Respiratory System

History and Symptomatology

History proforma

- Cough with expectoration
- Haemoptysis
- Dyspnoea
- Rule out CVS causes of breathlessness
- Chest pain
- Fever, evening rise of temperature, weight loss, night sweats
- Features suggestive of right heart failure (cor pulmonale)
- If suspecting bronchiectasis, complications of bronchiectasis
    - Oedema
- Past history of TB?
- Occupational history**

Chest pain- Unilateral chest pain usually points towards RS causes. Pleuritic chest pain characteristically increases on inspiration (coughing). If the pleural effusion is minimal, the layers of the pleura are more in contact which causes more irritation leading to more pain. Pleural disease can be associated with dry cough and stabbing type of chest pain.

Dyspnoea- MRC grading is preferred in RS. The advantages are: distance is checked, peer comparison and slope vs. level ground
Acute onset dyspnoea- pulmonary oedema, pulmonary embolism, acute exacerbation of COPD, acute severe asthma, status asthmaticus, one more asthma

Cough and Haemoptysis

Cough with expectoration history points:

- Onset, duration- TB, chronic bronchitis
- Postural variation- post nasal drip, bronchiectasis, left heart failure (**orthopnoea equalii)**, mass on trachea
- Diurnal variation: nocturnal cough- post nasal drip, paroxysmal nocturnal dyspnoea, bronchial asthma, cough variant asthma (no wheeze), GERD, Tropical pulmonary eosinophilia
- Colour of expectoration-
    - mucopurulent to purulent- bronchial/pneumonia infection;
    - Serous/frothy pink- pulmonary oedema;
    - blood stained- bronchiectasis, TB, pulmonary embolism, bronchogenic carcinoma
- Quantity- bronchorrhea>100ml/day (1 tumbler approx. 150-200 ml)
    - Causes-
        - bronchiectasis,
        - lung abscess,
        - empyema rupturing into a bronchus,
        - necrotising pneumonia
- Smell- foul smell in lung abscess, bronchiectasis
- Blood- frank haemoptysis vs. blood stained sputum

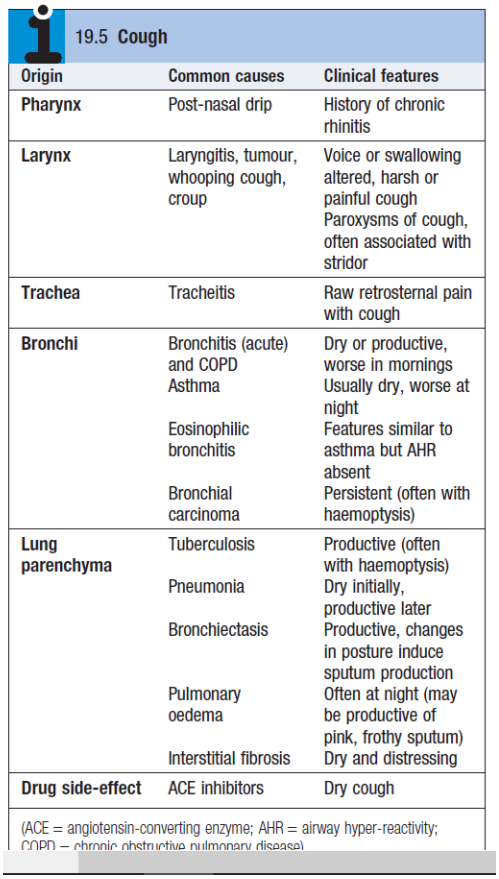
Haemoptysis

- Frank haemoptysis- expectoration of blood only
- Spurious haemoptysis- secondary to URTI
- Pseudohemoptysis- Serratia marcescens
- Endemic haemoptysis- Paragonimus westermani
- Severity
    - Mild: <100ml blood loss per day
    - Moderate: 100-150 ml
    - Severe: Up to 200 ml
    - Massive: >500 ml/day or Rate >150 ml/hr or 100 ml loss per day for more than 3 days.
- Causes*** (Dr. Vivekanandan)
    - RS
        - Airway-
            - Bronchitis(Ac & Chr),
            - Bronchogenic Ca,
            - Bronchiectasis and
            - Body(Foreign body)
            - *Note:*
                - *h/o repeated small haemoptysis/ blood streaked sputum is highly s/o carcinoma.*
                - *Bronchial bleed is more dangerous than a pulmonary artery bleed because the bronchial arteries are a part of the systemic circulation which are at a much higher pressure than the pulmonary circulation.*
        - Alveolar-
            - Pneumonia,
            - TB(**Fibrocavitary TB** (don't just say TB) due to rupture of **Rasmussen Aneurysms** (dilated pulmonary arteries surrounding the cavity)),
            - Lung abscess,
            - Acute pulmonary oedema (Pink frothy sputum)
        - Interstitial- ILD (rare)
        - Vascular- Pulmonary HTN, Pulmonary embolism, Pulmonary AV fistula
    - CVS-
        - Mitral stenosis,
        - Pulmonary embolism,
        - Pulmonary hypertension,
        - AV malformation
    - Collagen vascular disorders- Wegener's, Goodpasture syndrome, other vasculitis
    - Infective causes of haemoptysis- Weil's disease (severe Leptospirosis), Fibrocavitary TB, Invasive Aspergillosis, Paragonimiasis
- Massive Hemoptysis-
    - Causes: Bronchiectasis, Fibrocavitary TB, Aspergillosis, Paragonimiasis.
    - Management:
        - Intubate
        - Volume resuscitation
        - Blood transfusion
        - Bronchoscopy with balloon tamponade/Topical epinephrine or vasopressin

- Recurrent haemoptysis causes- bronchiectasis, chronic bronchitis, bronchogenic carcinoma
- Pseudohemoptysis- bleeding from upper respiratory tract or oral cavity
- Haemoptysis vs Haematemesis
    - Associated with cough, not with nausea and vomiting. Frothing present
    - pH Alkaline. Haematemesis is acidic. Food particles may be present in Haematemesis
    - Haematemesis may be associated with melaena

Cause of death in Hemoptysis: Asphyxia as d/t blood in bronchial tree.

h/s/o cor pulmonale- puffiness and swelling d/t pulmonary artery hypertension causing RV failure or even due to protein loss from the expectoration. Can also be due to amyloidosis.



19.5 **Cough**

Origin	Common causes	Clinical features
Pharynx	Post-nasal drip	History of chronic rhinitis
Larynx	Laryngitis, tumour, whooping cough, croup	Voice or swallowing altered, harsh or painful cough Paroxysms of cough, often associated with stridor
Trachea	Tracheitis	Raw retrosternal pain with cough
Bronchi	Bronchitis (acute) and COPD Asthma Eosinophilic bronchitis Bronchial carcinoma	Dry or productive, worse in mornings Usually dry, worse at night Features similar to asthma but AHR absent Persistent (often with haemoptysis)
Lung parenchyma	Tuberculosis Pneumonia Bronchiectasis Pulmonary oedema Interstitial fibrosis	Productive (often with haemoptysis) Dry initially, productive later Productive, changes in posture induce sputum production Often at night (may be productive of pink, frothy sputum) Dry and distressing
Drug side-effect	ACE inhibitors	Dry cough

(ACE = angiotensin-converting enzyme; AHR = airway hyper-reactivity; COPD = chronic obstructive pulmonary disease)

- Causes of dry cough-
    - pleural pathologies,
    - interstitial lung disease ILD,
    - cough-variant asthma,
    - tropical pulmonary eosinophilia,
    - atypical pneumonia,
    - GERD,
    - ACE inhibitors,
    - recurrent laryngeal nerve palsy
- Brassy cough- cough with metallic sound- compression over trachea
- Bovine cough- cough without its explosive nature- RLN palsy
- Post-tussive syncope: High intraThoracic pressure → decreases venous return → decreased cardiac output

Past history of TB

- Category
- Treatment taken? Treatment completed? Compliance?
- Side effects experienced?

