FIRST MBBS · INTEGRATED TEACHING · CARDIOVASCULAR SYSTEM

A E T C M

The Heart

An Integrated Story

Anatomy · Physiology · Biochemistry — told the simple way

Follow Anu, a first-year MBBS student, as her grandfather's chest pain takes her on a journey through the structure, the working and the chemistry of the heart — with tables, flowcharts, simple ECGs, online animations and the drugs doctors use, explained by how they work.

The story begins › | Where the heart lives › | Four rooms, four doors › | Coronary circulation ›
Embryology & fetal circulation › | Histology › | Electricity & action potentials › | Cardiac cycle › | ECG made simple ›
Cardiac output & Starling › | Blood pressure & capillaries › | Fuel of the heart › | Molecules of contraction ›
NO, endothelin, angiotensin › | Lipids & atherosclerosis › | Cardiac biomarkers › | Putting it together ›
Drugs for the heart › | Viva questions ›

TAP A TOPIC TO JUMP TO IT

ANATOMY — structure · vessels · development

PHYSIOLOGY — electricity · cycle · ECG · BP

BIOCHEMISTRY — fuel · proteins · lipids · markers

DRUGS — how they work

SEE IT ONLINE — TAP TO OPEN ANIMATION / AUDIO / IMAGE

Cardiac cycle (animation) ↗ | Wiggers diagram ↗ | Heart sounds (YouTube) ↗ | ECG forming (animation) ↗
Normal 12-lead ECG ↗ | Coronary arteries ↗ | Fetal circulation ↗ | Action potential ↗ | P–V loop ↗
All links — visual atlas ›

AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

For first-year MBBS students and teachers of integrated cardiovascular physiology

AETCM · ACADEMY FOR EMERGENCY, TRAUMA & CRITICAL CARE MEDICINE

The Heart — An Integrated Story

Anatomy · Physiology · Biochemistry for First MBBS

Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM) · First edition · October 2026

How to use this book

In the first year of MBBS you meet the heart three times — in the dissection hall (Anatomy), in the physiology lecture and laboratory (Physiology), and in the biochemistry classroom (Biochemistry). Most students learn these as three separate subjects. The examiner, and later the patient, expects you to see them as **one heart**. This little book tells the heart as a single story.

Each chapter opens with a short **story box** about Anu and her grandfather. Then comes the science in plain words, with tables to revise and flowcharts to draw. Look for the **Integration corner** — three columns that show how Anatomy, Physiology and Biochemistry explain the same thing. **Green boxes** are "remember" summaries; **blue boxes** are exam tips.

Tap to see it move

Wherever a moving or sounding thing is described — the cardiac cycle, heart sounds, an ECG — a small button opens a free online animation, image or audio: ▶ Animation ▶ Listen (YouTube) ▶ ECG. A one-page visual atlas with QR codes is given at the end for the printed copy.

The drug chapter explains **how drugs work** so that you can connect them to the physiology you are learning. Doses are deliberately not given — you will learn them in Pharmacology and Medicine. Tags used: A Anatomy P Physiology B Biochemistry C Clinical bridge.

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FOREWORD

Foreword

From Dr. Gireesh Kumar K.P.



Dear first-year students,

Every year, in the first weeks of MBBS, I see the same scene. A student stands in the dissection hall holding a heart, goes to the physiology lecture to hear about the cardiac cycle, and then sits in biochemistry learning about troponin and β-oxidation — and never realises that all three teachers were talking about the **same organ**. The heart gets learnt in three pieces, and the pieces rarely meet again until a patient with chest pain arrives in the emergency department years later.

I have spent most of my working life in emergency medicine. At three o'clock in the morning, when a man walks in clutching his chest, the doctor who saves him does not think in subjects. In one glance at the ECG she sees an artery (anatomy), a wall that has stopped moving (physiology) and cells running out of ATP (biochemistry) — and she acts. That joined-up thinking begins in the first year, or it struggles to begin at all.

This little book is my attempt to give you that joined-up picture from the very start. I have written it as a story — the story of Anu and her grandfather — because we remember stories far longer than we remember lists. Each chapter places Anatomy, Physiology and Biochemistry side by side, with simple tables, flowcharts you can redraw in the examination, hand-drawn ECGs, and links that let you watch the heart move and hear it beat. I have also included the common cardiac drugs, not to burden you with pharmacology, but to show you that every tablet is simply physiology and biochemistry put to work.

Read it slowly, draw the diagrams yourself, listen to the heart sounds, and keep asking the three questions on every page: *Where is it? How does it work? What molecule makes it happen?* If you do, the heart will never again be three separate subjects for you — it will be one living, beating organ that you understand.

I thank my colleagues in the departments of Anatomy, Physiology and Biochemistry, whose integrated teaching inspired this book, and the AETCM team for bringing it to you. Suggestions from students and teachers are always welcome.

With best wishes for a wonderful journey into medicine,

Dr. Gireesh Kumar K.P.
Academy for Emergency, Trauma & Critical care Medicine (AETCM)
October 2026

CHAPTER 1 · THE STORY BEGINS

One Heart, Three Lenses

Why the heart must be learnt as Anatomy + Physiology + Biochemistry together

📖 Sunday morning, Thrissur

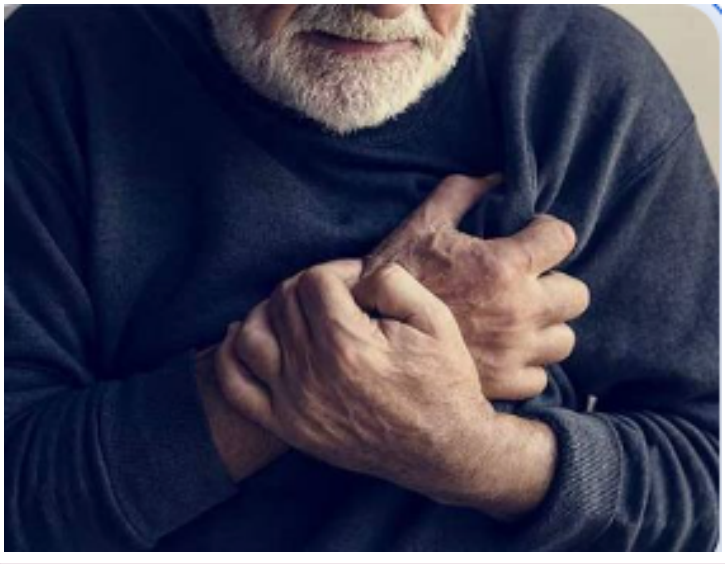
Anu had just finished her first month of MBBS. On Sunday morning her grandfather, Raghavan (62 years, a retired bank clerk, a smoker for 30 years), sat down heavily on the veranda, holding the centre of his chest. "It feels like an elephant is sitting on me," he whispered. He was sweating. His left arm ached. Anu's father rushed him to the hospital. An emergency physician stuck stickers on his chest — an ECG. Another drew blood — "send troponin." Within an hour he was in the cath lab. That night Anu opened her three textbooks and asked one question: ***what exactly happened inside Appooppan's heart?***

To answer Anu's question you need all three subjects. Anatomy tells you **which pipe** was blocked and **which part** of the heart it feeds. Physiology tells you **what stopped working** — the pumping and the electrical signals that the ECG picks up. Biochemistry tells you **why the cells died** and **what leaked out** into the blood for the laboratory to measure.

Lens	Question it answers	In Appooppan's heart attack
A **Anatomy**	Where? Which structure?	A clot blocked the left anterior descending (LAD) coronary artery, which supplies the front wall of the left ventricle
P **Physiology**	How does it work? What changed?	The front wall stopped contracting; the injured muscle changed the electrical current — ST elevation on the ECG
B **Biochemistry**	What happens in the molecules?	No oxygen → no ATP from mitochondria → lactic acid, calcium overload, cell membrane breaks → troponin leaks into blood
C **Clinical bridge**	So what does the doctor do?	Open the artery (angioplasty), prevent new clots (aspirin, clopidogrel), lower cholesterol (statin), rest the heart (beta blocker)

The heart in one paragraph

The heart is a **muscular pump about the size of your fist** (about 250–300 g). It has two sides working side by side. The **right side** collects used, dark blood from the body and pushes it to the lungs. The **left side** collects fresh, oxygen-rich blood from the lungs and pushes it to the whole body. It beats about **72 times a minute, 1 lakh times a day, 2.5 billion times in a life**, without ever resting — because it has its own electrical starter, its own blood supply and a fuel system that almost never runs out.



Appooppan holding the centre of his chest — the Sunday-morning chest pain where our story begins

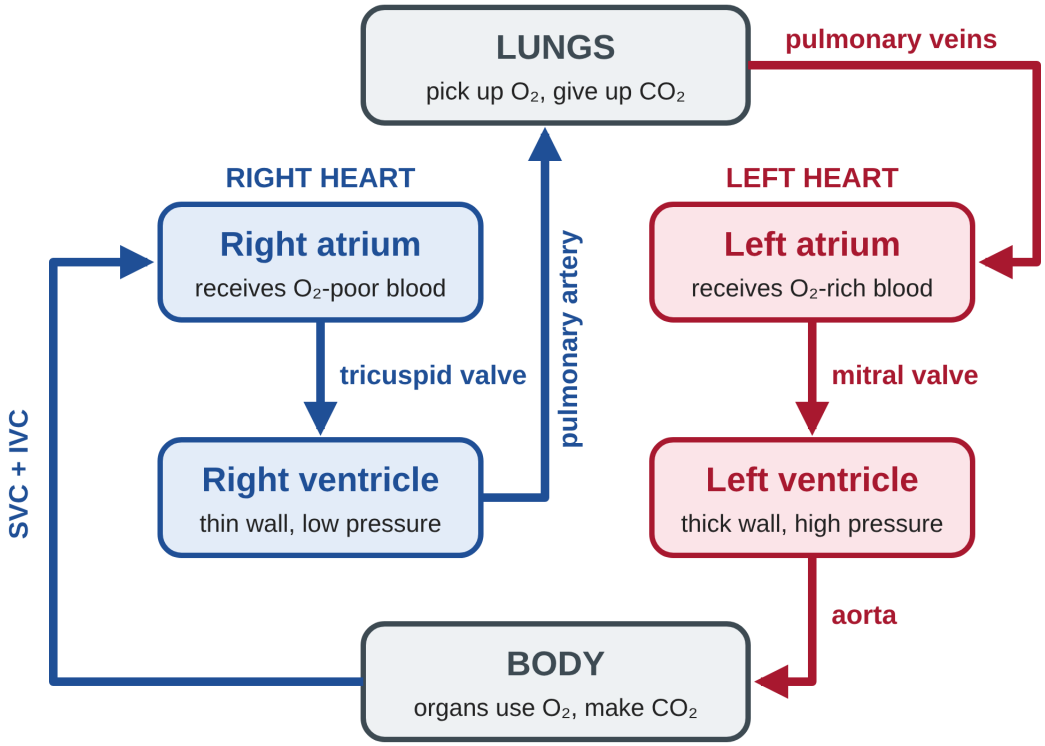


Figure 1.1 — Two pumps in series: body → right heart → lungs → left heart → body (red = O_2-rich, blue = O_2-poor)

Remember the four big ideas

- **Structure decides function** — the thick left ventricle makes high pressure; the thin right ventricle makes low pressure.
- **Electricity comes before contraction** — every beat starts as an electrical spark from the SA node.
- **The heart is an aerobic organ** — it burns mostly fat with oxygen; without oxygen it fails within minutes.
- **Injury leaves fingerprints** — ECG changes and enzymes in the blood tell us where and when damage occurred.

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CHAPTER 2 · ANATOMY

Where the Heart Lives

Position, surfaces and borders, mediastinum and pericardium

📖 The doctor's finger on the chest

Before the ECG, the emergency physician placed two fingers below Appooppan's left nipple. "I am feeling the apex beat," she said. Anu wondered — how does she know where the heart is without seeing it?

