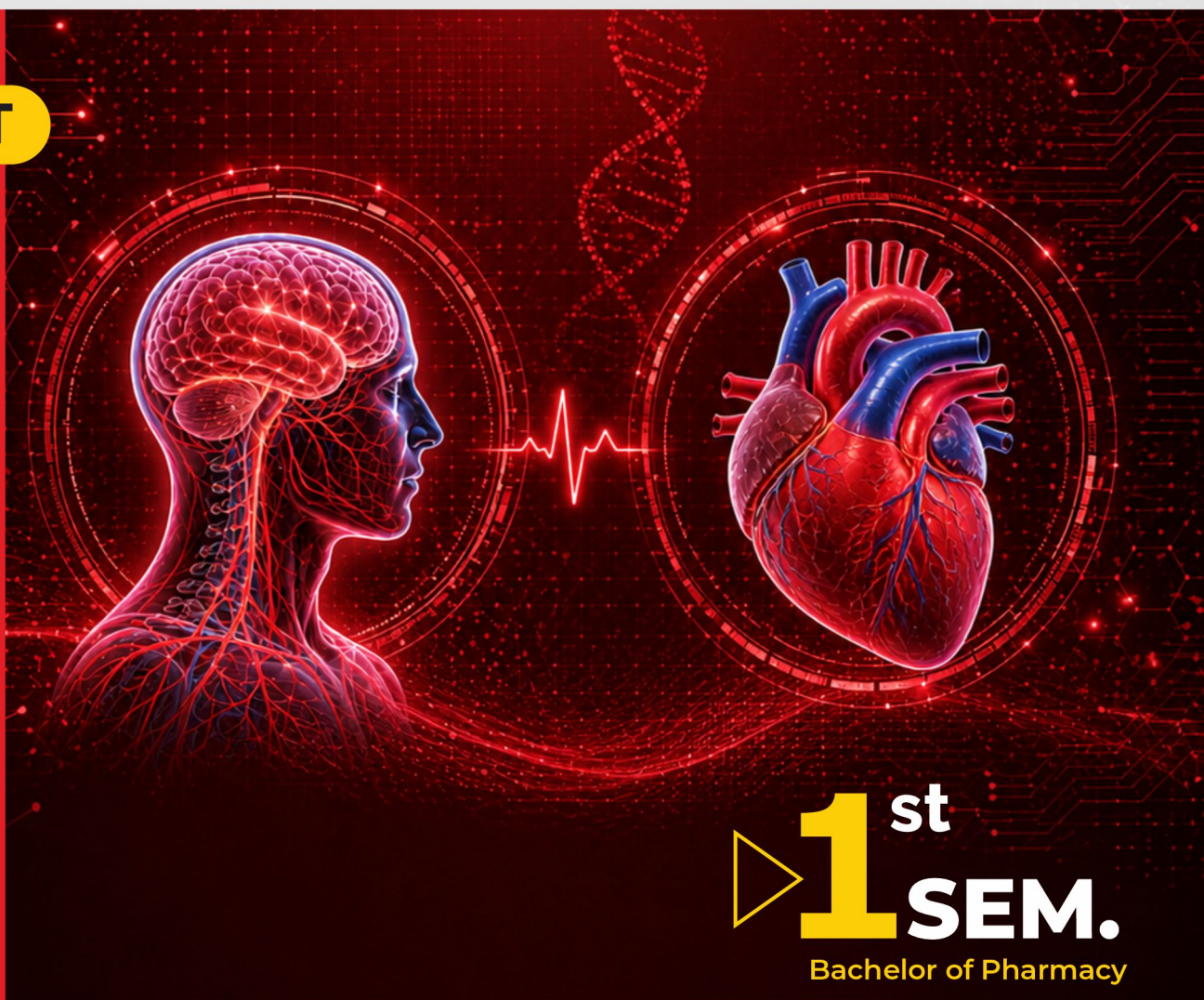


A Text Book of

HUMAN ANATOMY, PHYSIOLOGY AND PATHOPHYSIOLOGY - I

As per the New PCI Syllabus **NEP 2020**

BP104T



1st SEM.

Bachelor of Pharmacy



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First Semester

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*Dedicated
to the....*

MOTHER
Who Shaped Our World



Late Dr. Mridula Jaiswal
Educationalist

Our Inspiration & Best Teacher

Dr. Peeyush Jaiswal
Director,
GPAT Discussion Center

PREFACE

It gives us immense pleasure to present **Human Anatomy Physiology and Pathophysiology–I**, prepared in accordance with the latest syllabus prescribed by the **Pharmacy Council of India (PCI)** under the **National Education Policy (NEP) 2020**, for the First semester of Bachelor of Pharmacy programme. This book has been designed to provide pharmacy students with a clear understanding of the structure and functions of the human body.

Human Anatomy Physiology and Pathophysiology-I is vital for the further studies of Pharmacology, Pharmacotherapeutics, Clinical Pharmacy, Pharmacognosy, and Pharmacy Practice. A sound understanding of this subject is essential for comprehending disease mechanisms, drug actions, and therapeutic interventions.

The book covers all the prescribed units in a logical sequence, including the Introduction to human body and cellular adaptation, Integumentary system and wound healing, Skeletal system and joints, Body fluids, blood, lymphatic system, Inflammation and repair, Hematological diseases, Peripheral nervous system, sensory organs, and cardiovascular system and its pathophysiology. Each chapter has been presented in a systematic and student-friendly manner to facilitate conceptual learning.

To enhance understanding, the book includes high-quality coloured illustrations, well-labelled diagrams, flowcharts, tables, clinical correlations, and practical applications. Every chapter includes Assess Yourself which contains Multiple choice questions (MCQs), short answer questions, and long answer questions to help students prepare for university and competitive pharmacy examinations.

We express our sincere gratitude to all the co-authors, contributors, reviewers, and the publisher for their valuable support and guidance. We also extend our heartfelt thanks to our family members for their constant encouragement and patience throughout the preparation of this book. We hope this textbook will serve as a valuable learning resource for students and an effective teaching aid for faculty members. We welcome constructive suggestions and feedback from teachers, students, and readers for improving future editions.

Authors

INDEX

UNIT I Introduction to Human Body & Cellular Adaptations **1-56**

1.1. INTRODUCTION TO HUMAN BODY

- 1.1.1. Definition and Scope of Anatomy, Physiology and Pathophysiology
- 1.1.2. Levels of Structural Organization
- 1.1.3. Body Systems
- 1.1.4. Basic Life processes
- 1.1.5. Homeostasis
- 1.1.6. Basic Anatomical Terminologies

1.2. CELLULAR LEVEL OF ORGANIZATION

- 1.2.1. Structure & Function of the Cell
- 1.2.2. Transport across Cell Membrane
- 1.2.3. Cell Cycle and Cell Division
- 1.2.4. General Principles of Cell Communication
- 1.2.5. Forms of Intracellular Signaling
- 1.2.6. Cell Junctions

1.3. TISSUE LEVEL OF ORGANIZATION

1.4. BASIC PRINCIPLES OF CELL INJURY AND ADAPTATION

- 1.4.1. Components of Homeostatic System
- 1.4.2. Causes of Cell Injury
- 1.4.3. Pathogenesis of Cell Injury
- 1.4.4. Morphology of Cell Injury
- 1.4.5. Cellular Adaptations
- 1.4.6. Cell Death

UNIT II Integumentary System and Musculo- Skeletal System **57-114**

2.1 STRUCTURE AND FUNCTION OF SKIN

2.2 SKIN DISORDERS

2.3 WOUND HEALING

2.4 SKELETAL SYSTEM

- 2.4.1 Divisions of Skeletal System
- 2.4.2 Types of Bones
- 2.4.3 Salient Features and Function of Bones of Axial and Appendicular Skeleton

2.5 THE MUSCULAR SYSTEM

- 2.5.1 Muscle Group
- 2.5.2 Neuromuscular Junction (NMJ)

- 2.5.3 Muscle Contraction

2.6 JOINTS

2.7 DISEASES OF BONES & JOINTS

UNIT III Body Fluids & Hematological System **115-184**

3.1. BODY FLUIDS, BLOOD AND LYMPHATIC SYSTEM

- 3.1.1. Distribution of Body Fluid
- 3.1.2. Fluids and Electrolytes Balance
- 3.1.3. Blood & Its Components
- 3.1.4. Coagulation of Blood
- 3.1.5. Blood Grouping
- 3.1.6. Blood Transfusion
- 3.1.7. Lymphatic System

3.2. BASIC MECHANISM OF INFLAMMATION AND REPAIR

- 3.2.1. Acute Inflammatory Response
- 3.2.2. Chronic Inflammation
- 3.2.3. Tissue Repair

3.3. HEMATOLOGICAL DISEASES

- 3.3.1. Iron Deficiency Anemia
- 3.3.2. Megaloblastic Anemia
- 3.3.3. Sickle Cell Anaemia
- 3.3.4. Thalassemia
- 3.3.5. Acquired Hemolytic Anemias
- 3.3.6. Hereditary Haemolytic Anaemias
- 3.3.7. Haemophilia

UNIT IV Peripheral Nervous System & Sensory Organs **185-220**

4.1 CLASSIFICATION OF PERIPHERAL NERVOUS SYSTEM

4.2 COMPONENTS OF PERIPHERAL NERVOUS SYSTEM

4.3 SENSE ORGANS

- 4.3.1 Structure and Functions of the Eye
- 4.3.2 Structure and Functions of the Ear
- 4.3.3 Structure and Functions of the Nose
- 4.3.4 Structure and Functions of the Tongue

4.4 SENSORY DISORDERS

**UNIT
V****Cardiovascular System****221-276**

- 5.1. ANATOMY OF HEART**
- 5.2. HEART VALVES AND CIRCULATION OF BLOOD**
- 5.3. BLOOD VESSELS**
- 5.4. CARDIAC CONDUCTION SYSTEM**
- 5.5. CARDIAC CYCLE, HEART SOUNDS AND CARDIAC OUTPUT**

5.6. REGULATION OF CARDIOVASCULAR PARAMETERS**5.7. PATHOPHYSIOLOGY OF CARDIOVASCULAR DISEASES**

- 5.7.1. Hypertension
- 5.7.2. Cardiac Arrhythmias
- 5.7.3. Congestive Heart Failure
- 5.7.4. Angina
- 5.7.5. Myocardial Infarction
- 5.7.6. Atherosclerosis
- 5.7.7. Coronary Artery Disease

Unit I

Introduction to Human Body & Cellular Adaptations



Syllabus

Introduction to human body

Cellular and tissue level of organization. Definition and scope of anatomy, physiology and pathophysiology. Levels of structural organization and body systems, basic life processes, homeostasis, basic anatomical terminologies. Structure and functions of cell, transport across cell membrane, cell division, cell junctions. General principles of cell communication, forms of intracellular signalling-

a) Contact-dependent, b) Paracrine, c) Synaptic, d) Endocrine

Classification of tissues, structure, location and functions of epithelial, muscular and nervous and connective tissues.

Basic principles of cell injury and adaptation

Components and types of feedback systems, causes of cellular injury, pathogenesis (cell membrane damage, mitochondrial damage, ribosome damage and nuclear damage), morphology of cell injury – adaptive changes (atrophy, hypertrophy, hyperplasia, metaplasia, dysplasia), cell swelling, intracellular accumulation and cell death. Definitions of commonly used relevant medical terminologies.



INTRODUCTION TO HUMAN BODY & CELLULAR ADAPTATIONS



LEARNING OBJECTIVES

After studying this chapter, the learner will be able to:

- Explain the basic concepts of anatomy, physiology, and pathophysiology.
- Describe the organization, systems, life processes, and homeostasis of the human body.
- Apply anatomical terms, planes, positions, and movements in anatomical descriptions.
- Explain the structure and functions of cells, the cell membrane, cytoplasm, nucleus, and cellular organelles.
- Classify the major tissue types and relate their structure to function.
- Explain the cell cycle, mitosis, and cytokinesis in growth and tissue maintenance.
- Relate common pathophysiological terms to cellular and tissue changes in disease.
- Describe cellular injury, oxidative stress, intracellular accumulations, and cellular adaptations.
- Differentiate the major types of cell death and necrosis.

1.1. INTRODUCTION TO HUMAN BODY

1.1.1. Definition and Scope of Anatomy, Physiology and Pathophysiology

❑ Introduction of Anatomy

Anatomy (ana = up; - tomy = process of cutting) is the science of body structures and the relationships among them.

❑ Type of Anatomy

1. Macroscopic anatomy (Gross): Body structures that can be observed without microscope.

2. Microscopic anatomy: Body structures that can be observed with microscope.

1. Gross Anatomy, also called macroscopic anatomy, is separated into 5 major divisions:

A. Surface anatomy describes surface forms and marks that can be understood by visualization and touch.

B. Regional anatomy describes the organization of specific areas of the body such as the head or hand. This approach is used mostly in professional schools: medical, dental, physical therapy.

C. Systemic anatomy describes groups of organs that function together for a single purpose.

D. Developmental anatomy describes the structural changes in an organism from fertilized egg to maturity. Embryology is the anatomical study of early development.

E. Clinical anatomy describes various medical specialties, including medical anatomy (changes that occur during illness), and radiographic anatomy.

2. Microscopic Anatomy is divided into two major divisions:

A. Cytology, the study of cells and their structures.

❖ **Skeletal Muscles**

Skeletal muscles help in body movement, protect internal organs from injury, and maintain body stability.

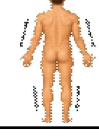
❖ **Nervous System**

The nervous system controls and coordinates body activities. It detects changes in the environment, sends signals for quick responses, and regulates vital functions necessary for maintaining homeostasis.

1.1.6. Basic Anatomical Terminologies

❑ Orientation and Directional Terms

Table 1.4: Orientation and Directional Terms

TERM	DEFINITION	ILLUSTRATION	EXAMPLE
Superior (upper)	Toward the head end or upper part of body; above		The forehead is superior to the nose.
Inferior (lower)	Away from the head end or toward the lower part of body; below		The stomach is inferior to lungs.
Ventral (anterior)	Toward or at the front of the body; in front of		The breastbone is anterior to the spine.
Dorsal (posterior)	Toward or at the backside of the body; behind		The heart is posterior to the sternum.
Median (middle)	Toward or at the midline of the body; on the inner side of		The heart is medial to the arm.
Lateral (side)	Away from the midline of the body; on the outer side of		The arms are lateral to the chest.
Ipsilateral	On the same side of the body as another structure		The gallbladder and ascending colon are ipsilateral.
Contralateral	On the opposite side of the body from another structure		The ascending and descending colons are contralateral.
Intermediate	Between a more medial and a more lateral structure		The collarbone is intermediate between breastbone and the shoulder.
Proximal	Close to the origin of the body part or the point of attachment of a limb to the body trunk		The elbow is proximal to the wrist.
Distal	Farther from the origin of a body part or the point of attachment of a limb to the body trunk		The knee is distal to the thigh.

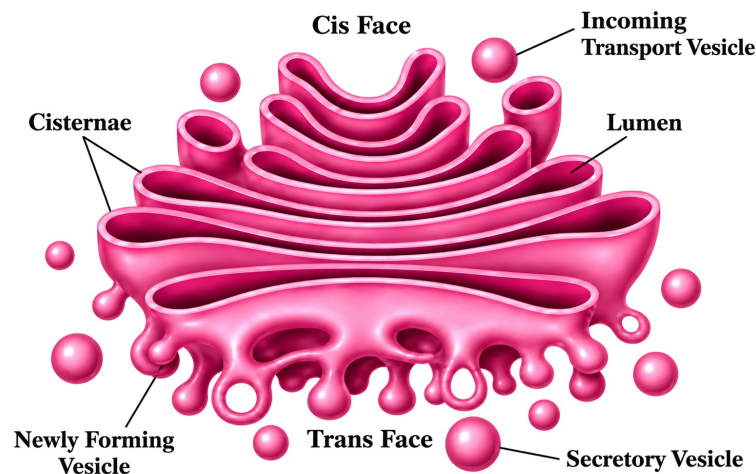


Fig.1.10: Structure of the Golgi Apparatus

- ✓ **Cisternae:**
 - These are flattened sacs arranged in stacks. In longitudinal view, they appear like tubules.
 - Each stack is called a dictyosome and usually contains 5–6 cisternae in animal cells.
- ✓ **Vesicles:**
 - **Transitional vesicles:** Small membrane-bound vesicles formed from the ER. They move to the cis face of the Golgi and fuse to form new cisternae.
 - **Secretory vesicles:** These vesicles become dense after filling with protein material. They release their contents outside the cell by exocytosis in response to signals.
 - **Clathrin-coated vesicles:** These are small, spherical vesicles with a rough surface, found at the periphery of the Golgi. They are different from secretory vesicles and help in **transport of materials within the cell** (intracellular transport/pinocytosis).
- ✓ **Functions of Golgi Apparatus**
 - It acts as a site for the **storage, processing, and packaging of cell products**.
 - It modifies, concentrates, and transports substances received from the endoplasmic reticulum (ER).
 - It helps in the formation of the plasma membrane, cell wall (in plants), and lysosomes.
 - It **produces secretory granules and vacuoles** containing substances such as hormones, enzymes, and proteins.
 - It synthesizes the polysaccharide part of glycoproteins.
- **Lysosomes**
 - Lysosomes are small membrane-bound sacs (0.25–0.50 micron in size) **that contain digestive enzymes**.
 - These enzymes break down large molecules like proteins, carbohydrates, lipids, and nucleic acids into smaller parts, which can then be used by the cell.
 - Sometimes, **lysosomes can digest the cell's own contents**, so they are also called “suicide bags.”

