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Human Anatomy, Physiology and Pathophysiology I

BP104T — B.Pharm Semester I

Exam Success Notes

Theory-exam mastery: complete answers, the way examiners want them

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 and pathophysiology: definitions and scope

Anatomy is the study of the structure of the body and the relationship between body parts. Physiology is the study of how those structures function — the chemical and physical processes that keep the body working. Pathophysiology is the study of what goes wrong: how normal function changes during disease.

Branches of anatomy you will meet: gross anatomy (structures visible to the eye), microscopic anatomy or histology (tissues), developmental anatomy (growth from fertilised egg to adult), and surface anatomy (what can be seen or felt on the body surface).

Why this matters for a pharmacy student: every drug chapter in pharmacology assumes this base. Structure tells you where a drug acts, physiology tells you what it changes, and pathophysiology tells you what is broken in the patient. You learn the disease mechanism here, and the drug action later.

Exam tip: Open any 10-mark answer in this unit with the definition first, then build outward. Examiners award the first 2 marks almost entirely for a clean definition.

Levels of structural organisation and body systems

From smallest to largest, there are six levels:

- 1 Chemical level — atoms and molecules (water, proteins, DNA).
- 2 Cellular level — the cell, the smallest living unit of the body.
- 3 Tissue level — groups of similar cells performing one job (muscle tissue, nervous tissue).
- 4 Organ level — two or more tissues forming a structure with a specific function (heart, liver).
- 5 Organ-system level — organs working together toward a common goal (cardiovascular system).
- 6 Organismal level — all systems together: the human being.

The body has 11 organ systems: integumentary, skeletal, muscular, nervous, endocrine, cardiovascular, lymphatic/immune, respiratory, digestive, urinary, and reproductive.

Life processes

All living things share these processes — a favourite one-mark question:

- Organisation — orderly arrangement of parts
- Metabolism — catabolism (breakdown) + anabolism (build-up)
- Responsiveness — reacting to changes (irritability)
- Movement — of the whole body or of substances within it
- Growth — increase in size
- Differentiation — unspecialised cells becoming specialised
- Reproduction — producing new cells or new individuals

Homeostasis and feedback systems

Homeostasis is the maintenance of a stable internal environment despite outside changes — body temperature around 37 °C, blood pH 7.35–7.45, fasting blood glucose 70–110 mg/dL. Every feedback loop has three parts:

- 1 Receptor — detects the change (e.g., thermoreceptors in skin).
- 2 Control centre — usually the brain or an endocrine gland; decides the response.
- 3 Effector — a muscle or gland that carries out the response.

Negative feedback — the response reverses the change. This is how almost everything works. Examples: high blood glucose triggers insulin release, which brings glucose down; rising body temperature triggers sweating and skin vasodilation, which cools the body.

Positive feedback — the response strengthens the change. Rare, used only when a rapid, complete event is needed. Examples: oxytocin release during childbirth (contractions → more oxytocin → stronger contractions), and blood clotting (activated platelets recruit still more platelets).

Exam tip: "Explain homeostasis with a suitable example" is a standard 5-mark answer. Draw the loop as: stimulus → receptor → control centre → effector → response, then work one example (blood glucose or temperature) through every box.

Anatomical terminologies

Anatomical position: body upright, feet together, arms at the sides, palms facing forward. All directional terms assume this position — never describe directions relative to how the patient happens to be lying.

Body planes:

- Sagittal plane — divides the body into left and right parts.
- Frontal (coronal) plane — divides the body into front and back parts.

- Transverse (horizontal) plane — divides the body into upper and lower parts.

Directional terms:

- Superior (toward the head) / inferior (toward the feet)
- Anterior (ventral) (front) / posterior (dorsal) (back)
- Medial (toward the midline) / lateral (away from the midline)
- Proximal (nearer the trunk or point of attachment) / distal (farther away)
- Superficial (near the surface) / deep (away from the surface)

Body cavities: the dorsal cavity (cranial cavity holding the brain + vertebral/spinal cavity holding the spinal cord) and the ventral cavity (thoracic cavity with pleural and pericardial cavities + the abdominopelvic cavity).

Abdominopelvic regions (a 3×3 grid): right and left hypochondriac, epigastric, right and left lumbar, umbilical, right and left iliac (inguinal), hypogastric. Clinicians also use four quadrants (RUQ, LUQ, RLQ, LLQ) — for example, RLQ pain suggests appendicitis.

Structure and functions of the cell

The cell is the structural and functional unit of life. Its main parts:

- Plasma (cell) membrane — a phospholipid bilayer with embedded proteins, cholesterol, and a carbohydrate coat (glycocalyx). Described by the fluid-mosaic model. Functions: selective barrier, transport, cell recognition, communication.
- Cytoplasm — the cytosol plus all organelles.
- Nucleus — contains DNA in chromosomes; controls the cell. Has a nuclear envelope with pores and a nucleolus that makes ribosomes.
- Mitochondria — the powerhouse; aerobic respiration produces ATP. They carry their own DNA.
- Ribosomes — protein synthesis; free ribosomes make proteins for use inside the cell, ribosomes on the rough ER make proteins for export.
- Endoplasmic reticulum — rough ER folds and transports proteins; smooth ER makes lipids, detoxifies drugs, and stores calcium (in muscle it is called the sarcoplasmic reticulum).
- Golgi apparatus — modifies, packages, and ships proteins in vesicles.
- Lysosomes — digestive enzymes; break down worn-out organelles and foreign particles.
- Cytoskeleton — microtubules, microfilaments (actin), and intermediate filaments; gives shape and moves materials inside the cell.
- Centrosome/centrioles — organise cell division.

Transport across the cell membrane

Passive transport — no energy, down the concentration gradient

- Simple diffusion — small lipid-soluble molecules (O_2 , CO_2) cross the membrane directly.
- Facilitated diffusion — via channel proteins (e.g., aquaporins for water) or carrier proteins (e.g., glucose via GLUT transporters). No energy needed, but the carriers can be saturated.
- Osmosis — water moves across a semipermeable membrane toward higher solute concentration. Key pharmacy concept: isotonic (no net movement), hypotonic (cell swells), hypertonic (cell shrinks) solutions.
- Filtration — water and solutes pushed through by hydrostatic pressure (e.g., at the glomerulus of the kidney).

Active transport — needs ATP, moves against the gradient

- Primary active transport — the sodium-potassium pump (Na^+/K^+ ATPase): pumps 3 Na^+ out and 2 K^+ in. It maintains the resting membrane potential and cell volume. Digoxin acts by inhibiting this pump — remember this for pharmacology.
- Secondary active transport — uses the Na^+ gradient created by the primary pump. Symport moves two substances in the same direction (Na^+ /glucose in the intestine — this is how oral rehydration salts work); antiport moves them in opposite directions (Na^+/Ca^{2+} exchanger).

Vesicular transport — for large particles

- Endocytosis: phagocytosis (cell eating — neutrophils engulfing bacteria), pinocytosis (cell drinking), receptor-mediated endocytosis (e.g., LDL uptake).
- Exocytosis: release of vesicle contents (neurotransmitters, insulin).
- Transcytosis: transport across the cell (IgA across the gut lining).

Exam tip: A two-column comparison table of passive vs active transport with one example each is exactly what a 5-mark answer on this topic should contain. Put the Na^+/K^+ pump (3 out, 2 in) at the top of the active column.

Cell division

- Interphase (G_1 growth, S DNA replication, G_2 preparation) is where the cell spends most of its life.
- Mitosis — one cell → two identical diploid cells, used for growth and repair. Phases: prophase (chromosomes condense, spindle forms), metaphase (chromosomes line up at the equator), anaphase (sister chromatids pull apart), telophase (nuclei reform), then cytokinesis (cytoplasm splits).
- Meiosis — one cell → four haploid gametes; happens only in the gonads and reduces the chromosome number from 46 to 23.

Pharmacy link: anticancer drugs target dividing cells — antimetabolites act in S phase, vinca alkaloids block the mitotic spindle in M phase.

Cell junctions

Five types, each matched to its job:

- Tight junctions — seal neighbouring cells so nothing leaks between them (gut lining, blood-brain barrier).
- Adherens junctions — anchor cells together through actin filaments.
- Desmosomes — strong "spot welds" using intermediate filaments; found in skin and cardiac muscle, tissues under mechanical stress.
- Gap junctions — channels (connexons) letting ions and small molecules pass directly between cells; found in cardiac and smooth muscle so they contract as a unit.
- Hemidesmosomes — anchor cells to the basement membrane.

Exam tip: "Tight junctions seal, desmosomes weld, gap junctions connect" — learn the junction-to-tissue match, because examiners ask for it that way.

General principles of cell communication

Cells signal each other in the four forms named in the syllabus:

- Contact-dependent (juxtacrine) — cells touch; the signal stays on the cell surface (e.g., immune cell recognition).
- Paracrine — the signal acts on nearby cells (e.g., histamine in inflammation).
- Synaptic — the signal crosses a synapse to one specific target (neurotransmission).
- Endocrine — a hormone travels in blood to distant targets (e.g., insulin, thyroid hormone).

(Autocrine signalling — a cell signalling itself — is sometimes added as a fifth.)

Classification of tissues

There are four tissue types.

Epithelial tissue

Covers surfaces, lines cavities, forms glands. Classified by number of layers and cell shape:

- Simple squamous — one flat layer; alveoli of lungs, endothelium of blood vessels, Bowman's capsule (filtration and diffusion).
- Simple cuboidal — kidney tubules, gland ducts (secretion and absorption).
- Simple columnar — stomach-to-rectum lining; often with goblet cells that secrete mucus.

- Stratified squamous — multiple layers; keratinised (skin — protection against abrasion) vs non-keratinised (mouth, oesophagus, vagina).
- Pseudostratified columnar — looks layered but every cell touches the basement membrane; ciliated, in the trachea (sweeps mucus upward).
- Transitional — stretches; urinary bladder.

Connective tissue

The most abundant tissue; cells scattered in a matrix:

- Connective tissue proper — loose (areolar, under the skin) and dense (tendons, ligaments).
- Cartilage — hyaline (nose, trachea, articular ends of bones), elastic (ear pinna, epiglottis), fibrocartilage (intervertebral discs, menisci).
- Bone — mineralised matrix; support and protection.
- Blood — fluid matrix (plasma); transport.

Muscular tissue

- Skeletal — striated, voluntary, multinucleated.
- Cardiac — striated, branched, intercalated discs, involuntary.
- Smooth — spindle-shaped, involuntary; walls of the gut and blood vessels.

Nervous tissue

Neurons (conduct impulses) plus neuroglia (supporting cells).

Exam tip: One labelled sketch per epithelial type is a classic 5-mark question. Practise drawing them from memory — location + function under each sketch earns full marks.

Cell injury: causes, pathogenesis, morphology

Causes of cellular injury

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

Pathogenesis — how injury happens at the molecular level

- ATP depletion — the Na^+/K^+ pump fails → Na^+ and water enter the cell → cell swelling; protein synthesis drops.
- Cell membrane damage — loss of phospholipids and lipid peroxidation by free radicals; the membrane becomes leaky.

- Mitochondrial damage — opening of the mitochondrial permeability transition pore and release of cytochrome c (which triggers apoptosis); ATP production collapses.
- Cytosolic calcium overload — activates destructive enzymes (phospholipases, proteases, endonucleases).
- Ribosome damage — protein synthesis falls.
- Nuclear damage — DNA injury; if severe and irreparable, the cell dies.

Morphology of cell injury

Reversible injury — the cell recovers if the cause is removed:

- Cell swelling (hydropic degeneration) — the earliest change, from Na⁺/water entry.
- Fatty change (steatosis) — lipid droplets accumulate in liver cells (e.g., in alcoholism).

