Supportive module 3 "Basics of diagnosis, treatment and prevention of major pulmonary diseases"

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<td>Respiratory failure. Chronic pulmonary heart</td>
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Approved at a meeting of the Department of Internal Medicine protocol № 1/1  30.08.2016
Supportive module 3 "Basics of diagnosis, treatment and prevention of major pulmonary diseases"

Chronic Obstructive Pulmonary Disease

LECTURE IN INTERNAL MEDICINE FOR IV COURSE STUDENTS

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V.N. Karazin National University Medical School’ Internal Medicine Dept.
Plan of the Lecture

- Definition
- Epidemiology
- Risk Factors and Etiology
- Mechanisms
- Classification
- Clinical presentation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic guidelines

https://s-media-cache-ak0.pinimg.com/originals/27/8c/c4/278cc463c56cc824d9eb2666e8f3863d.jpg
Definition

Chronic Obstructive Pulmonary Disease (COPD) is a type of lung disease by a decline in lung function over time in which subsets of patients may have dominant features of chronic bronchitis and/or emphysema characterized by long-term airflow obstruction that is not fully reversible with the main symptoms include shortness of breath and cough with sputum production typically worsens over time, some significant extra-pulmonary effects, and important comorbidities which may contribute to the severity of the disease in individual patients.

Chronic bronchitis is defined clinically as the presence of a chronic productive cough for 3 months during each of 2 consecutive years (other causes of cough being excluded).

Emphysema is defined as an abnormal, permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis.

http://www.who.int/respiratory/copd/GOLD_WR_06.pdf
en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease
emedicine.medscape.com/article/297664-overview?pa=%2FOOPqamlew%2Fp1dAMq%2BJKjXjrTEuzO%2FMsNWCiPMRsOneLeqn1hB%2BsABfchCz uV8Svl8jYv73GUYW5rsbWA%3D%3D#showall
Epidemiology

- Globally, as of 2010, COPD affected approximately 329 million people (4.8% of the population)
- In England, an estimated 0.84 million people (of 50 million) have a diagnosis of COPD; this translates into approximately one person in 59 receiving a diagnosis of COPD at some point in their lives
- In the United States, approximately 6.3% of the adult population, totaling approximately 15 million people, have been diagnosed with COPD
- The disease affects men and women almost equally, as there has been increased tobacco use among women in the developed world
- COPD is more common in older people
- The number of deaths from COPD in 2012 became the third leading cause of death.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Epidemiology
Epidemiology

COPD - Deaths / 1000 1990 Data projected to 2000.
Epidemiology

Risk Factors and Etiology

- Smoking: smokers are 13 times more likely to die from the COPD
- Air Pollution: indoor and outdoor pollutants
- Occupational Dusts and Chemicals: industrial dust, chemicals, and gases
- Genetics: in rare cases, genetic factors can cause people who have never smoked (a lack of the protein $\alpha_1$-antitrypsin (AAT), Salla disease, etc.)
- Intravenous drug use: the pulmonary vascular damage that results from the insoluble filler contained in methadone or methylphenidate
- Connective tissue disorders: Cutis laxa, Marfan syndrome, Ehlers-Danlos syndrome, Vasculitis syndrome, etc.
- Immunodeficiency syndromes
- Age: incidence COPD increases with age.

http://www.healthline.com/health/copd/risk-factors#Overview1
Risk Factors and Etiology

Healthy lungs

Healthy alveoli

Smoking and Emphysema

Inflammatory response triggered

Harmful particles trapped in alveoli

Large air cavity lined with carbon deposits formed

Inflamatory chemicals dissolve alveolar septum

Emphysema

https://www.consumerhealthdigest.com/wp-content/uploads/2015/01/smoking-asthma.jpg
Pathologic changes in COPD occur in the large (central) airways, the small (peripheral) bronchioles, and the lung parenchyma. Most cases of COPD are the result of exposure to noxious stimuli, most often cigarette smoke. The normal inflammatory response is amplified in persons prone to COPD development. Increased numbers of activated polymorphonuclear leukocytes and macrophages release elastases in a manner that cannot be counteracted effectively by antiproteases, resulting in lung destruction.
Mechanism
General 2

• The primary offender has been found to be human leukocyte elastase, with synergistic roles suggested for proteinase-3 and macrophage-derived matrix metalloproteinases (MMPs), cysteine proteinases, and a plasminogen activator.

