

Medicine for 4th stage

CVS & Respiratory

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#Cough

- Duration
- Onset
- During day or night
- Severity
- Pattern
- Dry or productive (Associated with sputum or not)
- Frequency
- Odor of breathing
- Associated symptoms (hemoptysis, breathlessness, fever, chest pain, weight loss)

Examples on cough: للاطلاع

- Bad breathing odor → in bronchiectasis - infection
- Dry cough at night (that disrupting sleep) → typical of asthma
- Productive cough in morning → in COPD
- Prolonged wheezy cough → severe asthma and COPD
- Cough on rising in the morning → could be rhinosinusitis and post-nasal drip
- Paroxysmal cough → in pertussis
- Several months of Paroxysmal dry cough after viral infection → bronchial hyperreactivity
- Bovine cough with hoarseness → in lung cancer or muscle weakness due to neuromuscular disorder
- Harsh, barking or painful cough + hoarseness + strider → laryngeal inflammation or infection or tumor
- Persistent moist (smoker's cough) → typical of chronic bronchitis
- Dry, centrally painful and non-productive cough → Tracheitis and pneumonia
- Chronic dry cough → in interstitial lung disease like idiopathic pulmonary fibrosis
- Coughing during and after swallowing liquids → in neuromuscular disease of oropharynx
- Cough syncope → result from raised intra-thoracic pressure
- cough on lying down in the evening → due to gastro-esophageal reflux
- chronic productive cough + hypertension → ACEI, B-blockers

#Sputum

- Color
- Amount
- Odor and taste
- Day or night
- Onset and Duration
- Content and Consistency
- Mixed with mucus, blood
- Type of sputum
- Presence of solid material (food, teeth, tablets)

Examples on sputum: للاطلاع

- Clear or mucoid sputum → in chronic bronchitis and COPD
- Yellow sputum → in acute lower respiratory tract infection and asthma
- Green purulent sputum (dead neutrophils) → in COPD or bronchiectasis
- Rusty red sputum → in early pneumococcal pneumonia (cause lysis of RBC)
- Large volumes of purulent sputum that varies with posture → bronchiectasis
- Suddenly coughing up large amounts of purulent sputum on single occasion suggests → rupture of lung abscess or empyema into the bronchial tree
- Large volume of watery sputum & dyspnea → pulmonary edema
- Large volume of watery sputum & dyspnea (over weeks) → suggests alveolar cell cancer
- Foul-tasting or smelling sputum → anaerobic bacterial infection, bronchiectasis, lung abscess, empyema
- Worm like solid material in the sputum → asthma, allergic Broncho-pulmonary aspergillosis

#Hemoptysis

- Amount
- Appearance
- Presence of clots
- Color and odor
- Mixed with mucus or purulent secretions
- Duration
- Frequency
- Associated symptoms (chest pain, dyspnea, others)

Examples on hemoptysis: للاطلاع

- Blood-streaked clear sputum more than week → lung cancer



7.3 Types of sputum

Type	Appearance	Cause
Serous	Clear, watery Frothy, pink	Acute pulmonary oedema Alveolar cell cancer
Mucoid	Clear, grey White, viscid	Chronic bronchitis/chronic obstructive pulmonary disease Asthma
Purulent	Yellow Green	Acute bronchopulmonary infection Asthma (eosinophils) Longer-standing infection Pneumonia Bronchiectasis Cystic fibrosis Lung abscess
Rusty	Rusty red	Pneumococcal pneumonia

- Clots in sputum more than week → lung cancer
- Daily hemoptysis more than week → lung cancer, TB, lung abscess
- Hemoptysis with purulent sputum → suggest infection
- Coughing up large amount of pure blood → lung cancer, bronchiectasis, TB, lung abscess, cystic fibrosis, aortobronchial fistula, granulomatosis, polyangitis
- Intermittent hemoptysis + copious purulent sputum → bronchiectasis
- Single episodes of hemoptysis associated with pleuritic chest pain and dyspnea → pulmonary thromboembolism and infarction

#Breathlessness

- Onset
- Duration
- Progression
- Pattern
- Severity
- Related to position
- Related to activity
- Aggravated factors
- Relieving factors
- Presence of:
 - Orthopnea → breathlessness when lying flat
 - Platypnea → breathlessness when sitting up
 - Trepopnea → breathlessness when lying on one side
 - Paroxysmal nocturnal dyspnea → breathlessness that wakes patient from sleep
- Associated symptoms (chest pain, cough, wheeze)

Examples on breathlessness: للاطلاع

- Psychogenic breathlessness → occur suddenly at rest or while talking
- Orthopnea → occur in left ventricular failure, respiratory muscle weakness, large plural effusion, massive ascites, morbid obesity, any severe lung disease
- Platypnea → right to left shunting
- Trepopnea → in unilateral lung disease, dilated cardiomyopathy, tumors
- Paroxysmal nocturnal dyspnea → typical of asthma and left ventricular failure
- Dyspnea on exercise → in heart failure and exercise induced asthma
- Breathlessness worse on waking and may improve after coughing up sputum is typical of → COPD
- Breathlessness continues to worsen 5-10 min after stopping the activity → in exercise induced asthma
- Breathlessness improving at weekends or holidays → in occupational asthma

- Breathlessness may be caused by myocardial ischemia and is known as angina equivalent
- Breathlessness occurs in heart failure and it is associated with fatigue
- Breathlessness within minutes to hours → pneumothorax, foreign body, pulmonary embolism
- Breathlessness within hours to days → pneumonia, COPD, infections, pulmonary edema
- Breathlessness within weeks to months → asthma, chronic infection, pleural effusion, malignancy, pulmonary fibrosis
- Very important tables:

 7.7 Medical Research Council (MRC) breathlessness scale	
Grade 1	Breathless when hurrying on the level or walking up a slight hill
Grade 2	Breathlessness when walking with people of own age or on level ground
Grade 3	Walks slower than peers, or stops when walking on the flat at own pace
Grade 4	Stops after walking 100 metres, or a few minutes, on the level
Grade 5	Too breathless to leave the house
(Grade 5b)	Too breathless to wash or dress

 6.5 New York Heart Association classification of heart failure symptom severity	
Class I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitation (asymptomatic left ventricular dysfunction)
Class II	Slight limitation of physical activity. Such patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (symptomatically 'mild' heart failure)
Class III	Marked limitation of physical activity. Less than ordinary physical activity will lead to symptoms (symptomatically 'moderate' heart failure)
Class IV	Symptoms of congestive heart failure are present, even at rest. With any physical activity increased discomfort is experienced (symptomatically 'severe' heart failure)

#Chest pain ((SOCRATES – SIROD CASP))

- Site
- Onset
- Character (colicky, stabbing, constricting, dull, ...)
- Radiation
- Associated symptoms (sweating, vomiting, breathlessness, others)
- Timing (duration, course, pattern, special time of occurrence)
- Exacerbating (exertion, special food, hunger) and relieving factors (rest, meal, drugs)
- Severity
- Frequency
- Previous episodes

	Ischemic cardiac chest pain	Non-cardiac chest pain
Location	Central, diffuse	Peripheral, localised
Radiation	Jaw/neck/shoulder/arm occasionally back)	Other or no radiation
Character	Tight, squeezing, choking	Sharp, stabbing, catching
Precipitation	Precipitated by exertion and/or emotion	Spontaneous, not related to exertion, provoked by posture, respiration or palpation
Reliving factors	Rest Quick response to nitrates	Not relieved by rest Slow or no response to nitrates
Associated features	Breathlessness	Respiratory, astrointestinal, locomotor or psychological

Note: chest pain less than 30 min in angina / more than 30 min in MI

#Edema

- Onset
- Duration
- Site ((Unilateral, bilateral, generalized, periorbital, abdomen, leg, sacrum, scrotum))
- Associated with dyspnea, orthopnea, Paroxysmal nocturnal dyspnea
- Weight change
- Start from which area ((ascending upward in heart failure, descending downward in renal failure))
- The cause ((renal, cardiac, hepatic, venous, lymphatic))

#Respiratory sounds

- Onset
- Duration
- Special time of occurrence
- Frequency

- Aggravated factor: Related to activity
- Reliving factor: rest
- Nature and type of sound
 - Dysphonia (hoarseness) → in laryngitis, lung cancer
 - Wheeze (rhonchi) (during expiration) → in asthma, COPD ((precipitant factors of wheezing are: allergens, exercise, occupation))
 - Strider → inspiratory stridor indicates narrowing at the vocal cord, biphasic strider suggests tracheal obstruction, strider on expiration suggests tracheobronchial obstruction
Causes of strider: acute epiglottitis, tumor of the trachea or main bronchus, extrinsic compression by L.N, anaphylaxis, foreign body.
 - Stertor → it is muffled speech occurs with naso or oropharyngeal blockage

#Respiratory pattern

- Change of rate or pattern of breathing
- If patient has daytime sleepiness, ask the patient's bed partner about:
 - Apnea
 - Loud snoring
 - Nocturnal restlessness
 - Irritability
 - Personality change

#Palpitation

- The mode of onset and termination
- Duration of attacks
- Frequency
- Continuous or intermittent
- Rhythm (ask patient to tap out), Description (thumping, pounding, fluttering, jumping, racing, skipping)
- Aggravating factor (exercise, alcohol, caffeine, drugs)
- Reliving factor
- Any associated symptoms
- History of organic heart diseases

#Syncope

- Time of occurring
- Number of syncopes
- Duration of syncope
- Triggers
- History of cardiovascular disease or neurocardiogenic disease
- History of drugs like vasodilators
- Ask about recent intense emotional stimuli or warm environment
- Previous history of syncope
- Associated symptoms (lightheadness, tinnitus, nausea, sweating)

#Cyanosis

- Onset
- Duration
- Central or peripheral
- Localized or generalized
- Which area is affected
- Medication like beta-blockers
- Smoking
- Associated symptoms (dyspnea, strider, polycythemia)

Ask about smoking: because smoking aggravates Raynaud's phenomenon and peripheral vascular disease that can cause peripheral cyanosis. Cigarette smoking can also cause chronic bronchitis and emphysema which can cause central cyanosis.

 **2.20 Calculating pack years of smoking**

20 cigarettes = 1 packet

$$\frac{\text{Number of cigarettes smoked per day} \times \text{Number of years smoking}}{20}$$

For example, a smoker of 10 cigarettes a day who has smoked for 15 years would have smoked:

$$\frac{10 \times 15}{20} = 7.5 \text{ pack years}$$

Above 20 pack years → increase the risk of COPD

Above 40 pack years → increase the risk of malignancy

Inspection

#General inspection

- Age – gender – body mass
- General appearance: does the patient look healthy, unwell, or ill?
- Conscious state: reduced in type 2 respiratory failure
- Position: the patient should lie on one pillow and his shoulder outside the pillow and according to the presence or absence of orthopnea
- Oxygen mask - IV fluid
- Evidence of other respiratory symptoms like cough and audible wheeze and cyanosis
- Check the temperature of the patient (associated with respiratory infection)
- Any indications of recent weight loss like sunken cheeks and other signs

Note:

- During normal (resting) respiration: women are predominantly thoracic respiration but men are predominantly abdominal respiration
- In severe respiratory failure or bilateral phrenic nerve lesions causing diaphragmatic palsy, the abdomen and chest move paradoxically; during inspiration the abdomen moves inwards as the chest wall moves out.