Irreversible injury → cell death, by two routes:

- Necrosis — unplanned death with inflammation. Types: coagulative (commonest; heart, kidney — tissue architecture preserved), liquefactive (brain, abscesses — tissue turns to liquid), caseous (tuberculosis — cheese-like), fat necrosis (pancreatitis), fibrinoid (blood vessel walls in hypertension/vasculitis), gangrenous (limb ischaemia with infection).
- Apoptosis — programmed, tidy death: the cell shrinks, chromatin condenses, the membrane blebs, and apoptotic bodies are eaten by neighbours — no inflammation. Controlled by caspases; triggered by cytochrome c release.

Adaptive changes — cells adjusting to stress without dying

- Atrophy — decrease in cell size (disuse atrophy of muscle in a plaster cast, denervation).
- Hypertrophy — increase in cell size (left ventricular hypertrophy in hypertension; skeletal muscle with exercise).
- Hyperplasia — increase in cell number (benign prostatic hyperplasia, endometrial hyperplasia).
- Metaplasia — one adult cell type replaced by another (a smoker's bronchial epithelium changing from columnar to squamous; Barrett's oesophagus). Reversible if the stimulus stops.
- Dysplasia — disordered, abnormal growth; considered premalignant (cervical dysplasia). Note the difference from metaplasia — dysplasia is not a benign adaptation.

Intracellular accumulations: lipids (fatty liver), proteins, glycogen, and pigments — lipofuscin (the "wear and tear" pigment), melanin, haemosiderin (iron overload).

Exam tip: Atrophy/hypertrophy change size, hyperplasia changes number, metaplasia changes type, dysplasia is disordered and premalignant. One line of contrast between metaplasia and dysplasia wins marks in short answers.

Commonly used medical terminologies

Learn the building blocks — they decode hundreds of terms:

- Prefixes: hyper- (excess), hypo- (deficient), a-/an- (without), dys- (abnormal, painful), brady- (slow), tachy- (fast), endo- (inside), ecto-/exo- (outside), peri- (around), sub- (below).
- Suffixes: -itis (inflammation: arthritis, dermatitis), -oma (tumour: carcinoma), -pathy (disease: neuropathy), -emia (blood condition: anaemia), -lysis (breakdown: haemolysis), -plasia (growth: hyperplasia), -rrhage (bursting forth: haemorrhage), -stasis (standing still: haemostasis).
- Practise decoding: hypertension, hypotension, tachycardia, bradycardia, dyspnoea, gastritis, nephritis.

Integumentary system: structure and functions of skin

The skin is the largest organ — about 2 m², around 16% of body weight. It has three layers:

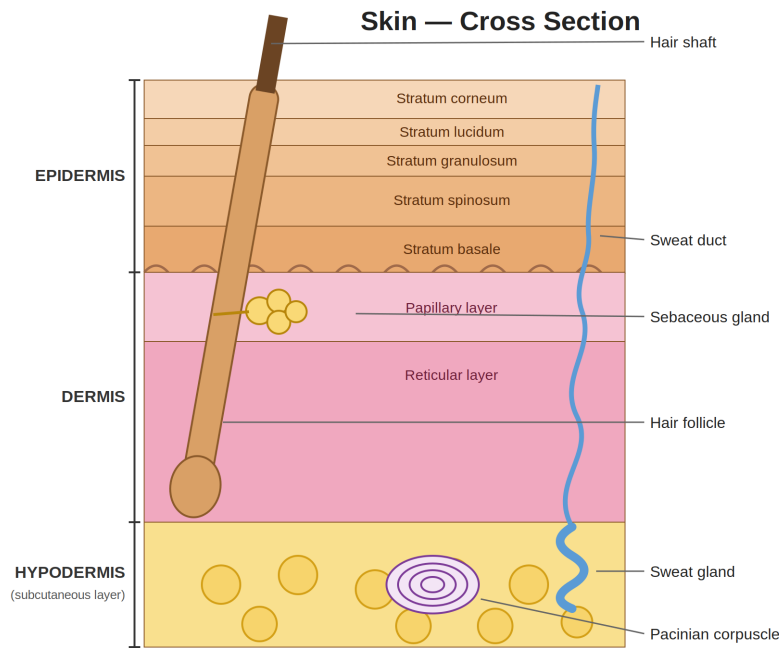
Epidermis — stratified squamous keratinised epithelium, with no blood vessels. Layers from deep to surface: stratum basale (stem cells divide here), stratum spinosum, stratum granulosum, stratum lucidum (thick skin only — palms, soles), stratum corneum (dead keratinised cells, constantly shed). Cells: keratinocytes (make keratin), melanocytes (make melanin — protection against UV), Langerhans cells (immune defence), Merkel cells (touch).

Dermis — connective tissue with blood vessels, nerves, hair follicles, and glands. The papillary layer forms dermal papillae (fingerprints); the reticular layer carries collagen and elastic fibres.

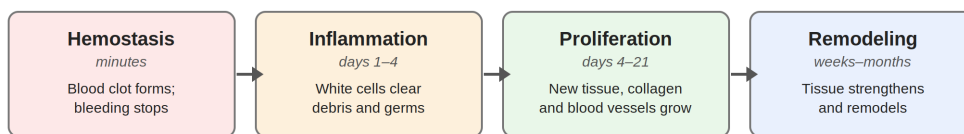
Hypodermis (subcutaneous layer) — fat and loose connective tissue; insulation and energy store.

Appendages: hair, nails, sebaceous glands (sebum oils the skin), and sweat glands — eccrine (all over, temperature control) and apocrine (axilla, groin).

Functions of skin: protection (microbes, UV, chemicals, water loss), temperature regulation (sweating, vasodilation/vasoconstriction), sensation (touch, pressure, pain, temperature), vitamin D synthesis (UV converts 7-dehydrocholesterol), excretion (sweat), absorption (the basis of transdermal drug delivery), and immune defence.



Wound Healing — Timeline



Skin in cross-section, showing epidermis, dermis and hypodermis

Skin disorders: psoriasis, dermatitis, leprosy

Psoriasis — a chronic autoimmune disorder; T-cell mediated hyperproliferation of keratinocytes. Thick, red plaques with silvery scales, typically on extensor surfaces (elbows, knees) and the scalp. Auspitz sign (pinpoint bleeding when a scale is removed), Koebner phenomenon (lesions appear at sites of skin trauma), nail pitting. Pharmacy link: treated with topical corticosteroids, vitamin D analogues (calcipotriol), and biologics.

Dermatitis — inflammation of the skin: redness, itching, swelling. Types: atopic dermatitis (eczema — allergic, often in children), contact dermatitis (irritant or allergic — e.g., nickel, cosmetics), seborrheic dermatitis (oily areas, dandruff).

Leprosy (Hansen's disease) — caused by *Mycobacterium leprae*; attacks skin and peripheral nerves (loss of sensation, deformities). Two poles: tuberculoid (paucibacillary) — few lesions, strong cell-mediated immunity; lepromatous (multibacillary) — many lesions, weak immunity, leonine facies. Treated with multidrug therapy: dapsone + rifampicin + clofazimine. Still relevant in India — know it well.

Principles of wound healing

The four phases overlap and run in order:

- 1 Haemostasis (minutes) — vasoconstriction, platelet plug, clot formation stops bleeding.
- 2 Inflammatory phase (1–3 days) — neutrophils, then macrophages, clear debris and bacteria.
- 3 Proliferative phase (day 3 to about 3 weeks) — fibroblasts lay down collagen (type III), new blood vessels form (angiogenesis), granulation tissue fills the wound, epithelial cells cover the surface.
- 4 Remodelling/maturation (weeks to months) — type III collagen is replaced by stronger type I; the scar contracts and strengthens (it never regains 100% of original strength).

Primary intention (clean surgical wound, edges closed — heals fast, thin scar) vs secondary intention (open wound healing by granulation — larger scar, e.g., pressure ulcers).

Factors affecting healing: nutrition (protein, vitamin C for collagen, zinc), infection, poor blood supply, diabetes, old age, smoking — and drugs: corticosteroids and NSAIDs delay healing. Pharmacy link: this is why diabetic foot care and perioperative drug review matter.

◆ **Exam tip: Mnemonic for the epidermal strata, deep to surface — "Brittany Spears Glitters Like Candy"**

Basale, Spinosum, Granulosum, Lucidum, Corneum. For wound healing: "HIPR" — Haemostasis, Inflammatory, Proliferative, Remodelling.

Very short questions (2 marks)

- 1 Define anatomy, physiology and pathophysiology in one line each.
- 2 Name the three components of a feedback system.
- 3 What does the sodium-potassium pump move, and in which directions?
- 4 Name the five types of cell junctions and one tissue where each is found.
- 5 List the layers of the epidermis from deep to superficial.

Short questions (5 marks)

- 1 Explain homeostasis with the help of one negative and one positive feedback example.
- 2 Compare passive and active transport across the cell membrane with examples.
- 3 Differentiate necrosis from apoptosis.

Long questions (10 marks)

- 1 Classify tissues. Describe the structure, location and functions of the different types of epithelial tissue with labelled diagrams.
- 2 Describe the structure and functions of the skin. Add a note on the pathophysiology of psoriasis and leprosy, and the phases of wound healing.

How to approach these answers

- Very-short Q1: three one-line definitions — keep each under 15 words, no examples needed.
- Very-short Q2: name receptor, control centre, effector in order; one example is a bonus.
- Very-short Q3: "3 Na⁺ out, 2 K⁺ in, uses ATP" — that single line is the full answer.
- Very-short Q4: junction + tissue as five pairs; spelling counts.
- Very-short Q5: five strata in order, deep to superficial — use the mnemonic.
- Short Q1: define homeostasis first, draw the loop, then work one negative example fully and name one positive one.
- Short Q2: two-column table (type, energy, gradient, example); end with the Na⁺/K⁺ pump line.
- Short Q3: definition of each + three contrasts (planned/unplanned, inflammation present/absent, morphology).
- Long Q1: intro (four tissue types), main body under 3 subheads — classification scheme, each epithelial type with location + function, labelled sketches — conclusion.
- Long Q2: intro (skin as largest organ), main body under 3 subheads — three layers with diagram, psoriasis + leprosy pathophysiology, wound healing phases — conclusion with pharmacy relevance.

Unit II — Skeletal System, Joints, Muscle Contraction and Joint Diseases

Divisions of the skeletal system

The adult skeleton has 206 bones, divided into two parts:

- Axial skeleton (80 bones) — skull (cranium 8 + face 14, plus 6 auditory ossicles and 1 hyoid), vertebral column (33 vertebrae), ribs (12 pairs), sternum.
- Appendicular skeleton (126 bones) — pectoral girdle (clavicle, scapula × 2), upper limbs (30 each), pelvic girdle (2 hip bones), lower limbs (30 each).

Functions of the skeleton: support, protection (skull guards the brain, rib cage guards heart and lungs), movement (bones act as levers for muscles), mineral storage (calcium, phosphorus), blood cell formation (red bone marrow), and fat storage (yellow marrow).

Types of bones

- Long bones — longer than wide, with a shaft (diaphysis) and ends (epiphyses). E.g., femur, humerus. The femur is the longest and strongest bone in the body.
- Short bones — cube-shaped; carpals (wrist), tarsals (ankle).

- Flat bones — thin and curved; skull bones, ribs, sternum, scapula. Protect organs; the sternum and pelvis produce blood cells.
- Irregular bones — complex shapes; vertebrae, facial bones.
- Sesamoid bones — embedded in tendons; the patella (kneecap) is the largest sesamoid.

Salient features of the axial skeleton

Skull: the cranium (frontal, 2 parietal, 2 temporal, occipital, sphenoid, ethmoid) protects the brain; the facial bones form the orbits, nasal cavity, and jaws. The foramen magnum in the occipital bone lets the spinal cord pass through. Sutures are fibrous joints between skull bones.

