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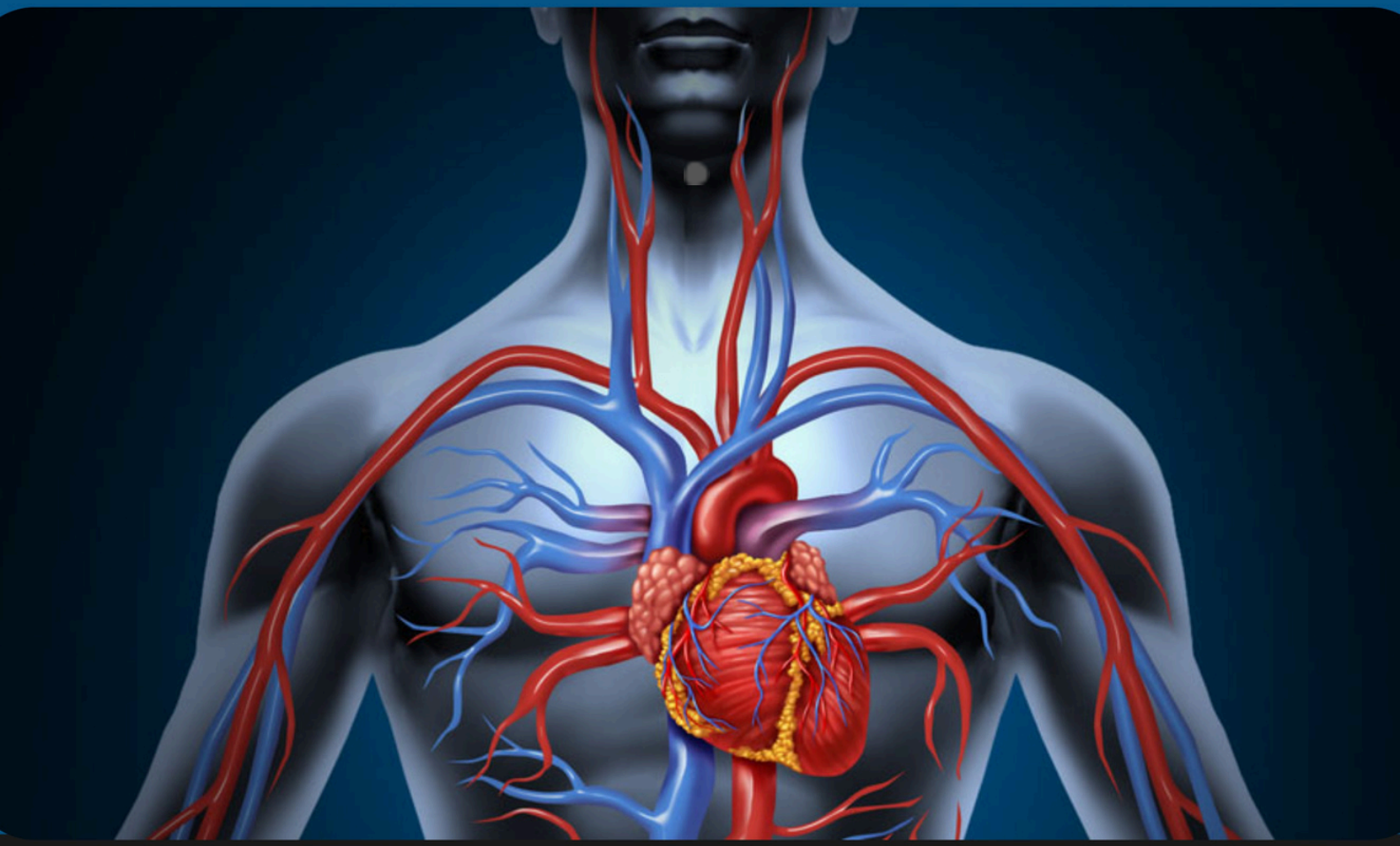
CARDIOVASCULAR SYSTEM

ANATOMY, PHYSIOLOGY & PATHOLOGY



TAILORED FOR MEDICAL STUDENTS, USMLE, PLAB, PA & NURSING

4th EDITION

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PDF

199 PAGES

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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:

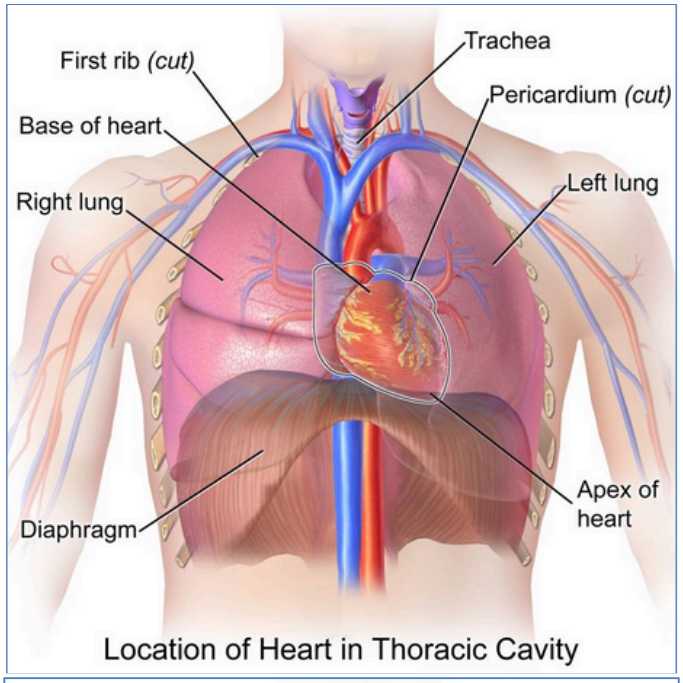
Pathology Notes:

ANATOMY OF THE HEART

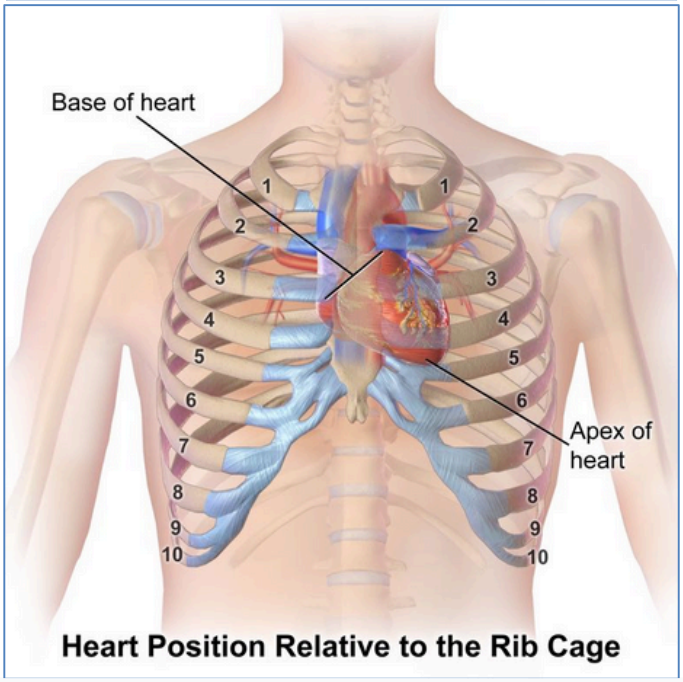
HEART ANATOMY:

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*.



Location of Heart in Thoracic Cavity



Heart Position Relative to the Rib Cage

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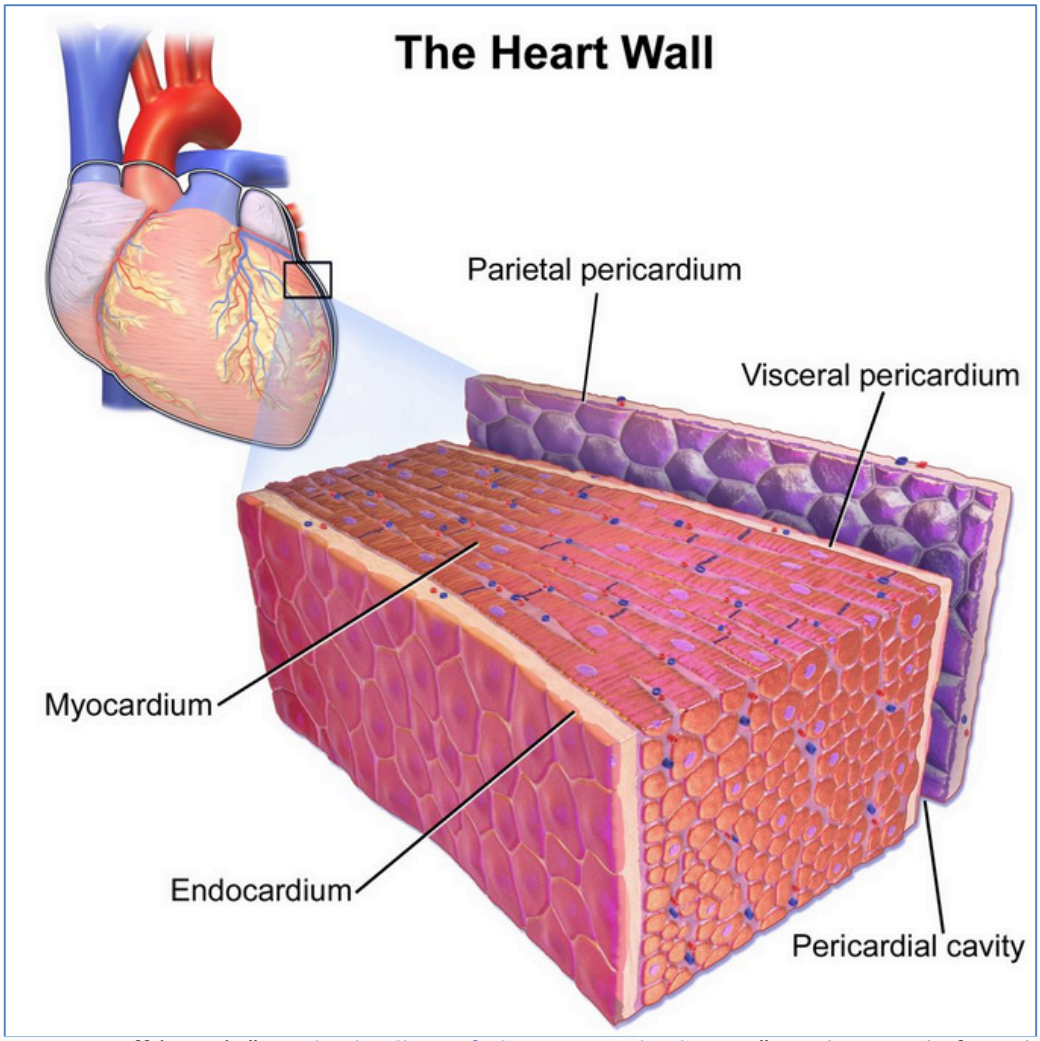
The Pericardium: (Coverings of the Heart)

- 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)



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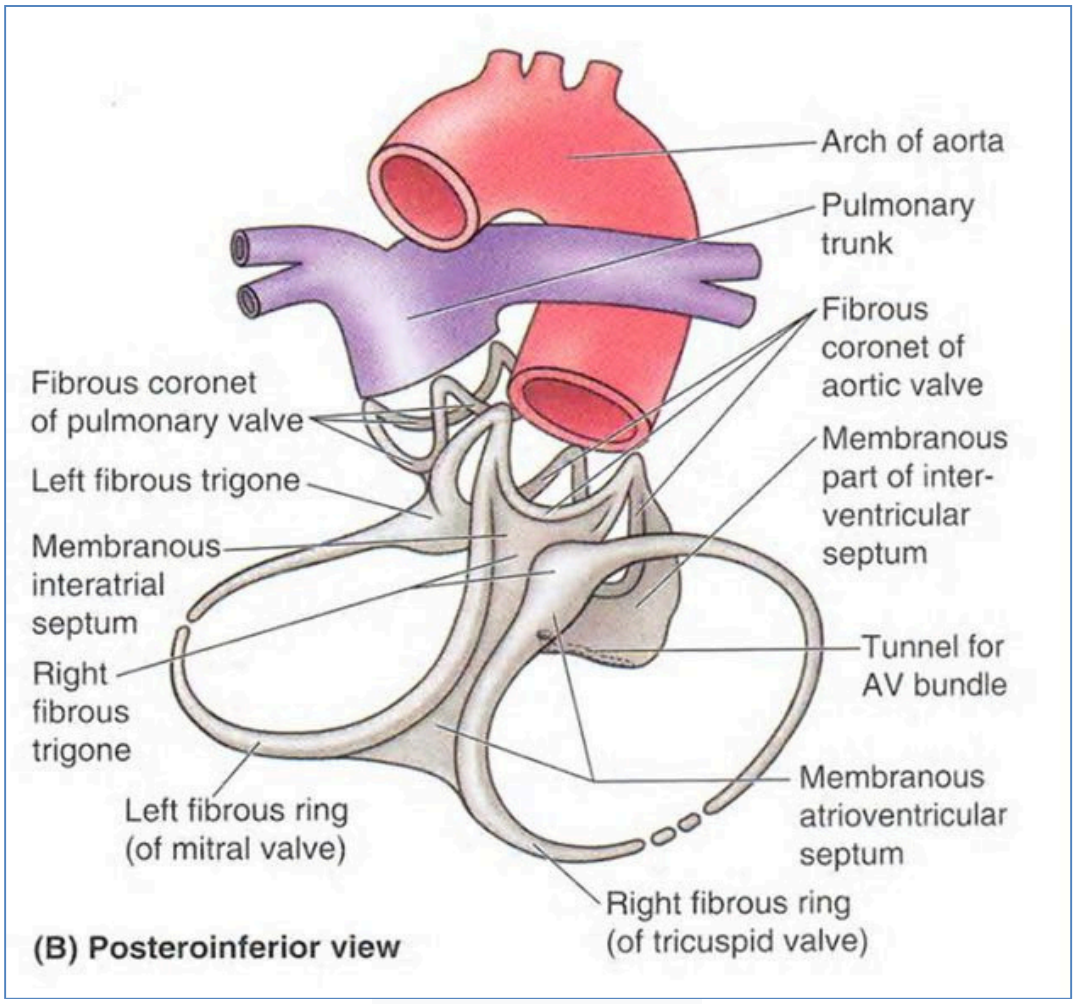
Fibrous Skeleton of the Heart:

- 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
    -



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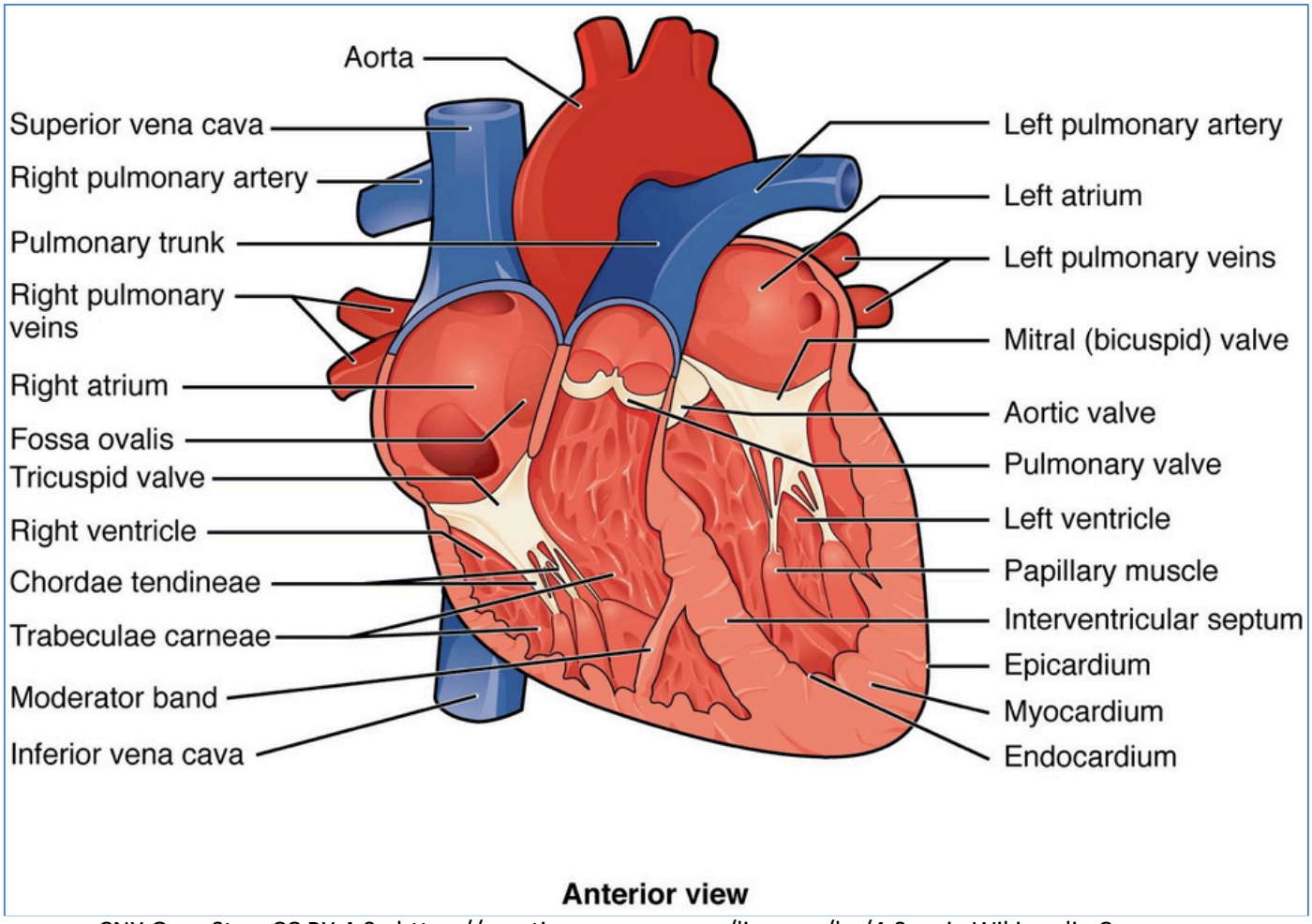
Chambers & Associated Great Vessels:

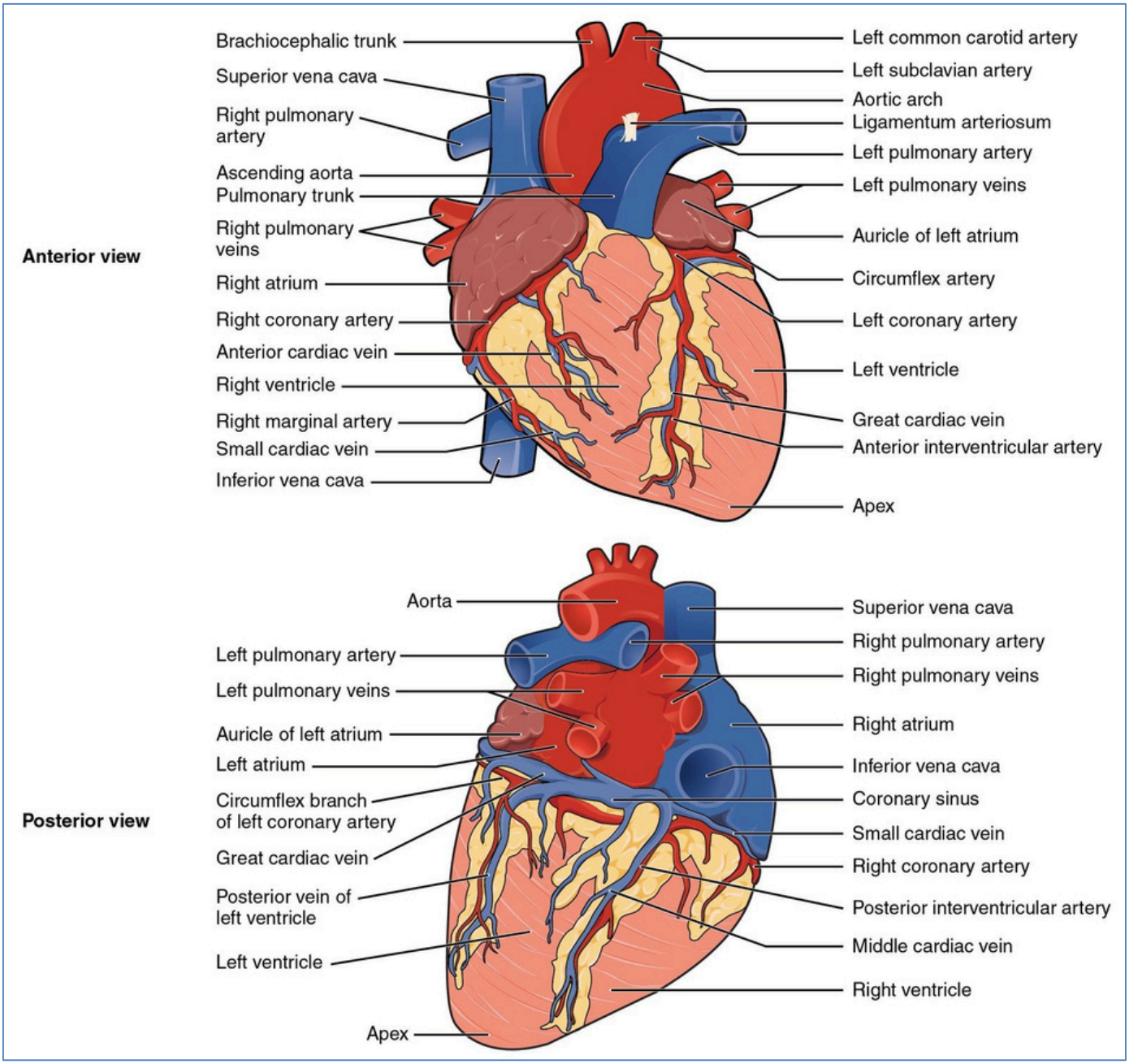
- **2 Atria (superior):** [Atrium = Entryway]
  - Thin-walled Receiving Chambers
  - On the superior aspect of heart (above the ventricles).
  - Each have a small, protruding appendage called **Auricles** – increase atrial volume.
  - Separated by Atrial Septum (Site of Foetal Shunt Foramen Ovale)
  - **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**



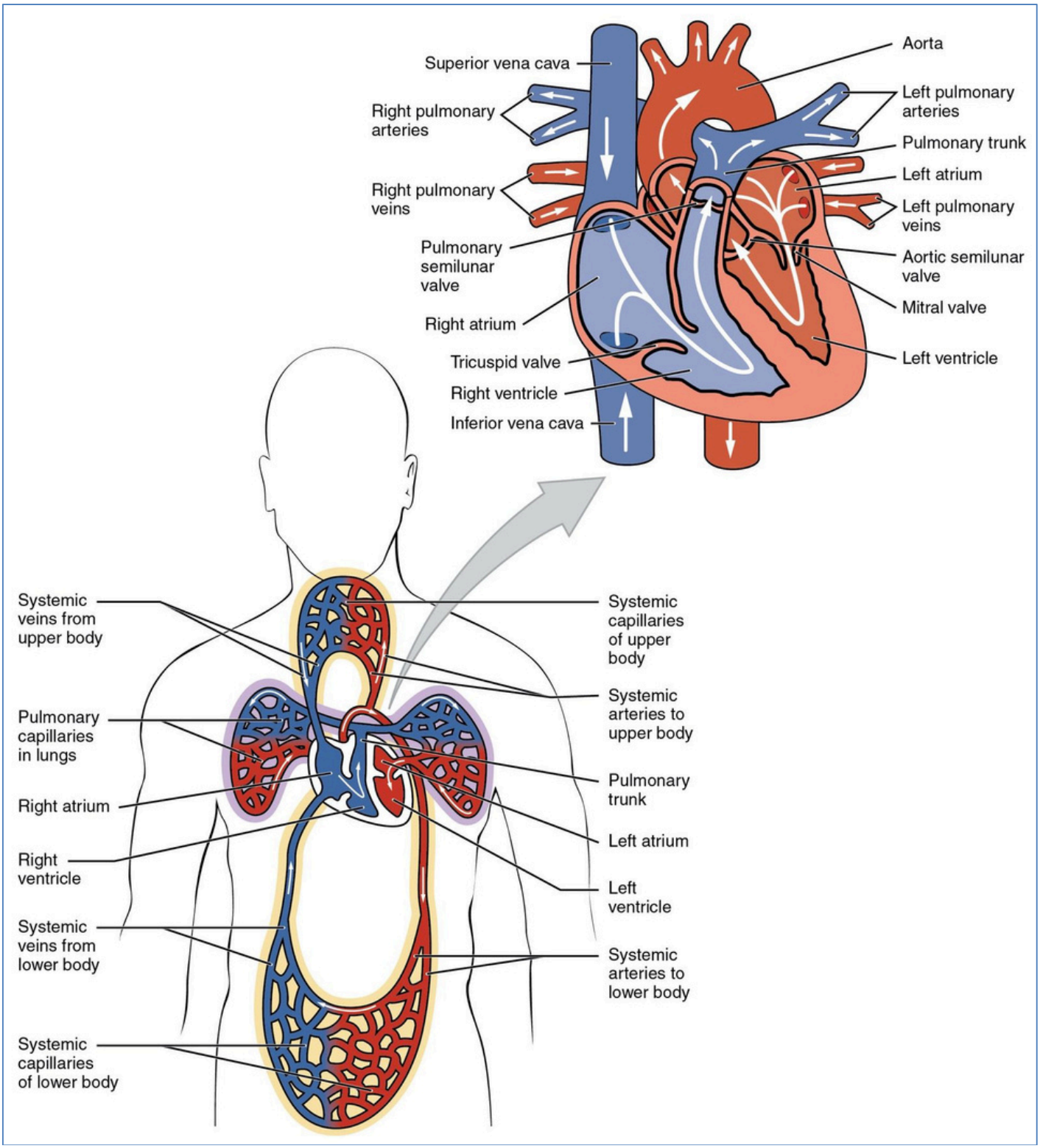
Landm arks of the Heart:

- **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:

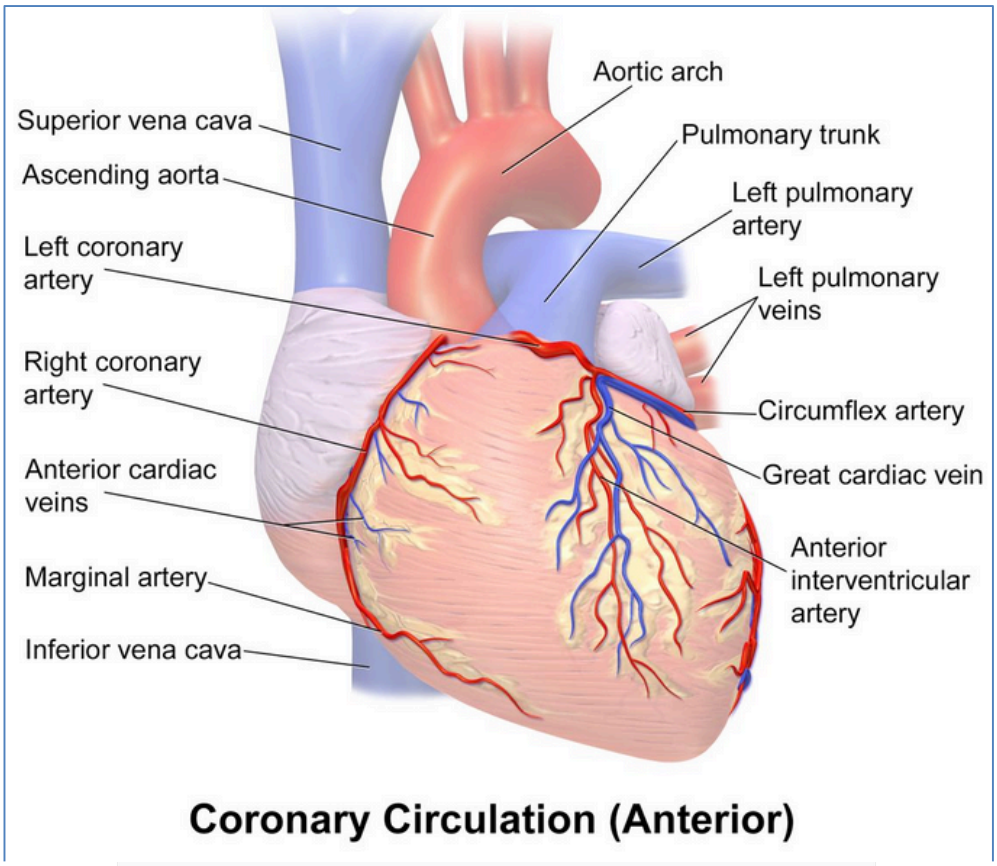
- **The right side** of the heart pumps blood through the pulmonary circuit (to the lungs and back to the left side of the heart).
    - Blood flowing through the pulmonary circuit gains oxygen and loses carbon dioxide, indicated by the colour change from blue to red.
- **The left side** of the heart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

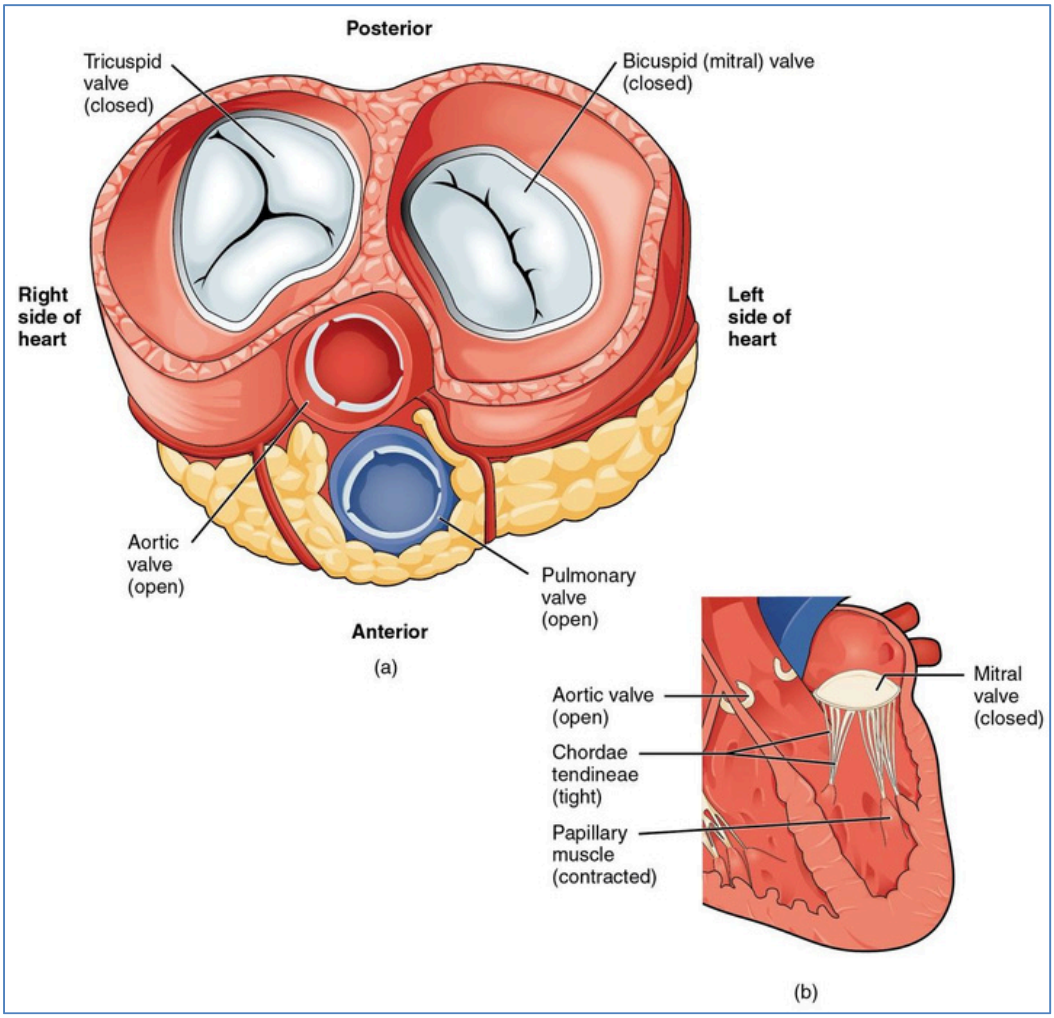
Heart Valves:

- Ensure *unidirectional flow of blood* through the heart.

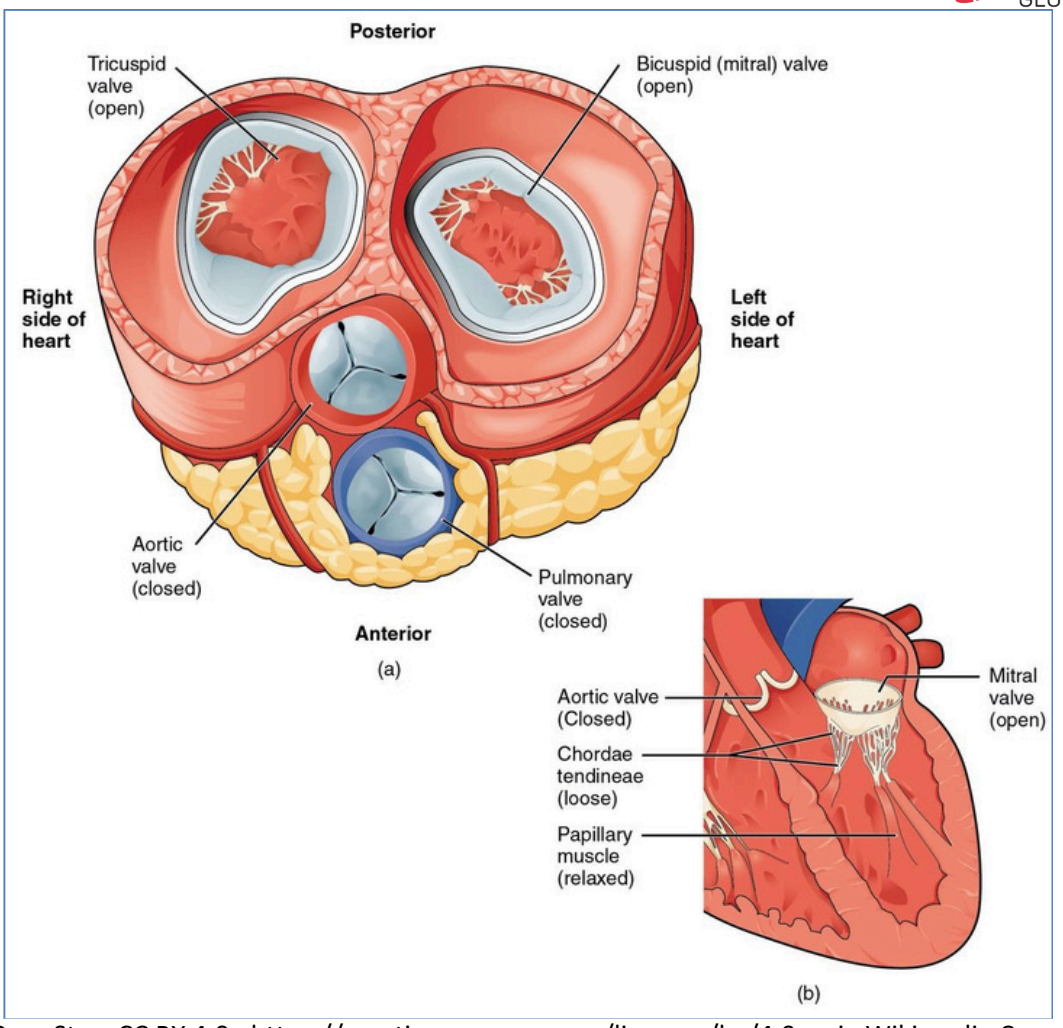
- **2x AtrioVentricular (AV) (Cuspid) Valves:**
  - **Location:**
    - At the 2 Atrial-Ventricular junctions
  - **Function:**
    - Prevent backflow into the Atria during Contraction of Ventricles
  - **Chordae tendinae** (tendinous cords) "heart strings" - Attached to each valve flap.
    - Anchor 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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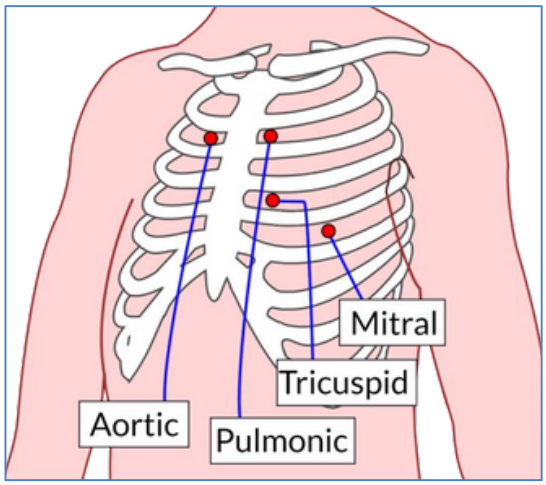
- **Valve Sounds:**
  - **1- "Lubb":**
    - Sound of AV Valve Closure
    - (M1 = Mitral Component)
    - (T1 = Tricuspid Component)
  - **2- "Dupp":**
    - Sound of Semilunar Valve Closure
    - (A2 = Aortic Component)
    - (P2 = Pulmonary Component)

- **W here to Listen:**



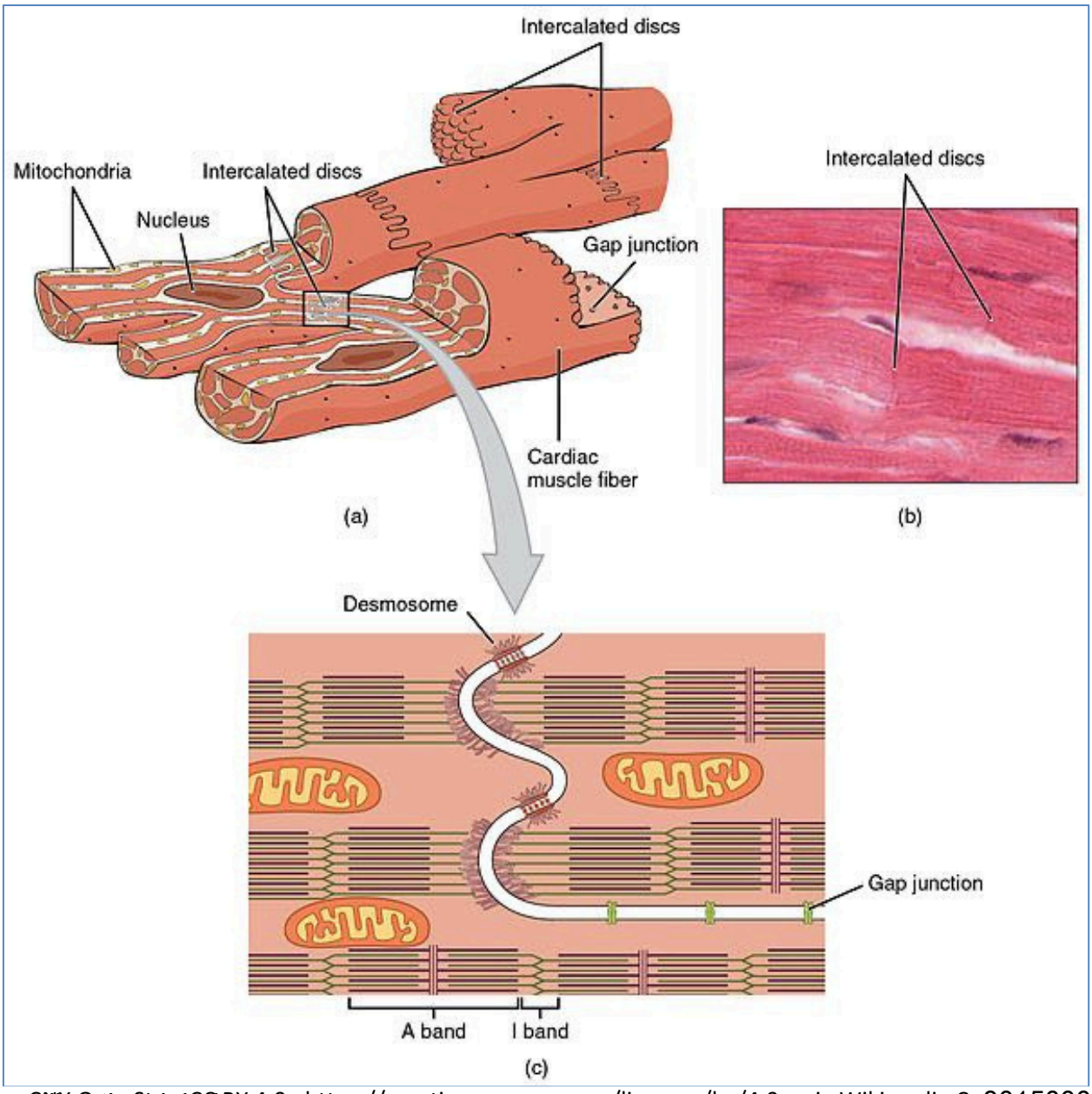
Adaptation of File:Precordial Leads 2.svg (by Jmarchn) and Rib_Cage (Jeroen Hut)

ELECTROPHYSIOLOGY OF THE HEART:

The Heartbeat:

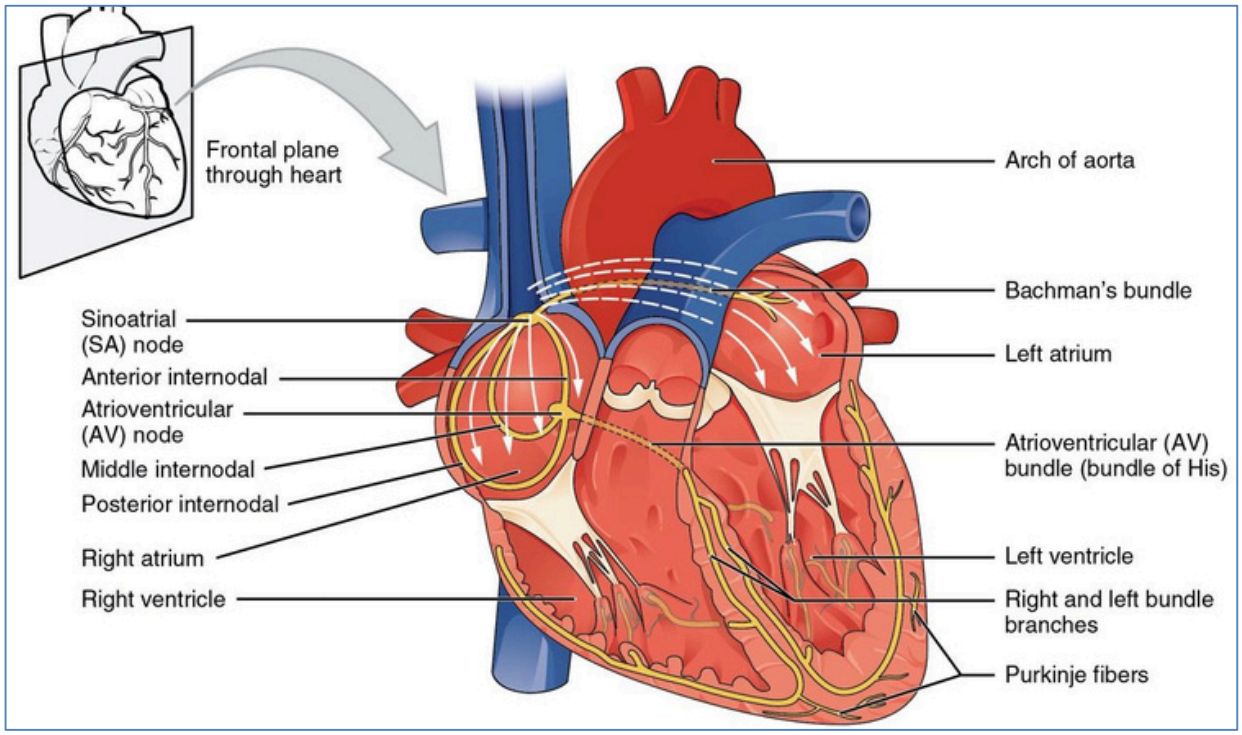
- **Heart is a Muscle & Requires:**
  - O2
  - Nutrients, &
  - **Action Potentials**; to function.
- **However,** these neural signals don't come from the brain;
  - Rather, the heart has its ***own*** conduction systems.
    - These systems ***allow it to contract autonomously***
  - Hence why a *transplanted heart* still operates (if provided with O2 & nutrients)
- **Cardiac Activity is Coordinated:**
  - To be effective, the Atria & Ventricles must contract in a ***coordinated manner***.
  - This activity is coordinated by the Heart's Conduction Systems.....

- **The Entire Heart is Electrically Connected**...**By:**
  - Gap Junctions:
    - Allows action potentials to move from cell to cell
  - 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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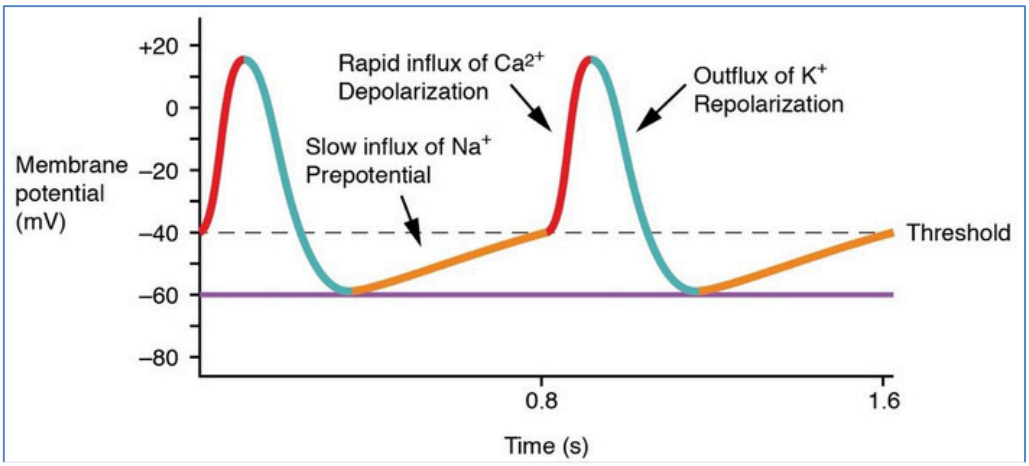
Conductile Cardiac Cell Physiology (SA/AV Node Cells):

- **Action Potentials: Slow 'Pacemaker' Type**
- **Have UNSTABLE Resting Membrane Potentials → *Spontaneous Electrical Activity:***
  - **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
  - **Depolarisation:**
    - Once Threshold is reached, Ca2+ channels open
    - → Influx of Ca+
    - → Causes an action potential.
  - **Repolarisation:**
    - Once peak MP is reached, Ca+ channels close, K+ channels open
    - → K+ Efflux makes MP more –ve
    - → Causes repolarisation
  - (Na+ brings to threshold, but Ca+ is responsible for Depolarisation.)
- **With a *Hierarchy* of control over the heart**.
  - Hierarchy based on *natural intrinsic rate*. (fastest node (SA node) takes control)



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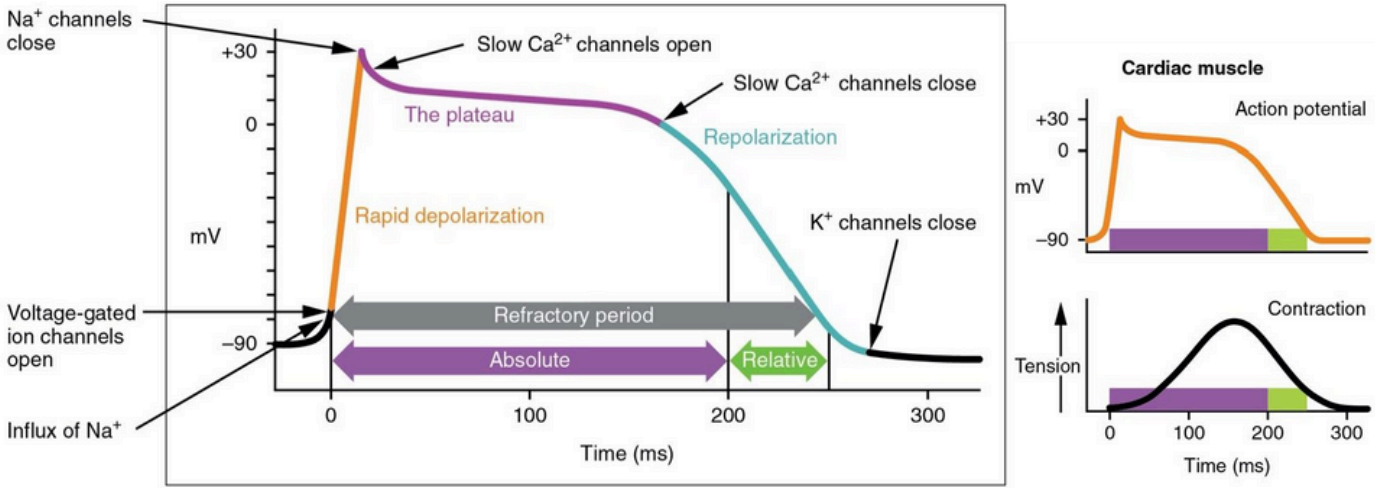
Contractile Cardiac Cell Physiology (Purkinje Fibres & Myocytes):

- **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 → +ve feedback → Threshold
  - o **Depolarisation:**
    - § If MP reaches threshold, all Fast Na+ channels open;
    - § → Massive influx of Na+ into cell
    - § → Membrane depolarises
  - o **Plateau:**
    - § Fast Na+ channels inactivate.
    - § → The small downward deflection is due to Efflux of K+ ions
    - § → 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;
      - → Result is a net *outward* flow of +ve charge. → Downward Deflection
      - → As the MP falls, more K+ channels open, accelerating depolarization.
      - → 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:

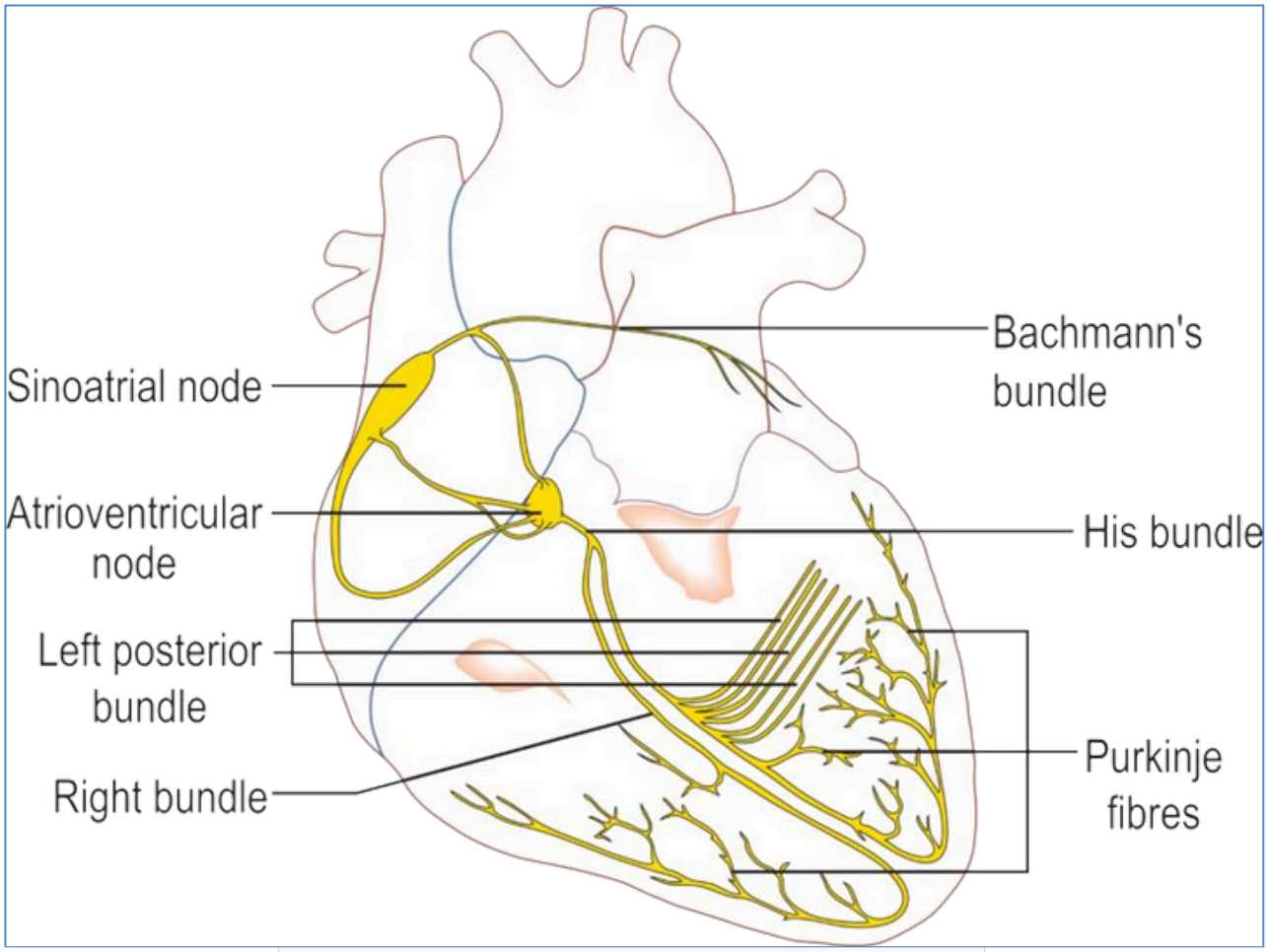
- In Cardiac Muscle, the *Absolute Refractory Period* continues until muscle relaxation; o Therefore summation isn't possible → tetany cannot occur (critical in heart) o Ie: The depolarised cell won't respond to a 2nd stimulus until contraction is finished.
- **Absolute Refractory Period:**
  - o Approx 200ms
  - o Duration: from peak → plateau → 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;
  - Parasympathetic NS lowers heart rate → Keeps Normal Resting HR at 70bpm
  - Sympathetic NS raises heart rate.
- **Location:**
  - *Posterior Wall* of the *Right Atrium* near the opening of the *Superior Vena Cava*
- **Nature of Action Potentials:**
  - Continually Depolarizing 90-100bpm
  - Takes 50ms for Action-Potential to reach the AV Node.
- **Role in Conduction Network:**
  - Sets the pace for the heart as a whole.
- **Portion of Myocardium Served:**
  - Contracts the Right & Left Atrium

The AtrioVentricular (AV) Node:

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

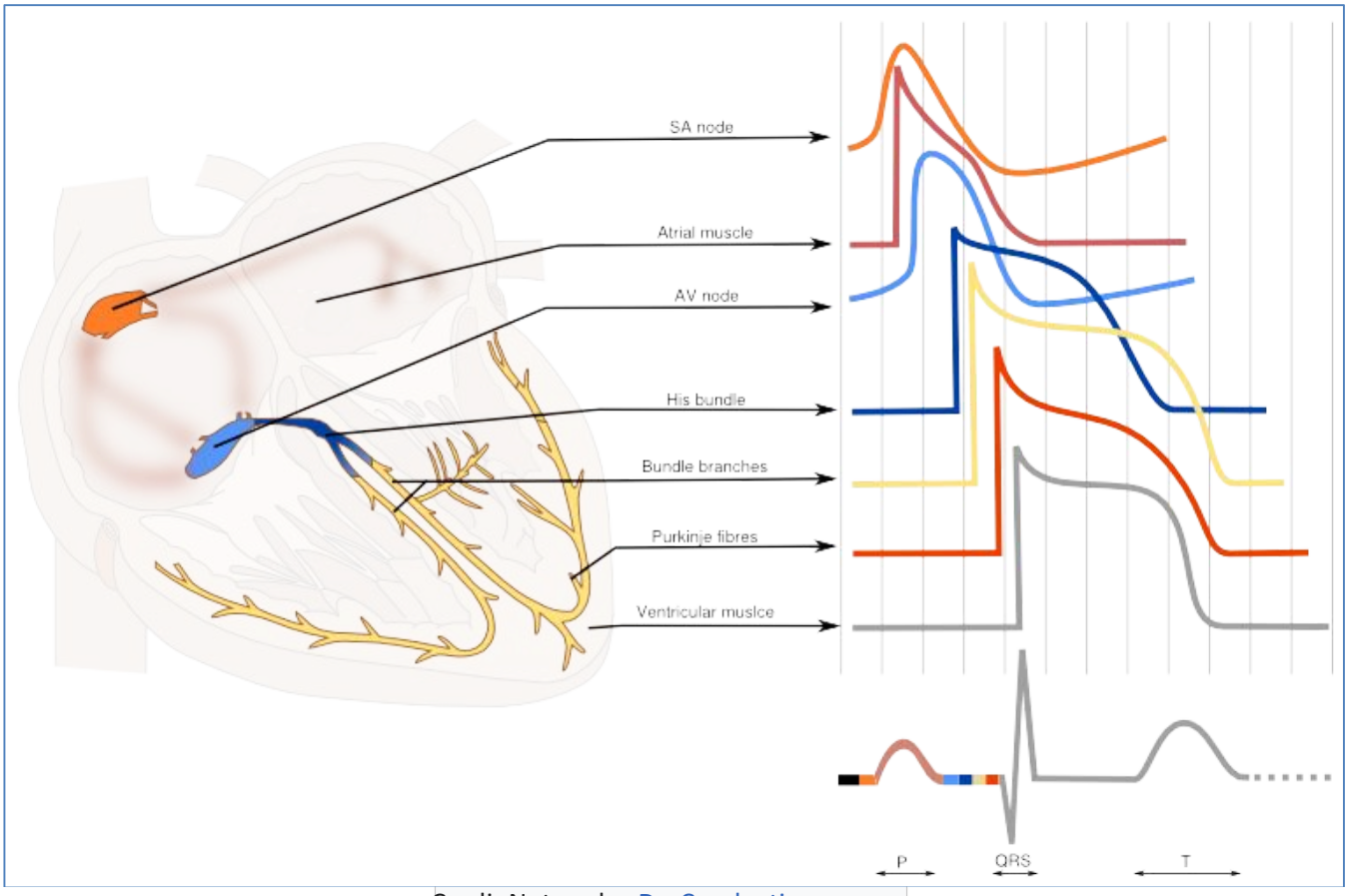


The Bundle Branches (Bundles of His):

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

The Purkinje Fibres:

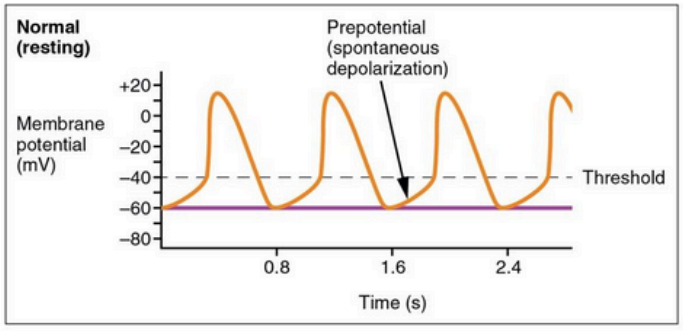
- **Specialised Myocytes with *very few myofibrils*** → don't contract during impulse transmission.
- **Location:**
  - The Inner Ventricular Walls of the Heart – just below the Endocardium
  - Begin at the heart apex, then turn superiorly into the Ventricular Walls.
- **Nature of Action Potentials:**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

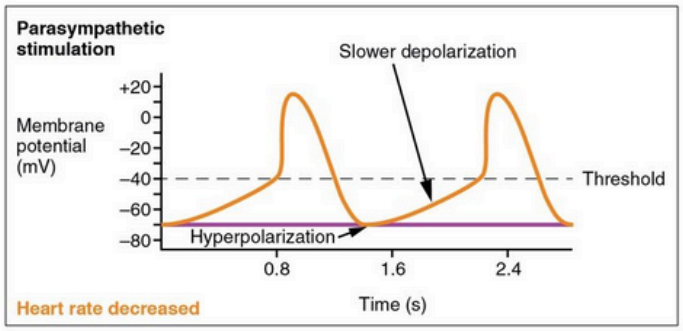


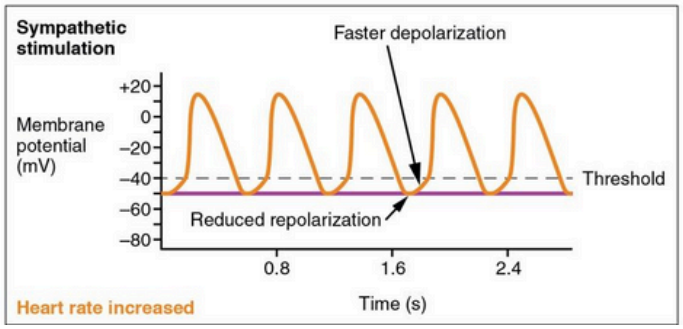
CardioNetworks: De-Conduction_ap.png

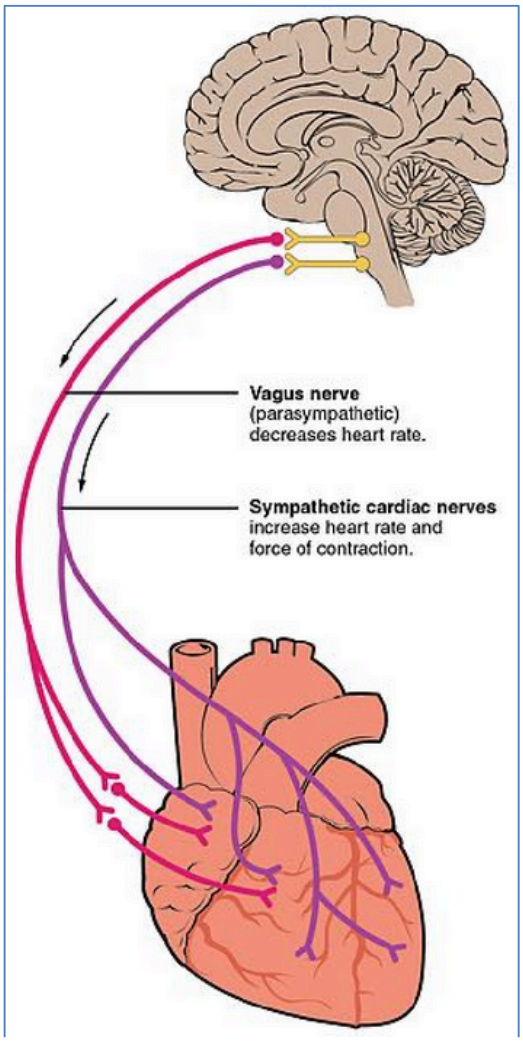
Effects of the Autonomic Nervous System (ANS):

- 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 & M uscles →
    - § 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







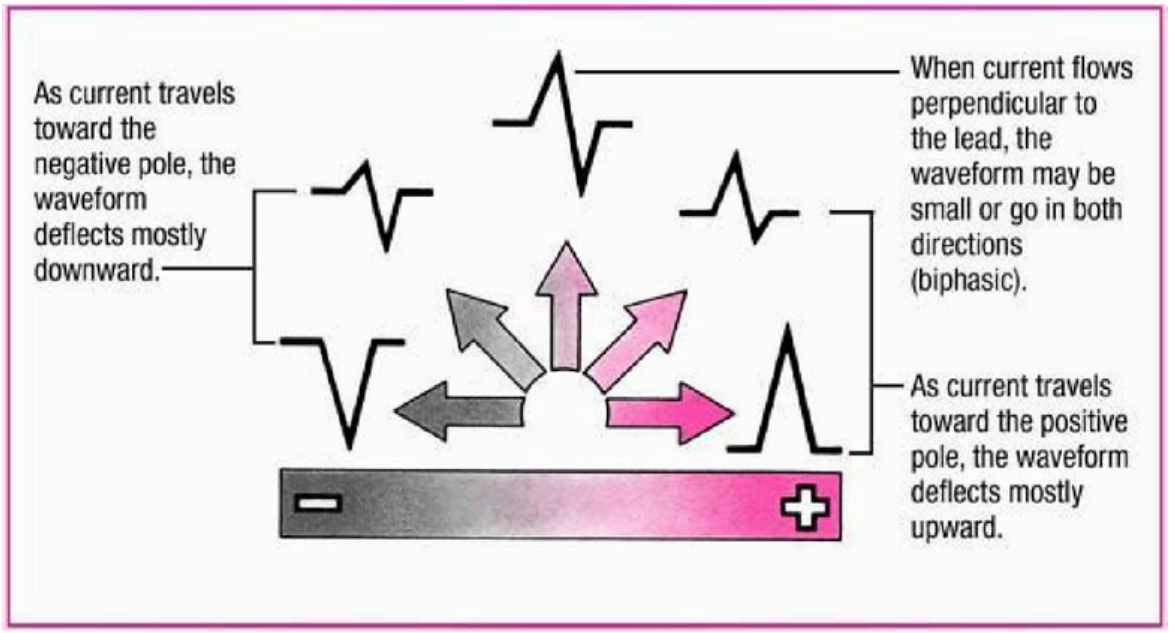


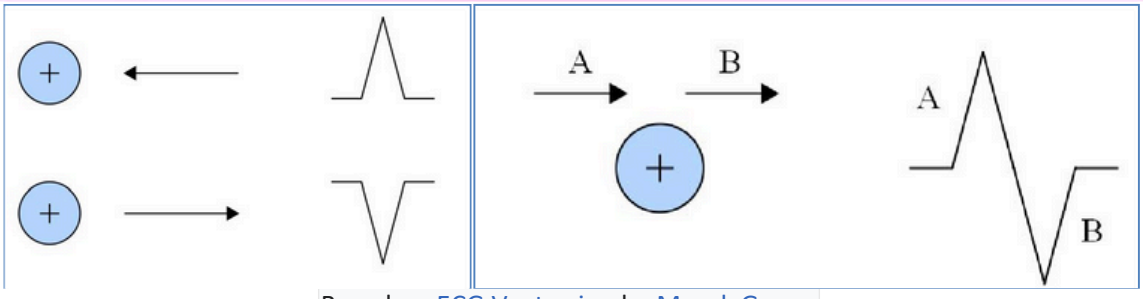
ELECTROCARDIOGRAM (ECG) PHYSIOLOGY:

ELECTROCARDIOGRAM (ECG) PHYSIOLOGY:

What Is An ECG?

- **A Recording of all Action Potentials by Nodal & Contractile Cells in the heart at a given time**.
  - NOTE: It IS NOT a single action potential.
  - 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:**
  - **3x Bipolar Leads**: Measure Voltages between the Arms...OR...Between an Arm & a LEg:
    - I = LA (+) RA (-)
    - II = LL (+) RA (-)
    - III = LL (+) LA (-)
  - **9x Unipolar Leads:**
    - Look at the heart in a '3D' Image.
  - (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:**
  - X-axis = Time
  - Y-axis = Amplitude (voltage) – Proportional to number & size of cells.
- **Understanding Waveforms:**
  - When a Depolarisation Wavefront moves toward a positive electrode, a *Positive* deflection results in the corresponding lead.
  - When a Depolarisation Wavefront moves away from a positive electrode, a *Negative* deflection results in the corresponding lead.
  - 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:

Get Direction GLOBAL

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:

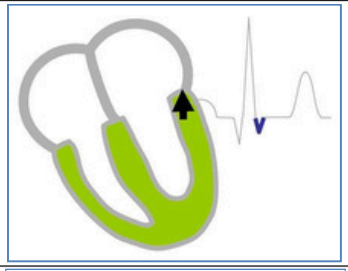
- Interventricular Septum Depolarization
- Wave direction (see blue arrow) is perpendicular to the Main Electrical Axis → results in a 'Biphasic' trace.
    - o Only the –ve deflection is seen due to signal cancellation by Atrial Repolarization.
    - o Sometimes this wave isn't seen at all

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:

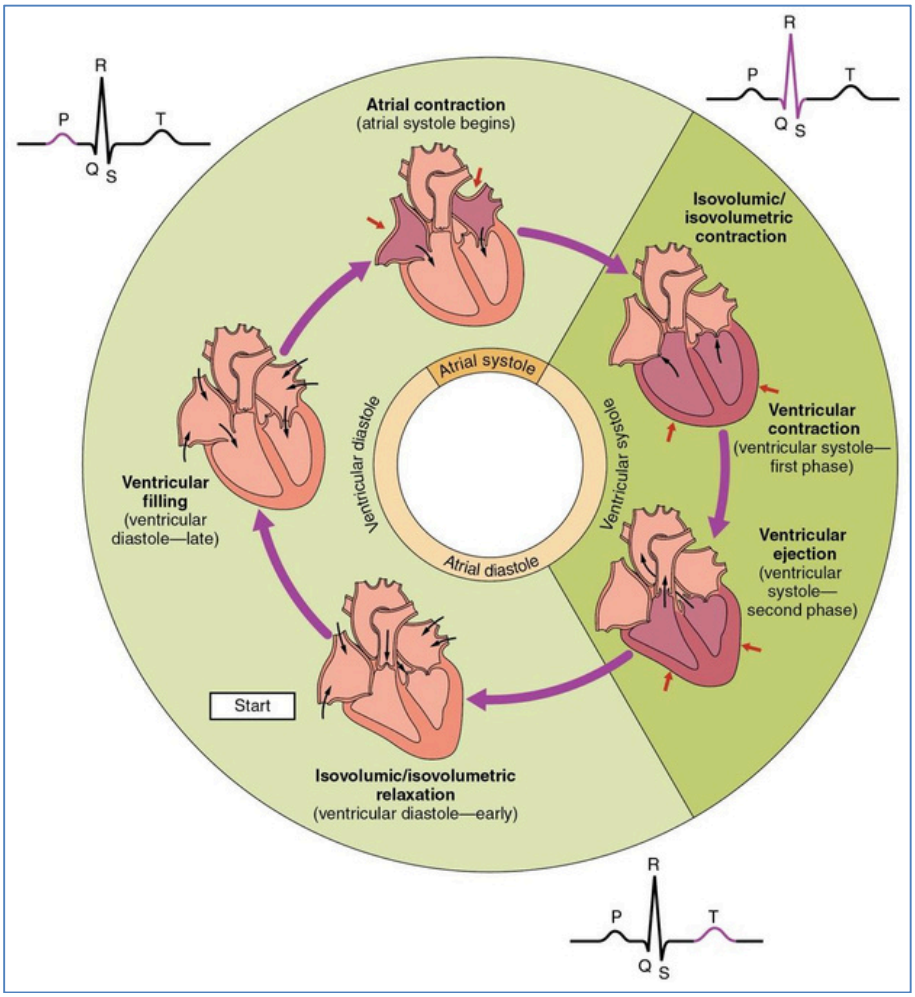
- Ventricular Contraction is occurring at this time.
    - o Due to the lag between excitation & contraction.

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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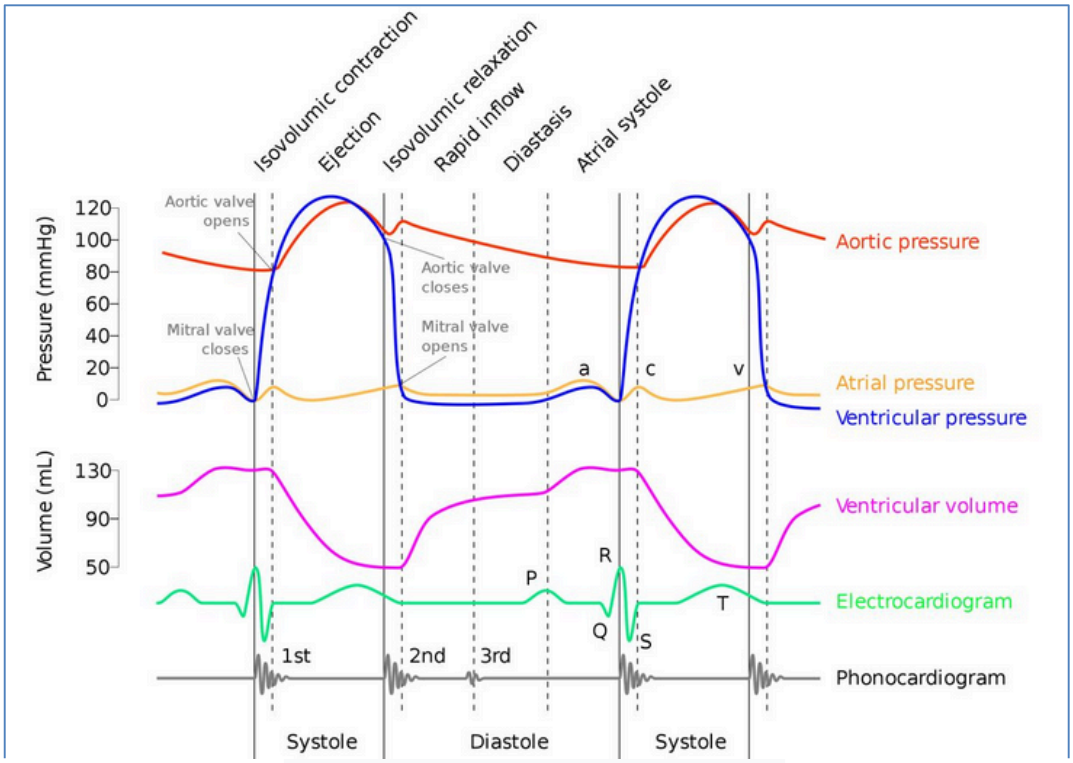

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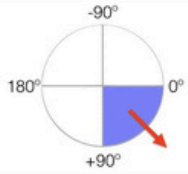
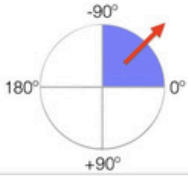
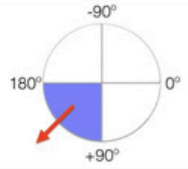
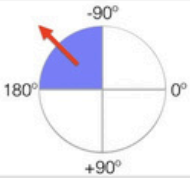
The Heart's Electrical Axis:

- 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 1	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 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:

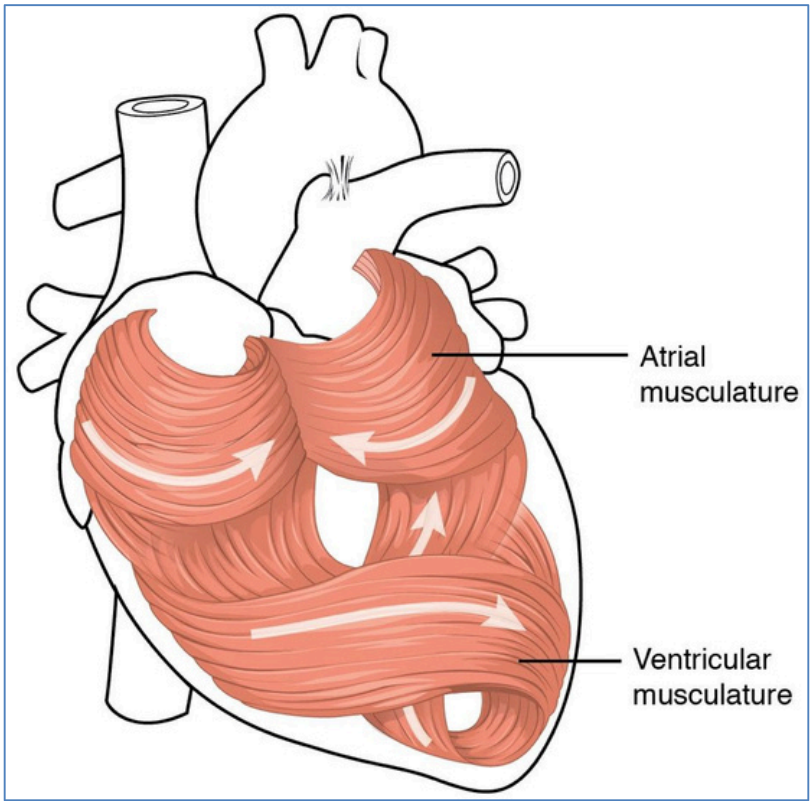
- **Check Pt ID**
- **Check Voltage & timing**
  - 25mm/sec
  - 1large square = 0.2s (1/5sec)
  - 1small square = 0.04s
- **What is the rate?**
  - 300/number of large squares between QRS Complexes
    - Tachycardia
    - >100bpm

    Bradycardia
    - <60bpm
- **What is the Rhythm?**
  - Sinus? (are there P-Waves before each QRS complex)
  - If Not Sinus?
    - Is it regular
    - Irregular?
    - Irregularly Irregular (AF)
    - Brady/Tachy
- **Atrial Fibrillation:**
  - Irregularly Irregular
  - P-Waves @ 300/min
- **QRS:**
  - Is there one QRS for each Pwave?
  - Long PR Interval? (1st degree heart block)
  - Missed Beats? (Second degree block)
  - No relationship? Complete heart block
- **Look for QRS Complexes:**
  - How wide – should be < 3 squares
  - If wide – It is most likely Ventricular
  - (Sometimes atrial with aberrant conduction (LBBB/RBBB)
  - IF Tachycardia, & Wide Complex → VT is most likely. (If hypotensive → Shock; if Normotensive → IV Drugs)
- **Look for TWaves:**
  - Upright or Inverted
- **Look at ST-Segment**
  - Raised, depressed or inverted
  - 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)
  - NOTE: Normal ECG Doesn't exclude infarct.
  - ST Depression → Ischaemia
  - ST Elevation → Infarction
  - If LBBB or Paced, you CANNOT comment on ST-Segment

MECHANICAL EVENTS OF THE CARDIAC CYCLE

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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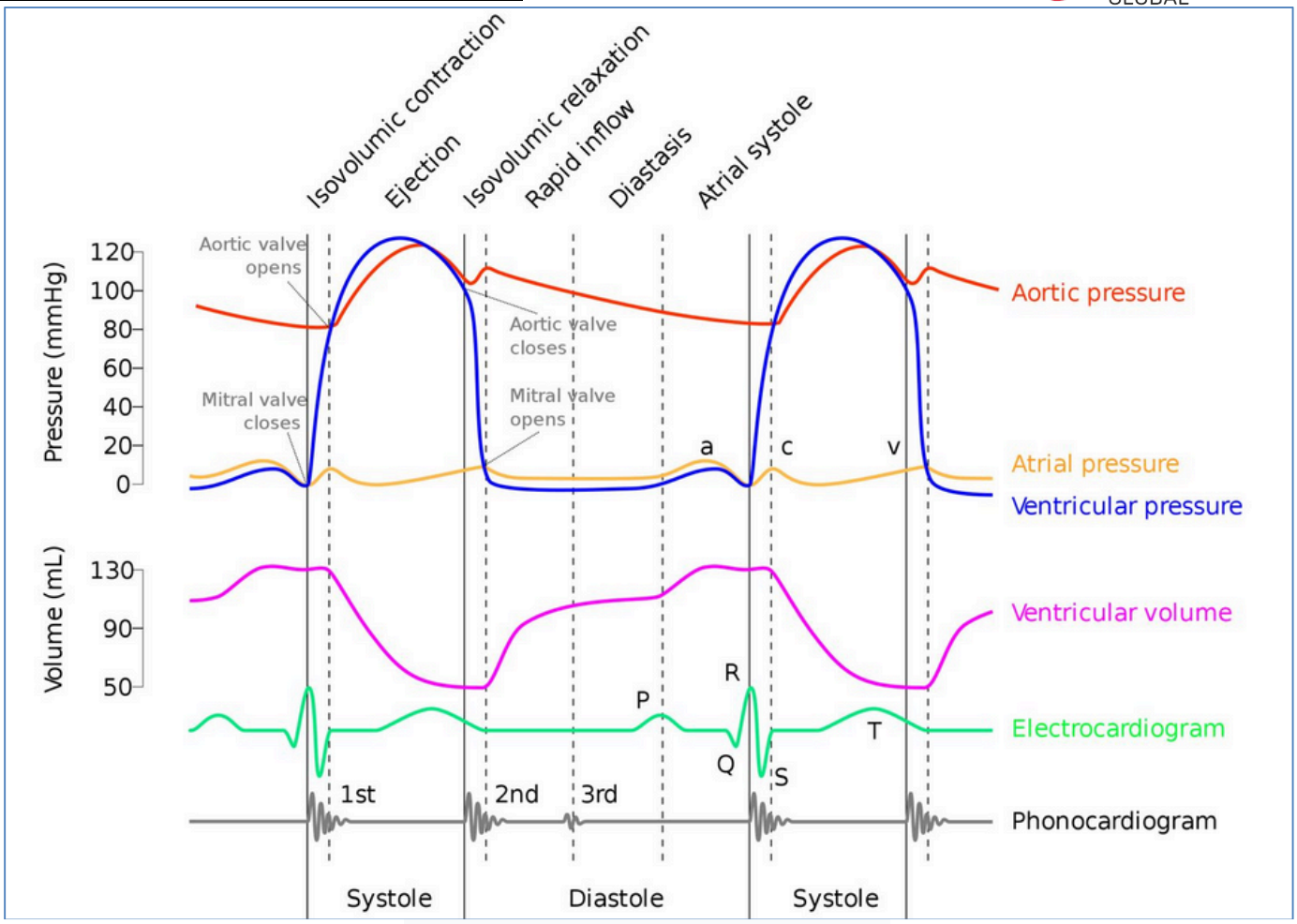
Terms:

- **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:



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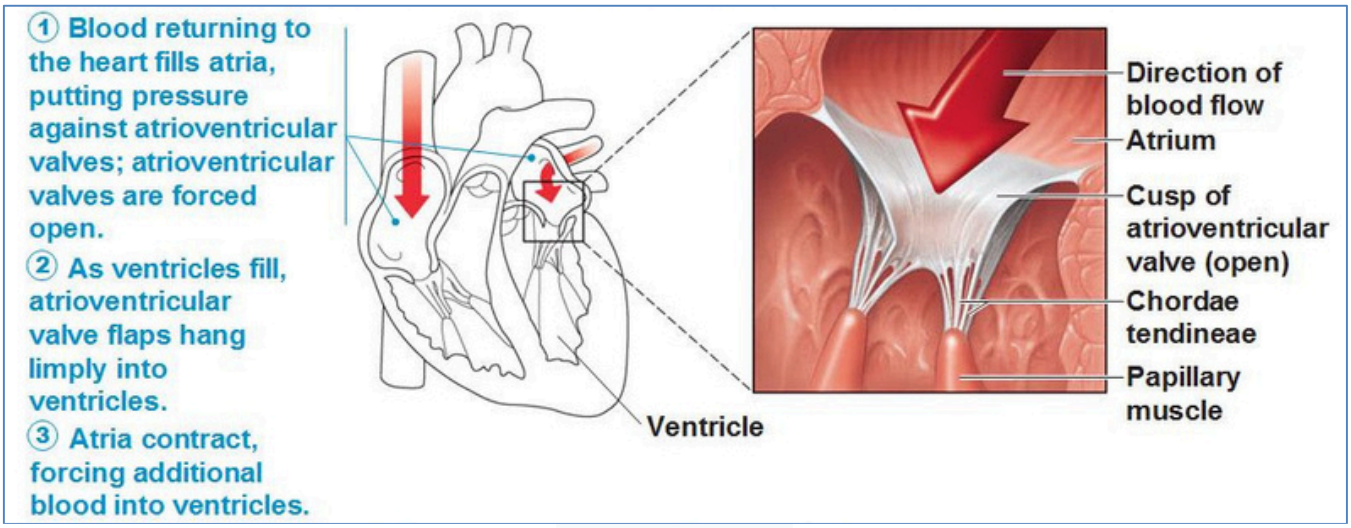
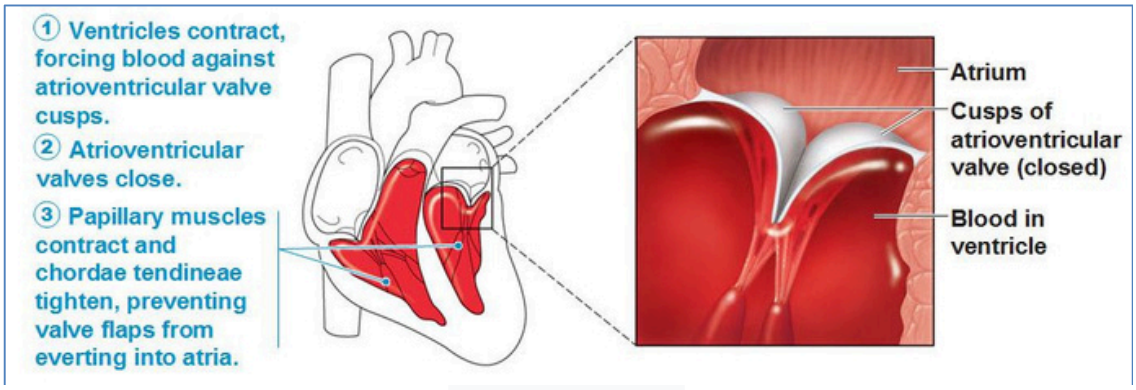
- **PHASE 1- Atrial Contraction (Systole) + Ventricular Filling (Diastole):**
  - **Contraction of Atria**
    - → IntraAtrial Pressure Increases
    - → Blood pushed into Ventricles through AV-Valves
  - **Note:** Ventricles are already 70% full from passive Venous Filling.
  - At End of Atrial Systole, Ventricles have EDV (End Diastolic Volume) ≈ 130mL



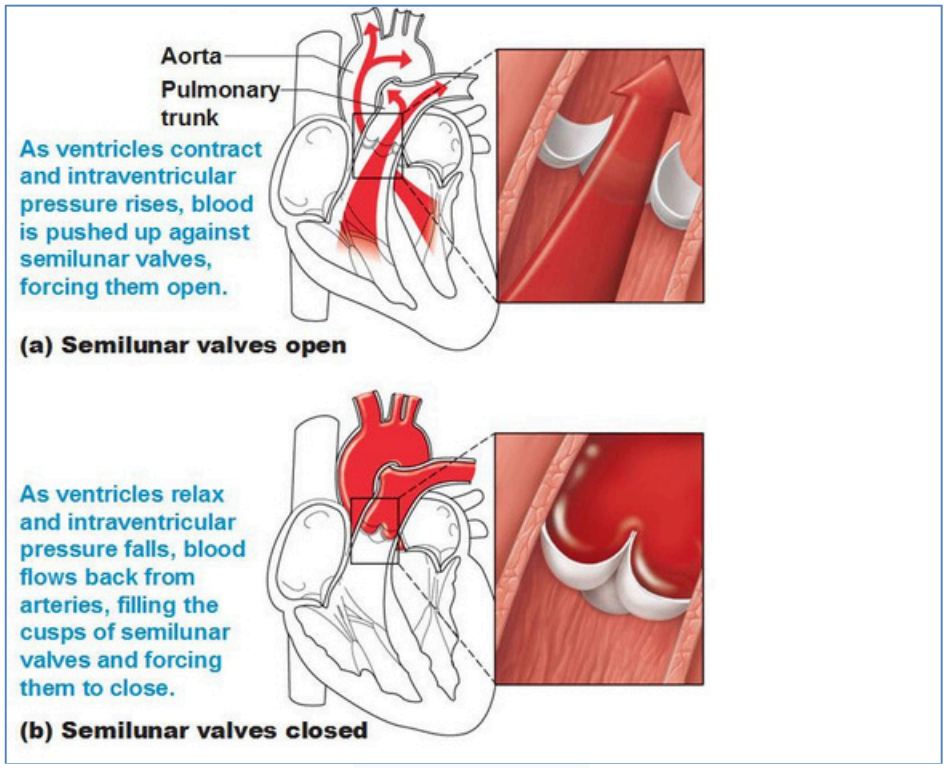
Source: antranik.org

- **PHASE 2- Ventricular Systole:**
  - **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.*



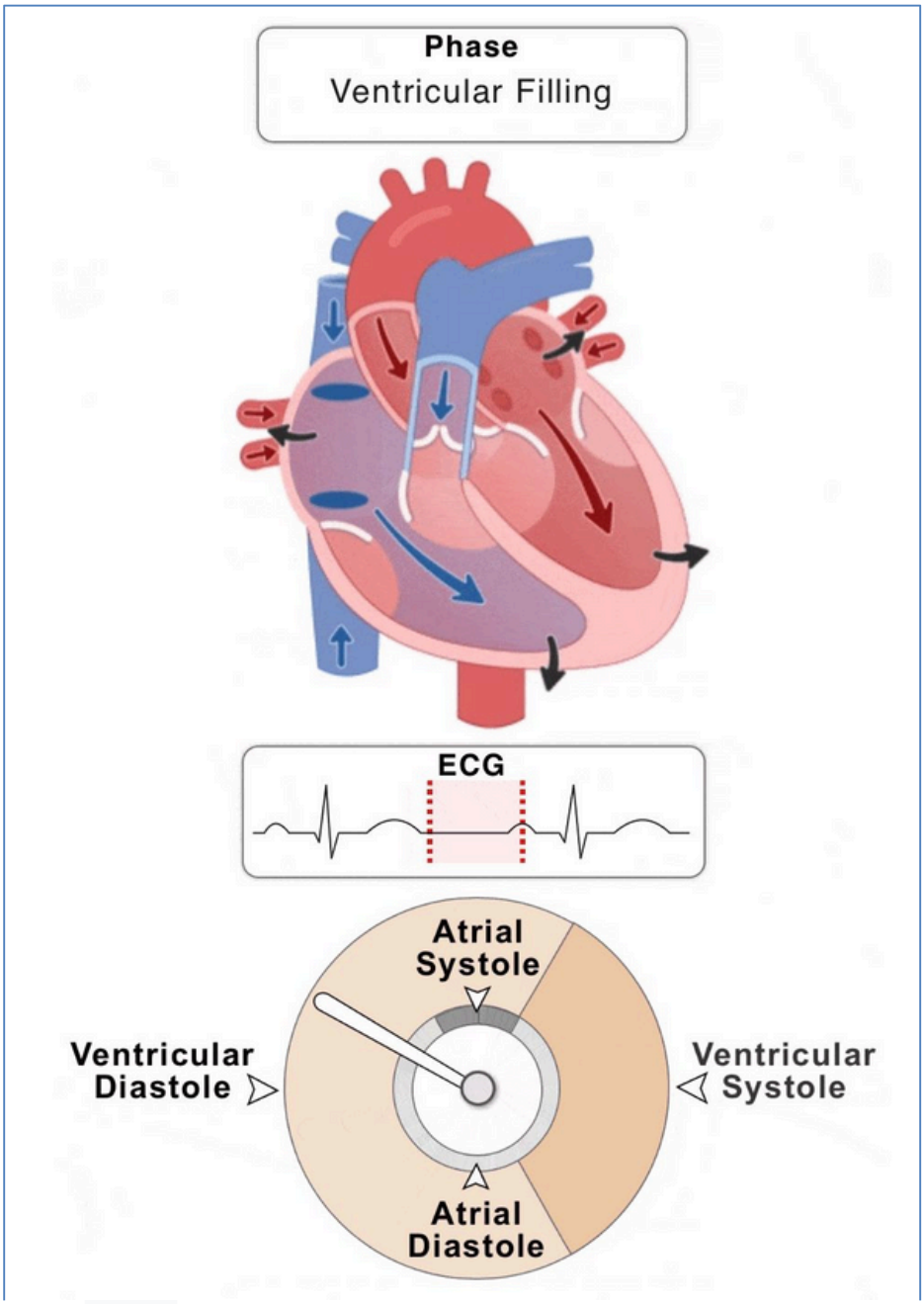
Source: antranik.org

  - **b) Semilunar Valves Open:**
    - § Ventricular Pressure Exceeds Aortic/Pulm Pressure → Blood Ejected
      - ≈80mL of blood ejected each time (Stroke Volume)
      - Ventricular Volume Decreases.
  - **c) Semilunar Valves Close:**
    - § Ventricular Pressure then falls Below Aortic/Pulm Pressure → Semilunar Valves Close.
      - Sudden closure of SemiLunar Valves causes the ***Dicrotic Notch***:
        - Result of Elasticity of the Aorta & Blood Rebounding off the Closed SL Valve.
        - 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:**
  - o 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/

CARDIO-DYNAMICS:

CARDIO-DYNAMICS:

Cardiac Output:

- Useful when examining cardiac function *over time*.
- **Determined by 2 Things:**
  - 1- Stroke Volume....&
  - 2- Heart Rate

Cardiac Output(mL/min) = Stroke Volume X Heart Rate

- **Average CO ≈ 5L/min** (Ie: The entire blood supply circulates once per minute)
- **Cardiac Output** Is regulated such that peripheral tissues receive adequate blood supply.

