

Unit 1: Cardiovascular System

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Pharmacotherapeutics-I, subject code MPP102T, is the second core theory subject of M.Pharm Pharmacy Practice, Semester 1. It carries 4 credits and 60 hours of theory, taught at 4 hours per week, for 100 marks, and its five units are of equal length at 12 hours each in the PCI scheme. Its scope, as the syllabus states it, is to enable students to understand the different treatment approaches in managing various disease conditions, and to impart skills in optimizing drug therapy by individualizing the treatment plan through evidence based medicine. Its objectives follow: to describe the rationale for drug therapy, to summarize the therapeutic approach for various disease conditions with reference to the latest evidence, to discuss clinical controversies and evidence based medicine, to prepare individualized therapeutic plans based on diagnosis, and to identify the patient specific parameters relevant in initiating and monitoring therapy. The subject head over the whole syllabus defines the method of every section: etiopathogenesis and pharmacotherapy of diseases of the following systems. The units follow the syllabus order: Unit 1 cardiovascular, Unit 2 respiratory and endocrine, Units 3 and 4 gastrointestinal and hematological, Unit 5 bone and joint, dermatological and ophthalmology. Every disease is therefore taught in the same order: what it is, what causes it, how it develops, how it presents, how it is recognised, and how its drug therapy works in principle. One discipline runs through the whole subject: these notes teach diseases and their drug therapy at standard textbook level, naming the standard drug classes and their roles and describing stepped management qualitatively. No doses or dose schedules are stated, and no patient specific advice is given. Drug therapy is always individualised on diagnosis, severity, comorbidity and response, which is the habit this subject exists to teach.

1.1 The pharmacotherapeutics method: from etiopathogenesis to an individualised plan

Pharmacotherapeutics is the study of drug therapy as it is actually chosen for a disease in a patient, and its method is the discipline that separates this subject from a list of drugs. The method begins with etiopathogenesis: the causes of the disease and the mechanism by which those causes produce the disordered physiology, because rational drug therapy is therapy aimed at a mechanism. A drug class earns its place in a treatment plan when its action interrupts, replaces or compensates for a named step in that mechanism, and the first question about any therapy in this subject is therefore which step it acts on. The second stage is presentation and diagnosis: the symptoms, signs and investigations that establish which disease is present, how severe it is, and what else will condition therapy, including other diseases, other medicines, organ function, allergy and adherence. The third stage is the therapeutic plan itself, built in the stepped pattern most chronic diseases in this subject follow: non drug measures and treatment of causes first and throughout, then drug therapy started with the class or classes the mechanism and the evidence put first, then review against stated goals, then addition, substitution or escalation where the response is inadequate, with long term therapy, prevention of complications and monitoring for adverse effects planned from the start rather than added later. Examinations reward answers that keep this order visible, and penalise answers that name drugs without the disease process they are meant to change.

The pharmacotherapeutics method used in every section

Etiopathogenesis: cause and mechanism of the disease

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Presentation and diagnosis: severity, comorbidity, organ function, other medicines, allergy, adherence

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Plan: non drug measures and causes treated throughout, first drug classes matched to the mechanism

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v

Review against goals: response, adverse effects, clinical and laboratory indices over time

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v

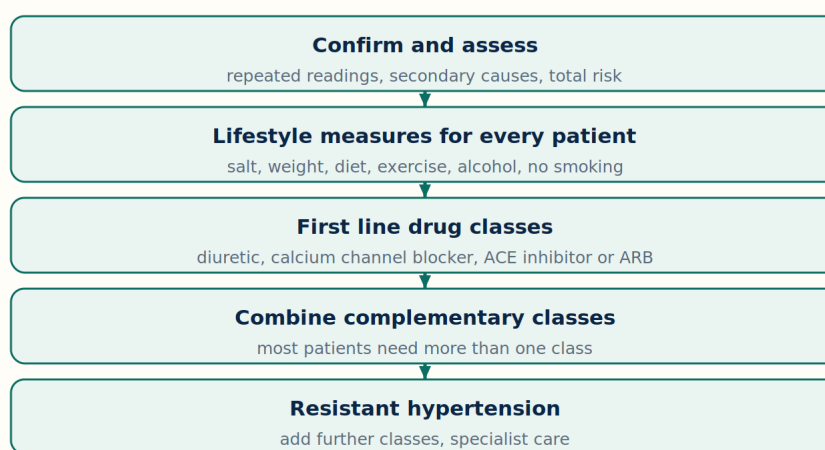
Adjust: add, substitute or escalate in steps, prevent complications, plan long term monitoring