#Face

- General appearance: Cushing's face result from long-term use of steroids.
- Central cyanosis (lips, buccal mucus membrane, tongue)
- Anemia (conjunctiva, face color) , polycythemia
- Horner's syndrome (possible apical lung cancer) = meiosis + partial ptosis + loss of hemifacial sweating

#Neck

- Jugular venous pressure JVP raised in:
 - Cor pulmonale ((Chronic hypoxia in COPD leads to pulmonary arterial vasoconstriction, pulmonary hypertension, right heart dilatation and peripheral edema with elevation of the JVP. This is cor pulmonale))
 - Tension pneumothorax

- Severe acute asthma
- Massive pulmonary embolism
- Right heart failure
- Evidence of Superior vena cava obstruction SVCO:
 - The JVP is raised and non-pulsatile, and the abdominojugular reflex is absent, Facial flushing, distension of neck veins and stridor can occur in SVCO when the arms are raised above the head
 - Causes of SVCO are: lung cancer compressing the superior vena cava, lymphoma, thymoma, mediastinal fibrosis
- Goiter: any possible tracheal obstruction
- Lymphadenopathy ((cervical, supra-clavicular, scalene lymph nodes)):
 - Scalene lymph node enlargement → may be the first evidence of metastatic lung cancer
 - Localized cervical lymphadenopathy → is a common presenting feature of lymphoma.
 - Rubbery L.N → In Hodgkin's disease
 - Tender L.N → in dental sepsis and tonsillitis
 - Matted L.N together to form a mass → in tuberculosis and metastatic cancer
 - Calcified lymph nodes feel stony hard → in malignancy
 - Fixed L.N to deep structures or skin → are usually malignant

#Hand

- Finger clubbing → lung cancer, bronchiectasis, interstitial lung disease, empyema, lung abscess, idiopathic pulmonary fibrosis
- Cyanosis → B-blockers, Raynaud syndrome, vasoconstriction, heart failure
- Tobacco staining → caused by tar not nicotine
- Yellow nail syndrome → associated with lymphedema and exudative pleural effusion
- Tremor → fine tremor due to anxiety or B-agonist, flapping tremor (asterixis) due to respiratory failure type 2
- Hypertrophic pulmonary osteoarthropathy → in lung cancer (squamous cancer)
- Pulse and blood pressure:
 - Tachycardia: in significant respiratory difficulty or overuse of B-agonist
 - Atrial fibrillation: in lung cancer
 - Pulsus paradoxus: in large pneumothorax or tension pneumothorax
 - Fall in pulse volume + systolic pressure >10 mmHg during inspiration: in cardiac tamponade
 - Diastolic pressure <60 mmHg: in community acquired pneumonia
 - Hypotension: in pneumothorax

#Lower limbs

- Swollen calf: possible DVT (unilateral edema) cor pulmonale (bilateral edema)

- Peripheral edema: lower legs if ambulant or sacral if bed-bound
- Erythema nodosum over the shin: in acute sarcoidosis and tuberculosis
- Raised, firm, non-tender subcutaneous nodules: in disseminated cancer

#Chest

- Respiratory rate: normal for adult is about 14/min
- Operation scars
- subcutaneous lesions: metastatic tumor nodules, neurofibromas, lipomas
- Vascular anomalies: dilated venous vascular channels of SVCO
- Paradoxical chest movement: may indicate a fractured rib
- Evidence of respiratory distress at rest or when walking like:
 - Obvious breathlessness
 - Talking in short phrases rather than full sentences
 - Use of accessory muscles (in asthma and COPD) (sternocleidomastoid, platysma, trapezius muscles are accessory muscles of respiration)
 - Exhalation with pursed lips (in severe COPD)
- Nature of breathing:
 - Kussmaul's breathing: deep and labored breathing, occur in severe metabolic acidosis
 - Cheyne-Stokes' breathing: progressively deeper breathing followed by temporary apnea, occur in heart failure and cerebrovascular disease and head injury and carbon monoxide poisoning and brain tumors and may be a normal variant during sleep or at high altitude
- Chest shape:
 - Overinflated (barrel shape) antero-posterior diameter greater than lateral diameter: in COPD or severe acute asthma
 - Asymmetry: the abnormality is on the side that moves less, occur in pneumothorax, collapse, consolidation, effusion
 - Pigeon chest (Pectus carinatum): in poorly controlled childhood asthma, rickets, osteomalacia ((Note: Harrison's sulci occur with Pectus carinatum))
 - Funnel chest (Pectus excavatum): usually asymptomatic but in severe cases the heart is displaced to the left and the ventilatory capacity is reduced
 - Kyphosis: is an exaggerated anterior curvature of the spine
 - Scoliosis: is an exaggerated lateral curvature of the spine
 - Both Kyphosis and Scoliosis are may be idiopathic or secondary to childhood poliomyelitis or spinal tuberculosis, and may be grossly disfiguring and disabling. It may reduce ventilatory capacity and increase the work of breathing. These patients develop progressive ventilatory failure with carbon dioxide retention and cor pulmonale at an early age.
- Respiratory sounds:
 - Dysphonia (hoarseness) → in laryngitis, lung cancer

- Wheeze (rhonchi) (during expiration) → in asthma, COPD
- Strider → in acute epiglottitis, tumor of the trachea or main bronchus, extrinsic compression by L.N, anaphylaxis, foreign body.
- Stertor → it is muffled speech occurs with naso or oropharyngeal blockage

Palpation

#Tracheal deviation

- Use the index finger to feel the trachea and to determine whether the trachea feels central or deviated ((normally the trachea is slightly deviated to the right side))
- Measure the distance between the suprasternal notch and cricoid cartilage, normally 3-4 finger breadths; any less suggests lung hyperinflation.
- The trachea is deviated away from → Tension pneumothorax, plural effusion
- The trachea is deviated towards → collapse, consolidation, fibrosis, pneumonectomy
- The trachea may also be deviated by → lymphoma, lung cancer, retrosternal goiter
- Shift of the upper mediastinum → causes tracheal deviation.
- Shift of the lower mediastinum → causes displacement of the cardiac apex beat
- Displacement of the cardiac impulse without tracheal deviation due to →
 - Left ventricular enlargement
 - Scoliosis
 - Kyphoscoliosis
 - Severe pectus excavatum
- Tracheal tug → is found in severe hyperinflation; resting on the patient's trachea, your fingers move inferiorly with each inspiration.

#Apex beat

- It is the most lateral and inferior position where the cardiac impulse can be felt
- It will be displaced if the mediastinum is displaced or distorted

#Chest expansion

- Usual chest expansion in an adult should be symmetrical and at least 5cm
- symmetrical reduction of the chest expansion:
 - overinflated lungs: bronchial asthma, COPD, emphysema
 - stiff lungs: pulmonary fibrosis
 - ankylosing spondylitis
- Asymmetrical reduction of the chest expansion:
 - absent expansion: empyema, pleural effusion
 - reduced expansion: pulmonary consolidation, collapse

#Tactile vocal fremitus

- Use the ulnar side of the hand while the patient say "ninety-nine"
- Tactile vocal fremitus increased → over areas of consolidation
- Tactile vocal fremitus decreased or absent → over areas of effusion or collapse

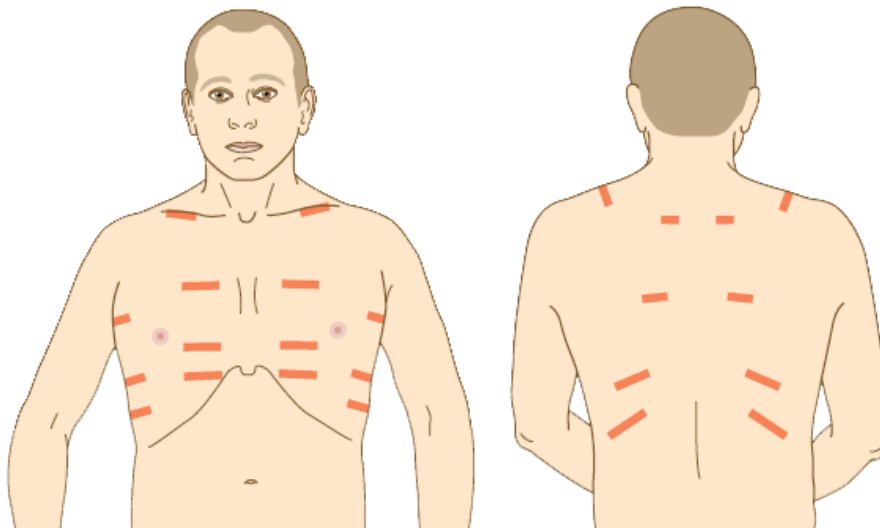
#Axillary L.N examination

#Other findings

- Paradoxical inward movement → in diaphragmatic paralysis, severe COPD
- Chest wall between the fractures become mobile or 'flail' → in double fracture of a series of ribs or of the sternum
- Crackling sensation over gas-containing tissue → in subcutaneous emphysema
- Systolic 'crunching' sound on auscultating the precordium (**Hamman's sign**) → in mediastinal emphysema
- Tenderness over the costal cartilages → in the costochondritis of Tietze's syndrome
- Localized rib tenderness → found over areas of pulmonary infarction or fracture

Percussion

- Resonant → Normal lung
- Hyper-resonant → Pneumothorax
- Dull → over solid structures like: Pulmonary consolidation, Pulmonary collapse, Severe pulmonary fibrosis, lung pneumonia, over the heart, over the liver
- Stony dull → over fluid like: Pleural effusion, Haemothorax
- Hepatic dullness → in adult is over the fifth rib in the mid-clavicular line. Resonance below this is a sign of hyperinflation (COPD or severe asthma)
- Cardiac dullness → over the left anterior chest may be decreased when the lungs are hyperinflated.
- Basal dullness → due to elevation of the diaphragm is easily confused with pleural fluid



Auscultation

#Introduction

- Most sounds reaching the chest wall are low-frequency and best heard with the stethoscope bell. The diaphragm locates higher-pitched sounds, such as pleural friction rubs.
- Stretching the skin and hairs under the diaphragm during deep breathing can produce anomalous noises like crackles, and in thin patients it may be difficult to apply the diaphragm fully to the chest wall skin.
- Listen with the patient relaxed and breathing deeply through his open mouth.
- Avoid asking him to breathe deeply for prolonged periods, as this causes giddiness and even tetany.
- Avoid auscultation within 3 cm of the midline anteriorly or posteriorly, as these areas may transmit sounds directly from the trachea or main bronchi.
- Listen:
 - Anteriorly from above the clavicle down to the 6th rib
 - Laterally from the axilla to the 8th rib
 - Posteriorly down to the level of the 11th rib

#Heart auscultation: Severe lung disease cause → pulmonary hypertension and loud S2

#Normal breath sounds

- Are called vesicular
- Described as quiet and gentle
- Usually there is no gap between the inspiratory and expiratory phase sounds
- The inspiration is longer than expiration
- Causes of diminished vesicular breathing:
 - Reduced conduction:
 - Obesity/thick chest wall
 - Pleural effusion or thickening
 - Pneumothorax
 - Reduced airflow
 - Generalized: in COPD
 - Localized: in collapsed lung due to occluding lung cancer
- If breath sounds appear reduced, ask the patient to cough. If the reduced breath sounds are due to bronchial obstruction by secretions, they are likely to become more audible after coughing.

#Bronchial breathing

- Abnormal sound that generated by turbulent air flow in large airways
- Similar sounds can be heard in healthy patients by listening over the trachea

- Sounds are harsh and poor in nature (hollow or blowing quality)
- There is gap between the inspiratory and expiratory phase sounds
- The inspiratory phase is of similar length and intensity of expiratory phase
- Causes:
 - Common: Lung consolidation (pneumonia)
 - Uncommon: Localized pulmonary fibrosis, at the top of a pleural effusion, Collapsed lung (where the underlying major bronchus is patent)

#Rhonchi (wheezes)

- Musical sound heard on expiration (in severe cases they may be both inspiratory and expiratory)
- It imply narrowing of the airways
- The loudness of rhonchi gives no indication of the severity of the condition
- Causes of rhonchi → asthma, COPD
- Precipitant factors of rhonchi are → allergens, exercise, occupation
- In severe airways obstruction wheeze may be absent because of reduced airflow, producing a '**silent chest**'
- Localized rhonchi → occur in fixed bronchial obstruction (due to lung cancer) and it does not clear on coughing.

