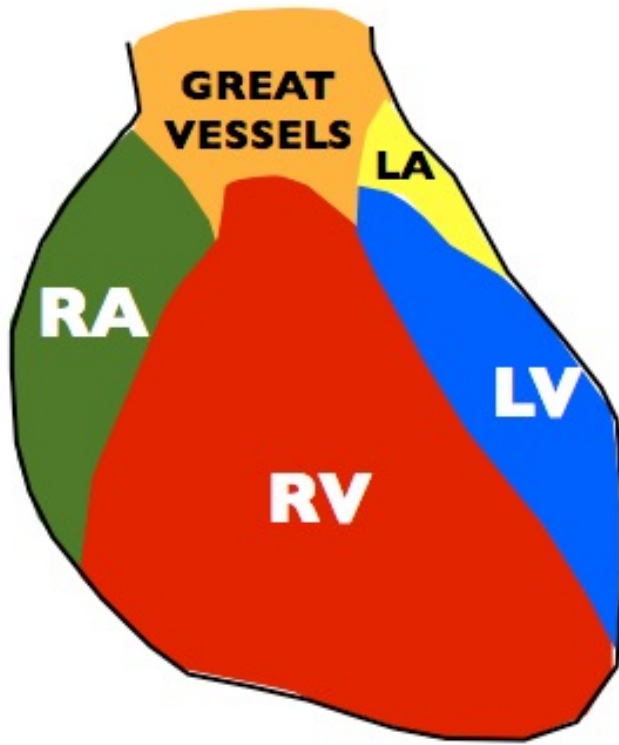
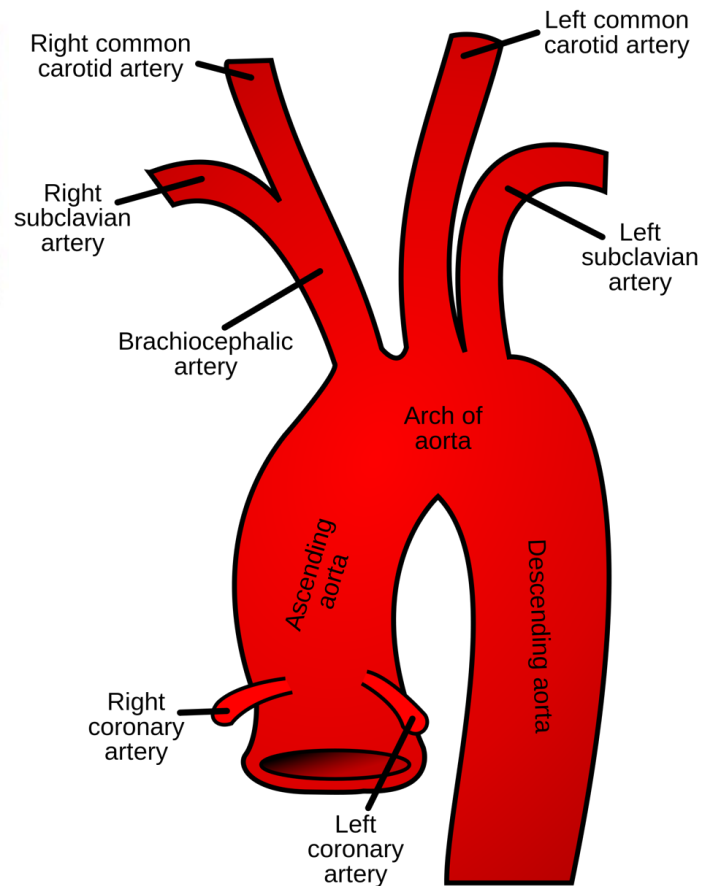


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#### Position of the heart in the chest

- **Anterior & inferior** border: right atrium + right ventricle
- **Left** border: left ventricle
- **Posterior**: left atrium + left ventricle
- **Right** border: right atrium + right ventricle



