MEDICINE 1
MINI-OSCE COLLECTED SLIDES - PART 2
JU 2021

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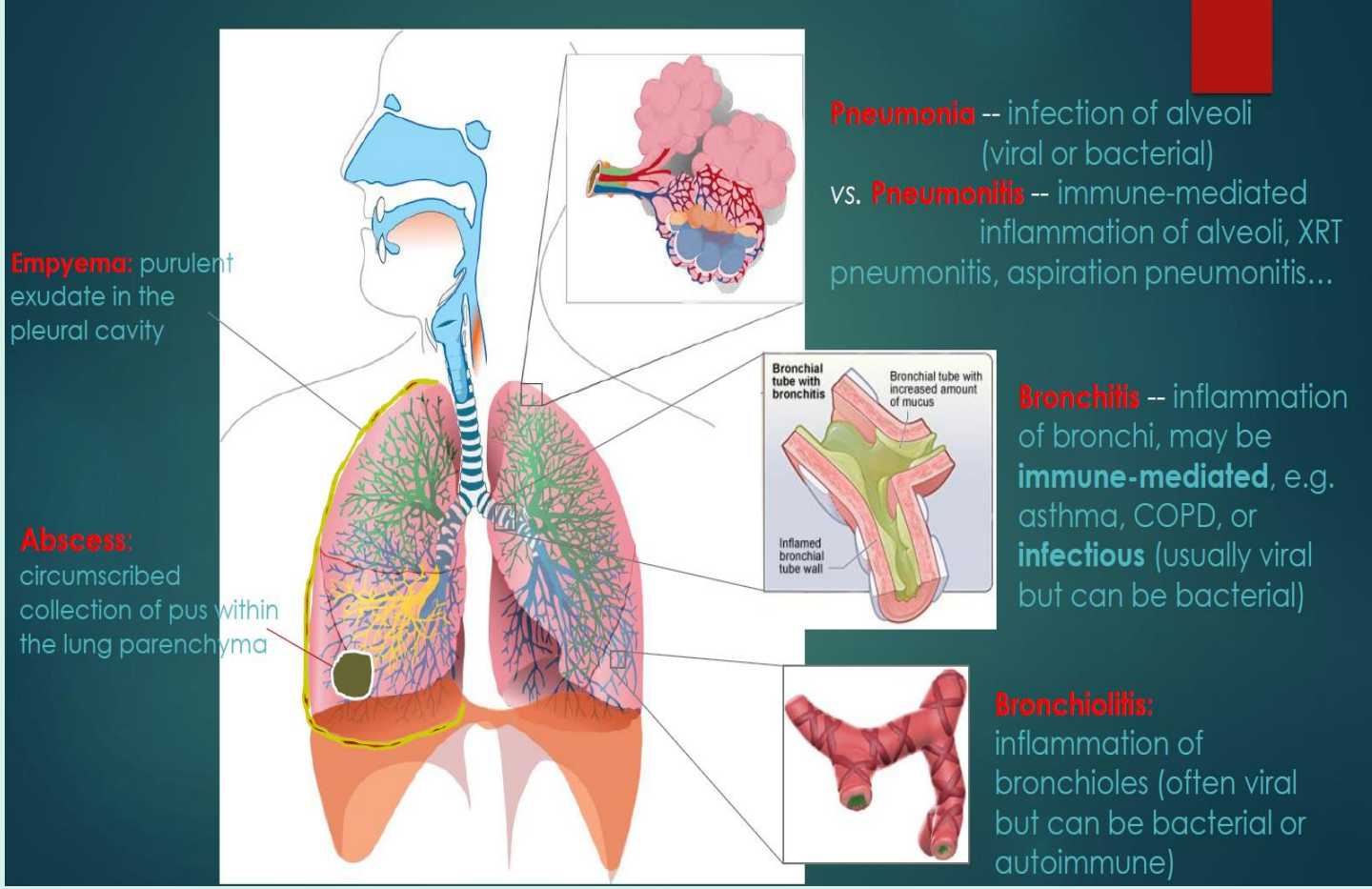
Contents

RESPIRATORY

PNEUMONIA

LOWER RESPIRATORY AND PLEURAL DISEASES



- RS infections occur on many different levels, so it's important to distinguish them since each type has a different treatment approach.
- Pneumonia is treated within 5-7 days.
- Empyema is infection of pleural surfaces, requires 6 weeks of treatment.
- Any severe infection can progress and cause Lung Abscess, we start treatment with Medications not drainage, if it fails then we remove it surgically.

An inflammatory process resulting from infection of the lung parenchyma by pathogenic microorganisms and usually associated with radiological evidence on CXR.

Patients present with Fever, Altered general well-being, along with Respiratory symptoms (Cough, Sputum production, Dyspnea, Pleuritic pain, and Hemoptysis).

In elderly and immunocompromised patients, the signs and symptoms of pulmonary infection may be muted and overshadowed by non-specific complaints.

On PE: Consolidation, Increased fremitus on affected side, Dullness, Bronchial breath sounds, Bronchophony and Crackles.

PNEUMONIA

CLASSIFICATION OF PNEUMONIA

The most common types of pneumonia are Community-acquired pneumonia and Nosocomial (Hospital-acquired and Ventilator associated) pneumonia.

Community-acquired pneumonia: the most common type, imaging shows consolidation.

Hospital acquired and Ventilator associated pneumonia: seen in patients hospitalized for at least 48 hours in a regular room or ICU on ventilator.

Pneumonia in immunocompromised patients: compromised to opportunistic organisms.

Atypical pneumonia: the types of bacteria that cause it tend to create less severe symptoms than those in typical pneumonia (C.pneumonia and M.pneumonia), it has atypical imaging showing bilateral opacities rather than consolidations.

PATHOLOGY AND PATHOGENESIS OF PNEUMONIA

PATHOLOGY AND PATHOGENESIS

- Aspiration of oropharyngeal or nasopharyngeal secretions is the main mechanism of contamination of lower airways by bacteria.
- The oropharynx of healthy individuals is colonized by diverse microorganisms that vary in their potential virulence.
- For example, Streptococcus pneumoniae, which contains multiple adhesions, binds to the receptor for platelet-activating factor on epithelial cells.
- Other pathogens enter the lower respiratory tract by inhalation.
- This interaction is enhanced by cigarette smoke, infection with respiratory viruses, and particulate air pollutants.
- Several mechanisms in the airways prevent adherence and colonization by potential bacterial pathogens:

✓Epithelial cells synthesize and secrete peptides, termed defensins and cathelicidins, that possess broad spectrum antimicrobial activity.

✓Pulmonary surfactant proteins A and C can inhibit bacterial binding to host cells and also promote phagocytosis of selected bacteria.

✓The presence of complement and immunoglobulins (particularly immunoglobulin A [IgA])

- Interactions between the virulence and quantity of aspirated or inhaled microorganisms and the individual's innate and adaptive immune responses determine whether pneumonia develops.
- Age , smoking , alcohol and comorbidity (resp. and non resp) all these factors increase risk of pneumonia.
- Geographic factors, seasonal timing, travel history, and occupational or unusual exposures modify the risk of various microbial aetiologies of CAP.

INVESTIGATIONS

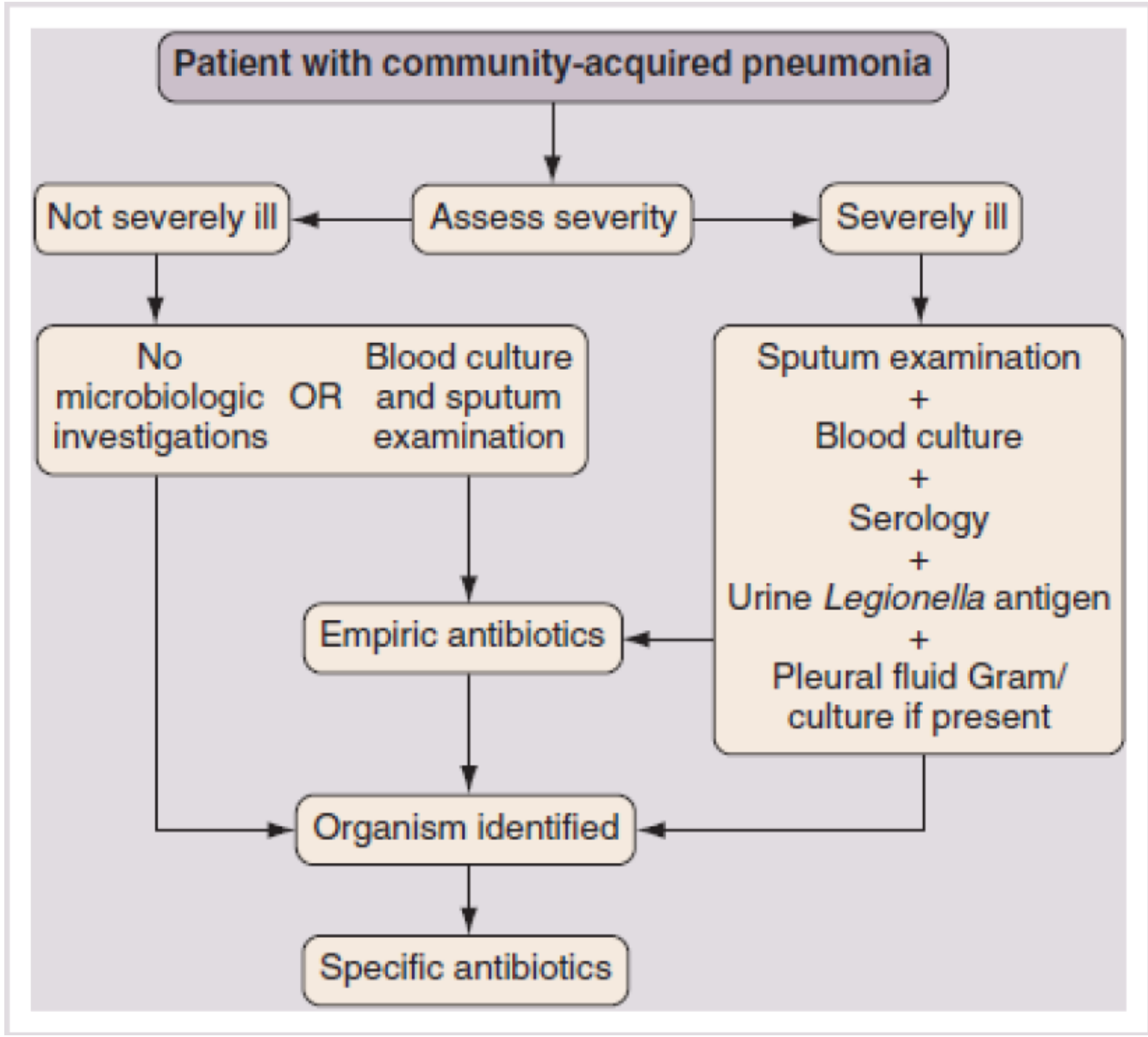
- CBC: Identifies bacterial infections- it shows left shift (increased WBC count), platelets are also important prognostic factors (high platelets → poor prognosis).
- CRP and ESR → inflammatory markers.
- Procalcitonin PCT: a marker of bacterial infection, if negative think of non-bacterial infection.
- We also do KFT for medications adjustment, and LFT because L.pneumophila can elevate liver enzymes.

- *Radiological*: CXR + CT
- *Laboratory*: CBC + Serum Glucose + Electrolyte measurements + Pulse oximetry or ABG + CRP + Procalcitonin.
- *Microbiological*: Sputum Culture + Blood Culture.
- *Invasive Procedures*: Pleural Tap + Bronchoscopy (has specific indications, Qualitative and Quantitative cultures).
- *Antigen detection and serology markers*: S.pneumonia (Urine antigen detection Sensitivity: 50-80%, Specificity: 90%, Pleural fluid antigen detection Sensitivity and Specificity: almost 100%), L.pneumophila serogroup 1 (Sensitivity: 60-80%, Specificity: >95%, Only testing Serogroup 1), Antigens for many common respiratory viruses (Influenza virus, Respiratory Syncytial virus, Adenovirus, Parainfluenza virus, → can be detected by Direct Immunofluorescence or by Enzyme linked Immunoassay), Some pathogens are detected by Nucleic acid Amplification tests (M.pneumonia, C.pneumonia, L.pneumophila, Bordetella Pertussis), Serology test used to be done to establish a microbiologic diagnosis for pneumonia caused by pathogens that cannot be readily cultured.

CRITERIA TO CONSIDER ADMISSION TO AN ICU FOR PATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA WITHOUT SHOCK OR RESPIRATORY FAILURE

- Respiratory rate > 30 breaths/min
- PaO_2/FIO_2 ratio < 250 or arterial saturation ≤90% on room air
- Multilobar or bilateral radiographic involvement or pleural effusion
- Confusion or disorientation
- Uremia (BUN level > 20 mg/dL)
- Leukopenia (WBC count < 4000 cells/dL) or extreme leucocytosis >20,000 cells/dL
- Thrombocytopenia (platelet count < 100,000 cells/dL
- Hypothermia (core temperature < 36° C
- Hypotension requiring aggressive fluid resuscitation
- Acidosis (pH < 7.30)
- Hypoalbuminemia (albumin < 3.5 g/dL)
- Hyponatremia (sodium < 130 mEq/L)
- Tachycardia (>125/min)

DIAGNOSTIC APPROACH TO THE PATIENT WHO HAS COMMUNITY-ACQUIRED PNEUMONIA



COMMON CAUSES OF COMMUNITY-ACQUIRED PNEUMONIA IN PATIENTS WHO DON'T REQUIRE HOSPITALIZATION

Mycoplasma pneumoniae

Streptococcus pneumoniae

Chlamydophila pneumoniae

Haemophilus influenzae

Respiratory viruses

COMMON CAUSES OF COMMUNITY-ACQUIRED PNEUMONIA IN PATIENTS WHO REQUIRE HOSPITALIZATION

Streptococcus pneumoniae
Mycoplasma pneumoniae
Chlamydophila pneumoniae
Haemophilus influenzae
Staphylococcus aureus
Mixed infections
Enteric gram-negative bacilli
Aspiration (anaerobes)
Respiratory viruses
Legionella species

COMMON CAUSES OF SEVERE COMMUNITY-ACQUIRED PNEUMONIA

Streptococcus pneumoniae

Enteric gram-negative bacilli

Staphylococcus aureus

Legionella species

Mycoplasma pneumoniae

Respiratory viruses

Pseudomonas aeruginosa (relative frequency determined by the presence or absence of specific risk factors)

COMMON CAUSES OF HOSPITAL-ACQUIRED PNEUMONIA

setting	pathogen
2 TO 5 DAYS IN HOSPITAL Mild to moderate pneumonia Severe pneumonia "low-risk"	Enterobacteriaceae Streptococcus pneumoniae Haemophilus influenza Methicillin-sensitive Staphylococcus
≥5 DAYS IN HOSPITAL Mild to moderate pneumonia	as above
≥5 DAYS IN HOSPITAL Severe HAP "low risk"	Pseudomonas aeruginosa Enterobacter spp. Acinetobacter spp.
≥2 DAYS IN HOSPITAL Severe HAP "high risk"	as above
Recent abdominal surgery or witnessed aspiration	Anaerobes

HIGH RISK GROUPS FOR HOSPITAL-ACQUIRED PNEUMONIA

- Age >65 years
- Pancreatitis
- COBD
- CNS dysfunction (Stroke, Drug overdose, Coma, Status Epilepticus)
- CHF
- Malnutrition
- DM
- Endotracheal intubation
- Renal Failure
- Complicated thoracoabdominal surgery
- Alcoholism

COMMON CAUSES OF VENTILATOR ASSOCIATED PNEUMONIA

Pseudomonas

S. aureus(MRSA 56%)

Enterobacteriacea

Haemophilus spp.

Streptococcus spp.

Acinetobacter spp.

S. pneumoniae

Neisseria spp.

S. Maltophilia

CAUSES OF NONRESPONDING PNEUMONIA

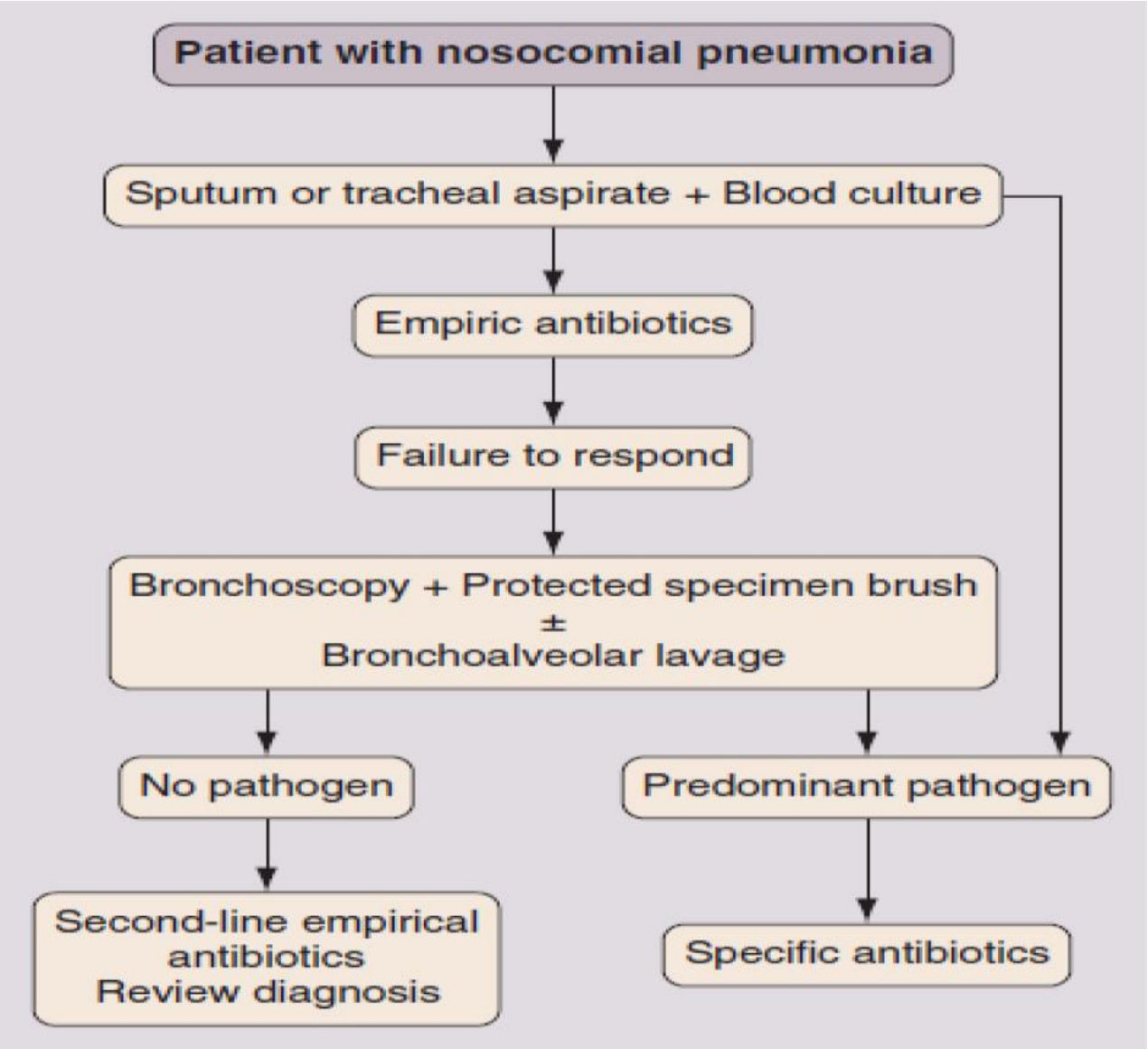
INFECTIOUS

1. Resistant microorganisms
2. Community-acquired pneumonia (e.g., *Streptococcus pneumoniae, Staphylococcus aureus*
3. Nosocomial pneumonia (e.g., *Acinetobacter*, methicillin-resistant *Staphylococcus aureus, Pseudomonas aeruginosa*)
4. Uncommon microorganisms (e.g., *Mycobacterium tuberculosis*, *Nocardia* spp., fungi, *Pneumocystis jirovecii*)
5. Complications of pneumonia
6. Empyema
7. Abscess or necrotizing pneumonia
8. Metastatic infection

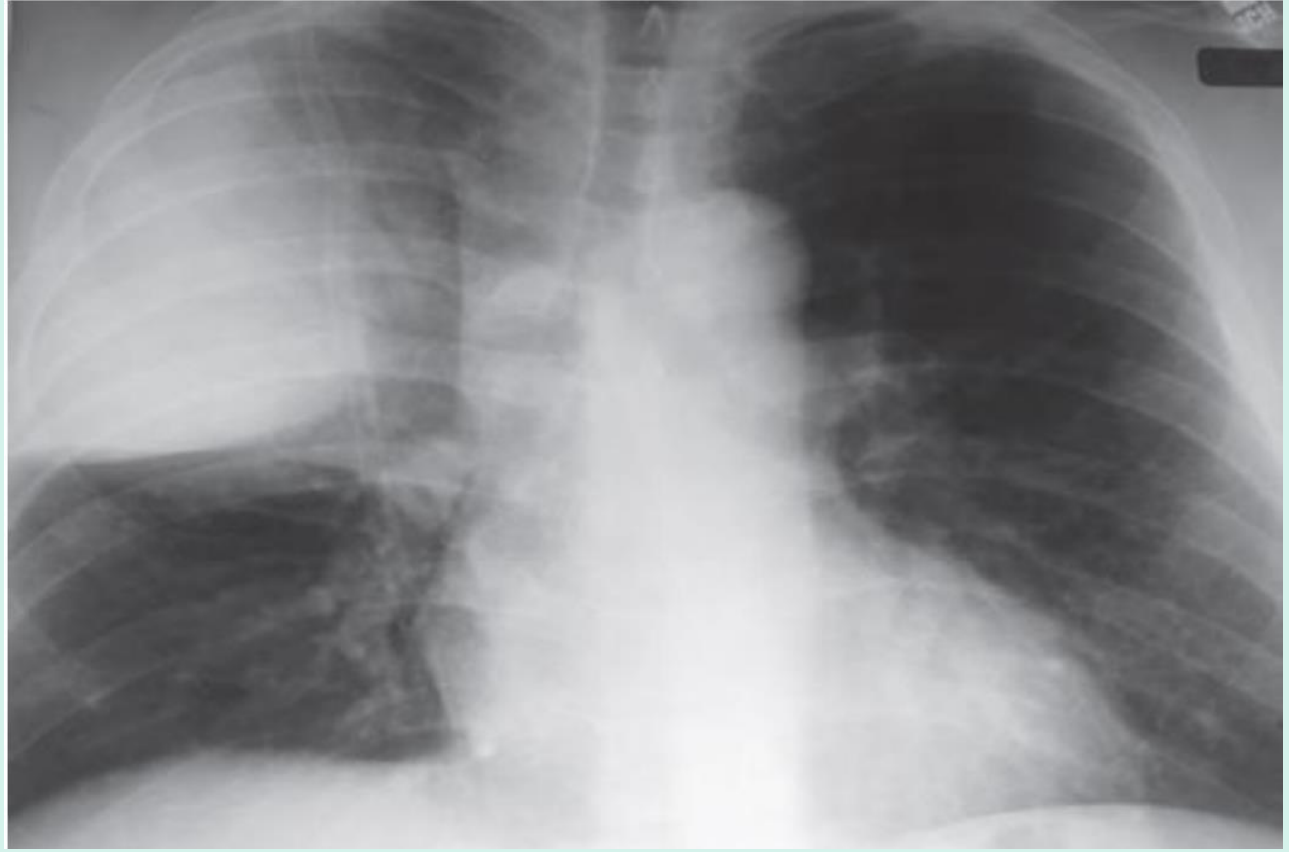
NONINFECTIOUS

1. Neoplasms
2. Pulmonary hemorrhage
3. Pulmonary embolism
4. Sarcoidosis
5. Eosinophilic pneumonia
6. Pulmonary edema
7. Acute respiratory distress syndrome
8. Organizing pneumonia
9. Drug-induced pulmonary disease
10. Pulmonary vasculitis

DIAGNOSTIC APPROACH TO THE PATIENT WHO HAS NOSOCOMIAL PNEUMONIA

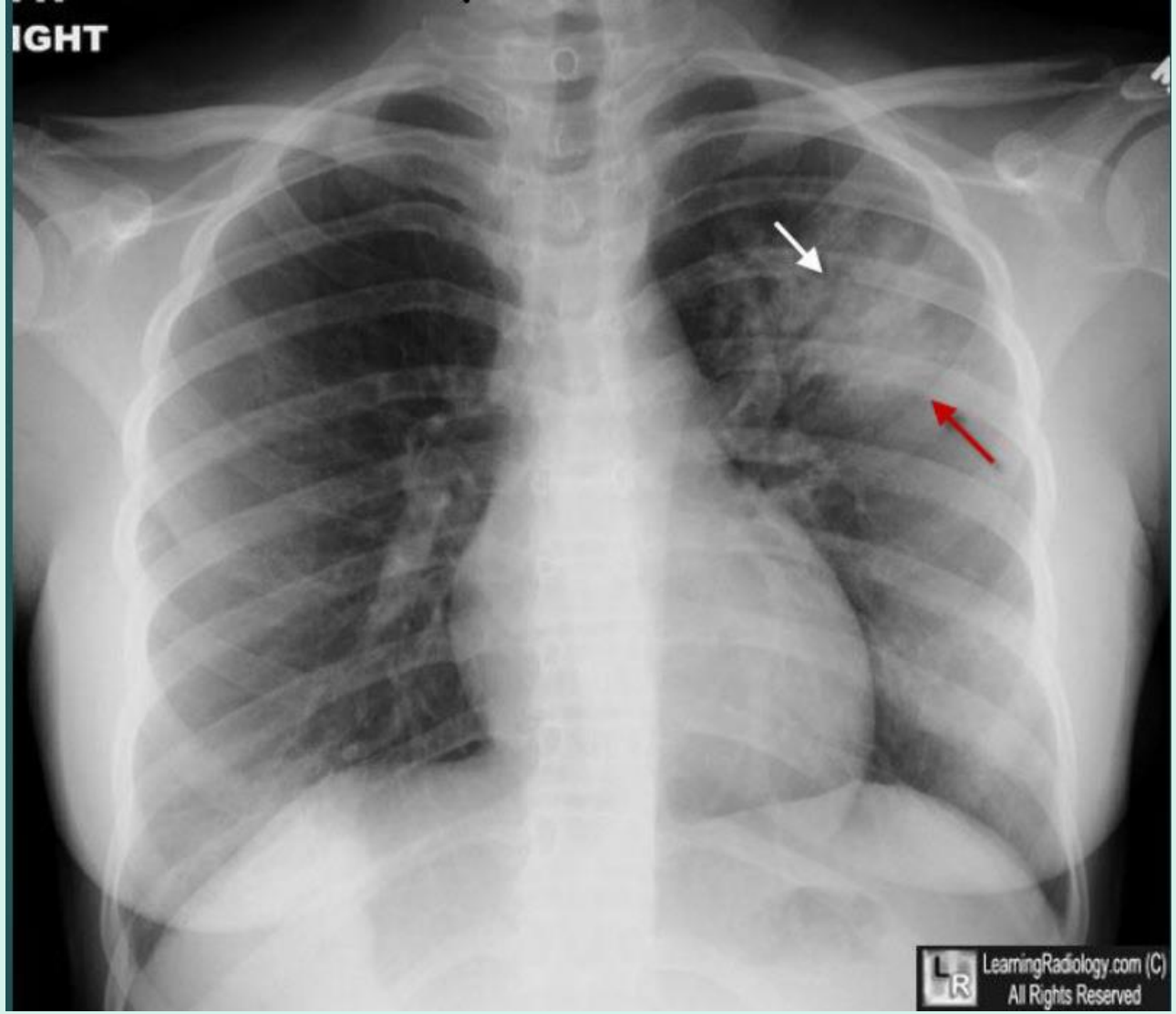


FRONTAL CHEST X-RAY - S.PNEUMONIA LOBAR PNEUMONIA



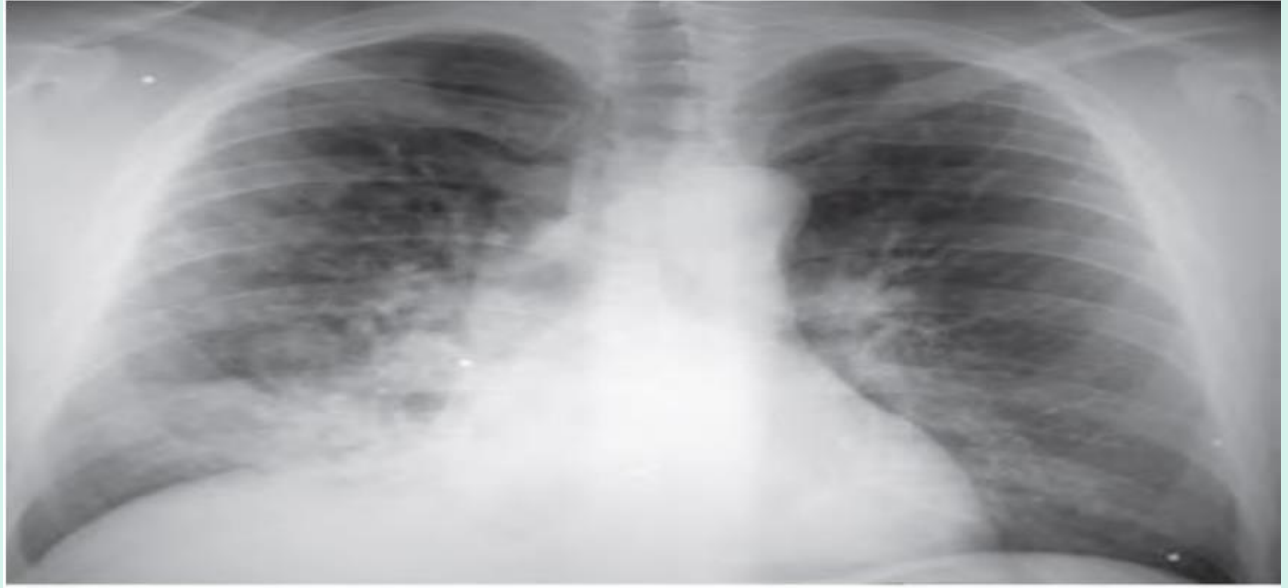
- The X-ray shows Homogenous increased opacity conforming to the shape of the Rt Upper lobe, extending to the pleural surfaces, associated with air bronchograms → Lobar Pneumonia (most commonly with S.pneumonia).
- Air Bronchograms: gas-filled bronchi surrounded by alveoli filled with fluid, pus or other materials. It's a very useful sign because it's highly sensitive and specific for the presence of lung consolidation (dense white area with Air bronchograms - black lines-).

TYPICAL LOBAR PNEUMONIA



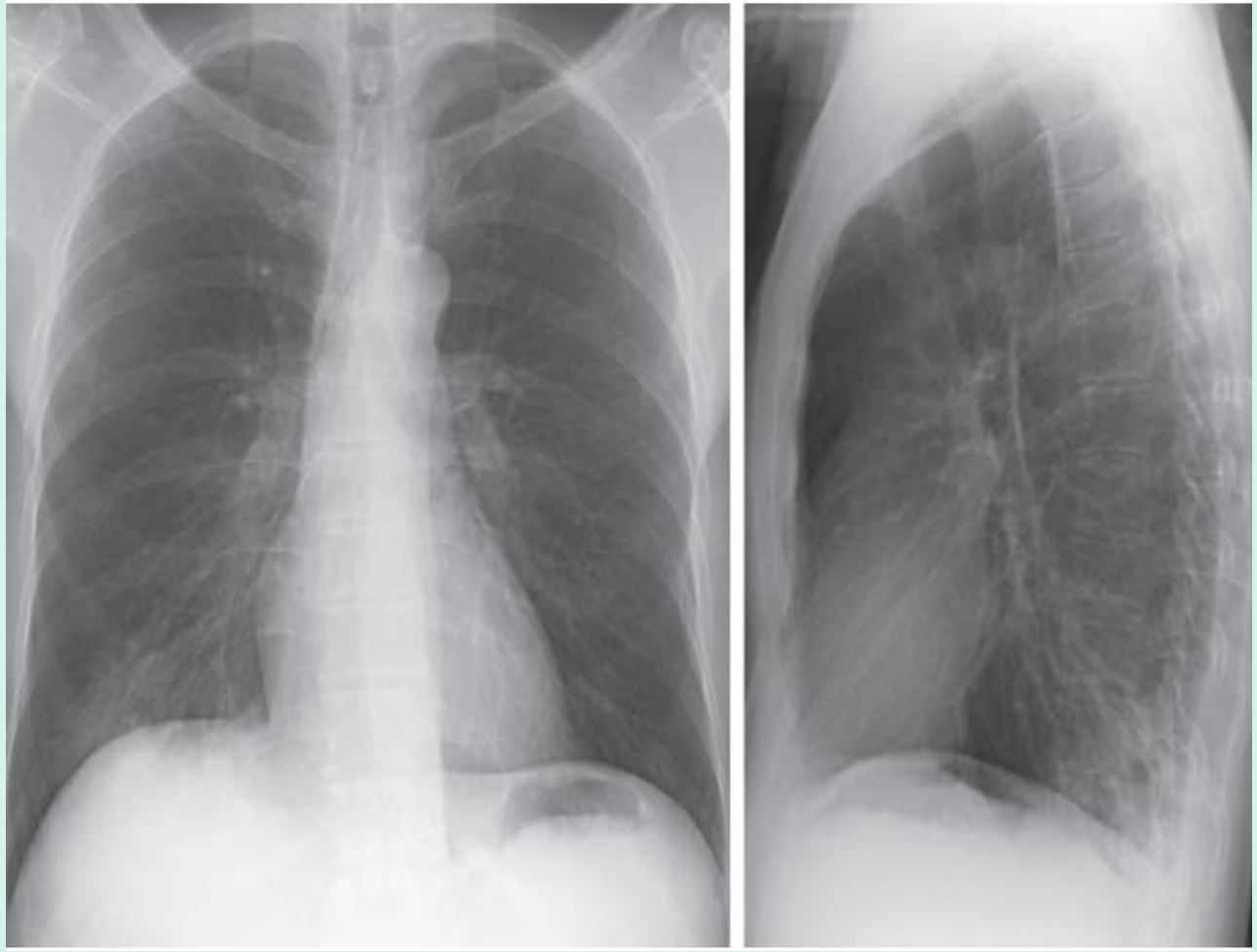
- Most seen with S.pneumonia infection

FRONTAL CHEST X-RAY AND CT - M.PNEUMONIA PNEUMONIA



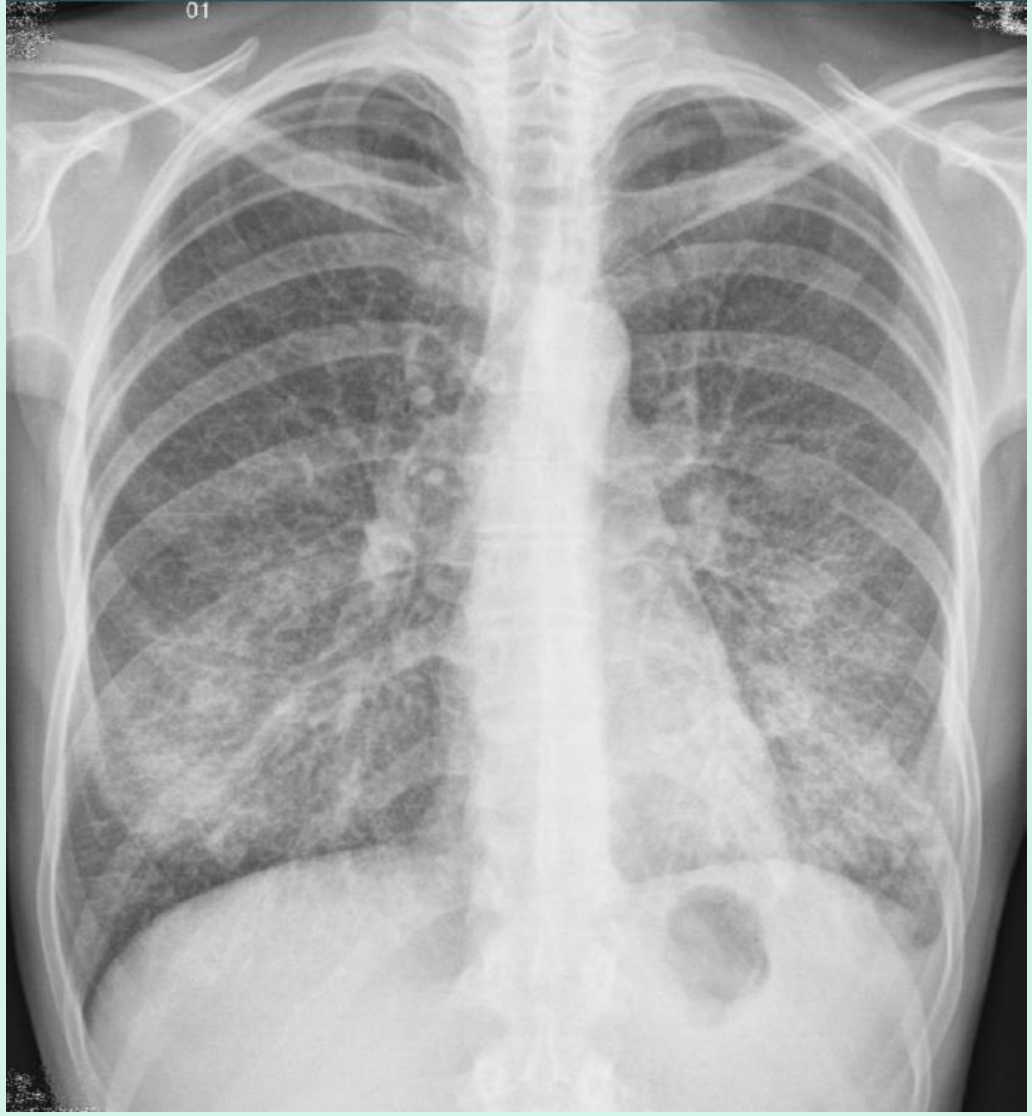
- The CXR shows Rt lower lobe consolidation associated with several small nodular opacities which is consistent with Acinar or Air Space nodules.
- Most seen with M.pneumonia infection.

CXR OF M.PNEUMONIA PNEUMONIA



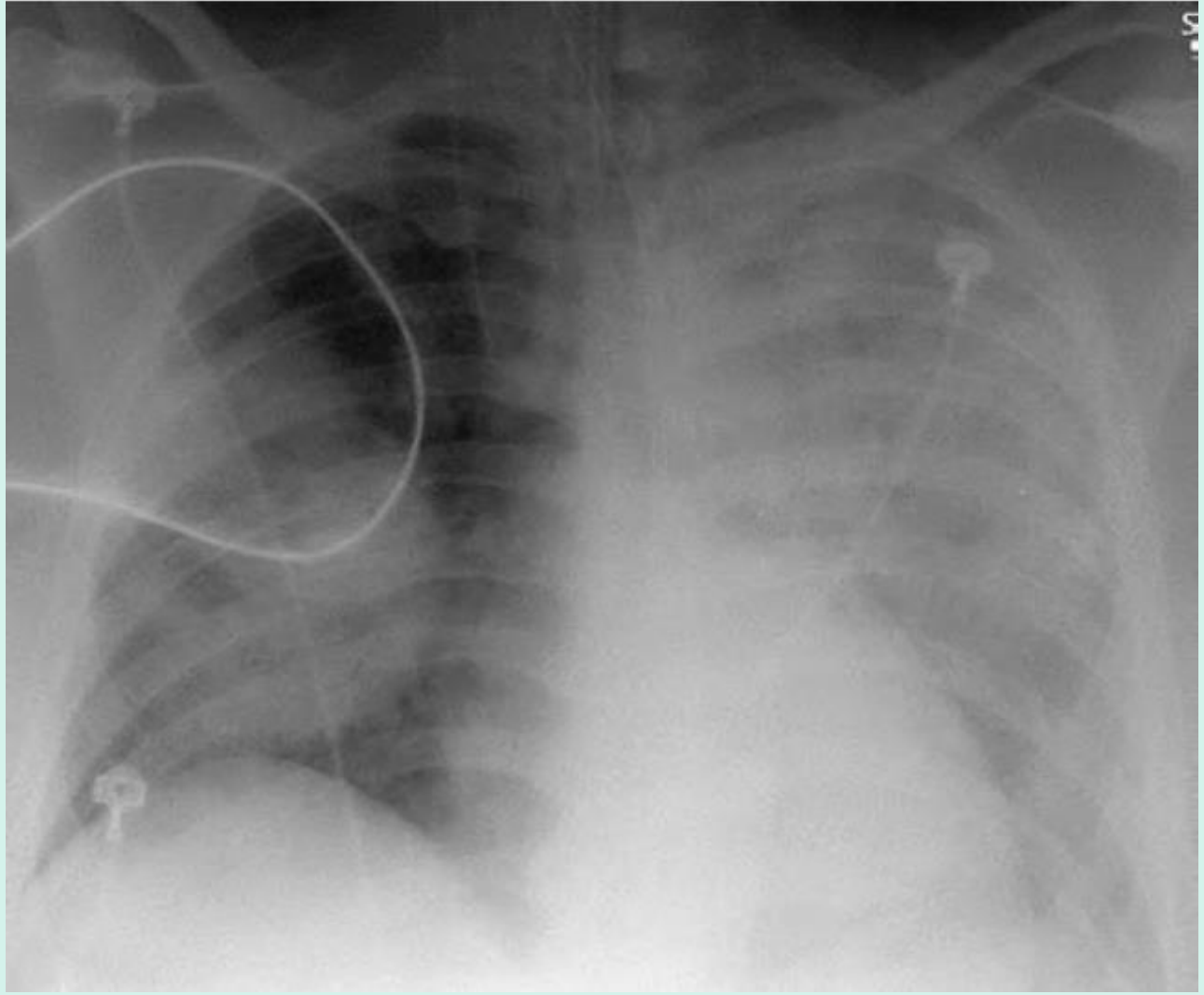
- This radiograph shows patchy Rt Lower lobe consolidation consistent with Bronchopneumonia.
- Mostly seen with M.pneumonia infection.

ATYPICAL PNEUMONIA CXR



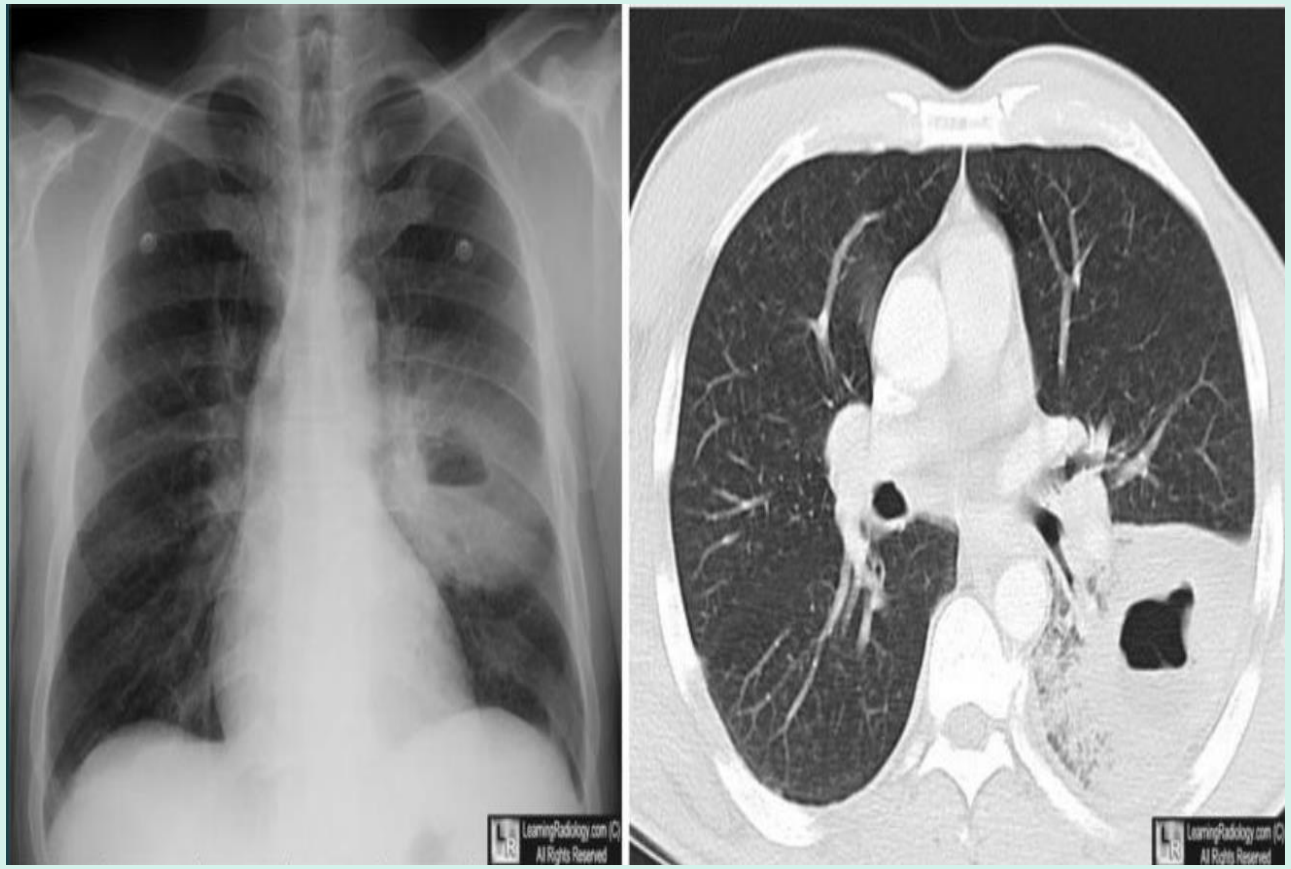
- CXR showing bilateral opacities and infiltrates consistent with Atypical Pneumonia.
- Mostly seen with M.pneumonia, L.pneumophila

FRONTAL CXR - L.PNEUMOPHILA PNEUMONIA AND RESPIRATORY FAILURE.



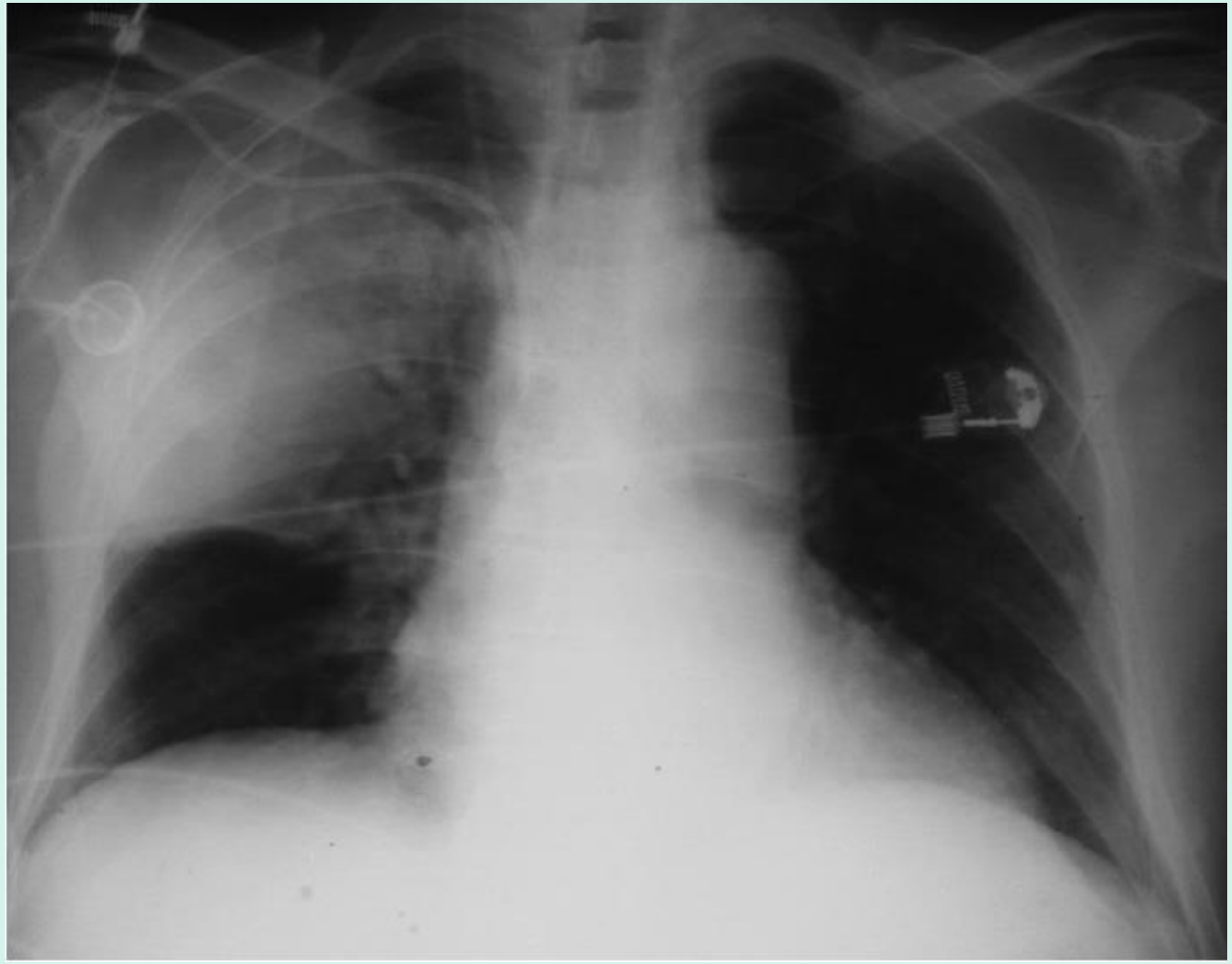
- This radiograph shows Left-greater-than-Right Multilobar consolidation.
- Most seen with L.pneumophila.

ABNORMAL CXR



- This radiograph shows Air-Fluid level in the left lung, which is suggestive of Lung Abscess, Hydropneumothorax, or Diaphragmatic Hernia

CXR OF K.PNEUMONIA PNEUMONIA



- This radiograph shows a Bulging fissure in the Rt Lung, suggestive of pneumonia K.pneumonia infection.

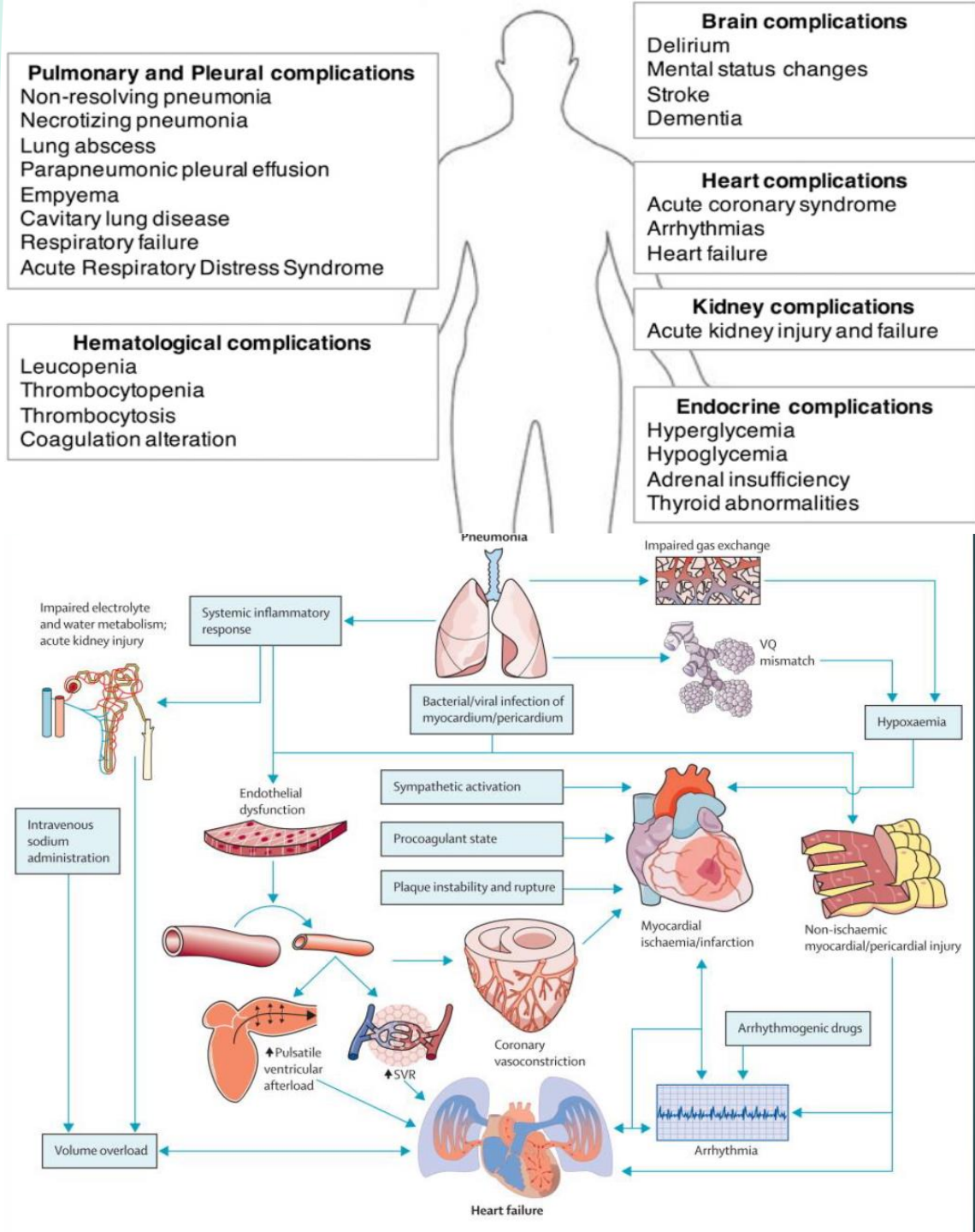
CURB-65 SCORING SYSTEM

CURB-65	Clinical Feature	Points
C	Confusion	1
U	Urea > 7 mmol/L	1
R	RR ≥ 30	1
B	SBP ≤ 90 mm Hg OR DBP ≤ 60 mm Hg	1
65	Age > 65	1

CURB-65 Score	Risk group	30-day mortality	Management
0 -1	1	1.5%	Low risk, consider home treatment
2	2	9.2%	Probably admission vs close outpatient management
3-5	3	22%	Admission, manage as severe

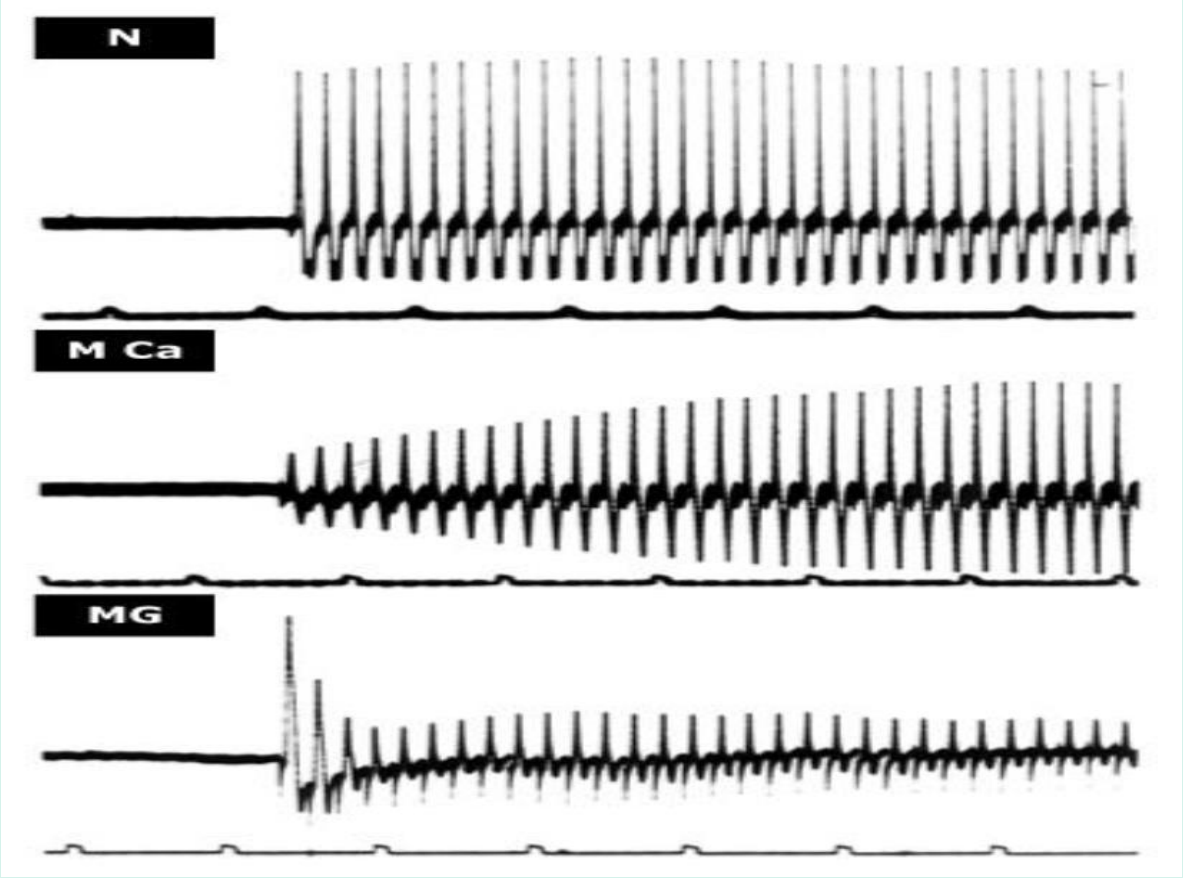
- This score is used to predict mortality from pneumonia.

COMPLICATIONS OF PNEUMONIA

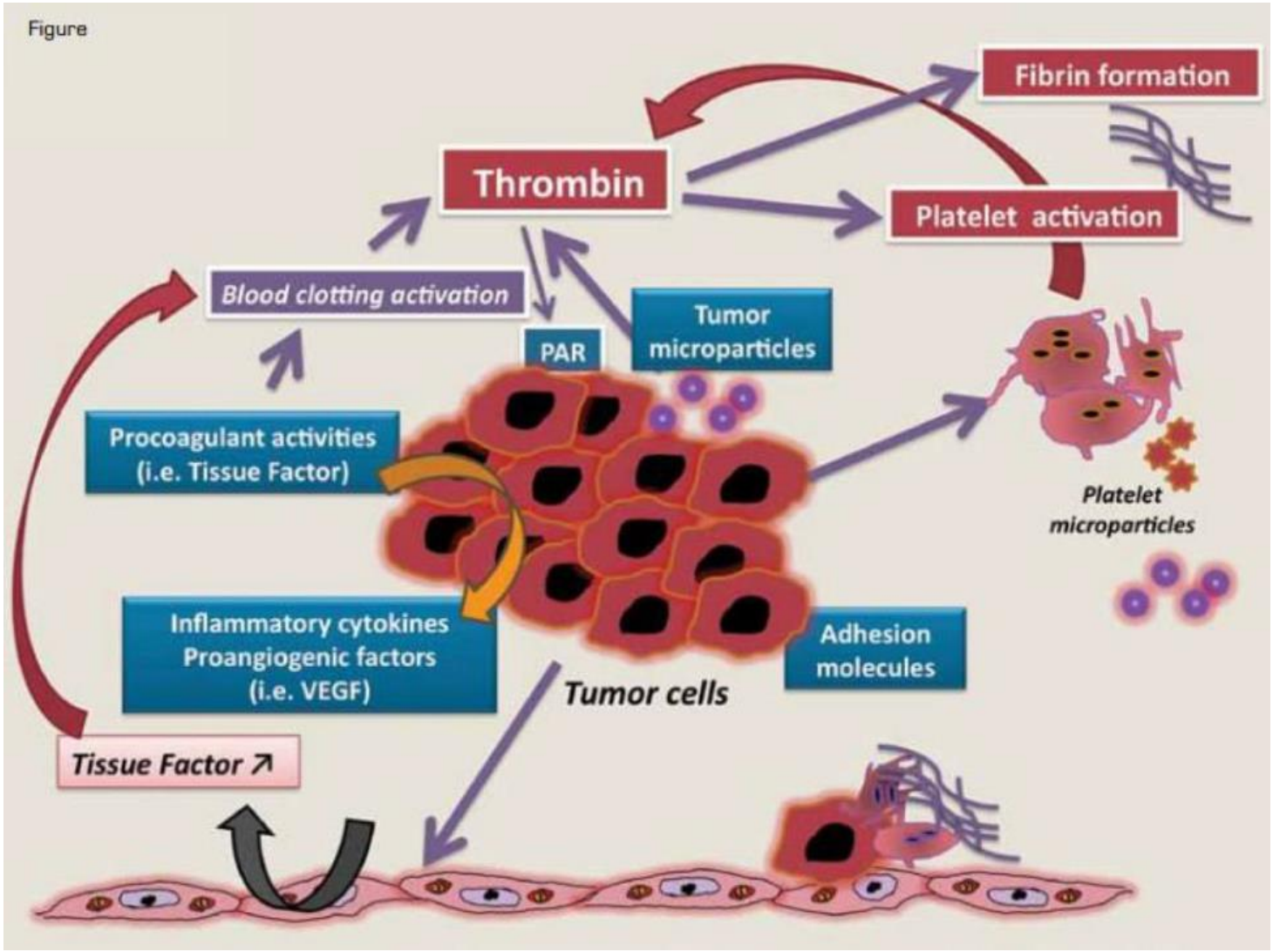


LUNG CANCER

NEUROLOGIC SIGNS AND SYMPTOMS OF PARANEOPLASTIC SYNDROME



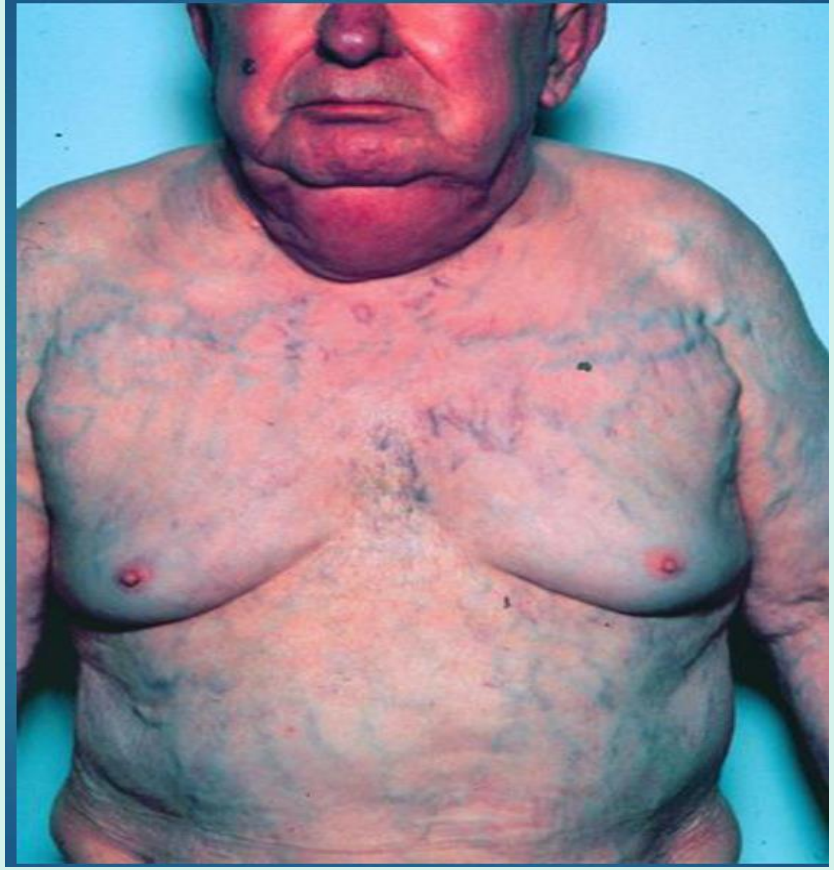
- In the first picture we have normal muscle fiber. Repetitive stimulation of a normal muscle fiber shows uniform strength.
- The second figure → Lambert-Eaton myasthenic syndrome. Repetitive stimulation of a muscle fiber in this case starts weak and becomes stronger.
- The third figure → Myasthenia Gravis. Repetitive stimulation of a muscle fiber in this case causes fatigue in the muscle.
- Lambert-Eaton syndrome is a condition in which the body's immune system attacks the connections between nerves and muscles.



PATHOPHYSIOLOGY OF HYPERCOAGULABILITY IN MALIGNANCY

- Tumor cells can cause hypercoagulable states due to their ability in activating the coagulation system on many levels.

SUPERIOR VENA CAVA SYNDROME



- This figure shows severe SVC obstruction.
- SVC syndrome:

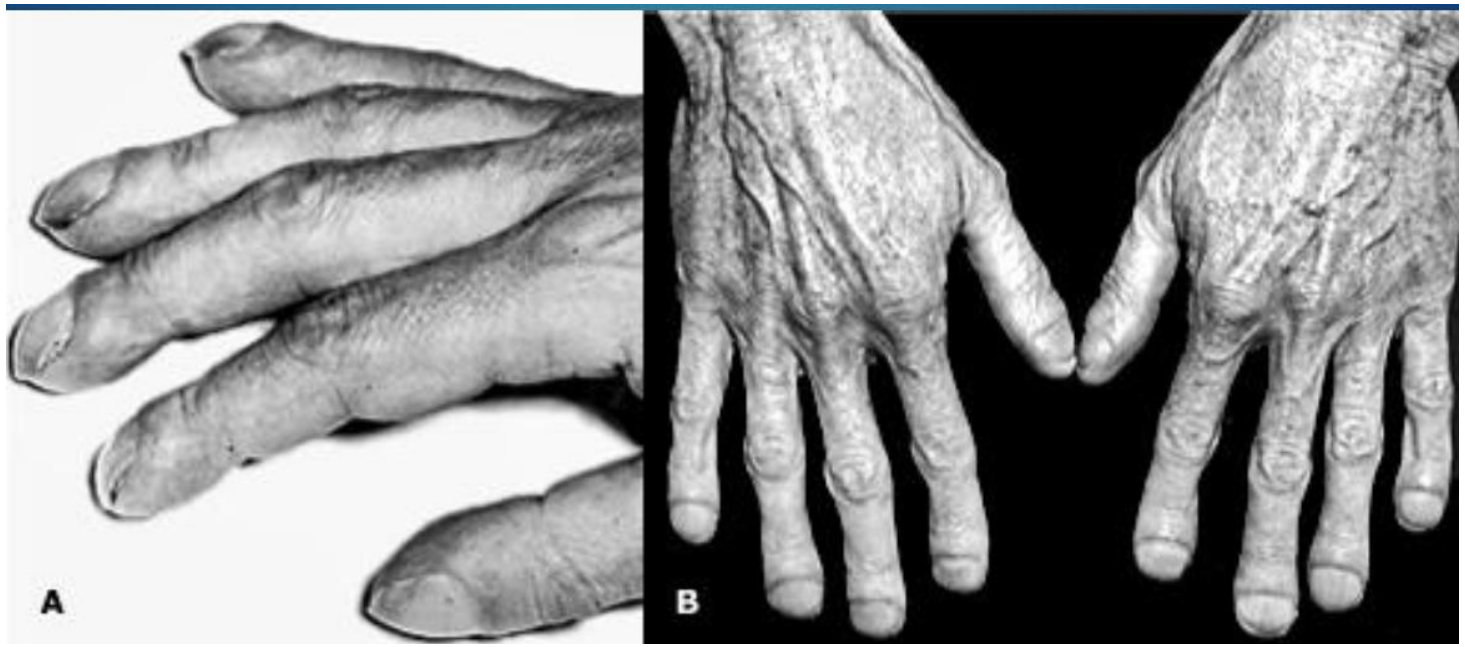
Patients present with sensation of fullness in the head and dyspnea. Cough, pain and dysphagia are less frequent.

