NEET SS ANESTHESIA

Updated Notes 2026

CARDIAC ANAESTHESIA

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CARDIAC PHYSIOLOGY : PART I

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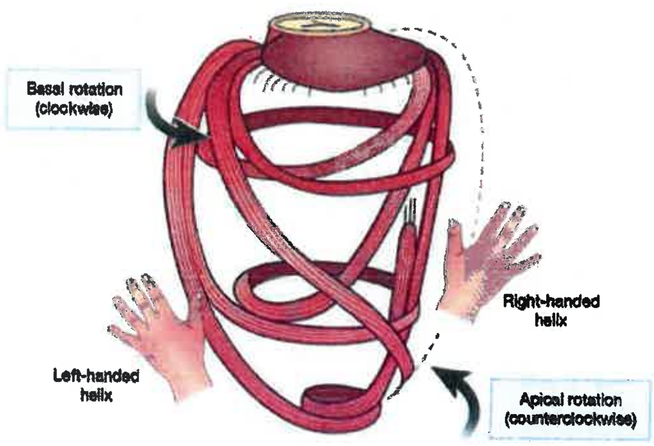
Anatomy

00:02:07

Functional implications of gross anatomy :

- Heart muscle has 3 main layers :
  - Endocardium, myocardium and the epicardium.
- Heart is composed of orthogonally oriented layers of myocardium :
  - Atria are thin & ventricles are thick (RV : 5mm, LV : 10mm).
  - Hence, LV has more capacity to contract, generate pressure & withstand higher pressure compared to atria.
- There are interdigitating deep sinospiral, superficial sinospiral & superficial bulbospiral muscles.
- Subendocardial & subepicardial muscle fibers of LV follow perpendicular, oblique, helical routes from base to apex.
  - Orientation of these interdigitating sheets reverses direction at LV midpoint.
- Contraction of obliquely arranged subepicardial & subendocardial fibers causes LV chamber shortening along its longitudinal axis, concomitant with characteristic twisting action that ↑ magnitude of force generated by LV during systole above that produced by basal-apical muscle fiber shortening alone.

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.

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- Contraction of circumferentially oriented mid myocardial fibers reduces chamber diameter.
- Elastic recoil of systolic wringing motion during LV relaxation is a crucial determinant of diastolic suction, facilitating adequate LV filling during hypovolemia & strenuous exercise.
- During evolving heart failure : Helical geometry → Spherical configuration contribute to ↓ systolic function.
- LV contraction is temporally uniform whereas RV contraction is peristaltic.
- RV contracts towards the interventricular septum (IVS) with a bellows like action with IVS & LV providing splint, against which RV free wall shortens and important contribution by LV contraction (Systolic ventricular interdependence).
- So there is mechanical advantage to less muscular RV to eject stroke volume.
- Thinner RV is more vulnerable to acute decompensation with modest ↑ in afterload, because thicker LV can generate pressure-volume work 5 to 7 times greater than what RV can produce.
- RV is more compliant & accommodates excess volume more easily than LV.

Relevance of twist motion :

- If myocardial fibers contracts directly, it produces an EF of 15-20%.
- Simultaneous contraction & wringing action of helical oriented myocardial fibers, results in an EF of 60-70%.

Laplace law

00:14:22

Formula :

$$T = \frac{P \times r}{2h}$$

- Internal pressure (P) : Orthogonal distending force exerted against chamber walls.
- Tension/ wall stress (T) : Shear force exerted around circumference of chamber resisting distension.
- h : Thickness of the wall.
- r : Radius.
- Relationship b/w wall stress & pressure in cardiac chamber is complex.

Application :

