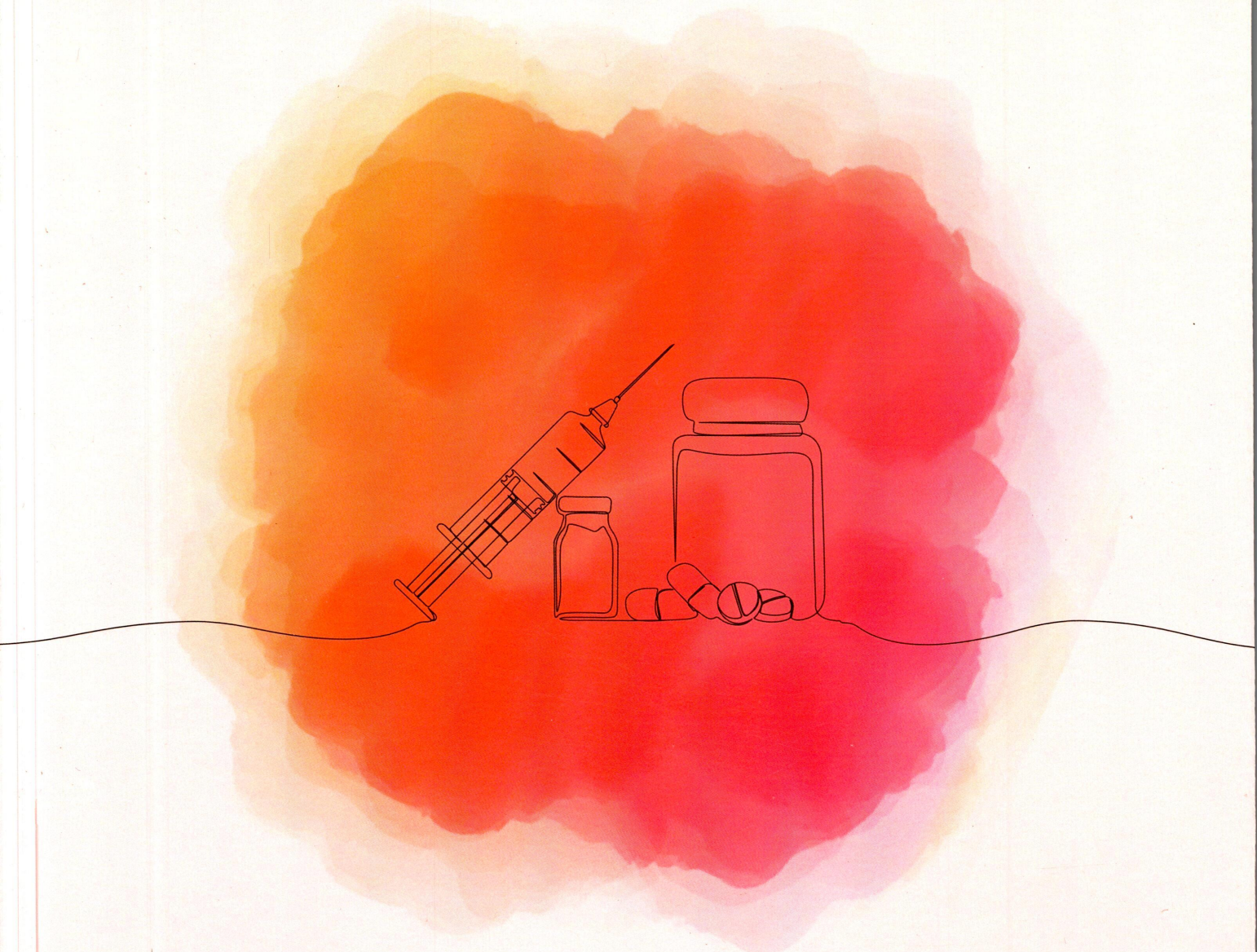
VOLUME-IV

PrepLadder



MEDICINE

NEET SS VERSION 4.0

PREFACE

Dear Dreamer,

Pat your back and reward yourself, today you've taken an important step towards reaching your dream. Mark Twain said, "the secret of getting ahead is getting started". You have started in the right direction.

The next few months until your targeted exam or exams are crucial. Every moment counts and each step you take now, will help you reach your goal, your dream.

PrepLadder is your partner in this journey. We are in this together. So, don't worry, do not stress. Just focus on your preparation and stay positive. Set daily targets, achievable and realistic ones. If you pass the target, celebrate. However, if you miss a daily target, forgive yourself, move on and do better the next day.

Always remember that you are special and unique. Do not compare your preparation, your speed, your abilities with others. Believe us, it doesn't help.

Practice a lot. Solve the QBank. Take Grand Tests. Review your performance. But don't let ranks and results drag you down. Rather use them as a guiding light to know your strengths and weaknesses, to increase your speed and accuracy, and to get yourself ready for the exam day.

If you have a doubt or a question, ask us. We are here.

Lastly and most importantly. take care of yourself. You and your health are precious. So, keep up with exercise, eat healthy, get enough sleep and care for yourself.

We're behind you, cheering you on to the finish line. You might be feeling stressed, overwhelmed, and so tired, but you're nearly there. Give it everything you've got, and know that whatever happens next, you've got what it takes for the life you want. You are a Champion.

Own Your Dream
Team PrepLadder

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CARDIOVASCULAR

1 JUGULAR VENOUS PRESSURE (JVP)

Introduction

00:01:27

- JVP is used to assess the volume status of the individual
  - To decide the need for diuretic therapy
    - → In CHF, an increase in JVP indicates to increase the dose of diuretic therapy
    - → Raised JVP secondary to RV infarction; diuretics are not given, as it may cause Hypotension
- JVP also indicates RA pressures
  - Pulsation delay is present between RA pressure changes and JVP waveforms.
    - → 60-110 msec
- JVP is not seen in normal individuals

History

00:04:15

- The jugular venous waveform was first described by Potain
- James Mckenzie
  - Named the JVP waves
  - Described the importance of JVP in clinical examination

Difference between Carotid Pulsations and JVP

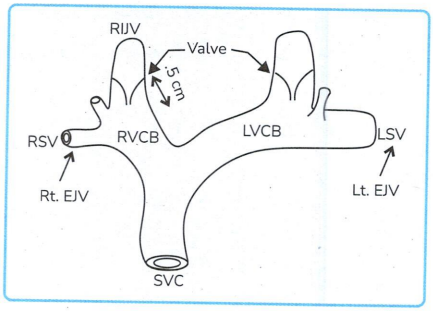
00:05:54

	Carotid pulsations	JVP
Relation with Sternocleidomastoid	Deep and medial	Superficial and lateral
Obliterate ability	Not obliterable	Obliterable
Varying with position & respiration	No variation	Increase on inspiration
Abdominal compression	No variation	Increase in compression at right hypochondrium
Pulsatile	Felt - pulsatile	Not felt
Waves	Felt	Seen
Peaks & Throughs	Has single upstroke	2 peaks and 2 throughs
Descend	Rapid	Slow

Anatomy of Jugular Vein

00:10:57

- There are two sets of jugular veins on either side
  - External jugular vein (EJV)
  - Internal jugular vein (IJV)
- Both the EJV drain into the respective subclavian veins
- The subclavian veins join with the respective IJV to form the Brachiocephalic vein
  - Brachiocephalic vein is also called innominate vein
- Right and left brachiocephalic veins together drain into the superior vena cava (SVC)
  - Then to the right atrium



Why choose the IJV over the EJV to assess RA pressure changes?

- The IJV is in direct line with the SVC
  - EJV does not have direct continuity with Right atrium
    - → It takes 2 bends of 90° → causes pressure changes
- The course of EJV to the SVC
  - Passes through several facial planes

- Thrombus formation in the bulb of the EJV
  - Can cause partial obstruction → rise in pressure in EJV
- On lateral movement of the head, there can be contraction of platysma muscle
  - Can cause partial obstruction of EJV
- Presence of valves in EJV
  - The pulsations are not seen due to the valves
- In cardiogenic shock and congestive cardiac failure
  - Vasoconstriction of EJV
    → Due to excessive activation of the sympathetic nervous system
  - EJV will be small and barely visible

Why the right IJV over the left IJV?