• Additionally, increased oxidative stress caused by free radicals in cigarette smoke, the oxidants released by phagocytes, and polymorphonuclear leukocytes all may lead to apoptosis or necrosis of exposed cells.

• Accelerated aging and autoimmune mechanisms have also been proposed.
Mechanism
General 2

• Cigarette smoke causes neutrophil influx, which is required for the secretion of MMPs; this suggests, that neutrophils and macrophages are required for the development of emphysema
• In addition to macrophages, T lymphocytes, particularly CD8+, play an important role in the pathogenesis of smoking-induced airflow limitation
• The dysregulation of apoptosis and defective clearance of apoptotic cells by macrophages play a prominent role in airway inflammation, particularly in emphysema
• In patients with stable COPD without known cardiovascular disease, there is a high prevalence of microalbuminuria, which is associated with hypoxemia independent of other risk factors.
Mechanism

Schema 1

http://www.nature.com/nri/journal/v8/n3/images/nri2254-f2.jpg

Immunology of COPD.
Multifaceted mechanisms in COPD.
Mechanism
Chronic Bronchitis 1

• Mucous gland hyperplasia is the histologic hallmark of chronic bronchitis
• Airway structural changes include atrophy, focal squamous metaplasia, ciliary abnormalities, variable amounts of airway smooth muscle hyperplasia, inflammation, and bronchial wall thickening
• Damage to the endothelium impairs the mucociliary response that clears bacteria and mucus
• Inflammation and secretions provide the obstructive component of chronic bronchitis
• Neutrophilia develops in the airway lumen, and neutrophilic infiltrates accumulate in the submucosa
Mechanism
Chronic Bronchitis 2

- The respiratory bronchioles display a mononuclear inflammatory process, lumen occlusion by mucus plugging, goblet cell metaplasia, smooth muscle hyperplasia, and distortion due to fibrosis.
- These changes, combined with loss of supporting alveolar attachments, cause airflow limitation by allowing airway walls to deform and narrow the airway lumen.
- In contrast to emphysema, chronic bronchitis is associated with a relatively undamaged pulmonary capillary bed.
- Eventually, hypercapnia and respiratory acidosis develop, leading to pulmonary artery vasoconstriction and cor pulmonale.
- With the ensuing hypoxemia, polycythemia, and increased CO₂ retention, these patients have signs of right heart failure and are known as "blue bloaters."

http://emedicine.medscape.com/article/297664-overview?pa=%2FOOPqamlew%2Fp1dAMq%2BJk7kjrTEuzO%2FMｐnwCIPMRsOneLeqn1hB%2BszABfchCzuV8Sjm8sjYv73GUyW5nbsWA%3D%3D#a3
Mechanism

Schema 3

Air tubes narrow as a result of swollen tissues and excessive mucus production.

Enlarged submucosal gland

Inflammation of epithelium

Mucus accumulation

Hyperinflation of alveoli

Airway lining swells up (inflammation)

Airway muscles tighten

Mucus builds up

Reduced airflow

Damaged airway
Mechanisms Underlying Airflow Limitation in COPD.
Mechanism
Emphysema 1

- Emphysema is a diagnosis defined by permanent enlargement of airspaces distal to the terminal bronchioles.
- This leads to a dramatic decline in the alveolar surface area available for gas exchange.
- Loss of alveoli leads to airflow limitation by 2 mechanisms: 1) loss of the alveolar walls results in a decrease in elastic recoil, which leads to airflow limitation, 2) loss of the alveolar supporting structure leads to airway narrowing, which further limits airflow.
- Emphysema has 3 morphologic patterns: centriacinar, panacinar, distal acinar (paraseptal).
Mechanism
Emphysema 2

- Centriacinar emphysema is characterized by focal destruction limited to the respiratory bronchioles and the central portions of the acini.
- Panacinar emphysema involves the entire alveolus distal to the terminal bronchiole.
- Distal acinar emphysema is the least common form and involves distal airway structures, alveolar ducts, and sacs.
- The gradual destruction of alveolar septae and of the pulmonary capillary bed in emphysema leads to a decreased ability to oxygenate blood.
- The body compensates with lowered cardiac output and hyperventilation.
- Eventually, patients develop muscle wasting and weight loss and are identified as "pink puffers."

