MEDICINE - CVS
NEET-SS

CONTENT

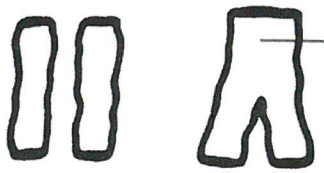
CARDIOLOGY BASICS – EMBRYOLOGY

Development of the heart

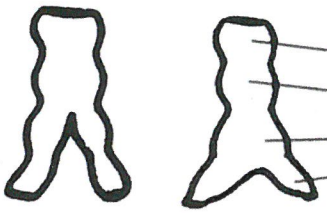
00:00:59

Cardiogenic area derived from splanchnic mesoderm and forms 2 endocardial tubes.

Fusion to form single cardiac tube on day 19.

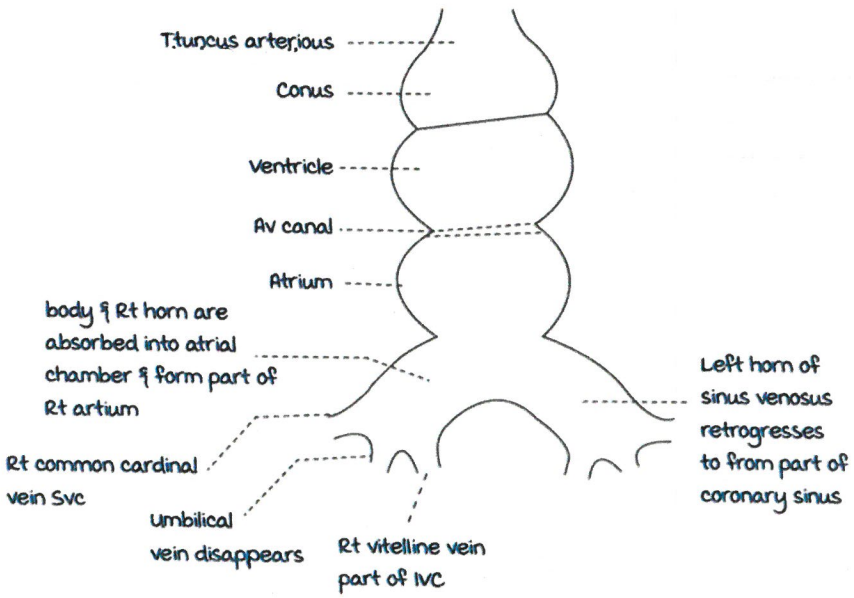


1. Endothelial tubes and fusion which begins cranially and caudally extends caudally



2. Heart tubes show serial dilatations
   - Bulbus cordis
   - ventricle
   - Atrium
   - Sinus venosus

Primitive heart tube has 5 dilatations : Truncus arteriosus
Bulbus/conus cordis
Primitive ventricle
Primitive atria
Sinus venosus



Heart starts beating by day 22.

Looping by day 23.

Active space

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]

Sinus venosus has 3 blood sources :

- Umbilical vein
- Vitelline vein
- Right common cardinal vein

Body and right horn of SV : 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 cardia :

It becomes smooth part of the ventricle.

Smooth part on right side : infundibulum

left side : aortic vestibule

Cardiac jelly forms the connective tissue of the endocardium.

Epicardium : neural crest cell derivative.

myocardium : lateral plate mesoderm.

