PULMONARY DISORDERS

(CHAPTERS 17, 18, AND 20)

How is pulmonary disease classified?

The "big one": is it **obstructive** or **restrictive**?

obstructive – blockage/shrinking of the airways

(e.g. asthma, bronchitis, emphysema)

restrictive – decrease in effective lung volume

(e.g. pulmonary fibrosis, pleural effusion, pneumonia)

Other classifications include:

- Time: is it **acute** or **chronic**?
- Infectivity: is it infectious or noninfectious?
- Cause: is it a **lung** problem or a **heart** problem?

Define dyspnea, orthopnea, paroxysmal nocturnal dyspnea, tachypnea, and eupnea.

dus- (bad)
pnoé (breathing)

DYSPNEA

"difficult or labored breathing; shortness of breath"

orthós (straight, upright)
pnoé (breathing)

ORTHOPNEA

"shortness of breath that occurs or worsens when lying down"

paroxusmós (severe fit of disease)

PAROXYSMAL NOCTURNAL DYSPNEA (PND)

"sudden, severe shortness of breath that occurs **at night**, caused by pulmonary disease or sleep apnea"

takhús (swift)
pnoé (breathing)

TACHYPNEA

"an increased **rate** of breathing"

eû- (good)
pnoé (breathing)

EUPNEA

"normal, unimpaired breathing"

For your reference, the opposite of tachypnea is **bradypnea** (abnormally **slow** breathing.)

Describe Kussmaul respiration and restricted breathing.

Kussmaul breathing is characterized by **deep**, **rapid** breaths without breaks in between.

This results in **hyperventilation**, or blowing off more CO₂ than the body produces.

It usually occurs as a compensatory response for **metabolic acidosis**. (Remember the acid/base unit?)

Restricted breathing, on the other hand, is characterized by **rapid** but **shallow** breaths.

Something is causing tidal volume to be decreased, leading to less gas exchange with each breath.

As a result, respiratory rate must increase (**tachypnea**) to maintain proper ventilation.

Describe Cheyne-Stokes breathing. Which what is this pattern of breathing associated?

Cheyne-Stokes breathing occurs when there is some sort of disruption to the normal breathing control of the central nervous system.

Breathing comes in "waves," gradually becoming faster and deeper, then slower and shallower, until it stops entirely (apnea).

Eventually, O₂ saturation drops enough to trigger breathing to resume, and another "wave" occurs.

It can be seen in patients who have suffered damage to the brainstem due to a **stroke**, as well as in **congestive heart failure** (CHF) and in patients who are near death.

What are the characteristics of labored or obstructed breathing?

Airway obstruction actually results in **slower** breathing, as it is more difficult to pass air through the occluded airway and it takes longer to fill the lungs.

There is increased **labor** (effort) of breathing, and tidal volume **increases** (deeper breaths).

What is the consequence of hypoventilation?

When **ventilation** (passage of air into/out of the lungs) decreases, **hypercapnia** (increased CO₂) results.

Remember **carbonic acid**? Decreased CO₂ expulsion leads to a **drop** in serum pH (more acidic.)

What is the consequence of hyperventilation?

When **ventilation** increases, we get the opposite effect —CO₂ levels drop, and **hypocapnia** occurs.

As a result, there is an **increase** in serum pH (more alkaline.)

Define hemoptysis and cyanosis. List some conditions that may cause cyanosis.

hemoptysis – coughing up blood or blood-stained sputum

This can occur in infections such as bronchitis, pneumonia, or TB, as well as lung cancer and trauma.

cyanosis – **bluish** discoloration of the skin, indicative of poor oxygenation

This can occur in anemia, heart problems, or respiratory problems (as well as some other causes such as cold temperature.)

What is (nail) clubbing?

Nail clubbing is a clinical sign that is indicative of long-term, unresolved **hypoxia**.

It presents as a deformation of the **distal ends** of the fingers in which the ends of the fingers **bulge out** rather than coming to a narrow tip.

Clubbing is almost always indicative of **chronic** cardiovascular or pulmonary disease.

How can sputum be clinically relevant?

A cough can be either **productive** (coughing up sputum) or **non-productive** (dry, hacking cough.)