- Right IJV is in a straight line with the SVC
  - It reflects the hemodynamic changes of RA pressure changes
  - Left IJV has angulations
- Right innominate vein
  - There is greater filling of the right innominate vein from the right side of head
- Left innominate vein
  - Prone for kinking / compression between the aortic arch and sternum

Interpretation of the Jugular Venous Pulse

00:19:50

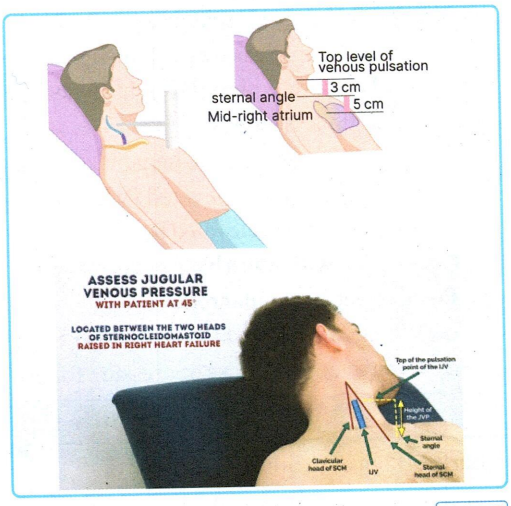
- Interpretation of JVP is done under the following:
  - Jugular venous pressure
  - Respiratory variation
  - Hepatojugular reflux/ abdominal compression test
  - Waveforms

Positioning the Patient

- The patient should be in a semi-reclining position
  - 45° between the trunk and bed
- Head should be slightly rotated towards the left

Why 45°inclination?

- In a healthy person,
  - In sitting posture - the jugulars are collapsed
  - In supine posture - the jugulars are distended to variable degree
- The intermediate position is 30°-60°.
  - Standard - 45°
- Sometimes, pulsations are going up to the jaw or even to the earlobes
  - Indicates RA pressure ≥25mmHg.



00:24:20

Measurement

- Two scales are required
  - One scale is placed horizontally to the upper level of pulsation
  - The second scale is placed perpendicularly to the first scale over the sternal angle - vertically
- The vertical distance from bifurcation of the scales to the sternal angle → JVP
  - Add 5cms to the value
    → Because the distance from the sternal angle to the center of RA is 5cms.
- In a normal individual
  - JVP is 6-8 cm of H_2O at 45°

Respiratory Variation

00:26:26

- During inspiration
  - Venous return increases → RA pressure increases → JVP increase
  - Mean JVP decreases

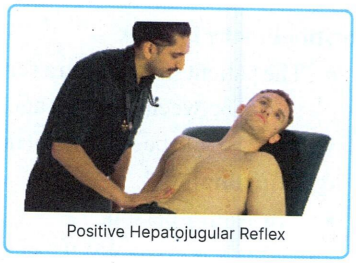
Kussumal Sign

- Mean JVP increases on inspiration
- Pulsatile increase in JVP
  - Seen in:
    - → Constrictive pericarditis
    - → Right heart failure
    - → TR
    - → Pulmonary stenosis
    - → Pulmonary hypertension
    - → Cor pulmonale
    - → Constrictive pericarditis
    - → Restrictive cardiomyopathy
- In right heart failure
  - Increase in RV end-systolic volume
  - Blood from RA is added during diastole → Increase in RV end-diastolic pressure
  - During inspiration, the venous return in RA increases
    - → Blood cannot flow to RV due to increased RV EDP
    - → RA pressure increases → SVC & IVC pressure increases → elevated JVP
- Kussumal's sign is not seen despite elevated JVP in: (non-pulsatile increase in JVP)
  - Cardiac tamponade
  - SVC obstruction

Hepatojugular Reflex

00:34:28

- Normal / Negative Hepatojugular Reflex
  - On applying pressure over the right hypochondrium
    - → Pressure within the right hypochondrium has to build up over 20-35mmHg within 15 seconds
    - The venous return to hepatic veins increases → VR to IVC increases → elevated RA pressure → elevated JVP
    - → JVP will be elevated by < 3mmHg
    - → JVP will return back to normal within 10 seconds
- Positive hepatojugular reflex
  - When JVP is elevated > 3cms of H_2O for the entire compression
  - Even after the release of pressure for > 15 secs.



Positive Hepatojugular Reflex

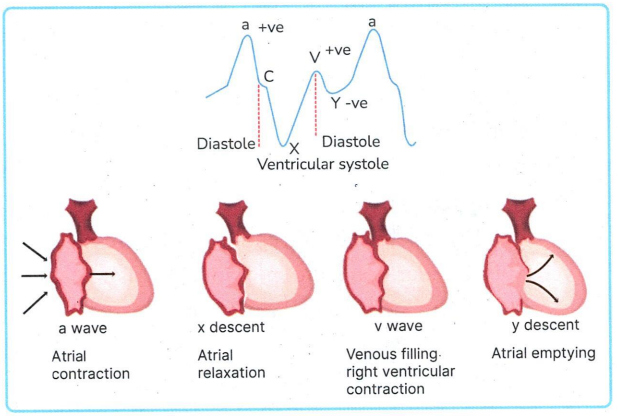
True Positive HJR	False Positive HJR
• Earliest sign of Incipient(latent) RHF ○ TR ○ Pulmonary hypertension ○ Pulmonary stenosis ○ Pulmonary embolism	• COPD • Lung conditions with loss of vital capacity • In individuals with excessive sympathetic stimulation • Extreme nervousness

Identification of Waves

00:39:28

- The waves are:
  - a, c, x, v, y and occasionally H
  - Positive waves - a, v
    - → Increase in right atrial pressure

- Negative waves - x, y
  - → Decrease in right atrial pressure
- When RV contracts, the pressure within the ventricles increases
  - The tricuspid valve leaflets are pushed into RA
    - → RA pressure increases → c wave
    - → C wave is present on the descent of the x wave
- x wave is due to atrial relaxation
- x' is due to the descent of the tricuspid valve ring and cusps
- H wave is present before a wave
  - Pre-systolic (atrial) wave
  - Seen in bradycardia
  - Occurs due to passive right heart filling during diastole



Events in Cardiac Cycle

00:49:16

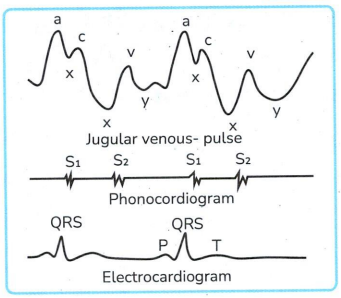
- Systole
  - Isovolumetric contraction
  - 1st rapid ejection
  - Slow ejection
- Diastole
  - Protodiastole / Isovolumetric relaxation
  - 1st rapid filling
    - → Due to the opening of the tricuspid and mitral valve
  - Diastasis - passive filling
  - 2nd rapid filling
    - → Due to atrial contraction

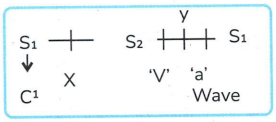
Wave Form	Phase of Cardiac Cycle	Mechanical Event
A wave	End diastole	Atrial systole Before S1
C wave	Early systole	Cusps - bulging → RA
X wave	Mid systole	Atrial relaxation
V wave	Late systole	Venous filling↑
Y wave	Early diastole	Atrial emptying

Heart Sounds & Cardiac Cycle Events

00:41:00

- S_1 corresponds to c wave
  - Isovolumetric contraction
  - Rapid upstroke
- Just before S_1 is a wave
- The peak seen after S_2 is v wave
- x wave is present between S_1 and S_2
  - Atrial relaxation
  - Mid-systole
- y wave is present between S_2 and S_1
  - Downstroke
- Palpate the opposite carotid artery simultaneously or auscultate over the pericardium
  - To make out the events of the cardiac cycle





Abnormal Waveforms

00:56:58

- Tall 'a' wave
  - Giant 'a' wave: 6-15 mmHg
  - Cannon 'a' wave: ≥16 mmHg
- Absent 'a' wave

Etiology of Giant 'a' Wave

00:57:30

- Tricuspid valve level
  - TS
    - → Due to elevated RA pressure
  - RA myxoma
    - → Will obstruct RV inflow tract

- Tricuspid atresia
- Thrombus in RA
- Lupus endocarditis
- Carcinoid syndrome

- Interventricular septum level
  - RV hypertrophy
    → Due to elevated RV end-diastolic pressure (RV EDP)
  - Sub valvular stenosis of pulmonary valve
    → Due to increase in RV pressure
- Pulmonary valve level
  - Pulmonary stenosis
    → Due to increase in RV systolic and end-diastolic pressure
  - Pulmonary hypertension
  - Pulmonary embolism
  - Pulmonary regurgitation

