13

Pulmonary Disorders

■ I. CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

A. Definition. COPD is a disorder characterized by expiratory flow limitation that does not change markedly over periods of several months of observation. The term *COPD* includes the following:

1. Chronic Bronchitis
   Chronic bronchitis is a clinical diagnosis made when chronic cough with sputum production is present on most days for at least 3 months of the year for at least 2 consecutive years. Major pathologic findings include airways inflammation and enlargement of the submucosal mucus glands.
2. Emphysema
   Emphysema is defined pathologically as an abnormal permanent enlargement of the air spaces distal to the terminal bronchiole, accompanied by destruction of their wall without obvious fibrosis. Clinically, it correlates with a reduction in the diffusing capacity (DL_{CO}).
3. Various degrees of both chronic bronchitis and emphysema coexist in most patients with COPD. The term *COPD* should not be used for other forms of obstructive lung disease such as bronchiectasis, cystic fibrosis, or major airway obstruction.

B. Etiology and Risk Factors. The pathogenesis of most cases of COPD remains unclear. The main risk factor associated with COPD is cigarette smoking, but most smokers do not develop COPD. Less than 1% of patients with emphysema have alpha$_1$-antitrypsin deficiency (serum A1AT <5 μM; normal values 20–48 μM).

C. Diagnostic Evaluation

1. Clinical Presentation
   a. Cough, sputum production, and dyspnea that usually have been present for several years. Symptoms consistent with severe COPD in a young and/or nonsmoking adult should prompt the consideration of other conditions such as alpha$_1$-antitrypsin deficiency, uncontrolled asthma, or other less common causes of obstructive lung disease (i.e., cystic fibrosis, immotile cilia syndrome, Young's syndrome [obstructive azoospermia with chronic bronchitis/bronchiectasis], congenital or acquired immunoglobulin deficiency).

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Table 13.1. Common Conditions Associated with COPD Decompensation

Respiratory Infections: Viral upper and lower respiratory tract (i.e., pharyngitis, tracheobronchitis, pneumonitis), aspiration, and bacterial pneumonia
Narcotics and sedatives
Inappropriately high fraction of inspired O_2 (FiO_2) (mainly in "CO_2 retainers")
Heart failure
Excessive diuresis with metabolic alkalosis and compensatory CO_2 retention
Pneumothorax (rupture of a bleb)
Hypophosphatemia, hypomagnesemia
Hypermetabolic states (i.e., sepsis, fever)

b. During an *acute decompensation* of COPD, there is an increase in dyspnea and cough, and there are changes in sputum volume, color, and consistency. Physical examination may reveal
   (1). Pursed-lip breathing
   (2). Rapid shallow breathing
   (3). Use of respiratory accessory muscles (i.e., sternocleidomastoid, pectoralis, abdominal muscles)
   (4). Thoracoabdominal paradoxical breathing pattern
   (5). Wheezes, coarse crackles, and almost undetectable breath sounds in severe cases
   (6). Increased jugular venous distention, hepatomegaly, peripheral edema, and right-sided S_3 and increased P_2 sounds are characteristic of patients with cor pulmonale due to severe COPD
   (7). Various degrees of changes in mental status may be present and related to hypoxemia, hypercapnia, infection, and/or drugs
   Other conditions that are commonly associated with or precipitate a worsening of COPD patients are depicted in Table 13.1.

2. Laboratory Findings
   a. Pulmonary Function Testing (PFT)
      (1). *Spirometry* reveals an obstructive pattern: reduction in the ratio of forced expiratory volume in the first second to forced vital capacity (FEV_1/FVC ratio); normal for a 50-year-old person is 70%). The severity of the expiratory airflow limitation can be assessed by the FEV_1 (as percent of normal predicted according to sex, race, and height). Commonly used values to assign severity of functional impairment are based on the Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD criteria) shown in Table 13.2.
      (2). In *intubated, mechanically ventilated* patients, the *interrupter technique* can be used to diagnose airflow limitation and assess the improvement of expiratory flow in response to bronchodilators.

Table 13.2. Classification of Severity of COPD—The GOLD Criteria

Stage	*Physiological characteristics*
I: Mild COPD	FeV_1/FVC <70% FeV_1 ≥80% of predicted
II: Moderate COPD	FeV_1/FVC <70% 50%≤ FeV_1 <80% of predicted
III: Severe COPD	FeV_1/FVC <70% 30%≤ FeV_1 <50% of predicted
IV: Very severe COPD	FeV_1/FVC <70% FeV_1 <30% of predicted

b. Radiologic Studies
   (1). Chest x-ray may demonstrate evidence of emphysema:
      (a). Flattening of the diaphragm
      (b). Increased retrosternal air space
      (c). Irregular areas of vascular attenuation and radiolucency (bullae: lucent areas in the lung parenchyma >1–2 cm in diameter).
      (d). Typical smoker's emphysema is mainly of apical distribution. Predominant lower lung zones changes are consistent with emphysema due to $alpha_1$-antitrypsin deficiency.
   (2). Computed tomography (CT) of the chest is the most sensitive way to detect emphysema, although it is not routinely recommended as an initial diagnostic test.
   (3). The roentgenographic features of chronic bronchitis are *nonspecific* and may include increased lung markings ("dirty lungs") and thickening of bronchial walls.
   (4). Chest x-ray during an acute COPD exacerbation can be helpful in the detection of associated processes such as pneumonia, atelectasis, or pneumothorax.
c. Arterial Blood Gases (ABGs)
   (1). Various degrees of *hypoxemia* with increased $P(A\text{-}a)O_2$ gradient are typical of COPD patients.
   (2). Chronic hypercapnia with compensatory metabolic alkalosis is seen in severe cases ("CO_2 retainers").
   (3). Finding chronic CO_2 retention in moderate COPD with FEV_1 >1–1.3 L is unusual and should raise the question of concomitant neuromuscular or sleep apnea disorders.
   (4). Common acid–base disturbances seen during an acute exacerbation of COPD include
      (a). Acute respiratory acidosis.
      (b). Partially compensated respiratory acidosis (acute-on-chronic).
      (c). Chronic respiratory acidosis (mild exacerbation in "CO_2 retainers").
      (d). Metabolic alkalosis induced by diuretics or continuous nasogastric aspiration may be a cause of persistent or worsening hypercapnia in COPD.

D. Management of Acute COPD Exacerbation
1. Ensure Adequate Oxygenation and Ventilation
   a. For most patients, the goal is to maintain a PaO_2 of 55–60 mmHg (arterial oxyhemoglobin saturation of 88–90%). In patients with concomitant coronary artery disease, an arterial saturation >90% is desirable.
      (1). *Spontaneously breathing patients* with acute COPD exacerbation can usually achieve those levels using a Venturi mask set to deliver 24–35% O_2 (preferred in "mouth breathers") or a nasal cannula with an O_2 flow of 1–2 L/min. (See Chapter 2, "The Basics of Critical Care.")
      (2). Some COPD patients will develop or worsen hypercapnia during O_2 therapy. A reduction of the hypoxic respiratory drive and a worsening of V/Q mismatch are the underlying mechanisms thought to mediate that response.
   b. Patients with significant acidemia, inadequate PaO_2, hypercapnia with changes in mental status, or hemodynamic instability should be assisted with mechanical ventilation (MV).
      (1). Noninvasive positive-pressure ventilation (NIPPV) as the initial form of ventilatory assistance has been reported effective in selected patients with acute respiratory failure. Candidates for NIPPV should
         (a). Tolerate a facial or nasal mask.
         (b). Cooperate with this form of therapy.
         (c). Have an intact upper airway function without excessive secretions, regurgitation, or vomiting.
         (d). Be hemodynamically stable.
         (e). It may also be offered to patients requiring endotracheal intubation who decline invasive procedures.
         NIPPV can be administered using a volume-cycled or pressure-controlled ventilator (i.e., BiPAP in S/T mode inspiratory positive airway pressure [IPAP] 10 cmH_2O, expiratory positive airway pressure [EPAP] 5 cmH_2O, rate 10 beats per minute). Close observation with ABGs and continuous monitoring of arterial O_2 saturation (SaO_2) are recommended to determine NIPPV efficacy and to avoid delays in endotracheal intubation.
      (2). When volume-cycled MV is instituted in intubated COPD patients, a major goal is to minimize dynamic hyperinflation (auto-positive end-expiratory pressure [PEEP]) and its hemodynamic consequences. In general, the ventilator should be set to lower the mean expiratory flow (VT/Te) through increases in inspiratory flow (i.e., 90 L/min) and expiratory time (reductions in machine rate, I:E ratio, or even sedation that leads to failure to trigger) and reductions in tidal volume (i.e., 6 mL/kg).
2. Bronchodilators
   a. Inhaled $Beta_2$-Agonists: When delivered by metered dose inhalers (MDI), these drugs are as effective as nebulized in intubated or spontaneously breathing patients (used with a spacer device). Albuterol (Proventil, Ventolin) 2–4 puffs may be administered initially q20 min × 3, followed by q1–2 h until improvement occurs, and then q4–6 h. The dose for albuterol nebulization is 2.5 mg (0.5 cc of 0.5% solution in 2–3 cc of normal saline).
   b. Anticholinergics: Ipratropium bromide (Atrovent) has shown to be as effective as $beta_2$-agonists with potentially fewer side effects. Ipratropium bromide should be added to inhaled albuterol during COPD exacerbations.