On PE: Dilated neck veins, Prominent venous pattern on the chest, Facial Edema, and a Plethoric appearance.

The chest radiograph typically shows widening of the mediastinum or a Rt hilar mass

CT scan often identifies the cause, level of obstruction, and extent of collateral venous drainage.

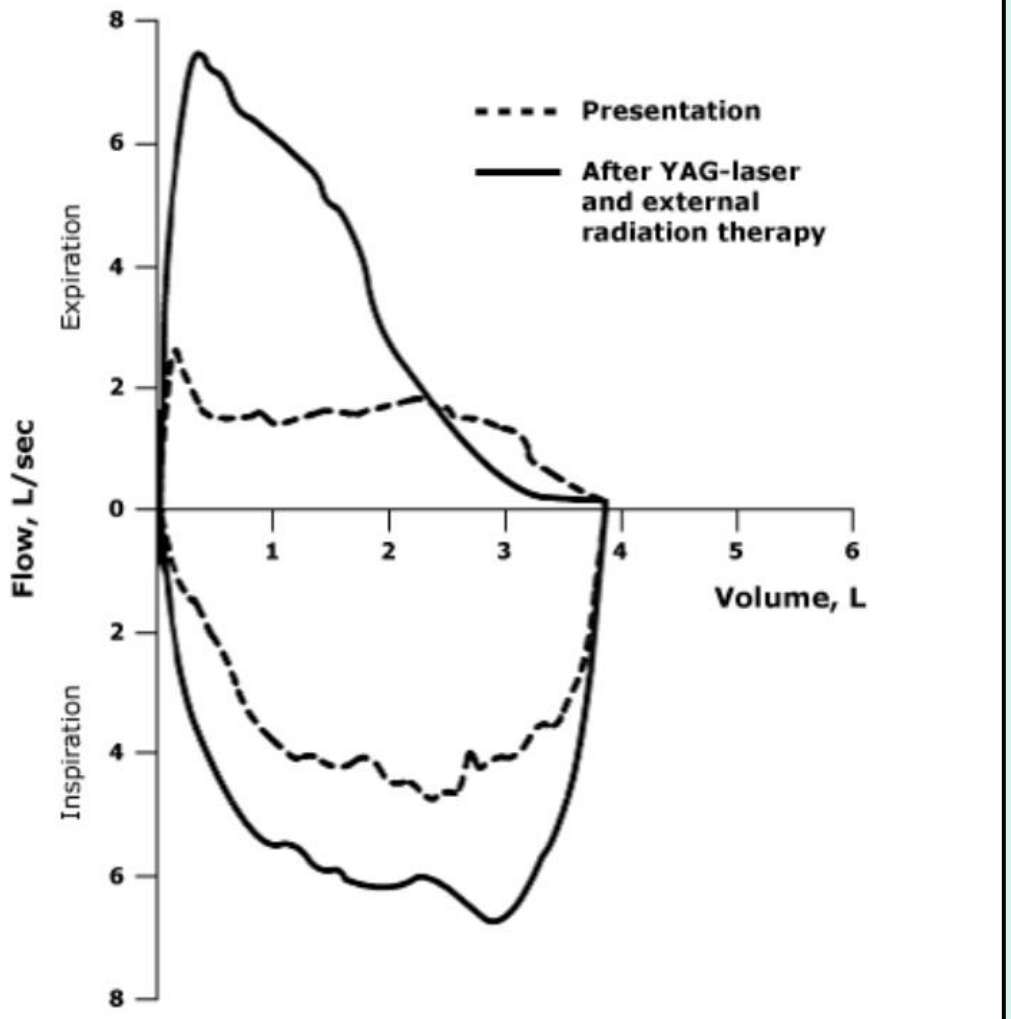
SVC syndrome is more common in SCLC than NSCLC.



FINGER CLUBBING

- One of the common clinical manifestations of lung cancer.

PFT OF A PATIENT WITH LUNG CANCER



- The PFT shows an obstructive pattern (major airway obstruction).

Clinically: through History and PE.

Laboratory: CBC, Electrolytes, Calcium, Alkaline phosphatase, ALT, AST, Total Bilirubin, Creatinine, Albumin, and Lactate dehydrogenase.

LFT abnormalities are possibly due to liver metastasis, and we should prompt evaluation of the liver with liver-directed imaging.

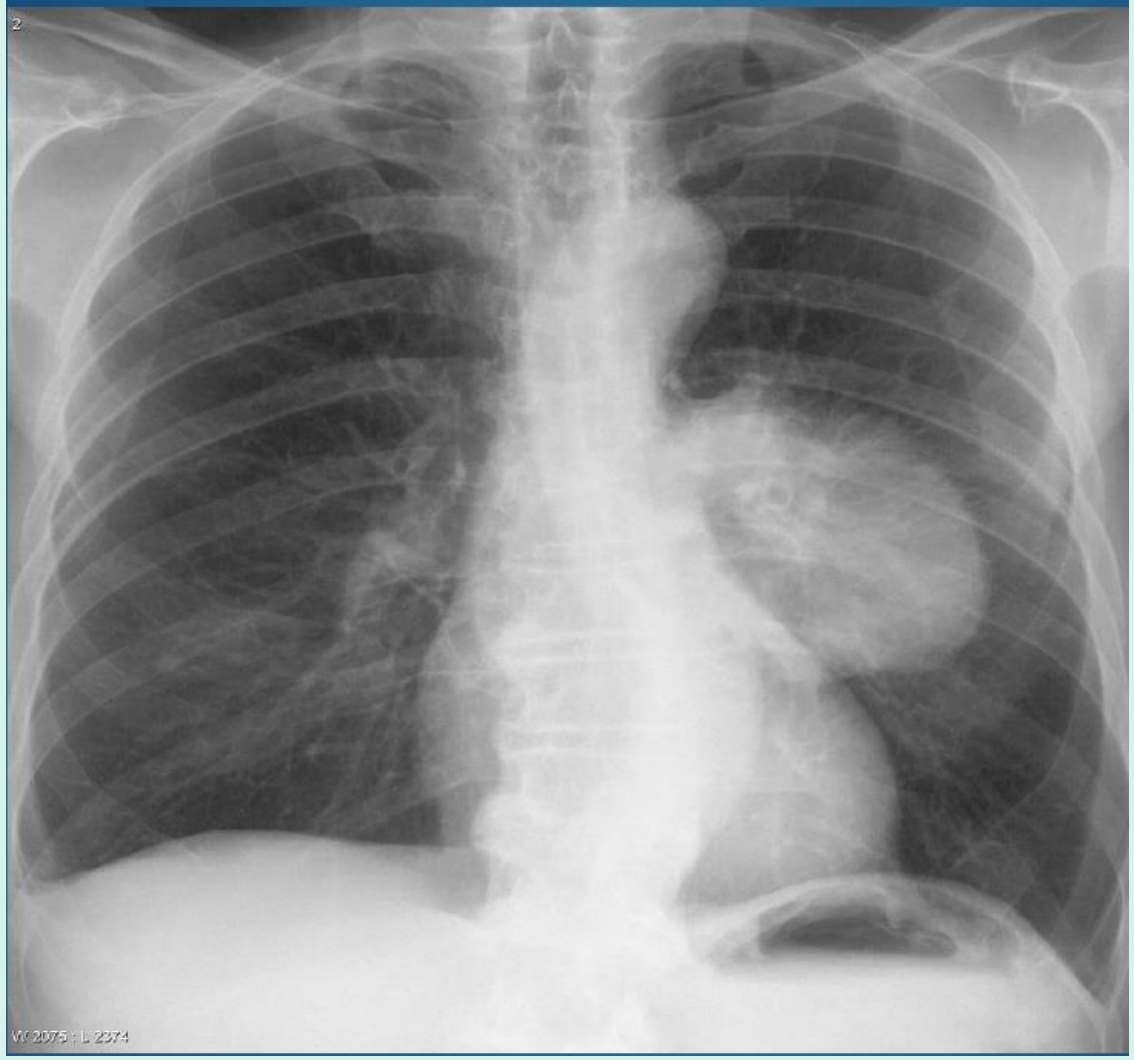
Calcium elevation should prompt additional imaging for bone metastasis and/or a work-up for a paraneoplastic manifestation of the primary tumor.

Elevation of alkaline phosphatase could be due to liver of bone mets and should prompt measurement Gama Glutamyl Transpeptidase (GGT). When GGT is normal, an evaluation for bone mets is indicated. When abnormal, an evaluation of Liver mets is indicated.

- Radiographic: CXR, CT, PET scan, Integrated PET/CT,

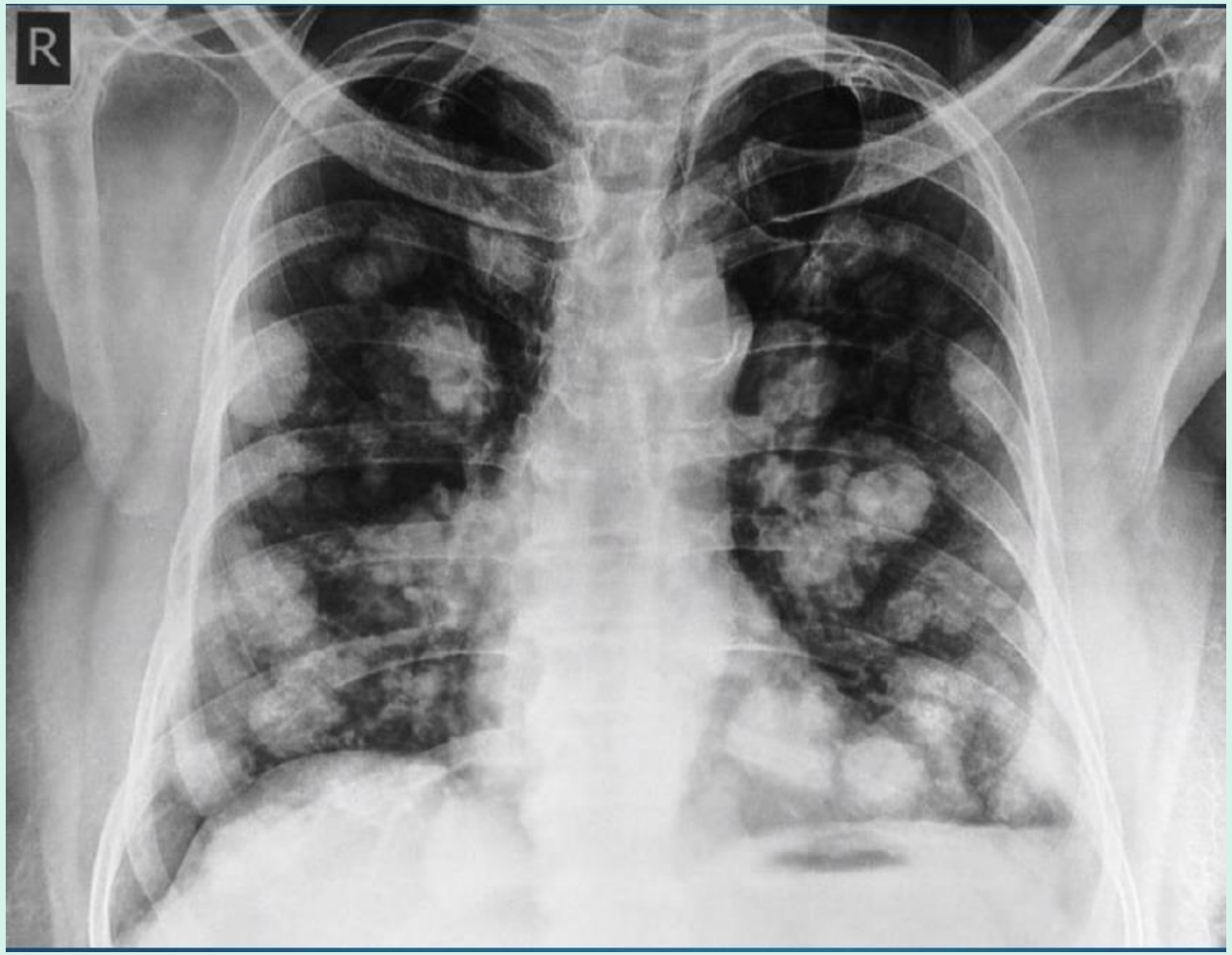
INITIAL EVALUATION OF LUNG CANCER

CXR OF A PATIENT WITH LUNG CANCER



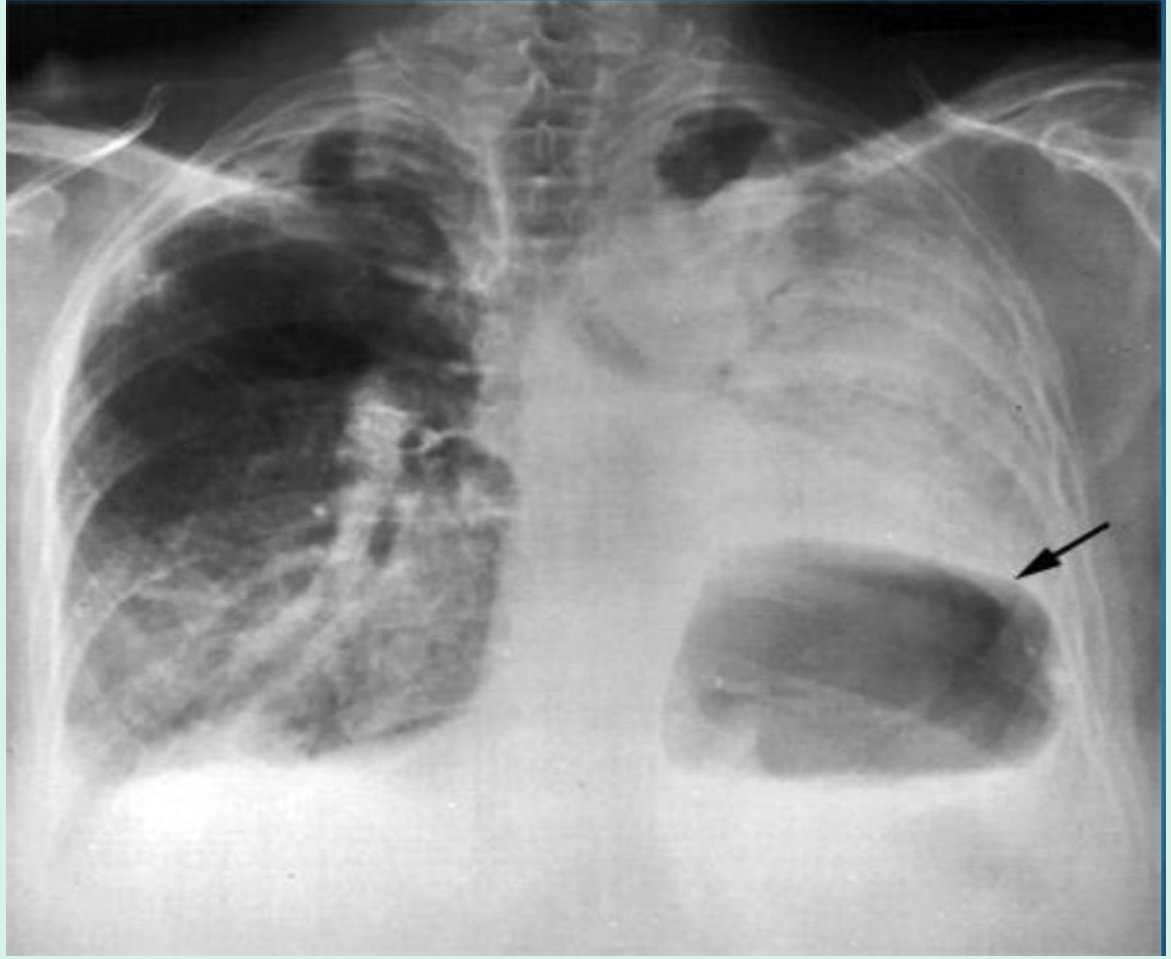
- The radiograph shows a central mass with a possibility of Phrenic nerve compression.
- When the mass is central, it's usually SCLC or Squamous cell carcinoma.
- Adenocarcinoma is usually peripheral

CXR OF A PATIENT WITH LUNG CANCER



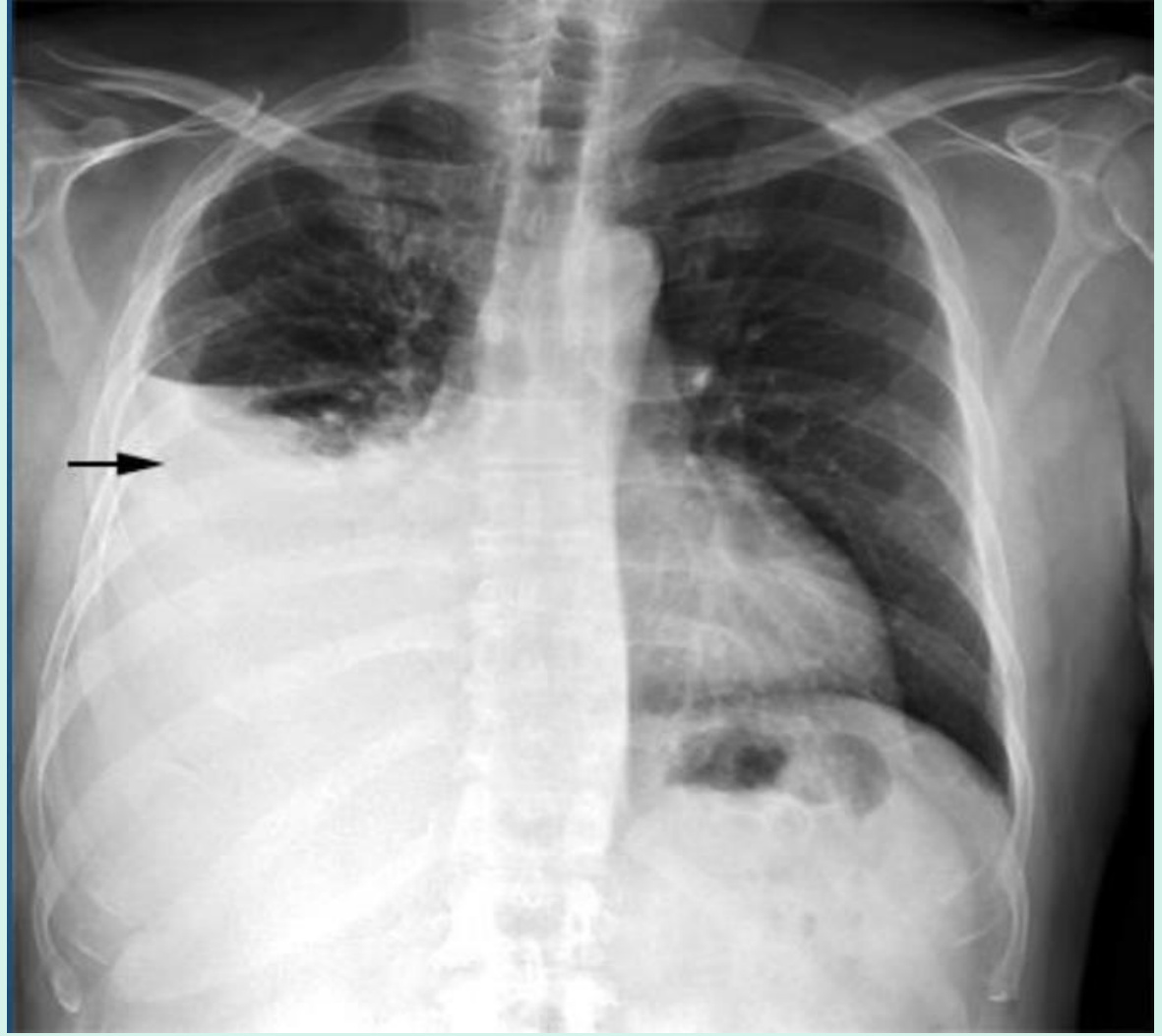
- *Canon Balls appearance*: Multiple metastasis, which can be either from the lung itself (primary) or from distant sites (secondary).

CXR OF A PATIENT WITH LUNG CANCER



- On this radiograph we can see that the diaphragm is elevated, and the tumor is above it (white) and it could be invading the phrenic nerve causing diaphragmatic paralysis.
- The Dark structure above the diaphragm is called Gastric bubble: a Radiolucent rounded area generally nestled under the Left hemidiaphragm representing gas in the fundus of the stomach.

PLEURAL EFFUSION

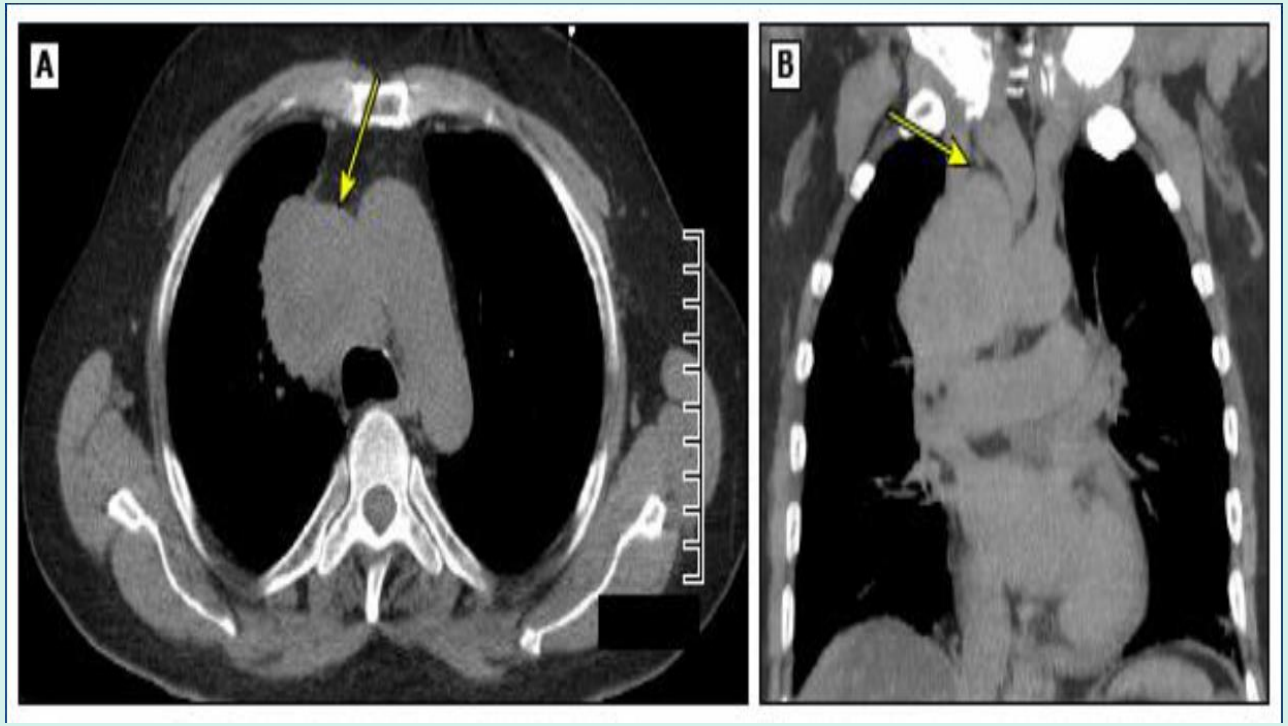


- Rt sided pleural effusion in a patient with Lung cancer.
- In patients with history of malignancy, Pleural effusions could be malignant indicating a Stage 4 cancer.

CT FINDINGS IN PATIENTS WITH LUNG CANCER

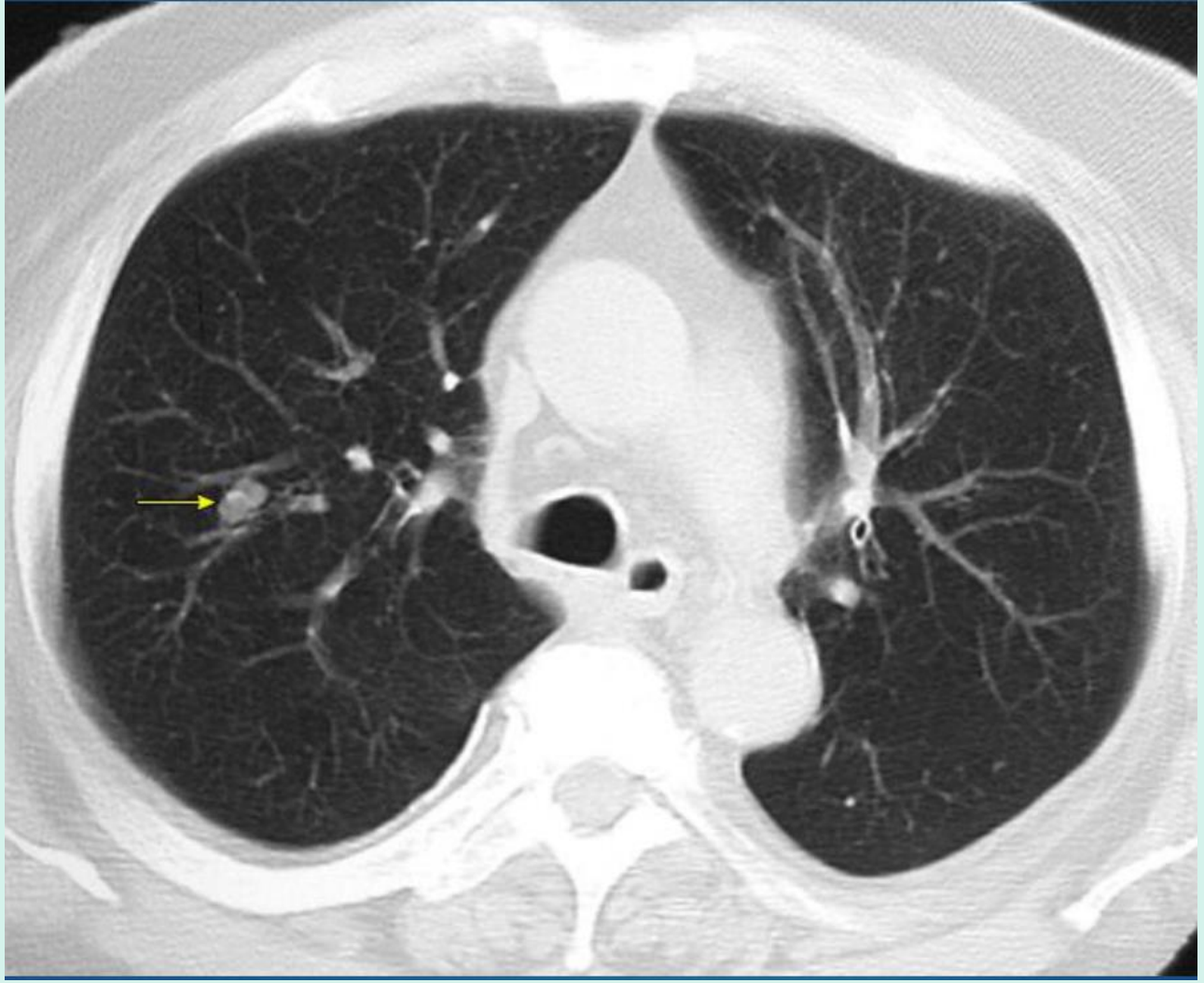
- Large Lesion size (>15 mm)
- Irregular or Speculated borders
- Upper lobe location
- Thick-walled cavitation
- Presence or development of a solid component within a ground glass lesion
- Detection of growth by follow-up imaging
- The finding of multiple nodules in a patient with a known or suspected extra-thoracic malignancy strongly suggests pulmonary metastasis

CT SCAN OF PATIENT WITH LUNG CANCER



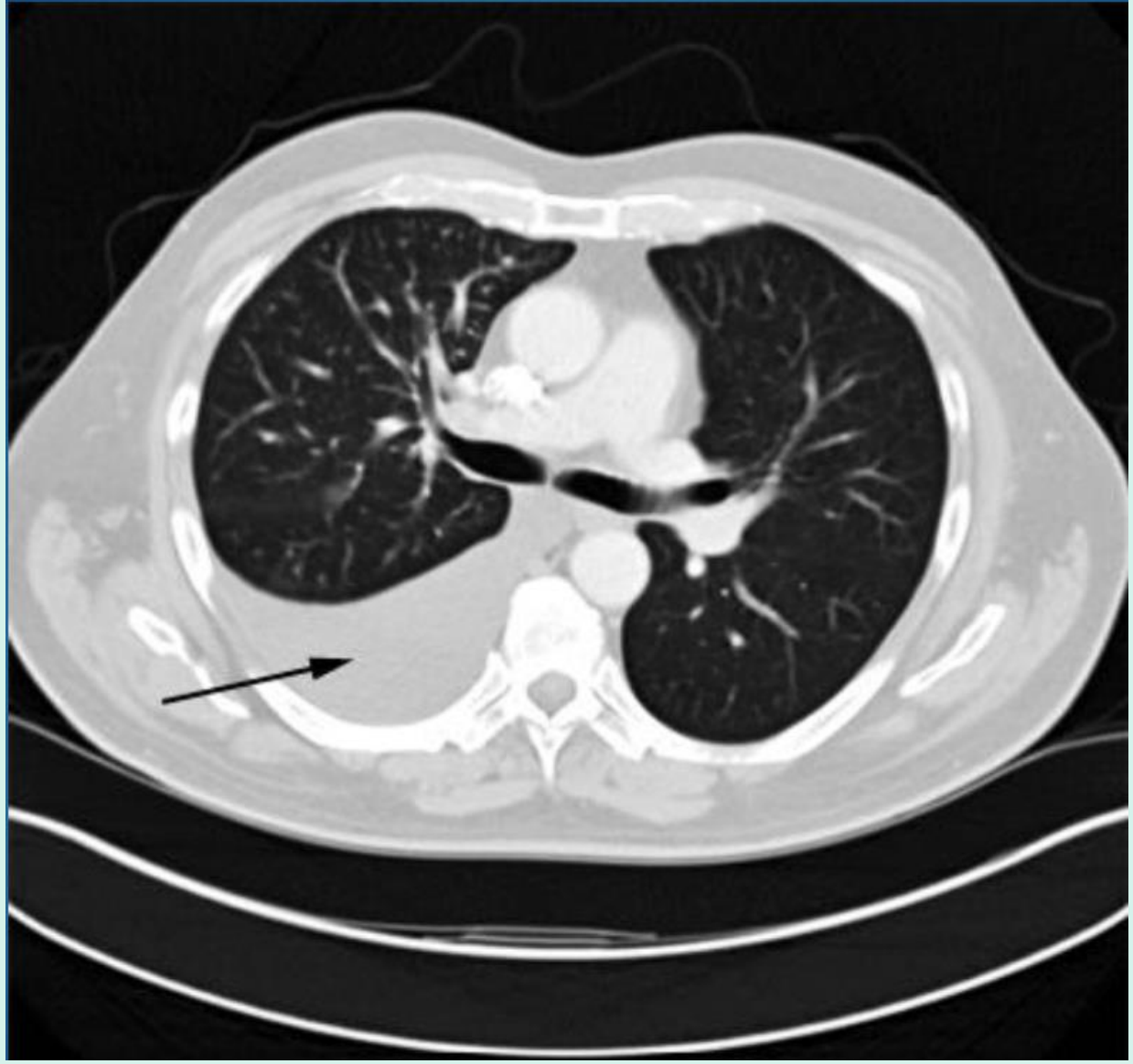
- The yellow arrows are pointing towards a Central Tumor.

CT SCAN OF A PATIENT WITH LUNG CANCER



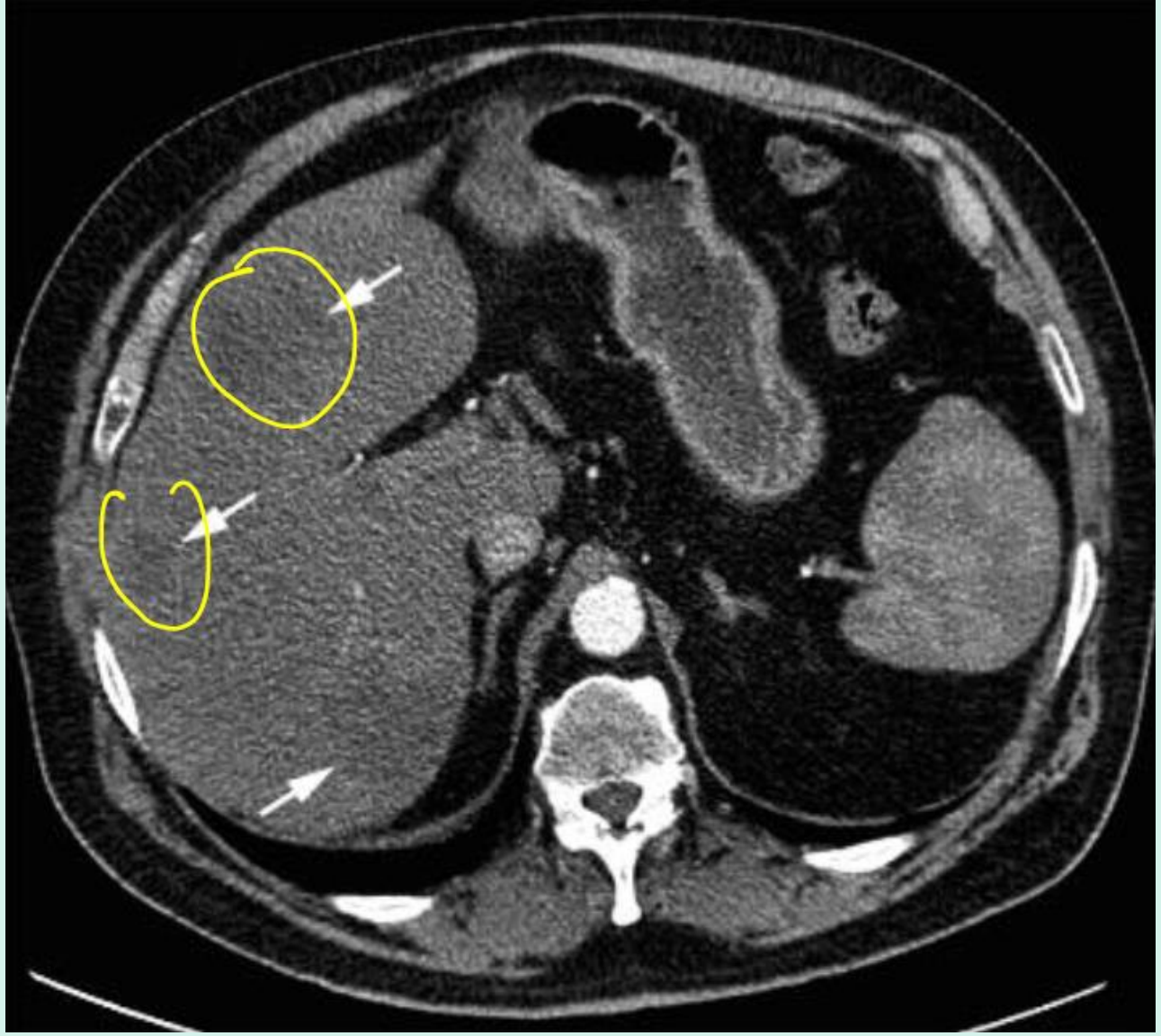
- The yellow arrow is pointing towards a small nodule.
- Nodules of this size won't show on CXR but will appear on CT.

CT SCAN OF A PATIENT WITH LUNG CANCER



- The black arrow is pointing to a very small effusion that appears only on CT not CXR.

CT SCAN OF A PATIENT WITH LUNG CANCER



- The scan shows small metastatic lesions on the Liver.

PET SCAN

Although whole body PET scan is more accurate than CT in detecting occult disease, its use hasn't been shown to improve survival.

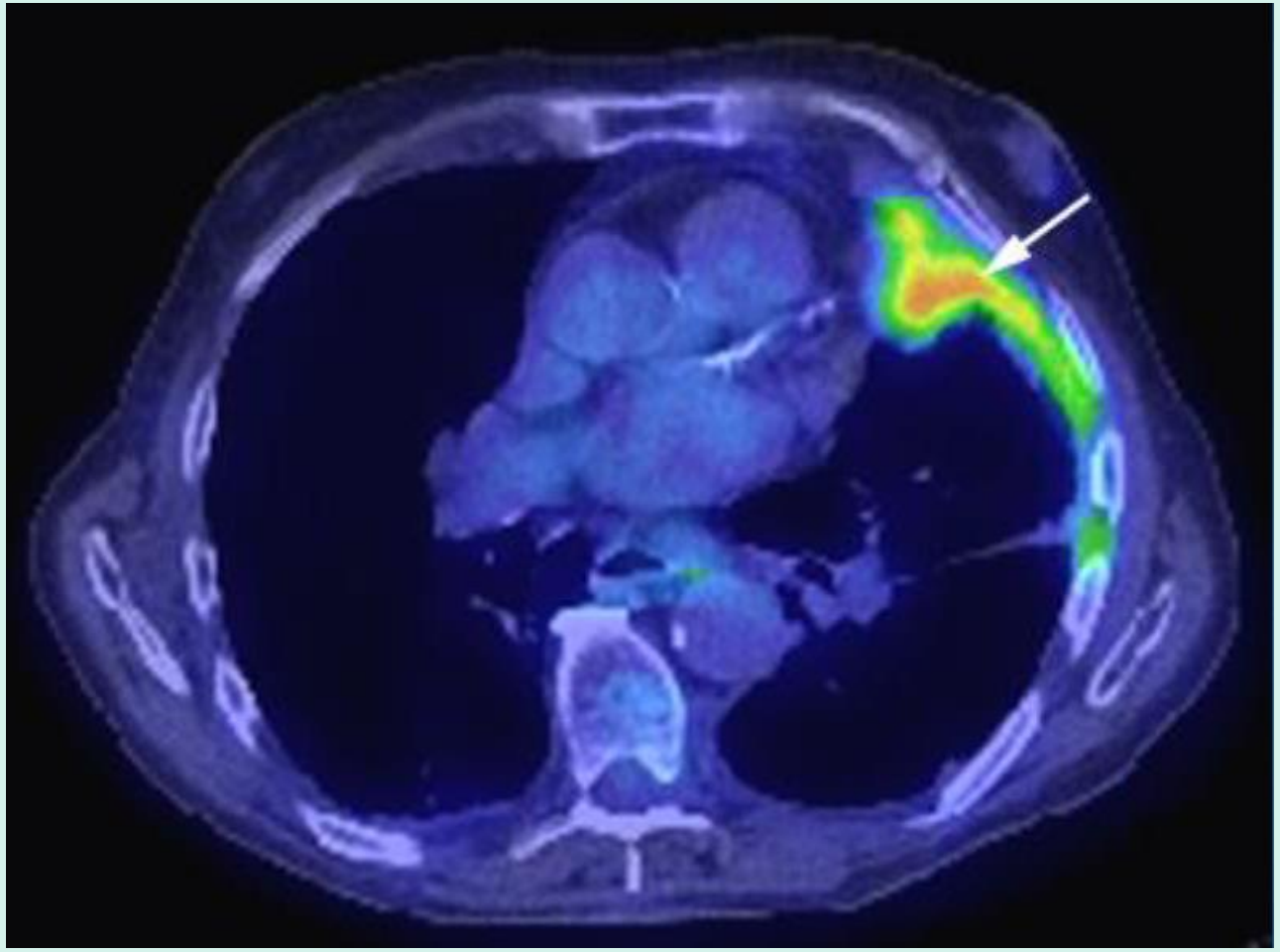
False positives can occur with benign FDG-avid lesions such as infections, inflammation, and granulomatous disease.

FDG: Fluorodeoxyglucose, is taken up whether it's a tumor, infection, or granulomatous disease.

False negatives typically occur when there are microscopic foci of metastasis, and in non-enlarged lymph nodes (<10mm).

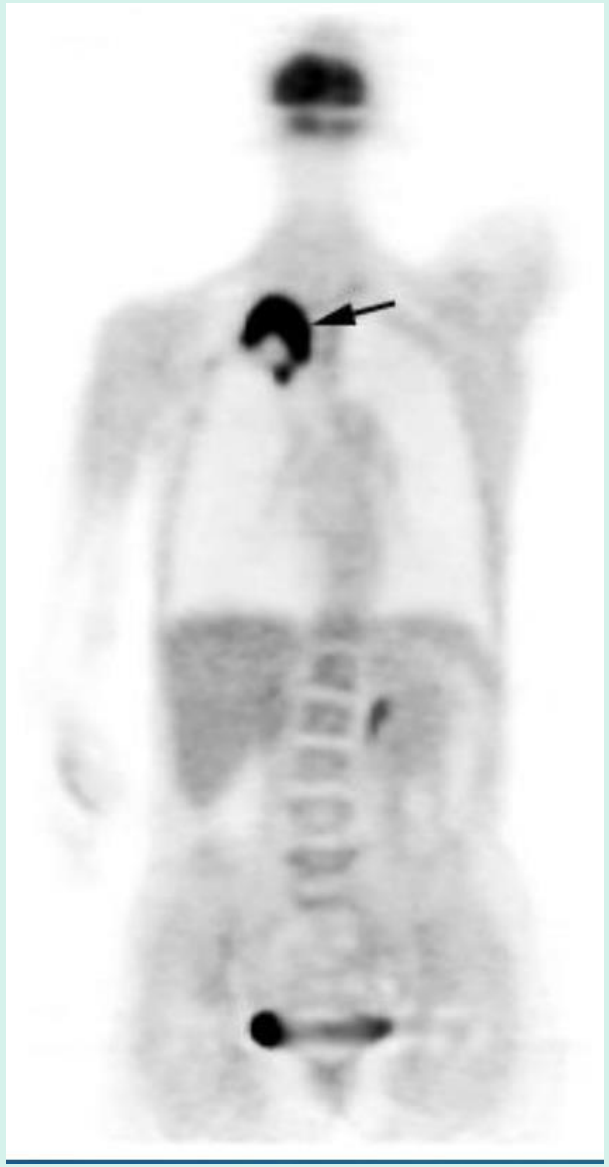
PET Scan isn't useful for brain mets since the brain cells are always active, i.e. it all light up, so we use MRI.

PET SCAN OF A PATIENT WITH LUNG CANCER

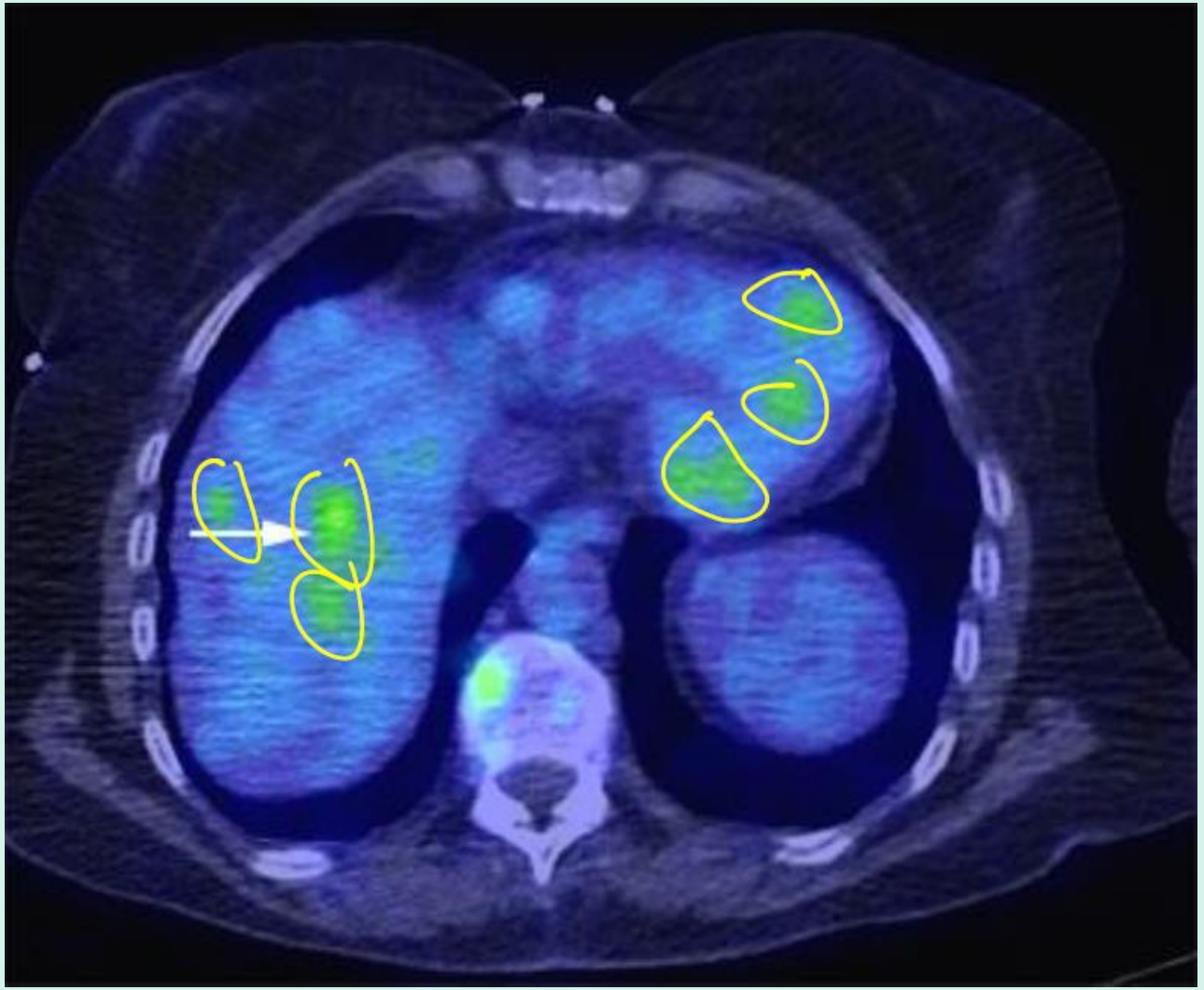


- This area can be a lung tumor, infection, granulomatous disease, etc.

PET SCAN

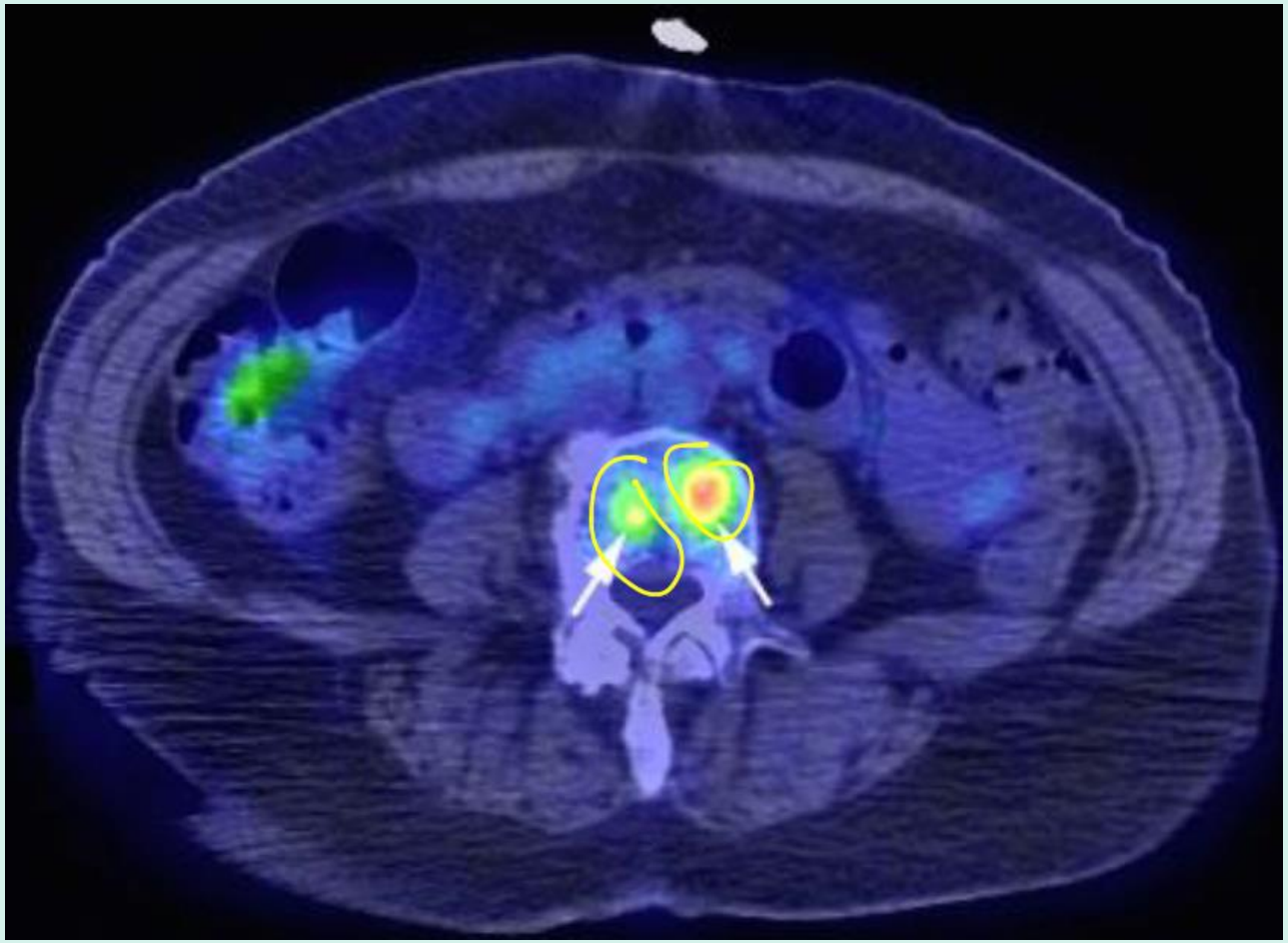


PET SCAN OF A PATIENT WITH LUNG CANCER



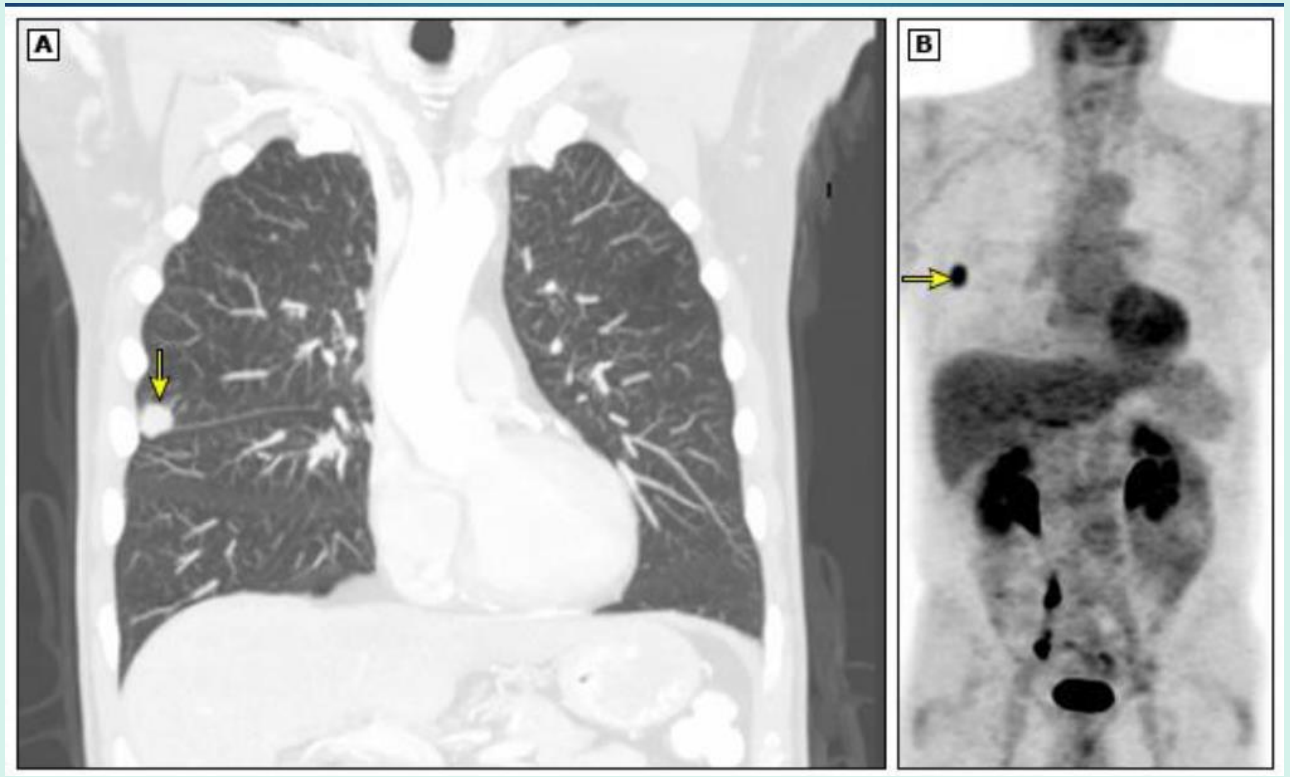
- This scan shows multiple liver metastatic lesions.

PET SCAN OF A PATIENT WITH LUNG CANCER

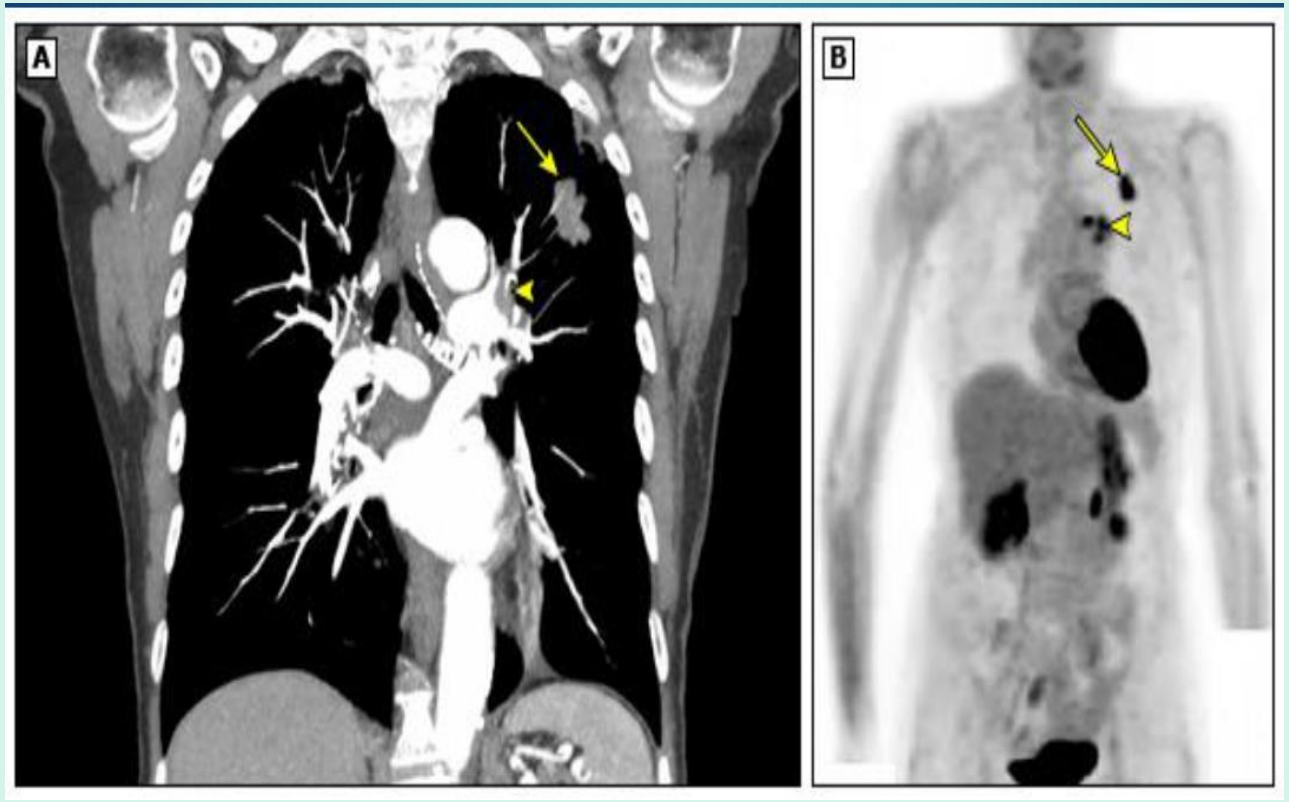


- This scan shows Bone metastatic lesions in a patient with Lung cancer.

INTEGRATED PET/CT SCAN

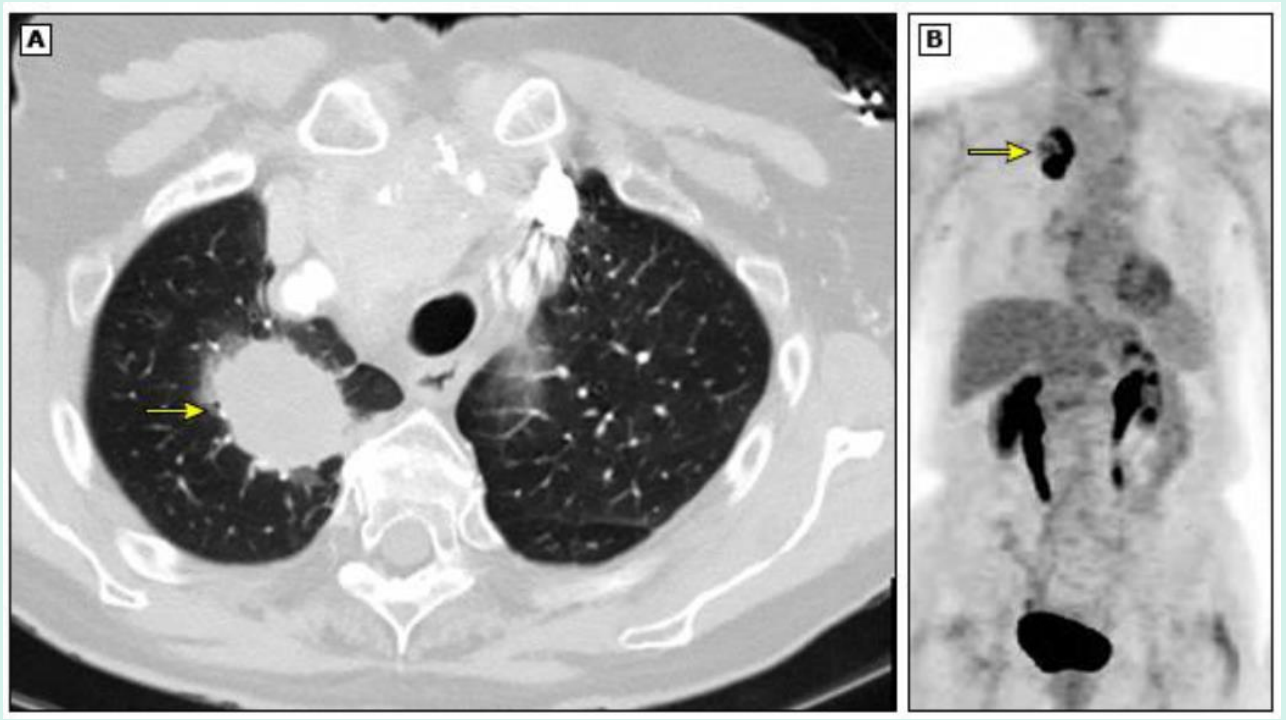


INTEGRATED PET/CT SCAN IN A PATIENT WITH LUNG CANCER



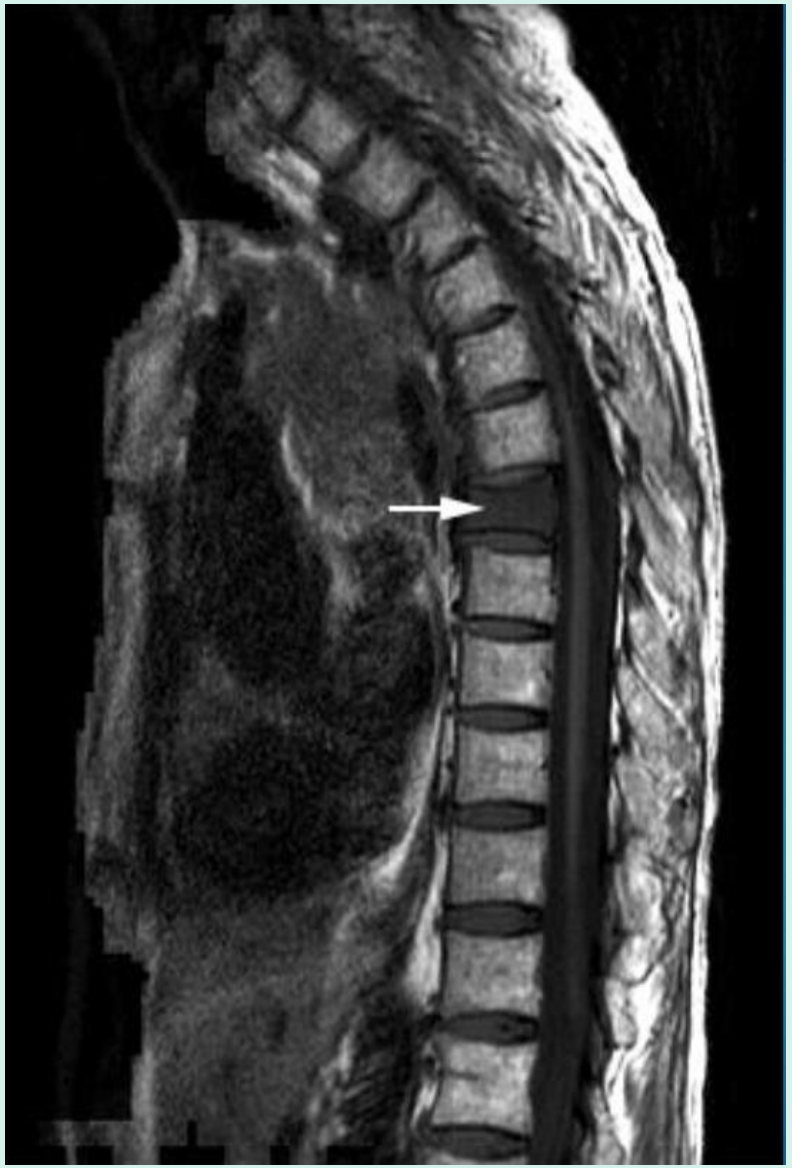
- This scan shows lung cancer with LN, Liver, and Adrenal involvement.

INTEGRATED PET/CT SCAN

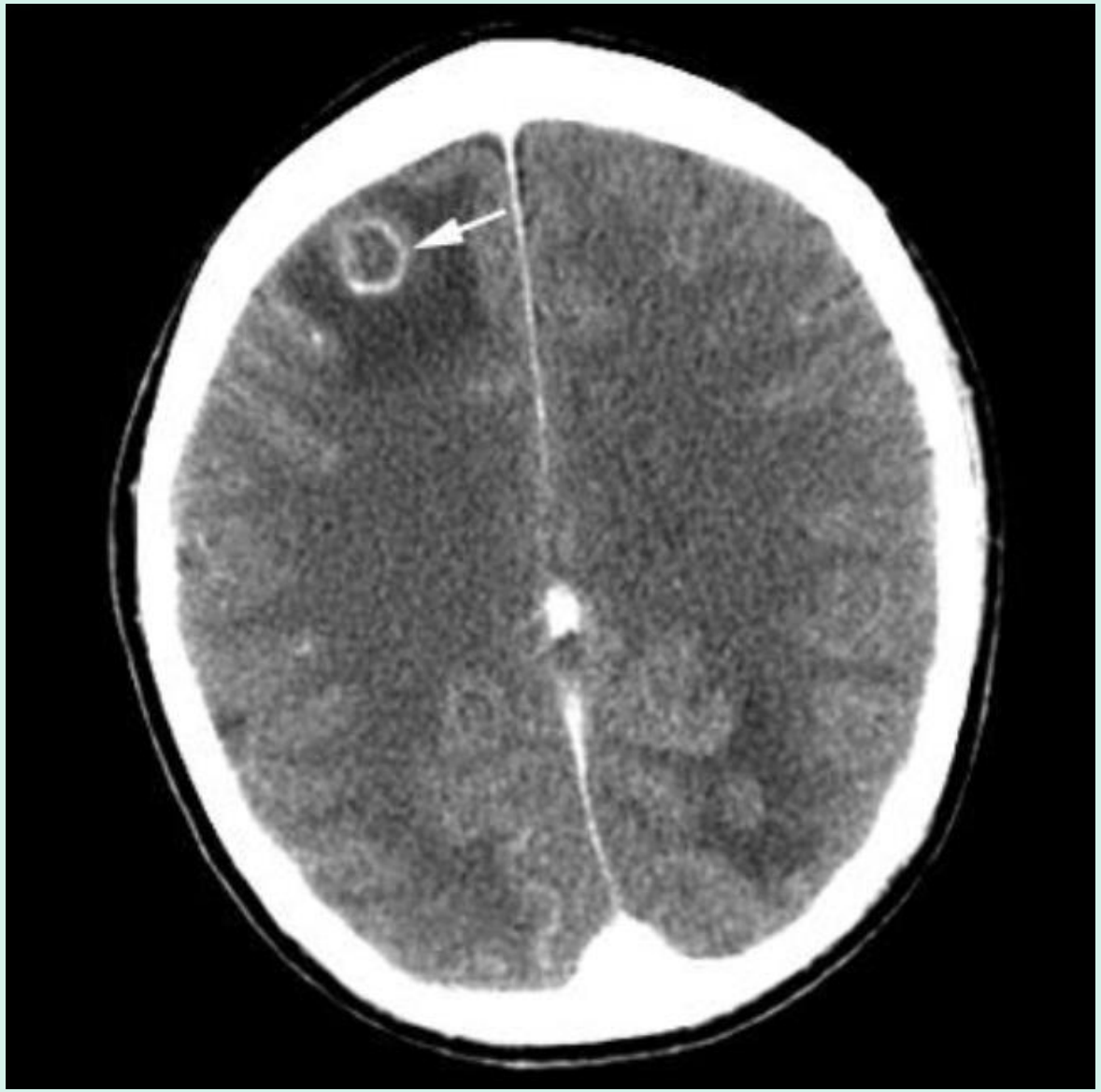


- The yellow arrow in the CT scan shows a Lung cancer, while in the PET scan it shows Suprarenal gland involvement.