Jugular Corrigans 01:02:19

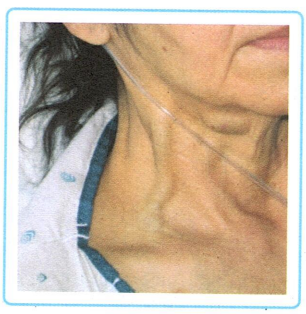
- If the amplitude of 'a' wave is 6-15cms higher than v wave

Cannon 'a' Waves 01:03:05

- Cannon waves offer diagnostic help in arrhythmias
- RA pressure ≥16mmHg
- When RA & RV contracts simultaneously (as in complete heart block)
  - Tricuspid valve closes due to contraction of RV
  - RA has to contract against a closed tricuspid valve
    → Elevated RA pressure

Irregular Cannon 'a' wave	Regular cannon 'a' wave
• Complete heart block • Ventricular ectopic beats	• Atrioventricular nodal reentrant tachycardia • Atrioventricular reentrant tachycardia • 2:1 AV block • Slow VT

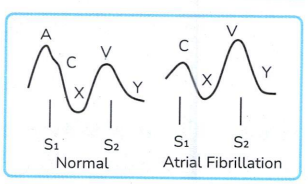
Frog Positive Sign



- Pronounced efflux of blood into jugular veins during simultaneous RA and RV contraction→ Prominent neck pulsations (Frog's neck)
  - Seen in cannon 'a' wave

Absent 'a' Wave 01:08:41

- The RA pressure does not increase with RA contraction
- Seen in:
  - Atrial fibrillation
    → Due to the feeble contraction of RA
    → Rate of RA contraction: 300-400/min
  - Ebstein's anomaly
    → Atrialization of RV → Giant silent RA
    → RA does not have effective mechanical systole

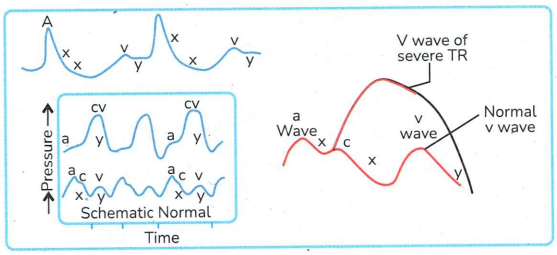


'x' Descent Abnormalities

01:11:27

- Attenuated/ Absent 'x'
  - Tricuspid regurgitation
- Increased depth of the 'x' descent - due to increase in RV EDP
  - Restrictive cardiomyopathy
  - Cardiac tamponade
  - Constrictive pericarditis

Attenuated / Absent 'x'

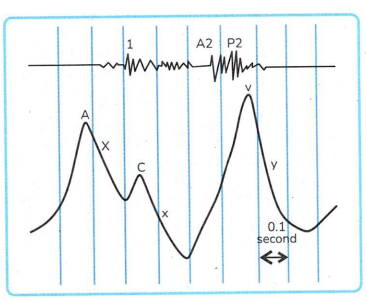


- When it is encroached upon by increasing degrees of TR
- In TR
  - Some blood will enter RA during RA relaxation
  - No fall in pressure of RA → absent 'x'

'v' Wave Abnormalities

01:15:01

- Increased 'v' wave amplitude
  - TR
    - → V wave is due to venous return
    - → In TR, RA receives blood from SVC, IVC, and blood through TR
    - → Increased v wave amplitude → Lancisi sign
- A large 'v' wave due to increased RA filling:
  - ASD
    - → Due to blood from LA → RA
    - → Also has deeper 'x' descent
    - → JVP in ASD: Prominent a, v, x and y
  - TAPVC
    - → Due to blood from pulmonary veins
  - Hyperdynamic circulatory states
  - Absent pericardium



'y' Descent Abnormalities

01:18:06

- Slow descent
  - Tricuspid stenosis
    - → Due to slow and delayed RA emptying
- Rapid & deep 'y' descent
  - Constrictive pericarditis
  - Restrictive cardiomyopathy
- Absent 'y' wave
  - Cardiac tamponade
    - → Due to rapidly abrupt elevation of pericardial pressure
    - → There is an increase in ventricular end-diastolic pressure
    - → Diastolic collapse of ventricles → Atria cannot empty the blood
- Prominent 'y' descent
  - Tricuspid regurgitation
    - → Due to the large volume of blood in RA

Friedrich's Sign

01:21:23

- JVP sign in constrictive pericarditis
  - Rapid and sharp 'y' decent without prominent 'v' wave
    → Rapid 'y' descent is due to diastolic dysfunction

Important Information

- JVP in TR
  - Absent 'x' wave
  - Prominent 'v' wave
  - Prominent 'y' descent
  - cv wave
    → c wave gets merged with v wave
- JVP in TS
  - Slow 'y' descent

Specifics

01:23:42

- Atrial septal defect
  - Prominent 'a' wave
  - Deep 'x' wave
  - Large steep 'v' wave
  - Deep 'y' descent
- Pericardial disease
  - In cardiac tamponade
    → Deep 'x' descent
    → Absent 'y' wave
  - In constrictive pericarditis
    → Prominent 'x' and 'y' descent
  - In effusive constrictive pericarditis (with pericardial effusion)
    → Dominant 'x' descent
    → Equal 'y' descent

May's Sign

- Engorged veins on the undersurface of the tongue
- Indicates elevated venous pressure



Clinical Scenario

01:27:00

Q. A 67-year-old man presents to accidental and emergency with a 3-day history of shortness of breath. On examination, you palpate the radial pulse and notice that the patient has an irregular heartbeat with an overall rate of 140 bpm. You request an electrocardiogram (ECG), which reveals that the patient is in atrial fibrillation. Which of the following would you expect to see when assessing the JVP?

A. Raised JVP with normal waveform
B. Large 'v' waves
C. Cannon 'a' waves
D. Absent 'a' waves
E. Large 'a' waves

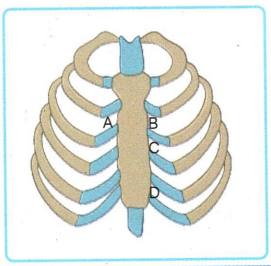
2 HEART SOUNDS

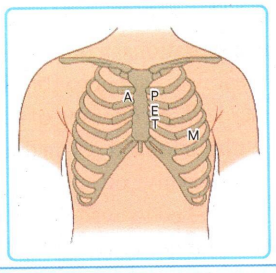
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Areas of Auscultation

Q. Where is the location of Erb's point during auscultation?

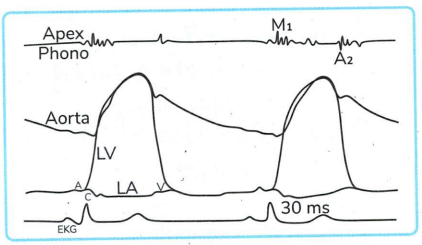
A. A = 2nd intercostal space right parasternal line
B. B = 2nd intercostal space left parasternal line
C. C = 3rd intercostal space left parasternal line
D. D = 5th intercostal space left parasternal line





Aortic area (A)	• 2nd intercostal space right parasternal line • Murmur of aortic stenosis/regurgitation is best heard here.
Pulmonary area (P)	• 2nd intercostal space left parasternal line • In pulmonary hypertension, loud P2 is heard in this area
Erb's point (E)	• 3rd intercostal space left parasternal line • 2nd heart sound is best heard in this area
Tricuspid area (T)	• 4th intercostal space left parasternal line • Murmur of tricuspid stenosis/regurgitation is best heard here.
Mitral area (M)	• Left 5th intercostal space in midclavicular line / ½ inch medial to midclavicular line. • Corresponds to the apex of heart • Murmur of mitral stenosis/regurgitation is best heard here.

First heart sound



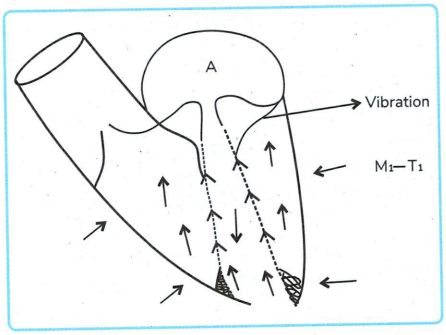
Q. The first heart sound signals the onset of left ventricular contraction and consists of?

