BP104T · B.PHARM THEORY

BP104T Human Anatomy, Physiology and Pathophysiology I

Unit V — Cardiovascular System

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From-zero teaching standard: every technical term is defined before use.

Syllabus coverage: Unit V (12 hours).

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CHAPTER 13: THE CARDIOVASCULAR SYSTEM — ANATOMY AND PHYSIOLOGY

Unit V · ~6 hours

13.1 Introduction

Your heart is a fist-sized pump that has been beating since before you were born and will beat about 3 billion times in your lifetime — without a single rest. Around it runs 100,000 kilometres of blood vessels, a plumbing network that delivers oxygen and food to every cell and carries waste away. This chapter builds the whole system from zero: the pump, the pipes, the electrical wiring that times each beat, and the controls that adjust pressure moment to moment.

Learning objectives — after studying this chapter, you should be able to:

- describe the anatomy of the heart: chambers, valves, and wall layers;
- trace systemic, pulmonary, and coronary circulation;
- compare arteries, veins, and capillaries in structure and function;
- name the elements of the heart's conduction system and explain how a heartbeat is generated;
- explain how the autonomic nervous system regulates the heartbeat;
- define cardiac output and describe the phases of the cardiac cycle;
- explain the regulation of blood pressure, what the pulse is, and the basic waves of the ECG.

13.2 Definitions

> **Scientific definition:** The cardiovascular system consists of the heart (a muscular pump), blood vessels (arteries, veins, capillaries), and blood, which together transport oxygen, nutrients, hormones, and waste products throughout the body.

> **Simple explanation:** It is the body's delivery-and-cleanup network — the heart is the pump, the arteries and veins are the delivery roads, and blood is the fleet of trucks carrying goods to every house (cell) and bringing rubbish back.

Cardiac = relating to the heart (from the Greek *kardia*, heart).

13.3 Key Terminology

Term	Meaning	Simple explanation
Atrium (plural: atria)	Upper chamber of the heart that receives blood	The "receiving room"
Ventricle	Lower chamber of the heart that pumps blood out	The "pumping room" — thicker walls
Valve	One-way flap preventing backflow of blood	A "one-way door"
Systole	Phase of heart-muscle contraction	The "squeeze"
Diastole	Phase of heart-muscle relaxation	The "rest and refill"
Cardiac output	Volume of blood pumped by a ventricle per minute	"Litres per minute the pump delivers"
Stroke volume	Volume pumped per single beat	"Litres per squeeze"
Blood pressure	Force of blood against vessel walls	"How hard the blood pushes the pipes"
Pulse	The pressure wave felt in an artery with each heartbeat	The "throb" you feel at the wrist
ECG (electrocardiogram)	Recording of the heart's electrical activity	The "electrical signature" of each beat

13.4 Detailed Explanation

13.4.1 Anatomy of the heart — chambers, valves, layers

Location and size: The heart sits in the chest between the lungs, slightly to the left, about the size of your closed fist. It is enclosed in a protective sac called the **pericardium**.

Wall layers (outside → inside):

1. **Pericardium** — the outer sac (fibrous outer layer + two thin serous layers with lubricating fluid, so the heart slides smoothly as it beats).
2. **Epicardium** — the thin outer surface of the heart muscle itself.
3. **Myocardium** — the thick middle layer of **cardiac muscle**; this is the actual pump. Thickest in the left ventricle (it works hardest).
4. **Endocardium** — the smooth inner lining; its smoothness lets blood flow without clotting.

Chambers — four rooms, two sides:

- **Right atrium (RA)** — receives oxygen-poor blood returning from the body (via the superior and inferior vena cava).
- **Right ventricle (RV)** — pumps that blood to the **lungs** (via the pulmonary artery).
- **Left atrium (LA)** — receives oxygen-rich blood returning from the lungs (via pulmonary veins).
- **Left ventricle (LV)** — pumps oxygen-rich blood to the **whole body** (via the aorta). Its wall is the thickest — about three times thicker than the right ventricle's — because it pushes blood through the entire body.

Valves — the one-way doors (four):

- **Tricuspid valve** — between RA and RV (three flaps).
- **Bicuspid (mitral) valve** — between LA and LV (two flaps).
- **Pulmonary semilunar valve** — between RV and pulmonary artery.
- **Aortic semilunar valve** — between LV and aorta.
- Valves open and close with pressure changes; their closing makes the **heart sounds** ("lubb" = AV valves closing at the start of ventricular systole; "dubb" = semilunar valves closing at the start of diastole). Leaky or narrowed valves cause **murmurs**.

13.4.2 Blood circulation — three circuits

Pulmonary circulation (heart ↔ lungs): RV → pulmonary artery → lungs (picks up oxygen, drops carbon dioxide) → pulmonary veins → LA. *Note the naming twist: the pulmonary artery carries oxygen-poor blood and pulmonary veins carry oxygen-rich blood — "artery" and "vein" are defined by direction (away from / towards the heart), not by oxygen content.*

Systemic circulation (heart ↔ rest of body): LV → aorta → body tissues (delivers oxygen, collects waste) → vena cava → RA. This is the big circuit serving every organ.

Coronary circulation (heart feeding itself): The heart muscle needs its own blood supply — the **right and left coronary arteries** branch off the aorta just above the aortic valve and feed the myocardium; **coronary veins** drain back to the right atrium. Blockage here causes a heart attack (see Chapter 14).

