PULMONARY PHYSIOLOGY

BASIC EQUATIONS	**Partial Pressure O_2**	**= $F_iO_2 \times P_b$**
	P_AO_2	**= $[(P_b - P_{H2O}) \times F_iO_2] - [P_aCO_2/0.8]$** ➡ *Respiratory quotient* **(0.8)** = *CO_2 production/minute & O_2 consumption*
	$D_{A\text{-}a}O_2$	**= $P_AO_2 - P_aO_2$**

TERMINOLOGY	**P_AO_2**	• *Partial pressure of O_2 in the alveoli*
	F_iO_2	• *Fraction of inspired oxygen =* **20.9%** *at sea level → decrease with higher altitude*
	P_b	• *Atmospheric pressure =* **760** *mmHg at sea level*
	P_aO_2	• *Partial pressure of O_2 in the arterial blood* (commonly called PO_2) **= 75–100** *mmHg*
	P_aCO_2	• *Partial pressure of CO_2 in the arterial blood* (commonly called PCO_2) **= 38–42** *mmHg*
	S_aO_2	• *O_2 saturation of hemoglobin in the arterial blood →* **95–100%**
	S_vO_2	• *O_2 saturation of mixed venous blood* (in the pulmonary artery) **→ 65–75%**
	$S_{cv}O_2$	• *O_2 saturation of central venous blood* (from the superior vena cava) **→ >70%**
	$D_{A\text{-}a}O_2$	• *The alveolar–arterial gradient* (A-a gradient/ A-a O_2 /$D_{A\text{-}a}O_2$) *= the difference between O_2 partial pressure in the alveoli* (A) *& that in arterial blood* (a) **= 5–10** *mmHg*

6 CAUSES OF HYPOXEMIA

VENTILATION/PERFUSION (V/Q) MISMATCH

PATHOLOGY	➡ *The main cause of hypoxemia in chronic lung diseases due to –* **a.** *Inadequately perfused airspaces/saddle PE or* **b.** *Perfused areas that are inadequately ventilated*
O2 RESPONSE	➡ *Responds well to* **100%** *O_2*
CAUSES	**1.** *Asthma* **2.** *COPD* **3.** *Alveolar disease* (pneumonia) **4.** *Interstitial disease* **5.** *Pulmonary vascular disease* (pulmonary hypertension – pulmonary embolism)

RIGHT-TO-LEFT (R-TO-L) SHUNTING

PATHOLOGY	➡ *Due to perfusion of nonventilated alveoli (↑ A-a gradient)*
O2 RESPONSE	➡ *Does not respond well to* **100%** *O_2 but responds better to positive end-expiratory pressure* (PEEP)*– PEEP can worsen R-to-L intracardiac shunt by ↑ shunt fraction due to ↑ right-sided pressures*
CAUSES	**1.** *ARDS* **2.** *Alveolar collapse* **3.** *Intracardiac shunt* **4.** *Intraalveolar filling* (pneumonia – pulmonary edema) **5.** *Vascular shunt*

DECREASED ALVEOLAR VENTILATION (HYPOVENTILATION)

PATHOLOGY	➡ *Seen with –* **1.** *↓ tidal volumes* **2.** *↓ respiratory rates* **3.** *Apnea* **4.** *High P_aCO_2 with hypoxemia*
CAUSES	• *Drug overdose* • *Neuromuscular diseases* • *CNS disorders*
A-a GRADIENT	➡ *Normal*

HIGH ALTITUDE

PATHOLOGY	➡ *↓ atmospheric pressure → reduced P_AO_2 (so responds to supplement O_2)*
A-a GRADIENT	➡ *Normal unless lung disease is present*

DECREASED DIFFUSION

PATHOLOGY

➡ *The key is membrane thickening or loss of the alveolar-capillary interface → ↓ O_2 diffusion → little effect on hypoxemia at rest (as it has to be significant loss of the interface) but major role in exercise-induced desaturations*

➡ *DLCO test → measures how efficient inspired CO diffuses from the alveoli to RBC hemoglobin (surrogate marker for CO_2 & oxygen diffusion) → if ≤30% of predicted = Hypoxemia at rest*

O2 RESPONSE

➡ *With Low DLCO → symptoms improve with supplemental O_2*

CAUSES

LOW DLCO	HIGH DLCO
1. *ILDs*	1. *Alveolar hemorrhage*
2. *Emphysema*	2. *Polycythemia*
3. *Pulmonary vascular disease*	3. *Sometimes → acute asthma attack ↑ Cardiac output (limited diffusion time) → ↓ O_2 transfer*
4. *Marked anemia*	

LOW MIXED VENOUS O_2

PATHOLOGY

➡ *Caused by –*

1. *Low cardiac output or*
2. *Increased tissue extraction of oxygen (as in sepsis)*

➡ *Lower P_vO_2 → hypoxic vasoconstriction → increasing R-to-L baseline anatomical shunting (5%) → reduces P_aO_2 during resting conditions & exaggerate all other causes of low P_aO_2*

OXYHEMOGLOBIN DISSOCIATION CURVE

PHYSIOLOGY

➡ *Oxygen delivery to the tissues based on both –*

a) *Amount of O_2 transported to the tissues*

b) *Amount of transported O_2 that is taken up & subsequently utilized by the mitochondria &/or cells*

➡ *3 factors to assess in critically ill patient requires better oxygen delivery*

1. *Cardiac output (heart rate × stroke volume)*
2. *Hgb*
3. S_aO_2

DO_2 = cardiac output × (1.34 × Hgb × S_aO_2)

