NEET SS ANAESTHESIA
CARDIAC
ANAESTHESIA

CONTENT

CARDIAC PHYSIOLOGY I

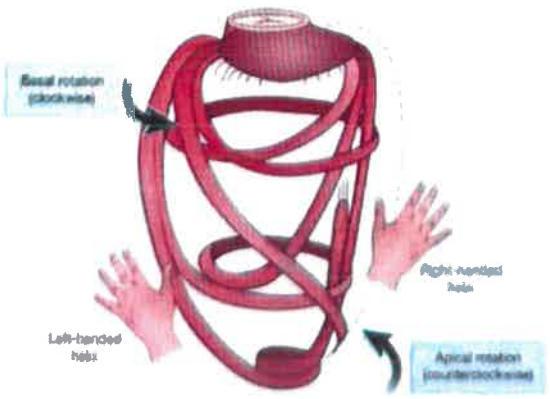
Gross muscle geometry of heart

00:01:21

Functional implications of gross geometry of heart :

- The skeleton's foundation includes the annuli of valves, roots of aortic and pulmonary arteries, central fibrous body, left and right fibrous trigones.
- Protein elastin interwoven in thick type I collagen crosslinked with thin type III collagen provides additional flexibility and elasticity without compromising strength.
- Composed of orthogonally oriented layers of the myocardium (RV 5 mm, LV 10 mm) : Interdigitating deep sinospiral, superficial sinospiral and superficial bulbospinal.
- Left ventricle can tolerate high pressures, but right ventricle is more compliant.
- Subendocardial and subepicardial muscle fibres of LV follow perpendicular, oblique and helical routes from base to apex but the orientation of these interdigitating sheets reverses direction at the LV midpoint.
- Contraction of obliquely arranged subepicardial and subendocardial fibres causes LV chamber shortening along its longitudinal axis, concomitant with a characteristic twisting action that increases magnitude of force generated by LV during systole above that produced by basal-apical muscle fibre shortening alone.
- Contraction of circumferentially oriented mid-myocardial fibres reduces chamber diameter.

Myocardial fiber orientation and direction of rotation



Myocardial fiber orientation and direction of rotation. Myocardial fibers in the subepicardium helically run in a left-handed direction, fibers in the mid layer run circumferentially, and fibers in the subendocardium helically run in a right-handed direction.

- Elastic recoil of the systolic wringing motion during LV relaxation is a crucial determinant of diastolic suction, facilitating adequate LV filling during hypovolemia and strenuous exercise.
- Helical geometry : Spherical configuration, contribute to decreased systolic function during evolving heart failure.
- Twisting motion :
  - If myocardial fibres contract directly, produce ejection fraction (EF) of 15–20%.
  - Simultaneous contraction and wringing action of helically oriented myocardial fibres results in 60–70% EF.
- LV contraction is temporally uniform, whereas RV contraction is peristaltic.
- RV contracts toward IV septum (IVS) with a bellows-like action with IVS & LV providing splint against which RV free wall shortens and essential contribution by LV contraction (Systolic ventricular interdependence) → mechanical advantage to less muscular RV to eject SV (Stroke volume) = LV.
- Thinner RV is more vulnerable to acute decompensation with modest ↑ in afterload because thicker LV can generate pressure-volume work upto 5 to 7 times greater than RV can produce.
- RV is more compliant & accommodates excess volume more quickly than LV.

