

Obstructive and Restrictive lung disorders

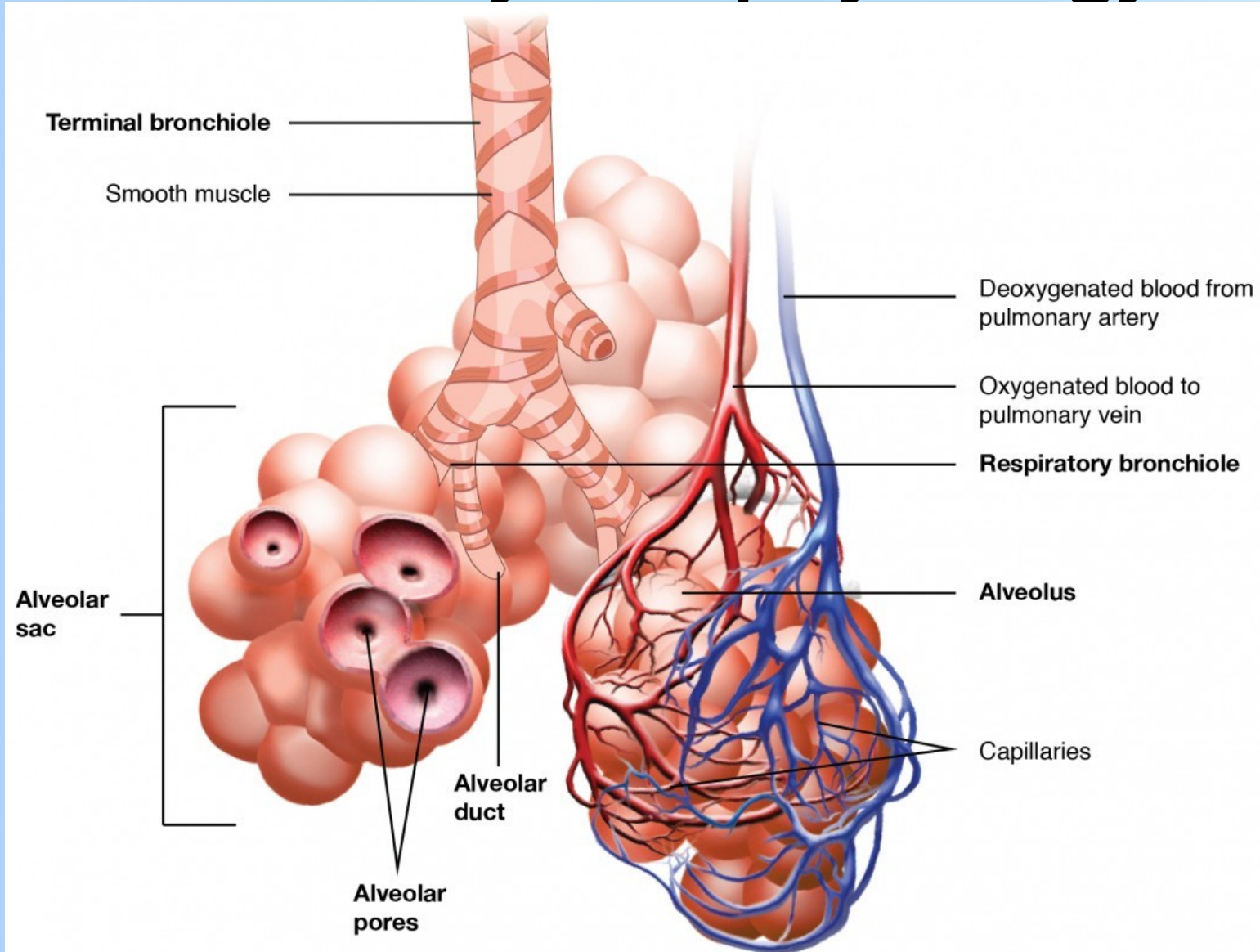
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Goals

- Distinguish obstructive vs restrictive lung disease using pathophysiology
- Predict spirometry patterns
- Explain why symptoms occur (dyspnea, wheeze, cough)
- Apply concepts to exam-style questions

Exam mindset: “What is the primary problem – airflow or lung expansion?”

Anatomy and physiology

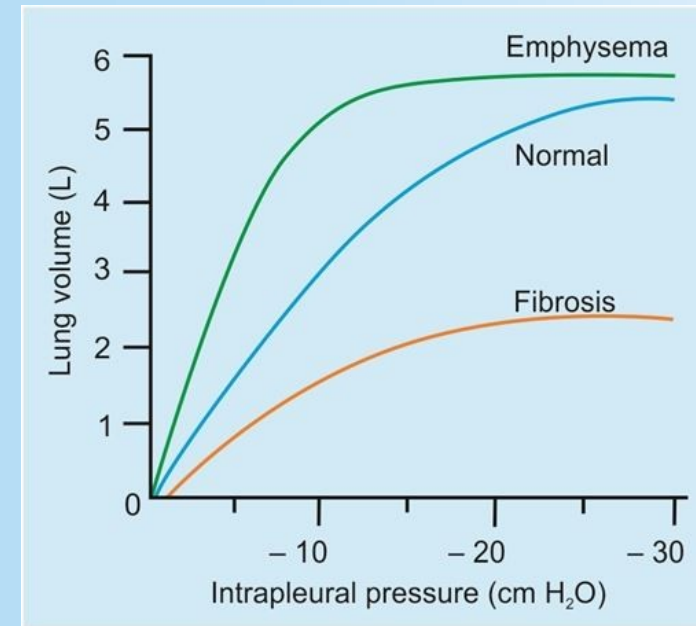


Normal physiology

Exam pearl: Expiration is normally passive — if it becomes active, pathology is present.

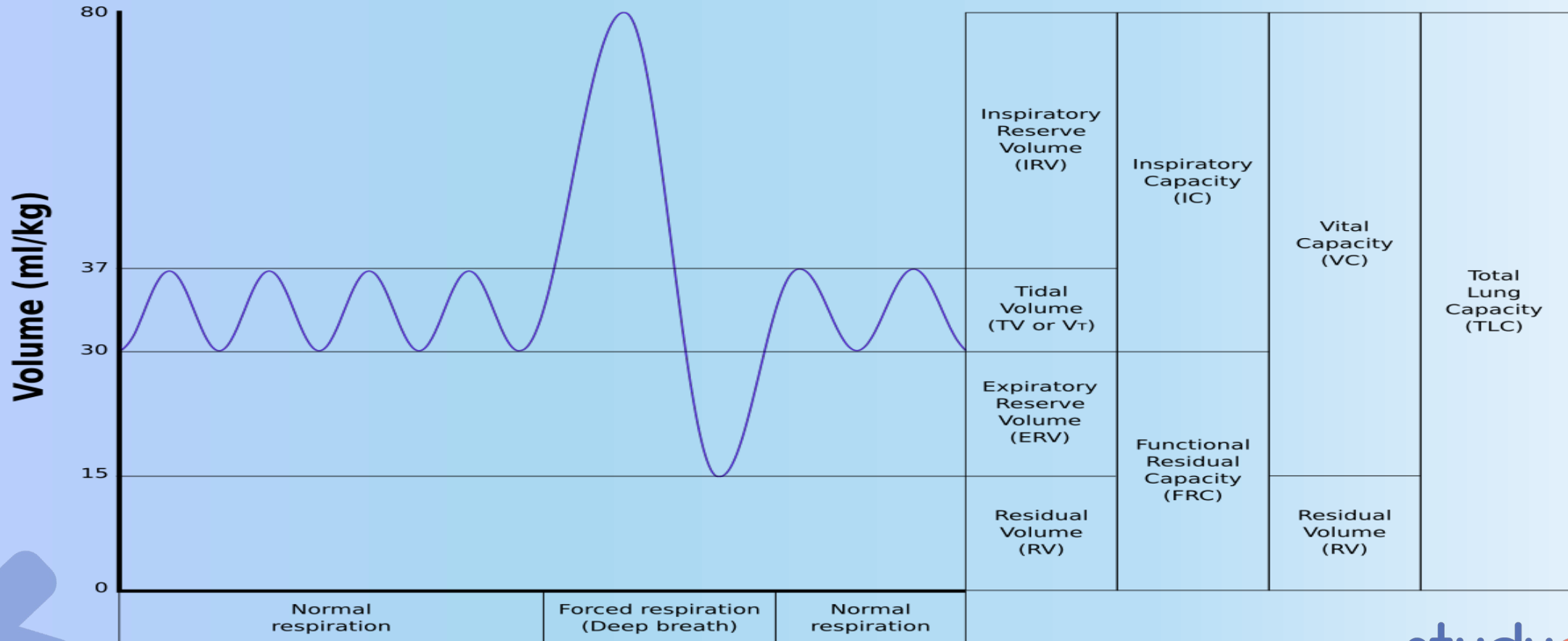
- Normal mechanics
- Airflow depends on:
 - Airway radius
 - Elastic recoil
- Lung volumes depend on:
 - Compliance ($\Delta V / \Delta P$)
 - Chest wall + lung interaction
- High-yield reminders
 - Small changes in airway radius → **huge resistance changes**
 - Elastic recoil helps **exhalation**

