RESPIRATORY EXAM:

1. General inspection [at 45°]

- **Patient position = URPIGHT > TRIPOD > SUPINE (worst)**
  - Impending sense of doom?
- **SURROUNDINGS**: **Sputum mug** (clear, blood, pus, etc.), **inhalers**, **O_2 supply (nasal prongs, non-rebreather)**, **chest physiotherapy devices (e.g. CF, Bronchiectasis),** walking aids, cig packs
- **BREATHING WORK: Breathing work @ rest + accessory muscles** *(esp. intercostal, intraclavicular space, pursed lips)*
- **Cyanosis** (blue lips, blue fingertips)
- **Type of cough (dry/productive/bovine)**,
  - **Dry = ILD, ACEi, GORD, Post-nasal drip, asthma, sarcoidosis**
  - **Wet** = pneumonia, lung abscess, bronchiectasis, APO, TB, lung cancer, PE
- **Voice** = hoarseness (RLN palsy) – *DDx*: laryngitis, oeseophageal / lung cancer (+ throat)
- **Body Habitus**
  - **Tall thin males (marfan's)** - ++ pneumothorax risk → elongated blebs + ↑ difference in intrapleural pressure b/w apex and base
  - **T1RF** = agitated, tachypnoea, +WOB, confusion → CPAP
  - **T2RF** = Blue bloaters = flushed skin, bounding pulse, asterixis, drowsy → **BiPAP**

2. Hands *(distal → proximal)*

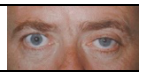
Inspect	• **Clubbing = 80% of cases** (NOT COPD) → lung cancer, CF, **Bronchiectasis, lung abscess** ○ *Gradual UWL, anaemia, haemoptysis* • **Cyanosis (peripheral)** • **Tar staining** in-between fingers= **SMOKING** • **Thinned skin** = steroid usage • **Muscle Wasting of small hand muscles → apical Pancoast's lung tumour spreading** to brachial plexus **invasions of T1 nerve root** → weakening finger abduction/adduction

Palpate	• **Wrist tenderness/clubbing** = ***hypertrophic pulmonary osteoarthropathy* (HPOA)** → periosteal inflammation (**swelling and tenderness)** at the distal wrists [Wrist clubbing]
	• **Fine tremor**:
	  ○ SABA (B2 agonists)
	  ○ Steroids / Cushing's
	• **Asterixis/Flapping tremor**
	  ○ (CO_2 retention, hepatic encephalopathy)
	  ○ Ureamic encephalopathy
	• **Pamberton's sign:** SVC obstruction

Pulse	• *Tachycardia* (asthma, COPD, PE or infection) • *Bounding pulse* (CO2 retention Type 2 resp. failure)
RR	• *Tachypnoea (lung disease, infection, fever, PE, stress)* • *Bradypnea (CNS depression)*

3. Face *(Top → bottom)*

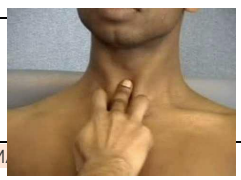
Face	• **Facial plethora**—smoker, SVC obstruction [acute SOB] • **Cushingoid appearance** – steroid usage
Eyes	• **Conjunctival pallor** → anaemia → non-respiratory cause of SOB (O_2 transport issue) • **Horner's syndrome [miosis, ptosis, anhidrosis]**
Nose	• nasal polyps (**Asthma**), engorged turbinates (**asthma – allergies**), septal deviation (nasal blockage), **Sinus tenderness** (palpate maxillary, frontal sinus)
Mouth	• peripheral/central cyanosis, • poor dentition or loose teeth (**lung abscess**) OR **oral candida [UNCONTROLLED ASTHMA]** • URTI (**red pharynx) + crowded oropharynx** ○ Check for tonsillitis (URTI), tongue depression required



4. Neck/ Trachea

Warn patient of any discomfort [STERNAL NOTCH = LANDMARK]

Cervical LN	• **Lymphadenopathy**: infection (TB), lung cancer, sarcoidosis
JVP	• **Raised in cor pulmonale** • **DDx: RVF, TR, PHTN, CCF**
Tracheal deviation	• **towards side of lung lesion** = (atelectasis | fibrosis | pneumonectomy) • **away from side of lung lesion** = (Massive pleural effusion | tension pneumothorax) • **upper mediastinal masses**= (Retrosternal goitre) ○ **DDx: neck mass – branchial cyst, pharyngeal pouch**
Cricosternal distance & tracheal tug [pull downwards]	• Airflow obstruction ➔ Gross CHEST expansion [**possible COPD]** • Fingers on supraclavicular fossa feel contracted during inspiration (use of the accessory muscles – scalenes → **dyspnoea → impending sign of resp. Failure)**

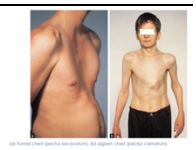


5. Posterior Chest (IPPA + ask patient to cough)

Inspect ** Video-assisted thoracoscopic surgery (**VATS**)*

General	• **Scars** (under arms), → thoracotomy, sternotomy (central), chest drains (M • **Skin changes (*erythema/rash*), radiotherapy tattoos** • **Prominent veins** (SVC obstruction, caput medusae)
Shape /symmetry	• **Barrel-chest** (increased AP diameter = **COPD**), • **Pectus excavatum (CT disorder)** • **Pectus carinatum (childhood resp. disease – bronchiectasis, CF, asthma)** • **Kyphoscoliosis** (exaggerated curvature of spine – **vertebral TB)** - risk of T2RF • **Harrison's sulcus**: Lower rib depression due to severe childhood asthma or rickets

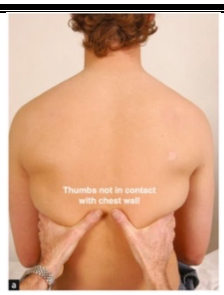
Chest wall movement	• **Mainly upwards** = *emphysema*
	• **Symmetric**, movement for **COPD**
	• **Assymetrical →**
	  ○ ***Pneumonectomy*** → *absent BS (initially) – BS present as lung tissue develops*
	  ○ ***Rib fracture*** – *Subcutaneous emphysema (crackles under skin)*
	  ○ ***fibrosis***,
	  ○ ***atelectasis, effusion, pneumothorax***

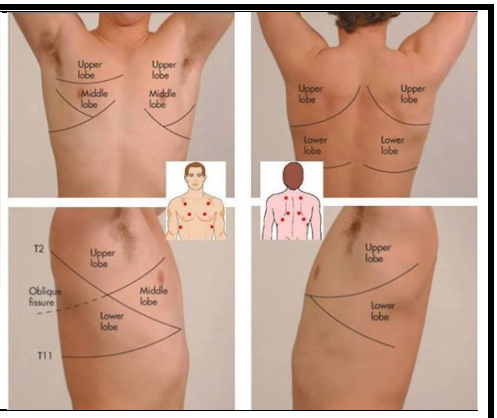


6. Anterior chest [Recline back at 45°]

Palpate (asymmetry and reduction of movement)

- **Chest expansion (fingers surround sides of chest BUT do not touch):**
  - **normal ≥5cm (CHECK posteriorly)**
  - **Hoover's sign = COPD** (thumbs closer together during inspiration, and further apart in expiration)
- Feel for **RV heave (pulmonary HTN)**
- **Palpate Ribs/** Costochondral joints → tender = fractures, prolonged cough
- **Subcutaneous emphysema**: diffuse swelling on chest wall or neck + crackling sensation on palpation (due to air beneath skin → ? pneumothorax in lung or rib #)





Percuss:

'Compare both sides

Ask patient to fold their arm or hug pillows

- rotates scapulae **anteriorly** out of the way [compare L/R]
- **Percuss bilaterally**, NB: liver begins at 5th IC space on right

	Tension Pneumothorax	Consolidation - Pneumonia	Pleural Effusion	Atelectasis (collapse)	COPD / asthma
Tracheal deviation	Away	None	Away	Towards	None
Chest expansion	All reduced ipsilaterally				
Vocal Fremitus	↓	↑	↓	↑	
Percussion	Hyper-Resonant	Dull	**Stony Dull "blood, lymph,or fluid"**	Dull	Hyper-Resonant (bullae areas) Dull (consolidation)

Auscultate

(same location as percussion → compare both sides)

'Emergency = 'silent' chest = narrowed airway about to collapse

Standard	• Patient breaths in/out deeply → compare both sides for symmetry
Vocal resonance	• **Repeat auscultation but patients says "99"**

1) Breath sound

- **Intensity → reduced diffuse (COPD) or reduced localised (pleural effusion, pneumothorax)**
- **Quality (vesicular** = normal (I:E ratio = 3:1) → **bronchial = periphery (hollow) = consolidated (lobar pneumonia) or collapsed lung or pleural effusion**

2) Added sounds

Reduced airway entry (↓BS)	Emphysema, pneumothorax, pleural effusion, collapse
Inspiratory stridor (*high pitched monophonic)*	• *ACUTE*: tracheal, laryngeal obstruction (e.g. food, foreign object), epiglottis, anaphylaxis • *CHRONIC*: laryngeal cancer, tracheal cancer, laryngeal palsy
Expiratory Wheeze (*polyphonic vs monophonic***)**	• **COPD** [low-pitch polyphonic or monophonic] • **Asthma** [high-pitch polyphonic • **Monophonic** = Tumour
Coarse crackles (*mid-insp, variable***)**	Bronchiectasis, consolidation (Esp. vocal resonance)
Early inspiratory crackles	COPD (esp. chronic bronchitis)
Coarse inspiratory crackles	Pulmonary oedema, consolidation
Fine-end-inspiratory crackles [*Velcro***]**	Pulmonary fibrosis, ILD (upper vs lower lobe)
Pleural rub (*grating colicky sound***)**	Pleurisy, pulmonary infraction, pneumonia, pleural malignancy
Whispered petroliquy (transmitted upper airways sounds)	Consolidation "sound of words head through chest wall"

Auscultate Heart

Recognise P2 *(loud = pulmonary HT, soft = pulmonary stenosis)*

- Loud P2 "ba-boom"
- RV heave/thrill
- ↓JVP
- Systolic murmur (TR)

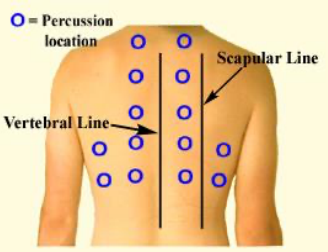


Figure 2. Areas to percuss during the posterior respiratory exam.

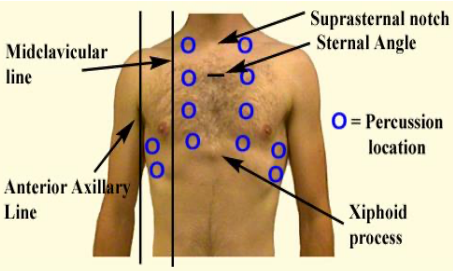
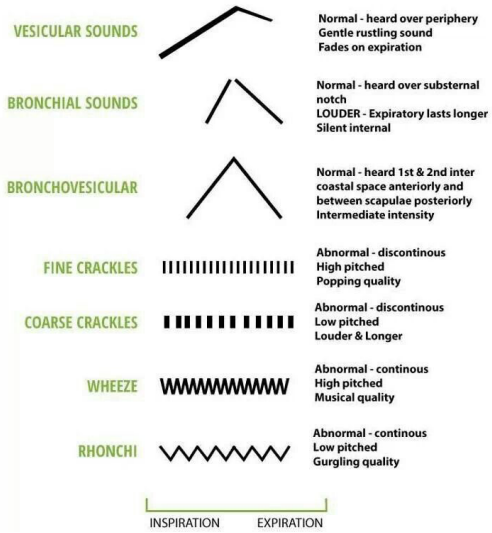


Figure 5. Landmarks and areas to percuss for the anterior component of the respiratory examination.

Breath sounds



Dynamic

- Ask **to blow hard** (FEV1) – check if if > 3seconds
  - DDx: COPD, asthma or **gas trapping**

Abdomen + legs

- Liver = ptosis (hyperinflated chest), hepatomegaly (RVF)
- Splen =splenomegaly (portal HTN secondary to CF or portal vein thrombosis)
- Peripheral oedema – sacral, tibial, medial malleolus (**cor pulmonale**)
- Swollen/tender calves (**DVT**)

Summary

Today I performed a respiratory exam on _____ :

- on inspection, any peripheral stigmata of resp. disease? Regular pulse? Normal RR?
- On percussion, symmetrical on front and chest? Any dullness? Any resonance?
- On auscultation, normal vesicular breathing? Added sounds?

To complete the examination, I would like to:

- **Review observation checks (esp. checking O2 sats and temp. for possible infection)**
- **Send off a sputum sample**
- **Measure peak flow (if asthmatic)**
- **Full Cardiac and Peripheral vascular exam**
- FBC, EUC, ABG
- **Possible CXR**: for possible malignancy or pleural effusion:
- **CTPA or V/Q perfusion scan**
- High res CT scan
- Lung biopsy

RESPIRATORY PHYSIOLOGY

Why study the respiratory membrane?

- **In emphysema, what effect does the larger size but smaller number of alveoli have on gas exchange?**
  Less efficient gas exchange
- **Why are babies born before 28 weeks at risk from respiratory distress?**
  Babies have started to produce surfactant to prevent alveolar collapse. No surfactant produced would cause substantial alveolar collapse and causing respiratory distress.
  - ➢ Give steroids for lung maturity
  - ➢ Surfactant and caffeine to help with respiratory distress

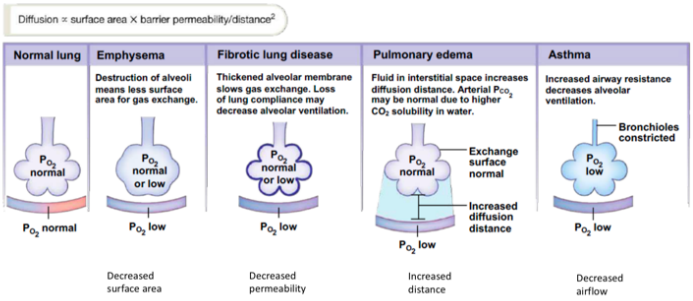
In a non-valvular pneumothorax, why doesn't the other lung collapse as well?

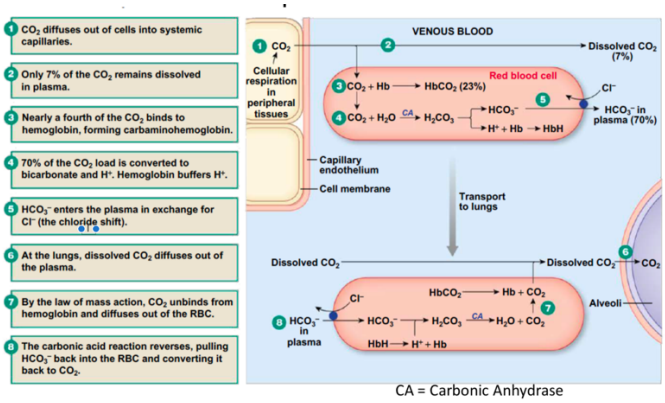
- Hole is made into chest → air gets into pleural cavity →lung collapses due to elasticity and negative pressure lost (**Pip** =0)(, allowing air to enter IN and OUT (no valve)
- Remaining lung is not collapsed to keep person alive as there is a midline partition (created by CT from anterior heart to sternum and from posterior part of pericardium back to spine) to separate the left and right pleura cavities (so air entering in one pleura does not enter to the other pleura)
- **Valvular pneumothorax** = flap of tissue created to allow air in but cannot get out. Inspiration allows more air in creating more tension and pressure to push heart against good lung
- **Tension pneumothorax** = 14G needle decompression in 2nd IC MCL (1ST LINE) → 5th IC space AAL

Why doesn't the lung completely collapse when you breath out (i.e. completely exhaust ERV)?

- Since there is a residual volume (≈ 1L), which is essential to prevent lung collapse

Some lung conditions associated with hypoxia



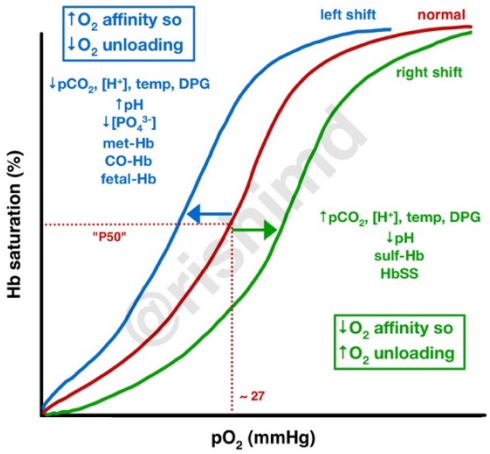


CA = Carbonic Anhydrase

What's the deal with foetal haemoglobin?

Foetal haemoglobin holds oxygen very tightly and is able to attract oxygen from maternal haemoglobin in order to deliver oxygen to the foetus for tissue metabolism

- ➢ HbF > HbA (causes physiological jaundice in 1st day of life)



Why is carbon monoxide so bad?

