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Human Anatomy, Physiology and Pathophysiology I

BP104T — B.Pharm Semester I

Rapid Revision Notes

One-night-pass companion: every topic, only the core

As per PCI B.Pharm (NEP 2020) syllabus

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Unit I — Introduction to Human Body, Cell & Tissue Organisation, Cell Injury, Integumentary System

Anatomy, physiology, pathophysiology — what each means

Anatomy = study of body structure and the relation between parts. Physiology = study of how those parts work. Pathophysiology = study of what goes wrong in disease — this is your bridge to pharmacology: disease mechanism here, drug action later.

Levels of structural organisation

Six levels, small to big: chemical (atoms, molecules) → cellular (the cell) → tissue (groups of similar cells) → organ (heart, liver) → organ-system (cardiovascular) → organismal (the whole human). The body has 11 organ systems — integumentary, skeletal, muscular, nervous, endocrine, cardiovascular, lymphatic/immune, respiratory, digestive, urinary, reproductive.

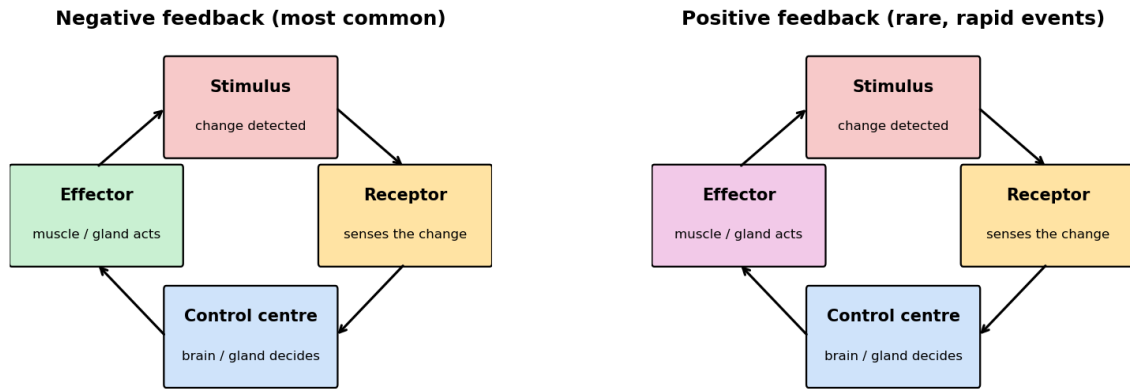
Basic life processes

Organisation, metabolism (catabolism + anabolism), responsiveness, movement, growth, differentiation, reproduction. Favourite one-mark list — learn it in order.

Homeostasis and feedback systems

Homeostasis = keeping the internal environment steady (temperature ~37 °C, blood pH 7.35–7.45, fasting glucose ~70–110 mg/dL) despite outside changes. Every feedback loop has three parts: receptor (detects change) → control centre (brain or gland, decides) → effector (muscle or gland, acts).

- Negative feedback — response reverses the change; most common. High glucose → insulin → glucose falls. Heat → sweating and vasodilation.
- Positive feedback — response strengthens the change; rare, for rapid complete events. Oxytocin in childbirth (contractions → more oxytocin → stronger contractions); platelet plug formation in clotting.



Example: high blood glucose → insulin released → glucose falls (change reversed)

Examples: oxytocin in childbirth (contractions strengthen); platelet plug in clotting

Negative vs positive feedback loops

Remember: Exam favourite — "Explain homeostasis with an example." Draw the loop: stimulus → receptor → control centre → effector → response.

Basic anatomical terminologies

- Anatomical position: upright, feet together, palms facing forward — all direction terms assume this.
- Planes: sagittal (left/right), frontal or coronal (front/back), transverse or horizontal (upper/lower).
- Directions: superior/inferior (toward head/feet), anterior (ventral)/posterior (dorsal), medial/lateral (toward/away from midline), proximal/distal (nearer/farther from trunk), superficial/deep.
- Body cavities: dorsal cavity (cranial + vertebral, holds brain and spinal cord); ventral cavity (thoracic with pleural and pericardial cavities + abdominopelvic).
- Abdominopelvic regions: nine regions (hypochondriac, epigastric, lumbar, umbilical, iliac/inguinal, hypogastric) or four quadrants (RUQ, LUQ, RLQ, LLQ) — RLQ pain suggests appendicitis.

Structure and functions of the cell

The cell is the structural and functional unit of life. Plasma membrane — phospholipid bilayer (fluid-mosaic model), selective barrier. Nucleus — DNA in chromosomes, controls the cell; nucleolus makes ribosomes. Mitochondria — ATP from aerobic respiration, own DNA. Ribosomes — protein synthesis (free or on rough ER). Rough ER — protein folding; smooth ER — lipid synthesis, detox, calcium storage. Golgi apparatus — modifies and packages proteins. Lysosomes — digestive enzymes. Cytoskeleton — microtubules, actin microfilaments, intermediate filaments. Centrosome/centrioles — organise cell division.

Transport across the cell membrane

Passive (no energy, down the gradient): simple diffusion (O_2 , CO_2), facilitated diffusion (aquaporins, GLUT carriers — no energy but saturable), osmosis (water toward higher solute concentration — know isotonic, hypotonic, hypertonic), filtration (pressure-driven, e.g. glomerulus).

Active (ATP, against the gradient): Na^+/K^+ ATPase pumps 3 Na^+ out and 2 K^+ in — maintains membrane potential; digoxin inhibits this pump. Secondary active transport uses that Na^+ gradient: symport (same direction, e.g. Na^+ /glucose in gut — how ORS works) and antiport (opposite, e.g. Na^+/Ca^{2+}).

Vesicular transport (large particles): endocytosis — phagocytosis (cell eating), pinocytosis (cell drinking), receptor-mediated (LDL); exocytosis (neurotransmitters, insulin out); transcytosis (across the cell).

Remember: The Na^+/K^+ pump (3 out, 2 in) is the single most exam-worthy transport protein — and it is exactly what digoxin targets in the heart.

Cell division

Interphase ($G_1 \rightarrow S \rightarrow G_2$): cell grows, DNA replicates — most of the cell's life. Mitosis (prophase $\rightarrow$ metaphase $\rightarrow$ anaphase $\rightarrow$ telophase $\rightarrow$ cytokinesis): one cell $\rightarrow$ two identical diploid cells; growth and repair. Meiosis: gonads only; one cell $\rightarrow$ four haploid gametes (46 $\rightarrow$ 23 chromosomes). Pharmacy link: anticancer drugs hit dividing cells — antimetabolites act in S phase, vinca alkaloids block the spindle in M phase.

Cell junctions

Tight junctions — seal cells (gut lining, blood-brain barrier); nothing leaks between. Adherens junctions — anchor with actin filaments. Desmosomes — strong "spot welds" with intermediate filaments; skin and cardiac muscle. Gap junctions — connexon channels let ions pass directly; cardiac and smooth muscle contract as a unit. Hemidesmosomes — anchor cells to the basement membrane.

Mnemonic: Tight = seal, Desmo = weld, Gap = connect. Match each junction to its tissue — a standard 2-mark question.

Forms of cell communication

Four syllabus forms: contact-dependent (juxtacrine) — cells touch, signal stays on the surface (immune recognition). Paracrine — acts on nearby cells (histamine in inflammation). Synaptic — crosses a synapse to a specific target (neurotransmission). Endocrine — hormone travels in blood to distant targets (insulin, thyroid hormone).

Classification of tissues

Four types. Epithelial — covers surfaces, lines cavities, forms glands: simple squamous (alveoli, endothelium — filtration/diffusion), simple cuboidal (kidney tubules), simple columnar (gut lining with goblet cells), stratified squamous keratinised (skin) vs non-keratinised (mouth, oesophagus), pseudostratified ciliated columnar (trachea), transitional (bladder — stretches). Connective — cells scattered in matrix: loose/dense proper (areolar, tendons, ligaments), cartilage (hyaline, elastic, fibrocartilage), bone, blood. Muscular — skeletal (striated, voluntary, multinucleated), cardiac (striated, branched, intercalated discs, involuntary), smooth (spindle-shaped, involuntary; gut, vessel walls). Nervous — neurons conduct impulses, neuroglia support.

Causes of cellular injury

Hypoxia and ischaemia (most common — myocardial infarction), physical agents (trauma, heat, radiation), chemicals and drugs, infectious agents, immunologic reactions (autoimmunity, hypersensitivity), nutritional imbalance, genetic defects.

Pathogenesis of cell injury

ATP depletion → Na^+/K^+ pump fails → Na^+ and water enter → cell swelling. Cell membrane damage — lipid peroxidation by free radicals, membrane goes leaky. Mitochondrial damage — permeability transition pore opens, cytochrome c released → apoptosis; ATP collapses. Cytosolic calcium overload — activates destructive enzymes (phospholipases, proteases, endonucleases). Ribosome and nuclear damage — protein synthesis stops, DNA injured; irreparable DNA injury = cell death.

Morphology of cell injury — reversible changes

Cell swelling (hydropic degeneration) — the first change, Na^+ /water entry. Fatty change (steatosis) — lipid droplets in liver cells, e.g. alcoholism. Reversible if the cause is removed.

Cell death — necrosis vs apoptosis

Necrosis — unplanned death with inflammation. Types: coagulative (heart, kidney — architecture preserved), liquefactive (brain, abscesses — turns to liquid), caseous (TB — cheese-like), fat necrosis (pancreatitis), fibrinoid (vessel walls in hypertension), gangrenous (limb ischaemia + infection).

Apoptosis — programmed, tidy death: cell shrinks, chromatin condenses, membrane blebs, fragments eaten by neighbours — no inflammation. Driven by caspases, triggered by cytochrome c release.