V: Volume
P: Pressure



Lung volumes and spirometry

- **FVC:** The maximum amount of air that can be inhaled after a forceful expiration
- **FEV1:** Forced expiratory volume after 1 second
- **FEV1/FVC:** Ratio, proportion of VC that can be expired in 1 second



DLCO

Diffusing Capacity of the Lung for Carbon Monoxide

- DLCO measures how well gas transfers from the alveoli → pulmonary capillary blood. It reflects the efficiency of the alveolar-capillary membrane.
- Carbon monoxide (CO) is used because it binds hemoglobin strongly and is diffusion-limited, making it ideal to test the membrane.

- **What DLCO Depends On (High Yield)**

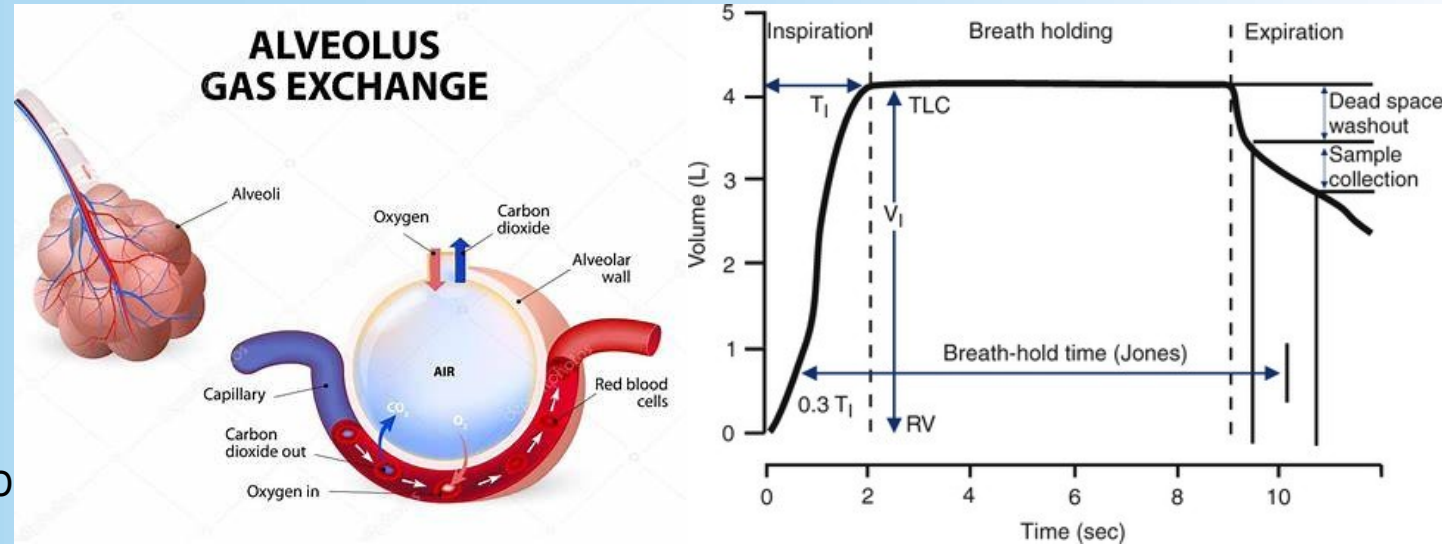
- DLCO is determined by:
 - Alveolar surface area
 - Thickness of the alveolar-capillary membrane
 - Pulmonary capillary blood volume
 - Hemoglobin concentration

- **Exam shortcut:**

$DLCO \propto \text{surface area} / \text{membrane thickness}$

- **How the Test Works**

- Patient inhales a small, harmless amount of CO
- Holds breath for ~10 seconds
- Exhaled CO is measured
- Difference = how much crossed into blood
- Low uptake → impaired diffusion

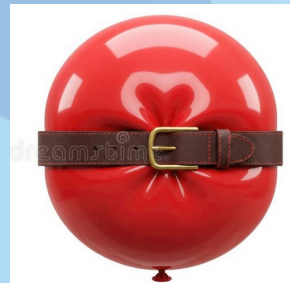


What is the difference?

Obstructive = can't get air out



Restrictive = can't get air in

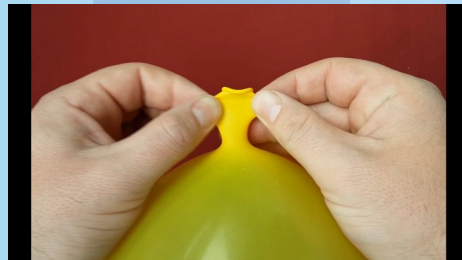


Big picture comparison

Feature	 Obstructive	 Restrictive
Main problem	Airflow out	Lung expansion
Primary phase affected	Expiration	Inspiration
TLC	Normal or ↑	↓
FEV1	↓↓↓	↓
FEV1/FVC	↓	Normal or ↑
Compliance	Normal or ↑	↓

Obstructive disorders

Asthma & COPD



Core pathophysiology

Exam pearl:
Patients “breathe at higher lung volumes” to keep airways open.

