MEDICINE - CVS
NEET-SS

CONTENT

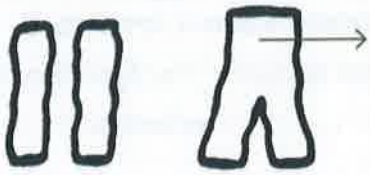
CARDIOLOGY BASICS – EMBRYOLOGY

Leave Feedback

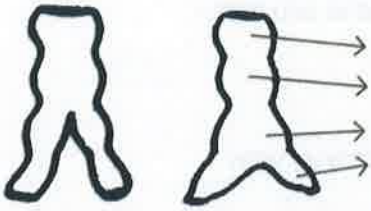
Development of the heart 00:00:59

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

Fusion to form single cardiac tube on day 19.

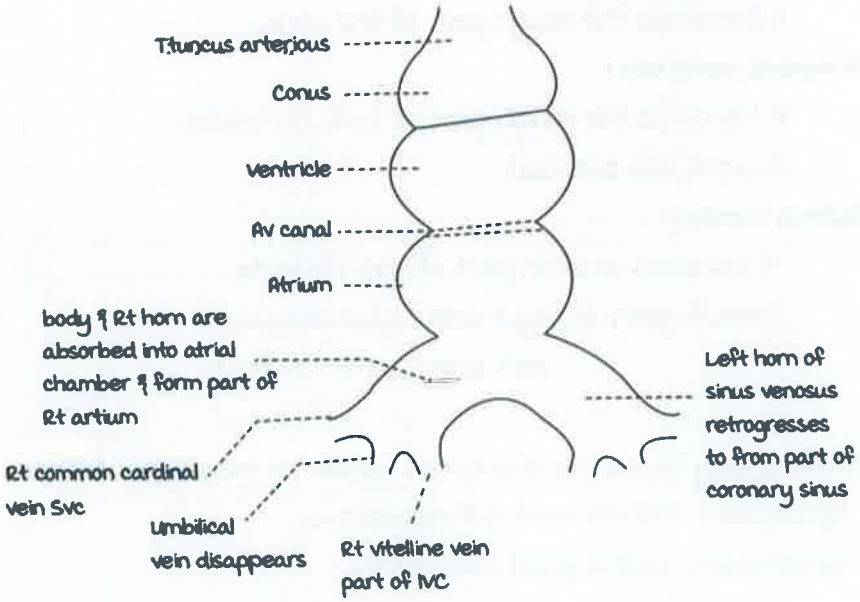


1. Endothelial tubes and fusion which begins cranially and caudally extends caudally



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

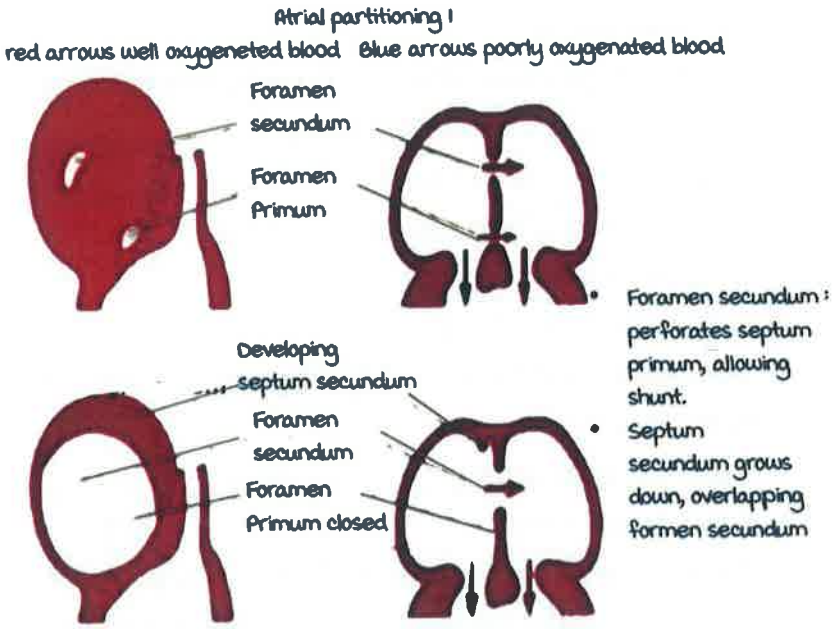
- Hepatic vein

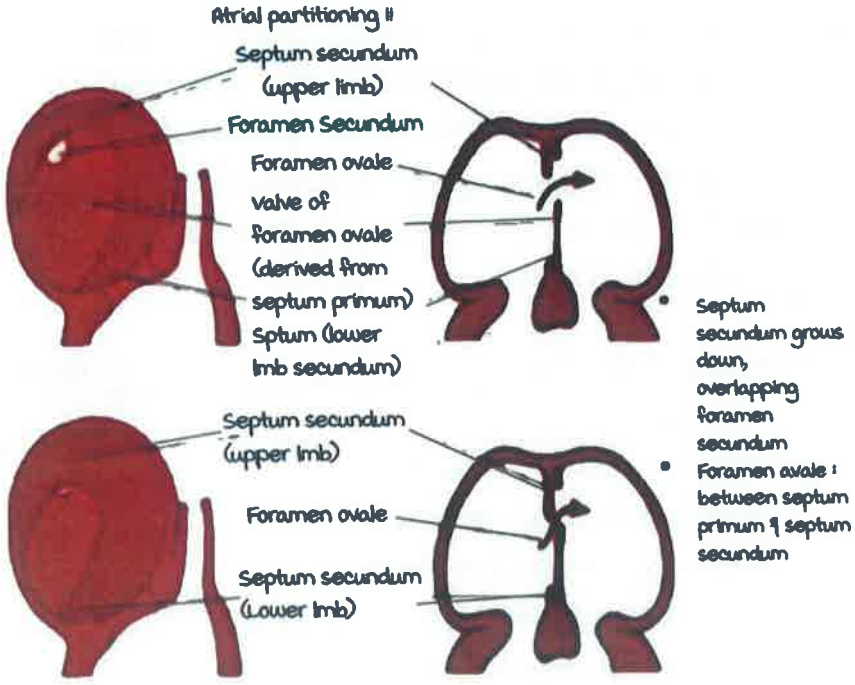
Leave Feedback

- 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.





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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

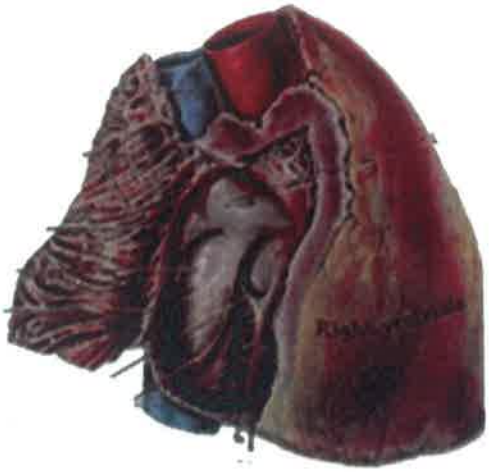
They are separated by crista terminalis.

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

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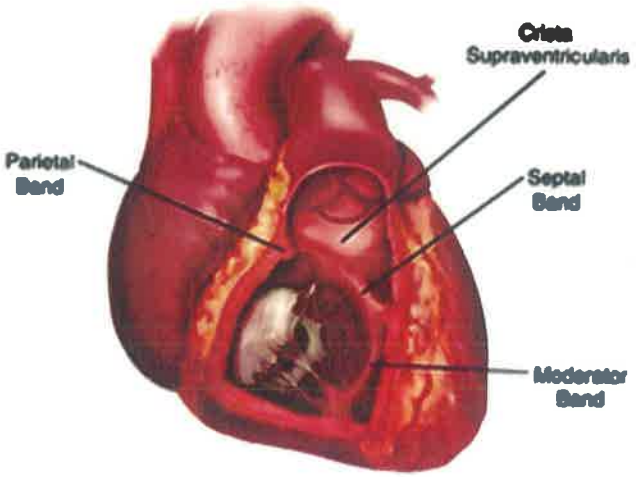
Left ventricle :

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.

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).

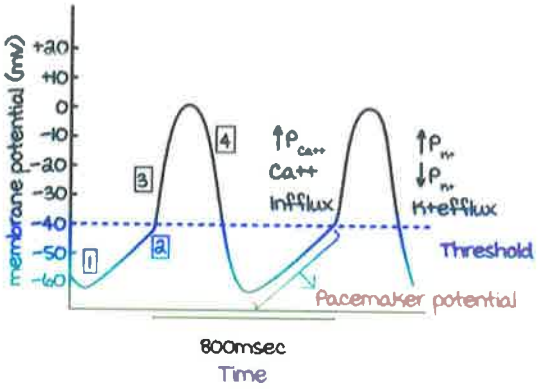
Intrinsic firing rate of SAN is 100/min.
But resting HR is 70-80/min (because of resting vagal tone).

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

Leave Feedback

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^{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.

