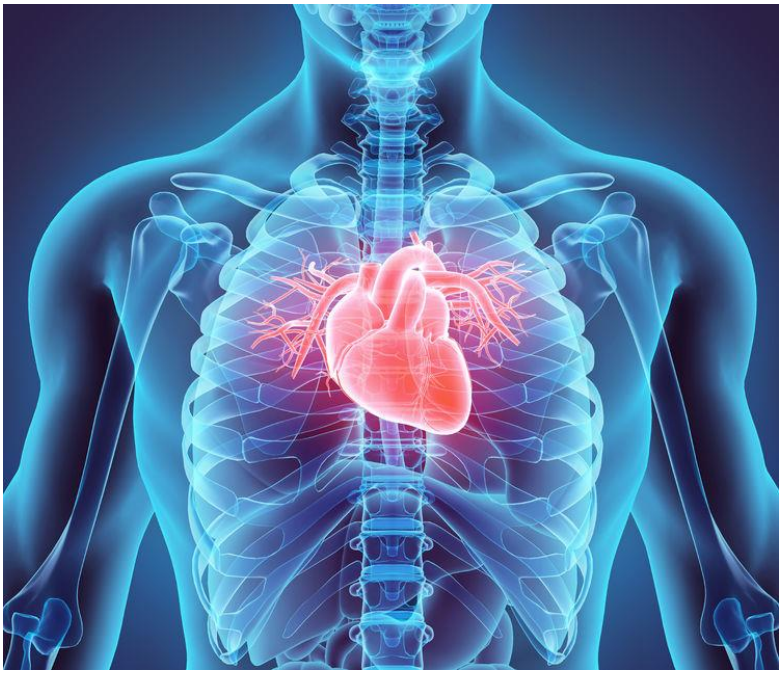
Cardiovascular System – Clinical Notes



Abdulrahman Aldabobi
CLINICOSIS

The Heart – Clinical Notes

1-Anginal Chest Pain

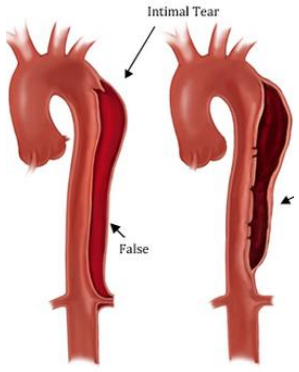
a) Site → Retrosternal
b) Onset → Progressive, increase in intensity over 1-2 minutes
c) Character → Constricting
d) Radiation → Neck, arm epigastrium
e) Associated Symptoms → Breathlessness
f) Timing → Intermittent, each episode lasts 2-10 minutes
g) Types
    a. Stable Angina → Angina that occurs upon exacerbation only (Excxersise, emotion, cold windy weather), and relived by rest and nitrates
    b. Unstable Angina → Angina at rest, not relieved by nitrates
    c. Prinzmetal's Angina → Angina due to vasospasm in the coronary arteries rather than obstruction, relived by calcium channel blockers and not nitrates

2-Myocardial Infarction (MI)

a) Site → Retrosternal
b) Onset → Rapid over a few minutes
c) Character → Constricting
d) Radiation → Neck, arm epigastrium
e) Associated Symptoms → Breathlessness, autonomic symptoms (sweating, nausea, vomiting) and angor animi
f) Timing → Acute presentation with prolonged duration (up to 30 minutes)
g) Exacerbated by stress and exercise, not relived by rest or nitrates
a) Cause → Coronary artery obstruction
b) Angina Equivalent → Dyspnea to myocardial ischemia

3-Aortic Dissection

a) Tearing in the layers of the aorta, forming a false lumen in which the blood will be trapped
b) Symptoms
    a. Tearing chest pain radiates to the back (intrascapular), with very sudden onset
    b. The trapped blood in the false lumen causes hypoperfusion of some organs, and depending on the site of the tearing, it may cause
        i. Focal Neurological Signs (Stroke) and Syncope → If the trapping occurs before the origin of the carotid arteries
        ii. Limb Ischemia
        iii. Upper limb pulse asymmetry
        iv. Mesenteric Ischemia (signs and symptoms of mesenteric ischemia will be discussed)

