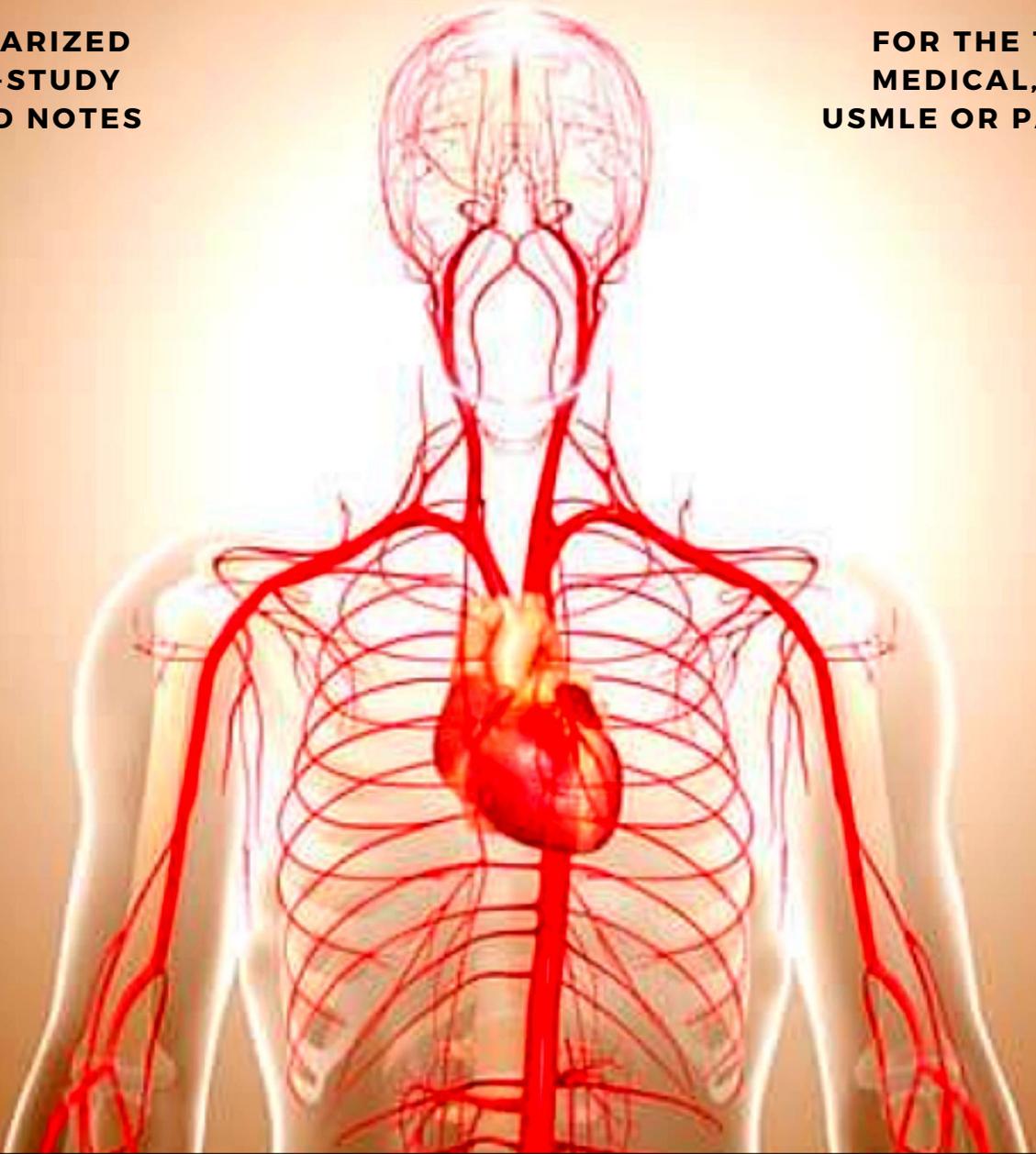


ANATOMY, PHYSIOLOGY & PATHOLOGY NOTES OF THE **CARDIOVASCULAR** SYSTEM

FOURTH EDITION

PRE-SUMMARIZED
READY-TO-STUDY
HIGH-YIELD NOTES

FOR THE TIME-POOR
MEDICAL, PRE-MED,
USMLE OR PA STUDENT



PDF



201 PAGES

A Message From Our Team

Studying medicine or any health-related degree can be stressful; believe us, we know from experience! The human body is an incredibly complex organism, and finding a way to streamline your learning is crucial to succeeding in your exams and future profession. Our goal from the outset has been to create the greatest educational resource for the next generation of medical students, and to make them as affordable as possible.

In this fourth edition of our notes we have made a number of text corrections, formatting updates, and figure updates which we feel will enhance your study experience. We have also endeavoured to use only open-source images and/or provide attribution where possible.

If you are new to us, here are a few things to help get the most out of your notes:

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Table Of Contents:

What's included: Ready-to-study anatomy, physiology and pathology notes of the cardiovascular system presented in succinct, intuitive and richly illustrated downloadable PDF documents. Once downloaded, you may choose to either print and bind them, or make annotations digitally on your iPad or tablet PC.

Anatomy & Physiology Notes:

- ANATOMY OF THE HEART
- ELECTROPHYSIOLOGY OF THE HEART
- ELECTROCARDIOGRAM (ECG) PHYSIOLOGY
- MECHANICAL EVENTS OF THE CARDIAC CYCLE
- CARDIO-DYNAMICS
- HAEMODYNAMICS / HEMODYNAMICS
- BLOOD PRESSURE PHYSIOLOGY
- ANATOMY & PHYSIOLOGY OF BLOOD VESSELS
- PHYSIOLOGY OF HYPERTENSION
- PHYSIOLOGY OF SHOCK
- PHYSIOLOGY OF MYOCARDIAL ISCHAEMIA / ISCHEMIA
- THE EFFECTS OF AGEING ON THE HEART

Pathology Notes:

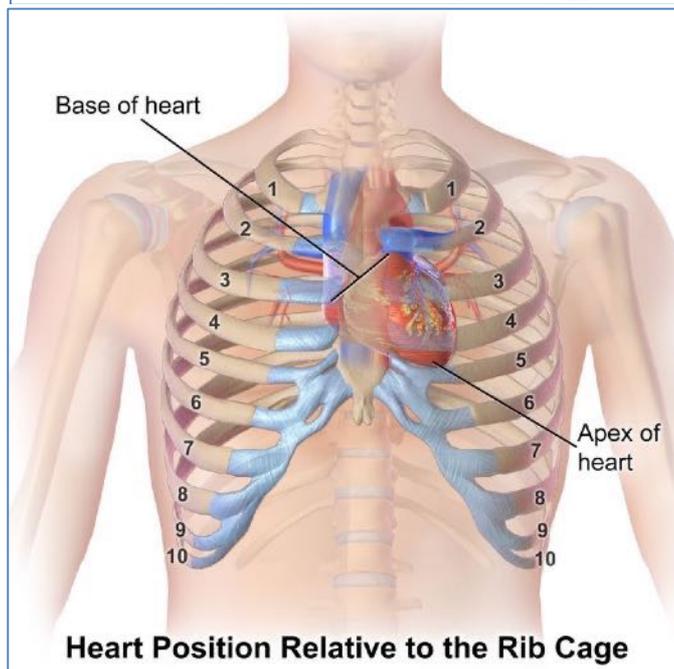
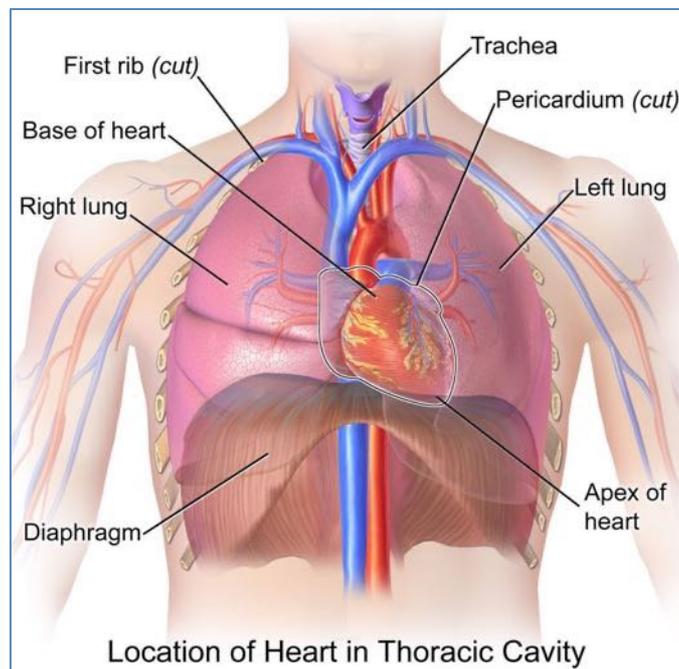
- CONGENITAL HEART DEFECTS
- ANEURYSMS & DISSECTIONS
- ARRHYTHMIAS
- DRUG CLASSES FOR TREATING ARRHYTHMIAS
- DYSLIPIDAEMIA
- ATHEROSCLEROSIS
- ISCHAEMIC HEART DISEASE
- ACUTE CARDIOGENIC PULMONARY OEDEMA
- HEART FAILURE
- CARDIOMYOPATHIES ("HEART MUSCLE DISEASES")
- PATHOLOGY OF HYPERTENSION
- PATHOLOGY OF SHOCK
- DVT & PE
- CARCINOID HEART DISEASE
- INFECTIVE ENDOCARDITIS
- NON-INFECTIVE ENDOCARDITIS (NBTE - Non Bacterial Thrombotic Endocarditis)
- LYMPHANGITIS
- MYOCARDITIS – VIRAL & TOXIC
- PERICARDITIS
- PERICARDIAL EFFUSION
- CARDIAC TAMPONADE
- ACUTE ARTERIAL OCCLUSION ("CRITICAL LIMB ISCHAEMIA")
- PERIPHERAL VASCULAR DISEASE
- VARICOSE VEINS
- CHRONIC SKIN ULCERS
- TUMOURS OF VESSELS
- IMPORTANT VASCULITIDES
- RHEUMATIC FEVER & RHEUMATIC HEART DISEASE
- VALVULAR HEART DISEASE & MURMURS
- CARDIOVASCULAR DISEASE & OBESITY; NUTRITION & PHYSICAL EXERCISE

ANATOMY OF THE HEART

HEART ANATOMY:

Anatomical Location of the Heart:

- Snugly enclosed within the ***middle mediastinum*** (medial cavity of thorax). Contains:
 - Heart
 - Pericardium
 - Great Vessels
 - Trachea
 - Esophagus
- Middle Mediastinum – located in the inferior mediastinum (lower than the sternal angle)
- Extends obliquely from 2nd rib → 5th intercostal space.
- Anterior to Vertebrae
- Posterior to Sternum
- Flanked by 2 lungs
- Rests on the diaphragm
- 2/3 of its mass lies to the LHS of the *midsternal line*.

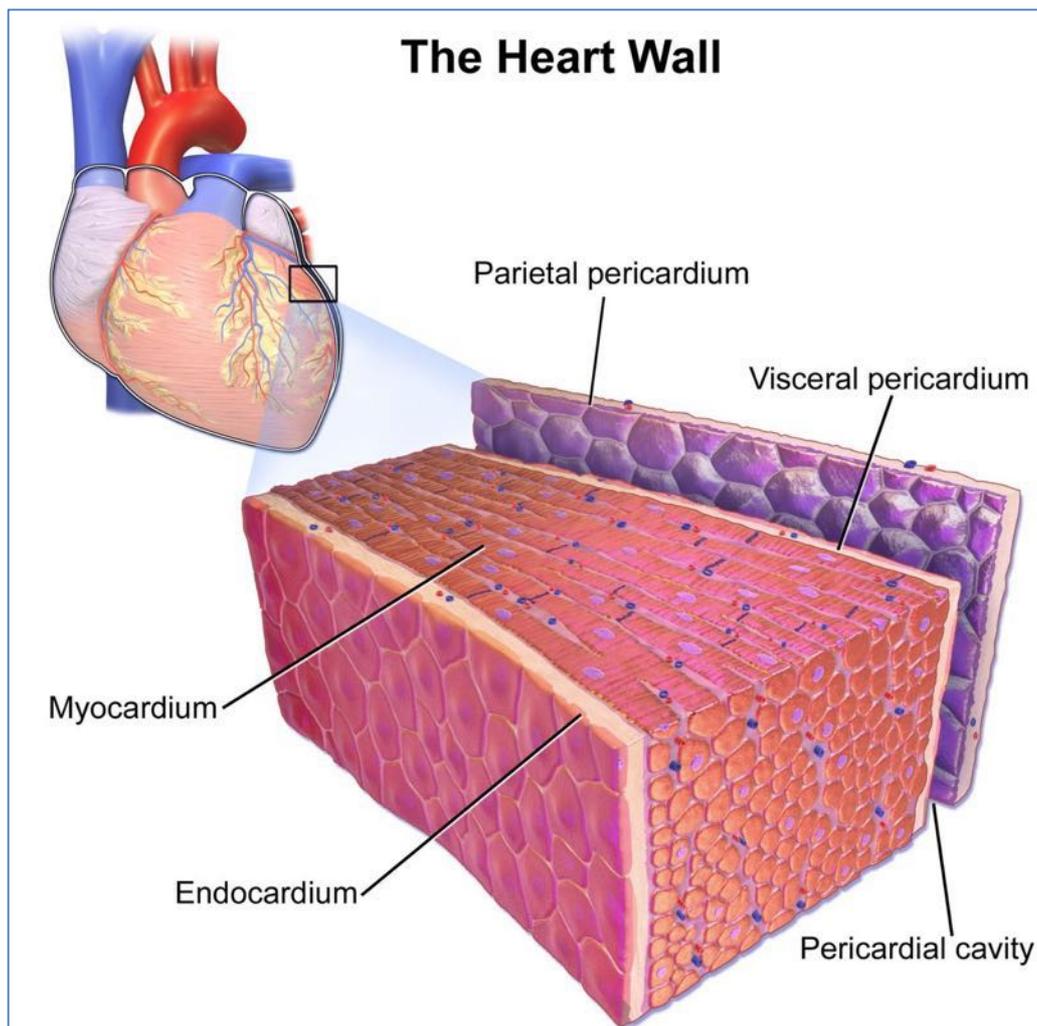


The Pericardium: (Coverings of the Heart)

