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 O₂ 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

![Diagram of the heart with fibrous skeleton](image)

• **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 (O₂ 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)
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 Ca²⁺ 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 Tricuspid 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 Ca²⁺ channels to open →
        - Ca Influx + Ca release from Sarcoplasmic Reticulum
        - ↑[Ca²⁺] 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:**
  
  ![Current direction and wave deflection](image)

  - **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.
o **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.

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

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

**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:

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**Quadrant method**

This chart will help you quickly determine the direction of a patient's electrical axis. Just observe the deflections of the QRS complexes in leads I and aV6. Then check the chart to determine whether the patient’s axis is normal or has a left, right, or extreme axis deviation.
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 & O₂.
  - 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 O₂ or ↑CO₂ 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:
  \[ \text{Flow (F)} = \frac{\text{Pressure Gradient (AP)}}{\text{Resistance}} \]

Effects of Vasomotion on Rate & Velocity of Flow:
- Changes Vessel Diameter:
  - The Flow Rate is directly proportional to the 4\textsuperscript{th} Power of the Vessel Diameter.
  - I.e. Small changes in diameter \(\rightarrow\) Large changes in Flow Rate (by \(\times^4\)).

Factors Influencing Blood Pressure (Long Term):
- Cardiac Output
- Peripheral Resistance
- Blood Volume

\[ \therefore \text{BP} = \text{Cardiac Output} \times \text{Total Peripheral Resistance} \]

Types of Blood Pressures:
- **Systolic**: Peak Aortic pressure during ventricular systole.
- **Diastolic**: Lowest Aortic pressure during ventricular diastole.
- *Pulse 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 \(\rightarrow\) ↑HR & Contractility \(\rightarrow\) ↑MAP
      - Parasympathetic \(\rightarrow\) ↓Heart Rate \(\rightarrow\) ↓MAP
  - 3. Endocrine Mechanisms (Kidney Level):
    - **Antidiuretic Hormone (ADH) – AKA. Vasopressin**:
      - ADH \(\rightarrow\) Water Retention Released \(\rightarrow\) ↑MAP
    - Angiotensin II:
      - AT-II \(\rightarrow\) Vasocostriction \(\rightarrow\) ↑MAP
    - Erythropoietin:
      - EPO \(\rightarrow\) Haematopoiesis \(\rightarrow\) ↑Blood Volume \(\rightarrow\) ↑MAP
    - Natriuretic Peptides (Released by the heart):
      - ↑Stretch on Heart \(\rightarrow\) NP Release \(\rightarrow\) ↑Diuresis \(\rightarrow\) 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 Epithelium)
  - **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.
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.
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
  - **Fenomen 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.