13.4.3 Blood vessels — artery, vein, capillary

Feature	Artery	Vein	Capillary
Direction	Carries blood AWAY from heart	Carries blood TOWARDS heart	Connects smallest arteries to smallest veins
Wall	Thick, muscular, elastic	Thin, less muscle; has VALVES	One cell thick (endothelium only)
Pressure	High	Low	Very low
Function	Withstand pumping pressure; stretch and recoil	Return blood to heart; act as blood reservoir	Exchange of oxygen, nutrients, waste with tissues
Blood type	Mostly oxygen-rich (except pulmonary artery)	Mostly oxygen-poor (except pulmonary veins)	Mixed — exchange happens here
Example	Aorta, coronary arteries	Vena cava, saphenous vein	Networks in every tissue

Why the differences make sense: arteries need thick elastic walls to survive the heart's pounding; veins need valves because low-pressure blood flowing uphill (legs → heart) would fall back without them; capillaries must be paper-thin so gases and nutrients can diffuse across.

13.4.4 Conduction system of the heart — the electrical wiring

Heart muscle contracts when an electrical signal sweeps through it. The heart generates its own signal — it does not wait for the brain. The specialised pathway:

1. **SA node (sinoatrial node)** — the natural **pacemaker**, in the right atrium wall. It fires spontaneously about 70–75 times per minute, starting each heartbeat. The signal spreads across both atria → atria contract.
2. **AV node (atrioventricular node)** — between atria and ventricles. It **delays** the signal briefly (~0.1 second) so the ventricles fill completely before they contract.
3. **Bundle of His (AV bundle)** — carries the signal down the wall between the ventricles.
4. **Right and left bundle branches** — split the signal to each ventricle.
5. **Purkinje fibres** — spread the signal rapidly through the ventricular muscle → ventricles contract from the bottom up, squeezing blood upward into the arteries.

Order to remember: **SA → AV → Bundle of His → bundle branches → Purkinje fibres.**

13.4.5 Regulation of heartbeat by the autonomic nervous system

The SA node sets the basic rhythm, but the ANS adjusts it to the body's needs:

- **Sympathetic** (fight or flight) → releases noradrenaline → SA node fires faster, heart muscle squeezes harder → heart rate and force INCREASE (exercise, fear, fever).
- **Parasympathetic** (via the **vagus nerve**, X) → releases acetylcholine → SA node slows → heart rate DECREASES (rest, sleep, digestion).
- This is why your pulse is ~70 at rest but can exceed 150 during hard exercise — same pump, different throttle setting.

13.4.6 Cardiac output

Cardiac output (CO) = the volume of blood one ventricle pumps per minute. **CO = Stroke volume × Heart rate.**

- **Stroke volume (SV)** = blood pumped per beat (~70 mL at rest — textbook standard).
- **Heart rate (HR)** = beats per minute (~72 at rest).
- So resting CO ≈ 70 mL × 72 = ~5,000 mL/min ≈ **5 litres per minute** — roughly the entire blood volume circulated every minute. During heavy exercise it can rise several-fold (faster rate + stronger squeeze).

13.4.7 The cardiac cycle — phases of one heartbeat

One full cycle (one heartbeat) lasts about **0.8 seconds** at 75 beats/min and has three main phases:

1. **Atrial systole (0.1 s)** — atria contract, pushing the last bit of blood into the ventricles. AV valves open.
2. **Ventricular systole (0.3 s)** — ventricles contract powerfully.

- First the AV valves slam shut ("lubb") so blood cannot flow back into the atria. - Then pressure builds until the semilunar valves open and blood is ejected into the pulmonary artery and aorta.

3. **Diastole / general relaxation (0.4 s)** — everything relaxes.

- Semilunar valves snap shut ("dubb") preventing backflow from the arteries. - AV valves reopen; ventricles refill from the atria. The heart muscle itself receives its coronary blood supply mainly during this phase.

Key logic: systole = squeeze and eject; diastole = relax and refill. The heart spends more time resting (diastole) than working — that is how it beats for a lifetime.

13.4.8 Regulation of blood pressure

Blood pressure must stay in a narrow range — high enough to reach the brain against gravity, low enough not to burst vessels. Three control layers:

A. Nervous (fast — seconds): baroreceptor reflex.

- **Baroreceptors** (pressure sensors) in the carotid arteries and aorta detect pressure changes and report to the brainstem's **vasomotor centre**.
- Pressure too high → parasympathetic slows the heart + vessels dilate → pressure falls.
- Pressure too low (e.g., standing up suddenly) → sympathetic speeds the heart + vessels constrict → pressure rises. (When this reflex lags, you feel dizzy on standing — **orthostatic hypotension**, relevant to several BP drugs.)

B. Hormonal (slower — minutes to hours):

- **RAAS (renin–angiotensin–aldosterone system):** kidneys sense low pressure → release **renin** → produces **angiotensin II** (a powerful vessel constrictor) → triggers **aldosterone**, which makes kidneys retain salt and water → blood volume rises → pressure rises. Many BP medicines block this chain (ACE inhibitors, ARBs).
- **ADH (antidiuretic hormone):** retains water → raises blood volume and pressure.
- **Adrenaline/noradrenaline:** from sympathetic activation/adrenal medulla → faster heart + constricted vessels → pressure rises.

