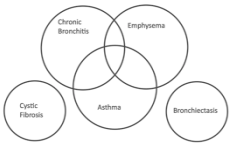
Chronic Obstructive Airway Diseases

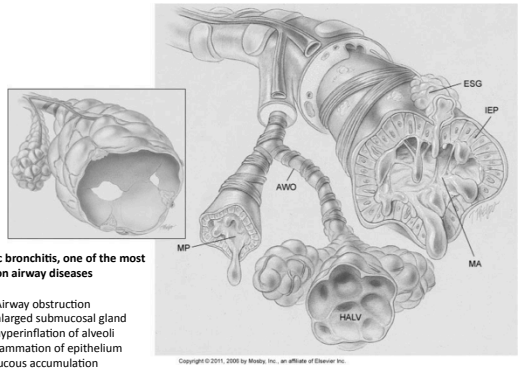
Chronic Bronchitis & Emphysema

RSPT 2310

Obstructive Airways Diseases

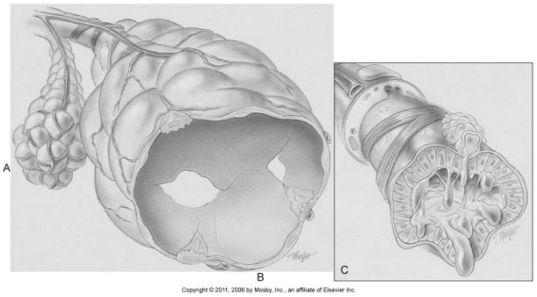
- Obstructive lung diseases are characterized by a variety of pathologic conditions
  - bronchial inflammation
  - excessive airway secretions
  - mucous plugging
  - bronchospasm
  - distal airway weakening
- The most common obstructive lung disorders
  - chronic bronchitis
  - emphysema
  - asthma
- Chronic bronchitis, emphysema, and asthma may appear alone, but often appear in combination
  - When chronic bronchitis and emphysema appear together as one disease complex, the patient is said to have chronic obstructive pulmonary disease (COPD)
  - Although asthma can be chronic, it is usually a more acute and intermittent respiratory disorder.
  - Other obstructive lung disorders include cystic fibrosis and bronchiectasis





Chronic bronchitis, one of the most common airway diseases

AWO, Airway obstruction
ESG, enlarged submucosal gland
HALV, hyperinflation of alveoli
IEP, inflammation of epithelium
MA, mucous accumulation
MP, mucous plug

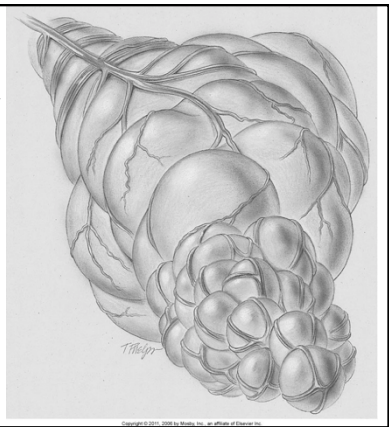


Panlobular emphysema

A, Normal alveoli for comparison purposes
B, Panlobular emphysema: abnormal weakening and enlargement of all air spaces distal to the terminal bronchioles
C, Excessive bronchial secretions from bronchitis, a common alteration of the lungs

Centrilobular emphysema

Abnormal weakening and enlargement of the respiratory bronchioles and alveoli in the proximal portion of the acinus.



American Thoracic Society Guidelines

American Thoracic Society Guidelines

- Chronic obstructive pulmonary disease is a preventable and treatable disease state characterized by airflow limitation that is not fully reversible
  - The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking
  - Although COPD affects the lungs, it also produces significant systemic consequences

American Thoracic Society Guidelines

- Chronic bronchitis is defined clinically as chronic productive cough for 3 months in each of 2 successive years in a patient in whom other causes of productive chronic cough have been excluded

American Thoracic Society Guidelines

- Emphysema is defined pathologically as the presence of permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis

Chronic Obstructive Diseases

- Chronic bronchitis and emphysema
  - Can each develop alone
  - Often occur together as one disease complex called *chronic obstructive pulmonary disease*
  - COPD refers to two diseases occurring concurrently
  - Patients with COPD demonstrate a variety of clinical manifestations associated with both disorders
  - The treatment of chronic bronchitis, emphysema, or a combination of both disorders (COPD) is essentially the same in the clinical setting

Anatomic Alterations

Anatomic Alterations of the Lungs

- Chronic Bronchitis:
  - Chronic inflammation and swelling of the wall of the peripheral airways
  - Excessive mucous production and accumulation
  - Partial or total mucous plugging of the airways
  - Smooth muscle constriction of bronchial airways (bronchospasm)
  - Air trapping and hyperinflation of alveoli—occasionally in late stages