- A double-walled sac
- contains a film of lubricating serous fluid
- **2 Layers of Pericardium:**
 - **Fibrous Pericardium:**
 - Tough, dense connective tissue
 - Protects the heart
 - Anchors it to surrounding structures
 - Prevents overfilling of the heart – if fluid builds up in the pericardial cavity, it can inhibit effective pumping. (Cardiac Tamponade)
 - **Serous Pericardium:** (one continuous sheet with '2 layers')
 - Parietal Layer – Lines the internal surface of the fibrous pericardium
 - Visceral Layer – (aka **Epicardium**) Lines the external heart surface

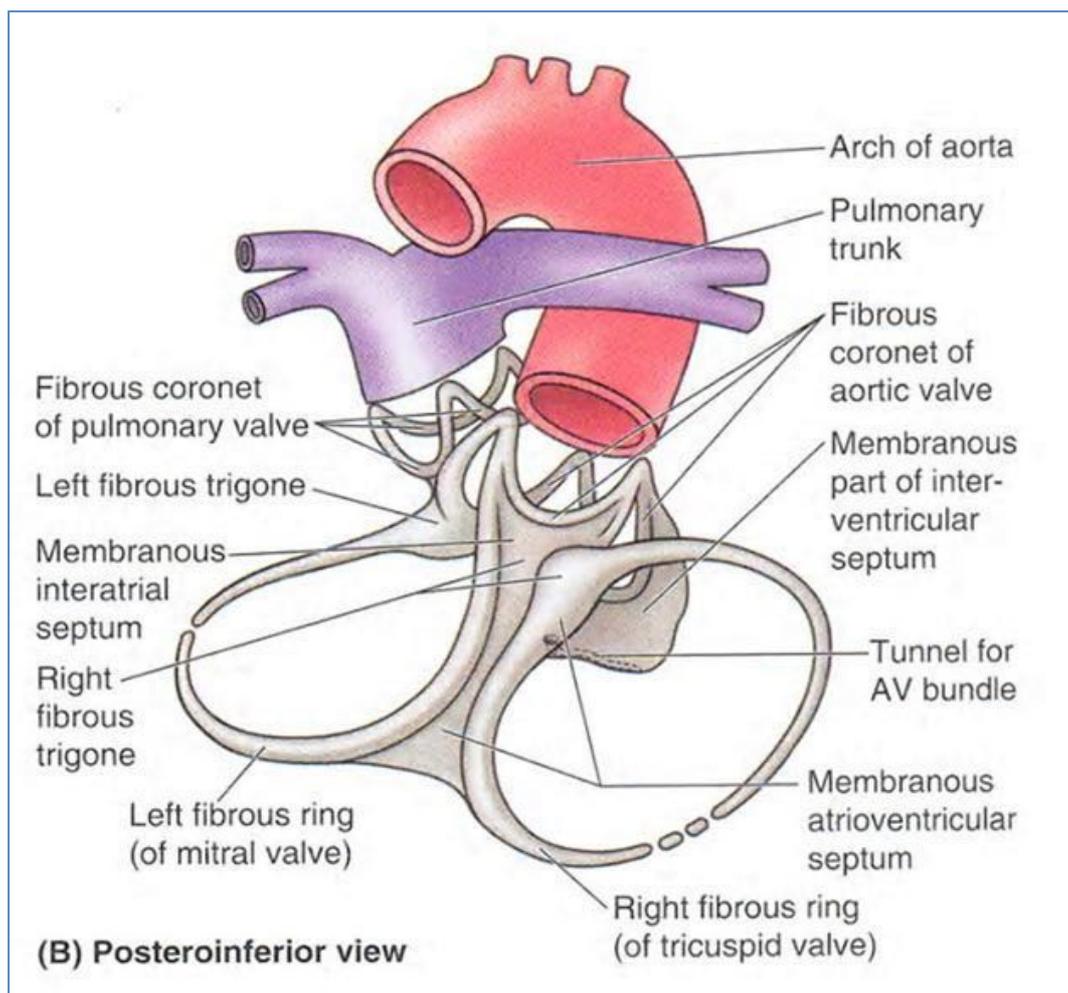
Layers of the Heart Wall:

- **Epicardium:**
 - Visceral layer of serous pericardium
- **Myocardium:**
 - Muscle of the heart
 - The layer that 'contracts'
- **Endocardium:**
 - Lines the chambers of the heart (Endothelial Cells)
 - Prevents clotting of blood within the heart
 - Forms a barrier between the O² hungry myocardium and the blood. (blood is supplied via the coronary system)



Fibrous Skeleton of the Heart:

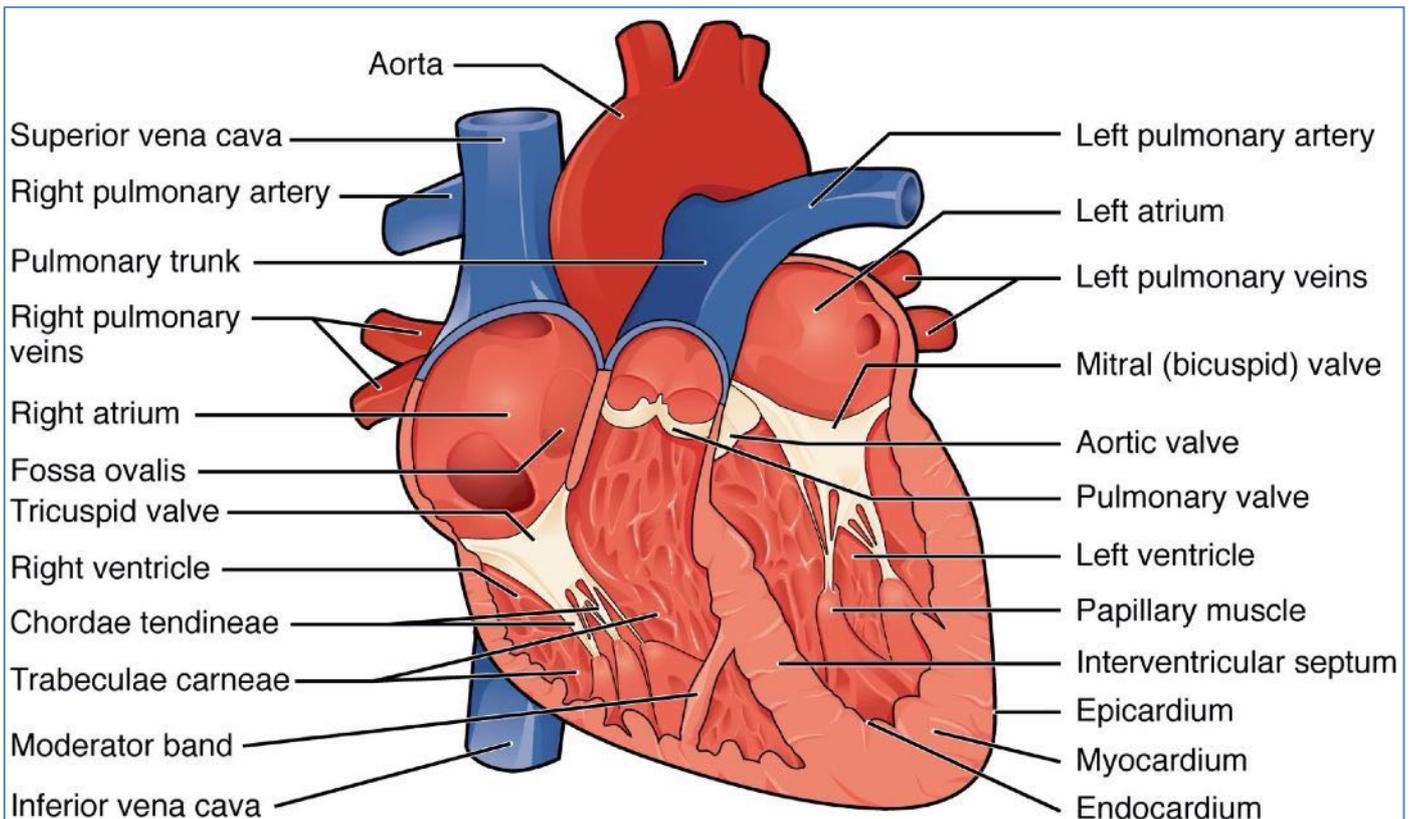
- The network of connective tissue fibers (collagen & elastin) within the myocardium
- Anchors the cardiac muscle fibers + valves + great vessels.
- Reinforces the myocardium
- Provides Electrical Isolation
- **2 Parts:**
 - **Septums:**
 - Flat sheets separating atriums, ventricles & left and right sides of the heart.
 - Electrically isolates the left & right sides of the heart (conn. Tissue = non-conductive)
 - Important for cardiac cycle
 - (interatrial septum/atrioventricular septum/interventricular septum)
 - **Rings:**
 - Rings around great vessel entrances & valves
 - stop stretching under pressure



Source: unable to attribute.

Chambers & Associated Great Vessels:

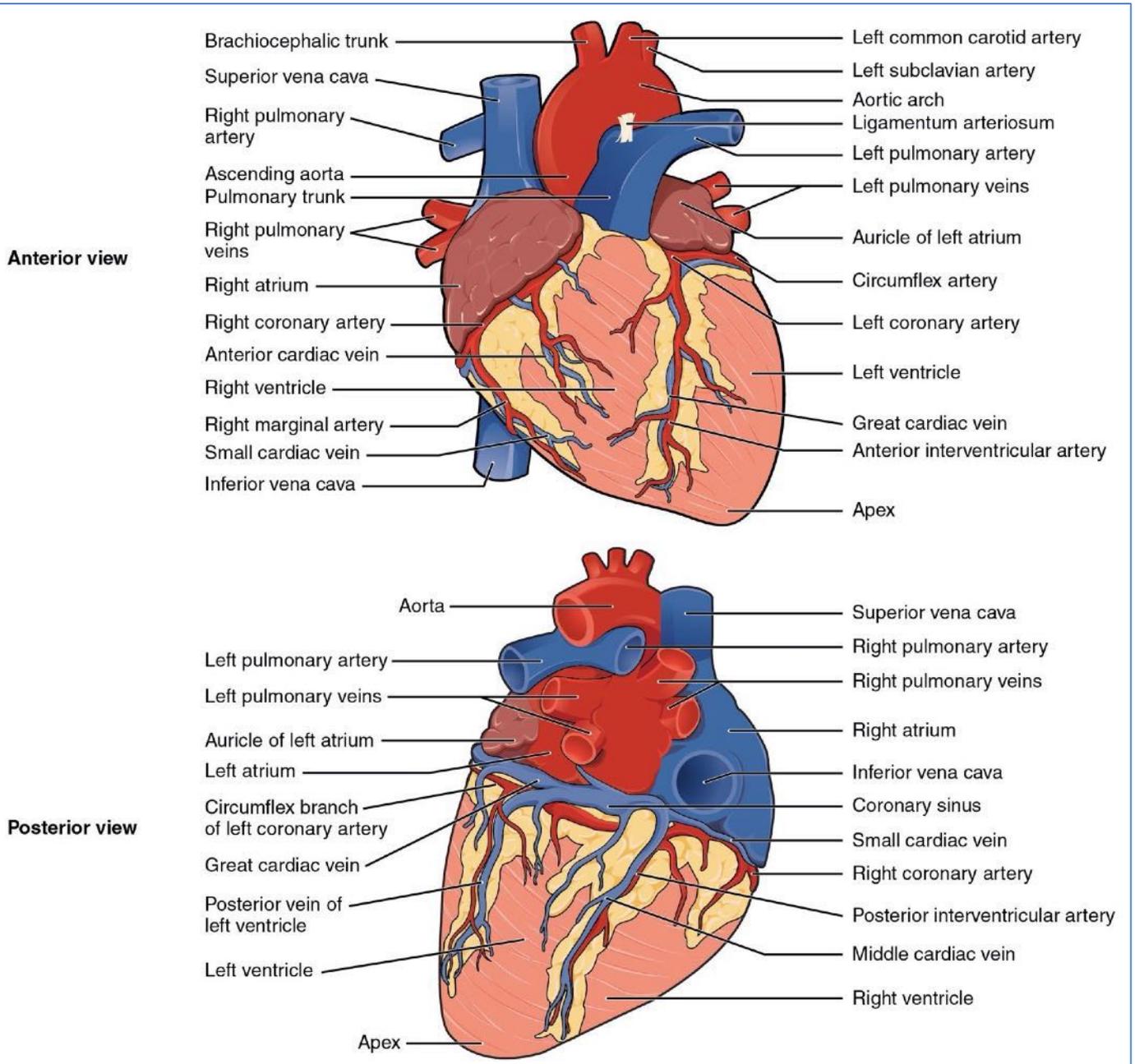
- **2 Atria (superior):** [Atrium = Entryway]
 - Thin-walled Receiving Chambers
 - On the superior aspect of heart (above the ventricles).
 - Each have a small, protruding appendage called **Auricles** – increase atrial volume.
 - Separated by Atrial Septum (Site of Foetal Shunt Foramen Ovale)
 - **Right Atrium:**
 - Ridged internal anterior wall – due to muscle bundles called **Pectinate Muscles**.
 - Blood enters via 3 veins:
 - **Superior Vena Cava**
 - **Inferior Vena Cava**
 - **Coronary Sinus** (collects blood draining from the myocardium)
 - **Left Atrium:**
 - Blood enters via:
 - **The 4 pulmonary veins** (O² blood)
- **2 Ventricles (inferior):** [Vent = Underside]
 - Thick, muscular Discharging Chambers
 - The ‘pumps’ of the heart
 - **Trabeculae Carneae** [crossbars of flesh] line the internal walls
 - **Papillary Muscles** play a role in valve function.
 - **Right Ventricle:**
 - Most of heart’s Anterior Surface
 - Thinner – responsible for the *Pulmonary Circulation* – Via **Pulmonary Trunk**
 - **Left Ventricle:**
 - Most of the heart’s Postero-Inferior Surface
 - Thicker – it is responsible for the *Systemic Circulation* – Via **Aorta**



