Chapter 18
Chronic Obstructive Pulmonary Disease

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Name and Synonyms

Chronic Obstructive Pulmonary Disease; COPD; Emphysema

Incidence/Epidemiology

- COPD affects about 5 % of the U.S. population.
- COPD is reported as the third- or fourth-leading cause of death in the U.S, depending upon the survey. COPD is directly responsible for about 120,000 deaths per year in the U.S.
- COPD is associated with a very high rate of medical resource utilization. The majority of these costs are for hospitalization of acute exacerbations of COPD.
- In the U.S., nearly 2 % of all hospital admissions are directly attributable to COPD. It is considered a major contributing factor in another 9 % of hospital admissions. In patients older than 65, the percentage of all hospital admission related to COPD approaches 20 %.
- COPD is the only major cause of death that is increasing in the U.S.

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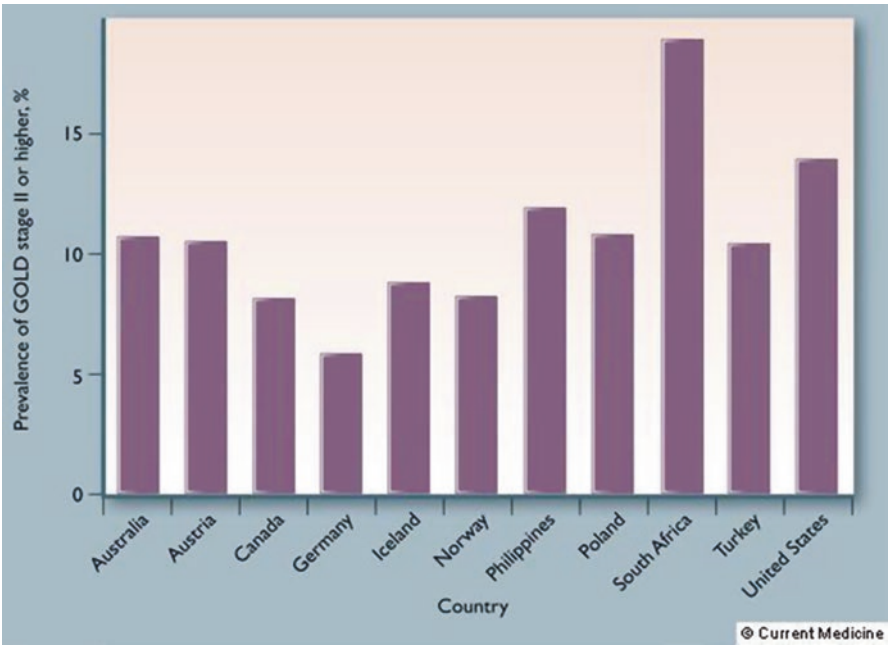
- The incidence and mortality of COPD in women is increasing worldwide. This is likely related to the increased incidence of smoking among women during the last 50 years.
- Tobacco smoke is the major risk factor for developing COPD, but only 15 % of smokers will develop COPD.
- Alpha-1 antitrypsin deficiency accounts for less than 1 % of COPD cases.



Worldwide prevalence of chronic obstructive pulmonary disease (COPD). The prevalence of COPD (in 2007) in different geographic regions based on the Burden of Obstructive Lung Disease data [Buist AS, McBurnie MA, Vollmer WM, et al. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. Lancet. 2007; 370:741 -750.] is presented based on results for specific cities within the countries listed. The reasons for these regional differences are not entirely clear and may be due to different environmental exposures (smoking, biomass, occupation), as well as possibly genetic factors. [Cho M, Silverman E. Genetics and Racial, Ethnic, and Gender Characteristics of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption from original*

Differential Diagnosis

- The primary symptoms of COPD are dyspnea (usually exertional early in the disease course), cough, and sputum production.

- The differential diagnosis of these symptoms is broad, and includes many diseases and organ symptoms.
- Patients may present with worsening fatigue, exercise or activity intolerance, and often complain of daily cough for many months.
- In patients without a current diagnosis of COPD, the differential includes:
    - Congestive heart failure
    - Interstitial lung disease
    - Thromboembolic Disease (pulmonary embolism)
    - Asthma
    - Bronchiectasis
    - Tuberculosis
    - Bronchiolitis
    - Airway obstruction from bronchogenic or metastatic cancer, lymphadenopathy (sarcoidosis), and tracheal stenosis/scarring.
- It is important to remember that many of these disorders can occur together.
- In patients with an existing diagnosis of COPD, who present with acute dyspnea (exacerbation of COPD), the cause of the exacerbation needs to be investigated. The differential for acute dyspnea in these patients is also broad, and includes:
    - CHF/pulmonary edema
    - Acute coronary syndrome
    - Pneumonia
    - Viral respiratory infection
    - Pulmonary embolism
    - Pleural effusion
    - Pneumothorax
    - Pericardial effusion
    - Mucous plugging of bronchi
    - Rib fracture from severe coughing
    - Electrolyte imbalance (especially hypokalemia and hypocalcemia)

