BP104T · UNIT II

BP104T Human Anatomy, Physiology and Pathophysiology I

Unit II — Integumentary System, Skeletal System and Joints

B.Pharm Semester I · Theory

12 hours · Chapters 5–7

Pills & Python

Pharmacy • Data • AI • Healthcare

CHAPTER 5: THE INTEGUMENTARY SYSTEM — SKIN, WOUND HEALING AND DISORDERS

Unit II · ~4 hours

5.1 Introduction

The skin is the largest organ of the body — about 2 square metres in an adult — and the first thing every drug applied as a cream, ointment, or patch must cross. For a pharmacist, the skin is both a *barrier* to drug delivery and a *route* for it. This chapter covers how skin is built, how wounds heal, and what goes wrong in common skin diseases.

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

- describe the layers of the skin and its appendages;
- list the functions of the skin;
- explain the phases of wound healing;
- describe the pathophysiology of psoriasis, dermatitis, and leprosy at textbook level.

5.2 Definitions

Scientific definition: The integumentary system consists of the skin (epidermis, dermis, hypodermis) and its appendages (hair, nails, sweat and oil glands), forming the body's outer covering.

Simple explanation: The integumentary system is the body's **outer suit of armour** — it keeps germs and water out, keeps body fluids in, senses the world, and helps control temperature.

5.3 Key Terminology

Term	Meaning	Simple explanation
Epidermis	Outermost, thin layer of skin (stratified squamous epithelium)	The "paint" — waterproof outer coat
Dermis	Thick middle layer with blood vessels, nerves, glands	The "leather" — tough, living layer
Hypodermis	Fatty layer under the dermis	The "cushion" — insulation and padding
Keratin	Tough waterproof protein in skin, hair, nails	The "raincoat protein"
Melanin	Pigment made by melanocytes; gives skin colour, protects from UV	The "natural sunscreen"
Appendage	A structure attached to skin (hair, nail, gland)	The "accessories" of the skin

5.4 Detailed Explanation

5.4.1 Structure of the skin — three layers

1. Epidermis (outer, ~0.1 mm on most of the body):

- Made of **stratified squamous keratinized epithelium** — many layers of flat cells.
- Bottom layer (stratum basale) constantly divides, pushing new cells upward; surface cells die, fill with **keratin**, and shed — the whole epidermis renews roughly every month.
- Contains **melanocytes** (make melanin pigment), and no blood vessels (nourished by diffusion from the dermis).

2. Dermis (middle, tough and elastic):

- Dense connective tissue with **collagen and elastic fibres**.
- Contains blood vessels, nerve endings (touch, pain, temperature), **hair follicles**, **sweat glands** (cooling), **sebaceous (oil) glands** (keep skin supple), and arrector pili muscles (make hair "stand up" in cold — goosebumps).

3. Hypodermis / subcutaneous layer (deepest):

- **Adipose (fat) tissue** — insulation, energy store, shock absorber; anchors skin to underlying muscle. This is where subcutaneous injections are given.

Skin appendages: hair (protection, sensation), nails (protect fingertips, help grip), sweat glands — **eccrine** (all over, cooling sweat) and **apocrine** (armpits, groin, scent), sebaceous glands (oil/sebum).

5.4.2 Functions of the skin

1. **Protection** — barrier against germs, chemicals, UV rays, and water loss.
2. **Temperature regulation** — sweating cools; blood-vessel constriction conserves heat.
3. **Sensation** — touch, pressure, pain, temperature via nerve endings.
4. **Vitamin D synthesis** — sunlight converts a cholesterol-like substance in skin to vitamin D.
5. **Excretion** — small amounts of salts and urea leave in sweat.
6. **Drug route** — transdermal patches deliver drugs (e.g., nicotine, nitroglycerin) through the skin.

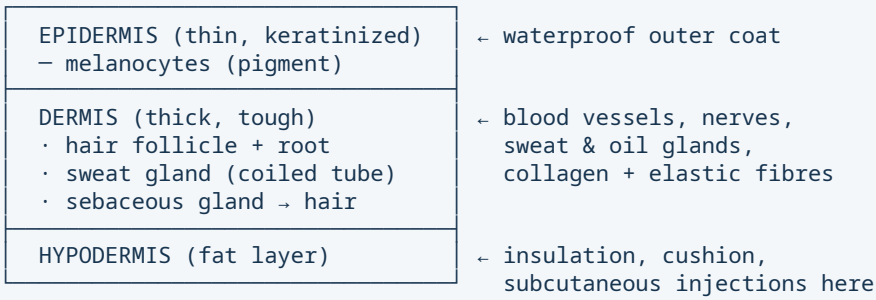
5.4.3 Wound healing — four overlapping phases

1. **Haemostasis (minutes):** blood vessels constrict; platelets form a clot — bleeding stops, a scab forms.
2. **Inflammation (days 1–4):** white blood cells clean the wound — redness, swelling, warmth; debris and germs are removed.
3. **Proliferation (days 4–21):** new tissue (**granulation tissue**) fills the wound; **fibroblasts** lay down collagen; new blood vessels grow; the wound contracts and skin cells cover the surface.
4. **Remodelling/maturation (weeks to months):** collagen reorganizes and strengthens; the scar fades but never reaches full original strength.