- CO has a greater affinity than oxygen to haemoglobin (close to irreversible binding), hence decreases the number of oxygen binding sites in red blood cells causing hypoxia (ironically appear bright red) ➔ 100% o2 sats
- Deliver oxygen at very high partial pressure to mitigate this (Dalton's Law)
- Shifts curve **LEFT + DOWN**

What the effect of increasing their 2, 3 DPG in anaemia?

- Typically 2,3 DPG disrupts binding of oxygen [increasing its release]
- This is however, useful for anaemia as it allows oxygen to released more easily to tissues to compensate for the low number of red blood cells.

Why study breathing in and out useful?

Why is someone with peritonitis not going want to take a breath in?

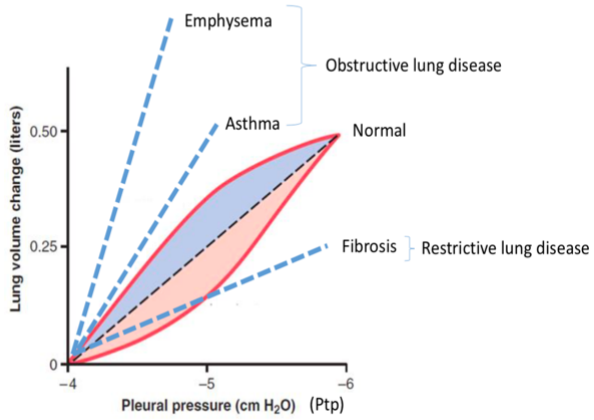
During inspiration, diaphragm moves down → moves liver down → hits and moves intestines. If you have inflamed membrane (PERITONEUM) around intestines, it will hurt when it moves (hence there is only shallow breathing)

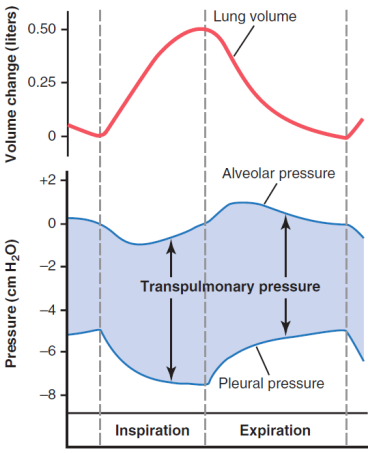
Does it matter that post-abdominal surgery patients don't want to cough?

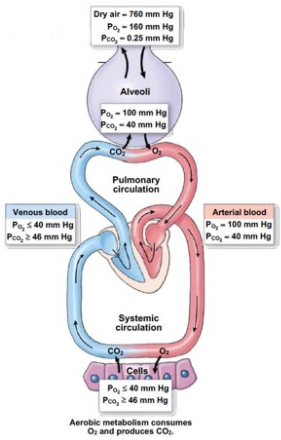
Despite coughing hurting, coughing is essential to remove any accumulation of mucus or fluid = minimise chance of respiratory infection (e.g. post-op pneumonia) ➔ aspiration pneumonia

Why do people with breathing difficulties often lean forward and grasp something immobile with both hands?

Mobilies upper limbs to provide extra assistance in inspiration from accessory respiratory muscles (e.g. scalenes). This is done unconsciously







Partial pressures

- Gases diffuse from areas of **high partial pressure → low partial pressure**
- Gases have a partial pressure if they are in the air or dissolved, but NOT if they are bound *e.g. to haemoglobin*
- For a mixture of gasses: the total pressure is the sum of their partial pressures represented as 'P' (Dalton's Law)

Does humidity affect P_{o2} and P_{CO2}?

E.g. **humidified air**: Added water vapour reduces % of O_2 and CO_2 in air = hence, decreased P_{o2} and P_{co2} (as per Dalton's Law)

What is P_{o2} in a 50% oxygen mixture given to a patient?

0.5 x 760mmHg = 380 mmHg (given by mask)

Calculating partial pressures

Air has a total pressure of 760 mmHg (at sea level)

Air contains 21% O_2 and 0.03% CO_2

Po_2 = 21% of 760 ie 150mmHg
Pco_2 = 0.03% of 760 ie 0.02 mmHg

Arterial (dissolved) blood gas Po_2 and Pco_2 are commonly used to indicate the effectiveness of pulmonary gas exchange.

As most O_2 is carried bound by haemoglobin, arterial oxygen saturation readings are also used to monitor pulmonary gas exchange.

Neural control of breathing Q's:

Why can't little Jonny (throwing a tantrum) hold his breath until he dies?

Although you can consciously override respiration, there is an automatic drive to breath will override conscious control of breathing. *Apneustic centres* **stimulate** **inspiration while** *Pneumotaxic centres* **inhibit** apneustic centres to allow expiration

Why is a spinal cord lesion above C3 so serious?

Although the higher neural levels are undamaged, the signals cannot be transmitted to the phrenic nerve or intercostal nerve. This essentially stops breathing as there is no diaphragm or chest movement causing death.

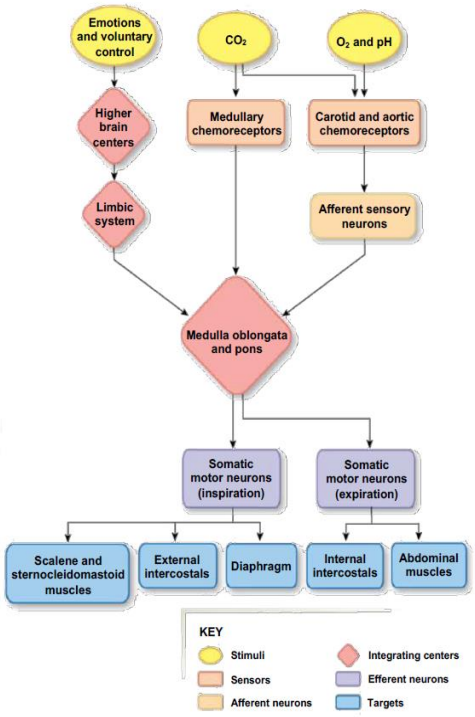
- **Phrenic nerve (formed from C3-5 spinal cord segments)** innervates left and right hemidiaphragm
- **Thoracic intercostal** nerves (T1-12) innervate accessory intercostals + abdo muscles for chest expansion and contraction

Why might you faint if you hyperventilate?

- Hyperventilating will cause excess CO_2 exhalation. This is dangerous as CO_2 is the main stimulus for respiratory centres to initiate breathing.
- Hence, it will take a longer period for CO_2 to accumulate to trigger activation of the chemoreceptors in the retrotrapezoid nucleus in the medulla to stimulate breathing, which by that time the person has already become hypoxic causing → syncope.

If you blood pH drops, what happens to your breathing?

Dropping pH means there is a high concentration of CO_2 →activates medullary centres to increased respiratory rate and depth of breathing volume to exhale excess CO_2 to decrease $PaCO_2$ to increase pH and compensate for this drop in pH.



CLINICAL IMPLICATIONS of **constant workload exercise** :

Respiratory adjustments to exercise:

- Constant exercise (ride bike for 5 mins)
- Oxygen consumption increases then falls
- Increase in ventilation = due to increase in tidal volume and increased in frequency

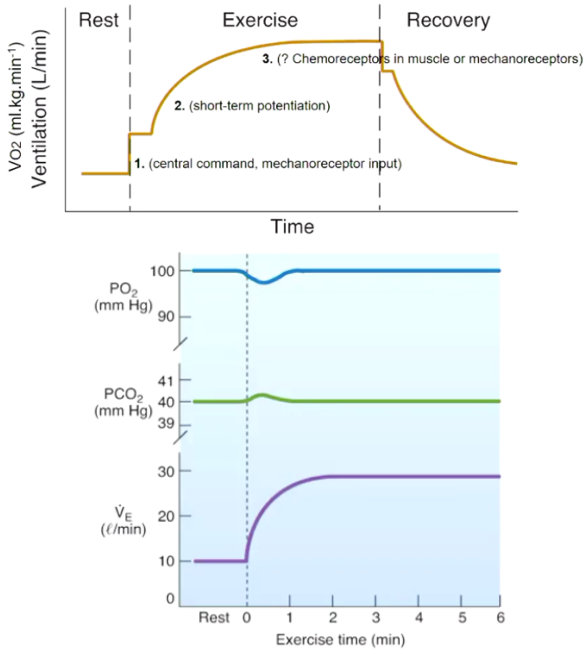
$$\uparrow \dot{V}_E = \uparrow V_T \times \uparrow \text{frequency}$$

What stimulates this breathing?

- Ventilation stimulated by mechanoreceptors in lung and partly by higher centres
- Initially, **mechanoreceptor input from limbs** increases central command (voluntary) to drive ventilation by
- *Short-term potentiation* (overflow into other cardio-respiratory centres to stimulate breathing) respiratory neurons in brain
- Possibly at end of exercise: *Chemoreceptors in muscle to maintain ventilation rate*

Changes in ventilation during constant workload

Driven by: *higher centres, mechanoreceptors*



Why is not the peripheral chemoreceptors during constant workload exercise?

- PaO_2 is **not low enough** (nor $PaCO_2$ increases **large enough to activate the anaerobic metabolism**) during constant workload exercise to stimulate peripheral chemoreceptors

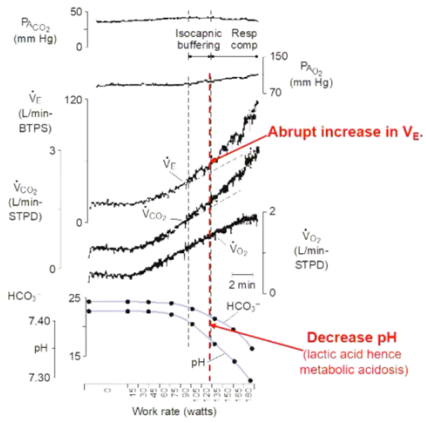
What about incremental exercise? Why is there an abrupt increase in ventilation?

- Increase in exercise = increase in ventilation →
- Abrupt increase in ventilation coincides with decreases in bicarbonate levels thus decreasing pH
- metabolic acidosis stimulates the peripheral chemoreceptors → increase ventilation rate at end of exercise in intense state

**Cardiac stress test, treadmill with increasing incline*

Changes in ventilation during incremental exercise

Driven by: *higher centres, mechanoreceptors + peripheral chemoreceptors*



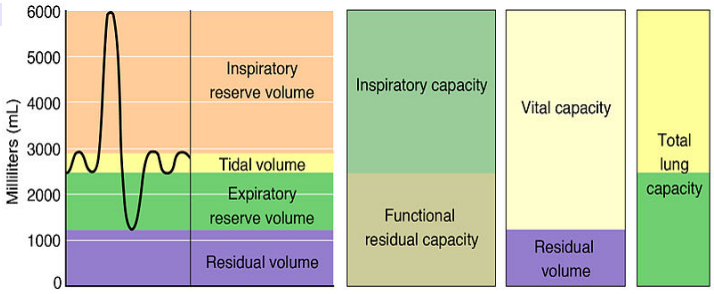
***TRANSITION INTO ANAEROBIC RESPIRATION = INCREASES LACTIC ACID PRODUCTOIN**

LUNG FUNCTION

At any one time, 3 major forces that determine lung volume:

	(1) Resp. muscles	(2) Chest wall	(3) Lung
Forcing out to RV (below FRC)	Smaller lung vol.	Bigger lungs [Recoil outwards at small lung vol.]	Smaller *[intrinsically elastic]*
Near TLC	Bigger lung vol.	Smaller vol.	Smaller vol.

- *ILD* = lung is stiffer, highly elastic → lower TLV, V/Q mismatch
- *Emphysema* = lung weakly elastic → high TLV

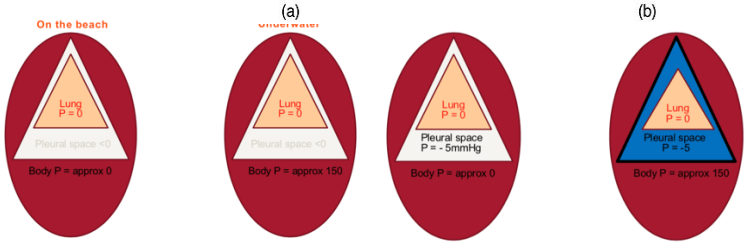


Reason for difference:

****In snorkelling**, we need a huge trans-diaphgramatic pressure to breathe*

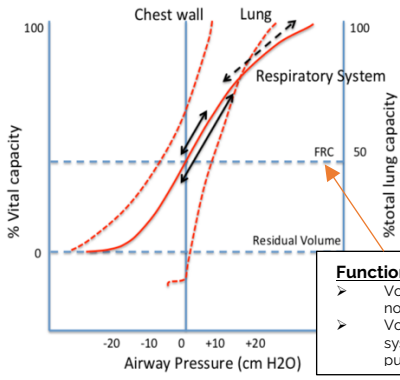
*****Elephants** = 10x thicker than diaphragm → generated pressure gradient to overcome body pressure gradient to make pleural pressure negative*

- ***do NOT have a pleural space*** → transformed during fetal development into loose CT
- Prevents any pneumothorax & avoid any pleural effusion → allows snorkeling



Man snorkelling with thorax at 2m depth VS elephant thorax at 2m depth

Obstructive & Restrictive Lung Disease



Compliance: $\Delta V / \Delta P$

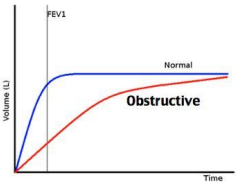
Emphysema
Increased Compliance

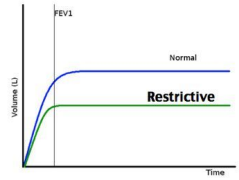
Restrictive disease
Reduced Compliance

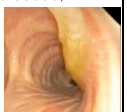
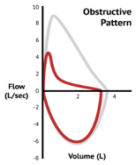
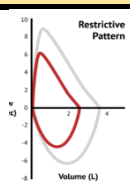
Functional residual capacity
- Vol. of gas remaining at end of normal expiration
- Vol. in which lung-thorax system exerts ZERO trans-pulmonary pressure

Normal:
- **Chest wall** = wants to spring out
- **Lung** = expands on positive pressure (collapses when pressure removed down to residual vol.)
- **Solid line** = lung stiffer at higher vol. (gradient decreases)
- **FRC** = vol. to counterbalance pressure required to keep lungs stretched (end of breath = most relaxing)
  - **On exercise** = *bigger breaths (slightly dip FRC)*
  - **Anxiety** =*breathing at higher lung vol. (increase SOB) – need to breathe out to minimise SOB*

Pathologies:
- **Restrictive** = **reduced compliance** → more pressure needed to move air as all volumes decreased (chest wall or lung issue)
- **COPD = Emphysema** = **increased compliance** → lung stiffer at higher vol. (gradient decreases = increased work) = higher +ve pressure needed at higher volumes despite less flow limitation