The heart lies in the **middle mediastinum** — the central compartment of the chest between the two lungs — wrapped in a bag called the **pericardium**. It sits obliquely: the broad **base** faces backwards and to the right, and the pointed **apex** faces forwards, downwards and to the left. The apex lies in the **left 5th intercostal space, just inside the midclavicular line** (about 9 cm from the midline) — this is where you feel the **apex beat**. ▶ Heart anatomy images

Feature	Formed mainly by	Why it matters
Apex	Left ventricle	Apex beat — shifted outward when the LV enlarges
Base (posterior surface)	Left atrium (mostly)	An enlarged LA presses on the oesophagus behind it
Anterior (sternocostal) surface	Right ventricle (mostly)	RV lies just behind the sternum — injured first in stab wounds
Inferior (diaphragmatic) surface	Left ventricle (2/3) and right ventricle	Supplied mainly by the RCA — inferior MI
Right border	Right atrium	Right heart shadow on chest X-ray
Left border	Left ventricle (and left auricle)	Left heart shadow on X-ray
Inferior border	Right ventricle (mainly)	Lies on the diaphragm

The mediastinum — the heart's neighbourhood

Part	Main contents
Superior mediastinum	Arch of aorta and its branches, SVC, brachiocephalic veins, trachea, oesophagus, thymus, phrenic and vagus nerves
Anterior mediastinum	Thymus remnants, fat, lymph nodes
Middle mediastinum	**Heart and pericardium**, ascending aorta, pulmonary trunk, lower SVC, phrenic nerves, main bronchi
Posterior mediastinum	Descending aorta, oesophagus, thoracic duct, azygos veins, sympathetic chain

The pericardium — a double bag

Think of pushing your fist into a soft balloon: the fist is the heart, the layer of balloon touching the fist is the **visceral layer**, the outer layer is the **parietal layer**, and the space between them holds a few millilitres of lubricating fluid so the heart can beat without friction. Outside all this is a tough **fibrous pericardium** fused to the diaphragm, which stops the heart from over-stretching. ▶ Pericardium images

Layer / space	What it is	Clinical bridge
Fibrous pericardium	Tough outer sac, fused to central tendon of diaphragm	Cannot stretch quickly — rapid fluid collection squeezes the heart (cardiac tamponade)
Serous — parietal layer	Lines the inside of fibrous sac	Supplied by the phrenic nerve — pericarditis pain felt in the shoulder

Serous — visceral layer (epicardium)	Covers heart surface; coronary vessels run in it	—
Pericardial cavity	Potential space with 15–50 mL fluid	Pericardial effusion
Transverse sinus	Passage behind aorta and pulmonary trunk, in front of SVC	Surgeons pass a finger/clamp here in cardiac surgery
Oblique sinus	Cul-de-sac behind the left atrium	—

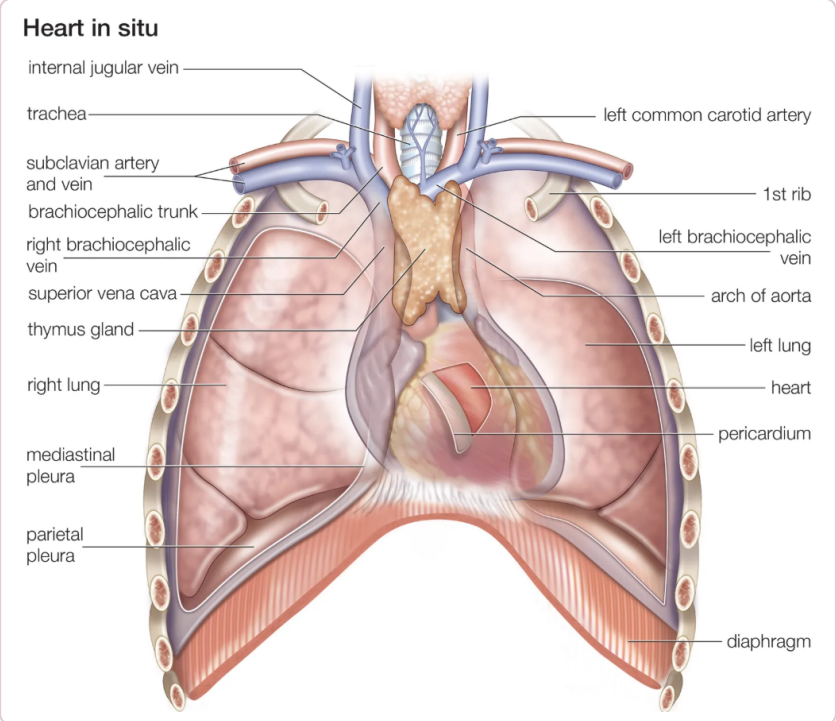


Figure 2.1 — The heart in situ — in the middle mediastinum between the two lungs, wrapped in pericardium, resting on the diaphragm

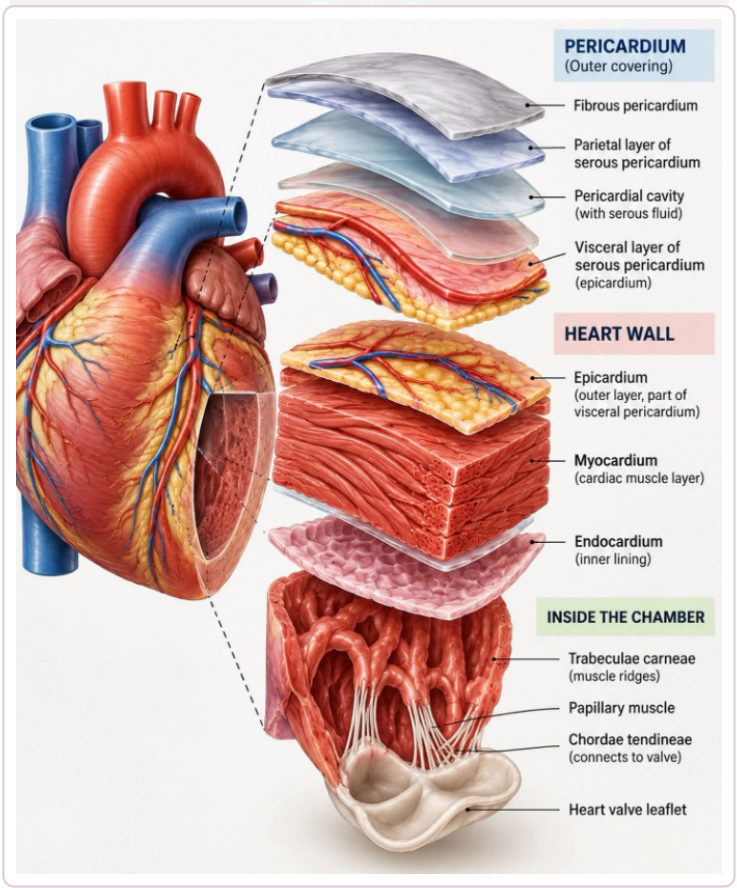


Figure 2.2 — Coverings and layers of the heart — fibrous and serous pericardium, epicardium, myocardium and endocardium

CHAPTER 3 · ANATOMY → PHYSIOLOGY

Four Rooms and Four Doors

Chambers, septa, valves and the path of blood

📖 A house with four rooms

Anu's anatomy teacher drew the heart as a house: two upstairs rooms (the atria) that receive guests, two downstairs rooms (the ventricles) that throw them out, a wall down the middle (the septum), and four one-way doors (the valves). "If a door leaks or sticks," she said, "the whole house suffers."

Chamber	Receives blood from	Sends blood to	Key internal features
Right atrium	SVC, IVC, coronary sinus	Right ventricle (through tricuspid)	Smooth part (sinus venarum) and rough part with pectinate muscles separated by crista terminalis; fossa ovalis on septum; **SA node** near SVC opening
Right ventricle	Right atrium	Pulmonary trunk	Trabeculae carneae; septomarginal trabecula (moderator band) carrying right bundle branch; smooth outflow (infundibulum)
Left atrium	Four pulmonary veins	Left ventricle (through mitral)	Mostly smooth wall; left auricle — where clots form in atrial fibrillation
Left ventricle	Left atrium	Aorta	Thickest wall (3× RV); two papillary muscles; smooth outflow (aortic vestibule)

	Right ventricle	Left ventricle
Wall thickness	3–5 mm	10–15 mm
Pressure	25/5 mmHg	120/10 mmHg
Pumps into	Lungs (low resistance)	Whole body (high resistance)
Shape in section	Crescent	Circle

Why is the LV wall thicker?

Both ventricles pump the **same volume** each beat (~70 mL). But the LV must push against **5–6 times more pressure**. Muscle grows in proportion to the work it does — like the arm of a weight-lifter. That is also why long-standing high BP thickens the LV (left ventricular hypertrophy).

The four doors — the valves

Valve	Between	Cusps	Opens when	Closure makes
Tricuspid (AV)	RA → RV	3	Atrial pressure > ventricular pressure	Part of S1 ("lub")
Mitral / bicuspid (AV)	LA → LV	2 (anterior, posterior)	Atrial pressure > ventricular pressure	Main part of S1
Pulmonary (semilunar)	RV → pulmonary trunk	3	RV pressure > pulmonary artery pressure	P2 component of S2
Aortic (semilunar)	LV → aorta	3 (coronary arteries arise above two of them)	LV pressure > aortic pressure	A2 component of S2 ("dub")

How do valves stay shut? Valves have no muscle; they open and close passively when pressure on one side becomes higher. The AV valves would flip back into the atria like an umbrella in the wind — but the **chordae tendineae** (strings)

tied to the **papillary muscles** hold them. The papillary muscles contract with the ventricle and keep the strings tight. If a papillary muscle dies in a heart attack, the mitral valve leaks suddenly (acute mitral regurgitation).

The path of one red cell — draw this flowchart

SVC / IVC → Right atrium → Tricuspid → Right ventricle → Pulmonary valve → Pulmonary artery → Lungs (O_2 in)

Pulmonary veins → **Left atrium** → Mitral → **Left ventricle** → Aortic valve → **Aorta** → Body (O_2 out)

🔗 Integration corner — Valves

ANATOMY
Cusps, chordae tendineae and papillary muscles; fibrous skeleton of the heart holds the four valve rings.

PHYSIOLOGY
Valves open and close by pressure difference; their closure gives S1 and S2; a narrow valve (stenosis) or leaky valve (regurgitation) causes a murmur.

BIOCHEMISTRY
Valve tissue is collagen and elastin; in rheumatic fever, antibodies against streptococcal M-protein cross-react with valve proteins (molecular mimicry).

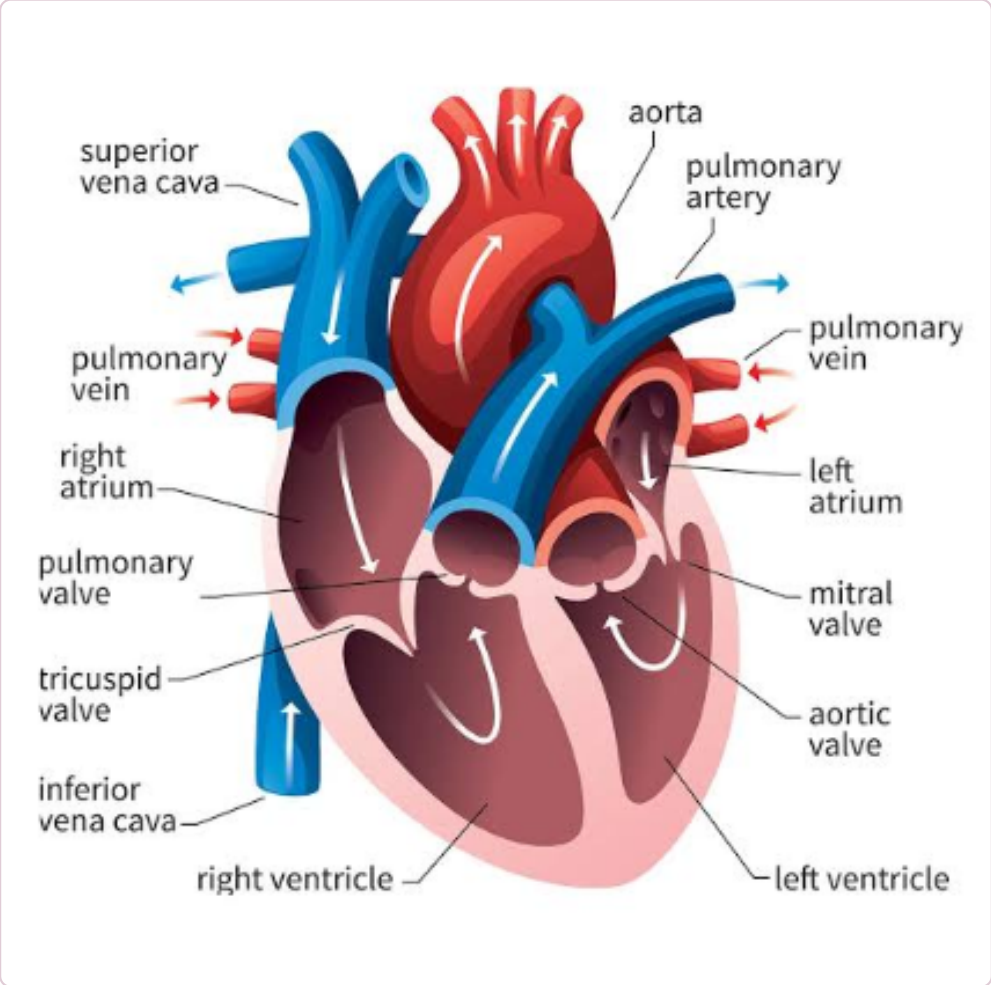


Figure 3.1 — The four chambers and four valves — arrows show the path of blood through the heart

CHAPTER 4 · ANATOMY → CLINICAL

The Heart Feeds Itself

Coronary arteries, dominance and venous drainage

📖 Which pipe was blocked?

The cardiologist showed Anu the angiogram. "See this artery running down the front groove of the heart? It is the left anterior descending — the LAD. It was totally blocked. We call it the widow-maker." Anu realised that although the heart is full of blood, its muscle cannot drink from the blood inside its chambers — it needs its own pipes.

The heart muscle gets its blood from two **coronary arteries**, the first branches of the aorta, arising from the **aortic sinuses** just above the aortic valve. They run in the grooves (sulci) on the surface of the heart under the epicardium, then send branches into the muscle. ▶ Coronary artery images

Artery	Origin and course	Main branches	Supplies
Right coronary artery (RCA)	Right (anterior) aortic sinus; runs in the right atrioventricular groove	SA nodal (≈60%), right marginal, AV nodal (≈80–90%), posterior interventricular (in right dominance)	Right atrium, right ventricle, inferior wall of LV, posterior 1/3 septum, SA and AV nodes
Left main coronary artery	Left (posterior) aortic sinus; short (1–2 cm), passes behind the pulmonary trunk	Divides into LAD and circumflex	—
Left anterior descending (LAD) = anterior interventricular	Runs down the anterior interventricular groove to the apex	Diagonal and septal branches	Anterior wall of LV, anterior 2/3 of septum, apex, bundle branches
Left circumflex (LCx)	Runs in the left AV groove round to the back	Obtuse marginal branches	Lateral and posterior wall of LV, left atrium

Dominance — who gives the posterior interventricular artery?

The artery that gives the **posterior interventricular (posterior descending) artery** is called dominant. **Right dominant: ~70–80%**, left dominant ~10%, co-dominant ~10%. That is why the RCA usually supplies the AV node — an inferior wall MI often causes **heart block**.