Vitelline vein : gives rise to

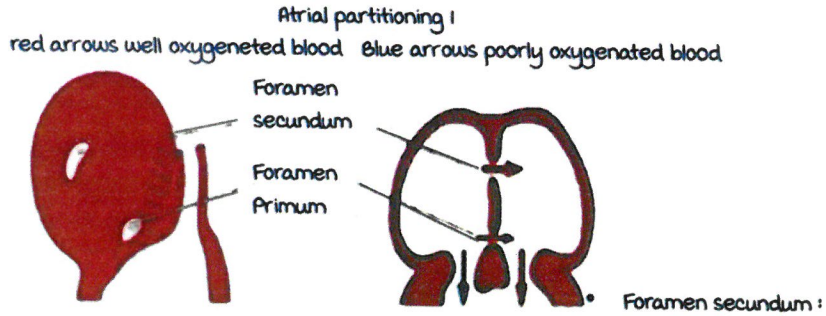
- Hepatic vein

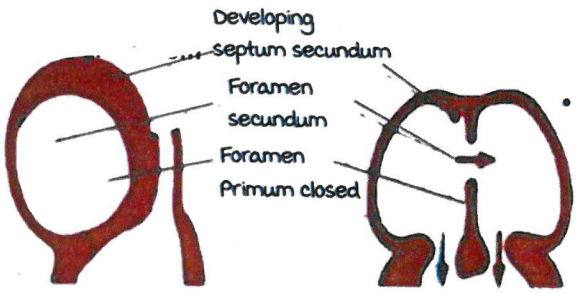
Active space

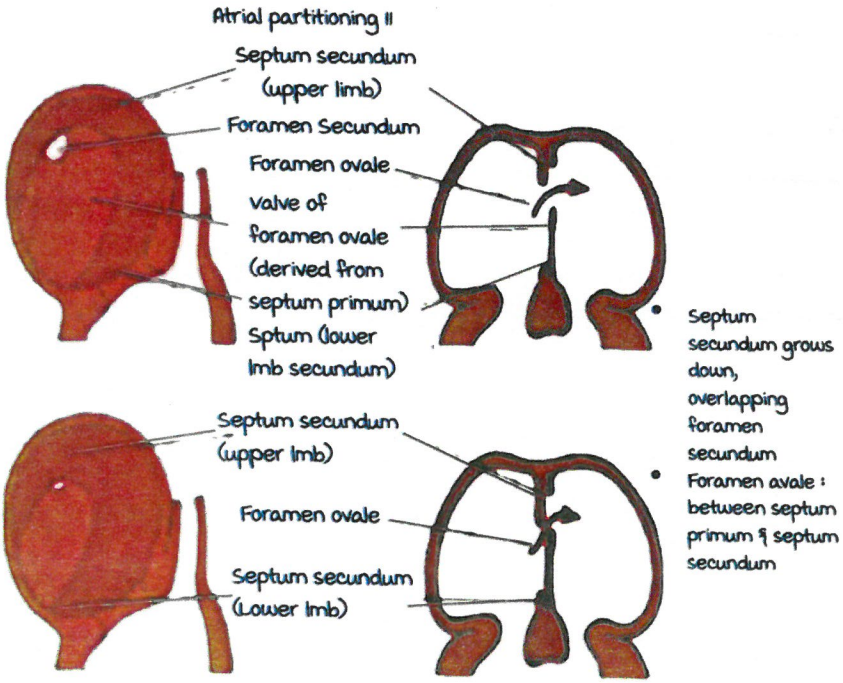
- Superior mesenteric vein
- Portal vein
- Inferior portion of IVC

Formation of interatrial septum 00:10:31

- Septum primum forms fossa ovalis after birth.
- Septum secundum forms limbus fossa ovalis after birth.
- Foramen ovale is formed between septum primum & septum secundum.







Active space

Leave Feedback

After birth :

- Ductus arteriosus closes : functionally 12 to 24 hours after birth.
  Anatomically 2 to 3 weeks after birth.
- Ductus arteriosus : ligamentum arteriosum.
- Left umbilical vein : ductus venosus.
- Umbilical arteries : medial umbilical ligament.
- Urachus : median umbilical ligament.

