



**PULMONARY DYSPNEA
BRONCHIAL DISEASES**

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Bronchial pathology

Rules useful in management of bronchial diseases:

- 1) Adjusting on ambulatory conditions;
- 2) Establishing evolution and severity;
- 3) Primary prevention (i.e. - no smoke);
- 4) Spirometrical monitoring;
- 5) Chronic utilization of drugs, on ambulatory care:
 - Antibiotherapy on chronic bronchitis acutization;
 - Specific drugs for broncho-spasm;
- 6) Home therapy, if necessary;
- 7) Quality of life (QualityLife).

Acute Bronchitis (AB)

Definition = acute inflammatory reaction on bronchial mucosa manifested by:

- pain (chest pain);
- cough;
- mucus, mucus–purulent sputum.

Etiology:

- Infections with: Strept. Pneumoniae, Haemophilus influenzae
- Chemical agents;
- **Allergy:** predisposing agents are – pollutions, smoke that means low efficiency of mucous-cilia system, low immunity.

Morphopathology: AB consists of inflammation on tracheal and bronchial mucosa with denudation of basal cells.

Acute Bronchitis - Clinical exam

First phase:

- Fever, subfebrility (in cold seasons);
- Myalgias, headaches;
- Chilly, influenced general state;
- Rhinorrhea;
- Irritative cough;
- Dysphonia, dyspnea.

Second phase: 4 – 5 days

- Mucous – purulent sputum;
- Better general state.

Third phase (remission): 10 days

- persisting coughing but no sputum.
- Examination → rare bronchial rales
- Thoracic X rays – normal
- Laboratory – leucopenia (viral infection).

Acute Bronchitis

Evolution: – complete remission;
– spastic cough – bronchial asthma
– persisted expectoration (on predisposing)

Clinical forms

On children: - larynx involvement with glottic edema
- acute bronchiolitis – sever evolution

On elderly – sever evolution due to low immunity

Differential diagnosis is made with all the forms of TB

Complications in evolution – sinusitis, otitis

Treatment: - Preventive attitude

- Vaccination for persons with predisposing factors
- Dietary/no cold
- Drugs: - Symptomatically – antitussives, expectorants
 - Anti-inflammatory drugs/antibiotics
 - Antipyretic drugs,

Chronic bronchitis

Clinic – chronic bronchitis syndrome with progressive dyspnea onset over 40 years.

Pathology obstruction of intrapulmonary airways

Anatomy – obstructive lesions (Reid Index) $> 0,52$

– emphysema (central lobule)

– thickness of bronchial wall – irreversible

Etiology – excluded – asthma, emphysema, cystic fibrosis, nocturnal apnea – hypopnea syndrom.

Dyspnea – stages:

I – large efforts, FEV1 close normal;

II – current efforts, FEV1, Life expectancy 10 years;

III – minimal efforts, FEV1 < 1 , expectancy 4 years.

IV – no exercises, no effort FEV1 $< 0,5$, expectancy 1-2 years.

Chronic bronchitis

Classification

- 1) Simple – only sputum (mucous).
- 2) Mucous – purulent bronchitis – purulent sputum chronically without signs of abscess.
- 3) Obstructive chronic bronchitis – on Spirometry
- 4) Spastic chronic bronchitis – episodic bronchial spasm - wheezing.

Differential diagnosis with asthma – hystorical wheeze and chronic bronchitis – hystorical cough with sputum

- 5) COPD – chronic obstructive pulmonary diseases with central acinary emphysema (monitoring of FEV1, PEFr).

Chronic bronchitis

Etiology – germs:

Haemophilus influenzae

Streptococcus pneumoniae

Brahmanella catharalis

Rhinoviruses

Mycoplasma pneumoniae

Risk factors:

smoking

pollutions

infections

genetically and family (deficiency of alfa1 chemotripsine)

Evolution to **COR PULMONALE CHRONICUM** and to
RESPIRATORY FAILURE.

Bronchial asthma

Definition: chronic inflammations of bronchial mucosa with, different triggers manifested on predisposed persons with bronchial hyper reactivity by: expiratory dyspnea in crisis and wheezing with remission (total remission) at bronchodilators or spontaneous.

Etiology – determined by:

Atopic

Bronchial hyperreactivity

- A) Allergens
- B) Medication
- C) Pollutions, atmospheric agents, professional factors
- D) Infection (viruses)
- E) Effort
- F) Psychological factors
- G) Gastro–esophageal factors
- H) Focal infections

Bronchial asthma - diagnosis

■ Clinical

Anamnesis wheezing in crises condition of appearance –
bronchial asthma with intermittent access

Can be

- „chronic“ asthma
- Status asthmaticus (cardiac involvement):
 1. – silent lung
 2. – important dyspnea
 3. – tachypnea $> 25/\text{min}$ with long expire using accessory muscles
 4. – tachycardia $> 110/\text{min}$
 5. – paradoxical pulses
 6. – PEF $< 110/\text{min}$

Bronchial asthma - diagnosis

■ Laboratory - *For allergic status*

1. *Eosinophilia: in blood and sputum*

Total and specific IgE, another Ig (A, G, M).

