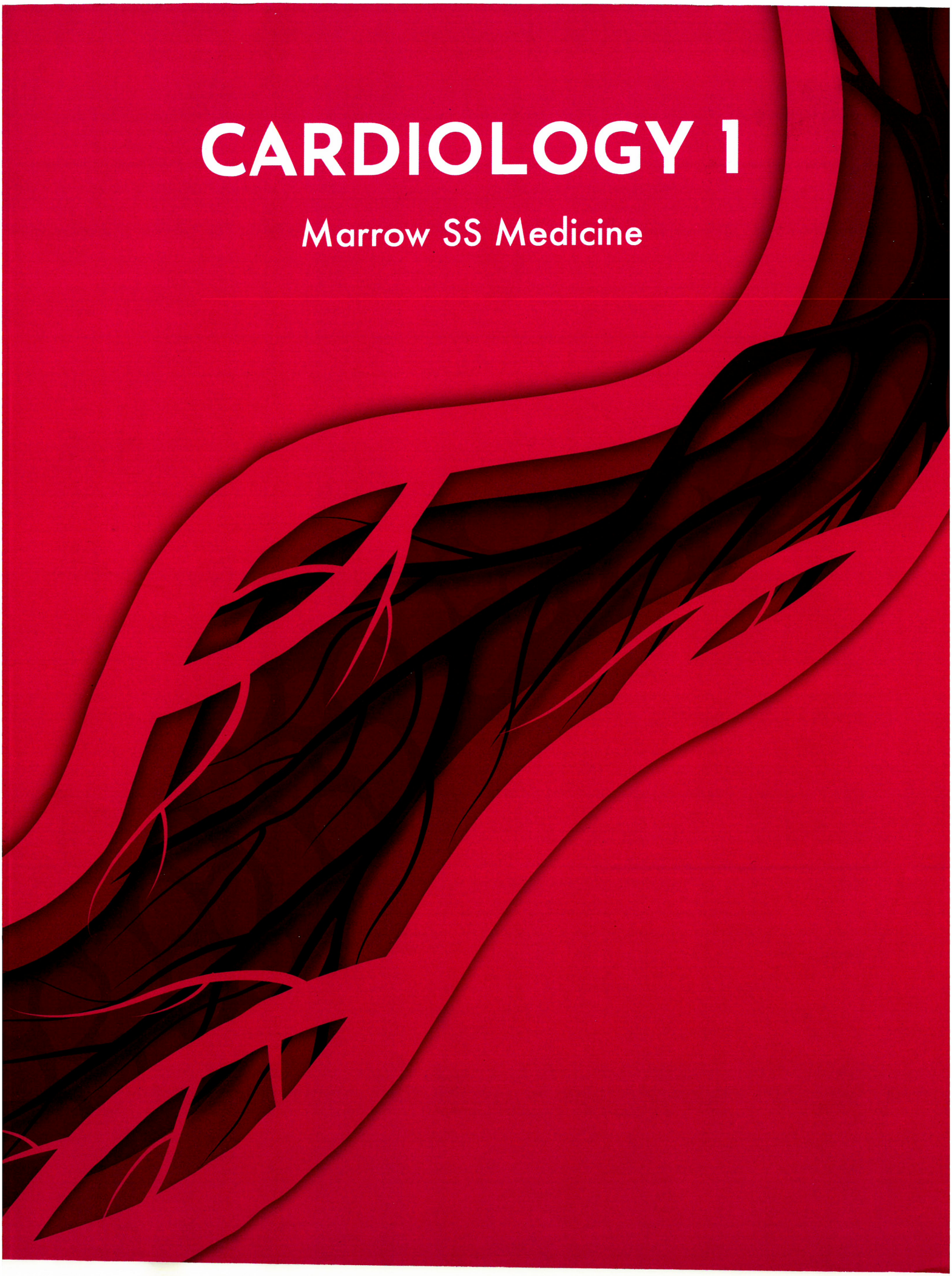
CARDIOLOGY 1

Marrow SS Medicine



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CARDIOLOGY BASICS - EMBRYOLOGY

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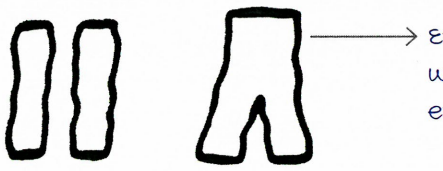
Development of the heart

00:00:59

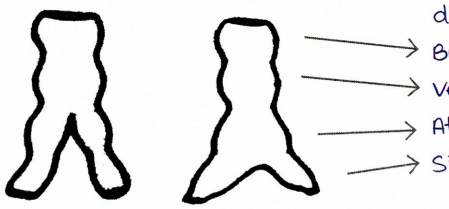
Cardiogenic area :

Derived from splanchnic mesoderm and forms 2 endocardial tubes.

Fusion to form single cardiac tube on day 19.



→ Endothelial tubes and fusion which begins cranially and extends caudally



Heart tubes show serial dilatations :