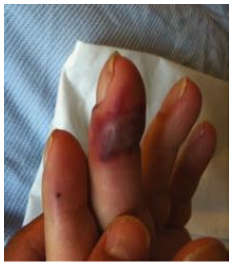


c) No relieving factors are present
d) Risk Factors Include Smoking and Connective Tissue diseases (Marfan, EDS)

4-Pericarditis

a) Inflammation of the pericardium
b) Symptoms
    a. Chest pain
        i. Sharp 'Stabbing' and pleuritic
        ii. Exacerbated by inspiration and lying down and Relieved by sitting up and leaning forward and with some NSAIDs
        iii. Radiate to the left shoulder or the back
    b. Fever
c) Causes
    a. Idiopathic
    b. Coxsackie B virus (think of a patient presets with pleuritic chest pain one week after a upper respiratory infection)
    c. Post-MI (Dressler Syndrome → Pericarditis two weeks after MI)
    d. Autoimmune diseases
    e. Uremia → Think with acute kidney injury who develops pleuritic chest pain after a while
    f. Connective Tissue diseases
    g. After a surgery, radiotherapy, and catheter ablation

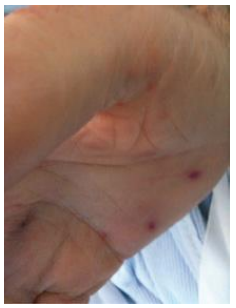
5-Coarctation of the Aorta

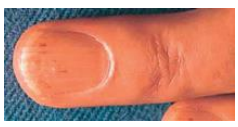


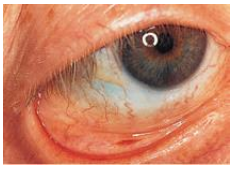
a) Congenital Narrowing in the aorta
b) Associated with turner syndrome
c) Cause Brachiofemoral Delay

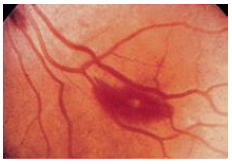
6-Endocarditis

a) Inflammation of the endocardium, which is usually the inner lining of the heart, formed by endothelial cells
b) Symptoms
    a. Fever
    b. Roth spots → Found in the retina with cotton-wool center, can also be found as a complication of HTN and DM
    c. Osler Nodes → painful nodes on the finger and the toe beds
    d. Janeway lesions → painless lesions on the palm or the sole
    e. Splinter Hemorrhage → Hemorrhages on the nail beds (found in case of trauma and other vasculitis disorders)
    f. Murmurs (Mitral Valve more commonly, but it can affect also the tricuspid valve)
    g. Petechial Hemorrhages May induce thrombus formation
    h. Hematuria
c) Risk factors
    a. Recent dental procedures
    b. Recent sepsis + IV drug abuse
    c. Prosthetic valves
    d. Colon cancers (induce Strep. Bovis sepsis)









7-Hypertension

a) Elevated systolic or diastolic blood pressure or both
b) Usually Asymptomatic, but if severe it may cause headaches and visual disturbances
c) Primary Hypertension
    a. Idiopathic hypertension
    b. Most common
d) White Coat Hypertension
e) Secondary Hypertension
    a. Hypertension due to secondary cause, and some of these causes are listed below
    b. Renal Artery stenosis → Think of a patient with widespread vascular disease and Renal Bruit
    c. Pheochromocytoma
        i. Tumor in the adrenal medulla lead to increased secretion of catecholamines (epinephrine and norepinephrine) which cause hypertension
        ii. Think of a patient with presents with extremely high and refractory (cannot be controlled by antihypertensive agents) blood pressure (180mmHg), headaches, palpitations, and sweating
        iii. Family history or past medical history of parathyroid and medullary thyroid neoplasms may be present, as pheochromocytoma can be present as part of the Multiple Endocrine Neoplasia 2A (MEN 2A)
    d. Primary Aldosteronism
        i. Think of HTN with symptoms of hypokalemia (abnormal heart rhythm, muscle weakness)
    e. Cushing Disease
        i. Think of patient with HTN, moony facies, central obesity, proximal muscle weakness, abdominal Striae
        ii. Chronic Glucocorticoid use may also cause Cushing disease
    f. Coarctation of the aorta → HTN with low lower limb pulses and Radiofemoral delay
    g. Acute Polycystic Kidney disease → HTN with bilateral palpable kidney (more about APKD in the renal chapter)

