Program	B. Pharmacy
Semester	1st
Course/Subject	Human Anatomy, Physiology & Pathophysiology - 1
Course/ Subject Code	BP104T
Faculty Name	Ms. Ravina Kumari
Mobile No.	+918679692189
Email Id	Ravinarajput17@gmail.com

CO No.	**Upon successful completion of this course, the students will be able to:**
1	Explain the fundamental concepts of Human Anatomy, Physiology and Pathophysiology.
2	Explain the gross morphology, structure and functions of various organs of the human body.
3	Understand the etiology and pathogenesis of diseases/disorders associated with integumentary system, peripheral nervous system and cardiovascular system
4	Understand the basic mechanism behind inflammation
5	Identify and differentiate the various tissues and organs of different systems of human body.

Sr. No.	Learning Outcome	CO
1	Describe the structural anatomy of the heart chambers, valves, circulatory loops, and histological layout of blood vessels.	**BP104.5**
2	Apply electrophysiological and physical principles to map pacemaker action potentials, cardiac cycle stages, and ECG tracings.	**BP104.5**
3	Dissect the homeostatic baroreceptor, chemoreceptor, and endocrine pathways regulating systemic blood pressure and cardiac output.	**BP104.5**
4	Evaluate the molecular defects, vascular cellular changes, and mechanical failures driving Hypertension, Arrhythmias, Heart Failure, and Ischemic Coronary Syndromes.	**BP104.5**

Table of Contents

❖ **Anatomy of the Heart and Haemodynamics**

➢ **Structural Anatomy of the Myocardium and Valvular Apparatus**

The heart is a four-chambered, self-adjusting muscular pump located within the middle mediastinum. Its wall features three distinct structural layers: the outer protective **Epicardium**, the middle contractile **Myocardium** (composed of branching cardiac muscle cells linked by intercalated discs containing gap junctions), and the inner lining **Endocardium**.

The Structural Valvular Apparatus

[Atrioventricular (AV) Valves]	**[Semilunar (SL) Valves]**
(Prevents backflow into Atria)	(Prevents backflow into Ventricles)
➔ **Tricuspid Valve**	➔ **Aortic Valve**
(Right side; 3 cusps)	(Left ventricular exit)
➔ **Bicuspid / Mitral Valve**	➔ **Pulmonary Valve**
(Left side; 2 cusps)	(Right ventricular exit)

- **Chamber Geometry:** The **Atria** feature thin walls as they receive blood under low pressures, while the **Ventricles** possess thick muscular walls designed to push blood into downstream vascular circuits. The left ventricular myocardium is roughly three times thicker than the right, reflecting the high resistance of the systemic circuit compared to the low-resistance pulmonary loop.
- **Valvular Mechanics:** The **Atrioventricular (AV) Valves** are anchored structurally to the ventricular floor via fibrous cords called **Chordae Tendineae**, which project from projecting **Papillary Muscles**. When the ventricles contract, the papillary muscles shorten, pulling the chordae tendineae taut to prevent the valve cusps from everting into the atria. The **Semilunar (SL) Valves** rely on crescent-shaped pockets that catch back-flowing blood, sealing the exits automatically when ventricles relax.

➢ **Systemic, Pulmonary, and Coronary Circulatory Pathways**

Blood travels concurrently through two distinct master loops, driven by the synchronized action of the dual muscular pumps:

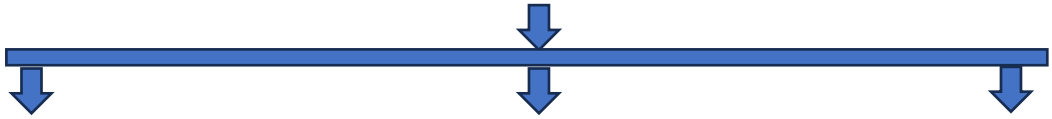
Right Ventricle ➔ Pulmonary SL Valve ➔ Pulmonary Arteries ➔ Alveolar Capillaries (Gas Exchange) ➔ Pulmonary Veins ➔ Left Atrium
Left Ventricle ➔ Aortic SL Valve ➔ Ascending Aorta ➔ Systemic Capillary Beds (Deoxygenation) ➔ Venae Cavae ➔ Right Atrium

- **Coronary Circulation:** The myocardium is highly metabolic and cannot extract oxygen from the blood inside its chambers. It relies on its own plumbing network—the **Coronary Circulation**. The **Right and Left Coronary Arteries** branch directly from the root of the ascending aorta. Blood flows through these arteries primarily during the **diastole phase** of the cardiac cycle, when the contracting myocardium relaxes and uncompresses the intramyocardial vessels. Deoxygenated structural blood collects in the **Coronary Sinus**, which empties directly back into the right atrium.

➢ **Histological Micro-Architecture and Functions of Blood Vessels**

The vascular highway is divided into three functional categories, with walls organized into three distinct structural coats (tunics):

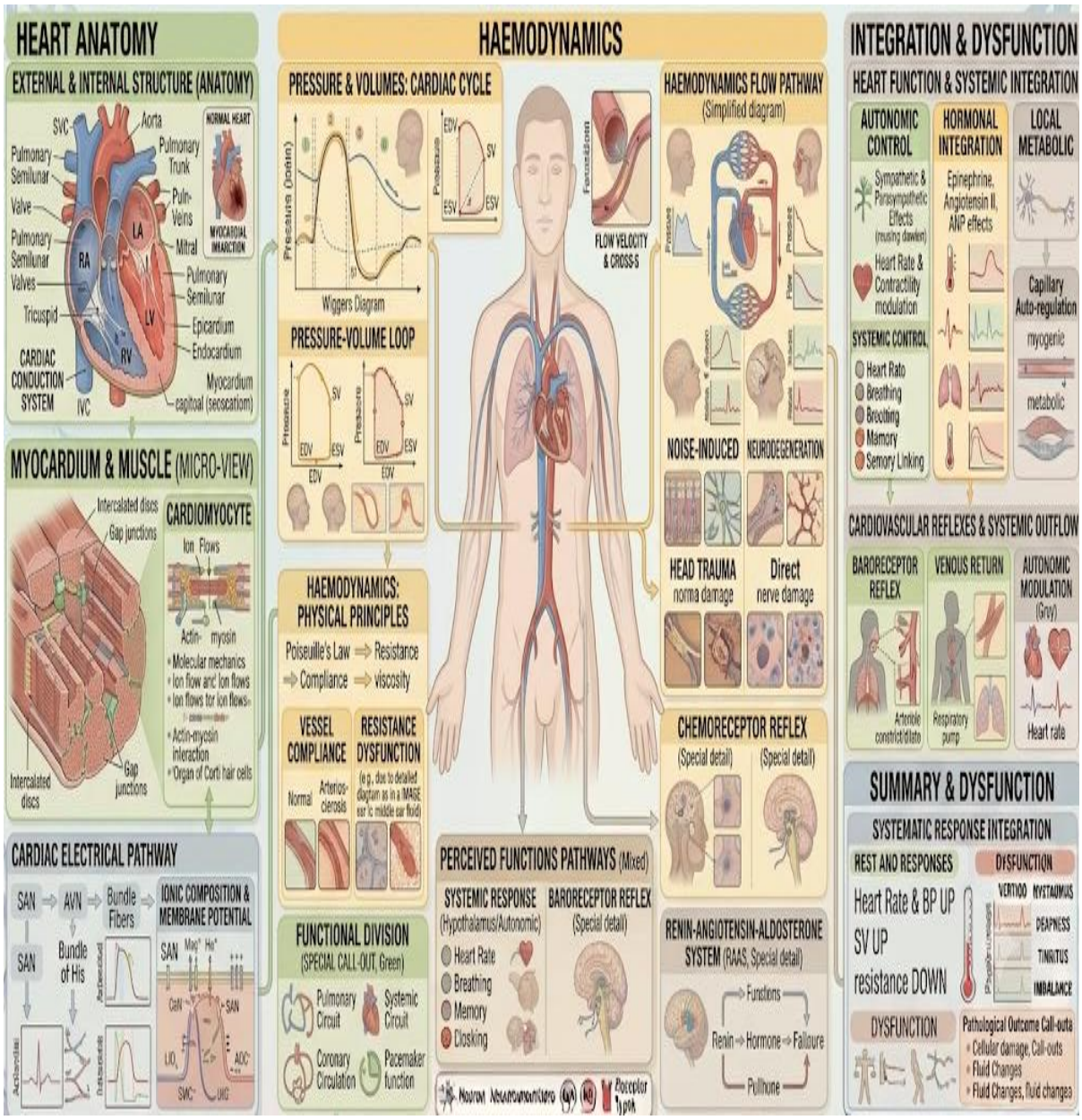
Histological Vascular Blueprint



[Tunica Intima]	**[Tunica Media]**	**[Tunica Adventitia]**
(Inner Endothelial Sheath)	(Middle Smooth Muscle Layer)	(Outer Connective Coat)
➔ Simple squamous cells	➔ Elastic fiber layers	➔ Collagen networks
➔ Minimal friction surface	➔ Regulates Vasoconstriction	➔Structural anchoring