→ Bulbus cordis

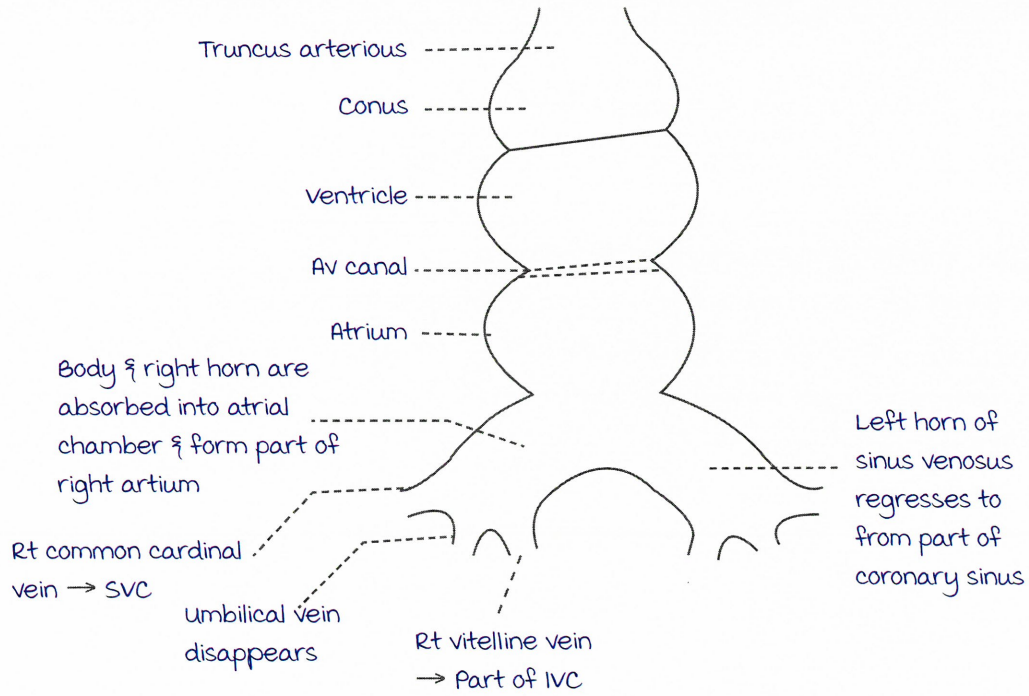
→ Ventricle

→ Atrium

→ Sinus venosus

Primitive heart tube has 5 dilatations :

- Truncus arteriosus.
- Bulbus/conus cordis.
- Primitive ventricle.
- Sinus venosus.



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Heart starts beating by day 22.
Looping occurs by day 23.

Fate of dilatations :

Truncus arteriosus :

It splits into aorta and pulmonary artery by fusion of spiral/conotruncal septum.

Clinical correlation :

- Anteriorly displaced septum : Tetralogy of Fallot.
- No spiral septum : Persistent truncus arteriosus.
- Failure of fusion of spiral septum : Transposition of great arteries.

Sinus venosus (SV) : Has 3 blood sources

- Umbilical vein.
- Vitelline vein.
- Right common cardinal vein.

Body and right horn of SV : Forms smooth part of right atrium (Sinus venarum).

Left horn of SV : Regresses to form part of coronary sinus.

Primitive atrium :

It becomes the rough part of the atria.

Primitive ventricle :

It becomes the rough part of both ventricles (Trabeculae carnae).

Bulbus cordis :

It becomes smooth part of the ventricle.

Smooth part on right side : Infundibulum, left side : Aortic vestibule.

Origin of other structures of the heart :

Cardiac jelly forms the connective tissue of the endocardium.

Epicardium : Neural crest cell derivative.

Myocardium : lateral plate mesoderm.

Vitelline vein gives rise to :

- Hepatic vein.
- Superior mesenteric vein.
- Portal vein.
- Inferior portion of IVC.

Formation of interatrial septum

00:10:21

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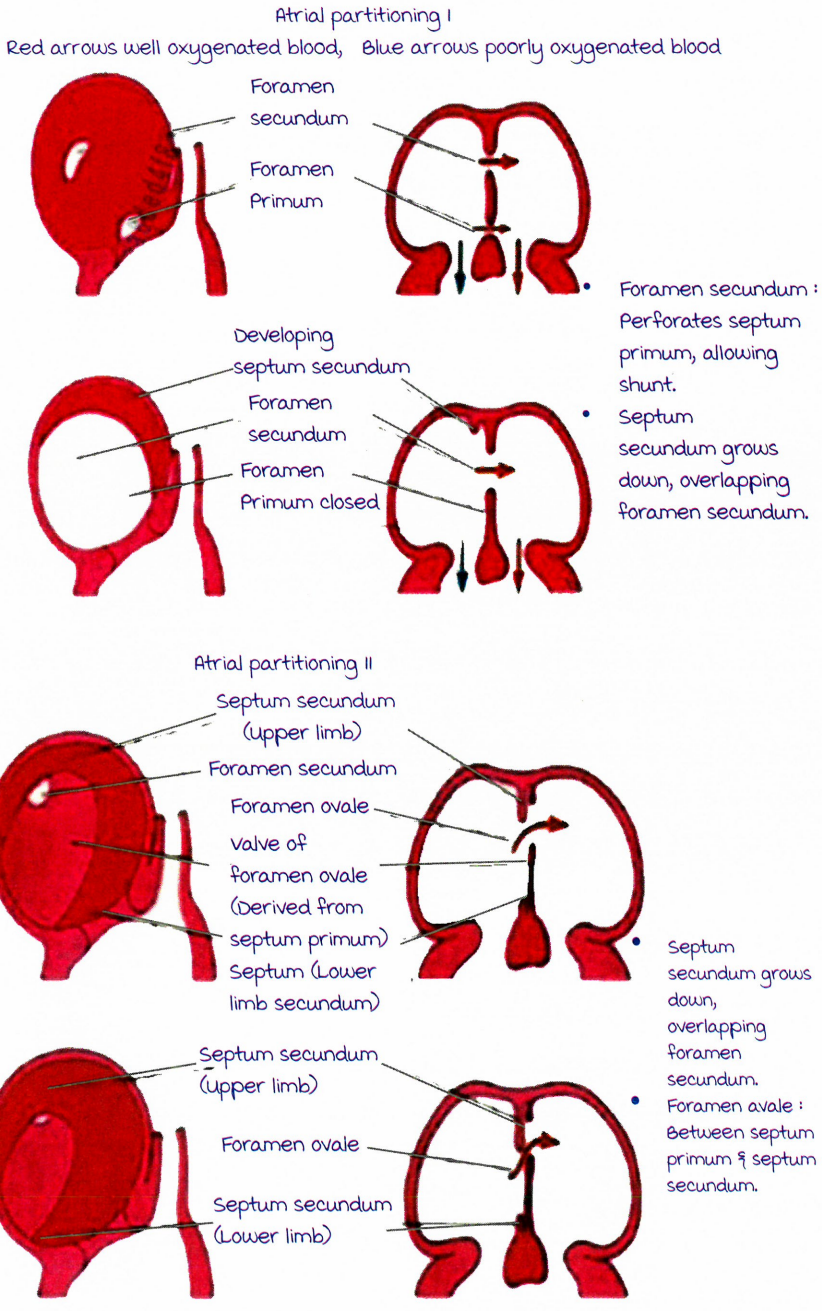
Septum primum : Forms fossa ovalis after birth.