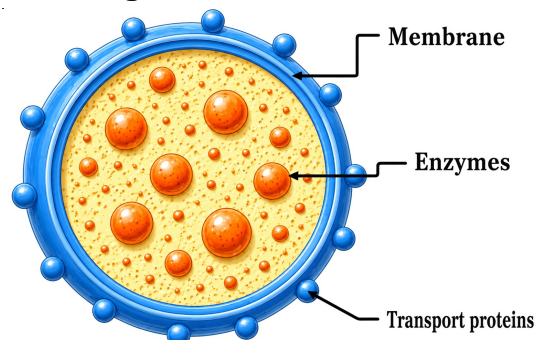


Fig.1.11: Structure of a Lysosome

➤ Stratified Epithelium

- It is composed of multiple layers of cells of different shapes. Its main function is protection.
- The greater the number of layers, the stronger its protective ability.
- It can resist abrasion and continuously renews itself.
- Stratified epithelium protects the underlying structures from mechanical damage and wear.

These are the three types of Stratified epithelium

✓ Pseudostratified Epithelium

- It is a single layer of cells of varying height giving a false appearance that looks multilayered because the nuclei lie at different levels.
- It consists of closely packed cells that appear to be arranged in layers but all of which are in fact attached to the basement membrane.
- It often bears cilia for moving and clearing substances or stereocilia for absorption, and is commonly found in the respiratory and parts of the reproductive system.

✓ Stratified Squamous Epithelium

- It is composed of multiple layers. The cells are of various shapes.
- It lines the esophagus, vagina and mouth cavity in non-keratinized form.
- The stratified squamous keratinized forms the epithelium of the skin, hair and nails.
- Here, the uppermost layers of cells are converted into keratin.

✓ Transitional Epithelium

- It lines most of the urinary tract. The uppermost layers are not flattened but are dome-shaped.
- The lower layers are pear shaped and columnar. This epithelium allows distension of the organ.

II. Connective Tissue

- It is the **most abundant tissue** in the body and mainly provides support by binding different tissues together.
- It contains cells embedded in a large extracellular matrix made of fibers and ground substance.
- Its functions include support, protection, transport, and insulation.
- The main cell, the fibroblast, produces both fibers and ground substance.
- Fat cells are common in adipose tissue. Macrophages, leukocytes, and mast cells help in body defense.
- Connective tissue contains three main fibers—collagen, elastic, and reticular distributed throughout the body.

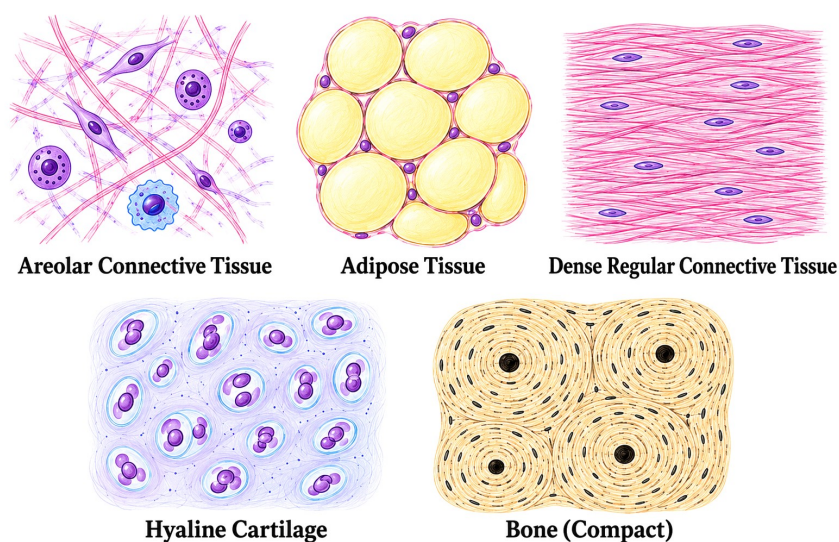


Fig.1.26: Microscopic Appearance of Different Connective Tissues

2. Positive Feedback Control System

Positive feedback control system reacts to increase the intensity of the change in the same direction. Positive feedback is less common than negative feedback. However, it has its own significance particularly during emergency conditions.

Examples of Positive Feedback Control System

Well known positive feedback mechanism occurs during blood clotting. Blood clotting is necessary to arrest bleeding during injury and it occurs in **three stages**:

I. Formation of prothrombin activator

II. Conversion of prothrombin activator

III. Conversion of fibrinogen into fibrin

- Thrombin formed in second stage stimulates the formation of more prothrombin activator in addition to converting fibrinogen into fibrin. It causes formation of more and more amount of prothrombin activator so that the blood clotting process is accelerated and blood loss is prevented quickly.
- Other processes where positive feedback occurs are **milk ejection reflex** and **parturition** and both the processes involve oxytocin secretion.

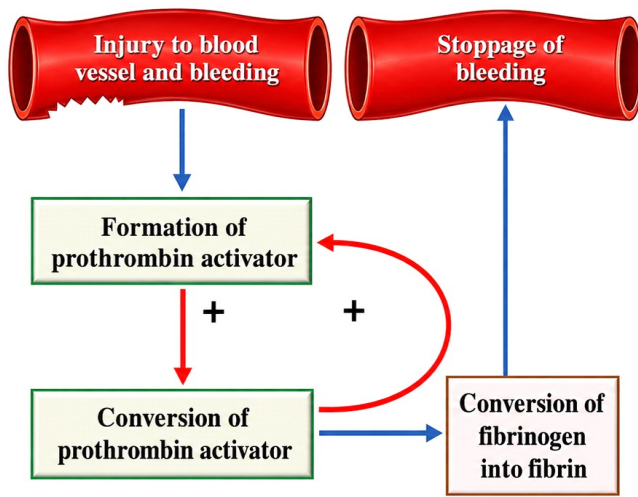


Fig.1.34: Positive feedback mechanism: Coagulation of blood. Once formed, thrombin induces the formation of more prothrombin activator.

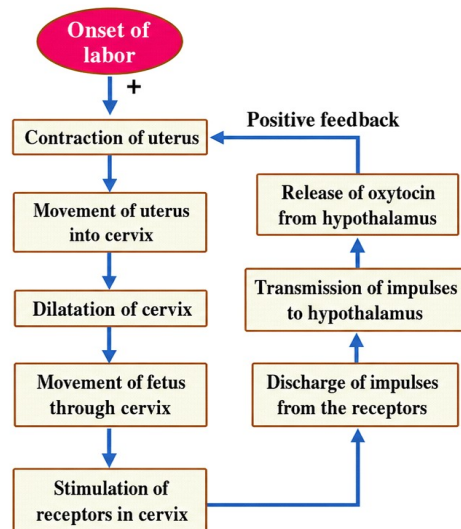


Fig.1.35: Positive feedback mechanism: Parturition

1.4.2. Causes of Cell Injury

A variety of stresses of factors may be responsible for cell injury. The important ones are described here.

- (a) Hypoxia:** Hypoxia (deficiency of oxygen) is the most common and most important cause of cell injury. Hypoxic cells are unable to carry aerobic metabolic reactions. Cells with a high energy turnover are particularly at risk of hypoxic injury. The **most common cause of hypoxia is ischemia** (inadequate blood supply) due to impaired arterial supply of venous drainage. However, hypoxia may result from other causes as well e.g. cardiac failure, respiratory failure, anaemia, carbon monoxide poisoning, etc

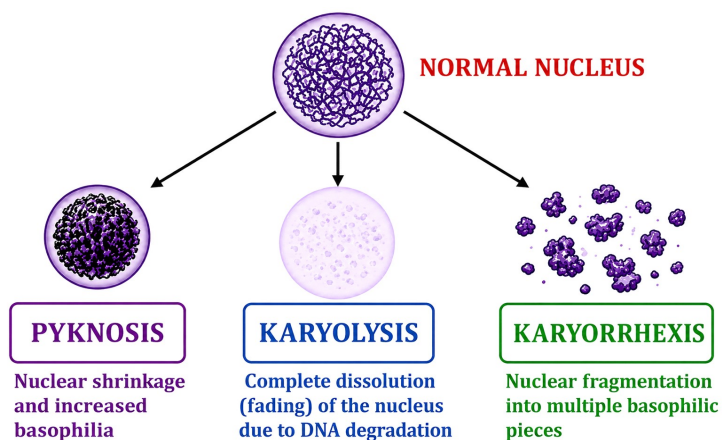
- It is also known as **unprogrammed demise of cells and living tissues**.
- The acidic pH of dying cells causes eosin dye to bind more strongly to cytoplasmic proteins.
- As a result, dead or wounded cells have a darker stain (eosin stain makes the cytoplasm pinker).
- Necrotic cell death occurs as a result of enzymatic digestion or protein denaturation.
- There are various diverse morphological patterns of necrosis based on the appearance of necrotic cell/tissue.

Table 1.12: Various Types of Necrosis

TYPE OF NECROSIS	FEATURES	EXAMPLES
Coagulative Necrosis	• Caused by irreversible focal injury, mostly from sudden cessation of blood flow. • Occurs due to denaturation of proteins.	Hypoxic cell death e.g. Myocardial infarction
Liquefaction Necrosis	• Digestion of dead cell to form viscous liquid mass. • Occurs due to ischaemic injury and bacterial or fungal infections	Hypoxic injury to cells of central nervous system
Caseous Necrosis	• Whitish appearance of central necrotic area • Combination of coagulative and liquefactive necrosis	Infection by <i>M. tuberculosis</i> and some fungi
Fat necrosis	• Action of lipases on fatty tissue	Traumatic fat necrosis of the breast
Fibrinoid necrosis	• Complexes of antigens and antibodies • Leakage of fibrinogen out of vessels	Immune complex vasculitis, autoimmune diseases
Gangrenous necrosis	• Dry gangrene → Ischemic injury occurs • Wet gangrene → Infection occurs	Diabetic and chain smokers are more susceptible to gangrene development

Table 1.13: Morphological changes in Nucleus during Necrosis

NUCLEAR CHANGES	FEATURES
Karyolysis 	• Due to activation of DNAase activity • Degradation of DNA or chromosome • Fading of basophilia of chromatin
Pyknosis 	• Shrinkage of nuclear components • DNA condense into solid shrunken mass • Increase in basophilia of chromatin
Karyorrhexis 	• Followed by fragmentation of nucleus • Pyknotic nucleus undergoes fragmentation

**Fig.1.40: Morphological changes in nucleus during irreversibly injured cell**

Unit II

Integumentary System and Musculo- Skeletal System



Syllabus

Integumentary system and wound healing

Structure and functions of skin. Skin disorders: Psoriasis and dermatitis and pathophysiology of Leprosy. Basic principles of wound healing.

Skeletal system and joints

Divisions of skeletal system, types of bones, salient features and functions of bones of axial and appendicular skeletal system. Organization of skeletal muscle, physiology of muscle contraction, neuromuscular junction. Structural and functional classification of joints.

Diseases of bones and joints

Pathophysiology of rheumatoid arthritis, osteoporosis and gout.



INTEGUMENTARY SYSTEM AND MUSCULO- SKELETAL SYSTEM



LEARNING OBJECTIVES

After studying this chapter, the learner will be able to:

- Explain the structure and functions of the integumentary system, skin, and its accessory organs.
- Describe the etiology, pathogenesis, and clinical features of common skin disorders, including psoriasis, dermatitis, and leprosy.
- Discuss the phases and factors involved in wound healing and tissue repair.
- Understand the organization, classification, and functions of the skeletal system, bones, and joints.
- Describe the structure and physiology of muscles, the neuromuscular junction, and the mechanism of muscle contraction.
- Correlate the pathophysiology and clinical manifestations of common disorders affecting bones, joints, and muscles.

❑ INTRODUCTION TO INTEGUMENTARY SYSTEM

The integumentary system is composed of the **skin** and its associated or accessory structures, including **hair, hair follicles, nails, glands (sebaceous and sweat glands), and sensory nerves**. It forms the outermost covering of the body and serves as a critical interface between the internal environment and the external surroundings.

❖ Functions of the Integumentary System

1. Protection

The integumentary system provides a **physical, chemical, and biological barrier** that protects the body from various external harmful agents:

- **Mechanical damage:** Protects against trauma such as cuts, abrasions, and bruises.
- **Chemical damage:** Acts as a barrier against harmful chemicals such as acids and bases.
- **Thermal damage:** Shields the body from extreme temperatures (heat and cold).
- **Ultraviolet (UV) radiation:** Melanin in the skin protects against harmful effects of sunlight.
- **Microbial defense:** Prevents entry of microorganisms through intact skin and supports immune responses.

2. Regulation of Body Temperature (Thermoregulation)

The skin helps maintain normal body temperature by:

- **Sweating**, which cools the body through evaporation
- **Vasodilation and vasoconstriction**, which regulate heat loss and retention

3. Sensory Function

The skin contains numerous **sensory receptors** that detect:

- Touch • Pressure • Pain • Temperature

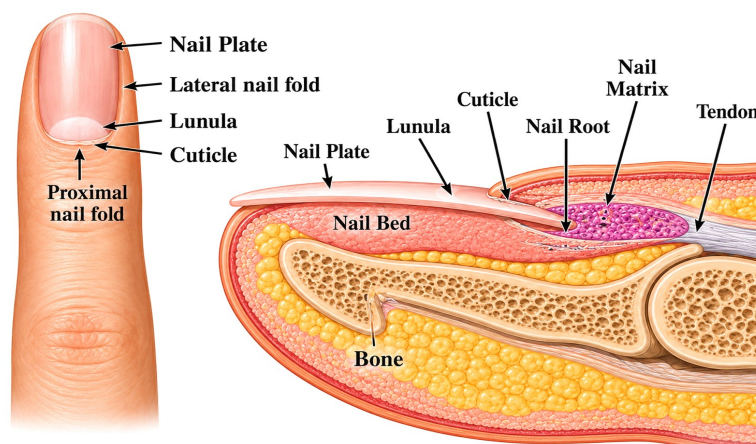
This allows the body to respond appropriately to environmental changes.

C. Nails:

- Nails are hard, keratinized structures found at the ends of fingers and toes.
- They serve to protect the distal phalanges and assist in precise movements by providing structural support.

➤ Structure of Nails:

- **Nail Plate:** Hard, translucent keratinized part lying on the nail bed.
- **Nail Bed:** Vascular skin beneath the plate, gives pink color.
- **Nail Matrix:** Growth region producing keratinocytes for nail formation.
- **Lunula:** Visible white crescent; part of the matrix.
- **Cuticle (Eponychium):** Protective seal over the matrix.
- **Nail Folds:** Surround and support the nail on three sides.

**Fig.2.6: Structure of Nails****D. Sebaceous gland:**

- Sebaceous glands are small, sac-like glands located in the dermis of the skin.
- These are holocrine glands, meaning the entire cell disintegrates to release its secretions.
- They are responsible for producing and secreting sebum, an oily substance that helps maintain skin and hair health.

➤ Location:

- Found throughout the skin, except on the palms and soles.
- Highly concentrated on the face, scalp, chest, and upper back.

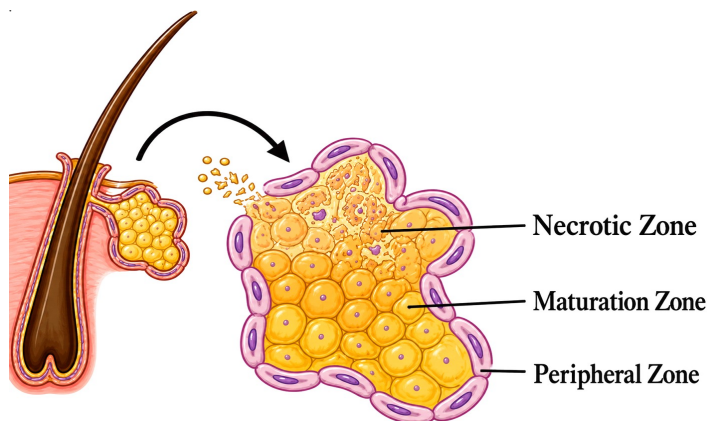
**Fig.2.7: Structure of Sebaceous gland**



Fig.2.15: Atopic Dermatitis

3. SEBORRHEIC DERMATITIS

Seborrheic dermatitis is an acute or chronic inflammatory skin disorder that mainly affects areas rich in sebaceous glands such as the scalp, face, sternum, and between the shoulder blades. It may also involve intertriginous areas like the armpits, groin, navel, and beneath the breasts. In infants, it presents as cradle cap with thick yellowish crusts on the scalp, while adults commonly develop dandruff and grey-yellow scaly lesions with mild itching. The condition is genetically determined and may worsen with infections. Dandruff is considered a mild form and is associated with colonization by *Pityrosporum ovale* yeast.



Fig.2.16: Seborrheic Dermatitis

4. EXFOLIATIVE DERMATITIS

This is a life-threatening complication. Rest, a high-protein diet to replace the serum proteins lost through the extensively damaged skin, and the use of high-dose systemic steroids comprise the normal treatment mode, plus the specific management of any underlying cause, if one can be identified. All nonessential drugs should be withdrawn.

It may also complicate psoriasis, lymphomas and leukaemias.



Fig.2.17: Exfoliative Dermatitis

❑ LEPROSY

- Leprosy, also called **Hansen's disease**, is a chronic infectious disease caused by **Mycobacterium leprae**. It was first described in ancient Indian texts, and the causative organism was discovered by Hansen in 1874.
- The disease mainly affects the cooler parts of the body, such as the skin, peripheral nerves, eyes, upper respiratory tract, lymph nodes, and testes. The earliest and most important involvement is seen in the skin and peripheral nerves, leading to skin lesions and nerve damage.