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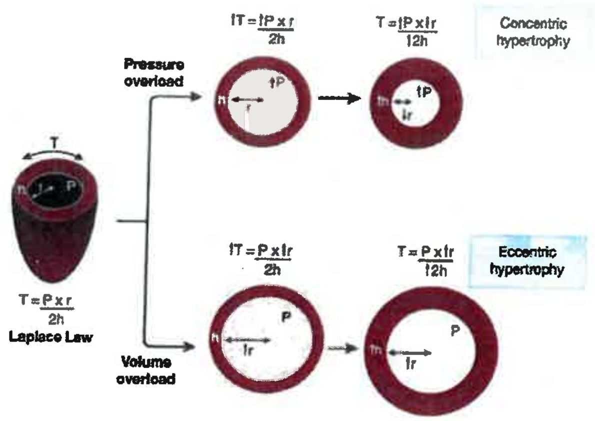
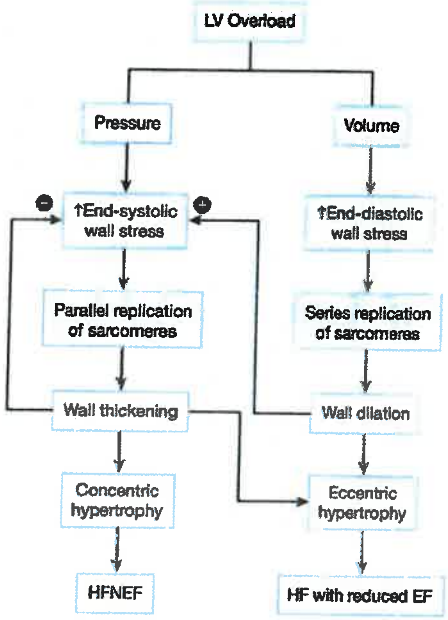
Laplace law



- In pressure overload, 'P' ↑ & hence 'T' also ↑ : To compensate that concentric hypertrophy occurs so that 'h' ↑ and later 'r' ↓ (Classic example : AS).
- In volume overload, radius (r) ↑ and hence T ↑ : To compensate, eccentric hypertrophy occurs and 'h' ↑ (Classic example : Chronic AR).

Pathophysiology of Heart failure



- In the intact LV, myocardial wall stress is heterogeneously distributed across its thickness, being maximal in the sub-endocardium and progressively decreasing toward the epicardial surface.
- Regional differences are important in LV pressure-overload hypertrophy due to aortic valve stenosis or poorly controlled essential HTN wherein sub-endocardium exposed to pronounced ↑ in interventricular pressure, concomitant with ↑ myocardial O_2 consumption making it susceptible to acute MI.

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Note :

- The Laplace law is not applicable to right ventricular volume due to its bellows-like geometry & relatively thin myocardial wall thickness.

Effect of PEEP on LV wall stress :

- Ventricular transmural pressure (P) : The difference between the intrathoracic pressure and the ventricular cavity pressure.
  - P = LVP (Intracavitary LV pressure) – ITP (Intrathoracic pressure).
  - ITP increases and decreases in accordance with PEEP.
- ↑ Transmural pressure (Negative intrathoracic pressure) ↑ afterload.
- ↓ Transmural pressure (Positive pressure ventilation) ↓ afterload.
- Clinical application : ↑ PEEP → ↑ ITP → ↓ Transmural pressure (P) → ↓ LV wall stress (T).

Cardiac cycle

00:31:18

Overview :

- At a heart rate of approximately 75–80 beats per minute, the duration of one cardiac cycle is about 0.8 seconds.
- Blood flows from LA to LV → LV volume (120–130 ml) and LV pressure start increasing.
- When the pressure of LV > LA → mitral valve close → First heart sound.

Left ventricular (LV) systolic phase :

- Isovolumetric contraction : All valves are closed; ventricular pressure rises sharply while volume remains constant.
- During systole, LV pressure rises rapidly to approximately 100–120 mmHg → Aortic valve open.
- Ejection phase :
  - Rapid ejection : ~70–75% of stroke volume is expelled.
  - Reduced (slow) ejection : Remaining 15–20% of stroke volume is ejected as ventricular pressure gradually declines.
- Aortic pressure increases → When pressure inside aorta > LV pressure → Aortic valve closes Second heart sound.
- By end of ejection phase LV end-diastole volume decreases (30–50 ml).
- LV pressure decrease gradually and aortic valve closes.

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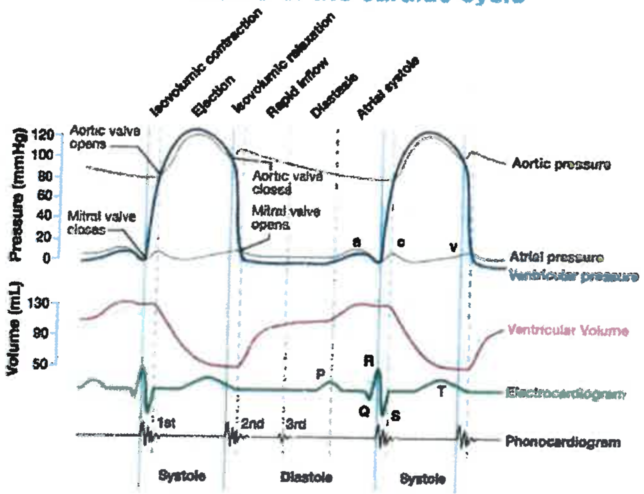
Events of the cardiac cycle