1.2 Hypertension

Hypertension is persistently raised arterial blood pressure, in the standard textbook definition a clinic blood pressure of 140 over 90 mmHg or more on repeated measurement. Secondary hypertension has a specific cause that may be treatable in its own right: renal parenchymal and renovascular disease, endocrine causes such as primary aldosteronism, Cushing syndrome and phaeochromocytoma, thyroid disorders, coarctation of the aorta, obstructive sleep apnoea, and medicines including oral contraceptives, non steroidal anti inflammatory drugs, decongestants and corticosteroids. Young or abrupt onset, severe or resistant hypertension should always prompt the search for a secondary cause. In established hypertension resistance is raised, arterioles remodel and stiffen, and sympathetic and renin angiotensin activity sustain vasoconstriction and sodium retention. The harm is that of sustained pressure and accelerated atherosclerosis: left ventricular hypertrophy and heart failure, coronary disease, stroke, chronic kidney disease, retinopathy and peripheral arterial disease.

Hypertension is usually asymptomatic and found on measurement, so technique and repeated readings, supplemented by home or ambulatory monitoring where indicated, are the foundation of diagnosis. Diagnosis confirms sustained elevation, excludes white coat and masked patterns, grades severity, seeks secondary causes where clues exist, and assesses total cardiovascular risk and target organ damage: renal function, electrolytes, glucose, lipids, urine testing and an electrocardiogram are the standard core, because treatment intensity follows total risk, not the pressure figure alone. Pharmacotherapy rests on lifestyle measures for every patient, weight reduction, salt restriction, a diet rich in fruit, vegetables and low fat dairy produce, regular exercise, alcohol moderation and stopping smoking, and on four first line drug classes. Thiazide and thiazide like diuretics reduce sodium and volume and, with continued use, peripheral resistance. Calcium channel blockers relax arteriolar smooth muscle, dihydropyridines acting mainly on vessels and rate limiting agents also on the heart. ACE inhibitors and angiotensin receptor blockers remove angiotensin mediated vasoconstriction and aldosterone mediated retention and protect heart and kidney beyond pressure lowering. Beta blockers reduce cardiac output and renin release and are first line chiefly where a compelling cardiac indication coexists. Most patients need combination therapy, complementary classes combined, often in a single tablet to support adherence; resistant hypertension adds agents such as aldosterone antagonists in specialist hands. Monitoring follows pressure, renal function and electrolytes after renin angiotensin and diuretic changes, and class adverse effects: cough and angioedema with ACE inhibitors, ankle swelling with dihydropyridines, electrolyte disturbance with diuretics, bradycardia and bronchospasm risk with beta blockers.

Hypertension: stepped care in principle



Beta blockers are first line chiefly with a compelling cardiac indication

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Figure: Hypertension stepped care. Lifestyle measures run throughout, drug classes are combined in steps, and resistant hypertension is a specialist problem.