- **Arteries (Pressure Reservoirs):** Possess a thick, highly developed **Tunica Media** rich in smooth muscle and elastic tissue templates. This architecture allows them to stretch to absorb high-pressure systolic surges and recoil elastically to maintain continuous baseline blood pressure during diastole.
- **Veins (Volume Reservoirs):** Characterized by a significantly thinner tunica media layer and a larger internal lumen diameter, allowing them to host up to **64% of total blood volume** at rest. Because they operate under low pressures, veins feature internal **one-way endothelial valves** that direct blood back toward the heart, preventing gravity-driven pooling.
- **Capillaries (Exchange Vessels):** Devoid of both a tunica media and adventitia; their wall consists exclusively of a single layer of endothelial cells resting on a delicate basement membrane (**Tunica Intima**). This ultra-thin layout minimizes diffusion

distances, allowing for rapid exchange of gases, nutrients, and cellular metabolic wastes between blood and interstitial fluid.



❖ **Cardiac Electrophysiology and Kinetics**

➢ **The Intrinsic Conduction System and Pacemaker Mechanics**

The heart possesses an intrinsic auto-rhythmic network capable of generating and propagating electrical action potentials completely independent of nervous input:

Sinoatrial (SA) Node ➔ Internodal Pathways ➔ Atrioventricular (AV) Node (0.1s Delay) ➔ AV Bundle of His ➔ Bundle Branches ➔ Purkinje Fibers

- **Sinoatrial (SA) Node:** Located in the superior wall of the right atrium. Functions as the dominant **Primary Pacemaker**, generating spontaneous action potentials at an intrinsic baseline rate of $\approx 70 - 80$ beats/minute.
- **Atrioventricular (AV) Node:** Located in the interatrial septum. Introduces a vital **0.1-second electrical delay**. This delay ensures that the atria have sufficient time to contract fully and pump their entire volume into the ventricles before ventricular depolarization begins.
- **AV Bundle of His & Purkinje System:** The Bundle of His is the only electrical bridge connecting the atria and ventricles through the fibrous skeleton. It splits into right and left bundle branches that travel down the interventricular septum, terminating in **Purkinje Fibers** that rapidly spread the electrical impulse upward through the ventricular walls.

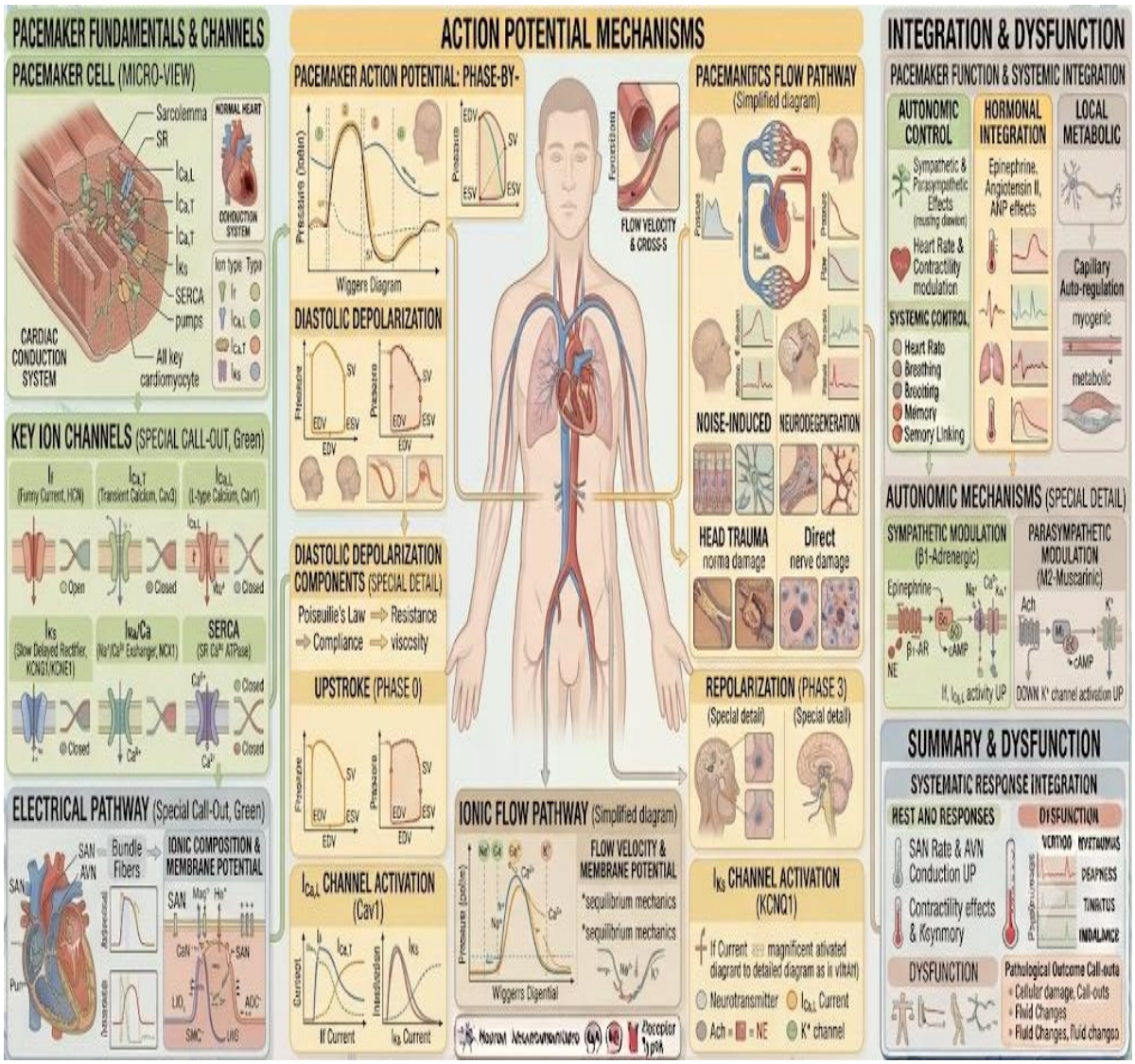
➢ **Cellular Pacemaker Action Potential Graph Channels**

Unlike contractile myocardial cells, auto-rhythmic pacemaker cells do not possess a stable resting membrane potential. Instead, they exhibit a continuous, spontaneous depolarization termed a **Pacemaker Potential**:

✓ **Phase 4 (Spontaneous Depolarization):** Following repolarization, hyperpolarization opens unique **Funny Channels (I_f)**, allowing a slow, steady influx of Sodium (Na^+) ions. As the potential climbs toward threshold, transient **T-type Calcium channels** open, allowing a brief influx of Ca^{2+} to push the membrane to its activation threshold (≈ -40 mV).

✓ **Phase 0 (Depolarization Phase):** Reaching threshold opens long-lasting **L-type Calcium channels**, triggering a rapid influx of extracellular Ca^{2+} ions that drives the membrane potential above zero.