Remember: Necrosis = messy death with inflammation; apoptosis = tidy programmed death without inflammation. Examiners ask this difference every year.

Adaptive changes of cells

Atrophy — smaller cells (disuse atrophy in a plaster cast). Hypertrophy — bigger cells (left ventricular hypertrophy in hypertension; muscle with exercise). Hyperplasia — more cells (benign prostatic hyperplasia). Metaplasia — one adult cell type replaced by another (smoker's bronchial epithelium: columnar → squamous; reversible). Dysplasia — disordered growth, premalignant (cervical dysplasia) — not a benign adaptation. Intracellular accumulations: lipids (fatty liver), proteins, glycogen, pigments (lipofuscin — "wear and tear", haemosiderin in iron overload).

Mnemonic: Atrophy/Hypertrophy change SIZE; Hyperplasia changes NUMBER; Metaplasia changes TYPE; Dysplasia is DISORDERED and dangerous.

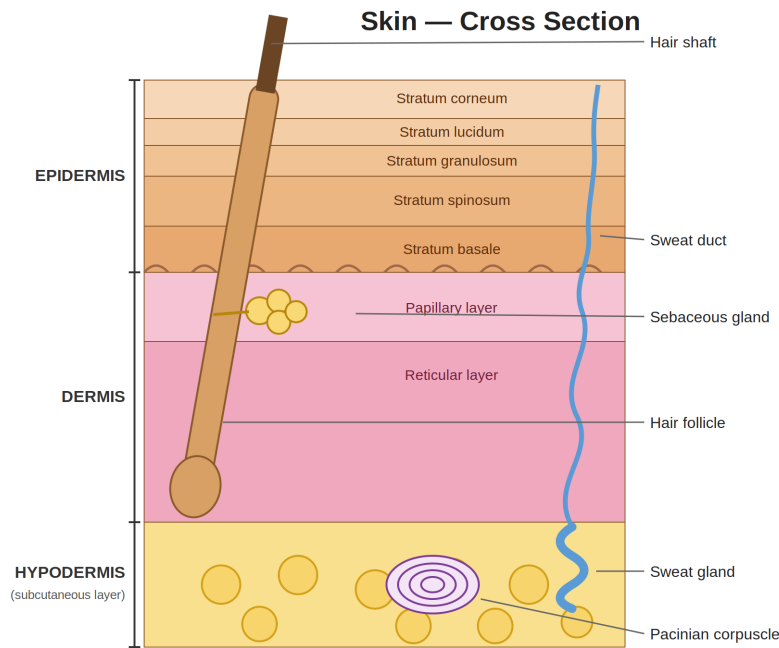
Common medical terminologies

Learn the blocks and you can decode hundreds of terms. Prefixes: hyper- (excess), hypo- (deficient), a-/an- (without), dys- (abnormal/painful), brady- (slow), tachy- (fast), endo- (inside), peri- (around), sub- (below). Suffixes: -itis (inflammation: arthritis, dermatitis), -oma (tumour), -pathy (disease), -emia (blood condition: anaemia), -lysis (breakdown: haemolysis), -plasia (growth), -rrhage (bursting forth: haemorrhage), -stasis (standing still: haemostasis). Practise: hypertension, tachycardia, bradycardia, dyspnoea, gastritis, nephritis.

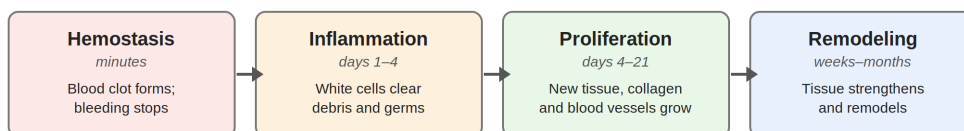
Structure and functions of skin

Largest organ (~2 m², ~16% of body weight). Epidermis — keratinised stratified squamous, no blood vessels; deep to surface: basale (stem cells divide) → spinosum → granulosum → lucidum (thick skin only: palms, soles) → corneum (dead cells, shed). Cells: keratinocytes, melanocytes (melanin, UV protection), Langerhans (immune), Merkel (touch). Dermis — connective tissue with vessels, nerves, hair follicles, glands; papillary layer (dermal papillae = fingerprints), reticular layer (collagen). Hypodermis — fat, insulation. Appendages: hair, nails, sebaceous glands (sebum), sweat glands — eccrine (temperature control) and apocrine (axilla, groin).

Functions: protection, temperature regulation (sweat, vasodilation), sensation, vitamin D synthesis (UV on 7-dehydrocholesterol), excretion, absorption (basis of transdermal delivery), immune defence.



Wound Healing — Timeline



Cross-section of the skin

Mnemonic: Epidermal layers deep→surface — "Brittany Spears Glitters Like Candy": Basale, Spinosum, Granulosum, Lucidum, Corneum.

Skin disorders — psoriasis, dermatitis, leprosy

Psoriasis — chronic autoimmune, T-cell mediated keratinocyte hyperproliferation; thick red plaques with silvery scales on elbows, knees, scalp; Auspitz sign (pinpoint bleeding on scale removal), Koebner phenomenon (lesions at trauma sites), nail pitting. Treated with topical steroids, calcipotriol, biologics.

Dermatitis — skin inflammation with redness, itching, swelling: atopic (eczema, allergic, children), contact (irritant/allergic — nickel, cosmetics), seborrheic (oily areas, dandruff).

Leprosy (Hansen's disease) — *Mycobacterium leprae*; attacks skin and peripheral nerves (sensation loss, deformities). **Tuberculoid (paucibacillary)** — few lesions, strong immunity; **lepromatous (multibacillary)** — many lesions, leonine facies. Treated with dapsone + rifampicin + clofazimine (MDT). Still relevant in India — know it cold.

Basic principles of wound healing

Four overlapping phases: 1. Haemostasis (minutes) — vasoconstriction, platelet plug, clot. 2. Inflammatory (1–3 days) — neutrophils then macrophages clean debris. 3. Proliferative (day 3 to ~3 weeks) — fibroblasts lay collagen III, angiogenesis, granulation tissue, re-epithelialisation. 4. Remodelling (weeks–months) — collagen III replaced by type I; scar never reaches full original strength.

Primary intention (clean closed surgical wound — thin scar) vs secondary intention (open wound healing by granulation — bigger scar). Speed it up: protein, vitamin C (collagen), zinc, good blood supply. Slow it down: infection, diabetes, old age, smoking, corticosteroids and NSAIDs.

Very short questions (2 marks)

- 1 Define homeostasis and name its three components.
- 2 What is the function of the Na^+/K^+ ATPase?
- 3 Name the five strata of the epidermis from deep to superficial.
- 4 What is the difference between hypertrophy and hyperplasia?
- 5 Give one example each of negative and positive feedback.

Short questions (5 marks)

- 1 Describe the transport mechanisms across the cell membrane with examples.
- 2 Differentiate between necrosis and apoptosis.
- 3 Explain the phases of wound healing and the factors affecting it.

Long questions (10 marks)

- 1 Describe the structure and functions of the skin with a labelled diagram. Add a note on psoriasis.
- 2 Discuss the causes, pathogenesis and morphology of cell injury, including adaptive changes.

How to approach these answers

- Very-short Q1: one-line definition (stable internal environment) + the three components; don't write a paragraph.
- Very-short Q2: state 3 Na^+ out / 2 K^+ in + one line on why it matters (membrane potential, digoxin target).
- Very-short Q3: list the five strata in order with the one-line mnemonic; lucidum only in thick skin.
- Very-short Q4: size change vs number change + one example each; one table row is enough.
- Very-short Q5: glucose/insulin for negative, childbirth oxytocin for positive; one line each.
- Short Q1: open with the passive vs active split, then list each type with one example, close with the digoxin line.

- Short Q2: open with planned vs unplanned, compare in 4–5 points (membrane, inflammation, examples), close with the caspase line.
- Short Q3: list the four phases in order with timings, then split factors into those that help and those that delay.
- Long Q1: intro, then three subheads — layers of skin, functions, psoriasis — plus the labelled skin diagram and a one-line conclusion.
- Long Q2: intro, then three subheads — causes, molecular pathogenesis, morphology (reversible, death, adaptations) — close with why this matters for pharmacy.

Unit II — Skeletal System, Joints, Bones and Joint Diseases

Divisions of the skeletal system

Adult skeleton = 206 bones. Axial (80) — skull (cranium 8 + face 14, plus 6 ossicles and 1 hyoid), vertebral column (33), ribs (12 pairs), sternum. Appendicular (126) — pectoral girdle, upper limbs, pelvic girdle, lower limbs.

Functions: support, protection (skull → brain, rib cage → heart/lungs), movement (levers for muscles), mineral storage (calcium, phosphorus), blood cell formation (red marrow), fat storage (yellow marrow).

Types of bones

Long — femur, humerus (shaft + ends); femur is the longest and strongest bone. Short — cube-shaped; carpals, tarsals. Flat — skull bones, ribs, sternum, scapula; protect organs, make blood cells. Irregular — vertebrae, facial bones. Sesamoid — in tendons; patella is the largest sesamoid.

Bones of the axial skeleton — salient features

Skull: cranium protects the brain; foramen magnum (occipital bone) lets the spinal cord pass; sutures are fibrous joints. Vertebral column: 7 cervical (atlas C1, axis C2 with dens — head rotation), 12 thoracic, 5 lumbar (bear weight), 5 fused sacral, 4 fused coccygeal; normal curves: cervical/lumbar lordosis, thoracic/sacral kyphosis; intervertebral discs (fibrocartilage, nucleus pulposus) cushion — a slipped disc is nucleus herniation. Ribs: 12 pairs — 7 true (direct to sternum), 3 false (via cartilage), 2 floating. Sternum: manubrium, body, xiphoid; site of marrow aspiration in adults.