#Rales (crackles or crepitation)

- Probably represent opening of small airways and alveoli
- They may be normal at lung bases if they clear on coughing or after taking a few deep breaths
- Fine basal crepitation are feature of → pulmonary congestion with left ventricular failure and resolving pneumonia, they may be more diffuse in pulmonary fibrosis
- Course crepitation may be heard in → bronchiectasis
- Causes of crackles:
 - In early inspiratory phase: Small airways disease, as in bronchiolitis
 - In middle inspiratory phase: Pulmonary edema
 - In late inspiratory phase: Pulmonary fibrosis (fine), pulmonary edema (medium), Bronchial secretions in COPD, pneumonia, lung abscess, tubercular lung cavities (coarse)
 - In Biphasic: Bronchiectasis (coarse)

#Pleural friction rub

- Creaking sound
- Caused by stiff pleural membranes such as with pleurisy and associated with pain
- Heard over areas of inflamed pleura in pulmonary infarction due to pulmonary embolism and in pneumonia or pulmonary vasculitis

- Pleural friction rubs disappear if an effusion separates the pleural surfaces
- Pleuro-pericardial friction rub: heard if the pleura adjacent to the pericardium is involved

#Vocal resonance

- Place the stethoscope at various levels and ask the patient to whisper "ninety-nine"
- The sound is muffled → over normal lung
- The sound is increased → if there is consolidation
- The sound is decreased or absent → if there is effusion or collapse

#Whispering pectoriloquy

- Is elicited as for vocal fremitus but ask the patient to whisper "one, two, three"
- It is the increased quality and loudness of whispers that are heard with a stethoscope over an area of pulmonary consolidation (pneumonia), at the top of a pleural effusion, over areas of dense fibrosis

#Aegophony

- Is a bleating or nasal sound
- Heard over consolidated lung (pneumonia) or at the upper level of a pleural effusion.
- It is due to enhanced transmission of high-frequency noise across abnormal lung, with lower frequencies filtered out

#Stridor

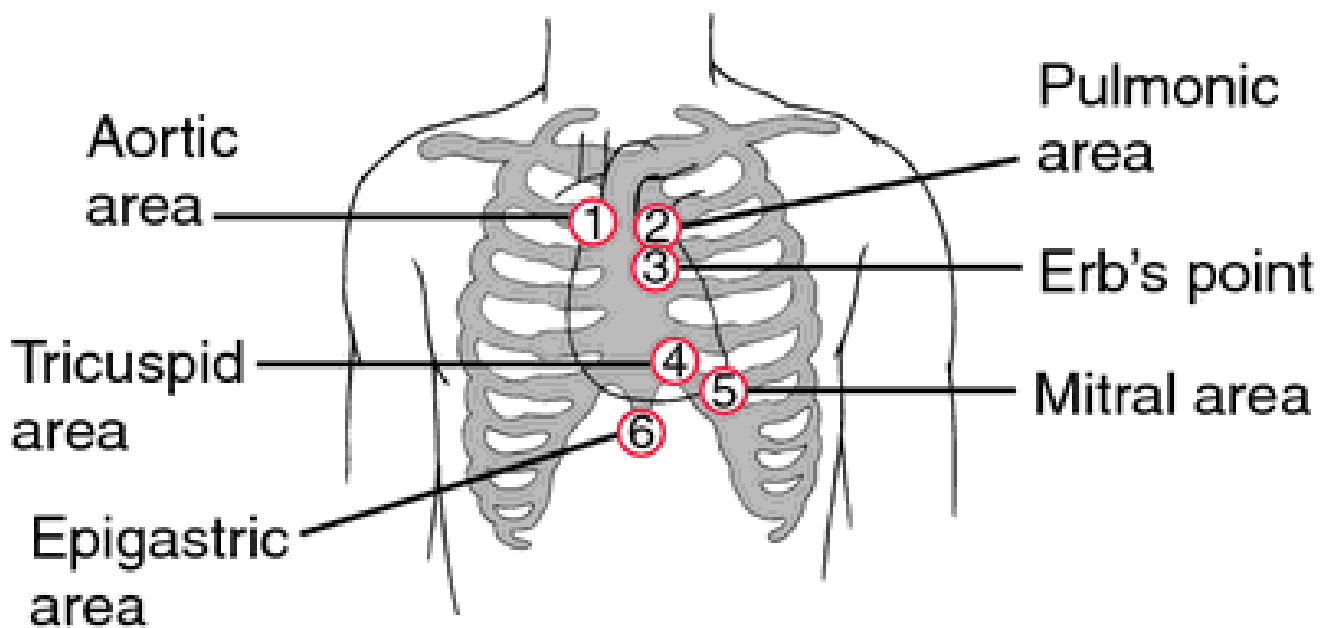
- Inspiratory stridor → suggests narrowing at the vocal cord
- Biphasic stridor → suggests tracheal obstruction
- Expiratory stridor → suggests tracheobronchial obstruction
- Causes of stridor:
 - Acute epiglottitis
 - Tumor of the trachea or main bronchus
 - Extrinsic compression by L.N, anaphylaxis, foreign body.

#Pneumothorax click

- Is a rhythmical sound, synchronous with cardiac systole, and produced when there is air between the two layers of pleura overlying the heart

#Mid-expiratory 'squeak'

- Is characteristic of obliterative bronchiolitis (a rare complication of rheumatoid arthritis) where small airways are narrowed or obliterated by chronic inflammation and fibrosis



The precordium: is the area on the front of the chest which relates to the surface anatomy of the heart.

- Pulmonary valve (to pulmonary trunk) → second intercostal space left upper sternal border
- Aortic valve (to aorta) → second intercostal space right upper sternal border
- Mitral valve (to left ventricle) → fifth intercostal space medial to left mid-clavicular line
- Tricuspid valve (to right ventricle) → fourth intercostal space lower left sternal border

Inspection

#Shape of the chest

- Pectus excavatum (funnel chest) → posterior displacement of the lower sternum
- Pectus carinatum (pigeon chest) → may displace the heart
- Bulging precordium → seen in huge cardiomyopathy

#Scars

- Midline sternotomy scar → in coronary artery bypass surgery or aortic valve replacement
- Left sub-mammary scar → in mitral valvotomy
- Infra-clavicular scar → seen after pacemaker or defibrillator implantation
- Scars due to valve replacement or mediastinal surgery or cabbage surgery

#Visible pulsation

- Visible apex beat: seen in thin patient and very forceful beat
- Epigastric beat: seen in thin patient, right atrial dilatation, right ventricular hypertrophy, abdominal aortic aneurysm
- Visible beat in the upper chest (neck) → in elevated JVP, aneurysm

Palpation

1-Palpation of apex beat

#Position of apex beat

- The most lateral and inferior position where the cardiac impulse can be felt
- Not palpable apex beat seen in → emphysema, pericardial effusion, poor left ventricular function, lung hyperinflation (asthma, COPD), obesity or muscular patient, dextrocardia, just below the rib
- Shift of the position of the apex beat → due to **cardiac** cause dilated left ventricle → shift the apex beat inferiorly and laterally (after MI, with aortic stenosis, severe hypertension, dilated cardiomyopathy) or **respiratory** cause (right lung effusion, right pneumothorax, left collapse)

#Character of apex beat

- Forceful apex beat (elevate the fingers) → there are two types as follow

Pressure overload	Volume overload
Not replace the apex beat	Replace the apex beat
Forceful and sustained	Forceful not sustained
Called heave apex or thrusting apex	Called hyper-dynamic apex
Causes: 1- Hypertension 2- Aortic stenosis 3- Hypertrophic cardiomyopathy like left ventricular hypertrophy	Causes: 1- aortic regurgitation 2- mitral regurgitation

- Tapping apex beat (just clicks, not elevate the fingers) → it is a palpable loud first heart sound S1 caused by mitral stenosis
- Double apical impulse → characteristic of hypertrophic cardiomyopathy

Note: Position of the apex beat → cardiac and respiratory causes // Character of the apex beat → cardiac causes only

2- Left parasternal heave

- Heave is a palpable impulse that noticeably lifts your hand
- Normally → no pulse felt on left parasternal area

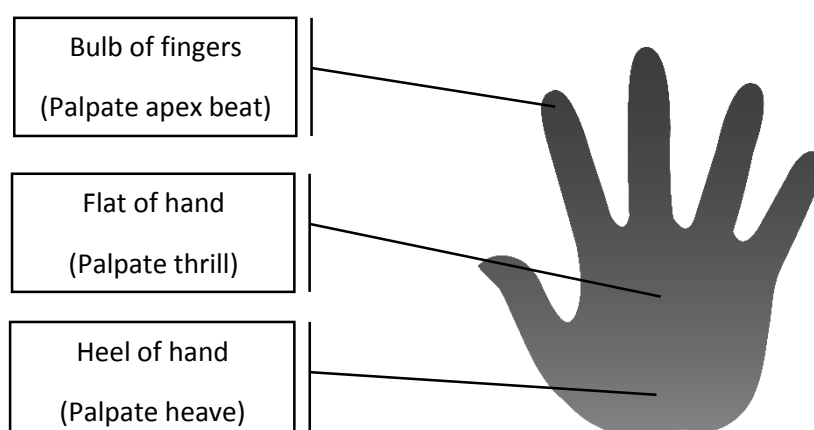
- Abnormally → felt due to:
 - Dilated cardiomyopathy (right ventricular dilatation)
 - Increase pulmonary hypertension (so right ventricle dilated)
 - Right ventricular hypertrophy

3- Thrill

- It is the tactile equivalent of a murmur and is a palpable vibration
- Normally not present
- Systolic thrill:
 - Due to systolic murmur
 - Between S1 and S2
 - With the carotid pulse
 - With apex beat
- Diastolic thrill (very rare):
 - Due to diastolic murmur
 - Between S2 and S1
 - Not with the carotid pulse
 - Not with the apex beat
- To differentiate between systolic and diastolic thrill put your hand on the apex or carotid artery to feel the pulsation
- Thrill caused by aortic stenosis → palpable at the apex, lower neck or lower sternum
- Thrill caused by ventricular septal defect VSD → best felt at the left and right sternal edges

4- Beat in the base of the heart

- In the right second intercostal space
- Normally not present
- Due to: atrial septal defect ASD or pulmonary hypertension



Auscultation

#Normal sounds

- First heart sound S1 →
 - 'lub' is caused by closure of the mitral and tricuspid valves at the onset of ventricular systole. It is best heard at the apex
 - S1= M1 (mitral) + T1(tricuspid)
 - normally together but S1 is mainly due to M1
- Second heart sound S2 →
 - 'dup' is caused by closure of the pulmonary and aortic valves at the end of ventricular systole and is best heard at the left sternal edge. It is louder and higher-pitched than the S1 'lub', and the aortic component is normally louder than the pulmonary one
 - S2= A2 (aortic) + P2 (pulmonary)
 - normally A2 before P2 and P2 normally hear in athletics

Physiological splitting of S2 occurs because left ventricular contraction slightly precedes that of the right ventricle so that the aortic valve closes before the pulmonary valve. This splitting increases at end-inspiration because increased venous filling of the right ventricle further delays pulmonary valve closure. This separation disappears on expiration. Splitting of S2 is best heard at the left sternal edge. On auscultation, you hear 'lub d/dub' (inspiration) 'lub-dub' (expiration)

#Abnormal sounds

- Soft (muffled) S1 → in Rheumatic mitral regurgitation, low cardiac output, poor left ventricular function, long P-R interval in first degree heart block
- Loud S1 → in mitral stenosis, increased cardiac output, large stroke volume, Short P-R interval, atrial myxoma
- Variable S1 → in atrial fibrillation, extrasystoles, complete heart block
- Soft S2 →
 - quiet or absent in calcific aortic stenosis
 - reduced in aortic regurgitation and low cardiac output
- Loud S2 → in pulmonary hypertension increase P2 and in systemic hypertension increase A2
- Splitting S2 →
 - Widen in inspiration (enhanced physiological splitting) → right bundle branch block, pulmonary stenosis, pulmonary hypertension, Ventricular septal defect VSD
 - Fixed S2 splitting (unaffected by respiration) → atrial septal defect ASD
 - Widens in expiration (Reversed S2 splitting) → in left bundle branch block, right ventricular pacing, aortic stenosis, hypertrophic cardiomyopathy