Left ventricular (LV) diastolic phase :

- Isovolumetric relaxation : Both aortic and MV valves are closed → LV pressure falls rapidly (~0–20 mmHg) while volume remains constant.
- Early passive filling : MV valves open → Blood flows rapidly from the left atrium (LA) into the LV, accounting for ~70% of diastolic filling.
- Diastasis (Reduced filling) : Slow, passive filling continues as pulmonary venous return gradually enters the LV (~5–10% of filling).
- Atrial contraction (Atrial Kick) : Atrial systole contributes the final 15–20% of LV filling, completing end-diastolic volume.

Pressure volume loop

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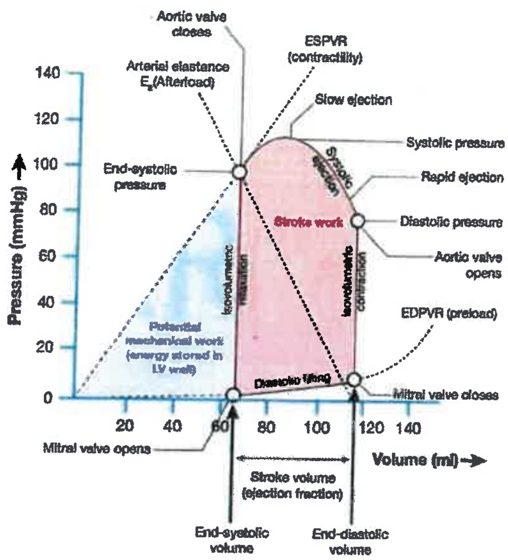
Overview :

Pressure-Volume Loop



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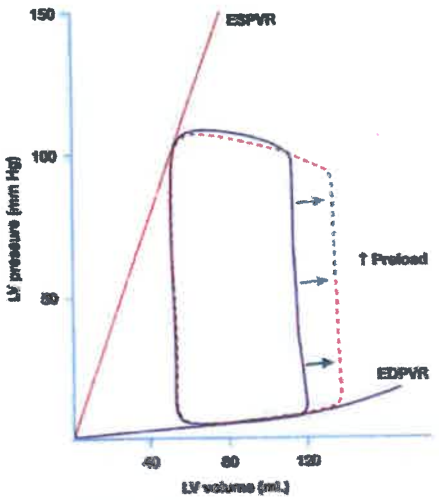
- mitral valve opening → Blood passes from LA to LV → LV pressure increases.
- Isovolumetric relaxation : Pressure in LV increases from 0 to around 20-30 mmHg Pressure inside LV > LA mv closes.
- Isovolumetric contraction : LV pressure is around 80-100 mm Hg → Aortic valve opens → LV ejection.
  - Early ejection : 120 mm of Hg.
  - Slow ejection : 80-100 mm of Hg.
- As pressure in LV decrease and gets back to 100 mmHg → Aortic valve closes → Second heart sound.
- End-diastolic pressure-volume relationship (EDPVR) (120 ml) describes ventricular elastance/compliance (Change in pressure per unit change in volume).
- End-systolic pressure-volume relationship (ESPVR) (50-60 ml) : Represents the maximum pressure that the left ventricle can generate at any given end-systolic volume, reflecting ventricular contractility independent of preload & afterload.
- Effective arterial elastance line connects point of end-diastolic pressure & volume to point of end-systolic volume & vaguely relates to afterload.
  - If the curve shifts to left : ↑ pressure generated leading to ↑ contractility.
  - Shift to right : Decreased contractility.

Interpreting pressure volume loops :

Pressure volume loop 1 :

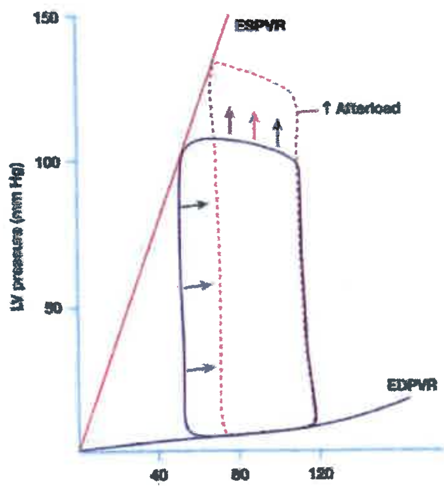


Lv compliance is maintained.