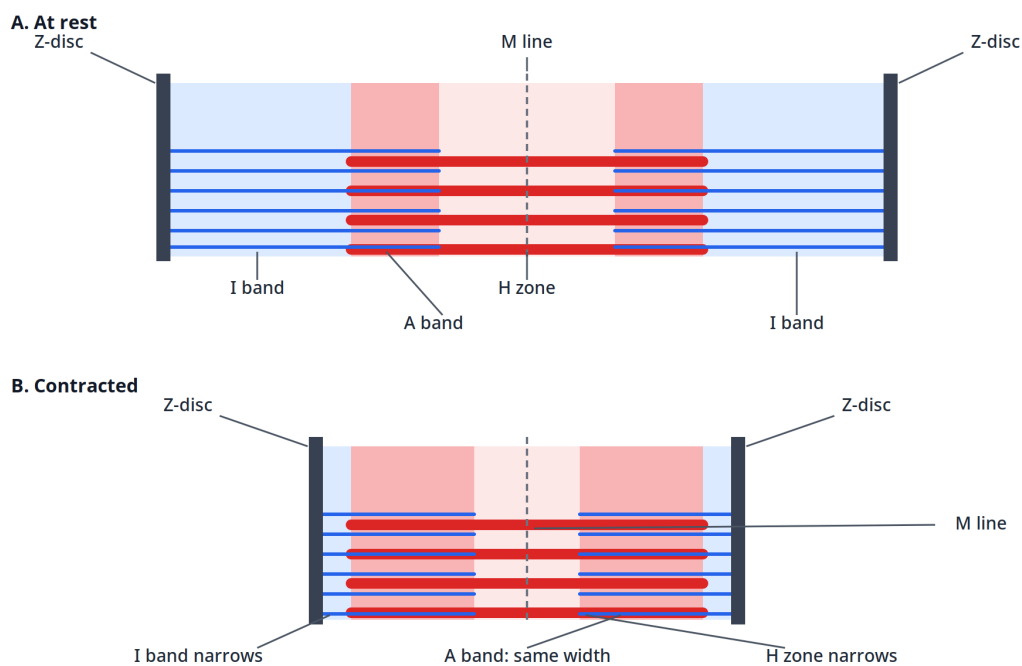
Bones of the appendicular skeleton — salient features

Pectoral girdle: clavicle (collarbone — most commonly fractured bone), scapula (glenoid cavity, acromion). Upper limb: humerus, radius (thumb side) and ulna, 8 carpals (scaphoid fractures most), 5 metacarpals, 14 phalanges. Pelvic girdle: ilium + ischium + pubis fuse at the acetabulum (hip socket); female pelvis wider with broader sciatic notch — for childbirth. Lower limb: femur (head in acetabulum), patella, tibia (medial, weight-bearing) and fibula (lateral), 7 tarsals (calcaneus = heel, largest tarsal), 5 metatarsals, 14 phalanges.

Organisation of skeletal muscle

Whole muscle in epimysium → fascicles (bundles) in perimysium → each fibre (cell) in endomysium. Tendons attach muscle to bone. Inside a fibre: myofibrils → sarcomeres (Z-disc to Z-disc, the contractile unit). Thin filaments (actin + troponin + tropomyosin) slide over thick filaments (myosin). Bands: A band (thick, dark), I band (thin only, light), H zone, M line.

Sarcomere: Rest vs Contracted (Sliding Filament Theory)



Key point: A band width is unchanged — filaments slide past each other; they do not shorten.

Legend: — Actin — thin filament — Myosin — thick filament

Sarcomere structure at rest and during contraction

Physiology of muscle contraction — sliding filament theory

- 1 Nerve impulse → Ca^{2+} released from sarcoplasmic reticulum.
- 2 Ca^{2+} binds troponin → tropomyosin moves off actin's binding sites.

- 3 Myosin heads form cross-bridges; ATP drives the power stroke — actin pulled to the centre.
- 4 New ATP detaches the head; cycle repeats; sarcomere shortens — I bands and H zone shrink, A band stays the same.
- 5 Ca^{2+} pumped back → relaxation.

ATP is needed for both contraction and relaxation — that is why rigor mortis (post-death stiffness) happens when ATP runs out. Twitch → summation → tetanus (sustained contraction). Isotonic (muscle shortens, e.g. lifting) vs isometric (length unchanged, e.g. pushing a wall).

Remember: The A band never changes length during contraction — a classic true/false trap. Calcium starts it, ATP powers and ends it.

Neuromuscular junction (NMJ)

The motor neuron-muscle fibre synapse: action potential arrives → Ca^{2+} enters the axon terminal → acetylcholine (ACh) released into the cleft → ACh binds nicotinic receptors on the motor end plate → Na^+ in → muscle action potential → contraction → acetylcholinesterase breaks down ACh, ending the signal. Pharmacy links: myasthenia gravis is treated with acetylcholinesterase inhibitors (neostigmine); tubocurarine-type blockers block nicotinic receptors in anaesthesia.

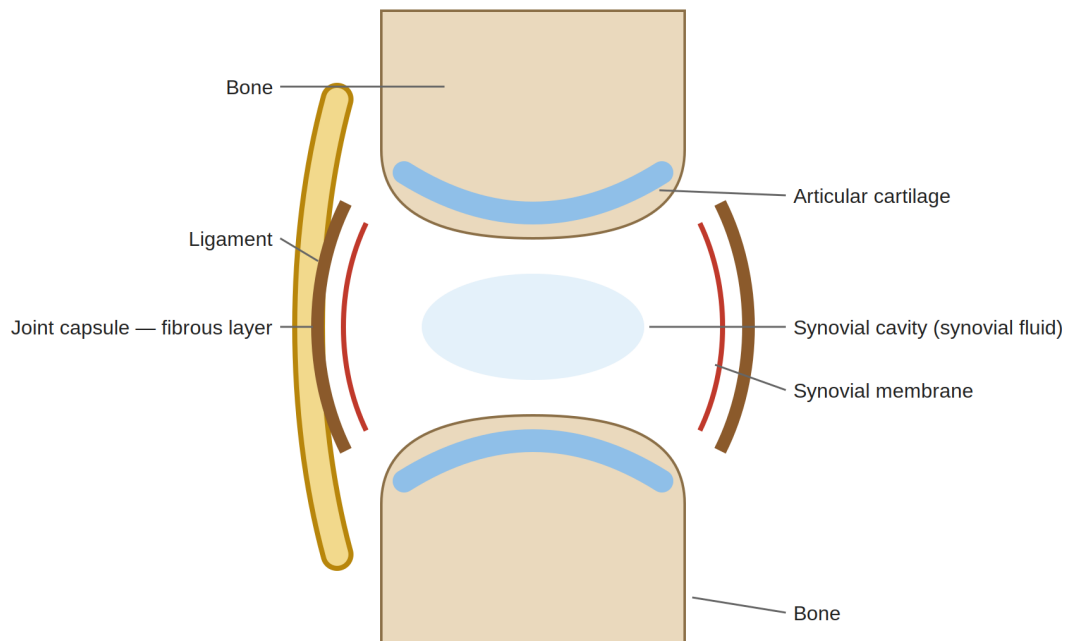
Classification of joints

Structural (by binding material): fibrous — dense connective tissue, no movement (skull sutures, tooth gomphosis). Cartilaginous — cartilage, slight movement (intervertebral discs, pubic symphysis). Synovial — fluid-filled cavity, freely movable; has articular cartilage, synovial cavity with synovial fluid, articular capsule, ligaments.

Functional: synarthrosis (immovable), amphiarthrosis (slightly movable), diarthrosis (freely movable).

Synovial subtypes (one example each — favourite exam list): ball-and-socket (shoulder, hip), hinge (elbow, knee, ankle), pivot (atlas-axis — head rotation), saddle (thumb carpometacarpal), condyloid (wrist), gliding/plane (between carpals/tarsals).

Synovial Joint — Cross Section



Most mobile joint type — e.g. knee, shoulder

Structure of a synovial joint

Pathophysiology of rheumatoid arthritis (RA)

Chronic autoimmune inflammation of the synovial membrane — small joints of hands and feet first, symmetrically. Trigger → synovitis → pannus (invasive granulation tissue) → cartilage and bone destruction → deformities (swan-neck, boutonnière, ulnar deviation). Markers: rheumatoid factor (RF) and anti-CCP (more specific). Hallmark: morning stiffness > 30–60 min, symmetric polyarthritis, rheumatoid nodules. Treated with NSAIDs, methotrexate (first-line DMARD), TNF- α inhibitor biologics.

RA vs osteoarthritis: RA = autoimmune, symmetric, small joints, worse with rest; OA = degenerative "wear and tear", weight-bearing joints, elderly, worse with activity.

Remember: Morning stiffness > 30 minutes points to RA; < 30 minutes points to OA. RF + anti-CCP = RA's lab signature.

Pathophysiology of osteoporosis

Reduced bone mineral density with deteriorated microarchitecture → fractures from trivial trauma. Resorption (osteoclasts) outpaces formation (osteoblasts). Main driver: oestrogen deficiency after menopause (oestrogen restrains osteoclasts); also ageing, low calcium/vitamin D, smoking, alcohol, corticosteroids, immobility. Silent until fracture — classic sites: hip (neck of femur), vertebrae (compression → kyphosis, height loss), distal radius (Colles' fracture). Diagnosis: DEXA scan; T-score ≤ -2.5 . Treated with calcium + vitamin D, bisphosphonates (alendronate — upright with water, empty stomach).