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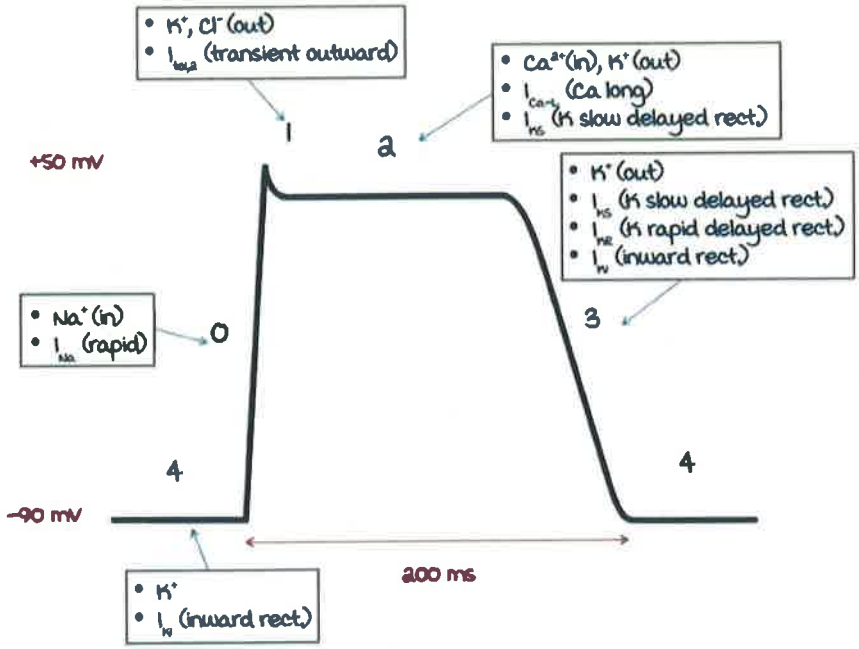
Called HCN (hyperpolarization activated cyclic nucleotide channel).
Responsible for prepotential
Seen in rods, cones, olfactory epithelium.
Ivabradine – Blocks funny current.
Decrease HR without affecting BP.
S/E : visual field disturbance.

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

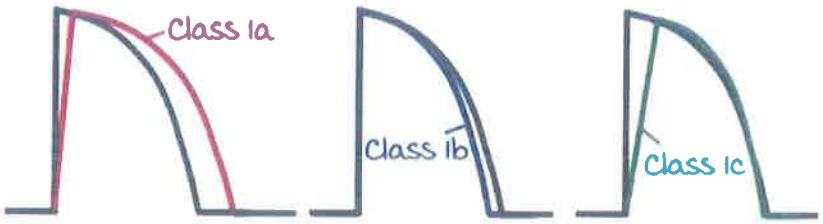
Based on predominant action of drugs.

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

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.

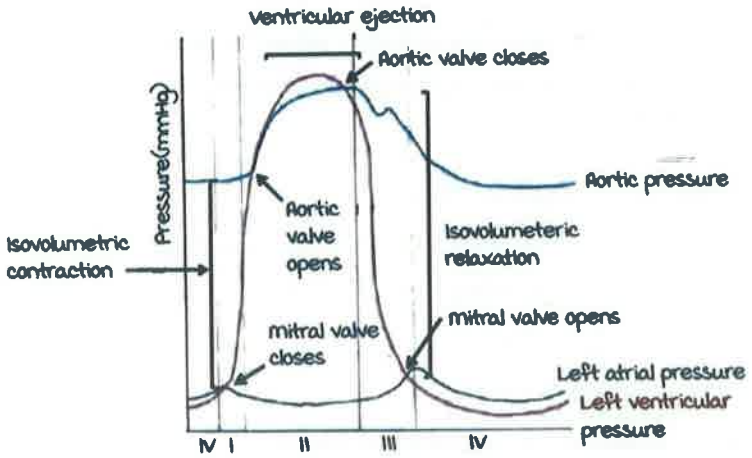
  Therefore by blocking fatty acid oxidation, it can be used for angina.

  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.

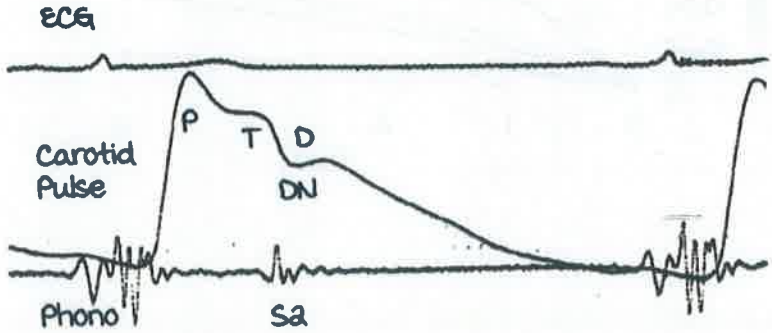
Parameters to assess the performance of LV :

- 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.

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Determinants of arterial pressure, pulse, and contour

Incident pressure wave is dependent on :

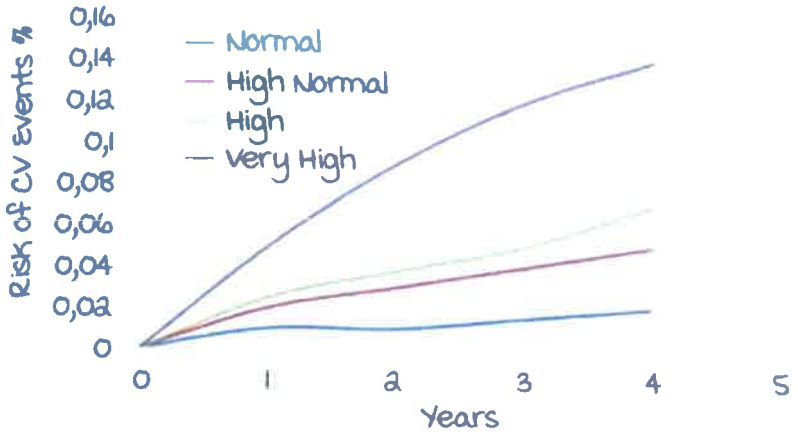
- Compliance of Aorta.
- The velocity of ejection.
- LV pump : Rate of change of pressure and peak aortic flow velocity.

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



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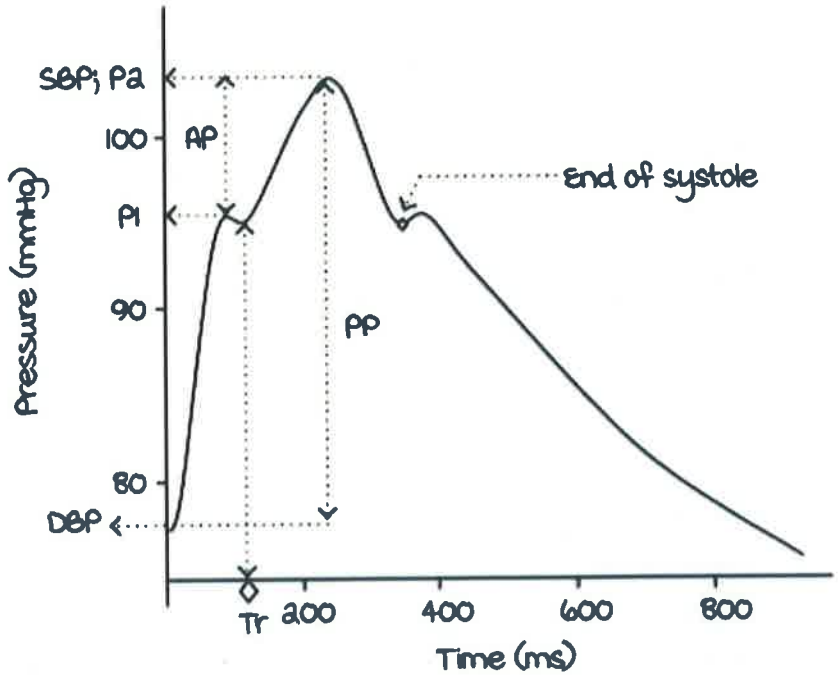
Central systolic blood pressure :

- 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 PI and Pa



Normal pulse

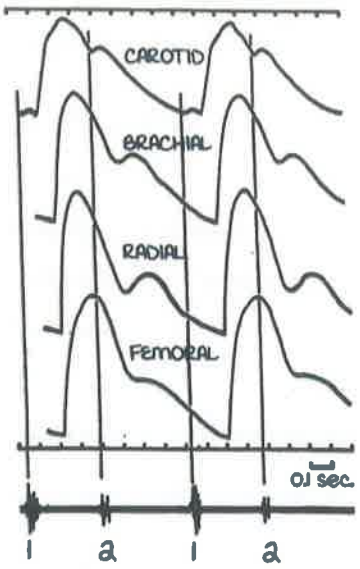
00:25:59

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.

Active space



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

- Causes :

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

Regularity and amplitude of pulse 00:41:31

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 00:45:53

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.

Severe Aortic stenosis : Slow rising/anacrotic/pulsus tardus.

- 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.
- 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.

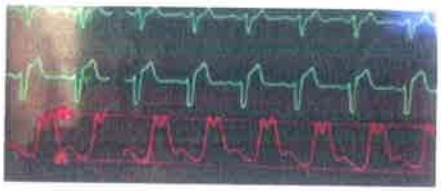
Upstroke high, amplitude low – severe mitral regurgitation.