Anterior view

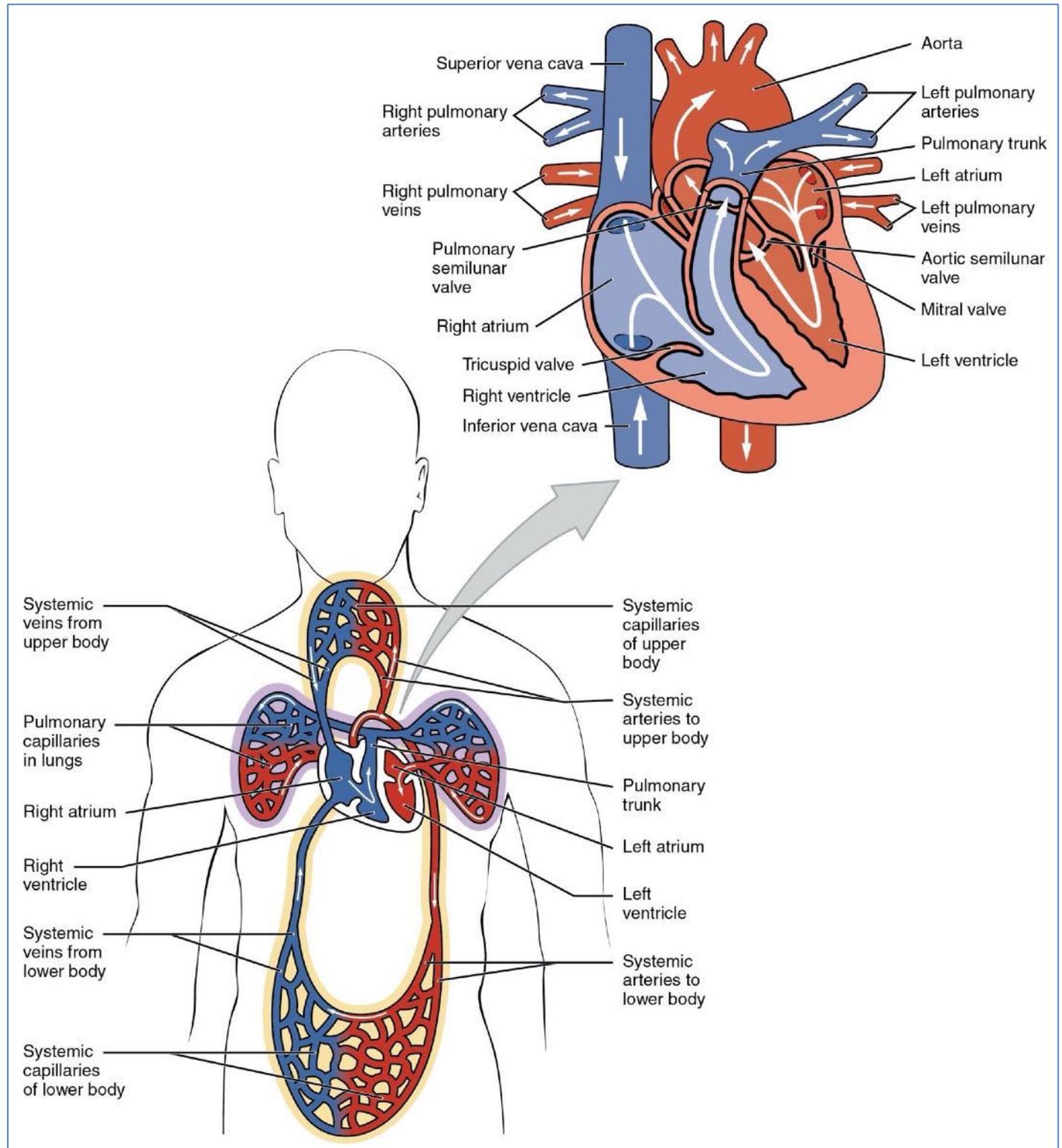
Landmarks of the Heart:

- **Coronary Sulcus (Atrioventricular Groove):**
 - Encircles the junction between the Atria & Ventricles like a 'Crown' (Corona).
 - Cradles the Coronary Arteries (R&L), Coronary Sinus, & Great Cardiac Vein
- **Anterior Interventricular Sulcus:**
 - Cradles the Anterior Interventricular Artery (Left Anterior Descending Artery)
 - Separates the right & left Ventricles anteriorly
 - Continues as the posterior Interventricular Sulcus.
- **Posterior Interventricular Sulcus:**
 - Cradles the Posterior Descending Artery
 - Continuation of the Anterior Interventricular Sulcus
 - Separates the right & left Ventricles posteriorly



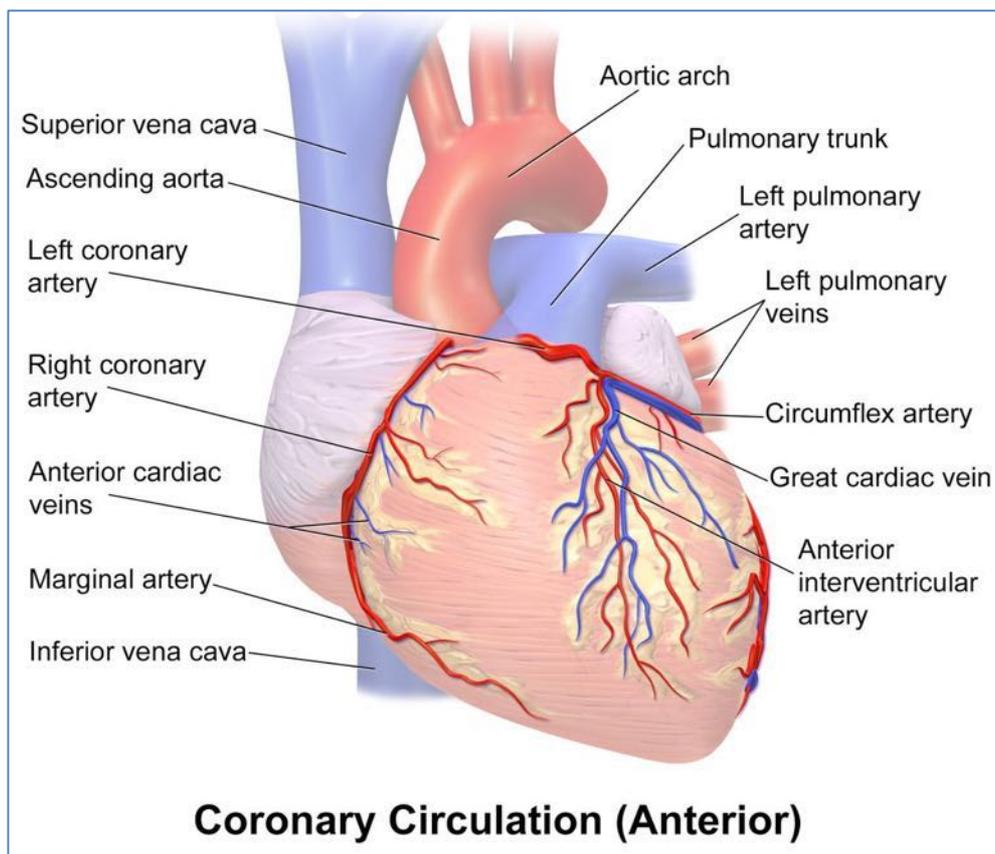
Pathway of Blood Through the Heart:

- **The right side** of the heart pumps blood through the pulmonary circuit (to the lungs and back to the left side of the heart).
 - Blood flowing through the pulmonary circuit gains oxygen and loses carbon dioxide, indicated by the colour change from blue to red.
- **The left side** of the heart pumps blood via the systemic circuit to all body tissues and back to the right side of the heart.
 - Blood flowing through the systemic circuit loses oxygen and picks up carbon dioxide (red to blue colour change)



Coronary Circulation:

- The myocardium's own blood supply
- The shortest circulation in the body
- Arteries lie in epicardium – prevents the contractions inhibiting bloodflow
- There is a lot of variation among different people.
- **Arterial Supply:**
 - Encircle the heart in the coronary sulcus
 - **Aorta** → Left & Right *coronary arteries*
 - **Left Coronary Artery** → 2 Branches:
 - **1- Anterior InterVentricular Artery** (aka. Left Anterior Descending Artery ...or LAD).
 - Follows the Anterior InterVentricular Sulcus
 - Supplies **Apex, Anterior LV, Anterior 2/3 of IV-Septum.**
 - **2- Circumflex Artery**
 - Follows the Coronary Sulcus (aka. AtrioVentricular Groove)
 - Supplies the **Left Atrium + Lateral LV**
 - **Right Coronary Artery** → 2 ('T-junction) Branches:
 - **1- Marginal Artery:**
 - Serves the Myocardium Lateral RHS of Heart
 - **2- Posterior Interventricular Artery:**
 - Supplies posterior ventricular walls
 - Anastomoses with the Anterior Interventricular Artery (LAD)



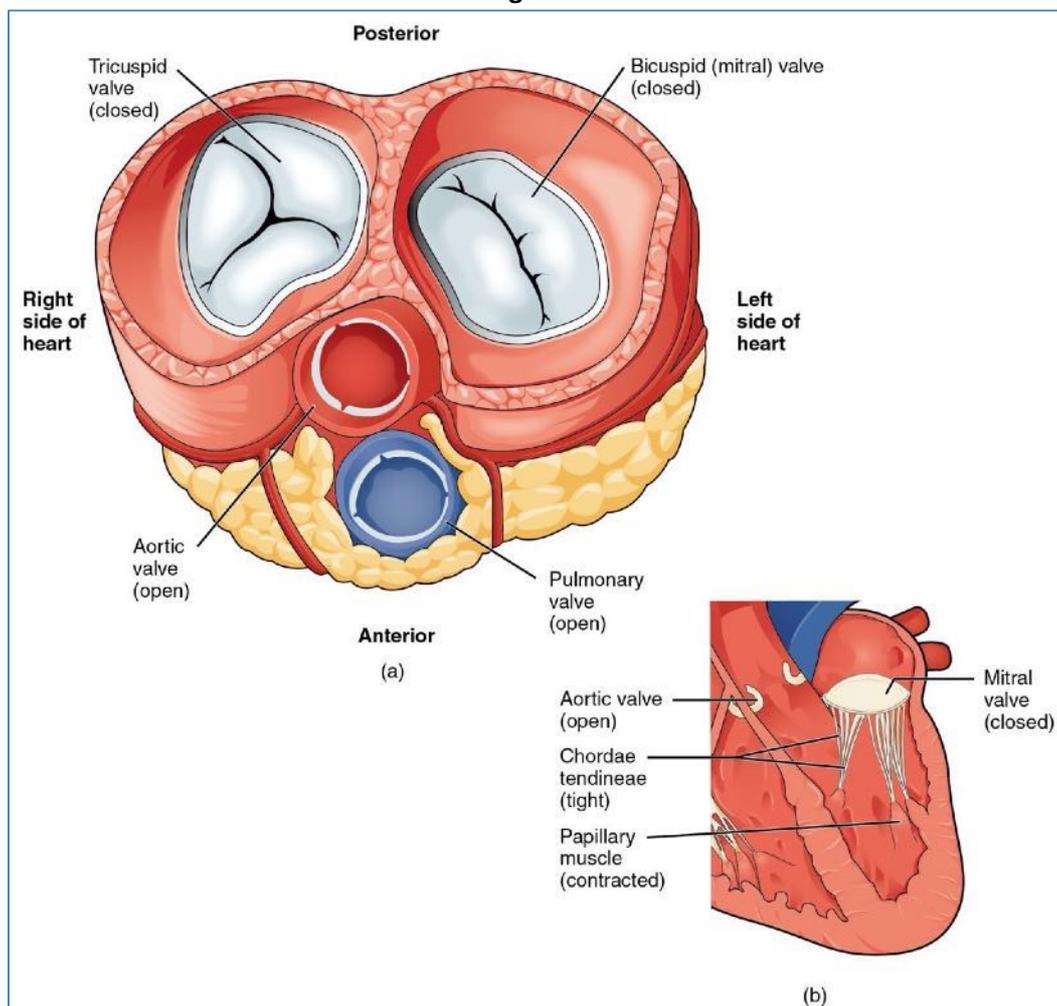
Blausen.com staff (2014). [Blausen Medical Communications, Inc.](#)

- **Venous Drainage:**
 - Venous blood – collected by the **Cardiac Veins:**
 - Great Cardiac Vein (in Anterior InterVentricular Sulcus)
 - Middle Cardiac Vein (in Posterior InterVentricular Sulcus)
 - Small Cardiac Vein (along Right inferior Margin)
 - - Which empties into the **Right Atrium.**

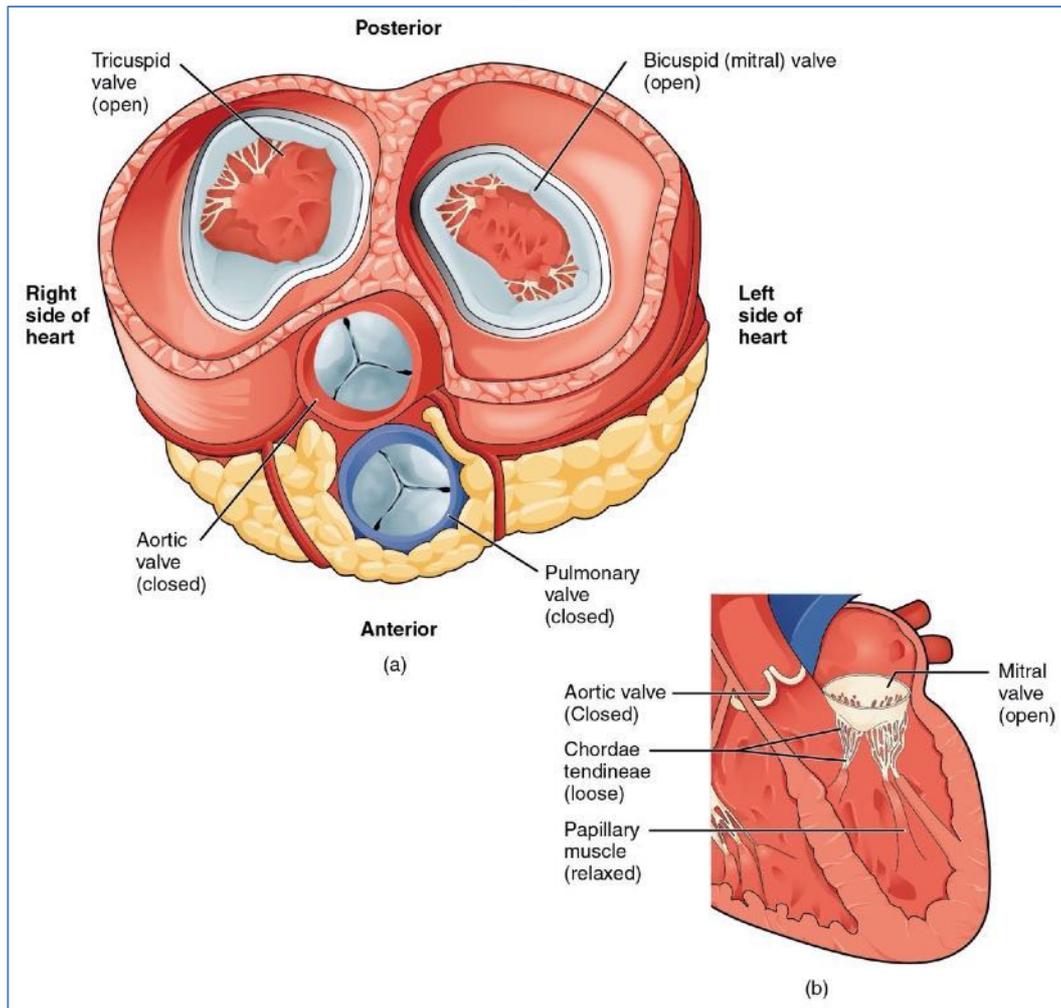
Heart Valves:

- Ensure *unidirectional flow of blood* through the heart.
- **2x AtrioVentricular (AV) (Cuspid) Valves:**
 - **Location:**
 - At the 2 Atrial-Ventricular junctions
 - **Function:**
 - Prevent backflow into the Atria during Contraction of Ventricles
 - **Chordae tendinae** (tendinous cords) “heart strings” - Attached to each valve flap.
 - Anchor the cusps to the **Papillary Muscles** protruding from ventricular walls.
 - Papillary muscles contract before the ventricle to tension the chordae tendinae.
 - Prevent inversion of valves under ventricular contraction.
 - **Tricuspid Valve (Right):**
 - 3 flexible ‘cusps’ (flaps of endocardium + Conn. Tissue)
 - **Mitral Valve (Left):**
 - (resembles the 2-sided bishop’s *mitre* [hat])
- **2x SemiLunar (SL) Valves:**
 - Located at the bases of both large arteries issuing from the Ventricles.
 - Each consists of 3 pocket-like cusps resembling a crescent moon (semilunar = half moon)
 - Open under Ventricular Pressure
 - **Pulmonary Valve:**
 - Between Right Ventricle & Pulmonary Trunk
 - **Aortic Valve:**
 - Between Left Ventricle & Aorta

Valve Positions During Ventricular Contraction



Valve Positions During Ventricular Relaxation

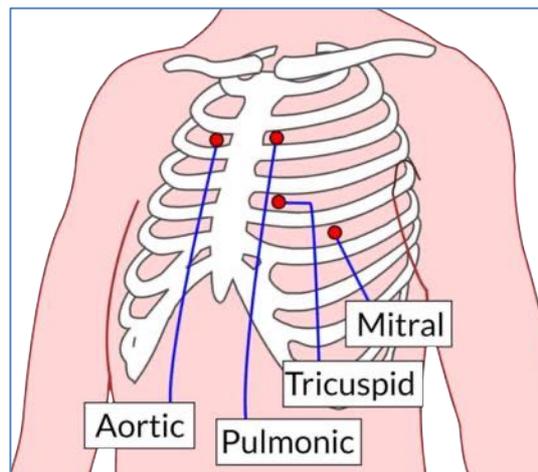


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- **Valve Sounds:**

- **1- "Lubb":**
 - Sound of AV Valve Closure
 - (M1 = Mitral Component)
 - (T1 = Tricuspid Component)
- **2- "Dupp":**
 - Sound of Semilunar Valve Closure
 - (A2 = Aortic Component)
 - (P2 = Pulmonary Component)

- **Where to Listen:**



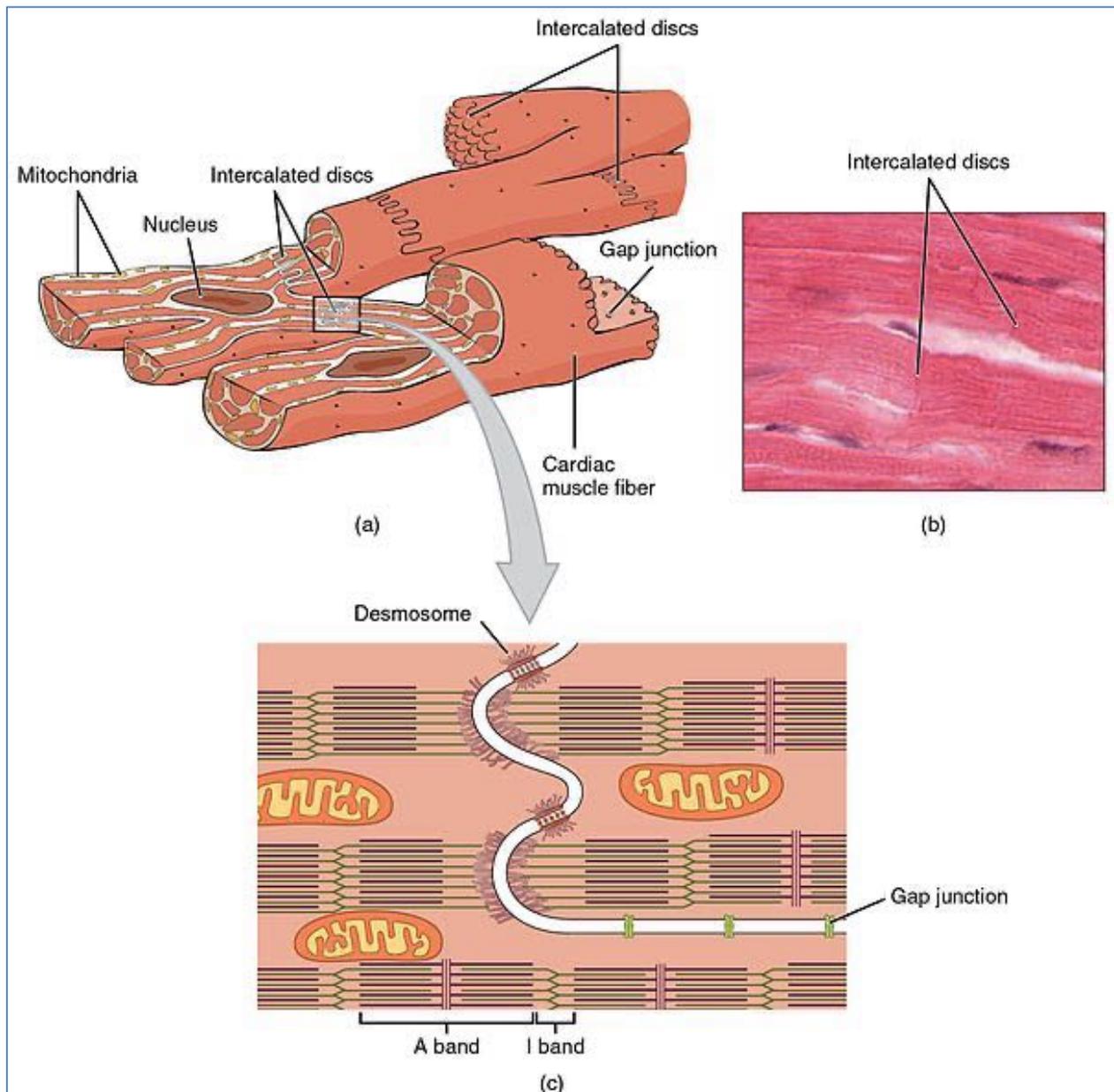
Adaptation of [File:Precordial Leads 2.svg](#) (by [Jmarchn](#)) and Rib_Cage (Jeroen Hut)

ELECTROPHYSIOLOGY OF THE HEART:

ELECTROPHYSIOLOGY OF THE HEART

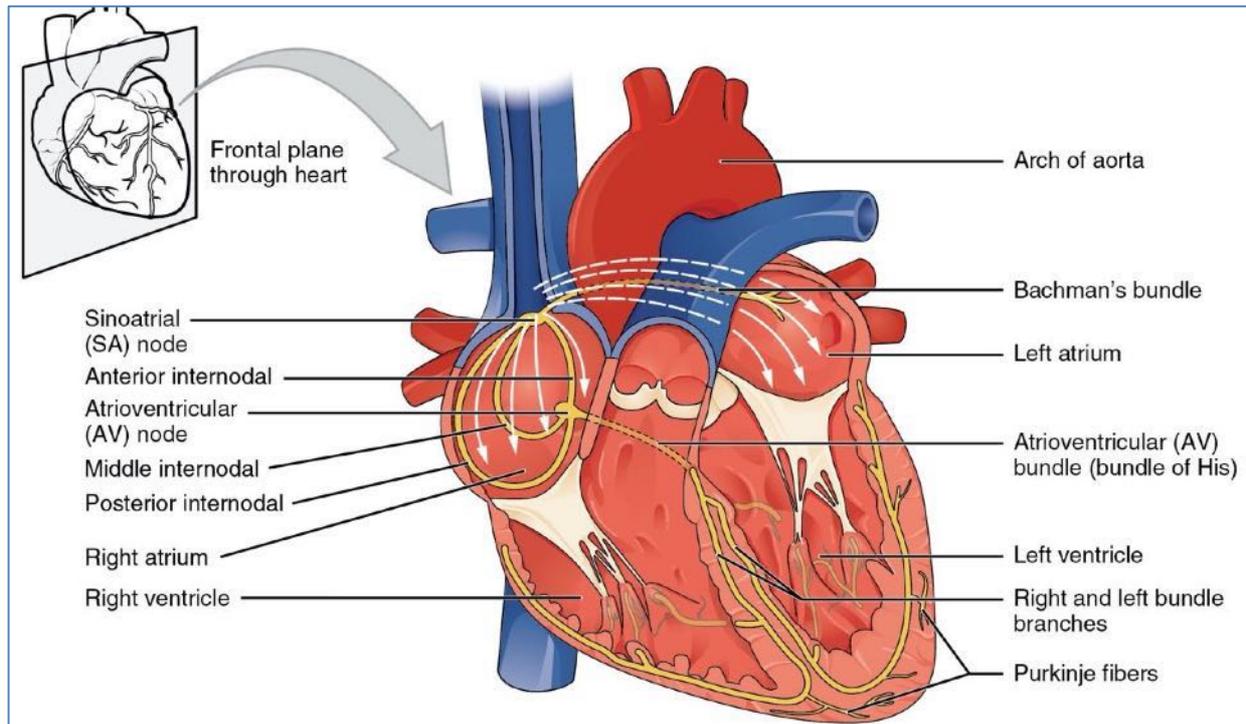
The Heartbeat:

- **Heart is a Muscle & Requires:**
 - o O₂
 - o Nutrients, &
 - o **Action Potentials**; to function.
- **However**, these neural signals don't come from the brain;
 - o Rather, the heart has its **own** conduction systems.
 - These systems **allow it to contract autonomously**
 - o Hence why a *transplanted heart* still operates (if provided with O₂ & nutrients)
- **Cardiac Activity is Coordinated:**
 - o To be effective, the Atria & Ventricles must contract in a **coordinated manner**.
 - o This activity is coordinated by the Heart's Conduction Systems.....
- **The Entire Heart is Electrically Connected...By:**
 - o Gap Junctions:
 - Allows action potentials to move from cell to cell
 - o Intercalated Discs:
 - Support synchronised contraction of cardiac tissue



The Heart's Conduction Systems:

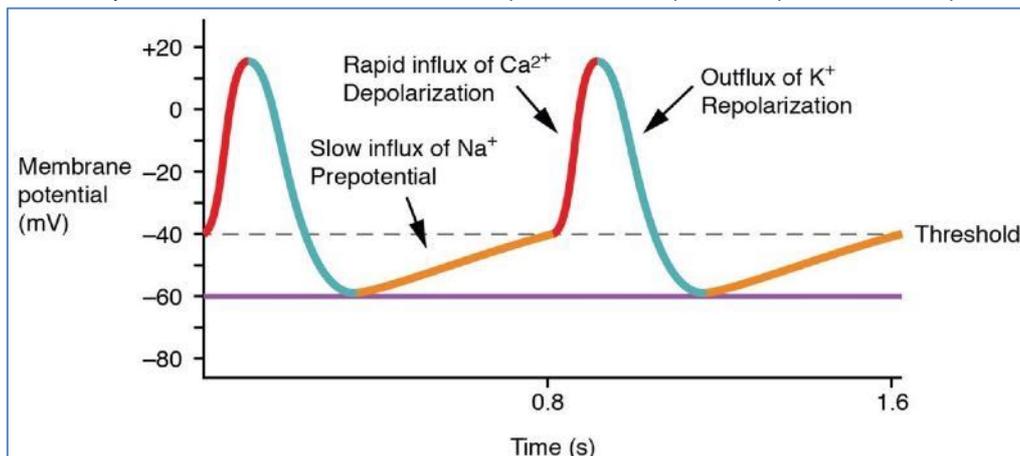
- SA Node → AV Node → Bundle Of His → R & L Bundle Branches → Purkinji Fibres → Myocyte Contraction



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Conductile Cardiac Cell Physiology (SA/AV Node Cells):

- **Action Potentials: Slow 'Pacemaker' Type**
- **Have UNSTABLE Resting Membrane Potentials → Spontaneous Electrical Activity:**
 - o **Spontaneously Depolarises to Threshold**
 - This gradual depolarisation is called a '*Prepotential*'.
 - Due to Leaky Na^+ Membrane Ion Channels
 - Therefore – Firing Frequency Depends on Na^+ Movement
 - o **Depolarisation:**
 - Once Threshold is reached, Ca^{2+} channels open
 - → Influx of Ca^+
 - → Causes an action potential.
 - o **Repolarisation:**
 - Once peak MP is reached, Ca^+ channels close, K^+ channels open
 - → K^+ Efflux makes MP more $\bar{\text{v}}$ e
 - → Causes repolarisation
 - o **(Na^+ brings to threshold, but Ca^+ is responsible for Depolarisation.)**
- **With a Hierarchy of control over the heart.**
 - o Hierarchy based on *natural intrinsic rate*. (fastest node (SA node) takes control)