Pathophysiology of gout

Hyperuricaemia → monosodium urate crystals deposit in joints → neutrophils attack → sudden severe inflammation. Overproduction (purine-rich food — red meat, seafood, alcohol) or underexcretion (kidney disease, diuretics). Classic: podagra — agonising nocturnal pain, redness, swelling of the big toe's first metatarsophalangeal joint. Chronic: tophi (chalky deposits in ears, elbows) and urate kidney stones. Acute: NSAIDs, colchicine; long-term: allopurinol (xanthine oxidase inhibitor). Avoid thiazides — they raise uric acid.

Very short questions (2 marks)

- 1 How many bones are in the axial and appendicular skeleton?
- 2 Name the parts of a sarcomere and state which band stays constant during contraction.
- 3 List the steps of transmission at the neuromuscular junction.
- 4 What are the markers of rheumatoid arthritis?
- 5 Which joint is classically affected in gout, and what crystal causes it?

Short questions (5 marks)

- 1 Explain the sliding filament theory of muscle contraction.
- 2 Classify joints structurally and functionally with one example each.
- 3 Differentiate rheumatoid arthritis from osteoarthritis.

Long questions (10 marks)

- 1 Describe the physiology of skeletal muscle contraction with a labelled diagram of the sarcomere, and explain the events at the neuromuscular junction.
- 2 Discuss the pathophysiology of any two of the following: rheumatoid arthritis, osteoporosis, gout.

How to approach these answers

- Very-short Q1: 80 axial + 126 appendicular = 206; one line, no paragraph.
- Very-short Q2: Z-disc to Z-disc, thin/thick filaments, then the A-band line — that last line is the mark.

- Very-short Q3: Ca^{2+} in $\rightarrow$ ACh out $\rightarrow$ nicotinic receptors $\rightarrow$ acetylcholinesterase clears; keep it to four steps.
- Very-short Q4: RF and anti-CCP, one line each on what they are.
- Very-short Q5: big toe MTP joint + monosodium urate; mention podagra by name.
- Short Q1: open with Ca^{2+} release, then troponin-tropomyosin-cross-bridge-power stroke in order, close with the ATP/rigor mortis line.
- Short Q2: open with the structural-vs-functional split, give the three structural types and three functional terms with one example each.
- Short Q3: compare in 5 points — cause, joints, symmetry, morning stiffness, markers — close with one line.
- Long Q1: intro, three subheads — sarcomere structure, sliding filament steps, NMJ events — plus the labelled sarcomere diagram, one-line conclusion.
- Long Q2: for each disease use cause $\rightarrow$ mechanism $\rightarrow$ clinical features $\rightarrow$ one pharmacy link; keep the two diseases balanced.

Unit III — Body Fluids, Blood, Lymphatic System, Inflammation, Haematological Diseases

Body fluids — compartments

Water $\approx$ 60% of body weight (adult male; less in females/elderly, more in infants). ICF (inside cells) $\approx$ 40% of body weight, K^+ -rich. ECF (outside cells) $\approx$ 20%, Na^+ -rich — split into plasma ($\sim$ 4–5%) and interstitial fluid ($\sim$ 15%), plus small volumes (lymph, CSF). Fluid shifts by osmosis and filtration (Starling forces); oedema = excess interstitial fluid from leaky capillaries, low plasma protein, or blocked lymph drainage.

Composition and functions of blood

$\sim$ 5 litres in an adult ($\sim$ 7–8% of body weight). Plasma ($\sim$ 55%) — 90% water; albumin (osmotic pressure, carries drugs — hypoalbuminaemia changes drug distribution), globulins (antibodies, transport), fibrinogen (clotting); plus nutrients, hormones, gases, waste, electrolytes. Formed elements ($\sim$ 45%) — RBCs 4.5–5.5 million/ μL (biconcave, no nucleus, haemoglobin; 120-day life; destroyed in spleen), WBCs 4,000–11,000/ μL (neutrophils — bacterial phagocytosis; eosinophils — parasites/allergy; basophils — histamine/heparin; lymphocytes — B cells make antibodies, T cells kill; monocytes $\rightarrow$ macrophages), platelets 1.5–4 lakh/ μL (from megakaryocytes; platelet plug).

Functions: transport (O_2 , CO_2 , nutrients, hormones, waste, drugs), regulation (pH, temperature, fluid balance), protection (clotting, immunity). Haematocrit: $\sim$ 40–54% men, $\sim$ 36–46% women;

risers in dehydration, falls in anaemia.

Haemopoiesis and haemoglobin formation

Blood cells form in red bone marrow (pelvis, vertebrae, sternum, ribs, skull) from haematopoietic stem cells. Erythropoiesis is driven by erythropoietin (EPO) from the kidney in response to low oxygen; needs iron, vitamin B₁₂, folic acid; erythroblast → reticulocyte → mature RBC (loses nucleus). Haemoglobin = 4 haem groups (each with one Fe²⁺) + 4 globin chains (2α + 2β in adult HbA). Iron absorbed in duodenum (vitamin C helps), carried by transferrin, stored as ferritin; old RBCs broken down in spleen/liver → haem becomes bilirubin (excess = jaundice), iron recycled.

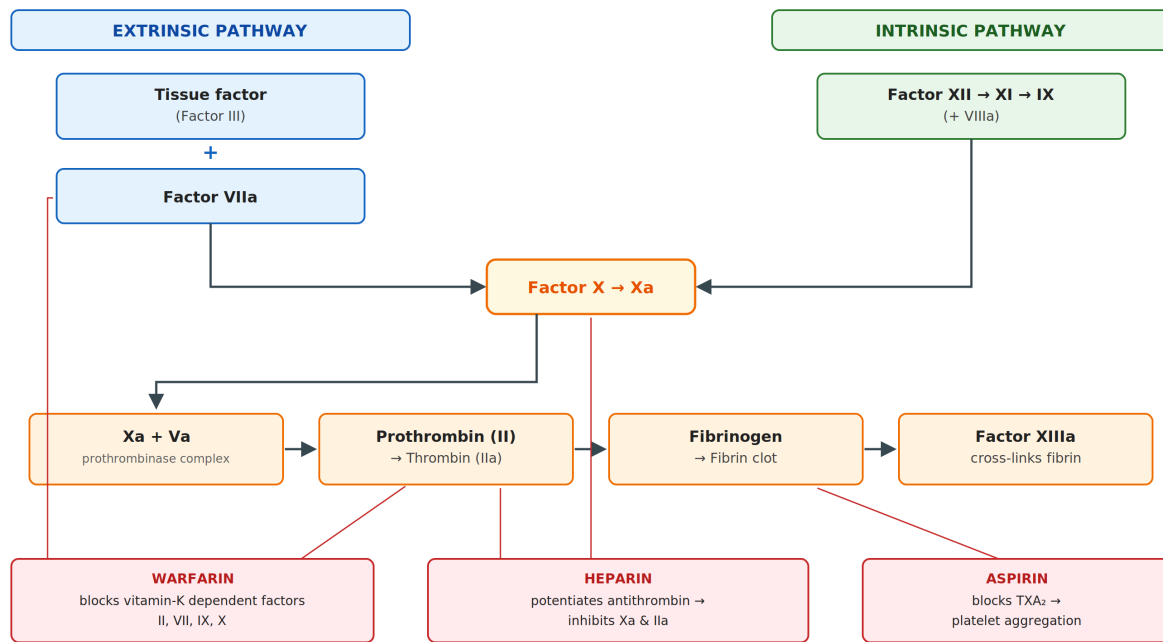
Mechanism of blood coagulation

Haemostasis in three stages: vascular spasm → platelet plug (platelets stick to collagen via von Willebrand factor, then aggregate) → coagulation (fibrin mesh stabilises the plug).

Cascade: extrinsic pathway (tissue factor/factor III from injury — fast; monitored by PT/INR, warfarin's test) and intrinsic pathway (contact with exposed collagen — slower; monitored by aPTT, heparin's test) both converge on factor X → Xa → prothrombin (II) → thrombin (IIa) → fibrinogen (I) → fibrin, cross-linked by factor XIII = stable clot. Must-name factors: I (fibrinogen), II (prothrombin), III (tissue factor), VIII (deficient in haemophilia A), IX (haemophilia B), calcium (IV), and vitamin K (needed to make II, VII, IX, X — why warfarin, a vitamin K antagonist, is an anticoagulant).

Fibrinolysis: plasmin dissolves the clot (streptokinase, alteplase activate plasminogen — used in MI and stroke).

Blood Clotting Cascade & Drug Action Sites



Red callouts mark drug action sites — a favourite exam focus.

The clotting cascade and where drugs act

Remember: One cascade explains three drug classes — heparin (intrinsic/aPTT), warfarin (extrinsic/PT-INR, vitamin K), aspirin/clopidogrel (antiplatelet — they hit the plug stage, not the cascade).

Blood grouping, Rh factor and transfusion

ABO (Landsteiner): RBCs carry A and/or B antigens; plasma carries antibodies against the antigens you lack. A has anti-B, B has anti-A, AB has neither (universal recipient), O has both antibodies (universal donor for RBCs). Rh: Rh-positive RBCs carry D antigen; Rh-negative mothers make anti-D after exposure → haemolytic disease of the newborn in a later Rh-positive pregnancy — prevented with anti-D immunoglobulin (RhoGAM). Always cross-match: ABO mismatch causes acute haemolytic reaction (agglutination, haemolysis, renal damage, shock).