Septum secundum : Forms limbus fossa ovalis after birth.

Foramen ovale : Formed between septum primum & septum secundum.



After birth :

- Ductus arteriosus closure : Functionally 12 to 24 hours after birth. Anatomically 2 to 3 weeks after birth.
- Ductus arteriosus : Ligamentum arteriosum.
- Left umbilical vein : Ductus venosus.

----- Active space -----

- Umbilical arteries : Medial umbilical ligament.
- Urachus : Median umbilical ligament.

Vascular embryology 00:13:46

Vascular embryology :

- Truncus arteriosus/Aortic sac is connected to dorsal aorta by 6 pairs of aortic arches.
- 1st arch artery forms maxillary artery.
- 2nd arch artery forms stapedial artery.
- 5th arch artery regresses.
- Left horn of aortic sac
- Left 4th arch artery
- Left dorsal aorta

  } Forms the aortic arch

- Right 4th arch artery
- Right dorsal aorta
- Right 7th cervical intersegmental artery

  } Forms the right subclavian artery.

- 3rd arch artery : Proximally gives rise to CCA and distally gives rise to internal carotid artery.
- Left 6th arch artery proximally gives rise to left pulmonary artery and distally ductus arteriosus.
- Right 6th arch artery forms right pulmonary artery.
- Right horn of aortic sac forms brachiocephalic trunk.
- Left 7th cervical intersegmental artery forms left subclavian artery.

Chambers of the heart 00:16:03

Right atrium (Internal features) :

Smooth part : Sinus venarum.

Rough part : Musculi pectinati.

They are separated by crista terminalis.

In the septal of wall of right atrium :

- Fossa ovalis.
- Limbus fossa ovalis.
- Triangle of KOCH : Location of AV node.

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Rt atrium : Internal features

Left atrium relations :

Anteriorly :

Ascending aorta & the pulmonary aorta.

Separated by transverse sinus of pericardium.

Posteriorly :

The descending aorta and the oesophagus.

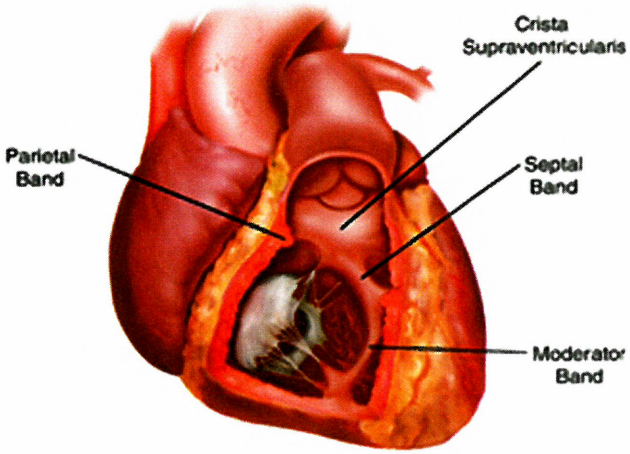
Separated by oblique sinus of pericardium.

Right ventricle :

- Rough inflow portion (Less) : made up of coarse trabeculae (Trabeculae carneae).
- Smooth outflow portion (Large) : Infundibulum.

They are separated by crista supraventricularis.

Right ventricle :



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Left ventricle :

- Fine trabeculae present.
- Smooth outflow portion : Aortic vestibule.
- No crista supraventricularis.

Right ventricle	Left ventricle
Upper and anterior part is called infundibulum or conus arteriosus.	Upper and anterior part is called aortic vestibule.
Wall of right ventricle is thinner than left ventricle (1 : 3) : 9 mm.	Wall of left ventricle is thicker : 27 mm.
3 papillary muscles : Anterior, posterior and septal.	2 papillary muscles : Anterior and posterior.
Moderator band present.	Moderator band absent.
Cavity : Crescentric in cross section.	Cavity : Circular in cross section.
Outflow part is infundibulum.	Outflow oart is aortic vestibule.
Supraventricular crest separates inflow part from outflow part.	No crest.

CARDIOLOGY BASICS - PHYSIOLOGY

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Terminology :

Chronotropy : Heart rate (SA nodal action potential).

Ionotropy : Myocardial contractility (myocardial action potential).

Dromotropy : Cardiac conduction velocity.

Bathmotropy : Cardiac excitability.

Lusitropy : Cardiac relaxation.

Sinoatrial node (SAN) action potential 00:03:25

Automaticity :

Ability to beat in absence of external stimulus.

Automatic tissues :

- SA node.
- AV node (Distal).
- His Purkinje system.
- Atrial cells near ostium of coronary sinus.

SAN is the pacemaker of heart because :

- It has the highest intrinsic firing rate.
- Maximum slope of pacemaker potential.
- Intrinsic firing rate of SAN is 100/min.
- But resting HR is 70-80/min (Because of resting vagal tone).

Effect of autonomic nervous system :

- Sympathetic system increases slope of pacemaker potential → ↑HR → Graph shifts to left.
- Parasympathetic system decreases slope of pacemaker potential → ↓HR → Graph shifts to right.

Pacemaker potential :

AKA prepotential/SA node action potential/Restless membrane potential/ Spontaneous diastolic depolarisation.

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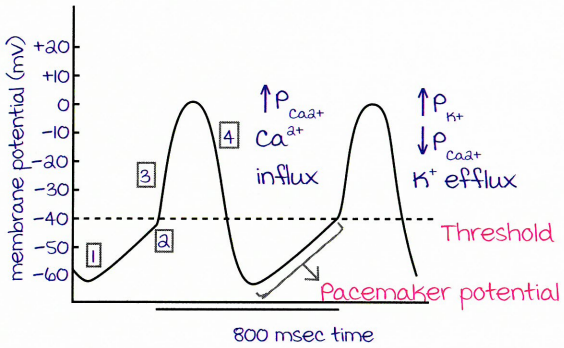
Ion channels during phases of SA node action potential :

1. 'Funny' sodium channels (I_f channels) are open ($\uparrow P_{Na+}$) and K^+ channels close.
2. Transient Ca^{2+} (T-type) channels open, pushing the membrane potential to threshold.
3. Long-lasting Ca^{2+} (L-type) channels open, giving rise to the action potential.
4. Opening of K+ channels, ($\uparrow P_{K+}$), and closing of Ca^{2+} (L-type) channels, hyperpolarising the cell.



a) Pacemaker potential :

Between -40 to -60 mV.

