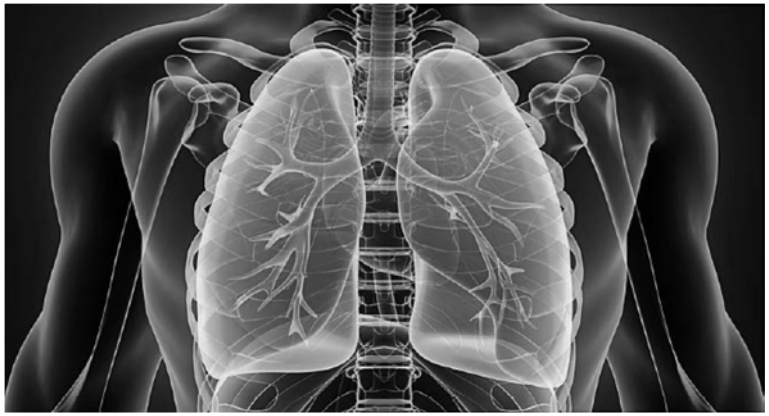


Advanced Pulmonary Pathophysiology

2022

COI Statement:

- ***I am a current member of the NBCRNA Board of Directors. The <u>content expressed</u> in my lecture is based on my own clinical expertise and in no way reflects any view, opinion or position of the NBCRNA. I HAVE NO ACCESS TO ANY OF THE NBCRNA ASSESSMENT ITEMS, CONTENT OR EXAM DEVELOPMENT, including, but not limited to, the CPCA.***

Objectives

The learner will be able to discuss the following advanced pulmonary pathophysiology and list the etiology and risk factors, clinical manifestations, pathological changes, and diagnostic results for:

- Bronchitis
- Pulmonary emphysema
- Asthma
- Bronchiectasis
- Pulmonary infections
- Acute respiratory distress syndrome (ARDS)
- Interstitial lung disease (ILD)
- Lung cancer
- Pulmonary Vascular Disorders
- Neuromuscular Disorders
- Pleural Diseases (including pneumothorax)

Classification of Pulmonary Disorders

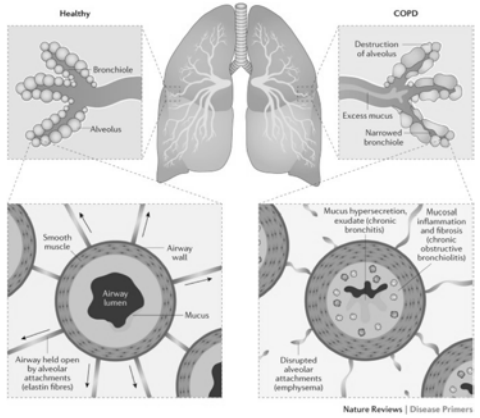
Obstructive disease

Causes a decrease in the rate of airflow in the conducting airways

Restrictive disease

Causes a decrease in the volume of lung, especially the inspiratory capacity and vital capacity

Obstructive Diseases



Chronic Obstructive Pulmonary Disease

- A group of disorders characterized by progressive **limitations in predominantly expiratory airflow** that are partially reversible by bronchodilator or anti-inflammatory therapy

Risk Factors for COPD

Definitions

Forced vital capacity (FVC):

- the determination of the vital capacity from a maximally forced expiratory effort

FEV_1 ($MMEF_1$):

- Volume that has been exhaled at the end of the first second of forced expiration

PEF :

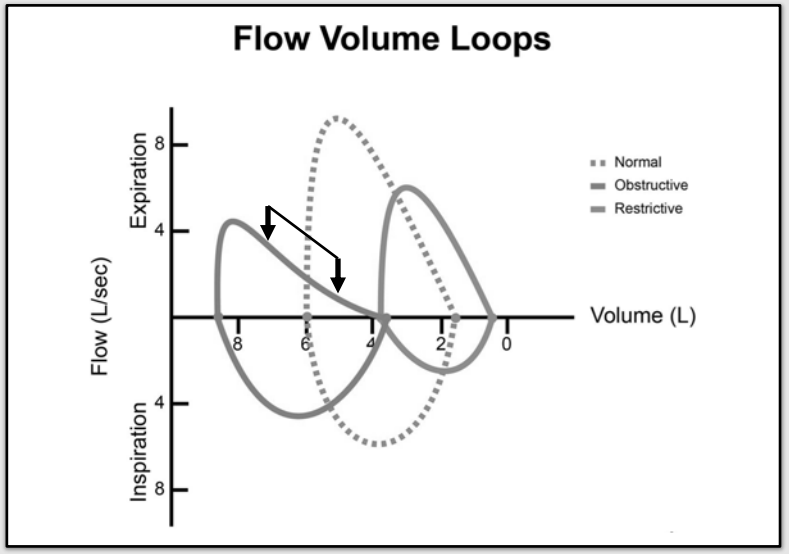
- The highest forced expiratory flow measured with a peak flow meter

MVV Maximal voluntary ventilation:

- **volume of air expired in a specified period during repetitive maximal effort (MMEF or $FEV_{25\%-75\%}$)**

MIP:

- Maximum inspiration (IC), used to assess diaphragm strength



Overall Classification of Pulmonary Disorders

- Obstructive Disease (COPD)
  - Causes a decrease in the rate of airflow in the conducting airways, causes an increase in residual volume due to air trapping

$FEV_1\downarrow$, FVC $\downarrow$, FEV_1/FVC < 70% of predicted, TLC > 120% of predicted,

RV > 120% of predicted, MMF $\downarrow$ DLCO < 80% of predicted, PEF $\downarrow$

DLCO: Diffusion capacity of lung for carbon dioxide

Overall Classification of Pulmonary Disorders

In obstructive lung disease, the FEV_1 is reduced due to obstruction to air escape. Thus, the FEV_1/FVC ratio will be reduced.

More specifically, the diagnosis of COPD is made when the FEV_1/FVC ratio is less than 70%.