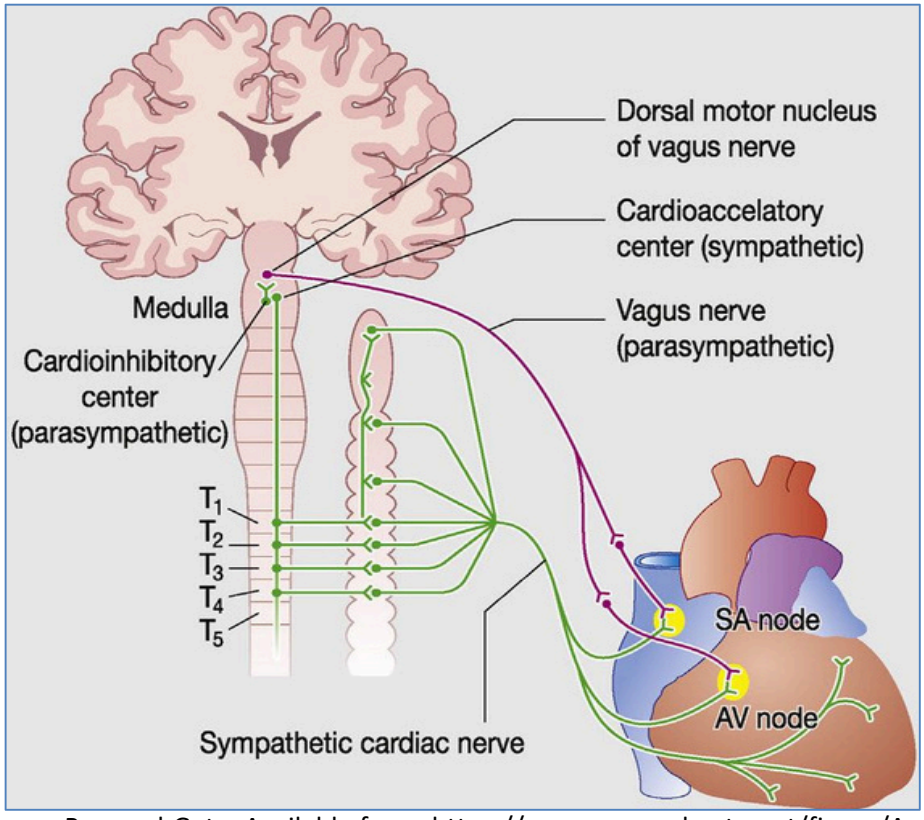
HEART RATE:

- Depends on Tissue-Satisfaction with Nutrients & O2.
- **Terms:**
  - **BradyCardia:** HR *Slower* than normal. (too fast → stroke volume & CO suffers)
  - **TachyCardia:** HR *Faster* than normal.

5 Things That Affect Heart Rate:

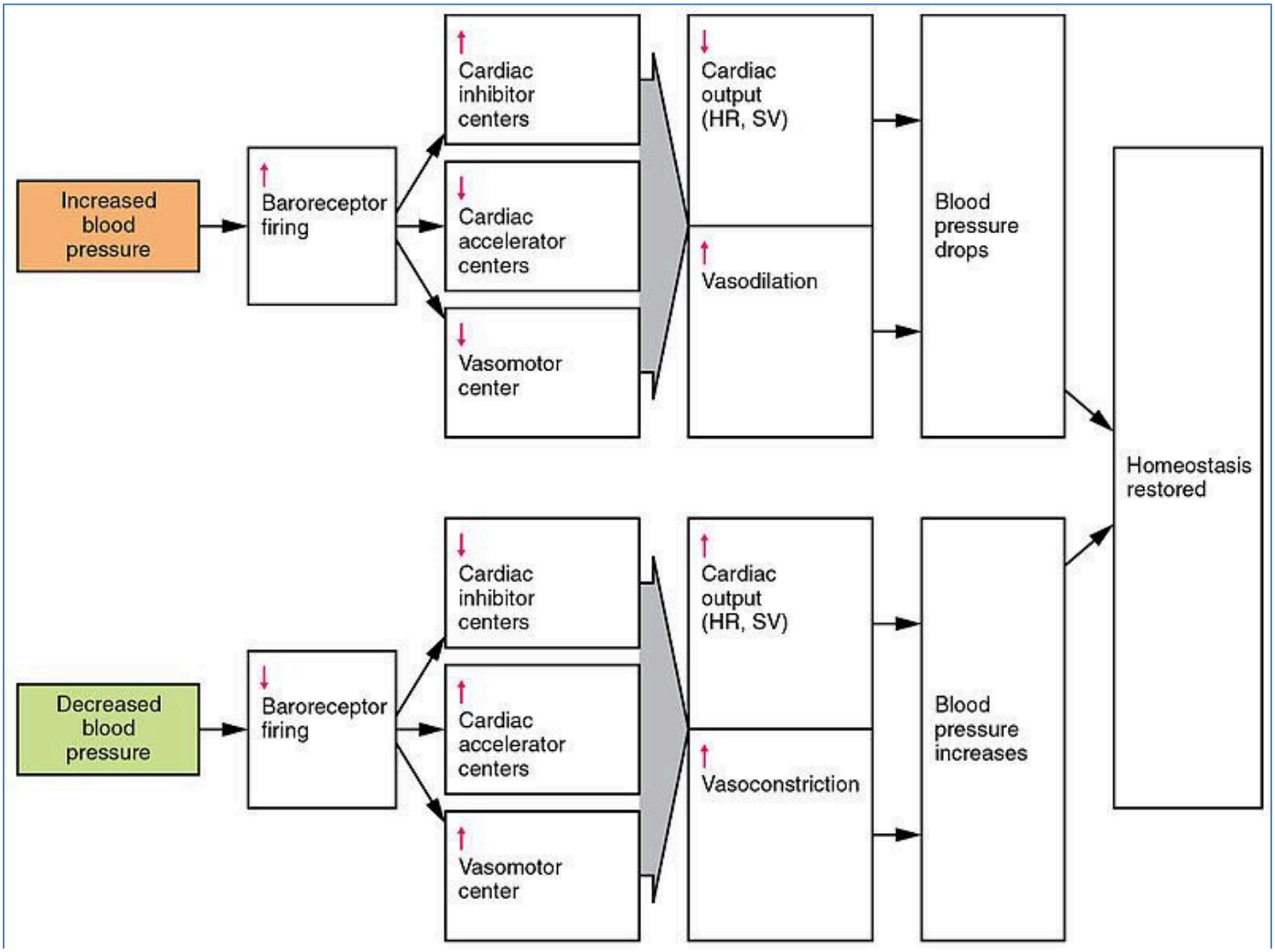
- **1- Alterations in SA-Node Firing:**
  - SA-Node is the Pacemaker.....therefore:
    - Change its rate → change Heart Rate (→change Cardiac Output)

- **2- Autonomic Nervous System:**
  - **Parasympathetic: (Vagus Nerve)**
    - *Decrease* Heart Rate (-ve Chronotropic Effect)
    - *Increase* AV-Node Delay (-ve Dromotropic Effect)
    - NOTE: ONLY A TINY EFFECT ON CONTRACTILITY
  - **Sympathetic: (Sympathetic Chains)**
    - *Increase* Heart Rate (+ve Chronotropic Effect)
    - *Increase* Force of Contraction (+ve Inotropic Effect).



Scientific Figure on ResearchGate. Available from: https://www.researchgate.net/figure/Anatomy-of-the-sympathetic-and-parasympathetic-innervation-of-the-heart-AV_fig1_321702138

- **3- Reflex Controls:**
  - o **Bainbridge Reflex (Atrial Walls)**:
    - § **Where an ↑Venous Return → ↑Heart Rate**
    - § (Stretch of Atrial Walls → Stretch Receptors → Sympathetic NS → ↑HR.)
    - § Responsible for 40-60% of HR increases.
  - o **ChemoReceptor Reflex:**
    - § ↓Low O2 or ↑CO2 in Peripheral-Tissue → ↑HR & ↑Resp Rate
  - o **BaroReceptor Reflex (Aortic & Carotids):**
    - § **Where an ↑BP → ↓HR & ↓Contractility (+ Vasodilation)**
    - § **2 Main Baroreceptors:**
      - **Aortic** → Vagus Nv. → CV Centre(medulla/pons)
      - **Carotid** → Hering's Nv. → CV Centre(medulla/pons)
    - § **Constantly responds to Blood Pressure Changes**
      - (via stretch in vessel walls)
      - More Stretch = More Firing:.leads to:
        - o Parasympathetic Activation
        - o Sympathetic De-activation
    - § Receptors Never Silent – constantly signalling
    - § Quick to respond
    - § In Hypertension → receptors recalibrate to the higher BP.
    - § Changes HR accordingly



- **4- Atrial Node Stretching (Similar to Baroreceptor Reflex, but in the Atrium):**
  - Venous Return Fills Atria With Blood.
  - § When Venous Return ↑, Atrial Walls Stretch → Stretches SA-Node.
  - Stretching of SA-Node Cells → More Rapid Depolarisation → ↑HR
  - Responsible for 15% of HR increases.

    Influenced by:
    - § Arterial Pressure
    - § Peripheral Compliance
    - § Local Blood Flow
    - § Capillary Exchange

- **5- Chemical Regulation:**
  - **Hormones:**
    - § Adrenaline
    - § Thyroxine
    - § Insulin
  - **Ions:** Na+
    - § K+
    - § Ca2+
    - §

- **(Other Factors):**
  - Age (Old → Lower Resting-HR)
  - Gender (Females → Higher Resting-HR)
  - Physical Fitness (Fit → Lower Resting-HR)
  - Temperature (Hot → Higher Resting-HR)

STROKE VOLUME:

- Blood output per heart-beat.
- Useful when examining the efficiency of a *single* cardiac cycle.

Stroke Volume (SV) = End Diastolic Volume (EDV) – End Systolic Volume (ESV)

- **Therefore, Stroke Volume is ↑'d by:**
  - ↑ Ventricular Filling Time (Duration of Ventricular Diastole)
  - ↑ Venous Return
  - ↓ Arterial BP (A High Arterial BP → harder to eject blood → ESV Increases)
  - ↑ Force of Ventricular Contraction.

2 Things That Affect Stroke Volume:

- **1- PreLoad: Degree of Stretch of Heart Muscle:**
  - The degree of *Stretching* of the Heart Muscle during Ventricular Diastole.
    - Caused by amounts of blood from venous return.
  - Influenced by:
    - Arterial Pressure
    - Peripheral Compliance
    - Local Blood Flow (depending on the demands of those tissues)
    - Capillary Exchange.
  - Preload ↑ as EDV↑. (Directly Proportional)
    - **↑End Diastolic Volume = ↑Stroke Volume (Frank-Starling Law)**
  - Affects % of actin/myosin contact in myocytes→ Affects cross-bridge cycling:
    - → Affects muscle's ability to produce tension.

    Preload Varies with demands placed on heart.

    Contractility:
    - Inotropy
    - Force produced during contraction *at a given Preload*.
    - Influences *End Systolic Volume* (↑Contractility = ↓ESV)

- **2- Afterload: Back Pressure Exerted by Arterial Blood:**
  - The tension needed by Ventricular Contraction to *Open Semilunar Valve.*
    - Ie: The pressure the heart must reach to eject blood.

    ↑Afterload = ↑ESV = ↓SV

    Afterload is increased by anything that *Restricts Arterial Blood Flow.*

	Factors Affecting Stroke Volume (SV)		
	Preload	**Contractility**	**Afterload**
Raised due to:	• fast filling time • increased venous return **Increases end diastolic volume,** **Increases stroke volume**	• sympathetic stimulation • epinephrine and norepinephrine • high intracellular calcium ions • high blood calcium level • thyroid hormones • glucagon **Decreases end systolic volume,** **Increases stroke volume**	• increased vascular restistance • semilunar valve damage **Increases end systolic volume** **Decreases stroke volume**
Lowered due to:	• decreased thyroid hormones • decreased calcium ions • high or low potassium ions • high or low sodium • low body temperature • hypoxia • abnormal pH balance • drugs (i.e., calcium channel blockers) **Decreases end diastolic volume,** **Decreases stroke volume**	• parasympathetic stimulation • acetylcholine • hypoxia • hyperkalemia **Increases end systolic volume** **Decreases stroke volume**	• decreased vascular resistance **Decreases end systolic volume** **Increases stroke volume**

HAEMODYNAMICS / HEMODYNAMICS

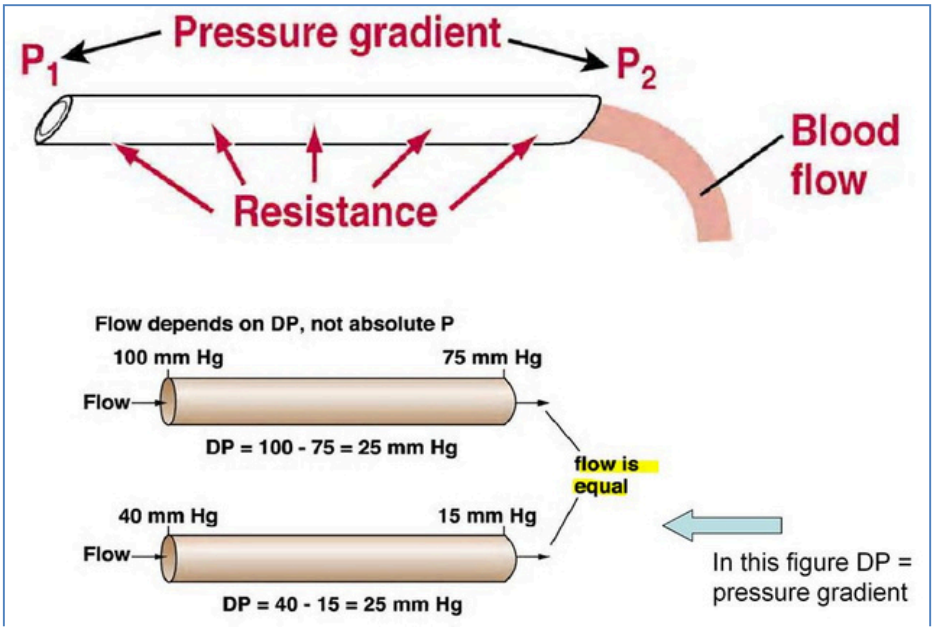
HEMODYNAMICS

Relationship Between *Flow, Pressure & Resistance:*

- 1- Flow is Directly Proportional to Pressure Gradient between 2 points (*Change* in Pressure)
- 2- Flow is Inversely Proportional to Resistance
- **Therefore:**

$$Flow\ (F) = \frac{Pressure\ Gradient\ (\Delta P)}{Resistance}$$

Note: Resistance is *far more important* in determining *local blood flow* than the Pressure Gradient.



Blood Flow Rate:

- **The *Amount of blood* flowing through a vessel/organ/system *per unit time*. (mLs/min)**
- o Determined by pressure gradient & resistance, NOT Velocity.
- **Systemic Blood Flow** = Cardiac Output (relatively constant)
  Specific Organ Blood Flow – may vary widely due to its immediate needs.

Velocity of Flow:

- **Velocity of Flow = *SPEED of flowing blood*. *(mm/sec)***
- **Eg:** A constricted vessel will have a ***lower flow rate***, but a ***higher velocity of flow***. (Ie: Garden hose)
- **Note:** Velocity tends to change by a greater magnitude than the change in Flow Rate.

Blood Pressure:

- The Pressure exerted on the vessel wall by contained blood. (mmHg)
- Decreases with distance from heart. (arterial system)
- Decreases with 10%+ decrease blood volume.
- Increases with vessel constriction (provided same blood volume)

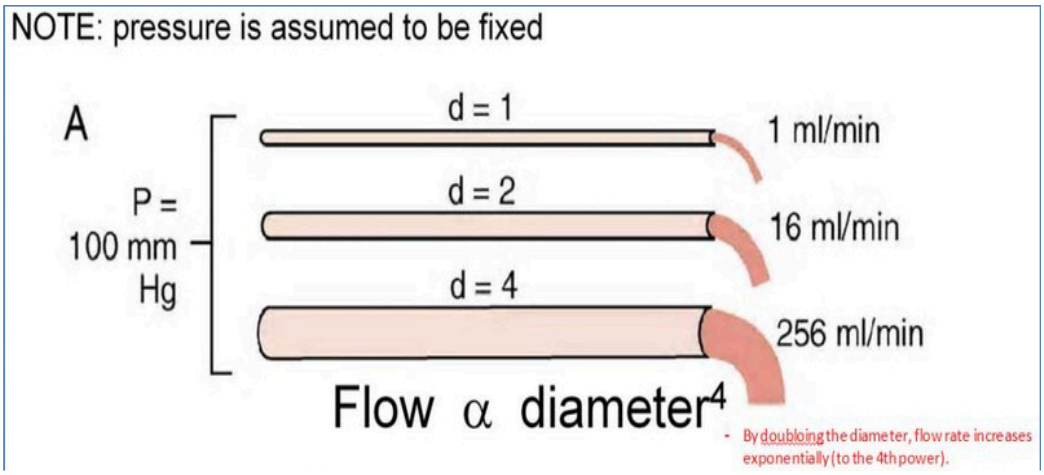
Resistance:

- The amount of *Friction* blood encounters *as it passes through the vessels*.
- **3 Factors Influencing Resistance:**
  - o **Blood Viscosity** (↑Viscosity = ↑Resistance) (Fairly Constant)
  - o **Total Vessel Length** (longer vessel = ↑ resistance) (Fairly Constant)
  - o **Vessel Diameter** (thinner vessel = ↑resistance) (Frequently Changes)
    - § Most Responsible for changes in BP
- **Systemic Vascular Resistance = Combination of the Above Factors**

Effects of Vessel Diameter (Vasomotion) on *Flow Rate:*

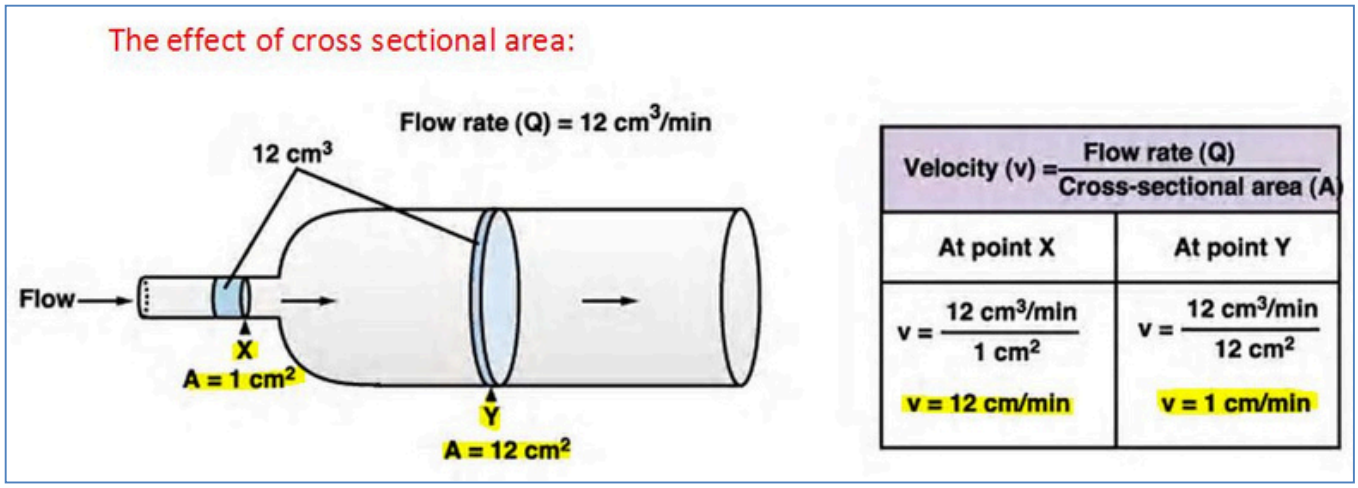
- The ***Flow Rate*** is directly ***proportional to*** the ***4th Power*** of the ***Vessel Diameter***.
- Ie: Small changes in vessel diameter → Changes Flow Rate by an exponent of 4.

(Poiseuille's Law)



E ffe cts o f V e sse l D ia m e te r (V a so m o tio n) o n *F lo w V e lo city :*

- ***Flow Rate*** is ***inversely proportional*** to the vessel's ***cross-sectional area***.
- Ie: An 'α' x Increase in cross-sectional area → Decreases Flow Velocity by a factor of 'α'.



BLOOD PRESSURE PHYSIOLOGY:

BLOOD PRESSURE IN DETAIL:

Get Direction GLOBAL

Factors Influencing Blood Pressure:

- **Cardiac Output:**
- o ↑Cardiac Output = ↑ BP

Peripheral Resistance:

- o Causes backpressure in blood (arterial system)
- o Eg: In Obesity, peripheral resistance increases.

- **Blood Volume:**
  - o (assuming constant vessel diameters) ↑Blood Volume = ↑BP
  - o Its affect depends on vessel compliance

BP = Cardiac Output X Total Peripheral Resistance

Types of Blood Pressures:

- **Systolic:**
  - o Peak Aortic pressure reached during ventricular systole.
  - o A Function of:
    - § Peak *rate* of ejection
    - § Vessel wall compliance
    - § Diastolic BP
  - o Normal = 120mmHg

- **Diastolic:**
  - o Lowest Aortic pressure reached during ventricular diastole, due to blood left after peripheral runoff.
  - o A Function of:
    - § Blood Volume
    - § Heart Rate
    - § Peripheral Resistance
  - o Normal = 80mmHg

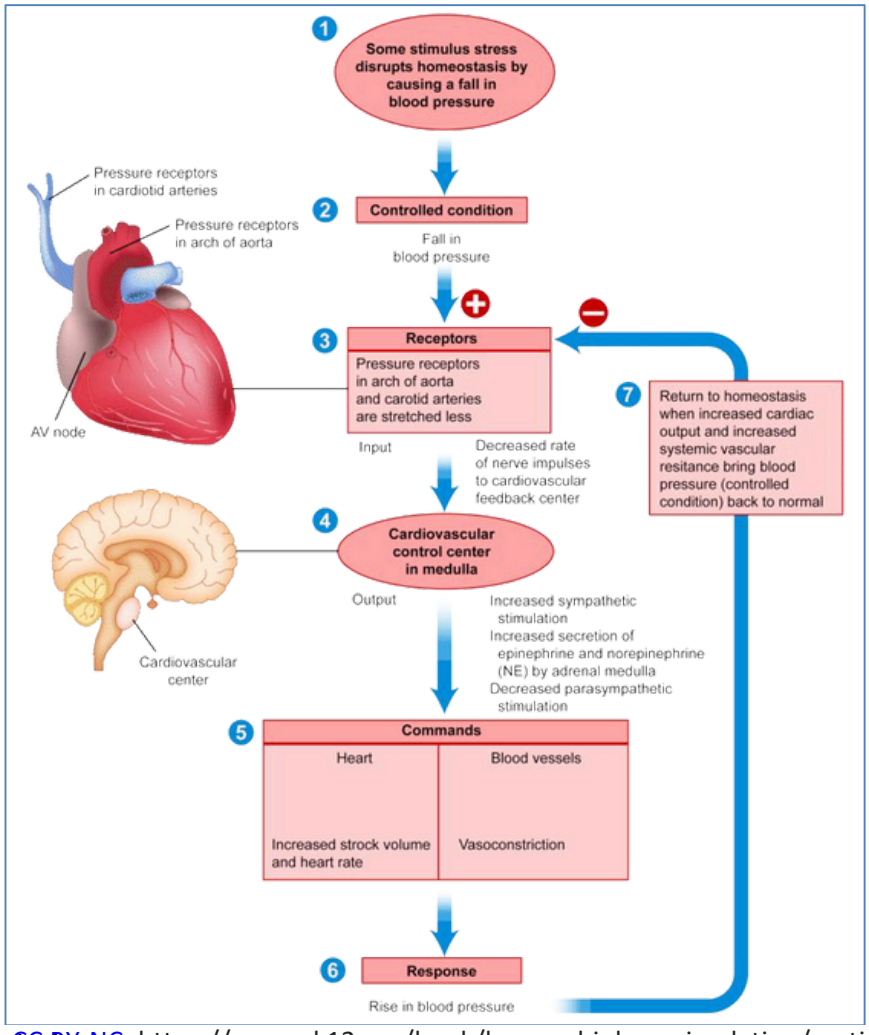
- ***Pulse Pressure:**
  - o **Pulse Pressure = Systolic Pressure - Diastolic Pressure**
  - o (Eg: 120mmHg – 80mmHg)
  - o Normal = 40mmHg
  - o If Lower – may be an indication of Aortic Stenosis or Atherosclerosis (slowed peripheral runoff)

- ***Mean Arterial Pressure (MAP):**
  - o MAP = Diastolic Pressure + 1/3(Pulse Pressure)
  - o *The Pressure that *Propels Blood to the Tissues* – maintains *Tissue Perfusion* (see below sections).
    - § Maintains flow through capillary beds
  - o Must be high enough to overcome peripheral resistance – (if not blood doesn't move)
  - o Finely Controlled

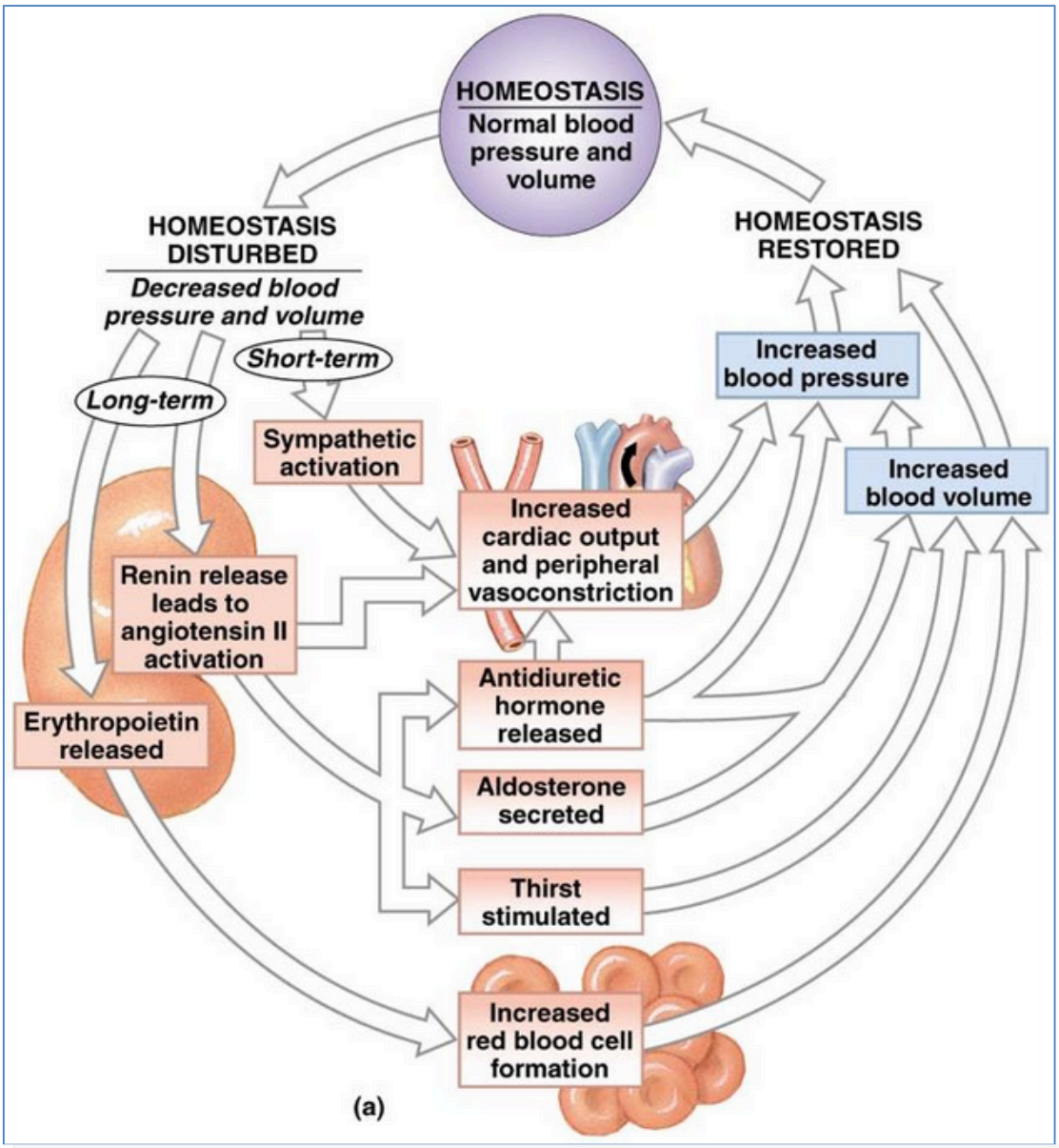
3 Main Regulators of Mean Arterial Pressure (MAP):

- **1- Autoregulation (@ the Tissue Level):**
  - o **Localised Automatic Vasodilation/constriction at the *Tissue* Level**
    - § Allows Control of flow within *a single capillary bed*.
    - § Ensures perfusion of the *'Needy'* Tissues
  - o **Metabolic Controls: → Vasodilation:**
    - § Low Oxygen/Nutrient levels
    - § Nitric Oxide
    - § Endothelin
    - § Inflammatory Chemicals: (histamine/kinins/prostaglandins)
  - o **Myogenic Control: → Vasoconstriction:**
    - § Sheer Stress: Vascular smooth muscle responds to passive stretch (↑vascular pressure) with increased tone.
      - Prevents excessively high tissue perfusion that could rupture smaller blood vessels.
    - § Reduced stretch promotes vasodilation → flow increases.

- **2- Neural Mechanisms:**
  - o **Vasomotor Centres (Medulla):**
    - § Take info from receptors:
      - Baroreceptors (primarily)
      - Chemoreceptors (lesser degree)
    - § Transmit impulses *via Sympathetic.NS*:
      - ↑ sympathetic activity = vasoconstriction = ↑ BP
      - ↓ sympathetic activity = vasodilation = ↓ BP
  - o **Cardiovascular Centres of the Autonomic Nervous System:**
    - § Sympathetic → ↑HR & Contractility → ↑MAP
    - § Parasympathetic → ↓Heart Rate → ↓MAP



- **3- Endocrine Mechanisms (Kidney Level):**
  - o More for Long Term BP & Blood-Volume regulation:
  - o ****Antidiuretic Hormone (ADH) – AKA. Vasopressin:**
    - § Released due to *Low* blood *volume*
    - § **ADH → Water Retention Increased → ↑MAP**
  - o **Angiotensin II:**
    - § Released due to *Low* blood *pressure*
    - § Potent VasoConstrictor
    - § Increases Cardiac Output & Blood volume
    - § **Angiotensin-II → VasoConstriction → ↑MAP**
    - § (NOTE: 'ACE' (Angiotensin I Converting Enzyme) activates it to Angiotensin II. Hence 'ACE-Inhibitors' are often used as *AntiHypertension* medicine)
  - o **Erythropoietin:**
    - § Released due to *Low Pressure & Low O2 Levels*
    - § Increases RBC production to increase Blood Volume.
    - § **EPO → Haematopoiesis → ↑Blood Volume → ↑MAP**
  - o **Natriuetic Peptides (Released by the heart):**
    - § Released *by the heart* due to *High Blood Pressure & Volume.*
    - § **↑Stretch on Heart → NP Release → ↑Diuresis → *Reduces BP & Volume*.**
    - § Also Inhibits ADH & Angiotensin II → *Reduces BP & Volume.*

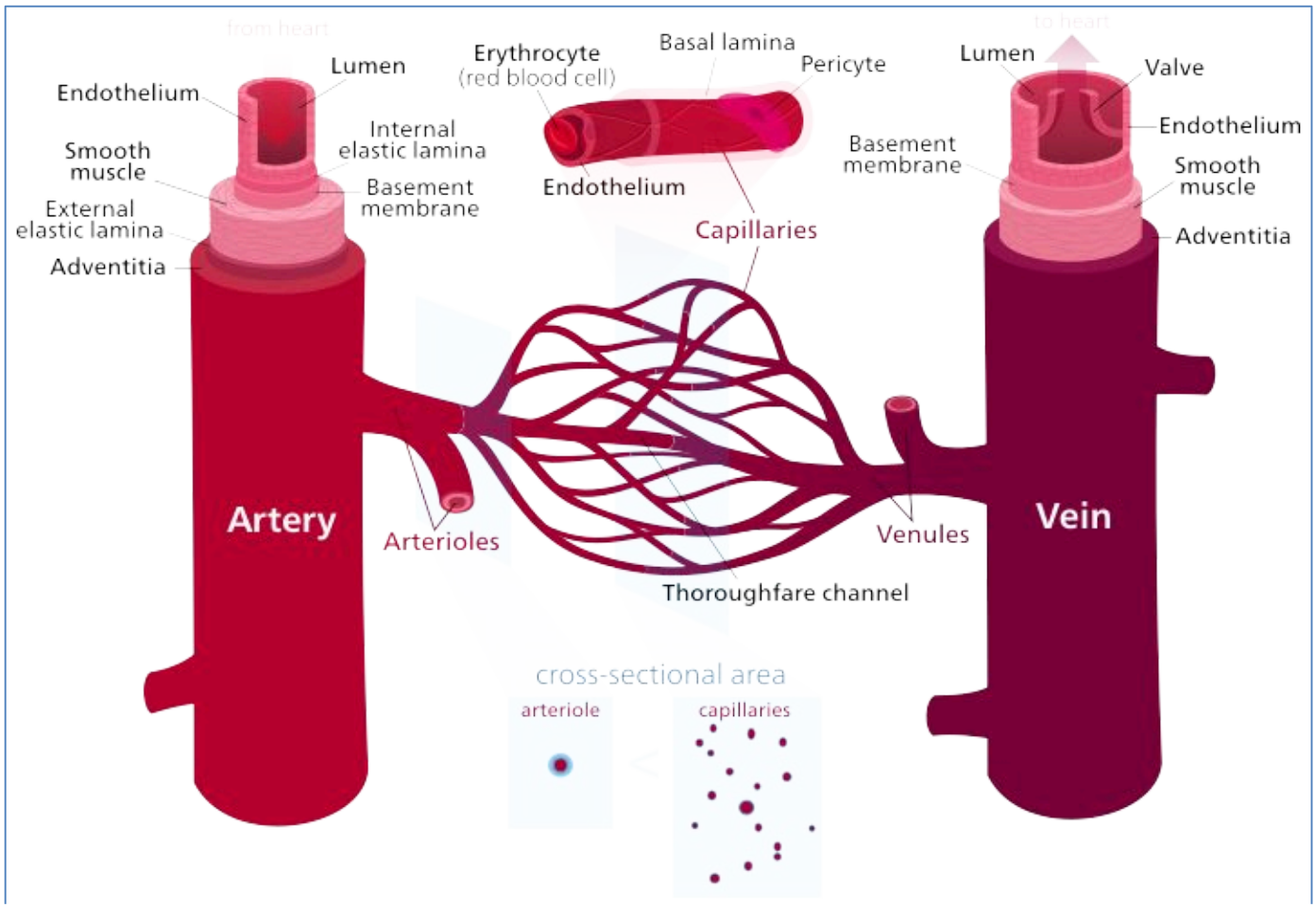


ANATOMY & PHYSIOLOGY OF BLOOD VESSELS

BLOOD VESSELS

Introduction to Blood Vessels:

- **3 Classes:**
  - o **Arteries – Carry blood away from the heart**
    - § Elastic Arteries Eg: Aorta & Major Branches (Conducting Vessels)
    - § Muscular Arteries Eg: Coeliac Trunk & Renal Arts. (Distributing Vessels)
    - § Arterioles Eg: Intra-Organ Arteries (Resistance Vessels)
    - § Terminal Arteriole Eg: Afferent Arteriole in kidney
  - o **Capillaries – Intimate contact with tissue → facilitate cell nutrient/waste transfer**
    - § Vascular Shunt
    - § True Capillaries
  - o **Veins – Carry blood back to the heart**
    - § Post-Capillary Venule (Union of capillaries) (Capacitance Vessels – 65% of body's blood is venous)
    - § Small Veins (Capacitance Vessels – 65% of body's blood is venous)
    - § Large Veins



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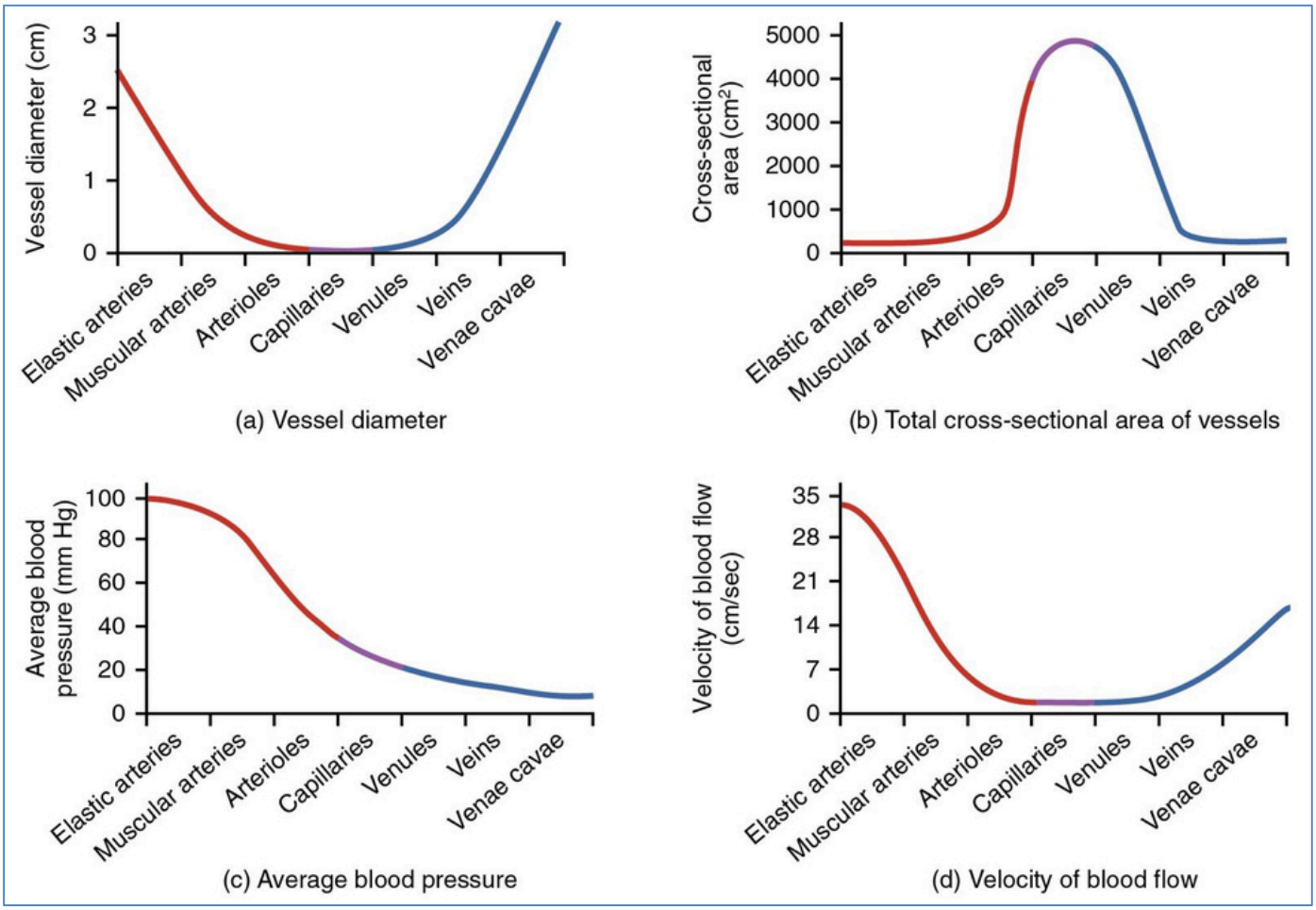
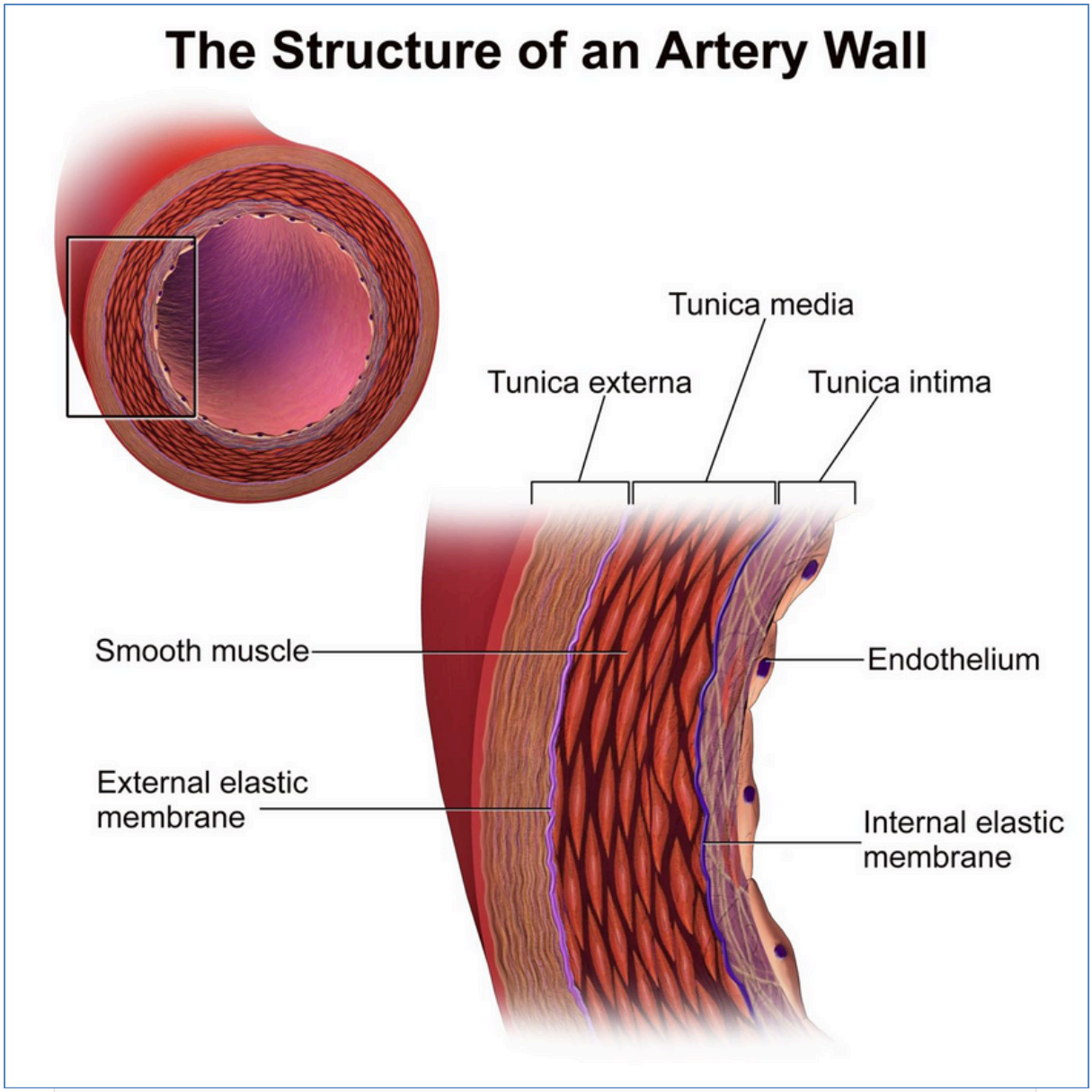
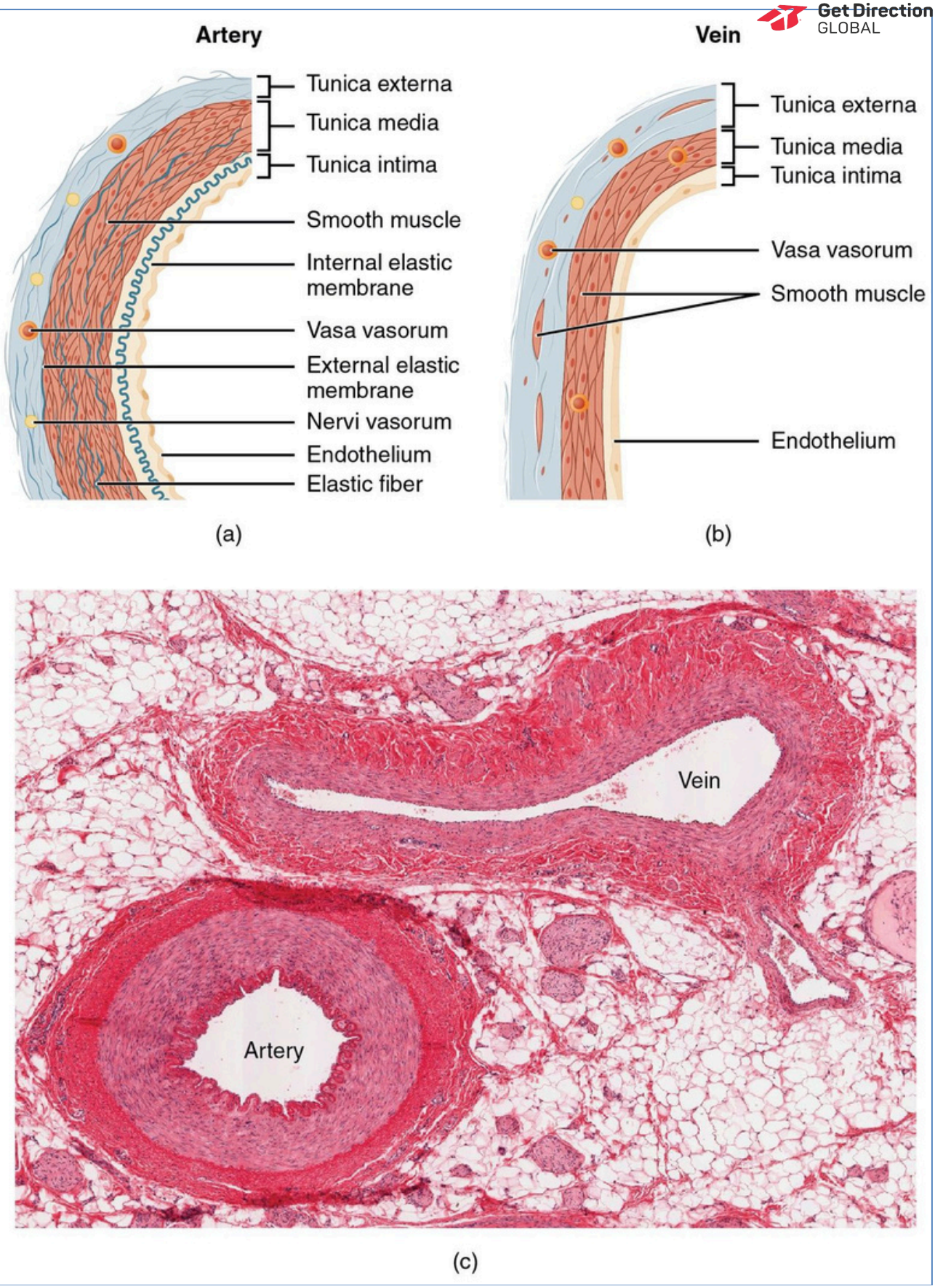


Blood Vessel Structure:

- **3-Layered Wall:**
  - o **Tunica Intima:**
    - § Ie: The layer in *intimate* contact with the blood (luminal)
    - § Consists of ***The Endothelium*** (Simple Squamous Epithelium)
    - § Larger vessels also have a ***Sub-Endothelial Layer***
  - o **Tunica Media:**
    - § Middle....& Thickest layer (*Smooth Muscle & Elastin*)
      - Circulating *Smooth Muscle*
      - Sheets of *Elastin*
    - § Regulated by Sympathetic Nervous System + Chemicals
    - § Contraction/Dilation *Maintains Blood Pressure.*
  - o **Tunica Externa:**
    - § Outermost Layer (Loose *collagen fibres*)
    - § (NOTE: Also Contains Nerve Fibres, Lymphatics, and Vasa Vasorum (In larger vessels))

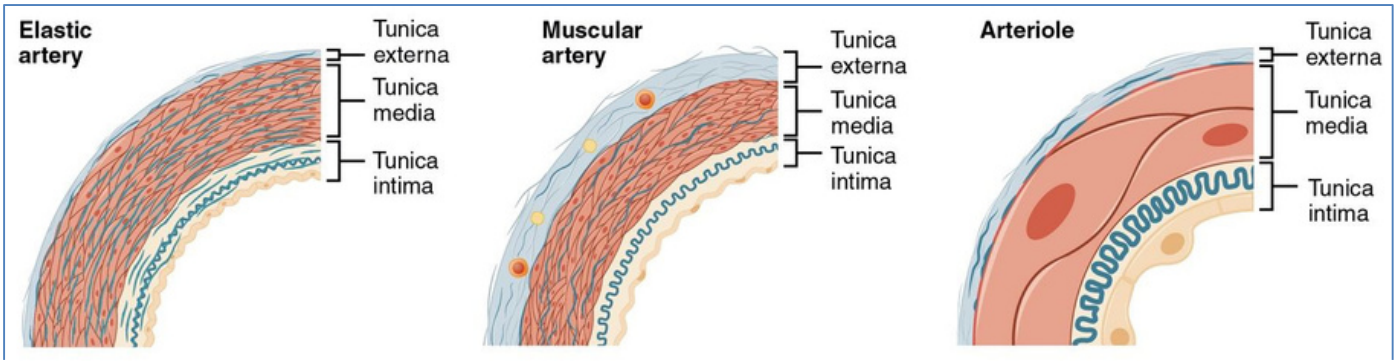


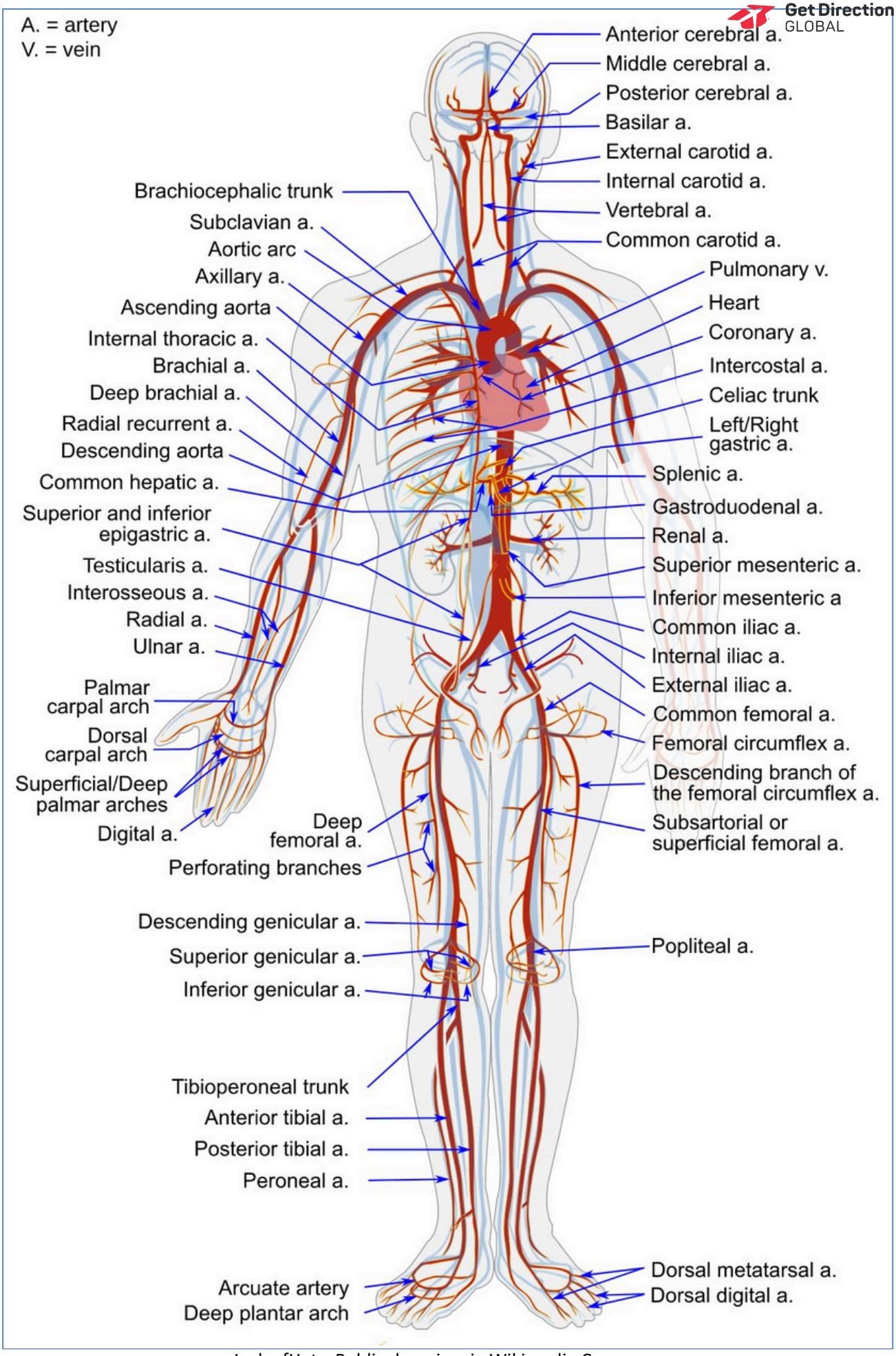
Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". *WikiJournal of Medicine*



The Arterial System:

- **Elastic (Conducting) Arteries:**
  - o **The Aorta + its major branches**
  - o Thick-Walled
  - o Large Lumen = Low resistance
  - o Highest Proportion of *Elastin:*
    - § Withstands Pressure Fluxes
    - § Smoothens out Pressure Fluxes
    - § 'Stretch' = potential energy → helps propel blood during diastole.
- **Muscular (Distributing) Arteries:**
  - o o **Distal to Elastic Arteries**
  - o Diameter: 0.3mm → 1cm o Deliver blood to specific body organs
  - Thickest Tunica Media:
    - § Due to smooth muscle
  - o Highest Proportion of *Smooth Muscle:*
  - Are § active in vasoconstriction
  - Are therefore less *distensible* (less elastin)
- **Arterioles:**
  - o **Smallest Arteries**
  - o **Larger Arterioles** have all 3 Tunics (Intima/media/externa).....
    - § Most of the Tunica Media is Smooth Muscle
  - o **Smaller Arterioles** – lead to capillary beds
    - § Little more than 1 layer of smooth muscle around the endothelial lining.
  - o **Autoregulation of Diameter:**
    - § Controlled by:
      - Neural (electrical) signals
      - Hormonal signals (NorAdrenaline/Epinephrine/Vasopressin/Endothelin-1/etc)
      - Local chemicals
    - § Controls blood flow to Capillary Beds
      - When constricted – tissues served are bypassed
      - When dilated – Tissues served receive blood.
  - o Biggest controller of Blood Pressure





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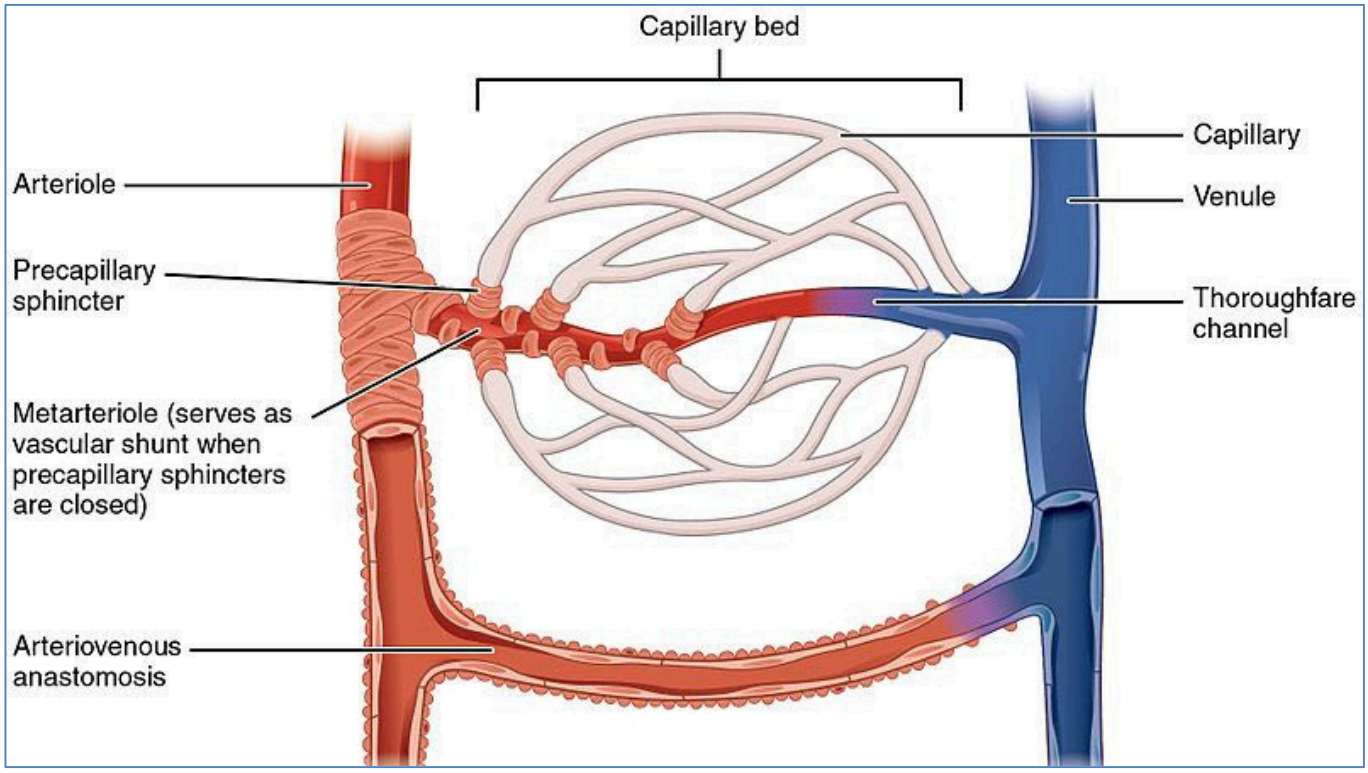
The Capillary System:

- **Smallest blood vessels - microscopic**
- Thin, thin walls – Tunica Intima Only – Ie: Only 1 layer thick.
- Average length = 1mm
- Diameter: The width of a single RBC.
    - RBC's flow through capillaries in single file
    - RBC's shape allows them to stack up efficiently against each other.
- Penetrate most tissues.
- (except tendons/ligaments/cartilage/epithelia)

Main Role:

- Exchange of Gases/Nutrients/Hormones/Wastes
- Exchange occurs between Blood & Interstitial Fluid.

- **Capillary Beds:**
    - Capillaries are only effective in large numbers:
        - Form networks called *'capillary beds'*
    - Facilitates ***'Microcirculation':*** Blood-Flow from an Arteriole → Venule
        - Consist of **2 Types of Vessels:**
            - **Vascular Shunt:**
                - From Metarteriole → Thoroughfare Channel
                - Short vessel – directly connects Arteriole with Venule.
            - **True Capillaries:**
                - The ones that actually take part in *exchange* with tissues.
                - Usually branch off the ***Metarteriole*** (proximal end of vascular shunt)
                - Return to the ***Thoroughfare Channel*** (distal end of vascular shunt)
                - **Precapillary Sphincters:**
                    - Smooth muscle *Cuffs*
                    - Surround the roots of each true capillary (arterial ends)
                    - Regulates blood flow into each capillary
                    - Ie: Blood can either go through capillary or through the shunt.
    - A Capillary Bed may be flooded with blood or bypassed, depending on conditions in that organ.



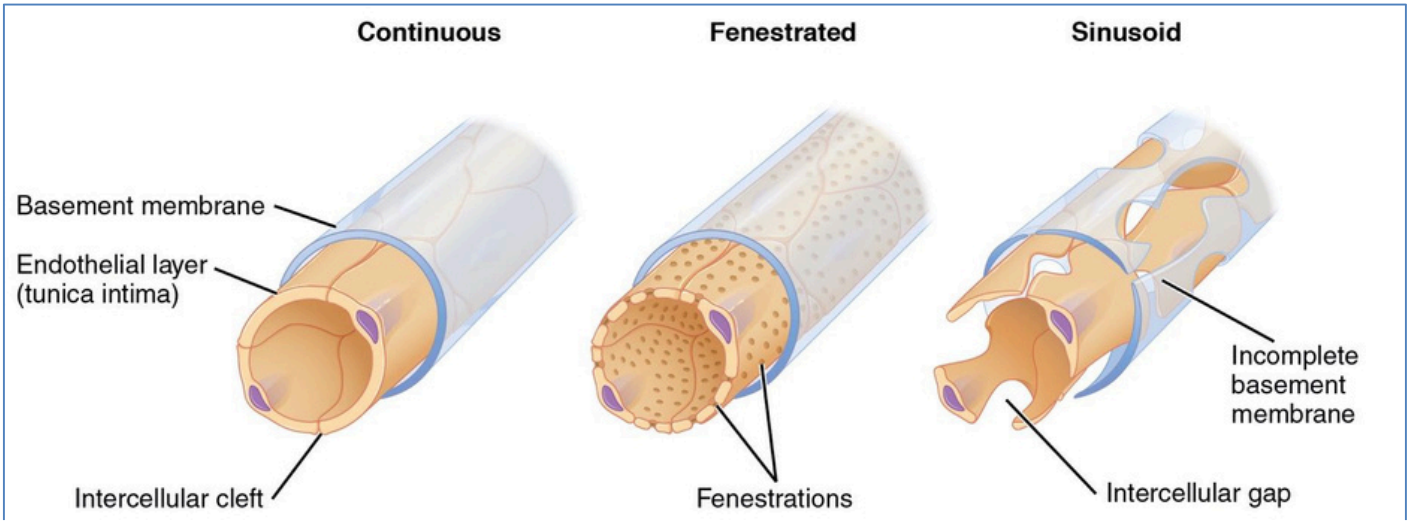
- **3 Types of Capillaries:**
  - **Continuous Capillaries:**
    - **'Continuous' = uninterrupted endothelial lining**.
      - Adjacent cells form ***Intercellular Clefts****:*
        - Joined by incomplete-*tight-junctions*
        - (Ie: Allow limited passage of fluids & solutes)
      - Note: in the brain, the *tight-junctions* are *complete* → blood brain barrier.

  - **Fenestrated Capillaries:**
    - **Endothelial cells are riddled with oval pores *(Fenestrations = windows)***
      - Much more permeable to fluids & solutes than *continuous capillaries.*

      Abundant wherever active absorption/filtration occurs.
      - Ie: Intestines
      - Kidneys
      - Endocrine organs (allow hormones rapid entry to blood)

  - **Sinusoids (Sinusoidal Capillaries):**
    - **AKA "Leaky Capillaries"**
    - **Found ONLY in:**
      - Liver
      - Bone Marrow
      - Lymphoid Tissues
      - Some Endocrine Organs
    - Large Irregularly-shaped lumens
    - Usually fenestrated
    - 'Discontinuous' = interrupted by *Kupffer Cells*:
      - Remove & destroy bacteria

      Intercellular clefts → larger + have fewer tight junctions
      - Allow *large molecules* & *leukocytes* passage through to Interstitial Space.



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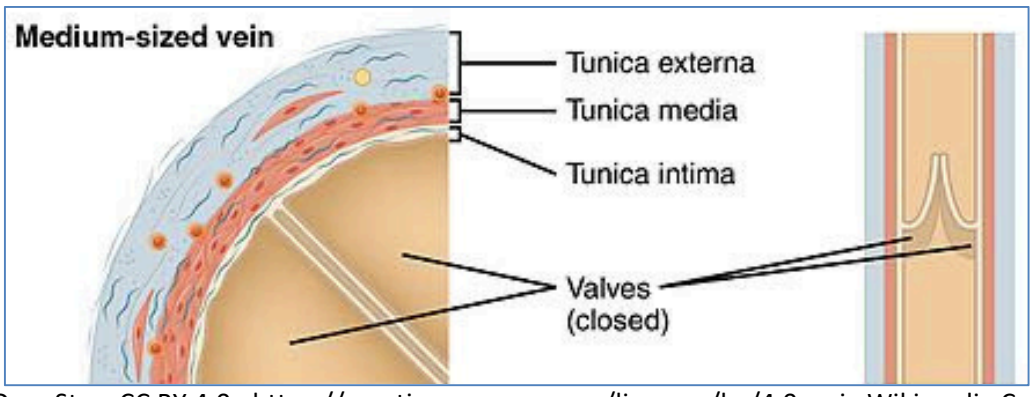
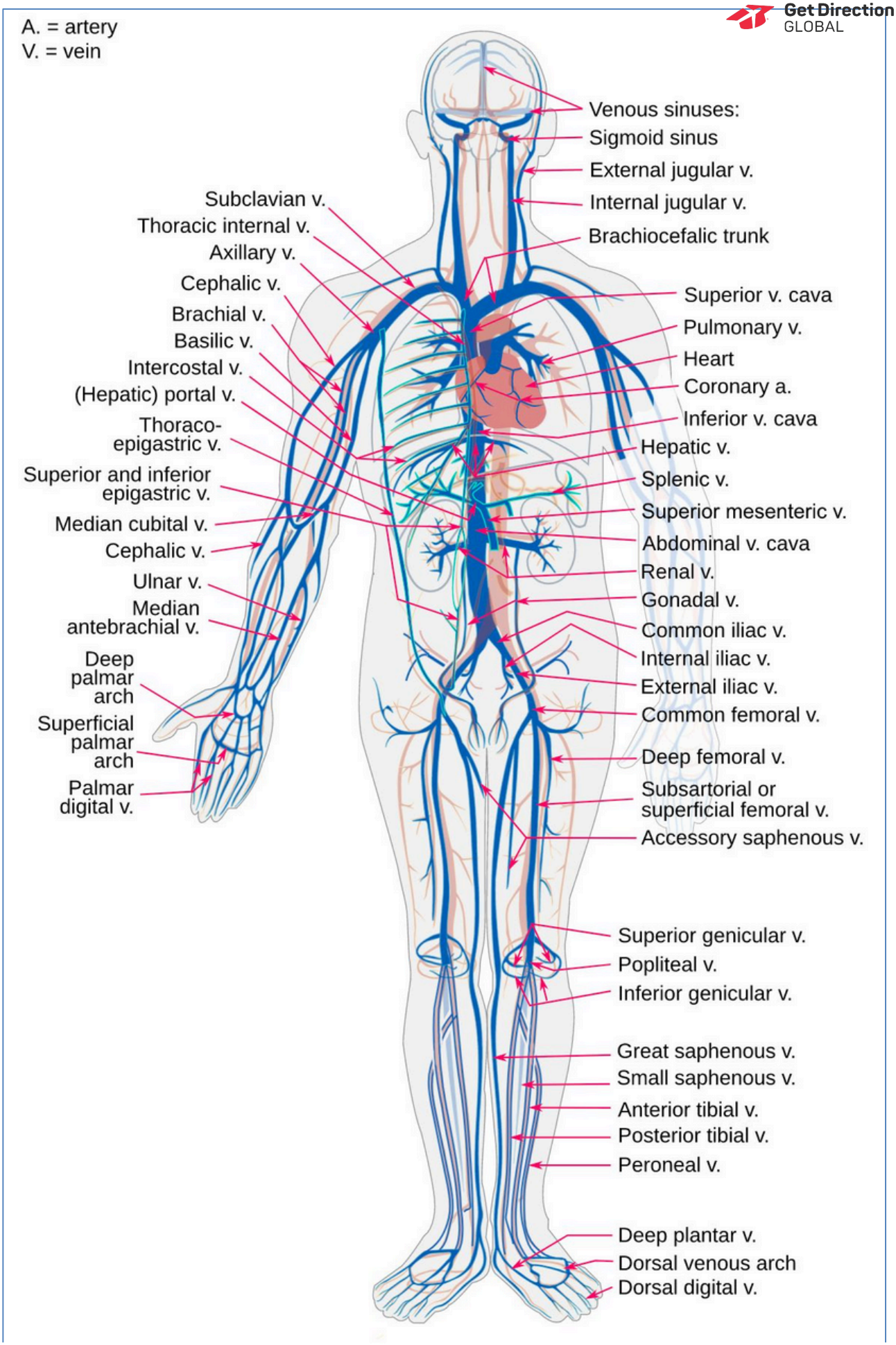
The Venous System:

- Vessels carry blood back towards the Heart. (From Capillary Beds)
- Vessels gradually increase in Diameter & Thickness towards the heart.
- **2 Types:**
  - **Venules:**
    - Formed by union of capillaries (post-capillary venules)
    - Consist entirely of Endothelium
    - Extremely porous; Allows passage of:
      - Fluid &
      - White Blood Cells (migrate through wall into inflamed tissue)
    - The larger venules:
      - Have 1or2 layers of smooth muscle (Ie: Tunica Media)
      - Have a thin Tunica Externa as well
  - **Veins:**
    - Formed by union of Venules
    - 3 distinct Tunics (but walls thinner than corresponding arteries)
    -
      - Thinner walls due to lower Blood Pressure

      Tunica Media:
      - Poorly developed
      - Some smooth muscle
      - Some elsastin
      - Tend to be thin even in large veins.
    - Tunica Externa:
      - Heaviest layer (thicker than Media)
      - Thick longitudinal collagen bundles
      - Thick elastic networks
    - Lumens larger than corresponding arteries
      - The reason 65% of body's blood is in the veins.
      - Therefore Veins: aka ***"Capacitance Vessels"***
    - **Lower Blood Pressure** than arteries:
      - Require structural adaptations to get blood → heart:
        - Large lumen (low resistance)
        - Valves
    - **Venous Valves:**
      - Folds of Tunica Intima (resemble Semilunar Valves)
      - Prevent blood flowing backward
      - Ensures unidirectional flow
      - Often have to work against gravity.
      - If Faulty:
        - Causes thrombosis (Eg: Varicose veins)





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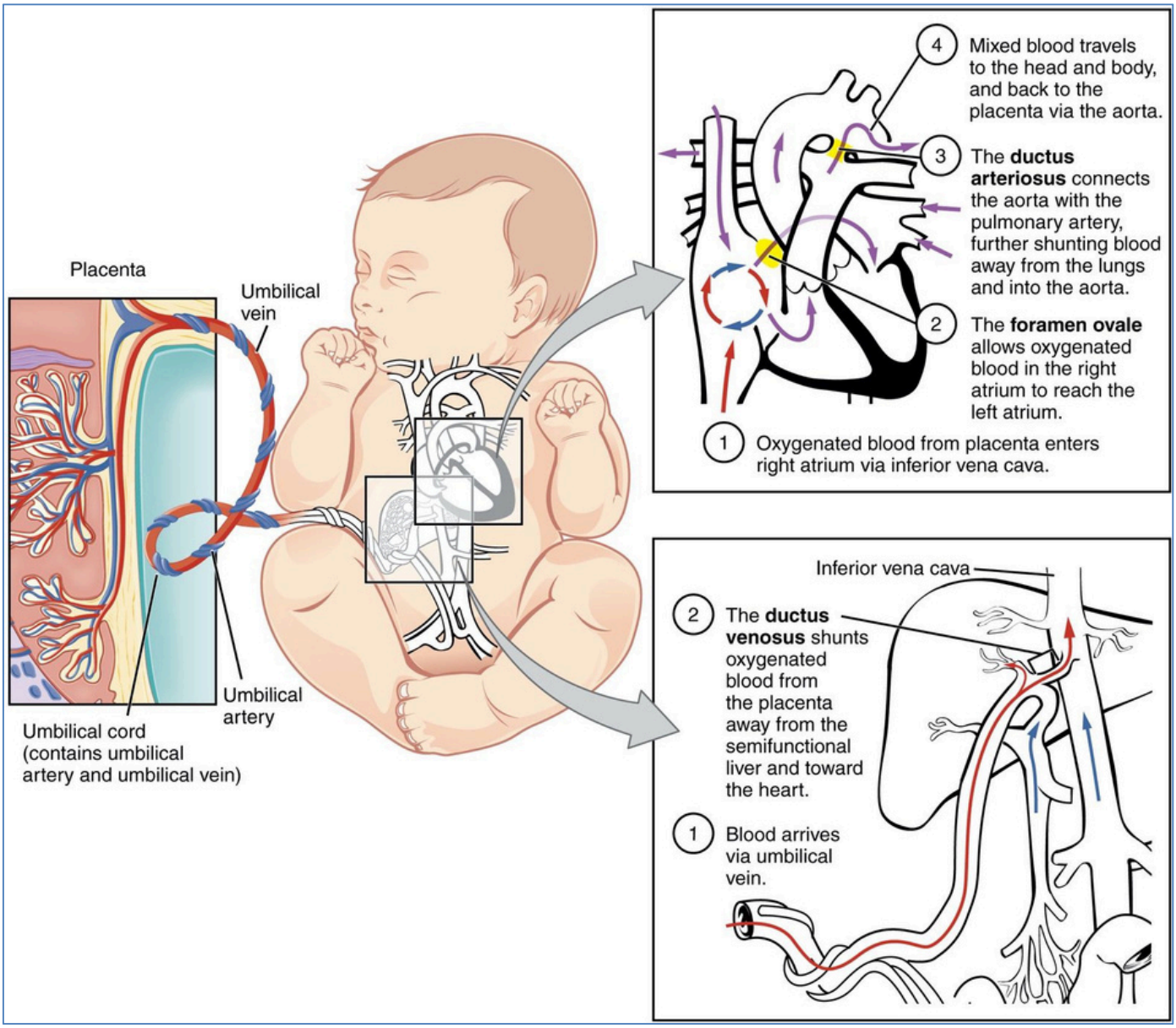
Foetal Circulation:

- **"Bypasses" / "Shunts" of foetal circulatory system:**
  - **Ductus Venosus**
    - Directs the oxygenated blood from the placental vein into inferior vena cava→heart
    - Partially bypasses the liver sinusoids
  - **Foramen Ovale**
    - An opening in the **interatrial septum** loosely closed by a flap of tissue.
    - Directs some of blood entering the right atrium into the left atrium → Aorta.
    - Partially bypasses the lungs.
  - **Ductus Arteriosus**
    - Directs most blood from right atrium of the heart directly into aorta
    - Partially bypasses the lungs
  - **All of these "shunts" are occluded at birth due to pressure changes.
    - NOTE: The Foramen Ovale can take up to 6 months to close.



Fluid Movements Across a Vessel:

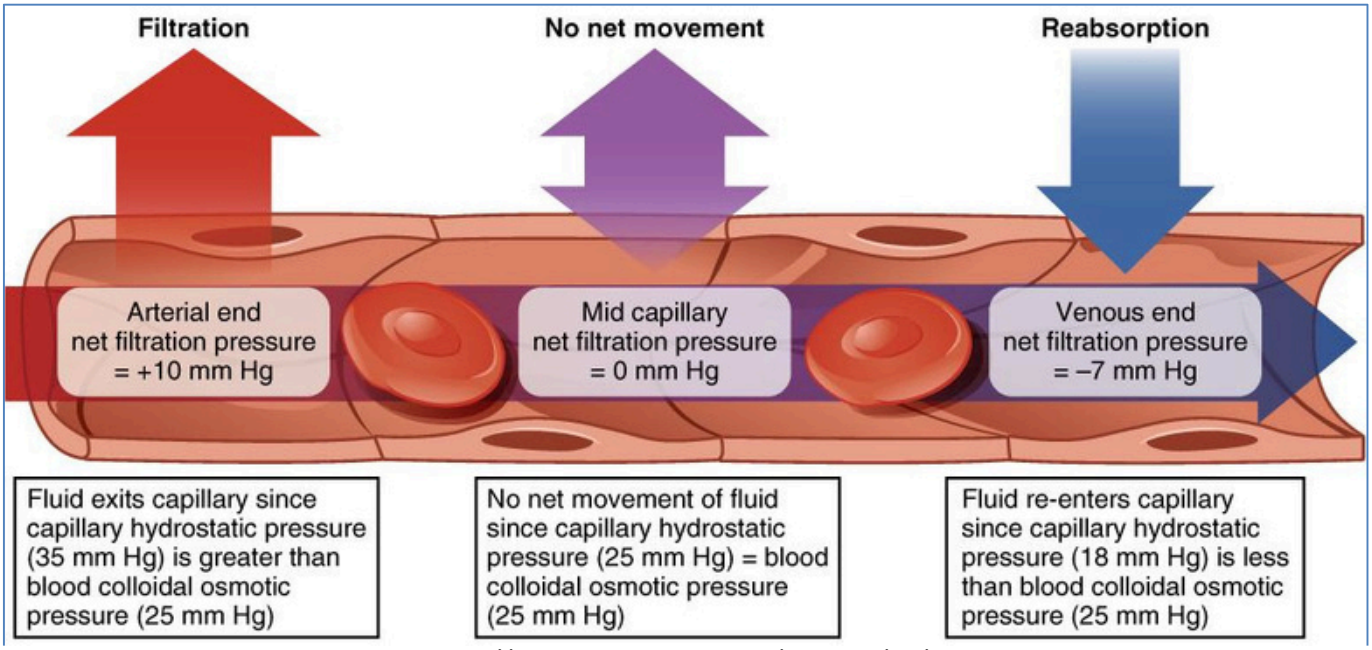
- **Determined by the balance of 2 forces:**
  - **Capillary Hydrostatic Pressure:**
    - The force the blood exerts against the capillary wall.
    - ***Hydrostatic pressure = capillary blood pressure ≈35mmHgArterial End /15mmHgVenous End***
    - Tends to force fluids through the capillary's ***Intercellular Clefts*** (between endothelial cells)
      - Capillary hydrostatic pressure drops as blood flows from arteriole → venule.