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

  } Aortic arch

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

  } Right subclavian artery

- 3rd arch artery : proximal 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 00:16:12

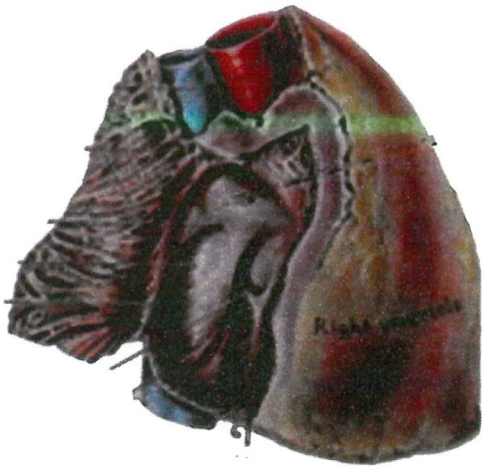
Right atrium – internal features :

Smooth part : sinus venarum.
Rough part : musculi pectinati
They are separated by crista terminalis.
In the septal of wall of right atrium :

Active space

- Fossa ovalis
- Limbus fossa ovalis
- Triangle of KOCH AV node

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.

Inflowing lower part	Outflowing upper part
It develops from primitive ventricle It is large size and lies below the supraventric crest. It is rough due to respond of it is smooth and forms upper I the muscular ridges – the inch conical part of the right trabeculae carneae.	It develops from bulbs cordis It is small in size and lies above the supraventric crest. It forms ventricular chamber– the most of the right ventricular infundibulum, whih gives rise chamber to pulmonary trunk

Active space

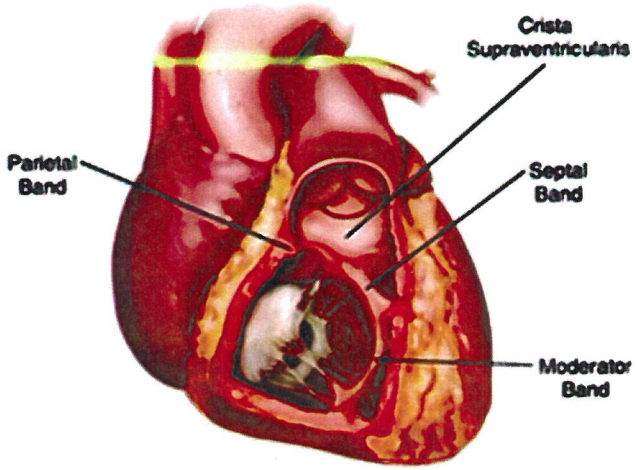
Leave Feedback

Left ventricle :

Fine trabeculae present.

Smooth outflow portion – aortic vestibule.

No crista supraventricularis.



Inflowing lower part	Outflowing upper part
It develops from primitive ventricle	It develops from bulbus cordis
It lies below the aortic vestibule	It is between the membranous part of the interventricular septum and anterior cups of the mitral valve
It is rough due to presence it is smooth and forms smooth of trabecule carneae and small upper part	The arotic forms most of the left vestibule, which gives rise to the ventricular chamber ascending aorta.

Active space

CARDIOLOGY BASICS - PHYSIOLOGY

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).

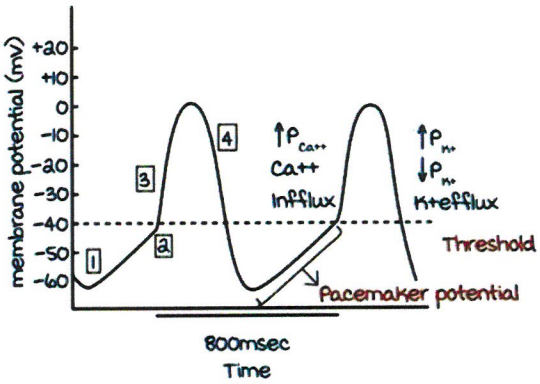
Intrinsic firing rate of SAN is 100/min.
But resting HR is 70-80/min (because of resting vagal tone).

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

- Parasympathetic system decreased slope of pacemaker potential → ↓HR → Graph shifts to right.