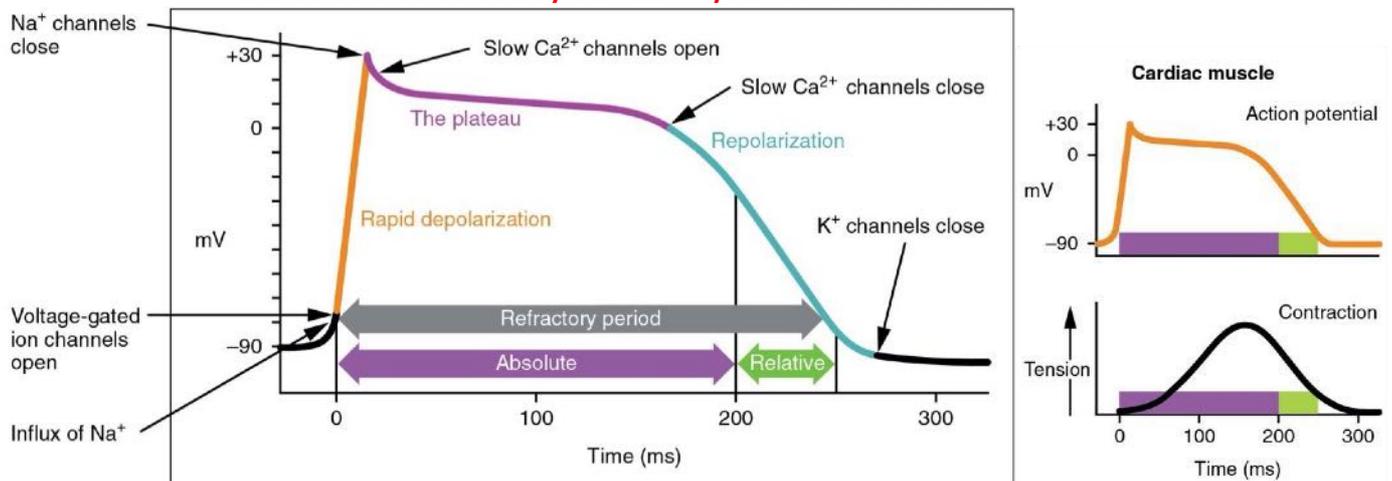


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Contractile Cardiac Cell Physiology (Purkinje Fibres & Myocytes):

- **Action Potentials: Fast 'Non-Pacemaker' Type**
- **Have STABLE Resting Membrane Potentials.**
 - o **Resting Membrane Potential (MP):**
 - Na^+ & Ca^+ channels are closed.
 - Any +ve change to MP causes Fast Na^+ channels to open \rightarrow +ve feedback \rightarrow Threshold
 - o **Depolarisation:**
 - If MP reaches threshold, all Fast Na^+ channels open;
 - \rightarrow Massive influx of Na^+ into cell
 - \rightarrow Membrane depolarises
 - o **Plateau:**
 - Fast Na^+ channels inactivate.
 - \rightarrow The small downward deflection is due to Efflux of K^+ ions
 - \rightarrow Action potential causes membrane Voltage-Gated Ca^+ channels to open
 - This triggers further Ca^+ release by the Sarcoplasmic Reticulum into the Sarcoplasm. ("Ca induced Ca Release")
 - o This increased myoplasmic Ca^+ causes muscular contraction.
 - Plateau is sustained by influx of Ca^+ , balanced by efflux of K^+ ions
 - o **Repolarization:**
 - Influxing Ca^+ channels close.....The effluxing K^+ channels remain open;
 - \rightarrow Result is a net *outward* flow of +ve charge. \rightarrow Downward Deflection
 - \rightarrow As the MP falls, more K^+ channels open, accelerating depolarization.
 - \rightarrow Membrane Repolarizes & most of the K^+ channels close.
 - o **What Happens to the Excess Ions??**
 - Excess Na^+ in the cell from depolarization is removed by the Na/K-ATPase.
 - Deficit of K^+ in the cell from repolarization is replaced by the Na/K-ATPase.
 - Excess Ca^+ from the Plateau Phase is eliminated by a Na/Ca Exchanger.

NOTE: There is a considerable delay between Myocardial Contraction & the Action Potential.



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Refractory Periods:

- In Cardiac Muscle, the *Absolute Refractory Period* continues until muscle relaxation;
 - o Therefore summation isn't possible \rightarrow tetany cannot occur (critical in heart)
 - o Ie: The depolarised cell won't respond to a 2nd stimulus until contraction is finished.
- **Absolute Refractory Period:**
 - o Approx 200ms
 - o Duration: from peak \rightarrow plateau \rightarrow halfway-repolarised.
- **Relative Refractory Period:**
 - o Na^+ channels are closed – but can still respond to a stronger-than-normal stimulus.
 - o Approx 50ms
 - o Duration: Last half of repolarisation

The SinoAtrial (SA) Node:

- = The **"PaceMaker"** of the Heart: Unregulated Rate: 90-100bpm.....however;
 - o Parasympathetic NS lowers heart rate → Keeps Normal Resting HR at 70bpm
 - o Sympathetic NS raises heart rate.
- **Location:**
 - o *Posterior Wall* of the *Right Atrium* near the opening of the *Superior Vena Cava*
- **Nature of Action Potentials:**
 - o Continually Depolarizing 90-100bpm
 - o Takes 50ms for Action-Potential to reach the AV Node.
- **Role in Conduction Network:**
 - o Sets the pace for the heart as a whole.
- **Portion of Myocardium Served:**
 - o Contracts the Right & Left Atrium

The AtrioVentricular (AV) Node:

- **2nd in Command:** Slower than the SA Node: 40-60bpm
- **Location:**
 - o Inferior portion of the InterAtrial Septum; Directly above the TriCuspid Valve.
- **Nature of Action Potentials:**
 - o Continually Depolarizing – but slower than the SA Node. (40-60bpm)
- **Role in Conduction Network:**
 - o To delay the impulse from the SinoAtrial Node → Bundle Branches;
 - o Delay allows the Atria to empty their contents before Ventricular Contraction
 - o Delay: Approx. 100ms
- **Portion of Myocardium Served:**
 - o Conducts the SA Node Impulses to the Purkinje Fibres (which supply the Ventricular Walls)

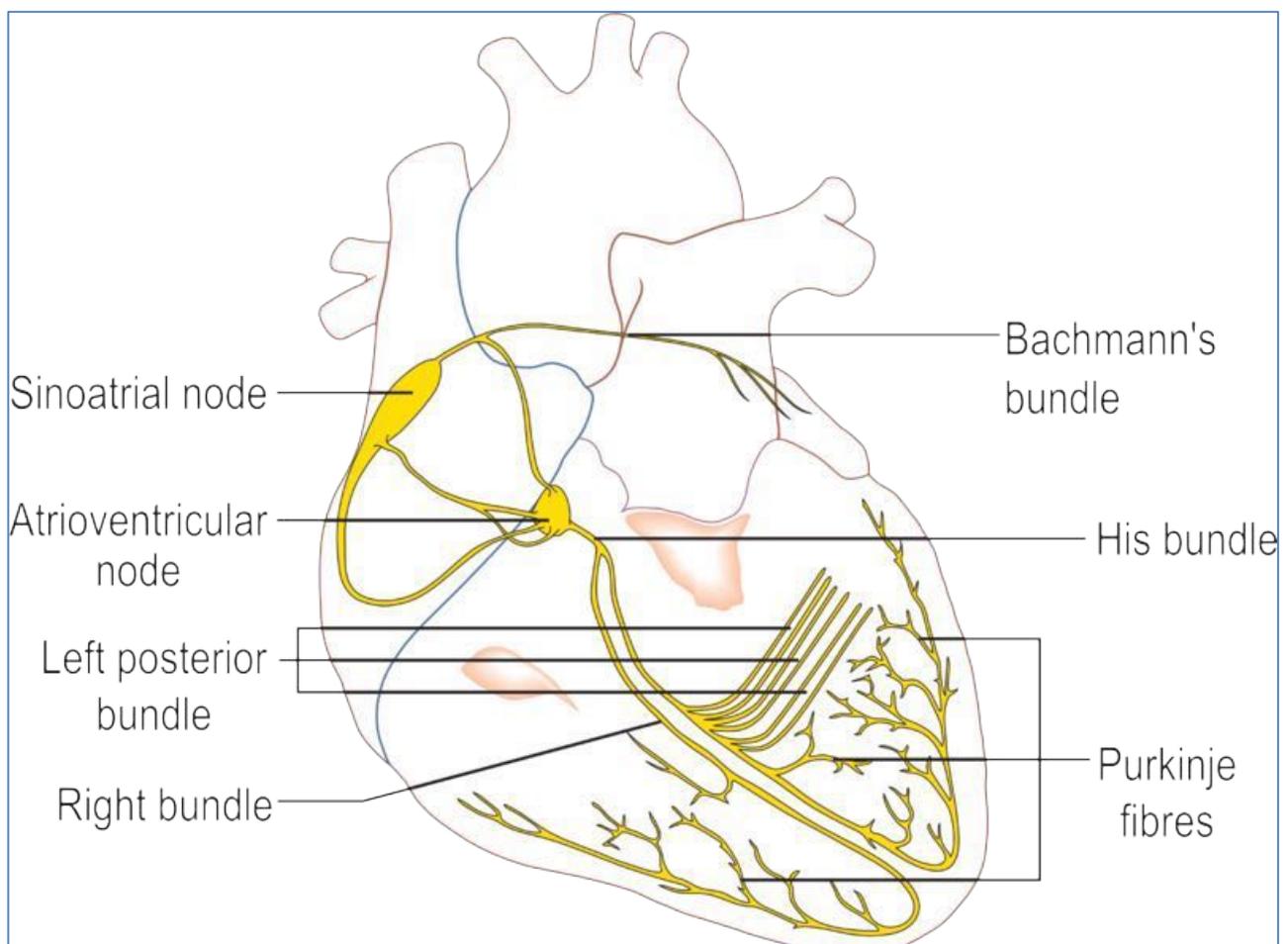


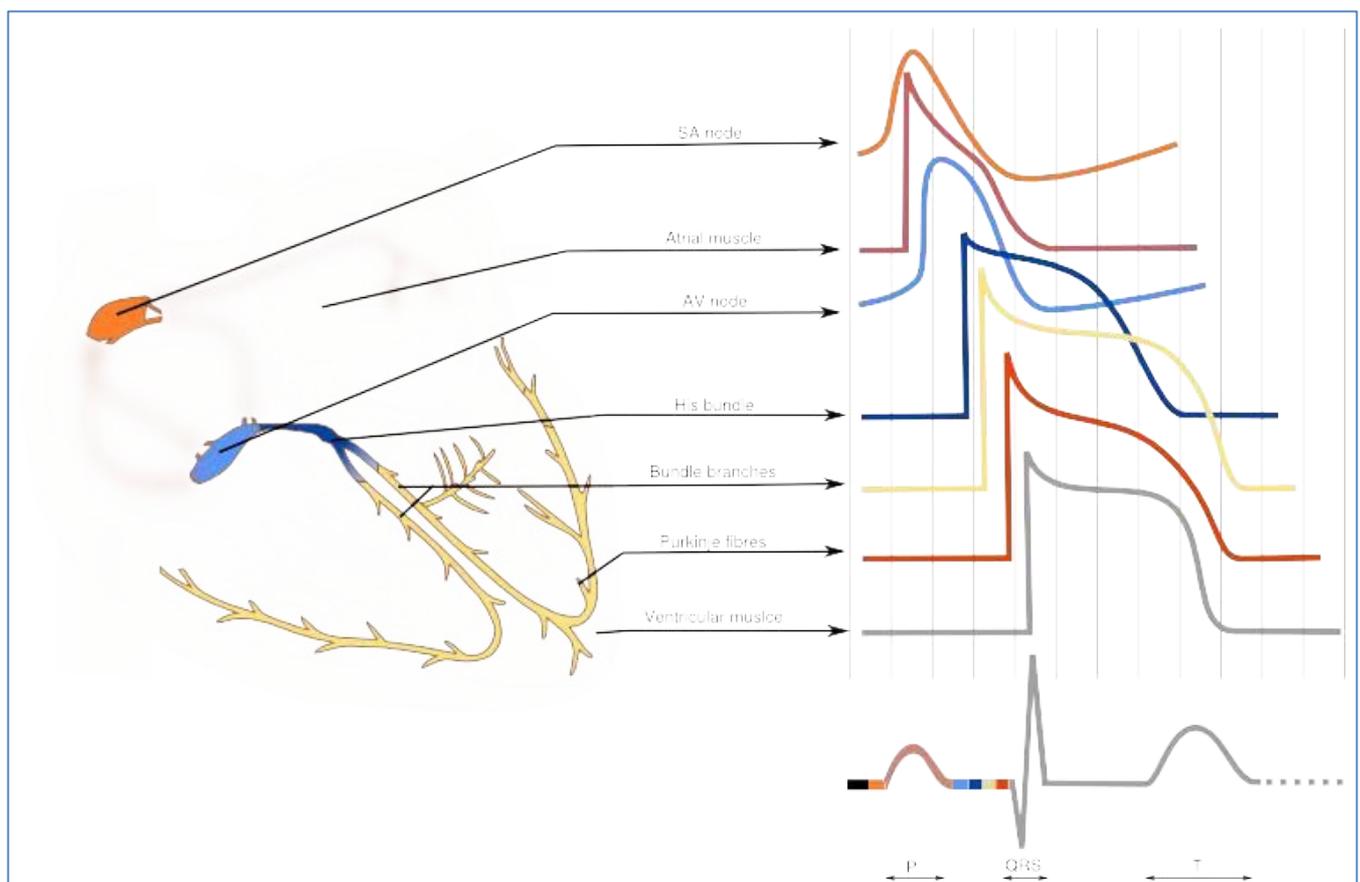
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The Bundle Branches (Bundles of His):

- **3rd in Command:** Slower than AV & SA Nodes: 20-40bpm
- **Location:**
 - o Fork of branches – Superior Portion of InterVentricular Septum
- **Nature of Action Potentials:**
 - o Continually Depolarising – Slower than AV & SA Nodes (20-40bpm)
- **Role in Conduction Network:**
 - o Serves as the only connection between the 2 Atria & 2 Ventricles.
 - o The 2 Atria & 2 Ventricles are isolated by the fibrous skeleton and lack of gap junctions.
- **Portion of the Myocardium Served:**
 - o Transmits impulses from the AV Node to the R & L Bundle Branches,
 - Then along the InterVentricular Septum → Apex of the Heart.