Arch of the aorta and its initial branches

#### Superior mediastinum:

4

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

5

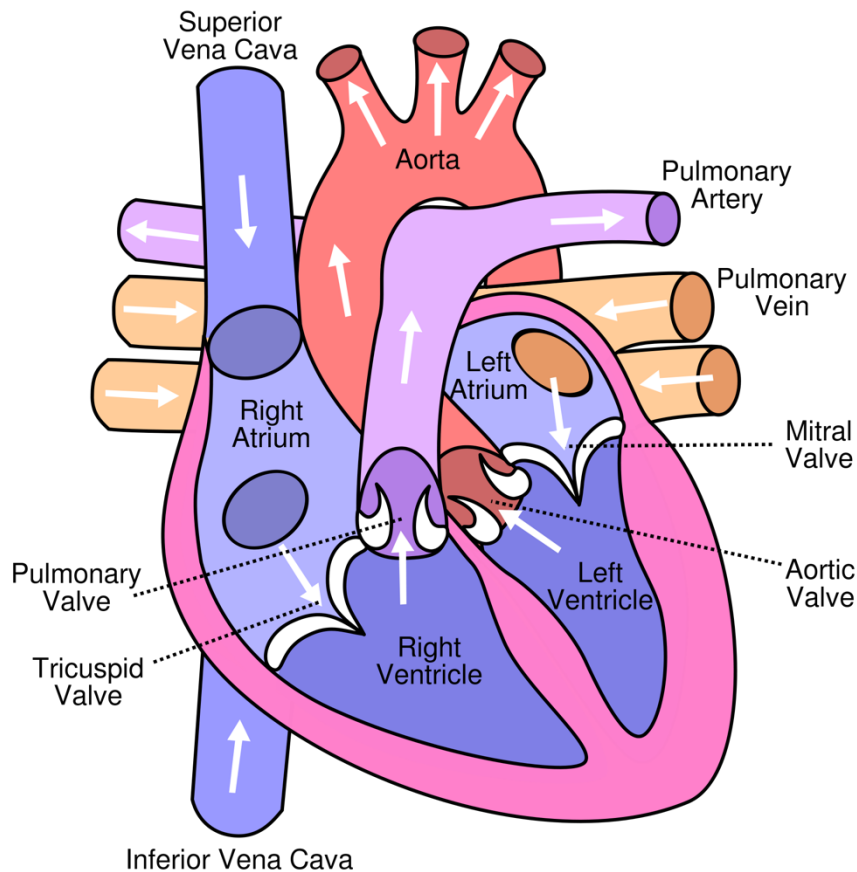
#### Nerves

- 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



## 6

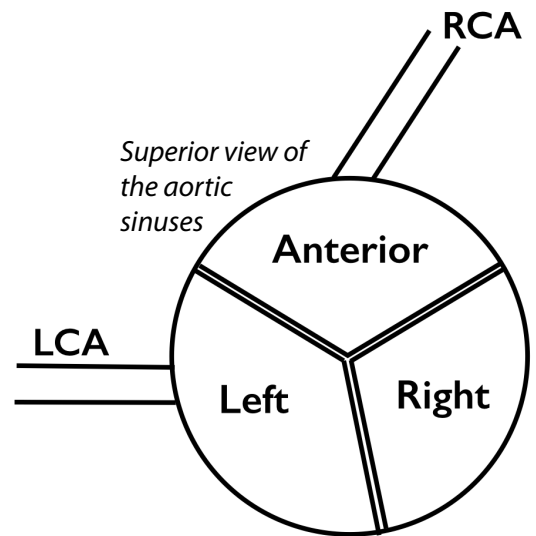
### Cardiac anatomy

- **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

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