Qualities of the sputum produced (amount, color, consistency, presence of blood, etc.) can be clinically significant.

For example, **frothy** or **foamy** sputum suggests pulmonary edema.

Rust-colored sputum suggests pneumonia or tuberculosis.

Sputum can also be **cultured** to determine the underlying pathogen and direct antibiotic therapy.

List some causes of hypercapnia.

Typically, hypercapnia is caused by pathologically **decreased** breathing. This can be linked to the side effects of CNS depressant drugs (such as opioids,) or to direct damage or alteration to the brainstem.

List some causes of hypoxia.

Again, hypoxia can be caused by any source of decreased breathing; however, it can also be caused by problems such as impaired **delivery** to the tissues.

- Decreased availability of oxygen in the air (e.g. mountain climbing)
- Mismatched ventilation-to-perfusion (V/Q) ratio amount of air entering lungs vs. blood flow to lungs
- Right-to-left shunting deformity allowing blood flow directly from right heart to left heart, bypassing lungs

What are the clinical findings in acute respiratory failure?

Respiratory failure results in alterations to the **arterial blood gas** (ABG) levels:

- P_aCO₂ > 50 mmHg (**hypercapnia**)
- $P_aO_2 < 50 \text{ mmHg (hypoxemia)}$
- Serum pH < 7.25 (acidosis)

What is the most common cause of pulmonary edema? What are its clinical manifestations?

The most common cause of pulmonary edema is **left heart failure** (LHF.)

Why left? Left side of the heart pumps blood **from** the lungs **into** the systemic circulation.

If the left ventricle isn't working efficiently, blood backs up **into the lungs** and the **increased CHP** causes edema.

Why is aspiration so serious? When do we see an increased risk of aspiration?

Aspiration of gastric contents has the potential for serious pulmonary damage due to the **acidity** of stomach contents.

It can also cause **infection** if microbiota from the stomach are aspirated into the lungs.

Increased risk is associated with decreased LOC (e.g. after anesthesia, neurological damage, etc.)

There is also a risk of aspiration associated with **NG** or **PEG tube** feeding, as well as with **tracheotomies**.

These risks can be mitigated by following proper procedures: verifying tube placement, checking residuals, and ensuring proper cuff pressure on trach tubes.

Describe the three main causes of atelectasis.

atelés (incomplete)

éktasis (extension)

ATELECTASIS

"collapse of all or part of a lung, leading to reduced lung capacity"

compression – fluid (pleural effusion) or air (pneumothorax) surrounding the lungs, or trauma

absorption – caused by **too much oxygen** in the air (not enough non-O₂ gas left behind)

post-surgical – anesthesia causes CNS changes and decreased surfactant production, leading to collapse

Describe bronchiectasis. How is it different from bronchiolitis?

brónkhos (bronchi)

éktasis (extension)

BRONCHIECTASIS

"a chronic, abnormal **enlargement** of the bronchi and bronchioles"

Bronchiectasis can occur as a sequela of infection (PNA, TB, etc.) or as a symptom of the genetic disorder **cystic fibrosis**.

The airways become inflamed and enlarged, but fill with mucus, leading to **dyspnea** and increased risk of subsequent **infection**.

Bronchiolitis, on the other hand, is an inflammatory narrowing of the airways that usually occurs in infants infected with respiratory syncytial virus (RSV.)

The narrowed airways pass air through less easily, also resulting in **dyspnea**. Severe cases may feature **intercostal retractions**.

Describe the types of pneumothorax.

Open pneumothorax results from an **open wound** in the chest wall from penetrating trauma.

This interferes with the ability to maintain pressure in the lungs, because air can leak in and out of the wound.

Typically, an occlusive dressing is used to prevent air ingress through the wound, helping to restore normal breathing.

Tension pneumothorax results from communication between the lungs and the **pleural space**, e.g. in blunt trauma.

The air in the pleural cavity **pushes** on the lungs from the outside, leading to **atelectasis**.

Treatment involves the placement of a **chest tube** to allow the extra air to escape.

Spontaneous (non-traumatic) pneumothorax occurs in the absence of any trauma or other causative lung disease.