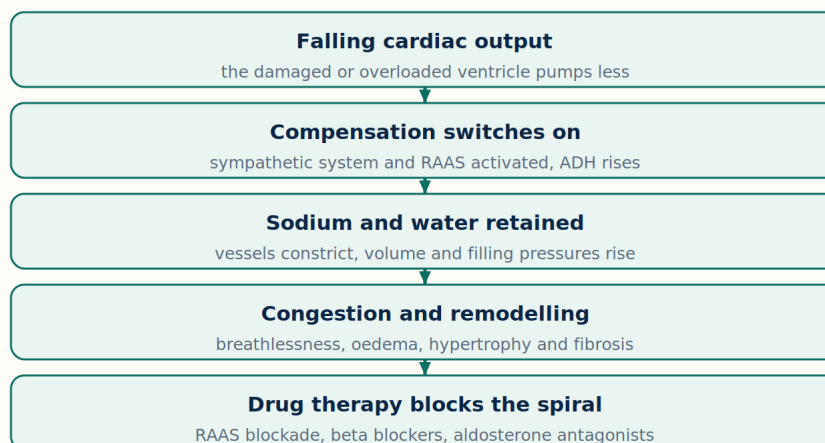
1.3 Congestive cardiac failure

Congestive cardiac failure, in modern usage heart failure, is a syndrome in which the heart cannot pump blood at a rate sufficient for the body's needs, or can do so only at raised filling pressures, so congestion in the lungs and systemic veins is its defining physical consequence. Causes are the diseases that damage, overload or stiffen the ventricle: coronary disease and previous infarction are commonest, with hypertension, valvular disease, cardiomyopathies, myocarditis, arrhythmias, alcohol, cardiotoxic medicines and systemic conditions such as thyroid disease and anemia. It is classified by ejection fraction into reduced ejection fraction failure, where the ventricle contracts weakly and dilates, and preserved ejection fraction failure, where the ventricle is stiff and relaxes poorly, usually in older patients with hypertension, obesity and diabetes; the drug evidence base is strongest in the reduced form. The pathophysiology is a compensatory spiral that becomes the disease: falling output activates the sympathetic system and the renin angiotensin aldosterone system and increases antidiuretic hormone, retaining sodium and water and constricting vessels; chronically these overload the ventricle, drive hypertrophy, dilation and fibrosis, provoke arrhythmias and damage the kidneys. Natriuretic peptides, released from the stretched heart wall, oppose this system, and one modern class exists to strengthen them. Congestion explains the presentation: exertional and then resting breathlessness, orthopnoea and paroxysmal nocturnal dyspnoea, fatigue, ankle oedema, raised jugular venous pressure and, in acute decompensation, pulmonary oedema. Diagnosis combines the syndrome with examination, chest imaging, electrocardiogram, echocardiography defining structure, function and cause, natriuretic peptide measurement, and tests for cause and precipitants such as anemia, thyroid disease, infection and non adherence.

Pharmacotherapy in reduced ejection fraction failure is built on classes that block the maladaptive neurohormonal systems, because those classes improve survival, not only symptoms. Renin angiotensin inhibition, by an ACE inhibitor, an angiotensin receptor blocker, or the angiotensin receptor and neprilysin inhibitor combination pairing renin angiotensin blockade with natriuretic peptide strengthening, is foundational. The beta blockers proven in this condition slow the heart, protect against arrhythmia and reverse remodelling. Aldosterone antagonists block aldosterone's fibrotic and potassium losing actions. SGLT2 inhibitors, a class that began in diabetes care, reduce hospitalisation and death across the ejection fraction range and are now standard. Loop diuretics relieve congestion and are adjusted to fluid state, but are not themselves survival therapy. Further options in selected patients include hydralazine with a nitrate,

digoxin, and device therapy where indicated. Management also treats cause and precipitants, and monitors weight, renal function and electrolytes, since the classes that protect the heart stress the kidney and potassium balance.

Heart failure: the neurohormonal spiral



Diuretics relieve the congestion; neurohormonal blockade improves survival

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Figure: The heart failure spiral. The compensations that restore pressure in the short term produce congestion and remodelling in the long term, and the survival drug classes block them.