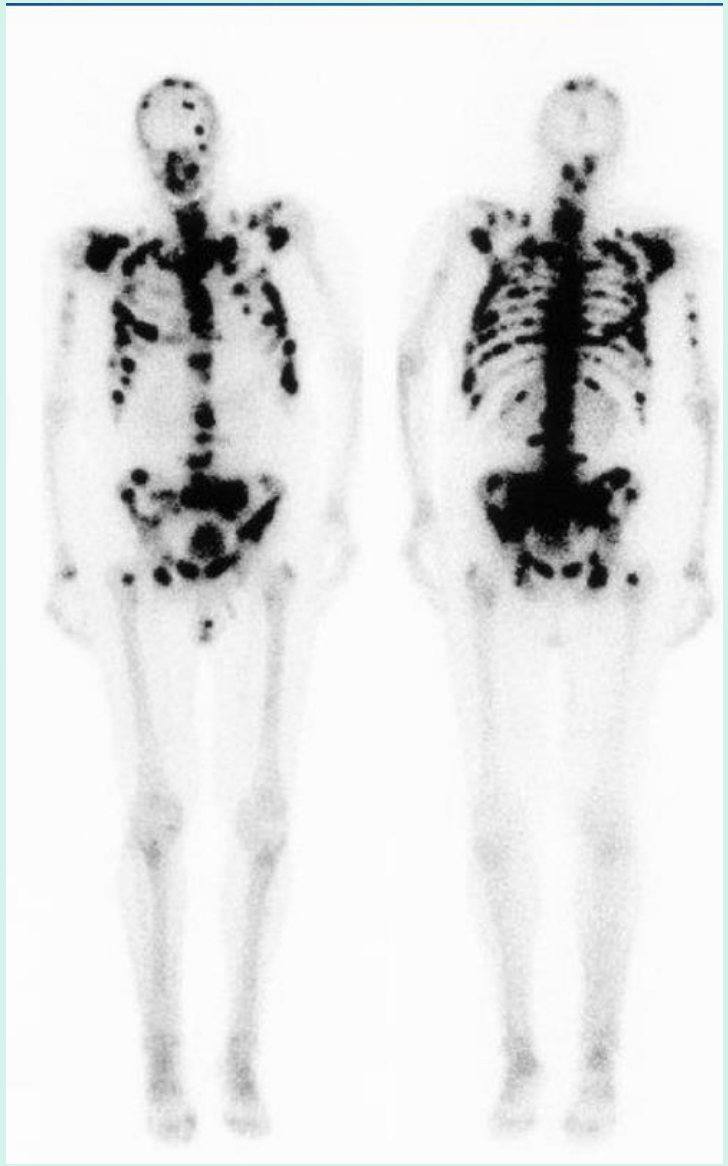
VERTEBRAL METASTASIS SEEN WITH MRI



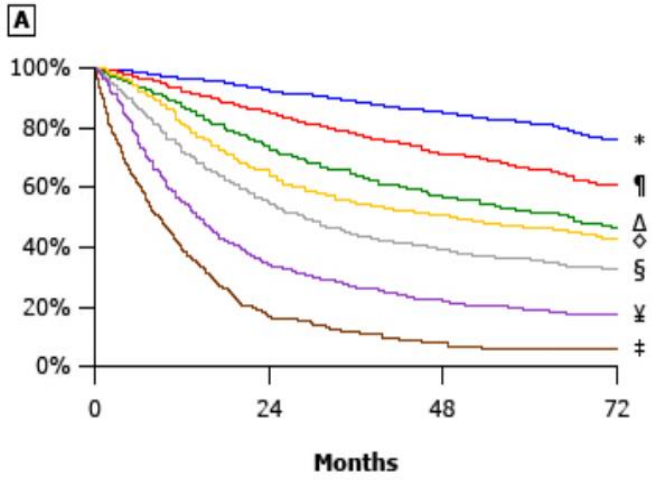
BRAIN METS ON MRI

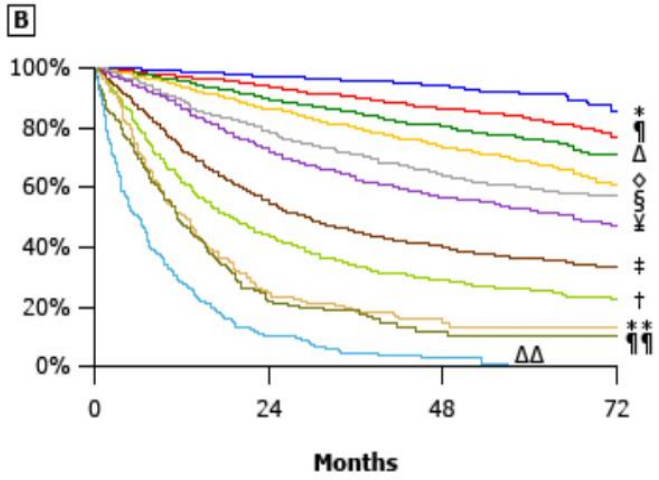


DIFFUSE BONE METS ON PET



PROGNOSIS OF SCLC





7th edition	Events / N	MST	24 month	60 month
* IA	1119 / 6303	NR	93%	82%
¶ IB	768 / 2492	NR	85%	66%
Δ IIA	424 / 1008	66.0	74%	52%
◇ IIB	382 / 824	49.0	64%	47%
§ IIIA	2139 / 3344	29.0	55%	36%
¥ IIIB	2101 / 2624	14.1	34%	19%
‡ IV	664 / 882	8.8	17%	6%

8th edition	Events / N	MST	24 month	60 month
* IA1	68 / 781	NR	97%	92%
¶ IA2	505 / 3105	NR	94%	83%
Δ IA3	546 / 2417	NR	90%	77%
◇ IB	560 / 1928	NR	87%	68%
§ IIA	215 / 585	NR	79%	60%
¥ IIB	605 / 1453	66.0	72%	53%
‡ IIIA	2052 / 3200	29.3	55%	36%
† IIIB	1551 / 2140	19.0	44%	26%
** IIIC	831 / 986	12.6	24%	13%
¶¶ IVA	336 / 484	11.5	23%	10%
ΔΔ IVB	328 / 398	6.0	10%	0%

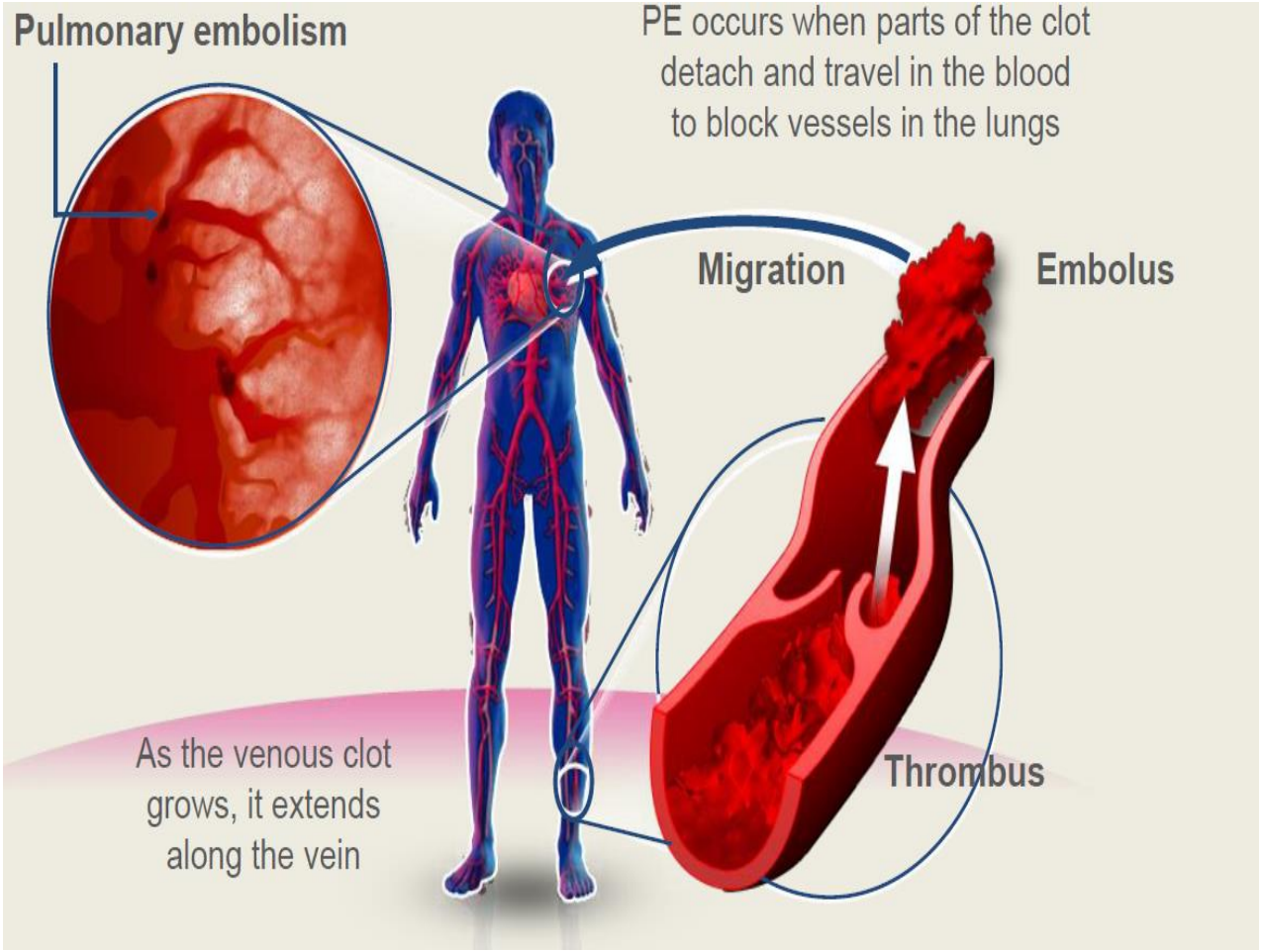
- The higher the stage, the worse the prognosis

PULMONARY EMBOLISM

PULMONARY EMBOLISM

- Partial or complete occlusion of a pulmonary arterial branch by blood clot (thrombus or multiple thrombi).
- DVT and PE are different presentations of the same underlying pathophysiological event, Venous Thromboembolism (VTE) → Hypercoagulable state (Vascular injury may cause a clot that dislodges from the lower limb and goes to the lung causing PE).
- Emboli Could be from a Thrombotic or Non-thrombotic sources.
- Thrombotic emboli account for most cases, it originates in the lower extremities and rarely from the lung itself, but it may happen.
- Most thrombi originate in the deep veins of the calf and propagate proximally to the popliteal vein → Femoral → Iliac → IVC → Lung.
- We can insert a filter in the IVC that forms a barrier stopping DVT from going to the lung.
- Emboli can also originate from atypical sites like upper extremity thrombosis associated with central venous catheters or intravascular cardiac devices or may be associated with thoracic outlet obstruction or effort thrombosis.

VTE: DVT AND PE



- Our aim is to prevent migration as the embolus ends up in the lungs.

CAUSES OF NON-THROMBOTIC PE

BOX 61-2 Causes of Nonthrombotic Pulmonary Emboli

Fat Embolism
Amniotic fluid embolism
Air Embolism
- Venous
- Arterial

Tumor embolism
Septic pulmonary embolism

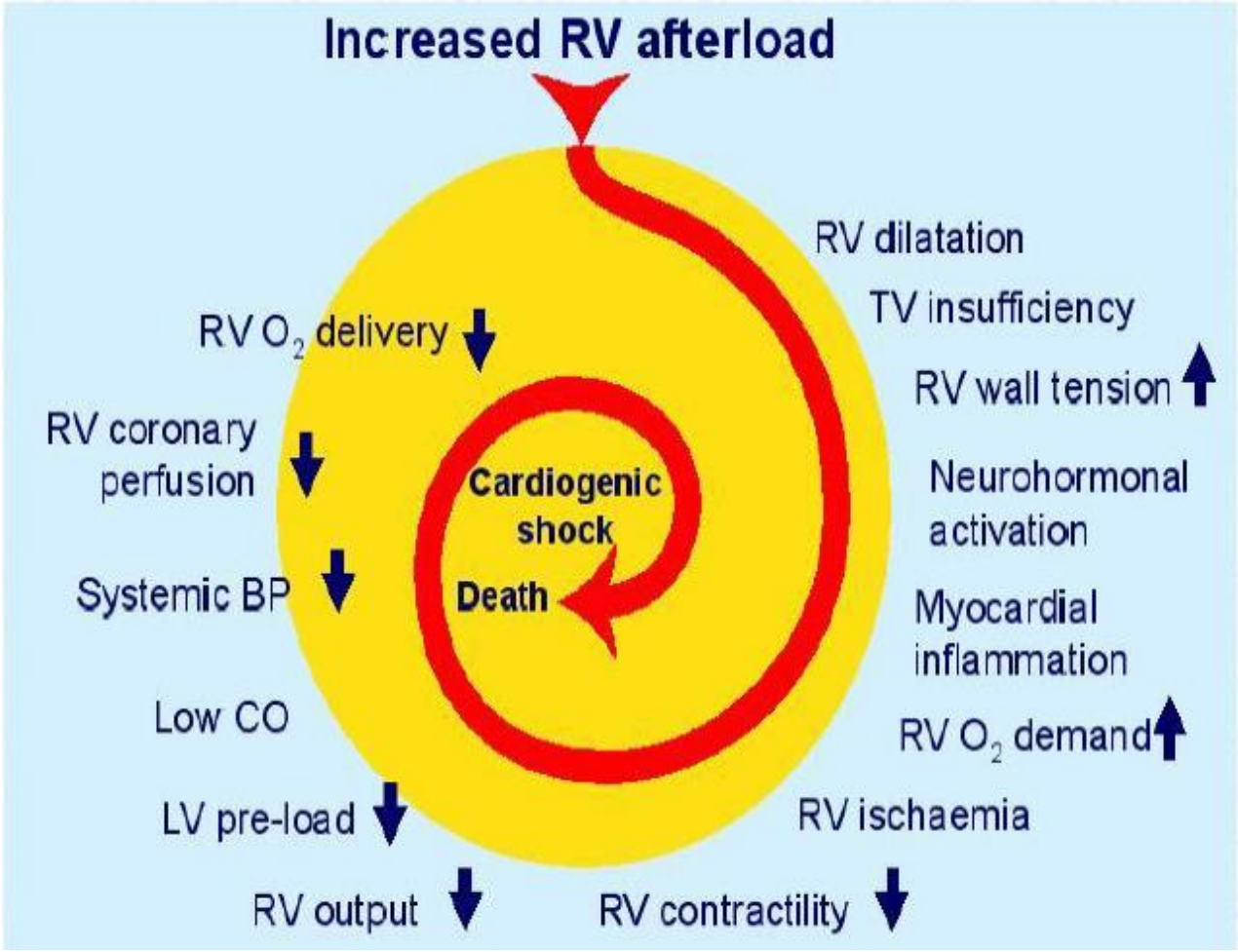
- Fat Embolism occurs mainly with fractures and the patients present with sudden hypoxia and SOB.
- Amniotic Fluid Embolism → in pregnant ladies.
- Septic Pulmonary Embolism → from infections.

PREDISPOSING FACTORS FOR VTE

Strong risk factors (OR>10)	Moderate risk factors (OR 2–9)
Fracture of lower limb	Arthroscopic knee surgery
Previous VTE	Autoimmune diseases
Spinal cord injury	Blood transfusion
Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months)	Central venous lines
Hip or knee replacement	Intravenous catheters and leads
Major trauma	Chemotherapy
Myocardial infarction (within previous 3 months)	Congestive heart failure or respiratory failure
	Erythropoiesis-stimulating agents
	Hormone replacement therapy (depends on formulation)

Moderate risk factors (cont'd)	Weak risk factors (OR<2)
In vitro fertilization	Bed rest >3 days
Oral contraceptive therapy	Diabetes mellitus
Postpartum period	Arterial hypertension
Infection (specifically pneumonia, urinary tract infection, and HIV)	Immobility due to sitting (e.g. prolonged car or air travel)
Inflammatory bowel disease	Increasing age
Cancer (highest risk in metastatic disease)	Laparoscopic surgery (e.g. cholecystectomy)
Paralytic stroke	Obesity
Superficial vein thrombosis	Pregnancy
Thrombophilia	Varicose veins

HEMODYNAMIC CONSEQUENCES

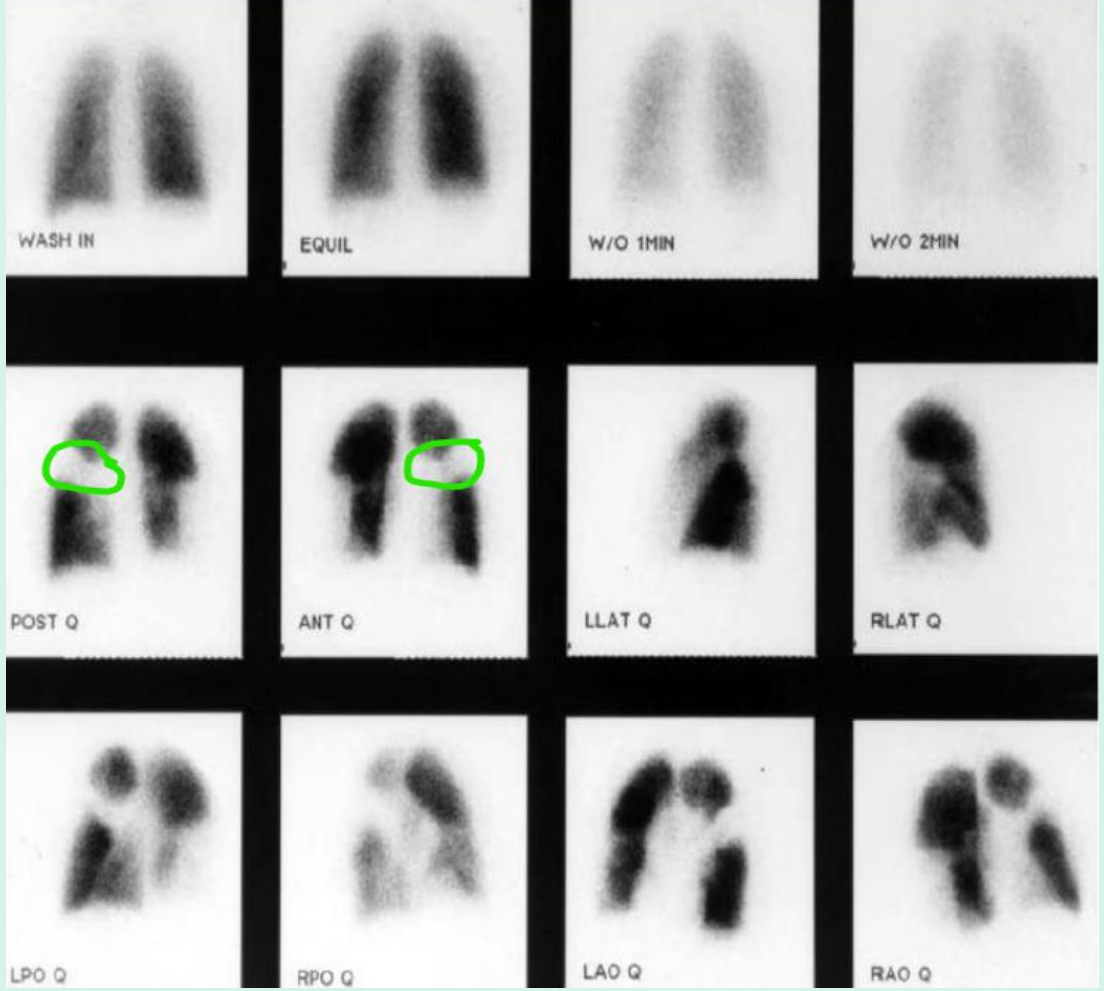


CLASSIFICATION OF PE BASED ON EARLY MORTALITY RISK

Early mortality risk		Indicators of risk			
		Haemo-dynamic instability	Clinical parameters of PE severity/ comorbidity: PESI III–V or sPESI≥1	RV dysfunction on TTE or CTPA	Elevated cardiac troponin levels
High		+	(+)	+	(+)
Interme-diate	Intermediate–high	-	+	+	+
	Intermediate–low	-	+	One (or none) positive	
Low		-	-	-	Assessment optional; if assessed, negative

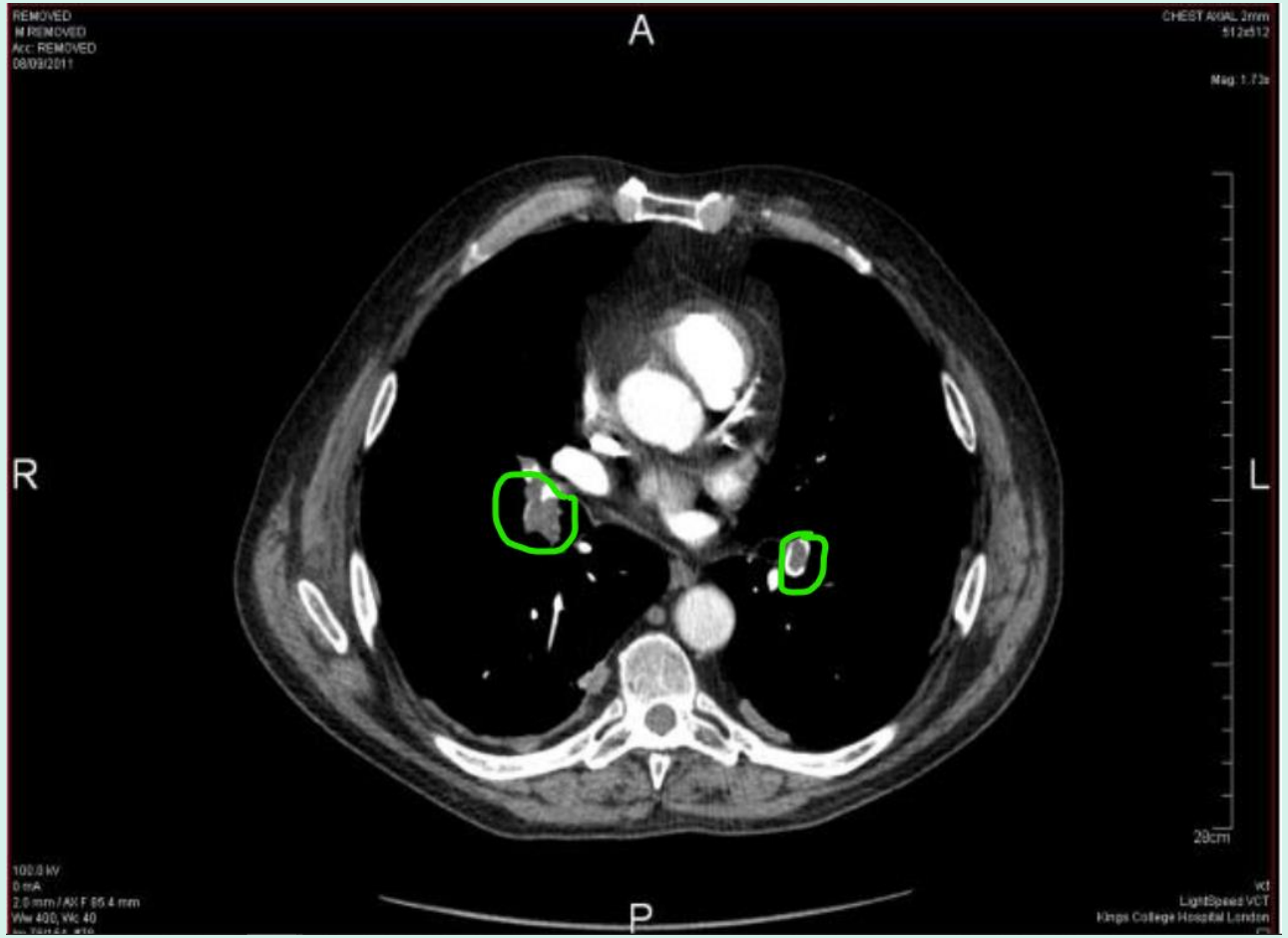
- Any patient with PE especially with Hypotension should have Troponin level test to rule out RV dysfunction (in patients with Right heart failure, the right side is larger than the left on imaging).
- Patients with low risk should be discharged to home with oral anticoagulants.

V/Q SCAN



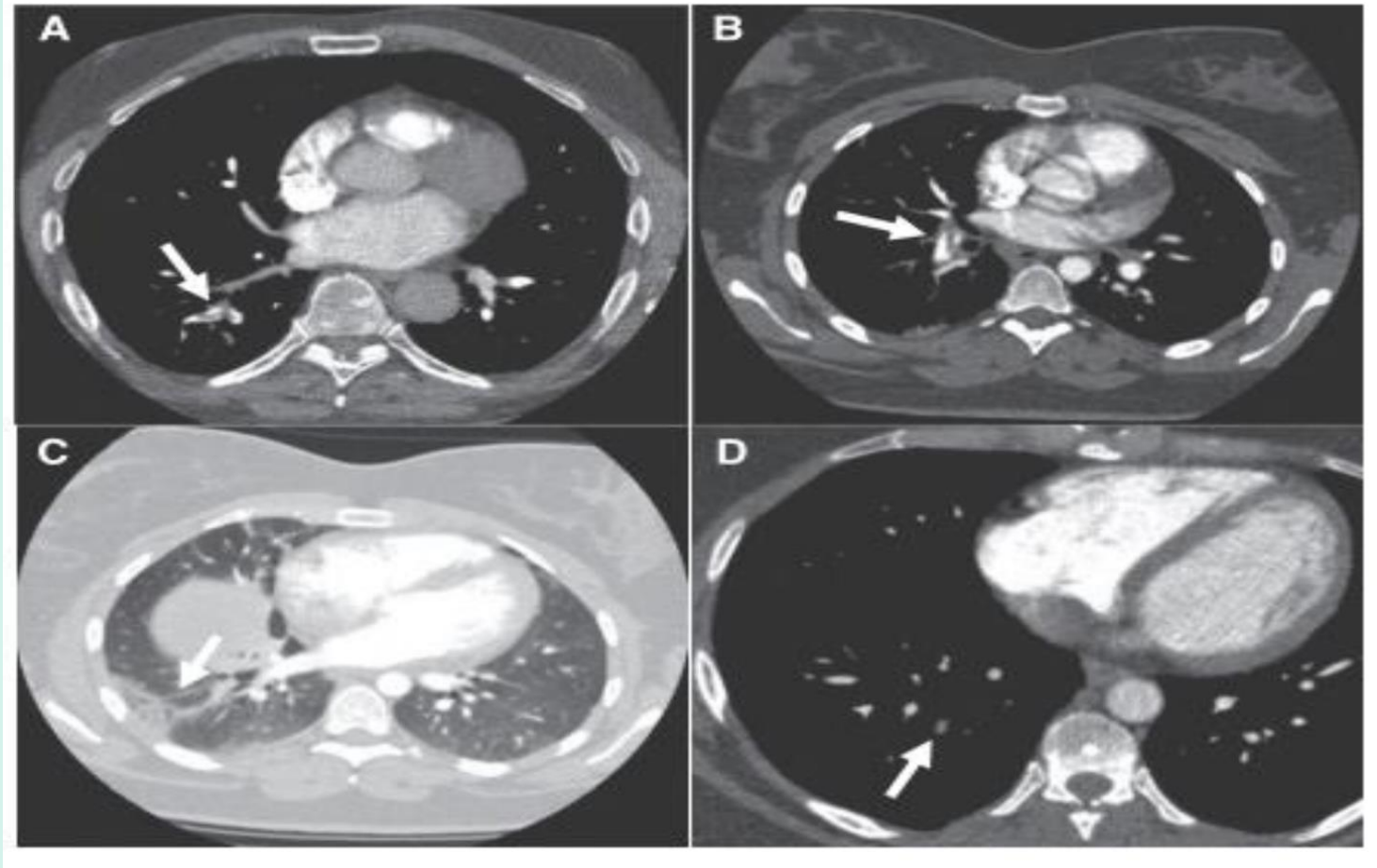
- The first row is the Ventilation scan
- The second row is the Perfusion scan
- The areas encircled with green circles are perfused areas but not ventilated → Mismatch indicating PE.

CT SCAN WITH CONTRAST OF A PATIENT WITH PE



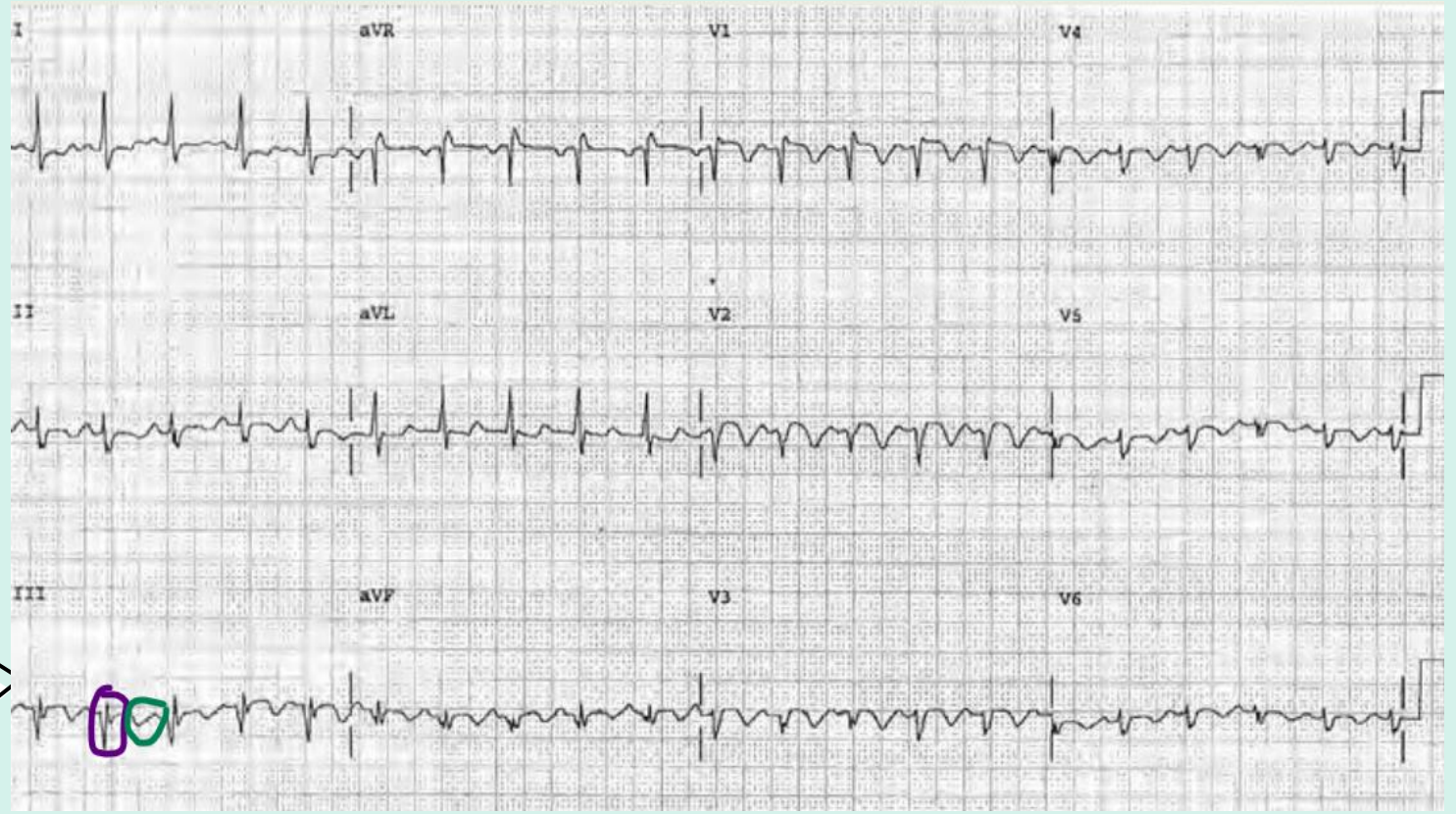
- The encircled structures are pulmonary arteries showing filling defects.
- Filling Defect: the clot is gray, and contrast is white.

CT SCAN OF A PATIENT WITH PE



- A → Clot
- B → Filling Defect
- C → Pulmonary infarction

ECG OF A PATIENT WITH PE



- ECGs are not diagnostic for PE, but it only gives hints.
- The most common ECG finding here is Sinus Tachycardia.
- Other findings: Incomplete RBBB, S1Q3T3 pattern, and Inverted precordial T waves.
- Notice the presence of the pathologic Q wave + T wave inversion

RESPIRATORY FAILURE

RESPIRATORY FAILURE

Respiratory dysfunction refers to the failure of gas exchange, i.e. decrease in arterial oxygen tension PaO2 (<60 mm Hg -Hypoxemia-)

It may or may not accompany hypercapnia, a PaCO2 >50 mm Hg (decreased CO2 elimination).

Any Lung disease can cause Respiratory failure, and all patients with RF must have an ABG test (PaO2 <60 mm Hg, O2 Sat <88%, PaCO2 >50 mm Hg).

Type 1 RF (Hyper-hypoxic): Arterial oxygen tension PaO2 <60 mm Hg with normal or low Arterial carbon dioxide tension PaCO2 (PaCO2 is equal to or less than 50 mm Hg).

Type 2 RF (Hypercapnic): Hypercapnic Respiratory failure characterized by a PaCO2 >50 mm Hg and Arterial oxygen tension PaO2 <60 mm Hg.

RF is further classified into Acute or Chronic.

Acute RF: characterized by life threatening derangements in ABGs and Acid-Base status.

Acute Type 2 RF develops over minutes to hours; therefore, pH is <7.3, and patient exhibits severe acidemia especially with decreased Level of consciousness.

In Acute Type 1 RF, patients are severely tachypneic and Toxic.

RESPIRATORY FAILURE

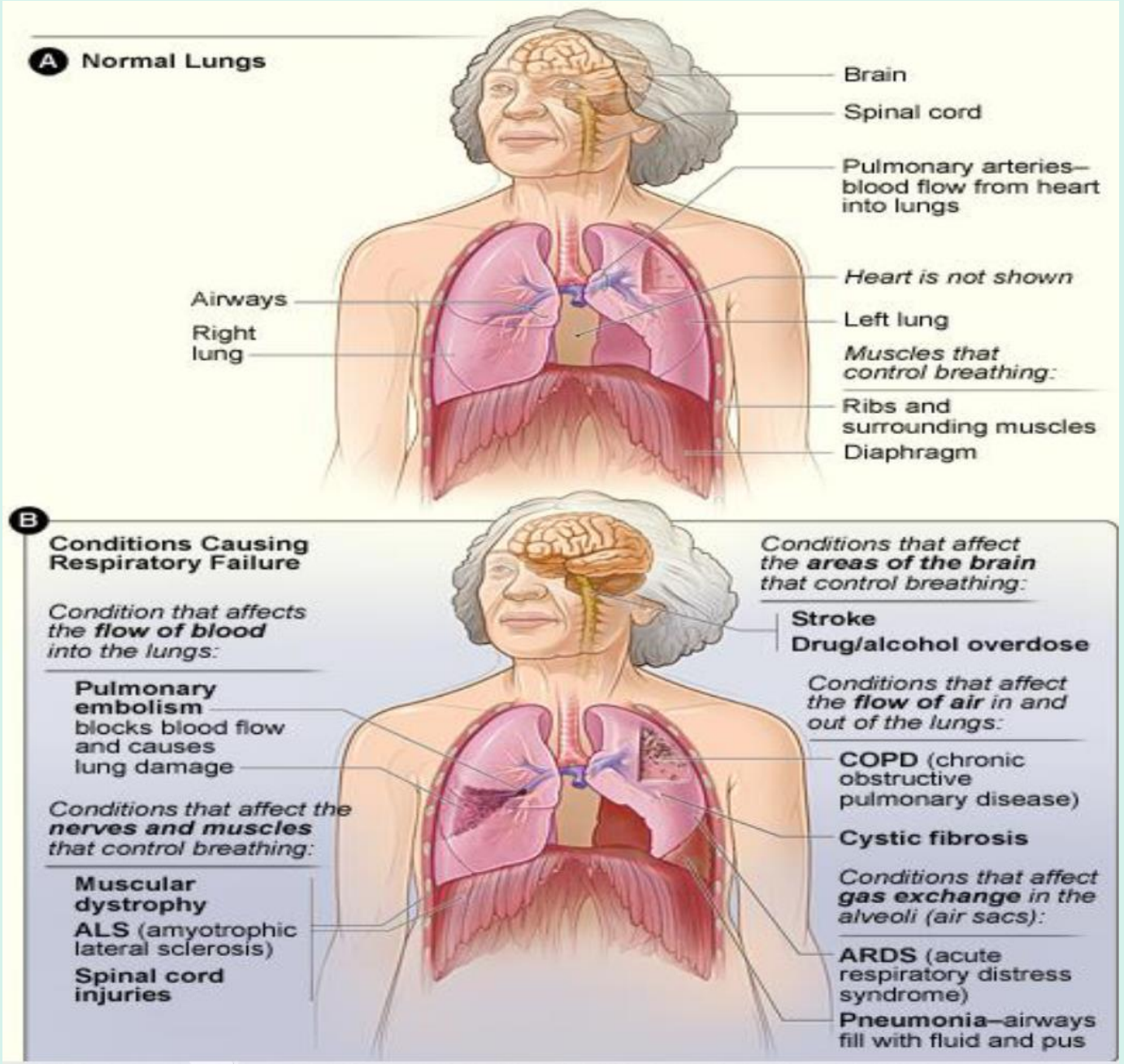
Chronic RF is less dramatic, since the patient get used to Hypoxia.

Develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH usually is only slightly decreased.

The clinical markers of chronic hypoxemia, such as Polycythemia (as a response to chronic hypoxemia, to compensate for hypoxia) or Cor-pulmonale (due to pulmonary HTN - vasoconstriction due to hypoxemia- leading to Rt sided heart failure) suggest a long-standing disorder.

In COPD → Long term O2 therapy improve survival in patient with COPD by preventing Vasoconstriction and Pulmonary HTN and Cor-pulmonale.

PATHOPHYSIOLOGY OF HYPOXEMIA



CAUSES OF HYPOXEMIA

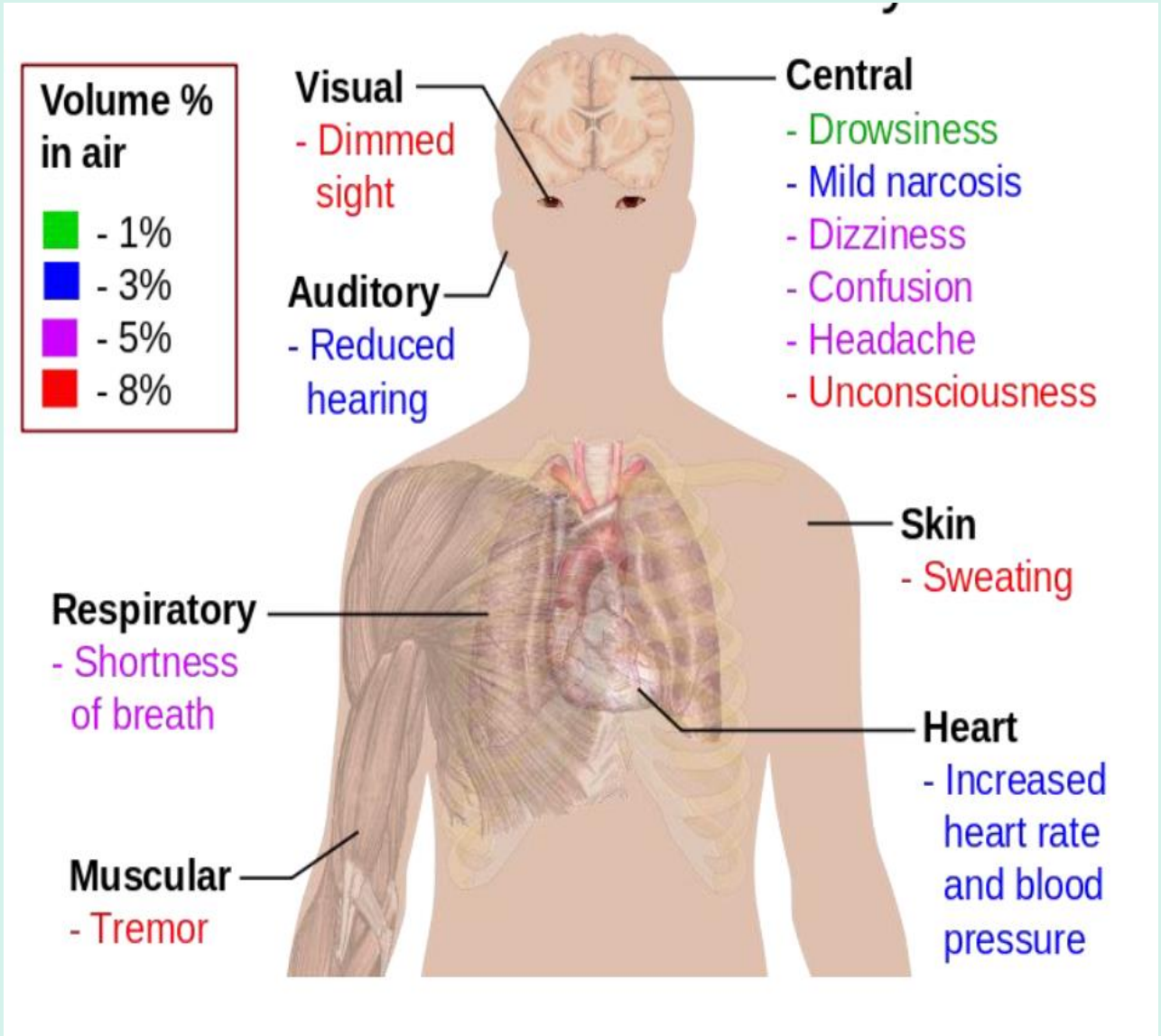
Cause	PaO_2	A-a gradient	PaO_2 response to supplemental oxygen
Hypoventilation	Decreased	Normal	Increases
Diffusion Impairment	Decreased	Increased	Increases
Shunt	Decreased	Increased	Does not increase.
V/Q Mismatch	Decreased	Increased	Usually increases (depends on V/Q mismatch type)
High Altitude	Decreased	Normal	Increases

- To differentiate between high altitude and hypoventilation hypoxemia, Check PaCO2:

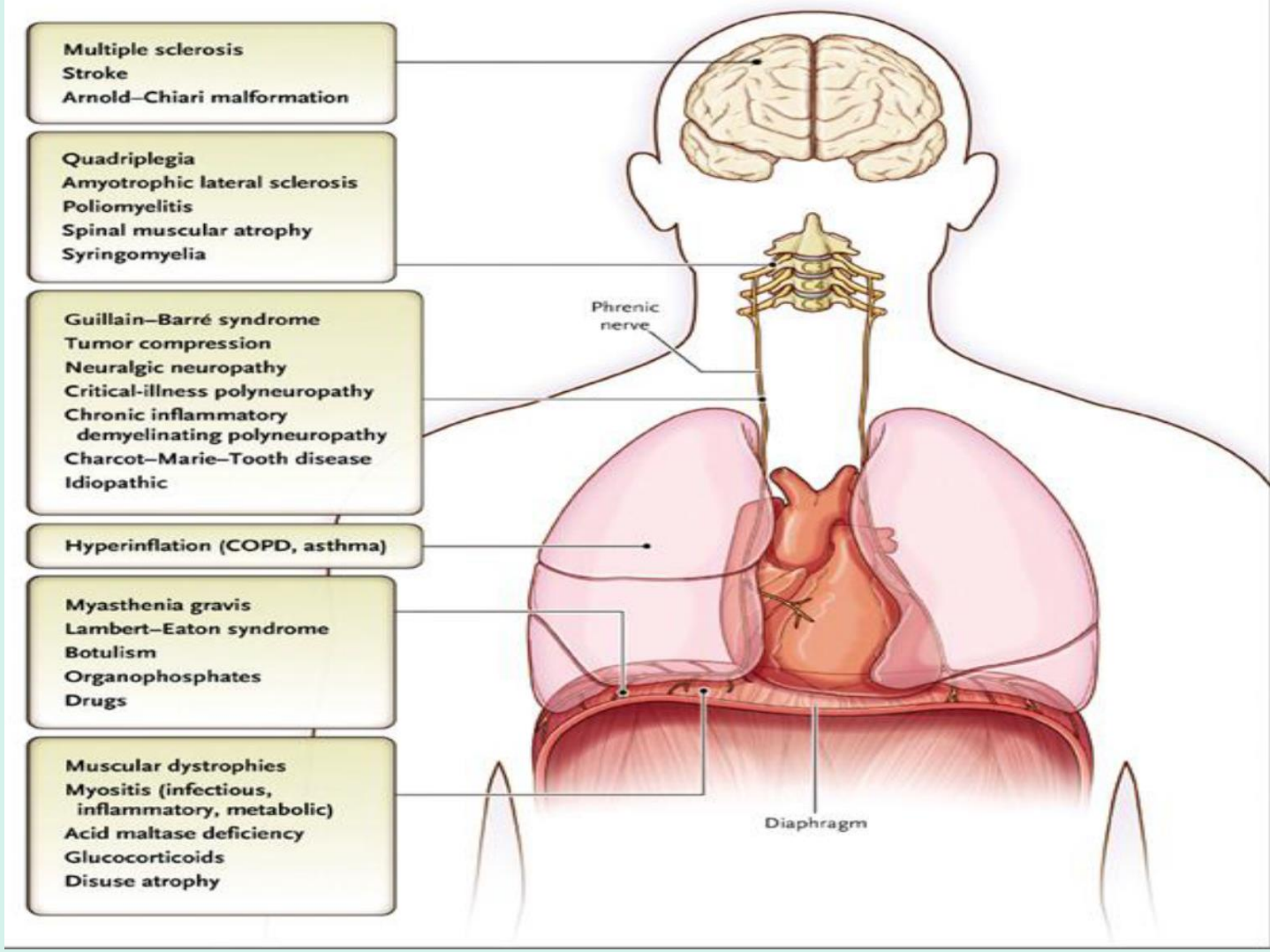
If PaCO2 is decreased → High Altitude Hypoxemia.

If PaCO2 is increased → Hypoventilation Hypoxemia.

MAIN SYMPTOMS OF CO2 TOXICITY



CAUSES OF TYPE 2 RF



- All of the above causes lead to Hypoventilation

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

A rapidly progressive non-cardiogenic pulmonary edema (confirmed by normal capillary wedge pressure) that initially manifests as Dyspnea, Tachypnea, and Hypoxemia, then quickly evolves into RF.

It's a very common cause of mortality in ICUs.

It occurs due to Infection, Inflammation, and Fluid accumulation.

Mechanism of Hypoxia in ARDS: Shunting due to poor ventilation but well perfusion.

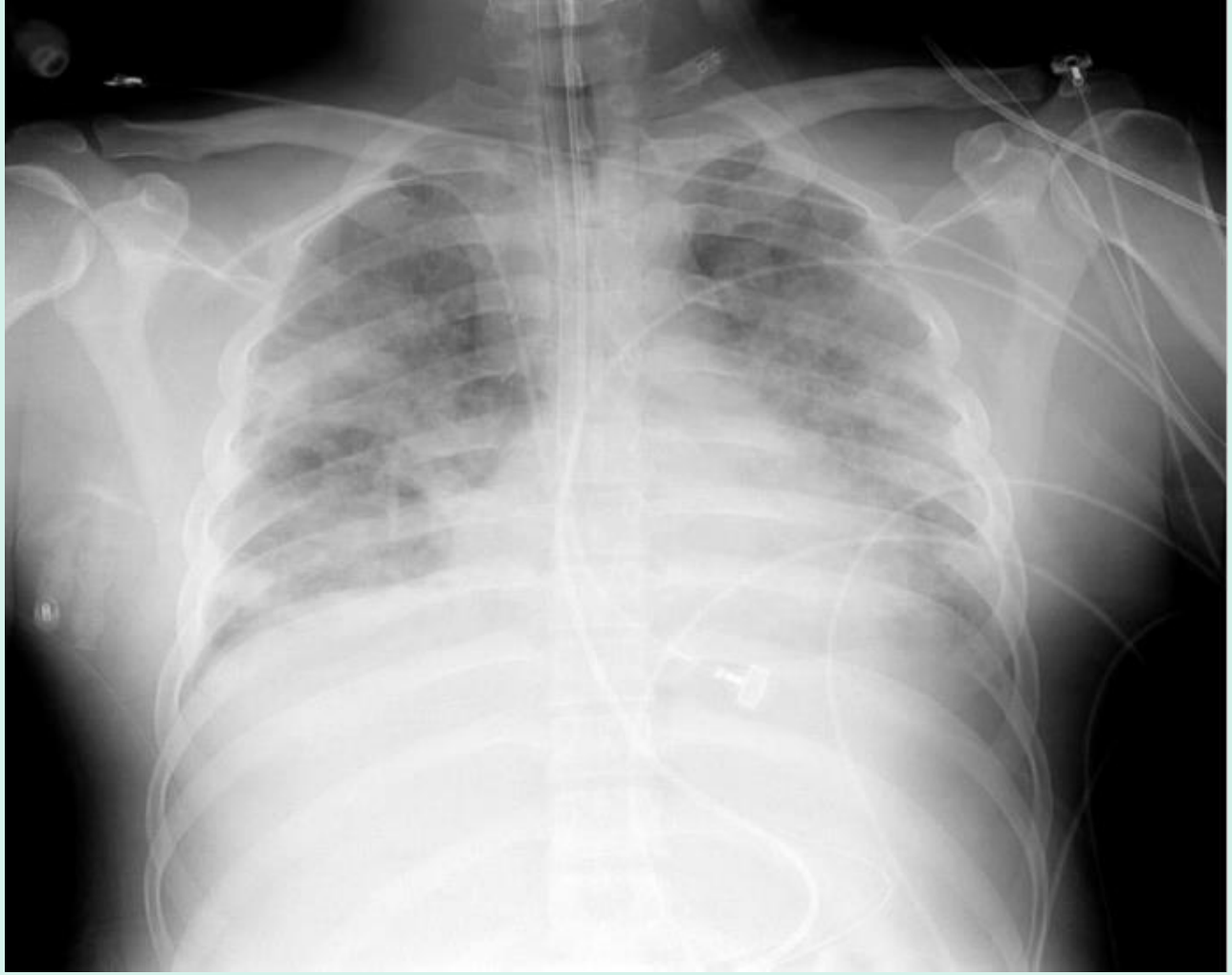
The criteria for the diagnosis are based on timing of symptom onset (within one week of known clinical insult or new or worsening respiratory symptoms):

1- Bilateral opacities on chest imaging that aren't fully explained by effusions, lobar or lung collapse, or nodules.

2- The likely source of pulmonary edema (RF not fully explained by cardiac failure of fluid overload).

3- Oxygenation as measured by the ratio of Partial pressure of arterial oxygen (PaO2) to fraction of inspired oxygen (FiO2) - normally PaO2 = 90 mm Hg, FiO2 = 0.21, Oxygenation Ratio= 450 mm Hg-

CXR OF A PATIENT WITH ARDS



- Note the bilateral air space opacification and lack of obvious vascular congestion.
- Typical CXR findings in ARDS:

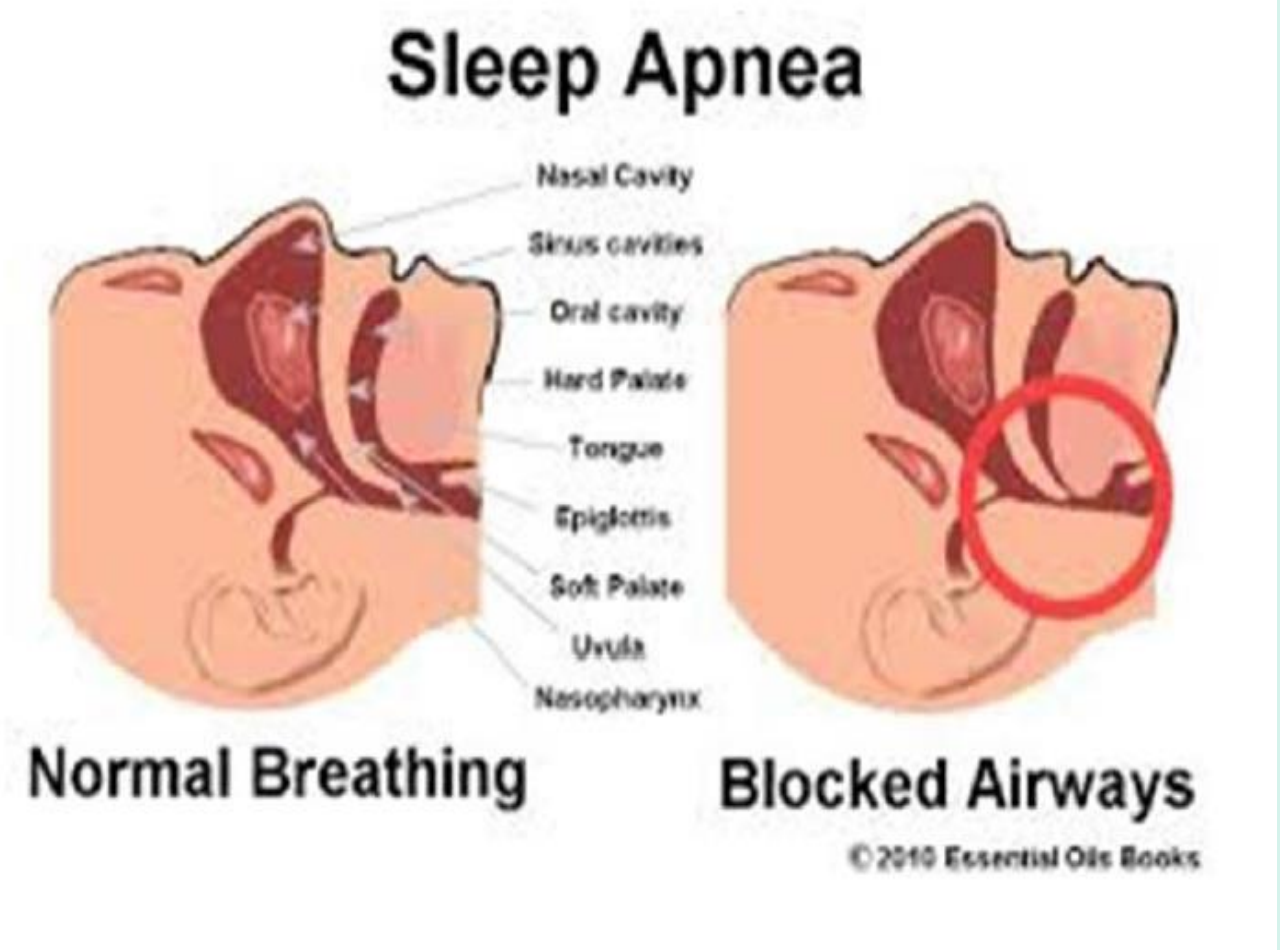
1- Right lung's mid and lower zones have white opacities and the Left side shows infiltrations.

2- Heart size is normal (non-cardiogenic).

OBSTRUCTIVE SLEEP APNEA / OBESITY HYPOVENTILATION SYNDROME

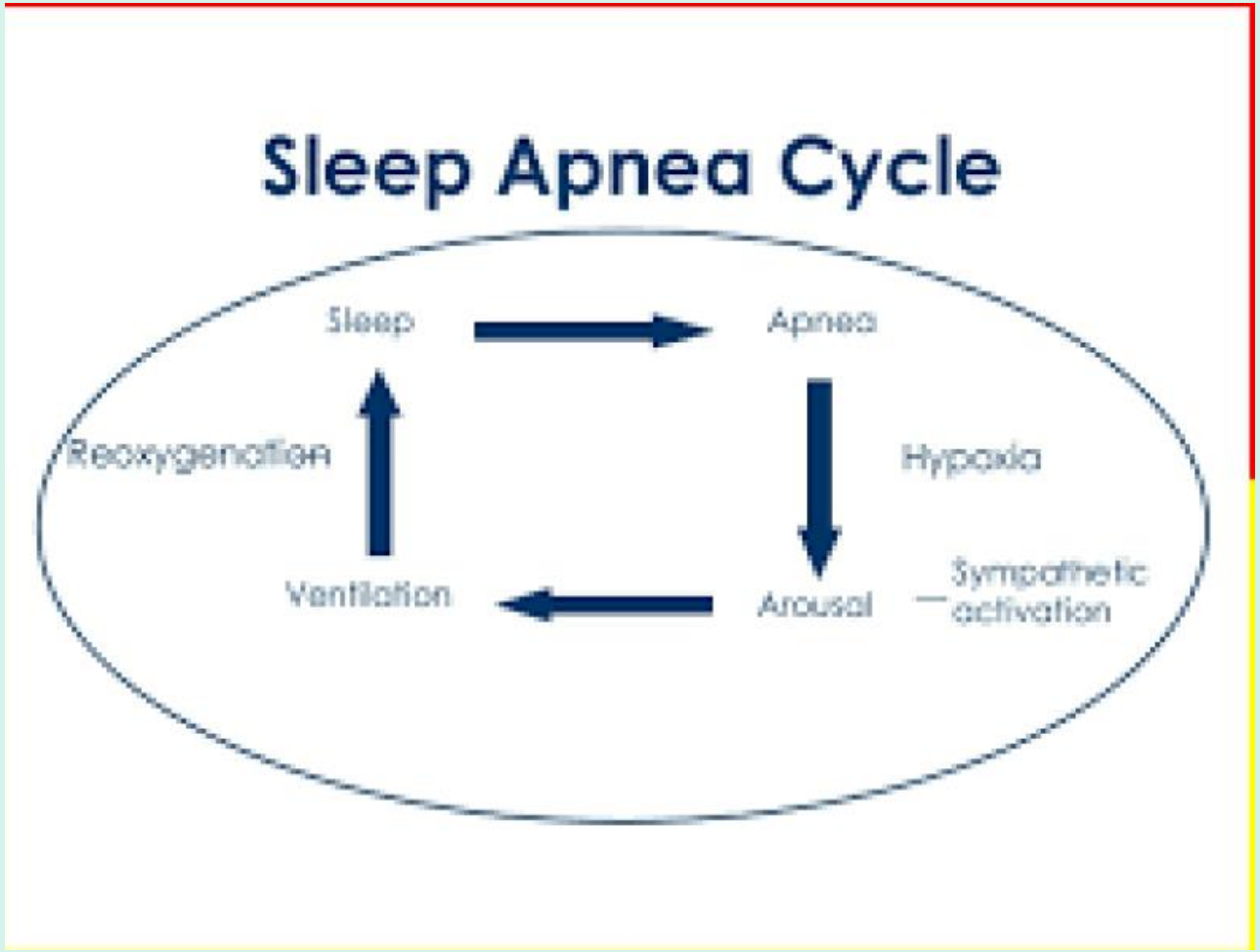
- OSA is characterized by repeated upper airway obstruction (at the level of the oropharynx behind the tongue) during sleep causing acute disruptions to blood oxygen levels, heart rate, blood pressure, intrathoracic pressure, and sleep quality (Drop in O2 levels leading to Tachycardia, Tachypnea, Increased intrathoracic pressure, and sleep quality is disturbed).

SLEEP APNEA



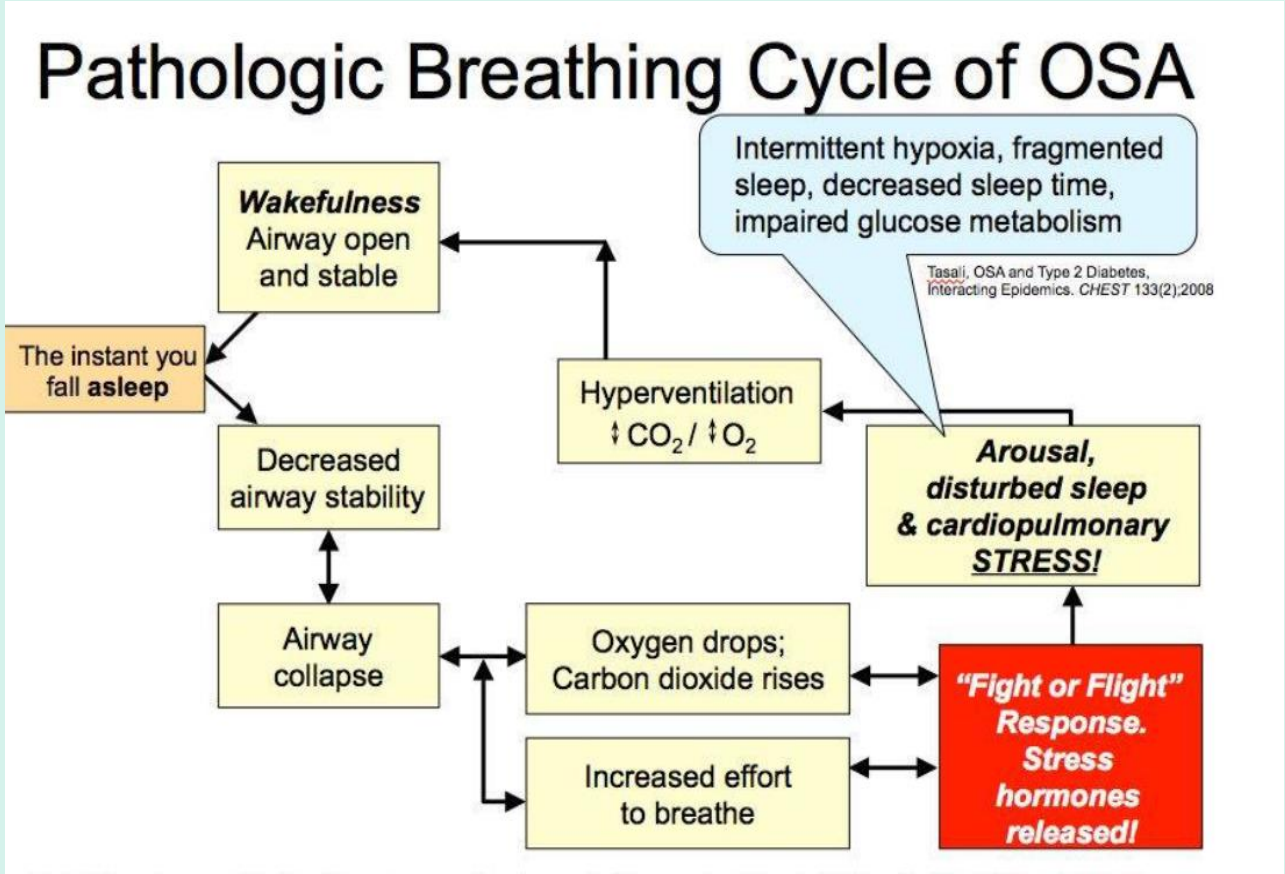
- Normally during sleep, muscles of the oropharynx and nasopharynx are pulled apart allowing breathing.
- In the case of OSA, the airways become blocked only during sleep due to muscle weakness (most importantly the tongue), aging, etc.

SLEEP APNEA CYCLE



- Sleep → Cessation of breath → Hypoxia → Arousal due to sympathetic activation against hypoxia → Ventilation → Reoxygenation → Sleep.

HEALTH EFFECTS OF OSA



- Some patients may not be aware of arousals during sleep (mild arousals).
- Signs of apnea that may awaken the patient from sleep:

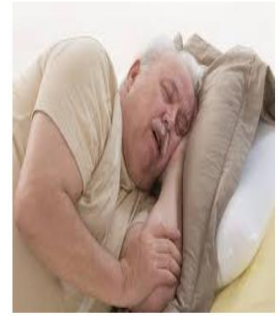
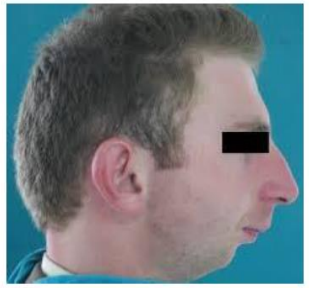
1- Feeling suffocated.

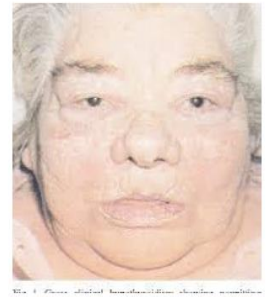
2- For frequent urination (due to the release of ANP).

3- Not knowing why they wake up.

RISK FACTORS FOR OSA

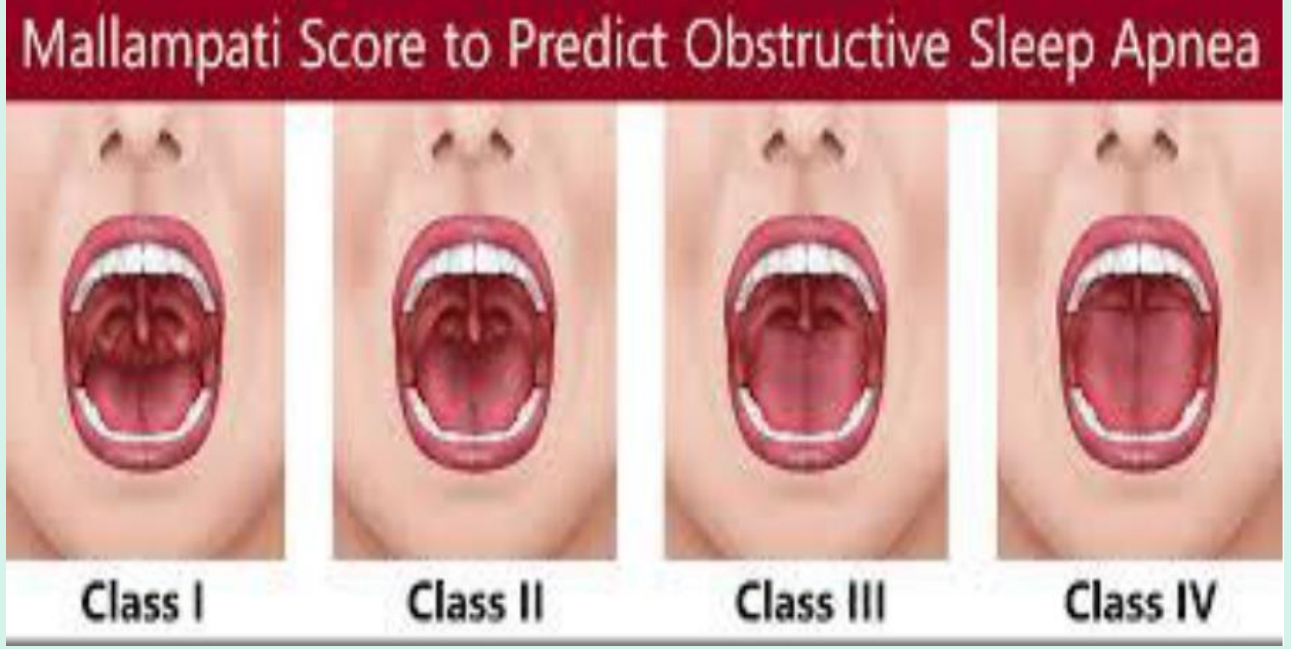


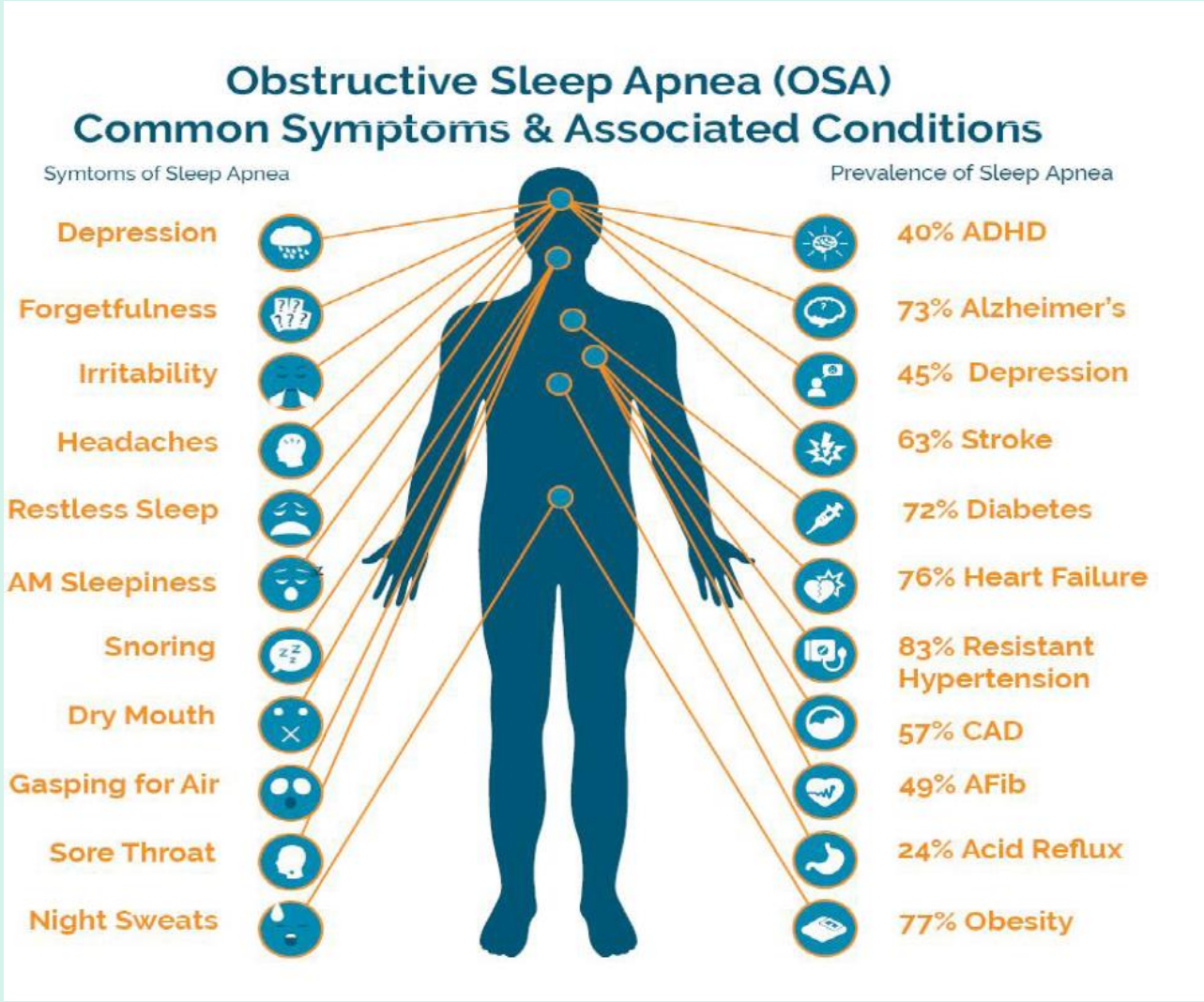
- Obesity (main risk factor), Being a mouth breather during sleep (may be due to adenoid or tonsillar enlargement), Alcohol (relaxes upper airway muscles), Smoking (weakens the respiratory muscles), Male gender, Age >50, Jaw structure (small), Menopause, Drugs (sedatives + anti-epileptics), Hypothyroidism, and Acromegaly.