- Third heart sound S3 →
 - Is a low-pitched early diastolic sound best heard with the bell at the apex.
 - It coincides with rapid ventricular filling immediately after opening of the atrioventricular valves and is therefore heard after the second as 'lub-dub-dum'.
 - It is a normal finding in healthy young adults, athletics, during pregnancy, in fever
 - Abnormally hear in heart failure (left ventricular failure → large, poorly contracting left ventricle) and mitral or aortic regurgitation
- Fourth heart sound S4 →
 - It is soft and low-pitched, best heard with the stethoscope bell at the apex. It occur just before S1 (da-lub-dub)
 - It is pathologic sound that heard in ischemic heart disease and left hypertrophic ventricles (due to hypertension or aortic stenosis or hypertrophic cardiomyopathy)
 - It cannot occur when there is atrial fibrillation

Third heart sound S3	Fourth heart sound S4
Ventricular sound	Atrial sound
In diastolic period	In diastolic period
Sometimes normal	Always abnormal

- Gallop rhythm →
 - It is quit S1 + quit S2 + S3 + Tachycardia (lub-da-dub)
 - Occur in heart failure
 - Note: both an S3 and an S4 cause a triple or gallop rhythm
- Ejection click →
 - Best heard by diaphragm, occur just after S1
 - Occur in congenital pulmonary or aortic stenosis in children and very rare in adult.
- Opening Snap →
 - Opening of the mitral valve give sound
 - Due to mitral stenosis (rarely tricuspid stenosis)
 - Occur just after S2
 - Best heard by the diaphragm at apex
- Mid systolic click →
 - Best heard by diaphragm at the apex
 - Occur in mitral valve prolapse
 - May be associated with late systolic murmur
- Mechanical heart sound →
 - It is loud metallic sound, could be heard without stethoscope, may be palpable
 - Occur due to valve replacement by mechanical valve
 - Mechanical mitral valve makes metallic S1 and a sound like loud opening snap
 - Mechanical aortic valve makes loud metallic S2 and an opening sound like an ejection click

- Pericardial rub (friction rub) →
 - Best heard using the diaphragm with the patient holding his breath in expiration
 - Audible over any part of precordium and it is localized
 - Heard in acute viral pericarditis and 24-72 hours after Myocardial infarction
 - It is vary in intensity with time and position of the patient
- Pleuro-pericardial rub →
 - Influenced by respiration, pleural in origin
 - A crunching noise can be heard caused by gas in the pericardium (pneumo-pericardium)

Notes:

- All of these sounds are without murmur
- In mitral stenosis there are = loud S1 + opening Snap + tapping apex beat
- Use the **diaphragm** of stethoscope to listen to → S1, S2, early diastolic murmur of aortic regurgitation, mechanical heart sound, click sound, pericardial friction rub
- Use the **bell** of the stethoscope to listen to → S3, S4, diastolic murmur of mitral stenosis

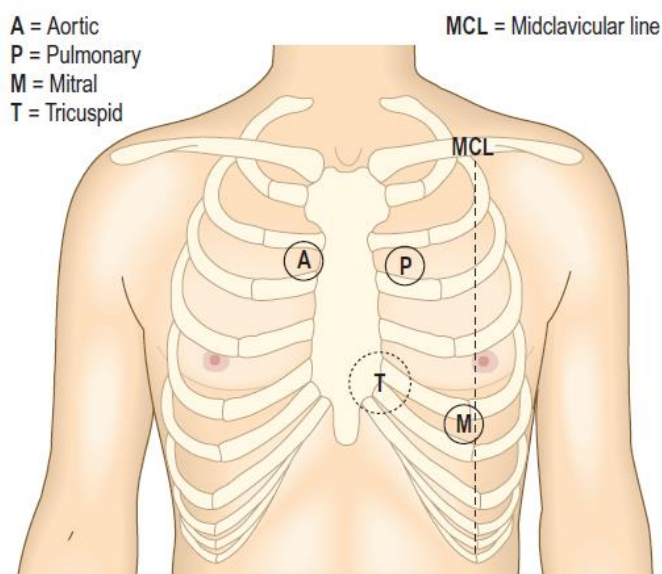


Fig. 6.21 Sites for auscultation. Sites at which murmurs from the relevant valves are usually, but not preferentially, heard.

6.25 Cardiac auscultation: the best sites for hearing abnormality	
Site	Sound
Cardiac apex	First heart sound Third and fourth heart sounds Mid-diastolic murmur of mitral stenosis
Lower left sternal border	Early diastolic murmurs of aortic and tricuspid regurgitation
Upper left sternal border	Second heart sound Opening snap of mitral stenosis Pulmonary valve murmurs Pansystolic murmur of ventricular septal defect
Upper right sternal border	Systolic ejection (outflow) murmurs, e.g. aortic stenosis, hypertrophic cardiomyopathy
Left axilla	Radiation of the pansystolic murmur of mitral regurgitation
Below left clavicle	Continuous 'machinery' murmur of a persistent patent ductus arteriosus

#Definition

It is a sound that produced by turbulent flow, murmur is continuous appear sound not like abnormal heart sounds that appear and disappear

#Causes

- Stenosis of heart valves
- Regurgitation of heart valves
- Congenital septal defects (ASD, VSD, PDA)
- Innocent murmurs caused by increased volume or velocity of flow through a normal valve occur when stroke volume is increased, e.g. during pregnancy, in athletes with resting bradycardia or children with fever.

#Grades of intensity of murmur

- Grade 1 → Heard by an expert in optimum conditions
- Grade 2 → Heard by a non-expert in optimum conditions
- Grade 3 → Easily heard; no thrill
- Grade 4 → A loud murmur, with a thrill
- Grade 5 → Very loud, often heard over wide area, with thrill
- Grade 6 → Extremely loud, heard without stethoscope

#Types

1- Mitral stenosis

- Type: Diastolic murmur
- Time: mid-diastolic murmur
- Best location to hear: at the apex (mitral area)
- Duration: hear after S2 (not immediately after S2 but after it by short time)
- Character: thunder رعد or rumbling قرقرة
- Radiation: no radiation
- Intensity (grade): depend on the patient status
- Note: cause left ventricle and atrium dilatation (enlargement) and cause loud S1 and opening snap and pre-systolic accentuation, best hear by bell and the patient rolled to his left side

2- Mitral regurgitation

- Type: Systolic murmur
- Time: Pan-systolic murmur
- Best location to hear: at the apex (mitral area)
- Duration: hear after S1 immediately, and continuous throughout the systole

- Character: loud and continuous blowing نفخ sound
- Radiation: to the left axilla
- Intensity (grade): depend on the patient status
- Note: cause left ventricle and atrium dilatation (enlargement) and cause soft S1 and thrill and S3 sound

3- Mitral prolapse

- Type: systolic murmur
- Time: mid-systolic murmur ((it is called **mid-systolic click**))
- Best location to hear: at the apex (mitral area)
- Duration: begins in mid-systole and producing a late systolic murmur
- Character: very low sound, difficult to hear
- Radiation: no radiation
- Intensity (grade): depend on the patient status
- Note: it is called click if there is no murmur, but if there is murmur it is called mid-systolic murmur

4- Aortic stenosis

- Type: systolic murmur
- Time: mid-systolic murmur ((also called **ejection systolic murmur**))
- Best location to hear: at aortic area ((sometimes hear at the mitral area))
- Duration: begins after S1 as soft sound then become loud in the middle of systole then become soft again and stopping before S2
- Character: saw منشار teeth sound or harsh خشن sound
- Radiation: to the carotid area in the neck and upper right sternal edge
- Intensity (grade): depend on the patient status
- Note: sometimes cause ejection click in children more than adult , may cause thrill

5- Aortic regurgitation

- Type: Diastolic murmur
- Time: Early-diastolic murmur
- Best location to hear: in the left sternal area
- Duration: hear after S2 immediately
- Character: collapsing quality
- Intensity (grade): depend on the patient status
- Note: has large volume pulse and cause left ventricular dilatation, mitral stenosis occur with it, S1 and S2 and systole are normal, to hear this murmur the patient should sit and take breath and hold I, it associated with systolic flow murmur

6- Tricuspid stenosis

- Type: Diastolic murmur ((Mid-diastolic murmur))

- Best location to hear: at tricuspid area
- Intensity (grade): depend on the patient status
- Note: rare murmur

7- Tricuspid regurgitation

- Type: systolic murmur
- Time: Pan-systolic murmur
- Best location to hear: at tricuspid area
- Duration: hear after S1 immediately, and continuous throughout the systole
- Radiation: no radiation
- Intensity (grade): depend on the patient status
- Note: more common, associated with v wave in the JVP and a pulsatile liver

8- Pulmonary stenosis

- Type: systolic murmur
- Time: ejection-systolic murmur ((also called flow murmur))
- Best location to hear: at the pulmonary area
- Duration: after S1 as soft sound then become loud in the middle of systole then become soft again and stopping before S2
- Character: softer than the sound of aortic stenosis murmur
- Radiation: no radiation but sometimes radiate to the right shoulder
- Intensity (grade): depend on the patient status
- Note: usually it is congenital

9- Ventricular septal defect VSD

- Type: systolic murmur
- Time: Pan-systolic murmur
- Best location to hear: at left 4th intercostal space (right sternal edge)
- Character: loud like the murmur of mitral or tricuspid regurgitation
- Radiation: all over the precordium
- Intensity (grade): depend on the patient status
- Note: it is congenital but sometime acquired, very clear normal S1, there is a thrill

10- Atrial septal defect ASD

- Type: systolic murmur
- Time: ejection-systolic murmur ((also called **pulmonary flow murmur**))
- Best location to hear: at the pulmonary area
- Intensity (grade): depend on the patient status
- Note: the defect occur in child age and the murmur appear at 40-50 years old, increase the blood volume in the right side of the heart lead to pulmonary hypertension, lead to wide and fixed splitting of S2

11- Patent ductus arteriosus PDA

- Type: Diastolic + systolic murmur
- Time: continuous murmur is systole and diastole ((called **machinery murmur**))
- Best location to hear: at the upper left sternal border
- Duration: start from the beginning of systole and stop at the end of diastole
- Character: high volume in systole and low volume in diastole
- Radiation: over the left scapula
- Intensity (grade): depend on the patient status

12- Other murmurs

- Pan-systolic murmur occur in VSD, mitral regurgitation, tricuspid regurgitation
- Flow murmur occur when high volume of blood pass through normal valve or when normal volume of blood pass through narrowed valve
- Combined valve disease murmur occur in mitral stenosis + regurgitation or in aortic stenosis and regurgitation
- Graham Steell murmur occur in pulmonary regurgitation, it is rare, caused by pulmonary artery dilatation in pulmonary hypertension or congenital pulmonary valve defect
- Austin Flint murmur is a mid-diastolic murmur that accompanies aortic regurgitation

#Causes of systolic murmur

- **Ejection systolic murmurs:**
 - Increased flow through normal valves
 - 'Innocent systolic murmur': fever, athletes (bradycardia → large stroke volume), pregnancy (cardiac output maximum at 15 weeks)
 - Atrial septal defect (pulmonary flow murmur)
 - Severe anemia
 - Normal or reduced flow through a stenotic valve
 - Aortic stenosis
 - Pulmonary stenosis
 - Other causes of flow murmurs
 - Hypertrophic cardiomyopathy (obstruction at subvalvular level)
 - Aortic regurgitation (aortic flow murmur)
- **Pan-systolic murmurs:**

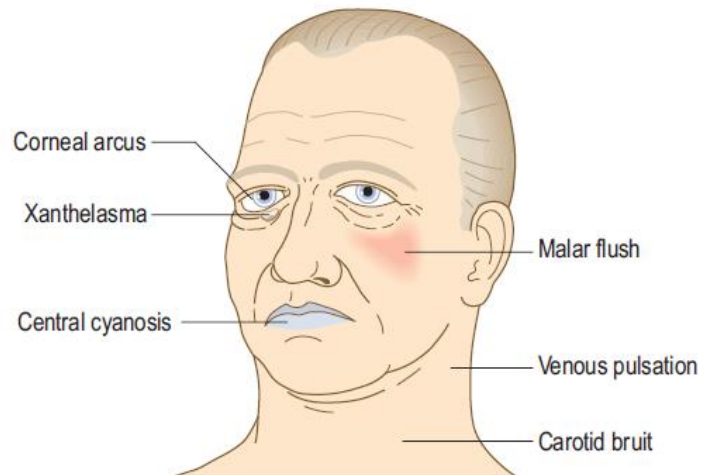
All caused by a systolic leak from a high- to a lower-pressure chamber:

 - Mitral regurgitation
 - Tricuspid regurgitation
 - Ventricular septal defect
 - Leaking mitral or tricuspid prosthesis

General examination

#General inspection

- Age and gender
- Conscious state
- Looks well or ill
- Breathlessness
- Cyanosis
- Frightened or distressed
- Check temperature



#Face and eyes

- Central cyanosis (in the mouth) → in heart failure, congenital heart disease (Right to Left shunt + clubbing)
- Xanthelasmata (at the eyelids) → indicate cardiovascular disease (can occur in normolipidaemic patient)
- Corneal arcus (at the iris) → caused by cholesterol deposition (can occur in normolipidaemic patient)
- Petechiae (in the conjunctivae) → in bleeding disorder and increased blood pressure
- Roth's spots (in the retina) → are flame-shaped retinal hemorrhages with cotton-wool centers, caused by a similar mechanism to splinter hemorrhage and can also occur in anemia or leukemia
- Features of hypertension and diabetes (in the Fundi)

#Hand and skin

- Temperature →
 - warm and sweaty hand: in infective endocarditis and pericarditis and after MI
 - cold and clammy hand: in hypotension and shock
- Tobacco staining
- Peripheral cyanosis → in peripheral vascular disease but normal in cold weather
- Petechial rash (in legs and conjunctiva) → in vasculitis and endocarditis
- Xanthomata (on the hand) → yellow skin or tendon nodules from lipid deposits
- Splinter hemorrhage → in infective endocarditis and some vasculitic disorders
- Nail fold infarcts and finger clubbing → uncommon features of endocarditis

- Janeway lesions → painless red spots, which blanch on pressure, on the thenar/hypothenar eminences of the palms, and soles of the feet
- Osler's nodes → painful raised erythematous lesions which are rare but found most often on the pads of the fingers and toes

#Urinalysis

- hematuria (endocarditis, vasculitis)
- glucose in urine (diabetes)
- protein in urine (hypertension, renal disease)

Arterial pulses

#General information

- When taking a pulse, assess:
 - Rate
 - Rhythm
 - Volume
 - Character
- Record individual pulses as:
 - Normal +
 - Reduced +/-
 - Absent -
 - Aneurysmal ++
- If you are in any doubt about whose pulse you are feeling, palpate your own pulse at the same time. If it is not synchronous with yours, it is the patient's.

#Surface markings of the arterial pulses

- **Radial:** At the wrist, lateral to the flexor carpi radialis tendon
- **Brachial:** In the antecubital fossa, medial to the biceps tendon
- **Carotid:** At the angle of the jaw, anterior to the sternocleidomastoid muscle
- **Femoral:** Just below the inguinal ligament, midway between the anterior superior iliac spine and the pubic symphysis (the mid inguinal point). It is immediately lateral to the femoral vein and medial to the femoral nerve
- **Popliteal:** Lies posteriorly in relation to the knee joint, at the level of the knee crease, deep in the popliteal fossa
- **Posterior tibial:** Located 2 cm below and posterior to the medial malleolus, where it passes beneath the flexor retinaculum between flexor digitorum longus and flexor hallucis longus

- **Dorsalis pedis:** Passes lateral to the tendon of extensor hallucis longus and is best felt at the proximal extent of the groove between the first and second metatarsals. It may be absent or abnormally sited in 10% of normal subjects, sometimes being 'replaced' by a palpable perforating peroneal artery

Note: Carotid pulse: Some clinicians consider routine examination of the carotid pulse is inappropriate because it may cause distal vascular events, e.g. transient ischemic attack, or induce reflex, vagally mediated bradycardia. In assessing a patient who may have had a cardiac arrest, however, it is the pulse of choice. If you do examine the carotid pulse do this gently and never assess both carotids simultaneously.

#Normal findings

Rate:

- Resting heart rate is normally 60–90 bpm
- Bradycardia is a pulse rate <60 bpm
- Tachycardia is a rate of >100 bpm
- A pulse rate of 40 bpm can be normal in a fit young adult
- A pulse rate of 65 bpm may be abnormally low in acute heart failure

Rhythm:

- Sinus rhythm originates from the sinoatrial node and produces a regular rhythm
- It varies slightly with the respiratory cycle, mediated by the vagus nerve, and is most pronounced in children, young adults or athletes (sinus arrhythmia).
- During inspiration: parasympathetic tone falls and the heart rate increases
- During expiration: the heart rate decreases

Volume

- Volume refers to the perceived degree of pulsation and reflects the pulse pressure

Character

- Character refers to the waveform or shape of the arterial pulse

#Abnormal findings

Rate:

- ❖ Fast (tachycardia, >100 bpm):
 - Sinus rhythm
 - Exercise
 - Pain
 - Excitement/anxiety

- Fever
- Hyperthyroidism
- Medication: Sympathomimetic, e.g. salbutamol Vasodilators
- Arrhythmia
 - Atrial fibrillation
 - Atrial flutter
 - Supraventricular tachycardia
 - Ventricular tachycardia
- ❖ Slow (bradycardia, <60 bpm):
 - Sinus rhythm
 - Sleep
 - Athletic training
 - Hypothyroidism
 - Medication: Beta-blockers, Digoxin, Verapamil, diltiazem
 - Arrhythmia
 - Carotid sinus hypersensitivity
 - Sick sinus syndrome
 - Second-degree heart block
 - Complete heart block

Notes:

Rapid regular heart beating with syncope → malignant arrhythmia like ventricular tachycardia

Rapid regular heart beating with polyuria → Paroxysmal Atrial Tachycardia

Rhythm:

- Causes of irregular pulse
 - Sinus arrhythmia
 - Atrial extrasystoles
 - Ventricular extrasystoles
 - Atrial fibrillation
 - Atrial flutter with variable response
 - Second-degree heart block with variable response
- Regularly irregular pulse → due to an ectopic beat occurring at a regular interval or missed beat at regular interval (in second-degree atrioventricular block)
- Irregularly irregular pulse → due to atrial fibrillation.
 - The rate in atrial fibrillation depends on the number of beats conducted by the atrioventricular node. Untreated, the ventricular rate may be very fast (up to 200 bpm).
 - atrial fibrillation occur due to SA block lead to irregular beat with irregular interval with normal volume and pulse deficit
 - Common causes of atrial fibrillation:
 - Hypertension
 - Heart failure
 - Myocardial infarction

- Thyrotoxicosis
- Alcohol-related heart disease
- Mitral valve disease
- Infection, e.g. respiratory, urinary
- Following surgery, especially cardiothoracic surgery

Pulse deficit = number of pulses at wrist - apical heart rate

- Pulse deficit → the variability of the pulse rate (and therefore ventricular filling) explains why the pulse volume varies and there may be a pulse deficit, with some cycles not felt at the radial artery. Calculate the pulse deficit by counting the radial pulse rate and subtracting this from the apical heart rate assessed by auscultation.
- the difference between atrial fibrillation and multiple ectopic is that the atrial fibrillation has pulse deficit while multiple ectopic not

Volume

- Volume refers to the perceived degree of pulsation and reflects the pulse pressure
- Causes of low pulse volume
 - Reduced stroke volume
 - Left ventricular failure
 - Hypovolemia, bleeding, shock
 - Peripheral arterial disease.
- Causes of large pulse volume
 - Physiological
 - Exercise
 - Pregnancy
 - Advanced age
 - Increased environmental temperature
 - Pathological
 - Peripheral vascular disease ((arteriosclerosis))
 - Hypertension
 - Fever
 - Thyrotoxicosis
 - Anemia
 - Aortic regurgitation
 - Paget's disease of bone
 - Peripheral atrioventricular shunt
- A compensatory pause → following a premature ectopic beat is sometimes felt by the patient.
- Coarctation is a congenital narrowing of the aorta. In children, the upper limb pulses are usually normal with reduced volume lower limb pulses, which are delayed relative to the upper limb pulses (radio-femoral delay). In adults, coarctation usually presents with hypertension and heart failure.

Radio-femoral delay

Constriction of the aorta lead to normal pulse in the upper limbs and low pulse in the lower limbs.

Normally the femoral pulse is stronger than radial pulse and there is no delay

Character

- A **collapsing pulse** → is when the peak of the pulse wave arrives early and is followed by a rapid descent. This rapid fall imparts the 'collapsing' sensation. This is exaggerated by raising the patient's arm above the level of the heart. It occurs in severe aortic regurgitation and is associated with wide pulse pressure (systolic BP – diastolic BP >80 mmHg)
- A **slow-rising pulse** → has a gradual upstroke with a reduced peak occurring late in systole, and is a feature of severe aortic stenosis
- **Pulsus bisferiens** → is an increased pulse with a double systolic peak separated by a distinct mid-systolic dip. Causes include aortic regurgitation, and concomitant aortic stenosis and regurgitation
- **Pulsus alternans** → is a beat-to-beat variation in pulse volume with a normal rhythm. It is rare and occurs in advanced heart failure.
- **Pulsus paradoxus** → is an exaggeration of the normal variability of pulse volume with breathing. Pulse volume normally increases in expiration and decreases during inspiration due to intra-thoracic pressure changes affecting venous return to the heart. This variability in exaggerated diastolic filling of both ventricles is impeded by increased intra-pericardial pressure. This occurs in cardiac tamponade because of accumulation of pericardial fluid and in constrictive pericarditis, pericardial effusion assess Pulsus paradoxus by measuring the systolic blood pressure during inspiration and expiration. a decrease in systolic blood pressure **>15 mmHg with inspiration** is pathological (Pulsus paradoxus)

Blood pressure

#General information

- BP is a measure of the pressure that the circulating blood exerts against the arterial walls.
- Systolic BP → is the maximal pressure that occurs during ventricular contraction (systole).
- Diastolic BP → During ventricular filling (diastole), arterial pressure is maintained at a lower level by the elasticity and compliance of the vessel wall. The lowest value (diastolic BP) occurs immediately before the next cycle.
- BP is usually measured using a sphygmomanometer. In certain situations, such as the intensive care unit, it is measured invasively using an indwelling intra-arterial catheter connected to a pressure sensor.
- BP is measured in mmHg and recorded as systolic pressure/diastolic pressure, together with where, and how, the reading was taken, e.g. BP: 146/92 mmHg, right arm, supine.
- Normal BP is defined as <130/85 mmHg

- BP is an important guide to cardiovascular risk and provides vital information on the hemodynamic condition of acutely ill or injured patients.
- Ambulatory BP measurement → using a portable device at intervals during normal daytime activity and at night, is better at determining cardiovascular risk.
- BP constantly varies and rises with stress, excitement and environment.
- 'White-coat hypertension' → occurs in patients only when a patient is seeing a healthcare worker.

