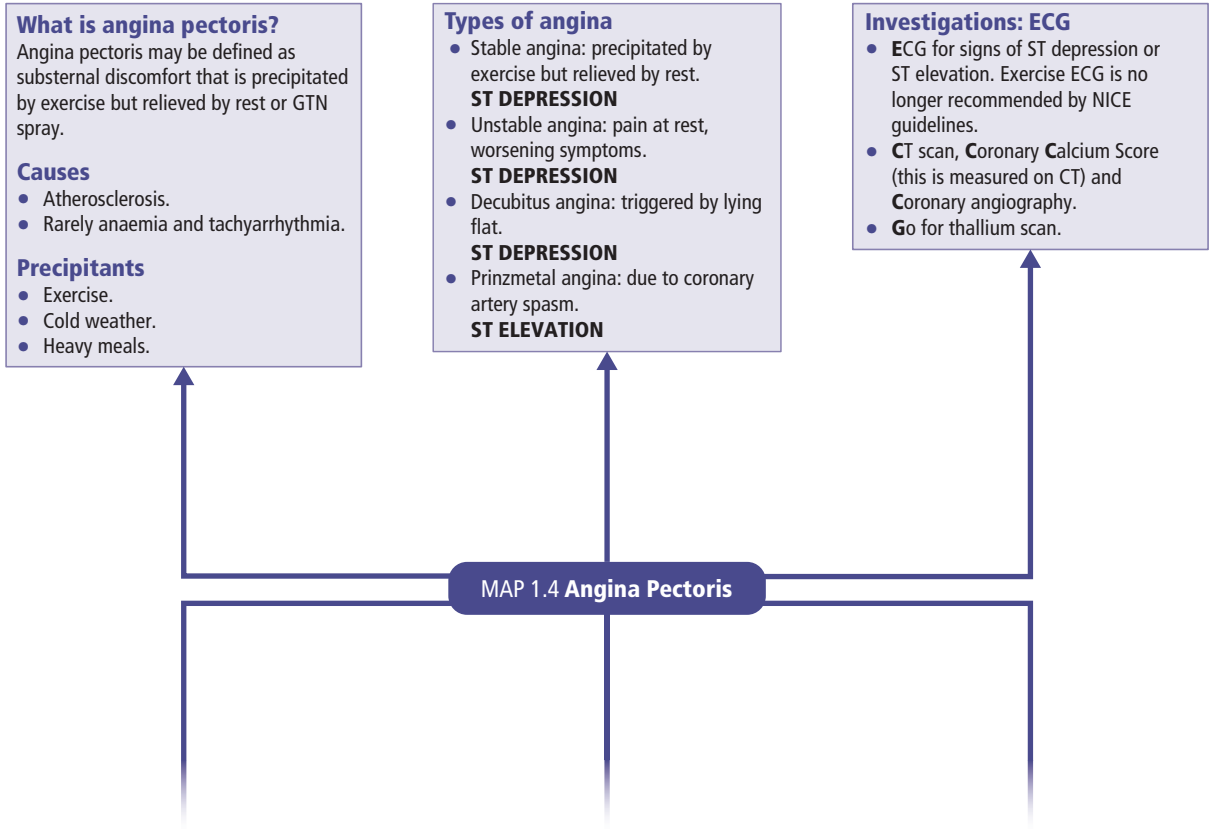
- Surgical: reperfusion with PCI if STEMI. PCI may also be used in NSTEMI but if NSTEMI patients are not having immediate PCI, fondaparinux (a factor Xa inhibitor) or a low molecular weight heparin (LMWH) may be given subcutaneously.

Complications

- Cardiogenic shock, Cardiac arrhythmia.

N.B. Atrial fibrillation (AF) increases a patient's risk of stroke. AF presents with an irregularly irregular pulse and an ECG with absent P waves, irregular RR intervals, an undulating baseline and narrow QRS complexes. Consider starting anticoagulation therapy in line with local hospital guidelines and in context with tools such as the HAS-BLED score, which estimates risk of major bleeding for patients on anticoagulation to assess risk–benefit in atrial fibrillation care.