MALLAMPATI SCORE

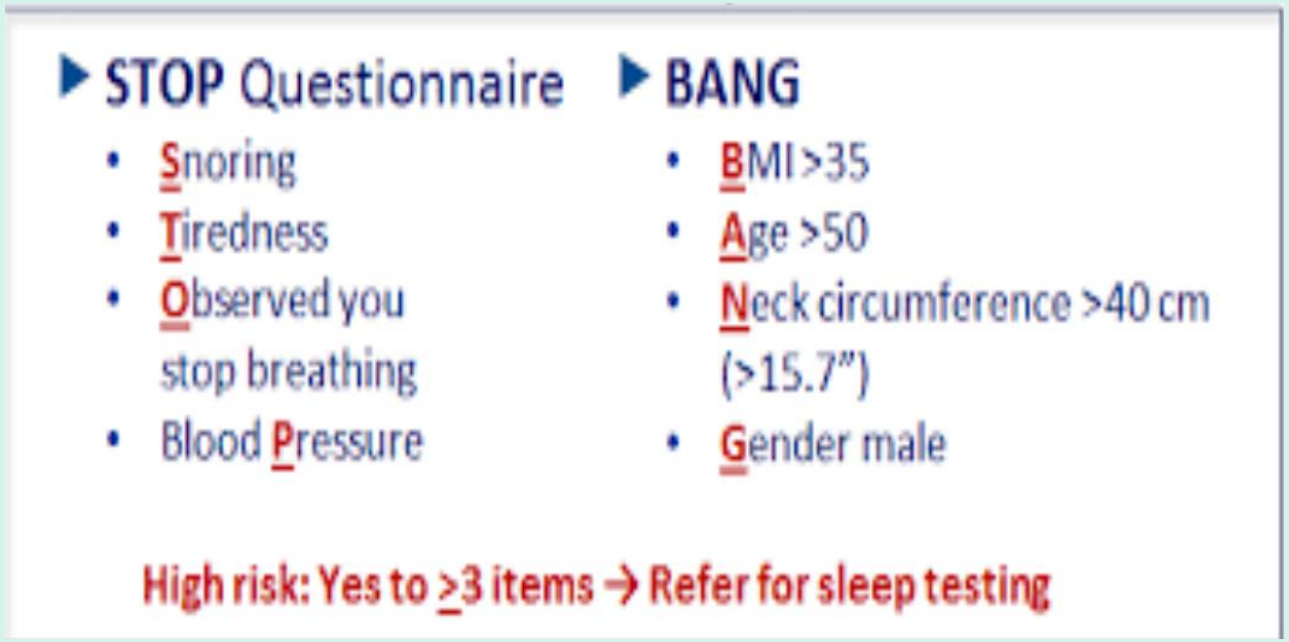


- The Mallampati score is a simple test that can be a good predictor of OSA. The assessment is performed with the patient sitting up straight, mouth open and tongue maximally protruded, without speaking or saying "ahhhh".
- Class 0: Ability to see any part of the epiglottis upon mouth opening and tongue protrusion.
- Class I: Soft palate, Fauces, Uvula, Pillars are all visible.
- Class II: Soft palate, Fauces, Uvula are visible.
- Class III: Soft palate, base of Uvula are visible.
- Class IV: Soft palate not visible at all.
- Class III and IV are the worst, and patients are at most risk of developing OSA.

COMMON SYMPTOMS AND ASSOCIATED CONDITIONS



STOP BANG QUESTIONNAIRE



- Used to assess the risk of sleep apnea, mainly pre-operatively.

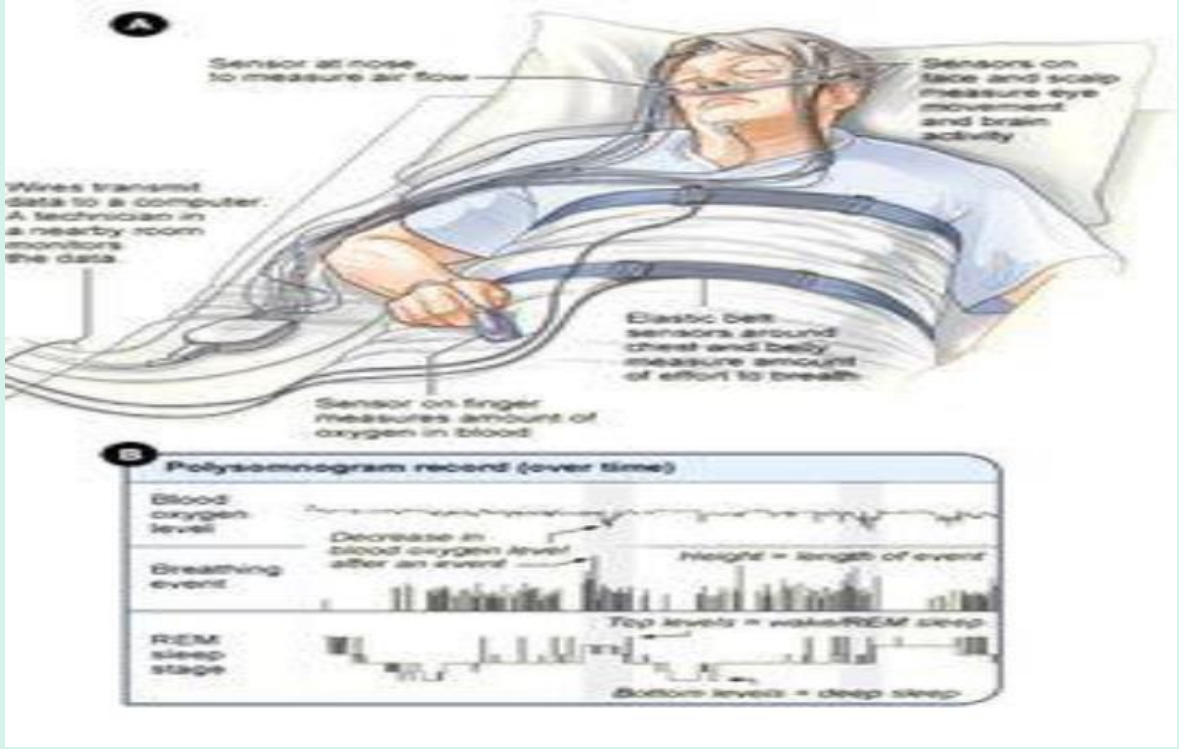
DIAGNOSIS OF OSA

Polysymnography (PSG) → Gold Standard.

Home sleep test.

Oximetry

PSG
VERY IMPORTANT TOPIC - TRY TO UNDERSTAND IT VERY WELL -

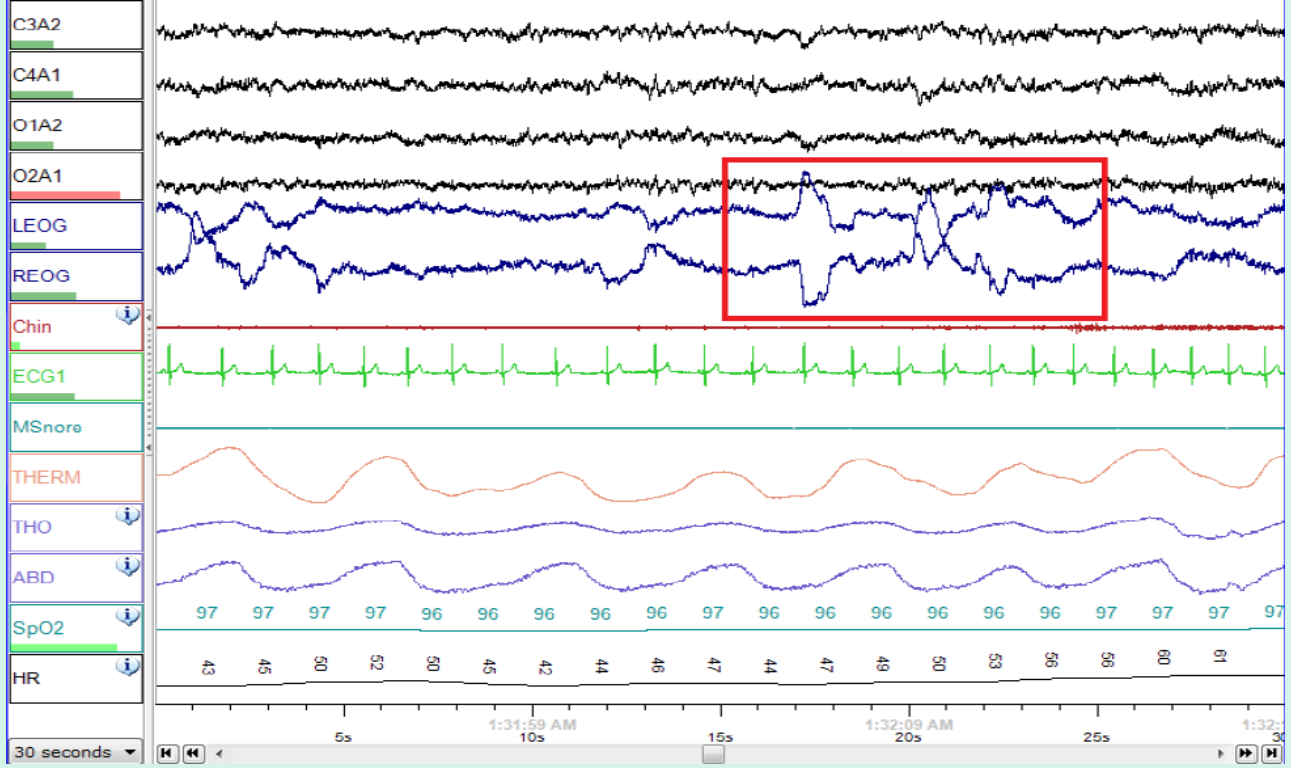


- Components:

* Electrodes on the head for EEG; to determine Sleep stages.
* Chin Microphone; for snoring.
* Nasal Canula; for the flow of air.
* Belts around the chest and abdomen; to detect their movements during breaths.

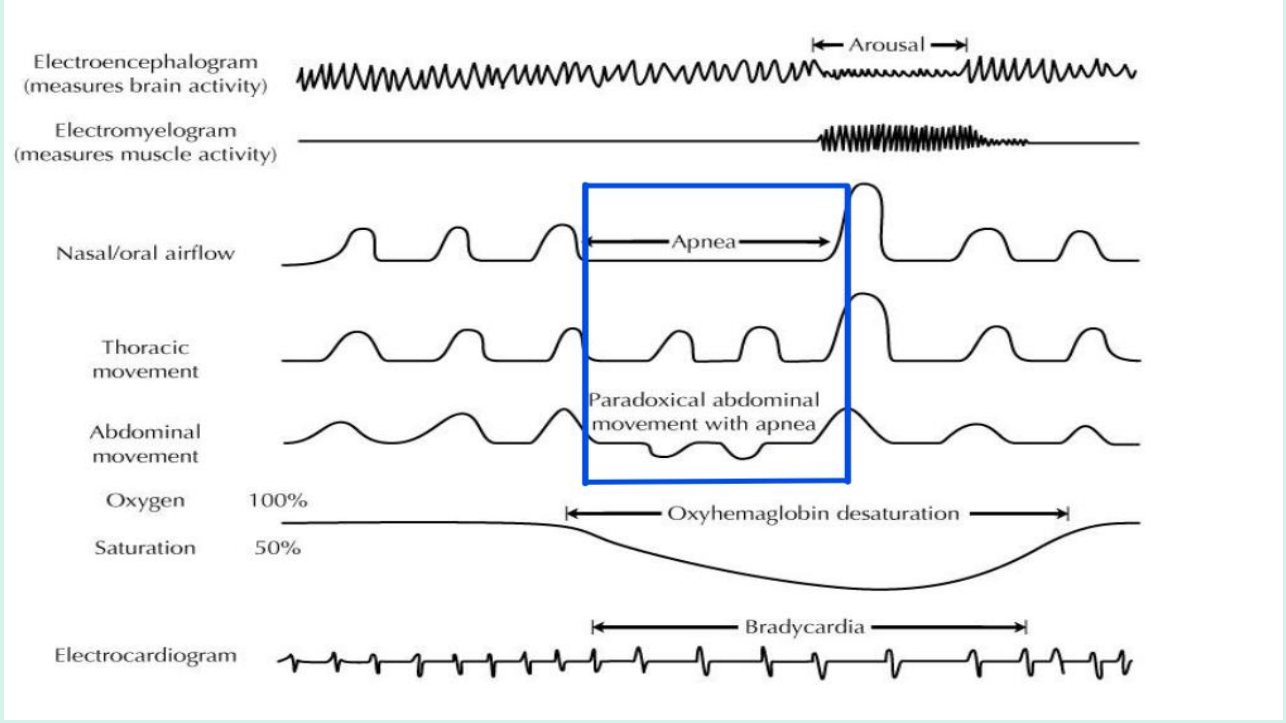
- Patients must not Drink coffee, take a nap at afternoon, or smoke.
- Patients can take medications to sleep → Benzodiazepine.

PSG



- C3A2, C4A1, O1A2, O2A1: EEG leads, each letter stands for where you put the electrode (C → central, O → Occipital, F → Frontal).
- LEOG, REOG: Rt and Lt eye electrogram → used to detect when the patient enters REM and Non-REM sleep.
- Chin: movement of Chin muscles.
- MSnore: shows snoring.
- THERM: for the nasal canula → determining air flow / waves , it's where apnea is shown.
- THO, ABD: Thorax and Abdominal movements →Abdomen and Thorax are in-phase movements since they move competitively.
- Regarding the above PSG, we can tell that the patient is male since the abdomen is moving more than the thorax during respiration.

ABNORMAL WAVES IN PSG

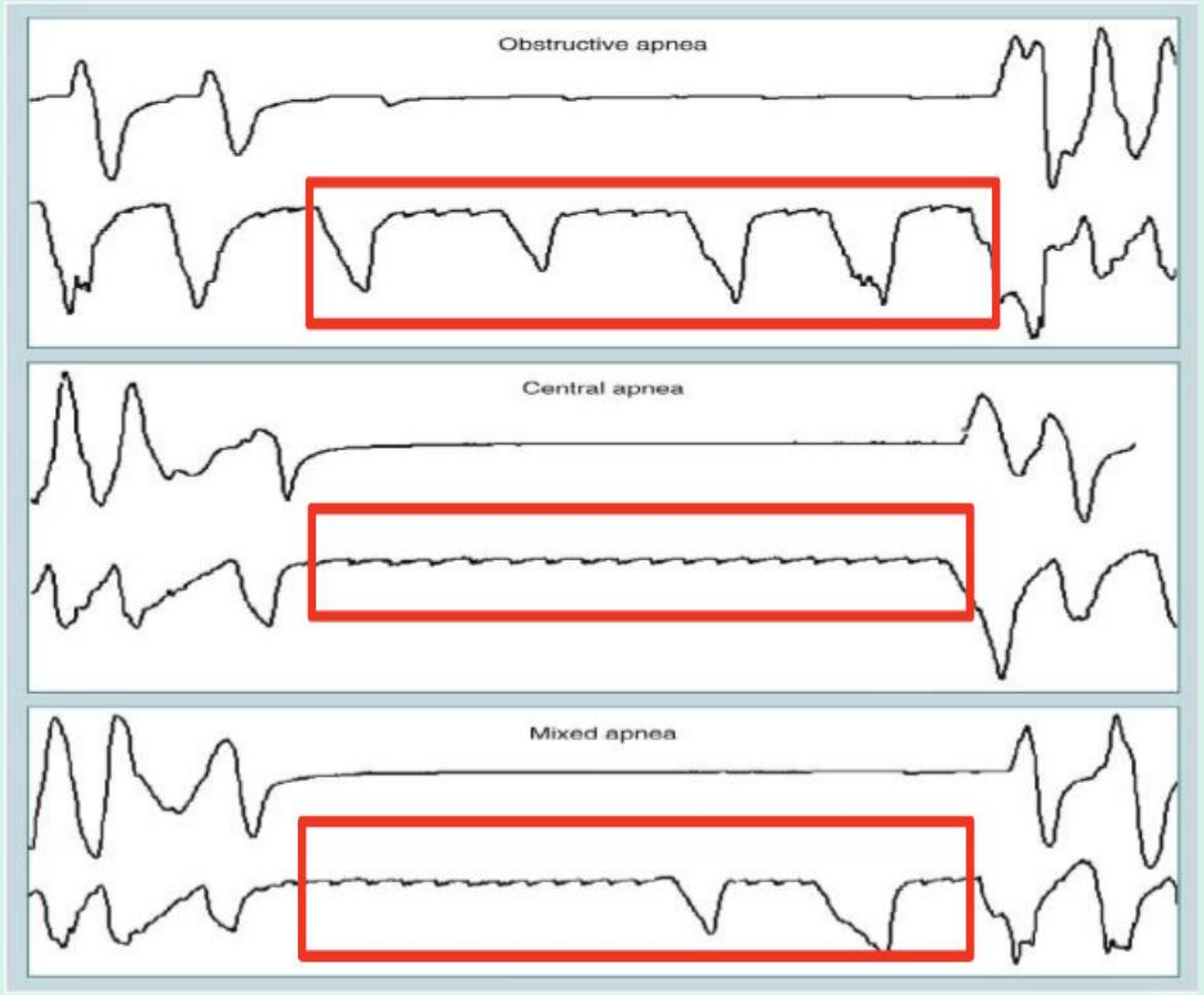


- In nasal/oral airflow waves, flat line means that no air entered or left there.
- At the same time if you looked at the thoracic movement waves you can tell that during apnea thorax movements persist (trying to resist) with abdominal paradoxical movements which supports Obstructive Sleep Apnea.
- Also, there is Paradoxical abdominal movements with apnea (Out of phase).
- By the end of the apnea, O2 Sat is at its lowest → this is a feature of OSA differentiating it from Central Apnea.
- In Central Apnea, O2 Sat returns to normal levels by the end of the event.
- During the phase of O2 desaturation there is Bradycardia.
- Notice on the EEG waves that at the end of Apnea there is a phase of arousal for Ventilation and reoxygenation.

SCORING RESPIRATORY EVENTS

- Apnea: 90% or more reduction in air flow or complete cessation of airflow for 10 seconds (02 Desaturation is not a criteria).
- Obstructive Apnea: Apnea with evidence of continued respiratory effort, i.e. Chest movement persists.
- Central Apnea: Apnea with absent respiratory effort, i.e. No chest movement (3 flat lines, no movement of Chest or Abdomen).
- Mixed Apnea: if inspiratory effort is absent at the beginning of the event but resumes in the second portion of the event.

TYPES OF APNEA



AHI = (# Apneas + # Hypopneas) / Sleep hours.

AHI <5: Normal

AHI 5 - 15: Mild

AHI 15 - 29: Moderate

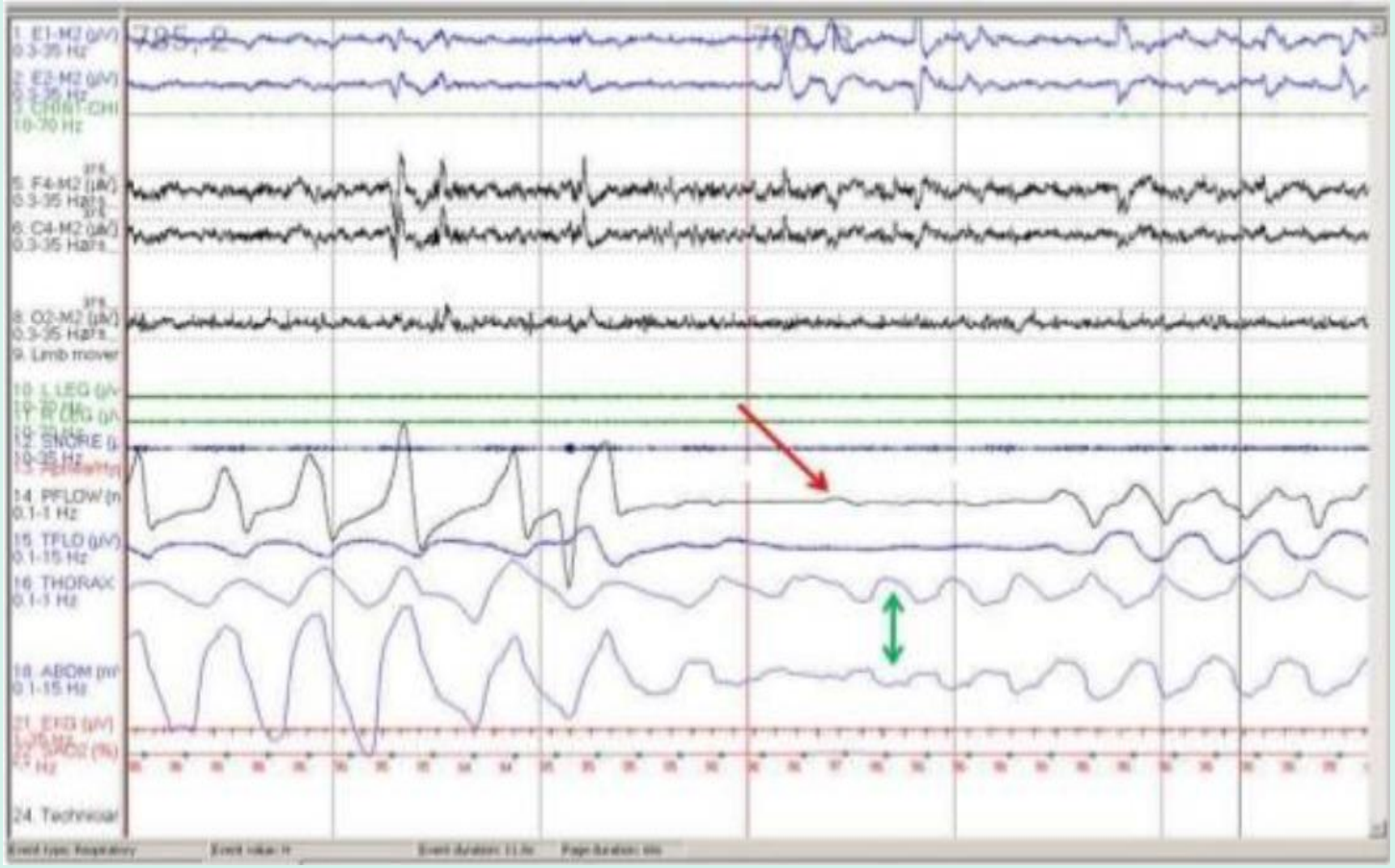
AHI >30: Severe

Respiratory Disturbance/Distress Index (RDI): a formula used in reporting polysomnography findings. Like the Apnea Hypopnea index, it reports on respiratory events during sleep, but unlike the AHI, it also includes respiratory-effort related arousals (RERAs).

RDI = (# Apneas + # Hypopneas + # RERAs) / Sleep hours.

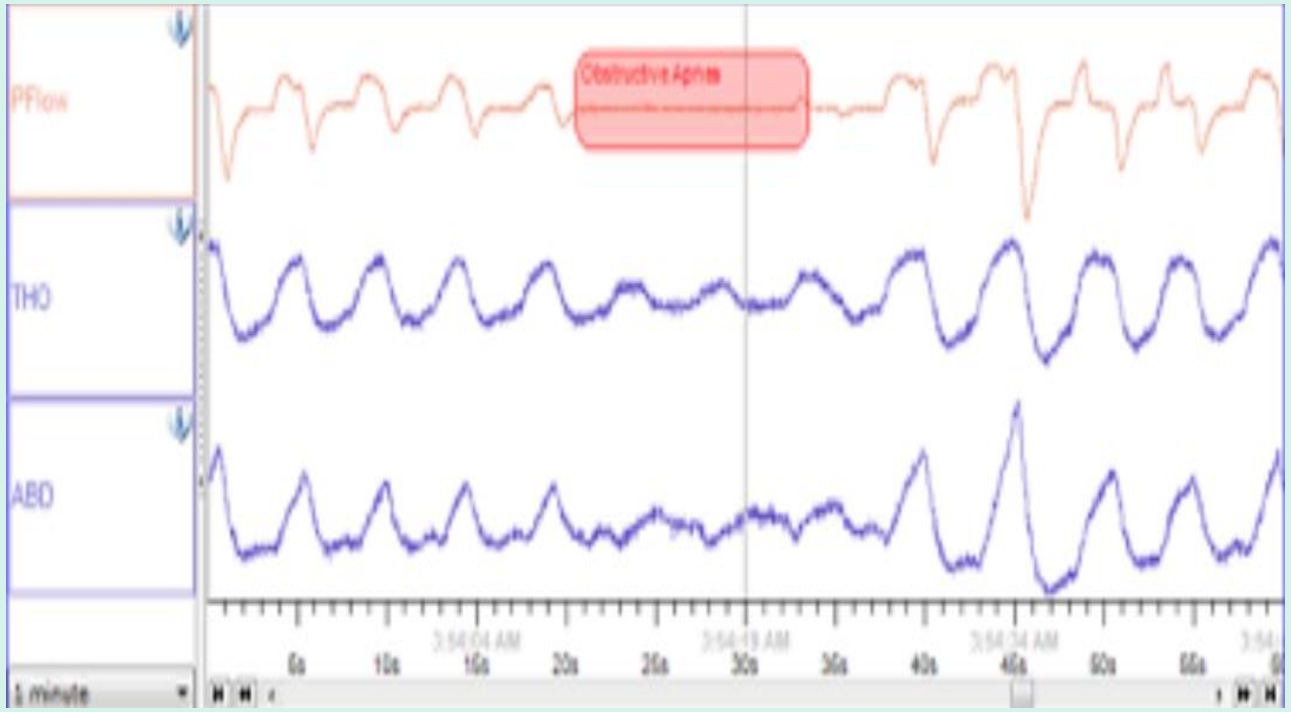
APNEA HYPOPNEA INDEX

OBSTRUCTIVE APNEA



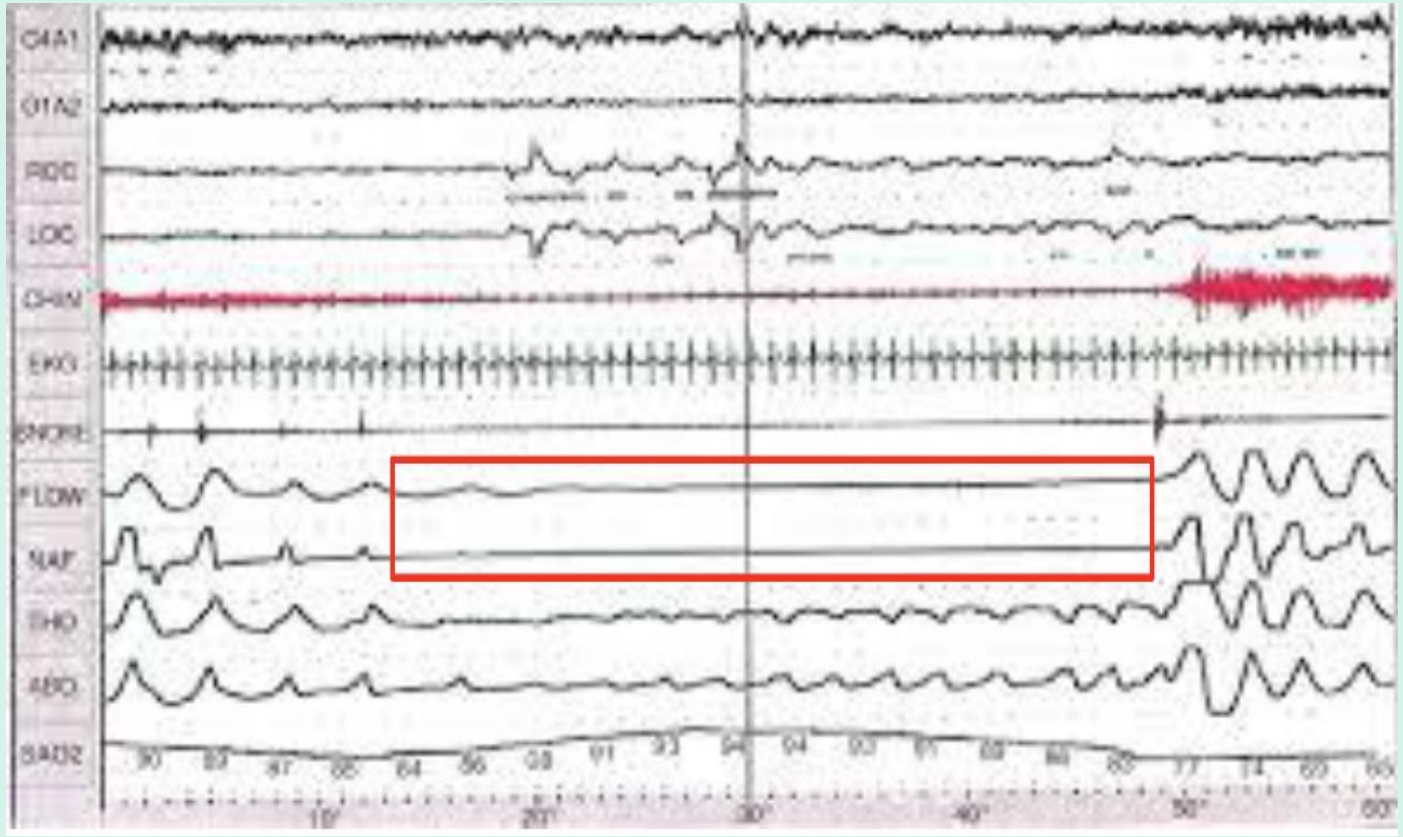
- The red arrow points towards a flat line → No air entry.
- Notice in this PSG we have two lines for Airflow, one of them is using temperature and the other is using pressure to measure airflow.
- Chest movements persisted / Paradoxical abdominal movements → out of phase

OBSTRUCTIVE APNEA



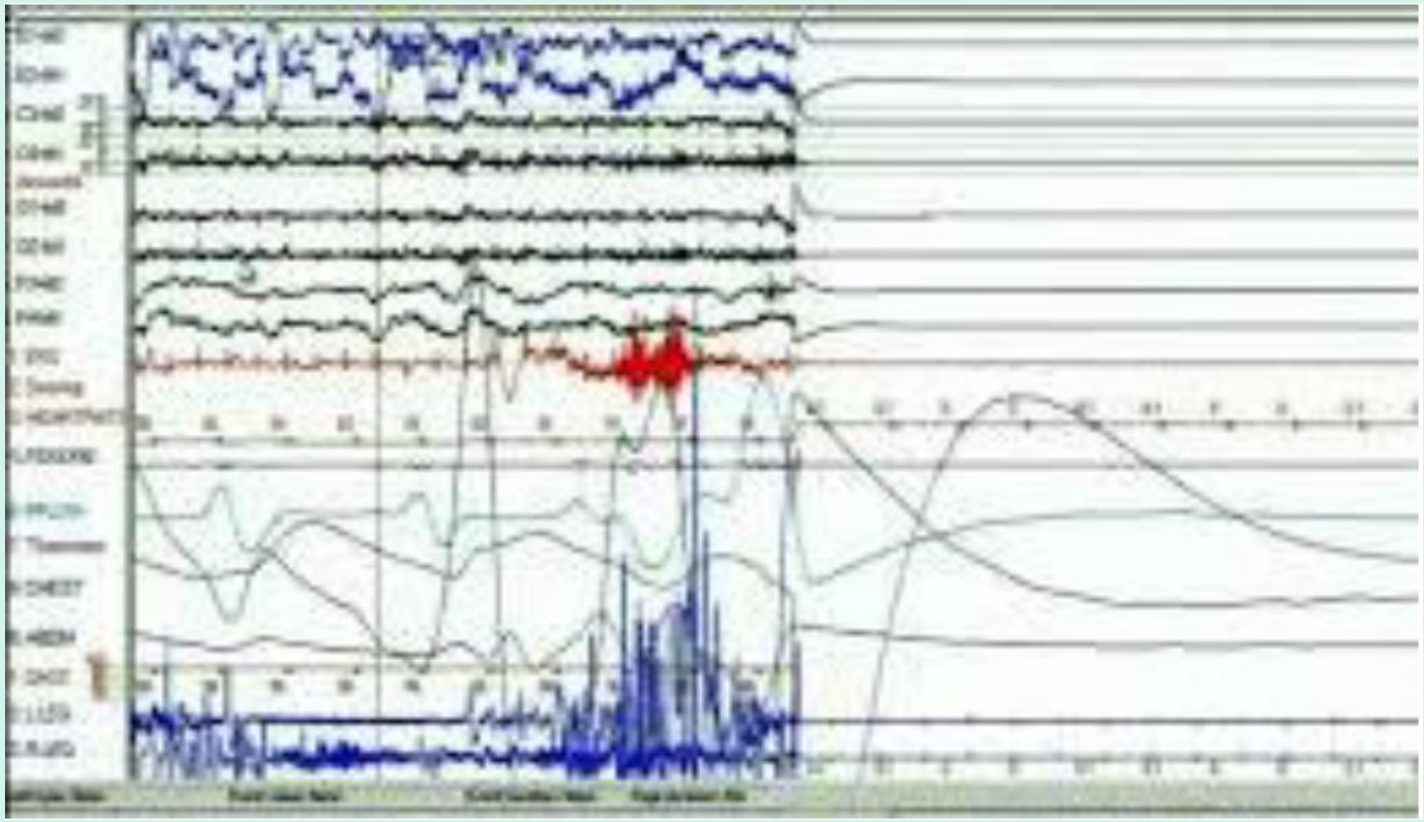
- Notice that we only have one flat line.
- Usually: 3 flat lines → Central.
- 1 flat and 2 moving lines → Obstructive.

OBSTRUCTIVE APNEA



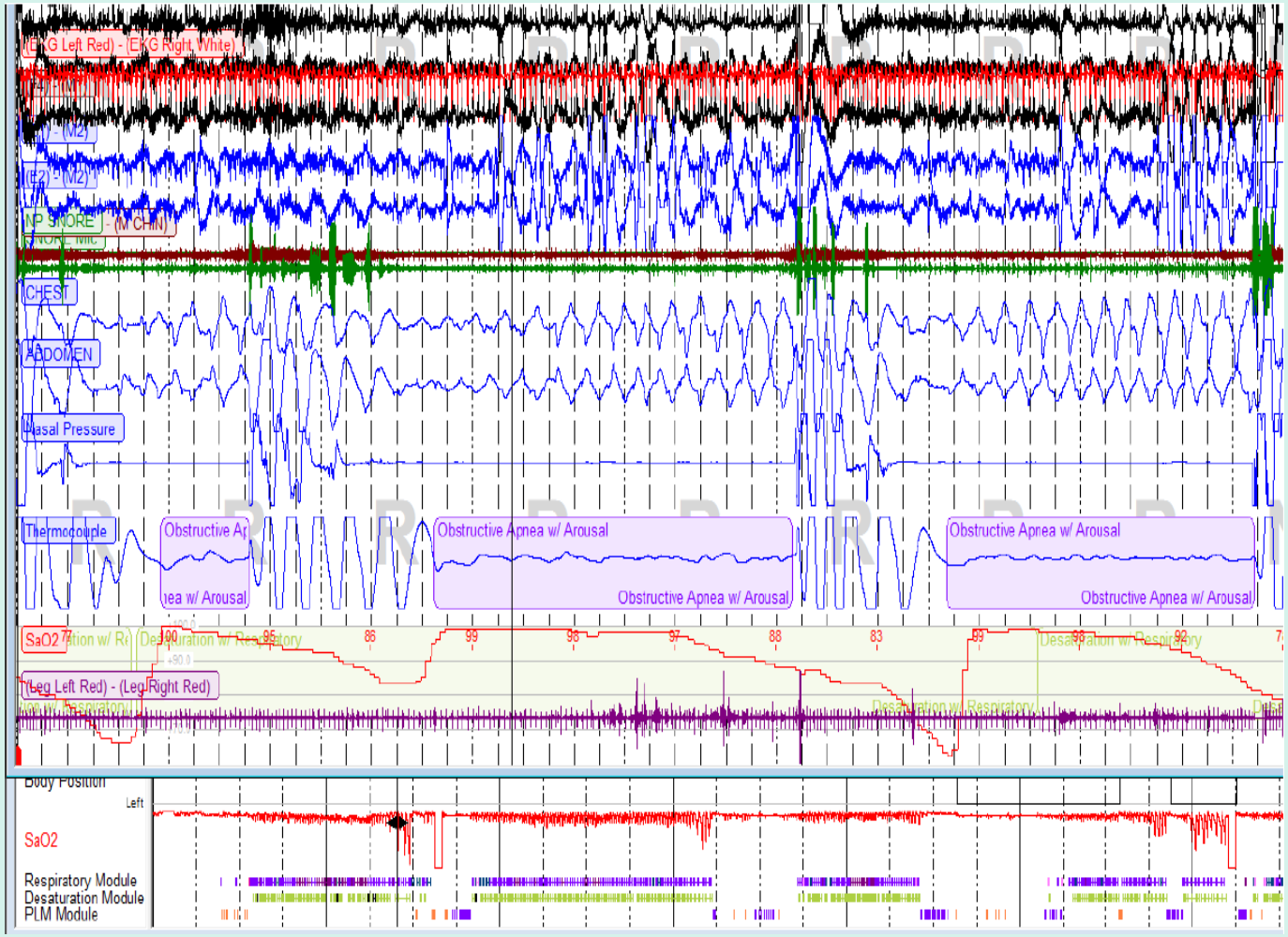
- PSG shows a very long period of apnea. In some cases, it can extend to 1.5 minutes.

ARTIFACT

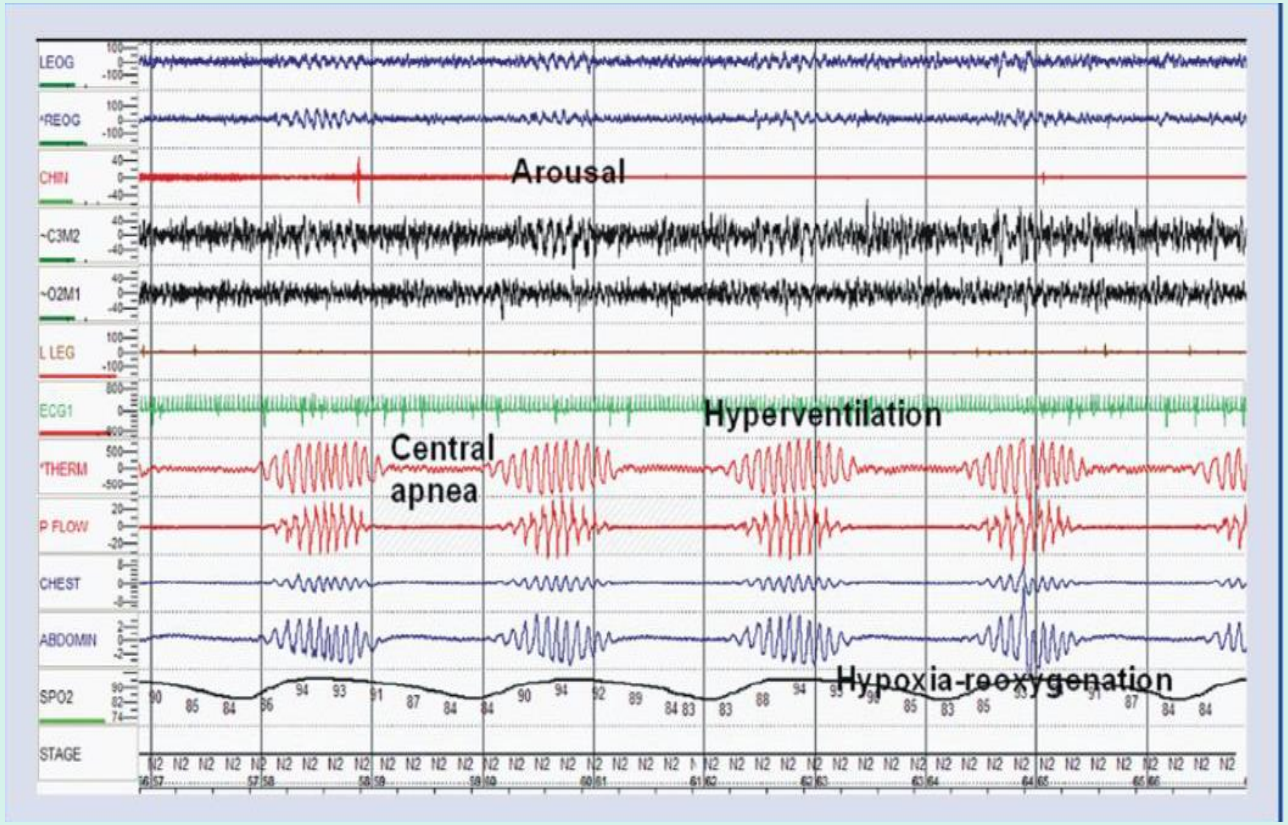


- All readings disappeared → this is an artifact, the patient may have removed the devices.

OBSTRUCTIVE APNEA

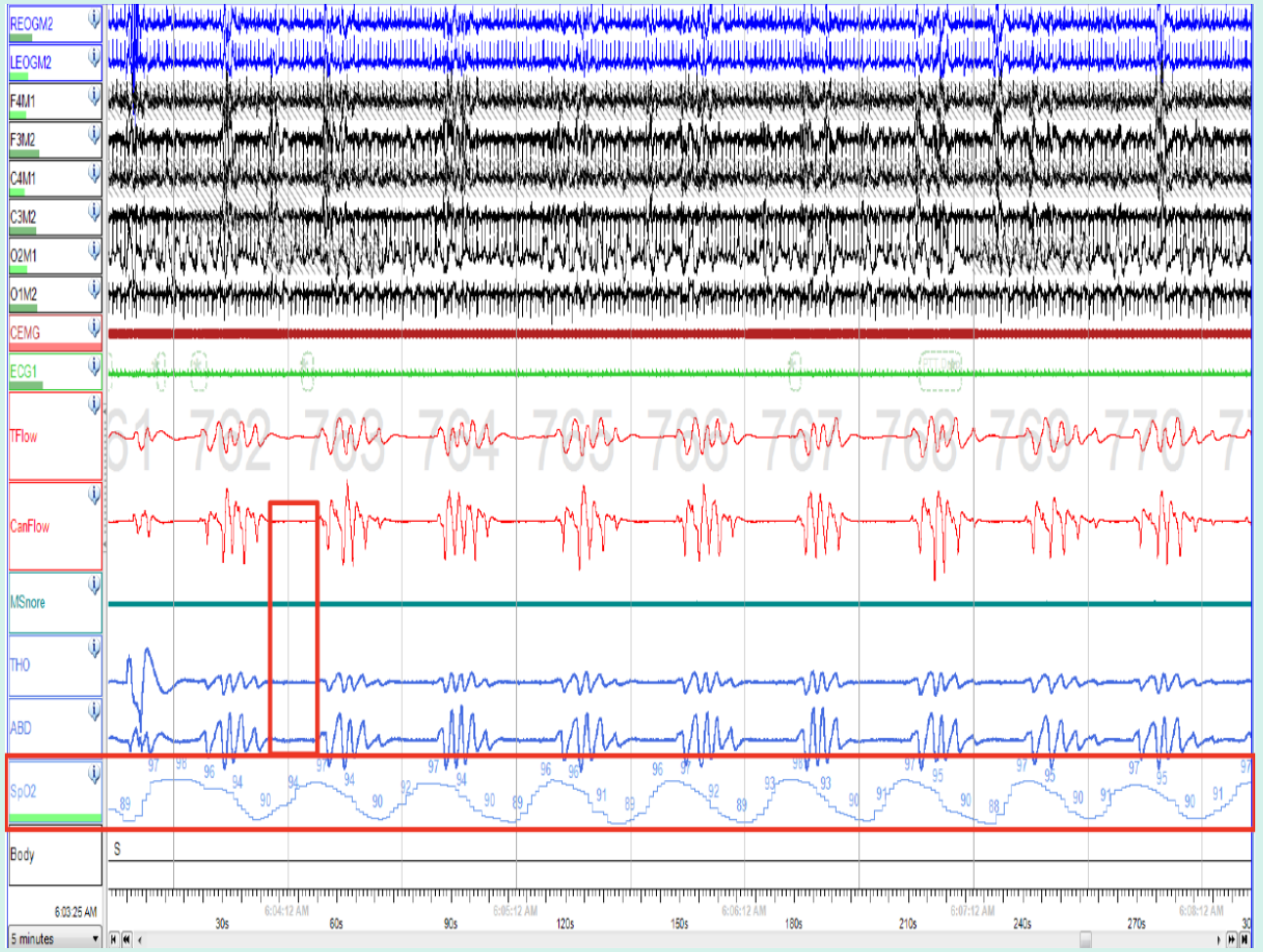


CENTRAL APNEA



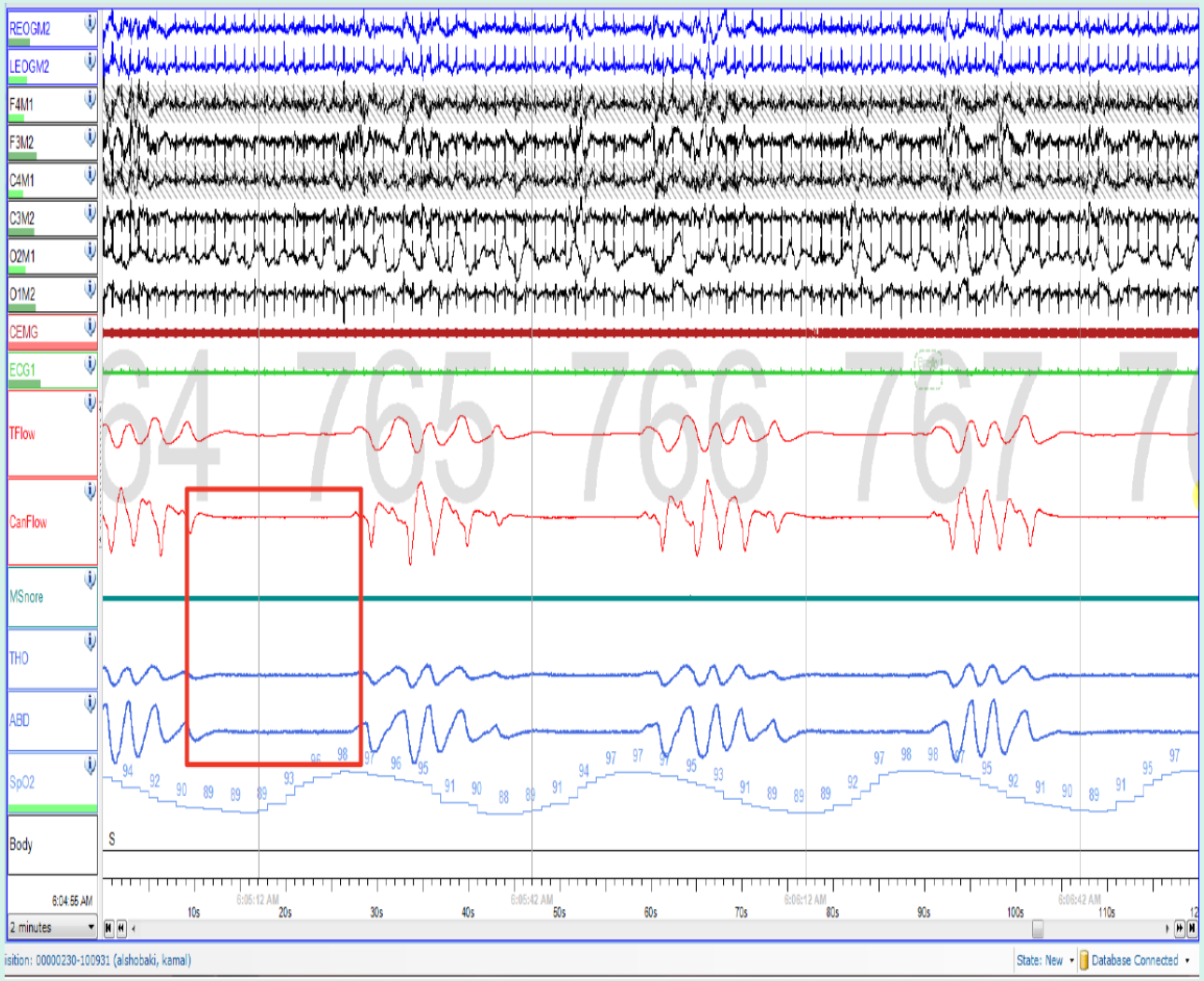
- Crescendo decrescendo changes in tidal breathing which sandwich central apneas.
- Note that the arousals occurred at the peak of hyperventilation. This contrasts with the temporal association of arousals occurring at the termination of obstructive apnea.
- Notice that all the lines are flat.
- Notice how 02 sat decreased halfway through the event returning to normal at the end of it - unlike OSA.

CENTRAL APNEA

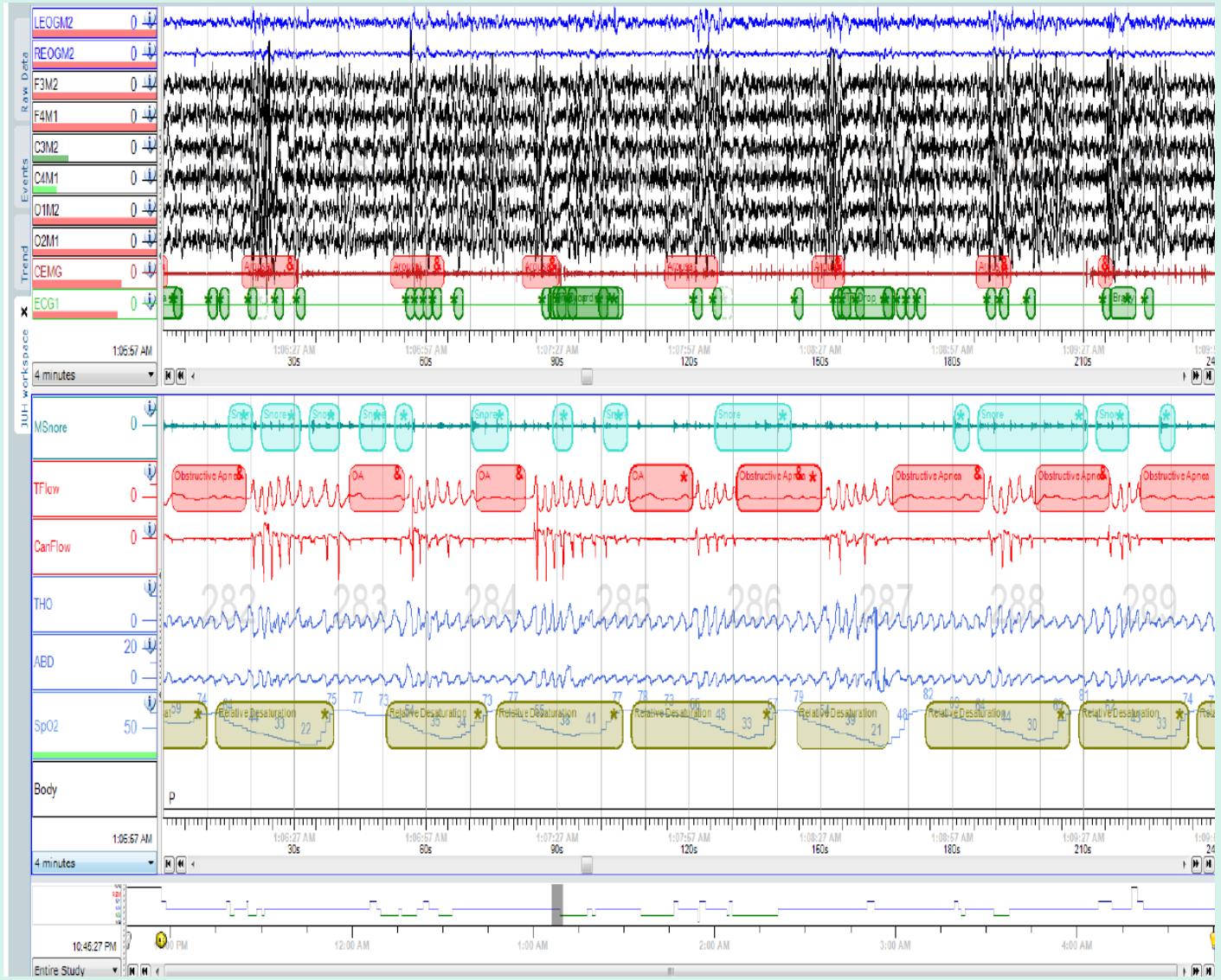


- 3 Flat Lines.
- 02 sat is normal by the end of the event.

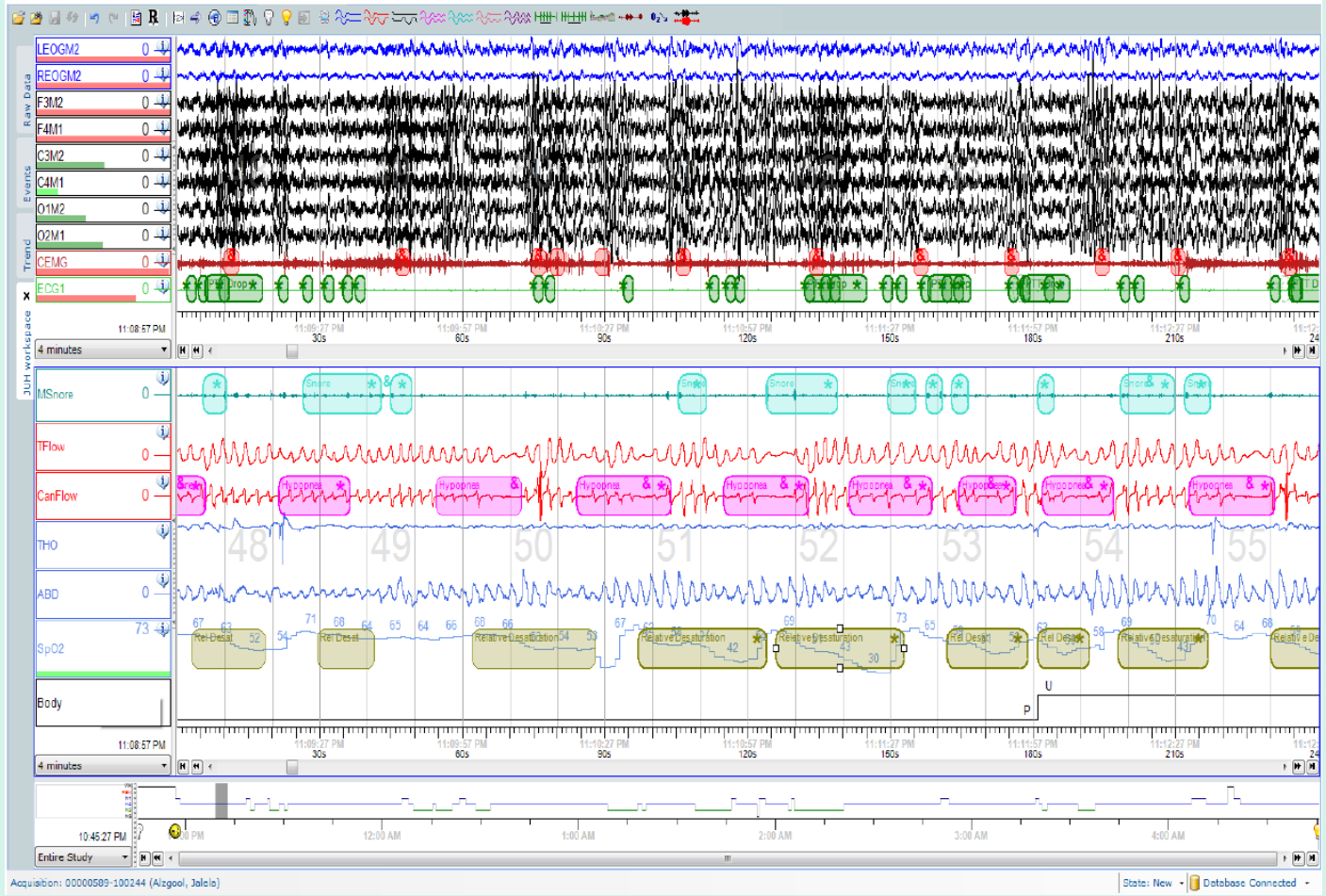
CENTRAL APNEA



OBSTRUCTIVE APNEA

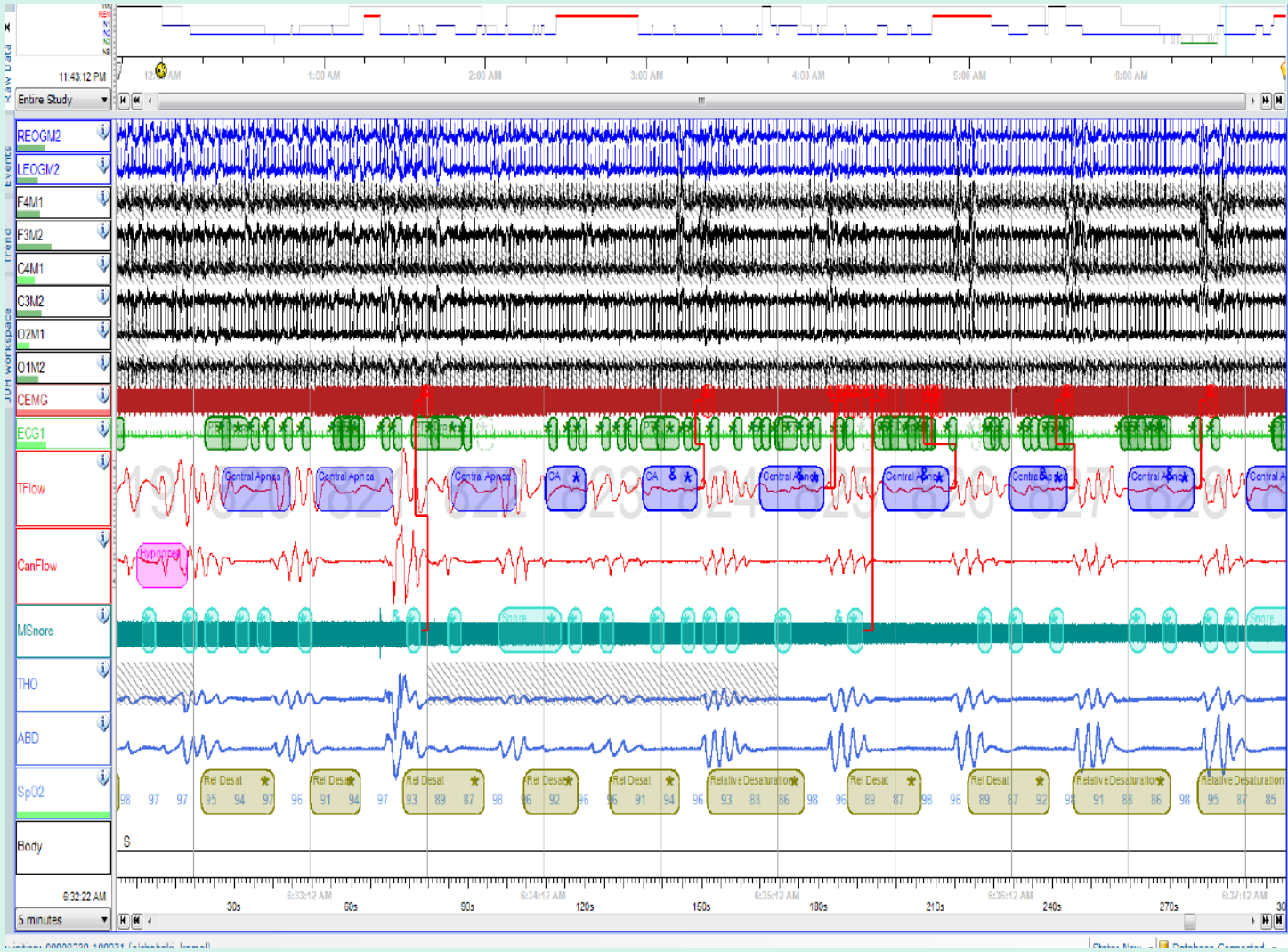


HYPOPNEA

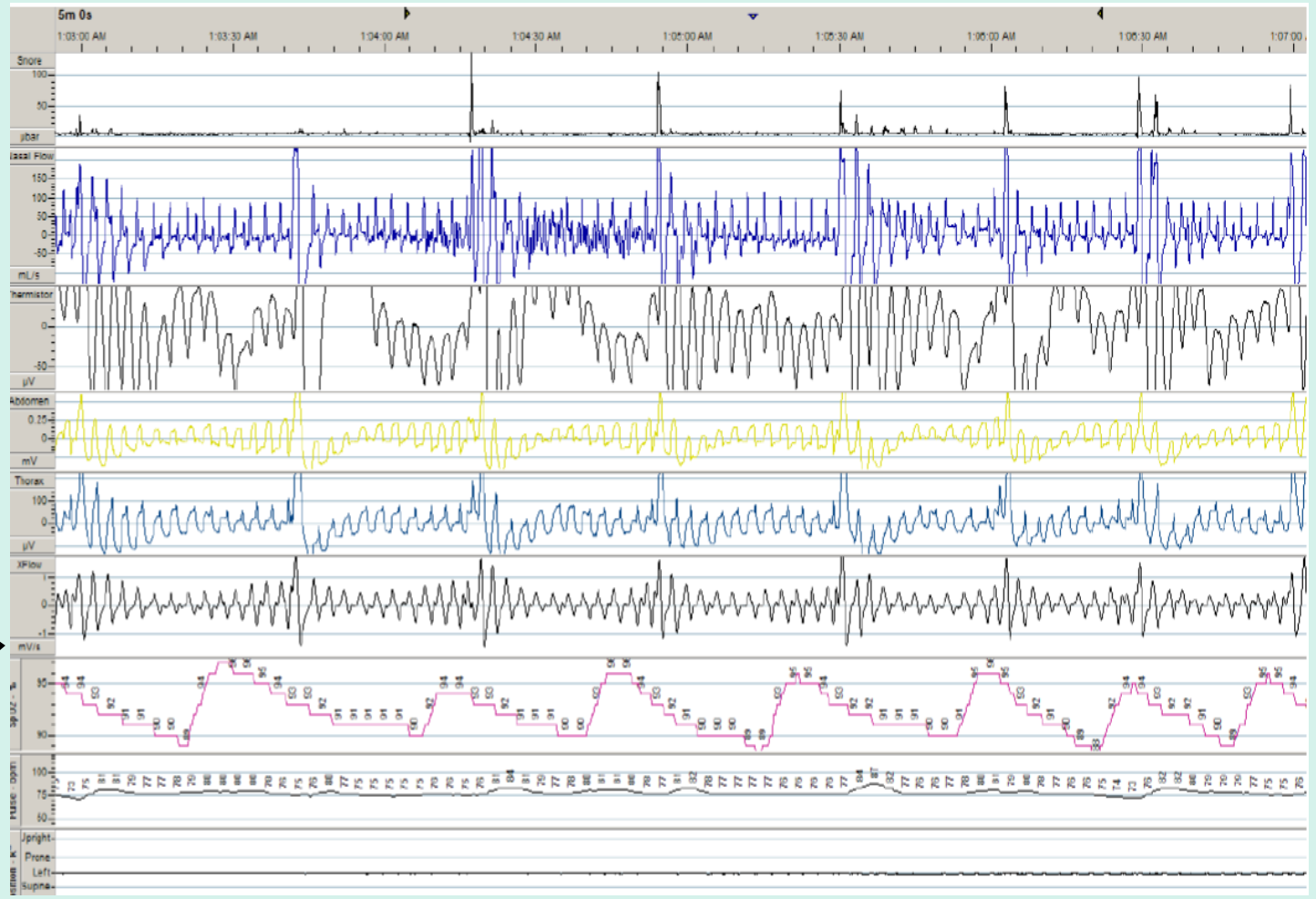


- Reduction in flow followed by arousal.
- Note that Sleep apnea and Hypopnea are different versions of the same sleep disorder. An Apnea is the complete blockage of air, while Hypopnea is the partial blockage of air.

CENTRAL APNEA



HOME SLEEP STUDY



- Done without EEG recordings → not useful in cases of sever apnea.
- Do:

O2 Sat, Abdomen, Chest, Canula flow from the nose, and a Microphon.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

- Flow generator with tubing and mask.
- It takes air from the room and push it into the airways.
- It pushes air out under pressure which keeps airways open and non collapsible

OBESITY HYPOVENTILATION SYNDROME (OHS)

A combination of obesity (BMI >= 30 kg/m2) and Daytime Hypercapnia (PaCO2 >=45 mm Hg) occurring in the absence of an alternative neuromuscular, mechanical or metabolic explanation for hypoventilation.