C. Kidney–fluid balance (slow — hours to days): long-term pressure follows blood volume, which the kidneys control by adjusting salt and water excretion.

13.4.9 The pulse

The **pulse** is the pressure wave you feel when an artery near the skin expands with each ventricular systole. It is normally equal to the heart rate. Common sites: **radial** (wrist — most used), carotid (neck), brachial, femoral, popliteal, dorsalis pedis. Pharmacists check the pulse before dispensing some heart medicines (e.g., digoxin — not given if the pulse is too slow).

13.4.10 Electrocardiogram — basic waves

The **ECG** records the heart's electrical activity from electrodes on the skin. One heartbeat produces three characteristic deflections:

- **P wave** — small bump: **atrial depolarisation** (atria electrically activated, about to contract).
- **QRS complex** — tall sharp spike: **ventricular depolarisation** (ventricles activated; they contract just after). It hides atrial repolarisation.
- **T wave** — rounded bump: **ventricular repolarisation** (ventricles electrically resetting for the next beat).

Reading logic: P before every QRS = normal rhythm starting in the SA node. Missing/abnormal P waves, widened QRS, or ST-segment shifts point to conduction problems or heart-muscle injury (ST elevation is the classic sign of a heart attack — Chapter 14). You are not expected to diagnose from ECGs — only to know what each wave represents.

13.5 Easy Analogy

> The cardiovascular system is a **city water supply**. The **heart** is the central pumping station with four tanks (two receiving, two pumping) and one-way valves so water never flows backwards. The **arteries** are the high-pressure mains (thick pipes), the **veins** are the return drains (with non-return valves where water must flow uphill), and the **capillaries** are the tiny house connections where water is actually delivered and wastewater collected. The **SA node** is the station's automatic timer, the **ANS** is the control room turning pressure up or down, and the **ECG** is the meter recording the pump's electrical pattern.

13.6 Diagrams (describe in PDF)

1. **Heart anatomy** — external and cut-open views labelled: RA, RV, LA, LV, tricuspid/mitral/pulmonary/aortic valves, aorta, pulmonary artery/veins, vena cava, pericardium, myocardium; arrows showing blood flow direction through all four chambers.
2. **Circulation map** — three colour-coded loops: pulmonary (heart ↔ lungs), systemic (heart ↔ body), coronary (aorta → heart muscle → right atrium).
3. **Vessel wall comparison** — cross-sections of artery (thick muscle/elastic), vein (thin wall + valve), capillary (single-cell wall) side by side.
4. **Conduction system** — heart outline with SA node, AV node, bundle of His, bundle branches, Purkinje fibres in firing order, numbered 1–5.
5. **Cardiac cycle timeline** — 0.8 s bar divided into atrial systole / ventricular systole / diastole, marking "lubb", "dubb", valve positions, and when coronary filling occurs.
6. **ECG strip** — labelled P wave, QRS complex, T wave with "atrial depolarisation / ventricular depolarisation / ventricular repolarisation" captions.
7. **BP regulation flowchart** — baroreceptor reflex loop and RAAS cascade (renin → angiotensin II → aldosterone → salt/water retention → BP up).

13.7 Pharmacy Relevance

- **BP measurement** is a core pharmacy skill — many Indian pharmacies now offer it; correct cuff size and patient rest matter for accuracy.
- **Drug logic from this chapter:** ACE inhibitors/ARBs block the RAAS chain; beta-blockers blunt sympathetic stimulation of the heart; calcium-channel blockers relax vessel muscle; diuretics reduce blood volume via the kidneys — every major BP drug class maps to a control point you just learned.
- **Pulse check before dispensing:** some heart drugs (e.g., digoxin, certain beta-blockers) are withheld if the pulse is too slow — the pharmacist is the safety checkpoint.
- **Nitroglycerin** (angina) dilates coronary and systemic vessels — patients must sit down when taking it (BP can dip suddenly).
- **Counselling:** "take BP medicine even when you feel fine" — hypertension has no symptoms but the vessel damage continues silently.

13.8 India Context

- Community pharmacies and health camps across India routinely screen blood pressure — pharmacists are often the first to detect hypertension in asymptomatic adults.
- Rheumatic heart disease (valve damage following untreated streptococcal throat infection in childhood) is still seen in India and is a preventable cause of valve disorders — prompt antibiotic treatment of sore throat matters.
- The government's Ayushman Bharat Health and Wellness Centres include BP screening as a core service, creating a growing role for pharmacists in follow-up and adherence support.

13.9 High-Yield Points

- Four chambers: RA → RV → lungs → LA → LV → body. Valves: tricuspid (RA–RV), mitral/bicuspid (LA–LV), pulmonary and aortic semilunar.
- Wall layers: pericardium → epicardium → myocardium (thickest in LV) → endocardium. Heart sounds: "lubb" (AV valves shut), "dubb" (semilunar shut).
- Artery = thick, elastic, high pressure, away from heart; vein = thin, valves, towards heart; capillary = one cell thick, exchange site.
- Conduction order: SA node (pacemaker) → AV node (delay) → bundle of His → bundle branches → Purkinje fibres.
- Sympathetic speeds the heart (noradrenaline); parasympathetic via vagus slows it (acetylcholine).
- Cardiac output = stroke volume × heart rate ≈ 5 L/min at rest.
- Cardiac cycle ≈ 0.8 s: atrial systole → ventricular systole → diastole (coronary filling happens in diastole).
- BP control: baroreceptor reflex (fast), RAAS + ADH + adrenaline (hormonal), kidney fluid balance (slow).
- ECG: P = atrial depolarisation, QRS = ventricular depolarisation, T = ventricular repolarisation.