Lymphatic system

Organs: lymph nodes (filter lymph, swell in infection), spleen (filters blood, destroys old RBCs), thymus (T-cell maturation, active in childhood), tonsils and Peyer's patches (MALT). Vessels: blind capillaries → through nodes → back to blood at the subclavian veins (thoracic duct left, right lymphatic duct right); one-way valves. Lymph formation: unreabsorbed capillary filtrate (~3 L/day) enters lymph capillaries. Functions: return tissue fluid to blood, absorb dietary fats (lacteals in intestine), immune surveillance.

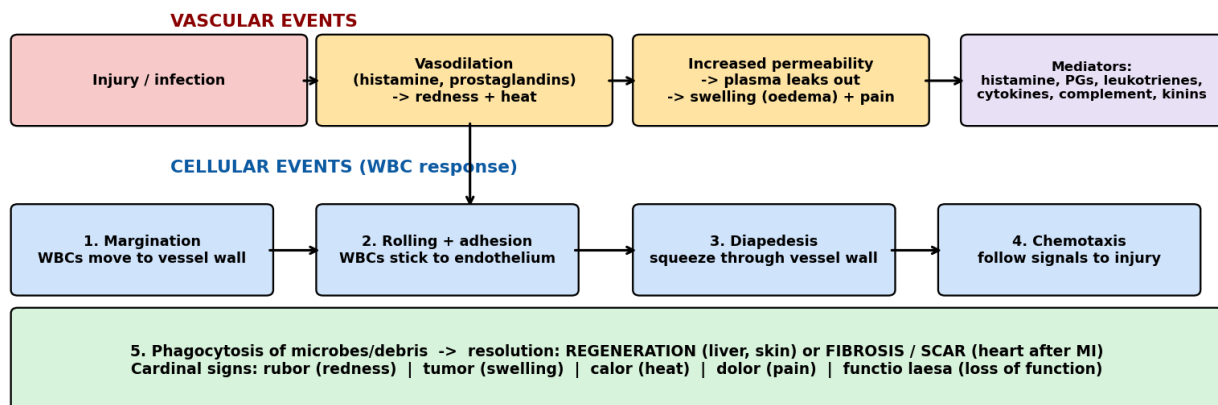
Inflammation — introduction and classification

The vascular + cellular response to injury or infection. Cardinal signs: rubor (redness), tumor (swelling), calor (heat), dolor (pain), functio laesa (loss of function). Acute (seconds-days; neutrophils; a cut, bacterial infection) vs chronic (weeks-years; macrophages + lymphocytes; TB, rheumatoid arthritis, granulomas).

Pathophysiology of inflammation

Vascular events: vasodilation (histamine, prostaglandins → redness, heat) → increased permeability → plasma leaks out (oedema, pain). Cellular events: margination → rolling → adhesion → diapedesis (WBCs squeeze through the wall) → chemotaxis (follow signals to the injury) → phagocytosis. Mediators (must-memorise list): histamine (mast cells, immediate vasodilation), prostaglandins (pain, fever — blocked by NSAIDs), leukotrienes (bronchoconstriction in asthma), cytokines (IL-1, IL-6, TNF- α — fever), complement (C3a, C5a — chemotaxis), kinins (bradykinin — pain), platelet-activating factor. Repair: regeneration (same tissue regrows — liver, skin) vs fibrosis/scarring (heart muscle after MI).

Inflammation: vascular and cellular events in order



Acute = neutrophils, seconds-days (cut, bacterial infection) | Chronic = macrophages + lymphocytes, weeks-years (TB, rheumatoid arthritis)

Pharmacy link: NSAIDs block prostaglandin synthesis (COX); corticosteroids block earlier steps (phospholipase A₂)

Vascular and cellular events of inflammation

Remember: NSAIDs block prostaglandin synthesis (COX); corticosteroids block earlier steps (phospholipase A₂) — same inflammation, different choke points.

Iron deficiency anaemia

Commonest anaemia worldwide. Cause: blood loss (menstruation, hookworm, piles), poor intake, pregnancy. Iron needed for haem → microcytic hypochromic RBCs (small, pale). Fatigue, pallor, koilonychia (spoon nails), pica (clay craving), angular cheilitis. Treated with ferrous sulphate/fumarate — blackens stools; best absorbed empty stomach with vitamin C.

Megaloblastic anaemia (vitamin B₁₂ and folic acid)

Both vitamins needed for DNA synthesis → precursors can't divide → large, immature, macrocytic RBCs. B₁₂ deficiency (pernicious anaemia — no intrinsic factor; strict vegetarian diets) also causes neurological symptoms (tingling, numbness — myelin damage); folate deficiency does not. Never give folate alone in B₁₂ deficiency — it masks the anaemia while nerve damage progresses silently.

Sickle cell anaemia

Autosomal recessive; one amino acid swap in β -globin (glutamate → valine) makes HbS, which polymerises in low oxygen → RBCs sickle. Sickled cells block small vessels (vaso-occlusive painful crises) and haemolyse early. Common in central Indian tribal belts (MP, Chhattisgarh, Maharashtra). Managed with hydroxyurea, folic acid, vaccination, crisis care.

Thalassemia

Reduced globin chain synthesis: β -thalassemia (β chains low → excess α chains damage RBCs) and α -thalassemia. Microcytic hypochromic anaemia, but iron studies are normal/high (unlike iron deficiency). Severe β -thalassemia major (Cooley's anaemia) needs lifelong transfusion → iron overload → chelation (deferoxamine/deferiprone).

Hereditary and acquired haemolytic anaemias

Hereditary spherocytosis — defective membrane proteins (spectrin/ankyrin) → fragile spherical RBCs destroyed in the spleen; treated by splenectomy. Acquired — autoimmune haemolytic anaemia and drug-induced haemolysis.

Haemophilia

X-linked recessive (males affected, females carriers). Haemophilia A — factor VIII deficiency (~85%); Haemophilia B (Christmas disease) — factor IX deficiency. Prolonged bleeding, haemarthrosis (bleeding into joints), easy bruising; PT normal, aPTT prolonged. Treated with factor concentrates; avoid NSAIDs and IM injections.

Remember: The three anaemia nutrients are iron, B₁₂ and folate — microcytic = iron problem; macrocytic = B₁₂/folate problem; B₁₂ alone adds nerve signs.

Very short questions (2 marks)

- 1 What is the difference between plasma and serum?
- 2 Which lab tests monitor heparin and warfarin therapy?
- 3 Why is blood group O the universal donor?
- 4 Name the five cardinal signs of inflammation.
- 5 Which clotting factor is deficient in haemophilia A and B?

Short questions (5 marks)

- 1 Describe the mechanism of blood coagulation with a flowchart.
- 2 Explain the vascular and cellular events of inflammation.
- 3 Compare iron deficiency anaemia with megaloblastic anaemia.

Long questions (10 marks)

- 1 Describe the composition and functions of blood. Add a note on haemoglobin formation and haemopoiesis.
- 2 Discuss the pathophysiology of any two haematological diseases from the syllabus, with their clinical features.

How to approach these answers

- Very-short Q1: plasma has fibrinogen/clotting factors, serum doesn't — one line, that's the mark.
- Very-short Q2: aPTT for heparin (intrinsic), PT/INR for warfarin (extrinsic) — name the pathway with each.
- Very-short Q3: O RBCs carry no A/B antigens, so no antibodies attack them — one line.
- Very-short Q4: rubor, tumor, calor, dolor, functio laesa — with the English meaning of each.
- Very-short Q5: VIII in A, IX in B; add "Christmas disease" for B.
- Short Q1: draw the cascade flowchart — extrinsic → X, intrinsic → X, common → thrombin → fibrin — then mark where heparin/warfarin act.
- Short Q2: vascular events first (vasodilation → permeability), then the cellular sequence in order ending at phagocytosis, close with the mediator list.
- Short Q3: compare in 5 points — cause, RBC size, one unique sign each, lab finding, treatment point.
- Long Q1: intro, three subheads — plasma, formed elements + functions, haemopoiesis/haemoglobin — close with one pharmacy link (albumin and drug binding).
- Long Q2: for each disease use cause → mechanism → clinical features → treatment; pick two with contrasting mechanisms (e.g. iron deficiency vs sickle cell).

Unit IV — Peripheral Nervous System and Special Senses

Divisions of the peripheral nervous system

PNS = everything outside brain and spinal cord (nerves + ganglia). Somatic (voluntary) — one neuron from CNS to skeletal muscle; transmitter is acetylcholine at the NMJ. Autonomic (involuntary) — two-neuron chain (preganglionic → ganglion → postganglionic) to smooth muscle, cardiac muscle, glands; split into sympathetic and parasympathetic. A third enteric division runs the gut.

Sympathetic vs parasympathetic nervous system

Feature	Sympathetic ("fight or flight")	Parasympathetic ("rest and digest")
Origin	Thoracolumbar (T1–L2)	Craniosacral (III, VII, IX, X + S2–S4)
Ganglia	Near spinal cord (paravertebral chain)	Near/inside the target organ
Postganglionic transmitter	Noradrenaline (mostly)	Acetylcholine (muscarinic)
Heart	Rate and force up	Rate down
Pupil	Dilates (mydriasis)	Constricts (miosis)
Bronchi	Dilate	Constrict
Gut	Motility down, sphincters contract	Motility and secretion up
Bladder	Retains urine	Voids urine
Saliva	Thick, scanty	Profuse, watery

Pharmacy links: beta-blockers blunt sympathetic effects on the heart; anticholinergics (atropine) block parasympathetic effects — dry mouth, dilated pupils, urinary retention; salbutamol mimics sympathetic bronchodilation in asthma.

Remember: Preganglionic transmitter is acetylcholine (nicotinic) in BOTH divisions — the difference is only in the postganglionic transmitter. A classic trap.