It's worse than OSA.

How to differentiate between OSA and OHS? We do ABGs while the patient is awake → If PaCO2 is normal: OSA, If PaCO2 is high >45 mm Hg: OHS, but the patient must be obese.

Patients with OHS usually present with:

1- Acute on top of chronic Type 2 RF.

2- Referral to respiratory clinic for: Suspected OSA, Unexplained Dyspnea, Pulmonary HTN.

3- They usually have HTN, DM, and other comorbidities.

- The prevalence of OSA is higher in men, while the prevalence of OHS is equal in Men and Women

Nearly 75% of the patients are misdiagnosed and treated for Obstructive lung disease → so do spirometry; if there is no obstruction then it's OHS, if there is obstruction then exclude OHS.

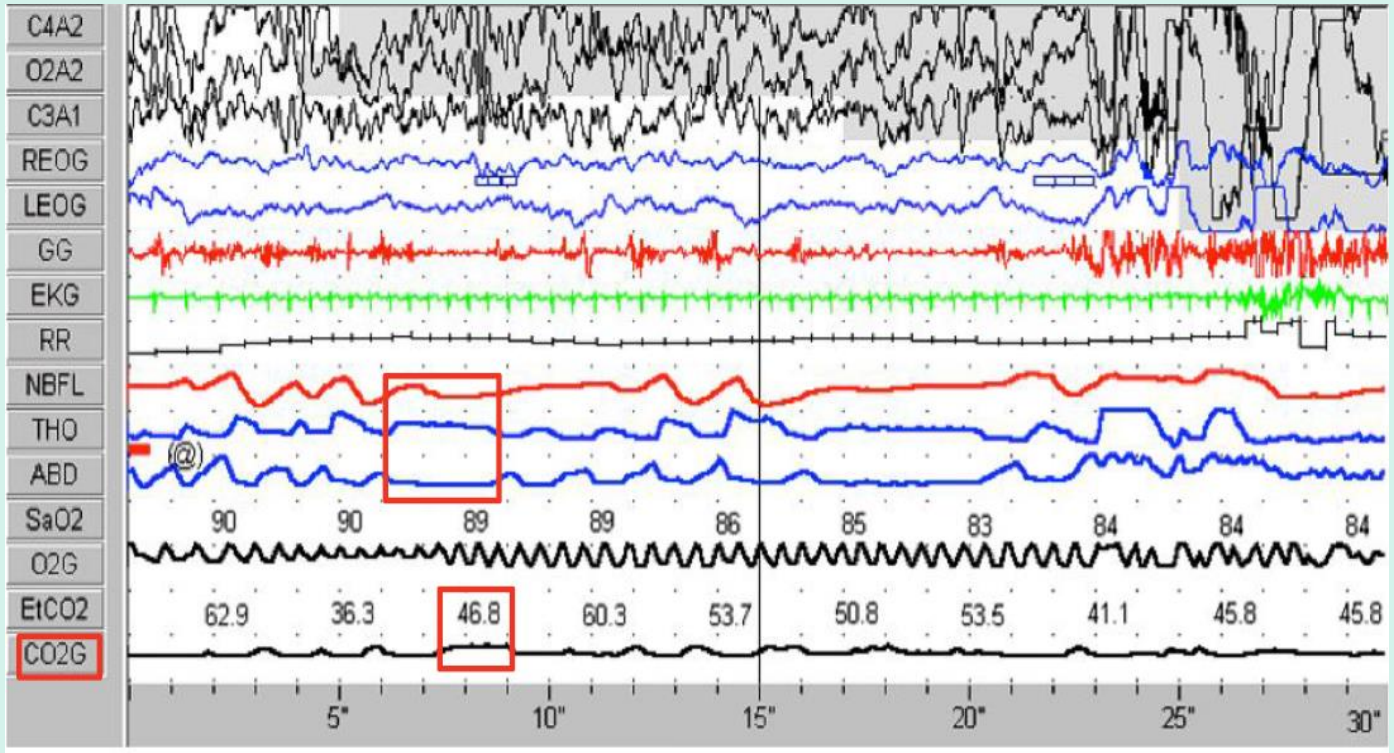
Make sure that the patient has no obstructive lung disease and no musculoskeletal disease.

Criteria for OHS with sleep hypoventilation: Just knowing that these patients' $PaCO_2$ elevates during sleep.

CO_2 levels usually measured using Transcutaneous carbon dioxide ($TcCO_2$).

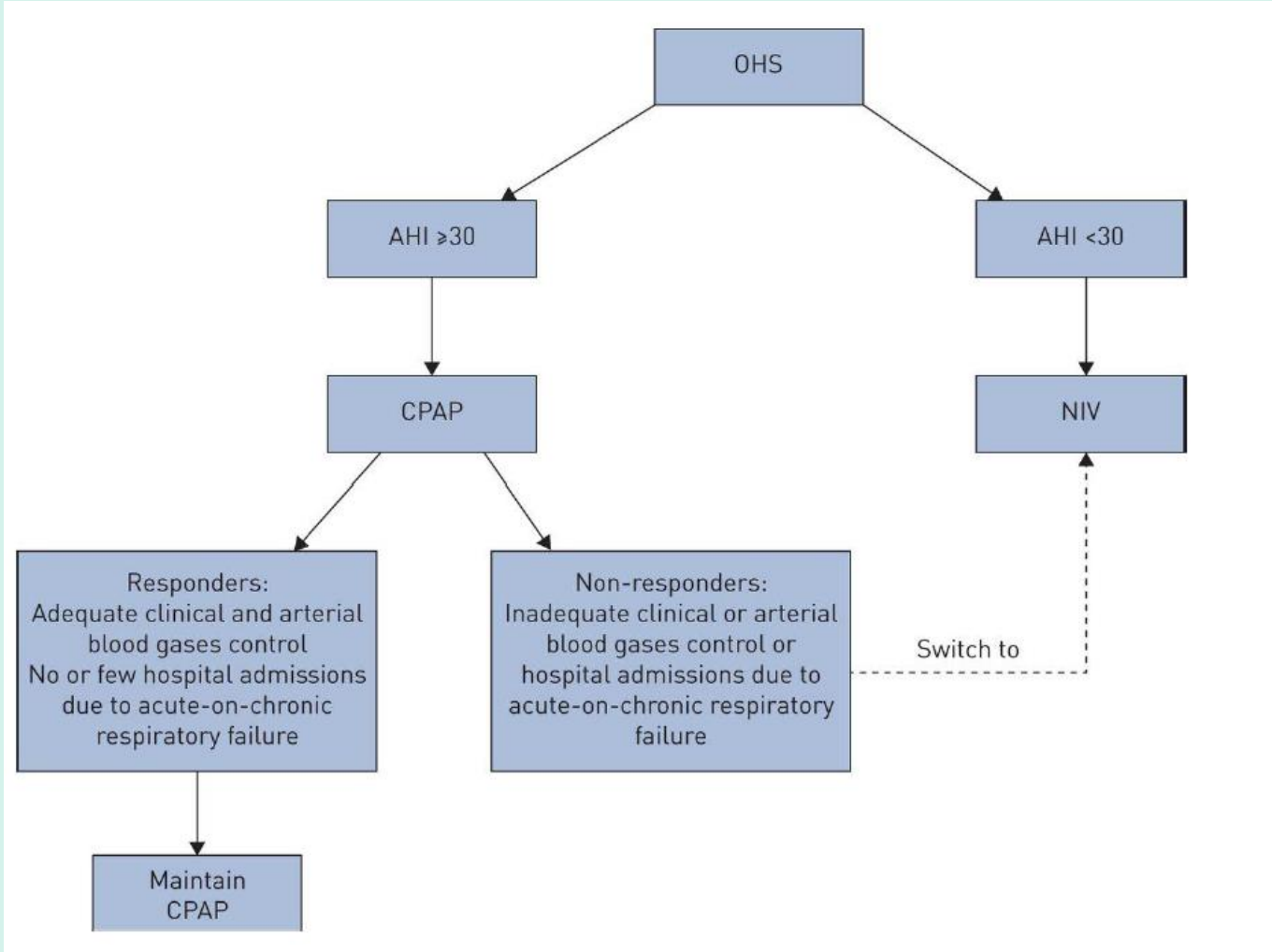
OHS

OHS



- Apnea associated with DAYTIME elevated PaCO2

OHS MANAGEMENT STRATEGY



COPD

COPD

A common (because smoking is common), preventable (by stopping smoking), and treatable disease.

The disease is characterized by persistent respiratory symptoms and airflow limitations that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

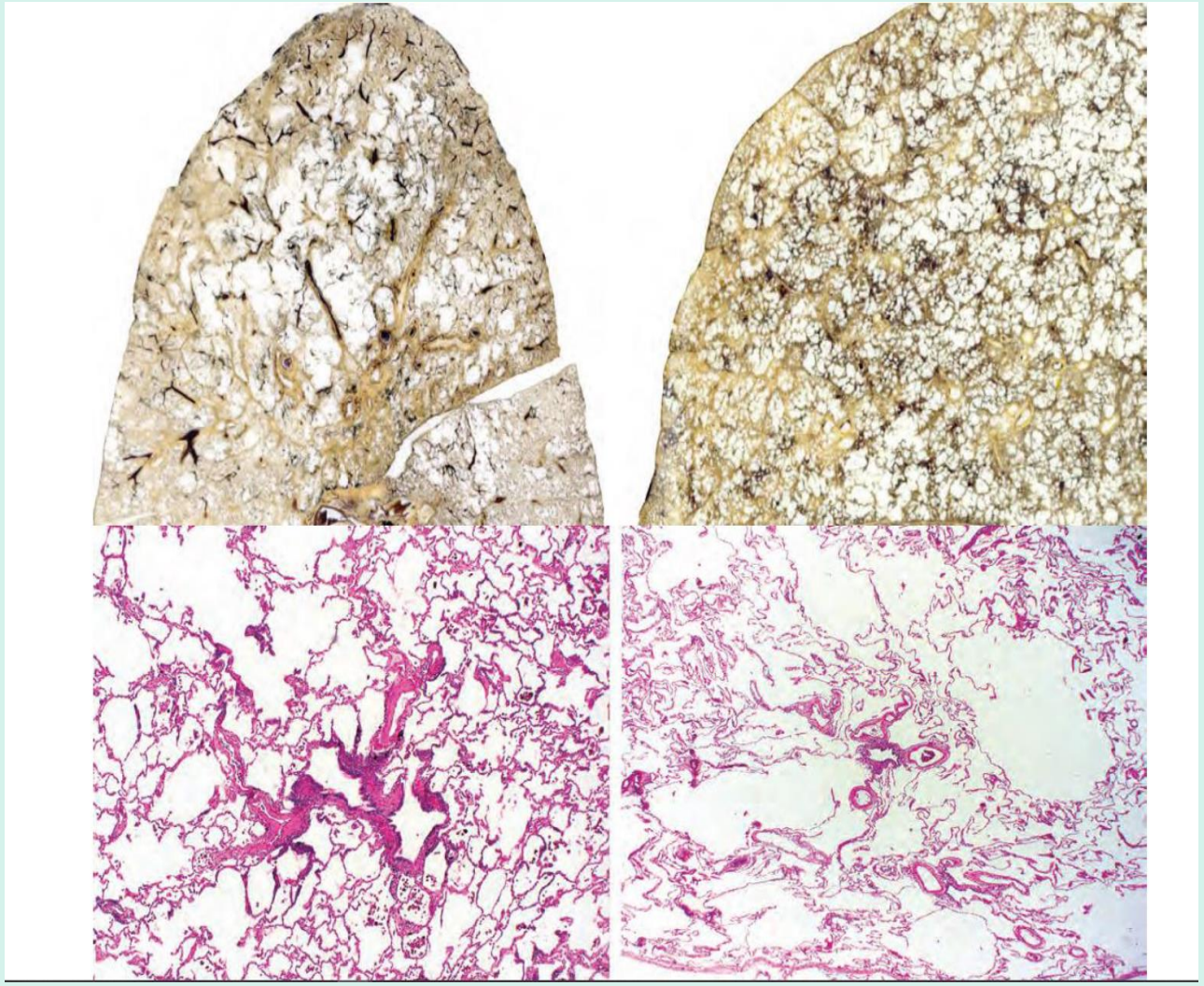
The chronic airflow limitation that is characteristic of COPD is caused by a mixture of small airways disease (e.g., obstructive bronchiolitis) and parenchymal destruction (emphysema).

Mixed chronic bronchitis and emphysema with different involvement from patient to patient.

Chronic bronchitis: defined in clinical terms as the presence of cough and sputum production for most days over 3 months for 2 consecutive years (Pathology: Thickened, Inflamed, and more mucous in airways).

Emphysema: defined as enlargement of the airspaces distal to the terminal bronchioles, due to destruction of the alveolar wall. Diagnosis mainly by Histopathological findings (Pathology: Loss of elasticity).

COPD



RISK FACTORS

- Tobacco smoke (the most common factor), including Cigarette, Pipe, Cigar, Water pipe.
- Indoor air pollution: Biomass fuel used for cooking and heating in poorly ventilated dwellings.
- Occupational exposures: inorganic dusts, chemical agents, fumes.
- Outdoor air pollution.
- Genetic factors: usually younger age with early presentation especially with heavy smoking, Mainly with Alpha 1 antitrypsin deficiency.
- Age and Sex
- Lung growth and development: in newborns.
- Socioeconomic Status
- Asthma and airway hyper-reactivity.
- Chronic bronchitis
- Infections

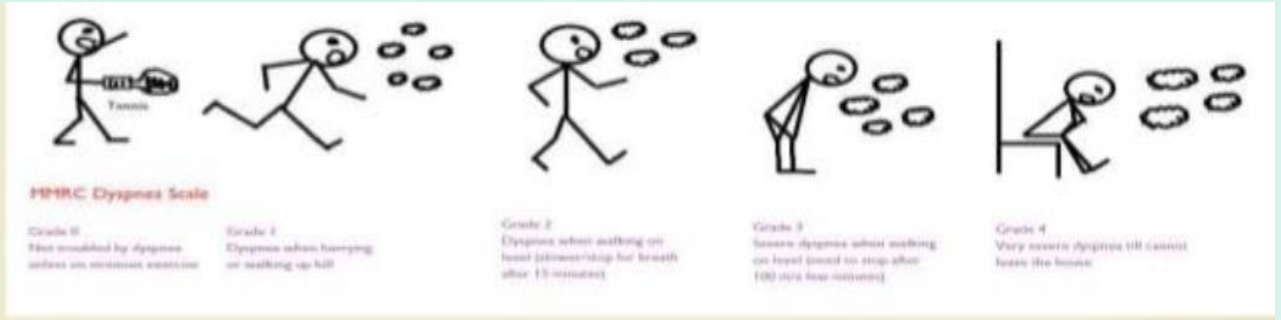
GOLD CLASSIFICATION OF SEVERITY OF AIRFLOW LIMITATION IN COPD, BASED ON POST-BRONCHODILATOR FEV1

In Patients with $FEV_1/FVC < 0.70$	
GOLD 1: mild	$FEV_1 \geq 80\%$ predicted
GOLD 2: moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3: severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4: very severe	$FEV_1 < 30\%$ predicted

- FEV1 / FVC <0.70 → means obstruction.

MMRC DYSPNEA SCALE

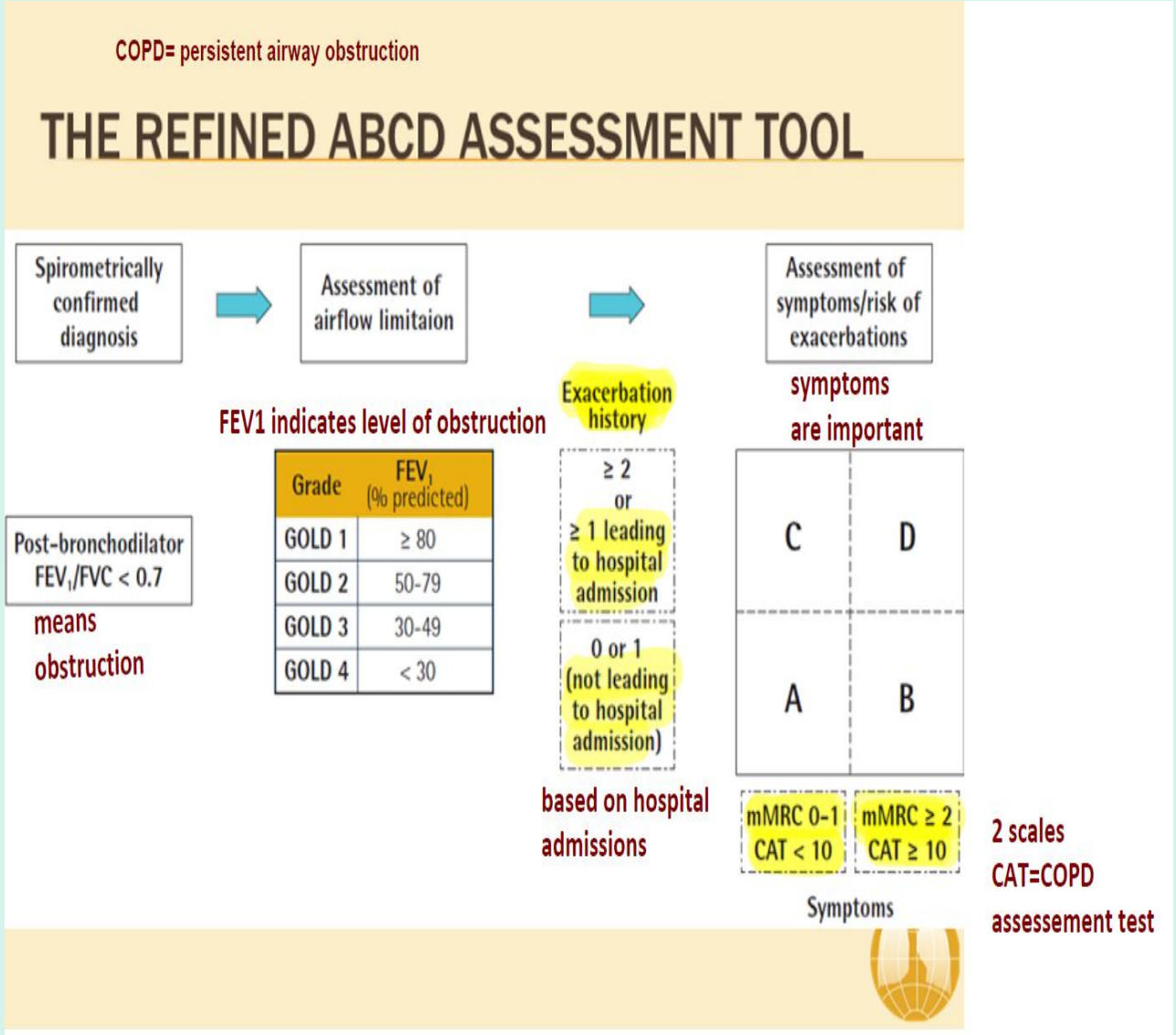
** VERY IMPORTANT - MEMORIZE IT- **



The Modified Medical Research Council (MMRC) Dyspnoea Scale

Grade of dyspnoea	Description
0	Not troubled by breathlessness except on strenuous exercise
1	Shortness of breath when hurrying on the level *or* walking up a slight hill
2	Walks slower than people of the same age on the level because of breathlessness *or* has to stop for breath when walking at own pace on the level
3	Stops for breath after walking about 100 m *or* after a few minutes on the level
4	Too breathless to leave the house *or* breathless when dressing or undressing

THE REFINED ABCD ASSESSMENT TOOL



ABCD ASSESSMENT TOOL

ABCD Assessment Tool

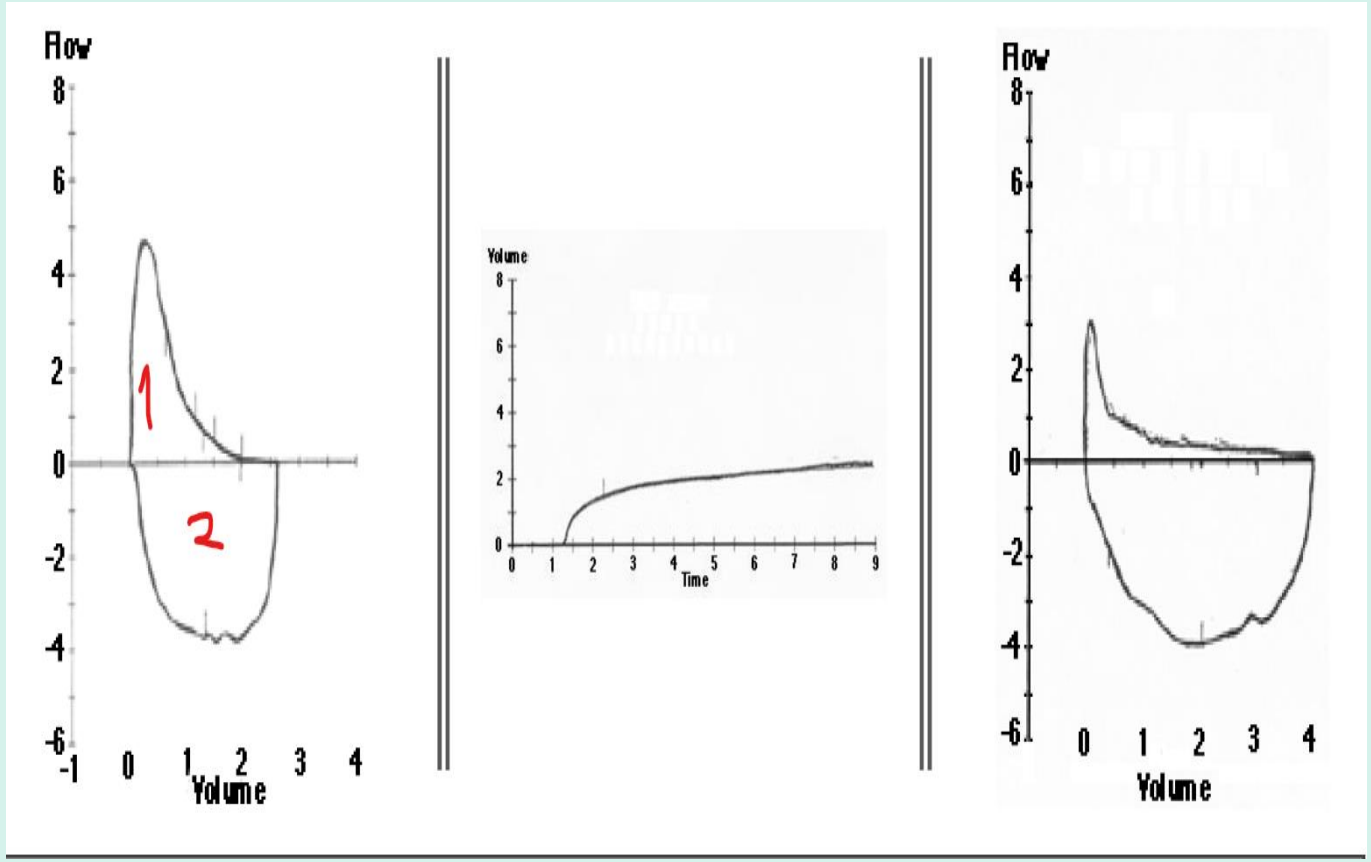
Example

- Consider two patients:
  - Both patients with FEV_1 < 30% of predicted
  - Both with CAT scores of 18
  - But, one with **0 exacerbations** in the past year and the other with **3 exacerbations** in the past year.

- Both would have been labelled **GOLD D** in the prior classification scheme.
- With the new proposed scheme, the subject with 3 exacerbations in the past year would be labelled **GOLD grade 4, group D**.
- The other patient, who has had no exacerbations, would be classified as **GOLD grade 4, group B**.

- Grouping Systems (MMRC and CAT) are important for treatment.
- The patient with 0 exacerbations → Group B
- The other patient → Group D
- Totally different prognosis and management.

SPIROMETRY SHOWING AIRFLOW OBSTRUCTION

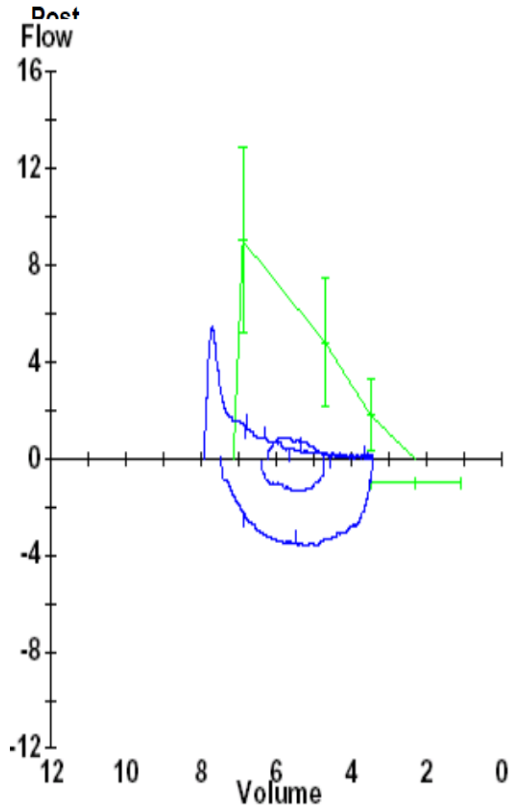


- #1 → Expiration
- #2 → Inspiration
- The picture on the left → Mild Airflow obstruction
- The picture on the Right → Severe Airflow obstruction

SPIROMETRY SHOWING AIRFLOW OBSTRUCTION

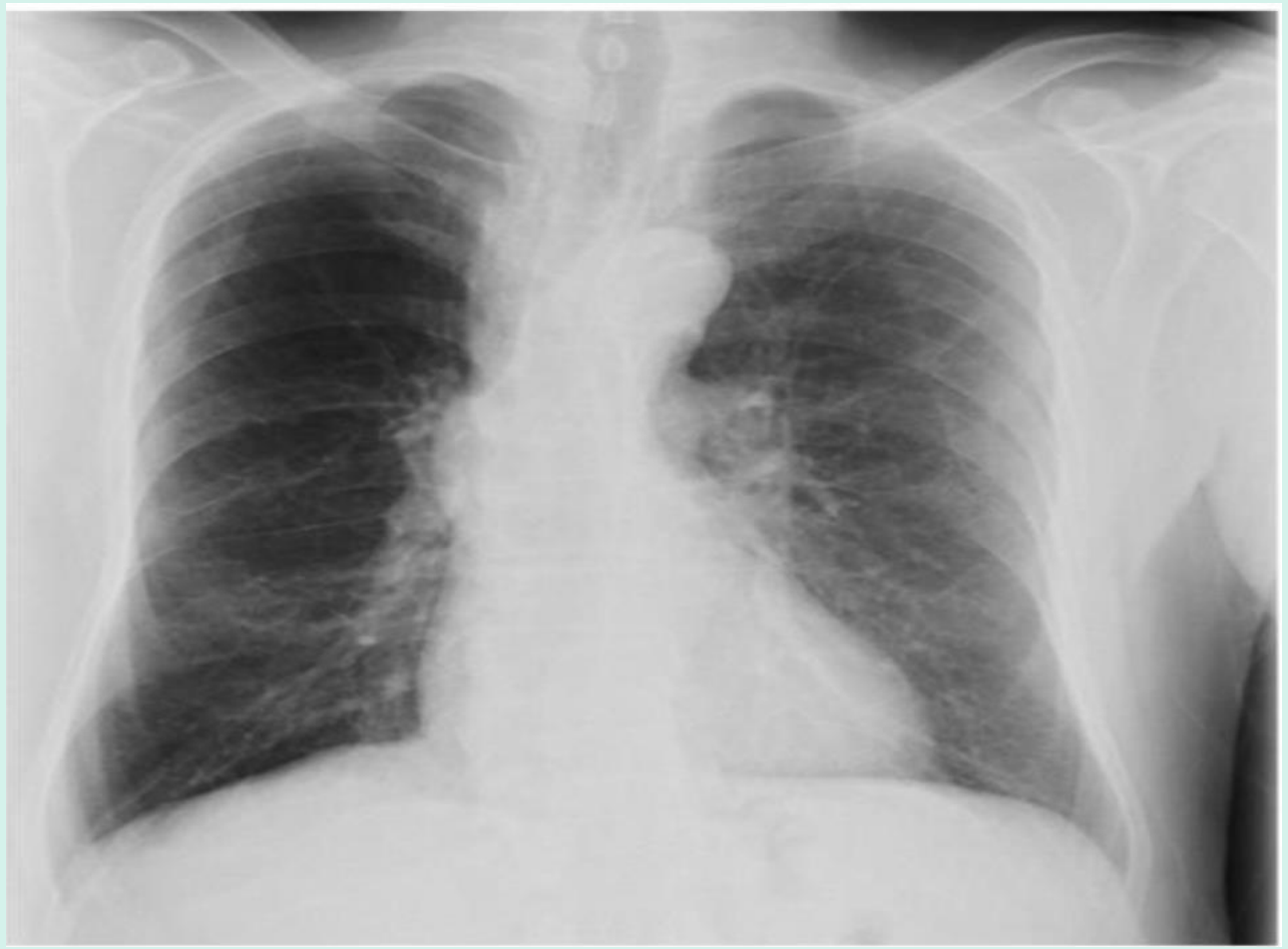
ID: CSM4166 Date: 10/03/04 Gender: Male Age: 62
Weight(kg): 79.0 Height(cm): 184 BMI: 23.33
PB: 753 Temp: 23

Spirometry	Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref
FVC	4.86	4.48	92		
FEV_1	3.38	(1.61)	(48)		
FEV_1/FVC	70.0	(36.0)			
FEF_{25-75}%	3.11	(0.35)	(11)		
PEF	9.02	5.43	60		
Lung Volumes					
TLC					
RV					
RV/TLC					
FRC PL					
ERV					
VC					
Resistance					
Raw					
sRaw					
Diffusion					
D_{LCO}					
D_{LCO} /V_A					
V_A					



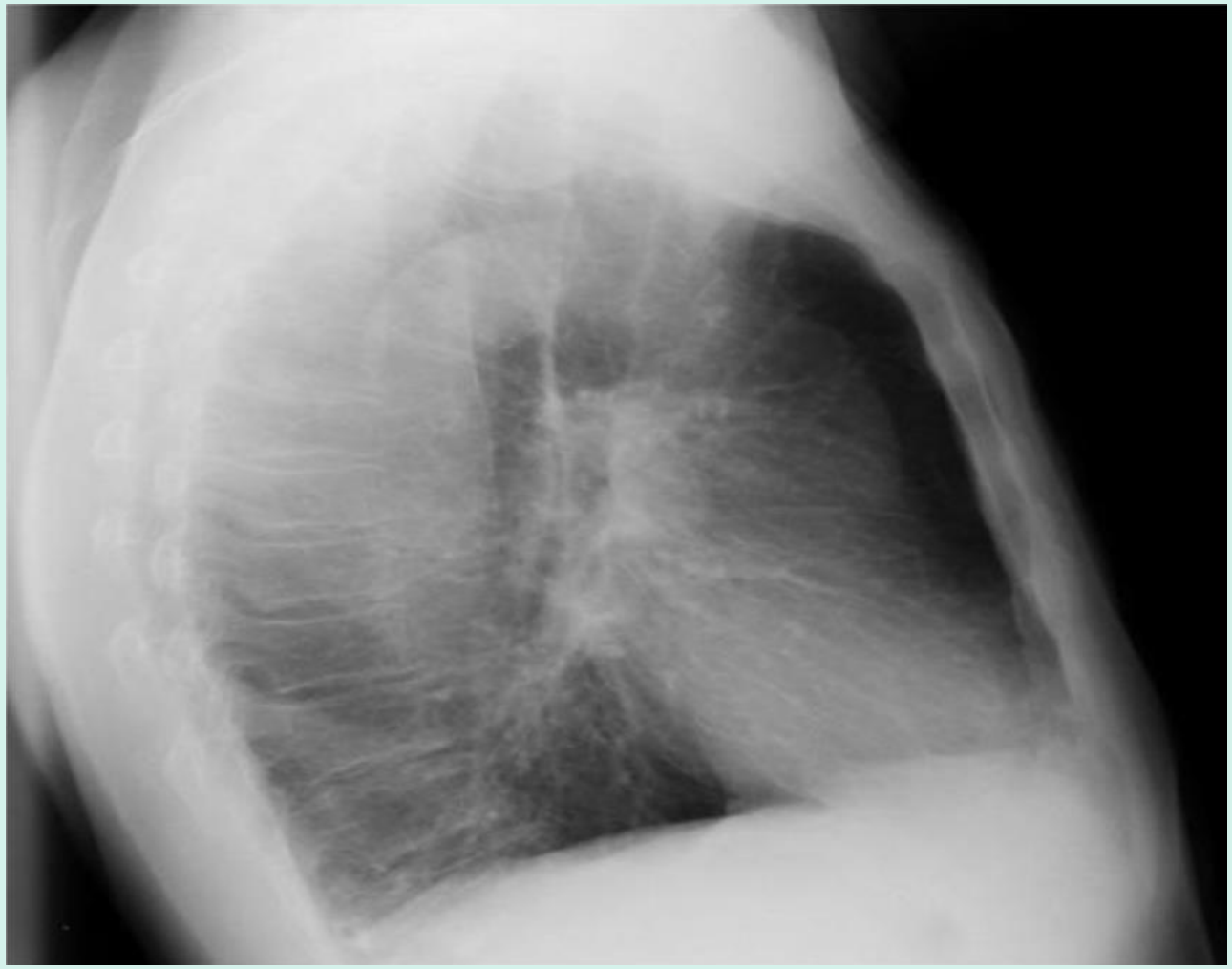
Comments: The patient could not fully expire during to FVC or SVC, therefore the results for both vital capacities may be underestimated. See attached FV loops

CXR OF A PATIENT WITH COPD



- X-Ray isn't diagnostic but it may give hints.
- This radiograph shows Hyperinflation (more obvious on lateral x-rays) + Flattening in Diaphragm + Increased Intercostal Spaces → Suggestive of COPD.

LATERAL CXR OF A PATIENT WITH COBD



- Hyperinflated lung
- Flattened Diaphragm
- Increased Antero-posterior diameter (Barrel chest)

DIFFERENTIATING COBD FROM ASTHMA

	Asthma	COPD
Onset	Anytime (often childhood or youth)	Later in life
Etiology	Allergic, family history	Smoking, other noxious exposures
Course	Intermittent	Chronic progressive
Clinical features	Wheeze, episodic dyspnea, cough	Persistent dyspnea, productive cough
Pattern of Symptoms	Variable day to day, more at night/early morning	Less variable, more on exertion
Inflammatory cells and mediators	Eosinophils, mast cells, Th-2 type	Neutrophils, macrophages, Th-1 type
Response to Bronchodilators	Largely reversible	Partially reversible or irreversible
Response to steroids	Substantial	Partial

INTERSTITIAL LUNG DISEASE

ILD

Interstitial lung disease is any disease that affects the area between the alveoli (doesn't affect blood vessels and airways directly).

Characterized by excess production of fibroblasts (increased fibroblasts activity) which replaces the normal lung parenchyma causing damage to the alveoli and blood vessels and affecting gas exchange by producing fibrin, elastin, and collagen.

Patients are usually hypoxic, and present with CO2 retention in advanced stages.

Patients also develop Pulmonary HTN due to Hypoxia that causes Vasoconstriction.

Pathophysiology: Repeated exposure to inflammatory agents or imperfect repair of damaged tissue leads to permanent damage.

Physiological impairment due to damage: V/Q mismatch, Shunt, Decreased DLCO, Decreased Lung compliance.

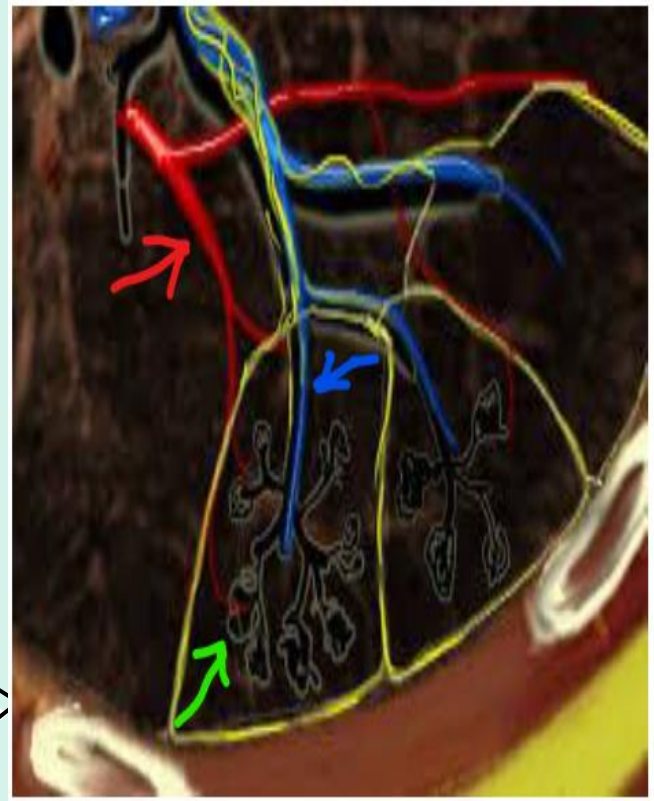
Patients present with Respiratory symptoms along with:

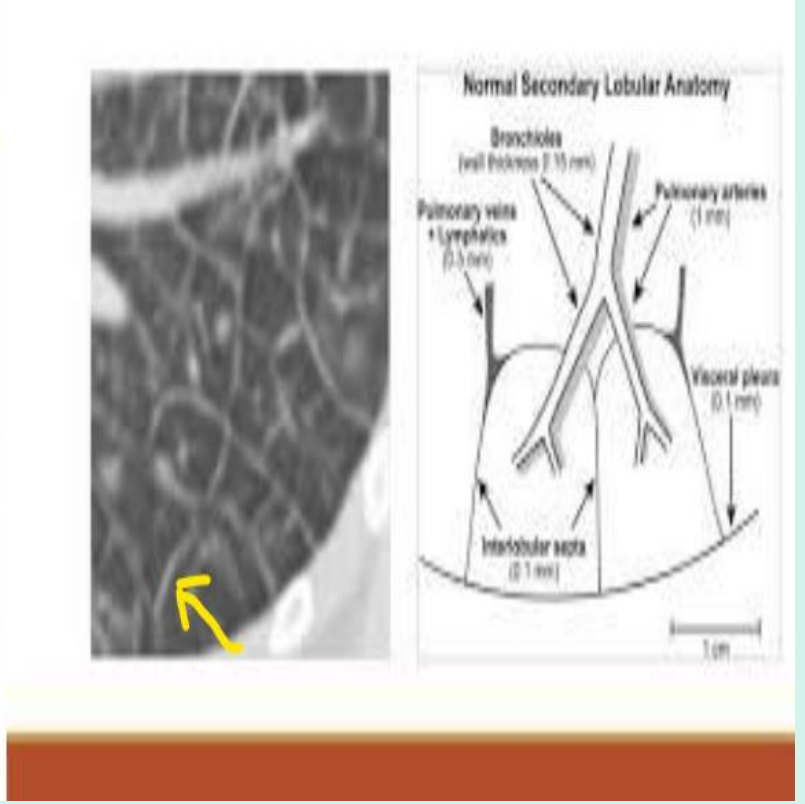
1- Radiologically diffuse infiltrates (on CXR + CT).

2- Histologically by distortion of the gas exchanging units.

3- Physiologically by restriction of the lung volumes and impaired oxygenation.

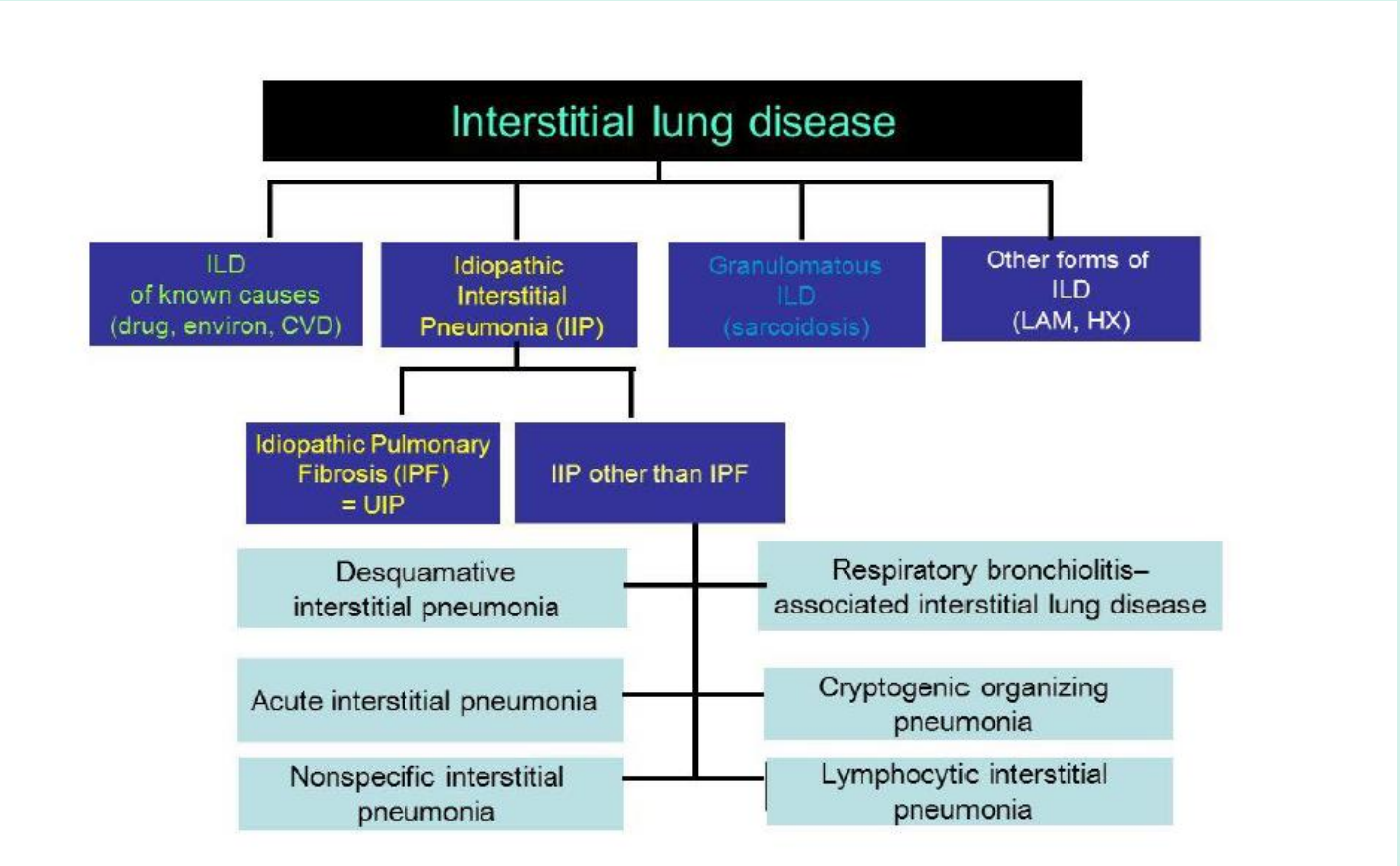
SECONDARY PULMONARY LOBULES





- Red Arrow → Pulmonary Artery.
- Blue Arrow → Pulmonary Vein.
- Green Arrow → Alveoli.
- The area surrounding the alveoli is where disease starts and it's full of fibroblasts.
- Yellow Arrow → Interlobular septal lines, those separate Secondary pulmonary lobules (they become prominent when affected by ILD, usually it doesn't appear like this especially peripherally)

CLASSIFICATION ATS/ERS



- We have 4 major classifications.
- HX stands for Histiocytosis X.
- Idiopathic Pulmonary Fibrosis: the most common form of lung fibrosis.
- Usual interstitial Pneumonia (UIP) is a histological definition.
- IPF is a disease that has UIP appearance → we refer the patients for biopsy after excluding other causes (not every UIP is IPF)
- IIP other that IPF is further classified depending on Histology.

DIAGNOSIS OF ILD

History

- Age >70 years ..... IPF(Idiopathic pulmonary fibrosis).
- Exertional dyspnea and nonproductive cough
- Rarely sputum production, hemoptysis, or wheezing

Duration :

Acute : AIP

Infection

Acute HP(hypersensitivity pneumonitis), acute EP (eosinophilic pneumonia).

Drug reaction COP, CTD (e.g. acute lupus pneumonitis)

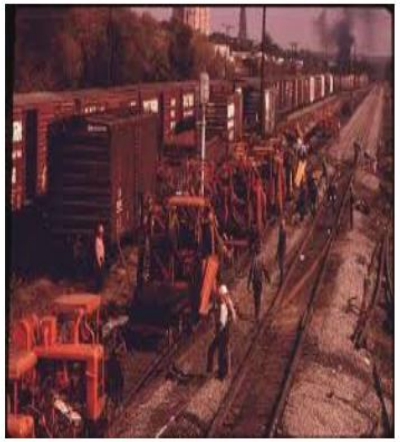
DAH (diffuse alveolar hemorrhage).

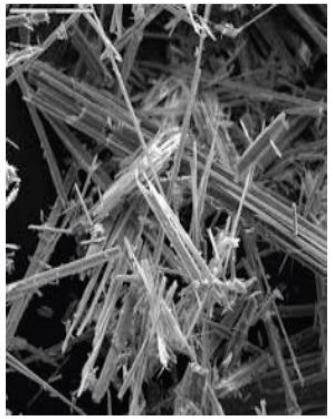
Chronic :IPF ,

- Non respiratory symptoms related to disease complications .
- Smoking related ILDs(Interstitial lung diseases).
- Exposure and occupation .

- Most patients are old, we rarely see the disease in young patients.
- There is no airway involvement but in advanced stages there will be Traction Bronchiectasis and patients will start producing sputum.
- Sputum production, Hemoptysis and Wheezing are rare symptoms.
- If ILD is Acute → Acute interstitial Pneumonia (AIP): Bilateral Infiltrate, ICU admission, Very ill and sick patients and require Ventilatory Support.

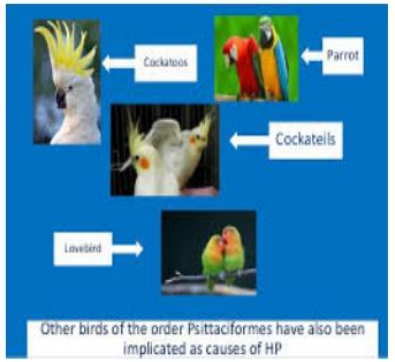
OCCUPATIONS RELATED TO ILD





- Isolation
- Building
- Industry
- ** any occupation that involves Exposure to Asbestos.

HYPERSENSITIVITY PNEUMONITIS (HP)

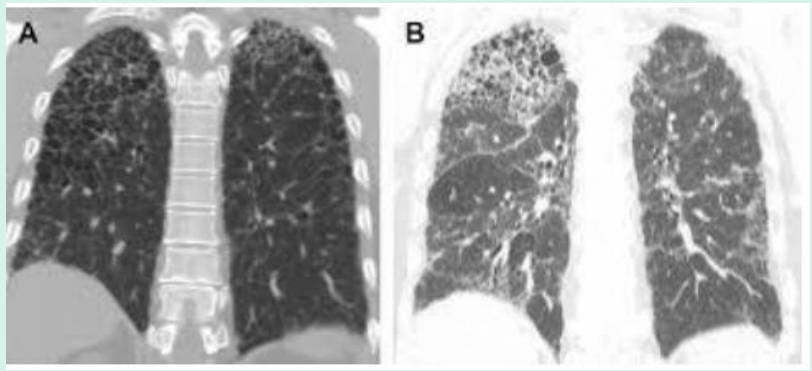


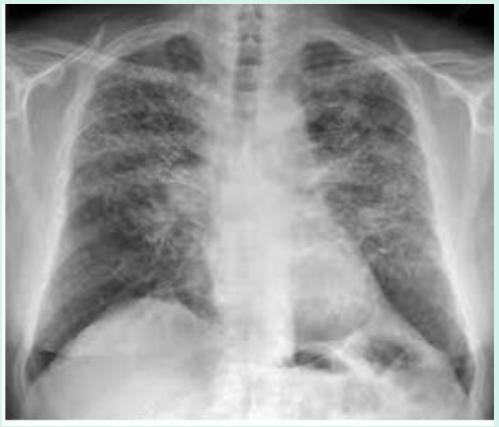
- Exposure to allergens in the environment (Birds, Cotton) not similar to Asthma allergens.
- On PE: Chest → Increased Tactile and Vocal Fremitus, Dull percussion note, Bronchial breath sounds, Crackles usually fine end inspiratory, Pleural friction rub, Whispered Pectoriloquy.

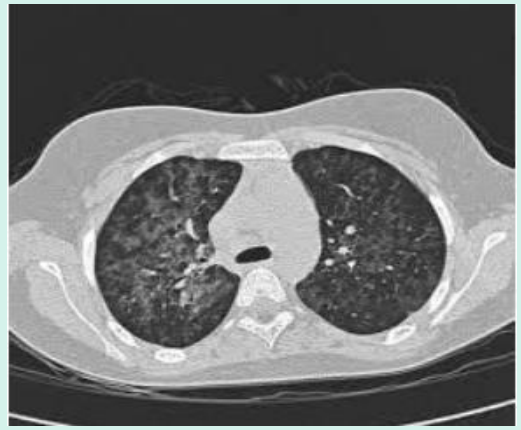
Chest US → B lines.

- The gold standard for the diagnosis is: High Resolution CT Scan.

HYPERSENSITIVITY PNEUMONITIS





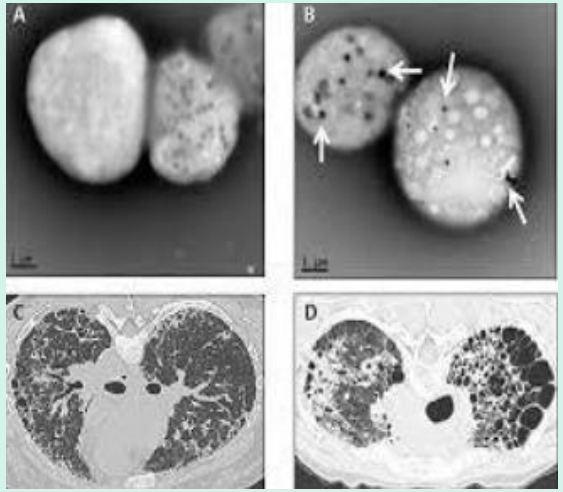


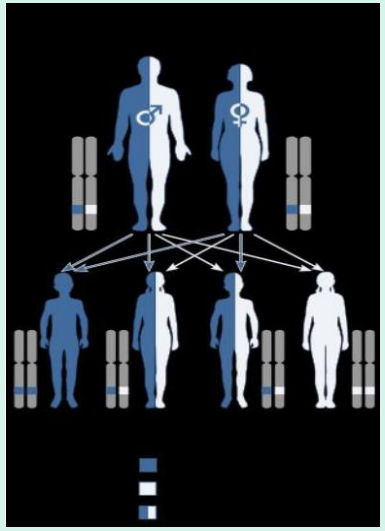
- Spares lower lobes due to allergens (birds), since anything you inhale usually goes to the upper part of the lung.
- Diseases affecting upper lobes: Sarcoidosis + Ankylosing Spondylitis+ Hypersensitivity Pneumonitis
- Diseases affecting lower lobes: Asbestosis + Connective tissue disorders + IPF.

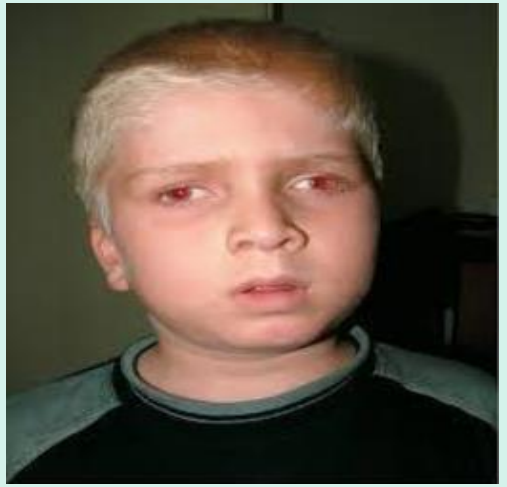
VELCRO CRACKLES

- Fine end inspiratory crackles.
- Indicates Scarring of the lung parenchyma

HERMANSKY PUDLAK SYNDROME

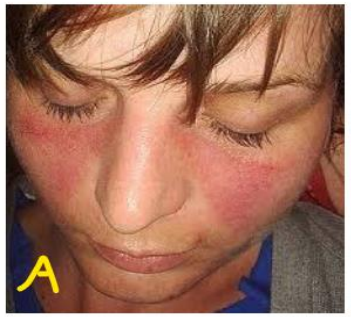


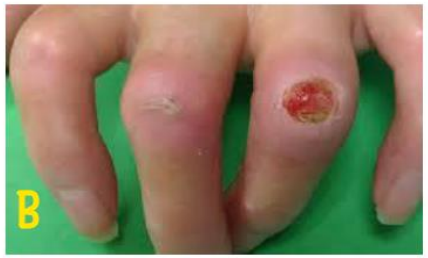


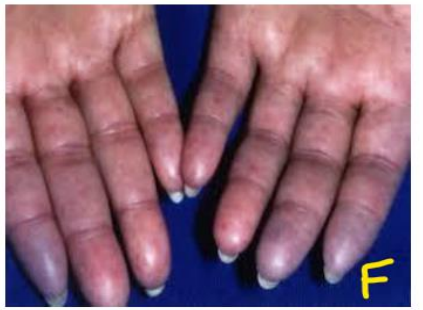


- A hereditary type of Pulmonary fibrosis.
- ** EXTRA** Characterized by a condition called Oculocutaneous albinism, which causes abnormal light coloring (pigmentation) of Skin, Hair, and Eyes.
- Presentation → young patients with white hair.
- Common in Puerto Rico
- It causes diffuse Fibrosis, Platelet dysfunction, and Albinism.

CONNECTIVE TISSUE RELATED ILD





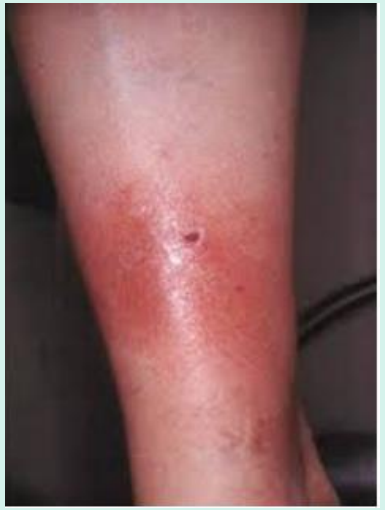


- Rash: indicates ILD when accompanied by respiratory symptoms.
- A → Rash over the cheeks, sparing Nasolabial folds (LUPUS).
- B → Scleroderma (digital ulcer).
- C → Rheumatoid Arthritis.
- D → Oral ulcer in a patient with Lupus.
- E → Scleroderma with small mouth, peaked nose and very tight skin.
- F → Digital Ischemia in a patient with Scleroderma.

LAB TESTS

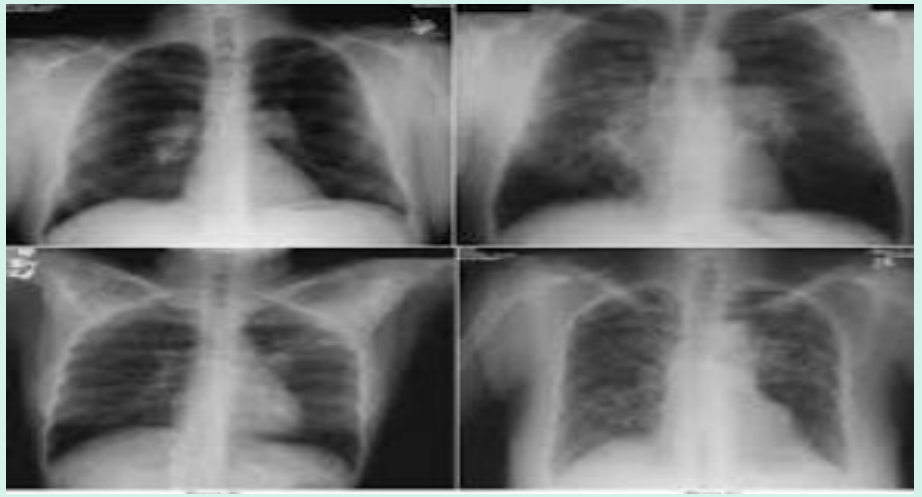
- RF(rheumatoid factor)
- ANA(anti nuclear antibody).
- Anti DS –DNA(Double stranded DNA)
- ENA (Extranuclear antibody).

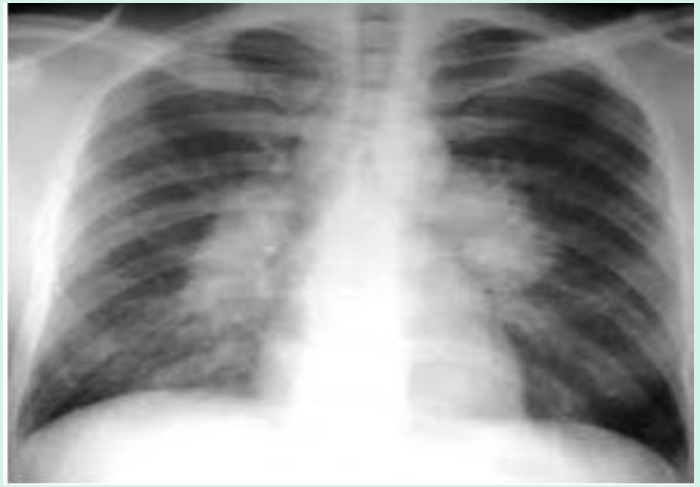
SARCOIDOSIS

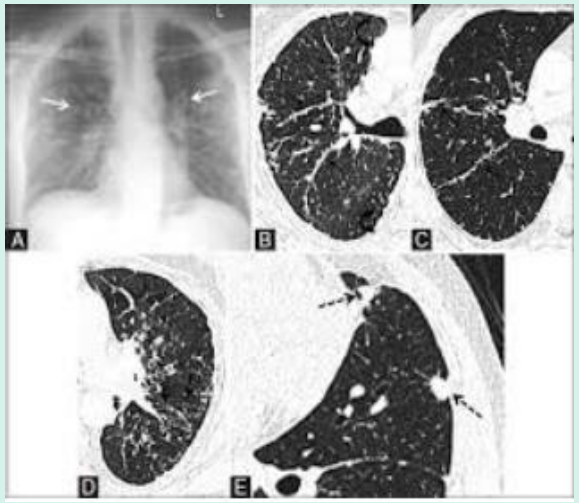


- **EXTRA** a disease characterized by the growth of tiny collections of inflammatory cells (Granulomas) in any part of our body.
- Accompanies Lung Fibrosis.
- Notice the redness of the sclera (Scleritis), as well as the rash on both upper and lower limbs.
- Labs: Serum ACEI Level + Hypercalcemia

SARCOIDOSIS







- CXR findings:

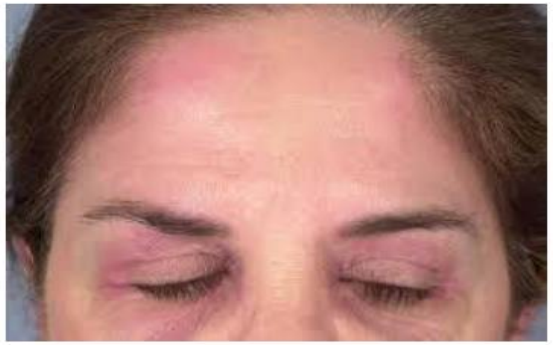
1- Symmetric Hilar and Mediastinal Lymphadenopathy.

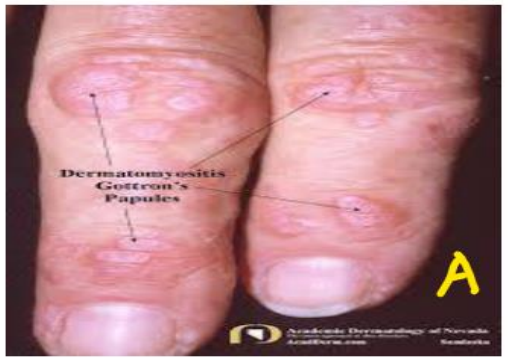
2- Reticulonodular opacities.

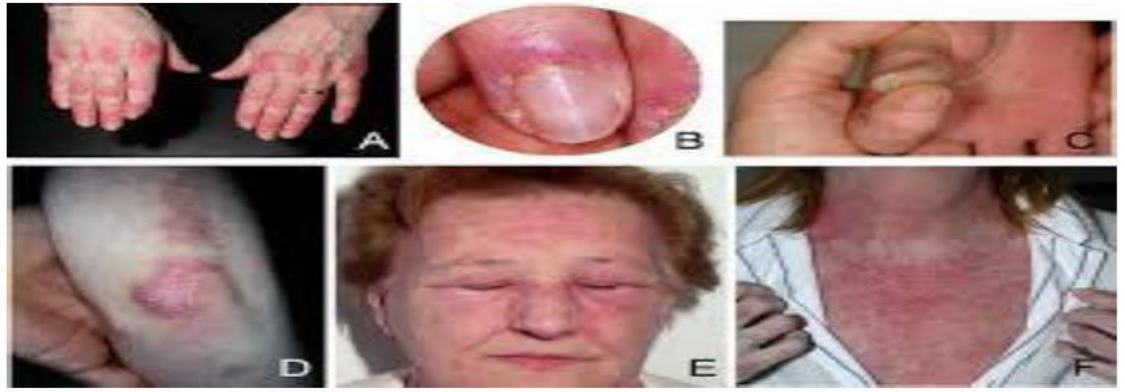
3- Nodules.

4- Pulmonary Fibrosis (mostly at upper and middle lung zones)

DERMATOMYOSITIS / POLYMYOSITIS



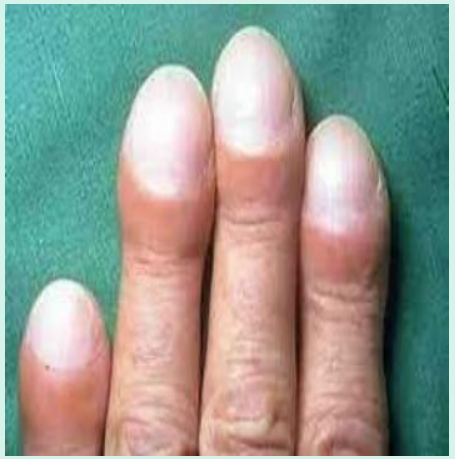




- A → Gottron's papules
- B → Digital Telangiectasia
- C → Mechanics Hands
- D → Calcinosis
- E → Photosensitive rash in Dermatomyositis (Involves the Forehead and Nasolabial Areas.
- F → V sign
- Labs: ANA (Antinuclear antibody) + ENA (Extranuclear antibody) + Myositis Panel.

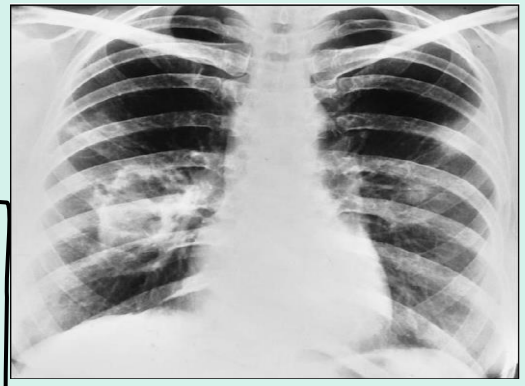
IDIOPATHIC PULMONARY FIBROSIS (IPF)

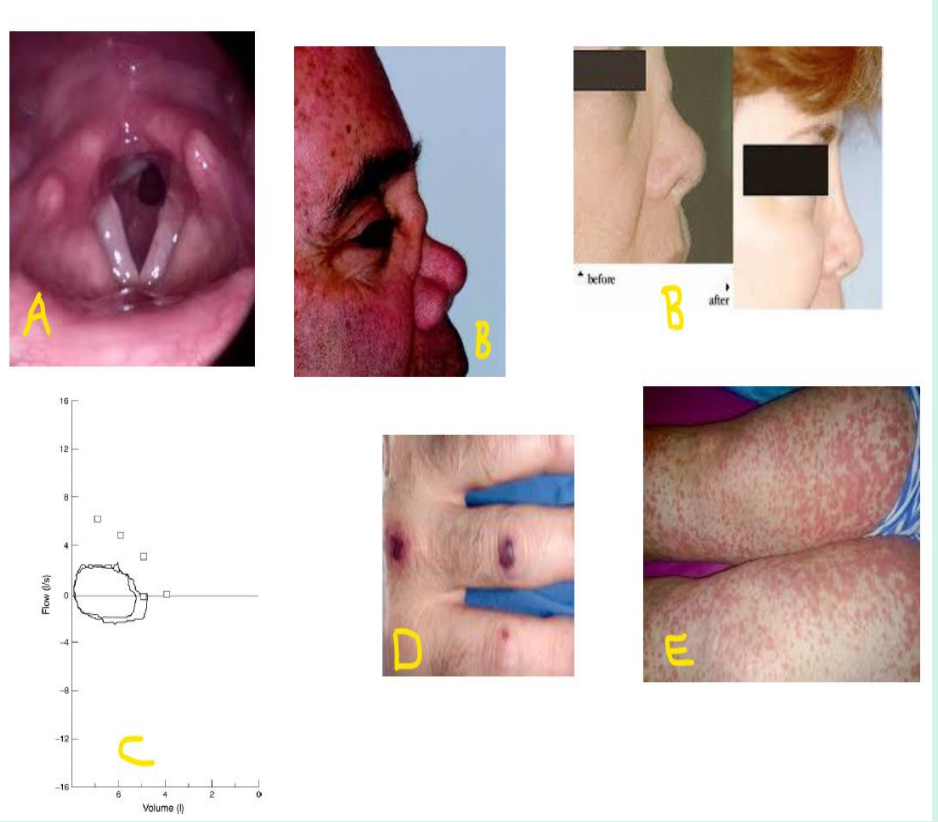




- Histologically know as Usual Interstitial Pneumonia (UIP)
- The most common form of ILD.
- Presentation: Old age man with progressive dyspnea (over 6 months), dry cough, and digital clubbing, No history of Smoking.