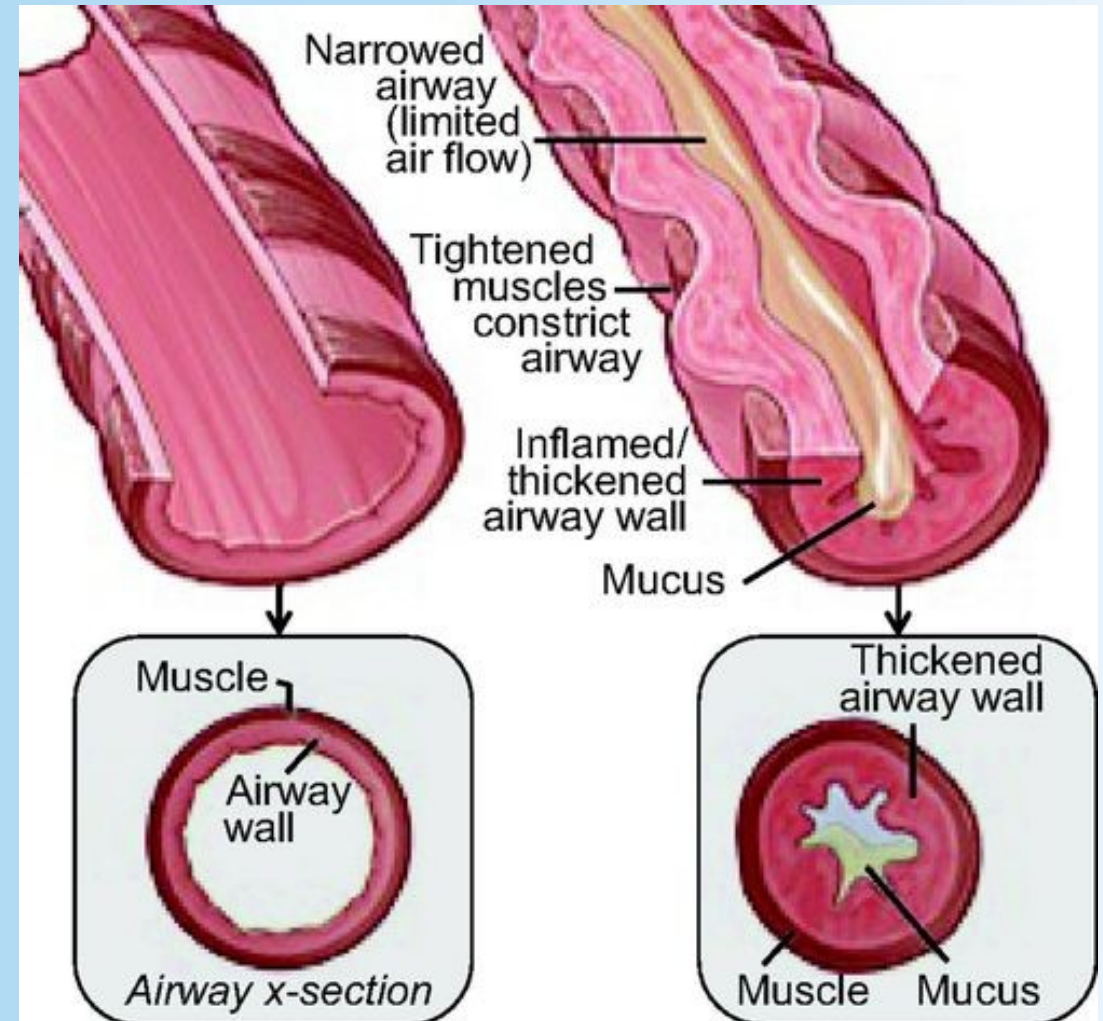
Shared mechanism

- $\uparrow$ Airway resistance $\rightarrow$ $\downarrow$ airflow during **expiration**
- Dynamic airway collapse
- **Air trapping $\rightarrow$ hyperinflation**
- **Why expiration is worse**
 - Loss of elastic recoil
 - Increased intrathoracic pressure during expiration collapses airways



Asthma

- Reversible
- Difficulty breathing
 - Upper respiratory tract infections
 - In relation to physical activity
- Persistent dry cough
- Wheezing on expiration!!
- Triad:
 - Inflammation
 - Bronchoconstriction
 - Mucus
- Triggered → episodic



Asthma

Allergic asthma

- Child
- Eczema
- Family history
- Responsive to ICS

Non-allergic asthma

- Adult
- Smoker
- Construction worker - living in cities with bad air quality
- Obese
- Stress
- Not responsive to ICS

ICS: Inhaled corticosteroids

Pathogenesis of allergic asthma

A type 1 hypersensitivity reaction (IgE)

1. Bronchial hyperresponsiveness

Triggered by inhaling antigen: dust, pollen, pet hair, mold spores

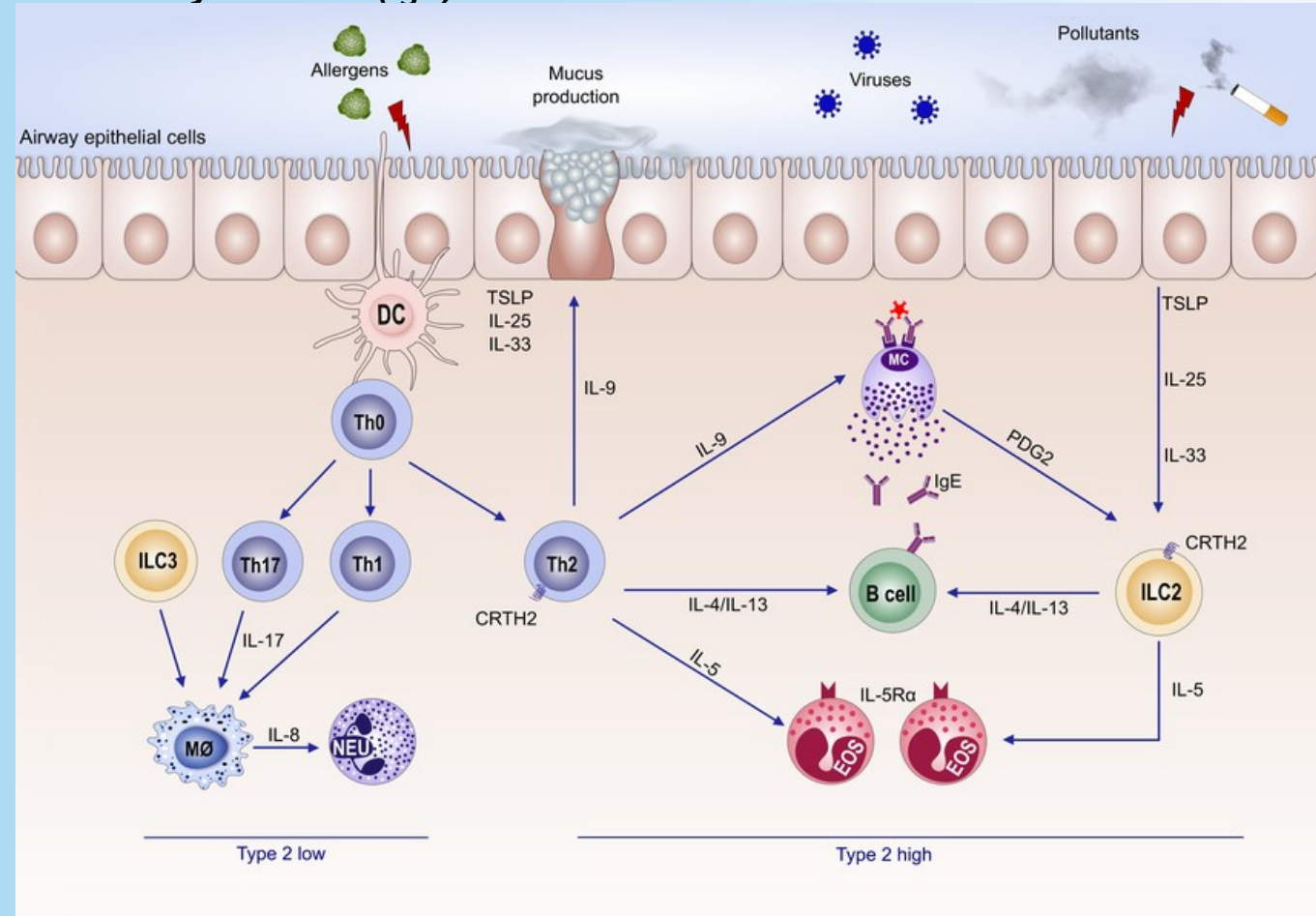
Leads to activation of Th₂-cells → Release of cytokines

2. Bronchial inflammation

- Edema of bronchioles & smooth muscle contraction → bronchial collapse
- Increased mucus production
- Activation of eosinophils and plasma cells → cellular immune response: ↑ vascular permeability