Laplace law

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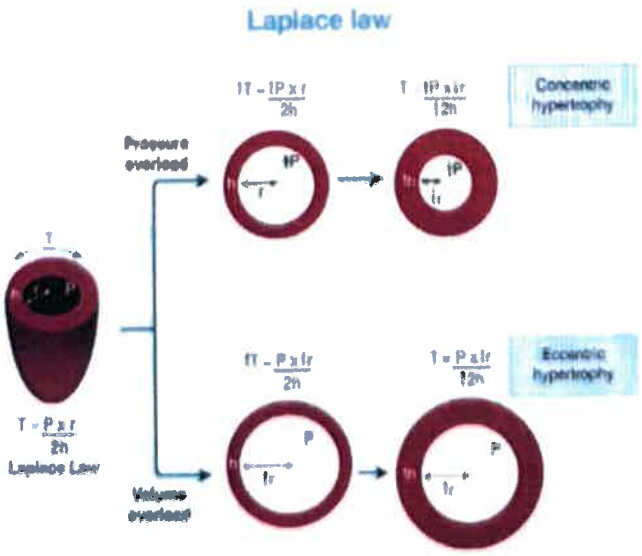
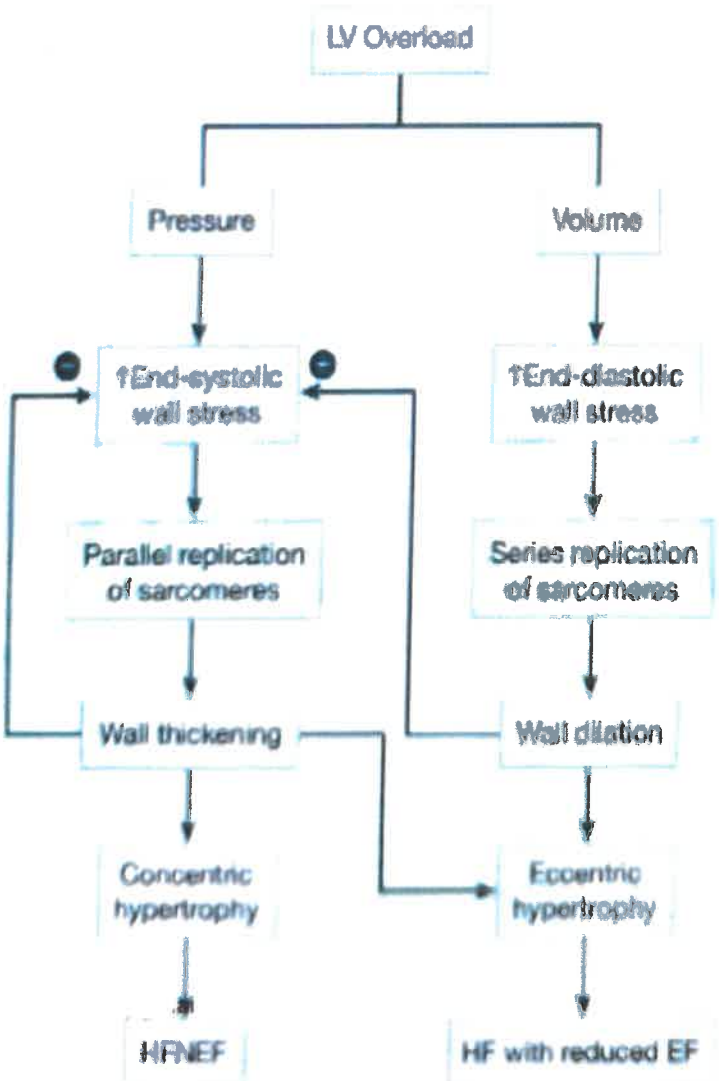
Internal pressure (P) = Orthogonal distending force exerted against chamber walls.
Wall stress = Shear force exerted around the circumference of the chamber resisting distension.



- The relationship b/w wall stress & pressure in the cardiac chamber is complex.
- Wall stress is not uniformly distributed across LV thickness in an intact heart, it is greatest in the subendocardium & progressively declines to minimum at the epicardial surface.
- Regional differences are important in LV pressure-overload hypertrophy due to aortic valve stenosis or poorly controlled essential hypertension wherein subendocardium exposed to pronounced ↑ interventricular pressure concomitant with greater myocardial O_2 consumption make it susceptible to acute MI (myocardial infarction).
- Not applicable for RV volume (Bellows-shape).

Pathophysiology of Heart failure



Application of Laplace law :

Severe aortic stenosis (AS) :

- Increased pressure overload of heart.
- Sarcomeres replicate in parallel direction.
- P↑, R↓, H↑ to maintain tension.
- Leads to concentric hypertrophy.

Chronic aortic regurgitation (AR) :

- Increased volume overload of heart.
- R↑, H↑ to maintain tension.
- Leads to eccentric hypertrophy.

Effect of PEEP on LV wall stress :

- PEEP = LVP (Left Ventricle Pressure) - Intrathoracic pressure.
- Increased transmural pressure (Negative intrathoracic pressure) increases afterload.
- Decreased transmural pressure (Positive pressure ventilation) decreases afterload.
- Increasing PEEP helps in reducing LV wall stress, but deteriorates RV wall stress.

Cardiac cycle

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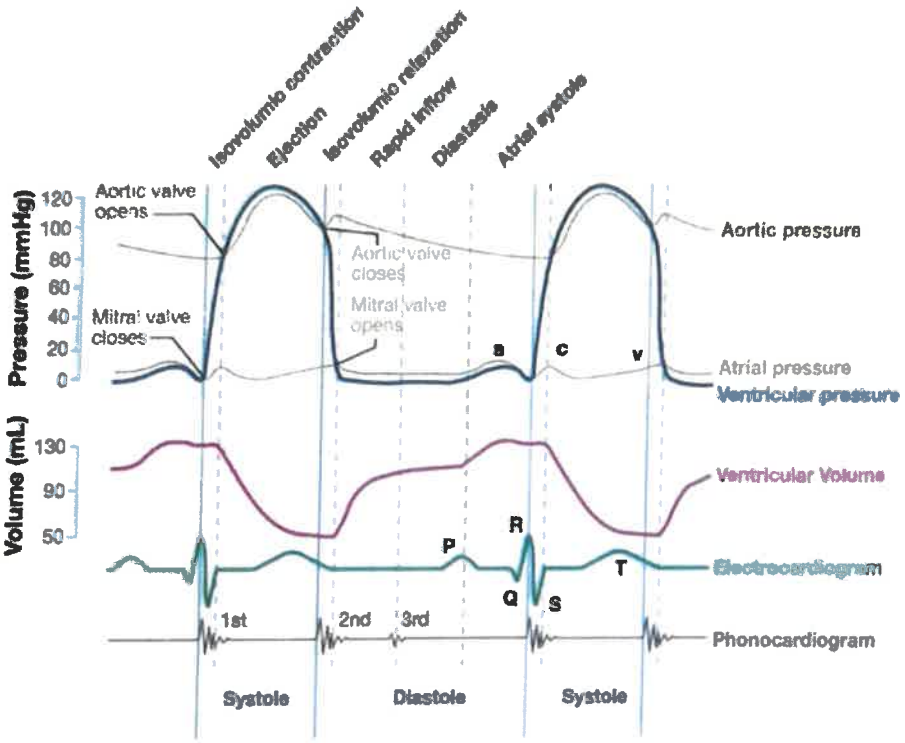
Duration : 0.8 sec at heart rate 75 bpm.