Vertebral column: 7 cervical (the atlas C1 and axis C2 allow head rotation via the dens of the axis), 12 thoracic (attach to ribs), 5 lumbar (large, bear body weight), 5 fused sacral, 4 fused coccygeal. Normal curves: cervical and lumbar lordosis (inward), thoracic and sacral kyphosis (outward). Intervertebral discs (fibrocartilage with a nucleus pulposus) cushion the vertebrae — a "slipped disc" is herniation of this nucleus.

Rib cage: 12 pairs — 7 true ribs (attach directly to the sternum), 3 false ribs (attach via cartilage), 2 floating ribs (no anterior attachment). Protects the heart and lungs; moves during breathing.

Sternum: manubrium, body, xiphoid process. A site of bone marrow aspiration in adults.

Salient features of the appendicular skeleton

Pectoral girdle: clavicle (collarbone — the most commonly fractured bone) and scapula (shoulder blade, with acromion and coracoid processes, glenoid cavity).

Upper limb: humerus (head fits into the glenoid cavity), radius and ulna (forearm — radius on the thumb side), 8 carpals (the scaphoid fractures most often), 5 metacarpals, 14 phalanges.

Pelvic girdle: each hip bone = ilium + ischium + pubis fused at the acetabulum (hip socket). The female pelvis is wider with a broader sciatic notch — an adaptation for childbirth.

Lower limb: femur (head fits into the acetabulum), patella, tibia (shinbone — medial, weight-bearing) and fibula (lateral, slender), 7 tarsals (the calcaneus is the heel bone and largest tarsal), 5 metatarsals, 14 phalanges.

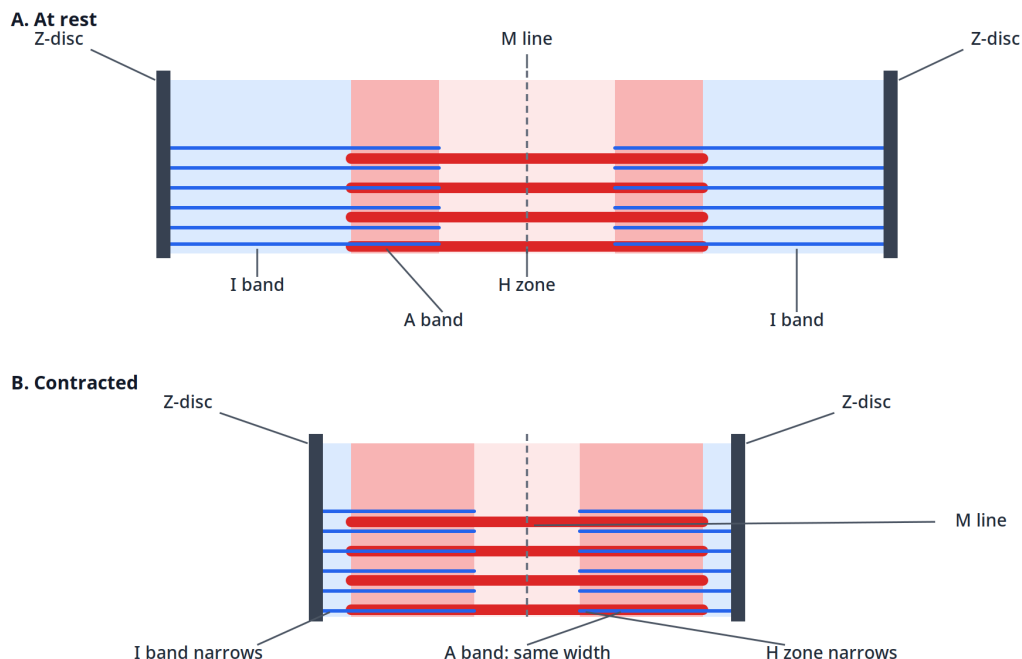
Organisation of skeletal muscle

From outside in: the whole muscle is wrapped in epimysium → bundles of fibres (fascicles) wrapped in perimysium → each muscle fibre wrapped in endomysium. Tendons (dense connective tissue, mainly collagen) attach muscle to bone.

A muscle fibre contains myofibrils, which contain sarcomeres — the contractile unit from one Z-disc to the next. Within a sarcomere, thin filaments (actin, with troponin and tropomyosin) slide over thick filaments (myosin). The bands: A band (thick filaments, dark), I band (thin

filaments only, light), H zone (centre of the A band), M line (middle).

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: the contractile unit of skeletal muscle

Physiology of muscle contraction — the sliding filament theory

- 1 A nerve impulse reaches the muscle; calcium ions (Ca^{2+}) are released from the sarcoplasmic reticulum.
- 2 Ca^{2+} binds troponin, moving tropomyosin off the actin binding sites.
- 3 Myosin heads attach to actin, forming cross-bridges; using ATP, the myosin head pivots — the power stroke — pulling actin toward the centre.
- 4 Fresh ATP binds, the head detaches, and the cycle repeats. The sarcomere shortens; the I bands and H zone shrink, but the A band stays the same length.
- 5 When stimulation stops, Ca^{2+} is pumped back into the sarcoplasmic reticulum and the muscle relaxes.

The point students miss most: ATP is needed both for the power stroke and for detachment/relaxation — this is why rigor mortis (stiffness after death) happens when ATP runs out.

A single stimulus gives a muscle twitch; repeated stimuli add up (summation) into tetanus (sustained contraction). Isotonic contraction shortens the muscle (lifting a weight); isometric

contraction keeps the length unchanged (pushing against a wall).

Exam tip: In the sliding filament answer, always state which bands change: I band and H zone shrink, A band stays constant. That one line separates a 7-mark answer from a 10-mark one.

The neuromuscular junction (NMJ)

The synapse between a motor neuron and a muscle fibre:

- 1 The action potential arrives at the axon terminal → voltage-gated Ca^{2+} channels open.
- 2 Ca^{2+} triggers release of acetylcholine (ACh) into the synaptic cleft.
- 3 ACh binds nicotinic receptors on the motor end plate → Na^+ influx → end-plate potential → muscle action potential → contraction.
- 4 Acetylcholinesterase breaks down ACh, ending the signal.

Pharmacy links you will meet in pharmacology: myasthenia gravis is treated with acetylcholinesterase inhibitors (neostigmine); tubocurarine and similar neuromuscular blockers block nicotinic receptors and are used in anaesthesia.

Structural and functional classification of joints

Structural classification (by the material binding the bones):

- Fibrous joints — bound by dense connective tissue; no movement. E.g., sutures of the skull, gomphosis (tooth in socket).
- Cartilaginous joints — bound by cartilage; slight movement. E.g., intervertebral discs, pubic symphysis.
- Synovial joints — bones separated by a fluid-filled cavity; freely movable. Features: articular cartilage, joint (synovial) cavity with synovial fluid, articular capsule, reinforcing ligaments.

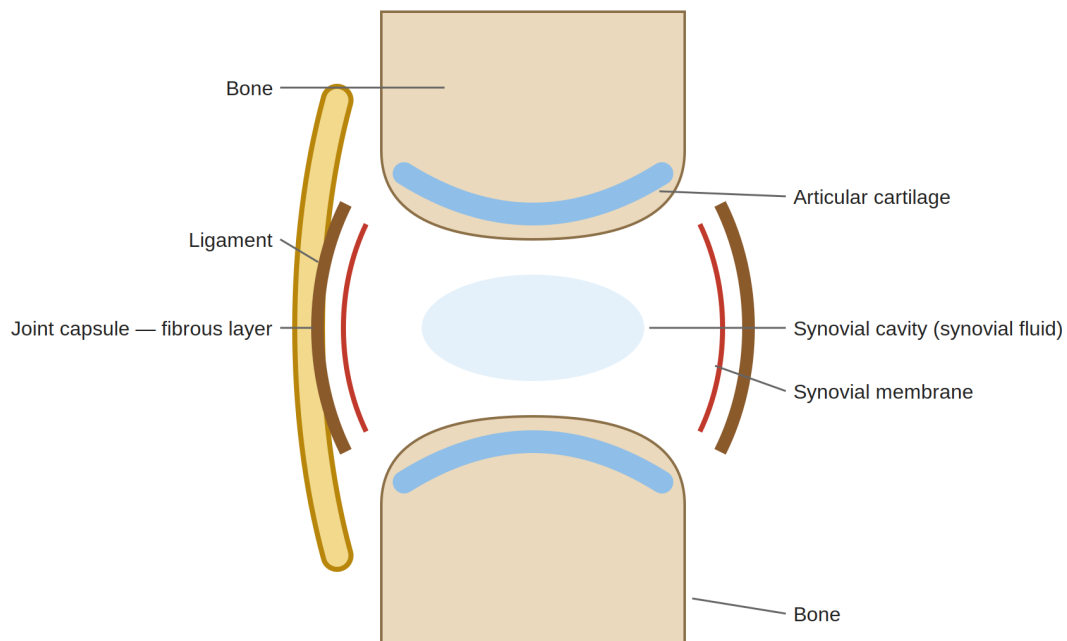
Functional classification: synarthrosis (immovable), amphiarthrosis (slightly movable), diarthrosis (freely movable).

Types of synovial joints — memorise one example each:

Joint type	Movement	Example
Ball-and-socket	All planes	Shoulder, hip
Hinge	Flexion/extension only	Elbow, knee, ankle
Pivot	Rotation	Atlas-axis joint (head rotation)
Saddle	Two planes	Thumb carpometacarpal joint
Condylloid (ellipsoid)	Two planes	Wrist joint

Joint type	Movement	Example
Gliding (plane)	Sliding	Between carpals, between tarsals

Synovial Joint — Cross Section



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

Synovial joint: articular cartilage, cavity, capsule and ligaments

Pathophysiology of rheumatoid arthritis (RA)

Definition: a chronic autoimmune inflammatory disorder attacking the synovial membrane of joints — the small joints of the hands and feet first, symmetrically.

Pathogenesis: an unknown trigger sets off an immune attack on the synovium → synovial inflammation (synovitis) → formation of pannus (invasive granulation tissue) → destruction of cartilage and bone → joint deformity (swan-neck and boutonnière deformities, ulnar deviation).

Markers: rheumatoid factor (RF) and anti-CCP antibodies (more specific for RA).

Clinical features: morning stiffness lasting more than 30–60 minutes (the key difference from osteoarthritis), symmetric polyarthritis, fatigue, rheumatoid nodules.

RA vs osteoarthritis (a favourite comparison): OA is degenerative "wear and tear" of weight-bearing joints in the elderly — worse with activity, better with rest, stiffness under 30 minutes. RA is autoimmune and symmetric — worse with rest (prolonged morning stiffness).

Pharmacy link: treated with NSAIDs, DMARDs (methotrexate is first-line), and biologics (TNF- α inhibitors).

Pathophysiology of osteoporosis

Definition: reduced bone mineral density with deteriorated bone microarchitecture → fragile bones that fracture from trivial trauma.

Pathogenesis: an imbalance between osteoblast (bone-forming) and osteoclast (bone-resorbing) activity — resorption wins. The main driver is oestrogen deficiency after menopause (oestrogen normally restrains osteoclasts); other factors are ageing, low calcium/vitamin D intake, smoking, alcohol, corticosteroid use, and immobility.

Clinical features: often silent until a fracture occurs. Classic fracture sites: the hip (neck of femur), vertebrae (compression → kyphosis and height loss), and distal radius (Colles' fracture). Back pain from vertebral crush fractures.

Diagnosis: DEXA scan; a T-score of -2.5 or lower means osteoporosis.

Pharmacy link: calcium + vitamin D supplementation, bisphosphonates (alendronate — taken upright with water on an empty stomach), and the link between long-term steroid therapy and bone loss.