✓ **Phase 3 (Repolarization Phase):** L-type Calcium channels close, and voltage-gated **Potassium (K^+) channels** open, driving a massive efflux of K^+ out of the cell to return the membrane potential to its baseline negative level.



➢ **The Cardiac Cycle: Chronological Mechanical Phases**

The cardiac cycle encompasses all the mechanical and electrical events that occur during a single complete heartbeat. It is divided into alternating periods of contraction (**Systole**) and relaxation (**Diastole**), progressing through five distinct phases:

1. Atrial Systole ➔ 2. Isovolumetric Contraction ➔ 3. Ventricular Ejection ➔ 4. Isovolumetric Relaxation ➔ 5. Ventricular Filling

(P-wave triggers contraction) (All valves closed; Pressure spikes) (SL valves open; Blood exits) (All valves closed; Pressure drops) (AV valves open; Passive flow)

1. **Atrial Systole:** Triggered by the P-wave; the atria contract to pump the final 25% of the ventricular blood volume into the ventricles. The total volume at the end of this phase is the **End-Diastolic Volume (EDV)** (≈ 130 mL).
2. **Isovolumetric Contraction:** Ventricular depolarization begins (QRS complex), causing ventricular pressure to rise. The instant ventricular pressure exceeds atrial pressure, the **AV valves snap shut (producing the First Heart Sound - S_1, "Lubb")**. For a brief window, all four valves are completely closed, and ventricular pressure spikes rapidly while blood volume remains constant.
3. **Ventricular Ejection:** Ventricular pressure rises above the pressure inside the aorta and pulmonary trunk, forcing open the **Semilunar valves**. Blood is forcibly ejected into the great vessels. The volume remaining behind at the end of this phase is the **End-Systolic Volume (ESV)** (≈ 60 mL).
4. **Isovolumetric Relaxation:** Ventricular repolarization occurs (T-wave). As the ventricles relax, pressure within the chambers drops below arterial pressure. Back-flowing blood catches the valve pockets, **snapping the Semilunar valves shut (producing the Second Heart Sound - S_2, "Dupp")**. All four valves are briefly closed again as pressure falls rapidly.
5. **Ventricular Filling:** When ventricular pressure drops below atrial pressure, the **AV valves open**. Blood that had accumulated in the atria rushes passively into the relaxing ventricles, accounting for 75% of ventricular filling before the next cycle begins.

➢ **Cardiac Output Mechanics and Regulation Frameworks**

Cardiac Output (CO) is the total volume of blood ejected by a ventricle into its respective arterial circuit each minute. It is determined by multiplying **Heart Rate (HR)** by **Stroke Volume (SV)**:

$$\text{CO (mL/min)} = \text{HR (beats/min)} \times \text{SV (mL/beat)}$$

Using baseline values (HR = 75 beats/min, SV = 70 mL/beat), normal resting cardiac output is approximately 5.25 L/min, meaning the entire blood volume circulates through the body once every minute. Stroke volume is regulated by three primary variables:

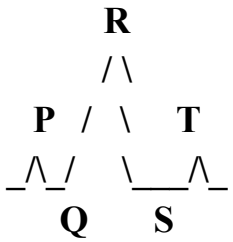
- **Preload (Frank-Starling Law):** The degree of stretch on the heart muscle before it contracts, which depends directly on the volume of blood returning to the heart (**Venous Return**). The *Frank-Starling Law of the Heart* states that

within physiological limits, the greater the volume of blood filling the heart during diastole, the more the myocardial fibers stretch. This alignment optimizes actin-myosin cross-bridge overlapping, generating a more powerful contraction to automatically eject the extra blood volume (Increased EDV → Increased Stroke Volume).

- **Contractility:** The structural force of contraction at any given preload level, driven by changes in intracellular Calcium availability. **Positive Inotropic Agents** (e.g., epinephrine, digitalis) increase extracellular Ca^{2+} influx to boost force, while **Negative Inotropic Agents** (e.g., calcium channel blockers) reduce it.
- **Afterload:** The pressure that must be overcome before the semilunar valves can open and eject blood. An increase in afterload (such as in systemic hypertension or aortic stenosis) shortens the ejection period, leaving more blood behind in the ventricle and **reducing stroke volume**.

➢ **Electrocardiogram (ECG) Waveform Interpretation**

An Electrocardiogram (ECG) is a graphic recording of the electrical currents generated by the depolarization and repolarization waves passing through the myocardium:



- **P-Wave:** A small upward deflection representing **Atrial Depolarization**, which spreads from the SA node through both atria, preceding atrial contraction.
- **QRS Complex:** Begins as a downward deflection (Q), continues as a tall triangular wave (R), and ends as a downward wave (S). It represents **Ventricular Depolarization**, which masks the simultaneous electrical signals of atrial repolarization.
- **T-Wave:** An upward deflection representing **Ventricular Repolarization**, indicating the recovery phase as ventricular muscle cells return to their resting electrical state.

- **PR Interval:** The time required for the depolarization wave to travel from the SA node, through the atria, across the AV node, and down into the ventricular pathways. A prolonged PR interval indicates an electrical conduction delay within the AV node framework.

❖ **Cardiovascular Regulation Systems**

➢ **Autonomic Regulation of the Heartbeat**

While the heart generates its own rhythm, the Cardiovascular Center in the medulla oblongata continuously adjusts heart rate and stroke volume to meet metabolic demands through the autonomic nervous system:

- **Sympathetic Acceleration Pathway:** Travels via Cardioaccelerator Nerves to release Norepinephrine (NE) onto the SA node, AV node, and myocardium. NE binds to β_1-adrenergic receptors, stimulating a cAMP second-messenger system that opens funny channels and calcium channels. This increases the rate of spontaneous pacemaker decay (increasing heart rate) and boosts intracellular Calcium availability to enhance contractility (increasing stroke volume).
- **Parasympathetic Braking Pathway:** Travels via Cranial Nerve X (Vagus Nerve), projecting primarily to the SA and AV nodes. The vagal terminals release Acetylcholine (ACh), which binds to M_2 muscarinic receptors. This downregulates cAMP synthesis and opens acetylcholine-regulated Potassium (K^+) channels, causing K^+ efflux that hyperpolarizes the pacemaker membrane. This slows the rate of spontaneous Phase 4 depolarization, lowering the heart rate (Vagal Tone).

➢ **Regulation of Systemic Blood Pressure and Arterial Pulse Mechanics**

Systemic blood pressure (BP) is the hydrostatic force exerted by blood against vascular walls. It is mathematically determined by the relationship:

Blood Pressure (BP) = Cardiac Output (CO) × Total Peripheral Resistance (TPR)

Total Peripheral Resistance (TPR) is the total resistance to flow throughout the systemic vascular network, heavily influenced by blood viscosity, total blood vessel length, and blood vessel radius. The vessel radius is the primary point of homeostatic control; according to Poiseuille's law, resistance is inversely proportional to the fourth power of the radius (Resistance $\propto 1/r^4$), meaning small adjustments in arteriole constriction cause large shifts in blood pressure.