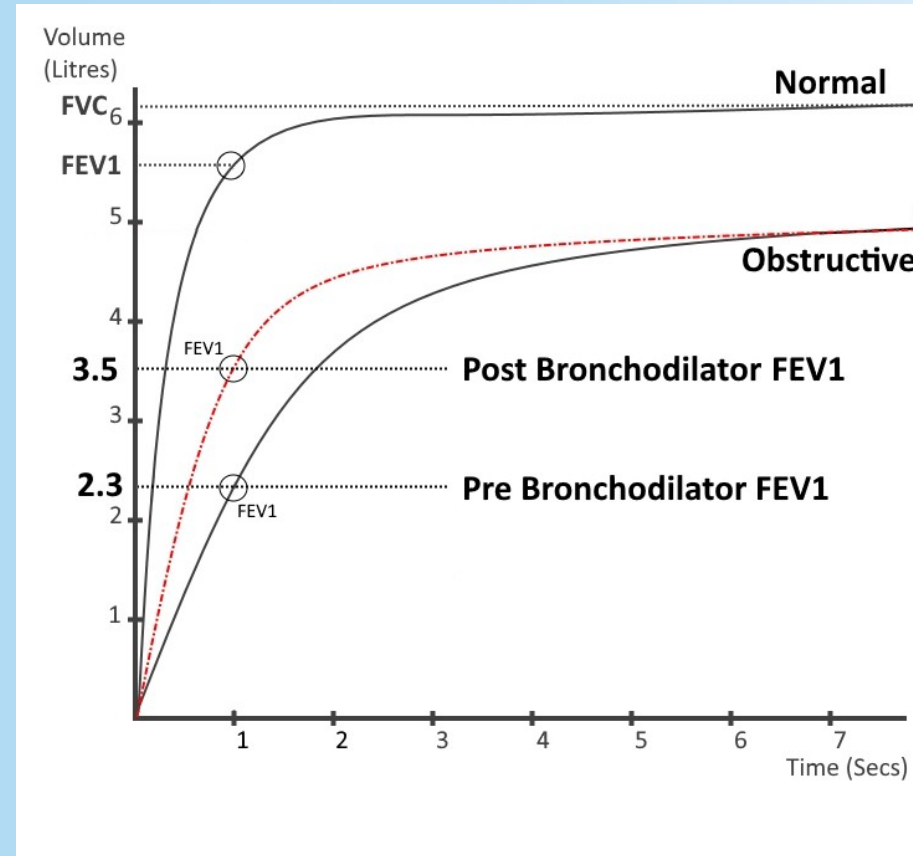
3. Endobronchial Obstruction

Remember? Can't breathe OUT!



Diagnosis of Asthma

- Typical clinical features
- Demonstration of reversible bronchoconstriction
 - We use inhaled bronchodilator
 - Significant improvement: $> 12\%$ improved FEV1



Chronic Obstructive Pulmonary Disorder (COPD)

- Chronic bronchitis
- Emphysema
- **Irreversible** airway obstruction
 - Due to chronic inflammation of the small airways, and destruction of pulmonary parenchyma
- Risk factors:
 - Smoking (90%)
 - Air pollution
 - Upper respiratory tract infections
 - α -1 antitrypsin deficiency (emphysema)

Clinically, patients usually present with elements of BOTH chronic bronchitis and emphysema, but distinguishing them is relevant for exams



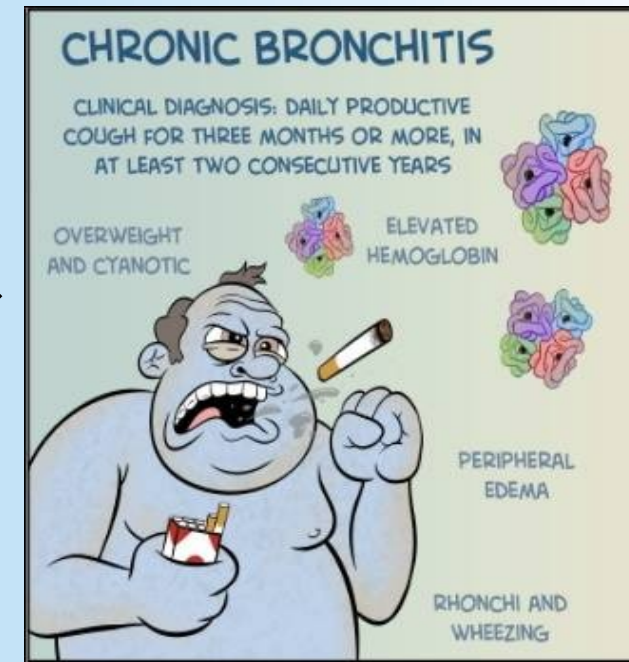
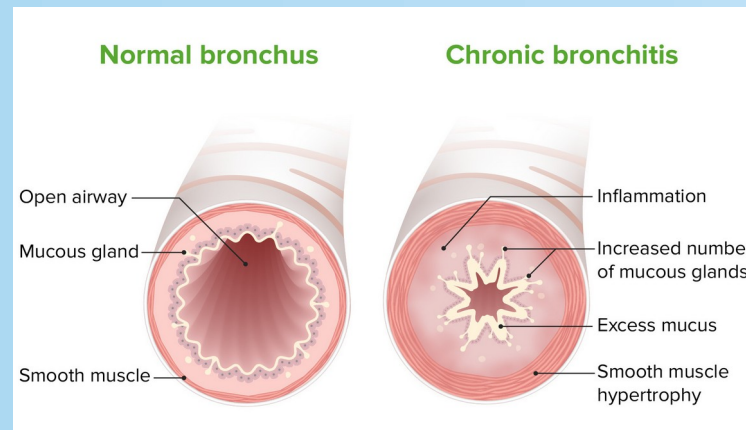
COPD: Chronic bronchitis

“The Blue Bloater”

1. Prolonged exposure to irritants
2. Hyperplasia and hypertrophy of mucus glands in the airways
 1. Bronchial mucus cells in bronchi
 2. Goblet cells in bronchioles
3. CO₂ buildup
4. Hypercapnia and hypoxemia
 1. Hypercapnia: ↑ CO₂ in arterial blood
 2. Hypoxemia: ↓ O₂ in arterial blood

→ Cyanosis

Decreased alveolar O₂, but with normal perfusion causes decreased systemic O₂.



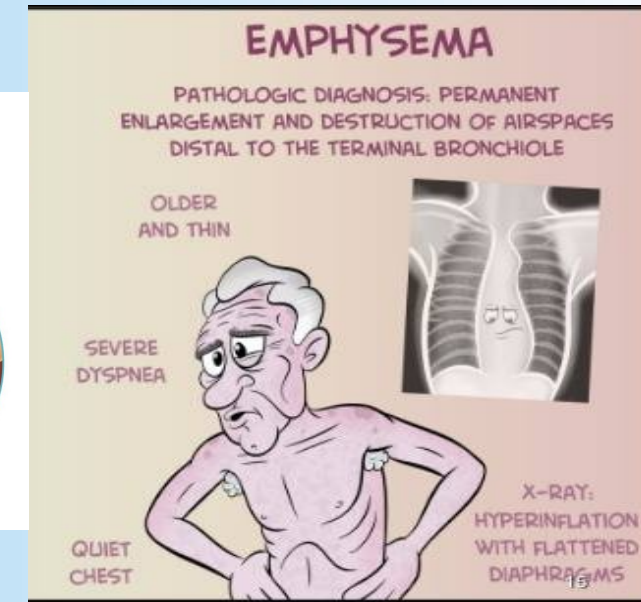
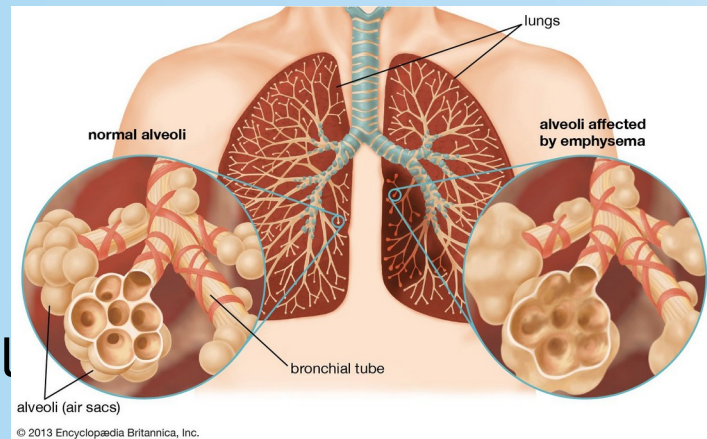
COPD: Emphysema

“The Pink Puffer”