Volume (Pre load) has **increased** but is able to eject the whole volume at around same pressure (100-120mmHg) : increase in stroke volume.

Contractility is maintained

Pressure volume loop 2 :



Preload is decreased

Higher pressure is generated to eject that volume (~140 mmHg)

Afterload is increased

Contractility is maintained

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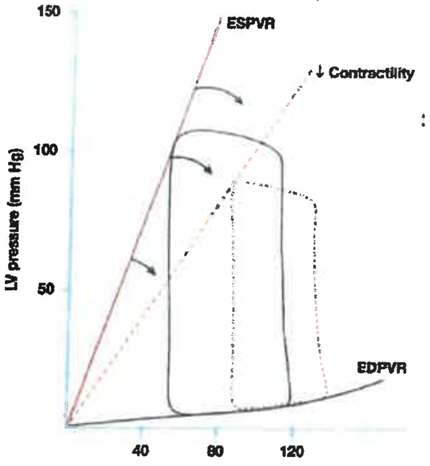
Pressure volume loop 3 :

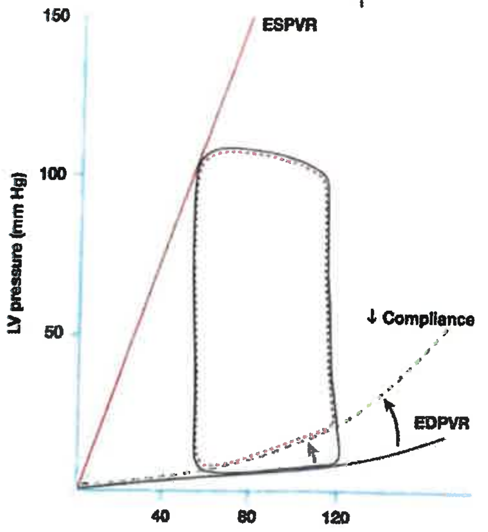


LV compliance is maintained

Volume is increased (Preload) but whole volume is not ejected

LV contractility is reduced (Shift to right)

Pressure volume loop 4 :



LV compliance has decreased

LV pressure has increased but for the same volume

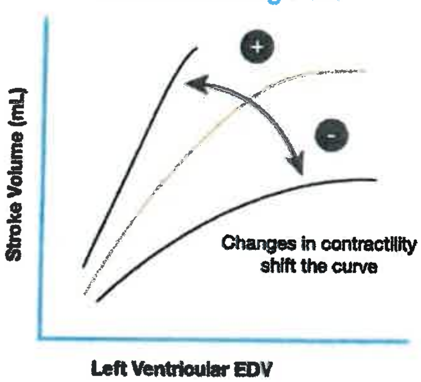
Frank Starling law

00:54:06

Overview :

- Within physiological limits, heart pumps all blood that returns to it by the way of veins (venous return).
- It is based on the length-tension relationship in cardiac muscle.
- Explains the relationship b/w EDV (End diastolic volume), contraction, strength & SV (Stroke volume).
- SV & CO directly correlates with EDV, and EDV correlates with VR.
- ↑ 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.

Frank-Starling Law



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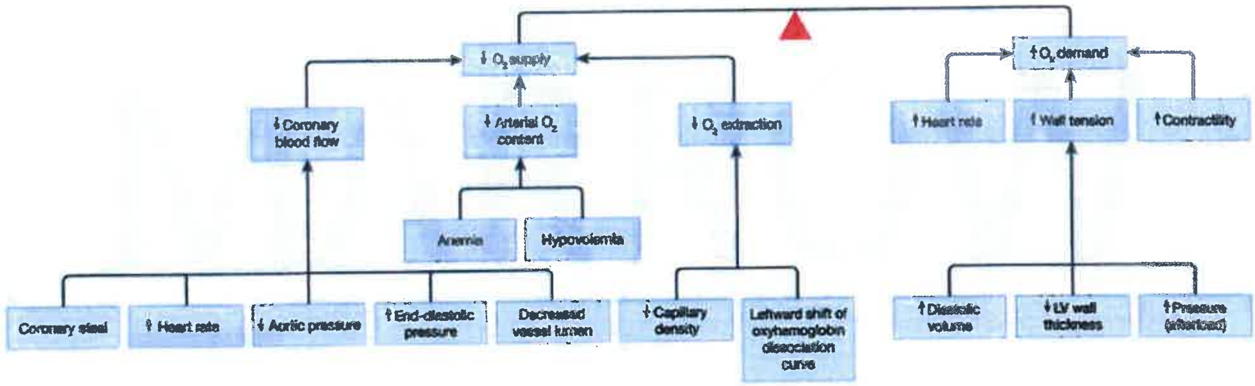
- Increase in length of muscle means increase in length of sarcomeres.
- Sarcomere has a length of approx 1.9 μm at rest.
- With increase in volume it increases to around 2.2 μm → Tension is developed → Contracts with a greater force.
- If the length increases >2.2 μm → Tension is not high/ decreases → Poor contraction.