2. *Skin tests*

IDR, scarification, prick – test (1/10 – in tract)

read to 15 min, 72 h

3. *Spyrometry* with provocation – specific

– Non specific (Prednisolone)

– Low flux expiratory flow (with large daily variability)

– High pulmonary volumes

– Normal capacity of diffusions, diseases like asthma:

1) Aspirin induced asthma (HLA – DQW2)

2) Churg–Strauss syndrome (et, IgE > 250 UI)

3) Aspergillosis.

Bronchial asthma - treatment

We must:

- 1) Control acute manifestations;
- 2) Prevent exacerbations;
- 3) Maintain normal pulmonary function and professional activity.

Methods of treatment

- education
- trigger control
- drugs
- immunotherapy.

Medications:

- 1) Bronchodilators
- 2) Anti-inflammatory drugs
 - Corticosteroids
 - Cromoglycate disodate
 - Nedocromyl
 - Antileukotrienes
- 3) Others:
 - Ketotifen, Methothrexat
 - Hydroxychloroquine
 - Specific and nonspecific imunomodulation

Bronchial asthma – treatment in crisis

A light form:

- Selective beta adrenergic drugs SABA–
Salbutamole, Terbutaline
p.o.
- Cromolyne, Ketotifen.

A medium form:

- Salbutamole (Ventoline)
aerosols, s.c., i.v.
- Theophyline 200 – 300 mg
i.v., slow (10 – 20 mg/ml)
- + Corticoids aerosols, i.v.
- + Anticholinergic – Ipratropium –
aerosols
- + Sedative (no morphine)
- + O₂ and rehydration
- severe forms – hospitalization or
intense monitoring

Bronchial asthma – treatment

■ At home

- Clinical assessment (PEFR < 150 l/min)
- Theofilin 240 – 500 mg i.v. slowly
- Hydrocortisone hemysuccinate - 200 mg/dose
- O₂

■ At hospital

1. Assessment

2. O₂

3. PEFR < 150 ml/min

4. Salbutamol – 5 microgr (and/or) every 4 h+ aerosols

5. Hydrocortisone HHC 200 mg every 4 h

After 24 h PDN 30 mg (2 weeks)