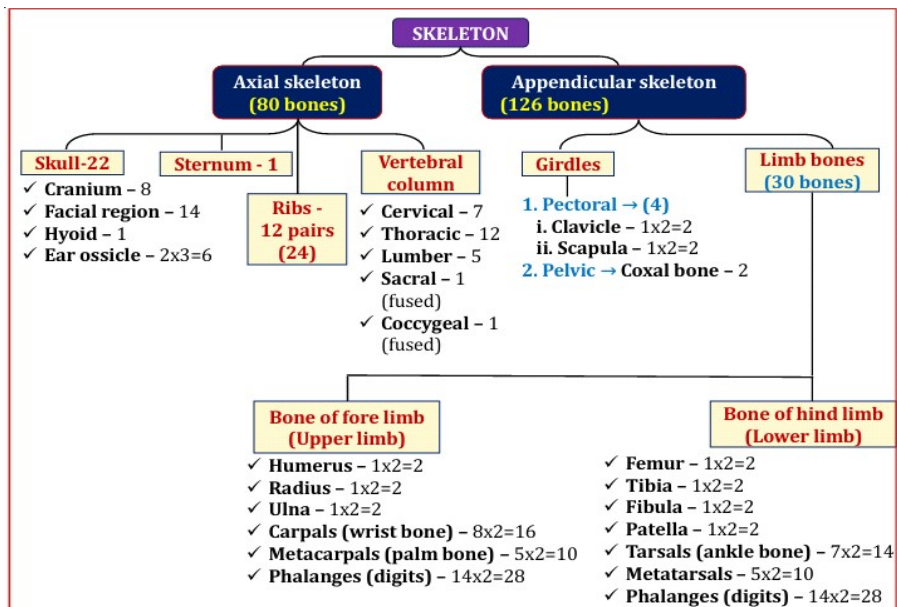


Fig.2.23: Classification and Distribution of Human Bones

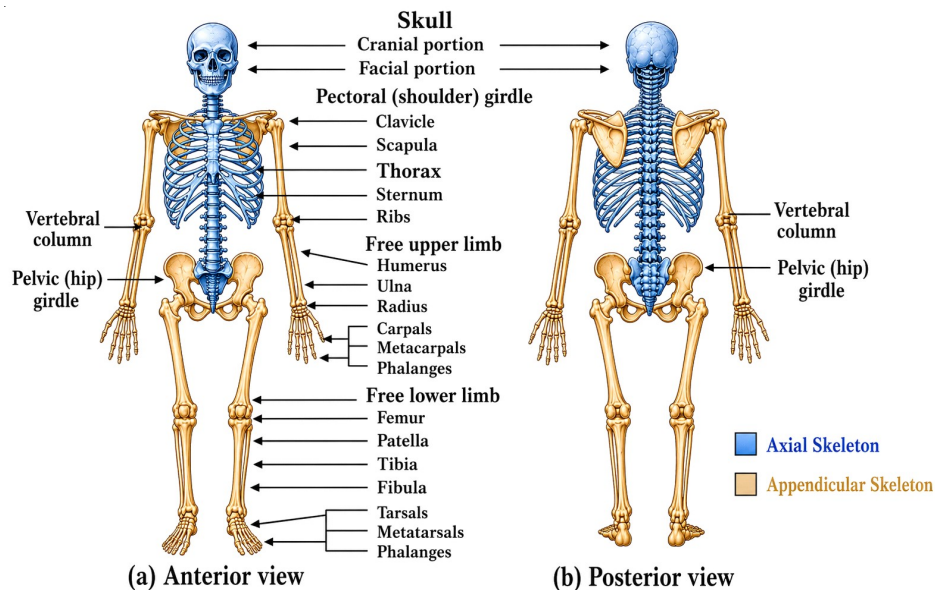


Fig.2.24: Structural Organization of the Human Skeleton

2.4.2. Types of bones

❖ Short bones

- These are cube-shaped bones and are equal in length and width.
- They consist of spongy bone tissue.
- Examples of short bones are the carpal bones (except pisiform) and the tarsal bones (except heel bone).

❖ Flat bones

- These are thin bones and are composed of two parallel plates of compact bone tissue enclosing a layer of spongy bone tissue.
- Flat bones give protection and provide extensive area for muscle attachment.
- Flat bones include the cranial bones, which protect the brain; the sternum (breast bone) and ribs, which protect organs in the thorax; and the scapulae (shoulder blades).

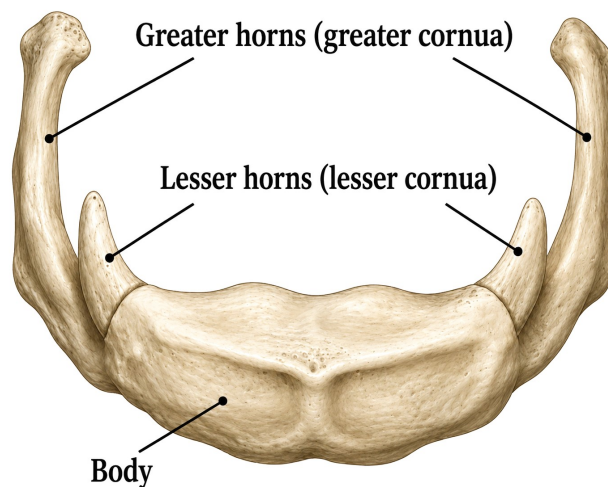


Fig.2.27: Hyoid Bone

3. STERNUM (Breast bone-1)

Table 2.12: Description and Anatomical Location of Sternum

Bone	Descriptions (Location)
Sternum	The sternum is a flat bone located in the middle of the anterior thoracic wall. • It has three parts: manubrium, body, and xiphoid process. • The junction of the manubrium and body forms the sternal angle. • The manubrium contains the jugular notch and clavicular notches. • The sternum articulates with the clavicles and ribs. • The xiphoid process remains cartilaginous in childhood and ossifies later in life.

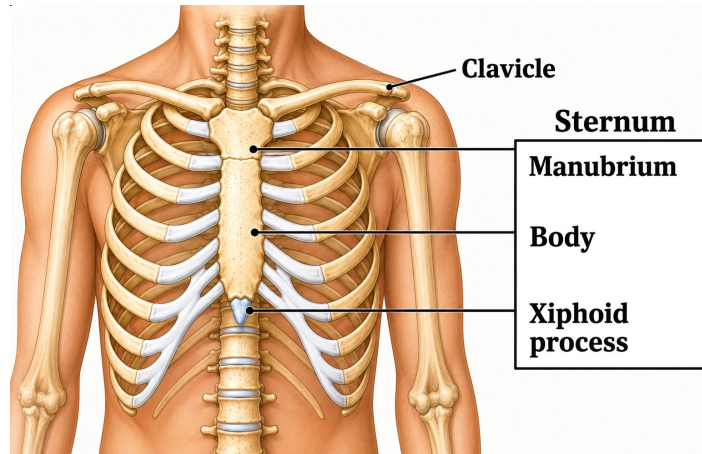


Fig.2.28: Thoracic Cage Showing the Sternum and Ribs

4. RIBS

Humans have 12 pairs of ribs that form the sides of the thoracic cavity. Posteriorly, each rib articulates with its corresponding thoracic vertebra. The first seven pairs of ribs attach directly to the sternum through costal cartilage and are known as true ribs. The 8th to 12th pairs are called false ribs because they do not attach directly to the sternum. Among these, the 11th and 12th pairs are referred to as floating ribs, as they have no anterior attachment to the sternum.

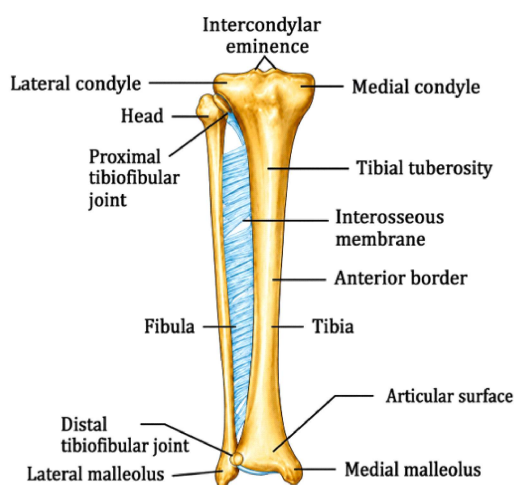


Fig.2.37: Left Tibia and Fibula (Anterior view)

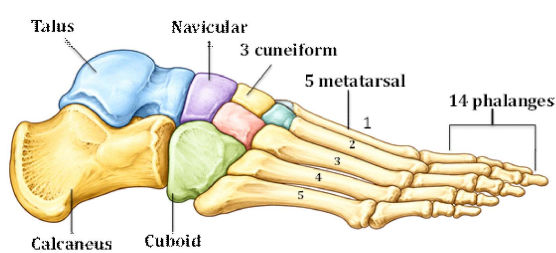


Fig.2.38: The Bones Of The Foot



Fig.2.39: Patella (Knee Cap)

2.5. THE MUSCULAR SYSTEM

Muscular system is an essential organ system in the human body that enables movement, provides stability, and supports vital bodily functions. It comprises specialized cells called muscle fibers that contract and relax to produce force. This system works in conjunction with the skeletal system to facilitate movement and maintain posture.

❑ TYPES AND STRUCTURE OF MUSCLE

- Muscular tissue is specialized for contraction and movement.
- It is classified into three main types based on structure, function, and control (voluntary or involuntary).

A. Skeletal Muscle:

Skeletal muscles are attached to bones with the help of tendons. Their muscle fibers are long, cylindrical, multinucleated, and show alternating light and dark bands known as striations. These muscles function under voluntary control, meaning their movements can be consciously controlled by a person.

❖ Structure of skeletal muscle

1. Muscle Fiber (Muscle Cell):

- A skeletal muscle fiber is a long, cylindrical muscle cell containing many nuclei.
- Each muscle fiber is enclosed by a thin membrane called the sarcolemma.

2. Fascicles:

- Muscle fibers are arranged in groups known as fascicles.
- Each fascicle is surrounded by a connective tissue layer called the perimysium.

3. Connective Tissue Layers:

- **Epimysium:** The outer connective tissue covering that encloses the entire muscle.
- **Endomysium:** A delicate connective tissue layer that surrounds each individual muscle fiber.

4. Organelles:

a. Myofibrils:

- Myofibrils are long, thread-like structures present inside muscle fibers.
- They are made up of repeating units called sarcomeres, which are responsible for muscle contraction.

b. Sarcoplasmic Reticulum (SR):

- The sarcoplasmic reticulum is a specialized network within the muscle fiber that stores calcium ions (Ca^{2+}).
- These calcium ions are essential for muscle contraction.

- Mitochondria
- Glycogen
- Myoglobin
- Enzymes required for energy production

Muscle fibers are highly specialized for contraction and contain numerous contractile proteins that help in movement.

2. Myofibrils

Myofibrils are long, rod-like contractile structures present within the sarcoplasm of muscle fibers. They run parallel to each other throughout the length of the muscle cell.

Each myofibril is composed of repeating segments called **sarcomeres**, which are responsible for the striated appearance of skeletal muscle.

Myofibrils contain:

- Thin filaments (actin)
- Thick filaments (myosin)

The arrangement of these filaments allows muscle shortening during contraction.

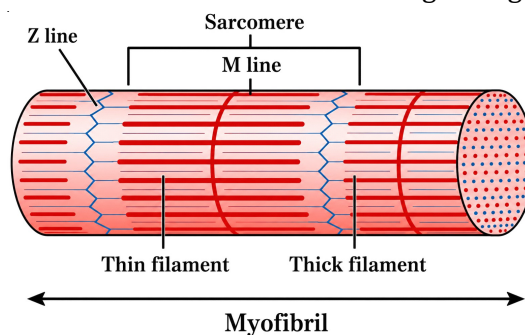


Fig.2.47: Organization of a Sarcomere in a Myofibril

3. Sarcomere

The sarcomere is the structural and functional unit of muscle contraction. It extends between two adjacent **Z-lines (Z-discs)**.

Important parts of the sarcomere include:

- **A-band:** Dark region containing thick myosin filaments and overlapping actin filaments
- **I-band:** Light region containing only thin actin filaments
- **H-zone:** Central lighter area containing only myosin filaments
- **M-line:** Middle part of the sarcomere that supports myosin filaments
- **Z-line:** Boundary of each sarcomere where actin filaments are attached

During contraction, the sarcomere shortens as actin filaments slide over myosin filaments.

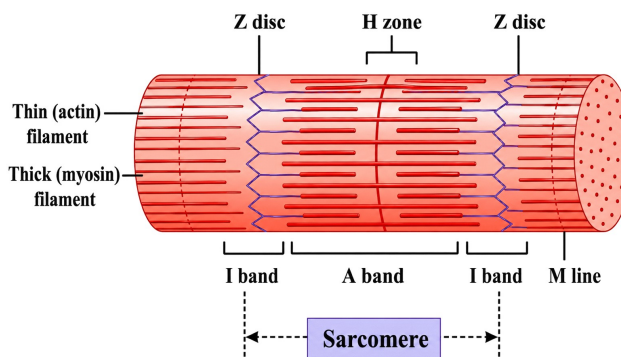


Fig.2.48: Structural Organization of a Sarcomere

1. Synarthroses (Immovable Joints)

Synarthroses are joints that do not allow movement. These joints are usually fibrous in nature and provide stability and protection.

Example:

- Sutures between the bones of the skull

2. Amphiarthroses (Slightly Movable Joints)

Amphiarthroses are joints that permit only a small amount of movement. Most of these joints are cartilaginous joints.

Example:

- Intervertebral discs between the vertebrae

3. Diarthroses (Freely Movable Joints)

Diarthroses are joints that allow free and wide movement. All synovial joints belong to this category. These joints are important for body movements and flexibility.

Examples:

- Shoulder joint
- Knee joint

❑ FIBROUS JOINTS

Table 2.23: Types of Fibrous Joints

Type of Fibrous Joint	Description	Location/Example	Movement
Sutures	Bones are connected by a thin layer of dense fibrous connective tissue	Found between skull bones (e.g., coronal suture between frontal and parietal bones)	Immovable (Synarthrosis)
Syndesmoses	Bones are joined by ligaments or an interosseous membrane	Between tibia and fibula; between radius and ulna	Slightly movable (Amphiarthrosis)
Gomphoses	Peg-like joint where a tooth fits into its socket and is held by periodontal ligament	Joint between teeth and alveolar sockets of maxilla and mandible	Immovable (Synarthrosis)

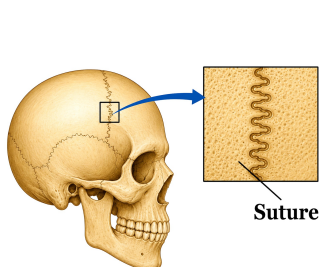


Fig.2.51: Suture between skull bones

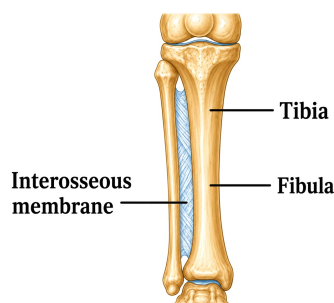


Fig.2.52: Syndesmoses between Tibia and Fibula

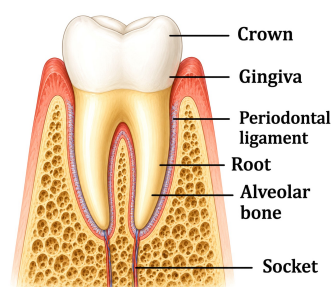


Fig:2.53: Gomphosis between tooth and socket of alveolar process

❑ CARTILAGINOUS JOINTS

A cartilaginous joint does not contain a synovial cavity and permits very little or no movement. In these joints, the bones are connected by either hyaline cartilage or fibrocartilage. Cartilaginous joints are of two types: **synchondroses** and **symphyses**.

➤ **Primary Osteoporosis**

Type I – Postmenopausal Osteoporosis

- Common in women after menopause
- Caused by estrogen deficiency
- Mainly affects trabecular bone and vertebrae

Type II – Senile Osteoporosis

- Occurs after 70 years of age
- Affects both men and women
- Mainly involves cortical bone loss

➤ **Contributing Factors**

- Genetic predisposition
- Female sex
- Reduced physical activity
- Hormonal deficiency
- Vitamin D deficiency
- Hyperparathyroidism

➤ **Clinical Complications**

- Bone fractures
- Reduced physical activity
- Joint and mobility problems
- Increased risk of pneumonia and blood clots in elderly patients

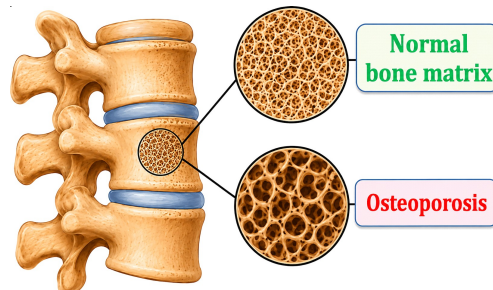


Fig.2.65: Comparison Between Normal and Osteoporotic Bone

❑ **GOUT**

Gout is a metabolic disorder caused by increased levels of uric acid in the blood. Excess uric acid forms monosodium urate crystals that deposit in synovial joints, leading to pain and inflammation. Uric acid levels above 7.0 mg/dL increase the risk of gout. The condition is commonly associated with increasing age, male gender, obesity, high blood pressure, alcohol intake, and elevated serum creatinine and blood urea nitrogen levels.