Exam tip: Osteopenia (T-score -1 to -2.5) vs osteoporosis (≤ -2.5) — one line on the DEXA cut-off shows the examiner you know the diagnostic standard, not just the theory.

Pathophysiology of gout

Definition: crystal-induced arthritis from hyperuricaemia (high blood uric acid) → monosodium urate crystals deposit in joints.

Pathogenesis: overproduction of uric acid (high-purine diet — red meat, seafood, alcohol; rapid cell turnover) or underexcretion (kidney disease, diuretics) → crystals deposit in the joint → neutrophils attack them → sudden, severe inflammation.

Clinical features: classic podagra — sudden agonising pain, redness, and swelling of the first metatarsophalangeal joint (big toe), often starting at night. Chronic gout forms tophi (chalky urate deposits) in the ears, elbows, and joints, plus urate kidney stones.

Pharmacy link: acute attacks — NSAIDs, colchicine (watch GI toxicity); long-term control — allopurinol (xanthine oxidase inhibitor, reduces uric acid production). Avoid thiazide diuretics, which raise uric acid.

Very short questions (2 marks)

- 1 How many bones are there in the axial and appendicular skeleton of an adult?
- 2 Name the thick and thin filaments of the sarcomere and the bands seen in it.

- 3 Which neurotransmitter acts at the neuromuscular junction, and which enzyme ends its action?
- 4 Name the six types of synovial joints with one example each.
- 5 Which joint is classically affected first in gout?

Short questions (5 marks)

- 1 Explain the sliding filament theory of muscle contraction.
- 2 Write a short note on the pathophysiology of rheumatoid arthritis.
- 3 Differentiate rheumatoid arthritis from osteoarthritis.

Long questions (10 marks)

- 1 Describe the physiology of skeletal muscle contraction and the events at the neuromuscular junction. Add the pharmacy relevance of both.
- 2 Describe the pathophysiology of osteoporosis and gout. Compare their causes, clinical features and treatment approaches.

How to approach these answers

- Very-short Q1: "206 total — 80 axial, 126 appendicular" is the whole answer.
- Very-short Q2: filaments (actin/myosin) + bands (A, I, H, M) as two short lists.
- Very-short Q3: acetylcholine; acetylcholinesterase — one line.
- Very-short Q4: six joint types as name-example pairs, no descriptions.
- Very-short Q5: first metatarsophalangeal joint (big toe) + the term podagra.
- Short Q1: five numbered steps in order (Ca^{2+} → troponin → cross-bridge → power stroke → relaxation), close with the A-band constant line.
- Short Q2: definition → pathogenesis in sequence → markers → one clinical feature (morning stiffness) → one drug class.
- Short Q3: a two-column table (cause, joints, stiffness pattern, markers) — the table itself is the answer.
- Long Q1: intro, 3 subheads — muscle organisation/sarcomere, sliding filament steps with the sarcomere diagram, NMJ steps — conclusion with the myasthenia gravis and muscle-relaxant links.
- Long Q2: intro, 3 subheads — osteoporosis (definition, oestrogen mechanism, fractures, DEXA), gout (hyperuricaemia, crystal mechanism, podagra/tophi), comparison table of cause/features/treatment — conclusion.

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

Body fluids: distribution and compartments

Water is about 60% of body weight in an adult male (slightly less in females and the elderly, more in infants). It sits in two main compartments:

- Intracellular fluid (ICF) — inside the cells; about 40% of body weight (two-thirds of total body water). Rich in K^+ , Mg^{2+} , and phosphate.
- Extracellular fluid (ECF) — outside the cells; about 20% of body weight (one-third of total water). Rich in Na^+ , Cl^- , and HCO_3^- . Subdivided into plasma (about 4–5% of body weight — the fluid part of blood) and interstitial fluid (about 15%, bathing the cells), plus small volumes such as lymph and CSF.

Fluid moves between compartments by osmosis and filtration (Starling forces at the capillaries). Oedema is excess interstitial fluid — from increased capillary permeability, low plasma protein, or blocked lymph drainage.

Composition and functions of blood

Blood is about 5 litres in an adult (roughly 7–8% of body weight). Its components:

Plasma (about 55%) — 90% water, carrying proteins: albumin (maintains osmotic pressure; carries drugs — hypoalbuminaemia changes drug distribution), globulins (antibodies, transport proteins), fibrinogen (clotting); plus nutrients, hormones, gases, waste products, and electrolytes.

Formed elements (about 45%):

- RBCs (erythrocytes) — 4.5–5.5 million/ μL ; biconcave discs with no nucleus, packed with haemoglobin; carry O_2 (and some CO_2); lifespan about 120 days; destroyed in the spleen.
- WBCs (leukocytes) — 4,000–11,000/ μL ; defence. Granulocytes: neutrophils (phagocytosis of bacteria), eosinophils (parasites and allergy), basophils (release histamine and heparin). Agranulocytes: lymphocytes (B cells make antibodies, T cells kill infected cells), monocytes (become macrophages in tissues).
- Platelets (thrombocytes) — 1.5–4 lakh/ μL (150,000–400,000); cell fragments from megakaryocytes; form the platelet plug in clotting.

Functions of blood: transport (O_2 , CO_2 , nutrients, hormones, waste, drugs), regulation (pH, temperature, fluid balance), and protection (clotting, immunity).

Haematocrit (PCV) — the percentage of blood volume occupied by RBCs: roughly 40–54% in men, 36–46% in women. It rises in dehydration and polycythaemia, and falls in anaemia.

Exam tip: Plasma vs serum is a 2-mark trap: plasma is the fluid of unclotted blood (contains fibrinogen and clotting factors); serum is what is left after blood clots (plasma minus fibrinogen and clotting factors).

Haemopoiesis and haemoglobin formation

Haemopoiesis (blood cell formation) takes place in red bone marrow (in adults: pelvis, vertebrae, sternum, ribs, skull). All blood cells arise from haematopoietic stem cells, which divide into myeloid and lymphoid lines.

Erythropoiesis (RBC formation) is stimulated by erythropoietin (EPO), made by the kidney in response to low oxygen. It needs iron, vitamin B₁₂, and folic acid. Development runs from erythroblast → reticulocyte → mature RBC, losing the nucleus along the way.

Haemoglobin formation: each Hb molecule = 4 haem groups (each carrying one Fe²⁺ iron) + 4 globin chains (2 alpha + 2 beta in adult HbA). Iron is absorbed in the duodenum (vitamin C helps absorption), transported by transferrin, stored as ferritin and haemosiderin. Old RBCs are broken down in the spleen and liver; haem becomes bilirubin (excreted in bile — excess causes jaundice), and iron is recycled.

Mechanism of blood coagulation

Haemostasis has three stages: vascular spasm (the vessel constricts), platelet plug formation (platelets stick to exposed collagen via von Willebrand factor, then aggregate), and coagulation (a fibrin mesh stabilises the plug).

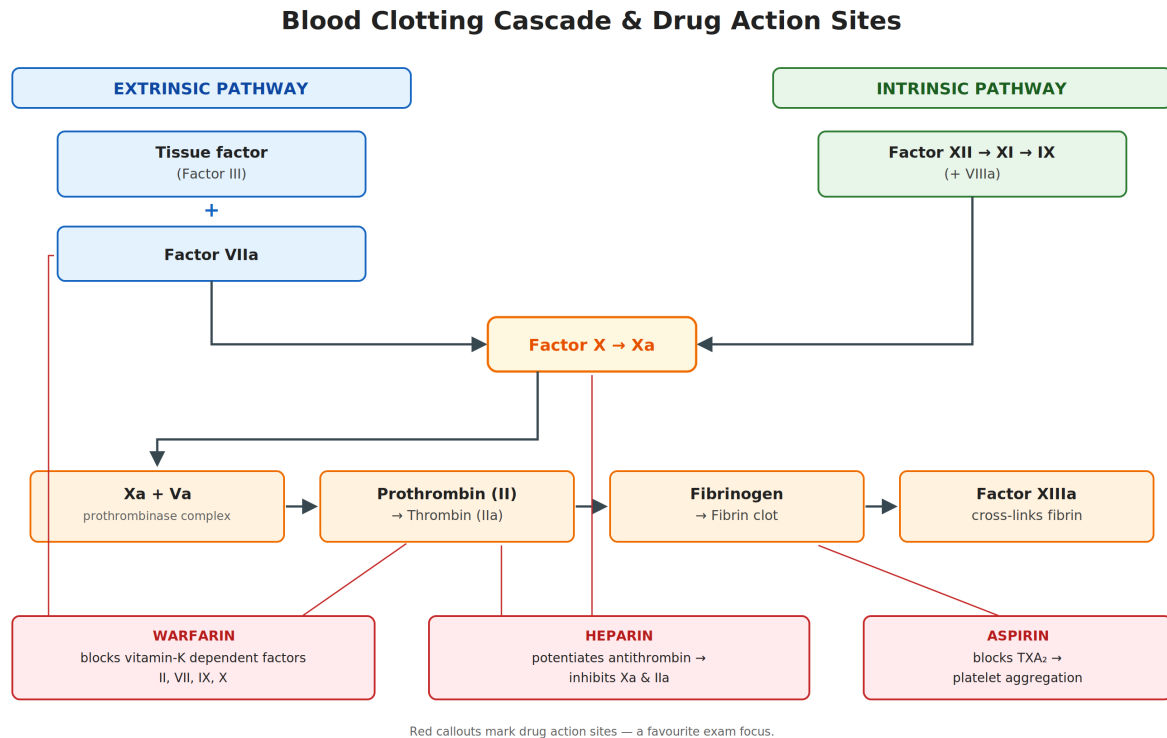
The clotting cascade (the simplified, exam-safe version):

- Extrinsic pathway — triggered by tissue injury releasing tissue factor (factor III); fast; measured by PT/INR (used to monitor warfarin).
- Intrinsic pathway — triggered by blood contacting exposed collagen; slower; measured by aPTT (used to monitor heparin).
- Common pathway — both converge: factor X → Xa converts prothrombin (II) to thrombin (IIa) → thrombin converts fibrinogen (I) to fibrin → fibrin mesh + factor XIII cross-linking = stable clot.

Key factors to name: I (fibrinogen), II (prothrombin), III (tissue factor), VIII (antihaemophilic — deficient in haemophilia A), IX (deficient in haemophilia B), plus calcium (factor IV) and vitamin K (needed to make factors II, VII, IX, X — this is why warfarin, a vitamin K antagonist, is an anticoagulant).

Fibrinolysis: plasmin dissolves the clot once healing is done (streptokinase and alteplase work by activating plasminogen — used in MI and stroke).

Pharmacy link: this one cascade explains heparin (intrinsic/aPTT), warfarin (extrinsic/PT-INR, vitamin K dependent), and aspirin/clopidogrel (antiplatelet — they block the platelet plug stage, not the cascade).



The clotting cascade: extrinsic, intrinsic and common pathways

Blood grouping, Rh factor and transfusion

ABO system (Landsteiner): RBCs carry A and/or B antigens; plasma carries antibodies against whichever antigens you lack. Group A has anti-B, group B has anti-A, group AB has neither antibody (universal recipient), group O has both antibodies (universal donor for RBCs).

Rh factor: Rh-positive RBCs carry the D antigen. Rh-negative people have no anti-D antibody naturally, but develop it after exposure (transfusion or pregnancy) — causing haemolytic disease of the newborn in a later Rh-positive pregnancy. Prevented with anti-D immunoglobulin (RhoGAM).

Transfusion rules: always cross-match. ABO incompatibility causes an acute haemolytic transfusion reaction — donor RBCs agglutinate and burst, with fever, chills, haemolysis, kidney damage, and shock.

Exam tip: For transfusion questions, draw a 4×4 compatibility table (donor rows × recipient columns with tick/cross marks). It is faster than reasoning each combination out, and examiners accept it as the answer.