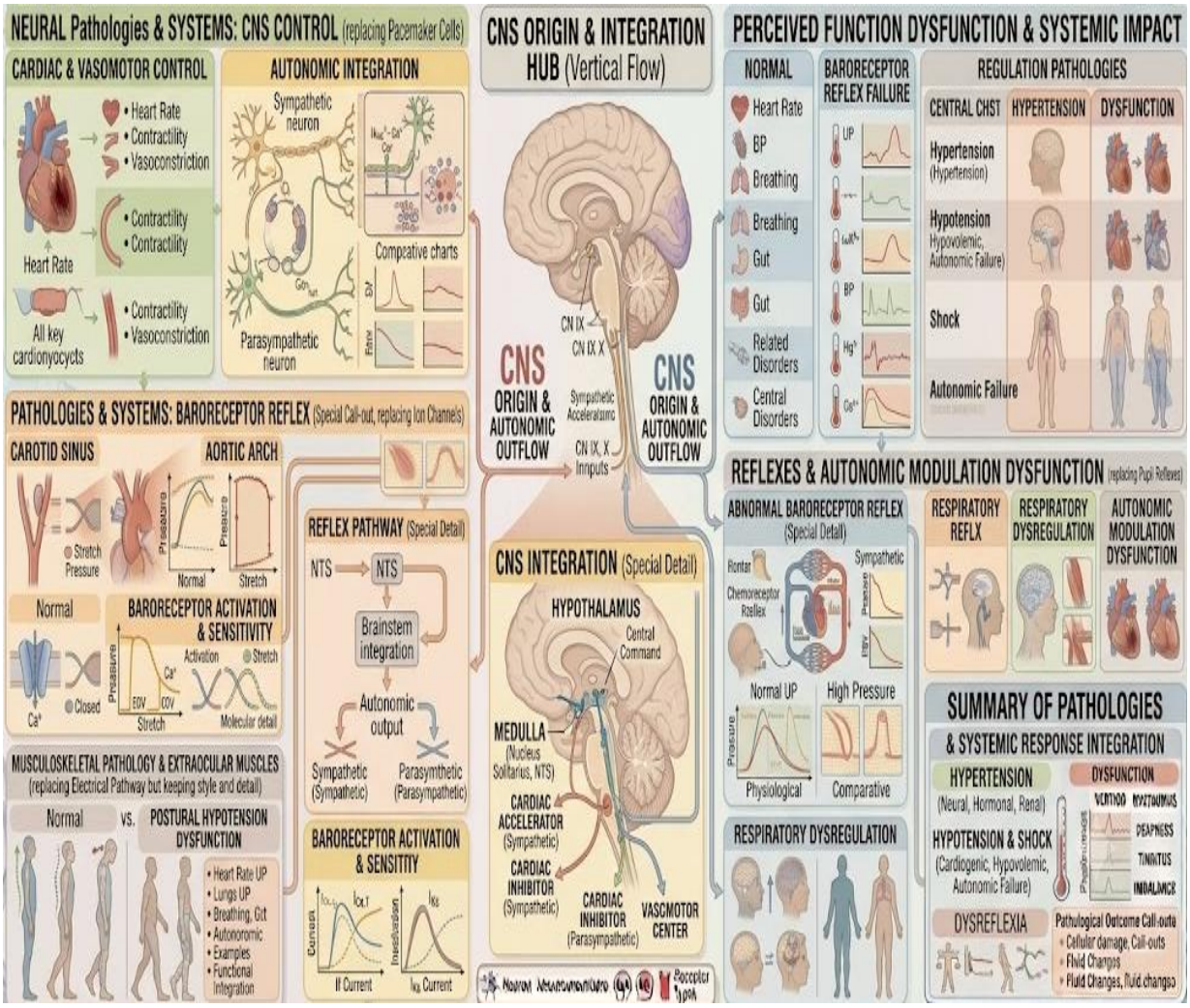
➢ **The Baroreceptor Reflex Arc**

Short-term regulation of blood pressure relies on a rapid neuro-feedback loop centered around specialized stretch receptors called Baroreceptors, located within the Carotid Sinus and the Aortic Arch:

[Systemic Blood Pressure Spikes] ➔ Baroreceptor Stretch Increases ➔ Sensory Firing up CN IX & CN X Increases

[Medulla Cardiovascular Center Integration] ➔ Vagal Parasympathetic Inflow Rises + Sympathetic Output Drops

[Heart Rate Slows + Arterioles Vasodilate] ➔ TPR and CO Fall ➔ Systemic Blood Pressure Restored to Baseline



- **Chemoreceptor Reflex Coordination:** Located in the carotid and aortic bodies, chemoreceptors monitor chemical shifts (O_2 drops, CO_2 climbs, H^+ rises). When they detect severe hypoxia or acidosis, they send signals to the cardiovascular center to stimulate sympathetic vasoconstriction, raising blood pressure to boost blood delivery to the lungs and brain.
- **Hormonal Regulation Mechanisms (RAAS System):** Long-term regulation relies on fluid volume adjustments managed by the endocrine system. When blood pressure or renal perfusion falls, the juxtaglomerular cells of the kidneys secrete the enzyme Renin. Renin cleaves plasma angiotensinogen into Angiotensin I, which is converted into Angiotensin II by Angiotensin-Converting Enzyme (ACE) in the lungs. Angiotensin II is a potent direct vasoconstrictor that raises TPR. It also stimulates the adrenal cortex to release Aldosterone (which increases renal sodium and water reabsorption) and triggers the posterior pituitary to secrete Antidiuretic Hormone (ADH), expanding total blood volume to restore long-term systemic blood pressure.

❖ **Pathophysiology of Cardiovascular Diseases**

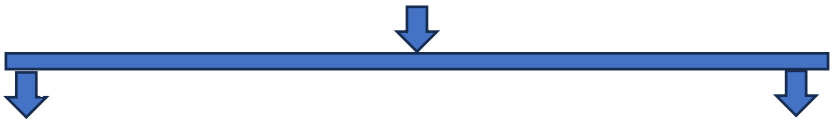
➢ **Hemodynamic Disorders: Hypertension**

- **Pathophysiology:** Hypertension is a chronic hemodynamic state marked by a persistent elevation of systemic arterial blood pressure (Systolic ≥ 130 mmHg or Diastole ≥ 80 mmHg). Over 90% of cases are classified as Primary (Essential) Hypertension, arising from a complex interaction of genetic factors, prolonged sympathetic over-activation, and chronic dysfunction of the RAAS system.
- This persistent pressure overload forces the cardiovascular system out of homeostatic balance. To push blood against elevated systemic resistance, the left ventricle must exert increased force, leading to adaptive Left Ventricular Hypertrophy.
- Over time, this chronic mechanical stress injures the endothelial lining of systemic arteries, accelerating the development of stiffening matrix layers (arteriosclerosis) and narrowing plaques (atherosclerosis), which elevates the risk of stroke, myocardial infarction, and chronic kidney disease.

➢ **Electrophysiological Alterations: Cardiac Arrhythmias**

Arrhythmias encompass any deviation from the normal electrical conduction sequence or rate of the heartbeat. They are caused by abnormalities in impulse generation (pacemaker automaticity errors) or impulse conduction pathways:

[Electrophysiological Arrhythmia Classes]



[Bradyarrhythmias]	**[Tachyarrhythmias]**
(Conduction Delays/Blocks)	(Ectopic Pacemaker Re-entry Circuits)
➔ **1st Degree AV Block** (PR > 0.2s)	➔ **Atrial Fibrillation** (Chaotic electrical waves)
➔ **3rd Degree Block** (Total AV dissociation)	➔ **Ventricular Fibrillation** (Zero cardiac output)

- **Heart Blocks (Conduction Delays):** Arise from ischemia or structural fibrosis within the AV node or Bundle of His. In a First-Degree AV Block, electrical conduction is delayed, causing the PR interval to lengthen beyond 0.20 seconds. In a Third-Degree (Complete) AV Block, electrical transmission between the atria and ventricles is entirely severed. The atria continue to beat

under the SA node's rhythm, while the ventricles adopt a slow, independent escape rhythm driven by downstream pacemaker sites.

- **Fibrillation Pathways:** Characterized by chaotic, rapid, uncoordinated electrical activity. Atrial Fibrillation involves multiple re-entrant wavelets circling the atria, causing them to quiver rather than contract effectively, which increases the risk of blood pooling and thrombus formation. Ventricular Fibrillation is a catastrophic event where chaotic electrical signals cause the ventricles to twitch randomly. This halt coordinated mechanical contraction, reducing cardiac output to zero and causing sudden cardiac death unless immediate electrical defibrillation is performed.