Why do coronaries fill in diastole?

During systole the contracting LV muscle squeezes its own vessels shut. So the LV gets most of its blood in **diastole**. A fast heart rate shortens diastole and reduces coronary flow — one reason beta blockers help angina.

Venous drainage

Vein	Runs with	Drains into
Great cardiac vein	LAD (anterior groove)	Coronary sinus
Middle cardiac vein	Posterior interventricular artery	Coronary sinus
Small cardiac vein	Right marginal artery	Coronary sinus
Coronary sinus	Posterior AV groove	Right atrium
Anterior cardiac veins	Front of right ventricle	Directly into right atrium

Venae cordis minimae (Thebesian)	Tiny, within the wall	Directly into all chambers

🔗 Integration corner — Coronary territory, ECG lead and enzyme

ANATOMY
LAD → anterior wall; RCA → inferior wall; LCx → lateral wall. Coronaries are functional end-arteries.

PHYSIOLOGY
Anterior MI → ST elevation in V1–V4; inferior → II, III, aVF; lateral → I, aVL, V5–V6. Loss of muscle → reduced stroke volume.

BIOCHEMISTRY
Dead cells release troponin and CK-MB; the larger the territory, the higher the enzyme peak.

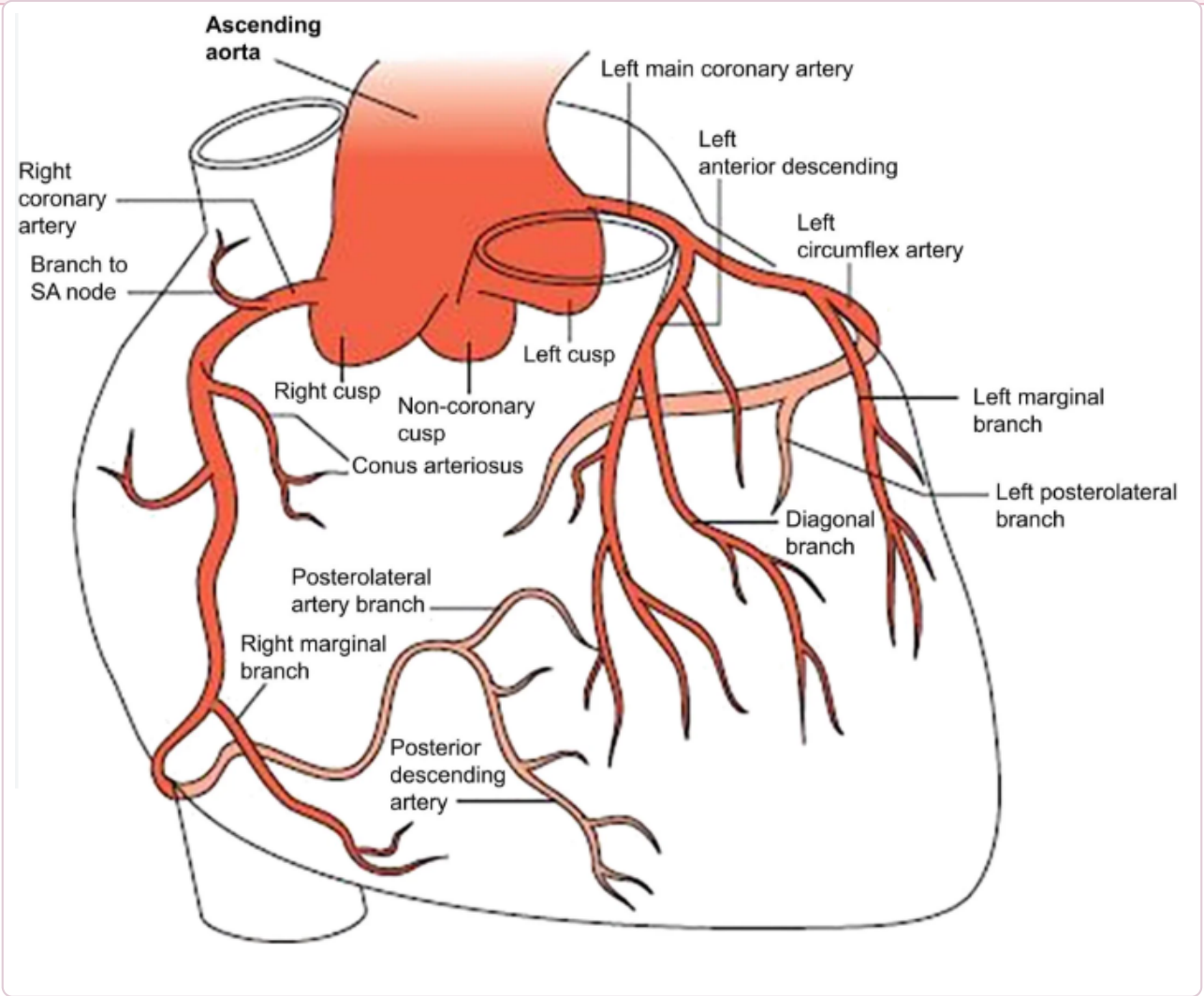


Figure 4.1 — The coronary arteries — right coronary artery and left main dividing into the LAD and circumflex, with their branches

CHAPTER 5 · EMBRYOLOGY

How the Heart Was Built

Heart tube, looping, septation, fetal circulation and the changes at birth

📖 A hole in the neighbour's baby

The baby in the next cubicle had a murmur. "A small hole between the two upper chambers — an ASD," said the paediatrician. "It should have closed after birth." Anu wondered why a hole should be there at all.

The heart is the **first organ to work** in the embryo — it begins beating at about **day 22** (end of the 3rd week), because the growing embryo is too big to get oxygen by diffusion alone. The story has four steps:

1. Heart tube — two endocardial tubes from cardiogenic mesoderm fuse into one tube (day 21–22)

↓

2. Looping — the tube bends to the right (dextro-looping) so the future ventricles come to lie in front and atria behind (week 4)

↓

3. Septation — walls grow to divide atria, ventricles and the outflow (weeks 4–7)

↓

4. Valves form from endocardial cushions; the outflow truncus splits into aorta and pulmonary trunk by a **spiral** aorticopulmonary septum (neural crest cells)

Structure	How it forms (simply)	If it goes wrong
Interatrial septum	Septum primum grows down leaving ostium primum; ostium secundum opens above; septum secundum grows on its right; the gap between them is the **foramen ovale** — a flap valve	Atrial septal defect (ASD)
Interventricular septum	Muscular part grows up from the floor; membranous part closes the top from endocardial cushions	Ventricular septal defect (VSD) — commonest congenital heart defect; usually membranous
Aorticopulmonary septum	Spiral septum divides truncus arteriosus	Transposition of great arteries; tetralogy of Fallot

Fetal circulation — the lungs are on leave

Before birth the baby does not breathe; the **placenta** is the lung. So the fetal heart uses three **shunts** to bypass the liver and lungs. ▶ Fetal circulation diagram

Placenta → **umbilical vein** (most oxygenated blood, ~80% saturation)

↓

Ductus venosus — bypasses liver → IVC → right atrium

↓

Foramen ovale — most of this blood crosses RA → LA → LV → aorta → **brain and heart get the best blood**

↓

SVC blood → RA → RV → pulmonary trunk → **ductus arteriosus** → descending aorta (bypasses lungs)

↓

Two **umbilical arteries** (from internal iliac arteries) carry blood back to the placenta

Fetal structure	What happens at birth	Adult remnant
Foramen ovale	Lungs expand → pulmonary resistance falls → LA pressure rises above RA → flap pushed shut (functional closure at birth)	Fossa ovalis
Ductus arteriosus	Higher O_2 and fall in prostaglandin E_2 make it contract (hours–days)	Ligamentum arteriosum
Ductus venosus	Umbilical flow stops	Ligamentum venosum
Umbilical vein	Flow stops	Ligamentum teres (round ligament of liver)
Umbilical arteries	Constrict	Medial umbilical ligaments

🔗 Integration corner — Congenital heart disease

ANATOMY
Failure of a septum to close (ASD, VSD) or of the ductus to close (PDA).

PHYSIOLOGY
Blood flows from high-pressure left side to low-pressure right side (left-to-right shunt) → more blood to lungs.

BIOCHEMISTRY
If the shunt reverses and blue blood reaches the body, hypoxia activates **HIF-1α** → kidney makes more **erythropoietin** → polycythaemia. Prostaglandin keeps the ductus open; NSAIDs (indomethacin) help close it.

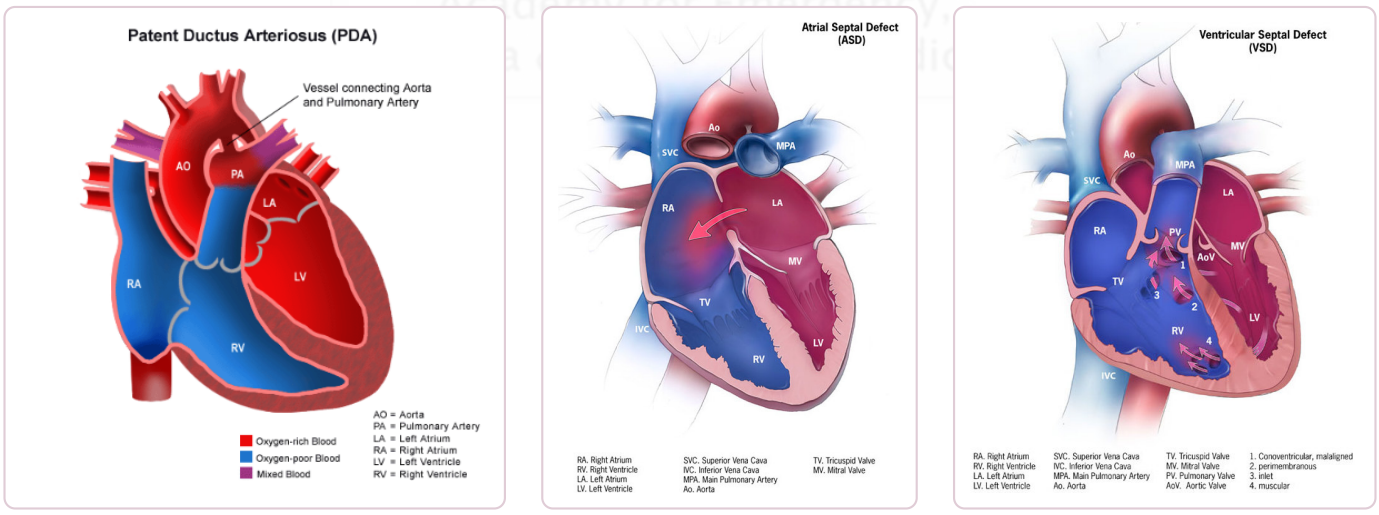


Figure 5.1 — Common congenital heart defects — patent ductus arteriosus (PDA), atrial septal defect (ASD) and ventricular septal defect (VSD)

CHAPTER 6 · HISTOLOGY

Bricks of the Wall

Cardiac muscle and the structure of blood vessels

> **📖 Under the microscope**
>
> *In the histology lab Anu saw cardiac muscle for the first time — striped like skeletal muscle, but the fibres branched and joined each other, with dark lines crossing them. "Those are intercalated discs," said the tutor. "They make the whole heart behave like one cell."*

Feature	Skeletal muscle	Cardiac muscle	Smooth muscle
Shape	Long, unbranched cylinders	**Branched** short cylinders	Spindle-shaped
Nuclei	Many, at the edge	**One or two, central**	One, central
Striations	Present	Present	Absent
Intercalated discs	Absent	**Present** — desmosomes, fascia adherens and **gap junctions**	Absent (gap junctions in some)
Control	Voluntary (somatic nerves)	Involuntary — own pacemaker, modified by autonomic nerves	Involuntary
Calcium source	Sarcoplasmic reticulum only	SR + calcium entering from outside (calcium-induced calcium release)	Mainly from outside
Regeneration	Some (satellite cells)	Practically none — dead heart muscle becomes scar	Good

Why gap junctions matter: they are tiny protein channels (connexins) that let ions flow from one cell to the next. So when one cell fires, the current spreads to its neighbours instantly, and the atria (or ventricles) contract together as a **functional syncytium**. ▶ Cardiac muscle histology

Purkinje fibres are special cardiac muscle cells under the endocardium — larger, paler (rich in glycogen, few myofibrils) and built for **fast conduction**, not for contraction.

The three coats of a blood vessel

Every vessel larger than a capillary has three layers: **tunica intima** (endothelium — the living lining that makes nitric oxide), **tunica media** (smooth muscle and elastic tissue) and **tunica adventitia** (connective tissue with tiny vessels — vasa vasorum).

Vessel	Special feature	Job	Example
Elastic (conducting) artery	Media full of elastic lamellae	Stretches in systole, recoils in diastole → keeps blood flowing continuously (Windkessel effect)	Aorta, pulmonary trunk
Muscular (distributing) artery	Thick smooth muscle; clear internal elastic lamina	Distributes blood to organs; **site of atherosclerosis**	Coronary, femoral, radial
Arteriole	1–2 layers of smooth muscle	**Resistance vessels** — decide BP and organ flow	—
Capillary	Only endothelium + basement membrane	Exchange of O_2, nutrients, waste	Continuous (muscle), fenestrated (kidney), sinusoidal (liver)
Vein	Thin media, wide lumen, **valves**	Capacitance vessels — hold ~65% of blood	Great saphenous vein

CHAPTER 7 · PHYSIOLOGY

The Spark

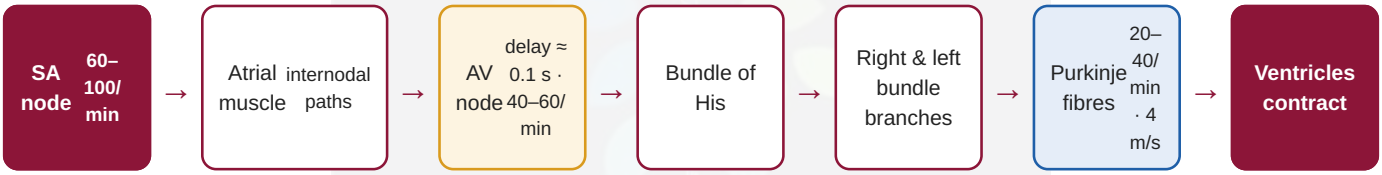
Properties of cardiac muscle, the conducting system and action potentials

📖 Why didn't his heart just stop?