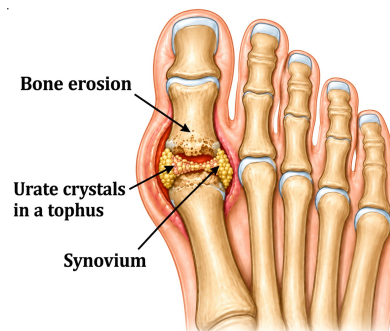
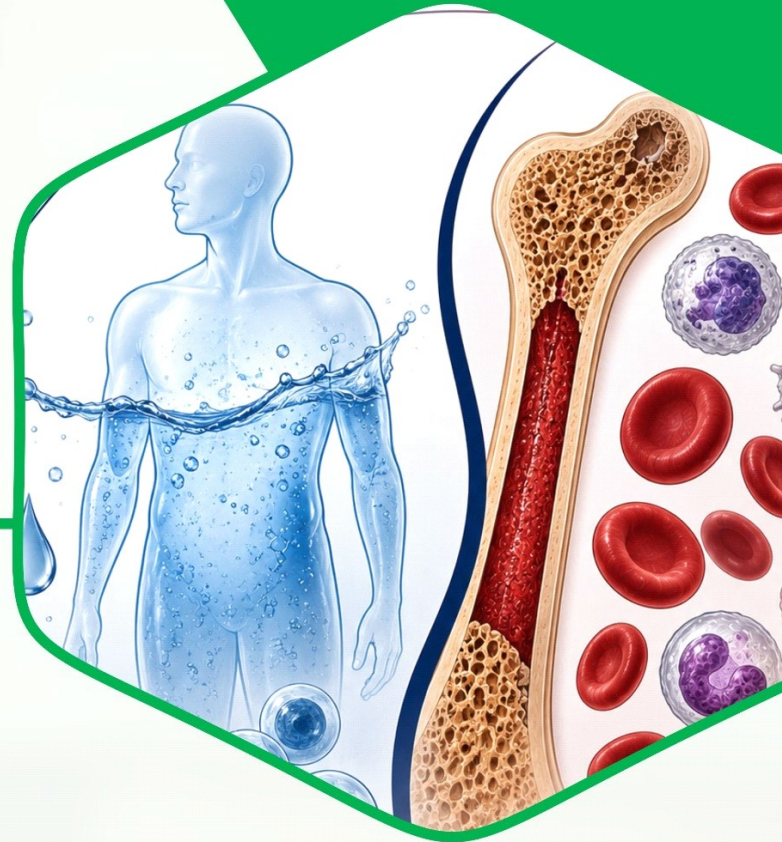


Fig.2.66: Crystal-Induced Joint Inflammation in Gout

Body Fluids & Hematological System

Unit III



Syllabus

Body fluids, blood and lymphatic system

Body fluids, composition and functions of blood, hemopoiesis, formation of haemoglobin, mechanisms of coagulation, blood grouping, Rh factors and transfusion. Lymphatic organs and tissues, lymphatic vessels, lymph formation, circulation and functions of lymphatic system.

Basic mechanism of inflammation and repair

Introduction, classification and pathophysiology of inflammation, mediators of inflammation.

Haematological diseases

Pathophysiology of iron deficiency, megaloblastic anaemia (Vit B12 and folic acid), sickle cell anaemia, Thalassemia, hereditary acquired anaemia and haemophilia.



BODY FLUIDS & HEMATOLOGICAL SYSTEM



LEARNING OBJECTIVES

After studying this chapter, the learner will be able to:

- Explain the physiology and regulation of body fluids, blood, electrolytes, acid–base balance, and the lymphatic system in maintaining homeostasis.
- Describe the structure, functions, hematopoiesis, hemostasis, coagulation, blood groups, and Rh factor.
- Explain the principles and clinical significance of blood typing, cross-matching, blood transfusion, and transfusion reactions.
- Discuss the mechanisms and significance of inflammation, tissue repair, regeneration, fibrosis, and wound healing.
- Describe the functions and immunological role of the lymphatic system and lymphoid organs.
- Explain the etiology, pathophysiology, clinical features, diagnosis, and management of anemia, including iron deficiency anemia, megaloblastic anemia, sickle cell anemia, thalassemia, and hereditary and acquired hemolytic anemias.
- Explain the etiology, pathophysiology, clinical features, diagnosis, and management of hemophilia (A, B, and acquired hemophilia), and correlate major hematological, coagulation, inflammatory, lymphatic, and wound-healing disorders with clinical conditions.

3.1. BODY FLUIDS, BLOOD AND LYMPHATIC SYSTEM

□ Introduction

- **Body fluids** are essential for maintaining life and ensuring the smooth functioning of all body processes. They consist primarily of **water** and are **dilute aqueous solutions containing dissolved substances** present inside and outside cells. In humans, body fluids mainly include **blood and lymph**, which circulate throughout the body and help maintain internal balance (homeostasis).
- **Blood** is a specialized connective tissue composed of plasma and blood cells. It performs vital functions such as transport of oxygen, nutrients, hormones, and waste products. It also plays a key role in immunity and regulation of body temperature and pH.
- The **lymphatic system** consists of lymph, lymph vessels, and lymphoid organs. Lymph is a colorless fluid formed from tissue fluid that helps in the transport of fats, removal of excess interstitial fluid, and defense against infections. Lymphatic organs like lymph nodes, spleen, and thymus are important for immune responses.
- Together, the blood and lymphatic system form a transport and defense network that supports circulation, protects the body from diseases, and maintains fluid balance.

❑ Water : The Essential Component of Body Fluids

- Water is the most essential and abundant component of the body fluid. It makes up nearly **70% of total body weight** and acts as a medium in which important ions are dissolved. Major cations present include sodium, potassium, calcium, hydrogen, and magnesium, while the main anions include chloride, bicarbonate, and proteins.
- Life cannot exist without water. It forms the **intracellular environment** where metabolic reactions necessary for life take place. Lack of water causes death more rapidly than lack of food. If water is available, a person may survive for several weeks even after losing most body fat and about 50% of body protein.



3.1.1. Distribution of Body Fluids

An average person weighing about 70 kg has nearly **40–45 liters of water** in the body. **In males**, water makes up about **65% of body weight**, while **in females it is about 10% less**. Babies and young people have a higher water content in their bodies. In older people, the amount of body water decreases with age.

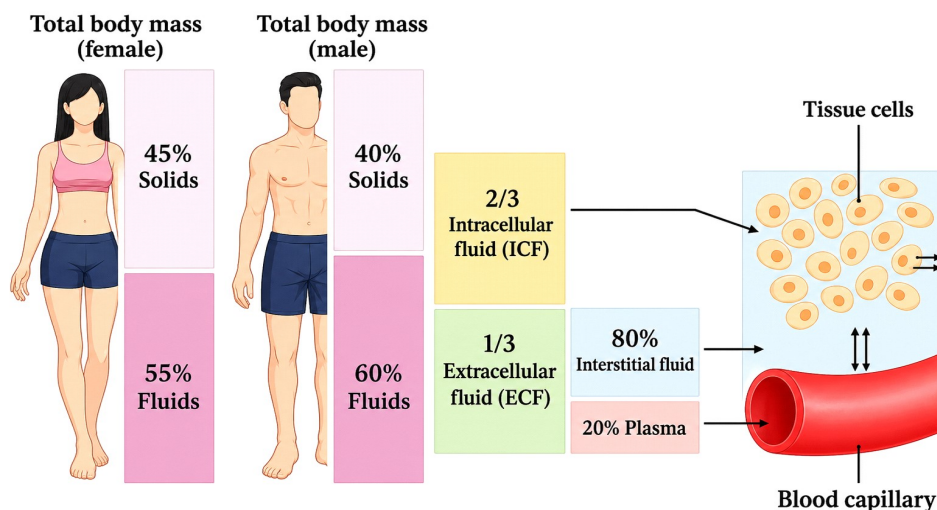


Fig.3.1: Distribution of Intracellular and Extracellular Fluids in the Human Body

❑ Body Fluid Compartments

Body fluid is distributed into two main compartments:

- I. **Intracellular Fluid (ICF), which is found within cells, and**
- II. **Extracellular Fluid (ECF), which is present outside cells**

- The ECF includes interstitial fluid (between tissues), blood plasma (within blood vessels), lymph (in lymphatic vessels), cerebrospinal fluid (surrounding the brain and spinal cord), synovial fluid (within joints), and the aqueous humor and vitreous body of the eyes.
- The cell membrane forms the boundary between these two compartments and allows water and small molecules to move freely between the intracellular and extracellular spaces, while larger molecules require specialized transport mechanisms for movement. The concentrations of substances in the **ICF and ECF remain different** because the cell membrane is selectively permeable, helping maintain a stable distribution of substances across the various body fluid compartments.

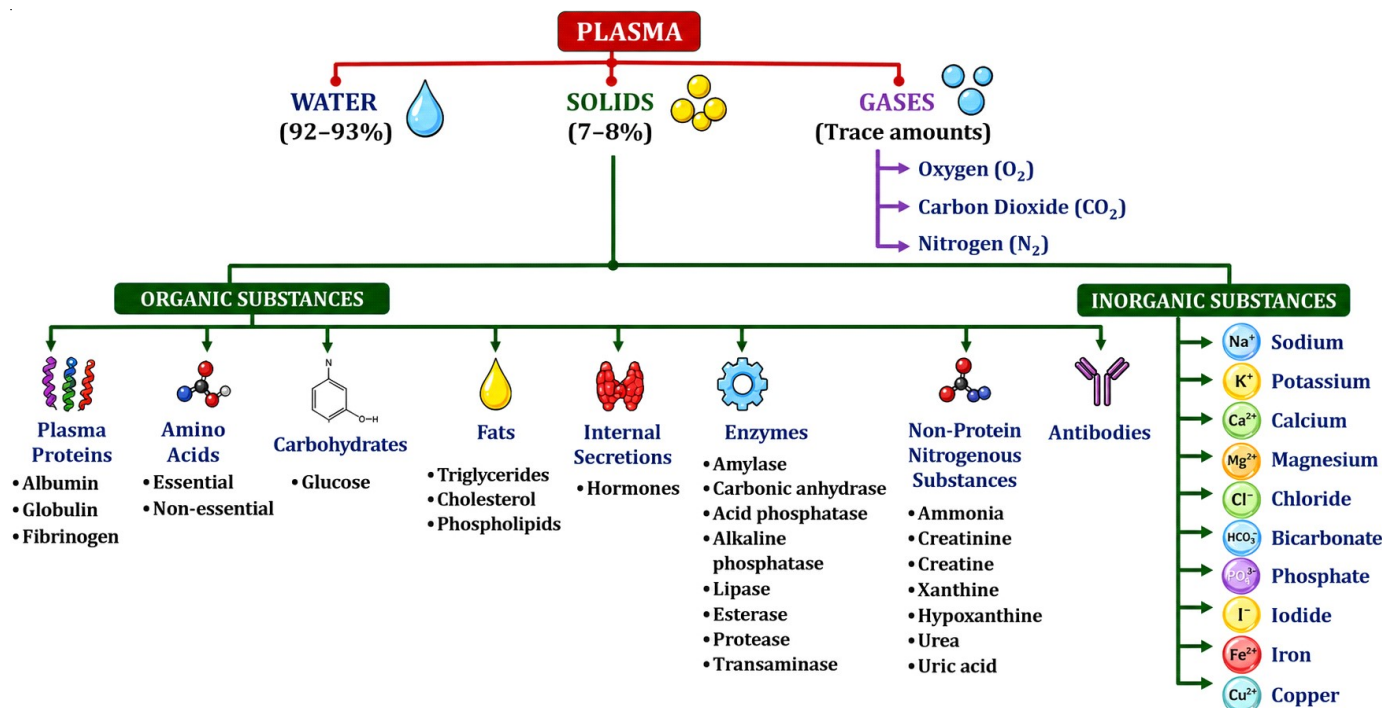


Fig.3.6: Organization of Blood Plasma Constituents

❖ **Formed Elements in Blood**

Formed elements are the cellular components of blood and include three main parts:

I. Red Blood Cells (RBCs)**II. White Blood Cells (WBCs)****III. Platelets**

RBCs and WBCs are complete cells, while platelets are small cell fragments formed from large cells called megakaryocytes.

Table 3.4: Formed Elements of Blood

BLOOD CELLS	CELL TYPE	ORIGIN	LIFE SPAN	TYPE OF NUCLEUS	FUNCTION
	Red Blood Cells (Erythrocytes)	Bone marrow	About 120 days	Anucleate (No nucleus)	Transport oxygen and carbon dioxide
	Platelets (Thrombocytes)	Bone marrow (from megakaryocytes)	5-9 days	Anucleate (No nucleus)	Help in blood clotting and stop bleeding
	White Blood Cells (Leukocytes)	Bone marrow and lymphoid tissues	Few hours to few days	Multilobed (Depends on type)	Defend the body against pathogens
COMPONENTS OF WHITE BLOOD CELLS					
	Neutrophils (50-70%)	Bone marrow	2-5 days	Multilobed (2-5 lobes)	Phagocytosis; destroy bacteria and microbes
	Eosinophils (2-4%)	Bone marrow	7-12 days	Bilobed (2 lobes)	Combat allergic reactions and parasites

- **Matching** = Recipient's RBCs + Test sera.
- **Cross-matching** = Recipient's serum + Donor's RBCs.

Table 3.8: Overview of ABO Blood Groups and Their Clinical Significance

Blood Group	Antigen on RBCs	Antibodies in Plasma	Population (%)	Can Donate To	Can Receive From	Remark
A	A antigen	Anti-B	25%	A, AB	O, A	—
B	B antigen	Anti-A	35%	B, AB	O, B	—
AB	A and B antigens	No antibody	7%	AB	O, A, B, AB	Universal Recipient
O	No antigen	Anti-A and Anti-B	33%	O, A, B, AB	O	Universal Donor

❑ Inheritance of ABO Agglutinogens and Agglutinins

- Blood group** is determined by two genes inherited from the parents, following the principles of Mendelian inheritance. **Gene A and Gene B are dominant**, whereas **Gene O is recessive**. The **A and B agglutinogens (antigens)** appear on red blood cells during the **6th month of fetal life** and gradually increase to reach adult levels by puberty.
- These agglutinogens are present not only on RBCs but also in various tissues and organs, including the **salivary glands, pancreas, kidneys, liver, and lungs**. In contrast, **agglutinins (α and β antibodies)** are not produced during fetal life and usually appear **2–3 months after birth**.
- They are produced in response to A or B agglutinin-like substances that enter the body through the respiratory or digestive tract, often carried by bacteria. **Agglutinins are gamma globulins, primarily of the IgG and IgM immunoglobulin classes, that play an important role in blood group compatibility and immune reactions.**

Table 3.9: Inheritance of ABO Blood Group

GENE FROM PARENTS	GROUP OF OFFSPRING	GENOTYPE
A + AA + O	A	AA or AO
B + BB + O	B	BB or BO
A + B	AB	AB
O + O	O	OO

❑ Rh Factor

- Rh factor** is an antigen present on **red blood cells (RBCs)** and was discovered by **Landsteiner and Wiener**. It was first identified in the **Rhesus monkey**, from which it derives its name. Although several Rh antigens exist, the D antigen is the most antigenic and clinically important in humans.
- Individuals possessing the D antigen are classified as Rh positive, while those lacking it are Rh negative. In the Indian population, approximately 85% are Rh positive and 15% are Rh negative, with Rh positivity being more common among Black populations. Unlike the ABO blood group system, the Rh system lacks a naturally **occurring anti-D antibody**.
- However, transfusion of Rh-positive blood into an **Rh-negative** individual can stimulate the production of **anti-D** antibodies. Conversely, an **Rh-positive** person can safely receive Rh-negative blood without the risk of Rh incompatibility.

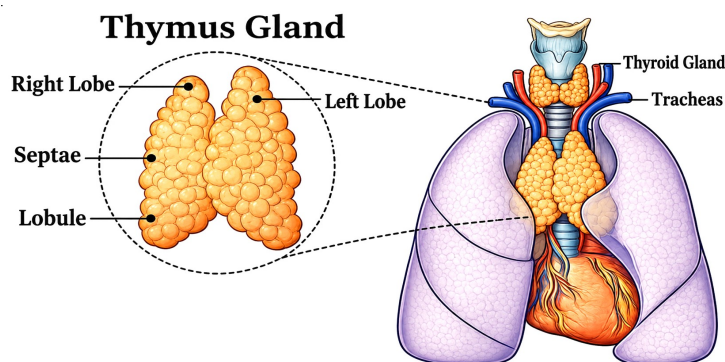


Fig.3.16: Structure And Mediastinal Position of the Thymus Gland

➤ Functions of Thymus

- Immature **T cells (thymocytes)** travel from the bone marrow to the thymus, where they mature into helper T cells and killer T cells.
- The thymus ensures that T cells recognize pathogens without attacking the body's own cells, preventing autoimmunity.
- The thymus produces hormones such as thymosin, which promote T-cell growth and development.
- Mature **naive T cells** are released into the bloodstream and move to secondary lymphoid organs like **lymph nodes and the spleen** to fight infections.

❖ Lymph Nodes

- Lymph nodes are **small, bean-shaped structures** located along lymphatic vessels, with about 600 nodes present in the body, mainly in groups near the **mammary glands, axillae, and groin**. Each node is enclosed by a connective tissue capsule that gives rise to trabeculae, providing support and allowing blood vessels to enter.
- A lymph node consists of an **outer cortex and an inner medulla**. The cortex contains B cells, T cells, dendritic cells, and lymphatic nodules with germinal centers that produce plasma cells and memory B cells after antigen exposure. The medulla contains plasma cells, B cells, and macrophages.
- Lymph enters through **afferent lymphatic vessels** and leaves through efferent vessels at the hilum. Macrophages filter foreign particles and pathogens, while lymphocytes initiate immune responses. The slow flow of lymph ensures efficient filtration before it returns to the bloodstream.

