Superior mediastinum:
- SVC → brachiocephalic veins = internal jugular + subclavian
- Arch of aorta, branches:
  - 1st: coronary arteries
  - 2nd: right brachiocephalic
  - 3rd: left common carotid
  - 4th: left subclavian
- Ligamentum arteriosum:
  - Remnant of ductus arteriosus
  - Connecting pulmonary artery and aorta
- Opposite the branch of left subclavian artery

Nerves
- Vagus - posterior to hilum of lung
  - Left: under arch of aorta to give recurrent laryngeal nerve → passes posteriorly to give cardiac & oesophageal plexi and pass into abdomen
  - Right: under right subclavian to give recurrent laryngeal nerve → passes posterior to give oesophageal plexus
- Phrenic nerves
  - Left and right both run anterior to hilum of lung and over anterior surface of heart
    - Right passes through diaphragm with IVC
    - Left innervates from above diaphragm

Recurrent laryngeal nerve palsy
(secondary to cardiac/thyroid surgery):
- Hoarse voice
- Vocal cord palsy
- Airway obstruction

Position of the heart in the chest
- Anterior & inferior border: right atrium + right ventricle
- Left border: left ventricle
- Posterior: left atrium + left ventricle
- Right border: right atrium + right ventricle

Anterior view of the heart

Arch of the aorta and its initial branches
Cardiac anatomy

- **Fibrous skeleton** at atrio-ventricular junction level

- **Right atrium**, contains:
  - Sino-atrial node (SAN) in wall of RA
  - SVC
  - IVC
  - Coronary sinus opens into RA
  - Fossa ovalis (remnant of foramen ovale) in atrial septum

- **Right ventricle**:
  - Tricuspid valve between RA and RV
  - **Infundibulum** is area proximal to the pulmonary valve (semi-lunar valve with 3 cusps)
  - **Pulmonary trunk passes anterior** to aorta → then divides into right and left pulmonary arteries

- **Left** side:
  - Atrium receives blood from 4 pulmonary veins
  - Mitral valve held in place by **chordae tendinae** (fibrous strands that attach cusps) connected to **papillary muscles**
  - Aortic arch loops around right pulmonary artery, moving posteriorly
  - Atrial auricles are extensions that open in high cardiac-output states

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**Posteromedial papillary muscle rupture**, secondary to inferior (right coronary) MI results in acute mitral valve incompetence, causing left ventricular failure
Coronary anatomy

- Aortic sinuses (of Valsalva): bulges in wall of aorta distal to the valve origin of right and left coronary artery

- Right coronary artery (RCA) divides into:
  - Right marginal artery
    - Runs over the lateral aspect of right ventricle
  - Posterior interventricular artery (PIV)

- RCA curves around to the posterior atrio-ventricular groove
- RCA supplies:
  - Right atrium & ventricle
  - Sinoatrial node (SAN)
  - Atrioventricular node (AVN)
  - Posterior interventricular septum

- Left coronary artery (left main stem) initially as the left main stem then becomes:
  - Left circumflex artery (LCx)
    - Runs in posterior atrio-ventricular groove
    - Gives off the left marginal artery
    - Runs over lateral aspect of left ventricle
  - Anterior interventricular artery (AIV, previously known as left anterior descending [LAD])

- LCA supplies:
  - Left atrium
  - Left ventricle
  - Anterior part of interventricular septum

Myocardial infarction

- Anterior MI = anterior interventricular artery
  - V1-V4
- Lateral MI = left marginal artery
  - I, aVL, V5-V6
- Antero-lateral MI = left main stem
  - I, aVL, V1-V6
- Inferior MI = right coronary (marginal)
  - II, III, aVF
- Posterior MI = posterior interventricular artery
  - V1-3 (ST-depression & upright T-waves)
Cardiac venous drainage

- **Coronary sinus**: in posterior atrioventricular groove, empties into right atrium.
- **Great cardiac vein**: in anterior interventricular groove and curves posteriorly to join coronary sinus
- **Middle cardiac vein**: in posterior interventricular groove
- **Short (small) cardiac vein**: lateral aspect of right ventricle

AVN dysfunction causes heart block, where atrial depolarisation is not conducted to the ventricles. Most due to ischaemic cardiomyopathy, but also with digoxin or beta-blocker overdose.