Events in cardiac cycle :

1. Atrial systole : P wave in ECG.
2. Ventricular systole :
   - Isovolmetric contraction (IVC) : QRS wave in ECG.
   - Rapid ejection : Aortic valve opens, stroke volume is generated, QT interval in ECG.
   - Slow ejection : When LV pressure < Aortic pressure, aortic valve closes.
3. Ventricular diastole :
   - Isovolumetric relaxation (IVR) : T wave on ECG.
   - Rapid filling (70-80%) : When LV pressure < LA pressure, mitral valve opens, no event in ECG.
   - Diastasis : Slow gradual filling from LA to LV, no event in ECG.
   - Atrial kick (15-20%) : Second rapid filling, corresponds to atrial systole, P

wave in ECG.

During heart failure, atrial kick contributes upto 40% of ventricular filling during diastole.



LV pressure and volume during cardiac cycle :

LV pressure :

- 120 mm Hg during ejection phase.
- 20 mm Hg during ventricular relaxation.

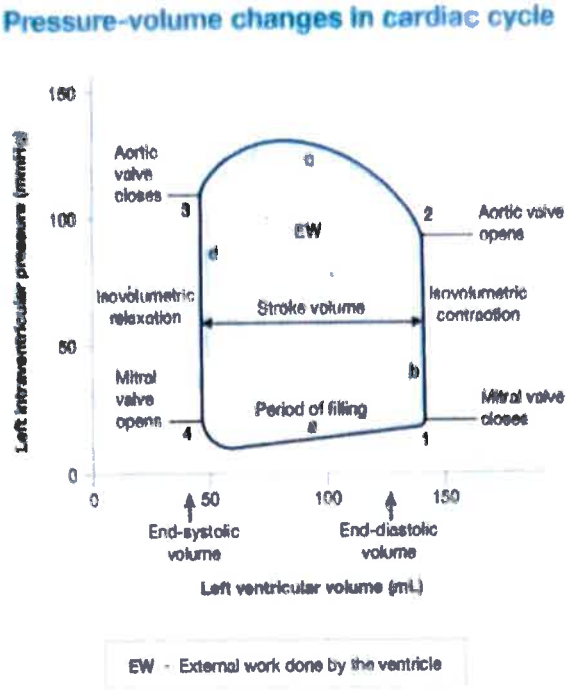
LV volume :

- End diastolic volume : 120-130 mL.
- End systolic volume : 40-50 mL.

Heart sounds :

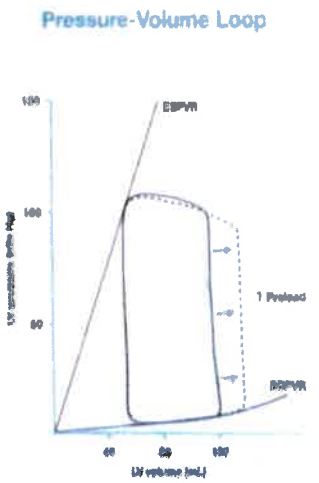
- S1 : Closure of mitral and tricuspid valves.
- S2 : Closure of aortic valves.
- S3 : Rapid inflow of blood from LA to LV.

Pressure-volume loop :

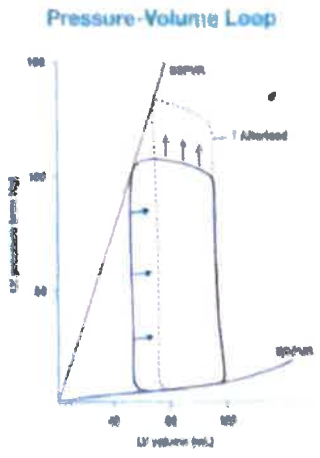


- End-systolic pressure-volume relationship (ESPVR) : maximal pressure that can be developed by the ventricle at any given LV volume (Contractility).
- End-diastolic pressure-volume relationship (EDPVR) : Describes ventricular elastance (Change in pressure per change in unit volume).
- Effective arterial elastance line connects point of end-diastolic pressure & volume to point of end-systolic volume & vaguely relates to afterload.
- Stroke work (Area under curve) = SV (stroke volume) x P (Pressure)
  = (LVEDV − LVESV) x P.