Spinal nerves — origin and functions

31 pairs: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal. Each forms from a dorsal (sensory) root + ventral (motor) root joining at the intervertebral foramen. Carry sensation in from skin/muscles/joints and motor commands out to skeletal muscle. A dermatome = skin area supplied by one spinal nerve — used to locate cord lesions (shingles follows a dermatome). Key plexuses: cervical (phrenic nerve → diaphragm), brachial (upper limb — Erb's palsy), lumbar (femoral nerve), sacral (sciatic nerve — largest nerve in the body; its compression = sciatica).

Cranial nerves — origin and functions

12 pairs from the brain (mostly brainstem). Names in order — "Oh, Oh, Oh, To Touch And Feel Very Green Vegetables, AH!": I Olfactory (smell), II Optic (vision), III Oculomotor, IV Trochlear, VI Abducens (eye movements; III also constricts pupil, lifts eyelid), V Trigeminal (face sensation + chewing muscles), VII Facial (facial expression, taste anterior tongue, salivary glands), VIII Vestibulocochlear (hearing + balance), IX Glossopharyngeal (taste posterior tongue, swallowing, parotid), X Vagus (the wanderer — parasympathetic to heart, lungs, most of gut), XI Accessory (trapezius, sternocleidomastoid — shrug, head turn), XII Hypoglossal (tongue movements).

Structure and functions of the eye

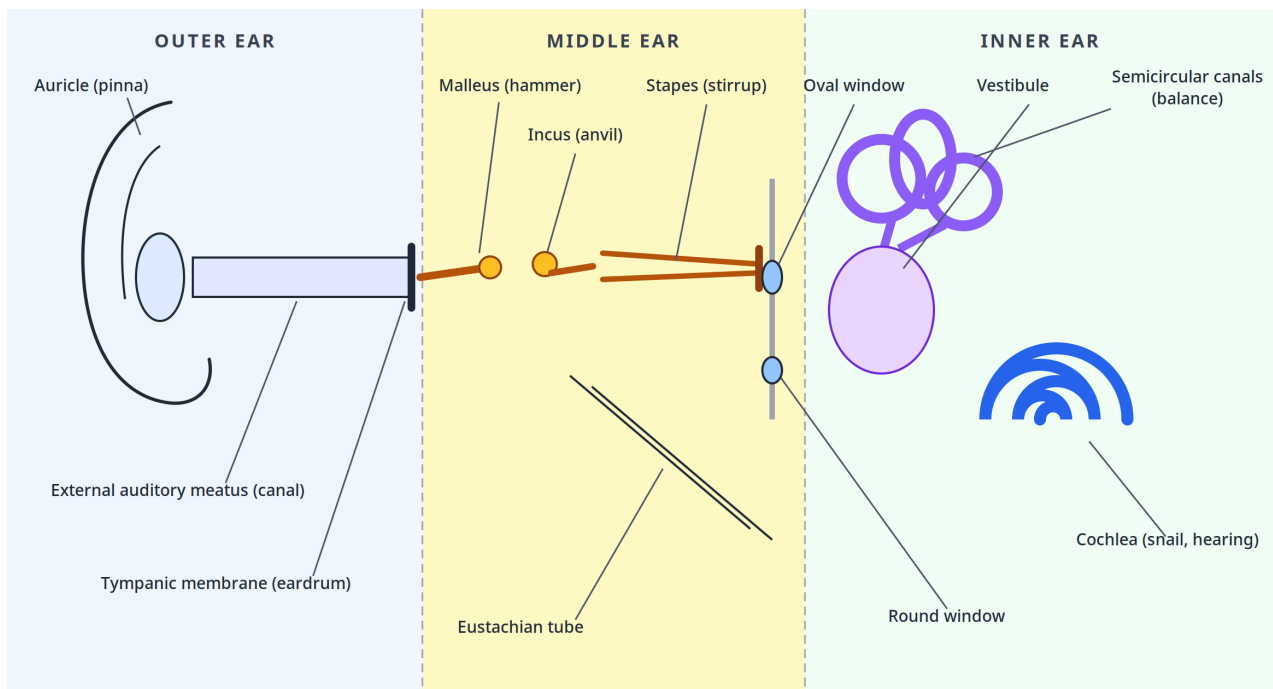
Three coats: sclera (outer, white, protective; continuous with cornea) → choroid (middle, vascular; continuous with ciliary body and iris — controls pupil size) → retina (inner, photoreceptors). Front to back: cornea (most focusing power) → anterior chamber with aqueous humor (drains via canal of Schlemm — blocked in glaucoma) → pupil → lens (fine focusing, accommodation) → vitreous humor (gel) → retina. Rods — dim light, black-and-white, need vitamin A (deficiency = night blindness); cones — colour, sharp vision, packed in the fovea (centre of macula). Optic disc (blind spot) — no photoreceptors, optic nerve leaves here. Visual pathway: retina → optic nerve → optic chiasma (nasal fibres cross) → optic tract → lateral geniculate body → visual cortex (occipital lobe).

Structure and functions of the ear

External ear: pinna (collects sound) → external auditory canal (cerumen/wax) → tympanic membrane (eardrum). Middle ear: three ossicles — malleus (hammer) → incus (anvil) → stapes (stirrup) — amplify sound to the inner ear; Eustachian (auditory) tube connects to the throat and equalises pressure (why ears "pop"; why colds cause middle-ear infections in children). Inner ear: cochlea (hair cells convert sound to impulses — sensorineural hearing), vestibule (utricle, saccule — static balance), semicircular canals (rotational balance); cranial nerve VIII carries both signals.

Hearing loss: conductive (external/middle ear — wax, otitis media; often treatable) vs sensorineural (cochlea/nerve — ageing, noise, ototoxic drugs like aminoglycosides and furosemide).

Human Ear — Three Divisions



Anatomy of the ear

Remember: Ossicle order is malleus → incus → stapes (hammer, anvil, stirrup) — and ototoxic drugs (aminoglycosides, furosemide) cause the permanent, sensorineural kind of hearing loss.

Structure and functions of the nose and tongue

Nose (olfaction): olfactory receptors in the olfactory epithelium high in the nasal cavity; smells dissolve in mucus → olfactory nerve (I) → olfactory bulb → brain. The only sense that reaches the cortex without passing through the thalamus — why smells trigger memory. Also warms, moistens, filters air.

Tongue (gustation): taste buds on papillae (fungiform, circumvallate, foliate); five tastes — sweet, salty, sour, bitter, umami. Facial nerve (VII) carries anterior two-thirds, glossopharyngeal (IX) the posterior third. Most of "taste" is actually smell — food is tasteless with a blocked nose.

Glaucoma

Raised intraocular pressure (aqueous humor can't drain) damages the optic nerve → gradual peripheral vision loss ("tunnel vision"); painless, so caught late. Open-angle (chronic, commonest) vs angle-closure (acute, painful emergency). Treated with timolol eye drops (beta-blocker), pilocarpine, latanoprost. Note: systemic anticholinergics can precipitate angle closure.

Cataract and myopia

Cataract — clouding of the lens (ageing, diabetes, UV, steroids) → painless blurring, glare. No drug reverses it — treated surgically (lens replacement). Myopia (near-sightedness) — eyeball too long or lens too powerful; distant rays focus in front of the retina; corrected with concave lenses. Contrast: hypermetropia (focus behind retina — convex lenses), presbyopia (ageing lens loses accommodation).

Otitis externa, otitis media and vertigo

Otitis externa — outer canal infection ("swimmer's ear"); pain when the pinna is pulled. Otitis media — middle-ear infection via the Eustachian tube, common in children; ear pain, fever, possible eardrum perforation. Vertigo — false sense of spinning from vestibular disturbance: BPPV (loose otolith crystals — brief episodes on head movement), Ménière's disease (endolymph buildup — vertigo + tinnitus + hearing loss). Distinguish from dizziness/light-headedness (often cardiovascular).

Anosmia

Loss of smell — causes: nasal polyps, sinusitis, head injury, viral infections, zinc deficiency. Matters in pharmacy because it kills appetite and appears as a side effect of some intranasal and systemic drugs.

Very short questions (2 marks)

- 1 Name the postganglionic neurotransmitters of the sympathetic and parasympathetic systems.
- 2 How many pairs of spinal nerves are there? Name the largest nerve in the body.
- 3 List the 12 cranial nerves in order.
- 4 What is the function of rods and cones?
- 5 What is the difference between conductive and sensorineural hearing loss?

Short questions (5 marks)

- 1 Compare the sympathetic and parasympathetic nervous systems with a table.
- 2 Describe the structure of the eye with its three coats.
- 3 Explain the pathophysiology of glaucoma and cataract.

Long questions (10 marks)

- 1 Describe the structure and functions of the ear with a labelled diagram. Add a note on otitis media and vertigo.
- 2 Classify the peripheral nervous system and describe the structure and functions of the sympathetic and parasympathetic divisions, with pharmacy examples.

How to approach these answers

- Very-short Q1: noradrenaline (sympathetic, mostly) vs acetylcholine muscarinic (parasympathetic) — plus the one line that preganglionic is ACh in both.
- Very-short Q2: 31 pairs (8-12-5-5-1) + sciatic nerve; one line each.
- Very-short Q3: write all 12 in order with the mnemonic — accuracy of order is the mark.
- Very-short Q4: rods = dim light/night vision (vitamin A), cones = colour/sharp vision (fovea); two lines.
- Very-short Q5: conductive = outer/middle ear, treatable vs sensorineural = cochlea/nerve, usually permanent; one example each.
- Short Q1: open with origin + transmitter, then the table (heart, pupil, bronchi, gut, bladder), close with one pharmacy link.
- Short Q2: list the three coats with what continues into what, then front-to-back order; keep it tight.
- Short Q3: glaucoma = pressure → optic nerve damage (two types); cataract = cloudy lens (surgical fix); one pharmacy line for each.
- Long Q1: intro, three subheads — external/middle/inner ear — plus the labelled ear diagram, then otitis media and vertigo as a closing note.
- Long Q2: intro, three subheads — classification, sympathetic, parasympathetic — the comparison table plus drug examples (beta-blockers, atropine, salbutamol).

