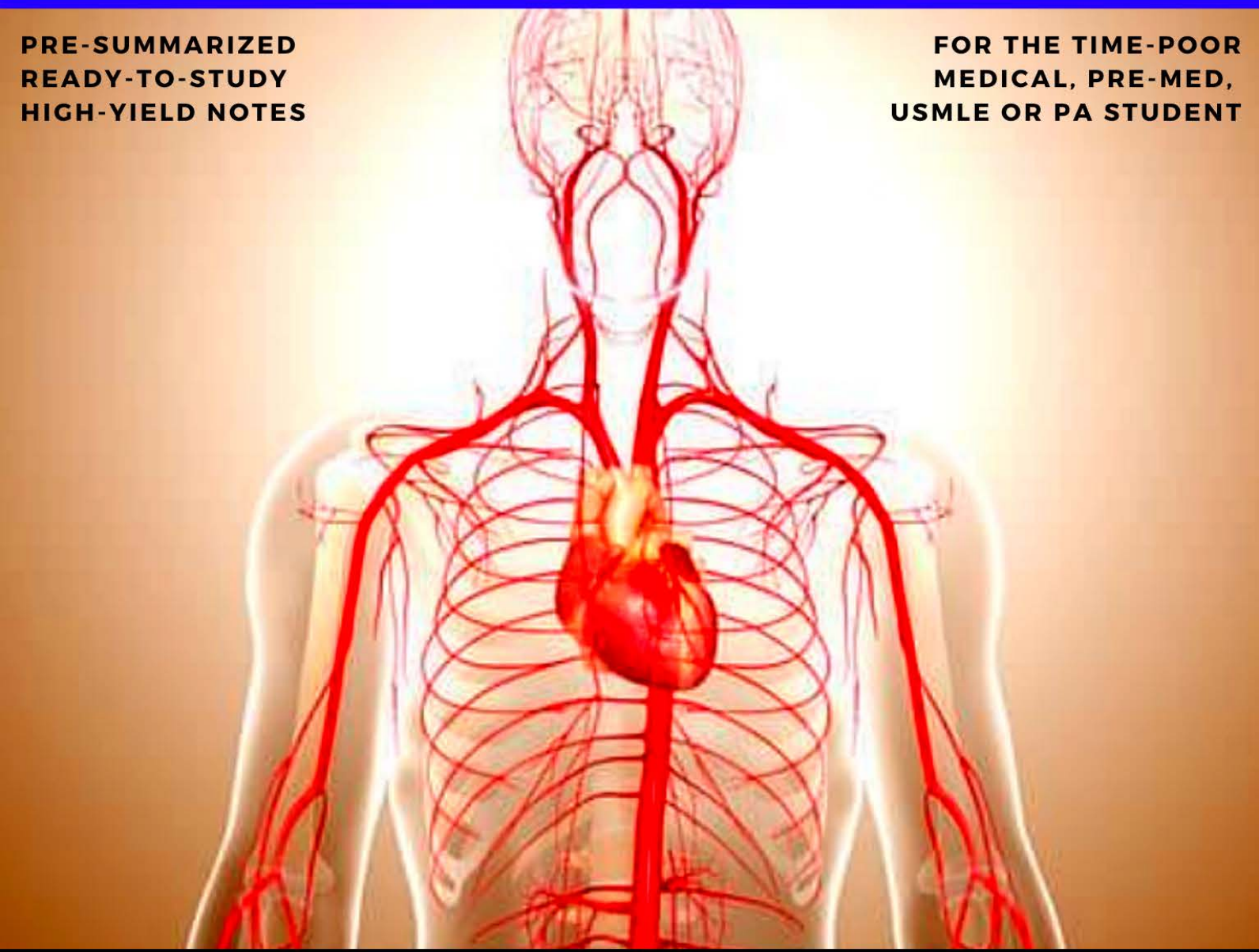
ANATOMY, PHYSIOLOGY & PATHOLOGY NOTES OF THE

CARDIOVASCULAR SYSTEM

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

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



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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.

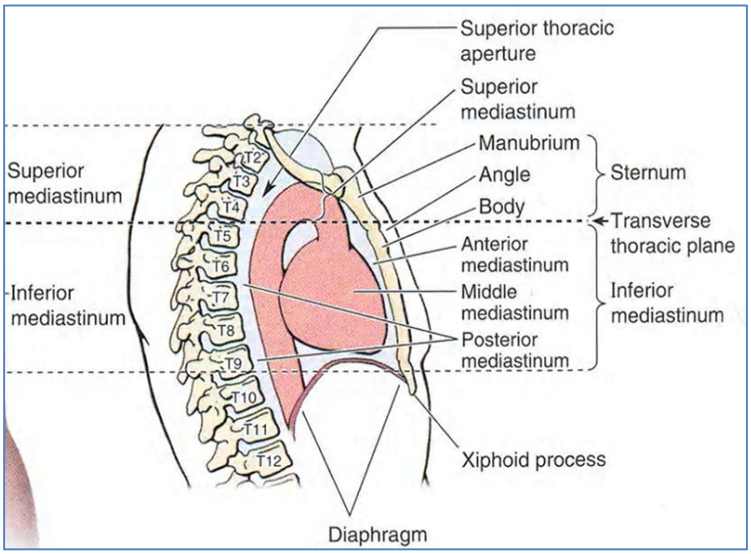
Free bonuses: 'Cardiology and Cardiac Surgery' chapter of Toronto Notes for reference and further detailed reading.

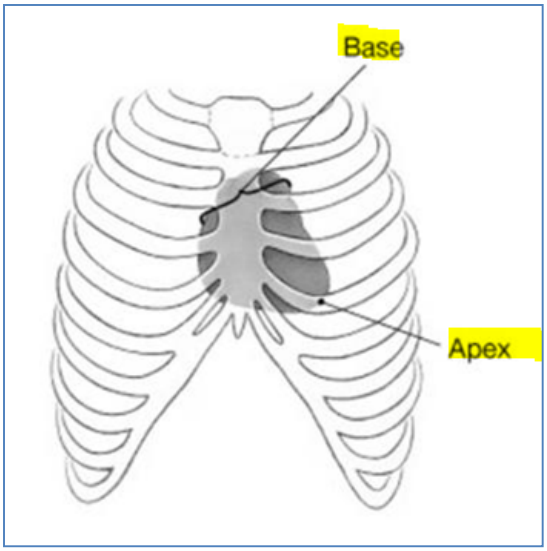
File List:

A&P
Heart Anatomy

Heart Anatomy:

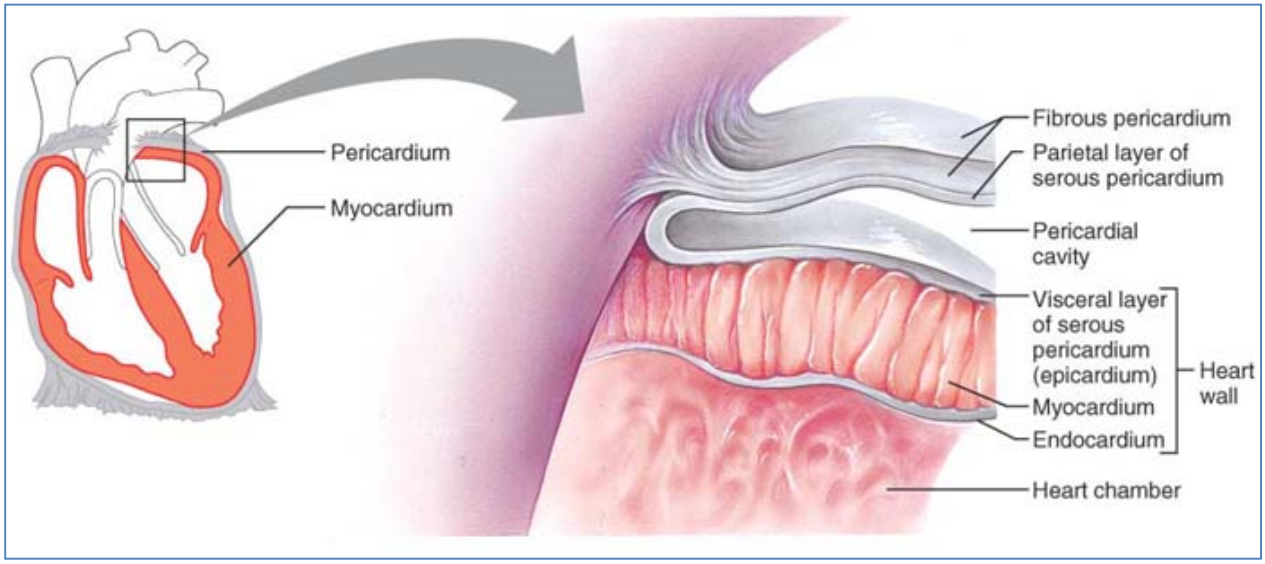
- **Location:**
  - Snugly enclosed within the ***middle mediastinum*** (medial cavity of thorax)
    - Contains the heart, pericardium, vessels to & from the heart & lungs, trachea & oesophagus.
    - M.Mediastinum – located in the inferior mediastinum (lower than the sterna angle)
  - Extends obliquely from 2nd rib → 5th intercostals space.
  - Anterior to Vertebrae
  - Posterior to Sternum
  - Flanked by 2 lungs
  - Rests on the diaphragm
  - 2/3 of its mass lies to the LHS of the *midsternal line*.



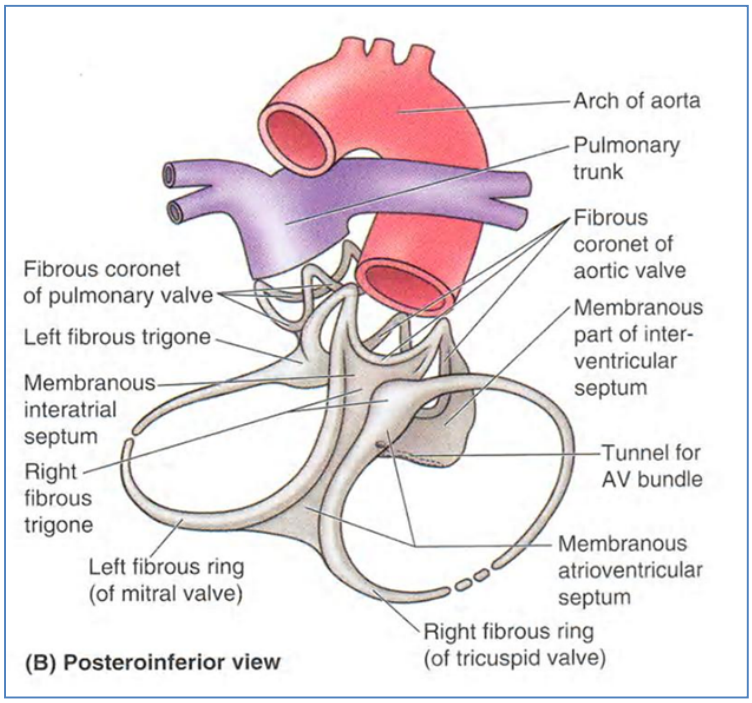


- **The Pericardium: (Coverings of the Heart)**
  - A double-walled sac
  - contains a film of lubricating serous fluid
  - **2 Layers of Pericardium:**
    - **Fibrous Pericardium:**
      - Tough, dense connective tissue
      - Protects the heart
      - Anchors it to surrounding structures
     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

- **Layers of the Heart Wall:**
  - **Epicardium:**
    - Visceral layer of serous pericardium
  - **Myocardium:**
    - Muscle of the heart
    - The layer that 'contracts'
  - **Endocardium:**
    - Lines the chambers of the heart
    - Prevents clotting of blood within the heart
    - Forms a barrier between the O^2 hungry myocardium and the blood. (blood is supplied via the coronary system)

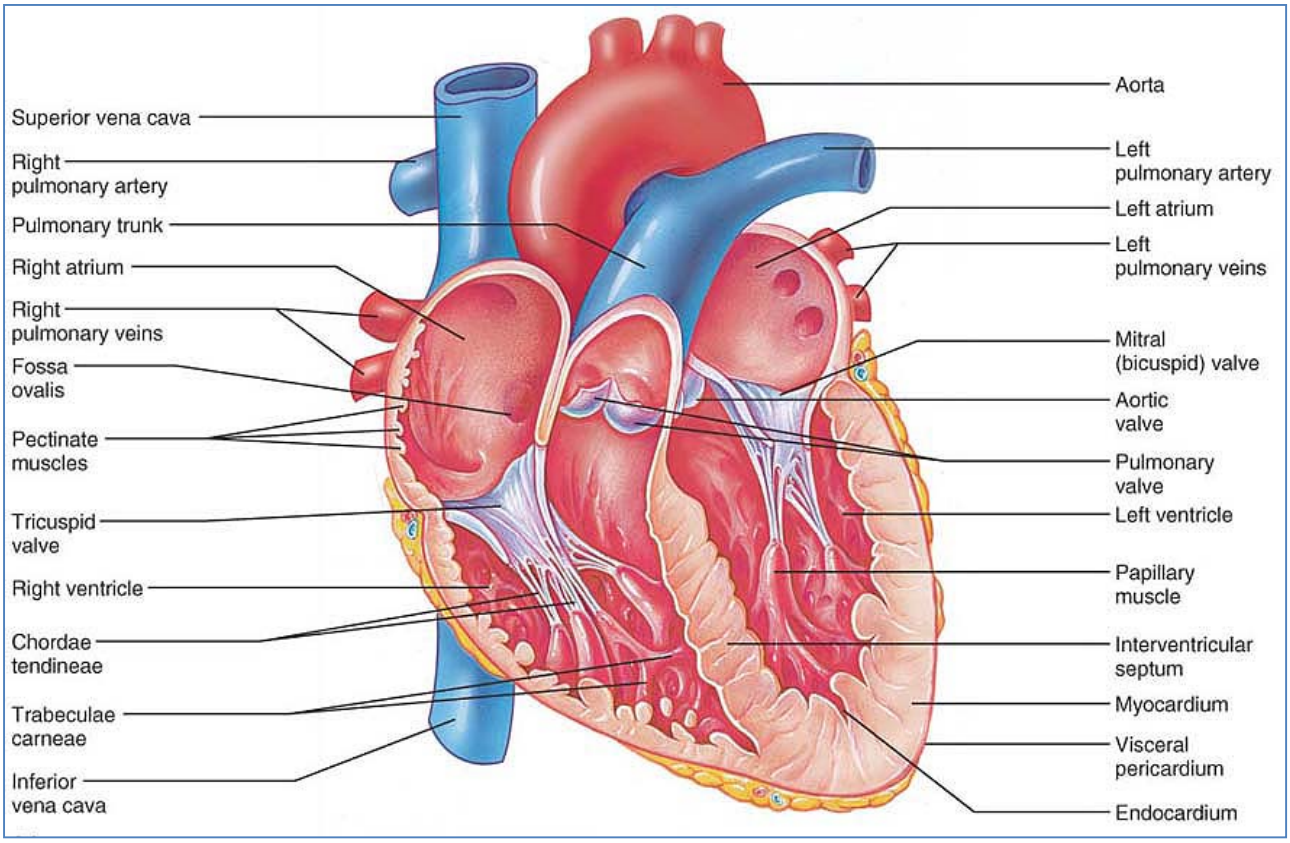


- **Fibrous Skeleton of the Heart:**
  - The network of connective tissue fibres (collagen & elastin) within the myocardium
  - Anchors the cardiac muscle fibres.
  - Reinforces the myocardium
  - **2 Parts:**
    - **Septums:**
      - Flat sheets separating atriums, ventricles & left and right sides 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

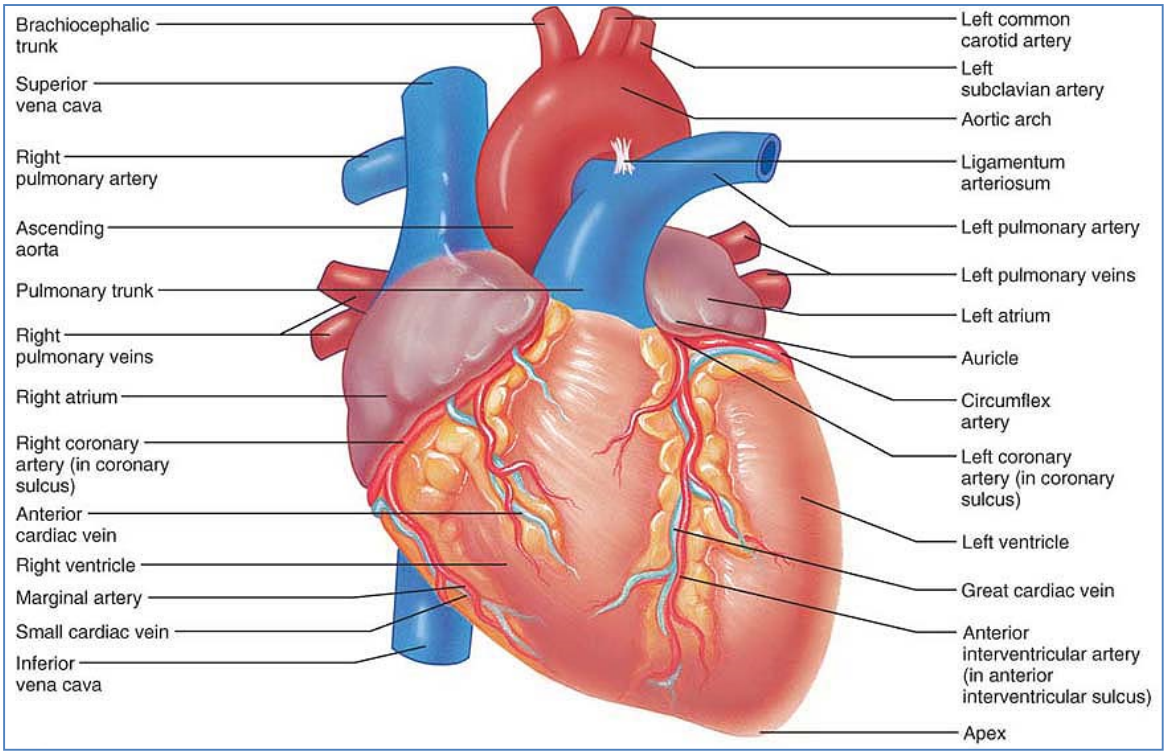


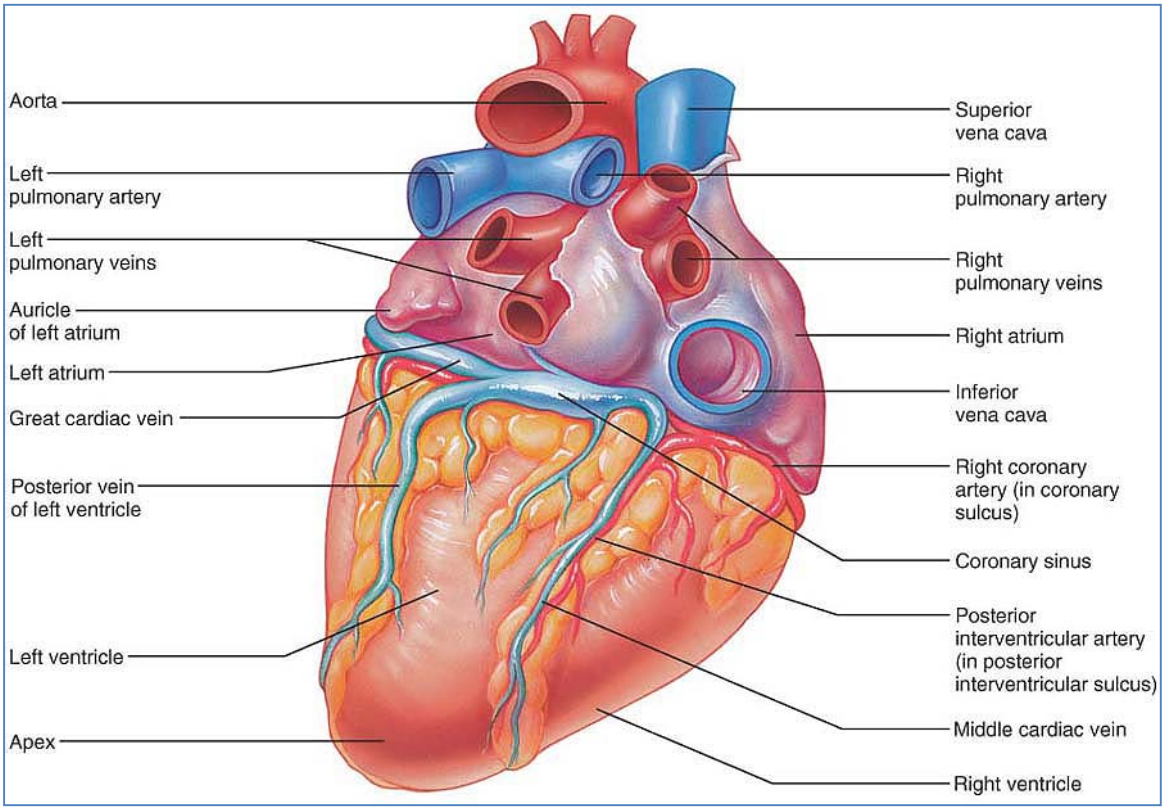
- **Chambers & Associated Great Vessels:**
  - **2 Atrias (superior):** [Atrium = Entryway]
    - Thin-walled Receiving Chambers
    - On the back & superior aspect of heart.
    - Each have a small, protruding appendage called **Auricles** – increase atrial volume.
    - Septal Area
      - Connective tissue dividing L & R atria. (Site of Foetal Shunt Foramen ovale)
    - **Right Atrium:**
      - Smooth internal posterior wall
        - Where veins drain into (either from body/lungs)
      - 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:**
      - Smooth internal post. & ante. walls.
      - Blood enters via:
        - The 4 pulmonary veins (O^2 blood) [Pulmonos = Lung]

  - **2 Ventricles (inferior):** [Vent = Underside]
    - Thick, muscular Discharging Chambers
    - The 'pumps' of the heart
    - Trabeculae Carnea [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:**
      - Thicker – it is responsible for the *Systemic Circulation* – Via **Aorta**
      - Most of the heart's PosteroInferior Survface



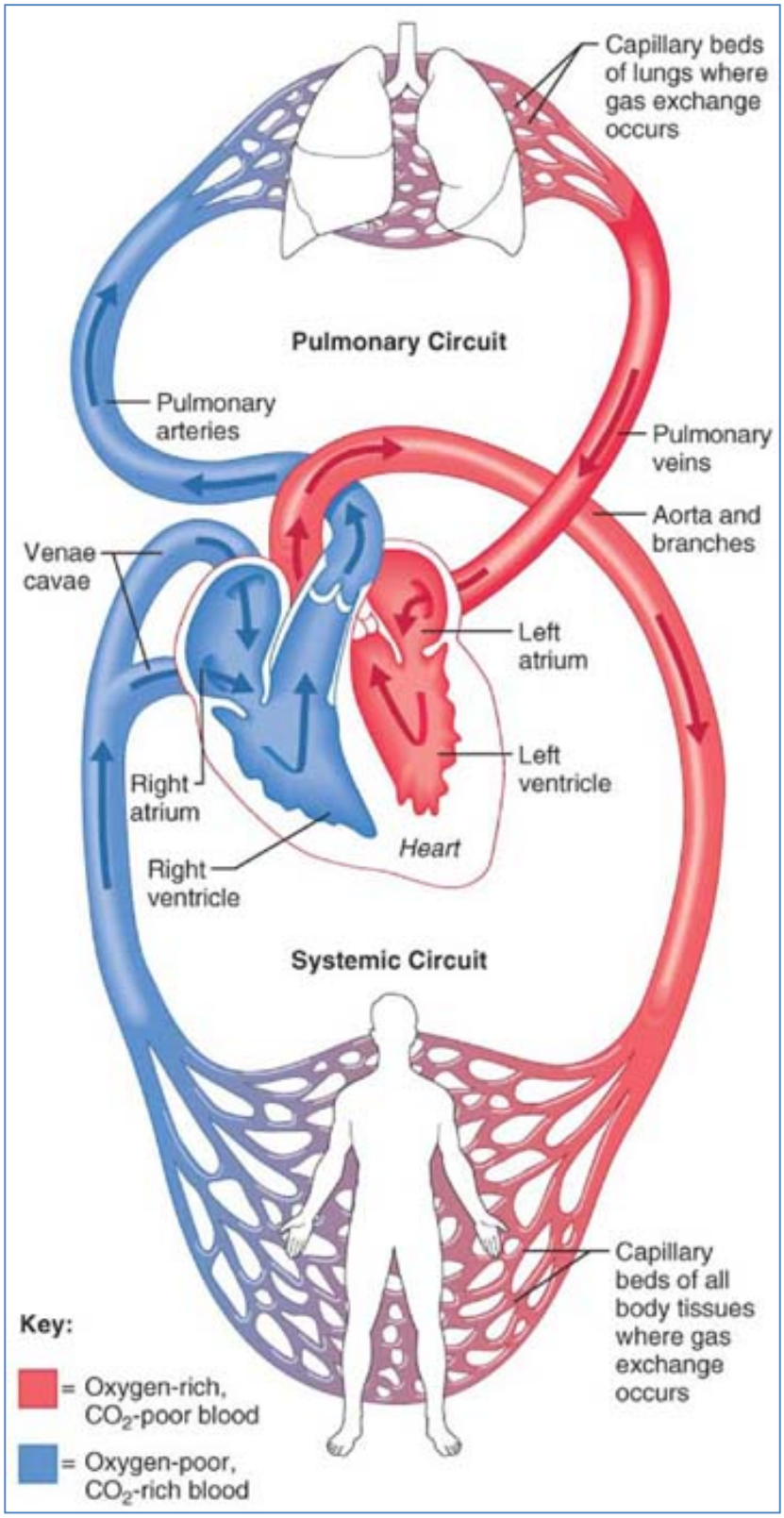
- **Landmarks of the Heart:**
  - **Coronary Sulcus (Atrioventricular Groove):**
    - Encircles the junction between the Atria & Ventricles like a 'Crown' (Corona).
    - Cradles the Coronary Arteries (R&L), Coronary Sinus, & Great Cardiac Vein
  - **Anterior Interventricular Sulcus:**
    - Cradles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





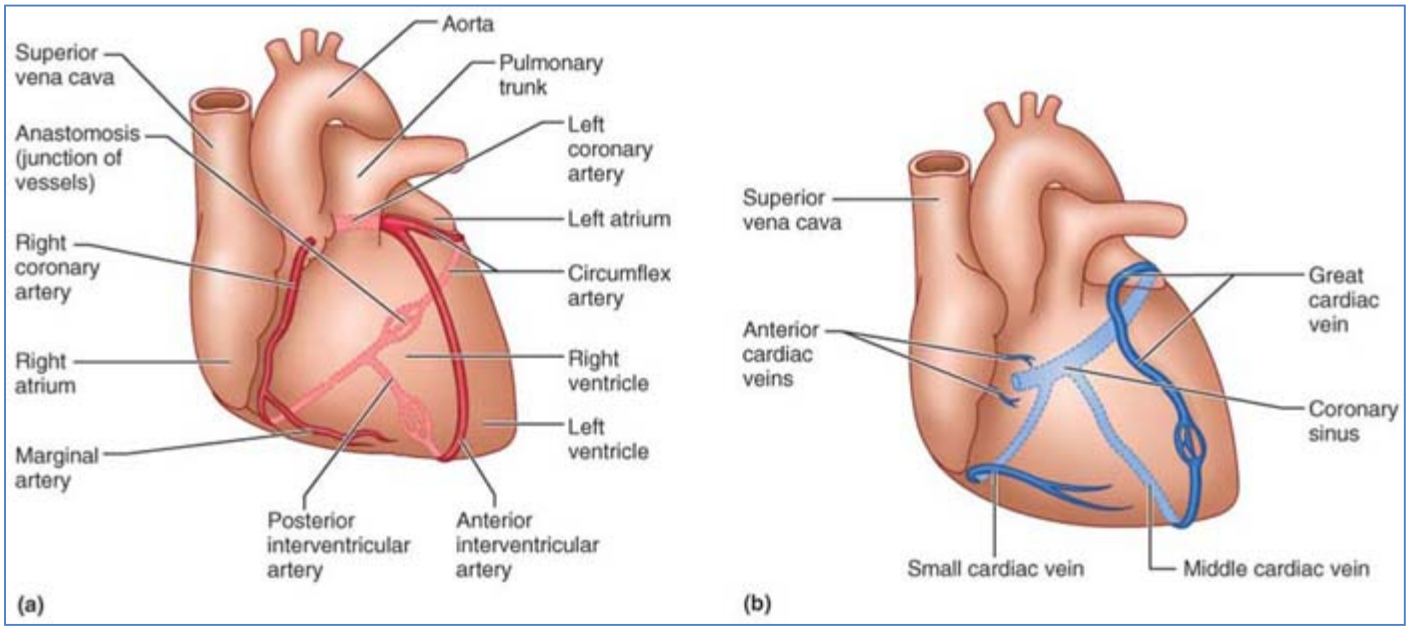
Pathway of Blood Through the Heart:

- **The systemic and pulmonary circuits:**
  - 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 color 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 color change)



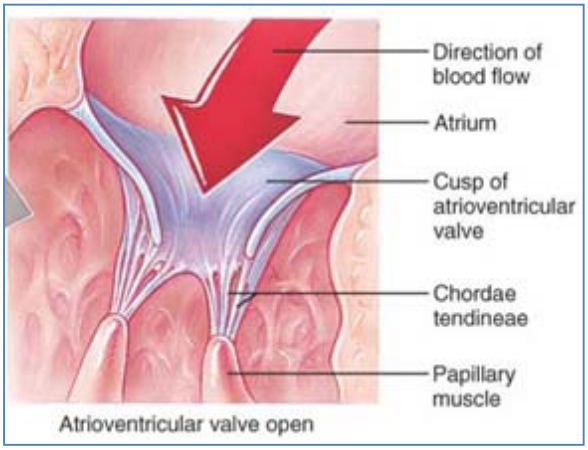
Coronary Circulation:

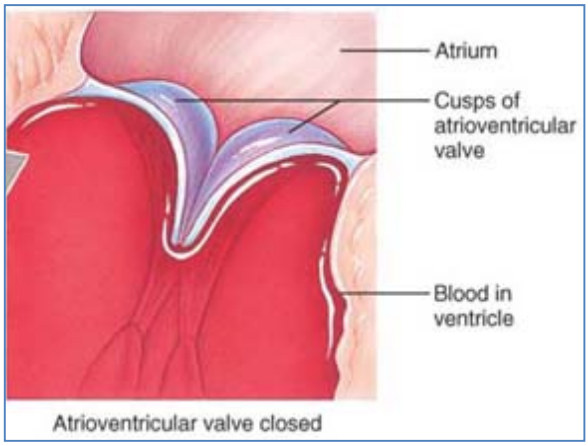
- The myocardium's own blood supply
- The shortest circulation in the body
- Arteries lie in epicardium – prevents the contractions inhibiting bloodflow
- There is a lot of variation among different people.
- **Arterial Supply:**
    - Encircle the heart in the coronary sulcus
    - **Aorta** → Left & 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 blood to InterVentricular Septum & Anterior walls of both Ventricles.
            - **2. Circumflex Artery**
                - Follows the Coronary Sulcus (aka. AtrioVentricular Groove)
                - Supplies the Left Atrium & Posterior walls of the Left Ventricle
        - **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
- **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.**



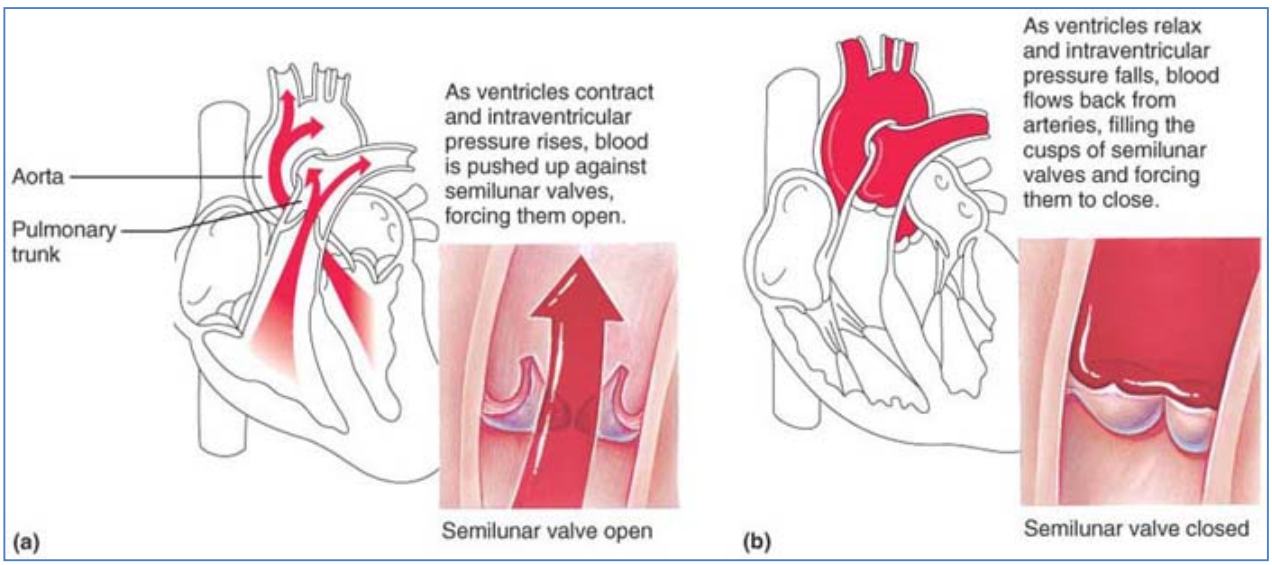
Heart Valves:

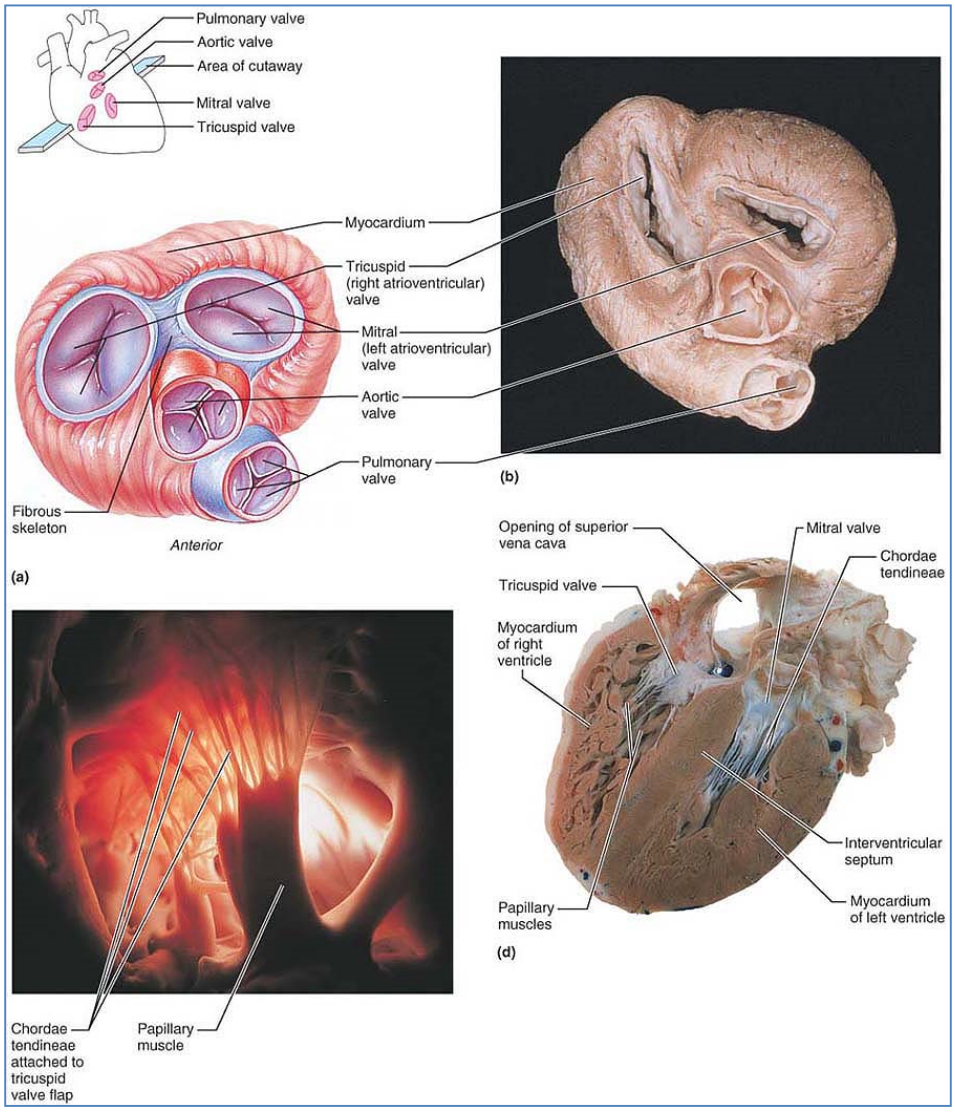
- Ensure *unidirectional flow of blood* through the heart.
- **2x AtrioVentricular (AV) (Cuspid) Valves:**
  - Located at the 2 Atial-Ventricular junctions
  - Prevent backflow into the Atria during Contraction of Ventricles
  - Attached to each valve flap are **chordae tendinae** (tendonous cords) "heart strings"
    - Anchor the cusps to the **Papillary Muscles** protruding from ventricular walls.
      - Papillary muscles contract before the ventricle to take up the slack in the chordae tendinae.
      - Prevent inversion of valves under ventricular contraction.
  - **Right AV Valve:**
    - **The "Tricuspid Valve"**
    - 3 flexible 'cusps' (flaps of endocardium + Conn. Tissue)
  - **Left AV Valve:**
    - **The "Mitral Valve" or "Biscupid Valve"**
    - (resembles the 2-sided bishop's *miter* [hat])





- **2x SemiLunar (SL) Valves:**
  - Guard the bases of the 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

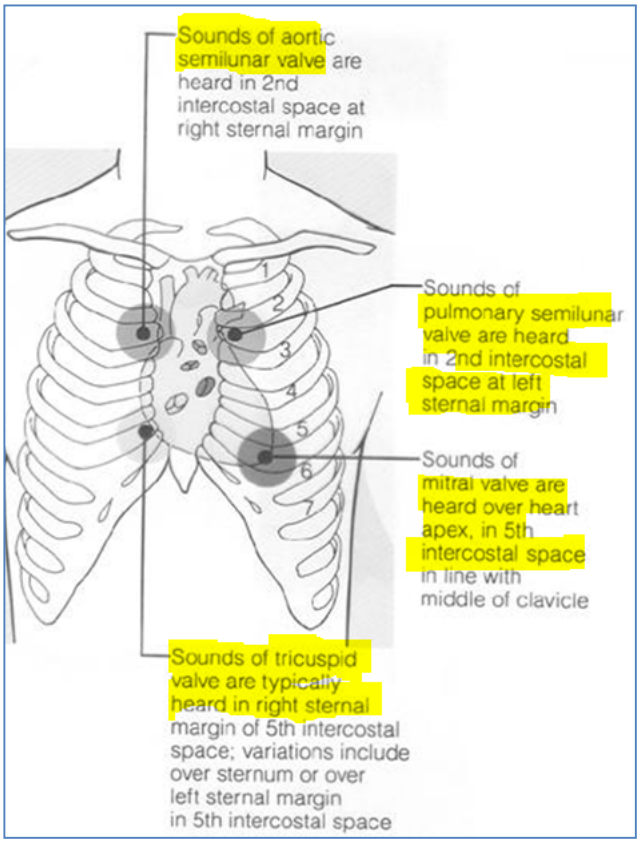




Valve Sounds:

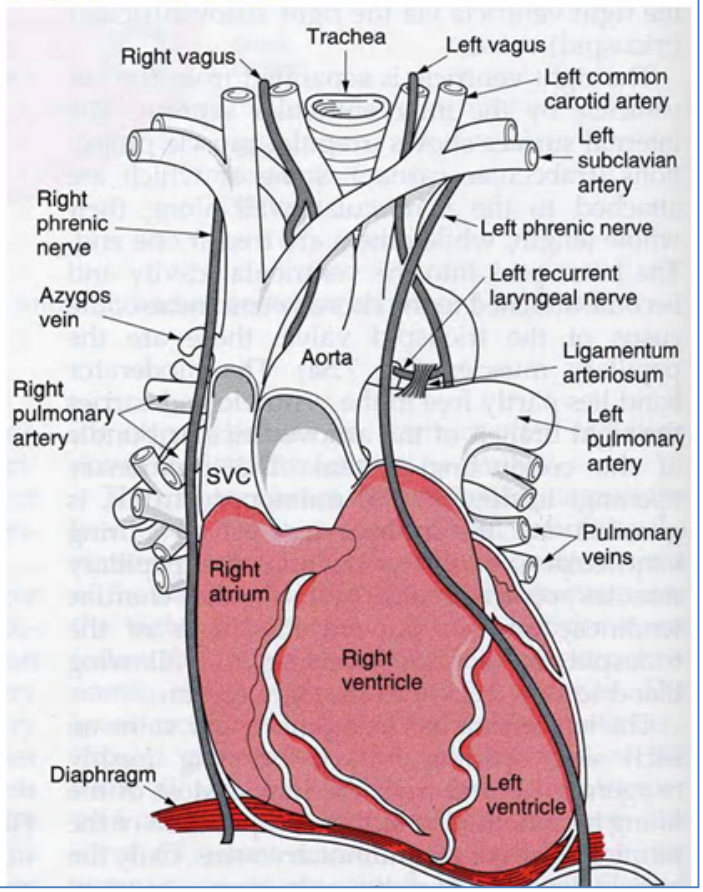
- **1. "Lubb":**
  - Sound of a Cuspid Valve closing
- **2. "Dupp":**
  - Sound of a Semilunar Valve Closing

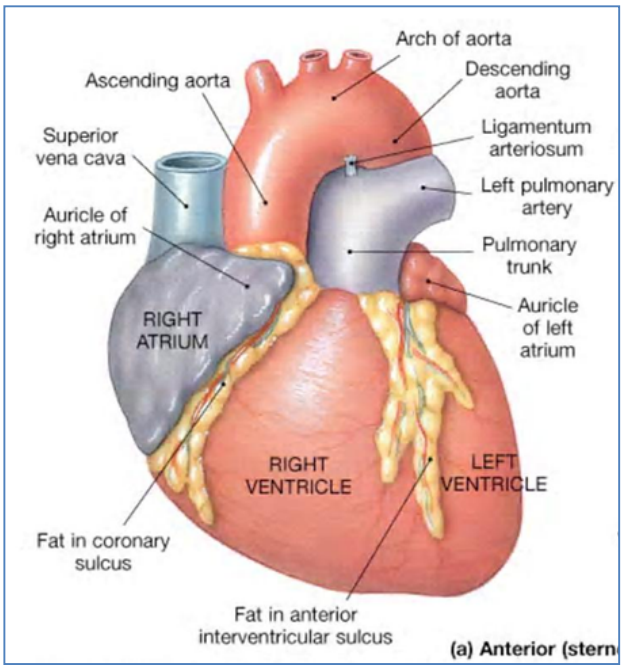
- **Where to Listen:**

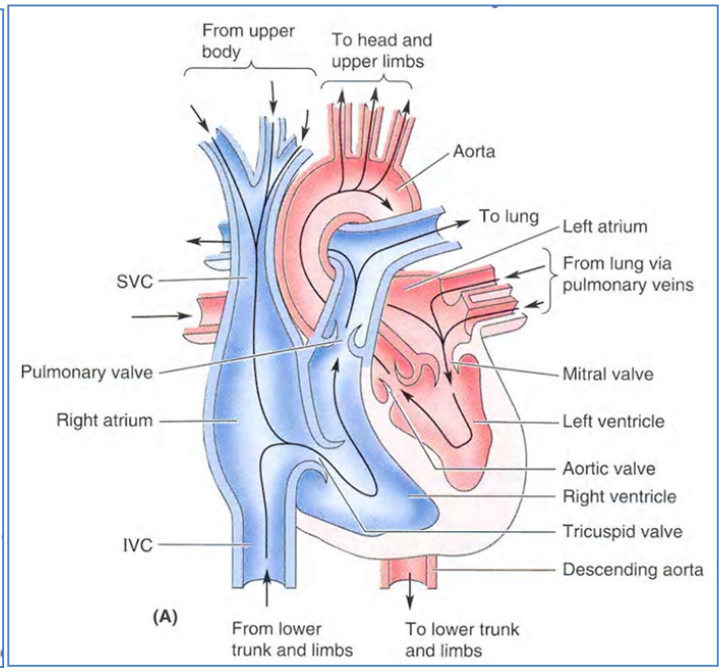


Relations

- Right and left phrenic nerves (C3, 4,5) pass anterior to lung root
- Right and left vagus (X) pass posterior to lung root
- Note relationship of nerves to the heart chambers



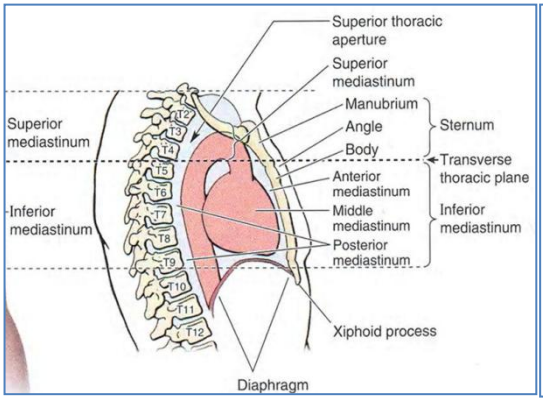


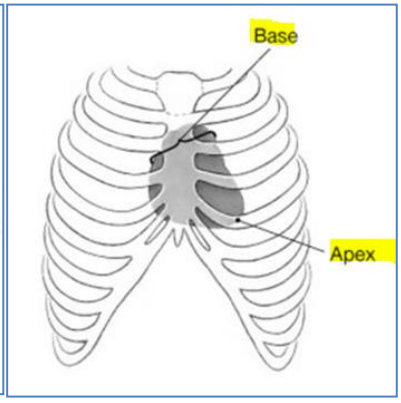


Basic Anatomy & Physiology:
Cardiovascular

Heart Anatomy:

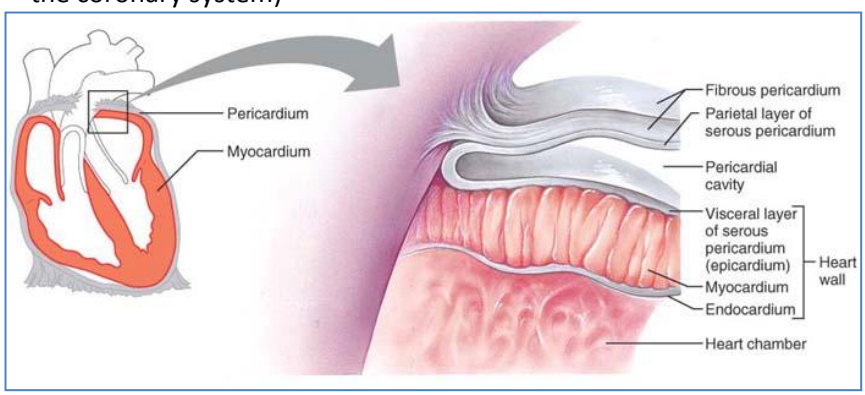
- **Location:**
  - Snugly enclosed within the ***middle mediastinum*** (medial cavity of thorax). Contains:
    - Heart
    - Pericardium
    - Great Vessels
    - Trachea
    - Esophagus.



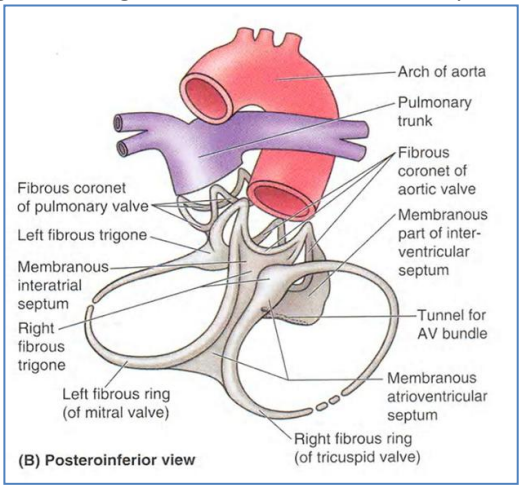


- **The Pericardium: (Coverings of the Heart)**
  - A double-walled lubricating sac
  - **2 Layers of Pericardium:**
    - **Fibrous Pericardium:**
      - Tough, dense connective tissue
      - Protects the heart
      - Anchors it to surrounding structures
    - **Serous Pericardium:** (one continuous 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 the serous pericardium
  - **Myocardium:**
    - Muscle of the heart
  - **Endocardium:**
    - Endothelium lining the chambers of the heart
    - Prevents clotting of blood within the heart
    - Forms a barrier between the O^2 hungry myocardium and the blood. (blood is supplied via the coronary system)



- **Fibrous Skeleton of the Heart:**
  - **Functions:**
    - Reinforces Myocardium
    - Anchors muscle fibres + valves + great vessels
    - Electrically isolates
  - **2 Parts:**
    - **Septums:**
      - Flat sheets separating atriums, ventricles & left and right sides of the heart.
      - Electrically isolates the L&R sides of the heart
    - **Rings:**
      - Rings around great vessels & valves → stop stretching under pressure



- **Chambers & Associated Great Vessels:**
  - **2 Atrias (Superior):**
    - Thin-walled Receiving Chambers
    - **Right Atrium:**
      - Blood enters via 3 veins:
        - SVC
        - IVC
        - Coronary Sinus (collects venous blood draining from the myocardium)
    - **Left Atrium:**
      - Blood enters via:
        - 4x Pulmonary Veins (O^2 blood)

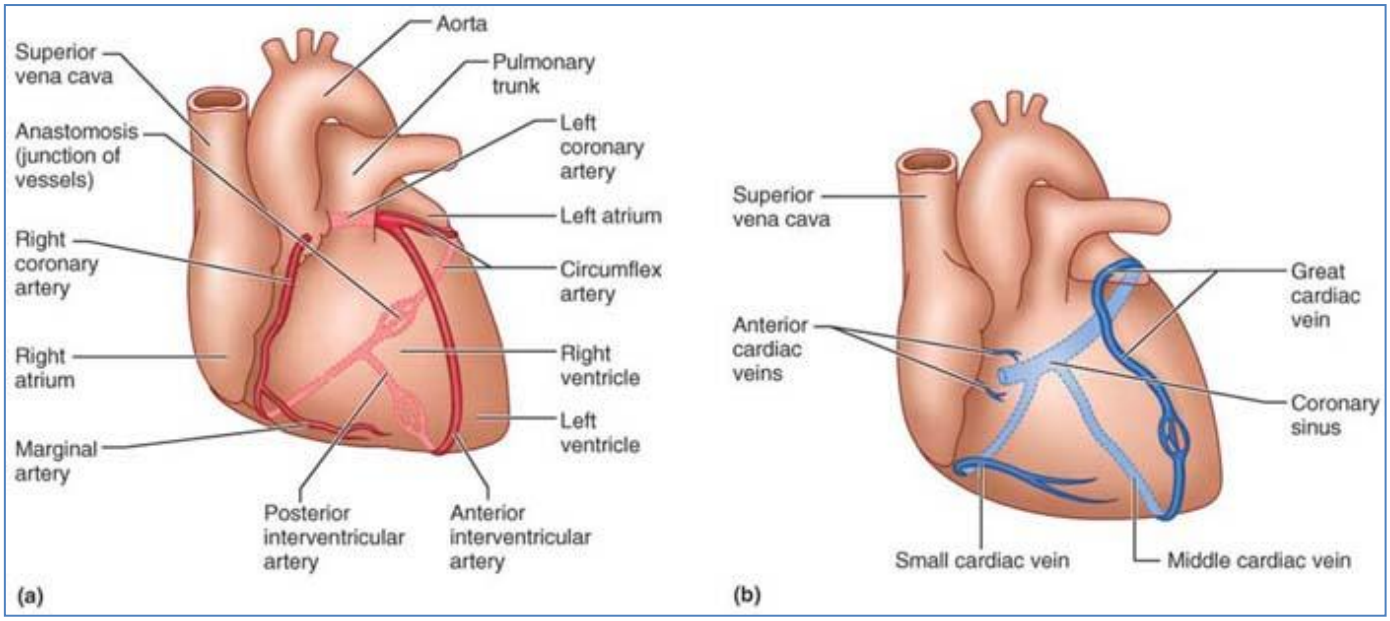
  - **2 Ventricles (Inferior):** [Vent = Underside]
    - Thick, muscular pumping chambers
    - **Right Ventricle:**
      - Anterior Surface of Heart
      - Thinner – *Low Pressure Pulmonary Circulation* – Via **Pulmonary Arteries**
    - **Left Ventricle:**
      - PosteroInferior Surface of Heart
      - Thicker – *High Pressure Systemic Circulation* – Via **Aorta**

Landmarks of the Heart:

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

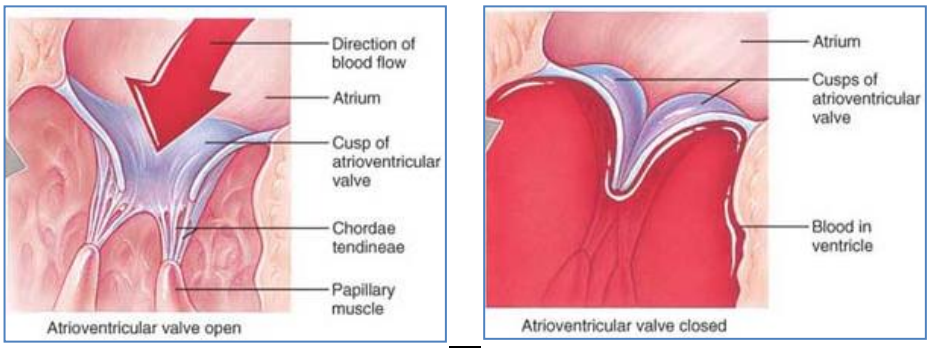
Coronary Circulation:

- The myocardium's own blood supply
- Arteries lie in epicardium – prevents the contractions inhibiting bloodflow
- **Arterial Supply:**
  - **Aorta** → Left & Right *coronary arteries*
    - **Left Coronary Artery** →
      - **1. Left Anterior Descending** → *Apex, Anterior LV, Anterior 2/3 of IV-Septum*.
      - **2. Circumflex Artery** → *L atrium + Lateral LV*
    - **Right Coronary Artery →Marginal & Post-Interventricular Artery →**
      - *R-Atrium*
      - *Entire R-Ventricle*
      - *Posterior 1/3 of IV-Septum*

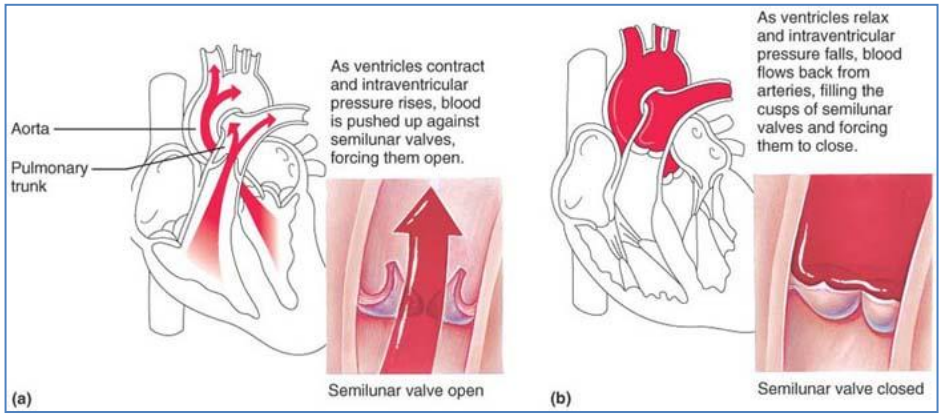


Heart Valves:

- Ensure *unidirectional flow of blood* through the heart.
- **2x AtrioVentricular (AV) (Cuspid) Valves:**
  - Prevent backflow into the Atria during Contraction of Ventricles
    - Papillary muscles contract before the ventricle to take up the slack in the chordae tendinae →Prevents ballooning of valves under ventricular contraction.
  - **Tricuspid Valve (Right):**
    - 3 flexible 'cusps' (flaps of endocardium + Conn. Tissue)
  - **Mitral Valve (Left):**
    - 2 Leaflets - resembles the 2-sided bishop's *miter* [hat]



- **2x SemiLunar (SL) Valves:**
  - Open under Ventricular Pressure
  - 3x Cusps each
  - **Pulmonary Valve:**
    - Between Right Ventricle & Pulmonary Trunk
  - **Aortic Valve:**
    - Between Left Ventricle & Aorta



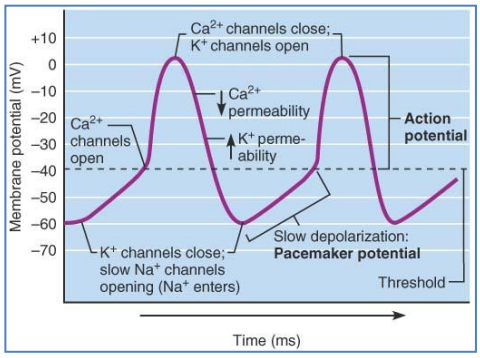
Valve Sounds:

- **S1 ("Lubb"):**
  - AV Valve Closure
  - (M1 = Mitral Component)
  - (T1 = Tricuspid Component)
- **S2 ("Dupp"):**
  - Semilunar Valve Closure
  - (A2 = Aortic Component)
  - (P2 = Pulmonary Component)

Electrophysiology & ECGs

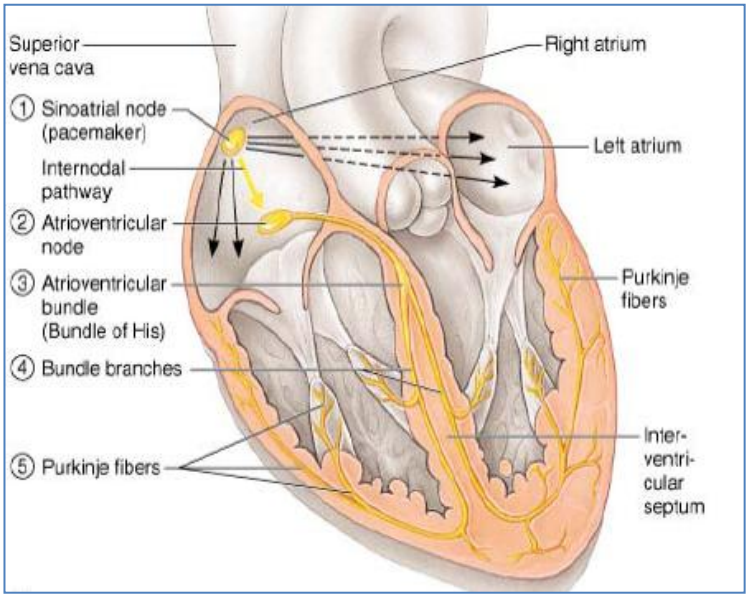
2 Types of Cardiac Muscle Cells:

- **Conductile/Nodal: (*Intrinsic*)**
  - **Have *Spontaneous Electrical Activity* – Cannot Maintain a Resting Membrane Potential**
    - Spontaneously Depolarises to Threshold (Due to Leaky Na^+ Membrane Ion Channels)
    - **NB:** ↑Na^+ brings to threshold, but Ca^+ is responsible for Depolarisation.
    - → Slow 'Pacemaker' Action Potentials
  - ***Heirarchy* of control depending on *Intrinsic Rate*.**
    - (SA is fastest :. takes control)

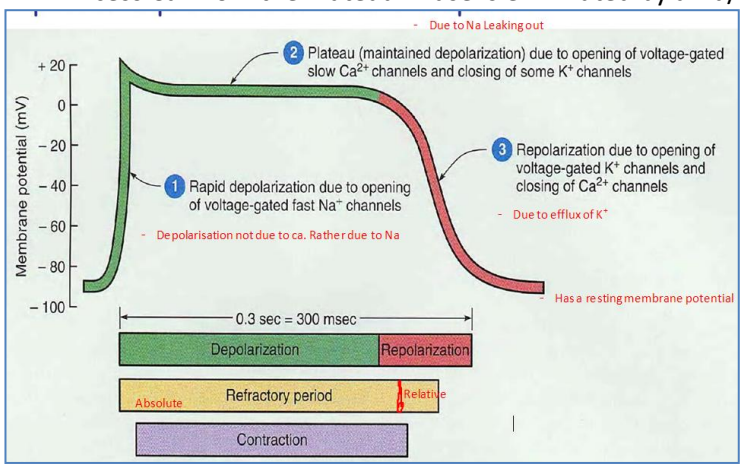


  - **The SinoAtrial (SA) Node:**
    - **= The "*PaceMaker*" of the Heart:** Unregulated Rate: 90-100bpm.....however;
    - **Location:** *Posterior Wall* of the *Right Atrium* near the opening of the *Superior Vena Cava*
  - **The AtrioVentricular (AV) Node:**
    - **2nd in Command:** Slower than the SA Node: 40-60bpm
    - **Location:** Inferior portion of the InterAtrial Septum; Directly above the TriCsupid Valve.
    - **Function:** Delays SA Node impulse Approx. 100ms → Bundle Branches;
      - →Allows Atrial emptying before Ventricular Contraction
  - **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
    - **Function:** Serves as the only connection between the 2 Atria & 2 Ventricles.

  - ***Conduction Path:***
    - **SA node→**
      - →AV node (delays signal – ensures coordinated contraction)
      - →Bundle of His (further delay 0.04secs)
      - →R and L bundle branches
      - →Purkinji fibres

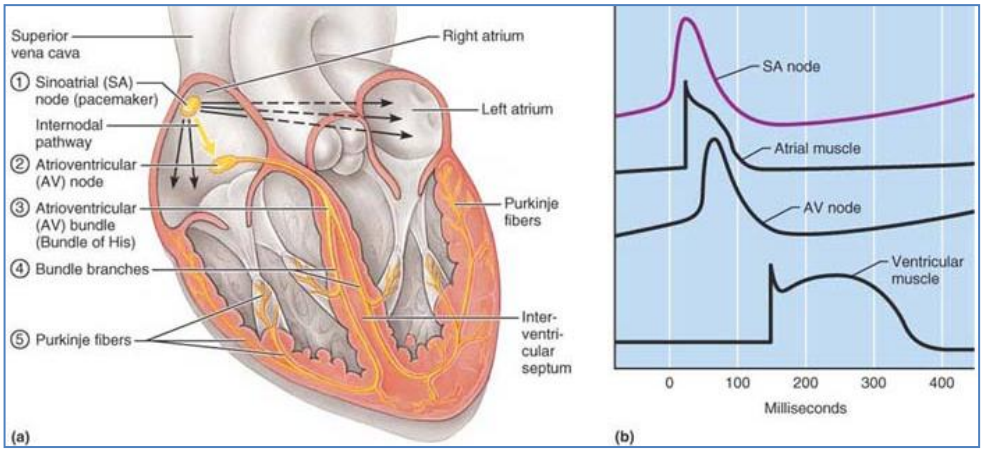


- **Contractile:**
  - **Fast 'Non-Pacemaker' Action Potentials**
  - **Have stable membrane potentials.**
    - **Depolarisation:**
      - Conductile AP → Opens Fast Na^+ Channels → Massive Na^+ influx →Depolarisation
    - **Plateau:**
      - Fast Na^+ channels close; Voltage-Gated Ca^+ channels to open →
        - →Ca Influx + Ca release from Sarcoplasmic Reticulum
          - ↑$[C^{a+}]$ causes muscular contraction.
        - (Plateau is balanced by Ca^+ influx & K^+ efflux)
    - **Repolarisation:**
      - Influxing Ca^+ channels close; but the effluxing K^+ channels remain open;
    - **Excess Ions?:**
      - Excess Na^+ & K^+ deficit is dealt with by the Na/K-ATPase.
      - Excess Ca^+ from the Plateau Phase is eliminated by a Na/Ca Exchanger.



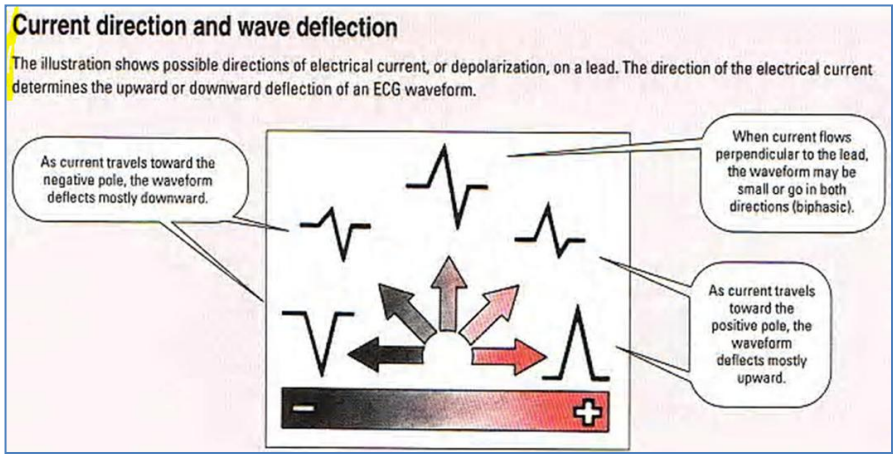
  - **The Purkinje Fibres:**
    - **= Specialised Myocytes with *very few myofibrils* (NOT contractile).**
      - Conductile; but...Resembles Ventricular Myocytes
      - Capable of Spontaneous Depolarisation – 15bpm
    - **Location:**
      - The Inner Ventricular Walls of the Heart – just below the Endocardium
    - **Role in Conduction Network:**
      - Impulse conduction from L & R Bundle Branches to the *Ventricles*;

  - **Atrial & Ventricular Myocytes:**
    - Cells with many ***myofibrils*** (contractile proteins)
    - Produce the contraction necessary for heart function.



ElectroCardioGrams (ECG):

- Recording of all Action Potentials by Nodal & Contractile Cells in the heart at a given time.
    - NB: It IS NOT a single action potential.
    - NB: 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:**



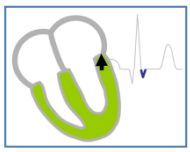
- **ECG Waves:**
    - **P – Wave:**
        - Depolarisation of the Atria
        - Presence of this waves indicates the SA Node is working

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

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

    - **R – Wave:**
        - Ventricular Depolarisation
        - 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.

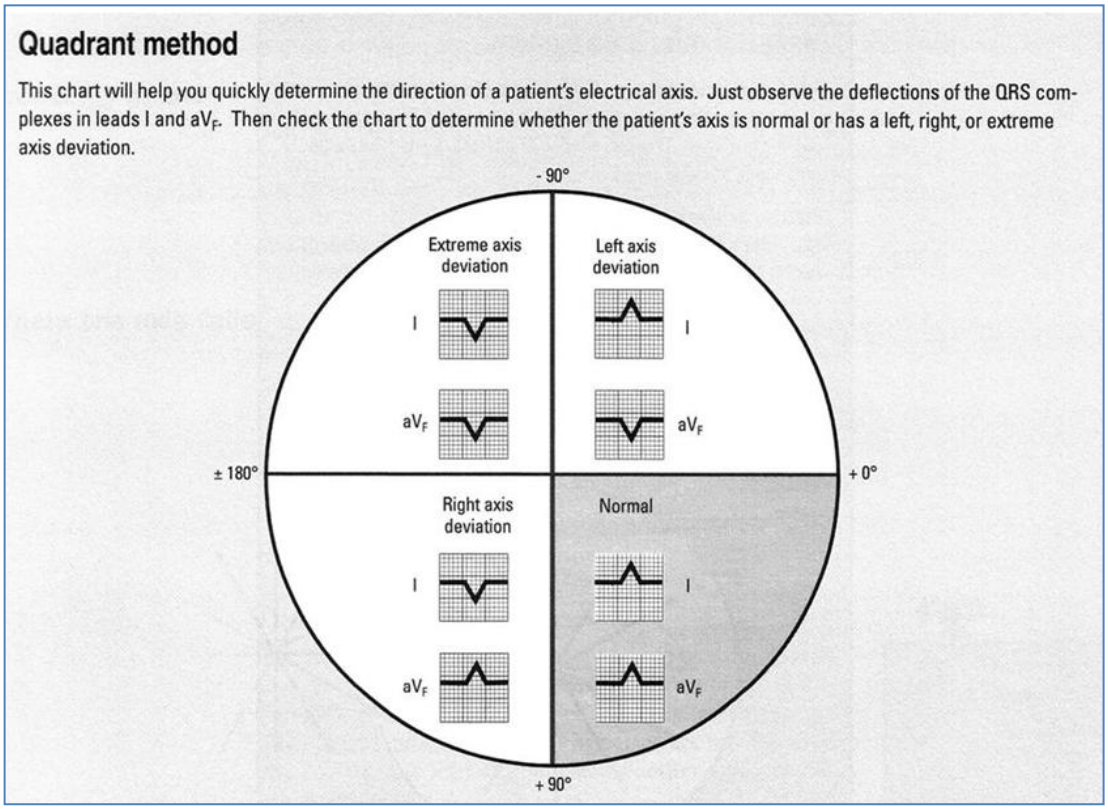


- **ST – Segment:**
  - Ventricular Contraction is occurring at this time.
    - Due to the lag between excitation & contraction.

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

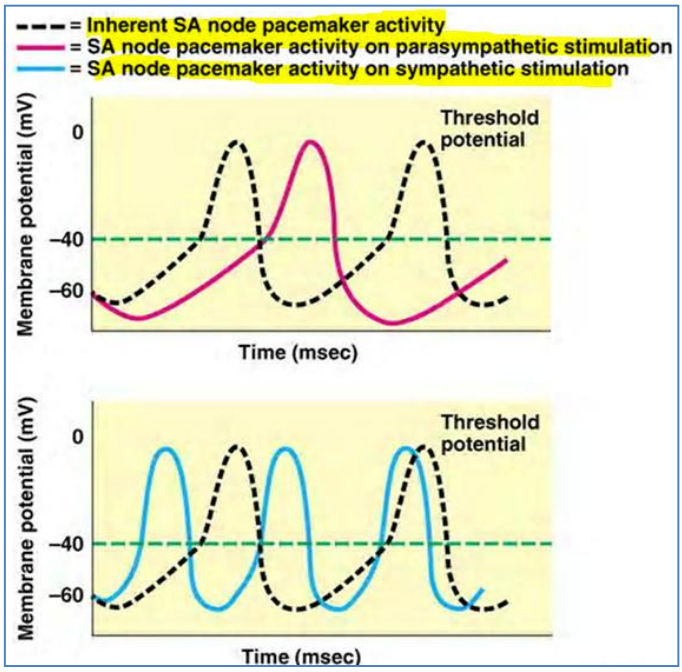
<u>The Heart's Electrical Axis:</u>

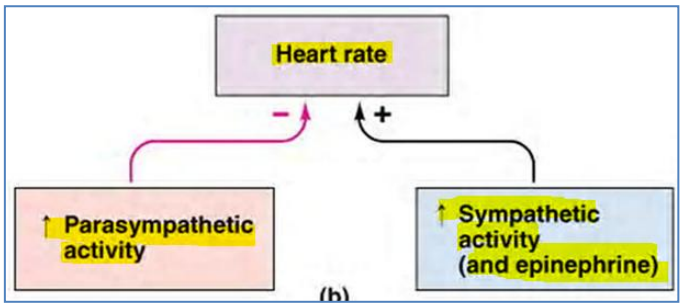
- Refers to the general direction of the heart's depolarisation wavefront (or 'mean electrical vector') in the frontal plane.
- It is usually oriented in a 'Right Shoulder to Left Leg' direction.
- **Determining The Electrical Axis From an ECG Trace:**
  - 3 Methods:
    - Quadrant Method (the one you're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

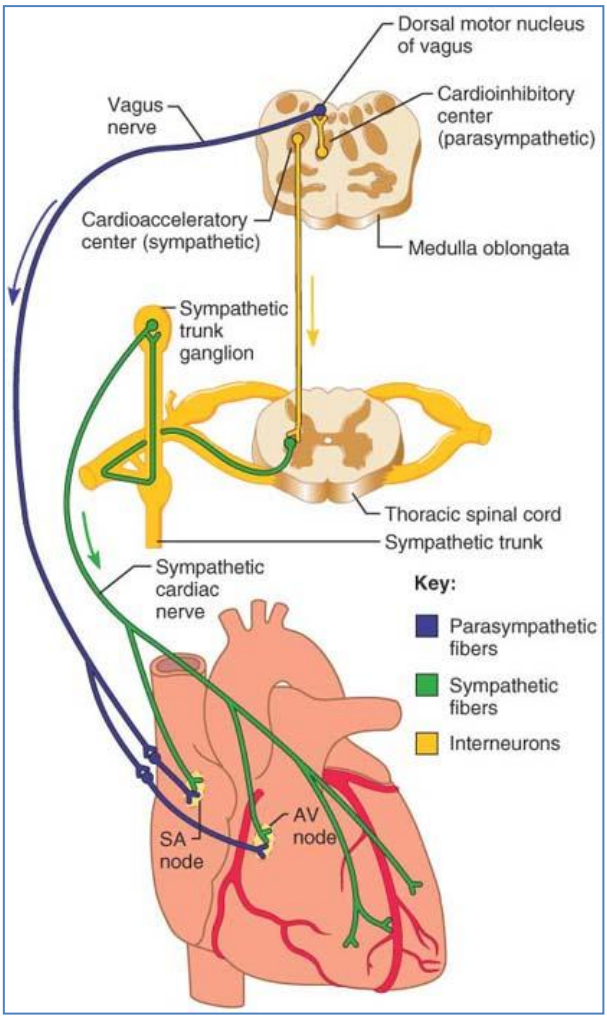


Effects of the Autonomic Nervous System:

- **Parasympathetic NS:**
  - **Innervates SA & AV Nodes →**
    - ↓Heart Rate
- **Sympathetic NS:**
  - **Innervates the SA & AV Nodes & Ventricular Muscle (& also via Noradrenaline).**
    - ↑Heart Rate
    - ↑Contractility



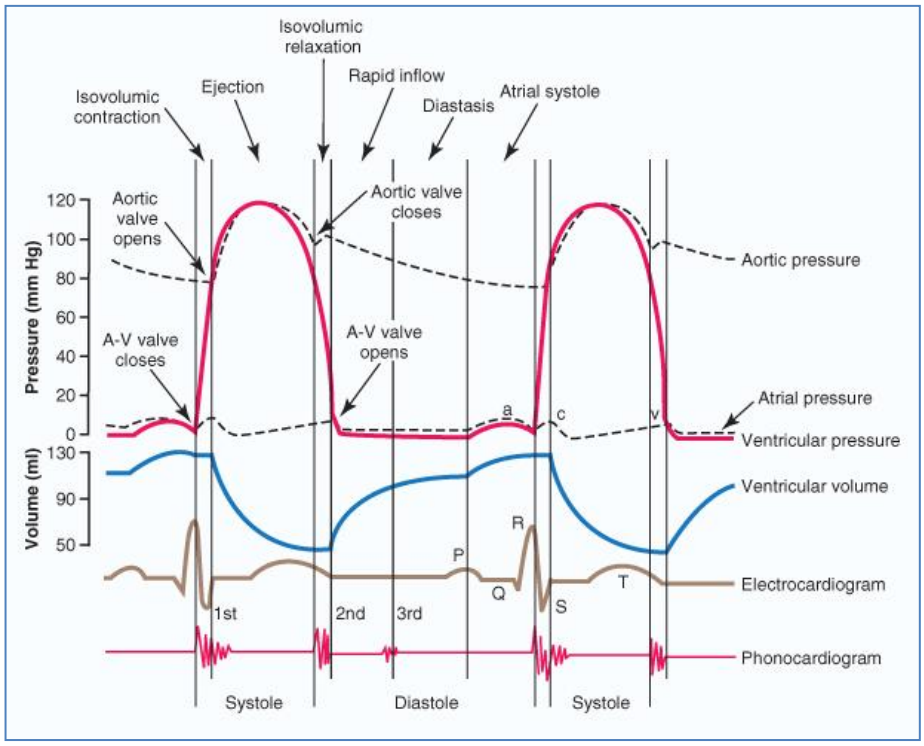




Mechanical Events of The Cardiac Cycle

Terms:

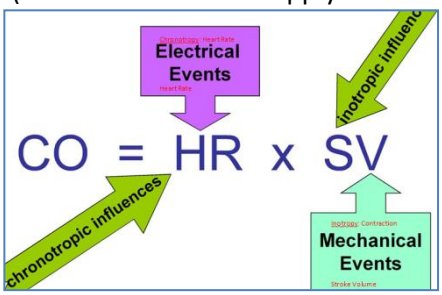
- **Systole =** *Myocardial Contraction*
- **Diastole =** *Myocardial Relaxation*
- **Stroke Volume =** Output of Blood from the Heart *Per Contraction* (≈80mL of blood)
- **Heart Rate =** *#Heart Beats/Minute*
- **Cardiac Output:**
  - Volume of Blood Ejected from the Heart *Per Minute* (Typically ≈5L/min)
  - ***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/Pulm.Art.
  - (↑Afterload = ↓SV due to ↓ejection time)



- **1. Atrial Systole/Ventricular Filling (Diastole):**
- **2. Ventricular Systole:**
  - **a) AV Valves Close:**
    - Ventricular Pressure Exceeds Atrial Pressure → AV Valves shut
  - **b) Semilunar Valves Open:**
    - Ventricular Pressure Exceeds Aortic/Pulm Pressure → Blood Ejected
  - **c) Semilunar Valves Close:**
    - Ventricular Pressure then falls Below Aortic/Pulm Pressure → Semilunar Valves Close.
- **3. Ventricular Diastole:**
  - Ventricles relax → Ventricular Pressure falls below Atrial Pressure → AV-Valves Open:
    - Blood → from Atria into Ventricles
    - (NB: Passive filling from venous return is responsible for 70% of ventricular filling.)

CardioDynamics:

- **Cardiac Output:**
  - **Determined by 2 Things:**
    - 1. Stroke Volume....&
    - 2. Heart Rate
  - **Average CO ≈ 5L/min** (ie. The entire blood supply circulates once per minute)



- **Heart Rate:**
  - Depends on Tissue-Satisfaction with Nutrients & O_2.
  - **Terms:**
    - **BradyCardia:** HR *Slower* than normal. (too fast → stroke volume & CO suffers)
    - **TachyCardia:** HR *Faster* than normal.
  - **Regulation of HR:**
    - **Autonomic Nervous System:**
      - **Parasympathetic: (Vagus Nerve)**
        - *Decrease* Heart Rate (-ve Chronotropic Effect)
        - *Increase* AV-Node Delay (-ve Dromotropic Effect)
        - NB: ONLY A TINY EFFECT ON CONTRACTILITY
      - **Sympathetic: (Sympathetic Chains)**
        - *Increase* Heart Rate (+ve Chronotropic Effect)
        - *Increase* Force of Contraction (+ve Inotropic Effect).
    - **Reflex Control:**
      - **Bainbridge Reflex (Atrial Walls)**:
        - ↑Venous Return → ↑Heart Rate
        - Responsible for 40-60% of HR increases.
      - **BaroReceptor Reflex (Aortic & Carotids):**
        - ↑BP → ↓HR & ↓Contractility (+ Vasodilation)
      - **ChemoReceptor Reflex:**
        - ↓Low O_2 or ↑CO_2 in Peripheral-Tissue → ↑HR & ↑Resp. Rate

- **Stroke Volume:**
  - Blood output per heart-beat.
  - **Stroke Volume = End Diastolic Volume – End Systolic Volume**
  - **:. SV is ↑by:**
    - ↑ Ventricular Filling Time (Duration of Ventricular Diastole)
    - ↑Venous Return
    - ↓Arterial BP (Higher → harder to eject blood → ESV Increases)
  - **NB: "*Frank Starling Mechanism*":**
    - **↑ Preload → ↑Contractility → ↑Stroke Volume**

Control of Circulation (Haemodynamics & BP Regulation)

Resistance:

- **3 Factors Influencing Resistance:**
  - **1. Blood Viscosity** (Fairly Constant)
  - **2. Total Vessel Length** (Fairly Constant)
  - **3. Vessel Diameter** (Highly Variable)

Relationship Between *Flow, Pressure & Resistance:*

- **Flow is:**
  - 1. Directly Proportional to Pressure Gradient
  - 2. Inversely Proportional to Resistance
- **Therefore:**

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

Effects of Vasomotion on *Rate* & *Velocity of Flow:*

- **Changes Vessel *Diameter*:**
  - The ***Flow Rate*** is directly ***proportional to*** the ***4th Power*** of the ***Vessel Diameter***.
  - Ie. *Small changes* in diameter → *Large changes* in Flow Rate (by x^4).

Factors Influencing Blood Pressure (Long Term):

- **Cardiac Output**
- **Peripheral Resistance**
- **Blood Volume**

:. BP = Cardiac Output X Total Peripheral Resistance

Types of Blood Pressures:

- **Systolic:** Peak Aortic pressure during ventricular systole.
- **Diastolic:** Lowest Aortic pressure during ventricular diastole.
- ***Pulse Pressure:**
  - **= Systolic Pressure - Diastolic Pressure**
  - (Eg. 120mmHg – 80mmHg)
  - Normal = 40mmHg
- ***Mean Arterial Pressure (MAP):**
  - MAP = Diastolic Pressure + 1/3(Pulse Pressure)
  - *The Pressure that *Propels Blood to the Tissues* – maintains *Tissue Perfusion* (see below sections).

Control of MAP:

- **3 Main Regulators:**
  - **1. Autoregulation (@ the Tissue Level):**
    - 'Automatic Vasodilation/constriction @ the tissue relative to metabolic requirements.'
  - **2. Neural Mechanisms:**
    - **Vasomotor Centres (Medulla):**
      - Baroreceptors & Chemoreceptors
    - **Autonomic Nervous System:**
      - Sympathetic → ↑HR & Contractility → ↑MAP
      - Parasympathetic → ↓Heart Rate → ↓MAP
  - **3. Endocrine Mechanisms (Kidney Level):**
    - ****Antidiuretic Hormone (ADH) – AKA. Vasopressin:**
      - ADH → Water Retention Released → ↑MAP
    - **Angiotensin II:**
      - AT-II → VasoConstriction → ↑MAP
    - **Erythropoietin:**
      - EPO → Haematopoiesis → ↑Blood Volume → ↑MAP
    - **Natriuetic Peptides (Released by the heart):**
      - ↑Stretch on Heart → NP Release → ↑Diuresis → *Reduces BP & Volume.*

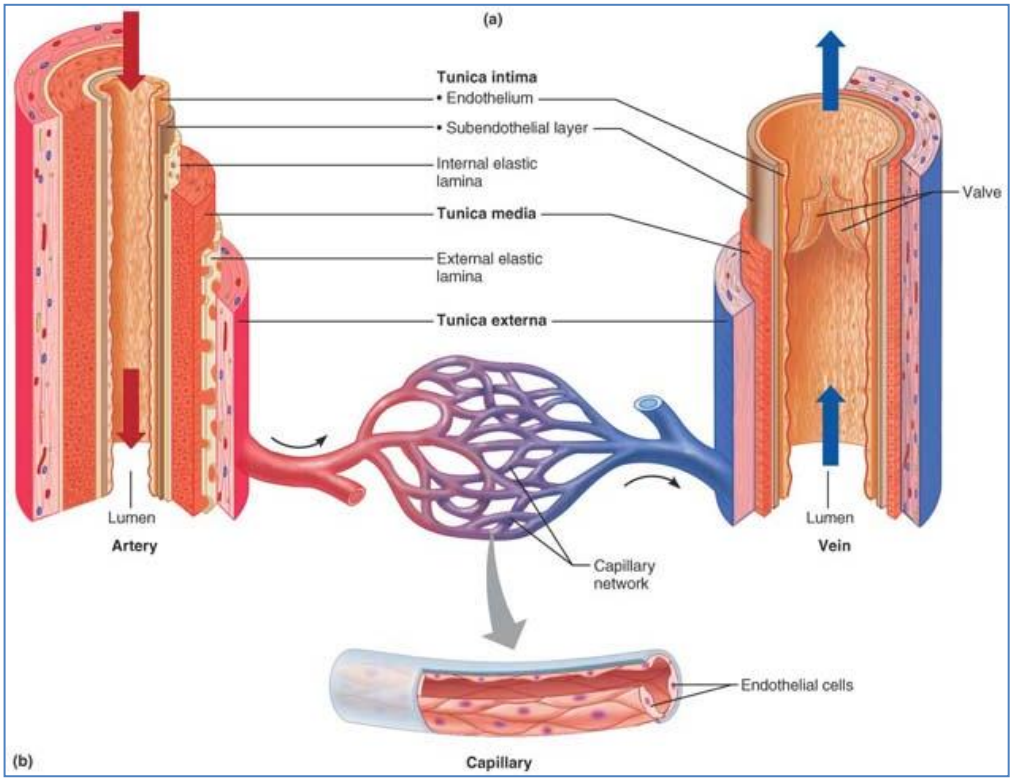
Blood Vessels

Introduction to Blood Vessels:

- **3 Classes:**
  - **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
  - **Capillaries – Intimate contact with tissue → facilitate cell nutrient/waste transfer**
    - Vascular Shunt
    - True Capillaries
  - **Veins – Carry blood back to the heart**
    - Post-Capillary Venule — (Union of capillaries)
    - Small Veins — (Capacitance Vessels – 65% of body's blood is venous)
    - Large Veins — (Capacitance Vessels – 65% of body's blood is venous)

Blood Vessel Structure:

- **3-Layered Wall:**
  - **Tunica Intima:**
    - Ie. The layer in *intimate* contact with the blood (luminal)
    - Consists of ***The Endothelium*** (Simple Squamous Epthelium)
  - **Tunica Media:**
    - Middle....& Thickest layer (*Smooth Muscle & Elastin*)
  - **Tunica Externa:**
    - Outermost Layer (Loose *collagen fibres*)
    - (NB: Also Contains Nerve Fibres, Lymphatics, and Vasa Vasorum (In larger vessels))



Fluid Movements Across a Vessel:

- **Determined by the balance of 2 forces:**
  - **1. Capillary Hydrostatic Pressure:**
    - Capillary blood pressure.
  - **2. Colloid Osmotic Pressure:**
    - Balance of Blood Osmolarity vs. Tissue Osmolarity
- ***(:. Net Filtration Pressure = Net Hydrostatic Pressure – Net Osmotic Pressure)***

FIGURE 19.20 **Major arteries of the systemic circulation. (a)** Schematic flowchart. **(b)** Illustration, anterior view.

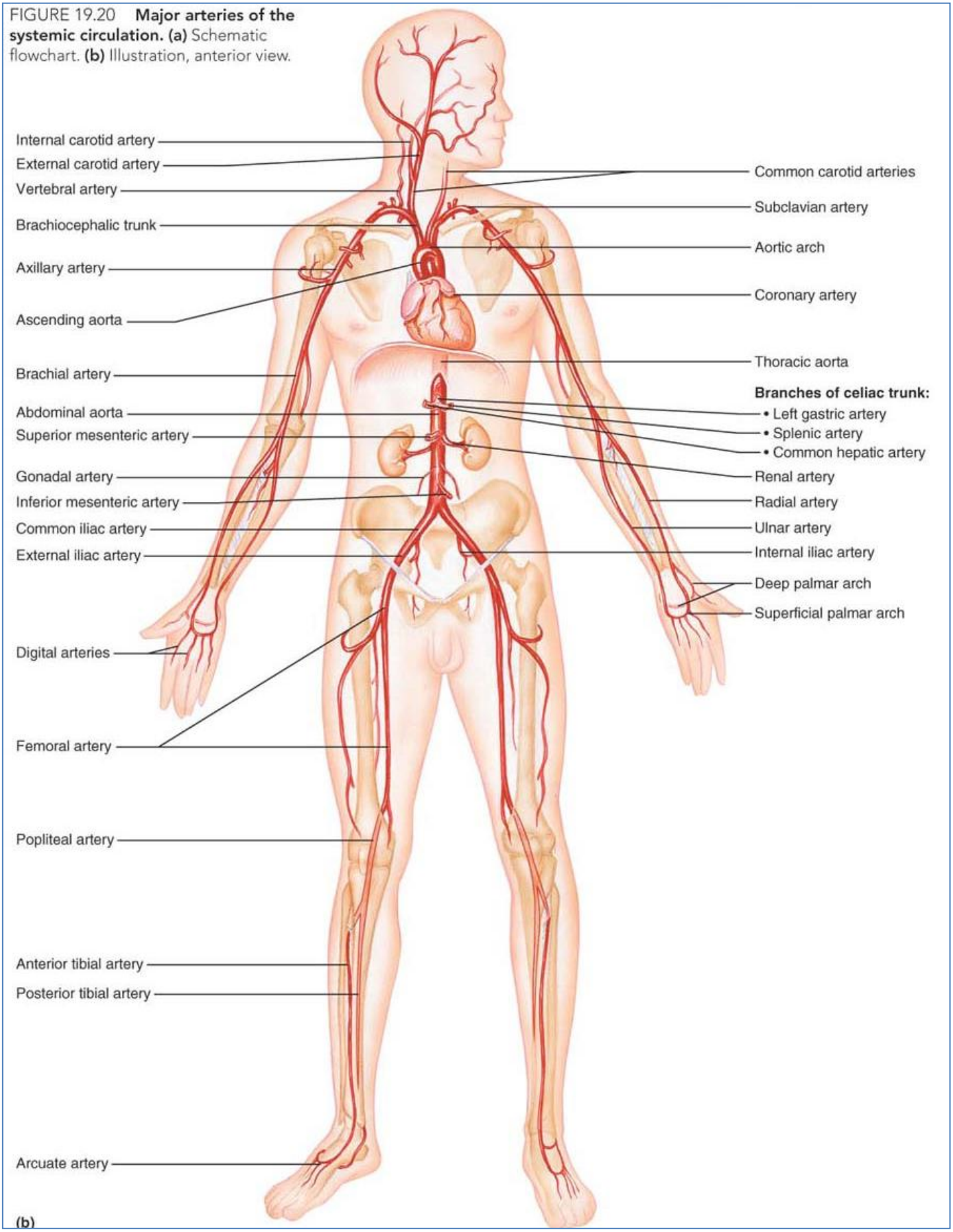
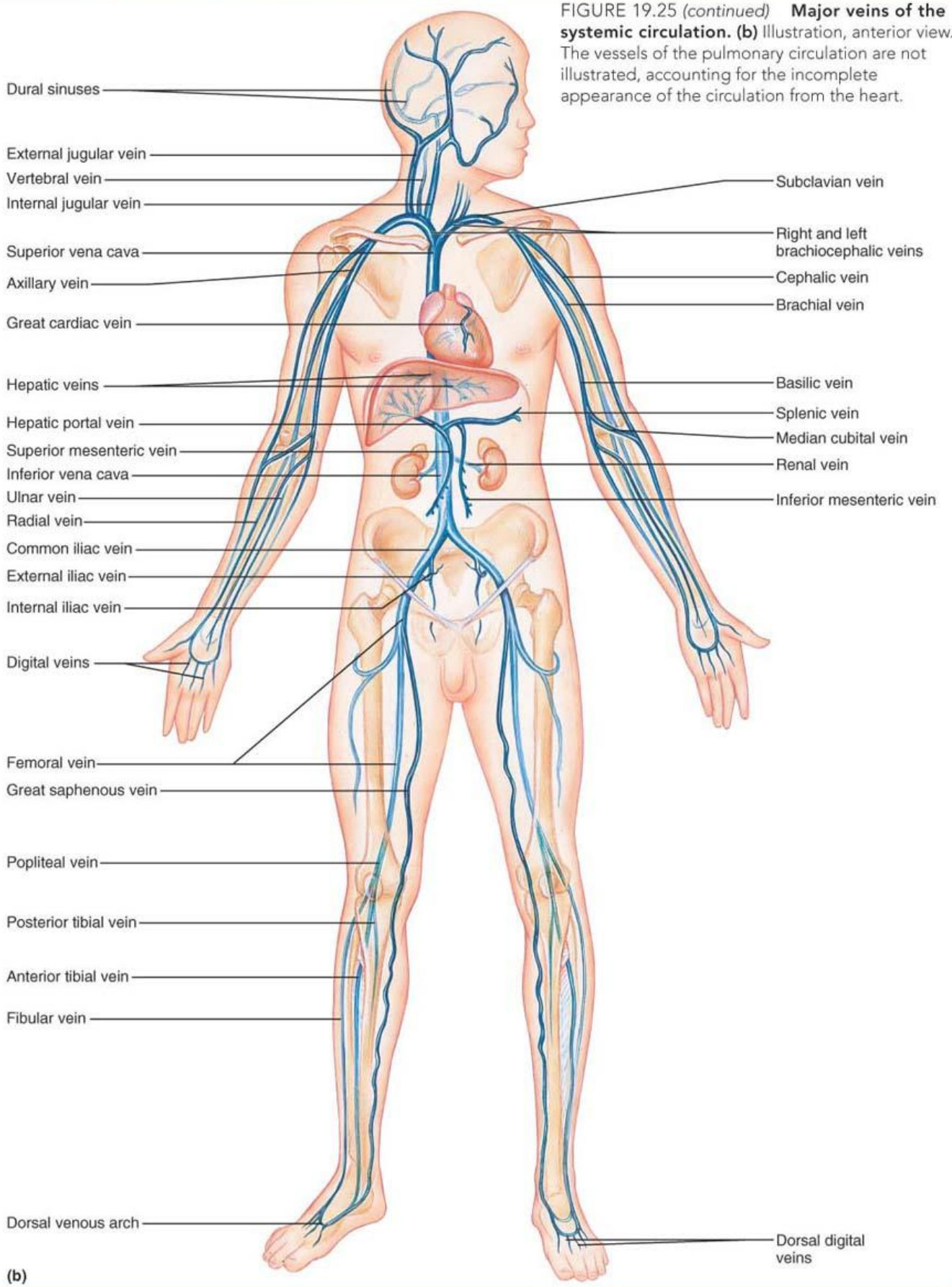


TABLE 19.9 *(continued)*

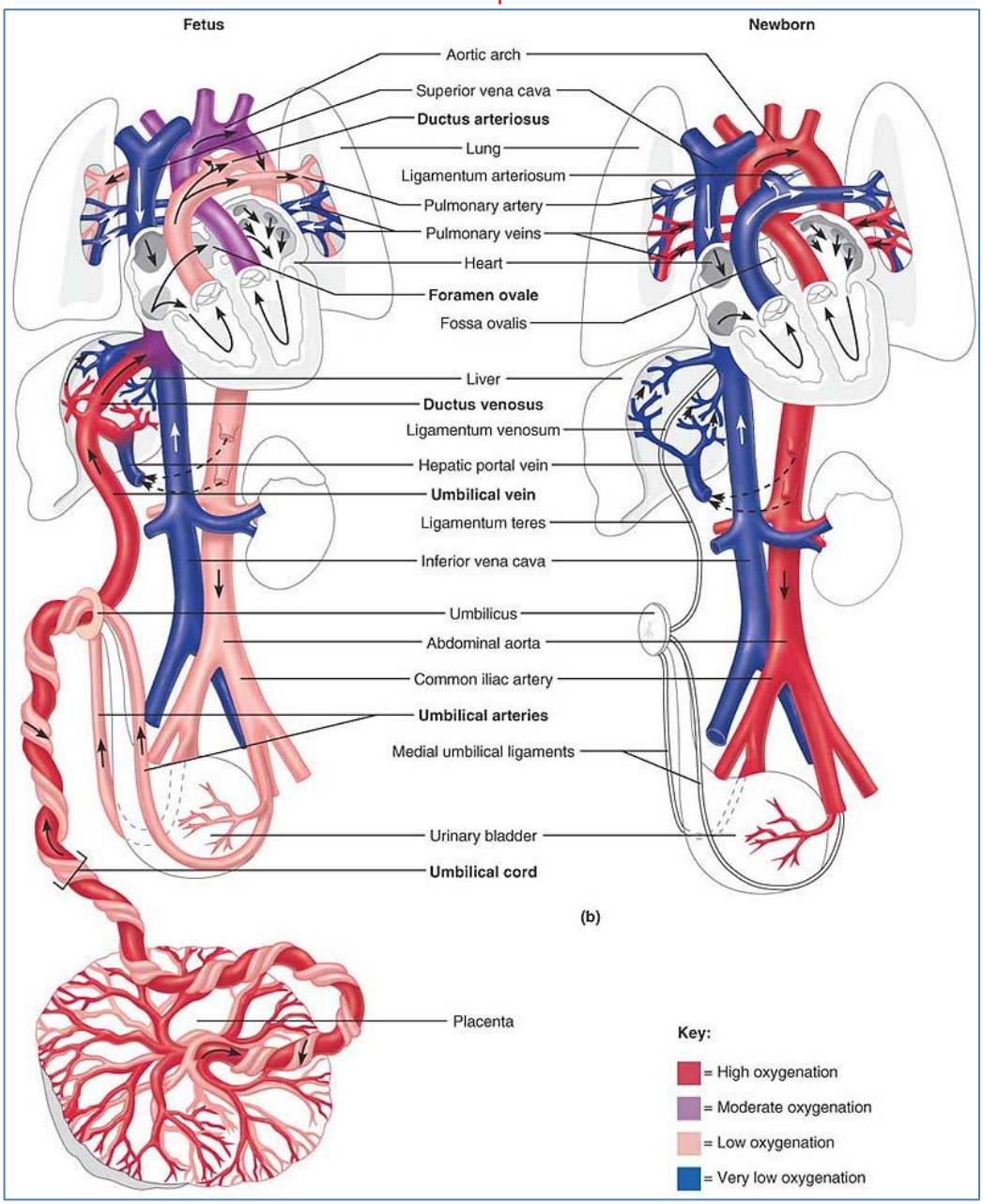
FIGURE 19.25 *(continued)* **Major veins of the systemic circulation. (b)** Illustration, anterior view. The vessels of the pulmonary circulation are not illustrated, accounting for the incomplete appearance of the circulation from the heart.



(b)

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.
    - NB: The Foramen Ovale can take up to 6 months to close.