α -1
antitrypsin
deficiency

1. Prolonged exposure to irritants
2. Hypersensitivity reaction
 1. Activation of neutrophils
 2. Release of proteases (elastase)
3. Elastase breaks down elastin
 1. Elastin is crucial for elastic recoil

📄👉 ➔ Collapse of the bronchiole
4. Airway collapse during ventilation
Causes “Air Trapping”
Patient can overcome this with pursed-lip breathing



Comparison

- Chronic bronchitis

- Defined by clinical presentation
- Productive cough for at least 3 months over 2 consecutive years

Clinical signs and symptoms:

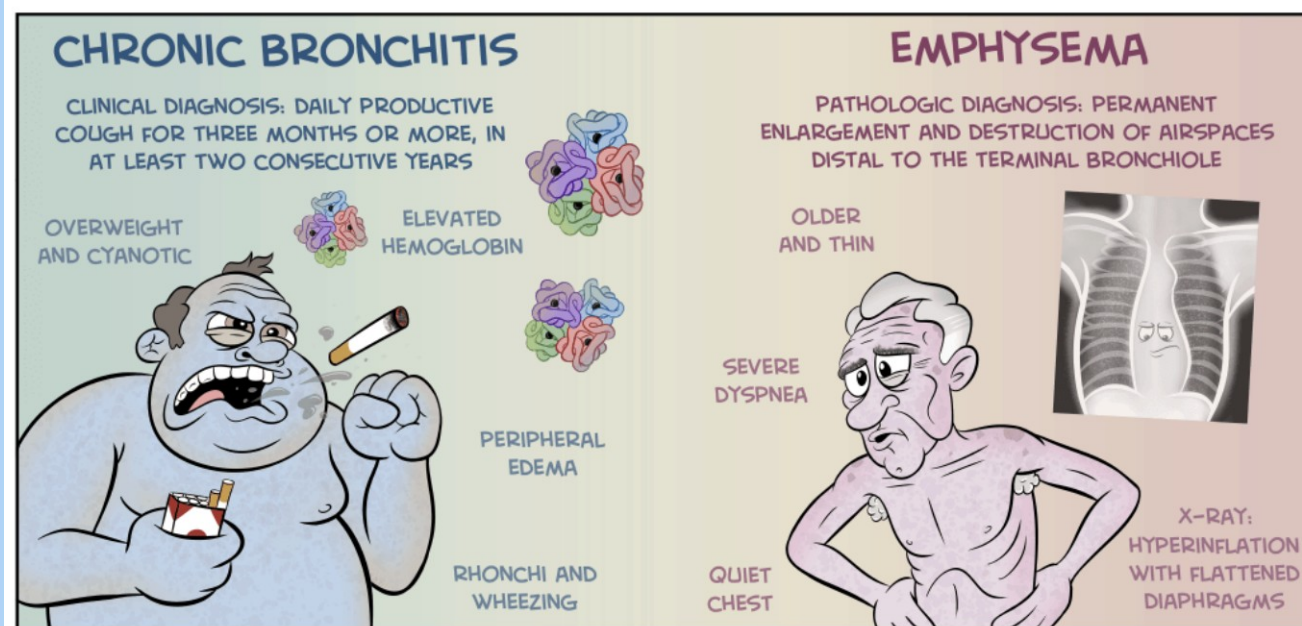
- Productive cough
- Cyanosis
- Wheezing/crackles on inspiration!!**
- Overweight/peripheral edema
- Increased hemoglobin levels
- Dyspnoea

- Emphysema

- Defined by structural presentation
- Permanent enlargement and destruction of pulmonary airways distally to the terminal bronchioles

Clinical signs and symptoms:

- Dyspnoea
- Acyanotic, but pink skin
- Huge “Barrel chest”
- Skinny

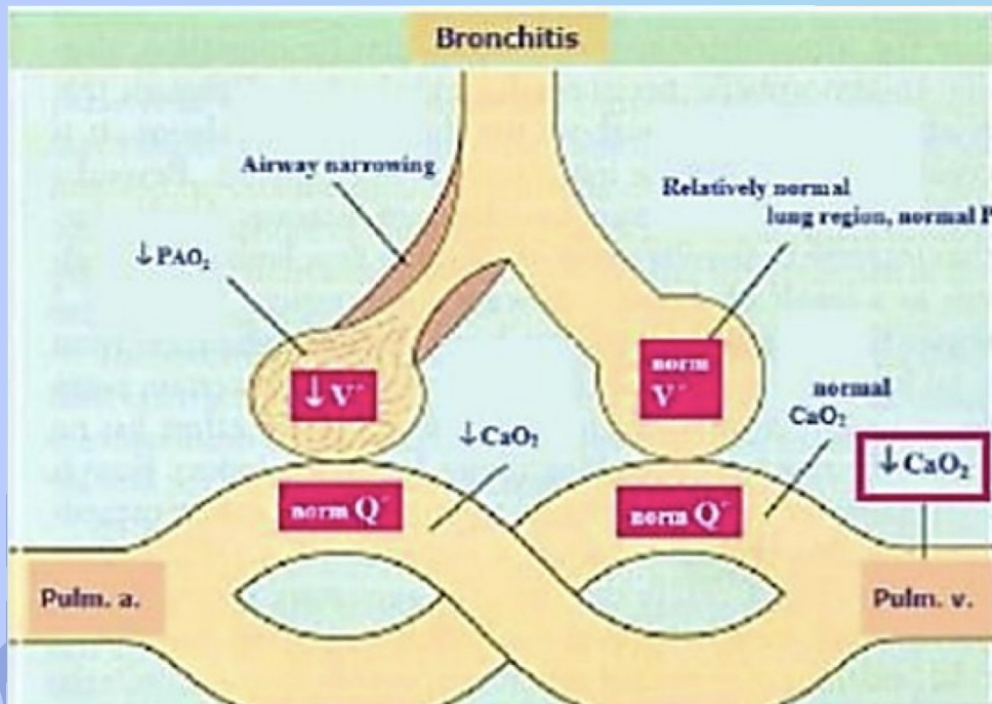


athing

Comparison

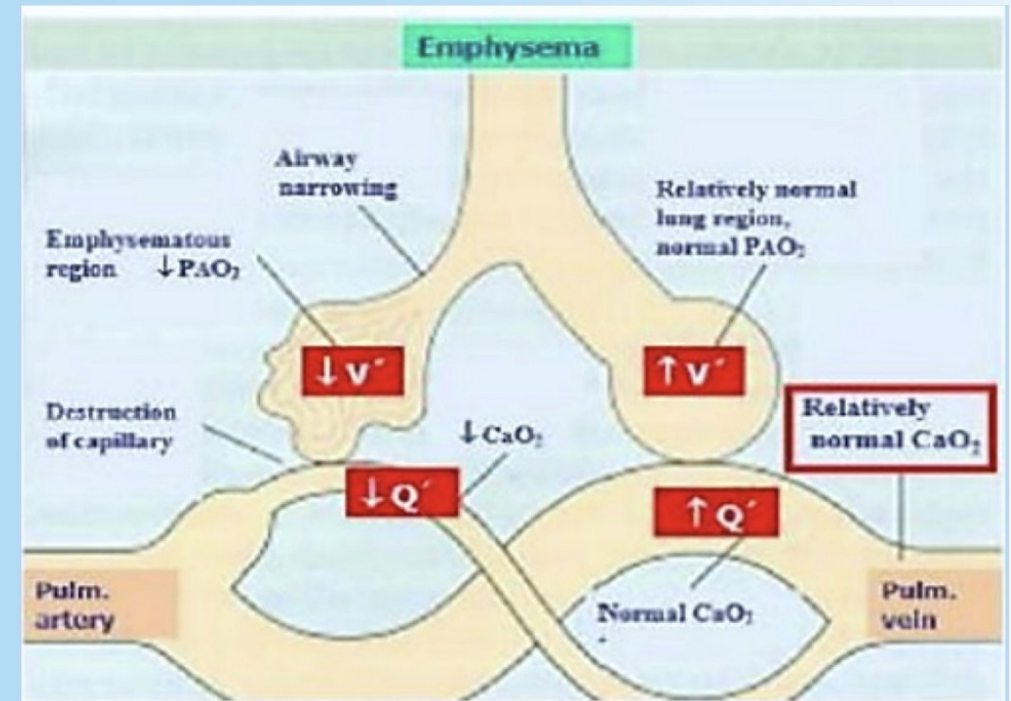
Chronic bronchitis: Cyanotic

Decreased alveolar O_2 , but with normal perfusion → decreased arterial O_2



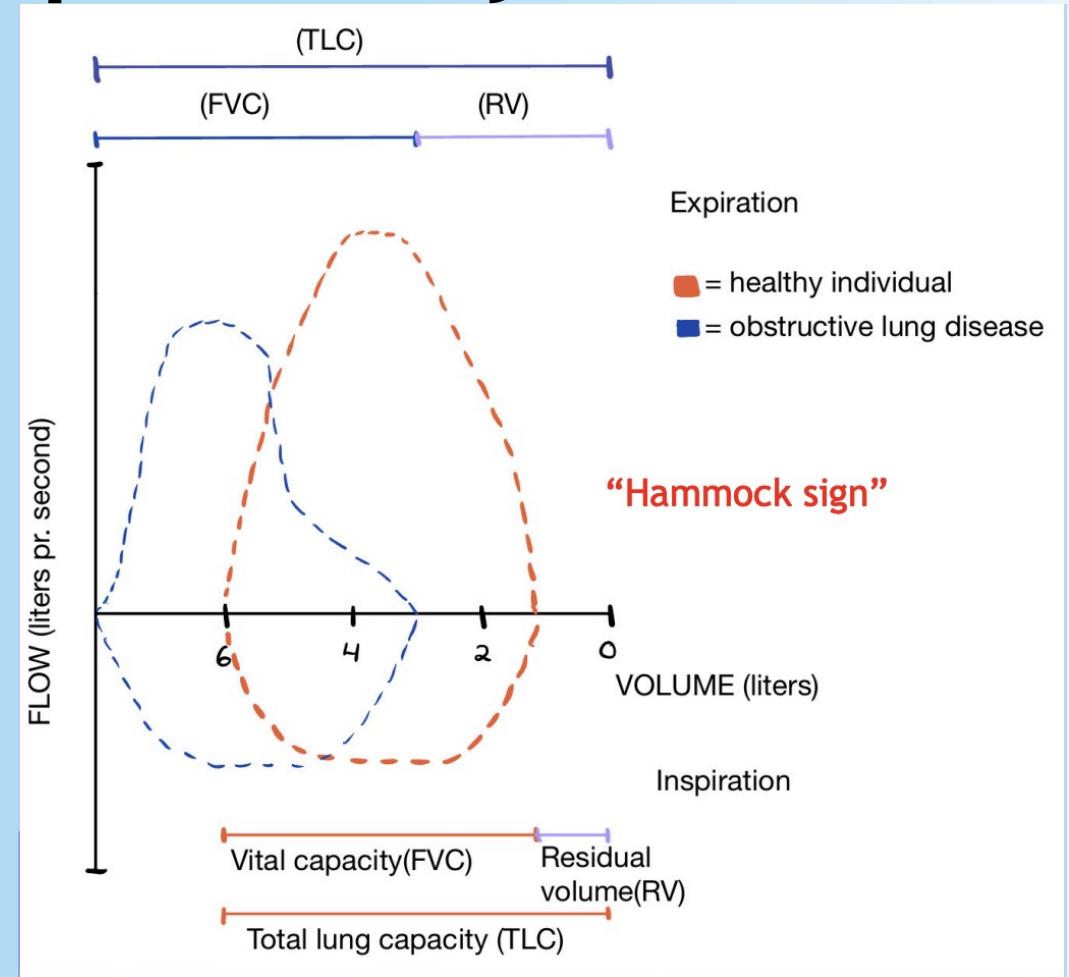
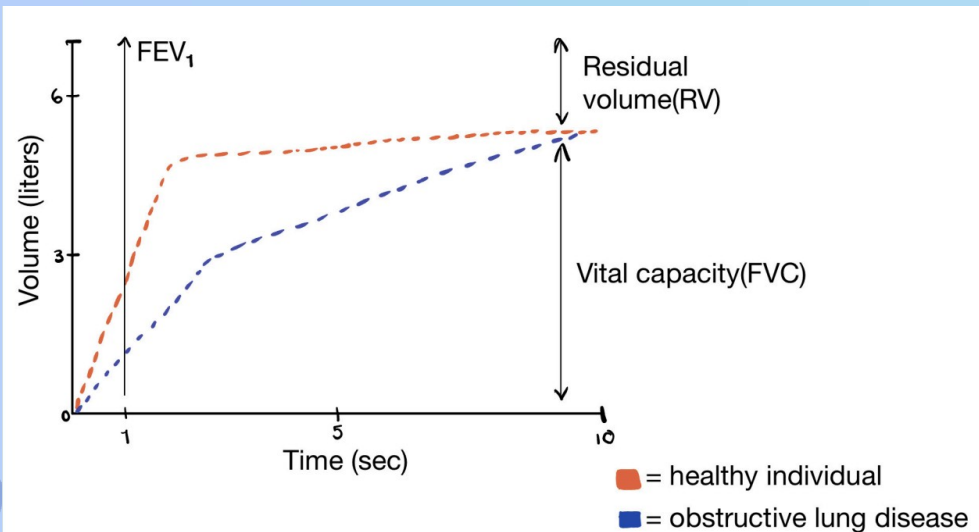
Emphysema: Acyanotic

Destruction of capillaries causes shunting towards functioning alveoli/parts of the lung
↓DLCO



Obstructive spirometry

- **↓↓↓ FEV₁**
 - Normal or slightly **↓ FVC**
 - **↓ FEV₁/FVC ratio, <70%**
 - **↑ RV and TLC**
- If the ratio drops, it's obstructive!



When you realize you
need to take a break



Restrictive disorders

Intrinsic, extrinsic and neuromuscular



Core pathophysiology

Exam pearl: Patients
can exhale, but can't
expand lungs

- Reduced **lung compliance**
 - Decreased lung expansion from the applied pressure
- High work of breathing
- Shallow, rapid breathing