	Obstructive	Restrictive
Desc	• Physical vs anatomical obstruction • Airways disease	• Pulmonary vs. extra-pulmonary • Altered compliance
PP	• **Intraluminal** = mucus, foreign body • **Airway wall** = bronchoconstriction, hypertrophy, oedema • **Outside airways** = emphysema, enlarged LN (previous TB infection)	• **Lung parenchyma** = ILD, Alveolar oedema, Pulm. fibrosis • **Pleural cavity** = PE, pleural thickening/infiltration [**Acquired** (e.g. silicosis, asbestosis, CWP) • **Chest wall/Neuro** = Kyphoscoliosis, morbid obesity, neuromuscular disorders (MND, GBS, MG, DMD) • **CV issues** → *CCF → pulm. Oedema, pleural effusion*
Cause	**Asthma (dx needs many things)** 1. Hx of atopy, episodic symptoms 2. Obstructive spirometry 3. Airway hyperresponsiveness via allergen 4. Biopsy (Airway inflammation -eosinophils) • **Types**: (allergic vs. non-allergic, eosinophilic, non-eosinophilic, childhood vs. adult-onset) • *Environmental asthma* • Non-specific irritants = cold, smoke • *Specific triggers* • HMW =hair dyes, animal dandur [HMW] • [LMW], aerosols cedar wood in farmers) 1. *sensitisation* 2. *early phase reaction (IgE)* 3. *Late phase (eosinophils)* • **a1-anti-trypsin deficiency** → **pan-acinar** emphysema + chronic liver disease • **Acute bronchitis → fever, productive cough, no lung disease** • **Bronchiolitis → wheeze (not asthma as normal lung function)** (**RSV**- children, graft vs host disease, whooping cough, , **HPMV** -adult) • **Cystic fibrosis** (AR 1/4000 = congenital) ○ **Gene therapy?** • **Foreign body inhalation** 	**COPD (Emphysema, chronic bronchitis)**, • SOB, cough due to airway inflammation and remodeling • ***Centro-acinar*** *Emphysema* =Parenchymal destroyed (**loss of alveoli + ↓ elastic recoil**) • *Chronic bronchitis* = 2-3mths/ productive cough for ≥ 2 consecutive years • *V/Q mismatch* • *CXR* • ***Dynamic Hyperinflation*** (gas trapping), • ***barrel chest*** (increased AP diameter), • **Peripheral vascular marking** loss (i.e. pruning) **Bronchiectasis** • Permanent bronchi dilation of larger airways = **scarring in airways** (not tissue) • Chronic productive cough + recurrent chest infections • *Congenital*: CF, PCD • *Acquired*: TB, whooping cough (esp. ATSI, 3rd world), UC, RA, hypogammaglobulinemia • **ABPA (allergic bronchopulmonary aspergillosis →type 3 hypersensitivity→central bronchiectasis** ○ ↑IgE + eosinophils ○ *Rx: Anti-fungal & Steroids* • **Accel. FEV_1 decline + ↑ mortality**: ○ Age, low QoL, long-term O2 use ○ Recent ICU admission ○ **P. Aeruginosa colonisation**	*Clinical entities (several classifications)* **Idiopathic**: • Idiopathic pulmonary fibrosis • Non specific interstitial pneumonia (NSIP) • Cryptogenic organising pneumonia (COP) • Hypersensitivity pneumonia • RB-ILD **Secondary**: • Autoimmune (RA, SLE) • Drugs (MTX, -mycins, amiodarone) • Radiation (6/12 after) **Upper lobe** (*malignancies' more common here)*: • Radiation • Sarcoidosis, • CWP, • AS, • TB, • Silicosis **Lower Lobe [more common]**: • Active RA → scarring similar to IPF (need to Rx for RA **not** IPF) • Asbestosis • Scleroderma • COP • Drugs (amiodarone, MTX, bleomycin nitrofurantoin)
Spiro	***SPIROMETRY*** [flow vs. volume curve] = 1ST line test for any airway disease (BUT ***effort dependent*** ***ONLY looking at 1st sec & need no breath for 5 sec*** & ***insensitive to small airway issues*** *e.g. bronchiolitis* • TLC = normal • FVC = normal • FEV_1 = **Reduced** • FEV_1/FVC **< 0.8** • **FLOW LIMITATION** • **RV - INCREASED**  **FEV_1 < 1 = BAD** **FEV_1/FVC = age-dependent** • 0.7 = cut-off • 0.75 = normal 65, abnormal teen	• TLC = **Reduced** • FVC = **Reduced** • FEV_1 = normal/ **Reduced** • FEV_1/FVC = normal • RV = normal (increased in NMD) 
Rx	**For asthma** 1. **ICS** (1st line preventer esp. against triggers) 2. **Bronchodilators** (SABA/LABA → SAMA/LAMA) 3. **Mast cell stabilisers** (i.e. cromones) 4. **Leukotriene blockers** (i.e. Montelukasts for children in allergic asthma) 5. **Biologics** (e.g. anti IgE, anti-Il-4,5,13) 6. **Low dose Macrolides** → long-term use minimises exacerbations but risk of microbial resistance **For severe asthma (pharmacological)** • **Immuno** (i.e. Anti-IgE, Anti IL5, Anti-IL-4,13) • **Low dose Macrolides**	**For Moderate COPD** • **Stop smoking & Pulm, rehab** • Pharmacological: ○ **Dual Bronchodilators (potent LABA + LAMA)** (symptomatic & exacerbation benefit) *If severe:* ○ **Inhaled CS** (for frequent exacerbations) ○ **Systemic CS** (*avoid long-term use)* **For Bronchiectasis –*good prognosis*** • **Antibiotics (Rx for chest infection)** • *No ICS → reduces local immunity = bad (but if asthma then need ICS)* • **Mucus clearance** (Physio device) • **Bronchodilators**	**Systemic steroids** **Exacerbations**: • COPD (mainly), • Asthma • ILD exacerbations **Inflammatory conditions**: • HP = farmer's lung • COP • Eosinophilic pneumonia • Sarcoidosis • ILD = 2° to autoimmune disease **Nintedanib (TKI) *or* Pirfenidone (Anti-TGFb)** **Conditional use**: • Idiopathic pulmonary fibrosis (IPF) → barrel chest

LUNG FUNCTION TESTS Part #1

***Pulse Oximetry DOES NOT work for patients with*:**

- Hypothermia ≤ 35˚C
- Hypotension ≤ 50mmhg
- Severe vascular disease – peripherally cyanosed patients
- Vasopressor therapy
- Anaemia (low Hb)
- Hyperbilirubinaemia or Abnormal Hb
- Carbon Monoxide Poisoning (make O2 sats appear 100%)
- Nail polish
- Cold weather

→ **Use Arterial Blood Gases = "*gold standard*'** arterial puncture in radial/femoral artery to monitor O_2 levels if **arterial oxygen saturation (SaO2) < 80% (e.g. breathless patients, DKA, cardiac arrest)**

Peak Expiratory Flow (PEF)

- Simple bedside test to measure a ***reproducible maximum speed*** in forceful expiration
- Indicates how narrow the airways are.
- Useful for home monitoring of a person's asthma
- ***Peak flow charts*** – recognise ***pattern*** over time, ***identify exacerbations***

	Relative to normal peak flow	Control
Green Zone	80-100%	asthma is under good control
Yellow Zone	50-79%	Caution → may mean respiratory airways are narrowing and additional medication may be required
Red Zone	<50%	medical emergency. Severe airway narrowing + needs immediate action.

Clinical Indications for Peak Flow →**Needs to be measured regularly to be useful**

Short-term monitoring (2-8 weeks)	Long-term monitoring
• To Dx or exclude asthma • Identify asthma triggers • Monitor response to new or changes in **treatment** • Calculate '**trigger point**' for written asthma **action plan**	• People with frequent exacerbations (esp. children, seasonal variations to identify environmental triggers) • **Moderate to severe asthma** with little warning of exacerbations • Asthma patients with anxiety/tendency to **over-treat** • Poor perceivers of airway narrowing (i.e. *those with chronic poor lung function)*

*contraindications: ***CV disease (e.g. waiting for bypass), previous MI***

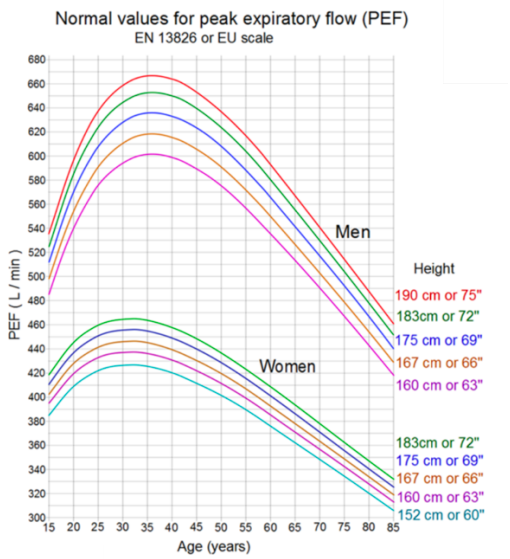
Interpretation of normal values

- Ensure 2 largest values should be within 5% of each other
- Compare with patient's own previous best reading
- When was medication last taken?
- Use same type of compressed chart every time to identify patterns in PEF (e.g. rises, falls, usual range)

What variables can affect normal range of results?

- Calculated for sex, age and height
- note: ***ethnicity***
- dependent on *sub-optimal effort*,
- technique (poor seal)
- diurnal variation

- *peak expiratory flow [PEF] ≈* ***500-600L/min (men)****,* ***300-400 L/min (women)*** *[always consult normal tables]*
- ***reduced and variable PEF*** *= COPD, asthma*



Perform Peak Flow [WASH HANDS BEFORE]

1. Connect a **new clean mouthpiece** to the meter
2. **Place the marker at the zero point** of the numbered scale
3. **Sit up straight and relaxed without** (avoid standing → cheating by adding extra force)
4. Hold flow meter **horizontally (avoid gravity) and steady** – place fingers either side of scale
5. Take a **very deep breath** in (essential to ensure that no low levels)
6. Put the meter in the mouth, between the teeth, and close the lips around the mouthpiece to create a seal
   a. **DO NOT** cover mouthpiece with tongue
   b. **DO NOT** cover the hole on the back end of the peak flow meter when holding it
7. **Maintaining a seal + blow out as hard and as fast as possible**
8. Repeat the **process 2 more times**

a. Ensure readings are close together

9. Document the best (**highest value**) of the 3 readings (unlike spirometry)
10. Thank patient and wash hands

PEAK flow vs Spirometry

Unlike spirometry, PEAK flow measurements have **poor quality control** and are **insensitive for detecting airflow limitation** since:

- since *difficult* to accurately *calibrate*
- hard to recognise what is *optimal* due to variable technique
- large inter and intra-specific *variations [fitness? – pilates, yoga]*

Interpret peak flow and spirometry results against normal ranges (spirometry with reference to obstructive and restrictive lung disease)

Test:	Measures volume and rate in which air can be ***forcefully exhaled*** in a single blow from full lungs.
Purpose	• Differentiate between ***obstructive*** lung disease and ***restrictive*** lung disease • Assess bronchodilator reversibility in asthma & COPD • *For those with frequent cough, >40, Hx of smoking, decreased lung function*
Measures	• **Forced Vital Capacity (FVC)** • **Forced Expiratory Volume over 1 second (FEV_1)** • **Peak Expiratory Flow (PEF)** • **Forced Expiratory Ratio (FER%)** → Calculated using FEV1/FVC
Contraindications	• **Uncooperative patients** (e.g. dementia, intellectual handicap, drug/alcohol intoxication) • **Recent eye, thoracic or abdominal surgery** ➔ any increases in intrathoracic pressure may cause haemorrhaging, pain and burst possible vessels, aneurysms etc. • **Uncontrolled HT + Unstable angina OR recent MI** (within a month) ➔ do not have CV strength to perform spirometry • Known intracerebral or thoracic/abdominal aortic aneurysm • Recent cerebrovascular accident (CVA/Stroke) • **People at risk of/with a pneumothorax** ◦ Includes those in 20's who may have blebs within parenchyma in lung apices ➔ potentially burst during spirometry leading to air in pleural space causing pneumothorax or collapse

Open Spirometry Technique [REPRODUCIBILITY POINTERS]

Counselling

Today we are going to use a machine called a spirometer which will test your lung function. This will require you to remove any tight clothing and inhale as deeply as possible and exhale as hard and as fast as possible through a mouthpiece.

Checking for contraindications:

I want to check that you don't have any conditions that could exacerbate any illness you may have. Any past eye, thoracic or abdominal surgeries? Any MI, angina, uncontrolled HT? Any previous pneumothorax, collapsed lung?

1. Place *nose clip* on patients nose
2. Inhale maximally + hold [**BIG BREATH IN → like hold breath underwater as long as possible**
3. Place mouth over mouthpieces to obtain a firm airtight seal [**TIGHT SEAL**]
4. Blow out as hard, as fast & for as long as possible [**BLOW HARD & FAST →** keep blowing UNTIL NO AIR IN LUNGS like blowing into balloon or blow the fire outs]
   - *No maximal effort: lower amplitude and shorter response (mimics obstructive with sub-maximal effort]
5. Ask the patient to take a **BIG BREATH IN** and to remove the mouthpiece from their mouth
6. Instruct the patient to repeat the procedure until 2-3 reproducible, reliable FVC & FEV1 results are obtained, with the 2 best results within 150mlm of each other.
7. Dispose mouth-piece and nose-clip
8. Repeat until you have 3 blows meeting reproducibility criteria (**No more than 8 blows in a session**)
9. Document and interpret best result ((**i.e. HIGHEST Reading**)

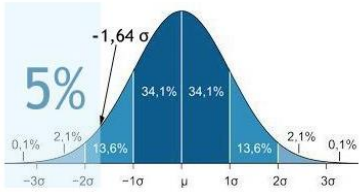
Reproducibility Criteria

At least ***3 blows*** must meet the ***reproducibility criteria*** to be accepted as valid measures of PFT:

1. The difference between the 2 best **FVC results** is ***<150 mL***
2. The difference between the 2 best **FEV_1 results** is ***<150 mL***
3. The **FER% (FEV_1/FVC)** is gender and age-adjusted to obtain a ***Lower Limit of Normal*** (LLN)

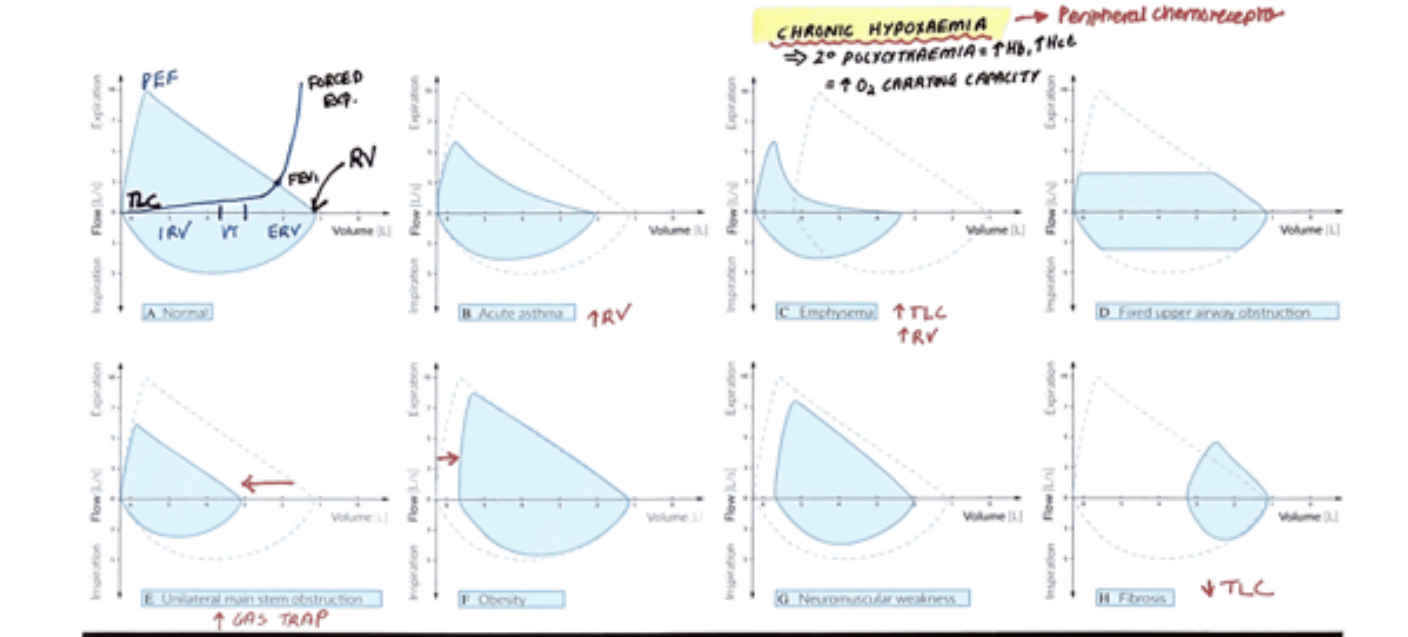
NOTE:

- Calculations of FER% use the ***best result*** for FEV1 & FVC from any spirometry recording (Results ***do not*** have to come from the same test)
- If the patient ***coughs*** during the first second of the procedure, the blow is ***not valid***