Anatomic Alterations of the Lungs

- Chronic Bronchitis
  - The conducting airways (particularly the bronchi) are the primary structures that undergo change in chronic bronchitis
  - As a result of chronic inflammation the bronchial walls are narrowed by vasodilation, congestion, and mucosal edema
  - This condition is often accompanied by bronchial smooth muscle constriction

Anatomic Alterations of the Lungs

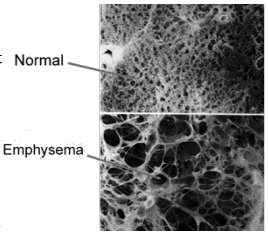
- Chronic Bronchitis
  - In addition, continued bronchial irritation causes the submucosal bronchial glands to enlarge and the number of goblet cells to increase, resulting in excessive mucous production
  - The number and function of cilia lining the tracheobronchial tree are diminished, and the peripheral bronchi are often partially or totally occluded by inflammation and mucous plugs, which in turn leads to hyperinflated alveoli

Anatomic Alterations of the Lungs

- Chronic Bronchitis
  - Major pathologic or structural changes
    - Chronic inflammation and swelling of the wall of the peripheral airways
    - Excessive mucous production and accumulation
    - Partial or total mucous plugging of the airways
    - Smooth muscle constriction of bronchial airways (bronchospasm)
    - Air trapping and hyperinflation of alveoli—occasionally in late stages

Anatomic Alterations of the Lungs

- Emphysema
  - Characterized by weakening and permanent enlargement of the air spaces distal to the terminal bronchioles and by destruction of the alveolar walls
  - Many adjacent pulmonary capillaries also are affected, resulting in decreased surface area for gas exchange

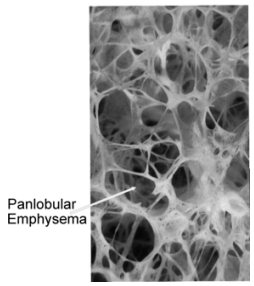


Anatomic Alterations of the Lungs

- Emphysema
  - Distal airways, weakened in the process, collapse during expiration in response to increased intrapleural pressure, trapping gas in the alveoli
  - Two major types of emphysema: panacinar (panlobular) emphysema and centriacinar (centrilobular) emphysema

Anatomic Alterations of the Lungs

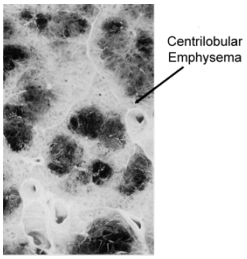
- Emphysema
  - In panacinar emphysema, or panlobular emphysema, there is an abnormal weakening and enlargement of all alveoli distal to the terminal bronchioles, including the respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli—the entire acinus is affected by dilatation and destruction



Anatomic Alterations of the Lungs

- Emphysema
  - The alveolar-capillary surface area is significantly decreased
  - Panlobular emphysema commonly is found in the lower parts of the lungs and often is associated with a deficiency of alpha$_1$-antitrypsin
  - Panlobular emphysema is the most severe type of emphysema and therefore the most likely to produce significant clinical manifestations

Anatomic Alterations of the Lungs



- Emphysema
  - In centriacinar emphysema, or centrilobular emphysema, the pathology involves the respiratory bronchioles in the proximal portion of the acinus
  - The respiratory bronchiolar walls enlarge, become confluent, and are then destroyed

Anatomic Alterations of the Lungs

- Emphysema
  - A rim of parenchyma remains relatively unaffected
  - Centriacinar emphysema is the most common form of emphysema and is strongly associated with cigarette smoking and with chronic bronchitis

Anatomic Alterations of the Lungs

- Emphysema
  - Major pathologic or structural changes
    - Permanent enlargement and destruction of the air spaces distal to the terminal bronchioles
    - Destruction of pulmonary capillaries
    - Weakening of the distal airways, primarily the respiratory bronchioles
    - Air trapping and hyperinflation

Etiology and Epidemiology

- Incidence of COPD
  - Precise incidence is unknown
    - Estimated that 10 to 15 million people in the United States have chronic bronchitis, emphysema, or a combination of both
    - Most authorities agree that COPD is underdiagnosed
      - If the people who have not been "officially" diagnosed with COPD are considered, the incidence is probably over 20 million people in the United States