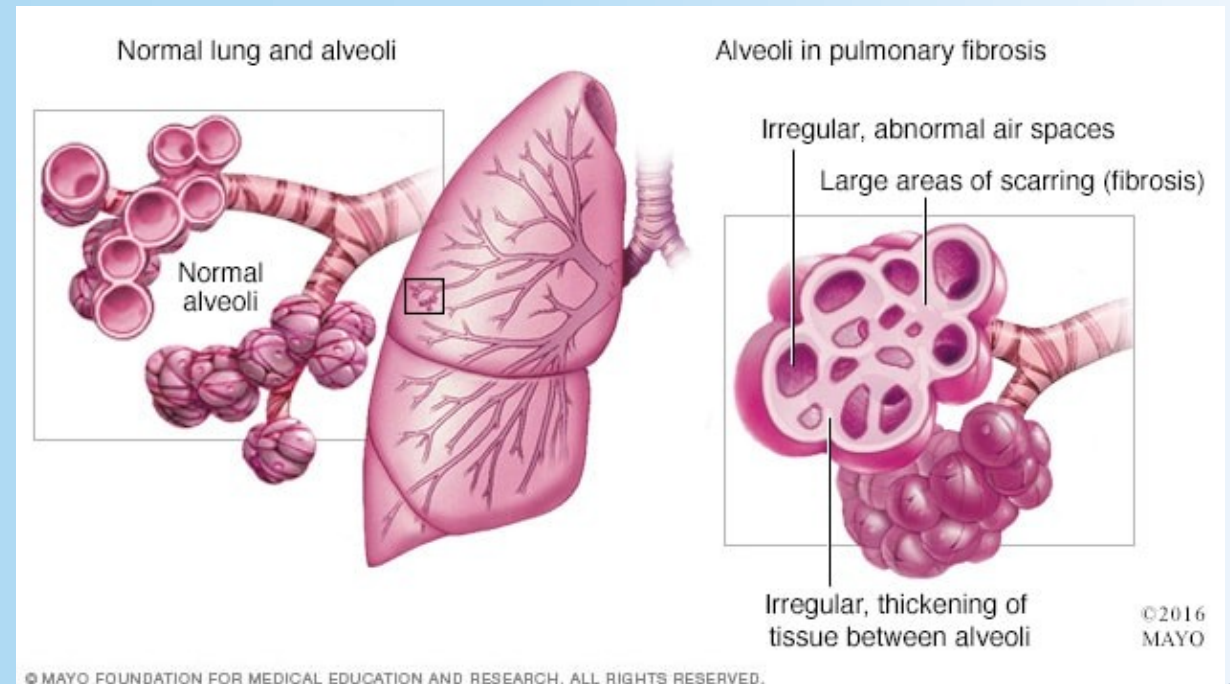


Intrinsic

- Thickening of the interstitial tissue in the lungs from fibrosis
 - ➔ Stiffer lung, meaning harder to expand, meaning **less compliance!**
 - Also means: harder for gases to diffuse accross the diffusion membrane
 - ↓ DLCO

Examples:

- Idiopathic Pulmonary Fibrosis
- Exposure related restrictive lung disorders
- Sarcoidosis



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Extrinsic

- Problems outside of the lung that **restrict** lung expansion

- Examples:
 - Obesity
 - Kyphoscoliosis
 - Pleural disease

Normal DLCO!



Neuromuscular

- Weakening of the respiratory muscles
 - → Less able to inhale sufficiently

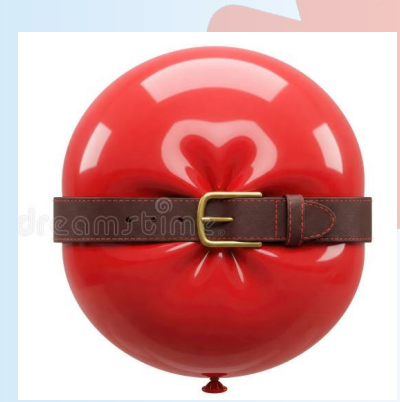
Examples:

- Myasthenia Gravis
- Amyotrophic Lateral Sclerosis (ALS)
- Guillain-Barré syndrome

Tip: Normal lungs + low volumes → Think neuromuscular

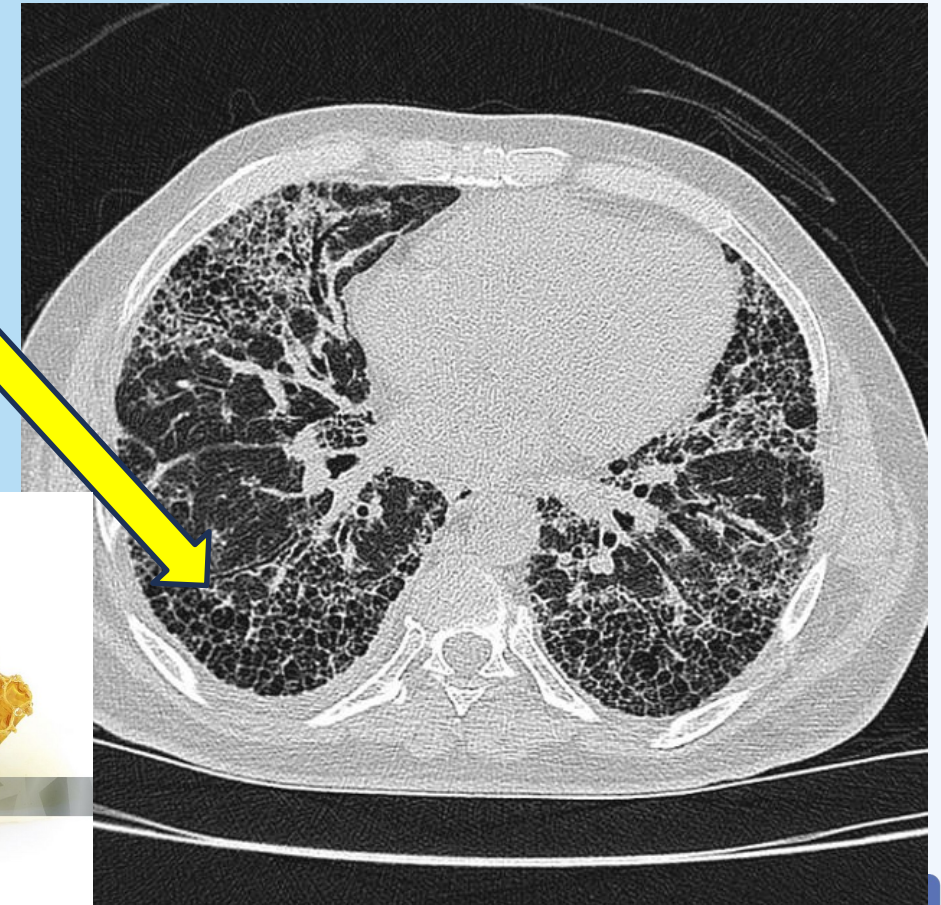
Idiopathic Pulmonary Fibrosis

IPF



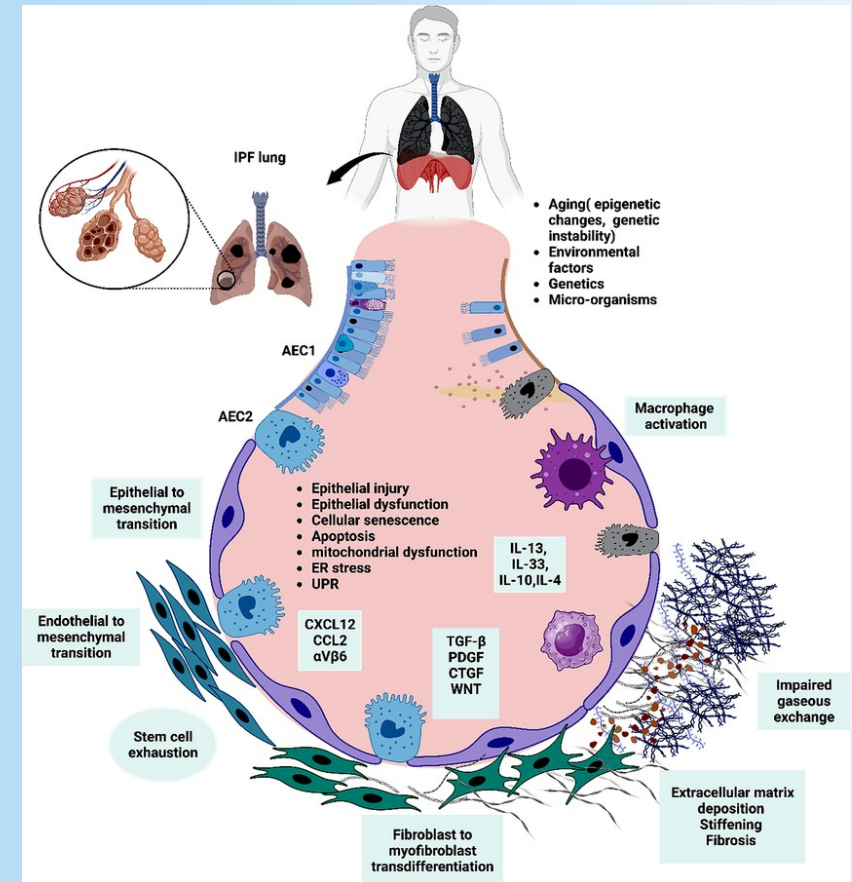
- Most common restrictive lung disorder
- Progressive - worsens over time
 - Coughing
 - Shortness of breath
 - Cyanosis
 - Over time: Lungs lose functional tissue → significant respiratory failure
- Affects males more than females
- Smoking is a risk factor (like for everything else...)