* The LLN corresponds with the 5th percentile for the gender/age

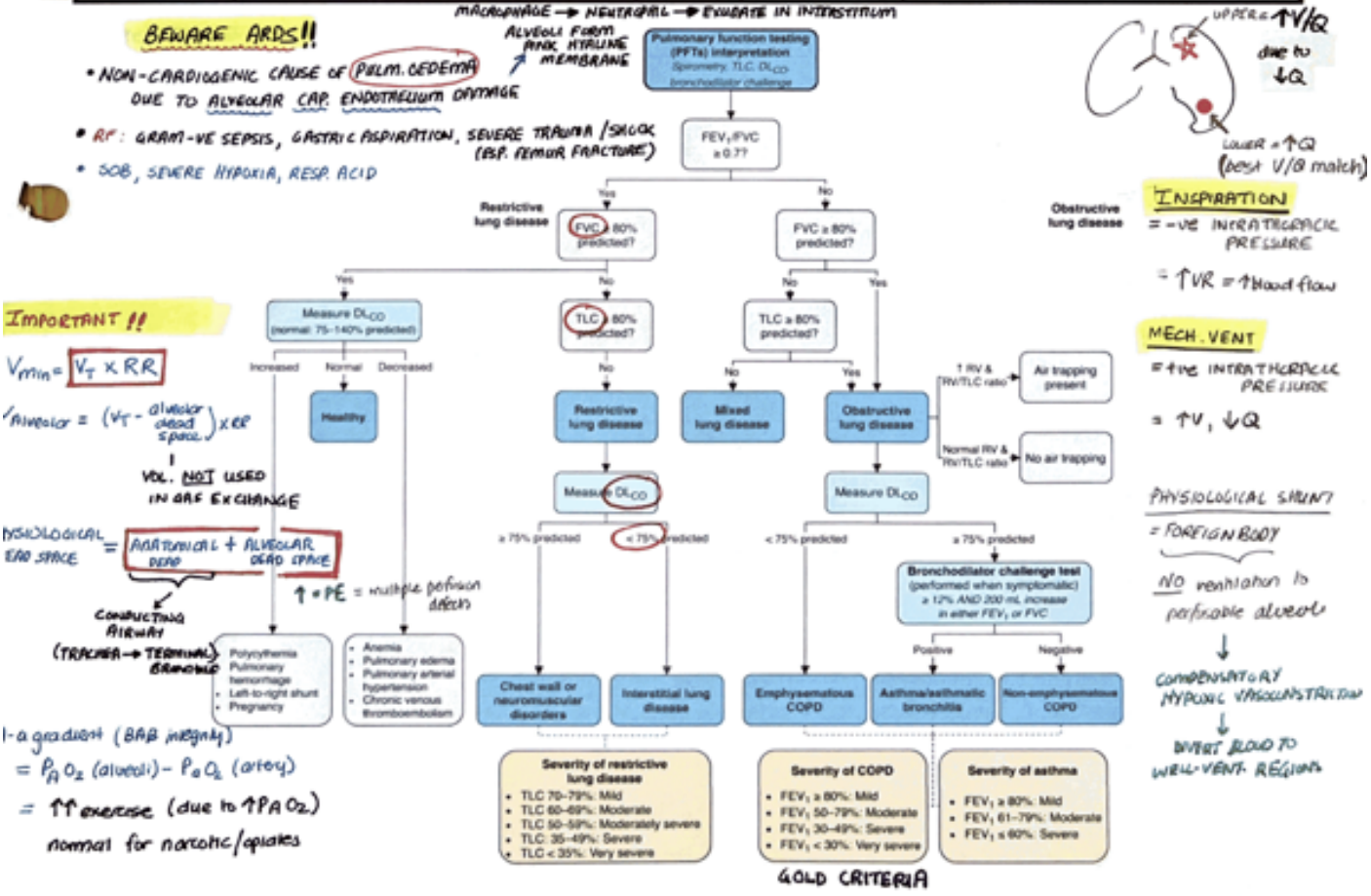
LUNG FUNCTION TESTS Part #2



	Pathophysiology	TLC	FEV1/FVC	RV
Asthma	Hyperresponsive airways to allergen (IgE, wheeze)	No change	decreased	Increased (as narrowed airway prevents full expiration)
Emphysema	Increased air trapping – loss of elastic recoil with enlarged alveoli area	Increase	decreased	Increase
Fixed airway obstruction	Foreign body obstruction → causes acute type 2 respiratory failure with no change in lung volumes (↓ FLOW RATE) = SHUNT	Normal	decreased	Normal
Unilateral main stem obstruction	Food entering the right main bronchus normal $PaCO_2$, ↓↓PaO_2	Normal	Decreased (air only enters unobstructed lung)	Increased (as narrowed airway prevents full expiration)
Obesity	Compressive effect – restricts ability to allow lung to expand fully	Decreased	Normal	Normal
Neuromuscular weakness	Due to myasthenia gravis, DMD → weak respiratory muscles in expiration and inspiration	Decreased	Normal	Increased (no strength to fully expire)
Fibrosis	Classic restrictive pattern where functional lung tissue replaced by non-functional scar tissue	Decreased	normal	Decreased/normal

BEWARE ARDS!!
- NON-CARDIOGENIC CAUSE OF PULM. OEDEMA DUE TO ALVEOLAR CAP. ENDOTHELIUM DAMAGE
- RF: GRAM-VE SEPSIS, GASTRIC ASPIRATION, SEVERE TRAUMA / SHOCK (ESP. FEMUR FRACTURE)
- SOB, SEVERE HYPOXIA, RESP. ACID

MACROPHAGE → NEUTROPHIL → EXUDATE IN INTERSTITIUM
ALVEOLI FORM PINK HYALINE MEMBRANE



IMPORTANT !!

$V_{min} = V_T \times RR$

$V_{Alveolar} = (V_T - \text{alveolar dead space}) \times RR$

↓ VOL. NOT USED IN GAS EXCHANGE

PHYSIOLOGICAL DEAD SPACE = ANATOMICAL + ALVEOLAR DEAD SPACE

↑ = PE = multiple perfusion defect

CONDUCTING AIRWAY (TRACHEA → TERMINAL BRONCHI)

Polycythemia
Pulmonary hemorrhage
Left-to-right shunt
Pregnancy

- Anemia
- Pulmonary edema
- Pulmonary arterial hypertension
- Chronic venous thromboembolism

A-a gradient (GAS integrity)
= P_AO_2 (alveoli) − P_aO_2 (artery)
= ↑↑ EXERCISE (due to ↑PAO_2)
normal for narcotic/opiates

INSPIRATION
= -VE INTRATHORACIC PRESSURE
= ↑VR = ↑blood flow

MECH. VENT
= +VE INTRATHORACIC PRESSURE
= ↑V, ↓Q

PHYSIOLOGICAL SHUNT
= FOREIGN BODY
NO ventilation to perfusable alveoli
↓
COMPENSATORY HYPOXIC VASOCONSTRICTION
↓
DIVERT BLOOD TO WELL-VENT. REGION

ACID-BASE BALANCE

	pH	pCO_2	HCO_3^-	Cause
Normal	7.35-7.45	35-45mmHg	22-26 mmHg	
Resp. acidosis	↓	↑	**Normal (ACUTE)** *mainly from intracellular Hb, PO4 buffering	**ACUTE HYPOVENTILATION** **CNS**: • Opiates, stroke, ICH, anaesthesia [CNS depression] **Lung**: • APO, Pneumonia • Upper airway obstruction (aspiration, laryngospasm after intubation)
			↑ (CHRONIC) * ↑↑ 1mM HCO_3 = ↑↑ 10mmHg $PaCO_2$ (occurs after 24 hrs w/ max response at 3-4 days)	**CHRONIC HYPOVENTILATION** **CNS**: • GBS, MND, MG, Obesity **Lung**: • COPD, Asthma, ILD
Resp. alkalosis	↑	↓	**Normal (ACUTE)** *mainly from intracellular Hb, PO4 buffering (within 10mins – max response at 6 hrs)	**ACUTE HYPERVENTILATION** **Lung**: • PE, pneumothorax → ↓PO_2 • XS ventilation (mechanical) → good V, poor Q • High altitude – Mt Everest **Other**: • **VERY ILL = Sepsis!!!** • Anxiety / panic attack → ↑PO_2 • Drugs – salicyclates
			↓ (chronic) ↓↓ 5mM HCO_3 = ↓↓ 10mmHg $PaCO_2$ (occurs (begin 6 hrs, maximal 2-3 days)	

Metabolic. Acidosis (L TKR)	↓	↓	↓	• *Below Stomach loss* = **Diarrhoea (HCO_3^- loss)**
				• **Toxins → MUDPILES** (HIGH-ANION GAP)
				  ○ EtOH
				  ○ Ethylene glycol (anti-freeze) = has osmolar gap
				• **Lactate** (IV adrenaline, metformin)
				• **Ketones** →DKA (T1DM, SGLT2i, starvation)
				• **XS H+ ions** → renal failure, Type 1 RTA or rhabdomyolysis
				• **Low bicarbonate** → diarrhoea, AKI, type 2 RTA

Metabolic. alkalosis	↑	↑	↑	• *Above Stomach loss* = **VOMITING, NGT/NPA aspiration** • **Exogenous HCO_3^-** = Overcorrection IV or Milk alkali (XS Mylanta – antacids) • ***K+ depletion*** (Conn's, Cushing, diuretic +/- diarrhoea) • ***Increased aldosterone activity*** ○ *Conn's , cirrhosis, HF, loop and thiazides*
Mixed acid base	• Mixed metabolic acidosis/alkalosis • Mixed metabolic and respiratory *(never for resp. acidosis/alkalosis)			• Lactic acidosis/DKA with vomiting (met acid + alkalosis) • Salicylate poisoning (met acid + resp. alkalosis)

How to interpret ABG/VBG?

1) Check pt details
2) **Is it venous or arterial**? → *Do I need ABG or VBG?*
   a. *If ventilating/sepsis = need ABG*
   b. *If chronic lung disease/DKA = VBG + SaO2*
3) **If resp. acidosis → check A-a gradient** (diffusion issue?)
   a. High A-a = lung issue (NOT CNS)
4) **If NO resp. or metabolic cause ➔ check Base Excess (-2.5 to +2.5)**
   a. Is there another cause of the acidemia or alkalemia?
5) **If metabolic acidosis → check Anion Gap to determine if:**
   a. (1) **intrinsic loss** (i.e. renal, DKA, Lactate) or
   b. (2) **extrinsic acid** (e.g. methanol, anti-freeze)
6) Respect buffering (HCO3 may be normal) → due to intracellular Hb, PO4 buffering

A-a gradient (difference b/w o2 conc. in alveoli and arterial system)

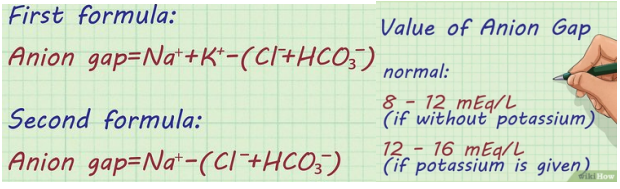
$$\text{A-a gradient} = P_{AO2} - PaO2 \cong \frac{Age}{4} + 4$$

Normal Anion Gap

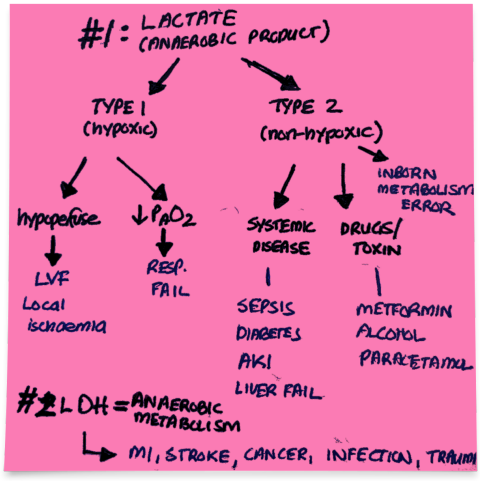
- HPTH,
- Addison
- RTA (Cl^- retention)
- Diarrhoea/Drugs
- Acetazolamide (CA inhibitor)
- Spironolactone
- Saline

Respiratory Alkalosis

- **C**NS disease (ICH, cirrhosis)
- **H**ypoxia
- **A**nxiety
- **M**ech. Overventilation
- **P**rogesterone
- **S**alicylate/Sepsis



Cause for hypoxia	A-a gradient	Corrected with high FIO2?	Causes
Low FIO2	Normal	Yes	High altitude, hypoxic gas mixture
Hypoventilation	Normal	Yes	Residual anesthetic, muscle relaxants
Diffusion	Elevated	Yes	Interstitial lung disease
V/Q mismatch	Elevated	Yes	Mucus plug, pulm embolism, COPD
Shunt	Elevated	No	Atelectasis, ARDS



ACID-BASE DISORDER QUESTIONS

1.1 A 17-year-old girl is admitted to Intensive Care. She is unconscious and ventilated following an overdose of benzodiazepine. Her blood gases are checked on admission to ICU. You are asked if you want to adjust the ventilation.

P_aO_2 (kPa)	pH	P_aCO_2 (kPa)	HCO_3 (mmol/l)	Base excess (BXS) (mmol/l)
13.3	7.60	2.66	19	0

P_aO_2 (mmHg)	pH	P_aCO_2 (mmHg)	HCO_3 (mmol/l)	Base excess (BXS) (mmol/l)
100	7.60	20	19	0

(a) What do the blood gas results show?

(a) What do the blood gas results show?
The P_aO_2 is normal. The pH reveals an *alkalaemia* – reference to the P_aCO_2 reveals it to be low (normal is 4.66–5.98 kPa or 35–45 mmHg) so there is a *respiratory alkalosis*. The base excess (BXS) is within normal limits (normal +2.5 to –2.5), so there is no metabolic component. This is *acute respiratory alkalosis*.

(b) Do you want to adjust the ventilation?
Yes. The alveolar minute ventilation needs to be reduced – this can be achieved by turning down the tidal volume or reducing the ventilation rate. Usually the rate is reduced. The alveolar partial pressure of CO_2 (P_ACO_2) is directly related to alveolar ventilation: halving the alveolar ventilation will be expected to result in a doubling of the P_aCO_2.

- *Resp alkalosis with NO metabolic compensation as base excess normal with no hypoxia (no signs of resp. failure – as ventilated)*
- *Likely Came in obtunded – intubated → over vigorous ventilation (too rapid) = overventilated?*
- *Base excess = 0 (suggests acute setting)*

Soln: *reduce ventilation rate (i.e. reduce tidal volume or reduce ventilation rate)*

- *Low co2 = vasoconstriction – end organ ischeamia?*
- *High co2 =vasodilation - can get cerebral oedema*

1.2 At 2 a.m. the night intern brings an arterial blood sample to ICU from a 71-year-old man who is on the medical ward with a diagnosis of lobar pneumonia. She is worried that he has received too much oxygen (40%) and the F_iO_2 should be reduced.

P_aO_2 (kPa)	pH	P_aCO_2 (kPa)	HCO_3 (mmol/l)	BXS
10.64	7.15	8.64	22	–8

P_aO_2 (mmHg)	pH	P_aCO_2 (mmHg)	HCO_3 (mmol/l)	BXS
80	7.15	65	22	–8

(a) What do the blood gas results show?

(a) What do the blood gas results show?
The P_aO_2 is within normal limits (although this requires 40% inspired oxygen to achieve, so the alveolar–arterial gradient is increased). The pH reveals a severe *acidaemia* – reference to the P_aCO_2 reveals a high P_aCO_2 so there is a *respiratory acidosis*. The base excess

(b) What will you tell the intern?
The patient does not have a metabolic alkalosis compensating for chronic CO_2 retention. This is respiratory failure complicated by metabolic acidosis (lactic acidosis due to sepsis in this case). Dropping the F_iO_2 is inappropriate: he is really sick and needs to be reviewed urgently and will probably require ICU admission (unless there are other contraindications).

Resp acidosis with no metabolic compensation (no hypoxia but have hypoxaemia)

- *Base excess is **negative** = suggests metabolic acidosis (very sick!)*
- *Mixed respiratory and metabolic acidosis (bi carb is lower than normal)*
- *Need ventilation*

1.3 Some days later the same intern shows you these arterial blood gas results from a 73-year-old lady who has been admitted to the medical ward with recent onset atrial fibrillation. She has been diagnosed with chronic obstructive airways disease (COAD) and has smoked heavily for more than 50 years. She asks you if these gases are consistent with chronic CO_2 retention.

P_aO_2 (kPa)	pH	P_aCO_2 (kPa)	HCO_3 (mmol/l)	BXS
6.65	7.35	8.512	34	+5

P_aO_2 (mmHg)	pH	P_aCO_2 (mmHg)	HCO_3 (mmol/l)	BXS
50	7.35	64	34	+5

(a) What do the blood gas results show?

(a) What do the blood gas results show?
The P_aC_2 is low (we don't know if she is on any supplemental oxygen). The pH reveals a *borderline acidaemia* – reference to the P_aCO_2 reveals a high P_aCO_2 so there is a *respiratory acidosis*. The base excess (BXS) is high, so there is also *metabolic alkalosis*. Since the pH is on the acidotic of 7.4 the acidosis is likely to be the primary abnormality and the metabolic alkalosis is therefore appropriate renal compensation.

(b) Is she right this time?
Yes, this is compensated respiratory acidosis and is entirely consistent with chronic CO_2 retention.
Hypoxic drive is possibly important for this lady and it is wise to monitor her arterial P_aCO_2 if increased oxygen is administered.