5.4.4 Skin disorders — pathophysiology (textbook level)

- **Psoriasis:** a chronic autoimmune condition — the immune system wrongly accelerates skin-cell production (cells mature in days instead of weeks), producing thick, red, scaly patches (plaques), commonly on elbows, knees, and scalp. It is *not* contagious.
- **Dermatitis (eczema):** inflammation of the skin — redness, itching, sometimes blisters — caused by irritants (soaps, chemicals — *contact dermatitis*), allergy, or unknown factors (*atopic dermatitis*). Scratching worsens it.
- **Leprosy (Hansen's disease):** a chronic infection caused by the bacterium *Mycobacterium leprae*, which prefers cool areas — skin and peripheral nerves. Nerve damage causes loss of sensation, so injuries go unnoticed; repeated unnoticed injuries lead to deformity. It spreads through prolonged close contact with untreated persons, is curable with multidrug therapy, and — importantly — is *not* highly contagious and *not* a curse.

5.5 Diagram — Layers of the Skin



5.6 Analogy

Skin is a **three-layer winter jacket**: the epidermis is the waterproof outer shell, the dermis is the tough insulated middle with all the wiring (nerves) and plumbing (vessels), and the hypodermis is the soft fleece lining that keeps you warm.

5.7 Pharmacy Relevance

Pharmacists dispense most skin medicines: corticosteroid creams for dermatitis, vitamin-D analogues and coal-tar preparations for psoriasis, multidrug therapy for leprosy (free under India's national programme). Counselling points — apply thin layers, don't scratch, complete the full leprosy course — come straight from this chapter. Transdermal patches are applied pharmacy: the stratum corneum is the rate-limiting barrier every patch formulation must overcome.

5.8 India Context

Leprosy was declared eliminated as a public-health problem in India in 2005, but new cases still appear and are treated free under the National Leprosy Eradication Programme (NLEP) — pharmacists must fight the stigma that it is incurable or a curse. Fungal skin infections (dermatophytosis) are extremely common in India's hot, humid climate — antifungal creams are among the most dispensed skin products.

5.9 High-Yield Points (Exam Pearls)

- 3 layers: epidermis (keratinized, no vessels) → dermis (vessels, glands, nerves) → hypodermis (fat).
- Melanocytes make melanin; keratin waterproofs.
- Wound healing: haemostasis → inflammation → proliferation → remodelling.
- Psoriasis = fast skin-cell turnover, autoimmune, scaly plaques, not contagious.
- Leprosy = *Mycobacterium leprae*, attacks skin + nerves, curable with multidrug therapy.

5.10 Common Confusions

- **Dermis vs epidermis:** epidermis = thin outer (no blood vessels); dermis = thick middle (has vessels, nerves, glands).
- **Eccrine vs apocrine sweat glands:** eccrine = everywhere, cooling; apocrine = armpits/groin, scent-related.
- **Psoriasis vs dermatitis:** psoriasis = thick scaly plaques from rapid cell turnover; dermatitis = itchy inflammation from irritants/allergy.

5.11 Common Mistakes

- Saying the epidermis has blood vessels — it does not; it is nourished by diffusion from the dermis.
- Calling leprosy "highly contagious" — it spreads only through prolonged close contact and is curable.
- Mixing up the order of wound-healing phases — haemostasis always comes first.

5.12 Mnemonic

"HIPR" for wound healing: **H**aemostasis → **I**nflammation → **P**roliferation → **R**emodelling. ("A wound needs HIPR care.")

5.13 Exam-Oriented Summary

The integumentary system is the skin plus hair, nails, and glands. Skin has three layers: the keratinized, vessel-free epidermis (with melanocytes); the tough dermis (vessels, nerves, hair follicles, sweat and sebaceous glands); and the fatty hypodermis. Functions include protection, temperature control, sensation, vitamin D synthesis, and drug delivery. Wounds heal in four phases — haemostasis, inflammation, proliferation, remodelling. Psoriasis is an autoimmune rapid-turnover disease with scaly plaques; dermatitis is skin inflammation from irritants or allergy; leprosy is a curable *Mycobacterium leprae* infection of skin and nerves.

5.14 One-Minute Revision

- Layers: epidermis (no vessels) / dermis (vessels + glands) / hypodermis (fat).
- Healing: HIPR — clot, clean, fill, strengthen.
- Psoriasis = fast + scaly; dermatitis = itchy inflammation; leprosy = nerve-damaging, curable.