Contour abnormalities

01:02:17

Pulsus bisferiens :

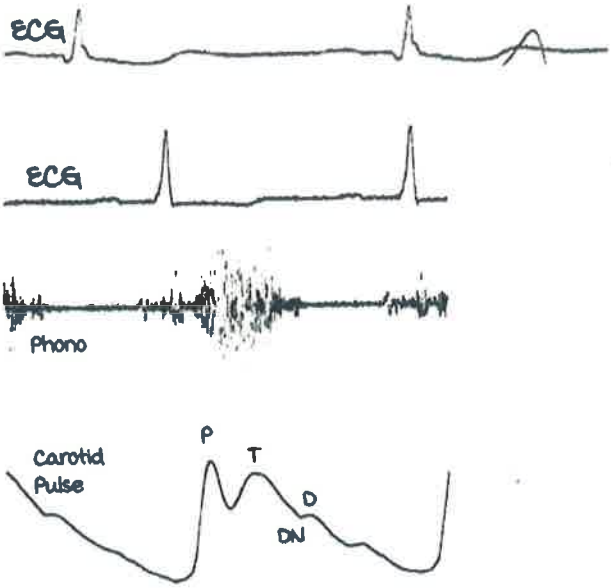
- Severe AR
- AR with mild AS
- HCM



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.

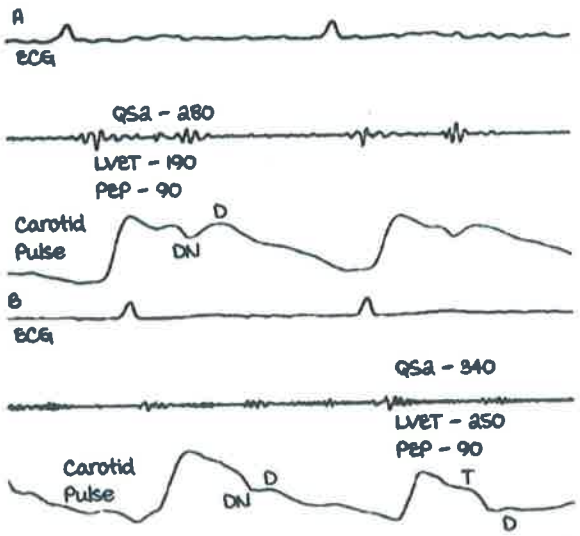
Pulsus bisferiens in Severe AR with AS



Hypertrophic cardiomyopathy :

- 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 :



a 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.

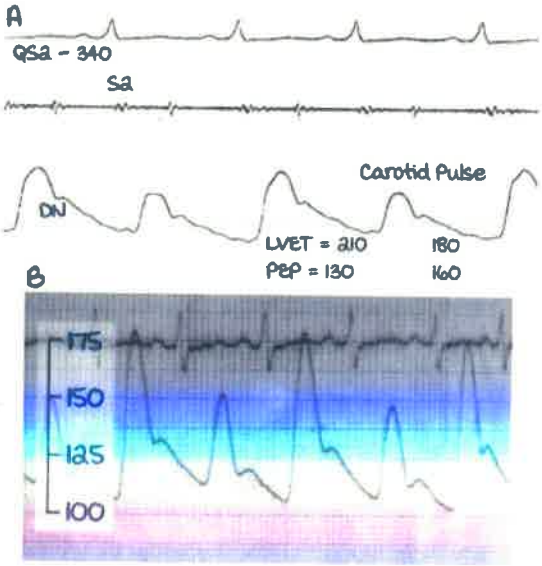
Active space

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

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.

Active space

Pulsus Paradoxus

01:21:09

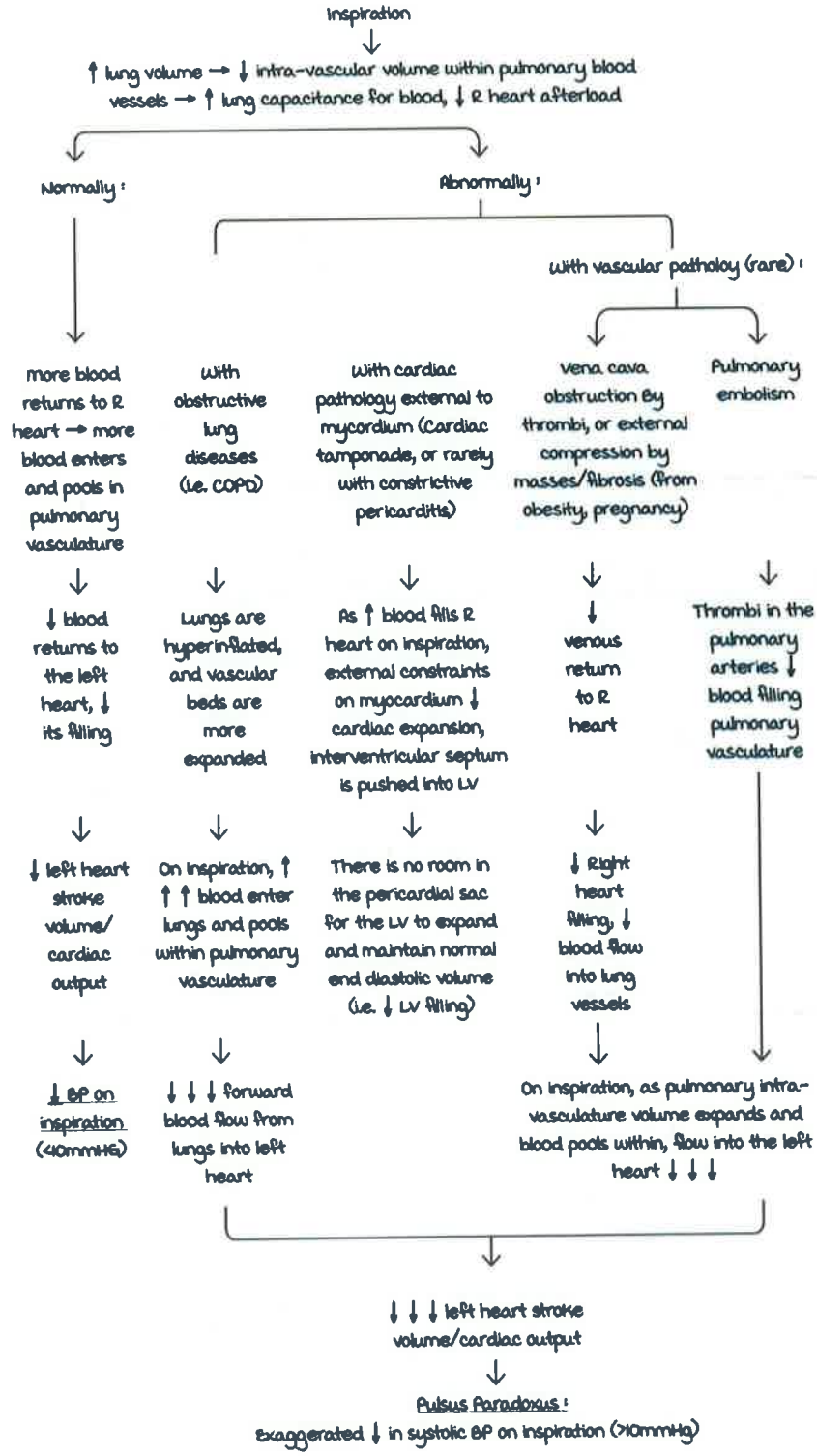
A/K/A Pulsus normalis aggregans.

- Normally, SBP falls by 10 mmHg during inspiration.

- If during inspiration, SBP falls > 10 mmHg → Pulsus paradoxus.
- Best felt at Femoral pulse.

Etiology:

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



JUGULAR VENOUS PRESSURE

It reflects the relationship between the right heart hemodynamics, volume of blood in the venous system and venous tone.

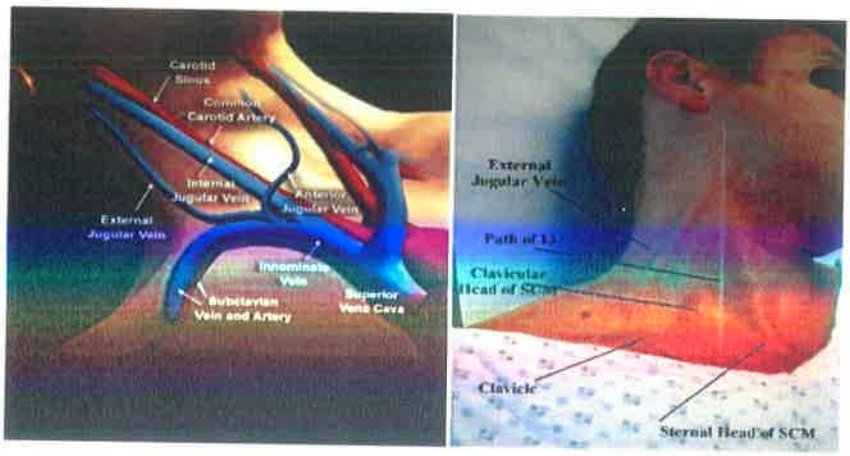
JVP describes ,

In diastole : Filling pressure/RVEDP.