In the ICU, Appooppan's monitor showed a slow rate of 38 for a few minutes. "His AV node is stunned — the lower centres have taken over," the doctor said, and kept the pacing wire ready. Anu learnt that the heart has backup generators.

Property	Meaning	Special point in heart
Excitability (bathmotropy)	Responds to a stimulus with an action potential	Long refractory period (~250 ms) — heart **cannot be tetanised**, so it always relaxes and refills
Rhythmicity / automaticity (chronotropy)	Generates its own impulses	SA node fastest → it is the **pacemaker**
Conductivity (dromotropy)	Spreads the impulse	Slowest in AV node (delay lets atria empty first), fastest in Purkinje fibres
Contractility (inotropy)	Shortens and generates force	Increased by sympathetic nerves and calcium; obeys Frank–Starling law

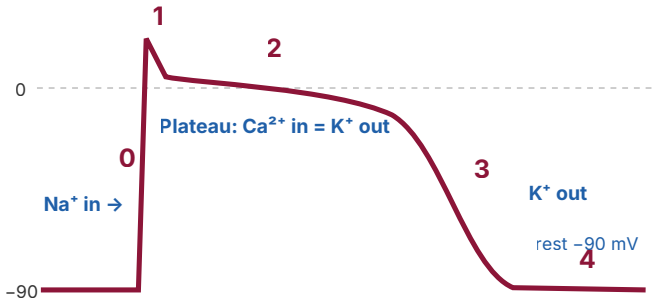
The conducting system — the heart's wiring



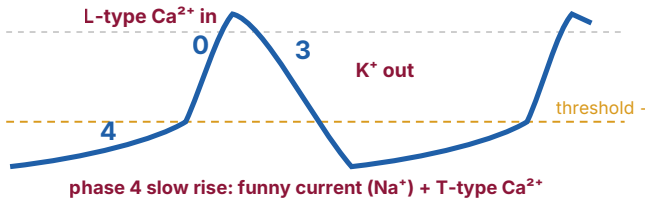
Part	Location	Intrinsic rate / speed
SA node	Wall of right atrium, near SVC opening (upper end of crista terminalis)	60–100/min — the natural pacemaker
AV node	Floor of RA, in the triangle of Koch near coronary sinus opening	40–60/min; slowest conduction (0.05 m/s) — gatekeeper
Bundle of His	Pierces fibrous skeleton, runs in membranous septum	Only electrical connection between atria and ventricles
Bundle branches	Either side of the muscular septum; right branch in moderator band	—
Purkinje fibres	Under endocardium of ventricles	20–40/min; fastest (≈ 4 m/s)

▶ Conducting system animation ▶ How each part makes the ECG (animation)

Two kinds of action potential



A. Ventricular muscle — fast response (≈ 250–300 ms)



B. SA node — slow response (no phase 1 or 2, no true rest)

Phase	Ventricular muscle (fast)	SA node (slow)
4 — resting / pacemaker	Stable at −90 mV (K^+ leak)	**Slow rise** from −60 mV: funny current (I_f, Na^+ in via HCN channels) then T-type Ca^{2+} — this is automaticity
0 — upstroke	Fast Na^+ entry — very steep	Slower Ca^{2+} entry (L-type)
1 — early repolarisation	Brief K^+ exit (notch)	Absent
2 — plateau	Ca^{2+} in balances K^+ out — long plateau triggers contraction and gives long refractory period	Absent
3 — repolarisation	K^+ exits	K^+ exits

Exam tip — never confuse the ions

Upstroke in the **SA node is Ca^{2+}-driven**; upstroke in the **ventricle is Na^+-driven**. Sympathetic stimulation (noradrenaline → β_1 → cAMP) makes phase 4 steeper → faster heart rate. Vagus (acetylcholine → M_2) opens K^+ channels and flattens phase 4 → slower rate. ▶ Action potential figures

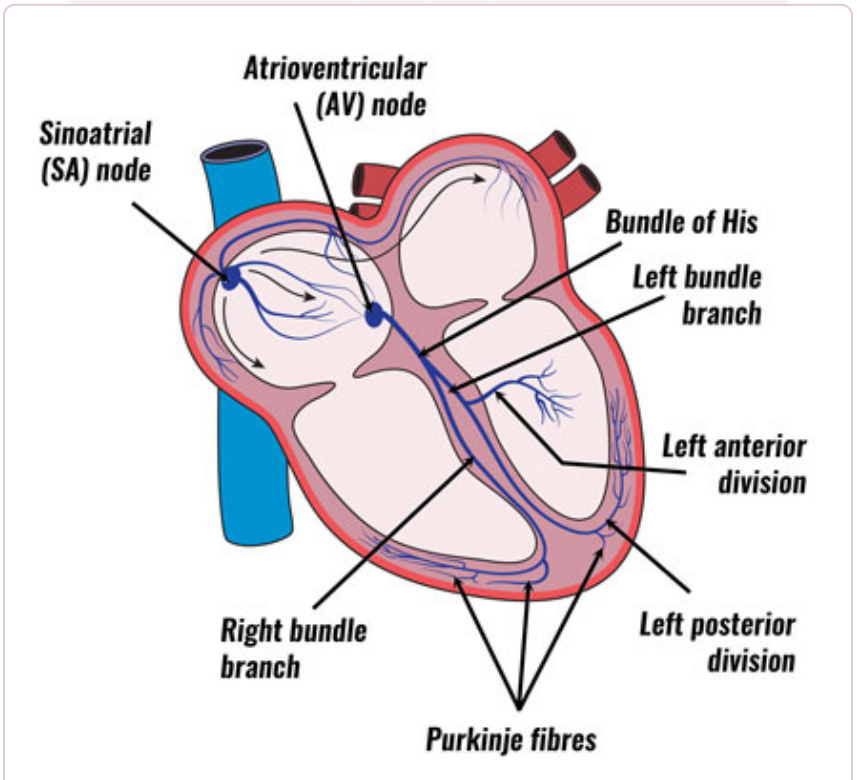


Figure 7.1 — The conducting system — SA node → AV node → bundle of His → right and left bundle branches → Purkinje fibres

CHAPTER 8 · PHYSIOLOGY

One Heartbeat

The cardiac cycle, pressure–volume changes and heart sounds

📖 Lub … dub

The resident let Anu listen with her stethoscope. "Lub-dub, lub-dub. The 'lub' is the mitral and tricuspid doors shutting; the 'dub' is the aortic and pulmonary doors shutting. Everything else you learn about the cardiac cycle happens between these two sounds."

One **cardiac cycle** is everything that happens from the start of one heartbeat to the start of the next. At a heart rate of 75/min it lasts **0.8 s**: ventricular systole ≈ 0.3 s and diastole ≈ 0.5 s. When the heart rate rises, it is mainly **diastole that shortens** — less time to fill and to feed the coronaries.

Watch it move first: ▶ Animated heart + Wiggers diagram ▶ Wiggers diagram explained ▶ Listen: S1 & S2 (YouTube) ▶ S1–S4 for beginners (YouTube)

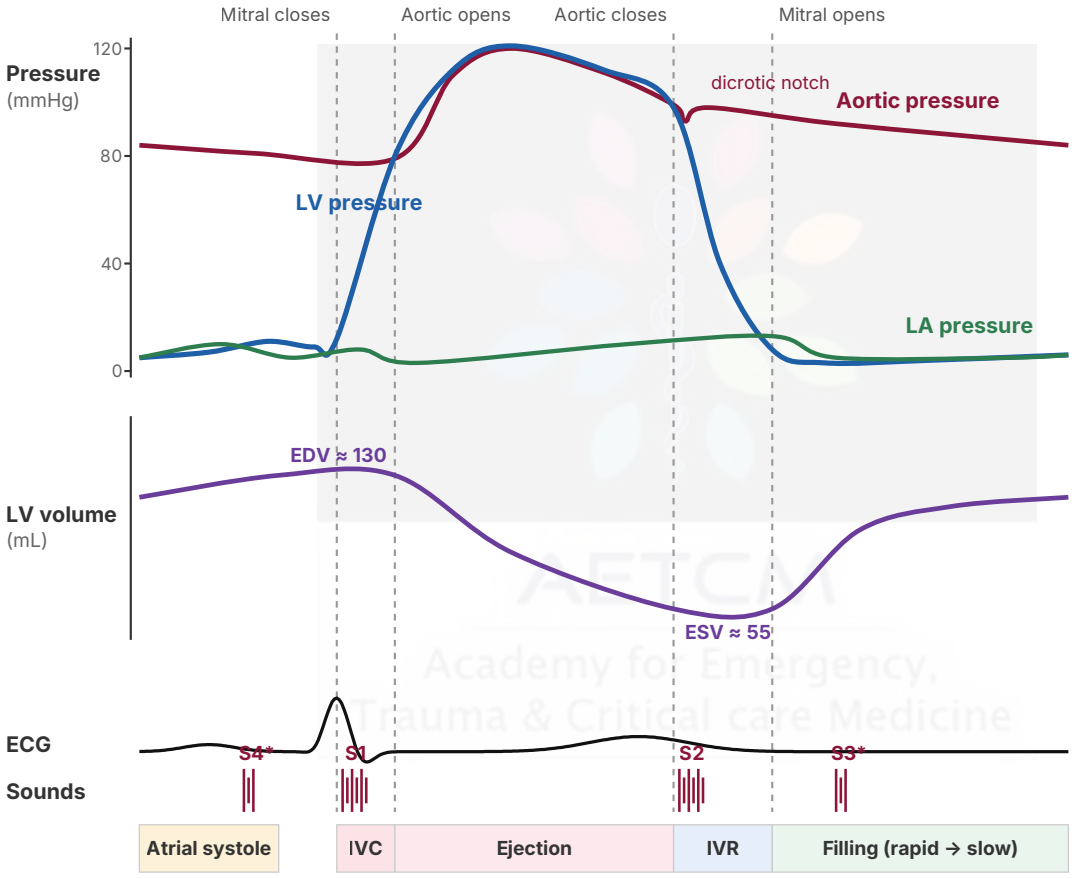


Figure 8.1 — A simplified Wiggers diagram of the left heart (IVC = isovolumetric contraction, IVR = isovolumetric relaxation; *S3 and S4 are not normally heard in adults)

Phase (in order)	Valves	What happens — in one line	Sound / ECG
1. Atrial systole	AV open, semilunar closed	Atria squeeze the last 20–30% of blood into ventricles ("atrial kick")	After P wave; S4 if ventricle stiff
2. Isovolumetric contraction	**All closed**	Ventricle contracts, pressure shoots up, volume unchanged	Just after QRS; **S1** at start
3. Ejection (rapid then slow)	Semilunar open	Blood goes into aorta; LV pressure peaks ~120 mmHg; ~70 mL ejected	ST segment and T wave

4. Isovolumetric relaxation	**All closed**	Ventricle relaxes, pressure falls, volume unchanged	**S2** at start; end of T wave
5. Rapid filling	AV open	Blood stored in atria rushes in	S3 (normal in children)
6. Slow filling (diastasis)	AV open	Slow trickle; then cycle repeats	—

Volume	Normal value
End-diastolic volume (EDV)	≈ 120–130 mL
End-systolic volume (ESV)	≈ 50–55 mL
Stroke volume = EDV − ESV	≈ 70 mL
Ejection fraction = SV/EDV	55–70%

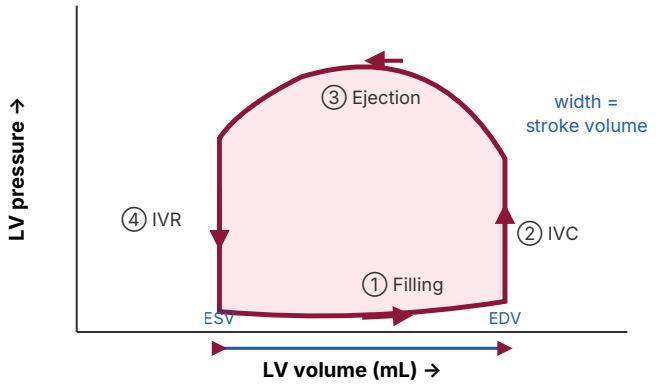


Figure 8.2 — Pressure–volume loop ▶ P–V loop

Sound	Cause	Best heard	Normal?
S1 — "lub"	Closure of mitral and tricuspid valves ▶ Listen	Apex (mitral area)	Yes
S2 — "dub"	Closure of aortic (A2) then pulmonary (P2) valves	Base (2nd intercostal spaces)	Yes; **splits on inspiration** — more blood returns to the right heart, so P2 comes later
S3	Rapid ventricular filling vibrating the wall ▶ Listen S3/S4	Apex, with bell	Normal in children and athletes; in adults suggests heart failure
S4	Atrium pushing blood into a stiff ventricle	Apex, with bell	Always abnormal (e.g., LVH, ischaemia)

Remember the jugular venous pulse (right atrial pressure)

a wave = atrial contraction · **c** wave = tricuspid bulging at start of systole · **x** descent = atrial relaxation · **v** wave = atrial filling with tricuspid closed · **y** descent = tricuspid opens. You can see the same waves on the LA curve in Figure 8.1.