13.10 Common Confusions

- **Pulmonary artery/vein oxygen content:** the pulmonary ARTERY carries oxygen-POOR blood (it goes away from the heart to the lungs); pulmonary VEINS carry oxygen-RICH blood. Artery/vein = direction, not oxygen.
- **"Lubb-dubb":** "lubb" = AV (tricuspid/mitral) valves closing at the START of ventricular systole; "dubb" = semilunar valves closing at the START of diastole. Not the reverse.
- **SA node vs AV node:** SA STARTS the beat (pacemaker); AV DELAYS it (lets ventricles fill). Students swap their jobs.
- **Systole vs diastole:** systole = contraction/ejection; diastole = relaxation/filling. Coronary arteries fill during DIASTOLE (when the muscle is relaxed).
- **Cardiac output vs stroke volume:** stroke volume = per BEAT; cardiac output = per MINUTE (SV × HR).

13.11 Common Mistakes

- Writing that veins always carry oxygen-poor blood (pulmonary veins are the exception).
- Saying the left ventricle wall is thin (it is the THICKEST — it pumps to the whole body).
- Placing the P wave as ventricular activity (P = atrial; QRS = ventricular).
- Forgetting the AV nodal delay — it is the reason atria finish emptying before ventricles contract.
- Describing blood pressure regulation with only the nervous system and omitting RAAS/kidneys.

13.12 Mnemonic

Valves in blood-flow order: *"Toilet Paper My Ass"* — **T**ricuspid → **P**ulmonary → **M**itral → **A**ortic (RA → RV → lungs → LA → LV → body).

Conduction order: *"SA-AV-His-Bundle-Purkinje"* — *"**S**end **A** **V**an **H**ome **B**efore **P**arty"* — SA node → AV node → His → Bundle branches → Purkinje.

ECG waves: *"**P**umps **A**tria, **QRS** **Q**uite **R**apidly **S**queezes ventricles, **T**ime to **T**est-reset"* — P = atrial depolarisation, QRS = ventricular depolarisation, T = ventricular repolarisation.

13.13 Chapter Summary

- The heart is a four-chambered double pump: right side sends blood to the lungs (pulmonary circuit), left side sends it to the body (systemic circuit); the coronary circuit feeds the heart muscle itself.
- Four one-way valves (tricuspid, pulmonary, mitral, aortic) keep flow forward; their closure makes the "lubb-dubb" sounds. Wall layers run pericardium → epicardium → myocardium → endocardium.
- Arteries (thick, elastic, high pressure), veins (thin, valved, low pressure), and capillaries (one cell thick, exchange site) form the vessel network.
- The heartbeat starts at the SA node (pacemaker), pauses at the AV node, then races down the bundle of His, bundle branches, and Purkinje fibres.
- Sympathetic action speeds and strengthens the beat; parasympathetic (vagus) slows it. Cardiac output = stroke volume × heart rate (≈5 L/min at rest).
- The 0.8-second cardiac cycle runs atrial systole → ventricular systole → diastole. Blood pressure is regulated by the baroreceptor reflex (fast), hormones including the RAAS (medium), and kidney fluid balance (slow). The pulse mirrors the heart rate; the ECG shows P (atrial depolarisation), QRS (ventricular depolarisation), T (ventricular repolarisation).

13.14 One-Minute Revision

Heart = 4 chambers (RA→RV→lungs→LA→LV→body), 4 valves (tricuspid, pulmonary, mitral, aortic — "Toilet Paper My Ass"), wall thickest at LV. Arteries thick/high-pressure/away; veins thin/valved/towards; capillaries one-cell exchange. Beat starts at SA node → AV delay → His → branches → Purkinje. Sympathetic speeds, vagus slows. CO = SV × HR ≈ 5 L/min. Cycle 0.8 s: squeeze, eject, rest-refill (coronaries fill in diastole). BP: baroreceptors fast, RAAS hormonal, kidneys slow. ECG: P = atria fire, QRS = ventricles fire, T = ventricles reset.

13.15 Questions

MCQs (answer key at end):

1. The natural pacemaker of the heart is the:

a) AV node b) SA node c) Bundle of His d) Purkinje fibres

2. Which valve lies between the left atrium and left ventricle?

a) Tricuspid b) Pulmonary c) Mitral (bicuspid) d) Aortic

3. In the ECG, the QRS complex represents:

a) Atrial depolarisation b) Ventricular depolarisation c) Ventricular repolarisation d) Atrial repolarisation

4. Cardiac output is calculated as:

a) Heart rate ÷ stroke volume b) Stroke volume × heart rate c) Blood pressure × heart rate d) Stroke volume − heart rate

5. The delay in conduction at the AV node allows:

a) The atria to refill b) The ventricles to fill completely before contracting c) The SA node to rest d) Blood to enter the coronary arteries

Viva:

1. Why is the left ventricular wall thicker than the right?
2. Name the vessels carrying blood: (a) from RV to lungs, (b) from lungs to LA, (c) from LV to the body.
3. What is the function of valves in veins?
4. During which phase of the cardiac cycle do the coronary arteries fill, and why?
5. How does the vagus nerve affect the heart rate?