Myocardial oxygen demand and supply

01:00:40

Oxygen demand and supply mismatch :

Coronary Physiology



Factors :

- Tachycardia : The demand increases but supply decreases → Decreased diastolic filling time → Decreased coronary perfusion duration → Decreased coronary blood flow.
- Oxygen delivery (DO_a) = Hb x 1.34 x SpO_a.
  - In anemia and hypovolemia : Decreased oxygen content → Decreased oxygen supply.
- Oxygen extraction decreased in :
  - Decreased capillary density.
  - Left shift of oxyhemoglobin dissociation curve : Acidosis, hypercarbia, decreased 2,3 DPG, increased temperature.

CARDIAC PHYSIOLOGY : PART II

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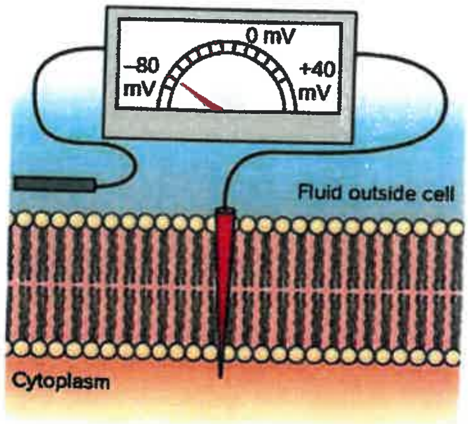
Cardiac action potential

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+ : Phase 0 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 (−ve) 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.

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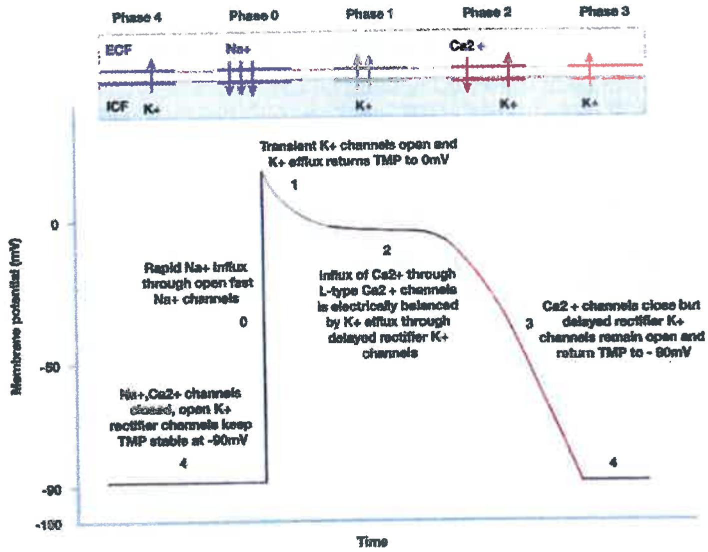
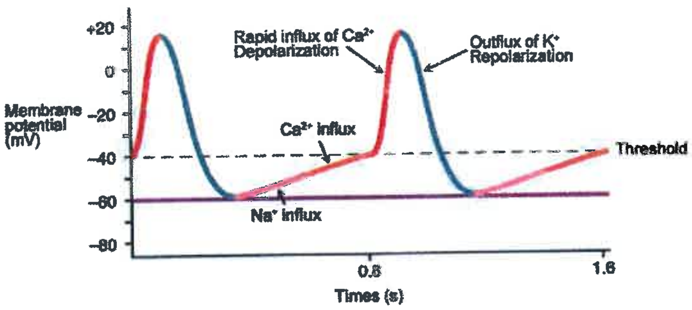
Action potential :

