I. Restrictive Pulmonary Disorders
   a. Due to decreased lung expansion
   b. Etiological classification
      i. Parenchymal lung disorders: fibrotic interstitial lung disease, atelectatic disorders
      ii. Pleural space disorders → between parietal/viseral
      iii. Chest wall disorders: kyphoscoliosis, ankylosing spondylitis, flail chest, disorders of obesity → adding fat to chest wall increases pressure and decreases space to expand
      iv. Neuromuscular disorders
      v. Pulmonary infection/inflammation: pneumonia, severe acute respiratory syndrome, pulmonary tuberculosis

II. Fibrotic Interstitial Lung Disease
   a. Etiology = unknown
   b. Alveolar wall infiltration by cells, fluid and connective tissue
   c. Incidence: 5/100,000
   d. Acute (allergies, minutes) subacute, chronic
   e. Can progress to irreversible fibrosis if untreated
   f. Pathogenesis
      i. Immune reaction: initial injury to alveolar epithelium or capillary endothelium, interstitial and alveolar wall thickening, increased interstitial collagen bundles
      ii. Inflammation: early and reversible, triggers cause inflammation and increased inflammatory cells (injury increases membrane permeability and fluid/debris movement into alveoli)
         1. Stretching capiilary pores
         2. Mast cells, neutrophils, macrophages, eaosiniphils
      iii. Fibrosis: fibroblastic proliferation and collagen deposition due to increased interstitial mesenchymal cells and fibroblasts, alveolar walls thicken with increased fibrous tissue → cracking at alveoli and destruction will eventually kill the patient
      iv. Destruction: end stage disease, loss of alveolar walls
   g. Clinical manifestations
      i. Progressive dyspnea with exercise and desaturation
      ii. Rapid, shallow breathing
      iii. Irritating, dry cough
      iv. Nail bed clubbing, bibasilar (both bases of lungs) end-expiratory crackles
      v. Late cyanosis
      vi. Anorexia, weight loss → no energy
      vii. Inability to increase cardiac output with exercise
   h. Diagnosis
      i. Chest X-ray → honeycomb pattern
      ii. Open lung biopsy → surgery, take out the ribs and take a piece of the lung

iii. Transbronchial biopsy → tube goes down to pinch a piece of the lung
iv. Bronchoalveolar lavage → spray fluid which picks up fibroblasts, immuelalveolar cells, debris and analyze under a microscope

i. Treatment
    i. Smoking cessation, avoid environmental exposure
    ii. Anti-inflammatory and immunosuppressive agents
    iii. Lung transplant
j. Sarcoidosis
    i. Etiology: idiopathic
    ii. Systemic disorder → affects all tissues
        1. Acute: common in women 20-30 years old → autoimmune that doesn't involve Ab
        2. Chronic: 30-40 years old
    iii. Trigger causes alveolar macrophage activation
    iv. Pathogenesis
        1. Multiple, uniform, noncaseating epithelioid granulomas → spread all over and change function of tissues
            a. Noncasesating = solid
        2. Multiorgan: lymph nodes and lung*, skin, eyes, spleen, liver, kidney, bone marrow
        3. Fibrotic, surrounded by large histiocytes
        4. Abnormal T-cell function
    v. Clinical manifestations
        1. Malaise, fatigue, weight loss, fever → tired, weak muscles
        2. Insidious dyspnea, dry cough
        3. Erythema nodosum, macules/papules, hyperpigmentation, subcutaneous nodules → subcutaneous nodules, red rash
        4. Hepatosplenomegaly, lymphadenopathy
            a. englarged
    vi. Diagnosis
        1. Blood tests
            a. Higher BP from ACE
            b. Leukopenia, increased eosinophils, anemia,
            c. High ESR, increased Ca (5% cases), high liver enzymes
            d. Anergy: decreased sensitivity to specific Ag like Candida and mumps (70% cases) → false
                i. Anergy = w/out energy due to leukopenia
            e. High angiotensin converting enzymes: active disease (40-80% cases)
        2. Bronchoalveolar lavage: monitor cell content, fluid has high lymphocytes and high CD4:CD8 cell ratio
        3. Transbronchial lung biopsy: noncaseating granulomas (definitive diagnosis)
        4. Chest X-ray: differentiates stages (start with this)

vii. Treatment
    1. Corticosteroids and immunosuppressants

III. Atelectatic Disorders
a. ARDS → secondary to something else
    i. Acute/Adult Respiratory Distress Syndrome
        1. Associated with other pathophysiological processes
        2. 125 000-150 000 cases/year in U.S., 30-60% mortality
    ii. Etiology
        1. Severe trauma, sepsis (>40% cases), shock
        2. Aspiration of gastric acid: >30% cases
    iii. Pathogenesis
        1. Widespread pulmonary inflammation
            a. Noncardiogenic pulmonary edema: leaky pulmonary capillaries
            b. Atelectasia associated with lack of surfactant
            c. Fibrosis: inflammatory protein deposition
        2. Sever hypoxemia due to intrapulmonary blood shunting
            a. Perfusion of large number of alveoli that are poorly ventilated (low ventilation-perfusion areas) or not ventilated (areas of shunt)
        3. Decrease in lung compliance
            a. Due to surfactant loss/inactivation with subsequent increased recoil pressure
            b. Proteinaceous fluid fills alveoli and impairs ventilation
        4. Diffuse, fluffy alveolar infiltrates
    iv. Clinical manifestations
        1. Early
            a. Sudden, marked, respiratory distress
            b. Mild tachycardia, dyspnea, low PaO_2
            c. Shallow, rapid breathing
        2. Late
            a. Tachycardia, tachypnea, hypotension
            b. Marked restlessness, cyanosis
            c. Crackles and ronchi on auscultation, use of accessory muscles, intercostal and sternal retractions
    v. Diagnosis
        1. Hallmark: hypoxemia refractory to increased supplemental oxygen levels
        2. ABG: hypoxia, acidosis, hypercapnia (high carbon dioxide)
        3. Chest X-ray: progressive, normal to diffuse “whiteout”
        4. Open lung biopsy: atelectasis, hyaline membranes, cell debris, interstitial and alveolar edema
    vi. Treatment
        1. Treat the sepsis first so the body has time to heal