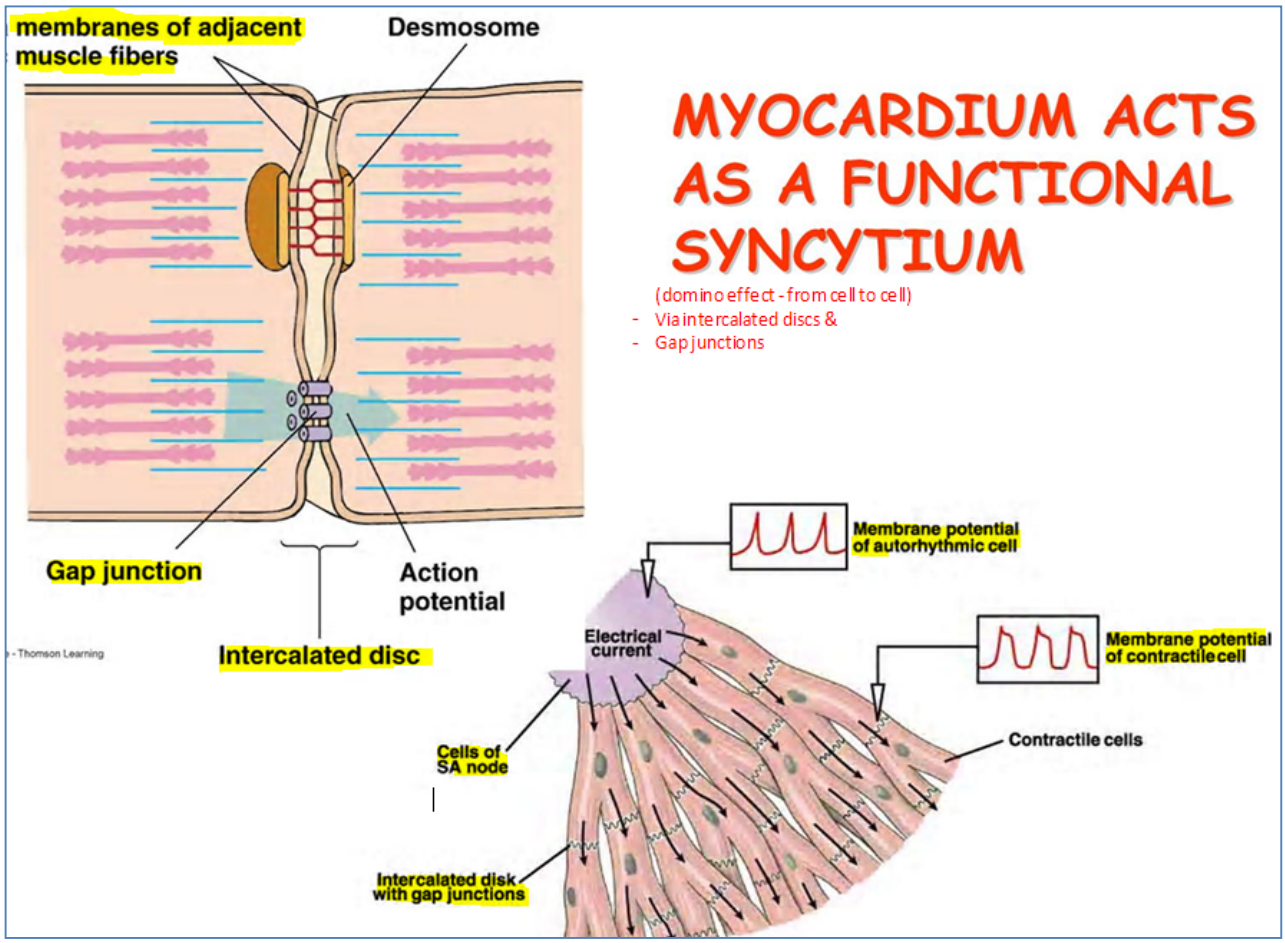


CardioVascular Medicine Notes
Electrophysiology & ECGs

The Heartbeat:

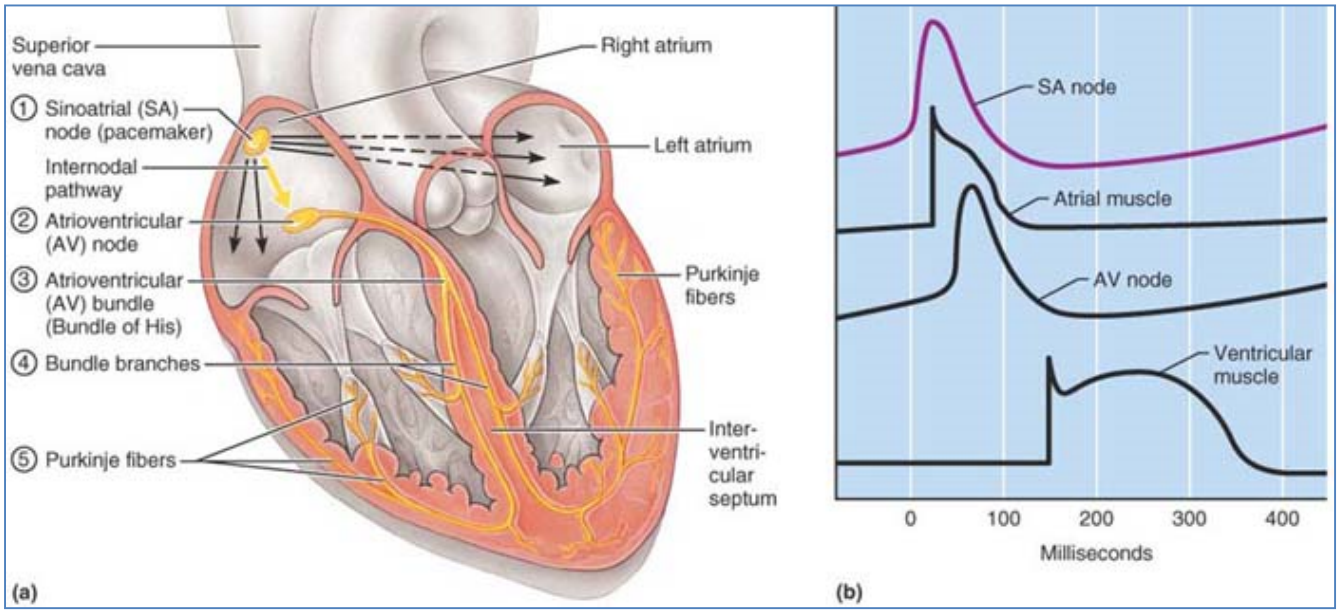
- Heart is a Muscle & Requires:
  - O_2
  - Nutrients, &
  - **Action Potentials**; to function.
- **However,** these neural signals don't come from the brain;
  - Rather, the heart has its ***own*** conduction systems.
    - These systems ***allow it to contract autonomously***
  - Hence why a *transplanted heart* still operates (if provided with O_2 & 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



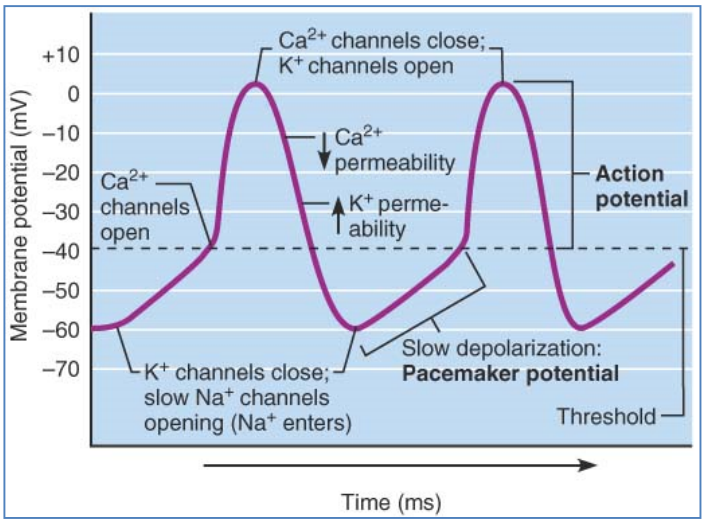
The Heart's Conduction Systems:

- NB: The Amplitude & Steepness of an action potential are important determinants of propagation velocity.

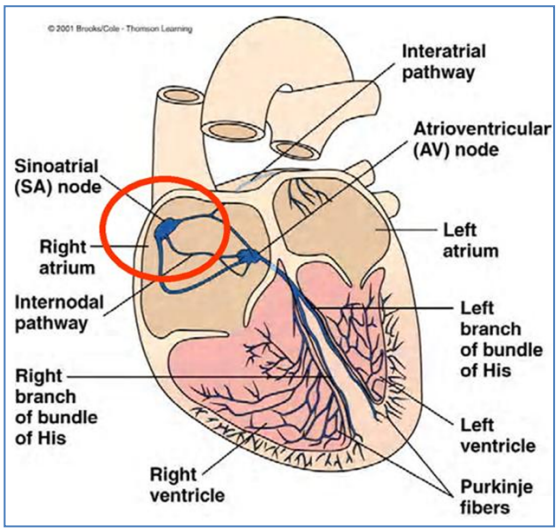


2 Types of Cardiac Muscle Cells:

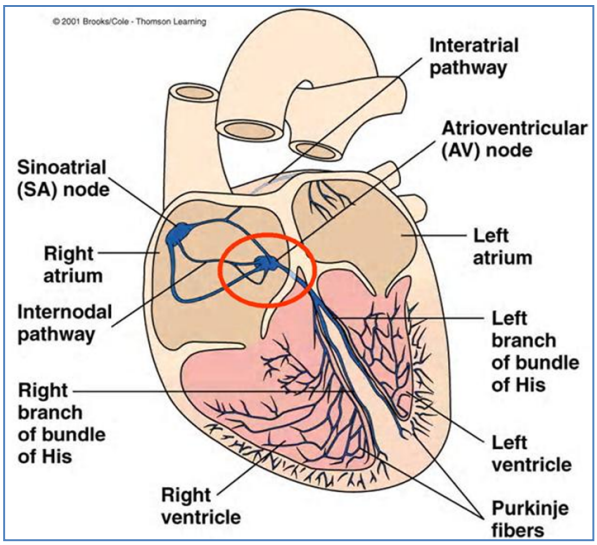
- **Conductile/Nodal: (*Intrinsic*)**
  - Slow 'Pacemaker' Action Potentials
  - Have *Spontaneous Electrical Activity* – Cannot Maintain a Resting Membrane Potential
    - Spontaneously Depolarises to Threshold
      - This gradual depolarisation is called a '*Prepotential*'.
      - Due to Leaky Na^+ Membrane Ion Channels
      - Therefore – Firing Frequency Depends on Na^+ Movement
    - Depolarisation:
      - Once Threshold is reached, Ca^{2+} 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
    - **I.e:** Na^+ brings to threshold, but Ca^+ is responsible for Depolarisation.
  - With a *Heirarchy* of control over the heart.
    - Heirarchy based on *natural intrinsic rate*. (fastest node (SA node) takes control)



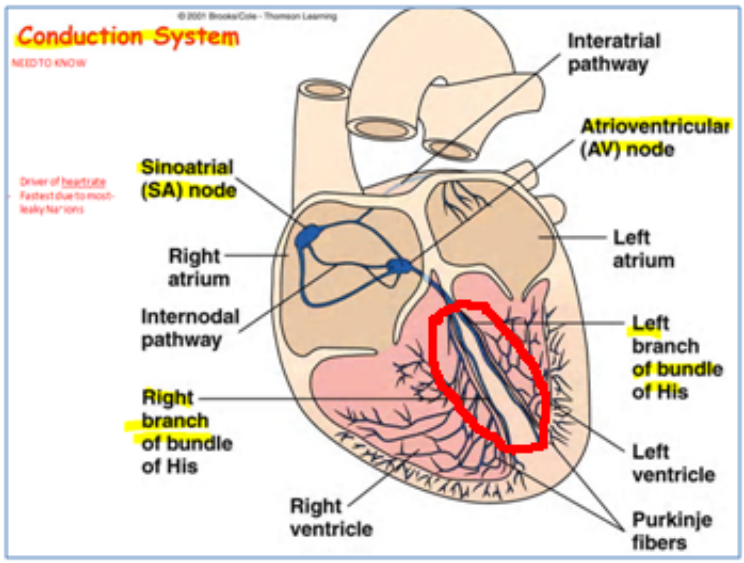
- **The SinoAtrial (SA) Node:**
  - The region that generates impulses at the highest frequency.
    - The "*PaceMaker*" - Driver of Heart Rate
    - Unregulated Rate: 90-100bpm.....however;
      - Parasympathetic NS lowers heart rate.
        - Keeps Normal Resting Heart Rate at 70bpm
      - Sympathetic NS raises heart rate.
    - Takes 50ms for Action-Potential to reach the AV Node.
  - Location:
    - Embedded in the *Posterior Wall* of the *Right Atrium* near the opening of the *Superior Vena Cava*
  - Nature of Action Potentials:
    - Continually Depolarising 90-100bpm
  - Role in Conduction Network:
    - Sets the pace for the heart as a whole.
  - Portion of Myocardium Served:
    - Contracts the Right & Left Atrium



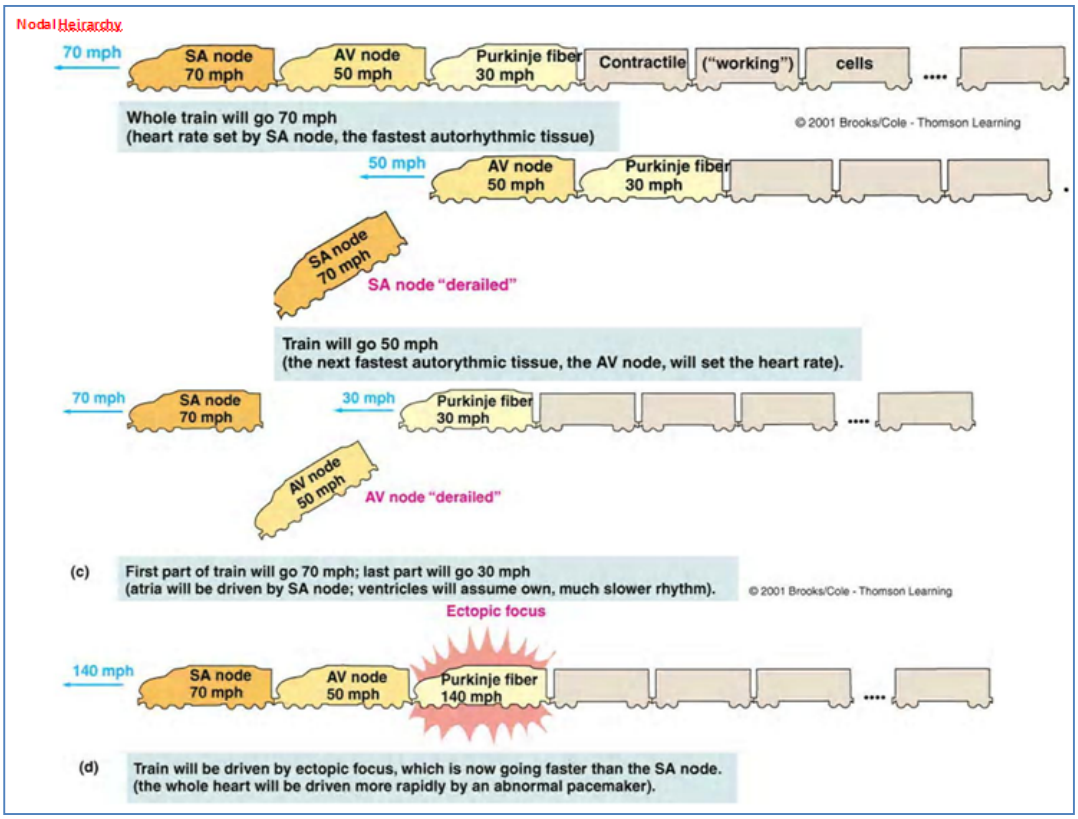
- **The AtrioVentricular (AV) Node:**
  - Location:
    - Inferior portion of the InterAtrial Septum; Directly above the TriCsupid Valve.
  - Nature of Action Potentials:
    - Continually Depolarising – but slower than the SA Node.
      - 40-60bpm
  - Role in Conduction Network:
    - To delay the 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:
    - Part of a conducting pathway; serves to pass on the SA Node Impulses to the Purkinje Fibres (which supply the Ventricular Walls)



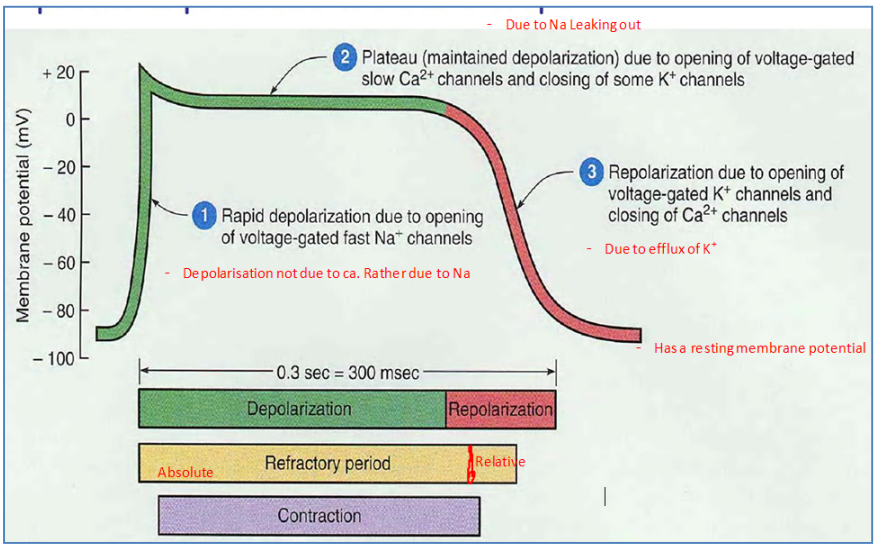
- **The Bundle Branches/AV Bundle Branches/Bundles of His:**
  - Location:
    - Fork of branches – Superior Portion of InterVentricular Septum
  - Nature of Action Potentials:
    - Continually Depolarising – Slower than AV & SA Nodes
      - 20-40bpm
  - Role in Conduction Network:
    - Serves as 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.
  - NB: Left Bundle is bigger due to larger L-Ventricle.



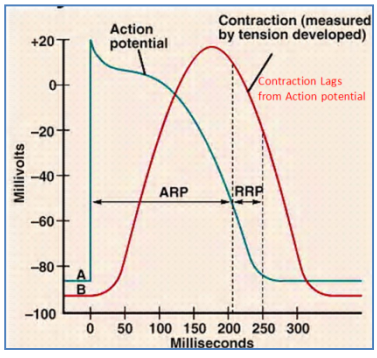
Nodal Heirarchy:



- **Contractile:**
  - Fast 'Non-Pacemaker' Action Potentials
  - Have stable membrane potentials.
    - Resting MP:
      - Na^+ & Ca^+ channels are closed.
      - Any +ve change to MP causes Fast Na^+ channels to open → +ve feedback → Threshold
    - Depolarisation:
      - If MP reaches threshold, all Fast Na^+ channels open;
      - Massive influx of Na^+ into cell
      - Membrane depolarises
    - 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")
          - This increased myoplasmic Ca^+ causes muscular contraction.
        - Plateau is sustained by influx of Ca^+, balanced by efflux of K^+ ions
    - Repolarisation:
      - 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 Repolarises & most of the K^+ channels close.
    - Excess Ions??
      - Excess Na^+ in the cell from depolarization is removed by the Na/K-ATPase.
      - Deficit of K^+ in the cell from repolarisation is replaced by the Na/K-ATPase.
      - Excess Ca^+ from the Plateau Phase is eliminated by a Na/Ca Exchanger.



  - NB: There is a considerable delay between Myocardial *Contraction* & the *Action Potential.*

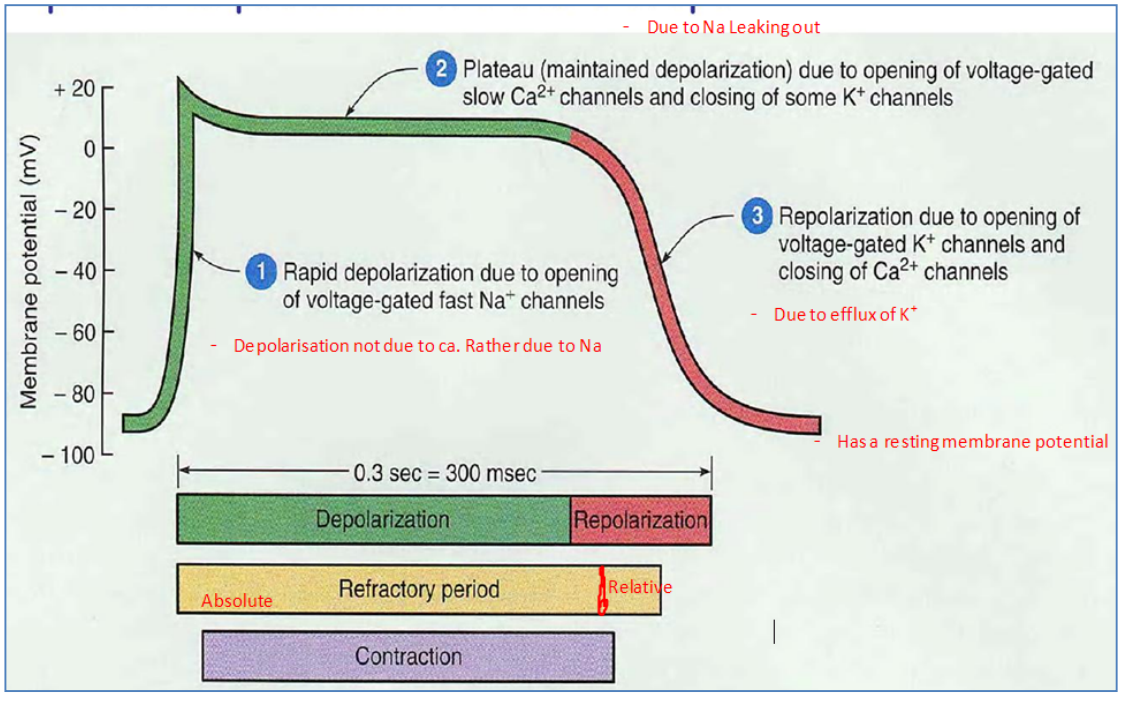


- **The Purkinje Fibres:**
  - What?:
    - Specialised Myocytes with *very few myofibrils* → don't contract during impulse transmission.
  - Location:
    - The Inner Ventricular Walls of the Heart – just below the Endocardium
    - Begin at the heart apex, then turn superiorly into the Ventricular 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.

- **Atrial & Ventricular Myocytes:**
  - Cells with many *myofibrils* (contractile proteins)
  - Produce the contraction necessary for heart function.

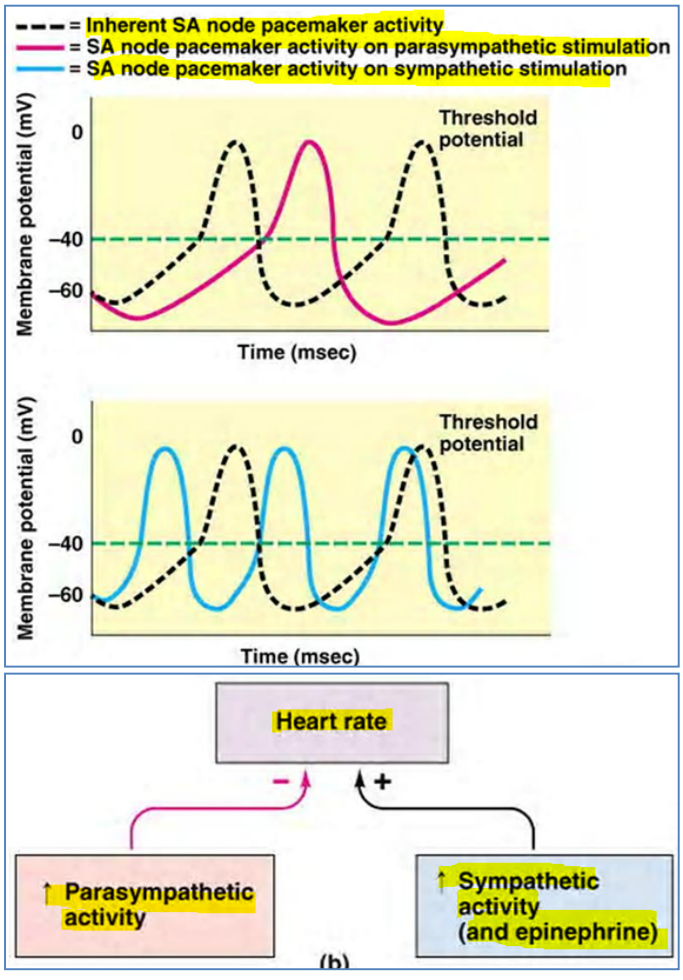
Refractory Periods:

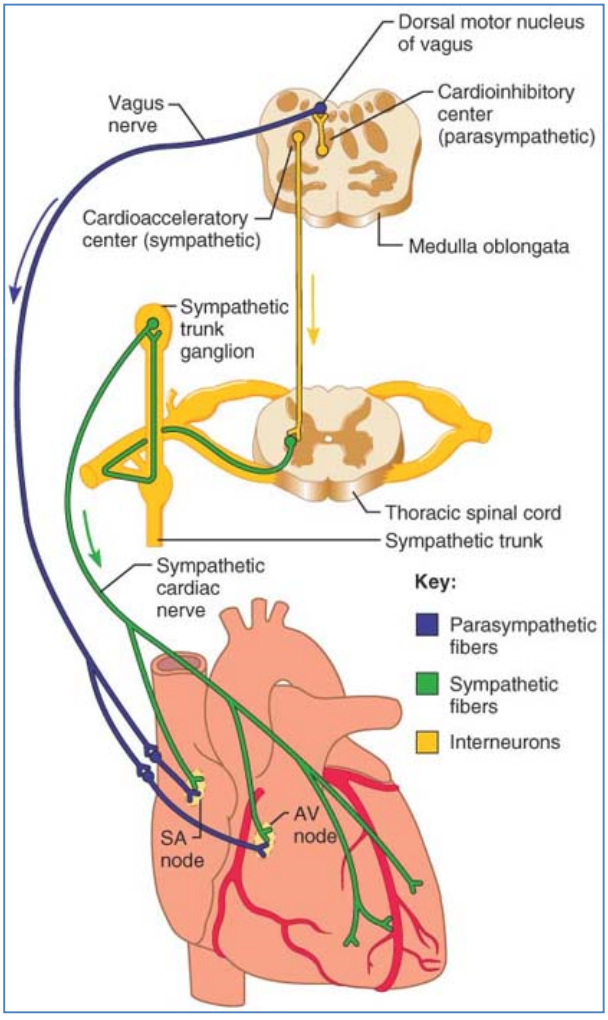
- In Cardiac Muscle, the *Absolute Refractory Period* continues until muscle relaxation;
  - Therefore summation isn't possible → tetanus cannot occur (critical in heart)
  - Ie. The depolarised cell won't respond to a 2nd stimulus until contraction is finished.
- **Absolute Refractory Period:**
  -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



Effects of the Autonomic Nervous System:

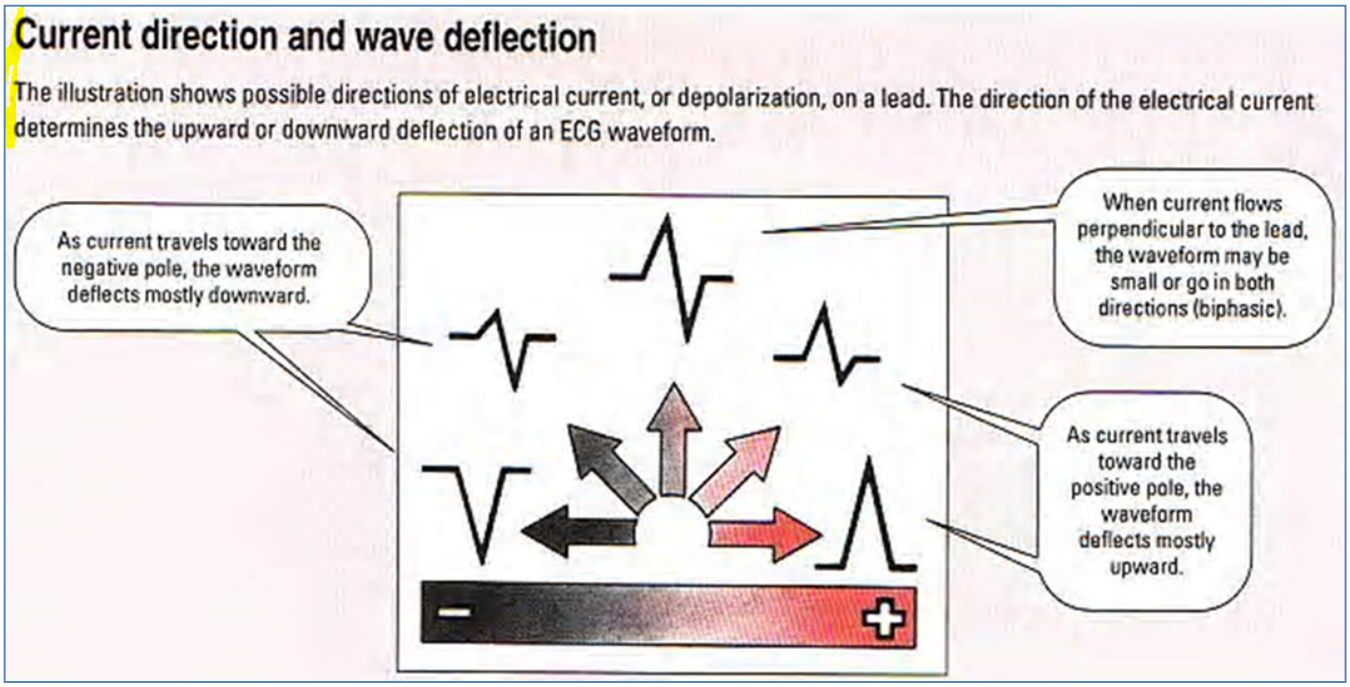
- Although the heart *can* operate on its own, It *normally* communicates with the brain via the A.N.S.
- **Parasympathetic NS:**
  - Innervates SA & AV Nodes
    - Slows Heart Rate
  - Direct Stimulation
  - Stimulation releases AcetylCholine → *Muscarinic* receptors in SA/AV Nodes →
    - Causes increased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

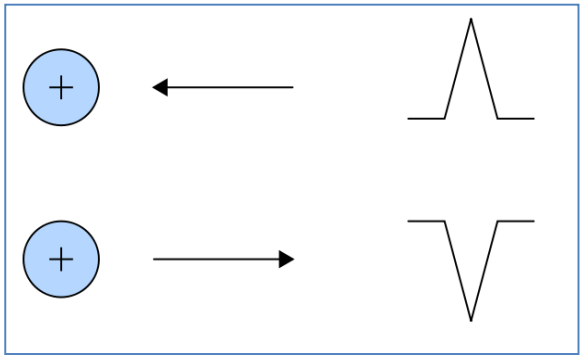


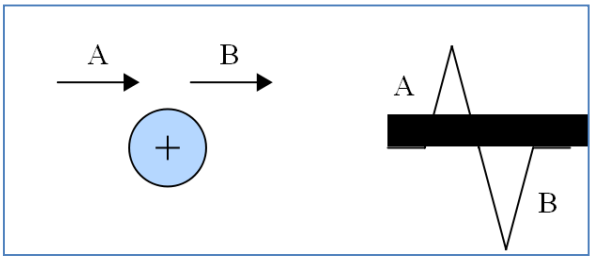


ElectroCardioGrams (ECG):

- Recording of all Action Potentials by Nodal & Contractile Cells in the heart at a given time.
  - NB: It IS NOT a single action potential.
- Measured by VoltMetres → record electrical *potential* across cells:
  - 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.
  - NB: 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.
  - A lead records electrical signals from a particular *combination of electrodes* placed at specific points on the body.
- **Graphic Output:**
  - X-axis = Time
  - Y-axis = Amplitude (voltage) – Proportional to number & size of cells.
- **Understanding Waveforms:**
  - When a Depol. Wavefront moves toward a positive electrode, a *Positive* deflection results in the corresponding lead.
  - When a Depol. Wavefront moves away from a positive electrode, a *Negative* deflection results in the corresponding lead.
  - When a Depol. Wavefront moves *perpendicular* to a positive electrode, it first creates a positive deflection, then a negative deflection.







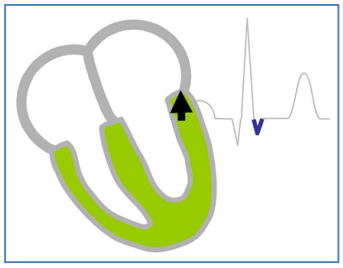
- **ECG Waves:**
  - **P – Wave:**
    - Depolarisation of the Atria
    - Presence of this waves indicates the SA Node is working

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

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

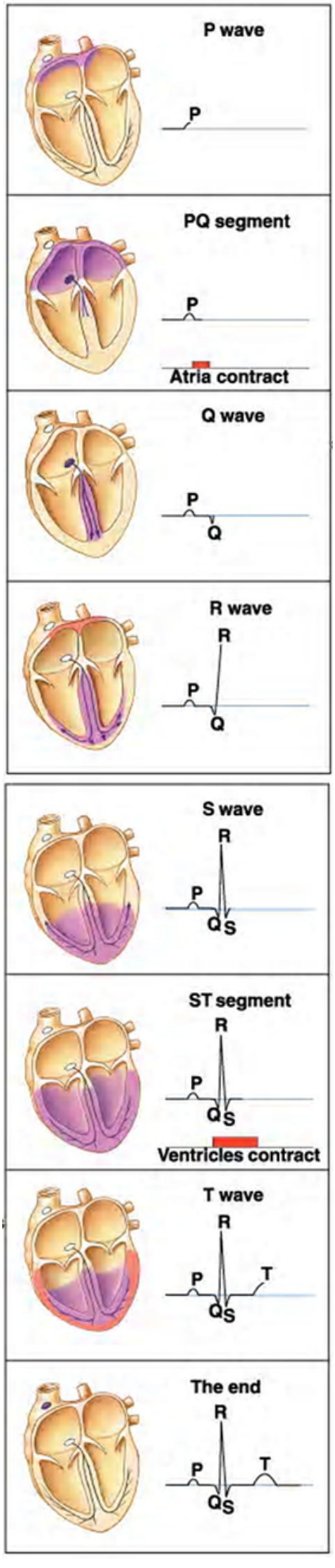
  - **R – Wave:**
    - Ventricular Depolarisation
    - Wave Direction (blue arrow) is the same as the Main Electrical Axis → Positive Deflection.
    - R-Wave Amplitude is large due to sheer numbers of depolarizing myocytes.

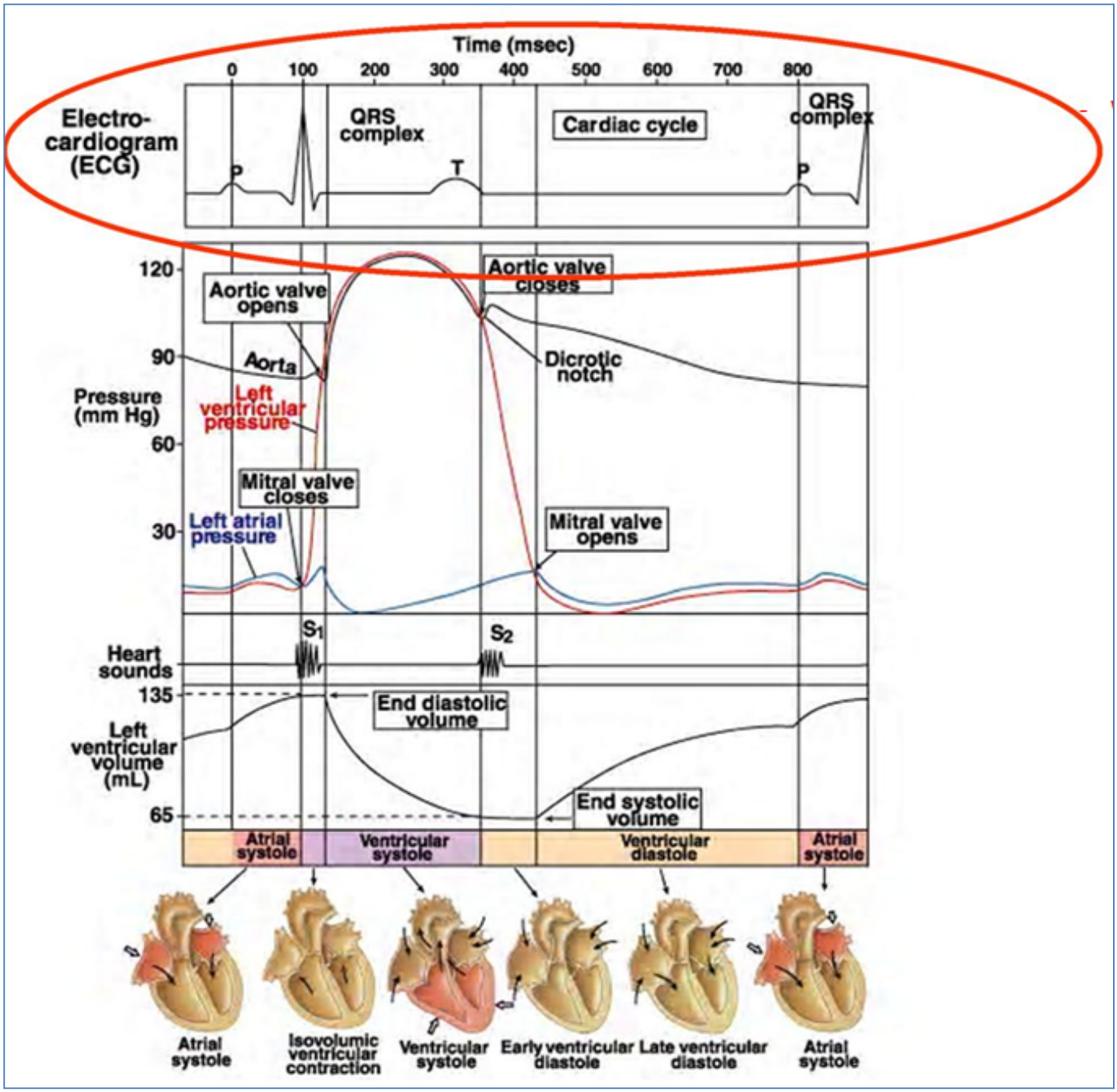
- **S – Wave:**
  - Depolarisation of the Myocytes at the last of the Purkinje Fibres.
  - Wave Direction (black arrow) opposes the Main Electrical Axis → Negative Deflection
  - This wave is not always seen.



- **ST – Segment:**
  - Ventricular Contraction is occurring at this time.
    - Due to the lag between excitation & contraction.

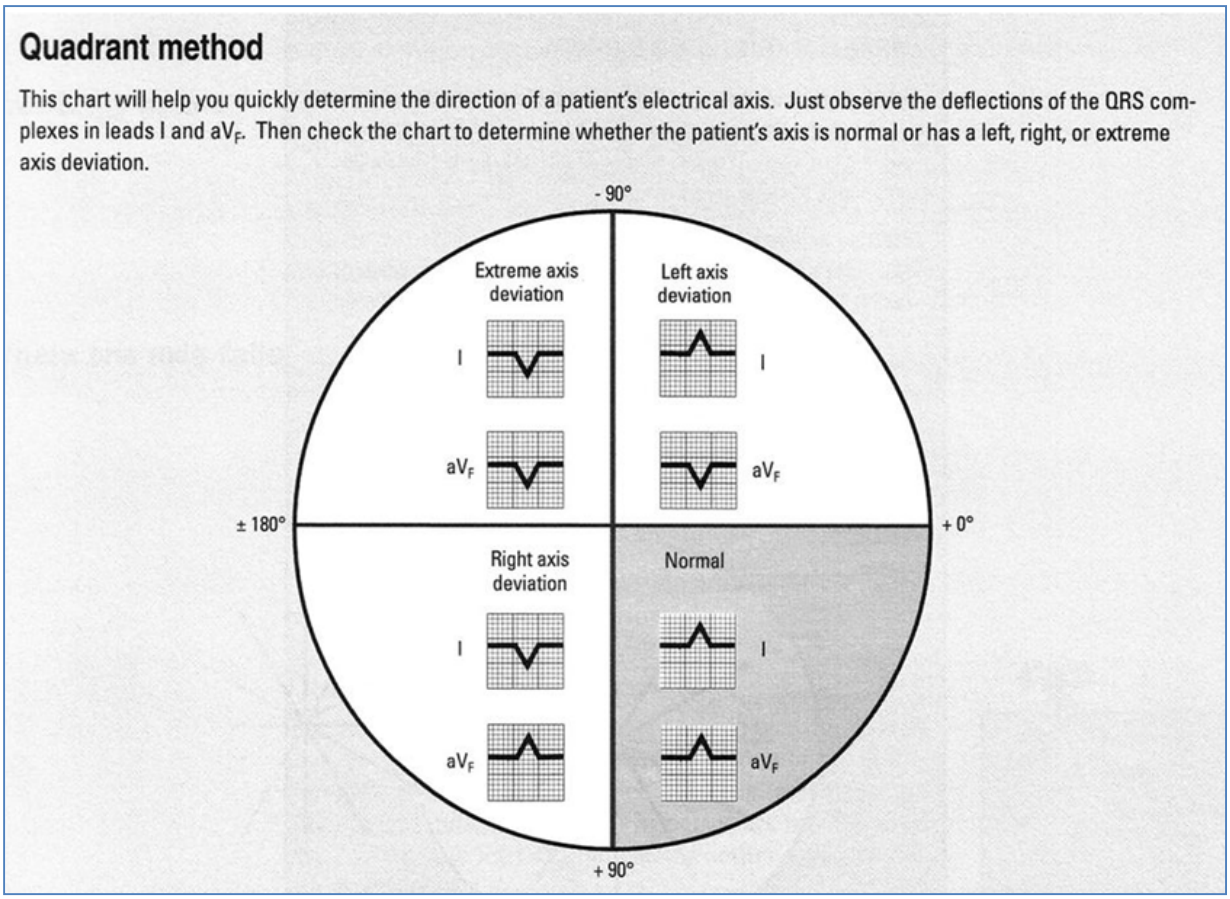
- **T – Wave:**
  - Ventricular Repolarisation
  - Positive deflection despite being a Repolarisation wave – because Repol. Waves travel in the opposite direction to Depol Waves.


P wave
P
PQ segment
P
Atria contract
Q wave
P
Q
R wave
R
P
Q
S wave
R
P
Q S
ST segment
R
P
Q S
Ventricles contract
T wave
R
P
T
Q S
The end
R
P
T
Q S


Time (msec)
0
100
200
300
400
500
600
700
800
Electro-cardiogram (ECG)
P
QRS complex
T
Cardiac cycle
QRS complex
P
120
90
60
30
Pressure (mm Hg)
Aortic valve opens
Aorta
Left ventricular pressure
Aortic valve closes
Dicrotic notch
Mitral valve closes
Left atrial pressure
Mitral valve opens
S1
S2
Heart sounds
135
65
Left ventricular volume (mL)
End diastolic volume
End systolic volume
Atrial systole
Ventricular systole
Ventricular diastole
Atrial systole
Atrial systole
Isovolumic ventricular contraction
Ventricular systole
Early ventricular diastole
Late ventricular diastole
Atrial systole

The Heart's Electrical Axis:

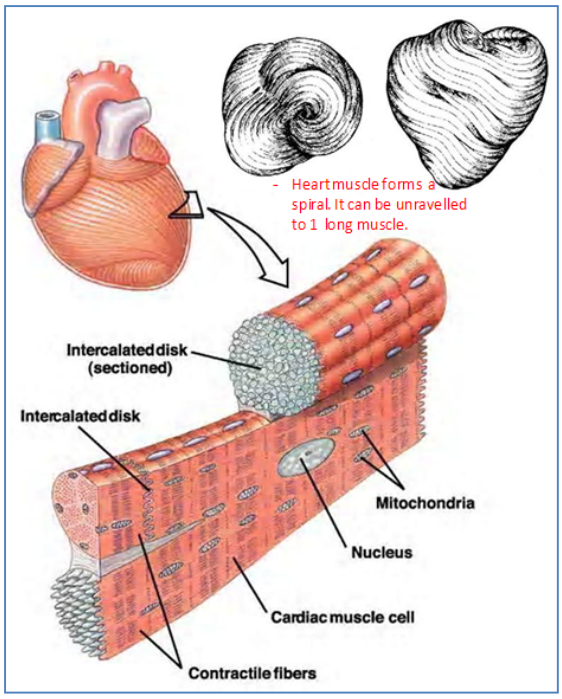
- Refers to the general direction of the heart's depolarisation wavefront (or 'mean electrical vector') in the frontal plane.
- It is usually oriented in a 'Right Shoulder to Left Leg' direction.
- **Determining The Electrical Axis From an ECG Trace:**
  - 3 Methods:
    - Quadrant Method (the one you're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



CardioVascular Medicine Notes
Mechanical Events of The Cardiac Cycle

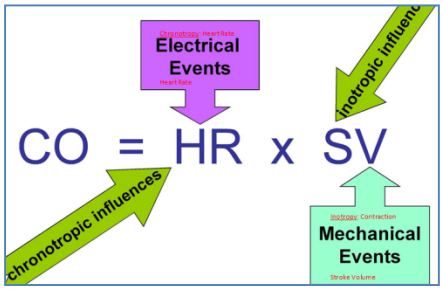
Structure-Function Relationship of the Heart

- 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.



Terms:

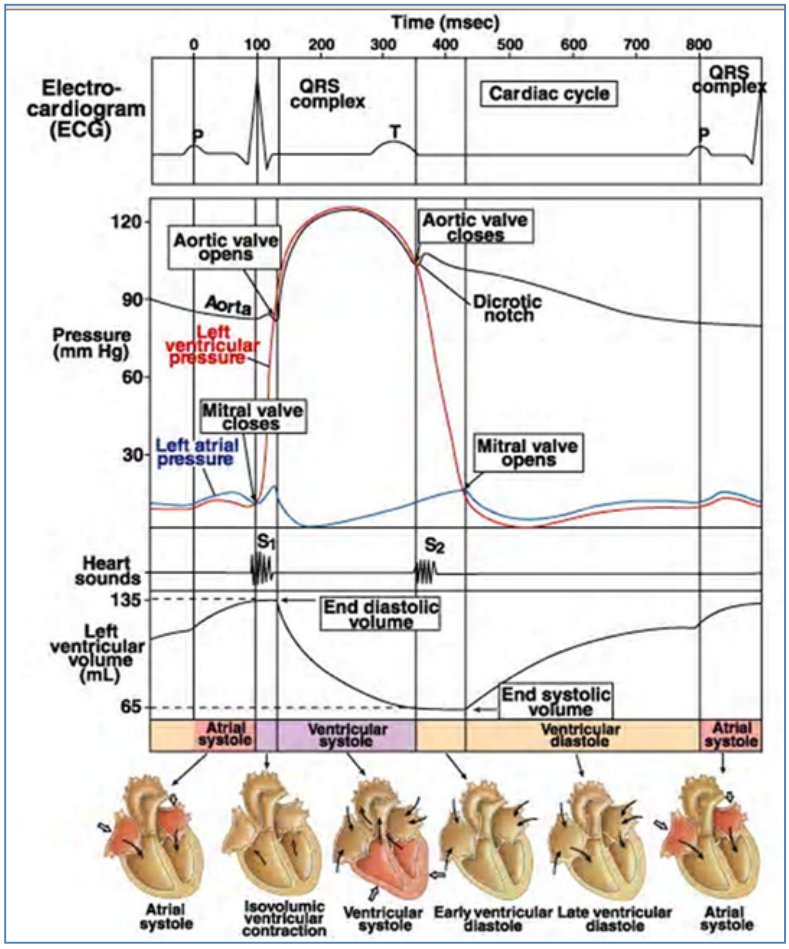
- **Systole:**
  - When Any Chamber of the Heart Contracts.
  - Blood → Out
- **Diastole:**
  - When Any Chamber of the Heart Relaxes.
  - Blood → In
- **Cardiac Output:**
  - Output of Blood from the Heart *Per Minute*
  - *Cardiac Output = Heart Rate x Stroke Volume*
  - **Chronotropic Influences:**
    - Factors affecting Heart Rate
  - **Inotropic Influences:**
    - Factors affecting Force of Contraction (& stroke volume)
  - **Dromotropic Influences:**
    - Factors affecting Conduction Velocity of AV-Node.



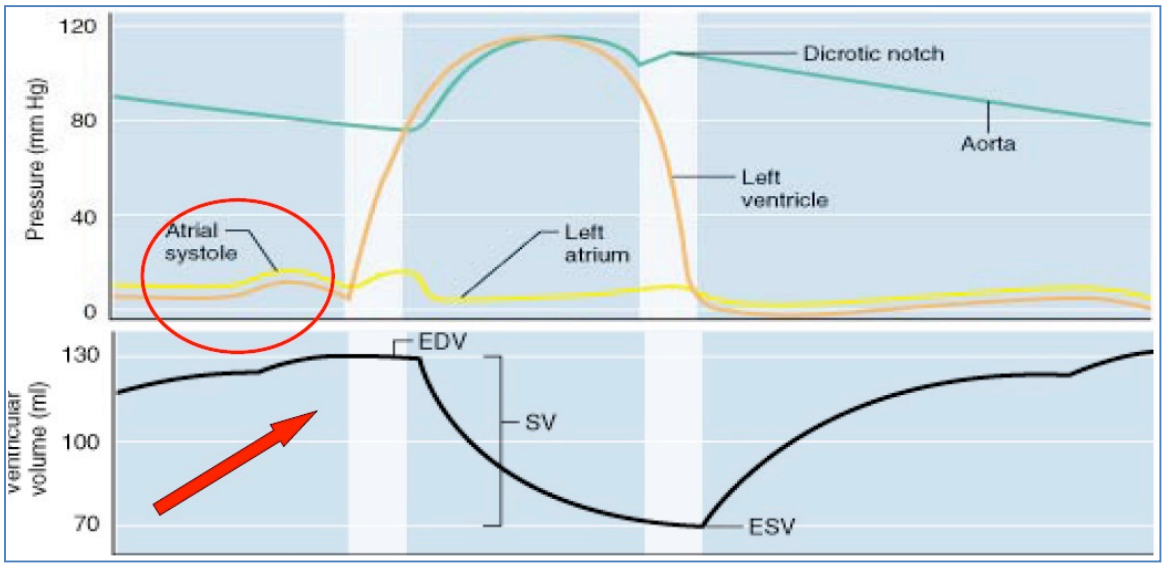
- **Stroke Volume:**
  - Output of Blood from the Heart *Per Contraction*
- **Heart Rate:**
  - #Heart Beats/Minute
- **End Diastolic Volume:**
  - Ventricular Volume during Complete Diastole (end of diastole).
  - When Ventricle is at its *Peak Fullness*
- **End Systolic Volume:**
  - Volume of blood left in Ventricles after Contraction
  - NB: not all ventricular blood is ejected during contraction (only 60-65%)

Mechanical Events of the Cardiac Cycle: + Relationships to ECG Trace:

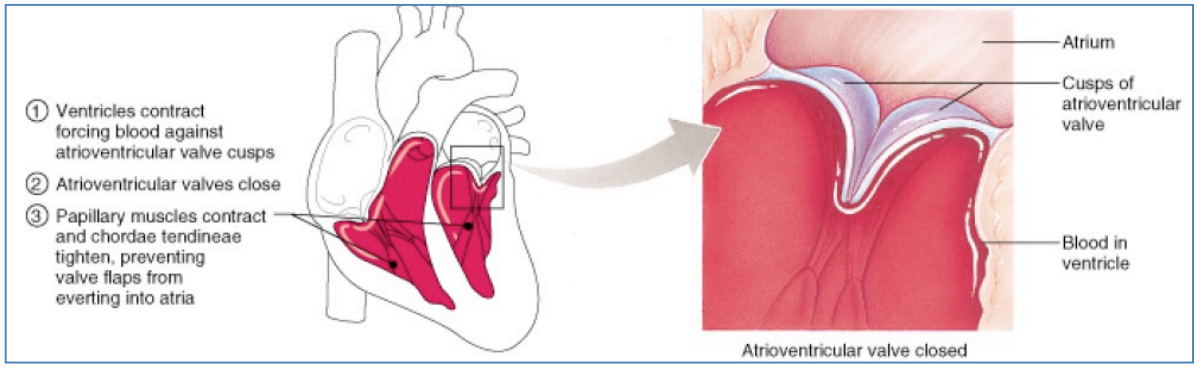
- NB: Contractions of the Heart ALWAYS Lag Behind Impulses Seen on the ECG.
- Fluids move from High Pressure → Low Pressure
- Heart Valves Ensure a *UniDirectional* flow of blood.
- Coordinated Contraction Timing – Critical for Correct Flow of Blood.



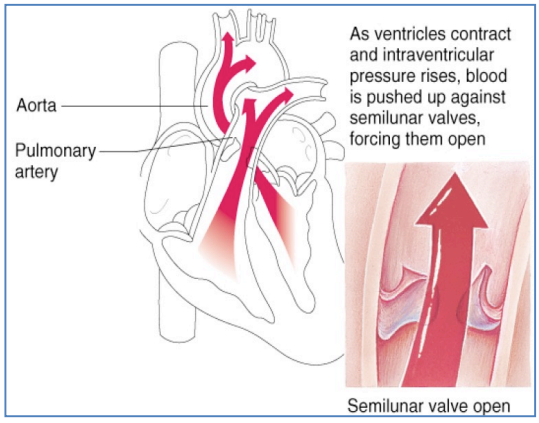
- **1. Atrial Systole/Ventricular Filling (Diastole):**
  - Contraction of Atria
  - IntraAtrial Pressure Increases
  - Blood pushed into Ventricles through AV-Valves
  - NB: Ventricles are already 70% full from passive Venous Filling.
  - At End of Atrial Systole, Ventricles have EDV (End Diastolic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

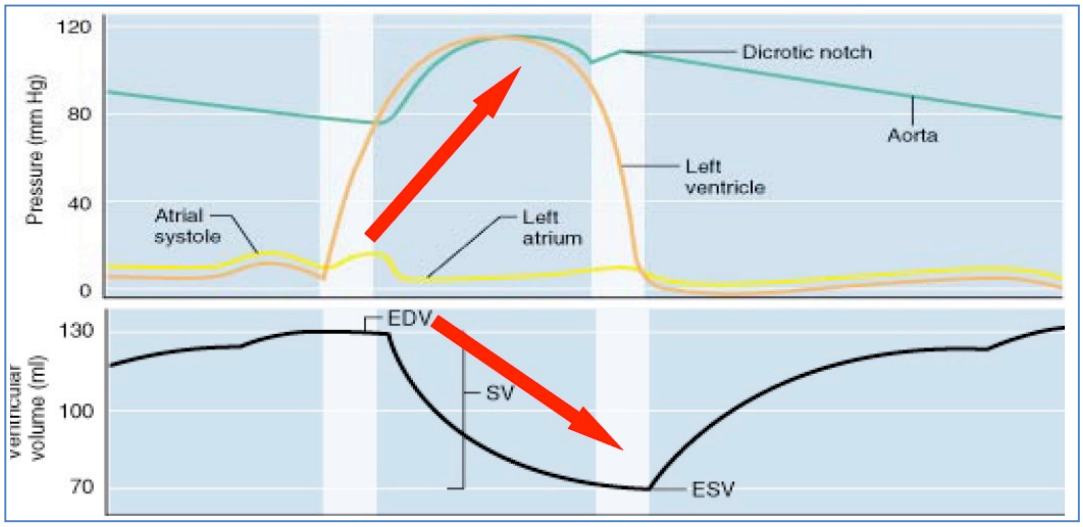


- **2. Ventricular Systole:**
  - **Early Phase:**
    - Ventricles Contract
    - Ventricular Pressure Exceeds Atrial Pressure → AV Valves shut
    - **IsoVolumetric Contraction Phase:**
      - The beginning of ventricular systole
      - All valves are *Closed.*
      - Where the ventricular pressure rises, but *Volume Stays Constant.*

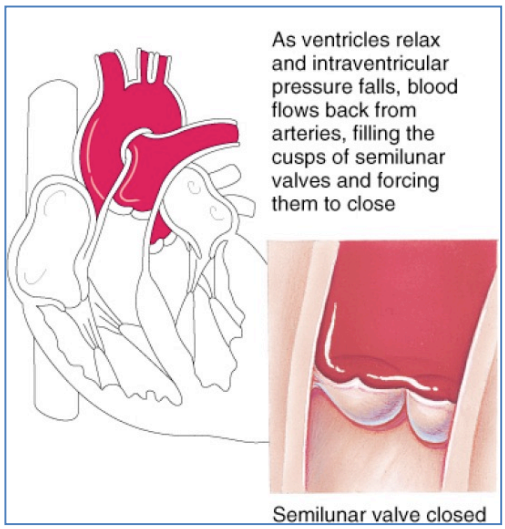


  - **Late Phase:**
    - Ventricular Pressure then Exceeds Aortic Pressure → Semilunar Valves Open → Blood Ejected
      - ≈80mL of blood ejected each time (Stroke Volume)
      - Ventricular Volume Decreases.
    - Ventricular Pressure then falls Below Aortic Pressure → Semilunar Valves Close.
      - 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*
    - Ventricles never *fully* empty:
      - ESV (End Systolic Volume) = Amount of blood left in ventricles → 50mLs.

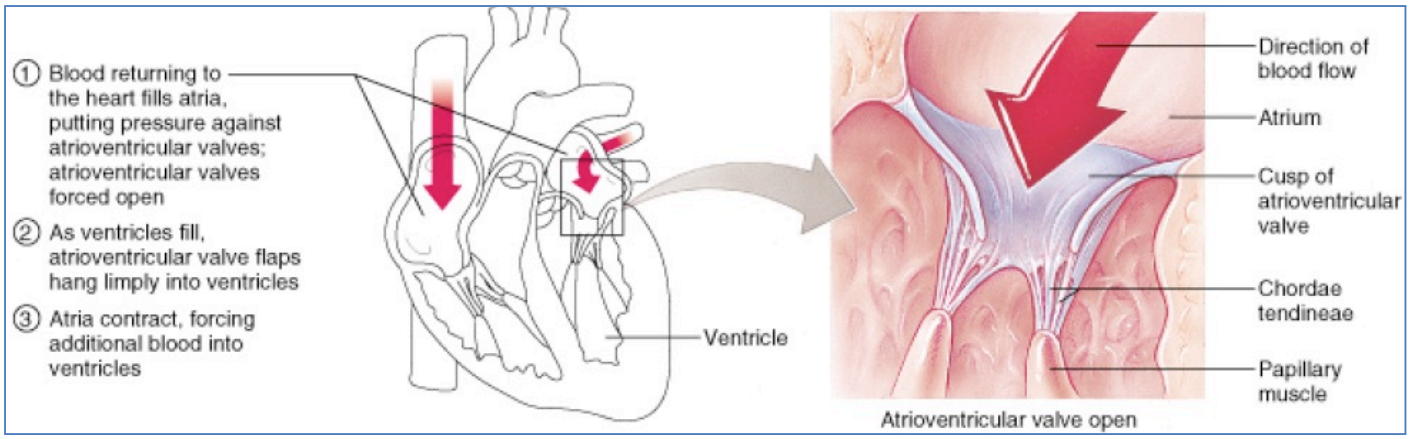




- **3. Ventricular Diastole:**
    - **IsoVolumetric Relaxation Phase:**
        - Initially when the Ventricle relaxes, both the Semilunar & AV-Valves are still closed.
            - Occurs at a pressure that is still slightly higher than the Atria, but also lower than Aortic/Pulmonary Arteries.
        - At this time, venous blood is already beginning to fill the atria



    - When Ventricular Pressure falls below Atrial Pressure → AV-Valves Open:
        - Blood → from Atria into Ventricles
        - Passive filling of the Atria & Ventricles – responsible for 70% of ventricular filling.

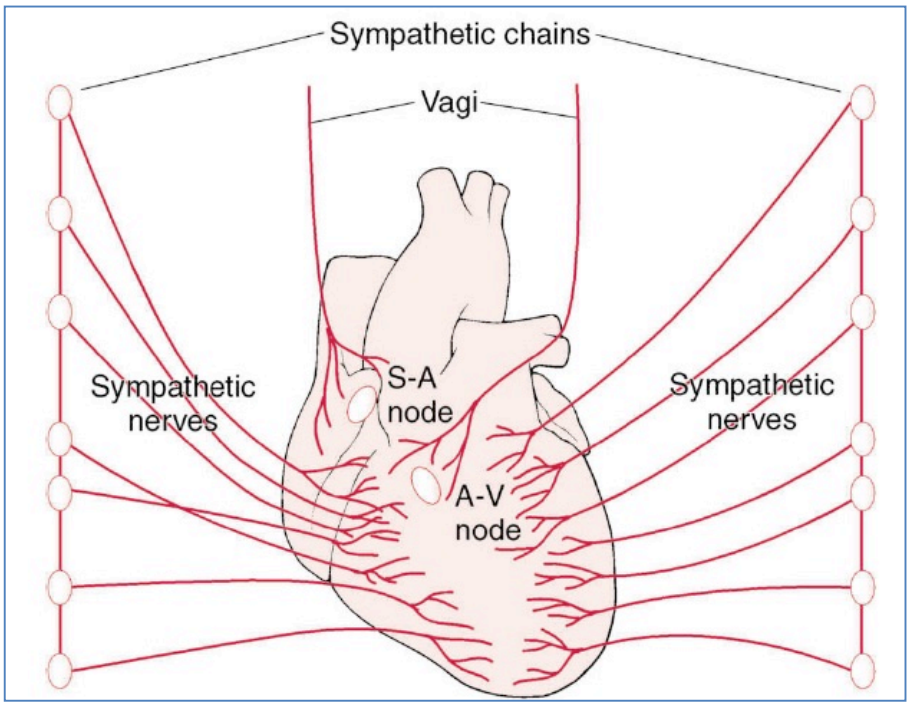


CardioDynamics:

- "The Movement & Forces Generated During Cardiac Contractions"
- **Cardiac Output:**
  - Useful when examining cardiac function *over time*.
  - **Determined by 2 Things:**
    - 1. Stroke Volume....&
    - 2. Heart Rate

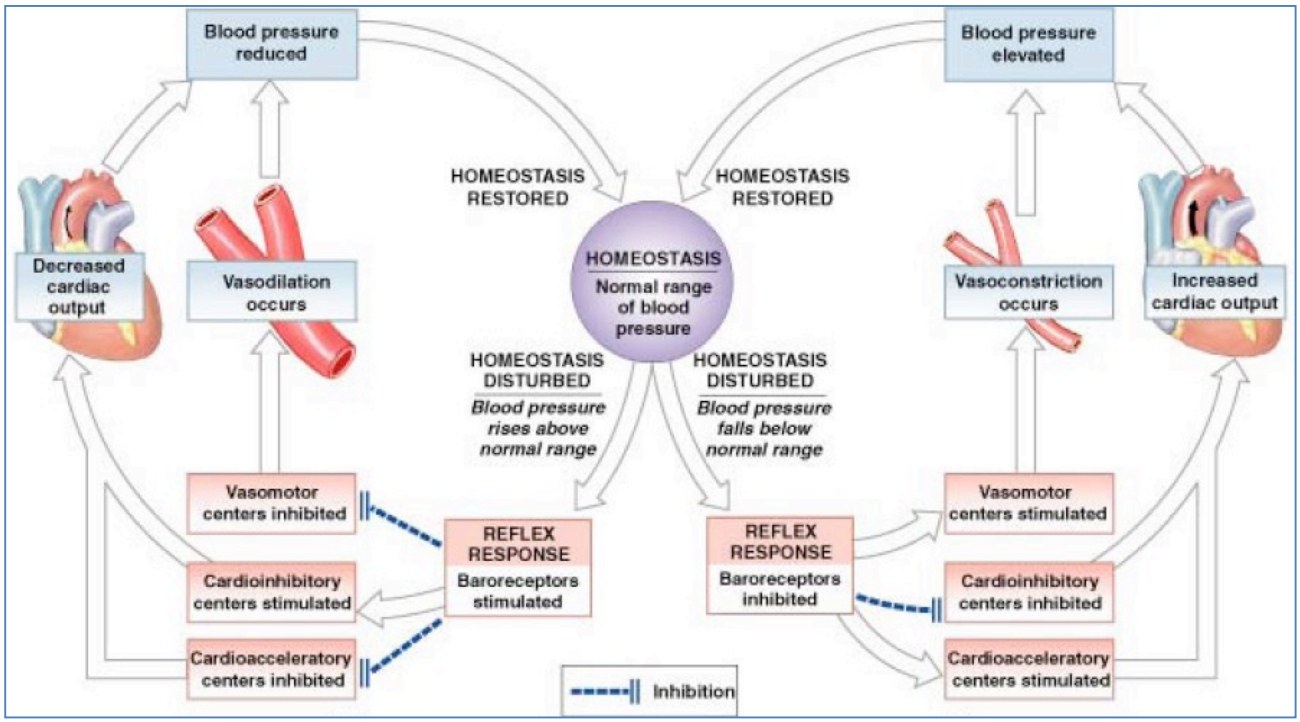
Cardiac Output$_{(mL/min)}$ = Stroke Volume X Heart Rate

  - Average CO ≈ 5L/min (ie. The entire blood supply circulates once per minute)
  - Is regulated such that peripheral tissues receive adequate blood supply.
  - **Heart Rate:**
    - Depends on Tissue-Satisfaction with Nutrients & O_2.
    - **Terms:**
      - **BradyCardia:** HR *Slower* than normal. (too fast → stroke volume & CO suffers)
      - **TachyCardia:** HR *Faster* than normal.
    - **Regulation:**
      - **Alterations in SA-Node Firing:**
        - SA-Node is the Pacemaker.....therefore:
          - Change its rate → change Heart Rate (→change Cardiac Output)
      - **Autonomic Nervous System:**
        - **Parasympathetic: (Vagus Nerve)**
          - *Decrease* Heart Rate (-ve Chronotropic Effect)
          - *Decrease* AV-Node Conduction (-ve Dromotropic Effect)
            - Increase delay between Atrial & Ventricular Contraction.
          - NB: ONLY A TINY EFFECT ON CONTRACTILITY
        - **Sympathetic: (Sympathetic Chains)**
          - *Increase* Heart Rate (+ve Chronotropic Effect)
          - *Increase* Force of Contraction (+ve Inotropic Effect).

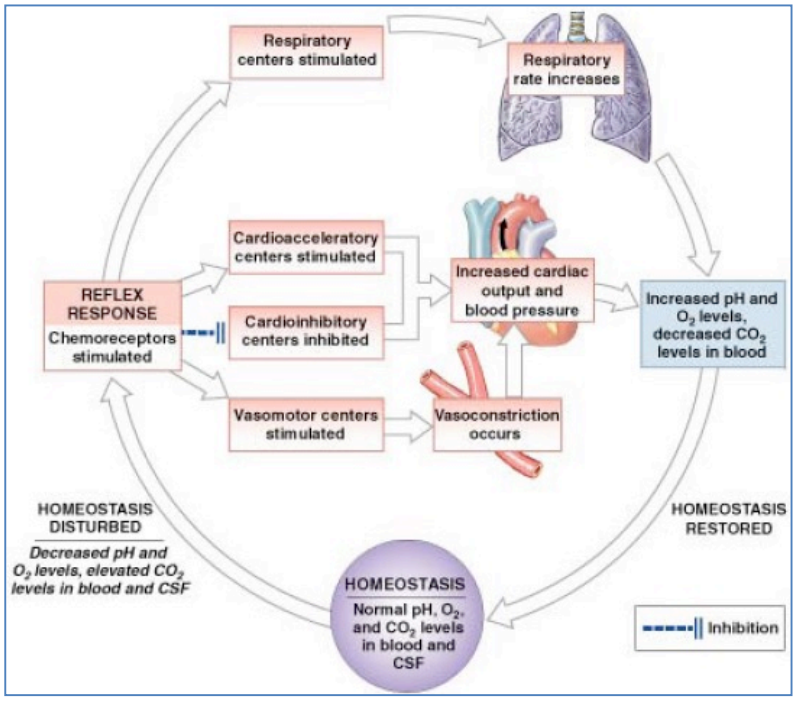


      - **Reflex Control:**
        - **Bainbridge Reflex (Atrial Walls)**:
          - Increase in HR in response to increased *Venous Return*
          - Stretch of Atrial Walls → Stretch Receptors → Symp.NS → ↑HR.
          - Responsible for 40-60% of HR increases.

- **BaroReceptor Reflex:**
  - **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:
      - Parasympathetic Activation
      - Sympathetic De-activation
  - Receptors Never Silent – constantly signalling
  - Quick to respond
  - In Hypertension → receptors recalibrate to the higher BP.
  - Changes HR accordingly



- **ChemoReceptor Reflex:**
  - Responds mostly to O_2 & CO_2 levels in Peripheral-Tissue.
  - If O_2 low → Respiratory Rate Increases (Small Increase in HR also)



- **Venous Return:**
  - Venous Return Fills Atria With Blood.
    - When Venous Return ↑, Atrial Walls Stretch → Stretches SA-Node.
  - Stretching of SA-Node Cells → More Rapid Depol. → ↑HR
  - Responsible for 15% of HR increases.
  - **Influenced by:**
    - Arterial Pressure
    - Peripheral Compliance
    - Local Blood Flow
    - Capillary Exchange

- **Chemical Regulation:**
  - **Hormones:**
    - Adrenaline
    - Thyroxine
    - Insulin
  - **Ions:**
    - Na^+
    - K^+
    - Ca^{2+}
  - **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 a *single* cardiac cycle.

Stroke Volume = End Diastolic Volume – End Systolic Volume

  - ↑End Diastolic Volume = ↑Stroke Volume
  - **EDV Influenced by:**
    - **1.** Filling Time (Duration of Ventricular Diastole)
    - **2.** Venous Return (Rate of Venous Flow during Filling Time)
  - **ESV Influenced by:**
    - **1.** Arterial BP (Higher → harder to eject blood → ESV Increases)
    - **2.** Force of V.Contraction (Higher → more blood ejected → ESV Decreases)

  - **Regulation of SV:**
    - **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.
      - 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)

    - **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.*

    - **Venous Return:**
      - What determines the preload of the heart
      - Influenced by:
        - Arterial Pressure
        - Peripheral Compliance
        - Local Blood Flow (depending on the demands of those tissues)
        - Capillary Exchange.

CardioVascular Medicine Notes
Blood Vessels

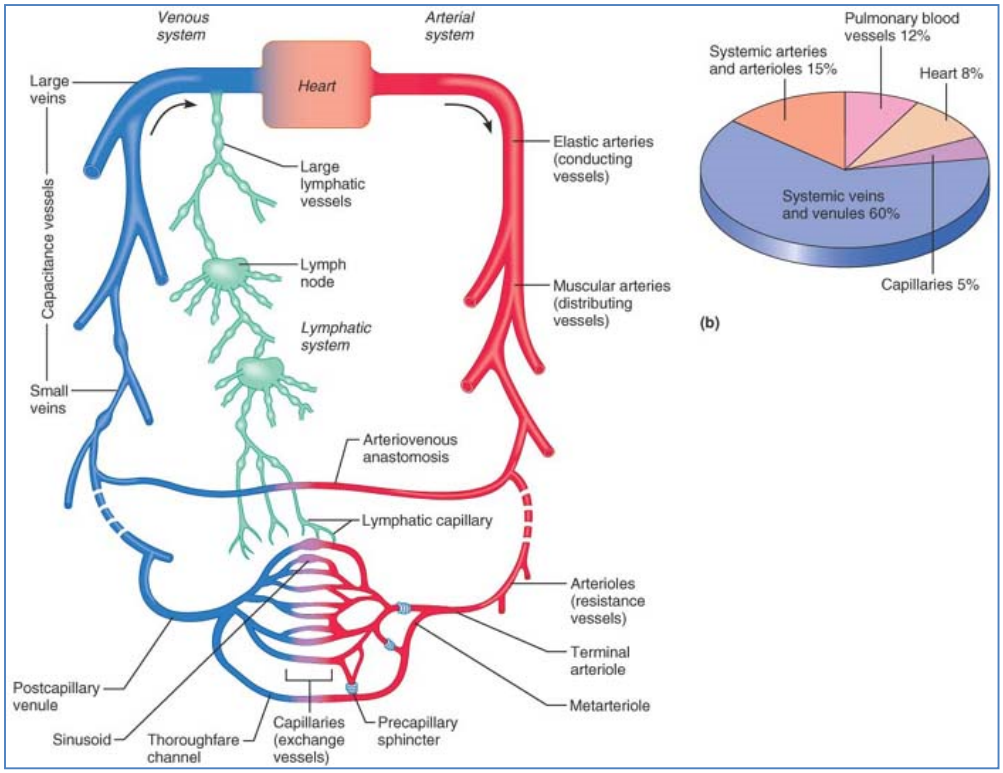
Objectives:

- Functional differences between arteries/veins/capillaries
- Anatomical & functional difference of each of the vessel layers
- How Capillary Beds Work
- Explain Net Filtration Pressure of a Capillary Bed
- How Oedema occurs.

Introduction to Blood Vessels:

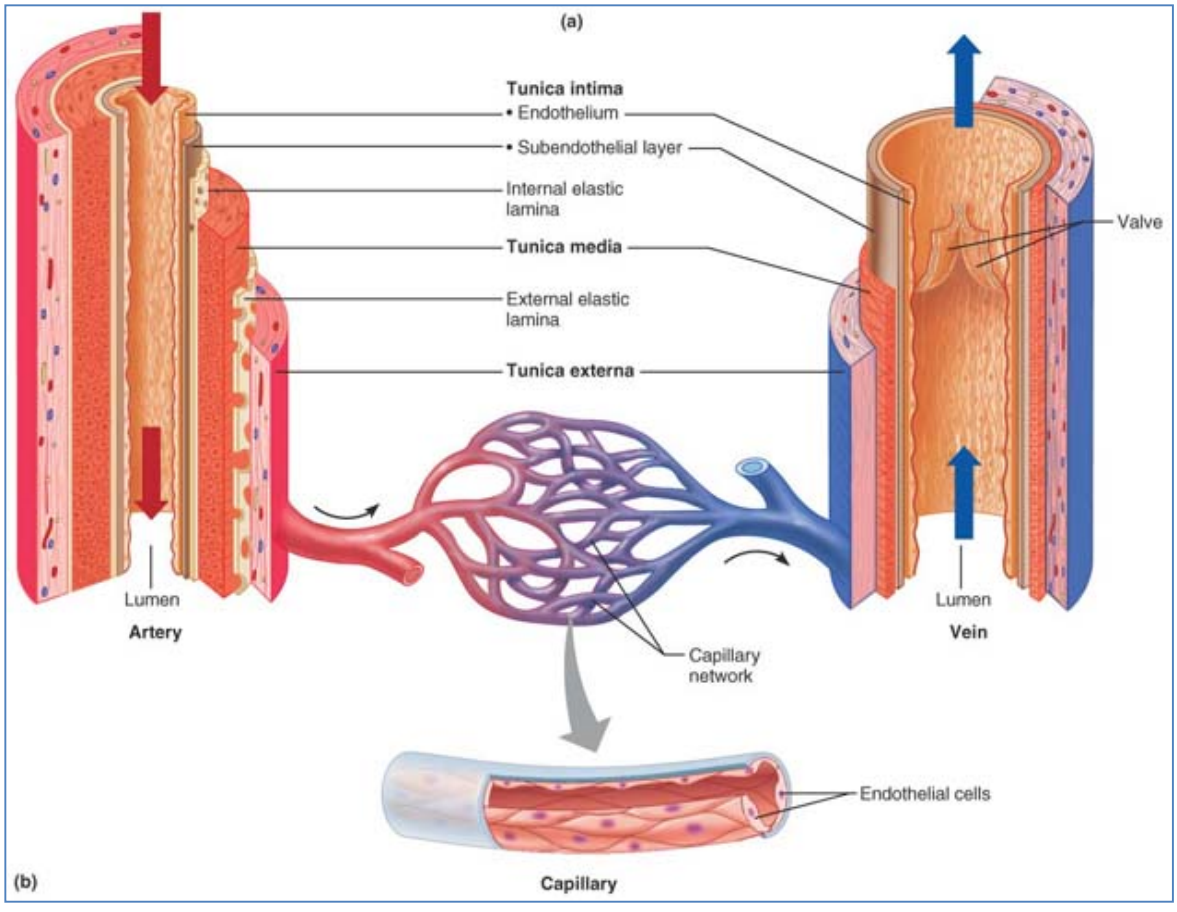
- **3 Classes:**
  - **Arteries – Carry blood away from the heart**
    - Elastic Arteries (Conducting Vessels)
    - Muscular Arteries (Distributing Vessels)
    - Arterioles (Resistance Vessels)
    - Terminal Arteriole
  - **Capillaries – Intimate contact with tissue → facilitate cell nutrient/waste transfer**
    - Precapillary Sphincters
    - Vascular Shunt
      - Metarteriol→Thoroughfare Channel
    - True Capillaries
      - Continuous Capillaries
      - Fenestrated Capillaries
      - Sinusoids (Leaky Capillaries)
  - **Veins – Carry blood back to the heart**
    - Post-Capillary Venule
    - Small Veins (Capacitance Vessels)
    - Large Veins (Capacitance Vessels)

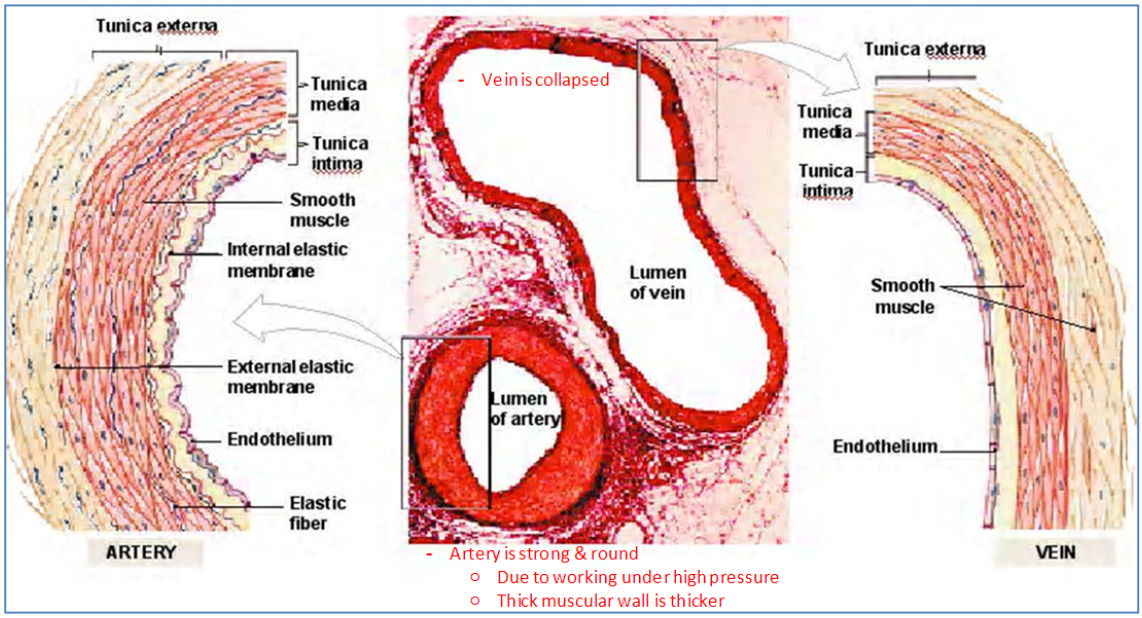
- **NB: Arterial blood isn't always oxygenated;** In Foetal & Pulmonary Circulation, placental & pulmonary arteries both carry deoxygenated blood.



Blood Vessel Structure:

- **3-Layered Wall:**
  - **Tunica Intima:**
    - Ie. The layer in *intimate* contact with the blood (luminal)
    - Minimises friction (flow resistance)
    - Consists of ***The Endothelium:***
      - Simple Squamous Epthelium
      - Lines the lumen
      - Continuation of the *Endocardium* (inside lining of the heart)
    - Larger vessels have a ***Sub-Endothelial Layer:***
      - Basement membrane...&
      - Loose Connective Tissue.
  - **Tunica Media:**
    - Middle....& Thickest layer
      - Circulating *Smooth Muscle*
      - Sheets of *Elastin*
    - Regulated by Sympathetic Nervous System + Chemicals
    - Contraction/Dilation *Maintains Blood Pressure.*
  - **Tunica Externa:**
    - Outermost Layer
    - Mostly loose *collagen fibres*
      - Protection
      - Reinforcement
      - Anchorage to tissues
    - Contains:
      - Nerve Fibres
      - Lymphatics
      - Vasa Vasorum (In larger vessels)
        - A system of tiny vessels
        - Nourish the tissues of the vessel wall





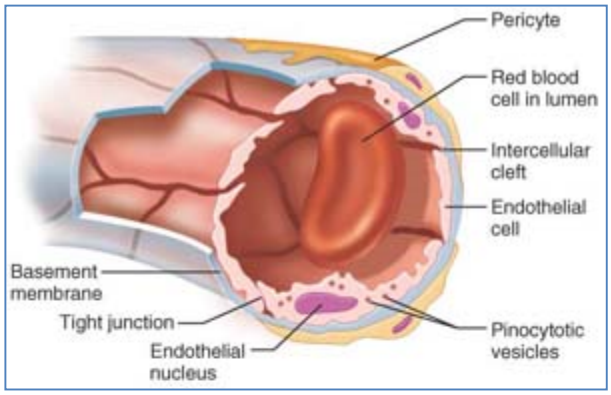
More Detail:

The Arterial System:

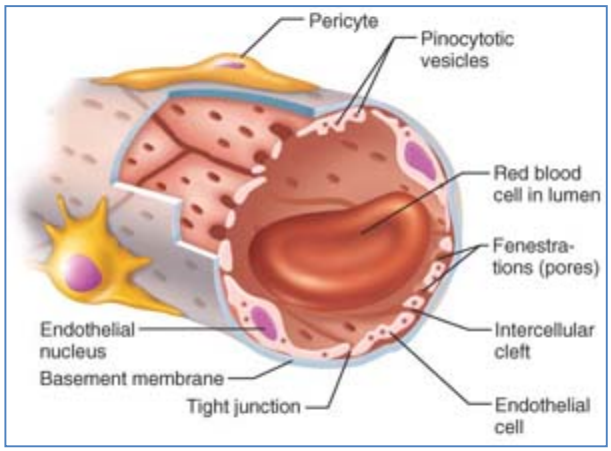
- **Elastic (Conducting) Arteries:**
  - Eg. The Aorta + its major branches
  - Close to the Heart.
  - Thick-Walled
  - Large Lumen = Low resistance
  - Highest Proportion of *Elastin:*
    - Withstands Pressure Fluxes
    - Smoothes out Pressure Fluxes
    - 'Stretch' = potential energy → helps propel blood during diastole.
  - Also Contain a lot of smooth muscle, but are relatively inactive in vasoconstriction.
    - Ie. Function of Elastic Arteries = simple elastic tubes.
- **Muscular (Distributing) Arteries:**
  - Distal to Elastic Arteries
  - Deliver blood to specific body organs
  - Diameter: 0.3mm→1cm
  - Thickest Tunica Media:
    - Due to smooth muscle
  - Highest Proportion of *Smooth Muscle:*
    - Are active in vasoconstriction
    - Are therefore less *distensible* (less elastin)
- **Arterioles:**
  - Smallest Arteries
  - Larger Arterioles have all 3 Tunics (Intima/media/externa).....
    - Most of the T.Media is Smooth Muscle
  - Smaller Arterioles – lead to capillary beds
    - Little more than 1 layer of smooth muscle around the endothelial lining.
  - 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.
  - Biggest controller of Blood Pressure

The Capillary System:

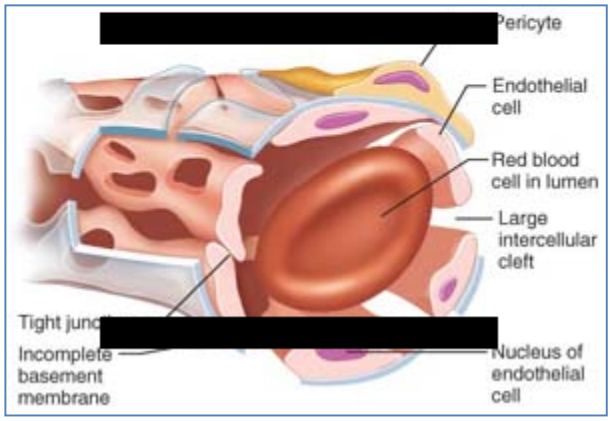
- Smallest blood vessels - microscopic
- Thin, thin walls – Tunica Intima Only – Ie. Only 1 layer thick.
- Average length = 1mm
- Diameter: The width of a single RBC.
    - RBC's flow through capillaries in single file
    - RBC's shape allows them to stack up efficiently against each other.
- Invades most tissues.
    - (except tendons/ligaments/cartilage/epithelia. – but receive nutrients from vessels nearby)
- Main Role:
    - Exchange of Gases/Nutrients/Hormones/Wastes
    - Exchange occurs between Blood & Interstitial Fluid.
- **3 Types:**
    - **Continuous Capillaries:**
        - Most common
        - Abundant in Skin & Muscles.
        - 'Continuous' = uninterrupted endothelial lining.
            - Adjacent cells form ***Intercellular Clefts****:*
                - Joined by incomplete-*tight-junctions*
                - (ie. Allow limited passage of fluids & solutes)
            - NB: in the brain, the *tight-junctions* are *complete* → blood brain barrier.



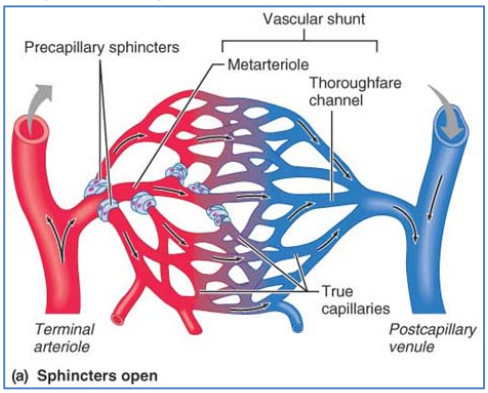
    - **Fenestrated Capillaries:**
        - Abundant wherever active absorption/filtration occurs.
            - Ie. Intestines
            - Kidneys
            - Endocrine organs (allow hormones rapid entry to blood)
        - Similar to Continuous Capillaries but….
            - Endothelial cells are riddled with oval pores *(Fenestrations = windows)*
            - Much more permeable to fluids & solutes than *continuous capillaries.*

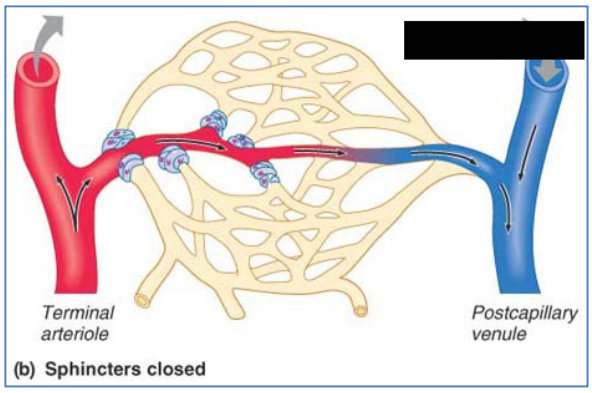


- **Sinusoids (Sinusoidal Capillaries):**
  - AKA "Leaky Capillaries"
  - Found ONLY in:
    - Liver
    - Bone Marrow
    - Lymphoid Tissues
    - Some Endocrine Organs
  - Large Irregularly-shaped lumens
  - Usually fenestrated
  - 'Discontinuous' = interrupted by *Kupffer Cells*:
    - Remove & destroy bacteria
  - Intercellular clefts → larger + have fewer tight junctions
    - Allow *large molecules* & *leukocytes* passage through to Interstitial Space.

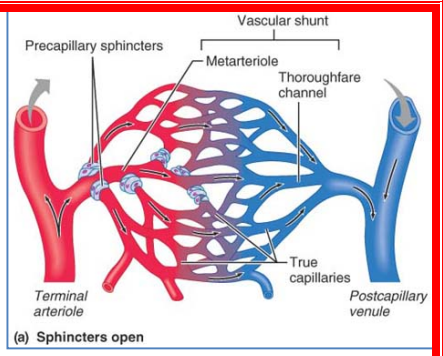


- **Capillary Beds:**
  - Capillaries are only effective in numbers:
    - Form networks called *'capillary beds'*
  - Facilitates ***'Microcirculation':*** Blood-Flow from an Arteriole → Venule
    - Consist of **2 Types of Vessels:**
      - **Vascular Shunt:**
        - From Metarteriole → Thoroughfare 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 Cap. Bed may be flooded with blood or bypassed, depending on conditions in the body or that specific organ.





Vascular Anastomosis:



- Anastomosis = "Coming Together"
- Hence...where vascular channels unite
- Arteries supplying the same territory often merge (*anastomose*)
    - Form alternate pathways
    - Aka. Collaterals
- Venous Anastomoses are also very common.
- Often occur around *Joints/Abdominal Organs/Brain/Heart*
- Also occur between Arterioles & Venules – "**Arteriovenous Anastomoses"**
    - Aka. = ***Metarteriole-Thoroughfare Channels*** See below sections...

The Venous System:

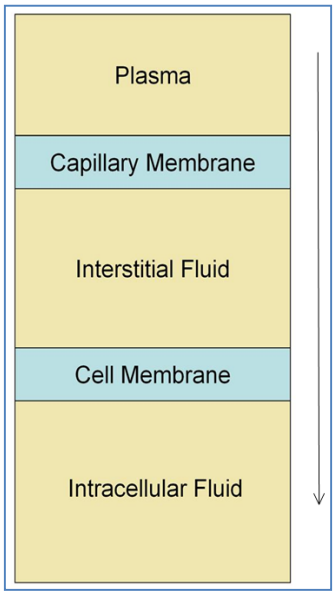
- Vessels carry blood back towards the Heart. (From Capillary Beds)
- Vessels gradually increase in Diameter & Thickness towards the heart.
- **2 Types:**
    - **Venules:**
        - Formed by union of capillaries (post-capillary venules)
        - Consist entirely of Endothelium
        - Extremely 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)

Body fluid – distributed between:

- Extracellular:
  - Blood Plasma
  - Interstitial Fluid
- Intracellular:
  - Cytoplasm

Interstitial Fluid:

- Contains Thousands of Substances:
  - Amino Acids
  - Sugars
  - Fatty Acids
  - Vitamins
  - Chem. Messengers
  - Salts
  - Wastes...etc.



Oedema:

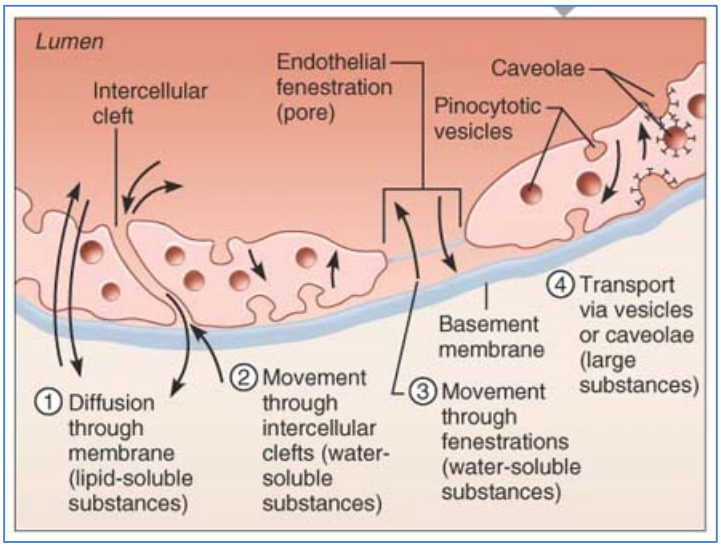
- Abnormal accumulation of fluid in the Interstitial Space = ie. ***Tissue Swelling***
- Caused by: increase in F*low of Fluid → Out of Vessel* OR *Lack of Re-Absorption → Into Blood Vessel*
- Usually reflects an imbalance in Colloid Osmotic Pressure on the 2 sides of the Capillary Membrane.
  - Eg. 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 a Inflammatory Response

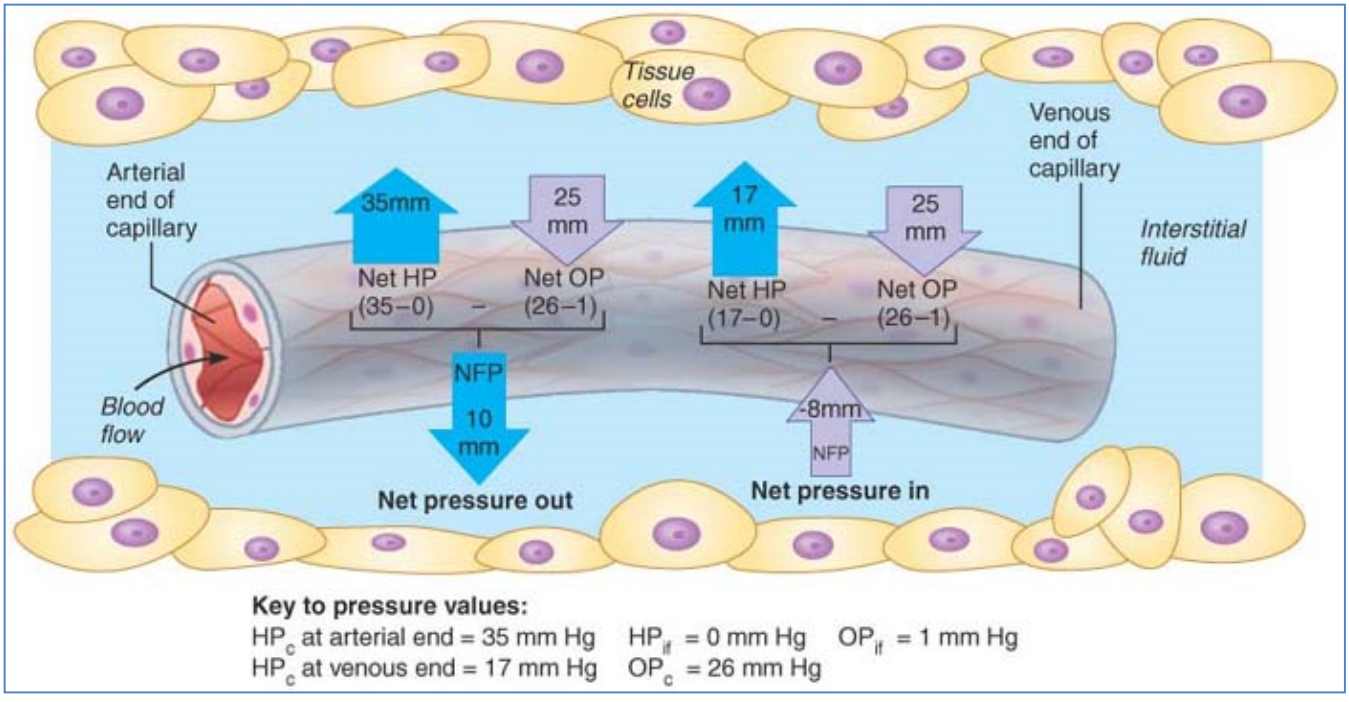
Fluid Movements:

- Fluid flows across capillary walls due to 2 forces:
  - **Capillary Hydrostatic Pressure:**
    - The force the blood exerts against the capillary wall.
    - ***Hydrostatic pressure = capillary blood pressure ≈35mmHg$_{Arterial\ End}$/15mmHg$_{Venous\ End}$***
    - 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.***
      - NB: 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.***
      - NB: Interstitial Osmotic Pressure ≈ 1mmHg

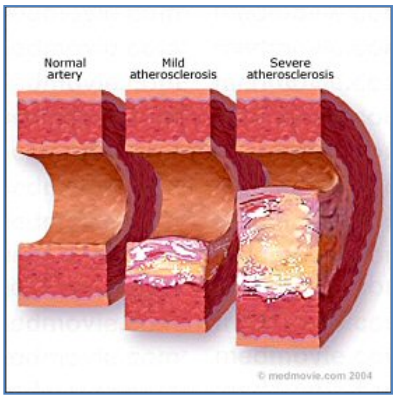
- Hence **Fluid** is **Forced Out @ Arterial End** & **Reabsorbed @ Venous End**
- The *amount* of fluid forced out – determined by the balance of net Hydrostatic & Osmotic forces.
  - Ie. ***Net Filtration Pressure = Net Hydrostatic Pressure – Net Osmotic Pressure***



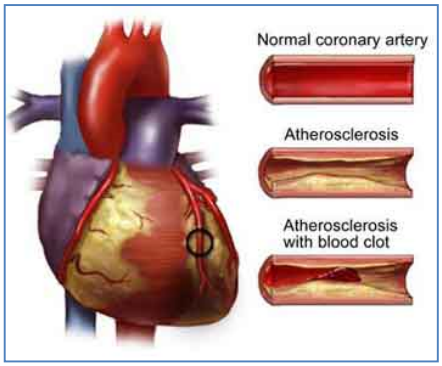


Injury to blood vessels

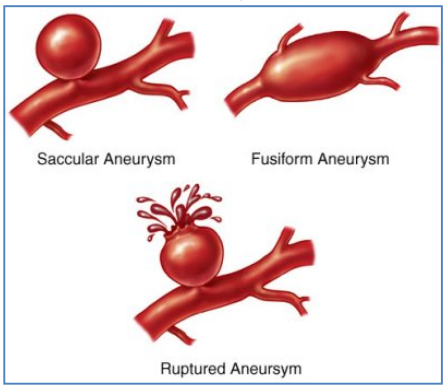
1. Early Athersclerotic lesions - lipid streaks.
   i. Ie the formation of fatty plaques
   ii. Fatty plaques begin to ulcerate
   iii. NB: Arterosclerosis is different = hardening of the vessel wall



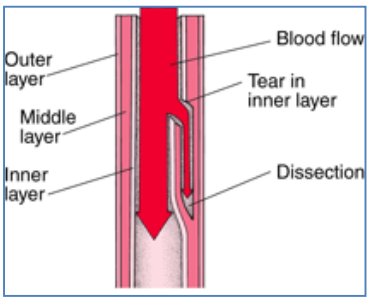
2. Coronary Blockages due to fatty plaque buildup
   i. Due to processed foods, saturated fats, bad diet.
   ii. Tends to be seen in many people these days



- **Elastic Arteries:**
  - Elastic due to concentric sheets of elastin
  - Can, however, lose their elasticity
  - Due to having thinner walls, they're more prone to **aneurysm** (bulging & potentially rupturing)
    - Results in pooling of blood --> eventual rupture.



- **Muscular Arteries:**
  - Those supplying specific organs
  - Less elastin, more muscle
  - Only a single sheet of elastin
    - More likely to have a tear in vessel wall.
  - **Dissecting aneurysms** (blood builds up between the layers of the wall & eventually press the vessel closed)



Smooth Muscle & Movement of Blood:

- **Elastic Vessels:**
  - Limited effect due to small muscle layer
  - Mainly elasticity mobilises blood
- **Muscular Vessels:**
  - Muscle will prevent rapid movement (vasoconstriction)
  - Control vessel diameter & influences BP (More next week)

Nervous System input in smooth muscle in arteries & its role in vasoconstriction:

- Alpha adrenergic receptors in smooth muscle of vasculature
- Sympathetic releases NA = vasoconstriction
- Beta Adrenergic receptors in coronary arteries --> Vasodilation --> increases blood flow to the heart
- Skeletal muscle blood vessels have Beta 2 recepors & some muscarinic receptors....why:?

Key Concepts of This Week:

- **Arterial System**
  - Vessel structure:
    - Layers
    - Wall structure
  - Movement of blood
    - In each of the 3 artery types:
    - Ie. Elastic arteries use their stretch...but in Muscular arteries & arterioles...blood moves due to pressure gradients
  - Smooth Muscle
    - Constriction
    - Dilation
    - Highly innervated by the sympathetic NS
      - Alpha Receptors (systemic vasculature)
      - Beta (coronary vasculature)
- **Venous System:**
  - Vessel Structure:
    - Larger lumen
    - Collapsible
  - Venous Return:
    - Valves
    - Muscular Pump
    - Respiratory Pump (by breathing in, the vessels in abdomen are compressed, milking blood back to the heart....while in the chest, the vessels are opened up...)
- **Capillary System:**
  - Vessel Structure:
    - 3 types
  - Capillary Bed:
    - Vascular shunt (metarteriole thoroughfare channel)
    - Precapillary sphincters
  - Movement of fluid:
    - Pressure gradients (arteriole end & venule end) IE. Capillary Hydrostatic Pressure
    - Coloid Osmotic Pressure - due to plasma proteins in blood drawing fluid from interstitial space.
- **Location of Major blood vessels:**
  - Coronary Vessels - all major ones
    - + what part of the heart these vessels are supplying
  - Others: use the page numbers below in marieb.
    - Know:
      - The blood vessels to this level of detail:
        - Marieb P.746-747 Table 19.4
        - Marieb P.758-9 Table 19.9

FIGURE 19.20 **Major arteries of the systemic circulation. (a)** Schematic flowchart. **(b)** Illustration, anterior view.