Unit V — Cardiovascular System

Anatomy of the heart

Fist-sized pump in the mediastinum, tilted left, enclosed in the pericardium (fibrous sac + serous layers with pericardial fluid). Wall: endocardium (inner) → myocardium (muscle — thickest in the left ventricle) → epicardium (outer). Chambers: right atrium → right ventricle → lungs → left atrium → left ventricle → body; right side = deoxygenated, left = oxygenated, septum separates them.

Valves: tricuspid (right AV, 3 cusps) and mitral/bicuspid (left AV, 2 cusps) — held by chordae tendineae + papillary muscles so they don't flip inside-out; pulmonary and aortic semilunar valves (3 cusps each, no chordae). Coronary circulation: right and left coronary arteries (from the aorta) feed the heart itself — blockage = MI; drains via the coronary sinus into the right atrium.

Blood circulation

Pulmonary: right ventricle → pulmonary trunk → lungs (CO₂ out, O₂ in) → pulmonary veins → left atrium. Systemic: left ventricle → aorta → tissues → venae cavae → right atrium. Portal: gut → liver via hepatic portal vein before the heart — why oral drugs undergo first-pass metabolism. The two exceptions: pulmonary arteries carry deoxygenated blood, pulmonary veins carry oxygenated blood.

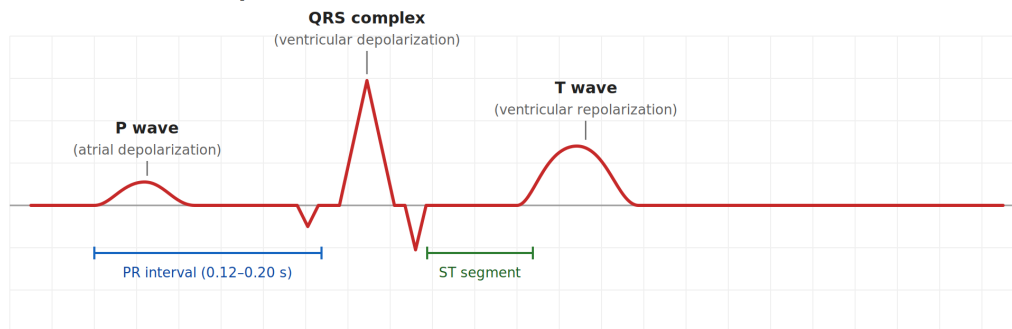
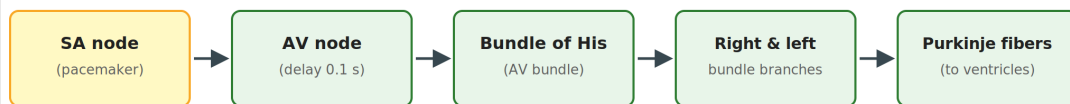
Remember: Artery = away from the heart, vein = toward the heart — oxygenation follows that rule everywhere EXCEPT the pulmonary vessels. Examiners love this trap.

Structure and functions of blood vessels

Three tunics: intima (endothelium), media (smooth muscle + elastic fibres), externa/adventitia (connective tissue). Arteries — thick walls, high pressure, carry blood away from the heart. Arterioles — main resistance vessels; control BP and local flow. Capillaries — one cell thick; site of exchange. Veins — thin walls, large lumens, valves against backflow; hold ~60% of blood (reservoirs); failed valves = varicose veins. Atherosclerosis starts with damage to the endothelium (tunica intima).

Conduction system of the heart

The heart beats on its own (autorhythmic); nerves only adjust the rate: SA node (pacemaker, right atrium; intrinsic ~100/min, vagal tone slows to ~72) → impulse across atria → AV node (delays ~0.1 s so ventricles fill) → bundle of His → right and left bundle branches → Purkinje fibres → ventricles contract apex-up. ANS regulation: sympathetic (noradrenaline, β_1) → faster + stronger; parasympathetic (vagus, acetylcholine) → slower. Pharmacy: beta-blockers slow the heart; atropine blocks vagal slowing; digoxin raises vagal tone and contractility.

A. Normal ECG complex**B. Cardiac conduction path**

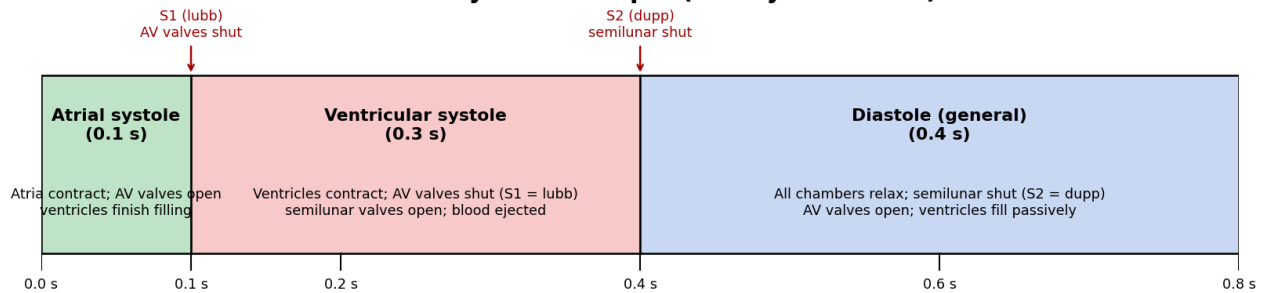
Impulse: SA node fires → atria contract → AV nodal delay → ventricles contract.

Conduction system of the heart and the ECG

Cardiac cycle

One cycle ≈ 0.8 s at 72 bpm: atrial systole (0.1 s — atria contract, AV valves open) → ventricular systole (0.3 s — AV valves shut = S₁ "lubb"; semilunar valves open; blood ejected) → diastole (0.4 s — semilunar shut = S₂ "dupp"; AV valves open; ventricles fill $\sim 70\%$ passively + $\sim 30\%$ atrial kick). Murmurs = abnormal turbulent-flow sounds (valve stenosis or regurgitation).

Cardiac cycle at 72 bpm (one cycle = 0.8 s)



Key facts: S1 = start of systole | S2 = start of diastole | Murmur = abnormal (turbulent flow)

Cardiac output = heart rate x stroke volume ~ 72 bpm x 70 mL ~ 5 L/min at rest

Stroke volume rises with preload (Frank-Starling law), falls with afterload, rises with contractility

Ventricular filling: ~70% passive in diastole + ~30% atrial kick

Phases of the cardiac cycle

Cardiac output

CO = heart rate × stroke volume ≈ 72 × 70 mL ≈ 5 L/min at rest — the whole blood volume every minute. Stroke volume depends on: preload (venous return — more stretch → stronger contraction, the Frank-Starling law), afterload (resistance pumped against — high BP cuts stroke volume), contractility (sympathetic, digoxin raise it). Ejection fraction (~55–70%) = SV ÷ end-diastolic volume — the key number in heart failure. Pharmacy: diuretics cut preload, vasodilators cut afterload, digoxin raises contractility — the three levers of heart-failure treatment.

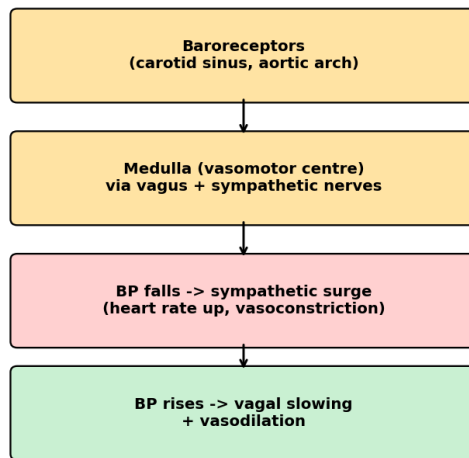
Regulation of blood pressure

BP = cardiac output × peripheral resistance; normal ~120/80 mmHg. Short-term (seconds-minutes): baroreceptors (carotid sinus, aortic arch) → medulla via vagus/glossopharyngeal → BP rises → vagal slowing + vasodilation; BP falls → sympathetic surge. Chemoreceptors sense low O₂/high CO₂/low pH. Long-term (hours-days) — RAAS: low BP/renal flow → kidney releases renin → angiotensinogen → angiotensin I → ACE (lungs) → angiotensin II → vasoconstriction + aldosterone (Na⁺/water reabsorbed, K⁺ lost) → volume and BP rise. Also ADH (water reabsorption) and ANP (opposes RAAS, released on atrial stretch). Pharmacy: ACE inhibitors (-prils), ARBs (-sartans), diuretics, beta-blockers each hit one of these steps.