Pacemaker potential/prepotential/SA node action potential/ Restless membrane potential/Spontaneous diastolic depolarisation :

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



a) Pacemaker potential :
Between -40 to -60 mV.
Determined by Transient (T-type) Ca^{a+} 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^{a+} channel.

c) Repolarization :
Delayed rectifying K^+ channel.
Closure of L-type Ca^{a+} channel.

Funny current :
mostly sodium channel.

Active space

Called HCN (hyperpolarization activated cyclic nucleotide channel).

Responsible for prepotential.

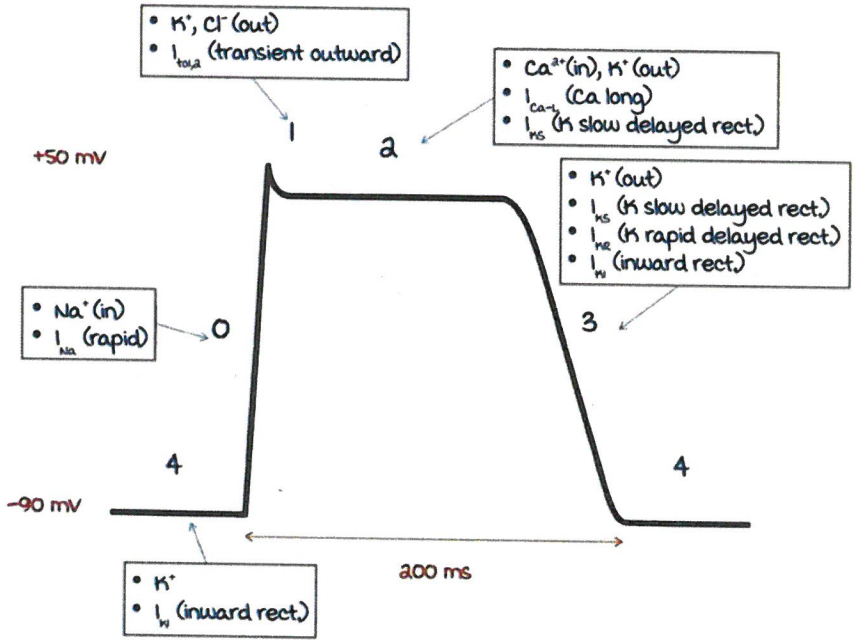
Seen in rods, cones, olfactory epithelium.

Ivabradine – Blocks funny current.

Decrease HR without affecting BP.

S/E : visual field disturbance.

Ventricular myocardial action potential 00:20:03



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

Active space

Vaughan William classification of anti-arrhythmic drugs

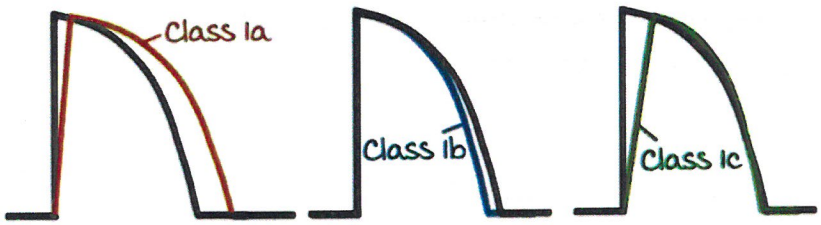
Based on predominant action of drugs.

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

Class I drugs :

Class I Antiarrhythmic Drug Effects

On the Ventricular Action Potential :



On the ECG :

↑QRS & ↑QT ↓QT ↑↑QRS

Active space

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.	Don't 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

Myocardial oxygen consumption is 8 ml/100gm/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.