Pathophysiology and Etiology

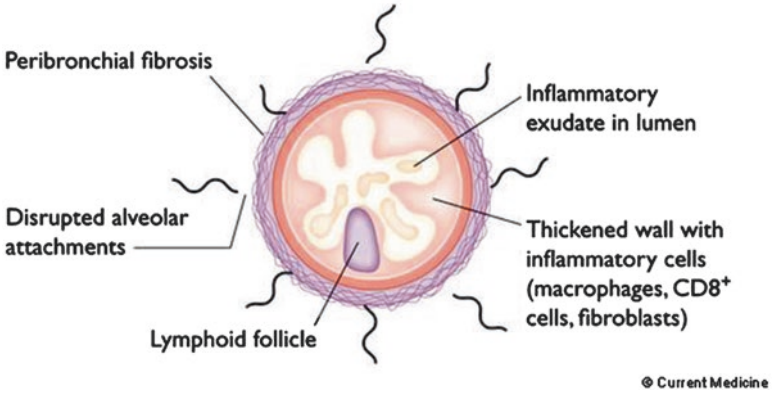
- Chronic obstructive pulmonary disease (COPD) is a disease characterized by chronic, progressive airflow obstruction.
- The Global Initiative for Chronic Obstructive Lung Disease (GOLD), sponsored by the National Heart, Lung, and Blood Institute (NHLBI) and the World Health Organization (WHO), defines COPD as follows: “Chronic obstructive pulmonary disease (COPD), a common preventable and treatable disease, is characterized by airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients.”

Global Initiative for Chronic Obstructive Lung Disease, GOLD [3]: $FEV_1/FVC < 70\%$		
I:	Mild COPD	$FEV_1 \geq 80\%$ predicted
II:	Moderate COPD	FEV_1 50- < 80% predicted
III:	Severe COPD	FEV_1 30- < 50% predicted
IV:	Very severe COPD	$FEV_1 < 30\%$ predicted
British Thoracic Society, BTS [2]: $FEV_1/VC < 70\%$ and $FEV_1 < 80\%$ predicted		
I:	Mild COPD	FEV_1 60- < 80% predicted
II:	Moderate COPD	FEV_1 40-59% predicted
III:	Severe COPD	$FEV_1 < 40\%$ predicted

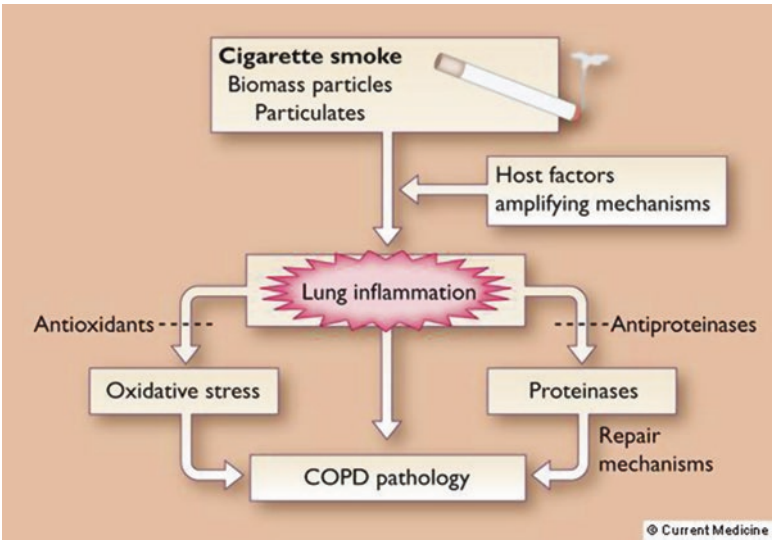
A group labelled BTS stage 0 was created for subjects with $FEV_1 \geq 80\%$ predicted: i.e. identical with mild COPD according to the GOLD criteria.

Severity criteria of COPD. [3] BTS: BTS guidelines for the management of chronic obstructive pulmonary disease. The COPD Guidelines Group of the Standards of Care Committee of the BTS. Thorax 1997, 52:S1-28. [2] Pauwels RA, Buist AS, Calverley PMA, Jenkins CR, Hurd SS: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: NHLBI and WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD): executive summary. Respiratory Care 2001, 46:798-825. [From article: Health-related quality of life is related to COPD disease severity. Health and Quality of Life Outcomes. 2005 Sep 9;3(1):56. https://doi.org/10.1186/1477-7525-3-56, at http://link.springer.com/article/10.1186%2F1477-7525-3-56/fulltext.html; by Elisabeth Ståhl, Anne Lindberg, Sven-Arne Jansson, Eva Rönmark, Klas Svensson, Fredrik Andersson, Claes-Göran Löfdahl, Bo Lundbäck, © Ståhl et al; licensee BioMed Central Ltd. 2005; licensed under Creative Commons Attribution License BY 2.0 http://creativecommons.org/licenses/by/2.0]