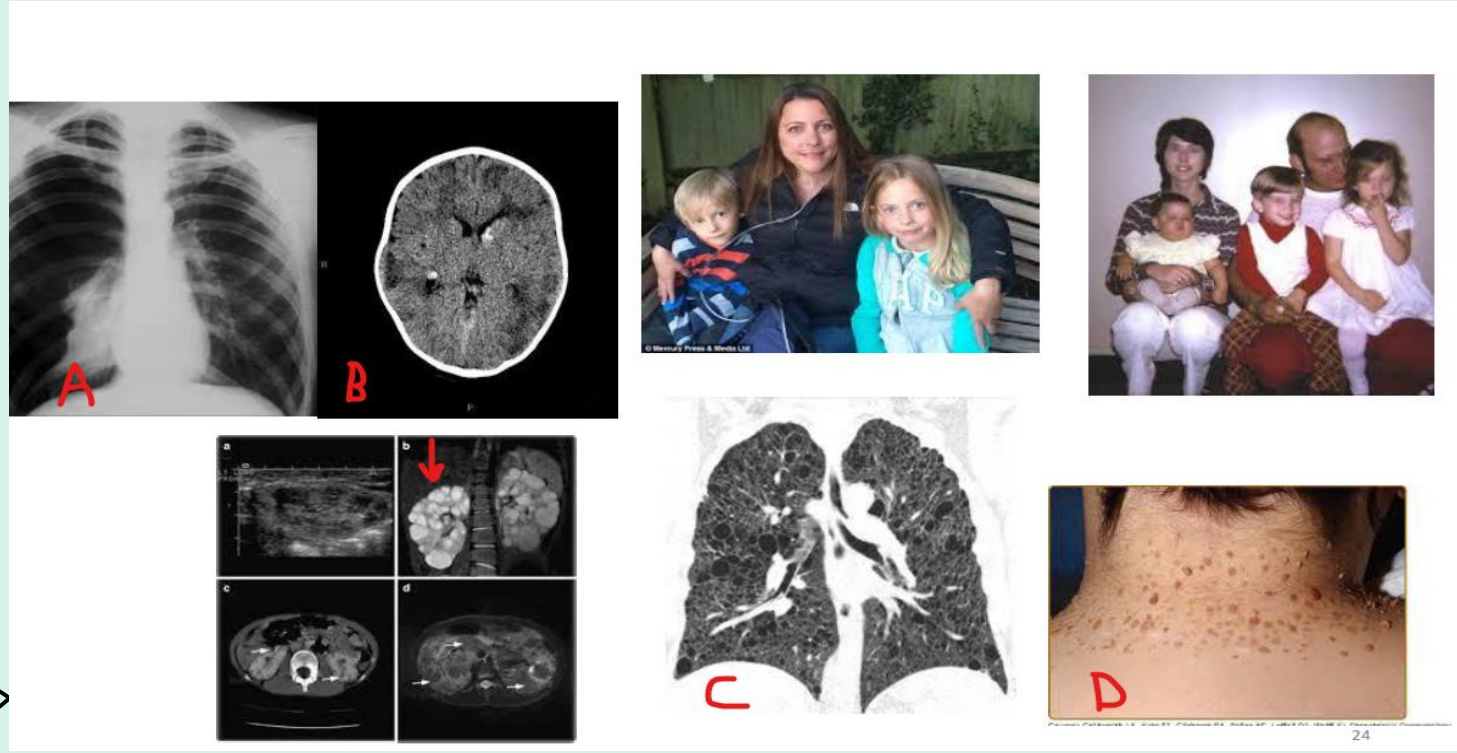
GRANULOMATOSIS WITH POLYANGIITIS (WEGENER'S GRANULOMATOSIS)





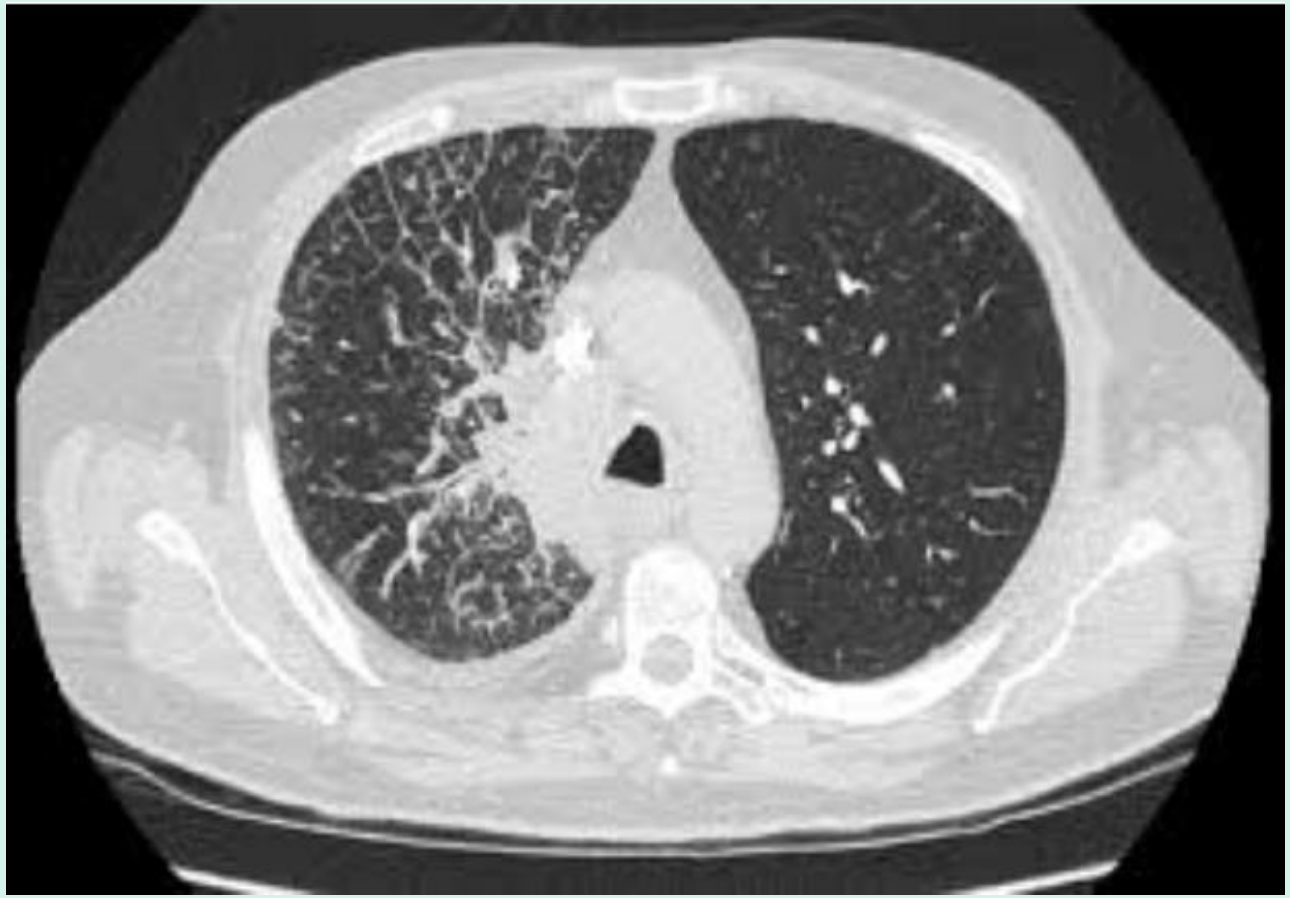
- A type of vasculitis with fibrosis.
- A → Vocal cords; Subglottic Stenosis
- B → Saddle nose deformity (destruction of the nasal septum).
- C → Box Shape PFTs, due to impairment of inspiration and expiration.
- D → Digital Infarction
- E → Purpuric rash.
- Labs: ANCA (Anti neutrophilic cytoplasmic antibodies).

LYMPHANGIOLYOMYOMATOSIS

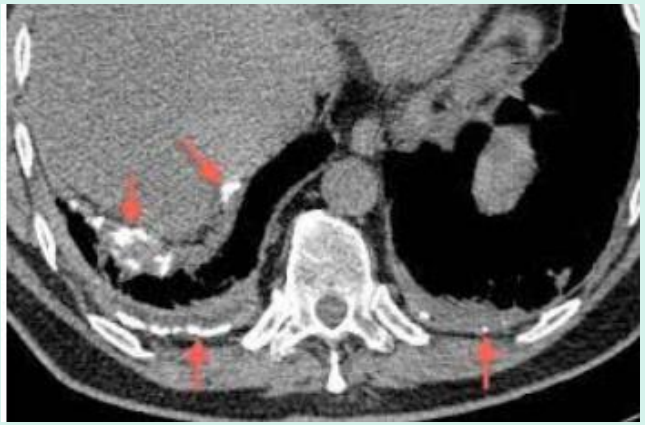


- Rare cause of fibrosis, mainly enlargements of the smooth muscle of lymphatics wall.
- Mostly affects females with history of epilepsy, they will develop thin cyst that when ruptures leads to pneumothorax.
- Presentation: Middle aged lady on O2 therapy with Pneumothorax and Angioma.
- A → Pneumothorax
- B → Problem in the brain (epilepsy)
- Red Arrow → Angioma
- C → Thin cysts Variable in size
- D → Skin lesions

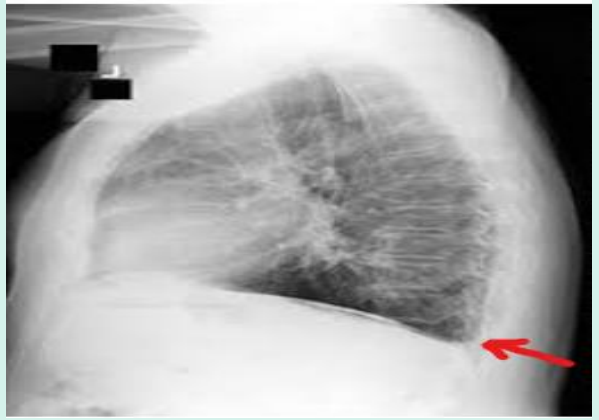
LYMPHANGITIS CARCINOMATOSIS



ASBESTOSIS



A



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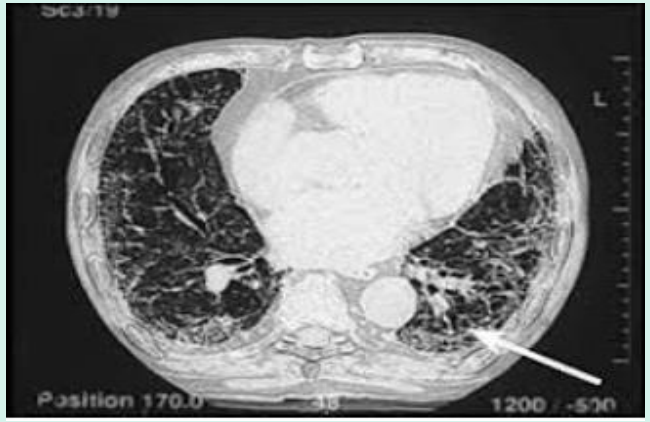
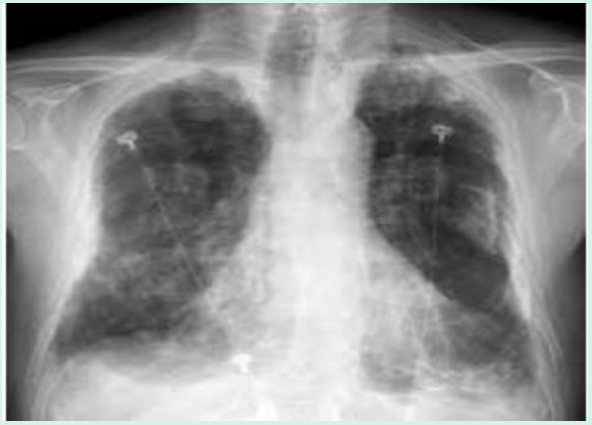
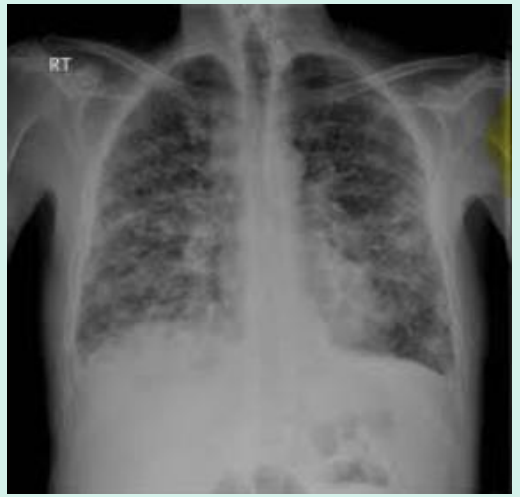


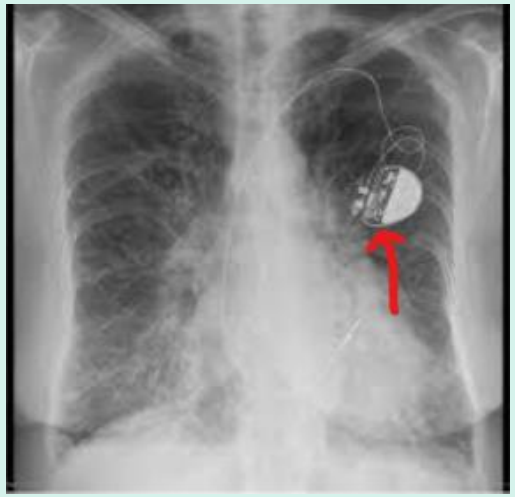


- A → Arrows show asbestos plaques deposits on pleura and diaphragm.
- B → Plaque on X-ray (in the lower zone).

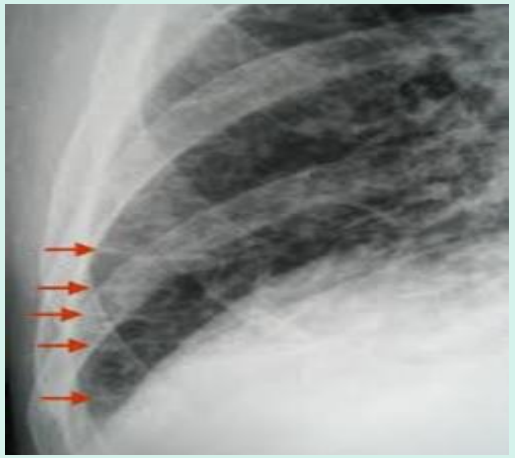
CONGESTIVE HEART FAILURE



A



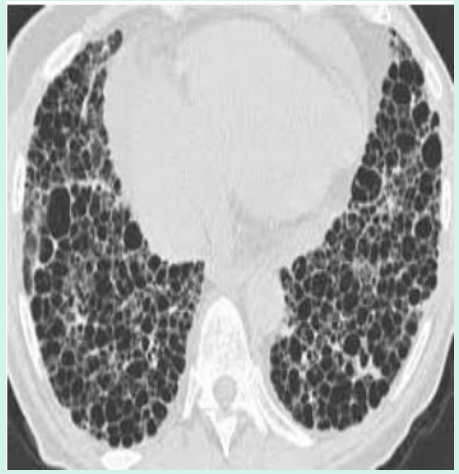
B

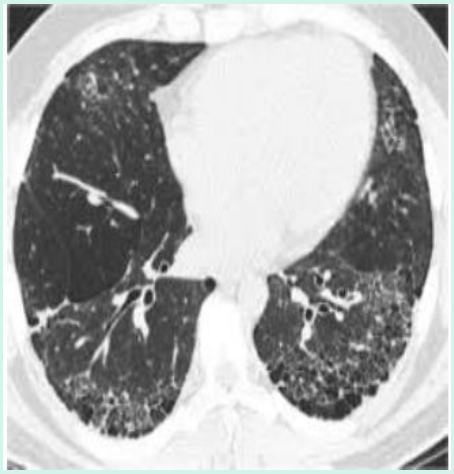


C

- Chronic interstitial edema with Heart failure and edema in the lung.
- A → Bilateral pleural effusion
- B → Pacemaker.
- C → Red arrows are pointing towards B lines (a kind of comet-tail artifact indicating subpleural interstitial edema)

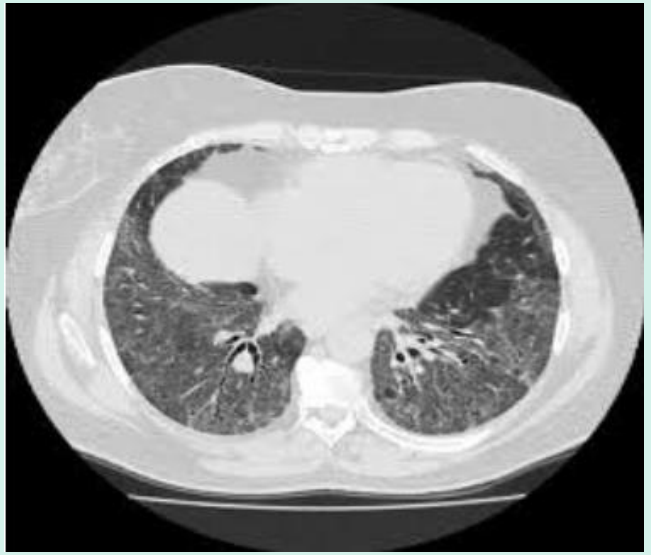
HONEYCOMBING

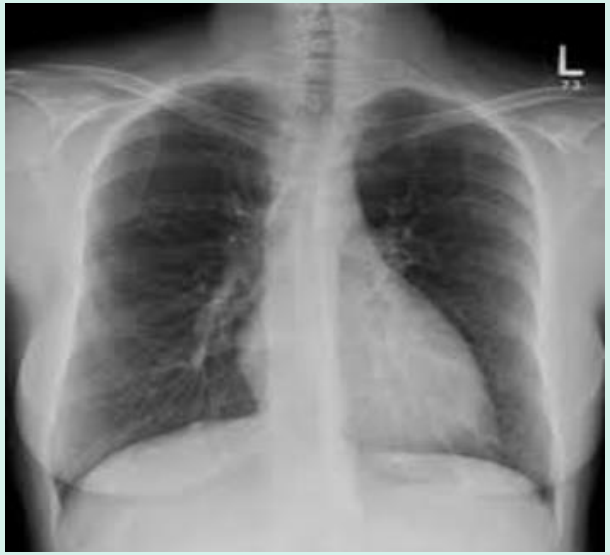




- A characteristic of IPF.
- Seen with any advanced Lung Fibrosis.

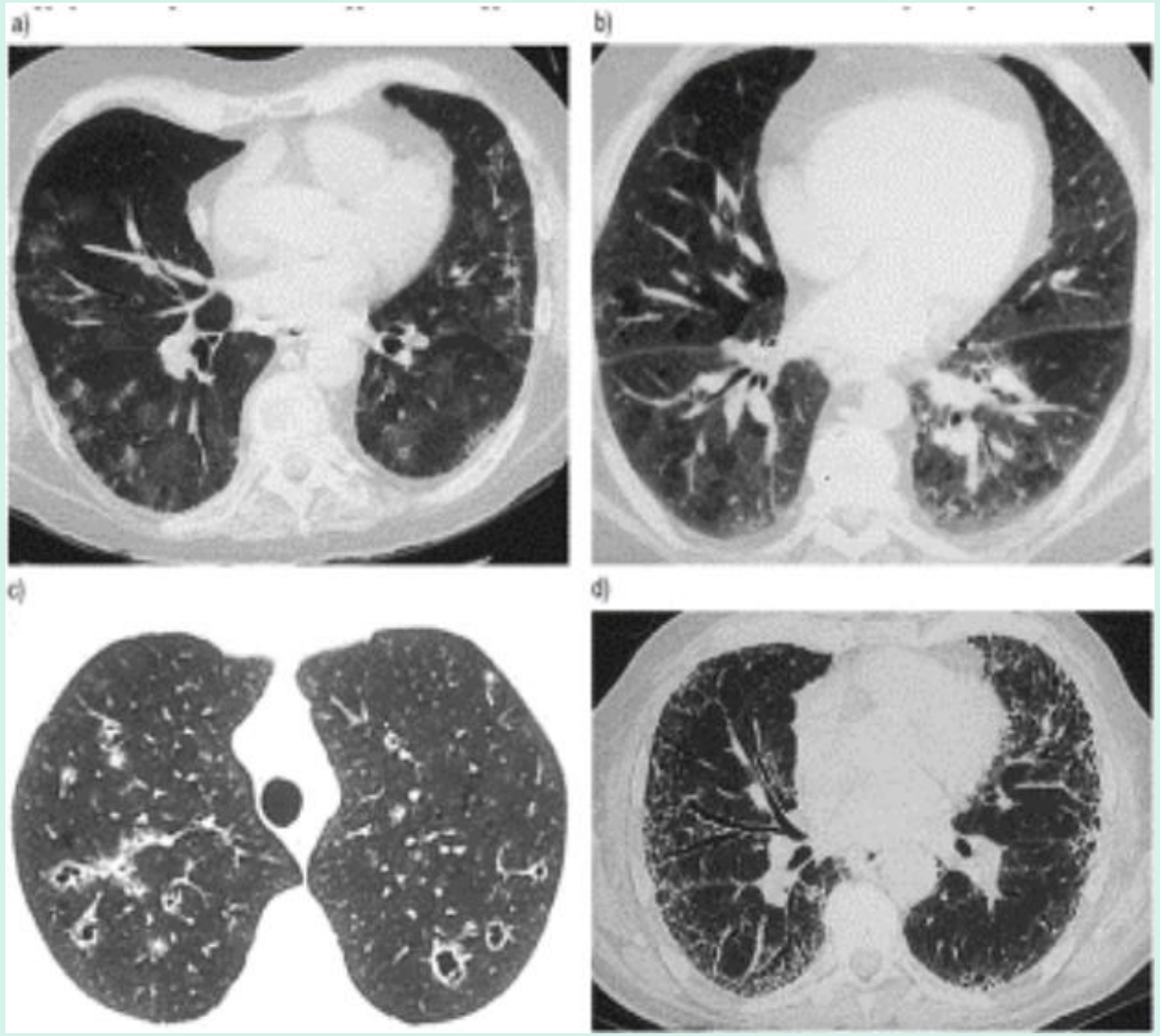
NONSPECIFIC INTERSTITIAL PNEUMONIA / GROUND GLASS APPEARANCE



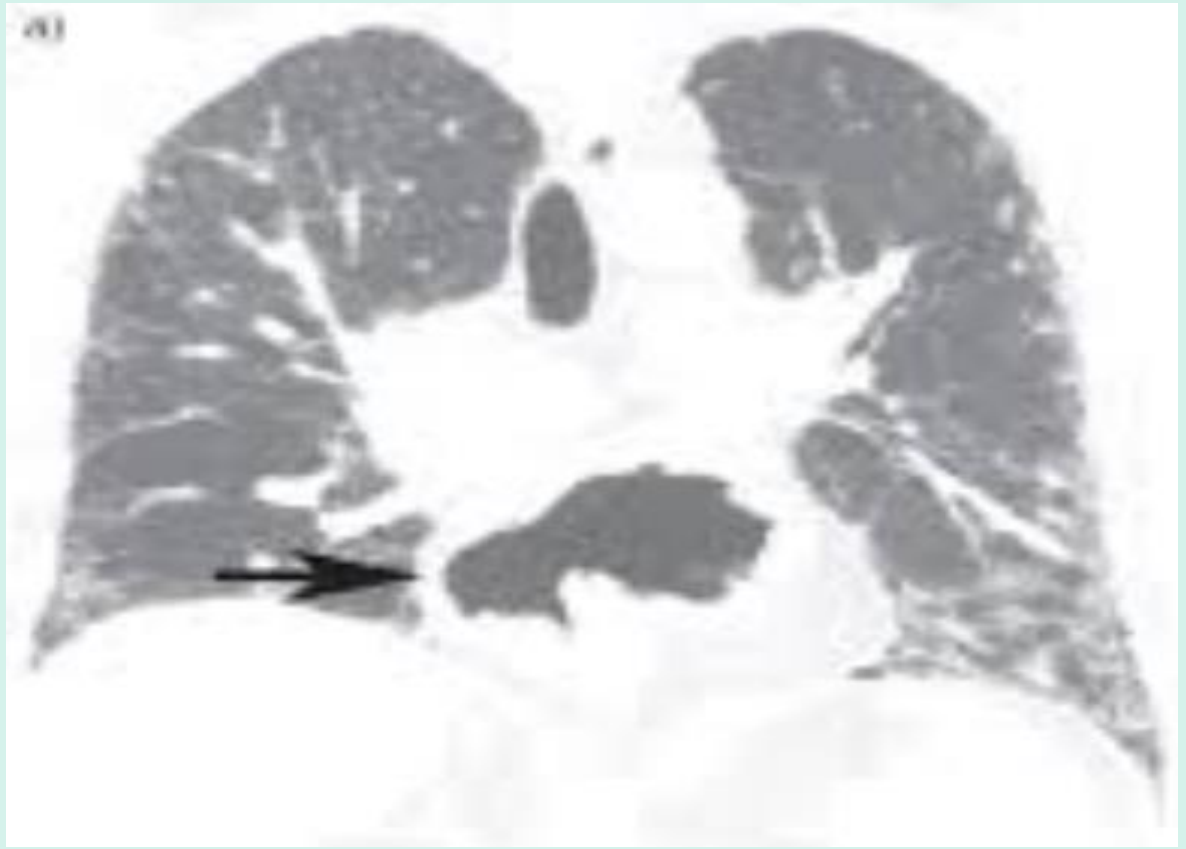


- In NSIP, we have Ground glass opacity instead of Honeycombing.

SMOKING RELATED INTERSTITIAL LUNG DISEASE

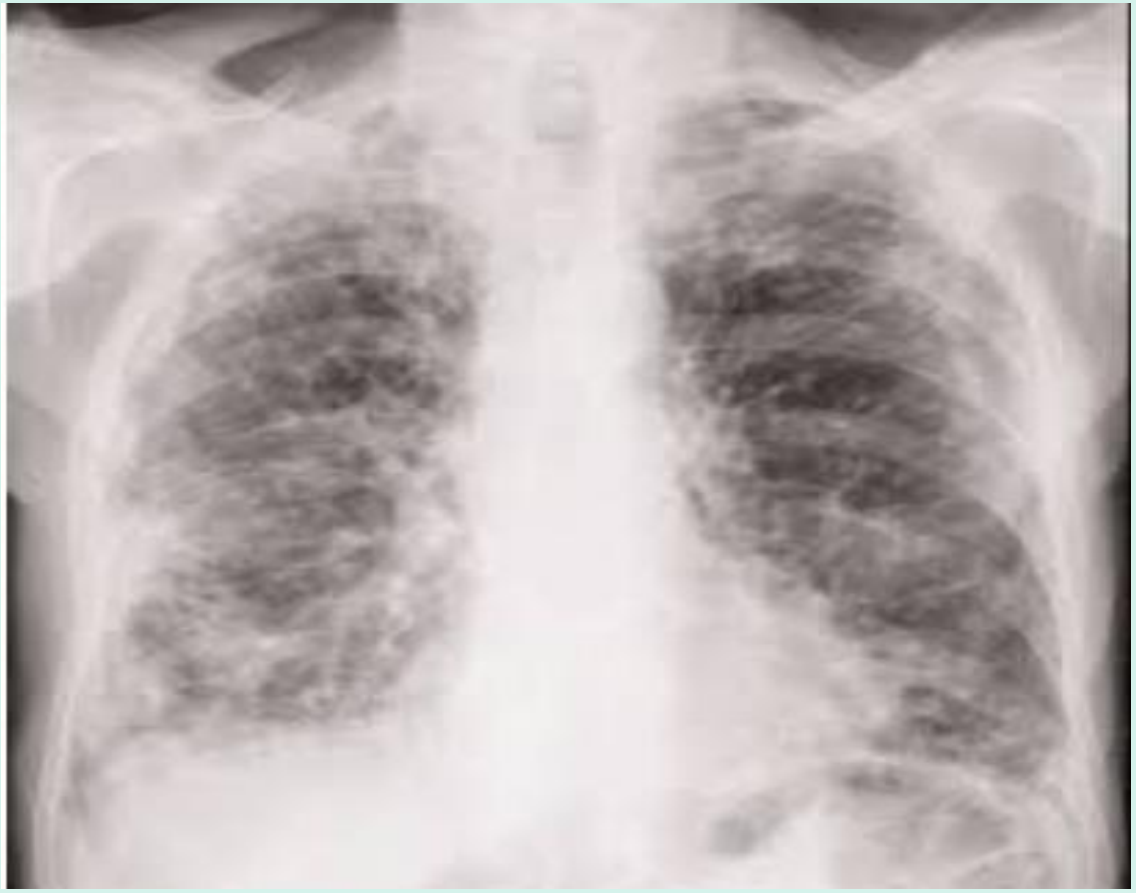


DIAGNOSIS??



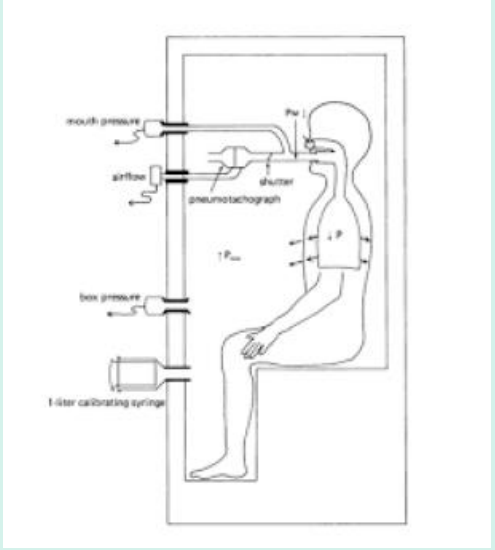
- Fibrosis + Hiatus Hernia + Micro-aspirations / reflux.

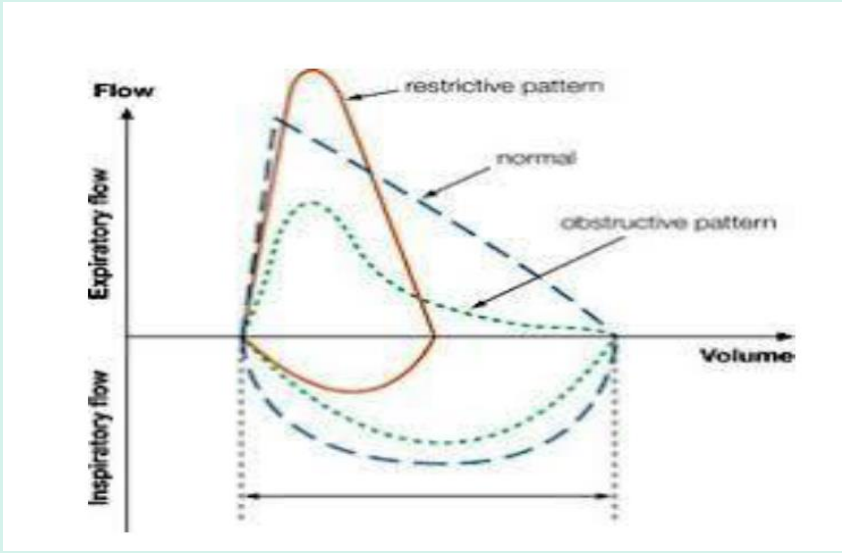
DRUG / RADIATION INDUCED ILD



- Diagnosed with proper History, and after excluding other causes

LUNG VOLUMES IN ILD





- Findings in Fibrosis:

1- Reduced Total Lung Capacity.

2- Low FVC

3- Normal FEV1 / FVC ratio

- Mainly Restrictive patterns.

ASTHMA

ASTHMA

it's a chronic disease characterized by recurrent attacks of shortness of breath and wheezing.

Heterogenous disease, usually characterized by airway inflammation

History of respiratory symptoms such as Wheezes, Chest tightness, Cough, and SOB that vary over time and in intensity, together with variable expiratory flow limitation.

Usually is associated with airway hyperresponsiveness and inflammation, but these are not necessary or sufficient to make a diagnosis

May become worse during physical activity or at night.

It begins early in life

Risk factors: Atopic disease, Recurrent wheezing, parental history of asthma and smoking.

Pathophysiology: Airway inflammation + Intermittent airflow obstruction + Bronchial Hyperresponsiveness.

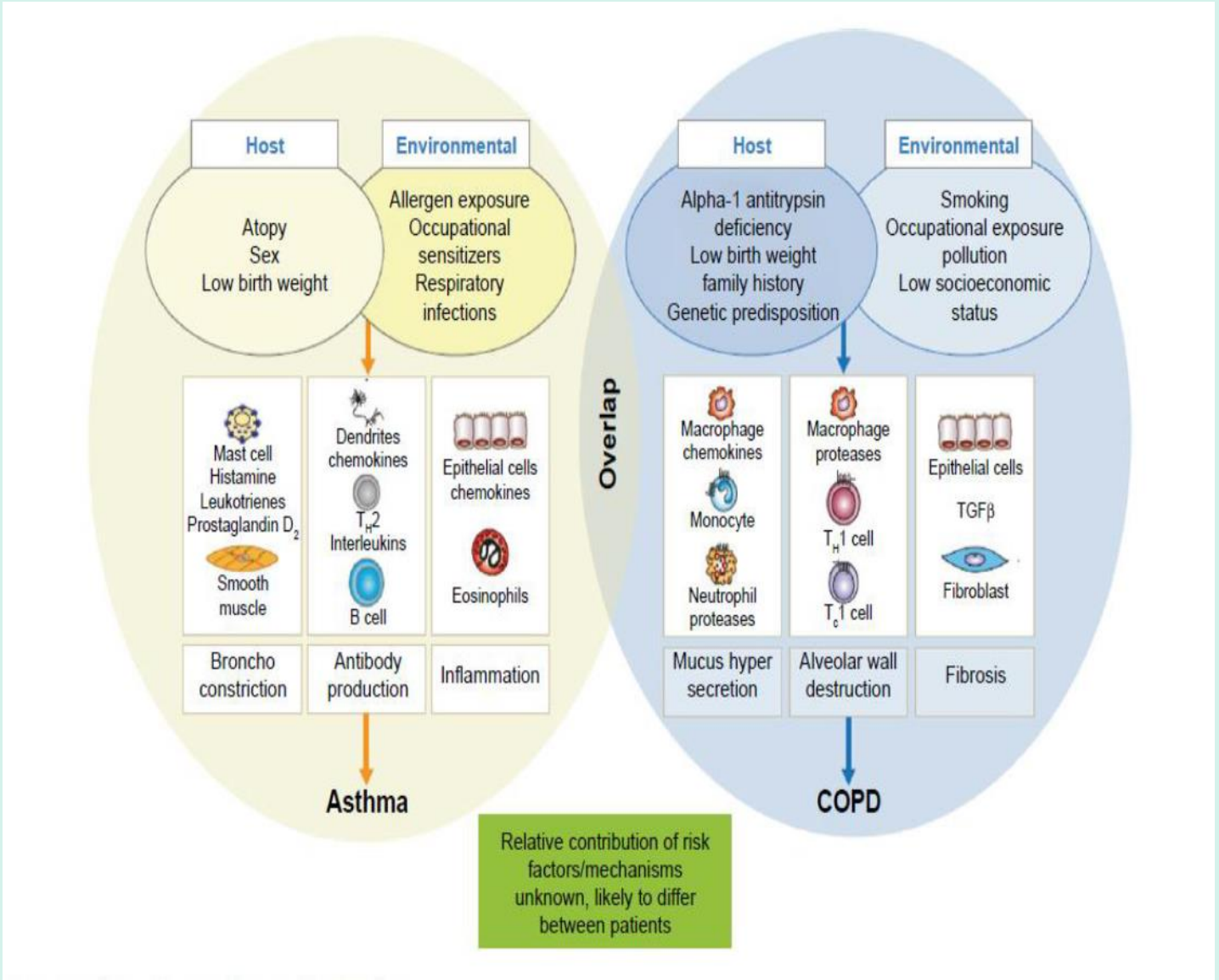
ASTHMA PHENOTYPES

Recognizable clusters of demographic, clinical and pathophysiological characteristic are often called Asthma Phenotypes.

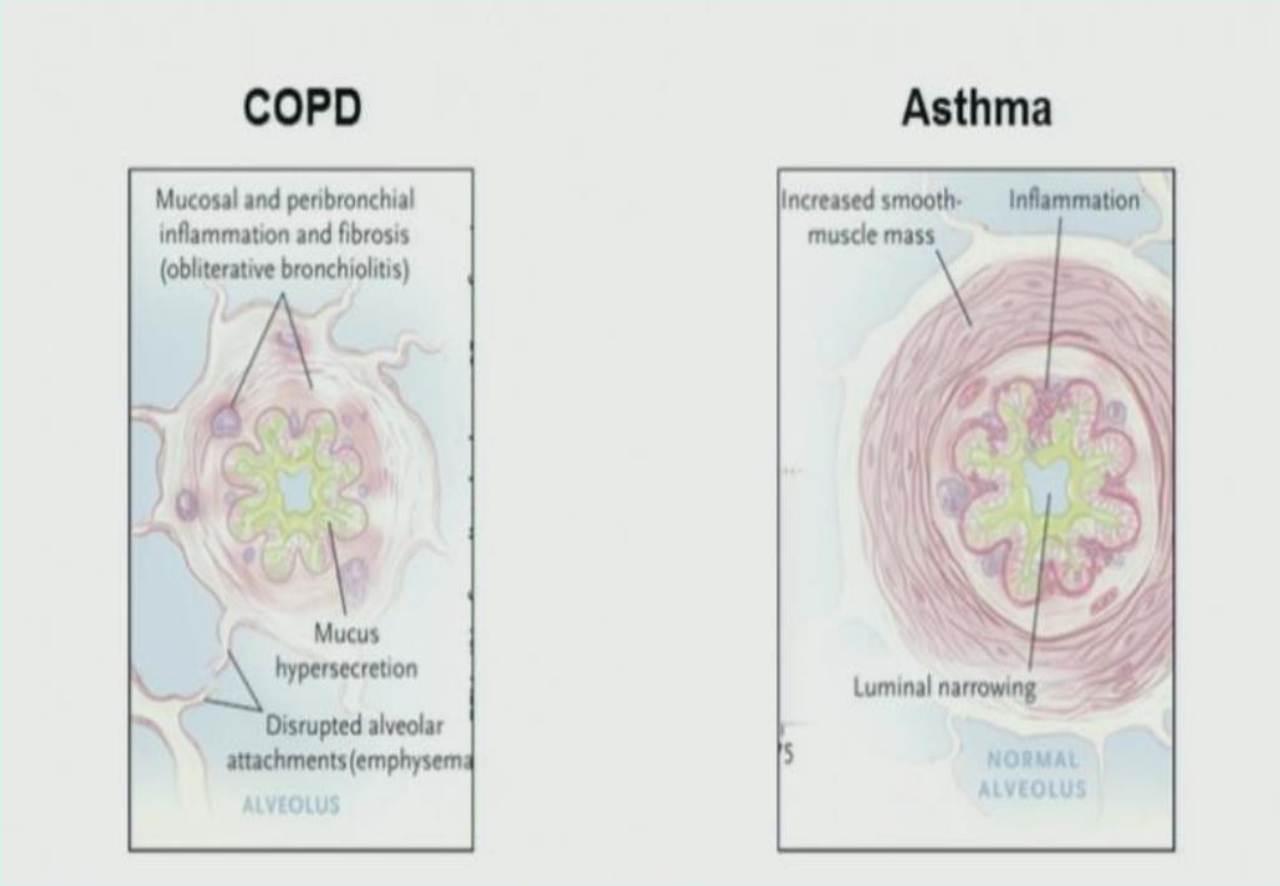
These don't correlate strongly with specific pathological processes or treatment responses.

Asthma Phenotypes: Allergic Asthma / Non-Allergic Asthma / Adult-Onset Asthma / Asthma with Persistent Airway Inflammation / Asthma with Obesity.

ASTHMA VS COPD



ASTHMA VS COPD



- COPD → Obliterative Bronchiolitis + Mucous Hypersecretion + Emphysema
- ASTHMA → Increased Smooth muscle mass + Inflammation + Luminal Narrowing

ASTHMA CLASSIFICATION

CLASSIFY SEVERITY Clinical Features before Treatment			
	Symptoms	**Nocturnal Symptoms**	**FEV_1 or PEF**
STEP 4 Severe Persistent	Continuous Limited physical activity	Frequent	< 60% predicted Variability > 30%
STEP 3 Moderate Persistent	Daily Attacks affect activity	> 1 time week	60 to 80% predicted Variability > 30%
STEP 2 Mild Persistent	> 1 time a week but < 1 time a day	> 2 times a month	> 80% predicted Variability 20 to 30%
STEP 1 Intermittent	< 1 time a week Asymptomatic and normal PEF between attacks	< 2 times a month	> 80% predicted Variability < 20%

- The severity of Asthma is classified as: Intermittent / Mild persistent / Moderate persistent / Severe persistent.
- Patients with asthma of any level of severity may have mild, moderate, or severe exacerbations.
- The presence of one severe feature is sufficient to diagnose severe persistent asthma.

CASE #1

History

- 40-year-old married lady , works as a teacher .
- A known case of bronchial asthma on salbutamol PRN only!!.
- Presented with S.O.B on exertion , chest tightness , wheezes that worse during night for the last 3 weeks.
- Headache that worse on leaning forward, Mild feeling of hotness . no chills or rigor
- She has nasal blockage , with no reflux symptom
- Seasonal allergy , family history and social history un-remarkable
- Nonsmoker , no Pets

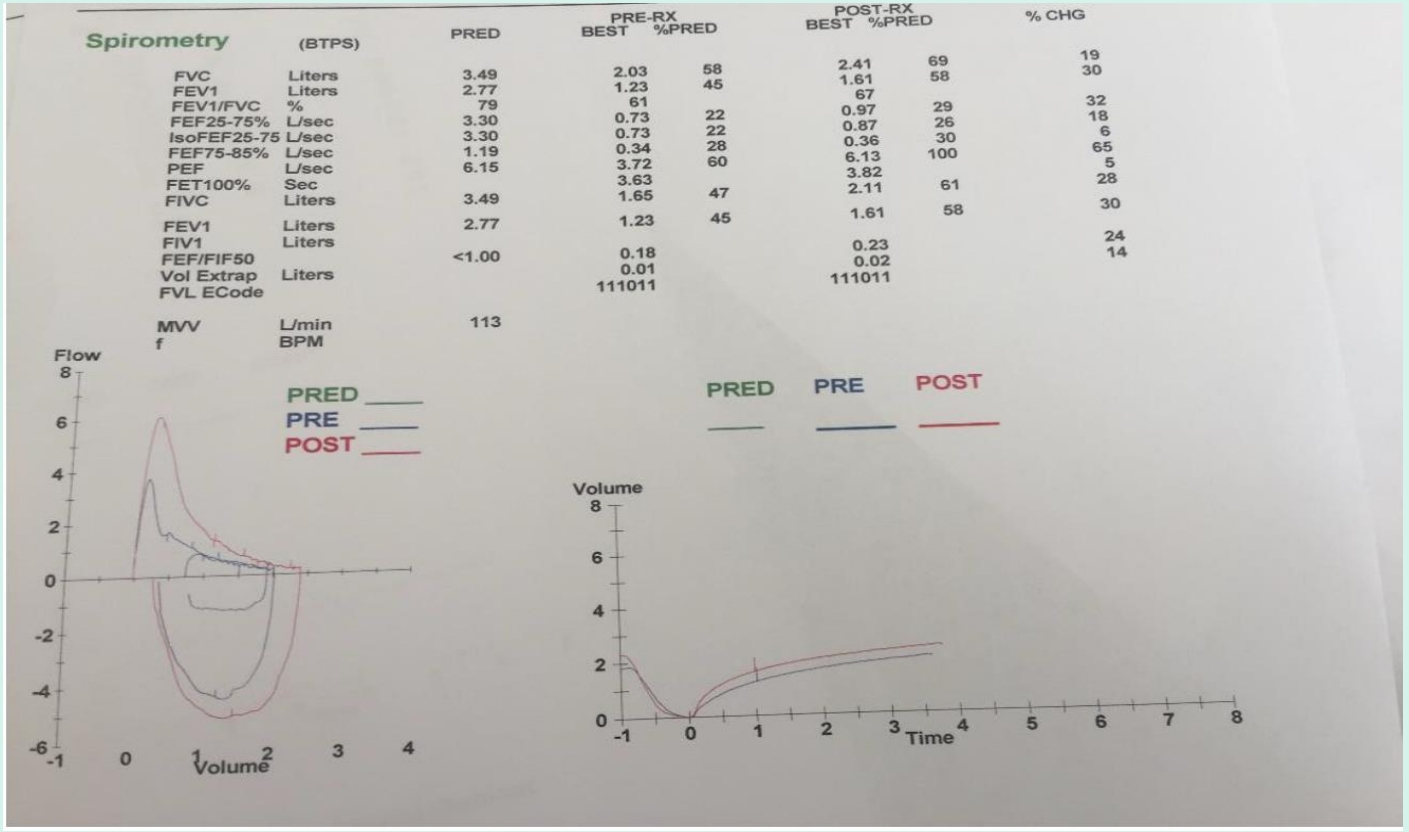
Physical exam

- HR:110 beats/min, RR 26 other vitals stable, O2% : 94

- **Upon auscultation**:

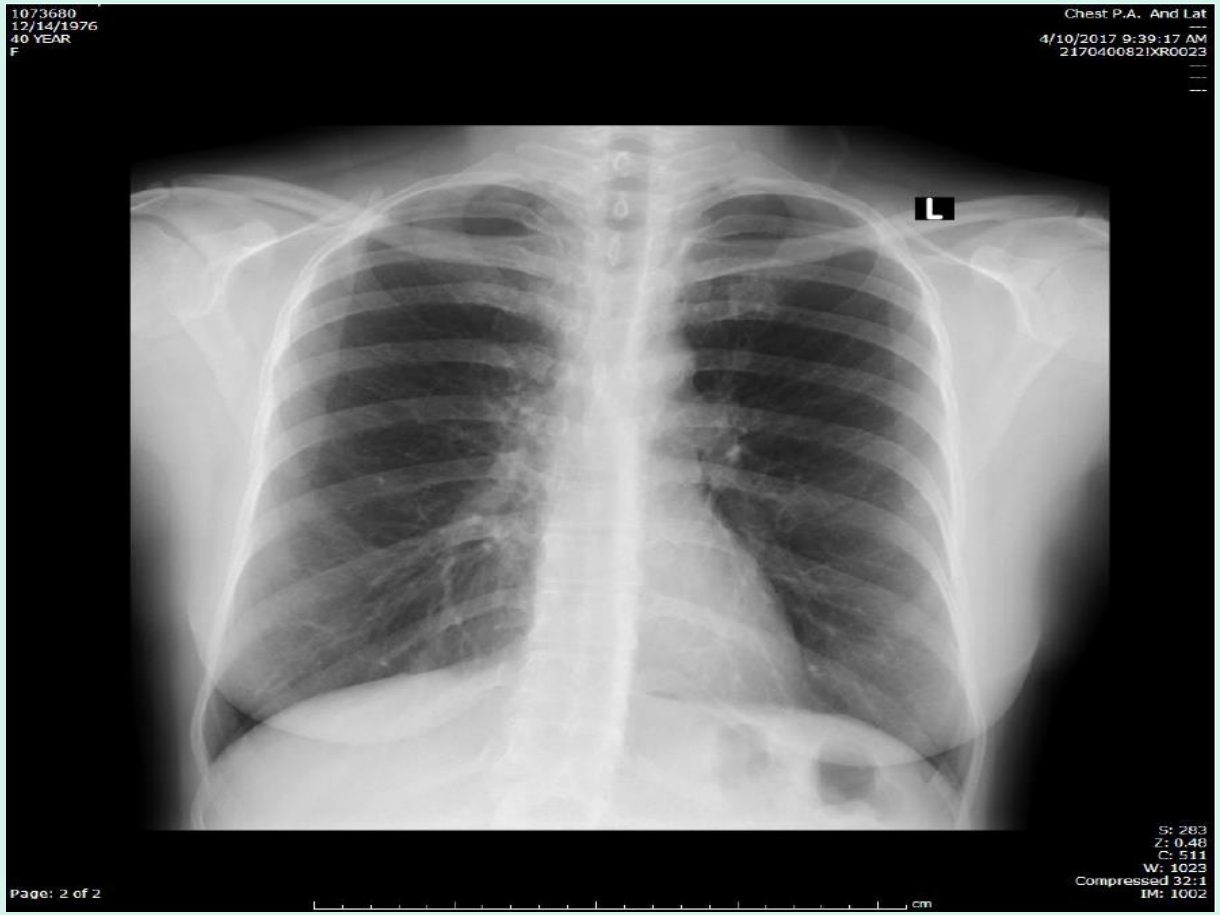
- Decrease air entry bilaterally
- Prolong expiration
- Bilateral expiratory and inspiratory wheezes , no other added sound

CASE #1 / PFT



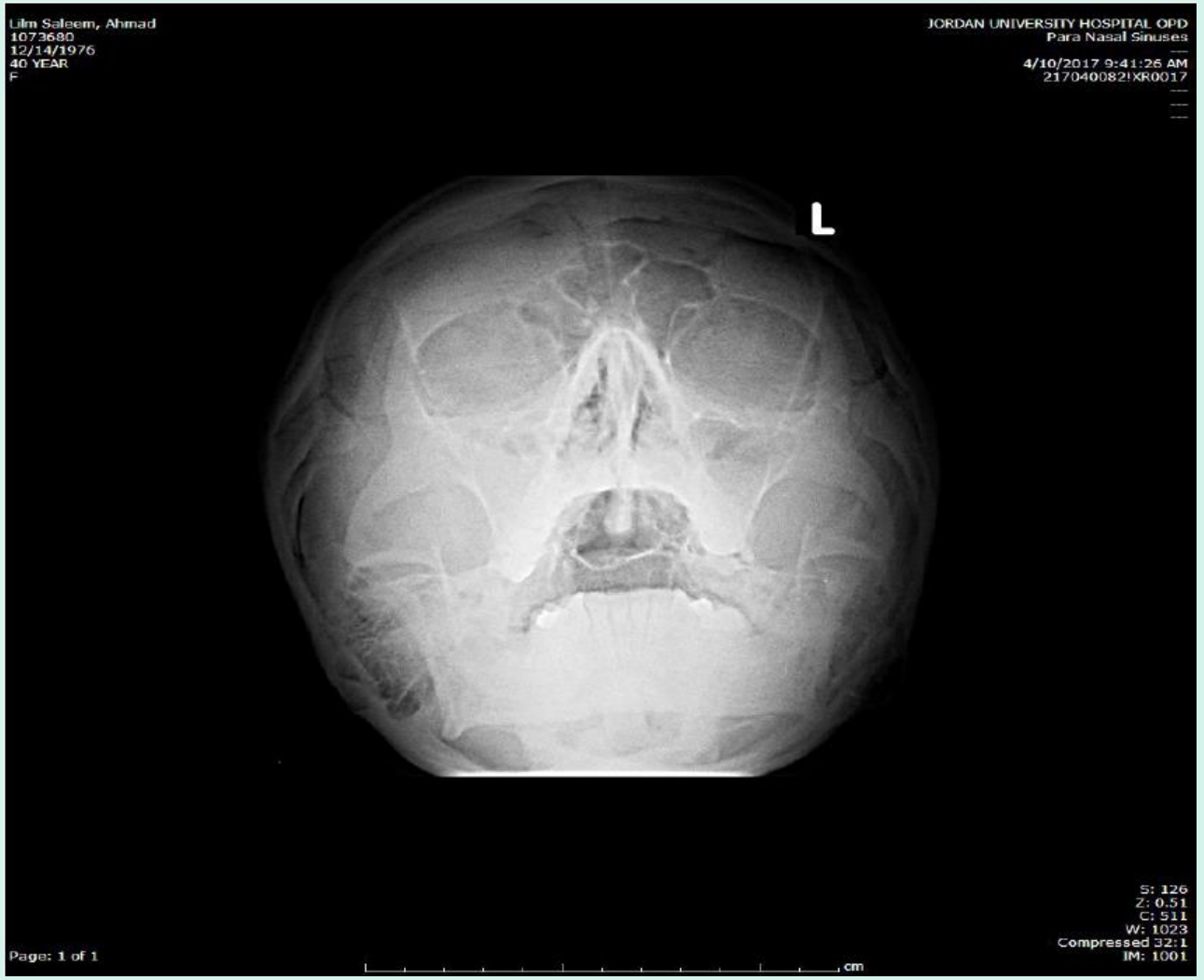
- Establish Asthma diagnosis.
- Prior to initiating treatment
- Should include measurements before and after inhalation of short acting bronchodilator.
- Reduced FEV 1 / FVC → Airway obstruction
- Reversibility: Increase of 12% and 200 ml after the administration of Sort-acting bronchodilator.

CASE #1 / CXR



- Reveals complications.
- Normal or Hyperinflation.
- Exclude Pneumothorax or Pneumomediastinum.

CASE #1 / PARANASAL SINUSES X-RAY



CASE #1 / MANAGEMNET

Management

- **Educational** :
- Explain to the patient about her diagnosis
- Inhaler education
- Asthma action plan

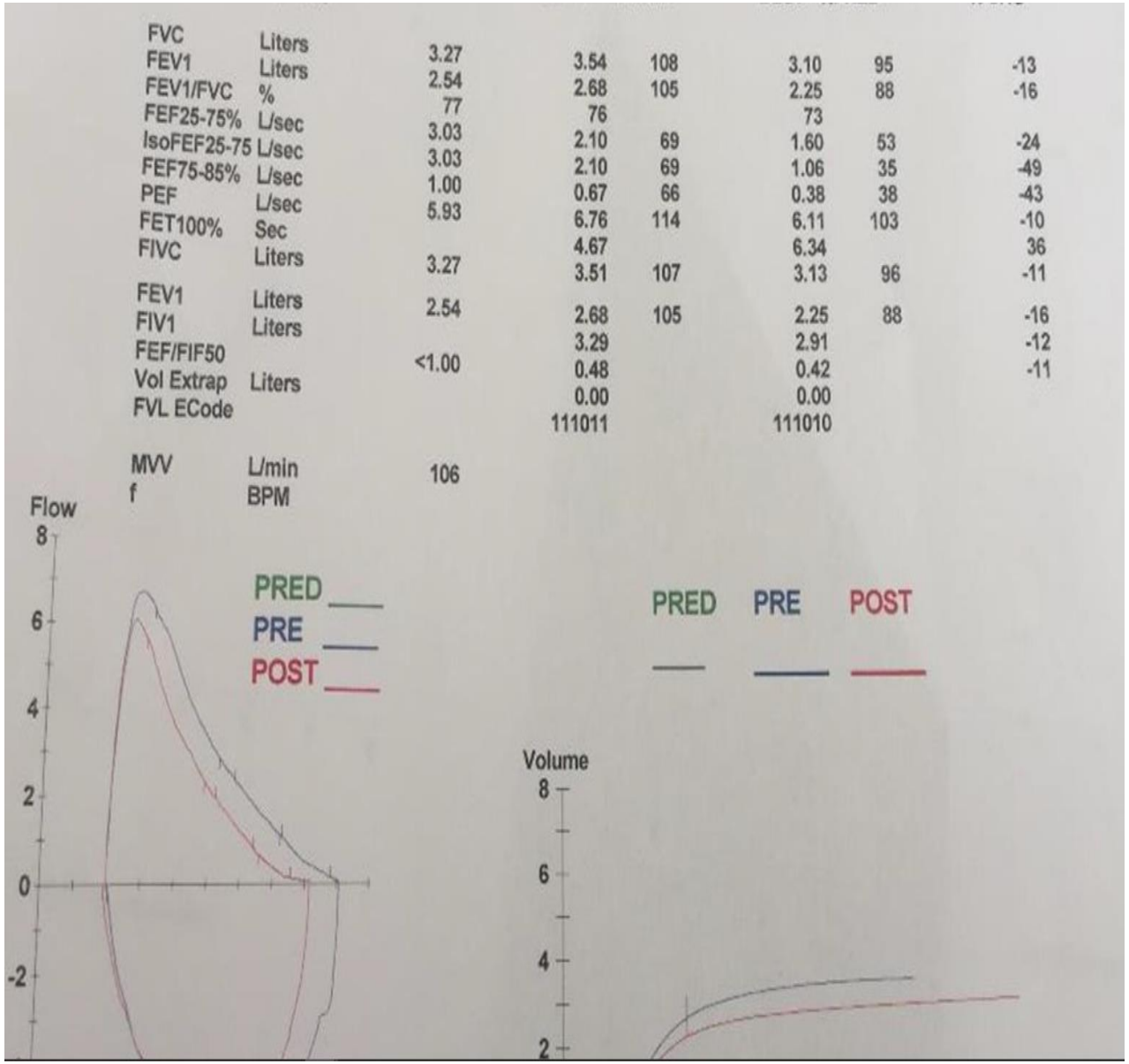
- Patient started on prednisone 50 mg P.O 1*1 for 5 days

- Budesonide/formoterol(160/4.5 2 puffs twice a day) and PRN .
- Oral antibiotics to treat sinusitis
- Rhinocort N.S 1*2
- Antihistamine
- RTC after 1 week

- One week follow up , patient improved on treatment

- Continue on Budesonide/formoterol(160/4.5 2 puffs twice a day) and PRN for 3 months with follow up appointment

CASE #1 / POST-TREATMENT PFT



FVC	Liters	3.27	3.54	108	3.10	95	-13
FEV1	Liters	2.54	2.68	105	2.25	88	-16
FEV1/FVC	%	77	76		73		
FEF25-75%	L/sec	3.03	2.10	69	1.60	53	-24
IsoFEF25-75	L/sec	3.03	2.10	69	1.06	35	-49
FEF75-85%	L/sec	1.00	0.67	66	0.38	38	-43
PEF	L/sec	5.93	6.76	114	6.11	103	-10
FET100%	Sec		4.67		6.34		36
FIVC	Liters	3.27	3.51	107	3.13	96	-11
FEV1	Liters	2.54	2.68	105	2.25	88	-16
FIV1	Liters		3.29		2.91		-12
FEF/FIF50		<1.00	0.48		0.42		-11
Vol Extrap	Liters		0.00		0.00		
FVL ECode			111011		111010		
MVV	L/min	106					
f	BPM						

CASE #2

History

- A 39 year old single female who is known case of resistant severe asthma for last 10 years on the following treatment :

1. Budesonide/formoterol(160/4.5 2 puffs 4 times a day)
2. Tiotropium caps once a day
3. Montelukast 10mg once a day
4. Theophylline SR 300 mg once a day
5. Omeprazole 40 p.o once a day
6. Multiple courses of oral prednisone (3-4 times a year)

- Still patient complains of attacks of S.O.B , wheezes and chest tightness

and not controlled on the above treatment.

- She is also a known case of non-specific colitis (labeled as ulcerative colitis on mesalamine)

- Symptoms partially controlled on treatment with exacerbation of her colitis .
- She is also a known case of hypertension(controlled on treatment)

And history of provoked DVT 2 years ago treated for 6 months.

- Recurrent visit to chest clinic because of uncontrolled symptoms .

CASE #2 / WORK-UP AND MANAGEMENT

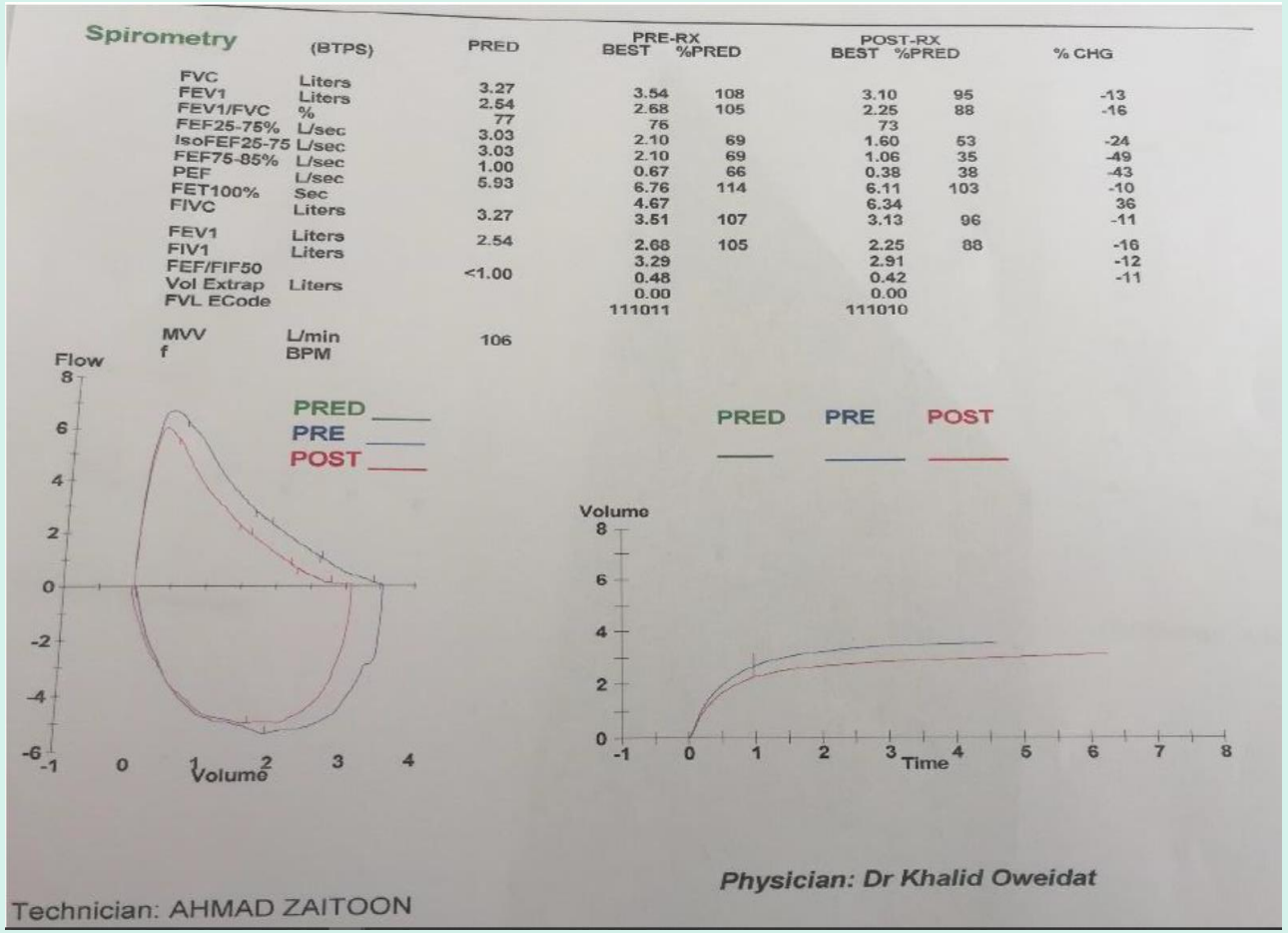
Work up and management

- Inhalers technique were checked
- Eosinophil count , IgE level, HRCT (no ABPA), bronchoscopy (no vocal cord paralaysis or endobronchial lesion, connective tissue work up and ANCA were negative.
- However , her spirometry stable!!!!

- Should be treatment by asthma specialist
- She has severe persistent asthma(5-10%)
- Think of immunetherapy treatment

- Anti IgE
- Anti IL 5
- Course of azithromysin
- Anti TSLP (thymic stromal lymphopoietin) antibody

CASE #2 / PFT



- Remember that Asthma can have normal Spirometry.

BRONCHIECTASIS

BRONCHIECTASIS

A chronic respiratory disease characterized by permanent and abnormal dilation of the bronchi and bronchioli (medium sized airways) which destruct muscular and elastic components of bronchial walls (because of that, Dilation is permanent).

Can be caused by airway obstruction, host defenses, and drainage effects.

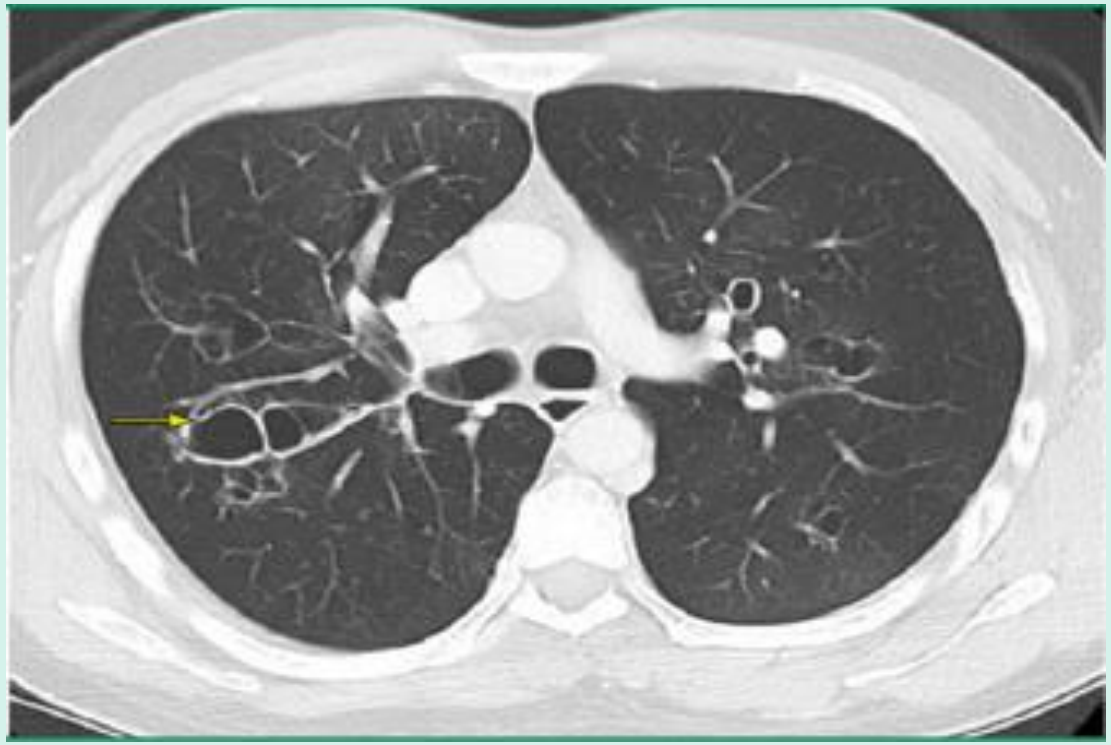
Causes of Airway Obstruction:

Congenital: Bronchomalacia + Tracheobronchomegaly

Acquired: Foreign body aspiration (with chronic cough) + Benign tumor + Hilar adenopathy (causes enlargement of Lymph nodes and affects adjacent airways, e.g., TB and Sarcoidosis) + Chronic bronchitis + Polychondritis (inflammation of cartilage every where in the body including the lungs) + Mucous impaction (ABPA)

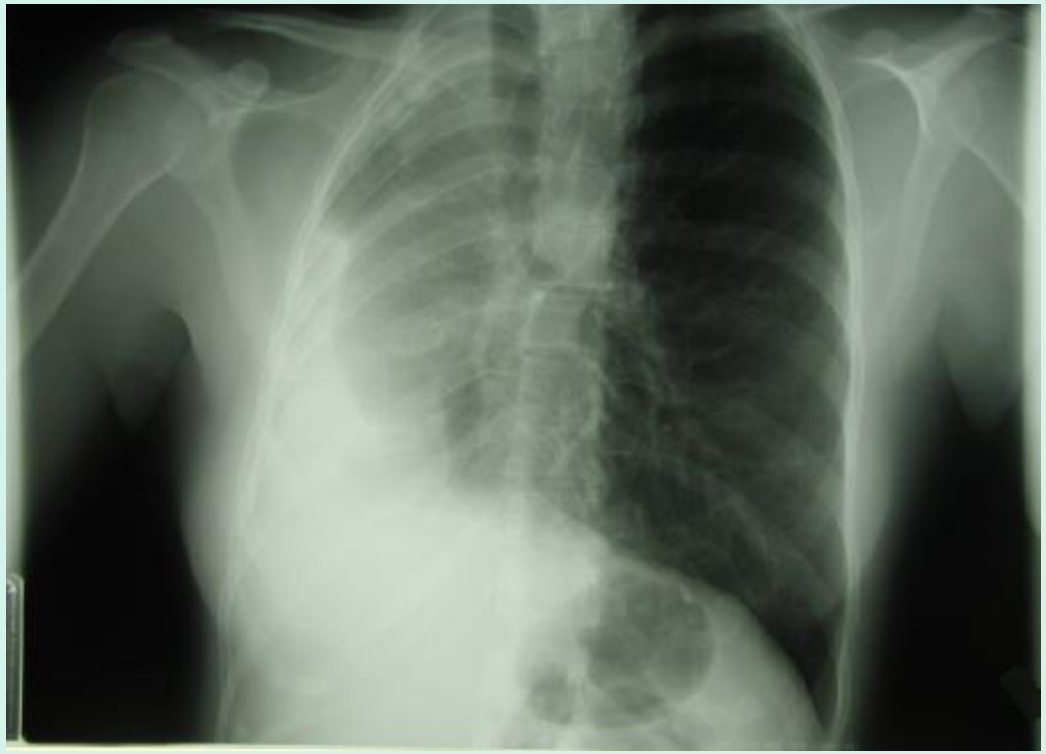
Allergic Bronchopulmonary Aspergillosis (ABPA): hypersensitivity response to the fungus Aspergillus that affects the bronchi and parenchyma of the lung. Diagnosed by very high serum IgE, presence of Aspergillus-specific IgE, Centrally located bronchiectasis, Finger in glove sign, and Green sputum. Occurs in patients with history of asthma. Suspected in people working in Farms, Gardens, or with Black mold on the walls.