Trauma & Critical care Medicine

CHAPTER 9 · PHYSIOLOGY

The ECG Made Simple

What the waves mean, the 12 leads and a few strips to recognise

📖 Stickers on the chest

The ECG machine printed a pink sheet with wiggly lines. The emergency physician pointed: "Look at V2, V3, V4 — the ST segment is lifted up like a tombstone. Anterior STEMI. Call the cath lab." Anu could not read a single wave. By the end of this chapter she could.

The **electrocardiogram (ECG)** is a recording, from the skin, of the electrical activity of the heart. It does **not** show contraction — it shows the electrical waves that cause contraction. A wave going **towards** a lead gives an upward deflection; going **away** gives a downward deflection. ▶ Animation: how the ECG forms ▶ ECG basics (LITFL)

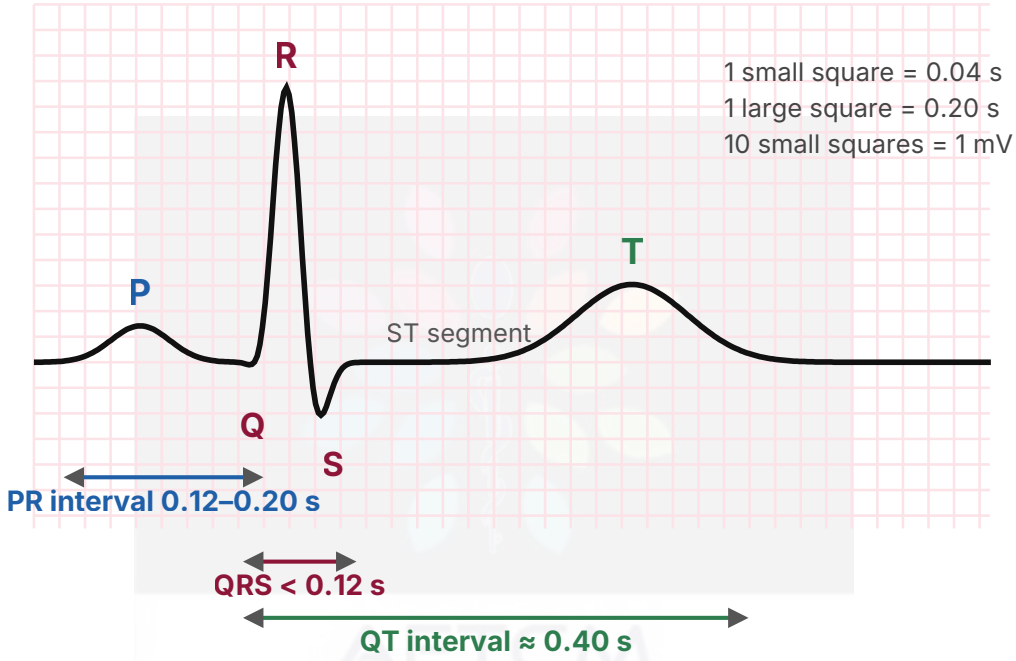


Figure 9.1 — One normal beat, labelled (drawn on standard paper: 25 mm/s, 10 mm/mV)

Part	Represents	Normal
P wave	Atrial depolarisation	< 0.12 s wide, < 2.5 mm tall; upright in lead II
PR interval	Time from atrial activation to ventricular activation (mostly AV-node delay)	0.12–0.20 s (3–5 small squares)
QRS complex	Ventricular depolarisation (atrial repolarisation is hidden in it)	< 0.12 s (usually 0.08–0.10 s)
ST segment	Plateau (phase 2) — all ventricular muscle is depolarised	Flat (isoelectric)
T wave	Ventricular repolarisation	Upright in most leads
QT interval	Total ventricular depolarisation + repolarisation	≈ 0.40 s; QTc < 0.44 s

Heart rate in 5 seconds

If the rhythm is regular: **HR = 300 ÷ number of large squares** between two R waves (or 1500 ÷ small squares). 1 large square → 300; 2 → 150; 3 → 100; 4 → 75; 5 → 60; 6 → 50.

Leads	Look at	Coronary artery
II, III, aVF	Inferior wall	RCA
I, aVL, V5–V6	Lateral wall	LCx
V1–V2	Septum	LAD
V3–V4	Anterior wall	LAD

The 12 leads: 3 **bipolar limb leads** (I, II, III — Einthoven's triangle), 3 **augmented unipolar limb leads** (aVR, aVL, aVF) — these six look at the heart in the vertical (frontal) plane; and 6 **chest leads** (V1–V6) that look in the horizontal plane, from the right side of the septum (V1) to the left lateral wall (V6).

Simple strips to recognise (lead II, drawn to scale)

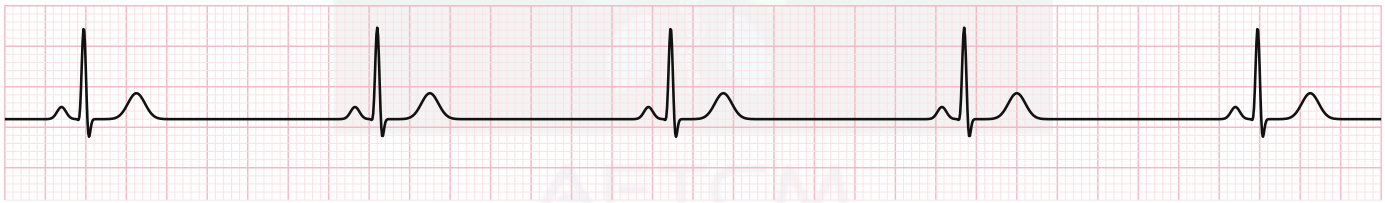
Normal sinus rhythm — rate 75, every P followed by QRS, PR constant



Sinus tachycardia — rate ≈ 125; normal beats, just faster — fever, pain, anxiety, exercise



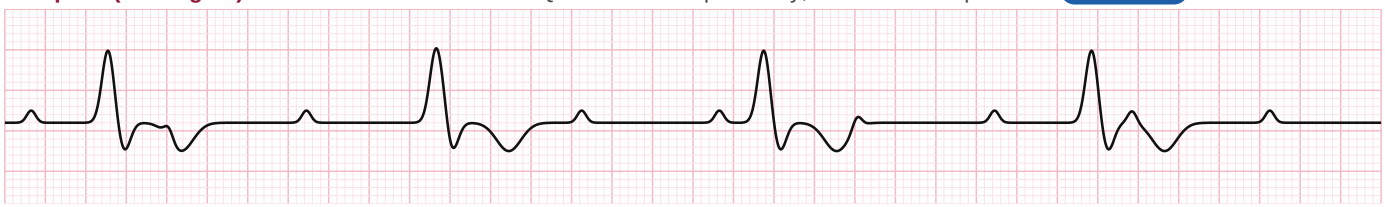
Sinus bradycardia — rate ≈ 40; athletes, sleep, beta blockers, inferior MI



Atrial fibrillation — no P waves, wavy baseline, irregularly irregular QRS ▶ real ECG



Complete (3rd-degree) heart block — P waves and QRS march independently; slow wide escape beats ▶ real ECG



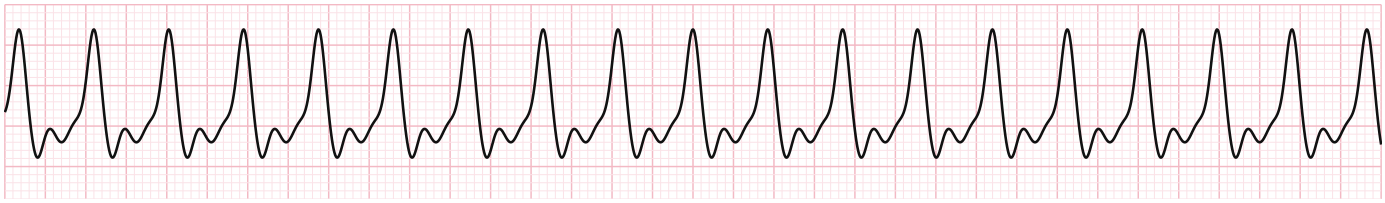
ST elevation (as in Appooppan's V2–V4) — ST segment lifted and merging with T — acute transmural injury ▶ real ECG



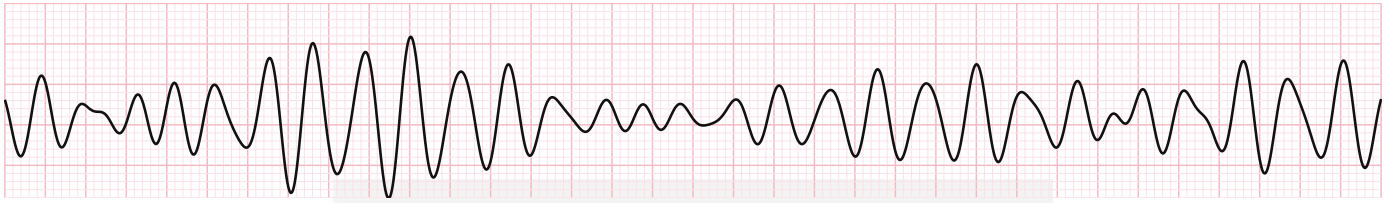
ST depression — flat/down-sloping ST — subendocardial ischaemia (angina on exertion)



Ventricular tachycardia — fast, regular, broad bizarre complexes — dangerous ▶ real ECG



Ventricular fibrillation — chaotic waves, no QRS — cardiac arrest: shock immediately



The strips above are simplified teaching drawings. To see real 12-lead tracings: ▶ Normal 12-lead ▶ Anterior STEMI ▶ LITFL ECG basics

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CHAPTER 10 · PHYSIOLOGY

How Much Does It Pump?

Cardiac output, preload, afterload, contractility and the Frank–Starling law

📖 A rubber band and a balloon

Anu's physiology professor stretched a rubber band. "Stretch it a little — it snaps back a little. Stretch it more — it snaps back harder. Heart muscle is the same. Fill the ventricle more, and it pumps more. That is Starling's law. But over-stretch it for years, like a balloon blown too many times, and it goes slack — that is heart failure."

Cardiac output = Stroke volume × Heart rate

≈ 70 mL × 72/min ≈ **5 L/min** at rest · up to 20–25 L/min in exercise · Cardiac index ≈ 3.2 L/min/m²

Factor	Simple meaning	Increased by	Effect on stroke volume
Preload	How much the ventricle is **stretched** before it contracts (≈ end-diastolic volume)	More venous return — lying down, IV fluids, exercise (muscle pump)	↑ (Frank–Starling)
Afterload	The **load the ventricle pushes against** to open the aortic valve (≈ aortic pressure/ resistance)	High BP, aortic stenosis	↓
Contractility (inotropy)	Strength of contraction at the **same** stretch	Sympathetic (β_1), adrenaline, calcium, digoxin, dobutamine	↑
Heart rate	Beats per minute	Sympathetic; reduced by vagus	↑ CO up to a point; very fast rates cut filling

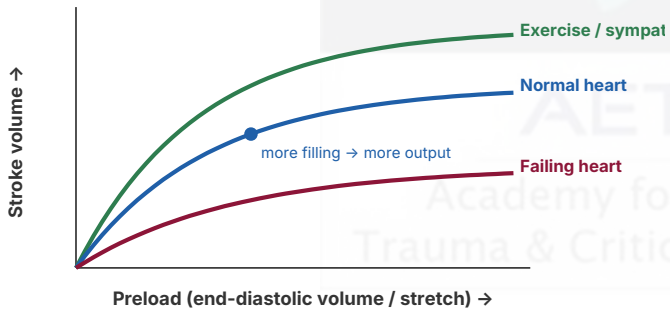


Figure 10.1 — Frank–Starling curves ▶ Explained

Frank–Starling law in one line

Within limits, **the more the heart is filled, the more forcefully it contracts**, so that "what comes in goes out." This keeps the right and left ventricles pumping exactly the same amount, beat after beat.

Why? (1) Stretch brings actin and myosin to the best overlap (sarcomere ≈ 2.2 µm). (2) Stretch makes **troponin C more sensitive to calcium** — a biochemical reason for a physiological law.

🔗 Integration corner — Heart failure

ANATOMY
The LV wall becomes hypertrophied (thick) or dilated (thin and baggy).

PHYSIOLOGY
Ejection fraction falls; the Frank–Starling curve shifts down; the body activates sympathetic system and RAAS to hold up BP — which overloads the heart further.

BIOCHEMISTRY
ATP shortage, poor calcium pumping by **SERCA2a**, and stretched ventricles release **B-type natriuretic peptide (BNP)** — a blood test for heart failure.

CHAPTER 11 · PHYSIOLOGY

Keeping the Pressure Right

Blood pressure — short-term and long-term control; capillary exchange

📖 Standing up too fast

On day 5 Appooppan stood up from the bed and felt dizzy for a second, then fine. The nurse smiled: "His baroreceptors caught it." Anu asked how a few nerve endings in the neck could save someone from fainting.