Ischaemic damage to part of the bundle of His causes bundle branch block. Ventricular contraction is less co-ordinated and this is seen as a prolonged QRS (>120ms) on ECG.

Electrical system of heart

- Initiated in **sino-atrial node** (SAN), then spreads across atria
  - No organised conduction system
- Reaches **atrio-ventricular node** (AVN), which **delays** the action potential to allow efficient ventricular filling
- Passes into specialised conducting fibres (**Purkinje fibres** - wide and no glycogen) in the **bundle of His**
- Divides into 3: right, left anterior, and left posterior bundles
  - Action potential passes to apex rapidly
  - Contraction from apex to base and endomycium to epimycium
Myocyte Ultrastructure
- Striated muscle
- Branching fibres
- Intercalated disks
- T-Tubules

Cardiac myocytes

Myocyte physiology
- Negative resting membrane potential
- Intracellular ions:
  - High $K^+$
  - Low $Na^+$
  - Low $Ca^{2+}$
- Calcium stored in sarcoplasmic reticulum by active uptake via SERCA pump

Pacemaker cells - electrically unstable
- Pre-potential $\uparrow P_{Na^+}$ (high sodium permeability - due to ‘funny channels’) causes a slow rise in membrane potential [4] (also known as ‘funny current’)
- Eventually passes threshold $[0] \rightarrow$ action potential fires due to opening of voltage-gated calcium channels
  - Causing $\uparrow P_{Ca^{2+}}$, and $Ca^{2+}$ influx
- At the peak of the action potential (AP) the voltage-gated $K^+$ channels open $[1] \rightarrow \uparrow P_{K^+}$
  - [No (plateau) phase 2]
  - This causes $K^+$ efflux $\rightarrow$ re-polarisation [3]

Cardiac muscle histology
- Gap junctions
- Desmosome junctions
- Adherens junctions

- Right ventricle forms inferior border; left ventricle forms left border
- Left main stem divides to circumflex (lateral wall) and anterior interventricular (anterior septum)
- Right coronary supplies SAN and posterior septum
- Coronary sinus receives all cardiac venous drainage
Ventricular cells
- Electrically stable [4]- under steady conditions there is no change membrane potential
- When threshold is passed, fast voltage-gated Na$^+$ channels open [0]:
  - $\uparrow$P$_{Na^+}$, with Na$^+$ influx
- Fast Na$^+$ close, causing a small fall in membrane potential [1]
- Slow Na$^+$ & Ca$^{2+}$ voltage-gated channels open, causing the plateau [2]:
  - $\uparrow$P$_{Ca^{2+}}$, with sodium and calcium influx
- Voltage-gated K$^+$ open during the plateau phase:
  - $\uparrow$P$_{K^+}$ → Re-polarisation and refractory periods [3] then returns to resting $E_m$ [4]

Refractory periods

Absolute
- Fast Na$^+$ channels in ‘inactive’ state
- No AP regardless of stimulus intensity
- Insufficient channels

Relative
- Some fast Na$^+$ channels available
- AP possible with large stimulus

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<thead>
<tr>
<th></th>
<th>Sympathetics</th>
<th>Parasympathetics</th>
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<tbody>
<tr>
<td>Heart rate</td>
<td>Increase</td>
<td>Decrease</td>
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<tr>
<td>AVN delay</td>
<td>Decrease</td>
<td>Increase</td>
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<tr>
<td>Ventricular contractility</td>
<td>Increase</td>
<td>No effect</td>
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<tr>
<td>Receptor involved</td>
<td>β-1-adrenoreceptor</td>
<td>Muscarinic acetylcholine</td>
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<tr>
<td>Mechanism</td>
<td>Increase cAMP</td>
<td>Reduce cAMP</td>
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<tr>
<td></td>
<td>Increase Na$^+$ &amp; Ca$^{2+}$ permeability</td>
<td>Increase K$^+$ permeability</td>
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