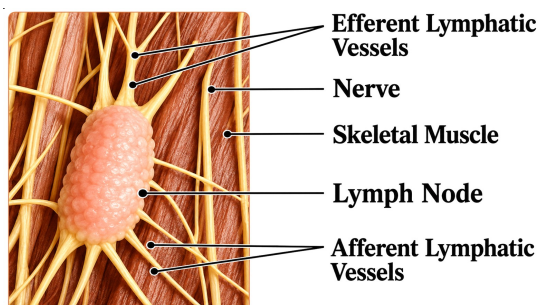


Fig.3.17: Typical Lymph Node and Its Lymphatic Connections

➤ Functions of lymph nodes

Lymph nodes filter lymph before it is returned to the bloodstream and help prevent foreign particles, such as bacteria and viruses, from entering the blood. They also play an important role in immunity by producing lymphocytes that help defend the body against infections.

iv. Stasis of Blood Flow

Loss of fluid from the blood vessels increases the viscosity of blood and slows microcirculation. As a result, blood flow becomes sluggish, a condition known as **stasis**.

v. Leukocyte Margination

When blood flow slows, **leukocytes**, particularly **neutrophils**, move toward the vessel walls, adhere to the **endothelium**, and then migrate into the surrounding tissues. This process occurs in the sequence of **margination** → **adhesion** → **migration**, resulting in the **attachment of WBCs to the endothelium** followed by **their movement into the tissues**.

➤ Triple Response

The **Lewis experiment** provides a clear demonstration of the haemodynamic changes that occur during inflammation. In this test, the inner surface of the forearm is stroked firmly with a blunt instrument, such as a pencil, and the resulting skin changes are observed. The response produced, known as the **triple response** or **red line response**, consists of a series of characteristic vascular reactions.

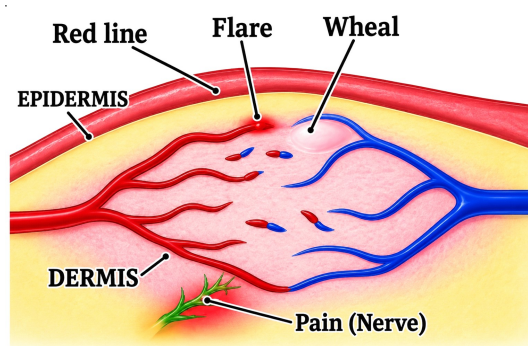


Fig.3.21: Vascular Changes in the Lewis Triple Response

Table 3.13: Triple Response in Acute Inflammation

RESPONSE	FEATURES	CAUSE (MECHANISM)	ONSET	APPEARANCE	RESULT / SIGNIFICANCE
Red line	A narrow red line appears at the site of injury.	Capillary dilation in the injured area.	Appears within seconds.		Indicates immediate vascular response.
Flare	Surrounding redness develops around the red line.	Dilation of nearby arterioles in surrounding skin.	Develops within seconds to a few minutes.		Increases blood flow to the area.
Wheal	Local swelling (raised, pale swelling) appears at the site.	Increased vascular permeability causes fluid accumulation in tissues.	Develops within minutes.		Causes swelling and may contribute to pain.

2. Altered Vascular Permeability

During inflammation, fluid accumulates in the interstitial tissues, leading to **edema**. In the early stage, **vasodilatation** increases blood flow and hydrostatic pressure, resulting in the leakage of protein-poor fluid known as **transudate**. As the inflammatory process continues, vascular permeability increases,

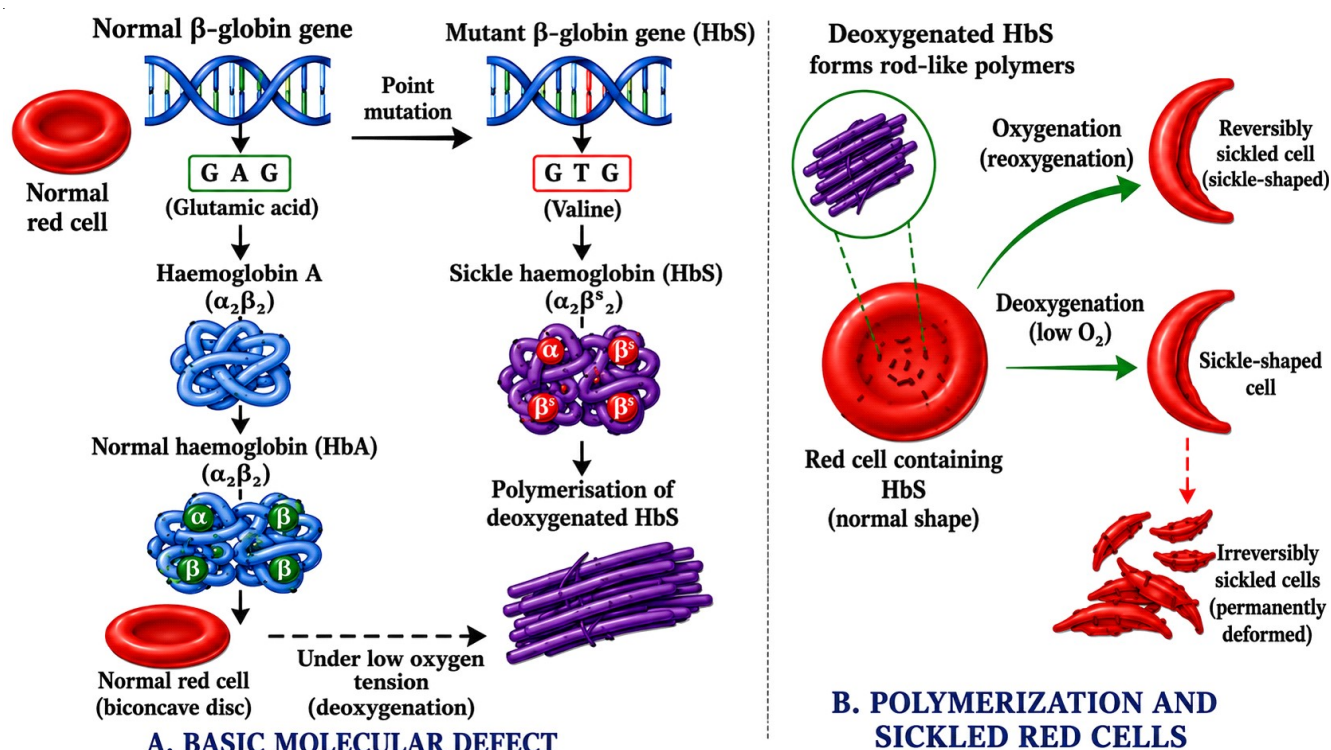


Fig.3.31: Molecular Basis and Polymerization Mechanism of Sickie Cell Disease.

3.3.4. Thalassemia

□ Introduction

- **Thalassaemia** is a group of inherited blood disorders characterized by reduced synthesis of one or more globin chains of hemoglobin, leading to defective red blood cell production and anemia. Normal adult hemoglobin (HbA) consists of two α -globin and two β -globin chains; mutations affecting either chain result in an imbalance of globin synthesis.
- Thalassaemias are quantitative disorders caused by decreased globin chain production. The condition was first described in Mediterranean populations and is therefore also known as **Mediterranean anemia**. The term thalassaemia is derived from the Greek word *thalassa*, meaning “sea.” Based on the affected globin chain, thalassaemia is classified into **α -thalassaemia** and **β -thalassaemia**.

□ Genetic Mechanisms and Classification of Thalassaemia

- Thalassaemia is an inherited disorder of hemoglobin synthesis. Normal adult hemoglobin (HbA) consists of two α -globin and two β -globin chains. The α -globin chains are encoded by **HBA genes on chromosome 16**, whereas the β -globin chains are encoded by **HBB genes on chromosome 11**.
- Based on the affected globin chain, thalassaemia is classified into **α -thalassaemia** and **β -thalassaemia**. In both conditions, the globin chains are structurally normal, but their synthesis is reduced. Each type may occur as a **minor (trait)** form in the heterozygous state or a **major** form in the homozygous state. The minor form is usually asymptomatic, whereas the major form presents as severe congenital hemolytic anemia.

49. Which antigen determines whether an individual is Rh-positive

- (a) A antigen (b) B antigen
(c) D antigen (d) H antigen

50. Yellow bone marrow can revert to red bone marrow primarily during

- (a) Hypercalcemia (b) Severe blood loss
(c) Hypertension (d) Hyperglycemia

ANSWER KEY

1-b	2-a	3-a	4-a	5-b	6-c	7-d	8-d	9-b	10-d
11-c	12-a	13-c	14-a	15-a	16-d	17-c	18-d	19-b	20-d
21-a	22-c	23-b	24-d	25-c	26-c	27-d	28-c	29-c	30-b
31-c	32-d	33-b	34-c	35-b	36-a	37-b	38-c	39-b	40-d
41-b	42-c	43-b	44-c	45-a	46-b	47-b	48-d	49-c	50-b

Most Important Short Answer Type Questions

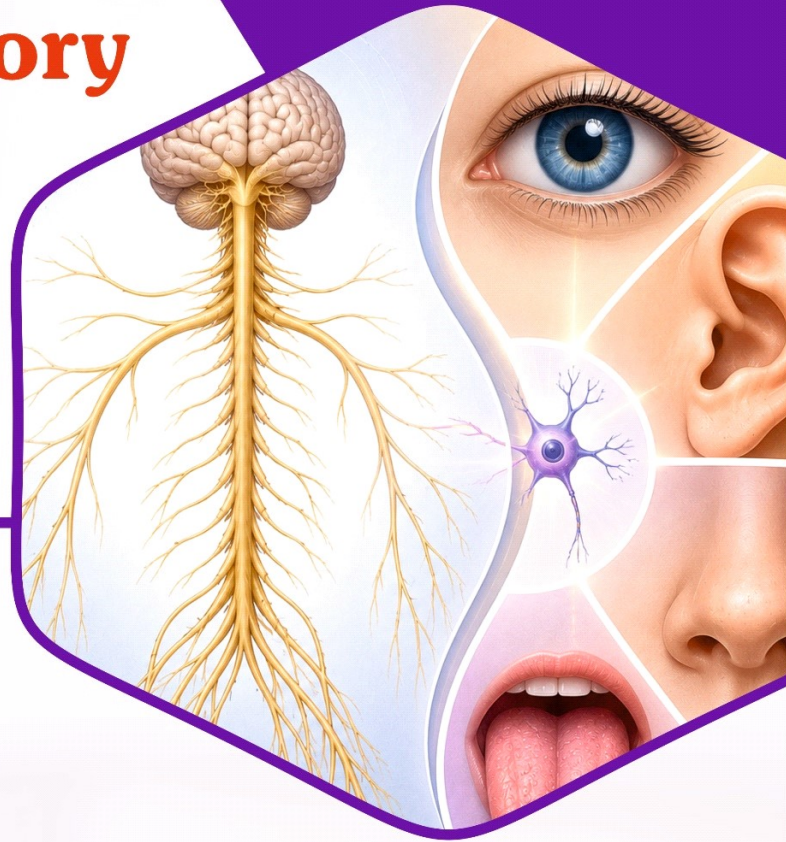
1. Differentiate between intracellular fluid (ICF) and extracellular fluid (ECF).
2. Write a short note on electrolyte balance.
3. Explain the role of erythropoietin in erythropoiesis.
4. Write the functions of hemoglobin.
5. Explain Landsteiner's law.
6. What is cross-matching? Why is it performed before blood transfusion.
7. Explain Rh incompatibility.
8. Write the functions of the lymphatic system.
9. Differentiate between acute and chronic inflammation.
10. Explain granulation tissue.
11. Write the factors affecting wound healing.
12. Explain the laboratory findings of iron deficiency anemia.
13. Differentiate between vitamin B₁₂ deficiency anemia and folic acid deficiency anemia.
14. Explain the laboratory findings of megaloblastic anemia.
15. Differentiate between blood typing and cross-matching.
16. Differentiate between α -thalassemia and β -thalassemia.
17. Write a short note on Paroxysmal Nocturnal Hemoglobinuria (PNH).
18. Explain the clinical features and laboratory diagnosis of Hemophilia A.
19. Explain the role of HbS in the development of sickle cell anemia.
20. Write a short note on hematopoietic growth factors.

Long Answer Type Questions

1. Describe blood with its composition, properties and physiological functions.
2. Explain hematopoiesis and blood cell formation.
3. Explain the mechanism of blood coagulation.
4. Describe the ABO blood group system and explain the importance of blood grouping and blood transfusion.
5. Describe the lymphatic system and explain the structure and functions of lymphoid organs.
6. Describe tissue repair and explain the stages of wound healing.
7. Explain anemia and discuss its classification, pathophysiology and clinical features.
8. Describe iron deficiency anemia with its pathophysiology, clinical features and laboratory findings.
9. Explain megaloblastic anemia and discuss vitamin B₁₂ deficiency anemia and folic acid deficiency anemia.
10. Explain thalassemia with its etiology, pathophysiology, clinical features, laboratory diagnosis and complications.

Unit IV

Peripheral Nervous System & Sensory Organs



Syllabus

Peripheral nervous system

Classification of peripheral nervous system: structure and functions of sympathetic and parasympathetic nervous system. Origin and functions of spinal and cranial nerves.

Special senses

Structure and functions of eye, ear, nose and tongue. Pathophysiology of special sense disorders- glaucoma, cataract, myopia, otitis externa, otitis media, vertigo and anosmia



PERIPHERAL NERVOUS SYSTEM & SENSORY ORGANS



LEARNING OBJECTIVES

After studying this chapter, the learner will be able to:

- Explain the organization and functions of the Peripheral Nervous System, including the somatic and autonomic divisions.
- Describe the structure and functions of cranial nerves, spinal nerves, ganglia, and nerve plexuses.
- Discuss the anatomy and physiology of the eye, ear, nose, and tongue as major sense organs.
- Explain the mechanisms of vision, hearing, balance, smell, and taste.
- Understand the role of sensory pathways and neural control in perception and coordination.
- Describe the etiology, clinical features, diagnosis, and management of common disorders affecting the eye, ear, and nose.

❑ INTRODUCTION TO PERIPHERAL NERVOUS SYSTEM

The **Peripheral Nervous System (PNS)** is the part of the nervous system that lies outside the central nervous system. It consists of all the nerves that arise from the brain and spinal cord and extend to different parts of the body.

The main function of the PNS is to establish communication between the central nervous system and the organs, muscles, glands, and tissues of the body. It carries sensory information from the body to the brain and spinal cord and sends motor commands from the central nervous system to the muscles and glands.

4.1. CLASSIFICATION OF PERIPHERAL NERVOUS SYSTEM

The Peripheral Nervous System is broadly classified into two main divisions:

1. **Somatic Nervous System (SNS)**
2. **Autonomic Nervous System (ANS)**

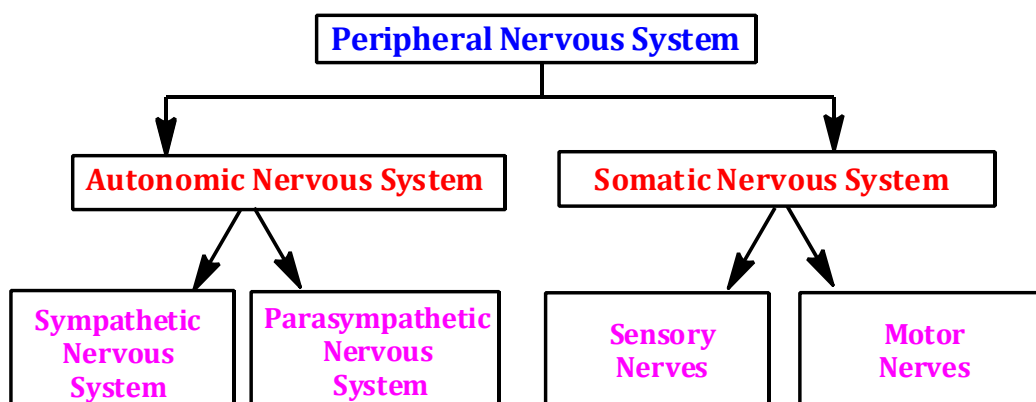


Fig. 4.1: Organisation of Peripheral Nervous System

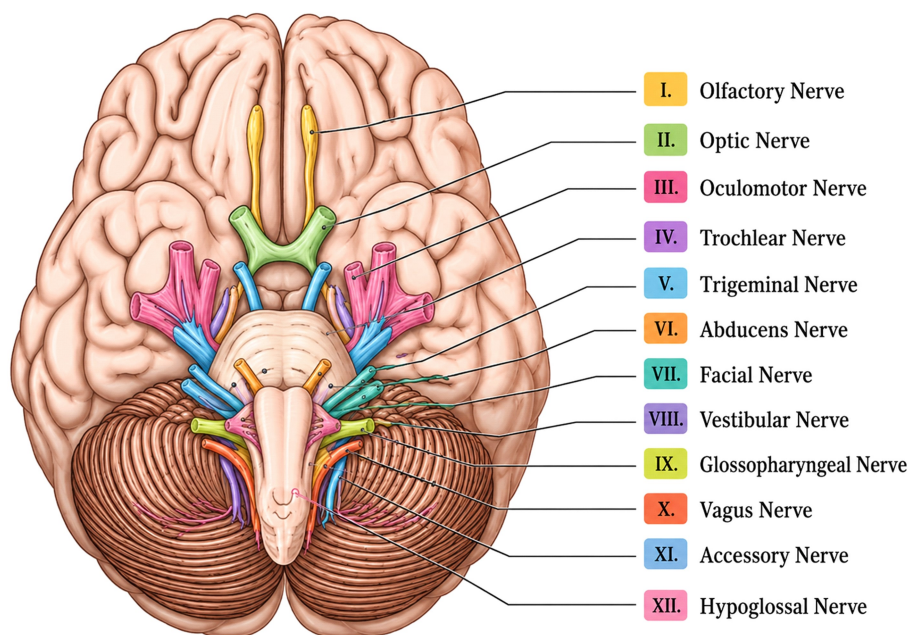


Fig. 4.6: 12 Pairs of Cranial Nerve

❑ SPINAL NERVES

Spinal nerves serve as the communication pathway between the spinal cord and different regions of the body. The spinal cord ends at the lower border of the L1 vertebra.