Determined by transient (T-type) Ca^{2+} channel/funny current (Na^+ channel).

K^+ channel (most important).

1. Closure of transient outward K^+
2. Opening of inward rectifying K^+

b) Depolarisation :

Slow inward L-type Ca^{2+} channel.

c) Repolarization :

Delayed rectifying K^+ channel.

Closure of L-type Ca^{2+} channel.

Funny current :

- Mostly sodium channel.
- K/A HCN (Hyperpolarization activated cyclic nucleotide channel).
- Responsible for prepotential.
- Seen in rods, cones, olfactory epithelium.
- Ivabradine :
  - Blocks funny current.
  - Decrease HR without affecting BP.
  - S/E : Visual field disturbance.

Ventricular myocardial action potential

00:20:03

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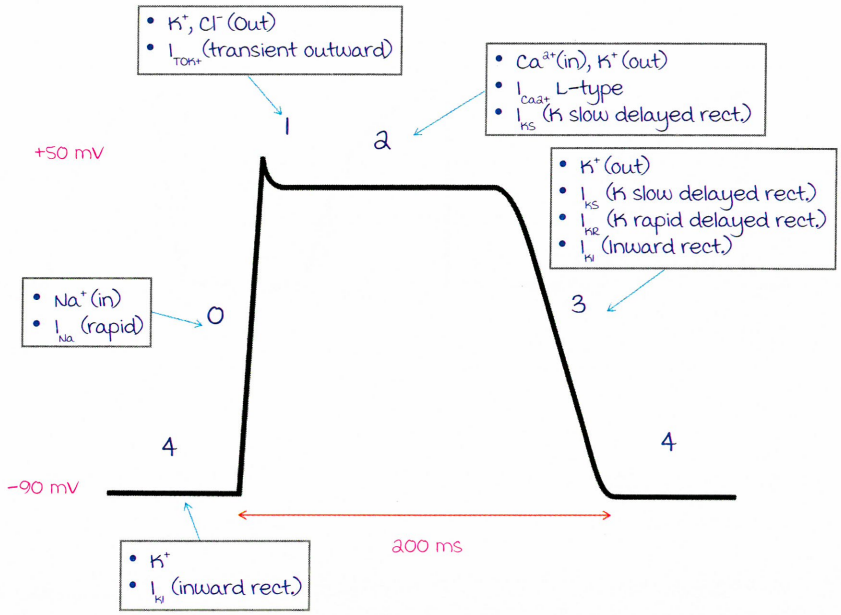

Phases of myocardial action potential :

Phase zero (Depolarisation) : Voltage gated fast acting Na^+ channel.

Phase 1 (Early repolarisation) : Closure of Na^+ channel & efflux of K^+ through TOK^+

Phase 2 (Plateau) : L-type Ca^{2+} channel & delayed rectifier K^+ channel

Phase 3 (Late repolarization) : Closure of L-type Ca^{2+} channel.

Phase 4 (Resting membrane potential) : Na^+-K^+ ATPase.

Vaughan William classification of anti-arrhythmic drugs

00:25:34

Based on predominant action of drugs.

Drug acting on	Mechanism	Class	Example
Phase 0	Na^+ channel blocker	I (I_A, I_B, I_C)	
Phase 4	Beta blocker	II	
Phase 3	K^+ channel blocker	III	BIDAS (Bretyllium, Ibutilide, Dofetilide, Amiodarone, Sotalol)
Phase 2	Ca^{2+} channel blocker	IV	Verapamil, Diltiazem

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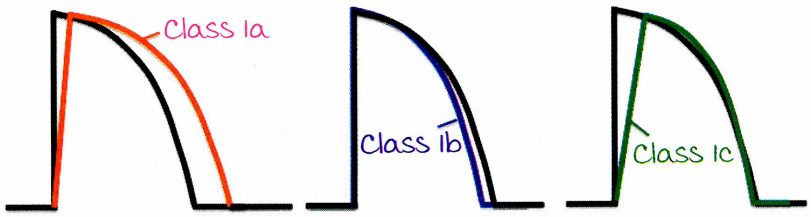
Class I drugs :

Class I Antiarrhythmic drug effects

On the ventricular action potential :



On the ECG :

↑QRS & ↑QT ↓QT ↑↑QRS

I_a	I_b	I_c
Quinidine Procainamide Disopyramide	Lignocaine Mexiletine Phenytoin	Propafenone Flecainide Encainide
Block Na^+ channel for 1–10 sec. Block in open state.	Block Na^+ channel for < 1sec. Block in closed state.	Block Na^+ channel for >10 sec. Block in open state.
Mild increase in QRS. Mild shift to right.	Do not shift phase zero. QRS duration unchanged.	Prolong QRS. Shift to right.
Block K^+ channel	Open K^+ channel	No action on K^+ channel
AP↑↑, QT interval ↑↑	QT interval short	QT interval normal

Miscellaneous concepts :

Myocardial oxygen consumption :

8 mL/100g/min.

- Most important parameter that determines oxygen consumption :
  - End diastolic volume.
  - Filling pressure.
- Consumption of fatty acid : Carbohydrate by heart is 70 : 30
  More oxygen requirement in fatty acid oxidation.
  Therefore by blocking fatty acid oxidation, it can be used for angina.
  PFOX Inhibitors (Partial fatty acid oxidation inhibitor) : Trimetazidine and Ranolazine.

Resting coronary blood flow :

60–90 mL/100 g/min or 225 mL/min.

Frank Starling law :

Force of contraction is directly proportional to the initial length of muscle fibre.