5.15 Questions

MCQs

1. Which skin layer contains blood vessels and sweat glands? (a) epidermis (b) dermis (c) hypodermis (d) stratum corneum — **Ans: (b)**
2. Melanin is produced by: (a) keratinocytes (b) melanocytes (c) fibroblasts (d) Langerhans cells — **Ans: (b)**
3. The correct order of wound healing is: (a) inflammation → haemostasis → proliferation → remodelling (b) haemostasis → inflammation → proliferation → remodelling (c) proliferation → inflammation → haemostasis → remodelling (d) haemostasis → proliferation → inflammation → remodelling — **Ans: (b)**
4. Psoriasis is characterized by: (a) bacterial infection of hair follicles (b) rapid skin-cell turnover with scaly plaques (c) loss of skin pigment (d) fluid-filled blisters — **Ans: (b)**
5. Leprosy is caused by: (a) a virus (b) *Mycobacterium leprae* (c) a fungus (d) an autoimmune reaction — **Ans: (b)**

Viva

1. Name the three layers of skin with one feature each.
2. Why does the epidermis not bleed when lightly scratched?
3. List the four phases of wound healing.
4. Why is psoriasis not contagious?
5. Which nerve-related symptom is characteristic of leprosy?

Application-based

1. A pharmacist is asked why a nitroglycerin patch works but a large protein drug cannot be given by patch. Explain using skin structure. *(Ans: small lipophilic molecules like nitroglycerin can cross the keratinized stratum corneum barrier; large protein molecules cannot pass this barrier, so they need injection.)*
2. A leprosy patient stops multidrug therapy after 2 months because skin patches faded. What would you counsel, using the pathophysiology you know? *(Ans: the bacteria, especially in nerves, are not yet eliminated; stopping early risks relapse and drug resistance — the full course must be completed even after visible improvement.)*

5.16 References

- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Skin chapters.)
- Tortora GJ, Derrickson B. Principles of Anatomy and Physiology. (Chapter 5.)
- Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology. (Skin disorder sections.)

CHAPTER 6: THE SKELETAL SYSTEM, MUSCLES AND JOINTS

Unit II · ~5 hours

6.1 Introduction

The skeleton is the body's framework — 206 bones that hold you up, protect your brain and heart, and make blood cells. But bones alone can't move; **skeletal muscles** pull on them across **joints** to produce every movement from walking to writing. For pharmacists, this system matters daily: calcium and vitamin D for bones, muscle relaxants, anti-inflammatory drugs for joints.

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

- state the divisions of the skeletal system and the functions of bones;
- classify bones by shape with examples;
- describe the salient features of the axial and appendicular skeleton;
- explain the sliding-filament theory of muscle contraction in simple steps;
- describe the neuromuscular junction;
- classify joints structurally and functionally with examples.

6.2 Definitions

Scientific definitions:

- **Skeleton** — the framework of bones and cartilage supporting and protecting the body.
- **Muscle contraction** — the shortening of muscle fibres generating force, explained by the sliding-filament theory (thin actin filaments slide past thick myosin filaments).
- **Joint (articulation)** — the point where two or more bones meet, allowing (or restricting) movement.
- **Neuromuscular junction** — the synapse where a motor nerve meets a skeletal muscle fibre, releasing acetylcholine to trigger contraction.

Simple explanation: Bones are the *poles of a tent*, joints are the *hinges*, and muscles are the *ropes that pull* — together they hold the tent up and let it move.

6.3 Key Terminology

Term	Meaning	Simple explanation
Axial skeleton	Skull, vertebral column, rib cage (80 bones)	The "central pillar" — head and trunk
Appendicular skeleton	Limbs and their girdles (126 bones)	The "attachments" — arms, legs, shoulders, hips
Diaphysis	The shaft of a long bone	The "handle" of the bone
Epiphysis	The ends of a long bone	The "knobs" at each end
Articular cartilage	Smooth cartilage covering joint surfaces	The "Teflon coating" for friction-free movement
Actin / Myosin	Thin and thick contractile proteins in muscle	The "ropes" (actin) and "oars" (myosin) of contraction
Sarcomere	The repeating contractile unit of a muscle fibre	The "engine cylinder" — smallest unit that shortens
Ligament	Connective tissue joining bone to bone	The "seatbelt" stabilizing a joint
Tendon	Connective tissue joining muscle to bone	The "tow rope" transmitting muscle pull

6.4 Detailed Explanation

6.4.1 Divisions and functions of the skeletal system

- **Axial skeleton (80 bones):** skull (cranium + facial bones — protects the brain), vertebral column (33 vertebrae — protects the spinal cord), thoracic cage (12 rib pairs + sternum — protects heart and lungs).
- **Appendicular skeleton (126 bones):** pectoral (shoulder) girdle + upper limbs; pelvic (hip) girdle + lower limbs.

Functions of bones: support (body framework), protection (skull → brain, ribs → heart/lungs), movement (levers for muscles), mineral storage (calcium, phosphorus), blood-cell formation (**haemopoiesis** in red bone marrow), fat storage (yellow marrow).