It is believed to be related to the formation of sacs of air called **blebs** in the lungs, which can spontaneously rupture and leak air into the pleural space.

Describe the three types of pleural effusion. What causes each of these types?

Pleural effusion refers to fluid buildup (effusion) that occurs in the **pleural cavity**, outside the lungs.

It can be categorized based on the **type** of fluid that causes it.

Transudative pleural effusion occurs when **normal fluid** (ECF) leaks out of intact blood vessels (similar to **edema**.)

Exudative pleural effusion occurs when **capillary permeability** increases, allowing proteins to leak out with the fluid.

Hemothorax occurs when trauma causes **blood** to pool up in the pleural space.

Describe empyema and pleurisy.

Pleural **empyema** (not "emphysema") or **pyothorax** refers to an infection within the pleural space outside the lungs.

Pleurisy or **pleuritis** refers to an inflammation of the pleura (lining of the lung space.)

What is compliance? Why would pulmonary fibrosis affect compliance?

Compliance refers to the "stretchiness" of the lungs and their ability to expand and contract normally.

The concept of compliance is strongly linked to **restrictive** lung diseases, such as pulmonary fibrosis.

Scar tissue makes the lungs tougher and less compliant, preventing them from expanding fully and hindering respiration.

Describe pneumoconiosis. Which occupations would be at risk for this disorder?

Pneumoconiosis refers to a range of diseases caused by the inhalation of **fine particulate** materials.

As such, its etiology is **environmental**, usually caused by occupational exposure to particulates without proper PPE.

Types of pneumoconiosis are characterized by the etiologic agent:

- Silicosis silica (ceramics, stone and concrete grinding)
- Coal worker's pneumoconiosis (CWP) or "black lung" – coal dust
- Asbestosis asbestos (old fireproofing/insulation material)

Describe acute respiratory distress syndrome (ARDS.) Why is COVID-19 related?

ARDS is a **severe medical emergency** and often requires mechanical ventilation in an ICU.

The mortality rate is estimated at approximately **50%**.

Pulmonary edema in ARDS results from increased **permeability** of the alveolar membrane, causing fluid to leak into the alveoli from the capillaries.

Diffuse alveolar injury also results in decreased production of **pulmonary surfactant**, which is required to maintain normal lung compliance and prevent collapse.

Alveolar damage in severe cases of COVID-19 can result in the development of ARDS, which is why we see so many severe COVID cases requiring ICU beds and ventilators.

Describe the clinical manifestations of ARDS.

Major symptoms are **dyspnea**/SOB and **dry cough** (unable to clear fluid from lungs.)

May see **tachypnea** as a compensatory response, and lack of surfactant can cause **atelectasis**.

Impaired gas exchange will also result in decreased oxygen saturation/hypoxemia.

List some common post-operative problems. How can they be prevented?

One very common problem that occurs after surgery is **atelectasis**.

You've probably learned about how we correct this in Concepts: **incentive spirometry**.

Regular use of an incentive spirometer can help "pop open" the alveoli and restore normal tidal volume.

Disruptions to respiration caused by surgery can also lead to more complex problems such as **pulmonary edema** and **pneumonia**.

Impaired circulation after surgery can also cause blood clots, which can result in **pulmonary embolism**.

This risk can be decreased by regular **position changes**, and **ambulating** patients as soon as possible after surgery.

Describe asthma and COPD.

ásthma (labored breathing)

ASTHMA

"a **chronic inflammatory disease** of the airways of the lungs"

Asthma is a **multifactorial** disease, involving both genetic predisposition and environmental factors.

There is a strong **genetic component**; those with at least one parent with asthma are **3-6 times** more likely to develop asthma themselves.

Asthma consists of three basic pathophysiologic processes, all of which work together to inhibit airflow:

inflammation of the airways

hypersecretion of mucus

bronchoconstriction (or bronchospasm)

Inflammation of the airways can be acute, but often progresses to a chronic inflammatory state where the airways are partially constricted even at baseline.

This long-term inflammation can lead to **airway remodeling**, where structural changes such as fibrosis permanently alter the function of the airways.