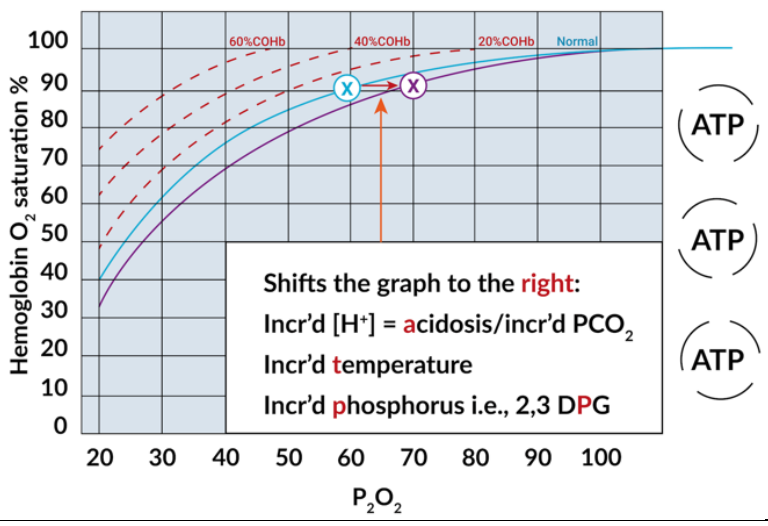
OXYHEMOGLOBIN DISSOCIATION CURVE

(Represent % of O_2 saturation of hemoglobin (S_aO_2) to certain P_aO_2)

➡ *Depends on 3 factors (ATP)*

1. *Acidosis (pH)*
2. *Temperature*
3. *Phosphorus (2,3-DPG) – high or low levels of s. phosphorus → ↑ or ↓ 2,3-DPG level*

SO_2 (%)	P_aO_2 (mmHg)	SO_2 (%)	P_aO_2 (mmHg)
80	44	91	62
81	45	92	65
82	46	93	69
83	47	94	73
85	49	95	79
86	50	96	86
87	52	97	96
88	55	98	112
89	57	99	145
90	60		



PRE-OP ASSESSMENT USING PFT

(PFT IS NOT ROUTINELY INDICATED IN PREOPERATIVE EVALUATION)

INDICATIONS	1. *If the surgical procedure is close to the diaphragm* (gallbladder) 2. *If the patient has moderate or severe lung disease* (FEV_1 < 1 L or elevated P_aCO_2 indicates high risk for postoperative pulmonary complications) 3. *If presurgical evaluation for lung cancer or lung resection required* ➡ *High risk of postoperative morbidity is suggested by predicted FEV_1 ≤ 0.8 L after surgery* 4. *If preoperative FEV_1 is ≤ 1.6 L → estimate the postoperative FEV_1 by –* a) *Obtaining quantitative ventilation & perfusion lung scan* b) *Multiply the percent perfusion* (or ventilation) *to the lobe to be removed by the FEV_1 in order to obtain the estimated postoperative FEV_1*

6-MINUTE WALK TEST

INDICATIONS	➡ *Provide reliable assessment of exercise capacity in lung disease patient or lung transplant patient* ➡ *Serial 6-minute walk test is used to assess treatment response in chronic pulmonary disorder* (especially PAH)
TECHNIQUE	➡ *Patient walks at own pace for 6 minutes & total walked distance is measured*

PULSE OXIMTERY

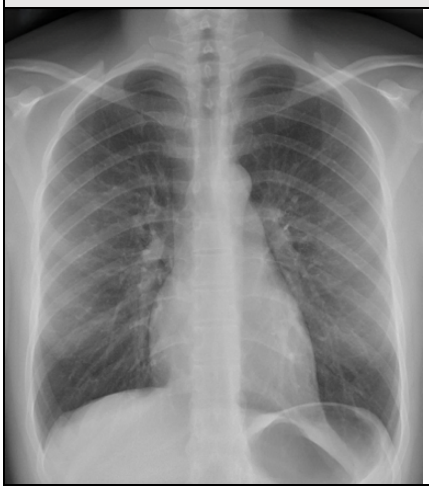
TECHNIQUE	➡ *Calculate the difference of infrared light absorption by oxygenated & deoxygenated blood*	
RESULT	**Normal**	➡ 95–100%
	Hypoxemia	➡ <90%
CAUSES OF ERROR READING (Use ABG instead)	• *Carboxyhemoglobin* • *Some topical anesthetics* • *Nail polish*	• *Methemoglobin* • *Cool extremities* • *Motion artifact*
		• *Methylene blue* • *Poor circulation* • *Race variability*

FRACTIONAL EXHALED NITRIC OXIDE

TECHNIQUE	➡ *Measurement of nitric oxide fraction in exhaled breath* (FeNO)	
INDICATIONS	1. *Noninvasive test to quantify eosinophilic airway inflammation* 2. *Predict glucocorticoid response & adherence to anti-inflammatory therapy (especially asthma as > 50% of asthma patients have eosinophilic [type2] airway inflammation)* 3. *Used in uncertain diagnosis of asthma after standard workup*	
RESULT	**FeNO > 50 ppb**	➡ *Indicate glucocorticoid-sensitive airway inflammation*
	FeNO < 25 ppb	➡ *Indicate less likely eosinophilic inflammation & glucocorticoid response*

IMAGING

CHEST RADIOGRAPHY (CXR)



TYPES	1. *Two views* (posteroanterior [PA] & lateral) 2. *One portable view* (anteroposterior [AP])
ADVANTAGES	• *Easy availability/portability* • *Low radiation*
LIMITATIONS	○ *Need for deep breath/breath hold* ○ *Imaging resolution*

Normal PA CXR

Source: Mikael Häggström MD

EXERCISE-INDUCED BRONCHOCONSTRICTION (EIB)