- Pericarditis.
- Emboli.
- Aneurysm formation.
- Rupture of ventricle.
- Dressler's syndrome: an autoimmune pericarditis that develops 2–10 weeks post MI. This is a triad of: 1) fever; 2) pleuritic pain; 3) pericardial effusion.
- Rupture of free wall.
- Papillary muscle rupture.
- Sudden death.
- Hypertension.
- Ventricular septal defect.
- Frozen shoulder and shoulder–hand syndrome.


What is angina pectoris?
Angina pectoris may be defined as substernal discomfort that is precipitated by exercise but relieved by rest or GTN spray.
Causes
Atherosclerosis.
Rarely anaemia and tachyarrhythmia.
Precipitants
Exercise.
Cold weather.
Heavy meals.
Types of angina
Stable angina: precipitated by exercise but relieved by rest.
ST DEPRESSION
Unstable angina: pain at rest, worsening symptoms.
ST DEPRESSION
Decubitus angina: triggered by lying flat.
ST DEPRESSION
Prinzmetal angina: due to coronary artery spasm.
ST ELEVATION
Investigations: ECG
ECG for signs of ST depression or ST elevation. Exercise ECG is no longer recommended by NICE guidelines.
CT scan, Coronary Calcium Score (this is measured on CT) and Coronary angiography.
Go for thallium scan.
MAP 1.4 Angina Pectoris

Pathophysiology of atherosclerosis

Atherosclerosis is a slowly progressive disease and is the underlying cause of ischaemic heart disease when it occurs in the coronary arteries.

There are 3 stages of atheroma formation:

1 **Fatty streak formation**
Lipids are deposited in the intimal layer of the artery. This, coupled with vascular injury, causes inflammation, increased permeability and white blood cell recruitment. Macrophages phagocytose the lipid and become foam cells. These form the fatty streak.

2 **Fibrolipid plaque formation**
Lipid within the intimal layer stimulates the formation of fibrocollagenous tissue. This eventually causes thinning of the muscular media.

3 **Complicated atheroma**
This occurs when the plaque is extensive and prone to rupture. The plaque may be calcified due to lipid acquisition of calcium. Rupture activates clot formation and thrombosis. If the coronary artery is partially occluded the result is myocardial ischaemia and therefore angina. If the coronary artery is completely occluded then the result is myocardial necrosis and MI.

Complications

- MI.
- Stroke.

Treatment

- Conservative: modify risk factors, e.g. control cholesterol, control diabetes, smoking cessation advice, weight loss, increase exercise and control hypertension.
  Identification of patients who are at risk, e.g. using QRisk score.
- Medical:
  - Nitrates: glyceryl trinitrate (GTN) spray. Side effects include headache and hypotension.
  - **A** – **A**spirin.
  - **B** – **B**eta-blockers but contraindicated in asthma and chronic obstructive pulmonary disease (COPD).
  - **C** – Ca^{2+} antagonists especially if beta-blockers are contraindicated.
  - K^{+} channel activator, e.g. nicorandil.
- Surgery: percutaneous transluminal coronary angioplasty or coronary artery bypass graft (CABG).

What is infective endocarditis?

It is an infection of the endocardium usually involving the heart valves, with 'vegetation' of the infectious agent.

The mitral valve is more commonly affected but the tricuspid valve is implicated in IV drug users.

Risk factors

- IV drug abuse.
- Cardiac lesions.
- Rheumatic heart disease.
- Dental treatment: requires antibiotic prophylaxis.

Pathophysiology

Infective endocarditis is a rare infection that usually affects patients who already have a structural valve abnormality. The reason why heart valves are targeted is because the valves of the heart have limited blood supply and consequently white blood cells cannot reach the valves through the blood. Circulating bacteria adhere to the valve causing vegetations.

Classification of infective endocarditis

Duke criteria: 2 major criteria *or* 1 major and 3 minor criteria *or* 5 minor criteria.