The Global Initiative for Obstructive Lung Disease (GOLD) criteria also require that values are after bronchodilator medication has been given to make the diagnosis

Dx: Pre-post bronchodilator testing with Spirometry testing. In Emphysema/Bronchitis small change less than 5%; Asthma typically changes >12% or 200 mL

Overall Classification of Pulmonary Disorders

- **Restrictive Disease**
  - Causes a decrease in the volume of lung, especially the inspiratory capacity and vital capacity

FEV1↓, FVC↓, FEV1/FVC↑ or normal, TLC < 80% of predicted, RV < 80% of predicted, MMF↑, DLCO > 120-140% of predicted, PEF normal or increased

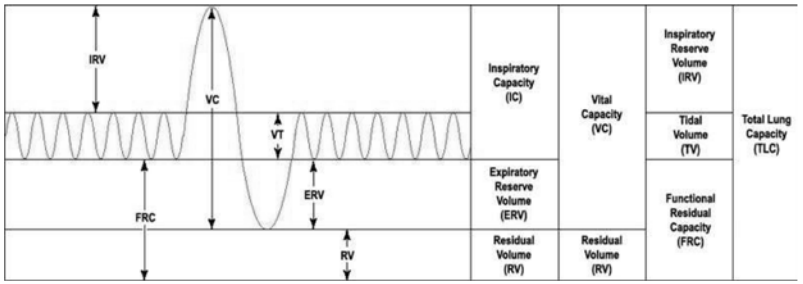
Overall Classification of Pulmonary Disorders

Restrictive lung disease:

the FEV_1 *and* FVC are equally reduced due to fibrosis or other lung pathology (not obstructive pathology).

FEV_1/FVC ratio should be approximately normal, or even increased due to an increased FEV_1 value (because of the decreased compliance associated with the presence of fibrosis in some pathological conditions).

Spirogram



Spirogram Capacities and Volumes

- TLC Total lung capacity: the volume in the lungs at maximal inflation
- RV Residual volume: the volume of air remaining in the lungs after a maximal exhalation
- ERV Expiratory reserve volume: the maximal volume of air that can be exhaled from the end-expiratory position
- IRV Inspiratory reserve volume: the maximal volume that can be inhaled from the end-inspiratory level

Spirogram Capacities and Volumes

IC Inspiratory capacity: the sum of IRV and TV

IVC Inspiratory vital capacity: the maximum volume of air inhaled from the point of maximum expiration

VC Vital capacity: the volume equal to TLC – RV

V_T Tidal volume: that volume of air moved into or out of the lungs during quiet breathing

FRC Functional residual capacity: the volume in the lungs at the end-expiratory position RV/TLC% Residual volume expressed as percent of TLC

FEV1/FVC ratio

The **FEV_1/FVC ratio**, also called Tiffeneau index, is a calculated ratio used in the diagnosis of obstructive and restrictive lung disease

It represents the proportion of the forced vital capacity exhaled in the first second

Normal values are approximately 80% of predicted

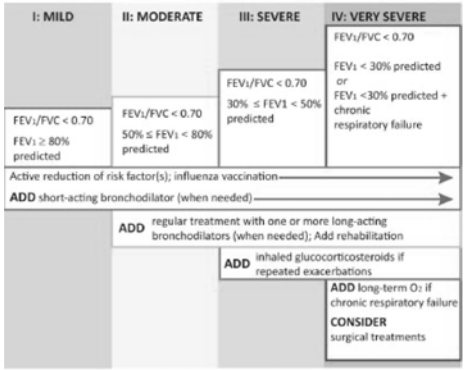
Predicted normal values are calculated based on age, sex, height, weight and ethnicity, sometimes smoking

A derived value of FEV_1% is **FEV_1% predicted**, which is defined as FEV_1% of the patient divided by the average FEV_1% in the population for any person of similar age, sex and body composition.

Chronic Obstructive Pulmonary Disease

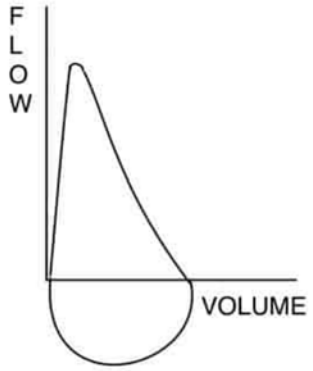
- May be preventable and treatable. Disease state characterized by airflow limitation that is not fully responsive to bronchodilator therapy. The airflow limitation is progressive and associated with an abnormal inflammatory response of the airway.
- ***Primary*** cause is cigarette smoking
- A significant response to the bronchodilator is considered by an increase in the FEV_1 by 12% AND an increase in VC by 200 mL.

Therapy at Each Stage of COPD



The following pressure volume loop indicates which pulmonary condition (Select all that apply);

a) Normal
b) Restrictive
c) Obstructive
d) Asthma



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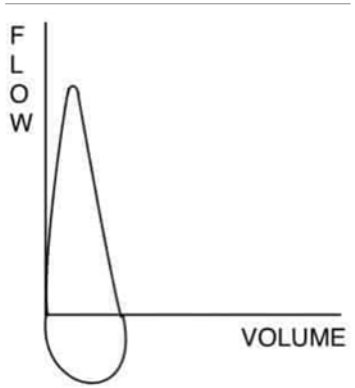

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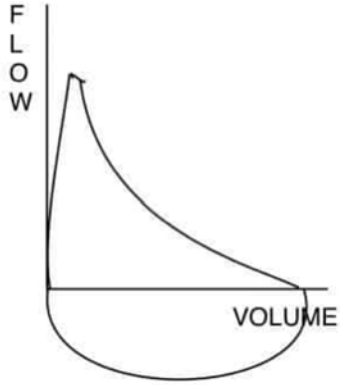


COPD: Epidemiology

- Some 16 Million Americans are affected
- COPD is the 3rd leading cause of death in the U.S.
- COPD caused 726,000 hospitalizations
- Total health expenditure of $32.1 Billion
- Most common form of COPD is Chronic Bronchitis

Risk Factors for COPD

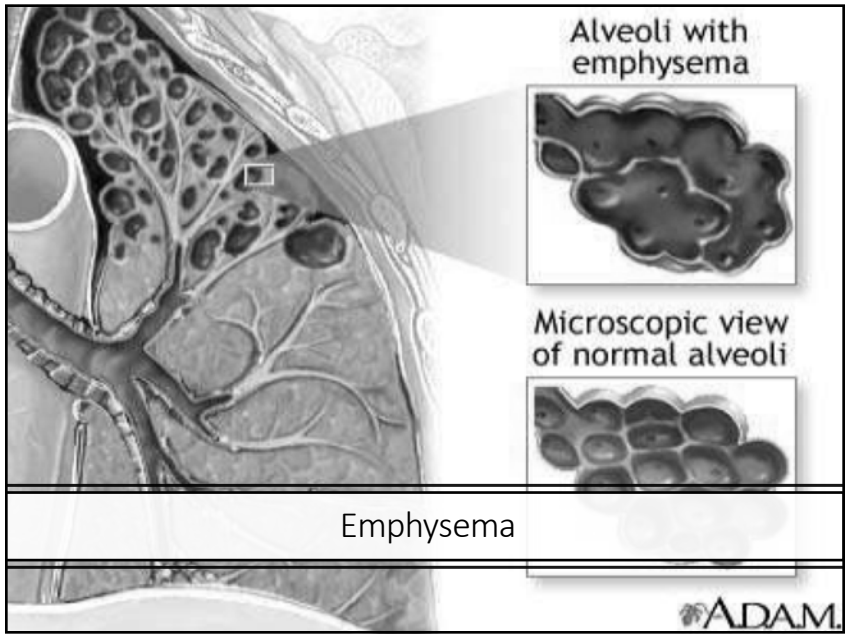
- Cigarette smoking/passive smoking
- Pollution
- Occupational exposure to dust and fumes
- Recurrent lung infections
- Hereditary factors
- Allergies
- Socioeconomic factors
- Alcohol ingestion
- Age