There are **31 pairs of spinal nerves**. These nerves are named according to the region of the vertebral column from which they arise. They include cervical, thoracic, lumbar, sacral, and coccygeal spinal nerves. The spinal nerves emerge from the spinal cord and pass through the intervertebral foramina of the vertebrae.

Since the spinal cord is shorter than the vertebral column, the nerve roots of the lumbar, sacral, and coccygeal nerves travel within the vertebral canal before exiting at their respective levels.

Table: 4.3 Classification of Spinal Nerves

SPINAL NERVES [C ₈ T ₁₂ L ₅ S ₅ Co ₁]	NUMBER	TOTAL
Cervical spinal nerves (C1-C8)	8	31
Thoracic spinal nerves (T1-T12)	12	
Lumbar spinal nerves (L1-L5)	5	
Sacral spinal nerves (S1-S5)	5	
Coccygeal spinal nerves (Co1)	1	

❖ Nerve Plexuses

A nerve plexus is a network formed by the anterior rami of spinal nerves. These plexuses distribute nerve fibres to different parts of the body and provide both sensory and motor functions. The major nerve plexuses are the cervical, brachial, lumbar, sacral, and coccygeal plexuses.

1. Cervical Plexus

The cervical plexus is formed by the anterior rami of spinal nerves **C1 to C4** with a contribution from **C5**. It is located deep in the neck and supplies the skin and muscles of the head, neck, and shoulder region. The most important branch is the **phrenic nerve**, which supplies the **diaphragm**, the main muscle responsible for breathing. It also provides sensory supply to parts of the shoulder and neck.

3. Eyelashes

Eyelashes are short, curved hairs present along the free margins of the eyelids. They protect the eyes by preventing dust and small foreign particles from entering.

4. Lacrimal Apparatus

The lacrimal apparatus consists of glands and ducts responsible for the production and drainage of tears. Tears keep the eye moist, clean, and protected.

➤ Components of the Lacrimal Apparatus

- Lacrimal gland and ducts
- Lacrimal canaliculi
- Lacrimal sac
- Nasolacrimal duct

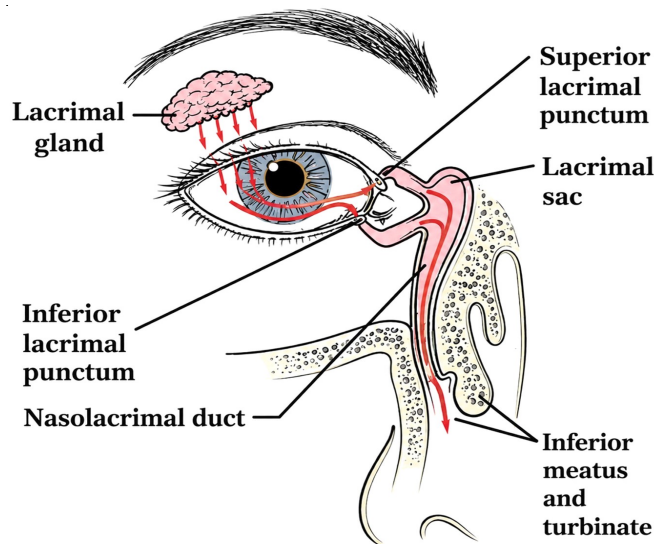


Fig. 4.11: Lacrimal Apparatus

5. Conjunctiva

The conjunctiva is a thin, transparent membrane that lines the inner surface of the eyelids and covers the front part of the eyeball. It forms a protective sac when the eyelids are closed and helps protect the cornea. The conjunctiva is highly vascular and made of columnar epithelium.

6. Eyelid Margins

The eyelid margins contain sebaceous and tarsal glands that secrete oily substances. These secretions help reduce evaporation of tears and keep the eyes lubricated.

7. Extrinsic Eye Muscles

There are six extrinsic eye muscles attached to the eyeball. These muscles are responsible for moving the eye upward, downward, medially, laterally, and in different directions.

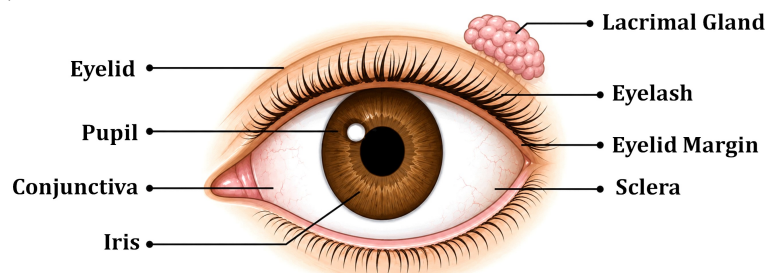


Fig. 4.12: Parts of Eye

The ear is anatomically divided into **three main parts**:

1. External Ear
2. Middle Ear
3. Internal (Inner) Ear

Table: 4.4 Parts and Description/Functions of Ear

Part of Ear	Components	Description / Functions
External Ear	Auricle (Pinna)	Visible cartilaginous part of the ear that collects sound waves and directs them into the external auditory canal.
	External Auditory Canal (External Acoustic Meatus)	A tube about 2.5 cm long lined with skin and ceruminous glands. These glands produce earwax (cerumen), which traps dust, microorganisms, and foreign particles.
	Tympanic Membrane (Eardrum)	Thin semitransparent membrane separating the external ear from the middle ear. It vibrates in response to sound waves and converts them into mechanical vibrations.
Middle Ear (Tympanic Cavity)	Auditory Ossicles	Three smallest bones of the body that transmit and amplify sound vibrations.
	Malleus	Attached to the tympanic membrane.
	Incus	Connects the malleus to the stapes.
	Stapes	Smallest bone of the body; its footplate fits into the oval window of the inner ear.
	Eustachian (Auditory) Tube	Connects the middle ear to the nasopharynx and equalizes air pressure on both sides of the tympanic membrane. Helps maintain normal hearing.
	Oval Window	Transmits vibrations from the middle ear to the inner ear.
	Round Window	Allows pressure release within the inner ear.
Inner Ear (Labyrinth)	Bony Labyrinth	System of hollow cavities within the temporal bone filled with perilymph fluid. Responsible for hearing and balance.
	Cochlea	Spiral-shaped structure responsible for hearing. Contains the organ of Corti with sensory hair cells.
	Vestibule	Central part of the inner ear involved in maintaining static balance and head position.
	Semicircular Canals	Three canals arranged at right angles that detect rotational movements of the head and help maintain dynamic balance.

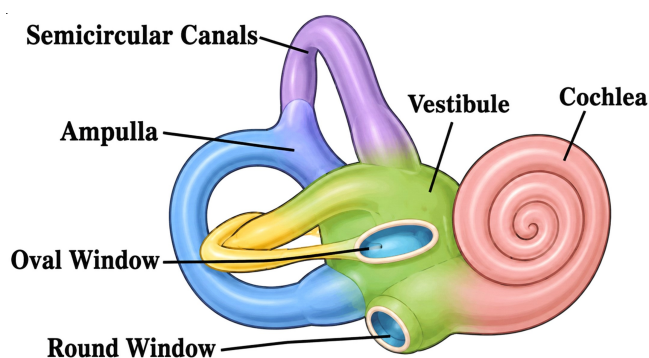


Fig. 4.16: Bony Labyrinth of Ear

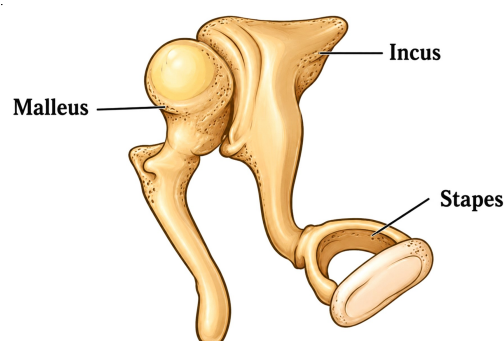


Fig. 4.17: Ossicles

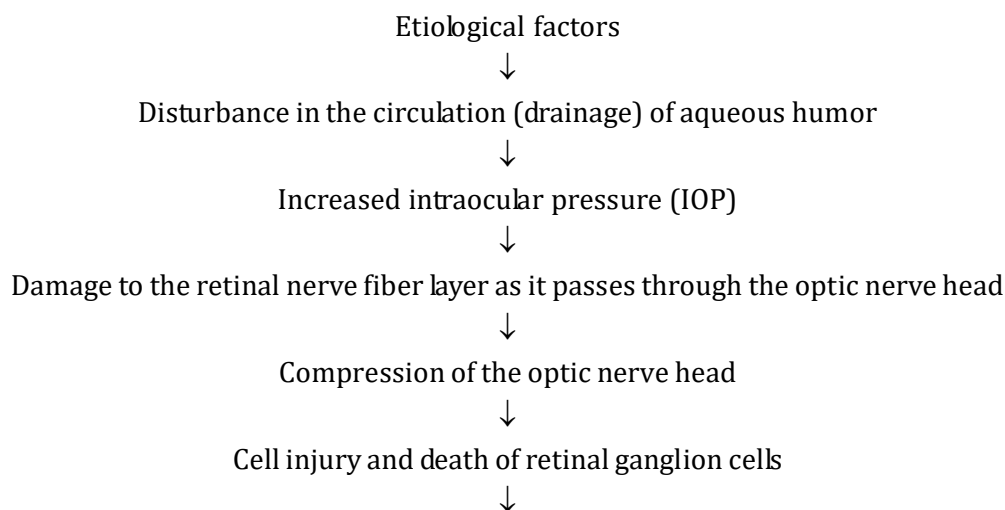
outflow of aqueous humor becomes gradually reduced. There is no visible blockage in the drainage pathway; however, intraocular pressure slowly increases over time. The exact cause of POAG is still not clearly understood. It is believed that microscopic obstruction within the drainage system beyond the limbus may interfere with the normal drainage of aqueous humor. The gradual rise in intraocular pressure can eventually damage the optic nerve.

2. Primary Angle-closure Glaucoma (PACG)

Primary angle-closure glaucoma (PACG) occurs when there is a visible blockage in the drainage pathway of aqueous humor. In this condition, the iris is pushed forward against the cornea, which obstructs the drainage angle and prevents the normal outflow of aqueous humor. As a result, intraocular pressure rises rapidly, usually within a few hours, leading to sudden damage to the optic nerve if not treated promptly.

❖ Pathophysiology of Glaucoma

Sequence of events in Glaucoma:



Progressive optic nerve damage and loss of vision (eventually blindness if untreated).

➤ Causes of Glaucoma

Major cause of glaucoma is the blockage in drainage system of aqueous humor in trabeculae, resulting in increased intraocular pressure. Glaucoma also develops secondary to other disorders, which affect the eyes. Common causes of secondary glaucoma are diabetes, inflammation or injury to eye and excess use of drugs such as corticosteroid.

➤ Symptoms of Glaucoma

Primary open-angle glaucoma is a silent chronic disease without any early symptoms. Symptoms that develop in later stages include heaviness around eyeball, headache and rapid reduction in visual acuity and visual field.

Early symptoms of angle-closure glaucoma are severe pain in eye or eyebrow, headache, nausea, blurred vision and rainbow halo (colored rings) around bulb light. Immediate care should be taken if two or more of these symptoms appear together.

➤ Treatment for Glaucoma

Treatment does not cure the disease but can prevent further damage of optic nerve. Treatment is aimed at lowering the intraocular pressure. It is achieved by using eye drops or medicines alone or in combination with laser treatment. If intraocular pressure cannot be controlled by these methods, surgery is required.

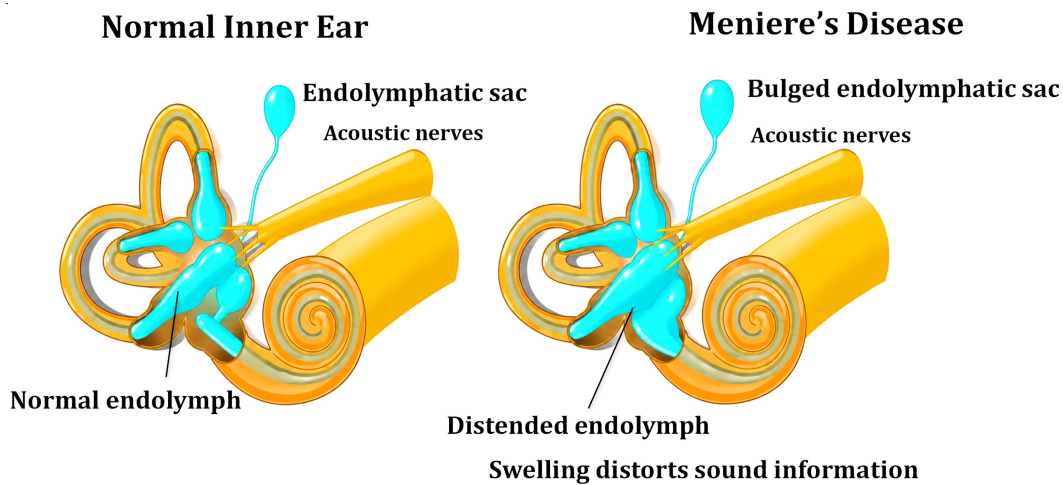


Fig. 4.30: Comparison of Normal Inner Ear and Meniere's Disease

➤ **Diagnosis**

Vertigo is diagnosed by:

- Medical history and physical examination
- Hearing tests
- Balance tests
- Dix-Hallpike test for positional vertigo
- CT scan or MRI in suspected neurological causes

➤ **Treatment**

Treatment depends on the underlying cause and may include:

- Vestibular suppressant medications
- Antiemetic drugs for nausea and vomiting
- Antibiotics for ear infections
- Vestibular rehabilitation exercises
- Canal repositioning maneuvers such as the Epley maneuver for BPPV
- Surgical treatment in severe cases

➤ **Prevention and Care**

The following measures may help reduce vertigo episodes:

- Avoid sudden head movements
- Maintain adequate hydration
- Reduce stress and fatigue
- Avoid excessive alcohol intake
- Treat ear infections promptly
- Follow prescribed vestibular exercises



❑ **Anosmia**

Anosmia is the complete loss or absence of the sense of smell. It may occur temporarily or permanently depending on the underlying cause. The condition develops when there is damage or obstruction affecting the olfactory receptors in the nasal cavity, the olfactory nerve, or the areas of the brain responsible for smell perception.

Smell plays an important role in daily life by helping individuals detect odors, enjoy food, and identify dangers such as smoke, spoiled food, or gas leaks. Since the senses of smell and taste are closely related, anosmia may also reduce the ability to appreciate flavors of food.

Unit V

Cardiovascular System



Syllabus

Anatomy of heart, blood circulation, blood vessels, structure and functions of artery, vein and capillaries, elements of conduction system of heart and heartbeat, its regulation by autonomic nervous system, cardiac output, cardiac cycle. Regulation of blood pressure, pulse, electrocardiogram.

Pathophysiology of hypertension, cardiac arrhythmias, congestive heart failure, ischemic heart disease (angina, myocardial infarction, atherosclerosis and coronary artery disease).



CARDIOVASCULAR SYSTEM



LEARNING OBJECTIVES

After studying this chapter, the learner will be able to:

- Describe the anatomy and functional organization of the heart and blood vessels.
- Explain heart valves, blood circulation, cardiac conduction, and the cardiac cycle.
- Discuss the electrocardiogram (ECG), heart sounds, blood pressure, and cardiovascular regulation.
- Differentiate the structure and functions of arteries, veins, and capillaries.
- Explain the pathophysiology and risk factors of atherosclerosis.
- Discuss the clinical features, diagnosis, and management of coronary artery disease (CAD), angina pectoris, and myocardial infarction (MI).
- Describe the pathophysiology, clinical features, and management of heart failure.
- Explain the classification, complications, and management of hypertension.

❑ Introduction

The **cardiovascular system** consists of three main components: **blood, heart, and blood vessels**. The term “**cardio**” means **heart**, while “**vascular**” refers to **blood vessels**. Blood transports oxygen, carbon dioxide, nutrients, hormones, and waste products throughout the body. It helps regulate important body processes and protects against diseases. The heart acts as a muscular pump that maintains continuous blood flow. It also helps regulate body temperature and meet the body’s oxygen needs.



5.1. ANATOMY OF HEART

- The human heart is a **muscular, fist-sized organ** located in the **middle mediastinum**. It lies **behind the sternum** and adjacent costal cartilages, with a **slight tilt toward the left** side of the body. The heart consists of four chambers and is enclosed within a protective sac known as the pericardium. Approximately **one-third of the heart lies to the right of the median plane, while two-thirds lies to the left**.
- The apex of the heart is the pointed tip of the left ventricle and rests on the diaphragm, whereas the base of the heart is formed **by the atria**. The right and left halves of the heart are separated by a **septum**. The heart works as a **dual-pump system** and circulates blood throughout the body through coordinated contraction and relaxation cycles. The heart measures approximately **12 × 9 cm**, and its average weight is about **300 g in males and 250 g in females**.

