Cardiac

Myoctes

- Striated muscle
- Connected by intercalated discs
- Gap junctions pass APs along
- Single nucleus

Baroreceptors

↑ stretch = ↑ rate of firing (parasympathetic output)

- *Carotid sinus/body* —> glossopharyngeal n (IX) —> vasomotor centre in brainstem
  - Responds to both increase and decrease in pressure
  - ↑ pressure = ↑ firing rate via CN IX —> ↑ parasympathettic tone to vasomotor centre in brainstem
- *Aortic arch —> vagus n (CN X)*

Vasoconstricting Agents	Vasodilating Agents
Endothelin-1	Nitric oxide
Thromboxane A2	Prostacyclin
Angiotensin II	Beta-agonists
Noradrenaline (alpha 1-receptors)	Calcium-channel blockers

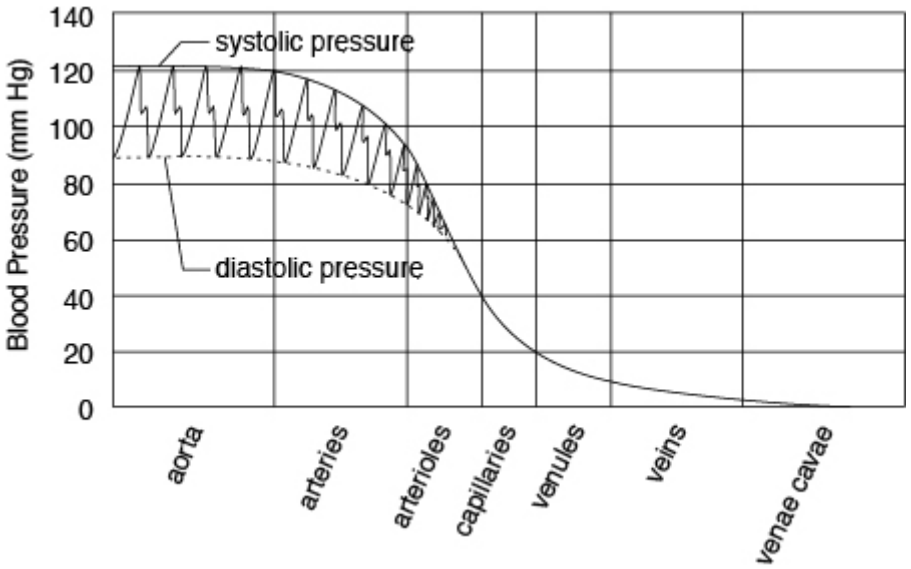
Blood volume around 70ml / kg
Stroke volume 70ml in average male
Cardiac output around 5L/min in average male

MAP and pulse pressure:

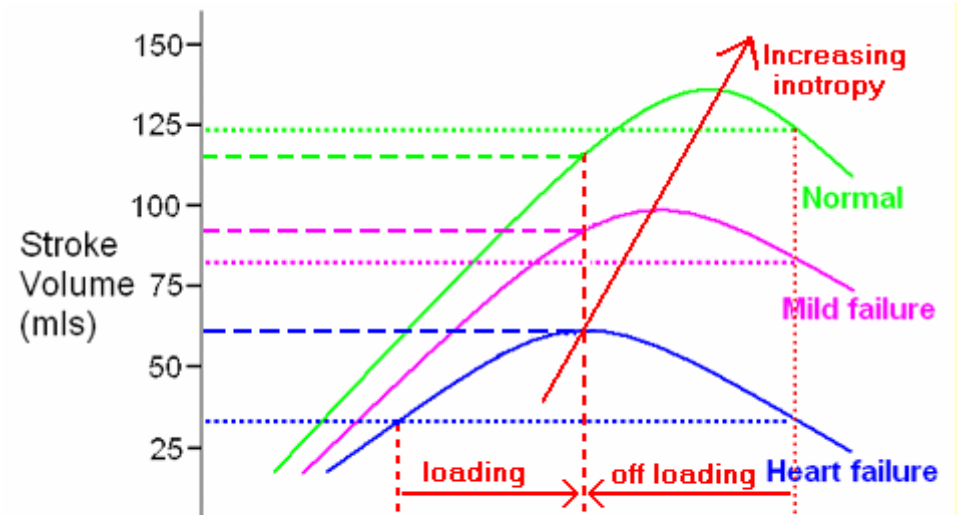
- Pulse pressure = difference between SBP and DBP
- Pressure gradient:
  - The pressure at aortic arch/arterial = around 120mmHg
  - Pressure at the IVC is around ZERO mmHg
- Mean arterial pressure:
  - Diastolic + a third of the pulse pressure
  - Reason that we cant just average out pressures is because heart is in diastole around 60% of time
  - MAP is considered to be the perfusion pressure of the body, clinically
  - MAP normally 65-110mmHg - even a reduction for a minute below this can have ischaemic effects on end organs

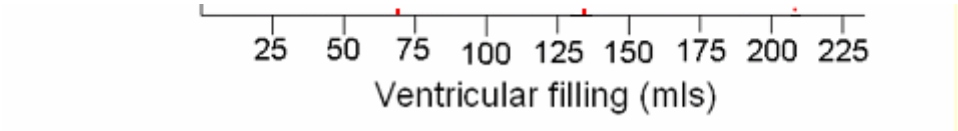
Normal distribution of cardiac output at rest