Application-based:

1. A patient's ECG shows a normal P wave before every QRS complex. What does this tell you about where the heartbeat is originating? *(Ans: the rhythm originates in the SA node — normal sinus rhythm; P = atrial activation preceding each ventricular beat.)*
2. A patient feels dizzy every time they stand up quickly. Which BP control mechanism is lagging, and which drug classes might worsen this symptom? *(Ans: the baroreceptor reflex (fast nervous control); drugs that lower BP or blunt sympathetic response — e.g., many antihypertensives — can worsen orthostatic dizziness; counsel to rise slowly.)*

MCQ answers: 1-b, 2-c, 3-b, 4-b, 5-b.

13.16 References

- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Cardiovascular system.)
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- Guyton AC, Hall JE. Textbook of Medical Physiology. (The heart; regulation of the heartbeat; the circulation.)

CHAPTER 14: CARDIOVASCULAR DISEASES — PATHOPHYSIOLOGY

Unit V · ~6 hours

14.1 Introduction

Chapter 13 built the normal machine. This chapter studies how it breaks — the textbook-level mechanisms behind the heart and vessel diseases a pharmacist meets every day. Hypertension, arrhythmias, heart failure, and ischemic heart disease account for a huge share of the prescriptions you will one day dispense. Understanding *what goes wrong step by step* is what turns a dispenser into a counsellor.

Learning objectives — after studying this chapter, you should be able to:

- define hypertension and explain its basic mechanisms (primary vs secondary) in simple words;
- classify cardiac arrhythmias and explain what goes wrong in each;
- describe the pathophysiology of congestive heart failure step by step;
- explain atherosclerosis progression and how it leads to angina, myocardial infarction, and coronary artery disease;
- connect each disease to its drug-therapy logic at a basic level.

14.2 Definitions

> **Scientific definition:** Pathophysiology is the study of the disordered physiological processes that cause, result from, or are associated with a disease — the "mechanism story" of how normal function becomes abnormal.

> **Simple explanation:** If physiology is "how the machine works," pathophysiology is "how the machine breaks, bolt by bolt."

Ischemia = tissue suffering from too little blood supply (and therefore too little oxygen). From the Greek *ischo* (to hold back) + *haima* (blood) — "blood held back."

14.3 Key Terminology

Term	Meaning	Simple explanation
Hypertension	Persistently high arterial blood pressure	"Pipes under too much pressure, all the time"
Arrhythmia	Abnormal heart rhythm	"The drummer loses the beat"
Heart failure	Heart cannot pump enough blood to meet body needs	"The pump is too weak for the workload"
Atherosclerosis	Thickening/hardening of arteries by plaque deposits	"Pipes furred up with fatty sludge"
Plaque	Fatty, cholesterol-rich deposit inside an artery wall	The "sludge" narrowing the pipe
Angina	Chest pain from temporary heart-muscle ischemia	"The heart muscle crying for oxygen"
Myocardial infarction (MI)	Death of heart-muscle tissue from prolonged ischemia	"Heart attack" — muscle dies
Coronary artery disease (CAD)	Narrowing/blockage of the coronary arteries, usually by atherosclerosis	"The heart's own supply pipes are choked"

14.4 Detailed Explanation

14.4.1 Hypertension — "the silent killer"

What it is: Blood pressure that stays abnormally high over time. It is called "silent" because there are usually **no symptoms** until damage is done — the patient feels fine while the high pressure quietly injures arteries, heart, brain, and kidneys.

Basic mechanism: BP = cardiac output × peripheral resistance. Hypertension results from an increase in either (or both):

- **More volume/output** — kidneys retain too much salt and water (RAAS overactivity), heart pumps more; or
- **Tighter pipes** — vessels constrict too much (excess sympathetic activity, excess angiotensin II) or stiffen with age/plaque.

Types:

- **Primary (essential) hypertension** — the common form; no single identifiable cause. It emerges from a mix of genetic tendency, high salt intake, obesity, inactivity, chronic stress, smoking, and ageing vessels. Think of it as many small pushes adding up.
- **Secondary hypertension** — caused by an identifiable disease: kidney disease, hormonal disorders (e.g., excess aldosterone), or certain medicines. Treating the underlying cause can cure it.

Why it matters (complications): years of high pressure thicken the heart muscle (it pumps against resistance), damage artery walls (accelerating atherosclerosis), and can rupture small brain vessels (stroke) or destroy kidney filters (kidney failure).

Drug-therapy logic: each drug class targets one mechanism — diuretics (reduce salt/water volume), ACE inhibitors/ARBs (block RAAS vessel-tightening), calcium-channel blockers (relax vessel muscle), beta-blockers (calm sympathetic drive on the heart).

14.4.2 Cardiac arrhythmias — "the drummer loses the beat"

What it is: any abnormality of heart rhythm — too fast, too slow, or irregular — caused by a fault in impulse **generation** (SA node misfires) or **conduction** (signal blocked or taking a wrong shortcut).