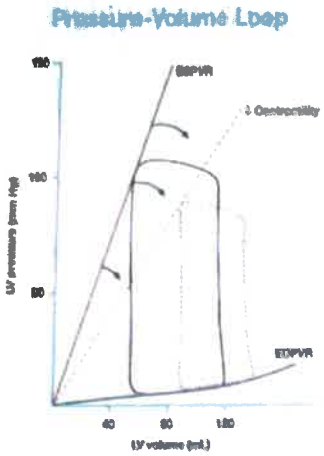
Examples :



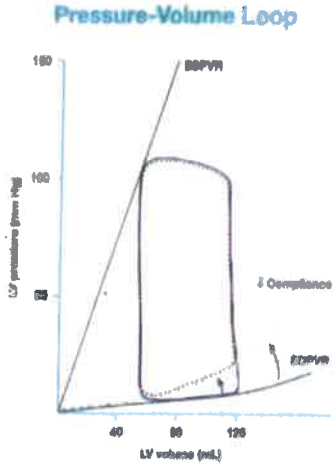
↑ Preload → ↑ SV & LV end-diastolic pressure.



↑ Afterload → ↑ LV pressure but ↓ SV.



ESPVR slope ↓ → LV systolic pressure ↓, SV ↓, ESV ↑.



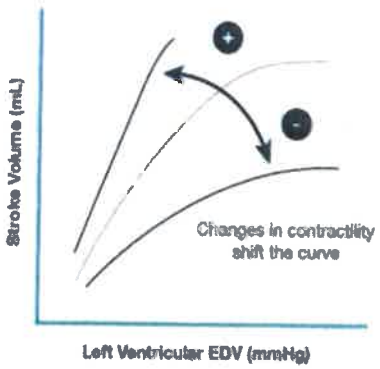
ESPVR is same, EDPVR slope ↑ → Compliance ↓, end diastolic pressure ↑.

Frank Starling law

00:41:40

Features :

- Determines relationship between length and contractility/tension of cardiac fibres.
- "Within physiological limits, the heart pumps all blood that returns to it by the way of veins (venous return)".
- Based on the length-tension relationship in cardiac muscle.



Implication :

- Explains relationship b/w EDV, contraction, strength & SV.
- SV & CO directly correlates with EDV and EDV correlates with venous return (CO = venous return).
- ↑ Venous return (Preload) → Cardiac muscle stretches to greater length → Ventricular muscle contracts with greater force → ↑ CO.
- Extreme stretching (HR >150) results in pulling apart of actin & myosin filament → ↓ Tension in cardiac muscles.
- In diseased heart, increase in volume leads to decrease in contractility : Optimisation of volume status is important.

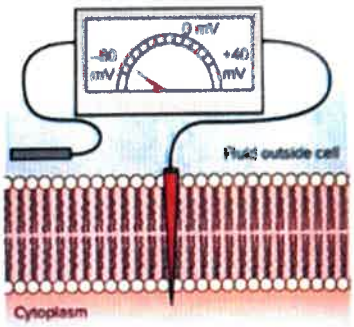
CARDIAC PHYSIOLOGY : II

Cardiac action potential

00:00:21

Transmembrane potential :

- Transmembrane potential (TMP) is the difference in electric potential between the interior and the exterior of a biological cell.
- When the cell is in a resting state this is known as the Resting membrane Potential (RMP).
- The transmembrane potential is due to the uneven distribution of ions between the inside and the outside of the cell.
- Skeletal muscle : -85 mV.
- Cardiac muscle : -90 mV.
- SA node : -60 mv.



Transmembrane potential.

Cardiac ion channels :

Sodium channels :

- Fast Na+ : Phase0 depolarization of non-pacemaker cardiac action potential
- Slow Na+ : Funny pacemaker current (If) in cardiac nodal tissue.
  Inhibited by Ivabradine.

Potassium Channels

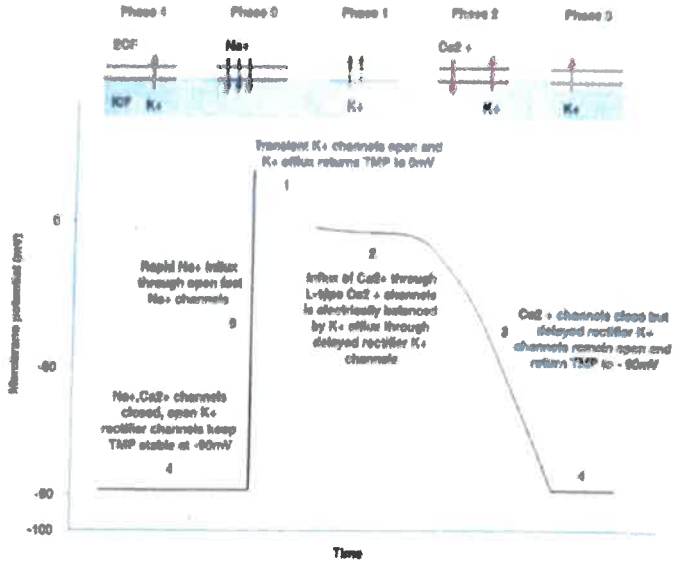
- Inward rectifier (Iir or IKi) : maintains phase 4 negative potential in cardiac cells.
- Transient outward (Ito) : Contributes to phase 1 of non-pacemaker cardiac action potentials.
- Delayed rectifier (IKr) : Phase 3 repolarization of cardiac action potentials.