6.4.2 Types of bones (by shape)

Type	Description	Examples
Long	Longer than wide; shaft + two ends	Femur (thigh), humerus (arm), tibia
Short	Cube-shaped	Wrist (carpals), ankle (tarsals)
Flat	Thin, flat, often curved	Skull bones, ribs, sternum, scapula
Irregular	Complex shapes	Vertebrae, facial bones
Sesamoid	Small, embedded in tendons	Patella (kneecap)

6.4.3 Salient features — bones worth knowing

- **Skull:** cranium (8 bones) protects the brain; facial bones (14) form the face; the **mandible** (lower jaw) is the only movable skull bone.
- **Vertebral column:** 7 cervical (neck) + 12 thoracic (chest) + 5 lumbar (lower back) + 5 fused sacral + 4 fused coccygeal = 33 vertebrae; the discs between them act as shock absorbers.
- **Thoracic cage:** 12 rib pairs + sternum (breastbone); protects heart and lungs and moves during breathing.
- **Upper limb:** humerus → radius + ulna → carpals (wrist) → metacarpals (palm) → phalanges (fingers).
- **Lower limb:** femur (longest, strongest bone) → patella → tibia + fibula → tarsals (ankle) → metatarsals → phalanges.
- **Pectoral girdle:** clavicle (collarbone) + scapula (shoulder blade). **Pelvic girdle:** two hip bones fused to the sacrum — supports body weight and protects pelvic organs.

6.4.4 Organization of skeletal muscle

A skeletal muscle (e.g., biceps) is a bundle of **muscle fibres** (long cells). Each fibre contains **myofibrils**, made of repeating units called **sarcomeres**. Each sarcomere has **thin filaments (actin)** and **thick filaments (myosin)** arranged in an overlapping pattern — this overlap creates the striations (stripes) seen under the microscope.

6.4.5 Physiology of muscle contraction — sliding-filament theory (basic level)

1. A nerve signal reaches the muscle (via the neuromuscular junction — see below).
2. **Calcium ions (Ca^{2+})** are released inside the muscle fibre.
3. Calcium uncovers the binding sites on actin.
4. **Myosin heads** (like tiny oars) attach to actin, pull (the "power stroke"), detach, and reattach — using ATP for each stroke.
5. Actin filaments **slide past** myosin toward the centre; the sarcomere shortens; the whole muscle shortens = **contraction**.
6. When the signal stops, calcium is pumped back and the muscle **relaxes**.

Key point: the filaments don't shorten — they *slide past each other*. That's why it's called the **sliding-filament** theory.

6.4.6 Neuromuscular junction (NMJ)

Where the motor nerve meets the muscle fibre:

1. The nerve impulse arrives at the nerve ending.
2. The nerve releases the neurotransmitter **acetylcholine (ACh)** into the synaptic gap.
3. ACh binds receptors on the muscle membrane, opening ion channels.
4. An electrical signal spreads over the muscle fibre → calcium release → contraction (steps above).
5. The enzyme **acetylcholinesterase** quickly breaks down ACh so the muscle can relax — ready for the next signal.

Pharmacy link: drugs like neostigmine (used in myasthenia gravis) block acetylcholinesterase, prolonging ACh action and strengthening weak muscles.

6.4.7 Joints — classification

A. Structural classification (by material joining the bones):

Type	Joining material	Movement	Examples
Fibrous	Dense connective tissue	Immovable (synarthrosis)	Skull sutures
Cartilaginous	Cartilage	Slightly movable (amphiarthrosis)	Between vertebrae; pubic symphysis
Synovial	Joint cavity with synovial fluid	Freely movable (diarthrosis)	Shoulder, hip, knee, elbow

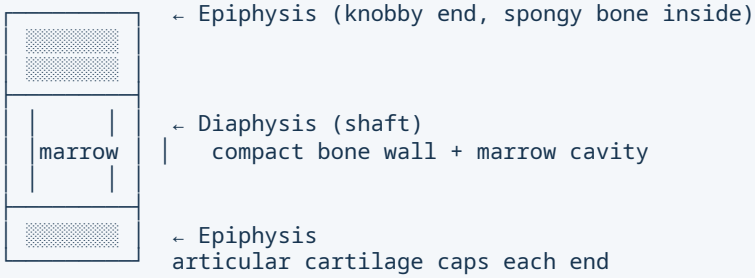
B. Functional classification (by amount of movement):

- **Synarthrosis** — immovable (skull sutures).
- **Amphiarthrosis** — slightly movable (vertebral joints).
- **Diarthrosis** — freely movable (all synovial joints).

Synovial joint subtypes (shapes decide the movement): ball-and-socket (shoulder, hip — movement in all directions), hinge (elbow, knee — like a door hinge), pivot (neck — rotation), gliding (wrist/ankle — sliding), saddle (thumb), condyloid (knuckle).

6.5 Diagrams

Long bone structure:



Sliding filament (sarcomere shortening):

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RELAXED:    |——actin——→|←—myosin—→|←——actin——|
                    (wide overlap zone)

CONTRACTED: |—actin—→|←myosin→|←—actin—|
                    (actin slid inward — sarcomere shorter)
  → filaments SLIDE; they do NOT shorten themselves
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6.6 Analogy

A muscle fibre is a **tug-of-war team**: actin ropes are pulled hand-over-hand by myosin "rowers" — each stroke (powered by ATP) drags the rope a little further, and the two ends of the rope (the sarcomere ends) come closer together.