Lymphatic organs, tissues, vessels and lymph

Lymphatic organs: lymph nodes (filter lymph; swell in infection), spleen (filters blood, destroys old RBCs, mounts immune responses), thymus (T-cell maturation — most active in childhood), tonsils and Peyer's patches (MALT — guard the body's entry points).

Lymphatic vessels: begin as blind capillaries, drain through nodes, and return lymph to blood at the subclavian veins (via the thoracic duct on the left and the right lymphatic duct on the right). One-way valves prevent backflow.

Lymph formation: plasma filtered out of capillaries becomes interstitial fluid; the part not reabsorbed (about 3 L/day) enters the lymph capillaries as lymph.

Functions of the lymphatic system: return tissue fluid to blood, absorb dietary fats (lacteals in the intestine), and immune surveillance (lymphocytes in nodes trap pathogens).

Inflammation: classification, pathophysiology, mediators, repair

Definition: inflammation is the vascular and cellular response to injury or infection. The five cardinal signs (Celsus + Galen): rubor (redness), tumor (swelling), calor (heat), dolor (pain), and functio laesa (loss of function).

Classification: acute (seconds to days; neutrophils; e.g., a cut, bacterial infection) vs chronic (weeks to years; macrophages and lymphocytes; e.g., tuberculosis, rheumatoid arthritis, granulomas).

Pathophysiology — vascular and cellular events:

- 1 Vasodilation (histamine, prostaglandins) → redness and heat.
- 2 Increased vascular permeability → plasma leaks out → swelling (oedema) and pain from pressure.
- 3 Cellular events: margination → rolling → adhesion → diapedesis (WBCs squeeze through the vessel wall) → chemotaxis (WBCs follow chemical signals to the injury) → phagocytosis of microbes and debris.

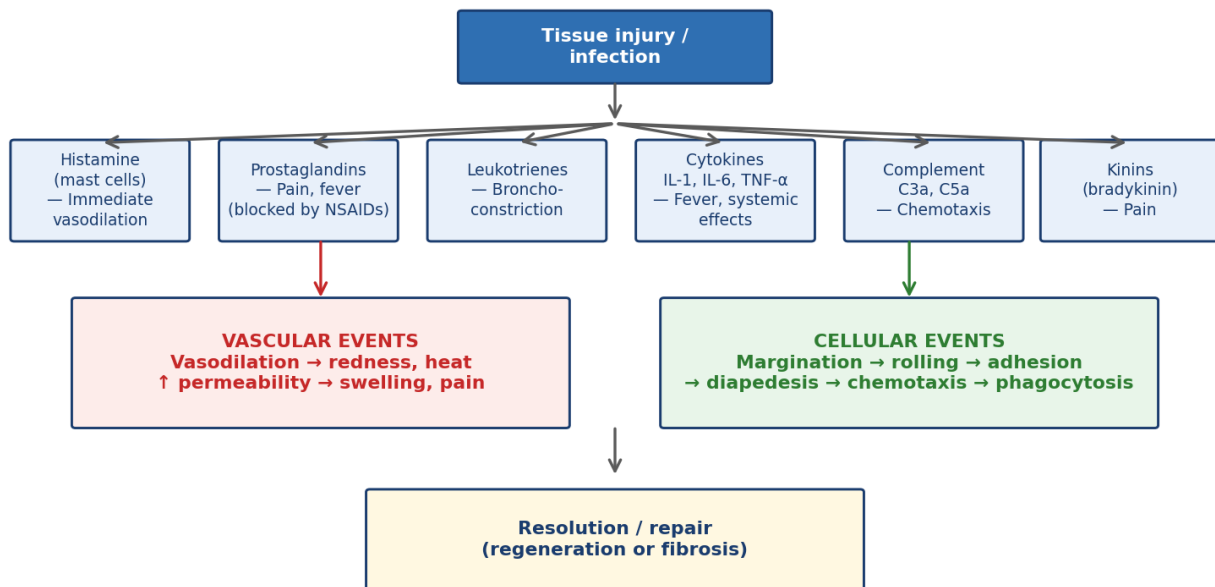
Mediators of inflammation — learn this as a list; examiners love it:

Mediator	Source / note	Main effect
Histamine	Mast cells	Immediate vasodilation
Prostaglandins	Arachidonic acid	Pain, fever (blocked by NSAIDs)
Leukotrienes	Arachidonic acid	Bronchoconstriction (asthma)
Cytokines (IL-1, IL-6, TNF- α)	Macrophages	Fever, systemic effects
Complement (C3a, C5a)	Plasma proteins	Chemotaxis
Kinins (bradykinin)	Plasma proteins	Pain
Platelet-activating factor	Platelets	Amplifies the response

Repair: regeneration (the same tissue regrows — liver, skin epithelium) vs fibrosis/scarring (connective tissue fills the gap — heart muscle after MI). Which one happens depends on the tissue's regenerative capacity and the extent of damage.

Pharmacy link: NSAIDs and corticosteroids both suppress inflammation but at different points — NSAIDs block prostaglandin synthesis (COX enzymes), steroids block earlier steps (phospholipase A₂).

Inflammation: mediators, vascular events, cellular events



Five cardinal signs: rubor (redness), tumor (swelling), calor (heat), dolor (pain), functio laesa (loss of function)

Inflammation: mediators, vascular events and cellular events

Haematological diseases: the anaemias

Iron deficiency anaemia

The commonest anaemia worldwide. Cause: blood loss (menstruation, hookworm, piles), poor intake, pregnancy. Iron is needed to make haem. Microcytic hypochromic RBCs (small, pale). Features: fatigue, pallor, koilonychia (spoon-shaped nails), pica (craving non-food items like clay), angular cheilitis. Treated with ferrous sulphate/fumarate — counsel patients that it blackens stools and is best absorbed on an empty stomach with vitamin C.

Megaloblastic anaemia (vitamin B₁₂ and folic acid deficiency)

Both vitamins are needed for DNA synthesis; without them, RBC precursors cannot divide → large, immature (megaloblastic), macrocytic RBCs. B₁₂ deficiency (pernicious anaemia — lack of intrinsic factor; strict vegetarian diets) additionally causes neurological symptoms (tingling, numbness, subacute combined degeneration of the cord) because B₁₂ maintains myelin — folate deficiency does not. Treat B₁₂ deficiency with B₁₂, never folate alone (folate masks the anaemia while nerve damage progresses silently).

Sickle cell anaemia

Autosomal recessive. A single amino-acid swap in beta-globin (glutamate → valine) makes HbS, which polymerises in low oxygen → RBCs sickle. Sickled cells block small vessels (vaso-occlusive painful crises), haemolyse early (haemolytic anaemia), and damage the spleen (infection risk). Common in central India (Madhya Pradesh, Chhattisgarh, Maharashtra tribal belts) — know it. Managed with hydroxyurea, folic acid, vaccination, and crisis care.

Thalassemia

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

Exam tip: Iron deficiency vs thalassemia is a classic confusion — both are microcytic hypochromic, but in iron deficiency the iron studies are low, while in thalassemia they are normal or high. One line of contrast earns the differentiation mark.

Hereditary and acquired haemolytic anaemias

A note on the syllabus phrase "hereditary acquired anaemia": it reads as a compressed phrase for hereditary haemolytic anaemias and acquired haemolytic anaemias, so both are covered here. The classic hereditary example is hereditary spherocytosis — defective membrane proteins (spectrin/ankyrin) make fragile spherical RBCs that are destroyed in the spleen; treated by splenectomy. Acquired examples include autoimmune haemolytic anaemia and drug-induced haemolysis.

Haemophilia

An X-linked recessive bleeding disorder (males affected, females carriers). Haemophilia A: factor VIII deficiency (commonest, about 85%). Haemophilia B (Christmas disease): factor IX deficiency. Features: prolonged bleeding after injury, haemarthrosis (bleeding into joints), easy bruising; PT normal, aPTT prolonged. Treated with factor concentrates; avoid NSAIDs and IM injections.

Very short questions (2 marks)

- 1 What is the difference between plasma and serum?
- 2 Name the three stages of haemostasis.
- 3 Which laboratory tests monitor heparin and warfarin therapy?
- 4 List the five cardinal signs of inflammation.
- 5 Why is folate never given alone in vitamin B₁₂ deficiency?

Short questions (5 marks)

- 1 Explain the clotting cascade with reference to the extrinsic, intrinsic and common pathways.

- 2 Describe the vascular and cellular events of acute inflammation with its mediators.
- 3 Differentiate iron deficiency anaemia from megaloblastic anaemia.

Long questions (10 marks)

- 1 Describe the mechanism of blood coagulation. Explain how heparin, warfarin and aspirin act at different points, with a labelled flowchart.
- 2 Classify the anaemias. Describe the pathophysiology of iron deficiency anaemia, sickle cell anaemia and thalassemia, with their distinguishing laboratory features.

How to approach these answers

- Very-short Q1: one line on unclotted vs clotted fluid; name fibrinogen as the difference.
- Very-short Q2: three stages in order — vascular spasm, platelet plug, coagulation.
- Very-short Q3: heparin → aPTT (intrinsic), warfarin → PT/INR (extrinsic) — pair each drug with its pathway.
- Very-short Q4: the five Latin terms with English meanings in brackets.
- Very-short Q5: "folate corrects the blood picture but lets nerve damage progress" — one line.
- Short Q1: three pathways as a flowchart in words (extrinsic → X, intrinsic → X, common → thrombin → fibrin); end with the PT/aPTT monitoring line.
- Short Q2: vascular events first, then cellular events in sequence (diapedesis → chemotaxis → phagocytosis), then the mediator table.
- Short Q3: a two-column table — cell size, iron studies, neurological signs, treatment.
- Long Q1: intro (three stages of haemostasis), main body under 3 subheads — the cascade with flowchart diagram, drug actions mapped onto it, fibrinolysis — conclusion.
- Long Q2: intro (anaemia = low Hb), 3 subheads — one per anaemia with cause → mechanism → features → labs — conclusion comparing microcytic vs macrocytic vs haemolytic patterns.

Unit IV — Peripheral Nervous System and Special Senses

Classification of the peripheral nervous system

The nervous system divides into the central nervous system (CNS — brain and spinal cord) and the peripheral nervous system (PNS — everything outside the CNS: nerves and ganglia). The PNS itself has two divisions:

- Somatic (voluntary) nervous system — sensory neurons carry information in; motor neurons carry commands out to skeletal muscle. A single neuron runs from CNS to muscle; the neurotransmitter is acetylcholine at the neuromuscular junction.
- Autonomic (involuntary) nervous system — controls smooth muscle, cardiac muscle, and glands. A two-neuron chain (preganglionic → ganglion → postganglionic). Split into sympathetic and parasympathetic divisions; a third, enteric division controls the gut.

Sympathetic vs parasympathetic nervous system

Feature	Sympathetic ("fight or flight")	Parasympathetic ("rest and digest")
Origin	Thoracolumbar (T1-L2)	Craniosacral (cranial nerves III, VII, IX, X + S2-S4)
Ganglia	Close to the spinal cord (paravertebral chain)	Close to or inside the target organ
Preganglionic transmitter	Acetylcholine (nicotinic)	Acetylcholine (nicotinic)
Postganglionic transmitter	Noradrenaline (mostly)	Acetylcholine (muscarinic)
Heart	Increases rate and force	Decreases rate
Pupil	Dilates (mydriasis)	Constricts (miosis)
Bronchi	Dilates	Constricts
Gut	Slows motility, contracts sphincters	Increases motility and secretion
Bladder	Relaxes detrusor, contracts sphincter (retains urine)	Contracts detrusor (voids urine)
Salivary glands	Thick, scanty saliva	Profuse watery saliva

Pharmacy links sit all over this table: beta-blockers blunt sympathetic effects on the heart; anticholinergics (atropine) block parasympathetic effects — dry mouth, dilated pupils, urinary retention is the classic side-effect list; salbutamol mimics sympathetic bronchodilation in asthma.