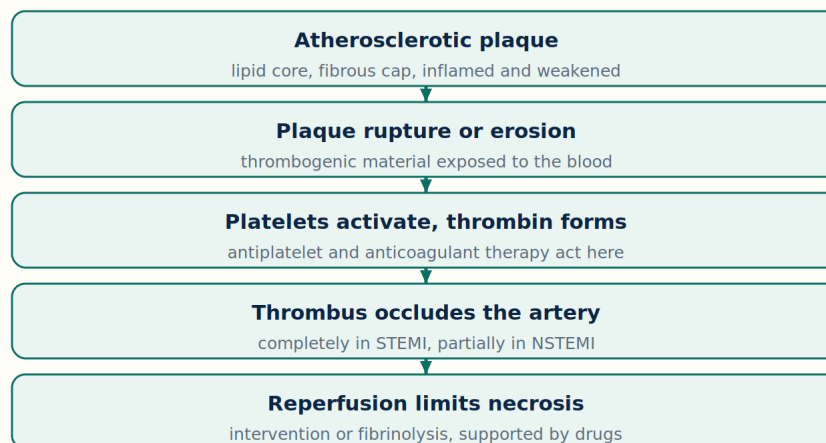
1.4 Acute coronary syndrome

Acute coronary syndrome is the spectrum of acute myocardial ischaemia caused by sudden disruption of a coronary atherosclerotic plaque: unstable angina, ischaemia without myocardial necrosis, and myocardial infarction, necrosis present, divided electrocardiographically into ST elevation infarction, with complete occlusion, and non ST elevation infarction, with partial or transient occlusion. Atherosclerosis develops over decades: endothelial injury and lipid accumulation form a plaque with a lipid core and fibrous cap, inflammation weakens the cap, and the precipitating event is plaque rupture or erosion exposing thrombogenic material. Platelets adhere and activate, coagulation generates thrombin and fibrin, and thrombus may occlude the artery completely, partially, or embolise downstream. Consequences follow the duration and territory of ischaemia: necrosis in a time dependent wavefront, electrical instability, pump failure and cardiogenic shock in large infarcts, and mechanical complications in the days that follow. Presentation is typically central chest pain or discomfort radiating to arm, neck or jaw, with sweating, nausea and breathlessness at rest or on minimal exertion; presentations are atypical more often in women, older patients and patients with diabetes, and some infarctions are silent. Diagnosis is urgent and structured: immediate electrocardiogram, which decides the ST elevation pathway, serial cardiac troponin, the biomarker of necrosis whose rise and fall defines infarction, risk assessment, and exclusion of mimics such as aortic dissection and pulmonary embolism where the picture suggests them.

Pharmacotherapy has two simultaneous aims: to limit necrosis by restoring flow, and to prevent thrombus extension and recurrence. Reperfusion is decisive in ST elevation infarction, by primary percutaneous coronary intervention where available in time or fibrinolytic therapy where it is not, and drug therapy supports it. Antiplatelet therapy pairs aspirin, blocking thromboxane mediated activation, with a P2Y₁₂ receptor blocker, blocking ADP mediated activation; the dual combination protects the artery and any stent placed in it. Anticoagulation with heparin class agents in the acute phase prevents thrombus propagation. Nitrates relieve ischaemic pain and preload, beta blockers reduce myocardial oxygen demand where not contraindicated, high intensity statin therapy stabilises plaque and begins the long term lipid strategy of

Section 1.6, and renin angiotensin inhibition follows in patients with ventricular impairment, hypertension or diabetes. Oxygen is given only where saturation is low, a standard examination point. Long term secondary prevention, continued antiplatelet therapy, statin and risk factor treatment including smoking cessation, is part of the same answer, because the syndrome declares a systemic arterial disease.

Acute coronary syndrome: plaque to thrombus



Time is muscle: the longer the occlusion, the larger the infarct

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Figure: From plaque rupture to reperfusion. Antiplatelet and anticoagulant therapy act on the thrombus steps, reperfusion on the occlusion itself.