6.7 Pharmacy Relevance

Bone and joint drugs are everyday pharmacy: calcium + vitamin D supplements for bone strength; bisphosphonates for osteoporosis (Chapter 7); NSAIDs (ibuprofen, diclofenac) for joint pain and inflammation; muscle relaxants for spasms. Understanding the NMJ explains both myasthenia gravis treatment (neostigmine) and why anaesthetic muscle relaxants work. Pharmacists also counsel on posture, exercise, and calcium-rich diet for bone health.

6.8 India Context

Vitamin D deficiency is widespread in India despite abundant sunshine (indoor lifestyles, darker skin needing longer exposure, clothing practices) — pharmacists frequently dispense vitamin D3 (cholecalciferol) supplements. Road-traffic fractures are a major burden; calcium, painkillers, and antibiotics for open fractures are core dispensing items in Indian hospitals.

6.9 High-Yield Points (Exam Pearls)

- Axial = 80 (skull, spine, ribs); appendicular = 126 (limbs + girdles). Total 206.
- Longest bone: femur. Only movable skull bone: mandible.
- Vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused), 4 coccygeal.
- Sliding filament: actin slides past myosin; myosin heads use ATP; Ca^{2+} starts it.
- NMJ transmitter: acetylcholine; broken down by acetylcholinesterase.
- Joints: fibrous (immovable, skull), cartilaginous (slightly movable, vertebrae), synovial (freely movable, knee/shoulder).

6.10 Common Confusions

- **Tendon vs ligament** (again — examiners love it): tendon = muscle → bone; ligament = bone → bone.
- **Actin vs myosin:** actin = thin filament; myosin = thick filament with heads that pull.
- **Axial vs appendicular:** axial = central axis (head + trunk); appendicular = appendages (limbs).
- **Synarthrosis/amphiarthrosis/diarthrosis** = functional (how much movement); **fibrous/cartilaginous/synovial** = structural (what joins them).

6.11 Common Mistakes

- Saying muscles "push" bones — muscles only *pull*; movement in the opposite direction needs the opposing muscle.
- Writing that filaments shorten during contraction — they slide; the *sarcomere* shortens.
- Counting 206 bones but forgetting the total splits as 80 + 126.

6.12 Mnemonic

"7-12-5" for vertebrae: **7** cervical (neck), **12** thoracic, **5** lumbar — "breakfast at 7, lunch at 12, dinner at 5, down the spine." For joints: **"FCS — Fixed, Curved (slightly), Swinging free"** (Fibrous, Cartilaginous, Synovial).

6.13 Exam-Oriented Summary

The 206-bone skeleton divides into the axial (80: skull, vertebral column, thoracic cage) and appendicular (126: girdles and limbs) divisions, serving support, protection, movement, mineral storage, and blood-cell formation. Bones are classified as long, short, flat, irregular, or sesamoid. Skeletal muscle fibres contain sarcomeres of actin (thin) and myosin (thick) filaments; contraction follows the sliding-filament theory — Ca^{2+} release, myosin power strokes using ATP, actin sliding inward. The neuromuscular junction uses acetylcholine (cleared by acetylcholinesterase) to trigger contraction. Joints are classified structurally (fibrous, cartilaginous, synovial) and functionally (synarthrosis, amphiarthrosis, diarthrosis).

6.14 One-Minute Revision

- 206 bones: 80 axial + 126 appendicular. Femur longest.
- Muscle: Ca^{2+} → myosin pulls actin → sarcomere shortens (filaments slide, don't shrink).
- NMJ: acetylcholine → contraction; acetylcholinesterase → relaxation.
- Joints: fibrous-fixed, cartilaginous-slight, synovial-free.

6.15 Questions

MCQs

1. The total number of bones in the adult human skeleton is: (a) 196 (b) 206 (c) 216 (d) 226 — **Ans: (b)**
2. In the sliding-filament theory, the protein with cross-bridge heads is: (a) actin (b) myosin (c) troponin (d) titin — **Ans: (b)**
3. The neurotransmitter at the neuromuscular junction is: (a) dopamine (b) serotonin (c) acetylcholine (d) GABA — **Ans: (c)**
4. The joint between vertebrae is: (a) fibrous (b) cartilaginous (c) synovial (d) ball-and-socket — **Ans: (b)**
5. Which bone is classified as sesamoid? (a) femur (b) scapula (c) patella (d) sternum — **Ans: (c)**

Viva

1. Name the two divisions of the skeleton with bone counts.
2. List the five bone shapes with one example each.
3. What is a sarcomere?
4. Why is it called the "sliding" filament theory?
5. Give one example each of synarthrosis, amphiarthrosis, and diarthrosis.

Application-based

1. Myasthenia gravis patients have antibodies that destroy acetylcholine receptors at the NMJ. Explain their muscle weakness and why neostigmine (an acetylcholinesterase inhibitor) helps. *(Ans: fewer receptors → weaker response to ACh → weakness; neostigmine prevents ACh breakdown, so more ACh stays in the gap longer, compensating for fewer receptors.)*
2. A weightlifter's biceps enlarge with training. Is this hypertrophy or hyperplasia, and why can't heart muscle undergo hyperplasia the same way skeletal-adjacent tissues do? *(Ans: hypertrophy — existing fibres enlarge (skeletal muscle fibres don't divide); cardiac muscle also hypertrophies under load for the same reason — its cells have very limited division capacity.)*

6.16 References

- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Muscle physiology chapters.)
- Tortora GJ, Derrickson B. Principles of Anatomy and Physiology. (Chapters 6–9.)
- Guyton AC, Hall JE. Textbook of Medical Physiology. (Muscle contraction chapters.)