A. Two major audible components (M1 and T1) and two inaudible components
B. Two major audible components (M1 and T1) and one inaudible component
C. One major audible component (M1 and T1) and two inaudible components
D. One major audible component (M1 and T1) and one inaudible component

- The first heart sound is heard in the Isovolumetric contraction phase of the cardiac cycle.
- All 4 heart sounds can be heard in phonocardiogram
- Only S1 and S2 can be heard in the stethoscope.
- The first heart sound has:
  - M1 - Mitral component
    → Coincides with the c wave on the left atrial pressure graph
  - T1 - Tricuspid component

00:06:04

Mechanism of first heart sound



- Isovolumetric contraction leads to increased pressure in ventricles
- This will push the AV valves into the atria
- Chordae Tendineae will also be pulled up along with AV valves
- Leads to stretching of papillary muscle
- After getting stretched to the maximum, the papillary muscle pulls the Chordae tendineae down along with AV valves.
- This leads to the vibration of the entire AV valve
  - In Mitral valve - M1
  - In Tricuspid valve - T1

Components of first heart sound

00:08:15

- The first inaudible low-frequency vibrations coincide with:
  - Beginning of left ventricular contractions on the left side
  - Beginning of right ventricular contractions on the right side.
- Audible M1 and T1 components:
  - Due to vibrations in mitral and tricuspid valves
- Inaudible low-frequency vibrations:
  - Coincides with opening of semilunar valves
    - → Pulmonary valves on the right side
    - → Aortic valve on left side

Q. An intern was asked to auscultate the first heart sound & he said he can't differentiate M1 & T1 separately. He cannot differentiate because the distance between M1 & T1 is?

A. > 30 msec

B. 20-30 msec

C. < 20 msec

D. 40-50 msec

- The human ear can differentiate two sounds when the gap between them is > 30 msec.
- The duration between M1 & T1 is 20-30 msec.

Characteristics of S1

00:11:25

Determinants of intensity of first heart sound

- Structural integrity of the AV valve
  - Thickness and mobility of leaflets
- Velocity of the valve closure
  - Depends on the position of AV valves
    - → If wide apart - high intensity
    - → If close to each other - low intensity
- Status of ventricular contraction
  - If the rate of pressure rise in the ventricle is less → low intensity
- Physical characteristics of the vibrating structures
  - Chordae tendineae
  - Papillary muscles
  - AV valve leaflets
- Transmission characteristics
  - Distance between AV valves and chest wall

Character	Findings
Frequency	Medium to High
Duration	0.14 sec
M1-T1 interval	20-30 msec
C-point of MV echocardiogram	Coincides with M1
Downstroke of C wave of LA pressure	Coincides with M1
Downstroke of C wave of RA pressure	Coincides with T1

Abnormalities of first heart sound

00:15:25

Causes of Soft S1

Poor Conduction	• Obesity • Emphysema • Left-sided pleural effusion • Pericardial effusions
Decreased LV contraction	• Myxoedema • Cardiomyopathy • Acute MI • Mitral regurgitation → LVF
PR interval (normal: 120 – 200 msec)	• Prolonged PR interval (200-500 msec) ○ 1st degree AV block → Due to lesser separation of mitral valve leaflets
Mobility of the valve	• Severe calcification ○ Leads to calcified mitral stenosis → reduced mobility

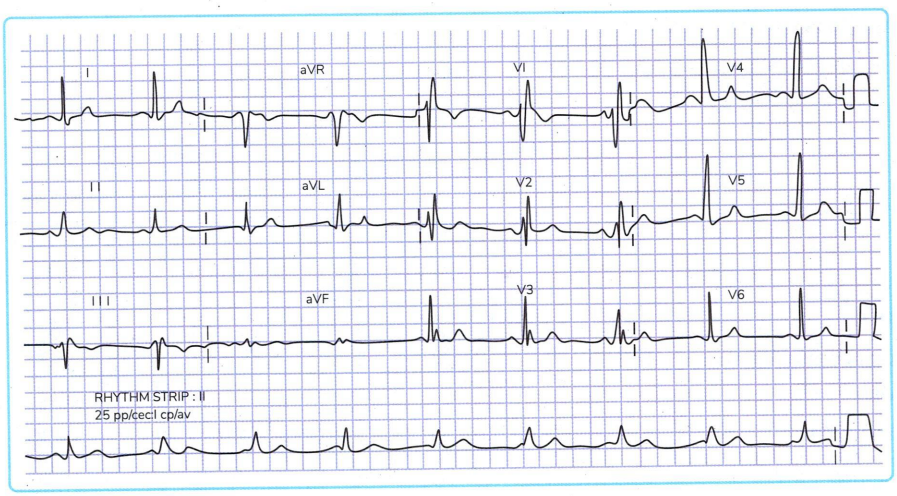
Causes of Loud S1

Tachycardia	• Anemia • Thyrotoxicosis • Beri-Beri • Fever • Pregnancy • AV fistula • Paget's disease
Prolonged AV flow	• Mitral stenosis • Tricuspid stenosis

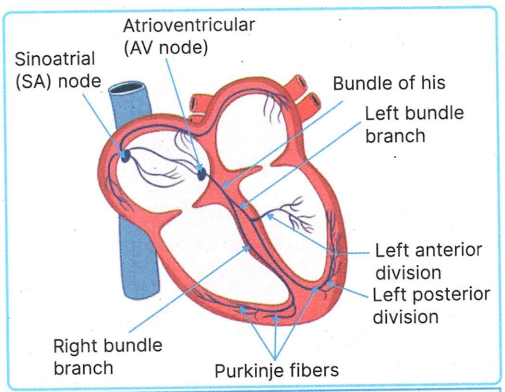
PR interval	• Short PR interval (< 120 msec)
	  ○ WPW (Wolff-Parkinson-White syndrome)
	  ○ LGL (Lown–Ganong–Levine syndrome)

Pathological Split S1

Q. A 27-year-old male patient presented with a cardiac history of ASD. ECG of the patient is as follows. What will be the abnormality in the first heart sound?



A. Reverse splitting of first heart sound
B. Pathological splitting of first heart sound
C. Soft S1
D. Loud S1



- RSR' pattern is observed in V1
  - Suggestive of Right Bundle Branch Block (RBBB)
  - Delayed T1
    - → The pressure rise in LV is more as compared to RV
    - → Thus, M1 is followed by T1
    - → Duration between M1 & T1: > 30msec

Normal splitting: M1 → T1 (> 30msec)	Reverse splitting: T1 → M1

• RBBB
• LV ectopic & paced beats
  ○ LV contraction occurs first, followed by RV contraction
• Ebstein's anomaly
  ○ Atrialization of RV
  ○ RV cavity is reduced and hypoplastic
	• LBBB
	• RV ectopic & paced beats
	  ○ RV contraction occurs first, followed by LV contraction
	• Severe mitral stenosis
	• Atrial myxoma

Important Information

- In normal individuals
  - LV pressure rise > RV pressure rise
- In very severe mitral stenosis
  - Mitral valve area (MVA) - <1 cm^2
  - LA → LV flow is grossly reduced
  - Leading to decreased LV pressure rise
  - RV pressure rise > LV pressure rise
  - T1 is followed by M1 → Reverse splitting
- Atrial myxoma
  - Tumor arising from interatrial septum
    → Will overlap the mitral valve annulus → obstructs LV inflow
  - RV pressure rise > LV pressure rise
    → Reverse splitting

Q. A 45-year-old female presented with syncopal episodes, angina, and dyspnea. Her ECG is as follows. What is the abnormality in the first heart sound?