Etiology and Epidemiology

- Incidence of COPD
  - Generally accepted that more people have chronic bronchitis than emphysema
    - National Center for Health Statistics estimates that in the United States about 9.5 million people have chronic bronchitis and 4.1 million people have emphysema
  - Annual cost related to COPD in the United States was about $37.2 billion—including $20.9 billion in direct costs, $7.4 billion in morbidity costs, and $8.9 billion in indirect costs

Etiology and Epidemiology

- COPD is the fourth leading cause of death, claiming more that 100,000 Americans each year
  - It is estimated that COPD will become the third leading cause of death by 2020
- Historically, more men than women have died from COPD each year
  - Since the year 2000, however, more women than men have died from COPD each year

Risk Factors

Risk Factors

- COPD risk factors are related to the total burden of inhaled particles a person encounters over his or her lifetime
  - This is why we questions patients about
    - Smoking history
      - how long did they smoke/how many packs per day
        - » 1 pack year = 1 ppd for 1 year
      - if they have quit smoking, when?
      - current and past employment
      - residence locales

Risk Factors

- Risk Factors for COPD
  - Tobacco smoke
    - Includes smoke from cigarette, pipe, cigar, and other types of tobacco smoking
  - Environmental tobacco smoke
  - According to GOLD, cigarette smoking is the most commonly encountered risk factor for COPD worldwide

Risk Factors

- Risk Factors for COPD
  - Occupational dusts and chemicals
    - Vapors, irritants, and fumes, when the exposures are sufficiently intense or prolonged
  - Indoor air pollution
    - From biomass fuel used for cooking and heating in poorly vented dwellings, a risk factor that particularly affects women in developing countries

Risk Factors

- Risk Factors for COPD
  - Outdoor air pollution
    - Also contributes to the lungs' total burden of inhaled particles and gases (e.g., silicates, sulfur dioxide, the nitrogen oxides, and ozone)
    - Appears to have a relatively small effect in *causing COPD*

Risk Factors

- Risk Factors for COPD
  - Conditions that affect normal lung growth
    - Any condition that affects lung growth during gestation and childhood (e.g., low birth weight, respiratory infections) has the potential for increasing the risk of developing COPD

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (alpha1-antitrypsin deficiency)
    - in about 1 out of every 50 cases of emphysema, there is a specific hereditary basis for panlobular emphysema called alpha1 (or α1)-antitrypsin deficiency

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - Major protein in the blood
    - Produced by the liver
    - Protects the lungs by blocking the effects of a powerful enzyme called elastase (carried by the body's white cells to help kill invading bacteria and to neutralize small particles inhaled into the lung)

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - When old white cells are destroyed in the lungs, elastase is released
    - Under normal circumstances, α1-antitrypsin works to inactivate the released elastase
    - When the α1-antitrypsin level is low, the elastase is free to attack and destroy the elastic tissue of the lungs

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency
    - The normal level of alpha1-antitrypsin is 200 to 400 mg/dL
    - Patients with normal levels of alpha1-antitrypsin are referred to genetically as having an MM phenotype or simply an M phenotype (homozygote)
    - The phenotype associated with severely low serum concentrations is the ZZ phenotype, or simply Z

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency
    - The heterozygous offspring of parents with the M and Z phenotypes have an MZ phenotype
    - This phenotype results in an intermediate deficiency of alpha1-antitrypsin, the precise effect which is unclear
    - It is strongly recommended, however, that individuals with this phenotype not smoke or work in areas having significant environmental air pollution

Risk Factors

- Risk Factors for COPD
  - One other possible risk factor is the remodeling of airways that occurs in asthma
  - Not yet proven

Diagnosis

Diagnosis

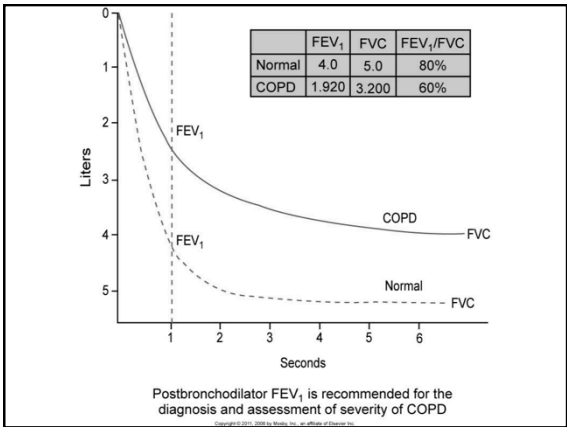
- Recommendations
  - The key indicators for considering a COPD diagnosis are as follows:
    - Over 40 years of age with
      - dyspnea
      - chronic cough
      - chronic sputum production
      - history of exposure to risk factors such as tobacco smoke