WILLIAMS-CAMPBELL SYNDROME



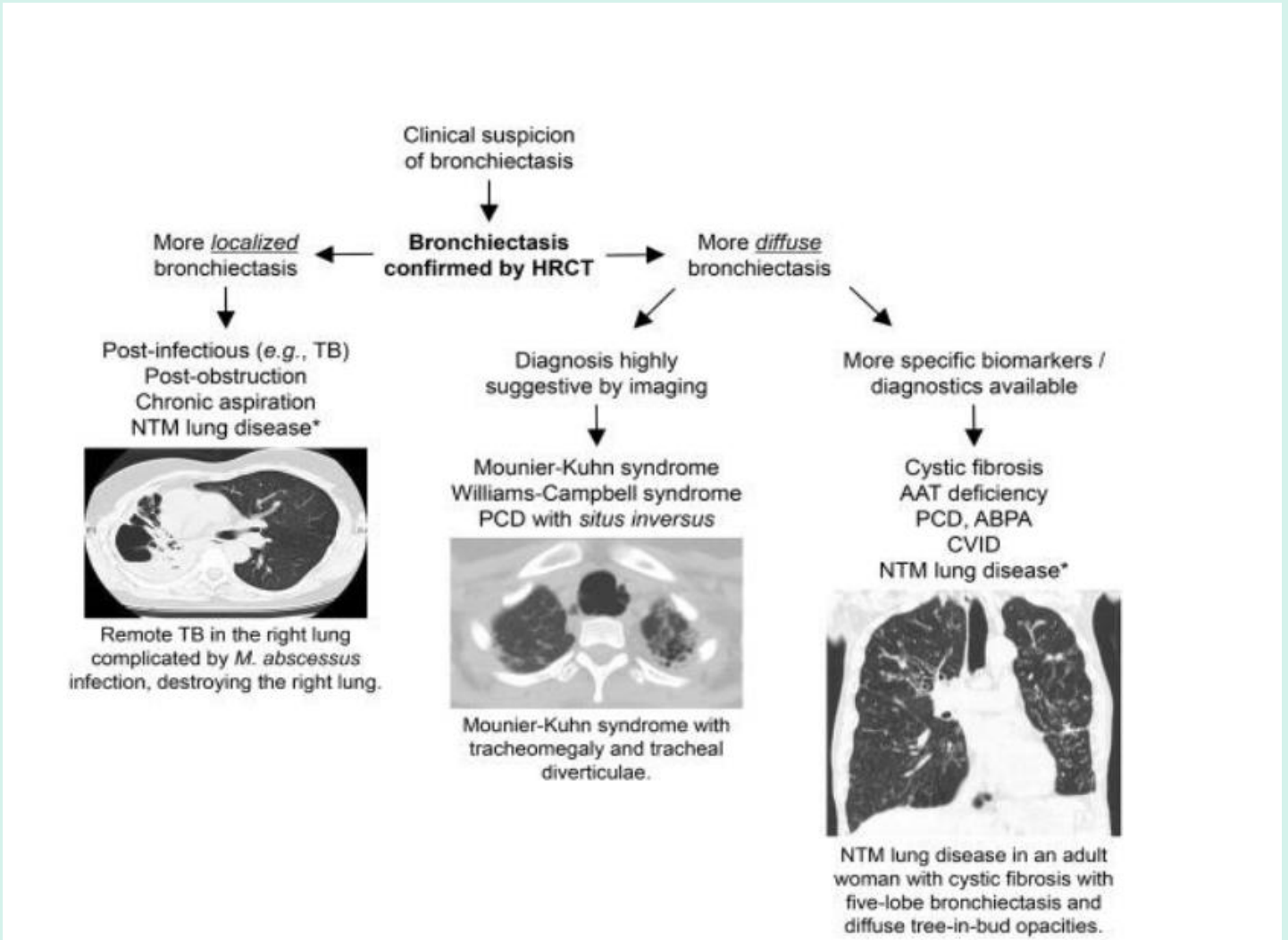
- Chest radiograph of bronchiectasis in Williams-Campbell syndrome.
- Williams-Campbell syndrome: Generalized tracheobronchomegaly due to deficiency of cartilage.
- Bronchomalacia: Abnormal dilation and weakness of bronchi

KARTAGENER'S SYNDROME



- This image shows Situs inversus, found in Kartagener's.
- Kartagener's syndrome is a rare, Autosomal recessive ciliary disorder comprising the triad of Situs inversus, Chronic Sinusitis, and Bronchiectasis. The basic problem lies in the defective cilia, leading to recurrent chest infections, ear / nose / throat symptoms and infertility.
- Kartagener's syndrome is one of the causes of impaired drainage leading to Bronchiectasis.
- Other causes of impaired drainage: Cystic Fibrosis (Autosomal recessive, mostly diagnosed in childhood, Upper lobes are more affected, Diagnosis: Sweat chloride test should be positive twice + mutation analysis) + Young's syndrome (a rare condition that is a combination of syndromes such as bronchiectasis, rhinosinusitis, and reduced male fertility) + Primary ciliary dyskinesia

DIAGNOSIS OF BRONCHIECTASIS



- The gold standard for the diagnosis is High resolution CT; it gives detailed lung parenchyma facilitating classification.
- Nontuberculous mycobacterial lung disease is a serious infection caused by bacteria that are common in the environment and can cause lung damage.

LAB TESTS

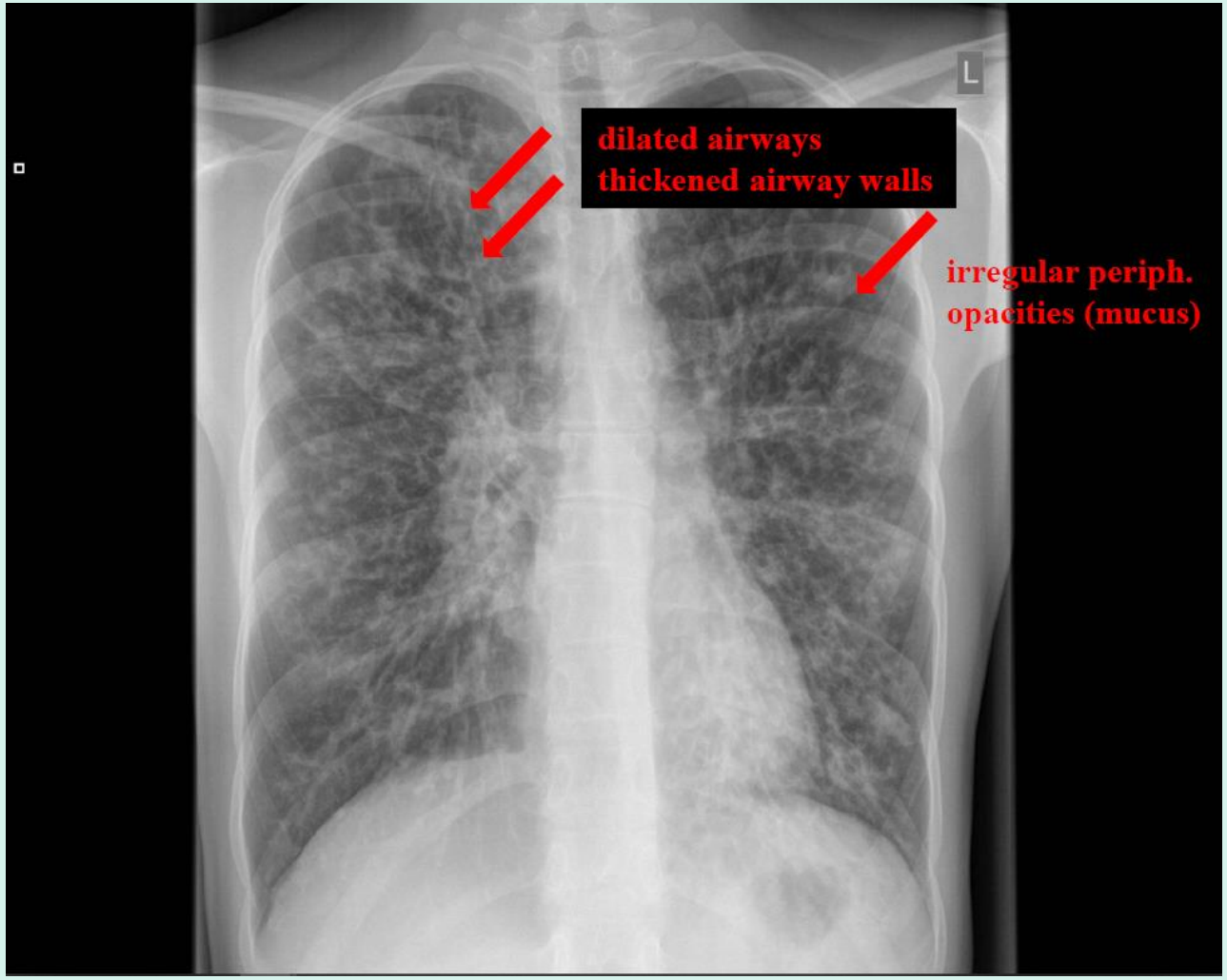
1. CBC, differential BC
2. immunoglobulin quantitation (levels of IgG, IgM, IgA)
3. Testing for cystic fibrosis:

   Sweat chloride

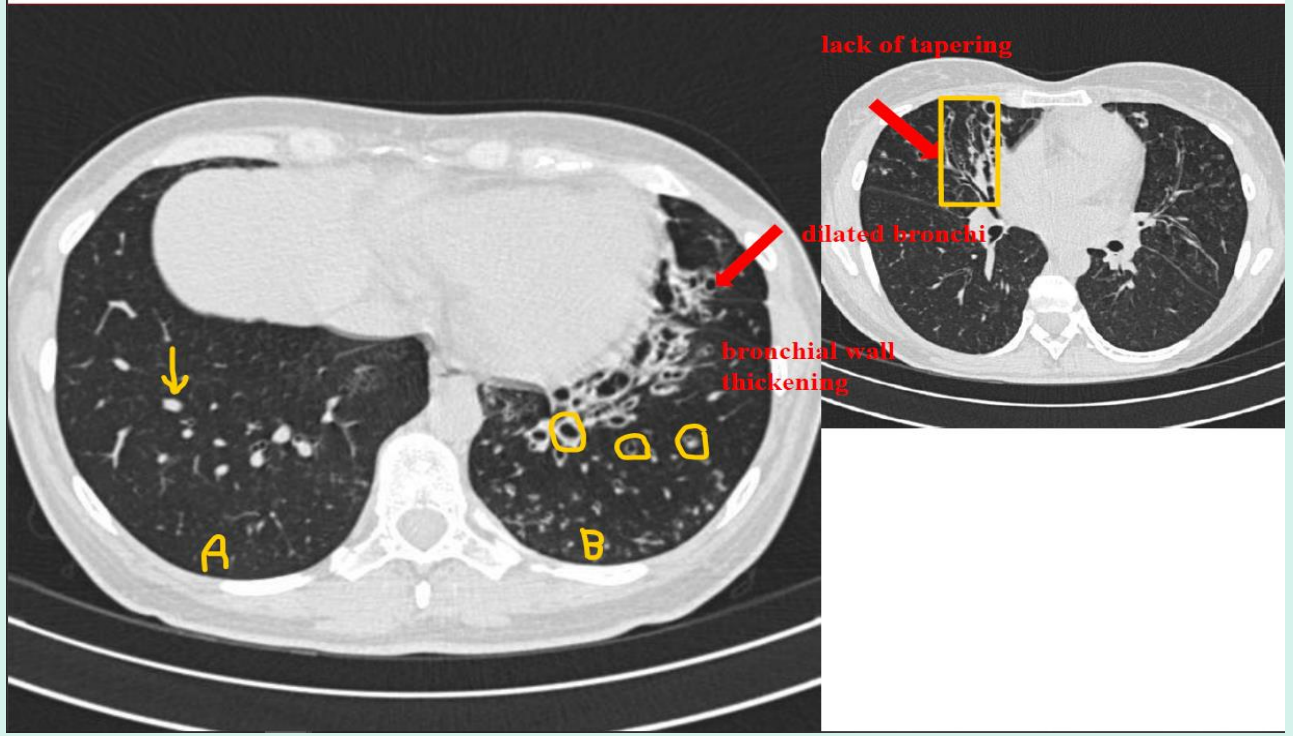
   mutation analysis of the cystic fibrosis transmembrane conductance regulator (CFTR) gene
4. sputum culture (bact. / TBC / fungi)

- Specific aspergillus IgE and IgG antibodies, total serum IgE level(Allergic bronchopulmonary asperjillosis) .
- IgG subclass levels .
- Alpha-1 antitrypsin level and/or genotype .
- Rheumatoid factor.

BRONCHIECTASIS CXR



BRONCHIECTASIS CHEST CT



- Bronchial Tapering: normal airways diminish in caliber as they extend towards the lung periphery.
- A → Normal side B → Abnormal side
- Yellow arrow → Vein, usually found alone
- A group of white filled circles → Arteries, found near airways (black circles).
- Features of bronchiectasis:

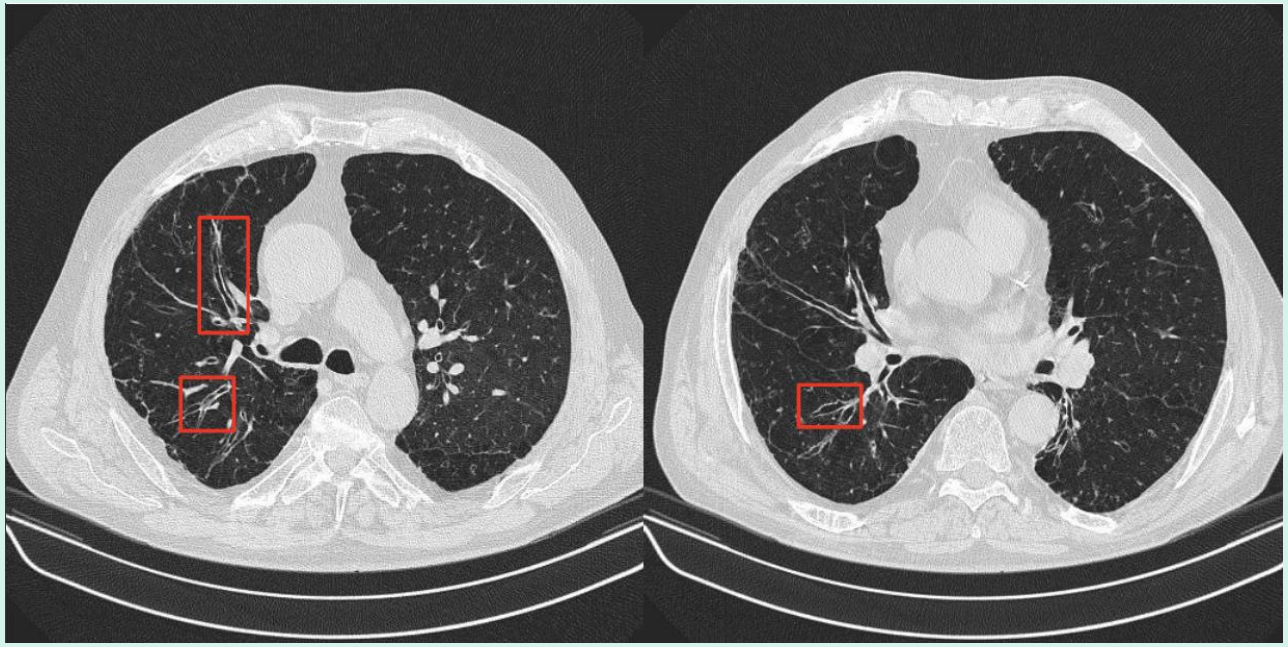
1- Lack of tapering

2- Signet Ring appearance (Yellow circles), normally artery to airway size 1:1, in bronchiectasis the airways become larger forming a ring shape.

3- Tram Lines (Yellow rectangle)

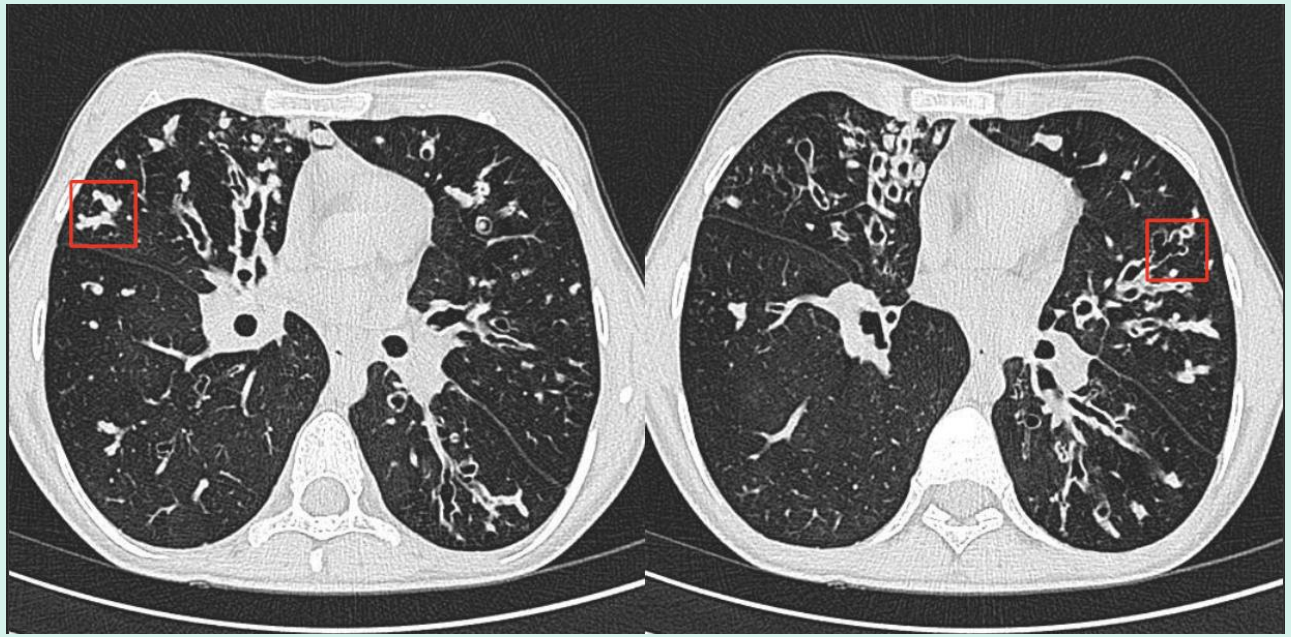
Advanced stages show Cystic changes.

BRONCHIECTASIS CHEST CT



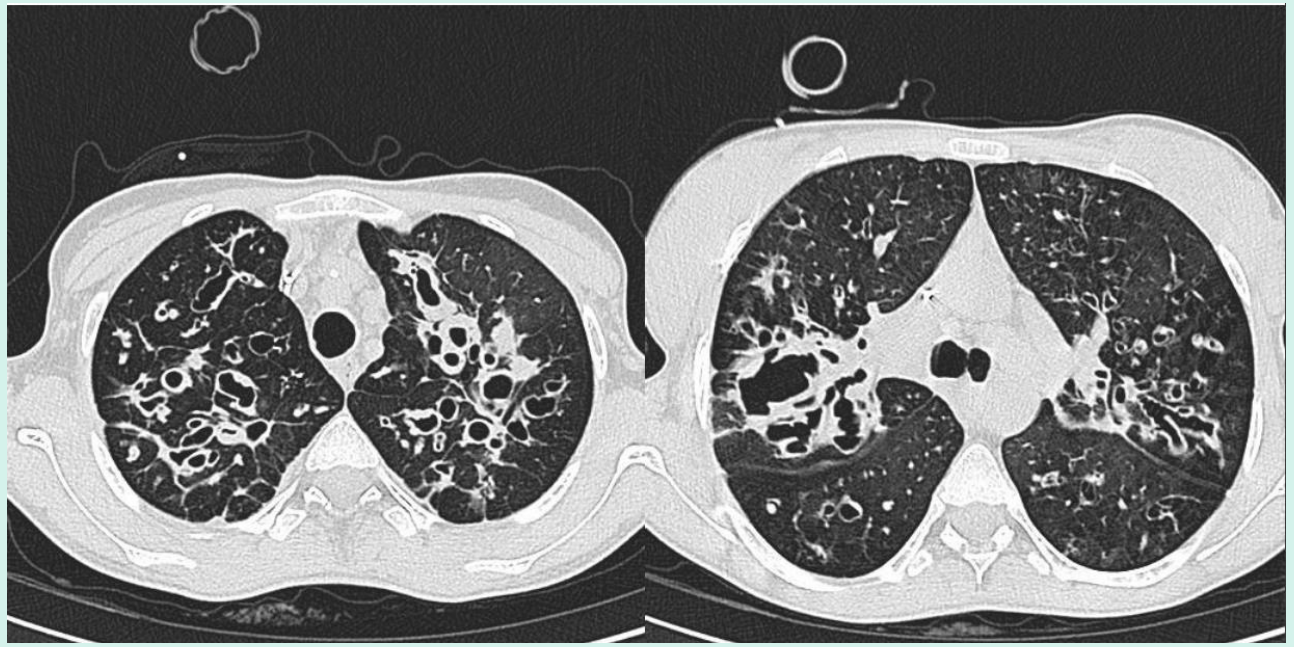
- Cylindrical Bronchiectasis (Bronchi are enlarged and cylindrical).
- Three basic morphologic types of bronchiectasis that are recognized on CT are Cylindrical, Varicose, and Cystic.

BRONCHIECTASIS CHEST CT



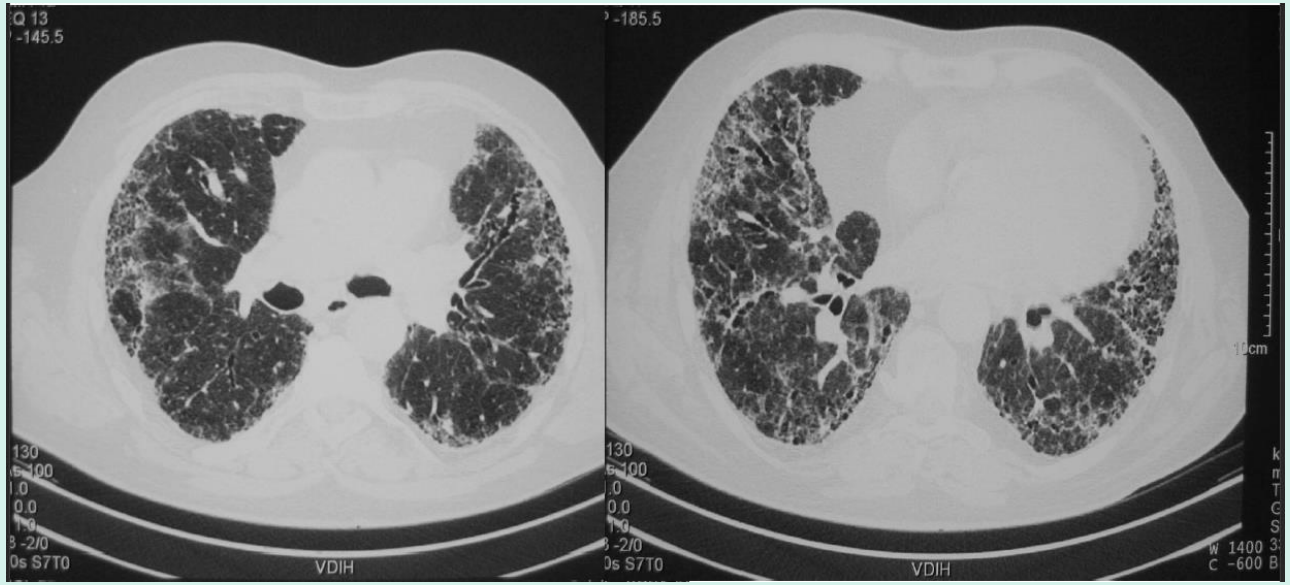
- Varicose Bronchiectasis; irregular or beaded bronchi, with alternating areas of dilation and constriction (narrow and wide)

BRONCHIECTASIS CHEST CT



- Cystic / Saccular Bronchiectasis (Sever); it has large cystic spaces and honeycomb appearance.

BRONCHIECTASIS CHEST CT



- Traction Bronchiectasis; it's the distortion of the airways secondary to mechanical traction on the bronchi due to fibrosis of the surrounding lung parenchyma.
- Although the airways may become dilated in this situation, the other manifestations of bronchiectasis are lacking.

PFT IN BRONCHIECTASIS

Obstructive spirometry

Low FVC in advanced disease; due to damaged lung parenchyma.

PLEURAL EFFUSION

PLEURAL EFFUSION

It is the abnormal excess fluid (Blood - Hemothorax -, Lymphatic fluid, Any Fluid) that accumulates in the pleural space, between the parietal and visceral pleura.

Normally, the pleural space contains a small amount of fluid (about 15 ml).

The pleural fluid's main function is Lubrication.

Human body makes 0.01 ml/kg/h, most reabsorbed into bronchial veins and lung lymphatics.

Normal Pleural fluid volume → 0.1 - 0.2 ml/kg (7 - 14 ml for a 70 kg person).

Normal Cell count → <1000 nucleated cells/μL, Neutrophils - 2%, Eosinophils - 0%, Lymphocytes - 7-11%, Monocytes - 61-77%, Mesothelial cells - 9-30%.

Normal Protein amount → 1.0 - 1.5 g/dl

Normal pH → 7.60 - 7.64

TRANSUDATE VS EXUDATE

Exudative Pleural effusions: Caused by inflammation or injury to the lung / pleura, e.g. Infections (in complicated infections, patient needs 6 weeks of antibiotics), Neoplasm (involvement of pleural space indicates stage 4 cancer), Pulmonary Embolism, Collagen vascular disease, Post-surgical, Uremia and Asbestos exposure.

If the fluid meets one of Light's criteria, then it's classified as exudate.

Usually, Exudate effusions causes are very serious, thus it's important to determine the type of effusion.

If you miss treatment of Exudative effusions for 1-2 days, patient may progress and become septic and die.

Light's criteria:

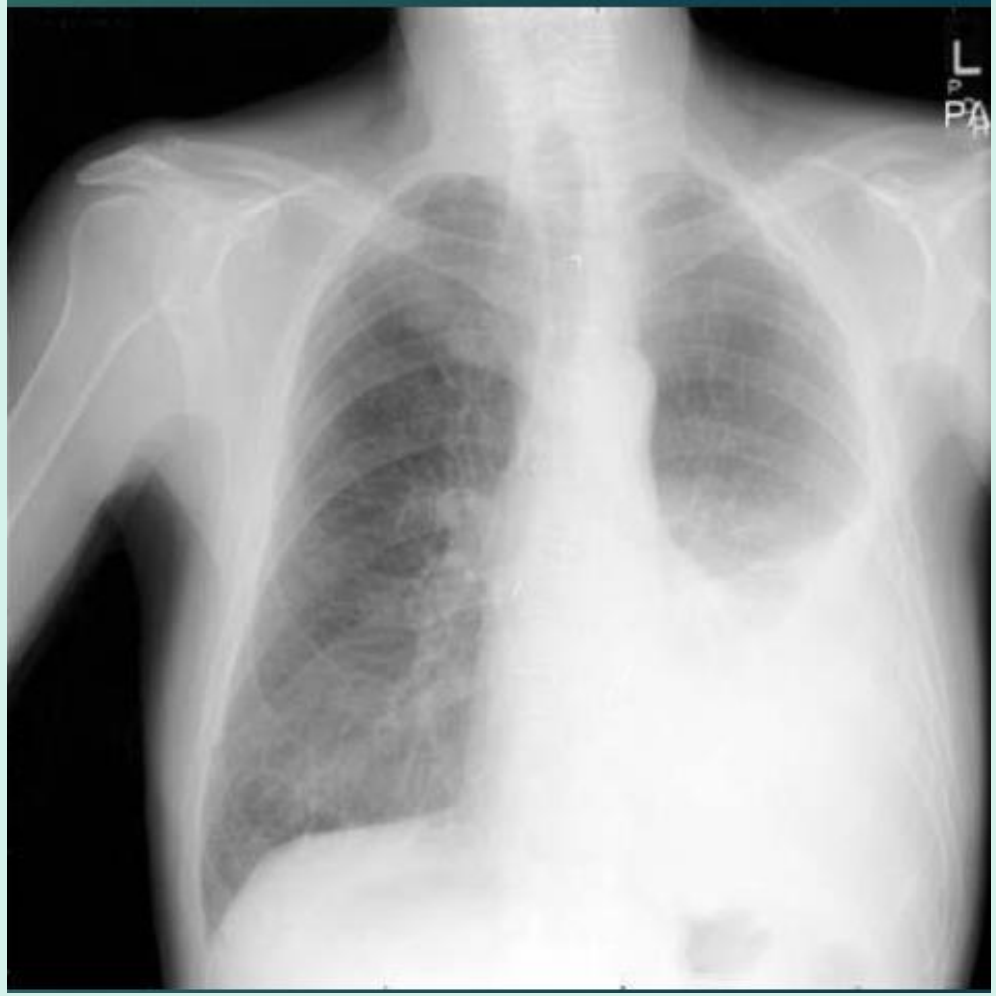
A- Pleural Fluid total protein / Serum total protein > 0.5

B- Pleural Fluid LDH / Serum LDH > 0.6

C- Pleural Fluid LDH value > 2/3 upper limit of normal for serum normal values.

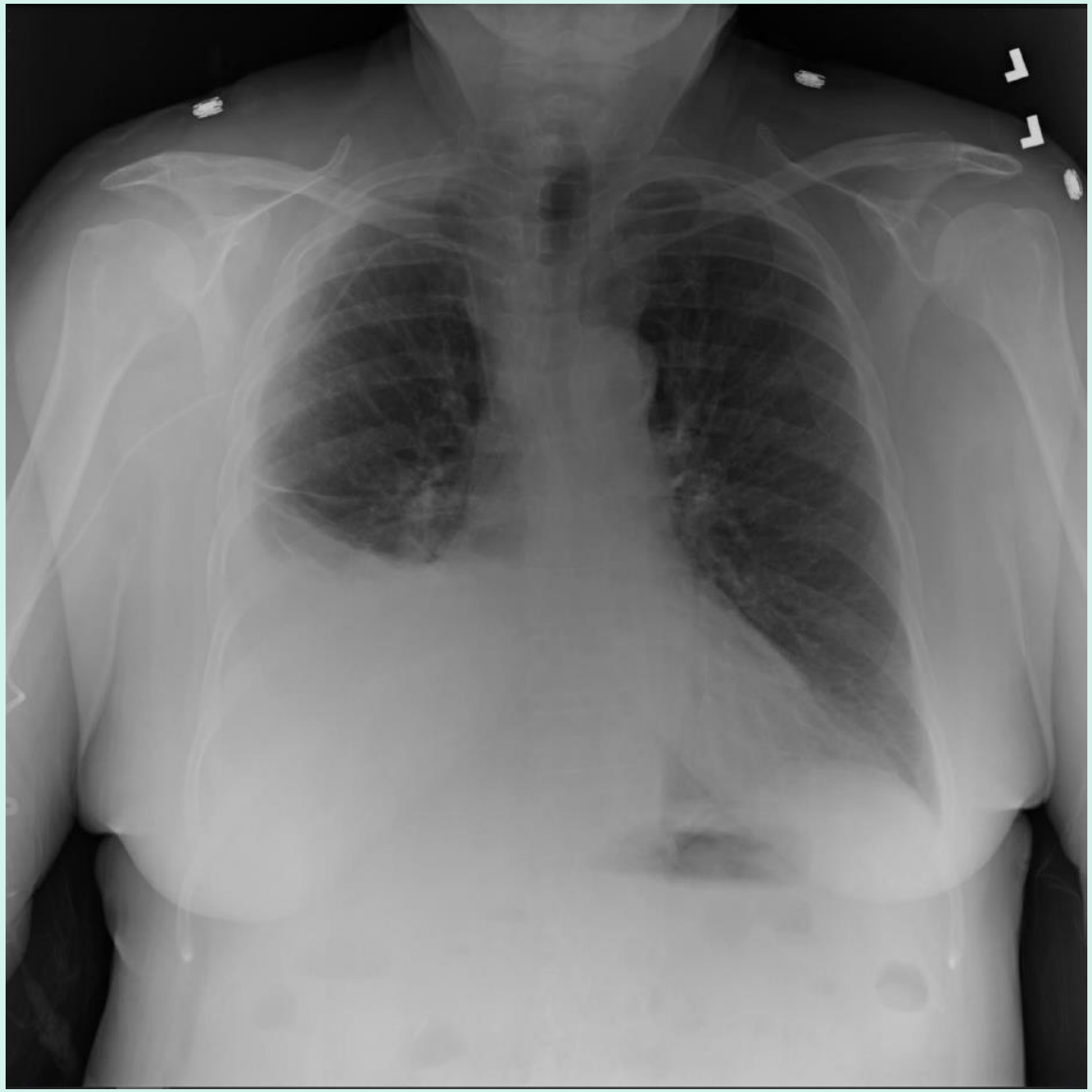
- Transudative Pleural effusions: Basically, caused by over production of water (patients usually respond to diuretics like Lasix and Spironolactone), e.g. CHF, Liver disease, Nephrotic Syndrome, Urinothorax, Peritoneal dialysis, and Myxedema.

PLEURAL EFFUSION - RADIOLOGICAL EVALUATION

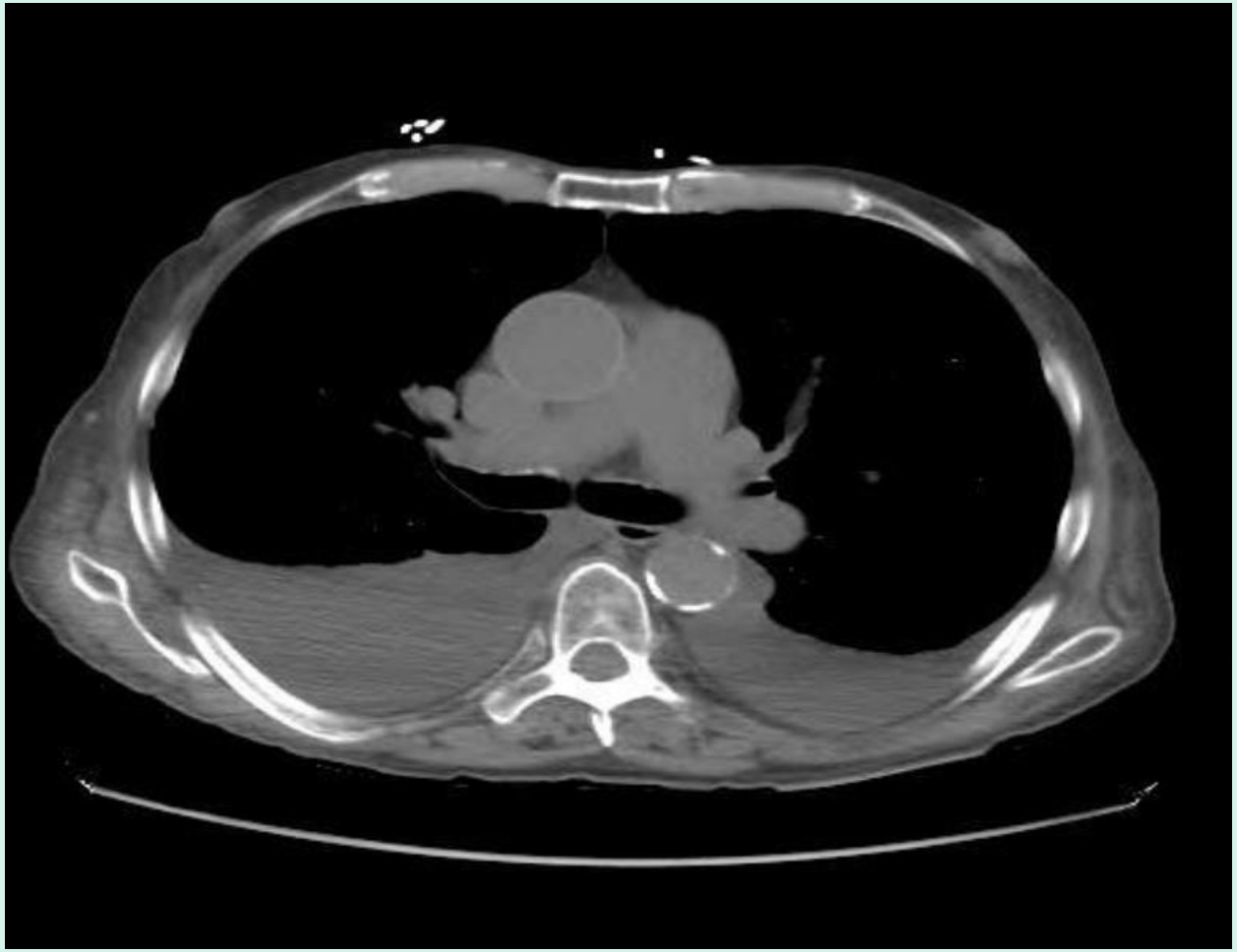


- CXR → AP and Lateral
- Ultrasound → To differentiate small effusions VS pleural thickening
- CT → Not routinely indicated but can evaluate Lung parenchyma / mass obscured by effusion.
- Lateral Decubitus → free flowing / Loculated.

PLEURAL EFFUSION - CXR

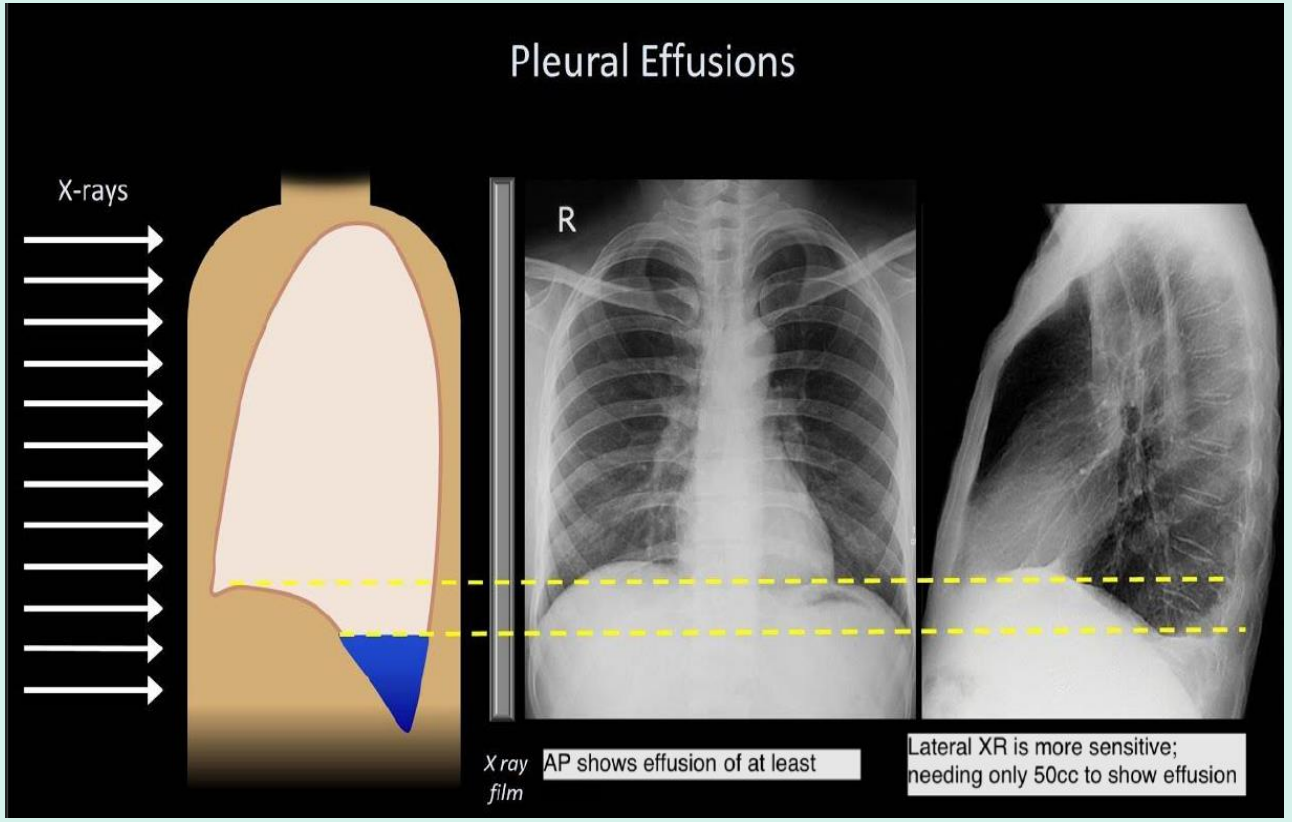


PLEURAL EFFUSION - CT



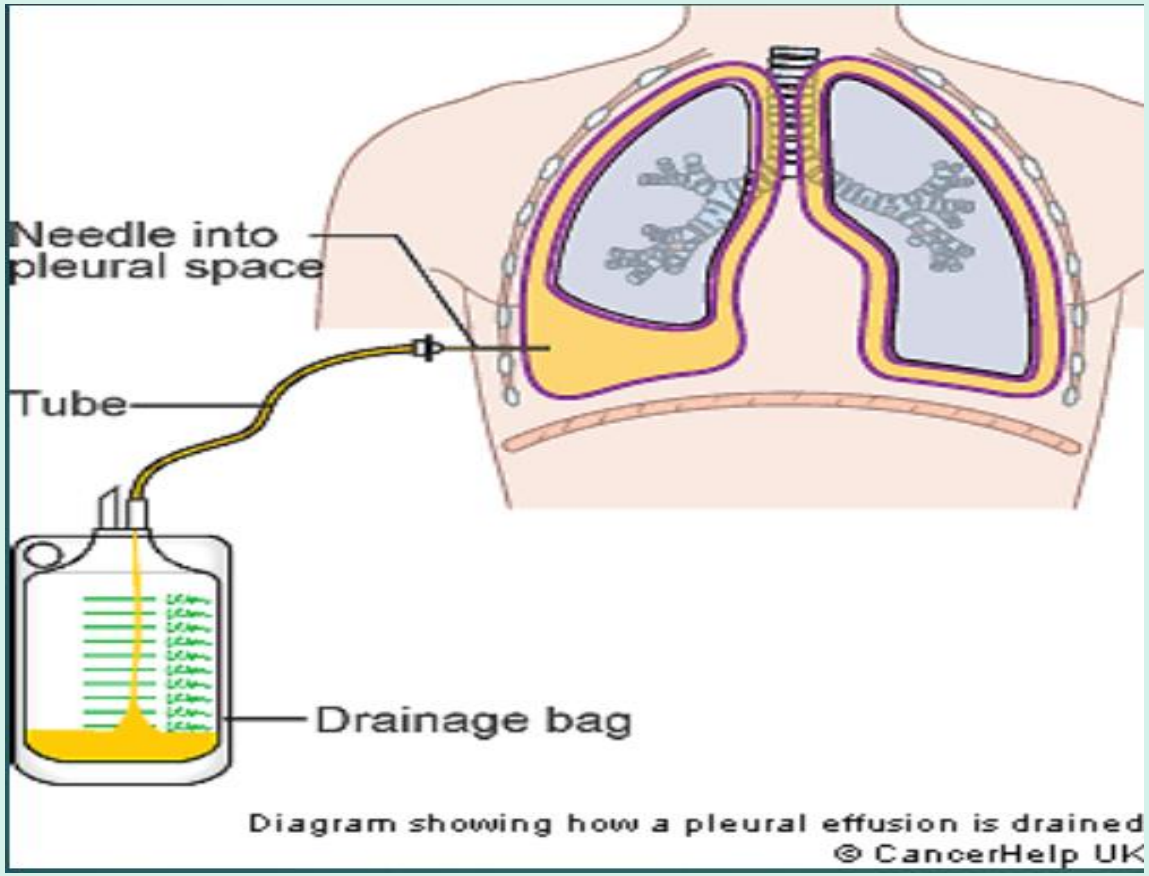
- CT is the gold standard and is very sensitive, but there is more radiation exposure and is more expensive, thus it's easier to start with CXR

PLEURAL EFFUSION - CXR



- CXR can also aid in determining the cause of the effusion (e.g. due to HF, Parapneumonic, Metastasis).

THORACOCENTESIS



- Drainage of the effusion, it can be both diagnostic and therapeutic.
- Any unexplained pleural effusion must be drained.
- Indicated in any pleural effusion of adequate size where there is no obvious cause.

PLEURAL FLUID APPEARANCE

Color	Suggested Diagnosis
Pale Yellow	Transudate
Red/Bloody	Malignancy, BAPE, PCIS, Pulmonary infarction, Trauma
White/Milky	Chylothorax/Cholesterol effusion
Brown	Chronic bloody effusion, rupture of amebic liver abscess
Black	Aspergillus
Yellow-green	Rheumatoid pleurisy
Color of tube feeds/IVF	Feeding tube/Catheter has reached pleural space

Characteristic	Suggested Diagnosis
Water Like	Duro-pleural fistula
Urine Like	Urinothorax
Pus	Empyema
Viscous	Mesothelioma, Empyema
Debris	Rheumatoid pleurisy
Turbid	Inflammatory exudate or lipids
Anchovy paste	Amebic liver abscess rupture
Satin-like sheen	Cholesterol effusion

- Please note that color alone cannot help in differentiating Exudate from Transudate.
- White / Milky Pleural fluid → indicates the presence of Lipids inside the fluid.

OTHER DIAGNOSTIC TESTING FOR PLEURAL EFFUSIONS

• Glucose	• May be reduced in **infections or RA**
• pH	• May be reduced in infections, malignancy, RA, esophageal rupture
• Cytology	• Malignancy
• Cultures	• Routine, AFB and fungal studies
• Amylase	• Elevated in **esophageal rupture, pancreatitis, and malignancy**
• Triglyceride	• Elevated in chylothorax (chylomicrons)
• Adenosine deaminase	• Elevated in **tuberculous pleuritis**, ADA < 40 U/L excludes tuberculosis

- Glucose levels in Pleural effusions < 60 mg/dl
- Cultures are done to rule out parapneumonic effusions / empyema .
- Amylase levels in Pleural effusions → > 200 mg/dl
- Triglyceride levels in pleural effusions → > 110 mg/dl
- Adenosine Deaminase levels in Tuberculous Pleuritis → > 50 mg/dl.

CELL COUNT IN PLEURAL EFFUSION

Cell Type	Value	Differential Dx
RBC	>100,000 per mm3	Malignancy, trauma, PE, parapneumonic effusion
Lymphocytes	>50%	Malignancy, PE, TB, Post CABG
	>80%	Post CABG, TB, Chylothorax, Yellow nail, Sarcoid, Lymphoma, Acute Lung Rejection, Chronic Rheumatoid Effusion
	>90%	TB, Lymphoma
Neutrophils	>50%	Parapneumonic effusion, PE, Abdominal disease, Acute tuberculous pleurisy (7%), Malignancy (20%), Viral infection
Eosinophils	>10%	PTX, HTX, BAPE, Parasitic, Drug induced, Fungal Disease, Churg-Strauss

- Normally, macrophages predominate, and lymphocytes are < 10%
- Increased Lymphocytes usually indicates malignancy or chronic infections.
- Increased Neutrophils indicates an acute inflammation (Infection, Lymphoma, Parapneumonic effusions).
- Any blood or air in the pleural space increases the eosinophils.

CASE #1

A 58-year old man is evaluated for dyspnea and is found to have a moderate sized right-sided pleural effusion. He undergoes a thoracentesis with the following findings.

Appearance	Serosanguinous
pH	7.48
Protein	**5.8g**/dL(serum protein 7.2g/dL)
LDH	**285** IU/L(serum LDH 320 IU/L)
Glucose	**66** mg/dL
WBC	3800/mm^3
RBC	24,000/mm^3
PMNs	10%
Lymphocytes	80%
Mesothelial cells	10%
Cytology	Lymphocytosis with chronic inflammation and no malignant cells or organisms identified

Which of the following is an unlikely cause of the pleural effusion in this patient?

A. Cirrhosis
B. Lung cancer
C. Mesothelioma
D. Pulmonary embolism
E. Tuberculosis

- The answer is A- Cirrhosis; because according to the labs, the effusion is exudative, and in the case of cirrhosis the effusion is Transudate.

CASE #2

A 62 year-old woman is admitted to the hospital with a community acquired pneumonia with a 4-day history of fever, cough, and right-sided pleuritic chest pain. The admission chest x-ray identifies a right lower and middle lobe infiltrate with an associated effusion.

All of the following indicate a complicated effusion that may require tube thoracostomy EXCEPT:

A. Loculated fluid
B. Pleural fluid pH less than 7.2
C. Pleural fluid glucose greater than 60 mg/dL
D. Positive Gram stain or culture of the pleural fluid
E. Frank pus on thoracentesis

- The Answer is C- Pleural Fluid Glucose > 60 mg/dl.
- There are three types of Parapneumonic effusions: Uncomplicated / Simple effusions + Complicated effusions + Empyema.

INDICATORS OF COMPLICATED PARAPNEUMONIC EFFUSION

Empyema-positive Gram stain of fluid or gross pus noted on thoracentesis

Multiple septations or loculations noted on ultrasound or CT chest

pH < 7.2 indicates complicated infection, < 7.0 will likely need drainage/decortication

Pleural fluid glucose < 60 mg/dL

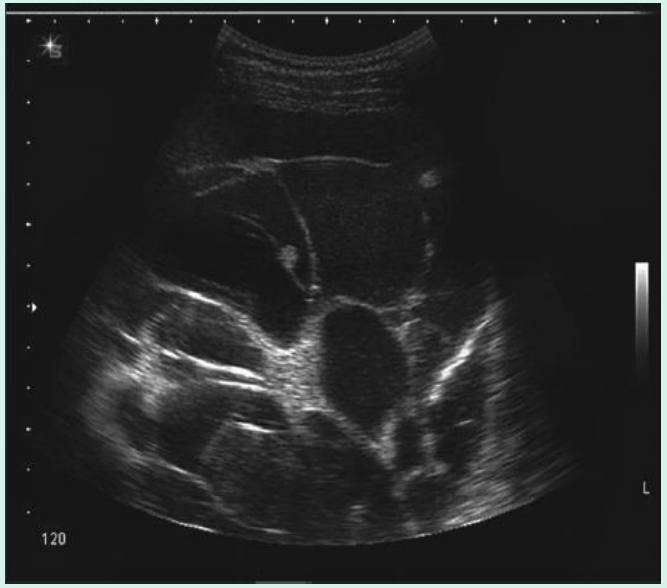
Treatment is **drainage** and antibiotic therapy.

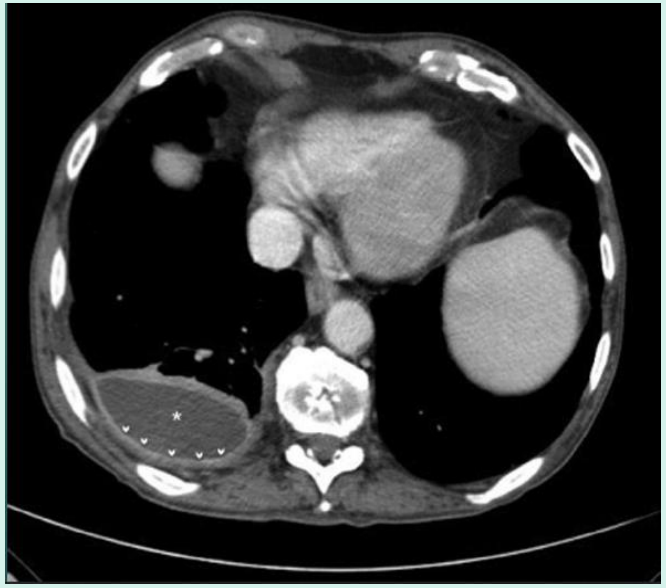
Includes either repeat thoracentesis, percutaneous catheter drainage, chest tube thoracostomy or possible surgical decortication. Can use **intrapleural thrombolytics** to help facilitate drainage.

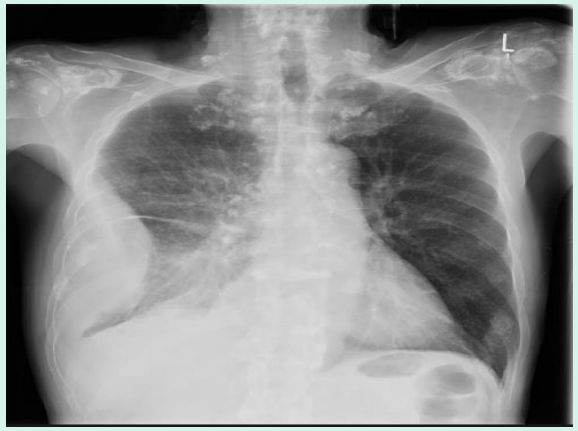
Early intervention improves outcomes.

- Complicated Parapneumonic effusions always require drainage.
- ** A common exam question: What are the indications to drain abnormal fluid in the setting of pneumonia??

RADIOLOGY OF COMPLICATED PLEURAL EFFUSION







- Shows Pocket of fluid or mass.
- CT shows split pleura with some contrast enhancement of the parietal pleura.
- Ultrasound images can show septations in the fluid

CASE #3

24 year old woman presents with several weeks of increasing cough and dyspnea. She denies chest pain, fever or chills. She has noted some weight loss but is unable to quantify it well. Physical exam reveals decreased breath sounds over the right chest with dullness to percussion and decreased tactile fremitus. A CXR reveals large mediastinal mass and right sided pleural effusion. She has no previous history of any trauma or recent surgery to the chest. She takes NSAIDS for dysmenorrhea. You perform a thoracentesis revealing turbid fluid. Pleural fluid analysis shows triglycerides of 600

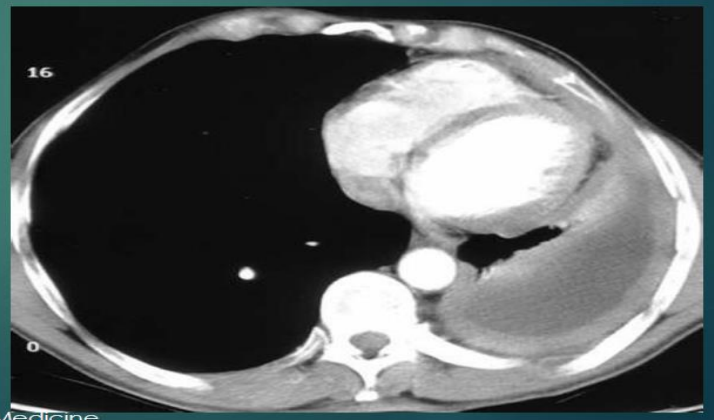
The most likely diagnosis is

A. Drug induced pleural effusion
B. Bronchogenic carcinoma
C. Lymphoma
D. Empyema
E. Rheumatoid arthritis

- The Answer is C

CASE #4

- 68 year old man, previously worked at a paper mill for 20 years where he applied insulation to large industrial boilers, however, he stopped when he was 50. presents with complaint of increased cough and dyspnea over the past three months. He has also had some dull continuous chest discomfort over the right side of his chest
- Physical exam reveals decreased breath sounds and dullness to percussion over the right chest
  CT chest reveals-



Murray and Nadel's Textbook of Respiratory Medicine

Most likely diagnosis:

- A. Yellow Nail syndrome
- B. Loculated empyema
- C. Tuberculous pleuritis
- D. Chylothorax
- E. Mesothelioma

- The answer is E

PULMONARY FUNCTION TEST (PFT)

PFT

2 types of PFTs:

1- Mechanical object: Volume, Flows, Bronchodilator response, airway hyperreactivity, Compliance, Resistance, Maximum Respiratory pressure, Work of breathing.

2- Gas Exchange object: PaO2, PaCO2, P(A-a)O2, Physiologic dead space ventilation, Diffusion capacity.

Indications: Evaluation of Pulmonary complaint or sign (diagnostic) + Quantification of impairment severity and need to initiate treatment, evaluate response to treatment + Preoperative assessment, for post op risk of complications, or tolerance of lung resection + Disability evaluation.

Contraindications: Chest or Abdominal pain of any cause + Oral or facial pain exacerbated by mouthpiece + Stress incontinence + Dementia or confusion state + Within 1 month of MI , or during unstable angina, asthma attack or chest infection.

SPIROMETRY

- Readily available, inexpensive, and highly reproducible.
- Used to assess:

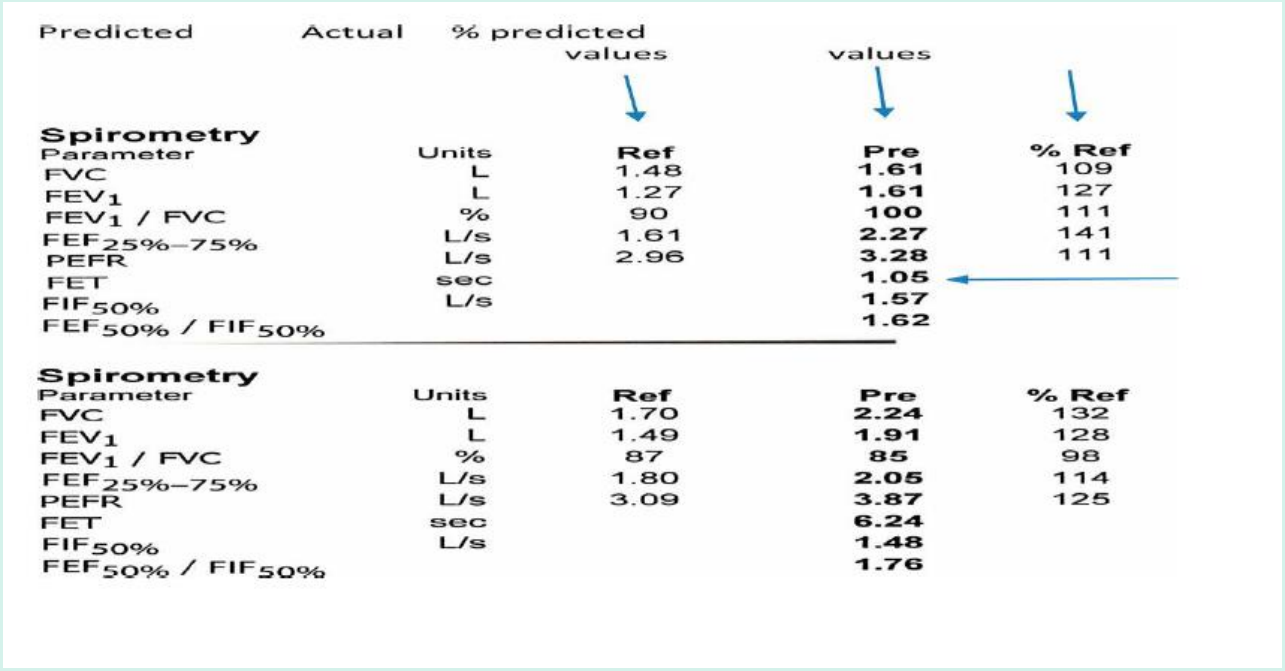
1- Forced Vital Capacity (FVC)

2- FEV1 (volume exhaled in the first second of FVC)

3- FEV1 / FVC ratio

- Predicted normal values depend on: Age, Hight and weight, Sex, Ethnic origin.
- Acceptability and reproducibility.
- Acceptability criteria: Smooth continuous curve, Good start of test, Good end of test.

SPIROMETRY



Predicted Actual % predicted values values

Spirometry Parameter	Units	Ref	Pre	% Ref
FVC	L	1.48	**1.61**	109
FEV_1	L	1.27	**1.61**	127
FEV_1 / FVC	%	90	**100**	111
$FEF_{25\%-75\%}$	L/s	1.61	**2.27**	141
PEFR	L/s	2.96	**3.28**	111
FET	sec		**1.05**	
$FIF_{50\%}$	L/s		**1.57**	
$FEF_{50\%}$ / $FIF_{50\%}$			**1.62**	

Spirometry Parameter	Units	Ref	Pre	% Ref
FVC	L	1.70	**2.24**	132
FEV_1	L	1.49	**1.91**	128
FEV_1 / FVC	%	87	**85**	98
$FEF_{25\%-75\%}$	L/s	1.80	**2.05**	114
PEFR	L/s	3.09	**3.87**	125
FET	sec		**6.24**	
$FIF_{50\%}$	L/s		**1.48**	
$FEF_{50\%}$ / $FIF_{50\%}$			**1.76**	

- Spirometry is a test used to assess how well the lungs are functioning by measuring how much air is inhaled, how much is exhaled and how quick the exhalation is after entering 4 factors, age, race, sex, and height; so that the references values get modeled based on them.
- It is used to diagnose asthma, chronic obstructive pulmonary disease (COPD), and other conditions that affect breathing

INTERPRETATION OF RESULTS

Inspect the flow volume loop for quality (good start, absence of artifact, >= 6 seconds effort, plateau).

Examine the FEV1 / FVC ratio. Diagnose Obstructive disease if the ratio is below lower limit of normal (LLN). Use <70% of LLN is unavailable.

Grade severity of obstruction:

1- FEV1 > 100% of predicted → Physiologic variant

2- 100% > FEV1 >= 70% of predicted → Mild obstruction

3- 70% > FEV1 >= 60% of predicted → Moderate obstruction

4- 60% > FEV1 >= 50% of predicted → Moderately severe obstruction

5- 50% > FEV1 >= 35% of predicted → Severe obstruction

6- FEV1 < 35% of predicted → Very severe obstruction.

- Be cautious in interpreting obstructive defect if Both FEV1 and/or FVC are above 100% even if the ratio is < LLN as this pattern can be seen in young healthy subjects and athletes (SVC > FVC)

PFT

- Forced Expiratory Time (FET): Checking the quality of the spirometry report is the first step before interpretation. It should have the following properties: a good start, absence of artifact, >6s expiratory time, and a plateau. Expiratory time is verified using the labeled graph where it shows Forced expiratory time (FET), which should be >6s.
- Spirometry Data: The most common parameters measured in spirometry are (what we will focus on):

a- Forced vital capacity (FVC): total volume of air expelled during forced expiration.

b- Forced expiratory volume (FEV1): forced expiratory volume in the first second.

c- FEV1/FVC: volume of air expelled in the first second with respect to the maximum air that can be expired.

Spirometry pre- and post-bronchodilator are performed to examine the reversibility, and effectiveness, of the bronchodilator (e.g. short-acting beta 2 agonists) on the breathing problem.

Significant bronchodilator reversibility is defined by a 12% and >200 mL increase in either FEV1 or FVC (or both), which is diagnostic of asthma.