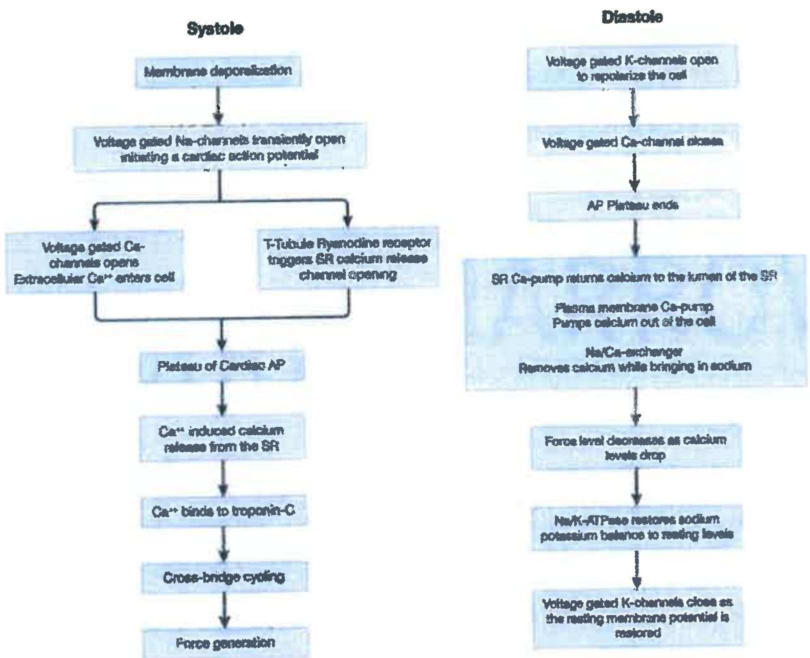
ventricular muscle :



Pacemaker cells :



Excitation contraction coupling :



Excitation-contraction coupling

Cardioplegia

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Clinical implication in cardioplegia :

- 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 cardioplegia :

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

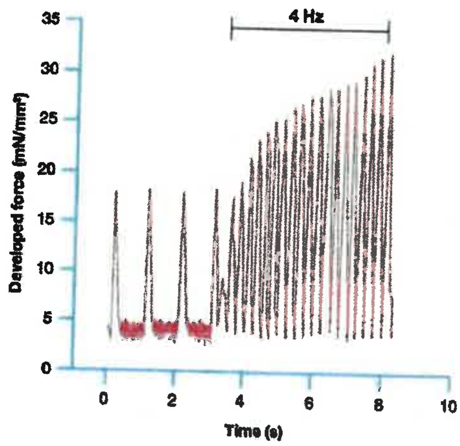
Cardiac reflexes

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 resequestered 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).

Bowditch effect



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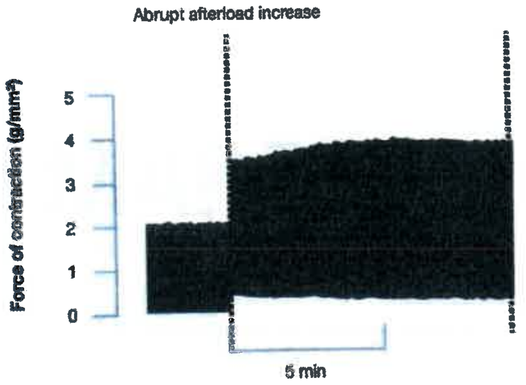
Anrep effect :

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

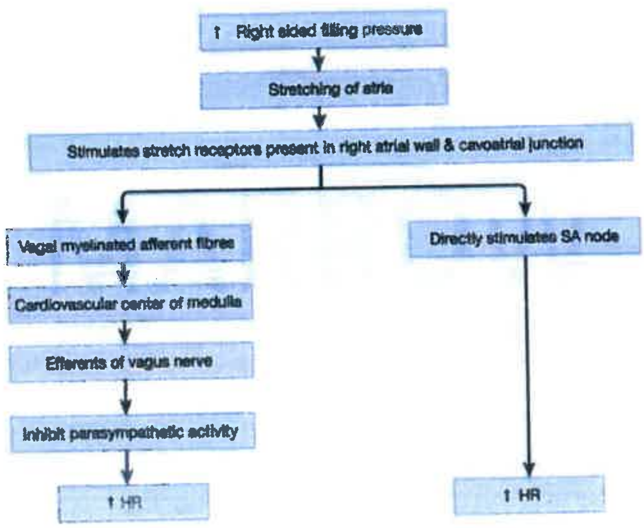
Anrep effect



Bainbridge reflex :

Also Known as atrial stretch reflex and volume reflex.