http://emedicine.medscape.com/article/297664-overview?pa=%2F0OOpqamlew%2Fp1dAMq%2BJkJrTEuzO2FMsNWClPMRs0neEqn1hB%2BszABfchCzuV8Sw8zjYy73GUyW5nWbWA%3D%3D#a3
Mechanism

Schema 5
Classification
International Classification of Diseases

- X Diseases of the respiratory system
- Chronic lower respiratory diseases (J40-J47)
  - J40 Bronchitis, not specified as acute or chronic
  - J41 Simple and mucopurulent chronic bronchitis
  - J42 Unspecified chronic bronchitis
  - J43 Emphysema
  - J44 Other chronic obstructive pulmonary disease
  - J45 Asthma
  - J46 Status asthmaticus
  - J47 Bronchiectasis

# Classification

GOLD staging of severity for COPD, based on the value of FEV\textsubscript{1}

<table>
<thead>
<tr>
<th>Stage</th>
<th>COPD Description</th>
<th>FEV\textsubscript{1}/FVC \textless 0.70</th>
<th>FEV\textsubscript{1} ≥ 80% normal</th>
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<tbody>
<tr>
<td>Stage I</td>
<td>Mild COPD</td>
<td>FEV\textsubscript{1} \textgtr 0.70</td>
<td>FEV\textsubscript{1} ≥ 80% normal</td>
</tr>
<tr>
<td>Stage II</td>
<td>Moderate COPD</td>
<td>FEV\textsubscript{1} \textgtr 0.70</td>
<td>FEV\textsubscript{1} 50-79% normal</td>
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<tr>
<td>Stage III</td>
<td>Severe COPD</td>
<td>FEV\textsubscript{1} \textgtr 0.70</td>
<td>FEV\textsubscript{1} 30-49% normal</td>
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<tr>
<td>Stage IV</td>
<td>Very Severe COPD</td>
<td>FEV\textsubscript{1} \textgtr 0.70</td>
<td>FEV\textsubscript{1} &lt;30% normal, or &lt;50% normal with chronic respiratory failure present*</td>
</tr>
</tbody>
</table>

* Usually, this means requiring long-term oxygen therapy.
Classification
Clinical GOLD staging of COPD severity

- **Stage 0**: at risk; chronic cough and sputum production; spirometry is normal
- **Stage I**: mild COPD; mild airflow limitation (FEV1/FVC) less than 70% but FEV1 80% or more than predicted; usually, but not always, chronic cough and sputum production
- **Stage II**: moderate COPD; worsening airflow limitation (FEV1 50-79% predicted) and usually progression of symptoms, with shortness of breath, especially on exertion
- **Stage III**: severe COPD; further worsening of airflow limitation (FEV1 30-50% predicted), increased shortness of breath, and repeated exacerbations
- **Stage IV**: very severe COPD; severe airflow limitation (FEV1 less than 30% predicted) or the presence of chronic respiratory failure

http://patient.info/doctor/chronic-obstructive-pulmonary-disease-pro
<table>
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<th>Severity</th>
<th>Classification</th>
</tr>
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<tbody>
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<td>Mild</td>
<td>FEV1/VC &lt;5th percentile of predicted and FEV1 ≥70% pred</td>
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<tr>
<td>Moderate</td>
<td>FEV1/VC &lt;5th percentile of predicted and FEV1 60–69% pred</td>
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<tr>
<td>Moderately severe</td>
<td>FEV1/VC &lt;5th percentile of predicted and FEV1 50–59% pred</td>
</tr>
<tr>
<td>Severe</td>
<td>FEV1/VC &lt;5th percentile of predicted and FEV1 35–49% pred</td>
</tr>
<tr>
<td>Very severe</td>
<td>FEV1/VC &lt;5th percentile of predicted and FEV1 &lt;35% pred</td>
</tr>
</tbody>
</table>

FEV1: forced expiratory volume in 1 second; VC: vital capacity; % pred: % of predicted value; FVC: forced vital capacity.
Symptoms and Signs

- The most important symptoms of COPD are breathlessness on exertion and chronic cough with or without phlegm.
- The dyspnea usually worsens over time but is often not present in mild or moderate COPD.
- The cough may be dry or productive.
- Cough and phlegm often precede dyspnea on exertion by many years.
- Other symptoms include wheezing and chest tightness.
- As the disease progresses and reaches the severe stages, fatigue, weight loss and anorexia may increase.