The Purkinje Fibres:

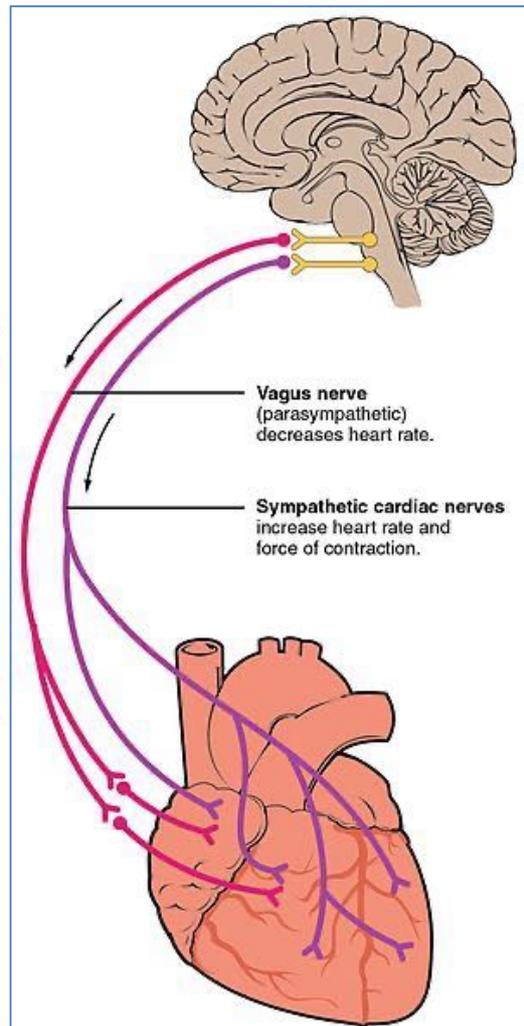
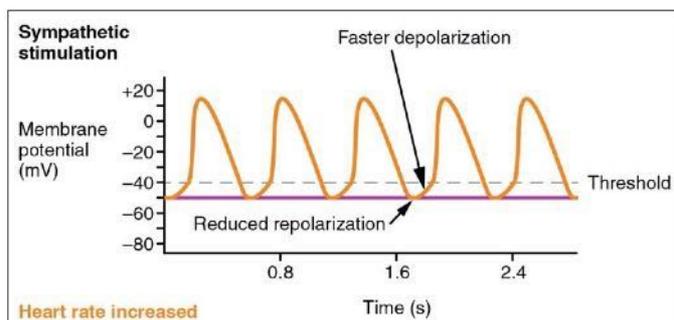
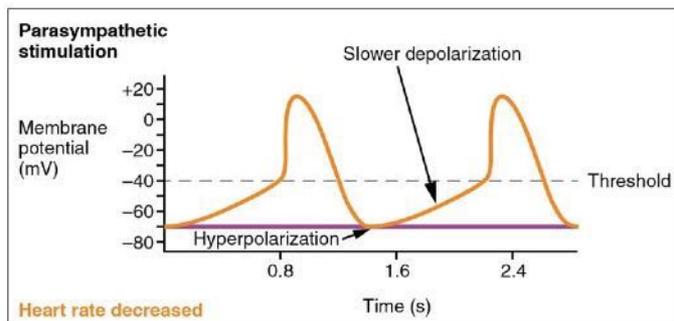
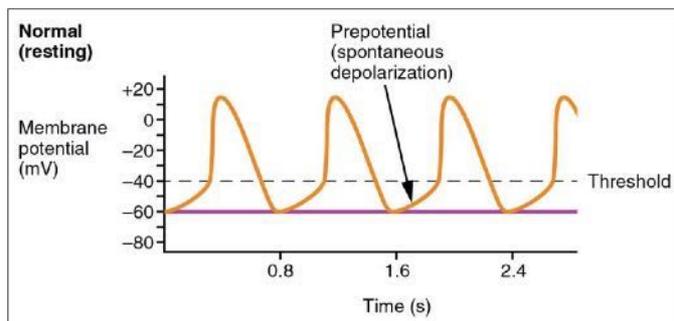
- **Specialised Myocytes with very few myofibrils** → don't contract during impulse transmission.
- **Location:**
 - o The Inner Ventricular Walls of the Heart – just below the Endocardium
 - o Begin at the heart apex, then turn superiorly into the Ventricular Walls.
- **Nature of Action Potentials:**
 - o Conductile; but...Resembles those of Ventricular Myocardial Fibers;
 - However the Depolarisation is more pronounced & Plateau is longer.
 - Long Refractory period
 - o Capable of Spontaneous Depolarisation – 15bpm
- **Role in Conduction Network:**
 - o Carry the contraction impulse from the L & R Bundle Branches to the *Myocardium* of the *Ventricles*;
 - o Causes Ventricles to Contract.
- **Portion of Myocardium Served:**
 - o R & L Ventricles.



CardioNetworks: [De-Conduction_ap.png](#)

Effects of the Autonomic Nervous System (ANS):

- Although the heart *can* operate on its own, It *normally* communicates with the brain via the A.N.S.
- **Parasympathetic NS:**
 - o Innervates SA & AV Nodes → Slows Heart Rate
 - o **Direct Stimulation** → Releases AcetylCholine → *Muscarinic* receptors in SA/AV Nodes →
 - Causes increased K^+ permeability (Efflux) → *Hyperpolarises* the cell →
 - Cell takes longer to reach threshold → **Lower Heart Rate**
- **Sympathetic NS:**
 - o Innervates the SA & AV Nodes & Ventricular Muscle.
 - → Raises Heart Rate
 - → Increases Force of Contraction
 - → Dilates Arteries
 - o **Indirect Stimulation** → Sympathetic Nerve Fibres Release NorAdrenaline (NorEpinephrine) @ their cardiac synapses → Binds to *Beta 1* Receptors on Nodes & Muscles →
 - Initiates a Cyclic AMP Pathway → Increases Na^+ + Ca^+ Permeability in Nodal Tissue & Increases Ca^+ Permeability_(Membrane & SR) in Muscle Tissue.
 - o **Effects on Nodal Tissue:**
 - ++Permeability to Na^+ → more influx of Na^+ → Membrane 'drifts' quicker to threshold → Increased Heart Rate
 - ++Permeability to Ca^+ → more influx of Ca^+ → Membrane Depolarisation is quicker → Increased Heart Rate
 - o **Effects on Contractile Tissue:**
 - ++ Membrane Permeability to Ca^+ → More influx of Ca^+ →
 - ++Sarcoplasmic Reticulum Permeability to Ca^+ → Efflux of Ca^+ into cytoplasm →
 - Increases available Ca^+ for contraction → Contractile Force Increases

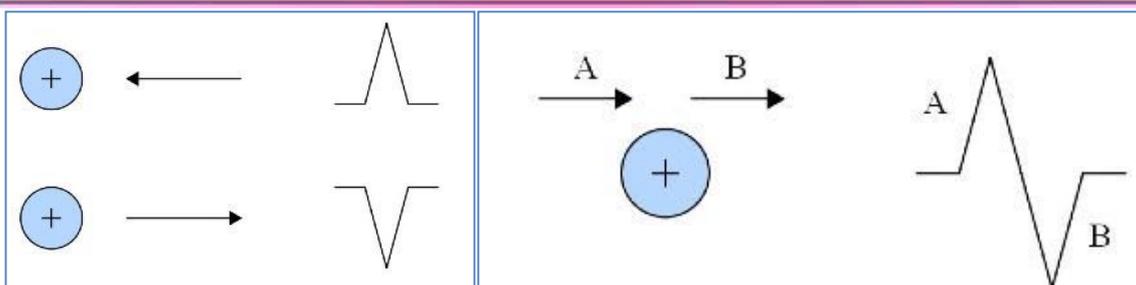
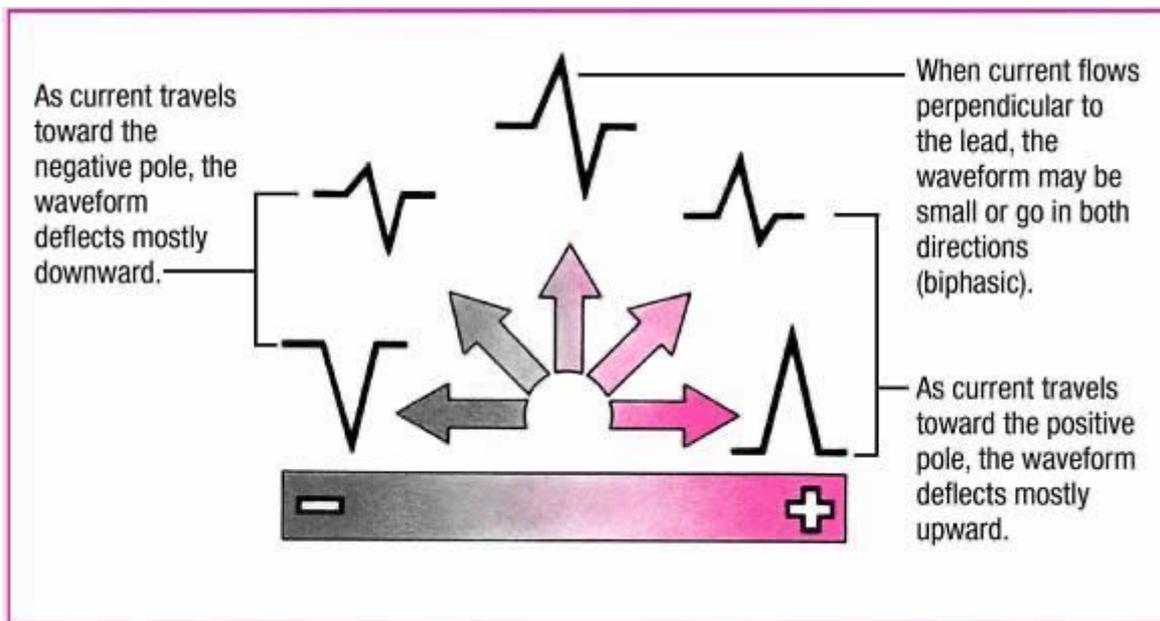


ELECTROCARDIOGRAM (ECG) PHYSIOLOGY:

ELECTROCARDIOGRAM (ECG) PHYSIOLOGY:

What Is An ECG?

- **A Recording of all Action Potentials by Nodal & Contractile Cells in the heart at a given time.**
 - o NOTE: It IS NOT a single action potential.
 - o NOTE: A "Lead" refers to a combination of *electrodes* that form an *imaginary line* in the body, along which the electrical signals are measured.
 - Ie: A 12 'lead' ECG usually only uses 10 electrodes.
- **Measured by VoltMetres → record electrical *potential* across 2 points:**
 - o **3x Bipolar Leads:** Measure Voltages between the Arms...OR...Between an Arm & a LEG:
 - I = LA (+) RA (-)
 - II = LL (+) RA (-)
 - III = LL (+) LA (-)
 - o **9x Unipolar Leads:**
 - Look at the heart in a '3D' Image.
 - o (A "Lead" refers to a combination of *electrodes* that form an *imaginary line* in the body, along which the electrical signals are measured. Ie: A 12 'lead' ECG usually only uses 10 electrodes.)
- **Graphic Output:**
 - o X-axis = Time
 - o Y-axis = Amplitude (voltage) – Proportional to number & size of cells.
- **Understanding Waveforms:**
 - o When a Depolarisation Wavefront moves toward a positive electrode, a *Positive* deflection results in the corresponding lead.
 - o When a Depolarisation Wavefront moves away from a positive electrode, a *Negative* deflection results in the corresponding lead.
 - o When a Depolarisation Wavefront moves *perpendicular* to a positive electrode, it first creates a positive deflection, then a negative deflection.



Based on [ECG Vector.jpg](#) by [MoodyGroove](#)

How Each Wave & Segment Is Formed:

P – Wave:

- Depolarization of the Atria
- Presence of this waves indicates the SA Node is working



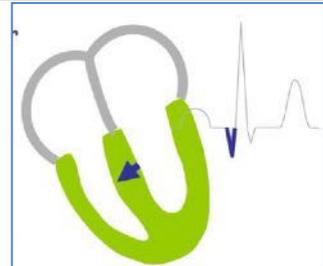
PR-Segment:

- Reflects the delay between SA Node & AV Node.
- Atrial Contraction is occurring at this time.



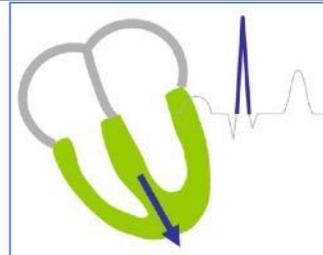
Q – Wave:

- Interventricular Septum Depolarization
- Wave direction (see blue arrow) is perpendicular to the Main Electrical Axis → results in a 'Biphasic' trace.
 - o Only the –ve deflection is seen due to signal cancellation by Atrial Repolarization.
 - o Sometimes this wave isn't seen at all



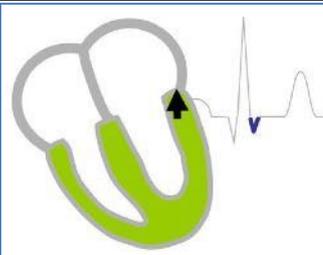
R – Wave:

- Ventricular Depolarization
- Wave Direction (blue arrow) is the same as the Main Electrical Axis → Positive Deflection.
- R-Wave Amplitude is large due to sheer numbers of depolarizing myocytes.



S – Wave:

- Depolarisation of the Myocytes at the last of the Purkinje Fibres.
- Wave Direction (black arrow) opposes the Main Electrical Axis → Negative Deflection
- This wave is not always seen.



ST – Segment:

- Ventricular Contraction is occurring at this time.
 - o Due to the lag between excitation & contraction.