3. Corticosteroids
   Unconfirmed trials have shown benefits from administration of steroids in acute COPD exacerbations. Methylprednisolone (Solu-Medrol) 0.5 mg/kg IV q6 h or prednisone 40–60 mg/d PO for 3 days and then tapered over a 2-week period is recommended.
4. Antibiotics
   A recognized upper or lower respiratory infection should be treated adequately. The initial antibiotic regimen should target likely bacterial pathogens (*H. influenzae, M. catarrhalis,* and *S. pneumoniae* in most patients) and take into account local patterns of antibiotic resistance Empiric antibiotic therapy (i.e., trimethoprim-sulfamethoxazole, levofloxacin, doxycycline, or amoxicillin for 7–10 days) in acute COPD exacerbation has been associated with an earlier resolution and fewer relapses.
5. Correct precipitating or associated problems (Table 13.1).

■ II. ASTHMA

A. Definition
   1. Asthma
      Asthma is a clinical syndrome characterized by increased responsiveness of the tracheobronchial tree to a variety of stimuli with slowing of forced expiration that changes in severity either spontaneously or as a result of therapy.
   2. Status Asthmaticus
      Status asthmaticus is a severe episode of asthma that does not respond to usually effective treatment requiring more aggressive therapy for reversal.

B. Pathophysiology. The key feature of asthma is *airway inflammation* with hyperresponsiveness leading to airway obstruction and in severe cases to hyperinflation, increased VD/VT, and V/Q mismatch, with subsequent hypoxemia and respiratory insufficiency.

C. Diagnostic Evaluation
   1. Clinical Presentation
      Dyspnea, wheezing, and coughing are the most common symptoms during an asthma attack. Other diagnostic considerations, especially when a prior history of asthma is absent, should include the following:
      a. Heart failure and ischemia with diastolic dysfunction
      b. Aspiration of foreign bodies
      c. Epiglottitis and croup
      d. Pulmonary embolism (rare)
      Table 13.3 shows several adverse prognostic indicators obtained by history, physical examination, and routine tests in acute life-threatening asthma.
   2. Laboratory Evaluation
      a. Spirometry: Bedside spirometry shows an obstructive pattern (see "COPD," section C2a). Serial FEV_1 determinations are indicated to objectively evaluate the response to treatment. If spirometry is not available, monitoring peak expiratory flow using a peak flowmeter is recommended.
      b. Arterial Blood Gases: Hypoxemia may be seen in cases complicated by respiratory failure, pneumonia, or pneumothorax. The most common acid–base

Table 13.3. Factors Associated with Severe Acute Asthma Attacks

Previous episode(s) of severe asthma (especially if associated with respiratory failure)
Changes in mental status
Use of accessory muscles of respiration
Very diminished or absent breath sounds
Pulsus paradoxus >10 mmHg
Tachycardia >130 beats per min
Cyanosis
Hypoxemia
Hypercapnia or normocapnia in the setting of tachypnea
FEV_1 <20% predicted

abnormality is acute respiratory alkalosis. *Normocapnia* or *acute respiratory acidosis* indicates impending or established respiratory failure.

c. Chest X-Ray: May show evidence of hyperinflation, increased bronchial markings, or associated conditions such as pneumonia or pneumothorax.
d. Other Tests: In addition to the usual admission tests, theophylline level and blood and sputum cultures should be done if clinically indicated.

D. Management of Asthma Attacks

1. Ensure Adequate Oxygenation
   a. Most asthma patients will maintain SaO_2 >90–92% during an acute attack with a low concentration of supplemental O_2 (Venturi mask or nasal cannula 2 L/min). Monitor patient with pulse oximeter and supplement O_2 as necessary.
   b. Mechanical Ventilation: Few patients with severe asthma will not respond to aggressive medical management and will require ventilatory support. The ventilatory strategy in patients with severe airway obstruction should provide adequate oxygenation and at the same time minimize the risk of barotrauma through the use of small tidal volumes (i.e., 5–8 mL/kg) and minute ventilation (even if $PaCO_2$ is allowed to climb: "controlled hypoventilation"). As in the case of COPD with expiratory flow limitation, reducing the Ventilator's mean expiratory flow (VT/Te) will improve air trapping and its deleterious effects.
2. Beta-Adrenergic Agonists
   Beta-adrenergic agonists are first-line therapy for acute asthma episodes. Selective $beta_2$-agonists such as albuterol and terbutaline administered by MDI with a holding chamber or by nebulization titrated to maximum effect are preferred. For dosing see COPD, section D2a. When drug delivery by aerosol is inadequate, SC epinephrine (0.3 mL 1:1,000 q20 min × 3 max.) or terbutaline (0.25 mg q20 min × 2 max.) can be used.

3. Anticholinergic agents
   Ipratropium bromide 500 mcg by nebulizer every 20 min, for 3 doses during acute asthma attack, then as needed.
4. Corticosteroids
   Methylprednisolone (Solu-Medrol) 40 mg q6 h IV or prednisone 60 mg orally q8 h are recommended for the first 36–48 h. Significant clinical benefits are usually present 6 h later. When the patient is stable, a prednisone-tapering program may consist of 60 mg/d for 4 days, reducing the dose to 40 mg/d and then by 10 mg/d every 4 days. At the same time, the patient should be started on inhaled corticosteroids (i.e., triamcinolone acetonide [Azmacort] 6–8 puffs bid).
5. Complicating Factors
   Treat any obvious associated precipitant or complicating conditions such as pneumonia and pneumothorax.
6. Other Forms of Therapy
   Other interventions that have been used in status asthmaticus but are not considered standard therapy include magnesium sulfate, general anesthetics, and bronchial lavage of thick secretions.

■ III. PULMONARY EMBOLISM

A. Clinical Presentation and Risk Factors
1. The clinical findings of pulmonary embolism (PE) are nonspecific. It most commonly presents as the acute onset of dyspnea with or without pleuritic chest pain, minor hemoptysis, tachypnea, and abnormal chest x-ray (although a normal chest x-ray is not uncommon either). Other forms of presentation include the following:
   a. Acute cor pulmonale (>40% of circulation compromised)
   b. Insidious onset of dyspnea (recurrent unrecognized PEs)
   c. Syncope, wheezing, fever, cough, dysrhythmias, and cardiopulmonary arrest.
   d. Asymptomatic
2. PE originates from thrombi in the deep venous system of the lower extremities (deep venous thrombosis [DVT]) in most cases. Important risk factors for venous thromboembolism include the following:
   a. Prolonged immobility or paralysis.
   b. Surgery (mainly orthopedic—hip, knee—and lengthy procedures).
   c. Trauma.
   d. Malignancy.
   e. Congestive heart failure (CHF), recent myocardial infarction (MI).
   f. Advanced age.
   g. Obesity.
   h. Pregnancy and estrogen therapy.
   i. Prior history of DVT/PE: Less often, DVT/PE is caused by antithrombin III, protein S and protein C deficiencies, or lupus anticoagulant syndrome.
3. Chest x-ray abnormalities may be subtle or even absent.
   a. Pulmonary Infiltrates
      (1). Only a minority represent pulmonary infarction, and they usually resolve over few days

(2). A pleural-based triangular infiltrate (Hampton's hump) may be seen with infarction. It usually persist for weeks.
   b. Pleural effusion(s)
   c. Elevated hemidiaphragm
   d. Platelike atelectasis
   e. Oligemia (Westermark's sign)
4. Arterial Blood Gases
   a. Hypoxemia in most cases (but 15% of PEs have PaO_2 >80 mmHg)
   b. $P(A\text{-}a)O_2$ gradient widened
   c. Hypocapnia
5. D-dimer
   This test has a good sensitivity and negative predictive value, but poor specificity and positive predictive value.
6. Electrocardiogram (ECG)
   a. Nonspecific QRS and ST-T changes
   b. Sinus tachycardia
   c. Atrial dysrhythmias (multifocal atrial tachycardia [MAT], atrial flutter)
   d. S1-Q3-T3 pattern (only 10% of cases)
   e. Pulseless electrical activity in massive PE
7. Echocardiography
   Up to 40% of patients with PE have echocardiographic anomalies. Some of these anomalies are
   a. Increase in RV size
   b. Decreased RV function
   c. Tricuspid regurgitation
   d. RV Thrombus