➢ **Functional Kinetics Failure: Congestive Heart Failure (CHF)**

- **Pathophysiology:** Congestive Heart Failure is a progressive systemic disorder where the myocardium cannot pump sufficient blood to satisfy the metabolic and oxygen demands of peripheral tissues. It typically develops as a chronic consequence of long-term ischemic heart disease or chronic untreated hypertension.
- **Left-Sided Heart Failure:** The left ventricle cannot empty effectively, causing blood to back up into the left atrium and the pulmonary veins. This increases hydrostatic pressure within pulmonary capillaries, forcing fluid into alveolar spaces and causing Pulmonary Edema, clinically marked by dyspnea, orthopnea, and coughing up frothy sputum.
- **Right-Sided Heart Failure:** Often caused by preexisting left-sided failure or chronic pulmonary disease. The right ventricle fails to empty effectively, causing blood to back up into the systemic venous circuits. This elevates pressure within the venae cavae, leading to Systemic Venous Congestion, clinically marked by jugular venous distension, hepatomegaly, and dependent peripheral edema pooling in the ankles.
- **Neurohormonal Compensation Failures:** As cardiac output falls, the body tries to compensate by activating the sympathetic nervous system and the RAAS axis. While intended to restore perfusion, the chronic elevation of norepinephrine and angiotensin II increases afterload and fluid retention, worsening the workload on the failing heart and accelerating myocardial remodeling and dilation.

➢ **Ischemic Heart Diseases (IHD)**

1) Atherosclerosis and Coronary Artery Disease (CAD)

- **Pathophysiology:** Atherosclerosis is a chronic inflammatory disease affecting large and medium-sized elastic and muscular arteries, serving as the primary cause of Coronary Artery Disease. The process begins with endothelial injury triggered by chronic hypertension, hyperlipidemia, or smoking.
- This injury allows circulating Low-Density Lipoproteins (LDL) to enter the subendothelial matrix of the tunica intima, where they undergo oxidation. The injured endothelial cells express adhesion molecules that recruit circulating monocytes into the vessel wall. These monocytes differentiate into tissue macrophages, which engulf the oxidized LDL particles via scavenger receptors.

  Endothelial Wall Injury ➔ Intimal LDL Infiltration/Oxidation ➔ Macrophage Scavenger Phagocytosis

  ⇩

  [Foam Cell Accumulation] ➔ Fatty Streak ➔ Smooth Muscle Migration ➔ Fibrous Cap Atheroma Plaque
- As these macrophages accumulate cholesterol, they transform into Foam Cells. The death of foam cells leaves behind a necrotic lipid core, forming a Fatty Streak.
- Over time, local smooth muscle cells migrate from the tunica media into the tunica intima, secreting collagen to build a firm Fibrous Cap over the lipid core, creating a mature Atheromatous Plaque. This plaque physically projects into the vessel lumen, narrowing the artery and restricting downstream blood flow.

2) Angina Pectoris

- **Pathophysiology:** Angina Pectoris is a clinical syndrome characterized by transient paroxysmal chest pain caused by temporary myocardial ischemia, developing when myocardial oxygen demand outpaces available coronary artery supply.
  - ***Stable Angina:*** Occurs when a fixed atheromatous plaque narrows a coronary artery by ≥ 70%. At rest, blood flow is sufficient; however, physical exertion or emotional stress increases heart rate and oxygen demand. The narrowed vessel cannot dilate to accommodate this demand, causing transient ischemia.

This shifts metabolism to anaerobic pathways, generating lactic acid that stimulates local nociceptors, producing transient chest pain that resolves with rest or nitroglycerin.

- ***Unstable Angina:*** Characterized by chest pain that occurs at rest, increases in frequency, or worsens over time. It is driven by plaque disruption, where the fibrous cap cracks or ruptures, exposing the underlying necrotic core to circulating blood and triggering acute, non-occlusive platelet aggregation and thrombus formation.

3) Myocardial Infarction (MI)

- **Pathophysiology:** A Myocardial Infarction occurs when a coronary artery undergoes sudden, prolonged, and complete thrombotic occlusion, leading to irreversible coagulative necrosis of the downstream myocardium. This event is typically triggered by the acute rupture of an unstable atheromatous plaque, which exposes the lipid core to circulating blood. This triggers immediate platelet adhesion, aggregation, and activation of the coagulation cascade, creating an occlusive thrombus that completely blocks downstream blood flow.

Patheroma Plaque Rupture ➔ Acute Thrombotic Occlusion ➔ Total Downstream Coronary Ischemia

⇩

Anaerobic ATP Depletion ➔ Myocyte Membrane Rupture ➔ Cardiac Biomarkers (Troponin) Spilled into Blood

- Within 60 seconds of complete ischemia, myocardial aerobic metabolism ceases, and cells shift to anaerobic glycolysis, dropping ATP levels and impairing contractility. If perfusion is not restored within 20–30 minutes, cells develop irreversible ischemic injury, leading to Coagulative Necrosis.
- The compromised cell membranes rupture, spilling intracellular cardiac enzymes—most notably Cardiac Troponins I and T and Creatine Kinase-MB (CK-MB)—into the bloodstream, which serve as definitive diagnostic biomarkers. Electrocardiographic mapping reveals signature shifts, classifying infarctions into ST-Elevation Myocardial Infarction (STEMI) (indicating transmural, full-thickness necrosis) or Non-ST-Elevation Myocardial Infarction (NSTEMI) (indicating subendocardial, partial-thickness necrosis)

Case-Based Learning (CBL) Assessment Board

📋 CBL Case Study 1: The High-Pressure Ventricular Remodelling Matrix

- **Patient Profile:** A 62-year-old male corporate executive presents for a routine executive health screening. He reports being entirely asymptomatic but notes a strong family history of premature cardiovascular events. On physical examination, his blood pressure is measured at **168/98 mmHg** in the right arm and **166/96 mmHg** in the left arm. A follow-up echocardiogram demonstrates significant **Left Ventricular Hypertrophy (LVH)**, with the interventricular septum thickening to 14 mm (Normal: ≤ 11 mm). His ejection fraction remains preserved at 60%. He is diagnosed with Stage 2 **Primary (Essential) Hypertension**.
- **Core Assessment Task:** Apply the principles of cardiovascular haemodynamics and neurohormonal pathways to trace systemic transformations and ventricular changes.

🧠 Investigative Questions:

1) **Vascular Hemodynamic Analysis:** Using the baseline formula $BP = CO \times TPR$, deduce which specific component is primary in driving this patient's blood pressure to 168/98 mmHg. Integrate Poiseuille's Law (Resistance $\propto 1/r^4$) to explain how microscopic reductions in systemic arteriole radius affect the heart's work.
2) **Cellular Remodeling Deconstruction:** Trace the mechanical cause-and-effect pathway linking a chronic elevation in vascular afterload to the structural development of Left Ventricular Hypertrophy. Explain what happens to myocardial protein synthesis and cross-bridge arrangements as a compensatory response to structural stress.
3) **Hormonal Axis Appraisal:** Critically evaluate how the chronic over-activation of the Renin-Angiotensin-Aldosterone System (RAAS) creates a loop that locks the body into a hypertensive state. Aligned with this pathway, explain the pharmaceutical mechanism of an ACE Inhibitor (e.g., Enalapril) in reducing tissue remodeling.

📋 CBL Case Study 2: The Retrograde Fluid Deluge

- **Patient Profile:** A 68-year-old female with a history of an extensive anterior wall myocardial infarction three years ago presents to the emergency department reporting severe shortness of breath that worsens dramatically

when lying flat (**orthopnea**). She reports needing to sleep propped up on three pillows. Physical examination reveals bilateral inspiratory crackles at the bases of both lungs, an elevated jugular venous distension (JVD) of 6 cm above the sternal angle, and 3 + pitting **peripheral edema** pooling around her ankles and shins. She is diagnosed with acute decompensated **Congestive Heart Failure (CHF)** showing biventricular failure.

- **Core Assessment Task:** Dissect retrograde fluid dynamics, kinetic failures, and compensatory mechanism breakdowns.

🧠 **Investigative Questions:**

1. **Left-Sided Retrograde Mechanics:** Trace the chronological, backward pathway of fluid pressure shifts that begins with a failing left ventricular stroke volume and ends with the development of **Pulmonary Edema**. Explain how elevated pulmonary capillary hydrostatic pressure disrupts normal Starling forces to drive fluid into alveolar spaces.
2. **Right-Sided Systemic Congestion:** Analyze the anatomical path connecting right ventricular kinetic failure to the clinical presentation of jugular venous distension and peripheral ankle edema. Identify the major systemic venous circuits that back up when right atrial pressures rise.
3. **The Compensation Paradox:** Appraise the body's neurohormonal responses (sympathetic activation and aldosterone release) to a dropping cardiac output. Evaluate how these compensatory reflexes act as a double-edged sword that ultimately accelerates myocardial dilation and heart failure progression.