Main patterns (textbook level):

- **Tachycardia** — resting rate abnormally fast (>100/min); the heart has less filling time, so output can fall despite speed. (Sinus tachycardia from fever/exercise is normal; the abnormal kind starts from faulty circuits.)
- **Bradycardia** — resting rate abnormally slow (<60/min); the brain may get too little blood → dizziness, fainting. Often from excessive vagal tone or conduction block.
- **Atrial fibrillation (AF)** — the atria quiver chaotically instead of contracting; blood stagnates in the atria and can form **clots**, which may travel to the brain (stroke risk). The ventricles beat irregularly.
- **Ventricular fibrillation (VF)** — the ventricles quiver uselessly; **no blood is pumped** — this is cardiac arrest and fatal within minutes without defibrillation (electric shock to reset the rhythm).
- **Heart block** — the AV node or bundle fails to pass the signal; atria and ventricles beat independently. Severe block needs an artificial pacemaker.

Drug-therapy logic: antiarrhythmics work by calming abnormal electrical activity (e.g., beta-blockers slow AV conduction; others stabilise the heart's electrical membranes); blood thinners are given in AF to prevent clot-related stroke; a defibrillator (not a drug) treats VF.

14.4.3 Congestive heart failure (CHF) — "the pump can't keep up"

What it is: the heart muscle is too weak (or too stiff) to pump enough blood to meet the body's needs. "Congestive" refers to the **congestion** — blood backing up and fluid leaking into tissues.

Step-by-step mechanism:

1. **Trigger:** the heart muscle is damaged (often after a heart attack) or chronically overworked (years of hypertension) → its pumping force falls.
2. **Backup:** blood the heart cannot push forward **backs up** behind it. Left-side failure → blood backs into the **lungs** (breathlessness, fluid in lungs — pulmonary edema). Right-side failure → blood backs into the **body veins** (swollen ankles, enlarged liver, neck veins bulge).
3. **Body's compensation (which worsens things):** sensing low output, the body activates the **sympathetic system** (heart beats faster) and the **RAAS** (retains salt and water to boost volume). Short-term this props up pressure; long-term the extra fluid overloads the weak pump further — a vicious cycle.
4. **Result:** fatigue (muscles get less blood), breathlessness, edema — the classic triad.

Drug-therapy logic: break the vicious cycle — diuretics (drain the excess fluid), ACE inhibitors/ARBs (block the harmful RAAS overdrive), beta-blockers (shield the heart from sympathetic overdrive), digoxin (strengthens each squeeze, used selectively).

14.4.4 Ischemic heart disease — angina, MI, atherosclerosis, CAD

These four terms describe one story at different stages: **atherosclerosis → coronary artery disease → angina → myocardial infarction.**

Atherosclerosis — "the pipes fur up" (the root process):

1. **Injury:** the inner lining (endothelium) of an artery is damaged — by high BP, smoking chemicals, or high LDL ("bad") cholesterol.
2. **Fatty streak:** LDL cholesterol and white blood cells collect under the lining; the cells gorge on fat and become **foam cells** — the earliest visible streak.
3. **Plaque formation:** smooth-muscle cells and fibrous tissue cover the fatty core, forming a raised **plaque** that bulges into the vessel and narrows it. Calcium hardens it over years ("hardening of the arteries").
4. **Rupture and clot:** a plaque's cap can crack; blood treats the crack like a wound and forms a **clot (thrombus)** on it — which can suddenly block the artery completely.

Coronary artery disease (CAD): atherosclerosis specifically in the **coronary arteries** — the heart's own supply lines narrow. This is the umbrella term.

Angina — "the heart cries for oxygen": when narrowed coronaries cannot deliver enough blood during exertion or stress, the heart muscle becomes **temporarily ischemic** → heavy, squeezing chest pain (often radiating to the left arm, neck, or jaw), relieved by rest. Types at textbook level: **stable angina** (predictable, with effort, relieved by rest/nitroglycerin), **unstable angina** (new, worsening, or at rest — warning of an impending heart attack), **variant (Prinzmetal) angina** (coronary spasm rather than plaque).

- **Drug logic:** nitroglycerin dilates vessels (including coronaries) and reduces the heart's workload → pain eases within minutes.

Myocardial infarction (MI) — "heart attack": when a coronary artery is **completely blocked** (usually plaque rupture + clot) and the ischemia lasts too long, heart-muscle cells **die** (necrosis) — dead muscle becomes scar and never contracts again. Symptoms: severe crushing chest pain not relieved by rest, sweating, breathlessness, nausea. The ECG shows characteristic **ST elevation**; blood tests show leaked heart-muscle proteins (troponins).

- **Drug logic:** "clot-busters" (thrombolytics) dissolve the clot if given early; antiplatelet drugs (e.g., aspirin) prevent further clotting; long-term medicines prevent recurrence.

14.5 Easy Analogy

> Think of the coronary arteries as **water pipes feeding a pumping station** (the heart muscle). **Atherosclerosis** is limescale and sludge building up inside the pipes over years. **CAD** is the diagnosis: "your station's supply pipes are scaled." **Angina** is the station sputtering whenever demand rises — it recovers when demand drops. **MI** is a pipe bursting shut completely: the station section it fed dies and never restarts. **Hypertension** is running the whole city's water at dangerously high pressure — silent until a pipe bursts. **Heart failure** is the main pump wearing out: water backs up and floods the streets (edema) while houses run dry (fatigue).