      Net Hydrostatc Pressure = Capillary Hydrostatic Pressure – Interstitial Hydrostatic Pressure.
      - Note: Interstitial Hydrostatic Pressure ≈ 0mmHg

  - **Colloid Osmotic Pressure:**
    - Opposes hydrostatic pressure
    - Due to large, non-diffusible molecules (Plasma Proteins) drawing fluid into capillaries.
    - ***Typically ≈25mmHg***
      - Relatively constant at both Arterial & Venous ends

      Net Osmotic Pressure = Capillary Osmotic Pressure – Interstitial Osmotic Pressure.
      - Note: Interstitial Osmotic Pressure ≈ 1mmHg

- Hence **Fluid** is **Forced Out @ Arterial End** & **Reabsorbed @ Venous End**
- The *amount* of fluid forced out – determined by the balance of net Hydrostatic & Osmotic forces.
  - Ie: ***Net Filtration Pressure = Net Hydrostatic Pressure – Net Osmotic Pressure***



Oedema:

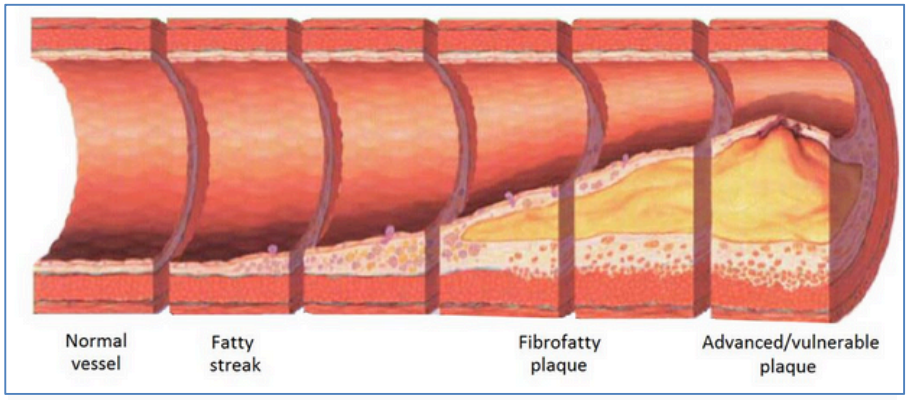
- **Abnormal accumulation of fluid in the Interstitial Space = Ie: *Tissue Swelling***
- **Caused by:** increase in F*low of Fluid → Out of Vessel* OR *Lack of Re-Absorption → Into Blood Vessel*
- Usually reflects an imbalance in Colloid Osmotic Pressure on the 2 sides of the Capillary Membrane.
- Eg: Low levels of plasma protein (reduces amount of water drawn into capillaries.

Contributing Factors:

- **High BP (Hydrostatic Pressure):**
  - Can be due to incompetent valves...OR
  - Localised Blood Vessel Blockage...OR
  - Congestive Heart Failure (Pulmonary Oedema – due to blockage in pulmonary circuit)...OR
  - High Blood Volume
- **Capillary Permeability:**
  - Usually due to an Inflammatory Response

Injuries to Blood Vessels

- **Eg: Atherosclerosis.**
  - o The formation of fatty plaques in the subendothelial layer
  - o Fatty plaques begin to ulcerate



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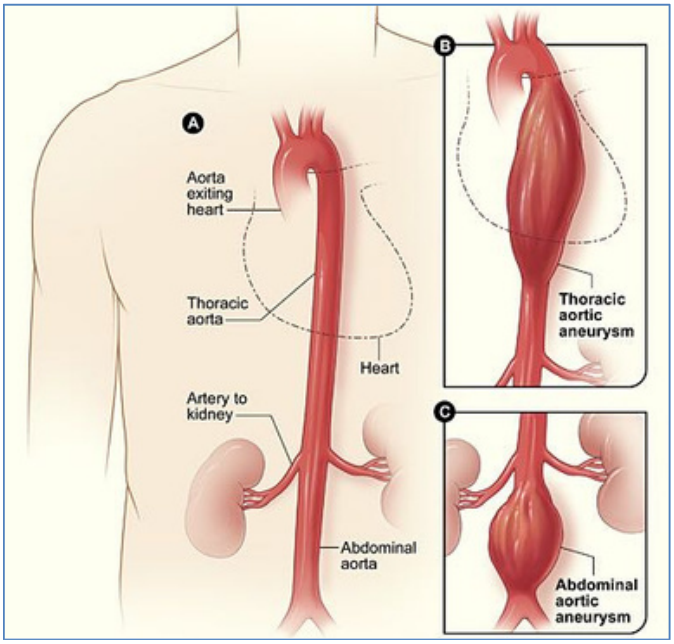
- o **Eg: Aneurysms:**
  - Elastic arteries can lose their elasticity
  - Due to having thinner walls, they're more prone to **aneurysm** (bulging & potentially rupturing)
    - § Results in pooling of blood --> eventual rupture.



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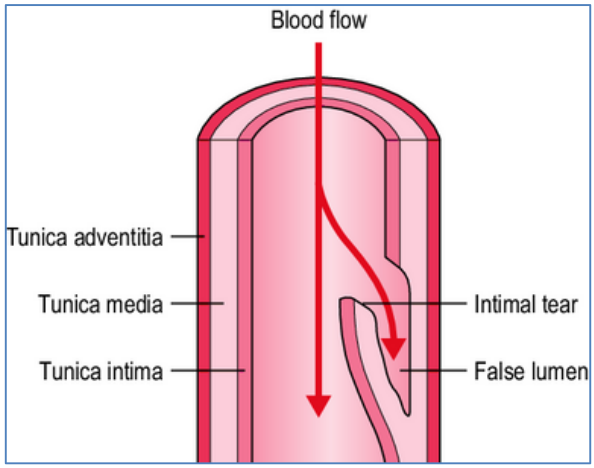
- o **Eg: Dissections:**
  - Blood builds up between the layers of the wall & eventually press the vessel closed)



Adapted from McKeeson Health Solutions 2002

PHYSIOLOGY OF HYPERTENSION

HYPERTENSION:

W hat is Hypertension?:

- Consistent **Diastolic of +90mmHg** **AND/OR**
- Consistent **Systolic of +140mmHg**.

General Info:

- **Is a Risk Factor *For*:**
  - Coronary Artery Disease
  - Stroke Heart Failure
  - Renal Failure
  - Peripheral Vascular Disease
  -
- *Usually Asymptomatic* – many don't know they have it.
- **Often Misdiagnosed Due To:**

Factor	Effect on BP reading
Cuff – too wide/ long	lower than actual
Cuff - too narrow/short	greater than normal
Arm - above heart	lower than normal
Arm - below heart	greater than normal
Arm - unsupported	greater than normal
Respiration rate	Lower during inspiration
"White coat" phenomenon	much greater than normal
smoking/caffeine/activity 30 min. prior to reading	greater than normal

- **Classifications (In Adults):**
  - **Different Classes Based on BP Ranges:**

Category	Systolic BP	Diastolic BP	% Population
Normal	<130	<85	83
Pre-Hypertensive	130-139	85-89	
Stage 1 Hypertension	140-159	90-99	13.5
Stage 2 Hypertension	160-179	100-109	2
Stage 3 Hypertension	180-209	110-119	
Stage 4 Hypertension	≥210	≥120	1

2 Types of Hypertension:

- (Based on Aetiology.)
- **1- Primary (Essential) Hypertension:**
  - 90-95% of cases
  - No specific cause.
  - But Related to:
    - Obesity
    - ↑Cholesterol
    - Atherosclerosis
    - ↑Salt Diet
    - Diabetes
    - Stress
    - Family History
    - Smoking
  - **Diastolic Hypertension:**
    - *Elevated Diastolic Pressure*
    - *Relatively Normal Systolic (or slightly elevated)*
    - Mostly Middle-Aged Men
  - **Isolated Systolic Hypertension:**
    - *Elevated Systolic Pressure*
    - *Normal Diastolic Pressure*
    - Ie: High Pulse Pressure

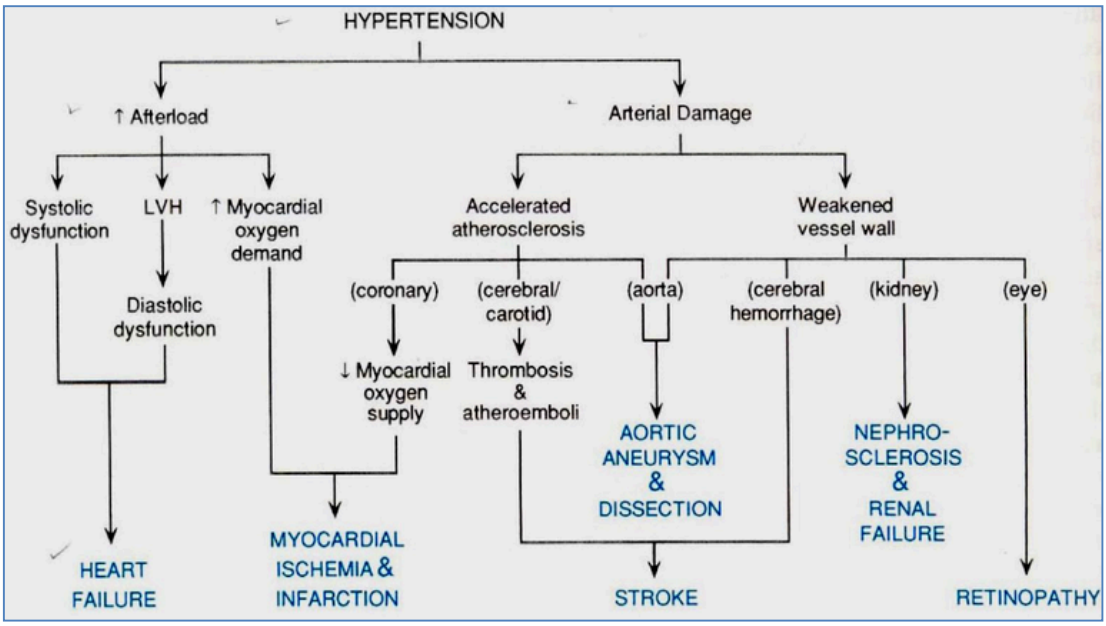
    In Older Adults (60yrs+):
    - May be due to reduced compliance of the aorta with increasing age.

    In Younger Adults (17-25):
    - May be due to Overactive Sympathetic NS → ↑Cardiac Output
    - Or Congenitally Stiff/Narrow Aorta

- **2- Secondary (Inessential) Hypertension:**
  - 5-10% of cases
  - Secondary to Another Diseases – Eg:
    - Renal Disease.
    - Endocrine Disorders
    - Pregnancy (Pre-Eclampsia) – in 10% of pregnancies. (@ 20wks of gestation)
    - Other –, Cancers, Drugs, Alcohol

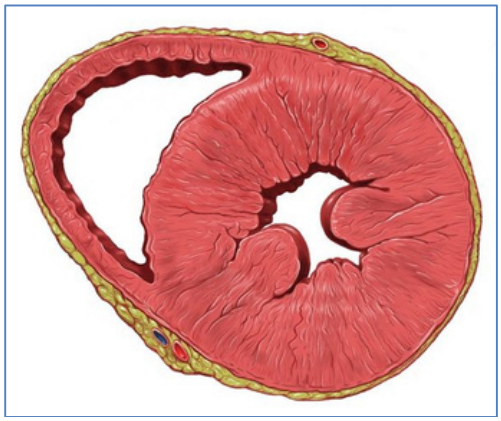
Organ Damage Caused By Hypertension:

- Relationship between *Degree* of hypertension & *Degree* of Complications.



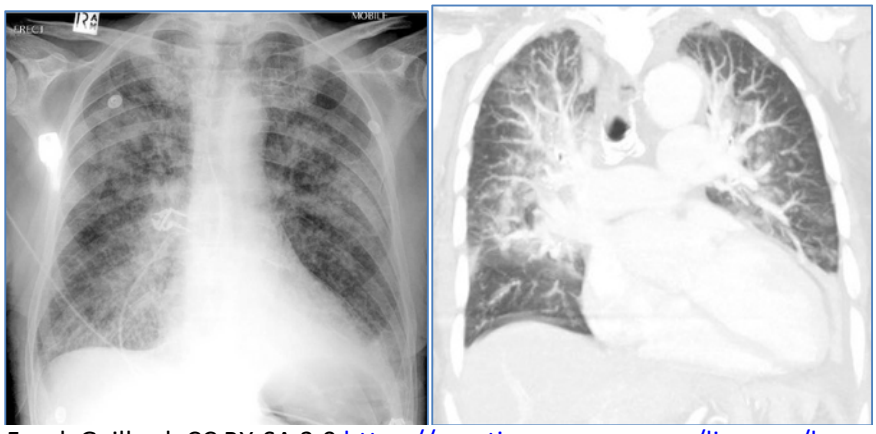
Source: Unattributable.

- **Heart:**
  - **Increased Afterload:**
    - § ↑ Workload of Heart → ↑Afterload → Pumps Harder → Hypertrophy → Failure
  - **L-Vent. Hypertrophy:**
    - § To compensate for higher workload
    - § → Compromised L-Ventricular Volume → ↓Stroke Volume →↓Cardiac Output



Patrick J. Lynch, medical illustrator, CC BY 2.5 <https://creativecommons.org/licenses/by/2.5

- **Lungs:**
  - o **Pulmonary Congestion:**
  - o Backing up of blood in Pulmonary Circuit.
  - o **Why:** ↑BP = ↑Aortic-BP = ↑Afterload = ↓SV = ↑ESV = ↓Pulmonary Blood Flow



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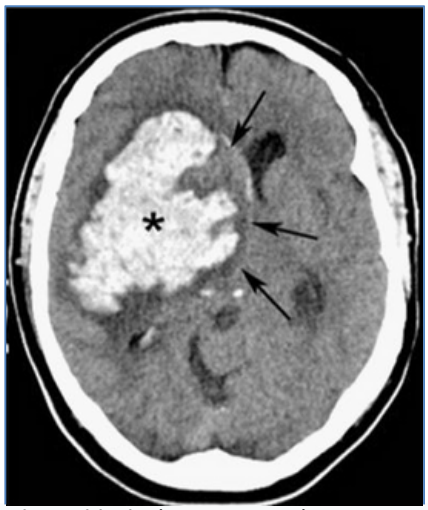
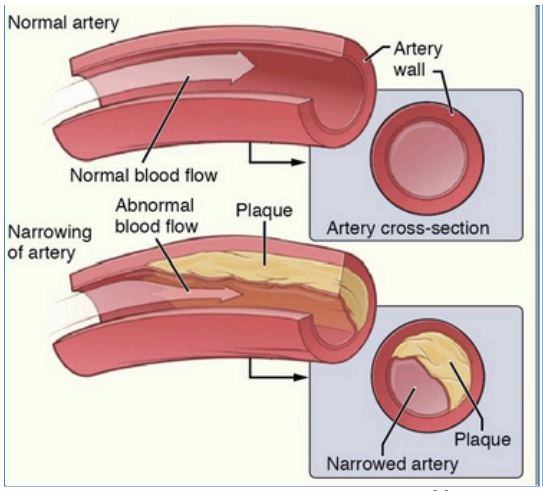
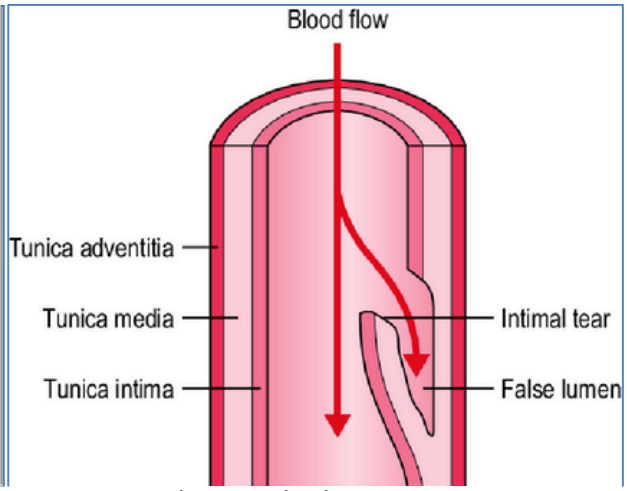
- **CerebroVascular:**
  - o Stroke – Typically *Intracerebral Haemorrhage*
  - o Rupture of Artery/Arterioles in brain



Shazia Mirza and Sankalp Gokhale (2016-07-25). Neuroimaging in Acute Stroke

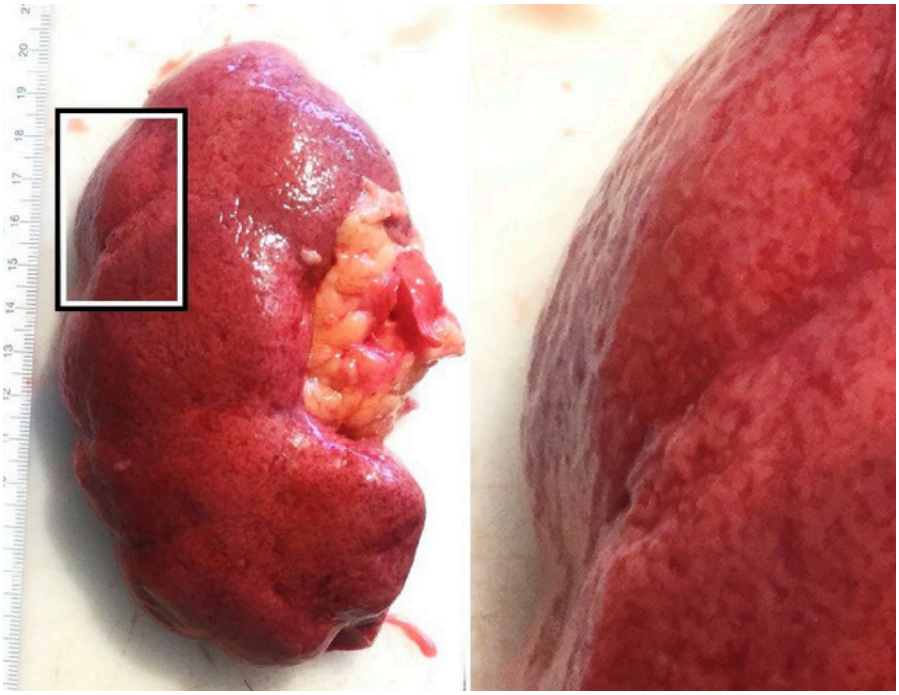
- **Aorta/Peripheral Vascular:**
  - o Arterial Mechanical Damage (Eg: Aneurysms/Dissecting Aneurysms)
  - o Accelerated Atherosclerosis





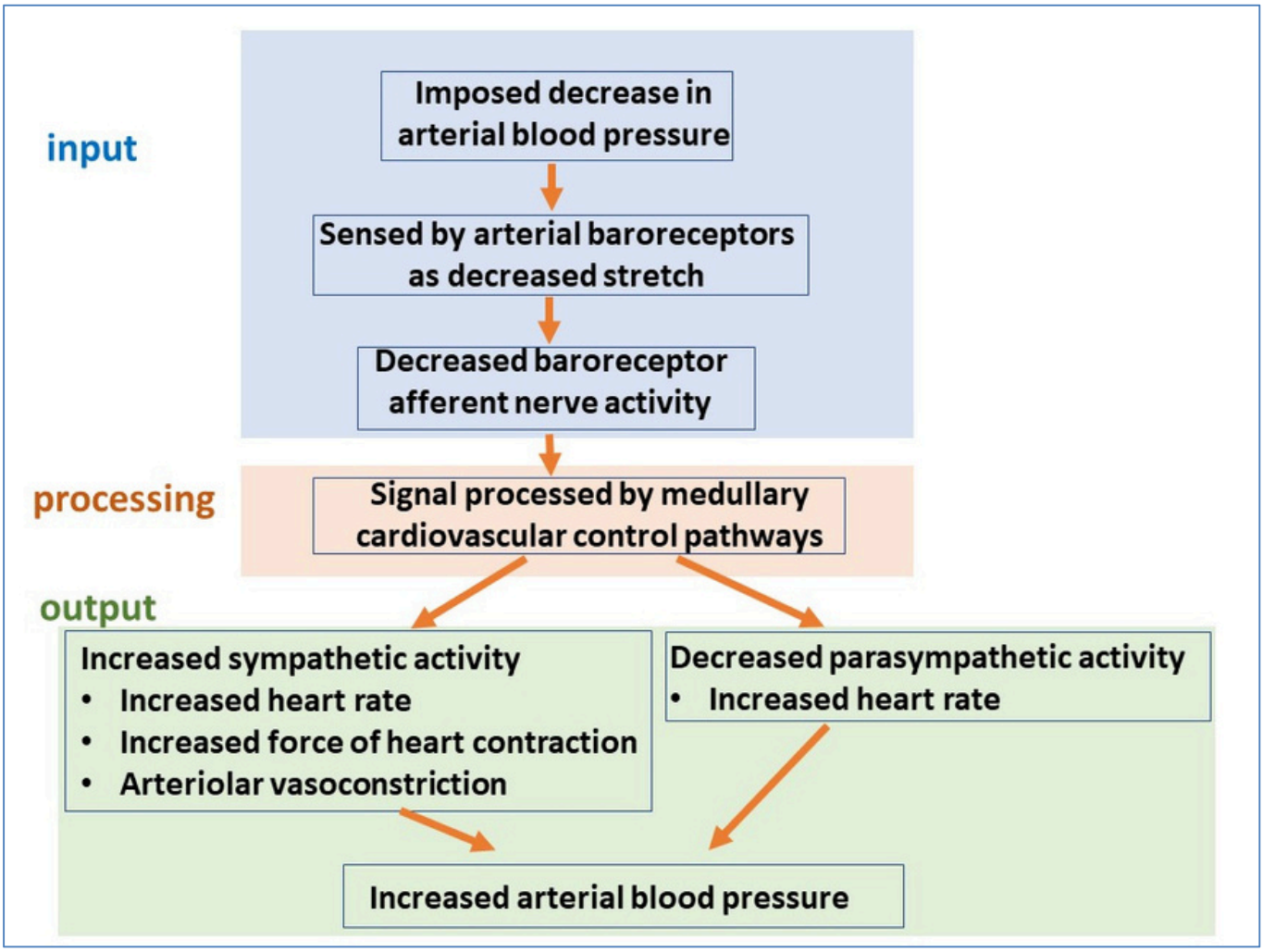
1. CNX OpenStax, CC BY 4.0 <https://creativecommons.org/licenses/by/4.0>, via Wikimedia Commons
2. Adapted from McKeeson Health Solutions 2002

- **Kidneys:**
  - o Nephrosclerosis – (hardening of kidney blood vessels)
  - o Renal Failure

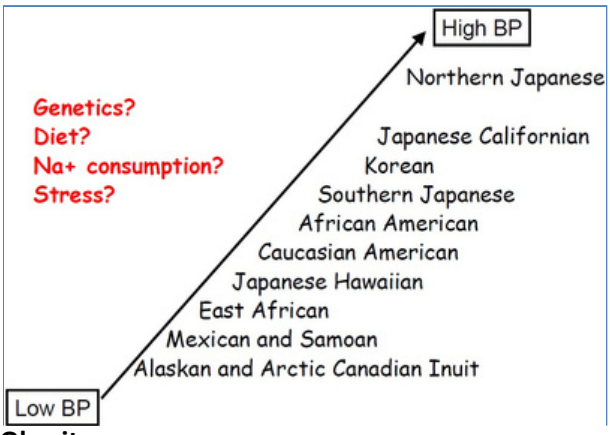


Mikael Häggström, CC0, via Wikimedia Commons;
https://commons.wikimedia.org/wiki/File:Gross_pathology_of_nephrosclerosis.jpg

- **Short-Term Physiological Control of BP:**
  - **The Baroreceptor Reflex:**

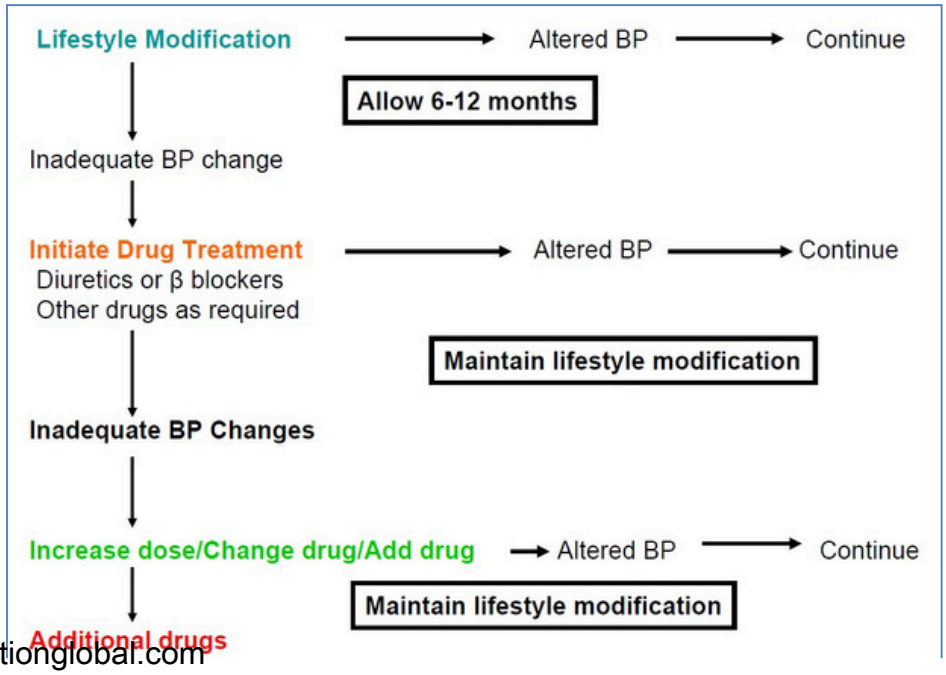


- **Risk Factors *Of* Hypertension:**
  - **Age:**
    - § BP normally increases with age.
      - Baby: 50/40
      - Child: 100/60
      - Adult: 120/80
      - Aged: 150/85 (quite normal)
    - § Due to Loss of Elasticity of Blood Vessels with age - Compliance↓.
    - § & Atherosclerosis
  - **Race:**

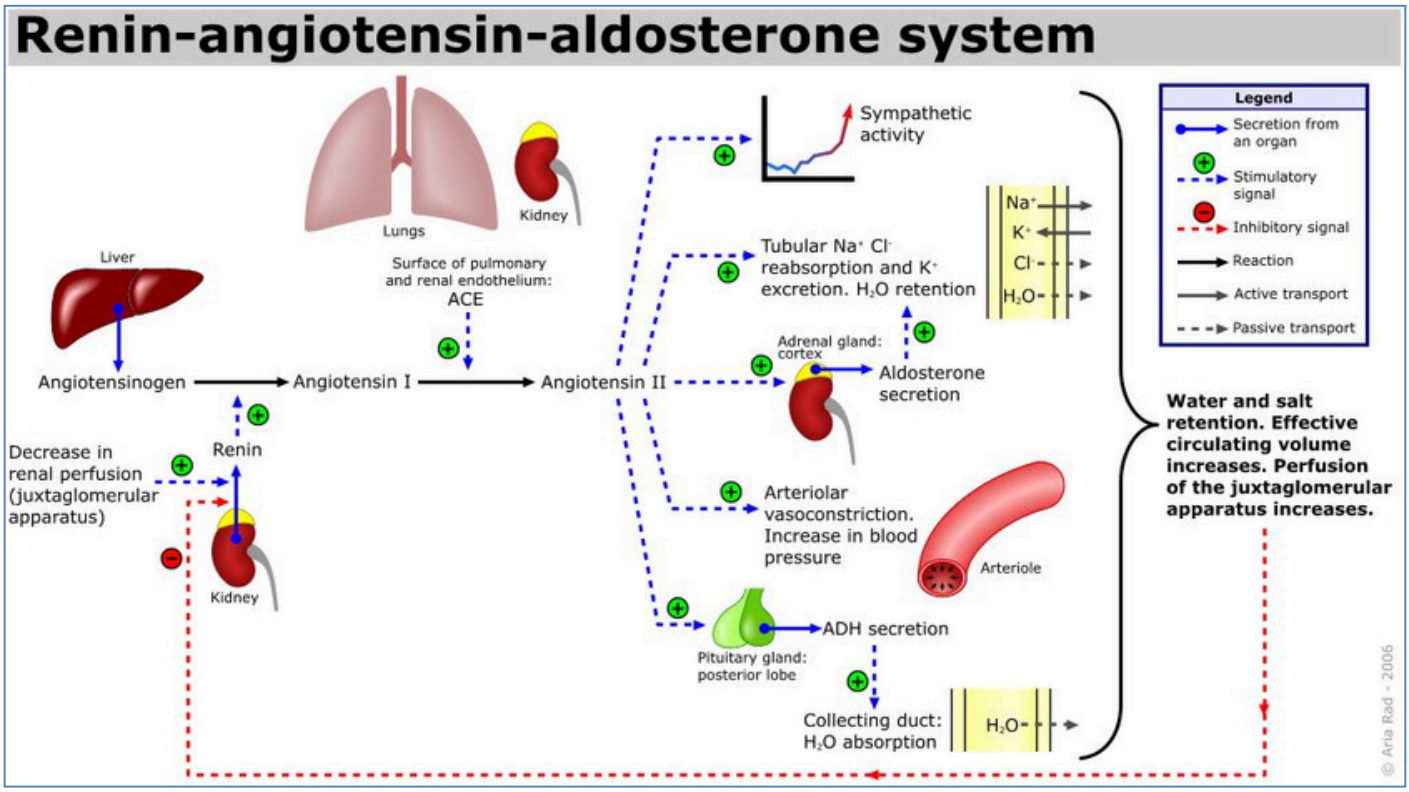


  - **Obesity:**
    - § Fatty Diet → Atherosclerosis
    - § Body Fat → kms more vessels → ↑Peripheral Resistance → Hypertension
    - § Physical Weight of fat – may impede venous return
    - § Kidney Dysfunction → Loss of long-term BP (Blood Volume) Control.
  - **Excess Na+ Intake:**
    - § **If Normal Kidney Function:**
      - Na+ intake → Slight BP increase (due to fluid retention)
      - But *Excess* Na+ & H2O excreted by kidneys → BP returns to normal.
    - § **If Impaired Kidney Function:**
      - Na+ intake → Larger BP increase...
      - Because *Excess* Na+ & H2O *Not* excreted by kidneys (less efficiently)

- **Basic Hypertension Treatment Plan:**



- **AntiHypertensive Drug Mechanisms:**
  - **Diuretics:**
    - Increases urination → ↓Blood Volume
    - Aim = To reduce workload on heart by reducing preload
  - **Sympatholytics:**
    - Reduces Sympathetic Activity (Prevents ↑HR/↑Contractility = Decrease in CO)
    - Eg: 'Beta-Blockers'.
  - **Vasodilators:**
    - Reduce Peripheral Resistance
    - → Reduce Afterload
    - → Reduce Workload on Heart.
  - **Renin-Angiotensin Antagonists (ACE Inhibitors):**
    - Decreases effects of Renin-Angiotensin System:
      - Decreases Sympathetic Drive
      - Decreases Vasoconstriction
      - Decreases Fluid Retention
      - Decreases Preload
      - Decreases Afterload



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PHYSIOLOGY OF SHOCK

SHOCK:

W hat is Shock?:

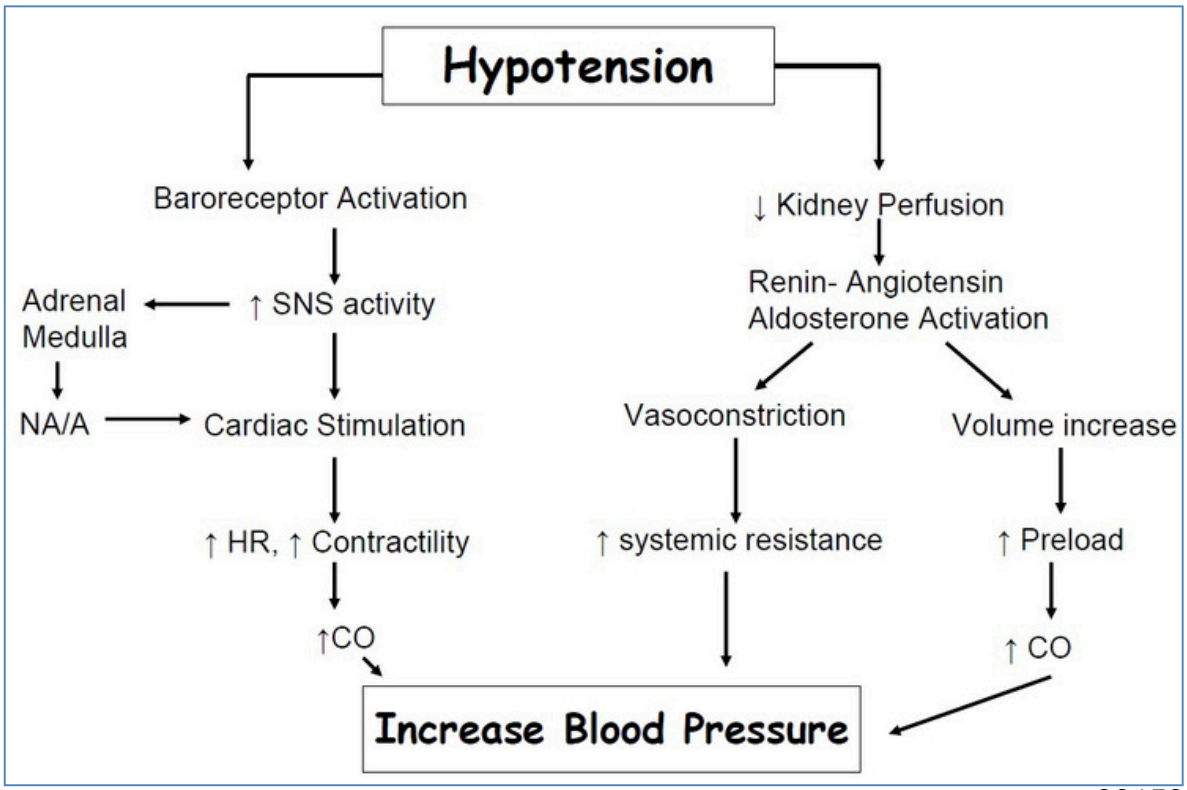
- Profound Haemodynamic/Metabolic Disorder due to Inadequate Blood Flow & O2 Delivery.

Common Causes of Shock:

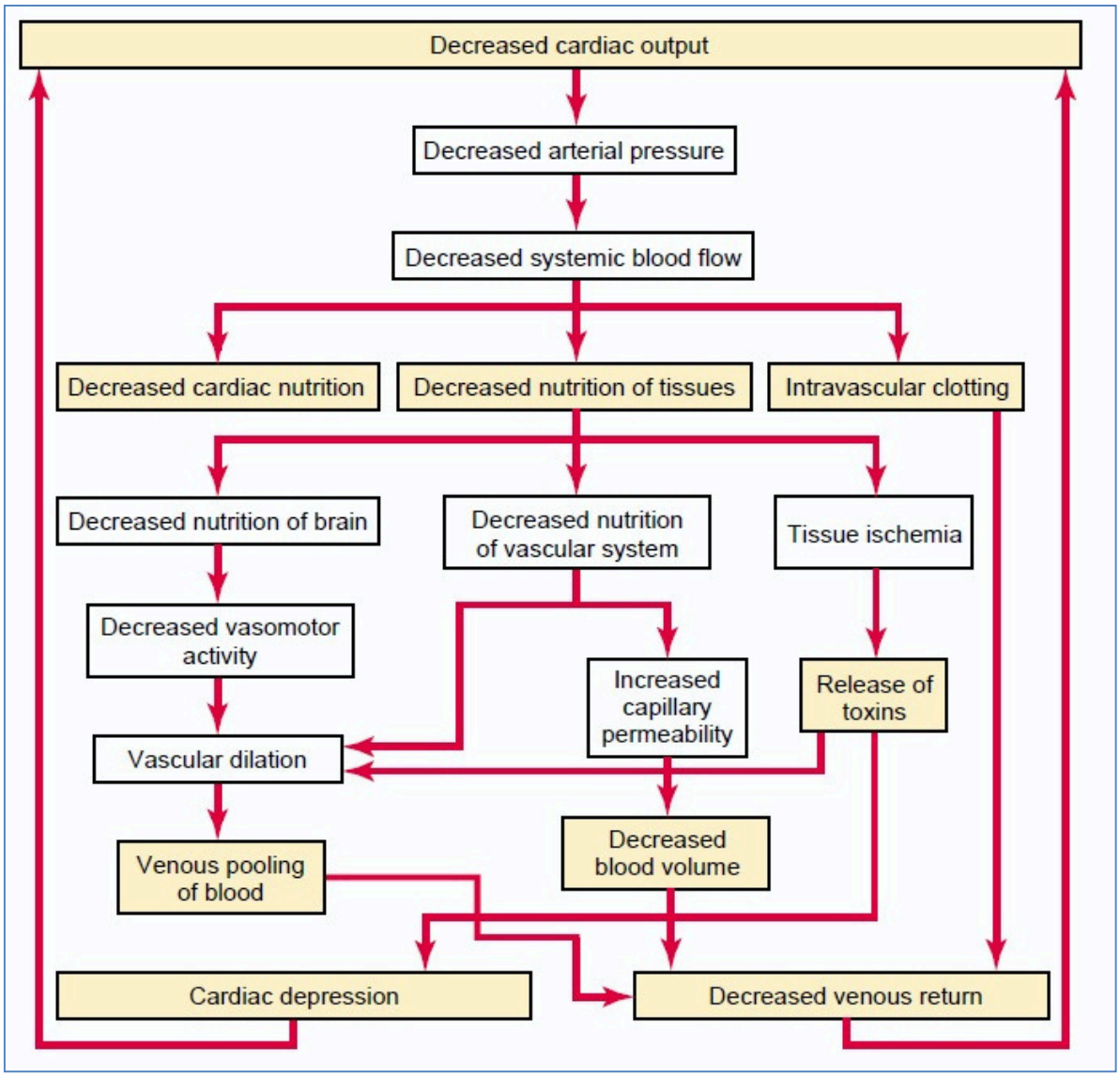
- **Hypovolemic Change:**
  - Severe Dehydration
  - Haemorrhage
- **Cardiogenic Change:**
  - Heart Failure (heart isn't getting enough blood out)
  - ↓Venous Return
- **Distributive Alteration:**
  - Excessive metabolism – Ie: Even a normal CO is inadequate.
  - Abnormal Perfusion Patterns – Ie: Most of CO perfuses tissues other than those in need.
  - Neurogenic Shock – Ie: Sudden loss of Vasomotor Tone → Massive VenoDilation.
  - Anaphylactic Shock – Drastic Decrease in CO & BP due to Allergic Reaction
  - Septic Shock – Disseminated bacterial infection in Body → Extensive Tissue Damage.

3 Stages of Shock:

- **1- Non-Progressive:**
  - Stable, not self-perpetuating.
  - **Symptoms:**
    - Hypotension (Low BP)
    - Tachycardia (High HR – body's attempt to compensate for poor perfusion)
    - Tachypnoea (High Breathing-Rate – Phrenic Nerve Stimulation – Diaphragm)
    - Oliguria (Low Urine Production by Kidney)
    - Clammy Skin
    - Chills
    - Restlessness
    - Altered Consciousness
    - Allergy symptoms (if anaphylaxis)
  - The Body's Compensatory Mechanisms (below) will prevail without intervention.
    - Aim to increase BP:

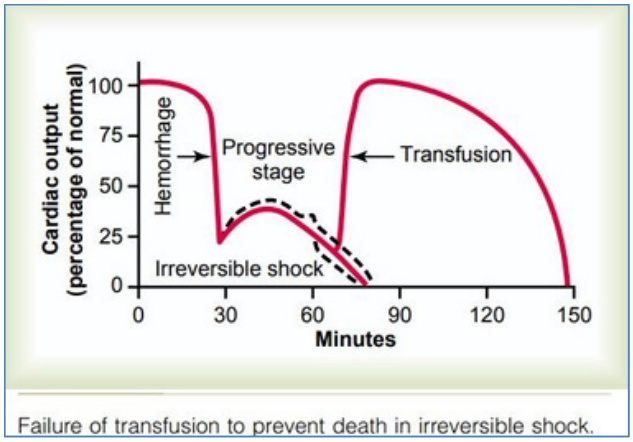


- **2- Progressive Stage:**
  - Unstable, vicious cycle of Cardiovascular Deterioration – Self-Perpetuating.
  - **Compensatory Mechanisms are insufficient to raise BP**.
  - Perfusion continues to fall → Organs become *more Ischemic* (Incl: Heart → Failure)
    - Cardiac Depression (due to O2 Deficit to Heart)
    - Vasomotor Failure (due to O2 Deficit to Brain)
    - "Sludged Blood" (Viscosity ↑. – Harder to move)
    - Increased Capillary Permeability
  - **Symptoms:**
    - Beginning of organ failure
    - Severely Altered Consciousness
    - Marked *Bradycardia* (initially tachycardic – but now the body is giving up)
    - Tachypnea (Fast Breathing) with Dyspnea (No breathing)
    - Cold, lifeless skin
    - Acidosis - (CO_2 equation affected)
  - **Treatment:**
    - Identify & Remove Causative Agents
    - Volume Replacement for Hypovolemia
    - If Septic Shock: Antibiotics
    - Sympathomimetric Drugs: If Neurogenic Shock (loss of vasomotor tone -vasodilation)
  - **Fatal if untreated**.



Abdel-Sater, Khaled. (2011). Physiological Positive Feedback Mechanisms. nwpii.com/ajbms. 3. 10.5099/aj110200145.

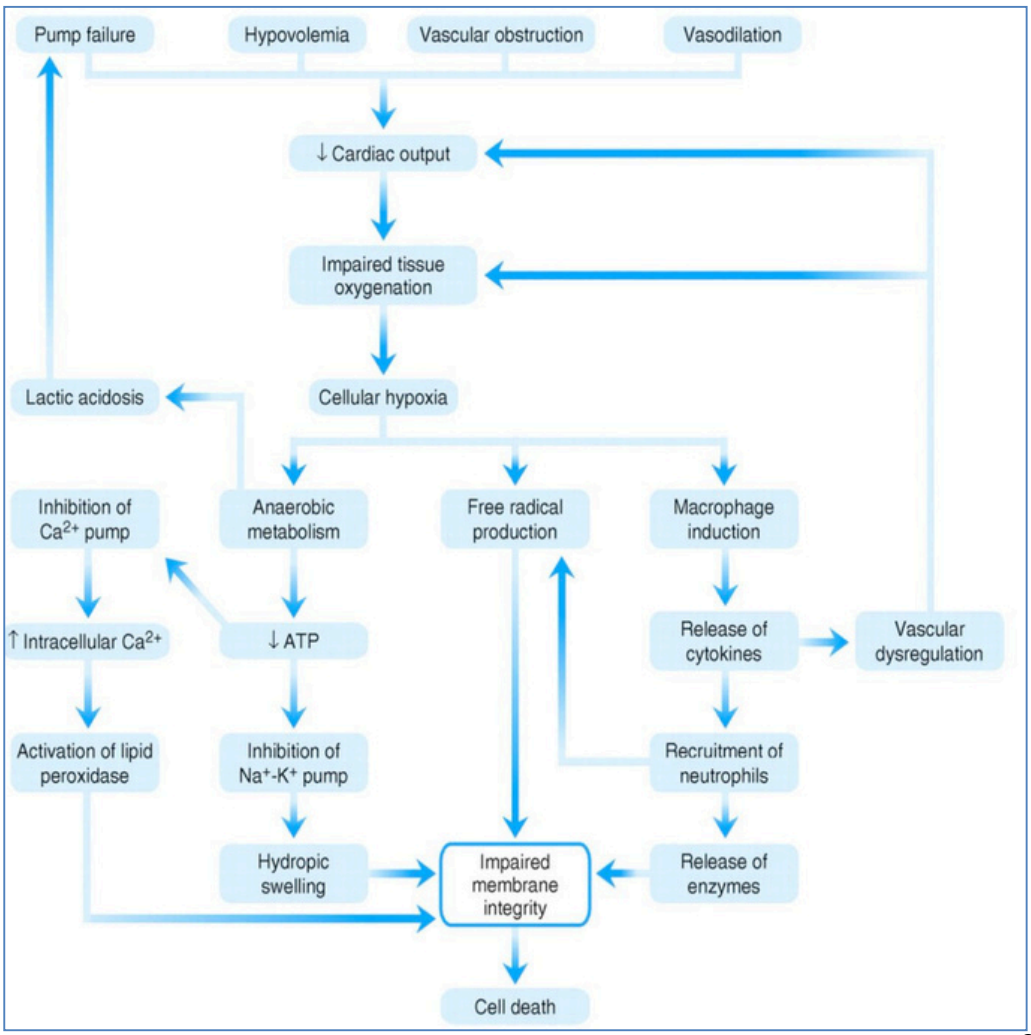
- **3- Irreversible Stage:**
  - o Advanced stage where the body is irrecoverable.
  - o Usually any form of therapy is ineffective.
    - § Eg: Transfusion is ineffective because the tissue/organ damage is too advanced.
  - o **Symptoms:**
    - § Organ Dysfunction (Renal/Cardiac/Pulmonary/CNS)
    - § Renal Failure Heart Failure
    - § Severely compromised CO & BP Worsening Acidosis
    - § Ischaemic Cell Death Coma
    - §
    - §
    - §



Failure of transfusion to prevent death in irreversible shock.

Shock-Induced Cell Death

- Self-Perpetuating Cascade



PHYSIOLOGY OF MYOCARDIAL ISCHAEMIA / ISCHEMIA

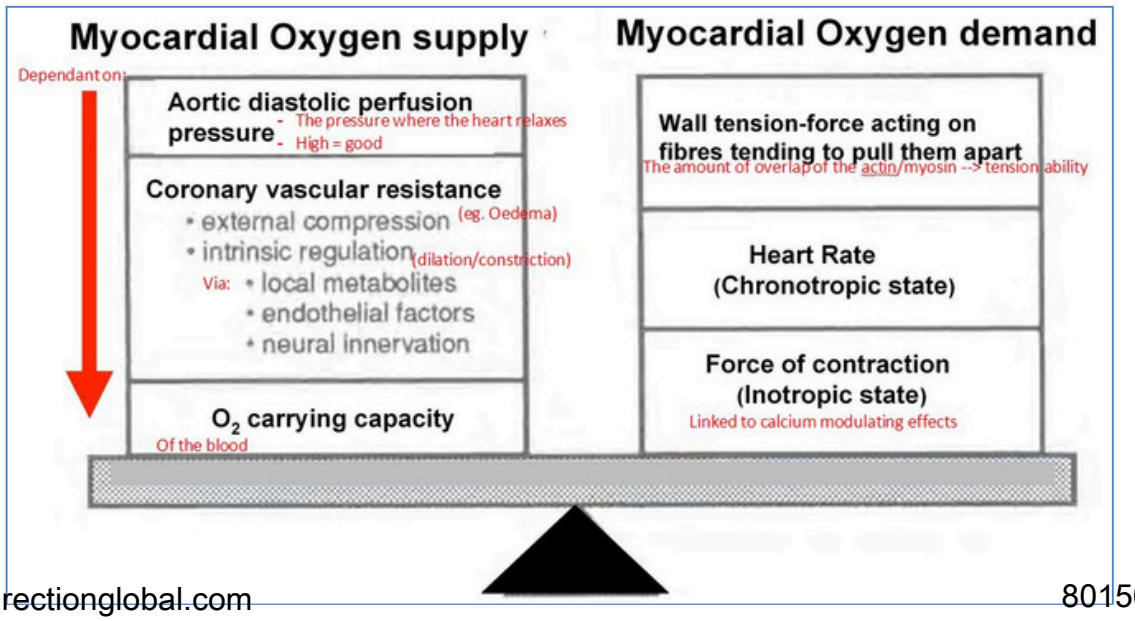
MYOCARDIAL ISCHAEMIA

Get Direction GLOBAL

W hat is 'Ischaem ia':

- = *Restraint of Blood* (Ie: Insufficient Blood)
- Leads to *Imbalance* Between Oxygen Supply & Demand.
- **Oxygen Supply – Increased By:**
  - o **↑Coronary Blood Flow:**
    - § **↑Aortic, Diastolic Perfusion Pressure:**
      - Aortic Pressure During L-Ventricular Diastole
      - If High → ↑Coronary Perfusion
      - Influenced by:
        - o Hypotension
        - o Aortic Regurgitation
    - § **↓Coronary Vascular Resistance:**
      - Resistance to Coronary Blood Flow
      - Depends on Vascular Diameter...
      - Influenced by:
        - o External Compression (Eg: Oedema)
        - o Intrinsic Regulation (Dilation/Constriction).
        - o Metabolites
        - o Neural
  - o **& ↑O2-Carrying Capacity of Blood:**
    - § Influenced By:
      - Hb Saturation
      - Hb Levels (Anaemia)
      - Blood pH
      - CO Poisoning
      - Lung Disease

- **Oxygen Demand – Increased By:**
  - o **↑Wall-Tension Force:**
    - § **↑Preload – (Degree of Stretch of Myocardium):**
      - The degree of *Stretching* of the Heart Muscle during Ventricular Diastole.
    - § **↑Afterload – (Back Pressure Exerted by Arterial Blood):**
      - The tension needed by Ventricular Contraction to *Open Semilunar Valve.*
  - o **↑Heart Rate (Chronotropic State)**
  - o **↑Force of Contraction (Inotropic State)**

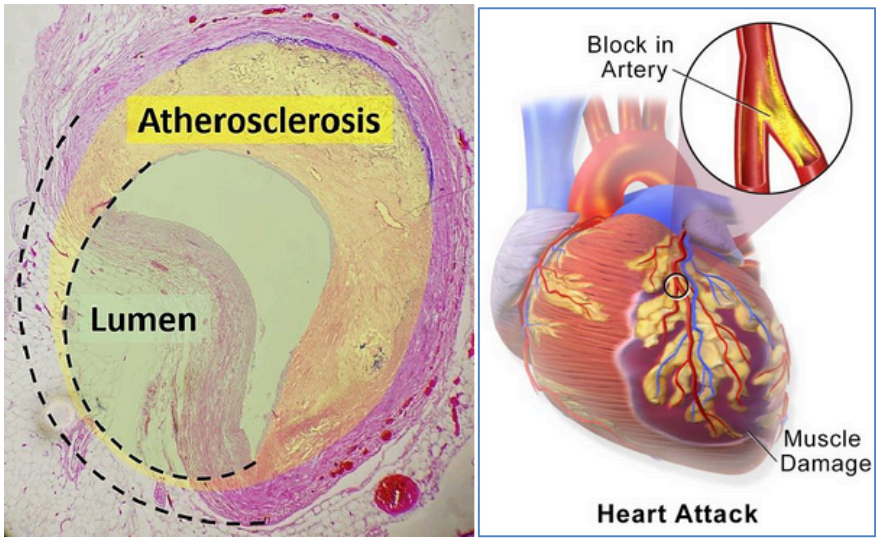


***Ischaemia Vs. Hypoxia Vs. Infarction:**

- **Ischaemia:** A '*FLOW' Limitation*, Typically due to Coronary Artery Stenosis (Narrowing)
- **Hypoxia:** An '*O2' Limitation,*Typically due to High-Altitude/Respiratory Insufficiency/etc.
- **Infarction:** *Irreversible Cell-DEATH*, Typically due to sustained Ischaemia.
- Note: Ischaemia can lead to Hypoxia & Infarction.

M yocardial Ischaem ia:

- Largest Cause of Deaths (50% of all deaths) in Western Society
- Mostly Attributed to ↓Coronary Blood Flow – Due to Plaque/Thrombosis.
- **Regional Ischaemia:**
  - o Ischaemia Confined to Specific *Region* of Heart.
  - o Due to Plaque/Thrombosis
- **Global Ischaemia (Rare):**
  - o Ischaemia of *Entire* Heart
  - o Due to Severe Hypotension/Aortic Aneurysm/Open-Heart-Surgery



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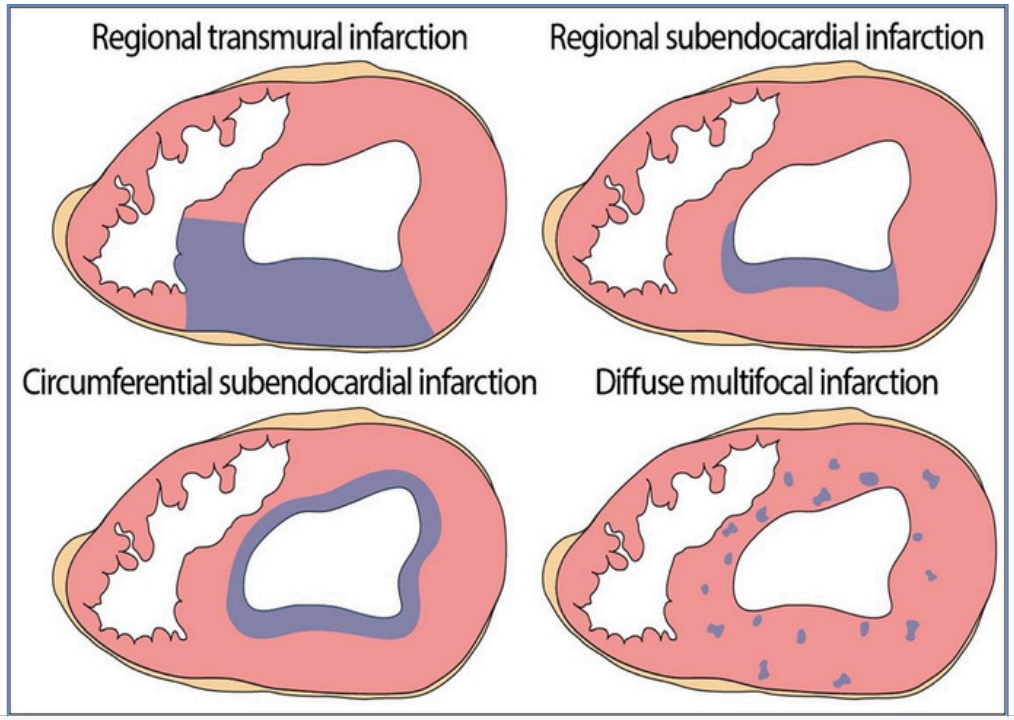
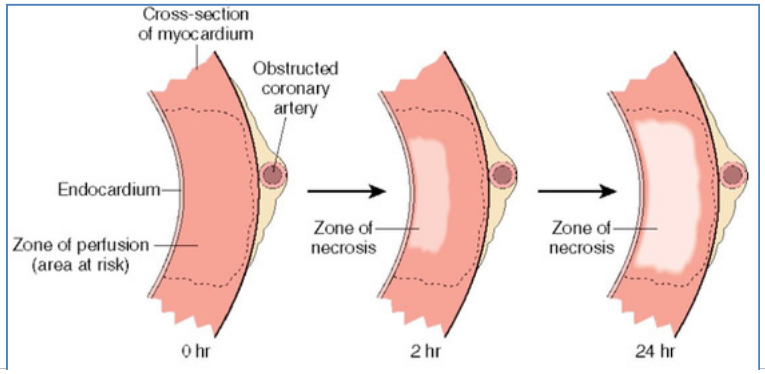


(2019). "Diagnosis of myocardial infarction at autopsy: AECVP reappraisal in the light of the current clinical classification". *Virchows Archiv*.

What Happens During Myocardial Ischaemia:

- **Myocardial Damage:**
  - o Inner-Myocardium will become Ischaemic first, then progress Outwards.
  - o (Same with necrosis/infarction)



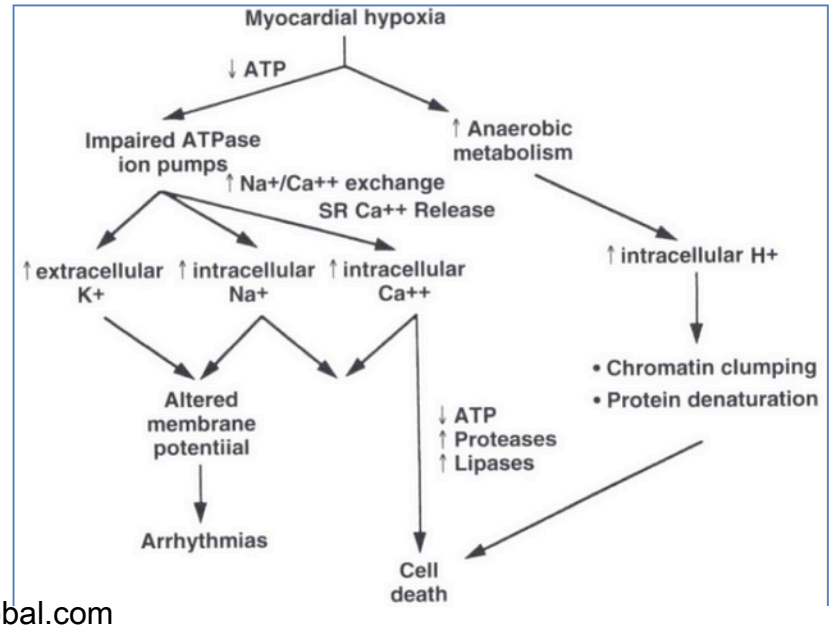
Adapted from *Schoen FJ, Mitchell, RN: The heart. In Kumar V, et al, editors:* Robbins and Cotran pathologic basis of disease.

- **Metabolic Changes – (Aerobic → Anaerobic):**
  - o ↑Lactate (Anaerobic Metabolism), ↓pH
  - o ↓ATP, ↑ADP, ↑Pi
  - o ↓Glycogen
- **Pain:**
  - o Nociceptor (pain receptor) Activation → Angina Pain
- **Acute Ischaemic Attack:**
  - o SNS & PNS Stimulation → Tachycardia, Sweating, Nausea.....
- **Reversible Cell Injury:**
  - o ↓Blood-Flow → ↓Myocardial Relaxation (diastolic) → Stiffening of L-Ventricle → ↑LVDP

Reperfusion Injury:

- o Cell Damage that occurs When Blood Supply is Restored (after being stopped)
- o Due to inflammation and oxidative damage through the induction of oxidative stress.

- **Pulmonary Congestion:**
  - o Stiffening of L-Ventricle & ↑LVDP → ↑Pulmonary Vascular Pressure
    - § → Pulmonary Congestion
    - § → Shortness of Breath
- **Ventricular Arrhythmias:**
  - o **Due to Myocyte Ion-Disturbances:**
    - § ↑ Extracellular K+
    - § ↑ Intracellular Na+
    - § ↑ Intracellular Ca+ ("Calcium-Loading") – If Ischaemia is Prolonged → Irreversible Damage
  - o → Alters Conduction Patterns of the Heart → Arrhythmias



Overview of Clinical Presentations of Myocardial Ischaemia:

- **Ischaemic Heart Failure:**
- o Weakness of Heart Muscle → Difficulty Breathing + Peripheral Oedema

Angina Pectoris:

o Substernal/Precordial Chest Pain – Due to Myocardial Ischaemia → No Cell Necrosis

o Pain Usually lasts up to 15min.

o **3 Subtypes:**

§ **Stable Angina (Typical):**

- Angina-Pain During Exertion/Stress
- No Permanent Injury
- ST-Depression (Indicates Subendocardial Ischaemia)
- Treated with Vasodilators

§ **Variant Angina (Prinzmetal):**

- Angina-Pain *Unrelated* to Activity
- Due to Coronary Vascular Spasm
- ST-Elevation (Indicates Transmural Ischaemia)

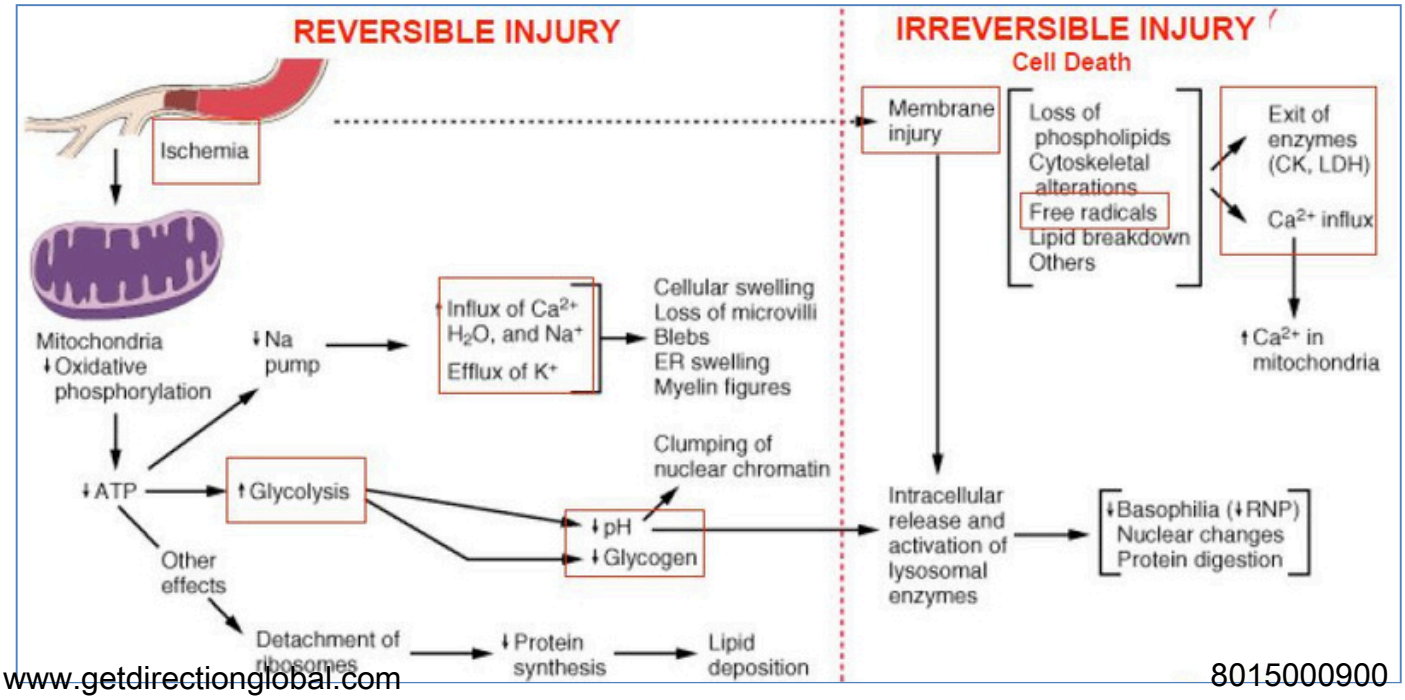
§ **Unstable Angina (Dangerous):**

- Occurs @ Rest – Prolonged Pain
- Increasing Frequency & Duration of Angina-Pain
- Due to unstable Atherosclerotic Plaque
- Can Lead to Myocardial Infarction (if untreated)

- **Silent Ischaemia:**
- o *No Pain*
- o Abnormal ECG (ST-Elevation)

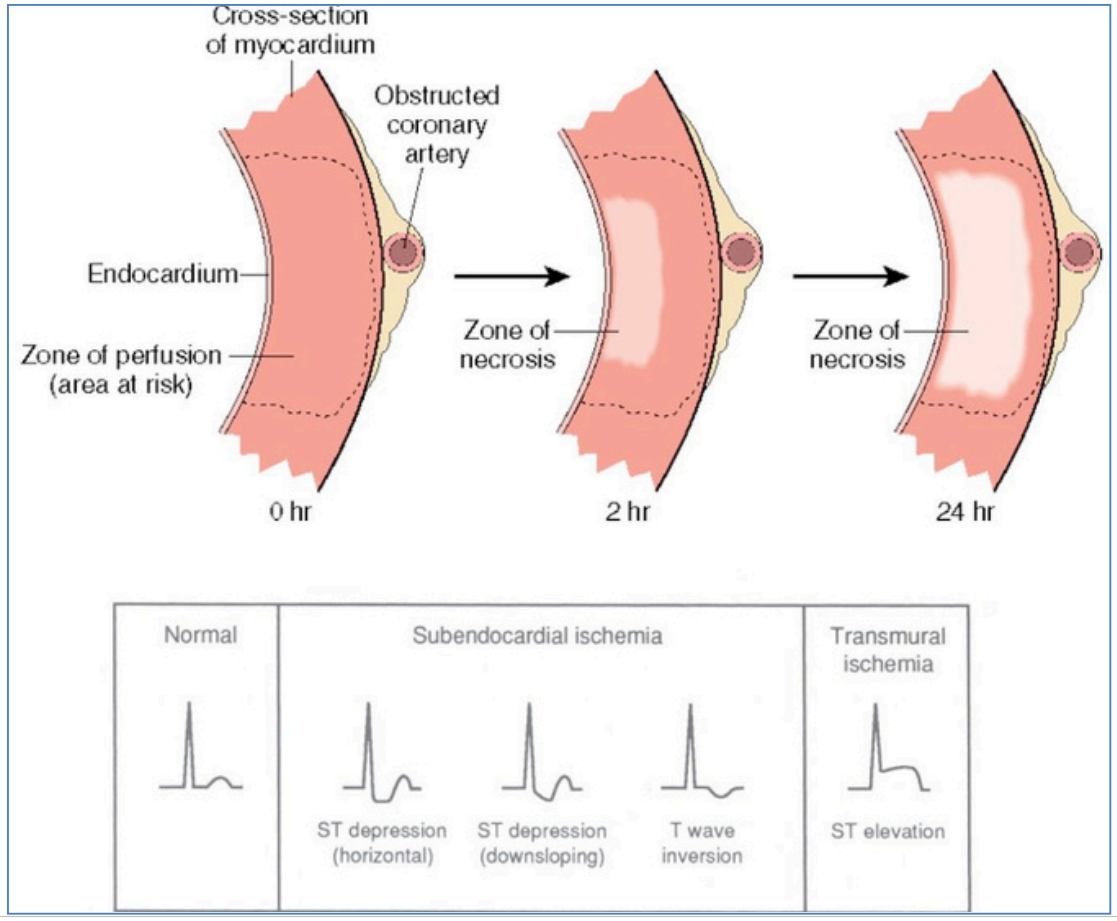
Prolonged Ischaem ic → Irreversible D am age → Leads to:

- **Ca+ Loading Within Cell:**
- o Ca+ Recycling Cycle (between Sarcoplasmic Reticulum, Sarcoplasm & Actin) Changes.
- o *Marks the transition between Reversible & Irreversible Damage.*
- **Heart Failure – Due to:**
- o Lethal Arrhythmias
- o ↑LVDP → Pulmonary Congestion → R-Heart Failure.
- **Infarction (Necrosis):**
- o Irreversible Cell Death – Due to Ischaemia/Acute Thrombus
- o Myocyte Membrane damage → Cell Enzymes/Proteins into Blood → Used as blood Markers:
- § Troponin I (Preferred)
- § Creatinine Kinase



ECG Changes Due to Ischaemia:

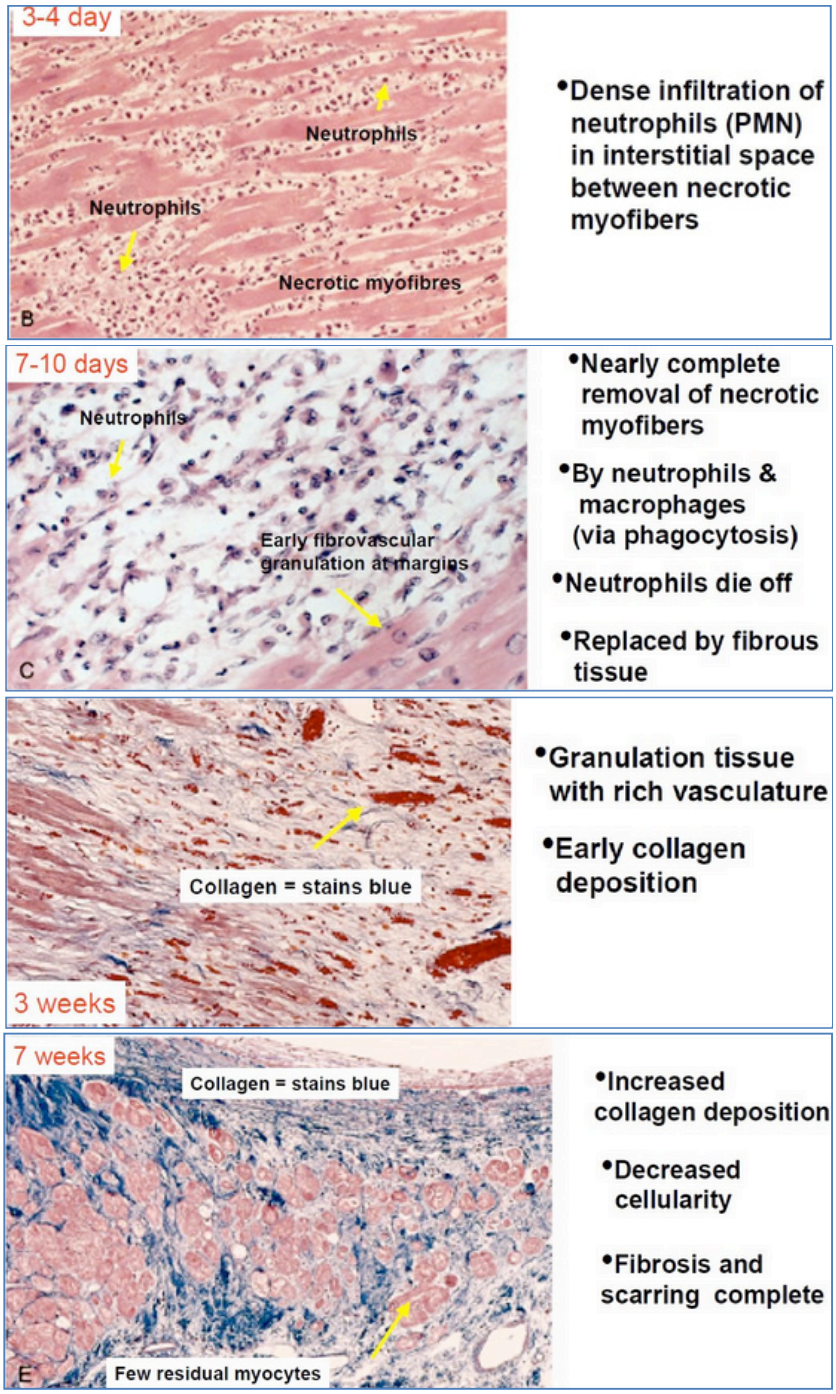
- **Normally:**
  - o QRS = Ventricular Depolarization
  - o T-Wave = Ventricular Repolarisation
    - § Note: Ventricular Repolarisation – Very sensitive to myocardial perfusion. (Ie: Lack of blood supply alters Ventricular Relaxation)
- **Subendocardial Ischaemia:**
  - o Poor Perfusion → Altered Ventricular Repolarisation →
    - § ST-*Depression*
    - § T-Wave Inversion
- **Transmural Ischaemia:**
  - o Full-thickness of the heart wall is damaged → Altered Ventricular Repolarisation →
    - § ST-*Elevation*

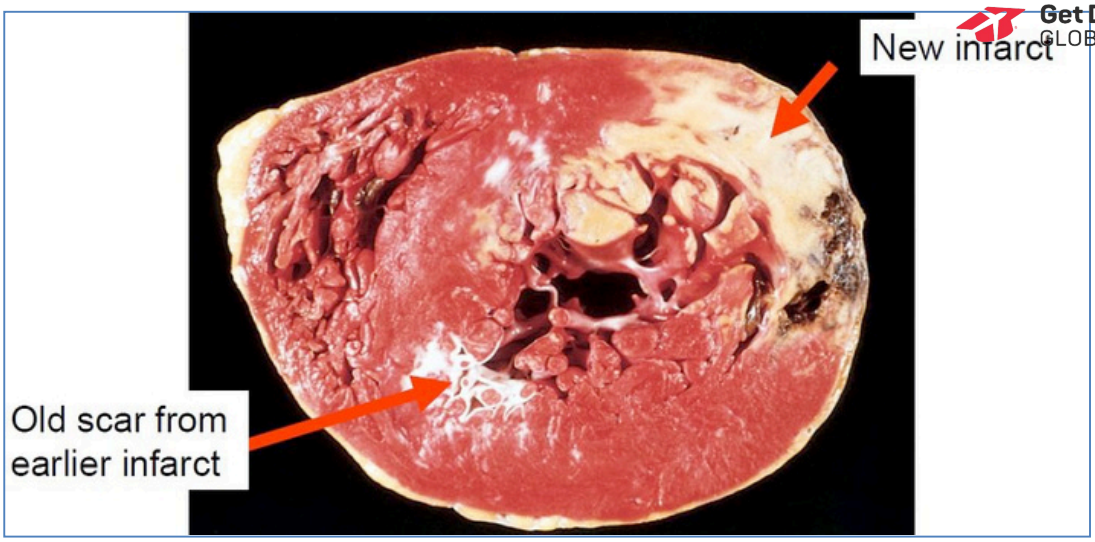


Adapted from *Schoen FJ, Mitchell, RN: The heart. In Kumar V, et al, editors:* Robbins and Cotran pathologic basis of disease.

M yocardial Infarction:

- *90% of Infarcts due to Thrombosis from Ruptured Atherosclerotic Plaque.
- **Diagnosis Requires 2 of the Following:**
  - o **History of Ischaemic-Related Chest Pain:**
    - § Eg: Angina
  - o **Changes on Sequential ECGs:**
    - § ST-Segment Elevation → Indicates ***Transmural Ischaemia*:**
      - Where the full-thickness of the heart wall is damaged.
      - Note: ST-Elevation isn't always due to MI.
  - o **Rise/Fall in Serum Cardiac Markers:**
    - § Spilt contents of dead cells → Blood
    - § Eg: Cardiac Troponin & Creatinine Kinase
- **Ensuing Inflammatory Response:**
  - o When Cells Die → Neutrophils Infiltrate Area → Attack/Decompose/Phagocytose Dead Cells
  - o After Inflammatory Response → Fibrosed Scar Tissue (Such Tissue in Heart is Non-Contractile*)





Contributed by Dr Shashider Venkatesh Murthy.

THE EFFECTS OF AGEING ON THE HEART:

THE AGEING HEART

Get Direction GLOBAL

W hat Happens in a Norm al Ageing Heart?:

- **Physical Changes:**
  - o Heart Dilation – (Lumen Size of L-Atrium & L-Ventricle Increases with Age.)
  - o Increased Capillary Density
  - o Valves become calcified – (Mitral Valve closes more slowly with age → ↑L-Vent. Filling Time)
  - o Fibrosis increases
  - o Arteries become less compliant
- **Histological Changes:**
  - o The number of myocytes decreases
  - o The remaining myocytes enlarge
  - o Heart Wall thickens to compensate for extra stress from stiffer blood vessels.
- **Functional Changes:**
  - o Decreased Heart Rate During Exercise
  - o Decreased Contractility
- **Physiologic Changes:**
  - o Myocardial metabolism decreases (Reduced mitochondrial metabolism)
  - o Altered Sarcoplasmic Reticulum function (Lower Ca+ in SR & Fewer Ca+ pumps/cell) → decreased contractility
- **Sensitivity Changes:**
  - o o β-Adrenergic Sensitivity Decreases
  - o Chemoreceptor Sensitivity Decreases
    - § (less Ca+ enters the cell → Max HR & Contractility decreases)
  - Baroreceptor Sensitivity Decreases
- **Conductivity Changes:**
  - o Conduction pathways become calcified
  - o Reduced Number of SinoAtrial Node Pacemaker-Cells → DECREASED HEART RATE
  - o Impaired Sinoatrial (Pacemaker) Function → Atrial Fibrillation, Arrhythmias

- **Note: These Changes = Normal = "Normal Ageing Myopathy"**

These Above Changes Make Old Age a Risk Factor For Heart Failure:

- **Incidence of Chronic Heart Failure Increases with Age...WHY?**
  - o **1-** The above changes may interact with each other → Heart Failure
    - § Eg: Decreased Myocytes (contractility) + Valve Calcification → ↓SV → Heart Failure
  - o **2-** The above changes may interact with an existing cardiovascular disease:
    - § Eg: Valvular Stenosis + Fibrosis + Less Compliant Arteries → ↓SV → Heart Failure
    - § Eg: Atherosclerosis + ↓Contractility → ↓Coronary Perfusion → Ischaemia → Heart Failure
    - § Eg: Hypertension + Calcified Valves + Less Compliant Arteries → ↓SV → Heart Failure
- **Ie: The normal physiological effects of ageing, even if healthy, increases the presence of many Heart-Failure Risk Factors**.

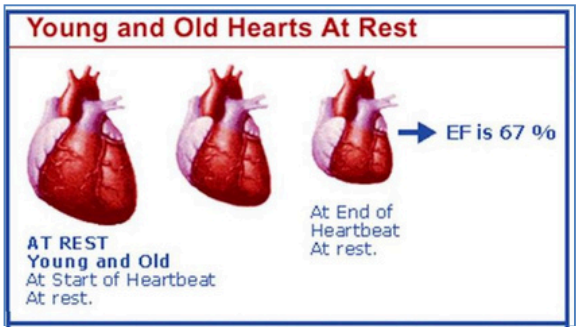
Healthy 20yr-old Vs. Healthy 80yr-old (At Rest)

- **Heart Rate (Resting) is 10% Lower in Old Heart:**
  - Older hearts have a 10% lower Resting Heart Rate than Young.

- **Stroke Volume (At Rest) is 10% Higher in Old Heart:**
  - Ie: An Old Heart pumps 10% more blood/beat than a Young Heart (At Rest), despite being a weaker pump.
  - ...**How?:**
    - Older Heart Compensates for its ↓Contractility by Dilating more during Diastole to Increase Ventricular Filling (Preload/End-Diastolic Volume) → ↑Stroke Volume.

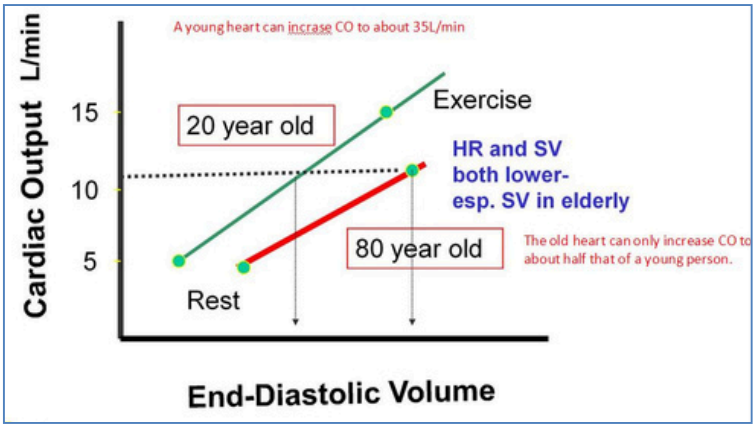
- **Same *Resting* Cardiac Output:**
  - Ie: An Old Heart pumps out the same amount of blood/min (at rest) as a Young Heart.
  - ...**How?:**
    - Older hearts have a 10% Higher Stroke Volume + but 10% Lower Heart Rate → Same Cardiac Output (At Rest) compensates for its ↓Contractility by Dilating more during Diastole to Increase Ventricular Filling (Preload/End-Diastolic Volume) → ↑Stroke Volume.
  - **However**, the older heart has a narrower *'Scope'* for Activity – Meaning it can only match a young heart's increase in Stroke Volume (during exercise) up until a point, after which the younger heart is superior.

- **Same *Resting* Ejection Fraction:**
  - Ie: An Old Heart has the same *'Ejection Fraction'* (≈67%) as a Young Heart.
    - Note: Ejection Fraction = The Percentage of The End Diastolic Volume Ejected Each Beat.
  - **However**, the older heart has a narrower *'Scope'* for Activity – Meaning it can only match a young heart's Increase in Ejection Fraction (during exercise) up until a point, after which the younger heart is superior.



Healthy 20yr-old Vs. Healthy 80yr-old (During Exercise):

- **Higher Preload/End-Diastolic Volume (During Exercise) in Older Heart:**
  - The older heart compensates for its ↓Contractility by Dilating more & Decreasing Heart Rate to Increase Filling Volume & Filling Time → ↑Preload
    - Increased Preload (End-Diastolic Volume) → ↑Stroke Volume.



- **Lower Max. Heart Rate (During Exercise) in Older Heart:**

Heart Rate During Exhaustive Exercise

Age (yrs)	20-29	30-39	40-49	50-59	60-69	70-79
Men	185	180	178	165	155	145
Women	182	176	169	165	155	145

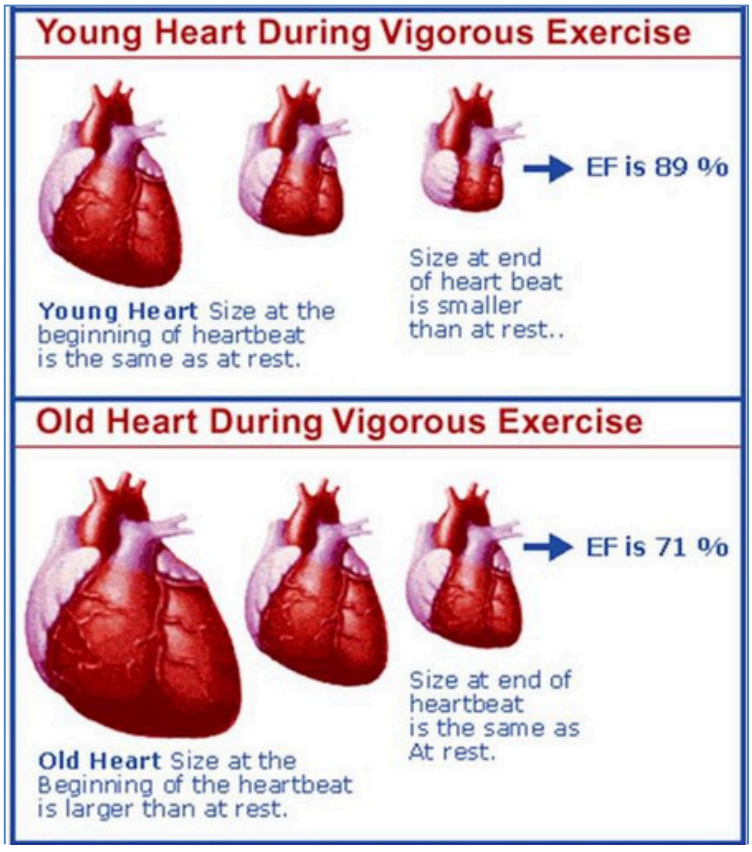
Max HR of 80 yr old is 25% lower than 20 yr old

- **Same Stroke Volume (During Exercise):**
  - o Due to Increased Preload/EDV via Dilation & ↓HR.

- **25% Lower Cardiac Output (During Exercise) in Older Heart:**
  - o Primarily Due to decreased heart rate. Note: SV stays same.
    - § (The young heart can increase CO from 5L/min @ rest to about 35L/min)
    - § (The old heart can only increase CO from 5L/min @ rest to about 15L/min)

- **Lower VO2max (Max O2 Consumption) in Older Person:**
  - o Old Person's VO2max is half that of a Young Person.
  - o **Due to:**
    - § Lower Muscle Mass (Ie: Less muscle uses less energy → ↓O2 Consumption)
    - § Changes in Muscle Metabolism (↓enzyme efficiency/manufacture etc.)
    - § Decreased Number of Mitochondria/Cell.

- **Lower Max. Ejection Fraction (During Exercise) in Older Heart:**
  - o Young heart can increase its ejection fraction from 67% → 89%.
    - § - by ↑Contraction & ↑Heart Rate.
  - o However, the Old heart can only increase its EF from 67% → 71%.
    - § - by Dilating.



Summary:

- **Young Heart:** In Exercise – its contractility is higher, so when the body requires a higher cardiac output, the heart contracts more than normal by balling up tighter in each contraction → decreasing End-Systolic Volum e → Increasing Stroke Volum e → Increasing Cardiac O utput.

- **Older Heart:** In Exercise – Its contractility is lower (Approx 60% lower than 20yr old heart – mostly due to sedentary lifestyle), so when the body requires a higher cardiac output, the heart compensates by dilating more to increase filling (End-Diastolic Volume) → Increasing Preload →Increasing Stroke Volume → Increasing Cardiac Output.