Diagnosis

- Recommendations
  - Although these indicators are not diagnostic by themselves, the presence of multiple indicators significantly increases the probability of a diagnosis of COPD
  - When multiple key indicators are present, the diagnosis of COPD should be confirmed by a pulmonary function study

Diagnosis

- The Pulmonary Function Study
  - The three main spirometry tests are used
    - Forced vital capacity (FVC)
    - Forced expiratory volume in 1 second (FEV_1)
    - Forced expiratory volume in 1 second/forced vital capacity ratio (FEV_1/FVC ratio)
      - clinically, the FEV_1/FVC ratio is also commonly called the forced expiratory volume 1 second percentage ($FEV_{1\%}$)



Postbronchodilator FEV_1 is recommended for the diagnosis and assessment of severity of COPD

Diagnosis

- COPD is confirmed when the both FEV_1 and FEV_1/FVC ratio are decreased
  - A post-bronchodilator FEV_1 is recommended for both the diagnosis and assessment of the severity of COPD
  - The degree of spirometric abnormality usually determines the severity of COPD
  - The extent of the symptoms should also be considered when developing individualized management programs

Diagnosis

- Additional Diagnostic Studies
  - In patients who are diagnosed with Stage II-IV COPD
    - Bronchodilator reversibility testing to rule out a diagnosis of asthma, particularly in patients with an atypical history
      - e.g., asthma in childhood and regular nocturnal night waking with cough and wheeze
    - Chest x-ray examination is seldom diagnostic in COPD but valuable to exclude alternative and/or additional diagnoses
      - e.g., pulmonary tuberculosis, and pneumonia, and to identify comorbidities such as cardiac failure

Diagnosis

- Additional Diagnostic Studies
  - In patients who are diagnosed with Stage II-IV COPD
    - Arterial blood gas measurement in patients with FEV_1 <50% predicted or with clinical signs suggestive of ventilatory failure or right-sided heart failure
      - the major clinical sign of ventilatory failure is cyanosis
      - clinical signs of right-sided heart failure include ankle edema and an increase in the jugular venous pressure
      - ventilatory failure is indicated by a PaO_2 <60 mm Hg, with or without a $PaCO_2$ >50 mm Hg while breathing room air

Diagnosis

- Additional Diagnostic Studies
  - In patients who are diagnosed with Stage II-IV COPD
    - Alpha1-antitrypsin deficiency screening: Perform when COPD develops in patients of Caucasian descent under 45 years of age or with a strong family history of COPD.

Diagnosis

- Chronic bronchitis or emphysema?
  - Can occur as one disease complex (COPD)
  - Can develop alone
- Clinical classifications
  - Patients with emphysema are classified as “pink puffers” (type A COPD)
  - Patients with chronic bronchitis are classified as “blue bloaters” (type B COPD)

Diagnosis

- Type A – “Pink Puffer”
  - Term comes from the reddish complexion rapid respiratory rate and pursed-lip breathing caused by
    - The progressive destruction of the distal airways and pulmonary capillaries leading to a reduced pulmonary blood flow
    - To compensate for the increased V/Q ratio the patient with emphysema hyperventilates
    - The increased respiratory rate, in turn, works to maintain a relatively normal arterial oxygenation level and causes a ruddy or flushed skin complexion
      - during the end stage of emphysema, the oxygenation status decreases and the carbon dioxide level increases

Diagnosis

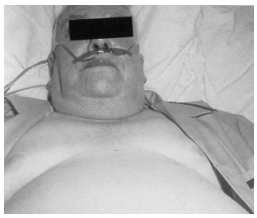
- Type A – "Pink Puffer"
  - In addition to the marked dyspnea and ruddy complexion, the pink puffer
    - Tends to be thin
      - muscle wasting and weight loss due to increased WOB
    - Has a barrel chest
      - overinflated lungs
    - Uses accessory muscles of inspiration
    - Exhales through pursed lips

Diagnosis

- Type B – "Blue Bloater"
  - Term comes from the cyanosis seen chronic bronchitis
    - The pulmonary capillaries in the patient with chronic bronchitis are not damaged
    - Patients with chronic bronchitis respond to the increased airway obstruction by decreasing ventilation and increasing cardiac output
    - This decreased V/Q ratio leads to decreased PaO_2, increased $PaCO_2$, and a compensated (normal) pH
      - compensated respiratory acidosis)
      - depressed respiratory drive
    - The low V/Q ratio and depressed respiratory drive both lead to a chronically reduced PaO_2 and polycythemia—which, in turn, causes cyanosis