- The pathophysiology of COPD involves the airways, lung parenchyma, and pulmonary vasculature.
  - Airways: The most significant pathologic change in the airways involves chronic inflammation from exposure to cigarette smoke and other pollutants, which leads to several permanent anatomic changes such as:
    - Mucous gland hyperplasia and increased numbers of goblet cells, resulting in increased mucous secretion. Increased mucous secretion and stasis leads to pathogenic bacterial colonization.
    - Increased mucous-secreting and goblet cells replace surfactant-secreting cells, which augments collapse and destruction of the small airways.
    - Increased induction of inflammatory cells with increased local protease production, accelerating tissue damage and breakdown.
    - Fibrosis with loss of elastic recoil, narrowing, collapse, and subsequent destruction and reduction in the number of small airways.
    - There is also squamous metaplasia within the airways resulting in an increased risk for cancer.

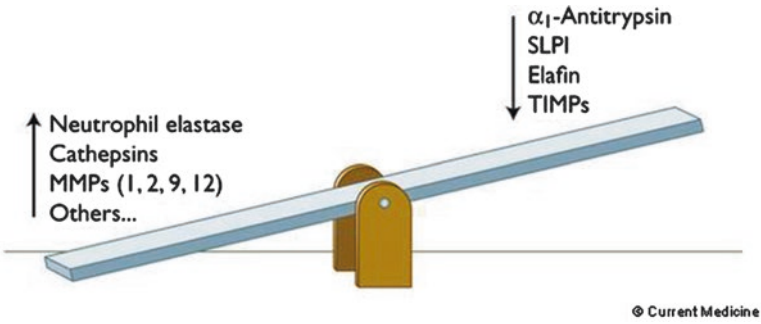


Overview of small airways in COPD: schematic view [Barnes PJ. Pathophysiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption adapted from original*

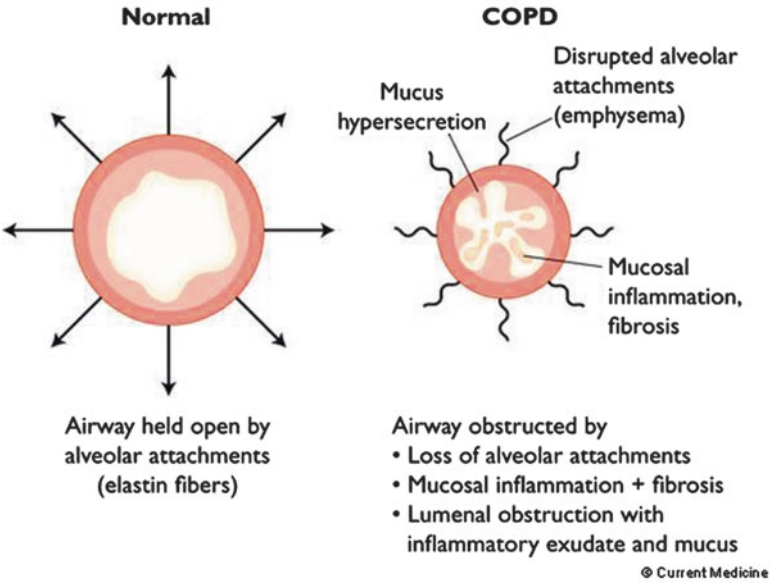


Link between inflammation and airway obstruction in chronic obstructive pulmonary disease (COPD). Chronic exposure to inhaled cigarette smoke and biomass particles (wood smoke) results in chronic inflammation of the lungs. Genetic and other unknown factors are responsible for the increased susceptibility to inhaled irritants. Inflammation generates reactive oxygen species (oxidative stress), which is normally counteracted by endogenous antioxidant mechanisms, but these may be defective in COPD. Inflammation also activates proteinases, which result in connective tissue destruction. This may be countered by antiproteinases and repair mechanisms that may also be defective in COPD.

[Barnes PJ. Pathophysiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption from original*