Active Problem-Based Learning (PBL) Interactive Board

🚨 PBL Problem Trigger: The Traumatic Asymmetrical Collapse

1. The Raw Problem Statement

- A 55-year-old overweight male schoolteacher is rushed to the emergency bay by paramedics after experiencing a sudden, tearing, substernal chest pain while shoveling heavy snow in his driveway. He describes the sensation as a heavy elephant sitting directly on his chest, with the pain radiating up into his left jaw and down the inside of his left arm. Unlike his usual episodes of mild chest discomfort during exertion, this pain has lasted for 45 continuous minutes and is not relieved by rest or sublingual nitroglycerin spray.
- On physical evaluation, he is diaphoretic, pale, and tachycardic (HR = 108 beats/min). An immediate 12-lead electrocardiogram (ECG) reveals a prominent, 3-mm **ST-segment elevation** across precordial leads V_1 through V_4. The emergency physician immediately orders a STAT cardiac biomarker panel and prepares the patient for emergency coronary angiography.

2. The Student 3-Column Brainstorming Matrix (Template)

WHAT WE KNOW	WHAT WE NEED TO KNOW	OUR HYPOTHESES
1. 55-year-old male presenting with crushing substernal chest pain. 2. Pain radiates to left jaw/arm; lasts >45 mins; ignores nitrates. 3. ECG shows 3-mm ST-segment elevation in leads V1-V4. 4. Tachycardic and diaphoretic.	• What cellular event transforms a stable plaque into an occlusive thrombus during physical stress? • Why does an ischemic shift to anaerobic pathways cause pain? • What specific proteins will spill into the blood from broken cells? into the blood from broken cells?	1. The patient has sustained an acute ST-Elevation Myocardial Infarction (STEMI). 2. Rupture of a coronary atheroma plaque triggered an occlusive thrombus, completely blocking the Left Anterior Descending artery..

🧠 Core Mechanistic Investigative Questions for Presentation:

1. **The Atherothrombotic Transition:** Analyze the path by which chronic Coronary Artery Disease transitions into an acute Myocardial Infarction. Deconstruct the mechanical and inflammatory forces that drive plaque rupture, and trace the subsequent cascade of platelet adhesion, activation, and fibrin mesh formation that causes complete vessel occlusion.
2. **The Bioenergetic Collapse:** Dissect the immediate cellular and metabolic changes that occur within myocardial tissue when aerobic coronary perfusion

drops to zero. Detail the chronological timeline of ATP depletion, the shift to anaerobic glycolysis, lactic acid accumulation, and the structural failure of membrane ion pumps (Na^+/K^+-ATPase) that leads to irreversible cell necrosis.

3. **The Diagnostic Fingerprint:** Explain the molecular basis behind using Cardiac Troponins I and T as definitive diagnostic blood markers for myocardial injury. Why are these proteins present in the circulation only after an infarction, and how does a transmural (STEMI) injury patterns alter the waves on an ECG tracing?

Multiple Choice Questions

1. Which structural layer of the heart wall contains the branching, contractile cardiac muscle cells linked by intercalated discs?
A) Epicardium B) Endocardium
C) Myocardium D) Pericardium

2. The fibrous cords that physically anchor the atrioventricular (AV) valve cusps to the ventricular papillary muscles are called:
A) Intercalated discs B) Chordae Tendineae
C) Purkinje nets D) Bundle bridges

3. During which specific phase of the cardiac cycle does blood flow through the coronary arteries to nourish the myocardium?
A) Ventricular Systole
B) Ventricular Diastole
C) Atrial Systole
D) Isovolumetric Contraction

4. Which layer of the blood vessel wall contains elastic fibers and smooth muscle layers to regulate vasoconstriction and vasodilation?
A) Tunica Intima B) Tunica Media
C) Tunica Adventitia D) Tunica Externa

5. Which vascular structures act as the primary "volume reservoirs," hosting up to 64% of total blood volume at rest?
A) Elastic Arteries B) Capillaries
C) Veins D) Arterioles

6. The vital 0.1-second electrical conduction delay that allows atria to empty fully into the ventricles occurs within the:
A) Sinoatrial (SA) Node
B) Atrioventricular (AV) Node
C) Bundle of His
D) Purkinje Fibers

7. Spontaneous Phase 4 pacemaker depolarization in auto-rhythmic cells is initiated by the opening of unique channels called:
A) L-type Calcium channels
B) Voltage-gated Potassium channels
C) Funny Channels (I_f)
D) Fast Sodium channels

8. The closure of the Atrioventricular (AV) valves at the start of ventricular contraction produces which heart sound?
A) First Heart Sound (S_1, "Lubb")
B) Second Heart Sound (S_2, "Dupp")
C) Third Heart Sound (S_3)
D) Fourth Heart Sound (S_4)

9. The total volume of blood remaining inside a ventricle at the immediate conclusion of the ventricular ejection phase is termed the:
A) End-Diastolic Volume (EDV)
B) Stroke Volume (SV)
C) End-Systolic Volume (ESV)
D) Cardiac Output (CO)

10. The Frank-Starling Law of the Heart establishes that increasing venous return stretches the myocardium to directly boost:
A) Afterload
B) Heart Rate
C) Total Peripheral Resistance
D) Stroke Volume

11. On a standard Electrocardiogram (ECG) tracing, the QRS complex represents the electrical wave of:
A) Atrial Depolarization
B) Ventricular Depolarization
C) Ventricular Repolarization
D) Atrial Repolarization

12. Vagal parasympathetic stimulation slows the heartbeat by releasing Acetylcholine to bind to which specific receptors?
A) β_1-adrenergic receptors
B) α_1-adrenergic receptors
C) M_2 muscarinic receptors
D) Nicotinic receptors

13. High-pressure stretch receptors that monitor arterial blood pressure are located within the aortic arch and the:
A) Carotid Sinus
B) Jugular Vein
C) Renal Glomerulus
D) Pulmonary Trunk

14. Which potent hormone is released via the renin-angiotensin-aldosterone axis to cause direct vasoconstriction and stimulate aldosterone release?
A) Angiotensin I
B) Angiotensin II
C) Atrial Natriuretic Peptide
D) Bradykinin

15. Left-sided heart failure causes blood to back up into the pulmonary circuits, leading directly to:
A) Jugular Venous Distension
B) Hepatosplenomegaly
C) Pulmonary Edema
D) Peripheral Ankle Edema

16. The microscopic, cholesterol-laden cells that accumulate within the tunica intima during early atherogenesis are:
A) Endothelial cells
B) Foam Cells
C) Smooth muscle fibers
D) Fibroblasts

17. The severe chest pain of Stable Angina pectoris manifests typically when coronary fixed plaque narrowing reaches or exceeds:
A) 20% B) 40%
C) 50% D) 70%

18. Which intracellular protein serves as a highly specific diagnostic blood biomarker for acute myocardial infarction?
A) Creatine Kinase-MM
B) Lactate Dehydrogenase-5
C) Cardiac Troponin
D) Alkaline Phosphatase

19. An electrical conduction delay inside the AV node that extends the PR interval beyond 0.20 seconds is classified as a:
A) First-Degree AV Block
B) Second-Degree AV Block
C) Third-Degree AV Block
D) Ventricular Fibrillation

20. Total Peripheral Resistance (TPR) increases dramatically when the internal radius of systemic arterioles undergoes:
A) Vasodilation
B) Vasoconstriction
C) Endothelial Denudation
D) Elastic Recoil

21. Which chamber of the heart possesses the thickest muscular walls to overcome high systemic resistance?
A) Right Atrium
B) Left Atrium
C) Right Ventricle
D) Left Ventricle