4.13 British Hypertension Society classification of blood pressure (BP) levels

BP	Systolic BP (mmHg)	Diastolic BP (mmHg)
Optimal	<120	<80
Normal	<130	<85
High normal	130–139	85–89
Hypertension		
Grade 1 (mild)	140–159	90–99
Grade 2 (moderate)	160–179	100–109
Grade 3 (severe)	>180	>110
Isolated systolic hypertension		
Grade 1	140–159	<90
Grade 2	>160	<90

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11-Signs of Hyperlipidemia

a) Xanthelasma
b) Tendon Xanthoma
c) Corneal Arcus

8-Heart Failure

a) Most common cardiovascular cause of both chronic and acute dyspnea ()
b) Decreased Heart functions, divided depending on the affected function to
    a. *Systolic Heart Failure*
        i. Decreased heart's ability to pump blood
        ii. Caused by Damage to cardiomyocytes (as in MI), dilated cardiomyopathy
    b. *Diastolic Heart Failure*
        i. Decreased heart's ability to get filled up
        ii. Caused by hypertrophy of the of the heart muscles (which decreases the space inside the heart's champers), causes of hypertrophy may be
            1. Primary → Hypertrophic Cardiomyopathy
            2. Secondary to increased afterload → Chronic Hypertension and Valve stenosis
c) Divided depending on the affected side of the heart to
    a. Left Sided heart failure
        i. Commonly due MI, HTN, Dilated Cardiomyopathy
        ii. Present with symptoms of pulmonary venous congestion
            1. Pulmonary Edema
            2. Orthopnea → Signifies advanced disease
            3. Paroxysmal Nocturnal Dyspnea → The patient awake after several hours of sleep due to breathlessness (Asthma patients awake at dawn)
    b. Right Sided Heart Failure
        i. Commonly due to left heart failure
        ii. Cor Pulmonale → Isolated right heart failure due to pulmonary cause (pulmonary hypertension and pulmonic valve stenosis)
        iii. Present with symptoms of systemic venous congestion
            1. Peripheral Edema (Bilateral Lower Limb Edema)
            2. Increased JVP
            3. Hepatomegaly → Decreased IVC drainage → Decreased Hepatic Vein drainage → Blood congestion inside the liver → Hepatomegaly
d) Patients with acute HF and pulmonary edema prefer to be upright, whereas patients with massive PE prefer lying flat
e) New York Heart Classification of heart Failure
    a. Class I → No Limitation
    b. Class II → Slight limitation, the patient is comfortable at rest, and ordinary physical activity may cause dyspnea
    c. Class III → Marked limitation, less then ordinary physical activity leads to symptoms
    d. Class IV → Symptoms even at rest
f) May cause frothy white or blood stained sputum
g) Advancer Heart Failure presents in a condition called cardiac cachexia, due to the prolonged catabolic state in the body, characterized by
    a. Ascites
    b. Weight Loss
    c. Muscle wasting
h) A well-know cause of central cyanosis

9-Palpitations

a) *Extrasystoles*
   a. Premature paroxysmal (sudden) extra contraction of the heart
   b. An electrical signal is released from the ventricular myocytes leads to an early contraction of the ventricles with low SV, then, when the SA node sends signals to the ventricles, they will not contract as they are still in the repolarization phase (Skipped beat) → The next beat will be forceful and with higher SV
   c. Described as missed beat by the patients
   d. Causes → Alcohol, caffeine and Fatigue
   e. Relieved by walking or any other mild physical activity

b) *Sinus Tachycardia*
   a. Increased HR due to increased firing of the SA node
   b. Character → Regular Fast
   c. Induced by exercise, anxiety, stress

c) *Supraventricular Tachycardia*
   a. Increased HR due to increased heart rhythm that originates above ventricles, either from the atrial myocytes or from the AV node
   b. Usually caused by the presence of pathways that allow signals to reach the ventricular myocytes bypassing the AV node
   c. Character → Regular, Fast
   d. Associated with lightheadedness, chest tightness and polyuria (thought to be due to increased ANP production from the atrial myocytes)