1.5 Arrhythmias

Arrhythmias are disorders of heart rate, rhythm or conduction, arising from abnormal impulse formation, abnormal conduction, or re entry circuits in which an impulse circles repeatedly through tissue with differing conduction and refractory properties. The standard classification divides them by rate and site: bradyarrhythmias, including sinus bradycardia and the degrees of heart block, and tachyarrhythmias, divided into supraventricular forms, including atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia, and ventricular forms, ventricular tachycardia and fibrillation, which are life threatening. Causes span ischaemic and structural heart disease, hypertension, heart failure, thyroid disease, electrolyte disturbance, infection, alcohol and medicines; several antiarrhythmic and non cardiac drugs themselves prolong repolarisation and provoke arrhythmia, a drug induced mechanism the pharmacist must watch for. Diagnosis rests on capturing the rhythm, a 12 lead electrocardiogram during symptoms and longer monitoring where episodes are intermittent, with blood tests for precipitants and assessment of the underlying heart. Atrial fibrillation, the commonest sustained arrhythmia, deserves its own emphasis: loss of organised atrial contraction causes an irregularly irregular pulse and, more importantly, atrial stasis and a substantial embolic stroke risk, so its management always has two parts, rate control or rhythm restoration, and anticoagulation assessed against stroke and bleeding risk.

Pharmacotherapy follows the mechanism and the aim. For rate control in atrial fibrillation, beta blockers and rate limiting calcium channel blockers slow atrioventricular nodal conduction, digoxin being an additional agent in sedentary patients and in heart failure. Antiarrhythmic drugs are classified by dominant action in the Vaughan Williams scheme: Class I agents block sodium channels, Class II are beta blockers, Class III agents block potassium channels and prolong repolarisation and the refractory period, Class IV are the rate limiting calcium channel blockers, with digoxin, adenosine and magnesium outside the scheme, adenosine being the acute terminator of paroxysmal supraventricular tachycardia by brief atrioventricular block.

1.6 Hyperlipidemias

The hyperlipidemias are raised levels of the blood lipids carried in lipoproteins: raised low density lipoprotein cholesterol, the principal atherogenic fraction, raised triglycerides, and often low high density lipoprotein cholesterol. They are primary, the familial and polygenic disorders, of which familial hypercholesterolaemia, an inherited defect of low density lipoprotein clearance presenting young with tendon xanthomas and premature coronary disease, is the standard example, or secondary, where diabetes, hypothyroidism, nephrotic syndrome, cholestasis, obesity, alcohol and medicines including thiazides and corticosteroids raise lipids and must be sought and treated. The pathophysiology is the atherosclerosis of Section 1.4 read from the lipid side: low density lipoprotein enters the arterial wall, is retained and modified, is taken up by macrophages to form foam cells, and builds the plaque whose rupture causes acute events; triglyceride rich remnants contribute to the same process, and severe hypertriglyceridaemia adds a distinct danger, acute pancreatitis. Hyperlipidemia is silent, found by testing and presenting through its complications, and is assessed as part of total cardiovascular risk, with a lipid profile, exclusion of secondary causes and family history.

Pharmacotherapy is matched to the fraction raised and the risk carried. Lifestyle measures, a diet reduced in saturated fat, weight management, exercise, alcohol moderation and smoking cessation, apply to every patient and continue with drug therapy. Statins, inhibitors of HMG CoA reductase, the rate limiting enzyme of cholesterol synthesis, are first line: reducing hepatic cholesterol raises hepatic low density lipoprotein receptor expression and clears low density lipoprotein from the blood, and their prevention evidence is the strongest in this section. Ezetimibe, which blocks intestinal cholesterol absorption, is added where the statin response is insufficient or the statin is not tolerated. PCSK9 directed therapies preserve low density lipoprotein receptors from degradation and give large additional reductions, reserved for high risk and familial patients under specialist care. Bile acid sequestrants bind bile acids in the gut, an older class limited by gastrointestinal intolerance and interactions. For triglycerides, fibrates activate PPAR alpha and increase lipoprotein lipase activity, with prescription omega 3 preparations an additional option; severe elevation is urgent in its own right because of pancreatitis. Monitoring covers lipid response and the liver and muscle adverse effects of statins, muscle symptoms being the commonest reason for intolerance, and interactions, since several statins share metabolic pathways with widely used inhibitors.

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