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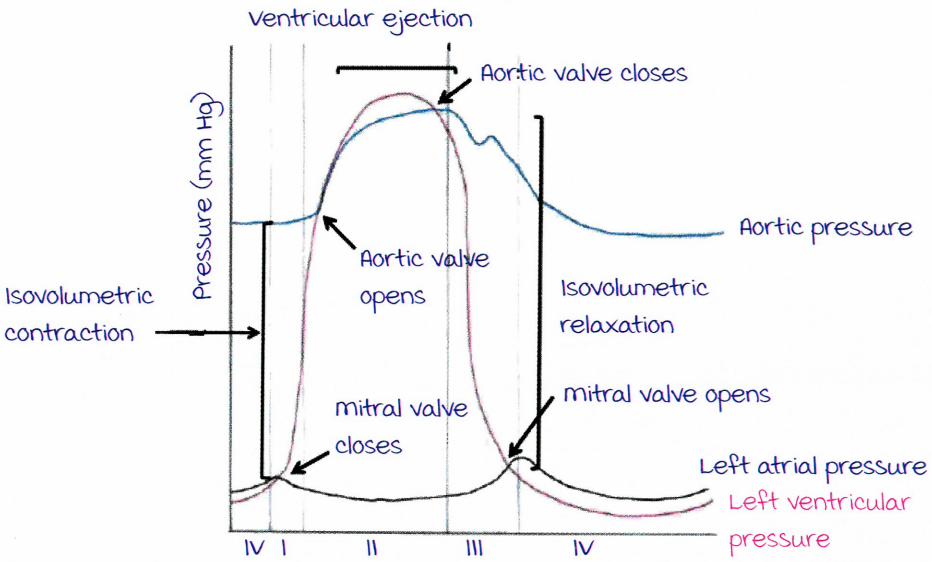
Ejection fraction (EF) :

EF = EDV – ESV/EDV = 120-50/120 = 70/120 mL.

The Cardiac cycle

00:38:27



Phase of cardiac cycle

Duration of cardiac cycle is 0.8 sec.

Systole	Diastole
0.3 s	0.5 s
1. Isovolumetric contraction. 2. Rapid ejection. 3. Reduced ejection.	1. Protodiastole. 2. Isovolumetric relaxation. 3. Rapid filling. 4. Reduced filling. 5. Atrial systole.

Atrial systole :

70-80% of filling of left ventricle is already completed before atrial systole.

AV valves open.

Active atrial contraction : Responsible for 20-30% of filling.

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Abnormality in atrial systole :

S4 : Vigorous contraction of atria.

Criteria for S4 :

- Normal healthy atria.
- Sinus rhythm.
- AV valve normal.
- Non-compliant hypertrophied non-dilated ventricle.

Isovolumetric contraction :

AV valve is closed (S1 is heard) just before isovolumetric contraction.

It is phase of systole where ventricular pressure increases.

At the end semilunar valves open (Ejection click).

Rapid ejection and reduced ejection flow.

Protodiastole (Hangout interval) :

Between the incidents when aortic pressure exceeds LV pressure and aortic valve closure (S2).

Isovolumetric relaxation :

At the end, AV valve opens.

Rapid filling and reduced filling flow.

PULSE

----- Active space -----

Definition and waveform of the pulse 00:01:06

Arterial pulse :

Pressure wave originating in the aorta due to ejection of blood during left ventricle systole and travels along the arterial wall at a rate of 5 m/s.

Column of blood : 50 cm/s.

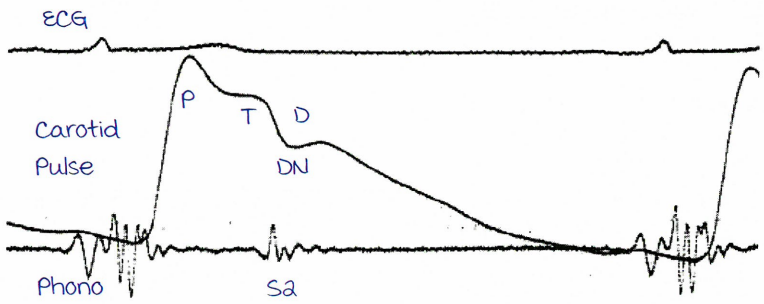
The upstroke of pulse coincides with S1 and the peak occurs well before S2.

Parameters to assess the performance of LV :

- Stroke volume : Increase correlates with a sharper upstroke and higher peak.
- Velocity of ejection : Increase correlates with a sharper upstroke and early peak.

Waveform :

- Percussion wave (P) : LV ejection (Stroke volume and velocity of ejection).
- Anacrotic notch : Between P and T.
- Tidal wave (T) : Aortic recoil (Vascular status).
- Dicrotic notch (DN)/Incisura : Corresponds to S2.
- Dicrotic wave (D) : Reflected wave from the periphery (Peripheral resistance).



Character and contour are always best felt at the carotids.

Ideal position :

Supine with the neck slightly turned to the site of palpation.

Simultaneous auscultation with palpation.

---- Active space -----

Determinants of arterial pressure, pulse and contour 00:15:10

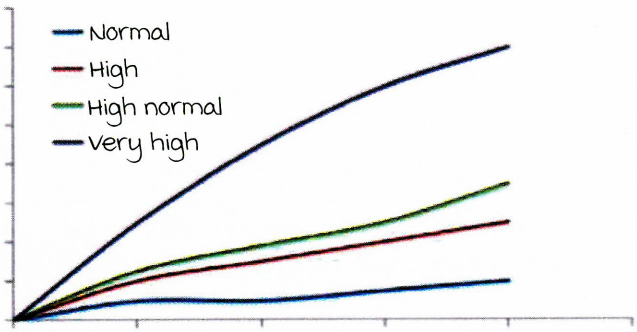
Incident pressure wave is dependent on :

- Compliance of aorta.
- The velocity of ejection.
- LV pump : Rate of change of pressure and peak aortic flow velocity.

Pulse wave velocity : Depends on stroke volume.

Arterial stiffness :

- Reflects true arterial wall damage.
- Has an independent predictive value for cardiovascular events.
- A marker of earlier target organ damage.
- Vessel wall cushioning : Healthy vessels absorb the energy of the pulse wave.
- Stiffness of vessel increases in aging vessels → ↑Pulse wave velocity.
- The gold standard to measure arterial stiffness : Pulse wave velocity



Central systolic blood pressure : High central SBP correlates with bad prognosis.