PATHOLOGY	➡ *Acute onset of bronchoconstriction during or more common within minutes after exercise*	
RISK FACTORS	➡ *Environmental factors –* 1. *Exercise in cold dry air* 2. *Trichloramines exposure in swimming pool* 3. *Airborne pollution*	
PRESENTATION	➡ *Chest tightness* ➡ *Dyspnea* ➡ *Wheezing*	
DDx	1. *Vocal cord dysfunction* 2. *Cardiac disorders*	
DIAGNOSIS	**CONFIRMATION**	➡ *By ↓ FEV_1 of ≥10% after graded exercise on treadmill /stationary bicycle*
	IF FALSE NEGATIVE	➡ *EIB that induced by cold air only can be false negative with exercise treadmill test so use –* • *Bronchoprovocation using methacholine - cold air – eucapnic voluntary hyperventilation to confirm the diagnosis of cold air–/EIB*
	EXCLUDE OTHER CAUSES	➡ *By the shape the flow-volume loop*
TREATMENT	**PHARMACOLOGIC**	1. *Inhaled glucocorticoid +SABA (not SABA alone) prn or before exercise or* 2. *Low dose glucocorticoid + Formoterol prn or before exercise* 3. *Leukotriene inhibitors (but not rapid relief as SABA)*
	NONPHARMACOLOGIC	• *Preexercise warm-up* • *Warming the cold air*

DIAGNOSIS

HISTORY & POSTBRONCHODILATOR IMPROVEMENT	➡ *Highly suggest the diagnosis (≥12% FEV_1 or ≥200 mL FVC improvement)*
PFTs	➡ *Measure FEV_1 – FVC & FEV_1/FVC – both before & after use of the bronchodilator (in all patients)*
FRACTIONAL EXHALED NITRIC OXIDE (FeNO) CONCENTRATION (FOR ≥ 5 YEAR AGE PATIENT)	1. *Used in the diagnosis of eosinophilic-induced asthma → levels >50 parts per billion (ppb) = considered elevated (in nonsmoker)* 2. *ATS is conditionally recommended if asthma therapy is considered* 3. *Used in uncertain asthma diagnosis*
BRONCHOPROVOCATION TESTS • **Methacholine** • **Histamine** • **Thermal (cold air) challenges**	➡ *Indicated with normal spirometry + ≥1 of asthma presentation –* 1. *Chronic cough* 2. *Intermittent symptoms of cough/wheeze &/or chest tightness* 3. *Exertional dyspnea without other cause*

TREATMENT

INITIAL TREATMENT

PROTOCOL	➡ *Assess severity (use FEV_1 – but not the peak flow) & determine treatment level based on that* ➡ *Start 1 or 2 levels above the presumed severity level → gradually stepped down* ➡ *If your patient's risks include >1 category → classify according to the most severe category*

CONTROL PHASE

THE KEY IS THE ASTHMA CONTROL & THE RESPONSE TO TREATMENT (NOT THE ASTHMA'S SEVERITY)

PROTOCOL

Based On 2021 Global Initiative For Asthma (Gina) Guidelines Of Control Of Asthma Symptoms

➡ *4 questions –*
1. *The presence of daytime symptoms >2×/week*
2. *Any nighttime awakening due to asthma*
3. *Use of SABA reliever >2×/week*
4. *Any activity limitation due to asthma*

Well-Controlled Asthma	➡ *None of these factors*
Partly Controlled Asthma	➡ *1–2 of these factors*
Uncontrolled Asthma	➡ *3–4 of these factors*

➡ *Reevaluate again in 1–2 months to assess the level of control using either FEV_1 or PEF*
- *If patient is well controlled for 3 months → assess the regimen required to maintain control & assign the category of severity that correlate with that step level of treatment (at that point – you can define the disease severity based on the treatment needed to control it)*
- *The control phase is dynamic (every 3 month) → step up if not controlled & step down if controlled*
- *The goal is to maintain control with fewest medications (ICS is the cornerstone of asthma in all steps)*

RESTRICTIVE LUNG DISEASE

DIFFUSE PARENCHYMAL LUNG DISEASE (DPLDs) (INTERSTITIAL LUNG DISEASES [ILDs])

PATHOLOGY

➡ *Primary or secondary collagen deposition in the interstitium (potential space between the capillaries & the alveoli) → creates fibrosis & alter the architecture of the alveoli and surrounding airways → increase elastic recoil (this recoil is ↓ in emphysema) → increase lung stiffness & decrease compliance*

CATEGORIES

KNOWN CAUSES

Drug-Induced	• *Amiodarone* • *Methotrexate* • *Nitrofurantoin* • *Chemotherapy*
Smoking-Related	○ *Respiratory bronchiolitis-associated ILD (RB-ILD)* ○ *Desquamative interstitial pneumonia (DIP)* ○ *Smoking-related pulmonary fibrosis* ○ *Combined emphysema with pulmonary fibrosis* ○ *Pulmonary Langerhans cell histiocytosis*
Radiation **Weeks To Months After Radiation**	1. *Radiation-induced lung injury* 2. *Radiation pneumonitis/radiation fibrosis*
Chronic Aspiration	➡ *Subclinical and may flare up other DPLDs* ➡ *Mainly in lower lobes (especially the right base)*
Pneumoconioses	**ORGANIC**: ➡ *Byssinosis* **INORGANIC**: • *Asbestosis* • *Berylliosis* • *Silicosis* • *Coal workers' pneumoconiosis*
Connective Tissue Diseases **Rheum Arthritis** **Systemic sclerosis** **Dermatomyositis** **Polymyositis** **MCTD** **SLE** **Sjögren syndrome** **Vasculitides**	1. *Nonspecific interstitial pneumonia (NSIP)* 2. *Organizing pneumonia* 3. *Usual interstitial pneumonia (UIP)* 4. *Lymphocytic interstitial pneumonia (LIP)*
Hypersensitivity Pneumonitis (HP)	• *Acute* • *Subacute* • *Chronic*
Idiopathic Pulmonary Hemosiderosis (IPH)	➡ *Abnormal accumulation of iron*
Eosinophilic ILDs	1. *Acute & chronic eosinophilic pneumonia* 2. *ABPA* 3. *EGPA*

UNKNOWN CAUSES

- *Idiopathic interstitial pneumonias (IIP)*
- *Idiopathic nonspecific interstitial pneumonia (INIP)*
- *Acute interstitial pneumonia (AIP)*
- *Idiopathic pulmonary fibrosis (IPF)*
- *Cryptogenic organizing pneumonia (COP)*
- *Sarcoidosis*