6. Oxymetry → ventilation

7. X ray – excludes a pneumonia.

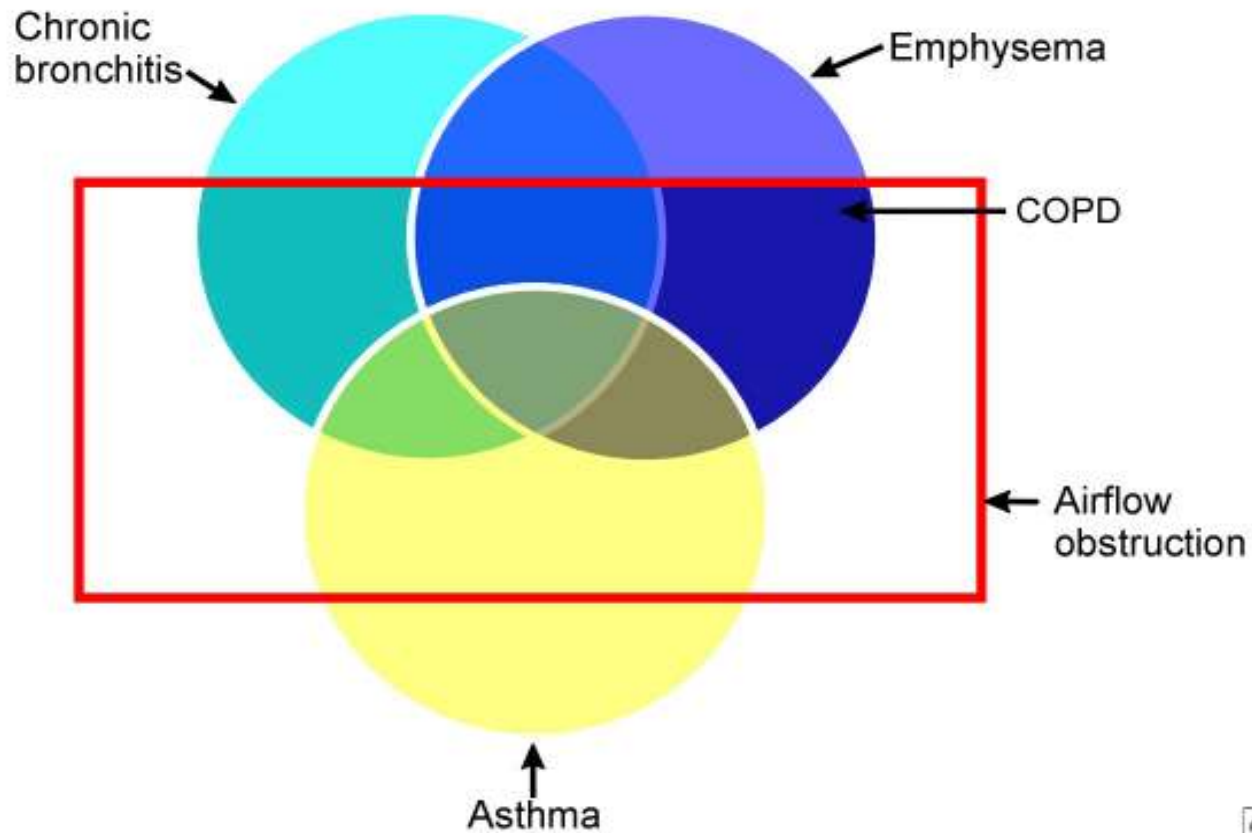
Chronic Obstructive Pulmonary Disease

Disorder that causes a huge degree of human suffering.

Definition:

- 1. Chronic bronchitis is defined clinically as the presence of a chronic productive cough for 3 months during each of 2 consecutive years.
- 2. Emphysema is defined pathologically as an abnormal, permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and.
- 3. The Global Initiative for Chronic Obstructive Lung Disease (*GOLD*) guidelines define COPD as a disease state characterized by airflow limitation that is not fully reversible, is usually progressive, and is associated with an abnormal inflammatory response of the lungs to inhaled noxious particles or gases.

Venn diagram of chronic obstructive pulmonary disease (COPD)



Clinical

History: have smoked at least 20 cigarettes per day for 20 or more years before the onset of the common symptoms of cough, sputum, and dyspnea. Presentation commonly occurs in the fifth decade of life.

- **A productive cough** or an acute chest illness is common. The cough usually is worse in the mornings and produces a small amount of colorless sputum.

- **Breathlessness** is the most significant symptom, but it usually does not occur until the sixth decade of life (although it may occur much earlier). By the time the FEV1 has fallen to 30% of predicted, the patient is usually breathless after minimal exertion.

- **Wheezing** may occur in some patients, particularly during exertion and exacerbations.

- Many patients with COPD may have decreased fat-free mass, impaired systemic muscle function, osteoporosis, anemia, depression, pulmonary hypertension, cor pulmonale, and even left-sided heart failure

Physical Exam

The respiratory rate increases proportionally to disease severity. Use of accessory respiratory muscles and paradoxical indrawing of lower intercostal spaces is evident (known as the Hoover sign).

In advanced disease, cyanosis, elevated jugular venous pulse (JVP), and peripheral edema are observed.

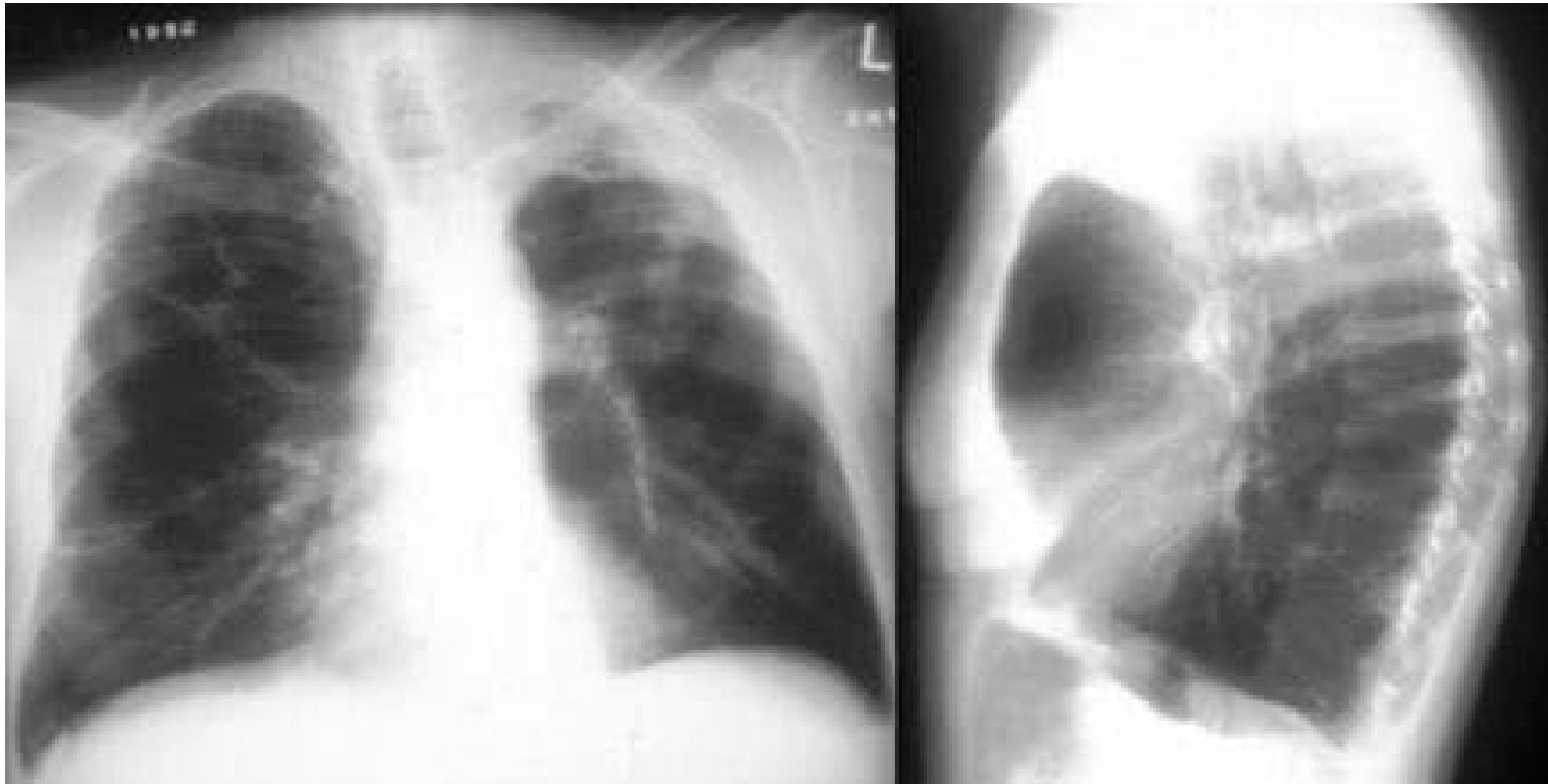
Measurement of forced expiratory time maneuver is a simple bedside test; a forced expiratory time of more than 6 seconds indicates considerable expiratory flow obstruction. Bedside spirometry, which can actually help quantify the severity of obstruction by virtue of the FEV1 .