CHAPTER 7: DISEASES OF BONES AND JOINTS — RHEUMATOID ARTHRITIS, OSTEOPOROSIS, GOUT

Unit II · ~3 hours

7.1 Introduction

Bones and joints can fail in three classic ways that every pharmacist must recognize: the immune system attacking joints (rheumatoid arthritis), bones becoming fragile (osteoporosis), and crystals inflaming joints (gout). These three diseases account for a huge share of painkiller, steroid, and calcium prescriptions — understanding their mechanisms tells you *why* each drug is chosen.

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

- describe the pathophysiology of rheumatoid arthritis, osteoporosis, and gout in simple steps;
- distinguish the three diseases by cause, joints affected, and key features;
- explain the rationale of drug therapy for each (at mechanism level);
- counsel patients on lifestyle measures for each condition.

7.2 Definitions

Scientific definitions:

- **Rheumatoid arthritis (RA)** — a chronic autoimmune inflammatory disease primarily attacking synovial joints, causing pain, swelling, and eventual joint deformity.
- **Osteoporosis** — a condition of reduced bone mass and deteriorated bone structure, making bones fragile and fracture-prone.
- **Gout** — an inflammatory arthritis caused by deposition of uric acid (monosodium urate) crystals in joints, due to high blood uric acid levels.

Simple explanation: In RA the body's *defence army attacks its own joints*; in osteoporosis the bones become *hollow and brittle like chalk*; in gout *sharp salt-like crystals* form inside joints and cause sudden, severe pain.

7.3 Key Terminology

Term	Meaning	Simple explanation
Autoimmune	Immune system attacking the body's own tissues	"Friendly fire" by the defence army
Synovium	The membrane lining a synovial joint, making lubricating fluid	The joint's "oil gland"
Pannus	Abnormal inflamed synovial tissue that invades joint structures in RA	"Invading weed" that eats the joint
Osteoblast	Bone-building cell	The "bricklayer"
Osteoclast	Bone-dissolving cell	The "demolition worker"
Uric acid	Waste product of purine breakdown, excreted by kidneys	A waste chemical that can crystallize
Tophus (plural: tophi)	A lump of urate crystal deposits under skin/in joints in chronic gout	"Chalky lumps" of stored crystals

7.4 Detailed Explanation

7.4.1 Rheumatoid arthritis — pathophysiology (step by step)

1. **Trigger:** in a genetically susceptible person, an unknown trigger (possibly infection) confuses the immune system.
2. **Autoimmune attack:** immune cells target the **synovium** (joint lining) as if it were foreign.
3. **Inflammation:** the synovium swells with immune cells; inflammatory chemicals (cytokines like TNF-α) flood the joint → pain, warmth, swelling, morning stiffness.
4. **Pannus formation:** the inflamed synovium thickens into an aggressive tissue (**pannus**) that grows over the cartilage.
5. **Destruction:** pannus enzymes eat away cartilage and then bone; ligaments weaken.
6. **Deformity:** joints become unstable and deformed (classic hand deformities); the process is **symmetrical** — both hands, both wrists.

Key features: chronic, symmetrical small-joint arthritis (hands, wrists, feet), morning stiffness lasting over an hour, systemic features (fatigue, anaemia). Blood often shows **rheumatoid factor** (an autoantibody).

Treatment rationale (mechanism level): NSAIDs and corticosteroids reduce inflammation and pain; **DMARDs** (disease-modifying drugs like methotrexate) suppress the immune attack to slow destruction; biologic agents block specific cytokines (e.g., TNF-α blockers).

7.4.2 Osteoporosis — pathophysiology (step by step)

1. **Normal bone remodelling:** throughout life, **osteoclasts** dissolve old bone and **osteoblasts** build new bone — a balanced cycle.
2. **Imbalance:** after menopause (oestrogen fall), in old age, or with low calcium/vitamin D, steroids, or inactivity, resorption **outpaces** formation.
3. **Bone loss:** bone mass and the internal scaffolding (trabeculae) thin out — bones look "porous" (hence *osteo-porosis*).
4. **Fragility:** bones break with minor trauma — classic sites: **hip, spine (vertebral crush), wrist**.
5. **Silent disease:** there is usually no pain until a fracture occurs — often discovered after a fall or as height loss/stooped posture from spinal fractures.

Key features: painless bone loss; fractures after trivial injury; risk factors — postmenopausal women, elderly, low calcium/vitamin D, smoking, alcohol, long-term steroid use, physical inactivity.

Treatment rationale: calcium + vitamin D (raw materials); **bisphosphonates** (e.g., alendronate — slow osteoclasts); weight-bearing exercise; fall prevention.

