I – Allergy – Many reactions are commonly referred to as allergies. We will limit our discussion to true type I hypersensitivity reactions (immediate hypersensitivity). Our incredibly complex immune system produces antibodies to fight off foreign antigens. The antibodies involved in allergic reactions are primarily IgE.

**Type I hypersensitivity** is also known as immediate hypersensitivity. This reaction is what we commonly refer to as an allergic reaction. The most dramatic (and potentially lethal) presentation of type I hypersensitivity is the anaphylactic reaction. The sequence of events of an allergic reaction is as follows. Upon exposure to a specific antigen, a genetically predisposed individual develops IgE antibodies to the antigen. These IgE antibodies bind to mast cells and basophils. When the person is re-exposed to the antigen the antibodies on the mast cells stimulate the mast cells to degranulate, or release granules containing various destructive chemicals. These chemicals include *histamine* and other vasoactive amines that lead to an **immediate reaction** of increased vascular permeability, vasodilation, smooth muscle contraction, bronchoconstriction, and increased mucous secretion. Lipid mediators such as prostaglandins and leukotrienes are also released during this immediate response. **Leukotrienes** are the most vasoactive and spasmogenic agents known! In addition to this immediate phase reaction, the mast cells are stimulated to secrete **cytokines** that lead to a **late phase reaction** that causes leukocyte infiltration, epithelial damage, and more bronchospasm. Milder forms of this type of reaction lead to typical allergies (including allergic conjunctivitis), asthma, and urticaria. The most devastating example of this type I reaction is anaphylaxis (a type of shock) with whole body urticaria, laryngeal edema and spasm, followed by systemic vasodilation, hypotension and circulatory collapse ending in death.

Treatment of allergic reactions includes:

* testing for and avoidance of the offending antigens
* antihistamines
* leukotriene inhibitors
* mast cell stabilizers
* corticosteroids
* bronchodilators
II - OBSTRUCTIVE LUNG DISEASE – Airway disease – with these conditions the FEV1/FVC is reduced, indicating a problem with moving air through the bronchial passages. The FEV1/FVC is typically decreased to less than 75%, and normal is usually 75% - 80% or so. With some of these conditions the Total Lung Capacity (TLC) is increased also.

Asthma – Episodic, reversible bronchospasm. Clinically this is manifested by episodes of shortness of breath, cough and wheezing. It affects nearly 5% of adults and 7-10% of children. The pathology here is an exaggerated bronchoconstrictor response called reactive airway disease. The most current thought is that bronchial inflammation is what leads to this hyperresponsiveness. There are two types of asthma:

1. **Extrinsic Asthma** – Due to allergy and type I immune reaction. Ig E and mast cells play a key role here. This type of asthma is more common in children and they often “outgrow” it.

2. **Intrinsic Asthma** – Triggers are nonimmune here. Things such as dust, weather changes, exercise, URIs, emotional stress, etc. trigger bronchospasm. Often there is overlap between these two types.

Treatment of asthma includes

*bronchodilators (albuterol, epinephrine, theophylline, etc.)
mast cell stabilizers
*steroids (oral or inhaled)
*leukotriene inhibitors
*Desensitization allergy shots.

Although antihistamines are used in allergic conditions in general they are not usually thought of as a treatment for asthma. Oxygen is typically only used in emergency situations.
COPD – Chronic Obstructive Pulmonary Disease is a clinical designation that includes both Emphysema and Chronic Bronchitis. These conditions often co-exist and therefore are grouped together.

1. Emphysema – this is characterized by permanent enlargement of the airspaces distal to the terminal bronchioles by destruction of their walls. The primary cause of emphysema is cigarette smoking, but there is a rare genetic form involving alpha 1 antitripsin deficiency. The classic example of someone with “pure” emphysema is a person who is “barrel-chested,” thin, puffing air, and not significantly hypoxic. These patients are referred to as “pink puffers.” Because they don’t usually become hypoxic early in the course of the disease, they often continue smoking.

2. Chronic Bronchitis – Defined as a persistent, productive cough for three consecutive months of at least two consecutive years. There is inflammation of the airways and hypersecretion of mucus and therefore airway size is compromised and obstructed. The leading cause is again cigarette smoking. As opposed to emphysema, with chronic bronchitis there is often retention of carbon dioxide and hypoxia. The classic patient here is obese, hypoxic, cyanotic, and edematous (from CHF) and is referred to as a “blue bloater.”

Treatment of these entities is similar to and uses most of the same medications as in asthma, with the notable exception of allergy shots, and often the addition of oxygen on a chronic basis. The airway obstruction here is not very reversible (unlike asthma where it is almost totally reversible). Also, these people tend to have additional medical conditions such as heart disease that must be managed to optimize their respiratory status. Most patients with COPD have anxiety, and this obviously will affect their treatment.