Breathlessness

Modified MRC grading scale:
0- No breathlessness, except with strenuous exercise
1- Breathlessness when hurrying on level ground/walking up a slight hill
2- Walks slower than peers on level ground because of breathlessness/ has to stop for breath while walking at own pace
3- Stops for breath after walking about 100m or after a few minutes on level ground
4- Too breathless to leave the house/breathless when dressing and undressing
Note: No "Breathlessness at rest"
The advantages vs NYHA are:

- ☐ slope vs. level ground
- ☐ peer comparison and
- ☐ Distance is checked

Other classification- Sherwood Jones classification

- Receptors involved in dyspnoea *JSCR*
    - J receptors- alveolar capillary junction
    - Stretch receptors- thoracic cage and lungs
    - Chemoreceptors- carotid artery, aorta, medulla
    - Respiratory muscle receptors

CVS Breathlessness	RS Breathlessness
Associated CVS cardinal symptom(s)	No associated CVS symptoms
Orthopnoea, PND	Orthopnoea may be present (Asthma, COPD), but PND is specific to CVS

19.8 Differential diagnosis of acute breathlessness

Condition	History	Signs	CXR	ABG	ECG
Pulmonary oedema	Chest pain, palpitations, orthopnoea, cardiac history*	Central cyanosis, ↑JVP, sweating, cool extremities, basal crackles*	Cardiomegaly, oedema/pleural effusions*	↓PaO_2 ↓$PaCO_2$	Sinus tachycardia, ischaemia*, arrhythmia
Massive pulmonary embolus	Risk factors, chest pain, pleurisy, syncope*, dizziness*	Central cyanosis, ↑JVP*, absence of signs in the lung*, shock (tachycardia, hypotension)	Often normal Prominent hilar vessels, oligaemic lung fields*	↓PaO_2 ↓$PaCO_2$	Sinus tachycardia, RBBB, $S_1Q_3T_3$ pattern ↑T(V_1–V_4)
Acute severe asthma	History of asthma, asthma medications, wheeze*	Tachycardia, pulsus paradoxus, cyanosis (late), JVP →*, ↓peak flow, wheeze*	Hyperinflation only (unless complicated by pneumothorax)*	↓PaO_2 ↓$PaCO_2$ (↑$PaCO_2$ in extremis)	Sinus tachycardia (bradycardia in extremis)
Acute exacerbation of COPD	Previous episodes*, smoker. If in type II respiratory failure, may be drowsy	Cyanosis, hyperinflation*, signs of CO_2 retention (flapping tremor, bounding pulses)*	Hyperinflation*, bullae, complicating pneumothorax	↓ or ↓↓PaO_2 ↑$PaCO_2$ in type II failure ± ↑H^+, ↑HCO_3 in chronic type II failure	Normal, or signs of right ventricular strain
Pneumonia	Prodromal illness*, fever*, rigors*, pleurisy*	Fever, confusion, pleural rub*, consolidation*, cyanosis (if severe)	Pneumonic consolidation*	↓PaO_2 ↓$PaCO_2$ (↑ in extremis)	Tachycardia
Metabolic acidosis	Evidence of diabetes mellitus or renal disease, aspirin or ethylene glycol overdose	Fetor (ketones), hyperventilation without heart or lung signs*, dehydration*, air hunger	Normal	PaO_2 normal ↓↓$PaCO_2$, ↑H^+	
Psychogenic	Previous episodes, digital or perioral dysaesthesia	No cyanosis, no heart or lung signs, carpopedal spasm	Normal	PaO_2 normal* ↓↓$PaCO_2$, ↓H^+*	

*Valuable discriminatory feature. (ABG = arterial blood gases; RBBB = right bundle branch block)

Chest pain

Can originate from the parietal pleura, chest wall and mediastinal structures.
NOT LUNG Because the lung has only autonomic innervation, it does not cause chest pain.

	Parietal pleura	**Chest wall**	**Mediastinal structures**
Character	sharp, stabbing, intensified by inspiration or coughing. Upper 6 ribs irritation → localised pain. Lower rib pleural irritation can radiate to the upper abdomen. Diaphragmatic pleural irritation is referred to the shoulder tip/neck.	dull, aching, unrelated to respiration, progressively worsens and hinders sleep.	central, retrosternal, no relation to respiration
Causes	pulmonary embolism, pneumonia, pneumothorax, fractured ribs	invasion, trauma, compression.	lung cancer, lymphoma, thymoma, mets.

Specific history points in a suspected case of Tuberculosis

- Occupation of husband- high risk group for HIV
- Evening rise of temperature- due to TNFα
- Contact history of tuberculosis

Examination

Pulse: Comment on pulsus paradoxus. **>10mmHg is significant**. It reduces on treatment.

Pallor in RS-

- recurrent haemoptysis
- bronchial ca
- Tb

Cyanosis- SpO_2 <85%

Clubbing-

- Clubbing in TB- post TB bronchiectasis, scar carcinoma (it's an **Adenocarcinoma**), empyema, endobronchial TB

Lymphadenopathy-

- **Epitrochlear node-** Palpable epitrochlear nodes are always pathologic.
  - NHL,
  - secondary syphilis,
  - sarcoidosis
  - infections of forearm , hand
  - tularaemia (rare) **but not in TB** (Dr. Hamide)
- **Scalene node**- Place index finger between SCM and clavicle. Ask the patient to tilt his head to the same side and press firmly down towards the first rib

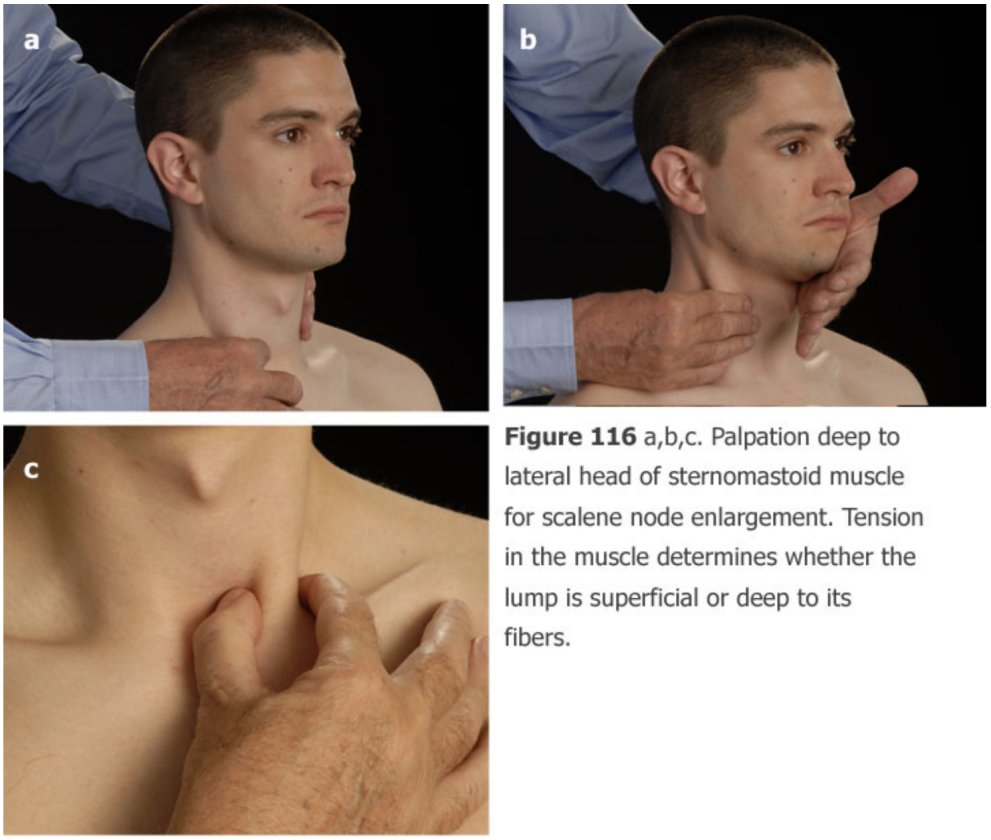


Figure 116 a,b,c. Palpation deep to lateral head of sternomastoid muscle for scalene node enlargement. Tension in the muscle determines whether the lump is superficial or deep to its fibers.

- Generalised lymphadenopathy- HIV, IM, sarcoidosis
- Mediastinal lymphadenopathy- dull percussion on manubrium sterni, bronchial breathing heard below the spine of T_4 (**d'Espine sign)**
    - *normally Tracheal bifurcation @ T4 → if sub carinal lymphadenopathy → conducts sound better (consolidation like) → bronchial breath*

Oedema- in RS, can be due to

- Cor pulmonale
- Protein loss in the sputum, esp in bronchiectasis
- Amyloidosis due to long standing bronchiectasis

Respiration: Normal rate-12 to 18/min
Women- thoraco-abdominal; Men- abdomino thoracic
If increased, check for **signs of respiratory distress**.