Organ	Percentage of cardiac output
Liver	25%
Kidneys	22%
Muscle	20%
Brain	14%
Heart	5%
Rest of the body	14%



Frank starling curve





Reduced cardiac output or heart failure *moves the curve of the frank starling curve* ***downward***

Myocyte

- Resting potential: -90 mV
- Threshold: -70 mV

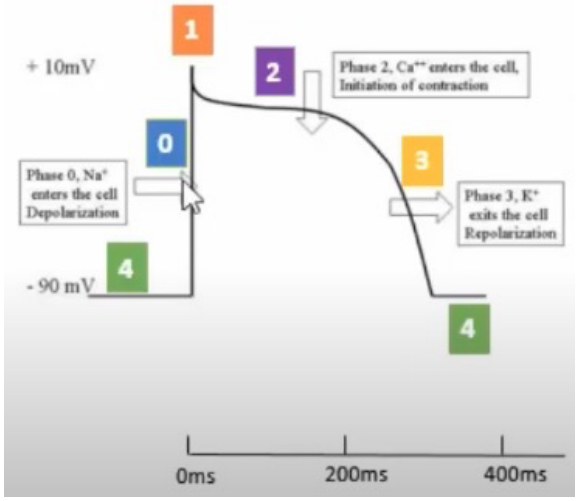
Pacemaker

- Resting potential: -60-70 mV
- Threshold: -40 mV

<u>Ventricular myocyte action potential</u>

Resting potential -90mv. Action potential 200-400ms long

Phase 4 = diastole. 0-3 = ventricular contraction

- <u>Phases</u>
- 

  - **0** - Na influx depolarises cell to +10 to 20 mV
  - **1** - Na channels close, K+ leak out of cell continues (cellular K > extracellular K)
  - **2** - Slow, voltage gated Ca2+ channels open, = **plateau phase -** as K+ is leaking out of cell and Ca2+ into cell (intracellular Ca2+ very low)
    1. Calcium binds to troponin C
    2. Causes conformational change in troponin-tropomyosin complex
    3. —> causes complex to move out of actin filament and expose myosin binding site
    4. —> myosin binds —> muscle contraction occurs
  - **3** - Ca2+ channels close, slow K+ channels open (+ leak of K+) —> repolarisation of cell
  - **4** - Return to resting membrane potential of -90mv

ROLE OF CALCIUM

- 25% of intracellular Ca2+ rise is from entry during action potential - the rest is from the efflux into cell from SR
  1. **Contraction**

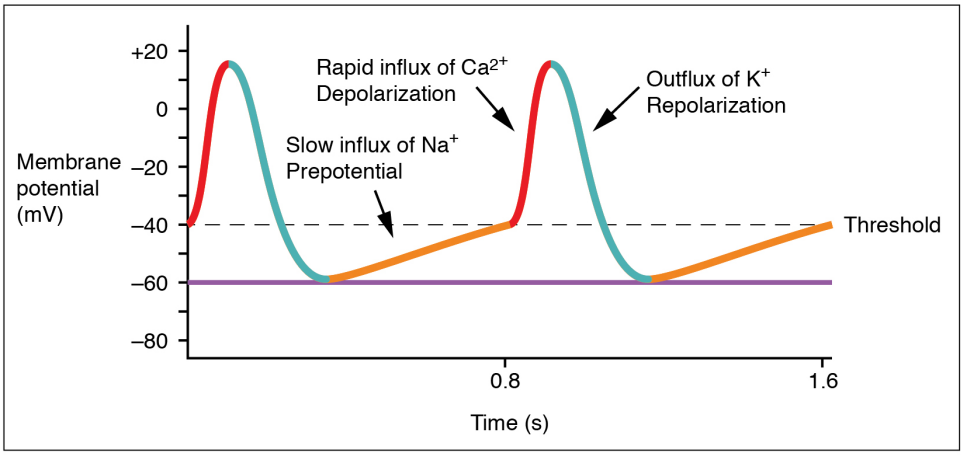
1. Influx of calcium into the cell from AP —> cause Ca2+ to be released from sarcoplasmic reticulum
2. Ca2+ floods into cytosol (amount depends on initial influx from AP)
    1. Calcium binds to troponin C
    2. Causes conformational change in troponin-tropomyosin complex
    3. —> causes complex to move out of actin filament and expose myosin binding site
    4. —> myosin binds —> muscle contraction occurs

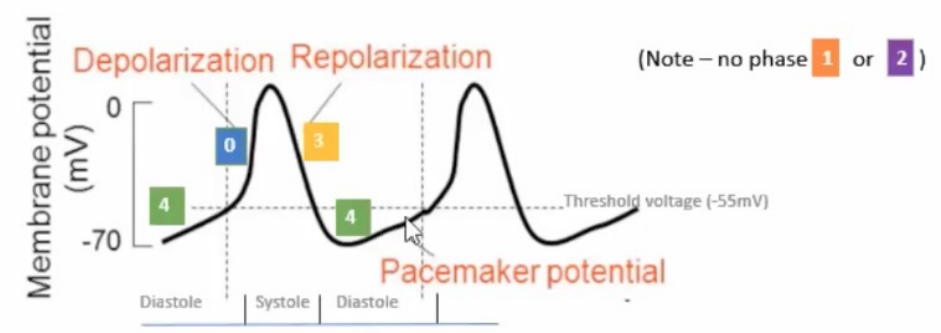
2. **Relaxation**
    1. 80% of Ca2+ is pumped BACK into the SR by Ca2+ ATPase pumps
    2. 20% is pumped out of cell by Na+/Ca2+ ATPase pumps
3. **Treppe effect**
    1. ↑ action potentials per unit time = ↑ calcium left in the cytosol and SR = ↑ HR and ↑ contractility