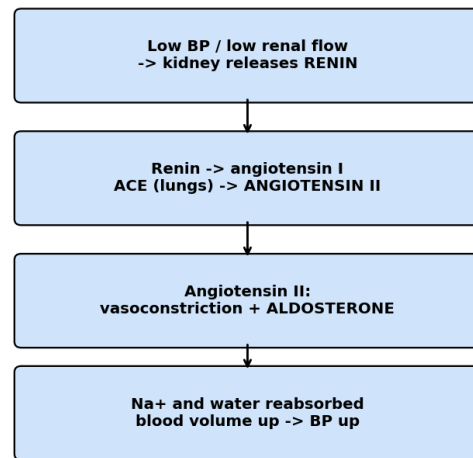
Regulation of blood pressure: short-term and long-term control

Chemoreceptors sense low O₂ / high CO₂ / low pH

SHORT-TERM (seconds-minutes)



LONG-TERM (hours-days): RAAS



Blood pressure = cardiac output x peripheral resistance | Also: ADH retains water; ANP (atria) opposes RAAS

Short-term and long-term regulation of blood pressure

Pulse and electrocardiogram (ECG)

Pulse — pressure wave felt where an artery crosses bone (radial artery standard); note rate, rhythm, volume (60–100/min normal); apex–radial pulse deficit suggests atrial fibrillation. ECG: P wave — atrial depolarisation; QRS complex — ventricular depolarisation (big spike; wide QRS = bundle-branch block); T wave — ventricular repolarisation. PR interval — AV nodal delay (prolonged = heart block); ST segment — elevation = acute MI, depression = ischaemia. Normal sinus rhythm: every P followed by QRS, regular, 60–100/min.

Remember: P = atria depolarise, QRS = ventricles depolarise, T = ventricles recover. ST elevation is the MI alarm on the ECG.

Pathophysiology of hypertension

Sustained BP $\geq$ 140/90 mmHg. Primary (essential) — ~90–95%; risk factors: age, family history, obesity, high salt, smoking, alcohol, stress, inactivity. Secondary — ~5–10%; kidney disease, aldosteronism, Cushing's, pheochromocytoma, coarctation of aorta, drugs (NSAIDs, steroids, oral contraceptives). Mechanism: raised cardiac output and/or peripheral resistance (sympathetic overdrive, RAAS overactivity, salt/water retention). The "silent killer" — no symptoms until target-organ damage: stroke, MI, heart failure, chronic kidney disease, retinopathy. Treatment: lifestyle first, then thiazides, ACE inhibitors/ARBs, calcium-channel blockers, beta-blockers matched to mechanism.

Pathophysiology of cardiac arrhythmias

Any abnormality of rate, rhythm, or conduction. Sinus tachycardia (>100 — fever, anxiety) / bradycardia (<60 — athletes, beta-blockers). Atrial fibrillation — chaotic atria, irregularly irregular pulse, lost atrial kick; danger is atrial clot $\rightarrow$ stroke $\rightarrow$ needs anticoagulation (warfarin/DOACs). Atrial flutter — sawtooth pattern. Ventricular tachycardia/fibrillation — VF = quivering ventricles, no cardiac output = cardiac arrest $\rightarrow$ immediate defibrillation. Heart blocks — first-degree (long PR), second-degree (dropped beats), third-degree/complete (atria and ventricles independent — needs pacemaker). Extrasystoles — premature beats, often benign. Pharmacy: antiarrhythmics (amiodarone, lidocaine, verapamil, adenosine) act on ion channels and conduction — learn the conduction system first.

Remember: AF risks stroke (anticoagulate); VF means no output (defibrillate); complete heart block needs a pacemaker. Three emergencies, three actions.

Pathophysiology of congestive heart failure (CHF)

The heart fails as a pump — can't meet the body's needs. Left-sided: blood backs into the lungs — dyspnoea, orthopnoea (breathless lying flat), paroxysmal nocturnal dyspnoea, pulmonary oedema (crackles), fatigue; causes: hypertension, MI, valve disease. Right-sided: blood backs into systemic veins — pedal oedema, raised JVP, enlarged tender liver, ascites; often follows left-sided failure or lung disease (cor pulmonale). Vicious cycle: initial insult $\rightarrow$ sympathetic + RAAS compensation (retain salt/water, vasoconstrict) + ventricular remodelling $\rightarrow$ helps briefly, worsens long-term. Systolic (low ejection fraction — weak squeeze) vs diastolic (preserved EF — stiff ventricle, can't fill). Treatment unloads the heart: diuretics (preload/oedema), ACE inhibitors/ARBs + beta-blockers (block harmful compensation — prolong life), digoxin (contractility).

Pathophysiology of ischemic heart disease

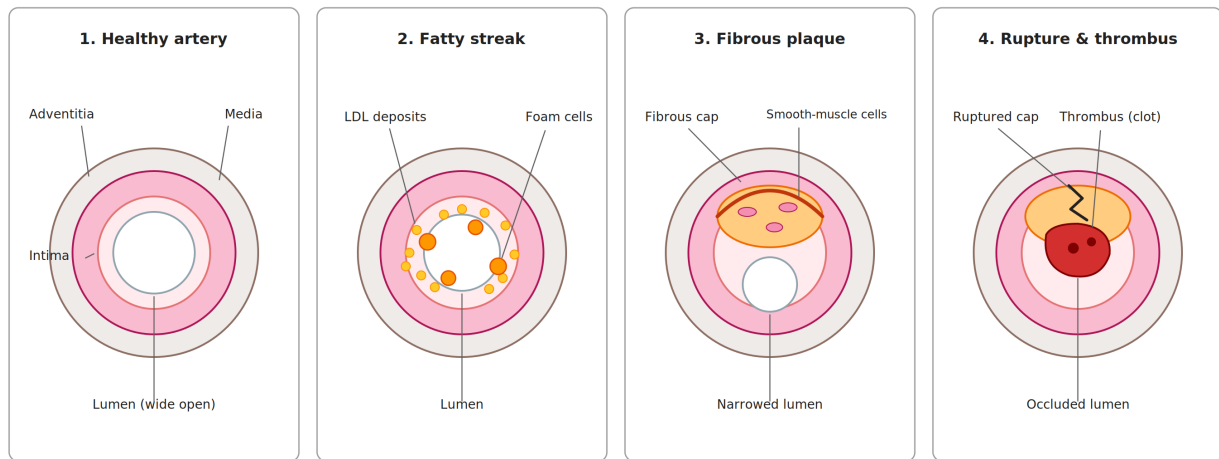
Atherosclerosis — the root process: 1. endothelial injury (hypertension, smoking, high LDL, diabetes) lets LDL into the intima. 2. Oxidised LDL $\rightarrow$ monocytes become macrophages $\rightarrow$ gorge on lipid $\rightarrow$ foam cells $\rightarrow$ fatty streak. 3. Smooth-muscle proliferation + collagen $\rightarrow$ fibrous plaque with lipid core. 4. Plaque rupture $\rightarrow$ thrombus $\rightarrow$ occlusion (stable plaques narrow = stable angina; ruptured plaques = MI).

Angina — ischaemic chest pain, relieved by rest/nitroglycerin: stable (predictable, exertional — fixed narrowing), unstable (new/severe/at rest — plaque disruption, pre-infarction emergency), Prinzmetal/variant (coronary spasm, at rest).

Myocardial infarction — prolonged ischaemia kills heart muscle: crushing retrosternal pain radiating to left arm/jaw, sweating, nausea; not relieved by rest or nitrates. Diagnosis: ECG (ST elevation in STEMI) + troponin I/T (most specific, rises in 3-6 h) and CK-MB. Complications: arrhythmias (commonest early death), heart failure, cardiogenic shock. Treatment: aspirin + clopidogrel, thrombolytics (streptokinase) or primary angioplasty — "time is muscle."

Coronary artery disease (CAD) — umbrella term for atherosclerotic coronary disease (angina, MI, sudden death); modifiable risks — smoking, hypertension, diabetes, dyslipidaemia, obesity, inactivity — are the pharmacist's counselling points.

Atherosclerosis: Sequence of Plaque Formation



LDL = low-density lipoprotein; foam cells = lipid-laden macrophages.

Sequence of atherosclerosis

Very short questions (2 marks)

- Trace the path of blood through the heart in order.
- What is the correct order of the heart's conduction system?
- What does each wave of a normal ECG represent?
- Define cardiac output and give its normal value.
- What is the difference between stable and unstable angina?

Short questions (5 marks)

- Describe the cardiac cycle with the heart sounds.
- Explain the short-term and long-term regulation of blood pressure.
- Differentiate left-sided from right-sided heart failure.

Long questions (10 marks)

- Describe the anatomy of the heart including chambers, valves and coronary circulation, with a labelled diagram. Add a note on the conduction system.
- Discuss the pathophysiology of atherosclerosis and explain how it leads to angina and myocardial infarction.

How to approach these answers

- Very-short Q1: venae cavae → right atrium → tricuspid → right ventricle → pulmonary valve → lungs → pulmonary veins → left atrium → mitral → left ventricle → aortic valve → aorta; keep it a single chain.
- Very-short Q2: SA node → AV node (delay) → bundle of His → bundle branches → Purkinje fibres; one line.
- Very-short Q3: P = atrial depolarisation, QRS = ventricular depolarisation, T = ventricular repolarisation; three short phrases.
- Very-short Q4: $HR \times SV \approx 5 \text{ L/min}$; state the formula and the numbers.
- Very-short Q5: stable = exertional, relieved by rest vs unstable = rest pain, pre-infarction emergency; two lines.
- Short Q1: use the 0.8 s timeline — three phases with durations, then place S_1 and S_2 on it.
- Short Q2: baroreceptors/chemoreceptors for short-term, RAAS step-by-step for long-term, close with the $BP = CO \times PR$ formula.
- Short Q3: left = lungs (dyspnoea, pulmonary oedema) vs right = body (oedema, JVP, liver); a two-column table works best.
- Long Q1: intro, three subheads — chambers + circulation, valves, coronary + conduction — plus the labelled heart diagram and a one-line conclusion.
- Long Q2: intro, three subheads — the four-step atherosclerosis sequence, angina types, MI (features + troponin + "time is muscle") — plus the atherosclerosis diagram.