Autonomic nervous system: sympathetic vs parasympathetic

SYMPATHETIC (<i>'fight or flight'</i>)		PARASYMPATHETIC (<i>'rest and digest'</i>)	
Thoracolumbar (T1–L2) · post-ganglionic = noradrenaline		Craniosacral (III, VII, IX, X, S2–S4) · post-ganglionic = acetylcholine (muscarinic)	
Heart	↑ rate, ↑ force	↓ rate	
Pupil	Dilates (mydriasis)	Constricts (miosis)	
Bronchi	Dilates	Constricts	
Gut	Motility ↓, sphincters contract	Motility ↑, secretion ↑	
Bladder	Retains urine	Voids urine	
Salivary glands	Dry, scanty saliva	Profuse watery saliva	

Exam link: beta-blockers blunt the sympathetic heart column; anticholinergics (atropine) block the parasympathetic column — dry mouth, dilated pupils, urinary retention.

Sympathetic vs parasympathetic: origin, transmitters and organ effects

Exam tip: Memorise this table cold. It answers most autonomic pharmacology questions you will ever face — and in anatomy exams it is usually asked as "compare sympathetic and parasympathetic", which is exactly this table.

Spinal nerves: origin and functions

31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal. Each forms from a dorsal (sensory) root and a ventral (motor) root joining at the intervertebral foramen.

Functions: carry sensory information from skin, muscles, and joints to the cord, and motor commands out to skeletal muscles. A dermatome is the skin area supplied by one spinal nerve — used clinically to locate cord lesions (e.g., shingles follows a dermatome).

Important plexuses (networks where nerves regroup): cervical (phrenic nerve → diaphragm), brachial (upper limb — injuries cause Erb's palsy), lumbar (femoral nerve), sacral (sciatic nerve — the largest nerve in the body; its compression causes sciatica).

Cranial nerves: origin and functions

12 pairs, numbered I–XII, arising from the brain (mostly the brainstem). Learn the names in order with the mnemonic "Oh, Oh, Oh, To Touch And Feel Very Green Vegetables, AH!":

- 1 Olfactory — smell.
- 2 Optic — vision.

- 3 Oculomotor — most eye movements; also constricts the pupil and lifts the eyelid.
 - 4 Trochlear — eye movement (superior oblique muscle).
 - 5 Trigeminal — face sensation + muscles of chewing.
 - 6 Abducens — eye movement (lateral rectus muscle).
 - 7 Facial — facial expression, taste from the anterior tongue, salivary glands.
 - 8 Vestibulocochlear — hearing and balance.
 - 9 Glossopharyngeal — taste from the posterior tongue, swallowing, parotid gland.
 - 10 Vagus — the wanderer: parasympathetic supply to the heart, lungs, and most of the gut; the workhorse of the parasympathetic column above.
 - 11 Accessory (spinal) — trapezius and sternocleidomastoid (shrugging, head turning).
 - 12 Hypoglossal — tongue movements.
-

Structure and functions of the eye

Three coats: outer sclera (white, protective; continuous with the cornea in front) → middle choroid (vascular, pigmented; continuous with the ciliary body and iris — the coloured part controlling pupil size) → inner retina (photoreceptors).

Front to back: cornea (transparent; provides most of the eye's focusing power) → anterior chamber with aqueous humor (drains through the canal of Schlemm — blockage raises pressure in glaucoma) → pupil → lens (fine focusing; changes shape — accommodation) → vitreous humor (gel filling the back of the eye) → retina.

Retina: rods (dim light, black-and-white vision; contain rhodopsin and need vitamin A — deficiency causes night blindness) and cones (colour and sharp vision; concentrated in the fovea at the centre of the macula). The optic disc (blind spot) is where the optic nerve leaves — no photoreceptors there.

Visual pathway (brief): retina → optic nerve → optic chiasma (nasal fibres cross) → optic tract → lateral geniculate body → visual cortex (occipital lobe).

Accessory structures: eyelids, eyelashes, lacrimal (tear) glands, and six extraocular muscles.

Structure and functions of the ear

External ear: pinna (collects sound) → external auditory canal (wax/cerumen) → tympanic membrane (eardrum).

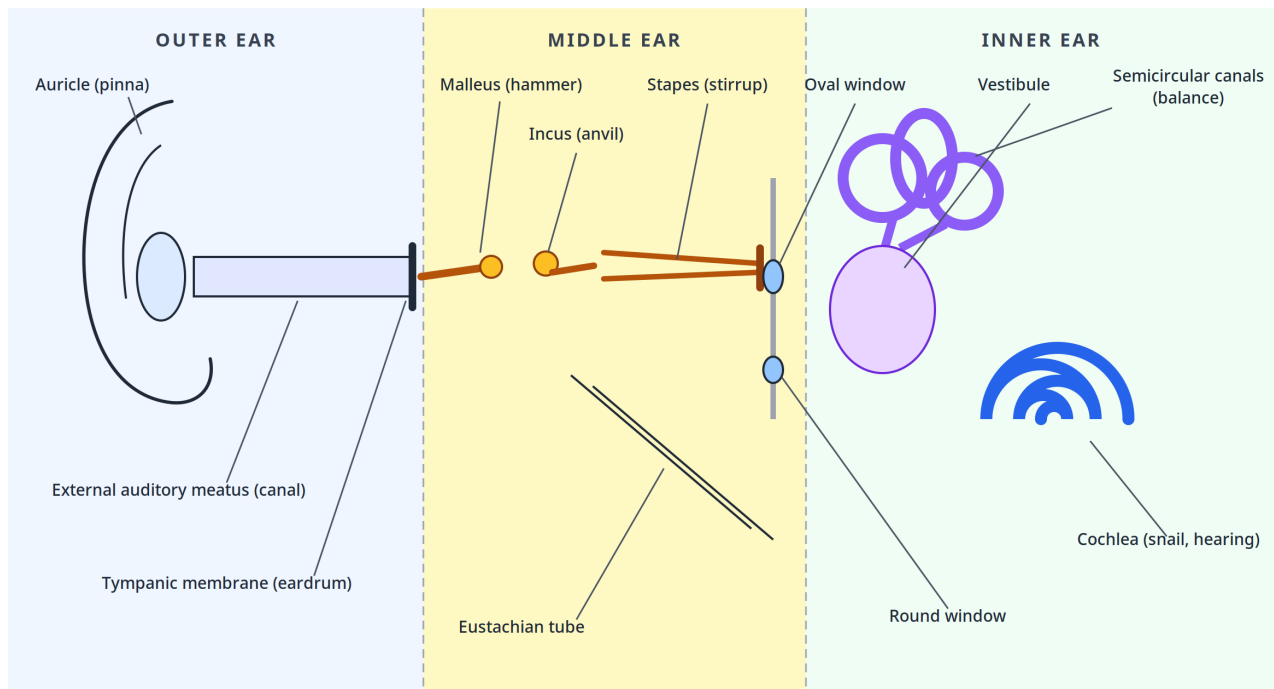
Middle ear: an air-filled cavity with three ossicles — malleus (hammer) → incus (anvil) → stapes (stirrup) — which amplify sound from the eardrum to the inner ear. It connects to the throat via the Eustachian (auditory) tube, which equalises pressure (this is why ears "pop", and why colds lead to middle-ear infections in children).

Inner ear: cochlea (snail-shaped; hair cells convert sound vibrations to nerve impulses — sensorineural hearing), vestibule (utricle and saccule — static balance and head position), and

semicircular canals (rotational balance). The vestibulocochlear nerve (VIII) carries both signals.

Hearing loss types: conductive (external/middle ear — wax, otitis media, ossicle damage; often treatable) vs sensorineural (inner ear/nerve — ageing/presbycusis, noise, ototoxic drugs like aminoglycosides and furosemide — a pharmacy link to remember; usually permanent).

Human Ear — Three Divisions



Ear: external, middle and inner ear with the ossicle chain

Structure and functions of the nose and tongue

Nose (olfaction): olfactory receptors sit in the olfactory epithelium high in the nasal cavity; smells dissolve in mucus, bind receptors → olfactory nerve (I) → olfactory bulb → brain. Smell is the only sense that reaches the cortex without passing through the thalamus — which is why smells trigger memory so strongly. The nose also warms, moistens, and filters air.

Tongue (gustation): taste buds sit mainly on papillae (fungiform, circumvallate, foliate). Five tastes: sweet, salty, sour, bitter, umami. Taste travels via the facial nerve (VII, anterior two-thirds) and glossopharyngeal nerve (IX, posterior third). Most of "taste" is actually smell — which is why food is tasteless with a blocked nose.

Pathophysiology of special sense disorders

Glaucoma — raised intraocular pressure (aqueous humor cannot drain) damages the optic nerve → gradual peripheral vision loss ("tunnel vision"); painless, so it is caught late. Types: open-angle (chronic, commonest) and angle-closure (acute, painful emergency). Pharmacy link: treated with timolol eye drops (beta-blocker), pilocarpine, latanoprost — and systemic anticholinergics can precipitate angle closure.

Cataract — clouding of the lens (ageing, diabetes, UV exposure, steroids) → painless blurring and glare. Treated surgically (lens replacement); no drug reverses it.

Myopia (near-sightedness) — the eyeball is too long or the lens too powerful; distant rays focus in front of the retina. Corrected with concave lenses. Contrast: hypermetropia (focus behind the retina, convex lenses) and presbyopia (ageing lens loses accommodation).

Otitis externa — infection of the outer ear canal ("swimmer's ear"); pain when the pinna is pulled.

Otitis media — middle-ear infection, common in children via the Eustachian tube; ear pain, fever, possible eardrum perforation.

Vertigo — a false sense of spinning or rotation from vestibular disturbance. Causes: BPPV (loose otolith crystals — brief episodes on head movement), Ménière's disease (endolymph build-up — vertigo + tinnitus + hearing loss). Distinguish from dizziness/light-headedness, which is often cardiovascular.

Anosmia — loss of smell; causes include nasal polyps, sinusitis, head injury, viral infections, and zinc deficiency. It matters in pharmacy because it kills appetite, and because intranasal drugs (and some systemic ones) list it as a side effect.

Exam tip: Glaucoma vs cataract is a favourite pair — glaucoma is a pressure problem damaging the optic nerve (timolol drops help), cataract is a lens problem (only surgery helps). State the anatomical site first and the confusion disappears.

Very short questions (2 marks)

- 1 Name the two divisions of the peripheral nervous system and what each controls.
- 2 Which cranial nerve carries parasympathetic supply to the heart and gut?
- 3 Name the three ossicles of the middle ear in order.
- 4 What is the difference between conductive and sensorineural hearing loss?
- 5 Which lens corrects myopia, and why?

Short questions (5 marks)

- 1 Compare the sympathetic and parasympathetic nervous systems.
- 2 Describe the structure of the eye with a labelled diagram.
- 3 Write short notes on glaucoma and cataract.

Long questions (10 marks)

- 1 Classify the peripheral nervous system. Describe the sympathetic and parasympathetic divisions in detail, with their effects on major organs and pharmacy relevance.
- 2 Describe the structure and functions of the eye and ear. Add a note on the pathophysiology of glaucoma, myopia, otitis media and vertigo.