<u>Pacemaker/SA node action potential</u>

Resting potential 70 mV, action potential threshold 40-55 mV

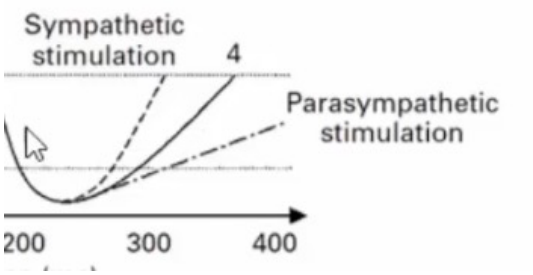
No phase 1 or 2



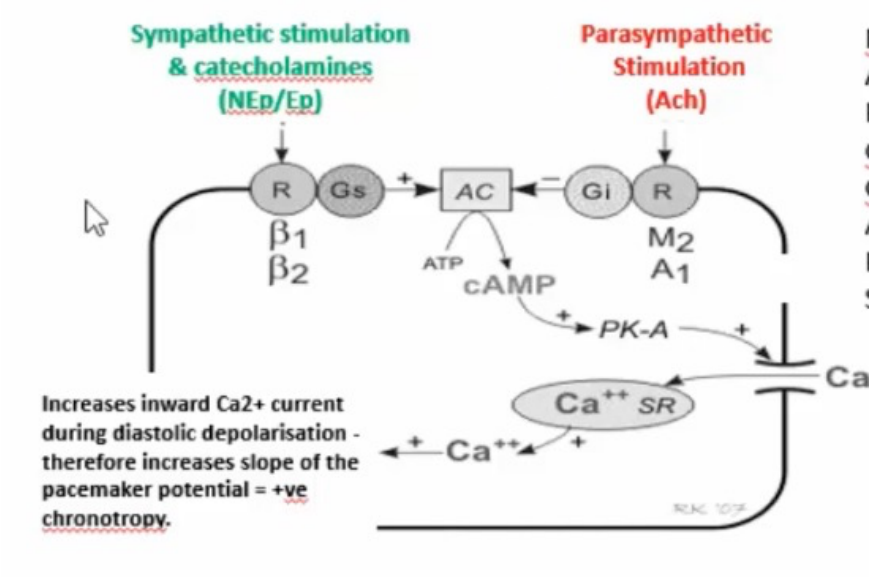


<u>Phases</u>

- 0 - T-type Ca2+ channel opens when threshold of 40mV is reached. Depolarises cell
- 3 - T-type channels close, slow K+ channels open —> starts to repolarise cell
- 4 - There is no resting potential, but instead a continually depolarising **pacemaker potential.**
    - Slow inward Na leak (and to a lesser extent, slow inward Ca2+ inward leak), causing a constant depolarisation toward threshold

- The steepness of this pacemaker potential dictates the HR
- 

SA node control - second messenger



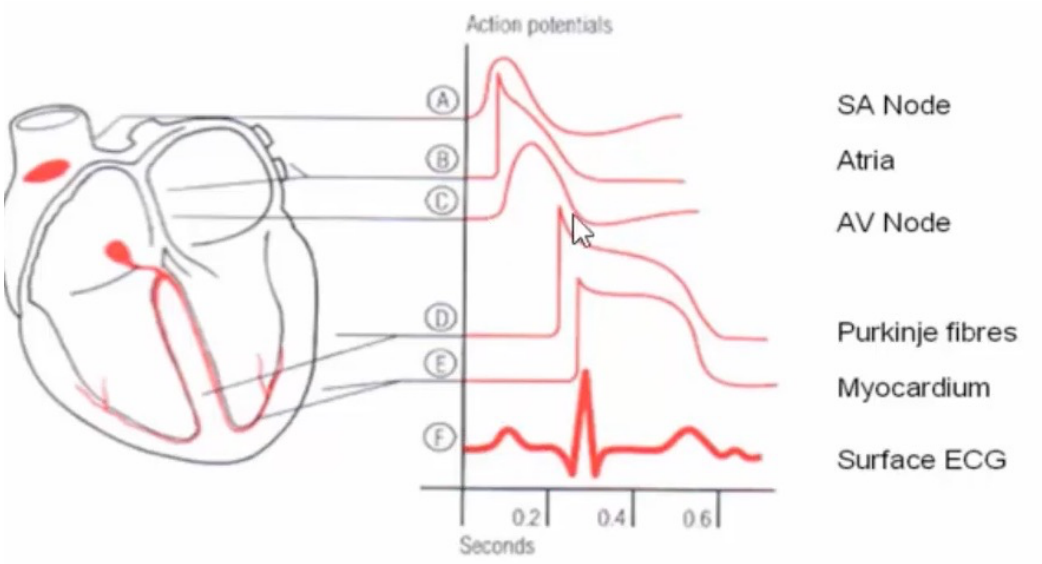
- Stimulation (G-protein linked adrenoreceptors)
  1. Adrenaline / noradrenaline bind beta-adrenergic receptors
  2. —> stimulates G-protein to activate adenyl cyclase
  3. —> adenyl cyclase converts ATP —> cAMP
  4. —> cAMP induces conformational change on L-type Ca2+ channel on cell
  5. —> influx of Ca2+ —> ↑ Ca2+ from SR = ↑ chronotropy of SA node cells and HR
- Inhibition
  1. Acetylecholine binds muscarinic receptors
  2. —> inhibits G-protein