- Major criteria:
  - 2 separate positive blood cultures.
  - Endocardial involvement.
- Minor criteria: **FIVE**
  - **F**ever >38°C.
  - **IV** drug user or predisposing heart condition, and
  - **I**mmunological phenomena, e.g. Osler's nodes or Roth's spots.
  - **V**ascular phenomena, e.g. mycotic aneurysm or Janeway lesions.
  - **E**chocardiograph findings.

Causative agents

- *Streptococcus viridans*.
- *Staphylococcus aureus*.
- *Staphylococcus epidermidis*.
- Diphtheroids.
- Microaerophilic streptococci.
- HACEK group: *Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikenella* and *Kingella*.

MAP 1.5
Infective Endocarditis

Investigations

- Blood cultures: take 3 separate cultures from 3 peripheral sites.
- Bloods for anaemia.
- Urinalysis; microscopic haematuria.
- CXR.
- Transoesophageal/transthoracic ECHO for vegetations.

Treatment

Depends on the causative agent. Check hospital antibiotic guidelines.

- Conservative: maintain good oral hygiene.
- Medical: follow local trust antibiotic prescribing policy but some examples of empirical therapy include **benzylpenicillin** and **gentamicin**.
  - Streptococci: **benzylpenicillin** and **amoxicillin**.
  - Staphylococci: **flucloxacillin** and **gentamicin**.
  - *Aspergillus*: **miconazole**.
- Surgical: valve repair or valve replacement.

Signs and symptoms

Remember this as **FROM JANE:**

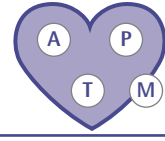
- **F**ever.
- **R**oth's spots (seen on fundoscopy).
- **O**sler's nodes (painful nodules seen on the fingers and toes).
- new **M**urmur.

- **J**aneway lesions (painless papules seen on the palms and plantars).
- **A**naemia.
- **N**ails: splinter haemorrhages.
- **E**mboli.

Complications

- Heart failure.
- Arrhythmias.
- Abscess formation in the cardiac muscle.
- Emboli formation: may cause stroke, vision loss or spread the infection to other regions of the body.

FIGURE 1.1 **Heart Valves**



Remember the heart valves as:
All Prostitutes Take Money
(Aortic, Pulmonary, Tricuspid, Mitral).

TABLE 1.1 **Aortic Valve Disease**

Valve lesion	Causes	Symptoms	Signs	Murmur	Investigations	Treatment	Complications
Aortic stenosis	Atherosclerotic-like calcific degeneration Congenital bicuspid valve Rheumatic heart disease	Syncope Dyspnoea Angina	Narrow pulse pressure Slow rising pulse	Crescendo–decrescendo ejection systolic murmur, which radiates to the carotids	ECG: left ventricular hypertrophy; AV block CXR: poststenotic dilation of the ascending aorta; may see calcification of valve on lateral view ECHO: confirms diagnosis; allows severity and valve area to be assessed	Conservative: manage cardiovascular risk factors, e.g. smoking cessation Medical: manage cardiovascular risk factors, e.g. control blood pressure Surgical: valve replacement is the treatment of choice	Sudden death Arrhythmia Heart failure Infective endocarditis
Aortic regurgitation	**Acute** Cusp rupture Connective tissue disorders, e.g. Marfan's syndrome	Dyspnoea Angina Heart failure	Waterhammer pulse Wide pulse pressure	Decrescendo early diastolic murmur	ECG: left ventricular hypertrophy	Conservative: manage cardiovascular risk factors, e.g. smoking cessation	Heart failure Arrhythmia Infective endocarditis

	Aortic dissection Perforation secondary to infection **Chronic** Rheumatoid arthritis Ankylosing spondylitis Syphilis		Traube's sign: a 'pistol shot' heard over the femoral artery De Musset's sign: head nodding in time with heart beat Quincke's sign: pulse felt in the nail Signs of systemic disease		CXR: may see cardiomegaly and pulmonary oedema if patient has heart failure ECHO: confirms diagnosis; allows severity and aortic root to be assessed	Medical: manage heart failure by following NICE guidelines Surgical: valve replacement is the treatment of choice	