Diagnosis

- Type B – "Blue Bloater"
  - In addition to the cyanosis and decreased F, the blue bloater
    - Tends to be stocky and overweight
    - Has a chronic productive cough
    - Has swollen ankles and legs and distended neck veins as a result of right-sided heart failure (cor pulmonale



Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Inspection		
Body build	Thin	Stocky, overweight
Barrel Chest	Common—classic sign	Normal

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Respiratory pattern	Hyperventilation & marked dyspnea; often occurs at rest Late stage: diminished respiratory drive & hypoventilation	Diminished respiratory drive Hypoventilation common, with resultant hypoxia and hypercapnia

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis (Cont'd)		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Pursed-lip breathing	Common	Uncommon
Cough	Uncommon	Common—classic sign
Sputum	Uncommon	Common—classic sign Copious amounts, purulent

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis (Cont'd)		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Cyanosis	Uncommon (reddish skin)	Common
Peripheral edema	Uncommon	Common Right-heart failure
Neck vein distention	Uncommon	Common Right-heart failure

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis (Cont'd)		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Use of accessory muscles	Common	Uncommon

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Auscultation	Decreased breath sounds, decreased heart sounds; prolonged expiration	Wheezes, crackles, rhonchi, depending on severity of disease
Percussion	Hyperresonance	Normal

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Chest radiograph	Hyperinflation, narrow mediastinum, normal or small vertical heart, low flat diagphragm, presence of blebs or bullae	Congested lung fields, densities, increased bronchial vascular markings, enlarged horizontal heart
Polycythemia	Uncommon	Common

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Infections	Occasionally	Common
Polycythemia	Uncommon	Common

Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis		
Clinical Manifestations	**Emphysema (Type A: Pink Puffer)**	**Chronic Bronchitis (Type B: Blue Bloater)**
Pulmonary Function Study		
DL_{CO} and DL_{CO}/VA	Decreased	Often normal
Other		
Pulmonary hypertension	Uncommon	Common
Cor pulmonale	Uncommon	Common Right-heart failure

Overview of the Cardiopulmonary Clinical Manifestations Associated with Chronic Bronchitis and Emphysema (COPD)

The following clinical manifestations result from the pathophysiologic mechanisms caused (or activated) by

- Excessive Bronchial Secretions
- Bronchospasm
- Distal Airway and Alveolar Weakening

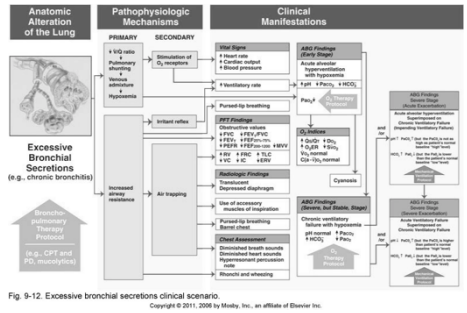


Fig. 9-12. Excessive bronchial secretions clinical scenario.

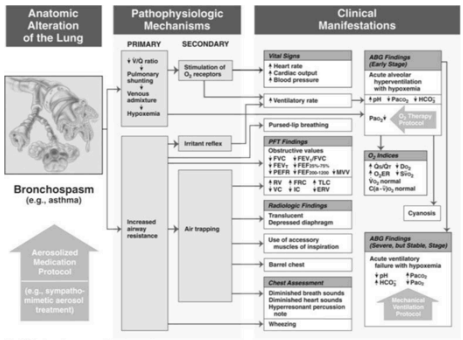


Fig. 9-11. Bronchospasm clinical scenario.

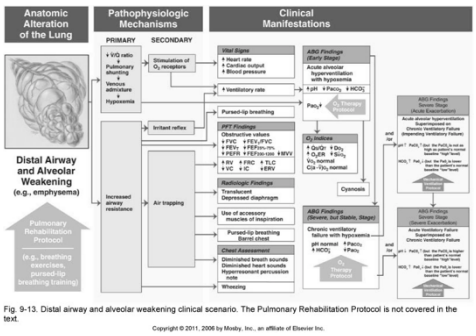


Fig. 9-13. Distal airway and alveolar weakening clinical scenario. The Pulmonary Rehabilitation Protocol is not covered in the text.