SA conduction

1. SA - fast spread across R and L atrium via inter-nodal tracts
2. SA —> AV
   1. AV node has slowest conduction (120-200ms) = PR interval
   2. Slow conduction allows atrial contractions to finish before ventricular
3. AV—> HIS bundle (fast conduction)
   1. Annulus fibrosus separates atrium and ventricle. Must travel via bundle of HIS
4. HIS —> (depolarises L to R)
   1. L bundle
      1. L posterior fascicle —> purkinje fibres at lateral and posterior of L ventricle
      2. L anterior fascicle —> purkinje fibres at apex L ventricle
   2. R bundles

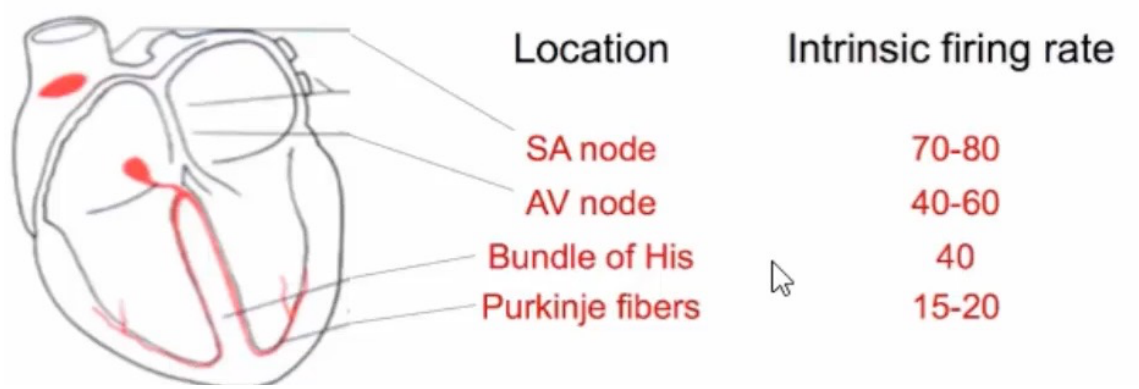
5. Bundles —> purkinje fibres
6. Purkinje —> ventriclular muscles

Action potential relations to ECG



- Note AV node corresponds to PR interval
- Plateau phase of purkinje/myocardium = ventricular contraction

INTRINSIC FIRING RATES



- P wave - atrial depolaristaion
- Q wave - inter ventricular depolarisation
- R wave - early ventricular depolarisation
- S wave - late ventricular depolarisation
- T wave - ventricular re-polarisation
- QT = total time of de and re polarisation of ventricles

RAD

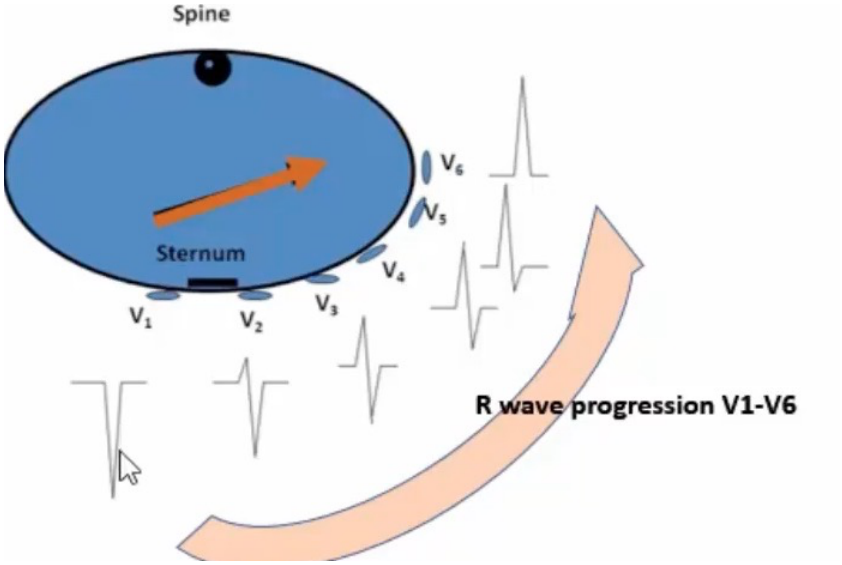
- Anything that causes hypertrophy or ↑ contraction of Rt ventricle
- R ventricular hypertrophy
- Rt heart strain e.g. PE
- Cor pulmonale

- L posterior fascicular block

LAD

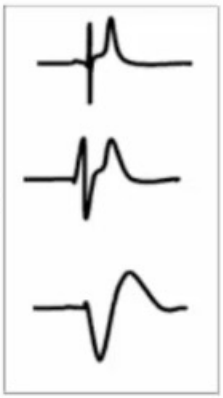
- L ventricular hypertrophy
- L anterior fascicular block