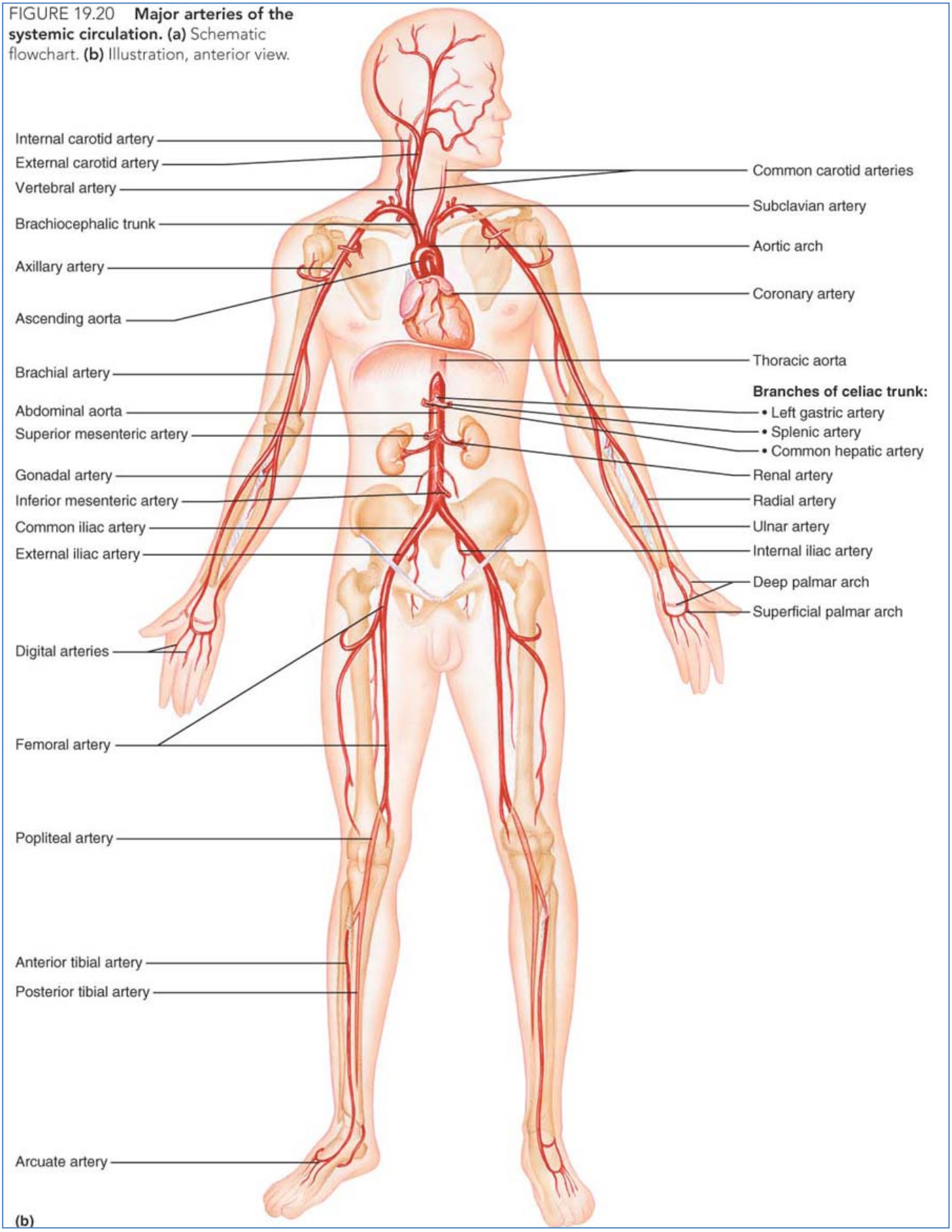
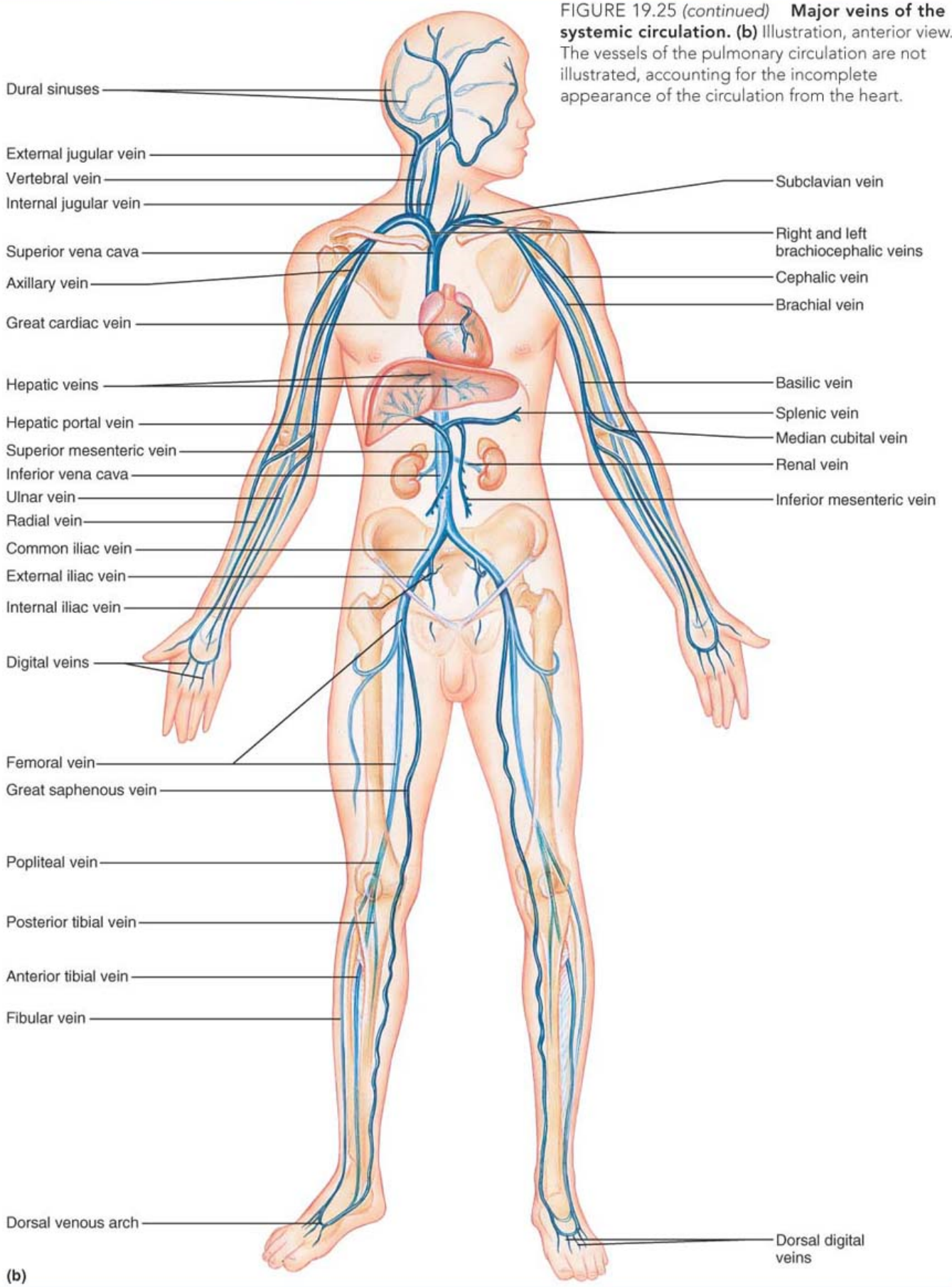


TABLE 19.9 *(continued)*

FIGURE 19.25 *(continued)* **Major veins of the systemic circulation. (b)** Illustration, anterior view. The vessels of the pulmonary circulation are not illustrated, accounting for the incomplete appearance of the circulation from the heart.



(b)

CardioVascular Medicine Notes
Control of Circulation (Haemodynamics & BP Regulation)

Blood Flow:
- The *Amount of blood* flowing through a vessel/organ/system *per unit time*. (mLs/min)
- Determined by pressure gradient & resistance.
- NB: NOT SPEED
    - 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 ≠ Blood Flow:**
    - **Blood Flow = *AMOUNT of flowing blood.* *(mL/min)***
    - **Velocity of Flow = *SPEED of flowing blood.* *(mm/sec)***
    - **NB:** A constricted vessel will have a ***lower flow rate***, but a ***higher velocity of flow*.** (ie. Garden hose)
    - NB: Velocity tends to change by a greater magnitude than the change in Flow Rate.

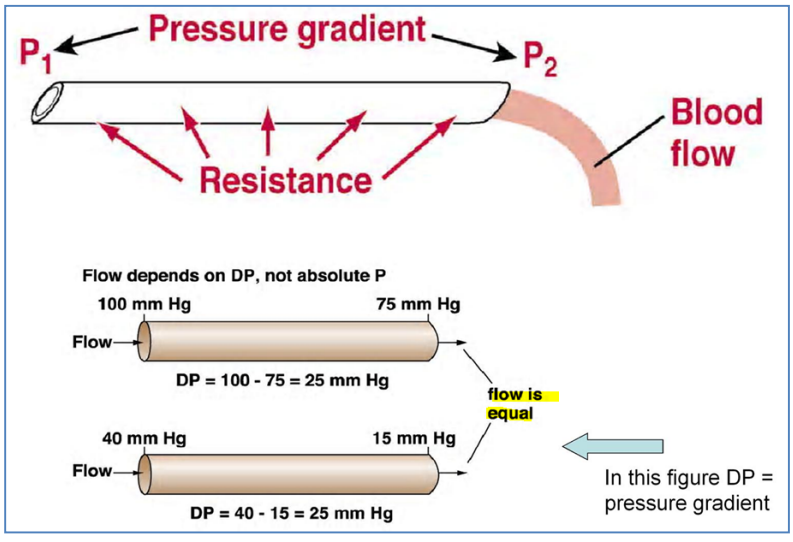
Blood Pressure: (**continued on page 4)
- The Pressure exerted on the vessel wall by contained blood. (mmHg)
- Decreases with distance from heart. (arterial system)
- Decreases with $10\%^{+}$ decrease blood volume.
- Increases with vessel constriction (provided same blood volume)

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

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:**

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

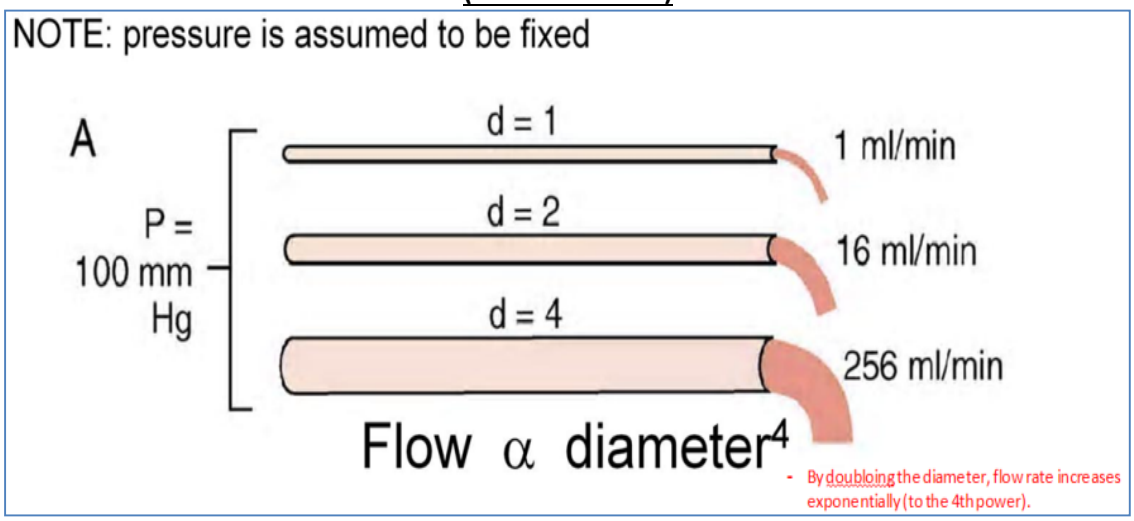
NB: Resistance is *far more important* in determining *local blood flow* than the Pressure Gradient.

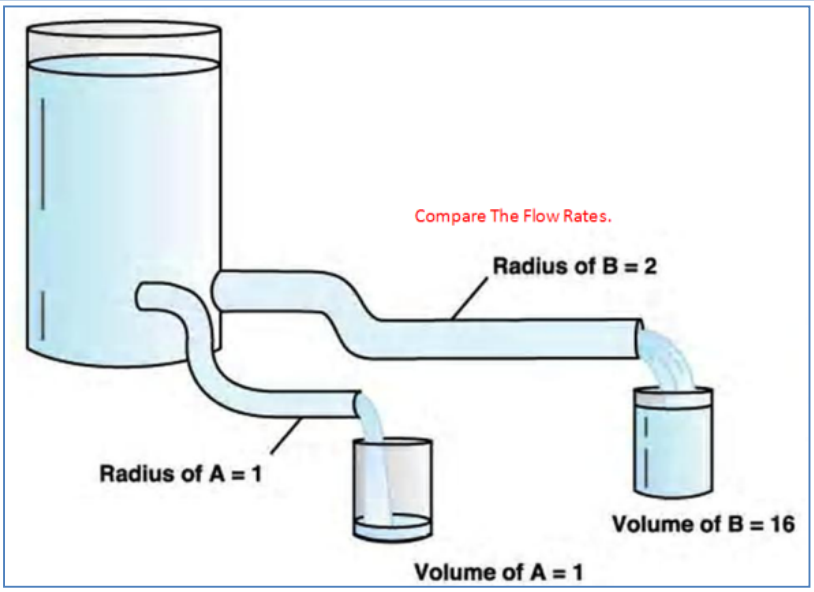


Effects of Vasomotion on *Rate* & *Velocity of Flow:*

- Constriction/Dilation:
- **Changes Vessel *Diameter*:**
  - **Influence 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.

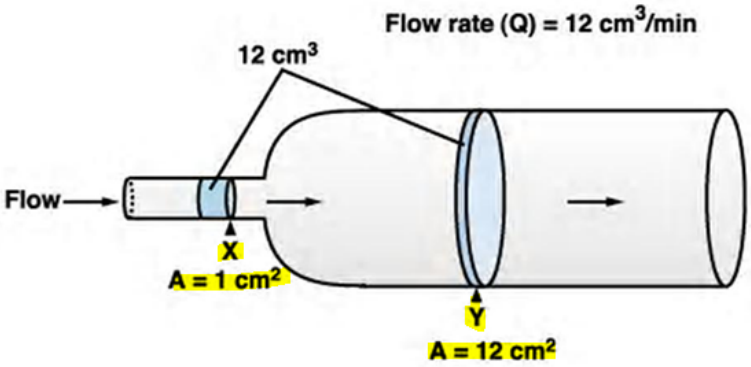
(Poiseuille's Law)





  - **Influence on *Flow Velocity:***
    - ***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'.

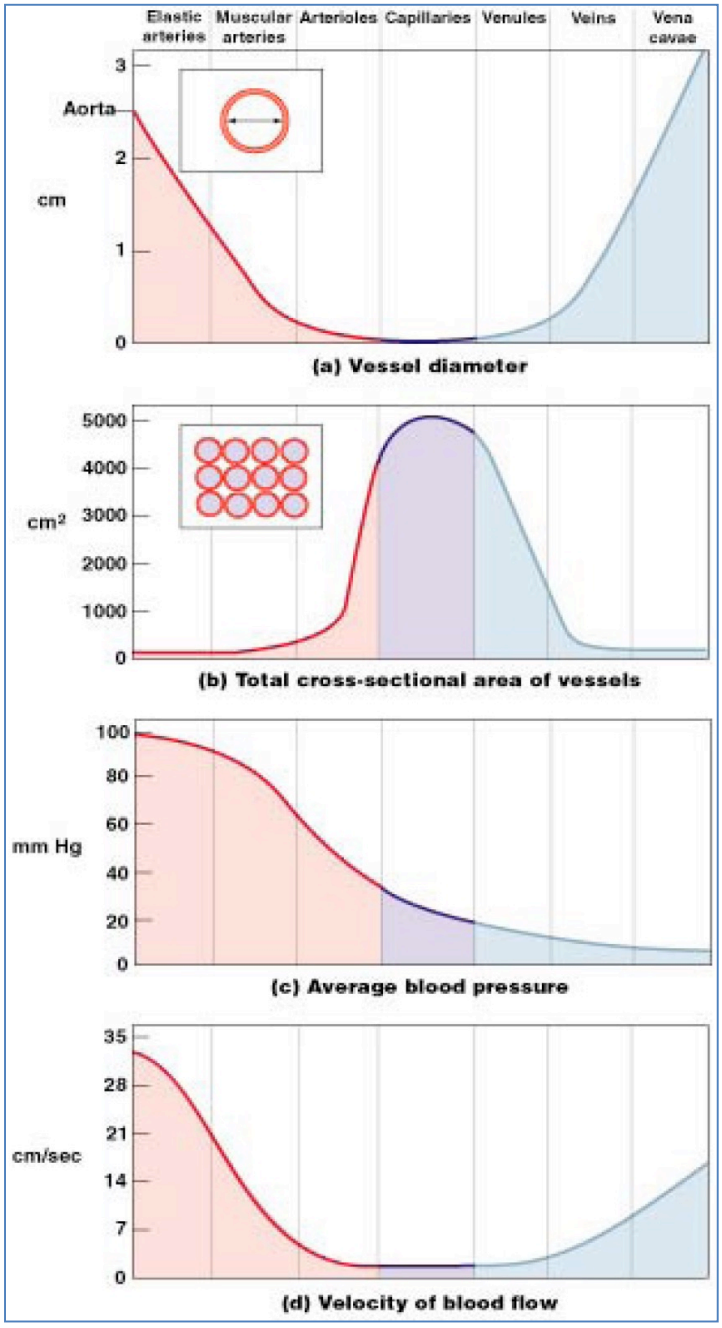
The effect of cross sectional area:



Velocity (v) = Flow rate (Q) / Cross-sectional area (A)	
At point X	At point Y
$v = \frac{12\ cm^3/min}{1\ cm^2}$	$v = \frac{12\ cm^3/min}{12\ cm^2}$
v = 12 cm/min	v = 1 cm/min

Properties of the Systemic Circuit:

- a) Individual Vessel Diameter:
- b) Total Combined Cross-Sectional Area of Vessels:
- c) Average Local Blood Pressure:
- d) Local Velocity of Blood Flow:



Blood Pressure Continued:

Factors Influencing Blood Pressure (Long Term):

- **Cardiac Output:**
  - ↑Cardiac Output = ↑ BP
- **Peripheral Resistance:**
  - Causes backpressure in blood (arterial system)
- **Blood Volume:**
  - (assuming constant vessel diameters) ↑Blood Volume = ↑BP
  - Its affect depends on vessel compliance

BP = Cardiac Output X Total Peripheral Resistance

Types of Blood Pressures:

- **Systolic:**
  - Peak Aortic pressure reached during ventricular systole.
  - Function of:
    - Peak *rate* of ejection
    - Vessel wall compliance
    - Diastolic BP
  - Normal = 120mmHg
- **Diastolic:**
  - Lowest Aortic pressure reached during ventricular diastole, due to blood left after peripheral runoff.
  - Function of:
    - Blood Volume
    - Heart Rate
    - Peripheral Resistance
  - Normal = 80mmHg
- ***Pulse Pressure:**
  - Difference in Systolic & Aortic Pressure (120mmHg – 80mmHg)
  - Normal = 40mmHg
  - If Lower – may be an indication of Aortic Stenosis or Atherosclerosis (slowed peripheral runoff)
- ***Mean Arterial Pressure (MAP):**
  - MAP = Diastolic Pressure + 1/3(Pulse Pressure)
  - *The Pressure that *Propels Blood to the Tissues* – maintains *Tissue Perfusion* (see below sections).
    - Maintains flow through capillary beds
  - Must be high enough to overcome peripheral resistance – (if not blood doesn't move)
  - Finely Controlled: See Below:

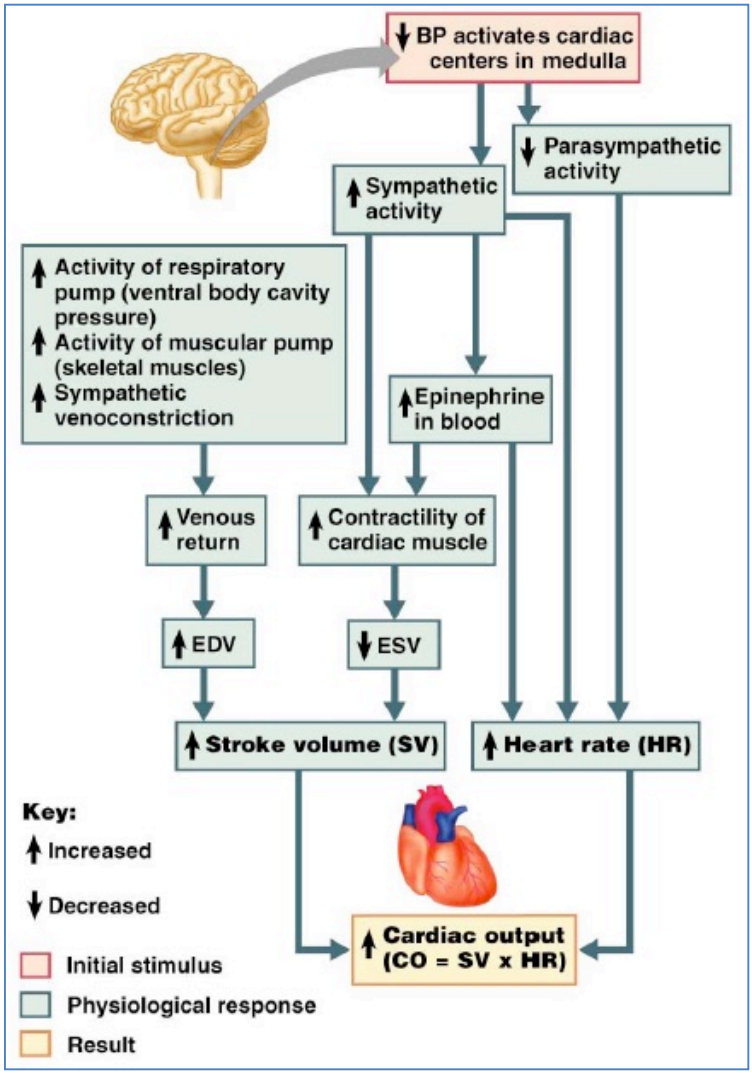
Tissue Perfusion:

- Blood flow to the tissues.
- Adequate Tissue Perfusion for:
  - Demands (O_2/nutrients) of organs/tissues.
  - Gas exchange in lungs
  - Nutrient Absorption in GIT
  - Urine formation in Kidneys.

Control of MAP:

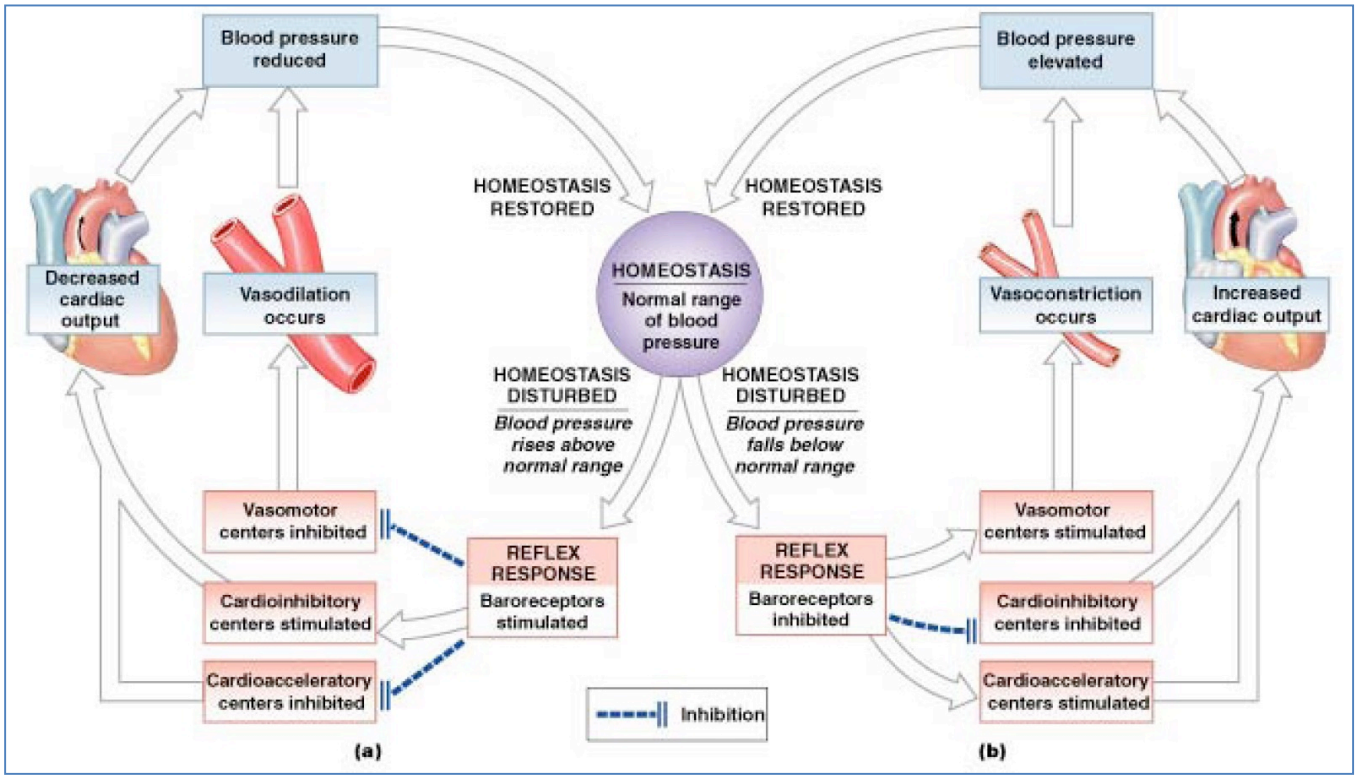
- **3 Main Regulators:**
  - **1. Autoregulation (Local):**
    - 'The automatic immediate adjustment of blood flow to each tissue relative to the tissue's requirements at any instant.'
    - Local (tissue bed) Level
      - Ie. Control of flow within *a single capillary bed*.
      - Ensures perfusion of the *'Needy'* Tissues.
    - **Short Term** – Immediate Adjustments – Due to:
      - **Metabolic Controls: → Vasodilation:**
        - Low Oxygen levels
        - Low Nutrient levels
        - Nitric Oxide
        - Endothelin
        - Inflammatory Chems: (histamine/kinins/prostaglandins)
        - NB: Was thought that Symp-ANS dilates → wrong! It only Constricts!
      - **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:**
  - **Short-term** Immediate adjustments:
  - Aims to:
    - Adjust Cardiac Output
    - Maintain adequate MAP by altering vessel diameter.
    - Alter blood distribution due to specific demands of various organs.
  - Mediated by the Nervous System.
    - Respond to changes in Arterial Pressure & Blood Gas Levels.
      - **Vasomotor Centres:**
        - Neuron Cluster in Medulla:
          - Take info from receptors:
            - Baroreceptors (primarily)
            - Chemoreceptors (lesser degree)
          - Transmit impulses *via SNS*:
            - ↑ sympathetic activity = vasoconstriction = ↑ BP
            - ↓ sympathetic activity = vasodilation = ↓ BP
      - **CardioVascular Centres of ANS:**
        - CardioAcceleratory (Sympathetic):
          - Active in times of Stress
          - ↑HR & Contractility
        - CardioInhibitory (Parasympathetic)
          - Active at Rest
          - ↓Heart Rate

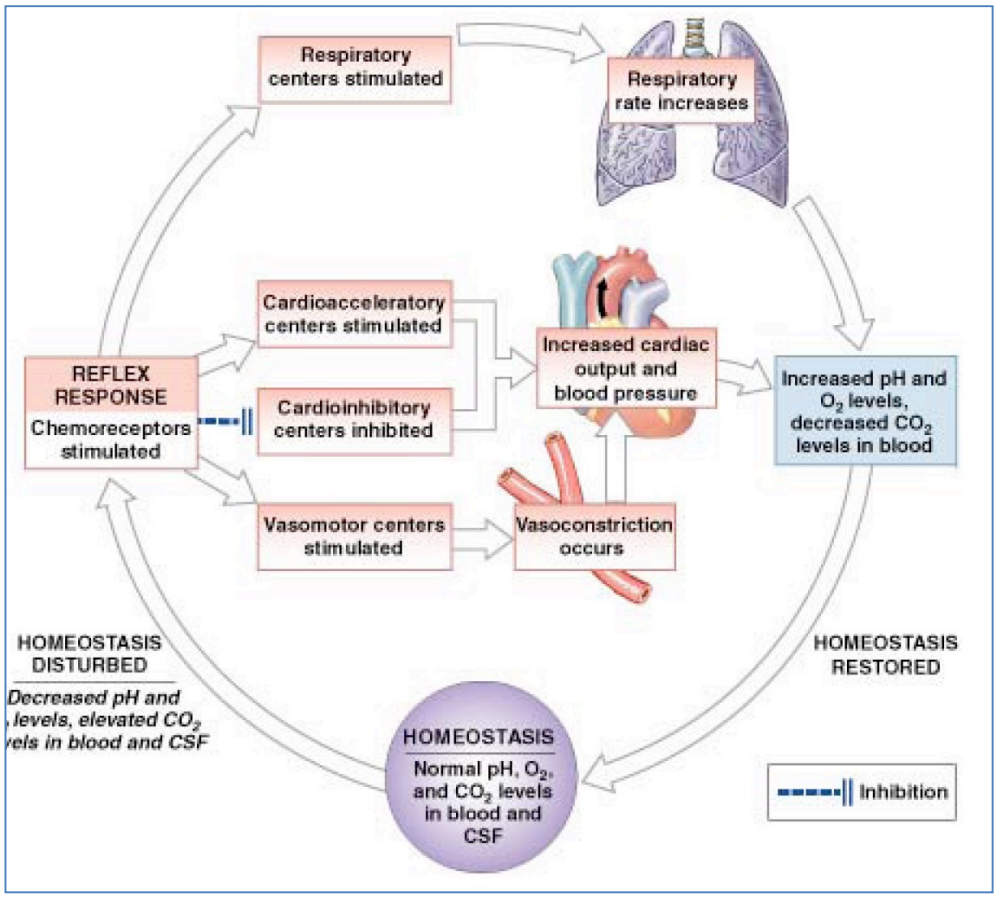


Effects of CV Centres on Cardiac Output

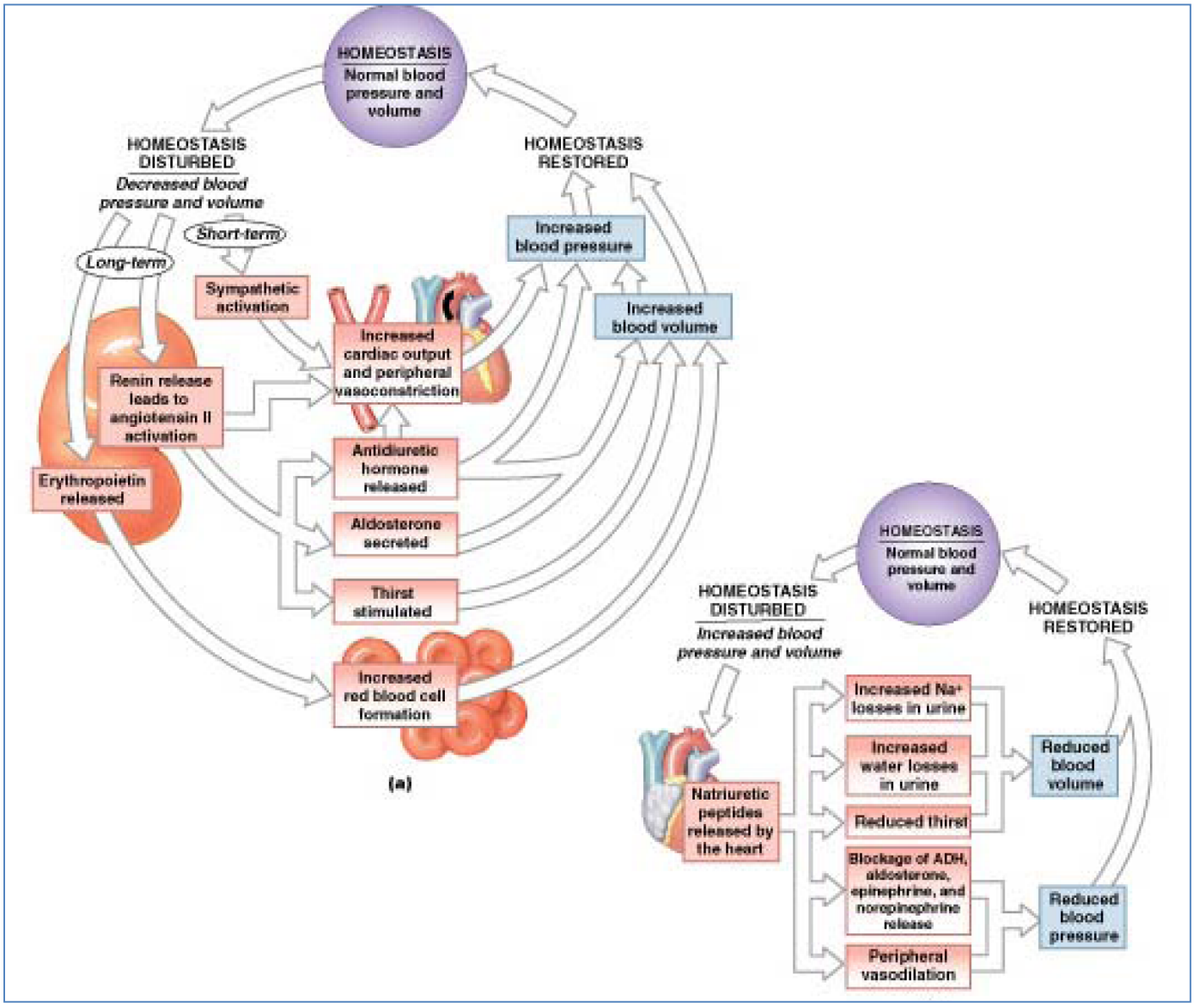
Baroreceptor Reflex



Chemoreceptor Reflex



- **3. Endocrine Mechanisms:**
  - More Long Term BP & Bl-Volume regulation:
  - AT THE KIDNEY LEVEL:
    - ****Antidiuretic Hormone (ADH) – AKA. Vasopressin:**
      - Released due to *Low* blood *volume*
    - **Angiotensin II:**
      - Released due to *Low* blood *pressure*
      - Potent VasoConstrictor
      - Increases CO & Blood volume
      - (NB: 'ACE' (Angiotensin I Converting Enzyme) activates it to Angiotensin II. Hence 'ACE-Inhibitors' are often used as *AntiHypertension* medicine)
    - **Erythropoietin:**
      - Released due to *Low Pressure & Low O_2 Levels.*
      - Increases RBC production to increase Blood Volume.
    - **Natriuetic Peptides:**
      - Released *by the heart* due to *High Blood Pressure & Volume.*
      - Inhibits ADH & Angiotensin II → *Reduces BP & Volume.*



Systolic Blood Pressure

Q. Compare the systolic blood pressure response to exercise in the individuals with quadriplegia and heart transplantation. How are they different and what accounts for this difference?

The systolic blood pressure response in the individual with quadriplegia is much lower than that in the individual with heart transplantation because of the poor stroke volume response to exercise. The reduced stroke volume response to exercise is due to the failure of cardiac performance to increase resulting from an absence of the muscle venous pump (frank starling law) and reduced muscle mass.

Systolic Blood Pressure

Q. Why is the systolic blood pressure lower in the individual with heart transplantation than that in the sedentary individual?

The systolic blood pressure response to exercise is lower in the individual with heart transplantation compared with the sedentary individual because of a lower stroke volume response to exercise (absence of cardiac sympathetic efferent activity).

Q. Compare systolic blood pressure in the athlete and the sedentary individual. How are they different and why?

The athlete has a much higher systolic blood pressure response to exercise than the sedentary individual because of a larger stroke volume and a more rapid rate of its ejection.

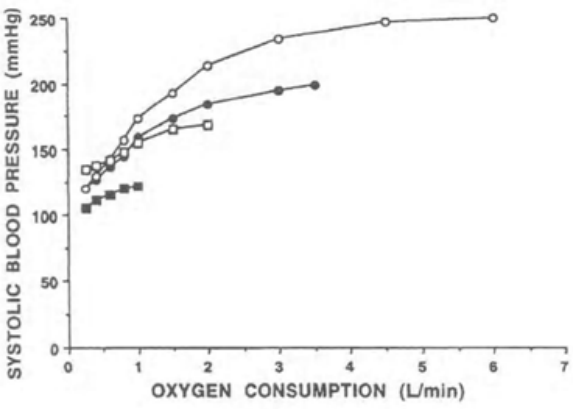


FIG. 8

Relationship between systolic blood pressure and increasing work loads in the 4 individuals. ○, athlete; ●, sedentary; □, cardiac transplant; ■, quadriplegia.

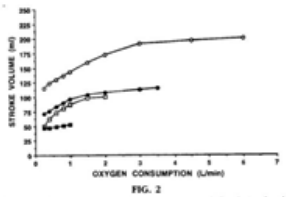


FIG. 2

Stroke volume response to increasing work loads in the 4 individuals. ○, athlete; ●, sedentary; □, cardiac transplant; ■, quadriplegia.

Diastolic Blood Pressure

Q. Explain the response of diastolic blood pressure to exercise in the athlete and the sedentary individual.

Total peripheral resistance decreases more in the athlete because the athlete has an increased cardiac performance and much higher systolic pressure, which maintains perfusion pressure. This means the baro/metabol-chem receptor reflexes are not activated to increase total peripheral resistance. Therefore diastolic pressure decreases (metabolic vasodilatation).

Q. Why might the individuals with heart transplant and quadriplegia have an increase in diastolic blood pressure?

Normally diastolic blood pressure decreases in response to exercise. However, in the individuals with heart transplantation and quadriplegia, the diastolic pressure rises in response to increasing work loads because of impaired cardiac performance. This activates the arterial baroreflex and muscle metaboreflex, which reflexly increase total peripheral resistance. Therefore, to maintain perfusion pressure, diastolic pressure will not decrease, and in severe cases it will increase because of an increase in total peripheral resistance.

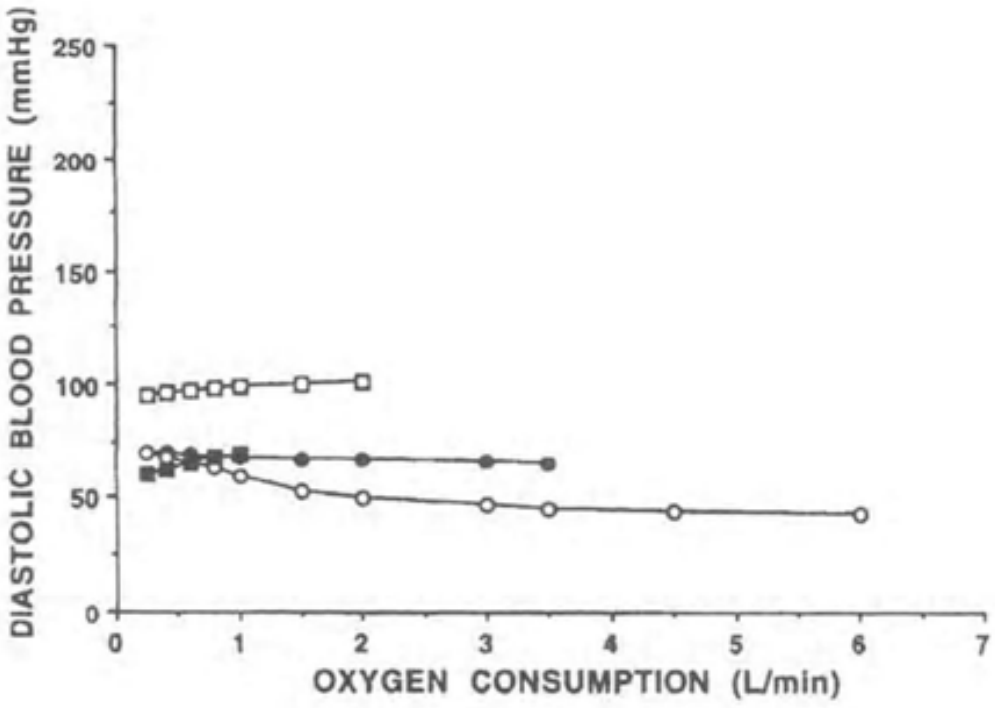


FIG. 9

Diastolic blood pressure response to exercise in the 4 individuals. ○, athlete; ●, sedentary; □, cardiac transplant; ■, quadriplegia.

CardioVascular Medicine Notes
Hypertension & Shock

Hypertension:

- **What is it?:**
  - 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
  - Big problem in Aus – 2.2Mil People → $1Bilion/Year
  - *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

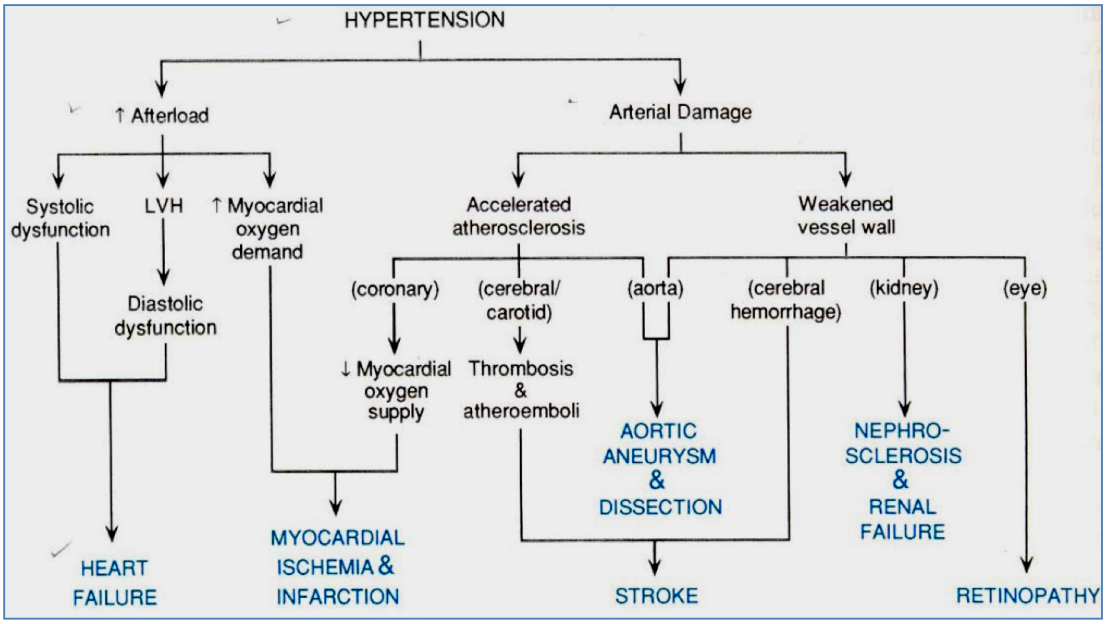
- **Classifications (In Adults):**
  - **Different Classes Based on BP Ranges:**

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

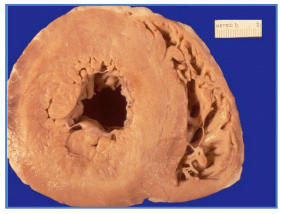
- **2 Types of Hypertension:**
  - (Based on Aetiology.)
  - **1. Primary (Essential) Hypertension:**
    - 90-95% of cases
    - No specific cause.
    - But Related to:
      - Obesity
      - ↑Cholesterol
      - Atherosclerosis
      - ↑Salt Diet
      - Diabetes
      - Stress
      - Family History
      - 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

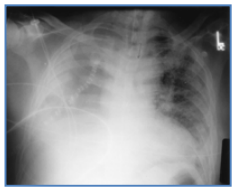
- **Organ Damage Caused By Hypertension:**
  - Relationship between *Degree* of hypertension & *Degree* of Complications.



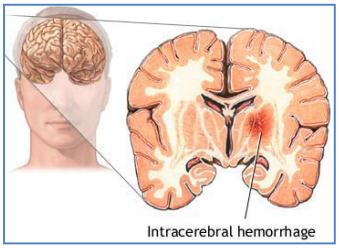
- **Heart:**
  - **Increased Afterload:**
    - ↑ Workload of Heart → ↑Afterload → Pumps Harder → Hypertrophy → Failure
  - **L-Vent. Hypertrophy:**
    - To compensate for higher workload
    - → Compromised L-Ventricular Volume → ↓Stroke Volume →↓Cardiac Output



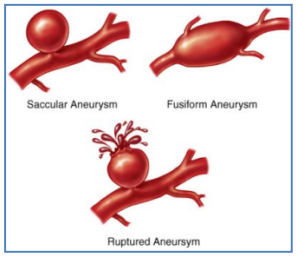
- **Lungs:**
  - **Pulmonary Congestion:**
  - Backing up of blood in Pulmonary Circuit.
  - **Why:** ↑BP = ↑Aortic-BP = ↑Afterload = ↓SV = ↑ESV = ↓Pulmonary Blood Flow

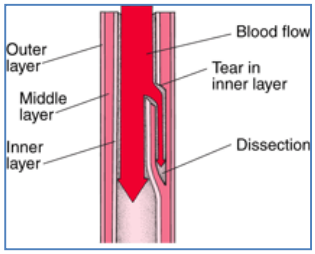


- **CerebroVascular:**
  - Stroke – Typically *Intracerebral Haemorrhage*
  - Rupture of Artery/Arterioles in brain

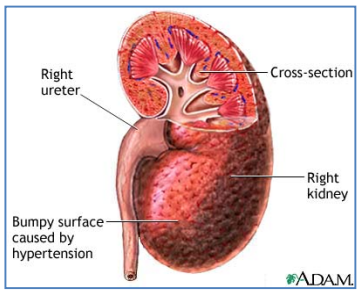


- **Aortia/Peripheral Vascular:**
  - Arterial Mechanical Damage (eg. Aneurysms/Dissecting Aneurysms)
  - Accelerated Atherosclerosis

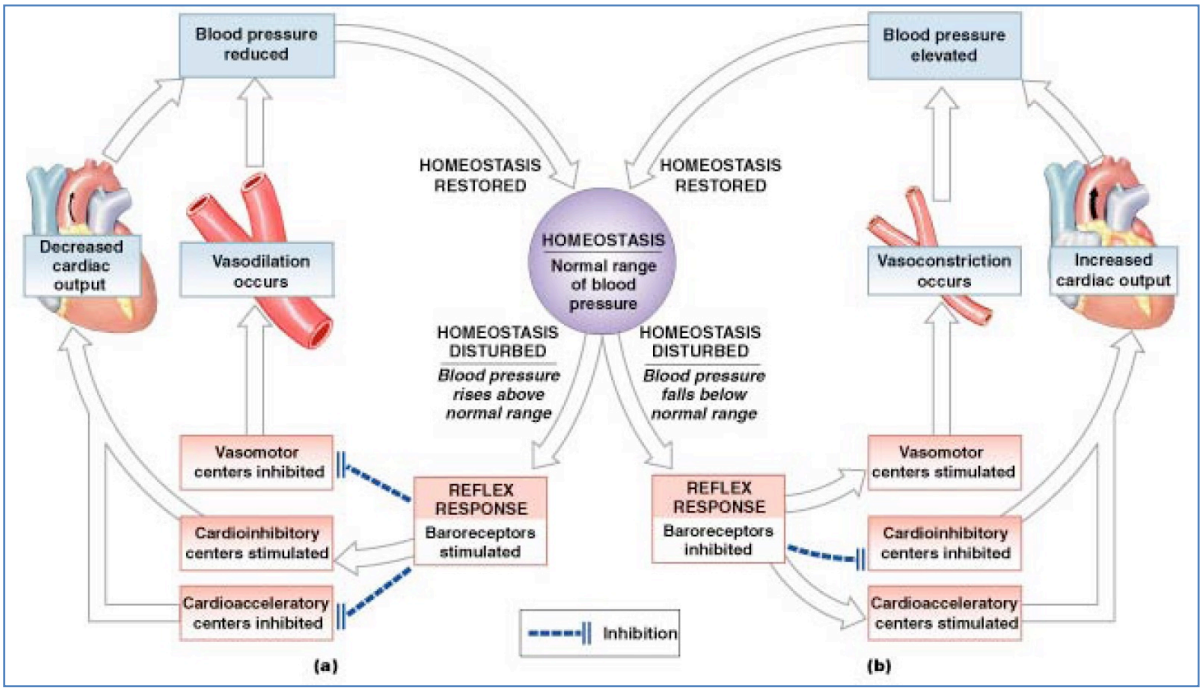


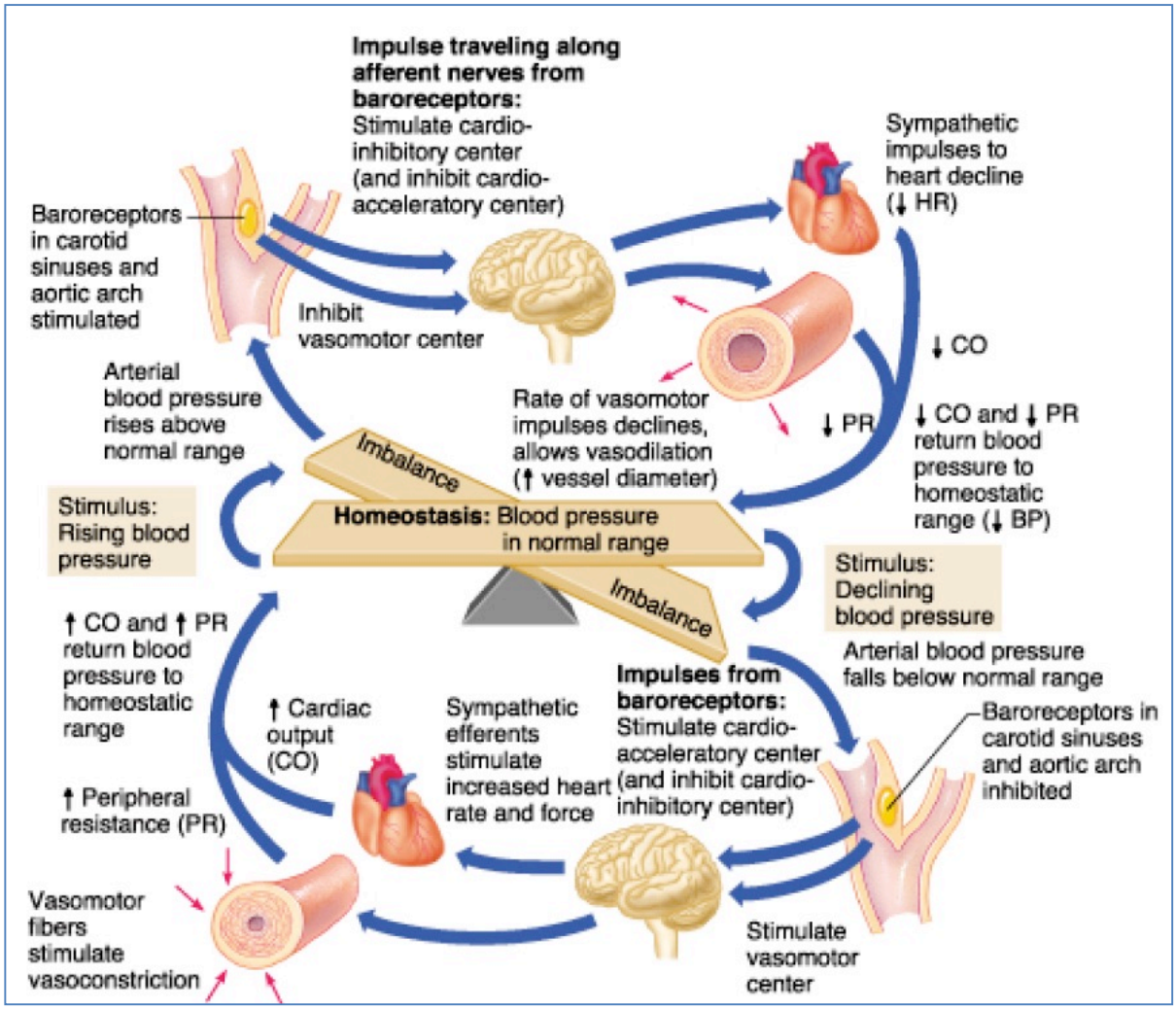


- **Kidneys:**
  - Nephrosclerosis – (hardening of kidney blood vessels)
  - Renal Failure



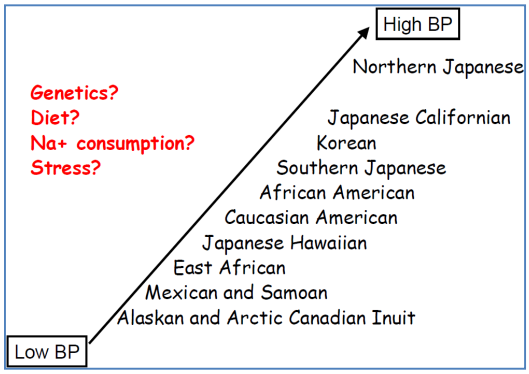
- **Short-Term Control of BP:**
  - **The Baroreceptor Reflex:**





- **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

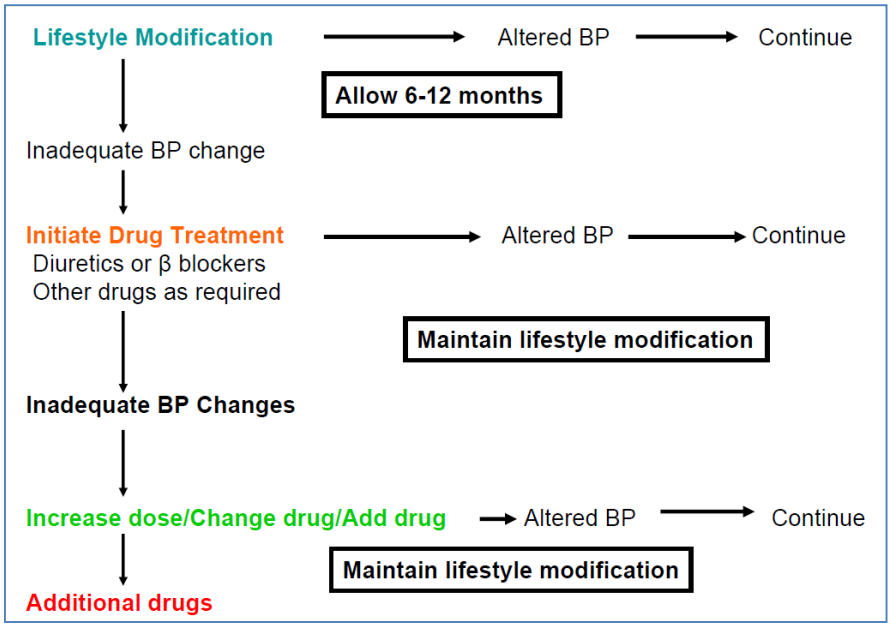
  - **Race:**



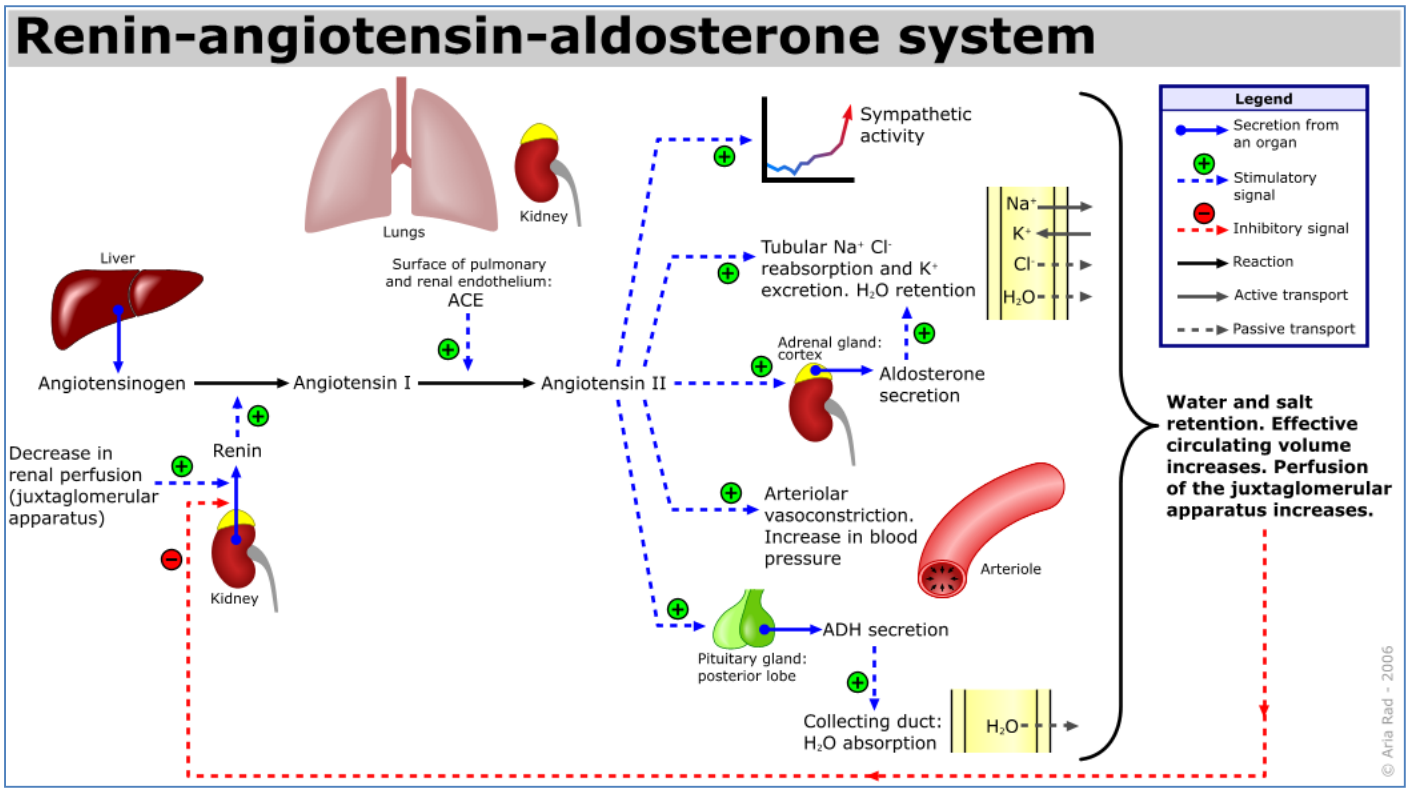
  - **Obesity:**
    - Fatty Diet → Atherosclerosis
    - Body Fat → kms more vessels → ↑Peripheral Resistance → Hypertension
    - Physical Weight of fat – may impede venous return
    - Kidney Dysfunction → Loss of long-term BP (Blood Volume) Control.

  - **Excess Na^+ Intake:**
    - **If Normal Kidney Function:**
      - Na^+ intake → Slight BP increase (due to fluid retention)
      - But *Excess* Na^+ & H_2O excreted by kidneys → BP returns to normal.
    - **If Impaired Kidney Function:**
      - Na^+ intake → Larger BP increase...
      - Because *Excess* Na^+ & H_2O *Not* excreted by kidneys (less efficiently)

- **Basic Hypertension Treatment Plan:**



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



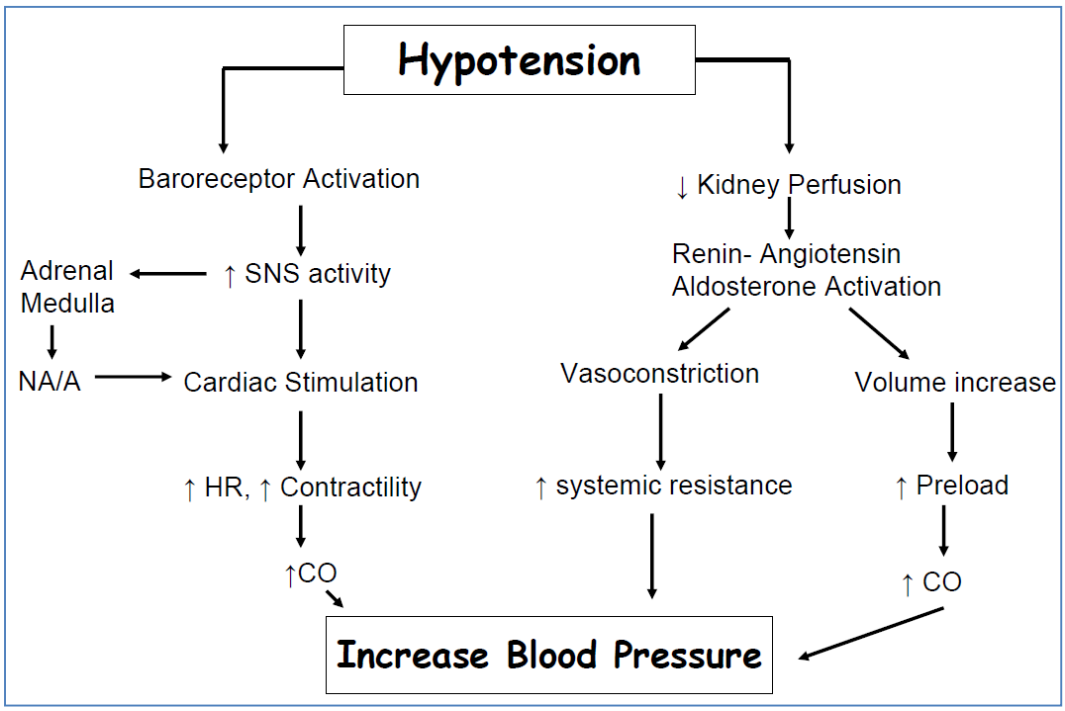
↑....For your own Interest....↑

Shock:

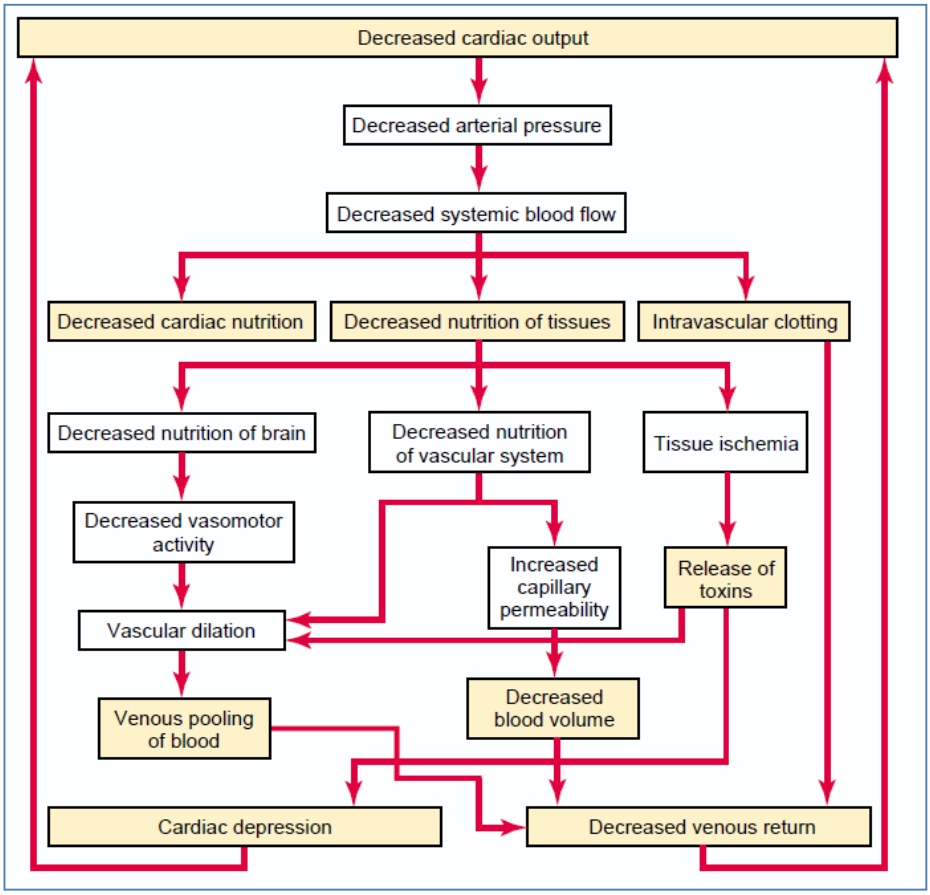
- **What is it?:**
  - Profound Haemodynamic/Metabolic Disorder due to Inadequate Blood Flow & O_2 Delivery.

- **Common Causes:**
  - **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.

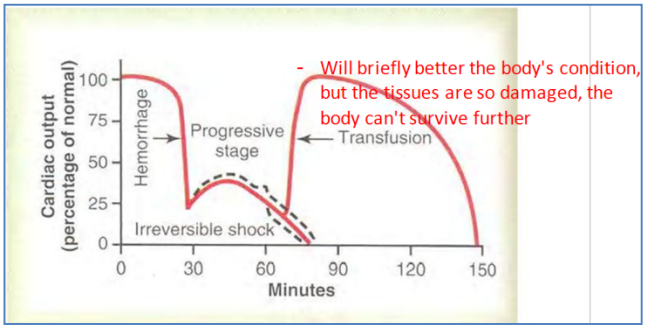
- **3 Stages of Shock:**
  - **1. Non-Progressive:**
    - Stable, not self-perpetuating.
    - **Symptoms:**
      - Hypotension (Low BP)
      - Tachycardia (High HR – body's attempt to compensate for poor perfusion)
      - Tachypnoea (High Breathing-Rate – Phrenic Nerve Stimulation – Diaphragm)
      - 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, viscous 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 O_2 Deficit to Heart)
    - Vasomotor Failure (due to O_2 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.**

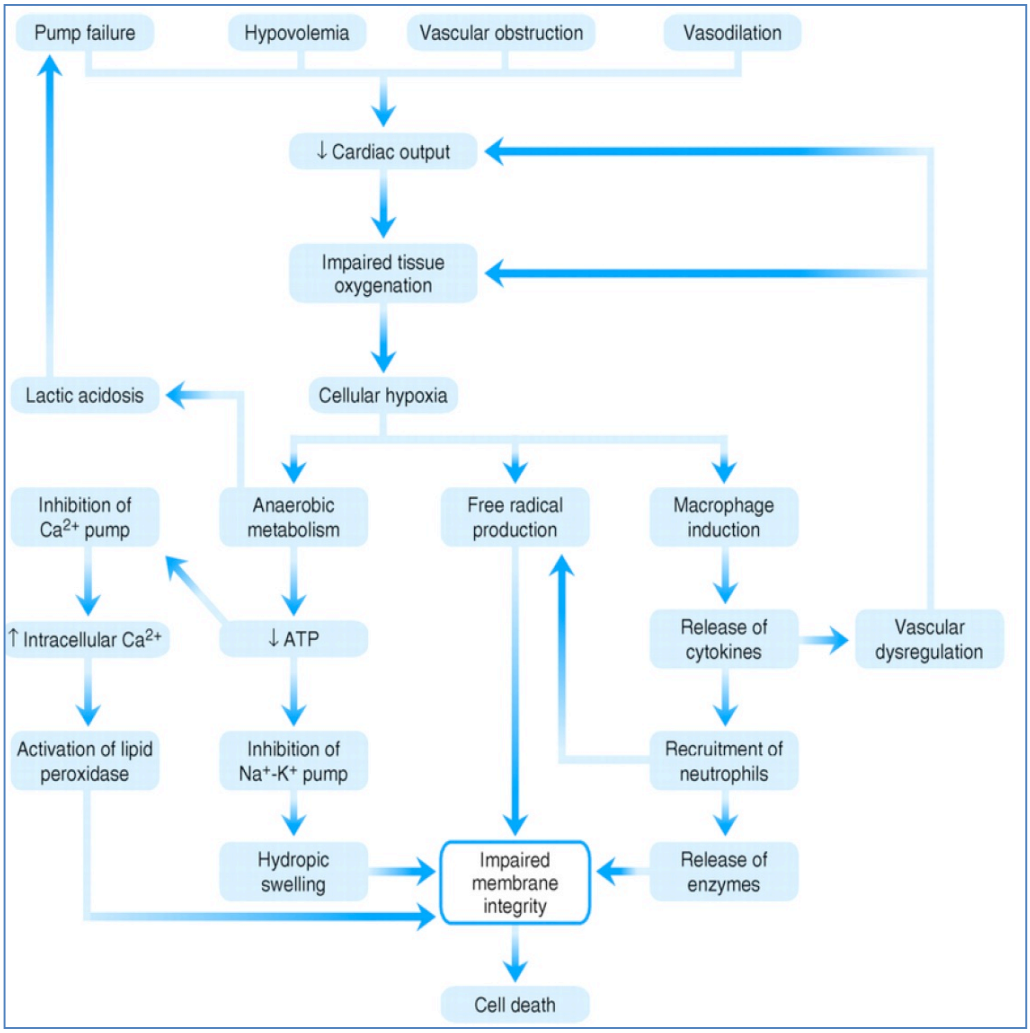


- **3. Irreversible Stage:**
  - Advanced stage where the body is irrecoverable.
  - Usually any form of therapy is ineffective.
    - Eg. Transfusion is ineffective because the tissue/organ damage is too advanced.
  - **Symptoms:**
    - Organ Dysfunction (Renal/Cardiac/Pulmonary/CNS)
    - Renal Failure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



Shock-Induced Cell Death

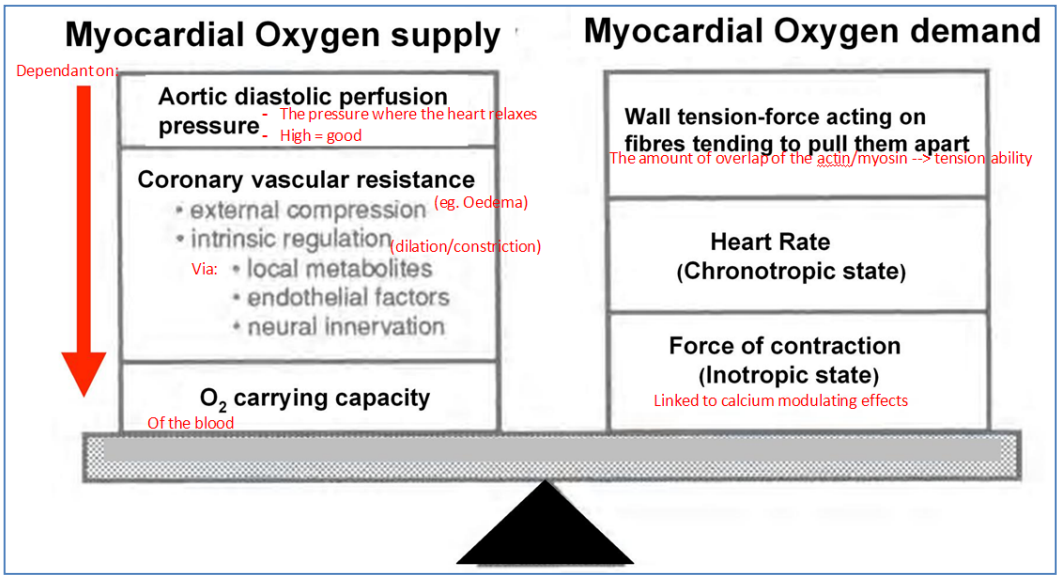
- Self Perpetuating Cascade



CardioVascular Medicine Notes
Myocardial Ischaemia

'Ischaemia':

- = *Restraint of Blood* (Ie. Insufficient Blood)
- Leads to *Imbalance* Between Oxygen Supply & Demand.
- **Oxygen Supply – Increased By:**
  - **↑Coronary Blood Flow:**
    - **↑Aortic, Diastolic Perfusion Pressure:**
      - Aortic Pressure During L-Ventricular Diastole
      - If High → ↑Coronary Perfusion
      - Influenced by:
        - Hypotension
        - Aortic Regurgitation
    - **↓Coronary Vascular Resistance:**
      - Resistance to Coronary Blood Flow
      - Depends on Vascular Diameter...
      - Influenced by:
        - External Compression (eg. Oedema)
        - Intrinsic Regulation (Dilation/Constriction).
          - Metabolites
          - Neural
  - **↑O_2-Carrying Capacity of Blood:**
    - Influenced By:
      - Hb Saturation
      - Hb Levels (Anaemia)
      - Blood pH
      - CO Poisoning
      - Lung Disease
- **Oxygen Demand – Increased By:**
  - **↑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.*
  - **↑Heart Rate (Chronotropic State)**
  - **↑Force of Contraction (Inotropic State)**

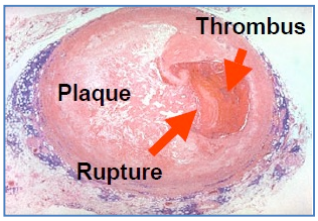


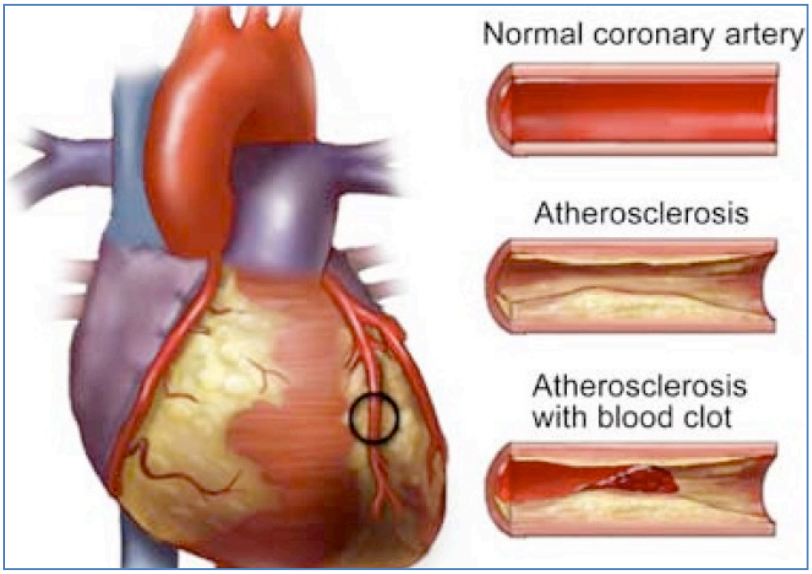
***Ischaemia Vs. Hypoxia Vs. Infarction:**

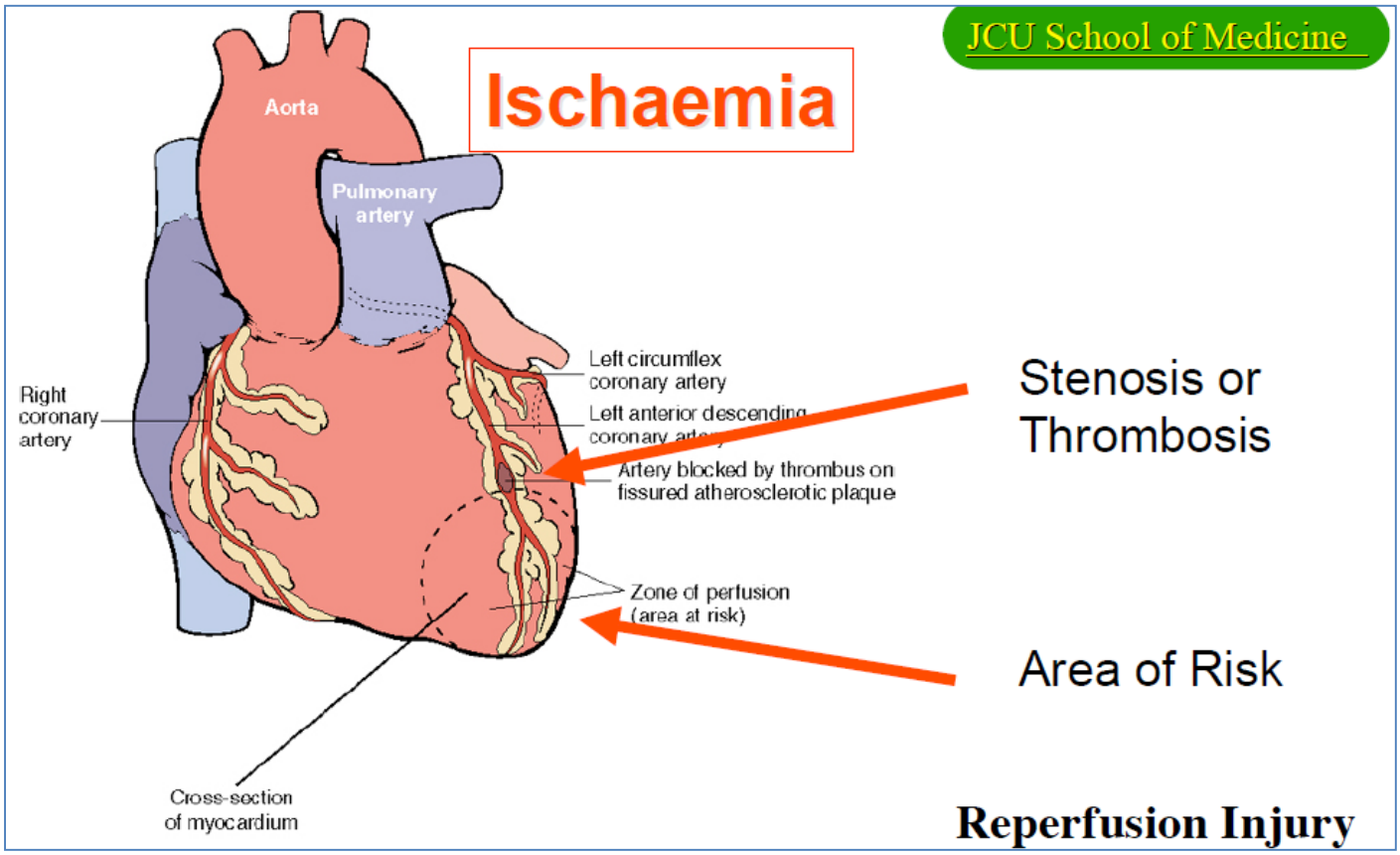
- **Ischaemia:** A *'FLOW' Limitation*, Typically due to Coronary Artery Stenosis (Narrowing)
- **Hypoxia:** An *'O_2' Limitation,* Typically due to High-Altitude/Respiratory Insufficiency/etc.
- **Infarction:** *Irreversible Cell-DEATH*, Typically due to sustained Ischaemia.
- NB: Ischaemia can lead to Hypoxia & Infarction.

Myocardial Ischaemia:

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

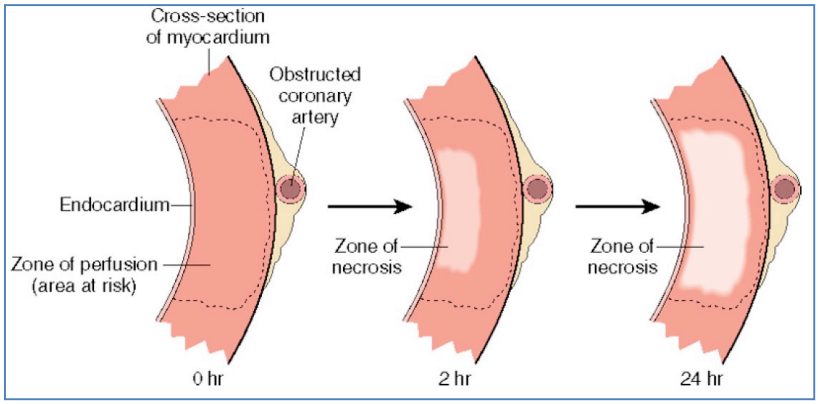




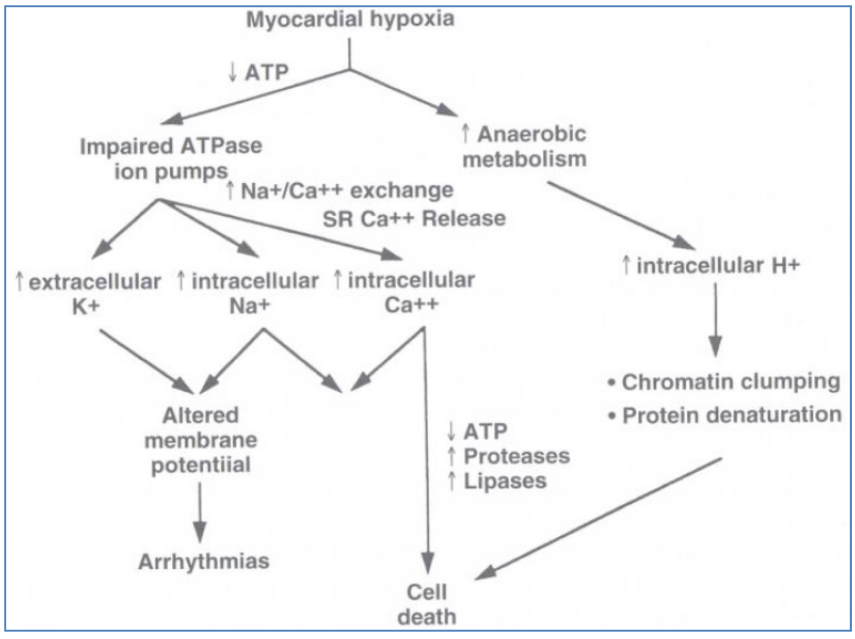


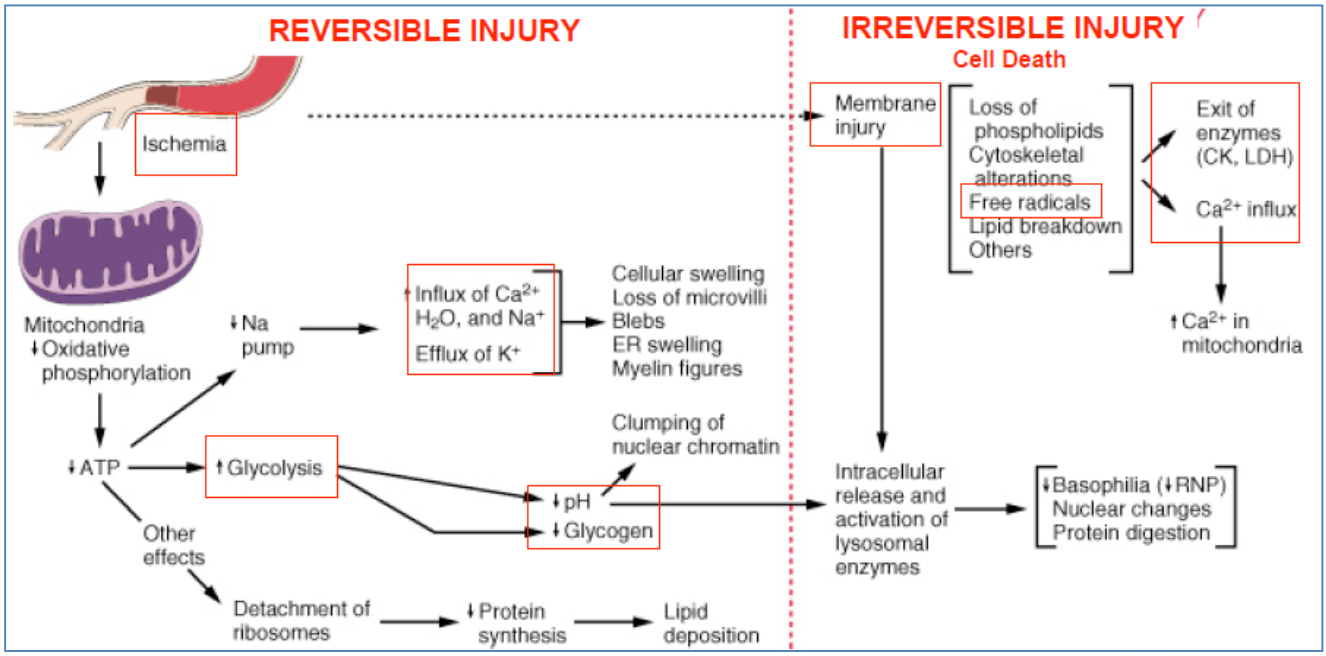
What Happens During Myocardial Ischaemia:

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



- **Metabolic Changes – (Aerobic → Anaerobic):**
  - ↑Lactate (Anaerobic Metabolism), ↓pH
  - ↓ATP, ↑ADP, ↑P_i
  - ↓Glycogen
- **Pain:**
  - Nociceptor (pain receptor) Activation → Angina Pain
- **Acute Ischaemic Attack:**
  - SNS & PNS Stimulation → Tachycardia, Sweating, Nausea.....
- **Reversible Cell Injury:**
  - ↓Blood-Flow → ↓Myocardial Relaxation (diastolic) → Stiffening of L-Ventricle → ↑LVDP
- **Reperfusion Injury:**
  - Cell Damage that occurs When Blood Supply is Restored (after being stopped)
  - Due to inflammation and oxidative damage through the induction of oxidative stress.
- **Pulmonary Congestion:**
  - Stiffening of L-Ventricle & ↑LVDP → ↑Pulmonary Vascular Pressure
    - → Pulmonary Congestion
    - → Shortness of Breath
- **Ventricular Arrhythmias:**
  - **Due to Myocyte Ion-Disturbances:**
    - ↑ Extracellular K^+
    - ↑ Intracellular Na^+
    - ↑ Intracellular Ca^+ ("Calcium-Loading") – If Ischaemia is Prolonged → Irreversible Damage
  - → Alters Conduction Patterns of the Heart → Arrhythmias





Clinical Presentations of Myocardial Ischaemia:

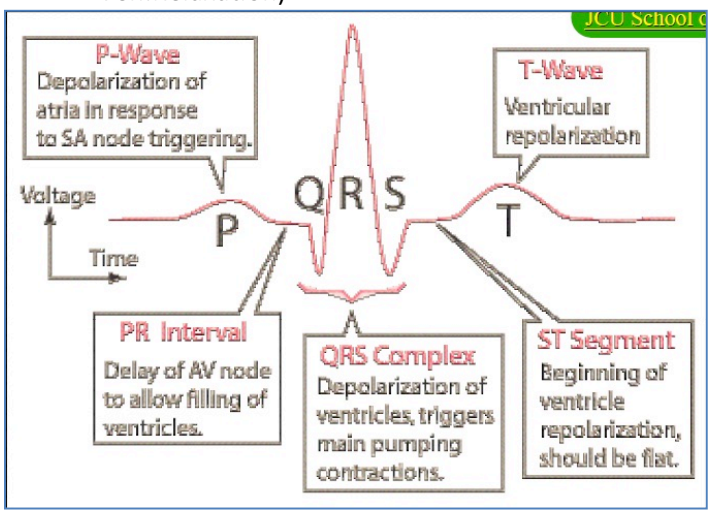
- **Ischaemic Heart Failure:**
  - Weakness of Heart Muscle → Difficulty Breathing + Peripheral Oedema
- **Angina Pectoris:**
  - Substernal/Precordial Chest Pain – Due to Myocardial Ischaemia → No Cell Necrosis
  - Pain Usually lasts up to 15min.
  - **3 Subtypes:**
    - **Stable Angina (Typical):**
      - 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:**
  - *No Pain*
  - Abnormal ECG (ST-Elevation)

Prolonged Ischaemic → Irreversible Damage → Leads to:

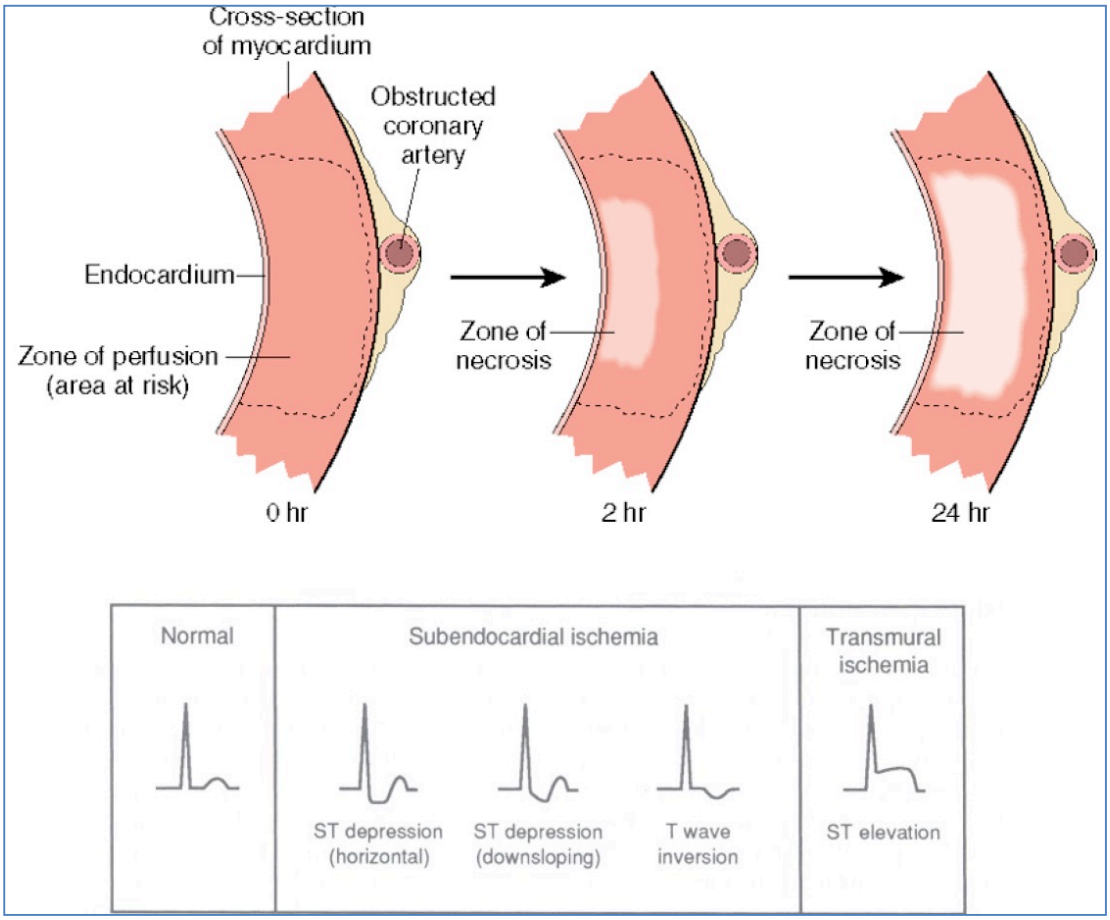
- **Ca^+ Loading Within Cell:**
  - Ca^+ Recycling Cycle (between Sarcoplasmic Reticulum, Sarcoplasm & Actin) Changes.
  - *Marks the transition between Reversible & Irreversible Damage.*
- **Heart Failure – Due to:**
  - Lethal Arrhythmias
  - ↑LVDP → Pulmonary Congestion → R-Heart Failure.
- **Infarction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

ECG Changes Due to Ischaemia:

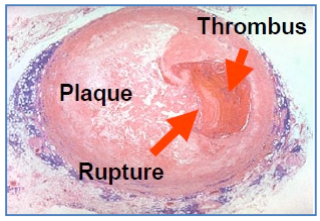
- **Normally:**
  - QRS = Ventricular Depolarisiation
  - T-Wave = Ventricular Repolarisation
    - NB: Ven.Repol – Very sensitive to myocardial perfusion. (ie. Lack of blood supply alters Ven.Relaxation)



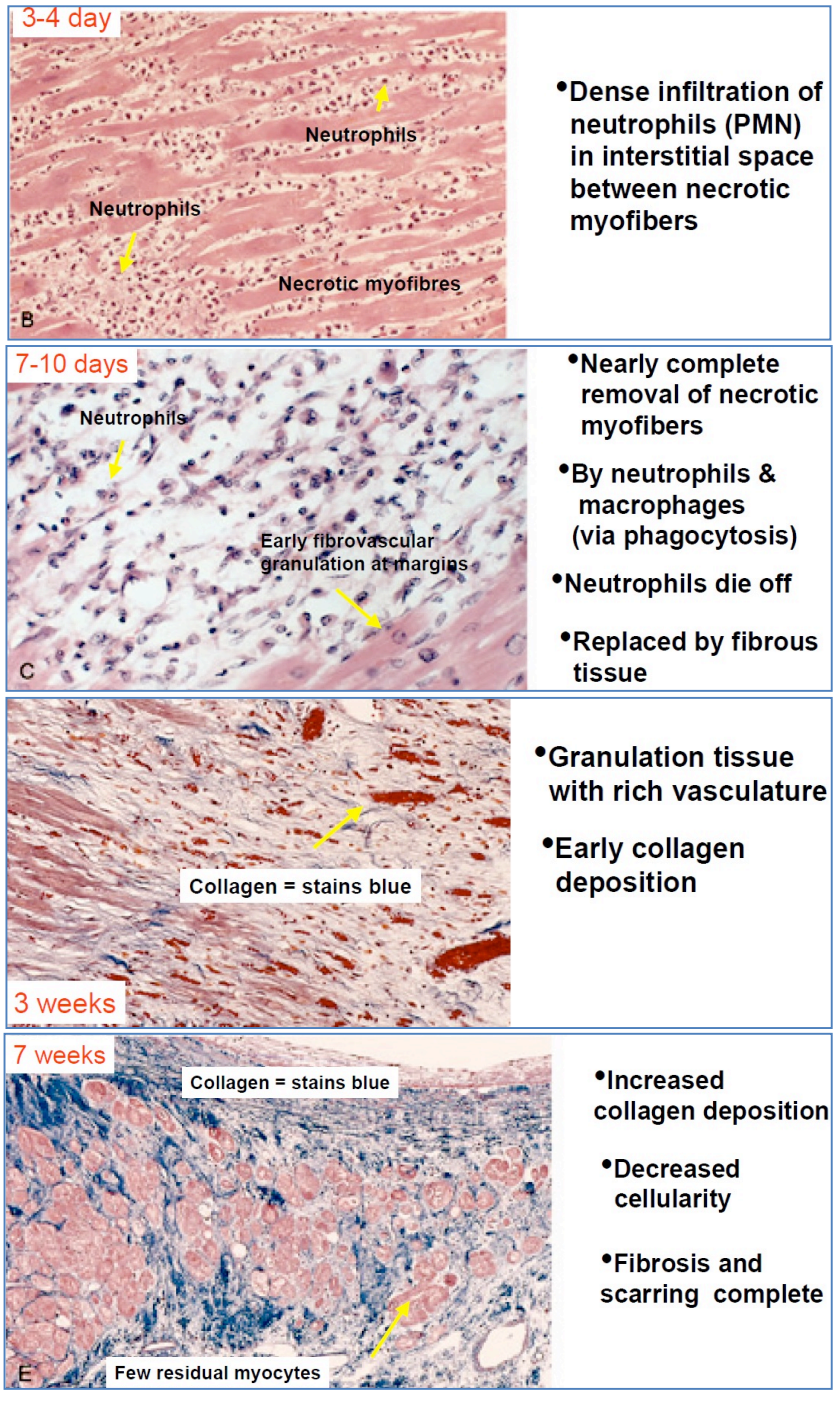
- **Subendocardial Ischaemia:**
  - Poor Perfusion → Altered Ven.Repolarisation →
    - ST-*Depression*
    - T-Wave Inversion
- **Transmural Ischaemia:**
  - Full-thickness of the heart wall is damaged → Altered Ven.Repolarisation →
    - ST-*Elevation*

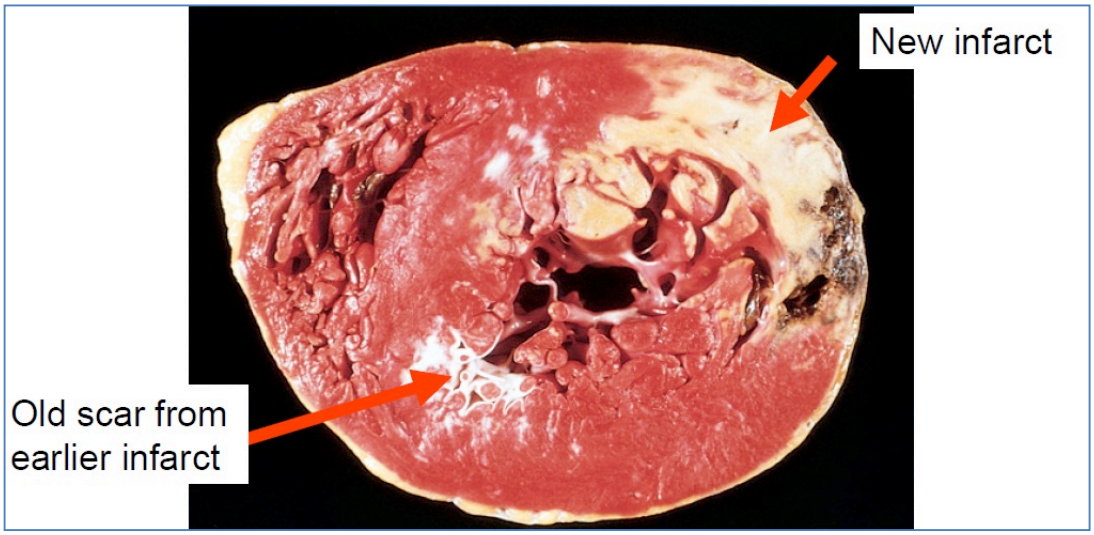


Myocardial Infarction (Heart Attack):



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




New infarct
Old scar from earlier infarct

CardioVascular Medicine Notes
The Ageing Heart

What Happens in an Normal Ageing Heart?:

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

- **NB: 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?**
  - **1.** The above changes may interact with each other → Heart Failure
    - Eg. Decreased Myocytes (contractility) + Valve Calcification → ↓SV → Heart Failure
  - **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.**

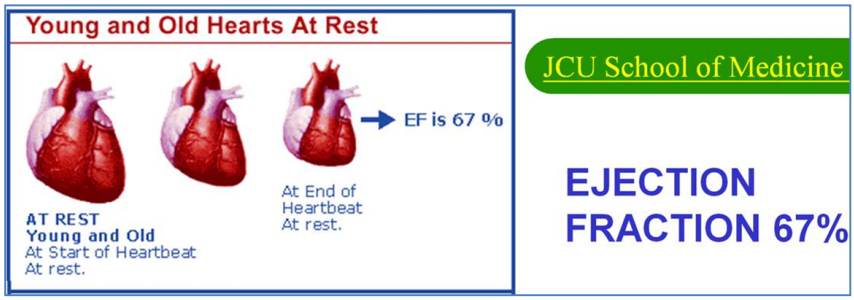
Healthy 20yr-old Vs. Healthy 80yr-old (At Rest)

- **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.

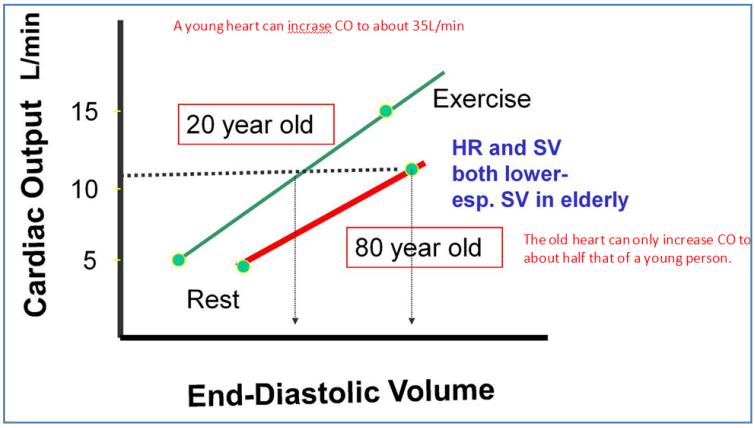
- **Same *Resting* Cardiac Output:**
  - Ie. An Old Heart pumps out the same amount of blood/min (at rest) as a Young Heart.
  - **...How?:**
    - Older hearts have a 10% Higher Stroke Volume + but 10% Lower Heart Rate → Same Cardiac Output (At Rest) compensates for its ↓Contractility by Dilating more 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.
    - NB: 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

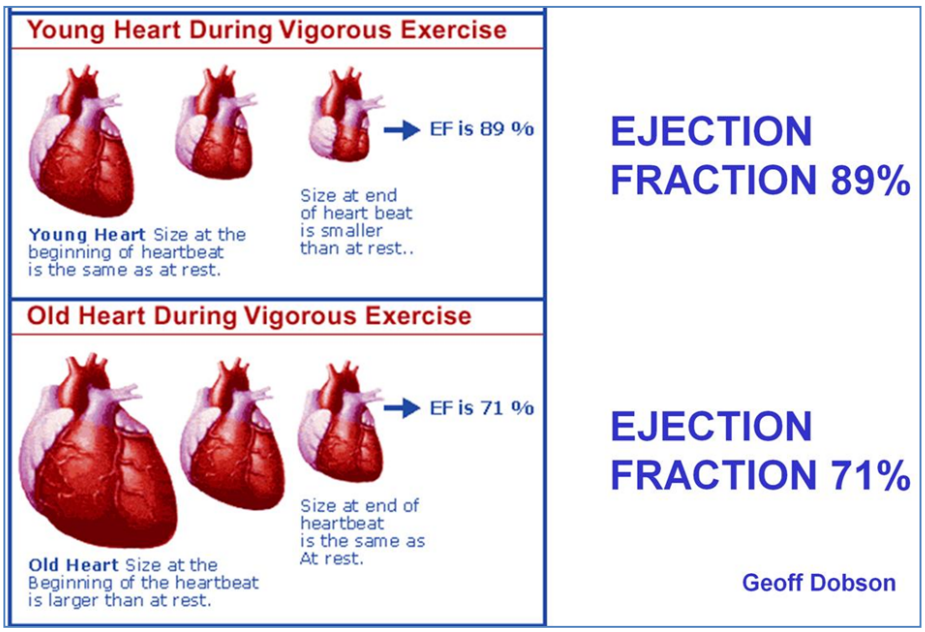
Max HR of 80 yr old is 25% lower than 20 yr old

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

- **25% Lower Cardiac Output (During Exercise) in Older Heart:**
  - Primarily Due to decreased heart rate. NB: 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 VO_{2max} (Max O_2 Consumption) in Older Person:**
  - Old Person's VO_{2max} is half that of a Young Person.
  - **Due to:**
    - Lower Muscle Mass (Ie. Less muscle uses less energy → ↓O_2 Consumption)
    - Changes in Muscle Metabolism (↓enzyme efficiency/manufacture etc.)
    - Decreased Number of Mitochondria/Cell.

- **Lower Max. Ejection Fraction (During Exercise) in Older Heart:**
  - Young heart can increase its ejection fraction from 67% → 89%.
    - - by ↑Contraction & ↑Heart Rate.
  - 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 Volume → Increasing Stroke Volume → Increasing Cardiac Output.

- **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.