In systole : Right atrial pressure.

Internal jugular vein

00:02:21



Locating the Carotid triangle.

It begins medial to the mastoid process, at the base of skull.

It runs inferiorly and joins the subclavian vein to form the right innominate vein (continues as SVC).

First vein preferred for cannulation to put a dialysis catheter.

EJV is not preferred over IJV because :

- EJV has valves which may interfere with measurement of JVP.
- Not visible due to vasoconstriction in hypotensive states Passes through multiple fascial planes.

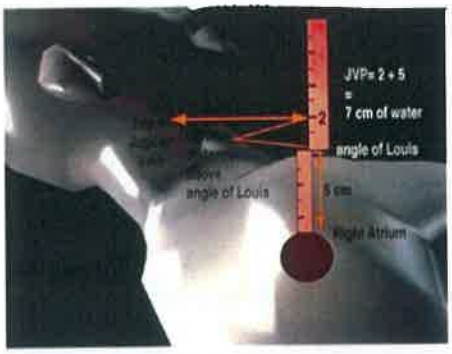
Right IJV is preferred because :

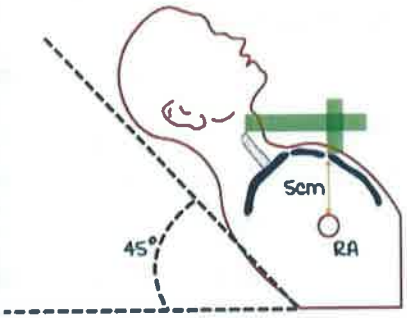
- Straight line course through innominate vein to the SVC and right atrium.
- Less likely for extrinsic compression from other structures in the neck.

IJV	Carotid pulse
• Superficial and lateral in the neck. • Better seen than felt. • 2 peaks and 2 troughs. • Descents more obvious than crests. • Digital compression abolishes venous pulse. • JVP falls during inspiration. • Abdominal compression elevates jugular pressure.	• Deeper and medial in the neck. • Better felt than seen. • Has single upstroke only. • Upstroke brisker & visible. • Digital compression has no effect. • Do not change with respiration. • Abdominal compression has no effect on carotid pulse.

Measuring the JVP

00:07:23





Supine position is the best position to appreciate the wave form.

The patient is kept in 45° position at the time of measuring JVP (the top most point comes under the clavicle).

Angle of Louis :

- At 5 cm from the center of the right atrium.
- Normally the top most point of the venous column is 2 cm from the Angle of Louis.

Normally the JVP = 7 cm of H_2O

method to measure : 2 scales and a tape.

Elevated JVP = > 9 mm of H_2O or > 7 mm Hg

Active space

Elevated JVP

00:12:18

1. Increased RV filling pressure and reduced compliance : RVH (PAH, RCM)
   RV failure
   RV infarction
   Pulmonary stenosis
2. RV inflow obstruction (Increased RAP) : RA myxoma
   Tricuspid stenosis
3. Fluid overload states : Cirrhosis
   Renal failure
   Excessive fluid administration
4. SVC obstruction : Pulseless rise in JVP

Kussmaul sign & Hepatojugular reflex

00:16:04

Kussmaul sign :

Failure of JVP to fall with inspiration (Normal JVP falls with inspiration).

Seen in : Chronic constructive pericarditis
RCM
RHF
RV infarction
} Increased RV filling pressure.

Absent in cardiac tamponade.

Hepatojugular reflex :

Firm pressure is applied to Periumbilical region for at least 10 – 30 seconds.

In a normal person : Transient rise in JVP to < 1 cm.

Positive Hepatojugular reflex :

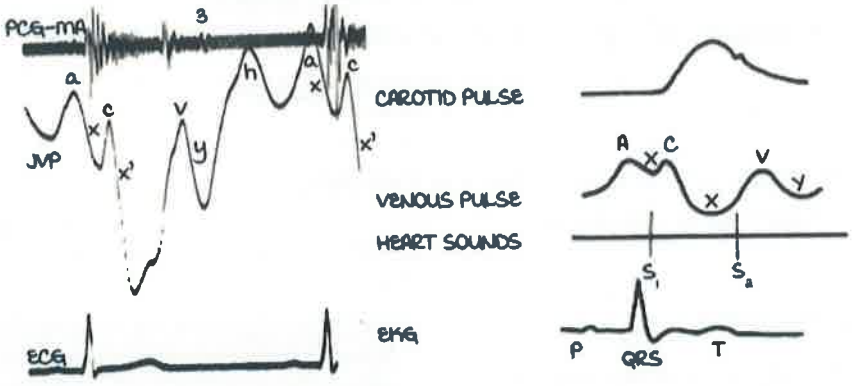
JVP elevated for > 3 cm for at least 15 seconds.

Seen in : Impending RVF
TR

False positive : COPD

False negative : Budd-Chiari syndrome.

Normal JVP waveforms : 'a' wave



3 positive waves : a, c & v.

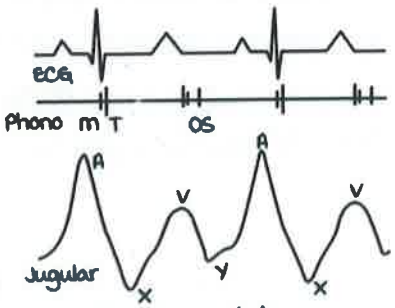
3 descents : x, x' & y.

Rarely : h wave.

The waveforms are based on the right atrial pressures.

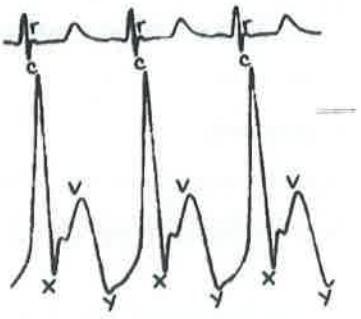
a wave :

- Because of atrial systole (effective RA contractions).
- most dominant positive wave on JVP.
- Pre-systolic wave.
- Follows P wave, precedes S_1 and carotid pulse in ECG.



Prominent 'a' wave

- Prominent 'a' wave :
  Forceful atrial contraction (RV inflow obstruction, decreased ventricular compliance).
- Giant 'a' wave/Cannon wave :
- Right atrium forcefully contract against a closed tricuspid valve.
  1. Irregular cannon wave :
     Ventriculoatrial dissociation.
     VT, CHB.



Regular cannon wave

a. Regular cannon wave :

AVRT, AVNRT or Junctional rhythm.

- Absent 'a' wave : When no effective atrial contraction as an atrial fibrillation.

Normal JVP waveforms : x descent, c wave & x' descent

00:39:05

x descent :

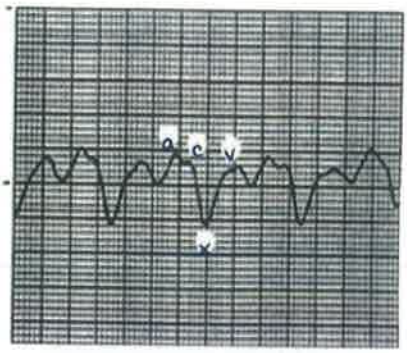
During ventricular systole (due to atrial relaxation).

most prominent negative wave.

Ends just before S2.

It is larger than y descent.

more prominent during inspiration.



Prominent x descent :
Cardiac tamponade

Normal x descent : Constrictive pericarditis

Prominent x descent : Cardiac tamponade
RCM

Absent/reduced x descent : Moderate - severe TR
Atrial fibrillation
Poor RV contraction

c wave :

Not usually visible.

Due to :

1. Upward bulge of closed TV, in IVC
2. Transmitted carotid artery pulsations.

x' descent :

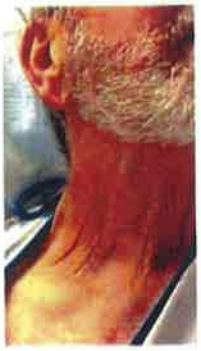
Systolic trough after 'c' wave.

Due to fall in right atrial pressure during early RV systole.

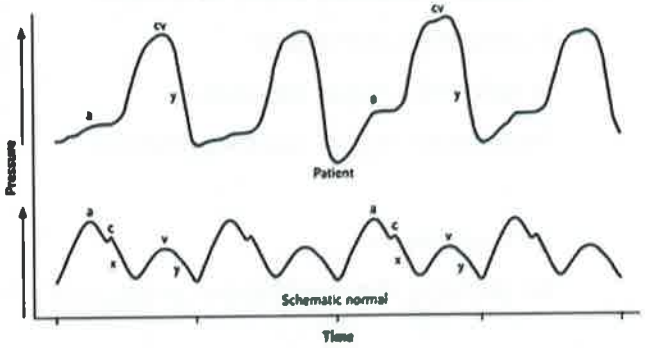
Active space

Normal JVP waveforms : v wave, y descent & h wave

v wave :
Begins in late systole and ends in early diastole.
Rise in RA pressure due to continued RA filling during ventricular systole when TV closed.
Roughly synchronous with carotid upstroke and corresponds to Sa.
Giant v wave : TR (Increased RA volume during systole).