The inflammatory process also leads to **hypersecretion** of mucus in the airways, with this excess fluid further contributing to respiratory obstruction.

These inflamed airways become **hypersensitive** to allergens, resulting in **bronchoconstriction** in which the smooth muscle around the airway constricts, further reducing the lumen and seriously obstructing airflow.

Chronic obstructive pulmonary disease (COPD) is an umbrella term encompassing two distinct diseases, which often overlap:

chronic bronchitis and emphysema

(We'll talk about each of these individually later.)

Compare type 1 and type 2 asthma. Are these extrinsic or intrinsic?

Type 1 asthma is the "normal" type of asthma that generally develops in childhood and may resolve itself over time with maturity.

Type 2 is also caused by environmental irritants, but usually develops in adults after repeated exposure.

These are both forms of **extrinsic** asthma, meaning that the trigger is **external** to the body (some form of allergen.)

What is intrinsic asthma? What is status asthmaticus?

Intrinsic (non-allergic) asthma covers any type of asthma that **lacks** an external trigger, and may be brought on by stress, exercise, cold/dry air, etc.

Status asthmaticus refers to an acute severe asthma exacerbation (asthma attack) that **does not respond** to initial bronchodilator treatment.

This is a **medical emergency** that is usually treated with alternate/higher-dose bronchodilators, corticosteroids, or even mechanical ventilation if necessary.

Describe chronic bronchitis, and list its clinical manifestations.

Chronic bronchitis, a subtype of COPD, is characterized by hypersecretion of mucus, which blocks (obstructs) airflow into the lungs and increases the risk of pulmonary insomnia such as pneumonia.

Is emphyesma classified as a restrictive or obstructive disorder?

Emphysema, as a form of COPD, is an **obstructive** pulmonary disorder.

What does alpha-antitrypsin have to do with emphysema?

Smoking increases the levels of **protease** in the lungs and decreases the action of **anti-proteases** (alphaantitrypsin,) causing the breakdown of the alveolar wall.

Proteases **break down proteins** and normally play an important role in cell regeneration and repair, but in emphysema, overactive proteases cause progressive damage.

What type of damage occurs to the lungs in emphysema?

Emphysema is charcterized by damage to the alveolar walls, resulting in the alveoli becoming **less elastic**, which reduces the "pushing force" that naturally expels CO₂.

This loss of elasticity results in the **collapse** of the airways, **obstructing** the flow of air within the lungs.

Major clinical signs include:

"barrel chest" – torso becomes rounder in crosssection, often nearly as deep in front-to-back dimension as it is wide

tripod positioning – patient naturally leans forward on outstretched arms to increase lung expansion

What do the terms "blue bloater" and "pink puffer" refer to? Which is associated with emphysema?

These terms are mnemonics for remembering the clinical presentations associated with COPD.

"Blue bloater" refers to the signs and symptoms of chronic bronchitis.

The airway congestion seen in chronic bronchitis leads to **poor oxygenation**, which can present as **cyanosis** (hence "blue.")

The long term effects of chronic bronchitis also lead to heart problems (**cor pulmonale**) which results in **edema** and **jugular venous distension** (JVD) (hence "bloater.")

"Pink puffer" refers to the signs and symptoms of emphysema.

Because the dyspnea associated with emphysema is constant, the body **adapts**. As such, COPD sufferers with only emphysema are **less likely** to be cyanotic (hence "pink.")

These adaptations include baseline **tachypnea** and **pursed-lip breathing**, which helps expel the excess CO₂ and prevent hypercapnia (hence "puffer.")

What are the three main types of bacterial pneumonia? How is pneumonia spread?

Community-acquired pneumonia (CAP) – usually caused by **Gram-positive** bacteria, e.g. Streptococcus

Hospital-acquired pneumonia (HAP) – usually caused by **Gram-negative** bacteria, e.g. Pseudomonas

Legionnaires' disease – rare but severe form of pneumonia caused by Legionella bacteria

As a **respiratory infection**, pneumonia spreads primarily through respiratory **droplets** expelled through coughing, sneezing, talking, etc.