Calcium channels :

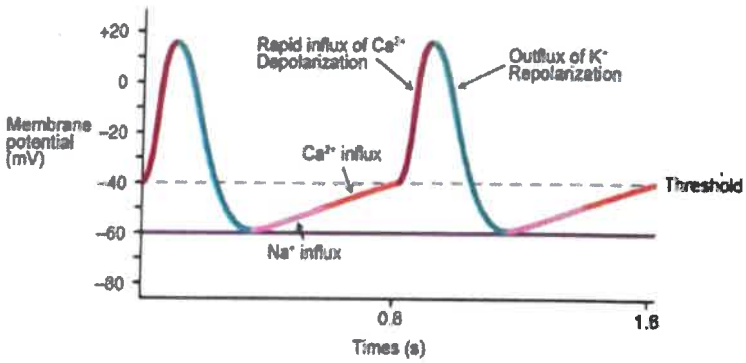
- L-type (ICa-L) : Slow inward, long-lasting current. Phase 2 non-pacemaker cardiac action potentials and late phase 4 and phase 0 of SA and AV nodal (pacemaker) cells.
- T-type (ICa-T) : Transient current that contributes to early phase 4 pacemaker currents in SA and AV nodal cells.

Action potential :

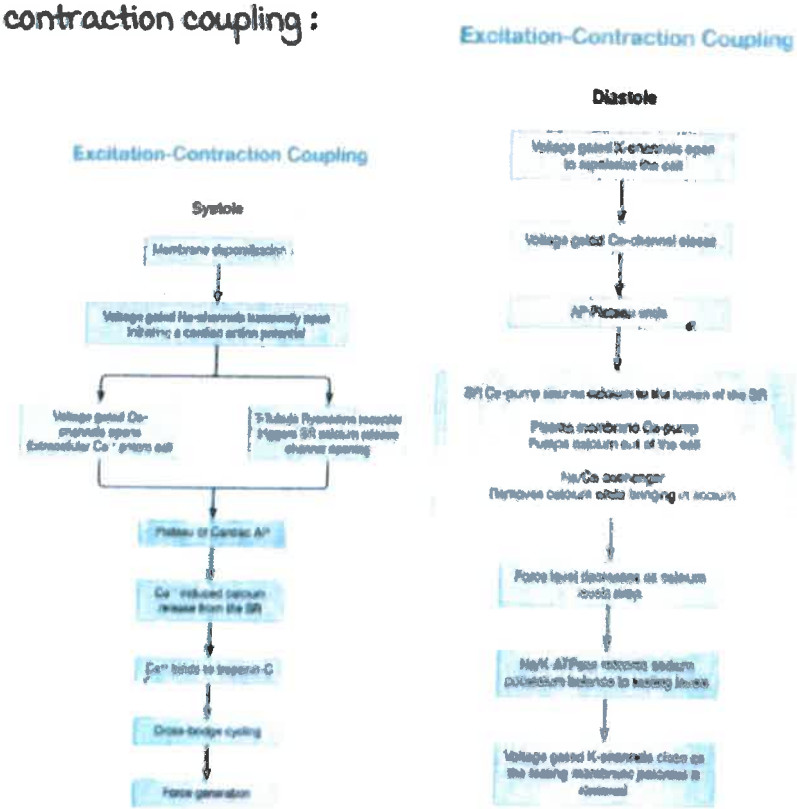
Ventricular muscle :



Pacemaker cells :



Excitation contraction coupling :



Cardioplegia

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Clinical implication in cardiopegia :

- Resting membrane potential is dominated by potassium channel equilibrium.
- NERNST equation :

$$\text{Equilibrium potential} = 61.5 \log \times \frac{\text{Concentration of potassium ion outside the cell}}{\text{Concentration of potassium ion inside the cell}}$$

- Normal resting membrane potential is -92 mv.
- In cardioplegia (Potassium rich) the resting membrane potential is -52 mv so there is early depolarisation of the cardiac muscle.
- Potassium rich cardioplegia arrest the heart in diastole phase.
- Potassium cardioplegias are :
  - Delnido cardioplegia.
  - St Thomas cardioplegia.

Hyponatremic cardiopegia :

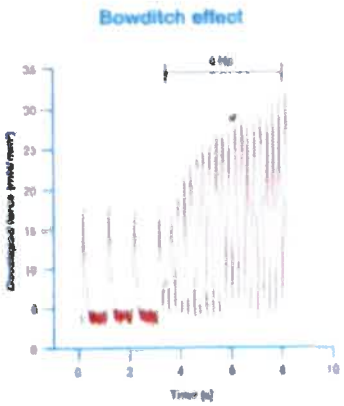
They are HTK and burschneider cardioplegia.
They have very low sodium levels.
They arrest the heart at hyperpolarised state.