R wave progression = most negative in V1 and most positive in V6



- **Hyperkalaemia**
  - Tall, tented narrow T waves
  - decreased P wave amplitude
  - widened QRS complexes
  - AV block
  - absent P waves
  - very broad, sinusoidal QRS
  - VT/VF

worsening hyperkalaemia



- **hypokalaemia** – (widening T waves, prolonged PR interval, ST depression, U waves and ventricular ectopics – ultimately SVT/VT)
- **hypercalcaemia** (shortened QT)
- **hypocalcaemia** (prolonged QT)

Important medical conditions & the ECG

- **Hypothyroidism** - bradycardia, low voltage QRS, QT prolongation, AV block
- **Hyperthyroidism** – sinus tachy, AF, LVH by voltage criteria (without strain)

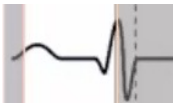
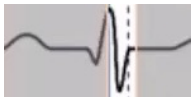
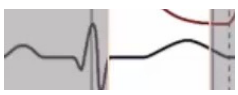
- **Hypothermia** – J (Osborn) waves
- **Tricyclic antidepressants** – wide QRS complexes, dominant R wave in aVR
- **Differential of ST elevation** – STEMI, LBBB, pericarditis, vasospasm, LV aneurysm
- **Digitalis** – bradycardia, 'reverse tick' ST depression, AV block
- **PE** – Sinus tachycardia, RV strain, AF, S1 Q3 T3,
- **SAH** – tachycardia, widespread ST/T changes

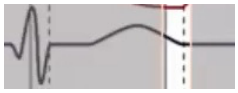
Normal pressures - must know (SBP / DBP)

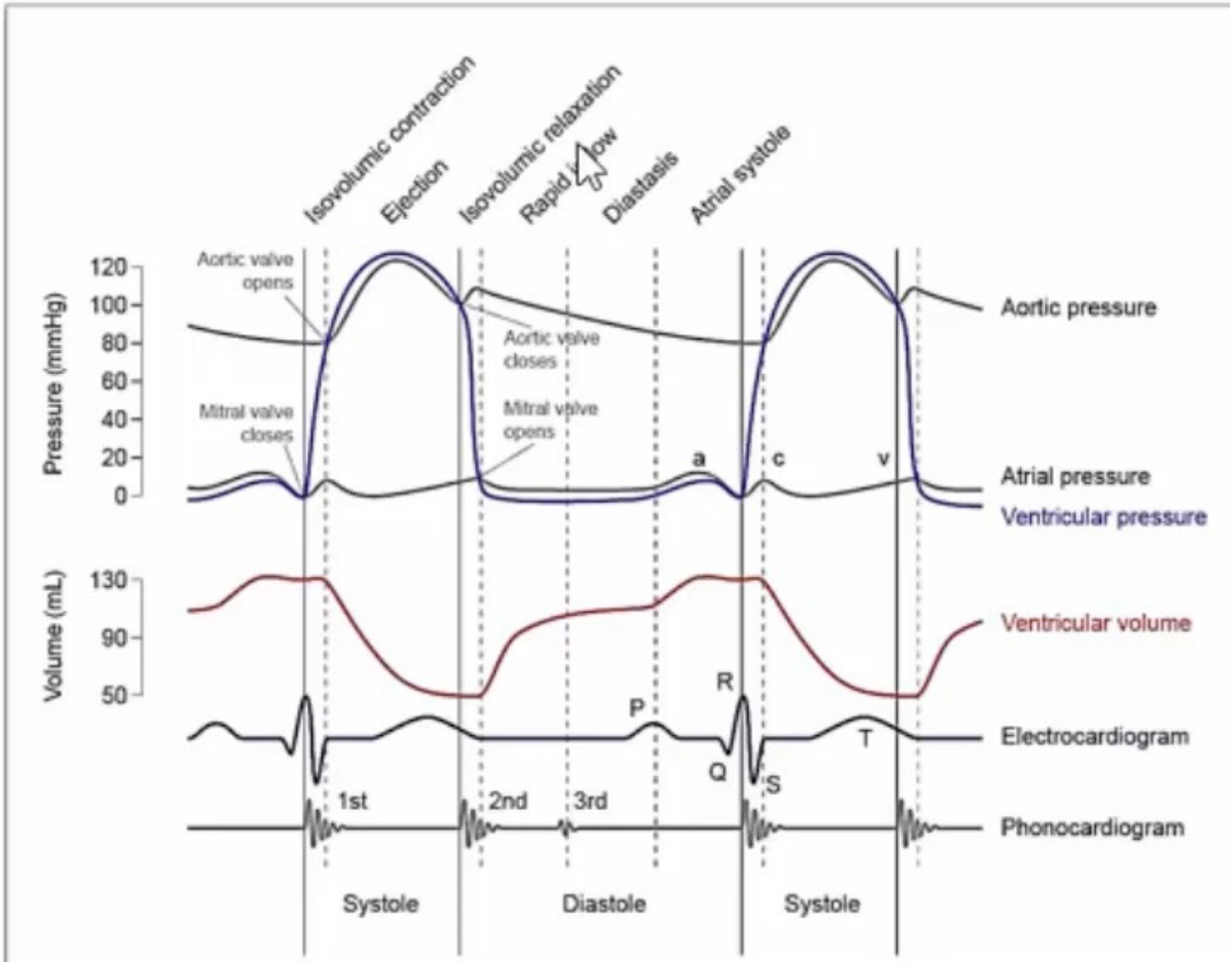
- Right Atrium: 5 / 3 mmHg
- Left Atrium: 10 / 8 mmHg
- Right Ventricle: 28 / 2 mmHg
- Left Ventricle: 125 / 6 mmHg
- Aorta: 120/70 mmHg
- Pulmonary Artery: 25 / 10mmHg

CARDIAC CYCLE

Four stages

1. **Atrial contraction** - PR interval
   - 
   - 20-30% of ventricular filling comes from atrial contraction
   - End-diastolic volume around 120-30 mls
   - 5mmHg for atria
2. **Isovulmentric ventricular contraction** - QRS
   - 
   - R ventricular pressure > pulmonary artery pressure (25mmHg) —> EJECTION
   - L ventricular pressure > aortic pressure (80mmHg) —> EJECTION
3. **Ejection** - ST
   1. 