Chronic Obstructive Pulmonary Disease

- Smoking
  - #1 cause of COPD
  - Increased mucous production
  - Inhibition of mucociliary clearance
  - Toxicity of inhaled gases and particulates
  - Bronchospasm
  - Decrease in macrophage activity
  - Disruption of the alveolar wall and capillary endothelium

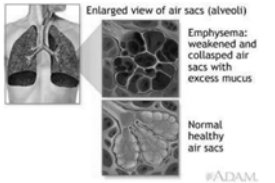
General Manifestations of COPD

- Small airways (< 2mm) are most susceptible to airway obstruction in COPD
- Diagnosed by PFT, clinical signs and symptoms
- Early to middle manifestations of COPD include:
  - Changes in pulmonary function testing
  - Shortness of breath with exertion
  - Changes in CXR
  - Increases in sputum production
  - Cough
  - Recurrent pulmonary infections
  - Wheezing
- Late manifestations of COPD include:
  - Accessory muscle usage
  - Edema from Cor Pulmonale
  - Mental status changes from chronic hypoxia/hypercapnea
  - Clubbing of fingers
  - Barrel Chest or Increased A-P Diameter



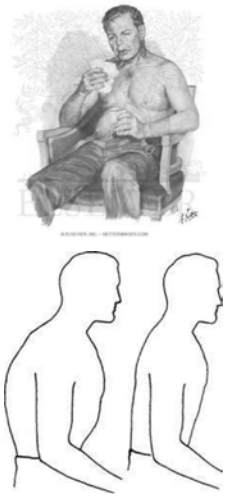
What is Emphysema?

- Loss of elastic recoil
- This loss of recoil leads to an increased compliance and inability to expel gas out of the alveoli
- Leading to trapped air in the lung
- Alveoli cluster together forming “blebs”



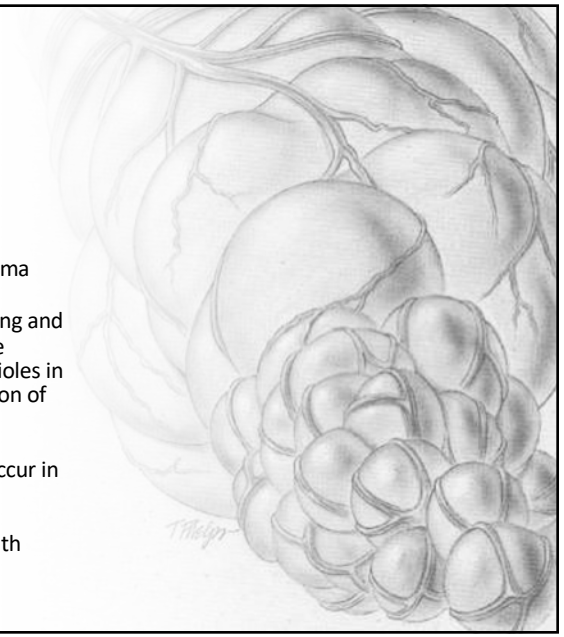
Emphysema Cont…

- Hence, the next breath is started with more air in the lungs.
- The trapped "old" air takes up space, so the alveoli are unable to fill with enough fresh air to supply the body with needed oxygen.



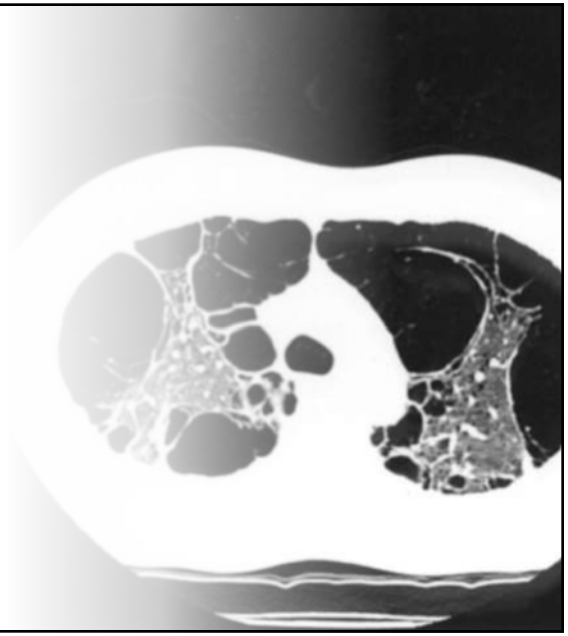
Pulmonary Emphysema

- Centrilobular emphysema
  - Abnormal weakening and enlargement of the respiratory bronchioles in the proximal portion of the acinus
  - Primary changes occur in upper lobes
  - High correlation with smoking



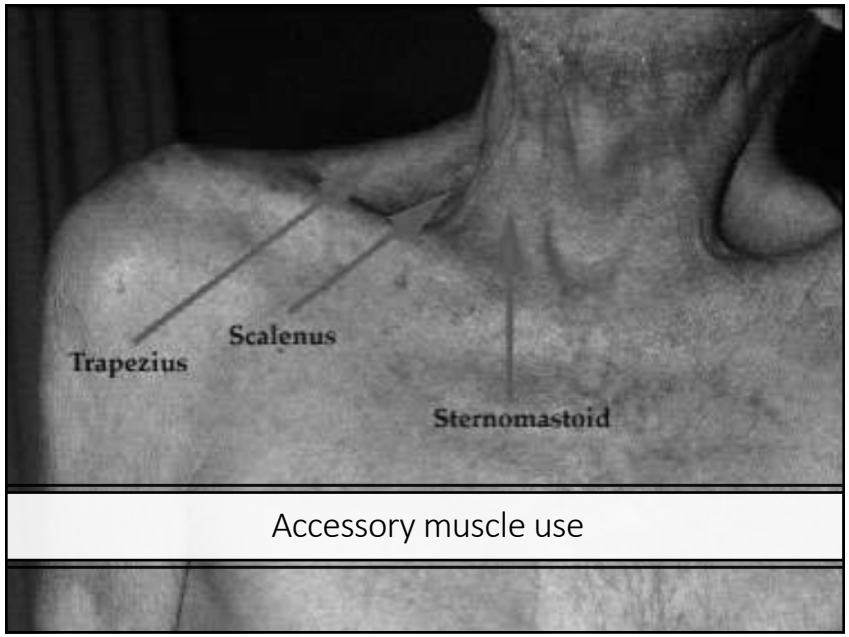
Pulmonary Emphysema

- Bullous emphysema
  - Changes seen at both respiratory bronchiole and alveolar levels
  - Prominent bullae formation (air spaces greater than 1 cm in diameter)



Emphysema Cont…

- A person with emphysema may feel **short of breath** during exertion and, as the disease progresses, even **while at rest**.
- Emphysema is one of several **irreversible lung diseases** that diminish the ability to exhale. This group of diseases is called **chronic obstructive pulmonary disease** (COPD). The two major diseases in this category are emphysema and **chronic bronchitis**, which often develop together.



Accessory muscle use

Emphysema

- Typically, symptoms of emphysema appear only after 30 to 50 percent of lung tissue is lost.
- Emphysema rates are highest for men age 65 and older.
- More people in the Midwest have emphysema than in any other region in the country.
- Emphysema is an irreversible disease that can be slowed but not reversed or stopped.

Causes

Generally, lungs become damaged because of reactions to irritants entering the airways and alveoli. Causes for emphysema:

1. Cigarette smoking
2. Alpha-1 antitrypsin deficiency

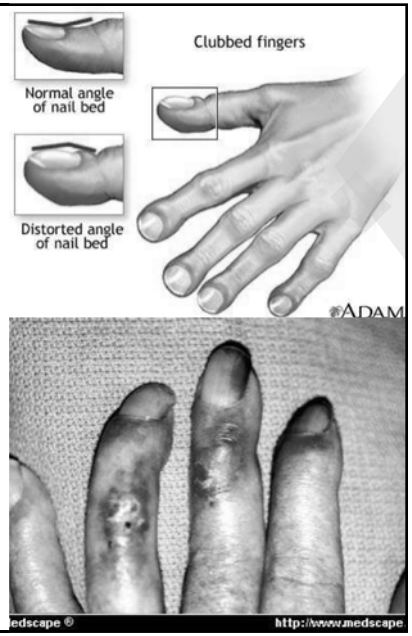
Symptoms

- The first sign of emphysema is shortness of breath during exertion. Eventually, this shortness of breath occurs while at rest. As the disease progresses, the following symptoms which are related to one of the other major lung diseases also caused by smoking – bronchitis may occur:

- Difficulty breathing (dyspnea)
- Coughing (with or without sputum)
- Wheezing (this can also be caused by emphysema itself)
- Excess mucus production
- A bluish tint to the skin (cyanosis)
- Hypoxemia
- Tachycardia
- Polycythemia

More Symptoms

- Clubbed fingers (chronic hypoxia)
- Right Heart Failure
- Stained yellow fingers, teeth



Diagnosis

- **History And Physical Examination**
- Smoking history (calculate pack years, # packs smoked times # years smoked)
- Working environment- breathing in any harmful chemicals?
- A physical examination will include an examination of your chest and breathing patterns; prolonged expiratory times
- Nasal flaring, accessory muscle usage (due to loss of diaphragm recoil from air trapping)

Diagnosis

X-Ray and/or CT of the Chest

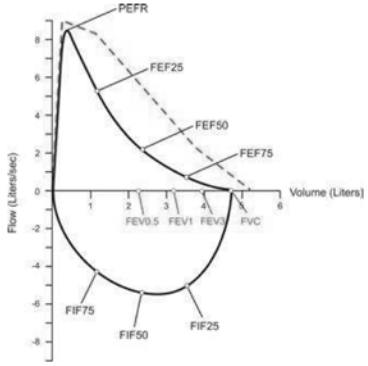
- Chest x-rays are a very useful tool to evaluate anatomy of the lung. In emphysema, there is evidence of increased air in the chest and destruction of some of the lung tissue. Bronchitis can be suspected on a chest x-ray by presence of thickening of the tissue around the large airways (bronchi). Chest x-rays are also useful as screening for lung cancer and heart disease.
- Computerized axial tomography or CAT scans indicate lung anatomy in greater detail. In some cases, this information is needed to fully evaluate lung disease.

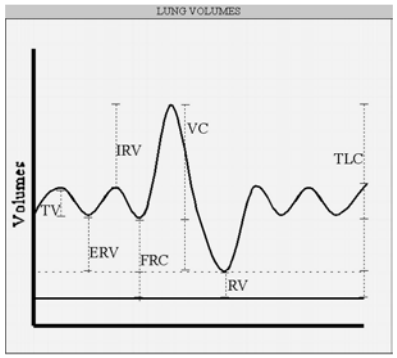
Lung Function Tests

Frequently, your physician will ask that spirometry and body plethysmography be repeated after administration of an inhaled **bronchodilator**

This test will help your physician determine if there is an asthmatic component present

Lung Volumes measures the amount of air in the lungs. This increases markedly in emphysema.





Lung Function Tests

Treatment for Emphysema

- There is no cure for emphysema. The goal of treatment is to slow the development of disabling symptoms. The most important step to take is to **stop smoking**.
- Treatments for emphysema caused by smoking include medication, breathing retraining, and surgery.
- People with inherited emphysema due to alpha-1 antitrypsin deficiency can receive alpha 1-proteinase inhibitor (A1PI), which slows lung tissue destruction.

Medications Used

- **Medications To Treat Emphysema**
- Emphysema cannot be cured and, except for oxygen, does not reverse with any medication. However, emphysema is frequently associated with bronchitis and asthma and the symptoms associated with these processes often can be alleviated with medication (hence, you can see the value of pulmonary function and other tests designed to discover if there is asthmatic component present:
- **Bronchodilator medication**
- **Corticosteroids**
- **Supplemental oxygen**

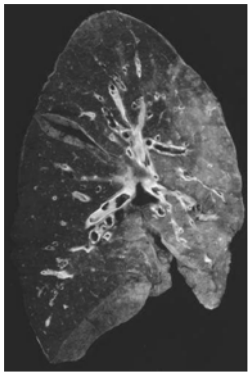
Medications Used

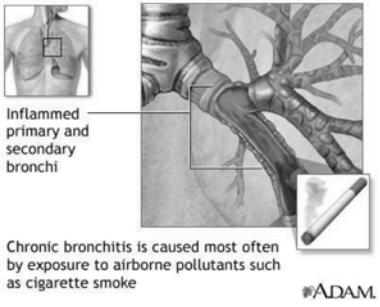
- **Bronchodilator Medication**
  - Bronchodilator medication may be prescribed for airway tightness. Bronchodilators react similar to norepinephrine through the sympathetic nervous system
  - The most commonly prescribed bronchodilators are beta2 agonists, the anti-cholinergic drug ipatropium bromide, and theophylline.
  - Anti-cholinergics block musacaric receptors which normally respond to acetylcholine and cause bronchoconstriction

Oxygen and Hypoxic Drive

Due to the chronic state of increased CO_2 in the blood (hypercapnia), the patient has adapted a breathing regulation in the brain that responds to changes in O_2 and not CO_2 like most people (Hypoxic Drive)

- If you give a patient with COPD more than 30% oxygen, they will slow their breathing
- Give low flow oxygen at 2 LPM by NC
- Or high flow oxygen with a venturi mask at 22-30%

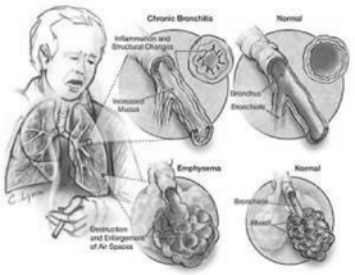




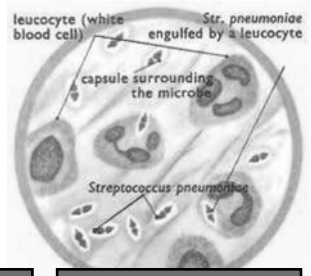
Chronic Bronchitis

Chronic Bronchitis

- Presence of cough and sputum production for **three or more months in two successive years**
- Etiology
  - Smoking
  - Air pollution
  - Chronic infections
  - Chronic Bronchitis Symptoms



Chronic Bronchitis



14 million Americans are affected	Most common causes are smoking/pollution	Repeated lung infections, especially in childhood increase risk
Common pathogens include ***Haemophilus influenzae*** **and**	***Streptococcus pneumoniae***	Gastroesophageal reflux disease (GERD) can lead to pneumonias from aspiration of stomach contents

Chronic Bronchitis – Pathophysiology

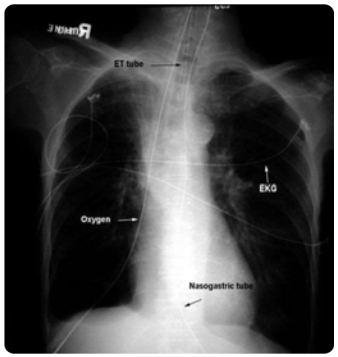
- Most changes in the lungs occur in the conducting airways
- Airway changes occur from:
  1. Chronic inflammation and swelling
  2. Excessive mucus production and accumulation
  3. Partial or total mucus plugging
  4. Hyperinflation of alveoli
  5. Smooth muscle constriction of airways

Chronic Bronchitis – Pathophysiology

- Changes in mucus glands
  - Increase in number of mucus secreting glands; goblet cells increase, causing decrease in ciliated columnar cells; submucosal glands hypertrophy
- Smooth muscle hypertrophy in bronchial airways
- Diminished airway radius

Chronic Bronchitis – Pathophysiology

- Increase in sputum production
- Accumulation of secretions
- Loss of ciliated cells
- Impairment of mucociliary escalator
- Decreased flow rates, VC, FVC, FEV_1, MMEF
- Increased RV, FRC, TLC



Chronic Bronchitis

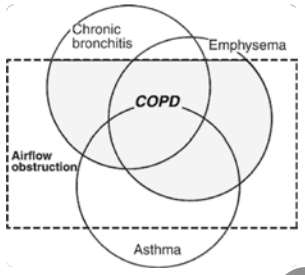
- Radiologic Findings
- Hyperinflation of the Lungs
- Flattened Hemi-diaphragm
- Peripheral Pulmonary Vasculature may be Prominent
- Pulmonary Vascular Engorgement
- Long and narrow heart (pulled down by the diaphragms)
- Enlarged heart

Chronic Bronchitis – Clinical Findings

- Typical appearance is of the "Blue Bloater"
  - Stocky build
  - Cyanotic
  - Increased A-P diameter
  - Jugular vein distension
  - Edema

Chronic Bronchitis – Clinical Findings

- Cough
  - Smoker's cough
  - Morning cough
  - Continual cough
- Sputum production
  - Volume increases slowly leading to abnormal production but typically less than a cup/day
  - Thick, gray, mucoid in nature
  - **Mucopurulent infections** leading to yellow or green sputum



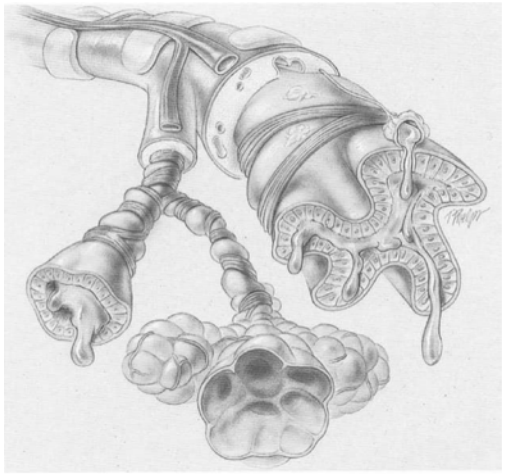
Chronic Bronchitis – Clinical Findings

- Increase in respiratory rate
  - Stimulation of peripheral chemoreceptors secondary to hypoxemia and chronic CO2 retention
- Decrease in lung compliance
  - Anxiety
- Increase in heart rate
- Dyspnea, especially on exertion
- Use of accessory muscles
- **BS: rhonchi, crackles, wheezing and decreased BS**

Chronic Bronchitis – Clinical Findings

- Pursed lip breathing
- Increase in A-P diameter of the chest (barrel chest) secondary to hyperinflation
- Clubbing
- Increased sputum production
- ABG results
  - Fully compensated pH unless in an acute exacerbation
  - Increase in P_aCO_2
  - Decrease in P_aO_2

Asthma



Asthma

- A disease of the airway "characterized by an increased responsiveness of the trachea and bronchi to various stimuli and is manifested by widespread narrowing of the airways that change in severity either spontaneously or as a result of treatment" (ATS)

Asthma

- Airway constriction *may* be partially or completely reversible either spontaneously or with treatment
- Affects more than 15 million Americans
- Recognized more than 2000 years ago
- More than 5,000 die per year - Teen dies of asthma
- The most common chronic illness of childhood
- May develop in adulthood with increased mortality
- May disappear at puberty

Asthma

Allergic or Extrinsic Asthma

- Results from an antigen-antibody reaction on mast cells causing a release of histamine, bradykinins, and other chemicals

Idiopathic or Intrinsic Asthma

- Cannot be linked to a specific antigen
- Results from an imbalance of the autonomic nervous system

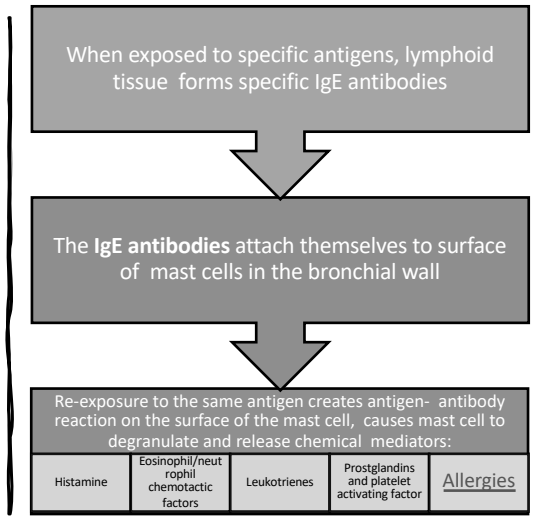
Non-specific Asthma

- Results from an unknown cause, possibly viral, emotional, or
- exercise

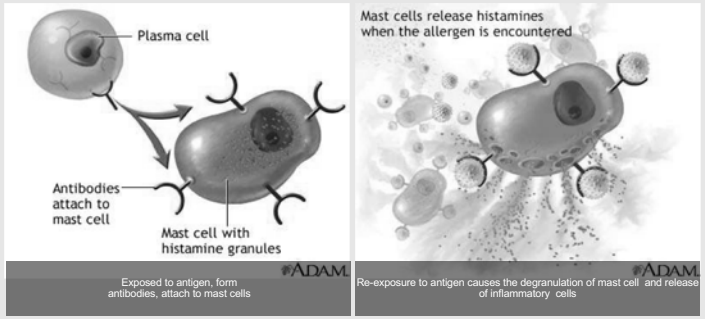
Asthma

- Seen predominantly in adults, more than 300 substances contribute to it.
- Sensitive work environments include:
  - Farming
  - Agricultural
  - Painting
  - Cleaning work
  - Plastic manufacturing

Immunologic Mechanism

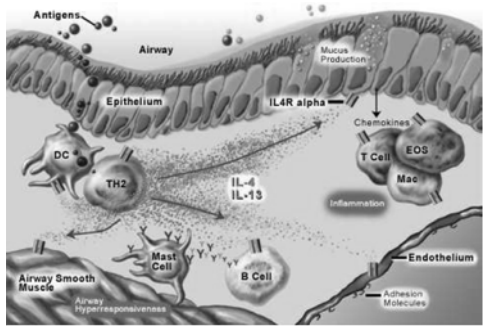


Mast Cell Degranulation



Mast Cell Degranulation

Following an Asthma attack; the patient will have congestion and increased sputum production for several days



Inflammatory cell release

- Release of chemical mediators from mast cell stimulates parasympathetic nerve endings in the bronchial airways leading to reflex bronchoconstriction and mucous hyper- secretion
- The mediators also increase permeability of capillaries causing dilation of blood vessels and tissue edema
- Early vs. late response (after steroids and bronchodilators have worn off)

Mast Cell inhibitors for asthma treatment

Cromolyn sodium (Intal) and nedocromil (Tilade) are used to prevent allergic symptoms like runny nose, itchy eyes, and asthma. The response is not as potent as that of corticosteroid inhalers.

How mast cell inhibitors work

- These drugs prevent the release of histamine and other chemicals from mast cells that cause asthma symptoms when you come into contact with an allergen (for example, pollen). The drug is not effective until four to seven days after you begin taking it.

Who should Use it

- Patients with extrinsic asthma, with known allergies
- Frequent dosing is necessary, since the effects last only six to eight hours. Mast cell inhibitors are available as a liquid to be used with a nebulizer, a capsule that is placed in a device that releases the capsule powder to inhale, and handheld inhalers

Intal and Tilade

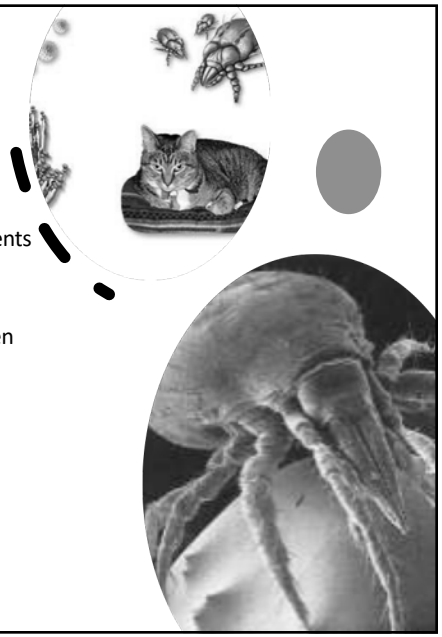
- Both drugs are used only for prophylaxis of asthma, not for treatment of the acute exacerbation or for the symptomatic patient

Anti-Leukotriens

- Do not prevent mast cell degranulation, as do Intal and Tilade
- They stop the inflammatory mediators once the mast cells is degranulated
- Leukotrienes are proinflammatory mediators with special significance in asthma. Released by numerous cell types, particularly after exposure to allergens, leukotrienes cause a potent contraction of bronchial smooth muscle, resulting in reduced airway caliber. Further, they cause plasma to leak from the vessels, resulting in edema, and enhance the secretion of mucus

Asthma

- Etiology
  - Heredity – one or more parents with disease
  - Allergies, especially if onset between ages five and fifteen
  - Inhaled irritants
    - Pollen
    - Dust mites
    - Grasses
    - Pollution
    - Animal dander
    - Chemicals



Asthma Risk Factors

Obesity: Certain mediators such as leptins may have an effect on airway function that can lead to development of asthma

Gender: Males up to 14, have a higher prevalence, due to possible lung size of boys vs. girls, after 14, girls have a higher prevalence

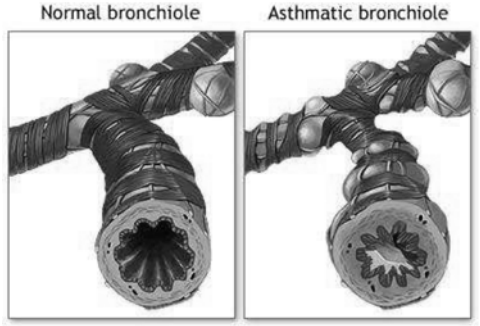
Infections: upper viral infections and bacterial infections contribute to asthma. Commonly seen in children after RSV, parainfluenza, rhinovirus.

Exercise induced: heat loss, water loss, increased osmolority increase inflammatory release

Asthma – Pathophysiology

- Airway Inflammation
  - Acute Phase Response – triggered by activation of mast
  - cells and the release of intracellular mediators

1. Bronchospasm
2. Increase in secretions
3. Mucosal edema
4. Significant reduction in airflow

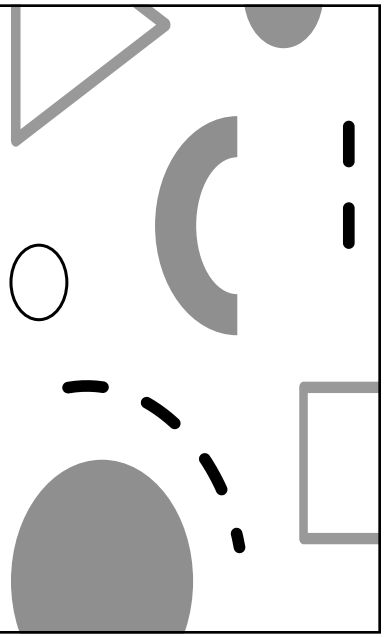


Asthma – Pathophysiology

- Airway Inflammation
  - **Subacute phase**
    - Continuous inflammatory pattern
    - Significant airflow limitation
    - Can continue for days to weeks

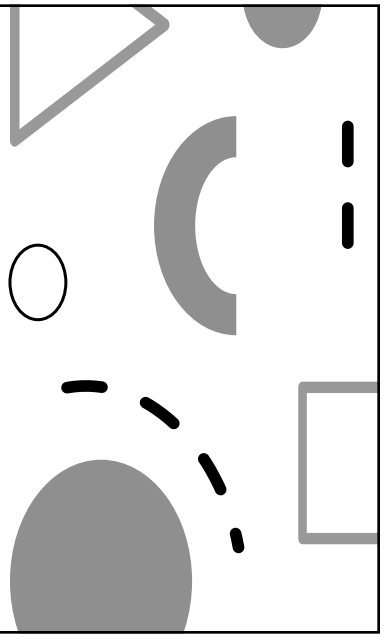
Asthma – Pathophysiology

- Airway Inflammation
  - **Chronic inflammation**
    - Present between episodes of exacerbation
    - Controlled by corticosteroids, mast cell modifiers, or leukotriene modifiers

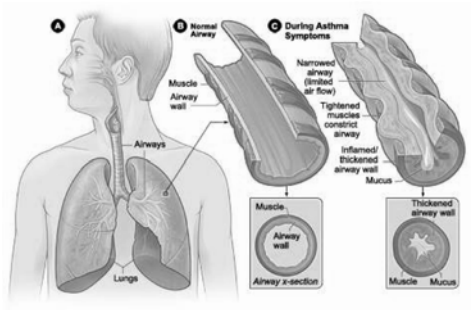


Asthma – Pathophysiology

- Airway Hyperresponsiveness
  - Usually most evident in acute phase
  - Increased sensitivity to both specific and non-specific causes
  - Release of **immunoglobulin E (IgE) mediators** into the cellular tissue causing bronchoconstriciton of the smooth muscle of the airway, degranulation of mast cells releasing histamines, leukotrienes, certain interleukins, prostaglandins and others
  - Treated with beta$_2$ agonists



Asthma – Clinical Findings



Asthma – Clinical Findings

- Auscultation – episodic wheezing
  - Absence of wheezing does not preclude asthma
  - Not all wheezing is asthma
  - Breath sounds may get worse but patient could be improving
- Shortness of breath
- Tachypnea
- Tachycardia
- Use of accessory muscles
- Pursed-lip breathing
- Anxiety
- Hypoxia
- Altered LOC
- Full Arrest
- BS – wheezes, crackles, rhonchi, decreased BS

Asthma – Clinical Findings

- Blood Gas Results
  - In **mild to moderate** episode:

    pH ↑ PCO_2 ↓ HCO_3 ↓ slightly PaO_2 ↓
  - In **moderate to severe** episode:
    - pH ↓ PCO_2 ↑ HCO_3 ↑ slightly PaO_2 ↓

Pharmacotherapy

Corticosteroids

- Most effective mediation in treatment of asthma
  - Reduces symptoms and mortality
- Use of inhaled steroids for long-term treatment preferred
  - Use spacer and rinse mouth to eliminate or minimize side effects
- Long-term use of oral steroids should be restricted to patients with asthma refractory to other treatment.
- Short-term oral steroid use during exacerbation reduces severity, duration, and mortality.

Pharmacotherapy

Inhaled Corticosteroids

- Beclomethasone (QVAR); 40 or 80 ug/puff BID
- Flunisolide (Aerobid); 250 ug/puff; BID
- Fluticosone (Flovent); 44, 110, or 220 ug/puff, BID
- Budesonide (Pulmicort); SVN 0.25 or 0.5 mg, BID
- Momestone furoate (Asmanex twisthaler) DPI 220 ug QD

Pharmacotherapy

Systemic Steroids Corticosteroids

- Prednisone (short term use following an acute attack) usually 3-5 days, BID
- Methylpredinsone (Solu-Medrol); Typically an IV potent systemic steroid, given during and after acute attacks

Pharmacotherapy (cont.)

Cromolyn (NSAID) non-steroidal anti-inflammatory drug
- Protective against allergens, cold air, exercise
- Administered prophylactically, CANNOT be used during an acute asthma attack
- Of limited use in adults
- Drug of choice for atopic children with asthma

Nedocromil (NSAID)
- Similar to Cromolyn, it is 4–10 times more potent in preventing acute allergic bronchospasm.

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Pharmacotherapy (cont.)

Leukotriene inhibitors
- Leukotrienes mediate inflammation and bronchospasms.
- Modestly effective to control mild to moderate asthma
- Accolate, Singular, Zyflo

Inhaled steroids remain the anti-inflammatory drug of choice for the treatment of asthma.

Methyxanthines (**use is controversial**)
- Oral or IV use if admitted for acute asthma attack
- Theophylline

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Pharmacotherapy (cont.)

β_2-Adrenergic agonists Short Acting

- Most rapid and effective bronchodilator
- Drug of choice for exercise-induced asthma and emergency relief of bronchospasms
- Used PRN
- Improves symptoms not underlying inflammation
  - Regular use may worsen asthma control and increase risk of death.
  - Albuterol (Proventil, Ventolin); SVN UD 0.5% Soln, or 2.5 mg (0.5 ml) give TID, QID, Q4, Q6 or PRN
  - Levalbuterol (Xopenex), SVN 0.31, 0.63, or 1.25 mg

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Pharmacotherapy (cont.)

β_2-Adrenergic agonists Ultra Short Acting

- Epinephrine (Epinephrine Mist, Primatene mist): SVN 1% soln (1:100), 0.25-0.5 ml QID; MDI 0.22 mg/puff
- Racemic Epinephrine; (Micronephrine, Nephrone); SVN 2.25% soln, 0.25-0.5 ml QID

  Last about 90 minutes, Racemic has a strong Beta and Alpha response, used for upper airway swelling

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Pharmacotherapy (cont.)