Lancisi sign



cv wave

Prominent v wave : ASD with MR.
RCM

y descent :
Diastolic collapse wave (down slope v wave).
It begins and ends during diastole well after Sa.
Decline of RA pressure due to RA emptying during early diastole when TV opens.
Early rapid feeling phase.
Prominent y descent : CCP (Rapid filling in the first 1/3rd of diastole - Friedrich's sign)
Absent y descent : CT, RCM] No diastolic filling.
Rapid y descent : TR
ASD with MR.
Slow y descent : Tricuspid stenosis
Right atrial myxoma.

Active space

h wave :

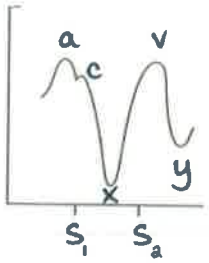
Small brief positive wave following y descent just prior to a wave.

Usually seen when diastole is long.

With increasing heart rate, y descent immediately followed by next a wave.

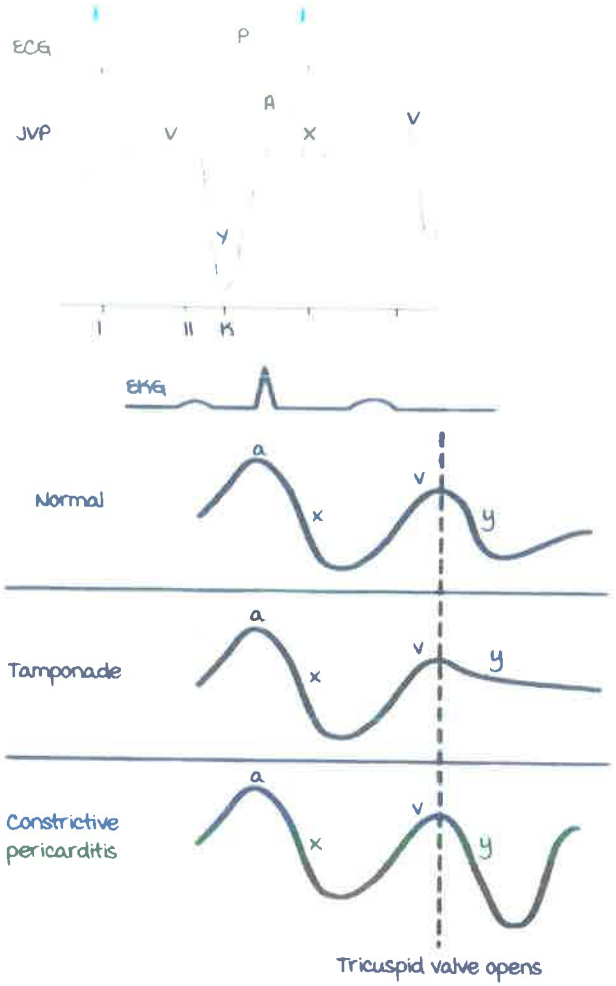
Constrictive pericarditis :

- JVP is elevated
- a wave is usually normal
- v wave is usually equal to a wave
- x descent : prominent
- y descent : rapid descent
- Kussmauls sign is usually positive



W/M pattern

On plotting the ventricular pressures : Square root sign



Active space

Leave Feedback

JVP in pulmonary hypertension :

Early RV decompensation :

- JVP maybe elevated.
- a wave is prominent.

Decompensated RVF :

- a and v wave prominent.
- v wave larger than a wave.
- x descent is diminished or absent.
- Rapid y descent due to TR.

Active space

EXERCISE AND ECG

Q : 39 year old male with chest pain,

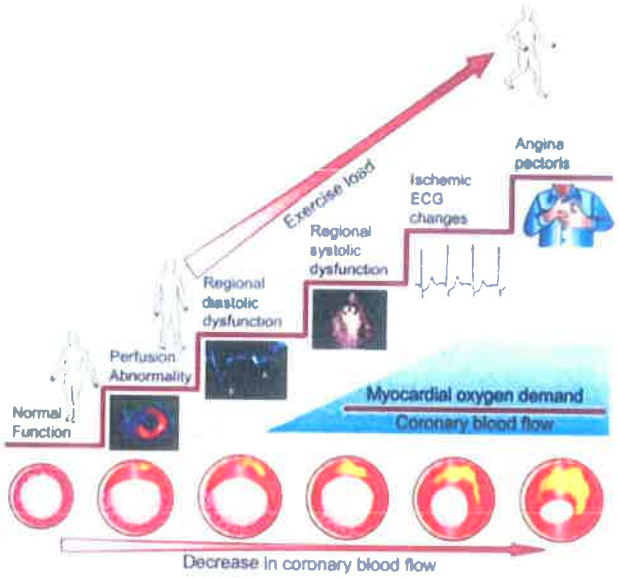
1 week prior TMT done as part of health insurance checkup- negative, ECG-echo was normal.

"Doctor told everything was normal. How can I develop an MI? "

- Is TMT the next step in chest pain evaluation after History, exam, ECG and ECHO ?
- What does TMT +ve or -ve mean?
- Does TMT negative status mean no CAD?
- Can a person develop an MI if he is TMT negative?
- Role of TMT in asymptomatic screening for CAD
  Eg: Health check up?

Ischemia cascade

00:01:38

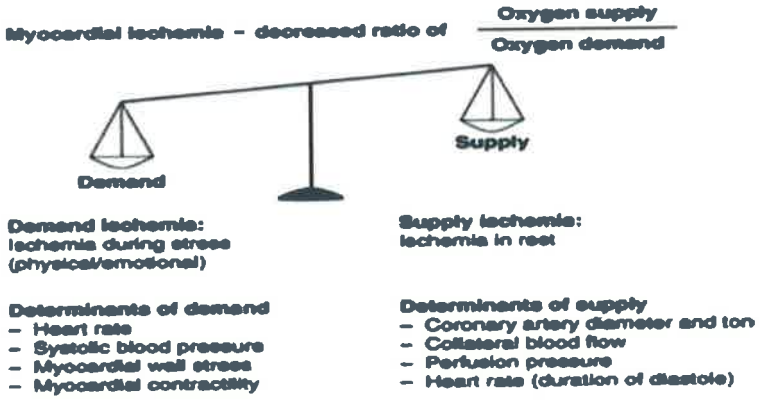


- Perfusion abnormality, it can be picked up doing SPECT
- Regional diastolic dysfunction and regional systolic

dysfunction by ECHO.

Leave Feedback

- Ischemic ECG changes by ECG.
- Chest pain.



Determinants of demand:

1. Heart rate
2. SBP = After load
3. myocardial wall sress = Preload
4. myocardial contractility = Ionotropy

Q. major determinant of MVO2 (myocardial oxygen demand) are all except (JIPMER 2020):

a) Ventricular wall tension

b) Hb – hemoglobin.

c) myocardial contractile state

d) HR

Parameters of demand

00:04:20

In Exercise Stress Test (EST):

- Increase demand and see if supply is proportionately increasing.

Active space

- Demand→ O2 consumption : Increased by Exercise.
- Supply→ measure coronary blood flow (invasive). So, Exercise induced symptoms and ECG changes.

Demand – 2 types :

- VO2 max : mets (metabolic equal Lents) .
- MVO2 max : Double product.

Supply :

- ECG changes.
- Exercise induced symptoms and signs.

What is demand ?

Exercise Test Terminology

Fick Equation

$Vo2max = (HRmax \times SVmax) \times (CaO2max - CvO2max)$

- Whole body O2 consumption-Vo2max——METs
- Myocardial Oxygen Consumption-Double product
- Coronary Flow x Coronary (a – v)O2 diff

Maximal Oxygen Consumption(VO2max)

- Greatest amount of oxygen an individual utilizes with maximal exercise (ml O2 /kg/ min)
- "Gold Standard" for cardiorespiratory fitness
- Fick Equation

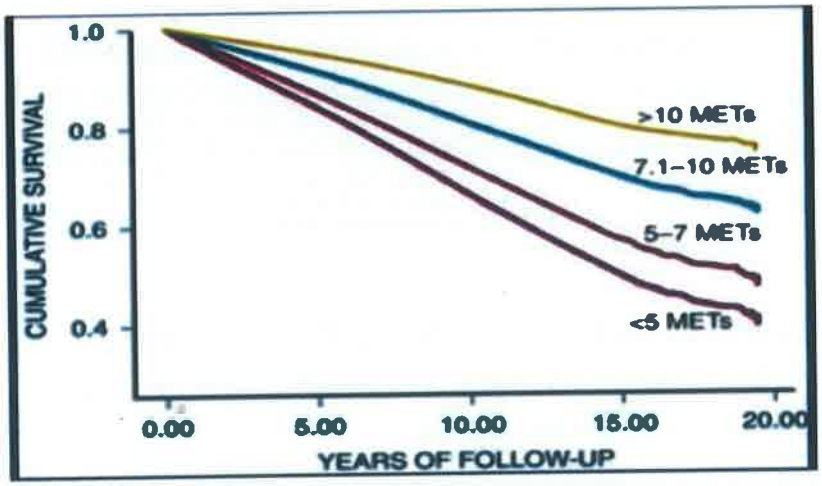
$Vo2max = (HRmax \times SVmax) \times (CaO2max - CvO2max)$

Active space

- 1 met : One metabolic equivalent (MET) is defined as the amount of oxygen consumed while sitting at rest and is equal to 3.5 ml O2 per kg body weight x min.
- Differs with Thyroid status, Disease state, Obesity, Muscle mass.