- **NB:** 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 O_2 Consuption
  - ↑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.**

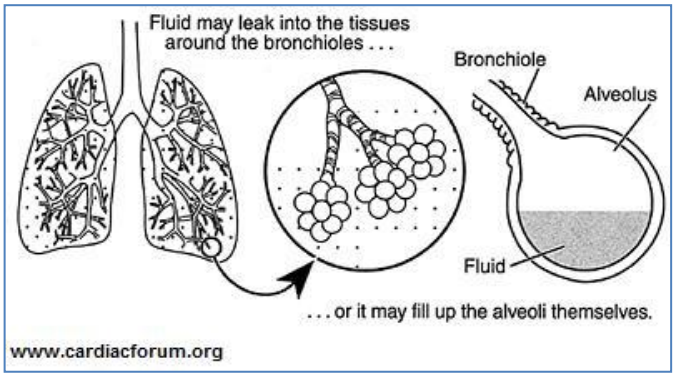
Combating CV-Ageing with Pharmaco-Therapies:

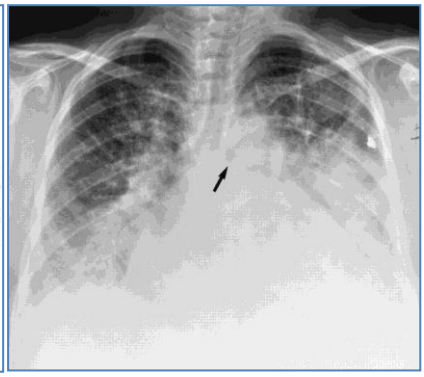
- Drugs that ↑Vascular Compliance
- Drugs that reduce Cardiac Fibrosis
- Drugs that reduce Ventricular Hypertrophy
- Antioxidants – Prevents damage due to free radicals
- Anti-Inflammatory Drugs – CV-Ageing has a small underlying inflammatory component.
- **Exercise**

CARDIOVASCULAR Pathology:
ACUTE CARDIOGENIC PULMONARY OEDEMA

ACUTE CARDIOGENIC PULMONARY OEDEMA

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

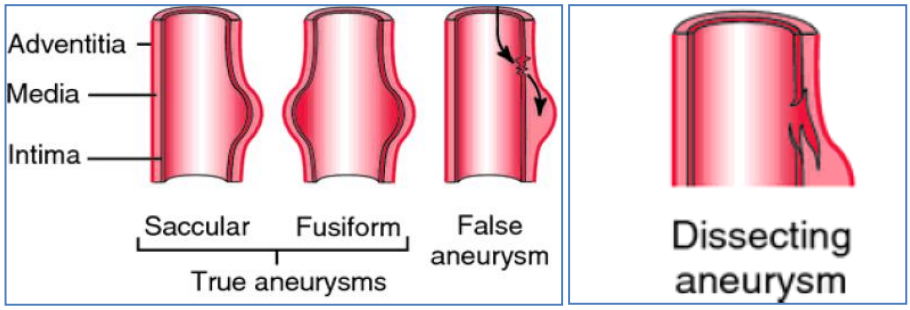




CVS Pathology:
Aneurysms & Dissections

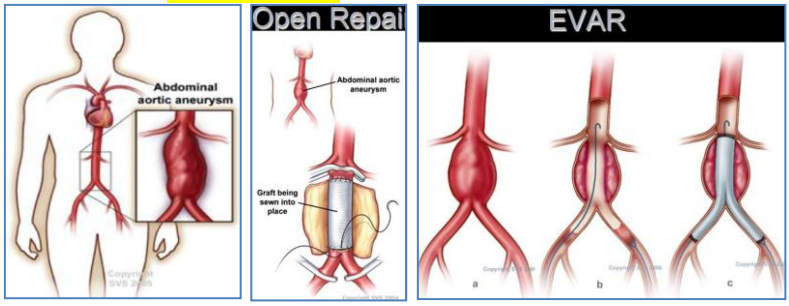
Aneurysms (General Info):

- **Definition:**
  - ***Dr. Martin – "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)
  - ***Robbins – "a Localised abnormal dilation of a BLOOD VESSEL OR THE HEART".***
- **True Vs. Pseudo- Aneurysms:**
  - **True Aneurysms –** *(Full Thickness Aneurysms)*
  - **False/Pseudo Aneurysms –** *(Partial Thickness Aneurysms)*
- **Classification (Size/Shape):**
  - **"Saccular Aneurysms":** Hemispherical Outpouchings involving ONLY PART of the vessel wall
  - **"Fusiform Aneurysms":** CIRCUMFERENTIAL Dilation of a vascular segment
  - **"Dissecting Aneurysms":**Blood within the Arterial wall *itself*.



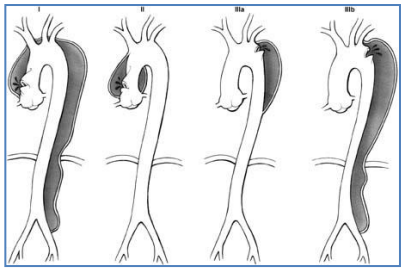
- **Aetiologies:**
  - **Atherosclerosis -** (Typically *AAAs*)
  - **Hypertension -** (Typically *Thoracic Aortic Aneurysms*)
  - **Myocardial Infarction -** (Typically *Ventricular* Aneurysms)
  - **(Others: Congenital – Eg. Downs/Marfan's/Ehlers-Danlos Syndrome/Conn.Tissue Disorders/Etc)**
- **Risk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

- **ABDOMINAL AORTIC ANEURYSM:**
  - **Aetiology:**
    - Atherosclerosis
  - **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:**
        1. Pulsatile Abdo Mass.
        2. Pain - Back/Flank/Abdo/Groin
        3. DVT (From Venous Compression)
        4. "Trash Foot" – from Thrombo-Emboli
  - **Investigations:**
    - Clinical Suspicion + Examination
    - ****Abdo USS – (100% Sensitive)**
    - CT/MRI
  - **Complications:**
    - **#AAA – (NB: SIZE = #1 Predictor of Rupture):**
      - **Classic Triad of Rupture:**
        1. Sudden Pain – (Abdo/Back)
        2. Shock – (Hypotension/ALOC)
        3. 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 → 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

- **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**
    - Radio-Radial Delay
    - **+/- 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:**
    - **1. Aggressive BP-Reduction (Nitrates + B-Blocker)** → Slows Progression
      - **If Ascending:** EMERGENCY SURGERY
      - **If Descending:** Initial Medical Mx

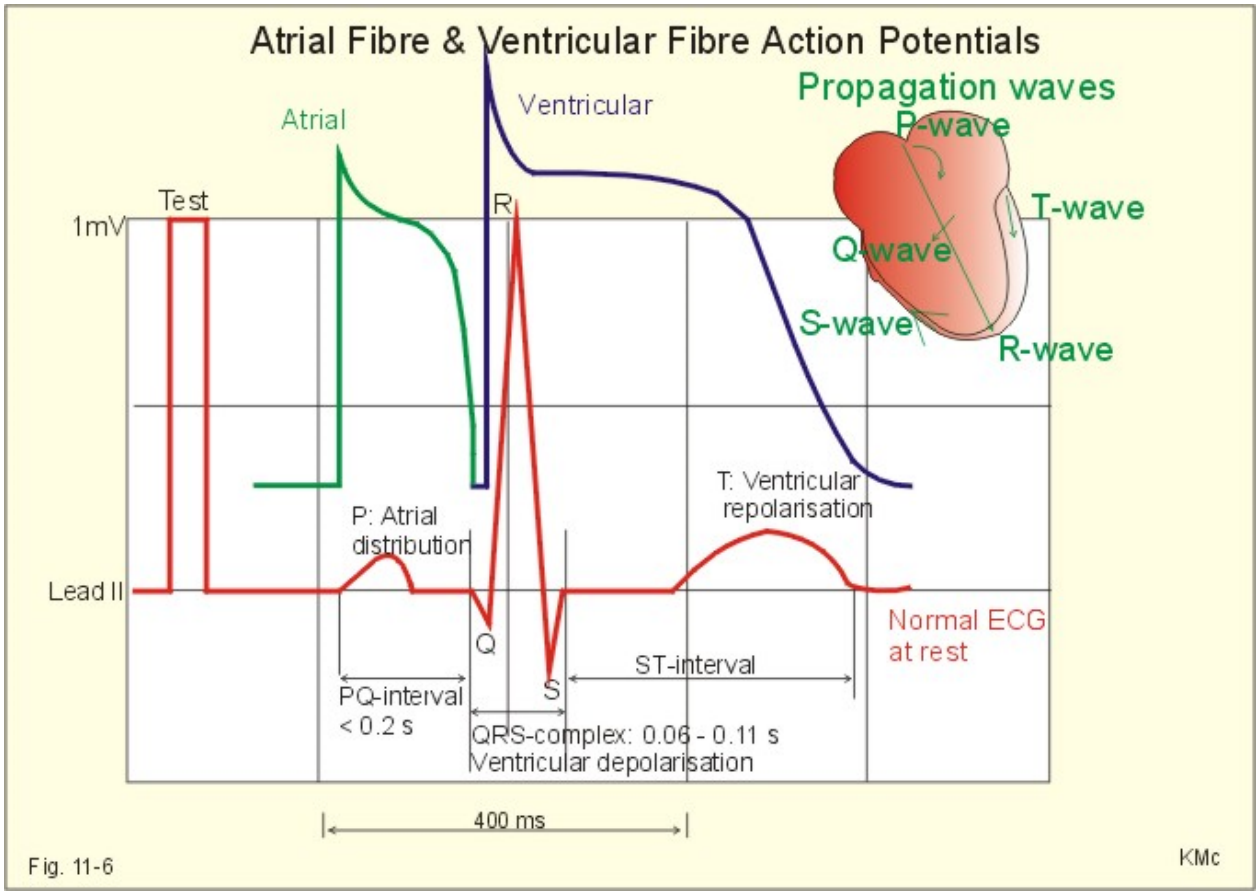


- **CEREBRAL ANEURYSM (Congenital Berry Anerysms – See Sub-Arachnoid Haemorrhage Notes):**
  - **Symptoms for an aneurysm that has not yet ruptured –**
    - Fatigue
    - Loss of perception
    - Loss of balance
    - Speech problems
  - **Symptoms for a ruptured aneurysm –**
    - Severe headaches
    - Loss of vision
    - Double vision
    - Neck and and/or stiffness
    - Pain above and/or behind the eyes.

CardioVascular Medicine Notes
Arrhythmias

Characteristics of a Normal ECG:

- **Sinus Rhythm/Rate:**
  - Between 60-100 bpm.
  - Initiated by SA-Node
  - NB: it's intrinsic rate is higher, but is suppressed by constant Parasympathetic-NS Influence
- **P-Wave:**
  - Rounded
  - Between 0.5-2.5 mm Tall
  - Less than 0.1 Seconds Duration
- **PR-Interval:**
  - Fixed
  - Between 0.12-0.20 Seconds
- **QRS-Complex:**
  - Clean & Sharp
  - Normally Less Than 25mm Tall
  - QRS Interval: Between 0.06-0.12 Seconds Duration
- **Q-T Interval:**
  - Between 0.35-0.45 Seconds Duration
- **S-T Segment:**
  - Normally ≈0.08 Seconds Duration
- **T-Wave:**
  - Prominent
  - Rounded
  - Less Than 5mm Tall (Limb) or Less Than 10mm Tall (Precordial)
  - Between 0.1-0.25 Seconds Duration
- **U-Wave**



Week 13
CardioVascular Medicine Notes
Arrhythmias

Characteristics of a Normal ECG:

- **Sinus Rhythm/Rate:**
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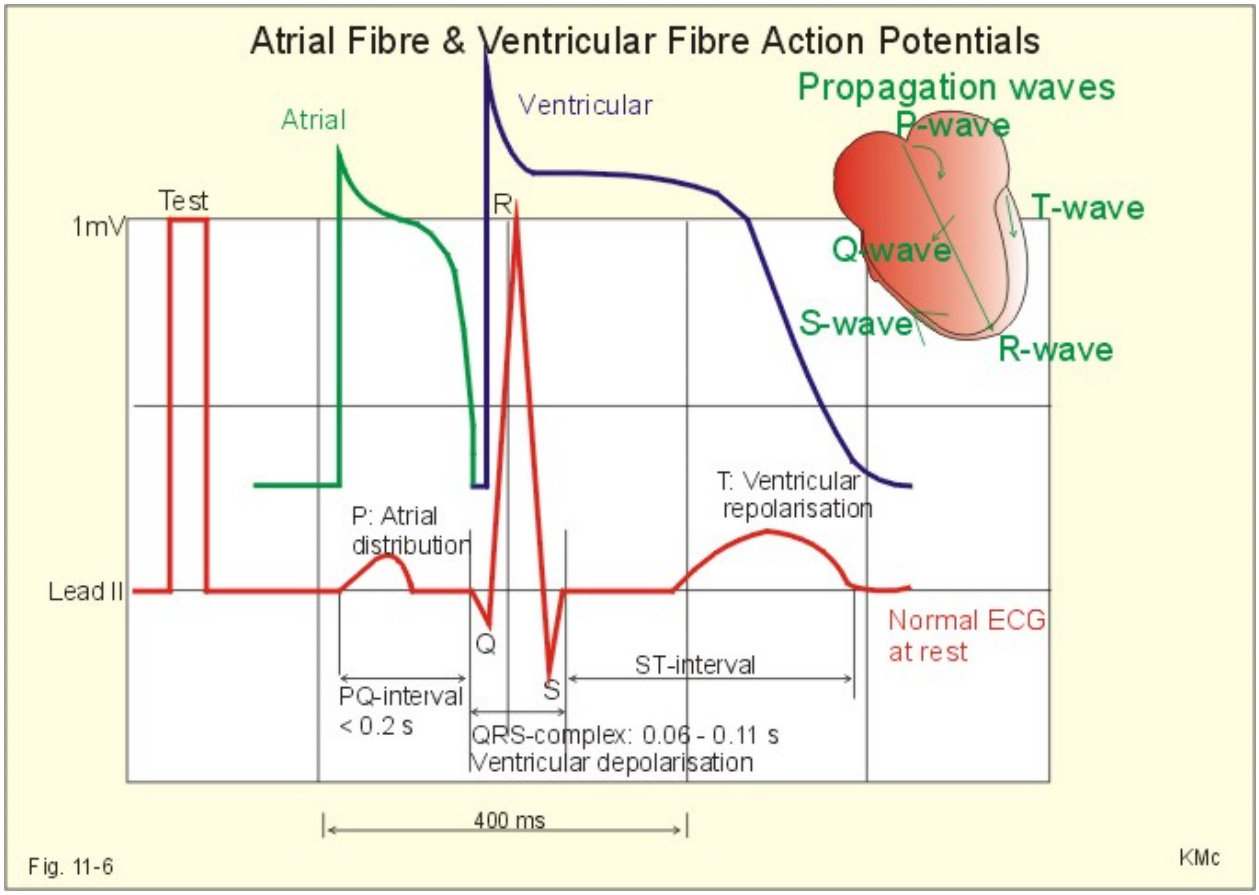


CVS Pathology:
Arrhythmias

BACKGROUND INFO:

- **Working knowledge of ECG and Cardiac Cycle is Essential:**
  - **P Wave =** Atrial Depolarisation
  - **QRS Complex =** Ventricular Depolarisation
  - **T Wave=** Ventricular Repolarisation
  - **Normal Rate ≈** 70bpm
  - **Tachycardia =** >100bpm
  - **Bradycardia =** <60bpm

- **Revision of the Cardiac Cycle:**
  - **P-Wave =** Atrial Systole
  - **QRS-Complex =** Start of Ventricular Systole
  - **T-Wave =** Ventricular Filling
  - **First & Second Heart Sounds:**
    - 1. Lubb (AV Valve Closure)
    - 2. Dupp (Semilunar Valve Closure)

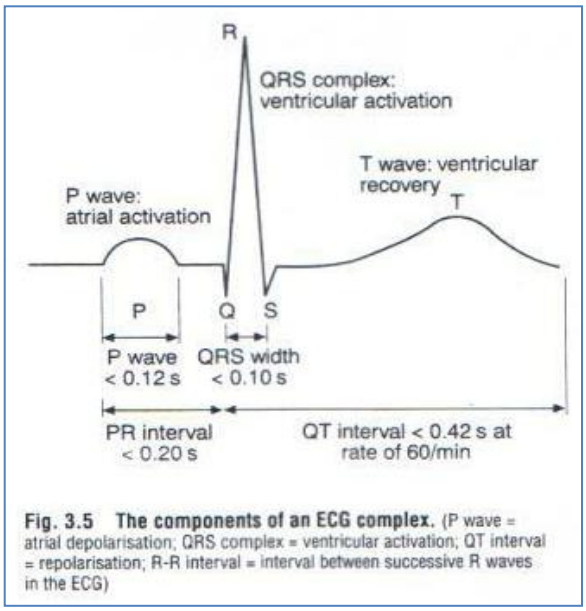
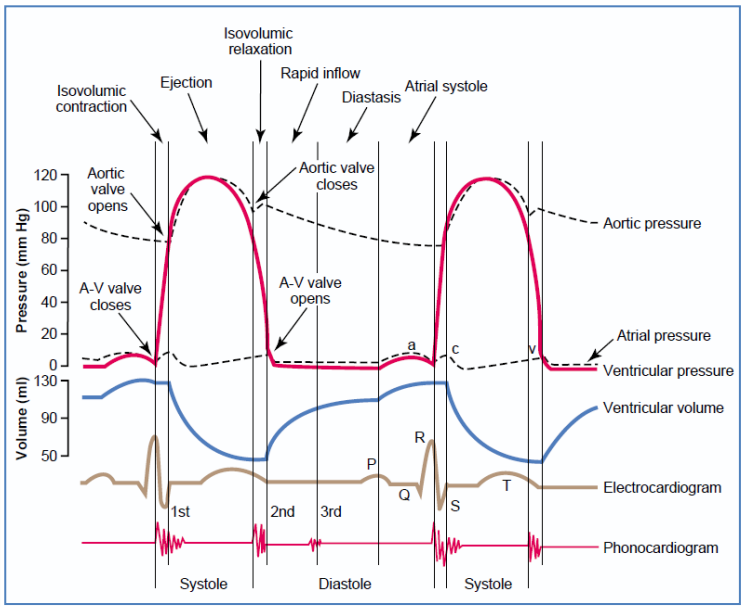


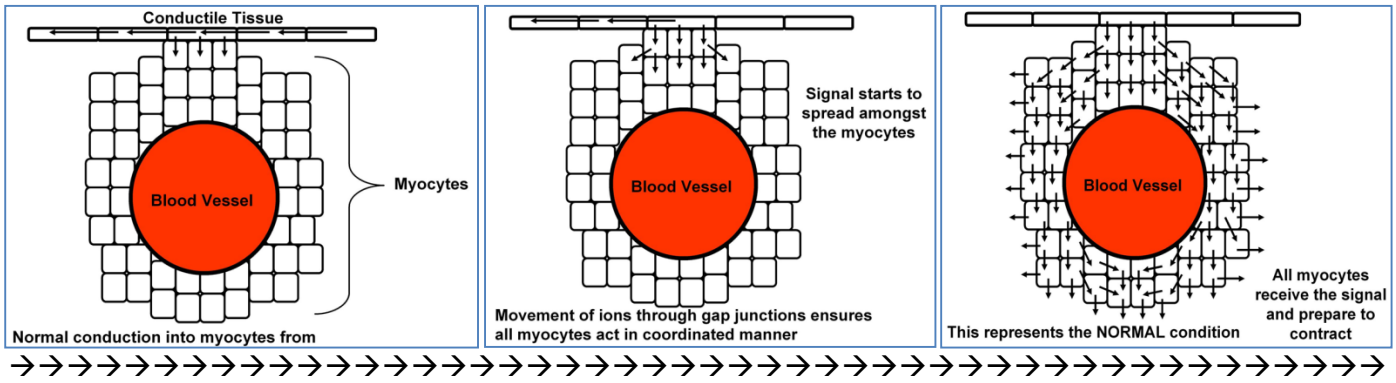
Fig. 3.5 The components of an ECG complex. (P wave = atrial depolarisation; QRS complex = ventricular activation; QT interval = repolarisation; R-R interval = interval between successive R waves in the ECG)



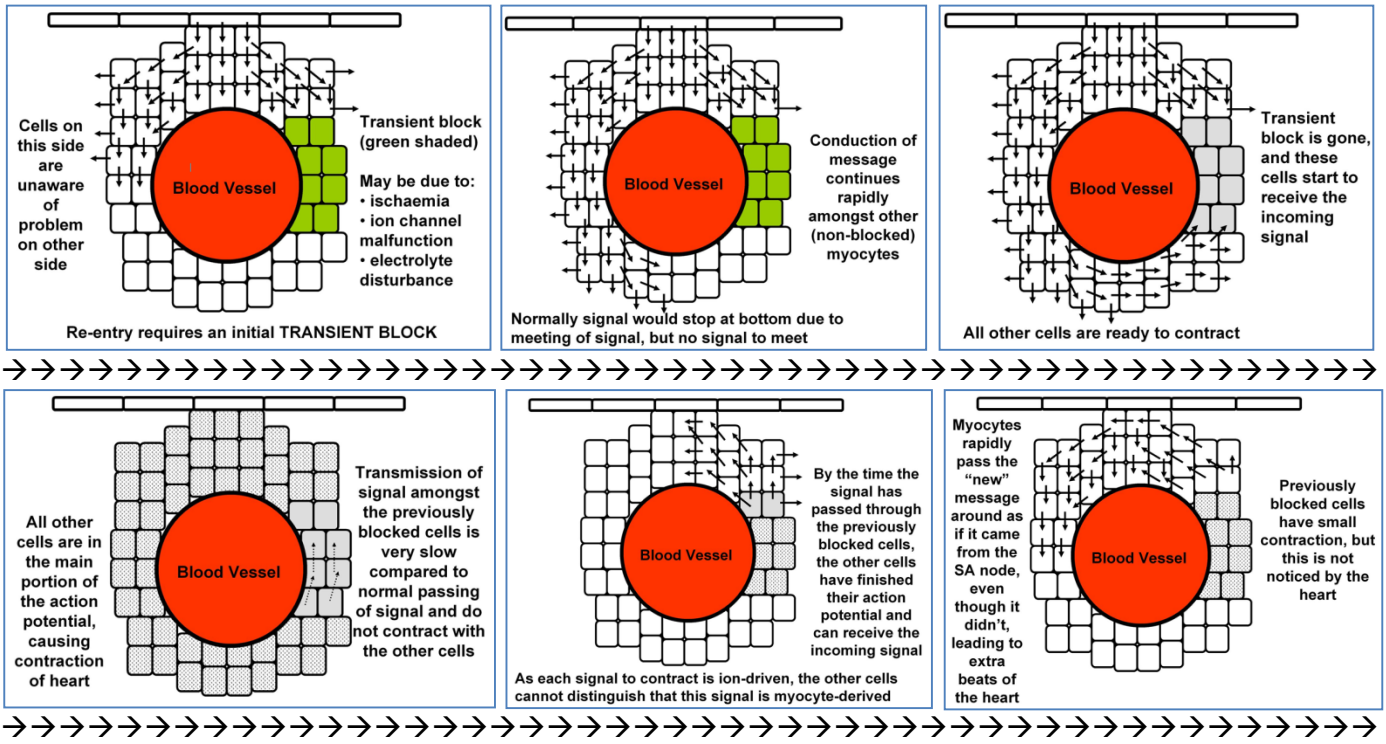
Mechanisms of TachyArrhythmias

Mechanism of Re-Entry:

- 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.
  - NB: Once a cell receives a signal, it won't receive another.

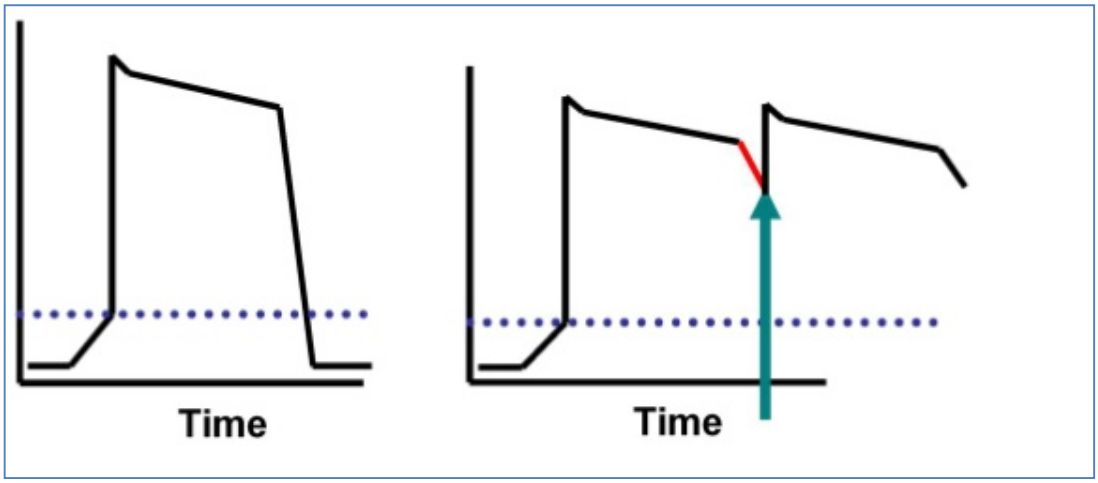


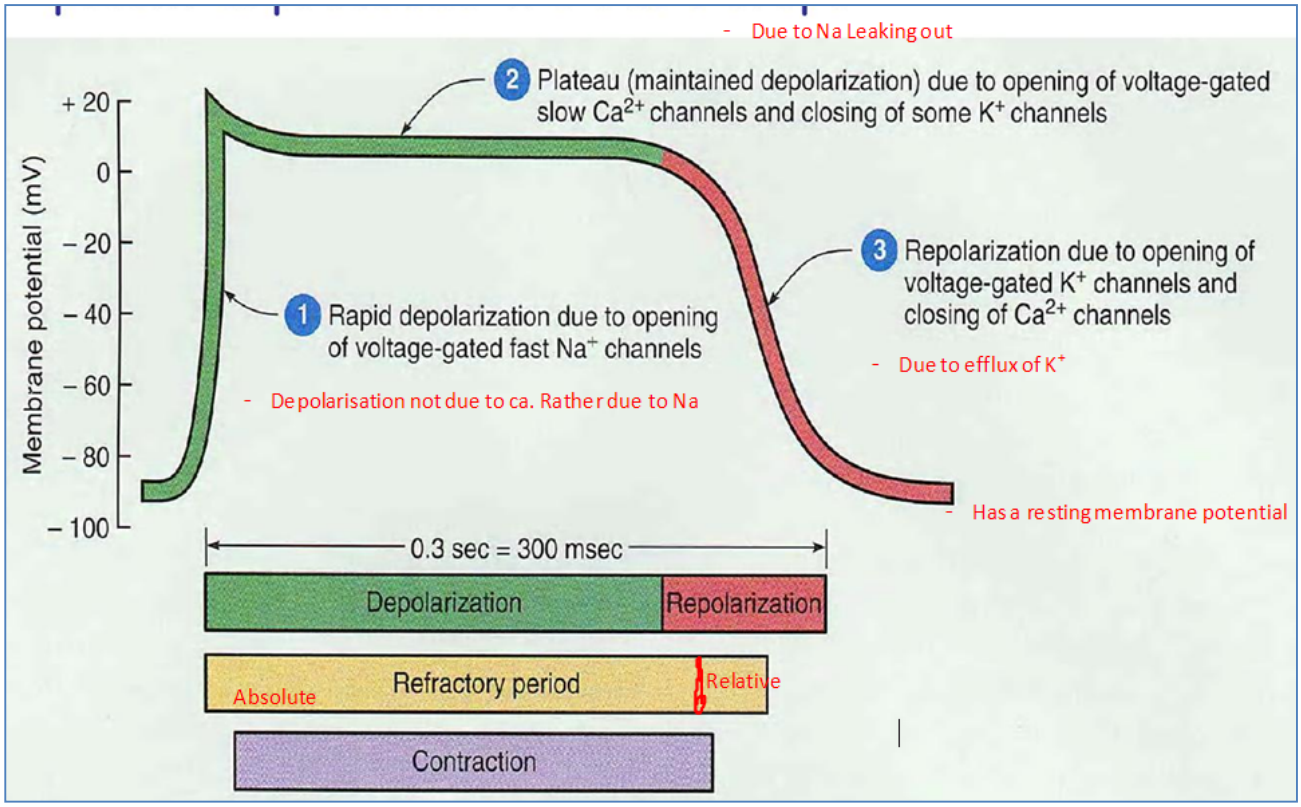
  - However, for Re-Entry to occur, an initial momentary/transient Block is required. See Below:



Early After-Depolarisations:

- 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



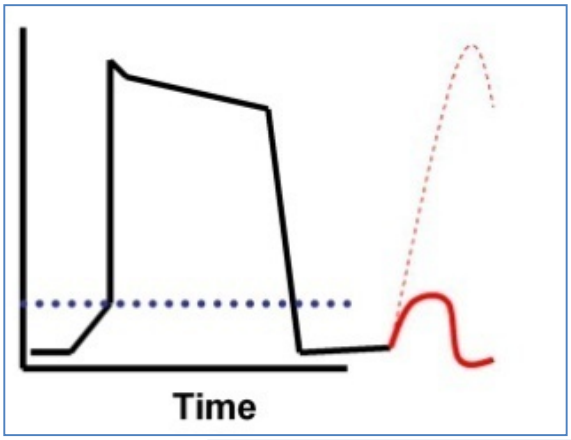


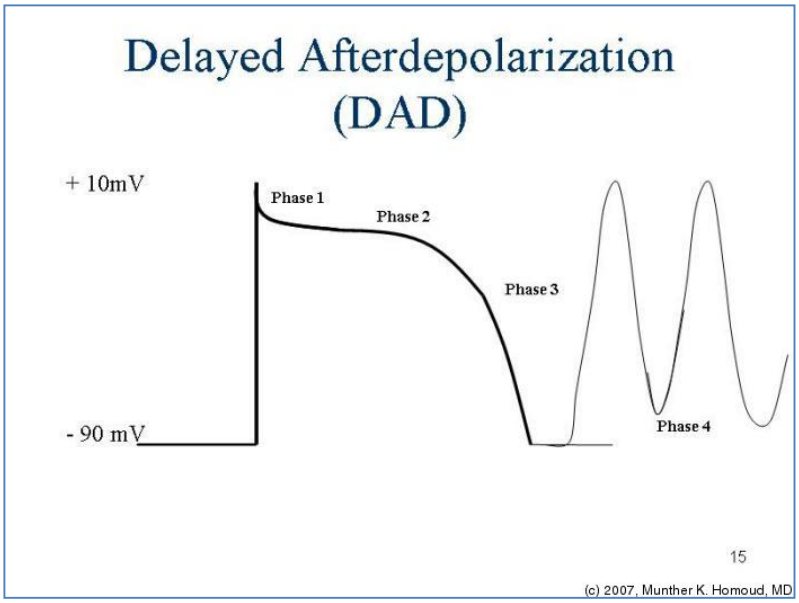
Delayed After-Depolarisations:

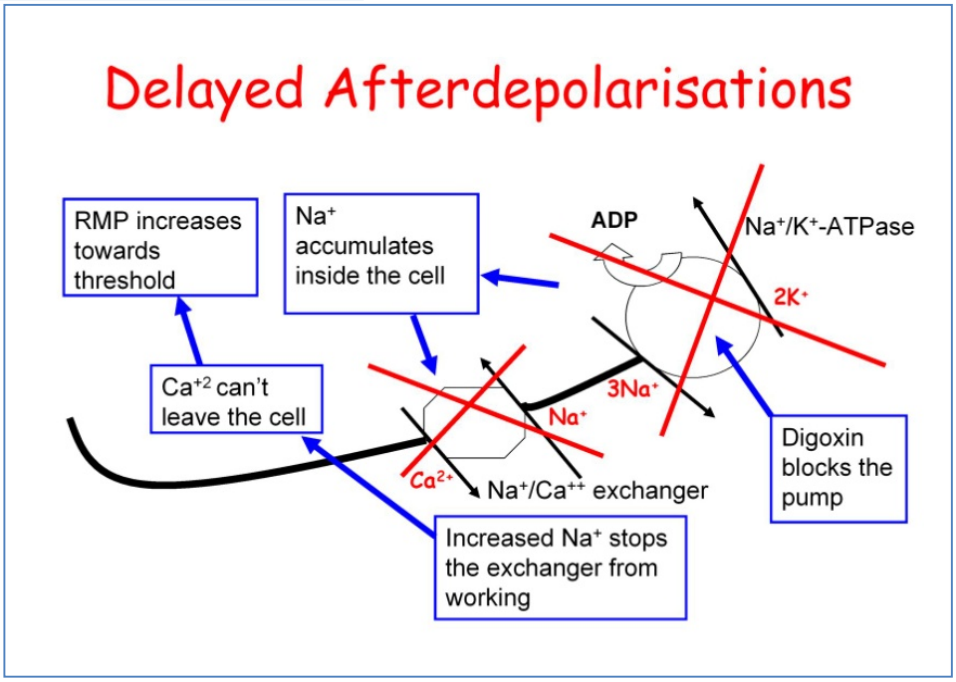
- Depolarisation during phase 4 (after repolarization is completed, but before another action potential would normally occur)
- Due to High Intracellular Ca^{2+} Concentrations Caused by TOO MUCH DIGOXIN.
    - NB: Digoxin is a drug used to treat Atrial Flutter & Atrial Fibrillation by Decreasing Conduction Through the AV-Node. Ie. DIGOXIN → DECREASED HEART RATE

- **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.

- **NB:** 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.







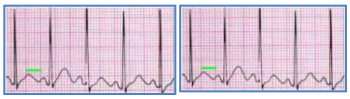
LOOKING AT ECGs:

- **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? (1^{st} 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)
  - NB: Normal ECG Doesn't exclude infarct.
  - ST Depression → Ischaemia
  - ST Elevation → Infarction
  - If LBBB or Paced, you CANNOT comment on ST-Segment

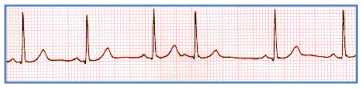
COMMON TACHYCARDIAS:

DDX #1 – *SUPRAVENTRICULAR* TACHYCARDIAS:

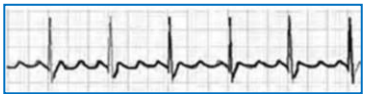
- **SINUS TACHYCARDIA:**
  - **= Sinus Rhythm of 100^{+}Beats/min**
    - Shortened T-P Interval – (But All Waves Visible)
  - **Management:**
    - Carotid Massage
    - (B-Blocker if Symptomatic)



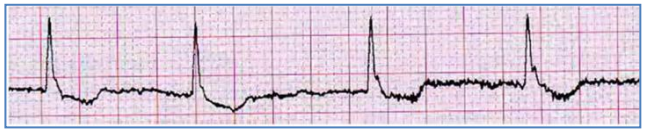
- **ATRIAL PREMATURE BEATS (APBs):**
  - **= Single Ectopic P-Wave → QRS**
  - **Management:**
    - Nil
    - (If Symptomatic → B-Blocker or Ca-Ch-Blocker)



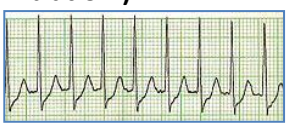
- **ATRIAL FLUTTER:**
  - **= Atrial Rate of ≈300bpm; But NOT Sinus *Rhythm*!**
    - 'Sawtooth' P-Waves
    - Ventricular Conduction Variable – (Eg. 2:1 / 3:1 / 4:1 Block etc)
  - **Mechanism:** Re-Entry (See End)
  - **Treatment:**
    - Rate Control – (B-Blocker, Ca-Ch-Blocker [*Verapamil*], *Digoxin*)
    - Electrical Cardioversion – (NB: Different to Defibrillation)



- **ATRIAL FIBRILLATION (AF):**
  - **= Sinus Rate of ≈350-600Beats/min; Irregular QRSs.**
    - NB: Poor Atrial Contraction → Thromboemboli Common. (Requires Warfarin)
  - **Causes – "PIRATE SHIV":**
    - PE, IHD, Rh-Heart Disease, Anaemia, ↑Thyroid, ETOH, Sepsis, HTN, Iatrogenic, Valvular
  - **Treatment:**
    - **Vent-Rate Control –** (B-Blocker / Ca-Ch-Blocker [Verapamil] / Digozin)
    - **Anticoagulation –** (*Warfarin*)
    - **Cardioversion –** (Medical [Sotalol/Amiodarone] or Electrical)

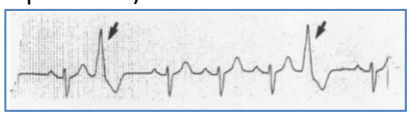


- **PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT):**
  - **= Sudden Onset Regular Tachycardias (Typically Atrial Re-Entry)**
    - Rate ≈ 130^{+}bpm (Regular)
  - **Diagnosis:**
    - **ECG**
    - **Adenosine Trial –** (Dromotropic → Slows SA-Node) :. If Rate slows = SVT.
      - (If not, consider ventricular cause)
  - **Management:**
    - **Rate Control –** (B-Blocker / Ca-Ch-Blocker [Verapamil] / Dogoxin)
    - **(Definitive – Catheter Ablation.)**

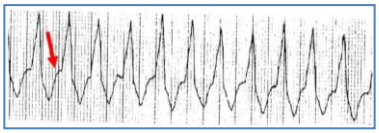


DDX #2 – *VENTRICULAR* TACHYCARDIAS:

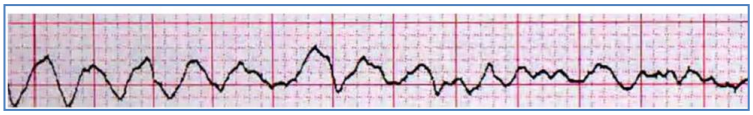
- **PREMATURE VENTRICULAR COMPLEXES (PVCs):**
  - **= Additional QRS's with No Preceding P-Wave.**
    - Wide QRS & Bizarre Shape
  - **Complication –** Consecutive PVCs = *VENTRICULAR TACHYCARDIA*.
  - **Management:**
    - Nil Necessary
    - B-Blocker – (if Symptomatic)



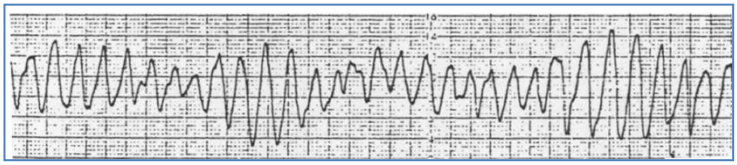
- **VENTRICULAR TACHYCARDIAS:**
  - **= 3 or more Consecutive PVCs**.
    - >30sec = *Sustained* VT
    - <30sec = *Non-Sustained* VT
  - **Mechanism:** Re-Entry (Typically due to IHD)
  - **Treatment – If *Sustained* (>30s):**
    - **Cardioversion**
    - **+/- Anti-Arrhythmic Drugs** (Type 1a Antiarrhythmics [Eg. Procainamide])



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



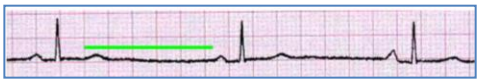
- **TORSADES DE POINTES ("TWISTING OF THE POINTS"):**
  - **= Polymorphic VT with QRS-Complexes of Changing Amplitude**
  - Rate ≈ 200-250bpm
  - **Causes:**
    - Long-QT-Syndrome (An inherited ion channel mutation)
    - (Drugs) eg. K^+ Channel Blockers
    - Electrolyte Disturbances – (Hypokalaemia / Hypomagnasaemia)
  - **Management:**
    - IV Magnesium
    - Temporary Pacing
    - DC Cardioversion - (If Haemodynamic Compromise)



COMMON BRADYCARDIAS:

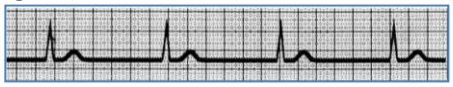
SINUS BRADYCARDIA:

- **= Sinus Rhythm of <60 Beats/min** (SA-Node is still the pacemaker)
  - Prolonged TP-Interval; All Waves Visible
- **Normal – (**At rest/Sleeping/In Elite Athletes)
- **Pathological – (**↓SA-Node Firing (Eg. IHD/Old Age), Cardiomyopathy**).**
- **Management:**
  - ***Atropine*** (If symptomatic) (+/- Pacing)



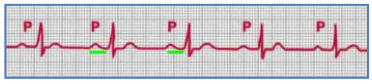
ESCAPE RHYTHMS (SINUS ARREST/EXIT BLOCK):

- **= SA-Node Failure (No P-Wave)** → AV-Nodal *'Escape Rhythm'*.
  - AV-Node *Rate* ≈ 40-60bpm (Compared to the SA-Node's 90-100bpm)
- **Management:**
  - **Cease Dromotropic Drugs** – (*B-Blockers* / *Ca-Ch-Blockers* / *Digoxin*)

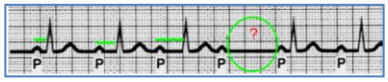


Conduction Blocks:

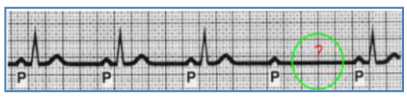
- **= Impaired AV-Conduction**
  - Often due to IHD
  - Often → Escape Rhythm.
- **Types of Conduction Blocks:**
  - **Vertical -** Between Atria & Ventricles
  - **Lateral -** Between L-Heart & R-Heart
- **AV-Conduction Blocks → 1 of 3 Degrees:**
  - **1. FIRST-DEGREE HEART BLOCK:**
    - **= Prolonged AV-Delay (Greater than 0.2sec)**
      - Ie. Prolonged PR-Interval – (But P:QRS ratio = 1:1)
    - **Management:**
      - **Nil.**



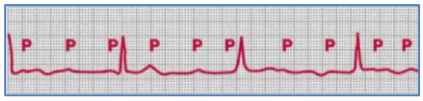
  - **2. SECOND-DEGREE HEART BLOCK:**
    - **MOBITZ TYPE-I (WENCKEBACH):**
      - **= Gradual Lengthening of PR-Interval until a QRS is lost.**
      - **Management:**
        - **Nil; (*Atropine* – if Symptomatic)**

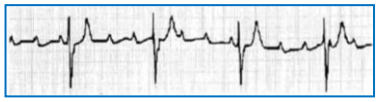


    - **MOBITZ TYPE-II:**
      - **= Intermittent Loss of AV-Conduction with FIXED PR-Interval**
        - Block may last for 2/more beats.
      - **Management:**
        - **Pacemaker**



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



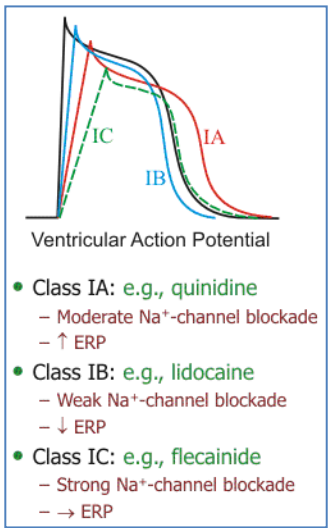


- **Bundle Branch (Lateral) Blocks (Ie. @ L/R Bundle-Branches):**
    - **LEFT BUNDLE-BRANCH BLOCK:**
        - = When Left Bundle-Branch is unable to conduct impulses to L-Ventricle.
            - Therefore, R-Bundle-Branch depolarizes R-Ventricle First, then the impulse travels to L-Ventricle causing it to depolarize.
            - Ie. Ventricles depolarize *Consecutively* rather than *Simultaneously*.
        - → Widened & Split QRS-Complex
    - **RIGHT BUNDLE-BRANCH BLOCK:**
        - = 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*.
        - → Widened & Split QRS-Complex

DRUGS FOR ARRHYTHMIAS:

Therapeutic Management of Dysrhythmias (Arrhythmias) – (Anti-Arrhythmics):

- **Class-I Antiarrhythmics (VG-Na^+ Channel Blockers):**
  - **General Info:**
    - **Indication:**
      - Ie. Typically Re-Entrant Tachycardias (But Not 1st line)
    - **Mechanism of Action:**
      - **Selective VG-Na^+ Channel Blockade (in Contractile Cells):**
        - Slows down Re-Entrant Foci → Restores SA-Nodal Control of HR.
    - **Typical Agents:**
      - **1a** – Quinidine, Procainamide (Intermediate Association/Dissociation)
      - **1b** – Lidocaine, Tocainide (Fast Association/Dissociation)
      - **1c** – Flecainide, Encainide (Slow Association/Dissociation)

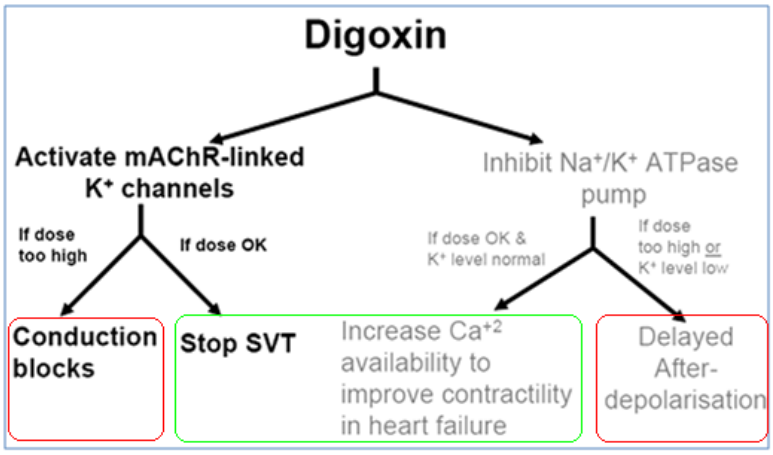


- **Class-II Antiarrhythmics (β1-Blockers):**
  - **Classical Agents:**
    - **Propanolol
    - Atenolol
  - **Mechanism of Action:**
    - β1-Adrenergic Receptor Blockade → Inhibit Sympathetic NS →
      - Conductile System → **↓HR**
      - Contractile Cells → **↓Contractility**
  - **Indications:**
    - 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 Effects:**
    - Sinus Bradycardia.
    - Bronchoconstriction in Asthmatic Patients.
    - (Rebound Tachycardia – if stopped abruptly; Must be weaned off)

- **Class-III Antiarrhythmics (VG-K^+ Channel Blockers):**
  - **Classical Agents:**
    - **Amiodarone
  - **Mechanism of Action:**
    - VG-K^+ Channel Blockers → Prolongs Plateau Phase of AP → ↓HR
  - **Indications:**
    - *1st Line in *Re-Entrant Tachycardias*. – V-Tac, V-Fib, A-Fib & A-Flutter.
  - ***KEY* Side Effect/s:**
    - Bradycardia
    - Early-After-Depolarisation (PVCs/Ectopic Beats)

- **Class-IV Antiarrhythmics (VG-Ca^+ Channel Blockers):**
  - **Classical Agents:**
    - **Verapamil – (Selective for the Heart)
    - Nifedipine – (Selective for Vessels) – (Used in Angina & Heart Failure)
  - **Mechanism of Action:**
    - ****Heart:**
      - **VG-Ca^+ Channel Blockade**
        - **@ SA/AV Nodes → ↓HR**
        - **@ Myocytes** → ↓Ca Influx →**↓ Contractility**)
    - **(Vessels – Used in Angina):**
      - **VG-Ca^+ Channel Blockade**
        - **@ Vascular Smooth Muscle** → Vasodilation → ↓BP & ↓Afterload
  - **Indications:**
    - SVT (Supraventricular Tachycardias)
    - Variant Angina
  - **Contraindications:**
    - β-Blockers– (Since Ca^+ Channel Blockers also Inhibit Ca^+ Influx) → Fatal Bradycardia.
  - ***KEY* Side Effect/s:**
    - Heart Block
    - Bradycardia.
    - (Also Hypotension/Dizziness due to ↓Contractility)

- **Other Agents:**
  - **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:**
      - NB: Not to be given if HR less than 60bpm → Brady/Heart Block.
      - NB: Also, Dosage is very important for reducing side effects.
      - *(NB: Also require K^+ Monitoring - & Supplements if on K^+ Wasting Diuretic)



  - **Adenosine:**
    - **Clinical Use:**
      - Diagnostically to distinguish V-Tac from SVT.
      - NB: Extremely short $T_{1/2}$ - 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

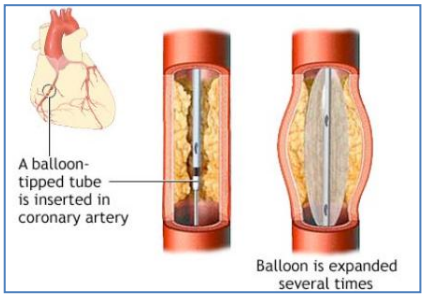
CVS Pathology:
Atherosclerosis

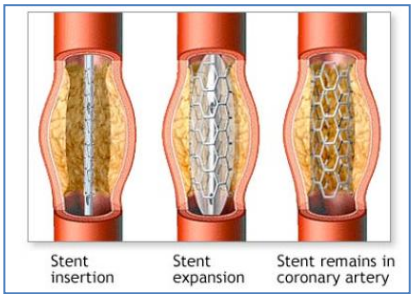
TERMINOLOGY:

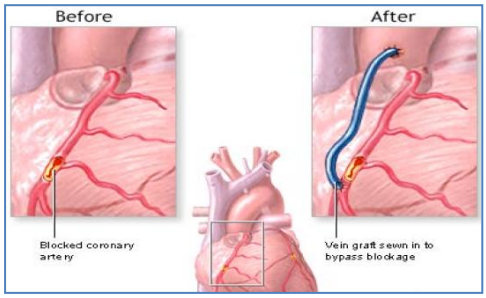
- **"Arteriosclerosis" – Hardening of Any Artery:**
  - ****Macroangiopathy = "Atherosclerosis"** - Hardening of *Large & Medium* Arteries
  - **Microangiopathy = "Arteriolosclerosis"** – Hardening of *Small Arteries*
    - Hyperplastic
    - Hyaline
- **"Arteritis" – Inflammation of Any Artery. (Next Week)**

****ATHEROSCLEROSIS:**

- ***= A Progressive Chronic Inflammation* of *Arteries* characterised by:**
  - ***1. Inflammation*,** (Macrophages engulf LDLs → "Foam Cells")
  - ***2. Fibrosis*,** (Conn. Tissue Matrix/Collagen/Elastin)
  - ***3. & Lipid Deposition*** (Cholesterol Esters & Cholesterol in Cells)
  - **("Athero"= Fat, "Sclerosis"= Hardening)**
- **Aetiology:**
  - BEGINS with Endothelial Injury
  - BIG Inflammatory Component
  - **Risk Factors:**
    - **Non Modifiable:** Age (40-60), Male, FamHx, Indigenous
    - **Modifiable:** ↑Cholesterol, HTN, Smoking, Diabetes, Obesity, Metabolic Syndrome
- **Pathogenesis**
  - **1. Endothelial Injury & Activation – (**HTN/Smoking/DM/Turbulence/Toxins/Infection/Immune).
  - **2. Endothelial Inflammation – (**Macrophage & Smooth Muscle Migration)
  - **3. Accumulation of Lipoproteins → Fatty Streak Formation.**
  - **4. Proliferation & Fibrosis – (Conversion of *Fatty Streak* into a Mature *Atheroma*)**
  - **5. Complicated plaque formation – (Thin Fibrous Cap** → Rupture → Thrombus → ACS)
- **Clinical Features/Complications:**
  - ***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:**
  - **Coronary Angiogram**
- **Management:**
  - **Risk Factor Modification:**
    - **Statins** - (↑Cholesterol)
    - **ACE-I/B-Blocker** - (HTN)
    - **Control DM**
    - **Diet & ↑Physical Exercise** (For Obesity)
    - ↓Smoking, Alcohol
  - **Prevent Thrombosis:**
    - Aspirin/Clopidogrel
  - **Surgical Intervention:**
    - **Balloon Angioplasty/Stent AngioplastyBypass Surgery:**







CardioVascular Medicine Notes
Atherosclerosis

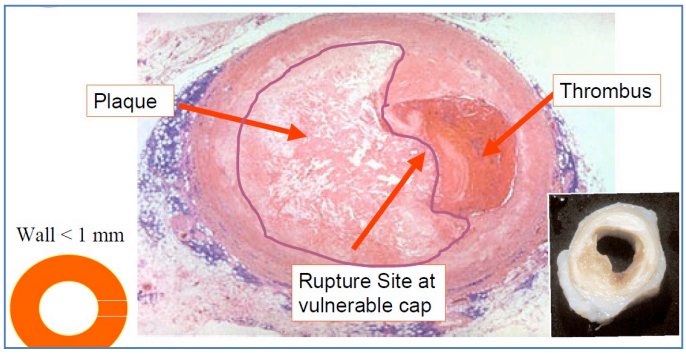
Atherosclerosis: A General Overview:

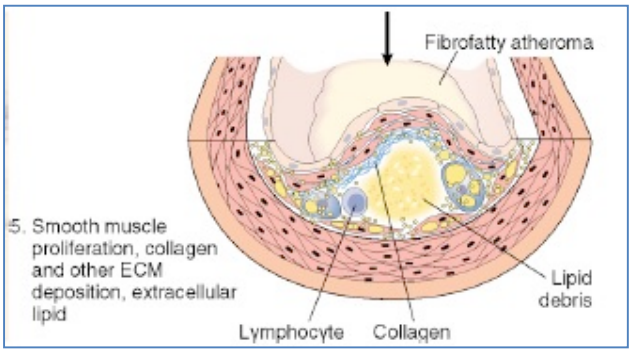
- **"Athero" =** Gruel/Porridge (ie. The fat in the blood)
- **"Sclerosis" =** Hardening

- **A *Progressive Chronic Inflammatory* Disease of the Blood Vessel Wall**
  - Due to Vessel Injury → Fatty ***Plaque Formation*** → Occlusion of Blood Vessel
    - Reduced blood flow to local area → Imbalance of Supply & Demand.

- **Occurs Silently Over Many Decades**

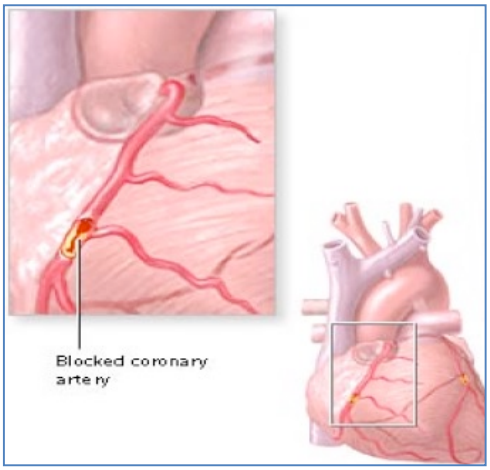
- **Advanced Plaque & Thrombus:**
  - If *'Apical Cap'* becomes unstable → Ruptures → Massive Thrombus (Clot) →
  - Thrombus *Completely* Occludes Vessel





- **Characterised By Accumulation of:**
  - **1. Lipids** (Cholesterol Esters & Cholesterol in Cells)
  - **2. Fibrous Elements** (Conn. Tissue Matrix/Collagen/Elastin) &
  - **3. Local Inflammatory Response** (Macrophages engulf LDLs → "Foam Cells")

- **Principle cause of Heart Disease & Stroke**
  - Cause of 90% Myocardial Ischaemia
    - Due to Occlusion of Coronary Circulation
  - Cause ≈50% of deaths in Western Society.



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

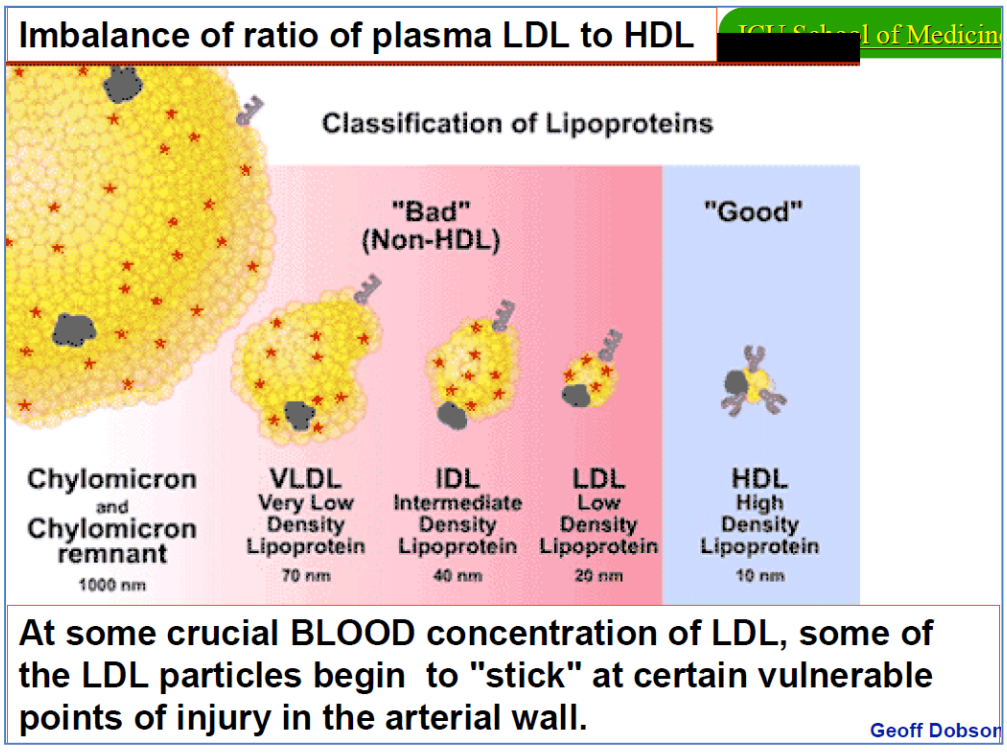
Particle	Source	Predominately transports
Chylomicron	gut	Triacylglycerol
Very-low density Lipoprotein (VLDL)	liver	Triacylglycerol
Intermediate density (IDL)	catabolism	Cholesterol
Low density (LDL)	catabolism	Cholesterol
High density (HDL)	catabolism	N/A
Lipoprotein A	liver,gut	N/A

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**

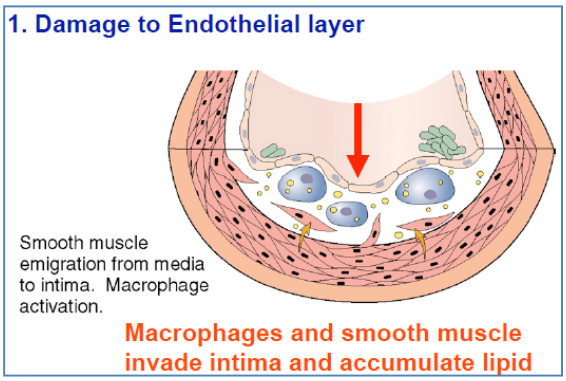
NB: LDLs Attribute to Atherosclerosis

NB: HDLs Help Prevent Atherosclerosis

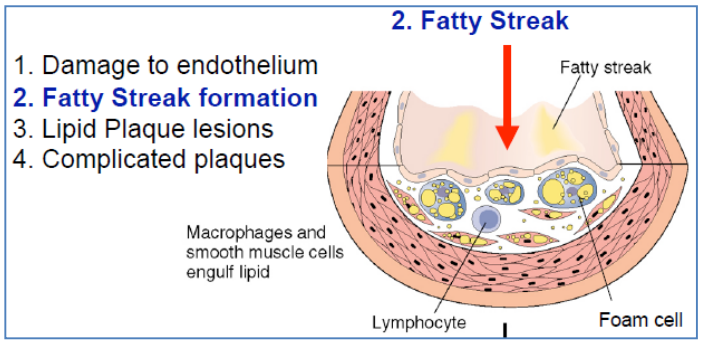


What Is The Process?:

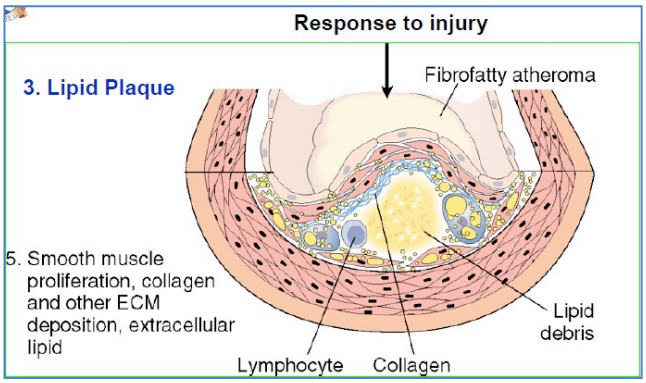
1. **Vessel Injury – Endothelial Damage:**
    a. **Risk Factors:**
        i. High Cholesterol
        ii. Hypertension – High Pressure can split arteries (Particularly where they branch)
        iii. Smoking – Toxins from cigarettes.
        iv. Toxins/Poisons
        v. Virus
        vi. Bacteria
        vii. Immune Reactions
        viii. Diabetes
    b. **Endothelium Becomes Activated:**
        i. Increased Vessel Permeability – Become 'Leaky'
        ii. Platelets Adhere
        iii. Monocytes Adhere → Transform to Macrophages
        iv. *Blood LDLs Enters → Bind to their Apolipoprotein 'Receptors' → Activated & Oxidised* →
        v. *Oxidised LDL Presence → Causes Inflammation:*
    c. **Local Inflammation:**
        i. Oxidised LDLs – Attract Immune Cells/Cytokines/Platelets/Smooth Muscle/Conn. Tissue
        ii. Macrophages Engulf *Oxidised LDL* → Transform to Lipid-Laiden *Foam Cells*.
        iii. *Plaque Building Slowly Begins.*

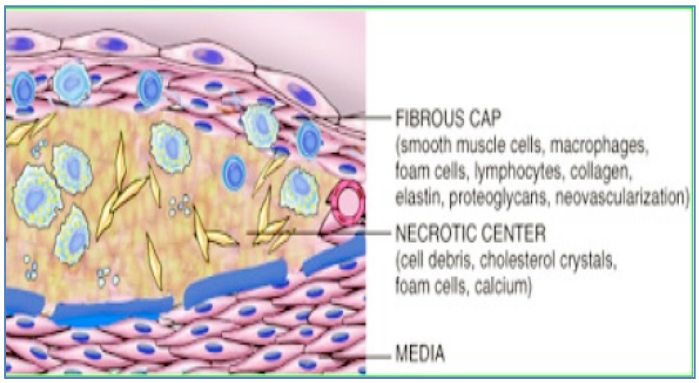


2. **Fatty Streak Formation:**
    a. **Fat Deposition** Under the Tunica-Intima Vessel-layer.
    b. ***The Typical Early Atherosclerotic Lesion*.**
        i. Majority Are *Clinically Silent*
        ii. Are Reversible – eg. If Diet Changes
    c. **Yellow Colour Reflects:**
        i. *Oxidized Lipids*
        ii. Presence of *'Foam Cells'*

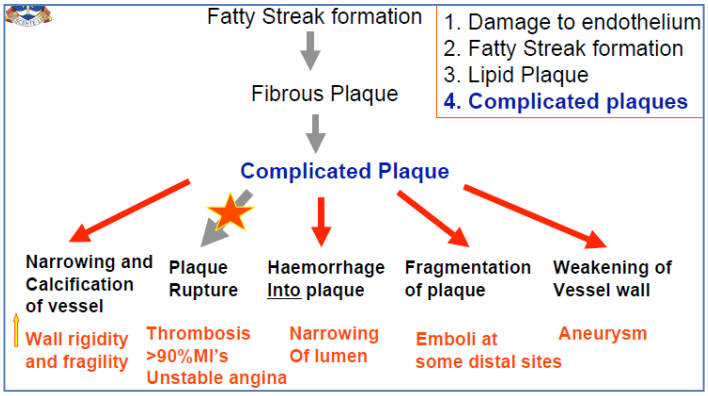


3. **Lipid Plaque:**
   a. Fatty Streak gets more profound
   b. 'Foam Cells' Unable to Digest Lipid Contents → Die
      i. → Extracellular Lipids
      ii. → Cell Debris
   c. Oxidised LDLs – Attract Immune Cells/Cytokines/Platelets/Smooth Muscle/Conn. Tissue
      i. Positive Feedback.
   d. Plaque Builds.

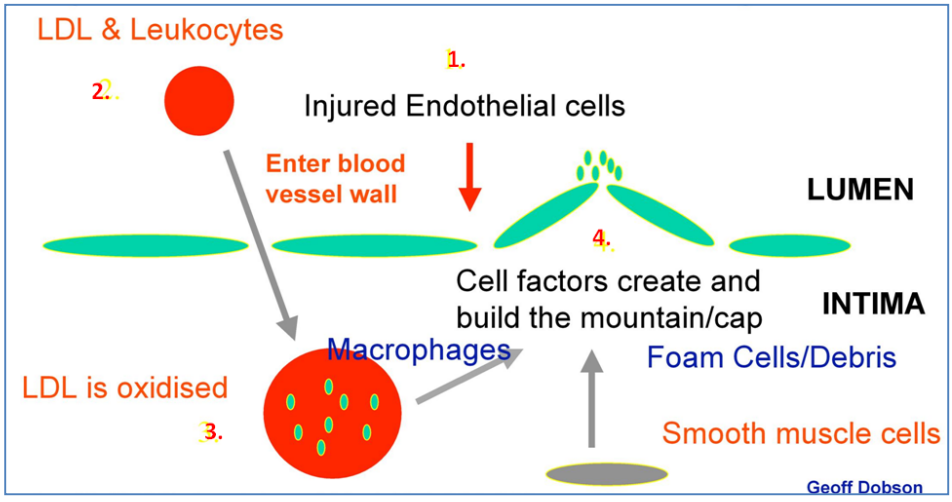




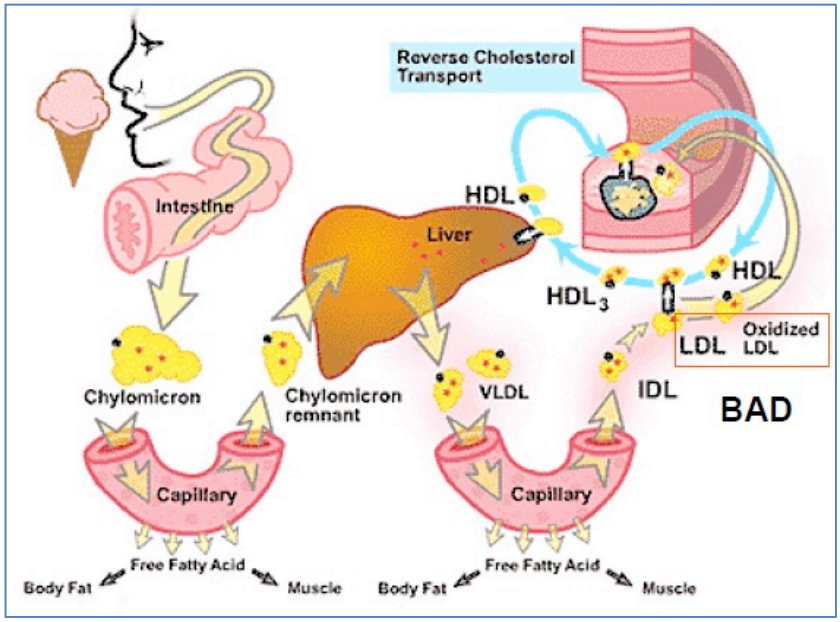
4. **Complicated Plaques:**
   a. **'Cap' forms on plaque** → Becomes more ***'Unstable'*** → May rupture → Thrombus → Occlusion
   b. **Clinical Manifestations (Different Types of 'Complicated Plaques'):**
      i. ****Plaque Rupture** → Thrombosis (Responsible for 90^{+}% of MI's)
      ii. **Narrowing/Calcification** → Vessel Rigidity & Fragility
      iii. **Haemorrhage *Into* Plaque** → Narrowing of Lumen
      iv. **Fragmentation of Plaque** → Distal Emboli
      v. **Weakening of Vessel Wall** → Aneurysms



Simplified Summary Of Process Of Atherosclerosis:



Whole Process

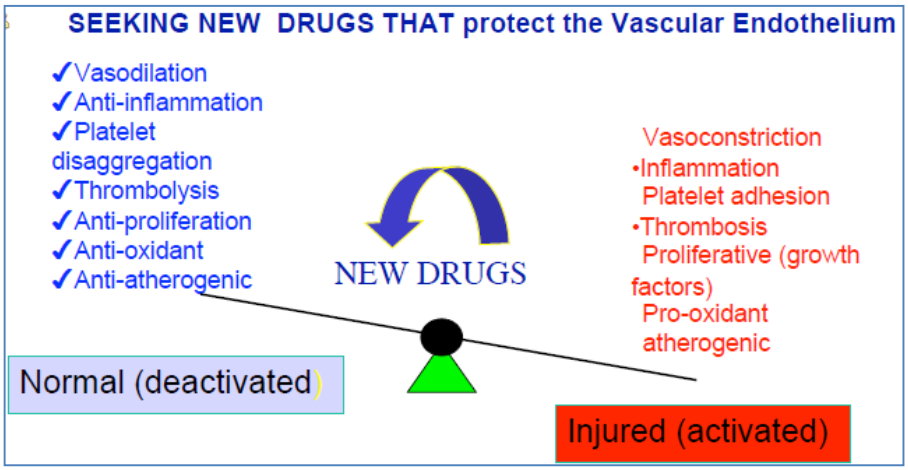


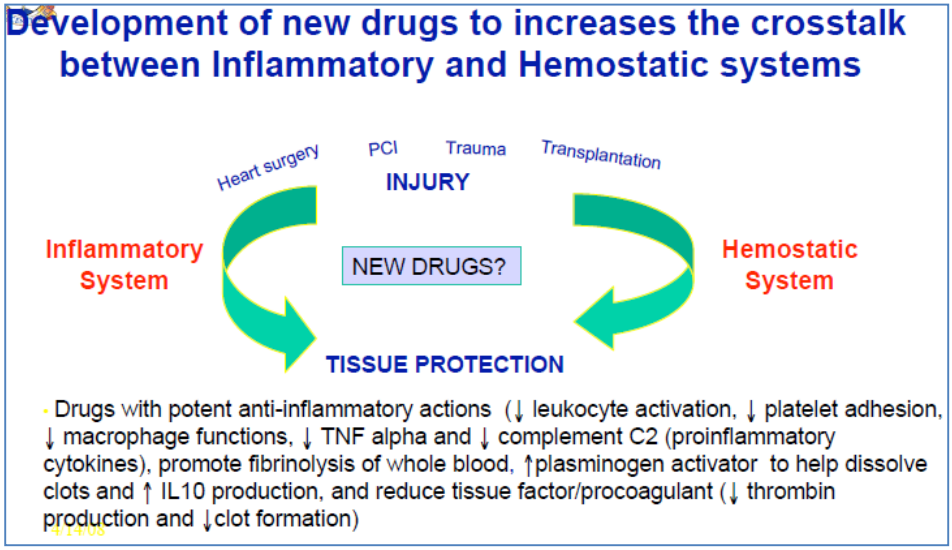
Diagnosis Of Atherosclerosis:

- **(Old) Invasive Method:**
  - Catheter via *Femoral Artery* → *Coronary Artery* → X-Ray Angiogram.
- **(Current) Non-Invasive Method:**
  - Contrast-Enhanced CT-Scan
  - Takes 15sec.

Preventing Atherosclerosis:

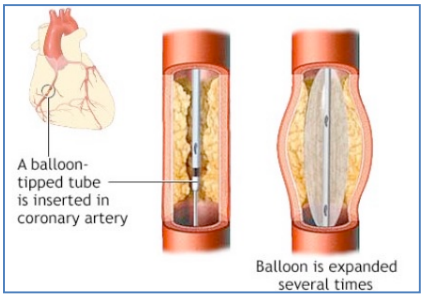
- **Drugs:**



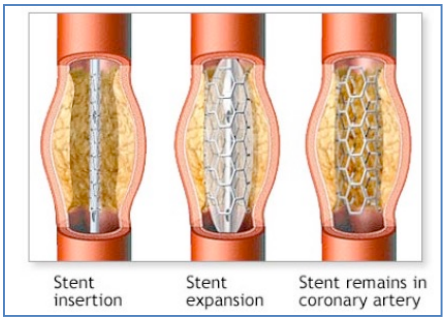


Reparative Surgery:

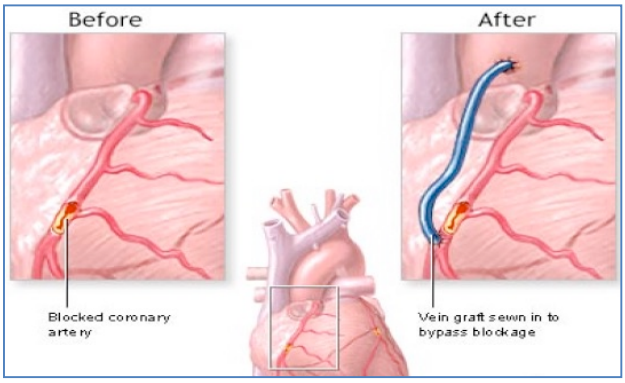
- **Balloon Angioplasty:**
  - Balloon-tipped tube inserted in coronary artery
  - Balloon Expanded multiple times – Stretches lumen.

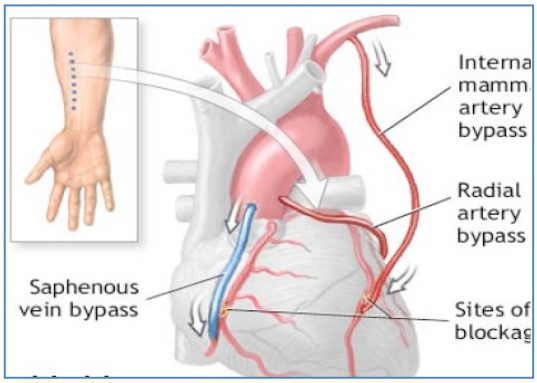


- **Balloon Stent Angioplasty:**
  - Stent-Tipped Balloon-Tipped Tube inserted in coronary artery
  - Balloon (+ Stent) expanded
  - Balloon deflated & withdrawn
  - Stent stays in & open.



- **Bypass Surgery:**
  - The use of a distal vessel as a substitute for Blocked Artery.
  - Eg. Radial Artery Bypass
  - Eg. Saphenous Vein Bypass





SS Questions:

1. **What is the difference between Arteriosclerosis & Atherosclerosis?**
    a. **Arteriosclerosis** is a general term describing any hardening (and loss of elasticity) of medium or large arteries, whereas...
    b. **Atherosclerosis** is the hardening of an artery specifically due to an *atheromatous (atherosclerotic) plaque.*

2. **What are the 3 Types of Arteriosclerosis?**

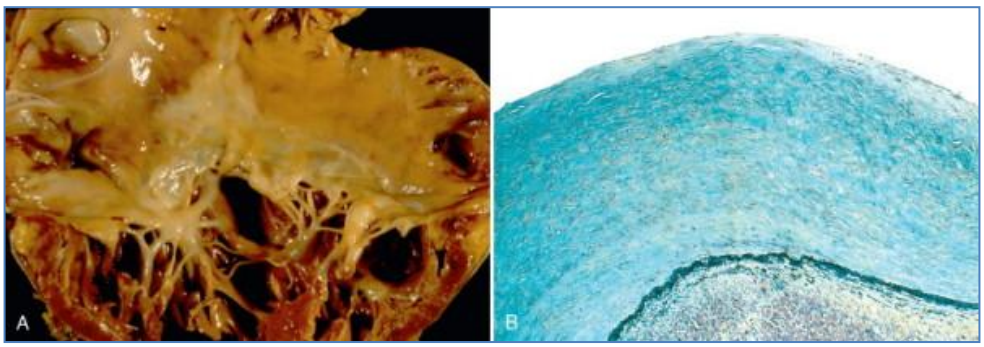
1. Atherosclerosis - Atheroma Plaque	2. Arteriosclerosis Obliterans - Fibrosis of intima _ Calcification of Media.	3. Medial Calcific Sclerosis - mostly in elderly - Calcification of *Internal Elastic Lamina*, but without thickening of intima or narrowing of vessel.

3. **What is the effect of Diabetes on the Vascular System?**
    a. Chronic elevation of blood glucose level leads to damage of blood vessels (angiopathy). The endothelial cells lining the blood vessels take in more glucose than normal, since they don't depend on insulin. They then form more surface glycoproteins than normal, and cause the basement membrane to grow thicker and weaker.

CVS Pathology:
Carcinoid Heart Disease

CARCINOID HEART DISEASE:

- **Aeitology:**
  - Cardiac Manifestation of the Systemic Syndrome caused by Carcinoid Tumours.
- **Pathogenesis:**
  - **Carcinoid Tumour Releases Vasoactive Hormones into Venous Circulation**
    - Serotonin / Bradykinin / Histamine / Prostaglandins
  - **Venous Drainage of these Hormones → R-Heart → R-Heart Endocardial & Valvular Fibrosis.**
    - (Generally → Fibrosis of R-Heart Valves (Tricuspid/Pulmonary))
- **Clinical Signs:**
  - **"Carcinoid Syndrome":**
    - Episodic flushing of skin
    - Cramps
    - Nausea/Vom/Diarr.
  - **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:**
  - **24hr Urinary 5-HIAA** (A Serotonin Metabolite)
  - **Abdo CT + Somatostatin Receptor Scintigraphy (SRS)** – (Tumour Localisation)
  - **Abdo MRI + Contrast**
  - **Cardiac Ix – (ECG, CXR, ECHO)**
- **Management:**
  - **Medical – Somatostatin Analogues (*Octreotide*)**
    - *+/- Interferon-A* in Palliative Pts.
  - **Surgery – Tumour Resection + Valuloplasty**
- **Prognosis:**
  - **Can Metastasize :. Early Removal is Essential**

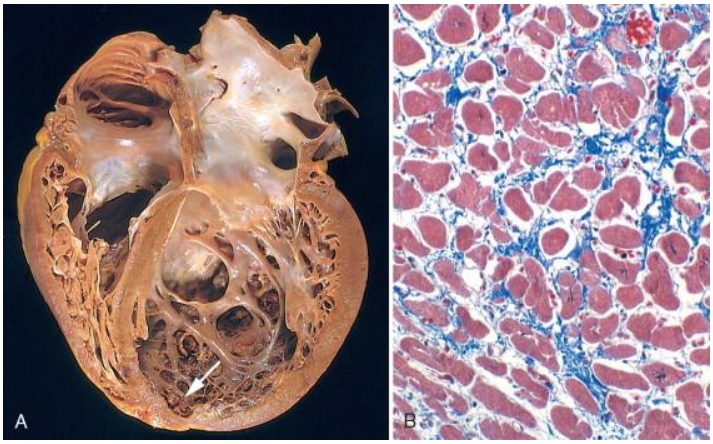


(**A**, Characteristic endocardial fibrotic lesion involving the right ventricle and tricuspid valve. **B**, Microscopic appearance of carcinoid heart disease with intimal thickening.)

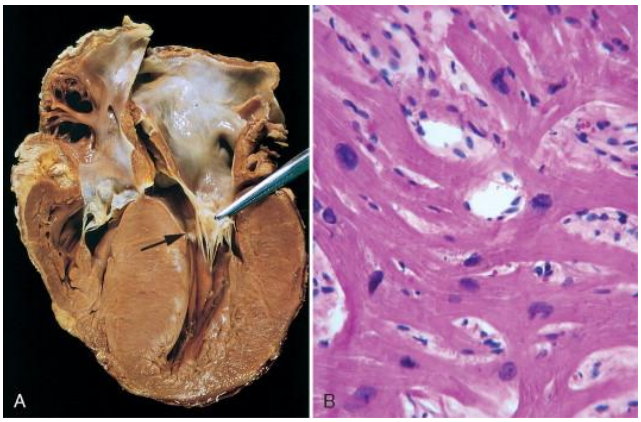
CVS Pathology:
CARDIOMYOPATHIES

CARDIOMYOPATHIES ("Heart Muscle Diseases"):

- **1. 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**.

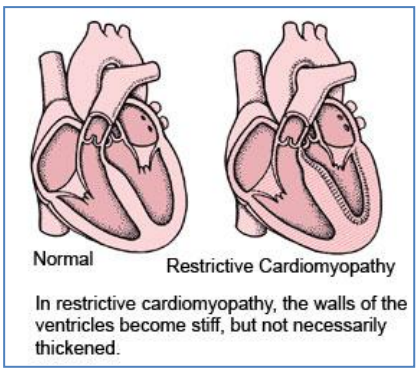


- **2. HYPERTROPHIC CARDIOMYOPATHY (AKA: HOCM – Hypertrophic Obstructive Cardiomyopathy)**
  - **Aetiology:**
    - **Genetic
  - **Pathogenesis:**
    - Genetic Mutation → Hypertrophy → Diastolic Dysfunction (↓Filling & ↓Chamber Size)
    - NB: End Stage can → Focal Ischaemia (Even in absence of Coronary Artery Disease)
  - **Clinical Features & Complications:**
    - CCF (Dyspnoea, Orthopnoea, PND, Cough)
    - Ventricular Outflow Obstruction → Syncope + Harsh Systolic Murmur
    - Angina
    - Arrhythmias
    - Mural Thrombus → Embolisation (eg. Stroke)
    - Sudden Death
  - **Investigations:**
    - **ECG** (LVH, Path Q Waves)
    - **Echo** (LVH, Diastolic Dysfunction, Poor EF)
  - **Management:**
    - **Medical** – **β-Blockers** → ↓Heart Rate + ↓Contractility
    - **Surgical** – Septal Myomectomy (Relieves the outflow tract obstruction)
    - **+/- ICD** – (If Arrhythmias)

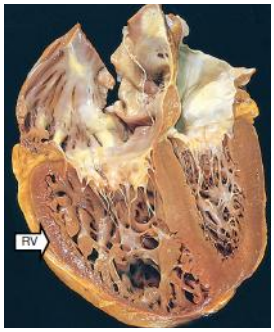


A, The septal muscle bulges into the left ventricule, left atrium is enlarged.
B, Extreme hypertrophy, branching of Myocytes, and the characteristic interstitial fibrosis (collagen is blue).

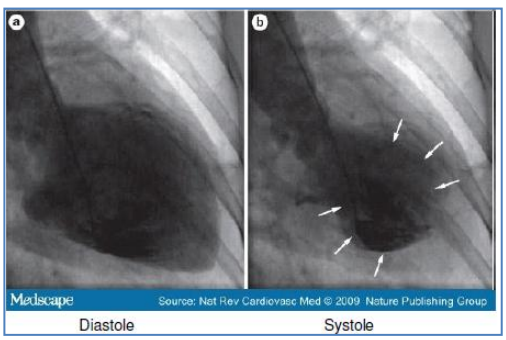
- **3. RESTRICTIVE CARDIOMYOPATHY**
  - **Aetiology:**
    - **Amyloidosis/Sarcoidosis/Scleroderma/Haemochromatosis
  - **Pathogenesis:**
    - → Stiffening of Myocardium → Diastolic Dysfunction (↓Filling) → Heart Failure
      - Ventricles ≈ Normal Size & Volume
      - Myocardium is Firm & Non-Compliant
  - **Clinical Features & Complications:**
    - **Heat Failure Symptoms:**
      - Cough, Dyspnoea, PND, Orthopnea
      - Fatigue
      - Chest Pain, Palpitations
    - **Signs:**
      - Elevated JVP
      - Lung Crepitations
      - Peripheral Oedema
      - Arrhythmias
  - **Investigations:**
    - **ECG –** (Low Voltage)
    - **CXR –** (CCF Signs)
    - **Echo –** (Diastolic Failure, Poor EF)
    - **Myocardial Biopsy (To Determine Aetiology)**
  - **Management:**
    - **Medical:**
      - **CCF Triple Therapy** – (ACEi/ARB + B-Blocker + Diuretics)
      - **Warfarin**
      - +/- Anti-Arrhythmics
    - **Definitive:** **Requires Heart Transplant.**



- **(4. "COR PULMONALE"):**
  - **RV Hypertrophy AND Dilation. (Secondary to COPD/Chronic Pulmonary Hypertension)**



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



CARDIOVASCULAR DISEASE & OBESITY; NUTRITION & PHYSICAL EXERCISE

CARDIOVASCULAR DISEASE:

What is CVD?

- Heart Disease
- Stroke
- Blood-Vessel Disease
- **NB: The *Leading Cause of Death*.**
- **NB: Signs & Symptoms are often *Silent* until Acute Event.**

Epidemiology:

- **CVD *Mortality* Has Declined Significantly:**
  - Better Drugs
  - Better Surgery
  - Falling Smoking Rates
- **However, CVD *Rates* are Increasing due to:**
  - Ageing Population
  - ↑Overweight/Obesity (Doubled since '80s)
  - ↑Diabetes (Doubled since '90s)
  - ↑Sedentary Behaviour
  - NB: Hypercholesterolaemia *Hasn't Changed*.
- **ATSI Populations Suffer Most:**
  - 3x Rate of Major Coronary Events (incl. Heart Attack)
  - 3x as likely to Die from CVD
  - 15-25% *More Likely* to die from ***Acute Rheumatic Fever*** & ***Chronic Rheumatic Heart Disease***.
  - **Why? –** Tend to have multiple CV Risk Factors & more of them.

THE 3 MAJOR RISK FACTORS FOR CVD:

- **Smoking**
- **Hypertension**
- **Hypercholesterolaemia**

***Other* Risk Factors for CVD:**

- ***Unavoidable* Factors:**
  - Sex
  - Age
  - Family history
  - Personality type
- ***Avoidable/Changeable* Factors:**
  - *Nutrition & Inactivity /Sedentary Lifestyle*
    - *Obesity*
    - *Hypertriglyceridaemia*
    - *Hypertension*
  - *Diabetes*
  - *Psychosocial factors* (depression, social isolation, poor social support)
  - ***NB: All of the above can be improved by 2 things*: - NUTRITION & PHYSICAL EXERCISE**.

Lifestyle Measures to Reduce Risk Factors for Chronic Disease:

- ***Control Blood Pressure:**
  - Lose Weight
  - Regular Physical Activity
  - Nutrition
- ***Maintain a healthy Weight:**
  - Regular Physical Activity
  - Nutrition
- ***Physical Activity:**
  - ↑Activity; ↓Sedentary Behaviour
- ***Nutrition:**
  - Adequate Fruit/Veg
  - 2x Fish/Week – (Omegas - Essential Fats)
  - Limit Alcohol
  - Limit Saturated Fats
  - Calcium (At least 800mg/day) → Helps reduced BP in Hypertension.

Role of the National Heart Foundation in CVD:

- **Aim:** To reduce Suffering & Death from Heart/Stroke/Vessel Disease in Australia.
- **How?:**
  - **Fund Scientific Research**
  - **Public Education Resources**
    - Public Awareness Programs
    - Assist in Making Healthier Choices
    - **Educate Community about *Recognising Warning Signs of Heart Attack*:**
      - Jaw Pain
      - Neck Pain
      - Back Pain
      - Shoulder Pain
    - **Especially Women** – Leading cause of death in Women (4x Breast Cancer)
  - **Guidelines for Health Professionals:**
    - Lipid Management
    - Acute Coronary Syndrome Management
    - Heart Failure Management
    - Hypertension Management
    - Guidelines for Physical Activity (For healthy people & with CVD)
    - Guidelines on Diagnosis/Management of Acute Rheumatic Fever & Rheumatic Heart Disease.
    - Guidelines for Reducing risk of Coronary Heart Disease.
    - 'Cardiovascular Risk Calculator'
  - **National Programs:**
    - "Tick Program" – (The Heart-Foundation Approved "*Tick*" on foods.)
    - "Time for Action"
  - **Advocates of Anti-Tobacco Legislation.**
  - **Advocates of Physical Activity for CVD & Obesity Prevention:**
    - Walking Groups – Encourage more people to walk.
  - **Advocate & Actively Contribute to Improving ATSI Health:**
    - Projects to Improve Smoking Cessation.
    - Promoting Physical Activity
    - Promoting Good Nutrition
    - Guidelines on Diagnosis/Management of Acute Rheumatic Fever & Rheumatic Heart Disease.

- **Cardiac Rehabilitation**
  - **Assist CVD Patients by promoting Treatment & Rehabilitation:**
    - "Heartmoves" – Safe exercise program for Stable CVD Pts/High-Risk Pts/Chronic Disease.
    - Walking Groups – Encourage more people to walk.
    - **"My Heart My Life" – Cardiac Rehabilitation:**
      - (An education booklet resource)
      - **Phase 1 – Counselling & Education of Cardiac Patients:**
        - Basic Understanding of Heart Disease
        - Angina Recognition & Home-Management.
        - Medication Education → ↑Compliance
        - Recognition & Modification of Risk Factors
      - **Phase 2 – Improving Condition & Education of Patient:**
        - Regain Pre-Hospital Activities.
        - Action Plans to Modify Risk Factors.
        - Psychological Recovery
        - Regular Physical Exercise.
      - **Physical Activity Guidelines:**
        - Walk 5-10mins Twice Daily (Post- Surgery/Cardiac-Event)
        - Avoid Upper Body Activity (Sternotomy)
        - Sleep on back (not side) for 4-6weeks post-op.
        - **Post-Discharge walking program:**

Week	Minimum time (Minutes)	Times per day	Pace
1	5-10	2	stroll
2	10-15	2	Comfortable
3	15-20	2	Comfortable
4	20-25	1-2	Comfortable/ stride out
5	25-30	1-2	Comfortable/ stride out
6	30+	1-2	Comfortable/ stride out

        - **NB: Shouldn't do *Too Much* Physical Activity:**
          - Walk & Talk test – (Tests breathlessness)
          - If Previous Day's Exercise has left you *Tired or Sore*, have a day off.
        - **Stairs?:**
          - Increase Gradually *Within Your Own Limits*.
          - (If you can walk normally, you should be fine with 2 Flights of Stairs)
        - **Sport?:**
          - Most sports are fine *After 6 Weeks*.
          - However, must check with Cardiologist.
        - **Sex?:**
          - Same as 2 flights of stairs.
          - (If you can walk normally, you should be fine with sex)

OVERWEIGHT & OBESITY:

Obesity is due to:

- ↑Processed (High GI) Foods & Drinks
- ↑Fatty foods
- ↑Sedentary Behaviour
- ↑Effort-saving inventions
- Possibly genetically determined (and Epigenetics)
- **Epigenetics** – events during childhood can change the *expression* of certain genes which predispose patient to Obesity (Changes are *Permanent* & *Heritable*)
- **Leptin Deficiency & Obesity:**
  - Leptin Deficiency causes Hyperphagia → Obesity
  - However, there have only ever been 12 cases worldwide

The General Effect of Fat on the Body:

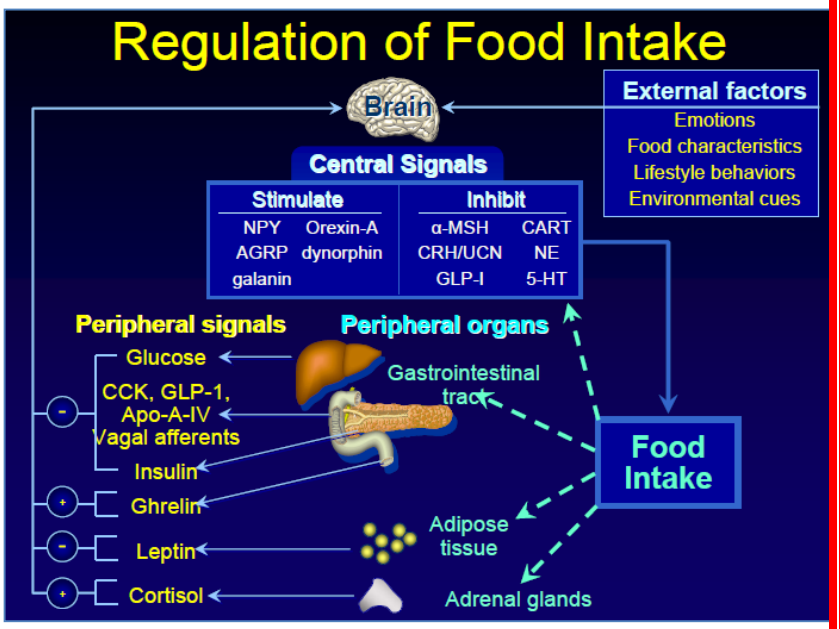
- ↑Fat Mass → ↑Blood Vessels →↑Peripheral Vascular Resistance → ↑Strain on the Heart →↑CVD
- ↑Fat Mass → ↑Body Weight → ↑Wear & Tear on Joints (Particular weight-bearing)→ Arthritis
- ↑Fat Mass → Endocrine Imbalances → Glucose Tolerance .... → Diabetes.
- Many More – Ie. The Whole Body has to work harder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

What is a Healthy Weight?

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

Regulation of Appetite:

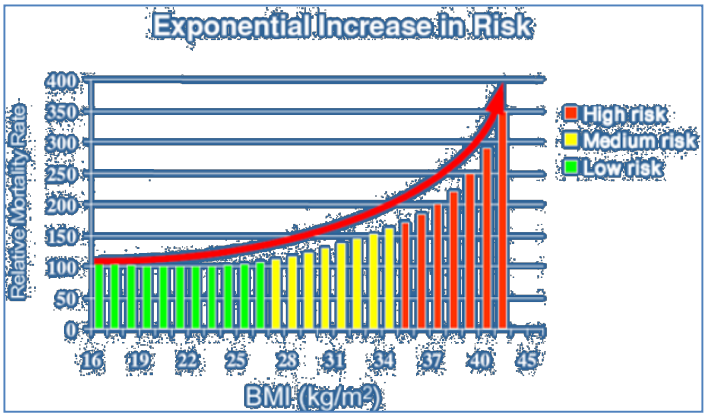
- **Central Signals:**
  - **Appetite Stimulating**
    - Neuropeptide Y
    - Agouti Related Peptide
  - **Appetite Inhibiting**
    - A-MSH
    - 5HT
    - NE
- **Peripheral Signals:**
  - **Positive Feedback:**
    - Ghrelin
    - Cortisol
  - **Negative feedback:**
    - Leptin
    - Insulin



- **NB. The CNS Appetite-Centre has evolved around the idea that "*there is not enough*".**
  - **Ie. There are several Safeguards *Against* Weight *Loss*.**
  - **Ie. There is NO Safeguard *Against* Weight *Gain*.**

↑Mortality increases exponentially with ↑BMI:

- Almost all body organs are affected



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
  - **NB: 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)
    - **NB: 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 (Movt is an opportunity rather than inconvenience)
  - Increase aerobic capacity
  - Resistance training
  - NB: Aerobic fitness almost halves risk of cardiovascular disease mortality.
  - NB: 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

BRUCE:

- 55yrs
- 84kg → 110kg due to sedentary job, too much food & alcohol. (BMI 35)
- Waist 110cm
- Tried many times to diet/exercise etc, but largely ineffective.
- Motivation – excercise intolerance, uncomfortable, avoid medication, avoid premature death, poor body image.
- Goal – to be under 90kg.
- Med Hx:
    - Impaired glucose tolerance (Elevated fasting BSL)
    - Unfavourable lipid profile
    - Recently hypertensive 155/90
    - Mild osteoarthritis of knees.
- Fam Hx:
    - Bowel Cancer
    - Myocardial Infarction
    - Ischaemic Heart Disease
- Social Hx:
    - Poor sleep
    - Late to bed, & tired in morning
- Treatment (Physical Exercise):
    - Bike riding
    - Walked to and from work
    - Pedometer
    - Supervised resistance training with Exercise Physiologist
    - Decreased screen time (Sedentary time)

LIFESTYLE FACTORS & CHRONIC DISEASE

NB: Lifestyle Measures in Treatment of Chronic Disease is INDIVIDUAL:

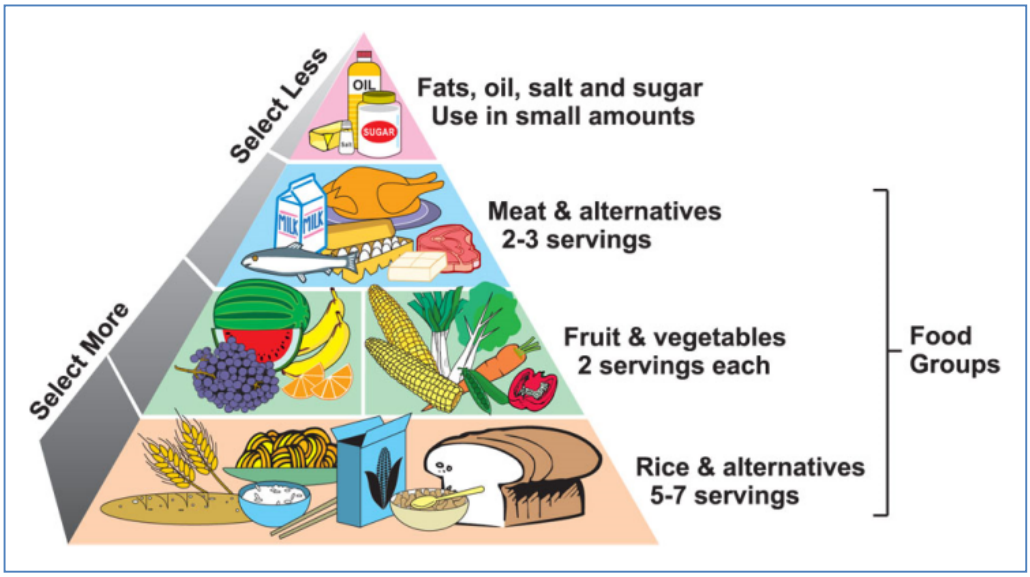
- **Ie. Not one size fits all –** (Ie. The same 'lifestyle prescription' doesn't suit everyone.)
- **Dietary Advice should be Adapted Depending on the Patient's Situation:**
  - Eg. Obese with CVD
  - Eg. Normal weight with CVD
  - Eg. Diabetes and obese
  - Eg. Diabetes and normal weight

Lifestyle Measures to Reduce Risk Factors for Chronic Disease:

- ***Control Blood Pressure:**
  - Lose Weight
  - Regular Physical Activity
  - Nutrition
- ***Maintain a healthy Weight:**
  - Regular Physical Activity
  - Nutrition
- ***Physical Activity:**
  - ↑Activity; ↓Sedentary Behaviour
- ***Nutrition:**
  - 2x Fruits/Day
  - 5x Vegs/Day
  - 2x Fish/Week – (Omegas - Essential Fats)
  - Legumes
  - Limit Alcohol
  - Limit Alcohol
  - Limit Saturated Fats
  - Calcium (At least 800mg/day) → Helps reduced BP in Hypertension.

- **NB: Notice how all of the above tie in with each other?**
- **Therefore, the 2 MOST IMPORTANT Lifestyle Measures that cover all the bases in reducing Chronic Disease are *Nutrition* & *Physical Activity*.**

NUTRITION:



- **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).
      - **NB:** 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)
      - (NB: 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 Fat:** Carbohydrates
      - **If Pt. Is Thin:** Poly- or mono-unsaturated fats
      - (Decision depends on BMI)

- **Role of Dieticians in Managing Patients with Chronic Disease - Giving Nutritional Advice:**
  - ***Tell them what to eat, rather than what not to eat:**
    - 2x Fruits/Day
    - 5x Vegs/Day
    - 2x Fish/Week – (Omegas - Essential Fats)
    - Legumes
    - Limit Alcohol
    - Limit Alcohol
    - Limit Saturated Fats
    - Calcium (At least 800mg/day) → Helps reduced BP in Hypertension
  - **IF you tell them what to avoid:**
    - Many will starve themselves
    - Others will find it too hard & give up

  - **Nutrition knowledge quiz - Hawkes and Nowak 1998**
    - **1. To reduce your cholesterol level do you think you should eat less:**
      - **Cakes/biscuits - yes**
      - **Ice cream - yes - (No if made with Skim Milk)**
      - **Fat on meat - yes**
      - **Peanuts - yes – (*Salted* Peanuts require oil for the salt to stick)**
      - **Coconut - yes**
      - Skim milk - (Has NO fat – so makes no difference)
      - Sugar – (Sugar will make you fat, but doesn't affect cholesterol)
      - Bread – (It's what you put *on* the bread)
      - Avocados – (Contains *Good Cholesterol*)
    - **2. Cholesterol is only found in animal products?**
      - **True** – Plants don't have sat-fats)
      - (**NB: *Saturated Fats*** apparently increases Bad (LDL) Cholesterol Levels)
    - **3. To reduce your blood cholesterol level is it more important to eat less saturated fat or less cholesterol?**
      - **Saturated fat**
      - (NB: Dietary Cholesterol has little/no effect)
    - **4. Which has LESS fat (pick one)**
      - Butter
      - Margarine
      - **They are equal –** (Fat content goes by *Weight*, therefore 1g of Marg = 1g of Butter)
        - (The Only difference is Margarine contains *Poly-Unsaturated Fats*)
    - **5. Which has LESS fat (pick one)**
      - Olive oil
      - Vegetable oil
      - **They are equal**
    - **6. The correct way to lose weight is to eat LESS:**
      - **Cheese - yes**
      - **Butter - yes**
      - **Cakes - yes**
      - **Margarine - yes**
      - Bread – (It's what you put *on* the bread)
      - Bananas – (Depends *how much* you eat)
      - Potatoes - (Depends *how much* you eat)
      - Grapes - (Depends *how much* you eat)
      - Rice yes - (Depends *how much* you eat)
      - **NB: While there are some things to avoid to lose weight *Faster*, it still comes down to *HOW MUCH* you eat – (Energy Balance)**

- **7. Are these foods HIGH in fat?**
  - **Toasted muesli - yes** – (Muesli is *Toasted* in Oil. NB: NON-Toasted is fine)
  - **Nuts - yes** – (50% Oil, 50% Protein; Where do you think they get peanut oil from?)
  - **Margarine - yes –** (Even though it contains poly-unsaturated fats, it still has fat)
  - **Olive oil - yes –** (Oil *IS* Fat in *Liquid Form*)
  - **Carob bar - yes –** (Requires oil to form it into *Bars* & *Resemble the Texture of Choc*)
  - Spaghetti – (No – Just carbs)
  - Rice – (No – Just carbs)
  - Bread – (No – Just carbs)
- **8. The Main Ingredient in a food is listed FIRST on a food label:**
  - **True**
- **9. Does one teaspoon of fat weigh:**
  - 0.1 gram
  - 1 gram
  - **4 grams**
  - 10 grams
  - not sure
- **10. Which of these foods contain fibre?**
  - **Oranges - yes**
  - **Bread - yes** – (NB: White Bread has very little; unless fortified with fibre)
  - **Baked beans – yes**
  - Steak – (NO – Just Protein)
  - apple juice – (NO – unless freshly squeezed ***with pulp***)
  - fish - (NO – Just Protein)

PHYSICAL ACTIVITY:

- **The Role of Physical Activity in Promoting Health & Preventing Chronic Disease:**
  - **NB: *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.