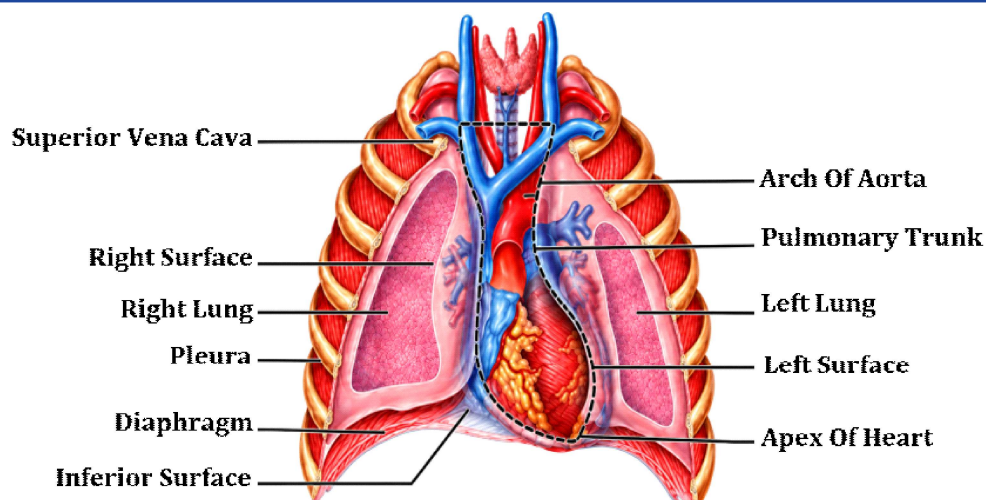


Fig.5.1: Anterior view of the Heart in the Thoracic cavity

➤ Pericardium

The pericardium is a **protective membrane** that surrounds the heart and holds it in position within the **mediastinum** while allowing normal movement during contraction. It consists of two layers:

I. The Fibrous Pericardium

II. The Serous Pericardium

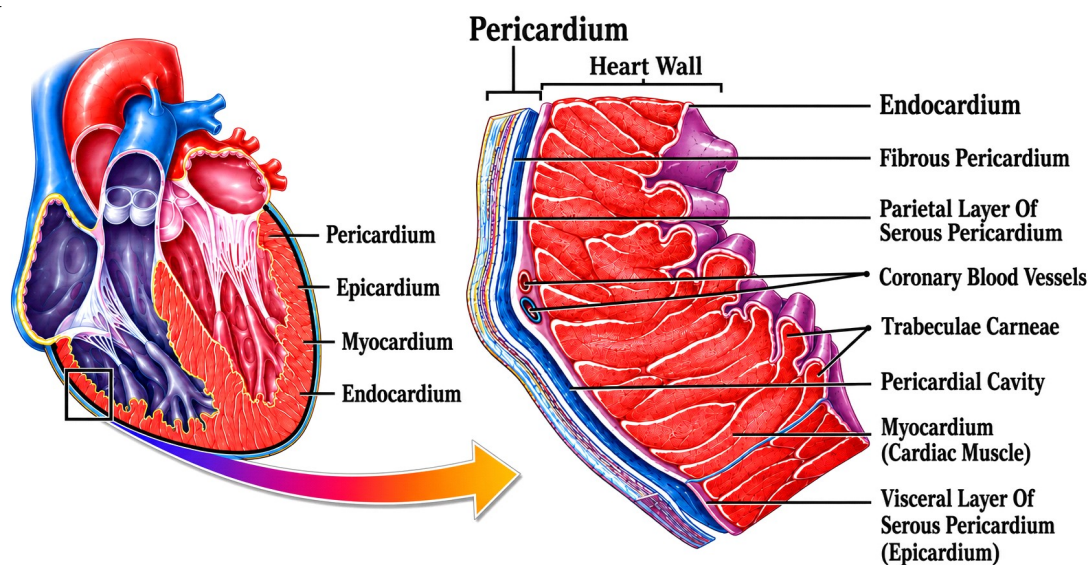


Fig.5.2: Structure of the Pericardium

- The **fibrous pericardium** is a tough, inelastic connective tissue layer that protects the heart, prevents overstretching, and anchors it to surrounding structures, including the diaphragm.
- The **serous pericardium** is a thin double-layered membrane consisting of an outer **parietal layer** and an inner **visceral layer (epicardium)**, which covers the heart surface. Between these layers lies the pericardial cavity, containing pericardial fluid that reduces friction as the heart beats.

❑ Layers of the Heart Wall

The wall of the human heart is composed of three distinct layers: **epicardium**, **myocardium**, and **endocardium**. These layers provide structural support, strength, and protection to the heart.

body except the lungs. These arteries divide into smaller arterioles that lead to systemic capillaries, where the exchange of nutrients and gases occurs through the thin capillary walls. During this exchange, **blood delivers oxygen (O_2)** to tissues and **collects carbon dioxide (CO_2)**. The capillaries then join to form venules, which carry **deoxygenated blood away from the tissues**. Venules merge into larger veins that return the blood to the right atrium.

❖ Pulmonary Circulation

The right side of the heart pumps **deoxygenated (oxygen-poor) blood** into the **pulmonary circulation**. The right ventricle ejects blood into the pulmonary trunk, which divides into **pulmonary arteries** carrying blood to the lungs. In the pulmonary capillaries, **blood releases carbon dioxide (CO_2) for exhalation and takes up oxygen (O_2) from inhaled air**. The freshly oxygenated blood then returns to the left atrium through the **pulmonary veins**.

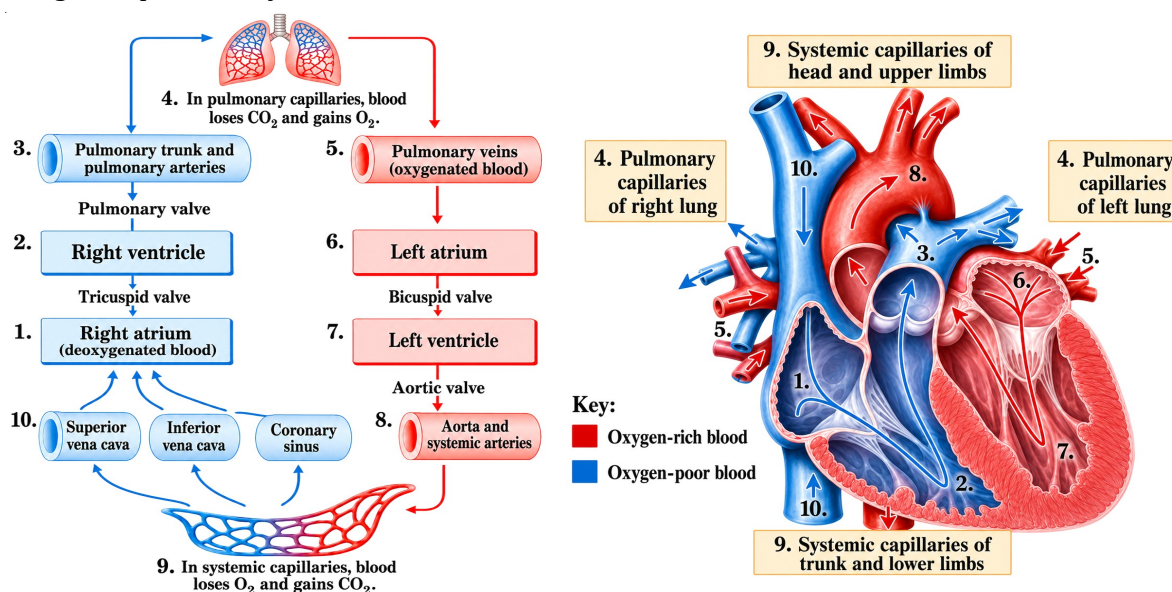


Fig.5.6: Overview of Pulmonary and Systemic Blood Circulation

❖ Coronary Circulation

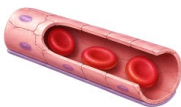
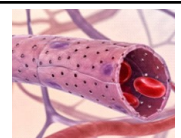
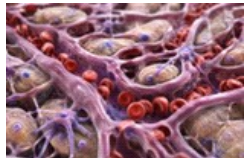
- Nutrients cannot diffuse rapidly enough from the heart chambers to supply all layers of the heart wall. Therefore, the **myocardium** has its own blood supply known as **coronary circulation (cardiac circulation)**. The coronary arteries arise from the ascending aorta and surround the heart like a crown.
- During relaxation of the heart, the high pressure in the aorta **pushes blood through the coronary arteries** to supply the myocardium. During heart contraction, blood flow in the coronary arteries decreases because these vessels are compressed. The blood then passes through the capillaries and finally drains into the **coronary veins**.

5.3. BLOOD VESSELS

- Blood vessels are **hollow, tubular channels** of the circulatory system that form a vast, closed, and pressurized network throughout the body. They transport blood between the heart and tissues, **carrying blood cells, oxygen, nutrients, hormones, and other essential substances** to cells while removing carbon dioxide and metabolic waste products.
- Blood vessels also help maintain blood circulation and **regulate blood pressure** and blood supply to various tissues. Some tissues, such as cartilage, epithelium, and the lens and cornea of the eye, are avascular because they do not contain blood vessels.

- Tissues with high metabolic activity, such as the muscles, brain, liver, kidneys, and nerves, have dense capillary networks, whereas tissues with low metabolic activity have fewer capillaries. **Capillaries are absent in the cartilage, cornea, lens, and epithelia.** Thus, they play a vital role in maintaining normal cellular function and tissue health.

Table 5.3: Types of Capillaries

TYPE / SYSTEM	ILLUSTRATION	FEATURES	LOCATION / EXAMPLES
Continuous Capillaries		Have continuous endothelial lining with small intercellular clefts and allow limited exchange of substances.	Skin, muscles, lungs, and central nervous system
Fenestrated Capillaries		Contain small pores (fenestrations) that increase permeability and allow rapid exchange.	Kidneys, intestinal villi, endocrine glands, and choroid plexus
Sinusoids		Wide and irregular capillaries with large gaps and incomplete basement membrane allowing passage of proteins and blood cells.	Liver, spleen, red bone marrow, pituitary gland, and adrenal glands
Portal System		Blood passes through two capillary networks before returning to the heart.	Hepatic portal and hypophyseal portal system

❖ Venules

- Venules are the **smallest veins** and are **formed by the joining of capillaries**. They collect blood from capillary networks within tissues and begin its return to the heart, directing it into progressively larger veins. Venules have **thin walls** and do not maintain their shape like arteries.

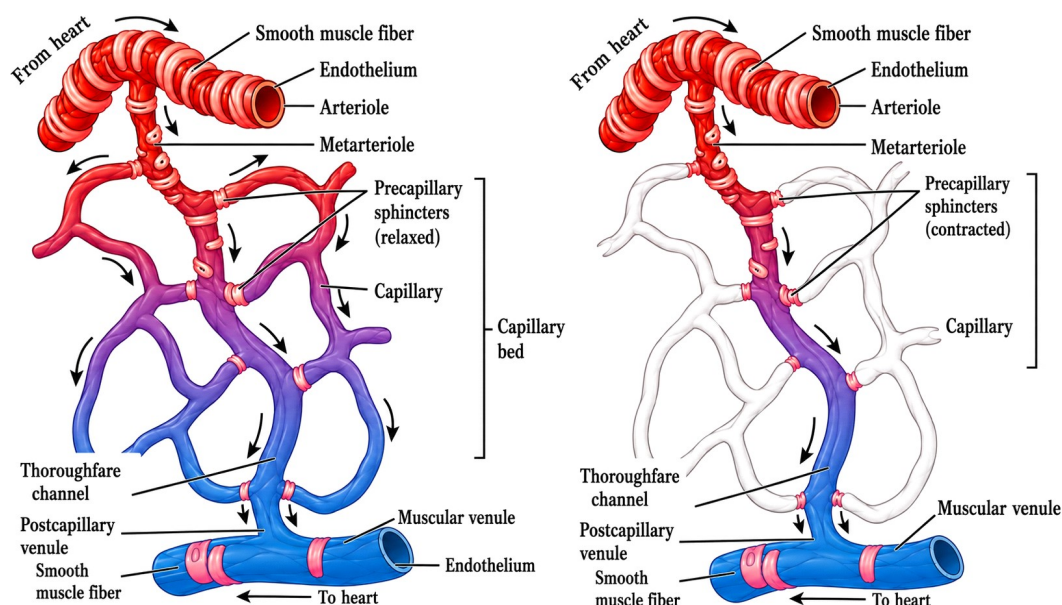


Fig.5.10: Microvascular Blood Flow in Relaxed and Contracted Precapillary Sphincter

(a) Sphincters relaxed: blood flowing through capillaries

(b) Sphincters contracted: blood flowing through thoroughfare channel

II. Vasodilation Effect

A. Discharge of Noradrenergic Sympathetic Nerves

Discharge of noradrenergic sympathetic nerve fibers causes vasodilation in most parts of the body.

B. Sympathetic Cholinergic Vasodilator Nerves

- Sympathetic **cholinergic vasodilator fibers** supply the **skeletal muscles, heart, lungs, kidneys, liver, and uterus** and act alongside **adrenergic vasoconstrictor sympathetic fibers**.
- They primarily release **acetylcholine** as their neurotransmitter, although **vasoactive intestinal peptide (VIP)** may also be released. These fibers originate from the **cerebral cortex**, do **not exhibit tonic discharge**, and are activated mainly during **exercise, childbirth, and other emergencies** to increase blood flow to specific organs as needed.

C. Parasympathetic Vasodilator Nerves

- Parasympathetic **vasodilator nerves** are present only in a few parts of the body and have a **limited role in general systemic circulation**.
- The **nervi erigentes (pelvic splanchnic nerves)** supply the erectile tissues, while **cranial parasympathetic fibers** innervate the salivary and other digestive glands. Their activation mainly supports **specific biological functions**, such as erection and glandular secretion.

5.5. CARDIAC CYCLE, HEART SOUNDS AND CARDIAC OUTPUT

The **cardiac cycle** is the sequence of events that occurs during **one complete heartbeat**, including the **systole (contraction)** and **diastole (relaxation)** of both the atria and ventricles. Under resting conditions, one cardiac cycle is completed in about **0.8 seconds**. During this cycle, **heart sounds** are produced, which can be heard using a **stethoscope**.

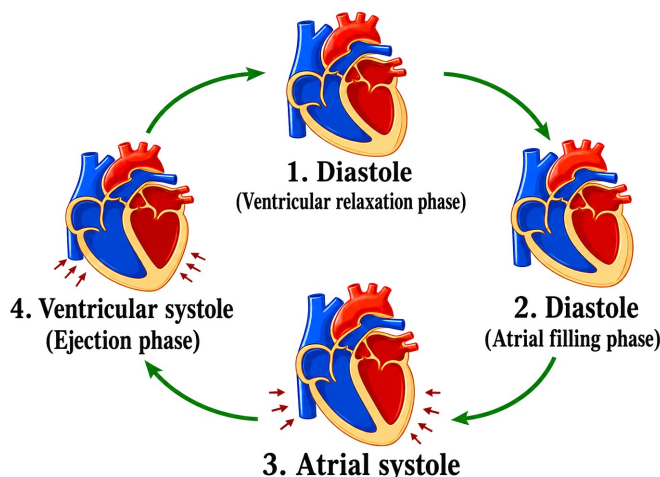


Fig.5.14: Mechanical Events of the Cardiac Cycle

□ Pressure and Volume Changes During the Cardiac Cycle

- During each **cardiac cycle**, the **atria and ventricles contract and relax alternately** to pump blood efficiently. Blood always flows from **areas of higher pressure to areas of lower pressure**, and contraction of a heart chamber increases the pressure within that chamber.
- The **pressure and volume changes** in the heart occur in coordination with the **electrocardiogram (ECG)**. The **left side of the heart generates higher pressure** than the right side, although **both ventricles pump the same amount of blood** with each heartbeat. At a heart rate of **75 beats per minute**, one complete cardiac cycle takes about **0.8 seconds**.

- The pulse is an important clinical indicator used to assess the **heart rate, rhythm, and strength of blood flow** and is usually measured in **beats per minute (BPM)**. The most common sites for measuring the pulse are the **radial artery at the wrist** and the **carotid artery in the neck**.

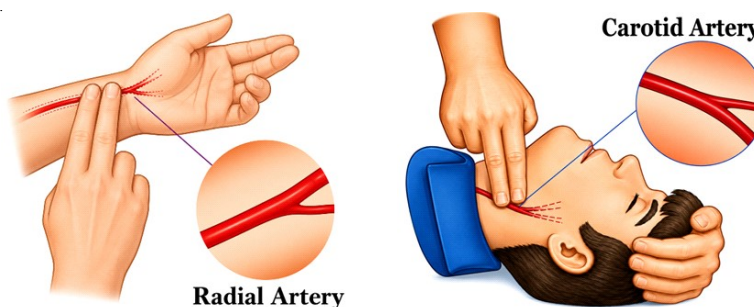


Fig.5.18: Sites for Palpation of the Radial and Carotid Pulse

❑ Blood Pressure

Blood pressure is the **force exerted by circulating blood on the walls of the arteries** during the pumping action of the heart. It is measured using an instrument called a **sphygmomanometer**, which is available in three types:

- I. **Mercury Sphygmomanometer**
- II. **Aneroid Sphygmomanometer**
- III. **Digital Sphygmomanometer.**

Under normal conditions, the **systolic blood pressure** is about **120 mm Hg**, while the **diastolic blood pressure** is about **80 mm Hg**. Therefore, a normal blood pressure reading is expressed as **120/80 mm Hg**.