http://www.erswhitebook.org/chapters/chronic-obstructive-pulmonary-disease/
Clinical Manifestations 1

- A characteristic of COPD is exacerbations or episodes of acute worsening of the respiratory symptoms.
- The most common causes of exacerbations are viral or bacterial infections.
- Increased air pollution also appears to precipitate exacerbations of COPD.
- Some patients are particularly prone to exacerbations while others are not.
- Two or more exacerbations during the previous year is the most important indicator of a future exacerbation.
Clinical Manifestations 2

• Exacerbations accelerate the decline in lung function that characterises COPD, resulting in reduced physical activity, poorer quality of life, and an increased risk of death; they are also responsible for a large proportion of the healthcare costs attributable to COPD

• Patients with COPD often suffer from other diseases (comorbidities)

• The comorbidities may share common risk factors with COPD, in particular cigarette smoking, and may also represent extrapulmonary manifestations or complications of COPD, such as muscle dysfunction due to inactivity

• Comorbidities may be secondary to treatment of COPD; for example, osteoporosis due to oral corticosteroid treatment

http://www.erswhitebook.org/chapters/chronic-obstructive-pulmonary-disease/
Clinical Manifestations 3

- The most common comorbidities in COPD are ischemic heart disease, anxiety and depression, osteoporosis, skeletal muscle dysfunction, gastro-esophageal reflux, anaemia, lung cancer, diabetes and metabolic syndrome.
- Comorbidities contribute to the overall severity and manifestations of the disease and can occur in mild, moderate or severe COPD.
- The clinical effects of COPD show considerable inter-individual variation, depending on which respiratory symptoms predominate, the frequency of exacerbations, the level and rate of lung function decline and the amount of emphysema, as well as comorbidities.
- Various subtypes of the disease are often termed phenotypes of COPD.
Complications

- Pneumonia
- COPD heart failure (Cor pulmonale)
- Lung cancer
- Diabetes
- Dementia
- Arrhythmias
- Osteoporosis
- Respiratory failure.
Diagnosis

• The diagnosis of COPD should be considered in anyone over the age of 35 to 40 who has shortness of breath, a chronic cough, sputum production, or frequent winter colds and a history of exposure to risk factors for the disease.

• Spirometry is then used to confirm the diagnosis.

• The formal diagnosis of COPD is made, when the ratio of forced expiratory volume in 1 second over forced vital capacity (FEV₁/FVC) is less than 70% of that predicted for a matched control, it is diagnostic for a significant obstructive defect.

• Screening those without symptoms is not recommended.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Signs_and_symptoms
Diagnosis

Spirometry 1

PEFR = peak expiratory flow rate; TLC = total lung capacity.

Diagnosis
Spirometry 2

![Spirometry Diagram](http://image.slidesharecdn.com/copyoflsqasthmamanagementptv050811piccomp-141113083910-conversion-gate02/95/spirometry-basics-2-30-638.jpg?cb=1415868230)
Diagnosis
Spirometry 3
Diagnosis
Arterial Blood Gas (ABG) Findings

• ABGs provide the best clues as to acuteness and severity of disease exacerbation
• Patients with mild COPD have mild to moderate hypoxemia without hypercapnia
• As the disease progresses, hypoxemia worsens and hypercapnia may develop, with the latter commonly being observed as the FEV₁ falls below 1 L/s or 30% of the predicted value
• pH usually is near normal; a pH below 7.3 generally indicates acute respiratory compromise
• Chronic respiratory acidosis leads to compensatory metabolic alkalosis.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWskqAfEbVp94c%2FW11%2B%2Fk7GZhKH%2B3snXYEMg6J2iYxQ%2FuxvzKUqypCxZhboX8IvI8zjW73GUyW5rsbWA%3D%3D#a1
Diagnosis
Frontal And Lateral Chest Radiographs

- Flattening of the diaphragm
- Increased retrosternal air space
- A long, narrow heart shadow
- Rapidly tapering vascular shadows accompanied by hyperlucency of the lungs
- Radiographs in patients with chronic bronchitis show increased bronchovascular markings and cardiomegaly.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWSkqAfubVp94c%2FW11%2B%2Fk7GZKKn%2B3nXYEMg6JZJtQ%2FxuvvKUqypCzzb0X8l8l8zjW+7GUyW5r5bWA%3D%3D#a1
Diagnosis