- *Respiratory acidosis with full metabolic compensation → COPD → Base excess = elevated → high bicarb content*

Repeat ABG
Chronically stable → likely compensated appropriately
**need venturi mask for fixed FiO2 but be careful to avoid loss of ventilation?*

T1 vs T2 Respiratory Failure

Hypercapnia:

$$PaCO_2 \propto \frac{VCO2}{VA} = \frac{Rate\ of\ CO2\ production\ (oxidative\ metabolism)}{Alveolar\ ventiilation\ (rate\ of\ CO2\ loss)}$$

Perfusion	1. Transfer O_2 to alveolus 2. Transport of O_2 to tissues
Alveolar Ventilation	3. Remove CO_2 from blood into alveolus & into environment
VBG	Na, K, pH, lactate → sepsis, DKA "cannot calculate A-a gradient"
ABG	PaO2 – check for resp. failure
O2 SATS	how much oxygen is bound to Hb

$$P_AO_2 = [F_iO_2 \times (P_B - P_{H_2O})] - \frac{P_aCO_2}{RQ}$$

$$P_AO_2 = 150 - \frac{P_aCO_2}{0.8}$$

If $P_aCO_2 \uparrow$, P_AO_2 must $\downarrow$

$$V_{minute} = RR \times V_{Tidal}$$

- **O2 delivery** = O_2 supply/content x CO
- **FiO2** = fraction of inspired oxygen (normal = 21%)
- **PAO2** = alveolar O2
- **PaO2** = arterial O2
- **O_2 content** = SaO_2 + [Hb]
- **Cyanosis** = [deoxyHb]
- **HCT** = % of RBC in blood (usu. 3x [Hb])
- **Carbon mono.= high SaO2 but low PaO2 on ABG**

Dx

Hypoxaemic respiratory failure (Type 1)
"lung issue"

- **Hypoxemia** (PaO2 <8.0 kPa (60 mmHg))
- Pallor, cyanosis, **cool**, reduced CRT
- ↓↓ **BP/tachycardia** + weak pulses,
- ↓↓**urine output**,
- altered mental state/**GCS**, angina/end organ ischaemic changes,
- ↓↓ **oxygen saturations**

Hypercapnic respiratory failure (Type 2)
"ventilator/pump issue" = **can't breath won't breath**

- **Hypoxia** (PaO2 <8.0 kPa (60 mmHg)) + **Hypercapnia** (>6 kPa (45 mmHg))
- **↓↓↓ alveolar ventilation (i.e. ↓↓ min. ventilation or ↑↑dead space)**
- ↑↑ WoB, ↑↑ RR, (Vmin = V_T x RR)
- Bounding pulse & Warm periphery
- Inadequacy of chest expansion/paradoxical breathing,
- Altered mental state & CO_2 retention → flapping tremor

DDx

	Example	A-a grad	Corrected by↑FiO2
V/Q mismatch [most common]	• **Low V/Q** = ILD obstructive lung diseases, atelectasis, pneumonia, ARDS, pulm. oedema • **High V/Q** = PE	↑ ↑ ↑	Yes
R → L Shunt	• *Anatomical (bypass alveoli)*: PFO, ASD, VSD, pulm. AVMs • *Physiological (non-ventilated alveoli perfused)I)* : PE, atelectasis, APO	↑ ↑ ↑	No
Diffusion impairment	ILD, ***pneumonia (inflammation)*** **(induced by exercise)**	↑ ↑ ↑	No
Hypoventilate	COPD, NMD	Normal	Yes
Low FiO_2	high altitude (↓PAO2)	↓↓↓	Yes
Other	Severe anaemia, sepsis, foreign body		

	Example
Reduced resp. drive (↓ RR or ↓ V_T)	• Drugs (opiates, benzodiazepines) • XS O2 therapy in chronically hypoxic pt • Traumatic or CNS surgery • TBI, TIA, stroke, encephalitis
Suboptimal ventilation (↓ V_T)	• ***Reduced chest wall compliance*** (OHS, flail chest, chest deformity (*kyphosco)*, burns, scars) • ***Nerves*** (MND, ALS, MG, phrenic palsy), • ***Muscles*** (DMD, GBS, Pompe's, GBS, ruptured diaphragm) • ***Airflow*** (COPD, large airway obstruction, mucus plugging)
Ventilation not meeting demands of over-production	• Sepsis • Malignant hyperthermia
Resp work & dead space	• Acute severe asthma • AECOPD or over-ventilation of COPD

AIM TO BUY TIME

- **1st line (improve breathing mechanics)** = **Optimise Position** (45 semi-fowler to improve V/Q) + **analgesia** + **appropriate FiO2**
- **Address symptoms affected by chest expansion*** (E.g. anything that enlarged beneath diaphragm → constipation, ascites)
  - Chest physio/airway clearance (remove mucus produced from infection
  - Antibiotics if evidence of infection
  - Diuretics if evidence of fluid overload

- **Hypoxic patients** → target 92-96%
- **CO2 retainers** → Venturi Mask → target 88-92%
- **If FIO2 requirement:**
  - > 40% (6L via HM) → senior clinical review
  - > 50% → HDU/ICU

- **Improve drive to breath**
  - stop/reverse suppressants
  - titrate FiO2 to achieve mild hypoxaemia
- **Improve airway patency** ➔ suction airway, neb. Bronchodilators (wheeze), theophylline (PDE43/4i), sildenafil (PDE5i)

Non-invasive ventilation involves using a full face mask or a tight fitting nasal mask to blow air forcefully into the lungs and ventilate them without having to intubate them (intermediate b/w O2 therapy and I/V)

CPAP

Patient ventilated at Continuous **SINGLE** pressure ABOVE atmospheric pressure

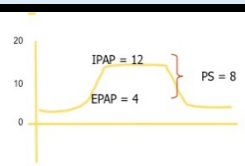
- *Overcomes* upper airway resistance
- *Recruit* (open up) alveoli (Esp. atelectasis and soggy lungs)
- *Reduce pre-load* in patients with HF
- **Patients have *NORMAL lung function***

Main indications for CPAP:

- **OSA**
- **CCF**
- **APO**

BIPAP

Patient ventilated at 2 pressures > Patm



- **IPAP** = Gives **Pressure support (PS) ➔ Increase by 2-5cm increments to resolve acidosis**
  ↑↑ V_{Tidal} or RR to ↑↑ V_{minute} = decrease CO_2
  - **Rests fatigued resp. muscles ➔ +++ ventilation**
- **CXR BEFORE insertion = confirm no pneumothorax**
- **ABG AFTER insertion (1hr then 4hrly)**

Contraindications of BiPAP:

- Untreated pneumothorax
- Structural abnormality affecting face, airway or GI tract

Last resort

Invasive mechanical ventilation (I +V) → via ETT or tracheostomy tube

- ***Indication*** = If $PaCO_2$ continues rising or safe PaO_2 cannot be achieved without severe hypercapnia/acidemia
- ***Benefits of BIPAP*** + eliminates metabolic cost of breathing
- Paralysis ➔ further reduces O2 consumption
- **Long-term use** = **barotrauma** (i.e. *alveolar overdistention = increased dead space & thoracic pressure* = ↓ VR = ↓ CO = HypoTN)
- **Myopathy** (diaphragm wasting)

A/E of increasing FiO2:

- **Hyperoxia** (oxidative stress, inflammation, systemic vasoconstriction = hypoperfusion)
- **May reverses systemic vasoconstriction** = vasodilatation = worsen V/Q mismatch
- **Loss of Hypoxic drive in CO2 retainers ➔ death**
- **ONLY AIM FOR 100% O2 sats if:**
  - **Tissue ischemia = STEMI or stroke**

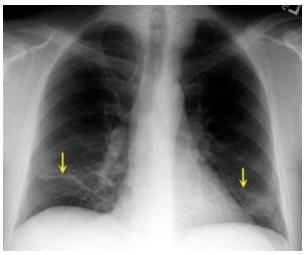
CASE EXAMPLES

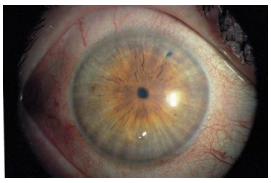
Must BALANCE **hypercapnia** and **coma** vs **severe hypoxaemia**, (i.e. fatal arrhythmias and hypoxic brain damage)

BARREL CHEST = lateral view = emphysema vs pulmonary fibrosis

ACUTE BRONCHITIS = Self-limiting (1-3wks) = no underlying lung disease

- common cold → headache, sore throat, nasal congestion → nasal decongestants and analgesia

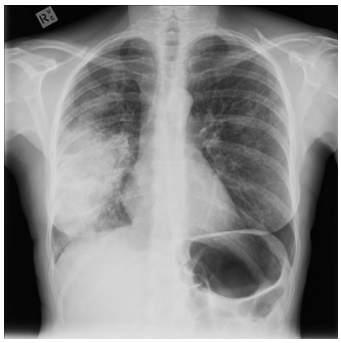




Case #1:

- 53 yo male, BMI 45
- D1 post 6 hour emergency laparotomy
- Morphine PCA + Drowsy and low sats, RR 6/min
- ABG – acute T2RF with low pH

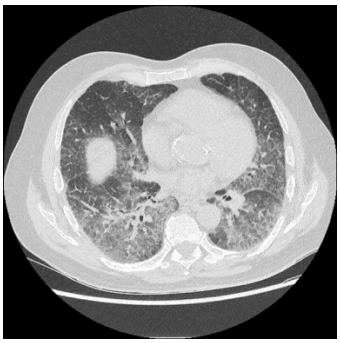
DDx	Post-op Linear atelectasis (bilateral lung base lines)	**Rx**	• Arousal + chest physio + walking around • Naloxone to treat morphine and pinpoint pupils
Ix	CXR		
Exam	Pinpoint pupils (miosis)		



Case #2:

- 40 yo previously healthy → Unwell for 7 days, productive cough
- T>39, sats 92% on 4L via NP
- Right sided pleurisy
- BP 86/44

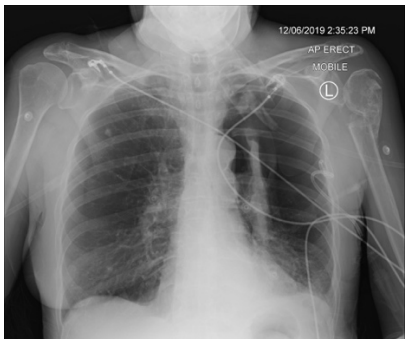
DDx	Severe Pneumonia + hemodynamically unstable
Ix	CXR → lobar pneumonia
Rx	Refer to high dependency unit



Case #3

- 78 year old male with known ILD → Still drives, keen to maintain independence as long as possible
- Restrictive lung function
- Sats 93% on room air, desaturates to 85% on 6MWT
- ABG – T1RF

DDx	ILD
Ix	CXR – ground glass changes ➔ V/Q mismatch
Rx	



- 78 year old lady with severe emphysema
- After left **upper lobe lung volume reduction** *with endobronchial valves*
- Heavy coughing fit, then rapid increase in breathlessness
- Hypoxic on 10L via facemask
- Resonant over left hemi-thorax
- ABG – compensated T2RF

DDx	Simple pneumothorax (due to shearing force of inserted valve)
Ix	CXR
Rx	Medical emergency – chest drain needed

Case Scenarios

	1st line Ix	2° diagnostic tests
Sudden onset of breathlessness and chest pain. Suspected pneumothorax in 22-year-old man with two previous pneumothoraces.	CXR	Chest drain
Suspected PE in 32-year-old woman recently returned from overseas. D-dimer positive.	CTPA	V/Q (if pregnant)
52-year-old woman with MND is wheelchair-bound. She is reported by family to be more sleepy.	MRI	Sleep Study, ABGs
22-year-old woman with severe asthma being managed in a severe asthma clinic.	Spirometry	Vitals check
45-year-old man with biopsy-confirmed interstitial lung disease (IPF) returns for review after 3 years and is now very breathless.	CXR, CT scan	Bloods
74-year-old man who worked for 5 years in the 1970's at the Wittenoom asbestos mine presents with chest pain and SOB. Clinically a right pleural effusion is present.	CXR, CT scan	High-res CT scan

RESPIRATORY EMERGENCIES

Pleural Effusion

Define

1) Collection of fluid in pleural cavity
2) Transudative or Exudative effusion

Cause / risk factors

Transudate	Exudate
➢ CCF	➢ PE
➢ Hypoalbumineamia (cirrhosis, CKD)	➢ Rheumatoid / RT
➢ Nephrotic syndrome	➢ Infection
➢ Constrictive pericarditis	➢ Neoplasia
➢ Meig's syndrome (ovarian cancer)	➢ Trauma
➢ Hypothyroidism	➢ CT disorder

Comp.

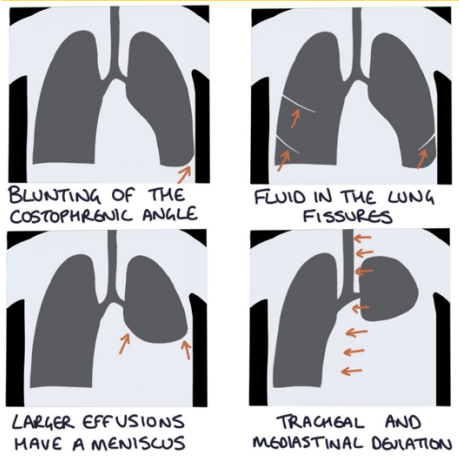
- Empyema (infected pleural effusion)
- Sepsis
- Death

SX

- SOB
- Stony dull percussion note over effusion site
- Reduced BS
- Tracheal deviation or mediastinal shift (if massive)

Ix

ERECT CXR:



Pleurocentesis – Light's criteria

- **Protein count**
- **Cell count**
- **pH**
- **glucose**
- **LDH**
- **M/C/S**

	Transudate	Exudate
pH	> 7.2	< 7.2 Low glucose
Protein (pleural:serum)	< 0.5	> 0.5
LDH (pleural:serum)	< 0.6	> 0.6
LDH: ULN	≤ 2/3	≥ 2/3

Acute Mx

- ***Conservative management*** for small effusions
- ***Pleural aspiration*** for **Larger** effusions → stick needle in and aspirate fluid. → ***May need repeat aspirations as effusion may recur***
- ***Chest drain*** for **Larger** effusions to drain the effusion and prevent it recurring. (risk of infection, pneumothorax)

Pneumothorax

Define

1) Air enters pleural cavity

Cause:

1) Spontaneously
2) Trauma
3) Iatrogenic (e.g. lung biopsy, mechanical ventilation, central line insertion)
4) *Lung pathology* (infection, asthma, COPD)

Risk factors:

1) Tall, thin young man
2) Contact sports

Comp.

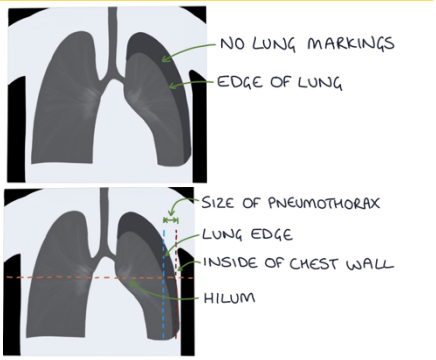
- Cardiorespiratory arrest
- Hypoxic end-organ damage
- Death

SX

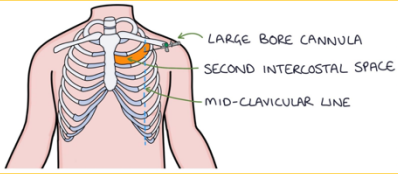
- Tracheal deviation (tension)
- Distended neck veins (TENSION)
- Pleuritic chest pain
- SOB
- Tachycardia, hypoTN
- Hyperesonant percussion note
- Reduced air entry on affected side

Ix

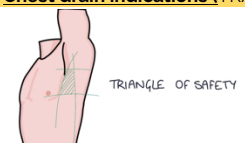
ERECT CXR:



Aspiration indications



Chest drain indications (TRAINGLE OF SAFETY)



- ***5th intercostal space*** (or the inferior nipple line)
- ***mid axillary line*** (or lateral edge of ***lat. dorsi***)
- ***Ant. axillary line*** (or lateral edge of ***pec. major***)

Nb: Needle inserted **above the rib** to avoid the neurovascular bundle

- Once drain insert → obtain a CXR to check the positioning.

Acute Mx

Conservative Mx:

- If no SOB and < 2cm rim of air on the CXR = NO Rx → Follow up in 2-4 weeks is recommended.

Aspiration indications (2nd IC space in MCL)

- If SOB and/or there is a > 2cm rim of air on CXR

Chest drain 5th IC space AAL ind. (TRAINGLE OF SAFETY)

- Unstable patients or bilateral or secondary pneumothoraces
- aspiration fails twice
- Tension pneumothorax

Pulmonary Embolism

Define

Clot within pulmonary arteries
Usu. secondary to DVT embolism

Risk factors:

1) Immobility - Post-op, Long-haul flight
2) Pregnancy
3) COCP/ HRT (w. estrogen)
4) Malignancy
5) Polycythaemia
6) SLE
7) Thrombophillia

Comp.

- Stroke
- MI
- Death

SX

- Pleuritic chest pain
- SOB
- Unilateral calf pain
- Tachypnoea
- Diaphoresis
- Tachycardia
- Altered mental state

Ix

Bloods:

- *FBC*
- *EUC*
- *Troponin*
- *ABG* – respiratory alkalosis (hyperventilation) BUT **low PO2**
- *Thrombophilia screen*

Well's score

- **Likely** = CTPA
  Helps to exclude pneumonia or malignancy
- **Unlikely** = D-dimer (if positive perform CTPA)

When do you use a V/Q scan?

- Renal impairment
- Contrast allergy
- Radiation risk (young adults, pregnant)

How long do you need to be anti-coag?