Thoracic examination reveals hyperinflation (barrel chest), wheezing, diffusely decreased breath sounds, hyperresonance on percussion, and prolonged expiration. Coarse crackles beginning with inspiration may be heard, and wheezes frequently are heard on forced and unforced expiration.

Laboratory

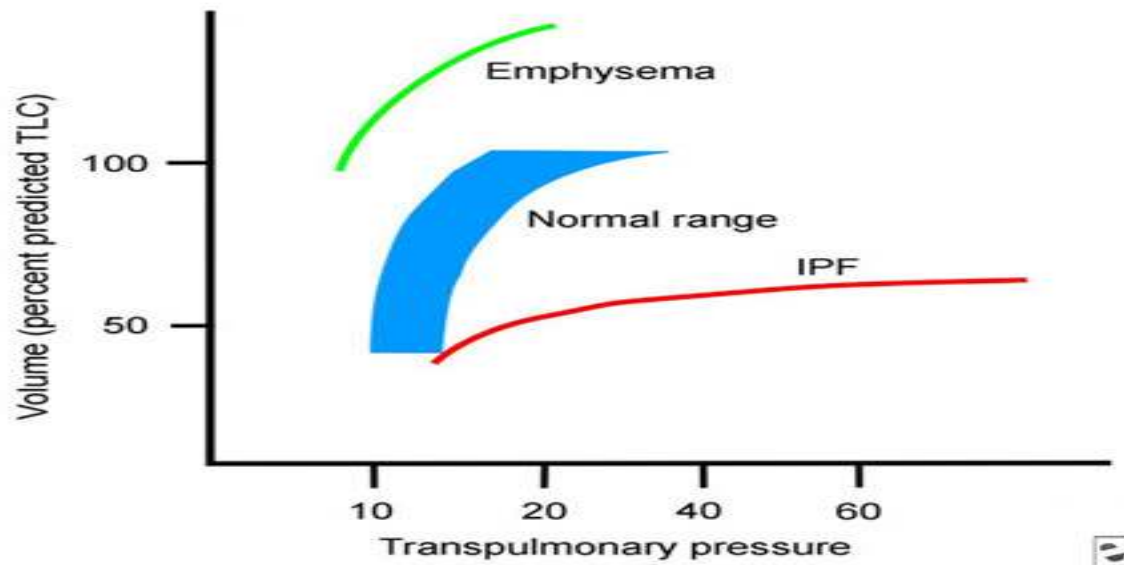
- Polycythemia - chronic hypoxemia;
- Hematocrit - > 52% in males, > 47% in female;
- Sputum: - stable chronic bronchitis: mucoid and macrophages
 - an exacerbation : - purulent and neutrophils
 - Strept. pneumoniae, H. influenzae,
Moraxella catarrhalis, Pseud. aeruginosa
- Imaging studies - Chest radiography
 - Pulmonary CT
 - Two-dimensional echocardiography

Posteroanterior (PA) and lateral chest radiograph in a patient with severe chronic obstructive pulmonary disease (COPD). Hyperinflation, depressed diaphragms, increased retrosternal space, and hypovascularity of lung parenchyma is demonstrated.