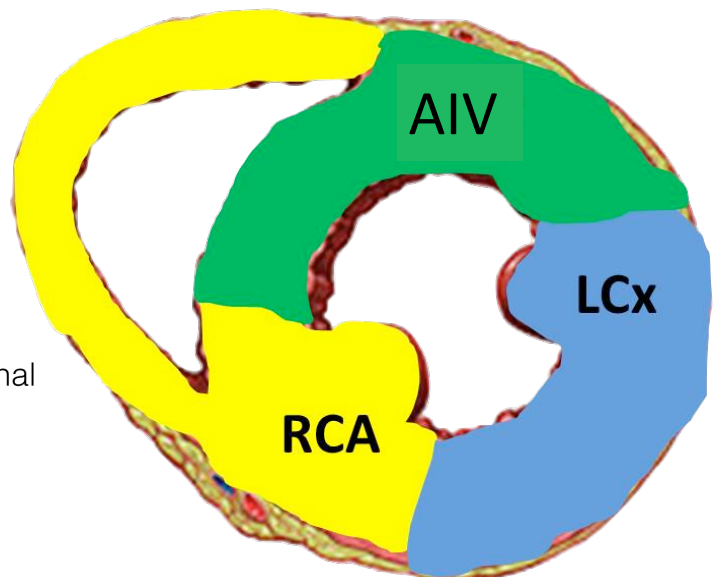
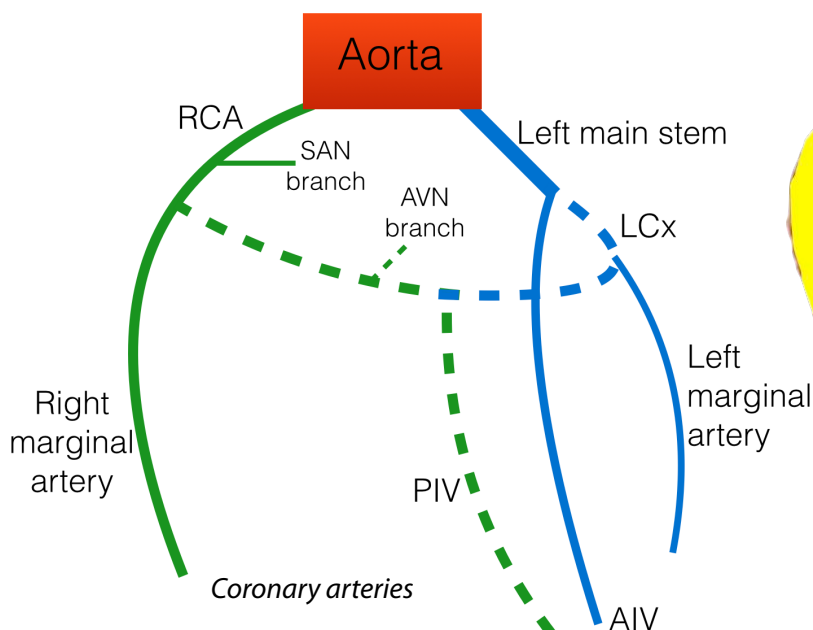
## Coronary anatomy

- **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 (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)

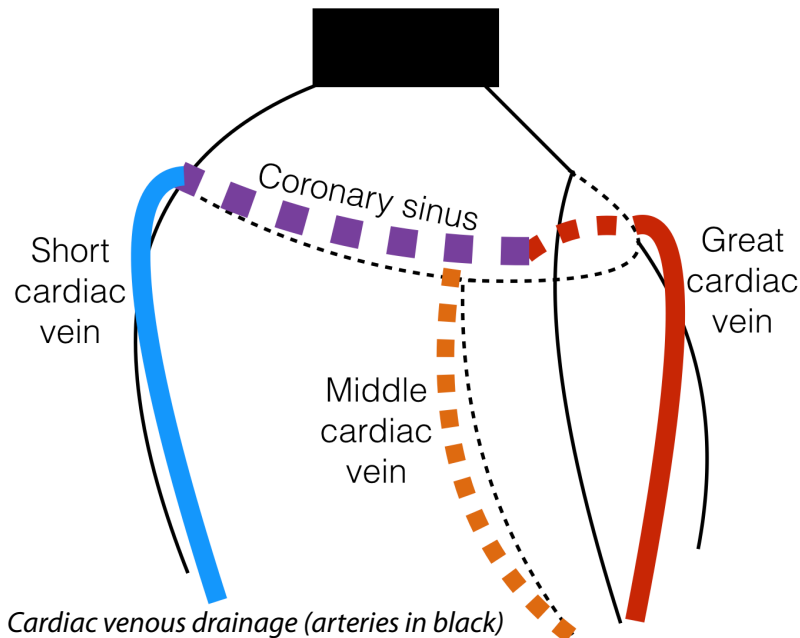


Blood supply to the myocardium

## 8

Cardiac venous drainage

- **Coronary sinus:** in posterior atrio-ventricular 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