B. Diagnostic Tests
1. Ventilation/Perfusion (V/Q) Scan
   A normal V/Q scan practically rules out pulmonary embolism. On the other hand, an abnormal V/Q scan is nonspecific and should be considered in the context of the clinical probability (see Figure 13.1). The V/Q scan and simultaneous chest x-ray findings are categorized as normal/very low, low, intermediate, or high probability, as depicted in Table 13.4 .
2. Lower Extremities Venous Studies (LEs)
   a. Duplex Ultrasound (DU): DU is Doppler ultrasound combined with real-time two-dimensional ultrasound to study the venous system. When available, it is the method of choice for diagnosing proximal DVT (positive predicted value of 94%). Diagnostic criteria of DVT include the inability to collapse the vein and to visualize the clot. DU can also assess flow augmentation, valvular incompetence, and other causes of pain and swelling (i.e., popliteal cysts and hematomas).
   b. Impedance Plethysmography (IPG): Rarely used anymore, IPG determines the changes in electrical impedance of the calf in response to blood volume changes produced by inflating a pneumatic thigh cuff. It is very sensitive for occlusive proximal DVT but insensitive for calf vein thrombosis.
   c. Venography: Once considered the gold standard for the diagnosis of leg DVT, is nowadays less frequently used. Disadvantages include its invasiveness, cost, and potential allergic reactions to contrast media. Definitive diagnostic findings include filling defects in a well-opacified vein and/or partially occluding defects surrounded by contrast media.

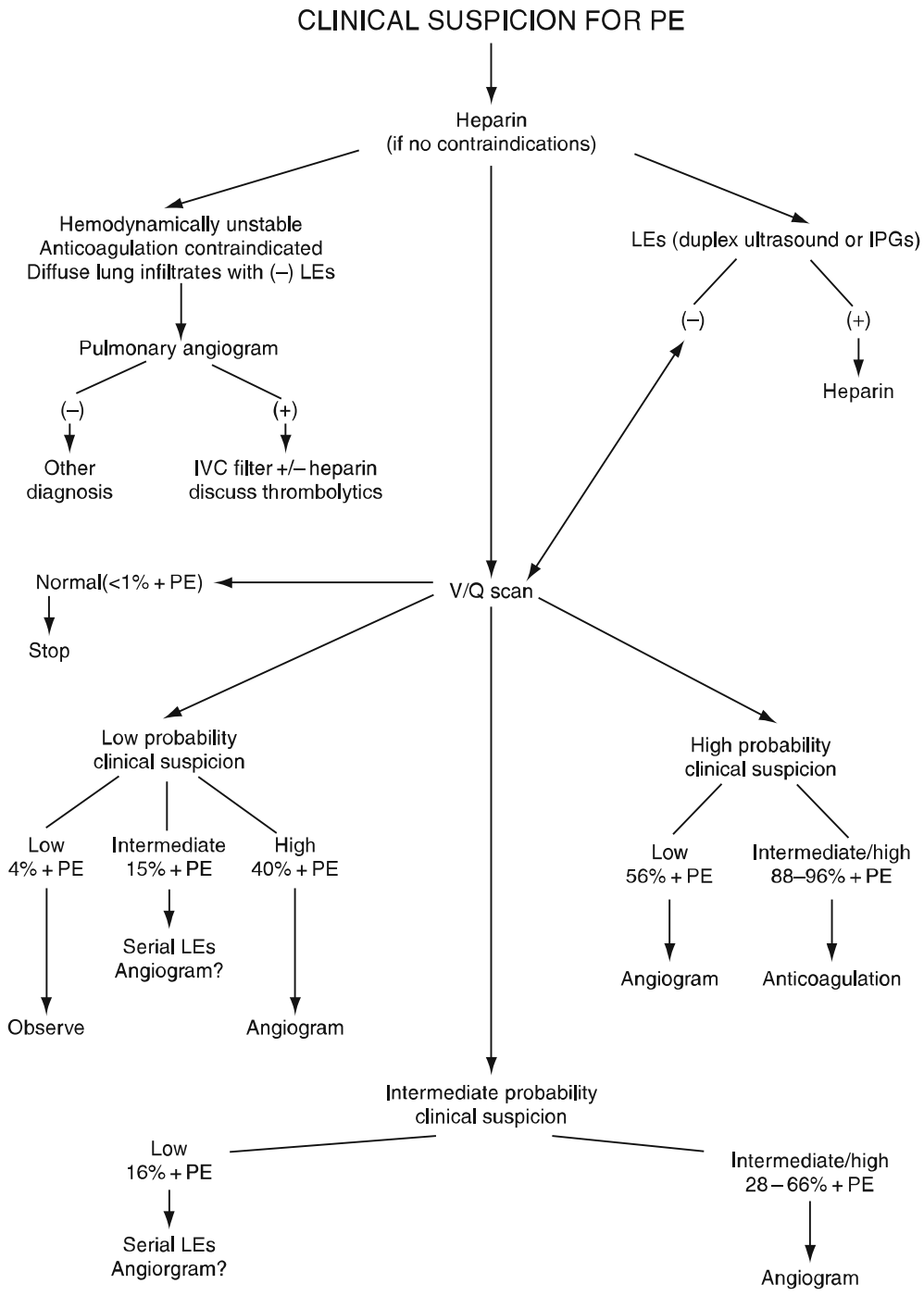

Figure 13.1. Diagnostic/treatment approach to pulmonary embolism.

3. Spiral CT with intravenous contrast
   This test has the ability to detect alternative pulmonary abnormalities that may explain patient's condition. The diagnosing accuracy varies from institution to institution, depending on the film's quality and radiologist's experience. When these two conditions are favorable, a CT is a very good diagnostic tool.

Table 13.4. V/Q Scan Interpretation Categories

Normal and very low probability
- No perfusion defects present (ventilation study and/or chest x-ray may be abnormal)
- Three small segmental (<25% of a segment) perfusion defects with normal chest x-ray

Low probability
- >Three small segmental perfusion defects with normal chest x-ray
- Large or moderate segmental perfusion defect involving no >4 segments in one lung and no >3 segments in one lung region with matching ventilation defects and chest x-ray normal or with abnormalities smaller than the perfusion defects
- Nonsegmental perfusion defect (small pleural effusion, cardiomegaly, enlarged aorta, mediastinum, hila)
- One moderate segmental (>25 to <75%) perfusion defect with normal chest x-ray mismatch)

Intermediate probability
- All V/Q scans not included in the above categories (borderline or difficult to categorize)

High probability
- Two large segmental (>75%) perfusion defects without corresponding ventilation or chest x-ray abnormality or smaller ventilation or chest x-ray abnormalities (mismatch)
- Two moderate segmental and one large segmental perfusion-ventilation
- Two moderate segmental and one large segmental perfusion-ventilation mismatches
- Four moderate segmental perfusion-ventilation mismatches

4. Pulmonary Angiography
   Still considered the gold standard for the diagnosis of pulmonary embolism. In general, pulmonary arteriography is a safe procedure (mortality <0.2%, morbidity 4%), even in patients with significant pulmonary hypertension. Definitive angiographic signs include the presence of intraluminal filling defects or cutoffs of pulmonary arteries. It is indicated in patients with suspected PE and
   a. Contraindications for anticoagulation (considering IVC filter)
   b. Hemodynamic instability (considering thrombolytic therapy or embolectomy)
   c. High clinical suspicion for PE with other than high-probability V/Q scan and negative leg venous studies
   d. Extensive pulmonary parenchymal disease or CHF

C. Treatment of Acute Thromboembolism
   1. Anticoagulation
      Anticoagulation should be started as soon as the clinical suspicion for PE is high enough to initiate a diagnostic evaluation.
      a. Heparin: Give an initial bolus of 10,000 U intravenously followed by a continuous infusion of 1,300 U/h (20,000 U in 500 mL D_5W at 33 mL/h). Check

the first activated partial thromboplastin time (aPTT) in 6 h, and maintain it between 1.5 and 2.5 times control. Modify heparin infusion according to the following:

(1). If aPTT >2.5 control, stop infusion for 1 h, reduce the dose by 100–200 U/h, and recheck it in 4–6 h.

(2). If aPTT is between 1.25 and 1.5 control, increase the dose by 100 U/h, and recheck it in 4–6 h.