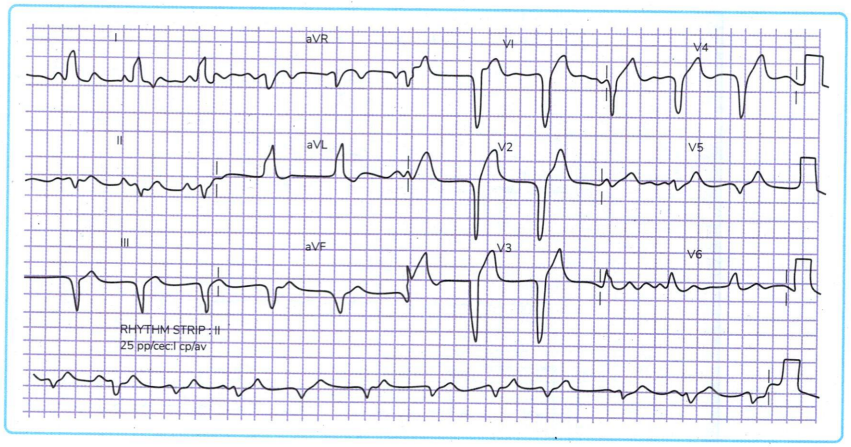
A. Reverse splitting of first heart sound

B. Pathological splitting of first heart sound

C. Soft S1

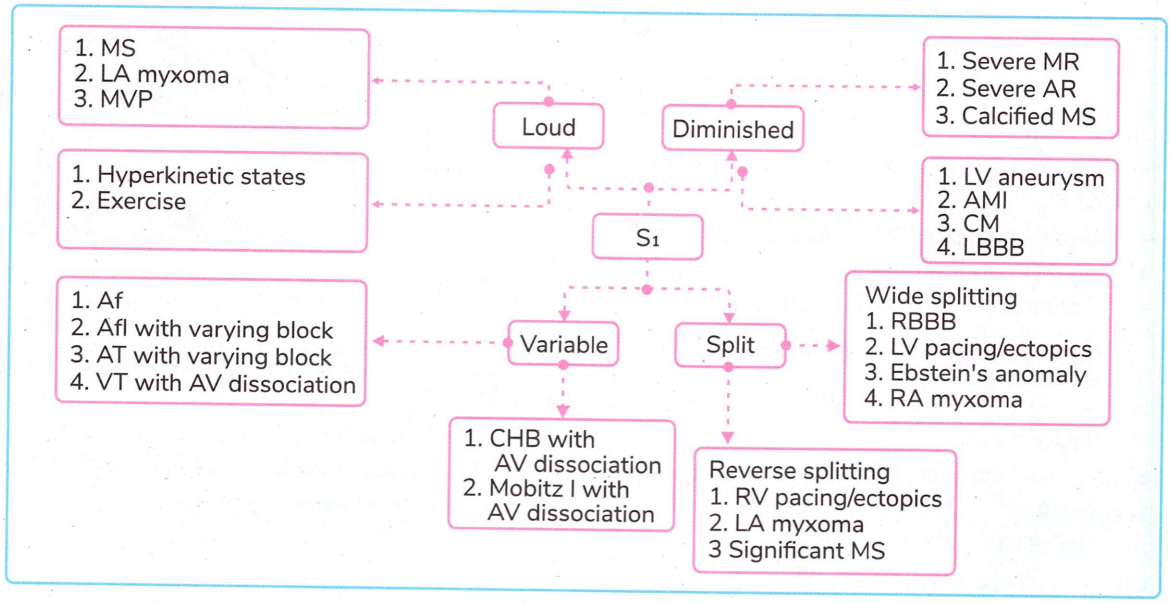
D. Loud S1

- In the ECG
  - Wide QRS complex
  - Deep and wide S wave in V1
  - Broad R wave in V6
- Suggestive of LBBB
  - RV contraction occurs first, followed by LV contraction
  - T1 is followed by M1
  - Reverse splitting



Summary

00:34:06



Second Heart Sound

00:36:11

- Heard during Isovolumetric relaxation phase/ Protodiastolic phase
- The second heart sound signals the onset of ventricular diastole
- Has two components:
  - A2 - Aortic valve closure
  - P2 - Pulmonary valve closure

Mechanism of Second heart sound

00:36:50

- In Isovolumetric relaxation phase
  - Pressure in ventricles reduces
- The blood will try to regurgitate back into the ventricles
  - The aortic valve closes → A2
- Similar mechanism on right side → P2
- A2 is followed by P2
  - Because left side pressure is more than right side pressure

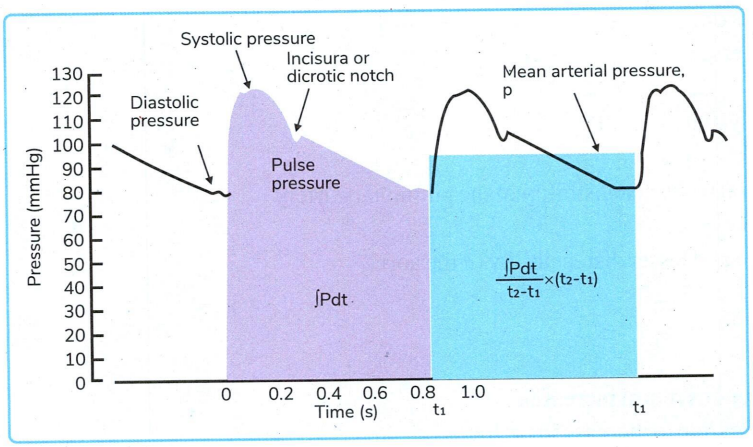
Characteristics of S2

00:38:09

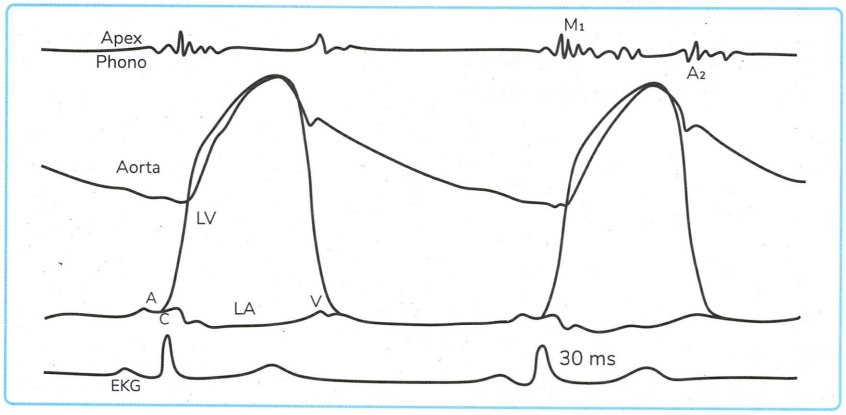
Character	Findings
Frequency	High
Duration	0.11 sec
Incisura of Aortic pressure coincides.	A2
Incisura of Pulmonary pressure coincides.	P2
A2-P2 interval	• Expiration: <30 msec • Inspiration: 40-50 msec
A2 is earlier than	P2
A2 is louder than	P2

Incisura

Q. Semilunar valve closure is represented by?

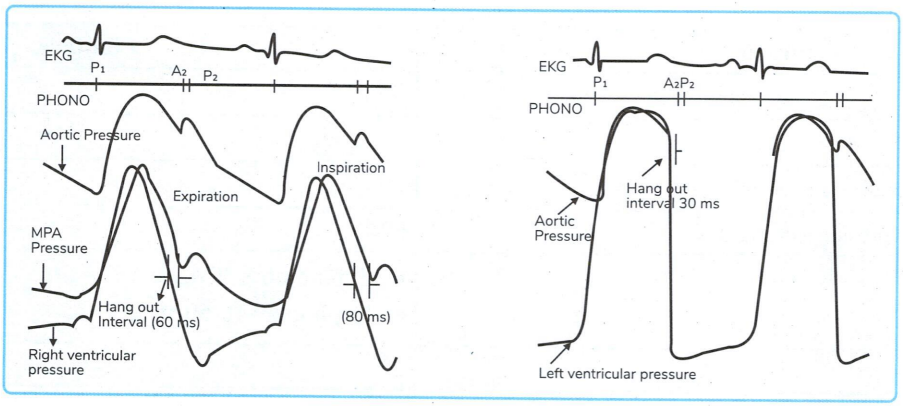


A. Systolic pressure
B. Diastolic pressure
C. Incisura
D. Pulse pressure



- A2 corresponds to incisura
  - Point where semilunar valves are closing
- Second heart sound is heard at the onset of ventricular diastole

Hangout Interval



- The semilunar valves are expected to close at the point of crossover of the pressures.
  - The point where ventricular pressure falls lower than the arterial pressure.
- In reality, these semilunar valves close slightly later than the expected point.
  - The time interval between the crossover of the pressures and the actual closure of semilunar valves → Hangout interval
- Hangout interval depends on:
  - Distensibility of the vessel
  - Vascular resistance
  - Recoil of the arterial system
- For P2, Hangout interval is 60ms
  - Due to less pressure and more distensibility of the pulmonary artery
- For A2, Hangout interval is 30ms
  - Due to higher pressure and lesser distensibility of the aorta