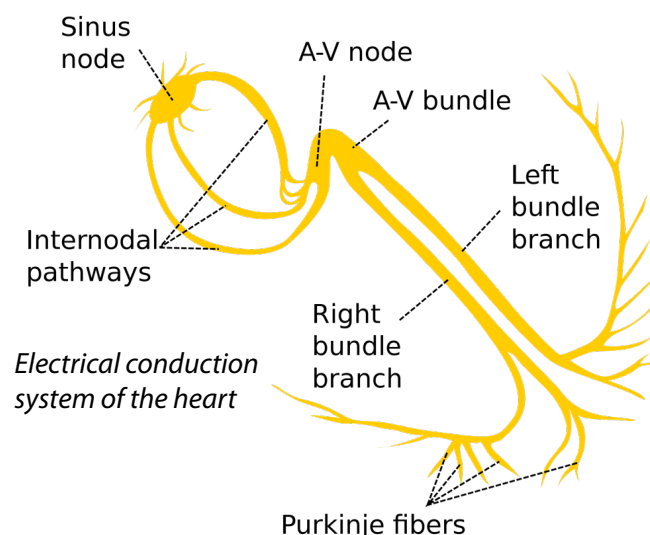
**AVN dysfunction** causes **heart block**, where atrial depolarisation is not conducted to the ventricles. Most due to ischaemic cardiomyopathy, but also with digoxin or beta-blocker overdose.

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.

## 9

Electrical system of heart

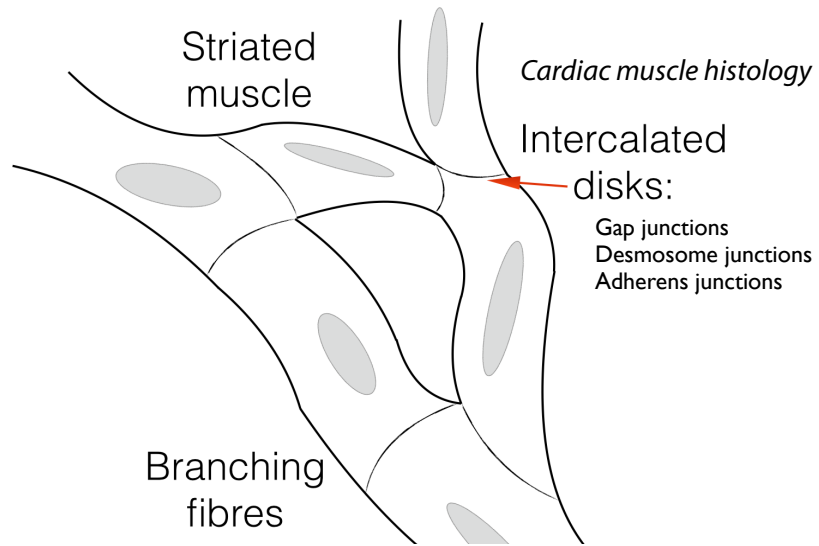
- Initiated in **sino-atrial node** (SAN), then spreads across atria
  - No organised conduction system
- Reaches **atrio-ventricular node** (AVN), which **delays** the action potential to allow efficient ventricular filling
- Passes into specialised conducting fibres (**Purkinje fibres** - wide and 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