Compliant vessel :

- ↓ Pulse wave velocity.
- Reflected wave (Dicrotic wave) returns to central aorta later in diastole.
- Augments diastolic BP.
- Increased coronary perfusion.

Stiff vessel :

- ↑ Pulse wave velocity.
- Reflected wave arrives earlier in the systole.
- Augments systolic BP.
- ↓ Diastolic BP and ↓ coronary perfusion.

Augmented pressure : Difference between P1 and P2.

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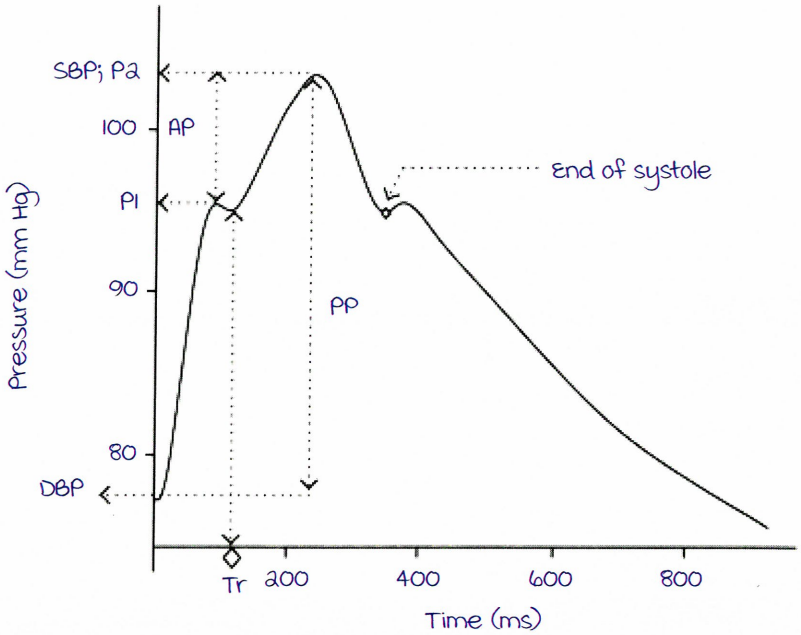


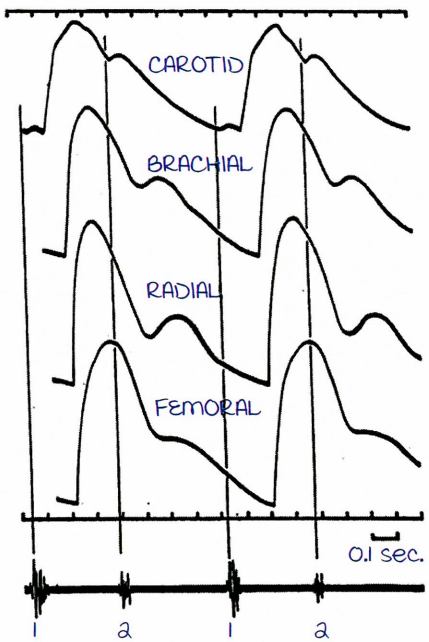
Normal pulse

00:25:59

Changes in pulse from the centre to the periphery :

1. Upstroke becomes steeper.
2. An anacrotic notch becomes less apparent.
3. The dicrotic notch becomes smoother.

Radio femoral delay is present in the coarctation of the aorta.



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The normal delay for pulse wave transmission (msec)	
Carotid	30
Brachial	60
Radial	80
Femoral	75

Rate of the pulse

00:30:30

Sinus tachycardia :

- Hypovolemia
- Sepsis
- Myocarditis.
- Cardiogenic shock (Anterior wall MI).
- High output states.

Sinus bradycardia : Regular rhythm <60 bpm.

- Drugs (Beta blockers).
- Myxedema.
- Hypothermia.
- Increased ICT (Cushing's reflex : Bradycardia + Hypertension).
- Inferior wall MI (RVMI).

Relative bradycardia (Faget sign) :

There is a decrease in heart rate with a rise in body temperature.

Causes :

Infectious	Non-infectious
Typhoid. Legionella. Q fever. Scrub typhus. Dengue (Rare). Malaria (Rare). Leptospirosis (Rare).	Drug fever. CNS : meningitis, encephalitis. Lymphomas. Factitious fever. Calcium channel blockers. β blockers.

Regularity and amplitude of pulse

00:41:15

----- Active space -----

Regularity of pulse :

- Rapid regular pulse : SVT/VT.
- Rapid irregular pulse : Atrial fibrillation, atrial flutter, or atrial tachycardia with varying block.
- Regularly irregular pulse : Ventricular premature complexes.

Amplitude/volume of pulse :

Low volume pulse : Hypokinetic pulse.

High volume pulse : Hyperkinetic pulse.

Pulse volume correlates with stroke volume.

Hyperkinetic pulse :

- The amplitude of upstroke increases.
- High cardiac output and low PR.
- The dicrotic wave becomes less prominent.
- Complete heart block & elderly : High volume pulse with low CO.

Corrigan's or water hammer pulse :

The brisk upstroke of the carotid pulse when felt in association with a large amplitude pulse with rapid descent.

- Aortic regurgitation.
- Aortic sinus rupture.
- Aortopulmonary window.
- Persistent ductus arteriosus.

Hypokinetic pulse :

- Small or diminished amplitude.
- Decreased LV stroke volume.
- LV dysfunction.

Slow rising/anacrotic/pulsus tardus :

- In severe aortic stenosis.
- The upstroke is slow & the peak is delayed nearer to S2.
- Ejection velocity is significantly increased due to obstruction.
- Volume increment is low : The increase in radius & the tension in the aortic wall will be expected to be slow.

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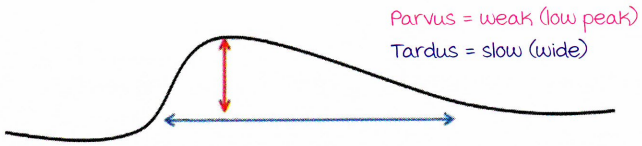
- Increased velocity of the aortic jet through the stenotic valve decreases the lateral pressure thereby contributing to a slower rate of pressure rise in the aorta.

Very severe AS : Pulsus parvus et tardus.