Mechanism of the split

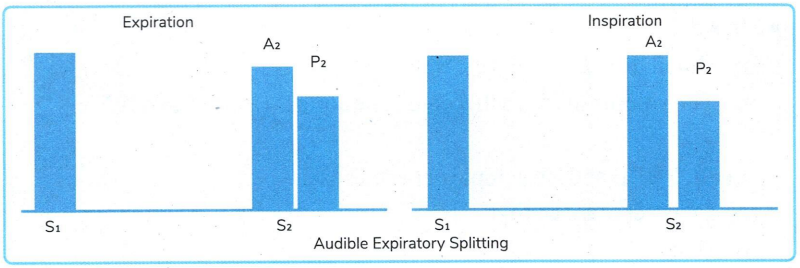
00:44:32

Inspiration

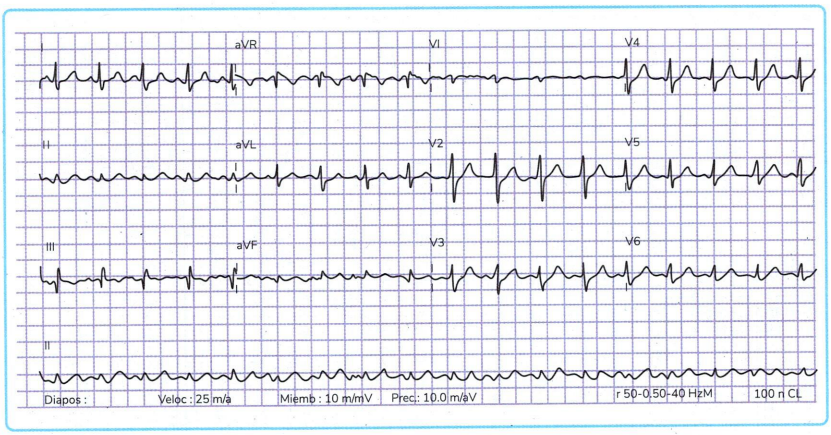
- During inspiration, the venous return increases
- RA & RV volume increases due to increase in preload
  - Delayed closure of P2
- LA & LV volume decreases
  - Early A2
- Duration between A2 and P2: > 40 -50 msec
  - Split present

Expiration

- LA & LV volume increases
  - Delayed A2
- RA & RV volume decreases
  - Early P2
- Duration between A2 and P2: < 30msec
  - No split



Q. A 24-year-old pregnant female presented with sudden onset dyspnea & palpitations. ECG is as follows. What is the abnormality in the second heart sound?



A. Narrow split S2

B. Wide S2

C. Reverse split S2

D. Absent S2

- Differential diagnosis of pregnant women with sudden onset dyspnea
  - Pulmonary embolism
- Most common ECG finding in PE – Sinus Tachycardia
- In the ECG
  - HR is 110/min
  - Right axis deviation
  - S1Q3T3 pattern

Wide Physiological Splitting of S2

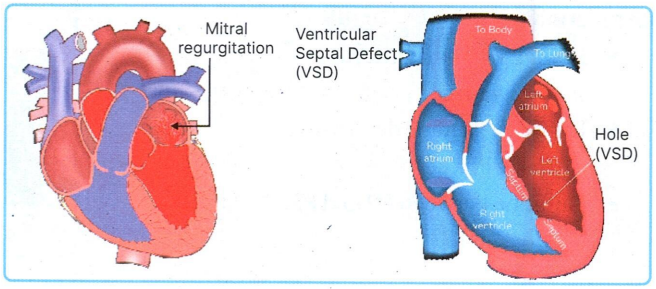
00:50:10

Early A2	Delayed P2

• Mitral regurgitation
• Ventricular septal defect
	• Pulmonary Embolism
	  ○ Increased afterload on RV
	• RBBB
	• LV ectopic or paced beats
	• Atrial septal defect
	  ○ Wide fixed spilt
	• Pulmonary hypertension
	• Pulmonary stenosis

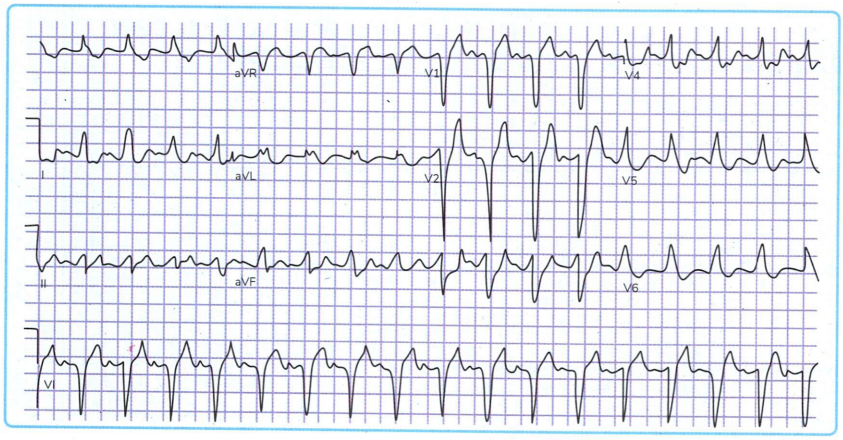
- In MR
  - During ventricular contraction
    → The volume of blood that enters the aorta is less → Early A2

- In VSD
  - Blood from LV enters the RV
  - The volume of blood that enters the aorta is less → Early A2



- In PE, RBBB and LV ectopic or paced beats
  - LV is depolarized first
  - Delayed P2

Q. A 54-year-old male, k/c/o DM presented with history of chest pain since 6 hours. O/E BP is 134/82 mmHG. Troponin is positive. ECG is as follows. What will be the abnormality in the second heart sound?



A. Normal S2
B. Wide split S2
C. Reverse split of S2
D. Absent A2

- ECG is suggestive of LBBB
  - RV will contract first - First P2
  - LV will contract later - Second A2

Reverse Splitting of S2

00:55:11

Delayed A2	Early P2
• LBBB • RV ectopic beats • RV paced beats • Patent ductus arteriosus	• WPW – Type B ○ The accessory pathway is towards the right ventricle

- In LBBB and RV ectopic or paced beats
  - RV is depolarized first
  - P2 followed by A2

Causes of fixed (wide) split S2

00:56:52

- Fixed split is seen in:
  - Atrial septal defect
  - Severe pulmonary stenosis
  - Acute RHF
  - TAPVC
- There is no variation with respiration
  - Normally, the split increases with inspiration and disappears in expiration

ASD-Mechanism of fixed split

- During inspiration
  - RA volume increases due to increased venous return
    → Split is present
- During expiration
  - The RA volume increases due to the flow of blood from LA to RA through ASD.
  - Delayed P2
    → Split present in expiration and inspiration

Causes of single S2

00:59:35

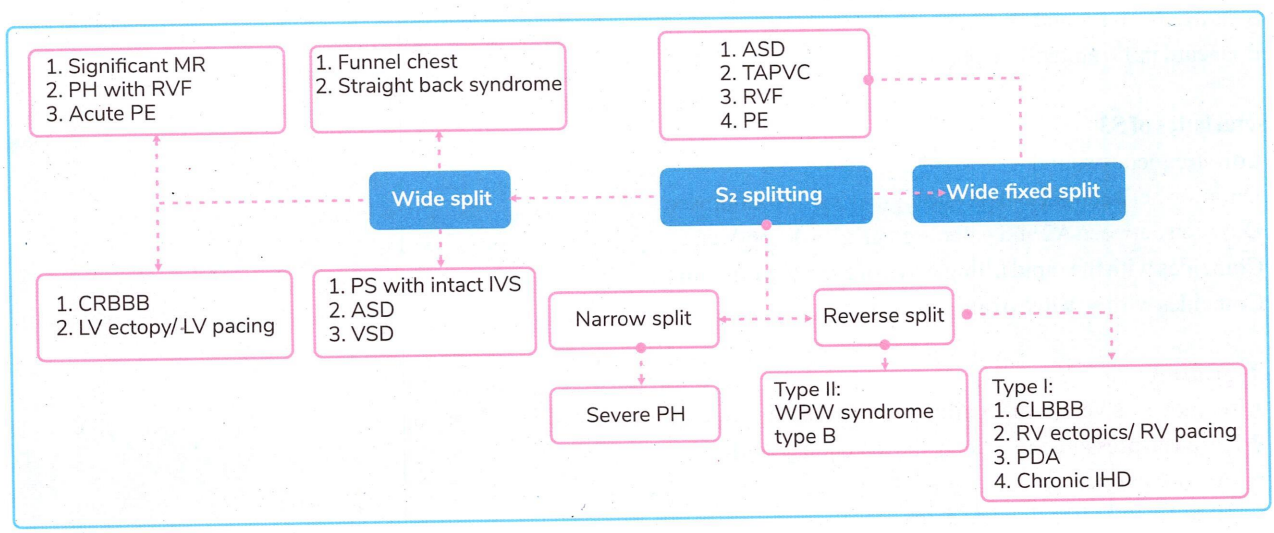
Causes of delayed A2 (A2 merges with P2)	• LBBB • RV ectopic beats • RV paced beats • Severe AS • Hypertensive cardiovascular disease