10

### Myocyte Ultrastructure

- Striated muscle
- Branching fibres
- Intercalated disks:
- T-Tubules



- **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

## Cardiac myocytes

11

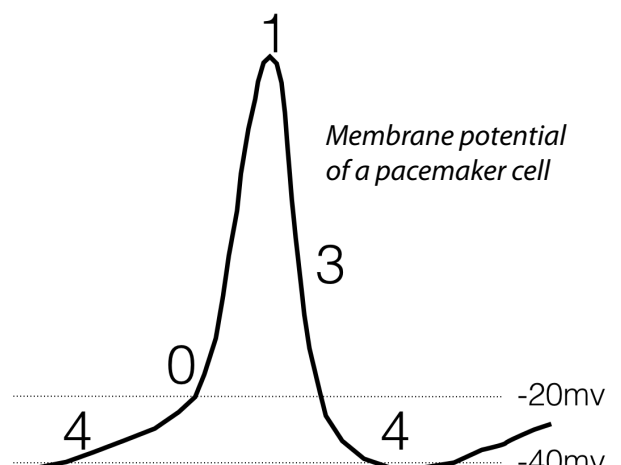
### Myocyte physiology

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

12

### Pacemaker cells - electrically unstable

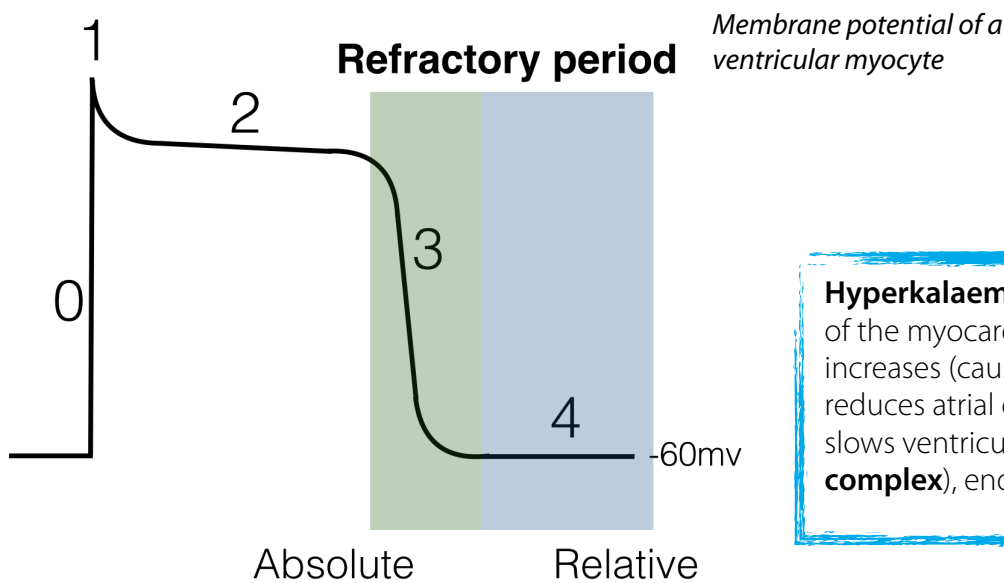
- **Pre-potential**  $\uparrow P_{Na^+}$  (high sodium permeability - due to 'funny channels') causes a slow rise in membrane potential [4] (also known as 'funny current')
- Eventually passes **threshold** [0]  $\rightarrow$  action potential fires due to opening of **voltage-gated calcium channels**
  - 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]



13

Ventricular cells

- **Electrically stable** [4]- under steady conditions there is no change membrane potential
- When threshold is passed, **fast voltage-gated Na<sup>+</sup> channels** open [0]:
  - $\uparrow P_{Na^+}$ , with Na<sup>+</sup> influx
- **Fast Na<sup>+</sup> close**, causing a small fall in membrane potential [1]
- **Slow Na<sup>+</sup> & Ca<sup>2+</sup> voltage-gated channels** open, causing the plateau [2]:
  - $\uparrow P_{Ca^{2+}}$  with sodium and calcium influx
- **Voltage-gated K<sup>+</sup>** open during the plateau phase:
  - $\uparrow P_{K^+} \rightarrow$  Re-polarisation and refractory periods [3] then returns to resting  $E_m$  [4]



**Hyperkalaemia** increases the 'refractoriness' of the myocardium. Hyperpolarisation [3] increases (causing **tall, tented T-waves**), reduces atrial depolarisation (**flat P-waves**), slows ventricular depolarisation (**broad QRS complex**), ending in asystole.

14

Refractory periods*Absolute*

- Fast Na<sup>+</sup> channels in 'inactive' state
- No AP regardless of stimulus intensity
- Insufficient channels

*Relative*

- Some fast Na<sup>+</sup> channels available
- AP possible with large stimulus

15

	Sympathetics	Parasympathetics
<i>Heart rate</i>	Increase	Decrease
<i>AVN delay</i>	Decrease	Increase
<i>Ventricular contractility</i>	Increase	No effect
<i>Receptor involved</i>	$\beta$ -1-adrenoreceptor	Muscarinic acetylcholine
<i>Mechanism</i>	Increase cAMP Increase Na <sup>+</sup> & Ca <sup>2+</sup> permeability	Reduce cAMP Increase K <sup>+</sup> permeability