Blood pressure = Cardiac output × Total peripheral resistance

Mean arterial pressure ≈ diastolic + ⅓ (systolic − diastolic) · e.g., 120/80 → MAP ≈ 93 mmHg

Short-term control — the baroreceptor reflex (seconds)

BP falls (standing up) → Less stretch of carotid sinus (IX) and aortic arch (X) → Fewer impulses to NTS in medulla → ↑ Sympathetic, ↓ vagus → ↑ HR, ↑ contractility, vasoconstriction, venoconstriction → **BP back to normal**

When BP rises, the opposite happens — slower heart, vessels relax. Chemoreceptors (carotid and aortic bodies) mainly sense low O_2 and high CO_2 and help when BP is very low. ▶ Baroreflex diagram

Long-term control — the kidney and RAAS (hours to days)

Low BP / low Na^+ / sympathetic stimulation → juxtaglomerular cells release renin

↓

Renin: angiotensinogen (liver) → angiotensin I

↓

ACE (mainly lungs): angiotensin I → **angiotensin II**

↓

Angiotensin II → vasoconstriction · aldosterone (Na^+ and water kept) · ADH and thirst · cardiac and vessel remodelling

↓

Blood volume and BP rise

▶ RAAS diagram Other long-term players: **ADH** (water), **ANP/BNP** from atria/ventricles (lose Na^+ and water — the heart's own diuretic), and **pressure natriuresis** (kidney passes more salt when BP is high).

Capillaries — Starling forces and lymph

Fluid leaves and re-enters capillaries depending on two pushes: **hydrostatic pressure** (pushes fluid out) and **oncotic pressure** of plasma proteins, mainly albumin (pulls fluid in).

	Arterial end	Venous end
Capillary hydrostatic pressure (out)	≈ 35 mmHg	≈ 15 mmHg
Plasma oncotic pressure (in)	≈ 25 mmHg	≈ 25 mmHg
Net result	**Filtration out**	**Reabsorption in**

Where does extra fluid go — and when do legs swell?

About 2–4 L/day more is filtered than reabsorbed; the **lymphatics** return it to the blood. **Oedema** occurs when hydrostatic pressure rises (heart failure, venous block), oncotic pressure falls (low albumin — liver disease, nephrotic syndrome, malnutrition), capillaries leak (inflammation), or lymphatics are blocked (filariasis).

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CHAPTER 12 · BIOCHEMISTRY

The Engine Room

What the heart burns, how it stores energy, and what happens without oxygen

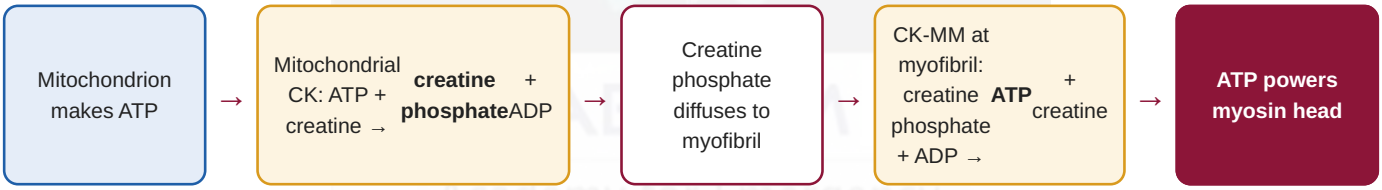
> **📖 Why does it hurt?**
>
> *"The pain is the muscle crying for oxygen," said the cardiologist. Anu's biochemistry teacher later explained it differently: "The pain is lactic acid and adenosine. The heart cell switched to the wrong fuel pathway because oxygen was cut off." Both were right.*

The heart uses about **6 kg of ATP every day** — many times its own weight — yet stores only enough for a few seconds of beating. So it must make ATP non-stop. Heart cells are packed with **mitochondria** (about one-third of the cell volume) and with **myoglobin** to hold oxygen. The heart is a strictly **aerobic** organ.

Fuel	Share of ATP at rest	Comment
Fatty acids (β-oxidation)	**60–70%**	Main fuel; gives the most ATP but needs the most oxygen per ATP
Glucose (glycolysis → pyruvate → Krebs cycle)	≈ 20–30%	Rises after meals, with insulin, and in stress; more ATP per molecule of O_2
Lactate	≈ 10% (more in exercise)	Heart **uses** lactate made by skeletal muscle — converts it back to pyruvate (LDH-1)
Ketone bodies, amino acids	Small	Ketones rise in starvation and diabetes

The creatine phosphate shuttle — the heart's pocket battery

ATP is made in mitochondria but used at the myofibrils. Instead of carrying bulky ATP, the cell uses a small, fast carrier: **creatine phosphate**.



The enzyme is **creatine kinase (CK)**. It has isoenzymes: **CK-MM** (skeletal muscle), **CK-MB** (heart — about 15–20% of heart CK) and CK-BB (brain). When heart cells die, CK-MB leaks out — that is why it is a heart-attack marker.

What happens when the oxygen stops — the ischaemic cascade

Coronary artery blocked → no O_2

↓

Oxidative phosphorylation stops → ATP falls within seconds; creatine phosphate used up first

↓

Switch to **anaerobic glycolysis** → lactate + H^+ → acidosis (pain, with adenosine)

↓

Na^+/K^+-ATPase fails → Na^+ and water enter → cell swells; K^+ leaks out (changes ECG)

↓

Ca^{2+} overload → enzymes activated, mitochondria damaged

↓

Muscle stops contracting within ~1 minute (reversible)

↓

After ~20–40 minutes → membrane ruptures → irreversible death → troponin, CK-MB released

Clinical bridge — "time is muscle"

Because death begins at about 20–40 minutes and spreads over hours, opening the artery early (angioplasty or clot-dissolving drugs) saves muscle. The drug **trimetazidine** works in this engine room: it partly blocks fatty-acid oxidation so the heart burns more glucose, which gives more ATP per molecule of oxygen.

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CHAPTER 13 · BIOCHEMISTRY + PHYSIOLOGY

Molecules That Pull

Contractile proteins, calcium and excitation–contraction coupling

📖 Rowers in a boat

Anu imagined each sarcomere as a boat. The myosin heads are rowers, the actin filament is the water they pull on, and calcium is the coach's whistle — no whistle, no rowing. ATP is the rowers' lunch: without it they cannot even let go of the oar (that is why muscles go stiff after death — rigor mortis).

Protein	Where	Job
Myosin	Thick filament	Heads have **ATPase**; bind actin and pull (power stroke)
Actin	Thin filament	Track that myosin pulls on
Tropomyosin	Lies along actin	Covers myosin-binding sites at rest
Troponin C	On tropomyosin	**Binds Ca^{2+}** — the switch
Troponin I	On tropomyosin	**Inhibits** actin–myosin binding at rest
Troponin T	On tropomyosin	**Tethers** troponin complex to tropomyosin

Cardiac troponin I and T have a slightly different amino-acid sequence from skeletal troponins. Laboratory antibodies detect only the cardiac forms — so a raised troponin means **heart** muscle damage, not leg muscle damage.

Excitation–contraction coupling — from spark to squeeze

Action potential spreads into T-tubules

↓

Phase 2: **L-type Ca^{2+} channels** (dihydropyridine receptors) open — a little Ca^{2+} enters

↓

This Ca^{2+} opens **ryanodine receptors (RyR2)** on the sarcoplasmic reticulum → large Ca^{2+} release (**calcium-induced calcium release**)

↓

Ca^{2+} binds troponin C → tropomyosin moves → myosin binds actin

↓

Cross-bridge cycling using ATP → **contraction**

↓

Relaxation: **SERCA2a** pumps Ca^{2+} back into SR (uses ATP; controlled by phospholamban) and **Na^+/Ca^{2+} exchanger** pushes Ca^{2+} out

🔗 Integration corner — Calcium — one ion, three subjects

ANATOMY

T-tubules lie at the Z-lines; SR surrounds the myofibrils; intercalated discs link the cells.

PHYSIOLOGY

More Ca^{2+} = stronger beat (inotropy). Sympathetic β_1 → cAMP → PKA phosphorylates L-type channels (more Ca^{2+} in) and phospholamban (faster relaxation).

BIOCHEMISTRY

Relaxation also needs ATP (SERCA). In ischaemia and heart failure SERCA2a is weak → slow, incomplete relaxation.

Digoxin

blocks Na^+/K^+-ATPase → less Ca^{2+} leaves through the exchanger → stronger beat.

Cardiac muscle — from tissue to T-tubule

Zoom in step by step: the striped tissue under the microscope, the branching cells joined by intercalated discs, and finally the myofibrils wrapped in sarcoplasmic reticulum with short, broad T-tubules — the stage for the calcium story.

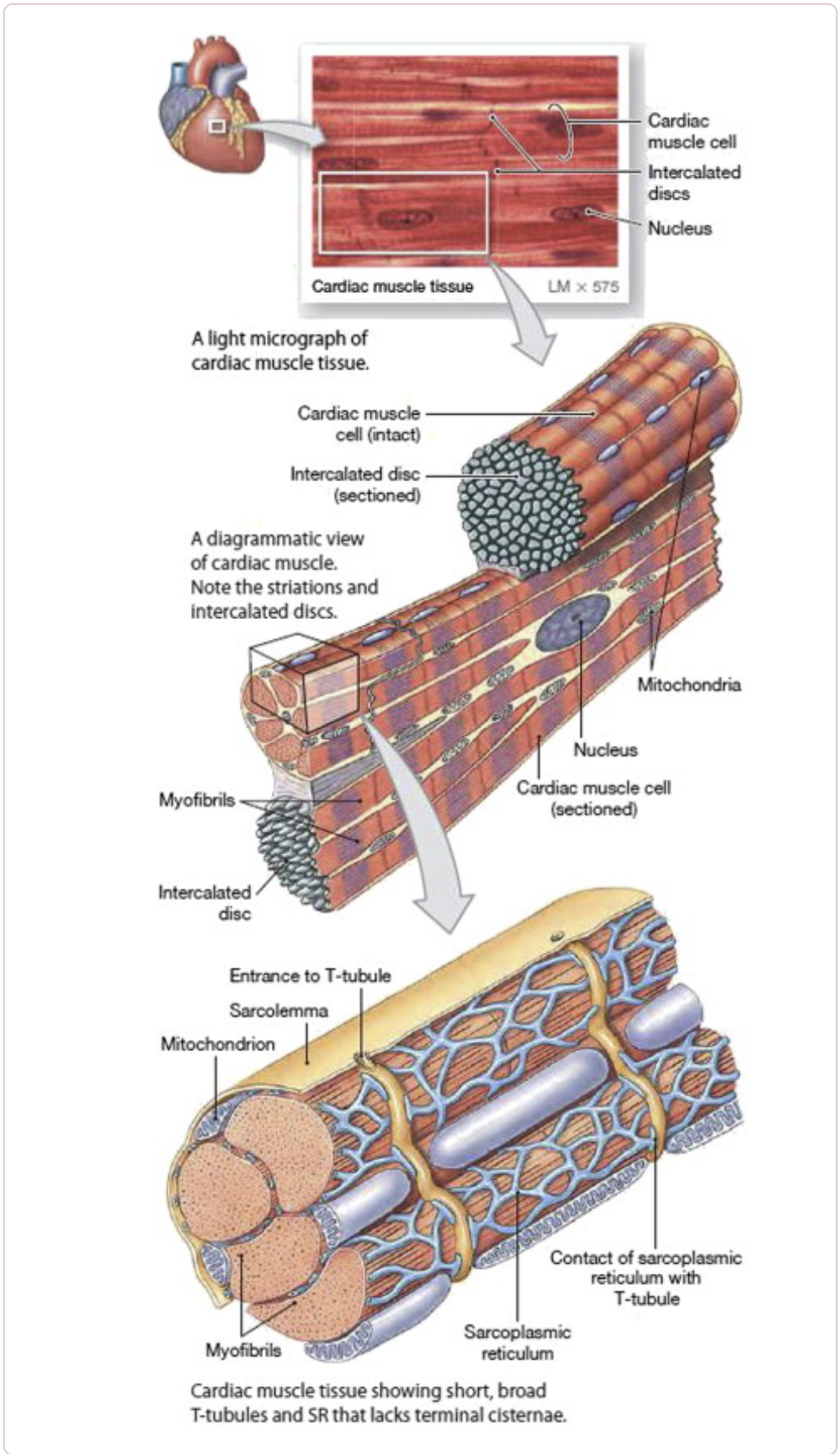


Figure 13.1 — Structure of cardiac muscle: light micrograph, cells with intercalated discs, and the T-tubule / SR system

CHAPTER 14 · BIOCHEMISTRY

Chemistry of Vessel Tone

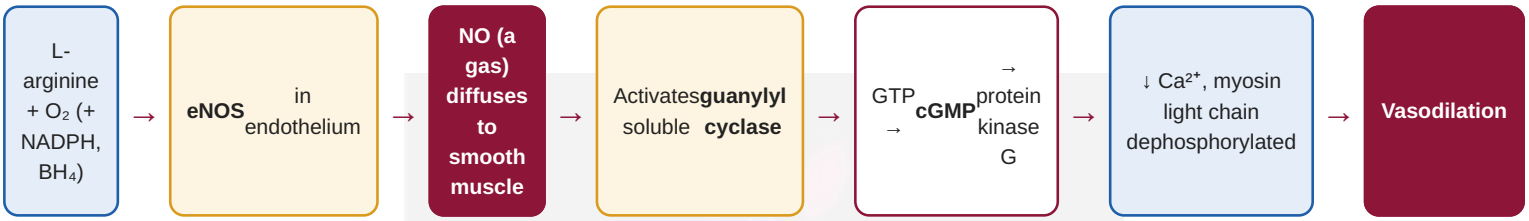
Nitric oxide, endothelin and angiotensin II

📖 The tablet under the tongue

Appooppan's discharge kit had a tiny bottle: "Sorbitrate — put one under the tongue if chest pain comes." The pharmacist said it works in two minutes by making a gas inside the blood vessel. Anu found the gas in her biochemistry book: nitric oxide.

The endothelium is not just a lining; it is a **gland** that constantly releases chemicals to relax or tighten the smooth muscle below it.