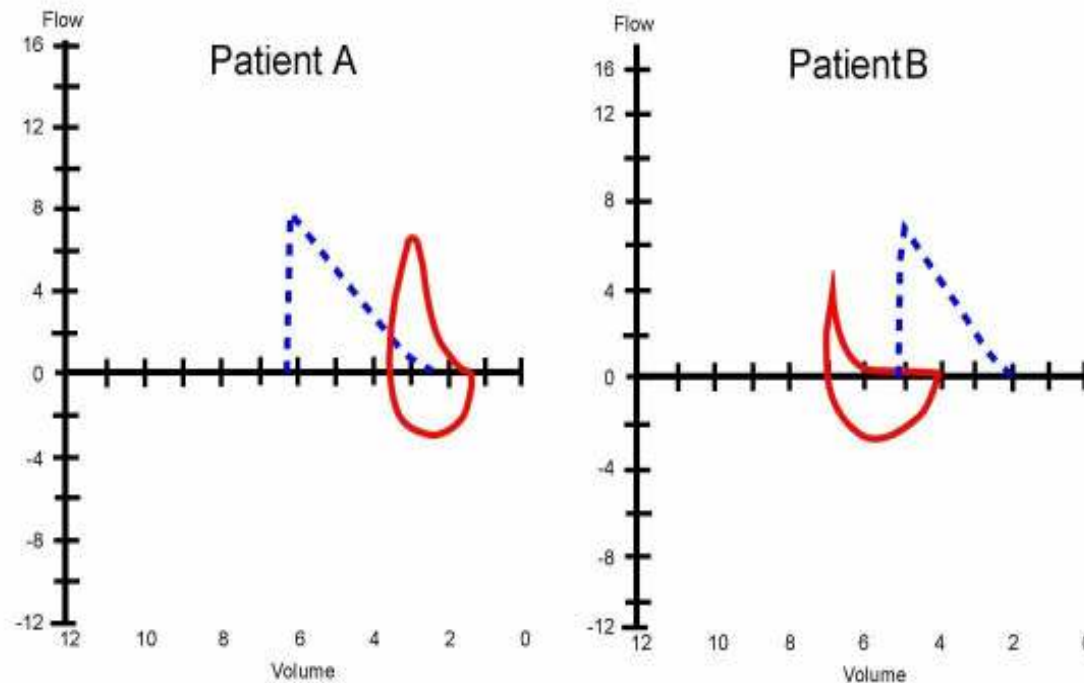


[Pulmonary function tests]

- For the diagnosis and assessment of the severity of disease, and they are helpful in following its progress.
- Forced expiratory volume in 1 second (FEV1) is a reproducible test and is the most commonly used index of airflow obstruction.
- Lung volume measurements often show an increase in total lung capacity, functional residual capacity, and residual volume. The vital capacity often decreases.
- Carbon monoxide diffusing capacity is decreased in proportion to the severity of emphysema.
- As many as 30% of patients have an increase in FEV1 by 15% or more after inhalation of a bronchodilator. However, the absence of bronchodilator response does not justify withholding therapy.



Pressure volume curve comparing lungs with emphysema lungs and restrictive lungs to normal lungs.



Flow volume curve of lungs in emphysema shows marked decrease in expiratory flows, hyperinflation, and air trapping (patient B) compared to a patient with restrictive lung disease, who has reduced lung volumes and preserved flows (patient A).

Staging - ATS criteria

- **Stage I (mild)** - FEV1 greater than or equal to 80% of predicted.
- **Stage II (moderate)** - FEV1 less than 80% and greater than or equal to 50% of predicted
- **Stage III (severe)** - FEV1 less than 50% and greater than or equal to 30% of predicted
- **Stage IV (very severe)** - FEV1 less than 30% of predicted or FEV1 less than 50% and chronic respiratory failure

GOLD guidelines

Stage I (mild obstruction) - Reduction of risk factors (influenza vaccine) plus short-acting bronchodilator as needed.

Stage II (moderate obstruction) - Stage I plus short-acting bronchodilator as needed plus long-acting bronchodilator(s) plus cardiopulmonary rehabilitation.

Stage III (severe obstruction) – Stage II plus cardiopulmonary rehabilitation plus inhaled glucocorticoids if repeated exacerbations.