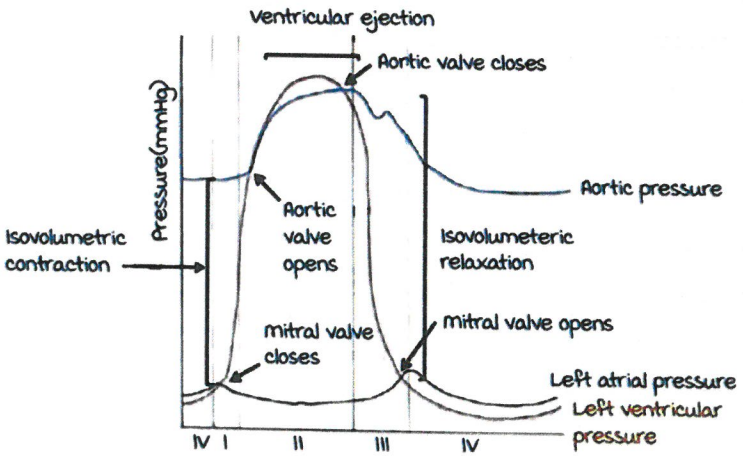
  PFOX Inhibitors (Partial fatty acid oxidation inhibitor) :

  Trimetazidine and Ranolazine.

- Resting coronary blood flow: 60–90 ml/100g/min or 225 ml/min.
- Force of contraction is directly proportional to the initial length of muscle fibre.
- EF = EDV − ESV/EDV = 120–50/120 = 70/120 ml.

The Cardiac cycle

00:38:27



Phase of cardiac cycle

Leave Feedback

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 left ventricle is already completed before atrial systole. AV valves open.
  Active atrial contraction – responsible for 20-30% of filling. Abnormality in atrial systole : S4 (vigorous contraction of atria)
  Criteria for S4 : Normal healthy atria.
  Sinus rhythm
  AV valve normal
  Noncompliant Hypertrophised nondilated 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 follow.

- Protodiastole (hangout interval)
  Between the incidents when aortic pressure exceeds LV pressure and aortic valve closure (S2).
- Isovolumetric relaxation:
  At the end AV valve open.
- Rapid filling and reduced filling follow.

Active space

PULSE

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 : 50cm/s.

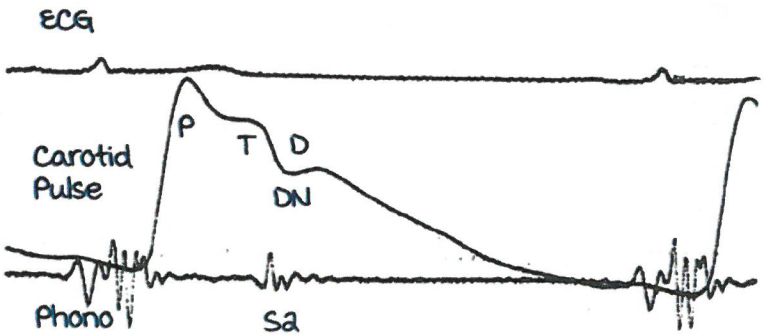
The upstroke of pulse coincides with S1 and the peak occurs well before S2.

Parameters to assess the performance of LV :

- Stroke volume : Increase correlates with a sharper upstroke and higher peak.
- The 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)/Insisura : Corresponds to S2.
- Dicrotic wave : 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

Leave Feedback

00:15:10

Incident pressure wave is dependent on :

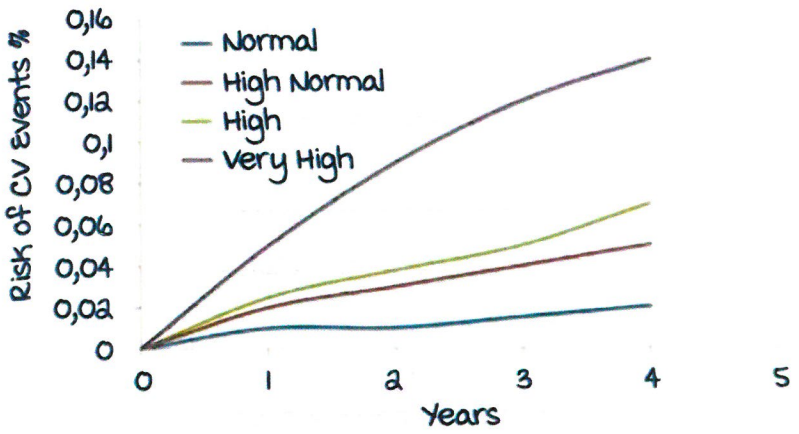
- Compliance of Aorta.
- The velocity of ejection.
- LV pump : Rate of change of pressure and peak aortic flow velocity.