Clinical Data Obtained at the Bedside

Vital Signs	Chronic Bronchitis & Emphysema
Heart rate and respiratory rate	Stable patients: normal vital signs Exacerbations: Usually acute increase in heart rate and respiratory rate (Tachypnea) Classic sign of hypoxemia

Chest Assessment Findings	Emphysema	Chronic Bronchitis
Inspection		
General body build	Thin, underweight	Stocky, overweight
Altered Sensorium—anxiety, irritability	Common—severe stage Classic sign of hypoxemia	Common—during moderate and severe stage Classic sign of hypoxemia
Barrel Chest	Yes—classic sign	Occasionally
Digital Clubbing	Late-Stage	Common

Chest Assessment Findings (Cont'd)	Emphysema	Chronic Bronchitis
Inspection		
Cyanosis	Uncommon—often reddish skin	Common
Peripheral edema and venous distention	End-stage emphysema	Common—Because polycythemia & cor pulmonale are common, the following are often seen: Distended neck veins Pitting edema Enlarged & tender liver

Chest Assessment Findings (Cont'd)	Emphysema	Chronic Bronchitis
Inspection		
Use of accessory muscles	Common Especially during exacerbations	Uncommon End-stage in some chronic bronchitis
Hoover's Sign - The inward movement of the lower lateral chest wall during each inspiration—indicates severe hyperinflation	Common—Severe Stage	Uncommon

Chest Assessment Findings (Cont'd)	Emphysema	Chronic Bronchitis
Inspection		
Pursed-lip breathing	Common	Uncommon
Cough	Uncommon during mild and moderate stage Some coughing during severe-stage with infection	Classic sign More severe in the mornings
Sputum	Uncommon Little, mucoid	Common Classic sign; copious amounts, purulent

Chest Assessment Findings (Cont'd)	Emphysema	Chronic Bronchitis
Inspection		
Palpation of the Chest	Decreased tactile fremitus Decreased chest expansion PMI often shifts to the epigastric area	Normal

Chest Assessment Findings (Cont'd)	Emphysema	Chronic Bronchitis
Inspection		
Percussion of the Chest	Hyperresonance Decreased diaphragmatic excursion	Normal
Auscultation of the Chest	Diminished breath sounds Prolonged expiration Diminished heart sounds	Rhonchi Crackles Wheezes

Clinical Data Obtained from Laboratory Tests and Special Procedures

Pulmonary Function Test Findings

Moderate to Severe Chronic Bronchitis & Emphysema
(Obstructive Lung Pathophysiology)

Forced Expiratory Flow Rate Findings

FVC	FEV_T	FEV_1/FVC ratio	$FEF_{25\%-75}$
↓	↓	↓	↓

$FEF_{50\%}$	$FEF_{200-1200}$	PEFR	MVV
↓	↓	↓	↓

Pulmonary Function Test Findings

Moderate to Severe Chronic Bronchitis & Emphysema
(Obstructive Lung Pathophysiology)

Lung Volume & Capacity Findings

VT	IRV	ERV	RV	VC
N or ↑	N or↓	N or↓	↑	↓

IC	FRC	TLC	RV/TLC ratio
N or↓	↑	N or ↑	N or ↑

Diffusion Capacity (DL_{CO})

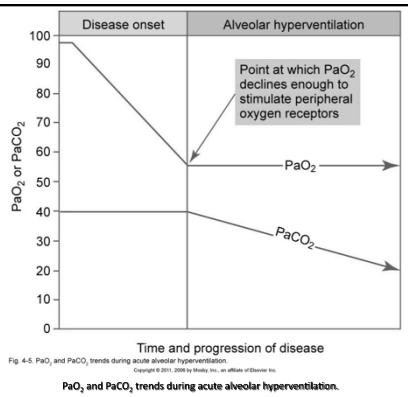
Emphysema	Chronic Bronchitis
Decreased • A decreased DL_{CO} is a classic diagnostic sign of emphysema	Normal

Arterial Blood Gases

Chronic Bronchitis & Emphysema

Mild to Moderate Stages
Acute Alveolar Hyperventilation with Hypoxemia

pH	$PaCO_2$	HCO_3	PaO_2
↑	↓	↓ (slightly)	↓

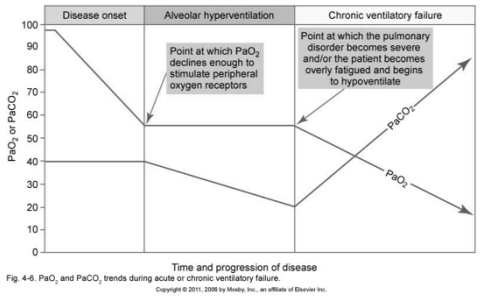


PaO_2 and $PaCO_2$ trends during acute alveolar hyperventilation.

Arterial Blood Gases
Chronic Bronchitis & Emphysema

Chronic Ventilatory Failure with Hypoxemia

pH	$PaCO_2$	HCO_3	PaO_2
N	↓	↓ (significantly)	↓



PaO_2 and $PaCO_2$ trends during acute or chronic ventilatory failure.