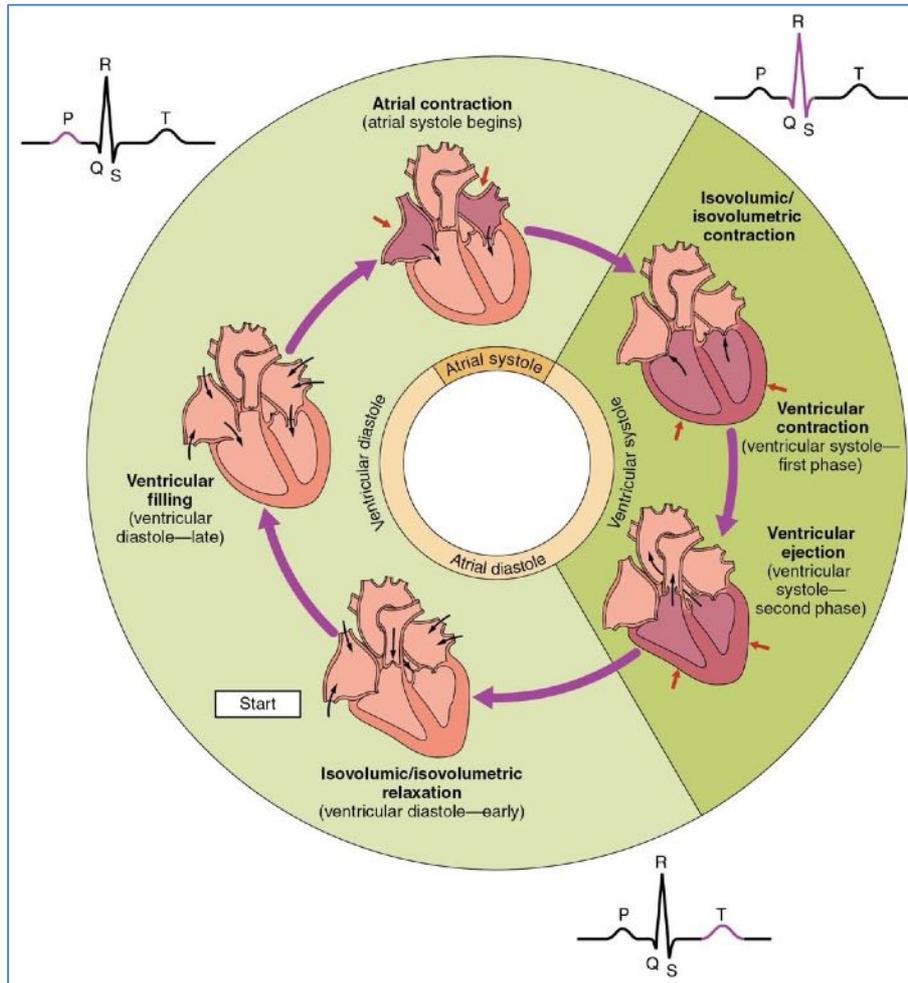
T – Wave:

- Ventricular Repolarisation
- Positive deflection despite being a Repolarisation wave – because Repol. Waves travel in the opposite direction to Depol Waves.

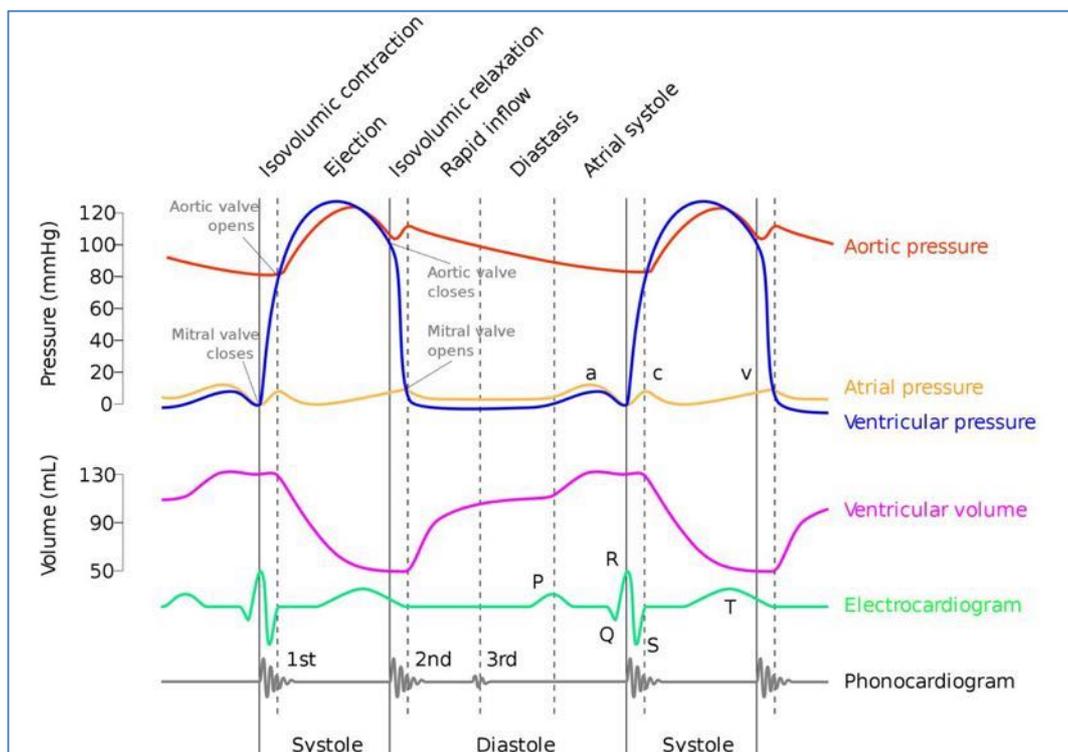


Relating ECG Waves To Events In The Cardiac Cycle:

- Contractions of the Heart ALWAYS Lag Behind Impulses Seen on the ECG.
- Fluids move from High Pressure → Low Pressure
- Heart Valves Ensure a *UniDirectional* flow of blood.
- Coordinated Contraction Timing – Critical for Correct Flow of Blood.



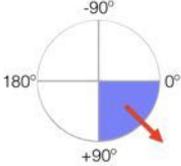
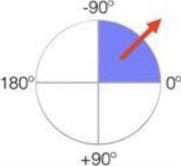
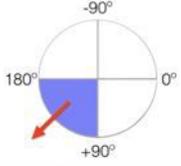
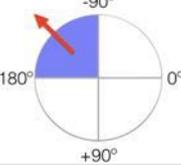
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Wikimedia Commons: Wiggers Diagram.svg

The Heart's Electrical Axis:

- Refers to the general direction of the heart's depolarisation wavefront (or 'mean electrical vector') in the frontal plane.
- It is usually oriented in a 'Right Shoulder to Left Leg' direction.
- **Determining The Electrical Axis From an ECG Trace:**
 - **3 Methods:**
 - Quadrant Method (the one you're concerned with)
 - Peak Height Measurement Method
 - The Degree Method
 - **The Quadrant Method:**

Lead I	Lead aVF	Quadrant	Axis
POSITIVE	POSITIVE		Normal Axis (0 to +90°)
POSITIVE	NEGATIVE		**Possible LAD (0 to -90°)
NEGATIVE	POSITIVE		RAD (+90° to 180°)
NEGATIVE	NEGATIVE		Extreme Axis (-90° to 180°)

Source: unable to attribute.

- **Normal Axis.** QRS positive in I and aVF (0 to 90 degrees). Normal axis is actually 30 to 105 degrees.
- **Left Axis Deviation (LAD).** QRS positive in I and negative in aVF, 30 to 90 degrees
- **Right Axis Deviation (RAD).** QRS negative in I and positive in aVF, +105 to +180 degrees
- **Extreme RAD.** QRS negative in I and negative in aVF, +180 to +270 or 90 to 180 degrees

Algorithm For Looking At ECGs:

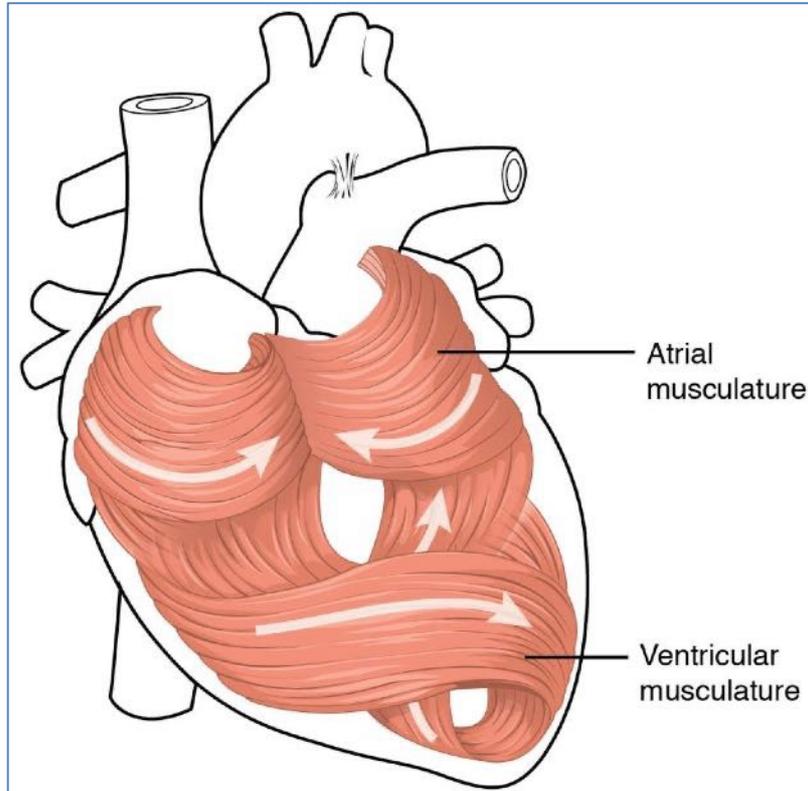
- **Check Pt ID**
- **Check Voltage & timing**
 - o 25mm/sec
 - o 1large square = 0.2s (1/5sec)
 - o 1small square = 0.04s
- **What is the rate?**
 - o 300/number of large squares between QRS Complexes
 - **Tachycardia**
 - >100bpm
 - **Bradycardia**
 - <60bpm
- **What is the Rhythm?**
 - o Sinus? (are there P-Waves before each QRS complex)
 - o If Not Sinus?
 - Is it regular
 - Irregular?
 - Irregularly Irregular (AF)
 - Brady/Tachy
- **Atrial Fibrillation:**
 - o Irregularly Irregular
 - o P-Waves @ 300/min
- **QRS:**
 - o Is there one QRS for each Pwave?
 - o Long PR Interval? (1st degree heart block)
 - o Missed Beats? (Second degree block)
 - o No relationship? Complete heart block
- **Look for QRS Complexes:**
 - o How wide – should be < 3 squares
 - o If wide – It is most likely Ventricular
 - o (Sometimes atrial with aberrant conduction (LBBB/RBBB)
 - o IF Tachycardia, & Wide Complex → VT is most likely. (If hypotensive → Shock; if Normotensive → IV Drugs)
- **Look for TWaves:**
 - o Upright or Inverted
- **Look at ST-Segment**
 - o Raised, depressed or inverted
 - o ST Distribution → Tells you which of the coronaries are blocked/damaged
 - Inferior ischaemia (II, III, AVF)
 - Lateral ischaemia (I, II, AVL, V5, V6)
 - Anterior ischaemia (V, leads 2-6)
 - o NOTE: Normal ECG Doesn't exclude infarct.
 - o ST Depression → Ischaemia
 - o ST Elevation → Infarction
 - o If LBBB or Paced, you CANNOT comment on ST-Segment

MECHANICAL EVENTS OF THE CARDIAC CYCLE

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Structure-Function Relationship of the Heart

- **The Myocardium is essentially *one long muscle* orientated in a spiral-like fashion**
 - This allows the heart to be electrically integrated
 - Allows the heart to 'wring out' the blood within it
 - This setup facilitates a Strong Pumping Action.

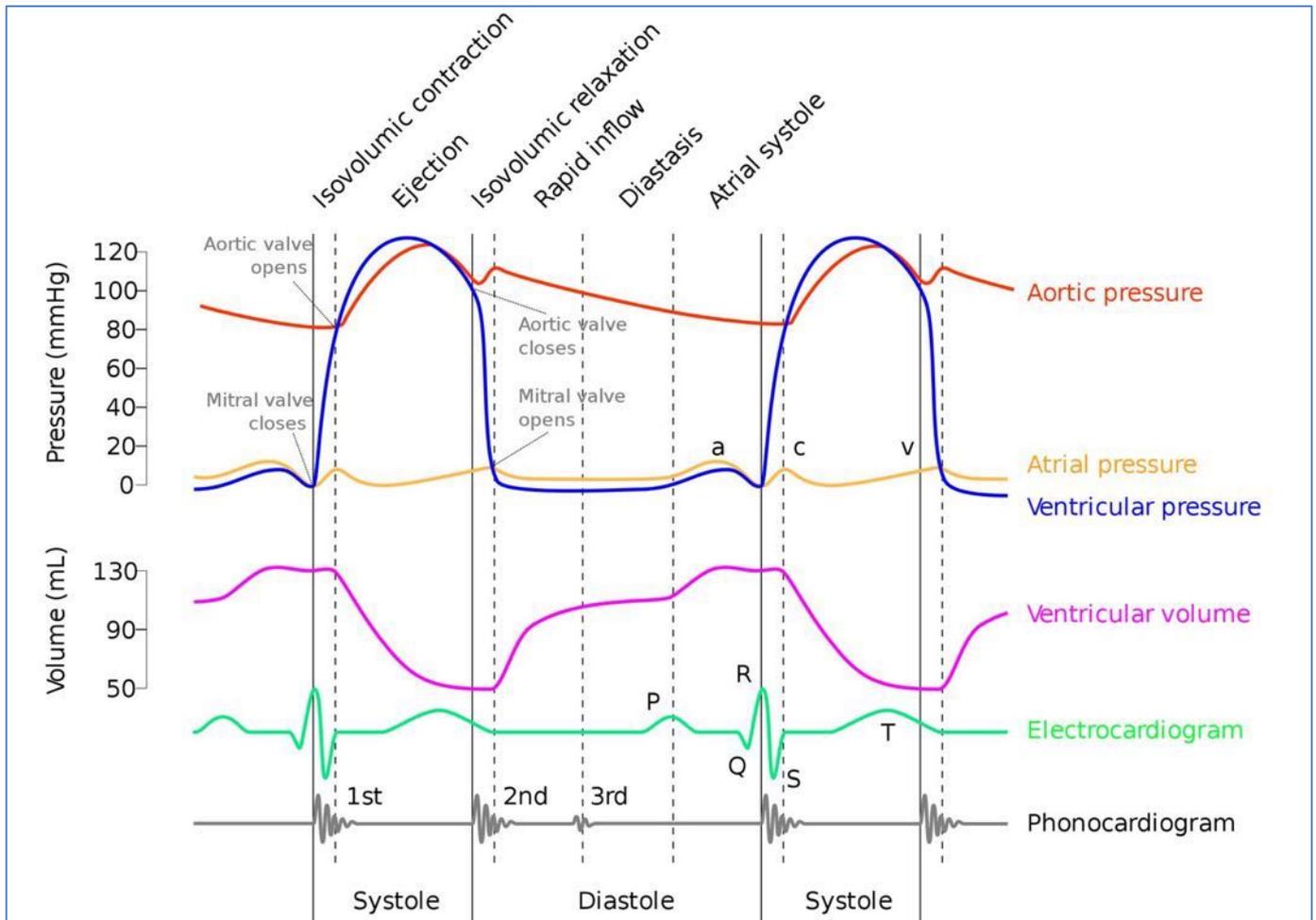


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Terms:

- **Systole** = Myocardial Contraction
- **Diastole** = Myocardial Relaxation
- **Stroke Volume** = Output of Blood from the Heart *Per Contraction* (≈80mL of blood)
- **Heart Rate** = #Heart Beats/Minute
- **Cardiac Output:**
 - Volume of Blood Ejected from the Heart *Per Minute* (Typically ≈5L/min)
 - **Cardiac Output = Heart Rate x Stroke Volume**
 - **Chronotropic Influences:**
 - Affect *Heart Rate*
 - **Inotropic Influences:**
 - Affect *Contractility* (& ∴ stroke volume)
 - **Dromotropic Influences:**
 - Affect *AV-Node Delay*.
- **End Diastolic Volume** = Ventricular Volume @ end of Diastole (When Ventricle is *Fullest*)
- **End Systolic Volume** = Ventricular Volume After Contraction (Normal ≈ 60-65%)
- **Preload** = The degree of *Stretching* of the Heart Muscle during Ventricular Diastole.
 - (↑Preload = ↑cross linking of myofibrils = ↑Contraction ("*Frank Starling Mechanism*")
- **Afterload** = The Ventricular Pressure required to *Eject* blood into Aorta/Pulmonary Art.
 - (↑Afterload = ↓SV due to ↓ejection time)

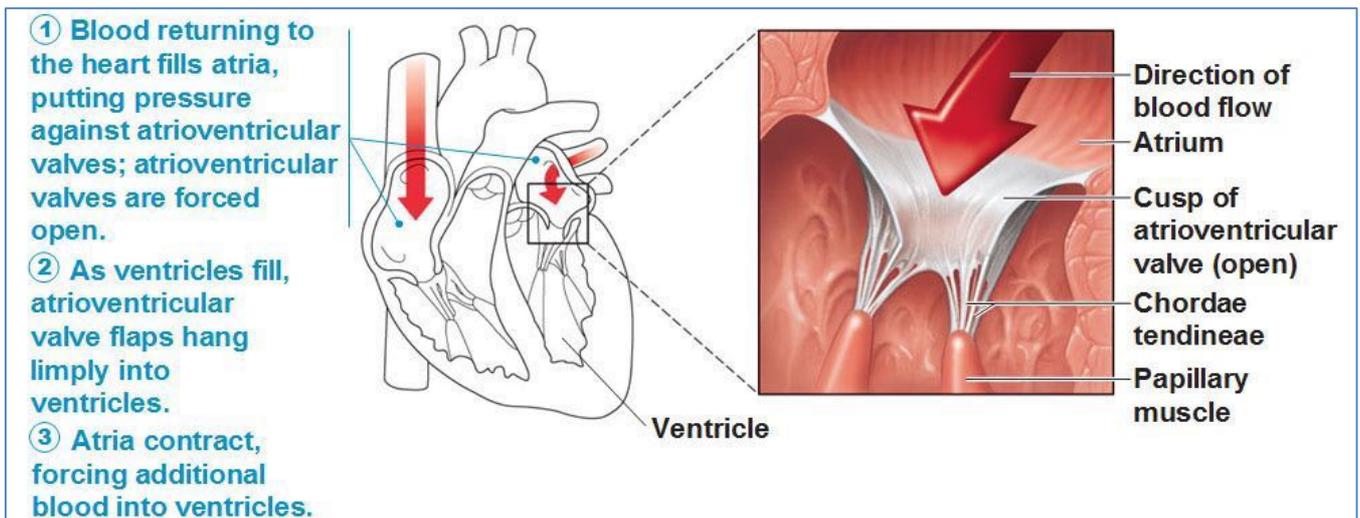
Overview of the Cardiac Cycle in One Diagram:



Wikimedia Commons: Wiggers Diagram.svg

PHASE 1- Atrial Contraction (Systole) + Ventricular Filling (Diastole):

- **Contraction of Atria**
 - → IntraAtrial Pressure Increases
 - → Blood pushed into Ventricles through AV-Valves
- **Note:** Ventricles are already 70% full from passive Venous Filling.
- At End of Atrial Systole, Ventricles have EDV (End Diastolic Volume) ≈ 130mL

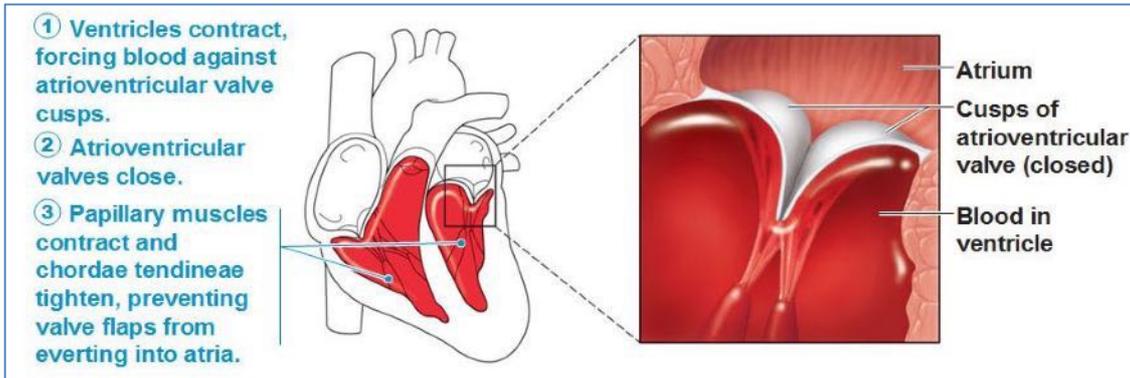


Source: antranik.org

- **PHASE 2- Ventricular Systole:**

o **a) AV Valves Close:**

- Ventricular Pressure Exceeds Atrial Pressure → AV Valves shut
- **Brief period of 'IsoVolumetric' Contraction:**
 - Where the ventricular pressure rises, but *Volume Stays Constant*.
 - The beginning of ventricular systole
 - All valves are still *Closed*.



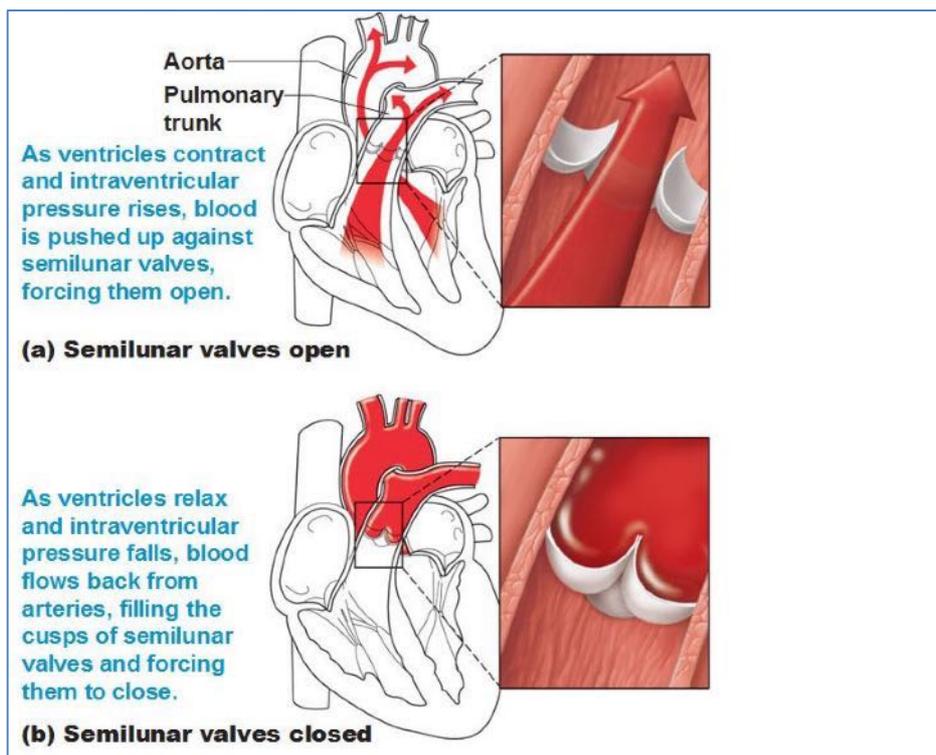
Source: antranik.org

o **b) Semilunar Valves Open:**

- Ventricular Pressure Exceeds Aortic/Pulm Pressure → Blood Ejected
 - ≈80mL of blood ejected each time (Stroke Volume)
 - Ventricular Volume Decreases.

o **c) Semilunar Valves Close:**

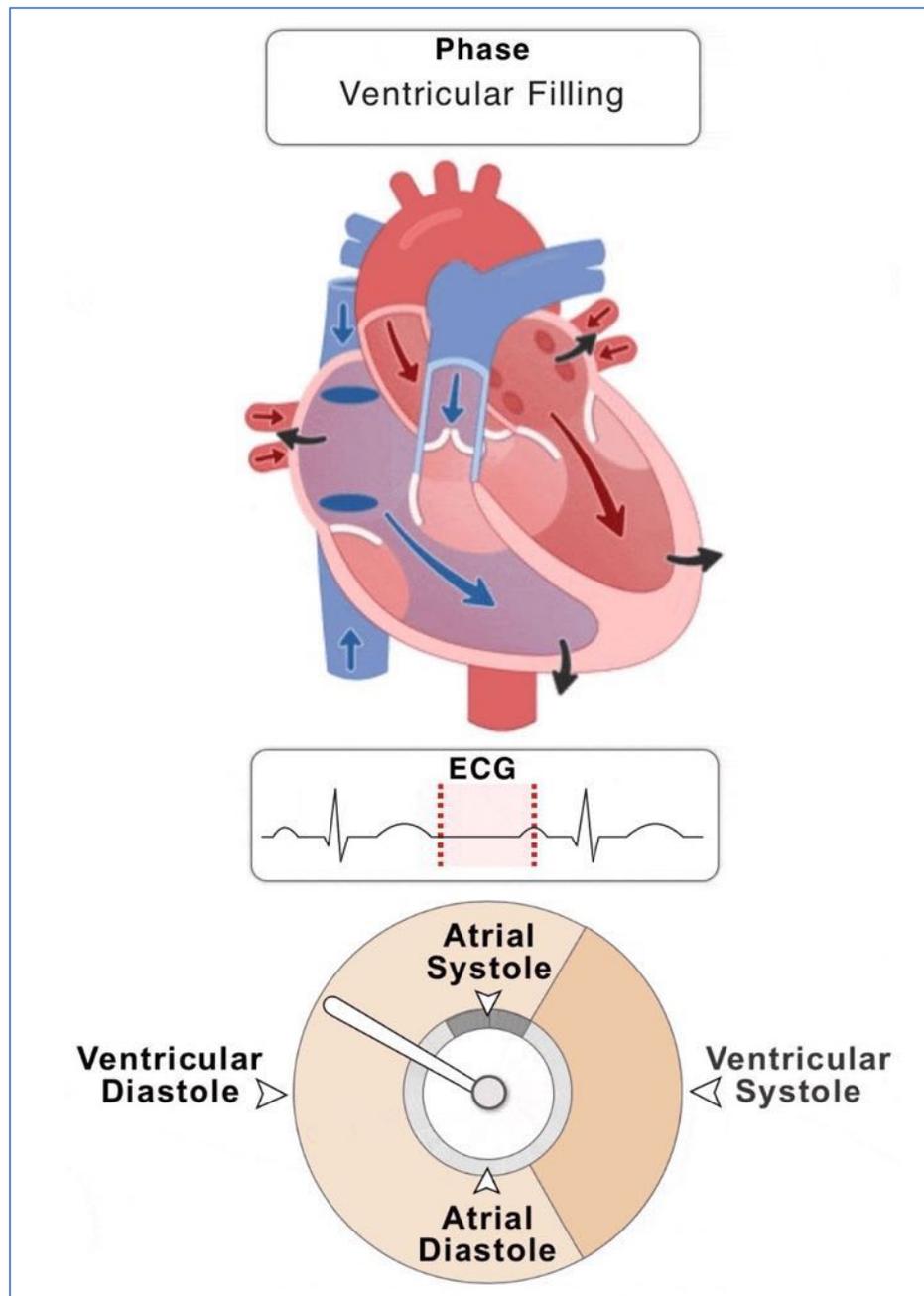
- Ventricular Pressure then falls Below Aortic/Pulm Pressure → Semilunar Valves Close.
 - Sudden closure of SemiLunar Valves causes the **Dicrotic Notch**:
 - o Result of Elasticity of the Aorta & Blood Rebounding off the Closed SL Valve.
 - o Causes a slight peak in *Aortic pressure*
- **Note:** Ventricles never *fully* empty:
 - ESV (End Systolic Volume) = Amount of blood left in ventricles → 50mLs.



Source: antranik.org

- **PHASE 3- Ventricular Diastole:**

- Ventricles relax → Ventricular Pressure falls below Atrial Pressure → AV-Valves Open:
 - Blood → from Atria into Ventricles
 - (NOTE: Passive filling from venous return is responsible for 70% of ventricular filling.)



Source: <https://www.humanbiomedia.org/cardiac-cycle-lesson/>

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