RARE DISORDERS WITH WELL-DEFINED FEATURES

1. *Lymphangioleiomyomatosis*
2. *Chronic eosinophilic pneumonia*
3. *Pulmonary alveolar proteinosis*

PRESENTATION

- *Subacute to exertional dyspnea*
- *Nonproductive cough*

EXAMINATION

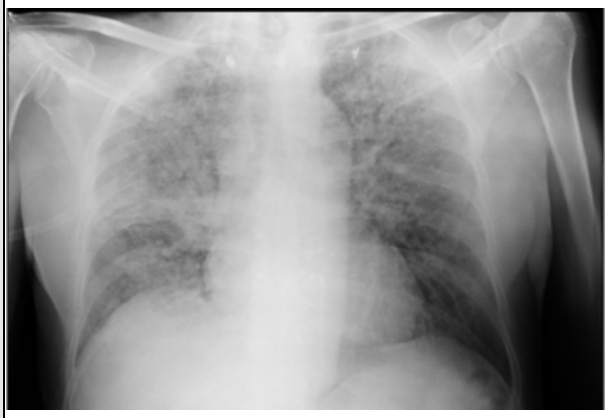
➡ *Normal findings*
➡ *Inspiratory Velcro crackles*
➡ *Inspiratory squawk (fibrosis)*
➡ *Normal resting oximeter but desaturation >4% on ambulation = diffusion limitation (the hallmark of DPLD)*

DIAGNOSIS

IMAGING

CXR

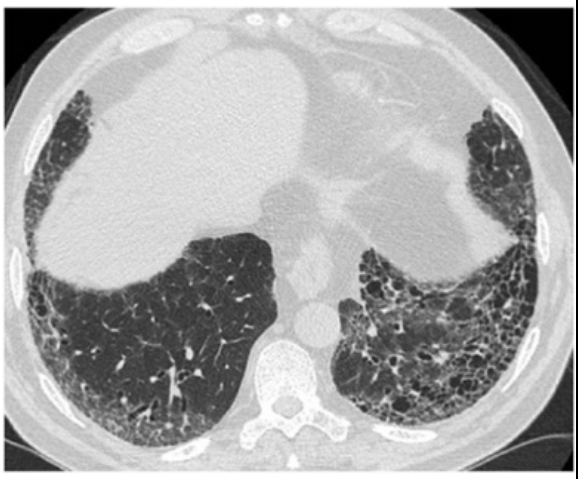
➡ *Bilateral diffuse reticular & reticulonodular pattern (common) is 1st suggestive of the disease (correlates poorly with the severity of disease)*



Bilateral ILD
Source: Yolanda Del Castillo

HRCT

➡ *Diagnostic for ILD*



Honeycombing in ILD
Source: Cécile Daccord

CXR FINDINGS IN ILDs

Asbestosis	*Lower lung field predominance of infiltrates* *+/− pleural calcifications & plaques*
Silicosis	*Hilar eggshell calcifications − nodular lesions*
Sarcoidosis	*Bilateral symmetrical hilar & paratracheal lymphadenopathy*
Lymphangioleiomyomatosis (LAM)	*ILD with Pneumothorax in Premenopausal woman* *Chylous effusions* *Characteristic diffuse thin-walled cysts on CT*

PFTs

1st TEST	• **DLCO** *(1st abnormal test)* → *use this parameter to follow patients are on potentially lung-toxic drugs (amiodarone − chemotherapy)*
PATTERN	• *Classic restrictive pattern = normal airway flow rates but decreased lung volumes*

SEROLOGY

➡ *ATS recommends excluding CTD-associated DLPD in new cases with unknown cause −*

• *CRP* • *ESR* • *ANA* • *RA* • *Anti-CCP* • *Myositis panel*

BIOPSY

BRONCHOSCOPY WITH TRANSBRONCHIAL BIOPSY

USAGE	1. *If uncertain diagnosis after PFT & HRCT* 2. *Exclude (along bronchoalveolar lavage) →* a) *Infections* b) *Lymphangitic spread of carcinoma* c) *Cryptogenic organizing pneumonia (COP)* d) *Sarcoidosis* e) *Airway centered DPLD like respiratory bronchiolitis-DPLD*
LIMITATION	➡ *Small tissue specimens acquired if used alone*

VIDEO-ASSISTED THORACOSCOPIC SURGICAL (VATS) BIOPSY

TECHNIQUE	➡ *Minimal invasive thoracoscope through the chest wall & open lung biopsy*
USAGE	➡ *The best yield to diagnose interstitial pneumonitis*

NONSPECIFIC INTERSTITIAL PNEUMONIA (NSIP)

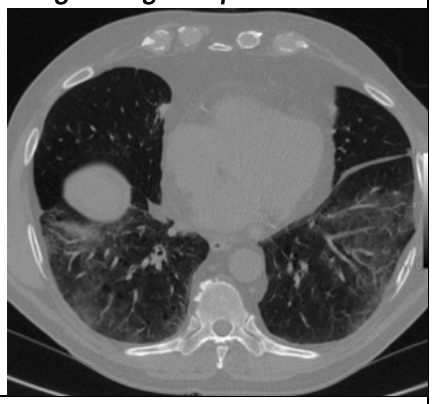
PATHOLOGY
- ➡ *Variant CTD-associated DLPD but idiopathic NSIP has distinctive clinical character*

FORMS
- **CELLULAR**
  - *Inflamed cells between alveoli*
- **FIBROTIC**
  - *More fibrosis progression in the lungs (worse prognosis)*

PRESENTATION
- ➡ *Affect younger patients than IPF but IPF has worse prognosis*

DIAGNOSIS

CHEST HRCT
- *Bilateral lower lobe reticular changes with scattered ground glass opacities + progression to fibrosis & honeycombing* (rare)



NSIP
Source: Mluisamtz11

BIOPSY
- *Confirm the diagnosis and exclude other causes*

TREATMENT
- **IMMUNOSUPPRESSANT**
  - ➡ *With better response in cellular form*
- **LUNG TRANSPLANTATION**
  - ➡ *In selected patients* (as IPF)