1) 3/12 = if obvious reversible cause
2) >3/12 = unclear cause, recurrent VTE or irreversible cause (e.g. Thrombophilia)
3) > 6/12 = if active cancer (then review)

Acute Mx

SUPPORTIVE MX

- Admit to hospital
- Vitals → O2
- Analgesia

INITIAL MX

1ST line = DOAC (Xa inhibitor)
2nd line = LMWH (if APS or CI for DOAC)

THROMBOLYSIS

- Massive PE + haem unstable
- IV fibrinolytic med
- Catheter directed thrombolysis into pulmonary arteries

LONG-TERM MX:

1st line – DOAC
2nd line – LMWH (pregnant or cancer)

PNEUMONIA

Def

Pneumonia	Infection of lung tissue causing inflammation of the lung tissue and sputum filling airways and alveoli
Community ACQUIRED (CAP)	Pneumonia outside hospital
HOSPITAL ACQUIRED (HAP)	Pnuemonia developed > 48 hr after hospital admission
Aspiration pneumonia	Pneumonia developed after inhaling foreign material (e.g. food)

Risk factors + complications

Known risk factors

- **Immunocompromised** (HIV, CF, T2DM, Steroid usage, elderly)
- **Sick contacts**
- **Smoking/tobacco consumption**
- **Occupational exposure** (asbestos, arsenic, mustard gas, radon…)
- **Hx of lung diseases** e.g. ILD, fibrosis or emphysemas

Complications :

1) Pleural effusion
2) Empyema
3) Lung abscess
4) Sepsis + bacteremia
5) Death

H+E

General Sx

- SOB,
- Productive sputum cough (+/- haemoptysis)
- Fever
- Pleuritic chest pain
- Delirium (acute confusion)
- Sepsis

General Signs

- Fever
- Hypoxia
- Tachypnoea + WOB
- Tachycardia
- Hypotension (late sign)
- Confusion / altered mental state

On examination

- Bronchial breath sounds (harsh BS on inspiration & expiration)
- Focal coarse crackles
- Dull percussion note (lung collapse or consolidation)

DDX:

Community Acquired:

- **Strep. Pneumonia (50%)**
- **HiB (20%)**
  - (young, acute, multilobular infiltrates, severe cold-flu like Sx)
- **Staph. Aureus** (CF)
- **Burkholderia Pseudomallei** (NT)

Hospital Acquired:

- **Pseudomonas Aeruginosa** (CF, bronchiectasis)
- **Klebsiella pneumonia** (DM, alcohol) – red current jelly sputum
- **Moraxella Catarrhalis** (immunocompromised or chronic pulmonary disease)
- **MRSA**

Atypicals:

- **Legionella = IV azithromycin + cipro**
- **Mycoplasma = 100mg doxy or macrolide**
- **Chlamydia = 100mg doxy or macrolide**
- **Q fever (Coxiella burnetti)**
- **Fungal (Pneumocystis jiroveci) = HIV low cd4 COUNT = co-trimoxazole for treatment and prophylaxis**

Ix

Bloods

- FBC + CRP (acute infection) – Nb: immunocompromised patients may not display raised CRP
- EUC
- ABG

CXR (1st line investigation)

- Focal consolidation

SPECIAL

- Sputum culture M/C/S
- Blood culture M/C/S
- Legionella and pneumococcal urinary antigens

Severity (CURB -65 -MORTALITY score

- Confusion (**new** disorientation to place, time and person)
- Ureamia (> 7)
- RR > 30
- BP < 90/60
- Age > 65yo
  - Score 0/1 (<5%): Consider treatment at home
  - Score ≥ 2 (: Consider hospital admission
  - Score ≥ 3 (>15%): Consider intensive care assessment

Mx

All treatment

- Follow local area guidelines
- Check Abx resistance specific to population
- Re-assess within 72 hours

	CURB-65 score	Where?	Rx options
Mild CAP	0-1	Home	5 DAY 1g amoxicillin tds PO or macrolide PO • PLUS: 100mg doxycycline PO • Vit C + D + sleep (boost immune system)
Mod CAP	≥ 2	Inpatient	
Severe CAP	≥ 3	Hospital	7-10 DAY 1g benPen qid PO or macrolide PO • PLUS: 100mg doxycycline PO
Emergency	≥ 4	ICU	➢ IV azithromycin (atypicals) ➢ IV ceftriaxone (MRSA, pneumococcus, enterobacter)

HYPERSENSITIVE TO PENICILLIN

➢ 500mg cefuroxime PO bd

ANAPHYLAXIS TO PENICILLIN

➢ 400mg moxifloxacin PO od

Case scenario:

56 year old indigenous man man living in a NSW Regional Centre. He is a university-trained Social Worker working with youth who have interacted with Juvenile Justice. Presents to the Base Hospital unwell with breathlessness, cough and fever. You are JMO in ED and he is allocated to your care. It is February.

Background history:-

- **Known limited left lower lobe bronchiectasis with only mildly impaired lung function**
- **Smoked until 6 years ago**
- **Type 2 diabetes under much better control over the past two years during which time he has been more adherent to disease management strategies**
- **Penicillin allergy; developed rash 35 years ago; tolerant of cephalosporin antibiotics**

1. Attended an international workshop for Indigenous advocates in India 8 weeks previously
2. Had proven UTI with pyelonephritis and was hospitalised for 10 days. Eventually recovered enough for travel and returned to Australia 4 weeks ago. Urine and blood cultures were positive for fully-sensitive E. coli
3. stopped off in Darwin on the way home and spent some time with a cousin who lives 30 minutes out of Darwin. He was there for three weeks and returned home one week ago.

Management course:

1. vitals → Ceftriaxone + azithromycin --→ DVT prophylaxis, → BSL monitor
2. **Blood culture results (+ve for strep. Pneumo). Urine culture (+ve for pneumococcal)**
3. Sudden deterioration after Rx→ presumed sepsis → ID consult → extending Ab coverage with **meropenem**
4. **Blood cultures return =** ESBL coliform; carbapenemase producing (meropenem resistant

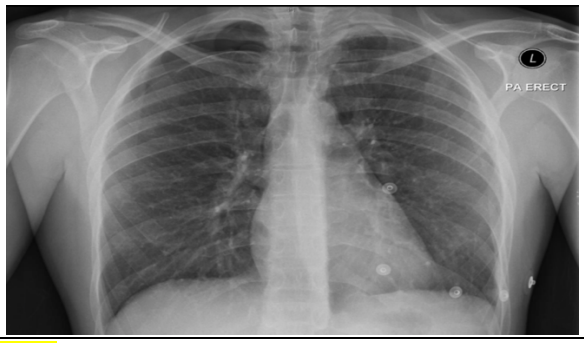
Where did this organism come from?

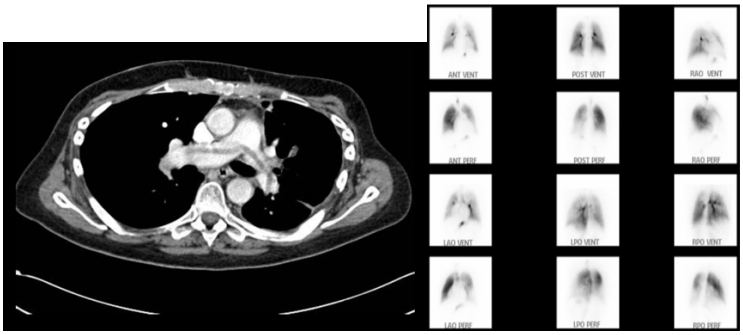
1. **Most likely**: Contaminated water and food (from India) + lack of Abx stewardship

Pneumonia, TB, Pleural Effusion

	Case 1	Case 2	Case 3
Scenario	*Sudden onset of breathlessness and chest pain. Suspected pneumothorax in 22 year old man with two previous PTX*	*Suspected pulmonary embolism in 32 year old woman recently returned from overseas. D-dimer positive.*	*52 year old woman with MND is wheelchair-bound. Reported by family to be more sleepy*
Primary Ix	**CXR**	**CT pulmonary angiography**	**MRI**
Other Ix / checks	• Chest CT • V/Q perfusion test (safer than CTPA for age)	• V/Q perfusion scan [if pregnant] • D-dimer test [already conducted]	○ Sleep study → OSA (hypercapnic) ○ ABGs ○ Nerve conduction study / EMG
	Case 4	**Case 5**	**Case 6**
Scenario	*22 year old woman with severe asthma being managed in a severe asthma clinic*	*45 year old man with biopsy-confirmed interstitial lung disease (IPF) returns for review after 3 years → is now very breathless* • *Crackles on chest & cyanosed*	*74 year old man who worked for 5 years in the 1970's at the Wittenoom asbestos mine presents with chest pain and breathlessness. Clinically a right pleural effusion is present.*
Primary Ix	Lung function test / Spirometry	CXR or chest CT	CXR /Chest CT
Other Ix / checks	○ Med compliance ○ # of hospital Ax (related to asthma)	○ High resolution CT scan ○ Spirometry ○ Smoking Hx?	○ **DDx**: Mesothelioma, Silicosis, CWP

Interpret results of these respiratory investigations and imaging for common respiratory pathologies





CASE 1:

Right apical pneumothorax (lung margin)
Left lung comparably normal

CASE 2:

- ***CTPA: Black line*** *along both pulm. Arteries indicates clot*
- High ventilation but low perfusion (high V/Q) in right lung ➔ PE

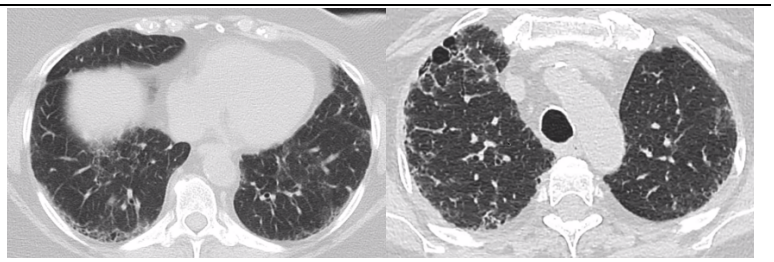
Respiratory Function Laboratory

Last Name:	Example	First Name:	Case
Identification:	812334	Date of Birth:	3/3/1996
Gender:	female	Age:	20 Years
Weight:	60.0 kg	Height:	168 cm
BMI:	23.9	Ref. Physician:	Dr Williams
Medication:	None taken today	Date:	20/1/2018
Clinical Notes:	SOB		

Spirometry

Post test performed after 4 puffs (400ug) of salbutamol via spacer.

		Pre	% Ref	Post	% Ref	% Chg	Ref	Ref LL
FEV 1	L	2.12	59	3.15	88	48	3.57	2.87
FVC	L	3.30	80	3.87	95	12	4.09	3.27
FEV 1 % FVC	%	64		81			88	78
Measurement time		11:00AM		11:15AM				



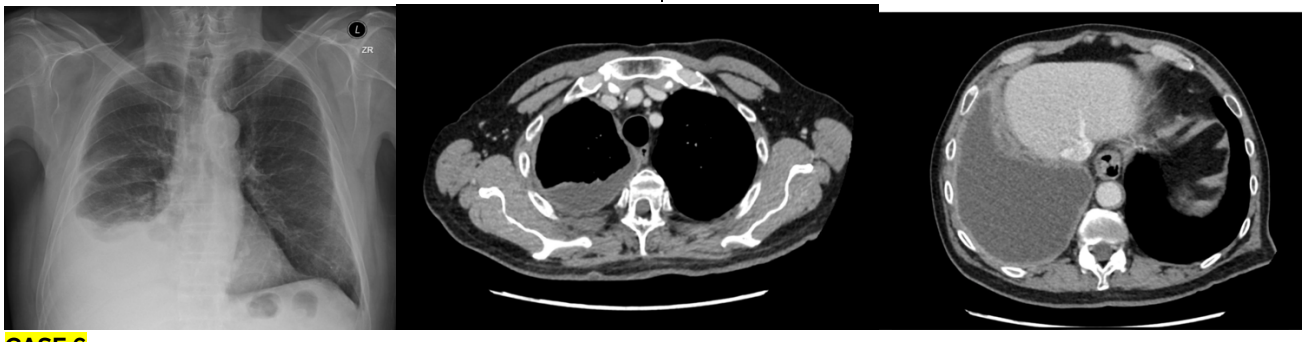
CASE 4:

- Moderate **airflow limitation** = **obstructive pattern [loss of elastic recoil]**
- $FEV_1/FVC < 50\%$ = severe
- Reversible obstruction w/ bronchodilator (>12%,
- *Need **ALL** lung vol. to diagnose restrictive disease*

CASE 5: lung window → see vasculature of lung

Diffuse prominent scarring w/ honeycombing at periphery at posterior lung base → radiological progression

- *Bronchiectasis → airway larger than vessels*
- *Cannot see artery or vein → need to use contrast*



CASE 6:

- *R-sided pleural effusion in RLL → use Light' criteria to determine if:*
  - *Exudate (e.g PE, pneumonia, DVT/PE) → needs catheter drainage*
  - *Transudate (e.g. CHF, cirrhosis, nephrotic syndrome) → conservative Mx*

***Supine CT** =fluid flows downwards due to gravity & pleural thickening*

ASTHMA VS COPD

Define

ASTHMA

- Chronic **reversible** airway inflammation and bronchoconstriction due to hypersensitive airways causing periodic attacks of wheeze, SOB, cough, chest tightness
  - FEV1 > 12% or > 200mL after bronchodilator
- *1st presentation* = **reactive airway disease** until recurrent

COPD

Umbrella term of chronic progressive **irreversible** obstructive lung disease characterised by limited airflow due to either::

- *Enlarged air sac* = emphysema,
- *Lung tissue destruction* = chronic bronchitis – persistent cough >3/12 past 2 years

Key Q's

ASTHMA

Timing
- Intermittent vs Persistent
- Worse at night (diurnal variability)

Severity

Mild-Mod	• ONLY sats < 94% • (can talk/walk normally
Severe	• RR> 25, HR >110 • cannot complete sentences, • sats < 94%
Life-threaten	• <92 >33 CHEST • Tired or in shock • No wheeze, "silent chest"

Control
- <2 daytime Sx/ week
- <2 SABA relief/week
- **NO** activity limitation
- **NO** nocturnal waking

- Good = All
- Sub-optimal/Partial = 1 or 2
- Poor = 3 or more

COPD

Timing Intermittent vs Persistent

Severity

Mild	• SOBOE, • recurrent chest infections
Mod	• +COPD-X needs oral ABx and CS, • SOB on walking on flat surface
Severe	• SOB at rest – tripoding, pursed lip breathing • dependent living • +++ freq. exacerbations

Control
- Functional capacity =(FEV1 changes)
- Exacerbation frequency
- # of hospital admissions

Severity of obstruction based on FEV1 (spirometry) – GOLD Criteria

Stage 1: FEV1 >80% of predicted
Stage 2: FEV1 50-79% of predicted
Stage 3: FEV1 30-49% of predicted
Stage 4: FEV1 <30% of predicted

RF

ASTHMA
- **Atopy** = dermatitis, allergic rhinitis, food allergies
- **FHx of asthma**

COPD
- **Lung cancer**
- **Heavy smoker**
- **Co-morbidities** → CVD, A+D, DM, OP, OSA, GORD, cancer

Trigger

ASTHMA
- **Environment irritants** (dust, cold/damp, exercise), animal dandur
- **Night time or early morning**
- **Infection** = URTI
- **Strong emotions**
- **Iatrogenic** = cigarette smoke, BB/ASA

COPD
- **Environment irritants** (dust, cold air, exercise)
- **Occupational exposure** (firefighter, miner, asbestos, cleaner)
- **Infection** = URTI
- **Medical** = a1-antitrypsin def.

Ix

ASTHMA
- **Bilateral polyphonic widespread wheeze**
- FBC – eosinophils +++
- IgE
- CXR = Hyperinflated during asthma attacks
- ***Spirometry (FEV1/FVC < 0.7) – BRONCHODILATOR REVERSIBLE***
  - ***Exacerbated with cholinergic***

COPD
- ***FBC** (High Hb), **EUC**, **LFT** (hepatic failure – cor pulmonale), **CRP**, **trops, ABG***
- ***Sputum culture and blood culture (if septic or significant infection)***
- ***ECG** (R heart strain – V1-V3)*
- ***Serum alpha-1 anti-trypsin***
- ***Spirometry (FEV1/FVC < 0.7)** and **TLCO to check CO diffusion***
- CT = *thickened wall dilated bronchi (bronchiectasis)*

CXR normal:
- ***Emphysema*** = hyperinflated lungs + bullae
- ***Chronic bronchitis*** = ++ bronchial markings

Acute Mx

ASTHMA
- DR ABCD → Refer to ED → **O SHIT ME**
  - *Cannot lie flat w/o SOB, FEV1 < 60, +WoB, cyanotic*
- **FiO2 -NRBM → maintain > 95% (6L/min)**
  - Monitor sats, RR, HR every 15 mins
- Inhaled ***bronchodilators*** (burst SABA + SAMA)
  - 12x puffs 100µg Salbutamol (x3 every 20mins) – 6x (<6yo)
  - 8x puffs 250µg Ipratropium (x3 every 20mins) – 4x (<6yo)
- Oral ***prednisolone or IV hydrocortisone***
  - Child = 2mg/kg oral (3-5 days then stop) or IV 4.5mg/kg
  - Adult = 50mg oral (5-10 days then stop) or IV 100mg qid
- ***IV Theophylline/aminophylline*** (PDE3/4i – SMC relaxant) – *fast onset of action but*
  - *low TI = means (NEED TO measure toxicity 5 days after starting)*
- ***IV MgSO4*** (SMC Relaxant)
  - 0.1-0.2mM/kg (child) – max 10mM
  - 10mM in adult
- ***Escalate care if not responding or COMA*** → arrange T/F to tertiary or ICU (alert senior staff and anesthetist to prepare for I+V if not responding)

Once haem stable
1. Monitor response to Rx based on RR, WOB, Peak flow, O2 sats, chest auscultation
2. Ventolin stretch (every 1 hr → 2hr → 3hr)
3. Asthma action plan (continue pred + stretch to every 6 h)