- Anticholinergics
  - Can be used as adjunct to first-line bronchodilators if there is an inadequate response
  - Has an additive affect to Beta$_2$-agonists
  - Blocks muscarinic receptors (Acetylcholine)
  - Ipatropium Bromide (Atrovent); SVN 0.5 mg, 0.02% solution MDI 18 ug/puff; dose TID, Q6
  - Tiotropium (Spiriva), used through a handi-haler, QD

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Special Considerations in Asthma Management (cont.)

- **Aspirin sensitivity**
  - 5% of adult asthmatics will have severe, life-threatening asthma attacks after taking NSAIDs.
  - All asthmatics should avoid; suggest Tylenol use.
- Asthma during pregnancy
  - A third of asthmatics have worse control at this time.
  - Much higher fetal risk associated with uncontrolled asthma than
  - that of asthma medications
  - Theophyllines, B_2-agonists, and steroids can be used without significant risk of fetal abnormalities.

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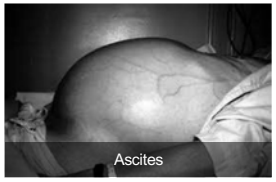
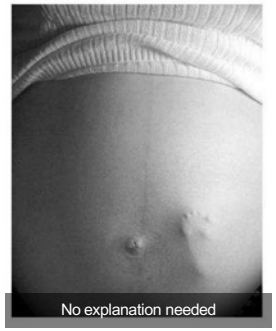
Restrictive Diseases





Restrictive Diseases

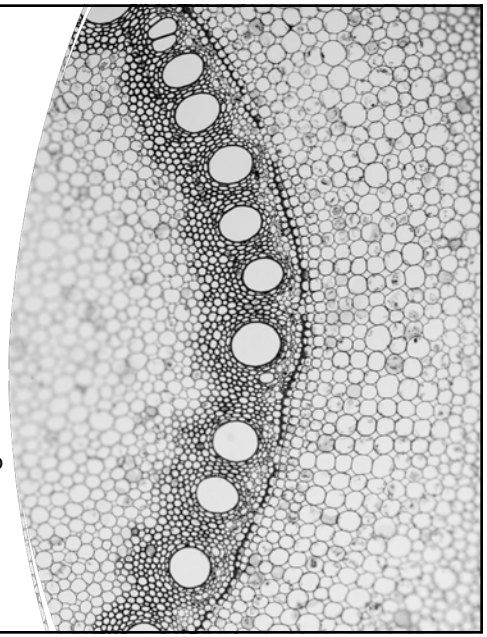
- Restrictive diseases generally:
  - Causes a decrease in lung compliance
  - Decrease in lung volumes
  - Can be caused by any alveolar filling lung defect
  - Can be intrinsic or extrinsic

Respiratory Distress Syndrome – Etiology

- Primary Risk Factors – Direct Lung Injury
  - Pneumonia
  - Aspiration of gastric contents
  - Inhalation of toxic substances, including high concentrations of oxygen
  - Near drowning
  - Lung contusion

Respiratory Distress Syndrome – Pathophysiology

- Three Phases of ARDS
  - Exudative phase: generally last three to five days
  - Fibroproliferative phase: can last for a few days to weeks
  - Resolution phase



Respiratory Distress Syndrome – Pathophysiology

Exudative phase

- Consolidative process with injury to the alveoli
- Inflammatory process secondary to the presence of activated macrophages
- Destruction of type I pneumocytes
- Migration of interstitial fluid, protein, fibrin, neutrophils, and red blood cells through the damaged alveolar wall

Respiratory Distress Syndrome – Pathophysiology

Fibroproliferative phase

- Lung repair begins
- Macrophage and lymphatic cleanup of cellular debris
- Hyperplasia of alveolar type II pneumocytes, proliferation of fibroblasts within alveolar basement membrane and intraalveolar spaces.
- Variable lung fibrosis can occur depending on the extent of fibroblast involvement

Respiratory Distress Syndrome – Pathophysiology

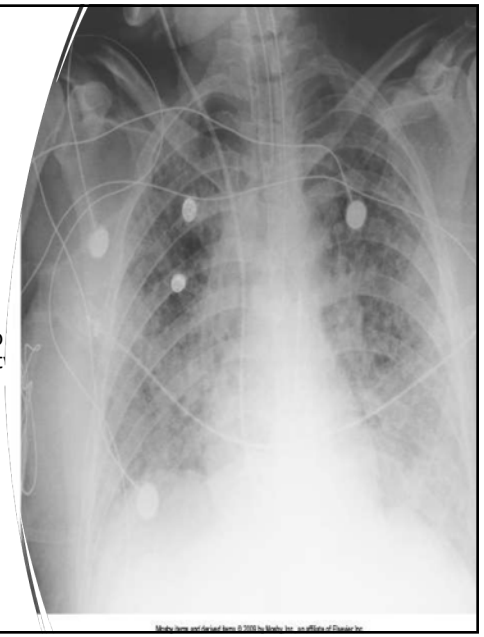
Resolution phase

- Increases in lung compliance
- Decreases in oxygen requirements
- Clearing of CXR
- Weaning of ventilatory support

Respiratory Distress Syndrome

Radiologic Findings

- Increased opacity due to the increased lung densit
- Ground glass appearance
- Infiltrates of one or both lungs



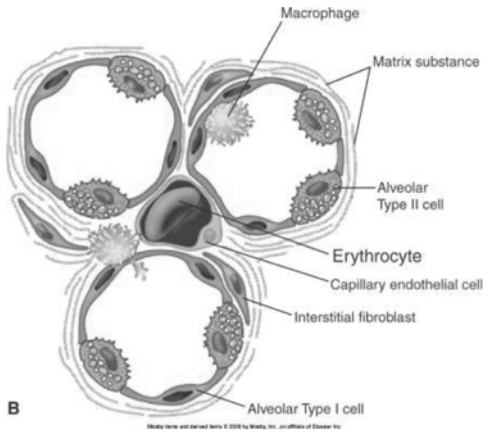
Respiratory Distress Syndrome

Clinical Findings

- Tachypnea disproportionate to blood gas changes
- Progressive hypoxemia
- Increase in A-a gradient
- Decrease in vital capacity
- Pulmonary capillary wedge pressure of < 18 mmHg
- A downward trending of static and dynamic compliance

Pulmonary Interstitial Disease – Pathophysiology

- Interstitial space is the area between the Type I and Type II alveolar cells and the vascular endothelial cells
- Contains macrophages, interstitial fibroblasts, and matrix substance.



Thank You