7.4.3 Gout — pathophysiology (step by step)

1. **High uric acid (hyperuricaemia):** the body makes too much uric acid (purine-rich foods, alcohol, obesity) or the kidneys excrete too little.
2. **Crystal formation:** when blood uric acid exceeds its solubility limit, needle-shaped **monosodium urate crystals** deposit in joints and tissues.
3. **Acute attack:** immune cells try to "eat" the crystals but are injured by their sharp edges → sudden, severe inflammation — typically the **big toe joint at night** (podagra): red, hot, swollen, exquisitely painful.
4. **Chronic gout:** repeated attacks deposit crystal lumps (**tophi**) in joints, ears, and tendons; chronic joint damage follows.

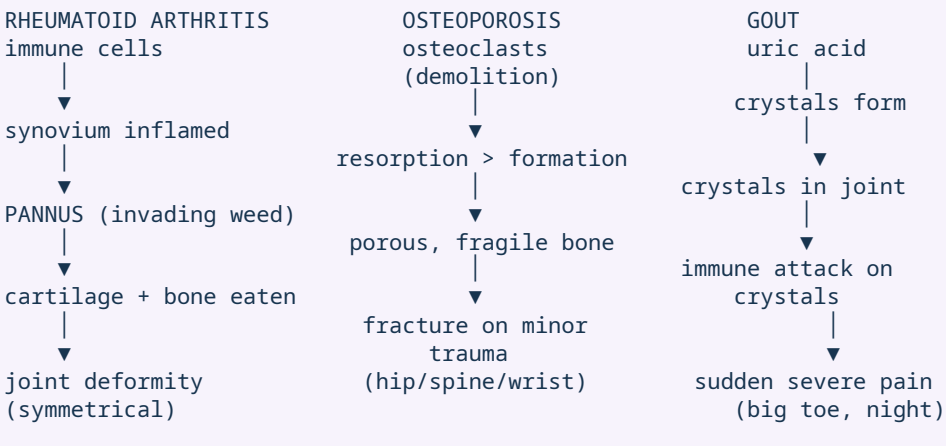
Key features: sudden night-time attacks, big-toe first joint most classic; triggered by alcohol, rich food, dehydration, certain diuretics.

Treatment rationale: acute attack — NSAIDs or colchicine (calms the crystal-triggered inflammation); long-term — **allopurinol** (reduces uric acid production) or uricosurics (increase excretion); lifestyle — less alcohol and purine-rich food, more water.

7.4.4 Comparison table — the three diseases at a glance

Feature	Rheumatoid arthritis	Osteoporosis	Gout
Basic nature	Autoimmune inflammation	Bone thinning	Crystal deposition
Main target	Synovial joints (symmetrical, small joints)	Whole skeleton (hip, spine, wrist)	Joints (big toe classic)
Pain pattern	Chronic ache + morning stiffness	Silent until fracture	Sudden severe attacks
Cause	Immune attack on synovium	Resorption > formation	Uric acid crystals
Key drugs	NSAIDs, steroids, methotrexate (DMARD), TNF blockers	Calcium + vitamin D, bisphosphonates	NSAIDs/colchicine (acute), allopurinol (chronic)

7.5 Diagram — The Three Disease Mechanisms



7.6 Analogy

Think of a building: RA is **vandals smashing the hinges** (joints) from inside; osteoporosis is **termites hollowing the wooden beams** (bones) until they snap under normal weight; gout is **broken glass scattered on the floor** (crystals) — every step is agony.

7.7 Pharmacy Relevance

These three diseases fill prescriptions daily: NSAIDs and steroids for RA flares, methotrexate requiring pharmacist counselling (weekly dosing, folic acid, liver monitoring), calcium/vitamin D and weekly alendronate (must be taken on an empty stomach, sitting/standing upright — key counselling), and allopurinol started *after* the acute gout attack settles. Pharmacists also spot drug-induced risks — long-term steroids causing osteoporosis, diuretics triggering gout.

7.8 India Context

Osteoporosis is underdiagnosed in India — many elderly hip fractures follow trivial falls at home; pharmacists can promote calcium, vitamin D, and fall-proofing. Gout is rising with changing diets and alcohol use. RA patients in India often rely on long-term steroids without DMARDs due to cost — pharmacists should encourage proper rheumatology follow-up and never stop steroids abruptly.

7.9 High-Yield Points (Exam Pearls)

- RA = autoimmune, symmetrical small joints, pannus, morning stiffness; drug: methotrexate (DMARD).
- Osteoporosis = resorption > formation; silent till fracture (hip/spine/wrist); drug: bisphosphonates + calcium/vitamin D.
- Gout = urate crystals; sudden night pain, big toe (podagra); acute: NSAID/colchicine; chronic: allopurinol.
- Osteoblast builds, osteoclast dissolves.