- **Alar nasii** enlargement,
- **pursed lip** breathing - help like PEEP
- **indrawing** of trachea
- use of accessory muscles of respiration like **sternocleidomastoid** and recti,
- intercostal **retraction**

Muscles of respiration - I always with E (insp → external ; vice versa)

- **Inspiration**
    - Principal- ***External*** intercostal muscles, diaphragm
    - Accessory- Sternocleidomastoid (elevates sternum), Scalenes (elevate upper ribs), Pectoralis minor, Trapezius
- **Expiration**
    - Quiet breathing- passive
    - Active- ***internal*** intercostals (pulls ribs down), Abdominal muscles (pull ribs down, compress abdominal contents pushing diaphragm up), Quadratus lumborum (pull ribs down)

RS Examination
External markers of Tuberculosis-

1. phlycten : small pinkish yellow nodule near the limbus
2. choroid tubercles - not visible to naked eye
3. scars/sinuses,
4. scrofula : tuberculous lymphadenitis(Painless) in cervical region usually , can be non Tb
5. Skin
    a. lupus vulgaris - painful cutaneous granuloma with nodular appearance(brown jelly/apple jelly nodules) : nose, eyelids , cheek ,ear neck
    b. erythema nodosum,
    c. Tuberculous Verrucous cutis: Direct inoculation of TB over skin → Solitary plaque with central Involution and atrophic scar.
    d. ScrofuloDerma: Direct skin extensions from TB of LN, Bones and joints.
6. cold abscess/collar stud abscess,
7. epididymo orchitis

Other things in G/E:

1. Horner's syndrome in lung malignancies
2. Signs of neurovascular compression : Wasting of small muscles of hands

3. Dahl's sign (also Thinker's sign or Target sign):
    a. areas of darkened (hyperpigmentation) and thickened (hyperkeratotic) skin are seen on the lower thighs and elbows.
    b. Air trapping in the lungs of COPD patients causes the diaphragm to be pushed down and flattened, which reduces the effect of contraction of the diaphragm during inspiration. Sitting forwards pushes the abdominal contents upwards, increasing the curvature of the diaphragm and improving its effectiveness.
    c. Long standing COPD pts tend to sit forwards with their arms resting on their thighs, leading to chronic erythema of the skin at the points of contact. Over time, hemosiderin released from red blood cells trapped in the skin is released causing a brown discolouration of the skin.

Inspection

Inspection of upper respiratory tract:

oral cavity- thrush (immunocompromised state) , tonsils; poor oral hygiene , halitosis → lung abscess
nose- DNS, polyps; pharynx- post nasal drip - cause even these can cause respiratory symptoms and breathing difficulty

Tracheal position: affected mainly by upper lobes
Trail's sign- undue prominence of the clavicular head of SCM on the side to which the trachea is deviated. The pretracheal fascia encloses the clavicular head of SCM on both sides. When the trachea is shifted to one side, the fascia on that side relaxes, making the clavicular head more prominent.
If no deviation "Trachea appears central"

Apex beat position- mediastinal shift.

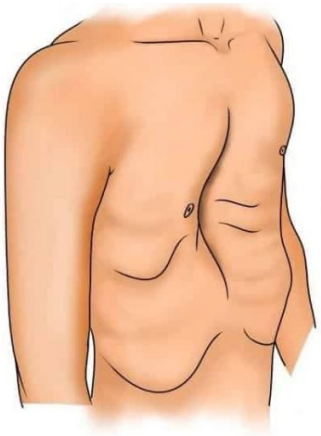
Skin over chest wall- engorged veins (malignancy), intercostal scar (drained pleural effusion, empyema, pneumothorax), discharging sinuses (TB)

Signs of lung volume loss-

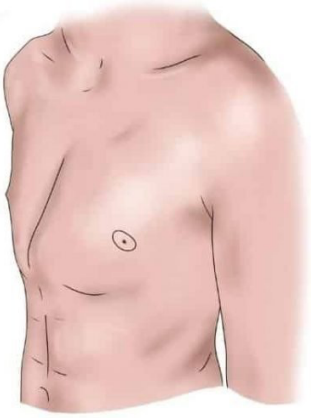
- shoulder **droop**,
- **prominence** of medial border of scapula (don't call it winging),
- **crowding** of ribs.

Symmetry of chest: Normal chest is symmetrical and elliptical in cross section. AP to transverse diameter ratio is **5:7**.

- **Flat chest-** AP to transverse diameter is **1:2**. Seen in **pulmonary TB, fibrothorax**
- **Barrel** chest- **1:1**. Infancy, old age, emphysema, acromegaly
- **Pectus carinatum** (pigeon chest)- Associated with **rickets**
    - forward protrusion of sternum
    - depression on either side of the sternum.
- **Pectus excavatum** (funnel chest, cobbler's chest-
    - sunken appearance of sternum
    - most commonly a cup-shaped concavity

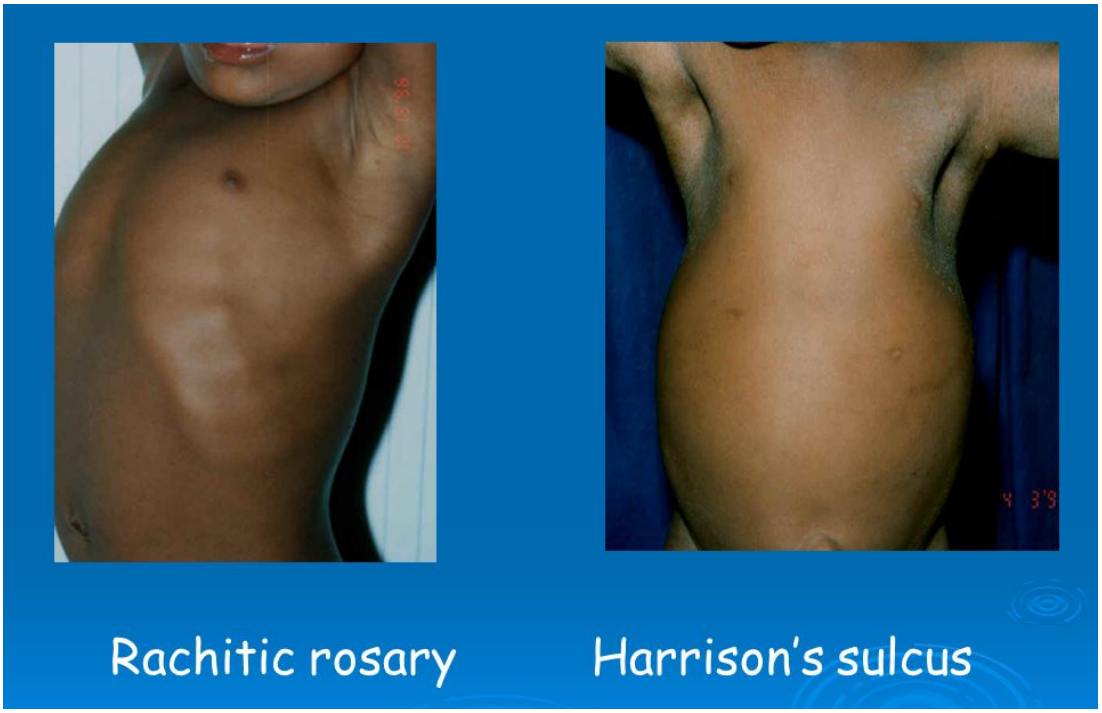


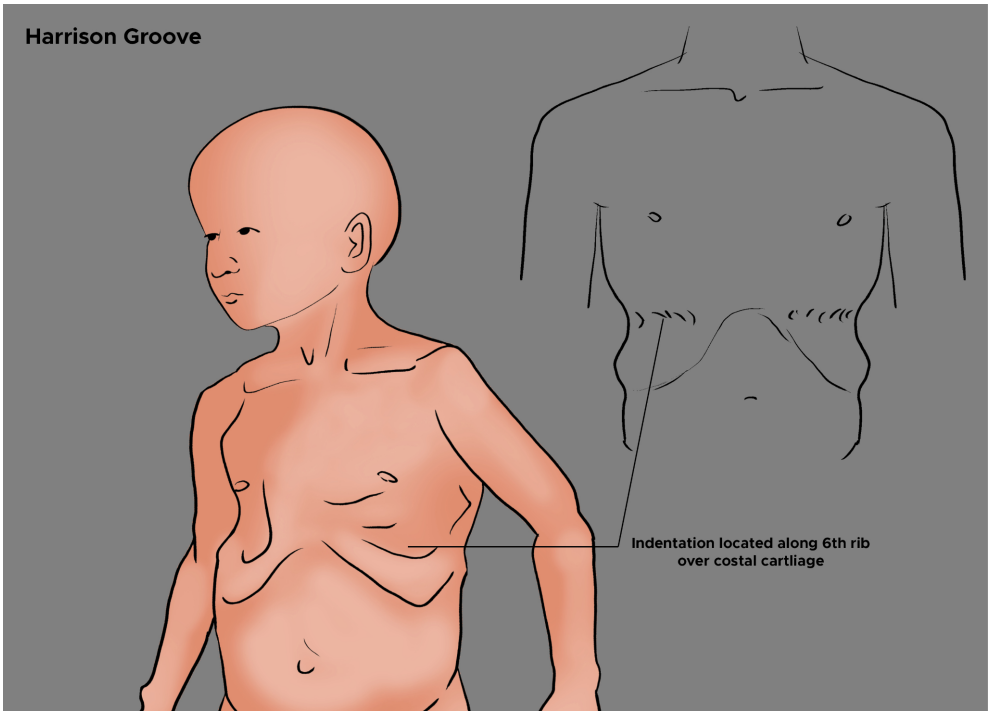
Pectus Excavatum



Pectus Carinatum

- **Harrison's sulcus**- chronic respi disease in child ; rickets
    - symmetrical horizontal grooves above the costal margin along the line of attachment of diaphragm.
    - d/t lung hyperinflation and repeated strong diaphragmatic contraction like in chronic respiratory disease in childhood.
- **Rachitic rosary**- **bead** like enlargement of **costochondral** junction





- **Flail Chest:** secondary to multiple rib fractures, depression of diaphragm causes the injured area to cave inward producing a "paradoxical thoracic movement" in breathing.