- **Note:** This compensatory mechanism of Dilating to increase L-Heart Pressures can lead to *Symptoms* of Heart Failure (Ie: Shortness of Breath, Loss of Pump Function & Pulmonary Oedema). However, this is not strictly Heart Failure, because Cardiac Output is not Severely Compromised.

Benefits of Aerobic Exercise on CardioVascular Ageing:

- **Huge Benefits:**
  - ↑Max O2 Consumption
  - ↑Ejection Fraction
  - ↑Contractility
  - (↑Contractility → Less need to Dilate for Increased Stroke Volume)
  - Less Dilation → ↓EDV & ↓LAP.
  - Less Arterial Stiffness.
- **Ie: It seems that a large part of CV-Ageing is Related to a Sedentary Lifestyle**.

Combating CV-Ageing with Pharmaco-Therapies:

Drugs that ↑Vascular Compliance
Drugs that reduce Cardiac Fibrosis
Drugs that reduce Ventricular Hypertrophy
Antioxidants – Prevents damage due to free radicals
Anti-Inflammatory Drugs – CV-Ageing has a small underlying inflammatory component.
(Plus Exercise)

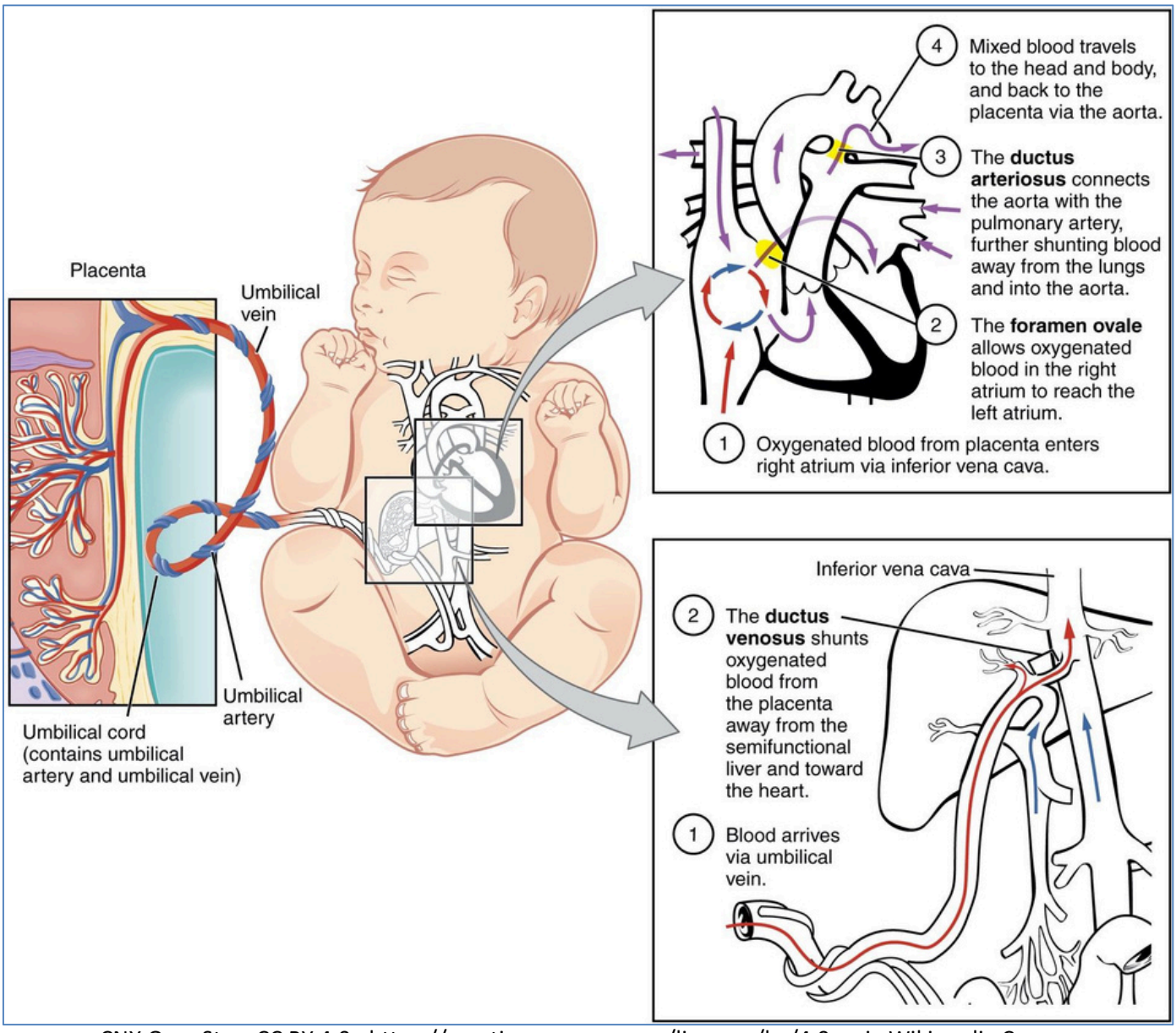
CARDIOVASCULAR PATHOLOGY:

Review of Foetal Circulation (Physiological Bypasses/Shunts):

- **Ductus Venosus**
  - o Shunts O2-Blood from Placental Vein → IVC →R-Atrium
  - o Bypasses the Liver
- **Foramen Ovale**
  - o Shunts O2-Blood from R-Atrium → L-Atrium.
  - o Bypasses the Lungs.
- **Ductus Arteriosus**
  - o Shunts O2-Blood from Pul-Artery → Aorta
  - o Bypasses the Lungs
- (**All of these "shunts" are should close after birth due to pressure changes)
  - o Note: The Foramen Ovale can take up to 6 months to close.

At Birth, the Pulmonary Vascular Resistance Falls Due to:

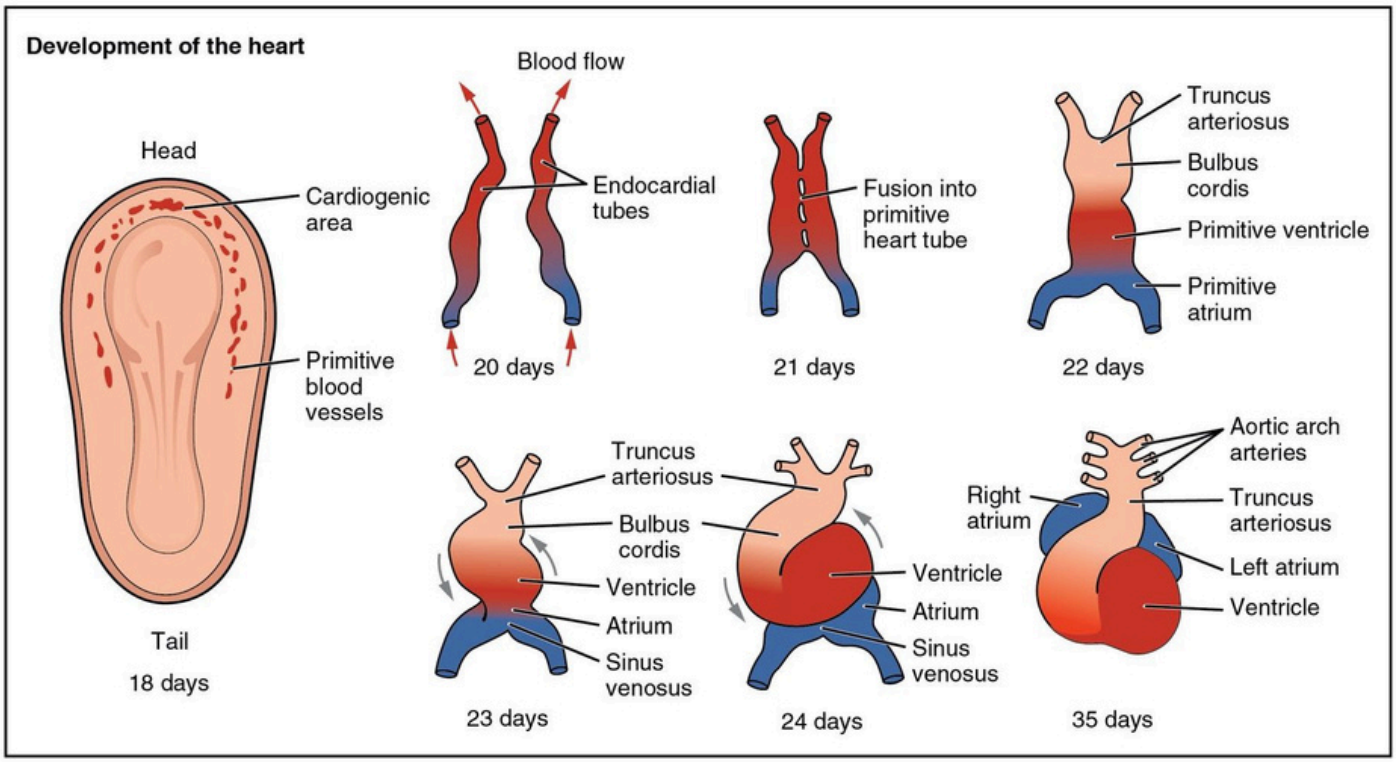
- **1- Mechanical of Lung Inflation** → Increased Radial Traction of Vessels
- **2- Vasodilation** – due to ↑Oxygen-Tension in the Lungs

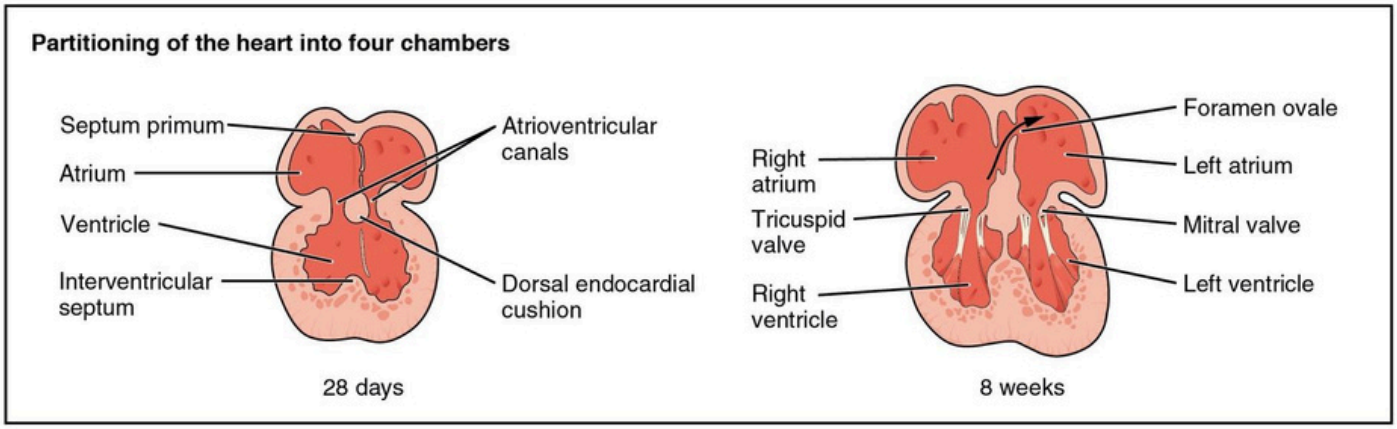


Basics of Foetal Development of the Heart:

- Begins at Wk 3 of Gestation
- Why? Because by this stage, the foetus is too large for nutrient/gas exchange to be via simple diffusion.
    - Therefore, an active nutrient/gas distribution system is needed for continual growth of Foetus.
- Beating occurs @ week 4/5

- **Starts as The "Cardiac Tube":**
    - The primordial tubular heart in the embryo, before its division into chambers.
    - This cardiac tube begins to fold & twist on itself until it is laid out in the basic heart-like structure.
- **Then Undergoes Septation:**
    - Between 4-6 weeks



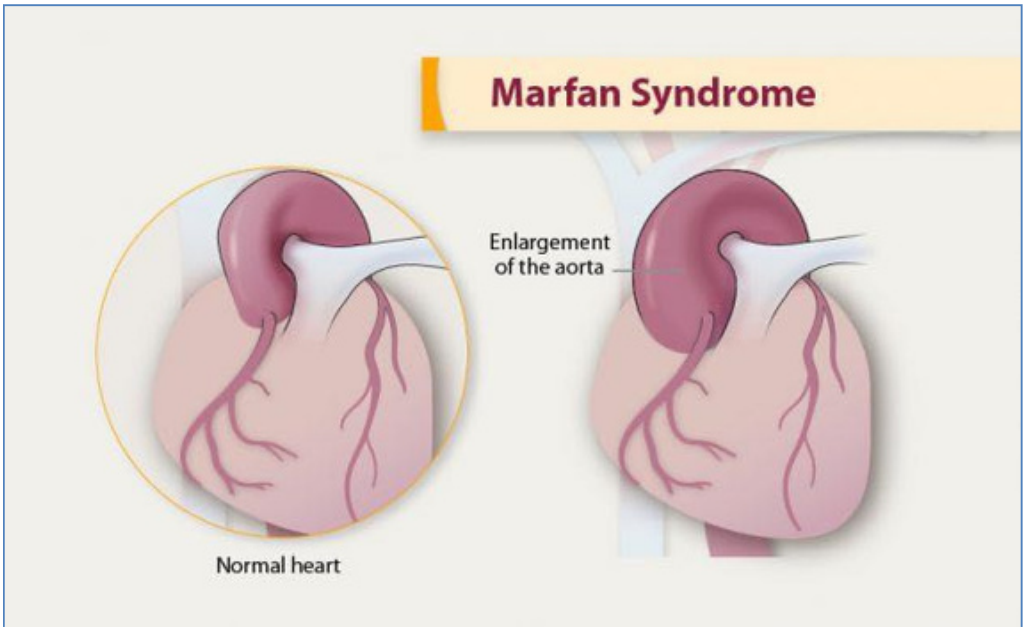


CONGENITAL HEART DEFECTS OVERVIEW:

- **Note: ~50-80% of Children have "Innocent" Heart Murmurs;**
- **Red Flag = Murmur + Cyanosis/↓Perfusion**
- **Left→Right (Non-Cyanotic) Shunts – (ASD, VSD, PDA)**
- o VSD = Commonest
- **Right→Left (Cyanotic) Shunts – (TETRALOGY & TRANSPOSITION)**
  Obstructive Defects – (COARCTATION, Valvular Stenoses)

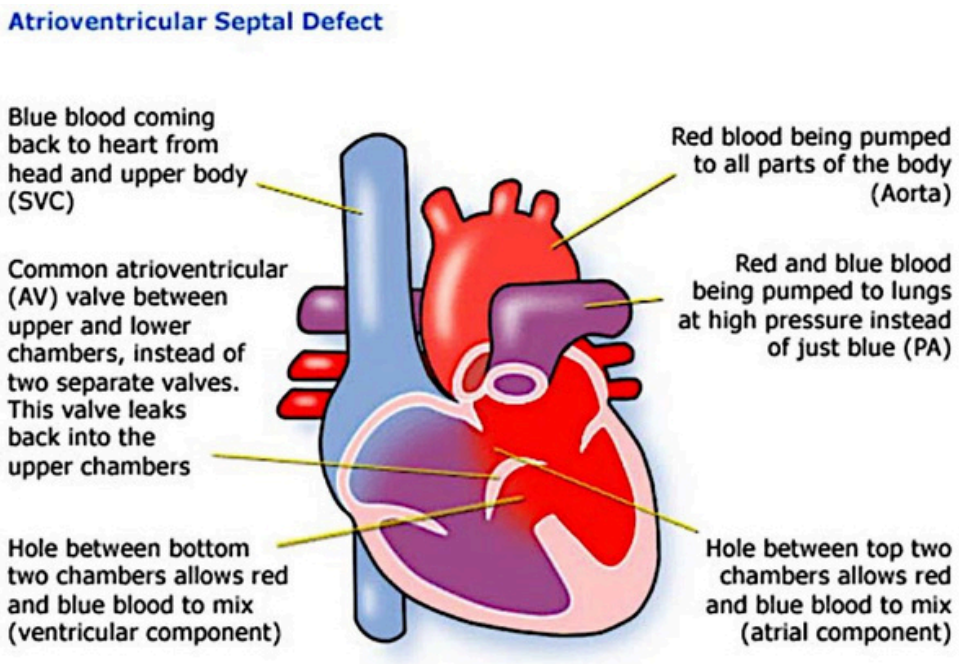
Common Genetic Associations:

- **MARFAN'S SYNDROME:**
  - o **Autosomal Dominant Disorder**
  - o **CV-Defects: - (**Aortic aneurysm/Mitral Prolapse / Tricuspid Prolapse / ASD / Others**)**



https://www.cdc.gov/heartdisease/marfan_syndrome.htm

- **DOWN'S SYNDROME:**
  - o **= Trisomy 21**
    - § 40% of Down's Syndrome Patients have congenital Heart Defects
  - o **CV-Defects: - (**Valvular Malformations / ASD + VSD**)**



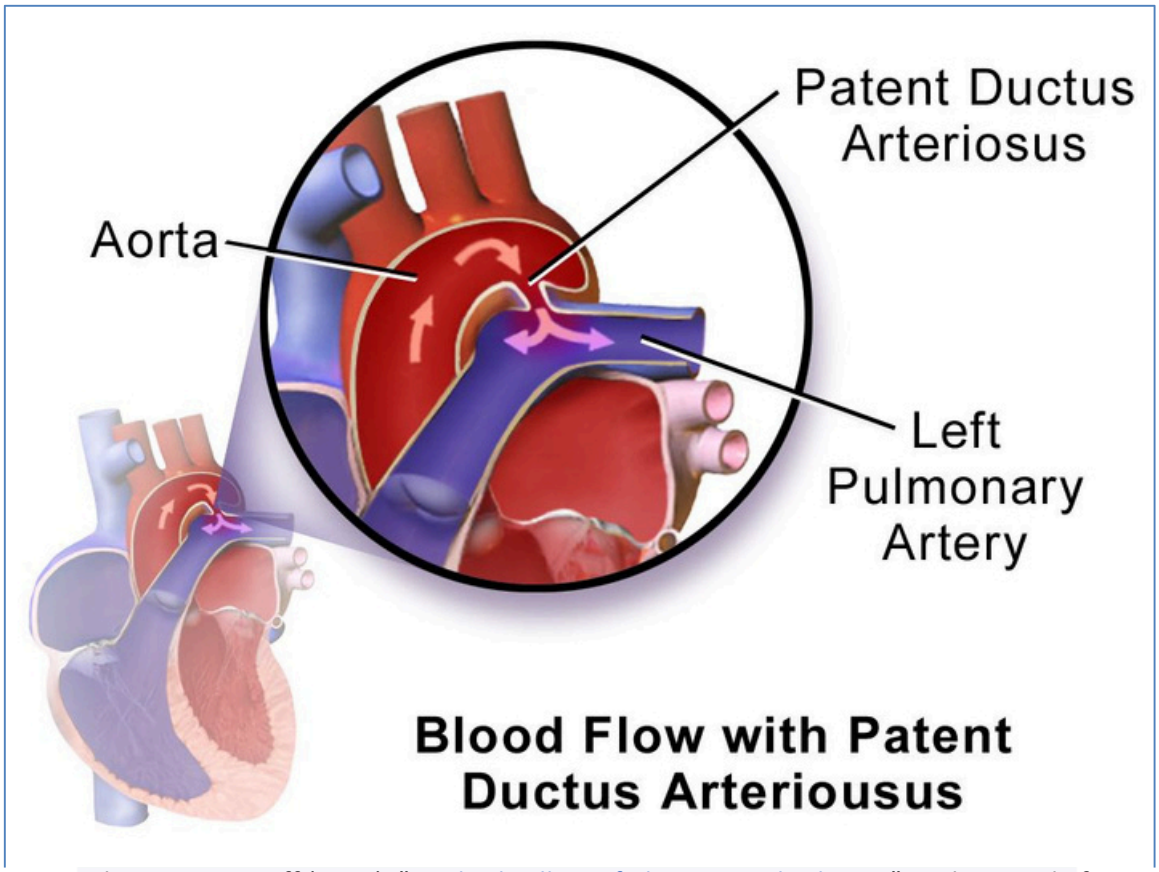
https://www.chfed.org.uk/wp-content/uploads/2018/08/AVSD-1.pdf

LEFT→RIGHT (NON-CYANOTIC) SHUNTS.

- **PATENT DUCTUS ARTERIOSUS (PDA):**
  - o = **Malocclusion of Ductus Arteriosus after birth**.
    - § **L→R Shunt from Aorta → Pulmonary Artery**
    - § • → Pulmonary Hypertension

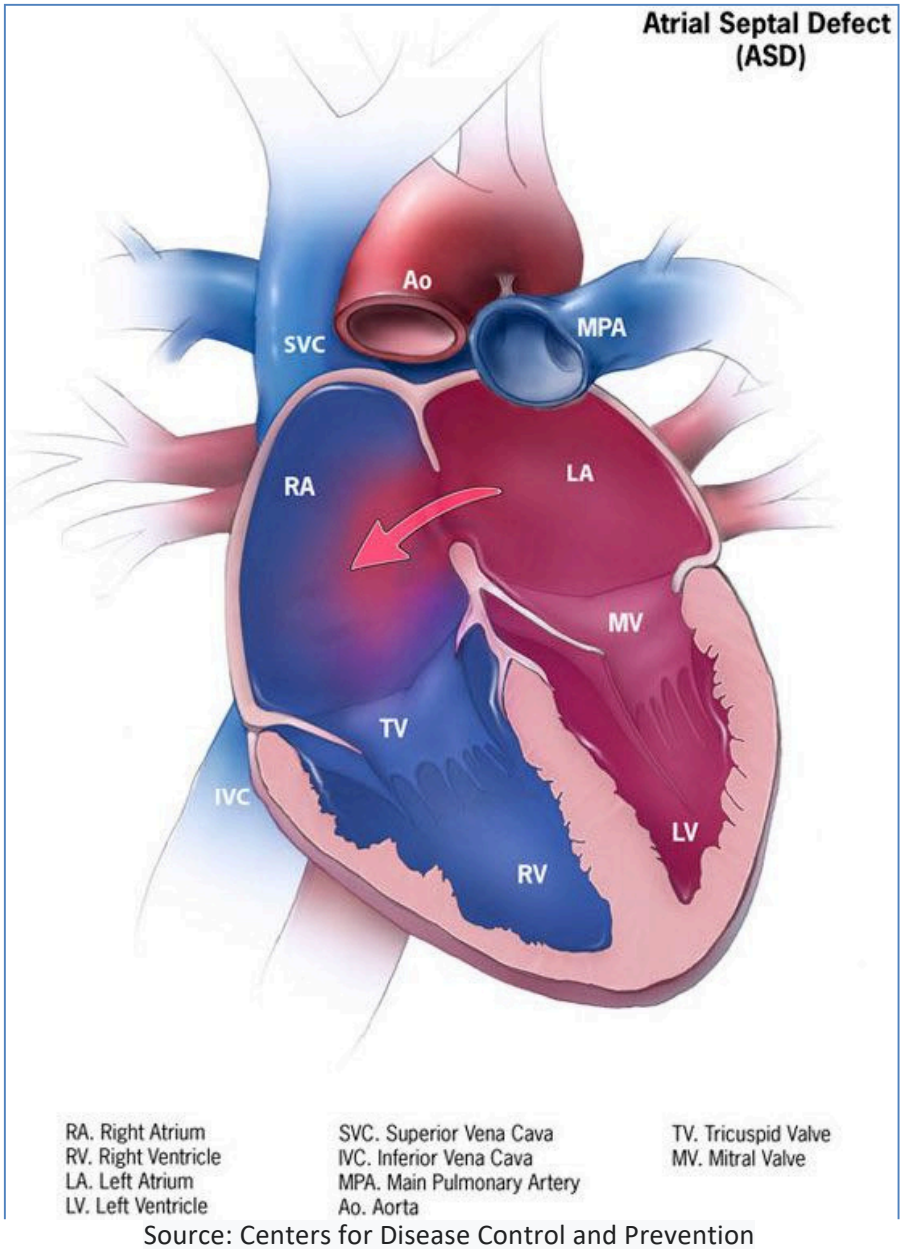
      Normally, Rising O2 Tension & Decreasing Prostaglandins cause it to Close.
  - o **Clinical Features:**
    - § **Murmur** *(Continuous*, *Harsh* "**Machinery-like**" *Murmur*).
  - o **Complications:**
    - § Soon After → **Irreversible Obstructive Pulmonary Vascular Disease** (Pulmonary Vessel Hypertrophy & Vasoconstriction →↑Resistance) → SHUNT REVERSAL → Cyanosis
  - o **Investigations:**
    - § **CXR – (**Pulmonary Congestion, Cardiomegaly**)**
    - § **ECG – (**LVH, RVH**)**
    - § **ECHO – (Definitive)**
  - o **Management:**
    - § **(*PDAs should be closed as early in life as possible)**
    - § **Medical:** Indomethacin (Prostaglandin Inhibitor)
    - § • (Note: In Cyanotic Heart Defects, Prostaglandin is actually *Given* to maintain a PDA)

      Surgical: Surgical Ligation



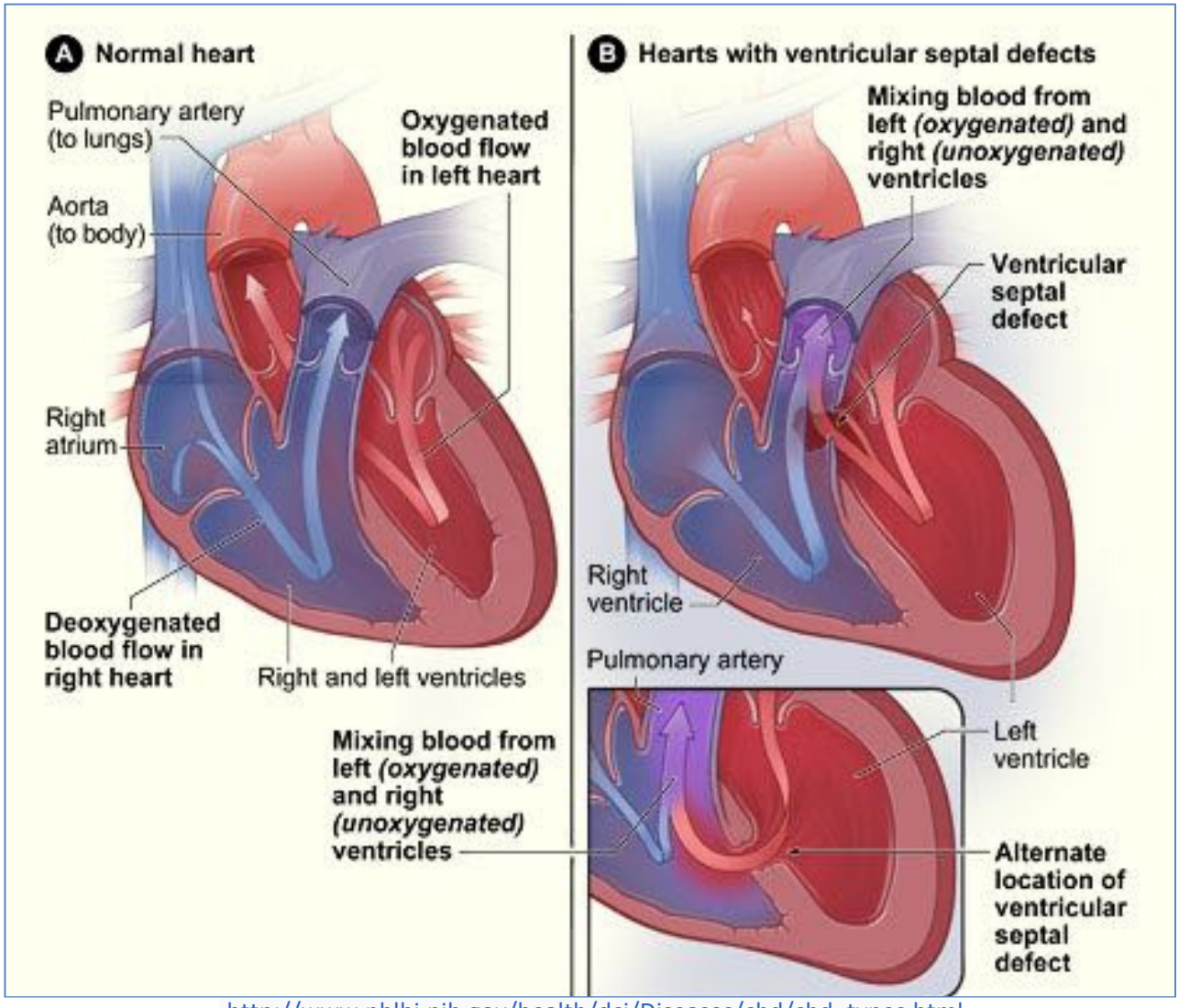
Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". *WikiJournal of Medicine* **1** (2). DOI:10.15347/wjm/2014.010. ISSN 2002-4436

- **ATRIAL SEPTAL DEFECT (ASD):**
  - o **= Hole in the Interatrial Septum. (Note: NOT a patent Foramen Ovale)**
    - § **→ Shunt from L-Atria → R-Atria:**
      - → RV-Hypertrophy & Pulmonary HTN.
  - o **Clinical Features:**
    - § ***Asymptomatic in Childhood*** *(Symptom onset ≈ 30yrs).*
    - § **Murmurs:**
      - Diastolic ASD Murmur (During Atrial Contraction)
      - (Systolic Pulmonary Flow-Murmur (Hyperdynamic))
      - (Splitting of S2 (Delayed P2))
    - § **RV-Hypertrophy → Parasternal Heave**
  - o **Complications are Rare, but Include:**
    - § **CCF** → Pulmonary Oedema (Dyspnoea) + Peripheral Oedema, Ascites, etc.
    - § **"Paradoxical Embolisation"** (DVT → Stroke)
  - o **Investigations:**
    - § **ECG –** (RV-Hypertrophy, RAxDev)
    - § **CXR –** (Pulmonary Congestion & Oedema)
    - § **ECHO – (Definitive)**
  - o **Management:**
    - § Surgical Endovascular Closure of Defect



Source: Centers for Disease Control and Prevention

- **VENTRICULAR SEPTAL DEFECT (VSD):**
  - o (*The Most Common Congenital Heart Disease)
  - o = **Hole in the Interventricular Septum**.
    - § **→ Shunt from L-Vent. → R-Vent.**
      - → LV-Failure (CCF, Pulmonary HTN, RV-Hypertrophy).
  - o **Clinical Features:**
    - § **Asymptomatic if Small (& Close Spontaneously)**
    - § ***Failure to Thrive* if Large (& Requires Surgical Closure)**
    - § **Murmurs:**
      - Pansystolic VSD Murmur (+/- L-Sternal Thrill)
      - Pulmonary Valve Flow Murmur
    - § **CCF –** (Dyspnoea, Cough, Peripheral Oedema)
  - o **Complications:**
    - § Initially a L-R-Shunt → **Pulmonary HTN** → RV-Hypertrophy
    - § **Later → Irreversible Obstructive Pulmonary Vascular Disease** → SHUNT REVERSAL:
      - R-L Shunt (↓O2 Blood → Systemic Circulation → Cyanosis/Death)
  - o **Investigations:**
    - § **CXR –** (Pulmonary Congestion, Cardiomegaly)
    - § **ECG – (**LVH & RVH)
    - § **ECHO – (Definitive)**
  - o **Management:**
    - § Early surgical intervention is Critical for normal lifespan



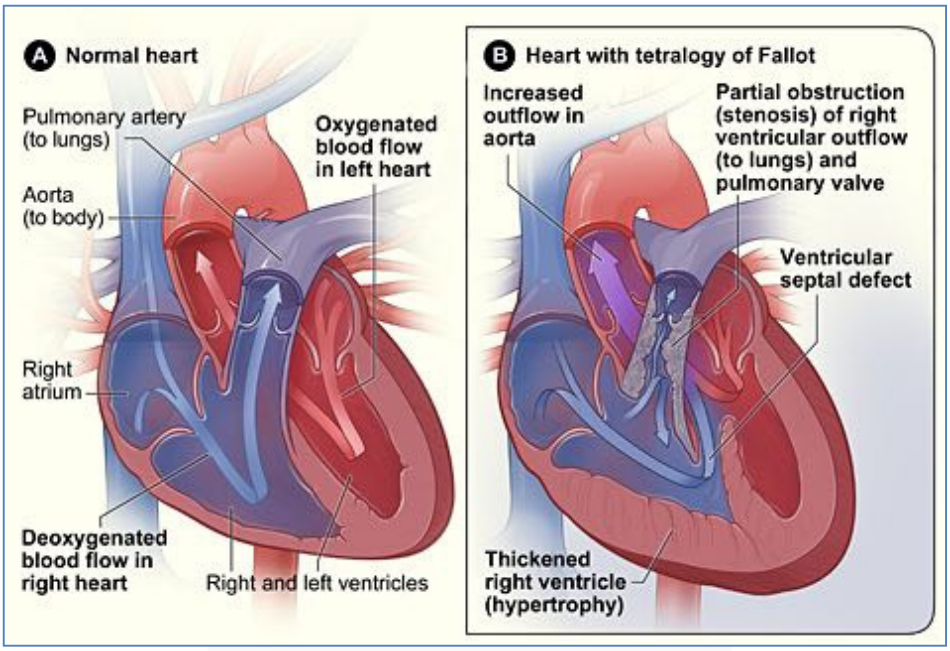
http://www.nhlbi.nih.gov/health/dci/Diseases/chd/chd_types.html

RIGHT→LEFT (CYANOTIC – SPO2 <75%) SHUNTS

- **TETRALOGY OF FALLOT (Cyanotic Heart/"Blue Baby Syndrome"):**
  - ***4 Features:***
    - **1- VSD**
    - **2- Overriding Aorta:**
      - Aortic Valve sits *above* the VSD :. Connected to both the R & L-Ventricle.
    - **3- Subvalvular Pulmonic Stenosis:**
      - → RV-Outflow Obstruction → R-L-Shunt → Hyopxemia/Cyanosis

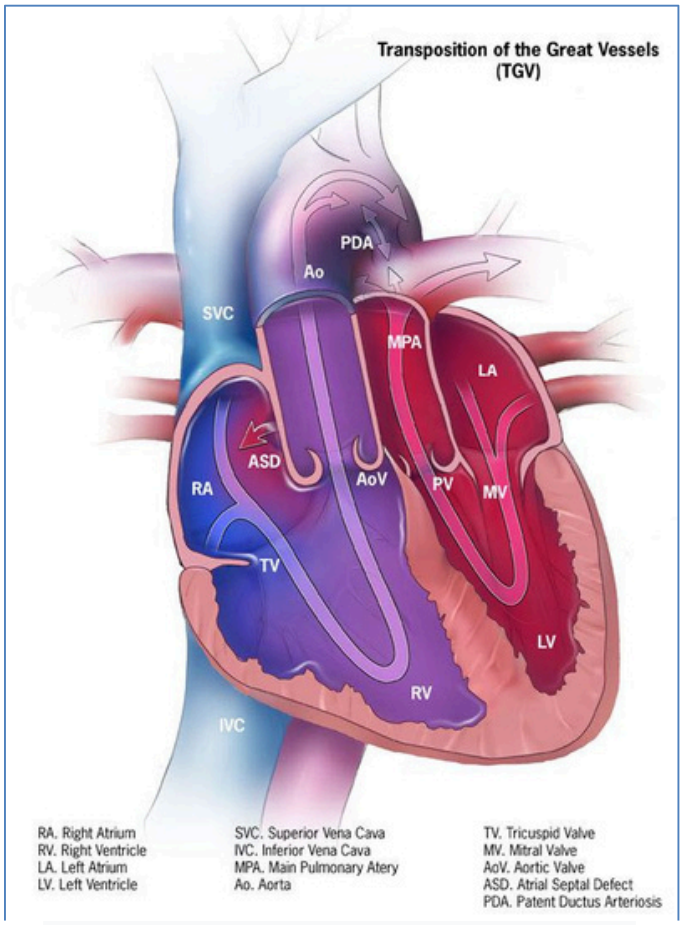
      4- R-Ventricular Hypertrophy:
      - Due to ↑R-Vent. Worlkload

      (5- Sometimes Patent Ductus Arteriosus)
  - **Clinical Features:**
    - **If Mild Pulmonary Stenosis →** Resembles an isolated VSD. (L-R-Shunt) [Non Cyanotic]
    - **If Severe Pulmonary Stenosis** → R-L-Shunt →
      - Chronic Cyanosis SpO2 <75%
      - Fingernail Clubbing
      - Polycythaemia (↑RBC)
    - **Symptoms:**
      - Blue Baby
      - Paroxysmal Tachypnoea
      - Irritability/Crying
    - **Classical Sign: Symptoms alleviated** by squatting (kinking femoral artery) thereby **increasing System ic Vascular Resistance**
      - → Means more blood being ejected via the Pulmonary Artery → Better Oxygenation.
  - **Complications:**
    - *"Paradoxical Embolism"* – (DVT → Stroke)
    - Seizures
  - **Investigations:**
    - **ECG – (**RV-Hypertrophy**)**
    - **CXR – (**Boot-Shaped Heart**)**
    - **ECHO – (Definitive)**
  - **Management:**
    - **Medical:**
      - **Supplemental O2**
      - **B-Blocker**
    - **Surgical:**
      - **Definitive Repair**



National Heart Lung and Blood Institute (NIH)

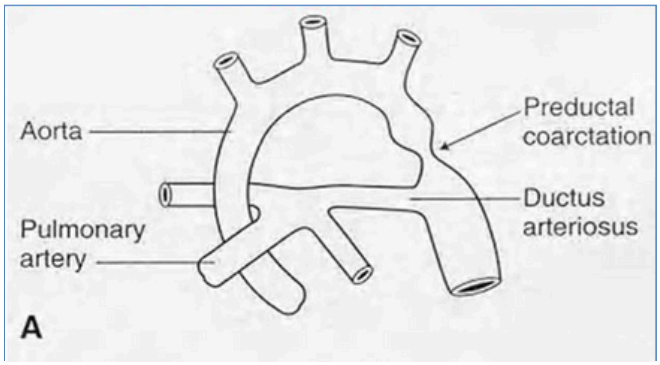
- **TRANSPOSITION OF GREAT VESSELS:**
  - **Where the Aorta comes off the R-Ventricle & the Pulmonary Artery comes off the L-Ventricle**.
  - **= Aorta & Pulmonary Artery are switched**.
    - (Note: Atrioventricular Connections are still correct)
    - (Note: Venous Return is correct – IVC/SVC & Pulmonary Veins)
    - **Hence, the Pulmonary & Systemic Circuits run in *Parallel*, rather than *Series***.
  - **Note: Incompatible with Post-Natal Life *Unless* a Shunt exists for Mixing of Blood:**
    - Eg: TGV + VSD = Stable Shunt. (Adequate mixing)
    - Eg: TGV + Patent Foramen Ovale = Unstable Shunt (Tends to close).
  - **Clinical Features:**
    - **The Most Common Cause of Cyanosis ("Blue Babies") *during* Infancy**
    - **Severe Hypoxemia & Cyanosis →**
      - Blueness of skin & mucous membranes
      - Fingernail Clubbing
      - Polycythaemia (↑RBC)
  - **Complications**
    - Prominent R-Ventricular Hypertrophy (R-V Pressure overload)
    - Atrophy/Thinning of L-Ventricle
  - **Investigations:**
    - **ECG – (**RAxDev, RVH**)**
    - **CXR – (**Egg-Shaped Heart with Narrow Mediastinum – "Egg on a string heart"**)**
    - **ECHO – (Definitive Dx)**
  - **Management:**
    - **Prostaglandin Infusion – (**To maintain PDA & allow mixing of blood**)**
    - **Surgical repositioning of Great Vessels**
  - **Prognosis:**
    - **Typically Incompatible with Life:**
      - 30% die within a week
      - 90% die within a year
    - **90% 1yr Mortality without Surgery**.



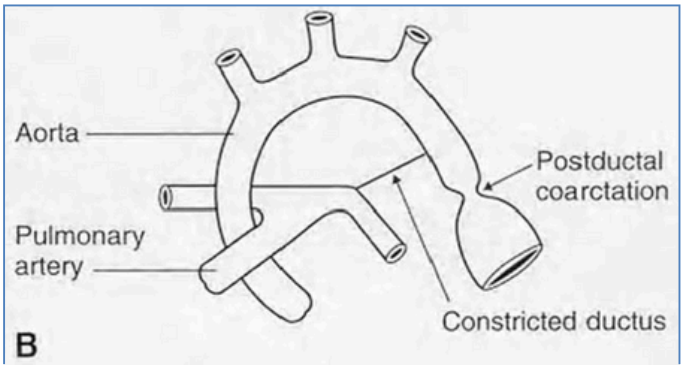
Source: Centers for Disease Control and Prevention

'OBSTRUCTIVE' CONGENITAL HEART DEFECTS:

- **COARCTATION OF AORTA**
  - **= Narrowing/Constriction of the Aorta.**
    - 2Male:1Female
    - *50%* have *Bicuspid Aortic Valve*
  - **Pathophysiology – 2 Types:**
    - **Pre-ductal:**
      - Proximal to Ductus Arteriosus
        - → R-L-Shunt (Pulmonary Artery → Aorta).
        - → Cyanosis of *Lower Half* of body.
    - **Post-ductal:**
      - Distal to Ductus Arteriosus.
        - → L-R-Shunt from Aorta→Pulmonary Artery
        - → Pulmonary HTN & CCF
    - **Leads to:** ↑Afterload → ↑L-Ven. End Systolic Volume → ↓Cardiac Output → Hypotension
      (↓Cardiac Output → Backup of Blood in Lungs → Pul.Congestion)
      → L-Ventricular Hypertrophy → Possibly Left Heart Failure
      →Decreased Perfusion to Abdominal Organs & Lower Limbs.
  - **Clinical Features:**
    - **Symptoms:**
      - **Leg Claudication**
      - **Note: Presentation may take up to 10 years** – As the Coarctation doesn't grow with the rest of the body → Only symptomatic when peripheral demand > Aortic Flow.
    - **Signs:**
      - **Upper limb BP > Lower limb BP**.
      - **RF-Delay**
      - **Cold Legs & ↑CRT**
      - **Systolic Murmur**
      - **LV-Hypertrophy**
  - **Investigations:**
    - **ABI - (**Asymmetrical**)**
    - **ECG – (**LV-Hypertrophy**)**
  - **Management:**
    - **Surgery** – (Balloon Angioplasty & Stenting).

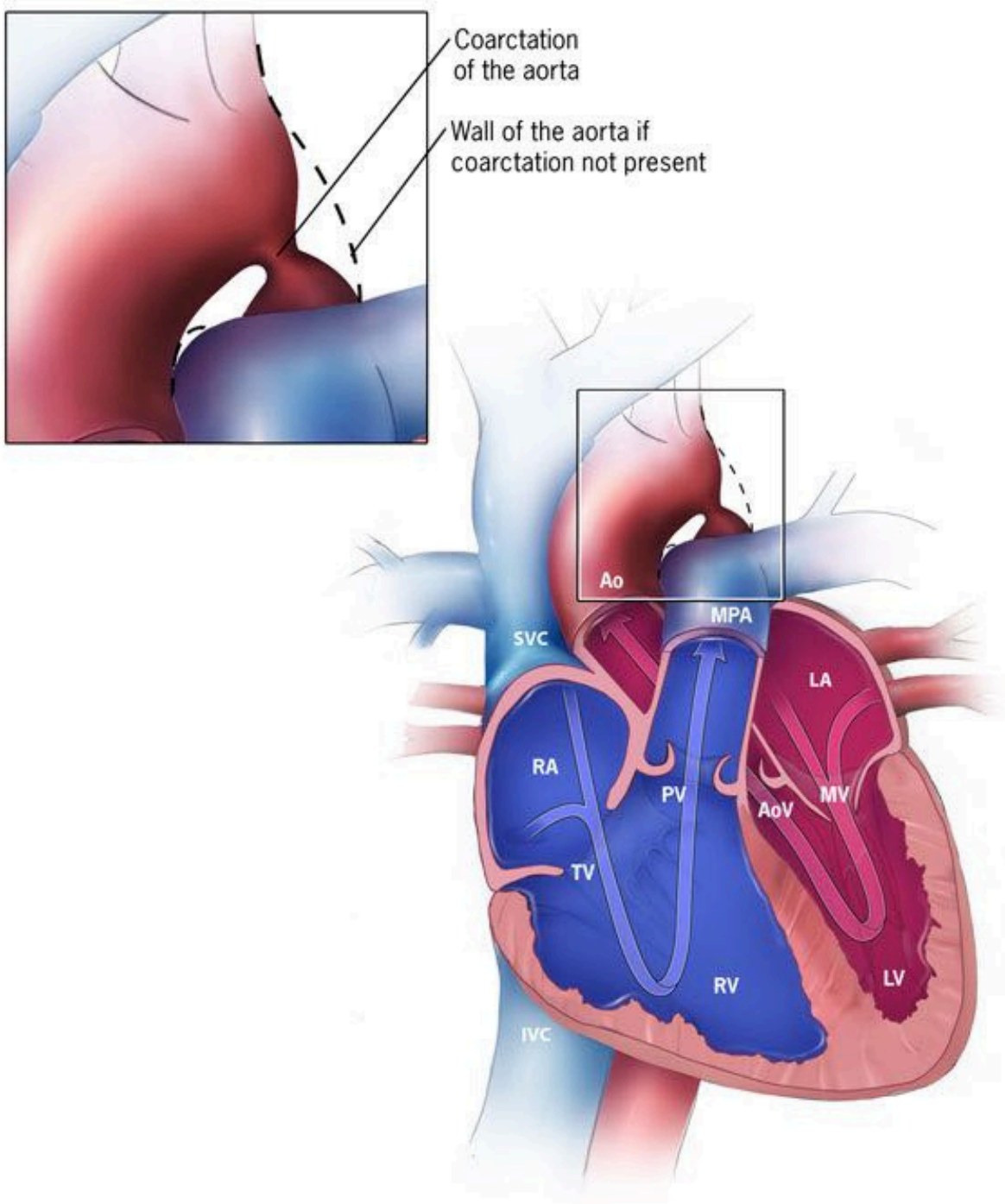


Preductal Coarctation



Postductal Coarctation

Coarctation of the Aorta



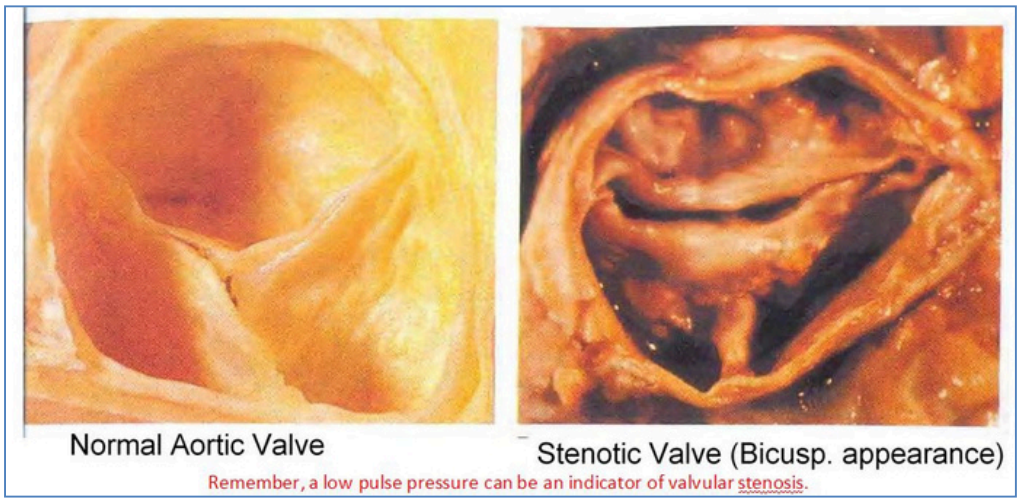
RA. Right Atrium
RV. Right Ventricle
LA. Left Atrium
LV. Left Ventricle

SVC. Superior Vena Cava
IVC. Inferior Vena Cava
MPA. Main Pulmonary Artery
Ao. Aorta

TV. Tricuspid Valve
MV. Mitral Valve
PV. Pulmonary Valve
AoV. Aortic Valve

Source: Centers for Disease Control and Prevention, National Center on Birth Defects and Developmental Disabilities

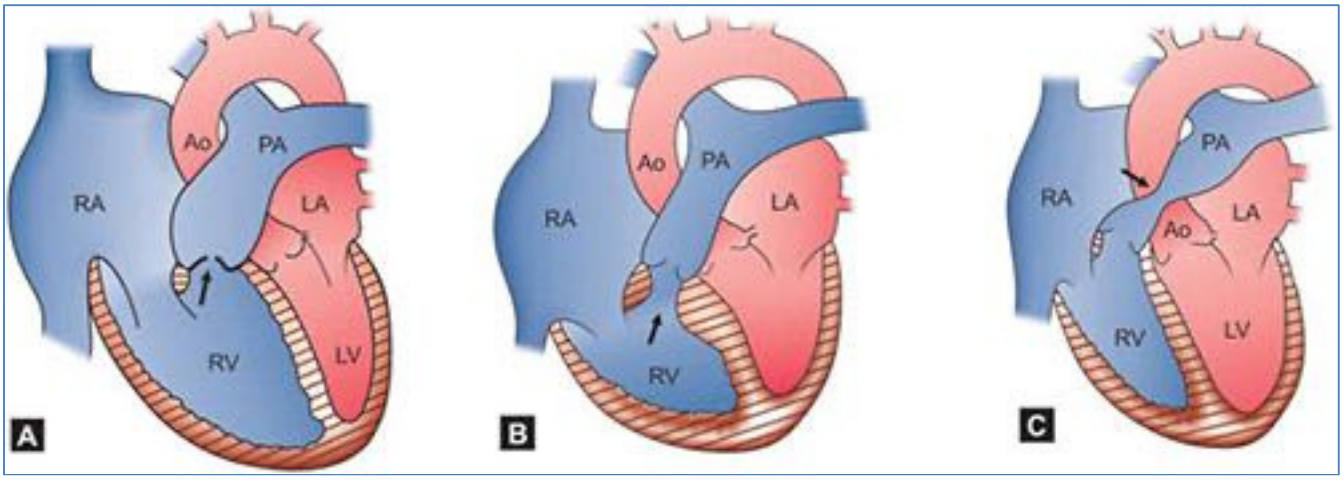
- **AORTIC STENOSIS:**
  - o **= Narrowing/Obstruction of the Aortic Valve**.
  - o Typically presents as a Bi-Leaflet, instead of the normal Tri-Leaflet Formation.
  - o Most common in males.
  - o **Leads to** → ↑LV-Afterload → ↓Cardiac Output → LV-Failure (Pul.HTN, Dyspnoea)



Source: Unattributable

- **PULMONIC STENOSIS:**
  - o **= Narrowing/Obstruction of the Pulmonary Valve OR Artery**.
    - § 90% = Valvular
    - § 10% = Elsewhere in the Pulmonary Artery
  - o **Leads to** → ↑RV-Afterload → ↓Pulmonary Output → RV-Failure (Peripheral Oedema)

A: Valve Stenosis; **B: Subvalve Stenosis;** **C. Supravalve Stenosis**



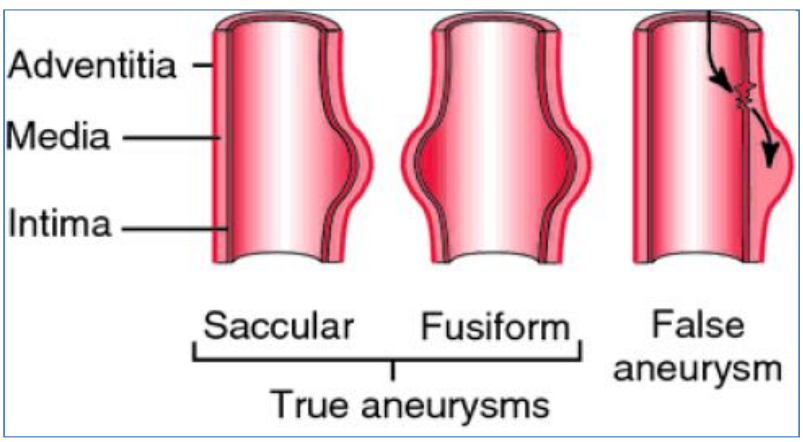
Source: Unattributable

Aneurysms (General Info):

Definition:

- o ***Most vascular surgeons: "A >50% Increase in the Size of an Artery Above its Normal Size"***
  - § Eg: Normal Infra-Renal Aorta = 2cm :. An Aneurysm would be >3cm.
  - § (90% of AAAs are Infra-Renal)
- o ***Robbins – "a Localised abnormal dilation of a BLOOD VESSEL OR THE HEART".***

- **True Vs. Pseudo- Aneurysms:**
  - o **True Aneurysms –** *(Full Thickness Aneurysms)*
  - o **False/Pseudo Aneurysms –** *(Partial Thickness Aneurysms)*
- **Classification (Size/Shape):**
  - o **"Saccular Aneurysms":** Hemispherical Outpouchings involving ONLY PART of the vessel wall
  - o **"Fusiform Aneurysms":** CIRCUMFERENTIAL Dilation of a vascular segment
  - o **"Dissecting Aneurysms":** Blood within the Arterial wall *itself*.



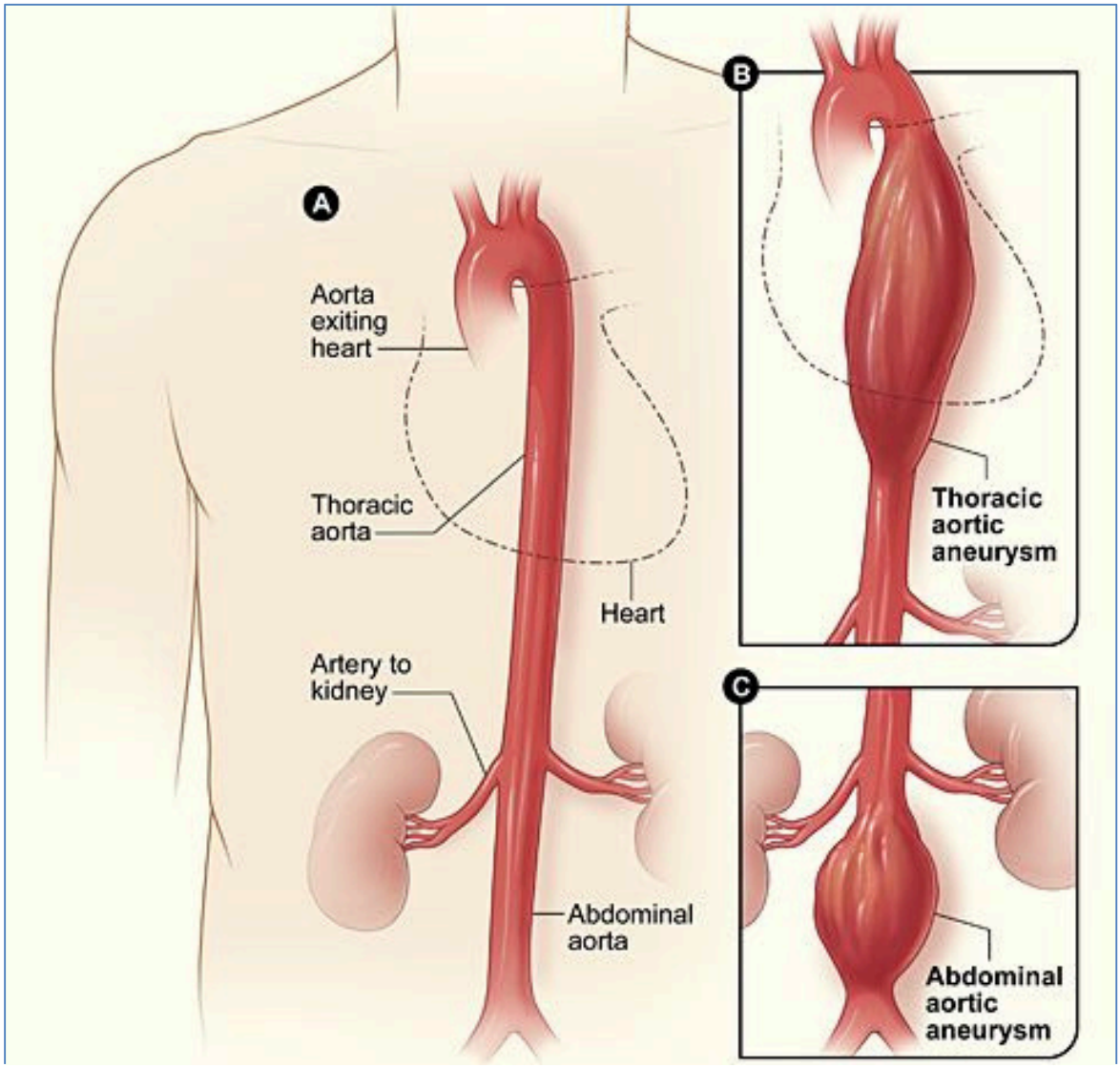
- **Aetiologies:**
  - o **Atherosclerosis -** (Typically *AAAs*)
  - o **Hypertension -** (Typically *Thoracic Aortic Aneurysms*)
  - o **Myocardial Infarction -** (Typically *Ventricular* Aneurysms)
  - o **(Others: Congenital – Eg: Downs/Marfan's/Ehlers-Danlos Syndrome/Connective Tissue Disorders/Etc)**
- **Risk Factors:**
  - o **Age >65**
  - o **Male**
  - o **Atherosclerosis**
  - o **↑Cholesterol**
  - o **HTN**
  - o **Smoking**
  - o **FamHx**

ABDOMINAL AORTIC ANEURYSM:

- **Aetiology:**
  - Typically Atherosclerosis (But can be due to other causes)
- **Pathogenesis:**
  - Atherosclerotic Plaque → Weakening of Vessel Wall → Aneurysm
- **Morphology:**
  - 90% of AAAs are ***INFRA-RENAL***
  - Saccular OR Fusiform
- **Clinical Features:**
  - **Presentation:**
    - **Typically Asymptomatic (Hence *"Sudden Death"*)**
    - **But Symptoms Include:**
      - Pulsatile Abdo Mass.
      - Pain - Back/Flank/Abdo/Groin
      - DVT (From Venous Compression)
      - "Trash Foot" – from Thrombo-Emboli
- **Investigations:**
  - Clinical Suspicion + Examination
  - ****Abdo USS – (100% Sensitive)**
  - CT/MRI
- **Com plications:**
  - **#AAA – (Note: SIZE = #1 Predictor of Rupture):**
    - **Classic Triad of Rupture:**
      - Sudden Pain – (Abdo/Back)
      - Shock – (Hypotension/ALOC)
      - Pulsatile Mass
    - **+ Acute Abdomen**
    - **+ Grey Turners Sign**
  - **Occlusion of a Branch-Vessel:**
    - Eg: Pre-Renal Failure
    - Eg: Mesenteric Ischaemia
  - **Thromboemboli:**
    - Renal Infarction
    - Mesenteric Infarction
    - "Trash Foot" – Focal Gangrene.
- **Management:**
  - **AAAs <5cm Diameter → Watchful Waiting (6mthly)**
    - + Risk Factor Modification
  - **AAAs >5cm Diameter → Surgical Repair (Due to ↑ Rupture Risk)**
    - (Open Vs. Endovascular Repair)
  - **#AAA (Ruptured AAA) → EMERGENCY SURGERY:**
    - + 2x Large Bore Cannulas
    - + Fluid Resuscitation (Bolus + Maintenance; Target BP ≈ 80 Systolic)
    - + Group & Hold + X-Match for Transfusion
- **Prognosis:**
  - **Pre-Rupture:** Good Prognosis
  - **Post Rupture: 95% Mortality** – (Only 30% Make it to Hospital; 20% of those Survive).

THORACIC AORTIC ANEURYSMS:

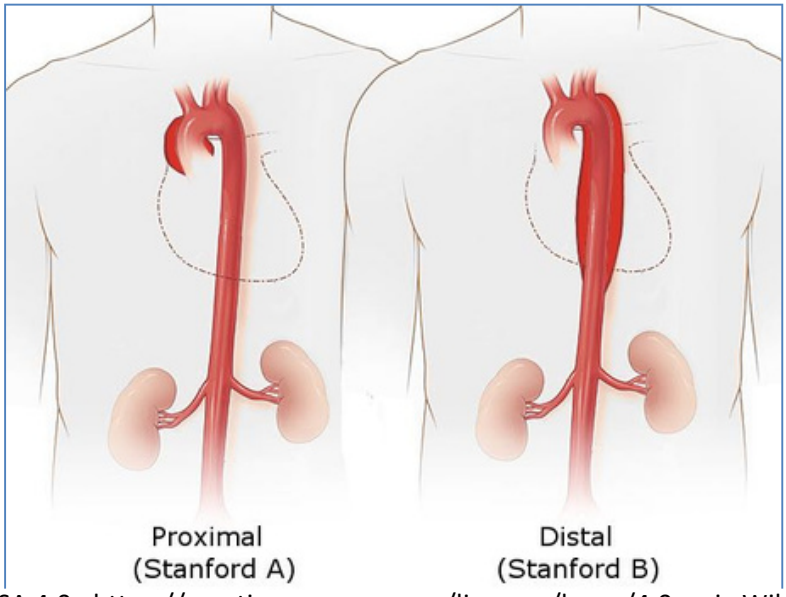
- **Aetiology:**
  - Hypertension
- **Clinical Features:**
  - **Complications:**
    - Mediastinal Compression (Heart & Lungs)
    - Dysphagia
    - Cardiac Disease (Eg: Aortic Regurgitation, Myocardial Ischaemia/Infarction)
    - Rupture



Public domain image: http://www.nhlbi.nih.gov/health/health-topics/topics/arm/types.html

AORTIC DISSECTION:

- **Aetiology:**
  - Hypertension
  - M:F = 4:1
- **Pathogenesis:**
  - Hypertension → Intimal Tear → Blood Enters False Lumen → Dissection Continues
- **Morphology:**
  - **#1- Ascending Type (*Ascending Aorta*):**
    - Bad because can → Occlude *Brachiocephalic Trunk/Internal Carotid/Subclavian*.
  - **Descending Type (*Descending Aorta*):**
    - Bad because can → Dissect all the way to legs → GI/Renal/Limb Ischaemia
- **Clinical Features:**
  - **Sudden Excruciating Chest Pain Radiating to the Back between Scapulae**
  - **+/- Signs of Complications:**
    - **Rupture** → Cardiac Tamponade & Shock
    - **Valvular** → Aortic Regurgitation → Diastolic Murmur (Due to Dilation)
    - **Vessel Occlusion** →MI, Stroke, Limb Ischaemia, Mesenteric Ischaemia, Renal Fail
- **Investigations:**
  - **CXR –** Wide Mediastinum, L-Pleural Effusion
  - **CT –** 100% Sensitive
  - **TOE (Echo) –** 100% Sensitive, but slow.
- **Management:**
  - **Aggressive BP-Reduction (Nitrates + B-Blocker)** → Slows Progression
    - **If Ascending:** EMERGENCY SURGERY
    - **If Descending:** Initial Medical Mx



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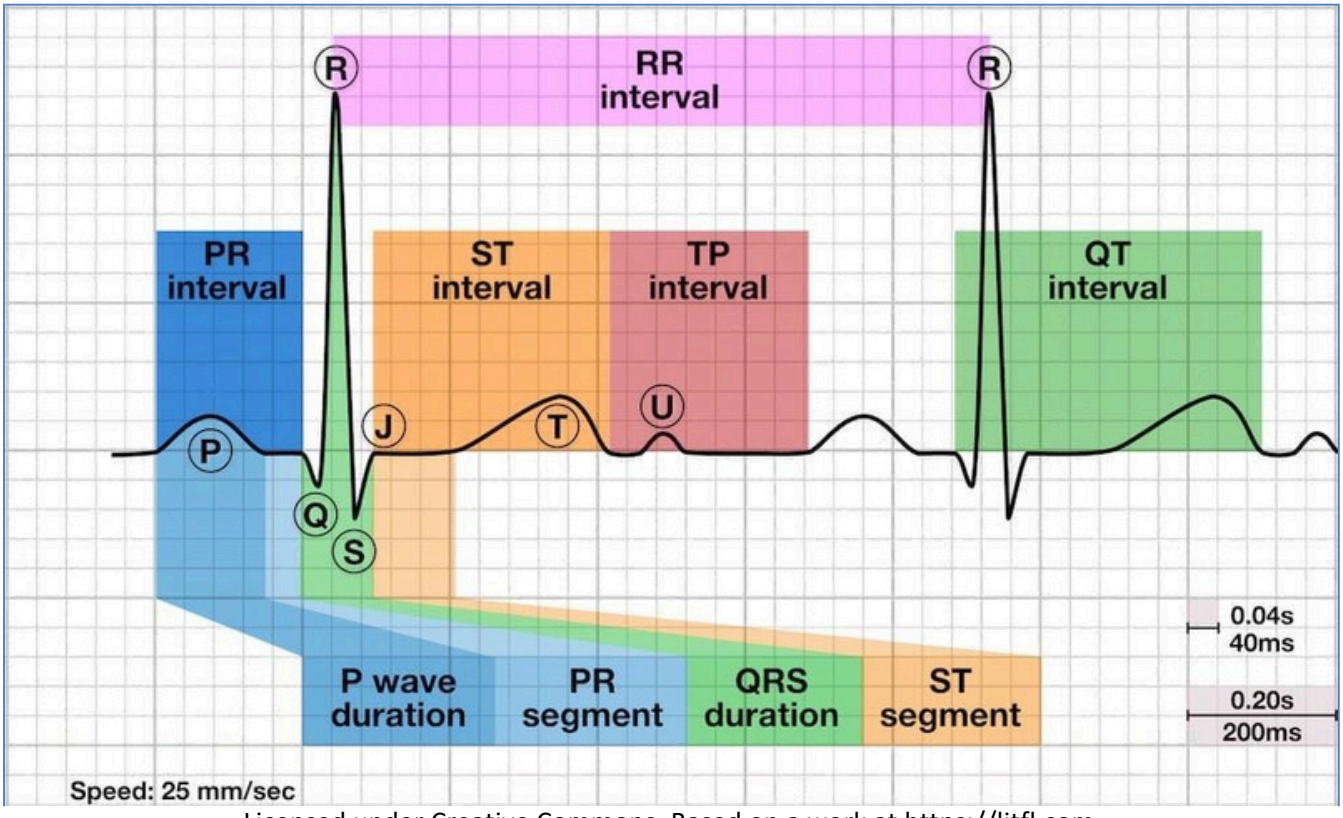
CEREBRAL ANEURYSM (Congenital Berry Aneurysms – See Sub-Arachnoid Haemorrhage in Nervous System Notes):

- **Symptoms for an aneurysm that has not yet ruptured –**
  - Fatigue
  - Loss of perception
  - Loss of balance
  - Speech problems
- **Symptoms for a ruptured aneurysm –**
  - Severe headaches
  - Loss of vision
  - Double vision
  - Neck and and/or stiffness
  - Pain above and/or behind the eyes.

Characteristics of a Normal ECG:

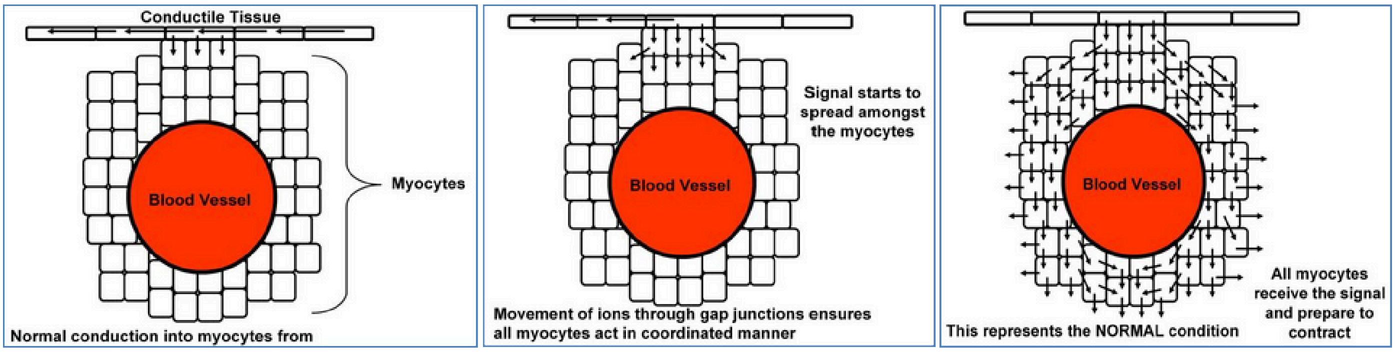
- **Sinus Rhythm/Rate:**
  - o Between 60-100 bpm.
  - o Initiated by SA-Node
  - o Note: it's intrinsic rate is higher, but is suppressed by constant Parasympathetic-NS Influence
- **P-Wave:**
  - o Rounded
  - o Between 0.5-2.5 mm Tall
  - o Less than 0.1 Seconds Duration
- **PR-Interval:**
  - o Fixed
  - o Between 0.12-0.20 Seconds
- **QRS-Complex:**
  - o Clean & Sharp
  - o Normally Less Than 25mm Tall
  - o QRS Interval: Between 0.06-0.12 Seconds Duration
- **Q-T Interval:**
- o Between 0.35-0.45 Seconds Duration
- **S-T Segment:**
  - o Normally ≈0.08 Seconds Duration

  T-Wave:
  - o Prominent
  - o Rounded
  - o Less Than 5mm Tall (Limb) or Less Than 10mm Tall (Precordial)
  - o Between 0.1-0.25 Seconds Duration
- **U-Wave**
  - o Small (0.5mm) deflection immediately following the T-Wave.
  - o Usually in the same direction as the T-wave
  - o Best seen in leads V2 & V3

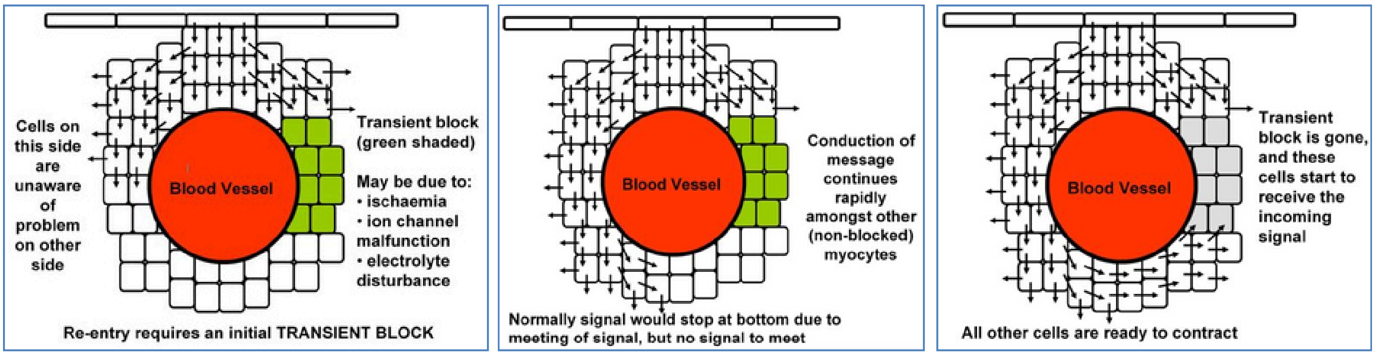


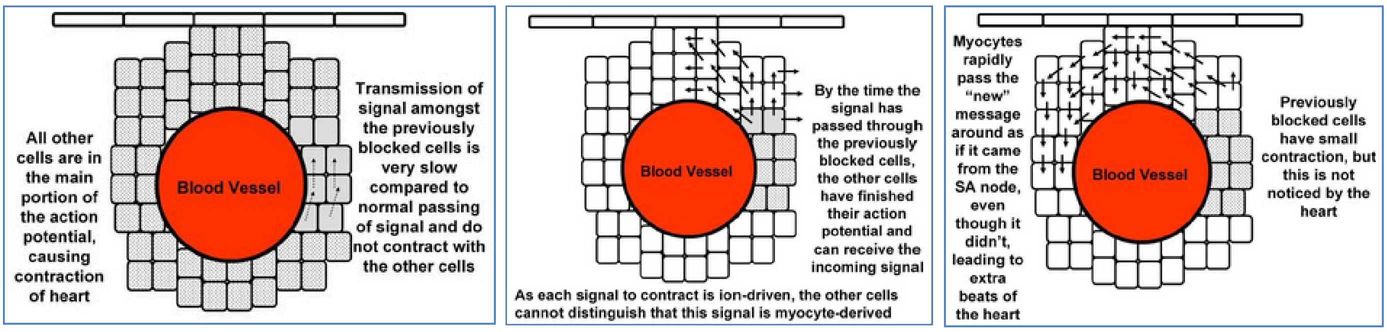
Common Mechanisms of TachyArrhythmias

- **1- Re-Entry (AKA: Re-entrant tachycardias):**
  - Accounts for ≈75% of Tachycardias
  - **Causes of Re-Entry:**
    - Ischaemic Heart Disease
    - Ion-Channel Mutations
    - Electrolyte Disturbances
  - **Results in an "Ectopic Focus":**
    - = An area in the heart that initiates abnormal beats. (Aka: An Ectopic Pacemaker)
    - Ectopic foci may occur in both healthy and diseased hearts
    - Usually associated with irritation of a small area of myocardial tissue.
    - Creates a Single Additional Beat, OR a Full Rhythm.
  - **How It Occurs:**
    - Normally, an Impulse from Conductile Tissue transmits into Myocytes (Contractile Cells),
    - then spreads amongst the myocytes. All Myocytes receive the Impulse and Contract. Note: Once a cell receives a signal, it won't receive another.



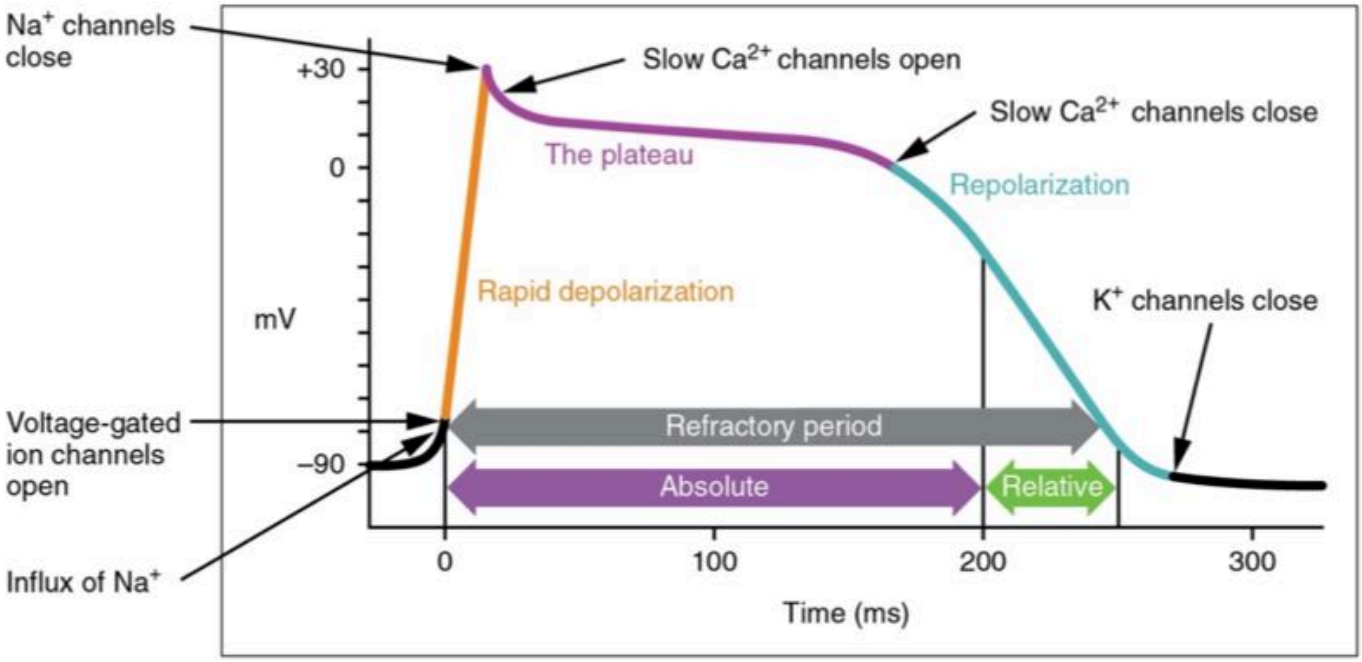
- However, for Re-Entry to occur, an initial momentary/transient Block is required. See Below:





- **2- Early After-Depolarisations (EAD):**
  - **Occur During Repolarisation Phase**
    - (Where K+ is Flowing OUT)
    - (Where Ca+ has STOPPED Flowing IN)
  - **More Likely to occur when Action-Potential Duration is Increased...WHY?**
    - The Absolute Refractory Period for the Na+ Channels (those responsible for depol) only lasts
    - for a small period of time. Usually this period is enough for repolarisation to occur. However, if the AP-Duration is increased, the membrane will still be in *Plateau* when the Na+ Channels enter the Relative Refractory Period, meaning a further stimulus will cause another action potential.
  - **Early After-Depolarisations can result in:**
    - Torsades de pointes (Twisting of the Points)
    - Tachycardia
    - Other Arrhythmias

Fernández-Velasco, María & Benitah, Jean-Pierre & Gomez, Ana & Neco, Patricia. (2012). Ryanodine Receptor Channelopathies: The New Kid in the Arrhythmia Neighborhood. 10.5772/25800.



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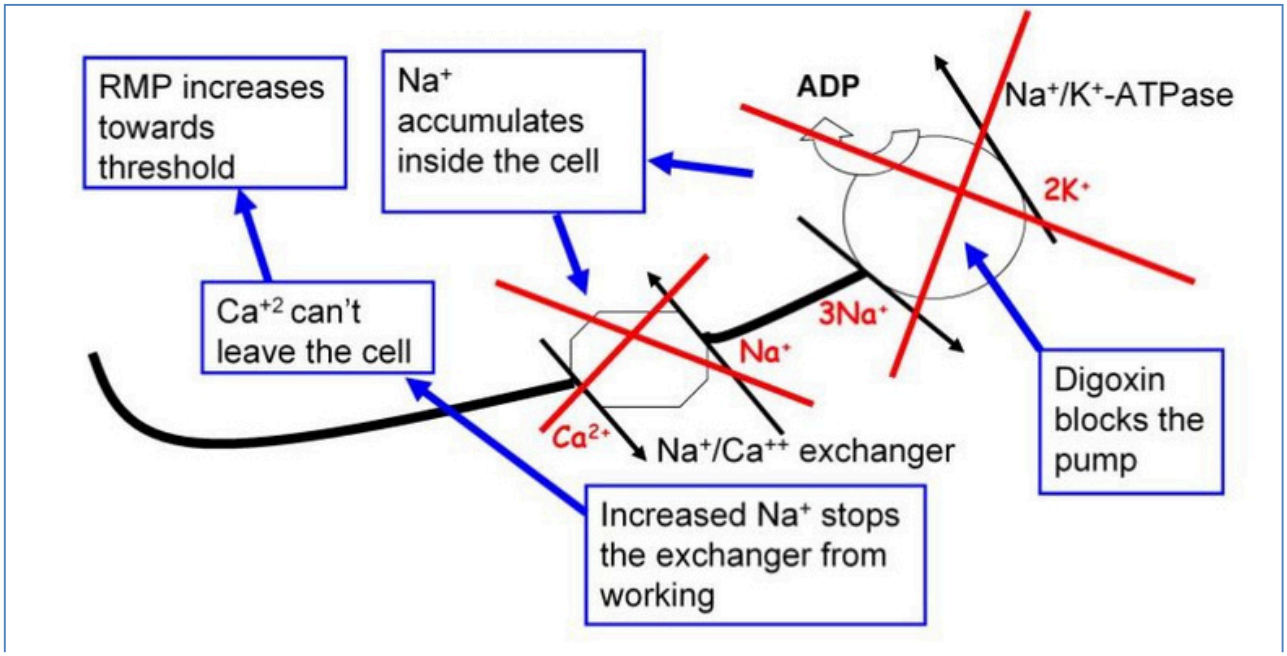
- **3- Delayed After-Depolarisations (DAD):**
  - o Depolarisation during phase 4 (after repolarization is completed, but before another action potential would normally occur)
  - o Due to High Intracellular Ca2+ Concentrations Caused by TOO MUCH DIGOXIN.
    - § Note: Digoxin is a drug used to treat Atrial Flutter & Atrial Fibrillation by Decreasing Conduction Through the AV-Node. Ie: DIGOXIN → DECREASED HEART RATE

  - o **(Digoxin – Mechanism of Action):**
    - § **1-** Blocks the Na+/K+-ATPase on the cell.
      - → Accumulation of Na+ inside the cell
      - → Deficit of K+ inside the cell
    - § **2-** The Secondary Active Na/Ca-Exchanger (That normally relies on the High Extracellular Na+ Gradient to remove Ca+ from the cell) ceases to work.
      - → Accumulation of Ca+ inside the cell → ↓Rate of Depol & Repol of *Pacemaker* Action Potentials → Stops Atrial Flutter/Fibrillation/other atrial tachycardia.

  - o **Note:** This accumulation of Na+ & Ca+ in the cell makes the Resting Membrane More Positive.
    - § → Action Potentials are easier to stimulate
    - § Can Lead to A Series of Rapid Depolarisations.

Fernández-Velasco, María & Benitah, Jean-Pierre & Gomez, Ana & Neco, Patricia. (2012). Ryanodine Receptor Channelopathies: The New Kid in the Arrhythmia Neighborhood. 10.5772/25800.