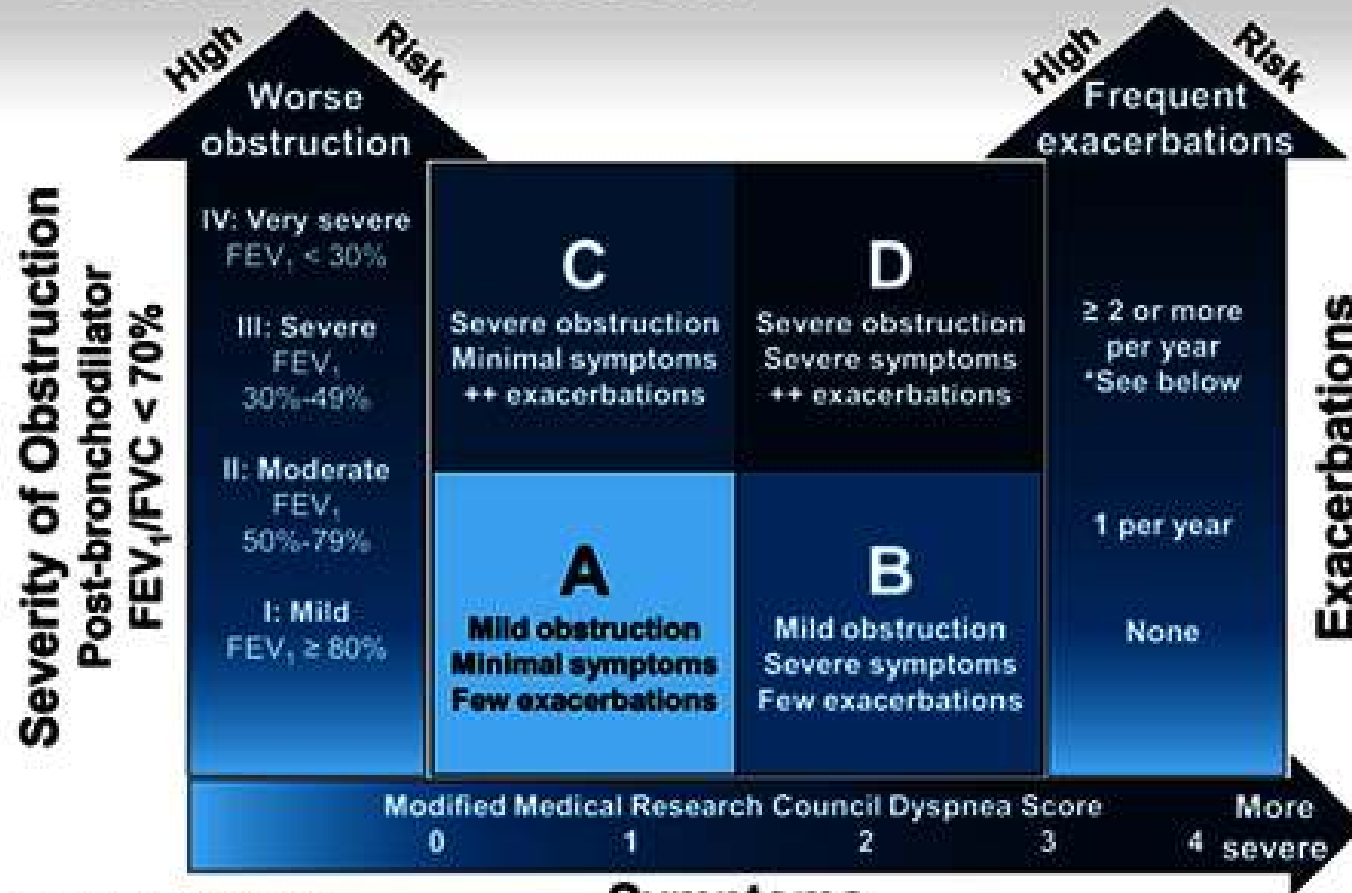
Stage IV (very severe obstruction or moderate obstruction with evidence of chronic respiratory failure) – Stage III plus long-term oxygen therapy (if criteria met); also consider surgical options.

Treatment

The goal of chronic obstructive pulmonary disease (COPD) - improve daily living and the quality of life by preventing symptoms and the recurrence of exacerbations by preserving optimal lung function.

Smoking cessation continues to be the most important therapeutic intervention. The use of the antidepressant bupropion (Zyban) is also effective for smoking cessation. The most recent drug to receive approval for smoking cessation is varenicline (Chantix).

COPD Assessment

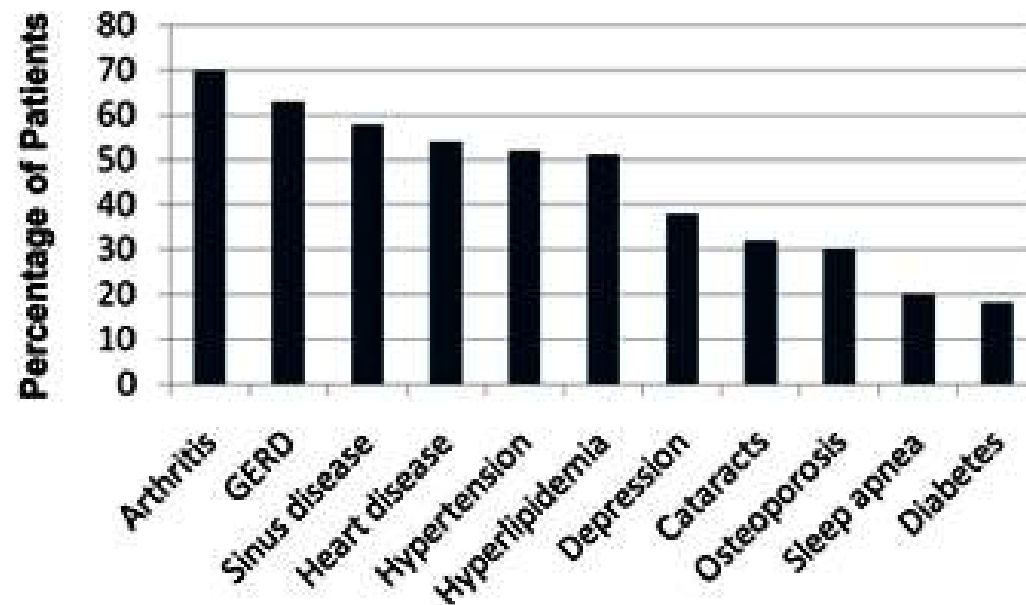


++ = frequent exacerbation

Slide provided by Sandra G. Adams, MD, MS and WipeCOPD.

Altered by Medscape Education and National Jewish Health from the *Global Strategy for Diagnosis, Management and Prevention of COPD 2011*, Global Initiative for Chronic Obstructive Lung Disease (GOLD), <http://www.goldcopd.org>.

COPD and Comorbidities



GERD = gastroesophageal reflux disease
Barr RG, et al. *Am J Med.* 2009;122:348-355.

Treatment

- **Bronchodilators**: the use of bronchodilators is guided by some very important concepts. In some patients, the change in forced expiratory volume in 1 second (FEV1) may be small; however, benefit may be seen by some other mechanism, such as decreased hyperinflation (hence, lack of a bronchodilator response on pulmonary function testing should not preclude their use if clinically warranted). Furthermore, some patients may have difficulty achieving effective delivery of the medication using a metered-dose inhaler; hence, use of a spacer may be of benefit to the patient. Inhaled delivery of medications is preferred over the oral route to help minimize potential adverse effects.

Treatment

- **Beta-agonists:** Inhaled beta2-agonist bronchodilators activate specific B2-adrenergic receptors on the surface of smooth muscle cells, which increases intracellular cyclic adenosine monophosphate (AMP) and smooth muscle relaxation. Patients, even those who have no measurable increase in expiratory flow, may benefit from treatment using beta2 agonists.

- mild intermittent symptoms - short-acting beta2 agonist
- persistent symptoms - long-acting beta agonist

Long-acting beta agonists have been shown to increase exercise endurance, prevent nocturnal dyspnea, and improve quality of life.

Treatment

- **Anticholinergic:** anticholinergic drugs compete with acetylcholine for postganglionic muscarinic receptors, thereby inhibiting cholinergically mediated bronchomotor tone, resulting in bronchodilatation. They block vagally mediated reflex arcs that cause bronchoconstriction. The clinical benefit is gained through a decrease in exercise-induced dynamic hyperinflation.

Ipratropium bromide is administered 2-4 puffs every 6-8 hours.

Tiotropium is a once-daily, long-acting anticholinergic medication that has been shown to have significant clinical benefit and is a first-line therapy in patients with persistent symptoms.

Treatment

- **Steroids:** corticosteroids are potent anti-inflammatory medications that affect the inflammatory cascade at multiple points. In the oral form, their primary role is for the treatment of exacerbations. Note that oral steroids are not as effective in treating COPD exacerbations as they are for bronchial asthma exacerbations.

The goal, however, is to wean from the steroid as soon as the patient can clinically tolerate it because of the concern for potential well-known systemic adverse effects. However, a small portion of patients may require long-term corticosteroid use to keep their symptoms under control. Inhaled corticosteroids provide a more direct route of administration to the airways.

Consequently, aside from the development of thrush, the systemic adverse effects of these medications at standard doses are negligible.

Treatment

- **Nonsteroidal anti-inflammatory medications:** macrolide antibiotics have been shown to have anti-inflammatory effects in the airways of COPD patients. More specifically, *azithromycin* has been shown to improve phagocytic function of pulmonary macrophages and be a potent anti-inflammatory. Azithromycin is clinically used for its anti-inflammatory effects in patients with cystic fibrosis and in lung transplantation patients with chronic rejection.
- **Phosphodiesterase:** Methylxanthines (ie, theophylline) are nonspecific phosphodiesterase inhibitors that increase cyclic AMP within the airway smooth muscle of the airways. Additionally, they may improve diaphragm muscle contractility and stimulate the respiratory center.

Treatment

- **Antibiotics:** empiric antimicrobial therapy is recommended in patients with an acute exacerbation (as evidenced by an increase in baseline dyspnea and/or a change in the quantity or quality of cough) and evidence of an infectious process, such as fever, leukocytosis, or an infiltrate on chest radiograph. The antibiotic choice must be comprehensive and should cover all likely pathogens in the context of the clinical setting and local resistance patterns.
- **Oxygen therapy:** COPD commonly is associated with progressive hypoxemia; oxygen administration reduces mortality rates in patients with advanced COPD because of the favorable effects on pulmonary hemodynamics.

Reducing the Risk for COPD Exacerbations

- Vaccination
 - Influenza
 - Pertussis
 - Pneumococcal
- Long-acting beta-agonist/inhaled corticosteroid combination therapy
- Long-acting anticholinergic agents
- Phosphodiesterase-4 inhibitors
- Antibiotics

Global Initiative for Chronic Obstructive Lung Diseases (GOLD). Global strategy for diagnosis, management and prevention of COPD 2011. Available at: www.goldcopd.org

Albert RK, et al. *N Engl J Med*. 2011;365:689-698.

Kretsinger K, et al. *MMWR Recomm Rep*. 2006;55:1-17.

Management of Acute Exacerbations of COPD

- **Short-acting beta₂-agonist**
 - Increase dose and/or
 - Frequency
- **Oral corticosteroids**
 - In patients with FEV₁ < 50% 30-40 mg of prednisone per day for 7-10 days may be added to regimen
 - Budesonide may be considered as an alternative
- **Antibiotics should be prescribed**
 - In patients experiencing: increased dyspnea, sputum volume, and sputum purulence
 - In patients requiring mechanical ventilation
- **Consider adding an anticholinergic**

Global Initiative for Chronic Obstructive Lung Diseases (GOLD). Global strategy for diagnosis, management and prevention of COPD 2011. Available at: www.goldcopd.org

Therapy Recommendations

Group	First Choice	Second Choice	Alternative Choice
Low risk, less symptoms	<ul style="list-style-type: none"> • Short-acting anticholinergic as needed (pm) or • Short-acting beta₂-agonist pm 	<ul style="list-style-type: none"> • Long-acting anticholinergic or • Long-acting beta₂-agonist or • Short-acting beta₂-agonist + short-acting anticholinergic 	<ul style="list-style-type: none"> • Theophylline
Low risk, more symptoms	<ul style="list-style-type: none"> • Long-acting anticholinergic or • Long-acting beta₂-agonist 	<ul style="list-style-type: none"> • Long-acting anticholinergic + long-acting beta₂-agonist 	<ul style="list-style-type: none"> • Theophylline • Short-acting beta₂-agonist and/or short-acting anticholinergic
High risk, less symptoms	<ul style="list-style-type: none"> • Inhaled corticosteroid + long-acting beta₂-agonist or • Long-acting anticholinergic 	<ul style="list-style-type: none"> • Long-acting anticholinergic + long-acting beta₂-agonist 	<ul style="list-style-type: none"> • Theophylline • Short-acting beta₂-agonist and/or short-acting anticholinergic • Consider phosphodiesterase-4-inhibitor
High risk, more symptoms	<ul style="list-style-type: none"> • Inhaled corticosteroid + long-acting beta₂-agonist or • Long-acting anticholinergic 	<ul style="list-style-type: none"> • Inhaled corticosteroid + long-acting anticholinergic + long-acting beta₂-agonist or • Inhaled corticosteroid + long-acting anticholinergic + phosphodiesterase-4-inhibitor or • Long-acting anticholinergic + phosphodiesterase-4-inhibitor or • Inhaled corticosteroid + long-acting anticholinergic or • Long-acting anticholinergic + long-acting beta₂-agonist 	<ul style="list-style-type: none"> • Theophylline • Short-acting beta₂-agonist and/or short-acting anticholinergic • Carbocisteine

Further Outpatient Care

- Pulmonary rehabilitation
- Pulmonary rehabilitation, a multidisciplinary team approach
- Benefits of pulmonary rehabilitation: As a result of rehabilitation, improvements occur in the objective measures of quality of life, well being, and health status, including a reduction in respiratory symptoms and an increase in exercise tolerance and functional activities (eg, walking, less anxiety and depression, increased feelings of control, self-esteem). An observational study has also shown that pulmonary rehabilitation improves the BODE score in patients with COPD and is associated with better outcomes.[41]Pulmonary rehabilitation also results in substantial savings in healthcare costs by reducing use of hospital and medical resources.
- Components of pulmonary rehabilitation

Differential Diagnosis

Alpha 1-Antitrypsin Deficiency

Asthma

Bronchiectasis

Bronchitis

Chronic Bronchitis

Cyanosis

Diaphragmatic Paralysis

Emphysema

Farmer's Lung

Hypersensitivity Pneumonitis

Injecting Drug Use

Nicotine Addiction

Perioperative Pulmonary Management

Pneumonia, Bacterial

Pneumonia, Community-Acquired

Pneumonia, Viral

Pneumothorax

Pulmonary Embolism

Pulmonary Fibrosis, Idiopathic

Pulmonary Fibrosis, Interstitial

Respiratory Failure

Restrictive Lung Disease

Tracheomalacia

Ventilation, Mechanical

Ventilation, Noninvasive