d) *Atrial Fibrillation*
   a. Increased HR due to spontaneous depolarization of the atria
   b. Character → Irregularly Irregular
   c. Same associated symptoms of the supraventricular tachycardia (not a well-known cause for syncope)
   d. Caused by
      i. Alcohol (Holiday Syndrome)
      ii. Hyperthyroidism → Always check for TSH in patients with A Fib
      iii. Ectopic Foci → Abnormal myocardium that generated electrical signals independently of the SA node
      iv. Scarring of the myocytes (Post-MI)
   e. Complications (Stasis of blood in some parts of the left heart leads to thrombus formation, which may be then transmitted to any part of the body as the heart contract and pumps it)
      i. Strokes
      ii. Mesenteric Ischemia
   f. WPW and Atrial Fibrillation
      i. Wolff-Parkinson Syndrome is characterized by the presence of an electrical pathway that transmits signals from the atria to the ventricle, independently from the AV node
      ii. Atrial Fibrillation does not cause ventricular fibrillation as the AV node slows and decreases the number of signals transmitted form the atria to the ventricles → But in case of WPW, the secondary pathway will transmit these signals inducing ventricular tachycardia
      iii. WPW + A Fib = V Fib (this is how A fib may cause syncope)

e) *Ventricular Fibrillation*
   a. Increased HR dur to depolarization of the ventricles, independently of the SA nodes
   b. Character → Regular Fast
   c. Causes presyncope, syncope, can be fatal
   d. Affects patients with cardiomyopathy and MI
   e. Usually, fatal

10-Syncope and Presyncope

a) Syncope → Transient loss of consciousness mainly due to cerebral hypoperfusion
b) Presyncope → Lightheadedness without loss of consciousness
c) Causes
   a. Postural Hypotension
      i. Fall of more than 20mmHg in systolic blood pressure
      ii. Causes
         1. Hypovolemia
         2. Drugs (such as vasodilators, ACEIs. Beta blockers, Calcium channel blockers, digoxin, amiodarone)
         3. Autonomic neuropathy (such in diabetes)
         4. elderly
   b. Neurocardiogenic Syncope
      i. Abnormal Autonomic reflexes produce slow heart rate and vasodilation → Cerebral hypoperfusion → Syncope (Vasovagal Attack)
      ii. Triggered by standing long time in warm environment, or by strong emotional stimuli
      iii. Preceded typically a prodrome of lightheadedness, tinnitus, nausea, sweating and facial pallor, and a darkening of vision from the periphery as the retinal blood supply
      iv. Raising legs may improve cerebral perfusion and the patient wakes up → often flushing from vasodilatation and nauseated or even vomiting due to vagal overactivity.
      v. Hypersensitive carotid sinus syndrome is another cause
   c. Arrhythmias
      i. Stokes-Adams Attacks → Syncope due to any degree of heart block
      ii. Ventricular Fibrillation
   d. Obstruction of the cardiac output
      i. Severe Aortic Stenosis
      ii. Hypertrophic Cardiomyopathy
      iii. Cardiac Tumors (Myxoma)
      iv. Thrombosis
      v. Failure of the prosthetic heart valves to open
      vi. Massive PE

11-Esophageal pain (as a cause of chest pain)

a) Esophageal Pain that presents as chest pain, has three main causes
   a. Esophageal Spasms
   b. GERD
   c. Hiatus Hernia (the fundus of the stomach herniates through the diaphragm to reach the thoracic cavity)

b) Lying flat/some foods may trigger, Not relieved by rest; nitrates sometimes relieve
c) Presents as heartburn and acid reflux (more details are covered in the GI chapter summary

Arterial Pulses

1-Causes of increased pulse volume

a) Physiological Causes, Pregnancy, Exercise, Advanced age, Increased Temperature
b) Pathological causes, Hypertension, Fever, Thyrotoxicosis, Anemia, Aortic Regurgitation, Paget's disease, Peripheral AV shunts