How to approach these answers

- Very-short Q1: somatic (voluntary, skeletal muscle) vs autonomic (involuntary, glands/smooth/cardiac) — one line each.
- Very-short Q2: vagus (X) — name plus "parasympathetic to heart, lungs, gut".
- Very-short Q3: malleus → incus → stapes in order; the order is the mark.
- Very-short Q4: site of lesion (external/middle vs inner ear/nerve) + one example each.
- Very-short Q5: concave lens, because rays focus in front of the retina — site + correction.
- Short Q1: reproduce the comparison table (origin, transmitter, 4-5 organ effects); end with one pharmacy link.
- Short Q2: three coats → front-to-back structures → retina (rods/cones) with the eye diagram.
- Short Q3: two subheads — glaucoma (pressure mechanism, open vs angle-closure, timolol) and cataract (lens clouding, surgery only).
- Long Q1: intro (PNS classification), 3 subheads — somatic vs autonomic, sympathetic detail, parasympathetic detail with the ANS figure — conclusion with beta-blocker/atropine/salbutamol links.
- Long Q2: intro, 3 subheads — eye structure with diagram, ear structure with diagram, four disorders as cause → mechanism → feature — conclusion.

Unit V — Cardiovascular System

Anatomy of the heart

The heart is a fist-sized muscular pump in the mediastinum, tilted to the left, enclosed in the pericardium (fibrous outer sac + serous layers with pericardial fluid for frictionless beating). Its wall has three layers: endocardium (inner lining), myocardium (muscle — thickest in the left ventricle), epicardium (outer layer).

Chambers: right atrium → right ventricle → (lungs) → left atrium → left ventricle → body. The right side handles deoxygenated blood, the left side oxygenated blood; the septum keeps them apart.

Valves (prevent backflow):

- Atrioventricular (AV) valves: tricuspid (right, 3 cusps) and mitral/bicuspid (left, 2 cusps). They are held by chordae tendineae attached to papillary muscles, which stop the valves flipping inside-out during ventricular contraction.
- Semilunar valves: pulmonary (right) and aortic (left), each with 3 cusps and no chordae.

Coronary circulation: the heart feeds itself via the right and left coronary arteries (branching from the aorta just above the aortic valve); blockage here causes myocardial infarction. Deoxygenated blood drains via the coronary sinus into the right atrium.

Blood circulation

- Pulmonary circulation: right ventricle → pulmonary trunk → lungs (CO₂ out, O₂ in) → pulmonary veins → left atrium.
- Systemic circulation: left ventricle → aorta → body tissues → venae cavae → right atrium.
- Coronary circulation (above) and portal circulation (gut → liver via the hepatic portal vein before reaching the heart — this is why many oral drugs undergo first-pass metabolism in the liver; a direct pharmacy link).

Note the two exceptions students mix up: pulmonary arteries carry deoxygenated blood and pulmonary veins carry oxygenated blood — the only places where artery/vein does not match oxygenation.

Exam tip: "Trace the path of blood through the heart" is asked almost every year. Write it as one chain: venae cavae → right atrium → tricuspid → right ventricle → pulmonary valve → lungs → pulmonary veins → left atrium → mitral → left ventricle → aortic valve → aorta. Valves included — examiners check for them.

Structure and functions of blood vessels

Arteries and veins share three layers (tunics): tunica intima (endothelium), tunica media (smooth muscle + elastic fibres), tunica externa/adventitia (connective tissue).

- Arteries — thick muscular/elastic walls that withstand high pressure; carry blood away from the heart. Elastic arteries (aorta) stretch and recoil; muscular arteries distribute blood.
- Arterioles — the main resistance vessels; their constriction and dilation control blood pressure and local flow.
- Capillaries — one endothelial cell thick; the site of exchange (O₂, nutrients, waste) by diffusion and filtration.
- Veins — thin walls, large lumens, valves to prevent backflow; carry blood toward the heart and act as blood reservoirs (about 60% of blood volume). Varicose veins are failed venous valves.

Atherosclerosis begins with damage to the endothelium (tunica intima) — keep this layer in mind for the IHD section.

Conduction system of the heart and the heartbeat

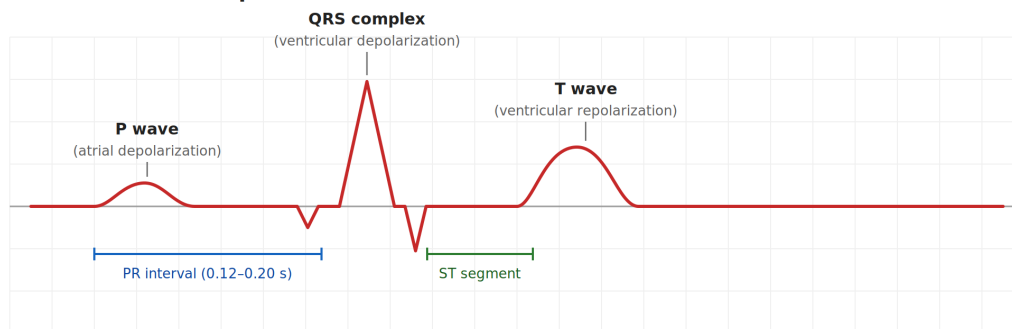
The heart beats on its own (autorhythmic); nerves only adjust the rate:

- 1 SA (sinoatrial) node — the pacemaker, in the right atrium; fires fastest (about 100/min intrinsic, slowed to about 72 by vagal tone).

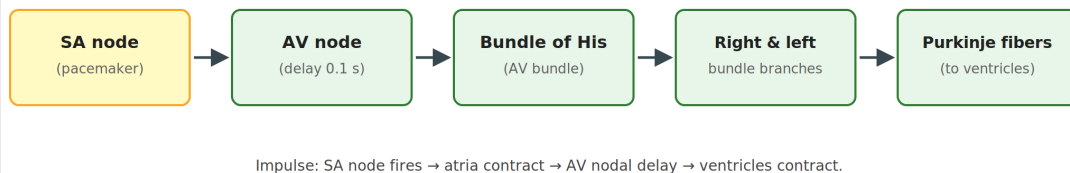
- 2 The impulse spreads across the atria (they contract) → reaches the AV (atrioventricular) node, which delays it about 0.1 s so the ventricles fill.
- 3 Down the bundle of His → right and left bundle branches → Purkinje fibres → the ventricles contract from the apex upward.

Regulation by the autonomic nervous system: sympathetic (noradrenaline, β_1 receptors) → faster rate, stronger contraction; parasympathetic (vagus, acetylcholine) → slower rate. Pharmacy links: beta-blockers (propranolol, metoprolol) slow the heart; atropine blocks vagal slowing; digoxin increases vagal tone and contractility.

A. Normal ECG complex



B. Cardiac conduction path



Conduction system of the heart: SA node to Purkinje fibres

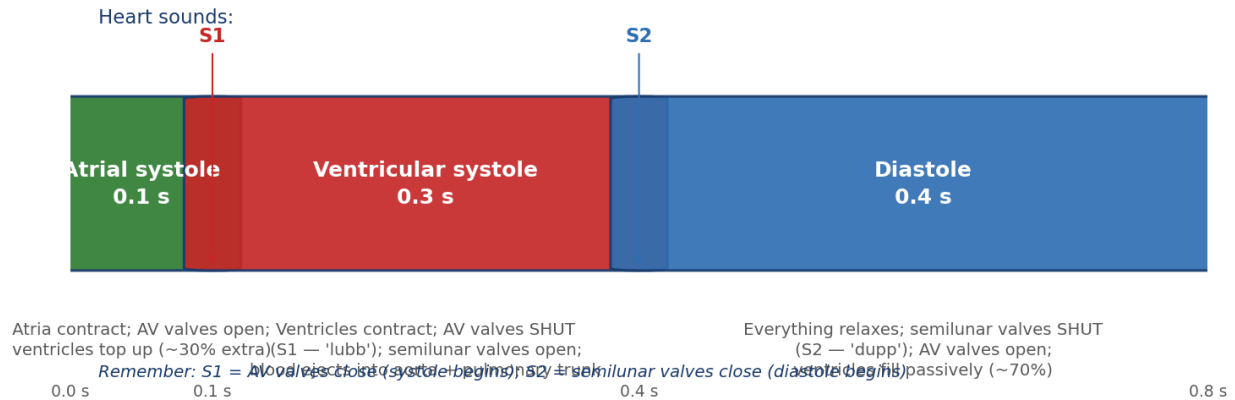
Cardiac cycle

One cycle (about 0.8 s at 72 bpm) has three stages:

- 1 Atrial systole (0.1 s) — atria contract, topping up the ventricles; AV valves open.
- 2 Ventricular systole (0.3 s) — ventricles contract; AV valves shut (first heart sound, S_1 — "lubb"); pressure opens the semilunar valves; blood ejects into the aorta and pulmonary trunk.
- 3 Diastole (0.4 s) — everything relaxes; semilunar valves shut (second heart sound, S_2 — "dupp"); AV valves open; ventricles fill passively (about 70%) plus the atrial kick (about 30%).

Heart sounds: S_1 = AV valves closing (systole begins), S_2 = semilunar valves closing (diastole begins). Murmurs are abnormal sounds from turbulent flow (valve stenosis or regurgitation).

The cardiac cycle (0.8 s at 72 bpm) — what happens when



The cardiac cycle: atrial systole, ventricular systole and diastole with S_1 and S_2

Cardiac output

Cardiac output (CO) = heart rate $\times$ stroke volume $\approx 72 \text{ bpm} \times 70 \text{ mL} \approx 5 \text{ L/min}$ at rest — the entire blood volume, every minute.

Stroke volume depends on three factors:

- Preload — venous return / end-diastolic volume; more stretch $\rightarrow$ stronger contraction (the Frank-Starling law).
- Afterload — the resistance the ventricle pumps against; high blood pressure reduces stroke volume.
- Contractility — sympathetic stimulation and drugs like digoxin increase it.

Ejection fraction (about 55–70%) = stroke volume $\div$ end-diastolic volume; the key number in heart failure assessment.

Pharmacy link: diuretics reduce preload, vasodilators reduce afterload, digoxin raises contractility — the three levers of heart-failure treatment map directly onto this equation.

Regulation of blood pressure

Blood pressure = cardiac output $\times$ peripheral resistance. Normal: about 120/80 mmHg (systolic/diastolic).

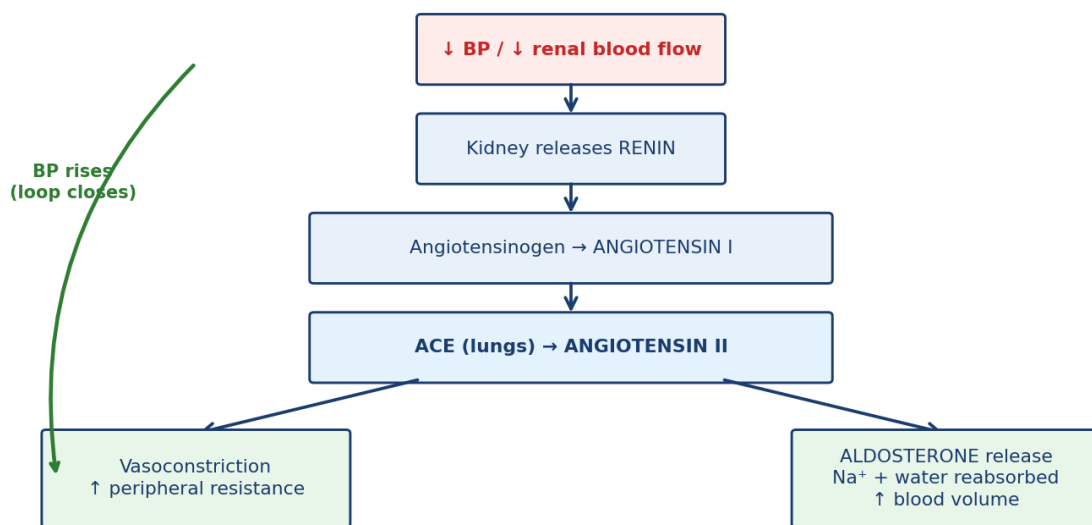
Short-term regulation (seconds–minutes):

- Baroreceptors (carotid sinus, aortic arch) sense stretch $\rightarrow$ signals via the vagus and glossopharyngeal nerves to the medulla $\rightarrow$ autonomic outflow adjusts. BP rises $\rightarrow$ vagal slowing + vasodilation; BP falls $\rightarrow$ sympathetic surge.
- Chemoreceptors respond to low O_2 , high CO_2 , and low pH.