Cardiac reflexes

00:22:08

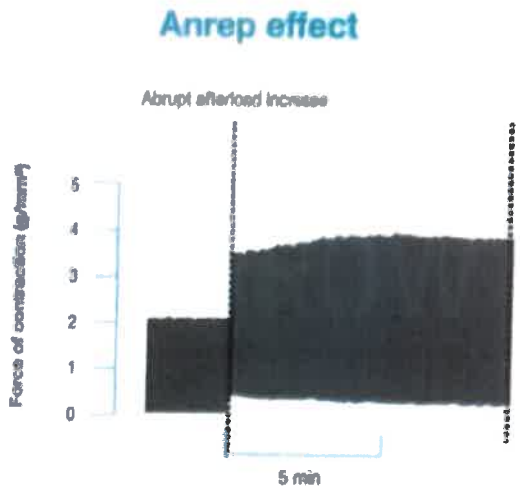
Bowditch effect :

- With increased heart rate, the time left for the removal of calcium is decreased.
- Residual calcium will increase the contractility of the myocytes wherever a high heart rate is sustained.
- Treppe phenomenon, the staircase phenomenon (Treppe being the German word for staircase).
- myocyte contraction is the consequence of significant calcium influx into the myocytes.
- Relaxation is mainly due to this calcium being ejected back out of the cell, or re-sequestered into the sarcolemma.
- This expulsion of calcium is a chemical process with a finite reaction time.
- This reflex is absent in patients with cardiomyopathy and CAD (inverse staircase phenomenon).



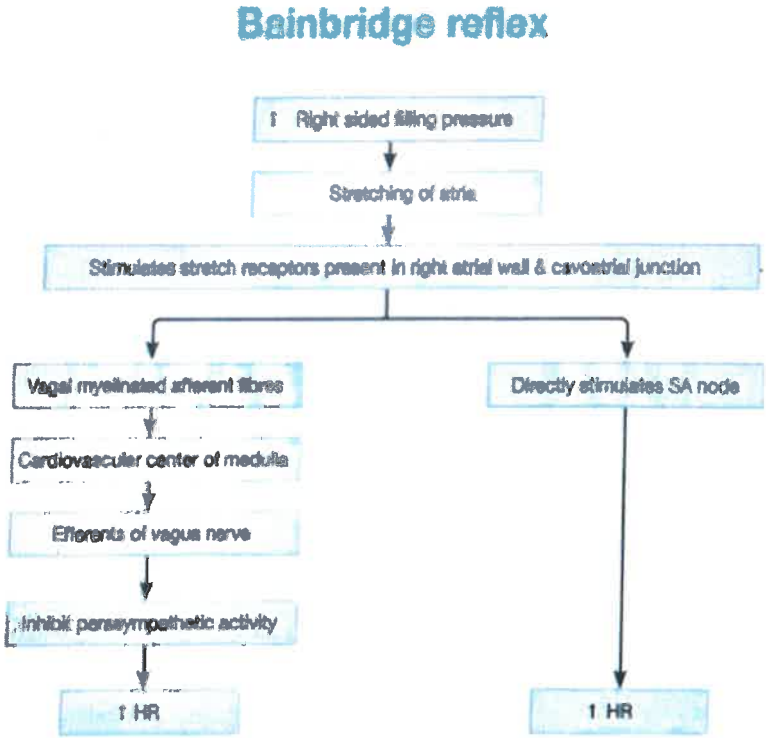
Anrep effect :

- Increased afterload.
- Increased end-systolic volume.
- Increased sarcomere stretch.
- That leads to an increase in the force of contraction.



Bainbridge reflex :

Also known as atrial stretch reflex and volume reflex.



Cushing reflex :

- Afferent : mechanosensors in the rostral medulla.
- Processor : Rostral ventrolateral medulla.
- Efferent : Sympathetic fibres to the heart and peripheral smooth muscle.
- Effect : Hypertension and baroreflex-mediated bradycardia.

Bezold jarisch reflex :

- Afferent : Vagus (mechanical/chemical sttimuli to the cardiac chambers).
- Processor : Nucleus of the solitary tract.
- Efferent : Vagus nerve and sympathetic chain.
- Effect : Hypotension and bradycardia in response to atrial stimulation.

CORONARY CIRCULATION : ANATOMY & PHYSIOLOGY

Coronary arteries

00:00:20

Features :

- Lie on the epicardial surface.
- Constitute 5% of the cardiac output.
- Two types :

i. Right coronary artery.

ii. Left coronary artery :

Left anterior descending artery.

Left circumflex artery.

Left main artery :

- Widow artery.
- Length : 0-40mm.
- Arise from left/posterior coronary sinus.
- Emerge between PA and left atrium.

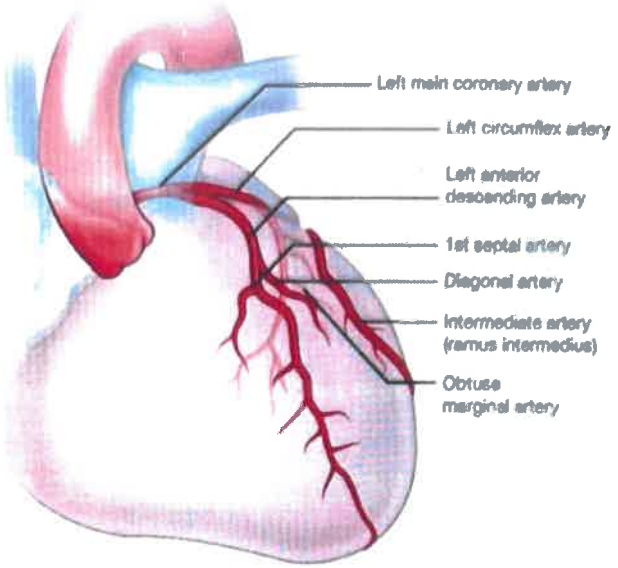
Left anterior descending :

- Runs directly into the anterior inter-ventricular grove, turns downwards and reaches the posterior suface of heart.
- Diagonal branch : Anterior & lateral part of LV.
- Septal branch : Anterior wall and 2/3 anterior IVS.