Describe the stages of pneumococcal pneumonia.

congestion

 $\mathbf{\Psi}$

red hepatization

 \downarrow

gray hepatization

 $\overline{\Psi}$

resolution

Congestion is the first stage, in which the congested vasculature of the lungs causes fluid to build up in the alveoli.

The first symptoms, such as coughing, appear, and the infection begins to spread throughout the lungs.

Red hepatization follows 2–3 days later, in which lungs swell and become dense and firm ("liver-like.")

Congestion persists, and blood leaks into alveoli causing pink sputum. Dyspnea worsens.

Gray hepatization follows in another 2–3 days when bloodflow to the lungs slows and RBCs in the lungs begin to break down.

The lungs begin to dry out and fibrin continues to build up in the alveoli.

Resolution is the final stage, in which enzymes work to break down all of the debris that has been exuded into the lungs.

This debris is then absorbed by macrophages and eventually coughed up in the sputum, after which the symptoms fade.

What is the causative agent in tuberculosis? How is it spread?

Tuberculosis is a **bacterial** lung infection caused by the bacterium **Mycobacterium tuberculosis**.

It is spread by (aerosolized) airborne droplets, similar to bacterial pneumonia but **smaller** (N95 recommended.)

What is a granulomatous lesion?

A **granuloma** is a small nodule consisting primarily of macrophages that can develop during the inflammatory response to infection.

Macrophages pile on and surround the infected area in an attempt to "quarantine" the foreign material.

In TB, the center of these granulomas undergo caseous necrosis and are then referred to as tubercles.

Describe the evaluation score for tuberculosis.

- 0 patient has not been exposed to TB at all
 - 1 patient exposed but uninfected (- PPD)
- 2 patient infected but no symptoms (+ PPD)
 - 3 clinically active disease **present** (+ PPD)
- 4 history of TB but no sign of current disease (+ PPD)
 - 5 suspicion of TB exposure but diagnosis pending

Describe pulmonary vascular disease.

Pulmonary vascular disease refers to disorders of the **blood vessels** of the lungs, which ultimately cause an increase in pulmonary blood pressure.

The resulting decrease in bloodflow through the heart (**perfusion**) decreases gas exchange and results in increased workload of the **right ventricle**.

What is cor pulmonale?

Cor pulmonale (pulmonary heart disease) is a common cause of right heart failure (RHF,) in which changes in the pulmonary circulation (stenosis, pulmonary hypertension) cause the right ventricle to have difficulty pumping blood through the lungs.

Remember that this is a **heart problem** that is caused by the circulation **in the lungs**!

How is pulmonary embolism treated? What are the clinical manifestations of massive occlusion?

Treatments depend on the severity of the blockage. Typically, **anticoagulants** will be involved to prevent the clot from getting worse.

In some patients, **thrombolytics** (clot-busters) such as tPA will be used to break up the clot to restore blood flow.

In the most severe cases ("massive" PE,) **obstructive shock** can occur. Systemic BP plummets and pulmonary BP rises dramatically as blood is unable to pass through the lungs.

This results in **tachypnea** and **tachycardia** as the body struggles to maintain oxygenation of tissues.

These patients will typically need high flow-rate supplemental O₂ to maintain oxygenation status until the clot can be broken up with tPA or surgical intervention.

What is pulmonary hypertension? What are the consequences of long-term pulmonary hypertension?

Normal pulmonary arterial pressure (PAP) is **15–25** mmHg (systolic) over **8–15** mmHg (diastolic.)

Note that this is **much** lower than the systemic blood pressure (e.g. 120/80!)

Pulmonary hypertension is defined as a systolic PAP of **over 25 mmHg**.

Chronic pulmonary hypertension can result in:

- Right ventricular hypertrophy (RVH)
- Cor pulmonale/right heart failure (RHF)
- Cardiac arrythmias, pulmonary embolism

Describe the tyes of lung cancer.

Small cell carcinoma — most aggressive form, high mortality rate but very rare

Non-small cell lung cancers (NSCLC):

- Adenocarcinoma Most common type (40%); peripheral, grows slowly
- Squamous cell carcinoma Second most common (30%); bronchi/hilar region
- Large cell carcinoma any NSCLC that isn't one of the other types