4. **Isovolumetric ventricular relaxation -**
    1. 
5. **Ventricular filling**
    1. 



JVP

Internal jugular vein - no valves between R atrium and top of internal JV = proxy of right sided atrial pressure

>3cm above sternal notch is abnormal

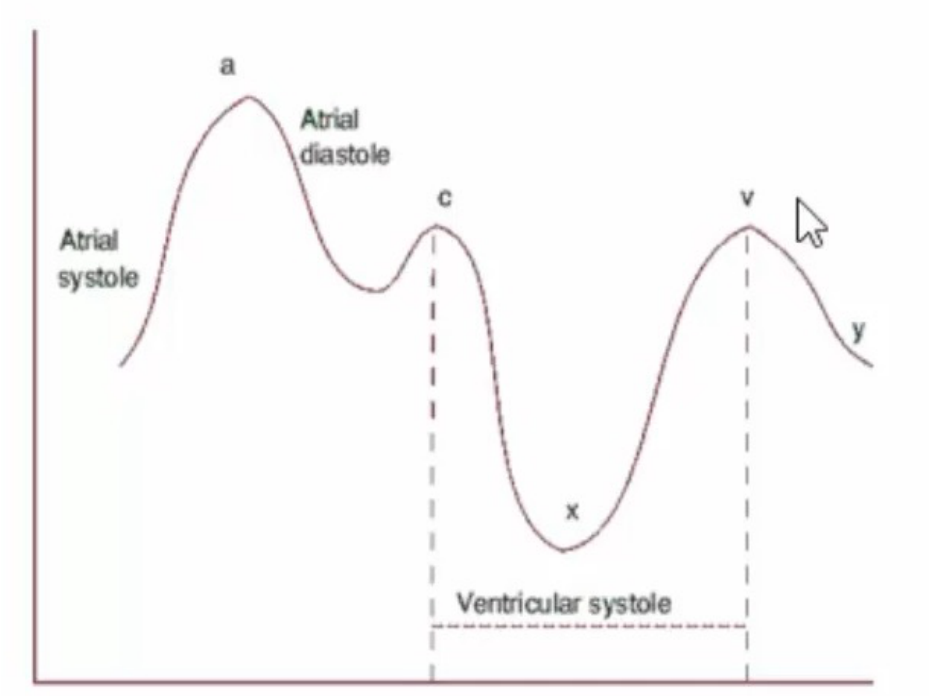
Causes of ↑JVP

- Cor pulmonale
- Rt sided heart failure
- Pericarditis
- Tamponade
- Tricuspid stenosis

Phases of the JVP

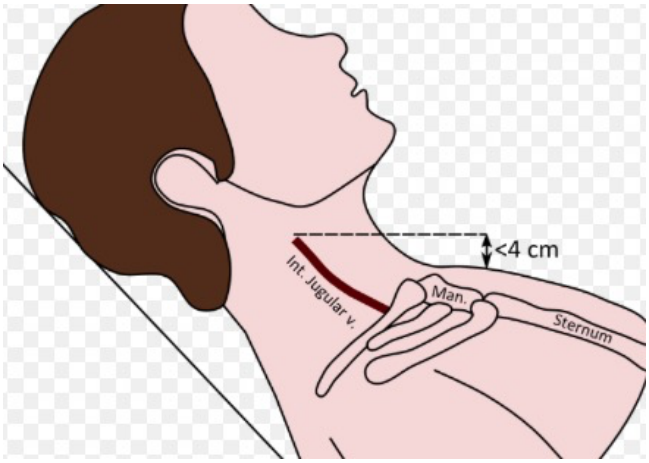
- **A wave** - atrial contraction - increased in R atrial hypertrophy and tricuspid stenosis
- **C wave** - R ventricular contraction. Shutting of tricuspid valve, bulges into atrium in ventricular contraction (NOT VISIBLE TO EYE)
- **X -** ventricular ejection
- **V wave -** atrial filling against closed tricuspid valve

- **Y** - tricuspid valve opening and filling of ventricle - increased in tricuspid incompetence