Digoxin & Delayed After-Depolarisations:

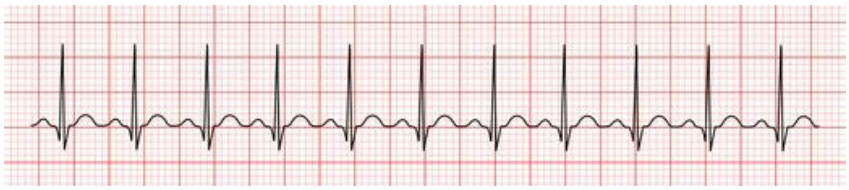


COMMON TACHYCARDIAS:

SUPRAVENTRICULAR TACHYCARDIAS:

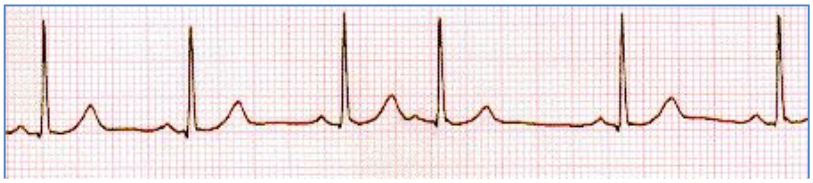
SINUS TACHYCARDIA:

- **= Sinus Rhythm of 100+Beats/min**
  - Shortened T-P Interval
  - All waves clear & visible – Ie: Sinus Rhythm is still very much present
- **Normal During:**
  - Exercise
  - Stimulants (Caffeine)
  - Sympathetic NS Response
- **Pathological Causes:**
  - Fever (Increases Permeability of Ions)
  - Hypovolemia (Eg: Haemorrhagic Shock)
  - Pulmonary Emboli
- **Management:**
  - Carotid Massage
  - (B-Blocker if Symptomatic)



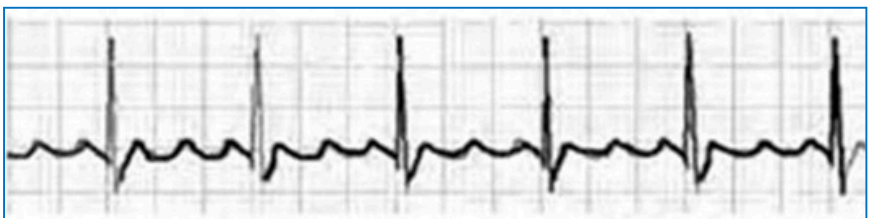
ATRIAL PREMATURE BEATS (APBs):

- **= Single Ectopic P-Waves → Single Ectopic QRS-Complexes**
- **Management:**
  - Nil required
  - (If Symptomatic → B-Blocker or Ca-Ch-Blocker)



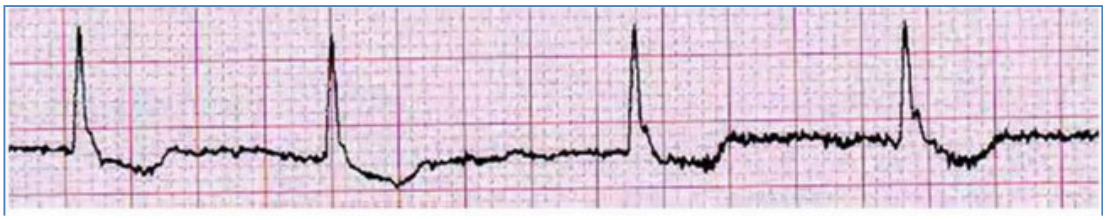
ATRIAL FLUTTER:

- **= Atrial Rate of ≈300bpm; But NOT Sinus *Rhythm*!**
  - Not all waves are conducted to the Ventricles (AV-Node only lets through some of these impulses) → Varied Ventricular Rate
- **P-Waves have a 'Sawtooth' appearance**
  - Ventricular Conduction Variable – (Eg: 2:1 / 3:1 / 4:1 Block etc)
- **Mechanism:** Re-Entry
  - Most Common in Patients with Pre-Existing Heart Disease.
- **Treatment:**
  - **Rate Control –** (B-Blocker, Ca-Ch-Blocker [*Verapamil*], *Digoxin*)
  - **Electrical Cardioversion –** (Different to Defibrillation)
    - To Restore Rhythm (the use of an electric shock)
  - Overdrive Pacing
  - Catheter Ablation (Removal of Blocked Tissue via femoral catheter)



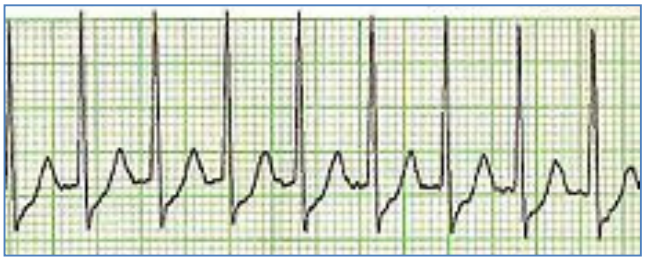
This is an example of a 4:1 (Atria:Ventricle) Conduction Ratio

- **ATRIAL FIBRILLATION (AF):**
  - o **= Sinus Rate of ≈350-600Beats/min; Irregular QRSs**.
    - § Atrial Depolarisations are Disorganised → ineffective Atrial Contraction
    - § Only Partial Signal Transmission to Ventricles → Irregular Pulse Rate.
    - § P-Waves are Unclear
  - o **Causes – "PIRATE SHIV":**
    - § PE, IHD, Rh-Heart Disease, Anaemia, ↑Thyroid, ETOH, Sepsis, HTN, Iatrogenic, Valvular
  - o **Presence of Atrial Fibrillation → ↑Risks of:**
    - § Hypotension (due to ↓Cardiac Output)
    - § Pulmonary Congestion (Due to L-Heart Failure)
    - § Thrombus Formation (Due to pooling of blood in Atria)
  - o **Treatment:**
    - § **Ventricular Rate Control –** (B-Blocker / Ca-Ch-Blocker [Verapamil] / Digoxin)
    - § **Anticoagulation –** (*Warfarin* or other)
    - § **Cardioversion –** (Medical [Sotalol/Amiodarone] or Electrical)



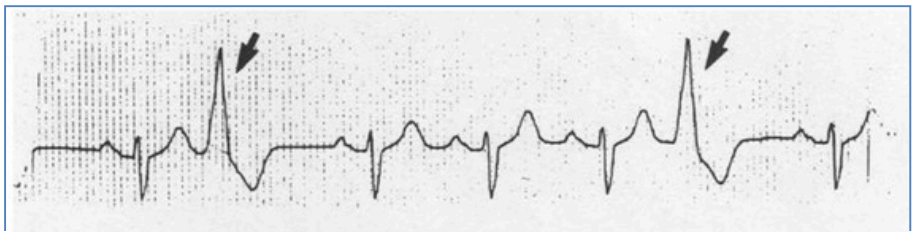
- **PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT):**
  - o **= Sudden Onset Regular Tachycardias (Typically Atrial Re-Entry)**
  - o § Rate ≈ 130+bpm (Regular)

  Diagnosis:
    - § **ECG**
    - § **Adenosine Trial –** (Dromotropic → Slows SA-Node) :. If Rate slows = SVT.
      - (If not, consider ventricular cause)
  - o **Management:**
    - § **Rate Control –** (B-Blocker / Ca-Ch-Blocker [Verapamil] / Digoxin)
    - § **(Definitive – Catheter Ablation.)**

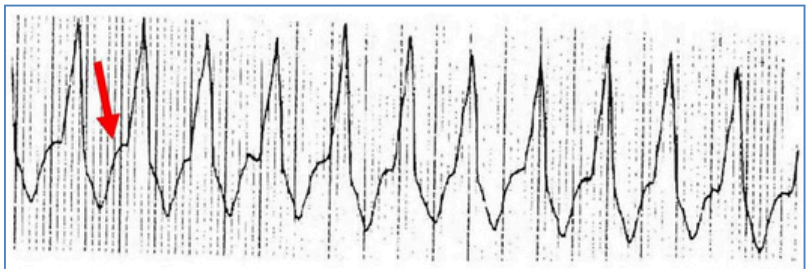


VENTRICULAR TACHYCARDIAS:

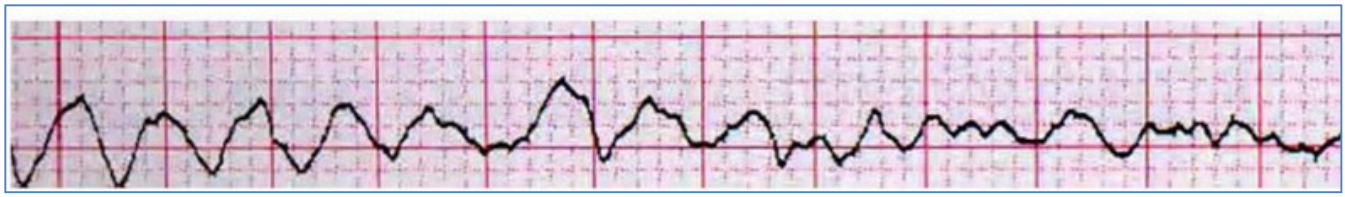
- **PREMATURE VENTRICULAR (QRS) COMPLEXES (PVC):**
  - o **= Additional QRS's with No Preceding P-Wave**.
    - § Wide QRS & Bizarre Shape
  - o **Complication –** Consecutive PVCs = *VENTRICULAR TACHYCARDIA*.
  - o **Causes:**
    - § Normal in Adolescents/Young Adults (Once/twice a day)
    - § Heart Disease
    - § Hypokalaemia (Low K+ levels) → Hyperpolarises the cell
    - § Hypoxia
  - o **Treatment:**
    - § Sometimes Requires no Treatment (If only occasional)
    - § Potassium Supplements
    - § B-Blocker – (if Symptomatic)



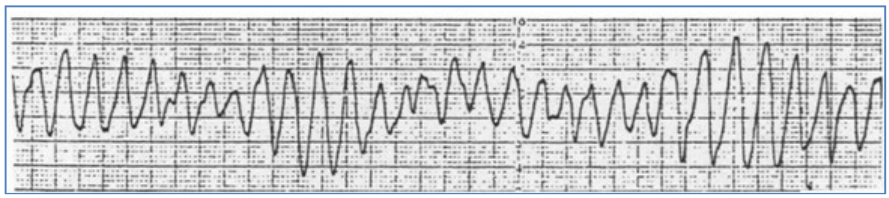
- **VENTRICULAR TACHYCARDIAS:**
  - o **= 3 or more Consecutive Premature Ventricular Complexes**.
    - § ***Sustained* Ventricular Tachycardia** = If it persists for more than 30s.
    - § ***Non-Sustained* Ventricular Tachycardia** = if it self-terminates
  - o SA-Node Activity is often overwhelmed by QRS Complex.
  - o T-Waves & P-Waves are Unclear.
  - o **Mechanism:** Re-Entry
  - o **Treatment – If *Sustained* (>30s):**
    - § **Cardioversion**
    - § **+/- Anti-Arrhythmic Drugs** (Type 1a Antiarrhythmics [Eg: Procainamide])



- **VENTRICULAR FIBRILLATION:**
  - o **= Disordered, Rapid Ventricular Depolarisation with NO Coordinated Contraction**.
    - § **No Coordinated Contraction → No Cardiac Output**
    - § **A Cause of "Sudden Death"**
  - o Often **Triggered by** an episode of **Premature Ventricular Complexes** or **Ventricular Tachycardia**.
  - o **Treatment:**
    - § **Defibrillation** – (Much Stronger than Cardioversion & isn't timed)
    - § **+/- CPR**
    - § +/- Anti-Arrhythmic Drugs.



- **TORSADES DE POINTES "Twisting of the Points":**
  - **= A Polymorphic Ventricular Tachycardia with QRS-Complexes of Changing Amplitude**
    - Rate ≈ 200-250bpm
    - ECG *appears* to be 'twisting around'.
  - **Causes:**
    - Long-QT-Syndrome (An inherited ion channel mutation)
    - (Drugs) Eg: K+ Channel Blockers
    - Electrolyte Disturbances – (Hypokalaemia / Hypomagnasaemia)
  - **Management:**
    - IV Magnesium
    - Temporary Pacing
    - DC Cardioversion - (If Haemodynamic Compromise)

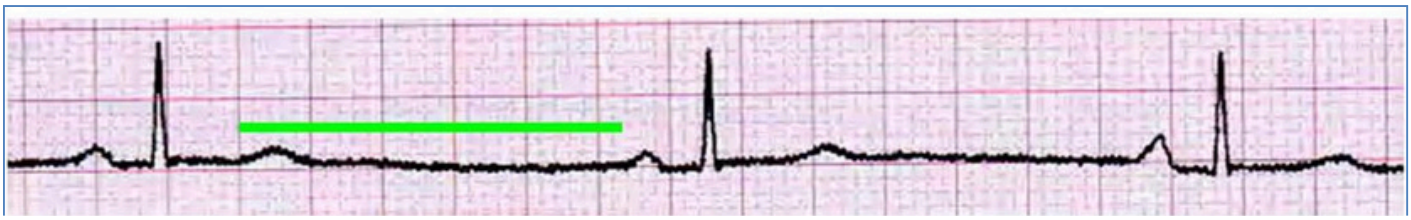


COMMON BRADYCARDIAS:

Get Direction GLOBAL

SINUS BRADYCARDIA:

- **= Sinus Rhythm of <60 Beats/min** (SA-Node is still the pacemaker)
  - o Prolonged TP-Interval; All Waves Visible
  - o All waves clear & visible
- **Occurs Normally:**
  - o At rest/Sleeping (Parasympathetic-NS)
  - o In Elite Athletes (Because SV is Higher)
  - o With Negative-Chronotropic Drugs (Ie: Meds that depress SA-Node Activity)
- **Pathological Causes:**
  - o Depressed Intrinsic Automatic SA-Node Firing (Eg: Due to Ischaemic Heart Disease/Old Age)
  - o Cardiomyopathy
- **Management:**
  - o ***Atropine*** (If symptomatic) (+/- Pacing)

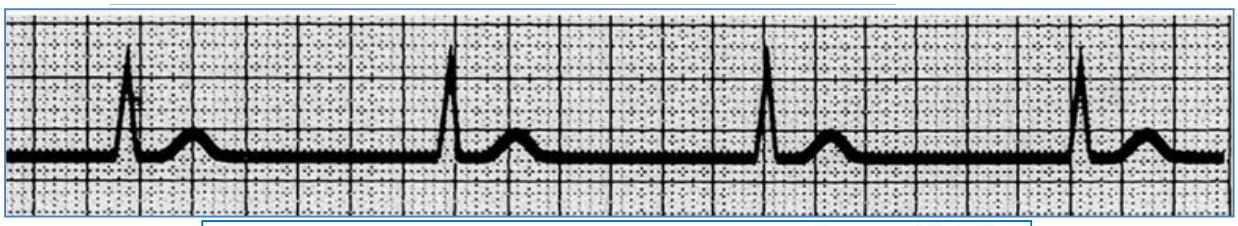


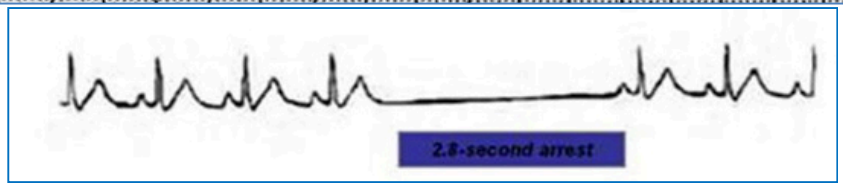
ESCAPE RHYTHMS (SINUS ARREST/EXIT BLOCK):

= SA-Node Failure (No P-Wave)

- o → Hence, the pace is set by the next available Node, the AV-Node → AV-Nodal *'Escape Rhythm'*.
- o → The 'Pacemaker' Impulse is Initiated by the AV-Node → Sets the rhythm.
- o **AV-Node has a slower *Intrinsic Rate* ≈ 40-60bpm (Compared to the SA-Node's 90-100bpm)**

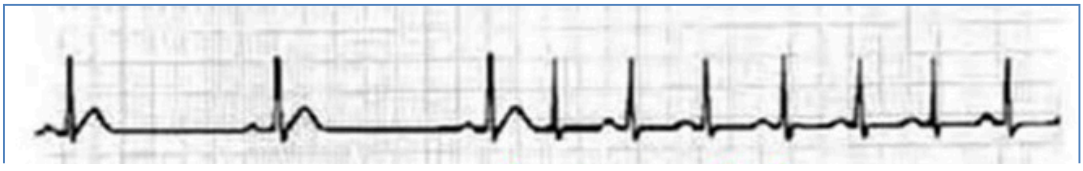
- **Management:**
  - o **Cease Dromotropic Drugs** – (*B-Blockers* / *Ca-Ch-Blockers* / *Digoxin*)
  - o In patients with complete AV block, high-grade AV block, or symptomatic sick sinus syndrome (ie, sinus node dysfunction), **a permanent pacemaker** may be needed





BRADY-TACHY SYNDROME:

- **= Intermittent Episodes of SA-Node Bradycardia & Tachycardia**
  - o Due to SA-Node Instability
  - o Common in Elderly
- **Management**
  - o Requires a pacemaker



CONDUCTION BLOCKS:

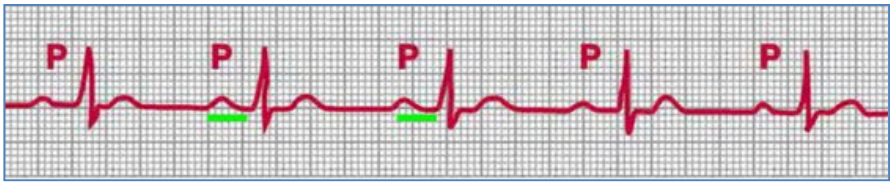
Get Direction GLOBAL

General Info About Conduction Blocks:

- **= Impaired Conduction between Atria & Ventricles**
  - o Commonly resulting from Ischaemic Damage to Nodal Tissue
  - o Often Involves an Escape Rhythm.
- **Types of Conduction Blocks:**
  - o Between Atria & Ventricles (Ie: Vertical (AV) Conduction Block)
  - o OR...Between L-Heart & R-Heart (Ie: Lateral Conduction Block)

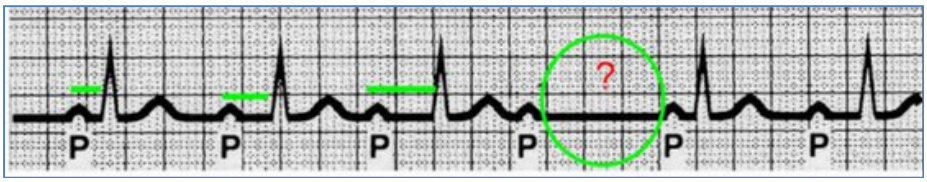
AV-CONDUCTION BLOCKS → 1 OF 3 DEGREES:

- **1- FIRST-DEGREE HEART BLOCK:**
  - o **= Prolonged delay between Atrial & Ventricular Depolarisation. (Greater than 0.2sec)**
    - § → Prolonged PR-Interval
  - o **1:1 Relationship between P-Waves & QRS-Complex is Maintained.**
  - o No Real Symptoms (Treatment Not Necessary)

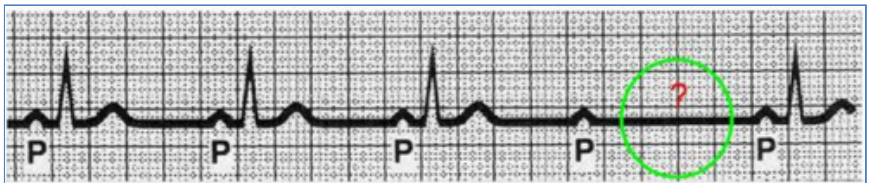


- **2- SECOND-DEGREE HEART BLOCK:**
  - o **MOBITZ TYPE-I (WENCKEBACH):**
    - § **= Gradual Lengthening of PR-Interval until a QRS is blocked.**
    - § • →Some P-Waves aren't followed by QRS-Complexes
    - § Minimal Symptoms

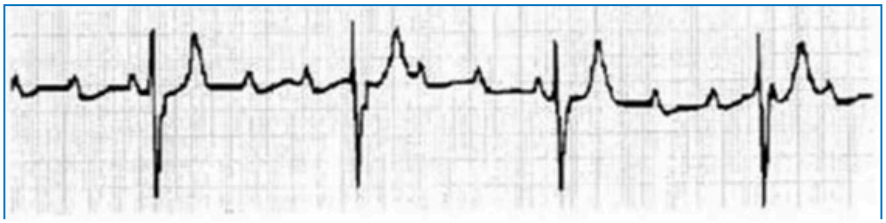
      Management: Nil; (*Atropine* – if Symptomatic)



  - o **MOBITZ TYPE-II:**
    - § **= Loss of AV-Conduction WITHOUT lengthening of PR-Interval (PR-Interval is Fixed)**
    - § Block may last for 2/more beats.
    - § **Management: Pacemaker**



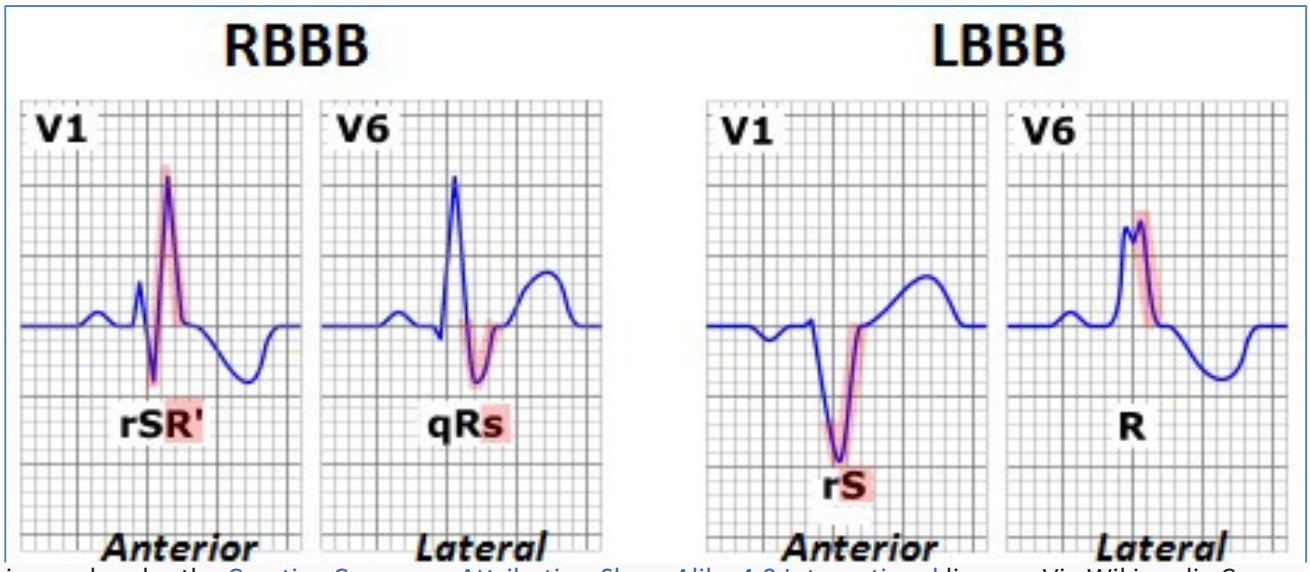
- **3- THIRD-DEGREE HEART BLOCK (AKA: COMPLETE HEART BLOCK):**
  - o **= Complete AV-Conduction Failure.**
    - § No P:QRS Relationship
    - § ↓Cardiac Output (Disordered Contraction of Atria & Ventricles)
  - o **Management: Pacemaker.**



BUNDLE BRANCH (LATERAL) BLOCKS (IE: @ L/R BUNDLE-BRANCHES):

- **RIGHT BUNDLE-BRANCH BLOCK:**
  - o = **When Right Bundle-Branch is unable to conduct impulses to R-Ventricle**.
    - § Therefore, L-Bundle-Branch depolarizes L-Ventricle First, then the impulse travels to R-
    - § Ventricle causing it to depolarize.
      Ie: Ventricles depolarize *Consecutively* rather than *Simultaneously*.
  - o → **Widened QRS-Complex**

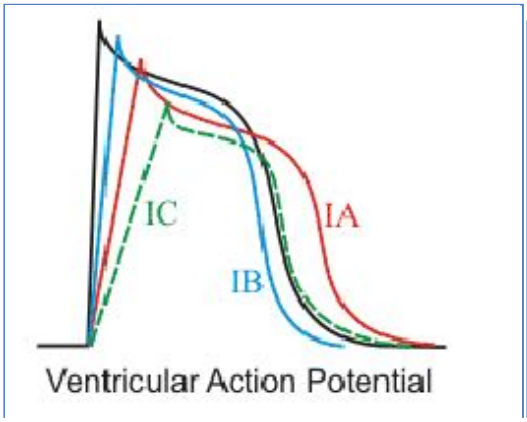
- **LEFT BUNDLE-BRANCH BLOCK:**
  - o = **When Left Bundle-Branch is unable to conduct impulses to L-Ventricle**.
    - § Therefore, R-Bundle-Branch depolarizes R-Ventricle First, then the impulse travels to L-
    - § Ventricle causing it to depolarize.
      Ie: Ventricles depolarize *Consecutively* rather than *Simultaneously*.
  - o → **Widened QRS-Complex**



DRUG CLASSES FOR TREATING ARRHYTHMIAS:

Class-I Antiarrhythmics (Voltage-Gated-Na+ Channel Blockers):

- **Indication:**
  - Typically Re-Entrant Tachycardias (But Not 1st line)
    - SupraVentricular Tachycardia
    - Ventricular Tachycardia
    - Preventing Ventricular Fibrillation
- **Mechanism of Action:**
  - **Selective only for Voltage-Gated-Na+ Channel Blockade (in Contractile Cells):**
    - Slows down Re-Entrant Foci → Restores SA-Nodal Control of HR.
    - (Blocking the Fast-Na+ Channels reduces the *Rate* of depolarisation, prolonging the Action Potential duration → Therefore Reducing Ventricular Rate)
- **Typical Agents:**
  - **1a** – Quinidine, Procainamide (Intermediate Association/Dissociation)
  - **1b** – Lidocaine, Tocainide (Fast Association/Dissociation)
  - **1c** – Flecainide, Encainide (Slow Association/Dissociation)



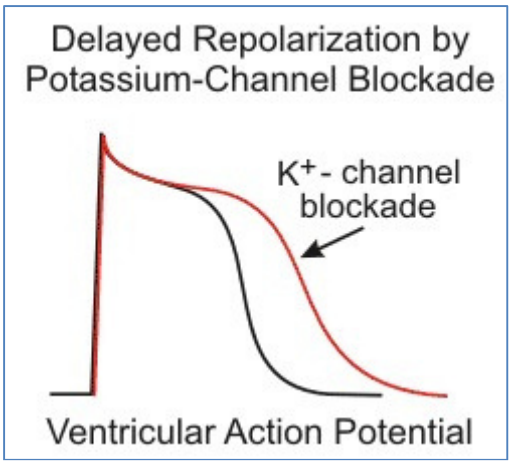
- Class IA: e.g., quinidine
  - Moderate Na^+-channel blockade
  - ↑ ERP
- Class IB: e.g., lidocaine
  - Weak Na^+-channel blockade
  - ↓ ERP
- Class IC: e.g., flecainide
  - Strong Na^+-channel blockade
  - → ERP

Class-II Antiarrhythmics (β1-Blockers):

- **= β1-Adrenergic Receptor Antagonists**
  - Note: There are 3 types of β-Receptor:
    - β1-Receptors: in Heart & Kidneys
    - β2-Receptors: in Lungs, GIT, Liver, Vascular Smooth Muscle, Skeletal Muscle
    - β3-Receptors: in Adipose Tissue
- **Classical Agents:**
  - **Propanolol
  - Atenolol
- **Mechanism of Action:**
  - β1-Adrenergic Receptor Blockade → Inhibit Sympathetic NS →
    - Conductile System → **↓HR**
    - Contractile Cells → **↓Contractility**
- **Indications:**
  - Atrial Fibrillation (Or other Sinus Tachycardia)
  - SVT
  - (Hypertension.)
  - Ischaemic Heart Disease →↓Cardiac Workload (Ie: ↓Metabolic Demands)
- **Contraindications:**
  - Asthma → Can cause Bronchoconstriction.
  - Ca+ Channel Blockers (Verapamil/Nifedipine) → Can cause Fatal Bradycardia.
- **Side Effect:**
  - Reduced Renin Release → ↓Aldosterone → ↓Na & H2O Retention → ↓Blood Pressure.
  - Sinus Bradycardia.
  - Bronchoconstriction in Asthmatic Patients.
  - (Rebound Tachycardia – if stopped abruptly; Must be weaned off)

Class-III Antiarrhythmics (VG-K+ Channel Blockers):

- Affect *VOLTAGE-GATED* K+ Channels in Nodal Cells & Myocytes.
- **Classical Agents:**
  - o **Amiodarone
- **Mechanism of Action:**
  - o VG-K+ Channel Blockers → Prolongs Plateau Phase of AP → ↓HR
  - o (Blocking K+ Channels prevents/slows K+-Efflux during Repolarisation of Cardiac Action Potentials. This prolongs the Repol. Phase → ↓Heart Rate.)
  - o Prevent Re-Entrant Arrhythmias (Atrial Flutter, Atrial Fibrillation, Ventricular Tachycardias) by prolonging the repolarisation phase of the action potential (Therefore prolonging the Refractory Period).
- **Indications:**
  - o *1st Line in *Re-Entrant Tachycardias*.
    - § Atrial Flutter
    - § Atrial Fibrillation
    - § Ventricular Tachycardias
    - § Ventricular Fibrillation
- ***KEY* Side Effect/s:**
  - o Bradycardia
  - o Early-After-Depolarisation (PVCs/Ectopic Beats)

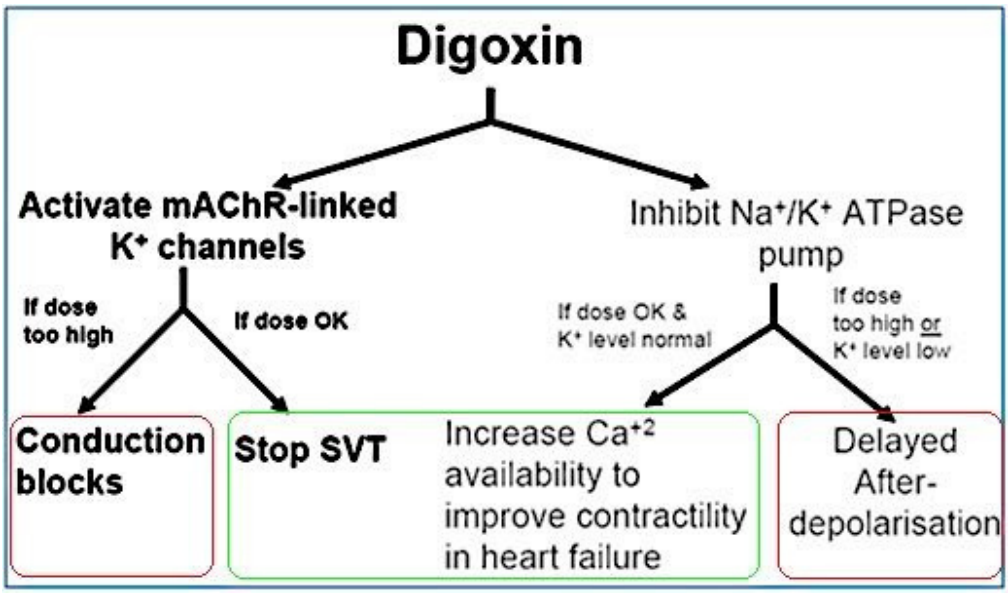


Class-IV Antiarrhythmics (VG-Ca+ Channel Blockers):

- **Affects *VOLTAGE-GATED* Ca+2 Channels in BOTH Nodal Cells & Myocytes**
  - o **Effect on Nodal Cells (Conductile Cells):**
    - § Blocking Ca+ Channels will slow the Depolarisation of Conductile Cells, thereby reducing their firing rate → Decreases Heart Rate (Negative Chronotropic Effect)
  - o **Effect on Myocytes (Contractile Cells):**
    - § Blocking Ca+ Channels will decrease Ca+ Influx into the Myocyte during the Plateau Phase of the Action Potential → Decreased Contractility (Negative Inotropic Effect)
- **Classical Agents:**
  - o **Verapamil – (Selective for the Heart)
  - o Nifedipine – (Selective for Vessels) – (Used in Angina & Heart Failure)
- **Indications:**
  - o SVT (Supraventricular Tachycardias)
  - o Variant Angina (Works on **Vascular Smooth Muscle** → Vasodilation → ↓BP & ↓Afterload)
- **Contraindications:**
  - o β-Blockers– (Since Ca+ Channel Blockers also Inhibit Ca+ Influx) → Fatal Bradycardia.
- ***KEY* Side Effect/s:**
  - o Heart Block
  - o Bradycardia
  - o (Also Hypotension/Dizziness due to ↓Contractility)

Digoxin:

- **2x Clinical Uses:**
  - **1- Heart Failure** (Especially Pts with coincident Atrial Fib. – 'Kill 2 birds')
  - **2- Long Term SVT (Eg: AF) Management**
- **2x Mechanisms of Action:**
  - **1- Inotropic: Myocytes: Na/K-ATPase Inhibitor** → ↑Contractility.
    - **Use:** Heart Failure
    - **Side Effect:** "Early After Depolarisations" (Ectopic Beats/SVT)
  - **2- Dromotropic: AV Node: K+ Channel Agonist** → Slows AV Conduction.
    - **Use:** SVT (Supraventricular Tachycardia)
    - **Side Effect:** Heart Block (if HR <60bpm)
- **Summary of Actions & Potential Side Effects:**
  - Note: Not to be given if HR less than 60bpm → Brady/Heart Block.
  - Note: Also, Dosage is very important for reducing side effects.
  - *(Note: Also require K+ Monitoring - & Supplements if on K+ Wasting Diuretic)



Adenosine:

- **Clinical Use:**
  - Diagnostically to distinguish V-Tac from SVT.
  - Note: Extremely short T1/2 - Only Effective in Emergency Situations to stop SVT.
    - (Digoxin is used for long-term SVT Management)
- **Mechanism of Action:**
  - **Adenosine Receptor Agonist @ SA & AV Nodes → Delays AV-Node Conduction.**
  - (HR will slow if it is an SVT) / (If HR is unchanged, then it is V-Tac)
- **Side Effect/s:**
  - *IMPENDING DOOM*!!! (Pts literally feel like they're dying).

Atropine:

- **Clinical Use:**
  - Acute Bradycardias/Asystole →↑HR. (However can cause V-Tac).
- **Mechanism of Action:**
  - **Chronotropic:** Anti-Muscaranic (Blocks Parasympathetic NS)→↑HR.
- ***KEY* Side Effect/s:**
  - Overdose → Ventricular Tachycardia

DYSLIPIDAEMIA

Dyslipidaemia = a blanket term for Elevated Blood levels of Fats (cholesterol and/or triglycerides).

- **Review of physiology of cholesterol and other lipids:**
  - **Five Lipid Transporters:**
    - **1- Chylomicrons** – Made by Small Intestine:
      - Transport Dietary Fats from SI → Liver (Via Lymph).
    - **2- Very Low Density Lipoproteins (VLDL's)** – Made by Liver:
      - Transports Fats from Liver → Tissues.
    - **3- Intermediate Density Lipoproteins (IDL's)** – Made by Liver:
      - Essentially a VLDL with some lipid and protein removed.
    - **4- Low Density Lipoproteins (LDL's) – BAD**
      - Delivers Cholesterol to Liver and Tissues.
      - Note: ↑Fat Consumption → ↑[LDL] →ATHEROSCLEROSIS
    - **5- High Density Lipoproteins – GOOD**
      - Cholesterol Re-Uptake from Tissues → Liver
- **Aetiologies:**
  - **Primary Hyperlipidaemias (Genetic):**
    - Eg: Familial Hyperlipidaemia
    - Eg: Lipoprotein lipase deficiency
  - **Secondary Hyperlipidaemia (Acquired):**
    - Eg: Obesity
    - Eg: Hypothyroidism
    - Eg: Diabetes mellitus
    - Eg: Nephrotic syndrome
    - Eg: Liver Failure
    - Eg: Drugs: (Eg: Oral contraceptives/Retinoids/thiazide diuretics)
- **Diagnosis & Screening (for high risk Pts):**
  - **FamHx** of CVD/IHD/MI/↑Cholesterol
  - **Physical Signs** – (Xanthomata, Xanthelasma)
  - **Comorbidities** – (Eg: Obesity, Diabetes, HTN, Hypothyroid).
- **Investigations:**
  - **Serum TGLs** - *(Normal = <2mmol/L)*:
    - ***>6mmol/L → Requires Intervention - (<6mths Lifestyle Modification → Statin Therapy).**
  - **Cholesterol** - *(Normal = <4mmol/L)*:
    - ***>6.5mmol/L → Requires Intervention - (<6mths Lifestyle Modification→Statin Therapy).**
    - **(Target = <4mmol/L total cholesterol or LDL-CK less than 1.8mmol/L)**
- **Management:**
  - ****1- Lifestyle Modification:**
    - **Diet – (**↓Saturated Fat/Cholesterol Intake, ↑Fibre intake, ↓Alcohol, ↓Smoking, Weight Loss**)**
    - **↑Exercise**
  - ****2- Pharmacological:**
    - ****Statins – (HMG-CoA Reductase Inhibitors):**
      - **Classical Agents:** (Simvastatin, Atorvastatin)
      - **MOA:** HMG-CoA Reductase Inhibitor → ↓Cholesterol Synthesis
      - **Side Effects:**
        - **Statin-Induced Myopathy/Myositis/Rhabdomyolysis** → Muscle Pain/Weakness + ↑CK-Levels
  - **(Other Lipid-Lowering Agents – (Only Recommended If CHD or Intolerant to Statins)):**
    - ***Fibrates:** (*Fenofibrate*)
    - **Bile Acid-Binding Resins – (Ion Exchange Resins):** (*Cholestyramine*)
    - **Ezetimibe:** (*Ezetimibe*)
    - **Fish Oil (***Omega-3***)** – Prophylactic?

ATHEROSCLEROSIS

TERMINOLOGY:

- **"Athero"** = Gruel/Porridge (Ie: The fat in the blood)
- **"Sclerosis"** = Hardening

Lipids: The Main Culprits! (A Review)

- **3 Types of Lipids in Plasma:**
  - **1- Cholesterol + Ch. Esters**
  - **2- Phospholipids**
  - **3- Triglycerides (Fatty Acids + Glycerol)**
- **Lipid Transport:**
  - **Insoluble In Water** → Must be *Packaged* to be suspended in plasma.
  - **Fats Absorbed in GI** → Packaged into ***Chylomicrons*** (in S.I.) → Lymphatics → Lymphatics → Circulation (Left Sub-Clavian Vein) →Liver.
  - **Liver Repackages** Chylomicron Remnants → ***Lipoproteins*** → Circulation

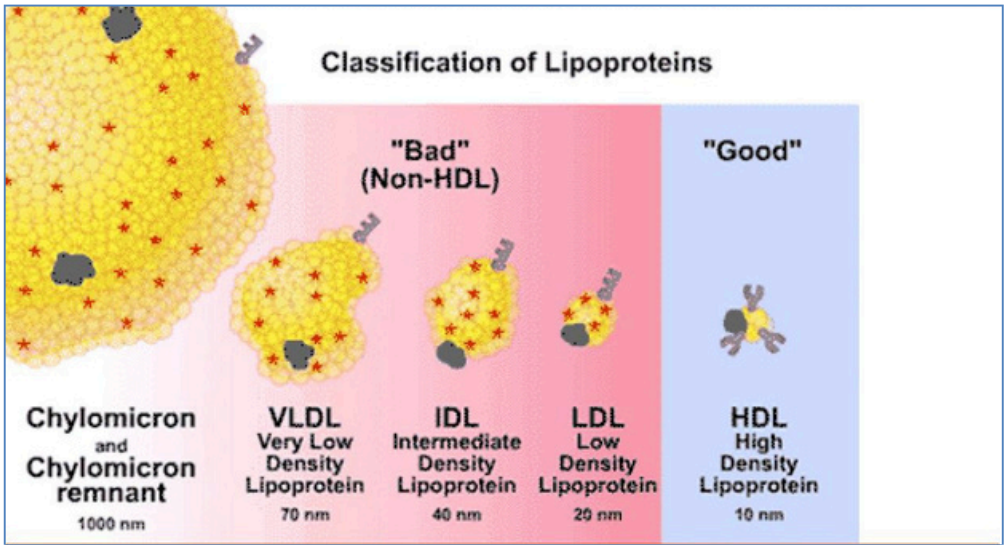
Classification of Lipoproteins-carriers for lipids in the blood

Particle	Source	Predominately transports
Chylomicron	gut	Triacylglycerol
Very-low density Lipoprotein (VLDL)	liver	Triacylglycerol
Intermediate density (IDL)	catabolism	Cholesterol
Low density (LDL)	catabolism	Cholesterol
High density (HDL)	catabolism	N/A
Lipoprotein A	liver,gut	N/A

HDL Inversely related with AS-mops up used cholesterol and also acts as direct cholesterol transport to the liver. HDL also transfers cholesterol into other lipoproteins for subsequent hepatic metabolism **GOOD**

LDL Correlate with AS **BAD**

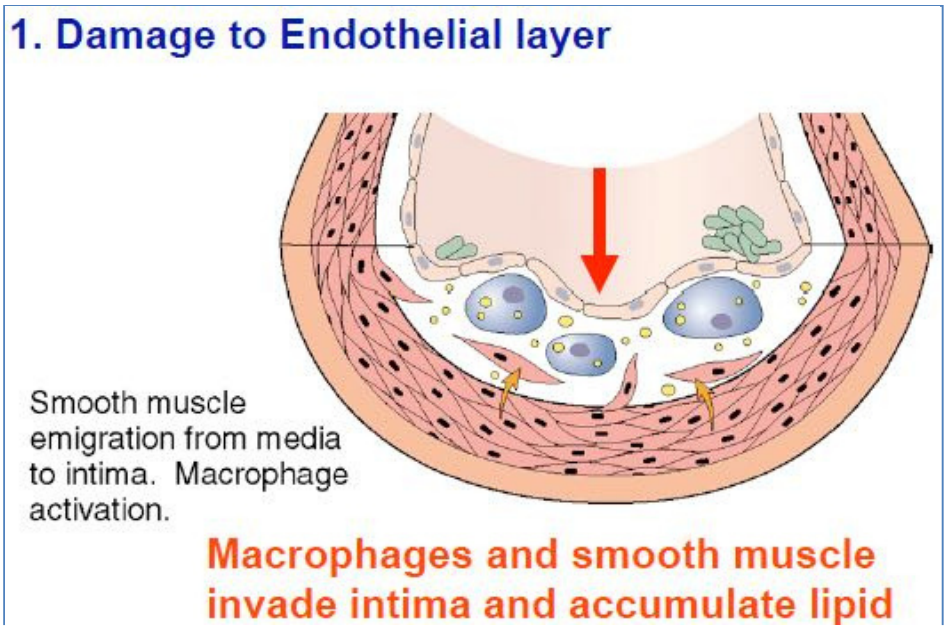
Note: LDLs Contribute to Atherosclerosis
Note: HDLs Help Prevent Atherosclerosis



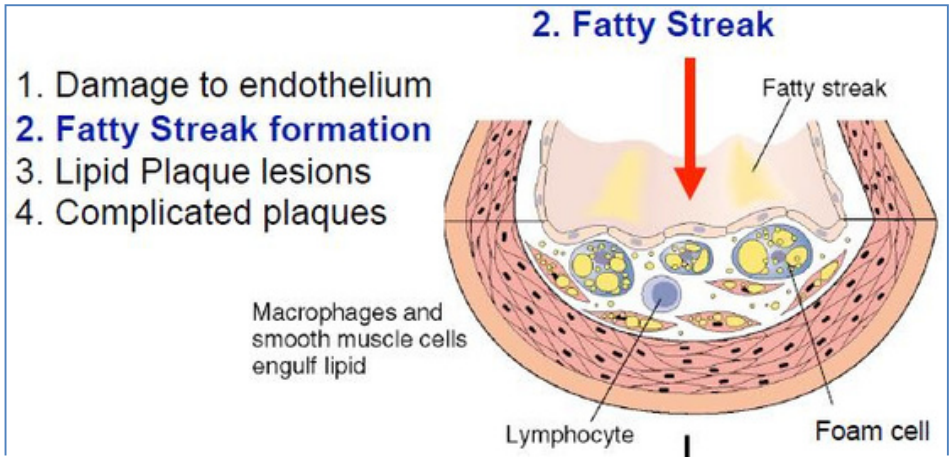
At some crucial BLOOD-concentration of LDL, some of the LDL particles begin to "stick" at certain vulnerable points of injury in the arterial wall.

ATHEROSCLEROSIS:

- ***= A Progressive Chronic Inflammation* of *Arteries* characterised by:**
  - o ***1- Inflammation*,** (Macrophages engulf LDLs → "Foam Cells")
  - o ***2- Fibrosis*,** (Conn. Tissue Matrix/Collagen/Elastin)
  - o ***3- & Lipid Deposition*** (Cholesterol Esters & Cholesterol in Cells)
  - o **("Athero"= Fat, "Sclerosis"= Hardening)**
- **Aetiology:**
  - o BEGINS with Endothelial Injury
  - o BIG Inflammatory Component
  - o **Risk Factors:**
    - § **Non Modifiable:** Age (40-60), Male, FamHx, Indigenous
    - § **Modifiable:** ↑Cholesterol, HTN, Smoking, Diabetes, Obesity, Metabolic Syndrome
- **Pathogenesis**
  - o **1- Endothelial Injury & Activation – (**HTN/Smoking/DM/Turbulence/Toxins/Infection/Immune).
  - o **2- Endothelial Inflammation – (**Macrophage & Smooth Muscle Migration)

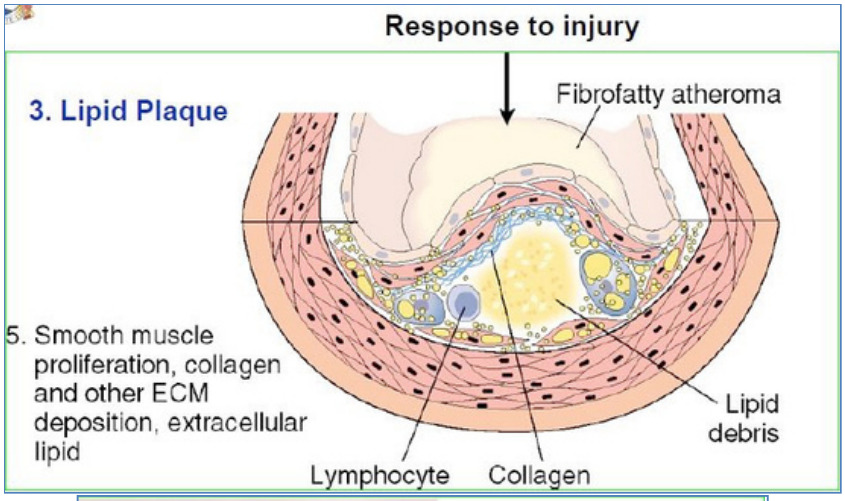


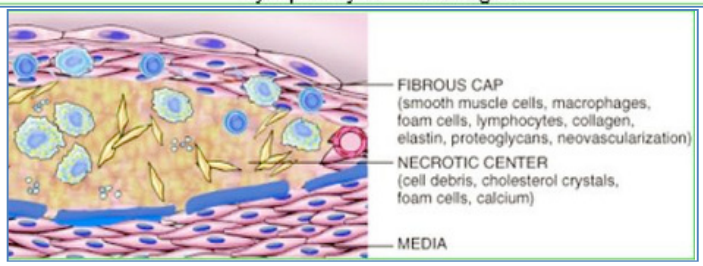
  - o **3- Accum ulation of Lipoproteins → Fatty Streak Form ation**.
    - § **Fat Deposition** Under the Tunica-Intima Vessel-layer.



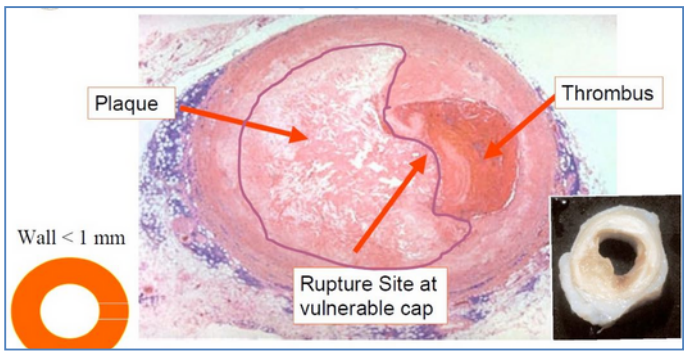
- **4- Proliferation & Fibrosis – (Conversion of *Fatty Streak* into a Mature *Atheroma*)**
  - Fatty Streak gets more profound
  - 'Foam Cells' Unable to Digest Lipid Contents → Die
    1. → Extracellular Lipids
    2. → Cell Debris
  - Oxidised LDLs – Attract Immune Cells/Cytokines/Platelets/Smooth Muscle/Conn. Tissue
  - 1. Positive Feedback.

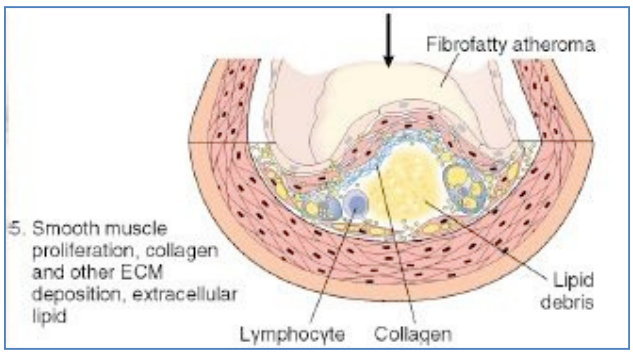
    Plaque Builds.

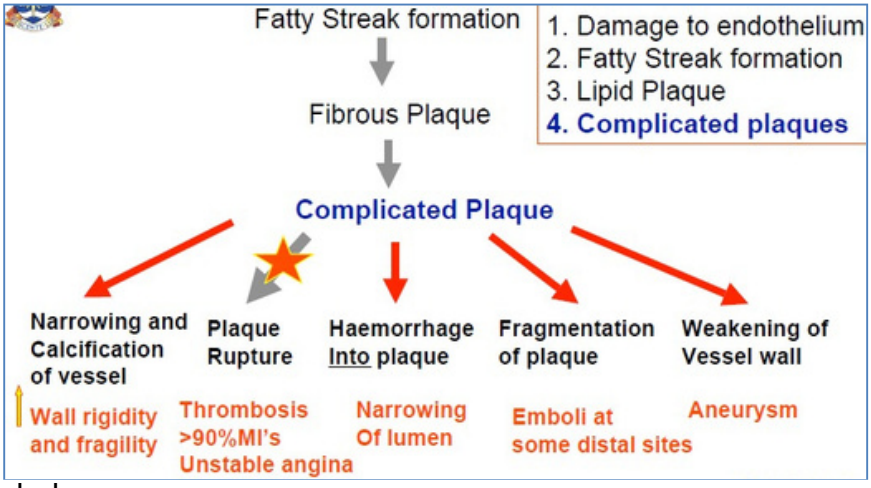




- **5- Complicated plaque formation – (Thin Fibrous Cap** → Rupture → Thrombus → ACS)
  - Reduced blood flow to local area → Imbalance of Supply & Demand.







Clinical Features/Complications:

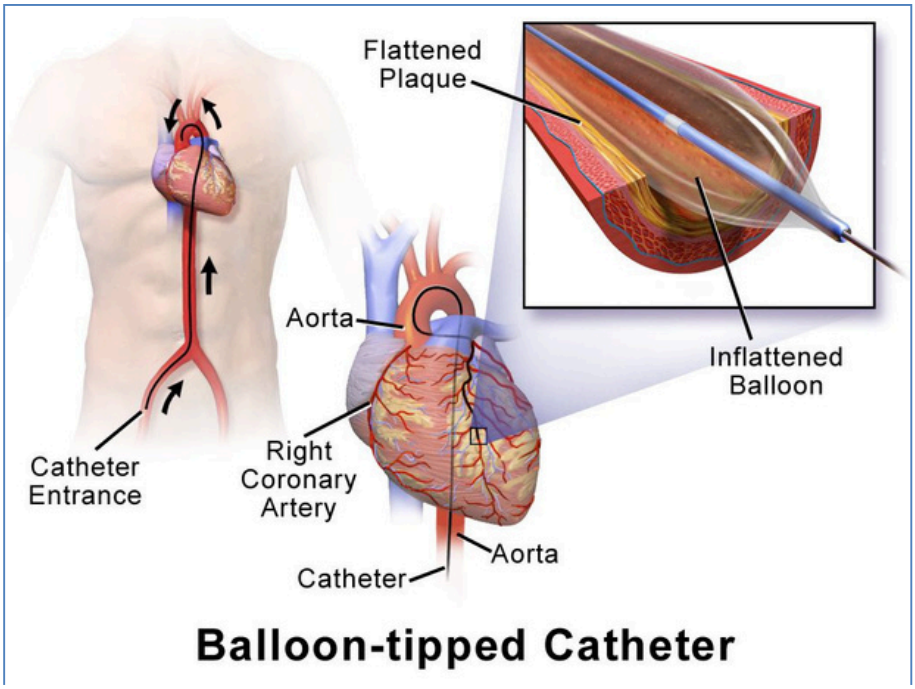
- o ***Multi-Organ*** **Disease:**
  - § **Heart** → IHD (Angina, MI).
  - § **Brain** → Cerebral Infarction (Stroke)
  - § **Kidneys** →Renal Infarction
  - § **GIT** → GI-Ischaemia/Infarction
  - § **Lower Extremities** → PVD (Eg: Claudication, Gangrene of Legs, Arterial Leg Ulcers)

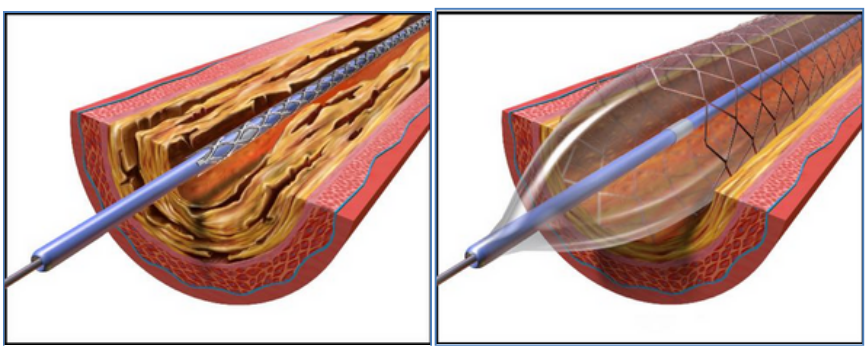
Investigations:

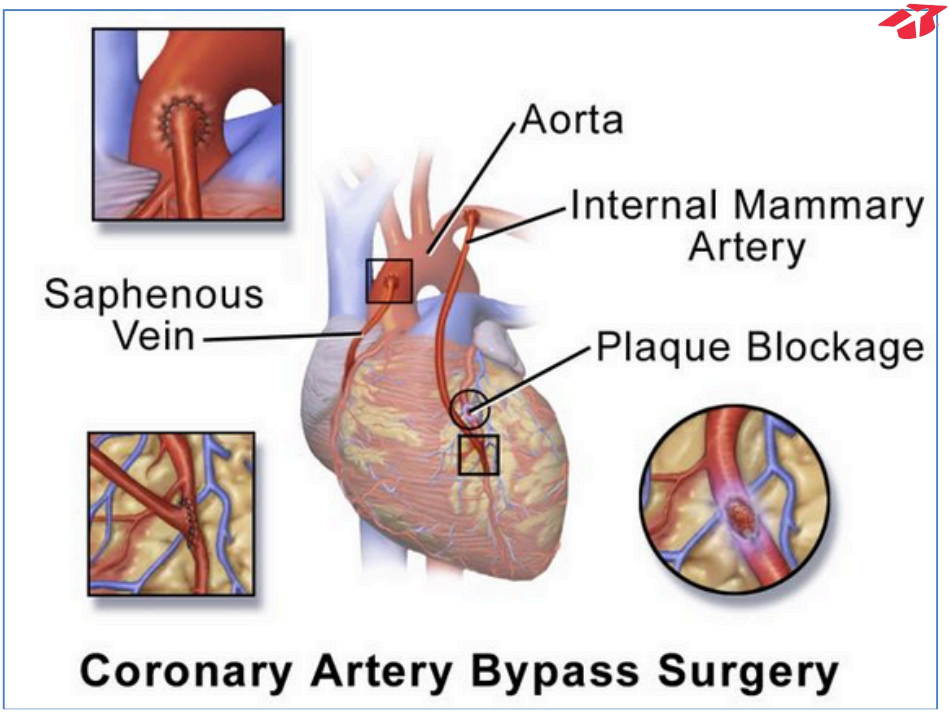
- o **Invasive Method:**
  - § Catheter via *Femoral Artery* → *Coronary Artery* → X-Ray Angiogram.
- o **Non-Invasive Method:**
  - § Contrast-Enhanced CT-Scan
  - § Takes 15sec.

Management:

- o **Risk Factor Modification:**
  - § **Statins** - (↑Cholesterol) **ACE-I/B-**
  - § **Blocker** - (HTN)
  - § **Improve diabetic control**
  - § **Diet & ↑Physical Exercise** (For Obesity)
  - § ↓Smoking, Alcohol
- o **Prevent Thrombosis:**
  - § Aspirin/Clopidogrel
- o **Surgical Intervention:**
  - § **Balloon Angioplasty/Stent AngioplastyBypass Surgery:**

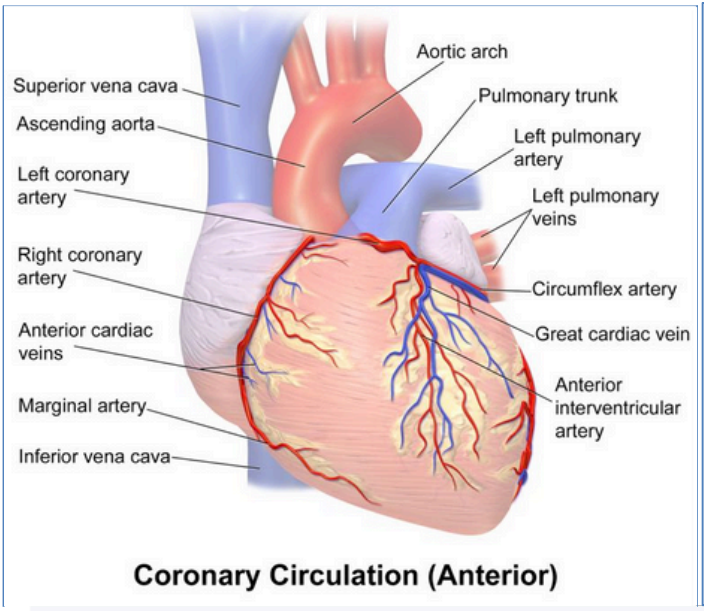


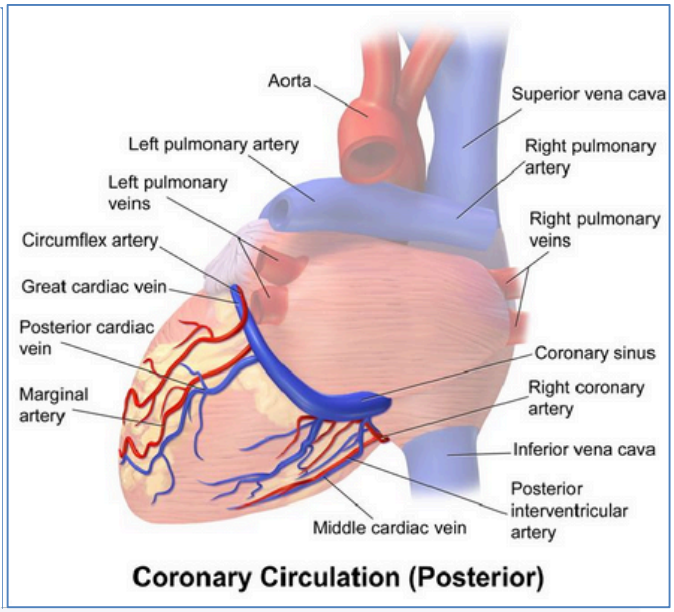




Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2).

Review of Coronary Anatomy:





LAD → Apex, Anterior LV, Anterior 2/3 of IV -Septum
LCX → Lateral LV
RCA → Entire RV, Postero-Superior LV, Posterior 1/3 of IV-Septum

- **Degrees of Coronary Blockage:**
  - **<70% Occlusion:** Asymptomatic
  - **70-75% Occlusion:** Angina
  - **90% Occlusion:** Chronic IHD
  - **Unstable Plaque:** Unstable angina +/- Rupture → Acute MI
  - **> 90% Occlusion:** MI

***Ischaemia Vs. Hypoxia Vs. Infarction:**

- **Ischaemia:** A '*FLOW' Limitation*, Typically due to Coronary Artery Stenosis (Narrowing)
- **Hypoxia:** An '*O2' Limitation,* Typically due to High-Altitude/Respiratory Insufficiency/etc.
- **Infarction:** *Irreversible Cell-DEATH*, Typically due to sustained Ischaemia.

Regional Vs. Global Myocardial Ischaemia:

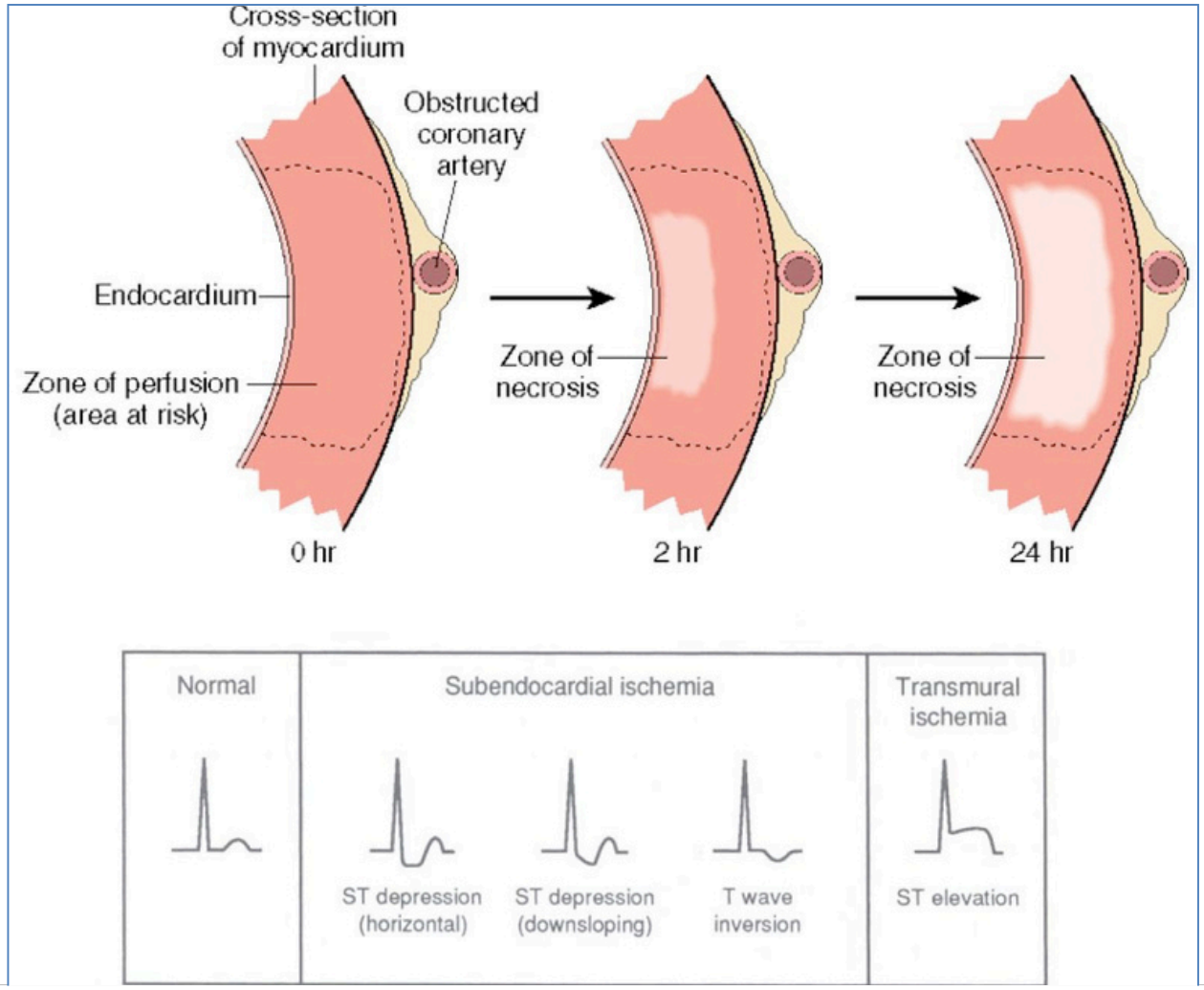
- **Regional Ischaemia:**
- o Local Atherosclerosis/Thrombosis → Ischaemia Confined to Specific *Region* of Heart.

Global Ischaemia (Rare):

o Severe Hypotension/Aortic Aneurysm → Ischaemia of *Entire* Heart

What Happens During Myocardial Ischaemia:

- **Metabolic Changes – (Aerobic → Anaerobic):** ↑Lactate (Anaerobic Metabolism) & ↓pH
- **Pain:** Nociceptor (pain receptor) Activation → Angina Pain
- **Global Autonomic Symptoms:** Tachycardia, Sweating, Nausea.
- **Pulmonary Congestion:** Eg: LV-Failure → Pulmonary Congestion → Shortness of Breath
- **Ventricular Arrhythmias:** Eg: SVT or VT or VF (due to Re-Entrant Focus & Altered Conduction Patterns)
- **Myocardial Damage:** Initially '*Subendocardial*'-Ischaemia/Infarction (ST-*Depression & T-Wave Inversion*)→ Progresses to *'Transmural'*-Ischaemia/Infarction (ST-*Elevation & Pathological Q-Waves*).



Adapted from *Schoen FJ, Mitchell, RN: The heart. In Kumar V, et al, editors:* Robbins and Cotran pathologic basis of disease.

ANGINA PECTORIS:

- **Aetiology:**
  - ↓**Myocardial Perfusion (relative to demand) due to Coronary Insufficiency**.
  - **Causes:** **Atherosclerosis / Vasospasm / Embolism / Ascending Aortic Dissection
  - **Exacerbated by –** (Vent-Hypertrophy, Tachycardia, Hypoxia, Coronary Arteritis (Eg: in SLE))
- **Pathogenesis:**
  - (= *A Late Sign of Coronary Atheroma – Symptoms Imply >70% Occlusion!!*)
  - ("Insufficient Coronary Perfusion *Relative to* Myocardial Demand")
  - ***Stable* Angina:**
    - **Due to:** Stable Atherosclerotic Coronary Obstruction (No Plaque Disruption)
    - **Presentation:** Chest Pain on Physical Exertion, which fades quickly with Rest (minutes)
  - ***Variant/Prinzmetal* Angina:**
    - **Due to:** Coronary Vasospasm (May not be Atheroma).
    - **Presentation:** Angina *Unrelated to Activity* (Ie: At Rest)
  - ***Unstable* Angina ("Pre-Infarction Angina"):**
    - **Due to:** Unstable Atherosclerotic Plaque (+/- Plaque Disruption & Thrombus).
    - **Presentation:** Prolonged Angina @ Rest (Either New-Onset/↑Severity/↑Frequency).
    - **Note: = Red Flag that MI may be Imminent
  - ***Silent* Ischaemia:**
    - **Due to:** Ischaemia masked by neuropathy (Eg: Diabetes/↓B12/etc)
    - **Presentation:** *Painless*, but may have Nausea, Vomiting, Diaphoresis + Abnormal ECG
- **Clinical Features of Angina:**
  - **Common Presentation:**
    - **<15mins of *Crushing, Central, Retrosternal* Chest Pain → Radiating to Arms, Neck or jaw:
      - (**Stable:** On exertion)(**Prinzmetal:** Rest)(**Unstable:** Worsening/Prolonged/@Rest)
    - +Dyspnoea (Pulmonary Congestion)
    - + Fear of Impending Doom
  - **Signs:**
    - ↑Sympathetic Drive → Diaphoresis
    - Hypotension → Cold/Clammy/Peripheral Shut-Down/Thready Pulse
    - Pulmonary Congestion → Dyspnoea, ↑JVP
- **Investigations:**
  - **(1st Line) Resting ECG:**
    - During Attack: ST-Depression, T-wave Inversion (Normal between Attacks)
    - (Path-Q-Waves if Previous MI).
  - **(2nd Line) Cardiac Stress Test + ECG:** Suggests Severity of CAD – (Any ST Depression is a +Ve Result)
  - **(3rd Line) Stress Echocardiography:** Assess Ventricular Function
  - **(4th Line) Coronary Angiography (Cath-Lab):** Pre-Angioplasty to Map the Coronary Anatomy
  - **(5th Line) Myocardial Perfusion Scans (Nuclear Medicine)**
- **Management/Treatment:**
  - **(Prevention/Management of CV Risk Factors):**
    - Smoking/Hypertension/Hyperlipidaemia/Diabetes/Obesity/Etc.
  - **Medical Therapy (*Maintenance*):**
    - **1- Anti-Anginal Therapy:**
      - **Nitrates (*GTN*)** – Coronary Vasodilation →↑Cardiac Perfusion
      - **B-Blockers (*Metoprolol*)** – To ↓Workload of the Heart
      - **Ca-Channel Blockers (*Diltiazem/Verapamil*)** – To ↓Afterload
    - **2- Antiplatelet Therapy:**
      - ***Aspirin / Clopidogrel***
    - **3- Lipid-Lowering Therapy:**
      - ***Atorvastatin/Simvastatin***
  - **Revascularisation (*Definitive*) - *OPTIONAL*:**
    - **PCI – (Per-Cutaneous Intervention)/Coronary Angioplasty:**
      - Balloon Dilation/Stenting of Coronary Arteries via Femoral Artery
    - **OR: CABG - (Coronary Artery Bypass Grafting):**
      - Harvested Vein (Saphenous/Wrist) → *Bypasses* the blockage

ACUTE CORONARY SYNDROMES - (STEMI/NSTEMI):

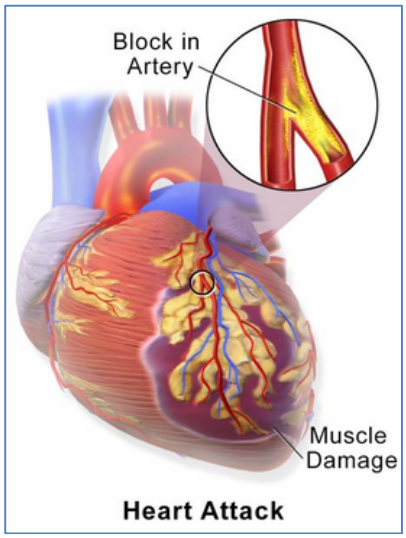
- **Aetiology:**
  - ***Unstable Atheroma***

Pathogenesis:

- ***Unstable Atheroma*** → **Rupture** → ***Prolonged*** **Ischaemia** → ***Necrosis/Death of Myocardium***.
  - (→ Sudden Death, Acute Systolic Dysfunction & Heart Failure, Vent.Rupture)
  - **Progression of Ischaemic Necrosis & "ST-ELEVATION?":**
    - **1-** ***Initially*** **"Subendocardial Necrosis" →** ***NON-ST-ELEVATION MI*****:**
      - *ST-Depression + T-Wave Inversion (As with Angina)*

    2- ***Then*** **"Transmural Necrosis" →** ***ST-ELEVATION MI*****:**
      - *ST-Elevation + T-Wave Inversion + Pathological Q-Waves*

    Note: The Endocardium is spared due to O2/Nutrients of Ventricular Blood.



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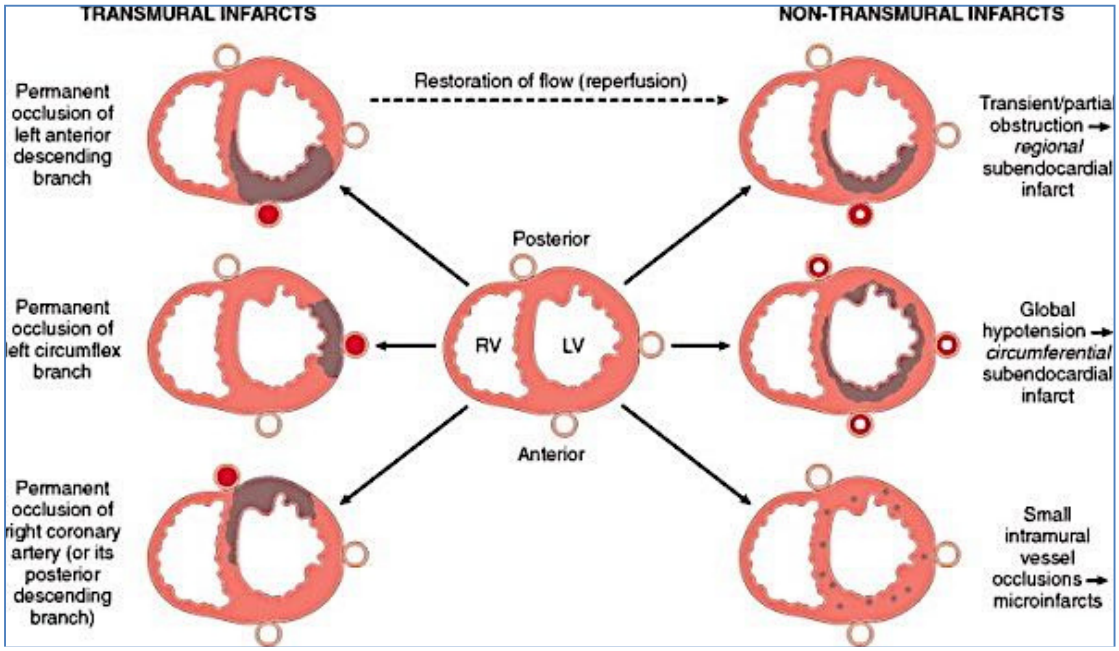
- **Most Common Coronary Obstruction & Locations of Ischaemia:**
  - **50% - LAD Obstruction:**
    - Anterior-LV + Apex + Ant.2/3 of IV-Septum

  30% - RCA Obstruction:
    - Posterior-LV + Posterior Septum + Free wall of RV.

  20% - LCX (Left Circumflex) Obstruction:
    - Lateral LV (except for the apex.)

  (Note: Nearly ALL Infarcts involve a portion of the LV)



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Clinical Features of NSTEMI/STEMI:

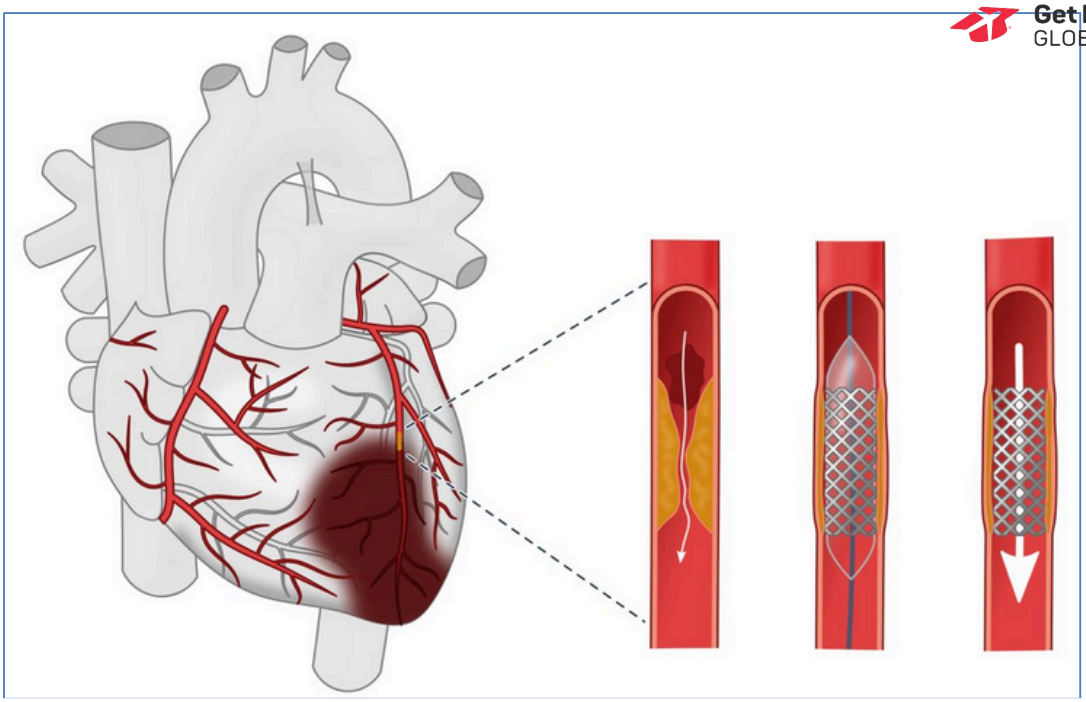
- o **Common Presentation:**
  - § **>20mins *Crushing, Central, Retrosternal* Chest Pain → Radiating to Arms, Neck or Jaw.
  - §
    - (Note: Some are "Silent" – Eg: Diabetes, Post Cardiac Surgery, Elderly)
  - § +Dyspnoea (Pulmonary Congestion)
    + Fear of Impending Doom
- o **Signs:** ↑Sympathetic Drive → Diaphoresis
  - § Hypotension → Cold/Clammy/Peripheral Shut-Down/Thready Pulse
  - § Pulmonary Congestion → Dyspnoea/Tachypnoea/↑JVP
  - § Signs of PVD
  - §

Investigations:

- o **(1st Line):**
  - § **Serial Resting 12Lead ECGs – (Every 15 Mins):**
    - **ST-Changes and Diagnosing MI:**
      - o **V1, V2, V3, V4 = Anterior MI**
      - o **II, III, AVF = Inferior Wall MI**
      - o **I, AVL, V5, V6 = Lateral**
  - § **3-Lead Cardiac Telemetry – (Screening for Arrhythmias)**
  - § **Serial Troponin Levels (Cardiac Troponin-I/T, or CK-MB):**
    - 1st. On Presentation
    - 2nd. @ 6hrs (↑Troponin = MI)
    - 3rd. Within 24hrs
  - § **+ Bloods – (**FBC, Serum Electrolytes, Glucose, Lipids**)**
- o **(2nd Line):**
  - § **TTE/TOE – Transthoracic/Transoesophageal Echo:**
    - Assess LV-Function
    - (+ Excludes DDXs - Aortic Dissection / Pericarditis / Pulmonary Embolism)
  - § **Myocardial Perfusion Scans (Nuclear Medicine):**
    - ? Location of Infarct

Management (As with Angina *PLUS MORPHINE, O2 & ANTICOAGULATION + DEFINITIVE Mx*):

- o ***(Simplified: MONA = Morphine, Oxygen, Nitrates, Aspirin)***
- o **1- Medical Therapy (*Maintenance*):**
  - § **1- Anti-Anginal Therapy:**
    - Nitrates (GTN/Isosorbide Mononitrate) – Coronary Vasodilation →↑Cardiac Perfusion
    - B-Blockers (Propanolol/Metoprolol) – To ↓HR & Contractility → ↓Cardiac Workload
    - Ca-Channel Blockers (Nifedipine/Verapamil) – To ↓Afterload → ↓Cardiac Workload
  - § **2- Antiplatelet Therapy:**
    - (Aspirin / Clopidogrel)
  - § **3- Lipid-Lowering Therapy:**
    - (Atorvastatin/Simvastatin)
  - § **+4- Morphine**: (Analgesia + Vasodilation)
  - § **+5- Oxygen:** (To Maximize O2 @ Myocardium)
  - **+6- Anticoagulation:** (Heparin/LMWH or Warfarin) – (Prevent Further Thrombogenesis).
- o **2- STAT Revascularisation (*Definitive*) – *WITHIN 4 HRS*:**
  - § ****PCI – (Per-Cutaneous Intervention)/Coronary Angioplasty:**
    - Balloon Dilation/Stenting of Coronary Arteries via Femoral Artery
  - § **OR... Thrombolysis/Fibrinolysis (With TPA – "*Tissue Plasminogen Activator*"/"*Alteplase*"):**
    - **Contraindicated in:** Hx of CVA, Stroke <3mths, Aortic Dissection, Active Bleeding.
  - § **+/- CABG:**



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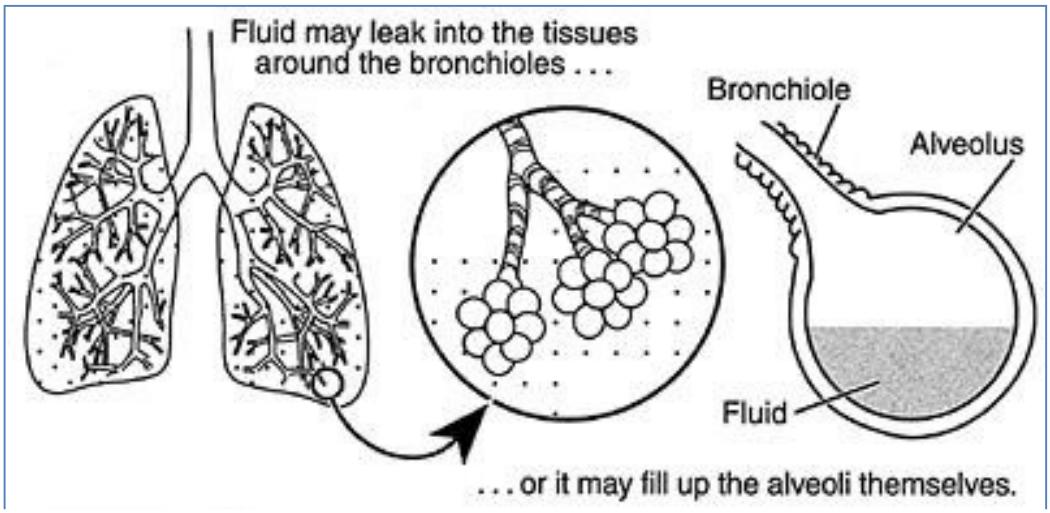
Complications:

- **Acute Complications:**
  - **LV-Failure:** → Acute Pulmonary Oedema, Shock (70% Mortality)
    Lethal Arrhythmias: → VT, VF
  - **Weakening of Necrotic Myocardium → Myocardial Rupture:** Tamponade / Acute VSD
  - **Stasis → Mural Thrombosis** → Embolization → Stroke
  -
- **Chronic Complications:**
  - **Ventricular Aneurysm, Papillary Muscle Rupture – Mitral regurgitation, CCF**.

ACUTE CARDIOGENIC PULMONARY OEDEMA

Get Direction GLOBAL

- **Aetiology:**
  - o Severe Decompensated LV-Failure (CCF)
- **Pathophysiology:**
  - o Severe Decompensated LV-Failure (CCF) → Fluid Accumulation in Alveoli & Interstitium→Dyspnoea
    - § →Impaired Gas Exchange & Respiratory Failure
- **Clinical Features:**
  - o **Symptoms:**
    - § Tachycardia
    - § Tachypnoea
    - § Diaphoresis
    - § Wet Cough with Frothy Sputum
  - o **Signs:** Respiratory Distress (↓SpO2)
    - § Bi-Basilar Crackles
    - § Splitting of S2
    - § Dullness to Percussion
    - § (+/- Signs of RV-Failure [↑JVP, Peripheral Oedema, Ascites])
    - §
- **Investigations:**
  - o **CXR –** (Pulmonary Congestion/Oedema, Cardiomegaly, Effusions)
  - o **ECG –** (Dx Previous/Current IHD, Rule out Arrythmias)
  - o **Echo (TTE) –** (Assess Ventricular Function [Ejection Fraction])
  - o **+(FBC [**↓Hb/Infection**], UEC, eLFT [**Alcohol**], TSH [**↑Thyroid**], Lipids [**IHD**], BSL/HbA1c [**Diabetes**])**
- **Management:**
  - o **Pt will most likely already be on CCF Regime. Ie:**
    - § **ACEi** (*Perindopril*) / **ARB** (*Candesartan*)
    - § **B-Blocker** (*Carvedilol*)
    - § **Diuretics** (*Frusemide / Spirinolactone*)
    - § **Fluid Balance** (Daily weights/Fluid restriction/↓Na diet)
  - o **"LMNOP" Protocol:**
    - § **L – Lasix (↑Diuresis & Fluid Restriction) – [***Frusemide / Spirinolactone***]**
    - § **M –** ***Morphine*** **(Anxiolytic & Vasodilation)**
    - § **N – Nitrates (***GTN***)**
    - § **O – Oxygen**
    - § **P – Positive Pressure Ventilation (CPAP / BiPAP)**



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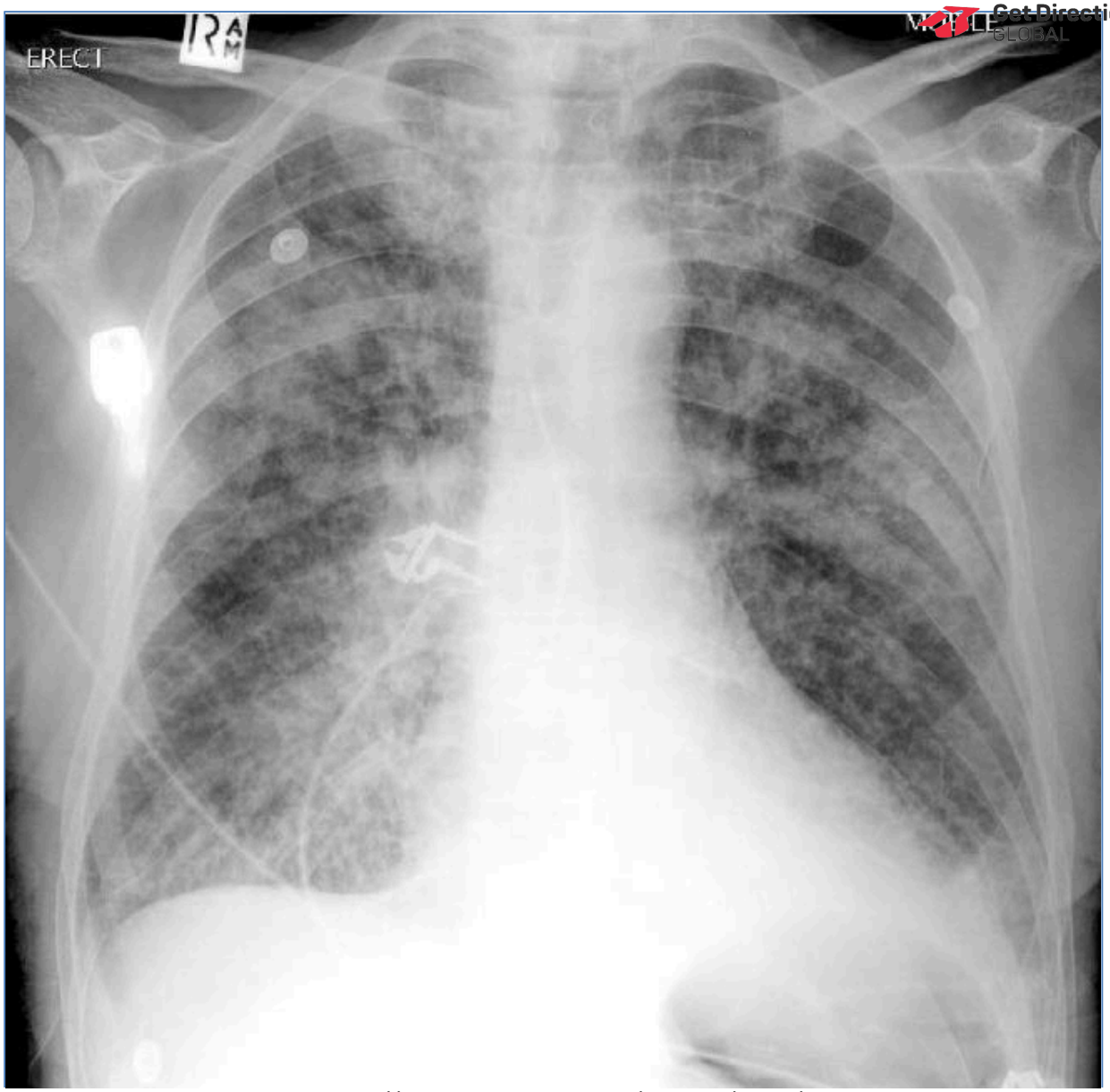

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HEART FAILURE

Get Direction GLOBAL

Background:

- ***Insufficient Cardiac Output*** to meet the demands of the body → ↓Organ Perfusion
- Note: 30% die within 1yr of Dx.