Bainbridge 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 stimuli 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

Anatomy of coronary circulation

00:00:34

Overview :

- Heart consists of 3 main epicardial arteries, which gives branches across the myocardium and then finally gives arterioles, which supplies the subendocardium.
- 3 main epicardial arteries are :
  - Right coronary artery (RCA).
  - Left main coronary artery (LMCA) divides into : Left anterior descending artery (LAD) & left circumflex artery (LCX).
- These coronary arteries arise from sinus of Valsalva (Outpouching present in the aortic root).
- The outpouching structure helps in :
  - Smooth opening & closing of aortic valve → Doesn't obstruct the flow across the coronary ostium.
  - Keeping a reserve of blood during the diastole, which maintains the diastolic aortic pressure, which in turn maintains the coronary perfusion pressure.
- In 10–15% cases, LMCA can give rise to a third branch : Ramus intermedius.

Left main coronary artery :

- Also known as widow artery.
- Arises from left sinus of Valsalva : Emerges between pulmonary artery & left atrium.
- After running for 0–40 mm, gives rise to LAD & LCX.

Left anterior descending artery :

- After arising from LMCA, runs in the anterior interventricular groove.
- In the anterior interventricular groove, gives rise to two branches : Diagonal (Supplies lateral wall of ventricle) & septal (Supplies the septum).
- Diagonal branch (Arise diagonally) : Supplies the anterior & lateral part of left ventricle.
- Septal branch (Arise perpendicularly) : Supplies the 2/3rd anterior interventricular septum.
- The number of diagonal & septal branches differ in each person : They are named sequentially (S1, S2, S3, etc).

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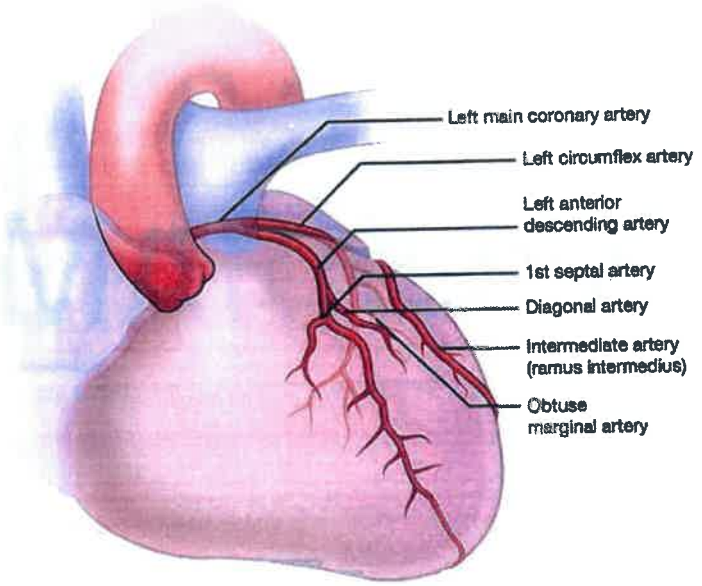
Segments of LAD :

- Proximal segment : From the origin from LMCA until the S1 branch is given.
- middle segment : From the S2 branch to half the distance between S1 & apex.
- Distal segment : Remaining part of LAD till the apex.

Types of LAD :

- Type 1 : LAD runs in anterior interventricular groove, but doesn't reach apex.
- Type 2 : LAD reaches apex.
- Type 3 : LAD reaches apex & wraps around to supply the posterior wall of left ventricle.



Left main coronary artery

Left circumflex artery :

- Runs in anterior atrioventricular groove, where it gives a branch to left atrium → Then, it rotates to run on the posterior wall of left ventricle & gives obtuse marginal branches (major branches of LCX).
- LCX can give rise SA nodal artery (38% Cases), AV nodal artery (10-15% Cases) & posterolateral branch (10% Cases).
- Supplies :
  - Left atrium.
  - Part of lateral left ventricle.
  - Inferior surface of left ventricle (15% Cases).
  - Posterior surface of left ventricle (15% Cases).
- The number of obtuse marginal branches differ in each person : They are named sequentially (OM1, OM2, OM3, etc).