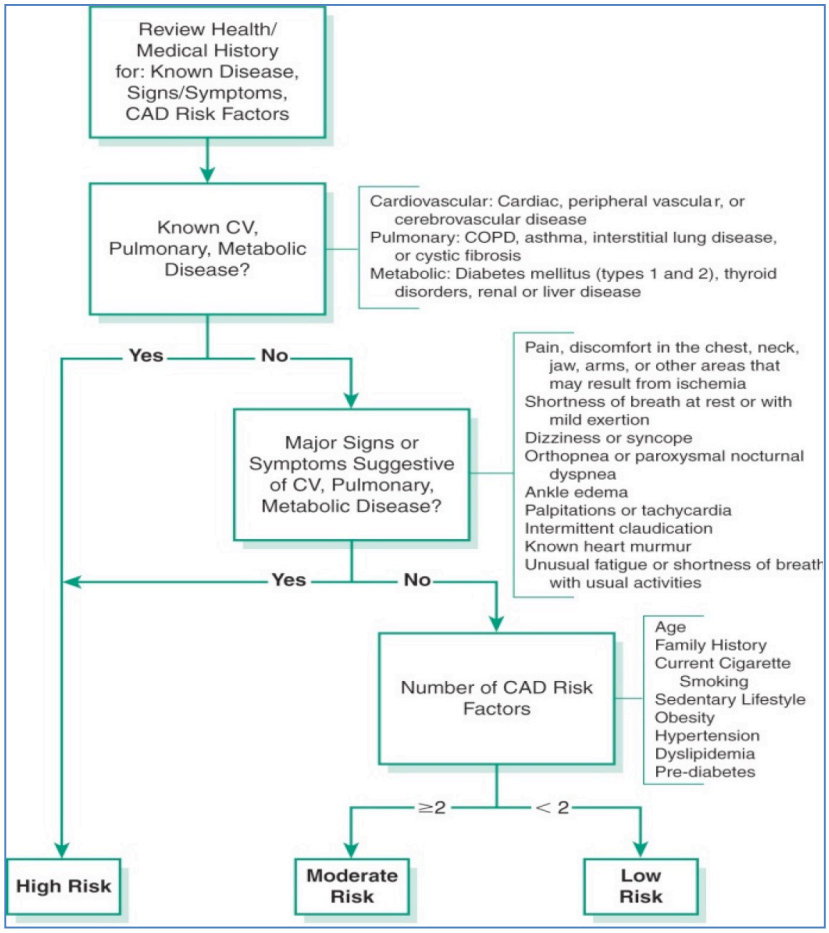
- **The Perils of Sedentary Behaviour:**
  - **Sedentary Behaviour is *DIRECTLY LINKED to*:**
    - ** CVD (NB: All of the below further contribute to CVD)
    - *Obesity
    - *Depression
    - *Diabetes (Typically type 2)
    - Osteoporosis
    - Stroke
    - Hypertension
    - High Cholesterol
  - **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:**
    - (**NB: Moderate Activities =** Brisk walk, a Bike ride or Active Play)
    - (**NB: 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**.
    - **NB: Children & Adolescents require almost *Double*.**
  - **12-18 year olds:**
    - At least **60mins** of Moderate to Vigorous Physical Activity Every Day.
    - **NB: 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.
    - **NB: If Exercise is being used as a disease *Intervention*, the recommendations are *Doubled*.**
  - **Elderly/Completely Sedentary:**
    - *ANY Physical Activity is Beneficial*.

- **Rate of Perceived Exertion – RPE – "Borg's RPE Scale":**
  - **Measures *Perceived* Exertion on a scale of 0-20:**
    - 0 = Falling Asleep; 6 = Very Very Light Exertion; 20 = Maximal Exertion
  - **Why? –** Because HR *alone* is an unreliable measure of exertion in patients on Cardio-Drugs.

- **How do we Ensure the Exercise is Safe for the Patient?:**
  - **NB: Low-Risk Patients can start exercise Immediately.**
    - **However, Moderate-High-Risk Patients should undergo medical testing before undertaking any exercise.**
  - **Risk Stratification Flow-Chart:**



NB: I Doubt you'll have to memorise this. For your interest & understanding only.

- **The Role of Physical Activity in Management of Chronic Disease:**
  - **PA & Management of Cardiovascular Disease (Post Myocardial Infarction):**
    - **Post-MI Exercise is *Immediate*:**
      - Ie. Within days after the MI.
      - **NB:** However, It is only *LOW INTENSITY*.
      - **NB:** 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

- **PA & Management of 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)
      - NB: Exercise + Normal dose of Insulin = Additive Effect; can cause hypoglycaemic shock. Therefore Necessary to ↓Insulin dose with Exercise.
      - NB: 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.

- **PA & Management of Depression:**
  - **Benefits:**
    - Improved Self-esteem
    - Improved Mood
    - Opportunity for participation in community/family events/tasks
  - **Recommendations:**
    - HIGH Intensity, FUN Exercise is recommended → Mental Distraction.
      - NB: The opposite (Anxiety), requires long, slow, exercise → Calming.
    - The longer the duration, the greater the benefit
    - Exercise is *As Effective* as psychotherapy
  - **Precautions:**
    - CVD risk factors
    - Stage of change & motivation
    - Must vary exercises, repetition leads to boredom.

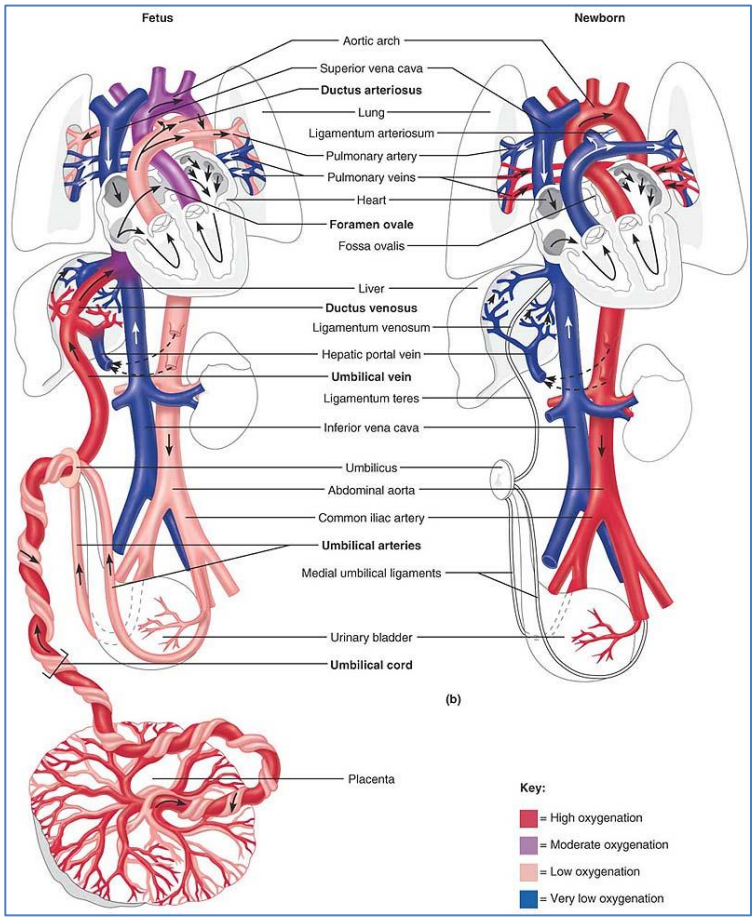
- **PA & Management of Cancers:**
  - **Benefits:**
    - Preservation/Improvement in Muscle Mass & Strength
    - Preservation/Improvement of CV fitness
    - Preservation/Improvement of Immune Function
    - Preservation/Improvement Self-Esteem
    - Decrease in side-effects & symptoms
    - Decreased hospitalisation length, depression & anxiety
  - **Recommendations:**
    - Large muscle groups (Moderate Daily Aerobic Exercise ≈ 20-30 min total)
  - **Precautions:**
    - Swimming (Infection/catheters/wounds)
    - Limited Balance/Coordination (eg. Treadmill, Yoga) when sensory neuropathy, dizzy.
  - **Contraindications:**
    - On days of IV Chemotherapy.
    - Platelets<50,000; WBC<3,000
    - Unusual weakness/malaise, dehydration, nausea

- **Allied Health Professionals & Physical Activity:**
  - **Exercise Scientists (3Yr Degree):**
    - **Mainly Involved in Disease PREVENTION in *Healthy Patients*:**
      - **Kids/teenagers:**
        - • active after school program
        - • sporting groups
      - **• Adults:**
        - • community projects (healthy hearts, 10,000 steps)
        - • workplace health promotion (corporate challenges)
      - **• Elderly:**
        - • group exercise (aqua)
  - **Accredited Exercise Physiologists (4Yr Degree):**
    - **Mainly Involved in Management & Rehabilitation of *Chronic Disease* Patients:**
      - **Musculoskeletal conditions**
        - Athletic/Workplace injury
        - Osteoarthritis/Osteoporosis/Back pain
      - **Neurological conditions:**
        - Multiple Sclerosis, stroke, ABI, SCI, Epilepsy, Cerebral Palsy, Parkinsons Disease
      - **Cardio-respiratory conditions:**
        - CVD, asthma, COPD, emphysema, cystic fibrosis
      - **Metabolic conditions:**
        - Obesity, diabetes
      - **Cancers**
    - **NB: AEPs Have *Medicare Provider Numbers* – Pts are covered.**

CVS Pathology:
Congenital Heart Defects

Review of Foetal Circulation:

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



CONGENITAL HEART DEFECTS:

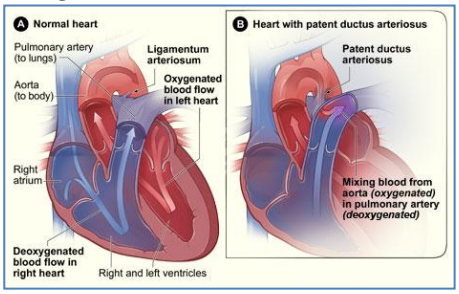
- **NB: 50-80% of Children have "Innocent" Heart Murmurs; Red Flag = Murmur + Cyanosis/↓Perfusion.**
- **L→R (Non-Cyanotic) Shunts – (ASD, VSD, PDA)**
  - VSD = Commonest
- **R→L (Cyanotic) Shunts – (TETRALOGY & TRANSPOSITION)**
- **Obstructive Defects – (COARCTATION, Valvular Stenoses)**

Genetic Associations:

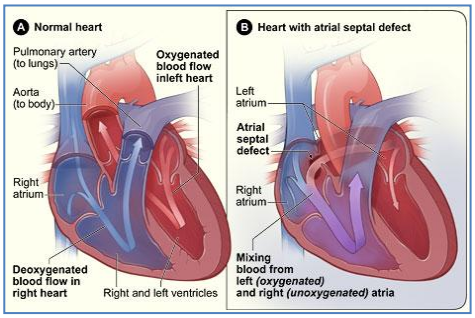
- **MARFAN'S SYNDROME:**
  - **Autosomal Dominant Disorder**
  - **CV-Defects: - (**Mitral Prolapse / Tricuspid Prolapse / ASD / Others**)**
- **DOWN'S SYNDROME:**
  - **Trisomy 21**
  - **CV-Defects: - (**Valvular Malformations / ASD / VSD**)**

Left→Right (NON-Cyanotic) Shunts.

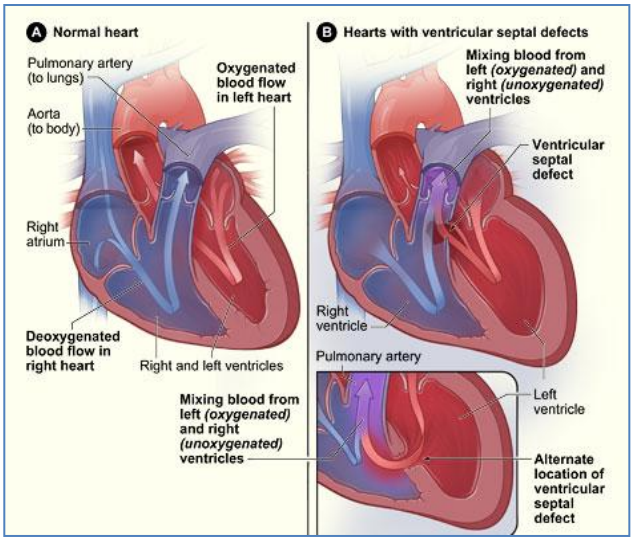
- **PATENT DUCTUS ARTERIOSUS (PDA):**
  - **= Malocclusion of Ductus Arteriosus after birth.**
    - **L→R Shunt from Aorta → Pulmonary Artery**
      - → Pulmonary Hypertension
  - **Clinical Features:**
    - **Murmur** *(Continuous, Harsh* "**Machinery-like**" *Murmur*).
  - **Complications:**
    - Soon After → **Irreversible Obstructive Pulmonary Vascular Disease** (Pulmonary Vessel Hypertrophy & Vasoconstriction →↑Resistance) → SHUNT REVERSAL → Cyanosis
  - **Investigations:**
    - **CXR – (**Pulmonary Congestion, Cardiomegaly**)**
    - **ECG – (**LVH, RVH**)**
    - **ECHO – (Definitive)**
  - **Management:**
    - **(*PDAs should be closed as early in life as possible)**
    - **Medical:** Indomethacin (Prostaglandin Inhibitor)
      - (NB: In Cyanotic Heart Defects, Prostaglandin is actually *Given* to maintain a PDA)
    - **Surgical:** Surgical Ligation



- **ATRIAL SEPTAL DEFECT (ASD):**
  - **= Hole in the Interatrial Septum. (NB: NOT a patent Foramen Ovale)**
    - **→ Shunt from L-Atria → R-Atria:**
      - → RV-Hypertrophy & Pulmonary HTN.
  - **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**
  - **Complications are Rare, but Include:**
    - **CCF** → Pulmonary Oedema (Dyspnoea) + Peripheral Oedema, Ascites, etc.
    - **"Paradoxical Embolisation"** (DVT → Stroke)
  - **Investigations:**
    - **ECG –** (RV-Hypertrophy, RAxDev)
    - **CXR –** (Pulmonary Congestion & Oedema)
    - **ECHO – (Definitive)**
  - **Management:**
    - Surgical Endovascular Closure of Defect

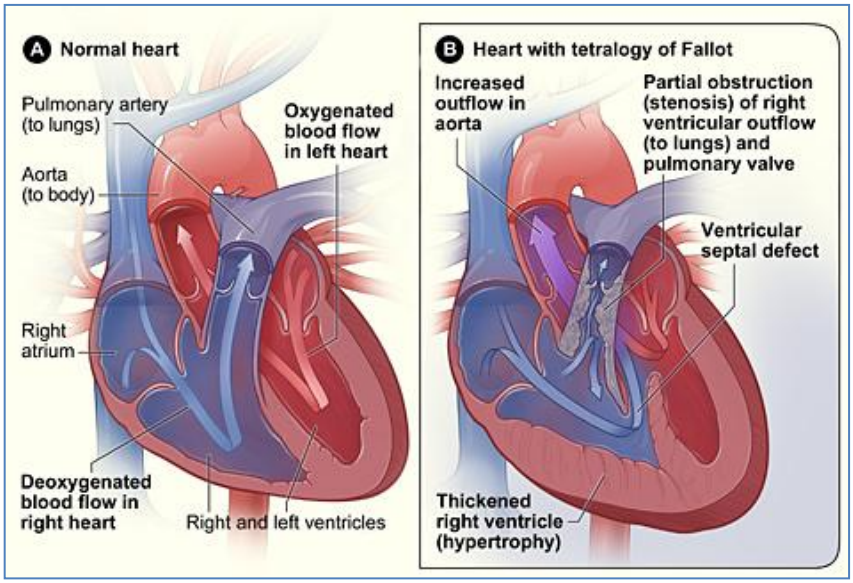


- **VENTRICULAR SEPTAL DEFECT (VSD):**
  - (*The Most Common Congenital Heart Disease)
  - **= Hole in the Interventricular Septum.**
    - **→ Shunt from L-Vent. → R-Vent.**
      - → LV-Failure (CCF, Pulm.HTN, RV-Hypertrophy).
  - **Clinical Features:**
    - **Asymptomatic if Small (& Close Spontaneously)**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

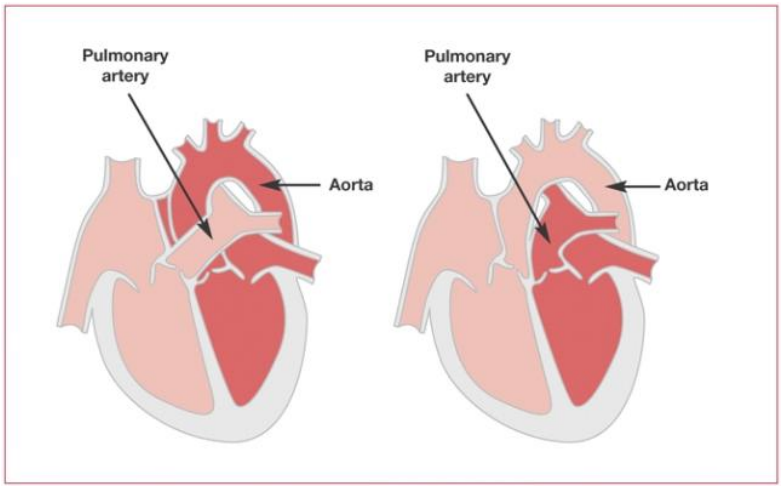


Right→Left (CYANOTIC – SpO2 <75%) Shunts

- **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 Pulm. Stenosis →** Resembles an isolated VSD. (L-R-Shunt) [Non Cyanotic]
    - **If Severe Pulm. Stenosis →** R-L-Shunt →
      - Chronic Cyanosis SpO2 <75%
      - Fingernail Clubbing
      - Polycythaemia (↑RBC)
    - **Symptoms:**
      - Blue Baby
      - Paroxysmal Tachypnoea
      - Irritability/Crying
  - **Complications:**
    - *"Paradoxical Embolism"* – (DVT → Stroke)
    - Seizures
  - **Investigations:**
    - **ECG – (**RV-Hypertrophy**)**
    - **CXR – (**Boot-Shaped Heart**)**
    - **ECHO – (Definitive)**
  - **Management:**
    - **Medical:**
      - **Supp O2**
      - **B-Blocker**
    - **Surgical:**
      - **Definitive Repair**

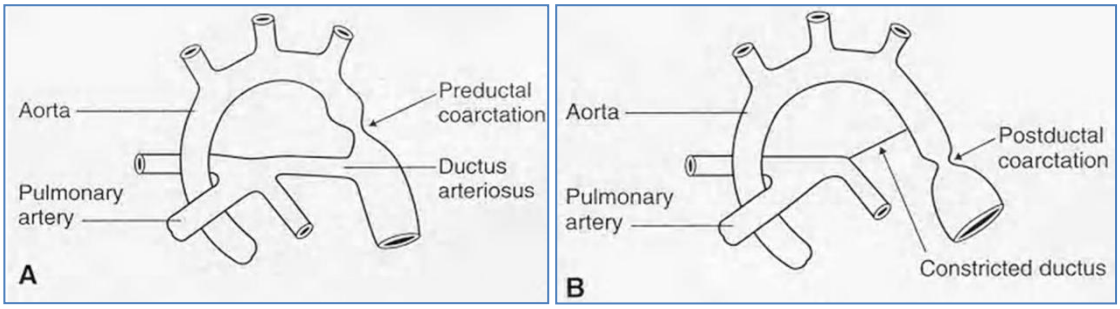


- **TRANSPOSITION OF GREAT VESSELS:**
  - **= Aorta & Pulmonary Artery are switched.**
    - (NB: Atrioventricular Connections are correct)
    - (NB: Venous Return is correct – IVC/SVC & Pulmonary Veins)
    - Hence, the Pulmonary & Systemic Circuits run in *Parallel*, rather than *Series*.
  - **NB: 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:**
    - **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 w. 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:**
    - **90% 1yr Mortality without Surgery.**



Obstructive Defects:

- **COARCTATION OF AORTA**
  - **= Narrowing/Constriction of the Aorta.**
    - 2M:1F
    - *50%* have *Bicuspid Aortic Valve*
  - **Pathophysiology – 2 Types:**
    - **Preductal:**
      - Proximal to Ductus Arteriosus
        - → R-L-Shunt (Pulm.Artery → Aorta).
        - → Cyanosis of *Lower Half* of body.
    - **Postductal:**
      - Distal to Ductus Arteriosus.
        - → L-R-Shunt from Aorta→Pulm.Artery
        - → Pulmonary HTN & CCF
  - **Clinical Features:**
    - **Symptoms:**
      - **Leg Claudication**
      - **NB: 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).



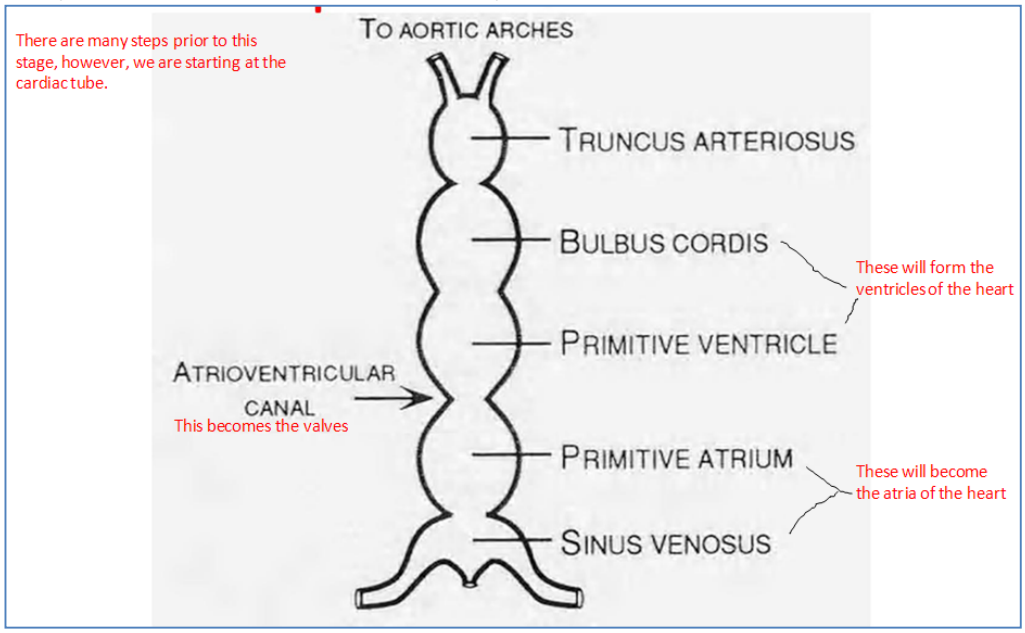
- **AORTIC STENOSIS:**
  - **= Narrowing/Obstruction of the Aortic Valve.**
    - → ↑LV-Afterload → ↓Cardiac Output → LV-Failure (Pul.HTN, Dyspnoea)
- **PULMONIC STENOSIS:**
  - **= Narrowing/Obstruction of the Pulmonary Valve OR Artery.**
    - → ↑RV-Afterload → ↓Pulmonary Output → RV-Failure (Peripheral Oedema)

CardioVascular Medicine Notes
Congenital Heart Defects

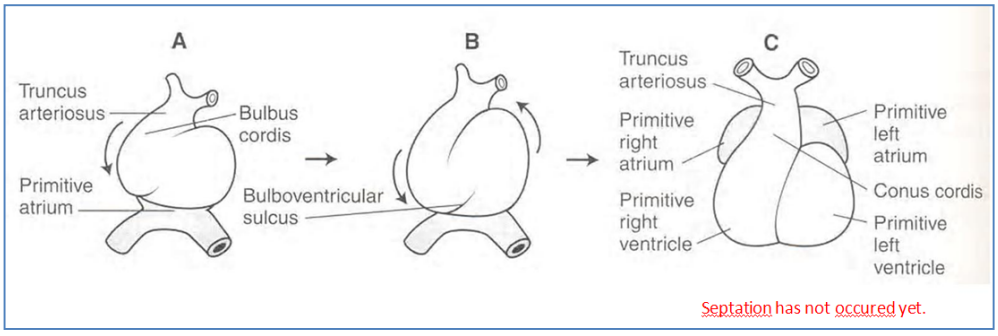
Basics of Foetal Development of the Heart:

- 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

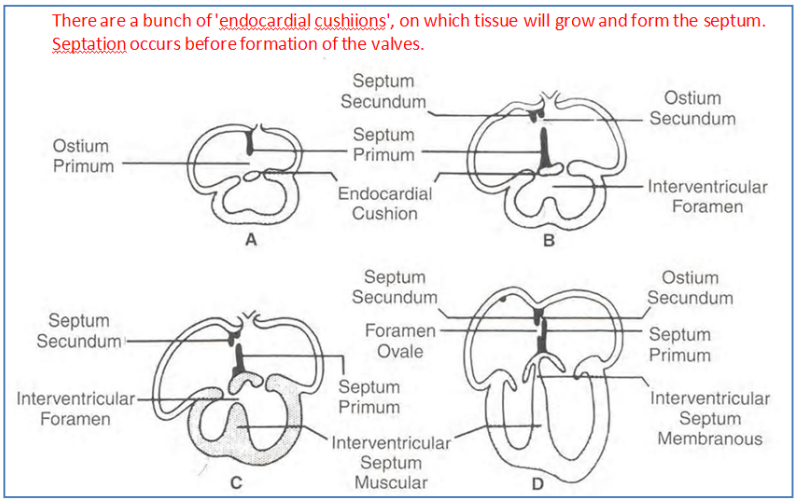
- **The "Cardiac Tube":**
    - The primordial tubular heart in the embryo, before its division into chambers.



    - This cardiac tube begins to fold & twist on itself until it is laid out in the basic heart-like structure.

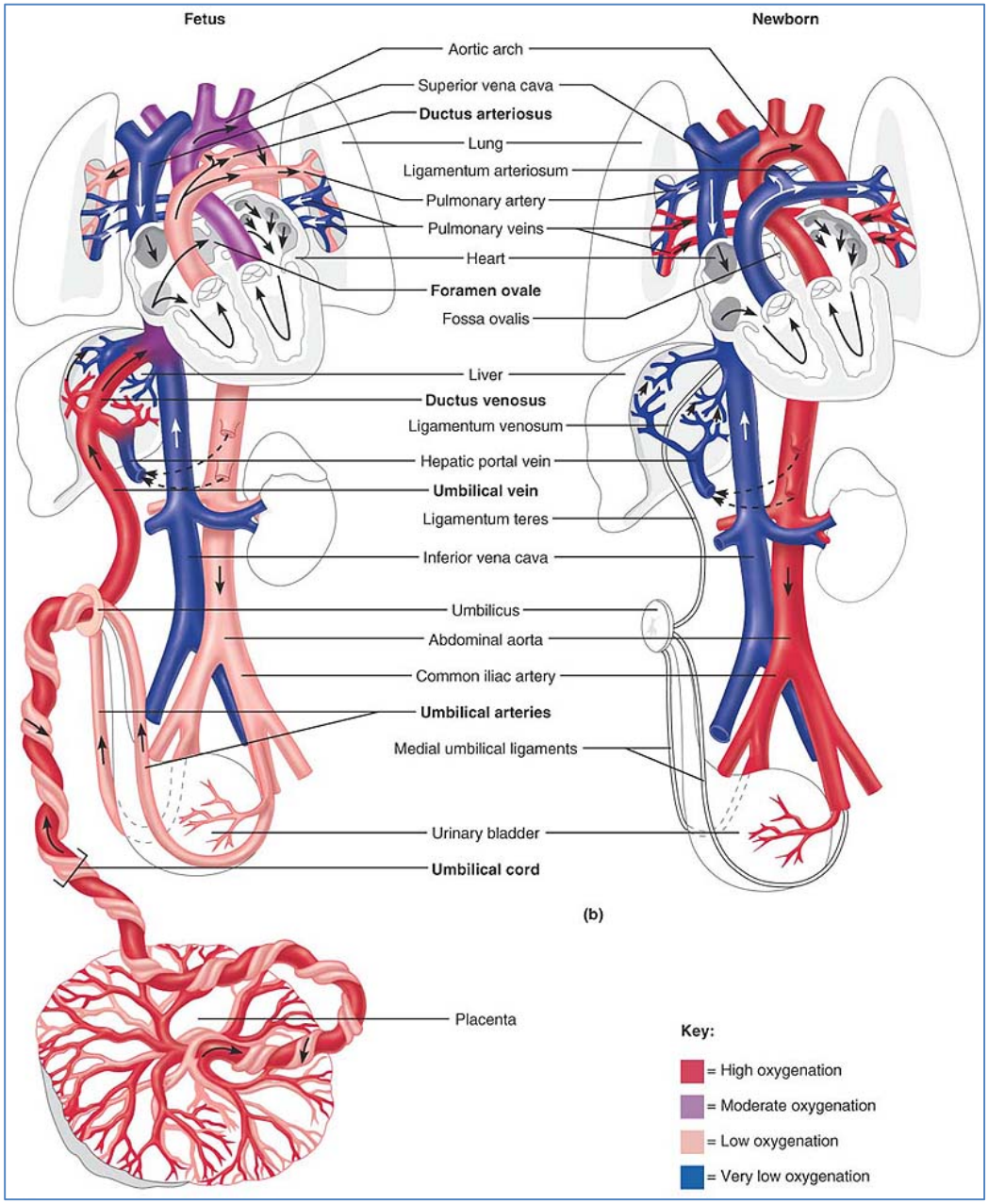


- **Septation:**
    - Between 4-6 weeks



Foetal Circulation:

- Foetal circulation is different from neonatal circulation.
  - Has to integrate circulation of placenta.
  - Blood flow to non-functional lungs & liver are partially bypassed.
- **"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.
    - NB: The Foramen Ovale can take up to 6 months to close.
- **NB: At Birth, the Pulmonary Vascular Resistance Falls Due to:**
  - **1. Mechanical Inflation** → Increased Radial Traction of Vessels
  - **2. Vasodilation** – due to ↑Oxygen-Tension in the Lungs

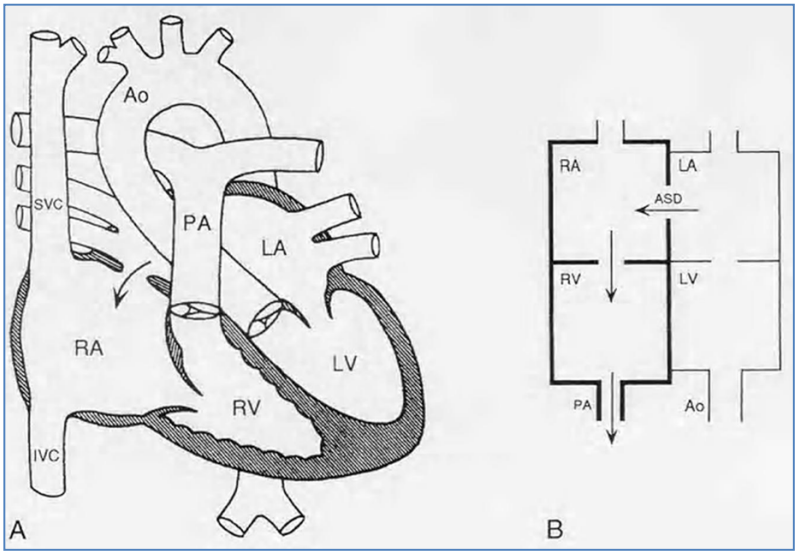


Types of Congenital Heart Defects:

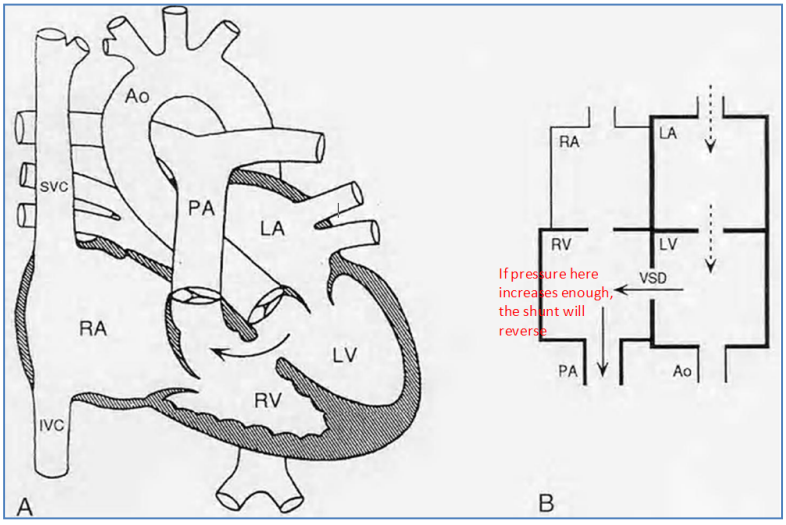
Septal Defects:

- A hole in the septum (Either Atrial/Ventricular) Leading to the Mixing of Blood from one side to the other.
  - This *'mixing'* (shunt) typically occurs from Left to Right (Due to Higher L-Heart Pressure)
  - **However, the shunt may reverse** to Right→Left (If Pulmonary Blood Volume Reaches a Critical Level)
- **Treatment:** Direct suture closure with a pericardial/synthetic patch.

- **Atrial Septal Defects:**
  - Opening in the Inter-Atrial Septum – Usually at the level of the Foramen Ovale.
  - NB: This is different from a *'patent foramen ovale'* (Normal for up to 6-12 months old) in that an 'Atrial Septal Defect' is just a hole, not a flap.
  - **Leads to:** Oxygenated Blood is shunted from the L-Atrium to the R-Atrium.

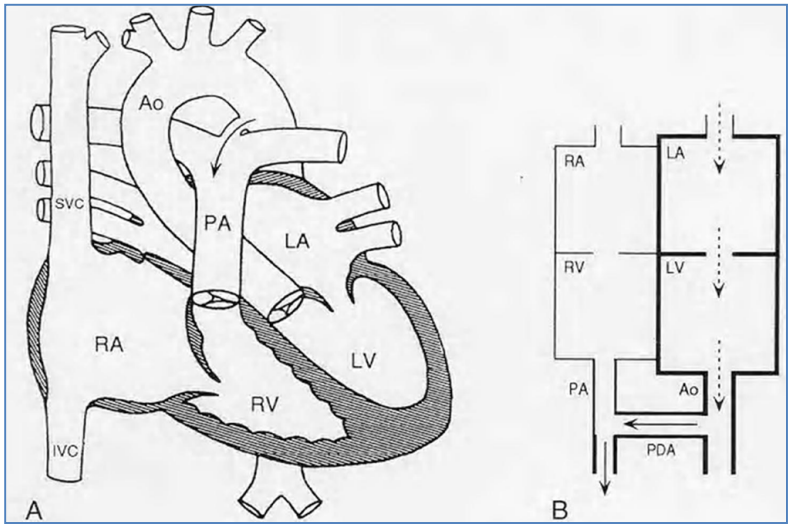


- **Ventricular Septal Defects:**
  - Opening in the Inter-Ventricular Septum – Vary greatly in size.
    - Most smaller shunts close spontaneously by 2 yrs.
    - Larger shunts tend to remain open
  - **Leads to:** Blood shunted from L-Ventricle to R-Ventricle →Increased output to Pulmonary Circulation
    - Can result in Pulmonary Hypertension → R-Heart failure & Hypertrophy
      → The Shunt may Reverse → Causing Deox. Blood to flow into L-Ventricle (Therefore Systemic Circulation) → Systemic Cyanosis. (Eisenmenger Syndrome)



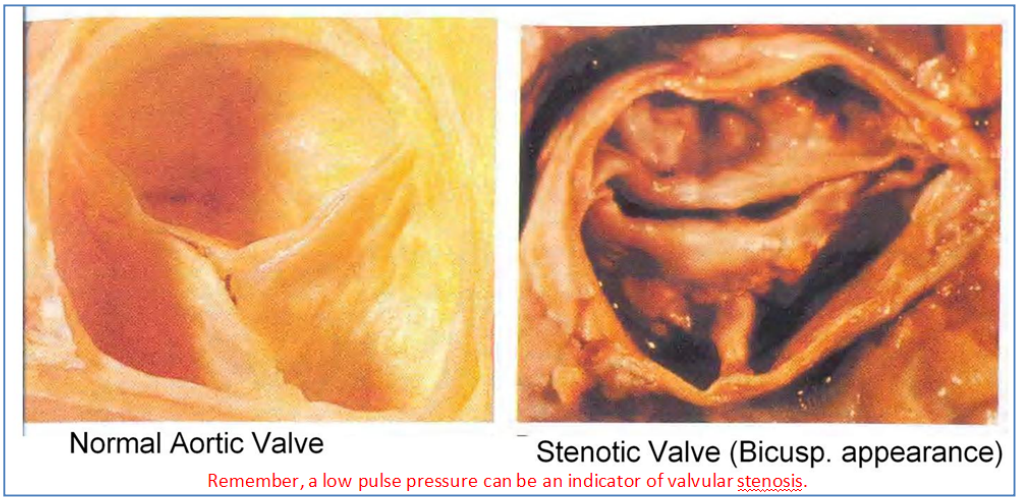
Patent Ductus Arteriosus:

- Where the Foetal Shunt that connects the Pulmonary Artery to the Aorta Doesn't Close off.
  - Normally, Rising O_2 Tension & Decreasing Prostaglandins cause it to Close.
- **Leads to:** Shunting of Oxygenated (Aortic) Blood back into the Pulmonary Artery (Deox) → Lungs.
  - **NB:** In Utero, Blood flows the other way (From the Pulmonary Artery to the Aorta)



Valvular Stenosis:

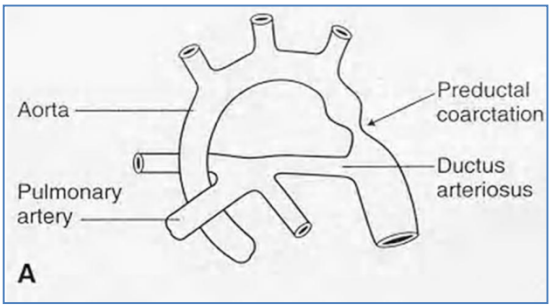
- **Aortic Stenosis:**
  - Narrowing/Obstruction of the Aortic Valve.
  - Typically presents as a Bi-Leaflet, instead of the normal Tri-Leaflet Formation.
  - Most common in males.
  - **Leads to:** Increased Afterload → ↑L-Ven. End Systolic Volume → ↓Cardiac Output → Hypotension
    (↓Cardiac Output → Backup of Blood in Lungs → Pul.Congestion)
    → L-Ventricular Hypertrophy → Possibly Left Heart Failure



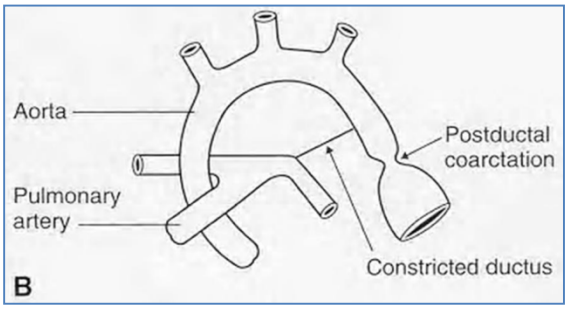
- **Pulmonic Stenosis:**
  - Narrowing/Obstruction of the Pulmonary Valve OR Artery.
    - 90% = Valvular
    - 10% = Elsewhere in the Pulmonary Artery
  - **Leads to:** Increased Afterload → ↑R-Ven. End Systolic Volume → ↓Pulmonary Output
    (↓Pulmonary Output → Backup of Blood in Body → Syst.Oedema)
    → R-Ventricular Hypertrophy → Possibly Right Heart Failure

Coarctation of the Aorta:

- Narrowing of the Aortic Lumen Either Before or After the Ductus Arteriosus.
  - "Preductal" Coarctation of the Aorta
  - "Postductal" Coarctation of the Aorta
- **Leads to:** Increased 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.
- **NB:** Clinical Presentation may take up to 10 years – Due to the fact that the Coarctic bit doesn't grow, while the rest of the heart grows around it. Hence, it only presents itself when the body's demand for increased cardiac output exceeds the flow rate of the Aorta.
- **Treated By:** Surgery or Balloon Angioplasty & Stenting.



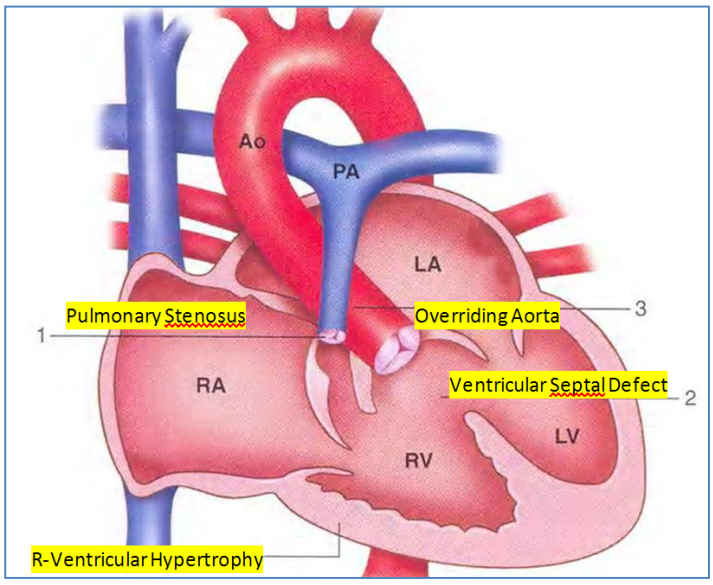
Preductal Coarctation

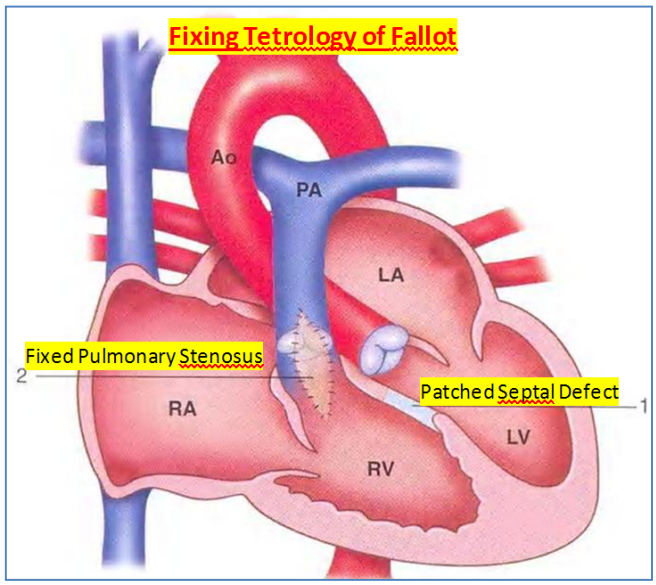


Postductal Coarctation

Tetrology of Fallot:.

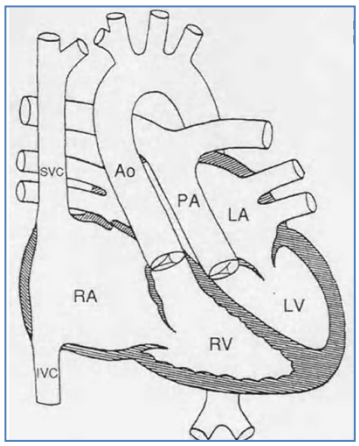
- **Consists of 4 Abnormalities:**
  - 1. Ventricular Septal Defect
  - 2. Subvalvular Pulmonic Stenosis
  - 3. R-Ventricle Hypertrophy (Due to Pulmonic Stenosis)
  - 4. Overriding Aorta (Valve sits over the Vent.Septal Defect – ie. Receives Blood from Both Ventricles)
- **Most Common Cause of CYANOSIS *after* Infancy.**
- **Symptoms:**
  - Cyanosis
  - Irritability
  - Hyperventilation (Due to Cyanosis–low [O_2] stimulates peripheral chemoreceptors → ↑Respirations)
  - Occasional Syncope (Fainting)
  - Occasional Convulsion
- **Symptoms Alleviated by Increasing Systemic Vascular Resistance:**
  - Eg. By squatting (kinking femoral artery)
  - → Means more blood being ejected via the Pulmonary Artery → Better Oxygenation.



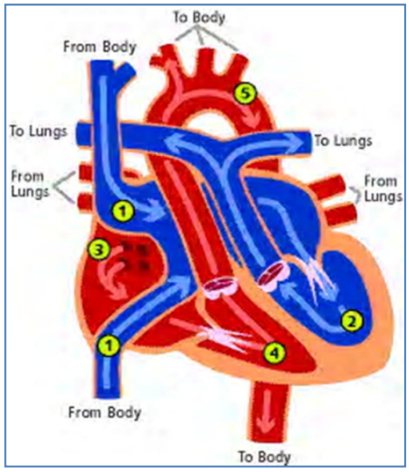


Transposition of the Great Vessels:

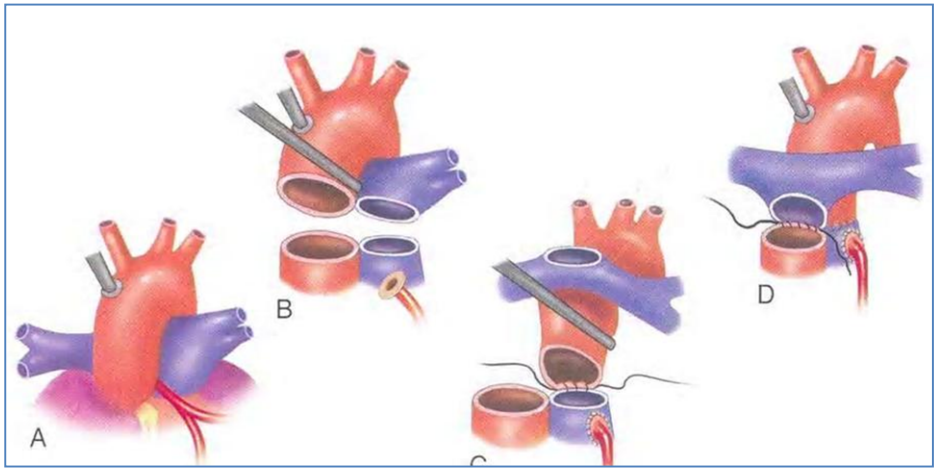
- Where the Aorta comes off the R-Ventricle & the Pulmonary Artery comes off the L-Ventricle.
- Hence, the 2 Circuits (Pulmonary & Systemic) are running in *Parallel*, rather than *Series*.
    - **Pulmonary Circuit:**
        - L-Vent. Blood → Pulmonary Artery → Lungs → Pulmonary Vein → L-Atrium → L-Vent.
    - **Systemic Circuit:**
        - R-Vent. Blood → Aorta → Systemic Circulation → Vena Cavae → R-Atrium → R-Vent.



- **The Most Common Cause of Cyanosis ("Blue Babies") *during* Infancy**
- **Leads to:**
    - Severe Hypoxia & Cyanosis
- **Typically Incompatible with Life:**
    - 30% die within a week
    - 90% die within a year
- **The Baby May Survive If:**
    - 1. The Foramen Ovale Remains Patent...and
    - 2. The Ductus Arteriosus Remains Patent
    - – These shunts maintain *some* communication between the parallel circuits → may prolong life.
    - – Therefore the doctor may give Prostaglandins to Maintain Patency of the Ductus Arteriosus as a short-term treatment until surgery is available.
- **Treatment (Surgery):**
    - ***Atrial* Switch (Mustard/Senning Procedure):**
        - Where the Systemic Venous Blood is diverted to the Left Atrium → L-Ventricle → Lungs.
        - And the Pulm-Venous (Oxy) Blood is diverted to the R-Atrium → R-Ventricle → Aorta
        - - **The Disadvantage =** The R-Heart becomes responsible for Systemic Circulation (which it is not designed to do)
    - ***Arterial* Switch:**
        - Where the Pulmonary Artery & Aorta are switched.



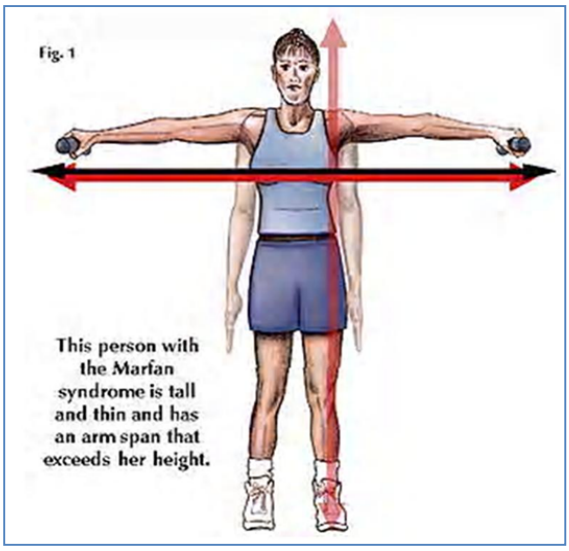
Atrial Swich Operation



Arterial Switch Operation

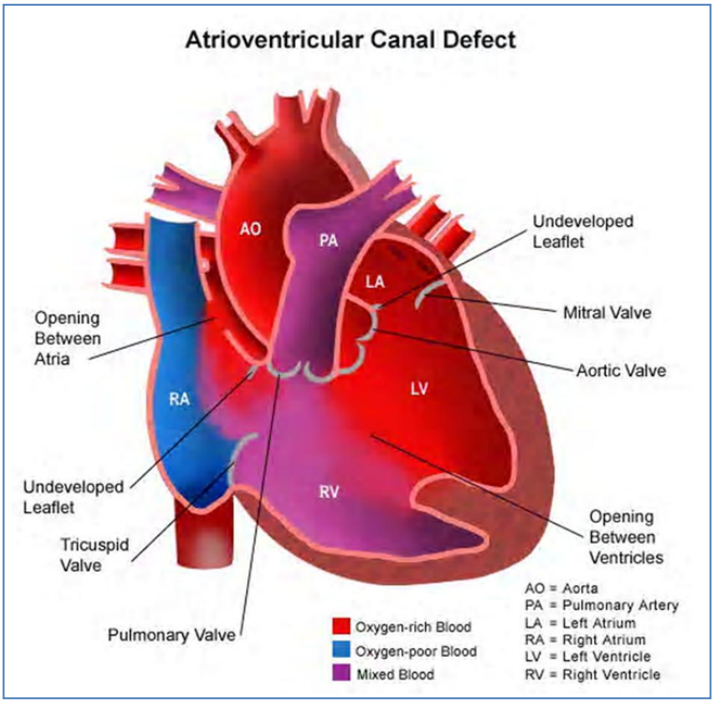
Marfan's Syndrome:

- Autosomal Dominant Disorder
- **Common CV Abnormalities**
    - Mitral Valve Prolapse - (L-AV Valve) this can lead to a mitral valve regurgitation. (blood will flow back from the ventricle to the atrium)
    - Tricuspid prolapse
    - ASD
    - Many More.....



Down's Syndrome:

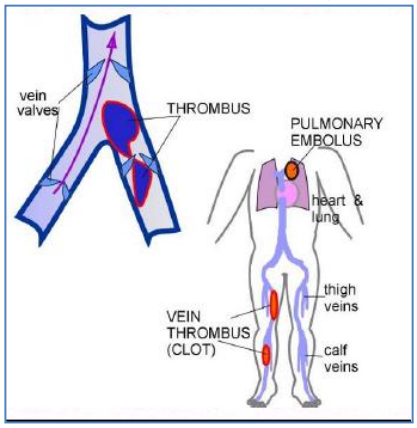
- 40% of Down's Syndrome Patients have congenital Heart Defects
- **Common CV Abnormalities:**
    - Atrio-Ventricular Canal - (Incomplete formation of AV Valves)
    - Atrial Septal Defects
    - Ventricular Septal Defects

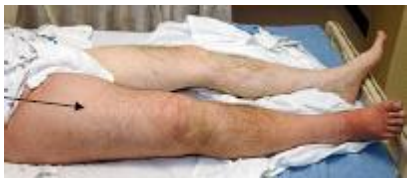
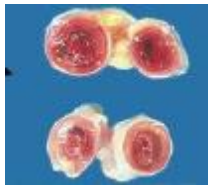
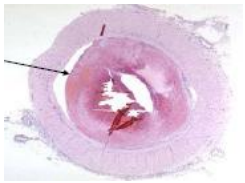


CVS Pathology:
DVT & PE

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

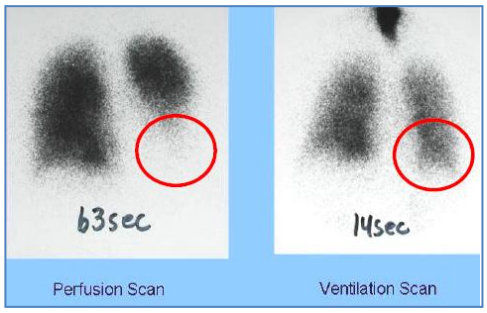
- **Aetiology:**
  - ****Deep* Venous Valve Incompetence of Lower Limbs:**
    - → Blood Stasis → Thrombosis
  - **+ ****Prolonged Immobilisation*:**
    - → Blood Stasis → Thrombosis
  - **+Risk Factors:**
    - **"Virchow's Triad"**
      - **1. Vessel Damage:**
        - Surgery/Smoking/Hypertension
      - **2. **Stasis**
        - Flight/Long Travel/Prolonged Bedrest/Surgery
        - Obesity/Pregnancy/Congestive Heart Failure
        - Post Operative
      - **3. Hypercoagulabiltiy**
        - Cancer (Eg. Adenocarcinoma → Paraneoplastic Syndrome)
        - Congenital: Eg. Antithrombin III Def./Factor 5 leiden
        - Drugs: Eg. Oral Contraceptive/HRT
        - Hyperviscosity: Eg. Pregnancy/Polycythaemia
- **Pathogenesis:**
  - **Failure/Inactivity of the Venous Calf Pump (Immobility/Valve Insufficiency)**
    - Blood Stasis & Pooling in Leg Veins → Coagulation → Thrombosis
- **Clinical Features:**
  - **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
        - → Sudden Chest pain, Dyspnoea, Haemoptysis, Collapse, Death.
- **Investigations:**
  - **Duplex Doppler USS –** (93% Sensitive; 98% Specific)
- **Management:**
  - ****Heparinization (LMW-Heparin) – 1wk**
    - **Then Convert to → Warfarin – 6mths**
  - **+/- Thrombectomy**
  - **+/- IVC Filter – (To Prevent Pulmonary Embolus)**

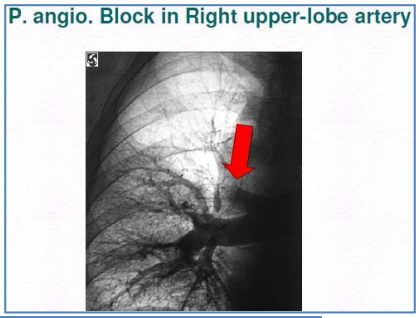


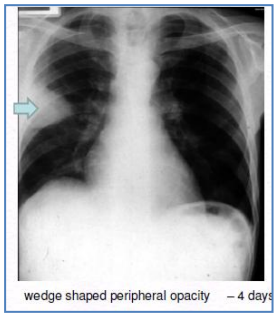




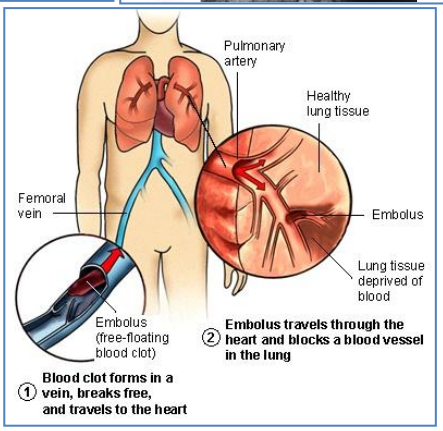
PULMONARY EMBOLISM

- **Aetiology:**
  - **95% = DVT → Thrombo-Emboli**
- **Pathogenesis:**
  - **DVT → Thrombo-Emboli Lodges in Pulmonary Arteries →**
    - ***1. → VQ-Mismatch*** *→ Respiratory Compromise* ***→ (Respiratory Failure)***
    - ***2. → ↑Pulm.Vas.Resistance*** *→ Haemodynamic Compromise* ***→ (Heart Failure).***
- **Clinical Features:**
  - Severity Depends on Size/Number of Emboli (Extent of Obstruction)
  - If Severe → Instant Death!! (Due to sudden Cardiac Failure)
  - **Symptoms →**
    - Pleuritic Chest Pain (+ Pleural Rub)
    - Dyspnoea/Tachypnoea
    - Cough/Haemoptysis
    - **(+ DVT Symptoms)**
  - **Signs:**
    - RV-Failure (↑JVP, Tricuspid Regurg)
    - Shock/Syncope
    - Fever
- **Diagnosis:**
  - ****CTPA (CT-Pulmonary Angiogram):** Shows Large Emboli lodged in Major Pulmonary Artery
  - **ECG:** Classical S1Q3T3 Pattern
  - **VQ Scan:** Shows VQ Mismatch
  - **CXR (Later >1day):** Shows Wedge-Shaped Pulmonary Infarct
- **Treatment:**
  - **Give Oxygen**
  - ****Heparinization (LMW-Heparin) – 1wk**
    - **Then Convert to → Warfarin – 6mths**
  - **TPA-Thrombolysis (If Haemodynamic Compromise)**
  - (+/- Trombectomy & IVC Filter)
- **Prevention (in High Risk Individuals):**
  - Elastic/Compression Stockings
  - Anticoagulation
  - If Severe Risk, Insertion of a IVC-Filter









CVS Pathology:
Dyslipidaemia

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.
      - NB: ↑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 – (**↓Sat.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/Rhabdomyelosis** → 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?

CVS Pathology:
HEART FAILURE

HEART FAILURE:

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

Where is the Failure?:

- **Myocardial Failure – (Ie. Systolic/Diastolic Dysfunction (Heart Muscle Itself)→ ↓Pumping Function):**
  - (Eg. Ischaemic Heart Disease, Myocarditis, Cardiomyopathies, etc.)
- **Valvular Heart Failure – (Ie. A problem with the Heart-Valves → ↓Pumping Function):**
  - (Eg. Stenosis/Regurgitation)
- **Circulatory Failure - (Ie. Defect in the Peripheral Circulation → Vascular System Dysfunction):**
  - (Eg. Haemorrhage/Shock)

What side is the Failure?

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

Signs of ↓Cardiac Output:

- **Thready Pulse** (Due to Low Arterial Pressure)
- **Tachycardia** (A Compensatory Mechanism)
- **Exercise Intolerance** (Due to ↓Tissue Perfusion & Pulmonary Congestion)
- **Dyspnoea** (Due to In Pulmonary Congestion)
- **Peripheral Oedema** (Venous Overload)

The Body's Responses to Heart Failure:

- **Short Term (Adaptive):**
  - **Peripheral Shutdown** (To maintain BP of Vital Organs. → ↑Afterload)
  - **Salt & H_2O Retention** (To ↑Blood Volume → ↑Preload)
  - **↑Preload** (To ↑ Stroke Volume)
  - **↑Sympathetic Tone** (To ↑ Heart Rate & Ejection)
  - **Hypertrophy** (To ↑ Muscle Mass to ↑ Contractile Strength)
- **Long Term (Maladaptive):**
  - **Peripheral Shutdown** → ↑Afterload → L-Heart Failure
  - **Salt & H_2O Retention** → Fluid Overload → Pulmonary & Peripheral Oedema
  - **Hypertrophy** → Myocardial Ischaemia + Diastolic 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**])**

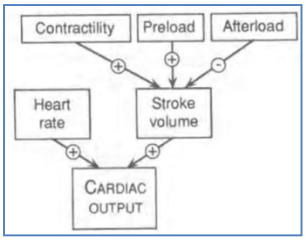
Heart Failure:

- Where an abnormality in cardiac function → ***poor cardiac output*** → isn't enough to supply the demands of the body → low perfusion of organs → hypoxia etc.
- Ie. The heart can't maintain circulation to tissues for normal metabolism.

Peripheral O_2 supply ≠ Peripheral O_2 demand

Revision of Cardiac Output

- **Cardiac Output = Heart Rate x Stroke Volume**
    - **= End Diastolic Volume – End Systolic Volume**
    - **≈ 70mL/min**
  - **Heart Rate Determined by:**
    - Chronotropic Factors
    - Autonomic Nervous System
  - **Stroke Volume Determined by:**
    - Contractility (NB: a direct effect of Ca^+ at the cellular level.)
    - Preload (Degree of Ventricular Filling During Diastole. High → ↑SV)
    - Afterload (ie. Resistance to Ventricular Ejection (ie. Due to ↑Aortic Blood Pressure)
- **'Ejection Fraction':** % of the End Diastolic Volume that is Ejected in Systole.
  - Stroke Volume/End Diastolic Volume
  - ≈60%



Types of Failure:

- **Heart Failure:**
  - Failure of the Heart *as an organ* to pump enough blood to satisfy the Periphery.
  - Myocyte Function May Be Normal.
    - **Eg. Heart Valve Failure:**
      - Ie. A problem with the Heart-Valves
      - Eg. Valve Incompetence/Regurgitation
      - Eg. Valve Stenosis (Narrowing)
      - Result = Changes in Preload/Afterload → Altered Cardiac Output
- **Myocardial Failure:**
  - Ie. Defect in Myocardial Contration (Heart Muscle Itself)→ Leads to deficit in Pump Function
  - Eg. Myocyte Death Due to Infarction →↓Myocyte Mass
  - Result = Decreased/Altered Pump Function
- **Circulatory Failure:**
  - Ie. Defect in the Peripheral Circulation
  - Eg. Haemorrhage/Shock/Cardiogenic Shock/Hypoxia
  - Result = Reduced Peripheral Perfusion
- **Congestion:**
  - Peripheral or Pulmonary Oedema - Due to:
    - Inadequate Pumping of the Heart
    - High Heart-'Filling'
    - High Venous Pressures → Backlog
    - Ie. When one side of the heart isn't '*pulling its weight*'→Backglog

Forward/Backward Heart Failure:

- **Forward Heart Failure:**
  - Reduced Output due to Inadequate Discharge of Blood into Arterial System.
- **Backward Heart Failure:**
  - Where One/Both Ventricle
    - 1. Fails to Discharge its Contents OR
    - 2. Fails to Fill Normally
  - Results in ↑Atrial Pressure + ↑Pressure in Venous System Behind the Failing Ventricle.
- **NB: Most Patients Have Both (Because Blood Flows in a Circle)**
  - Eg. Forward Heart Failure → Low Cardiac Output → Less Venous Return → Backward Heart Failure.

Left/Right Heart Failure:

- **Left Heart Failure:**
  - Inability of L-Heart to Pump into Systemic Circulation
  - **What Happens:**
    - Heart Tries to Compensate by Pumping Harder
    - →L- Heart Becomes Weaker
    - → ↑L-Ventricular End Systolic Volumes
    - → ↑L-Ventricular End Diastolic Volumes
    - → ↑L-Atrial Pressure
    - → ↑Pulmonary-Vein Pressure
    - → Shortness of Breath (Dyspnoea) & Pulmonary Oedema
    - → Fatigue

- **Right Heart Failure:**
  - Inability of R-Heart to Pump into Pulmonary Circulation
  - **What Happens:**
    - Blood Backs-Up in Peripheral Circulation
    - → ↑Venous Pressure
    - → Peripheral Oedema (Especially Legs & Abdominal Organs (Mainly Liver))
      - & Renal Insufficiency (Due to ↓Perfusion of Kidneys) – Hence Renal & Heart Failure go hand in hand.
    - Ie. A Congestive Heart Failure

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

Signs of ↓Cardiac Output:

- **Low Arterial Pressure** (Due to weaker heart muscle)
- **Tachycardia** (A Compensatory Mechanism)
  (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 – Grading of Heart-Failure Symptoms:

- 5 Classes

Class 1	No limitation to physical activity
Class 2	Slight limitation of activity. Dyspnea and fatigue with moderate exercise (eg walking upstairs quickly)
Class 3	Marked limitation of activity. Dyspnea with minimal activity
Class 4	Severe limitation of activity. Symptoms at rest
Class 5	Bed confinement. Life support monitoring

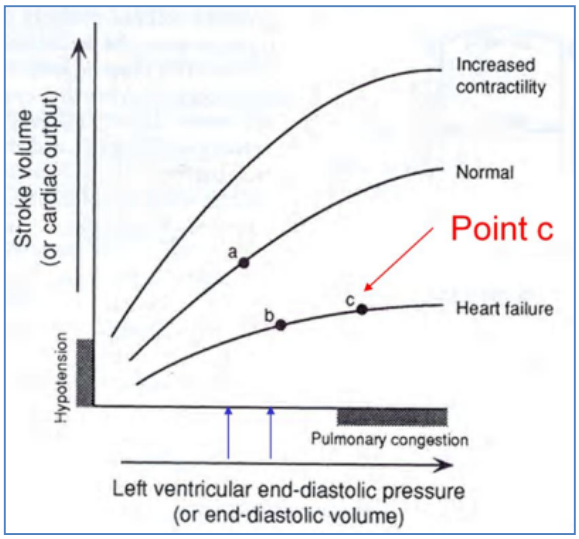
The Body's Responses to Heart Failure:

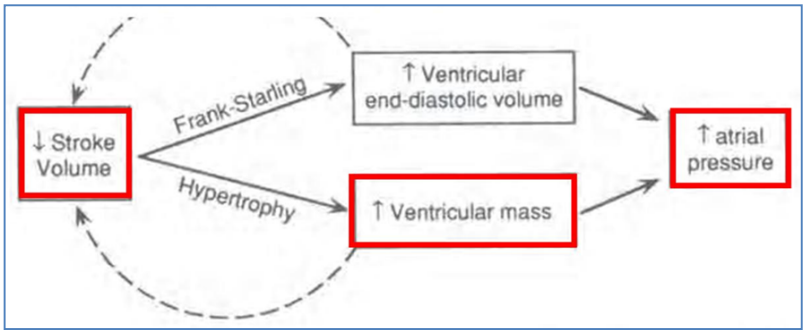
- **Short Term (ie. Acute Heart Failure):**
  - **Mainly Adaptive:**
    - Vasoconstriction (To maintain BP of Vital Organs. → ↑Afterload)
    - Salt & H_2O Retention (To ↑Blood Volume → ↑Preload)
    - Increased Preload (To ↑ Stroke Volume)
    - ↓Parasympathetic + ↑ Sympathetic (To ↑ Heart Rate & Ejection)
    - Increased HR (To ↑ Cardiac Output)
    - Hypertrophy (To ↑ Muscle Mass to ↑ Contractile Strength)

- **Long Term (ie. Chronic Heart Failure/Congestive Heart Failure):**
  - **Mainly Maladaptive:**
    Over a long period, the Heart simply Can't maintain the compensatory mechanisms of increasing CO.
    - Vasoconstriction → L-Heart Failure + ↑Myocardial Oxygen Consumption (MVO_2)
    - Salt & H_2O Retention → Pulmonary / Peripheral Oedema
    - Increased HR → ↑Energy Demand
    - Hypertrophy → Myocyte Energy Starvation + Impaired Relaxation

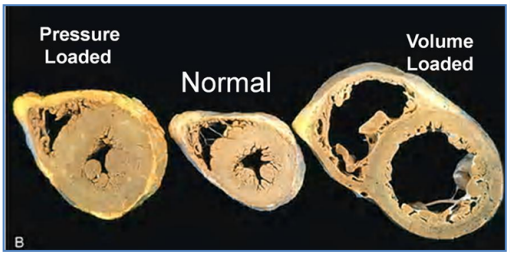
3 Compensatory Mechanisms:

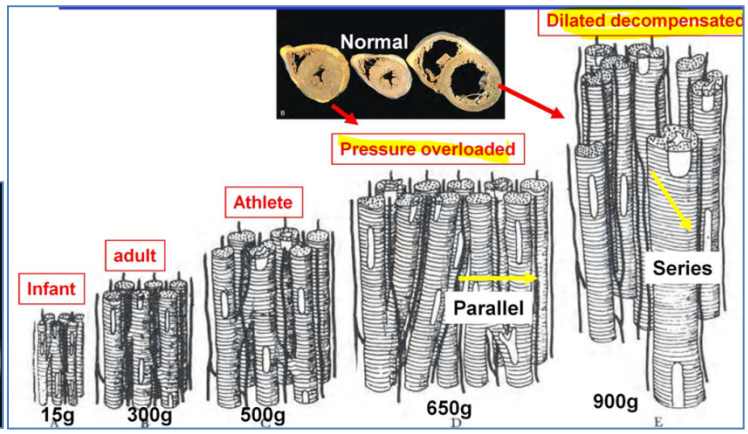
- **1. Frank-Starling Law/Mechanism:**
  - *"↑Preload → ↑Stroke Volume"*
  - Incomplete Chamber Emptying →↑PRELOAD → ↑Cardiac Output BY ↑STROKE-VOLUME.
  - BENEFICIAL in Short-Term
  - DETRIMENTAL in Long-Term
    - Ie. In Severe Heart Failure, Starling Curve is *Flatter* than normal.
    - →Even large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



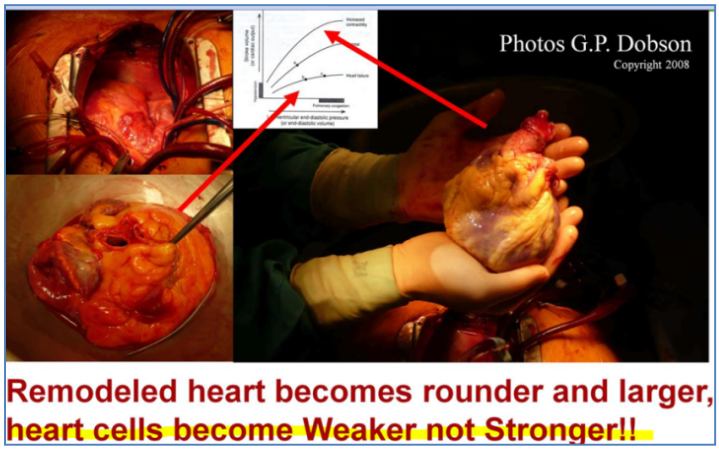


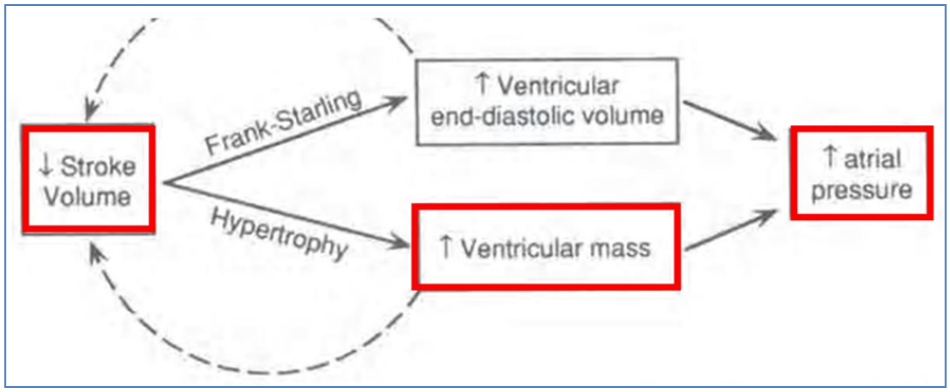
- **2. Myocardial Hypertrophy:**
  - **Pressure Overloaded Hypertrophy:**
    - In response to ↓Cardiac Output: When↓CO is due to ↑↑Afterload (↑Arterial Pressure)
    - **"Concentric Hypertrophy":** Muscle Thickens – Due to Synthesis of Sarcomeres in ***PARALLEL***.
  - **Volume Overloaded Hypertrophy:**
    - 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.***





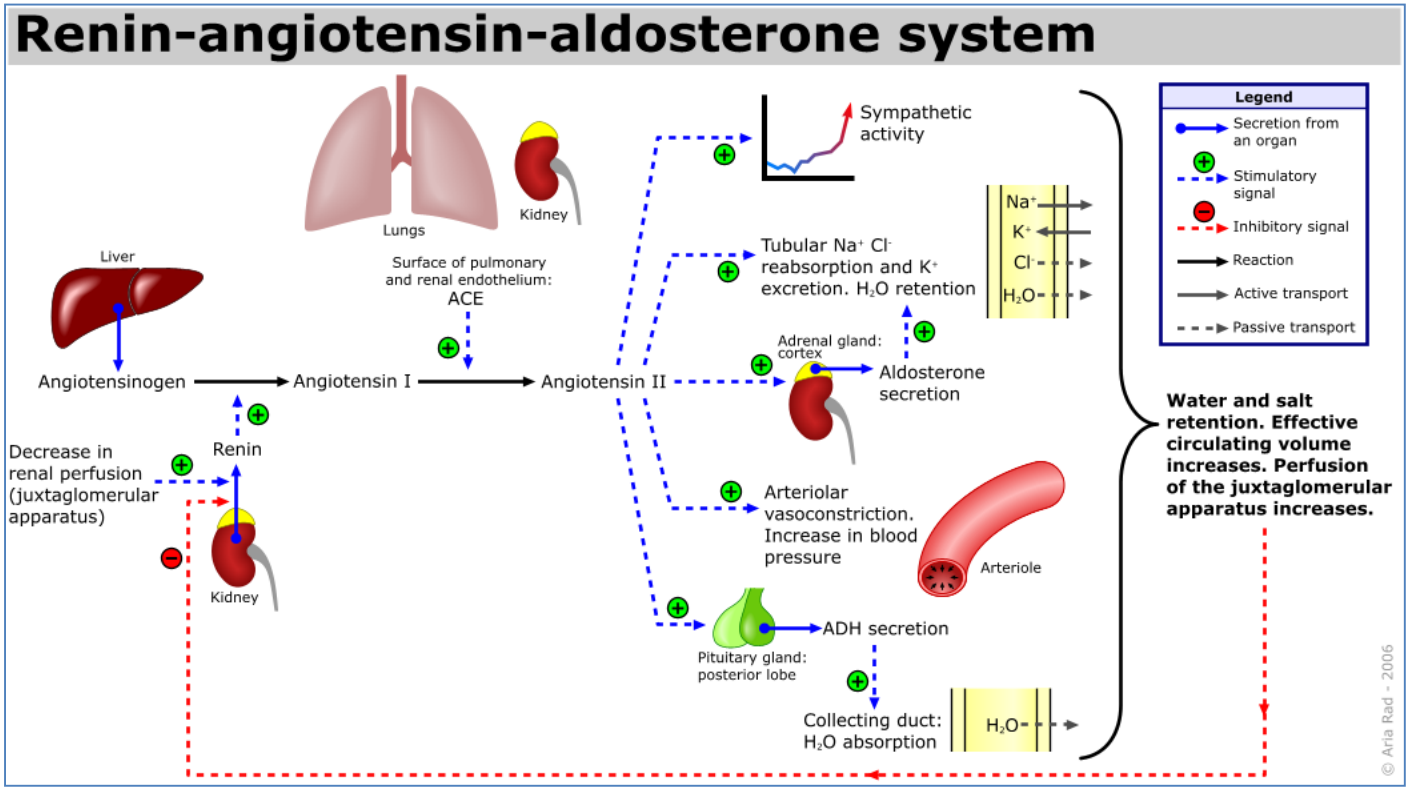
  - **Increased Ventricular Mass = Cell Hypertrophy (↑Size) & Hyperplasia (↑Numbers).**
    - Helps Maintain CO
    - Helps Reduce Wall Stress (By Increasing Thickness & Radius of Ventricle)
    - To Increase Contractility BUT Doesn't Work!
      - Heart becomes Rounder & Larger
      - Muscles Thicken, Lose Elasticity & Stiffen → Hard to relax.
        - →Decreased Compliance → ↑ESV → ↑Atrial Pressure → ↑Pul.Pressure.
      - Ventricle Stretches (Dilates) → Cannot Generate Enough Force to Pump Blood.
      - Cells Actually Become WEAKER!





- **3. Neurohormonal Systems:**
  - **Nor-Adrenaline/Epinephrine:**
    - *Baroreceptors* sense ↓CO as ↓Perfusion-Pressure →Stimulates Sympathetic:
      - →↑ Heart Rate
      - →↑ Contractility
      - →↑ Vessel Tone → To Increase Venous Return
      - →↑Preload (→SV →CO)

  - **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)



  - **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^+ & H_2O)

      } Therefore Inhibits RAAS.

  - **NB: The Neurohormonal Compensatory Mech = Viscious Cycle:**
    - Strain on heart → Activation of Neurohormonal Mechanisms →↑Preload & BP → Extra Strain on the heart.
    - Heart Responds by Remodelling → Larger & Rounder → Weaker.

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**

Complications:

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

Other Info:

Table 9. Signs and Symptoms of L vs. R Heart Failure

	Left Failure	Right Failure
low cardiac output (forward)	fatigue syncope systemic hypotension cool extremities slow capillary refill peripheral cyanosis MR Cheyne-Stokes breathing pulsus alternans S_3	TR S_3 (right-sided)
venous congestion (backward)	dyspnea orthopnea PND basal crackles cough hemoptysis	peripheral edema hepatomegaly hepatic tenderness pulsatile liver increased JVP positive HJR Kussmaul's sign

-
- **NYHA Functional Classification:**
  - **Class I:** No Limitation in ANY Activities; (Asymptomatic)
  - **Class II:** Slight Limitation of Activity; (Asymptomatic with Mild Exertion).
  - **Class III:** Marked Limitation of Activity; (Comfortable ONLY @ Rest).
  - **Class IV:** Symptoms Occur at Rest or with ANY Physical Activity.

CVS Pathology:
Hypertension

HYPERTENSION:

- **What is it?:**
  - Consistent **Systolic of +140mmHg.** **AND/OR**
  - Consistent **Diastolic of +90mmHg**

- **Aetiologies & Types:**
  - **95% = Primary/"Essential"/Idiopathic Hypertension:**
    - **Idiopathic** – Likely multifactorial (not curable)
    - **Risk factors for HT:**
      - GENETICS/FamHx
      - High Cholesterol/Salt Diet
      - Diabetes/Obesity
      - Smoking/Alcohol
      - Stress
      - Age
    - **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:**
    - **Cardio -** Coarctation, Hypervolaemia, Rigid Aorta
    - **Renal -** Acute Glomerulonephritis, CKD , Polycystic Kidneys, Renal Artery Stenosis
    - **Endocrine –** Hyper-Adrenalism, Acromegaly, Hypo/hyperthyroidism, Phaeo, Cushing's.
    - **Neurologic -** Psychogenic, Raised ICP, Sleep Apnea, Acute Stress
    - **Pre-Eclampsia:** (10% of pregnancies) - Placental Ischaemia → Placental vasoactive mediators → ↑Maternal BP in effort to ↑Placental Perfusion.
  - **(Accelerated/"Malignant" Hypertension):**
    - **= 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**
      - NB: "Malignant HTN" is rare, but can arise in HT of any Aetiology.
    - **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
    - **Symptoms Include:**
      - Vision Disturbance (Papilloedema/Retinal Bleed)
      - Headache, Drowsiness, Confusion
      - Nausea, Vomiting
    - **Management:**
      - *Smoothly* Reduce BP *over 24 to 36 hours* to <150 / 90
      - (NB: Excessive reduction may → Coronary/Cerebra/Renal Ischaemia)

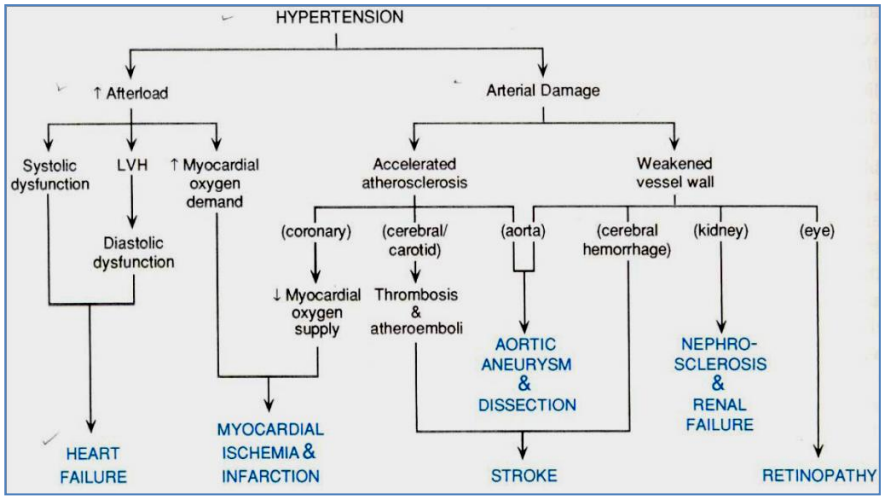
- **Clinical Features:**
  - **Symptoms:**
    - Typically Asymptomatic (Unless Malignant – Headache, Dizziness, N/V, Visual Changes)
  - **Signs:**
    - **Signs of 1° *causes*** – Eg. Thyroid, Cushing's, Acromegaly, Polycythaemia, CKD, Pregnancy.
    - **Abdomen:** Renal or Adrenal Masses (for possible causes), or for AAA
    - **Renal Bruit:** (Renal Artery Stenosis)
  - **Diagnosis – Essential Vs. Secondary?:**
    - **If Essential HT**: Diastolic Pressure will *RISE* on standing.
    - **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	80-90	83
Stage 1 Hypertension (Mild)	140-160	90-100	13.5
Stage 2 Hypertension (Moderate)	160-180	100-110	2
Stage 3 Hypertension (Severe)	180-210	110-120	
Stage 4 Hypertension (Severe)	≥210	≥120	1

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

- **Complications of Hypertension:**
  - **Is a Major Precursor For:**
    - CAD/IHD
    - Hypertensive Heart Disease (Heart Failure, Hypertrophic Cardiomyopathy)
    - Stroke
    - Aortic Dissection
    - PVD
    - Renal Failure
    - Microangiopathy/*'Arteriolosclerosis'* (Small Vessel Diseases)
  - 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)
  - **Aortia/Peripheral Vascular:**
    - Mechanical Arterial Damage (eg. Aneurysms/Dissecting Aneurysms/Atherosclerosis)
  - **Kidneys:**
    - Nephrosclerosis – (hardening of kidney blood vessels) → Renal Failure

CVS Pathology:
Infective Endocarditis

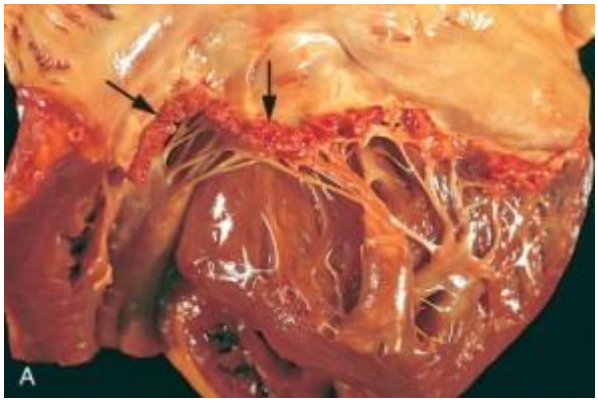
INFECTIVE ENDOCARDITIS

- **= 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
  - IV Access – (Haemodialysis, IVDU, Surgery)
  - Immunosuppression
- **Aeitologies:**
  - **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.

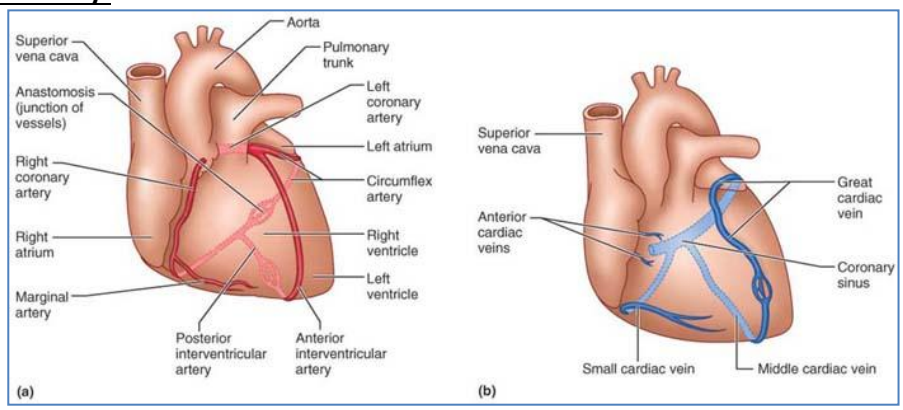
NON-INFECTIVE ENDOCARDITIS (NBTE - Non Bacterial Thrombotic Endocarditis):

- **Aetiology:**
  - **Hypercoaguable 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>Pulomonary
- **Clinical Signs:**
  - **Signs:**
    - **Of Hypercoaguable 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 2° Infective-BE:**
      - Fever + New Murmur
      - Septic Infarcts (Splinters, Oslers, Janeways, Roths, 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 2° 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



CVS Pathology:
Ischaemic Heart Disease

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:**

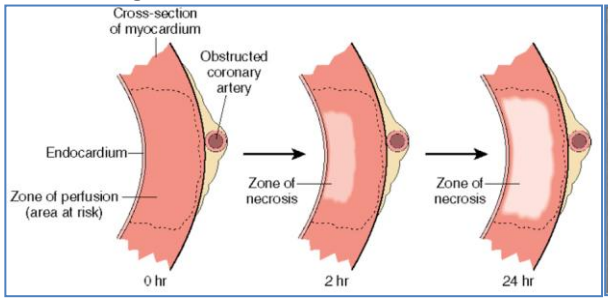
- **Ischaemia:** A '*FLOW' Limitation*, Typically due to Coronary Artery Stenosis (Narrowing)
- **Hypoxia:** An 'O_2' *Limitation,* Typically due to High-Altitude/Respiratory Insufficiency/etc.
- **Infarction:** *Irreversible Cell-DEATH*, Typically due to sustained Ischaemia.

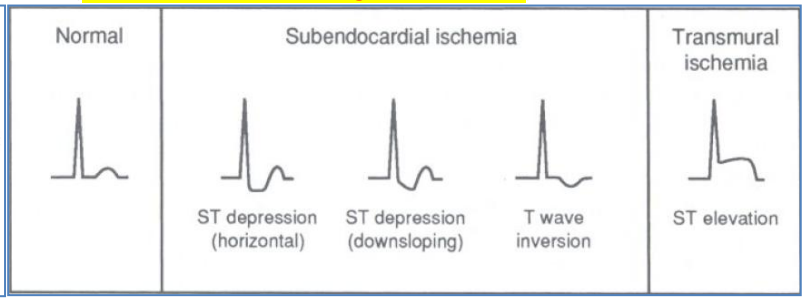
Regional Vs. Global Myocardial Ischaemia:

- **Regional Ischaemia:**
  - Local Atherosclerosis/Thrombosis → Ischaemia Confined to Specific *Region* of Heart.
- **Global Ischaemia (Rare):**
  - Severe Hypotension/Aortic Aneurysm → Ischaemia of *Entire* Heart

What Happens During Myocardial Ischaemia:

- **Myocardial Damage:** Initially '*Subendocardial*'-Ischaemia/Infarction (ST-*Depression & T-Wave Inversion*)→ Progresses to *'Transmural'*-Ischaemia/Infarction (ST-*Elevation & Pathological Q-Waves*).