Imaging Studies  2
Diagnosis
High-Resolution Computed Tomography

- Greater sensitivity than standard chest radiography
- High specificity for diagnosing emphysema (outlined bullae are not always visible on a radiograph)
- May provide an adjunctive means of diagnosing various forms of COPD (e.g., lower lobe disease may suggest alpha1-antitrypsin (AAT) deficiency
- May help the clinician determine whether surgical intervention would benefit the patient.
Diagnosis

Imaging Studies  3
Diagnosis

Imaging Studies  4
Diagnosis

Other Tests

• Hematocrit (patients with polycythemia)
• Serum potassium (diuretics, beta-adrenergic agonists, and theophylline act to lower potassium levels)
• Measure AAT in patients < 40 years or with a family history of emphysema at an early age
• Sputum evaluation (a transformation from mucoid in stable chronic bronchitis to purulent in acute exacerbations)
• Pulse oximetry provides instant feedback on a patient's status
• Electrocardiography can establish that hypoxia is not cardiac
• The distance walked in 6 minutes is a predictor of mortality in patients with moderate COPD
• Right-sided heart catheterization can confirm pulmonary artery hypertension.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWSkgAfueBv94c%2FW11%2B%2Fk7GZhKH%2B3nXYEMg6J2YlxQ%2FuxxzKUqypCxb0X8Iv8zjV73GUyW5rsbWA%3D%3D#a1
Diagnosis

Differentiation

- Asthma: diagnosed by establishing reversibility or variability of airflow obstruction either by spirometry or peak flow measurements after treatment with a bronchodilator or steroid.
- Other diagnoses to consider are congestive heart failure, bronchiectasis, allergic fibrosing alveolitis, pneumoconiosis, asbestosis or other restrictive lung conditions, tuberculosis, lung cancer, obliterative bronchiolitis, bronchopulmonary dysplasia, anaemia or generally poor physical condition.
Management

• Assessment and monitoring of disease, reduction of risk factors, management of stable COPD, management of exacerbations

• The goals of management are to relieve symptoms, prevent disease progression, improve exercise tolerance, improve health status, prevent and treat complications and exacerbations, reduce mortality and prevent or minimize side-effects from treatment

• Important components of management are smoking cessation, medical treatment with bronchodilators as well as inhibitors of inflammation, physical exercise and, in advanced disease, oxygen therapy, influenza vaccination once a year, pneumococcal vaccination once every 5 years

• Pulmonary rehabilitation is important, the most effective component of pulmonary rehabilitation is physical exercise.

http://www.erswhitebook.org/chapters/chronic-obstructive-pulmonary-disease/
Management
Approaches to Management by COPD Stage

• Stage I (mild obstruction): short-acting bronchodilator as needed
• Stage II (moderate obstruction): short-acting bronchodilator as needed; long-acting bronchodilator(s); cardiopulmonary rehabilitation
• Stage III (severe obstruction): short-acting bronchodilator as needed; long-acting bronchodilator(s); cardiopulmonary rehabilitation; inhaled glucocorticoids if repeated exacerbations
• Stage IV (very severe obstruction or moderate obstruction with evidence of chronic respiratory failure): short-acting bronchodilator as needed; long-acting bronchodilator(s); cardiopulmonary rehabilitation; inhaled glucocorticoids if repeated exacerbation; long-term oxygen therapy (if criteria met); consider surgical options such as lung volume reduction surgery (LVRS) and lung transplantation.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWSkqAfueBv94c%2FW11%2BfK7GzhKH%2B3snXYEMg6JZyixQ%2FuxvzKUqypCZhb0X8SlI8zjV73GUyW5rsbWA%3D%3D#a1
Management
Medications

- Short-acting beta_2-agonist bronchodilators (e.g., albuterol, metaproterenol, levalbuterol, pirbuterol)
- Long-acting beta_2-agonist bronchodilators (e.g., salmeterol, formoterol, arformoterol, indacaterol, vilanterol)
- Respiratory anticholinergics (e.g., ipratropium, tiotropium, aclidinium)
- Xanthine derivatives (i.e., theophylline)
- Phosphodiesterase-4 Inhibitors (i.e., roflumilast)
- Inhaled corticosteroids (e.g., fluticasone, budesonide)
- Oral corticosteroids (e.g., prednisone)
- Beta_2-agonist and anticholinergic combinations (e.g., ipratropium and albuterol,umeclidinium bromide/vilanterol inhaled)
- Beta_2-agonist and corticosteroid combinations (e.g., budesonide/formoterol, fluticasone and salmeterol).
Management
Bronchodilators 1