Nitric oxide (NO) — the relaxer



Shear stress of flowing blood, acetylcholine and bradykinin increase NO. Nitrates (nitroglycerin, isosorbide) **donate NO**. Sildenafil blocks the enzyme that breaks cGMP (PDE-5) — so taking it with nitrates can drop BP dangerously.

Molecule	Made by / from	Acts through	Effect
Nitric oxide	Endothelium (eNOS) from arginine	sGC → cGMP	Vasodilation; stops platelets sticking
Prostacyclin (PGI_2)	Endothelium (COX)	cAMP	Vasodilation; anti-platelet
Endothelin-1	Endothelium	ET_A receptor → Gq → IP_3 → Ca^{2+}	Very potent vasoconstriction (blocked by bosentan in pulmonary hypertension)
Angiotensin II	ACE from angiotensin I	AT_1 receptor → Gq → IP_3 → Ca^{2+}	Vasoconstriction, aldosterone, growth of heart and vessels
Thromboxane A_2	Platelets (COX-1)	Gq	Vasoconstriction, platelet aggregation (blocked by aspirin)

Remember

Healthy endothelium = lots of NO = relaxed, smooth, non-sticky vessels. Smoking, diabetes, high LDL and high BP damage the endothelium → less NO → the first step of atherosclerosis (**endothelial dysfunction**).

CHAPTER 15 · BIOCHEMISTRY → PATHOLOGY

Fat in the Pipes

Lipoproteins, cholesterol and how atherosclerosis grows

📖 A blockage built over 30 years

"The clot formed in a minute," said the cardiologist, "but the plaque under it took thirty years to build." Appooppan's lipid report showed LDL 168 mg/dL and HDL 32 mg/dL. Anu learnt why one was called 'bad' and the other 'good'.

Fats do not dissolve in blood, so they travel in packages called **lipoproteins** — a core of triglyceride and cholesterol ester wrapped in phospholipid, free cholesterol and **apoproteins**.

Lipoprotein	Made in	Main cargo	Main apoprotein	Job
Chylomicron	Intestine	Dietary triglyceride	B-48	Carries food fat to tissues
VLDL	Liver	Triglyceride made in liver	B-100	Carries liver fat to tissues → becomes IDL → LDL
LDL — "bad"	From VLDL in blood	**Cholesterol**	B-100	Delivers cholesterol to cells via **LDL receptor**; excess enters artery wall
HDL — "good"	Liver, intestine	Cholesterol (picked up)	A-I	**Reverse cholesterol transport** — brings cholesterol from tissues back to liver (LCAT esterifies it)

Cholesterol is made from acetyl-CoA; the **rate-limiting enzyme is HMG-CoA reductase**. When the liver cell makes less cholesterol, it puts more LDL receptors on its surface and pulls LDL out of the blood — exactly how **statins** work.

How a plaque grows — draw this

Endothelial injury (smoking, high BP, diabetes, high LDL)

↓

LDL enters the **tunica intima** and gets **oxidised**

↓

Monocytes enter → become macrophages → swallow oxidised LDL through **scavenger receptors** (not regulated!)

↓

Lipid-laden **foam cells** → **fatty streak** (visible even in teenagers)

↓

Smooth muscle cells migrate, make collagen → **fibrous cap** over a lipid core = plaque

↓

Cap thins and ruptures → platelets stick → clot → artery blocked → myocardial infarction

▶ Atherosclerosis images

🔗 Integration corner — Atherosclerosis

ANATOMY
Affects large elastic and muscular arteries — aorta, coronary, carotid, femoral; the intima thickens.

PHYSIOLOGY
Narrowing raises resistance (flow falls with the 4th power of radius — Poiseuille); stable plaque → angina on exertion; ruptured plaque → MI.

BIOCHEMISTRY
Oxidised LDL → scavenger receptors → foam cells; HDL protects; HMG-CoA reductase is the drug target.

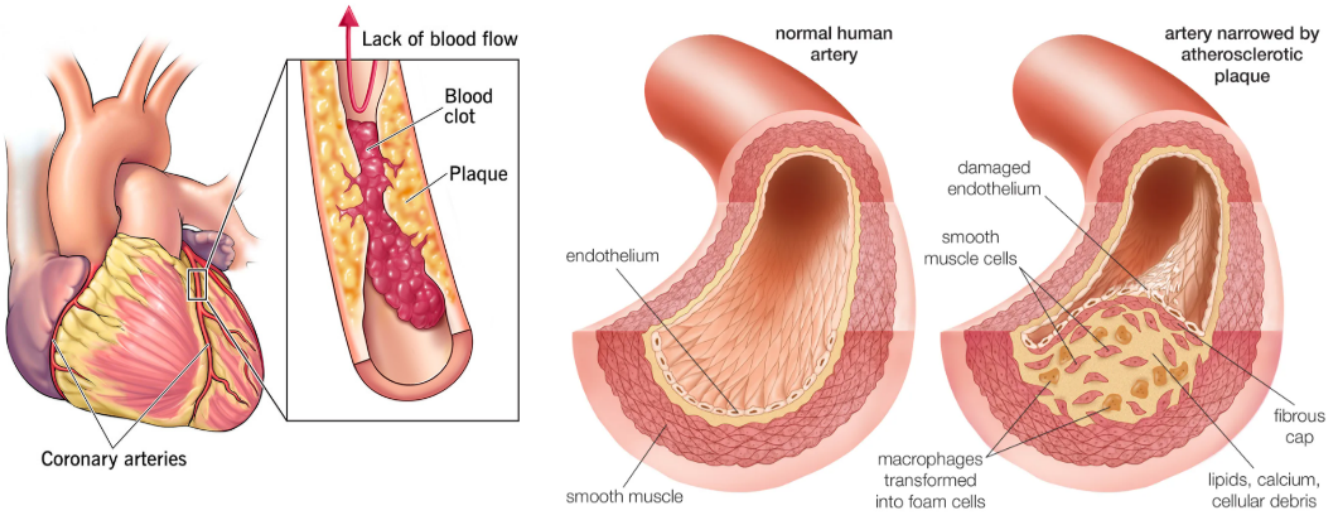


Figure 15.1 — From normal artery to blocked coronary — endothelial damage, foam cells and a fibrous-capped plaque; rupture with clot stops blood flow (Image: © Encyclopædia Britannica)

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CHAPTER 16 · BIOCHEMISTRY → CLINICAL

When Heart Cells Die

Cardiac biomarkers — what leaks out, when and why

📖 Send troponin

Appooppan's first troponin at 1 hour was slightly high; at 3 hours it was ten times higher. "The rise confirms that heart muscle is dying, not just short of blood," the doctor told the family. Anu noted: one value is a photograph; two values make a movie.

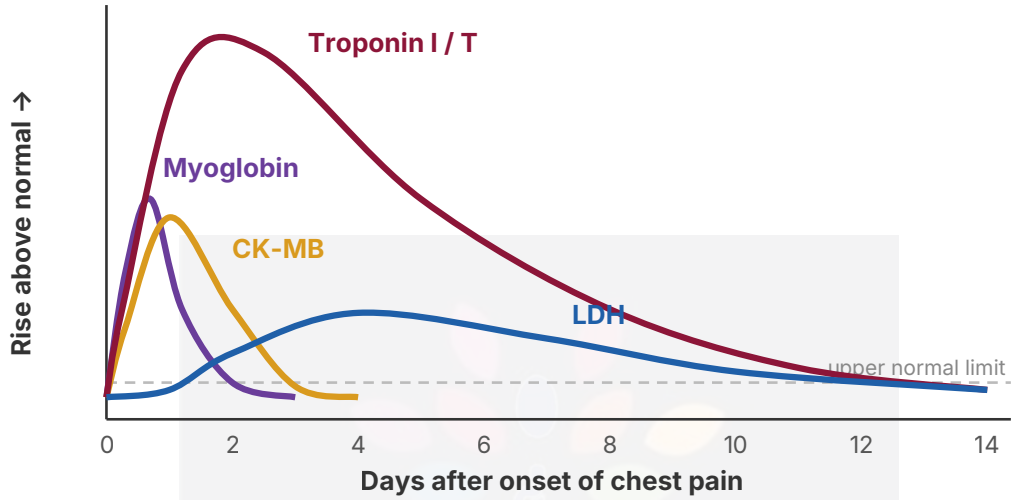


Figure 16.1 — Typical rise and fall of cardiac markers after a heart attack (schematic)

Marker	Starts rising	Peaks	Back to normal	Points to remember
Cardiac troponin I / T	3–4 h (high-sensitivity: 1–3 h)	24–48 h	7–14 days	**Marker of choice** — heart-specific, very sensitive; stays long, so it can detect a late-presenting MI
CK-MB	4–6 h	~24 h	48–72 h	Good for detecting a **re-infarction** (falls quickly)
Myoglobin	1–3 h	6–12 h	~24 h	Earliest but not specific (also skeletal muscle)
LDH (LDH-1)	24–48 h	3–6 days	8–14 days	Normally LDH-2 > LDH-1; after MI **LDH-1 > LDH-2 ("flipped" pattern)** — historical
BNP / NT-proBNP	—	—	—	Released by stretched ventricles — marker of **heart failure**, not of MI

Why troponin and not just "cardiac enzymes"?

Troponin is a **structural protein**, not an enzyme. Its cardiac forms (cTnI, cTnT) differ from skeletal forms, so the test is heart-specific. CK and LDH are **enzymes** present in many tissues; CK-MB is only relatively specific to heart.

CHAPTER 17 · INTEGRATION

Putting It All Together

Four integration stories for the examination and the viva

📖 Anu's notebook

Six weeks later Appooppan was walking 2 km a day. Anu rewrote everything she had learnt as one big table so she would never again learn the heart in three pieces.

Scenario	Anatomy	Physiology	Biochemistry
Myocardial infarction (heart attack)	Occlusion of a coronary artery — e.g., LAD → anterior wall and septum	Lost contraction of that wall; ST elevation in matching leads; may cause arrhythmia or heart block	No O_2 → no oxidative phosphorylation → anaerobic glycolysis → lactic acidosis → Ca^{2+} overload → membrane rupture → troponin, CK-MB released
Atherosclerosis	Endothelial damage in the intima of muscular/elastic arteries	Narrowed lumen → raised resistance → angina; stiff arteries → high systolic BP	Oxidised LDL → scavenger receptors on macrophages → foam cells → fatty streak → plaque
Congenital heart defect (ASD/VSD/PDA)	Septum or ductus fails to close	Left-to-right shunt → more pulmonary flow → later pulmonary hypertension and shunt reversal	Hypoxia → HIF-1α → erythropoietin → polycythaemia; prostaglandins keep ductus open
Heart failure	Hypertrophy or dilatation of ventricles	Low ejection fraction, Frank–Starling curve shifts down, RAAS and sympathetic activation, oedema (Starling forces)	ATP depletion, weak SERCA2a calcium handling, high BNP

The whole story of Appooppan — one flowchart

Risk factors: smoking, high LDL, low HDL (Biochemistry) → endothelial damage, less NO

↓

Plaque in the LAD (Anatomy) builds over 30 years

↓

Plaque ruptures → platelets (thromboxane A_2) → clot → LAD blocked

↓

Anterior wall stops contracting; ST elevation V1–V4 (Physiology)

↓

No ATP → cell death → troponin rises (Biochemistry)

↓

Angioplasty + drugs that act on platelets, cholesterol, heart rate and RAAS (Clinical bridge → next chapter)

High-yield study tips for First MBBS

- **Draw three diagrams from memory:** coronary arteries (Anatomy), Wiggers diagram (Physiology), β-oxidation / creatine phosphate shuttle (Biochemistry).
- **Learn the ions:** SA node upstroke = Ca^{2+}; ventricle upstroke = Na^{+}; plateau = Ca^{2+}; repolarisation = K^{+}.
- **Focus on the normal:** first-year examiners test how the healthy heart works and the first molecular signs of its injury.
- **Always link:** for every structure ask “what does it do?” and “what molecule makes it work?”

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CHAPTER 18 · CLINICAL BRIDGE

Drugs That Help the Heart

How common cardiac drugs work — linked to what you learnt (no doses — for understanding only)

📖 The discharge bag

Appooppan went home with seven medicines. Anu laid them out on the table and, using her first-year notes, wrote next to each one the exact page of physiology or biochemistry it was acting on. The medicines suddenly made sense.