(3). If aPTT is <1.25 control, rebolus with 5,000 U IV, increase the dose by 200 U/h, and recheck it in 4–6 h.

(4). Weight-based protocols are available in most hospitals

In most patients, continue heparin for at least 5 days (provided that Coumadin was started on day 1 or 2). Seven to ten days of heparin infusion is recommended for patients with massive PE or extensive iliofemoral thrombosis.

Low molecular weight heparin (1 mg/kg IBW q12 h) has been used instead of unfractionated heparin in PE with excellent results (see Chapter 7, "Hematological Disorders").

b. Coumadin: Oral anticoagulation started on day 1 or 2 at a dose of 5–10 mg/d is recommended. The goal is to prolong the prothrombin time (PT) to an International Normalized Ratio (INR) of 2.0–3.0. Coumadin should be continued for at least 3–6 months in most patients. For those with a continuing risk factor or recurrent thromboembolism, anticoagulation should be given indefinitely. In cases where Coumadin may be contraindicated (i.e., pregnancy during the first and third trimester), adjusted-dose SC heparin can be effectively used.

2. Thrombolytic Therapy

Thrombolytic drugs dissolve thrombi by activating plasminogen to plasmin, which in turn degrades fibrin (see Chapter 7, "Hematologic Disorders"). In contrast to thrombolytic therapy for MI, complete emboli resolution in the pulmonary vessels is not accomplished frequently. Although a reduction in PE mortality has not been shown with this form of therapy, it should be considered in patients with acute massive PE and hemodynamic instability without significant risk factors for bleeding. The role of thrombolytic therapy in DVT and submassive PE is less well established. Agents used for PE/DVT include the following:

a. Streptokinase (SK): 250,000 IU loading dose followed by 100,000 IU/h for 24 h in PE and 48–72 h in DVT.

b. Urokinase (UK): 4,400 IU/Kg loading dose followed by 4,400 IU/kg/h for 12 h in PE and 24–48 h in DVT.

c. Tissue Plasminogen Activator (tPA): 100 mg over 2 h.

The efficacy and bleeding complications of SK, UK, and tPA are equivalent. When using SK or UK, checking a thrombin time or an aPTT 2–4 h into the infusion is recommended to verify a fibrinolytic state. Heparin should be restarted when the aPTT is <2 times control.

3. IVC Filter

Indications for placement of an IVC filter (Greenfield, Mobin-Uddin, Bird's nest) include the following:

a. Contraindications to anticoagulation

b. Acute massive PE

c. Recurrent PE on adequate anticoagulation therapy

d. Chronic thromboembolism with pulmonary hypertension

e. Following pulmonary embolectomy or thromboendarterectomy

4. Embolectomy

Embolectomy may be considered for documented massive PE with documented occlusion of main pulmonary artery.

Catheter embolectomy: Intrapulmonary arterial techniques, using rheolytic embolectomy catheter (i.e., AngioJet embolectomy system), have been used with success in some cases. This procedure has been called by some the Varon-Strickman procedure.

Surgical embolectomy: This procedure is advised only if an experienced surgical team is immediately available.

■ IV. ADULT RESPIRATORY DISTRESS SYNDROME (ARDS)

A. Definition. ARDS is a form of acute lung injury characterized by a high-permeability (noncardiogenic) pulmonary edema. In clinical practice, it is defined by the presence of the following:

1. Acute respiratory distress in a patient with predisposing conditions
2. Diffuse bilateral infiltrates on chest x-ray (pulmonary edema pattern)
3. Hypoxemia (PaO_2 <55 mmHg with FiO_2 >0.5)
4. Reduced respiratory system static compliance (<40–50 mL/cmH_2O)
5. Low or normal pulmonary artery occlusion pressure (pulmonary capillary wedge pressure [PCWP] <16 cmH_2O)

B. Etiology. ARDS is most commonly associated with

1. Sepsis
2. Bronchial aspiration of gastric content
3. Trauma
4. Nosocomial pneumonia

Major risk factors for the development of ARDS are listed in Table 13.5.

Table 13.5. Conditions Associated with ARDS

Air embolism
Aspiration of gastric contents
Burns
Cardiopulmonary bypass
Disseminated intravascular coagulation
Drugs (cocaine, heroin, methadone, acetylsalicylic acid)
Multiple fractures (fat embolism)
Multiple transfusions
Near drowning
Pancreatitis
Pneumonia (bacterial, viral, fungal)
Prolonged hypotension
Sepsis
Toxin inhalation
Trauma

C. Pathophysiology. The basic abnormality in ARDS is the disruption of the alveolar-capillary barrier. The endothelial injury in ARDS is frequently part of a more generalized permeability defect. An initial exudative phase is followed by proliferation of type II pneumocytes and fibrosis seen as early as the end of the first week.

D. Clinical Presentation
1. ARDS may develop insidiously over hours or even days after the initiating insult (i.e., pneumonia evolving into ARDS). Occasionally, it coincides with the precipitating event (i.e., gastric contents aspiration).
2. The signs and symptoms of ARDS are specific and usually include
   a. Dyspnea
   b. Tachypnea (rapid shallow breathing)
   c. Coarse lung crackles
   d. Cyanosis
   e. Agitation
3. Systemic manifestations of other organ dysfunctions may be related to the precipitating cause (i.e., burn, trauma) or may represent the generalization of the inflammatory response:
   a. Disseminated intravascular coagulation
   b. Encephalopathy
   c. Acute renal failure
   d. Acute liver failure
   e. Sepsis (gut bacterial translocation)
4. ABGs show marked hypoxemia and hypocapnia with either acute respiratory alkalosis or acute metabolic acidosis.
5. Despite the chest x-ray appearance of diffuse bilateral infiltrates, chest CT reveals a patchy, nonhomogeneous distribution of affected lung mixed with normal parenchyma. Small pleural effusions can be seen in ARDS. Usually, the cardiovascular silhouette on chest x-ray is within normal limits.
6. The pulmonary artery occlusion pressure or wedge pressure measured by a balloon-tipped, flow-directed catheter (Swan-Ganz catheter) in the past was used to detect the hydrostatic component of the pulmonary edema (cardiogenic). In pure ARDS, the wedge pressure should be <16–20 cmH_2O.

E. Management
1. Treatment of the Precipitating Condition(s)
   Specific treatment for the underlying disorder should be instituted as soon as possible (i.e., antimicrobials for infections, sepsis; drainage of abscesses, transfusion for hypovolemic shock, etc.).
2. Supportive Care
   a. Ventilatory Support (see Section V, below)
   b. Hemodynamic Monitoring and Support
      (1). The use of pulmonary artery catheter (PA catheter or Swan-Ganz catheter) is controversial, as no study has ever proved that this technique improves survival in suspected or established ARDS. However, the information derived from hemodynamic monitoring using this catheter can be used in the following:
         (a). Differentiation of cardiogenic vs. noncardiogenic pulmonary edema.
         (b). Management of intravascular volume (avoiding volume overload).

(c). Assessment of the cardiovascular effects of PEEP titration (cardiac index, stroke volume).
(d). Unfortunately, overtreating or confusing parameters is common while using a PA catheter.
(2). In severe cases of ARDS, where high levels of extrinsic PEEP or dynamic hyperinflation (auto-PEEP) are necessary to maintain adequate oxygenation, a reduced cardiac index should be corrected with the use of inotropes (i.e., dobutamine or dopamine) to maintain an adequate O_2 delivery.

c. Nutritional Support (see Chapter 10, "Nutrition")
d. Diagnosis and Treatment of Complications
(1). Barotrauma (i.e., tube thoracostomy for pneumothorax)
(2). Acute renal failure (i.e., hemodialysis)
(3). DIC (i.e., transfusions)
(4). Infections: line sepsis, urinary tract infection (UTI), cellulitis (i.e., antibiotics, change central lines)

3. Other Therapeutic Modalities
a. *Pharmacologic and immunologic agents* targeted to arrest a specific step in the inflammatory cascade or pathophysiologic process characteristic of ARDS and sepsis have been extensively evaluated without success: i.e., monoclonal antibodies against bacterial lipopolysaccharide and tumor necrosis factor (TNF), soluble interleukin-1 (IL-1) and TNF receptors, prostaglandin E1, pentoxifylline, nonsteroidal anti-inflammatory drugs (NSAIDs) (i.e., ibuprofen), synthetic surfactant mixtures (Exosurf), inhaled nitric oxide, etc.
b. *Extracorporeal oxygenation and CO_2 removal* (IVOX, $ECCO_2R$, ECMO), as currently implemented in some centers.

F. Prognosis
1. The mortality of ARDS has remained unchanged over the past two decades in spite of advances in supportive therapy.
2. Early mortality is usually related to the underlying condition(s); later, mortality is mainly related to multiple organ failure rather than pulmonary dysfunction.
3. Most *ARDS survivors* surprisingly have minimal long-term impairment of lung function (mild restrictive and diffusion capacity [DLCO] defects). Occasionally, reversible airway obstruction may develop).

■ V. ACUTE RESPIRATORY FAILURE

A. Definition. Acute respiratory failure is the inability to maintain adequate blood oxygenation and/or alveolar ventilation in the absence of an intracardiac shunt. Provided the baseline ABGs are close to predicted normal values, this usually means an acute increase in $PaCO_2$ >50 mmHg with arterial acidemia and/or a PaO_2 <55 mmHg while breathing room air.

B. Classification and Etiology. Two clinical and pathophysiologic distinct types of acute respiratory failure can be described:
1. Hypoxemic Respiratory Failure
The hallmark of this type of respiratory failure is the inability to adequately oxygenate the blood. The main pathophysiologic mechanisms involved are

V/Q mismatch (response to 100% O_2) and intrapulmonary shunting (no significant improvement with 100% O_2). The patients exhibit a rapid shallow breathing pattern and a low or normal $PaCO_2$. This form of respiratory failure is commonly the result of a diffuse acute lung injury with high-permeability pulmonary edema (ARDS), severe pneumonic infiltrates, or cardiogenic pulmonary edema.

2. Hypercapnic Respiratory Failure (Pump Failure)
   The hallmark of ventilatory pump failure is hypercapnia with acute respiratory acidosis. The $P(A\text{-}a)O_2$ gradient is useful in determining if the hypoxemia present in this form of respiratory failure is due to hypoventilation (normal gradient) only or to additional parenchymal lung disease (elevated gradient). The hypercapnia is the result of abnormalities in one or more of the determinants of the $PaCO_2$

$$PaCO_2 = kVCO_2/VE(1 - VD/VT)$$

   a. *Increased CO_2 production (VCO_2)* in patients with fever, sepsis, agitation, or excessive carbohydrate load, associated with a limited ventilatory capacity (high VD/VT, low VE)
   b. *Increased dead space (VD/VT)* in severe COPD, cystic fibrosis, and severe asthma
   c. *Decreased total minute ventilation (VE)* due to ventilatory pump dysfunction:
      (1). Decreased Central Respiratory Drive: CVA, drugs (narcotics, sedatives, anesthetics), central hypoventilation, hypothyroidism
      (2). Abnormal Respiratory Efferents
         (a). Spinal Cord: trauma, poliomyelitis, amyotrophic lateral sclerosis, tetanus, rabies
         (b). Neuromuscular: myasthenia gravis, multiple sclerosis, botulism, Guillain-Barré syndrome, hypophosphatemia, hypomagnesemia, drugs (streptomycin, amikacin, neuromuscular blockers), polyneuropathy of critical illness, bilateral phrenic nerve injury
      (3). Abnormal Chest Wall and/or Muscles: severe kyphoscoliosis, ankylosing spondylitis, massive obesity, muscular dystrophy, polymyositis, respiratory muscles fatigue, acid maltase deficiency
      (4). Airways, Upper Airway Obstruction: epiglottitis, fixed and variable upper airway obstruction due to tumors, post-extubation, tracheomalacia, bilateral vocal cord paralysis

C. Management. The management of acute respiratory failure is initially supportive, aimed at the correction of hypoxemia or hypercapnia until specific actions are implemented to correct, if possible, the factors that lead to the respiratory failure (i.e., antibiotics for pneumonia; diuretics, morphine, nitroglycerin, and afterload-reducing agents for cardiogenic pulmonary edema; naloxone for narcotics overdose).
   1. Hypoxemic Respiratory Failure
      a. Patients with V/Q mismatch abnormalities without significant intrapulmonary shunt will usually respond to noninvasive O_2 supplementation (i.e., nasal cannula, Venturi mask).

b. In patients with cardiogenic pulmonary edema, the use of continuous positive airway pressure (CPAP) (5–10 cmH_2O) via a face mask in addition to O_2 supplementation can be beneficial by reducing the transmural pressure of the left ventricle, and therefore afterload, but also by decreasing the preload.
c. The ventilatory management of patients with diffuse acute lung injury (i.e., ARDS) requires mechanical ventilation and should be viewed as a balance between *adequate oxygenation* on one hand, and the *risk for barotrauma* and *cardiovascular compromise* on the other. The following section pertains to specific aspects of the ventilatory management of ARDS.
   (1). Adequate Oxygenation: For most patients with ARDS, this means a PaO_2 of 55–60 mmHg or O_2 saturation of 88–90% with a cardiac index >2.5 L/min/m^2 and hemoglobin 10 g/dL. Arterial O_2 saturation can be increased in ARDS by the following:
      (a). *Raising the fraction of inspired oxygen (FiO_2)*: To avoid potential O_2 toxic effects, it is recommended not to use 100% O_2 for more than a few hours and to maintain an FiO_2 $\leq$0.6. A particular effort should be made to decrease FiO_2 to the minimum acceptable in patients exposed to drugs that may increase O_2 toxicity (i.e., bleomycin, amiodarone).
      (b). *Increasing the end-expiratory lung volume* to recruit collapsed or flooded alveoli. This can be achieved by adding *extrinsic PEEP* and/or setting the ventilator to create *dynamic hyperinflation (auto-PEEP)*. It is unclear if one strategy is more effective than the other in ARDS. The goal is to maximize oxygenation while at the same time *avoiding* hypotension, reduced cardiac pump function and a *plateau pressure >35 cmH_2O*. With these considerations
         i. *Extrinsic PEEP* is usually started at a level of 5 cmH_2O and titrated up by 2 cmH_2O increments to a level of 15–20 cmH_2O along with the use of other strategies to minimize barotrauma (see "Avoiding Barotrauma," below).
         ii. *Extended-ratio ventilation* (prolonged I:E ratio, known as inverse ratio ventilation when I/E >1:1) is a technique used to increase mean alveolar pressure and transpulmonary pressure. It can be implemented with either volume–controlled or pressure-controlled ventilators. Increasing the inspiratory time (see Table 13.6) increases the mean airway pressure (MAP) and allows the recruitment of lung units with long time constants, therefore, improving oxygenation. Extended-ratio ventilation is more easily implemented with volume–controlled than pressure–controlled ventilators. Both require heavy sedation with or without paralysis; i.e., morphine sulfate, midazolam (Versed), lorazepam (Ativan), or propofol (Diprivan) by continuous infusion with or without cisatracurium (Nimbex). Monitoring of plateau pressure to keep it <35 cmH_2O, auto-PEEP (end-expiration occlusion method).
   (2). *Avoiding barotrauma:* Barotrauma in the form of extra-alveolar air or worsening of acute lung injury is the result of alveolar overdistention (increased transmural pressure or alveolar pressure [P_{alv}]—pleural pressure [P_{pl}]). Thus, it seems reasonable to avoid lung volumes above total lung capacity (TLC) to prevent lung damage.

Table 13.6. Prolonging Inspiratory Time (Ti) and I/E Ratio in Volume-Controlled Ventilators*

Reduce inspiratory flow (i.e., to 40 L/min)
Use decelerating inspiratory flow waveform
Add an inspiratory pause (i.e., 0.2–0.5 s)
Increase the percent inspiratory time†

* Applying these changes in a stepwise manner will allow progressive extension of Ti and I:E ratio to the degree that is tolerated or needed. Adjustment in tidal volume (VT) as recommended (see below) and monitoring the plateau pressure (Pplateau), auto-PEEP, and its hemodynamic effects are required to avoid complications.

† Direct way of setting the I:E ratio (i.e., Siemens Servo Ventilator).

(a). Because P_{alv}, P_{pl}, and lung volumes are difficult to determine at the bedside, monitoring the *plateau pressure* (*end-inflation hold pressure*) is recommended as the best approximation of the peak alveolar pressure.

(b). A plateau pressure of 35 cmH_2O or more, in the absence of significantly decreased chest wall compliance, should be avoided.

(c). Peak airway pressure (Ppeak) reflects not only the elastic but also the flow-resistive pressures of the respiratory system, and it should be used only as a gross estimate for the risk of barotrauma (i.e., high Ppeak may be due to a small endotracheal tube, bronchospasm, secretions, high inspiratory peak flow, or worsening of lung or chest wall compliance) that may or may not mean alveolar overinflation.

(d). Determining the tidal volume (VT): ARDS is a nonhomogeneous process with collapsed and flooded areas mixed with relatively normal aerated lung, resulting in a reduction in the TLC. Thus, it makes sense to ventilate ARDS patients with smaller than conventional VT. The VT chosen should be one that prevents lung overinflation (i.e., plateau pressure <35 cmH_2O) and alveolar derecruitment at the end of expiration (inadequate oxygenation). This usually means an initial VT of 5–6 mL/kg.

(e). Setting the respiratory rate: The machine rate should be determined considering the patient's metabolic demands, intrinsic rate, and desired I:E ratio. It is usually set between 25 and 40 breaths per minute. Even with these rates (plus the low VT used), minute ventilation may not be high enough for ARDS patients. Allowing CO_2 retention (i.e., 60 mmHg) and respiratory acidemia (i.e., pH 7.2–7.25) in an effort to limit barotrauma is referred as *permissive hypercapnia.*

(3). *Controlling the hemodynamic effects of mechanical ventilation:* The increase in end-expiratory lung volume and mean alveolar pressure produced by the ventilatory strategies described above can have deleterious hemodynamics consequences. It is important to document that a ventilator change aimed at increasing O_2 saturation does not reduce the total amount of O_2 delivered to the tissues via a reduction in cardiac index. In severe cases, the cardiac index should be maintained >2.5 L/min/m^2 with the use of inotropic agents (vasopressin 1–6 units per hour if hypotension is present).

2. Hypercapnic Respiratory Failure
   The main goal in treating patients with hypercapnic respiratory failure is to improve alveolar ventilation through the use of mechanical ventilation. This is most commonly done through an endotracheal tube using a volume-controlled ventilator (usual initial settings are VT 5–7 mL/kg, A/C mode, rate 8–10 breaths per minute, and FiO_2 1.0). ABGs are checked 10–20 min later to detect inadvertent and potentially life-threatening acute alkalosis secondary to overcorrection of the hypercapnia and to adjust the FiO_2. Noninvasive mechanical ventilation has been effective in patients with neuromuscular conditions. The management of hypercapnic respiratory failure in asthma and COPD is discussed above.

■ VI. BAROTRAUMA

A. Definition. Barotrauma is lung injury that is related to high *alveolar* pressures (and volumes). In the intensive care unit (ICU) setting, *barotrauma* specifically refers to positive-pressure ventilator-induced lung damage. Occasionally, a patient may be admitted to the ICU after a diving accident (sudden decompression) or foreign body aspiration (ball-valve mechanism).

B. Clinical Manifestations
   1. *Classic mechanical ventilator-induced barotrauma* is manifested by *extra-alveolar air* in the form of
      a. Pulmonary interstitial emphysema (PIE)
      b. Subpleural air cysts
      c. Pneumomediastinum
      d. Pneumothorax (PTX)
      e. Subcutaneous emphysema
      f. Pneumopericardium
      g. Pneumoretroperitoneum
      h. Pneumoperitoneum
      i. Gas emboli (main clinical manifestation in diving accidents)
   2. *Tension PTX* occurs in 30–97% of all PTXs in mechanically ventilated patients and is characterized by worsening hypoxemia, hypotension, or even cardiovascular collapse with pulseless electrical activity (PEA). Chest x-ray shows lung collapse with hemithorax expansion and contralateral mediastinal shift.
   3. Physical examination reveals
      a. Absent breath sounds, hyperresonance to percussion, and decreased chest excursion on the affected side in cases of PTX.
      b. Crepitation on palpation or auscultation is found in cases of subcutaneous emphysema.
      c. Mediastinal "crunch" in pneumomediastinum.
      d. Changes in mental status or neurologic deficits are usually found in patients with gas embolism.
   4. Development or worsening of *diffuse lung injury* (in the form of noncardiogenic pulmonary edema) also has been associated with positive-pressure ventilation.

C. Pathophysiology
   1. Alveolar rupture occurs at the site where alveoli attach to the bronchovascular sheath. From there, extra-alveolar air may dissect the peribronchovascular

tissues into different planes to produce the clinical manifestations listed above. Alternatively, direct rupture of a subpleural cyst into the pleural cavity may also cause PTX.

2. Positive-pressure mechanical ventilation has also shown to produce
   a. Increased lung microvascular permeability and filtration pressure
   b. Alveolar epithelial injury
   c. Alteration of surfactant function and turnover

D. Diagnosis
1. A high index of suspicion should be maintained in those patients at risk for barotrauma (i.e., use of high VT, high plateau pressure, peak pressure, PEEP, dynamic hyperinflation, extensive lung damage, and prolonged mechanical ventilation).
2. Chest x-ray will usually confirm a diagnosis of extra-alveolar air. PIE (seen as linear radiolucent streaks) is the first radiologic sign and should alert the physician for the risk of progression to PTX
3. The classic radiologic sign of PTX (pleural line separated from the apicolateral chest wall) may not be present in ICU patients when an antero-posterior (AP) portable chest x-ray is taken in the supine or semirecumbent position. In those patients, attention should also be paid to the mediastinal and subpulmonic recesses where air accumulates.

E. Management
1. *Extra-alveolar air without PTX* is managed conservatively. Observation and avoidance of risk factors, if possible, is indicated (see "Avoiding Barotrauma," above).
2. In general, all PTXs in a mechanically ventilated patient should be treated with *tube thoracotomy* (see Chapter 15, "Special Techniques").
3. The use of *"prophylactic" tube thoracotomy* for high-risk mechanically ventilated patients is controversial and not recommended. Instead, it is advised to follow these patients closely and be prepared for immediate placement of a chest tube should PTX develop.
4. Persistent *bronchopleural fistula(s)* despite chest tube suction should be managed with the lowest VT, Ppeak, and Pplateau that permit adequate ventilation. High-frequency jet ventilation (HFJV), independent lung ventilation, or surgery should be considered if the air leak is massive and does not respond to the usual management.
5. Recompression in a hyperbaric chamber is indicated for diving accidents resulting in *air embolism.*

■ VII. MASSIVE HEMOPTYSIS

A. Definition. Hemoptysis is the expectoration of blood that originates from below the larynx. It is considered massive when the rate of bleeding is at least 400 mL in 6 h or 600 mL in a 24-h period.

B. Etiology. The most common causes of massive hemoptysis are
1. Tuberculosis (rupture of Rasmussen's aneurysm)
2. Bronchiectasis (erosion of bronchial vasculature)
3. Bronchogenic carcinoma (invasion of pulmonary vessels)

4. Lung abscess (destruction of fairly normal vessels due to inflammation)
   Other etiologies include bronchial carcinoids, cystic fibrosis, broncholithiasis, aspergilloma, trauma, bronchovascular and arteriovenous (A-V) fistulas, mitral stenosis, and the pulmonary-renal syndrome.

C. Evaluation
1. Differentiate Hemoptysis From Hematemesis
   Hemoptysis is usually bright red blood, frothy, with an alkaline pH. In contrast, hematemesis is usually darker with an acidic pH. At times this differentiation cannot be made easily, because hematemesis may produce blood aspiration into the tracheobronchial tree, which in turn causes "hemoptysis," and, on the other hand, patients with hemoptysis may swallow blood and vomit it after coughing.
2. Localize the Bleeding Site
   Localization of the bleeding site is important to adequately plan any interventional procedure. Bleeding coming from the upper airways can be excluded by performing an ear, nose, and throat (ENT) examination.
   a. Chest x-ray may suggest the bleeding site
      (1). Lung masses, apical cavitary lesions, or infiltrates in the chest x-ray should point to these sites as the source of bleeding.
      (2). A normal chest x-ray is consistent with bleeding arising from the airways.
      (3). Roentgenograms showing bilateral or diffuse disease are not helpful in pointing out the origin of bleeding.
   b. *Bronchoscopy* should be performed to further identify the site and cause of bleeding and to achieve temporary control. The type and timing of the procedure depend on the rate of bleeding.
      (1). When *flexible fiberoptic bronchoscopy (FFB)* is done, the patient must be intubated with a large endotracheal tube that may be used to tamponade the affected lung if necessary,
      (2). *Rigid bronchoscopy* is the preferred temporizing method to evaluate and control a massive bleeding. This procedure should be done under general anesthesia in the operating room by trained physicians.
   c. *Angiography* of the bronchial and pulmonary circulation is recommended as it can determine the bleeding site and simultaneously provide therapy (i.e., coiling).
3. Laboratory Determinations
   Obtain ABGs to determine if acidosis exists and to confirm level of oxygenation. The obtain a complete blood cell count, PT, partial thromboplastin time (PTT), bleeding time, creatinine, serum urea nitrogen (BUN), and request blood type and cross-matching.