Inspection of the back

- Kyphosis- forward curvature
- Scoliosis- lateral curvature
    - In fibrosis, the fibrosed lung pulls both ends of the vertebral column towards te fibrosed side, so the convexity of the curvature is towards the opposite side

PALPATION

Movements

Chest expansion- Normal= 5 to 8cm. **Emphysema, ILD, Ank Spon- <1 cm**

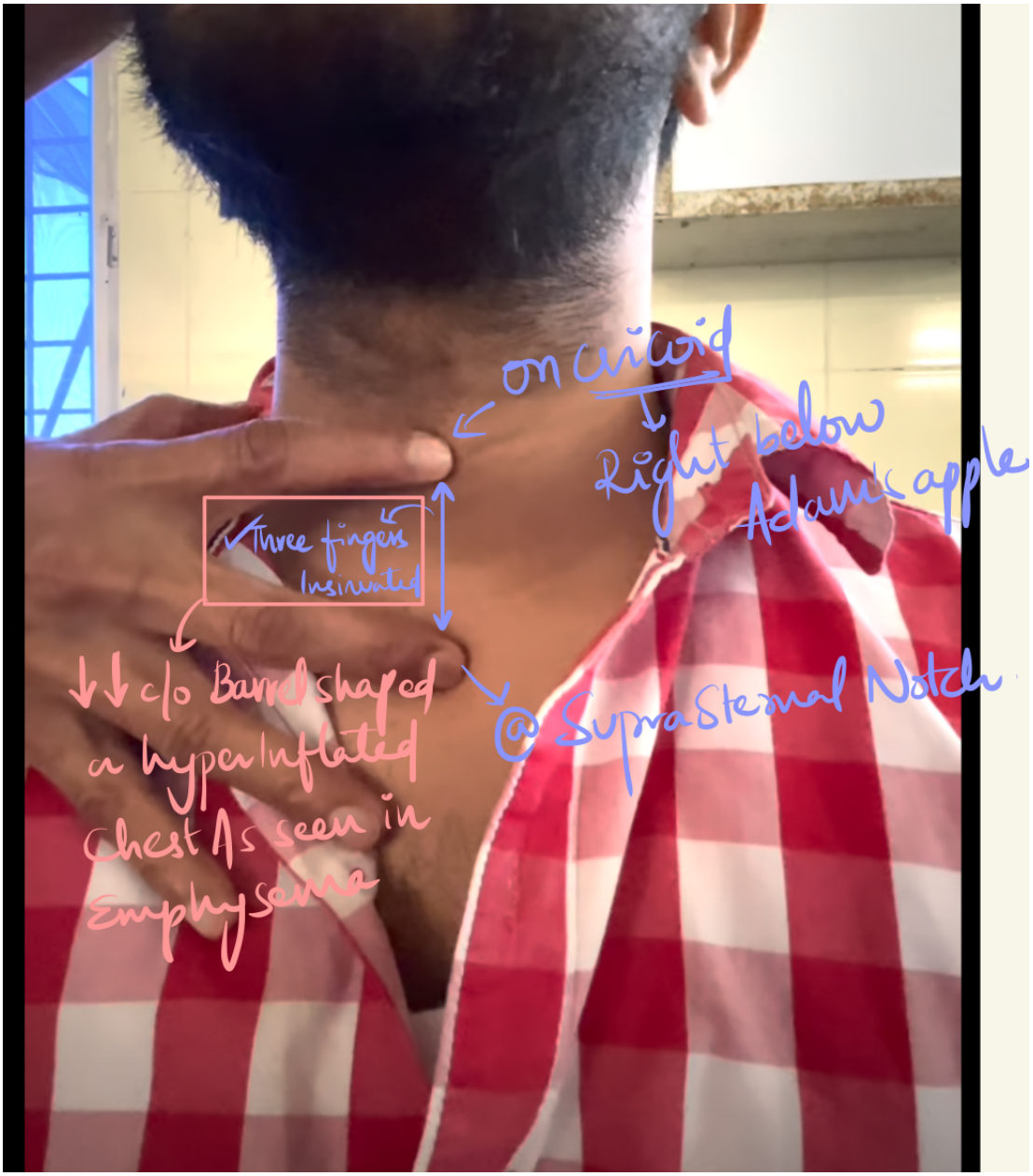
- Chest circumference in inspiration: ____ cm
- Chest circumference in expiration: ____ cm
- Hemithoracic expansion
- Apical chest expansion-
    - put hands over supraclavicular fossae
- Anterior thoracic movement
    - hand placed on side of chest with thumbs infront & separation of thumbs with respiration is observed
- Posterior thoracic movement
    - same as anterior for lower lobes ,
    - upper lobes - place hand over the TRAPS and compare movements
- Vertebroscapular distance
- Note:
    - Metal end of the tape is kept in front while measuring chest circumference
    - Metal end of the tape is kept on the spines while measuring hemithoracic because that is the fixed point
    - Measure in full expiration first

Tracheal position:

- Slight tracheal deviation to the right is normal.
- Pushed to the opposite side-
    - pleural effusion,
    - pneumothorax,
    - apical tumour
- Pulled to the same side-
    - Fibrosis,
    - collapse
- Shifted to the same side in effusion- co existent fibrosis/collapse, **mesothelioma**

CricoSternal distance:

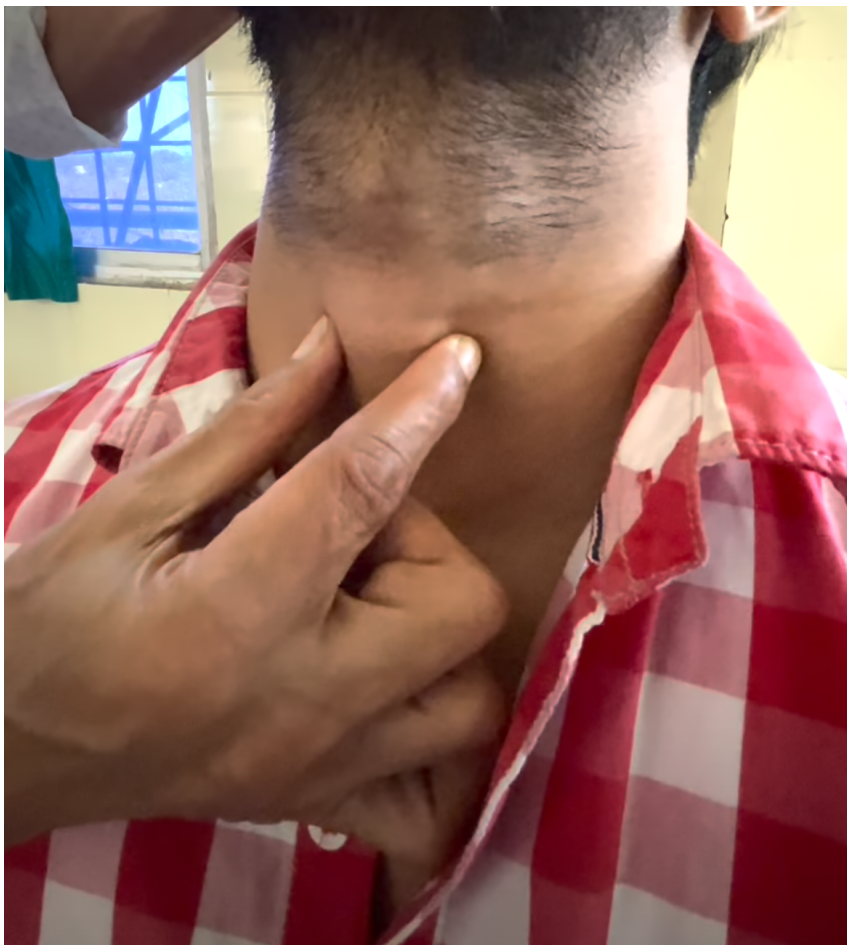
Measured as number of fingers that can be insinuated. Reduced in COPD bc the Mediastinum is pulled down pulling the cricoid also downward.