COPD
1. DR ABCD → Refer to ED
2. **FiO2 -venturi mask** → titrate to > 88% (BIPAP + O2) = improve resp. acidosis
   - **CI = vomiting, facial trauma, altered mental state**
3. **Inhaled bronchodilators** (burst SABA + SAMA)
4. **Oral Steroids** (50mg once daily for 5 days then stop)
5. **500mg Amoxil if** infection present (see sputum M/C/S)
6. ***Escalate care if not responding***
   - *Will need non-invasive BIPAP + I/V*
   - *IV aminophylline*
   - *Doxapram (if NIV or intubation not appropriate)*

Once haem stable
1. Pulmonary Rehab → (e.g. chest physio, mech. Vibration)
2. Further tests:
   - **FBC** = polycythaemia VS anaemia
   - **Mantoux test** = TB (ENDEMIC nation)
   - ECG = R heart strain (v1-v3) → +++ risk of IHD
   - CXR = *pneumothorax, pneumonia, pulm. fibrosis*
   - Polysomnography = OSA
3. F/U = in 1 week ➔ **complete 40mg pred course over 5 days**
   - respiratory physicians r/v
   - **Vit D and Ca suppl** (if long-term steroid usage)

Long-term Mx

ASTHMA

DKA-E	• Use inhalers w/ spacers • **Dry powder vs aerosols (MDI)** • Educate carer/parent/patient

Non-pharm Mx	• **Stop smoking**
	• If concurrent cold
	  ○ FESS + saline irrigation
	  ○ Codral cold + flu to sleep
	  ○ Betadine gargle
	• **Preventer AM prior to any activity**
	• **Create non-sensitive environment:**
	  ○ Avoid sick contacts
	  ○ Warm (20-21°C)
	  ○ Humidifier

Pharm Mx	• **1st line** = **Symbicort (LABA/ICS – dry powder**)-adults OR **salbutamol + Seretide** for children SABA A/E = tremor, tachycardia, tachyphylaxis, hypoK • **2nd line** = SABA PRN + **LOW** dose ICS/LABA -*fostair* • **3rd line** = SABA PRN + ***HIGH*** dose ICS/LABA – *breo elliptica* • **4th line** = Add theophylline, anti-IgE, IL-4,5,13 *__montelukast__ = if concurrent allergic rhinitis or hayfever: > A/E = nightmare, mood change, aggression
Px	➢ Yearly flu/COVID jab and asthma review
Info	National Asthma Council,
Refer	➢ Paediatrician or respiratory physician
Safety net	**Asthma Action Plan** (self-mx) + **rescue pack for Xcerbations**
FU	➢ Yearly LFT – spirometry (FEV1 tracking) if poor control ➢ Yearly flu jab and asthma review ➢ Advise exercise and avoid smoking ➢ Consider stepping down if improving

COPD

DKA-E	What is it? → complications and risks of COPD ➢ Poor prognostic factors = BMI < 21, smoker, O2 sats < 92%
Non-pharm Mx	• **Stop smoking → nicotine replacement therapy** + vernacaline + CBT • **Nutrition + exercise** • **Manage co-morbidities** (e.g. A+D, DM, IHD, OP)
Pharm Mx	**Home O2 therapy** (must NOT be smoking) indicated if ➢ PaO2 < 55 (chronic hypoxia) ➢ PaO2 < 60 w/ PHTN, , polycytheamia, cor pulmonale **LAMA/SABA PRN *plus*:** 1st line = low dose ICS + vit D/Ca 2nd line = low dose ICS/LABA 3rd line =High dose ICS/LABA 4th line = oral CS + theophylline + immunotherapies *__Long-term prophylactic Abx__ = azithromycin **__Oral mucolytics (e__.g. carbocisteine)
Surgery	**Endoscopic lung volume reduction surgery (ELVR)** ➢ Thermal ablation or coils
Px	➢ **Yearly flu/COVID jab and asthma review**
Info	**Lung foundation Australia,** Quitline
REFER	• Pulmonary rehab – improve exercise tolerance • pharmacist home medicine review • palliative care services
Safety net	• GPMP +/- advanced care directives • COPD action plan • Non-urgent review = rapid FEV1 decline, COPD<40, recurrent chest infections
FU	• Yearly LFT – spirometry (FEV1 tracking) • 4 -6/52 after medications

DDx – wheezing

- **Anaphylaxis or FB**
- **Infection** (bronchitis, pneumonia)
- **OSA**
- **GORD**
- **Asthma vs COPD**
- *Rare* = CF, malignancy, CHF, vocal cord dysfn

Medication A/E

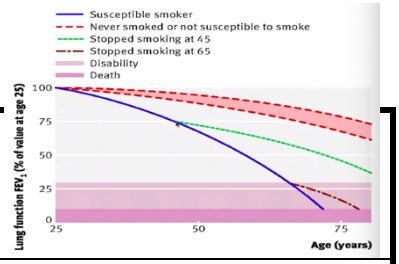
- **SABA = tachycardia +tachyphylaxis (builds up tolerance) hypoK** (myalgia, parasthesias, fatigue, N/V, polyuria)
- **Inhaled CS = oral thrush**
- **Oral CS = OP**, *wt gain, immunosupp. ↑BSL, VTE risk, insomnia, psychosis, cataracts, fatigue and light-headed*

Drugs to avoid
- **BB = avoid in asthma** (bronchoconstrict)
- **NSAID/aspirin & Bee products**

Indications for referral

- **Severe URT disease (***adenoids, severe rhinitis)*
- **Resp. distress in early life (***PCD, TTN, bronchopulm. Dysplasia)*
- **Persistent crepitation**(e.g. empyema, fibrosis, atelectasis)
- **Severe chest wall deformity** (pectus excavatum, carinatum, Harrison's sulcus - rickets)
- **Screen for CF** (CFTR1 gene, sweat test) → *clubbing, enlarged nasal polyps, recurrent chest infections, infertile, DM*

COPD management:



1. Stop smoking

Use **Champix / vernacaline (partial nicotine agonist)**

- Improves abstinence than patches
- Avoid inhaled particular matter
- **A/E**: nightmares + N+V

2. Drugs

COPD Mx:

- ***Mild (FEV₁ 60-80%):*** *LAMA*
- ***Mod: (FEV₁ 40-59%):*** *LABA/LAMA*
- ***Sev: (FEV₁ <40%):*** *triple therapy = LAMA + ICS/LABA (Seretide)*

**Use SABA/SAMA as reliever in any cases*

Beta agonists [SMC relaxation]	***SABA*** *salbutamol, terbutaline*	• **Symptom relievers → A/E tremors, palpitations (tachycardia), hypoK** • *Rapid onset – 4-6hr effect (BUT may develop tolerance)*
	LABA *(-erols)= salmeterol*	• Can be added with SABA with caution • *Ultra LABA* = indacaterol (aim to increase compliance)
Anti-cholinergic	**SAMA** ipratropium bromide	• **1ST line reliever** → greater increased in FEV1 than SABA/LABA • Slower than SABA – 4-6hr effect
	LAMA tiotropium bromide	• **1ST line for COPD** = mild airflow obstruction ○ **Add LABA if** mod airflow or exacerbations • Improves health status + effectiveness of pulm. Rehab

Low dose macrolides	**Low dose azithromycin** *"reduce chronic airway inflammation & mucus production"*	• **Anti-Inflammatory** = ↓↓↓ chronic airway inflammation
		  ○ ↑↑↑ phagocytosis of dead neutrophils, and pro-inflammatory production, defensins
		  ○ ↓↓↓ ROS, adhesion molecule, chemotaxis
		• **Anti-secretory** = ↓↓↓ mucus production
		• **Anti-Microbial** = ↓↓↓ pathogen load → reduce infective exacerbations

ICS	**Fluticasone**	• If increased exacerbation and frequency • Combined w/ LABA (e.g. **Seretide = Fluticasone / salmeterol**) for severe diseases in asthma/COPD overlap syndromes ○ Assoc. with hoarseness, oral candidiasis, pneumonia risk

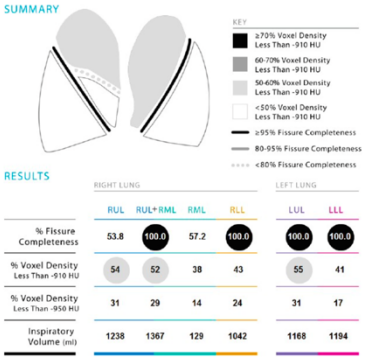
3. Pulm. Rehab

- improves exercise tolerance/capacity, gas exchange and QoL
- Mucus excretion techniques
- Important to do prior to ELVR
- Consider Transplantation? (if death within 3 years)

4. Long-term O_2 Therapy

prolong survival

- **(1) PaO_2 < 55mmHg** OR
- **(2) PaO_2 < 60mmHg + pulm . HTN OR** Polycythaemia
  - For type 2 resp. failure → need BiPAP at night w/ O_2 therapy
- **Only if *smoking ceased***

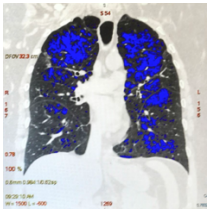


5. Endoscopic lung volume Reduction (ELVR)

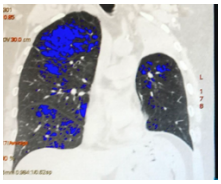
= targeted lung collapse (atelectasis)

- ***AIM***: Deflate non-functional lobes causing mass compression effect
  - Reduce pulm. HTN & improve V/P matching & gas transfer (↑DLCO)
- ***GOAL***: Improve lung function, QoL and mortality w/ advanced COPD
- ***Fissure analysis*** *[to ensure **NO** collateral ventilation between lobes for reliable collapse of targeted area]*
  - ***Fissure > 95% = YES (minimal holes) → proceed ELVR***
  - *Fissure 70-95% → Perform CHARTS*
  - *Fissure < 70% → NOT-indicated*

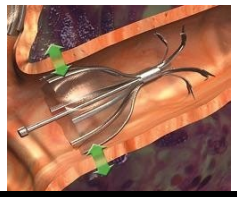
	Description	Location	Adv.	
Endobronchial 1-way Valves	Occlude lobe destroyed by emphysema **Indications**: • *FEV_1 = 20-45%,* • *RV > 175% (hyperinflation)* • *6MWT > 100-500m*	**Upper** + **lower** emphysema	Fully **reversible** **[can remove]**	• Pneumothorax • No collateral ventilation (need sealed fissures) • Should be already having pulm. Rehab + med Mx
RePneu Coils	Placed into **smaller airways** to collapse them	**Upper** + **lower** emphysema	No collateral ventilation or fissure concern	**Irreversible** after 4 weeks • Haemoptysis • Irritation & infection → COPD exacerbations
Thermal vapour ablation	**Targeted heat damage** to bronchi → *induce* localised inflammation & fibrosis/scarring to non-ventilated airways	**Upper** lobe **ONLY**		**Irreversible** **Local/systemic reaction** = • Pneumonia • COPD exacerbation

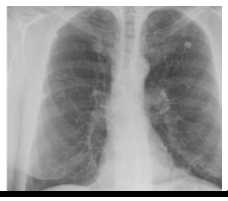


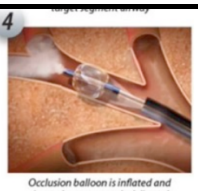
***Blue** = DEAD space (non-functional tissue)*



***After upper lobe lobectomy** → raised left hemidiaphragm & left trachea midline shift*







LUNG CANCER (HIGHEST MORTALITY CANCER)

Def

Small cell lung cancer	Non-small cell carcinoma (NSCLC)		
	Adenocarcinoma	Squamous cell carcinoma	Large cell carcinoma
• 15% of all lung cancer • >60% metastatic presentation. • Staged as "limited" or "extensive • **SMOKERS**	**Most common NSCLC** • **MOST common lung cancer in non-smokers** • **SMOKERS (current or ex-)**	• 20% of NSCLC • **More common in smokers**	
• **Central** • SCLC neuroendocrine cells contain **neurosecretory granules** releasing neuroendocrine hormones → paraneoplastic (ACTH, SIADH) • "**oat cell**" = Scant cytoplasm, ill defined borders • **neuroendocrine markers** (*CD56, Neural cell adhesion molecule, synaptophysin, chromogranin*) • **TTF-1 Positive**	• **Peripherally** • Involves mucus producing gland cells within lung [Glandular differentiation] • **Usu. EGFR-mutant lung cancers →** • **Young Asian Women non-smokers** • **TTF-1 Positive**	• **Central** • **Obstructs larger airway →** Epithelial cells change from columnar → cuboidal →squamous • Keratinization with intracellular bridges • PTHrP → HPTH • **CK7 and CK20 negative** • **TTF-1 Negative** • **Nap-A positive**	• Lack cytologic and architectural features of small-cell carcinoma and glandular or squamous differentiation • Rapidly grows, can present in peripheral OR proximal lung tissue

Risk factors

Known risk factors
- **Smoking/tobacco consumption**
- **Occupational exposure** (asbestos, arsenic, mustard gas, radon…)
- **Hx of lung diseases** e.g. ILD, fibrosis or emphysema
- **Mutations: CYP1A1 genetic polymorphism**, Rb, p53, T790M mutations

Unknown risk factors
- Low fruit and vegetable diet
- Ni exposure
- Passive smoking

H+E

General:
- SOB,
- cough +/- haemoptysis
- Finger clubbing
- Recurrent pneumonia
- UWL
- Lymphadenopathy (usu. supraclavicular LN)

Extrapulmnoary Mass effect
- **SVC obstruction = "pemberton's sign" = medical emergency**
- **Horner's syndrome** = Pancoast tumour (compress SNS chain)
- **Phrenic nerve palsy** = SOB
- **RLN palsy** = hoarseness
- **Metatastic spread**:
  - Bone (pain, radiculopathy)
  - Brain (AM nausea, FND)
  - Liver (abdo discomfort)

Paraneoplastic effects
- **SCLC** (SIADH - hypoNa, ACTH – cushing's, LEMS - VGCC)
  - LEMS = proximal muscle weakness
    intraocular weakness (diplopia),
    levator muscle weakness (ptosis)
    pharyngeal muscle weakness (slurred speech and dysphagia)
    autonomic dysfn (dry mouth, impotence, blurred vision)
- **SCC** (HPTH - ↑hyperCa = bone, stones, groans, overtones
- **Bronchial + gastric carcinoma = acanthosis nigricans**
- Bronchial + pancreatic carcinoma = migratory thrombophlebitis "trosseau's sign of malignancy"

DDX:

- **Resp**: **Lung cancer, COPD, asthma, bronchiectasis**
  - **Infection (*VIRAL* – pneumonia, *LATENT* – TB, *ATYPICAL* -fungal)**
  - **Sarcoidosis**
  - **Pulmonary oedema** (unlikely due to no haemoptysis)
  - **Pleural effusion (1° or 2°)**
  - **Pulmonary fibrosis / ILD**
  - ***Unlikely mesothelioma** (no asbestos work)
- **CV: CHF, tamponade, anamia, PHTN, IE,**
- **Renal impairment**
  - **Antibiotics → AKI**
  - **Diuretics (ACEi = cough, others = hypoTN)**
- **MSK: scoliosis, obesity, costochondritis (chest pain?)**
- **Endo**: hypothyroidism
- **Neuro**: Myasthenia gravis, GBS, MND
- **GI**: GORD (causing pneumonitis), 2° metastasis (from colon, liver?)