2-Causes of decreased pulse volume

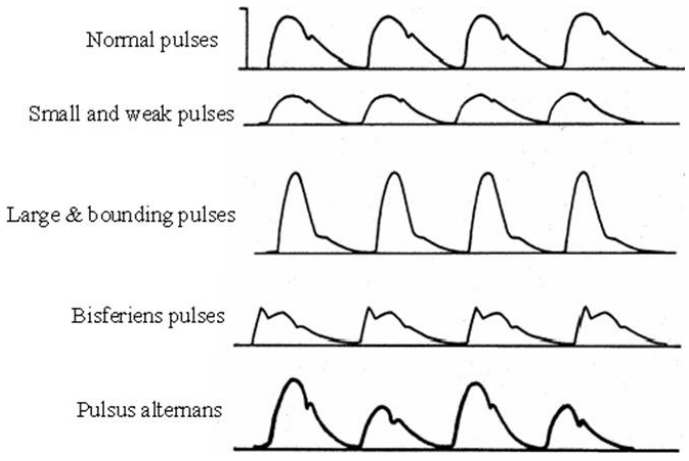
a) Severe heart Failure
b) Inadequate ventricular filling (Hypovolemia, Cardiac Tamponade, Mitral Stenosis)

3-Causes of Asymmetric pulses

a) Occlusive Peripheral arterial disease
b) Aortic Dissection
c) Coarctation of the aorta (Radiofemoral Delay)

4-Abnormal Pulses

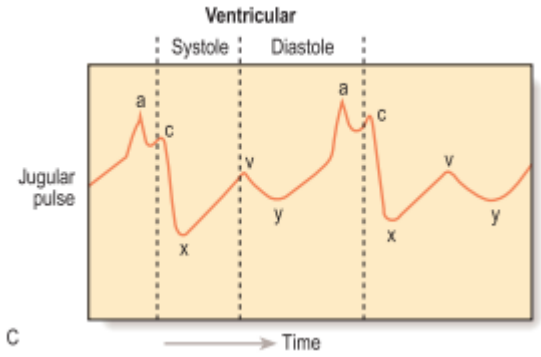
a) *Pulsus Parvus et tardus*
    a. Slow Rising pulse with reduced peak
    b. Severe aortic stenosis
b) *Collapsing Pulse*
    a. Rapidly rising pulse with increased peak, followed by rapid fall in the pressure
    b. Severe Aortic Regurgitation
c) *Pulsus Biseferiens*
    a. Increased pulse with double systolic pulse and mid-systolic dip
    b. Concomitant aortic stenosis and regurgitation
d) *Pulsus Alternans*
    a. Beat to Beat variation in pulse volume with normal rhythm
    b. Advanced HF
e) *Pulsus Paradoxus*
    a. Systolic blood pressure falls more than 10mmHg during inspiration
    b. Due to increased pressure on the heart
        i. Cardiac Tamponade
        ii. Pericardiac constriction
        iii. Severe Asthma



Jugular Venous Pressure

1-Normal JVP tracing curve will show

a) A wave → due to Atrial Contraction (Active filling of the atrium)
b) C wave → due to Ventricular contraction against closed tricuspid and pulmonic valve → Upward displacement of Tricuspid valve (pushed into the lumen of the atria → Increased atrial pressure)
c) X Descent → Downward displacement of the tricuspid valve, as the ventricle is now contracting against opened pulmonic valve
d) V Wave → filling of the atrium, closed tricuspid valved and opened pulmonic valve
e) Y Descent → Passive emptying of the atrium (Passive filling of the ventricle)



2-Abnormalities of JVP tracing curve

a) *Absent A wave* → Atrial fibrillation (no atrial contraction)
b) *Giant A wave* → Tricuspid Stenosis (the atrium contracts against stenosed valve, increasing the pressure inside the atrium)
c) *Cannon A wave*
    a. Irregular → Complete Heart block (the atrium contracts against closed tricuspid valve)
    b. Regular → Junctional rhythm and with some ventricular and supraventricular tachycardias
d) *Giant V waves* → Tricuspid Regurgitation (due to tricuspid regurgitation, during ventricular contraction, some blood comes back to the atrium, so when the atrium is being filled, it will be filled with higher than normal amount → Giant V wave),
e) *CV Waves* → Tricuspid regurgitation (same mechanism of the previous one)
f) *Prominent Y descent* → Pericardial Effusion (The effusion applies and external pressure on the atrium while it is being emptied, increasing its pressure)