#Hypertension

- Hypertension is asymptomatic although, rarely in severe hypertension, headaches and visual disturbances occur.
- It is associated with significant morbidity and mortality from vascular disease (heart failure, coronary artery disease, cerebrovascular disease and renal failure)
- In most hypertensive patients there is no identifiable cause so-called '**essential hypertension**'
- **Secondary hypertension** is rare, occurring in <1% of the hypertensive population, and caused by:
 - Renal arterial disease, including renal artery stenosis
 - Pheochromocytoma
 - Conn's syndrome
 - Cushing's syndrome
 - Coarctation of the aorta
 - Adult polycystic kidney disease
- Assess the hypertensive patient for:
 - Any underlying cause
 - End-organ damage:
 - cardiac: heart failure
 - renal: chronic kidney disease
 - eye: hypertensive retinopathy
 - Overall risk of vascular disease: like stroke, myocardial infarction, heart failure
- Physical signs which may be associated with hypertension:
 - Head: Cerebrovascular disease, Cushingoid facies, Retinopathy
 - Chest: Buffalo 'hump', Left ventricular hypertrophy, 3rd/4th heart sound, Basal crackles
 - Abdomen: Renal failure, Renal disease ± bruit, Signs of alcohol-related disease
 - Limbs: Atrial fibrillation, Radio-femoral delay, Dependent edema

6.19 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

#Korotkoff sounds

- These sounds are produced between systole and diastole because the artery collapses completely and reopens with each heartbeat, producing a snapping or

knocking sound. The first appearance of sounds (phase 1) during cuff deflation indicates **systole**. As pressure is gradually reduced, the sounds muffle (phase 4) and then disappear (phase 5). Inter-observer agreement is better for phase 5 and this is the **diastolic BP**. Occasionally, muffled sounds persist (phase 4) and do not disappear; in this case, record phase 4 as the diastolic pressure

#Common problems in BP measurement للاطلاع

- **BP is different in each arm** → a difference >10 mmHg suggests the presence of subclavian artery disease. Unequal brachial BP is a marker of increased cardiovascular morbidity and mortality. Record the highest pressure and use this to guide management
- **Wrong cuff size**: the bladder should be approximately 80% of the length and 40% of the width of the upper arm circumference.
- **Auscultatory gap**: up to 20% of elderly hypertensive patients have Korotkoff sounds which appear at systolic pressure and disappear for an interval between systolic and diastolic pressure. If the first appearance of the sound is missed, the systolic pressure will be recorded at a falsely low level. Avoid this by palpating the systolic pressure first
- **Patient's arm at the wrong level**: the patient's elbow should be level with the heart. Hydrostatic pressure causes ~5 mmHg change in recorded systolic and diastolic BP for a 7 cm change in arm elevation
- **Terminal digit preference**: record the true reading rather than rounding values to the nearest 0 or 5
- **Postural change**: the pulse increases by about 11 bpm, systolic BP falls by 3–4 mmHg and diastolic BP rises by 5–6 mmHg when a healthy person stands. The BP stabilizes after 1–2 minutes. Check the BP after a patient has been standing for 2 minutes; a drop of $\geq 20 \text{ mmHg}$ on standing is postural hypotension
- **Atrial fibrillation**: makes BP assessment more difficult because of beat-to-beat variability. Deflate the cuff at 2 mmHg per beat and repeat measurement if necessary.

Jugular venous pressure JVP

#General information

- The JVP is primarily a sign of right ventricular function
- The JVP can be estimated by observing the level of blood in either the internal or external jugular veins
- both veins have valves but as blood flow is towards the heart, they do not affect this and may even make the waveform easier to see

- the normal waveform has 2 peaks per cycle, which helps distinguish it from the carotid arterial pulse
- The JVP level reflects right atrial pressure (normally <7 mmHg/9 cmH₂O)
- The sternal angle is approximately 5 cm above the right atrium, so the JVP in health should be ≤ **4 cm** above this angle when the patient lies at 45 degree
- If right atrial pressure is low, the patient may have to lie flat for the JVP to be seen
- If high, the patient may need to sit upright

#Aids to differentiate the jugular venous waveform from arterial pulsation:

- **Abdomino-jugular test:** firmly press over the abdomen. This increases venous return to the right side of the heart temporarily and the JVP normally rises.
- **Changes with respiration:** the JVP normally falls with inspiration due to decreased intra-thoracic pressure.
- **Waveform:** the normal JVP waveform has two distinct peaks per cardiac cycle:
 - "a wave" corresponds to right atrial contraction and occurs just before the first heart sound. In atrial fibrillation the 'a' wave is absent
 - "v wave" is caused by atrial filling during ventricular systole when the tricuspid valve is closed
 - "c wave" may be seen due to closure of the tricuspid valve
- **Occlusion:** the JVP waveform is obliterated by gently occluding the vein at the base of the neck with your finger.

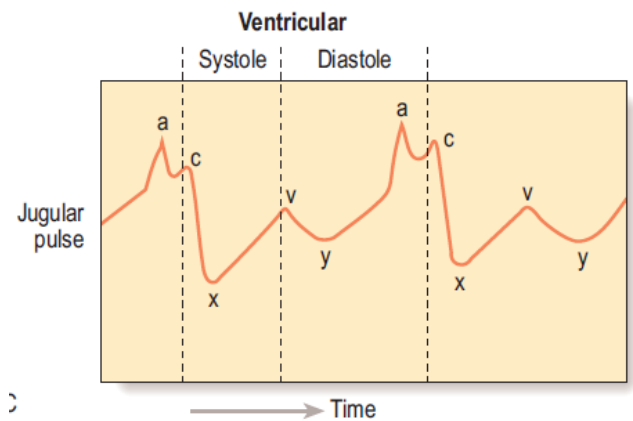
Differences between carotid artery and jugular venous pulsation	
Carotid	Jugular
Deep	Superficial
Jerky pulsatile character	Wavy sinusoid character
Rapid outward movement	Rapid inward movement
One peak per heart beat	Two peaks per heart beat (in sinus rhythm)
Palpable ((better to feel))	Impalpable ((better to see))
Pulsation unaffected by pressure at the root of the neck	Pulsation diminished by pressure at the root of the neck
Independent of respiration	Height of pulsation varies with respiration
Independent of position of patient	Varies with position of patient: decrease in upright position & increase in lying position
Independent of abdominal pressure	Rises with abdominal pressure
Not move the ear	Move the ear
Not limited	Limited in the upper part of the neck

"If JVP is not elevated, then the edema is not cardiogenic"

#Abnormalities of the jugular venous pulse Condition Abnormalities

- **Elevated JVP:**

- Fluid overload
- Right heart dilation
- COPD
- cor pulmonale
- Pulmonary embolism
- Heart failure
- Pericardial effusion
- Pericardial constriction
- Superior vena caval obstruction (most often caused by lung cancer)



- **Kussmaul's sign:** a paradoxical rise of JVP on inspiration seen in:

- Pericardial constriction or tamponade
- Severe right ventricular failure
- Restrictive cardiomyopathy

- **Prominent 'a' wave:** caused by delayed or restricted right ventricular filling:

- Pulmonary hypertension
- Tricuspid stenosis
- Atrial fibrillation → Absent 'a' waves

- **Cannon waves:** giant 'a' waves occur when the right atrium contracts against a closed tricuspid valve:

- Irregular cannon waves: in complete heart block and are due to atrio-ventricular dissociation
- Regular cannon waves: in junctional rhythm and with some ventricular and supraventricular tachycardias

- **'cv' wave:** a fusion of the 'c' and 'v' waves resulting in a large systolic wave and associated with a pulsatile liver is seen in tricuspid regurgitation

- **Giant 'v' waves:** occur in Tricuspid regurgitation

- **Prominent 'y' descent:** occur in Pericardial effusion

- **Abdominojugular reflux:**

- Heart failure → sustained abdominojugular reflux >10 seconds
- Superior vena caval obstruction (most often caused by lung cancer) → loss of pulsation, negative abdominojugular test

6.14 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

Peripheral arterial system

Lower limbs

#Classification of lower limb ischemia:

I. **Asymptomatic** → ankle to brachial pressure index (ABPI) <0.9 at rest

II. **Intermittent claudication** (leg angina) →

- Arterial claudication: is pain felt in the legs on walking due to arterial insufficiency and is the most common symptom of peripheral arterial disease
- Neurogenic claudication: is due to neurological and musculoskeletal disorders of the lumbar spine
- Venous claudication: is due to venous outflow obstruction from the leg, following extensive DVT

III. **Night/rest pain** → the patient goes to bed, falls asleep, but is then woken 1–2 hours later with severe pain in the foot, usually in the instep. The pain is due to poor perfusion resulting from the loss of the beneficial effects of gravity on lying down and the reduction in heart rate, BP and cardiac output that occurs when sleeping. Patients often obtain relief by hanging the leg out of bed or by getting up and walking around

IV. **Tissue loss (ulceration/gangrene)** → in severe lower limb PDA and injuries



6.34 The clinical features of arterial, neurogenic and venous claudication

	Arterial	Neurogenic	Venous
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve root or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to iliofemoral venous occlusion
Site of pain	Muscles, usually the calf but may involve thigh and buttocks	Ill defined. Whole leg. May be associated with numbness and tingling	Whole leg. 'Bursting' in nature
Laterality	Unilateral if femoropopliteal, and bilateral if aortoiliac disease	Often bilateral	Nearly always unilateral
Onset	Gradual after walking the 'claudication distance'	Often immediate on walking or standing up	Gradual, from the moment walking starts
Relieving features	On stopping walking, the pain disappears completely in 1–2 minutes	Bending forwards and stopping walking. May sit down for full relief	Leg elevation
Colour	Normal or pale	Normal	Cyanosed. Often visible varicose veins
Temperature	Normal or cool	Normal	Normal or increased
Oedema	Absent	Absent	Always present
Pulses	Reduced or absent	Normal	Present but may be difficult to feel owing to oedema
Straight-leg raising	Normal	May be limited	Normal

#Signs of acute limb ischemia [Very Important]

- Soft signs
 - Pulseless
 - Pallor
 - Perishing cold
- Hard signs (indicating a threatened limb)
 - Paraesthesia: means loss of light touch sensation
 - Paralysis
 - Pain on squeezing muscle
- Other signs
 - Absence of hair
 - Thin skin
 - Brittle nails
 - Rest pain
 - Tissue loss
 - Have ankle BP <50 mmHg
 - Positive Buerger's test
 - Muscle tenderness
 - The limb skin appears 'marble white' → then light blue or purple mottling which has a fine reticular pattern and blanches on pressure → coarser pattern of mottling which is dark purple, almost black, and does not blanch → Finally, large patches of fixed staining lead to blistering and liquefaction



6.36 Acute limb ischaemia: embolus versus thrombosis in situ

	Embolus	Thrombosis
Onset and severity	Acute (seconds or minutes), ischaemia profound (no pre-existing collaterals)	Insidious (hours or days), ischaemia less severe (pre-existing collaterals)
Embolic source	Present (usually atrial fibrillation)	Absent
Previous claudication	Absent	Present
Pulses in contralateral leg	Present	Often absent
Diagnosis	Clinical	Angiography
Treatment	Embolectomy and anticoagulation	Medical, bypass surgery, thrombolysis

#The commonest causes of acute limb ischemia are:

- Thromboembolism: usually from the left atrium in association with atrial fibrillation
- Thrombosis in situ: thrombotic occlusion of an already narrowed atherosclerotic arterial segment

#Compartment syndrome

- Occurs where there is increased pressure within the fascial compartments of the limb, most commonly the calf
- The two commonest causes are lower trauma, e.g. fractured tibia, and reperfusion following treatment of acute lower limb ischemia.
- Failure to recognize and treat compartment syndrome may require limb amputation. The key
- Symptom is severe pain often unrelieved by opioids and exacerbated by active or passive movement. Peripheral pulses are usually present

Stroke

Transient ischemic attack (TIA) describes a stroke in which symptoms resolve within 24 hours.

The term 'stroke' is reserved for those events in which symptoms last for more than 24 hours.

#Carotid artery territory (anterior circulation)

- Clinical features vary according to the cerebral area involved
- Motor deficit
- Visual field defect: like homonymous hemianopia
- Difficulty with speech (dysphasia)

#Vertebrobasilar artery territory (posterior circulation)

- Giddiness
- Collapse with or without loss of consciousness
- Transient occipital blindness or complete loss of vision in both eyes
- Subclavian steal syndrome → Signs of this include asymmetry of the pulses and BP in the arms, sometimes with a bruit over the subclavian artery in the supraclavicular fossa.