PROGNOSIS
- ➡ *5-year mortality of idiopathic NSIP is 15–25% (better than IPF)*

ACUTE INTERSTITIAL PNEUMONIA (AIP)

PATHOLOGY
- ➡ *Diffuse alveolar damage* (clinically/pathological/radiologic indistinguishable from ARDS)
- ➡ *ARDS without risk factors → suspect AIP*

PRESENTATION
- ➡ *Acute respiratory failure occur rapidly over days-weeks with bilateral alveolar opacities on chest HRCT*

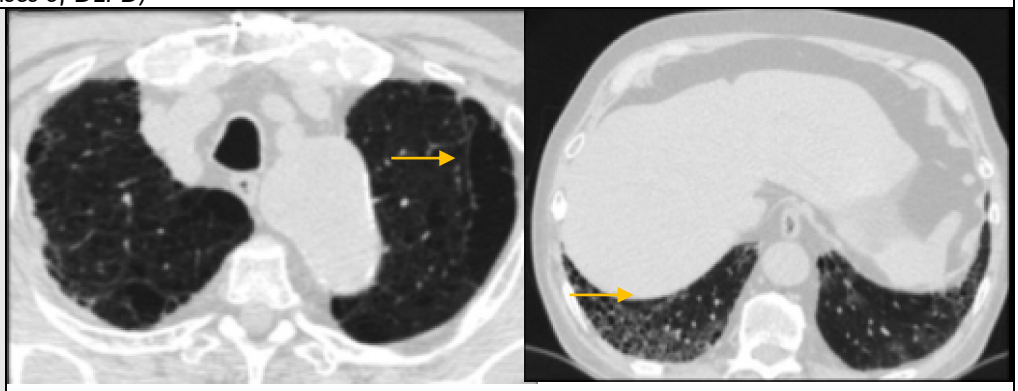
TREATMENT
- ➡ *Ventilatory support (as ARDS) + High-dose glucocorticoids* (often used but not strong evidence)

COMBINED PULMONARY FIBROSIS & EMPHYSEMA (CPFE)

PATHOLOGY
- ➡ *Fibrotic lung changes* (as in IPF) *+ Emphysematous changes in the same patient* (so part of known & unknown causes of DLPD)

DIAGNOSIS

CHEST HRCT



CPEF
Upper Zone Emphysema + Lower Zone Fibrosis
Source: Olívia Meira Dias

PFT
- ➡ ↓ *DLCO*

TREATMENT
- ➡ *Lung transplant is the only curative treatment*

ANTICOAGULATION

INDICATIONS	**VTE TREATMENT**	➡ *CHEST recommends DOACs over VAKs* ➡ *Avoid UFH due to unpredictable bioavailability over LMWH* ➡ *UFH indicated initially in case of unstable patient that will require emergent surgery or thrombolytics (due to short half-life)*
	VTE PROPHYLAXIS	➡ *Discussed below*
USAGE	**OVERLAP WITH UFH**	• *Warfarin* • *Dabigatran* • *Edoxaban*
	MONOTHERAPY	• *Apixaban* • *Rivaroxaban*
RELATIVE CONTRAINDICATIONS	**UNCORRECTED BLEEDING DISORDER**	1. *Thrombocytopenia* 2. *Hemophilias* 3. *Liver failure* 4. *Renal failure*
	POTENTIAL BLEEDING LESIONS	1. *Active peptic ulcer* 2. *Esophageal varices* 3. *Ruptured aneurysm* 4. *Recent trauma/surgery to the head/orbit/spine* 5. *Recent stroke* 6. *Intracranial or intraspinal bleed*
	BLOOD PRESSURE	➡ *Uncontrolled severe hypertension (systolic BP >200 mmHg &/or diastolic BP >140 mmHg)*
	MEDICATIONS	➡ *NSAIDs → increases risk of GI bleeding (stop NSAIDs if possible)*
	FALLS	➡ *Repeated falls or unstable gait*

HEPARIN

Unfractionated Heparin (UFH)	**Low Molecular Weight Heparin (LMWH)** **(Dalteparin – Enoxaparin)**
MECHANISM OF ACTION	
➡ *Binds with antithrombin (AT) to make it 1,000–4,000× more effective in inactivating thrombin & Factor 10a* ➡ *UFH dosage is based on weight-based nomogram → adequate anticoagulation* ➡ *Check aPTT levels every 6–8 hours after dosage change to allow time to achieve steady state → adjust the dose of IV UFH to keep the aPTT at least 1.5× control for 7–10 days – if greater → ↑ incidence of bleeding*	➡ *Depolymerization of heparin → molecular fragments with 30–50% the weight and more anticoagulant activity* ➡ *Solely inactivates Factor 10a → so PTT is not sensitive to LMWH anticoagulant effect* ➡ *Renal cleared (dose adjusted in renal dysfunction or switched to another agent as UFH)*
Titratable	**Non titratable**
Short Half-life	**Long Half-life**
Less predictable pharmacodynamic profile	**Immediate therapeutic Dosing**
INDICATIONS	
➡ *Preferred in treating VTE –* 1. *UFH is no longer the drug of choice for DVT → still used for the initial treatment of PE/DVT in case with unstable hemodynamics & might require rescue thrombolysis (as in hypotensive patients)* 2. *↓ creatinine clearance < 30 mL/minute* 3. *Severe obesity* 4. *Life-threatening PE – because subcutaneous LMWH → requires normal blood pressure & tissue perfusion for optimal delivery → so not reliable in unstable patients* 5. *In setting of rapid reversal need in planned procedures*	➡ *Preferred in treating VTE –* 1. *Use subcutaneous full-dose LMWH – whenever possible to treat PE (inpatients & outpatients) →* a) *Lower risk of major bleeding compared to UFH* b) *Preferred – esp. with hemodynamically stable patient → as LMWH reaches the therapeutic state faster* 2. *Pregnant women* 3. *Cancer patients* 4. *Patients with non–life-threatening PE* 5. *For extended period treatment because → the risk of osteoporosis is much lower than with UFH*
CONTRAINDICATION	
General Contraindications	*In Cr Cl <30 mL/minute → use UFH as not renal dependent*
DOSING & ROUTE	
80–100 U/kg then follow weight based monogram *IV (VTE treatment) & Subcutaneous (VTE Prophylaxis)*	*More predictable dosing* *Only subcutaneous (for treatment & prophylaxis)*