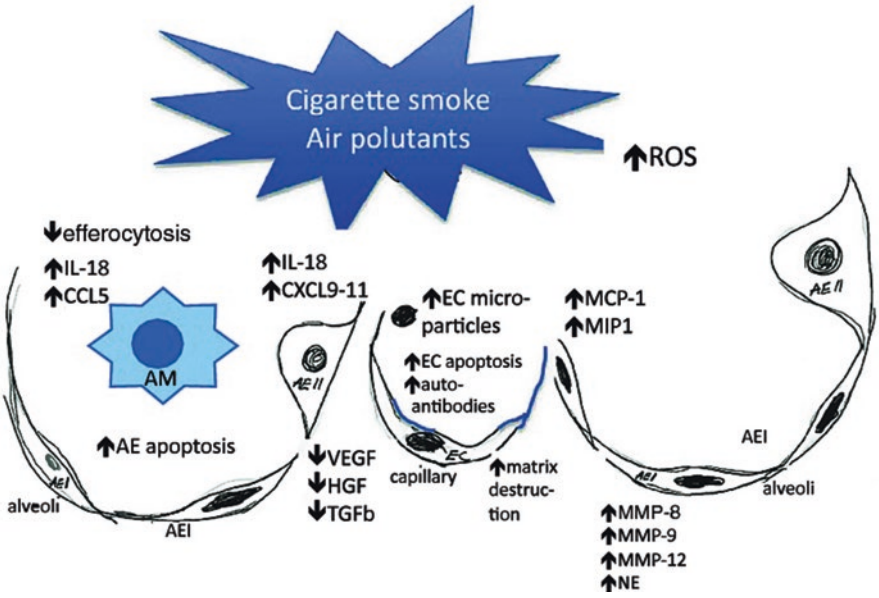
Protease-antiprotease imbalance in COPD: schematic view. Protease–antiprotease imbalance in chronic obstructive pulmonary disease (COPD). In COPD the balance appears to be tipped in favor of increased proteolysis, either because of an increase in proteases, including neutrophil elastase, cathepsins, and matrix metalloproteinases (MMPs), or a deficiency in antiproteases, which may include α_1-antitrypsin, elafin, secretory leukoprotease inhibitor (SLPI), and tissue inhibitors of matrix metalloproteinases (TIMPs). [Barnes PJ. Pathophysiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption from original*



Mechanisms of airflow limitation in COPD: schematic view. Mechanisms of airflow limitation in chronic obstructive pulmonary disease (COPD). The airway in normal

subjects is distended by alveolar attachments, which contain elastin fibers during expiration, allowing alveolar emptying and lung deflation. In COPD these attachments are disrupted due to emphysema, thus contributing to airway closure during expiration, trapping gas in the alveoli and resulting in hyperinflation. Peripheral airways are also obstructed and distorted by airway inflammation and peribronchiolar fibrosis (chronic obstructive bronchiolitis), and by occlusion of the airway lumen by inflammatory exudate and mucus secretions that may be trapped in the airways due to poor mucociliary clearance. [Barnes PJ. Pathophysiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption from original*

- Lung Parenchyma. The lung parenchymal changes are also called emphysema. The changes mostly affect the acinus, the functional unit distal to the terminal bronchiole that includes the respiratory bronchiole, alveolar ducts, alveolar sacs, and alveoli. There is destruction of this unit leading initially to collapse and then subsequent enlargement as air becomes trapped within the destroyed unit. The loss of elastic recoil prevents the air from escaping. There are three subtypes of emphysema:
  - Centrilobular: This involves the proximal acinus (predominately the respiratory bronchioles.) It is the form most strongly associated with cigarette smoking.
  - Panacinar: This involves all parts of the acinus, and is mostly seen with alpha-1 ant-trypsin deficiency.
  - Distal (paraseptal): Affects predominately the alveolar ducts.
- Pulmonary vasculature. Chronic hypoxia leads to reflex vasoconstriction of the small pulmonary arteries, which results in intimal hyperplasia, smooth muscle hypertrophy and hyperplasia, and ultimately pulmonary hypertension.



Destruction of the lung maintenance program in emphysema. Chronic exposure to cigarette smoke leads to a change in the local milieu: increased release of proinflammatory mediators (IL-8, IL-18, CCL5, IL-8, IFNgama, MIP1) that contribute to paracrine and autocrine signaling, impairment in efferocytosis, decreased levels of growth and survival factors (most notably VEGF and HGF), and extracellular matrix degradation (increased levels of matrix metalloproteases MMP-9 and MMP-12). Increased apoptosis of endothelial and epithelial cells might contribute to autoantibody formation. Reduced levels of TGF might signal delay repair processes and contribute to inflammatory cell activation. AM – alveolar macrophage; AEI – alveoli type I cell; AEII – alveoli type II cell; EC – capillary endothelial cell; ROS – reactive oxygen species [Taraseviciene-Stewart L, Voelkel NF. Immunopathology of COPD. In: Rogers TJ, Criner GJ, Cornwell WD, editors. Smoking and Lung Inflammation [Internet]. Springer New York; 2013 [cited 2016 May 16]. p. 1–27. Available from: http://link.springer.com/chapter/10.1007/978-1-4614-7351-0_1] *Caption from original*