MET Values :

1 MET	Resting
2 METs	Level walking at 2 mph
4 METs	Level walking at 4 mph
<5 METs	Poor prognosis; peak cost of basic activities of daily living
10 METs	Prognosis with medical therapy as good as coronary artery bypass surgery; unlikely to exhibit significant nuclear perfusion defect
13 METs	Excellent prognosis regardless of other exercise responses
18 METs	Elite endurance athletes
20 METs	World-class athletes



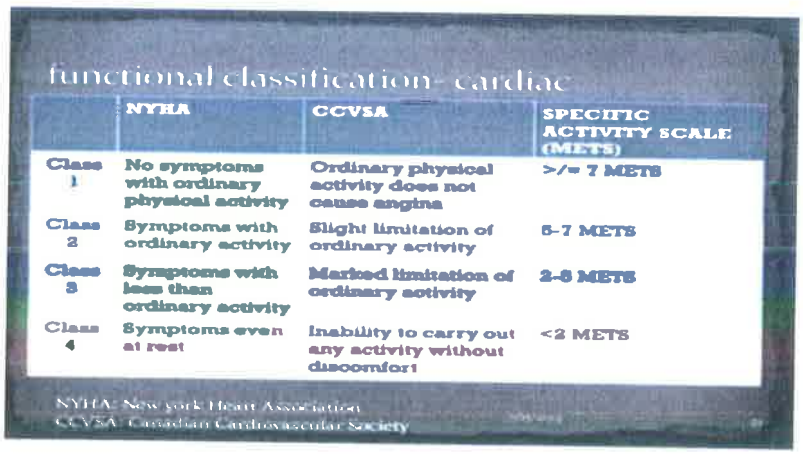
- Predicted mets (male) = 18 - (0.15 x Age)
- Predicted mets (female) = 14.7 - (0.13 x Age)

Active space

Age	Poor	Fair	Average	Good	High
Women					
< 29	<7.5	8-10	10-13	13-16	>16
30-39	< 7	7-9	9-11	11-15	>15
40-49	< 6	6-8	8-10	10-14	>14
50-59	< 5	5-7	7-9	9-13	>13
60-69	< 4.5	4.5-6	6-8	8-11.5	>11.5
70-79	< 3.5	3.5-4.5	4.5-6.5	6.5-8	> 8
>80	< 2.5	2.5-4	4-5.5	5.5-7	> 7
Men					
< 29	<8	8-11	11-14	14-17	>17
30-39	<7.5	7.5-10	10-12.5	12.5-16	>16
40-49	<7	7-8.5	8.5-11.5	11.5-15	>15
50-59	<6	6-8	8-11	11-14	>14
60-69	<5.5	5.5-7	7-9.5	9.5-13	>13

ENERGY REQ	ACTIVITY
2-4 METS	TAKING CARE OF SELF WALKING INDOORS WALK AT 2-3 mph
4 METS	LIGHT WORK AROUND THE HOUSE WALKING AT 3-4 mph
>4-<10 METS	CLIMB 1 FLIGHT OF STAIRS/UP HILL WALK>4 mph, SHORT RUNNING SCRUBBING FLOOR,MOVING FURNITURE
>10 METS	RUNNING> 6-7 mph HEAVY LABOUR SWIMMING,FOOTBALL

It is useful for giving Pre-Op fitness. If person is able to perform > 5 meters of physical activity, most of the surgeries can be uncomplicated.



Active space

Leave Feedback

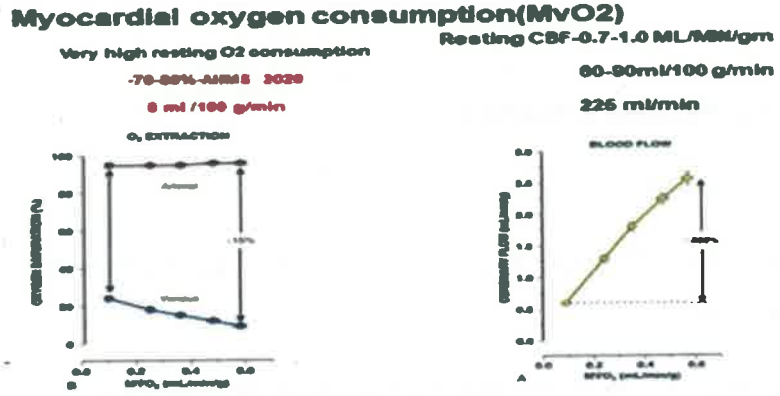
Calculation of METs on the Treadmill

$$METs = \frac{Speed \times [0.1 + (Grade \times 1.8)] + 3.5}{3.5}$$

Calcul automatically by device

Speed in meters/minute
= MPH x 26.8
Grade expressed as a fraction

Myocardial oxygen consumption(MvO2)



Myocardial oxygen consumption(MvO2)

Determinants of demand
- Heart rate
- Systolic blood pressure
- Myocardial wall stress
- Myocardial contractility

- 2 fold increase requires 50% increase in CBF.

Myocardial Oxygen Consumption

- Accurate measurement - cardiac catheterization
  - Coronary Flow x Coronary (a – v)O2 diff
- Indirectly measured as the "Double Product"
- "Double Product" = HR x SBP
  - A normal value is greater than 20,000 – 25,000
  - < 20,000 is low heart work load
  - > 29,000 indicates high heart work load
- Angina & ST↓ occur at the same DP for an individual

Demand=O2 consumption :

- VO2-mets
- MO2-Double product

Active space

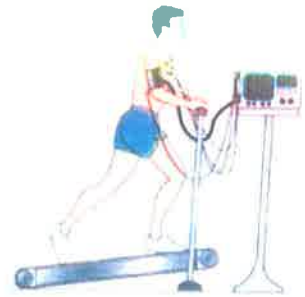
In EST – Exercise stress test :

- Increase demand and see if supply is proportionately increasing.
- Demand-O2 consumption -> Exercise
- Supply-measure blood flow -> ECG changes
- What type of exercise : Isotonic exercise

Increase demand = Exercise.

Types of exercise

- Isometric : If angle of joint does not change it is isometric exercise.
- Isotonic : If angle of joint changes then it is Isotonic exercise.



Comparison between isometric and isotonic exercises

00:04:20

Isometric exercise	Isotonic exercise
• HR-increases	• HR increases
• PVR increases	• PVR decreases
• SBP and DBP-Sharply increases	• SBP-moderate increase,DBP-unchanged or slight fall-PP increases
• CO-increases	• CO –increases
• SV unchanged(AIIMS 13)	• SV-Decreases
• 02 consumption increases	• 02 consumption increases

All increase in stress test except ? : JIPMER 2020

Leave Feedback

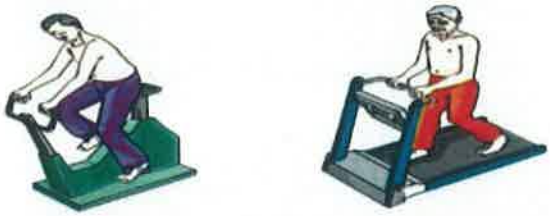
a) SBP

b) DBP

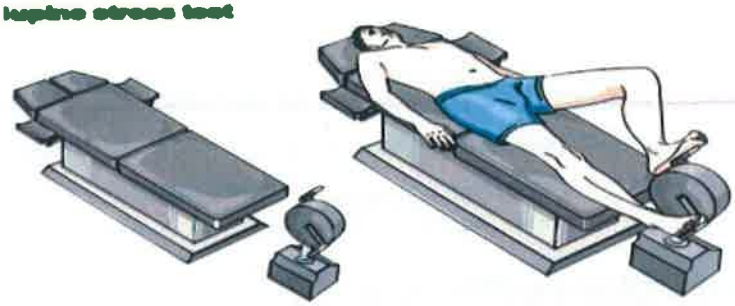
c) Pulse pressure

d) Double product

Types of EST



supine stress test



Parameters of supply

00:20:33

What do you look for in TMT

- ECG ST changes
- Arrythmias

- Functional capacity
- Exercise induced symptoms
- Exercise induced signs
- HR response
- BP response
- Double product
- Duke treadmill score

Active space

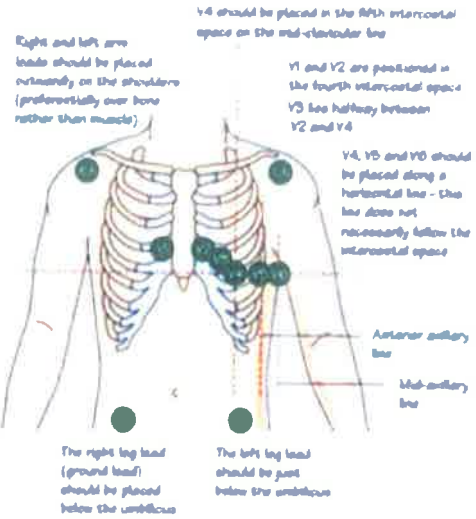
ST changes are to be considered in context of clinical data and non ST segment data.