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 → Increased pulse wave velocity.
- The gold standard to measure arterial stiffness : Pulse wave velocity

Aortic Pulse Wave Velocity and Probability of a CV Event



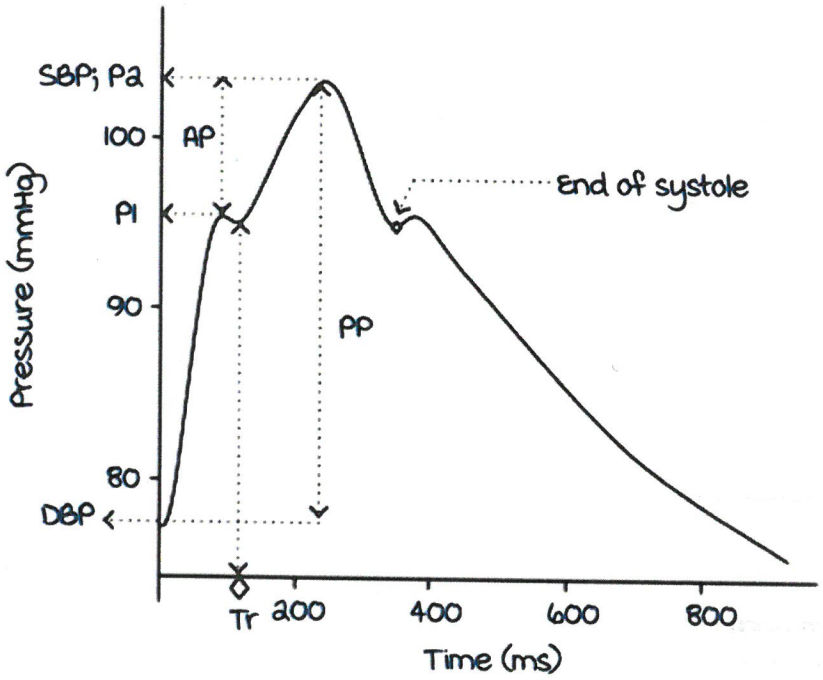
Active space

Central systolic blood pressure :

- High central SBP correlates with a bad prognosis.

Compliant vessel :	Stiff vessel :
↓ Pulse wave velocity	↑ Pulse wave velocity
Reflected wave (Dicrotic wave) returns to central Aorta later in diastole.	Reflected wave arrives earlier in the systole.
Augments diastolic BP.	Augments systolic BP
Increased coronary perfusion	↓ Diastolic BP and ↓ coronary perfusion.

- Augmented pressure : Difference between P1 and P2



Normal pulse

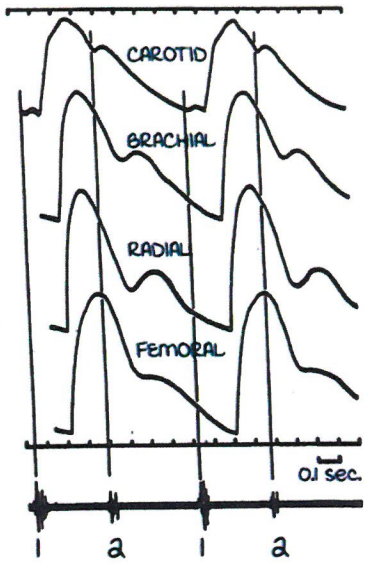
00:25:59

Changes in pulse from the center 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.

Leave Feedback



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 (β 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.

Active space