14.6 Diagrams (describe in PDF)

1. **Atherosclerosis progression strip** — four panels: healthy artery → fatty streak (foam cells) → fibrous plaque narrowing the lumen → ruptured plaque with clot blocking flow; each labelled with the stage name.
2. **Angina vs MI comparison illustration** — partially narrowed coronary (angina: pain with effort, relieved by rest) vs fully blocked coronary with dead muscle zone (MI: prolonged pain, necrosis, scar).
3. **Heart failure backup diagram** — weak left ventricle with arrows showing blood backing into lungs (breathlessness) and weak right ventricle with arrows to body veins (ankle edema, liver enlargement); RAAS/sympathetic vicious-cycle loop inset.
4. **Arrhythmia ECG strips** — normal sinus rhythm vs tachycardia (fast), bradycardia (slow), atrial fibrillation (irregular, no clear P waves), ventricular fibrillation (chaotic) — simplified textbook-style traces.
5. **Hypertension damage map** — body silhouette marking target organs: heart (thickening), brain (stroke), kidneys (failure), eyes (retinal damage), arteries (accelerated atherosclerosis).

14.7 Pharmacy Relevance

- **Adherence is the therapy:** hypertension and post-MI medicines work only if taken daily for life — the pharmacist's counselling ("no symptoms ≠ no disease") directly prevents strokes and second heart attacks.
- **Know the red flags:** crushing chest pain >15 minutes not relieved by rest = possible MI → emergency, not the pharmacy counter. Pharmacists must recognise and refer, never dispense painkillers and wait.
- **Nitroglycerin counselling:** keep tablets in the original dark bottle, sit down before taking (BP dip → fainting), seek emergency care if pain persists after the advised doses.
- **Drug interactions that matter:** NSAID painkillers can raise BP and blunt antihypertensives; potassium supplements + ACE inhibitors can dangerously raise potassium — classic pharmacist checkpoints.
- **Lifestyle advice is dispensing-adjacent:** less salt, no tobacco, daily walking, and weight control are as evidence-based as the tablets for hypertension and CAD.

14.8 India Context

- Cardiovascular disease is the leading cause of adult deaths in India, and it strikes Indians at younger ages than in Western populations — so screening and adherence in the 30s–50s age group is a national priority (stated at general textbook level, without invented figures).
- The high prevalence of diabetes, tobacco use (including smokeless forms), and high-salt diets are recognised Indian risk amplifiers for CAD and hypertension.
- Government programmes (NPCDCS — National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases and Stroke) run NCD clinics at district level offering free BP/diabetes screening and medicines — pharmacists support follow-up and adherence there.

14.9 High-Yield Points

- Hypertension = silent, sustained high BP; primary (no single cause — lifestyle + genes + age) vs secondary (kidney/hormonal/drug cause). BP = cardiac output × peripheral resistance.
- Arrhythmias: tachycardia (fast), bradycardia (slow), AF (chaotic atria → clot → stroke risk), VF (chaotic ventricles → no output → fatal without defibrillation), heart block (signal blocked → pacemaker).
- Heart failure: weak pump → blood backs up (lungs = breathlessness; body veins = edema) → sympathetic + RAAS compensation worsens overload → fatigue, breathlessness, edema triad.
- Atherosclerosis stages: endothelial injury → fatty streak (foam cells) → fibrous plaque → rupture + clot.
- CAD = coronary atherosclerosis; angina = temporary ischemia (stable/unstable/variant), relieved by rest/nitroglycerin; MI = prolonged blockage → muscle death (ST elevation, troponins).
- Drug logic: diuretics/ACE-I/ARB/CCB/beta-blockers for hypertension; break the RAAS–sympathetic vicious cycle in failure; clot-busters + antiplatelets for MI.

14.10 Common Confusions

- **Angina vs MI:** angina = TEMPORARY ischemia, pain relieved by rest, no muscle death; MI = PROLONGED blockage, muscle DIES, pain not relieved by rest. An MI is not "a bad angina."
- **Heart failure vs heart attack:** failure = pump too weak over time (chronic plumbing problem); attack (MI) = sudden blockage killing muscle (acute event). An MI can *cause* heart failure later.
- **Hypertension vs "tension":** hypertension means high BLOOD pressure, not mental stress — though stress can nudge it up.
- **Atherosclerosis vs arteriosclerosis:** atherosclerosis = plaque-specific (fatty deposits); arteriosclerosis = general stiffening/hardening of arteries with age. Atherosclerosis is one type of arteriosclerosis.
- **AF vs VF:** atrial fibrillation = serious but survivable (stroke risk from clots); ventricular fibrillation = immediately fatal without defibrillation. Never equate them.

14.11 Common Mistakes

- Saying hypertension always has symptoms like headache (it is usually symptomless — that is why it is "silent").
- Writing that angina pain is relieved by exercise (it is relieved by REST).
- Claiming heart failure means the heart has stopped (it means it pumps inadequately, not zero).
- Mixing up stable and unstable angina (stable = predictable with effort; unstable = new/worsening/at rest = emergency warning).
- Forgetting that MI diagnosis rests on the triad: typical pain + ECG changes (ST elevation) + blood markers (troponins).

14.12 Mnemonic

Atherosclerosis stages: *"**I**njured **F**oam **F**orms **R**upture"* — **I**njury → **F**atty streak (foam cells) → **F**ibrous plaque → **R**upture + clot.

Heart failure triad: *"**F**atigue, **B**reathlessness, **E**dema"* — *"**F**ailing **B**eats **E**verywhere"* (blood fails to go forward, backs into lungs, backs into veins).