3-Kussmaul's Sign

a) Normally, the JVP is decreased during inspiration
b) But in some cases, as in pericardial constriction, the JVP is increased during inspiration
c) THE TABLE TO THE RIGHT IS IMPORTANT

4.10 Haemodynamic effects of respiration

	Inspiration	Expiration
Pulse/heart rate	Accelerates	Slows
Systolic blood pressure	Falls (up to 10 mmHg)	Rises
Jugular venous pressure	Falls	Rises
Second heart sound	Splits	Fuses

4-Causes of elevated JVP

a) HF → the abdominojugular reflux will be sustained for more than 10 seconds
b) Pulmonary embolism –
c) Pericardial Effusion
d) Pericardial Constriction
e) SVC obstruction → not pulsatile

Precordium

1-Apex Beat

a) Displaced → Enlarged Ventricles
b) Impalpable → Hyperinflated lungs and overweight patients
c) Palpable on the right side → Dextrocardia
d) Tapping Apex Beat → Mitral Stenosis
e) Double apical impulse → Hypertrophic Cardiomyopathy

2-Ventricular Heave

a) Left Parasternal Heave → RV Hypertrophy and Pulmonary HTN
b) Apical Heave → LV hypertrophy, Aortic Stenosis and systemic HTN

3-Thrills

a) Right Upper sternal → Aortic Stenosis
b) Right and Left sternal borders → VSD
c) Apex thrill → Mitral Regurgitation

Heart Sounds

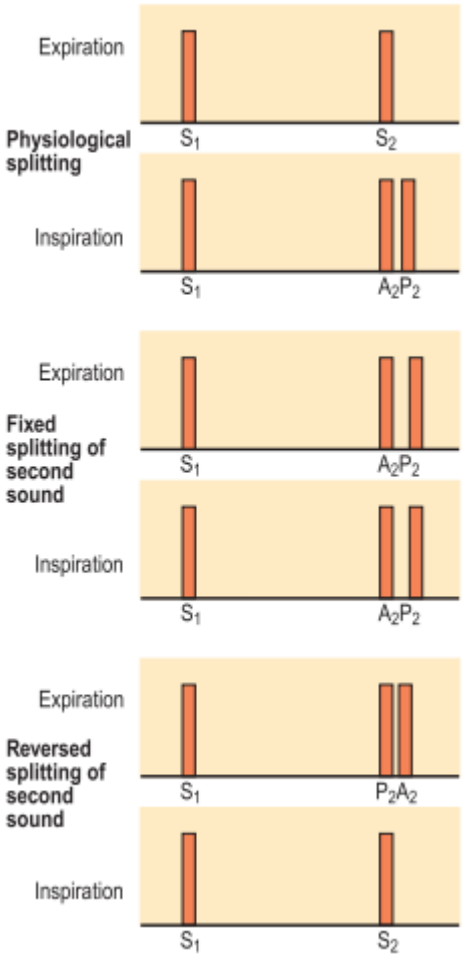
1-S1 Heart Sound

a) Marks the beginning of the systole
b) Due to closure of the mitral and tricuspid valve
c) Best heard at the apex
d) Quiet S1 heart sound is caused by (anything decreases the turbulence of blood around the closed mitral and tricuspid valves)
    a. Low CO (as in HF)
    b. Poor ventricular function
    c. Mitral Regurgitation
    d. Long PR intervals
e) Loud S1 is due to (Anything increases the turbulence of blood around the closed mitral and tricuspid valves)
    a. Increased CO
    b. Large SV
    c. Mitral Stenosis
    d. Short PR interval
    e. Atrial Myxoma
f) Variable S1 is due to
    a. A Fib
    b. Complete heart block
    c. Extrasystoles

2-S2 Heart sound

a) Marks the end of the systole
b) Due to closure of the Aortic and Pulmonic Valves

c) Best heard at the left sternal angle
d) Splitting of S2
    a. Remember, the S2 has two components, an aortic and pulmonic, normally the closure of the aortic valve precedes the closure of the pulmonic valve
    b. Anything increases the time needed for the pulmonic valve to close increases the splitting
    c. Physiological Splitting
        i. The S2 splitting is increased during inspiration
        ii. Inspiration increases the venous return to the heart → Increases the preload in the right ventricle → Increases the time required for the right ventricle to eject all the extra blood → Delayed closure of the pulmonic valve → Increased splitting