7.10 Common Confusions

- **Rheumatoid arthritis vs osteoarthritis:** RA = autoimmune, symmetrical, small joints, morning stiffness; osteoarthritis = wear-and-tear, asymmetrical, weight-bearing joints (knee, hip), worse with use.
- **Osteoporosis vs osteomalacia:** osteoporosis = *less* bone (quantity); osteomalacia = *soft* bone from poor mineralization (vitamin D deficiency in adults).
- **Gout vs pseudogout:** gout = urate crystals; pseudogout = calcium pyrophosphate crystals (knee, elderly) — different crystals, different disease.

7.11 Common Mistakes

- Starting allopurinol *during* an acute gout attack — it can worsen the attack; start it after the attack settles.
- Saying osteoporosis is painful — it is silent until fracture.
- Confusing DMARDs (slow disease, take weeks to act) with NSAIDs (quick pain relief).

7.12 Mnemonic

"R-O-G": **R**A = **R**ogue immunity; **O**steoporosis = **O**utpaced (resorption beats formation); **G**out = **G**lass-like crystals.

7.13 Exam-Oriented Summary

Rheumatoid arthritis is a chronic autoimmune attack on the synovium producing inflammation, pannus, cartilage/bone destruction, and symmetrical deformity (treated with NSAIDs, steroids, DMARDs like methotrexate). Osteoporosis is bone loss from resorption exceeding formation — silent until hip, spine, or wrist fracture (treated with calcium, vitamin D, bisphosphonates). Gout is urate-crystal arthritis causing sudden severe attacks, classically the big toe at night (acute: NSAIDs/colchicine; long-term: allopurinol).

7.14 One-Minute Revision

- RA: autoimmune → pannus → deformity; methotrexate.
- Osteoporosis: less bone, silent, fractures; bisphosphonates.
- Gout: urate crystals, big toe at night; colchicine acute, allopurinol chronic.

7.15 Questions

MCQs

1. Pannus formation is characteristic of: (a) osteoporosis (b) gout (c) rheumatoid arthritis (d) osteoarthritis — **Ans: (c)**
2. The classic first joint affected in gout is the: (a) knee (b) big toe (c) shoulder (d) hip — **Ans: (b)**
3. Osteoporosis results from: (a) excess bone formation (b) resorption exceeding formation (c) urate deposition (d) autoimmune attack — **Ans: (b)**
4. Allopurinol acts by: (a) reducing inflammation (b) reducing uric acid production (c) building bone (d) suppressing all immunity — **Ans: (b)**
5. Bisphosphonates are used in: (a) gout (b) rheumatoid arthritis (c) osteoporosis (d) anaemia — **Ans: (c)**

Viva

1. Define pannus.
2. Why is osteoporosis called a "silent disease"?
3. What is podagra?
4. Name one acute and one long-term drug for gout.
5. Differentiate osteoblast and osteoclast.

Application-based

1. A 65-year-old woman on long-term steroids for asthma suffers a hip fracture after slipping in the bathroom. Connect the drug history to the fracture using pathophysiology, and list two preventive measures a pharmacist could have suggested. *(Ans: chronic steroids accelerate bone resorption → osteoporosis → fracture on trivial trauma; prevention: calcium + vitamin D supplementation, bone-density monitoring, fall-proofing the home.)*
2. A patient with a first gout attack is prescribed allopurinol immediately along with an NSAID. Is the timing correct? Justify. *(Ans: the NSAID for the acute attack is correct, but allopurinol should be started after the attack fully settles — starting during acute inflammation can mobilize crystals and prolong/worsen the attack.)*

7.16 References

- Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology. (Musculoskeletal chapters.)
- Sembulingam K, Sembulingam P. Essentials of Medical Physiology. (Bone physiology sections.)
- Tortora GJ, Derrickson B. Principles of Anatomy and Physiology. (Chapters 6–9.)

END MATTER — UNITS I & II

Unit Infographic Concept — "The Body in Seven Chapters"

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┌──────────────────────────────────────────────────────┐
│  BP104T · UNITS I & II · 24 HOURS · 7 CHAPTERS       │
├──────────────────────────────────────────────────────┤
│ Ch 1  BODY BLUEPRINT   structure→function→           │
│                            disease; homeostasis      │
│ Ch 2  THE CELL         membrane transport,           │
│                            division, signalling      │
│ Ch 3  TISSUES          4 types: cover, connect,      │
│                            move, signal              │
│ Ch 4  CELL INJURY      adapt or die: necrosis        │
│                            vs apoptosis              │
│ Ch 5  SKIN             3 layers; HIPR healing;       │
│                            psoriasis/leprosy         │
│ Ch 6  BONES+MUSCLES    206 bones; sliding            │
│                        filament; joints FCS          │
│ Ch 7  BONE DISEASES    RA / osteoporosis / gout      │
│                            (R-O-G)                   │
├──────────────────────────────────────────────────────┤
│  ONE PRINCIPLE: form decides function —              │
│  and every drug acts on cells, tissues,              │
│  or systems you now know.                            │
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Combined References

1. Sembulingam K, Sembulingam P. Essentials of Medical Physiology.
2. Tortora GJ, Derrickson B. Principles of Anatomy and Physiology.
3. Guyton AC, Hall JE. Textbook of Medical Physiology.
4. Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology.