



Respiratory System Disorders

Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Acute Respiratory Distress Syndrome (ARDS)
- Severe Acute Respiratory Syndrome (SARS)
- Asthma
- Bronchiectasis
- Chronic Bronchitis
- Cystic Fibrosis
- Emphysema
- Histoplasmosis
- Influenza
- Legionnaires' Disease
- Lung Cancer

- Mesothelioma
- Pleural Effusion
- Pneumonia
- Pneumothorax
- Pulmonary Embolism
- Sarcoidosis
- Tuberculosis (TB)

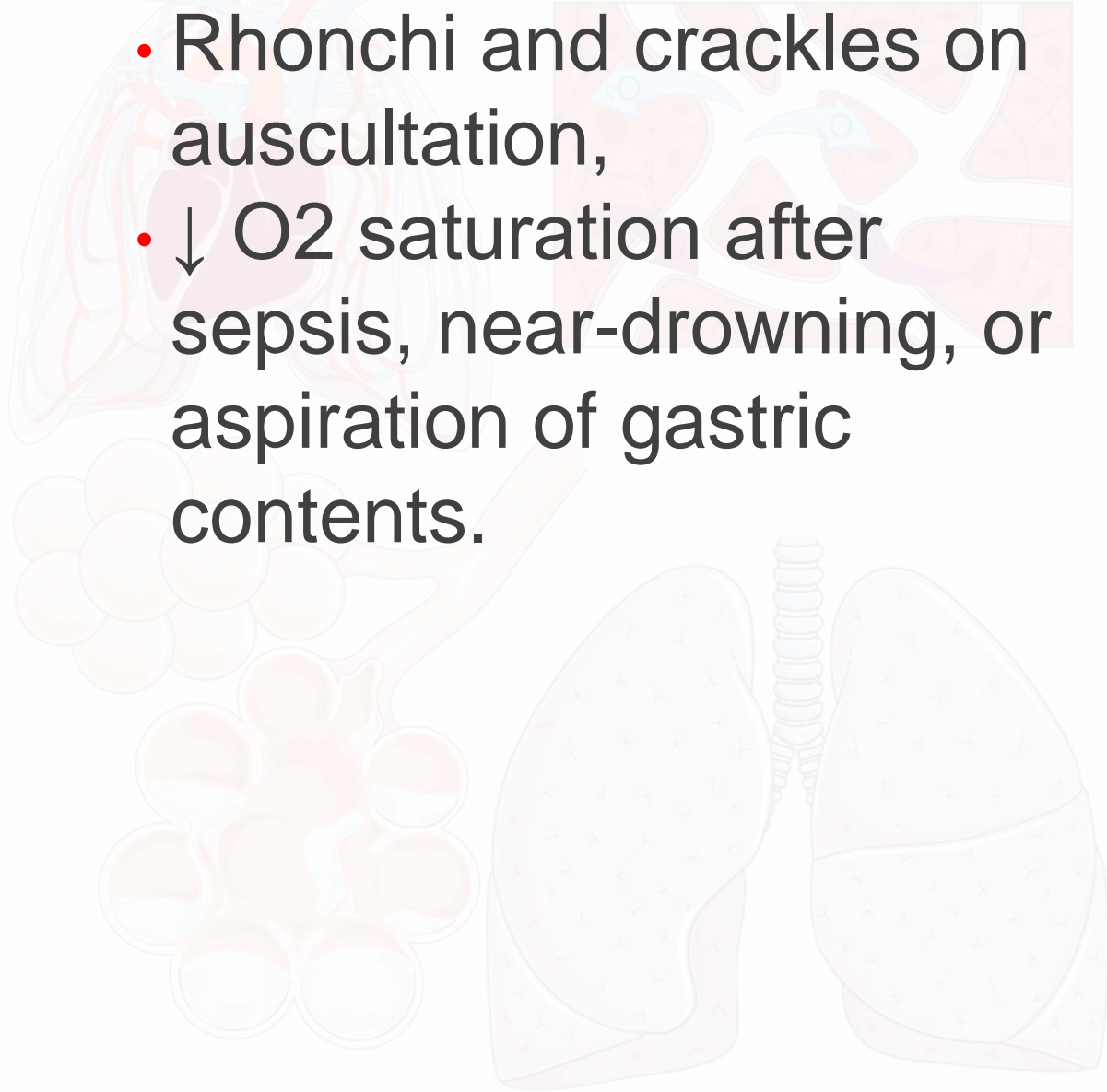
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Acute Respiratory Distress Syndrome (ARDS)

- Rhonchi and crackles on auscultation,
- ↓ O₂ saturation after sepsis, near-drowning, or aspiration of gastric contents.

Pathophysiology

- Destruction of alveolar walls and capillary beds caused by stimulation of immune mediators that ↓ O₂ exchange by fibrosis and edema.
- Immune mediators are also stimulated by traumatic events.



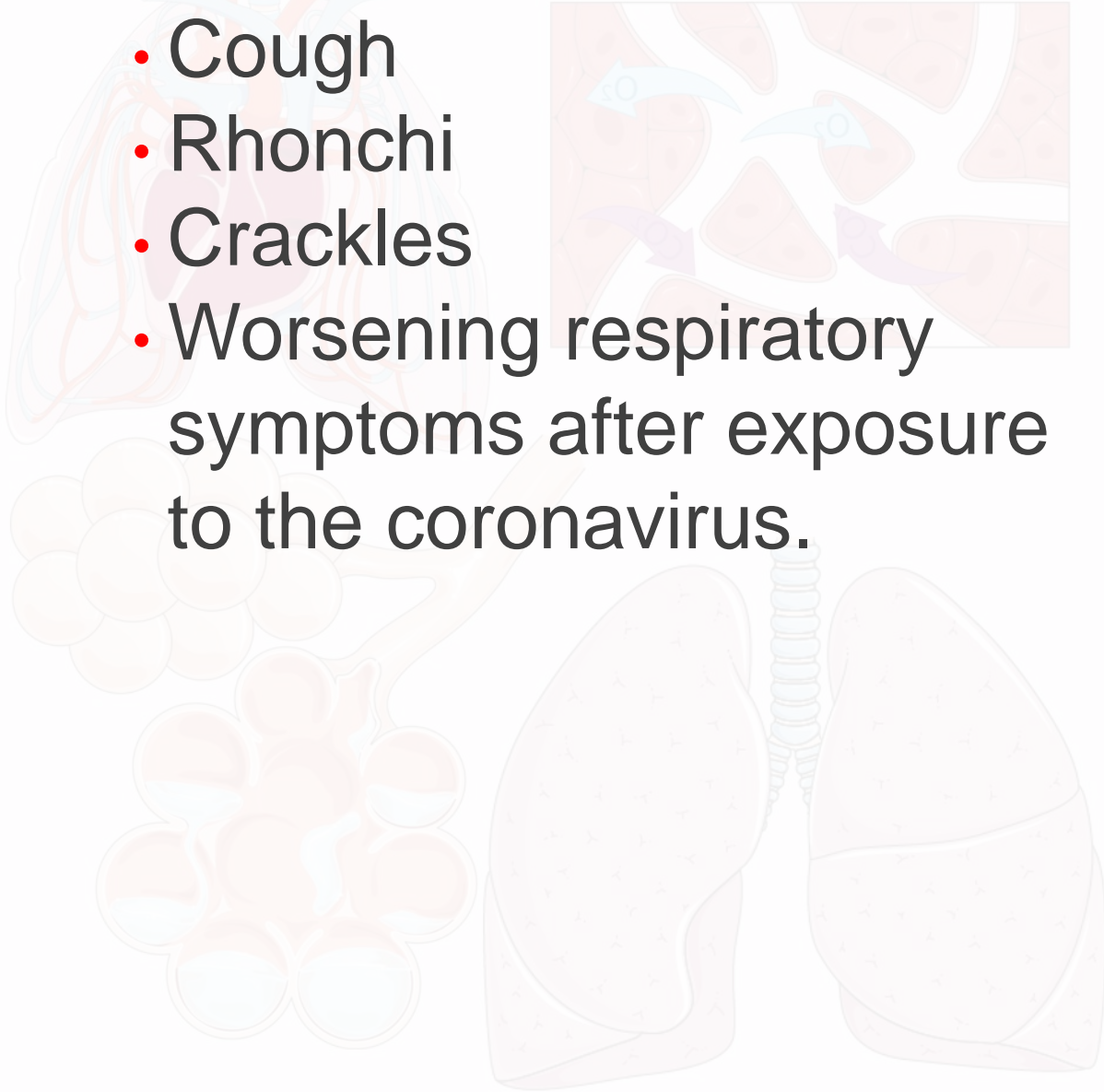
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Severe Acute Respiratory Syndrome (SARS)

- Cough
- Rhonchi
- Crackles
- Worsening respiratory symptoms after exposure to the coronavirus.

Pathophysiology

- Exposure to the coronavirus by droplet inhalation or contact. The SARS virus can live 6 hours on the hands.
- After contact with the SARS coronavirus, immune mediators cause inflammation, edema, and pneumonia by blocking gas exchange and resulting in filling of the alveoli with fluid.



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Asthma

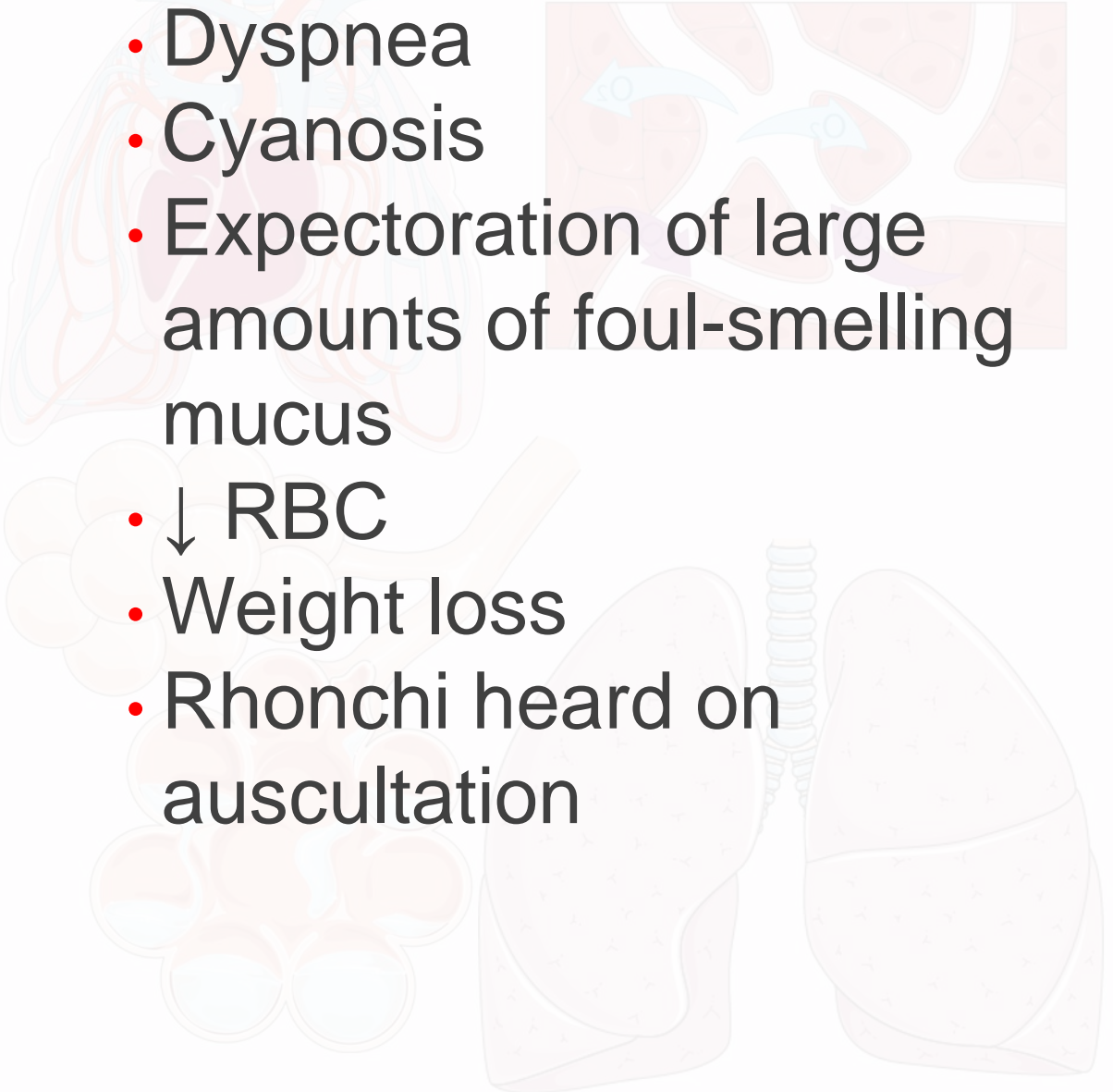
- Expiratory wheeze on auscultation
- Rapid onset
- Difficult expiration
- Nonproductive cough
- “chest is tight,”
- ↓ O₂ saturation.

Pathophysiology

- Reactive inflammatory disorder associated with exposure to allergens, viral infection, pollution, smoking, or extremes of temperature.
- Chronic inflammation results in fibrosis and narrowing of bronchiole passageways, leading to air trapping, bronchospasm, and increased dead air space.
- Mast cells, active in inflammation, release histamine, prostaglandins, leukotrienes, and bradykinin.

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Bronchiectasis

- Dyspnea
 - Cyanosis
 - Expectoration of large amounts of foul-smelling mucus
 - ↓ RBC
 - Weight loss
 - Rhonchi heard on auscultation
- 

Pathophysiology

- Chronic dilation of the bronchi and bronchioles due to inflammation.
- Inflammatory process destroys elasticity of smooth muscle in the airways.
- Chronic infections occur in dilated areas that retain mucus and obstruct airways.
- Can be localized or diffuse; associated with childhood diseases (e.g., measles), influenza, or tuberculosis.

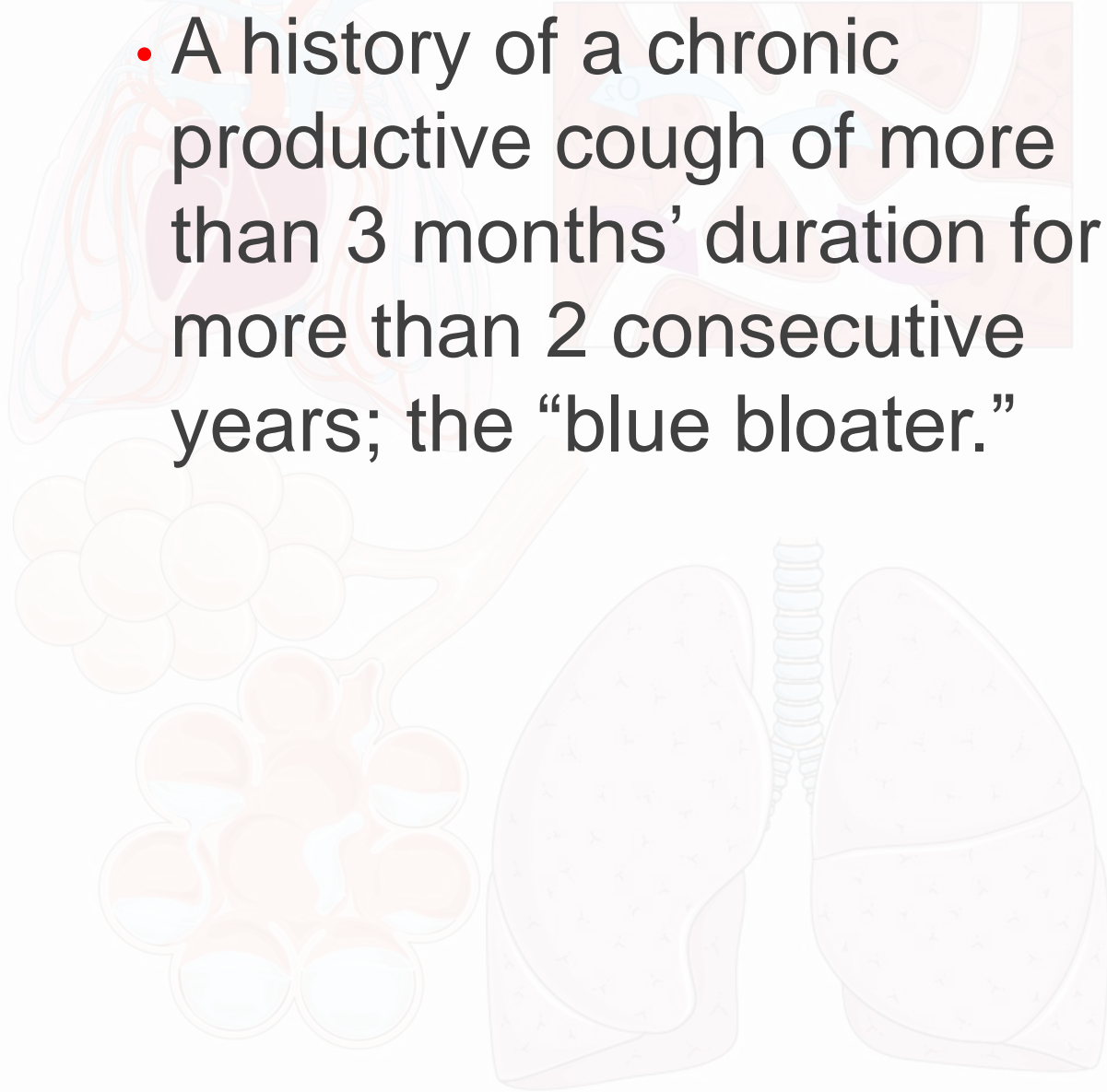
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Chronic Bronchitis

- A history of a chronic productive cough of more than 3 months' duration for more than 2 consecutive years; the “blue bloater.”

Pathophysiology

- Chronic Inflammation by IL-8 and cytokines of the mucous membrane lining the bronchi and bronchioles. Excess mucus is produced, and the mucociliary pump ceases to function properly, causing chronic congestion. Common in smokers.
- Chronic bronchitis results in a fibrotic, noncompliant airway and pulmonary hypertension.



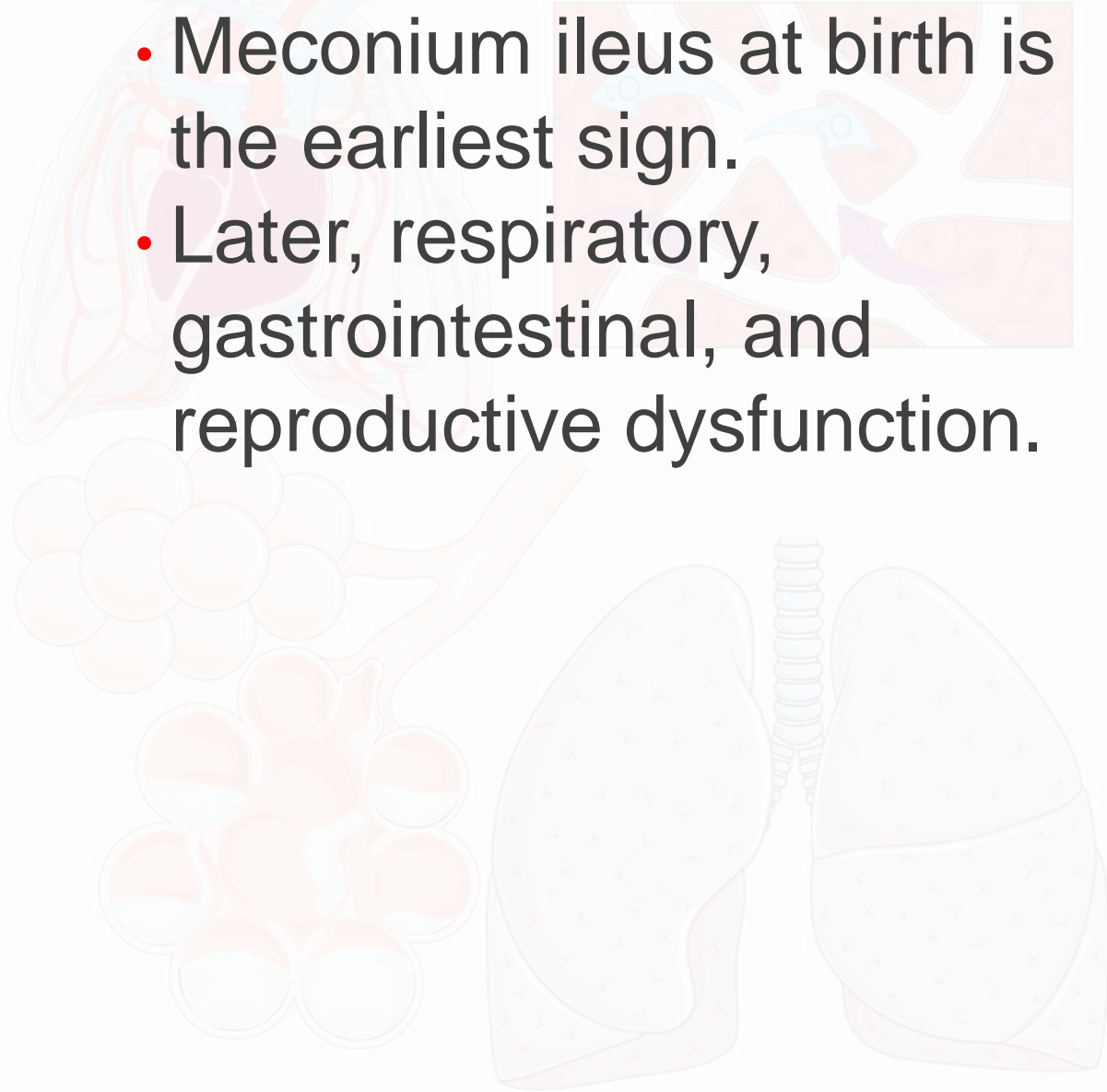
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Cystic Fibrosis

- Meconium ileus at birth is the earliest sign.
- Later, respiratory, gastrointestinal, and reproductive dysfunction.

Pathophysiology

- Autosomal recessive disorder that affects chromosome 7, which normally produces a protein CFTR that affects movement of Na^+ and Cl^- ions.
- All secretions of exocrine glands of the respiratory, gastrointestinal, and reproductive tracts become thick and obstruct normal flow.
- Sweat glands do not reabsorb sodium, so salt depletion in sweat can occur.



Emphysema

- Barrel or pigeon chest,
- Dyspnea,
- The “pink puffer,”
- \uparrow PaCo₂,
- Chronic respiratory acidosis
- Hypoxic respiratory drive.

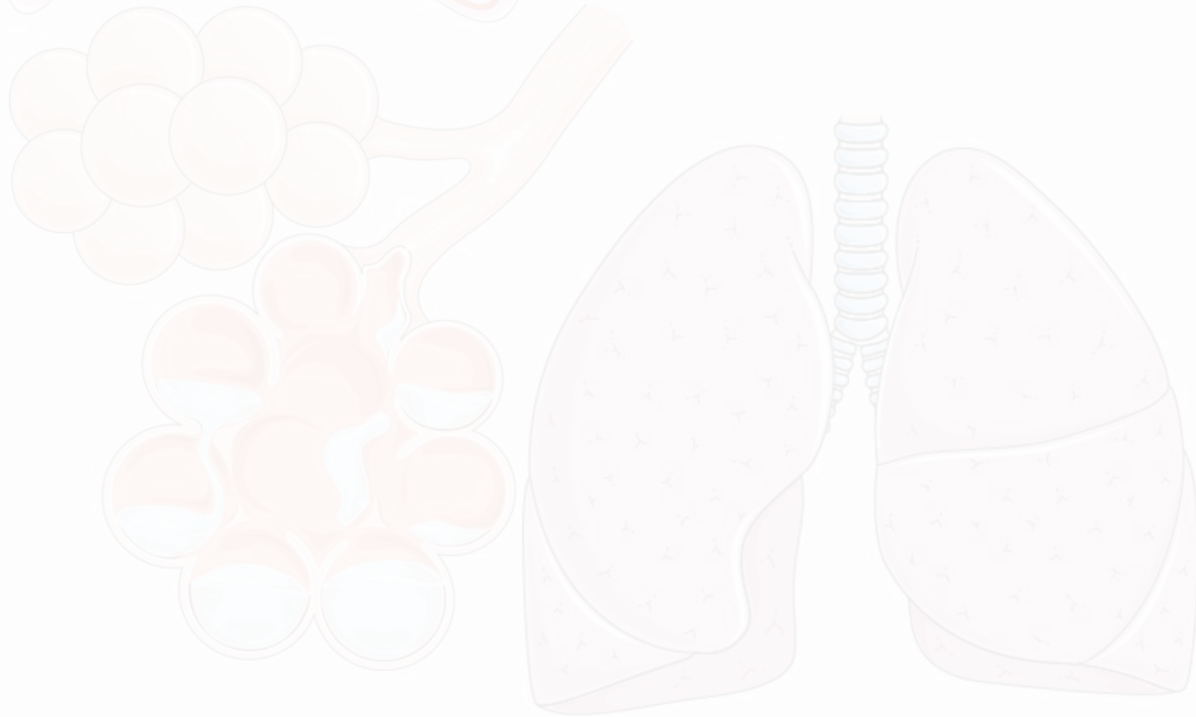
Pathophysiology

- A chronic disorder in which the alveolar structures distend, lose elasticity, rupture, or coalesce, resulting in damage and destruction to the pulmonary capillary bed, air trapping, and increased dead air space.
- Cigarette smoking and an inherited deficiency of α 1-antitrypsin are cocontributors to the disease. \downarrow α 1-antitrypsin results in elastase secreted from neutrophils that can digest elastin and other alveolar structures.

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Histoplasmosis

- Cough
- Fever



Pathophysiology

- Systemic fungal disease caused by dimorphic fungus *Histoplasma capsulatum*.
- Organism grows in soil enriched with bird droppings. Fungal spores form that are then inhaled.
- Once at body temperature, fungal spores change to the yeast form in the alveoli. The yeast is then absorbed through the regional lymphatics and into the bloodstream. Cellular immunity occurs 2–3 weeks after infection.

Influenza

- Fever
- Myalgia
- Respiratory and gastrointestinal symptoms.

Pathophysiology

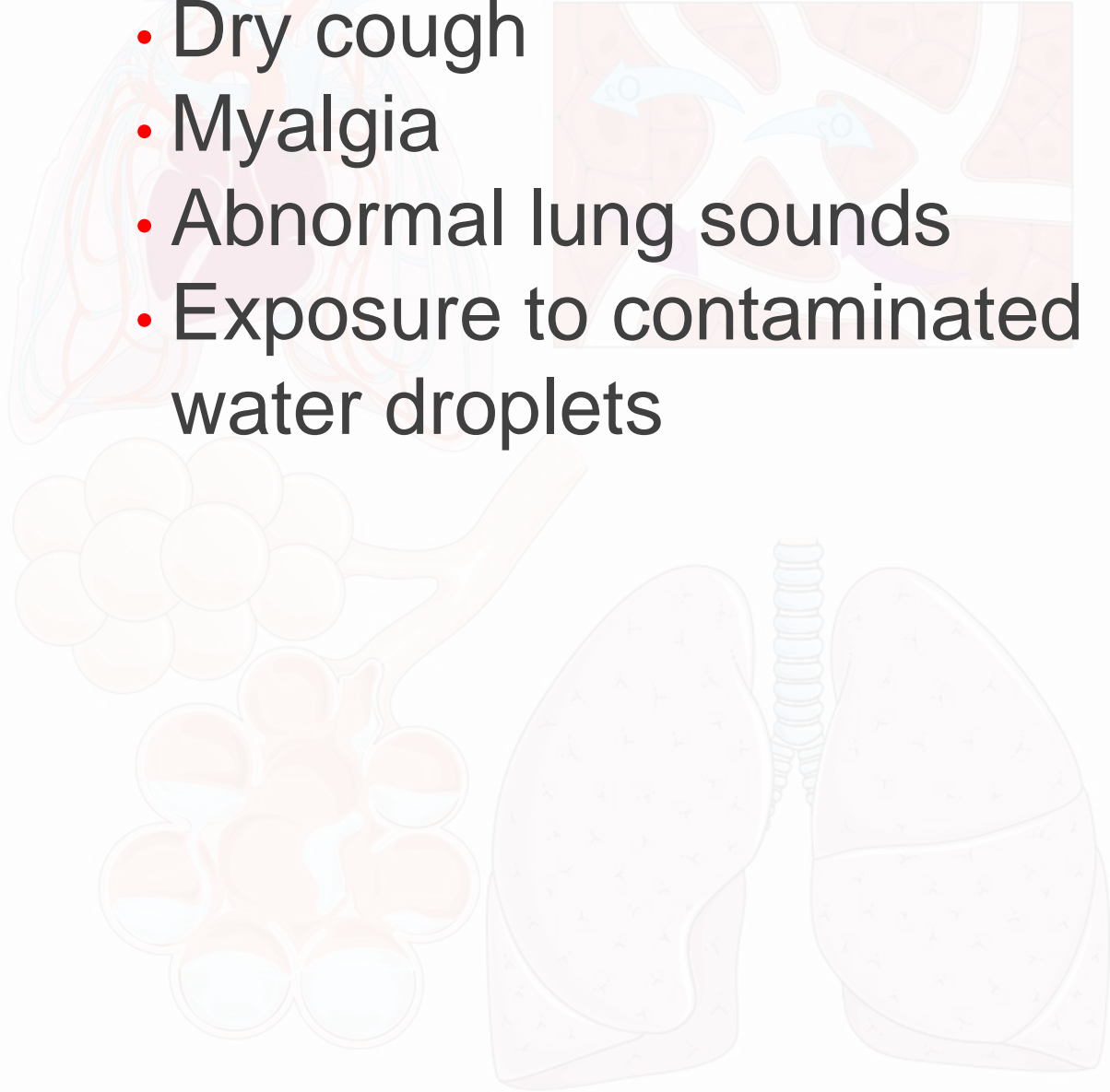
- A viral syndrome that is spread as aerosolized particles (airborne) and causes systemic inflammatory reactions of myalgia, fever, respiratory symptoms, and gastrointestinal symptoms.
- Strains are varied, and influenza vaccine is developed anew each season on the basis of identified strains. H1N1 influenza (swine flu) and H5N1 (bird flu) are relatively new strains causing concern.
- Influenza can easily become pandemic without vaccination.
- The very young, very old, and those with chronic disease are at most risk for death from complications.

10 Legionnaires' Disease

- Dry cough
- Myalgia
- Abnormal lung sounds
- Exposure to contaminated water droplets

Pathophysiology

- *Legionella* bacteria is inhaled from contaminated water supplies (e.g., from air conditioner vents, spas, respiratory equipment), causing pneumonialike symptoms. Thrives at temperatures from 90°–105° F.
- Headache, myalgia, fever, diarrhea. Incubation period of 2–10 days.
- Results in Legionnaire's disease or a lesser influenza-like illness known as Pontiac fever.



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Lung Cancer

- Persistent cough
- Weight loss
- History of or current cigarette smoking.

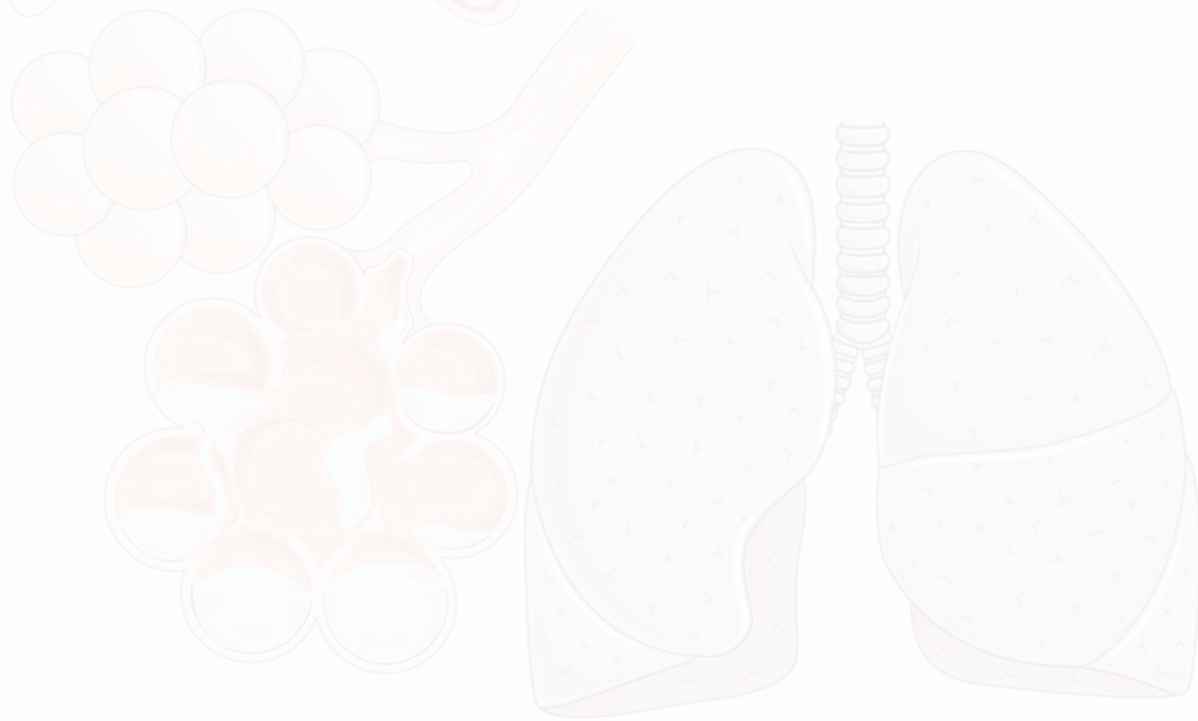
Pathophysiology

- Exposure to chronic irritants or carcinogens cause cell mutation, resulting in oncogene stimulation and loss of genetic material from chromosome 3.
- Cells in early differentiation that mutate are more aggressive than more mature cells.
- Cancers are evaluated using the TNM method.
- Cancer cells divide more rapidly and are more metabolic than normal body cells. Secreting cancers cause damage to the body by hypersecretion.
- Cancer in the lung may be the primary site or a metastatic secondary site from a distant body area.

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Mesothelioma

- Cough
- SOB
- History of asbestos exposure



Pathophysiology

- Mesothelia is a single layer of flat cells that line the pleural, peritoneal, and pericardial cavities. Exposure to asbestos through inhalation causes infiltration by the short asbestos fibers into these cells. Peritoneal infiltration is thought to occur by coughing up and swallowing the asbestos fibers.
- Cells mutate causing changing DNA, and activating oncogenes.

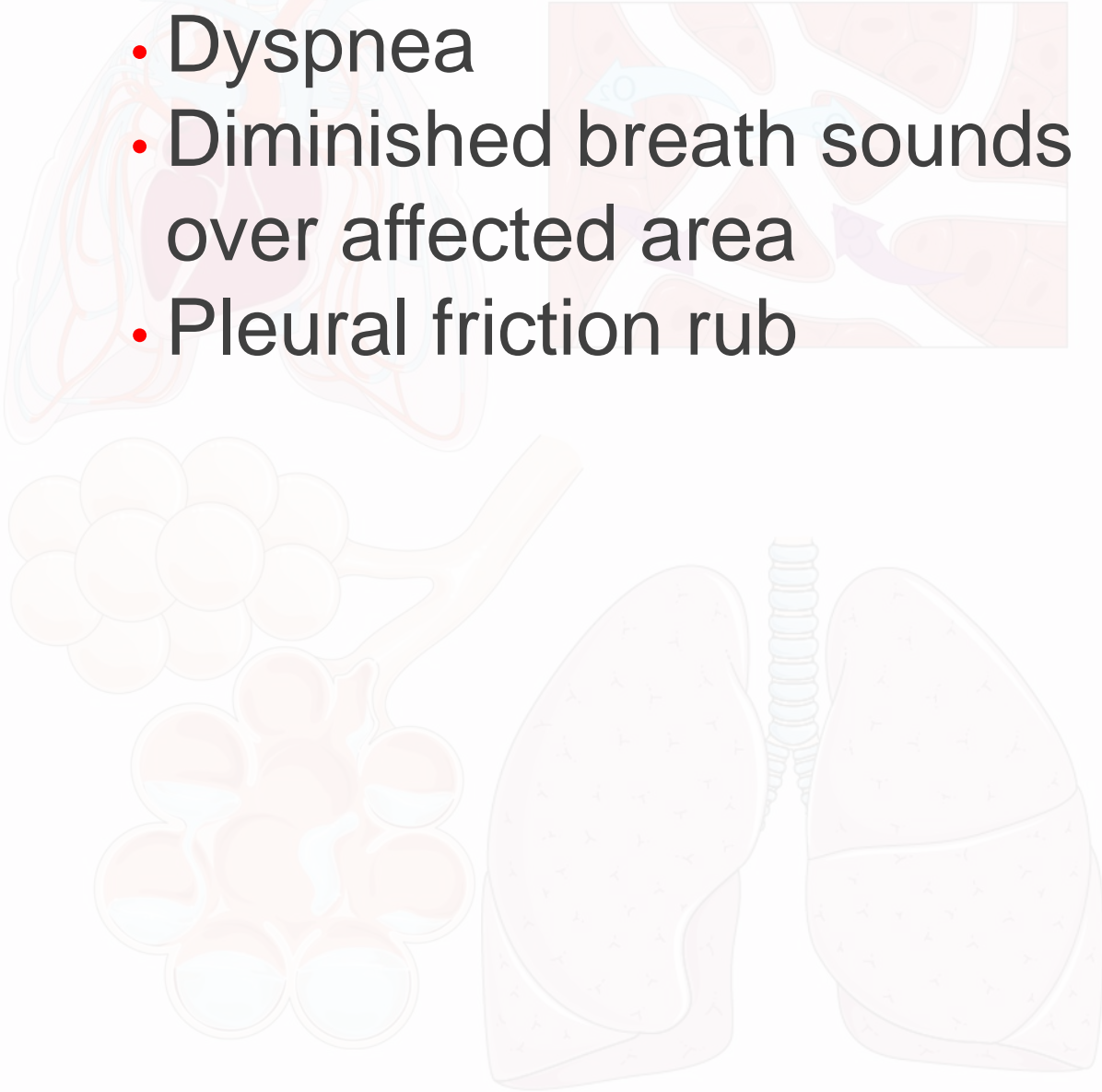
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Pleural Effusion

- Dyspnea
- Diminished breath sounds over affected area
- Pleural friction rub

Pathophysiology

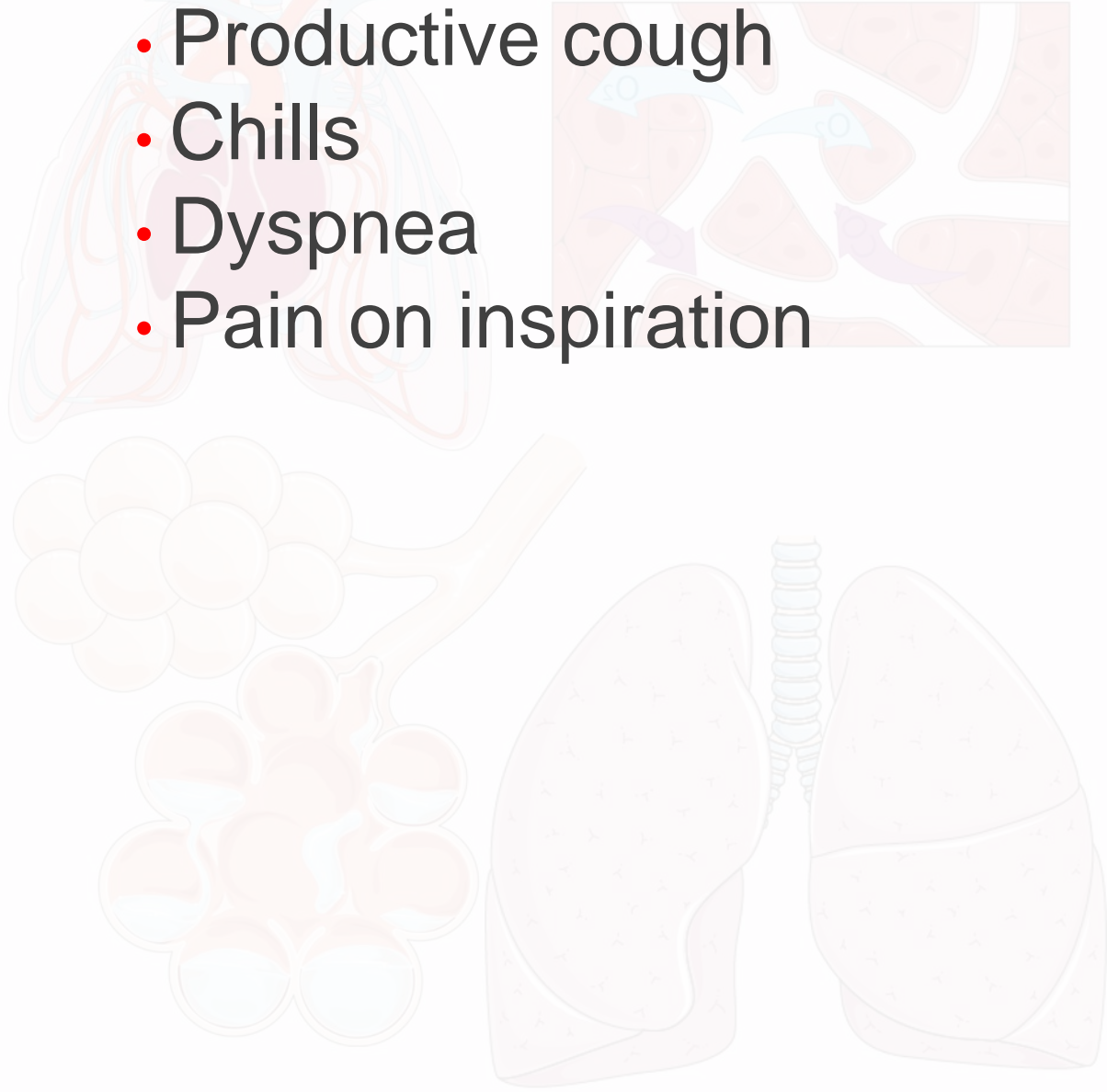
- The pleural space is the visceral and parietal lining of the outer lungs. Negative pressure or a vacuum exists in this space.
- If the lining becomes damaged or diseased, or experiences oncotic pressure changes (lung cancer, pulmonary tuberculosis [TB], lung abscess, congestive heart failure, ascites, chronic renal disease, chest trauma), the space loses its negative pressure and expands into a space that presses on the lung in that cavity.



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Pneumonia

- Productive cough
- Chills
- Dyspnea
- Pain on inspiration



Pathophysiology

- Acute inflammation of lung tissue by inhalation of droplets containing viral particles, bacteria, fungi, parasites, or irritating chemicals.
- Inflammatory mediators in lung tissue cause edema and filling of alveoli with serous fluid and mucus.

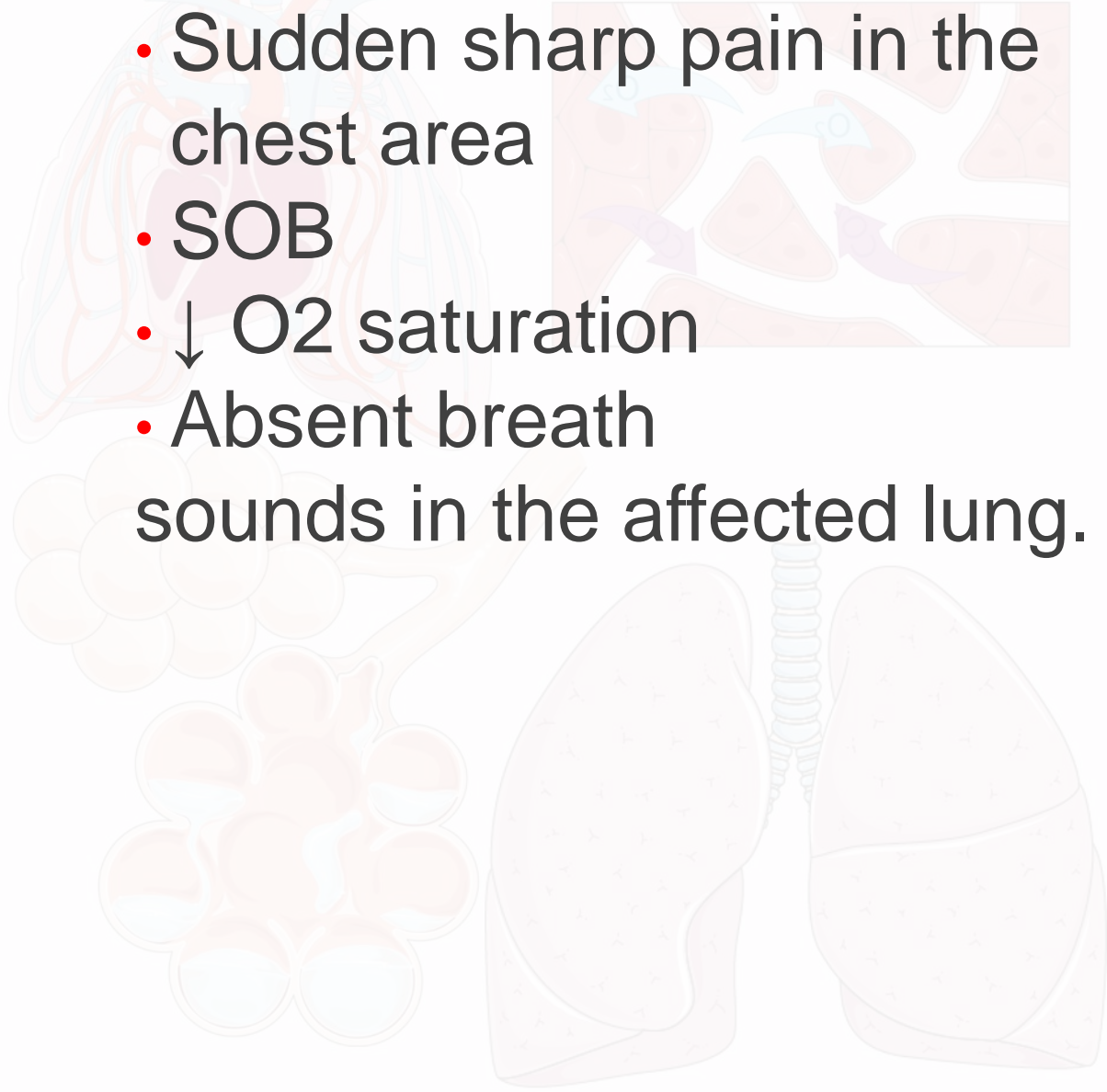
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Pneumothorax

- Sudden sharp pain in the chest area
- SOB
- ↓ O2 saturation
- Absent breath sounds in the affected lung.

Pathophysiology

- Potential space created by the visceral and parietal pleura creates negative pressure in that area. Once breached by trauma or a pathologic event, negativity is lost and the potential space becomes an actual space that fills with air (pneumothorax) or blood (hemothorax).
- Positive pressure in the pleural space presses against the lung tissue, causing atelectasis ↓ gas exchange.



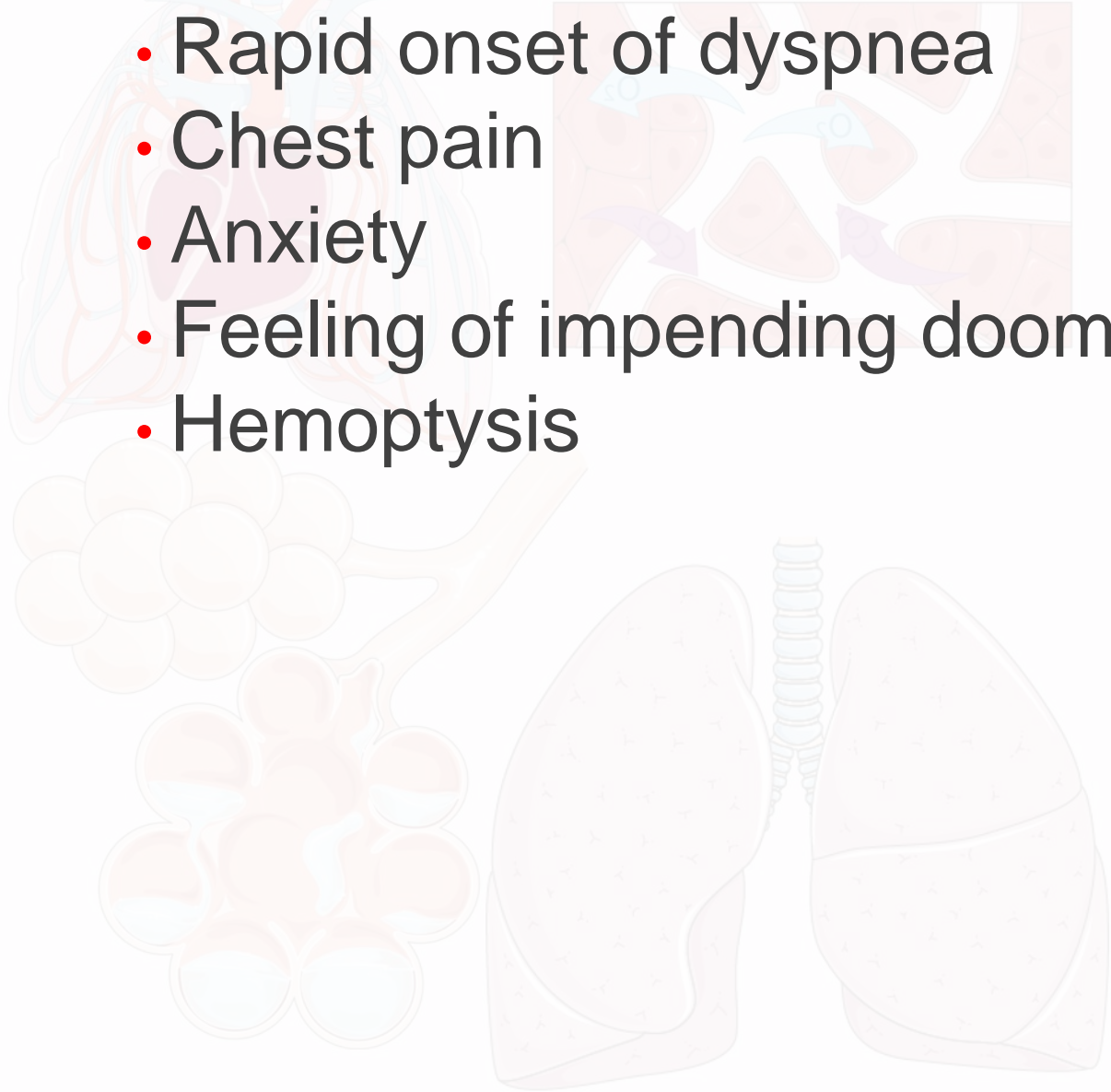
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Pulmonary Embolism

- Rapid onset of dyspnea
- Chest pain
- Anxiety
- Feeling of impending doom
- Hemoptysis

Pathophysiology

- Thrombus formation in the deep veins from an ineffective cardiac pump; atrial fibrillation; the presence of increased clotting factors; or lack of movement of the musculoskeletal pump, delaying blood movement back to the heart.
- Emboli may also consist of air, fat, amniotic fluid, and bacteria.
- The thrombus occludes pulmonary circulation, impairing gas exchange.



Sarcoidosis

- Fever
- Myalgia
- Night sweats
- Anorexia
- Weight loss
- Fatigue with
- Progressive
- Lung noncompliance and
- SOB



Pathophysiology

- Granulomatous disorder primarily of the lungs, skin, eyes, and lymphatics thought to have a genetic link. Other organs affected are the heart, bones, joints, liver, and kidneys.
- Genetic clusters include mainly African Americans and Scandinavians. Environmental influences are considered as genetic triggers. Affects those 40 years of age and younger.
- Hilar lymphadenopathy occurs, then progresses to lymphocytic alveolitis. Skin lesions, peripheral lymphadenopathy, interstitial nephritis, iritis, hepatomegaly and splenomegaly can also occur.
- Symptoms and complications are related to malabsorption.
- Can result in pulmonary fibrosis or associated right-sided heart failure (cor pulmonale).

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Tuberculosis (TB)

- Fatigue
- Sudden Weight loss
- Anorexia
- Night Sweats
- Low-grade fever
- Productive Cough
- Hemoptysis
- Chest pain
- Anxiety

Pathophysiology

- Mode of transmission: The tubercle bacilli are spread by the airborne route. The mycobacterium-containing droplet nuclei circulate in the air. A T-cell-mediated response occurs, walling off the lesion (Ghon tubercle), inactivating the disease.
- The Ghon tubercle affects the hilar region first. If the client becomes immunosuppressed, the Ghon necrose cavitates then may release the organism into the lung.