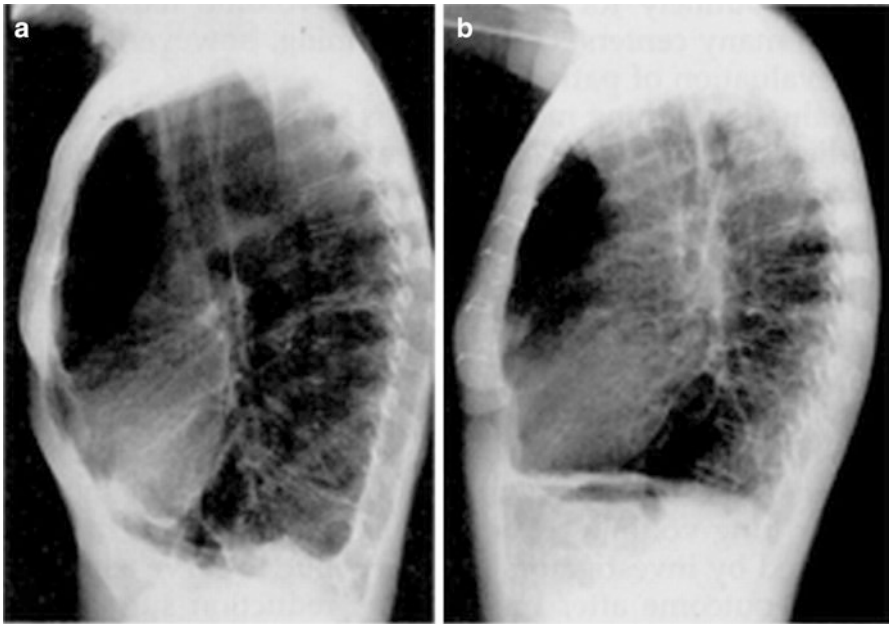
- The single largest risk factor for the development of COPD is cigarette smoking. In the United States, nearly 80 % of patients diagnosed with COPD have history of cigarette smoking. The amount and duration of smoking are the largest predictors of disease severity. The single most reliable predictor for finding airflow obstruction on spirometric testing is a greater than 40 packs-per-year history of cigarette use.
- However, only 15 % of smokers will develop COPD.
- Smoking is the largest risk factor for the development of COPD, but not the only risk factor. Twenty percent of patients with COPD do not have a significant history of cigarette smoking. Other causes of COPD include occupational exposures (such as coal mining, gold mining, and cotton dust). There are also genetic causes, such as alpha-1 anti-trypsin deficiency, although this accounts for less than 1 % of cases of COPD.
- There is currently no strong evidence linking air pollution or second-hand smoke to the development of COPD.

Presentation

Typical/"Classic"

- The most common presenting symptoms of COPD are exertional dyspnea and chronic cough with sputum production.
- COPD is a slowly progressive disease with a prolonged asymptomatic phase. Patients are often unaware of the dyspnea, or just relate it to aging or poor fitness. They will often adjust their activities to limit the symptoms of dyspnea.
- Wheezing is often a later symptom.

- The course is one of slow progression with intermittent exacerbations of dyspnea and cough.
- Early in the course of the disease, the patient may have no physical signs of the disease.
- As the disease progresses, patients may develop the more classic signs of COPD, such as a barrel chest (increased AP diameter from air trapping), prolonged expiration with expiratory wheezing, decreased breath sounds, tachypnea, and pursed lip breathing.



A lateral chest X-ray is shown. (a) Depicts the patient before lung volume reduction surgery (LVRS): the thorax is barrel-shaped with high transparency of the lungs, a large retrosternal air-filled space, and a flattened to concave diaphragm. The chest X-ray of the same subject (b) is shown 3 months after LVRS: the lung fields are less transparent, the air-filled retrosternal space decreased significantly, and the diaphragm exhibits an almost normal, convex shape. [Sullivan EA. Lung Volume Reduction. In: Slinger P, editor. Principles and Practice of Anesthesia for Thoracic Surgery [Internet]. Springer New York; 2011 [cited 2016 May 16]. p. 511–21. Available from: http://link.springer.com/chapter/10.1007/978-1-4419-0184-2_36] *Caption from original*

- Patients will often be noted to need to sit up and lean forward with their hands on their knees (tripod position) as this increases accessory muscle use (sternocleidomastoid, scalene, and intercostal muscles) and diaphragmatic excursion, both of which help to improve lung volumes.

- Patients with advanced disease will often have significant wasting with loss of muscle mass and subcutaneous fat. This is an independent predictor of a very poor prognosis.
- Clubbing of the digits is not a sign of COPD. If a patient with COPD develops clubbing, there is usually another cause, such as the interval development of lung cancer.

Atypical

- COPD less commonly presents initially with wheezing.
- COPD can present with chest tightness as the predominant symptom.

Primary Differential Considerations

- Initial consideration in patients with an acute presentation should be given to these differential diagnoses:
  - Asthma
  - Congestive heart failure
  - Pulmonary hypertension
  - Pulmonary embolism
  - Pneumonia

History and Physical Exam

Findings That Confirm Diagnosis

- There are no historical or physical exam findings that can confirm COPD. There is a large overlap with other disease states that cause dyspnea, such as asthma, congestive heart failure, and bronchiectasis.
- The diagnosis can be nearly confirmed when a patient presents with the typical symptoms of dyspnea on exertion with a chronic cough and sputum production associated with findings on spirometry of a forced expiratory volume in one second (FEV_1) less than 80 % of predicted and a ratio of FEV_1 to the forced vital capacity (FVC) of <0.7.

Assess and Monitor Disease: Key Points
Consider a diagnosis of COPD in any patient with a cough, dyspnea, or exposure to risk factors and confirm with spirometry.
Spirometry is the gold standard for the diagnosis and assessment of COPD and a postbronchodilator $FEV_1/FVC < 0.70$ confirms the presence of airflow limitation that is not fully reversible.
Health care workers involved in the diagnosis and management of COPD should have access to spirometry.
Severity of COPD is determined by symptoms, spirometric abnormalities, and complications. Changes in therapy should also be based on these factors.
Blood gas measurements should be made in patients with $FEV_1 < 50\%$ predicted or in those with evidence for right heart failure.
Comorbidities are common in COPD and are often the cause of death. They should be actively identified.

Assessment of COPD. [Dransfield MT. Diagnosis of COPD and the GOLD Guidelines. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption adapted from original*

Factors That Suggest Diagnosis

- A patient who presents with the typical symptoms of dyspnea, cough, and sputum production, and has a greater than 40 pack-per-year history of cigarette smoking, has a high likelihood of having COPD.

Factors That Exclude Diagnosis

- Normal spirometric findings can exclude the diagnosis in a patient with the classic symptoms.

Ancillary Studies

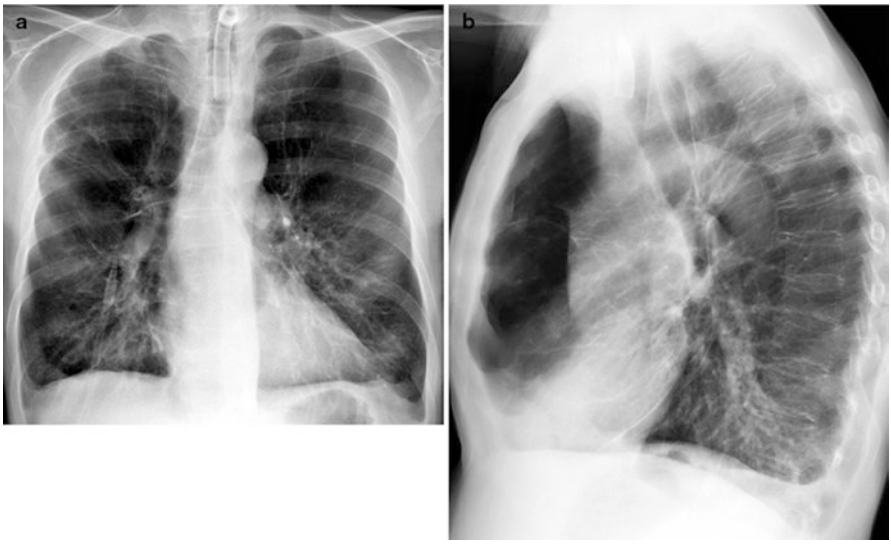
Laboratory

- There are no lab tests that are diagnostic for COPD. However, many lab tests are helpful in the evaluation of the dyspneic patient to evaluate for other causes of the dyspnea.
- A complete blood count can help assess for anemia as the cause of dyspnea. Patients with advanced COPD may demonstrate polycythemia induced by chronic hypoxemia.

- A BNP (brain natriuretic peptide) level may help to distinguish between COPD and congestive heart failure as the cause of dyspnea. It is important to remember, however, that these diseases may often be present in the same patient.
- An alpha-1 anti-trypsin level should be measured in any patient with early-onset (before age 45) COPD, a non-smoker with COPD, or a family history of premature development of COPD.

Imaging

- Plain chest x-rays are unreliable in the diagnosis of COPD. Only about one-half of patients with moderate COPD will have any findings of COPD on a chest x-ray.
- The classic CXR findings of COPD include flattened diaphragms, an increased antero-posterior diameter of the chest, and increased radiolucency of the lungs. These are usually only seen in moderate-to-severe COPD, and are due to hyperinflation and air trapping.
- CXR can also be used to evaluate for alternative diagnoses (such as lung cancer, CHF, bronchiectasis), co-morbidities (the list also includes lung cancer, CHF, and bronchiectasis), and complications (such as pneumonia, pneumothorax) of COPD that are apparent on chest x-ray.
- CT is more sensitive at detecting the pathologic changes of COPD, but is not often needed to make the diagnosis. CT can be very helpful in the evaluation for alternative diagnoses, co-morbidities, and complications of COPD.

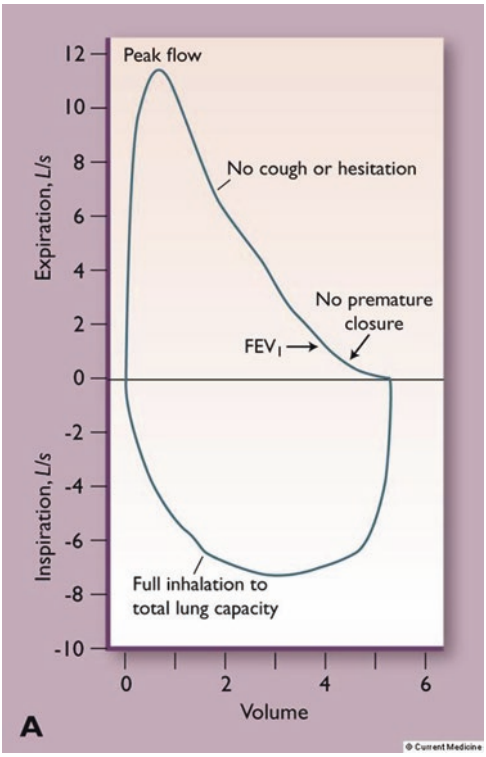


Chest X-rays of a 75-year-old patient with a clinical diagnosis of COPD. Posteroanterior (a) and lateral views (b) show radiographic signs consistent with pulmonary emphysema. Thickening of the bronchial wall is also evident in the lung bases. Tracheostomy was previously placed due to respiratory failure [Larici AR, Franchi P, Occhipinti M, Devicienti E, Mereu M, del Ciello A, Bonomo L. Airway Disease. In: Guglielmi G, Peh WCG, Guermazi A, editors. Geriatric Imaging [Internet]. Berlin, Heidelberg: Springer Berlin Heidelberg; 2013 [cited 2016 May 16]. p. 319–52. Available from: http://link.springer.com/10.1007/978-3-642-35579-0_14] *Caption from original*

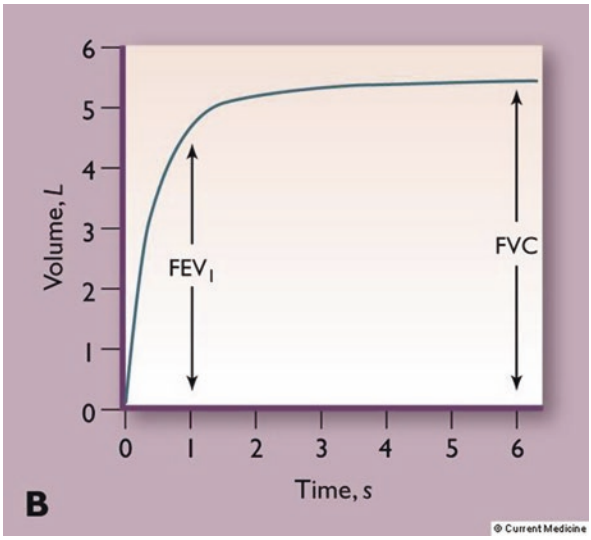
Pulmonary Function Tests

- PFT's, especially spirometry, are the most helpful studies in diagnosing COPD. They are also useful in assessing the severity of disease, response to treatment, and for following progression.
- Spirometry (the measuring of breathing) is the most useful pulmonary function test in diagnosing and following COPD. Specifically, the forced expiratory volume in one second (FEV1), and the forced vital capacity and their ratio are the most helpful diagnostic tests for COPD.
- Spirometry performed both before and after inhaled bronchodilator use can determine whether airflow limitation is present, and whether it is at all reversible. Airflow limitation that is reversible is characteristic of asthma; airflow that is only partially reversible or is irreversible is consistent with COPD.
- Generally, COPD is felt to be present in a patient with an FEV1 less than 80 % of predicted, and and FEV1/FVC ratio of less than 0.7.

https://www.youtube.com/watch?v=s8pXdtp_Duw
Video demonstrating spirometry procedure, including the patient interview.



Spirometry in diagnosis of COPD [Hegewald M, Crapo R, Jensen R. Clinical Physiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption adapted from original*



Spirometry in diagnosis of COPD [Hegewald M, Crapo R, Jensen R. Clinical Physiology of COPD. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. 2009 edition. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption adapted from original*

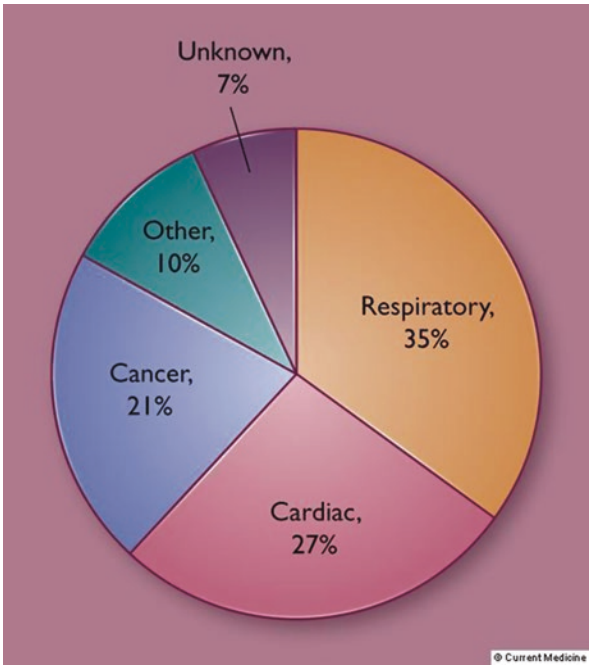
Special Populations

Age

- COPD is a slowly progressive disease that often presents later in life. It is uncommon to diagnose before the sixth decade of life.
- Patients who are diagnosed with COPD before the age of 45 need to be evaluated for alpha-one antitrypsin deficiency.

Co-morbidities

- As a disease of older patients, COPD is often is seen in combination with other diseases, such as coronary artery disease, congestive heart failure, and cancer.



Causes of death in patients with COPD [Make BJ, Crapo JD. The Worldwide Epidemic of COPD: Clinical Phenotypes. In: Crapo JD, editor. Atlas of Chronic Obstructive Pulmonary Disease. Philadelphia, PA: Current Medicine Group; 2009. 160 p. ISBN: 978-1-57340-294-1] *Caption adapted from original*

Pitfalls in Diagnosis

Critical Steps Not to Miss

- It is imperative to consider the diagnosis, especially early in the course of the disease when the symptoms may be non-specific and subtle. Interventions such as smoking cessation can help to slow and stop the progression of disease if instituted early in the course.
- It is imperative to consider to alternative diagnoses, such as lung cancer and congestive heart failure.

Mimics

- Any disease that causes dyspnea can mimic COPD.
- Early COPD can sometimes be confused with atypical angina, as COPD, like angina, can cause chest tightness and dyspnea. Smoking is a significant risk factor for both diseases.
- Early in the course it is easily mimicked by asthma (although asthma has reversible airflow obstruction and COPD has irreversible airflow obstruction).
- Later in the course, COPD can mimic CHF, lung cancer with airway obstruction, bronchiectasis, pulmonary fibrosis, and other disorders causing dyspnea and cough.

Time-Dependent Interventions

- COPD cannot be reversed, so it is important to initiate treatment as early as possible to try and prevent progression of symptoms and pathology.
- Smoking cessation is critically important to these patients. It is also very difficult and may require a multi-modal treatment approach that includes pharmacologic and behavioral interventions.

Overall Principles of Treatment

- Therapy for compensated COPD involves numerous modalities including: oxygen therapy when indicated, bronchodilators, corticosteroids, reducing mucous secretion, smoking cessation, and pulmonary rehabilitation.
- Oxygen therapy in chronically hypoxic patients reduces mortality. The goal is to keep the PaO2 $\leq$60 mmHg of oxygen saturation $\geq$90 % at rest. The generally accepted criteria for oxygen therapy are: PaO2 $\leq$55 mmHg, an oxygen saturation of $\leq$88 % on room air, or a PaO2 of 56–59 mmHg in the presence of pulmonary hypertension, cor pulmonale, and polycythemia.
- Bronchodilator therapy does not affect the progression of the disease, but it can provide symptomatic relief and control and reduce exacerbations. Bronchodilator therapy also can improve quality of life. Patients would typically be maintained on long-acting inhaled beta agonists such as salmeterol and formoterol. Short-acting beta agonists such as albuterol are usually reserved for symptom control and for use during acute exacerbations. Inhaled anticholinergics (ipratropium) also cause bronchodilatation. The use of inhaled beta agonists combined with an inhaled anticholinergic improves FEV1 and symptoms better than either of these alone.

- Systemic corticosteroids can be useful in helping to control acute exacerbations, but most authorities don't recommend their long-term use. Only about 20–30 % of patients will note any improvement with the use of oral steroids. Some patients may improve with the use of inhaled steroids, especially those with an FEV1 <50 % of predicted.
- The only measures shown to help with mucous handling are adequate hydration and room humidification. Antitussives, antihistamines, and decongestants are all drying agents, and their use should be limited. Mucolytics and expectorants are of no clear benefit.
- Smoking cessation is the only intervention proven to reduce the rate of disease progression. Smoking cessation also reduces mortality. A multi-modal approach is often necessary and may include both pharmacologic and behavioral therapies.
- Pulmonary rehabilitation can increase exercise tolerance and improve quality of life, and is indicated for moderate-to-severe COPD.
- It is important that patients with COPD receive a pneumococcal vaccine, and also receive yearly influenza vaccination.

Disease Course

- COPD is a chronic, slowly progressive disease. It is marked by periods of relative stability interrupted by acute exacerbations, and a slow, steady decline in lung function.
- The frequency of exacerbations is a surrogate marker for disease progression.

Related Evidence

Papers of particular interest have been highlighted as:
*** Of key importance*

Practice Guideline

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