

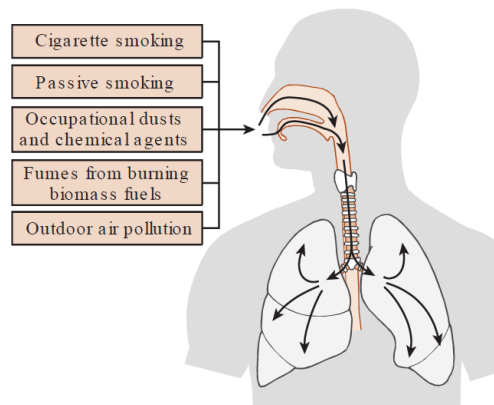
COPD

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a disease characterized by persistent air flow limitation that is usually progressive.

Previous terminology for COPD has been ‘chronic obstructive airways disease (COAD)’, ‘chronic obstructive lung disease (COLD)’ and ‘chronic air ow limitation (CAL)’ Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnea, cough, sputum production and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction. (GOLD,2023).

Aetiology



Pathophysiology

The inhalation of noxious particles causes lung inflammation and results in the following changes within the lungs:

1. Mucous gland hypertrophy with consequent increased mucus production
2. Destruction of ciliated epithelial cells in the airway walls
3. Chronic inflammatory changes and associated small airway fibrosis with consequent narrowing in the small airways
4. Increase in bronchial smooth muscle ([Chung 2005](#))
5. Loss of alveolar walls with consequent destruction of associated capillary beds.

The pathophysiological changes in the mucus secreting glands and the destruction of cilia in the airways result in increased sputum production and reduced mucociliary clearance. Chronic cough and sputum expectoration as a consequence of these changes are indicative of chronic bronchitis, for which the following clinical definition is used: sputum expectoration on most days for at least 3 months of the year over 2 successive years.

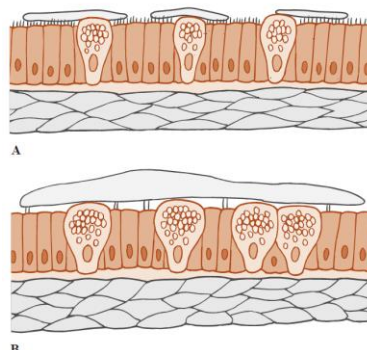


FIGURE 5-2 ■ Bronchial mucosa in chronic bronchitis. (A) Normal bronchial mucosa. (B) Bronchial mucosa showing enlarged mucus secreting glands and loss of ciliated epithelial cells.

The loss of alveolar walls is indicative of emphysema, for which the following anatomical definition is used: enlargement of the air spaces distal to the terminal bronchiole, with destruction of their walls.

Anatomic Alterations of the Lungs Associated with Chronic Bronchitis

Chronic inflammation and thickening of the walls of the peripheral airways.

- Excessive mucous production and accumulation.
- Partial or total mucous plugging of the airways.
- Smooth muscle constriction of bronchial airways (bronchospasm)—a variable finding.
- Air trapping and hyperinflation of alveoli may occur in late stages.

Anatomic Alterations of the Lungs Associated with Emphysema

Permanent enlargement and destruction of the air spaces distal to the terminal bronchioles.

- Destruction of the alveolar-capillary membrane.
- Weakening of the distal airways, primarily the respiratory bronchioles.
- Air trapping and hyperinflation.

There are three subtypes of emphysema: centrilobular, panlobular, and distal acinar (or subpleural):

- Centrilobular emphysema describes proximal dilation of the respiratory bronchioles, with alveolar ducts and sacs remaining normal. It is most common in the upper lobes and posterior portions of the lung.
- Panlobular emphysema describes dilation of all the respiratory airspaces in the acinus and occurs most frequently in the lung bases. Panlobular emphysema is seen in emphysema associated with α 1-antitrypsin deficiency.
- Distal acinar (subpleural) emphysema describes dilation of airspaces underneath the apical pleura, associated with apical bullae (large air collection contained within a thin outer wall), which can lead to spontaneous pneumothorax.

Clinical Features

The main symptoms of COPD are progressive dyspnoea, cough and sputum. On examination a person with COPD may have the following signs depending on disease severity. The more severe the disease the more obvious the following signs will be:

- Barrel chest, where the antero- posterior diameter of the chest wall is enlarged (like a barrel) due to hyperinflation.
- Accessory muscle use, particularly the inspiratory accessory muscles, e.g., sternocleidomastoid, pectoralis major and minor, serratus anterior, latissimus dorsi and trapezius.
- In severe disease or during an exacerbation accessory muscle may be used at rest. In less severe disease accessory muscle use may only occur during exercise. The accessory muscles are recruited due to the diaphragm being less available to contribute to ventilation as a consequence of hyperinflation.
- Chest radiograph showing signs of hyperinflation
- Decreased breath sounds on auscultation due to loss of alveoli and hyperinflation. There may be coarse crackles if sputum is present in the airways.
- Reduced functional exercise capacity measured by a reduced distance walked in either the 6-minute walk test or incremental shuttle walk test compared to predicted.
- Oxygen desaturation during exercise. In severe disease oxygen saturation may be low at rest.
- Clinically, the patient with emphysema is sometimes classified as a “**pink puffer**,” and the patient with chronic bronchitis is sometimes classified as a “**blue bloater**,”.

“Pink Puffer”

The term *pink puffer* is derived from the reddish complexion and the “puffing” (pursed-lip breathing) commonly seen in the patient with emphysema. The major pathophysiologic mechanisms responsible for the red complexion and puffing are the following:

- Emphysema is caused by the progressive destruction of the distal airways and pulmonary capillaries.

- The progressive elimination of the distal airways and pulmonary capillaries leads to ventilation perfusion mismatches.
- To compensate for these ventilation-perfusion ratio mismatches, the patient 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, however, the patient's oxygenation status decreases and the carbon dioxide level increases.
- Thus, the patient with emphysema, who has both a red complexion and a rapid respiratory rate, is called a *pink puffer*.

In addition to the marked dyspnea and ruddy complexion, the pink puffer tends to be thin (because of the muscle wasting and weight loss associated with the increased work of breathing), has a barrel chest (because of hyperinflated lungs), uses accessory muscles of inspiration, and exhales through pursed lips.

“Blue Bloater”

The term *blue bloater* is derived from the cyanosis—the bluish color of the lips and skin—commonly seen in the patient with chronic bronchitis.

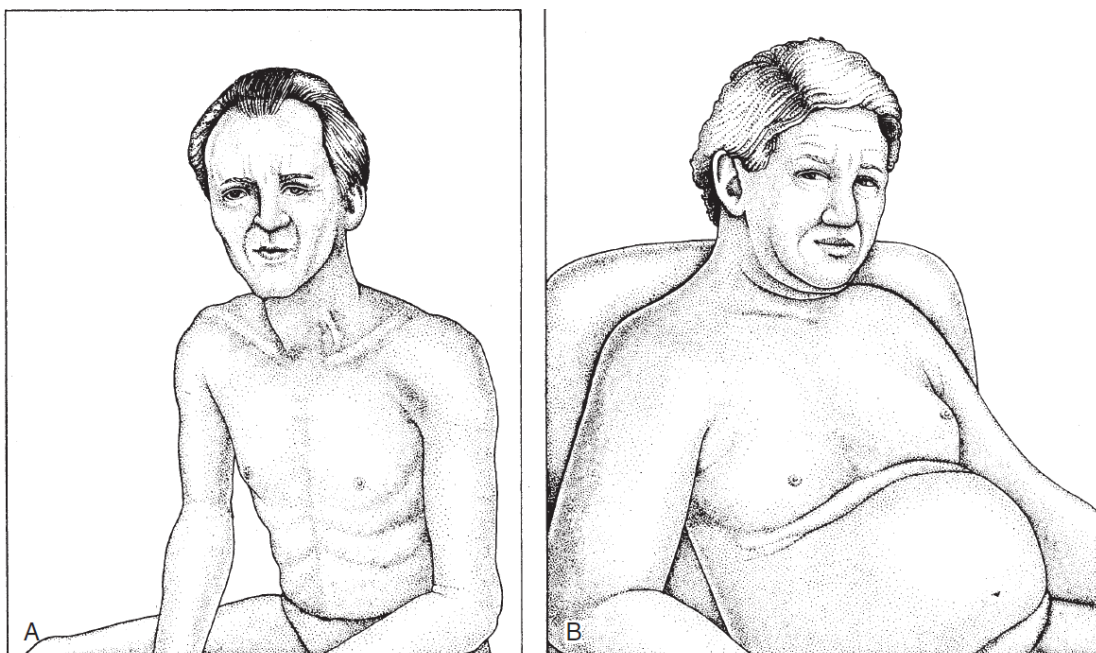


Figure 6-14 Comparison of appearance of individuals with emphysema-dominant (*left*) and chronic bronchitis-dominant (*right*) chronic obstructive pulmonary disease. (From Goodman CC, Snyder TEK. *Differential Diagnosis in Physical Therapy*. Philadelphia, 1990, Saunders.)

TABLE 12-4 Key Features Distinguishing Emphysema from Chronic Bronchitis*

Clinical Manifestation	Emphysema (Type A COPD: Pink Puffer)	Chronic Bronchitis (Type B COPD: Blue Bloater)
Inspection		
Body build	Thin	Stocky, overweight
Barrel chest	Common, classic sign	Normal
Respiratory pattern	Hyperventilation and marked dyspnea; often occurs at rest Late stage: diminished respiratory drive and hypoventilation	Diminished respiratory drive Hypoventilation common, with resultant hypoxia and hypercapnia
Pursed-lip breathing	Common	Uncommon
Cough	Uncommon	Common; classic sign
Sputum	Uncommon	Common; classic sign Copious amounts, purulent
Cyanosis	Uncommon (reddish skin)	Common
Peripheral edema	Uncommon	Common Right-sided heart failure
Neck vein distention	Uncommon	Common Right-sided heart failure
Use of accessory muscles	Common	Uncommon
Auscultation		
	Decreased breath sounds, decreased heart sounds, prolonged expiration	Wheezes, crackles, depending on severity of disease
Percussion		
	Hyper resonance	Normal
Laboratory Tests		
Chest radiograph	Hyperinflation, narrow mediastinum, normal or small vertical heart, low flat diaphragm, presence of blebs or bullae	Congested lung fields, densities, increased bronchial vascular markings, enlarged horizontal heart
Polycythemia	Uncommon	Common
Infections	Occasionally	Common
Pulmonary Function Study		
DLCO and DL/VA	Decreased	Often normal
Other		
Pulmonary hypertension	Uncommon	Common
Cor pulmonale	Uncommon	Common Right-sided heart failure

*The clinical features of emphysema and chronic bronchitis are not always clear-cut because many patients have a combined disease process (COPD—this is especially the case during the late stages of emphysema and chronic bronchitis).

Diagnosis/ Investigations

The following aid in the diagnosis of COPD.

- Symptoms of dyspnoea, chronic cough and/ or sputum production
- History of exposure to tobacco smoke, noxious fumes, occupational dusts and chemicals
- Older age
- Lung function tests

Spirometry: forced expiratory volume in 1 second (FEV1) to forced vital capacity (FVC) ratio <0.7 indicating airway obstruction, combined with the following FEV1 per cent age predicted to indicate disease severity. Transfer factor for carbon monoxide (TLCO) is often reduced due to the loss of surface area for gas exchange as a consequence of destruction of the alveolar walls and associated capillary bed.

- Chest radiograph showing signs of hyperinflation

Medical Management

- Smoking Cessation
- Vaccinations
- Medications

Medications for people with COPD include inhaled short- and long-acting bronchodilators, inhaled corticosteroids and antibiotics during an acute infective exacerbation.

- Surgical Interventions
- Oxygen Therapy
- Ventilatory Support

Non-invasive ventilation (NIV) may be used in some patients with chronic respiratory failure, obstructive sleep apnoea or during an acute exacerbation.

Physical Therapy Treatment

- Secretion clearance techniques, when indicated (postural drainage, acapella instruction, and active cycle of breathing)
- Controlled breathing techniques (pursed lips and paced breathing) at rest
- Controlled breathing techniques coordinated with position changes, ambulation, and stair climbing
- Ambulation with use of a rolling walker
- Instruction in the use of recovery from shortness of breath positions
- Endurance exercise training
- Strength and weight training
- Thoracic stretching exercises
- Postural reeducation to avoid round-shouldered postures.

REFERENCES

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