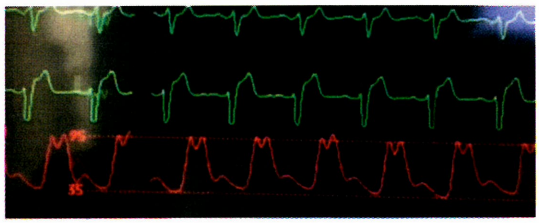
Upstroke high, amplitude low : Severe mitral regurgitation.

Contour abnormalities

01:01:50

Pulsus bisferiens :

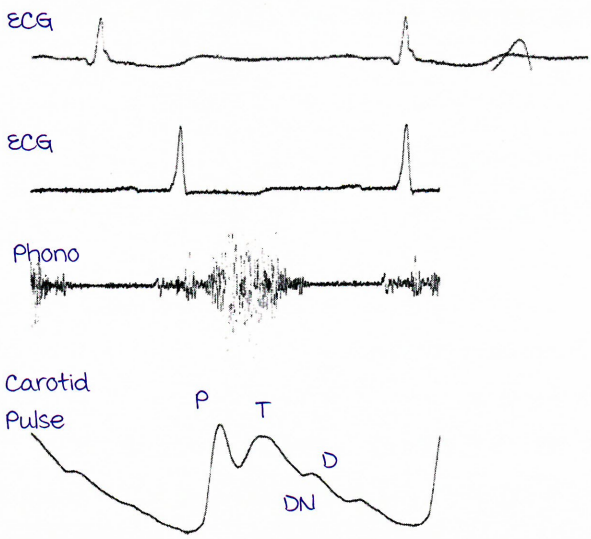
- Severe AR.
- AR with mild AS.
- HCM.



2 peaks in systole :

- The high SV accompanying the AR will cause a large-amplitude pressure wave : P & T will be prominent.
- The increased velocity of turbulent flow at peak systole due to stenosis decrease in lateral pressure in the aorta due to the Venturi effect : AS + AR.
- Leads to a drop in pressure rise during the middle of systole.
- Radial is more preferable as carotid shudder may mask it.

Pulsus bisferiens in severe AR with AS

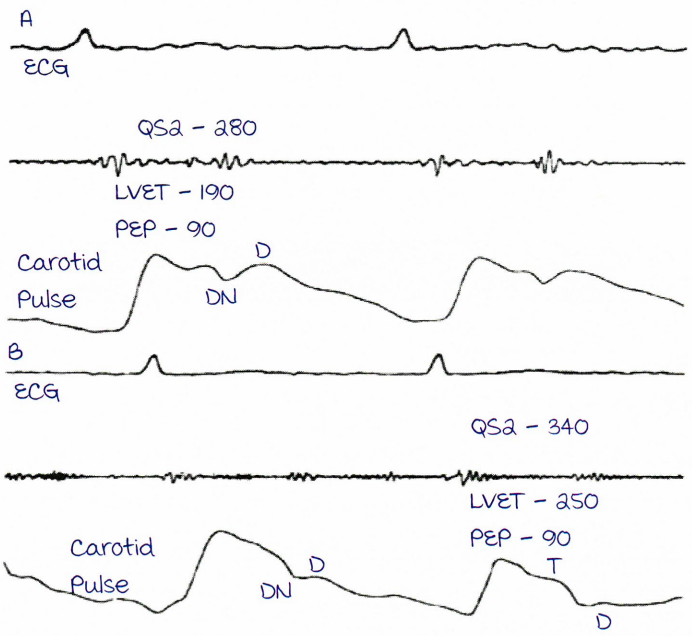


Hypertrophic cardiomyopathy :

----- Active space -----

- The ventricle is hypercontractile & ejects the blood very fast. This leads to a very rapid rise.
- The systolic anterior motion of the mitral leaflet → Obstructs ejection.
- In the late systole when the interventricular pressure begins to fall, it gives rise to a second peak in the pulse.

Dicrotic pulse :



2 peaks : One in systole, the other one in diastole → Radial artery.
Increased PVR → Prominent Dicrotic pulse.
Seen in LV dysfunction, conditions with low CO → Cardiac tamponade.

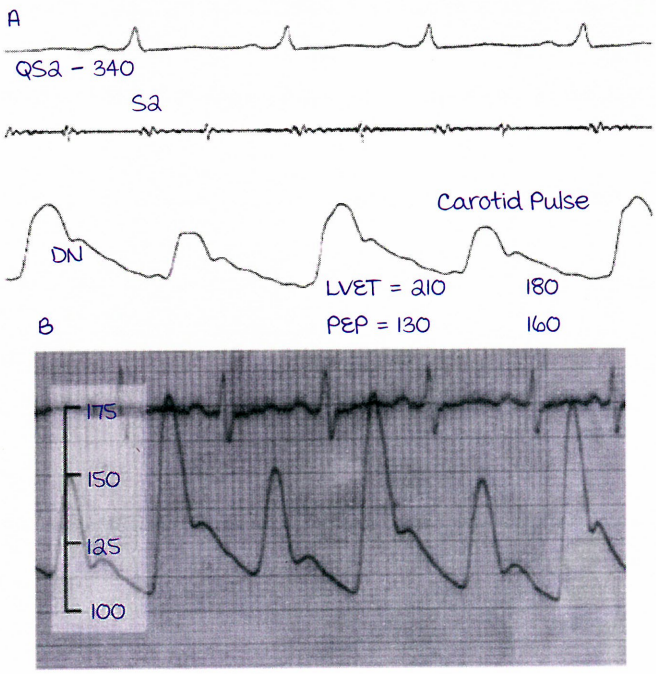
Pulsus alternans :

Amplitude changes beat to beat, alternating between higher & lower pulse amplitude as a result of alternating stroke volume. Seen in LV failure.

- Premature depolarization releases more calcium.
- The increased calcium is therefore available for the post premature beat, thereby increasing its contractile force.
- The calcium uptake & release may fluctuate alternatingly from beat to beat.

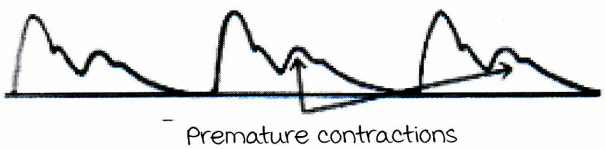
----- Active space -----

The sphygmomanometer is used to detect it.