Abdominal symptoms

#Mesenteric angina

- Severe central abdominal pain typically develops 10–15 minutes after eating
- The patient becomes scared of eating and significant weight loss is a universal finding
- Diarrhea may occur
- Acute mesenteric ischemia is a surgical emergency:
 - Severe abdominal pain
 - Shock
 - Bloody diarrhea
 - Profound metabolic acidosis
 - Renal angle pain occurs from renal infarction or ischemia
 - Microscopic or macroscopic hematuria

#Abdominal aortic aneurysm

- Abdominal and/or back pain
- Pulsatile abdominal mass
- Shock (hypotension)
- Athero-embolism from an AAA can cause '**blue toe syndrome**' characterized by purple discoloration of the toes and forefoot of both feet

#Vasospastic symptoms

- Raynaud's phenomenon: is digital ischemia induced by cold and emotion
- It has three phases:
 - Pallor: due to digital artery spasm and/or obstruction
 - Cyanosis: due to deoxygenation of static venous blood (this phase may be absent)
 - Redness: due to reactive hyperaemia
- Raynaud's phenomenon may be:
 - Primary (Raynaud's disease) and due to idiopathic digital artery vasospasm
 - Secondary (Raynaud's syndrome)

Signs suggesting vascular disease [Very Important]

#Hands and arms

- Tobacco stains → Smoking
- Purple discoloration of the fingertips → Atheroembolism from a proximal subclavian aneurysm
- Pits and healed scars in the finger pulps → Secondary Raynaud's syndrome
- Calcinosis and visible nail fold capillary loops → Systemic sclerosis and CREST (calcinosis, Raynaud's phenomenon, esophageal dysfunction, sclerodactyly, telangiectasia)
- Wasting of the small muscles of the hand → Thoracic outlet syndrome

#Face and neck

- Corneal arcus and xanthelasma → Hypercholesterolaemia
- Horner's syndrome → Carotid artery dissection or aneurysm
- Hoarseness of the voice and 'bovine' cough → Recurrent laryngeal nerve palsy from a thoracic aortic aneurysm
- Prominent veins in the neck, shoulder and anterior chest → Axillary/subclavian vein occlusion

#Abdomen

- Epigastric/umbilical pulsation → Aortoiliac aneurysm
- Mottling of the abdomen → Ruptured abdominal aortic aneurysm or saddle embolism occluding aortic bifurcation
- Evidence of weight loss → Visceral ischemia

#Buerger's test

- With the patient lying supine, stand at the foot of the bed. Raise the patient's feet and support the legs at 45 to the horizontal for 2-3 minutes.
- Watch for pallor with emptying or 'guttering' of the superficial veins.
- Ask the patient to sit up and hang the legs over the edge of the bed.
- Watch for reactive hyperaemia on dependency; the loss of pallor and spreading redness is a positive test.

#Ankle to brachial pressure index

- Assessing pulse status can be unreliable in patients with obesity or edema. Routinely measure ABPI in all patients with difficulty palpating lower limb pulses or where PAD is suspected on the basis of history.
- Examination sequence
 - Use a hand-held Doppler and a sphygmomanometer
 - Hold the probe over the posterior tibial artery
 - Inflate a BP cuff round the ankle
 - Note the pressure when Doppler signal disappears. This is the systolic pressure in that artery as it passes under the cuff
 - Repeat holding the probe over dorsalis pedis, and then the perforating peroneal
 - Measure the brachial BP in both arms, holding the Doppler probe over the brachial artery at the elbow of the radial artery at the wrist
- Normal findings:
 - The ratio of the highest pedal artery pressure to the highest brachial artery pressure is the ABPI
 - In health, the ABPI is >1.0 when the patient is supine
 - The popliteal artery is always hard to feel. if you feel it easily, consider an aneurysm.
- Abnormal findings:
 - <0.9 = intermittent claudication
 - <0.4 = critical limb ischemia
 - Patients with lower limb PAD, particularly those with diabetes mellitus, often have incompressible, calcified crural arteries with falsely elevated pedal pressures and ABPI
 - Use a Doppler ultrasound probe to detect the foot arteries while elevating the foot. The Doppler signal disappears at a height (in cm) above the bed that approximates to the perfusion pressure (in mmHg).

Peripheral venous system

Clinical presentation

Lower limb venous disease presents in four ways:

- Varicose veins
- Superficial thrombophlebitis
- DVT
- Chronic venous insufficiency and ulceration

- DVT (bluish color) → flagnesia cerulea
- DVT with increased pressure (pale color) → flagnesia alba

Pain

- Uncomplicated varicose veins → often complain of aching leg discomfort, itching and a feeling of swelling
- DVT → causes pain and tenderness in the affected part (usually the calf)
- Superficial thrombophlebitis → red, painful area overlying the vein involved
- Varicose ulceration → may be surprisingly painless; if it is painful, this may be relieved by limb elevation (but exclude coexisting arterial disease)

Note: Bandaging for a leg ulcer is contraindicated unless there is documented evidence of adequate arterial circulation. Do this by feeling the pulses or by measuring the ABPI.

Swelling

- Swelling (or edema), or a 'feeling of swelling', may be associated with lower limb venous disease

Discoloration

- Chronic venous insufficiency → lead to **lipodermatosclerosis** which varies in color from deep blue/black to purple or bright red

Chronic venous ulceration

- Causes: primarily due to venous disease. Other causes include pyoderma gangrenosum, syphilis, tuberculosis, leprosy, sickle cell disease and tropical conditions.
- Appearance: chronic venous ulceration usually affects the medial aspect, is shallow; is pink (granulation tissue) or yellow/green (slough); has an irregular margin; and is always associated with other skin changes of chronic venous insufficiency (varicose eczema, lipodermatosclerosis)

Deep vein thrombosis

- The leg: Most patients who die from pulmonary embolism have non-occlusive thrombosis and the leg is normal on clinical examination.

- The arm: Axillary subclavian vein thrombosis can occur due to trauma, and the symptoms include: arm swelling and discomfort, often exacerbated by activity, and the skin is cyanosed and mottled

Superficial venous thrombophlebitis

- Occur in patients with severe varicose veins and is more common during pregnancy
- Recurrent superficial venous thrombophlebitis → underlying malignancy
- It may propagate into the deep system → leading to DVT and pulmonary embolism

The Trendelenburg test

- is test to detect sapheno-femoral junction reflux
- examination sequence
 - Ask the patient to sit on the edge of the examination couch.
 - Elevate the limb as far as is comfortable for the patient and empty the superficial veins by 'milking' the leg towards the groin.
 - With the patient's leg still elevated, press with your thumb over the sapheno-femoral junction (2–3 cm below and 2–3 cm lateral to the pubic tubercle). A high thigh tourniquet can be used instead.
 - Ask the patient to stand while you maintain pressure over the sapheno-femoral junction.
 - If sapheno-femoral junction reflux is present, the patient's varicose veins will not fill until your digital pressure, or the tourniquet, is removed.

 6.43 Risk factors for deep vein thrombosis
• Recent bed rest or operations (especially to the leg, pelvis or abdomen) • Recent travel, especially long flights • Previous trauma to the leg, especially long-bone fractures, plaster of Paris splintage and immobilisation • Pregnancy or features to suggest pelvic disease • Malignant disease • Previous deep vein thrombosis • Family history of thrombosis • Recent central venous catheterisation, injection of drugs, etc.

6.42 Features of deep vein thrombosis of the lower limb		
Clinical feature	Non-occlusive thrombus	Occlusive thrombus
Pain	Often absent	Usually present
Calf tenderness	Often absent	Usually present
Swelling	Absent	Present
Temperature	Normal or slightly increased	Increased
Superficial veins	Normal	Distended
Pulmonary embolism	High risk	Low risk



6.41 Clinical features of venous and arterial ulceration

Clinical feature	Venous ulceration	Arterial ulceration
Age	Develops at age 40–45 but may not present for years; multiple recurrences common	First presents in over-60s
Sex	More common in women	More common in men
Past medical history	Deep vein thrombosis (DVT) or suggestive of occult DVT, i.e. leg swelling after childbirth, hip/knee replacement or long bone fracture	Peripheral arterial disease, cardio- and cerebrovascular disease
Risk factors	Thrombophilia, family history, previous DVT	Smoking, diabetes, hypercholesterolaemia and hypertension
Pain	One-third have pain (not usually severe) that improves with elevating the leg	Severe pain, except in diabetics with neuropathy; improves on dependency
Site	Gaiter areas; usually medial to long saphenous vein; 20% are lateral to short saphenous vein	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)
Margin	Irregular, often with neopithelium (appears whiter than mature skin)	Regular, indolent, 'punched out'
Base	Often pink and granulating under green slough	Sloughy (green) or necrotic (black), with no granulation
Surrounding skin	Lipodermatosclerosis always present	No venous skin changes
Veins	Full and usually varicose	Empty with 'guttering' on elevation
Swelling (oedema)	Usually present	Absent
Temperature	Warm	Cold
Pulses	Present, but may be difficult to feel	Absent

#Signs of hypoxia

- cyanosis
- tachycardia
- polycythemia
- pulmonary and systemic hypertension

#Signs of hypercapnia

- flapping tremor
- confusion
- bounding pulse
- large volume pulse
- papilledema
- warm peripheries

#Signs of cor pulmonale

- leg edema
- raised JVP
- congestive hepatitis
- right ventricular heave

#Red flag symptoms associated with cough

- Hemoptysis
- Breathlessness
- Fever
- Chest pain
- Weight loss

#Causes of cough

- Acute cough (<3 weeks)
 - Normal chest X-ray
 - Viral respiratory tract infection
 - Bacterial infection (acute bronchitis)
 - Inhaled foreign body
 - Inhalation of irritant dusts/fumes
 - Abnormal chest X-ray

- Pneumonia
- Inhaled foreign body
- Acute hypersensitivity pneumonitis
- Chronic cough (>8 weeks)
 - Normal chest X-ray
 - Gastro-esophageal reflux disease
 - Asthma
 - Post viral bronchial hyperreactivity
 - Rhinitis/sinusitis
 - Cigarette smoking
 - Drugs, especially angiotensin-converting enzyme inhibitors
 - Irritant dusts/fumes
 - Abnormal chest X-ray
 - Lung tumor
 - Tuberculosis
 - Interstitial lung disease
 - Bronchiectasis

#Causes of hemoptysis

- Tumor
 - Malignant: Lung cancer, Endobronchial metastases
 - Benign: Bronchial carcinoid
- Infection
 - Bronchiectasis
 - Tuberculosis
 - Lung abscess
 - Mycetoma
 - Cystic fibrosis
- Vascular
 - Pulmonary infarction
 - Arteriovenous malformation
 - Vasculitis
 - Goodpasture's syndrome
 - Iatrogenic
 - Bronchoscopic biopsy
 - Transthoracic lung biopsy
 - Bronchoscopic diathermy
 - Acute left ventricular failure
 - Anticoagulation
 - Polyangiitis
 - Trauma

- Inhaled foreign body
- Chest trauma
- Cardiac
- Mitral valve disease
- Hematological
- Blood dyscrasias

#Causes of breathlessness

- **Non-cardiorespiratory**

- Anemia
- Metabolic acidosis
- Obesity
- Psychogenic
- Neurogenic

- **Cardiac**

- Left ventricular failure
- Mitral valve disease
- Cardiomyopathy
- Constrictive pericarditis
- Pericardial effusion

- **Respiratory**

- Airways
 - Laryngeal tumor
 - Foreign body
 - Asthma
 - COPD
 - Bronchiectasis
 - Lung cancer
 - Bronchiolitis
 - Cystic fibrosis
- Parenchyma
 - Pulmonary fibrosis
 - Alveolitis
 - Sarcoidosis
 - Tuberculosis
 - Pneumonia
 - Diffuse infections, e.g. Pneumocystis jiroveci pneumonia
 - Tumor (metastatic, lymphangitis)
- Pulmonary circulation
 - Pulmonary thromboembolism
 - Pulmonary vasculitis

- Primary pulmonary
- hypertension Pleural
 - Pneumothorax
 - Effusion
 - Diffuse pleural fibrosis
- Chest wall
 - Kyphoscoliosis
 - Ankylosing spondylitis
- Neuromuscular
 - Myasthenia gravis
 - Neuropathies
 - Muscular dystrophies
 - Guillain–Barré syndrome

#Breathlessness: modes of onset, duration and progression

- Minutes
 - Pulmonary thromboembolism
 - Pneumothorax
 - Asthma
 - Inhaled foreign body
 - Acute left ventricular failure
- Hours to days
 - Pneumonia
 - Asthma
 - Exacerbation of COPD
- Weeks to months
 - Anemia
 - Pleural effusion
 - Respiratory neuromuscular disorders
- Months to years
 - COPD
 - Pulmonary fibrosis
 - Pulmonary tuberculosis