    d. Wide Splitting
        i. The S2 splitting is increased even with expiration, and inspiration causes further increase in the splitting
        ii. Seen in conditions that causes delay in the RV emptying such as
            1. Pulmonic Stenosis
            2. Right Bundle Branch Block → Decreased transmission velocity of the electrical signals in the right bundle branch that innervates the right ventricle → Delayed contraction of the right ventricle
            3. Pulmonary Hypertension
            4. VSD
    e. Fixed Splitting
        i. S2 Splitting that is not altered by inspiration
        ii. Heard in ASD → Blood moves from the left to the right atrium continuously → Increased blood in the right atrium and ventricle → Continuous delay in RV emptying
    f. Reversed Splitting
        i. S2 splitting in which the pulmonic component precedes the aortic components, and the splitting is decreased by inspiration
        ii. Caused in conditions that cause delay in the LV emptying
            1. Aortic Stenosis
            2. Left Bundle Branch Block
            3. Hypertrophic Cardiomyopathy
            4. Ventricular Pacing
e) Quiet S2 is heard in case of
    a. Low CO
    b. Aortic Stenosis
    c. Aortic Regurgitation
f) Loud S2 is heard in case of
    a. Systemic Hypertension (loud aortic component)
    b. Pulmonary Hypertension (loud pulmonic component)

3-S3 Heart Sound (S3)

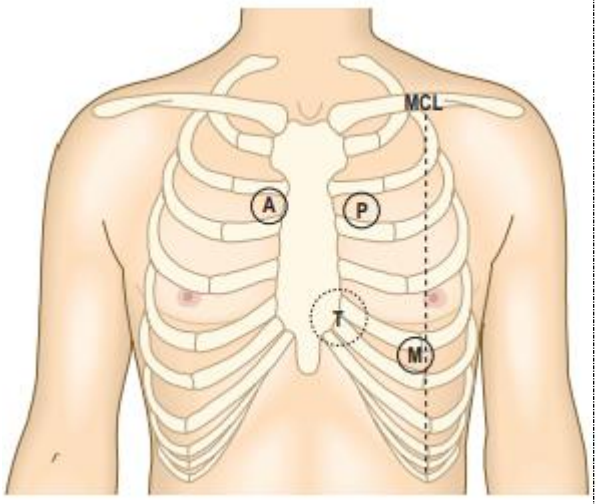
a) Early Diastolic Heart sound (low pitched)
b) Due to rapid ventricular filling after opening of the mitral and the tricuspid valves
c) Normal in children, young adults, pregnant woman and febrile patient
d) Pathological after the age of 40, seen in
    a. Left Ventricular Failure
    b. Mitral Regurgitation
e) If it was associated with tachycardia, it is termed as (S3 gallop), and may indicate Heart failure

4-S4 Heart sound

a) Late Diastolic heart sound (low pitched)
b) Best heard at the apex using the bells
c) Due to forceful atrial contraction against stiff ventricle
d) Always pathological and seen in
    a. Systemic Hypertension
    b. Aortic Stenosis
    c. Hypertrophic Cardiomyopathy
e) Lost in atrial fibrillation
f) S4 + Tachycardia → S4 Gallop
g) S3+S4+Tachycardia → Summation Gallop

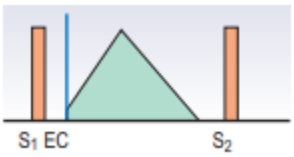
Heart Murmur

1-Approch to heart murmurs

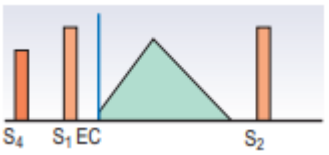


a) Each murmur is best heard at certain location in the precordium, depending on the affected valve
    a. Mitral Murmurs → Apex
    b. Tricuspid Murmur → Left lower parasternal edge
    c. Pulmonic Murmurs → Left upper parasternal edge
    d. Aortic Murmurs → Right upper parasternal Edge
b) The two main types of murmurs
    a. Stenosis → Abnormality in valve opening
    b. Regurgitation → Abnormality in valve closure
    c. How to determine if the murmur is systolic or diastolic During systole
        i. the aortic valve and the pulmonic valve must be open, abnormalities in their opening (Aortic/Pulmonic Stenosis) leads to systolic murmurs
        ii. The mitral and the Tricuspid valves must be closed, abnormalities in their closure (Mitral/Tricuspid Regurgitation) → Leads to systolic murmurs
    d. During Diastole
        i. The aortic and the pulmonic valves must be closed, abnormalities in their closure (Aortic/Pulmonic Stenosis) leads diastolic murmurs
        ii. The mitral and the tricuspid valves must be open, abnormalities in their opening (Mitral/Tricuspid Regurgitation) → Leads to diastolic murmurs