Measuring JVP

1. Patient at 45°
2. Measure from sternal notch to top visible part of JVP
3. Should be less than 3-4cm



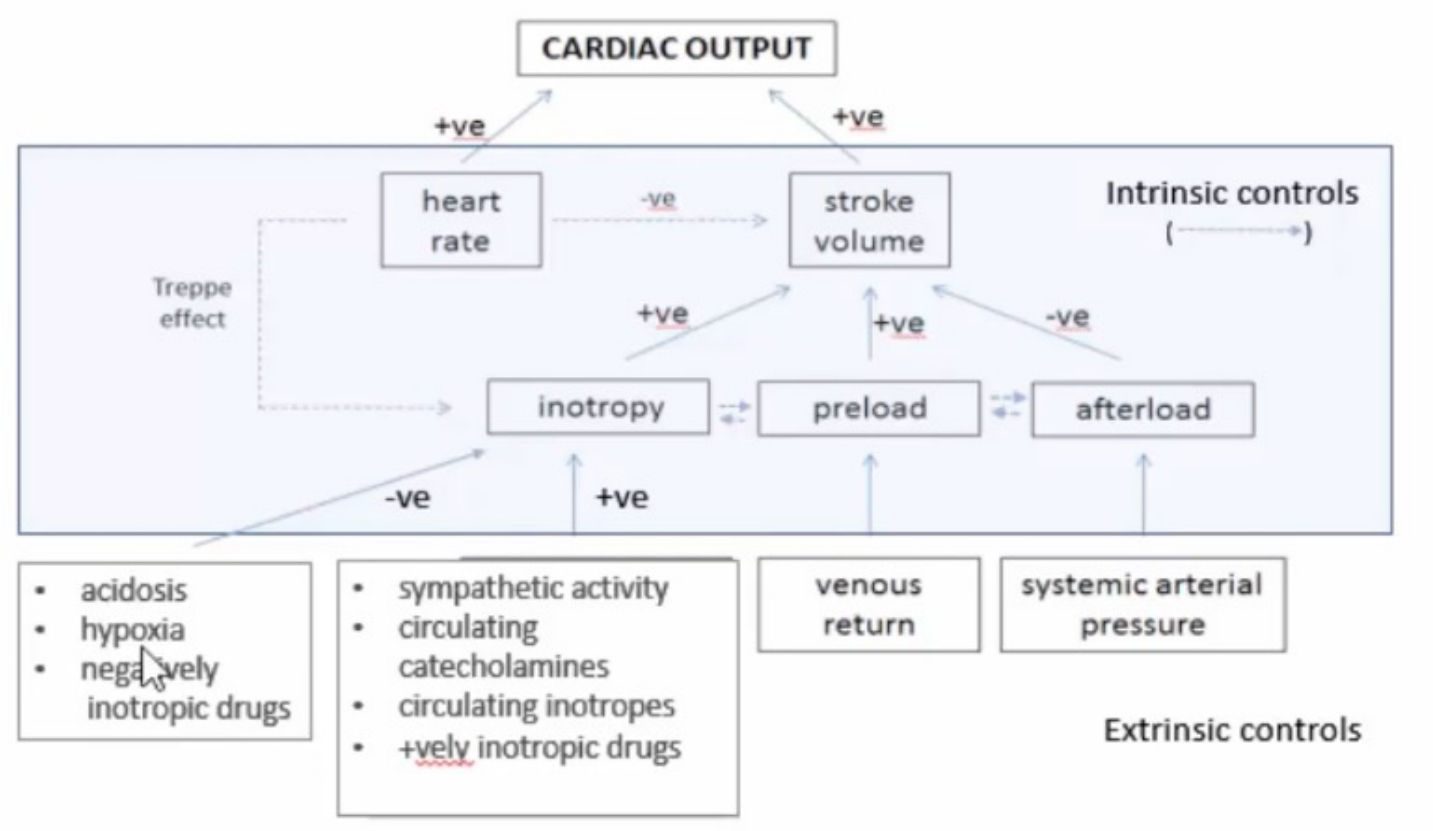
THE CARDIAC PUMP

Cardiac output = stroke volume x HR

- Stroke volume = average 70ml per cycle
  - EDV around 50ml
  - Ejection fraction normally around 0.6 / 60%
- HR = average 60-70bpm
- Cardiac output therefore around 5 litres / minute

Stroke volume dependant on

1. Pre-load
2. Inotropy
3. Afterload



Treppe effect

- If HR too high = ↓ time for ventricular filling = ↓ SV (theoretically)
- But ↑ HR reduces time for Ca2+ to be cleared, and so SV is maintained because intracellular Ca2+ levels remains high

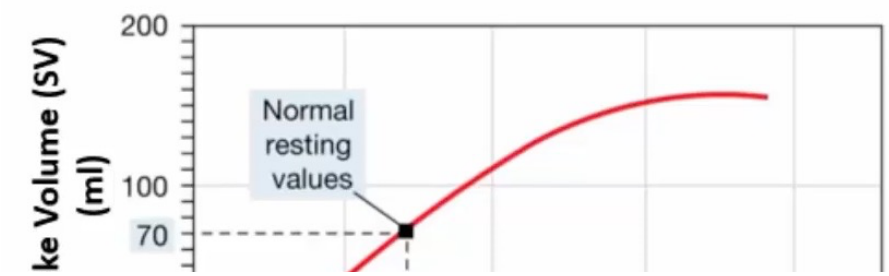
Frank-Starling forces

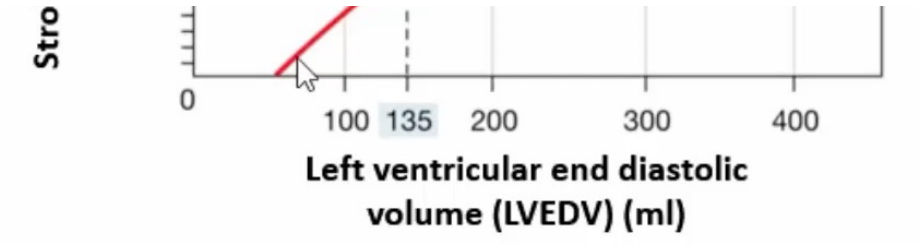
Stroke volume is proportionate to the stretch (pre-load) of cardiac myocytes.