#Acute breathlessness: commonly associated symptoms

- No chest pain
 - Pulmonary embolism
 - Pneumothorax
 - Metabolic acidosis
 - Hypovolemia/shock
 - Acute left ventricular failure/pulmonary edema

- Pleuritic chest pain
 - Pneumonia
 - Pneumothorax
 - Pulmonary embolism
 - Rib fracture
- Central chest pain
 - Myocardial infarction with left ventricular failure
 - Massive pulmonary embolism/infarction
- Wheeze and cough
 - Asthma
 - COPD

#Causes of Paroxysmal nocturnal dyspnea

- ischemic heart disease
- aortic valve disease
- hypertension
- cardiomyopathy
- atrial fibrillation
- mitral valve disease
- atrial tumors

#Symptoms of obstructive sleep apnea/ hypopnea syndrome (OSAHS)

- Snoring
- Witnessed apneas
- Unrefreshing sleep
- Restless sleep
- Nocturia
- Excessive daytime sleepiness
- Impaired concentration
- Choking episodes during sleep
- Irritability/personality change
- Decreased libido

#Causes of chest pain

☒ Non-central

- Pleural
 - Infection: pneumonia, bronchiectasis, tuberculosis
 - Malignancy: lung cancer, mesothelioma, metastatic
 - Pneumothorax
 - Pulmonary infarction

- Connective tissue disease: rheumatoid arthritis, SLE
- Chest wall
 - Malignancy: lung cancer, mesothelioma, bony metastases
 - Persistent cough/ breathlessness
 - Muscle sprains/tears
 - Bornholm's disease (Coxsackie B infection)
 - Tietze's syndrome (costochondritis)
 - Rib fracture
 - Intercostal nerve compression
 - Thoracic shingles (herpes zoster)

☒ **Central**

- Tracheal
 - Infection
 - Irritant dusts
- Cardiac
 - Massive pulmonary thromboembolism
 - Acute myocardial infarction/ ischemia
- Esophageal
 - Esophagitis
 - Rupture
- Great vessels
 - Aortic dissection
- Mediastinal
 - Lung cancer
 - Thymoma
 - Lymphadenopathy
 - Metastases
 - Mediastinitis

#Examples of drug-induced respiratory conditions

- **Bronchoconstriction:** Beta-blockers, Opioids, NSAIDs
- **Cough:** Angiotensin-converting enzyme inhibitors
- **Bronchiolitis obliterans:** Penicillamine
- **Diffuse parenchymal lung disease:**
 - Cytotoxic agents: bleomycin, methotrexate
 - Anti-inflammatory agents: sulfasalazine, penicillamine, gold salts, aspirin
 - Cardiovascular drugs: amiodarone, hydralazine
 - Antibiotics: nitrofurantoin
 - Intravenous drug misuse
 - Radiation
- **Pulmonary thromboembolism:** Estrogens

- **Pulmonary hypertension:** Estrogens, Dexfenfluramine, fenfluramine
- **Pleural effusion:** Amiodarone, Nitrofurantoin, Phenytoin, Methotrexate, Pergolide
- **Respiratory depression:** Opioids, Benzodiazepines

#Examples of occupational lung disease

- Pulmonary fibrosis → Asbestos, Quartz (silica), Coal, Beryllium
- COPD → Coal
- Malignancy → Asbestos, radon
- Byssinosis → Cotton, flax, hemp
- Hypersensitivity pneumonitis:
 - Farmer's lung → Fungal spores of thermophilic actinomycetes or Micropolyspora faeni
 - Malt worker's lung → Aspergillus clavatus
 - Bird fancier's lung → Bloom on birds' feathers/excreta
- Asthma → Animals, Grains, Hardwood dusts, Colophony, Enzymes, Isocyanates, Epoxy resins, Drugs, Formaldehyde, paraldehyde, latex

#Features of severe community-acquired pneumonia (CRB-65)

- Confusion
- Respiratory rate >30 breaths/min
- Blood pressure – diastolic <60 mmHg
- Age >65 years

#Clinical signs of common respiratory conditions

- **Lung cancer**
 - Ptosis/Horner's syndrome
 - Cervical lymphadenopathy/ scalene nodes
 - Raised, fixed JVP (SVCO)
 - Signs of effusion (Reduced expansion, Stony dull' percussion, Reduced/absent, breath sounds and vocal resonance)
 - Signs of collapse (Reduced chest expansion, Dull percussion, Reduced breath sounds and vocal resonance)
 - Clubbing
 - Tobacco staining
- **Asthma**
 - Agitation or drowsiness (in 'severe asthma')
 - Central cyanosis, (in 'severe asthma')
 - Intercostal indrawing (children)
 - Wheeze (silent chest in 'severe asthma')
 - Hyperinflated chest
 - Rapid pulse (slow in 'severe asthma')
- **Pneumonia**

- Confusion (in 'severe pneumonia')
- Fever (rigors)
- Central cyanosis
- Respiratory rate >30 (in 'severe pneumonia')
- Signs of consolidation (Reduced chest expansion, dull percussion, Bronchial breath sounds ± pleural rub, Increased vocal resonance)
- Hypotension (diastolic <60 mmHg in 'severe pneumonia')
- **Idiopathic pulmonary fibrosis (fibrosing alveolitis)**
 - Central cyanosis
 - Reduced chest expansion
 - Fine late inspiratory crackles
 - Clubbing
 - Ankle swelling in late stages
- **Chronic obstructive pulmonary disease**
 - Plethoric (secondary polycythemia)
 - Central cyanosis
 - Raised JVP (cor pulmonale)
 - Using accessory muscles
 - Hyperinflated 'barrel-shaped' chest
 - Wheeze
 - Reduced air entry
 - Tobacco staining
 - Ankle swelling (cor pulmonale)
- **Pulmonary thromboembolism**
 - In pain (pleuritic)
 - Pleural friction rub
 - Signs of effusion (Reduced expansion, 'Stony dull' percussion, reduced breath sounds and vocal resonance)
 - Signs of deep vein thrombosis (Unilateral, Edema, Warmth, Tenderness)

#Common symptoms of heart disease

- **Chest discomfort**
 - Cardiovascular causes
 - Myocardial infarction
 - Angina
 - Pericarditis
 - Aortic dissection
 - Other causes
 - Esophageal spasm
 - Pneumothorax
 - Musculoskeletal pain

- **Breathlessness**
 - Cardiovascular causes
 - Heart failure
 - Angina
 - Pulmonary embolism
 - Pulmonary hypertension
 - Other causes
 - Respiratory disease
 - Anemia
 - Obesity
 - Anxiety
- **Palpitation**
 - Cardiovascular causes
 - Tachyarrhythmias
 - Ectopic beats
 - Other causes
 - Anxiety
 - Hyperthyroidism
 - Drugs
- **Syncope/dizziness**
 - Cardiovascular causes
 - Arrhythmias
 - Postural hypotension
 - Aortic stenosis
 - Hypertrophic cardiomyopathy
 - Atrial myxoma
 - Other causes
 - Simple faints
 - Epilepsy
 - Anxiety
- **Edema**
 - Cardiovascular causes
 - Heart failure
 - Constrictive pericarditis
 - Venous stasis
 - Lymphedema
 - Other causes
 - Nephrotic syndrome
 - Liver disease
 - Drugs
 - Immobility

#Genetically determined cardiovascular disorders

- **Single-gene defects**
 - Hypertrophic cardiomyopathy
 - Marfan's syndrome
 - Familial hypercholesterolaemia
 - Muscular dystrophies
 - Long Q–T syndrome
 - Arrhythmogenic right ventricular cardiomyopathy (ARVC)
- **Polygenic inheritance**
 - Ischemic heart disease
 - Hypertension
 - Type 2 diabetes mellitus
 - Hyperlipidemia
 - Abdominal aortic aneurysm

#Occupational aspects of cardiovascular disease

- **Occupational exposure associated with cardiovascular disease**
 - Organic solvents: arrhythmias, cardiomyopathy
 - Vibrating machine tools: Raynaud's phenomenon
 - Publicans: alcohol-related cardiomyopathy
- **Occupational exposure exacerbating pre-existing cardiac conditions**
 - Cold exposure: angina, Raynaud's disease
 - Deep-sea diving: embolism through foramen ovale
- **Occupational requirements for high standards of cardiovascular fitness**
 - Pilots
 - Public transport/heavy goods vehicle drivers
 - Armed forces
 - Police

#Cardiovascular disease presenting with 'non-cardiac' symptoms

- Central nervous system (Stroke) → causes:
 - Cerebral embolism
 - Endocarditis
 - Hypertension
- Gastrointestinal (Jaundice, Abdominal pain) → causes:
 - Liver congestion
 - secondary to heart
 - failure
 - Mesenteric embolism
- Renal (Oliguria) → causes: heart failure

#Causes of unilateral and bilateral leg edema

- Unilateral
 - Deep vein thrombosis
 - Soft-tissue infection
 - Trauma
 - Immobility, e.g. hemiplegia
 - Lymphedema
- Bilateral
 - Heart failure
 - Chronic venous insufficiency
 - Hypoproteinaemia, e.g. nephrotic syndrome, kwashiorkor, cirrhosis
 - Lymphatic obstruction, e.g. pelvic tumor, filariasis
 - Drugs, e.g. NSAIDs, nifedipine, amlodipine, fludrocortisone
 - Inferior vena caval obstruction
 - Thiamine (vitamin B1) deficiency (wet beriberi)
 - Milroy's disease (unexplained lymphedema which appears at puberty; more common in females)
 - Immobility

#Symptoms related to medication

- **Dyspnea:** Beta-blockers in patients with asthma Exacerbation of heart failure by beta-blockers, some calcium channel antagonists (verapamil, diltiazem), NSAIDs
- **Dizziness:** Vasodilators, e.g. nitrates, alpha-blockers, angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor antagonists
- **Angina:** Aggravated by thyroxine or drug-induced anemia, e.g. aspirin or NSAIDs
- **Edema:** Steroids, NSAIDs, some calcium channel antagonists, e.g. nifedipine, amlodipine
- **Palpitation:** Tachycardia and/or arrhythmia from thyroxine, β 2 stimulants, e.g. salbutamol, digoxin toxicity, hypokalemia from diuretics, tricyclic antidepressants

#Features of infective endocarditis

- petechial rash
- hematuria
- splinter hemorrhage
- cerebral emboli
- weight loss
- tiredness
- fever, night sweating
- atrial fibrillation

#Clinical features of angina and MI

- Chest pain or discomfort
- Pain in your arms, neck, jaw, shoulder or back accompanying chest pain
- Nausea
- Fatigue
- Shortness of breath
- Sweating
- Dizziness

#Clinical signs of respiratory distress

- Tachypnea
- Expiratory grunting (from partial closure of glottis)
- Supraclavicular, intercostal, subcostal rescession
- Dusky appearance
- Cyanosis
- Nasal flaring
- Extremely immature in neonates may develop apnea and/or hypothermia

Notes هاهنا

- Clubbing + cyanosis occur in → bronchiectasis, malignancy, fibrosing lung diseases, cardiovascular (R to L shunt, fistula)
- Heart failure + dyspnea syndrome → congestive liver, medications, congestive uropathy, early features of uremia, inferior cardiac wall ischemia, congestive gastropathy
- Siluet sign → difficulty in determining the heart boundaries in X-ray due to lung diseases like fibrosis, consolidation, mass, effusion
- Examination of scalene L.N (Video)

Murmur	Systolic	Ejection systolic murmur	Pulmonary stenosis
			Aortic stenosis
			Atrial septal defect ASD
		Pan- systolic murmur	Mitral regurgitation
			Tricuspid regurgitation
			Ventricular septal defect VSD
	Systolic + diastolic	Machinery murmur	Patent ductus arteriosus PDA
	Diastolic	Early diastolic murmur	Pulmonary regurgitation
			Aortic regurgitation
		Mid-diastolic murmur	Mitral stenosis
Tricuspid stenosis			