Ix

CXR (1st line investigation)
- *Hilar enlargement*
- *Peripheral opacity (visible lesion in lung field)*
- *Unilateral pleural effusion*
- *Lung collapse*

Paraneoplastic check
- *EUC (hypoNa)*
- *CMP (HPTH)*

Secondary ix:
1) **Staging CT scan (+/- contrast enhanced – highlight different tissues)**
2) **PET-CT = metastasis check**
3) **Bronchoscopy w/ endobronchial USS (EBUS) – US guided biopsy**
4) Pleural fluid cytology, LDH, protein, glucose (need > 100mL = more the better)
5) **Histological diagnosis**
6) **Molecular testing**: EGFR, ALK, PD1 (Implications for treatment in stage IV disease

Histology

Diagnosis & staging of lung cancer = TNM staging

Important cut-offs for any primary lung tumour:
- N2 disease = at least stage IIIA
- N3 disease = at least stage IIIB
- M1 disease = certain stage IV

Stage of lung cancer → suitable Rx

	Suitable Rx	*5-year survival rate*	*Cure rate*
I-IIIA	Resectable *+/- adjuvant chemo/immuno*	*50%*	*25-80%*
IIIB	Concurrent chemo + RT *+/- immuno*	20% (18-24 MTHS)	20-25%
IV	Palliative systemic Rx	*1% (10-12 MTHS)*	*Palliative*

TABLE 5] Lung Cancer Stage Grouping (Eighth Edition)

T/M	Label	N0	N1	N2	N3
T1	T1a ≤1	IA1	IIB	IIIA	IIIB
	T1b >1-2	IA2	IIB	IIIA	IIIB
	T1c >2-3	IA3	IIB	IIIA	IIIB
T2	T2a Cent, Tive PI	IB	IIB	IIIA	IIIB
	T2a >3-4	IB	IIB	IIIA	IIIB
	T2b >4-5	IIA	IIB	IIIA	IIIB
T3	T3 >5-7	IIB	IIIA	IIIB	IIIC
	T3 Inv	IIB	IIIA	IIIB	IIIC
	T3 Satell	IIB	IIIA	IIIB	IIIC
T4	T4 >7	IIIA	IIIA	IIIB	IIIC
	T4 Inv	IIIA	IIIA	IIIB	IIIC
	T4 Ipsi Nod	IIIA	IIIA	IIIB	IIIC
M1	M1a Contr Nod	IVA	IVA	IVA	IVA
	M1a Pl Dissem	IVA	IVA	IVA	IVA
	M1b Single	IVA	IVA	IVA	IVA
	M1c Multi	IVB	IVB	IVB	IVB

Mx

All treatment
- requires MDT input (consultant, pathologist, surgeon, oncologist)
- Depends on stage and grade
- Stop smoking + TREAT symptoms (pain mx, SOB -SA morphine?, cough)

	Prognosis	Rx options
Small cell lung cancer	Poor Early dissemination "2-4 mths survival"	• Chemotherapy (High response rates to chemo (≈ 70%) • Radiotherapy (bare minimum) • Endobronchial Rx w. stents or debulking (part of palliative intent) → relieved bronchial obstruction

Non-small cell lung cancer	Better
	(less responsive to chemo)
		• 1st line = lobectomy / segmentectomy / wedge resection
		  ○ **CI** = Malignant cells on effusion and inadequate pulmonary reserve
		• 1st/2nd line = Radiotherapy (if caught early)
		  ○ AC = EGFR +ve or ALK +ve = osimetinib + chemo (if fit)
		  ○ AC = PDL-1 > 50% (immune + chemo), PDL -1 < 50% (immuno+ chemo)→ adjuvant chemo.
		• 3RD line = chemotherapy (adjuvant vs palliative intent) = aim to improve survival and QoL
		• Endobronchial Rx w. stents or debulking (part of palliative intent) → relieved bronchial obstruction

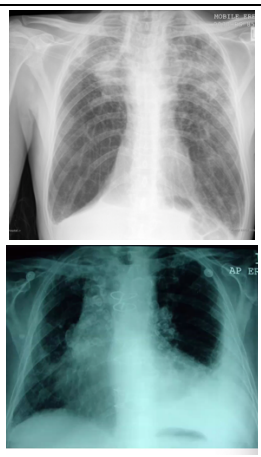
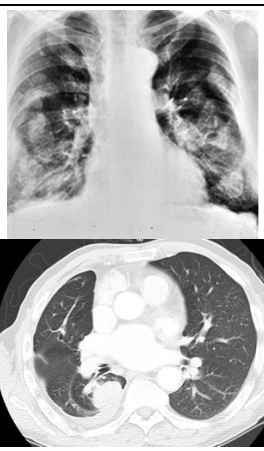
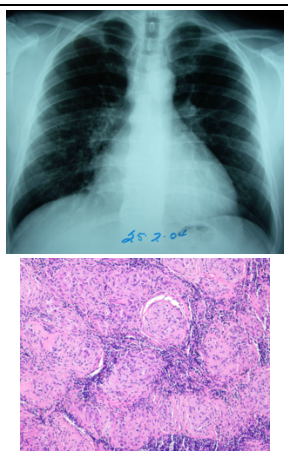
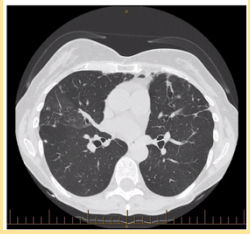
Mesothelioma	Very poor	• Chemotherapy (palliative intent but increases survival) • huge latent period between asbestos exposure to dx ≈ 45 years

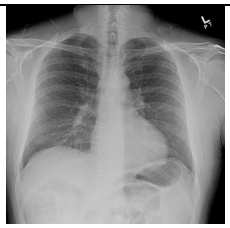
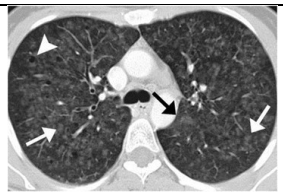
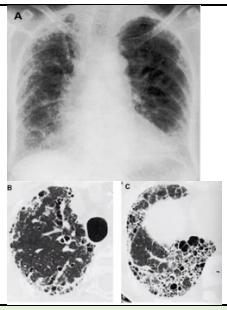
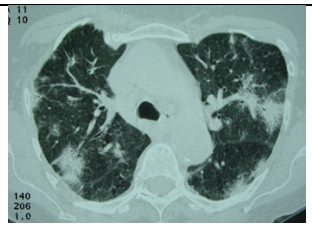
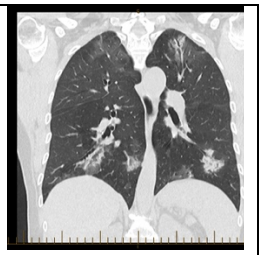
INTERSTITIAL LUNG DISEASE AND PULMONARY FIBROSIS

- **INTERSTITIAL LUNG DISEASE (ILD)** = umbrella term that affects lung parenchyma (lung tissue) causing **IRREVERSIBLE** inflammation and fibrosis (i.e. normal elastic functional lung tissue REPLACED BY stiff scar tissue)
- **Diagnosis = HRCT → ground glass appearance** = hazy areas of increased density
  - If diagnosis unclear ➔ lung biopsy → histology to confirm

General Mx for ILD:

1) Treat/remove underlying cause
2) Stop smoking
3) PT + pulm rehabilitation
4) IUTD – flu, COVID, pneumococcal
5) Advanced care planning (+/- lung transplant
6) Home oxygen if hypoxic at rest

				 **Bronchiectasis**
Dx	**Silicosis** *DDx for egg-shells: Sarcoidosis, TB*	**Asbestos-related pleural disease:** • **Asbestosis** = Diffuse ILD **ONLY due to** high exposure • **Mesothelioma** = pleura cancer (any asbestos exp) • **Lung cancer (adenocarcinoma)**	**Sarcoidosis** *[Chronic granulomatous disease] DDx: TB, berryliosis*	• Recurrent haemoptysis, LRTi in childhood • Pectus carinatum, **clubbing**, cyanosis • Assoc. CF, kartegener's • **CXR** = air bronchograms, dilated cystic changes • **CT** = honeycomb - Large-dilated airways in periphery of the lung (abnormal)
Risk Factors	• Sandblasting • Stone masons → kitchen marble tops • Quarries • Tunnels / coal workers	• Asbestos Mining (e.g. Woodsreef) • Insulation & steam pipes (hosp.) • Builders & Shipyards • **Mechanics** = Brake-pads for cars	• Systemic (fever, rash, erythema nodusum, uveitis) • HyperCa • Afro-carribean	**Drug induced pulm. Fibrosis** ➢ MTX ➢ Bleomycin / daptomycin ➢ Amiodarone ➢ Cyclophosphamide ➢ Nitrofurantoin
Ix	**MARKEDLY ABNORMAL** • Upper lobe predominant • Fibrosis/Scarring in upper lobe causing mediastinal upward shift as lower lobe dilates to compensate • +/- effusion • **Egg-shell calcification** = Calcified lymph nodes	**Asbestos is lung fibrosis due to inhalation of:** *Asbestos types* • *Serpentine* (white asbestos – chrysotile) • *Amphibole* (crocidolite =**blue** asbestos (Wittenoon), amosite= **brown** asbestos) **Asbestos is BOTH fibrogenic and oncogenic causing** 1. **Lung fibrosis** 2. **Pleural thickening → Calcified Pleural plaques (30-50%) (**does NOT follow anatomical lung boundaries) 3. **Pleural effusion** (usu. benign →unilateral)	• **Upper lobe** predominant • Bilateral hilar adenopathy • Pulmonary fibrosis **Pathology:** • Non-caseating granuloma (necrotising) • histiocytes, • giant cells, • lymphocytes	**2nd Pulmonary Fibrosis:** ➢ RA ➢ SLE ➢ Systemic sclerosis ➢ Alpha-1 antitripsin deficiency

					
Dx	**Hypersensitivity Pneumonitis** (extrinsic allergic alveolitis)	**Respiratory Bronchiolitis-associated ILD**	**IPF** (idiopathic pulmonary fibrosis)	**Cryptogenic Organising Pneumonia (COP)** *"bronchiolitis obliterans organising pneumonia"*	**Eosinophilic pneumonia**
Cause	• **Bird fancier's** (bird droppings) • **Farmers lung** (mouldy spores in hay) • **Mushroom worker** (mushroom antigens) • **Malt worker** (mould on barley)	**Smokers** (current and previous) • 3rd -6th decades • insidious onset **Airway inflammation that spreads to lung tissue*	• Ex-smokers • M> F in 60s-70s • Insidious onset dry cough for > 3 months *Assoc with:* • finger clubbing, • bibasal fine inspiratory crackles	• Median age: 50-60 • Non-smokers • SOB, fever, cough, lethargy • **Unresponsive to antibiotics** • Idiopathic • Triggers = infection, meds, RT, toxins, allergens	Similar imaging to COP
Ix	• **IgG driven (Type 3 hypersensitivity)** • Bronchoalveolar lavage – washing airways w/ fluid then collecting fluid to test ➔ ++lymphocytes, mast cells	• Centrilobular nodules with • **Ground glass change** = ***Pathology***: • Pigmented macrophages within bronchioles/ • peribronchiolar alveolar spaces	• **Lower lobe predominant** + sub pleural • Peripheral reticular opacity • **Honeycombing** • **Can look like RA → need to exclude and Rx for RA**	• **Focal/patchy Consolidation of lung tissue (lower lobes)** • Organizing pneumonia pattern = Alveoli full of mixture of inflammatory cells +/- fibrotic changes (**NO pus**)	Bronchial lavage: • 40% eosinophils (normal <3%) • Lots of eosinophils but NO bugs
Rx	• Remove allergen • O2 if needed • Systemic steroids		• **Rx**: Nintedanib (TKI) or pirfenidone (anti-TGFβ) • **Avoid**: Warfarin, endothelin antag, prednisone	**Systemic Steroids**	**Systemic Steroids**

PULMONARY HYPERTENSION, SARCOIDOSIS and OSA

Define

PULMONARY HTN

Increased resistance and pressure of blood in pulmonary arteries → R heart strain

SARCOIDOSIS

Granulomatous inflammatory condition consisting of granulomas (nodules of inflammation containing macrophages)

OSA

Collapse of pharyngeal airway during sleep causing periodic apnoea episodes

Cause / risk factors

PULMONARY HTN

Group 1	Primary PHTN, idiopathic or CT disease (SLE)
Group 2	L) heart failure (2° to MI, congenital HD or systemic HTN)
Group 3	Chronic lung disease (COPD)
Group 4	Pulm. Vascular disease (PE)
Group 5	Other – sarcoidosis, glycogen storage disease, haematological disorders

SARCOIDOSIS

Risk factors:
- Young adults + 60s
- Women
- Black

DDx
- TB
- Lymphoma
- HIV, toxoplasmosis, histoplasmosis
- Hypersensitivity pneumonitis

OSA

Risk factors:
- Middle age
- Male
- Obesity
- Alcohol
- Smoking
- T21, NMD, Pierre-Robin sequence (smaller lower jaw)

Comp.

PULMONARY HTN
- Empyema (infected pleural effusion)
- Sepsis
- Death

SARCOIDOSIS
- Asymptomatic (50%) to life-threatening
- *Death usu. due to infiltration to heart (arrythmias) or CNS*

OSA
- HTN → HF, AF
- Obesity, depression
- MI/ Stroke

SX

PULMONARY HTN
- SOB (main)
- Syncope
- Tachycardia
- Raised JVP
- Hepatomegaly
- Peripheral oedema

SARCOIDOSIS
- Progressive SOB -*PHTN and pulmonary fibrosis, Bilateral hilar LN, pulm. nodules*
- Systemic = fever, fatigue, UWL

Extra-pulm manifestations
- **EYES=** Anterior uveitis, optic neuritis, conjunctivitis
- **SKIN** = Erythema nodosum (painful shins), lupus pernio
- **HEART** = BBB, Heart block, infiltration
- **KIDNEYS** = stones, nephrocalcinosis, interstitial nephritis
- **CNS** = encephalopathy, DI, nodules
- **PNS** = facial nerve palsy, mononeuritis multiplex
- **Bones** = arthralgia, arthritis, myopathy
- **Lofgren's syndrome** = erythema nodosum + bilateral hilar LN, polyarthralgia

OSA

STOP BANG Criteria:

1) Clinical criteria ("A" criteria" ≥ 1 Sx:
- Snoring
- Laboured or apnoea episodes during sleep
- Daytime somnolence
- Hyperactive
- Behavioural issues

2) Sleep study (PSG) "B" criteria
- ≥1 obstructive apnoea
- ≥ 25% of total sleep time with hypercapnia
- ≥1 or more snoring, flattened nasal pressure waveform, thoracoabdominal motion

Ix

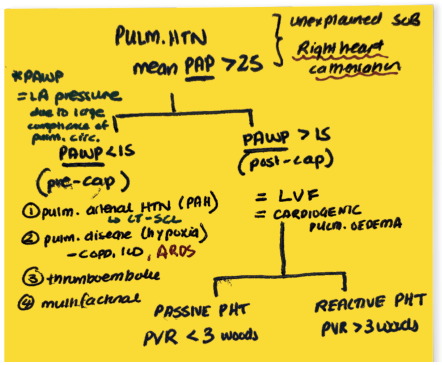
PULMONARY HTN

ECG changes:
- RVH
  (large R waves in V1-3 and S waves in V4-6)
- Right axis deviation
- RBBB

CXR changes:
- **Dilated pulmonary arteries**
- **RVH – cardiomegaly**

Special Ix
- Raised NT-proBNP = R) ventricular failure
- TTE – estimated pulmonary artery pressure

Right heart catherisation
- gold standard for PHTN diagnosis
- invasive but safe measure via R) IJV
- *Indications* = UNEXPLAINED SOB, abnormal non-invasive tests

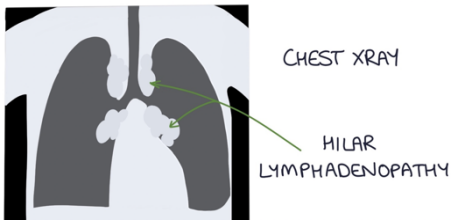


*PAP = pulmonary artery pressure
**PVR = Pulmonary vascular resistance

SARCOIDOSIS

Bloods
- FBC
- CMP – hypercalcemia
- ACE – elevated
- CRP – raised
- Immunoglobulins – raised
- Serum soluble IL-2 receptor – raised

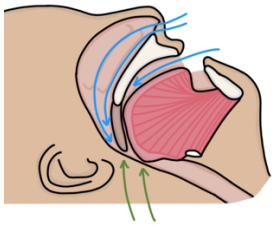
Imaging
- CXR = bilateral hilar LN
- HRCT thorax = hilar LN + pulm. Nodules
- MRI = CNS involvement
- PET = active inflamed areas



HISTOLOGY
- Gold standard to diagnose via biopsy (EBUS) on mediastinal LN

ORGAN INVOLVEMENT Tests:
- **EUC** = for kidney involvement
- **UA** or **URINE ACR** = proteinuria --→ nephritis
- **LFTs** = liver involvement
- **Ophthalmology** review = eye involvement
- **ECG and TTE** = heart involvement
- **USS abdomen** = liver and kidney involvement

OSA



General
- Vitals (BP)
- BMI + neck circumference

Bloods (anaemia screen)
- *FBC – anaemia*
- *Fe, B12, folate*
- *Vit D*
- *TFT*

Epsworth Sleepiness Scale

Assess symptoms sleepiness associated with OSA
- 0-7 = normal
- ≥ 8 = specialist review
- ≥16 = XS sleepy anytime

POLYSOMNOGRAPHY
- GOLD standard to confirm OSA
- Indicated if:
  - STOP-BANG SCORE ≥ 4
  - OSA-50 score ≥ 5

Mx

PULMONARY HTN

Poor prognosis – 30-40% 5 year survival from dx (primary PHTN w/ CT disease is the worst)

For primary PHTN → vasodilate + anti-proliferation
1) IV prostanoids (e.g. epoprostenol)
2) Endothelin receptor antagonists (e.g. macitentan, bosentan)
3) PDE5i (e.g. sildanefil)

For secondary PHTN:
1) Treat cause (E.g. PE, SLE)

SUPPORTIVE TREATMENT for complications:
1) respiratory failure
2) arrythmias
3) heart failure

SARCOIDOSIS

Prognosis = spontaneously resolves within 6 months in 60% of patients
- *Can lead to PHTN and pulmonary fibrosis*
- *Death due to infiltration to heart (arrythmias) or CNS*

1st line = No treatment = 1st line
- For those with no or mild symptoms

2nd line: medications
- Oral steroids for 6-24 months
- + Bisphosphonates to protect against OP
- 2nd line = MTX or azathioprine

3rd line = surgery
- Lung transplant (only for severe pulmonary disease)

OSA

CONSERVATIVE MX
- Lose weight (>10%) – PA, diet
- Stop drinking alcohol
- Stop smoking

INTERVENTIONS
- ENT referral or specialist sleep clinic
- CPAP machine
- Surgery – uvulopalatopharyngoplasty or remove adenoids, tonsils (?may worsen OSA)

MEDICO-LEGAL
- Truck drivers of heavy goods
- Crane operators
- Pilots / surgeons