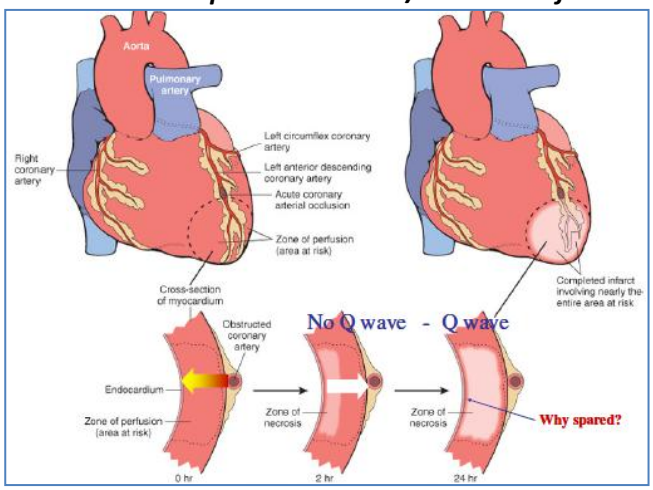
- **Metabolic Changes – (Aerobic → Anaerobic):** ↑Lactate (Anaerobic Metabolism) & ↓pH
- **Pain:** Nociceptor (pain receptor) Activation → Angina Pain
- **Global Autonomic Symptoms:** Tachycardia, Sweating, Nausea.
- **Pulmonary Congestion:** Eg. LV-Failure → Pulmonary Congestion → Shortness of Breath
- **Ventricular Arrhythmias:** Eg. SVT or VT or VF (due to Re-Entrant Focus & Altered Conduction Patterns)

ANGINA PECTORIS:

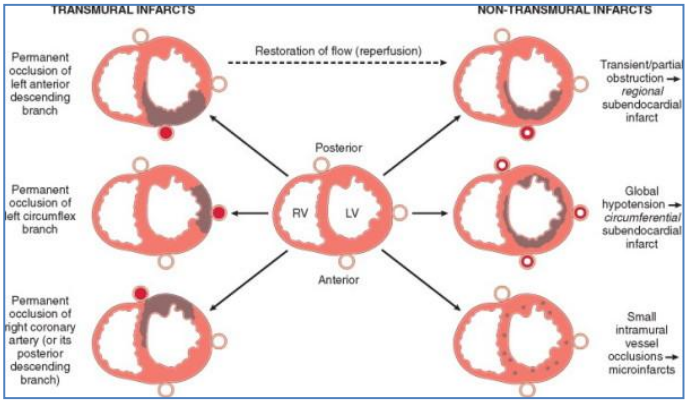
- **Aetiology:**
  - **↓Myocardial Perfusion (relative to demand) due to Coronary Insufficiency**.
  - **Causes:** **Atherosclerosis / Vasospasm / Embolism / Ascending Aortic Dissection
  - **Exacerbated by –** (Vent-Hypertrophy, Tachycardia, Hypoxia, Coronary Arteritis (e.g. in SLE))
- **Pathogenesis:**
  - (= *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).
    - **NB: = 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 (Pulm.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 - (Unstable Angina/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 + Path.Q-Waves*
    - ***NB: The Endocardium is spared due to O2/Nutrients of Ventricular Blood.***

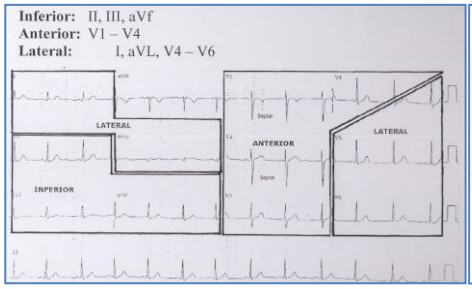


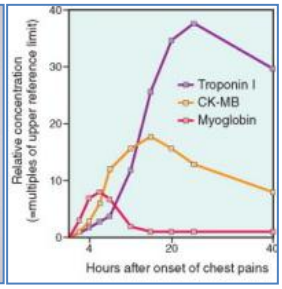
  - **Most Common Coronary Obstruction & Locations of Ischaemia:**
    - **50% - LAD Obstruction:**
      - Anterior-LV + Apex + Ant.2/3 of IV-Septum
    - **30% - RCA Obstruction:**
      - Posterior-LV + Posterior Septum + Free wall of RV.
    - **20% - LCX (Left Circumflex) Obstruction:**
      - Lateral LV (except for the apex.)
    - (NB: Nearly ALL Infarcts involve a portion of the LV)



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

- **Investigations:**
  - **(1st Line):**
    - **Serial Resting 12Lead ECGs – (Every 15 Mins):**
      - **ST-Changes and Diagnosing MI:**
        - **V1, V2, V3, V4 = Anterior MI**
        - **II, III, AVF = Inferior Wall MI**
        - **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**)**





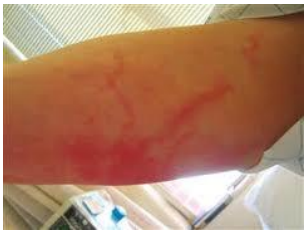
  - **(2nd Line):**
    - **TTE/TOE – Transthoracic/Transoesophageal Echo:**
      - Assess LV-Function
      - (+ Excludes DDXs - Aortic Dissection / Pericarditis / Pulmonary Embolism)
    - **Myocardial Perfusion Scans (Nuclear Medicine):**
      - ? Location of Infarct

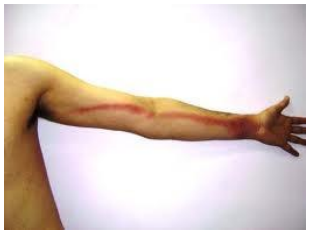
- **Management (As with Angina *PLUS MORPHINE, O2 & ANTICOAGULATION + DEFINITIVE Mx*):**
  - ***(Simplified: MONA = Morphine, Oxygen, Nitrates, Aspirin)***
  - **1. Medical Therapy (*Maintenance*):**
    - **1. Anti-Anginal Therapy:**
      - Nitrates (GTN/Isosorbide Mononitrate) – Coronary Vasodilation →↑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).
  - **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:**
- **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.**

CVS Pathology:
Lymphangitis

LYMPHANGITIS:

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





CVS Pathology:
Myocarditis - Viral & Toxic

MYOCARDITIS – VIRAL & TOXIC:

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

MYOCARDITIS

❑ inflammatory process involving the myocardium (an important cause of dilated cardiomyopathy)

Etiology

❑ idiopathic
❑ infectious
- viral: Coxsackie virus B, Echovirus, Poliovirus, HIV, mumps
- bacterial: S. aureus, C. *perfringens*, C. *diphtheriae*, Mycoplasma, Rickettsia
- fungi
- spirochetal (Lyme disease – B*orrelia burgdorferi*)
- Chagas disease (T*rypanosoma cruzi*), toxoplasmosis

❑ acute rheumatic fever (Group A β-hemolytic S*treptococcus*)
❑ drug-induced: emetine, doxorubicin
❑ collagen vascular disease: systemic lupus erythematosus (SLE), polyarteritis nodosa (PAN), rheumatoid arthritis (RA), dermatomyositis (DMY)
❑ sarcoidosis
❑ giant cell myocarditis

Clinical Manifestations

❑ constitutional illness
❑ acute CHF
❑ chest pain - associated pericarditis or cardiac ischemia
❑ arrhythmias (may have associated inflammation of conduction system)
❑ systemic or pulmonary emboli
❑ sudden death

Investigations

❑ 12 lead ECG
- non-specific ST-T changes +/– conduction defects

❑ blood work
- increased CK, Troponin, LDH, and AST with acute myocardial necrosis +/– increased WBC, ESR, ANA, rheumatoid factor, complement levels

❑ perform blood culture, viral titers and cold agglutinins for Mycoplasma
❑ chest x-ray
- enlarged cardiac silhouette

❑ echocardiography
- dilated, hypokinetic chambers
- segmental wall motion abnormalities

Natural History

❑ usually self-limited and often unrecognized
❑ most recover
❑ may be fulminant with death in 24-48 hours
❑ sudden death in young adults
❑ may progress to dilated cardiomyopathy
❑ few may have recurrent or chronic myocarditis

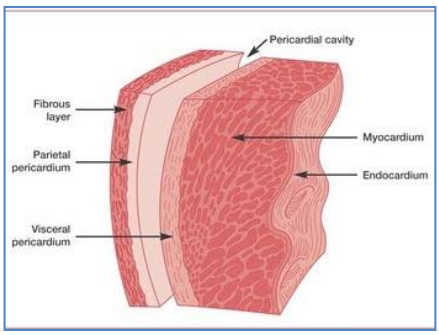
Management

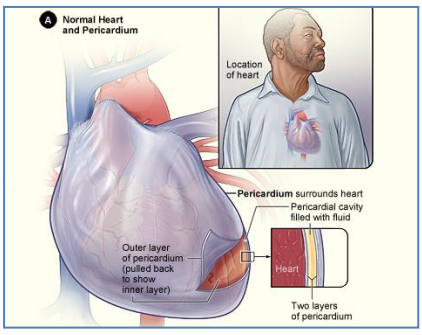
❑ supportive care
❑ restrict physical activity
❑ treat CHF
❑ treat arrhythmias
❑ anticoagulation
❑ treat underlying cause if possible

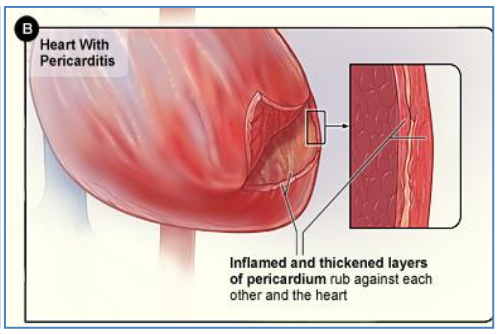
CVS Pathology:
Pericarditis

PERICARDITIS:

- **Aetiology:**
  - **Usually *Secondary* to:**
    - **Infection** (**Viruses**, Bacteria, Fungi, Parasites)
    - **Immuno** (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







CONSTRICTIVE PERICARDITIS

Definition
- ❑ chronic pericarditis resulting in fibrosed, thickened, adherent, and/or calcified pericardium

Etiology
- ❑ any cause of acute pericarditis may result in chronic pericarditis
- ❑ major causes are tuberculous, radiation-induced, post-cardiotomy, idiopathic

Symptoms
- ❑ dyspnea, fatigue, palpitations
- ❑ abdominal pain

Signs
- ❑ general examination - mimics CHF (especially right-sided HF)
  - ascites, hepatosplenomegaly, edema
- ❑ 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
- ❑ chest x-ray
  - pericardial calcification, effusions
- ❑ CT/MRI/TEE
  - pericardial thickening
- ❑ cardiac catheterization
  - equalization of RV and LV diastolic pressures, RVEDP > 1/3 of RV systolic pressure

Management
- ❑ medical: diuretics, salt restriction
- ❑ surgical: pericardiectomy

PERICARDIAL EFFUSION

Etiology
- ❑ two 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 hemopericardium (trauma, post MI myocardial rupture, aortic dessection)
- ❑ physiological consequences depend on type and volume of effusion, rate of effusion development, and underlying cardiac disease

Symptoms
- ❑ none or similar to acute pericarditis
- ❑ dyspnea, cough
- ❑ extra-cardiac (esophageal/recurrent laryngeal nerve/tracheo-bronchial/phrenic nerve irritation)

Signs
- ❑ JVP: increased with dominant "x" descent
- ❑ arterial pulse: normal to decreased volume, decreased PP
- ❑ auscultation: distant heart sounds +/– rub

Investigations
- ❑ 12 lead ECG
  - low voltage, flat T waves
- ❑ chest x-ray
  - cardiomegaly, rounded cardiac contour (water bottle)
- ❑ ECHO (procedure of choice)
  - fluid in pericardial sac
- ❑ pericardiocentesis
  - establishes diagnosis

Management
- ❑ mild: frequent observation with serial ECHO, treat the cause, anti-inflammatory agents for inflammation
- ❑ severe: may develop cardiac tamponade

CARDIAC TAMPONADE

- ❑ major complication of pericardial effusion
- ❑ cardiac tamponade is a clinical diagnosis

Pathophysiology and Symptomatology

- ❑ high intra-pericardial pressure —> decreased venous return —> decreased diastolic ventricular filling decreased CO —> hypotension + venous congestion
    - symptoms
        - tachypnea, dyspnea, shock

Signs

- ❑ "x" descent only, absent "y" descent
- ❑ hepatic congestion

Clinical Pearl

- ❑ **Classic quartet: hypotension, increased JVP, tachycardia, pulsus paradoxus.**
- ❑ **Beck's triad: hypotension, increased JVP, 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 and RV) in diastolic
- ❑ cardiac catheterization
    - mean RA, LA, LV and RV diastolic pressures all high and equal

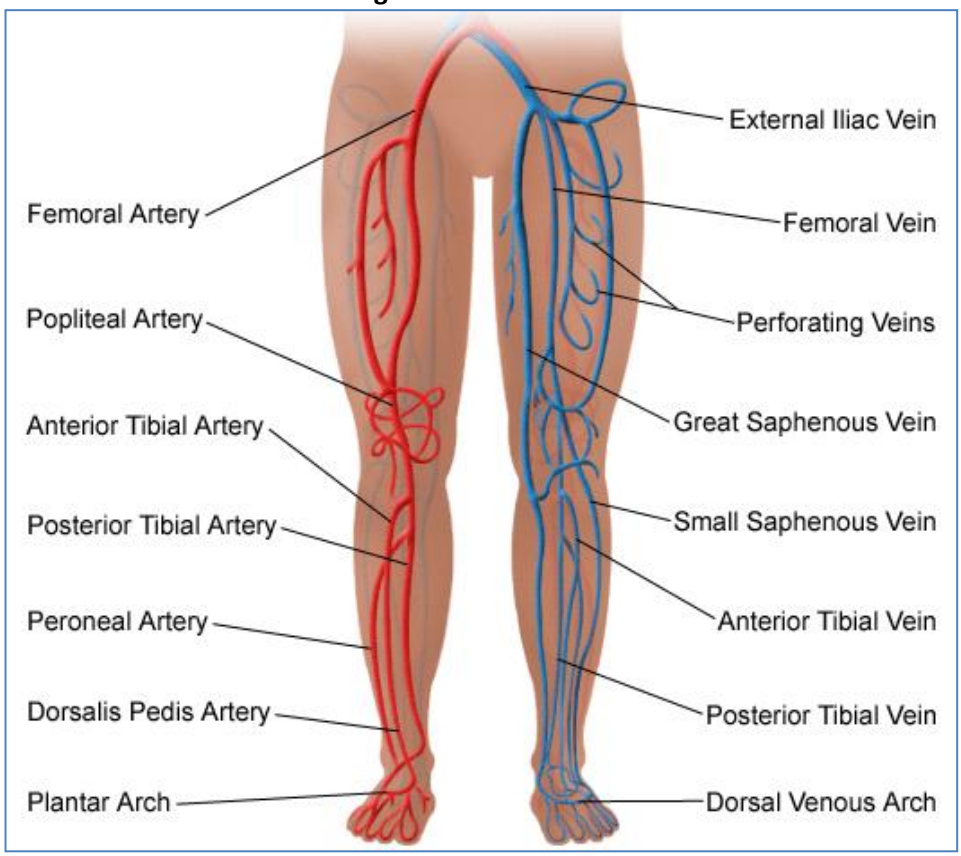
Management

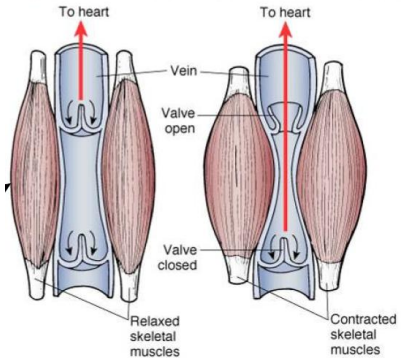
- ❑ pericardiocentesis – ECHO-, ECG-guided
- ❑ pericardiotomy
- ❑ avoid diuretics and vasodilators (these decrease venous return to already under-filled RV —> decrease LV preload —> decrease CO)
- ❑ fluid administration may temporarily increase CO
- ❑ treat underlying cause

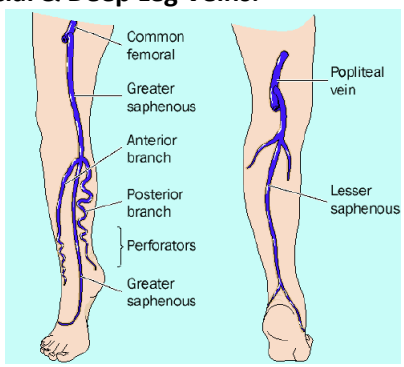
CVS Pathology:

Peripheral Vascular Disease, Varicose Veins & Chronic Leg Ulcers

Revision of Lower Limb Vascular Anatomy:

- **Arterial & Venous Circulation of the Legs:**
  - 

- **"The Muscle Pump":**
  - 

- **Superficial & Deep Leg Veins:**
  - 

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

ACUTE ARTERIAL OCCLUSION/INSUFFICIENCY

- ❑ due to embolus, arterial thrombosis or trauma. Time is of essence, after approximately 6 hours (depending on collaterals), ischemia and myonecrosis is irreversible to limb

Embolus

- ❑ etiology
  - cardiac is the source of 80-90% of embolic episodes; History of MI (< 3 months), rheumatic heart disease, abnormal or prosthetic valves, A fib, MS, cardiomyopathy, endocarditis, atrial myxoma
  - arterial source – proximal arterial source such as aneurysm, atheroembolism
  - paradoxical embolism with a history of venous embolus passing through intracardiac shunt
  - other including a history of medications (oral contraceptives), previous emboli, neurologic / TIAs
- ❑ presentation
  - sudden pain in lower extremity progressing within hours to a feeling of cold numbness, loss of function and sensation
  - no history of significant vascular claudication
  - pulses are present in contralateral limb
  - may have emboli to other locations (cerebral, upper limb, renal)

Arterial Thrombosis

- ❑ etiology
  - it is important to differentiate thrombosis from embolism because the treatment for the two may vary dramatically
  - thrombosis usually occurs in a previously diseased (atherosclerotic) artery, congenital anomaly, infection, hematological 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

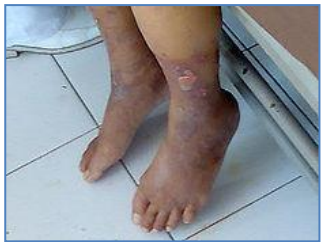
Trauma

- ❑ etiology
  - it is important to determine a history of arterial trauma, arterial catheterization, intra-arterial drug induced injection, aortic dissection, severe venous thrombophlebitis, prolonged immobilization, idiopathic
- ❑ symptoms
  - symptoms (**6 P's**)
    - **P**ain: absent in 20% of cases because of prompt onset of anesthesia and paralysis
    - **P**allour: replaced by mottled cyanosis within a few hours
    - **P**arasthesia: light touch goes first (small fibers) followed by other sensory modalities (large fibers)
    - **P**aralysis / **P**ower loss: heralds impeding gangrene
    - **P**olar (cold)
    - **P**ulselessness
  - do not expect all of the 6 P's to be present and do not rely on pulses
  - of the 6 P's the most important are paralysis / power loss
  - full cardiac exam including complete bilateral pulse examination
  - atrophic skin and nail changes - longstanding arterial insufficiency
- ❑ investigations
  - CXR, ECG, arteriography
- ❑ management
  - immediate heparinization at 5000iu bolus and continuous infusion to maintain PTT > 60
  - in the absence of power and sensation – need emergent re-vascularization: (i) for embolus – embolectomy; (ii) for thrombus – bypass
  - in the presence of power and sensation – need work-up – including angiogram: (i) for embolus – embolectomy; (ii) for thrombus - bypass
  - embolectomy: Fogarty catheter tied to fish embolus out of artery
  - bypass: bypass occlusion allowing blood flow to resume to distal site
  - identify and treat underlying cause
  - continue heparin post-op, start warfarin post-op day 1 for 3 months
  - re-perfusion phenomenon
  - toxic metabolites from ischemic muscle —> renal failure and multi-organ system failure
- ❑ complications
  - beware compartment syndrome with prolonged ischemia; requires fasciotomy
- ❑ treatment of irreversible ischemia is amputation
- ❑ prognosis
  - 12-15% mortality rate
  - 5-40% morbidity rate (amputation)

- **NB: Emboli Can also Deposit in *Other* Arteries Too:**
  - Eg. Mesenteric Ischaemia → Ischaemic Gut (++ Painful + Bloody Diarrhoea)
  - Eg. Renal Art.Thrombosis → Abdo/Back/Flank Pain, ARF, Oliguria, Hypertension

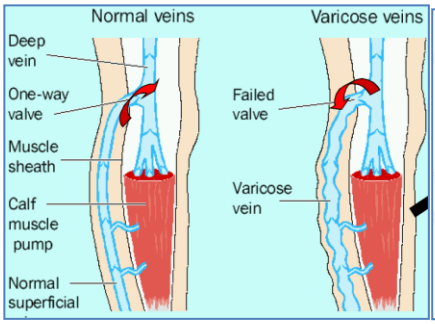
PERIPHERAL VASCULAR DISEASE (AKA: *Peripheral ARTERIAL Disease*):

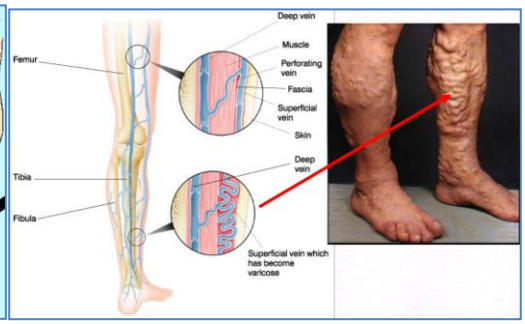
- **Definition:**
  - Obstruction of any of the *PERIPHERAL ARTERIES* (Not including Coronaries/Aortic Arch/Brain)
- **Aetiologies:**
  - ****Atherosclerosis (Most Common)**
  - **(Major Risk Factors):**
    - Smoking (10x) - the single greatest *modifiable* cause of PVD.
    - Diabetes
    - Dyslipidaemia
    - 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
  - (NB: 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



VARICOSE VEINS:

- **Aetiology:**
  - **Mechanical:** Prolonged Leg Dependence
  - (Risk Factors = Obesity, Pregnancy, Familial)
- **Pathogenesis:**
  - ***Superficial Valve Incompetence*** (Due to Incompetent Valves & Venous Dilation)
    - → Further Venous Stasis, Congestion, Oedema, Pain & Thrombosis.
  - **(NB: Can Also Occur in Oesophagus, Rectum, & Scrotum)**
- **Clinical Features:**
  - **Symptoms:**
    - Diffuse Aching, Tightness & Night-Cramping
    - Persistent Leg Oedema
    - ↓Wound Healing
  - **Signs:**
    - Distended, Tortuous Superficial Veins
    - Ischaemic Skin Changes (Eg. *Stasis Dermatitis*)
    - Venous Leg Ulcers
  - NB: Embolism is RARE since *only Superficial Veins* are affected!!!!
  - **Complications:**
    - Recurrent ***Superficial Thrombophlebitis*** (See Below)
    - Lipodermatosclerois
    - Haemorrhage
    - Ulceration
- **Investigation:**
  - **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:**
  - **Conservative:** Elevation + Compression Stockings
  - **Surgical:** **"**Stripping" of Varicose Veins



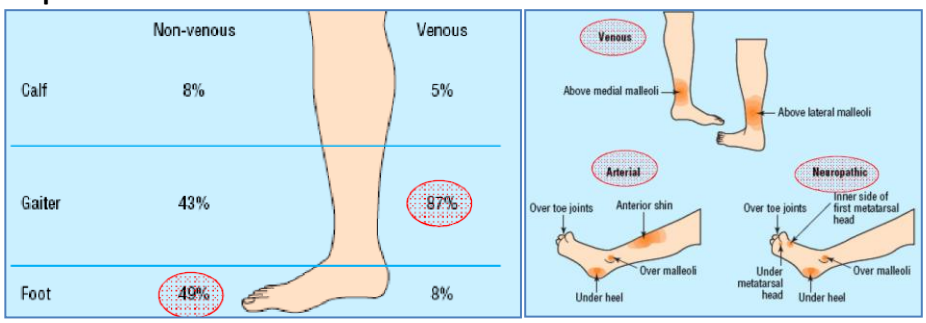


SUPERFICIAL THROMBOPHLEBITIS

- ❑ inflammation or thrombosis of any superficial vein
- ❑ etiology
  - trauma
  - association with varicose veins
  - migratory superficial thrombophlebitis
  - Buerger's disease
  - SLE
  - polycythemia
  - thrombocytosis
  - occult malignancy (especially pancreas)
  - idiopathic
- ❑ a pulmonary embolus is rarely present with superficial thrombophlebitis
- ❑ signs and symptoms
  - pain and cord-like swelling along course of involved vein;
  - most commonly involves long saphenous vein or its tributaries
  - red, warm, indurated vein
- ❑ investigations
  - non-invasive tests to exclude associated DVT (5-10%)
- ❑ treatment
  - conservative
    - bed rest and elevation of limb
    - moist heat, compression bandages, mild analgesic, anti-inflammatory and anti-platelet (e.g. ASA), ambulation
  - surgical excision of involved vein indicated if conservative measures fail
    - of suppurative thrombophlebitis - IV antibiotics and excise involved vein
- ❑ complications
  - chronic recurrent superficial thrombophlebitis

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

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

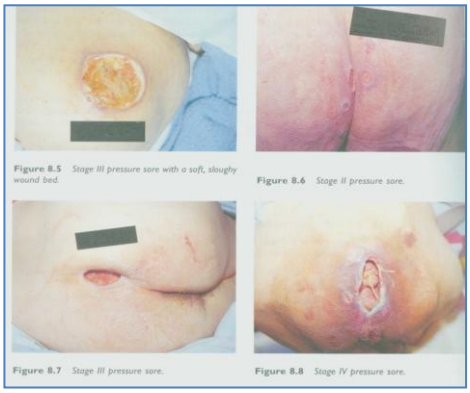


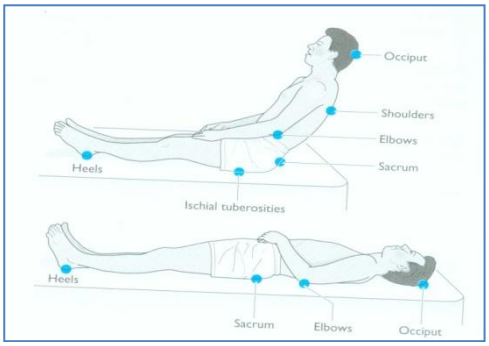
LEG ULCERS

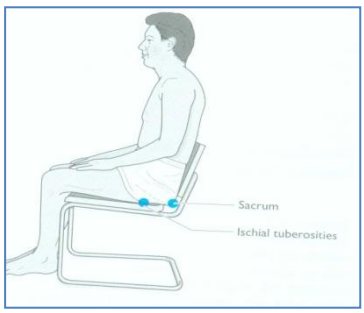
Table 11. Venous vs. Arterial Ulcers vs. Diabetic Ulcers

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

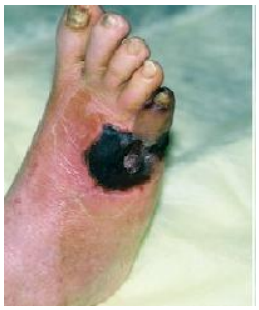
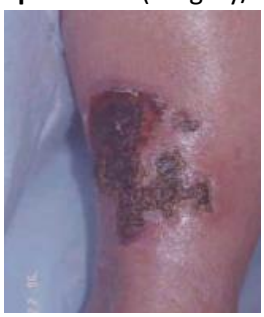
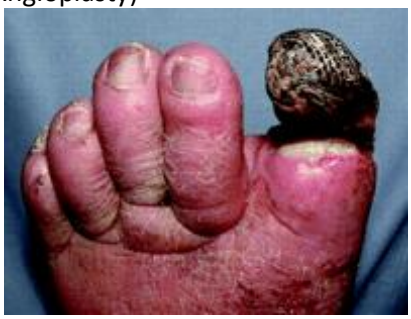
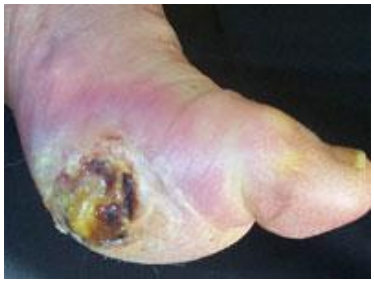
- **PRESSURE ULCERS:**
  - **Aetiology:**
    - **Long-Term Pressure** – (*Elderly, frail, bedridden, paraplegia, coma*)
  - **Pathogenesis:**
    - Long-term skin pressure → Skin Ischaemia → Necrosis → Ulcer
  - **Clinical Features:**
    - **Location & Appearance:**
      - Bony Prominences – (sacrum, 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



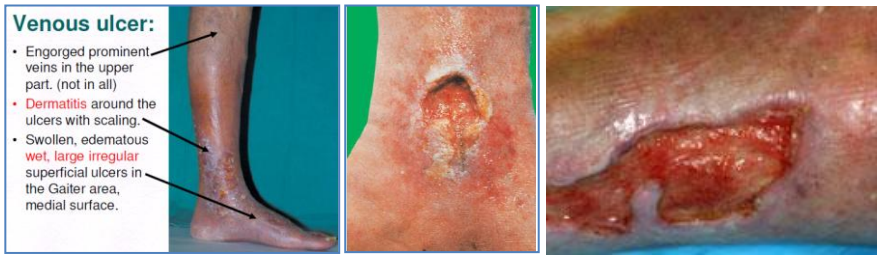




- **ARTERIAL ULCERS:**
  - **Aetiology:**
    - Arterial Insufficiency (PVD) – (Typically due to ***Atherosclerosis***)
    - **Common in Diabetes
  - **Pathogenesis:**
    - Arterial Insufficiency → Tissue Hypoxia/Ischaemia → Skin Necrosis + ↓Wound Healing
    - (NB: Often occurs following Trivial Trauma 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)

- **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

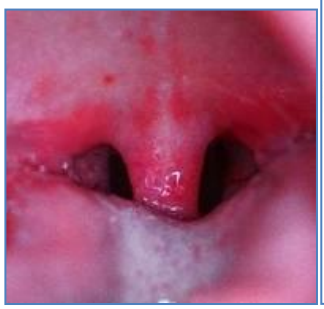


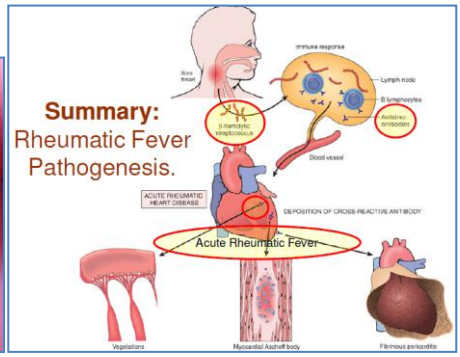
- **NEUROPATHIC ULCERS:**
  - **Aetiology:**
    - ****Diabetic Neuropathy + ****Arterial Insufficiency**
  - **Pathogenesis:**
    - **Diabetic Neuropathy**→ Damage/Injury goes Unnoticed → Further Tissue Damage/Necrosis
    - **(+ Arterial Insufficiency** → Tissue Hypoxia/Ischaemia → Tissue Damage/Necrosis**)**
  - **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)

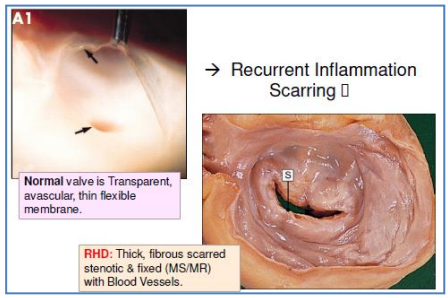
CVS Pathology:
Rheumatic Fever & Rheumatic Heart Disease

RHEUMATIC FEVER & RHEUMATIC HEART DISEASE

- **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)
  - **(NB: 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-Haemolitic 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 → Mitral Stenosis:**
  - **1. GABH Strep Pharyngitis** (In *HLA-DR2/3-Positive* Person)
  - **2. 2wks Later, Immune Response to GABH-Strep→ Rheumatic Fever → Carditis**
    - (NB: 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**







- **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)
      - ♥ **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)

- **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).
    - → **Pulm.Congestion & CCF –** (RV-Hypertrophy, Exertional Dyspnoea)
  - **Atrial Fibrillation** (From Atrial Stretch due to Mitral Stenosis)
  - **↑Risk of Infective Endocarditis**

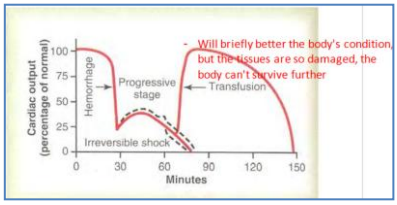
- **Management:**
  - **(Primary Prevention – Rx of Strep Pharyngitis):**
    - **10days PO Penicillin-V (Or Amoxicillin or Cephalexin)**
  - **Secondary Prevention:**
    - **Admit on Suspicion:**
      - Based on Jones Criteria
    - **Treating *Acute Rheumatic Fever*:**
      - **GABH Strep Eradication –** (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

- ***GLS Question - What is the difference between Rheumatic Fever (RF) & Rheumatic Heart Disease (RHD)?***
  - **(NB: 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.

CVS Pathology:
Shock

SHOCK:

- **EXAM Definition = "Indequate Perfusion of *Vital* Organs (Heart/Brain/Kidneys)"**
- **Aetiologies:**
  - **Hypovolemic Shock:**
    - Severe Dehydration - (Eg. Sweating, Vom/Dia, DKA & Diuresis, Seeping Burns)
    - Severe Blood Loss/Haemorrhage
  - **Cardiogenic Shock:**
    - Heart Failure - (Eg. Acute MI, Valvular, Cardimyopathy, Myocarditis)
  - **Distributive Shock:**
    - **Septic Shock** – (Extracellular Fluid Shift → Hypotension → Shock)
    - **Anaphylactic Shock** – (Extracellular Fluid Shift → Systemic Oedema & Hypotension).
    - **Neurogenic Shock** – (Sudden loss of Vasomotor Tone → Massive VenoDilation)
  - **Obstructive Shock:**
    - **Massive PE**
    - **Cardiac Tamponade –** (Massive Pericardial Effusion → ↓Ventricular Filling → ↓SV & CO)
    - **Tension Pneumothorax**
- **Compensatory Mechanisms:**
  - ***"CARDIAC RESERVE" =*** *Maximal % that CO can Increase Above Normal. (Typically 300-400%)*
  - **(IMMEDIATE) ↑Sympathetic Tone:**
    - Baroreceptors → ↑SNS → ↑HR & Contractility → ↑CO
  - **(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):**
    - **Stable & Reversible.**
    - **Signs of Compensated Hypovolaemia:**
      - *Tachycardia*
      - *Oliguria* (Low Urine Production)
  - **2. Progressive Stage (15-40% (750-2000mL)Blood Loss):**
    - **Unstable, Decompensating, Reversbile.**
    - **Signs of Decompensation:**
      - *Hypotension*
      - *Delayed CRT* (↓Peripheral Perfusion)
      - *Tachycardia*
      - *Organ Failure* (Anuria, Confusion/ALOC, Heart Failure, Tachypnoea, Acidosis)
    - **But *Still Reversible* with Treatment:**
      - Reverse Causative Agents + Volume Replacement ***(Bolus 2L IV)*** +/- Inotropes
      - **(Otherwise Fatal if Untreated)**
  - **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.



- **Recognising And Assessing Shock ?**
  - **Obvious Causes? (Eg. Bleeding, seeping burns)**
  - **Compensation? (Eg. Tachycardia)**
  - **Hypotension? (Despite Compensation)**
  - **Poor Tissue Perfusion?**
    - Peripheral Shutdown? (Eg. Cold, sweaty)
    - Renal Failure? (↓Urine Output)
    - Brain Hypoxia? (Confusion, ALOC, Coma)
    - Myocardial Ischaemia (Chest Pain, Heart Failure)

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 1^{o} Assessment, **IM/IV/SC Adrenaline**, +/- Steroids
- **Cardiogenic Shock:** **Inotropes**, Nitrates/Angioplasty/Reperfusion, Valvuloplasty, Transplant
- **Mechanical:**
  - **Tamponade:** **Pericardiocentesis**, Correct Cause (Trauma/Infection)
  - **Pneumothorax:** **Thoracocentesis** (Pleural Tap), Correct Cause (Trauma/Infection/Fluid Overload)
  - **PE:** **Thrombolysis (TPA/Alteplase), Thrombectomy**

FLUID RESUSCITATION PRINCIPLES – (See "Fluid Management [Surgical Context]" for more Info):

- **How Much???**
  - **1. Bolus (Vol. Of Estimated Acute Losses)**
  - **2. Maintenance ***(4,2,1 Rule)***:**
    - 4ml/kg/hr for 1^{st} 10kg
    - 2ml/kg/hr for 2^{nd} 10kg **(Ie. 60ml/hr for 1^{st} 20kg)**
    - 1ml/kg/hr for every kg thereafter. **(Ie. 100ml/hr for 1^{st} 60kg –Plus 1ml/kg/hr onwards)**
- **What happens to the Different IV Fluids?:**
  - **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.
  - **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.
  - **IV Dextrose → Actively taken into cells :. None Remains in Blood.**
    - :. NOT Suitable for Pressure Fluid Resuscitation. (Good for Hypoglycaemia & Post-Surgery)
- **Blood:**
  - **= The best fluid to replace blood loss**
  - But Saline/Hartmanns or Colloid are still ok.
  - BUT Blood has risks (immunogenic/infections/etc)

GLS – SHOCK CASES:

Case 1 - Bart:

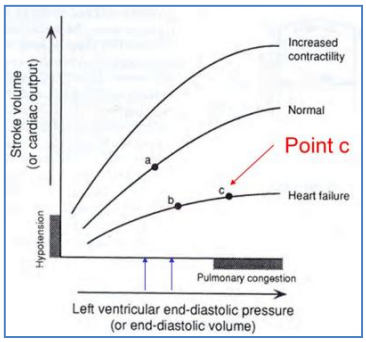
- He is pale and sweaty, has a distended abdomen and obvious bilateral femoral fractures. His pulse is 140 and his blood pressure is 75/40.

- **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 – (For the Bradycardia)

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:

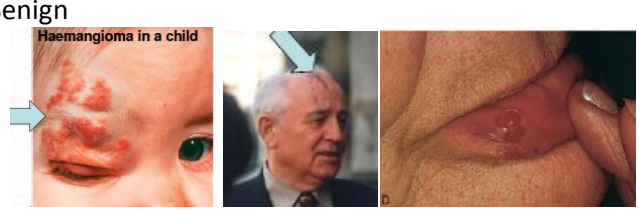
- Her dummy fell in dog poo. Now very sleepy. Her skin is a mottled grey colour. Pulse of 180 and blood pressure is 60/40. Angry inflamed area on her face which has pus in the middle of it.

- **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 Lactica Acid by Anaerobic Metabolic Pathways.

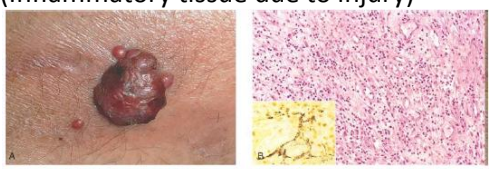
CVS Pathology:
Tumours of Vessels

Tumours of Vessels (Blood & Lymphatics): p520

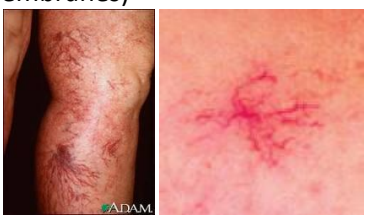
- **HAEMANGIOMA:**
  - **= Closely Packed Aggregates of Sub-Cutaneous Capillaries filled with Blood.**
  - Congenital & Benign



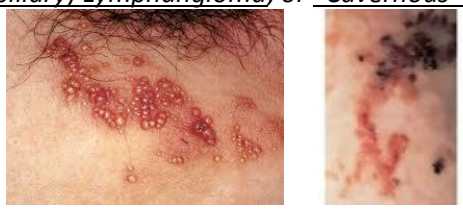
- **PYOGENIC GRANULOMA:**
  - **= A Granulating Haemangioma**
  - Rapidly Growing Cutaneous/Mucosal Red Nodule (Bleeds Easily & Often Ulcerated.)
  - Consist of Capillaries, Granulation Tissue & Bacteria
  - Often follow Trauma (Inflammatory tissue due to injury)


Moist growth over a wound - Inflammatory tissue, BV & bacteria.

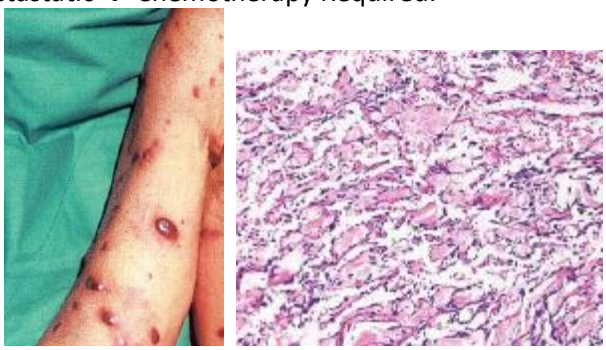
- **TELANGIECTASIA:**
  - **= A Tiny AV-Malformation**
  - *Blanching,* Spider-Like, Red Lesions.
  - Composed of Capillaries, Venules & Arterioles.
  - (Usually in Skin/Mucous Membranes)



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



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



CVS Pathology:
Valvular Heart Disease & Murmurs

VALVULAR HEART DISEASE:

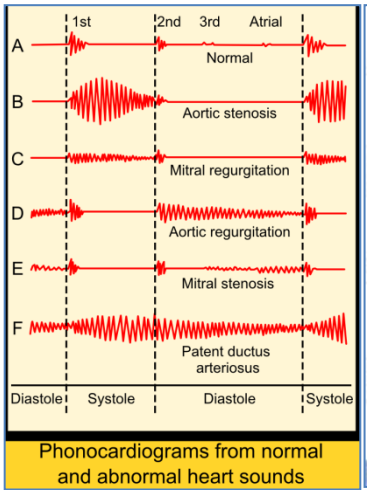
- **4 Most Common Murmurs & Their Causes:**

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 Marfan's Syndrome Syphilitic Aortitis Rheumatoid Arthritis Ankylosing Spondylitis

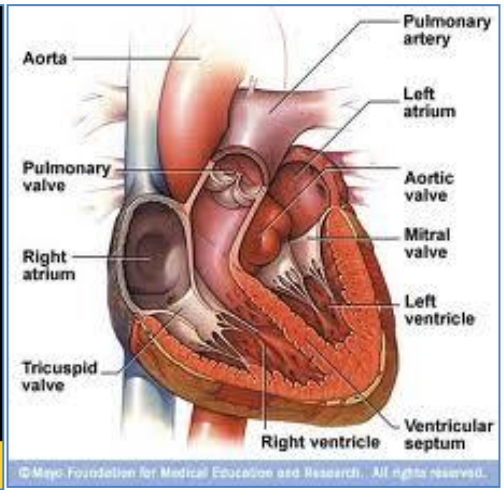
- (Red = Most Common)

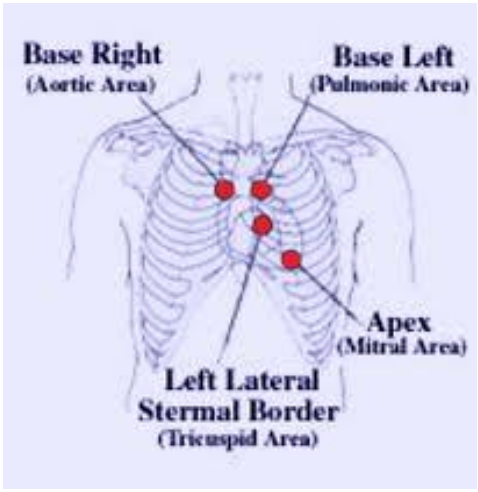
- **Other Less Common Murmurs:**

	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



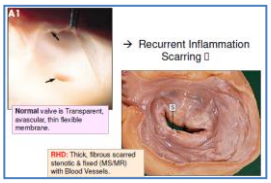
Phonocardiograms from normal and abnormal heart sounds

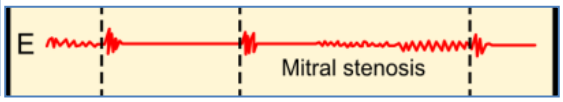




MITRAL STENOSIS:

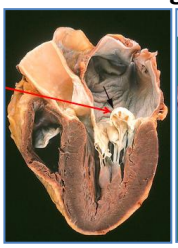
- **Aeitiology:**
  - 99% Rheumatic Heart Disease
- **Pathogenesis:**
  - Recurrent Acute Rheumatic Fever → Autoimmune Mitral Valve Fibrosis → Stenosis
- **Clinical Features**
  - **Symptoms:**
    - **CCF** – (Exertional Dyspnoea/Orthopnoea/PND/Wet cough (Pulmonary Oedema))
  - **Signs:**
    - **Low-Volume Pulse**
    - **Mid-Diastolic Rumbling Murmur** – (Loudest @ Apex on Expiration & → Axilla).
    - **"Mitral Facies" –** (Malar/Butterfly Rash over Cheeks & Nose)
    - **Pulm.Congestion & CCF** – (RV-Hypertrophy, Exertional Dyspnoea)
    - **If Cor-Pulmonale (RV-Failure)** → (↑JVP, Pulsatile Liver, Ascites, Peripheral Oedema)
- **Investigations:**
  - **ECHO –** (Diagnostic)
  - **ECG –** (May have A.Fib, LA-Hypertrophy, RVH)
  - **CXR –** (LA-Hypertrophy, Pulmonary Congestion)
- **Management:**
  - **Medical –** Treat A.Fib, Warfarin, CCF Triples (ACEi + B-Blocker + Diuretics)
  - **Surgical – Mitral Valvuloplasty (Repair) or Replacement**

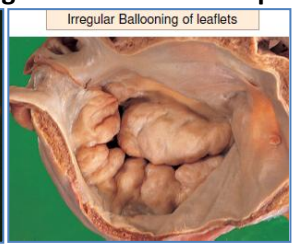


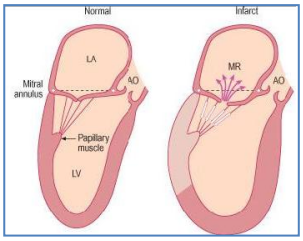


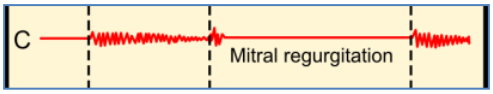
MITRAL INCOMPETENCE/REGURGITATION:

- **Aeitiology:**
  - Myxomatous Degeneration, Rheumatic Fever, Infective Endocarditis or **Ischaemia**
- **Pathogenesis:**
  - Myxomatous Degeneration – (Pathological weakening of valve connective tissue)
  - Rheumatic Fever → Autoimmune Mitral Valve Fibrosis → Stenosis **& Regurg**
  - Infective Endocarditis → Vegetations on Valve Edges → Improper Closure → Regurg
  - **Ischaemia** – (Post MI Papillary Rupture → Ballooning of Mitral Valve during Systole)
- **Clinical Features & Complications:**
  - **Symptoms:**
    - Exertional Dyspnoea
    - Wet Cough (Pulmonary Oedema)
  - **Signs:**
    - High-Pitched Pansystolic Murmur – (Loudest @ Apex on Expiration → Axilla)
    - L-Parasternal Heave (L-Atrial Hypertrophy)
- **Investigations:**
  - **ECHO –** (Diagnostic)
  - **ECG –** (LAH, LVH)
  - **CXR –** (LAH, LVH, Pulmonary Congestion)
- **Management:**
  - **Medical – CCF Triples (ACEi + B-Blocker + Diuretic)**
  - **Surgical – Mitral Valvuloplasty (Repair) or Replacement**



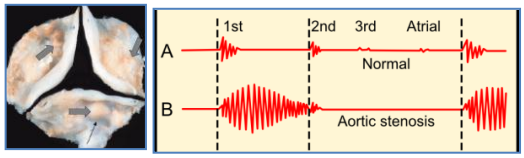






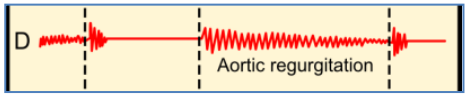
AORTIC STENOSIS:

- **Aeitiology:**
  - Age-Related Calcification (Wear & Tear)
  - (Also Rheumatic Heart Disease in 10% of cases)
- **Pathogenesis:**
  - Wear & Tear Degeneration + Calcification.
- **Clinical Features:**
  - **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)**
  - **Signs:**
    - **LV-Hypertrophy** → Displaced Apex Beat.
    - **Loud *Ejection Systolic Murmur* +/- Thrill** – (Loudest @ 2nd ICS R-Sternal Border)
      - Worse on Expiration
      - Radiates to Carotids
    - **Congestive Heart Failure** → Dyspnoea + Pulmonary Oedema
- **Investigations:**
  - **ECHO –** (Diagnostic)
  - **ECG –** (LV-Strain & LVH)
  - **CXR –** (Calcified Valve, LVH, CCF/Pulmonary Oedema)
- **Management:**
  - **If Symptomatic → Requires Cardiac Surgery**:
    - Aortic Valve Replacement.
    - Or Balloon Valvuloplasty



AORTIC INCOMPETENCE/REGURGITATION:

- **Aeitiology:**
  - **Age/Hypertension/"Syphilitic Aortitis"** → Aortic Root Dilation
- **Pathogenesis:**
  - Dilation of Aortic Root → Valve Leaflets Misalignment → Aortic Regurg
- **Clinical Features & Complications:**
  - **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)**
  - **Signs:**
    - "*Waterhammer Pulse*" – (Bounding and Rapidly Collapsing)
    - Displaced Apex Beat (Due to LV-Hypertrophy)
    - Diastolic Decrescendo Murmur – (Loudest @ R.2nd ICS on Expiration)
    - Tachycardia (Compensation for ↓CO)
- **Investigations:**
  - **ECHO –** (Diagnostic)
  - **ECG –** (LAH + LVH)
  - **CXR –** (LAH + LVH, CCF/Pulmonary Oedema)
- **Management:**
  - **Medicine:** Vasodilators + CCF Triple Therapy (ACEi + B-Blocker + Diuretic)
  - **Surgery:** Aortic Valve Replacement



TRICUSPID VALVE DISEASE

Etiology
- ❑ TS: rheumatic, congenital, carcinoid syndrome, fibroelastosis
- ❑ TR: RV dilatation (commonest cause), IE (iv drug users), rheumatic, Ebstein anomaly, AV cushion defects, carcinoid, tricuspid prolapse, trauma

Symptoms
- ❑ right heart failure
  - fatigue
  - pedal edema, abdominal pain (liver congestion), ascites
  - dyspnea (may reflect right heart forward failure)

Signs
- ❑ carotid pulse: irregular if A fib and low volume
- ❑ JVP
  - increased JVP
  - prominent "a" waves in TS
  - large "v" waves in TR ("cv" waves)
  - positive HJR and Kussmaul's sign (rise in JVP with inspiration)
- ❑ precordial palpation for left parasternal lift (RV) in TR
- ❑ precordial auscultation
  - note: all right sided sounds are louder with inspiration, except a pulmonary ejection click
  - TS: diastolic rumble in 4th left intercostal space (LICS)
  - TR: holosystolic murmur along LLSB (Carvallo's murmur); may behave like an ejection murmur
  - RV S3 along LLSB (with inspiration)
- ❑ abdominal examination
  - hepatomegaly (congestion) with systolic pulsations from TR
  - edema, ascites: 2° to fluid retention

Investigations
- ❑ 12 lead ECG
  - TS: RAE
  - TR: RAE, RVH, A fib
- ❑ chest x-ray
  - TS: dilatation of RA without pulmonary artery enlargement
  - TR: RA + RV enlargement
- ❑ ECHO
  - diagnostic

Management
- ❑ supportive
  - diuretics, preload reduction
  - TV surgery usually determined by need for other interventions (e.g. MVR of r associated MS)

PULMONARY VALVE DISEASE
- ❑ much less commonly involved

Etiology
- ❑ pulmonary stenosis (PS): usually congenital; rheumatic uncommon; carcinoid
- ❑ pumonary regurgitation (PR): secondary to dilatation of valve ring
  - pulmonary HTN (MS - most common, COPD, recurrent PE)
  - rheumatic, IE

Symptoms
- ❑ chest pain, syncope, dyspnea, leg edema (RV failure and CHF)

Signs
- ❑ PS
  - systolic murmur - maximum at 2nd LICS
  - pulmonary ejection click; normal/loud/soft P2; right sided S_4
- ❑ PR
  - early diastolic murmur at base
  - Graham Steel (diastolic) murmur at 2nd and 3rd LICS increasing with inspiration; no peripheral stigmata of AR

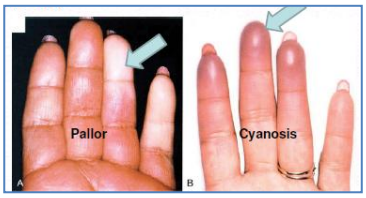
Investigations
- ❑ 12 lead ECG
  - RVH
- ❑ chest x-ray
  - prominent pulmonary arteries if pulmonary HTN
  - enlarged RV
- ❑ ECHO
  - diagnostic - RVH, RV dilatation; PS or PR by Doppler

Management
- ❑ IE prophylaxis
- ❑ PR
  - rarely requires treatment (well tolerated if systemic vascular resistance is normal)
  - valve replacement may be required
- ❑ PS
  - balloon valvuloplasty, depending on severity

CVS Pathology:
Vasculitides

(NB: Raynaud's Phenomenon – A common feature of Blood Vessel Disorders):

- **Aetiology:** Exaggerated Vasoconstriction of Digital Arteries (Cold/Emotional Trigger)
- **Clinical Features:** → Paroxysmal Pallor/Cyanosis of Digits (hands/feet)
    - Usually benign



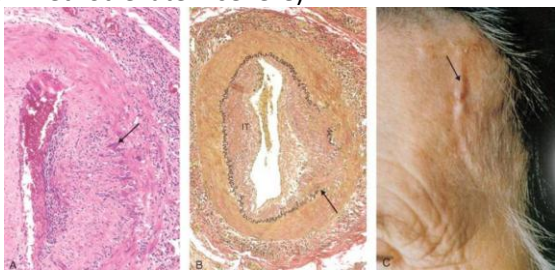
Vasculitis (General Vessel Inflammation):

- (NB: There are ≈20 different forms of Vasculitis – Only focus on those Highlighted)
- **2 Aetiologes:**
    - **Immune...OR...Infective**
    - **(NB: MUST distinguish between aetiologies since treatments Contradict each other)**
- **Signs/Symptoms:**
    - **Generals** – *Fever, Malaise, Myalgias & Arthralgias.*
    - **Specifics** - Depend on Vessels Affected.

VASCULITIS IN LARGE ARTERIES:

**** GIANT CELL (*TEMPORAL*) ARTERITIS:**

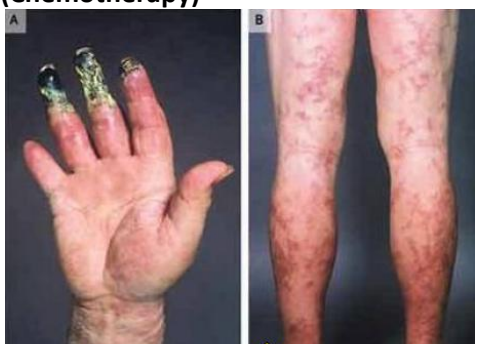
- **Aetiology:**
    - Chronic, Autoimmune Disease of *TEMPORAL and OPHTHALMIC Arteries*
- **Pathogenesis:**
    - Autoimmune Inflammation of Temporal & Ophthalmic Arteries
- **Clinical Features:**
    - (Typically in >50yo's)
    - **Temporal Arteritis Triad:**
        - **1. Headaches**
        - **2. Jaw Claudication**
        - **3. Tender Temples**
    - **+ Fever, Fatigue, Weight Loss**
    - **+/- Sudden Painless Blindness (**Transient or Permanent**)**
    - **Sometimes "*Polymyalgia Rheumatica*" – (**Neck, Shoulder & Hip Pain/Stiffness**)**
- **Complications:**
    - ****RED FLAG – If Untreated, can → BLINDNESS**
    - **Aortic Arch Syndrome → Aortic Aneurysm +/- Rupture.**
- **Investigation:**
    - **↑ESR + ↑CRP → **Temporal Artery Biopsy**
        - **(NB: Biopsy = Definitive Diagnosis)**
    - **+/- Cranial Angiography**
- **Treatment:**
    - **High-Dose Prednisone**
    - (+/- Azathioprine or Methotrexate if severe)



VASCULITIS IN MEDIUM ARTERIES (MUSCULAR ART):

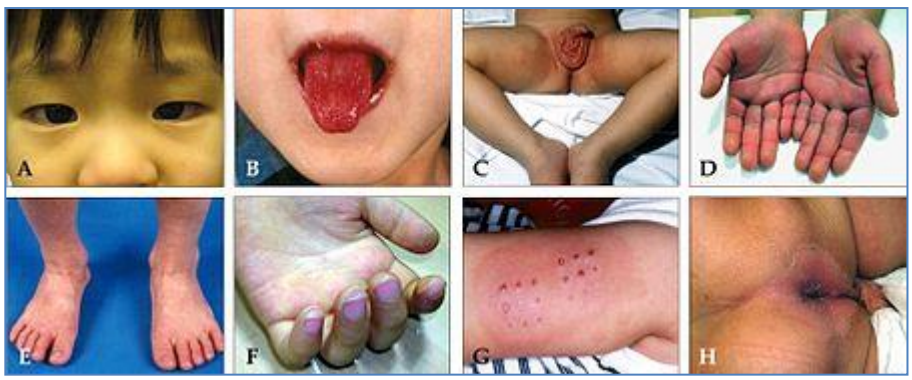
POLYARTERITIS NODOSA:

- **Aetiology:**
  - *SYSTEMIC* Autoimmune Inflammation of Medium Arteries.
- **Pathogenesis:**
  - Immune Complex Deposition in Arteries *(Particularly Renal Arteries)*
    - → Necrosis of Vessels → Rupture/Thrombosis/Aneurysms → Infarct/Ischaemia.
- **Clinical Features:**
  - **General Symptoms:**
    - **Fever
    - Rash
    - Malaise
    - Weight Loss
  - **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:**
  - Rupture/Thrombosis/Aneurysms → Localised Infarct/Ischaemia
- **Investigation:**
  - **↑ESR + ↑CRP**
  - **Vascular Biopsy**
  - **Or Angiogram**
- **Treatment:**
  - **Prednisone**
  - **+ Cyclophosphamide (Chemotherapy)**



KAWASAKIS DISEASE ("Mucocutaneous Lymph Node Syndrome"):

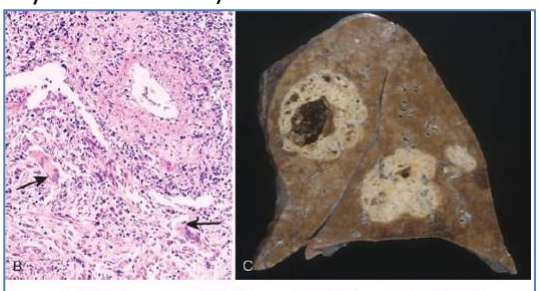
- See Paediatrics



VASCULITIS IN SMALL ARTERIES (CAPS & ARTERIOLES):

WEGENER'S GRANULOMATOSIS:

- **Aetiology:**
  - Autoimmune (Probably Hypersensitivity to Inhaled Agents)
- **Pathogenesis:**
  - Autoimmune Hypersensitivity Reaction to Inhaled Agent → Necrotizing Lung Granulomas (~TB)
    - (Also Renal → ***Glomerulonephritis***).
- **Morphology:**
  - **Granulomatous Inflammation in Lungs & URT**
    - URT Mucosal Granulomatous Lesions
    - Granulomas (which may cavitate) in the Lungs
  - **Necrotizing Vasculitis around Small Vessels (Particularly Renal/Glomerular).**
    - Focal (early) or Diffuse (late) Glomerular Necrosis → ***Glomerulonephritis***
- **Clinical Features:**
  - **Systemic:**
    - Fever, Malaise, Weakness, Myalgia, Rash.
  - **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
  - **Renal:**
    - **Glomerulonephritis** – (Nephrotic +/- Nephritic Syndrome)
- **Investigations:**
  - **American College of Rheumatology Criteria – (>2 of):**
    - URTI Inflammation – (Nasal/Oral)
    - CXR – (Nodules/Cavitations)
    - Urinalysis – (Protein/Casts)
    - Biopsy – (Granulomatous Inflammation)
  - **+ ANCA**
  - **↑ESR & ↑CRP**
- **Treatment:**
  - **Prednisone (+/- *Cyclophosphamide*)**
  - **+ High-Dose *Methotrexate***
  - (NB: 80% 1yr Mortality if Not Treated)



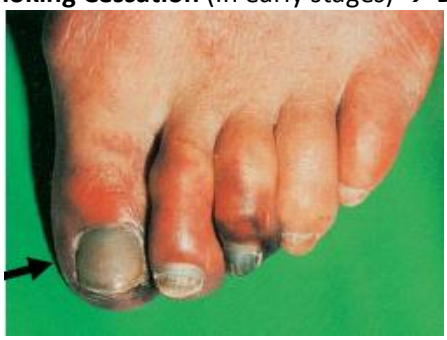
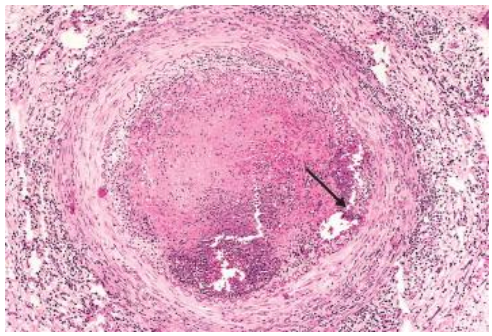
Granulomatous inflammation around small vessels with epitheloid cells and giant cells. Lung specimen showing cavitating grey white lesions. (similar to TB)

CHURG-STRAUSS SYNDROME:

- **Aetiology:**
  - Unknown
- **Pathogenesis:**
  - Granulomatous Inflammation of Small/Medium-Sized Vessels.
- **Clinical Features:**
  - **Churg-Strauss Triad:**
    - Systemic Vasculitis
    - Asthma
    - Allergic Rhinitis
  - **Others – (**Angina, Myocarditis, Neuropathy**)**
- **Investigation:**
  - **+ANCA**
  - **↑ESR**
  - **FBC – (**Eosinophilia**)**
- **Management:**
  - ***Prednisone +/- Cyclophosphamide***
  - **Then *Methotrexate***

BUERGER'S DISEASE ("THROMBOANGIITIS OBLITERANS"):

- **Aetiology:**
  - **Cigarette Smoking → Direct Endothelial Toxicity
- **Pathogenesis:**
  - **Cigarette Smoking → Direct Endothelial Toxicity → Vasculitis → Ischaemia → Gangrene
- **Clinical Features:**
  - ***Occurs in Chronic HEAVY Smokers
  - **Digital Infarcts - Gangrene**
  - **Distal Limb Ischaemia** – (Claudication, Arterial Ulcers, Gangrene)
- **Treatment:**
  - **Smoking Cessation** (In early stages) → Dramatic Relief.

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

Continue Reading For Bonus Supplementary Study Materials…

C Cardiology and Cardiac Surgery

David Armstrong, Mena Gewarges, and **Sagar Rohailla,** chapter editors
Hart Stadnick and **Kevin Yau,** associate editors
Alex Cressman, EBM editor
Dr. Chi-Ming Chow, Dr. Michael McDonald, and **Dr. Anna Woo,** staff editors

Acronyms

AAA	abdominal aortic aneurysm
ABI	ankle-brachial index
ACEI	angiotensin converting enzyme inhibitor
ACS	acute coronary syndrome
AFib	atrial fibrillation
AR	aortic regurgitation
ARB	angiotensin receptor blocker
ARDS	acute respiratory distress syndrome
AS	aortic stenosis
ASA	acetylsalicylic acid (Aspirin®)
ASD	atrial septal defect
AV	atrioventricular
AVM	arteriovenous malformation
AVNRT	atrioventricular nodal re-entrant tachycardia
AVRT	atrioventricular re-entrant tachycardia
BBB	bundle brunch block
BNP	brain natriuretic peptide
BP	blood pressure
BiVAD	biventricular assist device
CABG	coronary artery bypass graft
CAD	coronary artery disease
CCB	calcium channel blocker
CHF	congestive heart failure
CI	cardiac index
CO	cardiac output
COPD	chronic obstructive pulmonary disease
CTA	CT angiography
CVD	cerebrovascular disease
CXR	chest x-ray
DCM	dilated cardiomyopathy
DM	diabetes mellitus
DOAC	direct oral anticoagulant
DVT	deep vein thrombosis
ECASA	enteric coated ASA
ECG	electrocardiogram
Echo	echocardiogram
EPS	electrophysiology studies
EtOH	ethanol/alcohol
GERD	gastroesophageal reflux disease
HCM	hypertrophic cardiomyopathy
HFPEF	heart failure with preserved ejection fraction
HFREF	heart failure with reduced ejection fraction
HTN	hypertension
HR	heart rate
ICD	implantable cardiac defibrillator
IE	infective endocarditis
JVP	jugular venous pressure
LA	left atrium
LAE	left atrial enlargement
LBB	left bundle branch
LBBB	left bundle branch block
LICS	left intercostal space
LLSB	left lower sternal border
LMWH	low molecular weight heparin
LV	left ventricle
LVAD	left ventricular assist device
LVEF	left ventricular ejection fraction
LVH	left ventricular hypertrophy
MAT	multifocal atrial tachycardia
MI	myocardial infarction
MPI	myocardial perfusion imaging
MR	mitral regurgitation
MRA	MRI angiography
MS	mitral stenosis
NSR	normal sinus rhythm
NSTEMI	non-ST elevation myocardial infarction
OS	opening snap
PAC	premature atrial contraction
PCI	percutaneous coronary intervention
PCWP	pulmonary capillary wedge pressure
PDA	patent ductus arteriosus
PE	pulmonary embolism
PFO	patent foramen ovale
PIV	posterior-interventricular artery
PMI	point of maximal impulse
PND	paroxysmal nocturnal dyspnea
PUD	peptic ulcer disease
PVC	premature ventricular contraction
PVD	peripheral vascular disease
RA	right atrium
RAE	right atrial enlargement
RAO	right anterior oblique
RBB	right bundle branch
RBBB	right bundle branch block
RBW	routine blood work
RV	right ventricle
RVAD	right ventricular assist device
RVH	right ventricular hypertrophy
SA	sinoatrial
SCD	sudden cardiac death
SEM	systolic ejection murmur
SLE	systemic lupus erythematosis
STEMI	ST elevation myocardial infarction
SV	stroke volume
SVC	superior vena cava
SVR	systemic vascular resistance
SVT	supraventricular tachycardia
TAA	thoracic aortic aneurysm
TB	tuberculosis
TEE	transesophageal echocardiography
TIA	transient ischemic attack
TR	tricuspid regurgitation
TTE	transthoracic echocardiography
UA	unstable angina
VAD	ventricular assist device
VFib	ventricular fibrillation
VT	ventricular tachycardia
VTE	venous thromboembolism
WPW	Wolff-Parkinson-White

Basic Anatomy Review

Coronary Circulation

- conventional arterial supply to the heart arises from the right and left coronary arteries, which originate from the root of the aorta
  - right coronary artery (RCA)
    - acute marginal branches
    - atrioventricular (AV) nodal artery
    - posterior interventricular artery (PIV) = posterior descending artery (PD)
  - left main coronary artery (LCA): two major branches
    - left anterior descending artery (LAD)
      - septal branches
      - diagonal branches
    - left circumflex artery (LC)
      - obtuse marginal branches
- dominance of circulation
  - right-dominant circulation: PIV and at least one posterolateral branch arise from RCA (80%)
  - left-dominant circulation: PIV and at least one posterolateral branch arise from LC (15%)
  - balanced circulation: dual supply of posteroinferior LV from RCA and LC (5%)
- the sinoatrial (SA) node is supplied by the SA nodal artery, which may arise from the RCA (60%) or LCA (40%)
- most venous blood from the heart drains into the RA through the coronary sinus, although a small amount drains through Thebesian veins into all four chambers, contributing to the physiologic R-L shunt

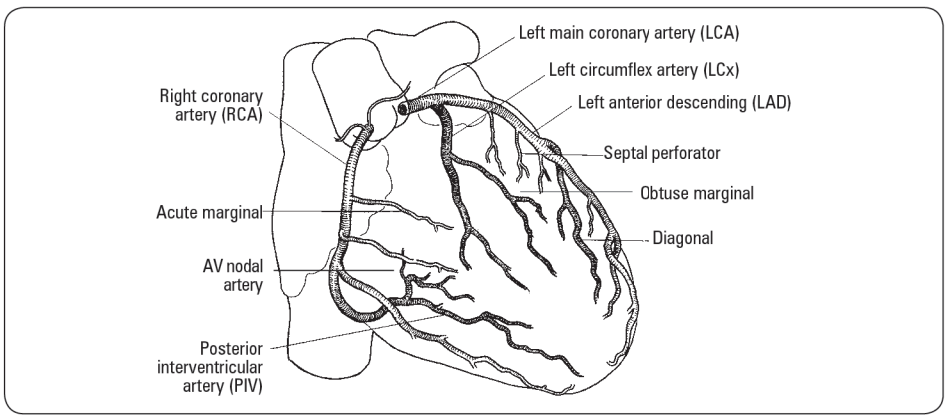


Figure 1. Anatomy of the coronary arteries (right anterior oblique projection)

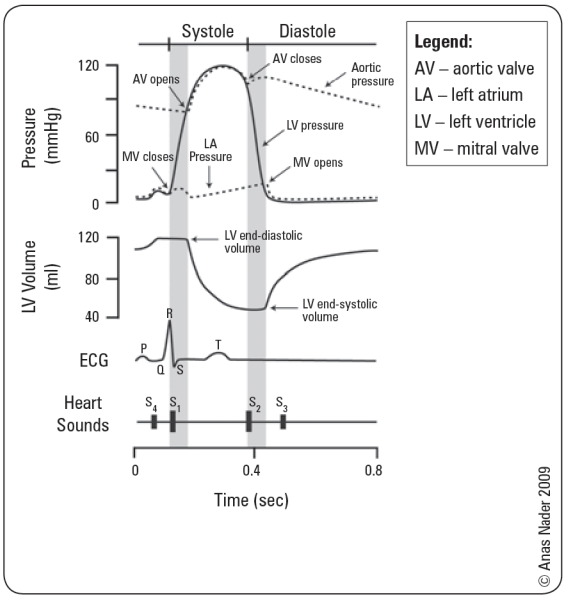


Figure 2a. Cardiac cycle
Grey shaded bars indicate isovolumic contraction (left) and isovolumic relaxation (right)

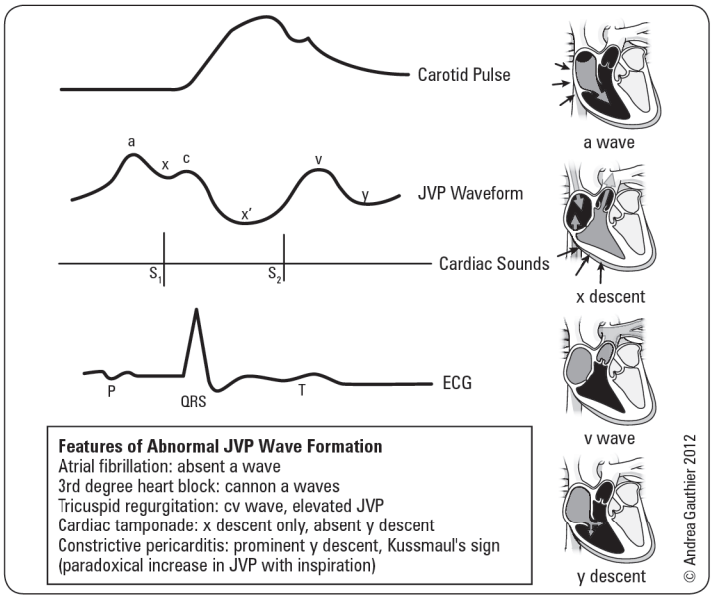


Figure 2b. JVP waveform

Cardiac Anatomy

- **layers of the heart**
  - endocardium
  - myocardium
  - epicardium
  - visceral pericardium
  - pericardial cavity
  - parietal pericardium
- **valves**
  - semilunar valves: no subvalvular apparatus present
    - aortic valve, 3 valve leaflets: separates LV and ascending aorta
    - pulmonic valve, 3 valve leaflets: separates RV and main pulmonary artery (PA)
  - atrioventricular valves: subvalvular apparatus present in the form of chordae tendinae and papillary muscles
    - tricuspid valve, 3 valve leaflets: separates RA and RV
    - mitral valve, 2 valve leaflets: separates LA and LV
- **conduction system**
  - SA node governs pacemaking control
  - anterior-, middle-, and posterior-internal nodal tracts carry impulses in the right atrium and along Bachmann's bundle in the left atrium
  - atrial impulses converge at the AV node
    - the AV node is the only conducting tract from the atria to the ventricles because of electrical isolation by the annulus fibrosis (except when accessory pathways are present)
  - the bundle of His bifurcates into left and right bundle branches (LBB and RBB)
  - LBB further splits into anterior and posterior fascicles
  - RBB and fascicles of LBB give off Purkinje fibres which conduct impulses into the ventricular myocardium

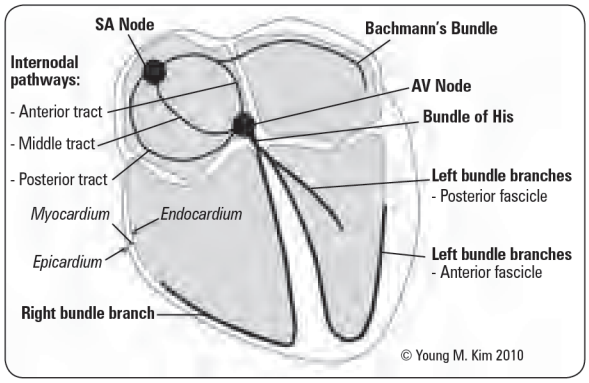


Figure 3. Conduction system of the heart

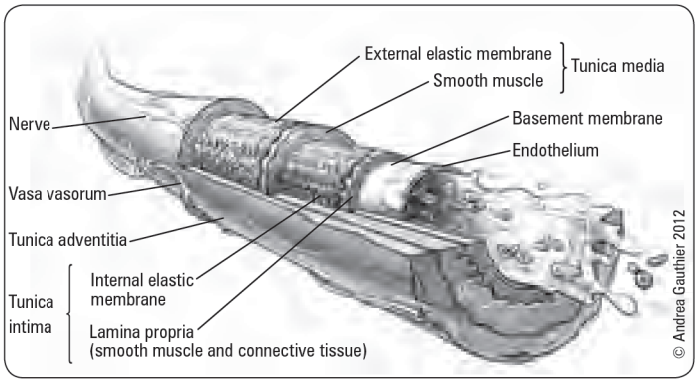


Figure 4. Blood vessel structure

- **cardiovascular innervation**
  - sympathetic nerves
    - innervate the SA node, AV node, ventricular myocardium and vasculature
    - SA node (β1) fibres increase pacemaking activity (chronotropy)
    - cardiac muscle (β1) fibres increase contractility (inotropy) to help increase cardiac output
    - stimulation of β1- and β2-receptors in the skeletal and coronary circulation causes vasodilatation
  - parasympathetic nerves
    - innervate the SA node, AV node, atrial myocardium but few vascular beds
    - basal vagal tone dominates the tonic sympathetic stimulation of the SA node and AV node resulting in slowing of pacemaker activity and conduction (i.e. reduced dromotropy – if only affecting AV node conduction)
    - parasympathetics have very little impact on total peripheral vascular resistance

Differential Diagnoses of Common Presentations

Note: **bold** text indicates most common, underlined text indicates life threatening condition

Chest Pain

- cardiac
  - MI/**angina**
  - myocarditis
  - pericarditis/Dressler's syndrome
  - cardiac tamponade
- pulmonary
  - **pneumonia**
  - PE
  - pneumothorax/hemothorax, tension pneumothorax
  - empyema
  - pulmonary neoplasm
  - bronchiectasis
  - TB
- gastrointestinal
  - esophageal: spasm, **GERD**, esophagitis, ulceration, achalasia, neoplasm, Mallory-Weiss syndrome, esophageal rupture
- gastrointestinal
  - PUD
  - gastritis
  - pancreatitis
  - biliary colic
- mediastinal
  - lymphoma
  - thymoma
- vascular
  - dissecting aortic aneurysm
  - aortic rupture
- surface structures
- costochondritis
- rib fracture
- skin (bruising, herpes zoster)
- breast
- anxiety/psychosomatic

Loss of Consciousness

- **hypovolemia**
- cardiac
  - structural or obstructive causes
    - ACS
    - AS
    - HCM
    - cardiac tamponade, constrictive pericarditis
  - arrhythmias (see *Arrhythmias*, C17)
- respiratory
  - massive pulmonary embolism
  - pulmonary hypertension
  - hypoxia
  - hypercapnia
- neurologic
  - stroke/TIA (esp. vertebrobasilar insufficiency)
  - migraine
  - seizure
- metabolic
  - **anemia**
  - **hypoglycemia**
- drugs
  - antihypertensives
  - antiarrhythmics
  - diuretics
- **vasovagal**
- autonomic dysfunction
  - diabetic neuropathy
- psychiatric
  - panic attack

Local Edema

- inflammation/infection
- venous or lymphatic obstruction
  - thrombophlebitis/deep vein thrombosis
  - venous insufficiency
  - chronic lymphangitis
  - lymphatic tumour infiltration
  - filariasis

Generalized Edema

- increased hydrostatic pressure/fluid overload
  - **heart failure**
  - pregnancy
  - drugs (e.g. CCBs)
  - **iatrogenic** (e.g. IV fluids)
- decreased oncotic pressure/ hypoalbuminemia
  - nephrotic syndrome
  - **liver cirrhosis**
  - malnutrition
- increased capillary permeability
  - severe sepsis
- hormonal
  - hypothyroidism
  - exogenous steroids
  - pregnancy
  - estrogens

Palpitations

- cardiac
  - **arrhythmias** (PAC, PVC, SVT, VT)
  - **valvular heart disease**
  - HCM
- endocrine
  - **thyrotoxicosis**
  - pheochromocytoma
  - hypoglycemia
- systemic
  - fever
  - **anemia**
- drugs
  - stimulants and anticholinergics
- psychiatric
  - panic attack

Dyspnea

- cardiovascular
  - acute MI
  - CHF/LV failure
  - aortic/mitral stenosis
  - aortic/mitral regurgitation
  - arrythmia
  - cardiac tamponade
  - constrictive pericarditis
  - left-sided obstructive lesions (e.g. left atrial myxoma)
  - elevated pulmonary venous pressure
- respiratory
  - airway disease
    - asthma
    - COPD exacerbation
    - upper airway obstruction (anaphylaxis, foreign body, mucus plugging)
  - parenchymal lung disease
    - ARDS
    - pneumonia
    - interstitial lung disease
  - pulmonary vascular disease
    - PE
    - pulmonary HTN
    - pulmonary vasculitis
  - pleural disease
    - pneumothorax
    - pleural effusion
- neuromuscular and chest wall disorders
  - C-spine injury
  - polymyositis, myasthenia gravis, Guillain-Barré syndrome
  - kyphoscoliosis
- anxiety/psychosomatic
- hematological/metabolic
  - anemia, acidosis, hypercapnia

Cardiac Diagnostic Tests

Electrocardiography Basics

Description

- a graphical representation (time versus amplitude of electrical vector projection) of the electrical activity of the heart

Indications

- detect myocardial injury, ischemia, and the presence of prior infarction
- palpitations, syncope, antiarrhythmic drug monitoring
- arrhythmia surveillance in patients with documented or potentially abnormal rhythms
- surveillance of non-sustained arrhythmias that can lead to prophylactic intervention

Contraindications

- no absolute contraindications
- patient refusal
- allergies (sensitivities to electrodelatex adhesive)

Risks

- no absolute risks

- on the ECG graph
  - the horizontal axis represents time (at usual paper speed 25 mm/s)
    - 1 mm (1 small square) = 40 msec
    - 5 mm (1 large square) = 200 msec
  - the vertical axis represents voltage (at usual standard gain setting 10 mm/mV)
    - 1 mm (1 small square) = 0.1 mV
    - 10 mm (2 large squares) = 1 mV
- leads
  - standard 12-lead ECG
    - limb leads: I, II, III, aVL, aVR, aVF
    - precordial leads: V1-V6 (V1-V2 septal, V3-V4 anterior, V5-V6 lateral)
  - additional leads
    - right-sided leads: V3R-V6R (useful in RV infarction and dextrocardia)
  - lateral = I, aVL, V5, V6; inferior = II, III, aVF; anterior = V1-V4

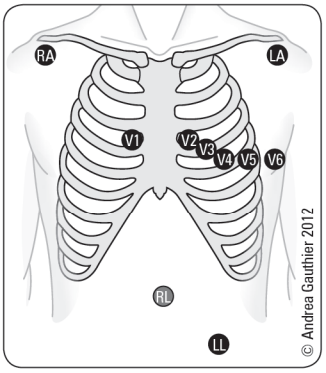


Figure 5. ECG lead placement

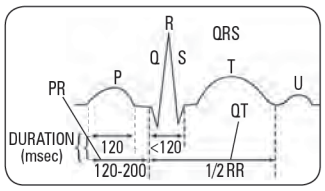


Figure 6. ECG waveforms and normal values

Approach to ECGs

Introduction

Historically, the electrocardiogram has been a tricky subject for medical students. For many years, the classical approach has been taught in medical schools, which has demystified the ECG. Below, we are presenting both the Classical Approach and the newer PQRSTU Approach to provide students with different ways to view the ECG. Despite methodological differences, the rigor and final result is the same. These two approaches should help you better understand the concepts of ECG interpretation and equip you with the necessary skills to interpret ECGs in exam scenarios and clinical practice

Rate Calculations
- Examples, practice

For more examples and practice visit www.ecgmadesimple.com

Classical Approach to ECGs

Classical Approach to ECG
- Rate
- Rhythm
- Axis
- Conduction abnormalities
- Hypertrophy/chamber enlargement
- Ischemia/infarction
- Miscellaneous ECG changes

RATE

- normal = 60-100 bpm (atrial rate: 150-250 bpm = paroxsymal tachycardia, 250-350 bpm = atrial flutter, >350 bpm = AFib)
- regular rhythm
  - to calculate the rate, divide 300 by number of large squares between 2 QRS complexes (there are 300 large squares in 1 min: 300 x 200 msec = 60 sec)
  - or remember 300-150-100-75-60-50-43 (rate falls in this sequence with the number of large squares between 2 QRS complexes)
- irregular rhythm
  - rate = 6 x number of R-R intervals in 10 s (the "rhythm strips" are 10 sec recordings)
  - types: wandering pacemaker, multifocal atrial tachycardia, AFib
- atrial escape = 60-80 bpm; junctional escape = 40-60 bpm; ventricular escape = 20-40 bpm

RHYTHM

- regular: R-R interval is the same across the tracing
- irregular: R-R interval varies across the tracing
- regularly irregular: repeating pattern of varying R-R intervals
- irregularly irregular: R-R intervals vary erratically
- normal sinus rhythm (NSR)
  - P wave precedes each QRS; QRS follows each P wave
  - P wave axis is normal (positive in 2 out of the 3 following leads I, aVF, II)
  - rate between 60-100 bpm

Differential Diagnosis for Left Axis Deviation (LAD)
- Left anterior hemiblock
- Inferior MI
- WPW
- RV pacing
- Normal variant
- Elevated diaphragm
- Lead misplacement
- Endocardial cushion defect

AXIS

- mean axis indicates the direction of the mean vector
- can be determined for any waveform (P, QRS, T)
- the standard ECG reported QRS axis usually refers to the mean axis of the frontal plane – it indicates the mean direction of ventricular depolarization forces
- QRS axis in the frontal plane
  - normal axis: -30° to 90° (i.e. positive QRS in leads I and II)
  - left axis deviation (LAD): axis <-30°
  - right axis deviation (RAD): axis >90°
- QRS axis in the horizontal plane is not routinely calculated – it is directed posteriorly and to the left
  - transition from negative to positive is usually in lead V3

Differential Diagnosis for Right Axis Deviation (RAD)
- RVH
- Left post hemiblock
- PE
- COPD
- Lateral MI
- WPW
- Dextrocardia
- Septal defects

Table 1. Conduction Abnormalities

Left Bundle Branch Block (LBBB)	Right Bundle Branch Block (RBBB)
Complete LBBB • QRS duration >120 msec • Broad notched R waves in leads V5, and V5, and usually I, aVL • Deep broad S waves in leads V1-2 • Secondary ST-T changes (-ve in leads with broad notched R waves, +ve in V1-2) are usually present • LBBB can mask ECG signs of MI	**Complete RBBB** • QRS duration >120 msec • Positive QRS in lead V1 (rSR' or occasionally broad R wave) • Broad S waves in leads I, V5-6 (>40 msec) • Usually secondary T wave inversion in leads V1-2 • Frontal axis determination using only the first 60 msec

Left Anterior Fascicular Block (LAFB) (Left Anterior Hemiblock)	Left Posterior Fascicular Block (LPFB) (Left Posterior Hemiblock)	Bifascicular Block
Left Axis Deviation (-30° to -90°) • Small q and prominent R in leads I and aVL • Small r and prominent S in leads II, III, and aVF	**Right Axis Deviation (110° to 180°)** • Small r and prominent S in leads I and aVL • Small q and prominent R in leads II, III, and aVF	RBBB pattern • Small q and prominent R • The first 60 msec (1.5 small squares) of the QRS shows the pattern of LAFB or LPFB • Bifascicular block refers to impaired conduction in two of the three fascicles, most commonly a RBBB and left anterior hemiblock; the appearance on an ECG meets the criteria for both types of blocks

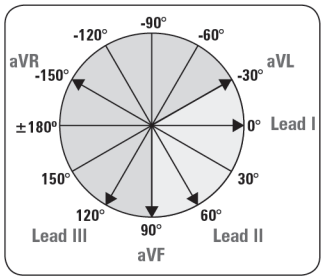


Figure 7. Axial reference system
Each lead contains a (+) area displayed by the bold arrows. Impulses traveling toward the positive region of the lead results in an upward deflection in that lead. Normal QRS axis is between -30° and +90°

Nonspecific Intraventricular Block

- QRS duration >120 msec
- absence of definitive criteria for LBBB or RBBB

Table 2. Hypertrophy/Chamber Enlargement

Left Ventricular Hypertrophy (LVH)	Right Ventricular Hypertrophy (RVH)
• S in V1 + R in V5 or V6 >35 mm above age 40, (>40 mm for age 31-40, >45 mm for age 21-30) • R in aVL >11 mm • R in I + S in III >25 mm • Additional criteria ▪ LV strain pattern (ST depression and T wave inversion in leads I, aVL, V4-V6) ▪ Left atrial enlargement • N.B. The more criteria present, the more likely LVH is present. If only one voltage criteria present, it is called minimal voltage criteria for LVH which could be a normal variant	• Right axis deviation • R/S ratio >1 or qR in lead V1 • RV strain pattern: ST segment depression and T wave inversion in leads V1-2
Left Atrial Enlargement (LAE)	**Right Atrial Enlargement (RAE)**
• Biphasic P wave with the negative terminal component of the P wave in lead V1 ≥1 mm wide and ≥1 mm deep • P wave >100 msec, could be notched in lead II ("P mitrale")	• P wave >2.5 mm in height in leads II, III, or aVF ("P pulmonale")

ISCHEMIA/INFARCTION

- look for the anatomic distribution of the following ECG abnormalities (see Table 3)
- ischemia
  - ST segment depression
  - T wave inversion (most commonly in V1-V6)
- injury
  - transmural (involving the epicardium)
    - ST elevation in the leads facing the area injured/infarcted
    - transient ST elevation may occur in patients with coronary artery spasm (e.g. Prinzmetal angina) which can be slight or prominent (>10 mm)
  - subendocardial
    - marked ST depression in the leads facing the affected area
    - may be accompanied by enzyme changes and other signs of MI
    - may also occur with angina

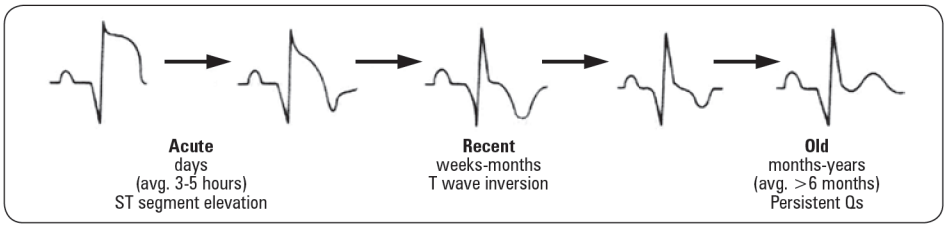


Figure 10. Typical ECG changes with infarction

- evolving infarction (ST elevation in contiguous leads in the same territory = acute MI)
- ST elevation: at least 1 mm in 2 adjacent limb leads or at least 1-2 mm in adjacent precordial leads in STEMI (signifies occlusion and transmural ischemic injury) vs. diffuse pattern in early pericarditis
- "typical" sequential changes of evolving MI
  1. hyperacute T waves (tall, symmetric T waves) in the leads facing the infarcted area, with or without ST elevation
  2. ST elevation (injury pattern) in the leads facing the infarcted area
     - usually in the first hours post infarct
     - in acute posterior infarction, there is ST depression in V1-V3 (reciprocal to ST elevation in the posterior leads, that are not recorded in the standard 12-lead ECG)
  3. significant Q waves: >40 msec or >1/3 of the total QRS and present in at least 2 consecutive leads in the same territory (hours to days post-infarct)
  4. inverted T waves (one day to weeks after infarction)
     - this classical sequence does not always occur
       – Q waves of infarction may appear in the very early stages, with or without ST changes
       – non-Q wave infarction: there may be only ST or T changes despite clinical evidence of infarction

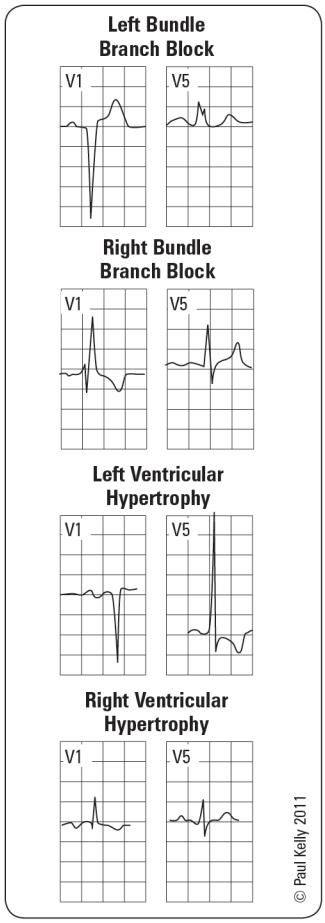


Figure 8. Complete LBBB, RBBB, LVH, and RVH (please see online examples for the full range of waveforms and the text for additional characteristics)

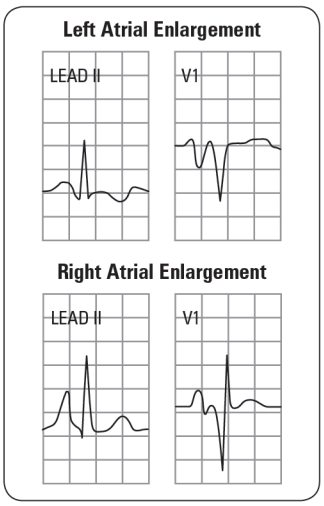


Figure 9. LAE, RAE (please see online examples and the text for characteristics)

- completed infarction
  - abnormal Q waves (Q waves may be present in leads III and aVL in normal individuals due to initial septal depolarization)
    - duration >40 msec (>30 msec in aVF for inferior infarction)
    - Q/QRS voltage ratio is >33%
    - present in at least 2 consecutive leads in the same territory
  - abnormal R waves (R/S ratio >1, duration >40 msec) in V1 and occasionally in V2 are found in posterior infarction (usually in association with signs of inferior and/or lateral infarction)

Table 3. Areas of Infarction/Ischemia (right dominant anatomy)

Vessel Usually Involved	Infarct Area (LAD and LC)	Leads (LAD and LC)
Left anterior descending (LAD)	Anteroseptal Anterior Anterolateral Extensive anterior	V1, V2 V3, V4 I, aVL, V3-6 I, aVL, V1-6
Right coronary artery (RCA)	Inferior Right ventricle Posterior MI (assoc. with inf. MI)	II, III, aVF V3R, V4R (right sided chest leads) V1, V2 (prominent R waves)
Left circumflex (LCX)	Lateral Isolated posterior MI	I, aVL, V5-6 V1, V2 (prominent R waves)

Low Voltage
- Definition: total QRS height in precordial leads <10 mm and/or limb leads <5 mm
- Differential diagnosis
  - Myocardial disease
    - Ischemia
    - Cardiomyopathy (usually infiltrative type), myocarditis
  - Pericardial effusion
  - Thick chest wall/barrel chest: COPD, obesity
  - Generalized edema
  - Hypothyroidism/myxedema
  - Inappropriate voltage standardization

MISCELLANEOUS ECG CHANGES

Electrolyte Disturbances

- hyperkalemia
  - mild to moderate (K^+ 5-7 mmol/L): tall peaked T waves
  - severe (K^+ >7 mmol/L): progressive changes whereby P waves flatten and disappear, QRS widens and may show bizarre patterns, axis shifts left or right, ST shift with tall T waves, eventually becomes a "sine wave" pattern
- hypokalemia
  - ST segment depression, prolonged QT interval, low T waves, prominent U waves (U>T)
  - enhances the toxic effects of digitalis
- hypercalcemia
  - shortened QT interval (more extracellular Ca^{2+} means shorter plateau in cardiac action potential)
- hypocalcemia
  - prolonged QT interval (less extracellular Ca^{2+} means longer plateau in cardiac action potential)

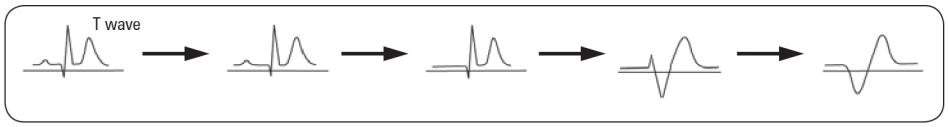


Figure 11. Hyperkalemia

Figure 12. Hypokalemia

Hypothermia

- sinus bradycardia
- when severe, prolonged QRS and QT intervals
- AFib with slow ventricular response and other atrial/ventricular dysrhythmias
- Osborne J waves: "hump-like" waves at the junction of the J point and the ST segment

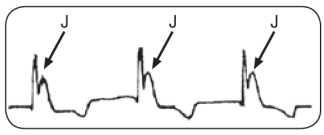


Figure 13. Osborne J waves of a hypothermic patient

Pericarditis

- early: diffuse ST segment elevation ± PR segment depression, upright T waves
- later: isoelectric ST segment, flat or inverted T waves
- ± tachycardia

Drug Effects

- digitalis
  - therapeutic levels may be associated with "digitalis effect"
    - ST downsloping or "scooping"
    - T wave depression or inversion
    - QT shortening ± U waves
    - slowing of ventricular rate in AFib

Pacemakers
- Demand pacemaker has discharge (narrow vertical spike on ECG strip) prior to widened QRS
- Atrial pacemaker has discharge prior to P wave
- Triggered pacemaker has discharge following the P wave but prior to the widened QRS
- Atrial and ventricular pacing have discharge before the P wave and widened QRS wave

- toxic levels associated with
  - arrhythmias: paroxysmal atrial tachycardia (PAT) with conduction block, severe bradycardia in AFib, accelerated junctional rhythms, PVCs, ventricular tachycardia (see *Arrhythmias*, C17)
  - "regularization" of ventricular rate in AFib due to a junctional rhythm and AV dissociation
- amiodarone, quinidine, phenothiazines, tricyclic antidepressants, antipsychotics, some antihistamines, some antibiotics: prolonged QT interval, U waves

Digitalis Side Effects
Palpitations, fatigue, visual changes (yellow vision), decreased appetite, hallucinations, confusion, and depression

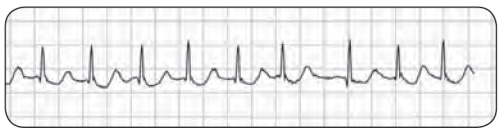


Figure 14. Atrial fibrillation, ST change due to digitalis ("digitalis effect")

Pulmonary Disorders

- *cor pulmonale* (often secondary to COPD)
  - low voltage, right axis deviation (RAD), poor R wave progression in precordial leads
  - RAE and RVH with strain
  - multifocal atrial tachycardia (MAT)
- massive pulmonary embolism (PE)
  - sinus tachycardia and AFib/atrial flutter are the most common arrhythmias
  - RAD, RVH with strain
  - most specific sign is S1Q3T3 (S in I, Q and inverted T wave in III) but rather uncommon

Alternative PQRSTU Approach to ECGs

Note: the information seen in this alternative approach – the PQRSTU Approach – is the same as the information in the Classical Approach; it is just organized in a slightly different way based on the anatomy of the ECG

PQRSTU approach to ECGs
P wave
P-R interval
QRS complex
ST segment
T wave
Q-T interval
U wave

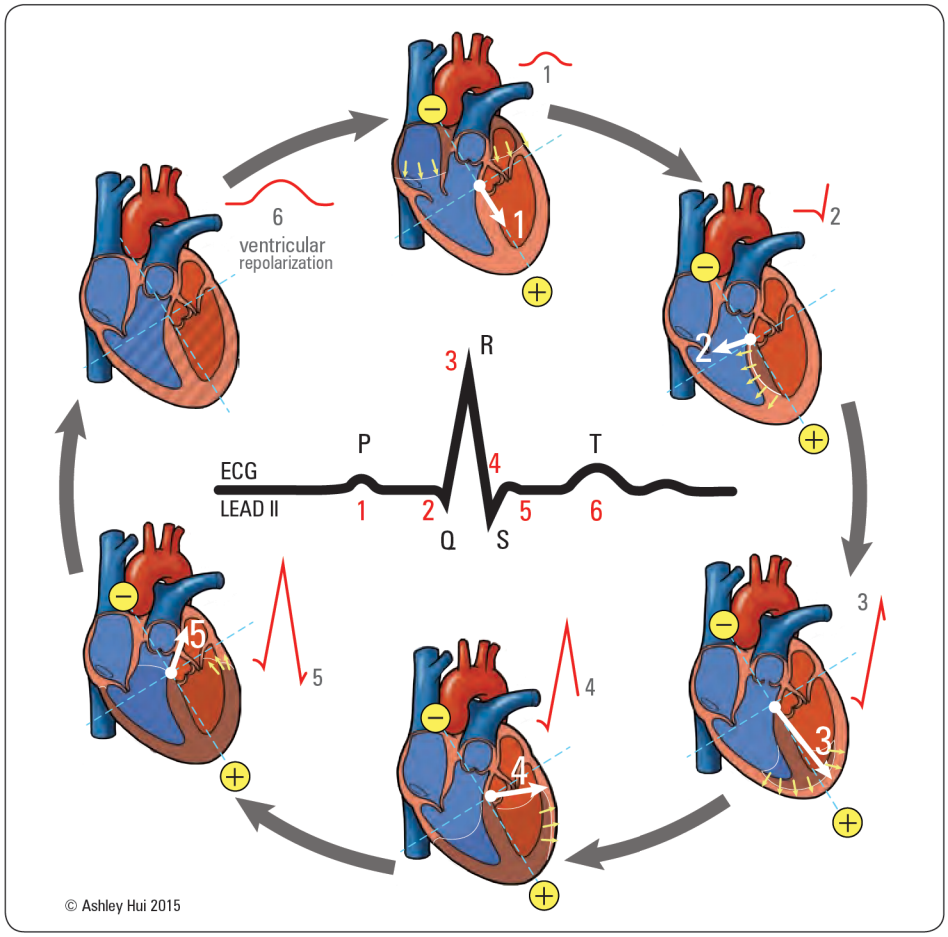


Figure 15. ECG correlations with heart activity

P WAVE

- the P wave provides a view into the atria of the heart and represents atrial contraction
- the best leads to view the P waves are II and V1
- assess the P waves for rate (based on the P-P interval relative to the R-R interval), rhythm (rounded, flutter/sawtooth, fibrillation) and axis
- lead II: the P wave should be rounded, <120 msec and <2.5 mm in height
- lead V1: the P wave is biphasic with a negative phase slightly greater than the positive phase

Common P Wave Pathology

- atrial flutter: sawtooth P wave (HINT: flip the ECG upside-down to see it better if unclear)
- atrial fibrillation: absent P wave, may have fibrillatory wave, irregular rhythm
- right atrial enlargement: tall P wave (>2.5 mm) in II or V1 (P pulmonale)
- left atrial enlargement: negative deflection >1 mm deep or >1 mm wide in V1, wide (>100 msec) notched P wave in II may be present (P mitrale)

P-R INTERVAL

- the P-R interval shows the delay between atrial and ventricular contraction that is mediated by the AV node; the magnitude of the delay is referred to as "dromotropy"
- positive dromotropy increases conduction velocity (e.g. epinephrine stimulation), negative dromotropy decreases velocity (e.g. vagal stimulation)
- P-R interval should be 120-200 msec
- long P-R interval (>200 msec)
  - heart block: first degree (fixed, prolonged P-R interval), second degree Mobitz I/Wenckebach (steadily prolonging to eventual dropped beat)
  - heart block
    - first degree: fixed, prolonged P-R interval
    - second degree Mobitz I/Wenckebach: steadily prolonging P-R interval to eventual dropped beat
    - second degree Mobitz II/Hay: fixed P-R interval with ratio of beat to dropped beat (e.g. for every 3 beats, there is one dropped beat [3:1])
    - third degree/complete: variable P-R intervals, P-P and R-R intervals individually constant but not in sync
  - atrial flutter
  - sinus bradycardia (normal to have long P-R if heart rate slow)
  - hypokalemia
  - trifascicular block
- short P-R interval (<120 msec)
  - pre-excitation syndrome (delta wave: upswooping of the P-R segment into the QRS complex indicating pre-excitation)
    - accessory pathways
    - WPW

Significant ECG Changes

- Look for ST changes starting at 60 msec from J point
- J point = the junction between the QRS complex and the ST segment
- ST elevation: at least 1 mm in 2 adjacent limb leads, or at least 1-2 mm in adjacent precordial leads
- ST depression: downsloping or horizontal
- Q Wave: pathological if Q wave ≥1 small square (≥40 msec) or >33% of the total QRS

QRS COMPLEX

- the QRS is where ventricular contraction is visualized
- rate: check the R-R interval to see if it matches the P-P interval
- amplitude: check for hypertrophy (see Table 2, C7)
- narrow width (<120 msec) QRS means that the His-Purkinje system is being used
- wide width (>120 msec) QRS means that the His-Purkinje system is being bypassed or is diseased
  - BBB, VT, ventricular hypertrophy, cardiomyopathy, WPW, ectopic ventricular beat, hyperkalemia, drugs (e.g. TCAs, antiarrhythmics)
- Q wave: the first downward deflection of the QRS complex
  - significant Q wave: >40 msec or >33% of total QRS amplitude; indicate myocardial necrosis (new or historical)
- R and S wave abnormalities typically show pathology in terms of BBB or intraventricular abnormalities

Insignificant Q Wave

- Septal depolarization by the left bundle
- Seen in leads I, II, III, aVL, V5, V6
- <40 msec

ST SEGMENT

- one of the more famous ECG personas mostly due to its role in detecting MI
- located between QRS complex and the T wave
  - corresponds to the completion of ventricular depolarization
- normally at the same level as "baseline/TP segment"
- ST elevation: at least 1 mm in 2 adjacent limb leads or at least 1-2 mm in adjacent precordial leads in STEMI (signifies occlusion and transmural ischemic injury) vs. diffuse pattern in early pericarditis
- ST depression: ischemia
  - ischemia which causes ST depression can result in myocardial damage (NSTEMI)
  - lateral ST depression (leads I, aVL, V5, V6) may actually indicate a STEMI in the right heart

T WAVE

- this is the repolarization phase of the ventricles (repolarization of the atria are obscured by the QRS complex)
- typically positive (except in aVR and V1) on ECG but normal isolated negative T waves may be present (esp. in V1 and V2)
- pathology when T wave variation occur in consecutive leads
  - inversion: BBB, ischemia, hypertrophy, drugs (e.g. digitalis), pulmonary embolism (lead III as part of S1Q3T3 sign)
  - elevation: infarction (STEMI, Prinzmetal, hyperacute), hyperkalemia (wider, peaked)
  - flattened: hypokalemia, pericarditis, drugs (e.g. digitalis), pericardial effusion
  - variations: T wave alterans; beat-to-beat variations due to PVC overlap (R on T phenomenon which may precipitate VT or VFib)
- appropriate T wave discordance: in BBB, T wave deflection should be opposite to that of the terminal QRS deflection (i.e. T wave negative if ends with R or R'; positive if ends with S)
  - inappropriate T wave concordance suggests ischemia or infarction

Q-T INTERVAL

- this represents the duration of ventricular depolarization and repolarization and is often difficult to interpret
- corrected QT (QTc) is often used instead in practice to correct for the repolarization duration; $QTc = QT \div \sqrt{RR}$
- normal QTc is 360-450 msec for males and 360-460 for females
  - increased (>450 msec for males and >460 for females): risk of Torsades de Pointes (a lethal tachyarrhythmia)
    - genetic Long QT Syndrome (often a channelopathy)
    - drugs: antibiotics, SSRIs, antipsychotics, antiarrhythmics
    - electrolytes: low Ca^{2+}, low Mg^{2+}, low K^+
    - others: hypothyroidism, hypothermia, cardiomyopathy
  - decreased (<360 msec): risk of VFib
    - electrolytes: high Ca^{++}
    - drugs: digoxin
    - others: hyperthyroidism

U WAVE

- origin unclear but may be repolarization of Purkinje fibres or delayed/prolonged repolarization of the myocardium
- more visible at slower heart rates
- deflection follows T wave with <25% of the amplitude
- variations from norm could indicate pathologic conditions:
  - prominent (>25% of T wave): electrolyte (low K^+), drugs (digoxin, antiarrhythmics)
  - inverted (from T wave): ischemia, volume overload

Differential Diagnosis of ST Segment Changes

ST Elevation I HELP A PAL
Ischemia with reciprocal changes
Hypothermia (Osborne waves)
Early repolarization (normal variant, need old ECGs to confirm)
LBBB
Post-MI
Acute STEMI
Prinzmetal's (Vasospastic) angina
Acute pericarditis (diffuse changes)
Left/right ventricular aneurysm

ST Depression WAR SHIP
WPW syndrome
Acute NSTEMI
RBBB/LBBB
STEMI with reciprocal changes
Hypertrophy (LVH or RVH) with strain
Ischemia
Post-MI

Cardiac Biomarkers

- provide diagnostic and prognostic information in acute coronary syndromes and in heart failure

Table 4. Cardiac Enzymes

Enzyme	Peak	Duration Elevated	DDx of Elevation
Troponin I, Troponin T	1-2 d	Up to 2 wk	MI, CHF, AFib, acute PE, myocarditis, chronic renal insufficiency, sepsis, hypovolemia
CK-MB	1 d	3 d	MI, myocarditis, pericarditis, muscular dystrophy, cardiac defibrillation, chronic renal insufficiency, etc.

- check troponin I at presentation and 8 h later ± creatine kinase-MB (CK-MB; depends on local laboratory protocol)
- new CK-MB elevation can be used to diagnose re-infarction
- other biomarkers of cardiac disease
  - AST and LDH also increased in MI (low specificity)
  - BNP and NT-proBNP: secreted by ventricles in response to increased end-diastolic pressure and volume
    - DDx of elevated BNP: CHF, AFib, PE, COPD exacerbation, pulmonary HTN

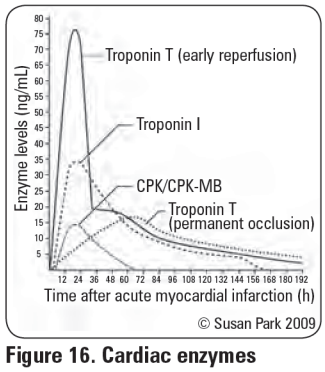


Figure 16. Cardiac enzymes

Ambulatory ECG

- **description**
  - extended ambulatory ECG of 24 or 48 hours or 14 or 30 days duration
  - provides a view of only two or three leads of electrocardiographic data over an extended period of time
  - permits evaluation of changing dynamic cardiac electrical phenomena that are often transient and of brief duration
  - ***continuous loop:*** a small, lightweight, battery operated recorder that records two or three channels of electrocardiographic data
    - patient activated event markers
    - minimum of 24-48 h
  - ***implantable device:*** subcutaneous monitoring device for the detection of cardiac arrhythmias
    - typically implanted in the left pectoral region and stores events when the device is activated automatically according to programmed criteria or manually with magnet application
    - can be used for months to years
- **indications**
  - evaluation of cardiac rhythm abnormalities
  - has also been used for assessing pacemaker and implantable cardioverter-defibrillator function, evidence of myocardial ischemia, late potentials, and heart rate variability
- **contraindications**
  - no absolute contraindications
  - patient refusal
  - allergies (sensitivities to latex adhesive)
- **risks:** no absolute risks

Use of B-Type Natriuretic Peptide in the Evaluation and Management of Acute Dyspnea (BASEL)
NEJM 2004;350;647-54
Study: Prospective, RCT.
Population: 452 patients (mean age 71 yr 58% male) with acute dyspnea; patients with severe renal disease or cardiogenic shock were excluded.
Intervention: Assessment including measurement of B-type natriuretic peptide or standard assessment.
Outcome: Time to discharge and total cost of treatment.
Results: Median time to discharge was significantly shorter in the intervention group when compared with the control group (8.0 vs. 11.0 d, p=0.001). Total cost was also significantly lower in the intervention group ($5410 vs. $7264, p=0.006). In addition, the measurement of B-type natriuretic peptide significantly reduced the need for admission to hospital and intensive care. The 30-d mortality rates were similar (10% vs. 12%, p=0.45).
Conclusions: In patients with acute dyspnea, measurement of B-type natriuretic peptide improves clinical outcomes (need for hospitalization or intensive care) and reduces time to discharge and total cost of treatment.