Left circumflex artery :

- Winds around the left lateral border and reaches the posterior surface.
- Obtuse marginal branch : Lateral part of LV.
- Posterolateral brach : Posterior part and anastamoses with the RCA.
- SA node artery : In 38% of the population.
- AVN artery : In 10-15% of the population.
- Also supplies :
  - Left atrium.
  - Part of lateral LV.
  - Inferior surface of LV (15%).

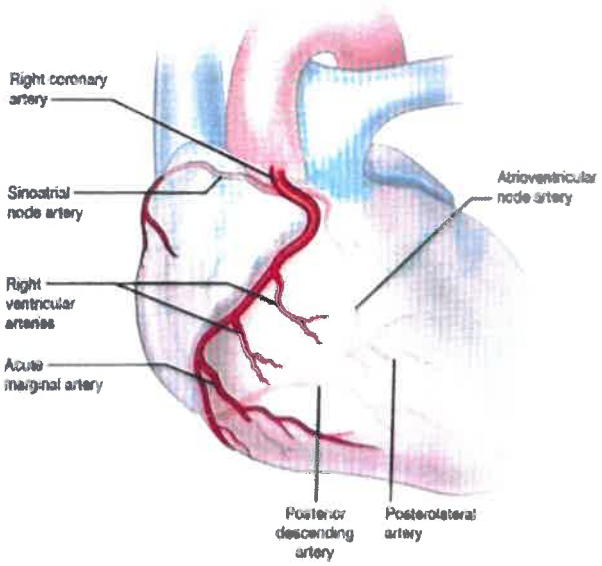
- Posterior surface of LV (15%).



Left coronary artery

Right coronary Artery :

- Arise from right/anterior coronary sinus.
- Emerge between PA and right atrium.
- Terminates along post IVS by anastomosing with LCX.
- Branches :
  - i. SA node artery.
  - ii. Acute marginal artery.
  - iii. Posterior descending artery.
  - iv. Postero-lateral artery.
- Areas of distribution
  - Right atrium.
  - most of RV except ant IVS.
  - Post IVS.
  - most of conduction bundle : SAN, AVN, BOH.



Right coronary artery

Coronary supply of conduction bundle :

- SAN : 60% RCA, 40% LCX.
- AVN : 90% RCA, 10% LCX.
- BOH : RCA.
- RBB : LAD.
- LBB : Mostly LAD.

Arterial supply of different anatomical regions :

Anatomic region of heart	Coronary artery
Inferior wall	RCA (85%), LCX (15%)
Anterioseptal wall	LAD
Anterioapical	Distal LAD
Anteriolateral	LCX
Posterior wall	RCA (85%), LCX (15%)

Cardiac veins

00:10:32

Venous drainage :

- Coronary sinus (85%).
- Anterior cardiac veins.
- Thebasian veins.

Coronary sinus :

Opens into the posterior wall of right atrium.

Major veins draining :

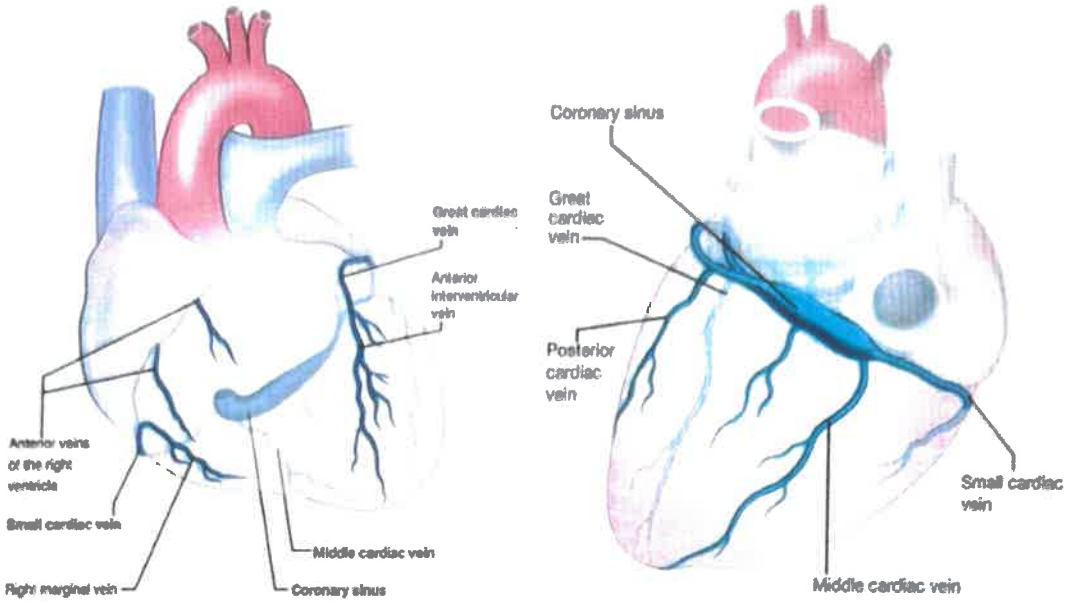
i. Great cardiac vein which runs along anterior interventricular grove.
ii. Middle cardiac vein which runs along the posterior interventricular septum.
iii. Small cardiac vein which runs along the right coronary artery.
iv. Posterior cardiac veins.