Signs of ↓Cardiac Output:

- **Low Arterial Pressure** (Due to weaker heart muscle)
- **Thready Pulse** (Due to Low Arterial Pressure) (A Compensatory Mechanism)
- **Tachycardia** (Due to [Carotid/Aortic] BaroReceptor-Reflex In Response to ↓BP) (Also due to the ↑Venous Pressure of Systemic Backlog (↑Systemic Blood Volume) →Atrial Stretch→ Bainbridge Reflex → Vagal (Parasympathetic) Withdrawal →↑HR)
- **Exercise Intolerance** (From ↓Tissue Perfusion)
- **Difficulty Breathing** (Eg: In Pulmonary Congestion)
- **Peripheral Oedema** (Eg: Due to R-Sided Heart Failure)

New York Heart Association – 5 Classes of Heart-Failure Symptoms:

- **Class 1: No limitation to physical activity**
- **Class 2: Slight limitation of activity + Dyspnoea & Fatigue with moderate exercise (Eg: Climbing stairs)**
- **Class 3: Marked limitation of activity + Dyspnoea with minimal activity**.
- **Class 4: Severe limitation of activity**. **Symptoms at rest**.
- **Class 5: Bed confinement**. **Life support monitoring**.

W here is the Failure?:

- **@ Myocardial Level – (Ie: Systolic/Diastolic Dysfunction (Heart Muscle Itself)→ ↓Pumping Function):**
- o (Eg: Ischaemic Heart Disease, Myocarditis, Cardiomyopathies, etc.)
- **@ Valvular Heart Level – (Ie: A problem with the Heart-Valves → ↓Pumping Function):**
  - o (Eg: Stenosis/Regurgitation)

@ Circulatory Level - (Ie: Defect in the Peripheral Circulation → Vascular System Dysfunction):

- o (Eg: Haemorrhage/Shock)

Forward/Backward Heart Failure:

- **Forward Heart Failure:**
- o Reduced Output due to Inadequate Discharge of Blood into Arterial System.

Backward Heart Failure:

- o Where One/Both Ventricle
  - § 1- Fails to Discharge its Contents OR
  - § 2- Fails to Fill Normally
- o Results in ↑Atrial Pressure + ↑Pressure in Venous System Behind the Failing Ventricle.

- **Note: Most Patients Have Both (Because Blood Flows in a Circle)**
  - o Eg: Forward Heart Failure → Low Cardiac Output → Less Venous Return → Backward Heart Failure.

The Body's Responses to Heart Failure:

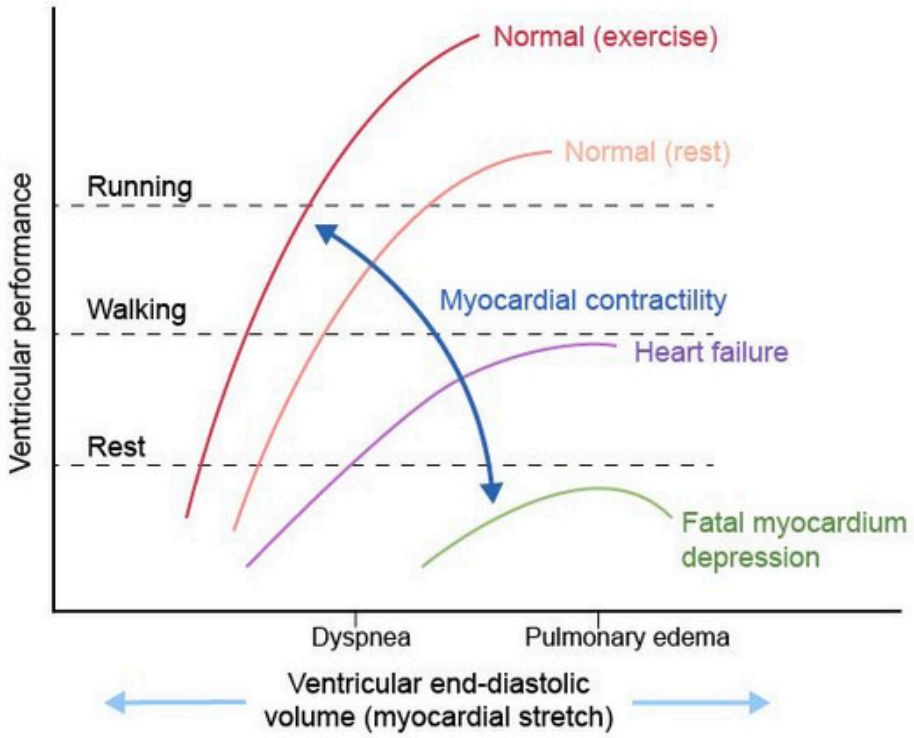
Short Term (Adaptive):

- o **Peripheral Shutdown** (To maintain BP of Vital Organs. → ↑Afterload)
- o **Salt & H2O Retention** (To ↑Blood Volume → ↑Preload)
- o **↑Preload** (To ↑ Stroke Volume)
- o **↑Sympathetic Tone** (To ↑ Heart Rate & Ejection)
- o **Hypertrophy** (To ↑ Muscle Mass to ↑ Contractile Strength)

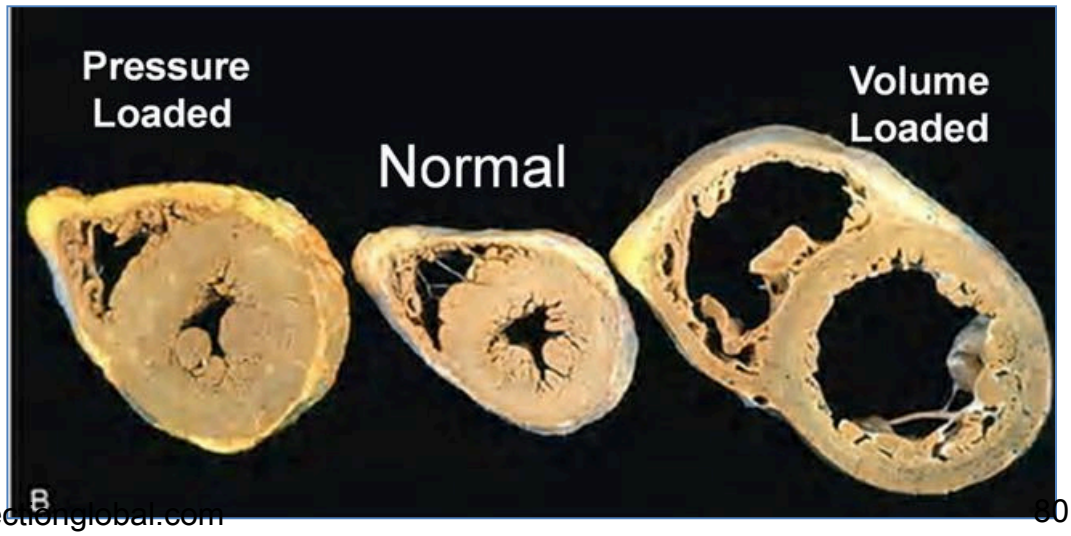
- **Long Term (Maladaptive):**
  - o (Over time, the Heart simply can't maintain the compensatory mechanisms of increasing CO)
  - o **Peripheral Shutdown** → ↑Afterload → L-Heart Failure
  - o **Salt & H2O Retention** → Fluid Overload → Pulmonary & Peripheral Oedema
  - o **Increased HR** → ↑Energy Demand
  - o **Hypertrophy** → Myocardial Ischaemia + Diastolic Failure

3 Compensatory Mechanisms:

- **1- Frank-Starling Law/Mechanism:**
  - o *"↑Preload → ↑Stroke Volume"*
  - o Incomplete Chamber Emptying →↑PRELOAD → ↑Cardiac Output BY ↑STROKE-VOLUME.
  - o **BENEFICIAL in Short-Term**
  - o **DETRIMENTAL in Long-Term**
    - § **Ie: In Severe Heart Failure, Starling Curve is *Flatter* than normal**.
    - § →Even large Increase in End-Diastolic Volume has *Little Effect* on Stroke Volume & CO.
    - § Also, ↑Vent-EDV → ↑Atrial Pressure → ↑Pulmonary Pressure

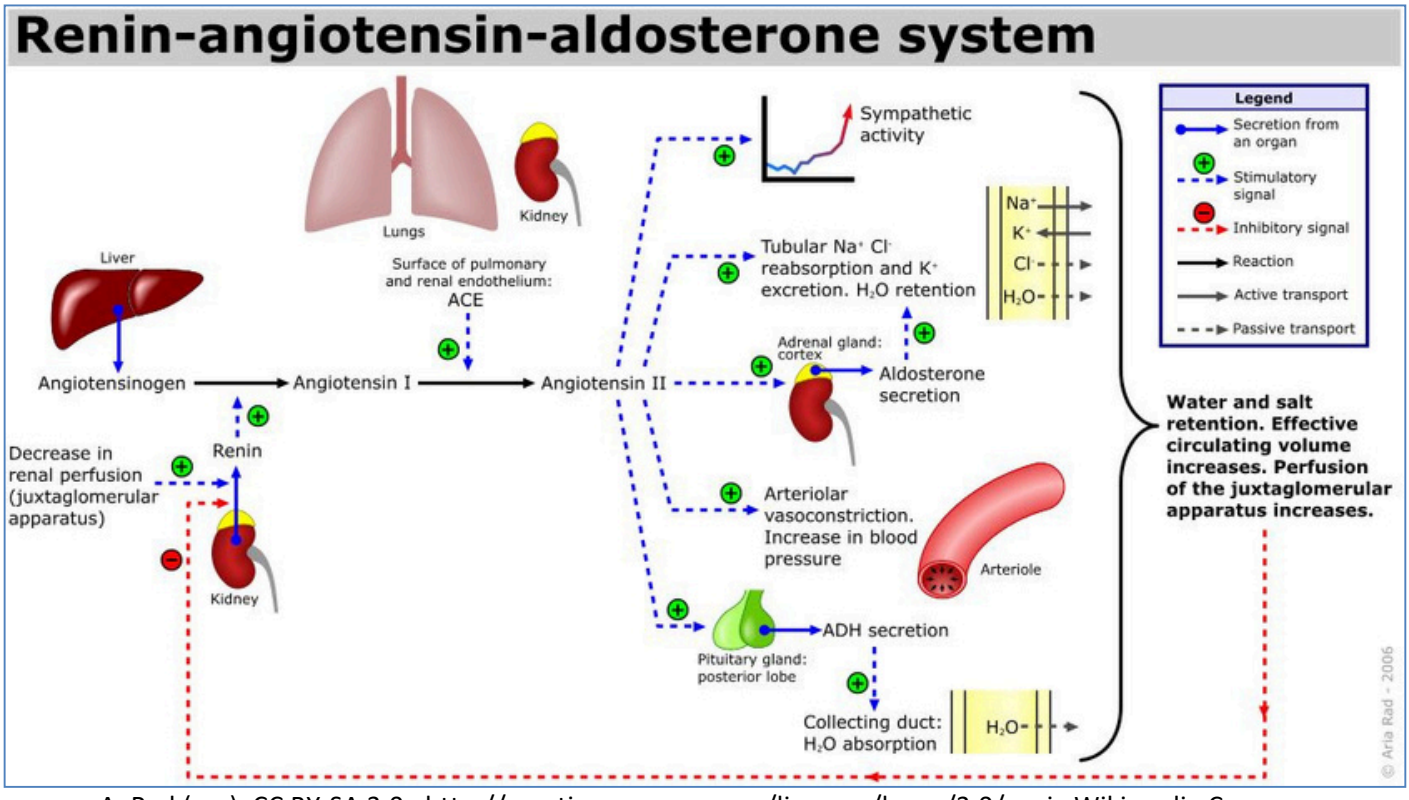


- **2- Myocardial Hypertrophy:**
  - o **Increased Ventricular Mass = Cell Hypertrophy (↑Size) & Hyperplasia (↑Numbers)**.
  - o **Pressure Overloaded Hypertrophy:**
    - § In response to ↓Cardiac Output: When↓CO is due to ↑↑Afterload (↑Arterial Pressure)
    - § **"Concentric Hypertrophy":** Muscle Thickens – Due to Synthesis of Sarcomeres in ***PARALLEL***.
      - →Decreased Compliance → ↑ESV → ↑Atrial Pressure → ↑Pul.Pressure.
  - o **Volume Overloaded Hypertrophy:**
    - § In response to ↑Volumes:
    - § Ie: ↑EDV → Ventricle Stretches (Dilates) → Cannot Generate Enough Force to Pump Blood.
    - § **"Eccentric Hypertrophy":** Heart *Balloons Out* – Due to Synthesis of Sarcomeres in ***SERIES***.



- **3- Neurohormonal Systems:**
  - **1- Nor-Adrenaline/Epinephrine:**
    - *Baroreceptors* sense ↓CO as ↓Perfusion-Pressure →Stimulates Sympathetic:
      - →↑ Heart Rate
      - →↑ Contractility
      - →↑ Vessel Tone → To Increase Venous Return
      - →↑Preload (→SV →CO)
  - **2- Atrial Natriuretic Peptide:**
    - Produced by Heart – But has NEGATIVE effects.
    - Released due to High *Filling Pressures* (within heart) – Via L-Atrial & Arterial Baroreceptors.
    - Important *INDICATOR* of Heart Failure
    - Function: → Reduce Fluid Retention (Ie: Diuretic)
      - →Vasorelaxation
      - →↓BP
      - →↑Renal Excretion (Na+ & H2O)

      } Therefore Inhibits RAAS.
  - **3- Renin-Angiotensin-Aldosterone System (RAAS)/Anti-Diuretic-Hormone Release:**
    - Due to ↓Renal Perfusion-Pressure → Stimulates Renin Secretion from Juxtaglomerular Cells.
      - →Vasoconstriction (Angiotensin-II = Potent Vasoconstrictor)
      - →↑Fluid Retention (Increases Intravascular Volume)
      - →↑Blood Pressure
      - →↑Preload (→SV →CO)



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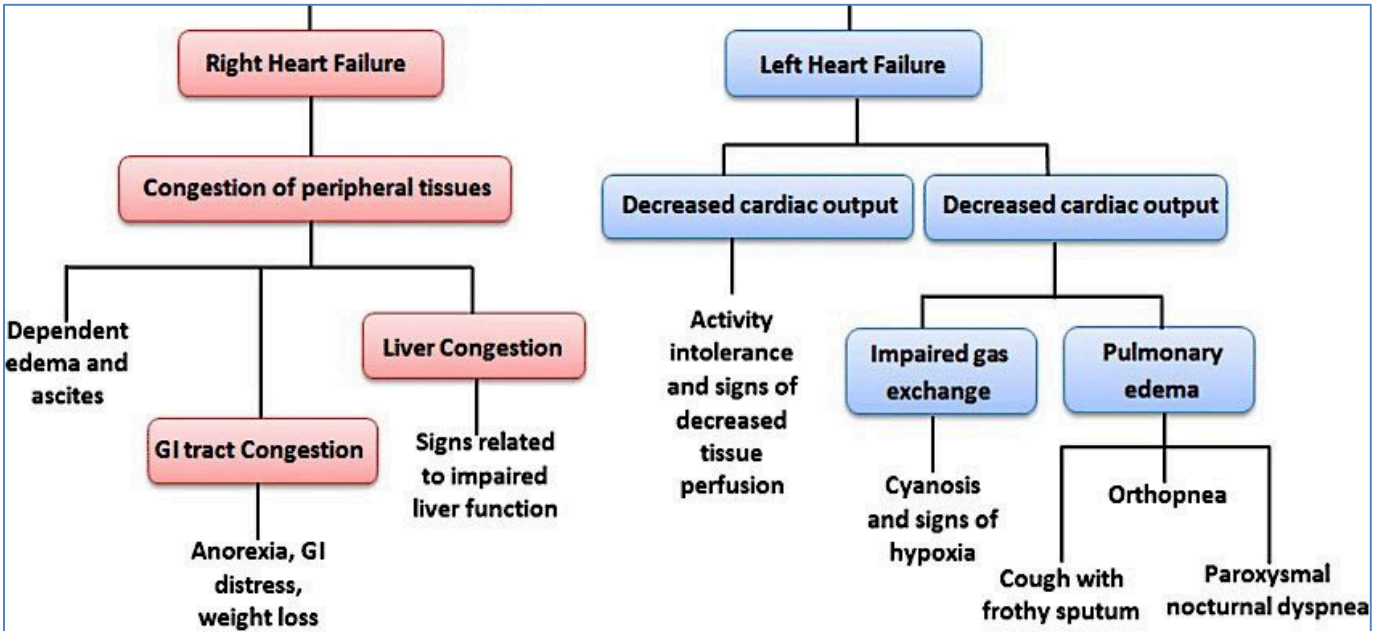
  - **Note: These Neurohormonal Compensatory Mechanisms = Vicious Cycles:**
    - Strain on heart → Activation of Neurohormonal Mechanisms →↑Preload & BP → Extra
    - Strain on the heart.
      Heart Responds by Remodelling → Larger & Rounder → Weaker.

Heart Failure Can Be LEFT or RIGHT Sided:

- **Left Heart Failure (LSHF):**
  - o = **↓L-Ventricle CO into *Systemic Circulation***
  - o **Common Causes:**
    - § **Systolic Failure:** Weak LV (IHD, Dilated Cardiomyopathy, Alcoholism, Myocarditis)
    - § **Diastolic Failure:** Stiff LV (Eg: Amyloidosis, Sarcoidosis, Hypertrophic Cardiomyopathy).
    - § **Valve Dysfunction:** (Aortic Stenosis/Regurg, Mitral Stenosis/Regurg)
    - § **Excessive Afterload:** (Eg: HTN, Coarctation of Aorta, Dissecting AAA)
  - o **Consequences & Clinical Features:**
    - § **Pulmonary Congestion →CCF →** Cough/Dyspnoea/Orthopnoea(Pt can't lie flat)/PND.
    - § **↓CO → (**Kidneys → Pre-Renal Failure), **(**Brain → Irritability, ALOC)
    - § **LV-Hypertrophy →** Initially Adaptive, then Weakens →Worse LV-Failure

- **Right Heart Failure (RSHF):**
  - o = **↓R-Ventricle CO into *Pulmonary Circulation***
  - o **Common Causes:**
    - § **Isolated RHF is Rare – (Typically caused by LSHF, Aka. *"Cor Pulmonale"*)**
    - § **"Cor Pulmonale":** LSHF →Pulmonary Hypertension →RSHF.
  - o **Consequences & Clinical Symptoms:**
    - § **Pulmonary Congestion →CCF →** Cough/Dyspnoea/Orthopnoea(Pt can't lie flat)/PND.
    - § ***PLUS Systemic* Congestion →** Peripheral Oedema/Organomegaly/Pleural Effusion/Ascites

- **NOTE: L-Failure can often lead to R-Failure:**
  - o Eg: L-Failure → Pulmonary Hypertension → ↑Afterload on R-Ventricle → R-Ventricular Failure.



Investigations:

- **B-Natriuretic Peptide (BNP) –** (If >500 = Heart Failure)
- **CXR –** (Pulmonary Congestion/Oedema, Cardiomegaly, Effusions)
- **ECG** – (Dx Previous/Current IHD, Rule out Arrythmias)
- **Echocardiogram (TOE/TTE)** – (Assess Ventricular Function [Ejection Fraction])
- **+(FBC [**Anaemia/Infection**], UEC, eLFT [**Alcohol**], TSH [**Hyperthyroid**], Lipids [**IHD**], BSL/HbA1c [**Diabetes**])**

Management of *Chronic CCF*:

- **1- Correct Systemic Factors & Comorbidities –** (Eg: Thyroid, Infection, Diabetes, COPD)
- **2- Lifestyle Mods –** (↓Smoking/Alcohol, Weight Loss)
- **3- Fluid Restriction -** (↓Salt Intake, Fluid Restriction, Daily Weights)
- **4- Antihypertensives –** (↓Preload & ∴ ↑CO)**:**
  - **ACE Inhibitors (*Perindopril*)/ARBs (*Candesartan*):**
    - **MOA:** ↓AT-II → Vasodilation + ↓Fluid Retention + ↓SNS → ↓Preload & ↓Afterload
    - **Dose:** Start Low &Go Slow.
    - **(Side Effects:** Persistent Dry Cough, Postural Hypotension, ↑K+, Renal Impairment)
  - **β-Blockers (*Carvedilol, Metoprolol, Bisoprolol*):**
    - **MOA:** ↓Workload of Heart (+ ↑Preload → ↑Cardiac Output) & Triggers Remodelling.
    - **(Side Effects:** Postural Hypotension, Dizziness)
- **5- Diuretics – (↓Fluid Overload):**
  - **Loop Diuretics (*Frusemide/"Lasix"*)**
  - **[IF SEVERE] Aldosterone Antagonists (*Spirinolactone*)– (Also K+ Sparing)**

- **(+/- Digoxin to ↑Contractility ; or Rate Control in AF)** – (Symptomatic Improvement, but no ↓Mortality)
- **(+/- Oxygen if SpO2 <88%)**
- **(+/- Vasodilators – Eg: Hydralazine / Nitrates)**
- **(+/- Internal Cardiac Defibrillator – as 50% of mortality is due to sudden lethal arrhythmias)**

Management of *Acute, Decompensated CCF*:

- **As Above (ACEi + B-Blocker)**
- **+ ↑Diuretics (*Frusemide*)**
- **+ Digoxin – (For Inotropic Support)**
- **+/- Nitrates**

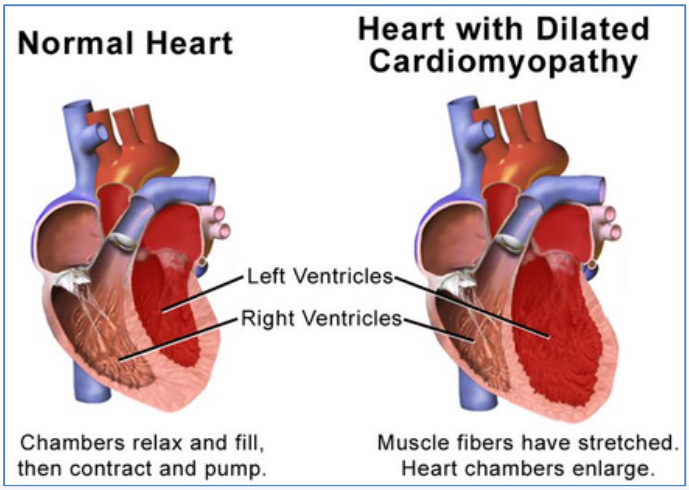
Com plications:

- **Sudden Lethal Arrhythmias (VT/VF) → Death**
- **Acute (Cardiogenic) Pulmonary Oedema** – (See CVS PATH – Acute Cardiogenic Pulmonary Oedema)

CARDIOMYOPATHIES ("HEART MUSCLE DISEASES")

DILATED CARDIOMYOPATHY (Most Common):

- **Aetiology:**
  - ***Idiopathic
  - **Chronic Alcoholism
  - *Post-Viral (Myocarditis)
  - Genetic
  - Chemotherapy
  - Chronic Anaemia
- **Pathogenesis:**
  - Progressive Dilation & Hypertrophy → **Systolic Dysfunction**.
    - → Enlarged, Flabby Heart
    - → Mural Thrombi (Can embolise)
    - → AV-Valve Regurgitation (Due to Chamber Dilation)
- **Clinical Features:**
  - Any Age (Incl: Childhood).
  - **Presentation: Congestive Heart Failure:**
    - Dyspnoea/Orthopnoea/PND
    - ↓Exercise Tolerance
    - Fatigue
    - Wet Cough
- **Complications:**
  - Mitral Regurgitation
  - Arrhythmias
  - Possible Thrombotic Embolism
- **Investigations:**
  - **ECG**
  - **CXR** – (Globular Heart)
  - **Echo** – (Assess Vent Function)
- **Management:**
  - **↓ETOH**
  - **CCF Triple Therapy:**
    - **ACEi** (*Perindopril*) / **ARB** (*Candesartan*)
    - **B-Blocker** (*Carvedilol*)
    - **Diuretic** (*Frusemide*)
  - **Warfarin** (Prevent Thromboembolism)
  - **FluVax & PneumoVax**
  - ****→ Heart Transplant**
- **Prognosis:**
  - **50% 5yr Mortality *Unless* Heart Transplant**.



Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". *WikiJournal of Medicine* **1** (2). DOI:10.15347/wjm/2014.010

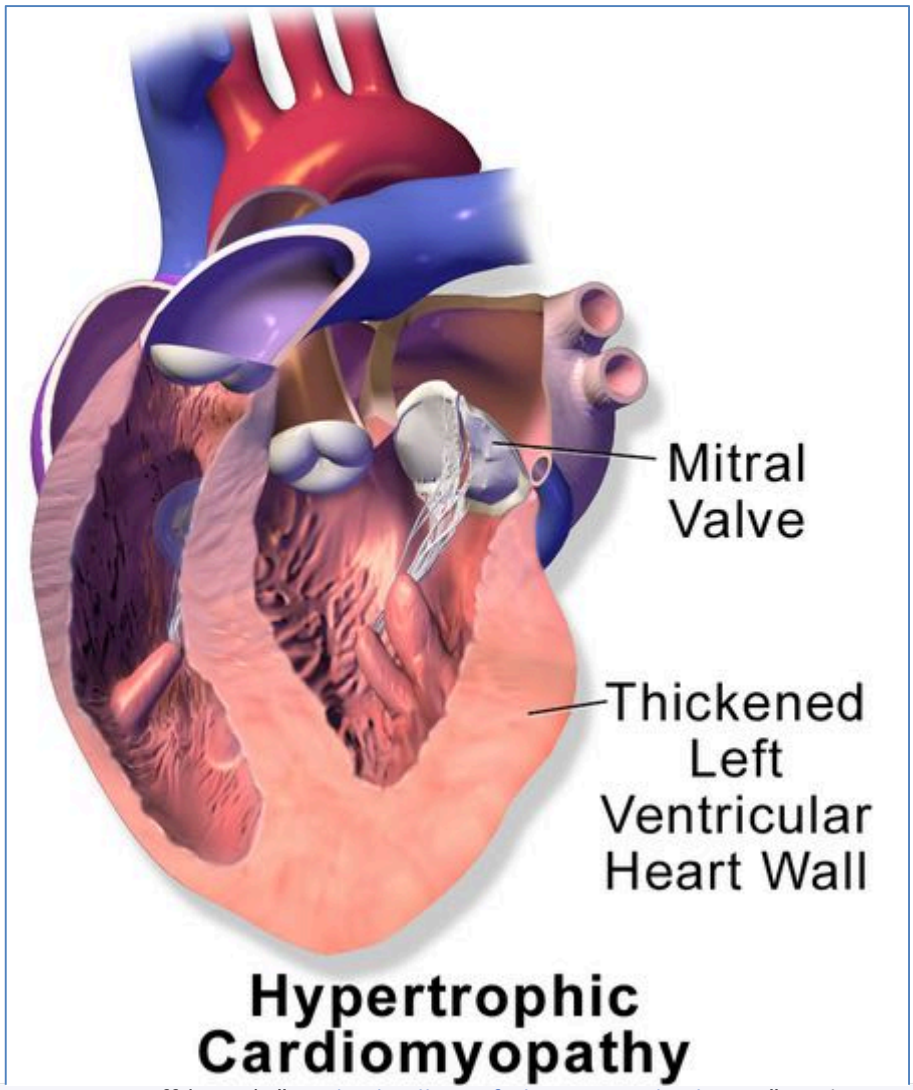
HYPERTROPHIC CARDIOMYOPATHY (AKA: HOCM – Hypertrophic Obstructive Cardiomyopathy)

- **Aetiology:**
- o **Genetic

Pathogenesis:

o Genetic Mutation → Hypertrophy → Diastolic Dysfunction (↓Filling & ↓Chamber Size)
o Note: End Stage can → Focal Ischaemia (Even in absence of Coronary Artery Disease)

- **Clinical Features & Complications:**
  - o CCF (Dyspnoea, Orthopnoea, PND, Cough)
  - o Ventricular Outflow Obstruction → Syncope + Harsh Systolic Murmur
  - o Angina
  - o Arrhythmias
  - o Mural Thrombus → Embolisation (Eg: Stroke)
  - o Sudden Death
- **Investigations:**
  - o **ECG** (LVH, Path Q Waves)
  - o **Echo** (LVH, Diastolic Dysfunction, Poor EF)
- **Management:**
  - o **Medical** – **β-Blockers** → ↓Heart Rate + ↓Contractility
  - o **Surgical** – Septal Myomectomy (Relieves the outflow tract obstruction)
  - o **+/- ICD** – (If Arrhythmias)



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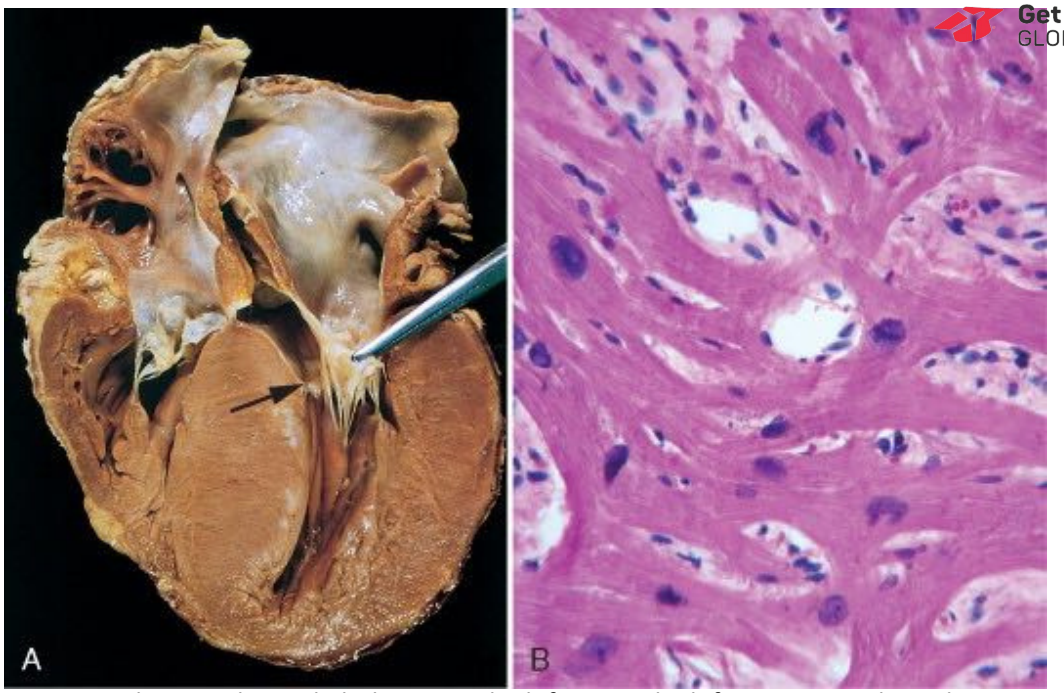


A, The septal muscle bulges into the left ventricle, left atrium is enlarged.
B, Extreme hypertrophy, branching of Myocytes, and the characteristic interstitial fibrosis (collagen is blue).

RESTRICTIVE CARDIOMYOPATHY

- **Aetiology:**
- o **Amyloidosis/Sarcoidosis/Scleroderma/Haemochromatosis

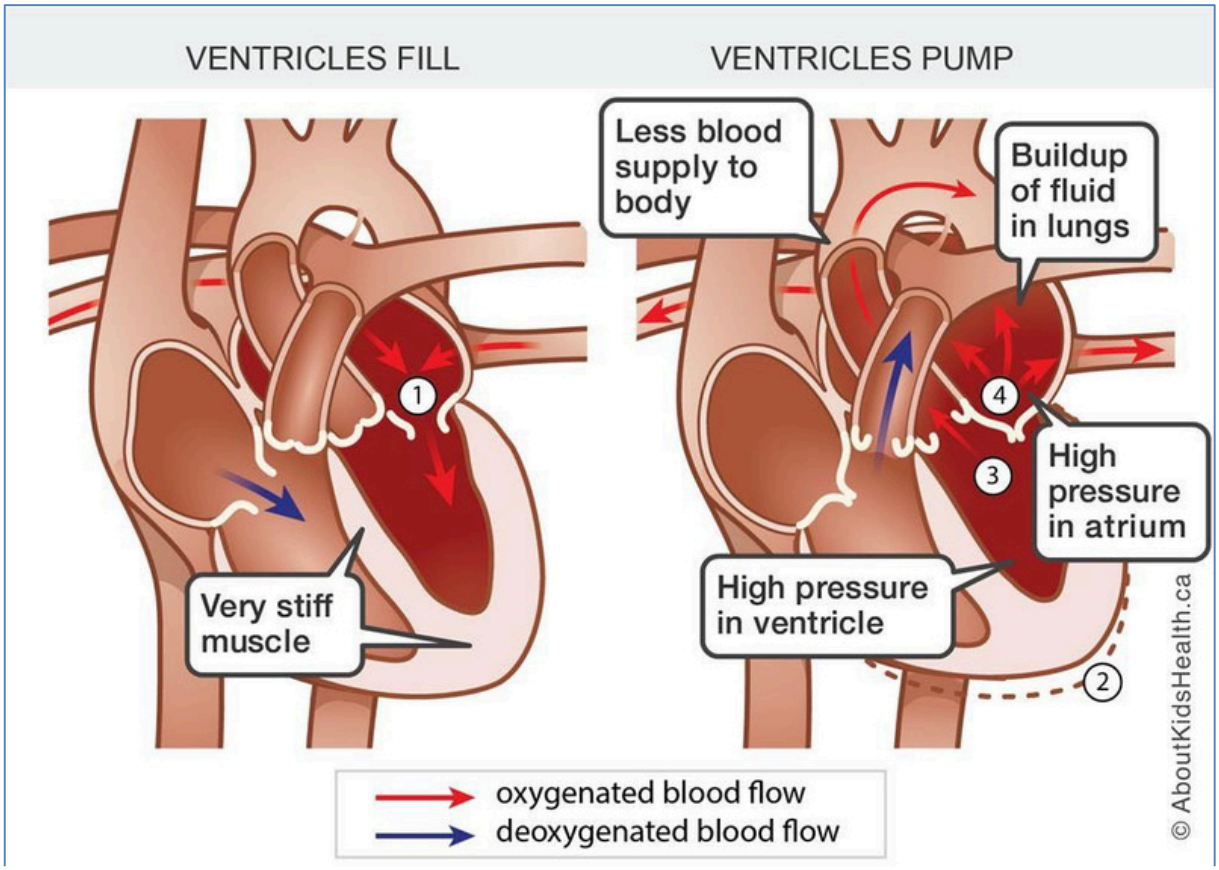
Pathogenesis:

- o → Stiffening of Myocardium → Diastolic Dysfunction (↓Filling) → Heart Failure
  - § Ventricles ≈ Normal Size & Volume
  - § Myocardium is Firm & Non-Compliant

- **Clinical Features & Complications:**
  - o **Heat Failure Symptoms:**
    - § Cough, Dyspnoea, PND, Orthopnea
    - § Fatigue
    - § Chest Pain, Palpitations
  - o **Signs:** Elevated JVP
    - § Lung Crepitations
    - § Peripheral Oedema
    - § Arrhythmias
    - §

- **Investigations:**
  - o o **ECG** – (Low Voltage)
  - o **CXR** – (CCF Signs)
  - o **Myocardial Biopsy (To Determine Aetiology)**
  - **Echo –** (Diastolic Failure, Poor EF)

- **Management:**
  - o **Medical:**
    - § **CCF Triple Therapy** – (ACEi/ARB + B-Blocker + Diuretics)
    - § **Warfarin**
    - § +/- Anti-Arrhythmics
  - o **Definitive: Requires Heart Transplant.**



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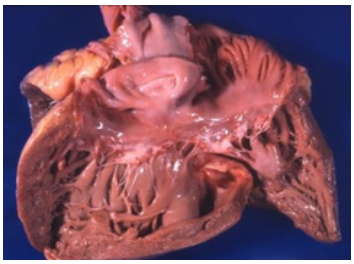
("COR PULMONALE"):

- **RV Hypertrophy AND Dilation. (Secondary to COPD/Chronic Pulmonary Hypertension)**



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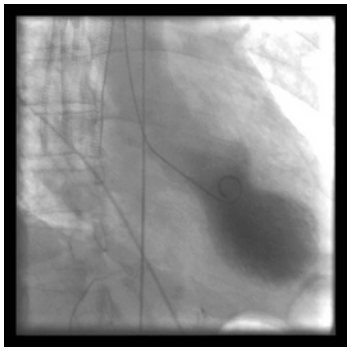
("STRESS CARDIOMYOPATHY"):

- **AKA:**
  - o "Broken Heart Syndrome"
  - o "Takotsubo Cardiomyopathy"
  - o "Apical Ballooning Cardiomyopathy"
- **Aetiology:**
  - o (NON-ischaemic)
  - o Stress-Related (High Catecholamines)
- **Pathogenesis:**
  - o Stress → High Catecholamines → Coronary Vasospasm → Myocardial *Stunning*
    - § →Bulging of the LV-Apex with Hypercontractile LV-Base. ("Octopus Trap" Shape)
- **Clinical Presentation & Complications:**
  - o **Acute, Reversible LV Systolic Dysfunction**
    - § Sudden Onset CCF
    - § Chest Pain
    - § Dyspnoea
  - o **Lethal Ventricular Arrhythmias + Other ECG Changes (Similar to MI)**
  - o **Ventricular Rupture**
- **Investigations:**
  - o **ECG –** (ST-Elevation)
  - o **Troponins –** (Elevated)
  - o **CXR**
  - o **Echo –** (Characteristic Regional Wall Motion Abnormalities)
  - o **Serum Catecholamines**
- **Management:**
  - o Supportive Therapy
  - o **CCF Triple Therapy:**
    - § **ACEi** (Perindopril)
    - § **B-Blocker** (Carvedilol)
    - § **Diuresis**
  - o **Inotropes (If Hypotensive) (***Dopamine***)**
  - o **Aspirin**
  - o **+/- Warfarin**



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HYPERTENSION:

Get Direction GLOBAL

W hat is Hypertension?:

- Consistent **Systolic of +140mmHg**. **AND/OR**
- Consistent **Diastolic of +90mmHg**

Aetiologies & Types:

- **95% = Primary/"Essential"/Idiopathic Hypertension:**
  - o **Idiopathic** – Likely multifactorial (not curable)
  - o **Risk factors for HT:**
    - § GENETICS/FamHx
    - § High Cholesterol/Salt Diet
    - § Diabetes/Obesity
    - § Smoking/Alcohol
    - § Stress
    - § Age
  - o **Subtypes:**
    - § **Isolated Diastolic HTN** (Typically Older Men)
    - § **Isolated Systolic HTN (Eg: >160/<90)**
      - In Young Adults – (**Due to Overactive Sympathetic NS** → ↑CO)
      - In Older Adults - (**Due to ↓Arterial Compliance** (Calcification/Fibrosis))
- **5% = Secondary Hypertension:**
  - o **Cardio -** Coarctation, Hypervolaemia, Rigid Aorta
  - o **Renal -** Acute Glomerulonephritis, CKD , Polycystic Kidneys, Renal Artery Stenosis
  - o **Endocrine –** Hyper-Adrenalism, Acromegaly, Hypo/hyperthyroidism, Phaeo, Cushing's.
  - o **Neurologic -** Psychogenic, Raised ICP, Sleep Apnoea, Acute Stress
  - o **Pre-Eclampsia:** (10% of pregnancies) - Placental Ischaemia → Placental vasoactive mediators → ↑ M aternal BP in effort to ↑ Placental Perfusion.
- **(Accelerated/"Malignant" Hypertension):**
  - o **= Rapid ↑in BP (>200/120mmHg) *Sufficient to cause Vascular Damage*→**
    - § **Retinopathy –** (Papilloedema, Haemorrhages, Bulging Discs)
    - § **Brain –** (Mental Status Changes)
    - § **Renal –** (Creatinine Rise)
    - § **Rapid Organ Failure**
    - § Note: "Malignant HTN" is rare, but can arise in HT of any Aetiology.
  - o **Pathophysiology Not well Understood:**
    - § **Common Causes:**
      - Cessation of Antihypertensives (Rebound HT)
      - Sympathetic Hyperactivity
      - Stimulants (Cocaine/Amphetamines)
      - Glomerulonephritis (Nephritic Syndrome)
      - Head Trauma (↑ICP)
      - Tumours (Eg: Thyroid, Phaeo, Adrenal)
      - Pre-Eclampsia
  - o **Symptoms Include:**
    - § Vision Disturbance (Papilloedema/Retinal Bleed)
    - § Headache, Drowsiness, Confusion
    - § Nausea, Vomiting
  - o **Management:**
    - § *Smoothly* Reduce BP *over 24 to 36 hours* to <150 / 90
    - § (Note: Excessive reduction may → Coronary/Cerebra/Renal Ischaemia)

Clinical Features:

- **Symptoms:**
  - o Typically Asymptomatic (Unless Malignant – Headache, Dizziness, N/V, Visual Changes)
- **Signs:**
  - o **Signs of 1o *causes*** – Eg: Thyroid, Cushing's, Acromegaly, Polycythaemia, CKD, Pregnancy.
  - o **Abdomen:** Renal or Adrenal Masses (for possible causes), or for AAA
  - o **Renal Bruit:** (Renal Artery Stenosis)
- **Diagnosis – Essential Vs. Secondary?:**
  - o **If Essential HT**: Diastolic Pressure will *RISE* on standing.
  - o **If Secondary HT:** Diastolic Pressure will *FALL* on standing.
- **Classification (Adults):**

Diagnostic Evaluation:

- **>3 Consecutive Readings of >140/>90 over 6mths = HTN**
- **BUT: Needs to be >*Stage 2* (>160/>100) to Prescribe Antihypertensives.**
- **+FBC** – (Eliminate Polycythaemia)
- **+Lipids** – (Screen ↑Risk Fx for IHD)
- **+UEC** – (Screen Renal Failure, Electrolyte Disturbances)
- **+Urinalysis** – (Screen Renal Failure & Urine Electrolytes)
- **+BSL** – (Screen Diabetes)
- **+ECG** – (Screen IHD)

Category	Systolic (mmHg)	Diastolic (mmHG)	% Population
Normal	120-140 140-	80-90 90-100	83
Stage 1 Hypertension (Mild)	160 160-180	100-110 110-120	13.5
Stage 2 Hypertension (Moderate)	180-210 ≥210	≥120	2
Stage 3 Hypertension (Severe)			
Stage 4 Hypertension (Severe)			1

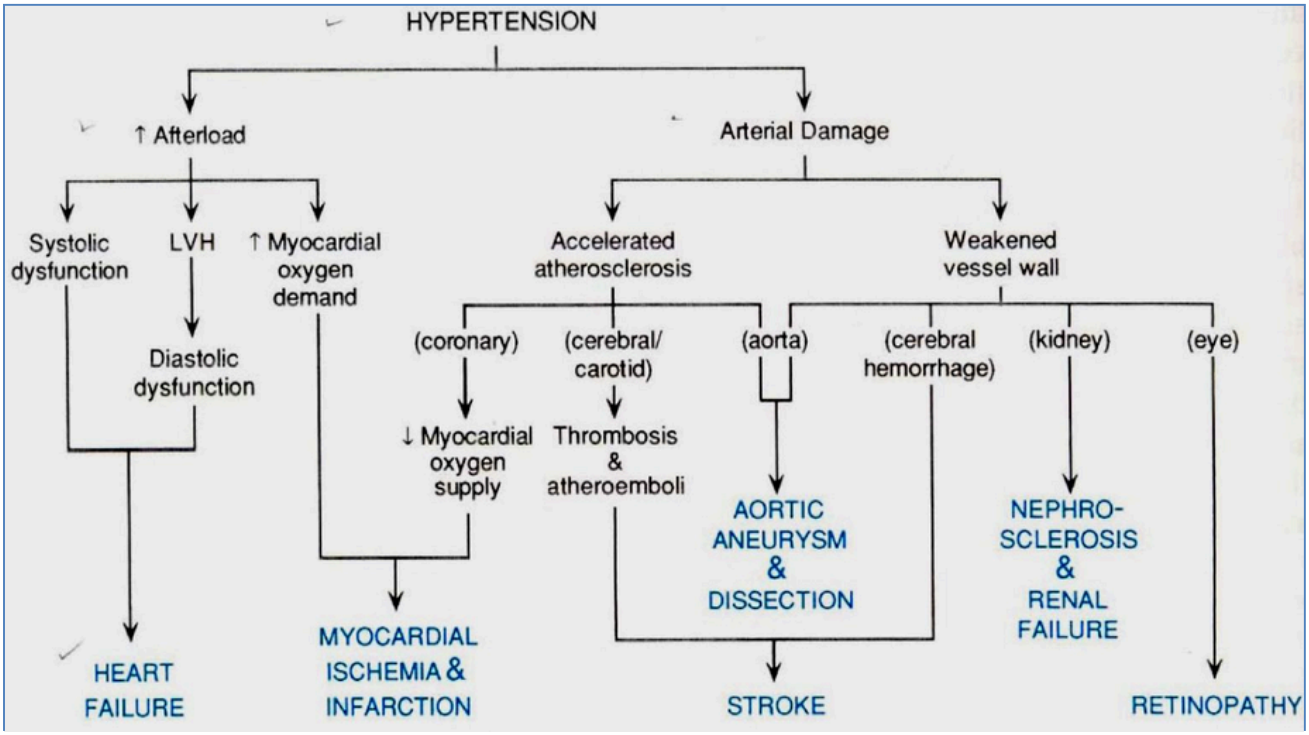
MANAGEMENT:

- **(Identify & treat underlying causes)**
- **(Note: Reduction should be SLOW, otherwise can be fatal)**
- **1- Lifestyle changes:**
  - o **Reduce Risk Factors** (Eg: Quit Smoking, ↓-Fat Diet, ↓Alcohol, ↓Salt, ↑Exercise)
- **2- Treatment drugs (If >Stage 2 [>160/>100]):**
  - o **Monotherapy First, Then Add ONE Other – (In Order of Recommendation):**
    - § **ACEi** (*Perindopril* ["Coversyl"]) / **ARB** (*Candesartan* ["Atacand"])
      - (Note: Beware ↓K+)
      - (Beware Dry Cough)
    - § **Ca-Ch-Blocker** (*Amlodipine* ["Norvasc"] / *Nifedipine* ["Adalat"])
    - § **Thiazide Diuretic** (*Hydrochlorothiazide* ["Amizide"])
      - (Note: Beware ↓K+)
    - § **{B-Blocker** (*Carvedilol* ["Dilatrend"] / *Atenolol* ["Noten"])**} Now Controversial!**
      - *Only used if Pt also has IHD / CCF.*
  - o **(Therapeutic Target *<140/90mmHg* or *<130/80mmHg in diabetics*)**
- **+ (3- Home BP Monitoring):**
  - o **If:** Non-Compliant / Diabetic / "White-Coat HTN"

Complications of Hypertension:

- **HTN Is a Major Precursor For:**
  - CAD/IHD
  - Hypertensive Heart Disease (Heart Failure, Hypertrophic Cardiomyopathy)
  - Stroke
  - Aortic Dissection
  - Microangiopathy/'*Arteriolosclerosis*' (Small Vessel Diseases)
    - PVD
    - Renal Failure

- Relationship between *Degree* of hypertension & *Degree* of Complications.



- **Heart:**
  - **↑Afterload** → LV-Hypertrophy → Eventually Diastolic Failure
  - **↑Workload** → ↑O2 Demand → Exacerbated Coronary Ischaemia
- **Lungs:**
  - **Pulmonary Congestion →** Pulmonary Oedema & RV-Hypertrophy
- **CerebroVascular:**
  - ***Intracerebral Haemorrhage*** **(**Rupture of Artery/Arterioles in brain)
- **Aorta/Peripheral Vascular:**
  - Mechanical Arterial Damage (Eg: Aneurysms/Dissecting Aneurysms/Atherosclerosis)
- **Kidneys:**
  - Nephrosclerosis – (hardening of kidney blood vessels) → Renal Failure

SHOCK:

EXAM Definition = "Inadequate Perfusion of *Vital* Organs (Heart/Brain/Kidneys)"

Aetiologies:

- **Hypovolemic Shock:**
  - o Severe Dehydration - (Eg: Sweating, Vomiting/Diarrhoea, DKA & Diuresis, Seeping Burns)
  - o Severe Blood Loss/Haemorrhage
- **Cardiogenic Shock:**
- o Heart Failure - (Eg: Acute MI, Valvular, Cardiomyopathy, Myocarditis)

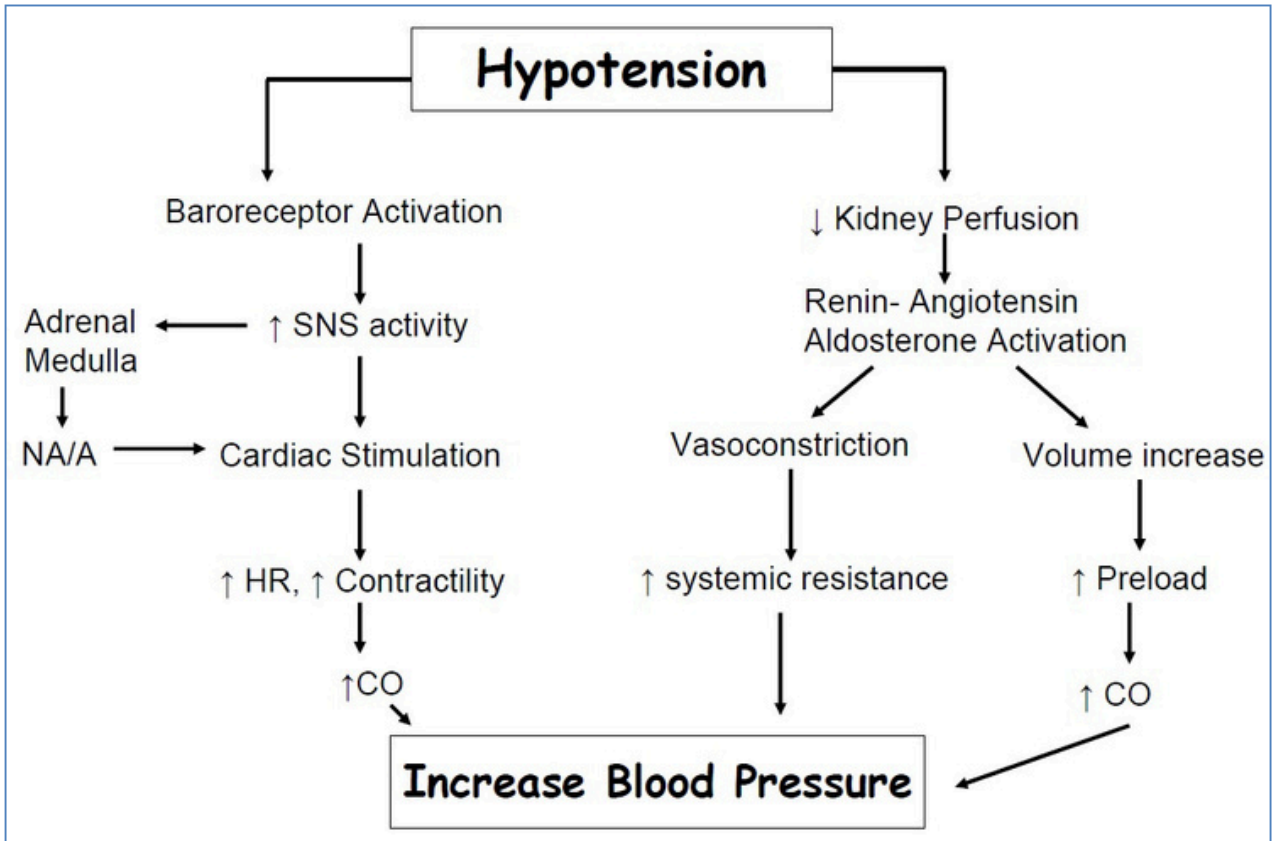
Distributive Shock:

- o **Septic Shock** – (Extracellular Fluid Shift → Hypotension → Shock)
- o **Anaphylactic Shock** – (Extracellular Fluid Shift → Systemic Oedema & Hypotension).
- o **Neurogenic Shock** – (Sudden loss of Vasomotor Tone → Massive VenoDilation)

- **Obstructive Shock:**
  - o **Massive PE**
  - o **Cardiac Tamponade –** (Massive Pericardial Effusion → ↓Ventricular Filling → ↓SV & CO)
  - o **Tension Pneumothorax**

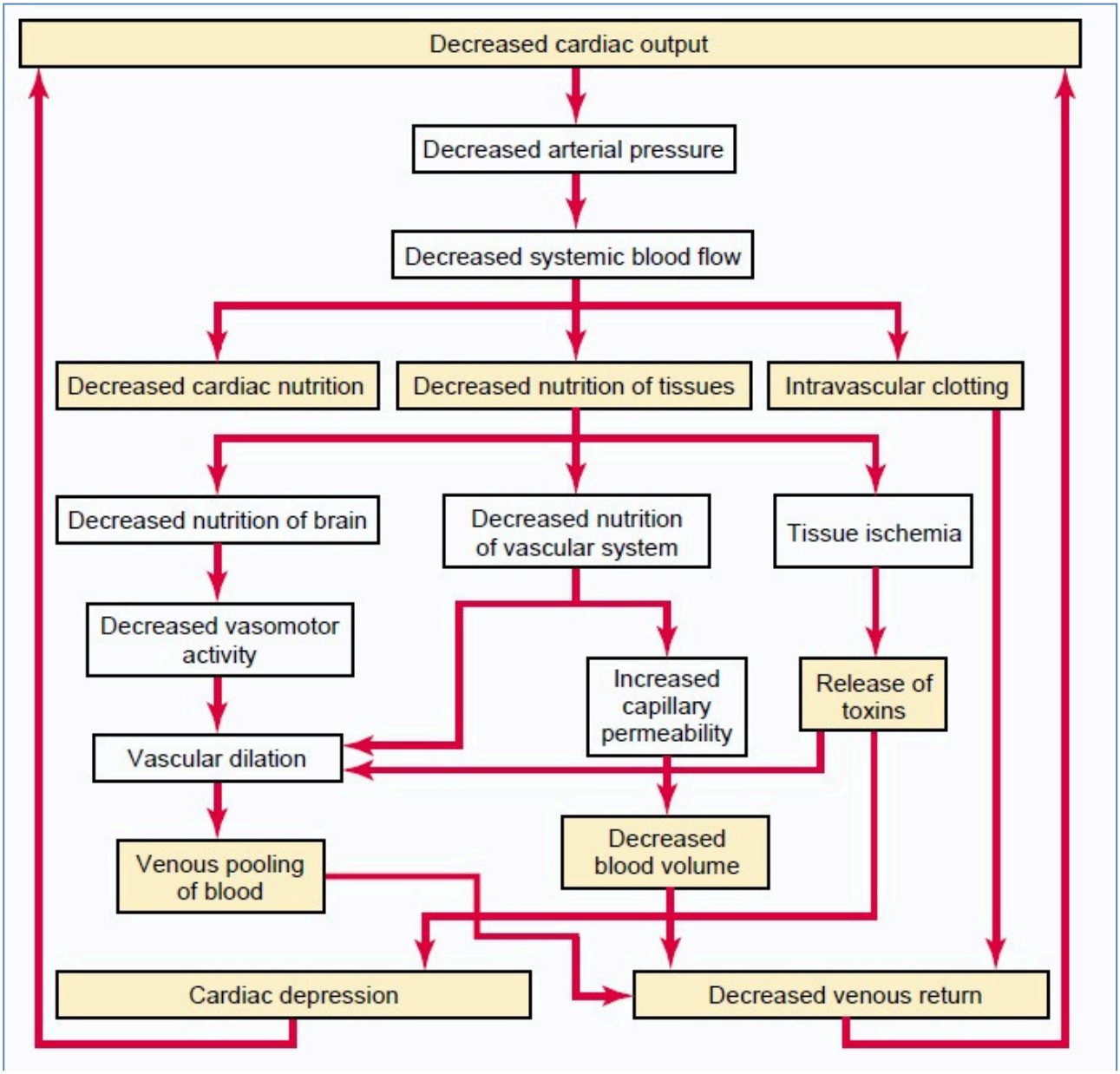
Compensatory Mechanisms:

- o ***"CARDIAC RESERVE" =*** *Maximal % that CO can Increase Above Normal. (Typically 300-400%)*
- o **(IMMEDIATE) ↑Sympathetic Tone:**
  - § Baroreceptors → ↑SNS → ↑HR & Contractility → ↑CO
- o **(DELAYED) Renal:**
  - § **Angiotensin-II** → General Vasoconstriction →↑BP
  - § **Vasopressin (ADH) →** ↓Urine Output → ↑Blood Volume → ↑BP
  - § **EPO →** ↑Haematopoiesis → ↑Blood Volume → ↑BP



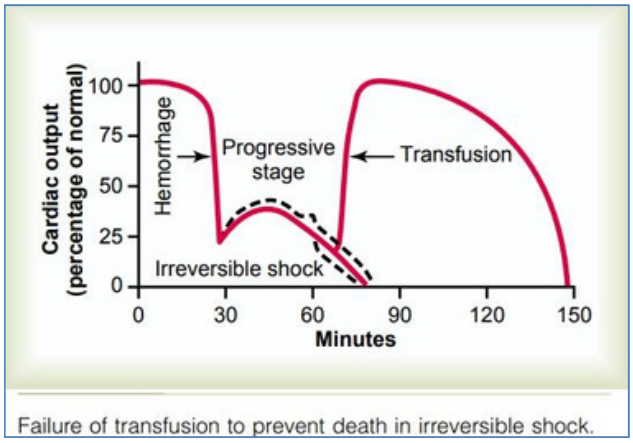
3 Stages of Shock:

- **1- Non-Progressive Stage (<15% (<750mL)Blood Loss):**
  - o **Stable & Reversible**.
  - o **Signs of Compensated Hypovolaemia:**
    - § *Tachycardia*
    - § *Oliguria* (Low Urine Production)
- **2- Progressive Stage (15-40% (750-2000mL)Blood Loss):**
  - o **Unstable, Decompensating, Reversbile**.
  - o **Signs of Decompensation:**
    - § *Hypotension*
    - § *Delayed CRT* (↓Peripheral Perfusion)
    - § *Tachycardia*
    - § *Organ Failure* (Anuria, Confusion/ALOC, Heart Failure, Tachypnoea, Acidosis)
  - o **But *Still Reversible* with Treatment:**
    - § Reverse Causative Agents + Volume Replacement ***(Bolus 2L IV)*** +/- Inotropes
    - § **(Otherwise Fatal if Untreated)**



Abdel-Sater, Khaled. (2011). Physiological Positive Feedback Mechanisms. nwpii.com/ajbms. 3. 10.5099/aj110200145.

- **3- Irreversible Stage (>40% (>2000mL) Blood Loss):**
  - **Unstable, Irrecoverable Organ Failure**.
  - **Pt WILL Die –** Treatment will delay death, but NO treatment will save Pt's life.
  - **Symptoms:**
    - Multi-Organ Failure (Renal/Cardiac/Pulmonary/CNS)
    - Acidosis
    - Anuria
    - Coma



Failure of transfusion to prevent death in irreversible shock.

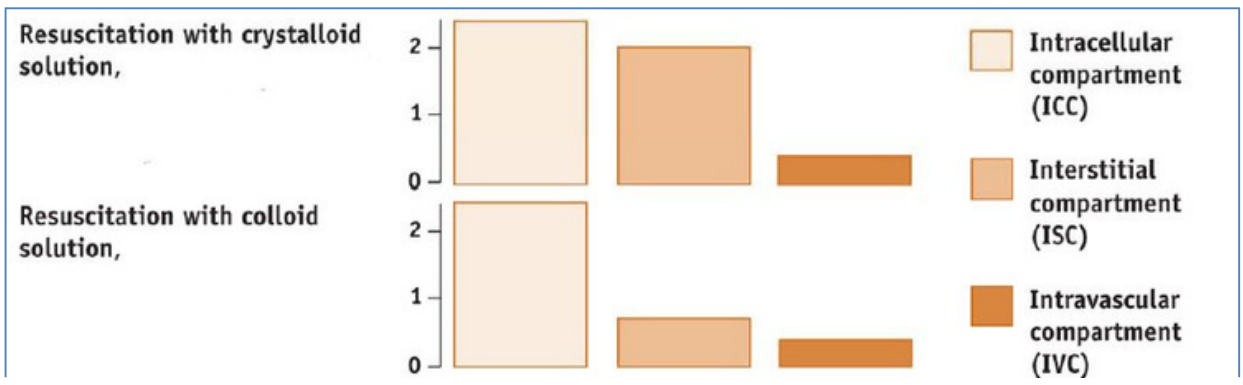
Basic Shock Management:

- **Hypovolaemic Shock:** Recognise Severity, **Replace Loss (Normal Saline)**, Stop Ongoing Losses
- **Septic Shock:** Blood Culture, **IV ABs, IV Fluids, Inotropes, Vasopressors**, Remove Infective Focus
- **Anaphylactic Shock:** ABC 1o Assessment, **IM/IV/SC Adrenaline**, +/- Steroids
- **Cardiogenic Shock:** **Inotropes**, Nitrates/Angioplasty/Reperfusion, Valvuloplasty, Transplant
- **Mechanical:** **Pericardiocentesis**, Correct Cause (Trauma/Infection)
  - **Tamponade:**
  - **Pneumothorax: Thoracocentesis** (Pleural Tap), Correct Cause (Trauma/Infection/Fluid Overload)
  - **PE:** **Thrombolysis (TPA/Alteplase), Thrombectomy**

FIRST LINE TREATMENT: FLUID REPLACEMENT THERAPY:

Crystalloid Vs. Colloid Solution:

- **Crystalloids:**
  - = Aqueous Solutions of Mineral Salts or other water soluble molecules.
  - Crystalloids have a *Low* Osmotic-Pressure in Blood due to Haemodilution.
- **Colloids:**
  - = Mixtures of Larger Insoluble Molecules. (Note: Blood *itself* is a colloid)
  - Colloids Preserve a *High* Colloid-Osmotic Pressure in the Blood.



Crystalloid Solutions:

- ***Saline:**
  - o The Most Commonly used Crystalloid.
  - o **Advantage –** Is *Isotonic* → Does not cause dangerous *fluid shifts*.
  - o **Disadvantage –** If you only replace fluid, O2 Carrying Capacity goes down (Dilution Anaemia)
    - § Also, since it raises Extracellular Fluid, it's not suitable for Pts. with Heart Failure/Oedema.
- **Dextrose:**
  - o Saline with 5% Dextrose – Used if Pt is at risk of Hypoglycaemia; or Hypernatraemia.
  - o Note: Becomes Hypotonic when Glucose is Metabolised → Can cause fluid overload.
- **Lactated Ringer's/Hartmann's Solution:**
  - o A Solution of Multiple Electrolytes:
    - § Sodium
    - § Chloride
    - § Lactate
    - § Potassium
    - § Calcium
  - o Used in Pts with Haemorrhage, Trauma, Surgery or Burns.
  - o Also used to Buffer Acidosis

Colloid Solutions:

- **Albumin:**
  - o Albumin 40g/100ml - Used in Liver Disease, Severe Sepsis, or Extensive Surgery.
  - o Albumin 200g/100ml – Used in Haemorrhage/Plasma loss due to Burns/Crush Injury/Peritonitis/ /Pancreatitis; or Hypoproteinaemia; or Haemodialysis
- **Polygeline (Haemaccel):**
  - o = Gelatin Cross-linked with urea.
  - o Used in Dehydration due to GI Upsets (Vom/Diarrhoea)

Blood Products:

- **Whole Blood:**
  - o RBCs, WBCs, Plasma, Platelets, Clotting Factors, Electrolytes (Na/K/Ca/Cl).
  - o Used to Replace Blood Volume & Maintain Haemoglobin Level → ↑O2-Carrying Capacity
- **RBCs:**
- o Used to Increase Haematocrit (proportion of RBCs) → ↑O2-Carrying Capacity

Plasma:

- o Plasma (With Plasma Proteins), Clotting Factors, Fibrinogen, Electrolytes (Na/K/Ca/Cl).
- o Used to restore Plasma Volume in Hypovolaemic Shock & Restore Clotting Factors.

Fluid Resuscitation Principles:

How Much???

- o **1- Bolus (Vol. Of Estimated Acute Losses)**
- o **2- Maintenance ***(4,2,1 Rule)***:**
  - § 4ml/kg/hr for 1st 10kg
  - § 2ml/kg/hr for 2nd 10kg **(Ie: 60ml/hr for 1st 20kg)**
  - § 1ml/kg/hr for every kg thereafter. **(Ie: 100ml/hr for 1st 60kg –Plus 1ml/kg/hr onwards)**

- **What happens to the Different IV Fluids?:**
  - o **Crystalloids (IV Saline/Hartmann's) → Na Redistributed into ECF & Blood due to Na/K-ATPase**.
    - § (25% remains in Blood)
    - § :. Somewhat useful in Pressure Fluid Resuscitation.
  - o **Colloid (Albumin, Gelatine) → Colloid Is Not Redistributed (Stays in blood)**.
    - § (ALL fluid given remains in Circulation) - (500mL of Colloid = 2L of Crystalloid)
    - § :. Most effective fluid in Pressure Fluid Resuscitation.
  - o **IV Dextrose → Actively taken into cells :. None Remains in Blood**.
    - § :. NOT Suitable for Pressure Fluid Resuscitation. (Good for Hypoglycaemia & Post-Surgery)
- **Blood:**
  - o **= The best fluid to replace blood loss**
  - o But Saline/Hartmanns or Colloid are still ok.
  - o BUT Blood has risks (immunogenic/infections/etc)

SHOCK CASES:

Case 1 - Bart:

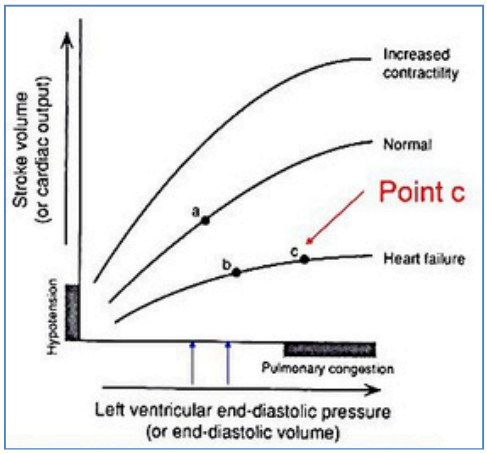
- He is pale and sweaty, has a distended abdomen and obvious bilateral femoral fractures. His pulse is 140 and his blood pressure is 75/40.

- **What signs of shock are evident?**
  - Pale and Sweaty
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Hypovolaemic (Haemorrhagic) Shock:
    - Seems to be bleeding into abdomen → Hypovolaemia → ↓CO →Hypotension + Compensatory Tachycardia
- **Could Bart be shocked without a change in BP?**
  - Yes. Young, healthy people are able to compensate for up to 1500mL of blood loss by Tachycardia & Vasopression, but then deteriorate rapidly afterwards.
- **Is this consistent with our definition of shock ?**
  - No - Our definition stipulates a loss of blood pressure.
  - (*Clinically important - Need to remember that relying on blood pressure changes alone to diagnose shock means that we will not recognise shock until a patient has lost 30 - 40 % of their blood volume (class 3)*)
- **Initial Treatment:**
  - Fluid Replacement – (For Hypovolaemia)

Case 2 – Homer:

- Suddenly collapsed and clutched his chest. He is pale and sweaty. His pulse is 40 and his blood pressure is 85/60. He is feeling short of breath. You note that his JVP is raised. Moe thinks that Homer has had a heart attack.

- **What signs of shock are evident?**
  - Pale & Sweaty
  - Hypotensive
  - Bradycardic → Suggests Cardiogenic Shock
- **What Type of Shock is This?**
  - → Cardiogenic Shock:
    - Myocardial Infarction → Heart Failure (↓CO) & Bradycardia→ ↓BP.
- ***Homer's ECG has shown an anterior myocardial infarction*. Why might this have caused him to be shocked?**
  - Myocardial Infarction → Disrupted heart Contraction & Conduction → ↓HR (in this case), and ↓CO
- **If Homer has a heart that is not pumping properly (decreased contractility) which direction will his Starling curve move?**
  - His starling curve will shift Downwards (Ie: Stroke Volume & CO will be Less @ any given End-Diastolic Volume)



- **Initial Treatment:**
  - Inotropes

Case 3 - Marge:

- Marge has bought a special new brand of extra strong hairspray. Begins to feel very itchy and notices small bumps coming up on her head. She collapses. She is conscious but confused. Skin is bright red & covered in raised lumps. Her pulse is 120 and her blood pressure is 90/60.

- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Distributive (Anaphylactic) Shock:
    - Itchy, red, bumps on skin + History of new Hairspray → Allergy (Systemic release of Histamine & Other Vasoactive Mediators → Loss of Vasomotor Tone → ↓BP & Compensatory Tachycardia.
- **What has happened to her:**
  - Venous Tone? Decreased
  - Venous Capacitance? Increased
  - Venous Return? Decreased
  - Preload? Decreased
  - Stroke Volume? Decreased
  - Cardiac Output? Decreased
- **Why has she collapsed?**
  - Due to Postural Hypotension → Hypo-Perfusion of Brain → Momentary loss of consciousness. (Regained once supine)
- **Initial Treatment:**
  - Adrenaline – (For the Anaphylaxis)

Case 4 – Lisa:

- Lisa has been playing her saxophone. She collapsed gasping for breath. Her pulse is 120 and her Blood Pressure is 65/45. Neck veins are distended. No breath sounds on the left side. Tension pneumothorax.
- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
- **What Type of Shock is This?**
  - → Obstructive Shock:
    - Spontaneous Tension Pneumothorax from Playing Saxophone → ↑Intra-Thoracic Pressure → Inhibits Cardiac Filling (Seen as raised JVP) → ↓CO → Hypotension & Compensatory Tachycardia
- **How might Lisa's tension pneumothorax cause her to be shocked?**
  - If pressure in the tension pneumothorax is high enough it may:
    - Compress (Decrease) Venous Return to the chest & heart → ↓CO → Shock
    - Shift the Mediastinum such that one/more of the Great vessels gets 'kinked' → ↓CO → Shock
- **Initial Treatment:**
  - Chest Drain – For the Pneumothorax.

Case 5 - Maggie:

- Her pacifier fell in dog poo and wasn't cleaned properly. Now very sleepy. Her skin is a mottled grey colour. Pulse of 180 and blood pressure is 60/40. Angry inflamed area on her face which has pus in the middle of it.

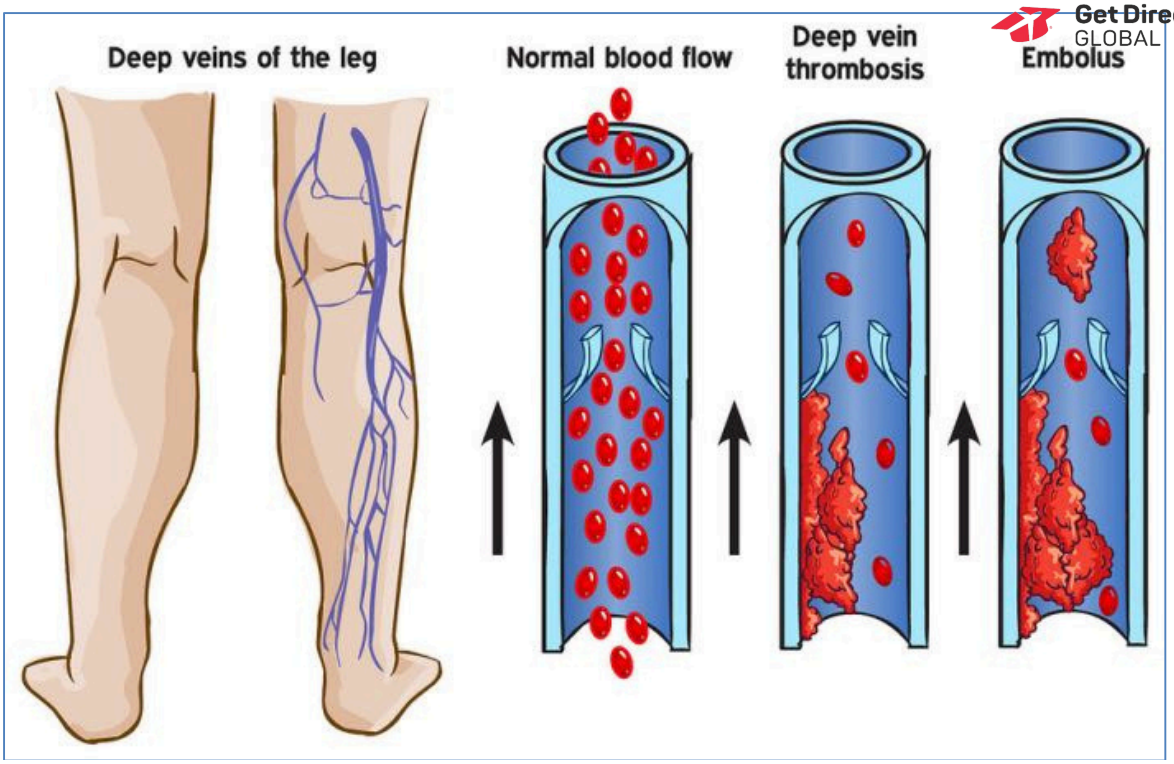
- **What signs of shock are evident?**
  - Tachycardic
  - Hypotensive
  - Grey, colourless skin
- **What Type of Shock is This?**
  - → Distributive (Septic) Shock:
    - Bacterial infection from dog faeces → Endo/Exo Toxin → Systemic Cytokine Release → Loss of Vasomotor Tone → ↓BP → Compensatory Tachycardia
- **How have the following been affected ?**
  - Venous tone? Decreased
  - Vessel Permeability? Increased
  - Myocardial function? Inotropic
- **Initial Treatment:**
  - Antibiotics
  - (Also check Lactic Acid Level):
    - High levels can indicate severe infection
    - & Can indicate lack of Tissue Perfusion & Production of Lactic Acid by Anaerobic Metabolic Pathways.

DEEP VEIN THROMBOSIS ("*PHLEBOTHROMBOSIS*"/"*THROMBOPHLEBITIS*"):

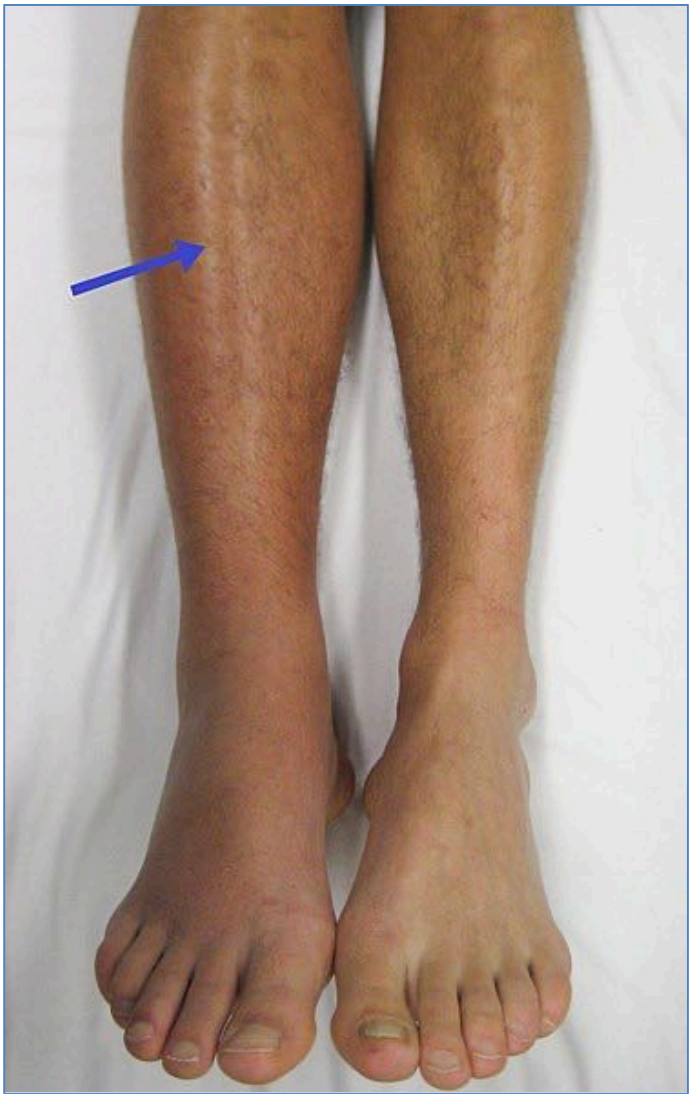
- **Aetiology:**
  - o ****Deep* Venous Valve Incompetence of Lower Limbs:**
    - § → Blood Stasis → Thrombosis
  - o + ****Prolonged Immobilisation*:**
    - § → Blood Stasis → Thrombosis
  - o **+Risk Factors:**
    - § **"Virchow's Triad"**
      - **1- Vessel Damage:**
      - o Surgery/Smoking/Hypertension
      - **2- **Stasis**
        - o Flight/Long Travel/Prolonged Bedrest/Surgery
        - o Obesity/Pregnancy/Congestive Heart Failure
        - o Post-Operative
      - **3- Hypercoagulability**
        - o Cancer (Eg: Adenocarcinoma → Paraneoplastic Syndrome)
        - o Congenital: Eg: Antithrombin III Deficiency /Factor 5 Leiden
        - o Drugs: Eg: Oral Contraceptive/HRT
        - o Hyperviscosity: Eg: Pregnancy/Polycythaemia
- **Pathogenesis:**
  - o **Failure/Inactivity of the Venous Calf Pump (Immobility/Valve Insufficiency)**
    - § Blood Stasis & Pooling in Leg Veins → Coagulation → Thrombosis
- **Clinical Features:**
  - o **Symptoms:**
    - § **Localized Symptoms – (Typically in Calf):**
      - Tenderness (Elicited by Pressure/Passive Dorsiflexion)
      - Heat, Redness, Swelling
      - Distal Oedema
      - Distal Cyanosis
      - Superficial Venous Dilation
    - § ****Pulmonary Embolism – May be the 1st Manifestation*:**
      - Thromboembolism into Pulmonary Artery → Biventricular Heart Failure
        - o → Sudden Chest pain, Dyspnoea, Haemoptysis, Collapse, Death.
- **Investigations:**
- o **Duplex Doppler USS –** (93% Sensitive; 98% Specific)

Management:

- o ****Oral Anticoagulation (or Heparin if contraindicated)**
- o **+/- Thrombectomy**
- o **+/- IVC Filter – (To Prevent Pulmonary Embolus)**



Deep Vein Thrombosis (DVT). Contributed by Creative Commons (CC BY-ND 2.0)
https://www.ncbi.nlm.nih.gov/books/NBK470381/figure/article-20301.image.f2/?report=objectonly



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PULMONARY EMBOLISM

- **Aetiology:**
  - o **95% = DVT → Thrombo-Emboli**

Pathogenesis:

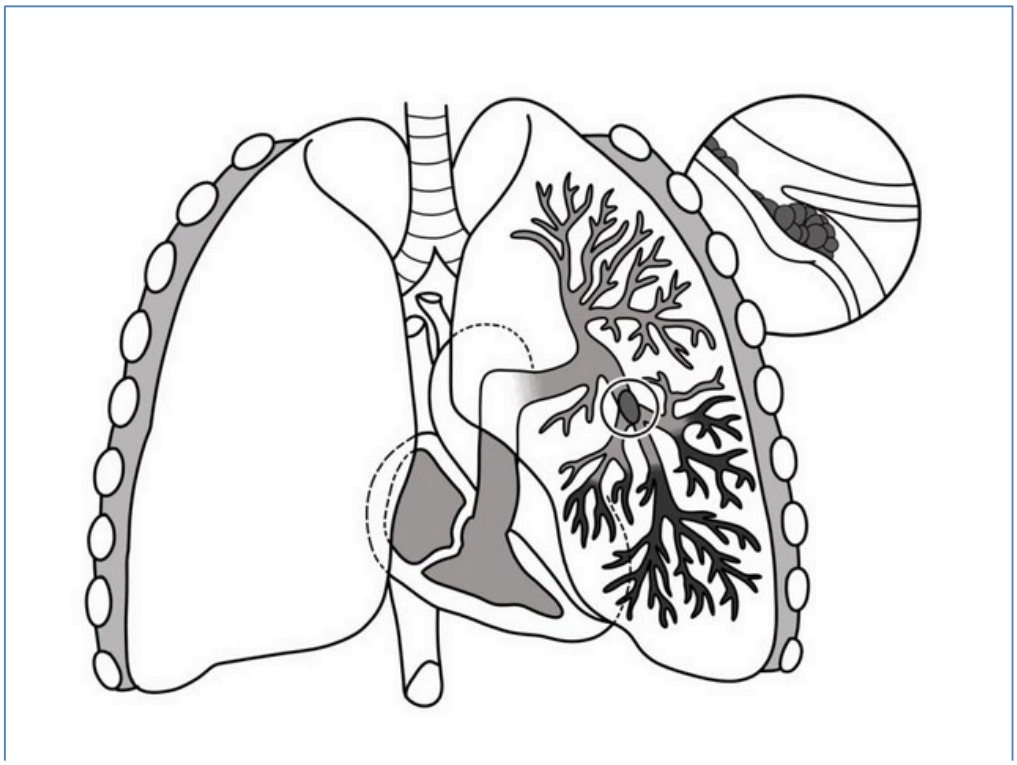
- o **DVT → Thrombo-Emboli Lodges in Pulmonary Arteries →**
  - § ***1- → VQ-Mismatch*** → *Respiratory Compromise* → ***(Respiratory Failure)***
  - § ***2- → ↑Pulmonary Vascular Resistance*** → *Haemodynamic Compromise* → ***(Heart Failure)***.