CT-scan:
- Honeycomb appearance



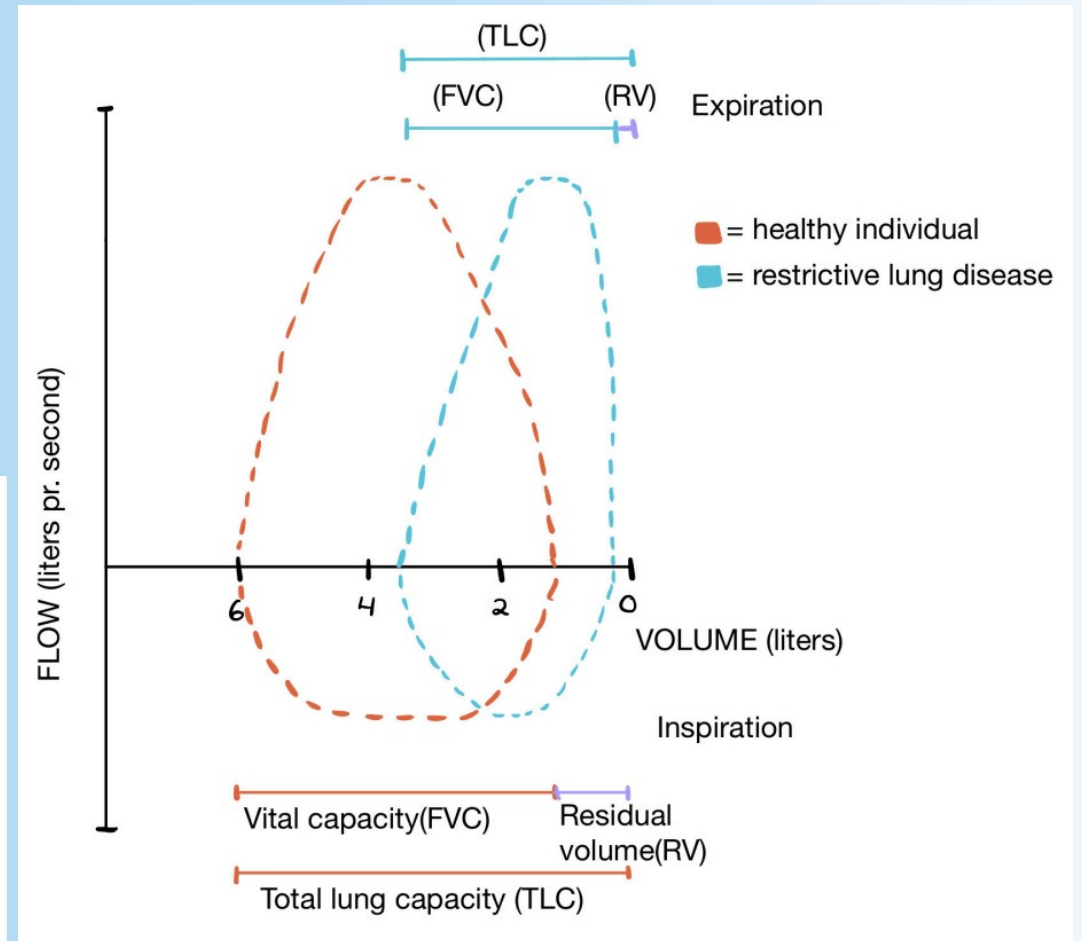
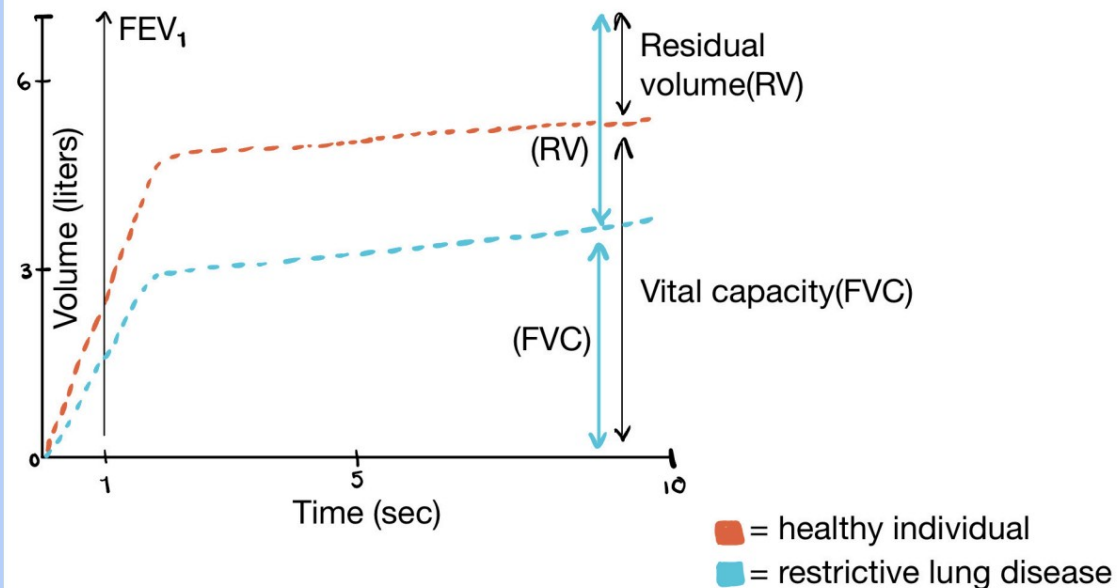
IPF Pathogenesis

1. Damage to alveolar membrane
Type 1-pneumocytes release TGF β -1
2. Type 1-cells stimulate type 2-cells
Type 2-cells proliferate uncontrollably
Type 2-cells stimulate production of fibroblast
3. Uncontrollable fibroblast proliferation
Produce reticular fibers and elastic fibers
4. Thickening of interstitial layer
Too much reticular and elastic fibers → Stiff lung with no compliance



Restrictive spirometry

- $\downarrow$ FEV₁
- $\downarrow\downarrow\downarrow$ FVC
- Normal or $\uparrow$ FEV₁/FVC ratio
- $\downarrow$ TLC



Summary table

	Obstructive Lung diseases		Restrictive Lung diseases	
	Asthma	COPD (Emphysema + Chronic Bronchitis)	Idiopathic Pulmonary Fibrosis	Sarcoidosis
Clinical Presentation	Young child, difficulty breathing (dyspnea), audible wheezing (on expiration) and chest tightness	Dyspnea with history of smoking, barrel shaped chest Productive cough	Older patient with progressive dyspnea and a dry cough	asymptomatic young woman.
Symptoms + Pathogenesis	<u>Dyspnea + chest tightness</u> – Bronchoconstriction from Bronchospasm (due to mast cell degranulation) <u>Wheezing</u> – mucus plugs that obstruct exhalation	<u>Dyspnea</u> – Collapse of bronchioles due to loss of elastic fibers (Emphysema) <u>Productive cough</u> – Mucus gland hyperplasia and hypersecretion	<u>Progressive dyspnea</u> – Excessive fibrosis cause decreased lung compliance	Non-caseating granulomas in different parts of the body may cause symptoms
Diagnosis	Clinical symptoms that are reversible on administration of Bronchodilators (β_2 -agonists)	Emphysema – X-Ray Chronic Bronchitis is clinical – Greater than 3 months of productive cough in 2 consecutive years	Exclusion “honeycomb appearance”	Exclusion
Spirometry	FEV1 = $\downarrow\downarrow$ FVC = $\downarrow$ or normal FEV1/FVC = $\downarrow$	FEV1 = $\downarrow\downarrow$ FVC = $\downarrow$ or normal FEV1/FVC = $\downarrow$	FEV1 = $\downarrow$ or Normal FVC = $\downarrow\downarrow$ FEV1/FVC = Normal or $\uparrow$	FEV1 = $\downarrow$ or Normal FVC = $\downarrow\downarrow$ FEV1/FVC = Normal or $\uparrow$