Anterior cardiac veins :

- Arise from the right atrium.

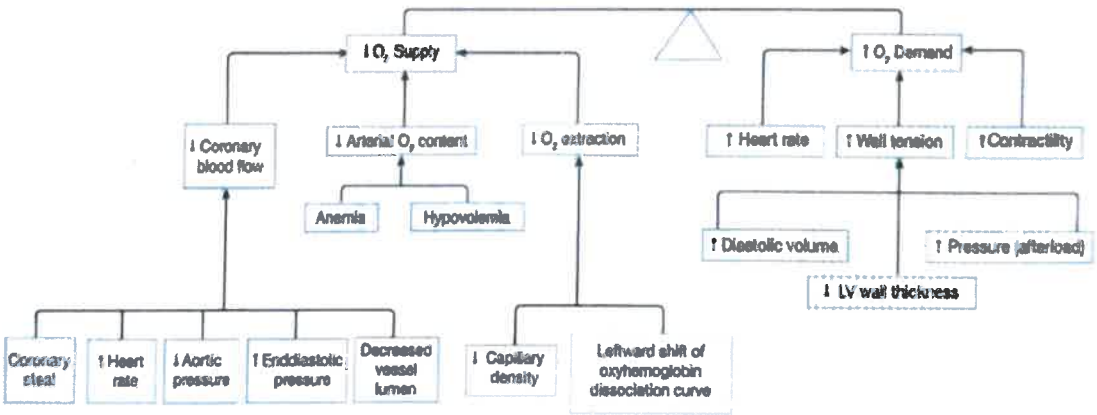
Thebasian veins :

- Drains all four chambers.
- Anterior cardiac veins and thebasian veins constitute 15% of venous drainage.



Venous drainage

Coronary physiology 00:14:02



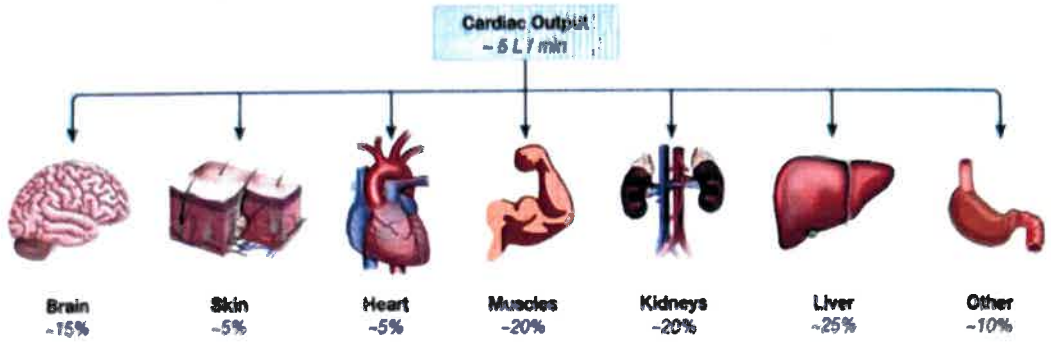
Myocardial oxygen supply and demand mismatch

Note :

- Coronary steal causes decreased blood flow to already ischemic area.
- CPP = DAP − LVEDP.
- Leftward shift of oxyhemoglobin dissociation curve seen in :
  - Hypothermia.
  - Hypercarbia.
  - Decrease in 2,3 DPG.
- Laplace law :
  - $T = P \times R \div 2H$.
  - Wall tension depends on diastolic volume, LV thickness and pressure.

Coronary blood flow :

- maximum capillary density (15% of mass).
- maximal extraction of any vascular bed : 65–75%.
- MVO_2 : 10 ml O_2 / 100g /min.
- CBF : 225ml/min (60–90ml/100g/min).
- Total coronary blood flow : 5% of cardiac output, 3x in exercise.



Determinants of coronary blood flow :

1. Perfusion pressure.
2. Myocardial extravascular compression.
3. Myocardial metabolism.
4. Neurohumoral control.

Coronary perfusion pressure :

- CPP = DAP – LVEDP.
- Any decrease in DAP or increase in LVEDP causes decrease in CPP.
- DAP > MAP for CBF.
- Factors reducing DAP : AR/large PDA.
- Factors increasing LVEDP : Impaired filling/stiffness/decreased LVEF/ delayed relaxation.

Myocardial compression :

- Coronary blood flow is intermittent than continuous.
- Complete occlusion of intramyocardial part of coronaries in systole.
- LV cavity systole pressure increases to 120 mmHg and RV intraventricular pressure increases to 30 mmHg.
- LV is perfused only in diastole.
- RV is perfused in systole and diastole.
- ST depression is seen when there is ischaemia to the subendocardial surface → NSTEMI.
- ST elevation is seen when there is transmural ischaemia (Epicardium, myocardium and subendocardium) → STEMI.