22. What prevents the atrioventricular valve cusps from everting (swinging upward) into the atria during ventricular contraction?
A) High blood velocity in the aorta
B) Contraction of papillary muscles pulling the chordae tendineae taut
C) The passive opening of the semilunar valves
D) The thick fibrous skeleton layout of the interatrial septum

23. Deoxygenated blood returning from the coronary circulation drains directly into the right atrium via the:
A) Superior Vena Cava
B) Inferior Vena Cava
C) Coronary Sinus
D) Pulmonary Vein

24. Which vascular structure consists exclusively of a single layer of endothelial cells resting on a delicate basement membrane?
A) Arterioles B) Capillaries
C) Venules D) Muscular Arteries

25. The mathematical relationship governing systemic blood pressure is expressed as:
A) $BP = HR \times SV$
B) $BP = CO \times TPR$
C) $BP = EDV - ESV$
D) $BP = CO/TPR$

26. Which structural component of the intrinsic conduction system functions as the primary pacemaker of the heart?
A) Atrioventricular (AV) Node
B) Sinoatrial (SA) Node
C) Bundle of His
D) Purkinje Fibers

27. Reaching the threshold potential (≈ -40 mV) in a cardiac pacemaker cell prompts the rapid opening of:
A) Fast Sodium channels
B) L-type Calcium channels
C) T-type Calcium channels
D) Voltage-gated Potassium channels

28. Phase 3 repolarization in both pacemaker and contractile myocardial cells is driven primarily by the:
A) Influx of Sodium ions
B) Influx of Calcium ions
C) Efflux of Potassium ions
D) Efflux of Chloride ions

29. The first heart sound (S_1) occurs chronologically at the immediate start of which phase?
A) Atrial Systole
B) Isovolumetric Contraction
C) Ventricular Ejection
D) Isovolumetric Relaxation

30. The volume of blood pumped out by a single ventricle during one individual contraction is the:
A) Cardiac Output
B) End-Diastolic Volume
C) End-Systolic Volume
D) Stroke Volume

31. On a standard ECG, the T-wave represents the electrical event of:
A) Atrial Depolarization
B) Atrial Repolarization
C) Ventricular Depolarization
D) Ventricular Repolarization

32. Sympathetic stimulation increases heart rate and contractility by releasing Norepinephrine to bind to:
A) α_1-adrenergic receptors
B) β_1-adrenergic receptors
C) β_2-adrenergic receptors
D) M_2 muscarinic receptors

33. According to Poiseuille's Law, if the internal radius (r) of a blood vessel is reduced by half, the resistance to flow:
A) Doubles
B) Increases by 4 times
C) Increases by 8 times
D) Increases by 16 times

34. The primary neurological control center that coordinates the baroreceptor reflex arc is localized within the:
A) Hypothalamus B) Cerebellum
C) Medulla Oblongata D) Midbrain

35. Long-term systemic blood pressure regulation balances blood volume via which primary endocrine cascade?
A) Sympathetic cardioaccelerator axis
B) Renin-Angiotensin-Aldosterone System (RAAS)
C) Kinin-bradykinin local loop
D) Histamine-mast cell degranulation path

36. Essential (primary) hypertension is clinically defined by a persistent elevation of systemic arterial blood pressure at or above:
A) 120/80 mmHg B) 130/80 mmHg
C) 140/90 mmHg D) 150/95 mmHg

37. Which cardiac condition features multiple chaotic electrical wavelets circling the atria, causing them to quiver rather than contract?
A) First-Degree AV Block
B) Atrial Fibrillation
C) Ventricular Fibrillation
D) Third-Degree AV Block

38. Right-sided heart failure leads to backup congestion within the systemic venous circuits, clinically manifesting as:
A) Pulmonary Edema
B) Symmetrical peripheral ankle edema and JVD
C) Dyspnea and orthopnea
D) Productive cough with frothy sputum

39. What is the immediate clinical result when ventricular fibrillation occurs?
A) Cardiac output drops to zero, causing sudden cardiac death if untreated
B) An increase in left ventricular stroke volume due to preload stretching
C) The PR interval extends beyond 0.20 seconds
D) Hypertrophy of the interventricular septum to 14 mm

40. The early structural lesion of atherosclerosis, characterized by a visible accumulation of foam cells beneath the endothelium, is a:
A) Fibrous Cap B) Fatty Streak
C) Thrombus D) Microvascular embolus

41. Angina pectoris that occurs at rest, increases in frequency, or worsens over time is classified as:
A) Stable Angina B) Unstable Angina
C) Prinzmetal Angina
D) Microvascular Angina

42. Irreversible coagulative necrosis of the myocardium due to sudden, prolonged, and complete thrombotic occlusion of a coronary artery is a:
A) Stable Angina episode
B) Transient ischemic attack
C) Myocardial Infarction
D) Left ventricular remodeling phase

43. A transmural myocardial infarction that involves full-thickness necrosis of the ventricular wall is typically classified on an ECG as a:
A) First-Degree Block B) NSTEMI
C) STEMI D) Atrial Flutter

44. The enzyme that proteolytically cleaves plasma Angiotensinogen into Angiotensin I is:
A) Angiotensin-Converting Enzyme (ACE)
B) Renin
C) Aldosterone
D) Thrombin

45. The second heart sound (S_2, "Dupp") is caused by the snapping shut of which valves?
A) Tricuspid and Mitral valves
B) Aortic and Pulmonary semilunar valves
C) Foramen ovale valves
D) The Eustachian venous valves

46. Cardiac output is mathematically calculated as the product of:
A) EDV − ESV
B) BP × TPR
C) HR × SV
D) SV/TPR

47. An accumulation of purulent exudate rising behind a blocked pediatric Eustachian tube is the diagnostic profile of:
A) Otitis Externa
B) Acute Otitis Media
C) Benign Paroxysmal Positional Vertigo
D) Anosmia

48. Dislodged calcium carbonate crystals (otoconia) migrating into the semicircular canals cause which condition?
A) Glaucoma
B) Cataract
C) Myopia
D) Vertigo

49. The progressive loss of lens matrix transparency due to oxidative clumping of crystallin proteins is a:
A) Cataract
B) Glaucoma
C) Myopia
D) Astigmatism

50. Postmenopausal estrogen withdrawal triggers checking of osteoclast life cycles, serving as the driver for:
A) Gouty Arthritis
B) Osteoporosis
C) Myasthenia Gravis
D) Rheumatoid Arthritis

ANSWER KEY (MCQs) 1. C | 2. B | 3. B | 4. B | 5. C | 6. B | 7. C | 8. A | 9. C | 10. D | 11. B | 12. C | 13. A | 14. B | 15. C | 16. B | 17. D | 18. C | 19. A | 20. B | 21. D | 22. B | 23. C | 24. B | 25. B | 26. B | 27. B | 28. C | 29. B | 30. D | 31. D | 32. B | 33. D | 34. C | 35. B | 36. B | 37. B | 38. B | 39. A | 40. B | 41. B | 42. C | 43. C | 44. B | 45. B | 46. C | 47. B | 48. D | 49. A | 50. B.