Table 5.10: Clinical Classification of Blood Pressure

BLOOD PRESSURE CATEGORY	SYSTOLIC (mm Hg)	DIASTOLIC (mm Hg)
Optimal	< 120	< 80
Normal	120–129	80–84
Prehypertension (High-Normal)	130–139	85–89
Grade 1 (Mild) Hypertension	140–159	90–99
Grade 2 (Moderate) Hypertension	160–179	100–109
Hypertensive Crisis (Severe Hypertension)	≥ 180	≥ 120

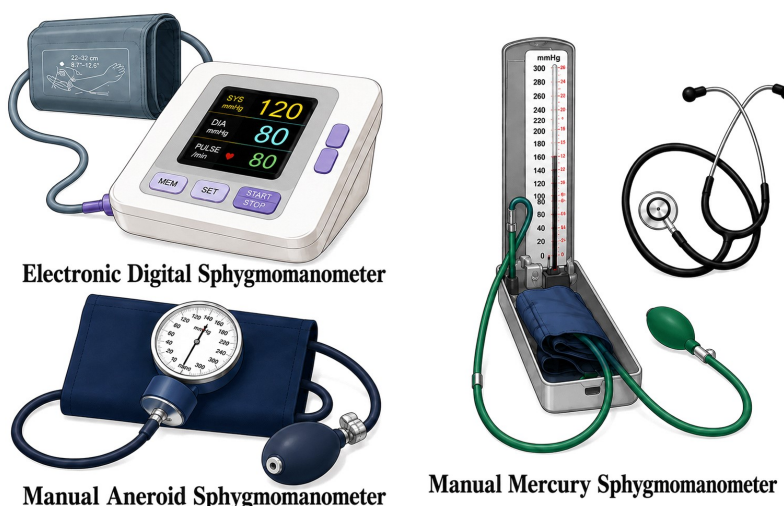


Fig.5.19: Types of Sphygmomanometers Used for Blood Pressure Measurement

- **Pulse Pressure:** Pulse pressure is the difference between systolic and diastolic blood pressure. It indicates the tension in the arterial walls, and its normal value is about **40 mm Hg**.

Mean Arterial Pressure (MAP): Mean arterial pressure (MAP) is the average arterial pressure maintained throughout the cardiac cycle. During the cardiac cycle, about **two-thirds of the time is spent in diastole and one-third in systole**.

Table 5.12: Different Representation of Blood Pressures

BLOOD PRESSURE TYPE	DEFINITION	NORMAL VALUE
Systolic Blood Pressure (SBP)	Pressure in the arteries during myocardial contraction.	120 mm Hg
Diastolic Blood Pressure (DBP)	Pressure in the arteries during myocardial relaxation.	80 mm Hg
Pulse Pressure (PP)	Difference between systolic and diastolic blood pressure.	40 mm Hg
Mean Arterial Pressure (MAP)	Average arterial pressure throughout the cardiac cycle. $MAP = 1/3 (SBP) + 2/3 (DBP)$	93.3 mmHg

❑ Types of Hypertension

1. Primary Hypertension

Primary hypertension develops without a clearly identifiable cause and is the most common type of hypertension. It accounts for approximately **90–95%** of all hypertension cases and occurs independently without being caused by another medical disorder.

2. Secondary Hypertension

Secondary hypertension occurs due to an underlying medical condition, and its exact cause is usually known. Treatment of the underlying disease can help control or cure hypertension.

3. Isolated Systolic Hypertension

Isolated systolic hypertension is characterized by an elevated **systolic blood pressure (≥ 140 mm Hg)** while the **diastolic blood pressure remains normal (≤ 90 mm Hg)**. It involves an increase only in systolic pressure without a significant rise in diastolic pressure.

4. Hypertensive Crisis

Hypertensive crisis is a severe elevation of blood pressure above **180/120 mm Hg**. It is classified into **hypertensive emergency** and **hypertensive urgency**.

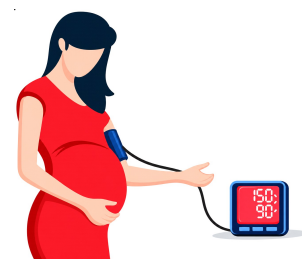
a. Hypertensive Emergency: Hypertensive emergency is a condition in which blood pressure rises to a dangerously high level and causes target organ damage. Complications may include **encephalopathy, intracranial hemorrhage, pulmonary edema, aortic aneurysm, unstable angina, and eclampsia**.

b. Hypertensive Urgency: Hypertensive urgency is characterized by a very high blood pressure without evidence of target organ damage. If left untreated, it may progress to a hypertensive emergency.

5. Hypertension in Pregnancy

Hypertension during pregnancy includes **gestational hypertension, preeclampsia, and eclampsia**.

a. Gestational Hypertension: Gestational hypertension develops **after 20 weeks of pregnancy** and is characterized by elevated blood pressure **without proteinuria**.



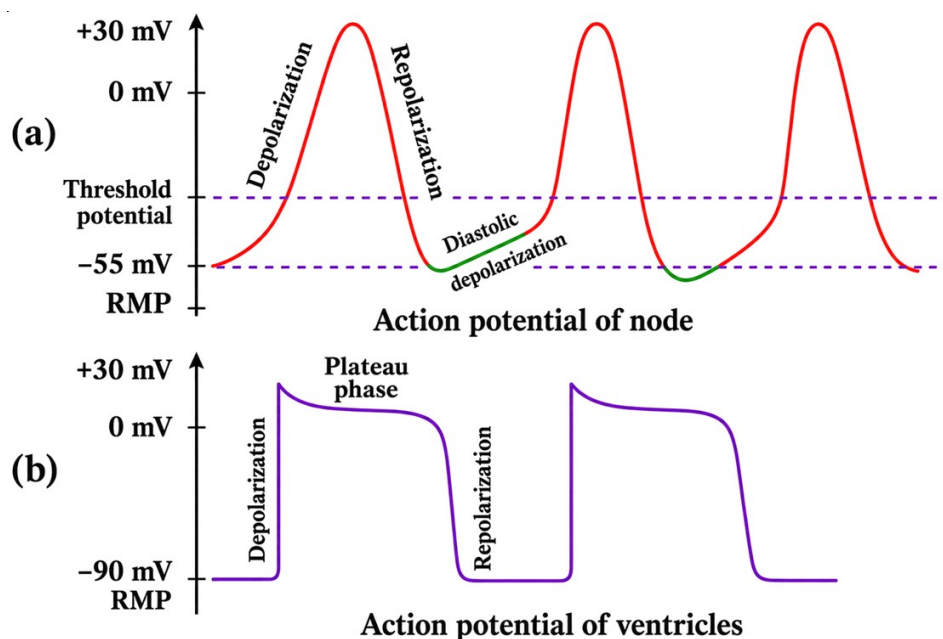


Fig.5.24: Normal Action potential of (a): SA node and (b): Ventricles

□ Types of Arrhythmias

(1) Tachycardia

Tachycardia is an increase in heart rate above **100 beats/min**, whereas the normal heart rate is about **72 beats/min**. It may be caused by increased body temperature, increased sympathetic activity, hyperthyroidism, or drugs such as atropine. Beta blockers, including atenolol and propranolol, may help control tachycardia.

(2) Bradycardia

Bradycardia is a condition in which the heart rate falls below 60 beats per minute. It may be caused by **hypothyroidism, increased parasympathetic activity, or toxicity from drugs** such as acetylcholinesterase inhibitors. Anticholinergic drugs, including atropine, are used to increase the heart rate.

(3) Atrial Fibrillation

- Atrial fibrillation is a type of arrhythmia in which the atria contract irregularly rather than as one coordinated unit. As a result, atrial impulses spread unevenly, causing loss of coordinated pumping.
- The atrial rate may increase up to **500 beats/minute**, but not all impulses reach the ventricles due to **AV nodal blockage**. Ventricular conduction may occur in ratios such as **3:1** or **4:1**, and the ventricular rate is usually around **125–150 beats/minute**.

- **Mechanism of Fibrillation:** Electrical impulses may split into multiple smaller impulses that stimulate different parts of the atria separately. **Functional re-entry** can also cause atrial fibrillation, and **atrial enlargement** creates long conductive pathways that promote **re-entry circuits**.
- **Effects on the Body:** The atria fail to pump blood effectively into the ventricles, but blood still flows passively from the atria to the ventricles. **Passive filling** contributes about **75% of ventricular filling**, while **ventricular pumping efficiency** decreases by about **20–30%**.
- **ECG Changes:** In atrial fibrillation, **P waves** are absent or not clearly visible, while **QRS complexes** usually remain normal. The rapid atrial activity makes the **P waves** indistinguishable on the ECG.

4. Microvascular Angina

- Microvascular angina occurs due to problems in the small blood vessels (microvessels) of the heart instead of the major coronary arteries. These tiny vessels do not function properly, reducing blood flow to the heart muscle.
- The chest pain may be triggered by daily activities, emotional stress, or sometimes without any obvious cause. The pain often lasts longer than 10 minutes and may not respond well to standard angina medicines, making it more difficult to manage.

5. Refractory Angina

- Refractory angina is persistent or recurrent chest pain that continues even after receiving the best available treatments, including medications, angioplasty, or stent placement. It occurs because the heart muscle still does not receive enough blood despite treatment.
- The condition can significantly affect a person's daily activities and quality of life. Management focuses on alternative therapies, lifestyle changes, and pain-relieving strategies to improve blood flow and reduce symptoms.

❖ Pathophysiology of Angina

Angina occurs due to an imbalance between myocardial oxygen supply and oxygen demand. The development of angina is multifactorial and complex, and it results from either **an inadequate oxygen supply to the myocardium or an increased myocardial oxygen demand**.

I. Factors Increasing Myocardial Oxygen Demand

Main determinants include:

- ✓ Increased heart rate
- ✓ Increased cardiac contractility
- ✓ Increased intramyocardial wall tension

According to Laplace's law, wall tension depends on intraventricular pressure. Therefore, an increase in heart rate, myocardial contractility, or ventricular pressure increases wall tension, thereby raising myocardial oxygen demand.

Double Product (DP)

Clinically, myocardial oxygen demand is assessed using:

- Heart rate
- Blood pressure

Double Product (DP) formula:

$$\text{DP} = \text{Heart Rate} \times \text{Blood Pressure}$$

Double product (DP) is an indirect indicator of **myocardial oxygen (O_2) demand** in patients. However, a major limitation of DP is that it does not consider certain factors influencing myocardial oxygen demand.

- Ventricular dilatation
- Ventricular contractility

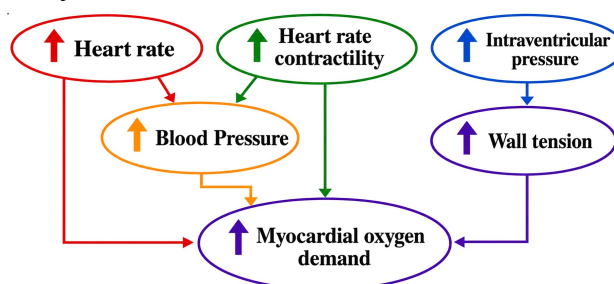


Fig.5.31: Factors that contribute in increasing myocardial oxygen demand.

Cardiac Troponin (cTn)	- Cardiac troponins confirm myocardial injury. - cTnI and cTnT are important cardiac markers. - cTnI is more specific for myocardial infarction. - Troponin levels rise within 2–4 hours. - Levels remain elevated for 4–7 days.
Lactate Dehydrogenase (LDH)	- LDH-1 is the cardiac-specific isoenzyme. - LDH levels rise after 24 hours of injury. - Levels remain elevated for about 7 days.
Creatine Kinase (CK)	- CK-MB is the cardiac-specific isoenzyme. - CK-MB helps diagnose myocardial infarction. - CK levels rise within 2–4 hours. - Levels return to normal within 48 hours.

❑ Types of Myocardial Infarction

1. ST Segment Elevation Myocardial Infarction (STEMI): STEMI is characterized by **ST-segment elevation** on the ECG, and **Q waves** commonly appear, which is why it is also called **Q-wave myocardial infarction**. It is known as **transmural infarction** because the damage involves the full thickness of the heart muscle. STEMI usually occurs due to complete blockage of a major coronary artery and is generally a more severe form of myocardial infarction.

2. Non-ST Segment Elevation Myocardial Infarction (NSTEMI)

NSTEMI does not show ST segment elevation on the ECG, although ST segment depression may be present in some cases. **Q waves are usually absent on the ECG**; therefore, it is also called non-Q wave myocardial infarction. NSTEMI is known as subendocardial infarction because the damage is limited to the inner layer of the heart muscle. It commonly occurs **due to partial blockage of a major coronary artery or complete blockage of a minor artery** and is usually less severe than STEMI.

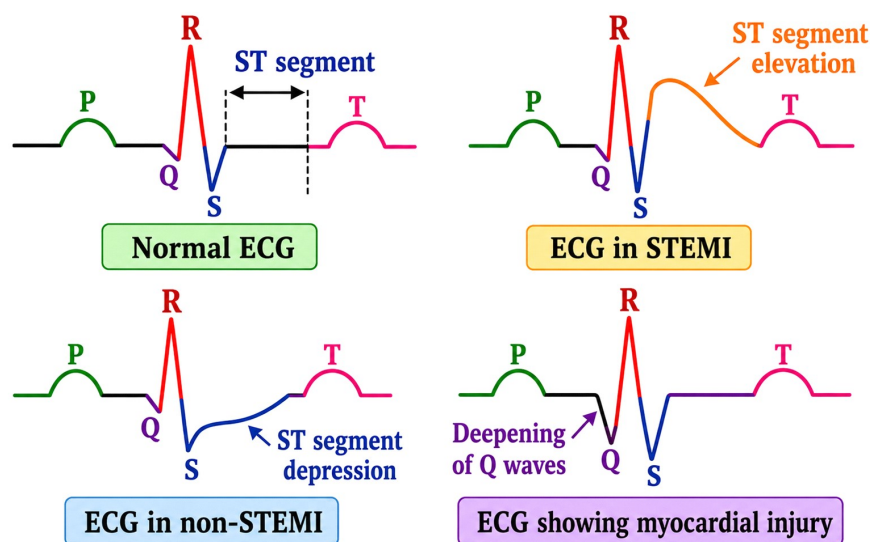
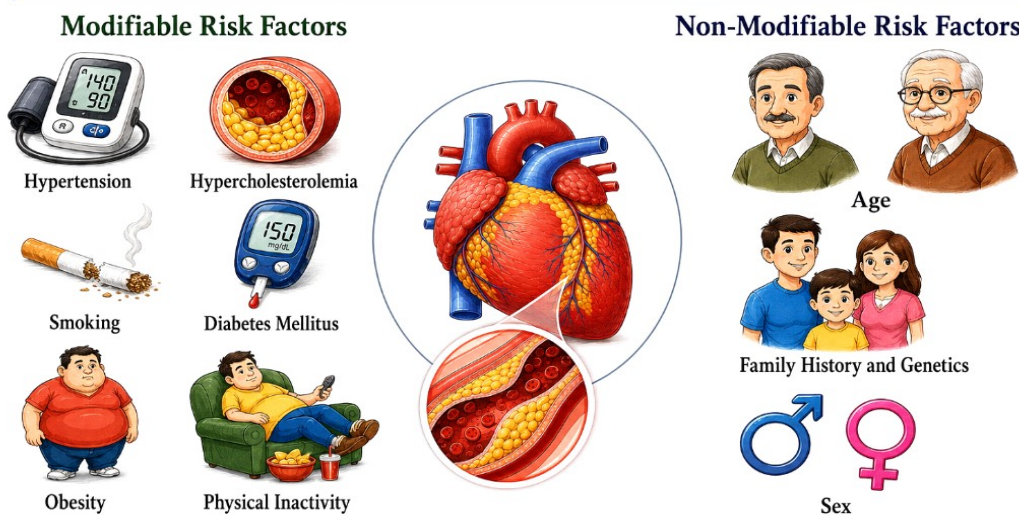


Fig.5.32: ECG changes appearing in STEMI and non-STEMI. Appearance of Q wave is indicative of myocardial injury and is observed in STEMI.

❑ Clinical Outcome

- The prognosis of myocardial infarction depends on the extent of tissue necrosis and the timing of treatment. Some patients may die before reaching the hospital due to **sudden cardiac death, with ventricular fibrillation** being one of the most common causes. Complications may occur immediately after a heart attack or develop gradually over time.

RISK FACTORS OF CORONARY ARTERY DISEASE



□ Pathophysiology of Coronary Artery Disease

1. Endothelial Injury

The pathogenesis of coronary artery disease (CAD) begins with damage to the endothelial lining of the coronary arteries. Endothelial injury is commonly initiated by hypertension, smoking, diabetes, and elevated LDL cholesterol.

2. Lipid Accumulation

LDL cholesterol enters the damaged arterial wall and undergoes oxidation. The oxidized LDL then triggers inflammatory and immune responses within the vessel wall.

3. Inflammatory Response

Monocytes migrate into the arterial wall and transform into macrophages. These macrophages engulf oxidized LDL and become foam cells. The accumulation of foam cells within the arterial wall leads to the formation of fatty streaks in the arteries.

4. Plaque Formation

Smooth muscle cells migrate to the injured area and proliferate, leading to the formation of a fibrous cap over the lipid-rich core and producing an atherosclerotic plaque. As the plaque progressively enlarges, it gradually narrows the arterial lumen.

5. Stenosis And Myocardial Ischemia

Significant narrowing of the coronary arteries reduces blood flow to the myocardium, resulting in a decreased oxygen supply that leads to myocardial ischemia, particularly during exertion. Clinically, myocardial ischemia commonly presents as angina pectoris.

6. Plaque Rupture and Thrombosis

Rupture of the fibrous cap exposes the underlying lipid core to the circulating blood, leading to platelet aggregation and rapid thrombus formation at the site of rupture. If the thrombus completely occludes the artery, it results in myocardial infarction, causing irreversible damage to the myocardial tissue.

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A Text Book of HUMAN ANATOMY, PHYSIOLOGY AND PATHOPHYSIOLOGY - I

As per the New PCI Syllabus NEP 2020 **BP104T**

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