Echocardiography

Transthoracic Echocardiography (TTE)

- **description:** ultrasound beams are directed across the chest wall to obtain images of the heart
- **indications**
  - evaluation of LVEF, wall motion abnormalities, myocardial ischemia and complications of MI
  - evaluation of chamber size, wall thickness, valve morphology, proximal great vessel morphology, pericardial effusion
  - evaluation of unexplained hypotension, murmurs, syncope, congenital heart disease
- **contraindications**
  - limited information retrieved from patients with a thick chest wall (obesity) or overcrowded ribs (underweight) due to penetration of ultrasound waves
- **risks:** No absolute risks

Transoesophageal Echocardiography (TEE)

- **description:** invasive procedure used to complement transthoracic echocardiography
  - ultrasound probe inserted into the esophagus to allow for better resolution of the heart and structures
  - better visualization of posterior structures, including left atrium, mitral and aortic valves, inter-atrial septum
  - use with Doppler to quantify degree of valvular stenosis or regurgitation
- **indications**
  - should be performed as the initial test in certain life-threatening situations, (e.g. aortic dissection) when other tests contraindicated (e.g. CT angiography in patient with renal failure) or in situations wherewhen TTE is likely to be non-diagnostic
  - intracardiac thrombi, tumours, valvular vegetations (infective endocarditis), aortic dissection, aortic atheromas, prosthetic valve function, shunt, technically inadequate transthoracic study
  - evaluation for left atrial/left atrial appendage thrombus in a patient with atrial fibrillation/atrial flutter to facilitate clinical decision making regarding anticoagulation, cardioversion, or ablation
- **contraindications**
  - suspected acute aortic pathology (i.e. dissection, transection, intramural hematoma)
  - suspected prosthetic valve dysfunction
  - suspected complications of endocarditis (fistula, abscess)
  - known serious esophageal pathology (e.g. esophageal stricture, bleeding esophageal varices)
- **risks**
  - serious complications are extremely rare (<1 in 5,000)
  - esophageal perforation
  - gastrointestinal bleeding
  - pharyngeal hematoma
  - methemoglobinemia (topical benzocaine and related agents used for posterior pharyngeal anaesthesia)

Stress Echocardiography (SE)

- **description:** echocardiography using either exercise (treadmill or bicycle) or pharmacologic agents (dobutamine) as the stress mechanism
- **indications**
  - when ECG cannot be interpreted appropriately
  - intermediate pre-test probability with normal/equivocal exercise ECG
  - post-ACS when used to decide on potential efficacy of revascularization
  - to evaluate the clinical significance of valvular heart disease
  - evaluation of myocardial viability, dyspnea of possible cardiac origin, mitral valve disease, aortic stenosis, mitral regurgitation, pulmonary hypertension in patients with hypertrophic cardiomyopathy (for LVOT obstruction)
  - dobutamine
    - pharmacologic stress for patients who are physically unable to exercise; same indications as exercise stress echo
    - low dose dobutamine stress echo can be used to assess myocardial viability and for assessing aortic stenosis with LV systolic dysfunction
- **contraindications**
  - contraindications to exercise testing
  - contraindications to dobutamine stress echocardiography: tachyarrhythmias and systemic hypertension
  - AAA has been considered as a relative contraindication to exercise testing or dobutamine stress echocardiograph
- **risks**
  - no known adverse effects
  - dobutamine: cardiac and non-cardiac side effects can occur; VF and MI are rare

Contrast Echocardiography with Agitated Saline Contrast

- **description:** improves resolution and provides real-time assessment of intracardiac blood flow
  - conventional agent is agitated saline (contains microbubbles of air)
  - allows visualization of right heart and intracardiac shunts, most commonly patent foramen ovale (PFO) and intrapulmonary shunt
- **indications**
  - cardiac shunt (ASD, VSD, etc.)
  - extra-cardiac shunt: PDA
  - pulmonary AV fistula
  - patent foramen ovale (PFO)
  - structure identification (persistent left SVC)
  - evaluation of complex congenital heart disease
  - evaluation of TR by enhancing the Doppler signal

Contrast Echocardiography with Transpulmonary Contrast Agents

- **description:** newer contrast agents are capable of crossing the pulmonary bed and achieving left heart opacification following intravenous injection; these contrast agents improve visualization of endocardial borders and enhance evaluation of LV ejection fraction, wall motion abnormalities, and intracardiac mass
- **indications**
  - visualization of the endocardial border when ≥2 consecutive segments are not seen on non-contrast images
  - potential assessment of myocardial profusion, viability
- **contraindications**
  - known hypersensitivity to perflutren (Definity)
  - for FS069 (Optison) only, known hypersensitivity to blood, blood products, or albumin
  - fixed right to left, bi-directional or transient right to left cardiac shunts
- **risks**
  - risk of non-fatal MI and death are rare
  - ultrasound contrast agents may cause back pain, headache, uticaria, and anaphylaxis

Stress Testing

EXERCISE TESTING

- **description:** cardiovascular stress test that uses treadmill or bicycle exercise with electrocardiographic and blood pressure monitoring
- **indications**
- patients with intermediate (10-90%) pretest probability of CAD based on age, gender, and symptoms
- ST depression <1 mm at rest, no left bundle branch block, no digoxin or estrogen use

Most Commonly Used Treadmill Stress Test Protocols

- **The Bruce Protocol**: 7 stage test with each stage lasting 3 min. With each successive stage, the treadmill increases in both speed (2.7 km/h to 9.6 km/h) and grade (10% with a 2% increase per stage up to 22%)
- **The Modified Bruce, Modified Naughton Protocol:** for older individuals or those with limited exercise capacity

- exercise test results stratify patients into risk groups
  1. low risk patients can be treated medically without invasive testing
  2. intermediate risk patients may need additional testing in the form of exercise imaging studies or cardiac catheterization
  3. high risk patients should be referred for cardiac catheterization
- **contraindications**
  - acute myocardial infarction (within two days)
  - unstable angina pectoris
  - uncontrolled arrhythmias causing symptoms of hemodynamic compromise
  - symptomatic severe valvular stenosis
  - uncontrolled symptomatic heart failure
  - active endocarditis or acute myocarditis or pericarditis
  - acute aortic dissection
  - acute pulmonary or systemic embolism
  - acute non-cardiac disorders that may affect exercise performance or may be aggravated by exercise
  - termination of exercise testing
    - patient's desire to stop
    - drop in systolic blood pressure of >10 mmHg from baseline despite an increase in workload, when accompanied by other evidence of ischemia
    - moderate to severe angina
    - ST elevation (>1 mm) in leads without diagnostic Q-waves (other than V1 or aVR)
    - increasing nervous system symptoms (e.g. ataxia, dizziness, or near syncope)
    - signs of poor perfusion (cyanosis or pallor)
    - technical difficulties in monitoring ECG or systolic blood pressure
    - sustained ventricular tachycardia
- **risks:** death, myocardial infarction, arrhythmia, hemodynamic instability, and orthopedic injury (<1-5/10,000 supervised tests)

Important Contraindications to Exercise Testing
- Acute MI, aortic dissection, pericarditis, myocarditis, PE
- Severe AS, arterial HTN
- Inability to exercise adequately

Important Prognostic Factor
Duke Treadmill Score (DTS) Weighted Index Score
- Treadmill exercise time using standard Bruce protocol
- Maximum net ST segment deviation (depression or elevation)
- Exercise-induced angina provides diagnostic and prognostic information (such as 1 yr mortality)

DTS = exercise time – (5 x MaxST) – (4 x angina index)
Angina index: 0 (no angina), 1 (angina but not exercise-limiting), 2 (exercise-limiting angina)
DTS ≥5: 0.25% 1 yr mortality
DTS 4 to -10: 1.25% 1 yr mortality
DTS ≤ -11: 5.25% 1 yr mortality

Ann Intern Med 1987;106:793-800

Patients with normal imaging (nuclear perfusion or stress echo) studies at peak stress have a <1%/yr incidence of death or nonfatal MI and are thus often spared further invasive evaluation

NUCLEAR CARDIOLOGY

- **description**
  - myocardial perfusion imaging (MPI) with ECG-gated single photon emission computed tomography (SPECT), using radiolabelled tracer
  - evaluates myocardial viability, detects ischemia, and assesses perfusion and LV function simultaneously
  - predicts the likelihood of further cardiac event rates independent of the patient's history, examination, resting ECG, and stress ECG
  - often denoted as MIBI scan with reference to radiolabelled tracer (sestamibi)
  - stress with either treadmill or IV vasodilator stress (dipyridamole, adenosine, regadenoson)
  - images of the heart obtained during stress and at rest 3-4 h later
  - tracers
    - Thallium-201 (^{201}Tl, a K^+ analogue)
    - Technetium-99 (^{99}Tc)-labeled tracer (sestamibi/Cardiolite® or hexamibi/Myoview®)
- **Indications**
  - exercise MPI
    - when ECG cannot be interpreted appropriately
    - intermediate pre-test probability with normal/equivocal exercise ECG
    - in patients with previous imaging whose symptoms have changed
    - to diagnose ischemia
  - dipyridamole/adenosine MPI
    - to diagnose CAD in possible ACS patients with non-diagnostic ECG and negative serum biomarkers
    - when ECG is cannot be interpreted appropriately due to LBBB or V-paced rhythm among patients unable to exercise, with the same indications as exercise MPI
- **contraindications**
  - contraindications to exercise testing
  - vasodilators (i.e. adenosine, regadenoson, and dipyridamole) are contraindicated in patients with hypotension, sick sinus syndrome, high-degree AV block (in the absence of backup pacemaker capability), and reactive airways disease
  - pregnancy
- **risks:** radiation exposure

STRESS ECHOCARDIOGRAPHY

- see *Echocardiography*, C12

Cardiac Catheterization and Angiography

Right Heart Catheterization (Swan-Ganz Catheter)

- **description:** also known as pulmonary artery catheterization
  - obtain direct measurements of central venous, right-sided intracardiac, pulmonary artery, and pulmonary artery occlusion pressures
  - can estimate cardiac output, systemic and pulmonary vascular resistance as well as mixed venous oxyhemoglobin saturation, oxygen delivery, and oxygen uptake
  - right atrial, right ventricular, and pulmonary artery pressures are recorded
  - can also be used to measure the Cardiac Index (CI)
    - CI = CO/body surface area
    - cardiac index is a measure of cardiac function
    - <1.8 L/min/m^2 usually means cardiogenic shock
    - 2.6-4.2 L/min/m^2 is considered normal
  - pulmonary capillary wedge pressure (PCWP)
    - obtained by advancing the catheter to wedge in the distal pulmonary artery
    - records pressure measured from the pulmonary venous system
    - in the absence of pulmonary venous disease reflects left atrial pressure
- **indications**
  - unexplained or unknown volume status in shock
  - severe cardiogenic shock (e.g. acute valvular disease, suspected pericardial tamponade)
  - suspected or known pulmonary artery hypertension
  - severe underlying cardiopulmonary disease (e.g. congenital heart disease, left-to-right shunt, severe valvular disease, pulmonary hypertension) and undergoing corrective or other surgery
- **contraindications**
  - lack of consent
  - infection at the insertion site
  - the presence of a right ventricular assist device
  - insertion during cardiopulmonary bypass
- **risks**
  - complications for diagnostic catheterization <1%
  - inadequate diagnostic procedures occur in <1% of cases
  - complications of insertion: atrial and/or ventricular arrhythmias (~3% of patients)
  - catheter misplacement or knotting (uncommon)
  - perforation of a cardiac chamber and rupture of a cardiac valve or the pulmonary artery (rare)
  - complications of catheterization: pulmonary artery rupture, pulmonary infarction, thromboembolic events, infection, and data misinterpretation
  - within 24 h of catheterization: death, MI, or stroke (0.2% to 0.3% of patients)

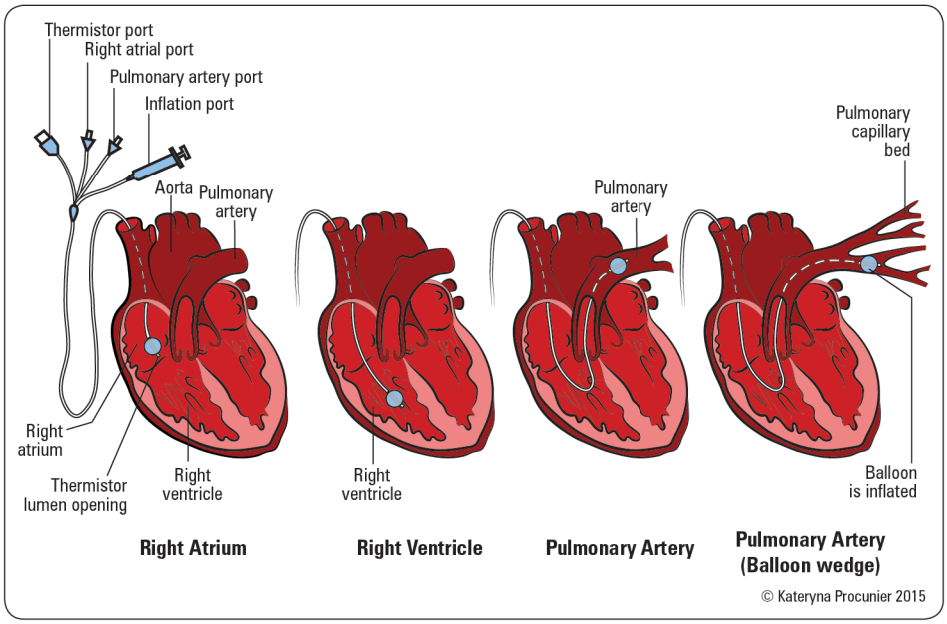


Figure 17. Swan-Ganz catheter placement

Left Heart Catheterization

- **description**
  - accomplished by introducing a catheter into the brachial or femoral artery and advancing it through the aorta, across the aortic valve, and into the left ventricle
  - evaluates mitral and aortic valvular defects and myocardial disease
  - systolic and end-diastolic pressure tracings recorded
  - LV size, wall motion and ejection fraction can be assessed by injecting contrast into the LV (left ventriculography) via femoral/radial artery catheterization
  - cardiac output (measured by the Fick oxygen method or the indicator dilution method)
- **indications**
  - identification of the extent and severity of CAD and evaluation of left ventricular function
  - assessment of the severity of valvular or myocardial disorders (e.g. aortic stenosis or insufficiency, mitral stenosis or insufficiency, and various cardiomyopathies) to determine the need for surgical correction
  - collection of data to confirm and complement noninvasive studies
  - determination of the presence of CAD in patients with confusing clinical presentations or chest pain of uncertain origin
- **contraindications**
  - severe uncontrolled hypertension
  - ventricular arrhythmias
  - acute stroke
  - severe anemia
  - active gastrointestinal bleeding
  - allergy to radiographic contrast
  - acute renal failure
  - uncompensated congestive failure (so that the patient cannot lie flat)
  - unexplained febrile illness or untreated active infection
  - electrolyte abnormalities (e.g. hypokalemia)
  - severe coagulopathy
- **risks**
  - complications for diagnostic catheterization <1%
  - inadequate diagnostic procedures occur in <1% of cases
  - within 24 h of catheterization: death, MI, or stroke (0.2% to 0.3% of patients)

Chambers	Pressure (systolic; mmHg)
Right atrium/ central venous	1-8
Right ventricle	1-8 (15-30)
Pulmonary artery	4-12 (15-30)
Left atrium/ pulmonary capillary wedge	4-12
Left ventricle end diastolic	4-12

Coronary Angiography

- **description**
  - radiographic visualization of the coronary vessels after injection of radiopaque contrast media
  - coronary vasculature accessed via the coronary ostia
- **indications**
  - to define the coronary anatomy and the degree of luminal obstruction of the coronary arteries
  - to determine the presence and extent of obstructive CAD
  - to assess the feasibility and appropriateness of various forms of therapy, such as revascularization by percutaneous or surgical interventions
  - can also be used when the diagnosis of CAD is uncertain and CAD cannot be reasonably excluded by noninvasive techniques
- **contraindications:** severe renal failure (due to contrast agent toxicity – must check patient's renal status)
- **risks:** major complications <2%, but increased in patients with pre-existing renal failure (especially in diabetic patients)

ACC/AHA 2011 Recommended Indications for Coronary Angiography

- Disabling (CCS classes III and IV) chronic stable angina despite medical therapy
- High-risk criteria on clinical assessment or non-invasive testing
- Serious ventricular arrhythmia or CHF
- Uncertain diagnosis or prognosis after non-invasive testing
- Inability to undergo non-invasive testing

Coronary Angiography
Gold standard for localizing and quantifying CAD

Hemodynamically significant stenosis is defined as 70% or more narrowing of the luminal diameter

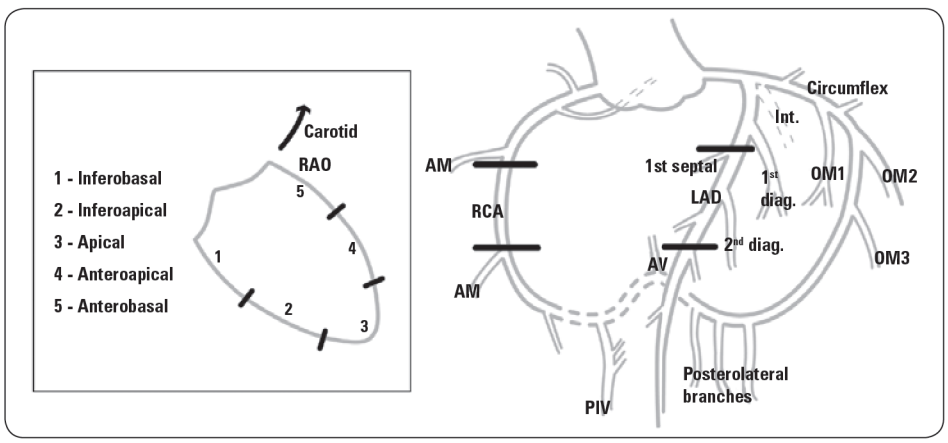


Figure 18. Coronary angiogram schematic
AM = acute marginal; LAD = left anterior descending; OM = obtuse marginal; RCA = right coronary artery

Diagnostic Catheterization

- complications for diagnostic catheterization <1%
- inadequate diagnostic procedures occur in fewer than 1% of cases
- provocative pharmacological agents can be used to unmask pathology
  - fluid loading may unmask latent pericardial constriction
  - afterload reduction or inotropic stimulation may be used to increase the outflow tract gradient in HCM
  - coronary vasoreactive agents (e.g. methylergonovine, acetylcholine)
  - a variety of pulmonary vasoreactive agents in primary pulmonary HTN (e.g. oxygen, calcium channel blockers, adenosine, nitric oxide, or prostacyclin)

Contrast-Enhanced CT Coronary Angiography

- **description:** fast ECG-synchronized multi-slice CT image acquisition in the heart to enable non-invasive imaging of the coronary arterial tree
- **indications:** often used to assess coronary artery and previous graft stenosis/viability that could not be seen during coronary angiography
- sensitivity = 85%, specificity = 90% for the diagnosis of obstructive coronary disease with >50% stenosis
- **contraindications:** allergy to contrast dye; severe renal dysfunction
- **risks:** radiation exposure

Magnetic Resonance Imaging

- **description:** offers high spatial resolution, eliminates the need for iodinated contrast, and does not involve exposure to ionizing radiation
- **indications**: valuable in assessment of congenital cardiac anomalies, abnormalities of the aorta, and assessment of viable myocardium
- **contraindications:** metallic foreign bodies/implants
- **risks:** hazards posed by certain metallic devices inside patients

CARDIAC DISEASE

Arrhythmias

Mechanisms of Arrhythmias

Alterations in Impulse Formation

A. Abnormal Automaticity

- automaticity is a property of certain cardiomyocytes to spontaneously depolarize to their threshold voltage to generate action potentials in a rhythmic fashion
- under normal circumstances only cells in the specialized conduction system (SA node, AV node, and ventricular conduction system) exhibit natural automaticity. These cells are pacemaking cells. The automaticity of these cells can become abnormally increased or decreased
- in disease (e.g. post-MI ventricular ischemia) cells in the myocardium outside the conduction system may inappropriately acquire the property of automaticity and contribute to abnormal depolarization. If these ectopic generators depolarize at a rate greater than the SA node, they assume pacemaking control and become the source of abnormal rhythm
- automaticity can be influenced by:
  - neurohormonal tone (sympathetic and parasympathetic stimulation)
  - abnormal metabolic conditions (hypoxia, acidosis, hypothermia)
  - electrolyte abnormalities
  - drugs (e.g. digitalis)
  - local ischemia/infarction
  - other cardiac pathology
- this mechanism is responsible for the accelerated idioventricular rhythm and ventricular tachycardia that often occurs 24-72 h post MI

Sinus Arrhythmia (SA)
- Normal P waves, with variation of the P-P interval by >120 msec due to varying rate of SA node

Respiratory SA
- Seen more often in young adults (<30 yr old)
- Normal, results from changes in autonomic tone during respiratory cycle
- Rate increases with inspiration, slows with expiration

Non-Respiratory SA
- Seen more often in the elderly
- Can occur in the normal heart; if marked may be due to sinus node dysfunction (e.g. in heart disease, or after digitalis toxicity)
- Usually does not require treatment

B. Triggered Activity due to Afterdepolarizations

1. Early Afterdepolarizations

- occur in the context of action potential prolongation
- consequence of the membrane potential becoming more positive during repolarization (e.g. not returning to baseline)
- result in self-maintaining depolarizing oscillations of action potential, generating a tachyarrhythmia (e.g. new baseline voltage is greater than threshold, which automatically triggers a new action potential after the refractory period ends)
- basis for the degeneration of QT prolongation, either congenital or acquired, into Torsades de Pointes

2. Delayed Afterdepolarizations

- occur after the action potential has fully repolarized, but before the next usual action potential, thus called a delayed afterdepolarization
- commonly occurs in situations of high intracellular calcium (e.g. digitalis intoxication, ischemia) or during enhanced catecholamine stimulation (e.g. "twitchy" pacemaker cells)

Alterations in Impulse Conduction

A. Re-Entry Circuits

- the presence of self-sustaining re-entry circuit causes rapid repeated depolarizations in a region of myocardium (see Figure 26, C22, for an example in the context of AV nodal re-entrant tachycardia)
  - e.g. myocardium that is infarcted/ischemic will consist of non-excitable and partially excitable zones which will promote the formation of re-entry circuits

B. Conduction Block

- ischemia, fibrosis, trauma, and drugs can cause transient, permanent, unidirectional or bidirectional block
- most common cause of block is due to refractory myocardium (cardiomyocytes are in refractory period or zone of myocardium unexcitable due to fibrosis)
- if block occurs along the specialized conduction system distal zones of the conduction system can assume pacemaking control
- conduction block can lead to bradycardia or tachycardia when impaired conduction leads to re-entry phenomenon

C. Bypass Tracts

- normally the only conducting tract from the atria to the ventricles is the AV node into the His-Purkinje system
- congenital/acquired accessory conducting tracts bypass the AV node and facilitate premature ventricular activation before normal AV node conduction
- see *Pre-Excitation Syndromes*, C23

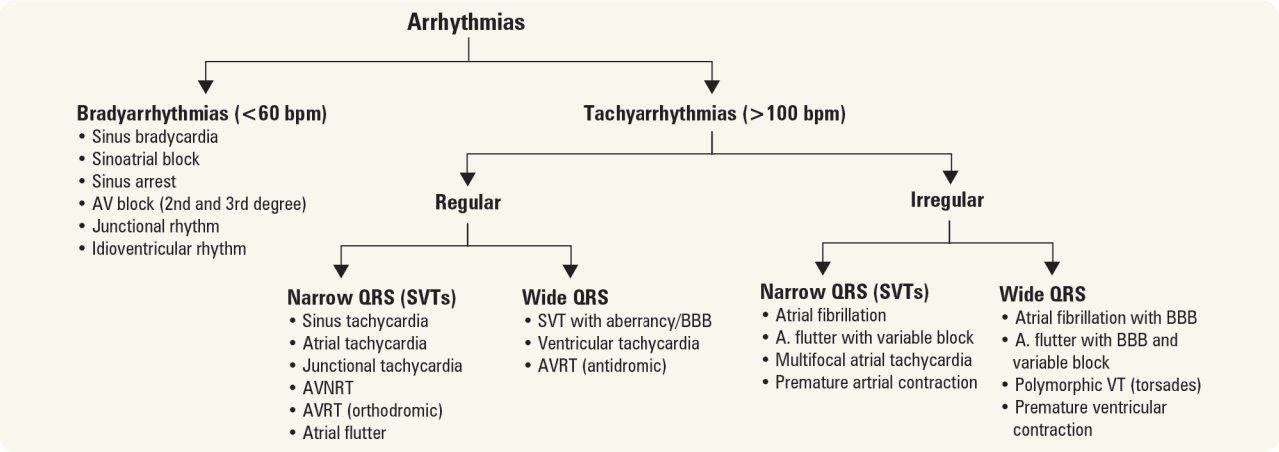


Figure 19. Clinical approach to arrhythmias

Bradyarrhythmias

Bradyarrhythmias
• Examples

1. SA NODAL DYSFUNCTION

A. A. Sinus Bradycardia

- P axis normal (P waves positive in I and aVF)
- Rate <60 bpm ; marked sinus bradycardia (<50 bpm)
- May be seen in normal adults, particularly athletes, and in elderly individuals
- Increased vagal tone or vagal stimulation; drugs (β-blockers, calcium channel blockers, etc.); ischemia/infarction

Atropine; pacing for sick sinus syndrome

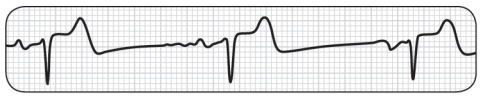


Figure 20. Sinus bradycardia

2. AV CONDUCTION BLOCKS

A. First Degree AV Block

- Prolonged PR interval (>200 msec)
- Frequently found among otherwise healthy adults

No treatment required

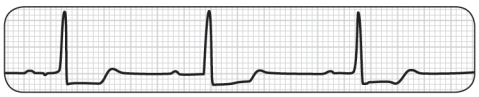


Figure 21. First degree AV block

B. Second Degree AV Block: Type I (Mobitz I)

- A gradual prolongation of the PR interval precedes the failure of conduction of a P wave (Wenckebach phenomenon)
- AV block is usually in AV node (proximal) triggers (usually reversible): increased vagal tone (e.g. following surgery), RCA-mediated ischemia

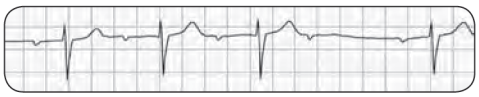


Figure 22. Second degree AV block with Wenckebach phenomenon (Mobitz I) (4:3 conduction) (lead V1)

B. Second Degree AV Block: Type II (Mobitz II)

- The PR interval is constant; there is an abrupt failure of conduction of a P wave
- AV block is usually distal to the AV node (i.e. bundle of His); increased risk of high grade or 3rd degree AV block

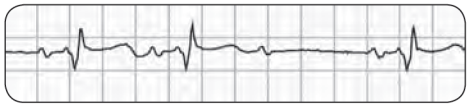


Figure 23. Second degree AV block (Mobitz II) (3:2 conduction) (lead V1)

B. Third Degree AV Block: Type II

- Complete failure of conduction of the supraventricular impulses to the ventricles; ventricular depolarization initiated by an escape pacemaker distal to the block
- Wide or narrow QRS, P-P and R-R intervals are constant, variable PR intervals; no relationship between P waves and QRS complexes (P waves "marching through")

Management (see *Electrical Pacing*, C25)

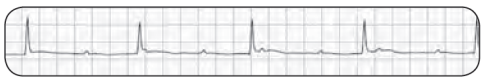


Figure 24. Third degree AV block (complete heart block) (lead II)

Supraventricular Tachyarrhythmias

Tachyarrhythmias
• Examples

Presentation for SVT (and Pre-Excitation Syndromes)

- presentation can include: palpitations, dizziness, dyspnea, chest discomfort, presyncope/syncope
- may precipitate CHF, hypotension, or ischemia in patients with underlying disease
- untreated tachycardias can cause cardiomyopathy (rare, potentially reversible with treatment of SVTs)
- includes supraventricular and ventricular rhythms

Supraventricular Tachyarrhythmias

- tachyarrhythmias that originate in the atria or AV junction
- this term is used when a more specific diagnosis of mechanism and site of origin cannot be made
- characterized by narrow QRS, unless there is pre-existing bundle branch block or aberrant ventricular conduction (abnormal conduction due to a change in cycle length)

Sinus Tachycardia

- sinus rhythm with rate >100 bpm
- occurs in normal subjects with increased sympathetic tone (e.g. exercise, emotions, pain), alcohol use, caffeinated beverages, drugs (e.g. β-adrenergic agonists, anticholinergic drugs, etc.)
- etiology: fever, hypotension, hypovolemia, anemia, thyrotoxicosis, CHF, MI, shock, PE, etc.
- treatment: treat underlying disease; consider β-blocker if symptomatic, calcium channel blocker if β-blockers contraindicated

Premature Beats
- premature atrial contraction
  - ectopic supraventricular beat originating in the atria
  - P wave morphology of the PAC usually differs from that of a normal sinus beat
- junctional premature beat
  - ectopic supraventricular beat that originates in the vicinity of the AV node
  - P wave is usually not seen or an inverted P wave is seen and may be before or closely follow the QRS complex (referred to as a retrograde, or "traveling backward" P wave)
- treatment usually not required

Atrial Flutter
- rapid, regular atrial depolarization from a macro re-entry circuit within the atrium (most commonly the right atrium)
- atrial rate 250-350 bpm, usually 300 bpm
- AV block usually occurs; it may be fixed (2:1, 3:1, 4:1, etc.) or variable
- etiology: CAD, thyrotoxicosis, mitral valve disease, cardiac surgery, COPD, PE, pericarditis
- ECG: sawtooth flutter waves (most common type of flutter) in inferior leads (II, III, aVF); narrow QRS (unless aberrancy); commonly see HR of 150
- in atrial flutter with 2:1 block, carotid sinus massage (first check for bruits), Valsalva maneuver, or adenosine may decrease AV conduction and bring out flutter waves
- treatment of acute atrial flutter
  - acute and if unstable (e.g. hypotension, CHF, angina): electrical cardioversion
  - if unstable (e.g. hypotension, CHF, angina): electrical cardioversion
  - if stable
    1. rate control: β-blocker, diltiazem, verapamil, or digoxin
    2. chemical cardioversion: sotalol, amiodarone, type I antiarrhythmics, or electrical cardioversion
  - anticoagulation guidelines same as for patients with AFib
- treatment of long-term atrial flutter: antiarrhythmics, catheter radiofrequency (RF) ablation (success rate dependent on site of origin of atrial flutter – i.e. whether right-sided isthmus-dependent or left-sided origin)

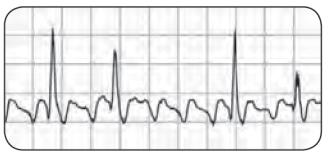


Figure 25. Atrial flutter with variable block

Multifocal Atrial Tachycardia (MAT)
- irregular rhythm caused by presence of 3 or more atrial foci (may mimic AFib)
- atrial rate 100-200 bpm – 3 or more distinct P wave morphologies and PR intervals vary, some P waves may not be conducted
- occurs more commonly in patients with COPD, and hypoxemia; less commonly in patients with hypokalemia, hypomagnesemia, sepsis, theophylline, or digitalis toxicity
- treatment: treat the underlying cause; calcium channel blockers may be used (e.g. diltiazem, verapamil), β-blockers may be contraindicated because of severe pulmonary disease
- no role for electrical cardioversion, antiarrhythmics, or ablation

Atrial Fibrillation
- see *CCS Atrial Fibrillation Guidelines 2014* for details (free mobile app – iCCS available on iOS and Android)
- most common sustained arrhythmia
- incidence increases with age (10% of population >80 yr old)
- symptoms: palpitations, fatigue, syncope, may precipitate or worsen heart failure
- **classification**
  - chronic/permanent: continuous AFib that is unresponsive to cardioversion; cardioversion should not be reattempted
  - lone: occurs in persons younger than 60 yr and in whom no clinical or echocardiographic causes are found
  - nonvalvular: not caused by valvular disease, prosthetic heart valves, or valve repair
  - paroxysmal: episodes that terminate spontaneously
  - persistent: AFib sustained for more than 7 d or AFib that terminates only with cardioversion
  - recurrent: two or more episodes of AFib
  - secondary: caused by a separate underlying condition or event (e.g. myocardial infarction, cardiac surgery, pulmonary disease, hyperthyroidism)
  - may be associated with thromboembolic events (stroke risk can be assessed by CHADS2 score in nonvalvular AFib)
- **initiation**
  - single circuit re-entry and/or ectopic foci act as aberrant generators producing atrial tachycardia (350-600 bpm)
  - impulses conduct irregularly across the atrial myocardium to give rise to fibrillation
  - in some cases, ectopic foci have also been mapped to the pulmonary vein ostia and can be ablated

Atrial Fibrillation – AFFIRM Trial
NEJM 2002;347:1825-1833
Study: Randomized, multicentre trial with mean follow-up of 3.5 yr.
Population: 4,060 patients (mean age 70 yr, 61% male, 89% white) with AF and a high risk of stroke or death.
Intervention: Rate control (β-blockers, calcium channel blockers, or digoxin alone or in combination) vs. rhythm control (antiarrhythmic drug chosen by the treating physician).
Primary Outcome: All cause mortality.
Results: There was no difference in mortality or disabling stroke, anoxic encephalopathy, major bleeding, and cardiac arrest between the two groups. There were more incidents of hospitalizations (80.1% vs. 73%, $p<0.001$) and adverse events (Torsades de Pointes (12 vs. 2, $p=0.007$), pulseless or bradycardic arrest (9 vs. 1, $p=0.01$), pulmonary event (108 vs. 24, $p<0.001$), gastrointestinal event (127 vs. 35, $p<0.001$), prolonged QT interval (31 vs. 4, $p\leq0.001$), bradycardia (105 vs. 64, $p=0.001$) in the rhythm-control group.
Conclusion: Rate-control was as effective as rhythm-control in AF and was better tolerated. There were more hospitalization incidents in the rhythm-control group.

- **maintenance**
  - the tachycardia causes atrial structural and electrophysiological remodelling changes that further promote AFib; the longer the patient is in AFib the more difficult it is to convert back to sinus rhythm
- **consequences**
  - the AV node irregularly filters incoming atrial impulses producing an irregular ventricular response of <200 bpm and the tachycardia leads to suboptimal cardiac output
  - fibrillatory conduction of the atria promotes blood stasis increasing the risk of thrombus formation – AFib is an important risk factor for stroke

Table 5. CHADS2 Risk Prediction for Non-Valvular AFib and Refer to AHA/ACC/HRS AFib Guidelines 2014 for more details

Risk Factor	Points	CHADS2 Score	Stroke Risk (%/Yr)	Anticoagulation Recommendation
Congestive Heart Failure	1	0	1.9 (low)	ASA 81-325 mg OD
Hypertension	1	1	2.8 (low-mod)	oral anticoagulants*
Age >75	1	2-3	4.0-5.9 (mod)	oral anticoagulants*
Diabetes	1	4-6	8.5-18.2 (high)	oral anticoagulants*
Stroke/TIA (prior)	2			

JAMA 2001;285:2864-70 and *Can J Cardiol* 2014 Oct;30(10):1114-30.
Oral anticoagulants *currently includes warfarin (INR 2-3) and direct oral anticoagulants (DOACs) e.g. apixaban, dabigatran, rivaroxaban
Note: recent CCS update recommends OAC if age ≥65 or if age <65 with at least one risk factor (hypertension, diabetes, CHF, stroke/TIA)

AFib on ECG

- no organized P waves due to rapid atrial activity (350-600 bpm) causing a chaotic fibrillatory baseline
- irregularly irregular ventricular response (typically 100-180 bpm), narrow QRS (unless aberrancy or previous BBB)
- wide QRS complexes due to aberrancy may occur following a long-short cycle sequence ("Ashman phenomenon")
- loss of atrial contraction, thus no "a" wave seen in JVP, no S4 on auscultation

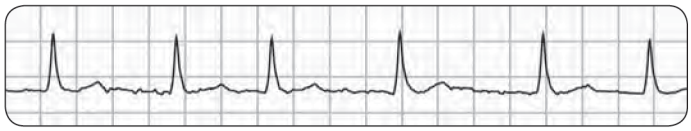


Figure 26. Atrial fibrillation (lead II)

Management (adapted from CCS Atrial Fibrillation Guidelines 2012 & 2014)

Major objectives (RACE): all patients with AF (paroxysmal, persistent, or permanent), should be stratified using a predictive index for stroke risk and for the risk of bleeding, and most patients should receive either an oral anticoagulant or ASA (see Table 5)

1. **R**ate control: β-blockers, diltiazem, verapamil (in patients with heart failure: digoxin, amiodarone)
2. **A**nticoagulation: use either warfarin or direct oral anticoagulant (DOACs) e.g. apixaban, dabigatran, rivaroxaban to prevent thromboembolism
3. **C**ardioversion (electrical)
   - if AFib <24-48 h, can usually cardiovert without anticoagulation
   - if AFib >24-48 h, anticoagulate for 3 wk prior and 4 wk after cardioversion due to risk of unstable intra-atrial thrombus
   - if patient unstable (hypotensive, active angina due to tachycardia, uncontrolled heart failure) should cardiovert immediately
4. **E**tiology
   - HTN, CAD, valvular disease, pericarditis, cardiomyopathy, myocarditis, ASD, post-operative, PE, COPD, thyrotoxicosis, sick sinus syndrome, alcohol ("holiday heart")
   - may present in young patients without demonstrable disease ("lone AFib") and in the elderly without underlying heart disease

Additional Management Points Regarding AFib

- studies of patients with AFib suggest that there is no difference in long-term survival when treating patients with a rhythm-control versus rate-control strategy
- however, many patients with a significant underlying structural heart lesion (e.g. valve disease, cardiomyopathy) will not tolerate AFib well (since may be dependent on atrial kick) and these patients should be cardioverted (chemical or electrical) as soon as possible

CHA2DS2-VASc Score
The European Society of Cardiology (ESC) and the Canadian Cardiovascular Society (CCS) have incorporated the Birmingham 2009 schema (CHA2DS2-VASc) for the prediction of stroke risk in latest guidelines. The CHADS2 (Table 5) score is to be applied first; followed by the VASc schema if the score is <2 to further grade the risk of stroke in patients at low risk. A score of 0 indicates the patient is very low risk of stroke and may require either ASA alone or no antithrombotic therapy, with the latter preferred. A score of 1 indicates the utility of either ASA or an oral anticoagulant, with the latter preferred. A patient with a score of ≥2 should receive an oral anticoagulant. For more information, please see Focused 2012 Update of the CCS AFib Guidelines, *Can J Cardiol* 2012;28:125-136.

Rivaroxaban for Stroke Prevention in AFib – ROCKET-AF Trial
NEJM 2011;365:883-891
Study: Prospective, non-inferiority, double blind, RCT, median follow-up of 1.9 yr.
Population: 14,264 patients with AFib (mean CHADS2=3.5). Patients either had previous thromboembolism or ≥3 risk factors.
Intervention: Patients were randomized to receiving rivaroxaban or warfarin.
Outcome: Composite of strokes and systemic thromboembolic event (STE).
Results: The hazard ratio of the primary outcome for rivaroxaban compared to warfarin was 0.88; 95% CI 0.74-1.03; p<0.001 for noninferiority; p=0.12 for superiority. Furthermore, the hazard ratio for major and non-major, but clinically relevant, bleeding was 1.03; 95% CI 0.96-1.11; p=0.44). There were also significant reductions in intracranial hemorrhage (0.5% vs. 0.7%, p=0.02) and fatal bleeding (0.2% vs. 0.5%, p=0.003) for rivaroxaban.
Conclusions: In patients with AFib, rivaroxaban is non-inferior to warfarin for stroke prevention and major and non-major bleeding.

Oral Anticoagulants vs. Antiplatelet Therapy for Preventing Stroke in Patients with Non-Valvular Atrial Fibrillation and No History of Stroke or Transient Ischemic Attacks
Cochrane DB Syst Rev 2009;3:CD006186
Study: Cochrane DB Syst Rev 8 RCTs with mean 1.9 yr of follow-up.
Population: 9,598 total patients with non-valvular AF and no history of stroke or transient ischemic attack.
Intervention: Long-term adjusted-dose warfarin versus ASA (dose ranging from 75-325 mg). **Outcome:** All cause mortality, all stroke, vascular death, MIs.
Results: Dose-adjusted warfarin therapy significantly reduced all stroke (OR 0.68, 95% CI 0.54-0.85), ischemic stroke (OR 0.53, 95% CI 0.41-0.68), and systemic emboli risk (OR 0.48, 95% CI 0.25-0.90). There was no significant difference in disabling or fatal strokes, MI, vascular death, or all cause mortality. There was a significantly increased risk of intracranial hemorrhage with warfarin therapy versus ASA (OR 1.98, 95% CI 1.20-3.28).
Conclusion: Long-term adjusted-dose warfarin significantly reduces all stroke and embolic risks but does not reduce risk of disability or mortality and carries a significant intracranial hemorrhage risk. The threshold of benefit for anticoagulation vs. antiplatelet therapy remains controversial.

Newly Discovered AFib

- anticoagulants may be beneficial if high risk for stroke
- if the episode is self-limited and not associated with severe symptoms, no need for antiarrhythmic drugs
- if AFib persists, 2 options
  1. rate control and anticoagulation (as indicated above)
  2. cardioversion (as above)

Recurrent AFib/Permanent AFib

- if episodes are brief or minimally symptomatic, antiarrhythmic drugs may be avoided; rate control and anticoagulation are appropriate
- patients who have undergone at least one attempt to restore sinus rhythm may remain in AFib after recurrence; permanent AFib may be accepted (with rate control and antithrombotics as indicated by CHADS2 score) in certain clinical situations
- if symptoms are bothersome or episodes are prolonged, antiarrhythmic drugs should be used
  - no or minimal heart disease: flecainide, propafenone, or sotalol
  - LV dysfunction: amiodarone
  - CAD: β-blockers, amiodarone

AV Nodal Re-Entrant Tachycardia (AVNRT)

- re-entrant circuit using dual pathways (fast conducting β-fibres and slow conducting α-fibres) within or near the AV node; often found in the absence of structural heart disease – cause is commonly idiopathic, although familial AVNRT has been reported
- sudden onset and offset
- fast regular rhythm: rate 150-250 bpm
- usually initiated by a supraventricular or ventricular premature beat
- AVNRT accounts for 60-70% of all paroxysmal SVTs
- retrograde P waves may be seen but are usually lost in the QRS complex (see Figure 27)
- treatment
  - acute: Valsalva or carotid massage, adenosine is first choice if unresponsive to vagal maneuvers; if no response, try metoprolol, digoxin, diltiazem, electrical cardioversion if patient hemodynamically unstable (hypotension, angina, or CHF)
  - long-term: 1st line – β-blocker, diltiazem, digoxin; 2nd line – flecainide, propafenone; 3rd line – catheter ablation

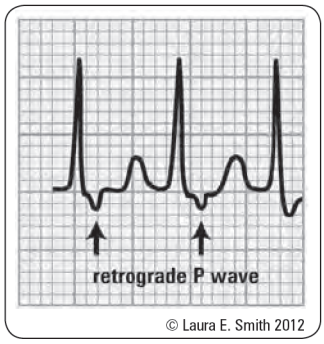


Figure 27. AVNRT

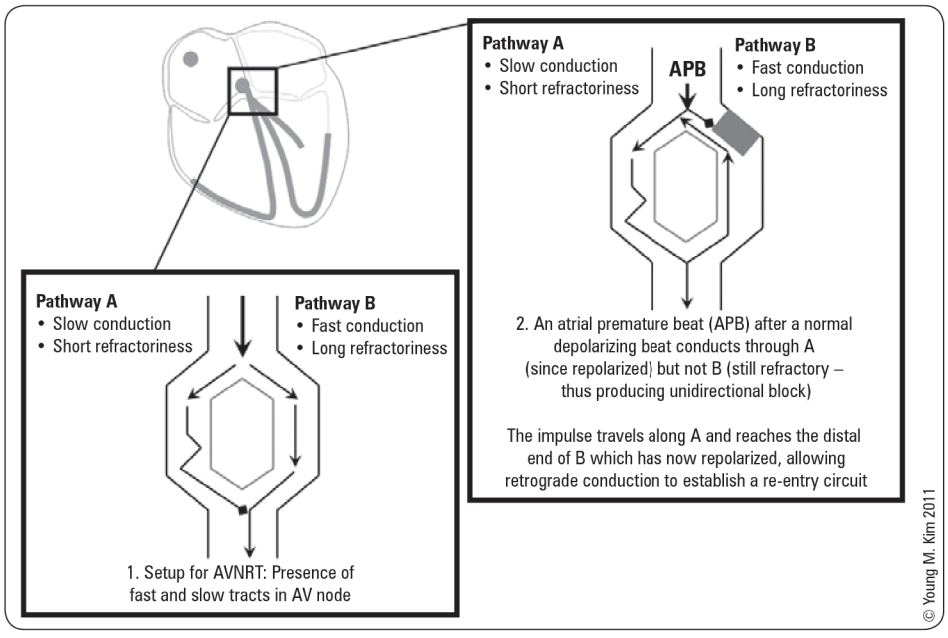


Figure 28. Mechanism for AVNRT

The carotid massage is a constant pressure directed posteriorly against the carotid artery for 5-10 s. Always listen for bruits before palpation

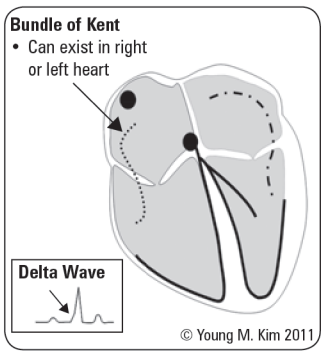


Figure 29. Accessory pathway conduction in WPW. Early ventricular activation leads to the appearance of a delta wave (slurred upstroke of the QRS) on the ECG before usual conduction across the AV node

Pre-Excitation Syndromes

- refers to a subset of SVTs mediated by an accessory pathway which can lead to ventricular pre-excitation

Wolff-Parkinson-White Syndrome

- congenital defect present in 1.5-2/1,000 of the general population
- an accessory conduction tract (Bundle of Kent; can be in right or left atrium) abnormally allows early electrical activation of part of one ventricle
- impulses travel at a greater conduction velocity across the Bundle of Kent thereby effectively 'bypassing' AV node
- since the ventricles are activated earlier, the ECG shows early ventricular depolarization in the form of initial slurring of the QRS complex – the so-called "delta wave"
- atrial impulses that conduct to the ventricles through both the Bundle of Kent and the normal AV node/His-Purkinje system generate a broad "fusion complex"
- ECG features of WPW
  - PR interval <120 msec
  - delta wave: slurred upstroke of the QRS (the leads with the delta wave vary with site of bypass)
  - widening of the QRS complex due to premature activation
  - secondary ST segment and T wave changes
  - tachyarrhythmias may occur – most often AVRT and AFib

AFib in WPW Patients

- AFib is the index arrhythmia in up to 20% of patients with WPW syndrome
  - it is usually intermittent rather than persistent or permanent
- rapid atrial depolarizations in AFib are conducted through the bypass tract which is not able to filter impulses like the AV node can
- consequently the ventricular rate becomes extremely rapid (>200 bpm) and the QRS complex widens
- treatment: electrical cardioversion, IV procainamide, or IV amiodarone
  - do not use drugs that slow AV node conduction (digoxin, β-blockers) as this may cause preferential conduction through the bypass tract and precipitate VF
  - long-term: ablation of bypass tract if possible

AV Re-Entrant Tachycardia

- re-entrant loop via accessory pathway and normal conduction system
- initiated by a premature atrial or ventricular complex
- **orthodromic** AVRT: stimulus from a premature complex travels up the bypass tract (V to A) and down the AV node (A to V) with narrow QRS complex (no delta wave because stimulus travels through normal conduction system)
- comprises 95% of the reentrant tachycardias associated with WPW syndrome
- **antidromic** AVRT: more rarely the stimulus goes up the AV node (V to A) and down the bypass tract (A to V); wide and abnormal QRS as ventricular activation is only via the bypass tract
- treatment
  - acute: similar to AVNRT except avoid long-acting AV nodal blockers (e.g. digoxin and verapamil)
  - long-term: for recurrent arrhythmias ablation of the bypass tract is recommended
    - drugs such as flecainide and procainamide can be used

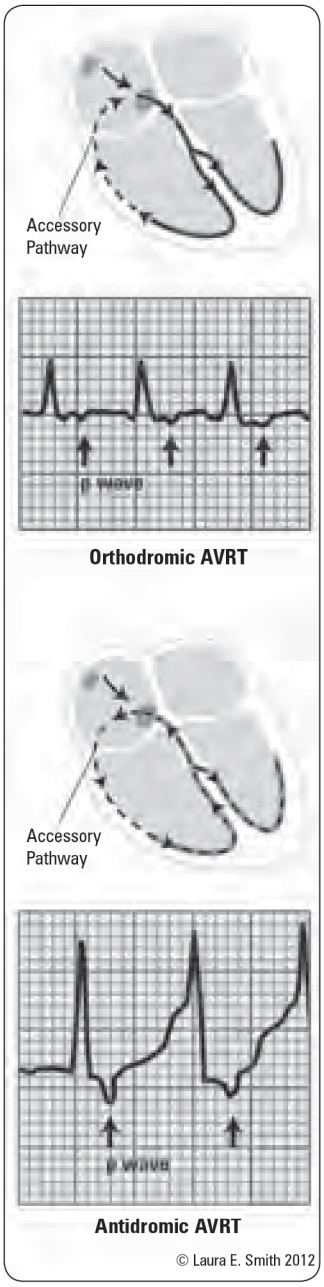


Figure 30. Orthodromic vs. antidromic AVRT

Ventricular Tachyarrhythmias

Premature Ventricular Contraction (PVC) or Ventricular Premature Beat (VPB)

- QRS width >120 msec, no preceding P wave, bizarre QRS morphology
- origin: LBBB morphology of VT = RV origin; RBBB morphology of VT = LV origin
- PVCs may be benign but are usually significant in the following situations
  - consecutive (≥3 = VT) or multiform (varied origin)
  - PVC falling on the T wave of the previous beat ("R on T phenomenon"): may precipitate ventricular tachycardia or VF

Accelerated Idioventricular Rhythm

- ectopic ventricular rhythm with rate 50-100 bpm
- more frequently occurs in the presence of sinus bradycardia and is easily overdriven by a faster supraventricular rhythm
- frequently occurs in patients with acute MI or other types of heart disease (cardiomyopathy, hypertensive, valvular) but it does not affect prognosis and does not usually require treatment

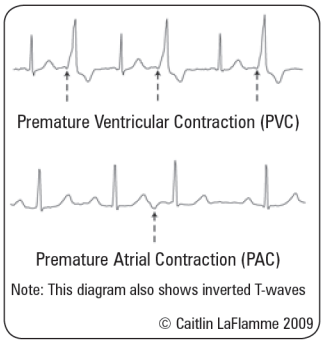


Figure 31. PVC (with bigeminy pattern) and PAC. Note the difference between the normal QRS/T wave and the PVC-generated QRS/T wave

Ventricular Tachycardia (VT)

- 3 or more consecutive ectopic ventricular complexes
  - rate >100 bpm (usually 140-200)
  - ventricular flutter: if rate >200 bpm and complexes resemble a sinusoidal pattern
  - "sustained VT" if it lasts longer than 30 s
  - ECG characteristics: wide regular QRS tachycardia (QRS usually >140 msec)
  - AV dissociation; bizarre QRS pattern
  - also favour Dx of VT: left axis or right axis deviation, nonspecific intraventricular block pattern, monophasic or biphasic QRS in V1 with RBBB, QRS concordance in V1-V6
  - occasionally during VT supraventricular impulses may be conducted to the ventricles generating QRS complexes with normal or aberrant supraventricular morphology ("ventricular capture") or summation pattern ("fusion complexes")
- **monomorphic VT**
  - identical complexes with uniform morphology
  - more common than polymorphic VT
  - typically result from intraventricular re-entry circuit
  - potential causes: chronic infarct scarring, acute MI/ischemia, cardiomyopathies, myocarditis, arrhythmogenic right ventricular dysplasia, idiopathic, drugs (e.g. cocaine), electrolyte disturbances
- **polymorphic VT**
  - complexes with constantly changing morphology, amplitude, and polarity
  - more frequently associated with hemodynamic instability due to faster rates (typically 200-250 bpm) vs. monomorphic VT
  - potential causes: acute MI, severe or silent ischemia, and predisposing factors for QT prolongation
- treatment
  - sustained VT (>30 s) is an emergency, requiring immediate treatment
  - hemodynamic compromise: electrical cardioversion
  - no hemodynamic compromise: electrical cardioversion, lidocaine, amiodarone, type Ia agents (procainamide, quinidine)

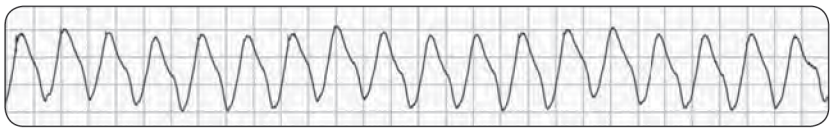


Figure 32. Ventricular tachycardia (monomorphic)

Table 6. Wide Complex Tachycardia: Clues for Differentiating VT vs. SVT with Aberrancy*

Clinical Clues		ECG Clues	
Presenting symptoms	Not helpful	AV dissociation	VT
History of CAD and previous MI	VT	Capture or fusion beats	VT
Physical exam		QRS width >140 msec	VT
Cannon "a" waves Variable S1	VT	Extreme axis deviation (left or right superior axis)	VT
Carotid sinus massage/adenosine terminates arrhythmia	SVT**	Positive QRS concordance (R wave across chest leads)	VT
		Negative QRS concordance (S wave across chest leads)	May suggest VT
		Axis shift during arrhythmia	VT (polymorphic)

*If patient >65 yr and previous MI or structural heart disease, then chance of VT >95%
**May terminate VT in some patients with no structural heart disease

Torsades de Pointes

- a variant of polymorphic VT that occurs in patients with baseline QT prolongation – "twisting of the points"
- looks like usual VT except that QRS complexes "rotate around the baseline" changing their axis and amplitude
- ventricular rate >100 bpm, usually 150-300 bpm
- etiology: predisposition in patients with prolonged QT intervals
  - congenital long QT syndromes
  - drugs: e.g. class IA (quinidine), class III (sotalol), phenothiazines (TCAs), erythromycin, quinolones, antihistamines
  - electrolyte disturbances: hypokalemia, hypomagnesemia
  - nutritional deficiencies causing above electrolyte abnormalities
- treatment: IV magnesium, temporary pacing, isoproterenol and correct underlying cause of prolonged QT, electrical cardioversion if hemodynamic compromise

Arrhythmias that May Present as a Wide QRS Tachycardia

- VT
- SVT with aberrant conduction (rate related)
- SVT with preexisting BBB or nonspecific intraventricular conduction defect
- AV conduction through a bypass tract in WPW patients during an atrial tachyarrhythmia (e.g. atrial flutter, atrial tachycardia)
- Antidromic AVRT in WPW patients (see *Pre-Excitation Syndromes*, C21)

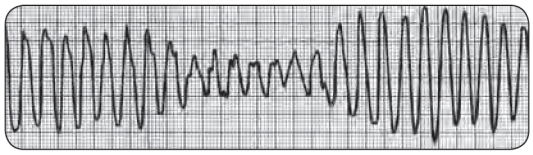


Figure 33. Torsades de pointes

Ventricular Fibrillation (VFib)

- chaotic ventricular arrhythmia, with very rapid irregular ventricular fibrillatory waves of varying morphology
- terminal event, unless advanced cardiac life-support (ACLS) procedures are promptly initiated to maintain ventilation and cardiac output, and electrical defibrillation is carried out
- most frequent cause of sudden death
- refer to ACLS algorithm for complete therapeutic guidelines

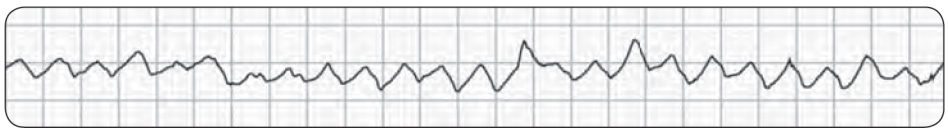


Figure 34. Ventricular fibrillation

Electrophysiology Studies

- invasive test for the investigation and treatment of cardiac rhythm disorders using intracardiac catheters
- provide detailed analysis of the arrhythmia mechanism and precise site of origin when ECG data are nondiagnostic or unobtainable
- bradyarrhythmias: define the mechanisms of SA node dysfunction and localize site of AV conduction block
- tachyarrhythmias: map for possible ablation or to assess inducibility of VT

Electrical Pacing

- the decision to implant a pacemaker usually is based on symptoms of a bradyarrhythmia or tachyarrhythmia in the setting of heart disease

Pacemaker Indications

- SA node dysfunction (most common): symptomatic bradycardia ± hemodynamic instability
- common manifestations include: syncope, presyncope, or severe fatigue
- SA node dysfunction is commonly caused by: intrinsic disease within the SA node (e.g. idiopathic degeneration, fibrosis, ischemia, or surgical trauma), abnormalities in autonomic nervous system function, and drug effects
- AV nodal-infranodal block: Mobitz II, complete heart block

Pacemaker Complications

- complications related to surgical implantation include venous access (pneumothorax, hemothorax, air embolism), pacemaker leads (perforation, malposition), pocket hematomas and infection
- complications specific to the pacemaker include a failure to pace, failure to sense, pulse generator failure, pacemaker syndrome and pacemaker mediated tachycardia

Pacing Techniques

- temporary: transvenous (jugular, subclavian, femoral) or external (transcutaneous) pacing
- permanent: transvenous into RA, apex of RV, or both
- can sense and pace atrium, ventricle, or both
- new generation: rate responsive, able to respond to physiologic demand
- biventricular

Implantable Cardioverter Defibrillators

- sudden cardiac death (SCD) usually results from ventricular fibrillation (VFib), sometimes preceded by monomorphic or polymorphic ventricular tachycardia (VT)
- ICDs detect ventricular tachyarrhythmias and are highly effective in terminating VT/VFib and in aborting SCD
- mortality benefit vs. antiarrhythmics in secondary prevention
- benefit seen in patients with ischemic and non-ischemic cardiomyopathy, depressed left ventricular ejection fraction (LVEF), prolonged QRS
- see *Heart Failure*, C36 for current treatment recommendations

CCS Consensus Conference 2003: Assessment of the Cardiac Patient for Fitness to Drive and Fly – Executive Summary
Can J Cardiol 2004;20:1313-1323
In both primary and secondary prevention ICD patients with private driving licenses, no restrictions to drive directly following implantation or an inappropriate shock are warranted. However, following an appropriate shock these patients are at an increased risk to cause harm to other road users and therefore should be restricted to drive for a period of 2 and 4 mo, respectively. In addition, all ICD patients with commercial driving licenses have a substantial elevated risk to cause harm to other road users during the complete follow-up after both implantation and shock and should therefore be restricted to drive permanently.

Systematic Review: Implantable Cardioverter Defibrillators for Adults with Left Ventricular Systolic Dysfunction
Ann Intern Med 2007;147:251-262
Study: Meta-review of 12 RCTs used for ICD efficacy, 5 RCTs and 48 observational studies for effectiveness, and 21 RCTs and 43 observational studies for safety review.
Population: 8,516 patients for ICD efficacy, 26,840 patients for effectiveness, and 86,809 patients for safety review with left ventricular ejection fraction ≤0.35.
Intervention: ICD implantation.
Outcomes: All-cause mortality and adverse events.
Results: ICDs reduced all-cause mortality by 20% (95% CI, 10%-29%; I^2=44.4%) with greatest reduction (54%) in sudden cardiac death (CI 37%-63%; I^2=0%). Observational studies had a reduced relative risk of 0.54 for all-cause mortality versus RCTs (CI 0.43-0.58, I^2=60.4%). Rates of success of ICD implantation were 99% (CI 98.8%-99.3%) with a 1.2% (CI 0.9%-1.5%) chance of peri-implantation death. Post-implantation complications (per 100 patient yr) were: 1.4 (CI 1.2-1.6) device malfunctions; 1.5 (CI 1.3-1.8) lead problems; 0.6 (CI 0.5-0.8) implant site infection and 19.1 (CI 16.5-22.0) inappropriate discharges in RCTs versus a rate of 4.9 (CI 4.5-5.3) inappropriate discharges in observational studies.
Conclusion: ICDs are safe and effective in reducing mortality in adult patients with LV systolic dysfunction but carry significant risks of inappropriate discharges. Differences between RCTs and observational studies show that improved risk stratification of patients may further improve outcomes and reduce adverse events.

Catheter Ablation

Techniques

- radiofrequency (RF) ablation: a low-voltage high-frequency form of electrical energy (similar to cautery); RF ablation produces small, homogeneous, necrotic lesions approximately 5-7 mm in diameter and 3-5 mm in depth
- cryoablation: new technology which uses a probe with a tip that can decrease in temperature to -20˚C and -70˚C. Produces small, necrotic lesions similar to RF ablation; when brought to -20˚C, the catheter tip reversibly freezes the area; bringing the tip down to -70˚C for 5 min permanently scars the tissue
  - advantage: can "test" areas before committing to an ablation
  - disadvantage: takes much longer than RF (5 min per cryoablation vs. 1 min per RF ablation)

Indications

- paroxysmal SVT
  - AVNRT: accounts for more than half of all cases
- accessory pathway (orthodromic reciprocating tachycardia): 30% of SVT
  - re-entrant rhythm, with an accessory AV connection as the retrograde limb
  - corrected by targeting the accessory pathway
- atrial flutter: reentry pathway in right atrium
- AFib: potential role for pulmonary vein ablation
- ventricular tachycardia: focus arises from the right ventricular outflow tract and less commonly originates in the inferoseptal left ventricle near the apex (note: majority of cases of VT are due to scarring from previous MI and cannot be ablated)

Major Complications

- 1% of patients
- death: 0.1-0.2%
- cardiac: high grade AV block requiring permanent pacemaker (less risk with cryoablation), tamponade, pericarditis
- vascular: hematoma, vascular injury, thromboembolism, TIA/stroke
- pulmonary: PE

Ischemic Heart Disease

Epidemiology

- most common cause of cardiovascular morbidity and mortality
- Canadian-led INTERHEART study showed that 9 modifiable risk factors accounted for >90% of MI
- atherosclerosis and thrombosis are the most important pathogenetic mechanisms
- M:F = 2:1 with all age groups included (Framingham study), 8:1 for age <40, 1:1 for age >70
  - according to the Framingham Heart Study, men develop coronary heart disease at a rate double that of women for age <60; incidence in women triples shortly after menopause
- peak incidence of symptomatic IHD is age 50-60 (men) and 60-70 (women)
- for primary prevention of ischemic heart disease see Family Medicine, FM7

Table 7. Risk Factors and Markers for Atherosclerotic Heart Disease

Non-Modifiable Risk Factors	Modifiable Risk Factors	Markers of Disease
Age	Hyperlipidemia*	Elevated lipoprotein(a)
Male, postmenopausal female	HTN*	Hyperhomocysteinemia
Family history (FHx) of MI*	DM*	Elevated high-sensitivity C-reactive protein (hsCRP)
First degree male relative <55	Cigarette smoking*	Coronary artery calcification
First degree female relative <65	Psychosocial stress	Carotid IMT/plaque
	Obesity	Ankle-brachial index
	Sedentary lifestyle	
	Heavy alcohol intake	

* Major risk factor

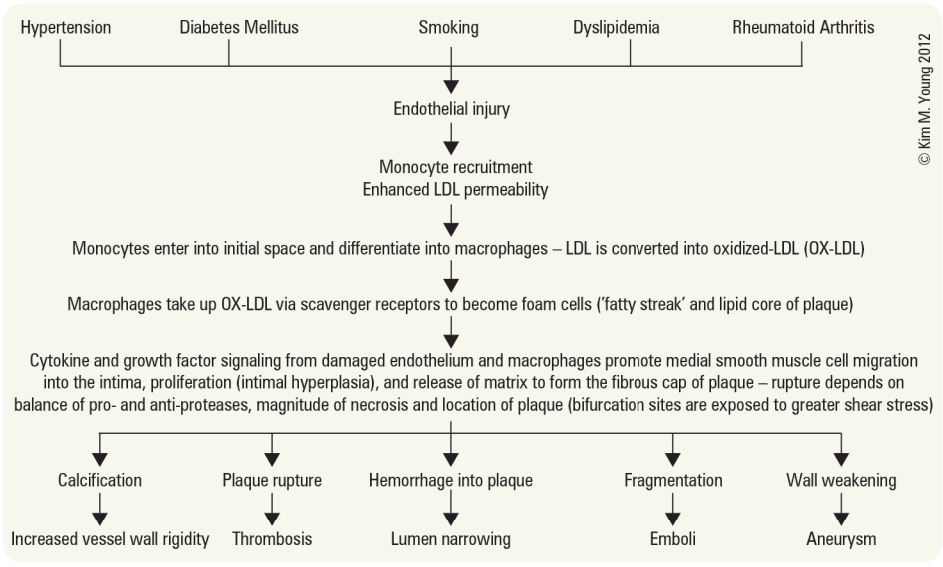


Figure 35. Pathophysiology of atherosclerosis

Chronic Stable Angina

Definition

- symptom complex resulting from an imbalance between oxygen supply and demand in the myocardium

Etiology and Pathophysiology

- factors that decrease myocardial oxygen supply
  - decreased luminal diameter: atherosclerosis, vasospasm
  - decreased duration of diastole: tachycardia (decreased duration of diastolic coronary perfusion)
  - decreased hemoglobin: anemia
  - decreased SaO_2: hypoxemia
  - congenital anomalies
- factors that increase myocardial oxygen demand
  - increased heart rate: hyperthyroidism
  - increased contractility: hyperthyroidism
  - increased wall stress: myocardial hypertrophy, aortic stenosis

Signs and Symptoms

- typical: (1) retrosternal chest pain, tightness or discomfort radiating to left (± right) shoulder/arm/neck/jaw, associated with diaphoresis, nausea, anxiety; (2) predictably precipitated by the "3 Es": exertion, emotion, eating; (3) brief duration, lasting <10-15 min and typically relieved by rest and nitrates
- atypical/probable angina (meets 2 of the above); non-cardiac chest pain (meets <1 of the above)
- Levine's sign: clutching fist over sternum when describing chest pain
- anginal equivalents: dyspnea, acute LV failure, flash pulmonary edema

Clinical Assessment

- history including directed risk factor assessment and physical exam
- labs: Hb, fasting glucose, fasting lipid profile
- ECG (at rest and during episode of chest pain if possible)
- CXR (suspected heart failure, valvular disease, pericardial disease, aortic dissection/aneurysm, or signs or symptoms of pulmonary disease)
- stress testing (see *Stress Testing*, C13) or angiography
- echo
- to assess systolic murmur suggestive of aortic stenosis, mitral regurgitation, and/or HCM
- to assess LV function in patients with Hx of prior MI, pathological Q waves, signs or symptoms of CHF

Differential Diagnosis

- see *Differential Diagnosis of Common Presentations*, C4

Chronic stable angina is most often due to a fixed stenosis caused by an atheroma

Acute coronary syndromes are the result of plaque rupture

Canadian Cardiovascular Society (CCS) Functional Classification of Angina

- **Class I:** ordinary physical activity (walking, climbing stairs) does not cause angina; angina with strenuous, rapid, or prolonged activity
- **Class II:** slight limitation of ordinary activity: angina brought on at >2 blocks on level or climbing >1 flight of stairs or by emotional stress
- **Class III:** marked limitation of ordinary activity: angina brought on at <2 blocks on level or climbing <1 flight of stairs
- **Class IV:** inability to carry out any physical activity without discomfort; angina may be present at rest

Treatment of Chronic Stable Angina

1. General Measures
- goals: to reduce myocardial oxygen demand and/or increase oxygen supply
- lifestyle modification (diet, exercise)
- treatment of risk factors: statins (see Endocrinology, E2, Family Medicine, FM9 for target lipid guidelines), antihypertensives, etc.
- pharmacological therapy to stabilize the coronary plaque to prevent rupture and thrombosis

2. Antiplatelet Therapy (first-line therapy)
- ASA
- clopidogrel when ASA absolutely contraindicated

3. β-blockers (first-line therapy – improve survival in patients with hypertension)
- increase coronary perfusion and decrease demand (HR, contractility) and BP (afterload)
- cardioselective agents preferred (e.g. metoprolol, atenolol) to avoid peripheral effects (inhibition of vasodilation and bronchodilation via β2 receptors)
- avoid intrinsic sympathomimetics (e.g. acebutolol) which increase demand

4. Nitrates (symptomatic control, no clear impact on survival)
- decrease preload (venous dilatation) and afterload (arteriolar dilatation), and increase coronary perfusion
- maintain daily nitrate-free intervals to prevent tolerance (tachyphylaxis)

5. Calcium Channel Blockers (CCBs, second-line or combination)
- increase coronary perfusion and decrease demand (HR, contractility) and BP (afterload)
- caution: verapamil/diltiazem combined with β-blockers may cause symptomatic sinus bradycardia or AV block

6. ACE Inhibitors (ACEI, not used to treat symptomatic angina)
- angina patients tend to have risk factors for CV disease which warrant use of an ACEI (e.g. HTN, DM, proteinuric renal disease, previous MI with LV dysfunction)
- benefit in all patients at high risk for CV disease (concomitant DM, renal dysfunction, or
- LV systolic dysfunction)
- angiotensin II receptor blockers (ARBs) can be used when ACEI contraindicated; avoid combining ACEI and ARB (e.g. hypersensitivity, angioedema)

7. Invasive Strategies
- revascularization (see *Coronary Revascularization*, C33 and COURAGE trial sidebar)

Optimal Medical Therapy with or without PCI for Stable Coronary Disease. COURAGE Trial
NEJM 2007;356:1503-1516
Study: Randomized, controlled trial with median follow-up of 4.6 yr.
Population: 2,287 patients who had objective evidence of myocardial ischemia and significant stable coronary artery disease.
Intervention: Patients were randomized to receive intensive pharmacologic therapy and lifestyle intervention with or without percutaneous coronary intervention (PCI).
Outcome: Primary outcome was all-cause mortality and nonfatal myocardial infarction (MI). Secondary outcome had additional events of stroke, all MI, and hospitalization for unstable angina with negative biomarkers.
Results: There was no significant difference in primary (unadjusted hazard ratio: 1.05; p=0.62) or secondary outcomes (hazard ratio: 1.05; p=0.62) between the PCI and non-PCI intervention groups. The PCI group had significantly lower rates of subsequent revascularization at 4.6 yr of follow-up (hazard ratio 0.60, p<0.001) and was more angina-free in the first 4 yr of follow-up.
Conclusions: PCI as an adjunct in initial management in patients with significant stable coronary artery disease does not reduce mortality, MI, stroke, or hospitalization for ACS, but does provide angina relief and reduced risk of revascularization.

VARIANT ANGINA (Prinzmetal's Angina)
- myocardial ischemia secondary to coronary artery vasospasm, with or without atherosclerosis
- uncommonly associated with infarction or LV dysfunction
- typically occurs between midnight and 8 am, unrelated to exercise, relieved by nitrates
- typically ST elevation on ECG
- diagnosed by provocative testing with ergot vasoconstrictors (rarely done)
- treat with nitrates and CCBs

SYNDROME X
- typical symptoms of angina but normal angiogram
- may show definite signs of ischemia with exercise testing
- thought to be due to inadequate vasodilator reserve of coronary resistance vessels
- better prognosis than overt epicardial atherosclerosis

Acute Coronary Syndromes

Definition
- ACS includes the spectrum of UA, NSTEMI, and STEMI; this distinction aids in providing the appropriate therapeutic intervention
  - **MI** is defined by evidence of myocardial necrosis. It is diagnosed by a rise/fall of serum markers plus any one of
    - symptoms of ischemia (chest/upper extremity/mandibular/epigastric discomfort; dyspnea)
    - ECG changes (ST-T changes, new BBB or pathological Q waves)
    - imaging evidence (myocardial loss of viability, wall motion abnormality, or intracoronary thrombus)
  - if biomarker changes are unattainable, cardiac symptoms combined with new ECG changes is sufficient
  - **NSTEMI** meets criteria for myocardial infarction without ST elevation or BBB
  - **STEMI** meets criteria for myocardial infarction characterized by ST elevation or new BBB

- **UA** is clinically defined by any of the following
  - accelerating pattern of pain: increased frequency, increased duration, decreased threshold of exertion, decreased response to treatment
  - angina at rest
  - new-onset angina
  - angina post-MI or post-procedure (e.g. percutaneous coronary intervention [PCI], coronary artery bypass grafting [CABG])

Investigations

- history and physical
  - note that up to 30% of MIs are unrecognized or "silent" due to atypical symptoms – more common in women, DM, elderly, post-heart transplant (because of denervation)
- ECG
- CXR
- labs
  - serum cardiac biomarkers for myocardial damage (repeat 8 h later) (see *Cardiac Biomarkers*, C11)
  - CBC, INR/PTT, electrolytes and magnesium, creatinine, urea, glucose, serum lipids
  - draw serum lipids within 24-48 h because values are unreliable from 2-48 d post-MI

MANAGEMENT OF ACUTE CORONARY SYNDROMES

1. General Measures

- ABCs: assess and correct hemodynamic status first
- bed rest, cardiac monitoring, oxygen
- nitroglycerin SL followed by IV
- morphine IV

2. Anti-Platelet and Anticoagulation Therapy

- see also CCS Antiplatelet Guidelines 2012 for details (free mobile apps available on iOS and Android platforms in the CCS app stores)
- ASA chewed
- NSTEMI
  - ticagrelor in addition to ASA or if ASA contraindicated, subcutaneous low molecular weight heparin or IV unfractionated heparin (UFH) (LMWH preferable, except in renal failure or if CABG is planned within 24 h)
  - clopidogrel used if patient ineligible for ticagrelor
- if PCI is planned: ticagrelor or prasugrel and consider IV GP IIb/IIIa inhibitor (e.g. abciximab)
  - clopidogrel used if patient ineligible for ticagrelor and prasugrel
  - prasugel contraindicated in those with a history of stroke/TIA, and avoidance of or lower dose is recommended for those >75 yr old or weighing under 60 kg (TRITON-TIMI 38)
- anticoagulation options depend on reperfusion strategy:
  - primary PCI: UFH during procedure; bivalirudin is a possible alternative
  - thrombolysis: LMWH (enoxaparin) until discharge from hospital; can use UFH as alternative because of possible rescue PCI
  - no reperfusion: LMWH (enoxaparin) until discharge from hospital
- continue LMWH or UFH followed by oral anticoagulation at discharge if at high risk for thromboembolic event (large anterior MI, AFib, severe LV dysfunction, CHF, previous DVT or PE, or echo evidence of mural thrombus)

3. β-blockers

- STEMI: contraindications include signs of heart failure, low output states, risk of cardiogenic shock, heart block, asthma or airway disease; initiate orally within 24 h of diagnosis when indicated
- if β-blockers are contraindicated or if β-blockers/nitrates fail to relieve ischemia, non-dihydropyridine calcium channel blockers (e.g. diltiazem, verapamil) may be used as second-line therapy in the absence of severe LV dysfunction or pulmonary vascular congestion (calcium channel blockers do not prevent MI or decrease mortality)

4. Invasive Strategies and Reperfusion Options

- UA/NSTEMI: early coronary angiography ± revascularization if possible is recommended with any of the following high-risk indicators:
  - recurrent angina/ischemia at rest despite intensive anti-ischemic therapy
  - CHF or LV dysfunction
  - hemodynamic instability
  - high (≥3) TIMI risk score (tool used to estimate mortality following an ACS)
  - sustained ventricular tachycardia
  - dynamic ECG changes
  - high-risk findings on non-invasive stress testing

- PCI within the previous 6 mo
- repeated presentations for ACS despite treatment and without evidence of ongoing ischemia or high risk features
- note: thrombolysis is NOT administered for UA/NSTEMI

- STEMI
  - after diagnosis of STEMI is made, do not wait for results of further investigations before implementing reperfusion therapy
  - goal is to re-perfuse artery: thrombolysis ("EMS-to-needle") within 30 min or primary PCI ("EMS-to-balloon") within 90 min (depending on capabilities of hospital and access to hospital with PCI facility)
  - thrombolysis
    - preferred if patient presents ≤12 h of symptom onset, and <30 min after presentation to hospital, has contraindications to PCI, or PCI cannot be administered within 90 min
  - PCI
    - early PCI (≤12 h after symptom onset and <90 min after presentation) improves mortality vs. thrombolysis with fewer intra-cranial hemorrhages and recurrent MIs
    - primary PCI: without prior thrombolytic therapy – method of choice for reperfusion in experienced centres (*JAMA* 2004;291:736-739)
    - rescue PCI: following failed thrombolytic therapy (diagnosed when following thrombolysis, ST segment elevation fails to resolve below half its initial magnitude and patient still having chest pain)

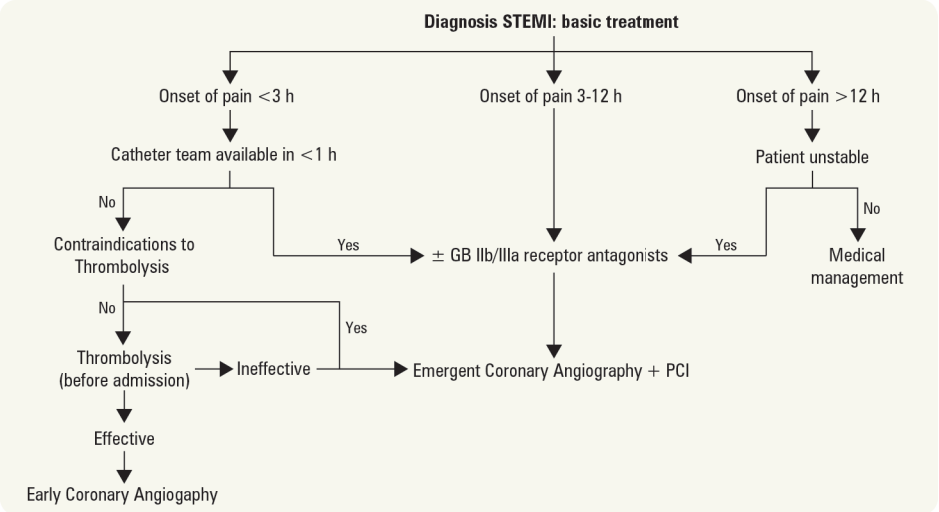


Figure 36. Reperfusion strategy in STEMI

Table 8. Contraindications for Thrombolysis in STEMI

Absolute	Relative
Prior intracranial hemorrhage	Chronic, severe, poorly controlled HTN
Known structural cerebral vascular lesion	Uncontrolled HTN (sBP >180, dBP >110)
Known malignant intracranial neoplasm	Current anticoagulation
Significant closed-head or facial trauma (≤3 mo)	Noncompressible vascular punctures
Ischemic stroke (≤3 mo)	Ischemic stroke (≥3 mo)
Active bleeding	Recent internal bleeding (≤2-4 wk)
Suspected aortic dissection	Prolonged CPR or major surgery (≤3 wk)
	Pregnancy
	Active peptic ulcer disease

Long-Term Management of ACS

- risk of progression to MI or recurrence of MI or death is highest within 1 mo
- at 1-3 mo after the acute phase, most patients resume a clinical course similar to that in patients with chronic stable coronary disease
- pre-discharge workup: ECG and echo to assess residual LV systolic function
- drugs required in hospital to control ischemia should be continued after discharge in all patients
- other medications for long-term management of ACS are summarized below

1. General Measures

- education
- risk factor modification

2. Antiplatelet and Anticoagulation Therapy
- see also CCS Antiplatelet Guidelines 2012 for details (free mobile apps available on iOS and Android platforms in the CCS app stores)
- ECASA 81 mg daily
- ticagrelor 90 mg twice daily or prasugrel 10 mg daily (at least 1 mo, up to 9-12 mo, if stent placed at least 12 mo)
- clopidogrel 75 mg daily can be used as alternatives to ticagrelor and prasugrel when indicated
- ± warfarin x 3 mo if high risk (large anterior MI, LV thrombus, LVEF <30%, history of VTE, chronic AFib)

3. β-Blockers (e.g. metoprolol 25-50 mg bid or atenolol 50-100 mg daily)

4. Nitrates
- alleviate ischemia but do not improve outcome
- use with caution in right-sided MI patients who have become preload dependent

5. Calcium Channel Blockers (NOT recommended as first line treatment, consider as alternative to β-blockers)

6. Angiotensin-Converting Enzyme Inhibitors
- prevent adverse ventricular remodelling
- recommended for asymptomatic high-risk patients (e.g. diabetics), even if LVEF >40%
- recommended for symptomatic CHF, reduced LVEF (<40%), anterior MI
- use ARBs in patients who are intolerant of ACEI; avoid combing ACE and ARB

7. ± Aldosterone Antagonists
- if on ACEI and β-blockers and LVEF <40% and CHF or DM
- significant mortality benefit shown with eplerenone by 30 d

8. Statins (early, intensive, irrespective of cholesterol level; e.g. atorvastatin 80 mg daily)

9. Invasive Cardiac Catheterization if indicated (risk stratification)

Is this Patient Having a Myocardial Infarction? From The Rational Clinical Examination
JAMA 2009; http://www.jamaevidence.com/content/3484335
Study: Systematic review of articles assessing the accuracy and precision of the clinical exam in the diagnosis of an acute myocardial infarction.
Results: In patients with normal or non-diagnostic ECG, no established CAD, and prolonged or recurrent chest pain typical of their usual discomfort, radiation of pain to the shoulder OR both arms had the highest positive likelihood ratio (+LR) of 4.1 (95% CI 2.5-6.5) and a negative likelihood ratio (-LR) of 0.68 (95% CI 0.52-0.89). Radiation to right arm had a +LR of 3.8 (95% CI 2.2-6.6) and –LR of 0.86 (95% CI 0.77-0.96), vomiting had a +LR of 3.5 (95% CI 2.0-6.2) and –LR of 0.87 (95% CI 0.79-0.97), while radiation to left arm only had a +LR of 1.3 (95% CI 0.93-1.8) and a –LR of 0.9 (95% CI 0.76-1.1).
Conclusions: The most compelling features that increase likelihood of an MI are ST-segment and cardiac enzyme elevation, new Q-wave, and presence of an S3 heart sound. In patients where the diagnosis of MI is uncertain, radiation of pain to the shoulder OR both arms, radiation to the right arm, and vomiting had the best predictive values, while radiation to the left arm is relatively non-diagnostic.

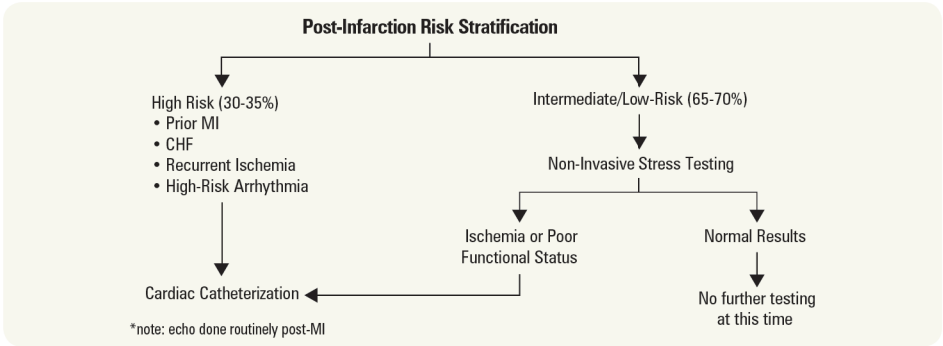


Figure 37. Post-MI risk stratification

Complications of MI

CRASH PAD
Cardiac **R**upture
Arrhythmia
Shock
Hypertension/**H**eart failure
Pericarditis/**P**ulmonary emboli
Aneurysm
DVT

Prognosis following STEMI

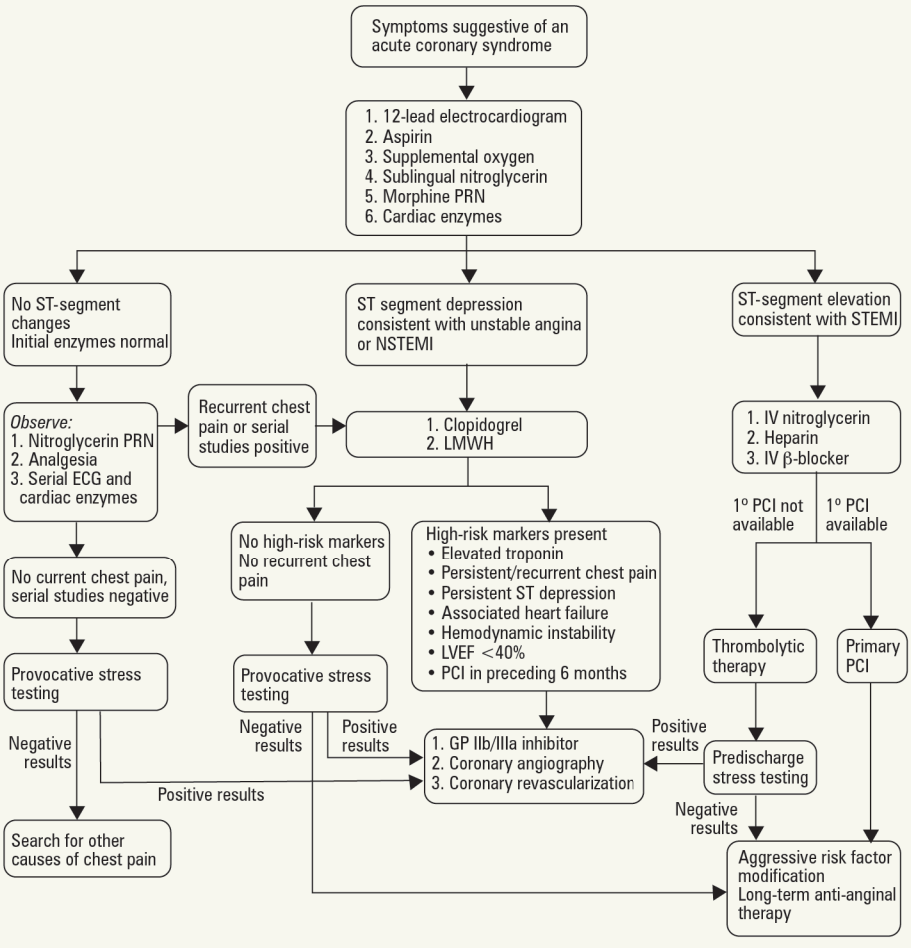
- 5-15% of hospitalized patients will die
  - risk factors
    - infarct size/severity
    - age
    - comorbid conditions
    - development of heart failure or hypotension
- post-discharge mortality rates
  - 6-8% within first year, half of these within first 3 mo
  - 4% per year following first yr
  - risk factors
    - LV dysfunction
    - residual myocardial ischemia
    - ventricular arrhythmias
    - history of prior MI

Resting LVEF is a useful prognostic factor

Table 9. Complications of Myocardial Infarction

Complication	Etiology	Presentation	Therapy
Arrhythmia			
1. Tachycardia	Sinus, AFib, VT, VFib	First 48 h	See *Arrhythmias*, C17
2. Bradycardia	Sinus, AV block	First 48 h	
Myocardial Rupture			
1. LV free wall	Transmural infarction	1-7 d	Surgery
2. Papillary muscle (→ MR)	Inferior infarction	1-7 d	Surgery
3. Ventricular septum (→ VSD)	Septal infarction	1-7 d	Surgery
Shock/CHF	Infarction or aneurysm	Within 48 h	Inotropes, intra-aortic balloon pump
Post-Infarct Angina	Persistent coronary stenosis Multivessel disease	Anytime	Aggressive medical therapy PCI or CABG
Recurrent MI	Reocclusion	Anytime	Aggressive medical therapy PCI or CABG
Thromboembolism	Mural/apical thrombus DVT	7-10 d, up to 6 mo	Anticoagulation
Pericarditis	Inflammatory	1-7 d	ASA
Dressler's Syndrome	Autoimmune	2-8 wk	

Treatment Algorithm for Chest Pain



ECG = electrocardiogram; LMWH = low-molecular-weight heparin; NSTEMI = non-ST-segment elevation myocardial infarction; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction

Figure 38. Treatment algorithm for patients with symptoms suggestive of an acute coronary syndrome
Adapted from: Andreoli TE, et al. Cecil Essentials of Medicine, 8th ed. 2011. Elsevier. With permission from Elsevier

Enoxaparin vs. Unfractionated Heparin with Fibrinolysis for ST-elevation Myocardial Infarction
NEJM 2006;354:1477-1488
Study: Prospective multicentre RCT.
Patients: 20,479 patients (median age 60 yr, 77% male) with STEMI who were scheduled to undergo fibrinolysis.
Intervention: Patients were randomized to receive either enoxaparin or weight based unfractionated heparin in addition to thrombolysis and standard therapies.
Primary Outcome: Death or recurrent nonfatal MI 30 d post-event.
Results: The composite primary outcome occurred less often in the enoxaparin group compared with those who received unfractionated heparin (9.9% vs. 12.0%, p<0.001, NNT=47). Taken separately, there was a trend toward reduced mortality (6.9% vs. 7.5%, p=0.11) and a significant reduction in nonfatal reinfarction (3.0% vs. 4.5%, p<0.001) in the enoxaparin group. The risk of major bleeding was significantly increased in the enoxaparin group (2.1% vs. 1.4%, p<0.001, NNH=142).
Conclusion: In patients with STEMI receiving thrombolysis, enoxaparin is superior to unfractionated heparin in preventing recurrent nonfatal MI and may lead to a small reduction in mortality.

Intensive vs. Moderate Lipid Lowering with Statins after Acute Coronary Syndromes
NEJM 2004;350:1495-1504
Study: Prospective, double blind, RCT; mean follow-up of 2 yr.
Population: 4,162 patients who had been hospitalized for an ACS within the preceding 10 d.
Intervention: Patients were randomized to receiving pravastatin 40 mg or atorvastatin 80 mg daily.
Primary Outcome: Composite of death from any cause, myocardial infarction, documented unstable angina requiring rehospitalization, revascularization (performed at least 30 d after randomization), and stroke.
Results: High dose atorvastatin was associated with a 16% hazard ratio reduction (p=0.005; 95% CI 5-26%) in the primary outcome compared to standard dose pravastatin.
Conclusions: In patients who recently experienced an ACS, high dose statin therapy provides greater protection against death and major cardiovascular events than standard dose therapy.

Sudden Cardiac Arrest

Definition
- unanticipated, non-traumatic cardiac death in a stable patient which occurs within 1 h of symptom onset; VFib is most common cause

Etiology
- primary cardiac pathology
  - ischemia/MI
  - LV dysfunction
  - severe ventricular hypertrophy
    - HCM
    - AS
  - congenital heart disease e.g. arrhythmogenic right ventricular dysplasia
  - mutations in cardiac ion channels e.g. long QT syndrome, Brugada syndrome

Management
- acute: resuscitate with prompt CPR and defibrillation
- investigate underlying cause (cardiac catheterization, electrophysiologic studies, echo)
- treat underlying cause
- antiarrhythmic drug therapy: amiodarone, β-blockers
- implantable cardioverter defibrillator (ICD)
- refer to ACLS guidelines (see Anesthesia, A33)

Coronary Revascularization

PERCUTANEOUS CORONARY INTERVENTION
- interventional cardiology technique aimed at relieving significant coronary stenosis
- main techniques: balloon angioplasty, stenting
- less common techniques: rotational/directional/extraction atherectomy

Indications
- medically refractory angina
- NSTEMI/UA with high risk features (e.g. high TIMI risk score)
- primary/rescue PCI for STEMI

Balloon Angioplasty and Intracoronary Stenting
- coronary lesions dilated with balloon inflation
- major complication is restenosis (approximately 15% at 6 mo), felt to be due to elastic recoil and neointimal hyperplasia
- majority of patients receive intracoronary stent(s) to prevent restenosis
  - bare metal stent (BMS) versus drug-eluting stents: PRAMI trial demonstrated stenting non-culprit lesions results in 14% absolute risk reduction of cardiac death, nonfatal MI, or refractory angina
    - coated with antiproliferative drugs (sirolimus, paclitaxel, everolimus)
    - reduced rate of neointimal hyperplasia and restenosis compared to BMS (5% vs. 20%)
    - complication: late stent thrombosis (5 events per 1,000 stents implanted)

Adjunctive Therapies
- ASA and heparin decrease post-procedural complications
- further reduction in ischemic complications has been demonstrated using GPIIb/IIIa inhibitors (abciximab, eptifibatide, tirofiban) in coronary angiography and stenting
- following stent implantation
  - dual antiplatelet therapy (ASA and clopidogrel) for 1 mo with BMS or ≥12 mo with DES
  - DAPT study showed benefit of dual antiplatelet therapy beyond 12 mo
  - ASA and prasugrel can be considered for those at increased risk of stent thrombosis

Procedural Complications
- mortality and emergency bypass rates <1%
- nonfatal MI: approximately 2-3%

CORONARY ARTERY BYPASS GRAFT SURGERY
- objective of CABG is complete reperfusion of the myocardium

TIMI Risk Score for UA/NSTEMI

Characteristics	Points
Historical	
Age ≥65 yr	1
≥3 risk factors for CAD	1
Known CAD (stenosis ≥50%)	1
Aspirin® use in past 7 d	1
Presentation	
Recent (≤24 h) severe angina	1
ST-segment deviation ≥0.5 mm	1
Increased cardiac markers	1

Risk Score = Total Points
If TIMI risk score ≥3, consider early LMWH and angiography
TIMI = thrombolysis in myocardial infarction
UA = unstable angina

JAMA 2000;284:835-842

Treatment of NSTEMI

BEMOAN
β-blocker
Enoxaparin
Morphine
O_2
ASA
Nitrates

Indications

- CABG
  - ≥50% diameter stenosis in the left main coronary artery
  - ≥70% diameter stenosis in three major coronary arteries
  - ≥70% diameter stenosis in the proximal LAD artery plus one other major coronary artery
  - survivors of sudden cardiac arrest with presumed ischemia-mediated VT caused by significant (≥70% diameter) stenosis in a major coronary artery
- other
  - ≥70% diameter stenosis in two major coronary arteries (without proximal LAD disease) and evidence of extensive ischemia
  - ≥70% diameter stenosis in the proximal LAD artery and evidence of extensive ischemia
  - multivessel CAD in patients with diabetes
  - LV systolic dysfunction (LVEF 35% to 50%) and significant multivessel CAD or proximal LAD stenosis where viable myocardium is present in the region of intended revascularization
- PCI
  - UA/NSTEMI if not a CABG candidate
  - STEMI when PCI can be performed more rapidly and safely than CABG
- CABG or PCI
  - one or more significant (≥70% diameter) coronary artery stenosis amenable to revascularization and unacceptable angina despite medical therapy

Table 10. Choice of Revascularization Procedure

	PCI	CABG
Advantages	Less invasive technique Decreased periprocedural morbidity and mortality Shorter periprocedural hospitalization	Greater ability to achieve complete revascularization Decreased need for repeated revascularization procedures
Indications	Single or double-vessel disease Inability to tolerate surgery	Triple-vessel or left main disease DM Plaque morphology unfavourable for PCI

Table 11. Conduits for CABG

Graft	Occlusion/Patency Rate	Considerations
Saphenous Vein Grafts (SVG)	At 10 yr, 50% occluded, 25% stenotic, 25% angiographically normal	Used when arterial grafts are not available or many grafts are required, such as triple or quadruple bypass
Left Internal Thoracic/Mammary Artery (LITA/LIMA) (LIMA to LAD)	90-95% patency at 15 yr	Most preferred option because of excellent patency Improved event-free survival (angina, MI) Decreased late cardiac events No increase in operative risk
Right Internal Thoracic/Mammary Artery (RITA/RIMA)	Pedicled RIMA patency comparable to LIMA Free RIMA patency less	Used in bilateral ITA/IMA grafting Patients receiving bilateral ITAs/IMAs have less risk of recurrent angina, late MI, angioplasty
Radial Artery (free graft)	85-90% patency at 5 yr	Prone to severe vasospasm post-operatively due to muscular wall
Right Gastroepiploic Artery	80-90% patency at 5 yr	Primarily used as an *in situ* graft to bypass the RCA Use limited because of the fragile quality of the artery, other technical issues, increased operative time (laparotomy incision), and incisional discomfort with associated ileus
Complete Arterial Revascularization		For younger patients (<60 yr of age) Is preferred due to longer term graft patency
Redo Bypass Grafting		Operative mortality 2-3x higher than first operation 10% perioperative MI rate Reoperation undertaken only in symptomatic patients who have failed medical therapy and in whom angiography has documented progression of the disease Increased risk with redo-sternotomy secondary to adhesions which may result in laceration to aorta, RV, IMA/ITA, and other bypass grafts

Percutaneous Coronary Intervention vs. Coronary-Artery Bypass Grafting for Severe Coronary Artery Disease: The SYNTAX Trial
NEJM 2009;360;961-972
Study: Prospective RCT.
Population: 1,800 patients with untreated three-vessel or left main coronary artery disease and anatomically equivalent for both Percutaneous Intervention (PCI) and Coronary Artery Bypass Graft (CABG).
Intervention: PCI vs. CABG.
Outcome: Composite of death from any cause, stroke, MI, or repeat revascularization in 12 mo post-intervention.
Results: Incidence of primary outcome was lower in the CABG intervention vs. PCI (12.4% vs. 17.8%, p=0.002, NNT=19). PCI was associated with significantly higher rates of repeat revascularization (13.5% vs. 5.9%, p<0.001) and cardiac death (3.7% vs. 2.1%, p=0.05), while CABG had higher rates of stroke (2.2% vs. 0.6%, p=0.03).
Conclusions: In patients with three-vessel or left main coronary artery disease CABG is superior to PCI in preventing major adverse cardiovascular and cerebrovascular events within 12 mo of intervention.

Operative Issues
- left ventricular (LV) function is an important determinant of outcome of all heart diseases
- patients with severe LV dysfunction usually have poor prognosis, but surgery can sometimes dramatically improve LV function
- assess viability of non-functioning myocardial segments in patients with significant LV dysfunction using delayed thallium myocardial imaging, stress echocardiography, PET scanning, or MRI

CABG and Antiplatelet Regimens
- please refer to CCS guidelines – 2012 update on antiplatelet therapy – for more information if possible
- prior to CABG, clopidogrel and ticagrelor should be discontinued for 5 d and prasugrel for 7 d before surgery
- dual antiplatelet therapy should be continued for 12 mo in patients with ACS within 48-72 h after CABG
- ASA (81 mg) continued indefinitely (can be started 6 h after surgery)
- patients requiring CABG after PCI should continue their dual antiplatelet therapy as recommended in the post-PCI guidelines

Table 12. Risk Factors for CABG Mortality and Morbidity (decreasing order of significance)

Risk Factors for CABG Mortality	Risk Factors for CABG Post-Operative Morbidity or Increased Length of Stay
Urgency of surgery (emergent or urgent)	Reoperation
Reoperation	Emergent procedure
Older age	Pre-operative intra-aortic balloon pump (IABP)
Poor left ventricular function (see below)	CHF
Female gender	CABG + valve surgery
Left main disease	Older age
Others include catastrophic conditions (cardiogenic shock, ventricular septal rupture, ongoing CPR), dialysis-dependent renal failure, end-stage COPD, DM, cerebrovascular disease, and peripheral vascular disease	Renal dysfunction
	COPD
	DM
	Cerebrovascular disease

Procedural Complications
- CABG using cardiopulmonary bypass (CPB)
  - stroke and neurocognitive defects (microembolization of gaseous and particulate matter)
  - immunosuppression
  - systemic inflammatory response leading to
    - myocardial dysfunction
    - renal dysfunction
    - neurological injury
    - respiratory dysfunction
    - coagulopathies

OFF-PUMP CORONARY ARTERY BYPASS SURGERY

Procedure
- avoids the use of CPB by allowing surgeons to operate on a beating heart
  - stabilization devices (e.g. Genzyme Immobilizer®) hold heart in place allowing operation while positioning devices (Medtronic Octopus® and Starfish® system) allow the surgeon to lift the beating heart to access the lateral and posterior vessels
  - procedure is safe and well tolerated by most patients; however, this surgery remains technically more demanding

Indications
- used in poor candidates for CPB who have: calcified aorta, poor LVEF, severe peripheral vascular disease (PVD), severe COPD, chronic renal failure, coagulopathy, transfusion objections (e.g. Jehovah's Witness), good target vessels, anterior/lateral wall revascularization, target revascularization in older, sicker patients
- **absolute contraindications**: hemodynamic instability, poor quality target vessels including intramyocardial vessels, diffusely diseased vessels, and calcified coronary vessels
- **relative contraindications:** cardiomegaly/CHF, critical left main disease, small distal targets, recent or current acute MI, cardiogenic shock, LVEF <35%

Outcomes
- OPCAB decreases in-hospital morbidity (decreased incidence of chest infection, inotropic requirement, supraventricular arrhythmia), blood product transfusion, ICU stay, length of hospitalization, and CK-MB and troponin I levels
- no significant difference in terms of survival at 2 yr, frequency of cardiac events (MI, PCI, CHF, recurrent angina, redo CABG), or medication usage compared to on-pump CABG

Safety and Efficacy of Drug-Eluting and Bare Metal Stents
Circulation 2009;119;3198-3206
Study: Meta-analysis of RCTs and observational studies. 22 RCTs and 34 observational studies.
Population: 9,470 and 182,901 patients in RCTs and observational studies respectively who underwent percutaneous coronary intervention.
Intervention: Drug-Eluting Stents (DES) versus Bare Metal Stents (BMS).
Outcome: All-cause mortality, myocardial infarction (MI), and target vessel revascularization (TVR).
Results: No difference in mortality was found between DES vs. BMS by RCTs, while observational studies showed significantly lower mortality rates in DES-treated patients (hazard ratio (HR) 0.78, $p<0.001$). No difference in MI incidence was found in RCTs, while lower incidences of MI were found in observational studies (HR 0.87, $p=0.014$). DES has a significantly lower TVR rate in both RCT (HR 0.45, $p<0.001$) and observational studies (HR 0.46, $p<0.001$).
Conclusions: DES significantly reduces rates of TVR compared to BMS. Although there is no difference in mortality or MI incidence as found by RCTs, observational studies suggest lowered mortality and MI rates in patients with DES over BMS.