Angina types: *"**S**table = **S**ame effort; **U**nstable = **U**nexpected; **V**ariant = **V**essel spasm."*

14.13 Chapter Summary

- **Hypertension** is persistently high BP, usually symptomless ("silent killer"); primary (multifactorial) vs secondary (identifiable cause); it damages heart, brain, kidneys, and arteries over years. Treated by reducing volume (diuretics), blocking RAAS (ACE-I/ARBs), relaxing vessels (CCBs), or calming sympathetic drive (beta-blockers).
- **Arrhythmias** are rhythm faults from abnormal impulse generation or conduction: tachycardia, bradycardia, atrial fibrillation (clot → stroke risk), ventricular fibrillation (fatal without defibrillation), heart block (may need pacemaker).
- **Congestive heart failure**: a weakened pump cannot meet demand → blood backs into lungs (breathlessness) and veins (edema) → sympathetic and RAAS compensation overloads the heart further → fatigue, breathlessness, edema. Therapy breaks this cycle.
- **Ischemic heart disease** is one story in stages: atherosclerosis (injury → fatty streak → plaque → rupture + clot) narrows coronaries (CAD) → temporary ischemia causes **angina** (stable/unstable/variant; relieved by rest/nitroglycerin) → complete prolonged blockage causes **myocardial infarction** (muscle death; ST elevation, troponins; clot-busters and antiplatelets).

14.14 One-Minute Revision

Hypertension = silent high BP (primary: lifestyle+genes; secondary: kidney/hormonal). Arrhythmias: fast (tachy), slow (brady), AF (clots→stroke), VF (fatal, shock it), block (pacemaker). Heart failure = weak pump → backup into lungs (breathless) and veins (edema) → RAAS/sympathetic vicious cycle → fatigue-breathlessness-edema. Atherosclerosis: injury → fatty streak → plaque → rupture+clot. CAD = coronary plaques. Angina = temporary ischemia, rest relieves. MI = blocked too long, muscle dies (ST up, troponins). Mnemonic: "Injured Foam Forms Rupture."

14.15 Questions

MCQs (answer key at end):

1. "Silent killer" refers to:

a) Ventricular fibrillation b) Hypertension c) Stable angina d) Bradycardia

2. The earliest visible lesion of atherosclerosis is the:

a) Fibrous plaque b) Fatty streak c) Thrombus d) Calcium deposit

3. Which arrhythmia carries a major risk of clot-related stroke?

a) Sinus bradycardia b) Atrial fibrillation c) First-degree heart block d) Sinus tachycardia

4. In heart failure, ankle edema results from:

a) Left-sided backup into the lungs b) Right-sided backup into the systemic veins c) Coronary spasm d) Valve infection

5. Myocardial infarction is distinguished from angina by:

a) Pain relieved by rest b) Death of heart-muscle tissue from prolonged ischemia c) Normal ECG d) Absence of chest pain

Viva:

1. Why is hypertension called "the silent killer"?
2. What is the difference between stable and unstable angina, and why does it matter?
3. Explain the vicious cycle in congestive heart failure involving RAAS and the sympathetic system.
4. What does ST elevation on an ECG suggest, and which blood test confirms MI?
5. Why are blood thinners given in atrial fibrillation?

Application-based:

1. A 55-year-old man has crushing chest pain for 40 minutes, not relieved by rest, with sweating. His ECG shows ST elevation. Is this angina or MI, and what is the immediate treatment logic? *(Ans: MI — prolonged unrelieved pain + ST elevation; emergency: restore blood flow — clot-busters if early/eligible, antiplatelets, hospital care; time is muscle.)*
2. A hypertensive patient on treatment says, "My BP was normal last month, so I stopped the tablets." Using the pathophysiology you learned, explain why this is dangerous. *(Ans: hypertension is symptomless and vessel damage continues silently; stopping treatment lets pressure rise again, raising stroke/MI/kidney risk — medicines control, not cure, primary hypertension.)*

MCQ answers: 1-b, 2-b, 3-b, 4-b, 5-b.

14.16 References

- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Cardiovascular diseases.)
- Tortora GJ, Derrickson BH. Principles of Anatomy and Physiology. (Disorders of the cardiovascular system.)
- Guyton AC, Hall JE. Textbook of Medical Physiology. (Cardiac failure; coronary circulation.)
- Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology. (Atherosclerosis; ischemic heart disease; heart failure; hypertension.)

Unit Infographic Concept Boxes (for the PDF designer)

UNIT V — "The Heart in One Page"

- Top: heart cross-section with 4 chambers, 4 valves in flow order ("Toilet Paper My Ass"), and the 5-step conduction path (SA → AV → His → branches → Purkinje).
- Middle: cardiac cycle bar (0.8 s: squeeze–eject–rest), CO = SV × HR ≈ 5 L/min, ECG strip labelled P/QRS/T.
- Bottom: disease chain — Atherosclerosis ("Injured Foam Forms Rupture") → CAD → Angina (rest relieves) → MI (muscle dies); plus Hypertension (silent), Arrhythmia types, Heart failure triad (fatigue–breathlessness–edema).

Combined References

- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Standard Indian physiology textbook.)
- Tortora GJ, Derrickson BH. Principles of Anatomy and Physiology. (Standard anatomy & physiology reference.)
- Guyton AC, Hall JE. Textbook of Medical Physiology. (Standard physiology reference.)
- Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology. (Standard pathology reference for disease mechanisms.)