PLEURAL DISEASES

PLEURAL EFFUSION

TYPES

TRANSUDATIVE EFFUSION (HYDROTHORAX)	➡ *Due to systemic disease → affect accumulation & absorption of pleural fluid* 1. *LV heart failure (most common)* (*common to see isolated right-sided effusion*) 2. *After abdominal surgery* 3. *Hypoalbuminemia* 4. *Cirrhosis* 5. *Nephrotic syndrome*
EXUDATIVE EFFUSION	➡ *Due to local disease process –* 1. *Bacterial pneumonia (1st most common)* 2. *Cancer (2nd most common)* 3. *Pulmonary Embolism*

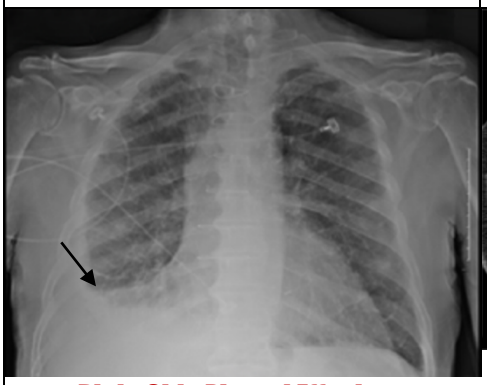
PRESENTATION

TYPICAL	• *Dyspnea*	• *Cough*	• *Pleuritic chest pain*
IN ELDERLY	▪ *Anemia*	▪ *Fatigue*	▪ *Failure to thrive*
EXAMINATION	○ *Dullness to percussion*	○ *Diminished or absent fremitus or breath sounds* (*if >200 ml*)	

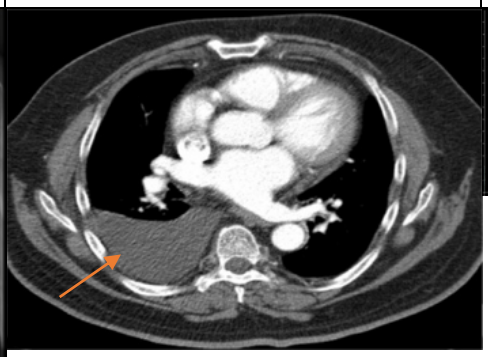
DIAGNOSIS

IMAGING

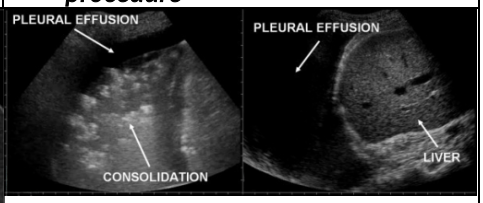
CXR (1st Test)	CT CHEST WITH CONTRAST	CHEST U/S
Detect effusion as 200 ml in PA view or 50 ml in lateral view	1. *Detect small pleural effusions* 2. *Detect coexisting lung pathology* 3. *Detect empyema* 4. *Detect PE* (*one of the causes*)	1. *Detect small effusions espicially in semirecumbinent patinet (ICU)* 2. *Determine the amount & if is loculated* 3. *Used in thorocentesis-guided procedure*



Right Side Pleural Effusion
Source: Mohsen Salih



Right Side Pleural Effusion
Source: Hellerhoff



Right Side Pleural Effusion
Source: Roberto Copetti

THOROCENTESIS

INDICATIONS	➡ *If unknown causes of pleural effusion* (*no indication for bilateral symmetric & duiretic responsive*) **&** *>1 cm thickness on U/S or lateral decubitus CXR*	
COMPLICATIONS	**TRANSIENT HYPOXIA**	• *Occurs in the first 12 hours* (*due to large amount removal of pleural fluid*) ***till the atelectatic alveoli reexpand and involve in gas exchange*** → *post-thoracentesis relief of dyspnea* (*that mostly due to diaphragm displacement and only 20% due to lung compression*)
	REEXPANSION PULMONARY EDEMA	• *If 1.4 – 2 L fluid removed*

PNEUMOTHORAX

CAUSES

PRIMARY SPONTANEOUS PNEUMOTHORAX (PSP)

➡ *Most commonly – tall slender & often smoking men 20–40 years of age → emphysematous blebs rupture → spontaneous pneumothorax*

RISK FACTORS

1. *Smoking*
2. *Tall stature*
3. *Family history*
4. *Thoracic endometriosis*

SECONDARY SPONTANEOUS PNEUMOTHORAX (SSP)

1. *COPD/Emphysema (most common cause)*
2. *Pneumocystis pneumonia in AIDS patients*
3. *Cystic fibrosis*
4. *Marfan syndrome*
5. *Langerhans cell histiocytosis (LCH) – smoking males*
6. *Lymphangioleiomyomatosis (LAM) – exclusively in premenopausal ♀*
7. *Barotrauma – reduced by using lung protective ventilation strategy with low TV of 6–8 mL/kg predicted body weight (PBW)*

PRESENTATION

➡ *Similar in PSP and SSP → sudden onset of dyspnea & sharp pleuritic chest pain (more severe in SSP due to underlying lung disease)*

EXAMINATION

SMALL PNEUMOTHORAX

➡ *No evident findings & could be limited to underling lung disease signs*

LARGE PNEUMOTHORAX

- *↓ Affected chest excursion*
- *Diminished breath sounds*
- *Hyperresonant percussion*
- *Enlarged affected hemithorax*
- *Absent tactile or vocal fremitus*
- *Subcutaneous emphysema (rarely)*