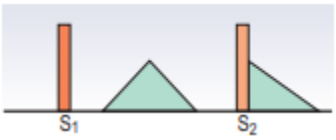
2 -*Aortic Stenosis*



a) Systolic Ejection murmur
b) Harsh, high pitched, musical
c) Ejection click is presents
d) Heard in all the systole, in a crescendo-decrescendo manner → Increased then decreasing in intensity
e) Common with advanced age
f) Associated with Pulsus Parvus et tardus
g) May radiate to the carotids

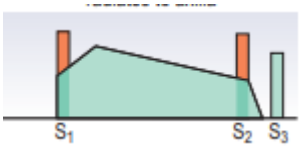


3-*Aortic Regurgitation*



a) Early Diastolic Murmur
b) Due to dilation of the root of the aorta, as in rheumatic fever
c) Usually, a systolic murmur is also present, due to increased flow in the aortic valve

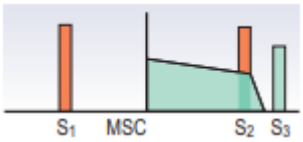
4-*Mitral Regurgitation*



a) Holosystolic murmur
b) Radiates to the axilla .

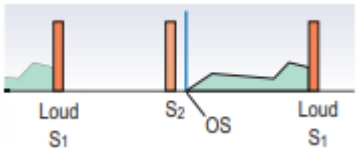
5-*Mitral Valve Prolapse*

a) Late Systolic Murmur
b) Midsystolic Click is present → Due to sudden tensing of chordae tendineae when the mitral leaflet prolapses into the left atrium



6-*Mitral Stenosis*

a) Early Diastolic Murmur
b) Opening snap is present → Due to sudden opening of the mitral valve during diastole
c) Associated with rheumatic fever

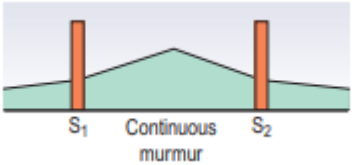


6-*VSD*

a) Holosystolic (Pansystolic)Harsh murmur

8-*PDA*

a) Patent Ductus Arteriosus → Opening between the aorta and the pulmonary artery
b) continuous (Systolic-Diastolic Murmur)
c) Best heard at the upper left sternal border
d) Radiates to the scapula



4-Pulmonary Stenosis

a) Systolic Ejection murmur
b) Presents with Ejection Clicks

10-*Metalic Heart sounds*

a) Mechanical Mitral Valve
    a. Metallic S1
    b. Loud Opening Snap
b) Mechanical Aortic Valve
    a. Ejection click
    b. Flow murmur

11-*Pericardial Friction Rub*

a) Best heard using the diaphragm with the patient holding their breath in expiration
b) Heard in acute pericarditis, extensive MI
c) Pneumopericardium Rub → Pericardial rub influenced by inspiration
d) Pneumopericardium → Gas in the pericardium

12-*ASD*

a) Ejection systolic murmur (Pulmonary flow murmur)
b) Diastolic Murmur (Tricuspid flow murmur)
c) Associated with Down Syndrome

12-Intensity of Heart Murmur

a) Does not correlate with the severity of the of the valve dysfunction , Intensity changes indicate progression of the disease ,Rapidly changing murmurs → Infective endocarditis
b) Grades of Heart murmurs
    a. Grade I →Heard by an expert in optimum conditions
    b. Grade II → Heard by non-expert in optimum conditions
    c. Grade III → Easily hear with no thrill
    d. Grade IV → Loud murmur with thrill
    e. Grade V → Very loud, heard over a wide area, with thrill
    f. Grade VI → Extremely loud, heard without stethoscope