Inspiratory tracheal descent- COPD- **Campbell's sign**

<u>Tracheal tug- Oliver's sign</u>

- Patient's chin is raised
- Cricoid cartilage held up on deglutition on either side with fingers of both hands
- Positive- downward tug felt- Aortic arch aneurysm
- False positive- mediastinal tumour attached to the arch
- False negative- non pulsatile thrombosed aortic aneurysm



Chest wall tenderness- Empyema, pleuritis, infiltration of chest wall
Subcutaneous emphysema

Fremitus

Vocal- increased in consolidation, decreased in effusion
Tactile- palpable added sounds- **crackles**, **rhonchi**
Friction- palpable pleural **rub**

Various conditions affecting VR/VF and Percussion

- Transmission(speed) of sound
    - Solid >liquid> air
- Ability to vibrate
    - Air > liquid > solid
- **VR / VF** : Measure the medium's ability to **transmit sound** (from vocal cords)
- **Percussion** : Measure medium's ability to **vibrate**
- Lesions where LUNG to SKIN distance is altered --- As distance increase ---- more attenuation of sound produced (VF/VR/BREATH SOUND) while nature of vibration of medium remain same

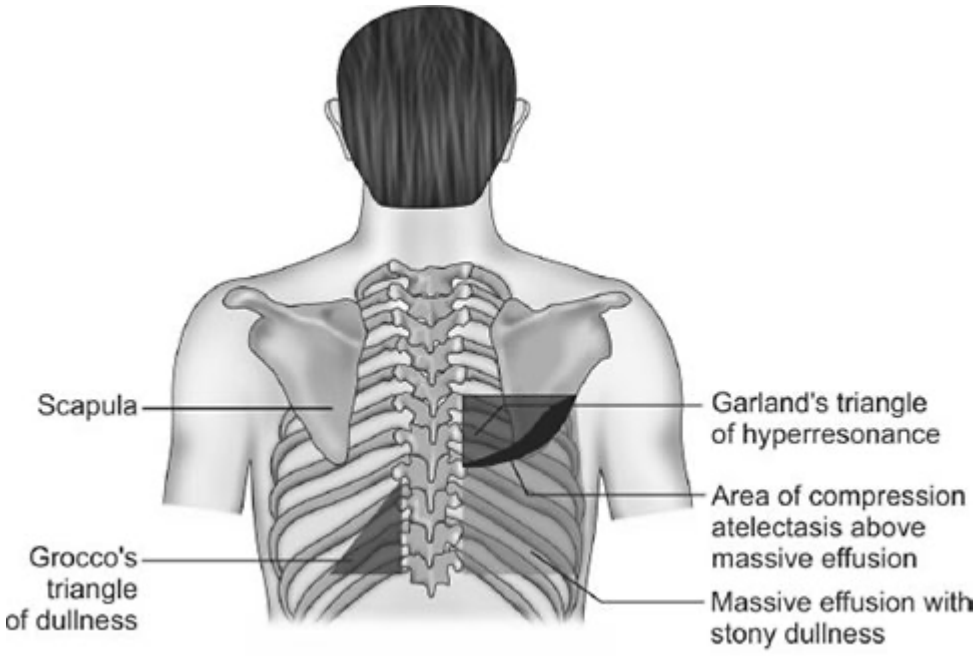
Pathology	What happens	VR/VF	Percussion
CONSOLIDATION	fluid in parenchyma	increased (fluid sound faster)	DULL (fluid vibrate less)
HYPERINFLATION	More air replaced in parenchyma	reduced (air sound slower)	RESONANT (air vibrate more)
PL.EFFUSION	Fluid around lung + lung far from chest wall	reduced (since lung - chest distance more)	STONY DULL
PNEUMOTHORAX	Air around lung + lung far from chest	reduced	Resonant (air vibrate more)
COLLAPSE	Lung far from chest	Reduced	Dull (collapse form solid like area)

<u>Percussion -</u> always compare with other side right after

- Anterior percussion- hand by the side
- Posterior percussion- <u>head bent forward</u>, hands on opposite shoulder
- Lateral percussion- hands held over head
- **<u>Areas:</u>**

<u>Anteriorly</u>	**<u>Posteriorly</u>**
Supraclavicular	Suprascapular
Clavicular	Interscapular
Infraclavicular- up to 2nd ICS	Infrascapular
Mammary 2nd to 6th ICS	
Axillary up to **4th** ICS	
Infra axillary **4th to 7th ICS**	

  - Also percuss for **Liver dullness.** Normal upper level of liver dullness is the 5th ICS. If <u>pushed down, it suggests emphysema (Primary or compensatory)</u>
  - In left sided pathologies, percuss over Traube's space also
  - Grocco's triangle- Area of dullness on percussion against the spine on the base of the opposite lung. Reason? (Possibly due to posterior mediastinal shift)
    - *In the presence of a pleural effusion, the posterior mediastinal pleura may bulge into the contralateral hemithorax. This may result in a triangle of dullness to percussion in the contralateral hemithorax. Grocco's triangle is considered pathognomonic of pleural effusion.*



Triangular area of dullness at the back of chest on the healthy side

Base – horizontally along the XII th rib

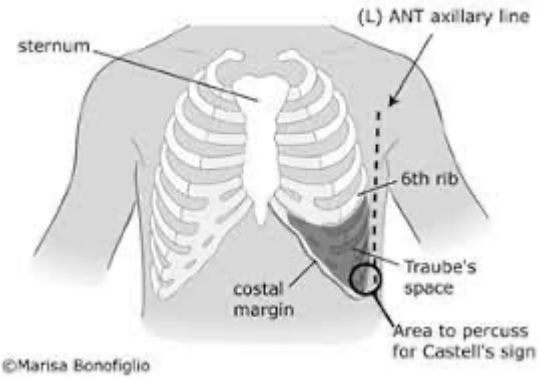
Apex – at the level of upper margin of fluid on diseased side

Internally – vertebral line

Externally – line joining the apex and lateral base

- **<u>Kronig's isthmus-</u>** Band of resonance 5-7cm over the supraclavicular fossa.
    - borders :
        - Scalenus medius,
        - acromion process laterally,
        - anteriorly clavicle,
        - posteriorly trapezius.
    - This percussion is done by **standing behind** the patient. from **lateral to medial** ; **clavicle not touched**
    - It indicates the condition of the lung apex.
        - Increase in size of this area- emphysema, decreased size- TB/apical lung tumour.
- Tidal percussion- infradiaphragmatic vs. supradiaphragmatic pathologies.

<u>TRAUBE'S SPACE</u>



6th rib superiorly, left midaxillary line laterally, left costal margin inferiorly
Right side- Left lobe of the liver
Left side- spleen
Superiorly- left lung resonance
Inferiorly- left costal margin
Content- Fundus of the stomach

- Obliterated in-
    - Left pleural effusion ,
    - massive splenomegaly,
    - enlarged left lobe of liver,
    - full stomach, massive pericardial effusion
- Shifted upwards in- left diaphragmatic paralysis, left lower lobe collapse, fibrosis of left lung

__Percuss the clavicle__- *__stretch the skin below__*. Percuss the medial 1/3 with your middle finger because there are less muscle attachments on the medial 1/3. Purpose of clavicular percussion is because it is near the apex of the lung. **So a dull note over here would represent a secondary TB.

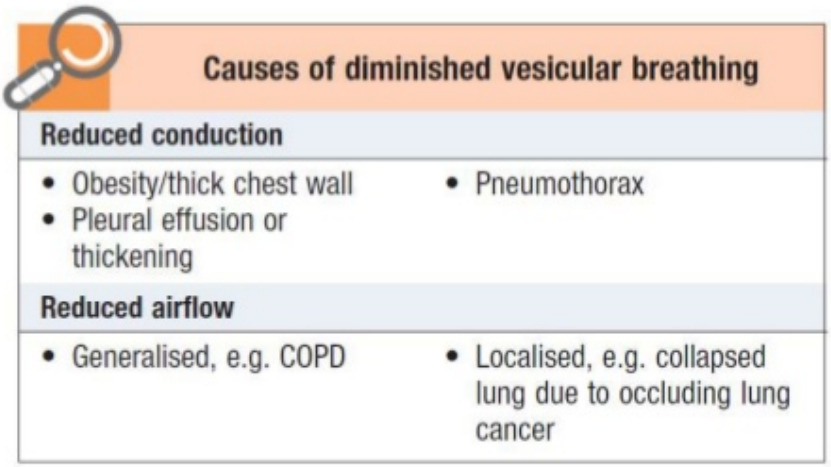
- **Shifting dullness**- Find the upper border of posterior dullness, then ask the patient to lean forward. If the dullness is immediately seen then it is suggestive of hydropneumothorax. Even effusion will have shifting dullness, but it will take time.
- **Straight line dullness**- hydropneumothorax
- **Succussion splash**- elicitable in hydropneumothorax. Percuss the upper limit of dullness. Place the stethoscope and hold it firmly just above the upper limit of dullness. Hug the patient and shake him moving along with him. You will hear a splash similar to the splash that can be heard over stomach containing air and fluid.
- **Silver coin sounds**- hit one coin with the other and place the steth on the back. The clinking sounds are audible in hydropneumothorax
- **S shaped curve of Ellis-** Seen in moderate pleural effusion. Uppermost dullness is highest in the axilla. Lowest in spine posteriorly and near sternum anteriorly.
    - Reason- ***capillary suction between two layers of pleura***. Also seen **radiologically**, believed to be due to scattering of X rays by the fluid

In shifting dullness due to hydropneumothorax , **__we don't wait for 20 seconds like ascites__** because in the abdomen the air and fluid are in different compartments, so we need to give time for the loops of bowel to move. Whereas in the lung, the air and fluid are in the same compartment so there is no need to give time for movement.