One component is absent/ inaudible	• Severe TOF
	  ○ Subvalvular pulmonary stenosis → Absent P2
	• Severe AS
	  ○ Soft A2 – inaudible
	• Severe Pulmonary stenosis → Soft P2
	• Pulmonary valve atresia → Soft P2
	• Eisenmenger syndrome
	  ○ Shunt: Right → Left
	  ○ Due to pulmonary hypertension
	  ○ Loud P2- overlaps A2

Inability to hear 2 components	• Emphysema • Obesity

Summary

01:02:40



Third heart sound

01:03:45

Q. 3rd heart sound are best heard with:

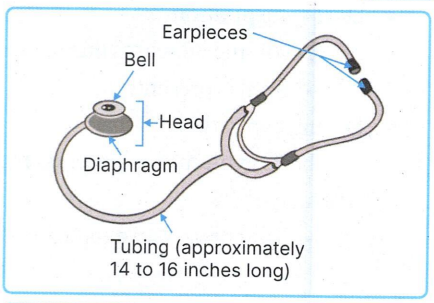
A. A - Bell

B. B - Head

C. C - Diaphragm

D. D - A & C

- All low-pitched sounds are best heard with the bell of the stethoscope
  - 3rd heart sound is a low-pitched sound.



Q. 3rd heart sound is best heard at?

A. A

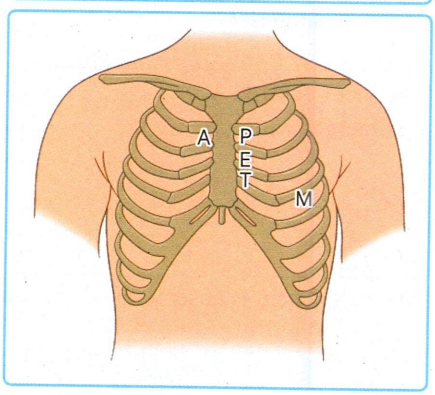
B. P

C. T

D. M

E. M & T

- RV S3 - Tricuspid area
- LV S3 - Mitral area
- Single area is the apex



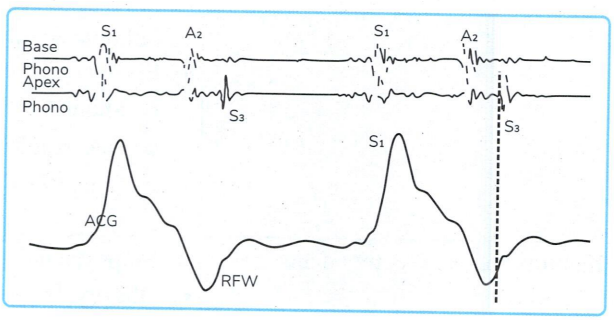
Q. Physiological S3 is heard in all except?

A. Age upto 40 years

B. Children

C. Pregnancy

D. Sumo fighters



- Physiological S3 can be heard in:
  - Young adults
  - Children
  - Pregnancy
  - Athletes
- Systolic phase of ventricle
  - Between S1 and A2
- Diastolic phase of ventricle
  - Between S2 and next S1
- S3 is a diastolic sound
  - Heard in 1st rapid filling phase

Characteristics of S3

01:06:40

- Low-frequency sound
- Duration between A2 and physiological S3: 120-200msec
- Duration between A2 and pathological S3: 140-160msec
- Coincides with the rapid filling wave of apex cardiogram
- Coincides with 'y' wave of atrial pressure

Physiological S3

- Also known as Ventricular gallop/ Ventricular diastolic gallop/ Protodiastolic gallop
- When atrial pressure is elevated due to venous return
  - AV valves open
  - Blood gushes into the ventricles
  - Blood will hit the ventricular wall → S3

- Diastolic sound
- Best heard in apex
  - In the left lateral position
  - Heard with the bell of the stethoscope

Q. A 54-year-old female patient is admitted with dyspnea. On examination, murmur is heard, which is suggestive of MR. What finding points to chronic severe mitral regurgitation?

A. Murmur louder on inspiration

B. Presence of S3

C. Corrigan's sign

D. Narrow pulse pressure

- In mitral regurgitation
  - During LV contraction
    → Blood enters the Aorta and LA
    → LA pressure is more than normal because of blood from the Pulmonary veins and LV
  - When the Mitral valve opens
    → The entire blood hits the LV wall → S3
    → Pathological S3
  - Leads to LV failure

01:12:04

Pathological S3- Ventricular dysfunction

- Cardiomyopathies
  - Ventricular dysfunction → LV end-systolic volume increases
  - LA blood is added to this volume
    → LV end-diastolic volume increases
  - Subsequently, in the next cycle, the blood from LA also enters LV
    → Leading to High-intensity S3
- Ventricular dysfunction is also seen in:
  - MR
  - AR
  - AS
- Pathological S3 is heard in all forms of cardiomyopathy:
  - Dilated cardiomyopathy
  - Hypertrophic cardiomyopathy
  - Restrictive cardiomyopathy

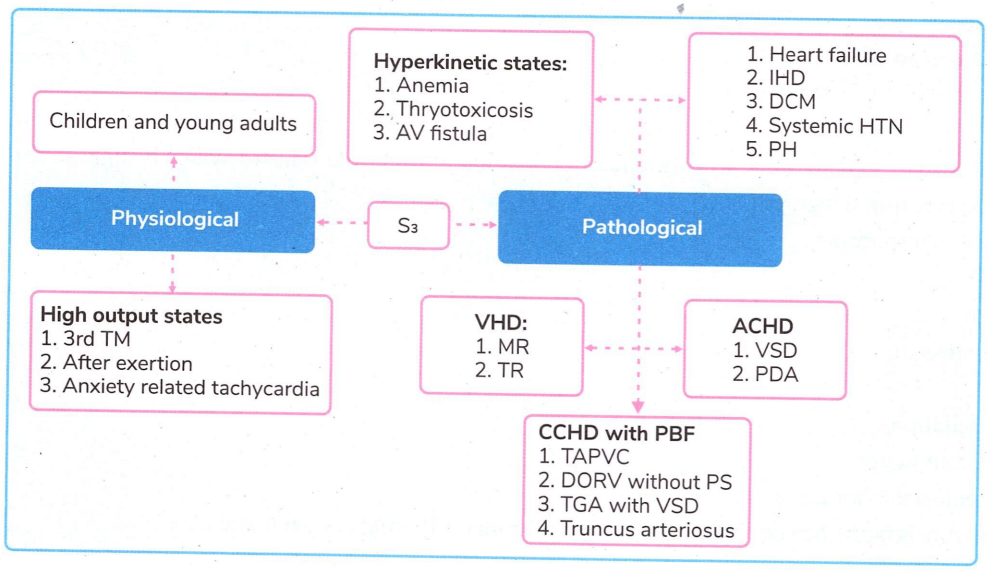
Pathological S3- Hyperkinetic states

- Anemia
- Thyrotoxicosis
- Beri-Beri
- Fever
- Pregnancy

Pathological S3- Left to Right shunt.

- Due to RV failure → RV S3
  - VSD
  - PDA
  - ASD

Summary



Fourth heart sound

01:16:32

Q. A 78-year-old woman is admitted with heart failure. The underlying cause is determined to be aortic stenosis. Which sign is most likely to be present?