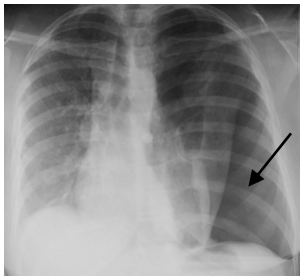
TENSION PNEUMOTHORAX

1. *Tracheal deviation (late sign but not always indicating pneumothorax)*
2. *Hemodynamic compromise (tachycardia – hypotension) = impending cardiopulmonary collapse*

DIAGNOSIS

CXR

1st test & often sufficient for diagnosis

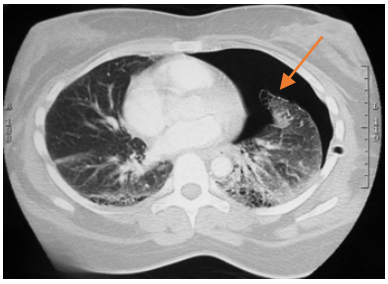


Left Side Pneumothorax

Source: Clinical Cases

CT CHEST

Useful if the diagnosis is not conclusive

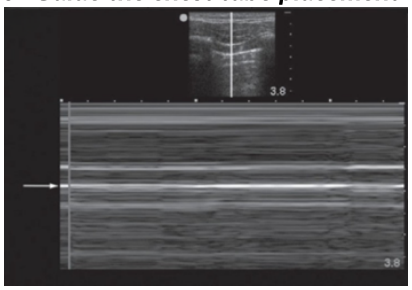


Left Side Pneumothorax

Source: Clinical Cases

CHEST U/S

1. *The most rapid & sensitive test for pneumothorax on ventilator*
2. *Guide the chest tube placement*



Stratosphere/Barcode Sign

M-mode image shows linear laminar pattern in the tissue superficial to the pleural line (arrow) & similar pleural pattern deep the pleural line = absent lung sliding that suggest pneumothorax

TREATMENT

SMALL PNEUMOTHORAX

➡ **< 2 cm in CXR + MINIMAL SYMPTOMS**

➡ *Needle aspiration + Observation admission + Supplement O_2 (increase rate of reabsorption vs. room air O_2)*

LARGE PNEUMOTHORAX

➡ **≥ 2 cm or SYMPTOMATIC**

➡ *Small bore (<14 Fr) thoracostomy tube insertion with connection to high-volume low-pressure suction system*

TENSION PNEUMOTORAX

HEMODYNAMIC INSTABILITY REGARDLESS THE SIZE

➡ *Emergent needle decompression in 2nd intercostal space then thoracostomy tube insertion*

INTRAVENOUS FLUID RESUSCITATION

	CRYSTALLOID	COLLOID
MOLECULE SIZE	*Small*	*Large*
COST	*Cheap*	*More cost*
EFFECT	*Immediate resuscitation*	*Rapid volume expansion*
SIDE EFFECTS	*Edema*	*Allergic reaction* *Kidney failure* *Blood clotting disorder*

INTRAVENOUS FLUID COMPARISON

FLUID	pH	OSMOLARITY (mOsm/L)	Na^+ (mEq/L)	Cl^- (mEq/L)	K^+ (mEq/L)
HUMAN PLASMA	7.4 ± 0.5	~ 290	140 ± 5	102 ± 5	4 ± 0.5
➢ CRYSTALLOIDS					
SALINE-BASED CRYSTALLOIDS					
Normal Saline (NS) 0.9%	5.5	308	154	154	0
½ Normal Saline (½ NS) 0.45%	5.5	155	77	77	0
¼ Normal Saline (¼ NS) 0.22%	-	86	39	39	0
Hypertonic Saline (HTS) 3%	5	1027	513	513	0
BALANCED CRYSTALLOIDS					
Lactate Ringer (LR)	6.5	273	130	109	4
Isolyte® S pH 7.4	7.4	295	141	98	5
Plasma-Lyte A	7.4	294	140	98	5
Normosol®-R	7.4	294	140	98	5
DEXTROSE-BASED CRYSTALLOIDS					
Dextrose 5% in Water (D5W)	4.3	253	0	0	0
Dextrose 10% in Water (D5W)	4.3	505	0	0	0
Dextrose 20% in Water (D5W)	4.3	1010	0	0	0
Dextrose 25% in Water (D5W)	4.5	1263	0	0	0
Dextrose 50% in Water (D5W)	4.3	2530	0	0	0
Dextrose 5% in NS (D5NS)	4.4	560	154	154	0
Dextrose 5% in ½ NS (D5 ½NS)	4.4	405	77	77	0
➢ COLLOIDS					
5% Albumin	7 ± 0.3	~ 290	145 ± 15	~ 150	0
25% Albumin	7 ± 0.3	~ 290	145 ± 15	~ 159	0
Hetastarch 6%	5.5	310	154	154	0
Pentastarch 6%	5	326	154	154	0
Dextran-40 (10% solution)	3.5-7	311	154	154	0
Dextran-70 (6% solution)	3-7	310	154	154	0
Haeaccel® (Gelatin)	7.4	293	145	145	5
Gelofusine® (Gelatin)	7.4	274	154	154	0

COMMON IV FLUID IN ED/ICU

COMPONENTS	NORMAL SALINE (NS)	RINGER LACTATE (RL)	PLASMALYTE
Na^+ (mEq/L)	154	130	140
Cl^- (mEq/L)	154	109	98
K^+ (mEq/L)	-	4	5
Ca^+ (mEq/L)	-	2.7	-
Mg^+ (mEq/L)	-	-	-

INVASIVE MECHANICAL VENTILATION

BREATH TYPES

TYPES	TRIGGER SOURCE	SUPPORT LEVEL
MANDATORY	**Ventilator** (time-triggered)	**Full**
ASSISTED	**Patient** (flow or pressure triggered)	**Full or Partial**
SPONTANEOUS	**Non-triggered**	**None**