__AUSCULTATION__

__Vesicular breath sounds__

- **Low pitched**, rustling in nature
- **Inspiration: Expiration is 3:1**
- **No pause** between end of inspiration and beginning of expiration
- Diminished VBS- bronchial asthma (silent chest), tumour, small effusion, pleural thickening



Causes of diminished vesicular breathing	
Reduced conduction	
• Obesity/thick chest wall • Pleural effusion or thickening	• Pneumothorax
Reduced airflow	
• Generalised, e.g. COPD	• Localised, e.g. collapsed lung due to occluding lung cancer

Bronchial breath sounds

- **Loud, high pitched**
- **I:E is 1:1**
- **Pause** between end of inspiration and beginning of expiration
- Causes of bronchial sounds- (**Dr. Vivekanandan)**
    - **Consolidation**- Tubular,
    - **Cavity** which is large and **superficial** lying and **thin walled**. Produces cavernous or amphoric (**Amphoric** is produced only in cases of **narrow** mouthed cavity). Lung abscess will not have bronchial sounds because it is **thick walled**
    - **Collapse**- provided major *bronchus is patent*
    - **Upper border of pleural effusion**- due to collapse with patent bronchus at the top. Fluid conducts the bronchial sounds as well but the sound waves get reflected, hence the bronchial sounds are best heard at the upper border where there is no fluid
    - **Fibrosis**. *Generally causes diminished BS*, but sometimes causes bronchial sounds when in the **upper lobe** because it **pulls the bronchus towards it**
    - **Pneumothorax**. *Generally causes reduced BS*, but in **bronchopleural fistula** there is bronchial breath sounds
- **Tubular** bronchial sounds - **high** pitched, heard in
    - consolidation,
    - collapse,
    - above the level of effusion
- **Cavernous** bronchial sounds- **low** pitched, heard in the presence of a **cavity**. *Can be mimicked by blowing into cupped palms*
- **Amphoric** bronchial sounds- **low pitched, high tone**. Seen in cavity, bronchopleural fistula, tension PTX. *Can be mimicked by blowing intensely across the mouth of a bottle*

Crackles=Crepts=Rales

- ***Non musical, interrupted, discontinuous added sounds of short duration****
- d/t bubbling of air through secretions or opening of airways that were occluded
- d/t sudden opening of previously closed alveoli (**Dr. Vivekanandan**)
- **Fine crackles**- less loud, **short** in duration, arise from **alveoli**. *Early consolidation* (indux crepitation), fibrosis
- **Coarse** crackles- **low pitched, loud**, arise from **bronchus** and **bronchioles**. *Resolving consolidation*
    - Seen in chronic bronchitis,
    - bronchiectasis,
    - fibrosis, ILD-(Velcro crackles),
    - pulmonary oedema
- *Crackles without sputum- ILD; crackles with sputum- parenchymal disease*
- **Death rattle-** coarse crackles heard in gross pulmonary oedema
- Consolidation, pulmonary oedema- fine/coarse crackles
- Pulmonary oedema, ILD- late inspiratory crackles
- ILD, fibrosis- fine crackles
- Bronchiectasis- coarse crackles
- Post tussive crepitations- crepitations evoked by coughing is suggestive of **cavity**

MacLeod table- causes of crackles

Causes of Crackles	
Phase of inspiration	**Cause**
Early	Small airways disease, as in bronchiolitis
Middle	Pulmonary edema
Late	Pulmonary fibrosis (fine crepitations) Pulmonary oedema (medium crepitations) Bronchial secretions in COPD, pneumonia, lung abscess, tubercular lung cavities (coarse crepitations)
Biphasic	Bronchiectasis (coarse crepitations)

Rhonchi- snoring quality

- **Musical, continuous added sounds**
- Sonorous- low pitched, arising from large airways
- Sibilant- high pitched, from smaller airways

Wheeze

- Wheeze is a symptom, Rhonchi is a sign (Dr. Vivekanandan)
- Cause of Rhonchi- vibration of narrow bronchus
- Wheeze is audible even without stethoscope
- Rhonchi are continuous musical sounds
- **Fixed monophonic** wheeze- single note of constant pitch, timing, site.
    - due to **localized** airway narrowing : tumour / foreign body
- **Random monophonic** wheeze- random single note : Bronchial **asthma**
    - varying in
        - phase of respiration
        - site,
        - duration and
        - pitch.
- **Expiratory polyphonic** wheeze- Complex sound with multiple notes with all its components starting together and ending in expiration due to **compression** of **central airways**- **Emphysema**
- Sequential inspiratory wheeze-

Vocal Resonance

- ***Bronchophony***-
    - "Phenomenon of the patient's voice **remaining loud** at the **periphery** of the lungs or **sounding louder than usual** over a distinct area of **consolidation d/t reduced acoustic filtering**, thus increasing transmission "
    - feels as if the sound is coming from the ear directly and not from chest piece of steth

- ***Aegophony***- E to A.
    - Differentiates between the crackles seen in consolidation (aegophony present) from IPF (absent) ; in both cases VR/VF reduced and percussion is impaired-dull in fibrosis
- ***Whispering pectoriloquy-*** whisper at the **end of expiration** and the words are transmitted **without distortion** and the individual words are recognised clearly. Seen in consolidation.

D'Espine sign

- Whispering pectoriloquy is not normally heard below T3/T4 - cause trachea bifurcates at this level
- D'Espine sign is said to be positive if it is heard below this level. Seen in
    - Mediastinal enlargement
    - Posterior mediastinal tumour
    - Patch of central pneumonia
        - these above mentioned conduct sounds from trachea causing whispering pectoriloquy

Notes

Breath sounds

- Mode of production- by **turbulent** airflow in the **upper airways** (ie pharynx and larger airways of the lungs)
- as transmission happens → breath sounds dampen → higher pitch lost → low pitch softer sounds heard
- Types of normal breath sounds

	VESICULAR	**TRACHEAL**	**BRONCHOVESICULAR**
quality	vesicular , normal lung	blowing or tubular	intermediate of these 2
Ins : Exp	3:1	1:1	
EXPIRATION compared to inspiration	lower pitch, low intensity	higher pitch , high intensity	
PAUSE b/w insp , exp	ABSENT	PRESENT	

Abnormal breath sounds

- Exaggerated/loud sounds-
    - Dyspnoea,
    - compensatory emphysema in response to effusion, pneumothorax etc
- Diminished/feeble sounds-
    - pleural effusion,
    - hydrothorax,
    - pneumothorax,
    - lung fibrosis/emphysema (loss of elasticity),
    - weakness of respiratory muscles like GBS

Percussion

- Types of percussion notes
    - Normal lung resonance
    - Impaired resonance- there is still resonance, but it is less- seen in Fibrosis
    - Dull- Absence of resonance- Consolidation, collapse
    - Stony dull- We feel pain or resistance because the impact is not absorbed due to fluid over the lung parenchyma. The note can be imitated by percussing over the thigh.
    - Tympanitic note-
        - drum like note, commonly elicited over stomach, intestine, trachea
        - Seen in superficial cavity or pneumothorax
    - Skodiac resonance
        - Hyperresonant note with a boxy quality heard just above the level of effusion or consolidation
        - Results from the fluid or consolidation causing the lung partially filled with air to relax upward and towards the hilum
    - Hyperresonance
        - Intermediate in pitch between normal and tympany
        - **Heard normally in full inspiration**
        - Bilateral hyperresonance-
            - emphysema
        - Unilateral-
            - pneumothorax,
            - compensatory emphysema (ex: if there is RUL collapse, there can be compensatory RLL emphysema)
- Cracked-pot resonance: type of resonance that is found in a cavity communicating with a bronchus. Imitated by clasping the hands loosely together and striking against the knee
- Tidal percussion.