A. Pleural effusion on chest X-ray

B. Raised jugular venous pressure (JVP)

C. Bilateral pedal oedema

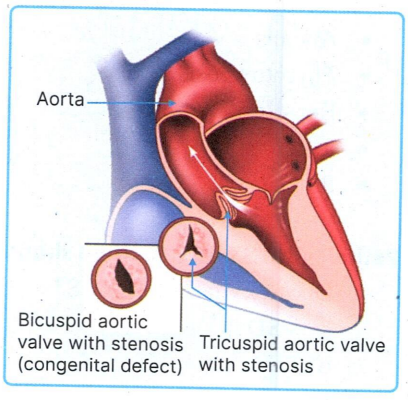
D. Prominent S4

- S4
  - Low-pitched sound
  - Heard during diastole
    - → In 2nd rapid filling phase
    - → Due to atrial contraction
  - Never present in normal individuals
    - → Always pathological.
    - → Heard in the apex
    - → With the bell of the stethoscope
  - Can be heard in normal individuals with phonocardiograph
  - Also known as S4 gallop/ atrial gallop

Causes of Fourth Heart Sound

01:18:22

- Mnemonic: HOCM
  - Due to high ventricular pressure:
    - → Hypertrophic obstructive cardiomyopathy
    - → Aortic stenosis and Pulmonary stenosis
    - → Cardiac Tamponade
  - Due to LV failure → volume overload
    - → Myocardial infarction
- In AS
  - LV pressure increases
  - LA has to contract strongly to push the blood into the LV
    - → Causes pathological S4



Q. A 79-year-old woman is admitted to the coronary care unit (CCU) with unstable angina. She is started on appropriate medication to reduce her cardiac risk. She is hypertensive, fasting glucose is normal, and cholesterol is 5.2. She is found to be in atrial fibrillation. What is the most appropriate sign?

A. Loud S1
B. Wide split S2
C. Absent S4
D. Pathological S3
E. Midsystolic click

- In atrial fibrillation, there is:
  - Variable S1
  - Absent S4
    - → Due to incomplete atrial contraction
    - → Cannot be heard even with phonocardiograph

Comparison of all heart sounds

01:22:08

Feature	S1	S2	S3	S4
Character	High-pitched	High-pitched	Low-pitched	Low-pitched
Duration	0.14 secs	0.11 secs	0.10 secs	0.10 secs
Frequency (Hz)	25-45	50	<20	<20
Cause	M1 T1	A2 P2	Rapid ventricular filling	Atrial contraction
Timings	Isovolumetric contraction	Isovolumetric relaxation	1st rapid filling phase	2nd rapid filling phase
Best heard with	Diaphragm	Diaphragm	Bell	Bell

Additional heart sounds

01:24:20

Q. A 45-year-old woman complains of increasing shortness of breath on exertion, as well as orthopnoea, for the previous 3-4 months. She has apparently recovered from pericarditis about a year earlier. On ECG, there is low voltage, especially in the limb leads, and the chest X-ray shows pericardial calcification. The presumptive diagnosis is constrictive pericarditis. Which of the following physical signs would be consistent with this?

A. Pericardial knock
B. Third heart sound
C. Fourth heart sound
D. Rales at both lung areas
E. Loud first and second heart sound

- In constrictive pericarditis - Pericardial knock is present

	Ejection click	Opening snap	Pericardial knock	Tumour plop
Condition	AS, PS	MS, TS	Constrictive pericarditis	Atrial myxoma
Phase-cardiac cycle	Systole	Diastole	Diastole	Diastole
Pitch	High	High	High	Low
Mechanism	Due to opening of stenosed semilunar valves	Elevated atrial pressure → opens AV valves with high intensity	The ventricle hits the thick and calcified pericardium during relaxation	• Atrial myxoma originates from the interatrial septum. • They overlap the AV valve opening. • Sound produced when myxoma hits the valve annulus.

Opening snap

01:29:00

- Heard after 0.03 – 0.15 secs after A2
  - The distance between A2 and the opening snap decides the severity of MS
    - → Narrow - Severe
    - → Wide - Mild
- The presence of opening snap indicates that
  - MS is organic
  - Valve cusps are pliable
- Opening snap is not present in severe AR, causing functional MS

Early diastolic intervals

01:30:40

Interval	Duration
A2-P2 during expiration	0.03 sec
A2-P2 during inspiration	0.04 - 0.05
A2-S3 (Physiological)	0.12 - 0.20
A2-S3 (Pathological)	0.14 - 0.16
A2-OS	0.03 - 0.15
A2 Pericardial knock	0.10 - 0.12

Q. A 59-year-old man presents for a well-person check. A cardiovascular, respiratory, gastrointestinal, and neurological examination is performed. No significant findings are found, except during auscultation a mid-systolic click followed by a late systolic murmur is heard at the apex. The patient denies any symptoms. The most likely diagnosis is:

A. Barlow syndrome

B. Austin Flint murmur

C. Patent ductus arteriosus

D. Graham Steell murmur

E. Carey Coombs murmur

- Mid-systolic click followed by a late systolic murmur is heard in Mitral valve prolapse
  - Also known as Barlow syndrome

Q. You see a 57-year-old woman who presents with worsening shortness of breath coupled with decreased exercise tolerance. She had rheumatic fever in her adolescence and suffers from essential hypertension. On examination, she has signs which point to a diagnosis of mitral stenosis. Which of the following is not a clinical sign associated with mitral stenosis?

A. Opening snap

B. Atrial fibrillation

C. Pan-systolic murmur, which radiates to the axilla

D. Tapping, undisplaced apex beat

E. Right ventricular heave

- In mitral stenosis:
  - Loud S1 is heard, opening snap
  - Atrial fibrillation
    - → Arrhythmogenic left atrium due to distended LA or LA pressure rise
  - Tapping, undisplaced apex beat
  - Right ventricular heave due to pulmonary hypertension

Added sounds

01:33:56

Systolic sounds	Diastolic sounds
• Ejection click • Mid systolic click	• Opening snap • S3 • S4 • Pericardial knock • Tumor plop

3 ARTERIAL PULSE

Definition

00:01:27

- Greek word
- Wave form produced after cardiac systole
  - Traverses in the peripheral direction of the vessel wall.
- The velocity of the arterial pulse is much higher than the blood column in the center of the vessel.
  - Speed of pulse – 5 m/sec or 18km/ hour
  - Blood flow – 0.5 m/sec or 1.8 km/ hour

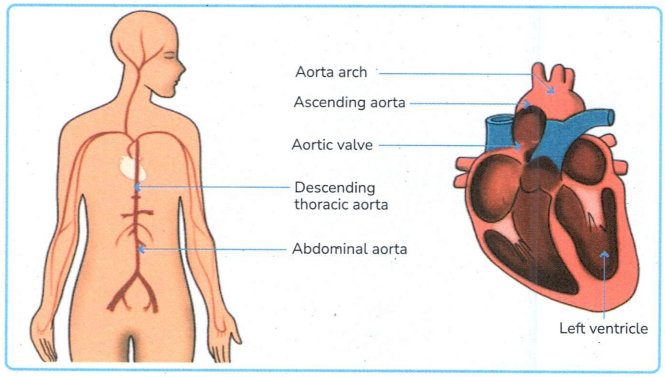
Time at which pulse wave arrives after cardiac systole

Carotid	30 milliseconds
Brachial	60 milliseconds
Femoral	75 milliseconds
Radial	80 milliseconds

Genesis of the Arterial Pulse

00:05:00

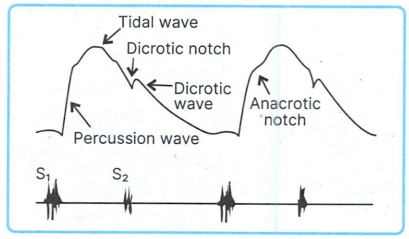
Contraction of the left ventricle
↓
Systolic pressure increases in LV
↓
Opens Aortic valve
↓
Aorta
↓
Radial artery



Normal waveforms of arterial pulse

00:07:30

- In systole (upstroke): 2 positive deflections
  - Percussion wave
    → Arrival of impulse after LV ejection
  - Tidal wave
    → 2nd wave
    → Deflection from the upper part of the body
- In diastole (descending limb) -1 positive deflection
  - Dicrotic / Diastolic wave
    → Deflection from the lower part of the body



Pulse wave patterns in central and peripheral arteries

00:10:55

Central arterial pulse	Peripheral arterial pulse

• Carotid artery
  ○ Palpable
  ○ Most accurate representation
• Ascending aorta
• Innominate vessel
  ○ Brachiocephalic trunk
	• Brachial artery
	  ○ Most suitable
	• Radial artery
	• Femoral artery
	• Popliteal artery
	• Dorsalis pedis artery