Pulsus bigeminy :

- Normal beat alternating with a premature contraction.
- SV of the premature beat diminished & pulse varies in amplitude accordingly.
- May masquerade as pulsus alternans, but regularly irregular rhythm.
- Pulse volume increases following a VPC due to longer pauses & more diastolic filling.



Premature contractions

Exception : HCM (Dynamic obstruction) → Pulse volume falls following a VPC → Brockenbrough sign.

Pulsus paradoxus

01:20:35

A/K/A Pulsus normalis aggregans.

- Normally, SBP falls by 10 mm Hg during inspiration.
- If during inspiration, SBP falls > 10 mm Hg → Pulsus paradoxus.
- Best felt at femoral pulse.

Etiology :

----- Active space -----

- Cardiac tamponade.
- Constrictive pericarditis.
- COPD.
- Thrombus or obstruction in the SVC.
- Pulmonary embolism.

Pulsus Paradoxus

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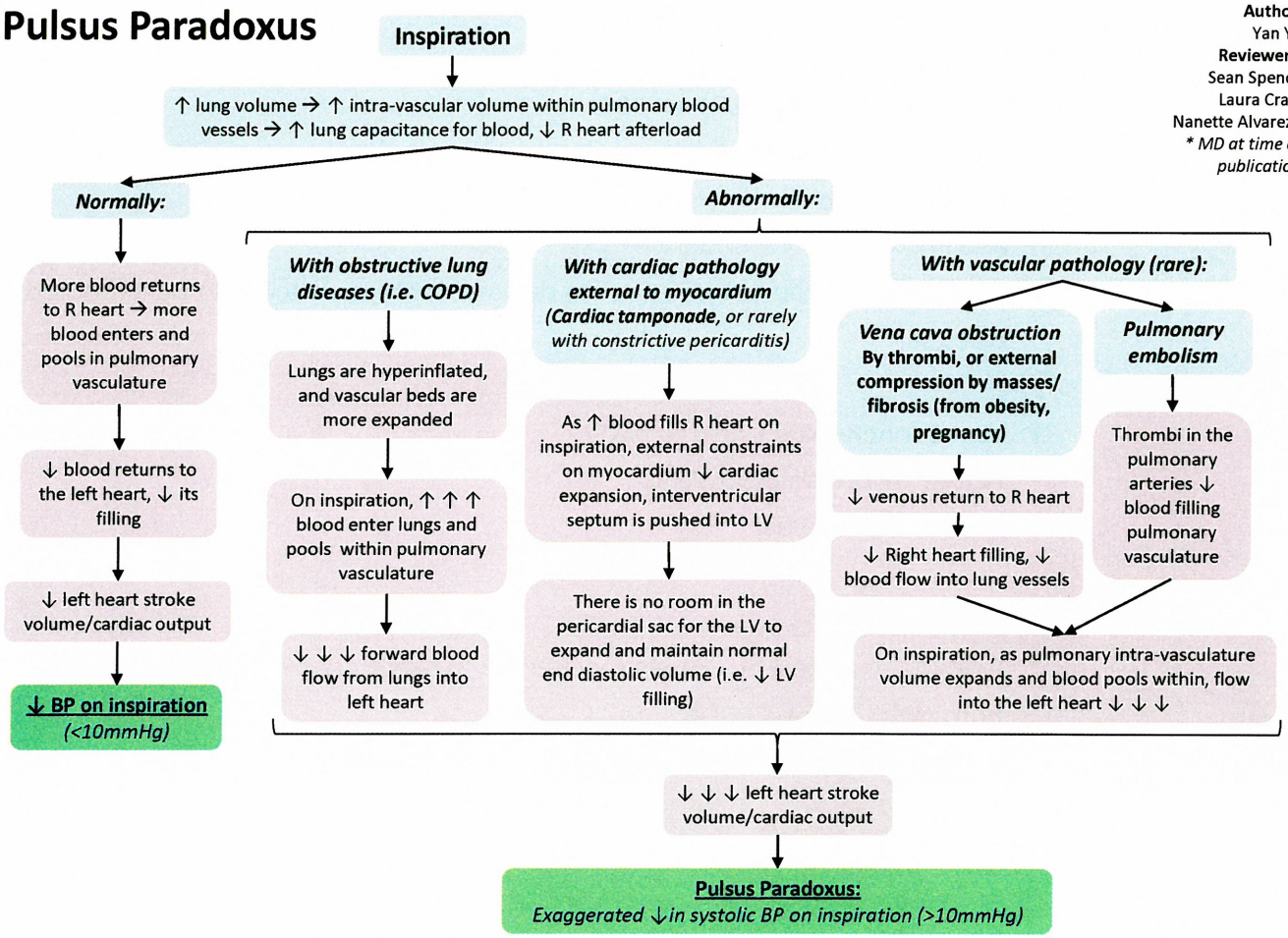

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JUGULAR VENOUS PULSE

Introduction

00:00:30

JVP : Jugular venous pulse/Jugular venous pressure.
A window into the right heart.

Giovanni Maria Lancisi (1654 to 1720) :

- "Systolic fluctuation of the jugular vein" in a patient who at necropsy had tricuspid regurgitation.
- Also known as Lancisi sign/Venous corrigan.
- Cor aegrotari non potest : First to declare that heart will also be affected in disease.

Sir James Mackenzie (1853 to 1925) :

- Father of british cardiology.
- Named JVP waves.

Polygraph :

- A device invented by James mackenzie to simultaneous measurement of both JVP & Carotid pulse.
- Inadvertently laid the foundation for the lie detector.

Articles :

- Sir James Mackenzie's heart : Article elaborating his Coronary artery disease progression from stable angina → Unstable angina → Death.
- Study of the pulse : Arterial, venous and hepatic and of the movements of the heart (1902) by James mackenzie : Describes the waves of JVP.

Thomas lewis : Father of modern cardiology.

Karl Frederik Wenckebach (1899) :

Wenckebach phenomenon (Type I 2nd degree heart block) was first described in JVP (1887).