Pleural rub vs. crackles

- Rub is **superficial** and **loud**
- **Continuous**
- Localised
- Unaffected by cough
- *Pressure with steth over chest wall increases the sound*
- Associated with **pain** and **tenderness**

Hamman's mediastinal crunch- Clicking rhythmical sound synchronous with cardiac cycle. Due to mediastinal emphysema

Diaphragmatic palsy

- Litten's sign
    - Diaphragm is attached to 7^{th}-10^{th} ribs
    - Observe the shadow of the ribs in sunlight and see the movement

Pleural effusion

Workup - Chest x ray, USG, Diagnostic tap

Diagnostic tap- Glucose, protein, LDH, cells, GeneXpert, Adenosine deaminase (ADA) ADA-2 isoenzyme is more specific for M.tb whereas ADA-1 can be elevated in other bacterial causes also

Presence of which cell in tap rules out TB? **Mesothelial cells** (**Dr. Hamide**)

- *because intense lymphocytic infiltration of pleural surfaces prevents mesothelial cell entry in pleural space - NCBI*

- Principle of treatment- Identify if exudate or transudate. If transudate, correct the underlying(systemic) cause. If exudate, consider TB, give ATT. Otherwise, if glucose <60 mg/dL, consider parapneumonic effusion, malignancy

*****Characteristics of a Tubercular Pleural Effusion**

- Usually associated with primary TB, believed to be a hypersensitivity reaction to the tuberculous protein in the pleural space.
- Straw coloured exudate, sometimes haemorrhagic
- Small lymphocytes predominate
- TLC 5000-10000
- Adenosine deaminase >40 U/L, IFN γ >140 pg/ml (Harrison's) (ADA> 40 is suggestive of a tubercular aetiology while >60 is highly suggestive)
- Mesothelial cells almost always absent
- GeneXpert can be done.
- Treatment is ATT

Light's Criteria- Pleural fluid is said to be an exudate if it **meets at least one** of the following

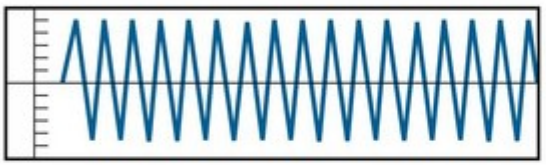
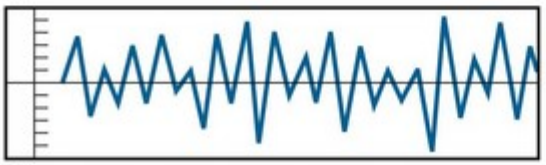
- Pleural fluid protein/Serum protein >0.5
- Pleural fluid LDH/Serum LDH >0.6
- Pleural fluid LDH more than 2/3 of the **normal upper limit** for serum

Light's criteria misidentifies almost 25% of transudates as exudates. If the patient is thought to have a transudative effusion despite Light's calling it an exudate then ***Serum Fluid Protein Gradient (SFPG) can be checked. >3.1 g/dL means transudative.***

- Pleural fluid **N-terminal pro-brain natriuretic peptide** is virtually diagnostic of an effusion is secondary to CHF.

The harmful effects of smoking cease in about 8 years (Dr. Hamide). Smoking has a protective effect vs. **ulcerative colitis and Parkinson's**.

Abnormalities in pattern of respiration

Cheyne-Stokes Respiration	 Tidal volume (V_T) with a progressive shallow-deep-shallow cycle (30 sec–2 min)	CVA, trauma, or masses involving a diffuse area of the forebrain, carbon monoxide poisoning, metabolic encephalopathy, altitude sickness, opiate effect
Biot's (Cluster) Respiration	 Periods of rapid respirations of nearly equal depth or V_T followed by regular periods of apnea	CVA or trauma involving the medulla or lower pons, mass effect on the medulla secondary to uncal or tentorial herniation, opiate effect
Kussmaul Respiration/ Hyperpnea	 Hyperventilation characterized by a consistent and deep respiratory pattern; the RR of patients with hyperpnea may be normal or rapid with increased V_T	Severe metabolic acidosis; Kussmaul respiration in diabetic ketoacidosis starts with a rapid and shallow pattern and later becomes rapid and deep
Apneustic Respiration	 Prolonged inspiratory phase followed by prolonged expiratory phases believed to be apneic phases	CVA, trauma, or mass effect involving the pons
Ataxic Respiration	 Erratic breathing pattern with irregular pauses and increasing episodes of apnea (progresses to agonal respiration)	CVA or trauma involving the medulla (indicates a very poor prognosis)

Cheyne-Stokes: Crescendo->Peak->decrescendo->apnoea-> repeat cycle.
Causes- **congestive heart failure**- decreased cardiac output leads to a lag between A-a CO_2. Lower alveolar CO_2 is reflected much later in the blood that bathes the medulla, and the medulla is highly sensitive to a decreased CO_2 concentration.
CNS disorders- meningitis, CVA, trauma: d/t heightened sensitivity of the centres to CO_2.

Biot's: Variant of Cheyne- Stokes. Hyperpnoea-apnoea cycles without crescendo-decrescendo pattern. Seen in meningitis, medullary compression. Worse prognosis

Apneustic breathing: Deep inspiratory phase->breath holding->rapid exhalation. Seen in brainstem, esp pons lesions.

Central Hyperventilation: Hyperpnoea and tachypnoea, but not as deep as Kussmaul's. Seen in brain stem lesions.

Ataxic ventilation: "Respiratory fibrillation". "Agonal respiration"- sign of impending death

Grunt:

- **Short** and **low pitched noise** produced by forced expiration against a closed glottis.
- *Grunting – Grunting is an end expiratory sound that occurs as the result of exhalation against a partially closed glottis. It slows expiratory flow and increases lung volume and alveolar pressures.* (UpToDate)
- Acts as a PEEP, like pursed lip breathing. Seen in adults in **respiratory muscle paralysis and in children in pneumonia**.
- Prevents **transudation in pulm oedema so also seen there**.

Tachypnoea- >20/min. Exertion, fever, pulm oedema, pneumonia, ARDS, acidosis
Apnoea- absence of respiration for at ***least 20 seconds while awake and 30 seconds while asleep.***
Hyperpnoea- Increase in **tidal volume** (usually with, but sometimes without increase in rate). Causes- **MAKE UP**-

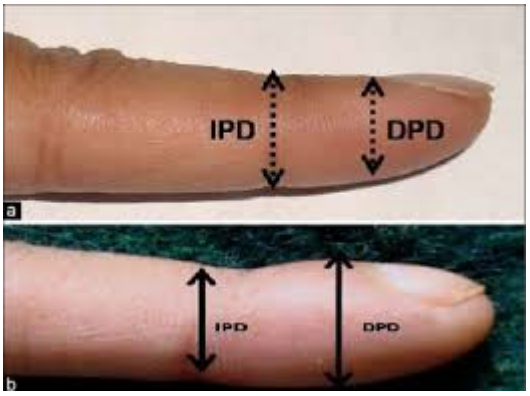
- methanol poisoning,
- aspirin overdose,
- KetoAcidosis ,
- ethylene glycol,
- uraemia,
- paraldehyde, lactic acidosis (HAGMA causes).

In cardiorespiratory disease, tachypnoea rather than hyperpnoea compensates because vital capacity is compromised.
Bradypnoea- <8/min. Causes-

- CNS depression- OP poisoning, Opiates;
- Nerve injury, muscle, NMJ, Alkalosis, Hypothyroidism

CLUBBING

Phalangeal Depth Ratio
Normally, DPD/IPD <1. In clubbing, >1. Excellent marker.



****Causes of clubbing-**

Congenital/Familial
Acquired
- Thoracic- Suppurative lung diseases (Bronchiectasis, Empyema, Lung Abscess, CF)
  - Malignancy- Bronchogenic carcinoma, Mesothelioma, Bronchial adenoma
  - Interstitial lung disease
  - Asthma and COPD do not cause**
- Cardiovascular- CCHD, Einsenmenger, IE, Aneurysm?, LA myxoma
- Gastrointestinal- Cirrhosis (esp PBC), Inflammatory bowel disease, Coeliac disease
- Others- Grave's disease- Thyroid acropachy, POEMS syndrome

Pseudoclubbing- increased curvature of the nail in longitudinal and transverse axes that **preserves Lovibond angle**. Seen in chronic lung disease like TB, bronchogenic ca.

HOA- (Marie Bamberger syndrome) Chronic, symmetric, proliferative periostitis of long bones often associated with clubbing. It is a paraneoplastic syndrome, usually of intrathoracic neoplasms like bronchogenic ca, lymphoma, mesothelioma or even mets. Diaphysis of the extremities are commonly affected. Rad>Ulna>Tibia>Femur.
Other features- symmetric arthritis like changes in one/more joints, coarsening of subcutaneous tissue in distal portions of arms and legs, neurovascular changes in arms and feet- sweating, erythema, paresthesias.

Thyroid acropachy- Thickening of peripheral tissues with clubbing, seen in 1% of Graves cases.