D. Management
1. Ensure Adequate Ventilation and Oxygenation
   This should be the main priority if we consider that the mode of death in massive hemoptysis is asphyxiation (as little as 150 cc of blood is needed to fill the airways).
   a. Depending on the rate of bleeding, it may only be necessary to administer supplemental O_2 through nasal cannula or face mask or, on the other extreme, it may be necessary to perform endotracheal intubation (single- or double-lumen [Carlen's tube]) to aspirate the blood and ventilate the patient while definitive therapy is being prepared.
   b. Position the patient in *the lateral decubitus position with the bleeding site down.*

2. Ensure Stable Hemodynamic Conditions
   a. Obtain adequate venous access.
   b. Administer fluids as needed (normal saline or blood).
3. Suppress Cough
   a. Codeine 60 mg PO q6 h.
   b. Sedatives may be added (i.e., midazolam 2 mg or lorazepam 1 mg, IV q2 h pm).
4. Control the Bleeding Site
   a. *Bronchoscopic procedures* include
      (1). Bronchial packing through a rigid bronchoscope
      (2). Tamponade of airway with a balloon-tipped (Fogarty) catheter through a rigid bronchoscope or alongside a flexible bronchoscope
      (3). Bronchial lavage with cold saline through a rigid bronchoscope
      (4). Coagulation of visible lesions with a Neodynium-YAG laser (not helpful in very active, brisk bleeding)
   b. *Bronchial artery embolization* is the method of choice for patients with massive or submassive hemoptysis and contraindications for surgery. Given its high success rate (90%), embolization has become a first-line treatment for all patients with massive or recurrent hemoptysis.
   c. *Surgical resection* is recommended for localized lesions that can be removed. It should not be offered to patients with
      (1). Metastatic lung cancer
      (2). Severe pulmonary or cardiovascular status
   d. *Correct coagulopathy,* if present (i.e., administer FFP and vitamin K if PT is prolonged, transfuse platelets in severe thrombocytopenia).

E. Prognosis
1. Although the underlying medical condition(s) affect the prognosis in massive hemoptysis, the best estimator of mortality is the rate of bleeding. A study by Cracco showed that hemoptysis of 600 cc occurring over
   a. 4 h has 71% mortality
   b. 4–16 h has 45% mortality
   c. 16–48 h has 5% mortality
2. Median operative mortality in massive hemoptysis is 17%. Actively bleeding patients at the time of the surgery have a higher mortality when compared with nonactively bleeding patients.

■ VIII. UPPER AIRWAY OBSTRUCTION

A. Acute upper airway obstruction can be a life-threatening situation that may lead to cardiac arrest. This situation requires immediate intervention. This is important among the pediatric population, having smaller airways that can quickly progress to complete obstruction.

B. An initial rapid assessment is pertinent to evaluate upper airway patency (audible speech, cough, drooling), respiratory distress (retractions, nasal flaring), and hypoxemia.

C. Immediate treatment: Chin tilt and lift maneuvers should be attempted, as well as removal of foreign body if suspected. Direct laryngoscopy can also be done,

preparing patient for emergent intubation. If endotracheal intubation is not successful on the first, but successful ventilation using bag mask, alternative modes may be used such as LMA, combitube, oral and nasal airways, or emergency surgical intervention (See Chapter 15, "Special Techniques").

■ IX. USEFUL FACTS AND FORMULAS

A. Lung Volumes. Normal values for pulmonary volumes and capacities in humans are depicted in Table 13.7 .
The *vital capacity* (VC) is calculated as follows:

$$VC = IRV + ERV + V_T$$

The *residual volume* (RV) is calculated as the difference between the functional residual capacity (FRC) and the expiratory reserve volume (ERV):

$$RV = FRC - EV$$

Alternatively, if the total lung capacity (TLC) and vital capacity (VC) are known, the following formula can be utilized:

$$RV = TLC - VC$$

The oldest method to measure *FRC* is the *equilibration technique,* utilizing the following formula:

$$FRC = [(C_1 \times V_1)/C_2] - V_1$$

Table 13.7. Normal Values for Lung Volumes in Upright Subjects

Volume or capacity	*Approximate value in upright subjects*
Total lung capacity (TLC)	6 L
Vital capacity (VC)	4.5 L
Residual volume (RV)	1.5 L
Inspiratory capacity (IC)	3 L
Functional residual capacity (FRC)	3 L
Inspiratory reserve volume (IRV)	2.5 L
Expiratory reserve volume (ERV)	1.5 L
Tidal volume (V_T)	0.5 L

where C_1 = known concentration of a test gas in the spirometer; V_1 = volume of gas in the spirometer; C_2 = the fractional value of the gas after the subject breathes in the spirometer until the concentration of the test gas equals that in the spirometer.

Another way to measure FRC is by utilizing the *nitrogen washout procedure* and the following formula:

$$FRC = (V_B \times C_B)/C_X$$

where V_B = amount of exhaled nitrogen volume in the bag; C_B = fractional concentration of nitrogen in the bag; C_X = subject initial fractional concentration of nitrogen (0.80).

Alternatively, FRC can be calculated using body plethysmography as follows:

$$FRC = (\Delta/\Delta P)(P_B + \Delta P)$$

where ΔV = change in volume; ΔP = change in pressure; P_B = atmospheric pressure minus water vapor pressure (P_{H2O}).

The *tidal volume* (V_T) is the sum of the dead space volume (V_D) and the alveolar volume (V_A):

$$V_T = V_D + V_A$$

The average *dead space volume* (V_D) is estimated as 1 mL/lb body weight. For an average 70-kg man

$$V_D = 70 \times 2.2 \times 1 = 154\text{mL}$$

B. Pulmonary Ventilation. The easiest way of estimating *minute ventilation* (V_E) is by using the following formula:

$$V_E = V_T \times RR = \text{mL/min}$$

where V_T = tidal volume; RR = respiratory rate.

Minute ventilation is also the sum of dead space (V_D) and alveolar ventilation (V_A):

$$V_E = V_A + V_D$$

The *alveolar ventilation* (V_A) can be calculated as

$$V_A = (V_T - V_D) \times N$$

where N = frequency of breathing in breaths per minute; V_D = dead space ventilation.

An alternative method requires knowledge of the CO_2 production by the patient. The *production of* CO_2 ($\dot{V}CO_2$) can be calculated as follows:

$$\dot{V}CO_2 = V_A \times F_{ACO2}$$

where F_{ACO2} = fractional concentration of CO_2 in the alveolar gas; and $V_A = VCO_2/F_{ACO2}$

Dead space ventilation (V_D) can be calculated if the minute ventilation (V_E) is known:

$$V_D = V_E([PaCO_2 - PECO_2])/PaCO_2$$

The *partial pressure of alveolar* CO_2 ($PACO_2$) is more convenient for these calculations and for practical purposes:

$$PACO_2 = FACO_2 \times (P_B - 47)$$

In normal lungs, the *arterial* CO_2 ($PaCO_2$) approximates the $PACO_2$. Therefore, the V_A formula can be rewritten as

$$V_A = K(VCO_2/PaCO_2)$$

where K = a factor (0.863) that converts CO_2 concentrations to pressure (mmHg).

C. Gas Transport in Blood. The difference between the inspired and expired fractional concentration of O_2 represents the *oxygen uptake* ($\dot{V}O_2$):

$$\dot{V}O_2 = (V_I \times FiO_2) - (V_E \times FEO_2)$$

where V_I = volume of gas inhaled; FiO_2 = fractional concentration of inspired oxygen; V_E = volume of gas exhaled; F_EO_2 = fractional concentration of expired oxygen.

The amount of O_2 *in solution in 100 mL of blood* is calculated as (assuming a partial O_2 pressure of 70 mmHg)

$$(PO_2/760) \times \alpha O_2 = 70/760 \times 2.3 = 0.21\text{mL}/100\text{mL}$$

The PaO_2 at which hemoglobin is 50% saturated (P_{50}) can be calculated from the venous pH and arterial blood gases as

$$P_{50} = \text{antilog}\frac{\text{Log}(1/k)}{n} = \text{normal}\,22 - 33\,\text{mm Hg}$$

where

$$(1/k) = (\text{antilog}\,[n \times \log \text{PaO2}_{7.4}]) \times (100 - \text{SaO}_2/\text{SaO}_2)$$

$$\text{antilog}\,[n \times \log \text{PaO2}_{7.4}] = \log \text{PaO}_2 - 0.5(7.4 - \text{venous pH})$$

$$n = \text{Hill constant} = 2.7 \text{ for hemoglobin A}$$

The *Fick equation* for *oxygen consumption* (VO_2) is calculated as follows:

$$VO_2 = Q(CaO_2 - C\bar{v}O_2)$$

where Q = cardiac output (L/min); CaO_2 = arterial oxygen content; CFA~vO_2 = mixed venous oxygen content.