- **Clinical Features:**
  - o Severity Depends on Size/Number of Emboli (Extent of Obstruction)
  - o If Severe → Instant Death!! (Due to sudden Cardiac Failure)
  - o **Symptoms →**
    - § Pleuritic Chest Pain (+ Pleural Rub)
    - § Dyspnoea/Tachypnoea
    - § Cough/Haemoptysis
    - § **(+ DVT Symptoms)**
  - o **Signs:**
    - § RV-Failure (↑JVP, Tricuspid Regurg)
    - § Shock/Syncope
    - § Fever
    - §

- **Diagnosis:**
  - o ****CTPA (CT-Pulmonary Angiogram):** Shows Large Emboli lodged in Major Pulmonary Artery
  - o **ECG:** Classical S1Q3T3 Pattern
  - o **VQ Scan:** Shows VQ Mismatch
  - o **CXR (Later >1day):** Shows Wedge-Shaped Pulmonary Infarct

- **Treatment:**
  - o o **Give Oxygen**
  - o **TPA-Thrombolysis (If Haemodynamic Compromise)** ****Oral Anticoagulation (or Heparin if contraindicated)**
    (+/- Trombectomy & IVC Filter)

- **Prevention (in High Risk Individuals):**
  - o Elastic/Compression Stockings
  - o Anticoagulation
  - o If Severe Risk, Insertion of a IVC-Filter



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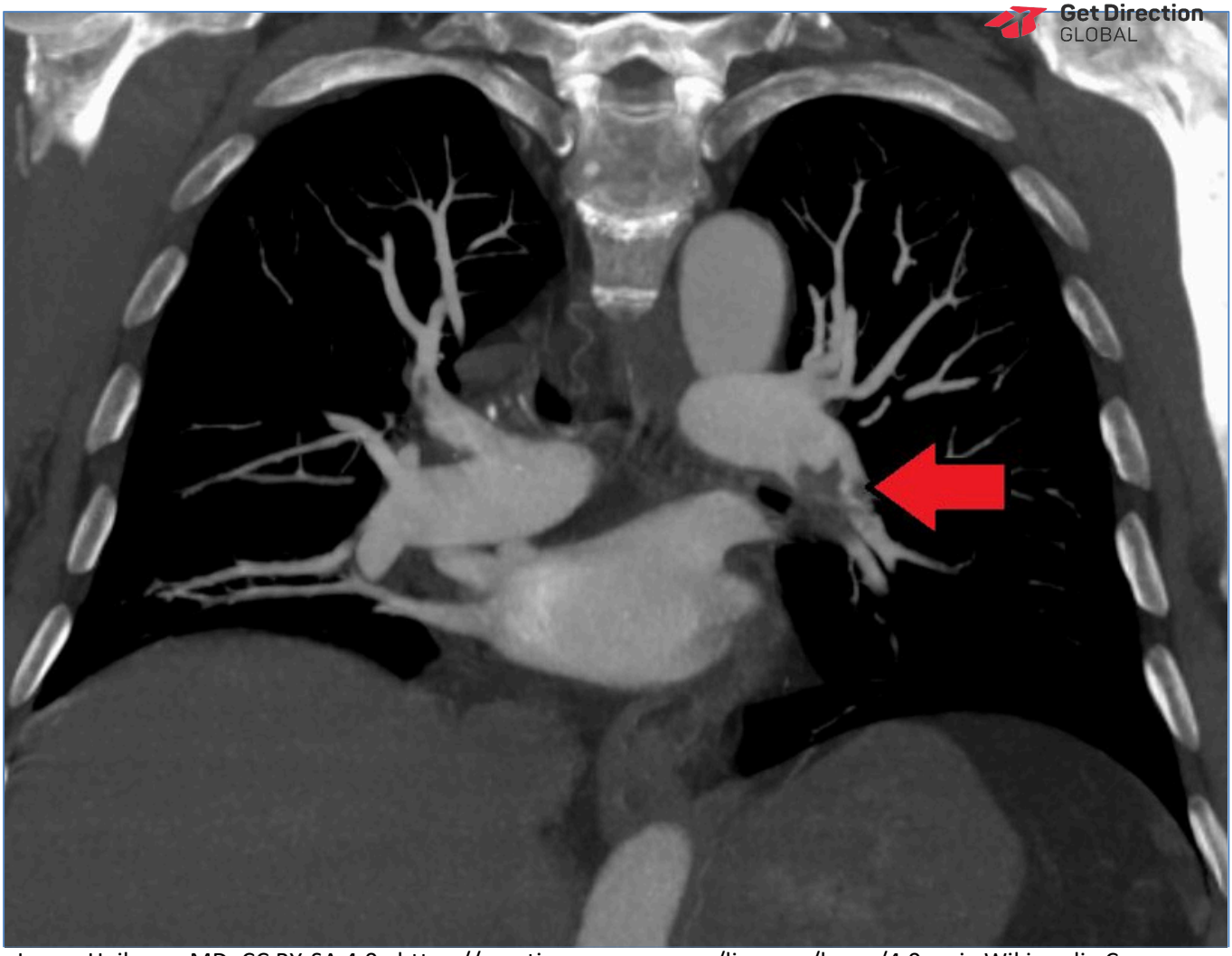

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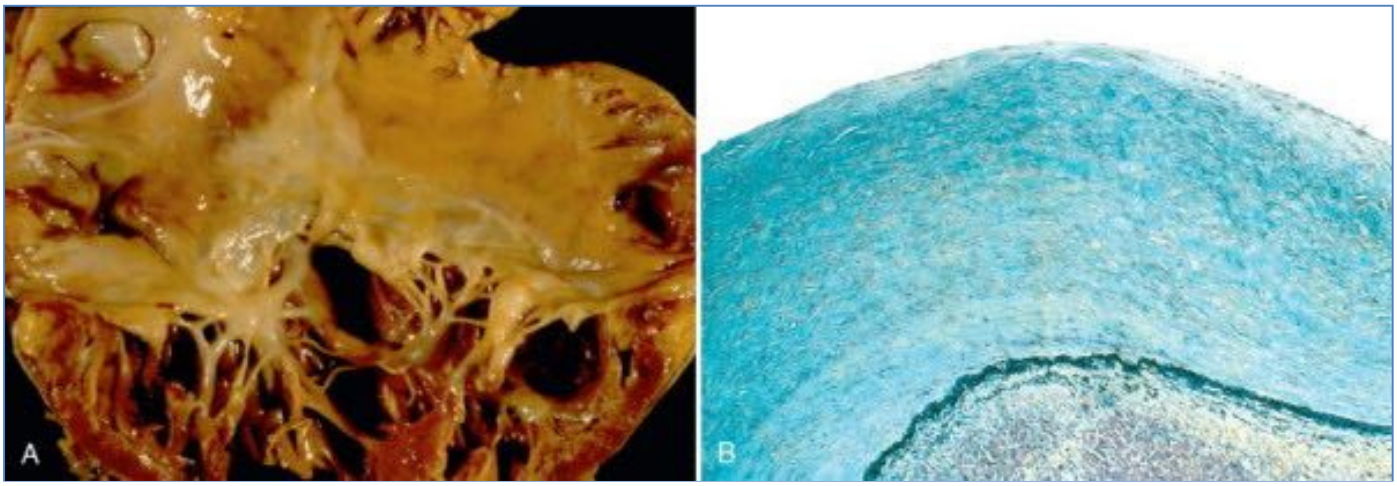
CARCINOID HEART DISEASE:

- **Aetiology:**
  - o Cardiac Manifestation of the Systemic Syndrome caused by Carcinoid Tumours.
- **Pathogenesis:**
  - o **Carcinoid Tumour Releases Vasoactive Hormones into Venous Circulation**
    - § Serotonin / Bradykinin / Histamine / Prostaglandins
  - o **Venous Drainage of these Hormones → R-Heart → R-Heart Endocardial & Valvular Fibrosis**.
    - § (Generally → Fibrosis of R-Heart Valves (Tricuspid/Pulmonary))
- **Clinical Signs:**
  - o **"Carcinoid Syndrome":**
    - § Episodic flushing of skin
    - § Cramps
    - § Nausea/Vom/Diarrhoea.
  - o **Heart Manifestations (RV-Failure due to..):**
    - § ***Tricuspid Regurgitation (Most Common)**
      - → Hepatomegaly/Pain
      - → Pulsatile Liver
      - → ↑JVP with Prominent V-Waves
      - → Systolic Murmur @ 4th ICS, L-Sternal Border, Louder on Inspiration
    - § ***Pulmonary Regurgitation**
      - → Dyspnoea
      - → Diastolic Murmur @ 2nd ICS, L-Sternal Border, Louder on Inspiration
    - § **(+ Features of RV-F):**
      - → Peripheral Oedema
      - → Organomegaly
      - → Portal HTN (Caput Medusa, Spider Naevi)
      - → ↑JVP
- **Investigations:**
  - o **24hr Urinary 5-HIAA** (A Serotonin Metabolite)
  - o **Abdo CT + Somatostatin Receptor Scintigraphy (SRS)** – (Tumour Localisation)
  - o **Abdo MRI + Contrast**
  - o **Cardiac Ix – (ECG, CXR, ECHO)**
- **Management:**
  - o **Medical – Somatostatin Analogues (*Octreotide*)**
    - § *+/- Interferon-A* in Palliative Pts.
  - o **Surgery – Tumour Resection + Valvuloplasty**
- **Prognosis:**
  - o **Can Metastasize :. Early Removal is Essential**



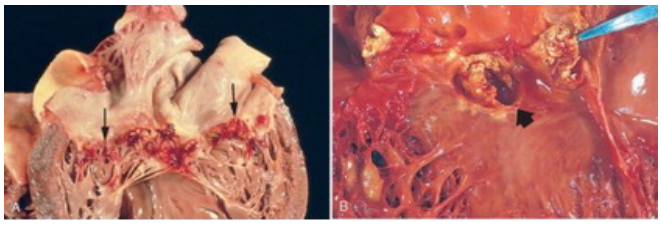
(**A**, Characteristic endocardial fibrotic lesion involving the right ventricle and tricuspid valve. **B**, Microscopic appearance of carcinoid heart disease with intimal thickening.)

INFECTIVE ENDOCARDITIS

= Infection of the Endothelial Lining of the Heart (including the heart valves)

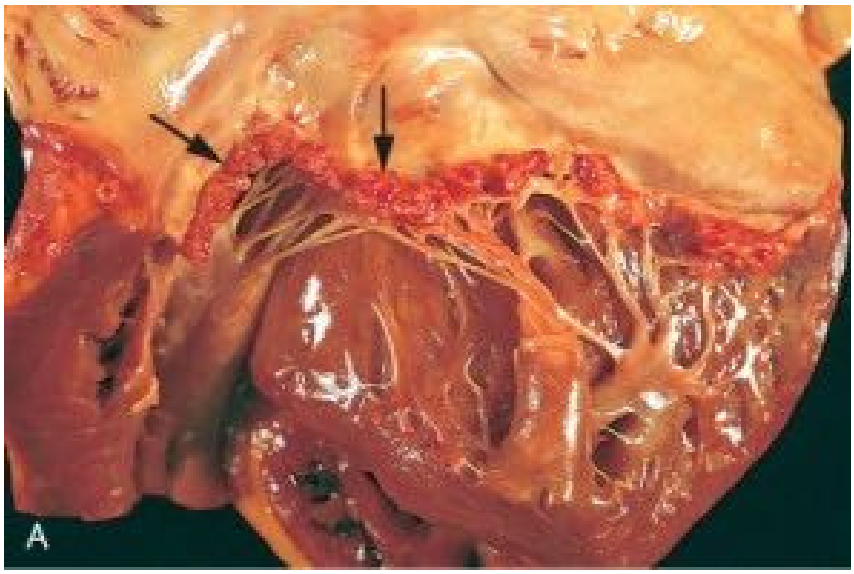
- **Risk Factors:**
  - Valve Abnormality – (Valve Murmurs, Calcification, Congenital, Artificial)
  - Open-Heart Surgery
  - Poor Dental Hygiene (Source of Bacteria) – Bacteraemia (Haemodialysis, IVDU, Surgery)
  - IV Access
  - Immunosuppression

- **Aetiologies:**
  - **Subacute Bacterial Endocarditis (Most Common - 50-60% of Cases):**
    - *(Oral) Strep Viridans/(Surgical) Strep Epidermidis* (Low Virulence)
    - **Epi:** Recent Oral Surgery, or Post-Prosthetic Valve Insertion.
  - **Acute Bacterial Endocarditis (Rare – 10-20% of Cases):**
    - *Staph. Aureus* (High Virulence - 50% Mortality)
    - **Epi:** IV Drug Users
- **Pathogenesis:**
  - **Bacterial Infection of Valves/Endocardium** → Vegetations on Valve Cusps
    - Typically *Strep. Viridans* (Subacute-BE) or *Staph Aureus/MRSA* (Acute-BE)
    - **Affects Aortic & Mitral Valves**; (RH-Valves may be affected in IV Drug Users)



- **Clinical Signs:**
  - **Symptoms:**
    - *****Fever + New Murmur***** **= Endocarditis until proven otherwise**
    - +Fatigue, Malaise, Weight Loss
  - **Physical Signs:**
    - **Septic Emboli → Infarcts:**
      - **Splinter Haemorrhages** (In the nail bed)
      - **Osler's Nodes** (painful erythematous nodules in pulp of digits)
      - **Janeway Lesions** (Red, nontender lesions on palms/soles)
      - **Roth Spots** (Retinal Haemorrhages - red ring lesions with a yellow centre)
    - Splenomegaly
    - Arrhythmia
  - **Complications – (Begin ≈2wks after onset):**
    - **Renal Failure** (Renal Emboli/Immune Complex Deposit → Glomerulonephritis, Haematuria)
    - **TIA** (Cerebral Septic Embolism → Ischaemia → TIA/Stroke)
    - **Septicaemia**
    - **CCF**
- **Investigations:**
  - **Clinical –** (Fever + New Systolic Murmur +/- Septic Emboli)
  - **3x Blood Cultures –** (@ Different Times & From Different Sites – Eliminate Contamination)
  - **ECG –** (Rule out Ischaemia/MI/Arrhythmias)
  - **Echo –** (Valvular Vegetations & Mitral Regurgitation)
- **Management:**
  - **2-6wks of High Dose IV *Vancomycin* -** (Initially Empirical; Then Culture-Directed Therapy).
  - **Refer to Cardiac Surgeon – (For ?Valve Replacement Surgery?):**
    - If IV-ABs are Unsuccessful.
    - Or If Valve is Destroyed (Ie: In Acute-BE) → Heart Failure
- **Prognosis:**
  - 30% Mortality with Rx.

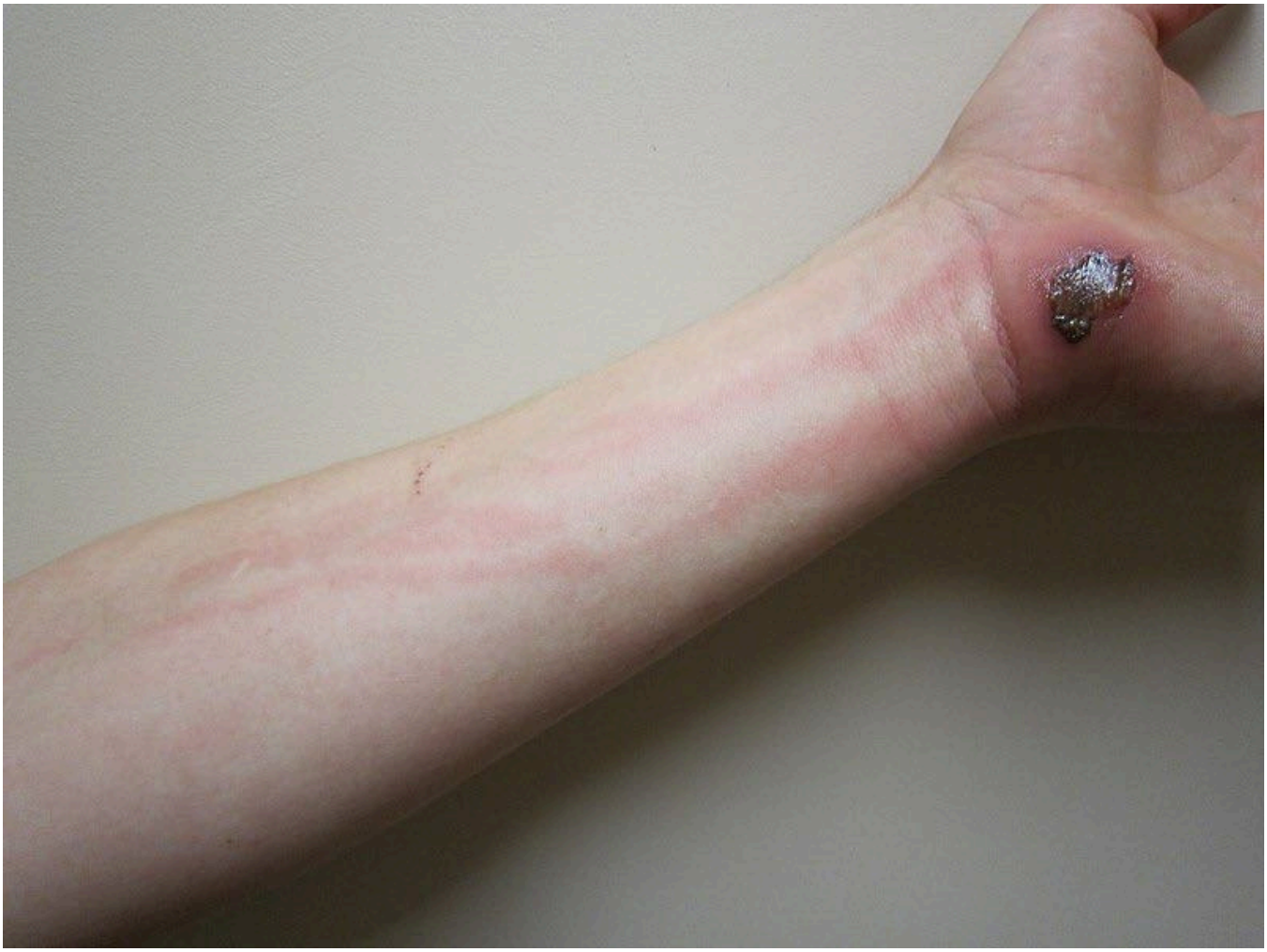
- **Aetiology:**
  - **Hypercoagulable States –** Eg: DIC, Malignancy, Sepsis, SLE, Pregnancy.
- **Pathogenesis:**
  - Deposition of small *Sterile* Thrombi on leaflets of Cardiac Valves (Ie: The suffix "itis" is NOT correct)
  - **Preference for Valves:** Mitral>Aortic>Tricuspid>Pulmonary
- **Clinical Signs:**
  - **Signs:**
    - **Of Hypercoagulable States:**
      - **DIC:** Acutely Ill, Shocked, Widespread Haemorrhage (Mouth, Nose, Bruising, Renal)
      - **Sepsis:** Fever, Acutely Ill, Shocked, Infective Focus
      - **SLE:** Fever, Fatigue, Malaise, Butterfly/Malar Rash, Lymphadenopathy, Arthritis
      - **Pregnancy:** Baby Bump, DVT
    - **Of NBTE:**
      - Heart Murmurs
      - Stroke
      - MI
    - **If 2o Infective-BE:**
      - Fever + New Murmur
      - Septic Infarcts (Splinters, Oslers, Janeways, Roth's, Abscesses, Haematuria)
  - **Symptoms are those of Systemic Arterial Embolism (Complications):**
    - Thrombo-Embolic Infarcts (Eg: Brain →Stroke; Heart →MI)
    - Secondary Bacterial Colonisation on Vegetations.
- **Investigations:**
  - **Clinical –** (Fever + New Systolic Murmur +/- Septic Emboli)
  - **3x Blood Cultures –** (@ Different Times & From Different Sites – Eliminate Contamination)
  - **+ Coags Screen!!**
  - **ECG –** (Rule out Ischaemia/MI/Arrhythmias)
  - **Echo –** (Valvular Vegetations)
- **Management:**
  - **Treatment of Underlying Aetiology**
  - **Anticoagulant Therapy** (*Heparin* Then *Warfarin*)
  - **+(If 2o Bacterial Endocarditis → 2-6wks of High Dose IV *Vancomycin*)**
  - **Refer to Cardiac Surgeon – (For ?Valve Replacement Surgery?):**
    - If IV-ABs are Unsuccessful.
    - Or If Valve is Destroyed (Ie: In Acute-BE) → Heart Failure



Source: Non-Attributable

LYMPHANGITIS:

- **Aetiology:**
  - o Commonly ***β-Haemolytic-Strep*** or ***Staphylococcus Aureus***
- **Pathogenesis:**
  - o Bacterial Infection Spread to Lymphatics → Acute Inflammation
    - § **If Severe** → Cellulitis/Abscesses
    - § **If Very Severe** → Bacteraemia/Sepsis
- **Clinical Features:**
  - o Fever/Chills/Malaise
  - o Painful Red Subcutaneous Streaks
  - o Painful Lymphadenitis (Swollen draining lymph nodes)
- **Com plications:**
  - o Abscesses
  - o Cellulitis
  - o Sepsis
- **Investigations:**
  - o Blood Culture + Swab any open wounds.
  - o FBC +/- CRP
- **Management:**
  - o Immobilisation of Limb
  - o Antibiotics
  - o Analgesia



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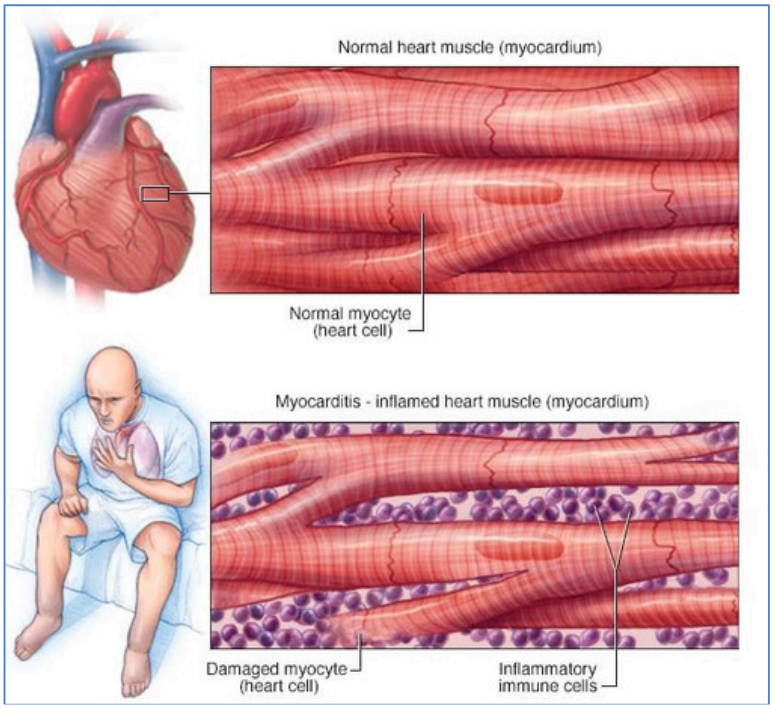
MYOCARDITIS – VIRAL & TOXIC:

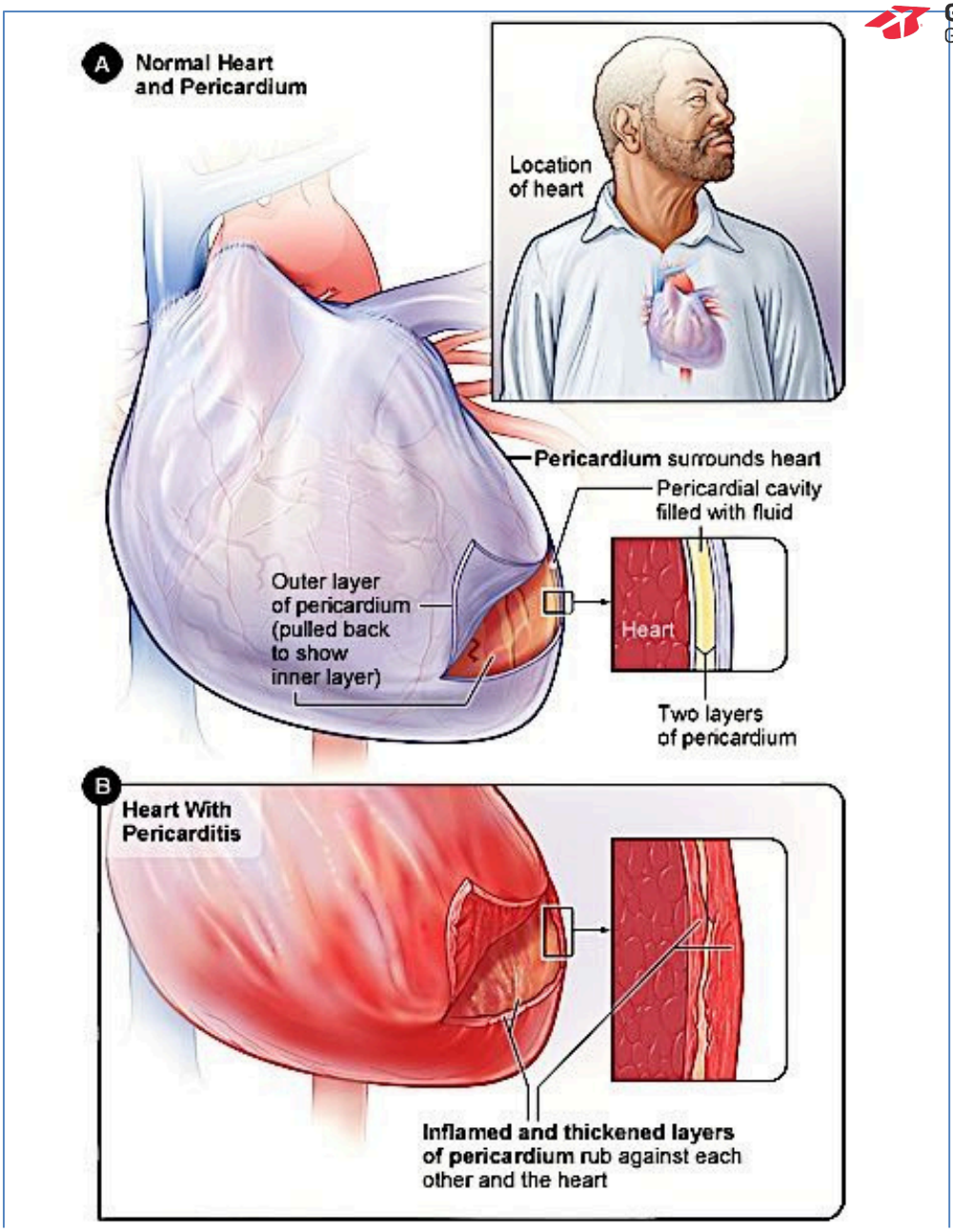
- **W hat is it?**
  - o **"Inflammation of the Heart Muscle"**
  - o + Characterized by Myocyte Necrosis – (Positive Troponin I results seen in 35% of Myocarditis)
- **2 M ain Aetiologies:**
  - o **VIRAL MYOCARDITIS**. (Eg: Coxsackievirus, Rhinovirus, Influenza, Parvovirus B19, Coronavirus)
    - § **Either *Direct Myocardial Injury* OR 2o *AutoImmune Response***
    - § →Heart Thickens & Weakens → Systolic Heart Failure
  - o **TOXIC MYOCARDITIS** (Eg: Chemo Drugs, Cocaine, Alcohol, Diuretics, Antibiotics, Venom, CO, Spike Protein)
    - § Myocardial Damage & Inflammation due to Either:
      - Hypersensitivity to Drugs
      - Direct Toxic Damage
- **Clinical Features:**
  - o (May be Asymptomatic)
  - o **Symptoms:**
    - § **Flu-Like Syx** - (Fever, Fatigue, Malaise)
    - § **LV-Failure** – (Dyspnoea/Orthopnoea/PND/Cough)
    - § **Chest Pain** – (Due to Myocarditis +/- Pericarditis)
    - § **Palpitations** (Arrhythmias)
- **Com plications:**
  - o **Cardiomyopathy** → Heart Failure
  - o **Arrhythmias** → ***Sudden Death***
  - o **Pericarditis** → Pericardial Effusion
- **Investigations:**
  - o **ECG & Continuous Telemetry**
  - o **Serial Troponins I/T** - (Immediately, then @6hrs, then @24hrs)
  - o **FBC** (↑WCC), **CRP** (↑), **ESR** (↑)
  - o **CXR** (Cardiomegaly)
  - o **Echo** (Dilated, Poor Vent-Function)
- **Management:**
  - o o **Bed Rest**
  - o ****GDMT Triple Therapy (ACEi/ARB + B-Blocker + Diuretics)
  - o **Warfarin** (Prevent Thromboembolism)
  - o Supportive Rx. (Fluids, Analgesia)
  - o Treat Underlying Cause if Possible



Mayo Foundation for Medical Education and Research (MFMER); IMG-20456507

PERICARDITIS:

- **Aetiology:**
  - **Usually *Secondary* to:**
    - **Infection** (**Viruses**, Bacteria, Fungi, Parasites)
    - **Immunological** (Rheumatic Fever, SLE, Post-MI, Drug Hypersensitivity)
    - **Other** (MI, Post-Cardiac Surgery, Neoplasia, Trauma, Radiation)
- **Classification:**
  - **According to Composition of Pericardial Exudate:**
    - **Serous** (Non-Infectious Inflammatory Diseases – SLE, Uraemia, Tumours)
    - **Purulent** (Infective - by Microbes)
    - **Fibrinous/Serofibrinous** (Due to Acute MI, Ch. Radiation, SLE, Trauma)
    - **Caseous** (Tuberculosis)
    - **Haemorrhagic** (Due to Metastasis, Cardiac Surgery).
- **Pathogenesis:**
  - Various Aetiologies → Inflammation of the Pericardium
    - → Thickening of Pericardium→ Pericardial Exudate (Serous Fluid + Pus/Fibrin/Blood)
      - →Rubbing of Parietal & Visceral Pericardium → Further Inflammation & Exudate.
- **Clinical Features & Complications:**
  - **Symptoms:**
    - Pleuritic Chest Pain (Better on Sitting Forward; Worse on Inspiration & Lying Down)
    - Fever, Fatigue
    - Dry Cough
    - Syx of CCF (Dyspnoea, Fatigue)
  - **Signs:** Fever, Tachycardia
    - Muffled Heart Sounds.
    - Friction Rub
    - ↑JVP
    - Heart Failure Signs if Tamponade
    -
  - **Complications:**
    - Cardiac Tamponade/Pericardial Effusion
    - If >3mths → Chronic → Constrictive Pericarditis (Requires Surgery)
- **Diagnosis:**
  - **ECG –** (Classical PR-Depression + ST-Elevation + Tachycardia)
  - **CXR –** (Pulmonary Congestion)
  - **ECHO –** (?Pericardial Effusion)
- **Management:**
  - Rx Underlying Cause
  - Anti-Inflammatories (NSAIDs / Steroids)
  - Analgesia



https://www.nhlbi.nih.gov/health-topics/heart-inflammation

CONSTRICTIVE PERICARDITIS:

- **Definition:**
  - Chronic pericarditis resulting in fibrosed, thickened, adherent, and/or calcified pericardium
- **Aetiology:**
  - Any cause of acute pericarditis may result in chronic pericarditis
  - Major causes are tuberculous, radiation-induced, post-cardiotomy, or idiopathic.
- **Symptoms:**
  - Dyspnoea
  - Fatigue
  - Palpitations
  - Abdominal Pain
- **Signs:**
  - General examination - mimics CHF (especially right-sided HF)
  - § Ie: Ascites, hepatosplenomegaly, oedema
  - Increased JVP, Kussmaul's sign (Paradoxical increase in JVP with inspiration), Friedrich's sign
  - (Prominent "y" descent > "x" descent)
    Pressures: BP normal to decreased +/- pulsus paradoxus
    Precordial Examination: +/- Pericardial knock (early diastolic sound)
- **Investigations:**
  - 12-lead ECG
  - § Low voltage, flat T-wave +/- A.Fib
  - CXR Pericardial calcification, effusions
  - §
    CT/MRI/TEE
    § Pericardial thickening
    Cardiac catheterization
    § Equalization of RV and LV diastolic pressures, RVEDP >1/3 systolic pressure
- **Management:**
  - Medical: Diuretics, salt restriction
  - Surgical: Pericardiectomy

PERICARDIAL EFFUSION:

Get Direction GLOBAL

Aetiology:

- **2 types of effusions:**
  - **Transudative (Serous):**
  - § CHF, hypoalbuminemia/hypoproteinemia, hypothyroidism
  - **Exudative (Serosanguinous or bloody):**
    - § Causes similar to the causes of acute pericarditis
    - § May develop acute effusion secondary to haemopericardium (Trauma, post MI myocardial rupture, aortic dissection)
- Physiological consequences depend on type and volume of effusion, rate of effusion development, and underlying cardiac disease.

Symptoms:

- None or similar to acute pericarditis
- Dyspnoea, cough
- Extra-cardiac (Oesophageal/recurrent laryngeal nerve/traceho-bronchial/phrenic nerve irritation)

Signs:

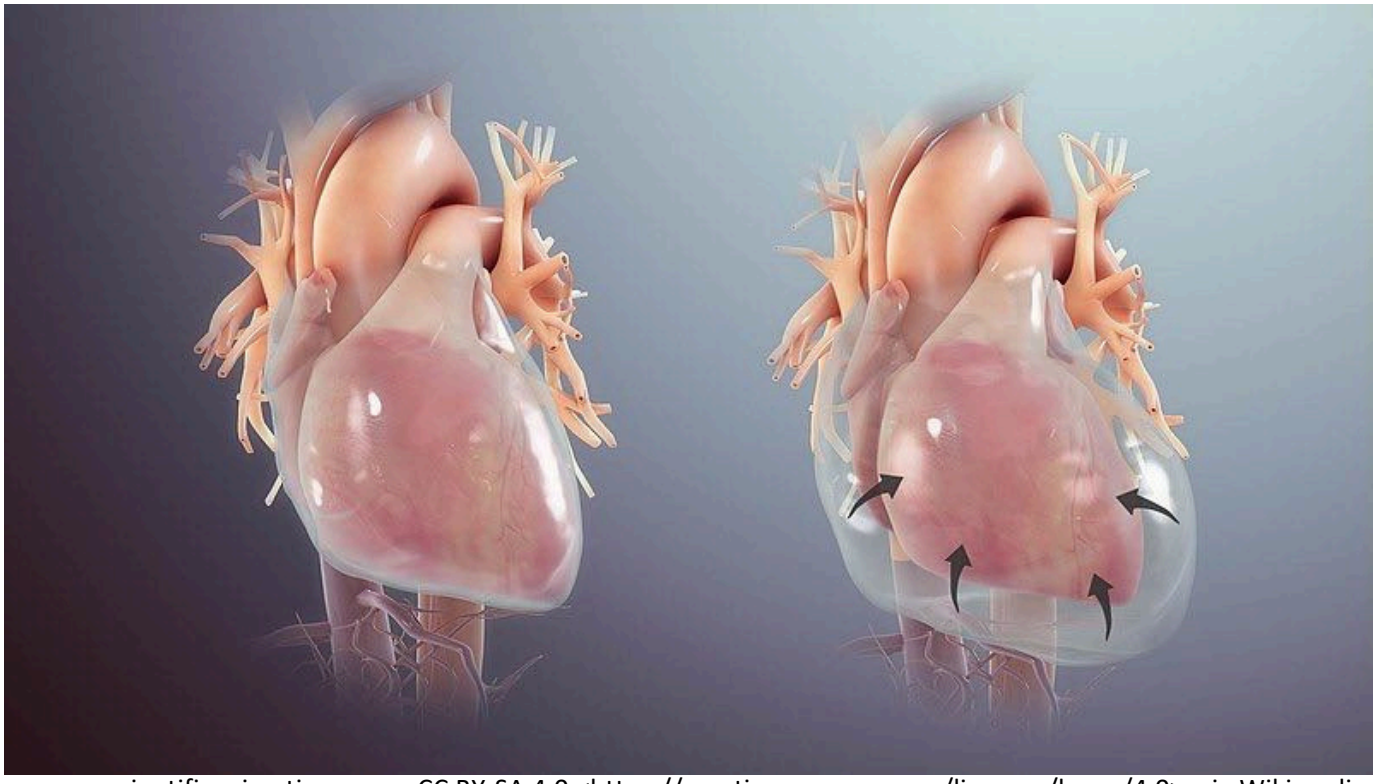
- JVP: Increased with dominant “x” descent
- Arterial Pulse: normal to decreased normal, decreased pulse pressure
- Auscultation: distant heart sounds +/- friction rub

Investigations:

- **12-lead ECG:** Low voltage, flat T-Waves
- **CXR:** Cardiomegaly, rounded cardiac contour (water bottle)
- **ECHO:** Fluid in pericardial sac
- **Pericardiocentesis:** Establishes diagnosis

Management:

- **Mild:** Frequent observation with serial ECHO, treat the cause, anti-inflammatory agents
- **Severe:** May develop cardiac tamponade.



CARDIAC TAMPONADE:

General Info:

- Cardiac tamponade is a major complication of pericardial effusion
- Cardiac tamponade is a clinical diagnosis

Pathophysiology:

- High intra-pericardial pressure → decreased venous return → decreased diastolic ventricular filling → decreased CO → hypotension + venous congestion

Symptoms:

- Tachypnoea
- Dyspnoea
- Shock

Signs:

- "x" descent only, absent "y" descent
- Hepatic congestion
- **Classic Quartet of symptoms:**
  - 1 – Hypotension
  - 2 – Increased JVP
  - 3 – Tachycardia
  - 4 – Pulsus paradoxus
- **Beck's Triad:**
  - 1 – Hypotension
  - 2 – Increased JVP
  - 3 – Muffled heart sounds

Investigations:

- **12-lead ECG:**
  - Electrical alternans (pathognomonic variation in R-wave amplitude)
  - Low voltage
- **ECHO:** Pericardial effusion
  - Compression of cardiac chambers (RA & RV) in diastolic
- **Cardiac catheterization:**
  - Mean RA, LA, LV, & RV diastolic pressures all high and equal

Management:

- Pericardiocentesis – ECHO-/ECG-guided
- Pericaridotomy
- Avoid diuretics & vasodilators (these decrease venous return to already under-filled RV → Decreased LV
- Preload → Decrease CO)
- Fluid administration may temporarily increase CO
  Treat underlying cause

ACUTE ARTERIAL OCCLUSION ("*CRITICAL LIMB ISCHAEMIA*"):

Get Direction GLOBAL

General Info:

- Due to embolus, arterial thrombosis or trauma
- Time is of the essence, after approx 6hrs (depending on collateral circulation), ischemia and myonecrosis is irreversible to the limb.

Those caused by Embolus:

- **Aetiology:**
  - Cardiac is 80-90% of embolic episodes;
    - Eg: History of MI (<3mths),
    - Eg: rheumatic heart disease,
    - Eg: abnormal or prosthetic valves,
    - Eg: A-fib,
    - Eg: cardiomyopathy,
    - Eg: endocarditis,
    - Eg: atrial myxoma
- **Presentation:**
  - Sudden pain in lower extremity progressing within hours to a feeling of cold numbness, loss of
  - function & sensation.
  - No history of significant vascular claudication
  - Pulses are present in contralateral limb
    May have emboli to other locations (cerebral, upper limb, renal)

Those caused by Arterial Thrombus:

- **Aetiology:**
  - It is important to differentiate thrombosis from embolism because the treatment for the two vary
  - dramatically
    Thrombosis usually occurs in a previously diseased (atherosclerotic) artery, congenital anomaly, infection, haematological disorders and low flow rates (CHF)
- **Presentation:**
  - Gradual progression of symptoms; but may have an acute-on-chronic event
  - Progression to loss-of-function and sensory loss may be less profound than with acute embolus
  - Past history of claudication
  - Atrophic changes may be present
  - Contralateral disease may be present

Those caused by Trauma:

- **Aetiology:**
  - Important to determine a history of arterial trauma, arterial catheterisation, intra-arterial drug induced injection, aortic dissection, severe venous thrombophlebitis, prolonged immobilisation, idiopathic.
- **Symptoms (6 'P's):**
  - Pain (although may be absent if prompt onset of anaesthesia and paralysis)
  - Pallor: replaced by mottled cyanosis within a few hours
  - Paraesthesia: light touch goes first (small fibers) followed by other sensory modalities (large fibers)
  - Paralysis/Power loss: Heralds impending gangrene
  - Polar (cold)
  - Pulselessness

Critical Limb Ischaemia (Clinical Context):

- **Investigations:**
  - CXR ECG
  - Arteriography
  -
- **Management:**
  - Immediate heparinization and continuous infusion to maintain PTT >60
  - In the absence of power and sensation - Patient needs emergent re-vascularization:
    - Eg: Embolectomy for embolus
    - Eg: Bypass if thrombus
  - Continue heparin post op; start warfarin post-op day 1 for 3mths
  - Amputation if irreversible ischaemia
- **Complications:**
  - Compartment syndrome if prolonged ischaemia (may require fasciotomy)
  - Re-perfusion syndrome (Toxic metabolites from ischaemic muscle → Renal failure & multi-organ system failure)
  - **Emboli Can also Deposit in *Other* Arteries Too:**
    - Mesenteric Ischaemia → Ischaemic Gut (++ Painful + Bloody Diarrhoea)
    - Renal Artery Thrombosis → Abdo/Back/Flank Pain, ARF, Oliguria, Hypertension
- **Prognosis:**
  - 12-15% mortality rate
  - 5-40% morbidity rate (amputation)

- **D e fin itio n :**
  - Obstruction of any of the *PERIPHERAL ARTERIES* (Not including Coronaries/Aortic Arch/Brain)
- **Aetiologies:**
  - ****Atherosclerosis (Most Common)**
  - **(Major Risk Factors):**
    - Smoking (10x) - the single greatest *modifiable* cause of PVD.
    - Diabetes
    - Dyslipidaemia
    - Hypertension
- **Pathogenesis:**
  - **Atherosclerosis** →Obstruction of Peripheral Arteries → Chronic Ischaemia
    - → Eg: Arterial Ulcers, Leg Claudication, Raynaud's Phenomenon.
- **Clinical Features:**
  - **Symptoms:**
    - **(Acute Critical Limb Ischaemia – See Prev. Page):**
    - • *Pain, Pallor, Paraesthesia, Paralysis, Pulseless*

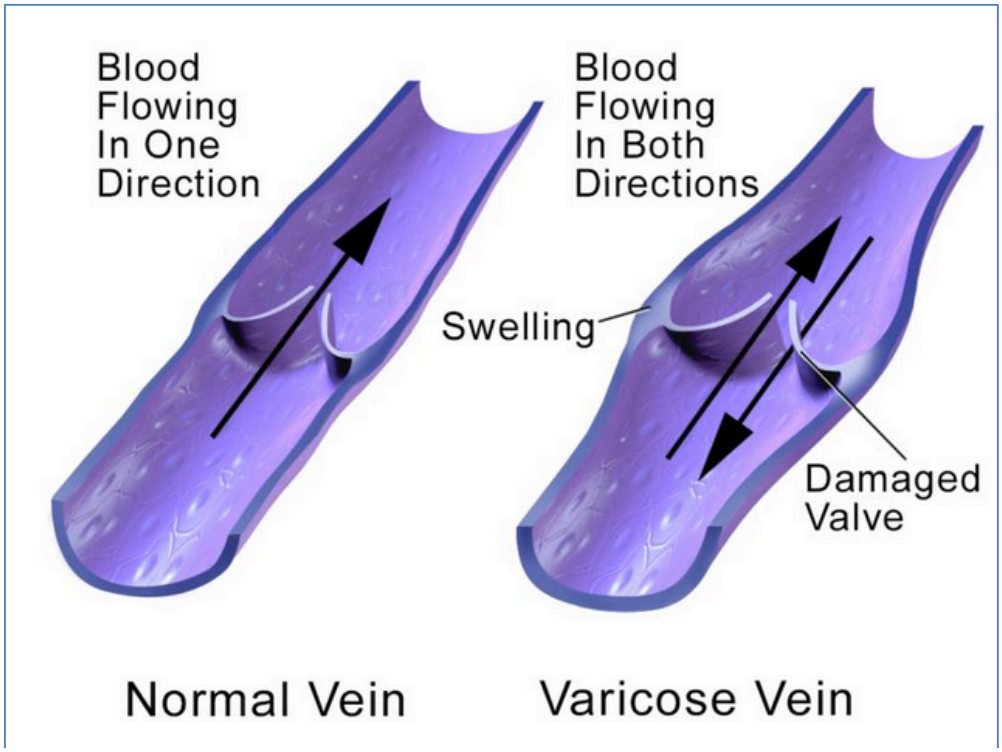
      Chronic:
      - **Mild-Severe Claudication** (Leg Pain/Cramping/Weakness on Exercise)
        - 1- On Exertion (Typically in Calves)
        - 2- Relived by Rest (2-3mins)
        - 3- Reproducible (Same "Claudication Distance")
        - (+ Rest Pain if SEVERE)
      - Distal Pulses Weak/Absent
      - Skin Changes (Hair-Loss, Atrophic Skin, Ulcerations, Gangrene)
      - Other Atherosclerotic Lesions – (Impotence, CVD, CAD)
- **Investigations:**
  - **Non-Invasive:**
    - **ABI (Ankle-Brachial Index):**
      - Ankle BP <90% of Brachial BP = Abnormal
      - ABI <0.3 → "Rest Pain & Night Pain" → *(↑Risk of Critical Limb Ischaemia)
    - **Doppler Ultrasound**
    - **Contrast CT-Angiogram**
  - **Invasive:**
  - ****Femoral Angiography (DSA Lab) =** Gold Standard

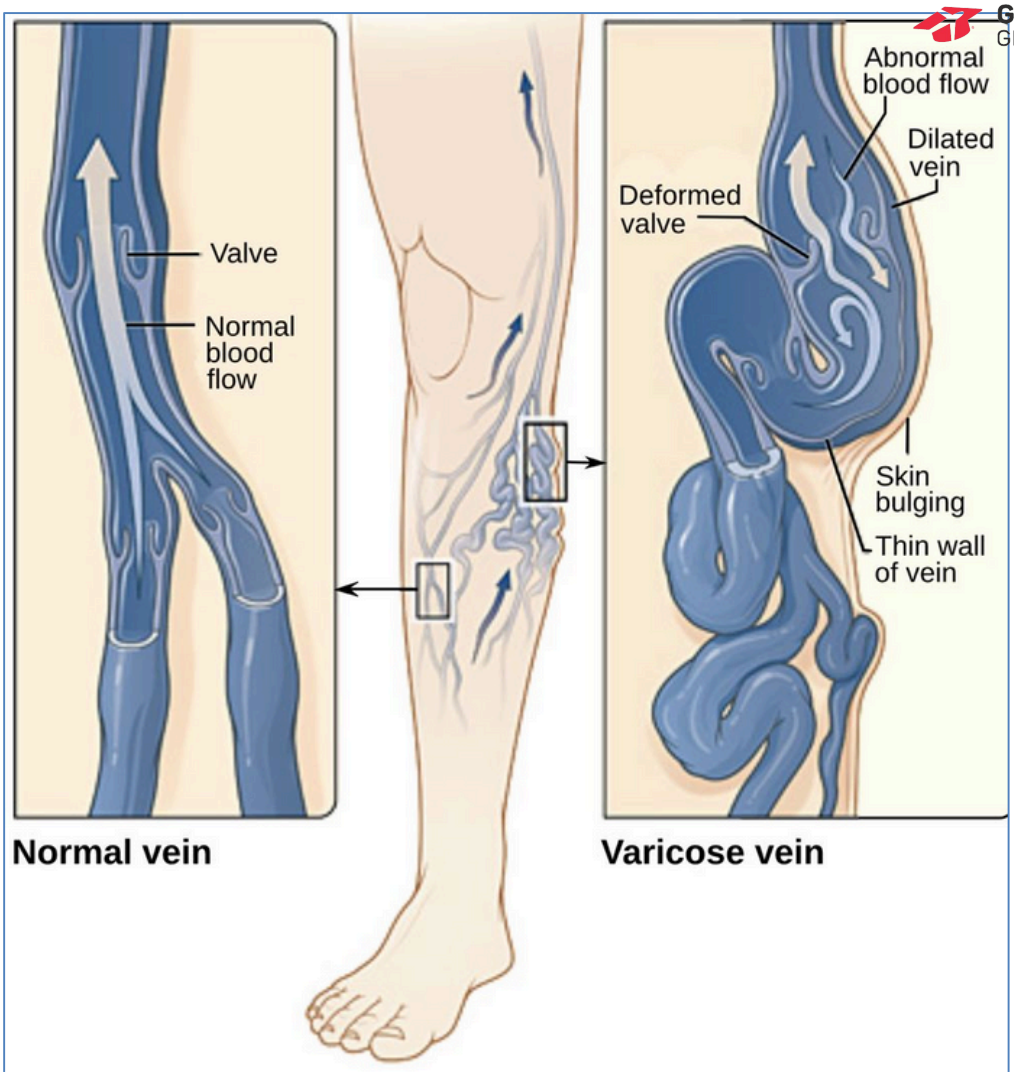
    (Note: Check for Carotid-Artery Stenosis!!)
- **Treatment:**
  - **1- Conservative Mx *Can* → 70% Improvement:**
    - Stop Smoking, ↓ETOH, Control Diabetes/↓Dietary Cholesterol/HTN.
    - ↑Exercise
  - **2- Medical Management:**
    - Cholesterol-Lowering (Statins/Fibrates/Bile-Resins(Cholestyramine)/Ezetimibe)
    - Antihypertensives (B-Blockers, ACE-Is/ARBs, Ca-Ch-Blockers)
    - Diabetes Mx
    - Champix
  - **3- Surgery:**
    - Angioplasty (Balloon + Stent)
    - Bypass Grafting (Eg: Femoral-Popliteal Bypass)
    - Plaque Excision (Endarterectomy)
    - Amputation

https://www.ncbi.nlm.nih.gov/books/NBK519606/

VARICOSE VEINS:

- **Aetiology:**
  - o **Mechanical:** Prolonged Leg Dependence
  - o (Risk Factors = Obesity, Pregnancy, Familial)
- **Pathogenesis:**
  - o ***Superficial Valve Incompetence*** (Due to Incompetent Valves & Venous Dilation)
    - § → Further Venous Stasis, Congestion, Oedema, Pain & Thrombosis.
  - o **(Note: Can Also Occur in Oesophagus, Rectum, & Scrotum)**
- **Clinical Features:**
  - o **Symptoms:**
    - § Diffuse Aching, Tightness & Night-Cramping
    - § Persistent Leg Oedema
    - § ↓Wound Healing
  - o **Signs:** Distended, Tortuous Superficial Veins
    - § Ischaemic Skin Changes (Eg: *Stasis Dermatitis*)
    - § Venous Leg Ulcers
    - §
  - o Note: Embolism is RARE since *only Superficial Veins* are affected!!!!
  - o **Complications:**
    - § Recurrent ***Superficial Thrombophlebitis*** (See Below)
    - § Lipodermatosclerois
    - § Haemorrhage
    - § Ulceration
- **Investigation:**
  - o **Trendelenberg Test – (**Pt Supine; Raise leg & occlude Saphenous Vein @ Thigh. Then convert to standing and let go. ***If Veins Fill From The Top = Positive Test*****)**
- **Management:**
  - o **Conservative:** Elevation + Compression Stockings
  - o **Surgical:** **"**Stripping" of Varicose Veins





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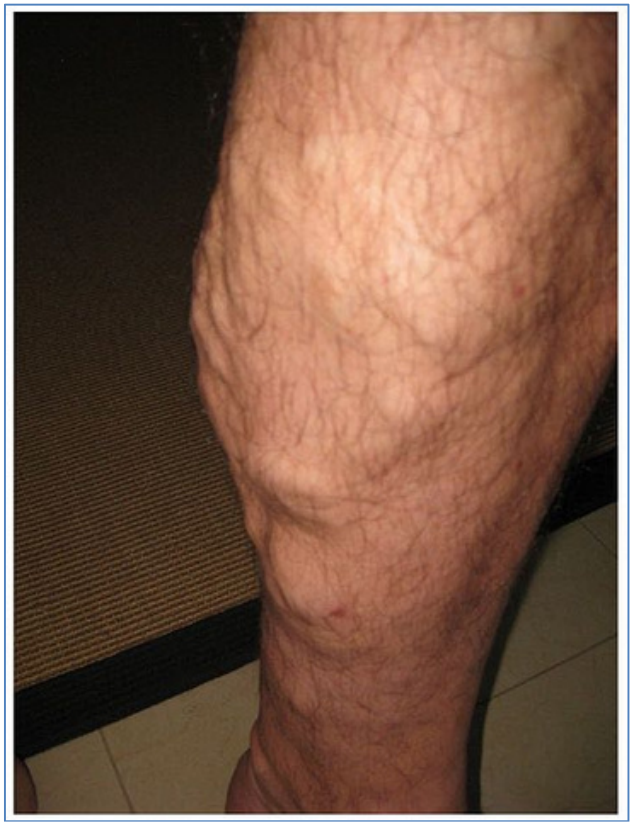

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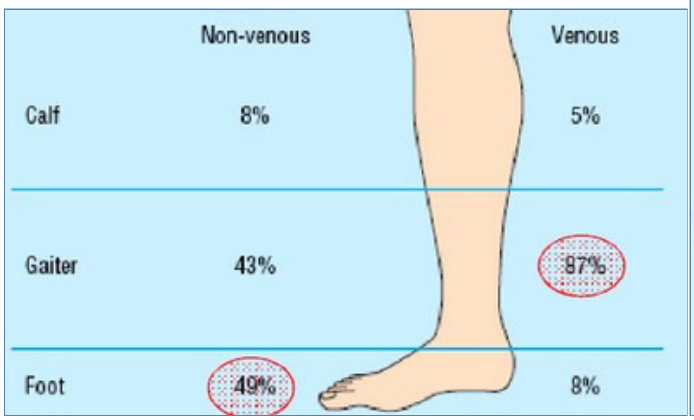
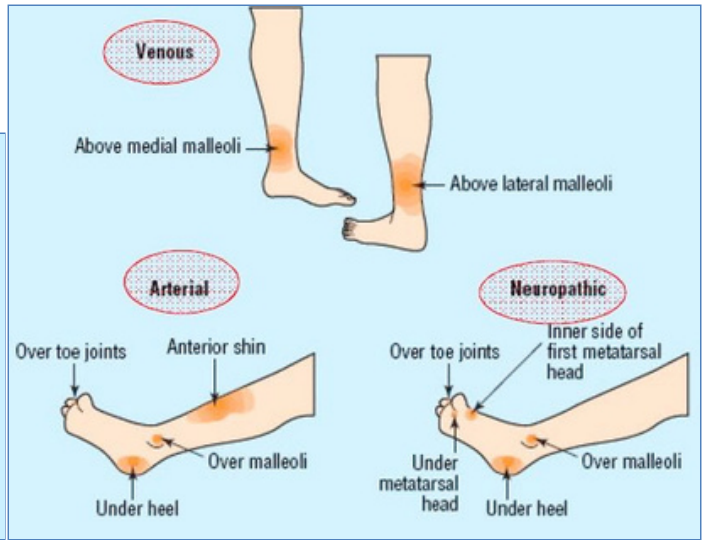
CHRONIC SKIN ULCERS (Lower Limbs *Most Common*):

- **Locations:**
  - **Venous** – "Gaiter" Region
  - **Arterial** – Foot Region, Anterior Shin & Pressure Points
  - **Neuropathic** – Pressure Points



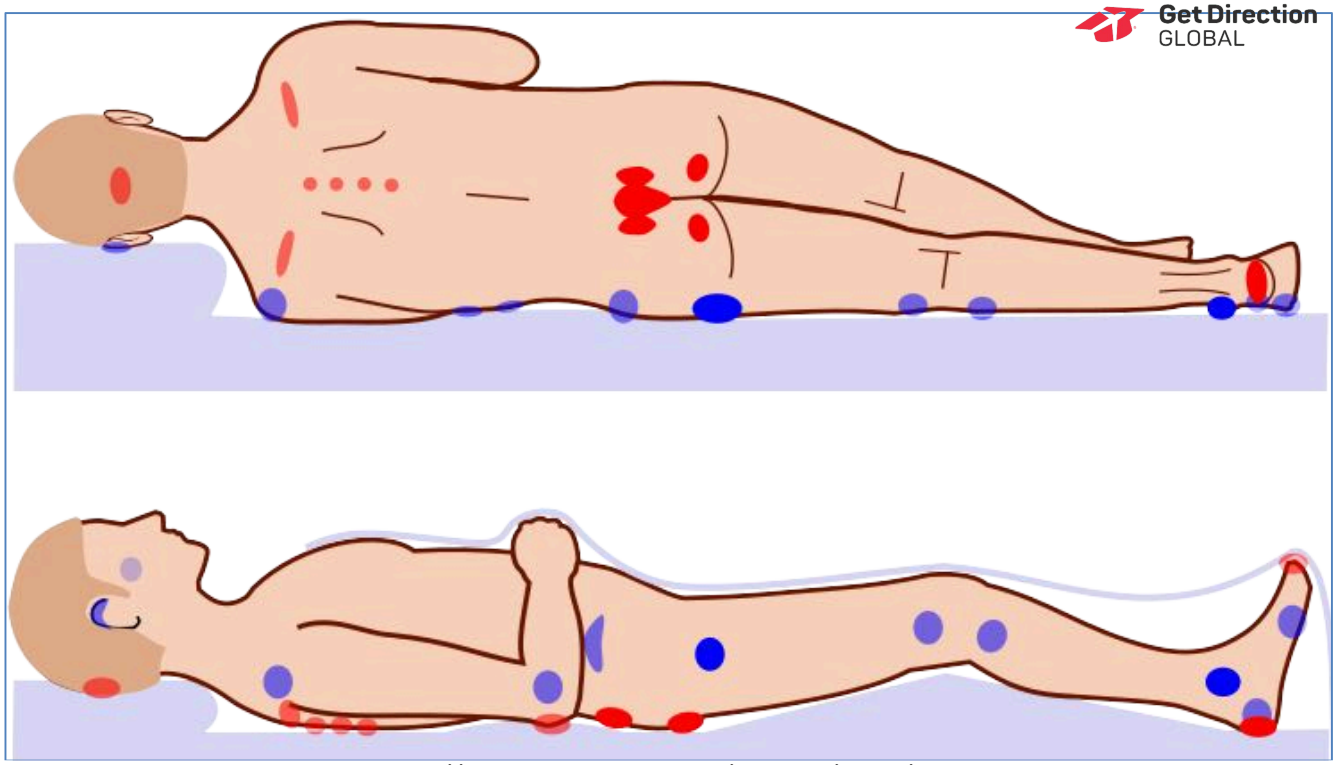


Venous (70% of vascular ulcers)	Arterial	Diabetic
Irregular wound margins	Even wound margins	Irregular wound margins
Superficial	Deep	Superficial
Moderately painful	Extremely painful	Painless
Yellow exudate + granulation tissue	Dry / necrotic base	Necrotic base
Gaiter distribution	Distal locations	Pressure point distribution
Venous stasis discoloration	Thin shiny dry skin	Thin dry skin
Normal distal pulses	Decreased distal pulses	Decreased pulses
No rest pain	Claudication / rest pain	No claudication / rest pain

- **PRESSURE ULCERS:**
  - **Aetiology:**
  - § **Long-Term Pressure** – (*Elderly, frail, bedridden, paraplegia, coma*)
  - **Pathogenesis:**
    - § Long-term skin pressure → Skin Ischaemia → Necrosis → Ulcer

    Clinical Features:
    - § **Location & Appearance:**
      - Bony Prominences – (sacrum, coccyx, heels, occiput, knee, elbow)
      - Initially Non-blanching Erythema → Wet, oozing ulcer.
    - § **Pain:** Often Painful unless Neuropathic/Paraplegia/etc.
      -

  - **Treatment:**
    - § Pressure Redistribution – (Regular Turning, Air Mattress)
    - § Debridement & Dressings
    - § Antibiotics



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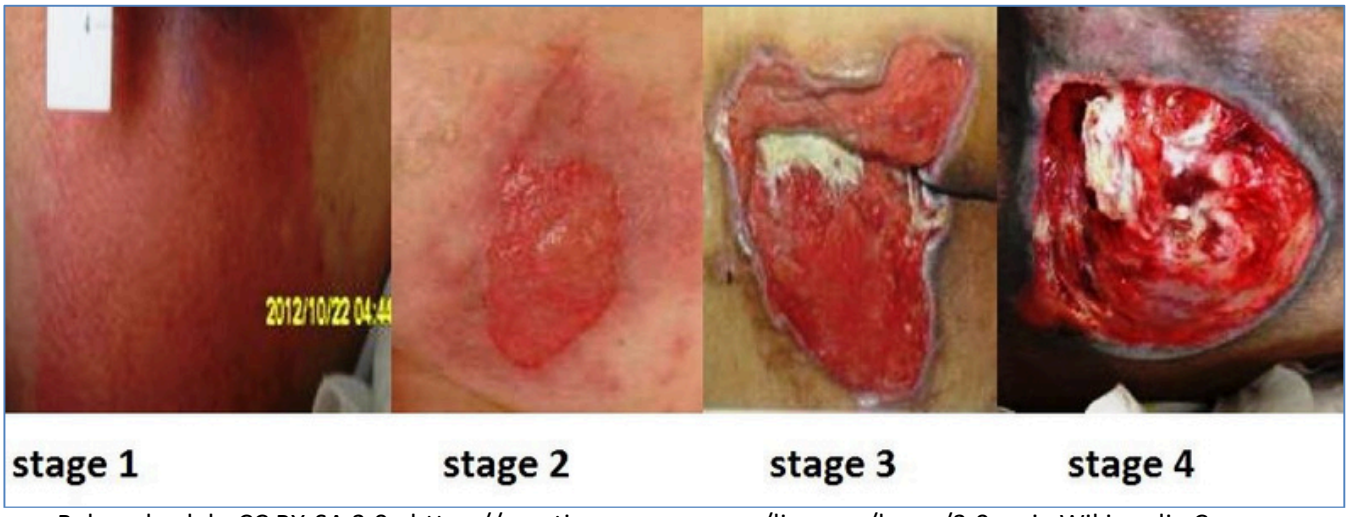

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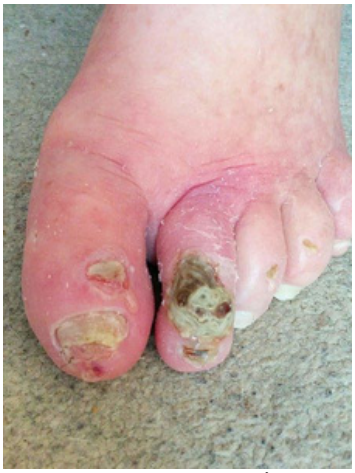
- **ARTERIAL ULCERS:**
  - **Aetiology:**
    - Arterial Insufficiency (PVD) – (Typically due to ***Atherosclerosis***)
    - **Common in Diabetes
  - **Pathogenesis:**
    - Arterial Insufficiency → Tissue Hypoxia/Ischaemia → Skin Necrosis + ↓Wound Healing
    - (Note: Often occurs following Trivial Trauma or Localised Pressure)
  - **Clinical Features:**
    - **Locations:**
      - Anterior Shin
      - Pressure Points of Ankle & Foot (Bony Prominences)
    - **Appearance:**
      - Superficial
      - Well Defined Edges
      - Pale, Non-Granulating Base (Often Necrotic)
      - Does not bleed to touch
      - *No surrounding dermatitis (As opposed to Venous Ulcers)
      - (Cold, Pale feet + Absent Pulses)
    - **Symptoms:**
      - **Severely Painful - ***Relieved by Depression***
      - (+ Claudication)
  - **Management;**
    - **(DO NOT use Compression Bandage!!)**
    - **Control Risk Factors** (Smoking/Diabetes/Hypertension/↑Lipids)
    - **Clean Wound** +/- **Debride**
    - **Reperfusion** (Surgery/Angioplasty)



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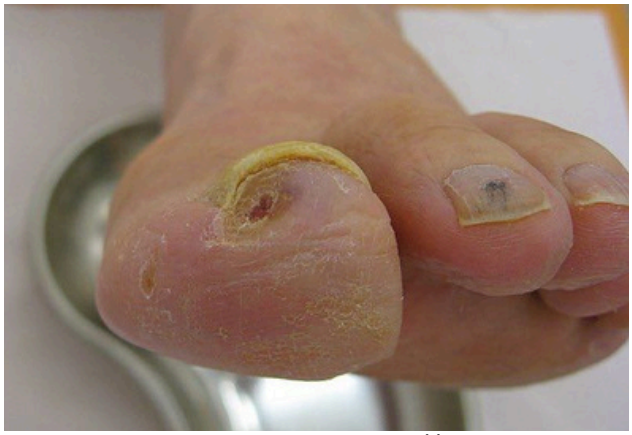

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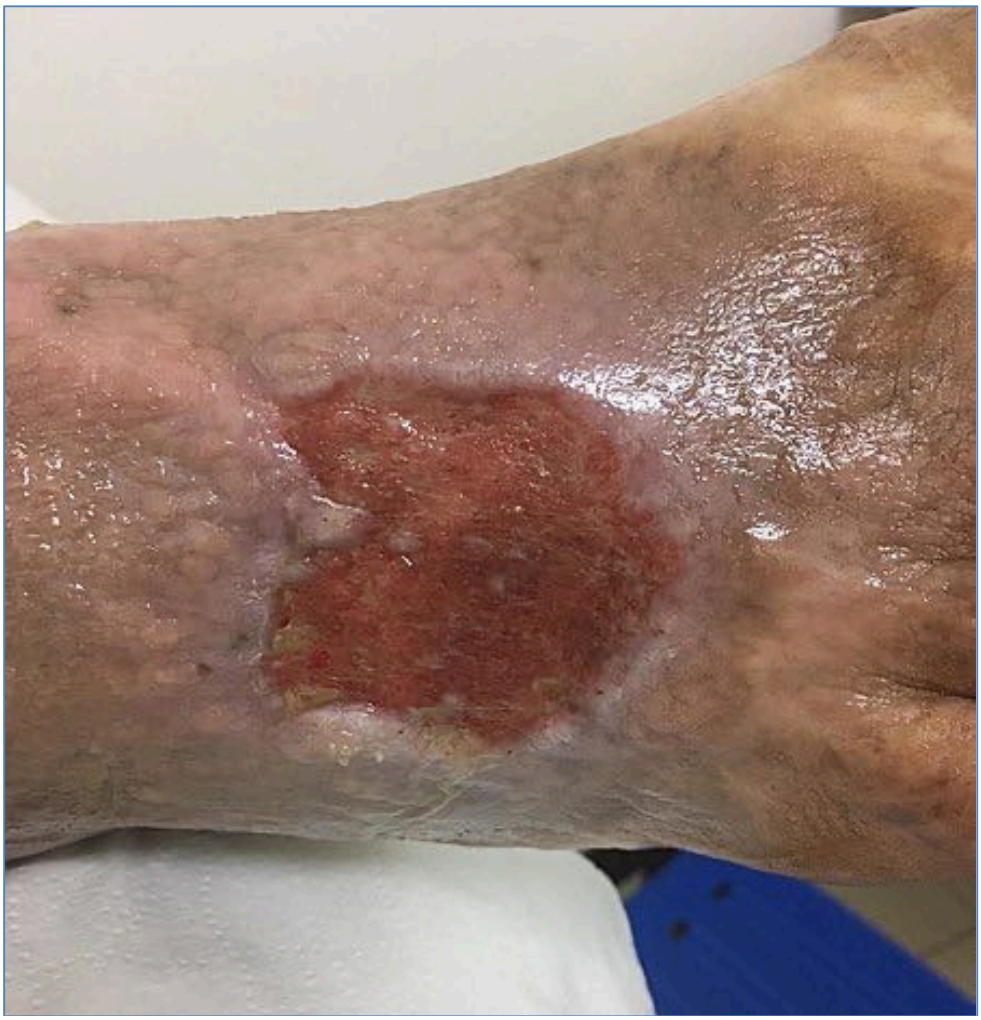
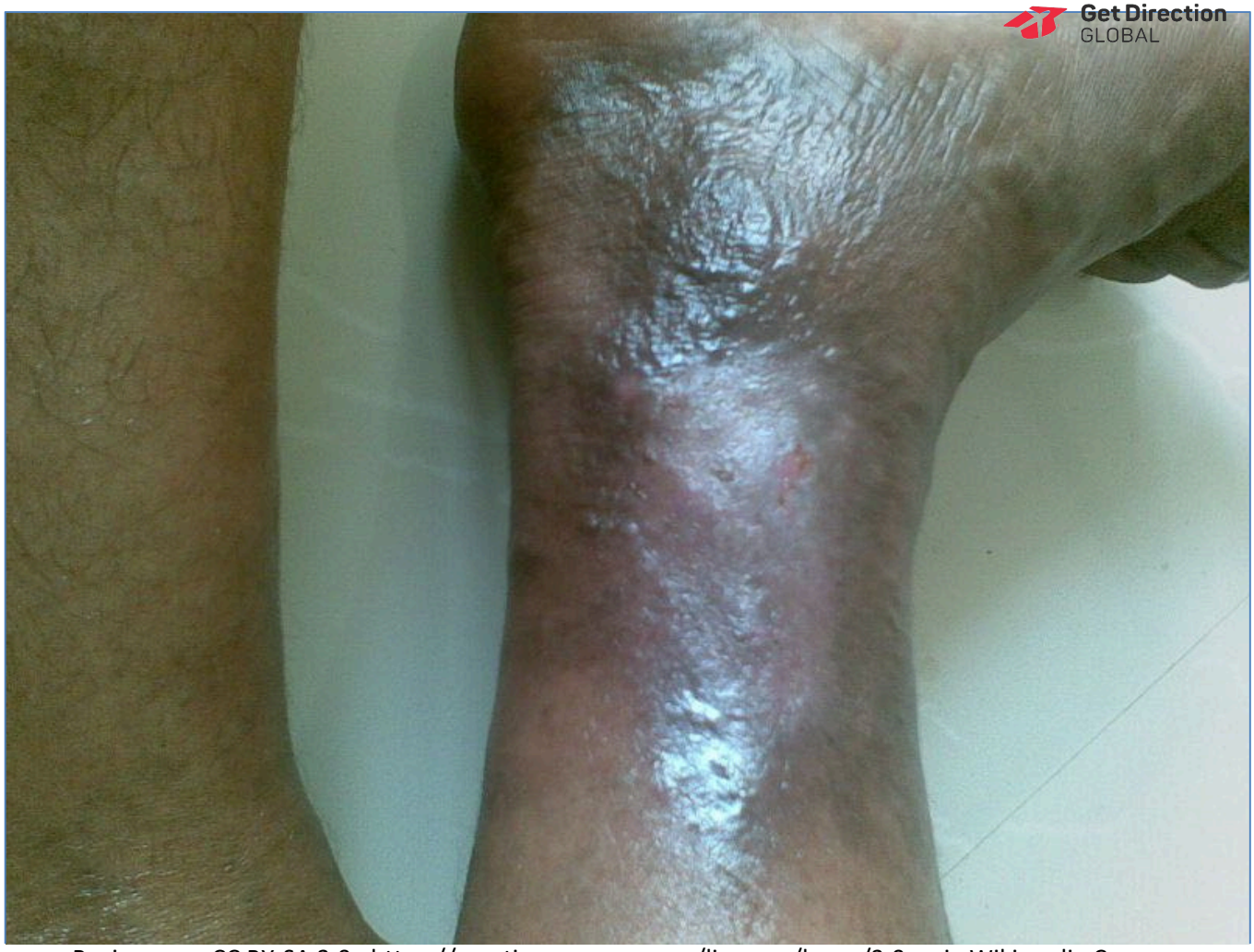
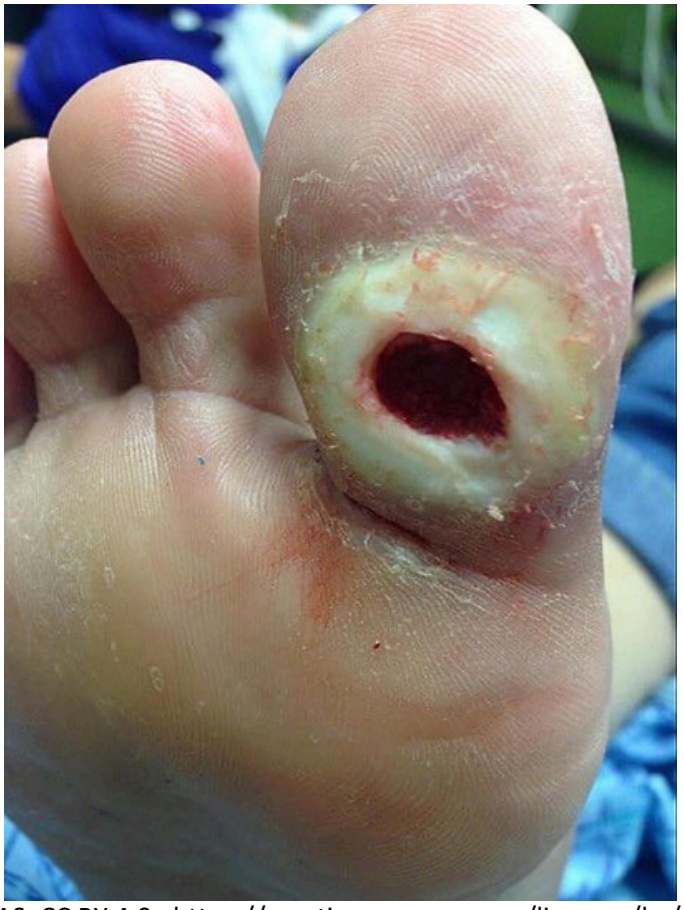
- **VENOUS ULCERS:**
  - **Aetiology:**
    - Venous Valve Insufficiency of the legs → **Sustained Venous Hypertension**
    - (May be Associated with *Varicose Veins*)
  - **Pathogenesis:**
  - Venous Insufficiency of legs → **Venous Hypertension & Stasis** → Ulceration

  Clinical Features:
    - **Location & Appearance:**
      - "Gaiter" Region Above Malleoli
      - Wet, Oozing**
      - Moist, Granulating Base – Bleeds on touch.
      - Surrounding "Stasis Dermatitis" (Eczematous)
      - Oedematous
      - Presence of Varicose veins
    - **Symptoms:**
      - **Only Mild Pain - ***Relieved by Elevation***
      - Dependent Oedema
    - **Treatment:**
      - Compression Bandage
      - Elevation @ Rest
      - Exercise
      - Clean Wound





- **NEUROPATHIC ULCERS:**
  - **Aetiology:**
  - § **Diabetic Neuropathy + Arterial Insufficiency
  - **Pathogenesis:**
    - § **Diabetic Neuropathy**→ Damage/Injury goes Unnoticed → Further Tissue Damage/Necrosis
    - § **(+ Arterial Insufficiency** → Tissue Hypoxia/Ischaemia → Tissue Damage/Necrosis**)**
  - **Clinical Features:**
    - § **Location & Appearance:**
      - Occurs over Pressure Points
      - Deep, "Punched-Out" ulcers
      - ***Often with surrounding Calluses (Hyperkeratosis)
      - Don't Bleed to Touch
    - § **Symptoms:**
      - **Painless
  - **Treatment:**
    - § **(DO NOT apply Compression Bandage!!)**
    - Debride (+/- Amputation)
    - Antibiotics
    - Fastidious Foot Care – (Clean Wound, Podiatrist)
    - Control Other Risk Factors – Esp. Diabetes (+ Smoking/Hypertension/↑Lipids)



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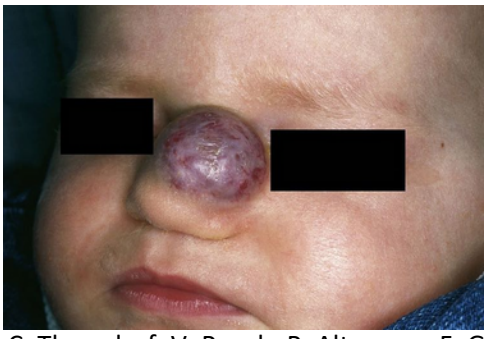
TUMOURS OF VESSELS

- **HAEMANGIOMA:**
  - o **= Closely Packed Aggregates of Sub-Cutaneous Capillaries filled with Blood**.
  - o Congenital & Benign



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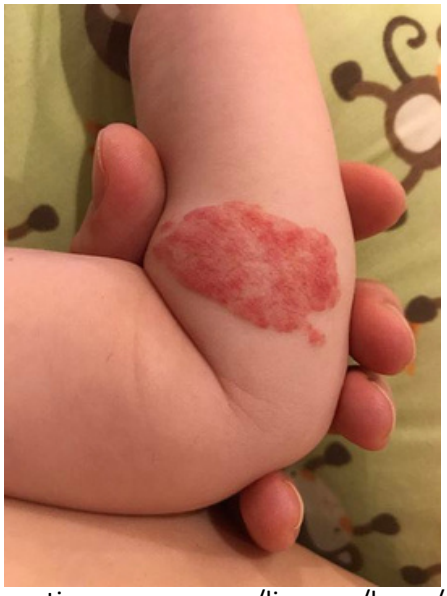

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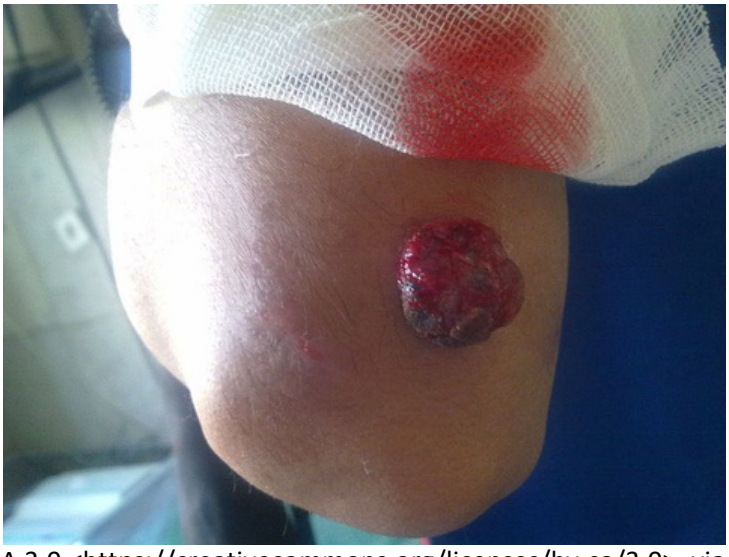
- **PYOGENIC GRANULOMA:**
  - o **= A Granulating Haemangioma**
  - o Rapidly Growing Cutaneous/Mucosal Red Nodule (Bleeds Easily & Often Ulcerated.)
  - o Consist of Capillaries, Granulation Tissue & Bacteria
  - o Often follow Trauma (Inflammatory tissue due to injury)



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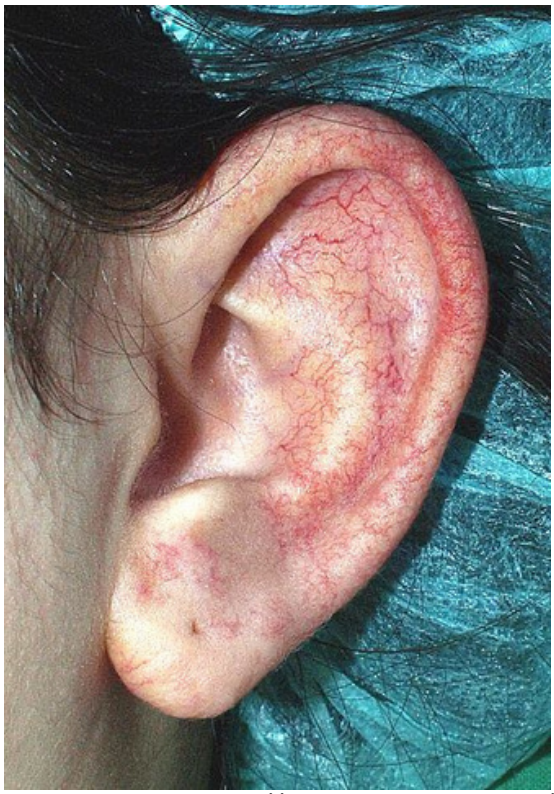
- **TELANGIECTASIA:**
  - o = **A Tiny AV-Malformation**
  - o *Blanching,* Spider-Like, Red Lesions.
  - o Composed of Capillaries, Venules & Arterioles.
  - o (Usually in Skin/Mucous Membranes)



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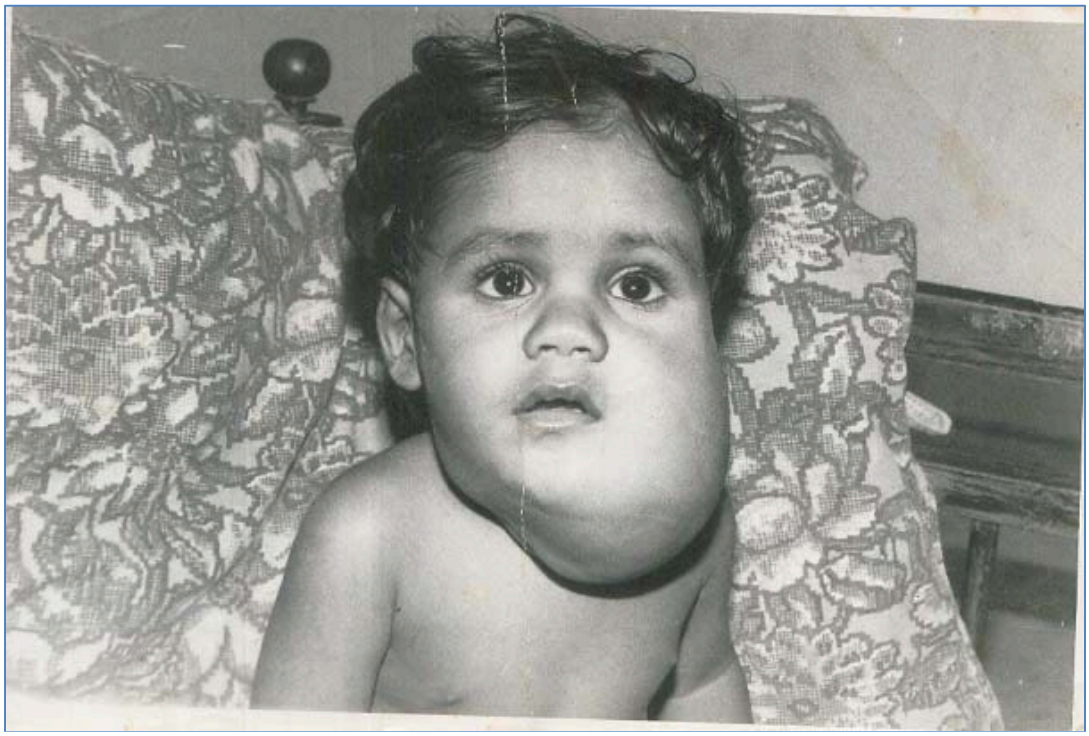
- **LYMPHANGIOMA:**
  - o = **Benign Lymphatic Version of a *Haemangioma* – (= Aggregations of Lymphatic Vessels)**
  - o *May be "Simple" (Capillary) Lymphangioma; or "Cavernous" Lymphangioma ("Cystic Hygroma").*



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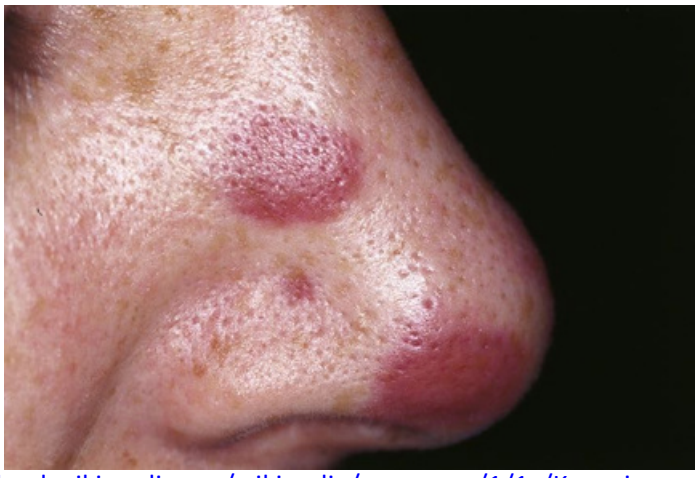
- **KAPOSI SARCOMA ("ANGIOSARCOMA"):**
  - o = **Highly Malignant Endothelial Tumour Caused by HHV-8 Infection**.
  - o Typically in Terminal AIDs Pts (Or other Immunodeficiency)
  - o Early Stages = Asymptomatic → Surgical Excision effective.
  - o Late Stages = Metastatic → Chemotherapy Required.



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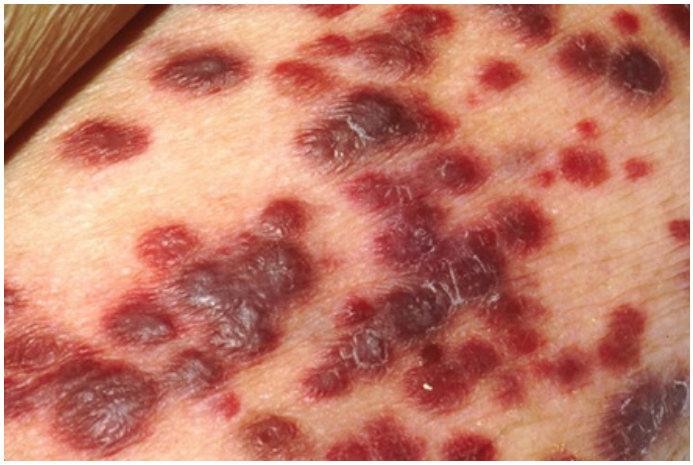

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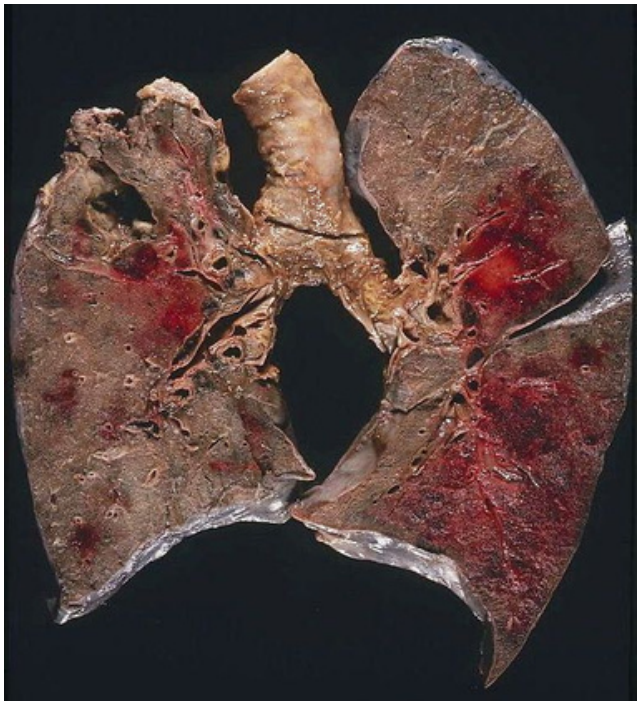

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IMPORTANT VASCULITIDES

Get Direction GLOBAL

Vasculitis (General Vessel Inflammation):

- (There are ≈20 different forms of Vasculitis – We'll focus on the most common ones)
- **2 Aetiologies:**
  - o **Immune…OR…Infective**
  - o **(Note: MUST distinguish between aetiologies since treatments Contradict each other)**
- **Signs/Symptoms:**
  - o **Generals** – *Fever, Malaise, Myalgias & Arthralgias.*
  - o **Specifics** - Depend on Vessels Affected.