Very Short Answer (2- Marks)

1) State the structural layer of the human heart wall that contains the contractile muscle tissue. (Bloom's Level 1: Remember)

A. Myocardium.

2) Name the specific heart valve located between the left ventricle and the ascending aorta exit point. (Bloom's Level 1: Remember)

A. Aortic Semilunar.

3) Identify the blood vessel class that completely lacks both a tunica media and a tunica adventitia layer. (Bloom's Level 1: Remember)

A. Capillaries.

4) Name the electrical component designated as the primary pacemaker of the human heart. (Bloom's Level 1: Remember)

A. Sinoatrial Node.

5) Which specific ion channels open to drive the rapid Phase 0 depolarization of a cardiac pacemaker cell? (Bloom's Level 1: Remember)

A. L-type Calcium.

6) Give the clinical term for the volume of blood trapped inside a ventricle at the immediate completion of filling. (Bloom's Level 1: Remember)

A. End-Diastolic Volume.

7) Identify the heart sound produced by the snapping shut of the two semilunar valves. (Bloom's Level 1: Remember)

A. Second Heart Sound.

8) State the mathematical formula used to determine systemic Cardiac Output. (Bloom's Level 1: Remember)

A. HR × SV.

9) Which wave on a standard electrocardiogram tracing represents the spread of atrial depolarization? (Bloom's Level 1: Remember)

A. P-wave.

10) Name the autonomic nerve pathway that acts as a continuous brake to slow the resting heart rate. (Bloom's Level 1: Remember)

A. Vagus Nerve.

11) Identify the primary enzyme secreted by juxtaglomerular cells in response to low renal perfusion. (Bloom's Level 1: Remember)

A. Renin.

12) State the structural classification of an arrhythmia where the PR interval exceeds 0.20 seconds. (Bloom's Level 2: Understand)

A. First-Degree Block.

13) Give the clinical term for the localized fluid backup into alveolar spaces caused by left-sided heart failure. (Bloom's Level 2: Understand)

A. Pulmonary Edema.

14) Identify the structural cell type created when a tissue macrophage phagocytoses oxidized LDL particles. (Bloom's Level 2: Understand)

A. Foam Cell.

15) Name the structural cap layer that isolates an atheromatous plaque's necrotic core from circulating blood. (Bloom's Level 1: Remember)

A. Fibrous cap.

16) State the vascular structures that act as the primary volume reservoirs, hosting up to 64% of blood volume at rest. (Bloom's Level 1: Remember)

A. Veins.

17) Enumerate the vital electrical conduction delay that allows atria to empty fully into the ventricles. (Bloom's Level 1: Remember)

A. 0.1-second delay.

18) Name the structural parameters that are used to multiply together to determine systemic blood pressure. (Bloom's Level 2: Understand)

A. CO × TPR.

19) Identify the highly specific intracellular protein that serves as a diagnostic blood biomarker for acute myocardial infarction. (Bloom's Level 1: Remember)

A. Cardiac Troponin.

20) Give the term for the uncoordinated atrial electrical waves that quiver rather than contract effectively. (Bloom's Level 2: Understand)

A. Atrial Fibrillation.

Short Answer Questions (5- Marks)

1. Differentiate between the structural wall thicknesses and hemodynamic roles of systemic arteries, veins, and capillaries.
   (Bloom's Level 4: Analyze)
2. Describe the coronary circulatory pathway, detailing its origin, phase-dependent flow, and final drainage into the right atrium.
   (Bloom's Level 2: Understand)
3. Explain the sequence of electrical structures that propagate an impulse through the heart's intrinsic conduction system.
   (Bloom's Level 2: Understand)
4. Dissect the ion channel currents that drive the spontaneous pacemaker potential and action potential in an auto-rhythmic cell.
   (Bloom's Level 4: Analyze)
5. Outline the mechanical events and valvular positions that characterize the phase of Isovolumetric Contraction.
   (Bloom's Level 2: Understand)
6. Explain how Stroke Volume is regulated by the physiological variables of Preload, Contractility, and Afterload.
   (Bloom's Level 2: Understand)
7. Map out the standard waves and intervals of an ECG tracing, correlating each with its underlying myocardial electrical event.
   (Bloom's Level 3: Apply)
8. Dissect the autonomic mechanisms by which sympathetic cardioaccelerator nerves elevate heart rate and contractility force via β_1 receptors.
   (Bloom's Level 4: Analyze)
9. Apply the Baroreceptor Reflex Arc framework to explain how the body responds to a sudden drop in systemic blood pressure.
   (Bloom's Level 3: Apply)
10. Outline the components of the Renin-Angiotensin-Aldosterone System (RAAS) and explain how it regulates long-term blood pressure volume.
    (Bloom's Level 2: Understand)
11. Explain the structural tissue transformations and ventricular overloading that lead to Left Ventricular Hypertrophy in primary hypertension.
    (Bloom's Level 4: Analyze)

12. Differentiate between the electrophysiological failures and clinical consequences of Atrial Fibrillation versus Ventricular Fibrillation.
(Bloom's Level 4: Analyze)
13. Dissect the cell-signaling errors and retrograde fluid dynamics that produce systemic venous congestion in right-sided heart failure.
(Bloom's Level 4: Analyze)
14. Chronologically outline the stages of Atherogenesis within the tunica intima, from initial endothelial injury to mature atheroma formation.
(Bloom's Level 2: Understand)
15. Explain the plaque dynamics and ischemic parameters that distinguish Stable Angina from an acute Myocardial Infarction.
(Bloom's Level 4: Analyze)

Long Answer Questions (10- Marks)

1. Write a detailed compendium on the structural anatomy, chambers, and internal valvular apparatus of the human heart. Discuss the systemic and pulmonary circulatory routes, supported by flowcharts. (Bloom's Level 2: Understand)
2. Write a comprehensive essay detailing the chronological mechanical and electrical events of the Cardiac Cycle. Compare pressures, volumes, and valvular positions across all five distinct phases, supported by reference diagrams. (Bloom's Level 4: Analyze)
3. Apply your knowledge of cardiovascular haemodynamics to analyze the homeostatic regulation of systemic Blood Pressure. Detail the baroreceptor reflex arc, chemoreceptor inputs, and Poiseuille's laws of peripheral resistance. (Bloom's Level 3: Apply)
4. Discuss the entire cellular and inflammatory pathogenesis of Atherosclerosis and Coronary Artery Disease. Trace the roles of endothelial damage, oxidized LDL accumulation, foam cell formation, and fibrous cap rupture mechanisms. (Bloom's Level 5: Evaluate)
5. Chronologically trace the pathophysiological cascade of an acute ST-Elevation Myocardial Infarction (STEMI). Analyze the transition from aerobic to anaerobic metabolism, cell membrane rupture, and the clinical release of cardiac biomarkers. (Bloom's Level 5: Evaluate)
6. Write a comprehensive essay on the complex etiology and systemic pathophysiology of Congestive Heart Failure. Compare the mechanical breakdowns, backwards congestion loops, and forward clinical presentations of left-sided versus right-sided failure. (Bloom's Level 5: Evaluate)
7. Dissect the intrinsic electrophysiology of the heart. Evaluate the auto-rhythmic mechanism of the SA node, explain the 0.1-second delay at the AV node, and outline the classification of first-, second-, and third-degree heart blocks. (Bloom's Level 5: Evaluate)
8. Evaluate the complete neurohormonal and endocrine loops that manage long-term blood pressure homeostasis, detailing the integrated actions of Renin, Angiotensin II, Aldosterone, ADH, and Atrial Natriuretic Peptide. (Bloom's Level 5: Evaluate)
9. Discuss the micro-anatomy and functional histology of the human vascular highway, comparing the structural adaptations of pressure-resisting arteries, volume-storing veins, and micro-exchange capillary beds. (Bloom's Level 4: Analyze)

10. Evaluate the physiological consequences of chemical interventions on the cardiovascular system, mapping out how beta-blockers, calcium channel blockers, and ACE inhibitors alter haemodynamics to manage chronic hypertension. (Bloom's Level 5: Evaluate)
11. Provide a detailed account of the mechanical and pressure changes that occur during the phase of Ventricular Ejection, correlating valvular states with aortic pressure changes. (Bloom's Level 4: Analyze)
12. Compare and contrast the complete tissue-level modifications, necrotic transformations, and clinical ECG findings that distinguish a STEMI from an NSTEMI presentation. (Bloom's Level 5: Evaluate)
13. Evaluate the cellular mechanisms that maintain the prolonged plateau phase (Phase 2) of a contractile myocardial action potential, contrasting it with skeletal muscle kinetics. (Bloom's Level 5: Evaluate)
14. Write a comprehensive essay on the physiological mechanisms that govern human Endocrine Regulation of Blood Sugar Homeostasis. Analyze the opposing feedback arms, glucose channel actions, and metabolic shifts run by Insulin and Glucagon. (Bloom's Level 5: Evaluate)
15. Discuss how target toxins act on the Neuromuscular Junction to cause synaptic failure. Compare the molecular mechanisms, cellular changes, and final clinical presentations of Organophosphates, Botulinum toxin, and Curare. (Bloom's Level 3: Apply)