E.g. If a patient has anaemia with Haemoglobin=6, as patient will have gross ST changes, TMT will be positive and in this context it doesn't mean patient has CAD. Once anemia is corrected TMT will be normal.

Normal ECG :

With Normal ECG testing method, due to presence of clamps, it is not possible for patient to run. So the following modifications are done to record ECG.

Mason Likar modification



- RAD
- ↑inf lead voltage
- Loss of inf lead q
- New Q in AVL

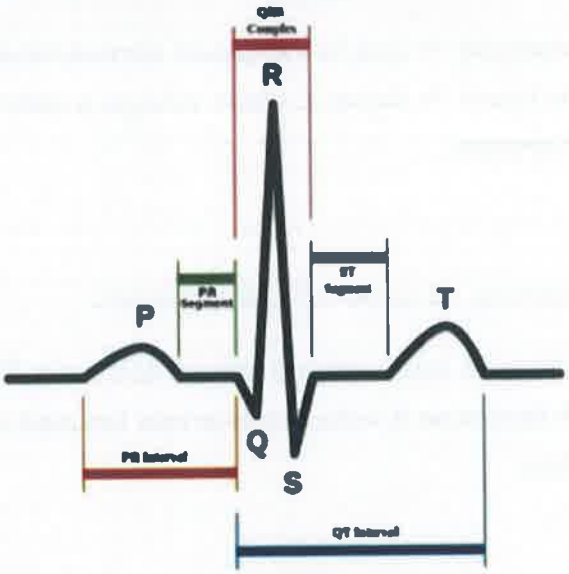
Active space

The ECG response

00:27:38

Leave Feedback

Exercise induced ECG changes



During Exercise, shortening of intervals is seen. i.e.,

1. PR interval, Interval, ST segment and QT interval shortens.
2. P wave remains unchanged.
3. R wave increases in amplitude, this effect is called Brody effect.
4. T wave increases in amplitude: due to shortening of QT. (similarly like in hyperkalaemia, QT shortens and this causes tall T waves).

All are true in exercise except – SCT19 :

a) PR shorten.

b) QT shorten.

c) R amplitude increases.

d) QRS duration shorten.

Brody effect :

Blood is good conductor of electricity. In AR, cor bovium is seen (cow like heart). So due to presence of so much blood

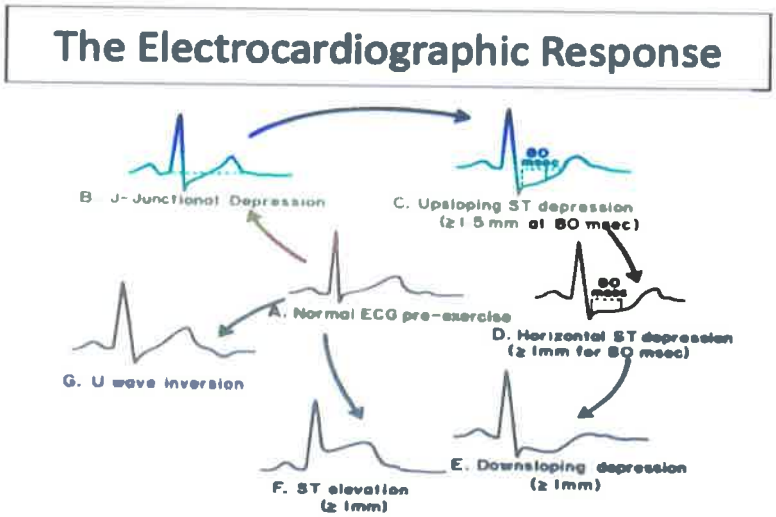
Active space

in heart higher R wave voltage.

Similarly in exercise → Due to increased venous return more blood in heart → Higher R wave voltage is seen. This is normal phenomenon.

- During exercise : R wave voltage increases.
- Post exercise : R wave voltage should decrease. If it doesn't decrease it indicates exercise induced LV dysfunction.

ECG changes – ST segment changes :



1. Exercise induced ST depression:

- Fast up sloping : Normal.
- Slow up sloping : Abnormal.
- Horizontal : Abnormal.
- Down sloping : Abnormal.

2. Exercise induced ST elevation : always abnormal.

- Leads with q wave.
- Leads without q wave.

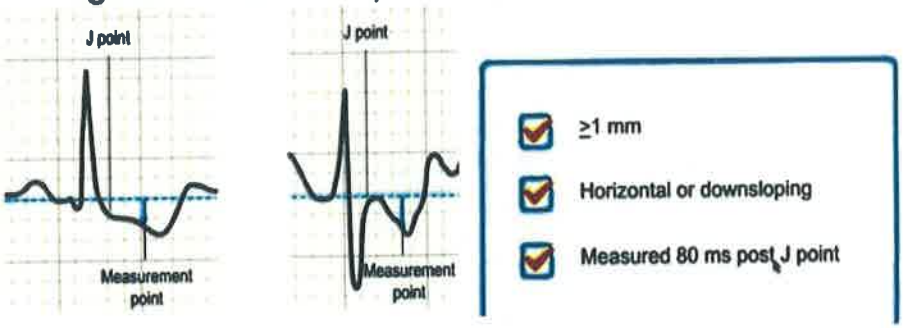
Leave Feedback

3. Exercise induced U wave inversion.

4. Exercise induced ST Elevation in aVR.

ST Depression -> no localizing value

- PQ junction-isoelectric point

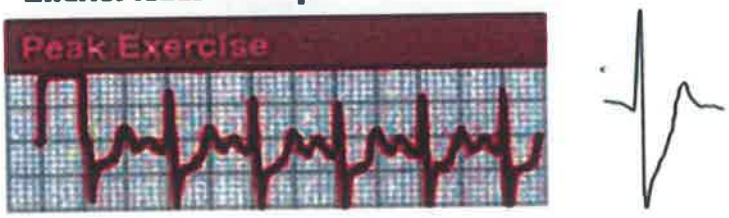


- ST depression and T inversions are manifestations of ischemia and they do not localize on ECG.

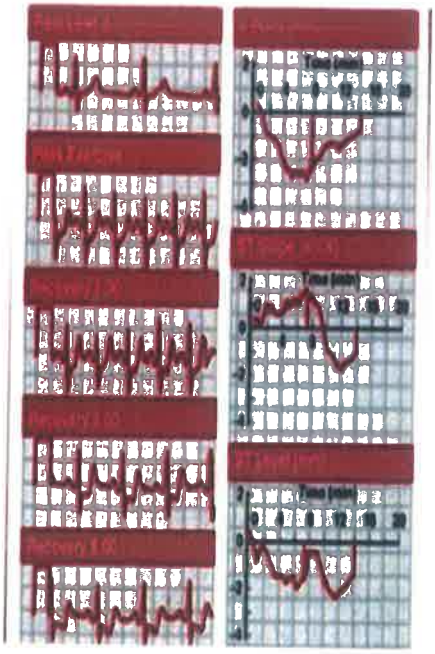
  ST elevation is a manifestation of injury on ECG and it localizes.

- Q wave is manifestation of infarct on ECG and is localized.

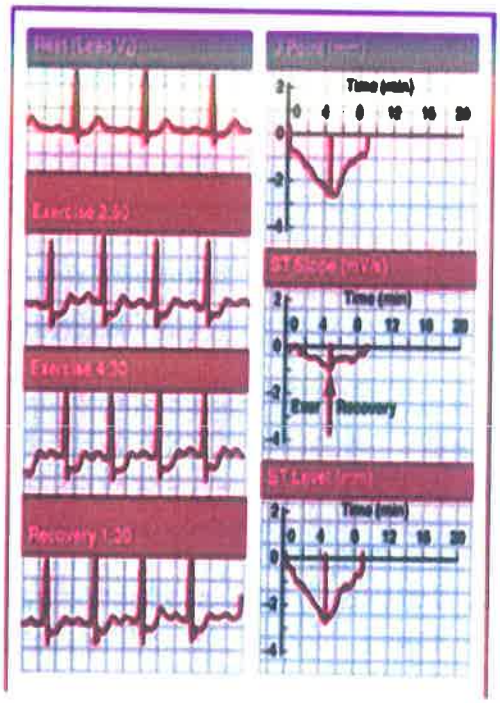
Lateral leads-most spc
Inferior leads-least spc



Active space



- Recovery equally important
  - ST changes only in recovery
  - ST changes take a long time to normalise



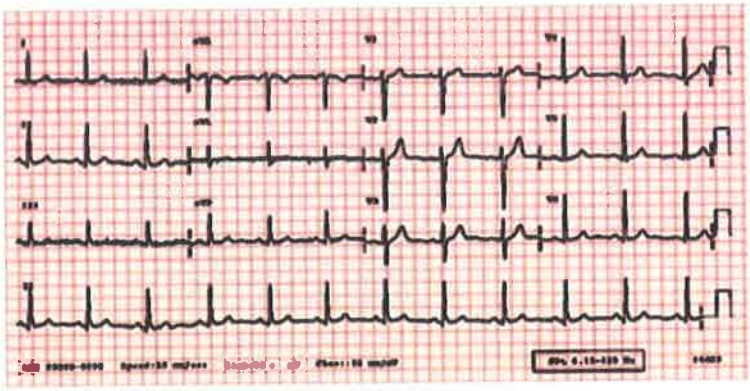
- Lateral leads-most spc
- Inferior leads-least spc

- ST changes taking a long time to normalise indicates a bad prognosis.