↑ stretch = ↑ contraction of myocytes

This works up until a point, and then myocyte contractility degrades (e.g. heart failure)

Frank-Starling Curve:





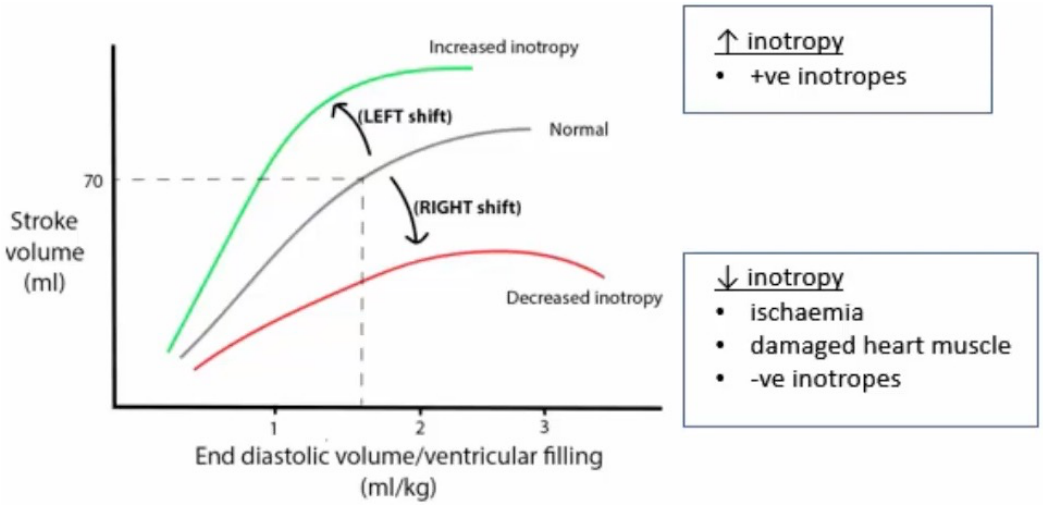
- Average L ventricular end diastolic volume = 135ml. This will produce around 70ml stroke volume
- Curve is almost linear and then declines with increasing preload
- Fluid resucitation aims to push the patient up the curve to ensure end organ perfusion - dont overfill patient

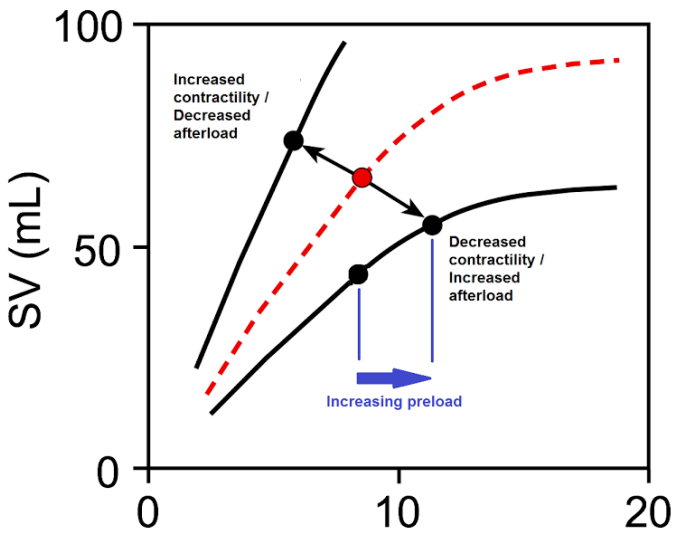
Factors affecting Frank starling curve:

1. Pre-load
    - ↑ pre-load = rightward shift along curve
2. Contractility/inotropy
    - ↑ contractility = upward shift on curve (more blood pumped out per beat)
3. After-load
    - ↑ after load = downward and leftward shift

Inotropy

- ↑ inotropy = increased stroke volume for same end diastolic volume (left and upward shift)
- ↓ inotropy = opposite to above





LVEDP (mmHg)

Positive inotropes

1. Sympathetic stimulation -
    - *Noradrenaline* on beta-receptors —> G-protein coupled receptors —> ↑ Ca2+ influx —> ↑ inotropy
2. Circulating inotropes
    - *Adrenaline* from adrenal medulla —> on beta 1 and beta 2 receptors
    - *Glucocorticoids (cortisol) and thyroxine —>* glucocorticords ↑ catecholamines (adrenaline, dopamine)
3. Pharmacological
    1. Catecholamines - e.g. noradrenaline, dopamine
    2. Sympatheticomimetics - e.g. ephedrine
    3. Digoxin - mild +ve inotropic effect (useful for rx tachyarrhythmia in poor cardiac function)

Negative inotropy:

1. Acidosis - H+ ions compete with Ca2+ for voltage gated Ca2+ channels = ↓ inotropy
2. Hypoxia reduces inotropy of heart, as heart muscle is aerobic
3. Pharmacological
    1. Beta-adrenergic receptor blockers (b-blockers)
    2. CCBs
    3. Fleicanide
    4. Anaesthetics e.g. propafol