Long-term regulation (hours-days) — the RAAS (renin-angiotensin-aldosterone system): low BP/renal flow → kidney releases renin → converts angiotensinogen to angiotensin I → ACE (lungs) makes angiotensin II → powerful vasoconstriction + aldosterone release (Na^+ and water reabsorbed, K^+ lost) → blood volume and pressure rise. Also ADH (water reabsorption) and ANP (opposes RAAS — released when the atria stretch).

Pharmacy links: ACE inhibitors (-prils) block angiotensin II formation; ARBs (-sartans) block its receptor; diuretics cut volume; beta-blockers cut renin and cardiac output.

RAAS — the long-term blood pressure control loop



Drug targets: ACE inhibitors (-prils) block formation · ARBs (-sartans) block the receptor · diuretics cut volume · beta-blockers cut renin

RAAS: the long-term blood pressure control loop

Pulse and electrocardiogram (ECG)

Pulse — the pressure wave felt where an artery runs over bone (the radial artery at the wrist is standard). Normal 60–100/min; rate, rhythm, and volume are all noted clinically. A pulse deficit (apex-radial difference) suggests atrial fibrillation.

ECG — records the heart's electrical activity via chest and limb leads:

- P wave — atrial depolarisation (contraction follows).
- QRS complex — ventricular depolarisation (the big spike; contraction follows). An abnormally wide QRS suggests bundle-branch block.
- T wave — ventricular repolarisation (recovery).
- PR interval — AV nodal delay; prolonged in heart block. ST segment — elevation signals acute MI; depression signals ischaemia.

Normal sinus rhythm: every P wave followed by a QRS, regular, 60–100/min.

Exam tip: Draw one perfect ECG complex and label P, QRS, T, the PR interval and the ST segment. Pair it with the conduction-path diagram (SA → AV → His → Purkinje). Those two diagrams together answer most Unit V short notes.

Pathophysiology of hypertension

Definition: sustained blood pressure $\geq 140/90$ mmHg (measured on more than one occasion).

Types: primary (essential) — about 90–95%, no single cause; risk factors are age, family history, obesity, high salt intake, smoking, alcohol, stress, and physical inactivity. Secondary — about 5–10%, from kidney disease, aldosteronism, Cushing's syndrome, pheochromocytoma, coarctation of the aorta, or drugs (NSAIDs, steroids, oral contraceptives).

Pathogenesis: increased cardiac output and/or peripheral resistance — via overactive sympathetic tone, RAAS overactivity, endothelial dysfunction, and salt/water retention.

Why it matters ("the silent killer"): usually no symptoms until complications appear — stroke, myocardial infarction, heart failure, chronic kidney disease, and retinopathy (target-organ damage). Malignant hypertension can cause papilloedema and encephalopathy.

Pharmacy link: lifestyle measures first; then drug classes matched to mechanism — thiazide diuretics, ACE inhibitors/ARBs, calcium-channel blockers, beta-blockers. Hypertensive emergency is treated with IV drugs (nitroprusside, labetalol).

Pathophysiology of cardiac arrhythmias

An arrhythmia is any abnormality of rate, rhythm, or conduction:

- Sinus tachycardia ($>100/\text{min}$) — fever, anxiety, exercise; sinus bradycardia ($<60/\text{min}$) — athletes, beta-blockers, hypothyroidism.
- Atrial fibrillation (AF) — chaotic atrial activity, irregularly irregular pulse, loss of the atrial kick; the danger is clot formation in the atria → stroke → needs anticoagulation (warfarin/DOACs).
- Atrial flutter — sawtooth pattern, fast regular atrial rate.
- Ventricular tachycardia / ventricular fibrillation — VF is chaotic ventricular quivering with no cardiac output — cardiac arrest; treated with immediate defibrillation.
- Heart blocks — AV nodal conduction fails; first-degree (long PR), second-degree (dropped beats), third-degree/complete (atria and ventricles beat independently — needs a pacemaker).
- Extrasystoles (ectopic beats) — premature beats from atria or ventricles; often benign.

Pharmacy link: antiarrhythmics (amiodarone, lidocaine, verapamil, adenosine) act on ion channels and conduction — which is why this unit's conduction system must be learned first.

Pathophysiology of congestive heart failure (CHF)

Definition: the heart cannot pump enough blood to meet the body's needs — not that it stops, but that it fails as a pump.

Left-sided failure — blood backs up into the lungs: dyspnoea, orthopnoea (breathless lying flat), paroxysmal nocturnal dyspnoea, pulmonary oedema (crackles), fatigue. Causes: hypertension, MI, valve disease.

Right-sided failure — blood backs up into the systemic veins: pedal oedema, jugular venous distension, enlarged tender liver (hepatomegaly), ascites. Often follows left-sided failure (via pulmonary hypertension) or lung disease (cor pulmonale).

Pathogenesis: an initial insult (MI, chronic hypertension) reduces output → compensatory mechanisms kick in — sympathetic activation and RAAS activation (retain salt/water, vasoconstrict) plus ventricular remodelling (hypertrophy, dilation). These help briefly but worsen the load long-term — a vicious cycle.

Systolic failure (reduced ejection fraction — weak squeeze) vs diastolic failure (preserved EF — a stiff ventricle that cannot fill).

Pharmacy link: treatment unloads the failing heart — diuretics (reduce preload/oedema), ACE inhibitors/ARBs + beta-blockers (block the harmful compensation — proven to prolong life), digoxin (raises contractility).

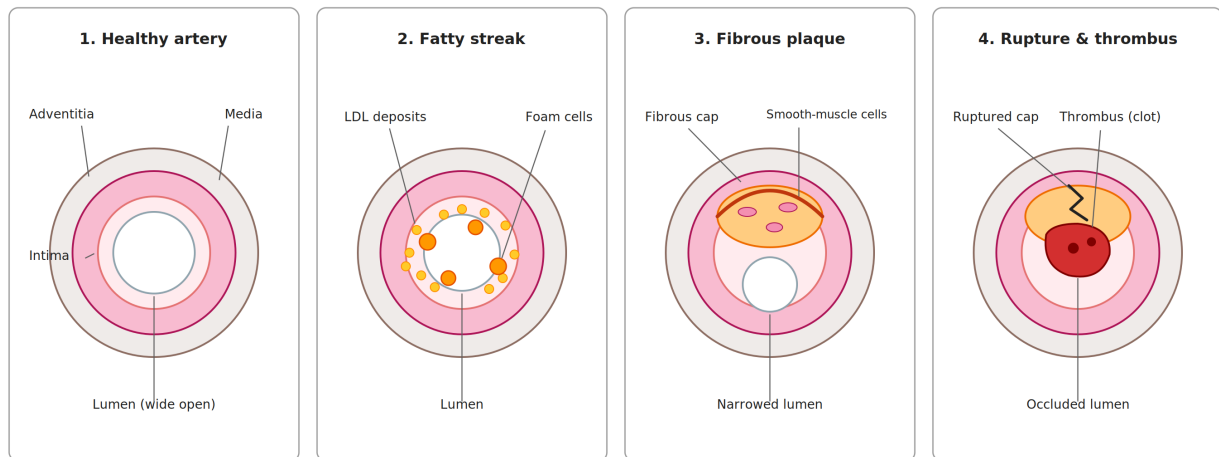
Exam tip: Left = lungs (breathlessness), right = rest of the body (oedema, JVP, liver). If you remember "left lung, right rest", the whole CHF answer writes itself.

Pathophysiology of ischemic heart disease

Atherosclerosis — the root process

- 1 Endothelial injury (hypertension, smoking, high LDL, diabetes) lets LDL into the intima.
- 2 LDL oxidises → monocytes enter, become macrophages, gorge on lipid → foam cells → fatty streak.
- 3 Smooth-muscle proliferation and collagen form a fibrous plaque with a lipid core.
- 4 Plaque rupture exposes thrombogenic material → thrombus → vessel occlusion. Stable plaques narrow the lumen (stable angina); ruptured plaques cause sudden occlusion (MI).

Atherosclerosis: Sequence of Plaque Formation



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

Atherosclerosis: from endothelial injury to plaque rupture

Angina pectoris

Chest pain from myocardial ischaemia (demand exceeds supply), relieved by rest or nitroglycerin:

- Stable angina — predictable, on exertion, relieved by rest; fixed plaque narrowing.
- Unstable angina — new, more severe, or at rest; plaque disruption without full occlusion — a warning of impending MI.
- Prinzmetal (variant) angina — coronary spasm, often at rest, can occur with normal coronaries.

Myocardial infarction (MI)

Prolonged ischaemia kills heart muscle ("heart attack") — usually plaque rupture + thrombus in a coronary artery. Features: severe crushing retrosternal chest pain radiating to the left arm/jaw, sweating, nausea, breathlessness; not relieved by rest or nitrates. Diagnosis: ECG (ST elevation in STEMI) + cardiac markers — troponin I/T (most specific, rises in 3–6 hours) and CK-MB. Complications: arrhythmias (commonest cause of early death), heart failure, cardiogenic shock, rupture. Treatment: aspirin + clopidogrel, thrombolytics (streptokinase — recall fibrinolysis from Unit III) or primary angioplasty (PCI) — "time is muscle."

Coronary artery disease (CAD)

The umbrella term for atherosclerotic disease of the coronary arteries, manifesting as angina, MI, or sudden death. Modifiable risk factors — smoking, hypertension, diabetes, dyslipidaemia, obesity, inactivity — are the prevention message pharmacists counsel on.

Very short questions (2 marks)

- 1 Trace the path of blood through the heart in one line.
- 2 What is the correct order of the heart's conduction system?

- 3 What does each wave (P, QRS, T) of a normal ECG represent?
- 4 Define cardiac output and give its normal resting value.
- 5 Why is troponin the preferred marker for myocardial infarction?

Short questions (5 marks)

- 1 Explain the cardiac cycle with reference to heart sounds.
- 2 How is blood pressure regulated in the short term and long term?
- 3 Differentiate stable angina, unstable angina and myocardial infarction.

Long questions (10 marks)

- 1 Describe the conduction system of the heart and the regulation of heartbeat by the autonomic nervous system. Add a note on ECG waves and their significance.
- 2 Describe the pathophysiology of atherosclerosis, angina, myocardial infarction and congestive heart failure, with their distinguishing clinical features.

How to approach these answers

- Very-short Q1: the single chain with valves included — the valves are what earn the second mark.
- Very-short Q2: SA node → AV node → His → bundle branches → Purkinje fibres, in that order.
- Very-short Q3: P = atrial depolarisation, QRS = ventricular depolarisation, T = ventricular repolarisation — three short phrases.
- Very-short Q4: formula ($HR \times SV$) + the 5 L/min figure.
- Very-short Q5: highly cardiac-specific and stays elevated for days — one line.
- Short Q1: three stages with timings, S1/S2 placed exactly where they occur.
- Short Q2: short-term (baroreceptors, seconds) vs long-term (RAAS, hours-days) as two subheads; draw the RAAS loop.
- Short Q3: a three-column table — trigger, pain pattern, relief, ECG/markers.
- Long Q1: intro (autorhythmic heart), 3 subheads — conduction path with diagram, ANS regulation (sympathetic vs vagus), ECG waves with a labelled complex — conclusion.
- Long Q2: intro (atherosclerosis as the root), 4 subheads — atherosclerosis sequence with diagram, angina types, MI (features, troponin, "time is muscle"), CHF (left vs right) — conclusion on modifiable risk factors.