The volume of carbon dioxide exhaled per unit time (*CO_2 production* or $\dot{V}CO_2$) is calculated as follows:

$$\dot{V}CO_2 = (V_E \times FECO_2) - (VI \times F_ICO_2)$$

where V_E = volume of gas exhaled per unit time; F_ECO_2 = fractional concentration of carbon dioxide in the exhaled gas; V_I = volume of gas inhaled per unit time; F_ICO_2 = fractional concentration of inspired carbon dioxide.

Since the inspired gas usually contains negligible amounts of carbon dioxide, another representation of this formula is

$$\dot{V}CO_2 = V_E \times F_ECO_2$$

D. Pulmonary Circulation. The *mean pulmonary artery pressure* (PAP) can be calculated utilizing the following formula:

$$PAP = (PVR \times PBF) + PAOP$$

where PVR = pulmonary vascular resistance; PBF = pulmonary blood flow (which typically equals the cardiac output). Reorganizing the above formula, the *pulmonary vascular resistance* (PVR) is then calculated as

$$PVR = (\text{Mean PAP} - PAOP)/CO$$

where PAOP = pulmonary artery occlusion pressure; CO = cardiac output.

The pressures that surround the vessels in the pulmonary circulation contribute to the *transmural pressure* (Ptm) represented as

$$Ptm = Pvas - Pis$$

where Pvas = vascular pressure; Pis = perivascular interstitial pressure.

Table 13.8. Pulmonary Blood Flow Zones

Blood flow zones	*Pressures*
I	Palv > Ppa > Ppv
II	Ppa > Palv > Ppv
III	Ppa > Ppv > Palv
IV	Ppa > Ppv > Palv

Abbreviations: Palv, pressure surrounding the alveolar vessels; Ppa, mean pulmonary arterial pressure; Ppv, mean venous (left atrial) pressure.

When the left atrial pressure (Pla) is available, the *driving pressure* responsible for producing *pulmonary blood flow* is then calculated as

$$(\text{Ppa} - \text{Pla}) = \text{Q} \times \text{Rvas}$$

where Ppa = mean pulmonary arterial pressure; Pla = mean left atrial pressure; Q = pulmonary blood flow; Rvas = pulmonary vascular resistance.

The *pulmonary vascular compliance* (Cvas) can be calculated utilizing the following formula:

$$\text{Cvas} = \Delta\text{Vvas}/\Delta\text{Pvas}$$

where ΔVvas = change in blood volume; ΔPvas = change in vascular pressure.

The *blood flow zones* in an idealized upright lung with normal pressure differences are depicted in Table 13.8 .

E. Mechanics and Gas Flow. The pressure inside the lungs relative to the pressure outside is known as the *transpulmonary pressure* (TP) and is calculated as

$$\text{TP} = \text{P}_{\text{alv}} - \text{P}_{\text{pl}}$$

where P_{alv} = alveolar pressure; P_{pl} = pleural pressure.

The change in volume (ΔV) for a unit pressure (ΔP) under conditions of no flow is the *static compliance:*

$$\text{Static compliance } (\text{C}_\text{s}) = \frac{\Delta\text{V}}{\Delta\text{P}}$$

Clinically, this formula can be simplified as follows:

$$\text{C}_\text{s} = \frac{\text{V}_\text{T}}{\text{Plateau airway pressure} - (\text{PEEP} + \text{autoPEEP})}$$

where V_T = tidal volume. Normal C_s value is 100 mL/cmH_2O.

The *dynamic compliance* (C_{dyn}) can be calculated utilizing the following formula:

$$C_{dyn} = \frac{V_T}{\text{Plateau airway pressure} - (\text{PEEP} + \text{autoPEEP})}$$

Normal C_{dyn} value is 100 mL/cmH_2O.

The specific compliance (C_{spec}) is calculated utilizing the following formula:

$$C_{spec} = C_{stat}/FRC$$

The *chest wall compliance* (Cw) can be calculated as

$$C_W = \frac{V_T}{\text{Airway pressure} - \text{Atmospheric pressure}}$$

Another formula that can be used under special circumstances (i.e., lung transplantation) is the *separate lung compliance* (C_X) and is calculated as

$$C_X = \frac{V_T}{\text{Airway pressure} - \text{Intrapleural pressure}}$$

The type of gas flow in the lung is *laminar flow* and is described mathematically by the *Poiseuille equation:*

$$\Delta P = \frac{8\mu lV}{\pi r^4}$$

where ΔP = hydrostatic pressure drop; V = gas flow; μ = gas viscosity; 1 = path length; r = radius of the tube. From this equation, resistance ($R = \Delta P/V$) can be calculated:

$$R = \frac{8\mu l}{\pi r^4}$$

On the other hand, *the pressure drop during turbulent flow* can be calculated utilizing the following formula:

$$\Delta P = \frac{\mu l^{-} \rho^{-}}{r^{19/4}}$$

where ΔP = pressure drop during turbulent flow; μ = viscosity; ρ = density.

The *Reynolds number* (Re) is the ratio of the pressure loss due to density-dependent or inertial flow vs. the pressure loss due to viscous flow. This number is used to predict the nature of a particular flow and is calculated as follows:

$$Re = \frac{2\rho rV}{\mu A}$$

The *airway resistance* (Raw) using body plethysmography can be calculated utilizing the following formula:

$$\text{Raw} = \frac{\Delta\text{Vbox}}{\text{V}} \times \frac{\text{Palv}}{\text{V}} = \frac{\text{Palv}}{\Delta\text{Vbox}}$$

where ΔVbox = volume changes in the box; V = flow; Palv = alveolar pressure.

The *work of the respiratory system* (W) can be calculated as

$$\text{W} = \text{P} \times \text{V}$$

where P = pressure; V = volume.

F. Ventilation/Perfusion. The physiological *dead space* can be calculated utilizing the classic *Bohr equation:*

$$\text{V}_\text{D}/\text{V}_\text{T} = \frac{\text{P}_\text{A}\text{CO}_2 - \text{P}_\text{E}\text{CO}_2}{\text{P}_\text{A}\text{CO}_2}$$

where $\text{P}_\text{A}\text{CO}_2$ = partial pressure of carbon dioxide in the alveolar gas; $\text{P}_\text{E}\text{CO}_2$ = partial pressure of carbon dioxide in mixed expired gas.

The above formula with the *Enghoff modification* is used in clinical practice:

$$\text{V}_\text{D}/\text{V}_\text{T} = \frac{\text{PaCO}_2 - \text{P}_\text{E}\text{CO}_2}{\text{PaCO}_2} = 0.30 \text{ in healthy subjects}$$

The quantity of blood passing through pulmonary right-to-left *shunts* (Qs/Q) is calculated as

$$\text{Qs}/\text{Q} = \frac{\text{Cc}'\text{O}_2 - \text{CaO}_2}{\text{Cc}'\text{O}_2 - \text{C}\bar{\text{v}}\text{O}_2}$$

where

$$\text{Cc}'\text{O}_2 = (\text{Hb} \times 1.38) + \text{P}_\text{A}\text{O}_2 \times \frac{\alpha}{760}$$

Therefore, the Qs/Q formula can be rearranged as

$$\text{Qs}/\text{Q} = \frac{(\text{P}_\text{A}\text{O}_2 - \text{PaO}_2) \times 0.0031}{(\text{P}_\text{A}\text{O}_2 - \text{PaO}_2) \times 0.0031 + (\text{CaO}_2 - \text{C}\bar{\text{v}}\text{O}_2)}$$

G. Alveolar Gas Equation. The *alveolar air equation* is based firmly on Dalton's law but is expressed in terms that emphasize alveolar O_2 and CO_2:

$$\text{P}_\text{A}\text{O}_2 = (\text{P}_\text{ATM} - \text{P}_{\text{H}_2\text{O}})\text{FiO}_2 - \text{PCO}_2/\text{RQ}$$

where P_AO_2 = partial pressure of O_2 in the alveolus under present conditions; P_{ATM} = current, local atmospheric pressure. P_{H_2O} = vapor pressure of water at body temperature and 100% relative humidity; FiO_2 = fraction of inspired O_2; PCO_2 = partial pressure of CO_2 in arterial blood; RQ = respiratory quotient.
At *sea level,* this equation can be simplified to

$$P_AO_2 = 150 - 1.25 \times PaCO_2$$

The *arterial oxygen tension (PaO_2) corrected for age* is calculated as

$$PaO_2 \text{age} - \text{corrected} = 100 - 1/3 \text{ age (in years)}$$

The *alveolar-arterial O_2 gradient* is *age-corrected* according to the following formula:

$$\text{Age correction} = 2.5 + (0.25 \times [\text{age in years}])$$