VASCULITIS IN LARGE ARTERIES:

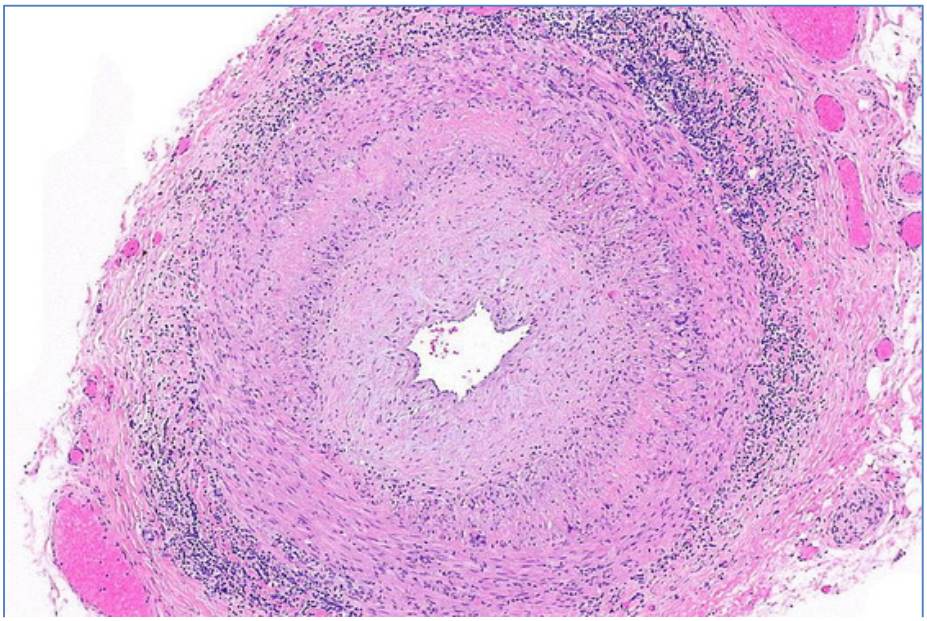
** GIANT CELL (*TEMPORAL*) ARTERITIS:

- **Aetiology:**
- o Chronic, Autoimmune Disease of *TEMPORAL and OPHTHALMIC Arteries*
- **Pathogenesis:**
  - o Autoimmune Inflammation of Temporal & Ophthalmic Arteries

Clinical Features:

- o (Typically in >50yo's)
- o **Temporal Arteritis Triad:**
  - § **1- Headaches**
  - § **2- Jaw Claudication**
  - § **3- Tender Temples**
- o **+ Fever, Fatigue, Weight Loss**
- o **+/- Sudden Painless Blindness (**Transient or Permanent**)**
- o **Sometimes "*Polymyalgia Rheumatica*" – (**Neck, Shoulder & Hip Pain/Stiffness**)**

- **Complications:**
  - o ****RED FLAG – If Untreated, can → BLINDNESS**
  - o **Aortic Arch Syndrome → Aortic Aneurysm +/- Rupture.**
- **Investigation:**
  - o **↑ESR + ↑CRP → **Temporal Artery Biopsy**
    - § **(Note: Biopsy = Definitive Diagnosis)**
  - o **+/- Cranial Angiography**
- **Treatment:**
  - o **High-Dose Prednisone**
  - o (+/- Azathioprine or Methotrexate if severe)



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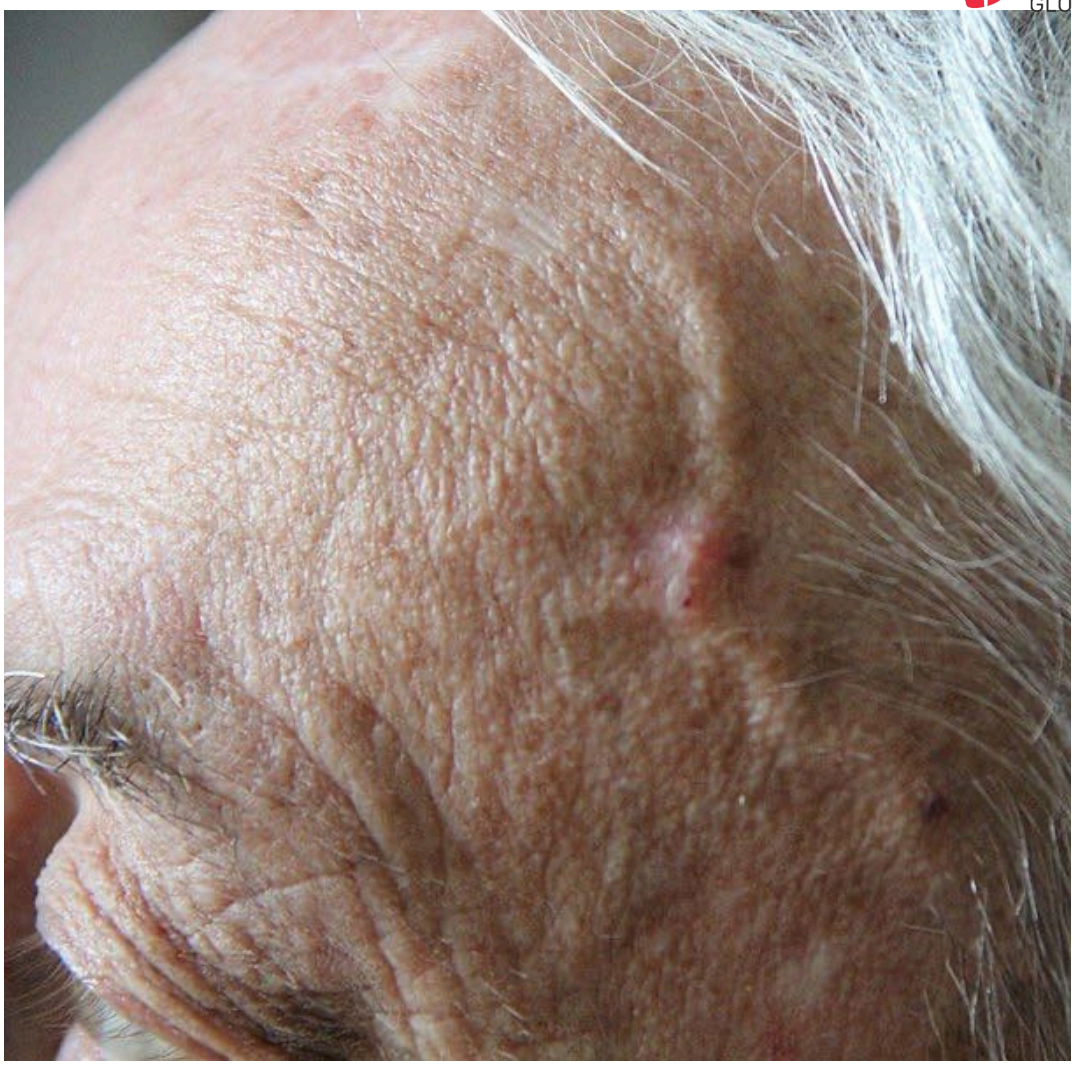

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POLYARTERITIS NODOSA:

Aetiology:

- o *SYSTEMIC* Autoimmune Inflammation of Medium Arteries.

Pathogenesis:

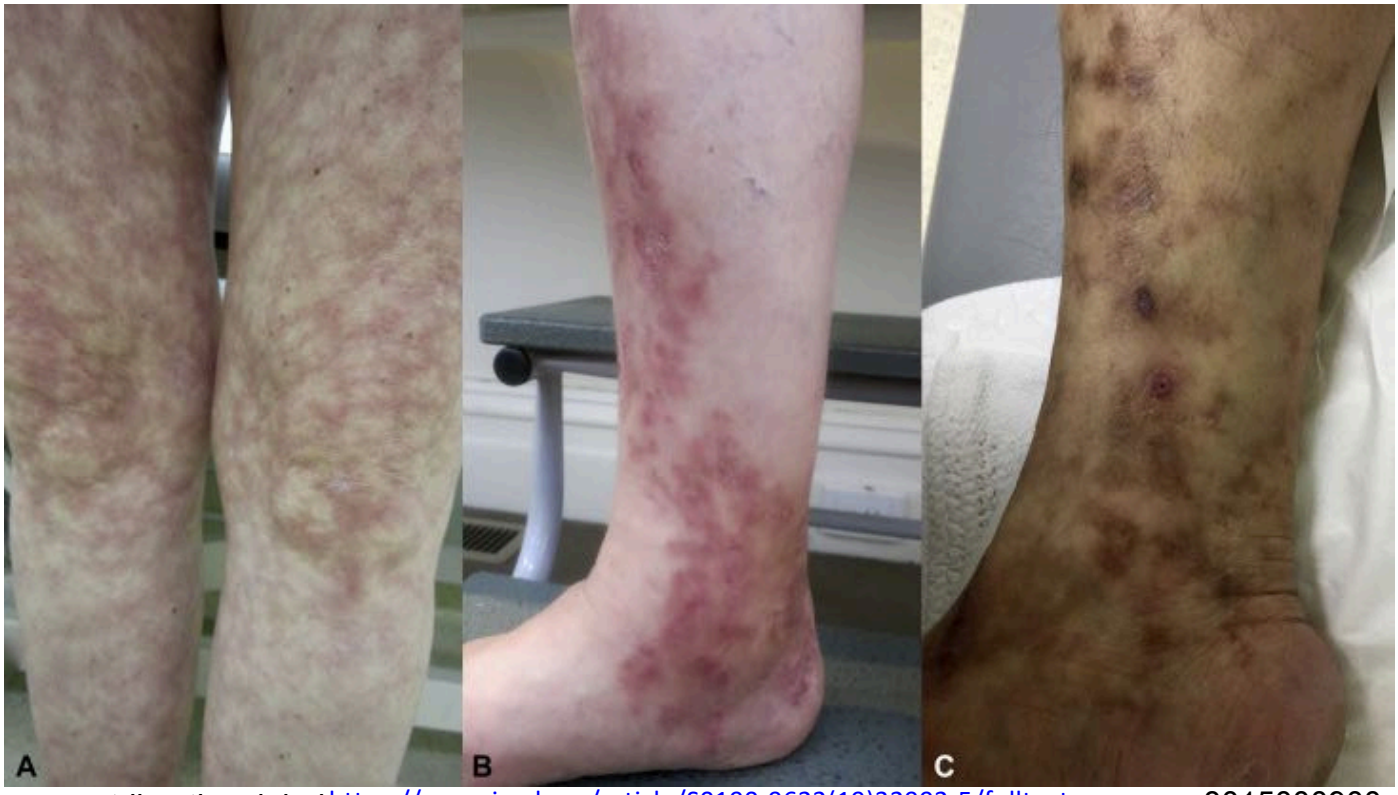
- o Immune Complex Deposition in Arteries *(Particularly Renal Arteries)*
  - § → Necrosis of Vessels → Rupture/Thrombosis/Aneurysms → Infarct/Ischaemia.

- **Clinical Features:**
  - o **General Symptoms:**
    - § **Fever
    - § Rash
    - § Malaise
    - § Weight Loss
  - o **Organs-Specific Symptoms:**
    - § **Skin** – Palpable Purpura, Ulcers
    - § **End-Arteries** – Gangrene, Digital Infarcts
    - § **Muscles** – Myalgia
    - § **Joints** - Arthralgia
    - § **Kidneys** - Hypertension
    - § **Heart** – Angina, MI, CCF
    - § **GIT** – Abdo Pain, Haematemesis, Malena, Ischaemic Gut
    - § **Liver** – Jaundice
    - § **Neuro** – Peripheral Neuropathy, Paraesthesia, Weakness
- **Complications:**
- o Rupture/Thrombosis/Aneurysms → Localised Infarct/Ischaemia

Investigation:

- o **↑ESR + ↑CRP**
- o **Vascular Biopsy**
- o **Or Angiogram**

- **Treatment:**
  - o **Prednisone**
  - o **+ Cyclophosphamide (Chemotherapy)**



https://www.jaad.org/article/S0190-9622(19)32992-5/fulltext

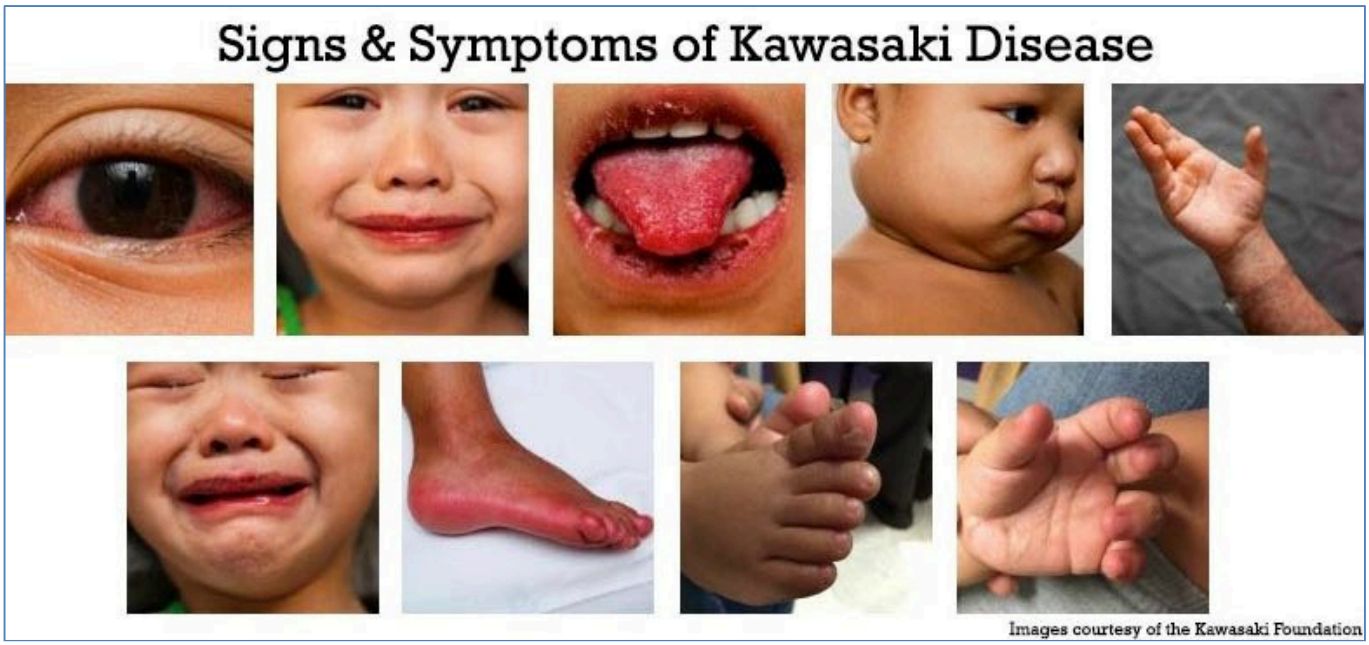
KAWASAKIS DISEASE ("MUCOCUTANEOUS LYMPH NODE SYNDROME"):

- **= An illness that causes inflammation in blood vessels throughout the body**.
- **Aetiology:**
  - o Most common type of vasculitis in children
  - o Usually self-limited
- **Risk Factors:**
- o Infants, children < 5 years old, Asian descent, biologically male

Complications:

- o Coronary artery aneurysm
- o Decreased myocardial contractility → heart failure
- o Myocardial infarction (MI)
- o Arrhythmias
- o Peripheral artery occlusion

- **Signs/Symptoms:**
  - o **First Phase (Lasts for up to 2 weeks) - Mnemonic: "Crash + Burn"**
    - § **C**onjunctivitis: bilateral, nonexudative
    - § Polymorphous **R**ash: desquamating
    - § Cervical lymph**A**denopathy
    - § **S**trawberry tongue: cracked red lips, oral mucositis Hand-foot erythema/ desquamation:
    - § oedema, erythema
      Fever: "**burn**" (Typically lasts for 5 days)
  - o **Second Phase (Usually begins around 2 weeks after fever):**
    - § Peeling skin on hands & feet
    - § Joint pain
    - § Diarrhoea/vomiting
    - § Abdominal pains
- **Diagnosis:**
  - o Clinical Diagnosis – If 4 of the 5 'CRASH' symptoms are present + fever of >5days
  - o ECG – Arrhythmias, Abnormal Q-waves, Prolonged PR & QT Intervals.
  - o CXR – Cardiomegaly
  - o ECHO – Check for coronary artery aneurysms, pericardial effusions, & reduced contractility.
  - o Bloods - ↑ CRP, ESR, platelet count (reactive thrombocytosis)
- **Treatment:**
  - o Aspirin
  - o IVIG (Intravenous Immunoglobulin)



https://kdfoundation.org.au/

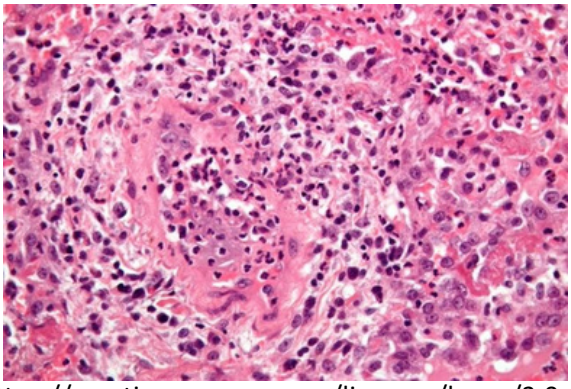
WEGENER'S GRANULOMATOSIS:

- **= Small-vessel vasculitis involving nasopharynx, lungs, kidneys**
- **Aetiology:**
- o Autoimmune (Probably Hypersensitivity to Inhaled Agents)

Pathogenesis:

- o Autoimmune Hypersensitivity Reaction to Inhaled Agent → Necrotizing Lung Granulomas (~TB)
  - § (Also Renal → ***Glomerulonephritis***).
- **Morphology:**
  - o **Granulomatous Inflammation in Lungs & URT**
    - § URT Mucosal Granulomatous Lesions
    - § Granulomas (which may cavitate) in the Lungs
  - o **Necrotizing Vasculitis around Small Vessels (Particularly Renal/Glomerular)**.
    - § Focal (early) or Diffuse (late) Glomerular Necrosis → ***Glomerulonephritis***
- **Clinical Features:**
  - o **Systemic:**
    - § Fever, Malaise, Weakness, Myalgia, Rash.
  - o **Respiratory:**
    - § **Initially – (Flu-like Illness):**
      - Fever
      - Cough
      - Rhinorrhoea
      - Otitis Media
    - § **Later – (Similar Features to TB):**
      - Haemoptysis
      - Chronic Pneumonitis
      - Bilateral Cavitary Granulomas in Lungs
      - Chronic Sinusitis
  - o **Renal:**
    - § **Glomerulonephritis** – (Nephrotic +/- Nephritic Syndrome)
- **Investigations:**
  - o **American College of Rheumatology Criteria – (>2 of):**
    - § URTI Inflammation – (Nasal/Oral)
    - § CXR – (Nodules/Cavitations)
    - § Urinalysis – (Protein/Casts)
    - § Biopsy – (Granulomatous Inflammation)
  - o **+ c-ANCA in 90% of cases**
  - o **↑ESR & ↑CRP**
- **Treatment:**
  - o **Prednisone (+/- *Cyclophosphamide/Rituximab*)**
  - o **+/- High-Dose *Methotrexate***
  - o (Note: 80% 1yr Mortality if Not Treated)

G ranulom atous inflam m ation around sm all vessels w ith epithelioid cells and giant cells.



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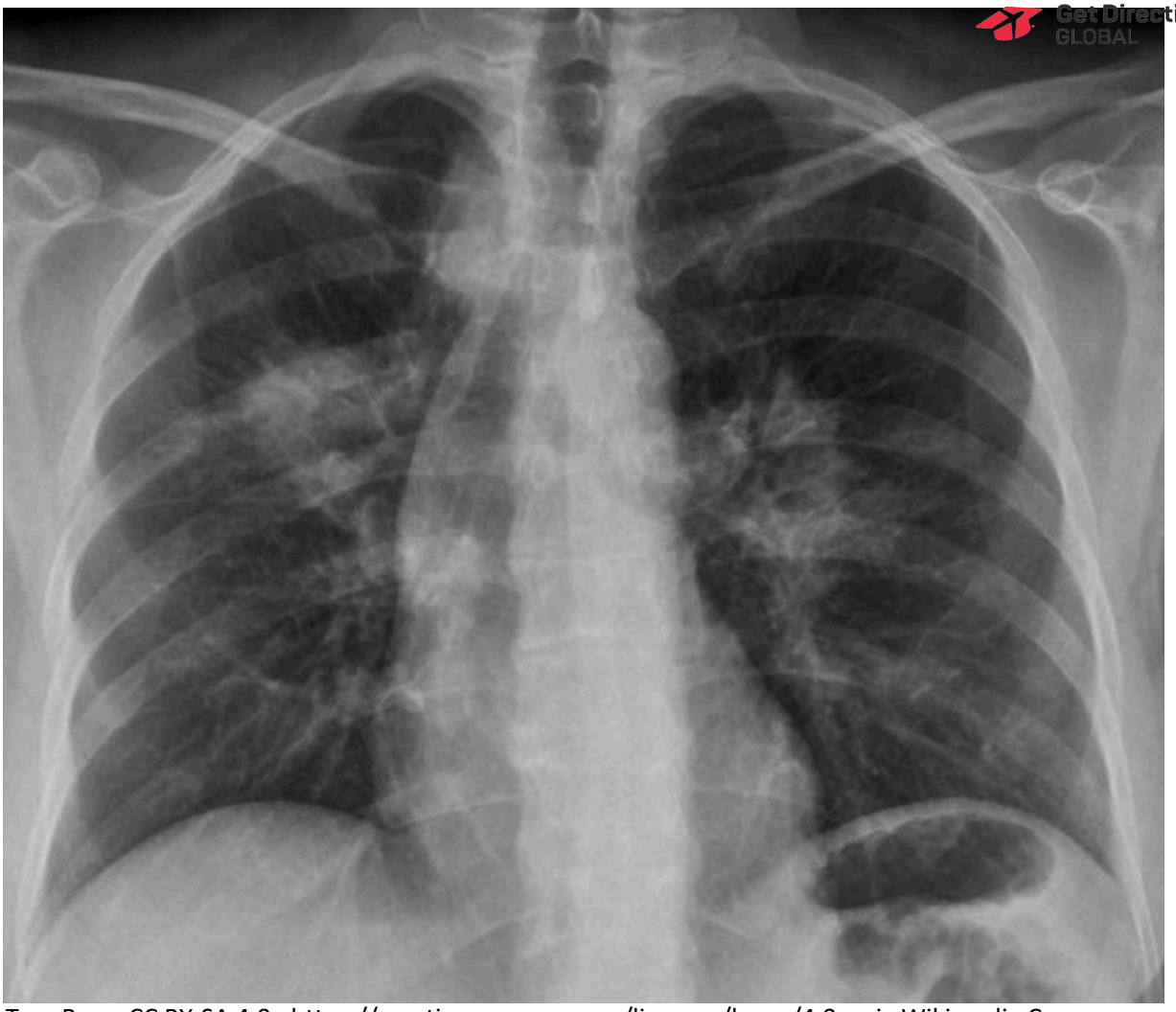

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CHURG-STRAUSS SYNDROME (AKA: *Eosinophilic Granulomatosis with Polyangiitis*; EGPA):

- **= Small, medium vessel granulomatous vasculitis**
- **Aetiology:**
- o Unknown

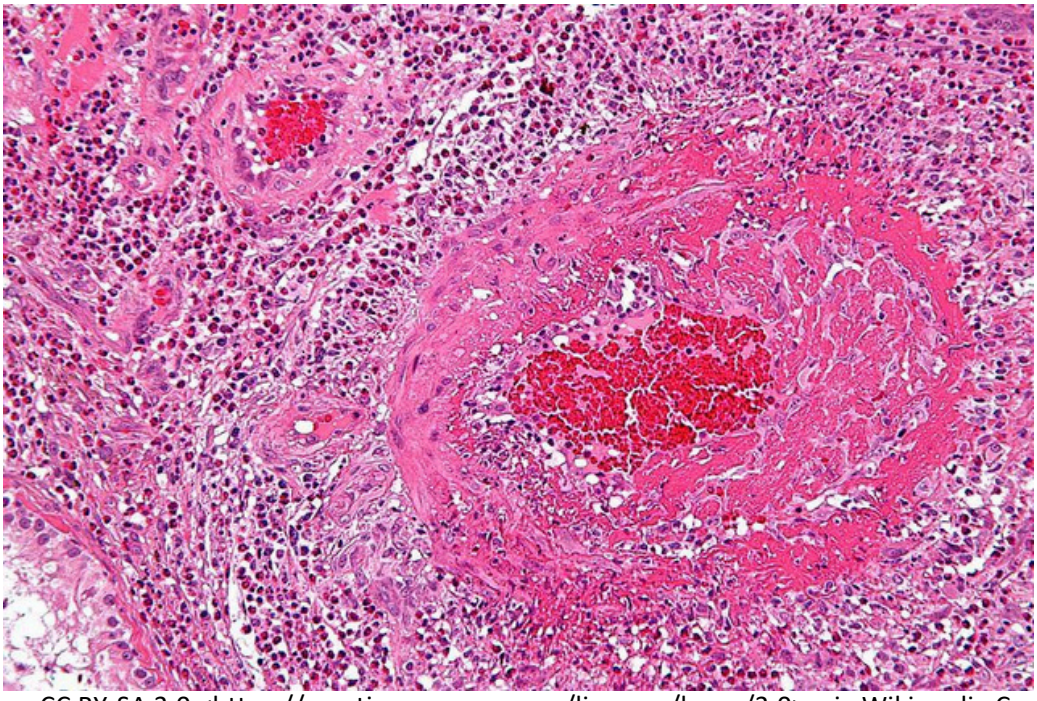
Pathogenesis:

o Granulomatous Inflammation of Small/Medium-Sized Vessels.

o **involving many organ systems**

- § **cardiac,**
- § **gastrointestinal,**
- § **respiratory,**
- § **skin,**
- § **renal,**
- § **neurologic**

- **Clinical Features:**
  - o **Age 30–50**
  - o **Churg-Strauss Triad:**
    - § Systemic Vasculitis
    - § Asthma
    - § Allergic Rhinitis
  - o **Others – (**Angina, Myocarditis, Neuropathy, Subcutaneous nodules, Palpable Purpura**)**
- **Investigation:**
  - o **+ P-ANCA**
  - o **+ MPO-ANCA Antibodies**
  - o **↑ESR & CRP**
  - o **FBC – (**Eosinophilia**)**
  - o **CXR -** Transient, patchy, symmetrical opacities, often in hilar/peripheral distribution
  - o **Pulmonary Function Tests –** Obstructive Picture consistent with asthma.
- **Management:**
  - o ***Prednisone +/- Cyclophosphamide***
  - o **Then *Methotrexate***
- **Prognosis:**
  - o **Poor -** five year survival, 25% without treatment; 50% with treatment



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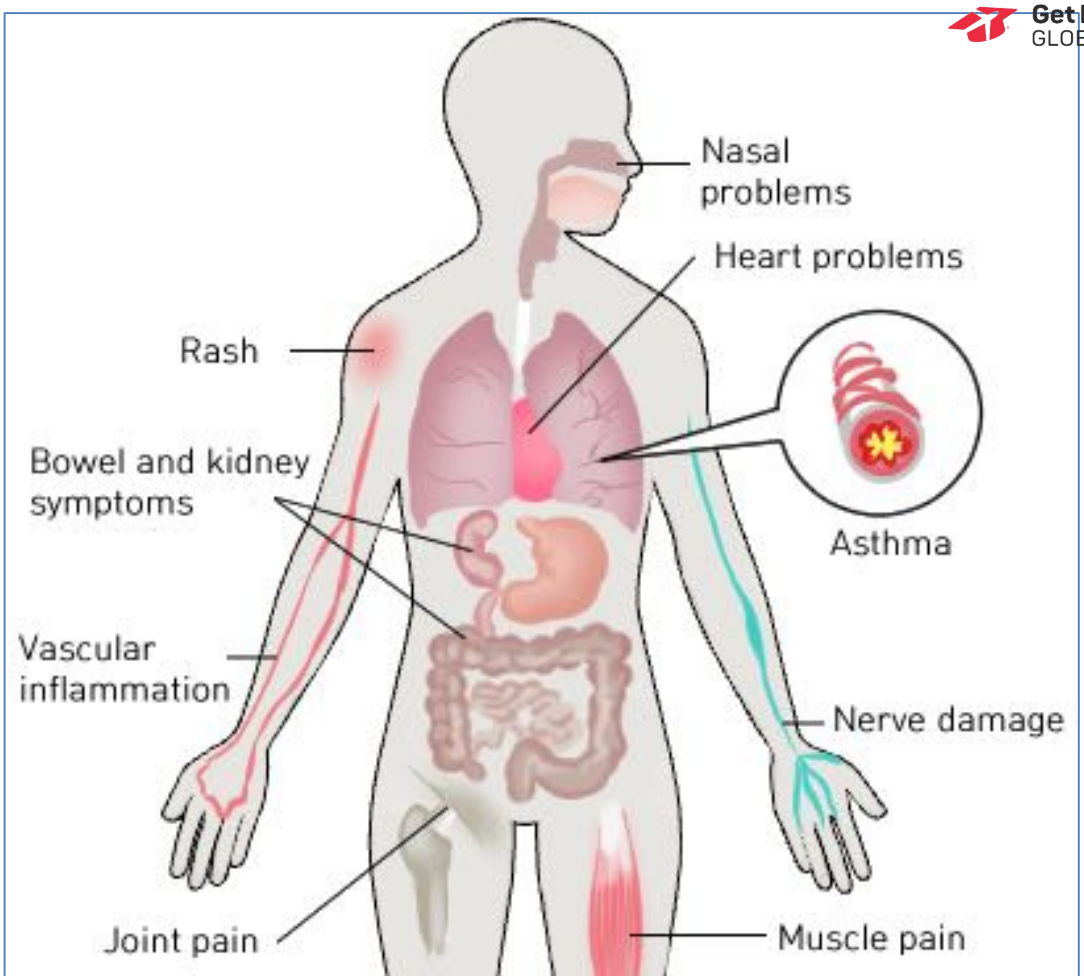


Brigham & Women's Hospital; https://www.brighamandwomens.org/

BUERGER'S DISEASE ("THROMBOANGIITIS OBLITERANS"):

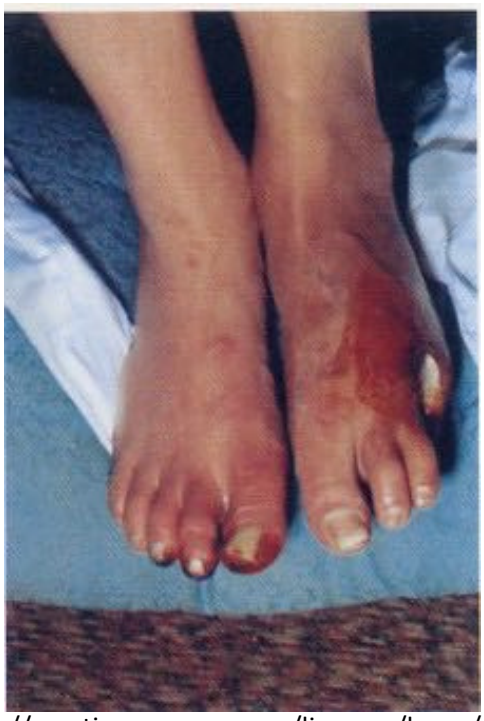
- **Aetiology:**
  - o **Cigarette Smoking → Direct Endothelial Toxicity

Pathogenesis:

- o inflammatory disease affecting small-, medium-sized veins, arteries of extremities → inflammatory occlusive throm bus → distal extrem ity ischem ia, digit ulcers/ gangrene → autoam putation
- o Strongly associated with Smoking.

- **Clinical Features:**
  - o ***Occurs in Chronic HEAVY Smokers
  - o o **Digital Infarcts – Gangrene & Ulceration**
  - o **Distal Limb Ischaemia** (Claudication, Arterial Ulcers, Gangrene) o **Subcutaneous nodules** o Paraesthesias of extremities o Raynaud phenomenon

- **Diagnosis:**
  - o **Angiogram** can be helpful to rule out atherosclerosis
  - o **But Biopsy is definitive**.
    - § acute-phase lesions show highly cellular, inflammatory thrombus with minimal inflammation of blood vessel
- **Treatment:**
  - o **Smoking Cessation** (In early stages) → Dramatic Relief.



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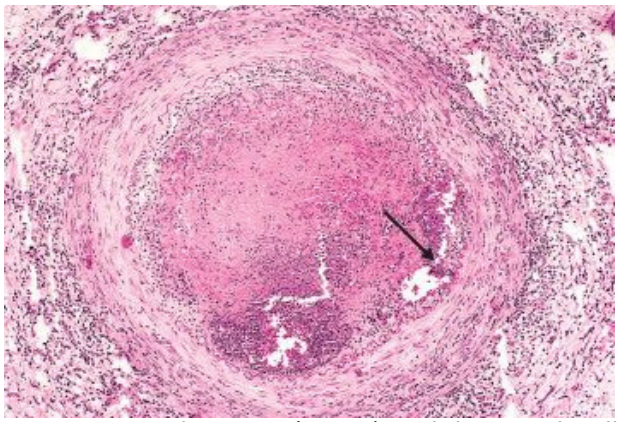
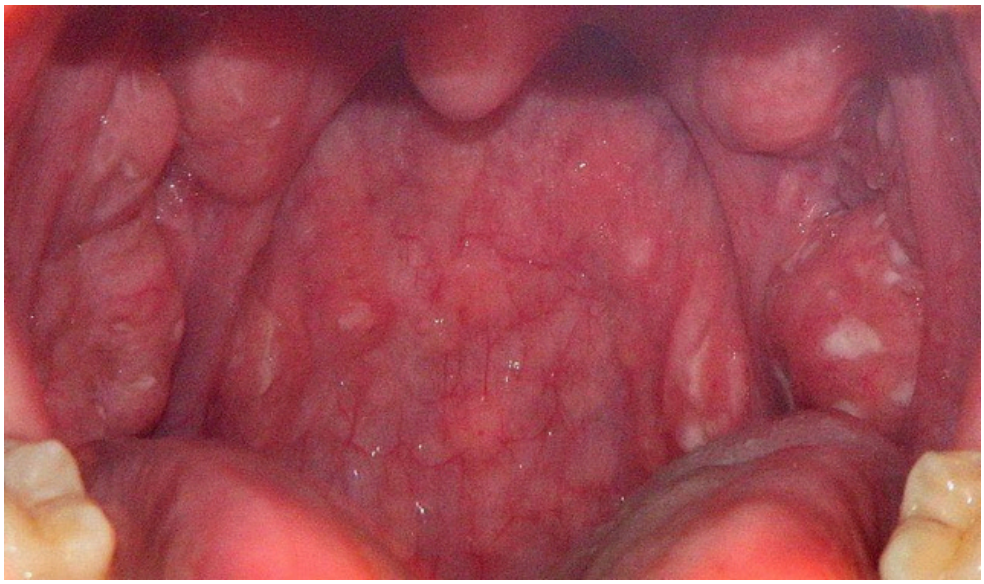


(Lumen is occluded by thrombus containing abscesses *(arrow)*, and the vessel wall is infiltrated with leukocytes.)

- **Background:**
  - **Rheumatic Fever (RF)** = Delayed Autoimmune Complication of a GAβH Strep Tonsillo/Pharyngitis.
    - → **Acute Rheumatic Fever / Carditis** (*Acute Phase* of Rheumatic Fever)
    - → **Chronic Rheumatic Heart Disease (RHD)** (Typically →Mitral Stenosis)
  - **(Note: Rheumatic Fever (RF) & Rheumatoid Arthritis (RA) are 2 different diseases)**
    - *RF - Licks joints but bites heart! (Temporary Arthritis, but Permanent Valvular Damage)*
    - *RA – Licks heart but bites joints! (Mild Myocarditis, but permanent Severe Arthritis)*
- **Aetiology – 3 Factors:**
  - **1- Environmental factor** – **Group-A-Beta-Haemolytic Strep (*Pyogenes*) Pharyngitis**
  - **2- Genetic Susceptibility (3% of Population)** – HLA DR-2 & DR-3 Positive
  - **3- Autoimmunity** – Autoantibodies (Antigenic Mimickery)
    - GABH-Strep → Production of Anti-M-Protein Ab's → Cross React with Cardiac Conn. Tissue.
- **Pathogenesis → M itral Stenosis:**
  - **1- GABH Strep Pharyngitis** (In *HLA-DR2/3-Positive* Person)
  - **2- 2wks Later, Immune Response to GABH-Strep→ Rheumatic Fever → Carditis**
    - (Note: 2wk Delay due to Lymphocyte Activation)
  - **3- Subsequent GABH-Strep Infections → Secondary (STRONGER) Immune Responses:**
    - **Recurrent Rh-Fever** → Cumulative Valve Damage (Fibrosis) → **Rheumatic Heart Disease**



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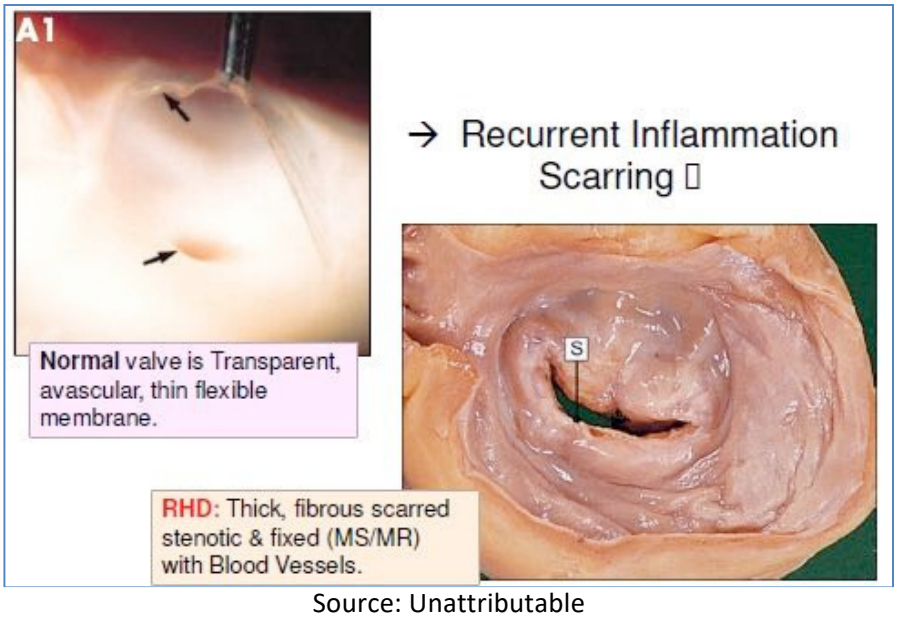
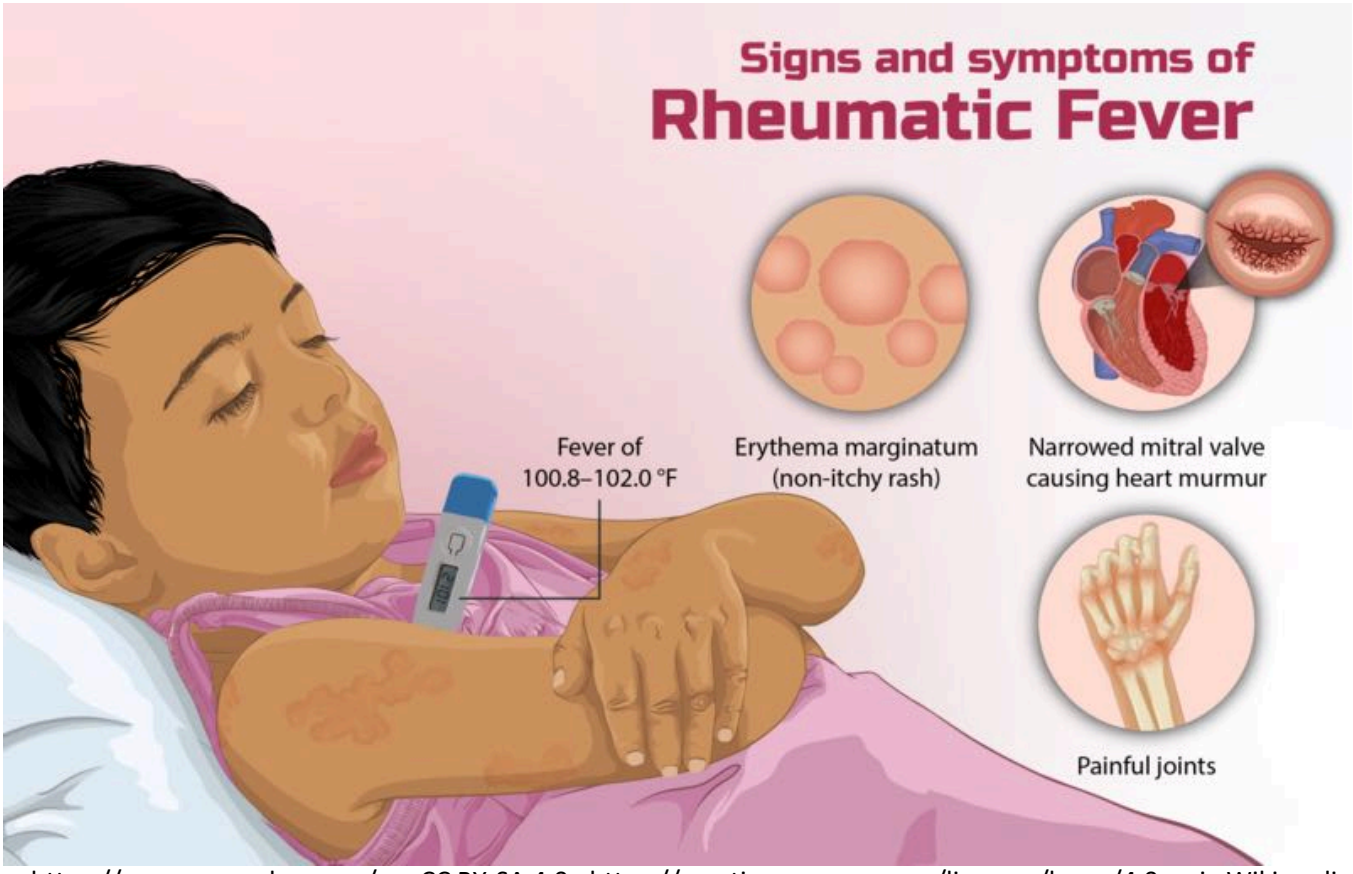


Source: Unattributable

- **Clinical Features & Diagnosis:**
  - **Acute Rheumatic Fever:**
    - **Jones Criteria Rules - Must Have:**
      - 1) Evidence of *Previous* GABH-Strep (Strep. Pyogenes) Infection
      - 2) (2x Major Criteria) OR (1 Major + 2 Minor)
    - **1- (Evidence of Previous Strep Infection):**
      - ↑Anti-Streptolysin-O Titre
      - ↑Anti-DNaseB Antibodies
      - Positive **Throat Swab Culture**
    - **2a. (Major Criteria)**
      - **J** **Joints** (Migratory Polyarthritis – Not necessarily arthralgia)
      - Y **Carditis** (Incl: Pericarditis – Friction Rub, Quiet Heart Sounds, Tachy)
      - **N** **Nodules** (Subcutaneous, painless, on extensor surfaces)
      - **E** **Erythema Marginatum** (Non-Pruritic, Tinea-like Rings on Trunk & Limbs)
      - **S** **Sydenham's Chorea** (Rapid, Involuntary Movements)
    - **2b. (Minor Criteria)**
      - (Fever)
      - (Arthralgia)
      - (Elevated ESR)
      - (Prolonged PR-Segment)



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- **Chronic Rheumatic Heart Disease:**
  - **Cardiac Murmurs (Typically L-Heart):**
    - Mitral Stenosis (+/- Regurg)
    - Aortic Stenosis (+/- Regurg)
  - **Mitral Stenosis:**
    - **→"Mitral Facies" –** (Malar/Butterfly Rash over Cheeks & Nose)
    - **→ Mid-Diastolic Rumbling Murmur –** (Loudest @ Apex on Expiration & → Axilla).
    - **→ Pulmonary Congestion & CCF –** (RV-Hypertrophy, Exertional Dyspnoea)
  - **Atrial Fibrillation** (From Atrial Stretch due to Mitral Stenosis)
  - **↑Risk of Infective Endocarditis**

- **Management:**
  - **(Primary Prevention – Rx of Strep Pharyngitis):**
    - **10days PO Penicillin-V (Or Amoxicillin or Cephalexin)**
  - **Secondary Prevention:**
    - **Admit on Suspicion:**
      - Based on Jones Criteria
    - **Treating *Acute Rheumatic Fever*:**
      - **GABH Strep Eradication –** (Single dose IM ***Benz-Pen-G***)
      - **Joint pain (Arthralgia) –** (*NSAIDs* or *Codeine*).
      - **Chorea –** (*Carbamazepine* or *Valproate* if Necessary)
      - **Carditis/Heart Failure –** (*ACEi* + *B-Blocker* + *Diuretics*)
    - **Treating to *Prevent Recurrent Attacks*:**
      - **Continuous AB-Prophylaxis for *Minimum 10 years after last ARF Episode*.**
        - ***First-line:** Monthly ***IM Ben-Pen-G***
        - **Second-line:** BD **Oral Pen-V**
        - ***(If Penicillin Allergy:** BD **Oral Erythromycin**).
  - **(Tertiary Prevention):**
    - **Cardiac Surgery -** Mitral Valve Replacement

- ***Question - What is the difference between Rheumatic Fever (RF) & Rheumatic Heart Disease (RHD)?***
  - **(Note: Neither RF or RHD is an Infection, and *Both* can affect the Heart.)**
    - **(The Distinction is whether it is *Reversible* (RF) or *Irreversible* (RHD).)**
  - **Rheumatic Fever:**
    - An acute, *Post*-GAS-Infection Inflammatory Disease.
    - Occurs a few weeks *After* a GAS Infection.
    - If not treated aggressively →Acute Rheumatic Carditis → Valvular Deformities.
  - **Rheumatic Heart Disease:**
    - The Chronic Stage which causes Irreversible Myocardial Damage & Heart Valve Damage.

VALVULAR HEART DISEASE:

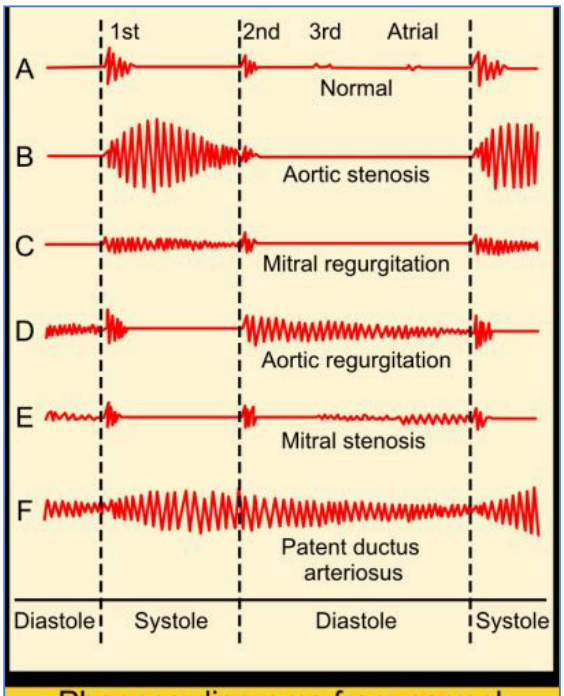
- **4 Most Common Murmurs & Their Causes:**

Valve Lesion	Aetiology/Pathological Cause
Mitral Stenosis	**Rheumatic Fever (Post Inflamm. Scarring)
Mitral Regurgitation	Mitral Prolapse ("Myxomatous Degeneration") Rheumatic Fever (Post Inflamm. Scarring) Infective Endocarditis MI (Papillary Muscle Fibrosis/Dysfunction) Rupture of Papillary Muscles/Chordae Tendineae Dilated Cardiomyopathy (Dilation of Valve Annulus) Congenital (Degeneration of Cusps)
Aortic Stenosis	Age-Related Calcification Rheumatic Fever (Post Inflamm. Scarring)
Aortic Regurgitation	Age-Related Dilation of the Ascending Aorta HT-Related Dilation of the Ascending Aorta Rheumatic Fever (Post Inflamm. Scarring) Infective Endocarditis M arfan's Syndrom e Syphilitic Aortitis Rheumatoid Arthritis Ankylosing Spondylitis

- (Red = Most Common)

- **Other Less Common Murmurs:**

	Cause	Diastolic/Systolic?
Pulmonary Stenosis	Congenital Heart Defect Rheumatic Heart Disease	Systolic
Pulmonary Regurgitation	Pulmonary Hypertension	Diastolic
Tricuspid Stenosis	Rheumatic Fever	Diastolic
Tricuspid Regurgitation	R-Ventricular Dilation (Eg: R-V Infarction)	Systolic



Phonocardiograms from normal and abnormal heart sounds

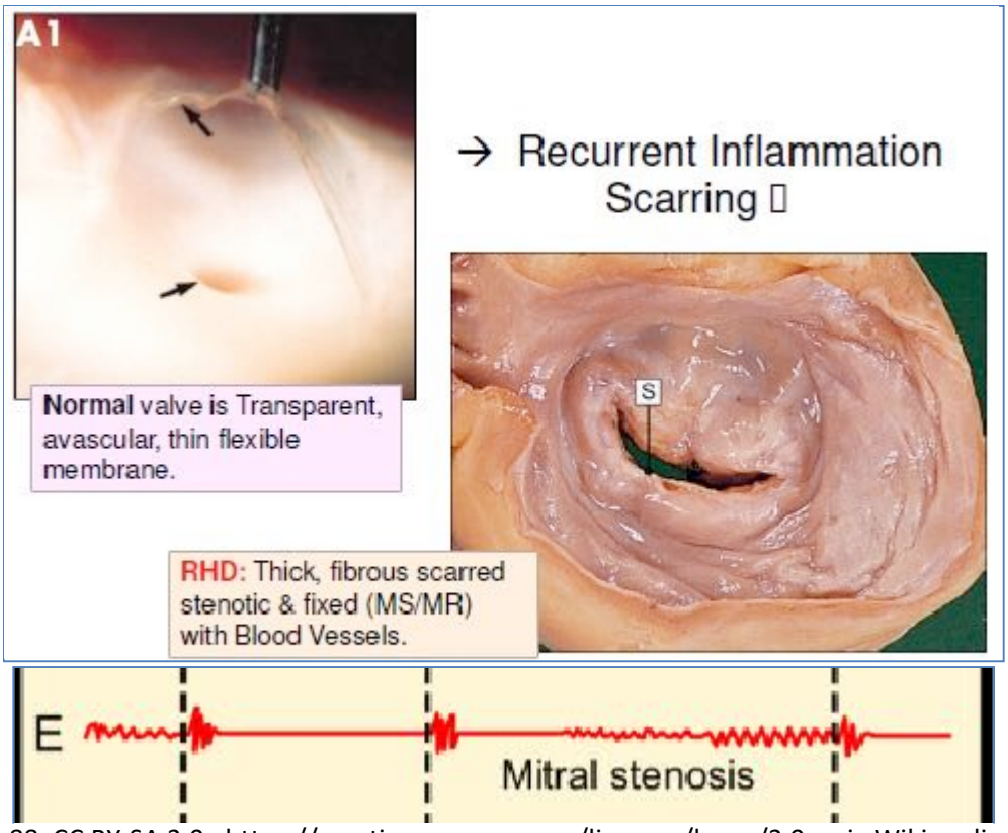
MITRAL STENOSIS:

- **Aetiology:**
  - o 99% Rheumatic Heart Disease
- **Pathogenesis:**
  - o Recurrent Acute Rheumatic Fever → Autoimmune Mitral Valve Fibrosis → Stenosis

Clinical Features

- o **Symptoms:**
  - § **CCF** – (Exertional Dyspnoea/Orthopnoea/PND/Wet cough (Pulmonary Oedema))
- o **Signs:** **Low-Volume Pulse**
  - § **Mid-Diastolic Rumbling Murmur** – (Loudest @ Apex on Expiration & → Axilla).
  - § **"Mitral Facies" –** (Malar/Butterfly Rash over Cheeks & Nose)
  - § **Pulmonary Congestion & CCF** – (RV-Hypertrophy, Exertional Dyspnoea)
  - § **If Cor-Pulmonale (RV-Failure)** → (↑JVP, Pulsatile Liver, Ascites, Peripheral Oedema)
  - §

- **Investigations:**
  - o **ECHO –** (Diagnostic)
  - o **ECG –** (May have A.Fib, LA-Hypertrophy, RVH)
  - o **CXR –** (LA-Hypertrophy, Pulmonary Congestion)
- **Management:**
  - o **Medical –** Treat A.Fib, Warfarin, CCF Triples (ACEi + B-Blocker + Diuretics)
  - o **Surgical – Mitral Valvuloplasty (Repair) or Replacement**



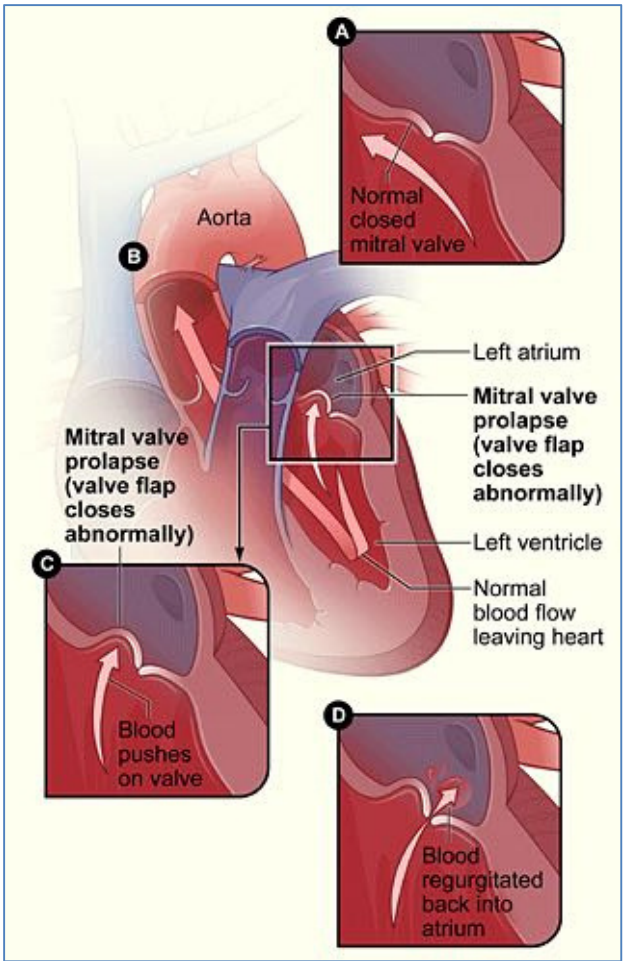
MITRAL INCOMPETENCE/REGURGITATION:

- **Aetiology:**
- o Myxomatous Degeneration, Rheumatic Fever, Infective Endocarditis or **Ischaemia**

Pathogenesis:

o Myxomatous Degeneration – (Pathological weakening of valve connective tissue) o Rheumatic Fever → Autoimmune Mitral Valve Fibrosis → Stenosis **& Regurg** o Infective Endocarditis → Vegetations on Valve Edges → Improper Closure → Regurg o **Ischaemia** – (Post MI Papillary Rupture → Ballooning of Mitral Valve during Systole)

- **Clinical Features & Complications:**
  - o **Symptoms:**
    - § Exertional Dyspnoea
    - § Wet Cough (Pulmonary Oedema)
  - o **Signs:** High-Pitched Pansystolic Murmur – (Loudest @ Apex on Expiration → Axilla)
    - § L-Parasternal Heave (L-Atrial Hypertrophy)
    - §
- **Investigations:**
  - o o **ECHO –** (Diagnostic)
  - o **CXR** (LAH, LVH, Pulmonary Congestion)
- **Management:**
  - o **Medical – CCF Triples (ACEi + B-Blocker + Diuretic)**
  - o **Surgical – Mitral Valvuloplasty (Repair) or Replacement**



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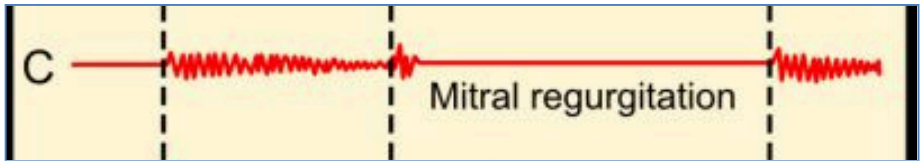

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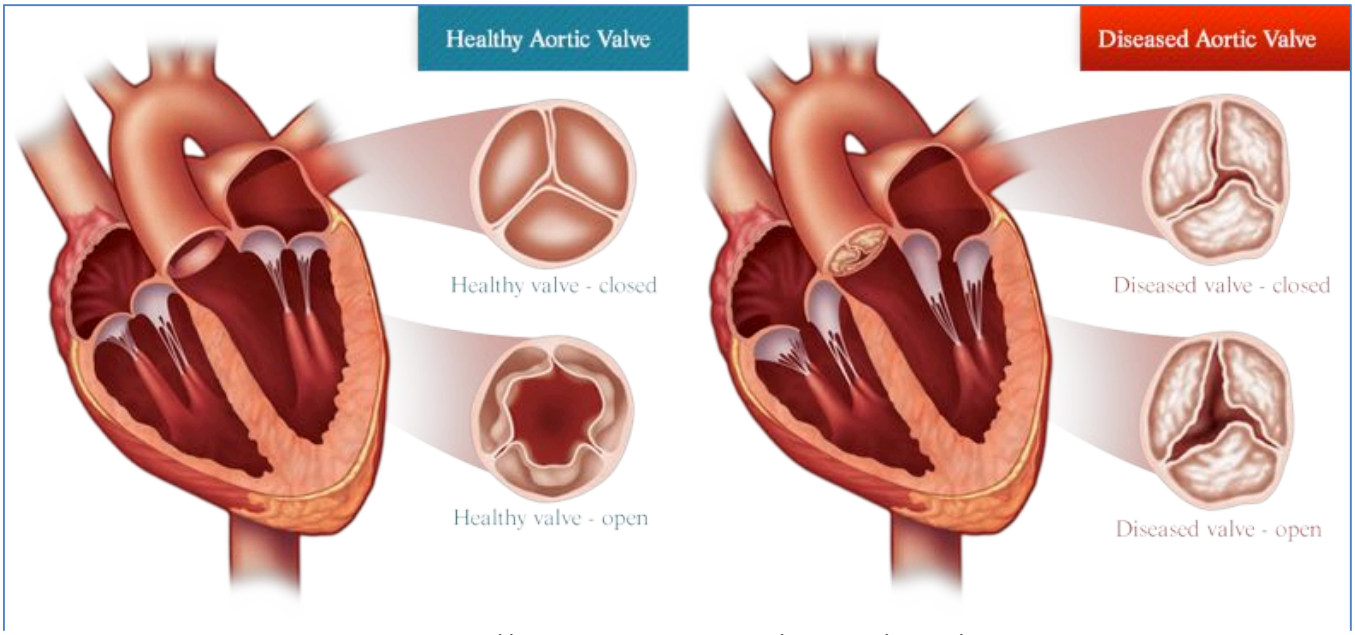
AORTIC STENOSIS:

- **Aetiology:**
  - o Age-Related Calcification (Wear & Tear)
  - o (Also Rheumatic Heart Disease in 10% of cases)
- **Pathogenesis:**
- o Wear & Tear Degeneration + Calcification.

Clinical Features:

- o **Symptoms:**
  - § **** "Aortic Stenosis Triad"****:
    - **1- Angina - (Due to LV-Hypertrophy & ↑Demand)**
    - **2- Exertional Dyspnoea - (Due to Congestive Heart Failure)**
    - **3- Syncope/Dizziness - (Due to ↓Cerebral Perfusion)**
- o **Signs:**
  - § **LV-Hypertrophy** → Displaced Apex Beat.
  - § **Loud *Ejection Systolic Murmur* +/- Thrill** – (Loudest @ 2ndICS R-Sternal Border)
    - Worse on Expiration
    - Radiates to Carotids
  - § **Congestive Heart Failure** → Dyspnoea + Pulmonary Oedema

- **Investigations:**
  - o **ECHO –** (Diagnostic)
  - o **ECG –** (LV-Strain & LVH)
  - o **CXR –** (Calcified Valve, LVH, CCF/Pulmonary Oedema)
- **Management:**
  - o **If Symptomatic → Requires Cardiac Surgery**:
    - § Aortic Valve Replacement.
    - § Or Balloon Valvuloplasty



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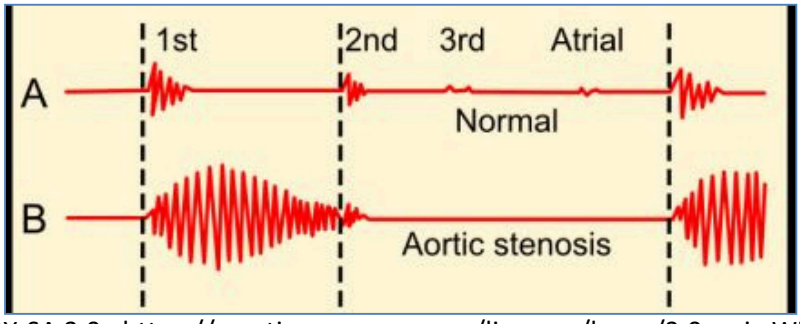

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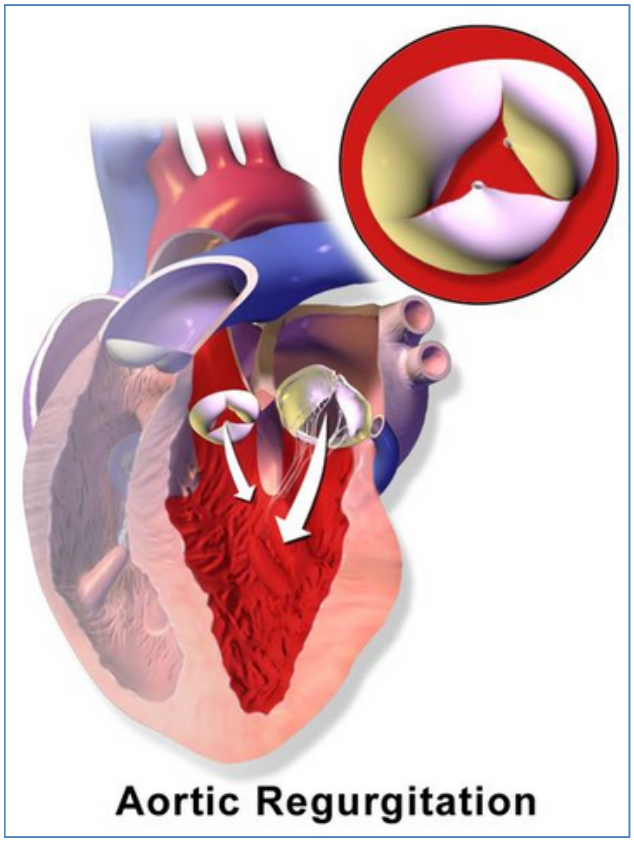
AORTIC INCOMPETENCE/REGURGITATION:

- **Aetiology:**
  - o **Age/Hypertension/"Syphilitic Aortitis"** → Aortic Root Dilation
- **Pathogenesis:**
  - o Dilation of Aortic Root → Valve Leaflets Misalignment → Aortic Regurg

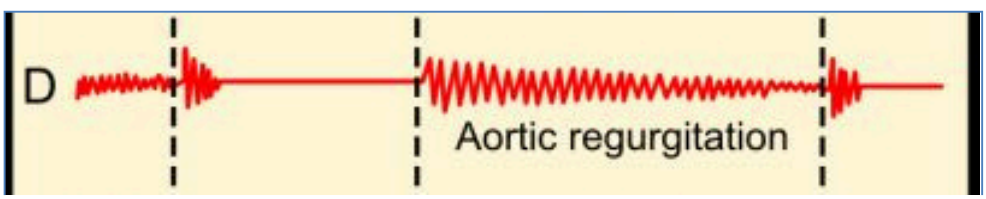
Clinical Features & Complications:

- o **Symptoms:**
  - § **Aortic Triad:**
    - **1- Angina - (Due to LV-Hypertrophy & ↑Demand)**
    - **2- Exertional Dyspnoea - (Due to Congestive Heart Failure)**
    - **3- Syncope/Dizziness - (Due to ↓Cerebral Perfusion)**
- o **Signs:**
  - § "*Waterhammer Pulse*" – (Bounding and Rapidly Collapsing)
  - § Displaced Apex Beat (Due to LV-Hypertrophy)
  - § Diastolic Decrescendo Murmur – (Loudest @ R.2ndICS on Expiration)
  - § Tachycardia (Compensation for ↓CO)

- **Investigations:**
  - o **ECHO –** (Diagnostic)
  - o **ECG –** (LAH + LVH)
  - o **CXR –** (LAH + LVH, CCF/Pulmonary Oedema)
- **Management:**
  - o **Medicine:** Vasodilators + CCF Triple Therapy (ACEi + B-Blocker + Diuretic)
  - o **Surgery:** Aortic Valve Replacement



"Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2). DOI:10.15347/wjm/2014.010. ISSN 2002-4436., CC BY 3.0 via Wikimedia Commons



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TRICUSPID VALVE DISEASE:

- **Aetiology:**
  - **Tricuspid Stenosis:**
  - § Rheumatic, congenital, carcinoid syndrome, fibroelastosis

    Tricuspid Regurg:
    - § RV dilatation (commonest cause), Infective Endocarditis, Rheumatic, Ebstein anomaly, AV cushion defects, carcinoid, tricuspid prolapse, trauma.
- **Symptoms:**
  - **Right Heart Failure:**
    - § Fatigue
    - § Pedal oedema, abdo pain (liver congestion), ascites
    - § Dyspnoea (may reflect right heart forward failure)
- **Signs:**
  - **Carotid Pulse:** Irregular if A-fib and low volume
  - **JVP:**
    - § Increased JVP
    - § Prominent 'A' waves in Tricuspid Stenosis
    - § Large 'V' waves in Tricuspid Regurg
    - § Positive Kussmaul's sign (rise in JVP with inspiration)
  - **Precordial palpation:** Left parasternal lift in Tricuspid regurg
  - **Precordial auscultation:**
    - § (Note: all right sided sounds are louder with inspiration, except a pulmonary ejection click)
    - § Tricuspid Stenosis: Diastolic rumble in 4th left intercostal space
    - § Tricuspid Regurg: Holosystolic murmur along left lower sternal border; may have an ejection murmur
- **Investigations:**
  - **12 lead ECG:**
    - § Tricuspid Stenosis: Right Atrial Enlargement
    - § Tricuspid Regurg: Right Atrial Enlargement, Right Ventricular Hypertrophy, A-Fib
  - **CXR:** Tricuspid Stenosis: Dilatation of right atrium without pulmonary artery enlargement
    - § Tricuspid Regurg: Right atrial and right ventricle enlargement
    - § Diagnostic
  - **ECHO:**
    - §
- **Management:**
  - **Supportive:**
    - § Diuretics, preload reduction
    - § Surgery usually determined by need for other interventions

PULMONARY VALVE DISEASE:

- **(Much less commonly involved)**
- **Aetiology:**
  - **Pulmonary Stenosis:** Usually congenital; uncommonly rheumatic; carcinoid
  - **Pulmonary Regurg:** Secondary to dilatation of valve ring
    - Pulmonary HTN (Mitral stenosis, COPD, recurrent PE
    - Rheumatic
    - Infective endocarditis
- **Symptoms:**
  - Chest pain, Syncope, Dyspnoea, Leg Oedema (RV failure and CHF)
- **Signs:**
  - **Pulmonary Stenosis:**
    - Systolic murmur (maximum at 2nd left intercostal space)
    - Pulmonary ejection click; normal/loud/soft P2; right sided P4
  - **Pulmonary Regurg:**
    - Early diastolic murmur at base
    - Graham Steel (diastolic) murmur at 2nd and 3rd left intercostal space increasing with inspiration.
- **Investigations:**
  - **12-lead ECG:**
  - Right ventricular hypertrophy
  - **CXR:** Prominent pulmonary arteries if pulmonary HTN
    - Enlarged Right Ventricle
    - Diagnostic – RVH, RV dilatation
  - **ECHO** Doppler
    -
    -
- **Management:**
  - Infective endocarditis prophylaxis
  - **Pulmonary Regurg:**
    - Rarely requires treatment (well tolerated if systemic vascular resistance is normal)
    - Valve replacement may be required
  - **Pulmonary Stenosis:**
    - Balloon valvuloplasty, depending on severity

CARDIOVASCULAR RISK FACTORS

OVERWEIGHT & OBESITY:

The General Effect of Obesity on the Body:

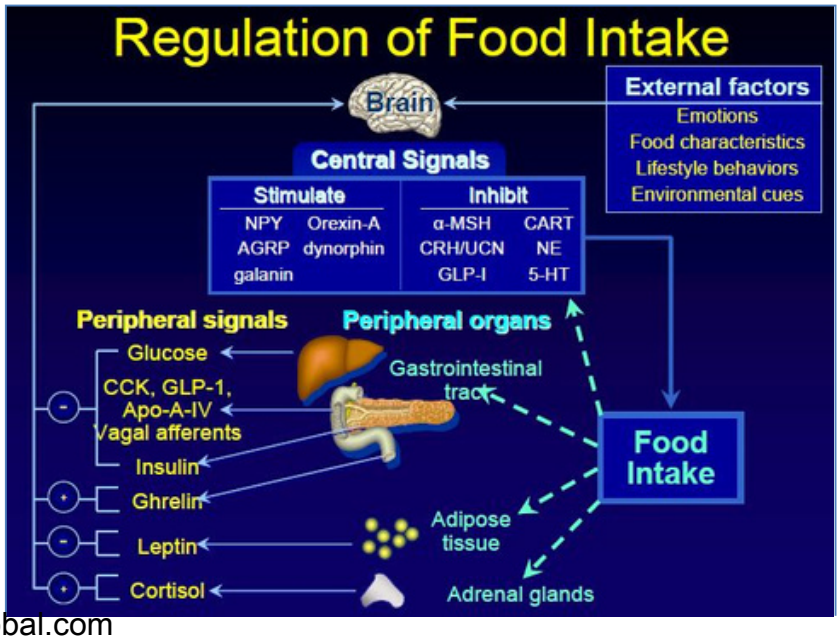
- ↑Fat Mass → ↑Blood Vessels →↑Peripheral Vascular Resistance → ↑Strain on the Heart →↑CVD
- ↑Fat Mass → ↑Body Weight → ↑Wear & Tear on Joints (Particular weight-bearing)→ Arthritis
- ↑Fat Mass → Endocrine Imbalances → Glucose Tolerance .... → Diabetes.
- Many More – Ie: The Whole Body has to work harder to compensate.

What is a Healthy Weight?

- **BMI:**
  - o Normal = 18.5-24.99
  - o Overweight = >25.00
  - o Obese = 30.00→
- **Waist Circumference:**
  - o Better than BMI
  - o Abdominal Adiposity, *Regardless of BMI*, Increases Risk of Certain Obesity-Related Conditions.
  - o Note: Fat deposited elsewhere (hips/buttocks) seems to be less of a risk.
  - o **Healthy Measurements:**
    - § **Women:** Waist Circumference of **88cm** or Less.
    - § **Men:** Waist Circumference **94cm** or Less.

Regulation of Appetite:

- **Central Signals:**
  - o **Appetite Stimulating**
    - § Neuropeptide Y
    - § Agouti Related Peptide
  - o **Appetite Inhibiting**
    - § A-MSH
    - § 5HT
    - § NE
- **Peripheral Signals:**
  - o **Positive Feedback:**
    - § Ghrelin
    - § Cortisol
  - o **Negative feedback:**
    - § Leptin
    - § Insulin



Managing Obese Patients:

- **Weight loss improves all of:**
  - Cholesterol
  - Glucose tolerance
  - HBA1C
  - Blood lipids
- **Obesity Treatment Pyramid:**
  - Lifestyle Mods at the foundation **(Nutrition/Dieting & Physical Exercise)**
  - Pharmacotherapy
  - Surgery (at the top)

- **Nutritional Advice & Food Diaries:**
  - Useful for recording what/how much/where/ate too much?/calories etc.
  - Also useful for monitoring alcohol intake
  - **Note: There is NO particular *Diet* that is proven to cause weight loss:**
    - Instead, it is an Energy Balance.
    - If by eating low-energy density foods, you create an energy deficit in your body, which is
    - supplemented by burning fats.

      Summary:

      - Low energy density, calorie controlled style of eating
        - Increased fruit & veg
        - Reduce sat fat
        - Reduce portion size
        - Regular meals especially breakfast
        - Eat slowly
        - Self-Monitoring (food diary)
    - **Note: Plateaus in weight loss charts are normal & predictable:**
      - Patients plateau after losing an amount of weight because their energy intake (which was previously creating an energy deficit) is now neutral since his body uses less energy to move the increased body mass. (which is now not there)

- **Physical Activity:**
  - ↑Incidental Movement (Mov't is an opportunity rather than inconvenience)
  - Increase aerobic capacity
  - Resistance training
  - Note: Aerobic fitness almost halves risk of cardiovascular disease mortality.
  - Note: Increased body fat increases CVD
  - However, even a fit, obese person has a lower risk of CVD than an unfit, thin person.
  - **Benefits of regular physical activity:**
    - ↓loss of fat-free mass associated with weight loss
    - Improves maintenance of weight loss
    - Improves cardiovascular risk regardless of weight loss.

- **Psychological Component of weight Loss:**
  - Self monitoring
  - Systematic approach to solving problems
  - Contingency plans for times of overeating
  - Stimulus control (identify triggers for overeating)
  - Stress management
  - Social Support (important for both exercise & maintaining dietary change) – Eg: Wife
  - **Cognitive Restructuring**
    - Changing style of thinking
    - Changing Dichotomous thinking (all or nothing; passed or failed; good or bad)
    - Reassessing Unrealistic Goal Setting
    - Body image issues.

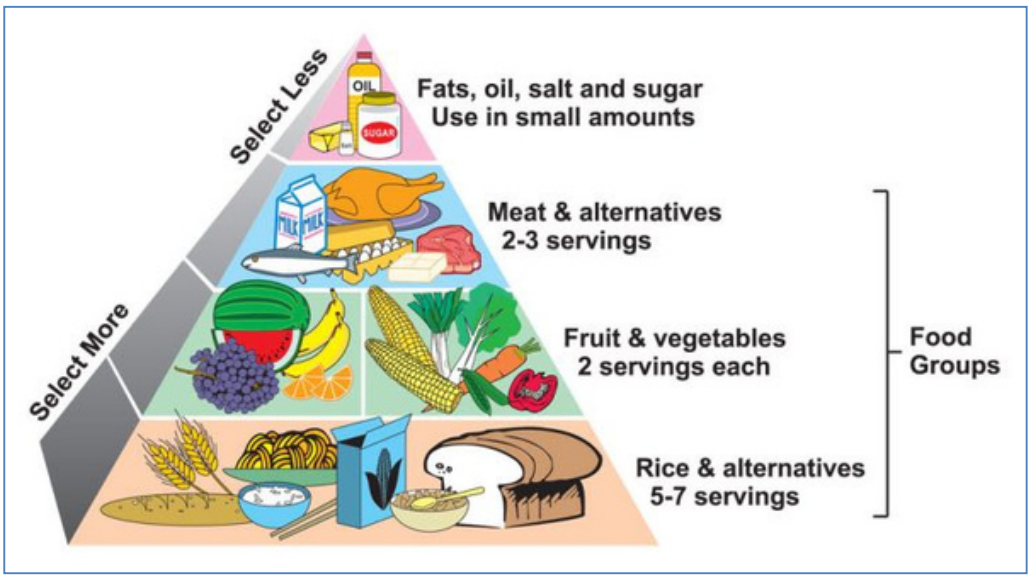
- **Bariatric Surgery:**
  - **Indications:**
    - § BMI over 40
    - § Or life-threatening CVD/diabetes/lifestyle impairment
    - § Failure to achieve adequate weight loss with non0surgical treatment
  - **Contraindications**:
    - § High Risk Heart Disease
    - § Uncontrolled Depression/Psychotic Illness
    - § Active Substance Abuse

Lifestyle Measures to Reduce Risk Factors for Chronic Disease:

- ***Control Blood Pressure:**
  - o Lose Weight
  - o Regular Physical Activity
  - o Nutrition
- ***Maintain a healthy Weight:**
  - o Regular Physical Activity
  - o Nutrition
- ***Physical Activity:**
- o ↑Activity; ↓Sedentary Behaviour

***Nutrition:**

o o 2x Fruits/Day
o 2x Fish/Week – (Omegas - Essential Fats) o Legumes o Limit Alcohol o 5x Veg/Day
Limit Alcohol o Limit Saturated Fats o
Calcium (At least 800mg/day) → Helps reduced BP in Hypertension.



- **The Role of Nutrition in Promoting Health & Preventing Chronic Disease:**
  - Helps Control Blood Pressure
  - Helps Control Hypercholesterolaemia
  - Helps Maintain/Achieve a *Healthy* Body Weight
  - Good Diet Promotes *Good Health* – by supplying the body with all essential vitamins/minerals.

- **The Role of Nutrition in Management of Chronic Disease:**
  - **Nutrition & Obesity – An Energy Balance:**
    - **Losing/Maintaining Weight is a simple *Energy Balance*:**
      - Ie: Energy Input (Caloric Intake) </= Energy Expenditure (Physical Activity).
      - **Note:** There *are* certain *Energy-Dense* foods to avoid (Sweets/Cheese/Butter/Etc), However, you can still get fat if you eat *LOTS* of "Healthy" foods.

  - **Nutrition & Cholesterol – A Problem of *SAT-FATs*:**
    - **Apparently Saturated Fats → ↑LDL Levels:**
      - Don't know how, Just Know that it Does. (Possible Controversy)
      - (Note: LDLs – Low density lipoproteins – are "Bad Cholesterol")
    - **SOURCES OF SATURATED FAT**
      - **ANIMAL PRODUCTS**
        - Fat on meat
        - Skin on chicken
        - Dairy fats
        - Some "deli meats"
      - **• VEGETABLE PRODUCTS**
        - Coconut (milk/cream/oil)
        - Palm oil
        - Tropical oil
        - Vegetable oil (unspecified) eg fish shops
        - Many roasted nuts
    - **REPLACING SATURATED FAT:**
      - **If Pt. Is Overweight:** Carbohydrates
      - **If Pt. Is Thin:** Poly- or mono-unsaturated fats
      - (Decision depends on BMI)

PHYSICAL ACTIVITY:

- **The Role of Physical Activity in Promoting Health & Preventing Chronic Disease:**
  - **Note: *DECREASING* Sedentary Behaviour is *MORE EFFECTIVE* than *INCREASING* Exercise**
    - Both are good, but doing exercise is pointless if you lead a Sedentary Lifestyle.
    - ***What you WANT to do is ↑PHYSICAL ACTIVITY***.
    - **Ie: Exercise ≠ Physical Activity:**
      - **Exercise =** Dedicated Physical Exertion
      - **Physical Activity =** Miscellaneous Day-to-Day Activity.

- **The Perils of Sedentary Behaviour:**
  - **Sedentary Behaviour is *DIRECTLY LINKED to*:**
    - ** CVD (Note: All of the below further contribute to CVD)
    - *Obesity
    - *Depression
    - *Diabetes (Typically type 2)
    - Osteoporosis
    - Stroke
    - Hypertension
    - High Cholesterol
  - **Exercise is known to a) Reduce the Risk of these conditions, but b) Also Decrease their Severity**.
    - Ie: Any Increase in Physical Activity (be it small/large) is immensely beneficial, *Even* in patients who already have these diseases.

- **The Rewards of ↑Physical Activity:**
  - ↑Lean body mass
  - ↑Bone density
  - ↑Cardiac output
  - ↑Oxygen carrying capacity & exchange
  - ↑Metabolism
  - Improved neurotransmitter regulation
  - Improved mood, self-efficacy
  - Improved QOL

- **Physical Activity Guidelines:**
    - (**Note: Moderate Activities =** Brisk walk, a Bike ride or Active Play)
    - (**Note: Vigorous Activities =** Anything that makes the kid "huff and puff" (Ie: Sports))
  - **5-12 year olds:**
    - Combination of Moderate and Vigorous Activities for At Least **60mins/day**.
    - **Note: Children & Adolescents require almost *Double***.
  - **12-18 year olds:**
    - At least **60mins** of Moderate to Vigorous Physical Activity Every Day.
    - **Note: Children & Adolescents require almost *Double***.
  - **Adults:** Step 1 – Think of movement as an opportunity, not an inconvenience
    - Step 2 – Be active every day in as many ways as you can
    - Step 3 – At least **30mins** of Moderate Physical Activity per day. (At least **5 days a week**)
    - Step 4 – Some Regular, Vigorous Activity for *extra* health and fitness.
    - **Note: If Exercise is being used as a disease *Intervention*, the recommendations are**
    - ***Doubled***.

  - **Elderly/Completely Sedentary:**
    - *ANY Physical Activity is Beneficial*.

- **The Role of Physical Activity in Management of Chronic Disease:**
  - **Cardiovascular Disease (Post Myocardial Infarction):**
    - **Post-MI Exercise is *Immediate*:**
      - Ie: Within days after the MI.
      - **Note:** However, It is only *LOW INTENSITY*.
      - **Note:** Cardiac Rehab is usually done as an *INPATIENT* under close supervision.
    - **Recommendations (MI)**
      - Begin ASAP
      - 3days/wk
      - 20-60 min (cardiac rehab) PLUS home-based
      - Start @ 40-60% Heart-Rate Reserve; progress to 85%
      - (HR Reserve = 220 – Age – Resting HR.)
    - **Benefits (MI):**
      - Improved cardio-respiratory function
      - Protection against exertional MI trigger
      - Reduced HR, BP, LDL, TC
    - **Contraindications (CVD):**
      - Change in Resting ECG Indicating Ischemia/MI/Unstable Angina/Uncontrolled Dysrhythmias.
      - Symptomatic Aortic Stenosis
      - Uncontrolled Heart Failure
      - Pulmonary Embolus/infarction
      - Myo-/Pericarditis

  - **Diabetes:**
    - **Diabetics are advised to Cycle/Swim instead of Running:**
      - Because Peripheral Vasculopathy & Neuropathy in Diabetes → Repetitive Trauma to feet + Little Sensation → Formation of Ulcers.
    - **Benefits:**
      - Improved Action of Insulin (Insulin Sensitivity)
        - Note: Exercise + Normal dose of Insulin = Additive Effect; can cause hypoglycaemic shock. Therefore Necessary to ↓Insulin dose with Exercise.
        - Note: Can even *Reverse* Type-2 Diabetes.
      - Improved Glucose tolerance
      - Improved weight management
      - Improved BP → Decreased CVD Risk
      - Improved Lipid profiles →Decreased CVD risk
    - **Recommendations:**
      - Aerobic Exercise: (20-60min @ Heavy Exertion (High RPE) At least 4 Times/wk)
      - Strength: (Low Resistance @ Moderate Exertion (RPE 11-16) 2-3 Times/wk.)
      - Flexibility, balance & coordination: (2-3xwk)
    - **Precautions:**
      - Effects of insulin & exercise are ADDITIVE - (Adjust insulin dose accordingly)
      - If BG <4 or >17 mmol/L, delay exercise until stable
      - Always have glucose handy (honey, jelly beans) in case of Insulin Overdose.
      - Illness, infection, retinal haemorrhage, peripheral neuropathy