• Inhaled bronchodilators are the primary medications used and result in a small overall benefit
• There are two major types, $\beta_2$-agonists and anticholinergics; both exist in long-acting and short-acting forms
• Short-acting agents are recommended on an as needed basis
• In those with more severe disease, long-acting agents are recommended
• If long-acting bronchodilators are insufficient, then inhaled corticosteroids are typically added
• With respect to long-acting agents, it is unclear if tiotropium (a long-acting anticholinergic) or long-acting beta agonists (LABAs) are better, and it may be worth trying each and continuing the one that worked best

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Bronchodilators 2

• There are several short-acting β₂ agonists available including salbutamol and terbutaline; they provide some relief of symptoms for four to six hours.

• Long-acting β₂ agonists such as salmeterol and formoterol are often used as maintenance therapy; when used with inhaled steroids they increase the risk of pneumonia.

• There are two main anticholinergics used in COPD, ipratropium and tiotropium; ipratropium is a short-acting agent while tiotropium is long-acting.

• Anticholinergics can cause dry mouth and urinary tract symptoms ND are also associated with increased risk of heart disease and stroke.

• Aclidinium, another long acting agent which came to market in 2012, has been used as an alternative to tiotropium.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Corticosteroids

- Corticosteroids are usually used in inhaled form but may also be used as tablets to treat and prevent acute exacerbations.
- While inhaled corticosteroids (ICS) have not shown benefit for people with mild COPD, they decrease acute exacerbations in those with either moderate or severe disease.
- By themselves ICS have no effect on overall one-year mortality.
- When used in combination with a LABA they may decrease mortality compared to either ICS or LABA alone.
- Inhaled steroids are associated with increased rates of pneumonia.
- Long-term treatment with steroid tablets is associated with significant side effects.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Other medication

• Long-term antibiotics, specifically those from the macrolide class such as erythromycin, reduce the frequency of exacerbations in those who have two or more a year
• This practice may be cost effective in some areas of the world
• Concerns include that of antibiotic resistance and hearing problems with azithromycin
• Methylxanthines such as theophylline generally cause more harm than benefit and thus are usually not recommended, but may be used as a second-line agent in those not controlled by other measures
• Mucolytics may help to reduce exacerbations in some people with chronic bronchitis
• Cough medicines are not recommended.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Oxygen

• Supplemental oxygen is recommended in those with low oxygen levels at rest (a partial pressure of oxygen of less than 50–55 mmHg or oxygen saturations of less than 88%)
• In those with normal or mildly low oxygen levels, oxygen supplementation may improve shortness of breath
• There is a risk of fires and little benefit when those on oxygen continue to smoke
• During acute exacerbations, many require oxygen therapy; the use of high concentrations of oxygen without taking into account a person's oxygen saturations may lead to increased levels of carbon dioxide and worsened outcomes
• In those at high risk of high carbon dioxide levels, oxygen saturations of 88–92% are recommended, while for those without this risk recommended levels are 94–98%.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Exacerbations 1

• Acute exacerbations are typically treated by increasing the use of short-acting bronchodilators; this commonly includes a combination of a short-acting inhaled beta agonist and anticholinergic.

• These medications can be given either via a metered-dose inhaler with a spacer or via a nebulizer with both appearing to be equally effective.

• Nebulization may be easier for those who are more unwell.

• Oral corticosteroids improve the chance of recovery and decrease the duration of symptoms; 5 days of steroids work as well as 10 - 14.

• In those with a severe exacerbation, antibiotics improve outcomes (amoxicillin, doxycycline and azithromycin).

• The FDA recommends against the use of fluoroquinolones when other options are available due to higher risks of serious side effects.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Exacerbations 2

• There is no clear evidence for those with less severe cases
• For those with respiratory failure with acutely raised CO₂ levels non-invasive positive pressure ventilation decreases the probability of death or the need of intensive care admission
• Additionally, theophylline may have a role in those who do not respond to other measures
• Fewer than 20% of exacerbations require hospital admission
• In those without acidosis from respiratory failure, home care ("hospital at home") may be able to help avoid some admissions.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Nebulizers and Spacers

Nebulizer

Spacer

http://apqualitycare.com/product/nebulizers-spacers/
Management

Surgery

- For those with very severe disease, surgery is sometimes helpful and may include lung transplantation or lung volume reduction surgery.
- Lung volume reduction surgery involves removing the parts of the lung most damaged by emphysema allowing the remaining, relatively good lung to expand and work better.
- Volume reduction surgery seems to be particularly effective if emphysema predominantly involves the upper lobe, but the procedure increases the risks of early death and adverse events.
- Lung transplantation is sometimes performed for very severe COPD, particularly in younger individuals.

https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease#Management
Management
Pulmonary Rehabilitation

- Patient and family education
- Smoking cessation
- Medical management (including oxygen and immunization)
- Respiratory and chest physiotherapy
- Physical therapy with bronchopulmonary hygiene, exercise, and vocational rehabilitation
- Psychosocial support.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWSkqAfuEbVp94c%2FW11%2B%2FK7GZhKH%2B3snXYEMg6JZyI%2FuxvzKUqyV0bX8iV8zjYv73GUyW5rsbWA%3D%3D#a1
Management
Patient Education

• It is important to educate the patient with COPD about the disease and to encourage his or her active participation in therapy
• The 2 most essential points for the patient to understand are as follows:
  • The dangers of smoking and the improvement in quality of life attainable with smoking cessation
  • The need to seek medical care early during an exacerbation and to not wait until they are in distress.

http://emedicine.medscape.com/article/297664-overview?pa=P4tWSkqAfueBv94c%2FW11%2BFk7GZhKH%2B3sXYEMg6JZyIY%2F%2FuxvzKUqypCzhb0X8%ii%8zjyV73GUyW5srbWA%3D%3DhA1
Prognosis

- COPD is a chronic, progressive disease showing great variation in its natural history.
- Spirometry providing data on FEV$_1$ and FVC is the most common measure of disease progression.
- A large cohort of patients with COPD of GOLD stage II+ followed up every 6 months for 3 years showed a mean annual decline in FEV$_1$ of 33 mL.
- An annual decline in FEV$_1$ >40 mL was seen in 38% of the patients, while 8% showed an average annual increase of 20 mL.
- Current smoking and emphysema are related to more rapid decline in FEV$_1$.
- Factors that indicate a poor prognosis are frequent exacerbations, respiratory insufficiency, nutritional status and comorbidities.
Prophylaxis

- Identification and reduction of exposure to risk factors are important steps in the prevention and treatment of COPD.
- All individuals who smoke should be encouraged to quit regardless of their disease status.
- Smokers without COPD should be offered smoking-cessation advice.
- Preventing passive smoking in fetal and early life is important to reduce the risk of COPD in adult life.
- Reduction of exposure to smoke from indoor biomass combustion, particularly among women and children, is important to reduce the prevalence of COPD.
- Prevention of COPD exacerbations is important: influenza and pneumococcal vaccination as well as treatment with inhaled long-acting bronchodilators and inhaled corticosteroids all work to reduce exacerbations and hospitalisations for COPD.

http://www.erswhitebook.org/chapters/chronic-obstructive-pulmonary-disease/
Abbreviations

AAT - protein $\alpha_1$-antitrypsin
ABG - Arterial Blood Gas
COPD - Chronic Obstructive Pulmonary Disease
FEV$_1$ - forced expiratory volume in 1 second
ICS - inhaled corticosteroids
LABAs - long-acting beta agonists
Diagnostic and treatment guidelines

Diagnosis and Management of Stable Chronic Obstructive Pulmonary Disease: A Clinical Practice Guideline Update from the American College of Physicians, American College of Chest Physicians, American Thoracic Society, and European Respiratory Society

Treatment of Stable Chronic Obstructive Pulmonary Disease: the GOLD Guidelines

Chronic obstructive pulmonary disease: Management of chronic obstructive pulmonary disease in adults in primary and secondary care

Chronic obstructive pulmonary disease in over 16s: diagnosis and management

Chronic-Obstructive-Pulmonary-Disease