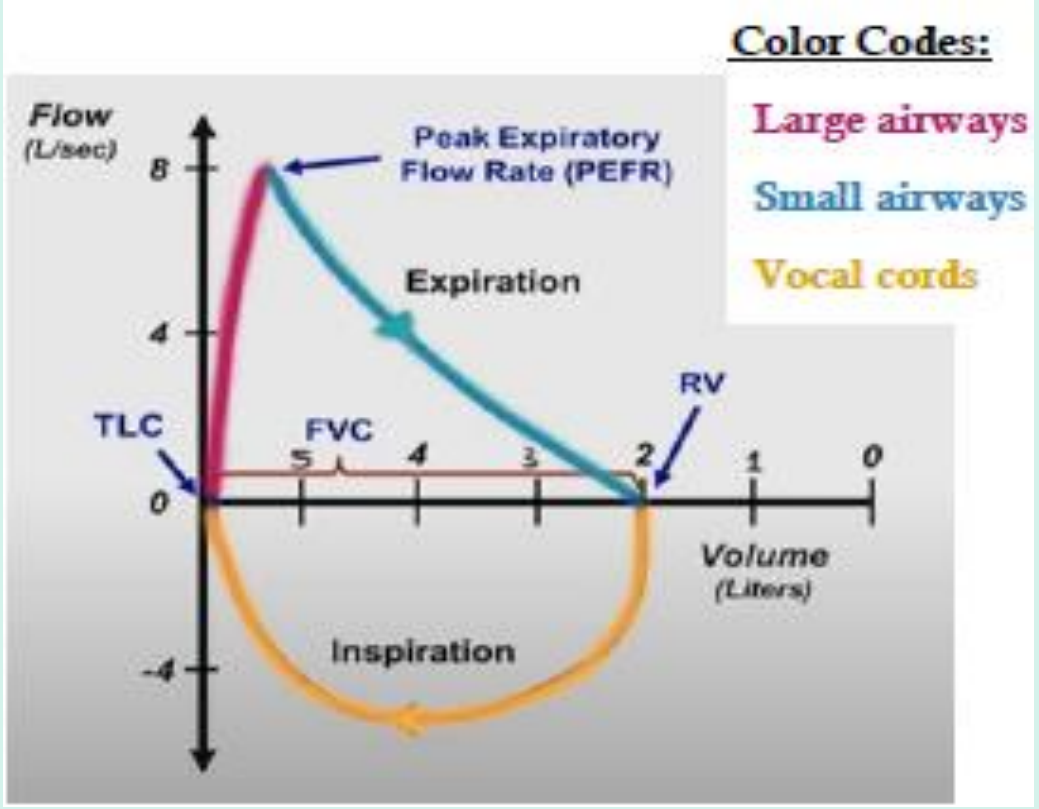
Criteria for normal spirometry (assessed by looking at the values in the red square):

a- FEV1: %predicted >80%

b- FVC: %predicted >80%

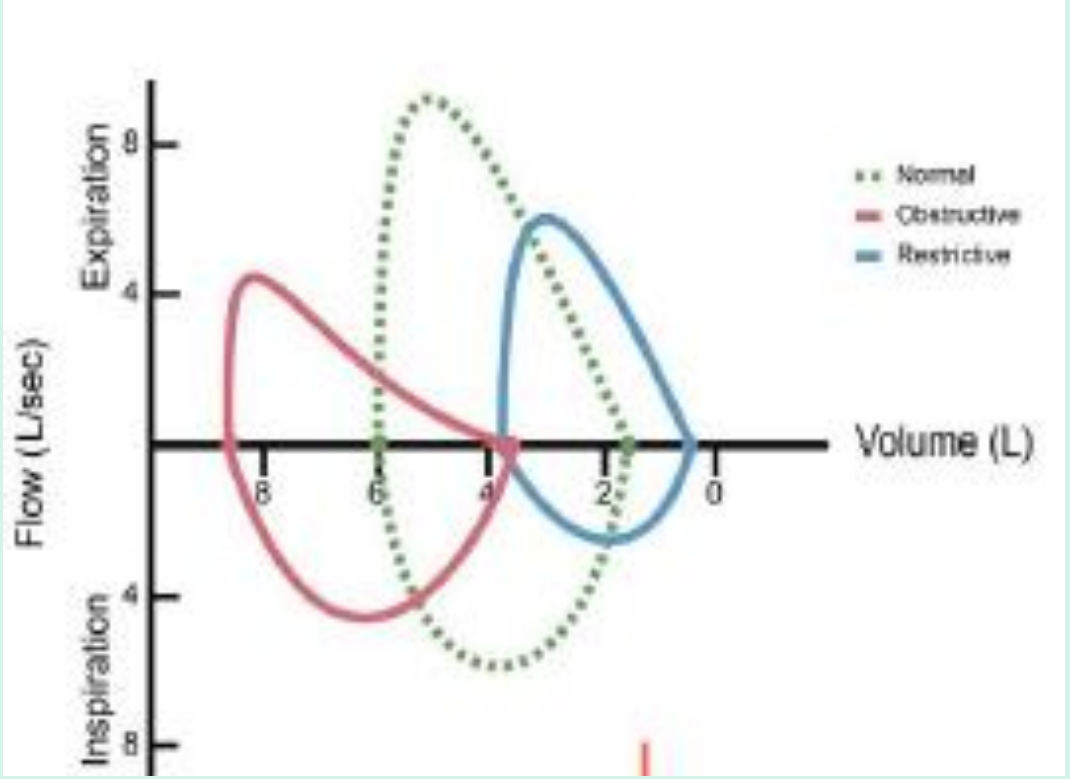
c- FEV1/FVC: >70% (0.7)

FLOW VOLUME LOOP



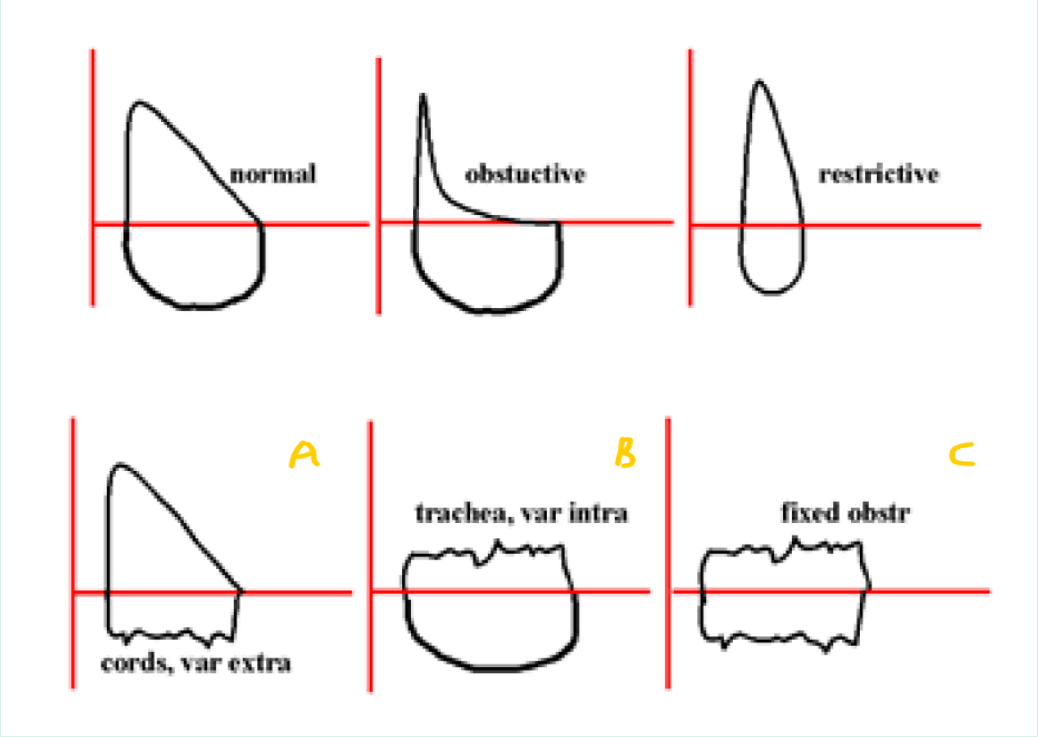
- Starting from the Residual Volume (RV), the patient takes a deep breath → reaching TLC → Forced Expiration starts expelling air from the large airways (e.g. trachea), followed by the small airways, until reaching RV again → the patient starts inspiration through taking deep breath again, which is affected by the functional vocal cords.

OBSTRUCTIVE VS RESTRICTIVE FLOW VOLUME LOOPS



- Obstructive Flow volume loop: Obstructive diseases are mostly due to small airways narrowing, which shows on the graph, where everything is normal except that the small airways limb requires more time to expel air (causing snooping or covering). The loop shifts to the left since volumes are > than normal.
- Restrictive Flow volume loop: In Restrictive Lung diseases, everything is normal except that the total Vital capacity is decreased. The loop shifts to the right since volumes are < than normal.

SPIROMETRY PATTERNS - FLOW VOLUME LOOPS



- A → Large and small airways are normal, vocal cords are abnormal (Wavy or Flat lines).

This is a case of Variable extra-thoracic obstruction, above the lung, e.g. Paradoxical vocal cord dysfunction, where they don't open directly upon inspiration.

- B → Large airways are abnormal.

This is a case of Intrathoracic obstruction, e.g. Tracheal polyp. The inspiratory limb isn't affected since the trachea normally dilates during inspiration.

- C → Both inspiration and expiration are affected.

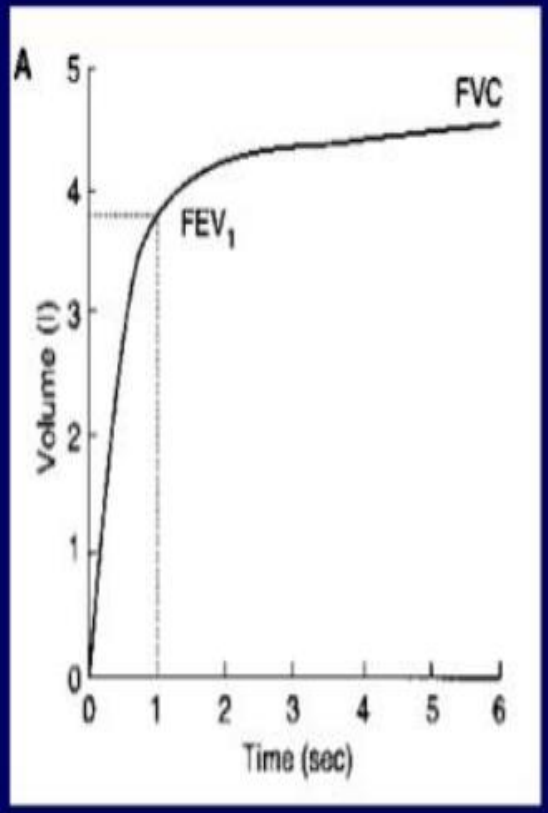
This is mostly due to the presence of fixed obstruction where the trachea cannot dilate even during inspiration, thus affecting both limbs, e.g. Wegener's granuloma, Tumor, Foreign objects.

FVC: TECHNIQUE

- The FVC begins with full lung inflation (TLC), followed by full exhalation with full effort (both maximum force and duration) to achieve the highest flow rates, down to RV , and the a rapid inhalation back up to TLC.

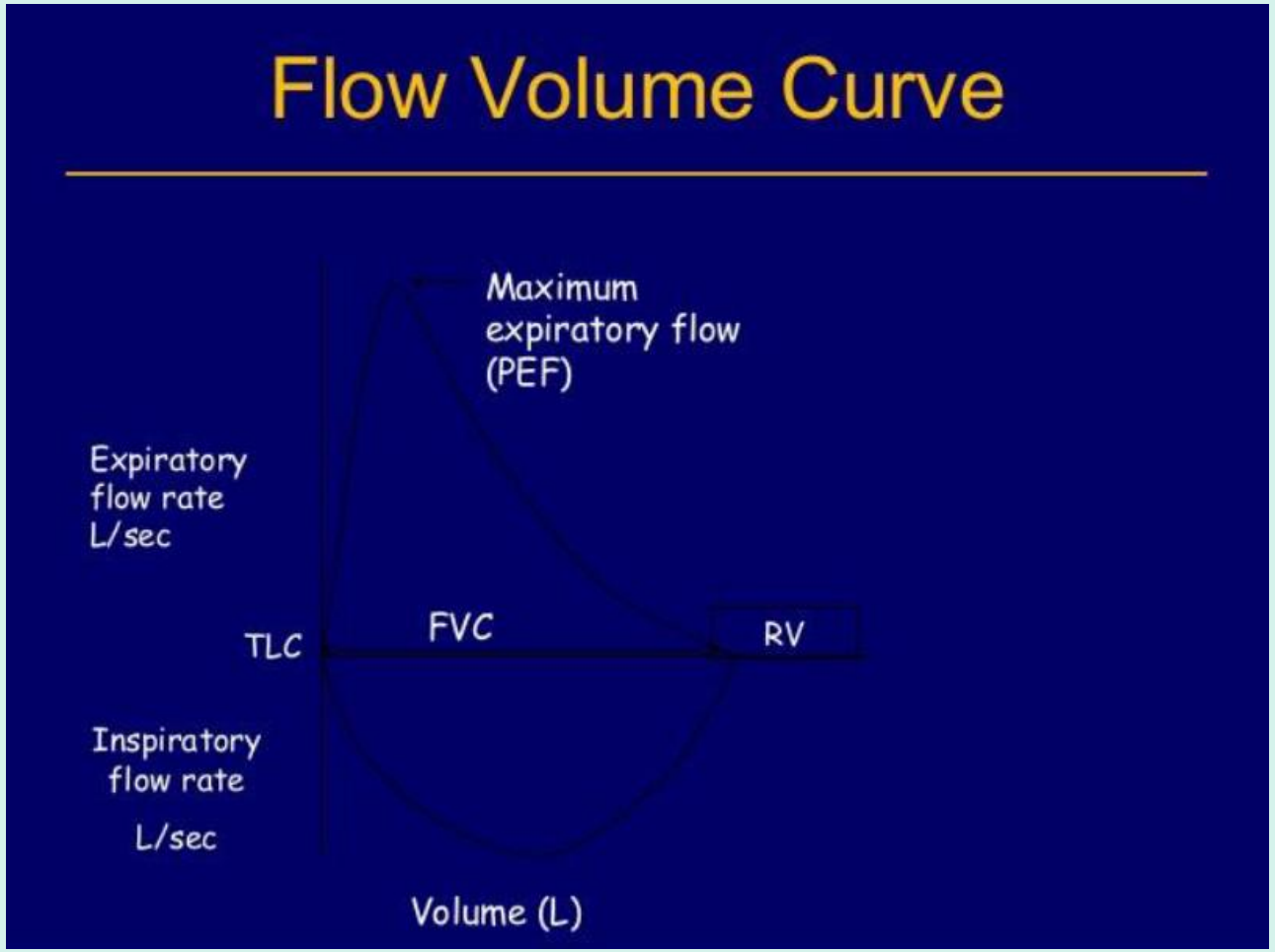
- This effort can be shown graphically as a flow-volume loop(FVL) , or volume –time curve (V-t curve) both represent the same FVC maneuver .

FEV1

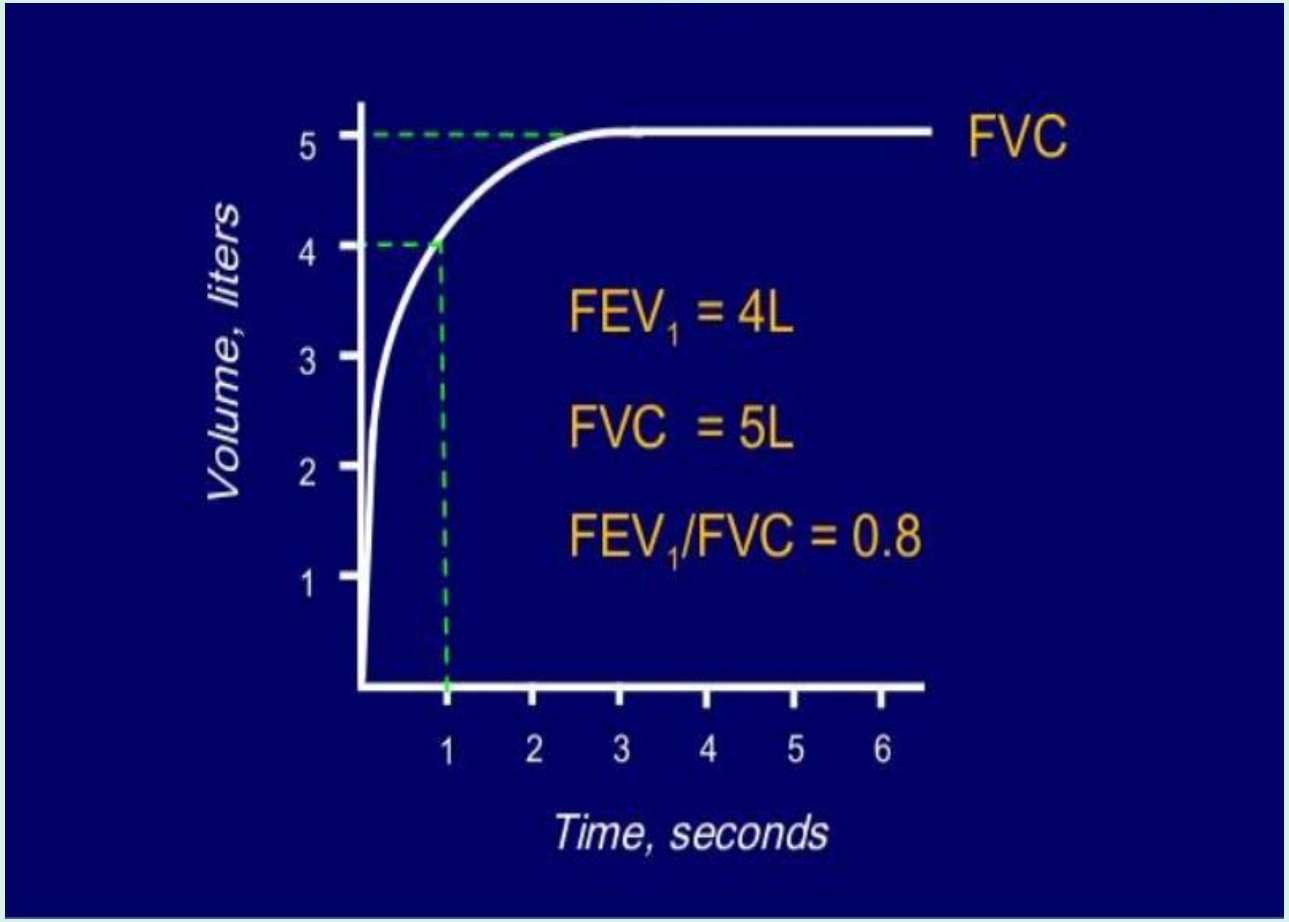


- Forced expiratory volume in 1 second: (FEV_1)
  - Volume of air forcefully expired from full inflation (TLC) in the first second
  - Measured in liters (L)
  - Normal people can exhale more than 75-80% of their FVC in the first second; thus the FEV1/FVC can be utilized to characterize lung disease

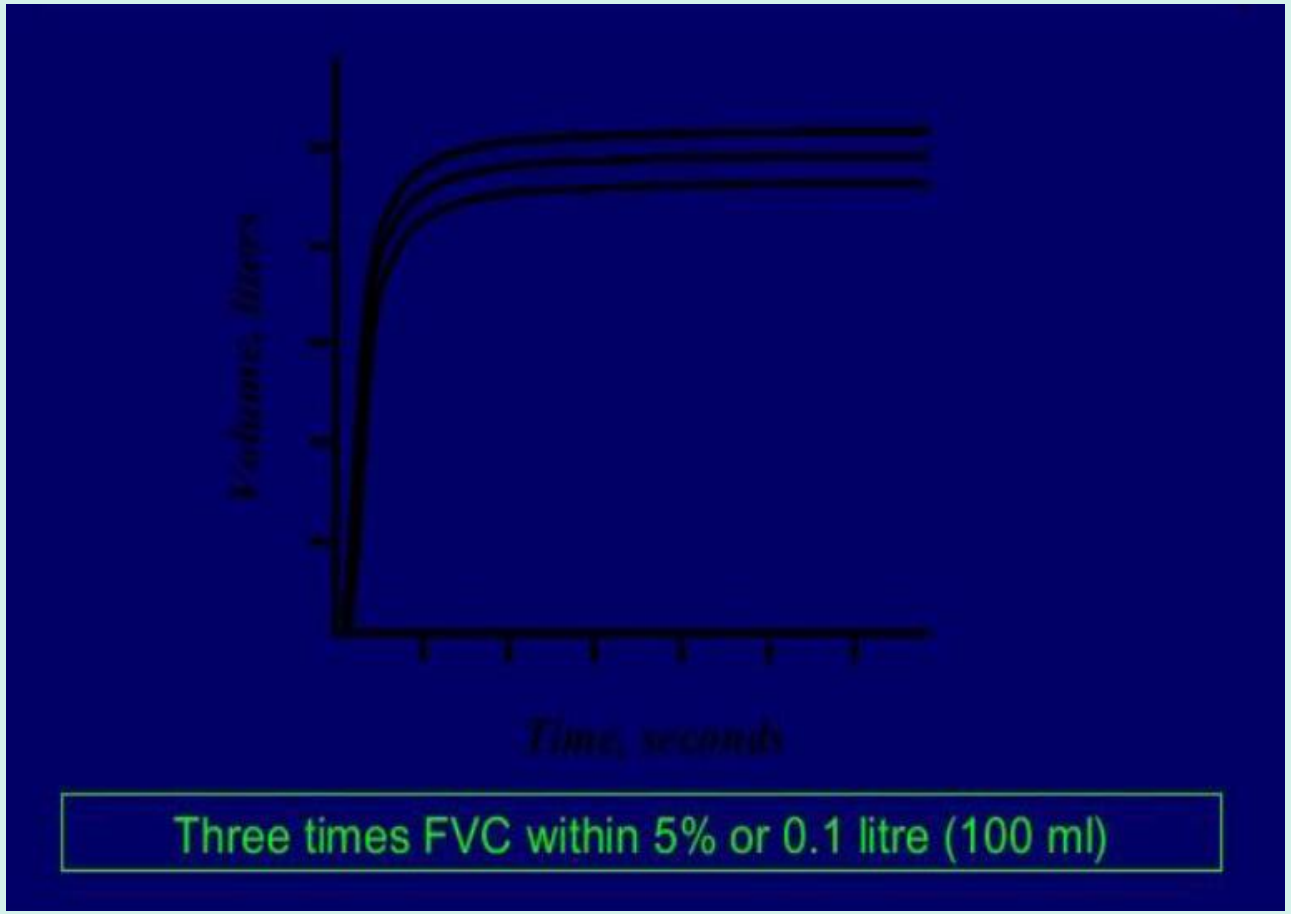
FLOW VOLUME CURVE



NORMAL TRACE SHOWING FEV1 AND FVC



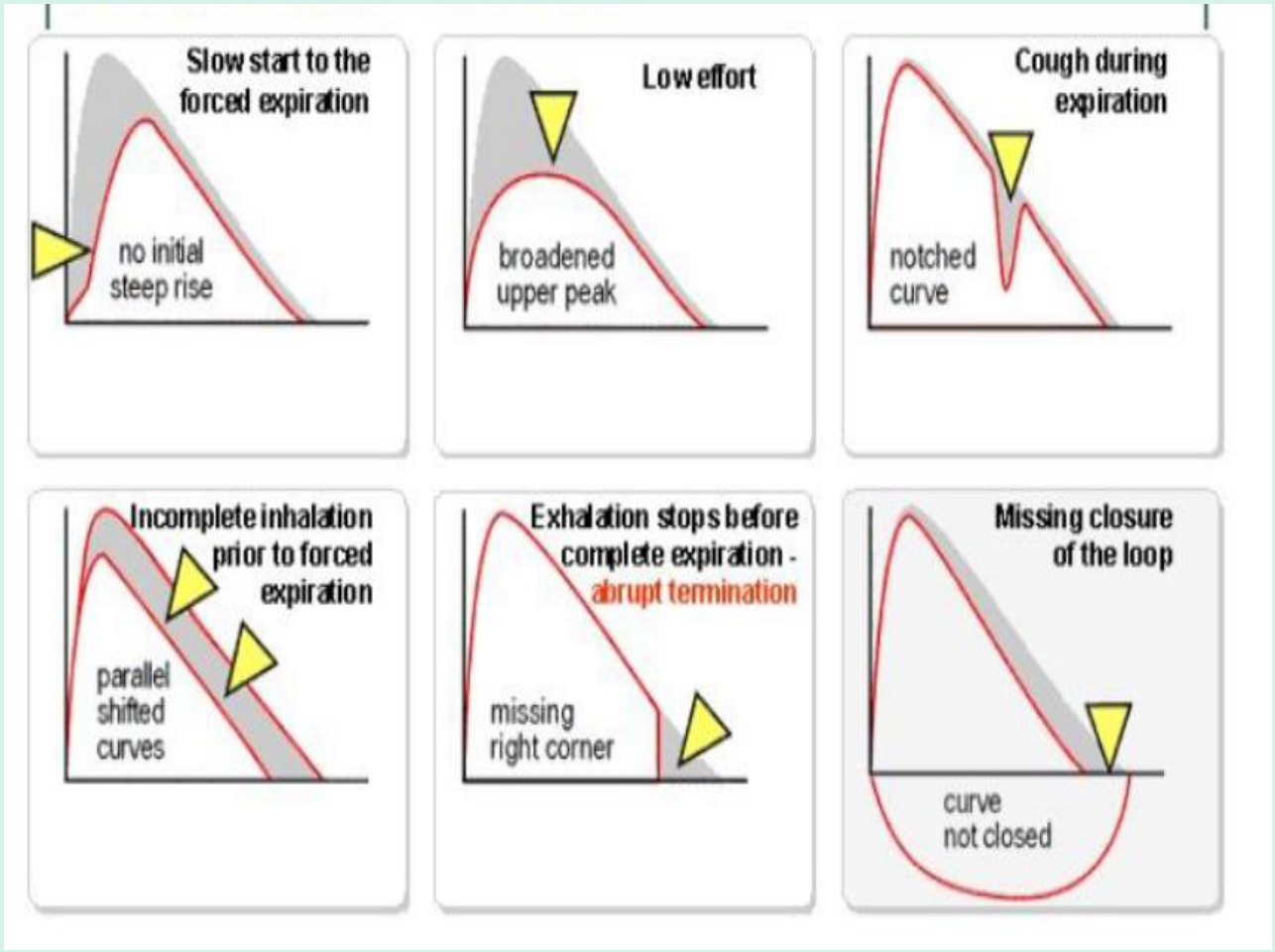
REPRODUCIBILITY - QUALITY OF RESULTS



SPIROMETRY - QUALITY CONTROL

- Most common cause of inconsistent readings is poor patient technique
  - ✓ Sub-optimal inspiration
  - ✓ Sub-maximal expiratory effort
  - ✓ Delay in forced expiration
  - ✓ Shortened expiratory time
  - ✓ Air leak around the mouthpiece
- Subjects must be observed and encouraged throughout the procedure

DETECTING POOR EFFORTS AND MISTAKES IN PERFORMING THE TEST



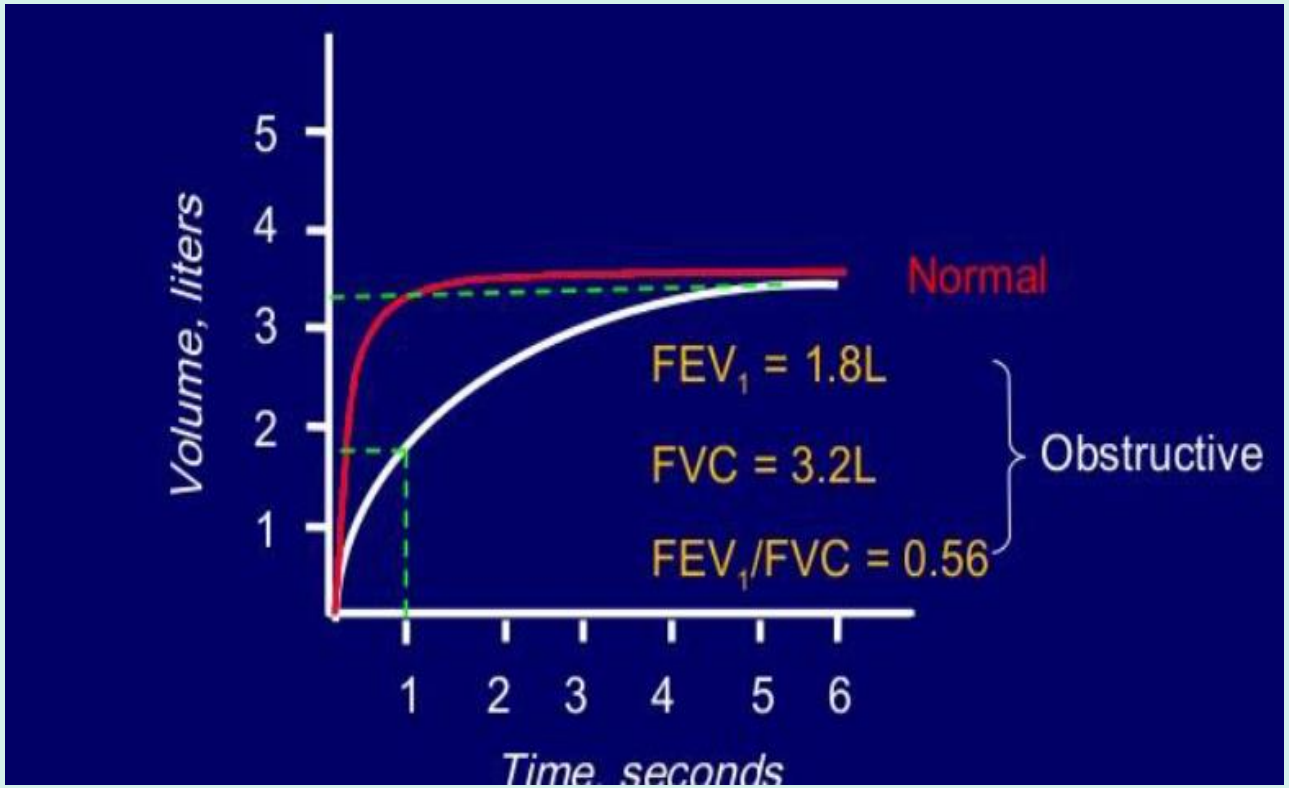
SPIROGRAM PATTERNS

- Normal
- Obstructive
- Restrictive
- Mixed Obstructive and Restrictive

CRITERIA FOR NORMAL POST-BRONCHODILATOR SPIROMETRY

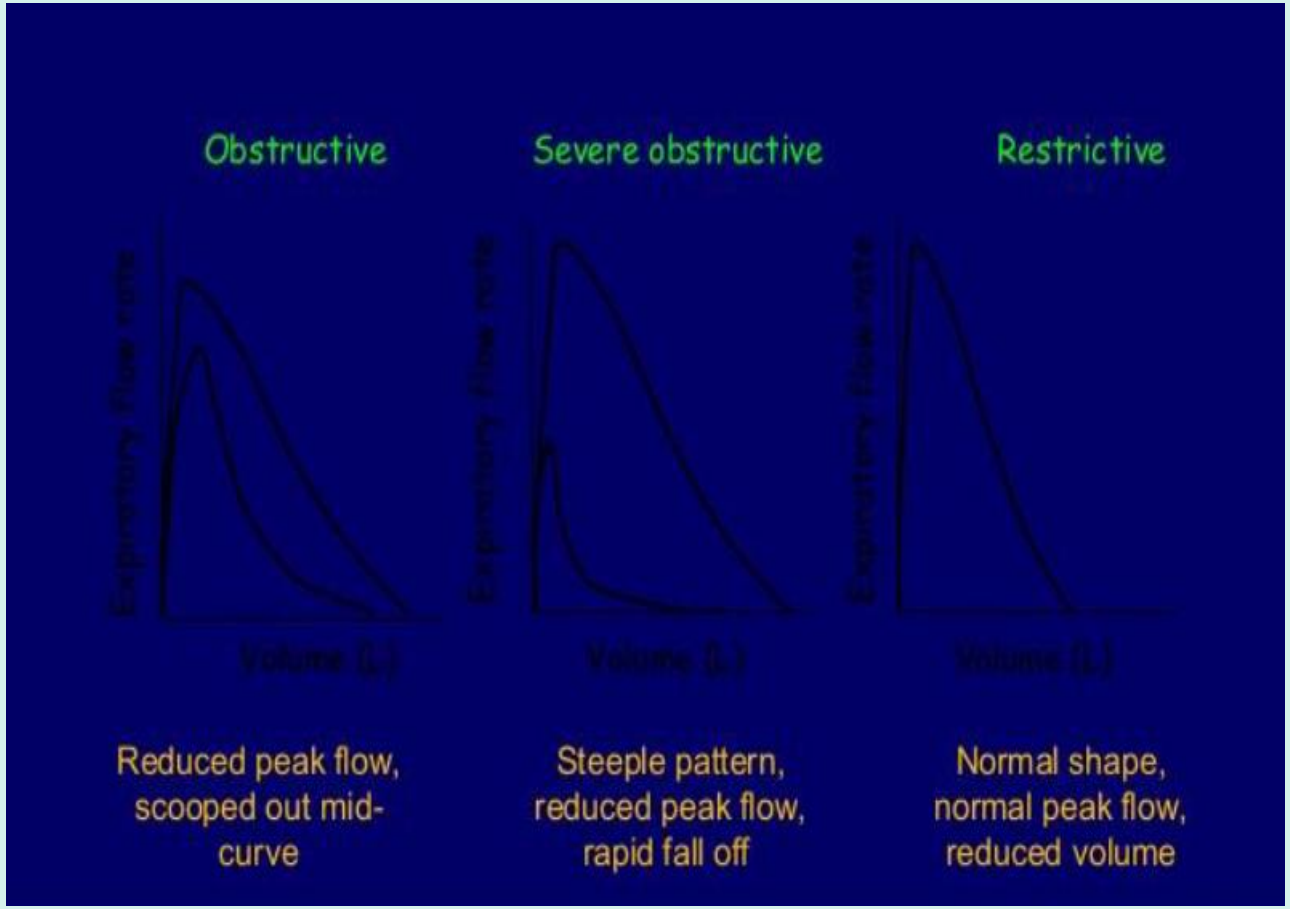
- FEV_1: % predicted $\geq$ 80%
- FVC: % predicted $\geq$ 80%
- FEV_1/FVC: > 0.7

SPIROMETRY - OBSTRUCTIVE DISEASE



- it's typical when the lung's airways are narrowed in some conditions, such as COPD, Asthma. Narrowing means that the air flows out of the lungs more slowly with less than 70% of the total amount in the first second.
- Low - Normal FVC, Lower FEV1, and Low FEV1 / FVC.

FLOW VOLUME CURVE PATTERNS - OBSTRUCTIVE AND RESTRICTIVE



SPIROGRAM - OBSTRUCTIVE

Pulmonary Function Test Results

Obeidah Masoudi MD. FCCP

Al- Khalidi hospital St. Al yasmin Medical Center
building 12
Pulmonary, Critical care &Sleep Medicine

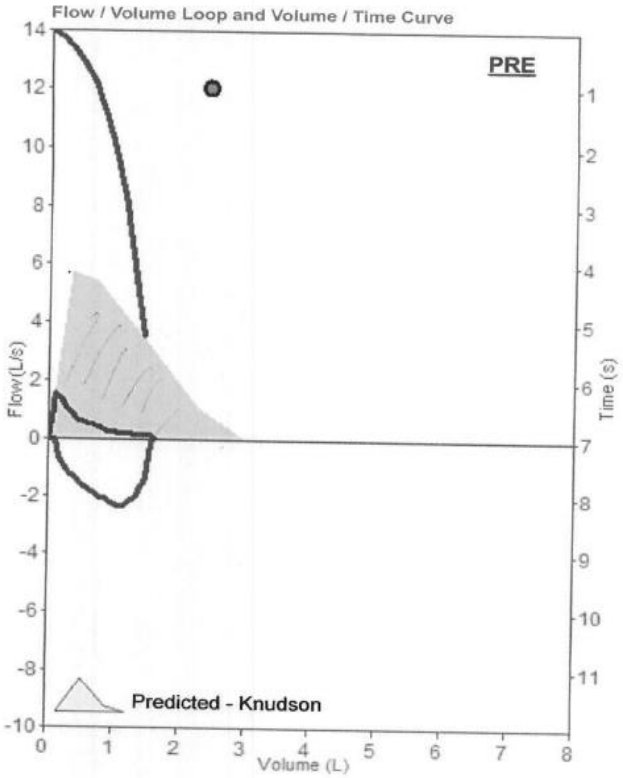
Visit date 5/7/2016

Patient code	WSP5380517840	Age	63
Surname	asi	Gender	Female
Name	[redacted]	Height, cm	164
Date of birth	5/7/19[redacted]	Weight, kg	60
Ethnic group	Caucasian	BMI	22.31
Smoke		Pack-Year	
Patient group			

Interpretation

FVC PRE | FEV1 PRE | FEV1% PRE

Very Severe Obstruction



Best values from all loops

Parameters		LLN	ULN	PRE	%Pred	Z-score	POST	%Chg
FVC	L	2.23	3.82	**1.57**	52	-3.01		
FEV1	L	1.78	3.07	**0.71**	29	-4.40		
FEV1%	%	67.4	90.7	**45.20**	57	-4.81		
PEF	L/s	3.09	8.37	**1.58**	28	-2.58		

PRE Trial date 5/7/2016 6:15:35 PM

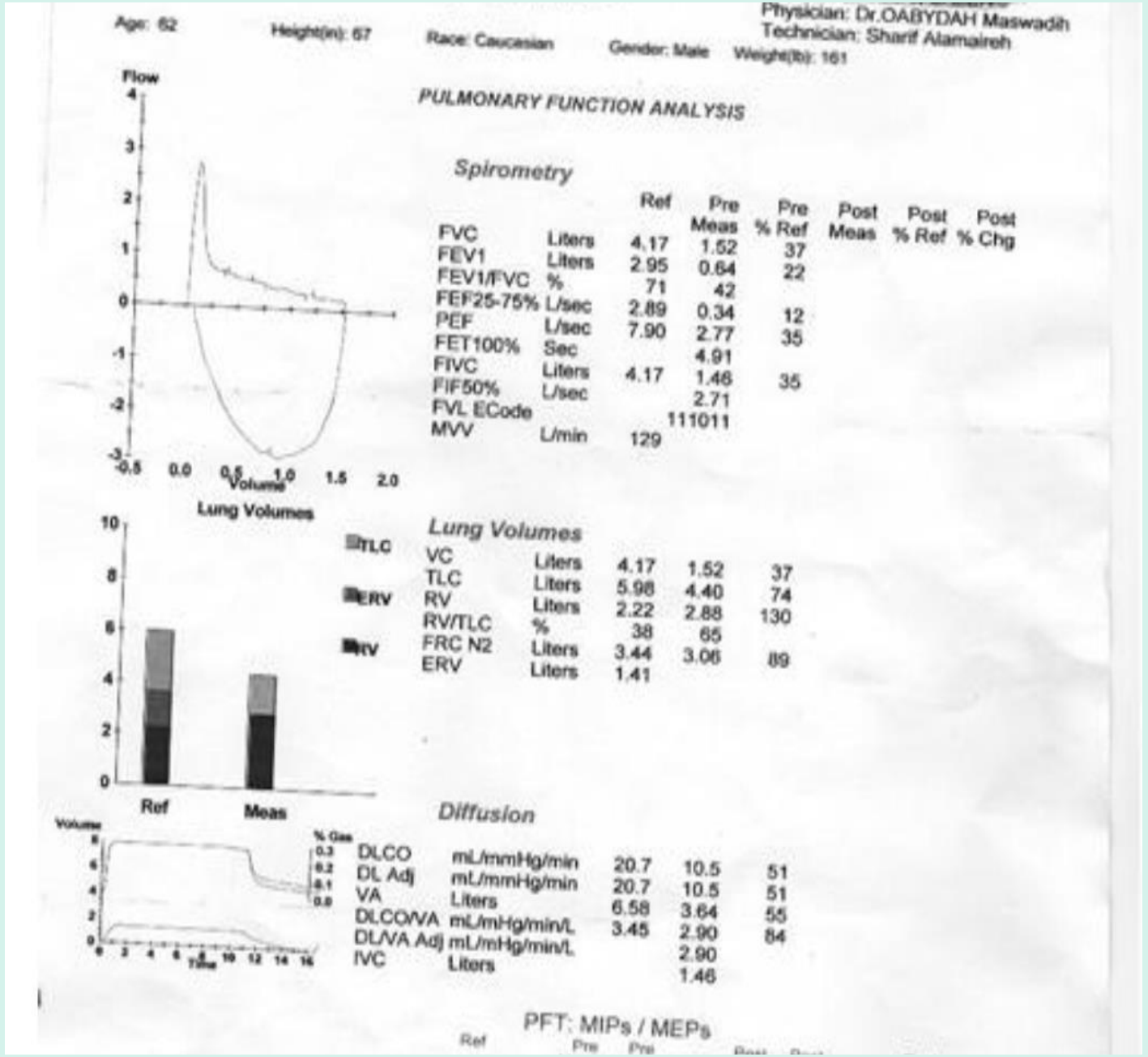
Parameters		LLN	ULN	Pred	PRE # 1	%Pred	Z-score	PRE # 2	PRE # 3	POST#1	%Pred	%Chg
FVC	L	2.23	3.82	3.02	**1.57**	52	-3.01					
FEV1	L	1.78	3.07	2.43	**0.71**	29	-4.40					
FEV1/FVC	%	67.4	90.7	79.1	**45.2**	57	-4.81					
PEF	L/s	3.09	8.37	5.73	**1.58**	28	-2.58					
ELA	Years			63	153	243						
FEF2575	L/s	1.16	3.97	2.57	**0.31**	12	-2.64					
FET	s			6.00	5.50	92						
FIVC	L	2.23	3.82	3.02	**1.43**	47	-3.30					
FEV1/VC	%	67.4	90.7	79.1								

BTPS 1.092 25 °C 77 °F

Conclusion / Medical report

Quality Report D

SPIROGRAM - OBSTRUCTIVE



Age: 62 Height(in): 67 Race: Caucasian Gender: Male Weight(lb): 161

Physician: Dr.OABYDAH Maswadih
Technician: Sharif Alamaireh

PULMONARY FUNCTION ANALYSIS

Flow

Volume

Lung Volumes

Ref Meas

Spirometry

		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	4.17	1.52	37			
FEV1	Liters	2.95	0.64	22			
FEV1/FVC	%	71	42				
FEF25-75%	L/sec	2.89	0.34	12			
PEF	L/sec	7.90	2.77	35			
FET100%	Sec		4.91				
FIVC	Liters	4.17	1.46	35			
FIF50%	L/sec		2.71				
FVL ECode			111011				
MVV	L/min	129					

Lung Volumes

		Ref	Pre Meas	Pre % Ref
VC	Liters	4.17	1.52	37
TLC	Liters	5.98	4.40	74
RV	Liters	2.22	2.88	130
RV/TLC	%	38	65	
FRC N2	Liters	3.44	3.06	89
ERV	Liters	1.41		

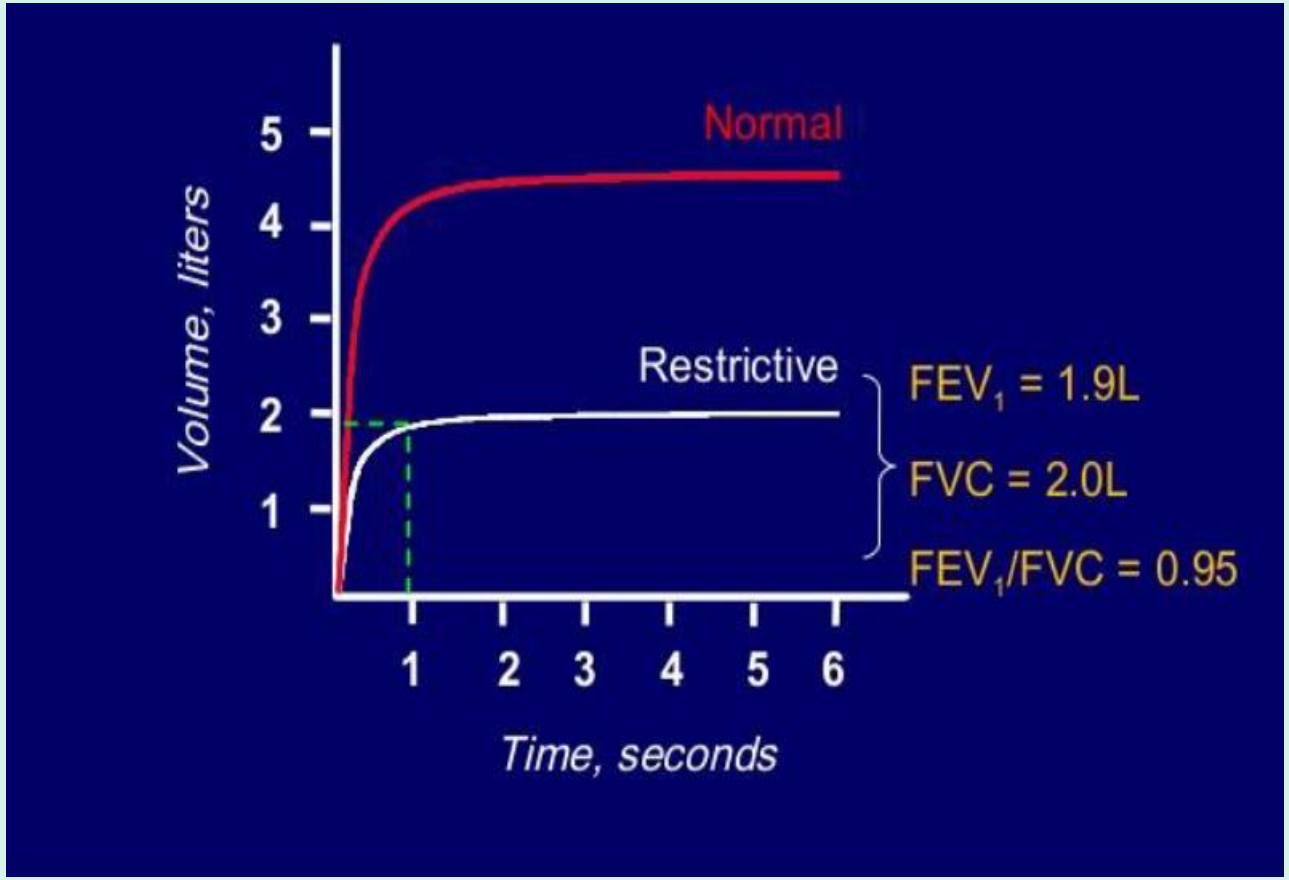
Diffusion

		Ref	Pre Meas	Pre % Ref
DLCO	mL/mmHg/min	20.7	10.5	51
DL Adj	mL/mmHg/min	20.7	10.5	51
VA	Liters	6.58	3.64	55
DLCO/VA	mL/mHg/min/L	3.45	2.90	84
DL/VA Adj	mL/mHg/min/L		2.90	
IVC	Liters		1.46	

PFT: MIPs / MEPs

Ref Pre Pre Post Post

SPIROMETRY - RESTRICTIVE DISEASE



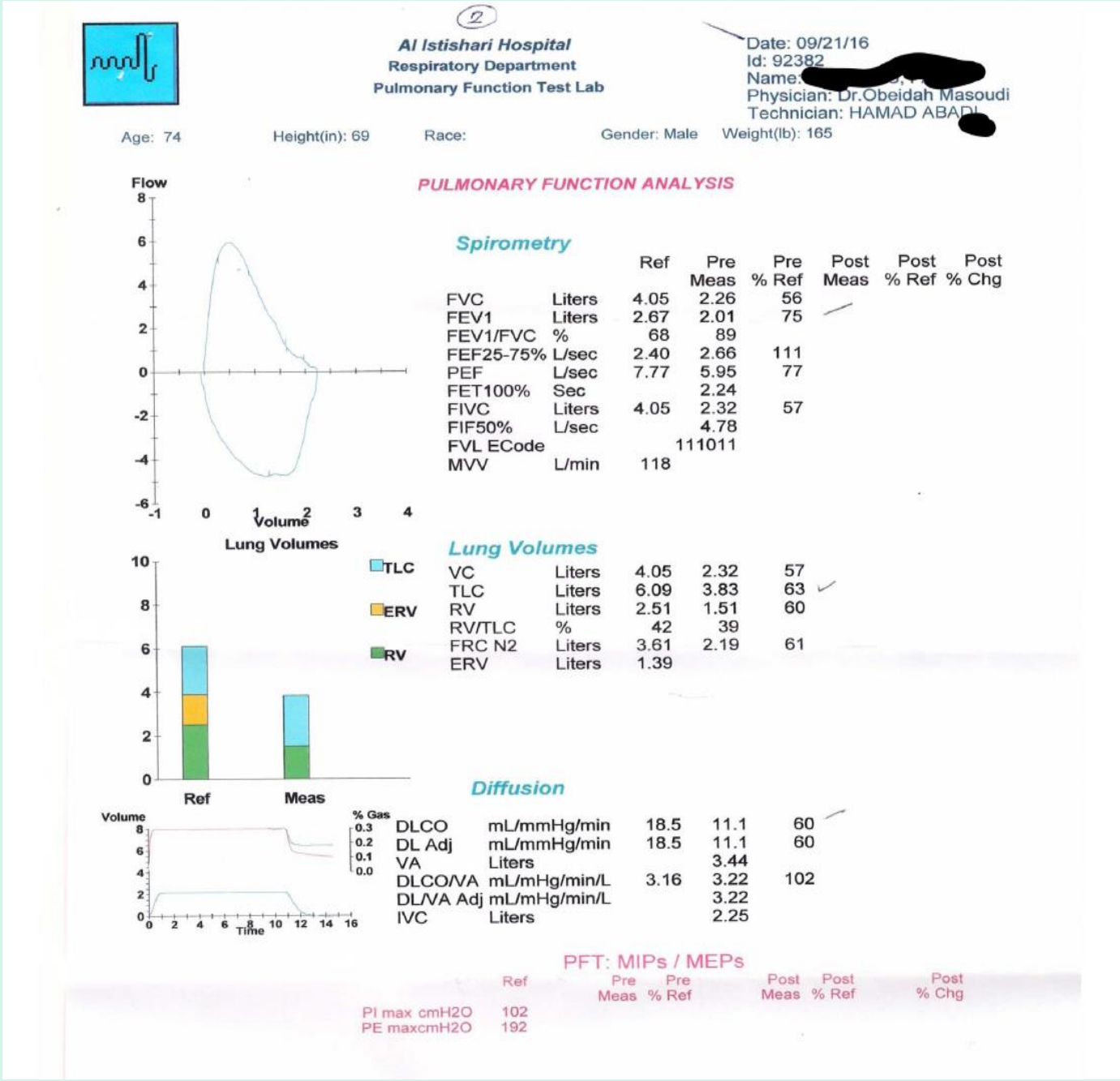
- The classic definition of a restrictive pattern on spirometry is low FVC in the presence of normal FEV1 / FVC ratio. Restrictive lung disease is characterized by a decrease in Total Lung Capacity (TLC); causing all volumes to decrease in proportion.
- Low FEV1 and FVC, Normal FEV1 / FVC

	FEV1	FVC	FEV1/FVC
Obstructive	↓↓	↓	↓
Restrictive	↓	↓	>80%

MIXED PATTERN

- Mixed lung disease has characteristics of both obstructive and restrictive lung diseases. Mixed lung disease most commonly occurs in people with chronic obstructive pulmonary disease (COPD), who also have congestive heart failure. It requires more functional tests to investigate.

SPIROGRAM - RESTRICTIVE



Al Istishari Hospital
Respiratory Department
Pulmonary Function Test Lab

Date: 09/21/16
Id: 92382
Name: [redacted]
Physician: Dr.Obeidah Masoudi
Technician: HAMAD ABADI

Age: 74 Height(in): 69 Race: Gender: Male Weight(lb): 165

PULMONARY FUNCTION ANALYSIS

Flow / Volume

Lung Volumes

Ref / Meas

Volume / Time / % Gas

Spirometry

		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	4.05	2.26	56			
FEV1	Liters	2.67	2.01	75			
FEV1/FVC	%	68	89				
FEF25-75%	L/sec	2.40	2.66	111			
PEF	L/sec	7.77	5.95	77			
FET100%	Sec		2.24				
FIVC	Liters	4.05	2.32	57			
FIF50%	L/sec		4.78				
FVL ECode			111011				
MVV	L/min	118					

Lung Volumes

VC	Liters	4.05	2.32	57
TLC	Liters	6.09	3.83	63
RV	Liters	2.51	1.51	60
RV/TLC	%	42	39	
FRC N2	Liters	3.61	2.19	61
ERV	Liters	1.39		

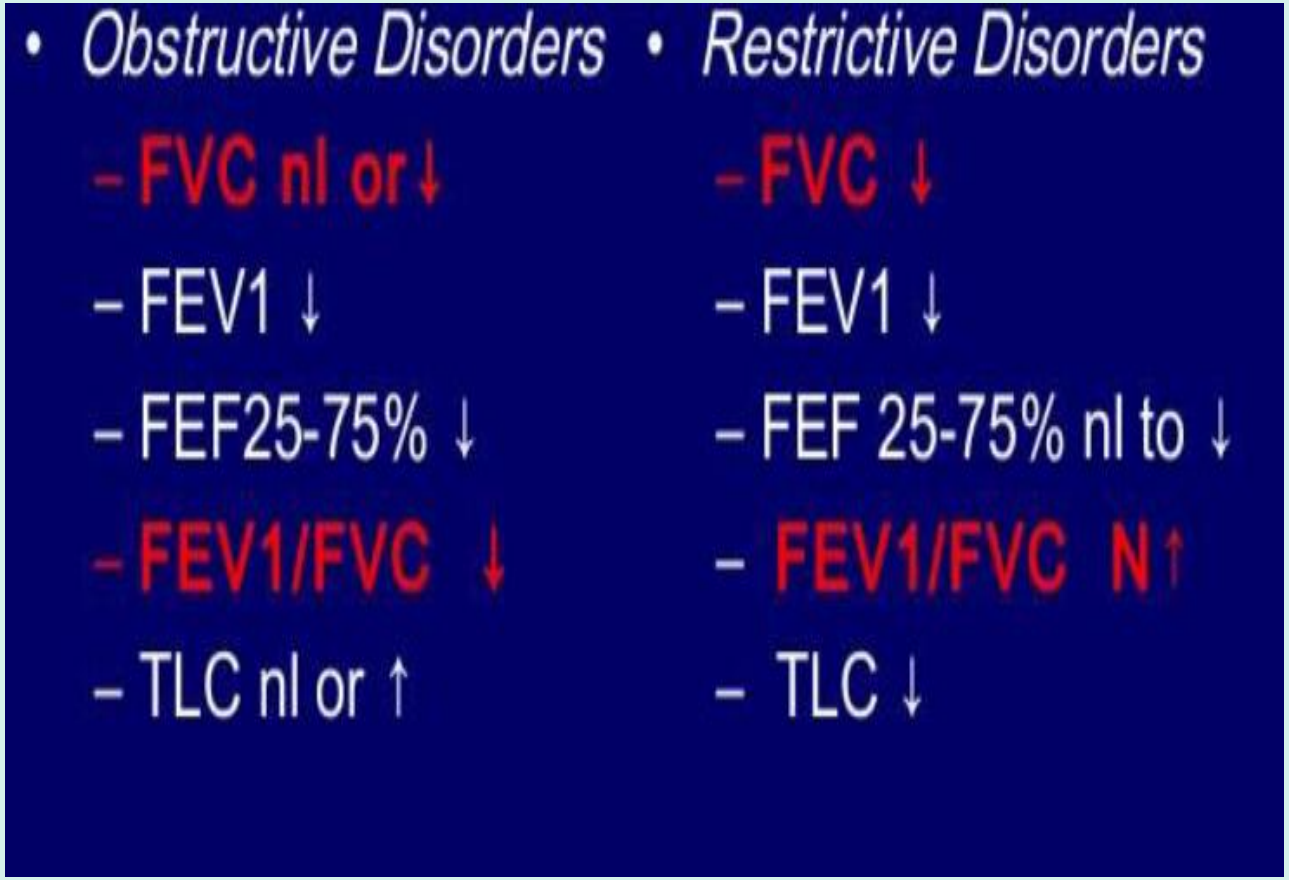
Diffusion

DLCO	mL/mmHg/min	18.5	11.1	60
DL Adj	mL/mmHg/min	18.5	11.1	60
VA	Liters		3.44	
DLCO/VA	mL/mHg/min/L	3.16	3.22	102
DL/VA Adj	mL/mHg/min/L		3.22	
IVC	Liters		2.25	

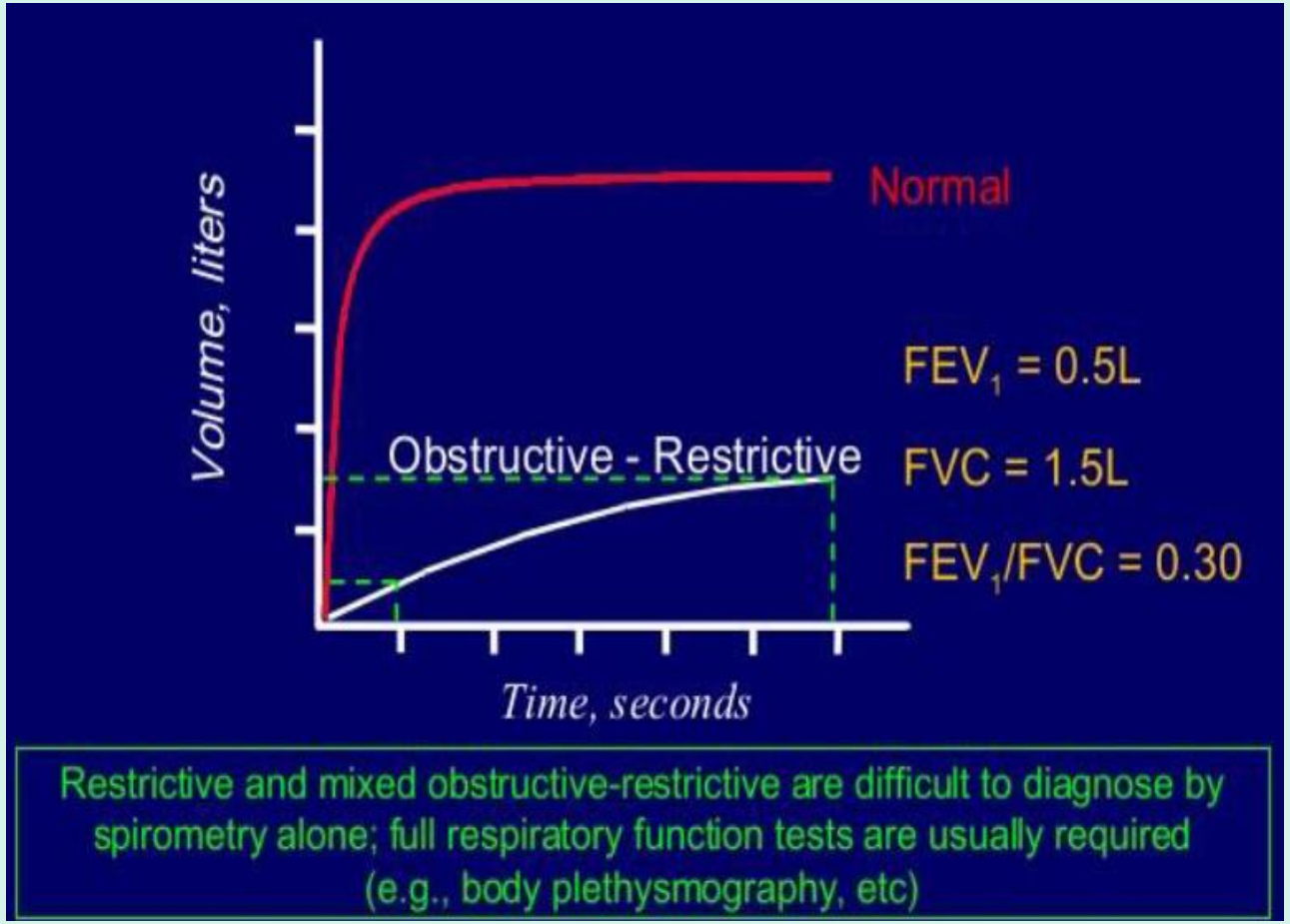
PFT: MIPs / MEPs

	Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
PI max cmH2O	102					
PE maxcmH2O	192					

SPIROMETRY INTERPRETATION: OBSTRUCTIVE VS RESTRICTIVE DEFECT

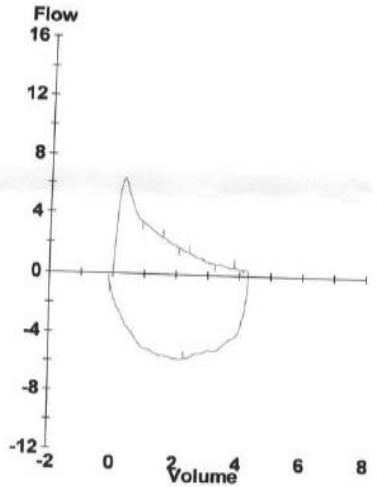


MIXED OBSTRUCTIVE AND RESTRICTIVE



OBSTRUCTIVE DEFECT

Race: Caucasian Gender: Male Weight(lb): 209

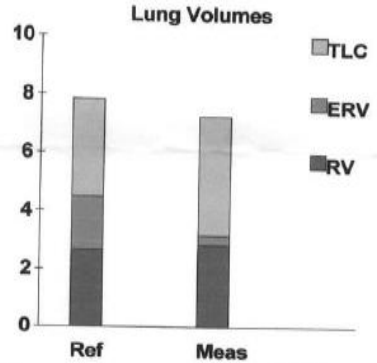


PULMONARY FUNCTION ANALYSIS

[redacted]

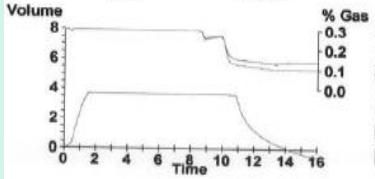
Spirometry

		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	5.40	4.28	79			
FEV1	Liters	3.80	2.45	64			
FEV1/FVC	%	70	57				
FEF25-75%	L/sec	3.46	1.34	39			
PEF	L/sec	9.87	6.45	65			
FET100%	Sec		4.34				
FIVC	Liters	5.40	4.41	82			
FIF50%	L/sec		5.74				
FVL ECode			111011				
MVV	L/min	160					



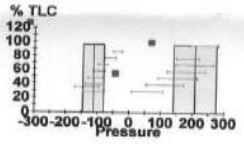
Lung Volumes

		Ref	Pre Meas	Pre % Ref
VC	Liters	5.40	4.41	82
TLC	Liters	7.85	7.25	92
RV	Liters	2.65	2.84	107
RV/TLC	%	36	39	
FRC N2	Liters	4.30	4.31	100
ERV	Liters	1.82	0.31	17



Diffusion

		Ref	Pre Meas	Pre % Ref
DLCO	mL/mmHg/min	27.8	14.2	51
DL Adj	mL/mmHg/min	27.8	14.2	51
VA	Liters	8.10	8.39	104
DLCO/VA	mL/mHg/min/L	3.54	1.70	48
DL/VA Adj	mL/mHg/min/L		1.70	
IVC	Liters		3.74	



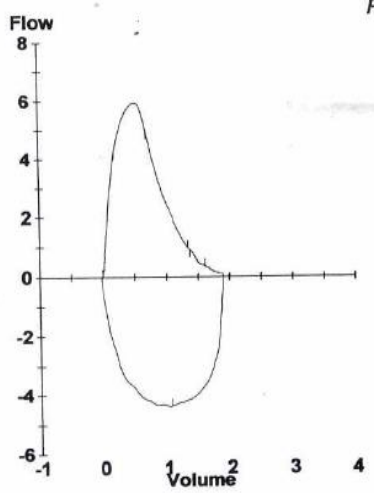
PFT: MIPs / MEPs

	Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
PI max cmH2O	112	44	39			
PE maxcmH2O	209	68	32			

RESTRICTIVE DEFECT

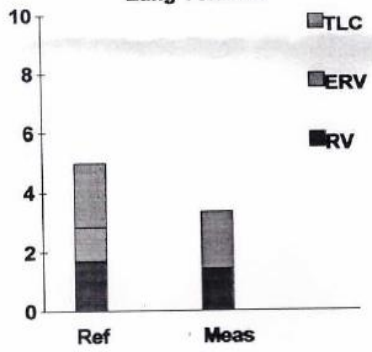
Age: 45 Height(in): 64 Race: Gender: Female Weight(lb): 229
Diagnosis: Medication:
Dyspnea Rest: No Dyspnea Exercise: No Cough: No Productive: [redacted]
Persistent: No Smoker: No How Long(pk/yrs): Stopped(yrs):
Cigarettes: No Cigars: No Temp: 21 PBar: 688

PULMONARY FUNCTION ANALYSIS



Spirometry

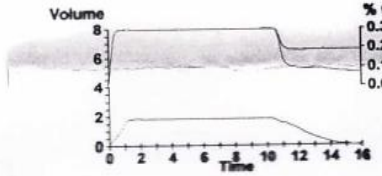
		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FVC	Liters	3.40	1.93	57			
FEV1	Liters	2.62	1.65	63			
FEV1/FVC	%	76	86				
FEF25-75%	L/sec	3.03	2.21	73			
PEF	L/sec	6.08	5.95	98			
FET100%	Sec		2.87				
FIVC	Liters	3.40	1.93	57			
FIF50%	L/sec		4.39				
FVL ECode			111011				
MVV	L/min	103					



Lung Volumes

		Ref	Pre Meas	Pre % Ref
VC	Liters	3.40	1.93	57
TLC	Liters	4.97	3.34	67
RV	Liters	1.69	1.40	83
RV/TLC	%	34	42	
FRC N2	Liters	2.11	1.78	85
ERV	Liters	1.12		

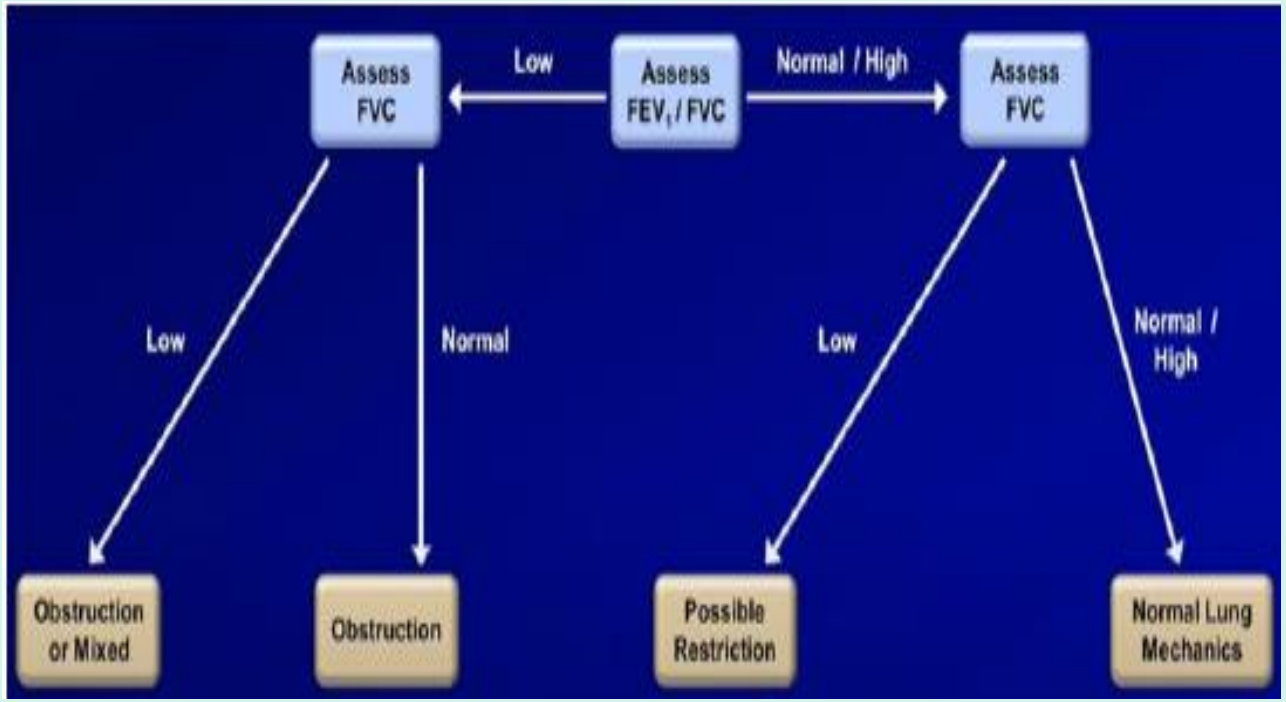
Hb:



Diffusion

		Ref	Pre Meas	Pre % Ref
DLCO	mL/mmHg/min	27.3	14.9	55
DL Adj	mL/mmHg/min	27.3	14.9	55
VA	Liters		2.93	
DLCO/VA	mL/mHg/min/L	3.82	5.10	133
DL/VA Adj	mL/mHg/min/L		5.10	
IVC	Liters		1.95	

SUMMARY



- To interpret the spirometry report:

1- First, we check the quality: Good start, absence of artifact, >6s effort, and plateau from the FET graph.

2- Then, we examine FEV1 / FVC ratio according to this figure.

- Regarding Possible restriction, we need to know the TLC to determine whether it's restrictive lung disease, or due to other causes such as obesity or rib fracture.

LUNG VOLUMES

Lung volumes are assessed using methods such as The Nitrogen washout, Helium Technique or Limb Plethysmography.

It can help in confirming spirometry diagnosis of restriction (TLC < LLN) or < 80%.

A test of the diffusing capacity of the lungs for carbon monoxide (DLCO) is one of the most valuable tests of lung function. Diffusing capacity is measured using small volumes of carbon monoxide (CO) and measures the transfer of CO across the alveolar-capillary membrane.

Should corrected for Hgb or Hct if available and should correct for carbon monoxide level if it's elevated. Abnormal if < LLN (or 80% of LLN unavailable).

Factors decreasing the DLCO (< LLN or <80%):

* Decreased total lung area, e.g. Restrictive lung disease (Fibrosis) + Pneumonia +Lung restriction.
* Decreased Alveoli Surface area, e.g. COPD (Emphysema) + Alveolitis.
* Damage to the capillary bed, e.g. Pulmonary embolism + Vasculitis.

DLCO is increased in the case of Polycythemia and Normal in Bronchial asthma

Grade severity of decrease in Diffusing capacity:

1- > 60% → Mildly decreased

2- 40 - 60 % → Moderately decreased

3- < 40% → Severely decreased

DIFFUSING CAPACITY

BEST OF LUCK ♡

Wait For Part 3 👀