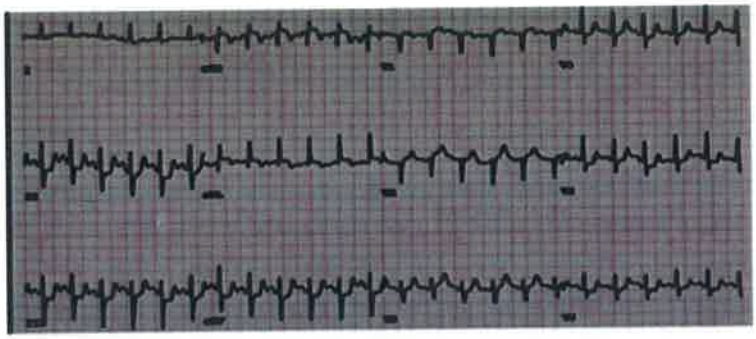
Active space

Leave Feedback

Q. 55 year old DM/SHT-chest pain not consistently related to exertion 3 months, with left shoulder pain.
ECHO-Normal



TMT-4 mets-reproducible chest pain.



This is suggestive of severe CAD.

ST elevation in TMT rare.

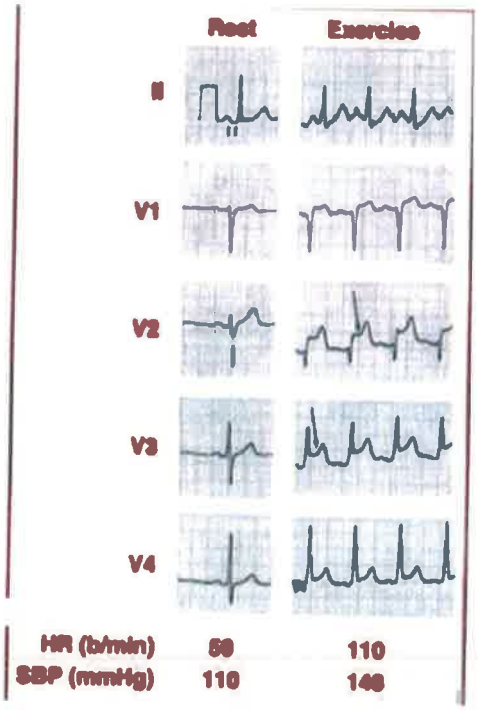
Leads with q wave :

- Peri-infarctonal ischemia.
- Dyskinetic.
- Aneurysmal segment.
- No localizing value.

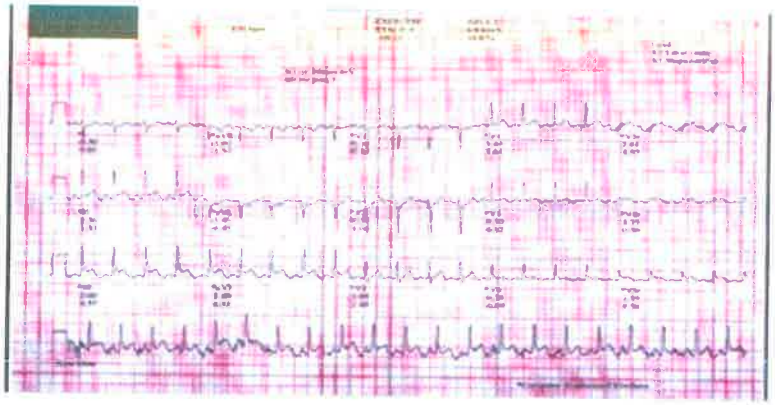
Leads without q wave

- Critical lesion.
- Coronary vasospasm.
- Localizing value.

Active space



No Q wave. This indicates either a critical LAD disease or LAD vasospasm.



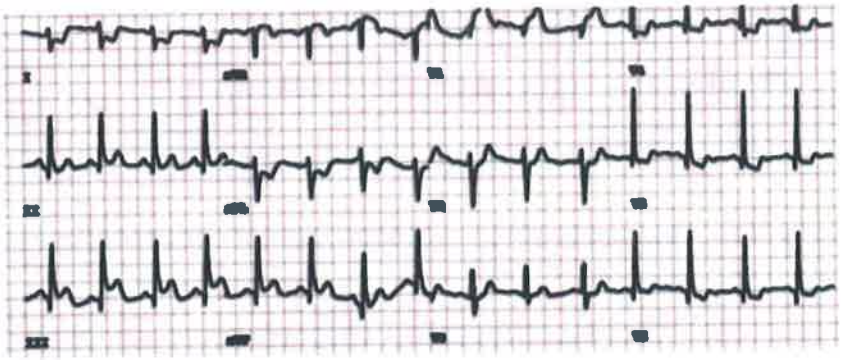
ST elevation in inferior leads and No Q wave critical RCA disease or RCA vasospasm.

When do you take him for CAG

While exercising patient develops ST elevation during

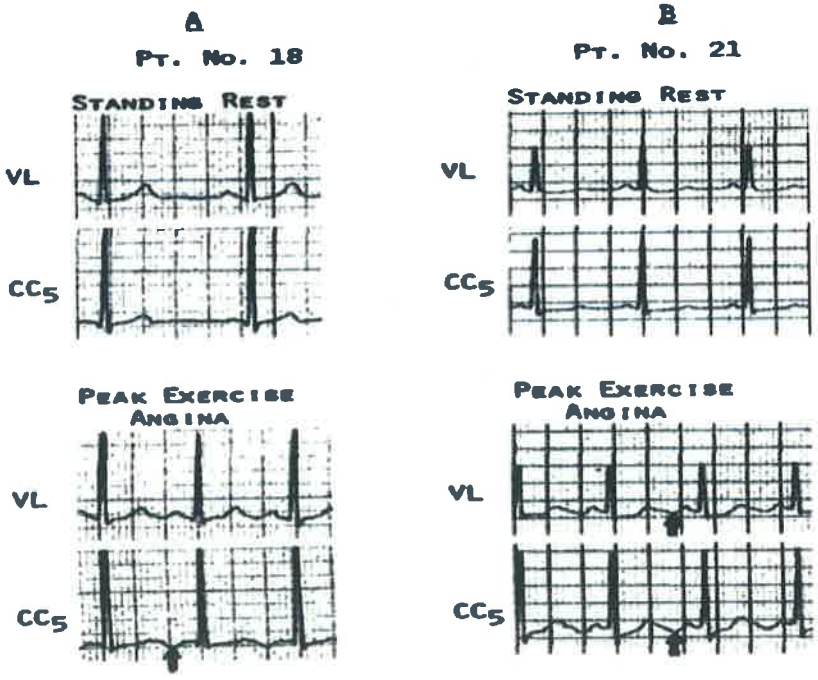
recovery ST elevation settle. CAG can be done in 3 or 4 days (it is an urgent CAG)

If ST is elevated continuously → emergency CAG should be done.



ST elevation in inferior leads with Q waves : Indicates dyskinetic aneurysmal segment.

Exercise induced u wave inversion :



- A patient with chest pain with U wave inversion – This indicates significant CAD, usually it involves LAD. (V1 to V6).
- If a patient develops new onset U wave inversion after

Active space

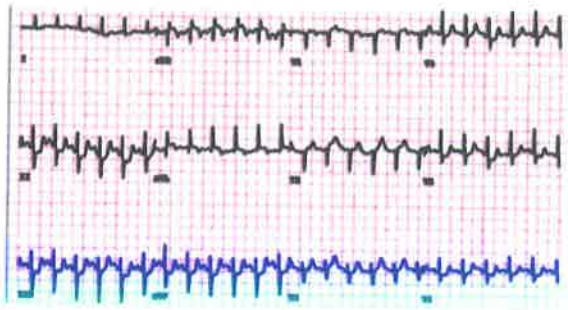
exercise : indicates significant CAD. It has localizing value,

V1 – V6 : LAD involved (anterior wall)

Lead 2, 3, aVF : RCA or circumflex.

No ST elevation in aVR : Rules out LMCA / triple vesseld disease (high negative predictive value).

- aVR ST elevation in isolation → 49% specificity.
- aVR ST elevation with V1 ST elevation → 83% specificity.
- aVR ST elevation with V5 ST depression → 85% specificity.



ST elevation lead aVR.

Preexisting ST depression

Can do :

- LVH with strain.
- RBBB.
- Hypokalemia with ST-T abnormalities.

Cannot do for diagnosis of CAD – Class 3 indications for TMT

- Greater than 1 mm ST depression at rest.
- Digitalis.
- LBBB.
- Ventricular preexcitation.
- Ventricular paced rhythm.