Arterial Blood Gases
Chronic Bronchitis & Emphysema

Acute Ventilatory Changes Superimposed On Chronic Ventilatory Failure

- Because acute ventilatory changes are frequently seen in patients with chronic ventilatory failure, the respiratory care practitioner must be familiar with and alert for the following:
  - Acute alveolar hyperventilation superimposed on chronic ventilatory failure
  - Acute ventilatory failure (acute hypoventilation) superimposed on chronic ventialtory failure.

Oxygenation Indices
Chronic Bronchitis and Emphysema
Moderate to Severe Stages

Q_S/Q_T	DO_2	VO_2	$C(a\text{-}v)O_2$	O_2ER	SvO_2
↑	↓	N	N	↑	↓

Hemodynamic Indices
Chronic Bronchitis and Emphysema
Moderate to Severe Stages

CVP	RAP	PA	PCWP	CO	SV
↑	↑	↑	N	N	N

SVI	CI	RVSWI	LVSWI	PVR	SVR
N	N	↑	N	↑	N

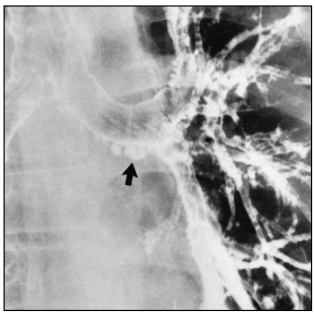
Laboratory Tests and Procedures

Test	Emphysema	Chronic Bronchitis
Hematocrit & Hemoglobin	Normal—mild moderate stage Elevated—late stage	Polycythemia common during the early and late stage
Electrolytes (abnormal)	Late stage: Hypochloremia (CL^-) When chronic ventilatory failure is present Hypernatremia (Na^+)	Early & Late stages: Hypochloremia (CL^-) When chronic ventilatory failure is present Hypernatremia (Na^+)

Laboratory Tests and Procedures		
Test	Emphysema	Chronic Bronchitis
Sputum examination (culture)	Normal	Streptococcus pneumoniae Haemophilus influsenzae Moraxella catarrhalis

Radiology Findings	
Test	Chronic Bronchitis
Chest Radiograph	Lungs may be clear if only large bronchi are affected Occasionally Translucent Depressed or flattened diaphragms Common Cor pulmonale
Bronchogram	Small spikelike protrusions

Chest X-ray film from a patient with chronic bronchitis. Note the translucent (dark) lung fields at the bases, depressed diaphragms, and long and narrow heart.



Chronic bronchitis. Bronchogram with localized view of left hilum. Rounded collections of contrast lie adjacent to bronchial walls and are particularly well demonstrated below the left main stem bronchus *(arrow)* in this film. They are caused by contrast in dilated mucous gland ducts

Radiology Findings	
Test	Emphysema
Chest Radiograph	Common Translucent Depressed or flattened diaphragms Long & narrow heart Increased retrosternal air space Occasionally cor pulmonale

Chest X-ray film of a patient with emphysema. The heart often appears long and narrow as a result of being drawn downward by the descending diaphragm.

Emphysema. Lateral chest radiograph demonstrates a characteristically large retrosternal radiolucency.

General Management of COPD

- Patient and Family Education
  - Help patient/family understand the disease and its effects on the body
  - Home care therapies and administration of medications
    - Although sympathomimetic, parasympatholytic, and xanthine agents are often prescribed, these drugs are minimally effective except in special cases
    - Excessive bronchial secretions may require expectorants, mucolytics and respiratory care modalities to mobilize secretions

General Management of COPD

- Patient and Family Education
  - Home care therapies and administration of medications
    - ICS therapy to reduce inflammation
    - Antibiotics to treat secondary respiratory tract infections
    - Prolastin may help patients with alpha$_1$ deficiency although long-term benefits have not been demonstrated
    - Oxygen therapy
      - If hypoxemia is present oxygen is usually required
      - Reevaluate patient on oxygen to avoid eliminating patients hypoxic drive

General Management of COPD

- Behavioral Management
  - Avoidance of smoking and inhaled irritants (smokingcessation clinics)
  - Avoidance of infections (immunizations, pneumococcal vaccine)
  - Encourage physician and patient involvement in a pulmonary rehabilitation, if available

General Management of COPD

- RC Treatment Protocols
  - Oxygen Therapy Protocol
  - Bronchial Hygiene Protocol
  - Aerosolized Medication Protocol
  - Mechanical Ventilation Protocol

GOLD STANDARDS

G lobal Initiative for Chronic

O bstructive

L ung

D isease

Components of Care: A COPD Management Program

The goals of COPD management include:

- Relieve symptoms
- Prevent disease progression
- Improve exercise tolerance
- Improve health status
- Prevent and treat complications
- Prevent and treat exacerbations
- Reduce mortality
- Prevent or minimize side effects from treatment

Four Components

- Assess and Monitor Disease
- Reduce Risk Factors
- Manage Stable COPD
- Manage Exacerbations

Component 1: Assess and Monitor

- Exposure to risk factors, including intensity and duration
- Past medical history, including asthma, allergy, sinusitis or nasal polyps, respiratory infections in childhood, and other respiratory diseases
- Family history of COPD or other chronic respiratory disease
- Pattern of symptom development
- History of exacerbations or previous hospitalizations for respiratory disorder.

Component 1: Assess and Monitor

- Presence of comorbidities, such as obesity, heart disease, malignancies, osteoporosis, and musculoskeletal disorders, which may also contribute to restriction of activity.
- Appropriateness of current medical treatments.

Component 1: Assess and Monitor

- Impact of disease on patient's life, including limitation of activity; missed work and economic impact; effect on family routines; and feelings of depression or anxiety.
- Social and family support available to the patient.
- Possibilities for reducing risk factors, especially smoking cessation.

Component 2: Reduce Risk Factors

- Counseling to quit smoking
- Pharmacotherapy
- Smoking prevention
- Occupational exposures
- Indoor and outdoor air pollution

Strategy to Help a Patient Quit Smoking

- Ask—Systematically identify all tobacco users at every visit.
- Advise—Strongly urge all tobacco users to quit.
- Assess—Determine willingness to make a quit attempt.
- Assist—Aid the patient in quitting.
- Arrange—Schedule follow-up contact.

Component 3: Manage Stable COPD

Management of stable COPD should be guided by the following general principles:

- Determine disease severity
- Implement a stepwise treatment plan that reflects this assessment of disease severity
- Choose treatments according to national and cultural preferences, the patient's skills and preferences, and local availability of medications

Component 3: Manage Stable COPD

- Patient education

Component 3: Manage Stable COPD

- Pharmacologic treatments
  - β_2-agnoists
    - Short-acting
      - Fenoterol
      - Levalbuterol
      - Salbutamol
      - Terbutaline

Component 3: Manage Stable COPD

- Long-acting
  - Formoterol
  - Saleterol
- Anticholinergics
  - Short-acting
    - Ipratropium bromide
    - Oxitropium bromide
  - Long-acting
    - Tiotropium

Component 3: Manage Stable COPD

- Combination short-acting β_2-agonists plus anticholinergic in one inhaler
  - Fenoterol/Ipratropium
  - Oxitropium bromide

Component 3: Manage Stable COPD

- Methylxanthines
  - Aminophylline
  - Theophylline

Component 3: Manage Stable COPD

- Inhaled glucocorticosteroids
  - Beclomethasone
  - Budesonide
  - Flutcasone
  - Triamcinolone

Component 3: Manage Stable COPD

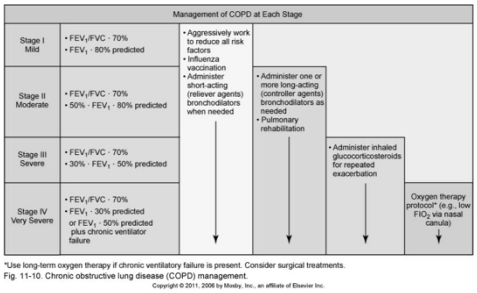
- Combination long-acting β_2-agonists plus glucocorticosteroids in one inhaler
  - Formoterol/Budesonide
  - Salmeterol/Fluticasone

Component 3: Manage Stable COPD

- Systemic glucocorticosteroids
  - Prednisone
  - Methyl-prednisolone

Component 3: Manage Stable COPD

- Glucocorticosteroids
- Vaccines
- Antibiotics
- Mucolytic
- Antitussives
- Non-pharmacologic treatment
- Rehabilitation
- Oxygen therapy
- Surgical treatment



*Use long-term oxygen therapy if chronic ventilatory failure is present. Consider surgical treatments.
Fig. 11-10. Chronic obstructive lung disease (COPD) management.

Component 4: Manage Exacerbation

- Exacerbation of COPD is defined as an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond normal-day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD

Additional Treatment Considerations for Emphysema

- $Alpha_1$ antitrypsin therapy
- Lung volume reduction surgery
- Lung transplantation