MODES

VOLUME-CYCLED VENTILATION

CONTINUOUS MECHANICAL VENTILATION (CMV)

SETTING	➡ *Set rate & TV (volume-cycled) → does not allow spontaneous breathing*
INDICATIONS	1. *Heavily sedated* 2. *Under anesthesia* 3. *Paralyzed with muscle relaxants* 4. *In deep coma*
DISADVANTAGES	➡ *Patient-ventilator asynchrony*

ASSIST/CONTROL (AC)

SETTING	➡ *Set rate & TV (volume-cycled) → allow spontaneous breathing when the ventilator senses the patient's attempt to take extra breath → it kicks in with full ventilator-supported breath with the preselected tidal volume (Breathing Over The Vent)*
INDICATION	➡ *Better for critically ill patient that need full ventilator support*
DISADVANTAGES	➡ *If patients are anxious/hyperventilating or has obstructive lung disease → trigger additional full machine breaths = hyperventilated → auto-PEEP*

INTERMITTENT MANDATORY VENTILATION (IMV)

SETTING	➡ *As AC with set rate & TV but the patient can take additional breath based on patient-determined rate with variable volume + adding pressure support ventilation (PSV) to overcome endotracheal tube resistance (add 5–20 cm H_2O)*
TYPES	➡ *Synchronized IMV (SIMV) is variation of IMV with the ventilator breath are synchronized with the patient's inspiratory efforts*
ADVANTAGES	1. *Better patient-ventilator synchrony* 2. *Better preserved respiratory muscle function* 3. *Better control over the support level* 4. *Lower mean airway pressure* 5. *Lower auto-PEEP*

PRESSURE REGULATED VOLUME CONTROL (PRVC)

SETTING	➡ *Types of volume control ventilation with set of tidal volume & the applied airway pressure changes (based on patient's effort & lung mechanics) to achieve the target tidal volume (variable flow is comfortable for the patient)*

PRESSURE-CYCLED VENTILATION

PRESSURE CONTROL VENTILATION (PCV)

SETTING	1. *Desired pressure for the patient on each breath* 2. *Inspiratory/expiratory ratio* 3. *Breath rate* ➡ *On spontaneous breath attempt → patient will get the preselected ventilator volume at the designated pressure*
INDICATIONS	1. *Helpful in limiting airway pressures with high-end inspiratory plateau pressures that occur in volume-cycled modes that can cause barotrauma* 2. *Allow to use low TV & high PEEP strategy for ARDS (as the peak inspiratory pressure is lower on PCV than on AC for any given achieved TV)*
DISADVANTAGE	➡ *Variable TV based on airway resistance & respiratory compliance (no guaranteed TV) → must be titrated carefully & monitored at the bedside to determine the proper pressure settings & achieved TV*

SHOCK

PATHOLOGY

➡ *Circulatory systemic tissue hypoperfusion → insufficient O_2 delivery to tissues → organ dysfunction*

- **EARLY STAGE**
  - *Hypoperfusion causing compensatory anerobic metabolism (reversible stage)*
- **LATE STAGE**
  - *Failure of the compensatory stages → persistent tissue hypoperfusion → cellular dysfunction & death*

4 TYPES

1. *Hypovolemic (reduced circulatory volume)*
2. *Cardiogenic (pump failure)*
3. *Distributive (inappropriate vasodilation)*
4. *Obstructive (circulatory obstruction)*

ASSESSMENT

ASSESS ORGAN HYPOPERFUSION SIGNS

- *Altered mentation*
- *Cool extremities/mottled skin*
- *Capillary refill time >2 sec*
- *Absent bowel sounds*
- *↓ Urine output <0.5 ml/kg/h*

ASSESS HEMODYNAMICS

- *Hypotension (SBP <90 mmHg or <70 mmHg MAP) or >30 mm Hg drop in SBP from baseline*
- *Tachycardia >100 HR/min*
- *Tachycardia >100 HR/min*

ORGAN HYPOPERFUSION MARKERS

- *↑ Serum lactate >3 mEq/L*
- *↓ Serum pH*
- *↑ Serum creatinine*
- *↑ Serum Troponin*

INITIAL TESTINGS

- *Cultures (blood – urine – sputum – wound)*
- *CBC/CMP*
- *EKG/Echo/Lung & abdominal ultrasound/Vascular studies*
- *Radiologic studies*

MONITORING

GOAL

➡ *Mean arterial pressure (MAP) ≥65 mm Hg = sufficient for organ perfusion (study with higher 80-85 mmHg MAP did not show mortality benefit)*

CUFF PRESSURE

➡ *Usual noninvasive method*

ARTERIAL LINE

1. *Used if cuff pressures <90 mm Hg*
2. *With frequent needed measurements*
3. *If unreliable cuff readings in –*
   - *Morbid obesity*
   - *Vascular anomalies of the extremities*
   - *Anatomic pathology limiting cuff accuracy*

PULMONARY ARTERY CATHETER

➡ *Not indicated routinely (due to ↑ risk)*

➡ *Indicated in selected patients to measure cardiopulmonary pressure or CO in–*

1. *Pulmonary hypertension*
2. *Pericarditis*
3. *Tamponade*

TREATMENT

➡ *Depends on underlying cause*

HYPOVOLEMIC SHOCK

CAUSES

➡ *Due to loss of blood volume or dehydration –*

BLEEDING

- *GI bleeding*
- *Ruptured aortic aneurysm*
- *Trauma*
- *Ruptured ectopic pregnancy*

GI

1. *Diarrhea*
2. *Poor po intake*
3. *Vomiting*

RENAL

➡ *Polyuria in DKA/HHS/post-ATN*

SKIN

- *Excessive sweating*
- *Burns*

MEDICATIONS

- *Diuretics*
- *Laxatives*

PRESENTATION

Vital Signs

➡ *Hypotensive & tachycardic*

Skin

➡ *Cold or clammy*

Urine Output

➡ *Low urine output*