A. Drugs for myocardial ischaemia (angina and heart attack)

Class	Examples	How it works	Links to
Nitrates	Nitroglycerin, isosorbide dinitrate / mononitrate	Release **NO** → cGMP → dilate veins (↓ preload, ↓ O_2 demand) and coronary arteries	Ch 14 (NO), Ch 10 (preload)
Beta blockers	Metoprolol, atenolol, bisoprolol	Block β_1 → ↓ heart rate and contractility → ↓ O_2 demand; longer diastole → more coronary filling	Ch 4, Ch 7, Ch 10
Calcium channel blockers	Amlodipine (vessels); verapamil, diltiazem (heart)	Block L-type Ca^{2+} channels → relax arteries / slow the heart	Ch 7, Ch 13
Aspirin	Aspirin	Irreversibly blocks platelet **COX-1** → no thromboxane A_2 → platelets stick less	Ch 14, Ch 15
P2Y₁₂ blockers	Clopidogrel, ticagrelor, prasugrel	Block platelet ADP receptor → less aggregation (given with aspirin after a stent)	Ch 15
Anticoagulants	Heparin, enoxaparin	Activate **antithrombin** → inhibit thrombin and factor Xa → stop clot growing	Clot formation
Thrombolytics	Streptokinase, tenecteplase	Convert plasminogen → **plasmin**, which dissolves fibrin in the clot	"Time is muscle" (Ch 12)
Statins	Atorvastatin, rosuvastatin	Inhibit **HMG-CoA reductase** → more LDL receptors → ↓ LDL; stabilise plaque	Ch 15
Others	Ivabradine; ranolazine; nicorandil; trimetazidine	Ivabradine blocks funny current (I_f) → ↓ HR; ranolazine blocks late Na^+ current; nicorandil opens K-ATP channels + NO; trimetazidine shifts fuel from fat to glucose	Ch 7, Ch 12

B. Drugs for heart failure

Class	Examples	How it works	Links to
Loop diuretics	Furosemide, torsemide	Block Na^+-K^+-$2Cl^-$ transporter in loop of Henle → remove excess fluid → relieve breathlessness and oedema	Ch 11 (Starling forces)
ACE inhibitors	Enalapril, ramipril	Block ACE → less angiotensin II and aldosterone; also ↑ bradykinin (dry cough)	Ch 11, Ch 14 (RAAS)
Angiotensin receptor blockers	Losartan, telmisartan	Block AT_1 receptor	Ch 14
ARNI	Sacubitril + valsartan	Sacubitril blocks neprilysin → more natriuretic peptide (BNP) action; valsartan blocks AT_1	Ch 10, Ch 16 (BNP)

Beta blockers	Carvedilol, bisoprolol, metoprolol succinate	Protect the heart from long-term sympathetic over-drive; reduce deaths	Ch 10, Ch 11
Mineralocorticoid receptor antagonists	Spironolactone, eplerenone	Block aldosterone → less Na^+ retention and less fibrosis; keep K^+	Ch 11
SGLT2 inhibitors	Dapagliflozin, empagliflozin	Block glucose-sodium reabsorption in proximal tubule → lose Na^+ and water; improve heart energetics	Ch 11, Ch 12
Digoxin	Digoxin	Inhibits **Na^+/K^+-ATPase** → more intracellular Ca^{2+} → stronger beat; ↑ vagal tone → slows AV node	Ch 13, Ch 7
Inotropes (in hospital)	Dobutamine	β_1 agonist → cAMP → more Ca^{2+} → stronger contraction	Ch 13

C. Antiarrhythmic drugs (Vaughan Williams classification)

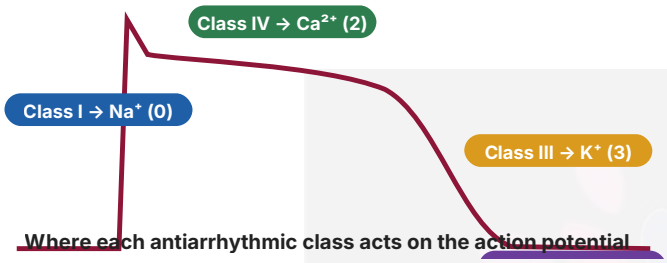


Figure 18.1 — Each class acts on a different phase

Memory hook — "Some Block Potassium Channels"

I = **S**odium channel blockers · **II** = **B**eta blockers · **III** = **P**otassium channel blockers · **IV** = **C**alcium channel blockers.

Class	Examples	How it works	Typical use (for understanding)
Ia	Procainamide, quinidine	Moderate Na^+ block + some K^+ block → slower upstroke, longer action potential	Some atrial and ventricular arrhythmias
Ib	Lidocaine, mexiletine	Weak Na^+ block, prefers ischaemic tissue; shortens action potential	Ventricular arrhythmia after MI
Ic	Flecainide, propafenone	Strong Na^+ block → markedly slow conduction	Atrial fibrillation in structurally normal hearts
II	Metoprolol, esmolol, propranolol	Block β_1 → flatter phase 4 in SA node, slower AV conduction	Rate control in AF, after MI
III	Amiodarone, sotalol	Block K^+ channels → longer phase 3 and refractory period (long QT)	AF and ventricular tachycardia
IV	Verapamil, diltiazem	Block L-type Ca^{2+} channels in SA/AV node → slower AV conduction	SVT, rate control in AF
Others	Adenosine; atropine; magnesium; digoxin	Adenosine opens K^+ channels in AV node → brief block; atropine blocks vagal M_2 → faster rate; Mg^{2+} stabilises membranes	Adenosine — SVT; atropine — bradycardia; Mg^{2+} — torsades

Why first-year students should know drugs at all

Every heart drug is a **physiology or biochemistry lesson in a tablet**. If you know the funny current, you understand ivabradine; if you know HMG-CoA reductase, you understand statins; if you know Na^+/K^+-ATPase and the Na^+/Ca^{2+} exchanger, you understand digoxin. Learn the mechanism now; doses and side-effects will come in later years.

CHAPTER 19 · VIVA VOCE

Viva Questions with Short Answers

40 questions across Anatomy, Physiology, Biochemistry and the clinical bridge

A **Q1. Where is the apex beat normally felt?**
Left 5th intercostal space, just medial to the midclavicular line (about 9 cm from midline); formed by the left ventricle.

A **Q2. Which chamber forms most of the anterior surface? The base?**
Right ventricle; left atrium.

A **Q3. What is the transverse pericardial sinus and why is it important?**
A passage behind the aorta and pulmonary trunk and in front of the SVC; surgeons pass a clamp through it to stop blood flow during cardiac surgery.

A **Q4. What prevents the AV valves from everting?**
Chordae tendineae attached to papillary muscles, which contract with the ventricle.

A **Q5. What is cardiac dominance? Commonest type?**
Dominance is decided by which artery gives the posterior interventricular artery; right dominance in ~70–80%.

A **Q6. Which artery usually supplies the SA node and the AV node?**
RCA — SA node in ~60%, AV node in ~80–90%.

A **Q7. Name the remnants of the fetal shunts.**
Fossa ovalis, ligamentum arteriosum, ligamentum venosum; umbilical vein → ligamentum teres; umbilical arteries → medial umbilical ligaments.

A **Q8. Why does the foramen ovale close at birth?**
Lungs expand → pulmonary resistance falls → more blood returns to LA → LA pressure exceeds RA → flap closes.

A **Q9. What are intercalated discs?**
Junctions between cardiac muscle cells containing desmosomes, fascia adherens and gap junctions — make the heart a functional syncytium.

A **Q10. Why is the aorta an elastic artery?**
Its elastic lamellae stretch in systole and recoil in diastole, keeping flow continuous (Windkessel effect).

P **Q11. Why is the SA node the pacemaker?**
It has the fastest rate of spontaneous phase-4 depolarisation (funny current).

P **Q12. What is the importance of AV nodal delay?**
It allows the atria to finish contracting before the ventricles contract.

P **Q13. Why can't cardiac muscle be tetanised?**
Its long refractory period (due to the plateau) lasts almost as long as contraction.

P **Q14. Ion responsible for the plateau phase?**
Calcium entry through L-type channels, balanced by K^+ exit.

P **Q15. What are the isovolumetric phases?**
Periods when all four valves are closed — isovolumetric contraction (after S1) and isovolumetric relaxation (after S2).

P **Q16. What causes physiological splitting of S2?**
On inspiration more blood returns to the right heart, so pulmonary valve closure (P2) is delayed.

P **Q17. When is S3 normal? Is S4 ever normal?**
S3 is normal in children, young adults and athletes; S4 is always abnormal.

P **Q18. What does each ECG wave represent?**
P — atrial depolarisation; QRS — ventricular depolarisation; T — ventricular repolarisation.

P **Q19. Why is atrial repolarisation not seen?**
It is hidden within the QRS complex.

P **Q20. Normal PR interval and QRS duration?**
PR 0.12–0.20 s; QRS less than 0.12 s.

P **Q21. State the Frank–Starling law.**
Within limits, the force of contraction is proportional to the initial length (stretch) of the muscle fibres.

P Q22. Define preload and afterload.
Preload — ventricular stretch at end of diastole (EDV); afterload — the resistance the ventricle must overcome to eject (aortic pressure).

P Q23. Describe the baroreceptor reflex for a fall in BP.
Less firing from carotid sinus (IX) and aortic arch (X) → medulla → ↑ sympathetic, ↓ vagal → ↑ HR, contractility and vasoconstriction.

P Q24. What are Starling forces?
Capillary hydrostatic pressure (pushes out) and plasma oncotic pressure (pulls in); their balance decides filtration or reabsorption.

B Q25. What is the main fuel of the resting heart?
Fatty acids by β-oxidation — about 60–70% of ATP.

B Q26. What is the role of creatine phosphate in the heart?
An energy shuttle and buffer: carries high-energy phosphate from mitochondria to myofibrils, regenerating ATP via creatine kinase.

B Q27. Name the troponin subunits and their roles.
Troponin C binds Ca^{2+}; troponin I inhibits actin–myosin binding; troponin T binds to tropomyosin.

B Q28. What is calcium-induced calcium release?
Ca^{2+} entering via L-type channels opens ryanodine receptors on the SR, releasing much more Ca^{2+}.

B Q29. Which pump relaxes the heart?
SERCA2a, which pumps Ca^{2+} back into the sarcoplasmic reticulum (uses ATP).

B Q30. How does nitric oxide relax blood vessels?
NO activates soluble guanylyl cyclase → cGMP → protein kinase G → lower Ca^{2+} → smooth muscle relaxation.

B Q31. Why is LDL 'bad' and HDL 'good'?
LDL carries cholesterol into tissues and the artery wall; HDL carries cholesterol back to the liver (reverse cholesterol transport).

B Q32. What is a foam cell?
A macrophage filled with oxidised LDL taken up through scavenger receptors.

B Q33. Best biomarker for MI? Best for re-infarction?
Cardiac troponin I/T; CK-MB (returns to normal within 2–3 days).

B Q34. What is the 'flipped' LDH pattern?
After MI, LDH-1 becomes higher than LDH-2.

Q35. How does aspirin prevent heart attacks?
Irreversibly inhibits platelet COX-1 → less thromboxane A_2 → less platelet aggregation.

Q36. How do statins lower LDL?
Inhibit HMG-CoA reductase → liver makes more LDL receptors → removes LDL from blood.

Q37. How does digoxin increase contractility?
Inhibits Na^+/K^+-ATPase → higher intracellular Na^+ → less Ca^{2+} extrusion by Na^+/Ca^{2+} exchanger → more Ca^{2+} for contraction.

Q38. How does ivabradine slow the heart?
Blocks the funny current (I_f) in the SA node, flattening phase 4.

Q39. Name the four Vaughan Williams classes.
I — Na^+ channel blockers; II — beta blockers; III — K^+ channel blockers; IV — Ca^{2+} channel blockers.

Q40. Why are nitrates and sildenafil not given together?
Both increase cGMP (one makes more, the other stops its breakdown) → severe fall in BP.

VISUAL ATLAS

See It Move — Tap or Scan

Free animations, images, ECGs and heart-sound audio from Wikimedia, LITFL, Kenhub, CV Physiology and other open sites

Cardiac cycle
▶ Animation
Animated heart synced with Wiggers

Wiggers diagram
▶ Image
Pressures, volume, ECG, sounds

Normal heart sounds
▶ YouTube
S1 and S2 — listen

S1, S2, S3, S4
▶ YouTube
Heart sounds for beginners

Pressure–volume loop
▶ Image
Normal LV P–V loop

Frank–Starling
▶ Article
Curves, preload, inotropy

Heart anatomy
▶ Images
Chambers, surfaces, valves

Coronary arteries
▶ Images
Arteries and cardiac veins

Pericardium
▶ Images
Layers and sinuses

Fetal circulation
▶ Diagram
Shunts and changes at birth

Cardiac muscle
▶ Histology
Intercalated discs, branching

Conducting system
▶ Animation
SA node to Purkinje

Action potential
▶ Figures
Pacemaker vs ventricular

How ECG forms
▶ Animation
Wave spreading with ECG

Normal 12-lead ECG
▶ ECG
Waves, intervals, segments

ECG basics
▶ ECG
LITFL library basics

Atrial fibrillation
▶ ECG
Real tracings

Complete heart block
▶ ECG
AV dissociation

Anterior STEMI
▶ ECG
LAD occlusion

Ventricular tachycardia
▶ ECG
Broad complex tachycardia

Atherosclerosis
▶ Images
Plaque, foam cells

Baroreceptor reflex
▶ Diagram
Short-term BP control

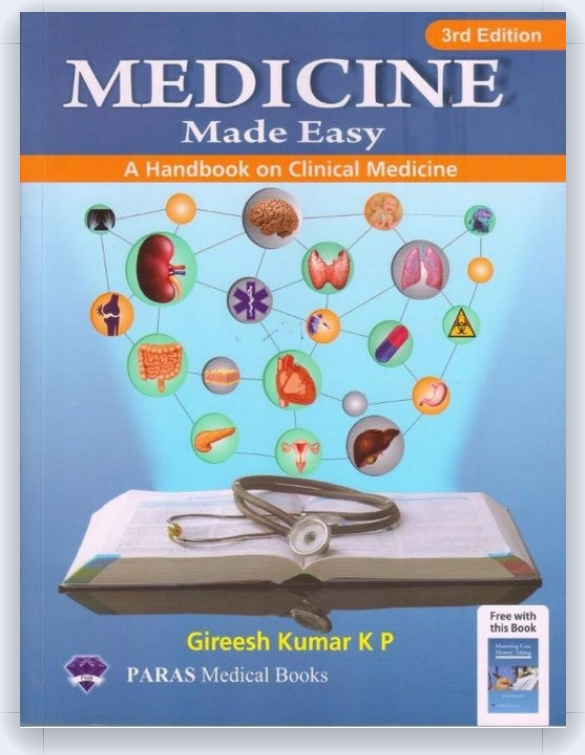
RAAS
▶ Diagram
Renin–angiotensin–aldosterone

Heart sounds lecture
▶ YouTube
Normal & abnormal, with audio

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