19.15 Pleural effusion: main causes and features

Cause	Appearance of fluid	Type of fluid	Predominant cells in fluid	Other diagnostic features
Tuberculosis	Serous, usually amber-coloured	Exudate	Lymphocytes (occasionally polymorphs)	Positive tuberculin test Isolation of *M. tuberculosis* from pleural fluid (20%) Positive pleural biopsy (80%) Raised adenosine deaminase
Malignant disease	Serous, often blood-stained	Exudate	Serosal cells and lymphocytes Often clumps of malignant cells	Positive pleural biopsy (40%) Evidence of malignancy elsewhere
Cardiac failure	Serous, straw-coloured	Transudate	Few serosal cells	Other signs of cardiac failure Response to diuretics
Pulmonary infarction	Serous or blood-stained	Exudate (rarely transudate)	Red blood cells Eosinophils	Evidence of pulmonary infarction Obvious source of embolism Factors predisposing to venous thrombosis
Rheumatoid disease	Serous Turbid if chronic	Exudate	Lymphocytes (occasionally polymorphs)	Rheumatoid arthritis: rheumatoid factor and anti-CCP antibodies Cholesterol in chronic effusion; very low glucose in pleural fluid
SLE	Serous	Exudate	Lymphocytes and serosal cells	Other signs of SLE Antinuclear factor or anti-DNA positive
Acute pancreatitis	Serous or blood-stained	Exudate	No cells predominate	Higher amylase in pleural fluid than in serum
Obstruction of thoracic duct	Milky	Chyle	None	Chylomicrons

(anti-CCP = anti-cyclic citrullinated peptide; SLE = systemic lupus erythematosus)

****Lymphatic Drainage of Lung**

- Left upper lobe- Left scalene to Left supraclavicular
- RUL, RLL, LLL, visceral pleura- Right scalene to Right supraclavicular
- Parietal pleura- axillary

*****RNTCP Daily Dose Regimen**

- Daily fixed dose combination of ATT is given according to weight brackets.
- New cases-
  - Intensive phase- 8 weeks of HRZE in daily dose. No need for extension of IP.
  - Continuation phase- 16 weeks of HRE daily..
- Extension of continuation phase by 12-24 weeks may be considered for TB meningitis, skeletal TB, disseminated TB

Weight	**Intensive phase (HRZE 75/150/400/275)**	**Continuation phase (HRE 75/150/275)**	**Inj. Streptomycin**
25-34kg	2 tablets	2	500 mg
35-49 kg	3 tablets	3	750 mg
50-64 kg	4 tablets	4	1g
65-75kg	5 tablets	5	1g
>75kg	6 tablets	6	1g

Community acquired pneumonia

- CURB-65
  - Confusion
  - Urea >7mmol/L
  - Respiratory rate ≥ 30/min
  - SBP <90 or DBP <60

Tx

- Pathological stages of pneumonia
    - Congestion
    - Red hepatisation
    - Grey hepatisation
    - Resolution
- Recurrent pneumonia- Chronic bronchitis, AIDS, Bronchiectasis, SLE etc.

Causes of dyspnoea

- Airway
- Alveoli
- Interstitium
- Vasculature
- Others
    - Pleural diseases
    - Chest wall diseases
    - Neuromuscular diseases
    - Neuropathy
    - Physiological
    - Abdominal distension
    - Laryngeal spasm, Laryngeal oedema
- Dyspnoea with normal CXR
    - Asthma
    - Pulmonary embolism

Summary of RS pathologies

Pathology	**cause**	**trachea**	**What happens**	**VR/VF**	**Percussion**	**sounds**
CONSOLIDATION	pneumonia	midline	fluid in parenchyma	increased (fluid sound faster)	DULL (fluid vibrate less)	1. tubular bronchial sounds 2. crackles
HYPERINFLATION	COPD, asthma	midline	More air replaced in parenchyma	reduced (air sound slower)	RESONANT (air vibrate more)	1. breath sounds maybe reduced - emphysema 2. wheeze - asthma ? 3. may have late inspiratory crackles in COPD - mcleod
PL.EFFUSION	TB- imp parapneumonic effusion transudative : CCF, Nephrotic cirrhosis etc	Shifted to opposite side in Massive.	Fluid around lung + lung far from chest wall	reduced (since lung - chest distance more) above the level increased ??	STONY DULL	1. diminished or absent BS 2. tubular above the level
PNEUMOTHORAX		opposite side	Air around lung + lung far from chest	reduced	Resonant (air vibrate more)	1. diminished /absent BS 2. no added sounds

COLLAPSE	compressive - PTX , large neoplasm absorptive (d/t onstruction) : F/B , musocus plugs , neoplasm , endobronch TB	deviated to same side	Lung far from chest	Reduced	Dull (collapse form solid like area)	1. diminished to absent breath sounds - if major bronchus occluded 2. tubular BS - if peripheral bronchus occluded 3. no added sounds
FIBROSIS	idiopathic drugs like Bleomycin	deviated to same side		variable	impaired note	1. diminished or bronchial BS 2. can have crackles
CAVITY	TB lung abscess necrotic degeneration	midline		increased	impaired	1. cavernous bronchial BS 2. fine / coarse crackles
BRONCHIECTASIS	post tb infective CF	midline		normal / increased	impaired	1. Vesicular/cavernous (large cystic cavities)/tubular breath sounds (associated consolidation) 2. coarse crackles

Fibrosis vs Collapse

Feature	Fibrosis	Collapse
Onset	Chronic	Sudden
Clubbing	Present	Absent
Percussion	Impaired	Impaired
Breath sound	Decreased	Absent
Added sounds	Crackles	None
Wasting	Yes	No

Pleural effusion

- Aetiology
  - Exudative
    - Infectious causes- Tuberculosis, Pneumonia
    - Autoimmune- Rheumatoid Arthritis, SLE
    - Neoplastic- Metastasis, Mesothelioma, Lymphoma
    - GI- Pancreatitis, Liver abscess, Subdiaphragmatic abscess
    - Uraemia
    - Yellow nail syndrome- effusion due to lymphoedema
  - Transudative
    - CCF, Cirrhosis, Nephrotic syndrome, Myxoedema, Constrictive pericarditis
- Investigations
  - CXR
    - Normal volume of pleural fluid is 25ml
    - Fluid detectable in PA view is 300ml
    - Smaller effusions are detected in the lateral decubitus position
    - 1500 ml effusion is required for mediastinal shift
    - Costophrenic and cardiophrenic angles are obliterated
    - Phantom tumour- interlobar effusion can mimic a solitary pulmonary nodule
  - Light's criteria- Any 1 is enough to say it is an exudate
    - Pleural fluid protein/Serum protein >0.5
    - Pleural fluid LDH/Serum LDH >0.6
    - Pleural fluid LDH> 2/3 of the upper limit of serum LDH
- Treatment
  - Tuberculous- ATT. Steroids can be added but are usually not added. (Steroids+ATT is more useful in TB pericarditis)
  - Malignant- Repeated tap can be done
  - **Re-expansion pulmonary oedema**- If more than 600ml pleural fluid is drained at a time, acute pulmonary oedema can be precipitated. The mechanism proposed is due to an acute-inflammatory response that damages the alveolar capillary membrane possibly secondary to reperfusion of a previously collapsed lung
- Complicated parapneumonic effusion
  - pH <7.0
  - Glucose <40 mg/dL
  - Positive gram stain or culture
  - Treatment- antibiotics+ intercostal drainage

- Predominantly right sided pleural effusion
    - Cirrhosis of liver
    - CCF
    - Rupture of amoebic liver abscess into pleural cavity
    - Meig's syndrome- fibroma of ovary with ascites and pleural effusion
    - Right sided subphrenic abscess
- Predominantly left sided effusion
    - Acute pancreatitis
    - Oesophageal rupture (Boerhaave's syndrome)
    - Left sided subphrenic abscess
    - Dressler's syndrome- post MI
- Drug induced pleural effusion- Methysergide, Amiodarone, Nitrofurantoin, Bromocriptine
- Pleural effusion with same sided tracheal shift
- Indications for tube thoracostomy
    - Presence of gross pus in the pleural space
    - Organisms visible on gram stain or culture positive
    - Glucose <40 mg/dL
    - pH< 7
    - Loculations
- Chylothorax
    - When thoracic duct is disrupted and chyle accumulates in the pleural space
    - Lesions above T5- left sided chylothorax, below T5- right sided chylothorax
    - Causes- Trauma (thoracic surgery), Lymphoma, TB, Lymphangioleiomyomatosis, Myxoedema (gold paint effusion)
    - Treatment- Pleuroperitoneal stent. Tube thoracostomy is contraindicated
- Acute pleural effusion causes
    - Acute pancreatitis
    - Pulmonary embolism
    - Oesophageal rupture
- Thickened pleura vs. Pleural effusion. Thickened pleura has
    - Depressed intercostal space
    - **Impaired resonance**
    - Patchy findings
    - **No Ellis curve**
    - Rib crowding
- Empyema
    - Aetiology-
        - Direct extension of infection- Bronchiectasis, Pneumonia, Lung abscess
        - Nearby infection- Subphrenic abscess, Liver abscess
        - TB
        - Chest wall infection
    - How to differentiate from pleural effusion? Sick patient, Clubbing, fever, intercostal tenderness
    - Criteria for Empyema
        - Thick pus
        - pH <7.2
        - LDH >1000 U/L
        - Glucose <40 mg/dL
        - Gram stain may be negative