Heart Failure

- see also CCS Heart Failure Guidelines 2012 for details (free mobile apps available on iOS and Android platforms in the CCS app stores) as well as the CCS Heart Failure Guidelines Compendium available at CCS.ca

Congestive Heart Failure

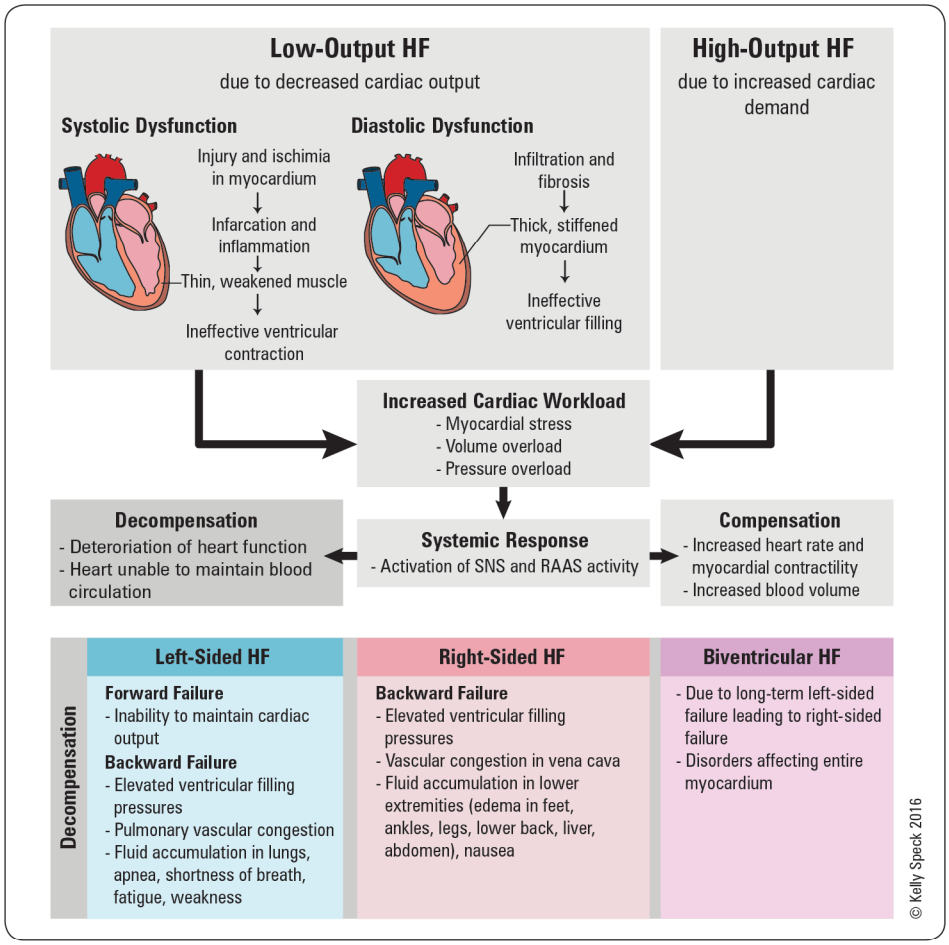


Figure 39. Congestive Heart Failure

Table 13. Signs and Symptoms of Left vs. Right Heart Failure

	Left Failure	Right Failure
Low Cardiac Output (Forward)	Fatigue Syncope Systemic hypotension Cool extremities Slow capillary refill Peripheral cyanosis Pulsus alternans Mitral regurgitation S3	Left failure symptoms if decreased RV output leads to LV underfilling Tricuspid regurgitation S3 (right-sided)
Venous Congestion (Backward)	Dyspnea, orthopnea, PND Cough Crackles	Peripheral edema Elevated JVP with abdomincjugular reflux, and Kussmaul's sign Hepatomegaly Pulsatile liver

Does this Dyspneic Patient in the Emergency Department have Congestive Heart Failure?
JAMA 2005;294:1944-1956

	LR + (95% CI)	LR – (95% CI)
Initial clinical judgment	4.4 (1.8-10.0)	0.45 (0.28-0.73)
Past Medical History		
Heart failure	5.8 (4.1-8.0)	0.45 (0.38-0.53)
Myocardial infarction	3.1 (2.0-4.9)	0.69 (0.58-0.82)
Coronary artery disease	1.8 (1.1-2.8)	0.68 (0.48-0.96)
Symptoms		
Paroxysmal nocturnal dyspnea	2.6 (1.5-4.5)	0.7 (0.54-0.91)
Orthopnea	2.2 (1.2-3.9)	0.65 (0.45-0.92)
Dyspnea on exertion	1.3 (1.2-1.4)	0.48 (0.35-0.67)
Physical Exam		
Third heart sound	11 (4.9-25)	0.88 (0.83-0.94)
Jugular venous distension	5.1 (3.2-7.9)	0.66 (0.57-0.77)
Rales	2.8 (1.9-4.1)	0.51 (0.37-0.70)
Lower extremity edema	2.3 (1.5-3.7)	0.64 (0.47-0.87)
Chest Radiograph		
Pulmonary venous congestion	12 (6.8-21)	0.48 (0.28-0.83)
Interstitial edema	12 (5.2-27)	0.68 (0.54-0.85)
Cardiomegaly	3.3 (2.4-4.7)	0.33 (0.23-0.48)
ECG		
Atrial fibrillation	3.8 (1.7-8.8)	0.79 (0.65-0.96)
Any abnormal finding	2.2 (1.6-3.1)	0.64 (0.47-0.88)

Dichotomies of Heart Failure
- Forward vs. backward
- Left-sided vs. right-sided
- Systolic vs. diastolic dysfunction
- Low output vs. high output

Use Ejection Fraction to Grade LV Dysfunction
- Grade I (EF >60%) (Normal)
- Grade II (EF = 40-59%)
- Grade III (EF = 21-39%)
- Grade IV (EF ≤20%)

Pathophysiology
- most common causes are ischemic heart disease, hypertension and valvular heart disease
- myocardial insult causes pump dysfunction/impaired filling leading to myocardial remodelling
  - pressure overload (e.g. AS or HTN) leads to compensatory hypertrophy (concentric remodelling) and eventually interstitial fibrosis
  - volume overload (e.g. AI) leads to dilatation (eccentric remodelling)
    - both processes lead to maladaptive changes contributing to disease process
- results in decreased volume cardiac output resulting in activation of the SNS and RAAS
- Na^+ and water retention, increasing preload and afterload, tachycardia
  - perpetuates cycle of increasing cardiac demand and decompensation

Heart Failure with Reduced Ejection Fraction
- impaired myocardial contractile function → decreased LVEF and SV → decreased CO

Volume Overload and Eccentric Remodeling is the Typical Phenotype
- findings: apex beat displaced, S3, cardiothoracic ratio >0.5, decreased LVEF, LV dilatation
- causes
  - ischemic (e.g. extensive CAD, previous MI)
  - non-ischemic
    - HTN
    - DM
    - alcohol (and other toxins)
    - myocarditis
    - dilated cardiomyopathy (multiple causes – see *Dilated Cardiomyopathy*, C42)

Heart Failure with Preserved Ejection Fraction
- previously known as "diastolic heart failure "
- concentric remodeling with a "stiff" left ventricle is the typical phenotype
- 1/2 of patients with heart failure have preserved EF; confers similar prognosis to HRrEF; more common in the elderly and females
- reduced LV compliance causes increased LV filling pressures, increased LA pressure/volume, and pulmonary congestion
- findings: HTN, apex beat sustained, S4, normal-sized heart on CXR, LVH on ECG/echo, normal EF
- causes
  - transient: ischemia (relaxation of myocardium is active and requires ATP)
  - permanent
    - severe hypertrophy (HTN, aortic stenosis, HCM)
    - restrictive cardiomyopathy (e.g. amyloid)
    - MI

High-Output Heart Failure
- caused by demand for increased cardiac output
- often exacerbates existing heart failure or decompensates a patient with other cardiac pathology
- differential diagnosis: anemia, thiamine deficiency (beriberi), hyperthyroidism, A-V fistula or L-R shunting, Paget's disease, renal disease, hepatic disease

Precipitants of Symptomatic Exacerbations
- consider natural progression of disease vs. new precipitant
- always search for reversible cause
- differential diagnosis can also be organized as follows:
  - new cardiac insult/disease: MI, arrhythmia, valvular disease
  - new demand on CV system: HTN, anemia, thyrotoxicosis, infection, etc.
  - medication non-compliance
  - dietary indisretion e.g. salt intake
  - obstructive sleep apnea

Investigations
- identify and assess precipitating factors and treatable causes of CHF
- blood work: CBC, electrolytes (including calcium and magnesium), BUN, creatinine, fasting blood glucose, HbA1c, lipid profile, liver function tests, serum TSH ± ferritin, BNP, uric acid
- ECG: look for chamber enlargement, arrhythmia, ischemia/infarction
- CXR: cardiomegaly, pleural effusion, redistribution, Kerley B lines, bronchiolar-alveolar cuffing
- echo: systolic function (LVEF), diastolic function (E/A ratio, E/e'), cardiac dimensions, wall motion abnormalities, RVSP (from TR jet), valvular disease, pericardial effusion
- radionuclide angiography: LVEF
- myocardial perfusion scintigraphy (thallium or sestamibi SPECT)

A Validated Clinical and Biochemical Score for the Diagnosis of Acute Heart Failure: the ProBNP Investigation of Dyspnea in the Emergency Department (PRIDE) Acute Heart Failure Score
Am Heart J 2006;151:48-54

Predictor	Possible Score
Age >75 yr	1
Orthopnea present	2
Lack of cough	1
Current loop diuretic use (before presentation)	1
Rales on lung exam	1
Lack of fever	2
Elevated NT-proBNP (>450 pg/mL if <50 yr, >900 pg/mL if >50 yr)	4
Interstitial edema on chest x-ray	2
Total	/14
Likelihood of heart failure Low = 0-5 Intermediate = 6-8 High = 9-14	

Brain natieuretic peptide (BNP) is secreted by ventricles due to LV stretch and wall tension. Cardiomyocytes secrete BNP precursor that is cleaved into proBNP. After secretion into ventricles, proBNP is cleaved into the active C-terminal portion and the inactive NT-proBNP. The above scoring algorithm developed by Baggish et al. is commonly used. A score of <6 has a negative predictive value of 98%, while scores ≥6 had a sensitivity of 96% and specificity of 84% ($p<0.001$) for the diagnosis of acute heart failure.

New York Heart Association (NYHA) Functional Classification of Heart Failure
- **Class I:** ordinary physical activity does not cause symptoms of HF
- **Class II:** comfortable at rest, ordinary physical activity results in symptoms
- **Class III:** marked limitation of ordinary activity; less than ordinary physical activity results in symptoms
- **Class IV:** inability to carry out any physical activity without discomfort; symptoms may be present at rest

Five Most Common Causes of CHF
- CAD (60-70%)
- HTN
- Idiopathic (often dilated cardiomyopathy)
- Valvular (e.g. AS, AR, and MR)
- Alcohol (dilated cardiomyopathy)

Precipitants of Heart Failure

HEART FAILED
Hypertension (common)
Endocarditis/environment (e.g. heat wave)
Anemia
Rheumatic heart disease and other valvular disease
Thyrotoxicosis
Failure to take meds (very common)
Arrhythmia (common)
Infection/**I**schemia/**I**nfarction (common)
Lung problems (PE, pneumonia, COPD)
Endocrine (pheochromocytoma, hyperaldosteronism)
Dietary indiscretions (common)

Acute Treatment of Pulmonary Edema

- treat acute precipitating factors (e.g. ischemia, arrhythmias)
- **L** – Lasix® (furosemide) 40-500 mg IV
- **M** – morphine 2-4 mg IV: decreases anxiety and preload (venodilation)
- **N** – nitroglycerin: topical/IV/SL - use with caution in preload-dependent patients (e.g. right HF or RV infarction) as it may precipitate CV collapse
- **O** – oxygen: in hypoxemic patients
- **P** – positive airway pressure (CPAP/BiPAP): decreases preload and need for ventilation when appropriate
- **P** – position: sit patient up with legs hanging down unless patient is hypotensive
- in ICU setting or failure of LMNOPP, other interventions may be necessary
  - nitroprusside IV
  - hydralazine PO
  - sympathomimetics
    - dopamine
      - low dose: selective renal vasodilation (high potency D1 agonist)
      - medium dose: inotropic support (medium potency β1 agonist)
      - high dose: increases SVR (low potency β1 agonist), which is undesirable
    - dobutamine
      - β1-selective agonist causing inotropy, tachycardia, hypotension (low dose) or hypertension (high dose); most serious side effect is arrhythmia, especially AF
    - phosphodiesterase inhibitors (milrinone)
      - inotropic effect and vascular smooth muscle relaxation (decreased SVR), similar to dobutamine
- consider pulmonary artery catheter to monitor pulmonary capillary wedge pressure (PCWP) if patient is unstable or a cardiac etiology is uncertain (PCWP >18 indicates likely cardiac etiology)
- mechanical ventilation as needed
- rarely used, but potentially life-saving measures:
  - intra-aortic balloon pump (IABP) - reduces afterload via systolic unloading and improves coronary perfusion via diastolic augmentation
  - left or right ventricular assist device (LVAD/RVAD)
  - cardiac transplant

Long-Term Management

- overwhelming majority of evidence-based management applies to HFREF
- currently no proven pharmacologic therapies shown to reduce mortality in HFPEF; controlcontrol risk factors (e.g. hypertension)

Conservative Measures

- symptomatic measures: oxygen in hospital, bedrest, elevate the head of bed
- lifestyle measures: diet, exercise, DM control, smoking cessation, decrease alcohol consumption, patient education, sodium and fluid restriction
- multidisciplinary heart failure clinics: for management of individuals at higher risk, or with recent hospitalization

Non-Pharmcological Management

- from CCS guidelines (2013 update)
- cardiac rehabilitation: participation in a structured exercise program for NYHA class I-III after clinical status assessment to improve quality of life (HF-ACTION trial)

Pharmacological Therapy

1.Renin-angiotensin-aldosterone blockade

- ACEI: standard of care – slows progression of LV dysfunction and improves survival
  - all symptomatic patients functional class II-IV
  - all asymptomatic patients with LVEF <40%
  - post-MI
- angiotensin II receptor blockers
  - second-line to ACEI if not tolerated, or as adjunct to ACEI if β-blockers not tolerated
    - combination with ACEI is not routinely recommended and should be used with caution as it may precipitate hyperkalemia, renal failure, the need for dialysis and increase (CHARM, ONTARGET)
  - combination angiotensin II receptor blockers with neprilysin inhibitors (ARNI) is a new class of medication that has morbidity and mortality benefit over ACE inhibitor alone; this may become standard first line therapy

2. β-blockers: slow progression and improve survival

- class I-III with LVEF <40%
- stable class IV patients
- carvedilol improves survival in class IV HF (COMET)
- note: should be used cautiously, titrate slowly because may initially worsen CHF

The most common cause of right heart failure is left heart failure

Measuring NT-Pro BNP

BNP is secreted by ventricles due to LV stretch and wall tension

Cardiomyocytes secrete BNP precursor that is cleaved into proBNP

After secretion into ventricles proBNP is cleaved into the active C-terminal portion and the inactive NT-proBNP portion

	NT-proBNP levels (pg/mL)
Age	HF very likely
<50	>450
50-75	>900
>75	>1800

Limitations: Age, body habitus, renal function, pulmonary embolism

Features of Heart Failure on CXR

HERB-B
Heart enlargement (cardiothoracic ratio >0.50)
Pleural **E**ffusion
Re-distribution (alveolar edema)
Kerley **B** lines
Bronchiolar-alveolar cuffing

Patients on β-blocker therapy who have acute decompensated heart failure should continue β-blockers where possible (provided they are not in cardiogenic shock or in severe pulmonary edema)

Can the Clinical Examination Diagnose Left-Sided Heart Failure in Adults?
From The Rational Clinical Examination
JAMA 2009; http://www.jamaevidence.com/content/3478992
Study: Systematic review of articles assessing the accuracy and precision of the clinical exam in the diagnosis of CHF.
Results: The diagnosis of left ventricular dysfunction in patient after an MI based on the presence of radiographic pulmonary venous congestion with edema, rales one-third up the lung fields in the absence of a chronic pulmonary disease, or a 3rd heart sound had a positive likelihood ratio (+LR) of 3.1 (95% CI 1.7-5.8) and a negative likelihood ratio (-LR) of 0.62 (95% CI 0.46-0.83). In inpatients the combination of clinical findings, ECG, and CXR had a +LR of 2.0 (95% CI 1.6-2.5) and a –LR of 0.41 (95% CI 0.30-0.56). Female sex (+LR, 1.6 [95% CI 1.2-2.2]) and sBP ≥160 mmHg (+LR, 1.8 [95% CI 1.3-2.6]) were most indicative for diastolic dysfunction. Heart rate ≥100/min (+LR 0.43 [95% CI 0.28-0.65]) and left atrial ECG abnormality (+LR 0.42 [95% CI 0.26-0.63]) were most indicative for systolic dysfunction.
Conclusions: Patients with signs, symptoms, and risk factors for systolic dysfunction should receive an ECG and CXR. Female sex and sBP ≥160 mmHg are suggestive of diastolic dysfunction; heart rate ≥100/min and left atrial ECG abnormality suggest systolic dysfunction.

3. **Mineralocorticoid receptor (aldosterone) antagonists:** mortality benefit in symptomatic heart failure and severely depressed ejection fraction
   - spironolactone or eplerenone symptomatic heart failure in patients already on ACEI, beta blocker and loop diuretic
   - note: potential for life threatening hyperkalemia
     - monitor K^+ after initiation and avoid if Cr >220 μmol/L or K^+ >5.2 mmol/L

4. **Diuretics:** symptom control, management of fluid overload
   - furosemide (40-500 mg daily) for potent diuresis
   - metolazone may be used with furosemide to increase diuresis
   - furosemide, metolazone, and thiazides oppose the hyperkalemia that can be induced by β-blockers, ACEI, ARBs, and aldosterone antagonists

5. **Digoxin and cardiac glycosides:** digoxin improves symptoms and decreases hospitalizations, no effect on mortality
   - indications: patient in sinus rhythm and symptomatic on ACEI, or CHF and AFib
   - patients on digitalis glycosides may worsen if these are withdrawn

6. **Antiarrhythmic drugs:** for use in CHF with arrhythmia
   - can use amiodarone, β-blocker, or digoxin

7. **Anticoagulants:** warfarin for prevention of thromboembolic events
   - prior thromboembolic event or AFib, presence of LV thrombus on echo

Procedural Interventions

- resynchronization therapy: symptomatic improvement with biventricular pacemaker
- consider if QRS >130 msec, LVEF <35%, and persistent symptoms despite optimal therapy
- greatest benefit likely with marked LV enlargement, mitral regurgitation, QRS >150 msec,
- ICD: mortality benefit in 1° prevention of sudden cardiac death
  - prior MI, optimal medical therapy, LVEF <30%, clinically stable
  - prior MI, non-sustained VT, LVEF 30-40%, EPS inducible VT
- LVAD/RVAD (see *Ventricular Assist Devices*, C40)
- cardiac transplantation (see *Cardiac Transplantation*, C40)
- valve repair if patient is surgical candidate and has significant valve disease contributing to CHF (see *Valvular Heart Disease*, C44)

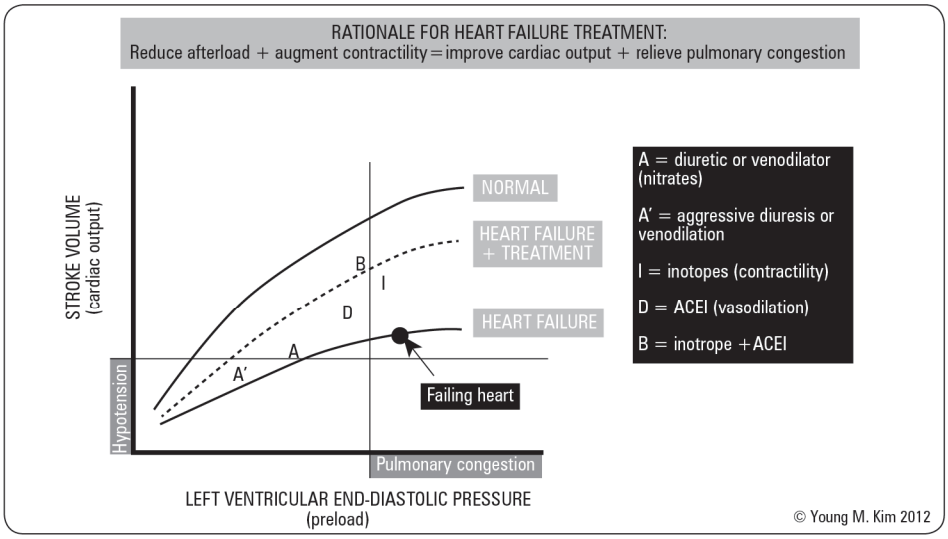


Figure 40. Effect of heart failure treatment on the Frank-Starling curve

Sleep-Disordered Breathing

- 45-55% of patients with CHF have sleep disturbances, including Cheyne-Stokes breathing and sleep apnea (central or obstructive)
- associated with a worse prognosis and greater LV dysfunction
- nasal continuous positive airway pressure (CPAP) is effective in treating symptoms of sleep apnea with secondary beneficial effects in cardiac function and symptoms

Chronic Treatment of CHF
- ACEI*
- β-blockers*
- ± Mineralocorticoid receptor antagonists*
- Diuretic
- ± Inotrope
- ± Antiarrhythmic
- ± Anticoagulant

*Mortality benefit

Influence of Ejection Fraction on Cardiovascular Outcomes in a Broad Spectrum of Heart Failure Patients
Circulation 2005;112:3738-3744
Purpose: Understand the relationship between ejection fractions and cardiovascular risk in patients with heart failure.
Methods: 7,599 patients from the CHARM study (Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity; RCT comparing placebo vs. candesartan in patients with NYHA class II to IV). Compared LVEF to cardiovascular outcomes and causes of death.
Results: All-cause mortality increased by 39% per 10% reduction in LVEF below 45% (Hazard ratio 1.39, 95% CI 1.32-1.46). For LVEF>45%, ejection fraction does not further contribute to assessment of cardiovascular risk in HF patients.
Conclusions: At LVEF<45%, lower ejection fractions were associated with poorer cardiovascular outcomes.

LVEF	CHF Hospitalization	All-Cause Mortality
≤22%	14.9%	15.4%
23-32%	10.9%	10.8%
33-42%	7.2%	7.4%
43-52%	5.7%	5.2%
>52%	6.9%	5.7%

Higher New York Heart Association Classes and Increased Mortality and Hospitalization in Patients with Heart Failure and Preserved Left Ventricular Function
Am Heart J 2006;151:444-450
Purpose: To establish the association between NYHA class and outcomes with heart failure and preserved systolic function.
Methods: Retrospective follow-up study (median 38.5 mo) of 988 patients with heart failure with ejection fracture >45%. Estimated risks of various outcomes using Cox proportional hazard models.
Results: Adjusted hazard ratio for all-cause mortality for NYHA class II, III, IV patients was 1.54, 2.56, and 8.46, respectively. Adjusted hazard ratio for all-cause hospitalization for NYHA class II, III, IV patients was 1.23, 1.71, and 3.4, respectively.
Conclusions: Higher NYHA classes were associated with poorer outcomes in patients with heart failure and preserved systolic function. Proportions of NYHA I, II, III, and IV patients who died of all causes during the study were 14.3%.

NYHA	Proportion of All-Cause Hospitalization	Proportion of All-Cause Mortality
I	60.7%	14.3%
II	65.2%	21.3%
III	77.7%	35.9%
IV	75.0%	58.3%

Cardiac Transplantation

- treatment for end-stage heart disease; due to ischemic or non-ischemic cardiomyopathy
- worldwide 1 yr survival is 85-90%, 5 yr survival about 60%, annual mortality rate of 4%
- matching is according to blood type, body size and weight (should be within 25%), and HLA tissue matching (if time allows)

Indications for Surgery

- severe cardiac disability despite maximal medical therapy (e.g. recurrent hospitalizations for CHF, NYHA III or IV, peak metabolic oxygen consumption <14 mL/kg/min in absence of β-blocker)
- symptomatic cardiac ischemia refractory to conventional treatment (e.g. unstable angina not amenable to CABG or PCI with LVEF <30%; recurrent, symptomatic ventricular arrhythmias)
- exclusion of all surgical alternatives to cardiac transplantation

Prerequisites

- psychosocial stability
- medically compliant and motivated
- relative contraindications: incurable malignancy, major systemic illness, irreversible major organ disease, active systemic infection, obesity, irreversible pulmonary HTN (pulmonary vascular resistance [PVR] >6 Wood units), severe COPD (FEV1 <1 L) or active drug addiction or alcoholism

Complications

- rejection
  - common, <5% have serious hemodynamic compromise
  - gold standard to detect rejection: endomyocardial biopsy
  - risk of acute rejection is greatest during the first 3 mo after transplant
- infection
  - leading cause of morbidity and mortality after cardiac transplantation
  - risk peaks early during the first few months after transplantation and then declines to a low persistent rate
- allograft CAD
  - approximately 50% develop graft CAD within 5 yr of transplantation
  - most common cause of late death following transplantation
- malignancy
  - develops in 15% of cardiac transplant recipients due to immunosuppressive medication
  - second most common cause of late death following transplantation
  - cutaneous neoplams most common, followed by non-Hodgkin's lymphoma and lung cancer
- immunosuppressive medication side effects (prednisone, cyclosporine, tacrolimus, sirolimus)

Effects of Donor Pre-Treatment with Dopamine on Survival After Heart Transplantation: A Cohort Study of Heart Transplant Recipients Nested in a Randomized Controlled Multicentre Trial
J Am Coll Cardiol 2010;58:1768-1777
Treatment of brain-dead donors with dopamine of 4 μg/kg/min will not harm cardiac allografts but appears to improve the clinical course of the heart allograft recipient.

Ventricular Assist Devices

- work to unload the ventricle while maintaining output; also results in decreased myocardial oxygen consumption permitting recovery of the myocardium that is not irreversibly injured
- can support the left (LVAD), right (RVAD), or both ventricles (BiVAD)
- indications
  - bridge to transplantation, bridge to decision (for transplant), or long term permanent therapy ("destination therapy")
  - post-operative mechanical support when unable to separate from cardiopulmonary bypass despite inotropic and intra-aortic balloon pump (IABP) support
    - IABP is a catheter based device inserted into the femoral artery and advanced to the descending aorta that decreases myocardial O_2 demand and increases blood flow to coronary arteries
    - inflation of the balloon occurs during diastole to increase ascending aorta and coronary artery perfusion pressure; deflation occurs at systole to reduce intra-aortic pressure thus reducing afterload
- post-operative cardiogenic shock

REMATCH Trial
NEJM 2001;345:1435-1443
Increased survival of 23% vs. 8% with LVAD vs. medical management of heart failure after 2 yr. Heartmate VAD has a biologic surface therefore does not require long-term anticoagulation but higher risk of infection

Canadian Cardiovascular Society Focused Position Statement Update on Assessment of the Cardiac Patient for Fitness to Drive: Fitness following Left Ventricular Assist Device Implantation
Can J Cardiol 2012;28:137-140
Patients with a continuous flow, NYHA class I-III, LVAD that are stable 2 mo post LVAD implantation qualify for private driving only and are disqualified from commercial driving.

Myocardial Disease

Definition of Cardiomyopathy
- intrinsic or primary myocardial disease not secondary to congenital, hypertensive, coronary, valvular, or pericardial disease
- functional classification: dilated, hypertrophic, or restrictive
- LV dysfunction 2° to MI often termed "ischemic cardiomyopathy", is not a true cardiomyopathy (i.e. primary myocardial disorder) since the primary pathology is obstructive CAD

Cardiomyopathy

HARD
Hypertrophic cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Restrictive cardiomyopathy
Dilated cardiomyopathy

Table 14. Summary Table for CHF and Myocardial Disease

Heart Failure Reduced Ejection Fraction		Heart Failure Preserved Ejection Fraction		
Dilated Cardiomyopathy	**Secondary Causes**	**Hypertrophic Cardiomyopathy**	**Restrictive Cardiomyopathy**	**Secondary Causes**
Idiopathic, infectious (e.g. myocarditis), alcohol, familial, collagen vascular disease, etc.	CAD, MI, DM, valvular (e.g. AR, MR)	Genetic disorder affecting cardiac sarcomeres (most common cause of sudden cardiac death in young athletes)	Amyloidosis, sarcoidosis, scleroderma, hemochromatosis, Fabry's, Pompe's Disease, Loeffler's, etc.	HTN, DM, valvular (e.g. AS), post-MI, transiently by ischemia, etc.

Myocarditis

Definition
- inflammatory process involving the myocardium ranging from acute to chronic; an important cause of dilated cardiomyopathy

Etiology
- idiopathic
- infectious
  - viral (most common): parvovirus B19, influenza, coxsackie B, echovirus, poliovirus, HIV, mumps
  - bacterial: *S. aureus, C. perfringens, C. diphtheriae, Mycoplasma, Rickettsia*
  - fungi
  - spirochetal (Lyme disease – *Borrelia burgdorferi*)
  - Chagas disease (*Trypanosoma cruzi*), toxoplasmosis
- toxic: catecholamines, chemotherapy, cocaine
- hypersensitivity/eosinophilic: drugs (antibiotics, diuretics, lithium, clozapine), insect/snake bites
- systemic diseases: collagen vascular diseases (SLE, rheumatoid arthritis, others), sarcoidosis, autoimmune
- other: giant cell myocarditis, acute rheumatic fever

Signs and Symptoms
- constitutional symptoms
- acute CHF - dyspnea, tachycardia, elevated JVP
- chest pain – due to pericarditis or cardiac ischemia
- arrhythmias
- systemic or pulmonary emboli
- pre-syncope/syncope/sudden death

Investigations
- ECG: non-specific ST-T changes ± conduction defects
- blood work
  - increased CK, troponin, LDH, and AST with acute myocardial necrosis ± increased WBC, ESR, ANA, rheumatoid factor, complement levels
  - blood culture, viral titres and cold agglutinins for Mycoplasma
- CXR: enlarged cardiac silhouette
- echo: dilated, hypokinetic chambers, segmental wall motion abnormalities
- cardiovascular magnetic resonance: functional and morphological abnormalities as well as tissue pathology (gadolinium enhancement)
- myocardial biopsy

Management
- supportive care
- restrict physical activity
- treat CHF
- treat arrhythmias
- anticoagulation
- treat underlying cause if possible

Prognosis
- often unrecognized, and may be self-limited
- myocarditis treatment trial showed 5 yr mortality between 25-50%
- giant cell myocarditis, although rare can present with fulminant CHF and be rapidly fatal, with 5 yr mortality >80%
- sudden death in young adults
- may progress to dilated cardiomyopathy

Dilated Cardiomyopathy

Definition
- unexplained dilation and impaired systolic function of one or both ventricles

Major Risks Factors for DCM
Alcohol, cocaine, family history

Etiology
- idiopathic (presumed viral or idiopathic) ~50% of DCM
- alcohol
- familial/genetic
- uncontrolled tachycardia (e.g. persistent rapid AFib)
- collagen vascular disease: SLE, polyarteritis nodosa, dermatomyositis, progressive systemic sclerosis
- infectious: viral (coxsackie B, HIV), Chagas disease, Lyme disease, Rickettsial diseases, acute rheumatic fever, toxoplasmosis
- neuromuscular disease: Duchenne muscular dystrophy, myotonic dystrophy, Friedreich's ataxia
- metabolic: uremia, nutritional deficiency (thiamine, selenium, carnitine)
- endocrine: hyper/hypothyroidism, DM, pheochromocytoma
- peripartum
- toxic: cocaine, heroin, organic solvents
- drugs: chemotherapies (doxorubicin, cyclophosphamide), anti-retrovirals, chloroquine, clozapine, TCA
- radiation

Abnormal Labs in DCM
- High BNP
- High Cr
- High LFTs
- Low bicarbonate
- Low Na^+

Signs and Symptoms
- may present as
  - CHF
  - systemic or pulmonary emboli
  - arrhythmias
  - sudden death (major cause of mortality due to fatal arrhythmia)

Investigations
- blood work: CBC, electrolytes, Cr, bicarbonate, BNP, CK, troponin, LFTs, TSH, TIBC
- ECG: variable ST-T wave abnormalities, poor R wave progression, conduction defects (e.g. BBB), arrhythmias (non-sustained VT)
- CXR: global cardiomegaly (globular heart), signs of CHF, pleural effusion
- echo: chamber enlargement, global hypokinesis, depressed LVEF, MR and TR, mural thrombi
- endomyocardial biopsy: not routine, used to rule out a treatable cause
- coronary angiography: in selected patients to exclude ischemic heart disease

Management
- treat underlying disease: e.g. abstinence from alcohol
- treat CHF: see *Heart Failure*, C36
- thromboembolism prophylaxis: anticoagulation with warfarin
  - indicated for: AFib, history of thromboembolism or documented thrombus
- treat symptomatic or serious arrhythmias
- immunize against influenza and S. pneumoniae
- consider surgical options (e.g. LVAD, transplant, volume reduction surgery) in appropriate candidates with severe, drug refractory disease
- consider ICD among patients with a LVEF <30%

Prognosis
- depends on etiology
- better with reversible underlying cause, worst with infiltrative diseases, HIV, drug-induced
- cause of death usually CHF (due to pump failure) or sudden death 2° to ventricular arrhythmias
- systemic emboli are significant source of morbidity
- 20% mortality in 1st yr, 10% per year after

Hypertrophic Cardiomyopathy

- see *2011 ACCF/AHA Guideline for the Diagnosis and Treatment of Hypertrophic Cardiomyopathy* for details

Definition
- defined as unexplained ventricular hypertrophy
- various patterns of HCM are classified, but most causes involve pattern of septal hypertrophy

Etiology and Pathophysiology
- histopathologic features include myocyte disarray, myocyte hypertrophy, and interstitial fibrosis
- cause is felt to be a genetic defect involving one of the cardiac sarcomeric proteins (>400 mutations associated with autosomal dominant inheritance, incomplete penetrance)
- prevalence of 1/500-1/1,000 in general population
- generally presents in early adulthood

Hemodynamic Classification
- hypertrophic obstructive cardiomyopathy (HOCM): dynamic LV outflow tract (LVOT) obstruction, either at rest or with provocation, defined as LVOT gradient of at least 30 mmHg
  - dynamic i.e. obstruction (and the murmur) is reduced with maneuvers that increase preload, and augmented with maneuvers that reduce preload
- non-obstructive HCM: no LVOT obstruction
- many patients have diastolic dysfunction (impaired ventricular filling secondary to LV hypertrophy which decreases compliance)

Signs and Symptoms
- clinical manifestations: asymptomatic (common, therefore screening is important), SOB on exertion, angina, presyncope/syncope (due to LV outflow obstruction or arrhythmia), CHF, arrhythmias, SCD
- pulses: rapid upstroke, "spike and dome" pattern in carotid pulse (in HCM with outflow tract obstruction)
- precordial palpation: PMI localized, sustained, double impulse, 'triple ripple' (triple apical impulse in HOCM), LV lift
- precordial auscultation: normal or paradoxically split S2, S4, harsh systolic diamond-shaped murmur at LLSB or apex, enhanced by squat to standing or Valsalva (murmur secondary to LVOT obstruction as compared to AS); often with pansystolic murmur due to mitral regurgitation

Investigations
- ECG/Holter monitor: LVH, high voltages across precordium, prominent Q waves (lead I, aVL, V5, V6), tall R wave in V1, P wave abnormalities
- transthoracic echocardiography and echo-Doppler study: asymmetric septal hypertrophy (less commonly apical), systolic anterior motion (SAM) of mitral valve and MR; LVOT gradient can be estimated by Doppler measurement
- genetic studies (± magnetic resonance imaging) can be helpful when echocardiography is inconclusive for diagnosis
- cardiac catheterization (only when patient being considered for invasive therapy)

Management
- avoid factors which increase obstruction (e.g. volume depletion)
  - avoidance of all competitive sports
- treatment of obstructive HCM
  - medical agents: β-blockers, disopyramide, verapamil (started only in monitored setting), phenylephrine (in setting of cardiogenic shock)
  - avoid nitrates, diuretics, and ACEI as they increase LVOT gradient and worsen symptoms
- patients with obstructive HCM and drug-refractory symptoms
  - surgical myectomy
  - alcohol septal ablation - percutaneous Intervention that ablates the hypertrophic septum with 100% ethanol via the septal artery
  - dual chamber pacing (rarely done)
- treatment of patients at high risk of sudden death : ICD
- first-degree relatives (children, siblings, parents) of patients with HCM should be screened (physical, ECG, 2D echo) every 12-18 mo during during adolescence, then serially every 5 yr during adulthood

Prognosis
- potential complications: AFib, VT, CHF, sudden cardiac death (1% risk/yr; most common cause of SCD in young athletes)
  - major risk factors for sudden death (consider ICD placement)
  - history of survived cardiac arrest/sustained VT
  - family history of multiple premature sudden deaths
  - other factors associated with increased risk of sudden cardiac death
    - syncope (presumed to be arrhythmic in origin)
    - non-sustained VT on ambulatory monitoring
    - marked ventricular hypertrophy (maximum wall thickness ≥30 mm)
    - abnormal BP in response to exercise (in patients <40 yr old with HCM)

RCM vs. Constrictive Pericarditis (CP)
Present similarly but CP is treatable with surgery

Restrictive Cardiomyopathy

Definition
- impaired ventricular filling with preserved systolic function in a non-dilated, non-hypertrophied ventricle secondary to factors that decrease myocardial compliance (fibrosis and/or infiltration)

Etiology
- infiltrative: amyloidosis, sarcoidosis
- non-infiltrative: scleroderma, idiopathic myocardial fibrosis
- storage diseases: hemochromatosis, Fabry's disease, Gaucher's disease, glycogen storage diseases
- endomyocardial
  - endomyocardial fibrosis, Loeffler's endocarditis, or eosinophilic endomyocardial disease
  - radiation heart disease
  - carcinoid syndrome (may have associated tricuspid valve or pulmonary valve dysfunction)

Key Investigations
- Echo: may show respiratory variation in blood flow in CP
- CT: may show very thickened pericardium and calcification in CP
- MRI: best modality to directly visualize pericardium and myocardium

Clinical Manifestations
- CHF (usually with preserved LV systolic function), arrhythmias
- elevated JVP with prominent x and y descents, Kussmaul's sign
- S3, S4, MR, TR
- thromboembolic events

Investigations
- ECG: low voltage, non-specific, diffuse ST-T wave changes ± non-ischemic Q waves
- CXR: mild cardiac enlargement
- Echo: LAE, RAE; specific Doppler findings with no significant respiratory variation
- cardiac catheterization: increased end-diastolic ventricular pressures
- endomyocardial biopsy: to determine etiology (especially for infiltrative RCM)

Management
- exclude constrictive pericarditis
- treat underlying disease: control HR, anticoagulate if AFib
- supportive care and treatment for CHF, arrhythmias
- heart transplant: might be considered for CHF refractory to medical therapy

Prognosis
- depends on etiology

Valvular Heart Disease

- see *Guidelines on the Management of Valvular Heart Disease. JACC Jun 10;63(22):2438-88* for details

Infective Endocarditis

- see Infectious Diseases, ID16
- American Heart Association (AHA) 2007 guidelines recommend IE prophylaxis
  - only for patients with
    - prosthetic valve material
    - past history of IE
    - certain types of congenital heart disease
    - cardiac transplant recipients who develop valvulopathy
  - only for the following procedures
    - dental
    - respiratory tract
    - procedures on infected skin/skin structures/MSK structures
    - not GI/GU procedures specifically

Rheumatic Fever

- see Pediatrics, P59

Prognosis

- acute complications: myocarditis (DCM/CHF), conduction abnormalities (sinus tachycardia, AFib), valvulitis (acute MR), acute pericarditis (not constrictive pericarditis)
- chronic complications: rheumatic valvular heart disease – fibrous thickening, adhesion, calcification of valve leaflets resulting in stenosis/regurgitation, increased risk of IE ± thromboembolism
- onset of symptoms usually after 10-20 yr latency from acute carditis of rheumatic fever
- mitral valve most commonly affected

Valve Repair and Valve Replacement

- indication for valve repair or replacement depends on the severity of the pathology; typically recommended when medical management has failed to adequately improve the symptoms or reduce the risk of morbidity and mortality
- pathologies that may require surgical intervention include congenital defects, infections, rheumatic heart disease as well as a variety of valve diseases associated with aging
- valve repair: balloon valvuloplasty, surgical valvuloplasty (commissurotomy, annuloplasty), chordae tendineae shortening, tissue patch
- valve replacement: typically for aortic or mitral valves only; repair is favored in younger individuals; percutaneous techniques being established

Mitral Valve Repair vs. Replacement for Severe Ischemic Mitral Regurgitation
NEJM 2014;370:23-32
Purpose: Ischemic mitral regurgitation is associated with significant mortality risk. The purpose of this study was to compare the effectiveness and safety of repairing versus replacing the mitral valve in patients with severe chronic ischemic mitral regurgitation.
Study Design: RCT with 251 patients with severe ischemic mitral regurgitation were randomly assigned to mitral valve repair or chordal-sparing replacement. The primary endpoint was the left ventricular end-systolic volume index (LVESVI) at 12 mo.
Results: There were no significant between-group differences in LVESVI, in the rate of major adverse cardiac or cerebrovascular events, in functional status, or in quality of life at 12 mo. The rate of moderate or severe mitral regurgitation recurrence at 12 mo was significantly higher in the repair group than in the replacement group (32.6% vs. 2.3%, respectively).
Conclusions: No significant difference in left ventricular reverse modelling or survival at 12 mo between patients who underwent mitral valve repair or replacement. Replacement provided more durable correction of mitral regurgitation, but there were no significant differences in clinical outcomes.

Choice of Valve Prosthesis

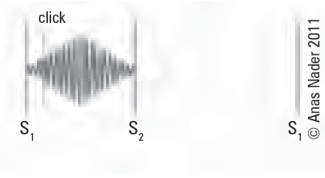
Table 15. Mechanical Valve vs. Bioprosthetic Valve

Mechanical Valve	Bioprosthetic Valve
Good durability	Limited long-term durability (mitral<aortic)
Less preferred in small aortic root sizes	Good flow in small aortic root sizes
Increased risk of thromboembolism (1-3%/yr): requires long-term anticoagulation with coumadin	Decreased risk of thromboembolism: long-term anticoagulation not needed for aortic valves
Target INR Aortic valves: 2.0-3.0 (mean 2.5) Mitral valves: 2.5-3.5 (mean 3.0)	Some recommendation for limited anticoagulation for mitral valves
Increased risk of hemorrhage: 1-2%/yr	Decreased risk of hemorrhage

Summary of Valvular Disease

Table 16. Valvular Heart Disease

Aortic Stenosis (AS)



Etiology
Congenital (bicuspid, unicuspid valve), calcification (wear and tear), rheumatic disease

Definition
Normal aortic valve area = 3-4 cm^2
Mild AS 1.5 to 3 cm^2
Moderate AS 1.0 to 1.5 cm^2
Severe AS <1.0 cm^2
Critical AS <0.5 cm^2

Pathophysiology
Outflow obstruction → increased EDP → concentric LVH → LV failure → CHF, subendocardial ischemia

Symptoms
Exertional angina, syncope, dyspnea, PND, orthopnea, peripheral edema

Physical Exam
Narrow pulse pressure, brachial-radial delay, pulsus parvus et tardus, sustained PMI
Auscultation: crescendo-decrescendo SEM radiating to R clavicle and carotid, musical quality at apex (Gallavardin phenomenon), S4, soft S2 with paradoxical splitting, S3 (late)

Investigations
ECG: LVH and strain, LBBB, LAE, AFib
CXR: post-stenotic aortic root dilatation, calcified valve, LVH, LAE, CHF
Echo: reduced valve area, pressure gradient, LVH, reduced LV function

Treatment
Asymptomatic: serial echos, avoid exertion
Symptomatic: avoid nitrates/arterial dilators and ACEI in severe AS
Surgery if: symptomatic or LV dysfunction

Surgical Options
Valve replacement: aortic rheumatic valve disease and trileaflet valve
– prior to pregnancy (if AS significant)
– balloon valvuloplasty (in very young)

Interventional Options
Percutaneous valve replacement (transfemoral or transapical approach) is an option in selected patients who are not considered good candidates for surgery

Aortic Regurgitation (AR)

Etiology
Supravalvular: aortic root disease (Marfan's, atherosclerosis and dissecting aneurysm, connective tissue disease)
Valvular: congenital (bicuspid aortic valve, large VSD), IE
Acute Onset: IE, aortic dissection, trauma, failed prosthetic valve

Pathophysiology
Volume overload → LV dilatation → increased SV, high sBP and low dBP → increased wall tension → pressure overload → LVH (low dBP → decreased coronary perfusion)

Symptoms
Usually only becomes symptomatic late in disease when LV failure develops
Dyspnea, orthopnea, PND, syncope, angina

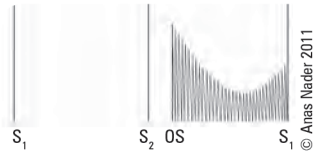
Physical Exam
Waterhammer pulse, bisferiens pulse, femoral-brachial sBP >20 (Hill's test wide pulse pressure), hyperdynamic apex, displaced PMI, heaving apex
Auscultation: early decrescendo diastolic murmur at LLSB (cusp pathology) or RLSB (aortic root pathology), best heard sitting, leaning forward, on full expiration, soft S1, absent S2, S3 (late)

Investigations
ECG: LVH, LAE
CXR: LVH, LAE, aortic root dilatation
Echo/TTE: quantify AR, leaflet or aortic root anomalies
Cath: if >40 yr and surgical candidate – to assess for ischemic heart disease
Exercise testing: hypotension with exercise

Treatment
Asymptomatic: serial echos, afterload reduction (e.g. ACEI, nifedipine, hydralazine)
Symptomatic: avoid exertion, treat CHF
Surgery if: NYHA class III-IV CHF; LV dilatation and/or LVEF <50% with/without symptoms

Surgical Options
Valve replacement: most patients
Valve repair: very limited role
Aortic root replacement (Bentall procedure):
– when ascending aortic aneurysm present, valved conduit used

Mitral Stenosis (MS)



Etiology
Rheumatic disease most common cause, congenital (rare)

Definition
Severe MS is mitral valve area (MVA) <1.2 cm^2

Pathophysiology
MS → fixed CO and LAE → increased LA pressure → pulmonary vascular resistance and CHF; worse with AFib (no atrial kick), tachycardia (decreased atrial emptying time) and pregnancy (increased preload)

Symptoms
SOB on exertion, orthopnea, fatigue, palpitations, peripheral edema, malar flush, pinched and blue facies (severe MS)

Physical Exam
AFib, no "a" wave on JVP, left parasternal lift, palpable diastolic thrill at apex
Auscultation: mid-diastolic rumble at apex, best heard with bell in left lateral decubitus position following exertion, loud S1, OS following loud P2 (heard best during expiration), long diastolic murmur and short A2-OS interval correlate with worse MS

Investigations
ECG: NSR/AFib, LAE (P mitrale), RVH, RAD
CXR: LAE, CHF, mitral valve calcification
Echo/TTE: shows restricted opening of mitral valve
Cath: indicated in concurrent CAD if >40 yr (male) or >50 yr (female)

Treatment
Avoid exertion, fever (increased LA pressure), treat AFib and CHF, increase diastolic filling time (β-blockers, digitalis)
Surgery if: NYHA class III-IV CHF and failure of medical therapy

Invasive Options
Percutaneous balloon valvuloplasty: young rheumatic pts and good leaflet morphology (can be determined by echo), asymptomatic pts with moderate-severe MS, pulmonary HTN
Contraindication: left atrial thrombus, moderate MR
Open Mitral Commissurotomy: if mild calcification + leaflet/chordal thickening
– restenosis in 50% pts in 8 yr
Valve replacement: indicated in moderate-severe calcification and severely scarred leaflets

Mitral Regurgitation (MR)

Etiology
Mitral valve prolapse, congenital cleft leaflets, LV dilatation/aneurysm (CHF, DCM, myocarditis), IE abscess, Marfan's syndrome, HOCM, acute MI, myxoma, mitral valve annulus calcification, chordae/papillary muscle trauma/ischemia/rupture (acute), rheumatic disease

Pathophysiology
Reduced CO → increased LV and LA pressure → LV and LA dilatation → CHF and pulmonary HTN

Symptoms
Dyspnea, PND, orthopnea, palpitations, peripheral edema

Physical Exam
Displaced hyperdynamic apex, left parasternal lift, apical thrill
Auscultation: holosystolic murmur at apex, radiating to axilla ± mid-diastolic rumble, loud S2 (if pulmonary HTN), S3

Investigations
ECG: LAE, left atrial delay (bifid P waves), ± LVH
CXR: LVH, LAE, pulmonary venous HTN
Echo: etiology and severity of MR, LV function, leaflets
Swan-Ganz Catheter: prominent LA "v" wave

Treatment
Asymptomatic: serial echos
Symptomatic: decrease preload (diuretics), decrease afterload (ACEI) for severe MR and poor surgical candidates; stabilize acute MR with vasodilators before surgery
Surgery if: acute MR with CHF, papillary muscle rupture, NYHA class III-IV CHF, AF, increasing LV size or worsening LV function, earlier surgery if valve repairable (>90% likelihood) and patient is low-risk for surgery

Surgical Options
Valve repair: >75% of pts with MR and myxomatous mitral valve prolapse – annuloplasty rings, leaflet repair, chordae transfers/shorten/replacement
Valve replacement: failure of repair, heavily calcified annulus
Advantage of repair: low rate of endocarditis, no anticoagulation, less chance of re-operation

Table 16. Valvular Heart Disease (continued)

Tricuspid Stenosis (TS)

Etiology
Rheumatic disease, congenital, carcinoid syndrome, fibroelastosis; usually accompanied by MS

Pathophysiology
Increased RA pressure → right heart failure → decreased CO and fixed on exertion

Symptoms
Peripheral edema, fatigue, palpitations

Physical Exam
Prominent "a" waves in JVP, +ve abdominojugular reflux, Kussmaul's sign, diastolic rumble 4th left intercostal space

Investigations
ECG: RAE
CXR: dilatation of RA without pulmonary artery enlargement
Echo: diagnostic

Treatment
Preload reduction (diuretics), slow HR
Surgery if: only if other surgery required (e.g. mitral valve replacement)

Surgical Options
Valve Replacement:
– if severely diseased valve
– bioprosthesis preferred

Tricuspid Regurgitation (TR)

Etiology
RV dilatation, IE (particularly due to IV drug use), rheumatic disease, congenital (Ebstein anomaly), carcinoid

Pathophysiology
RV dilatation → TR → further RV dilatation → right heart failure

Symptoms
Peripheral edema, fatigue, palpitations

Physical Exam
"cv" waves in JVP, +ve abdominojugular reflux, Kussmaul's sign, holosystolic murmur at LLSB accentuated by inspiration, left parasternal lift

Investigations
ECG: RAE, RVH, AFib
CXR: RAE, RV enlargement
Echo: diagnostic

Treatment
Preload reduction (diuretics)
Surgery if: only if other surgery required (e.g. mitral valve replacement)

Surgical Options
Annuloplasty (i.e. repair, rarely replacement)

Pulmonary Stenosis (PS)

Etiology
Usually congenital, rheumatic disease (rare), carcinoid syndrome

Pathophysiology
Increased RV pressure → RV hypertrophy → right heart failure

Symptoms
Chest pain, syncope, fatigue, peripheral edema

Physical Exam
Systolic murmur at 2nd left intercostal space accentuated by inspiration, pulmonary ejection click, right-sided S4

Investigations
ECG: RVH
CXR: prominent pulmonary arteries enlarged RV
Echo: diagnostic

Treatment
Balloon valvuloplasty if severe symptoms

Surgical Options
Percutaneous or open balloon valvuloplasty

Pulmonary Regurgitation (PR)

Etiology
Pulmonary HTN, IE, rheumatic disease, tetrology of Fallot (post-repair)

Pathophysiology
Increased RV volume → increased wall tension → RV hypertrophy → right heart failure

Symptoms
Chest pain, syncope, fatigue, peripheral edema

Physical Exam
Early diastolic murmur at LLSB, Graham Steell (diastolic) murmur 2nd and 3rd left intercostal space increasing with inspiration

Investigations
ECG: RVH
CXR: prominent pulmonary arteries if pulmonary HTN; enlarged RV
Echo: diagnostic

Treatment
Rarely requires treatment; valve replacement (rarely done)

Surgical Options
Pulmonary valve replacement

Mitral Valve Prolapse (MVP)

Etiology
Myxomatous degeneration of chordae, thick, bulky leaflets that crowd orifice, associated with Marfan's syndrome, pectus excavatum, straight back syndrome, other MSK abnormalities; <3% of population

Pathophysiology
Mitral valve displaced into LA during systole; no causal mechanisms found for symptoms

Symptoms
Prolonged, stabbing chest pain, dyspnea, anxiety/panic, palpitations, fatigue, presyncope

Physical Exam
Ausculation: mid-systolic click (due to billowing of mitral leaflet into LA; tensing of redundant valve tissue); mid to late systolic murmur at apex, accentuated by Valsalva or squat-to-stand maneuvers

Investigations
ECG: non-specific ST-T wave changes, paroxysmal SVT, ventricular ectopy
Echo: systolic displacement of thickened mitral valve leaflets into LA

Treatment
Asymptomatic: no treatment; reassurance
Symptomatic: β-blockers and avoidance of stimulants (caffeine) for significant palpitations, anticoagulation if AFib

Surgical Options
Mitral valve surgery (repair favoured over replacement) if symptomatic and significant MR

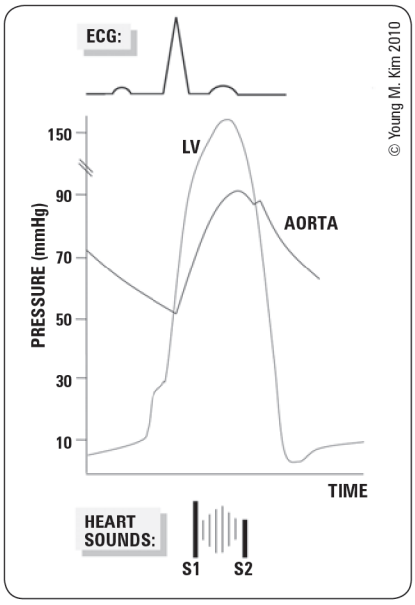


Figure 41. Hemodynamics of aortic stenosis
Stenosis across the aortic valve results in the generation of a significant pressure gradient between the left ventricle and the aorta and a crescendo-decrescendo murmur during systolic contraction. The stenosis decreases the intensity of aortic valve closure hence diminishing S2

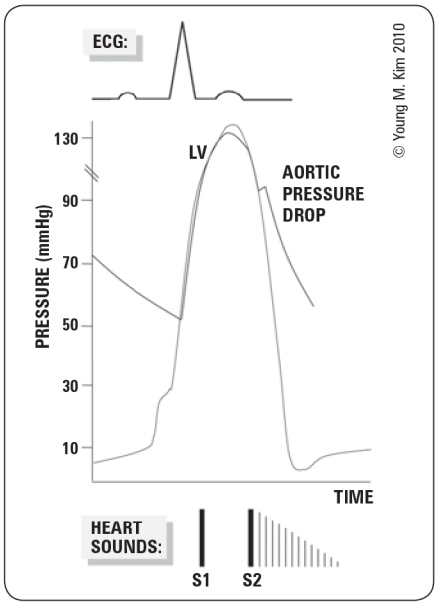


Figure 42. Hemodynamics of aortic regurgitation
Regurgitation across the aortic valve during diastole causes the aortic pressure to rapidly decrease and a decrescendo murmur can be heard at the onset of diastole (after S2 is audible). The presence of regurgitant blood from the aorta increases left-ventricular end-diastolic volume

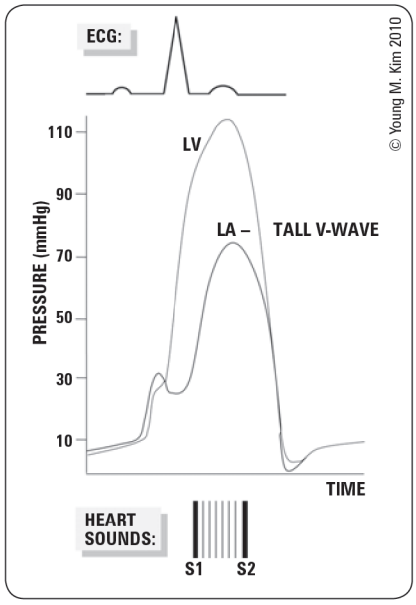


Figure 43. Hemodynamics of acute mitral regurgitation
During systolic contraction, blood regurgitates from the left ventricle into the left atrium across the incompetent mitral valve resulting in an audible holosystolic murmur between S1 and S2. The portion of left ventricular end diastolic volume that regurgitates into the left atrial myocardium increases left atrial pressures resulting in a tall V-wave (in the JVP)

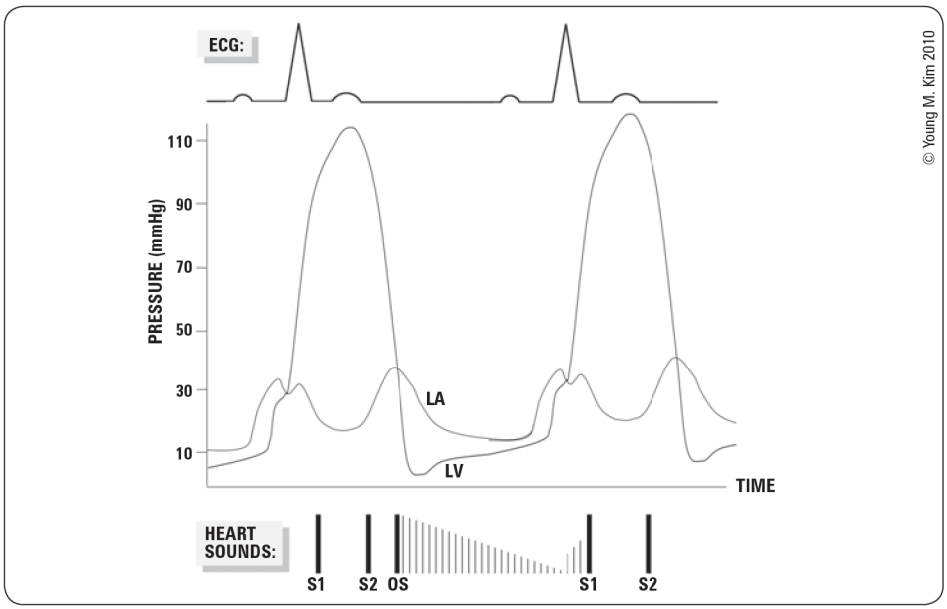


Figure 44. Hemodynamics of mitral stenosis
First note that the left atrial pressure exceeds the left ventricular pressure during diastole due to mitral stenosis and the consequent generation of a pressure gradient across the left atrium and left ventricle. In diastole the stenotic mitral valve opens which corresponds to the opening snap (OS) and the passage of blood across the mitral stenosis results in an audible decrescendo murmur. Left atrial contraction prior to S1 increases the pressure gradient resulting in accentuation of the murmur before S1 is audible

Pericardial Disease

Acute Pericarditis

Etiology of Pericarditis/Pericardial Effusion
- idiopathic is most common: presumed to be viral
- infectious
  - viral: Coxsackie virus A, B (most common), echovirus
  - bacterial: *S. pneumoniae, S. aureus*
  - TB
- fungal: histoplasmosis, blastomycosis
- post-MI: acute (direct extension of myocardial inflammation, 1-7 d post-MI), Dressler's syndrome (autoimmune reaction, 2-8 wk post-MI)
- post-cardiac surgery (e.g. CABG), other trauma
- metabolic: uremia (common), hypothyroidism
- neoplasm: Hodgkin's, breast, lung, renal cell carcinoma, melanoma
- collagen vascular disease: SLE, polyarteritis, rheumatoid arthritis, scleroderma
- vascular: dissecting aneurysm
- other: drugs (e.g. hydralazine), radiation, infiltrative disease (sarcoid)

Signs and Symptoms
- diagnostic triad: chest pain, friction rub and ECG changes (diffuse ST elevation and PR depression with reciprocal changes in aVR)
- pleuritic chest pain: alleviated by sitting up and leaning forward
- pericardial friction rub: may be uni-, bi-, or triphasic; evanescent and rare
- ± fever, malaise

Investigations
- ECG: initially diffuse elevated ST segments ± depressed PR segment, the elevation in the ST segment is concave upwards → 2-5 d later ST isoelectric with T wave flattening and inversion
- CXR: normal heart size, pulmonary infiltrates
- Echo: performed to assess for pericardial effusion

Acute Pericarditis Triad
- Chest pain
- Friction rub
- ECG changes

Treatment
- treat the underlying disease
- anti-inflammatory agents (high dose NSAIDs/ASA, steroids use controversial), analgesics
- colchicine reduces the rate of incessant/recurrent pericarditis (ICAP N Engl J Med 2013; 369:1522-1528)

Complications
- recurrent episodes of pericarditis, atrial arrhythmia, pericardial effusion, tamponade, constrictive pericarditis

Pericardial Effusion

Etiology
- transudative (serous)
- CHF, hypoalbuminemia/hypoproteinemia, hypothyroidism
- exudative (serosanguinous or bloody)
  - causes similar to the causes of acute pericarditis
  - may develop acute effusion secondary to hemopericardium (trauma, post-MI myocardial rupture, aortic dissection)
- physiologic consequences depend on type and volume of effusion, rate of effusion development, and underlying cardiac disease

Ewart's Sign
Bronchial breathing and dullness to percussion at the lower angle of the left scapula in pericardial effusion due to effusion compressing left lower lobe of lung

Signs and Symptoms
- may be asymptomatic or similar to acute pericarditis
- dyspnea, cough
- extra-cardiac (esophageal/recurrent laryngeal nerve/tracheo-bronchial/phrenic nerve irritation)
- JVP increased with dominant "x" descent
- arterial pulse normal to decreased volume, decreased pulse pressure
- auscultation: distant heart sounds ± rub
- Ewart's sign

Investigations
- ECG: low voltage (sum of QRS in I + II + III <155 or V1 +V2 +V3 <3010 MM), flat T waves, electrical alternans (classic, but not sensitive to exclude effusion)
  - be cautious in diagnosing STEMI in a patient with pericarditis and an effusion - antiplatelets may precipitate hemorrhagic effusion
- CXR: cardiomegaly, rounded cardiac contour
- ER: bedside ultrasound with subxiphoid view showing fluid in pericardial sac
- Echo (procedure of choice): fluid in pericardial sac
- pericardiocentesis: definitive method of determining transudate vs. exudate, identify infectious agents, neoplastic involvement

Treatment
- mild: frequent observation with serial echos, treat underlying cause, anti-inflammatory agents
- severe: treat as in tamponade (see *Cardiac Tamponade*)

Cardiac Tamponade

Etiology
- major complication of rapidly accumulating pericardial effusion
- cardiac tamponade is a clinical diagnosis
- any cause of pericarditis but especially trauma, malignancy, uremia, proximal aortic dissection with rupture

Pathophysiology
- high intra-pericardial pressure → decreased venous return → decreased diastolic ventricular filling → decreased CO → hypotension and venous congestion

Signs and Symptoms
- tachypnea, dyspnea, shock, muffled heart sounds
- pulsus paradoxus (inspiratory fall in systolic BP >10 mmHg during quiet breathing)
- JVP "x" descent only, blunted "y" descent
- hepatic congestion/peripheral edema

Investigations
- ECG: electrical alternans (pathognomonic variation in R wave amplitude), low voltage
- echo: pericardial effusion, compression of cardiac chambers (RA and RV) in diastole
- cardiac catheterization

Treatment
- pericardiocentesis: Echo-guided
- pericardiotomy
- avoid diuretics and vasodilators (these decrease venous return to already under-filled RV → decrease LV preload → decrease CO)
- IV fluid may increase CO
- treat underlying cause

Classic Quartet of Tamponade
- Hypotension
- Increased JVP
- Tachycardia
- Pulsus paradoxus

Beck's Triad
- Hypotension
- Increased JVP
- Muffled heart sounds

Constrictive Pericarditis

Etiology
- chronic pericarditis resulting in fibrosed, thickened, adherent, and/or calcified pericardium
- any cause of acute pericarditis may result in chronic pericarditis
- major causes are idiopathic, post-infectious (viral, TB), radiation, post-cardiac surgery, uremia, MI, collagen vascular disease

Signs and Symptoms
- dyspnea, fatigue, palpitations
- abdominal pain
- may mimic CHF (especially right-sided HF)
  - ascites, hepatosplenomegaly, edema
- increased JVP, Kussmaul's sign (paradoxical increase in JVP with inspiration), Friedreich's sign (prominent "y" descent)
- BP usually normal (and usually no pulsus paradoxus)
- precordial examination: ± pericardial knock (early diastolic sound)
- see Table 17 for differentiation from cardiac tamponade

Investigations
- ECG: non-specific – low voltage, flat T wave, ± AFib
- CXR: pericardial calcification, effusions
- echo/CT/MRI: pericardial thickening
- cardiac catheterization: equalization of end-diastolic chamber pressures (diagnostic)

Treatment
- medical: diuretics, salt restriction
- surgical: pericardiectomy (only if refractory to medical therapy)
- prognosis best with idiopathic or infectious cause and worst in post-radiation; death may result from heart failure

DDx Pulsus Paradoxus
- Constrictive pericarditis (rarely)
- Severe obstructive pulmonary disease (e.g. asthma)
- Tension pneumothorax
- PE
- Cardiogenic shock
- Cardiac tamponade

Table 17. Differentiation of Constrictive Pericarditis vs. Cardiac Tamponade

Characteristic	Constrictive Pericarditis	Cardiac Tamponade
JVP	"y" > "x"	"x" > "y"
Kussmaul's sign	Present	Absent
Pulsus paradoxus	Uncommon	Always
Pericardial knock	Present	Absent
Hypotension	Variable	Severe

Common Medications

Table 18. Commonly Used Cardiac Therapeutics

Drug Class	Examples	Mechanism of Action	Indications	Side Effects	Contraindications
ANGIOTENSIN CONVERTING ENZYME INHIBITORS (ACEI)					
	enalapril (Vasotec®), perindopril (Coversyl®), ramipril (Altace®), lisinopril (Zestril®)	Inhibit ACE-mediated conversion of angiotensin I to angiotensin II (AT II), causing peripheral vasodilation and decreased aldosterone synthesis	HTN, CAD, CHF, post-MI, DM	Dry cough, 10% hypotension, fatigue, hyperkalemia, renal insufficiency, angioedema	Bilateral renal artery stenosis, pregnancy, caution in decreased GFR
ANGIOTENSIN II RECEPTOR BLOCKERS (ARBs)					
	candesartan, irbesartan, valsartan	Block AT II receptors, causing similar effects to ACEI	Same as ACEI, although evidence is generally less for ARBs; often used when ACEI are not tolerated	Similar to ACEI, but do not cause dry cough	Same as ACEI
DIRECT RENIN INHIBITORS (DRIs)					
	aliskiren	Directly blocks renin thus inhibiting the conversion of angiotensinogen to angiotensin I; this also causes a decrease in AT II	HTN (exact role of this drug remains unclear)	Diarrhea, hyperkalemia (higher risk if used with an ACEI), rash, cough, angioedema, reflux, hypotension, rhabdomyolysis, seizure	Pregnancy, severe renal impairment
β-BLOCKERS					
β1 antagonists β1/β2 antagonists α1/β1/β2 antagonists β1 antagonists with intrinsic sympathomimetic activity	atenolol, metoprolol, bisoprolol propranolol labetalol, carvedilol acebutalol	Block β-adrenergic receptors, decreasing HR, BP, contractility, and myocardial oxygen demand, slow conduction through the AV node	HTN, CAD, acute MI, post-MI, CHF (start low and go slow), AFib, SVT	Hypotension, fatigue, light-headedness, depression, bradycardia, hyperkalemia, bronchospasm, impotence, depression of counterregulatory response to hypoglycemia, exacerbation of Raynaud's phenomenon, and claudication	Sinus bradycardia, 2nd or 3rd degree heart block, hypotension, WPW Caution in asthma, claudication, Raynaud's phenomenon, and decompensated CHF
CALCIUM CHANNEL BLOCKERS (CCBs)					
Benzothiazepines Phenylalkylamines (non-dihydropyridines)	diltiazem verapamil	Block smooth muscle and myocardial calcium channels causing effects similar to β-blockers Also vasodilate	HTN, CAD, SVT, diastolic dysfunction	Hypotension, bradycardia, edema Negative inotrope	Sinus bradycardia, 2nd or 3rd degree heart block, hypotension, WPW, CHF
Dihydropyridines	amlodipine (Norvasc®), nifedipine (Adalat®), felodipine (Plendil®)	Block smooth muscle calcium channels causing peripheral vasodilation	HTN, CAD	Hypotension, edema, flushing, headache, light-headedness	Severe aortic stenosis and liver failure
DIURETICS					
Thiazides	hydrochlorthiazide, chlorthalidone, metolazone	Reduce Na^+ reabsorption in the distal convoluted tubule (DCT)	HTN (drugs of choice for uncomplicated HTN)	Hypotension, hypokalemia, polyuria	Sulfa allergy, pregnancy
Loop diuretics	furosemide (Lasix®)	Blocks Na^+/K^+-ATPase in the loop of Henle	CHF, pulmonary or peripheral edema	Hypovolemia, hypokalemic metabolic alkalosis	Hypovolemia, hypokalemia
Aldosterone receptor antagonists	spironolactone, eplenerone	Antagonize aldosterone receptors	HTN, CHF, hypokalemia	Edema, hyperkalemia, gynecomastia	Renal insufficiency, hyperkalemia, pregnancy
INOTROPES					
	digoxin (Lanoxin®)	Inhibit Na^+/K^+-ATPase, leading to increased intracellular Na^+ and Ca^{2+} concentration and increased myocardial contractility Also slows conduction through the AV node	CHF, AFib	AV block, tachyarrhythmias, bradyarrhythmias, blurred or yellow vision (van Gogh syndrome), anorexia, N/V	2nd or 3rd degree AV block, hypokalemia, WPW

Table 18. Commonly Used Cardiac Therapeutics (continued)

Drug Class	Examples	Mechanism of Action	Indications	Side Effects	Contraindications
ANTICOAGULANTS					
Coumarins	warfarin (Coumadin®)	Antagonizes vitamin K, leading to decreased synthesis of clotting factors II, VII, IX, and X	AFib, LV dysfunction, prosthetic valves	Bleeding (by far the most important side effect), paradoxical thrombosis, skin necrosis	Recent surgery or bleeding, bleeding diathesis, pregnancy
Heparins	Unfractionated heparin LMWHs: dalteparin, enoxaparin, tinzaparin	Antithrombin III agonist, leading to decreased clotting factor activity	Acute MI; when immediate anticoagulant effect needed	Bleeding, osteoporosis, heparin-induced thrombocytopenia (less in LMWHs)	Recent surgery or bleeding, bleeding diathesis, thrombocytopenia, renal insufficiency (for LMWHs)
Direct thrombin inhibitors	dabigatran, melagatran	Competitive, direct thrombin inhibitor; thrombin enables fibrinogen conversion to fibrin during the coagulation cascade, thereby preventing thrombus development	AFib	Bleeding, GI upset	Severe renal impairment, recent surgery, active bleeding
Direct Factor Xa inhibitors	rivaroxaban apixaban edoxaban	Direct, selective and reversible inhibition of factor Xa in both the intrinsic and extrinsic coagulation pathways	AFib	Bleeding, GI upset, elevated liver enzymes	Hepatic disease, active bleeding, bleeding diathesis, pregnancy, lactation
ANTIPLATELETS					
Salicylates	ASA (Aspirin®)	Irreversibly acetylates platelet COX-1, preventing thromboxane A2-mediated platelet aggregation	CAD, acute MI, post-MI, post-PCI, CABG	Bleeding, GI upset, GI ulceration, impaired renal perfusion	Active bleeding or PUD
Thienopyridines	clopidogrel (Plavix®), ticlopidine (Ticlid®) prasugrel (Effient®)	$P2Y_{12}$ antagonist (block platelet ADP receptors	Acute MI, post-MI, post-PCI, CABG	Bleeding, thrombotic thrombocytopenic purpura, neutropenia (ticlopidine)	Active bleeding or PUD
Nucleoside analogues	ticagrelor (Brillinta®)	$P2Y_{12}$ antagonist (but different binding site than thienopyridines)			
GPIIb/IIIa inhibitors	eptifibatide, tirofiban, abciximab	Block binding of fibrinogen to Gp IIb/IIIa	Acute MI, particularly if PCI is planned	Bleeding	Recent surgery or bleeding, bleeding diathesis
THROMBOLYTICS					
	alteplase, reteplase, tenecteplase, streptokinase	Convert circulating plasminogen to plasmin, which lyses cross-linked fibrin	Acute STEMI	Bleeding	See Table 8, C30
NITRATES					
	nitroglycerin	Relax vascular smooth muscle, producing venous and arteriolar dilation	CAD, MI, CHF (isosorbide dinitrate plus hydralazine)	Headache, dizziness, weakness, postural hypotension	Concurrent use of cGMP phosphodiesterase inhibitors, angle closure glaucoma, increased intracranial pressure
LIPID LOWERING AGENTS					
Statins	atorvastatin (Lipitor®), pravastatin (Pravachol®), rosuvastatin (Crestor®), simvastatin (Zocor®), lovastatin (Meracor®)	Inhibit HMG-CoA reductase, which catalyzes the rate-limiting step in cholesterol synthesis	Dyslipidemia (1° prevention of CAD), CAD, post-MI (2° prevention of CV events)	Myalgia, rhabdomyolysis, abdominal pain	Liver or muscle disease
Cholesterol absorption inhibitor	ezetimibe (Ezetrol®)	inhibits gut absorption of cholesterol	Decreases LDL but does not reduceand mortality	Myalgia, rhabdomyolysis, abdominal pain	Liver or renal impairment
Miscellaneous	fibrates, bile acid sequestrates, nicotinic acid		Primarily in familial hypercholesterolemia	GI side effects common	
Investigational	PSCK9 inhibitor	monoclonal antibody	hypercholesterolemia		

Antiarrhythmics

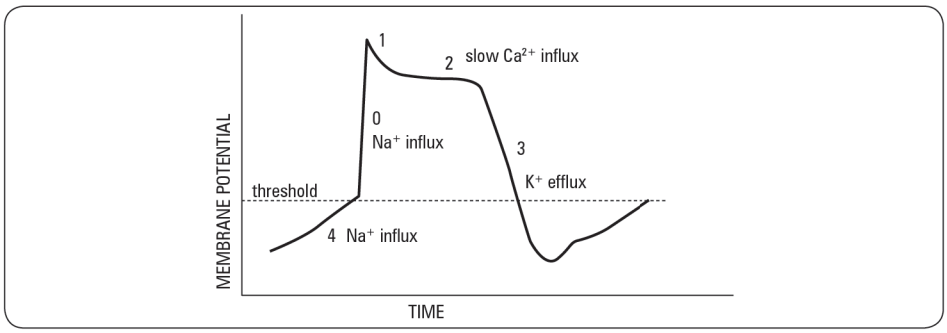


Figure 45. Representative cardiac action potential

Table 19. Antiarrhythmic* Drugs (Vaughan-Williams Classification)

Class	Agent	Indications	Side Effects	Mechanism of Action
Ia	quinidine procainamide disopyramide	SVT, VT	Torsades de Pointes (all Ia), diarrhea Lupus-like syndrome Anticholinergic effects	Moderate Na^+ channel blockade Slows phase 0 upstroke Prolongs repolarization, slowing conduction
Ib	lidocaine mexiletine	VT	Confusion, stupor, seizures GI upset, tremor	Mild Na^+ channel blockade Shortens phase 3 repolarization
Ic	propafenone flecainide encainide	SVT, VT AFib	Exacerbation of VT (all Ic) Negative inotropy (all Ic) Bradycardia and heart block (all Ic)	Marked Na^+ channel blockade Markedly slows phase 0 upstroke
II	propranolol metoprolol, etc.	SVT, AFib	Bronchospasm, negative inotropy, bradycardia, AV block, impotence, fatigue	β-blocker Decreases phase 4 depolarization
III	amiodarone** sotalol	SVT, VT AFib SVT, VT, AFib	Amiodarone: Photosensitivity, pulmonary toxicity, hepatotoxicity, thyroid disease, increased INR Amiodarone and Sotalol: Torsades de Pointes, bradycardia, heart block, β-blocker side effects	Blocks K^+ channel Prolongs phase 3 repolarization, which prolongs refractory period
IV	verapamil diltiazem	SVT AFib	Bradycardia, AV block Hypotension	Calcium channel blocker Slows phase 4 spontaneous depolarization, slowing AV node conduction

*All antiarrhythmics have potential to be proarrhythmic **Amiodarone has class I, II, III, and IV properties

Antiarrythmic Drug Classification

Some **B**lock **P**otassium **C**hannels
I – **S**odium channel blocker
II – β-**B**locker
III – **P**otassium channel blocker
IV – **C**CB

Table 20. Actions of α and β Adrenergic Receptors

	α RECEPTORS		β RECEPTORS	
Target System	**α1**	**α2**	**β1**	**β2**
Cardiovascular	Constriction of vascular smooth muscle Constriction of skin, skeletal muscle, and splanchnic vessels	Same as α1	Increased myocardial contractility Accelerate SA node Accelerate ectopic pacemakers	Decreased vascular smooth muscle tone
	Increased myocardial contractility Decreased heart rate	Peripherally act to modulate vessel tone Vasoconstrict and dilate; oppose α1 vasoconstrictor activity		
Respiratory				Bronchodilation
Dermal	Pilomotor smooth muscle contraction Apocrine constriction			
Ocular	Radial muscle contraction		Ciliary muscle relaxation	
Gastrointestinal	Inhibition of myenteric plexus Anal sphincter contraction			
Genitourinary	Pregnant uterine contraction Penile and seminal vesicle ejaculation Urinary bladder contraction	Smooth muscle wall relaxation	Stimulation of renal renin release	Bladder wall relaxation Uterine relaxation
Metabolic	Stimulate liver gluconeogenesis and glycogenolysis at the liver	Same as α1 Fat cell lipolysis	Fat cell lipolysis Glycogenolysis	Gluconeogenesis Fat cell lipolysis

Adapted from the Family Practice Notebook (www.fpnotebook.com/NEU194.htm)

Table 21. Commonly Used Drugs that Act on α and β Adrenergic Receptors

	α RECEPTORS			β RECEPTORS		
Mechanism of Action	**α1**	**α1 and α2**	**α2**	**β1**	**β1 and β2**	**β2**
Agonist	Phenylephrine Methoxamine	Epinephrine Norepinephrine	Clonidine Methyldopa	Norepinephrine Dobutamine	Isoproterenol Epinephrine	Albuterol Terbutaline
Antagonist	Prazosin Phenoxybenzamine	Phentolamine	Yohimbine Mirtazipine	Metoprolol Acebutolol Alprenolol Atenolol Esmolol	Propranolol Timolol Nadolol Pindolol Carvedilol	Butoxamine

Adapted from the Family Practice Notebook (http://www.fpnotebook.com/NEU194.htm)

Landmark Cardiac Trials

Trial	Reference	Results
ISCHEMIC HEART DISEASE		
ASCOT-LLA	*Lancet* 2003; 361:1149-58	In hypertensive patients with risk factors for CHD and average or below-average cholesterol, atorvastatin reduced nonfatal MI, fatal CHD, fatal/nonfatal stroke, coronary events but not all-cause mortality
CAPRIE	*Lancet* 1996; 348:1329-39	In atherosclerotic vascular disease clopidogrel reduced the primary combined endpoint of stroke, MI, or vascular death and improved PAD compared to ASA
CARE	*NEJM* 1996; 335:1001-9	Pravastatin reduced MI and stroke in patients with previous MI and average cholesterol
COURAGE	*NEJM* 2007; 356:1503-16	Compared with optimal medical therapy alone PCI + medical therapy did not reduce all-cause mortality and non fatal MI, and it did not reduce the incidence of major cardiovascular events
CURE	*NEJM* 2001; 345:494-502	Clopidogrel plus ASA reduced death from CV causes, non fatal MI, or stroke but increased bleeding complications
EUROPA	*Lancet* 2003; 362:782-88	With stable CAD and no CHF perindopril reduced cardiovascular death, MI, and total mortality
HOPE	*NEJM* 2000; 342:154-60	In high-risk patients without low LVEF or CHF ramipril reduced rates of death, MI, stroke, revascularization, new diagnosis of DM and complications due to DM; vitamin E had no effect on outcomes
HPS	*Lancet* 2002; 360:7-22	In high-risk patients with various cholesterol values simvastatin reduced all-cause mortality, coronary deaths, and major vascular events
INTERHEART	*Lancet* 2004; 364:937-52	Nine modifiable risk factors account for 90% of myocardial infarction
IMPROVE-IT	*N Engl J Med* 2015 Jun 3. [Epub ahead of print]	Ezetimibe added to statin reduces mortality in ACS patients
JUPITER	*NEJM* 2008; 359:2195-2207	With low to normal LDL-C and elevated hsCRP treatment with rosuvastatin significantly reduced major cardiovascular events; NNT with rosuvastatin for 2 yr to prevent one primary endpoint = 95
SYNTAX	*NEJM* 2009; 360:961-972	CABG has lower rate of major cardiac or cerebrovascular events; the rate of stroke was increased with CABG, whereas the rate of repeat revascularization was increased with PCI
TNT	*NEJM* 2005; 352:1425-35	Lipid-lowering therapy with atorvastatin 80 mg/d in patients with stable CHD provides clinical benefit beyond atorvastatin 10 mg/d
WHI	*JAMA* 2002; 288:321-333	Estrogen plus progestin therapy is associated with increased risks of cardiovascular disease and breast cancer but decreased risks of hip fracture and colorectal cancer in postmenopausal women
MYOCARDIAL INFARCTION		
BHAT	*JAMA* 1982; 247:1707-14	In acute MI propranolol reduced all-cause mortality, cardiovascular death, and sudden death from atherosclerotic heart disease
COURAGE	*NEJM* 2007; 356:1503-16	Compared with optimal medical therapy alone PCI + medical therapy did not reduce all-cause mortality and non fatal MI, and it did not reduce the incidence of major cardiovascular events
DAPT	*NEJM* 2014; 371: 2155-66	Dual antiplatelet therapy beyond one year confers additional benefit

Trial	Reference	Results
MYOCARDIAL INFARCTION		
ISIS-2	*Lancet* 1988; 2:349-60	Early therapy with streptokinase and ASA in patients with MI individually and in combination significantly reduced all-cause mortality and in combination demonstrated additive effect
ISIS-4	*Lancet* 1995; 345:669-85	In patients with suspected or definite acute MI early treatment with captopril reduced all-cause mortality at 35 d and during long-term follow up
OASIS-5	*NEJM* 2006; 354:1464-76	Compared to enoxaparin, fondaparinux reduced mortality rates, major bleeds at 9 and MI at 30 and 180 d
PEGASUS-TIMI54	*NEJM* 2015 EPUB	Ticagrelor on top of ASA reduces CV events and in patients with a history of MI
PLATO	*NEJM* 2009: 361:1045-57	ACS patients with either STEMI or NSTEMI, regardless of reperfusion strategy, ticagrelor reduced mortality, MI and stroke without increased bleeding compared to clopidogrel
PROVE IT – TIMI 22	*NEJM* 2004; 350:1495-1504	In patients hospitalized for ACS high-dose atorvastatin reduced all-cause mortality, MI, unstable angina, revascularization, and stroke compared with pravastatin
TRITON-TIMI 38	*NEJM* 2007; 357:2001-15	In ACS patients scheduled for PCI, prasugrel reduced ischemic events but increased major bleeding compared to clopidogrel; no change in mortality
HEART FAILURE		
AIRE	*Lancet* 1993; 342:821-8	Ramipril commenced 3-10 d after MI and continued for a mean 15-month period significantly reduced all-cause mortality in patients with non-severe CHF
CHARM	*Lancet* 2003; 362:759-66	Candesartan reduced overall mortality, cardiovascular death, and CHF hospitalizations
CIBIS II	*Lancet* 1999; 353:9-13	Bisoprolol reduced all-cause mortality, cardiovascular death, all-cause hospitalization, and CHF hospitalization
COMET	*Lancet* 2003; 362:7-13	Carvedilol was associated with a reduction in all cause mortality compared with metoprolol
CONSENSUS	*NEJM* 1987; 316:1429-35	Enalapril reduced all-cause mortality, death due to progression of heart failure
COPERNICUS	*NEJM* 2001; 344:1651-8	Carvedilol in addition to standard treatment significantly reduced the risk of death or hospitalization in patients with severe CHF
I-PRESERVE	*NEJM* 2008; 359:2456-2467	In patients with CHF and normal LVEF treatment with ARB (irbesartan) did not improve mortality or cardiovascular morbidity compared to placebo
MERIT-HF	*Lancet* 1999; 353:2001-7	Metoprolol CR/XL daily in addition to optimum standard therapy improved survival in clinically stable patients equating to prevention of 1 death per 27 patients treated per year
PARADIGM-HF	*NEJM* 2014; 371:993-1004	Novel drug (LCZ696 containing valsartan and a neprilysin inhibitor (prevents degradation of natriuretic peptides) reduces hospitalization and mortality
RALES	*NEJM* 1999; 341:709-17	In severe CHF (class III/IV) and LVEF $<35\%$ spironolactone reduced all-cause mortality, sudden death, and death due to progression of heart failure
SAVE	*NEJM* 1992; 327:669-77	Patients with LV dysfunction post-MI long-term captopril over 3.5 yr reduced the risk of death due to cardiovascular causes, recurrent MI, development of severe CHF, and CHF hospitalization
SCD-HeFT	*NEJM* 2005; 352:225-237	In mild-to-moderate CHF shock-only ICD significantly reduces risk of death; amiodarone had no benefit compared with placebo in treating patients with mild-to-moderate CHF
SOLVD	*NEJM* 1991; 325:293-302	In stable chronic CHF with decreased LVEF (<0.35) long-term enalapril reduced death due to all causes and death or hospitalization due to CHF
TRACE	*NEJM* 1995; 333:1670-6	In patients with LV dysfunction post-MI long-term trandolapril reduced the risk of death or progression to severe CHF and reduced risk of sudden death
V-HeFT II	*NEJM* 1991; 325:303-10	In chronic CHF enalapril reduced mortality more than hydralazine-isosorbide for at least 2 yr; treatment with either enalapril or hydralazine-isosorbide increased LVEF
DIABETES		
CARDS	*Lancet* 2004; 264:685-96	Atorvastatin reduces the risk of cardiovascular events in patients with type 2 DM
ONTARGET	*NEJM* 2008; 358:1547-59	In patients with vascular disease or DM without CHF telmisartan is equally as effective as ramipril, with telmisartan causing a reduced risk of cough and angioedema, and an increased risk of hypotensive symptoms; combination therapy offers no advantage

Trial	Reference	Results
ARRHYTHMIA		
AFFIRM	*NEJM* 2002; 347:1825-33	No significant difference in mortality rates between rate or rhythm control of AFib
AF-CHF	*NEJM* 2008; 358:2667-77	In patients with AFib and CHF there is no significant difference in mortality rates from cardiovascular causes between rate and rhythm control
ARISTOTLE	*NEJM* 2011: 365:981-92	AF patients treated with apixaban had a lower incidence of stroke, major bleeding and mortality compared to warfarin
ENGAGE AF-TIMI48	*NEJM* 2013: 369:2093-2104	AF patients treated with edoxaban had similar rates of stroke and lower rates of major bleeding compared to warfarin
RE-LY	*NEJM* 2009: 361:1139-51	AF patients treated with dabigatran had a lower incidence of stroke compared to warfarin, with similar rates of major bleeding
ROCKET-AF	*NEJM* 2011; 365:883-891	In patients with AFib rivoxabarin is non-inferior to warfarin for stroke prevention, and major and non-major bleeding
HYPERTENSION		
HYVET	*NEJM* 2008; 358:1887-98	In hypertensive patients >80 yr treatment with indapamide, with or without perindopril, showed a trend towards reduced relative risk of fatal or non-fatal stroke
SIPLICITY-HTN 3	*NEJM* 2014: 370:1393-1401	Renal denervation does not reduce blood pressure in patients with resistant hypertension compared to sham procedure
UKHDS (UKPDS)	*BMJ* 1998; 317:703-13	Hypertensive patients with DM and tight BP control at <150/85 mmHg by use of ACEI or β-blocker reduced risk of diabetic complications and death related to DM and reduced risk of end-organ damage
VALUE	*Lancet* 2004; 363:2022-2031	Valsartan group had higher incidence of MI than amlodipine group, whereas amlodipine had a higher incidence of new-onset DM

References

Ischemic Heart Disease

Cannon CP, Braunwald E, McCabe CH, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. NEJM 2004;350:1495-504.
Lindahl B, Toss H, Siegbahn A, et al. Markers of myocardial damage and inflammation in relation to long-term mortality in unstable coronary artery disease. NEJM 2000;343:1139-1147.
Pitt B, Remme W, Zannad F, et al. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. NEJM 2003;348:1309-1321.
Rauch U, Osende JI, Fuster V, et al. Thrombus formation on the atherosclerotic plaques: pathogenesis and clinical consequences. Ann Intern Med 2001;134:224-238.
Spinler S, Rees C. Review of prasugrel for the secondary prevention of atherothrombosis. J Manag Care Pharm 2009;15:383-95.
The Arterial Revascularization Therapies Study Group. Comparison of coronary-artery bypass surgery and stenting for the treatment of multivessel disease. NEJM 2001;344:1117-1124.
Turpie AGG, Antman EM. Low-molecular-weight heparins in the treatment of acute coronary syndromes. Arch Intern Med 2001;161:1484-1490.
Yeghiazarians Y, Braunstein JB, Askari A, et al. Review article: unstable angina pectoris. NEJM 2000;342:101-114.

Nuclear Cardiology

Lee TH, Boucher CA. Noninvasive tests in patients with stable coronary artery disease (review). NEJM 2000;344:1840-1845.

Cardiomyopathies

Feldman AM, McNamara D. Myocarditis (review). NEJM 2000;343:1388-1398.

Guidelines

2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. JACC 2013; 61:e78-140.
2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the diagnosis and management of patients with stable ischemic heart disease. JACC 2012;60:e44-e164.
ACC/AHA guidelines for percutaneous coronary intervention. Circulation 2001;103:3019-3041.
ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction. Available from: http://www.acc.org.
ACCF/AHA 2009 focused update on the guidelines for the diagnosis and management of heart failure in adults. Circulation 2009;119:1977-2016.
ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). Circulation 2004;110:e82-292.
American College of Cardiology (clinical guidelines, etc). Available from: http://www.acc.org.
Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial Infarction – summary: a report of the American College of Canadian Cardiovascular Society (CCS) mobile guidelines. Available from: http://www.ccs.ca/mobile.Free.
Aurigemma GP, Gaasch WH. Clinical practice, diastolic heart failure. NEJM 2004;351:1097.
Beard JD. Chronic lower limb ischemia. BMJ 2000;320:854-857.
Canadian Cardiovascular Society 2005 Consensus Conference Peripheral Arterial Disease (Draft). Available from: http:// www.ccs.ca.
Cardiology. American Heart Association Task Force on practice guidelines. Circulation 2004;110-588.
Cardiology Online. Available from: http:// www.theheart.org.
CCS focused 2012 update of the CCS atrial fibrillation guidelines: recommendations for stroke prevention and rate/rhythm control. Can J Cardiol 2012;28:125-136.
CCS. The use of antiplatelet therapy in the outpatient setting: CCS guidelines. Can J Cardiol 2011;27:S1-S59.
CCS. 2012 heart failure management guidelines update: Focus on acute and chronic heart failure. Can J Cardiol 2013;29:168-181.
CCS. 2001 Canadian cardiovascular society consensus guideline update for the management and prevention of heart failure. Can J Cardiol 2001;17(suppE):5-24.
CCS 2000 Consensus Conference: Women and ischemic heart disease. Can J Cardiol 2000:17(suppl D).
Harrington RA, Becker RC, Ezekowitz M, et al. Antithrombotic therapy for coronary artery disease: the seventh ACCP conference on antithrombotic and thrombolytic therapy. Chest 2004;126(3 suppl):513s-584s.
Heart valve repair. Available from: http:// www.heartvalverepair.net.
May J, White GH, Harris JP. The complications and downside of endovascular therapies. Adv Surg 2001;35:153-172.
Rutherford RB. Vascular surgery, 4th ed. Toronto: WB Saunders, 1995. Chapter: Atherogenesis and the medical management of atherosclerosis. p222-234.
Schmieder FA, Comerota AJ. Intermittent claudication: magnitude of the problem, patient evaluation, and therapeutic strategies. Am J Card 2001;87(Suppl):3D-13D.
Simpson C, Dorian P, Gupta A, et al. Assessment of the cardiac patient for fitness to drive and fly: executive summary. Can J Cardiol 2004;20:1313-1323.
Task Force on the Diagnosis and Treatment of Peripheral Artery Diseases of the European Society of Cardiology (ESC). Eur Heart J 2011;32:2851-2906.
Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Eur Heart J 2012;33:2551-2567.
Way LW, Doherty GM (editors). Current surgical diagnosis and treatment, 11th ed. Lange Medical Books/McGraw-Hill, 2004.
Welsh RC, Travers A, Huynh T, et al. Canadian Cardiovascular Society Working Group: providing a perspective on the 2007 focused update of the ACC/AHA 2004 guidelines for the management of ST elevation myocardial infarction. Can J Cardiol 2009;25:25-32.
Yang SC, Cameron DE (editors). Current therapy in thoracic and cardiovascular medicine. McGraw-Hill, 2004.

Ambulatory ECG

Kadish AH, Buxton AE, Kennedy HL. ACC/AHA clinical competence statement on electrocardiography and ambulatory electrocardiography: a report of the ACC/AHA/ACP-ASIM task force on clinical competence. Circulation 2001;104:3169-3178.
Krahn A, Klein G, Skane A, et al. Insertable loop recorder use for detection of intermittent arrhythmias. Pacing and Clinical Electrophysiol 2004;27:657-564.
Zimetbaum P, Josephson M. The evolving role of ambulatory arrhythmia monitoring in general clinical practie. Ann Intern Med 1999;130:848-856.

Stress Testing

Gibbons RJ, Balady GJ, Beasley JW, et al. ACC/AHA Guidelines for Exercise Testing. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). JACC 1997;30:260-315.

Echocardiography

Cheitlin M. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiograohy: summary article. J Am Soc Echocardiography 2003;16:1091-1110.
Gowda R, Khan I, Sacchi T, et al. History of the evolution of echocardiography. Int J Cardiol 2004;97:1-6.
Heatlie G, Giles M. Echocardiography and the general physician. Postgrad Med J 2004;80:84-88.
Picano E. Stress echocardiography: a historical perspective. AJM 2003;114:126-130.

Nuclear Cardiology

Beller G, Zaret B. Contributions of nuclear cardiology to diagnosis and prognosis of patients with coronary artery disease. Circulation 2000;101:1465-1478.
Sabharwal N, Lahiri A. Role of myocardial perfusion imaging for risk stratification in suspected or known coronary artery disease. Heart 2003;89:1291-1297.

MR

Danias P, Roussakis A, Ioannidis J. Cardiac imaging diagnostic performance of coronary magnetic resonance angiography as compared against conventional x-ray angiography: a meta-analysis. J Am Col Cardiol 2004;44:1867-1876.
Kim WY, Danias PG, Stuber M, et al. Coronary magnetic resonance angiography for the detection of coronary stenoses. NEJM 2001;345:1863-1869.

CT

Miller JM, Rochitte CE, Dewey M, et al. Diagnostic performance of coronary angiography by 64-row CT. NEJM 2008;359:2324-2336.
Schoepf J, Becker C, Ohnesorge B, et al. CT of coronary artery disease. Radiology 2004;232:18-37.
Somberg J. Arrhythmia therapy. Am J Therapeutics 2002;9:537-542.

Cath/EPS
Conti J. ACC 2005 Annual session highlight. Cardiac arrhythmias. J Am Coll Cardiol 2005;45:B30-B32.
Hayes D, Furman S. Cardiac pacing: how it started, where we are, where we are going. J Cardiovasc Electrophysiology 2004;15:619-627.
Keane D. New catheter ablation techniques for the treatment of cardiac arrhythmias. Card Electrophysiol Rev 2002;6:341-348.
Packer D. Evolution of mapping and anatomic imaging of cardiac arrhythmias. J Cardio Electrophysiol 2004;15:839-854.
Ryan TJ, Faxon DP, Gunnar RM, et al. Guidelines for percutaneous transluminal coronary angioplasty. A report of the American College of Cardiology/American Heart Association Task Force on assessment of diagnostic and therapeutic cardiovascular procedures (subcommittee on percutaneous transluminal coronary angioplasty). Circulation 1988;78:486-502.
Skanes A, Klein G, Krahn A, et al. Cryoablation: potentials and pitfalls. J Cardiovasc Electrophysiol 2004;15:528-534.
Wellens J. Cardiac arrhythmias: the quest for a cure. J Am Coll Cardiol 2004;44:1155-1163.
Zipes D. The year in electrophysiology. J Am Coll Cardiol 2004;43:1306-1324.
Zipes DP, DiMarco JP, Gillete PC, et al. ACC/AHA task force report guidelines for clinical intracardiac electrophysiological and catheter ablation procedures. JACC 1995;26:555-573.

Arrhythmias
Bernstein AD. The NASPE/BPEG pacemaker code. Tex Heart Inst J 1991;18:299-300.
Camm AJ, Kirchof P, Lip GYH, et al. Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Guidelines for the management of atrial fibrillation. Eur Heart J 2010;31:2369-2429.
Gutierrez C, Blanchard DG. Atrial fibrillation: diagnosis and treatment. Am Fam Phys 2011;83:61-68.
Garcia TB, Miller GT. Arrhythmia Recognition: The Art of Interpretation. Sudbury: Jones & Bartlett, 2004.
Thijssen J, Borleffs CJ, van Rees JB, et al. Driving restrictions after implantable cardioverter defibrillator implantation: an evidence-based approach. Eur Heart J 2011;32:2678-2687.
Lip GYH, Nieuwlaat R, Pisters R, et al. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the Euro heart survey on atrial fibrillation. Chest 2010;137:263-72.
Patel MR, Mahaffey KW, Garg J, et al. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. NEJM 2011;365:883-891.
Prystowsky EN, Topol EJ, Califf RM, et al. (editors). Textbook of cardiovascular medicine, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2007. Chapter: Electrophysiology and pacing.

Percutaneous Angiography/PCI
Baim D. New devices for percutaneous coronary intervention are rapidly making bypass surgery obsolete. Curr Opin Cardiol 2004;19:593-597.
Bashore TM, Bates ER, Berger PB, et al. ACC/SCA&I expert concensus document. American College of Cardiology/Society for Cardiac Angiography and Interventions Clinical Expert Consensus Document on Cardiac Catheterization Laboratory Standards. J Am Coll Cardiol 2001;37:2170-214.
Guidelines for percutaneous transluminal coronary angioplasty: a report of the American College of Cardiology/Am Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures Subcommittee on percutaneous transluminal coronary angioplasty. J Am Coll Cardiol 1988;12:529-545.
O'Neil W, Dixon S, Grines C. The year in interventional cardiology. J Am Coll Cardiol 2005;45:1117-1134.
Serruys PW, Morice MC, Kappetain AP, et al. Percutaneous coronary intervention versus coronary-artery bypass grafting for severe coronary artery disease. NEJM 2009;360:961-972.

Cardiovascular Surgery
Alexander P, Giangola G. Deep venous thrombosis and pulmonary embolism: diagnosis, prophylaxis, and treatment. Ann Vasc Surg 1999;13:318-327.
American College of Cardiology. Available from: http://www.acc.org.
Beard JD. Chronic lower limb ischemia. BMJ 2000;320:854-857.
Bojar RM. Manual of perioperative care in cardiac surgery, 3rd ed. Massachusetts: Blackwell Science, 1999.
Cardiology Online. Available from: http://www.theheart.org.
Cheng DCH, David TE. Perioperative care in cardiac anesthesia and surgery. Austin: Landes Bioscience, 1999.
Coulam CH, Rubin GD. Acute aortic abnormalities. Semin Roentgenol 2001;36:148-164.
Crawford ES, Crawford JL, Veith FJ, et al. (editors). Vascular surgery: principles and practice, 2nd ed. Toronto: McGraw-Hill, 1994. Chapter: Thoracoabdominal aortic aneurysm.
Fuchs JA, Rutherford RB (editors). Vascular surgery, 4th ed. Toronto: WB Saunders, 1995. Chapter: Atherogenesis and the medical management of atherosclerosis. p222-234.
Freischlag JA, Veith FJ, Hobson RW, et al. (editors). Vascular surgery: principles and practice, 2nd ed. Toronto: McGraw-Hill, 1994. Chapter: Abdominal aortic aneurysms.
Hallett JW Jr. Abdominal aortic aneurysm: natural history and treatment. Heart Dis Stroke 1992;1:303-308.
Hallett JW Jr. Management of abdominal aortic aneurysms. Mayo Clin Proc 2000;75:395-399.
Harlan BJ, Starr A, Harwin FM. Illustrated handbook of cardiac surgery. New York: Springer-Verlag, 1996.
Heart Valve Repair. Available from: http://www.heartvalverepair.net.
Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. J Am Coll Cardiol 2010;55:e27-e129.
May J, White GH, Harris JP. The complications and downside of endovascular therapies. Adv Surg 2001;35153-35172.
Pitt MPI, Bonser RS. The natural history of thoracic aortic aneurysm disease: an overview. J Card Surg 1997;12(Suppl):270-278.
Powell JT, Brown LC. The natural history of abdominal aortic aneurysms and their risk of rupture. Adv Surg 2001;35:173-185.
Rabi D, Clement F, McAlister F, et al. Effect of perioperative glucose-insulin-potassium infusions on mortality and atrial fibrillation after coronary artery bypass grafting: a systematic review and meta-analysis. Can J Cardiol 2010;26:178-184.
Rosen CL, Tracy JA. The diagnosis of lower extremity deep venous thrombosis. Em Med Clin N Am 2001;19:895-912.
Schmieder FA, Comerota AJ. Intermittent claudication: magnitude of the problem, patient evaluation, and therapeutic strategies. Am J Card 2001;87(Suppl):3D-13D.
Verma S, Szmitko PE, Weisel RD, et al. Clinician update: should radial arteries be used routinely for coronary artery bypass grafting? Circulation 2004;110:e40-e46.
Way LW, Doherty GM. Current surgical diagnosis and treatment, 11th ed. Lange Medical Books/McGraw-Hill, 2004.
Yang SC, Cameron DE. Current therapy in thoracic and cardiovascular medicine. McGraw-Hill, 2004.