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Факультет VI по підготовці іноземних студентів

ЗАТВЕРДЖЕНО

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МЕТОДИЧНІ ВКАЗІВКИ

для студентів англійською мовою

з дисципліни «Внутрішня медицина (в тому числі з ендокринологією)

студенти 4 курсу I, II, III медичних факультетів, V та VI факультетів по підготовці іноземних студентів

Хронічна обструктивна хвороба легенів

Харків 2016

Study subject **"chronic obstructive pulmonary disease."**

1. Hours - 4

Current Amount and *COPD,* which includes *chronic obstructive bronchitis (COPD) and emphysema (EL),* in Ukraine and in the world as a whole make up a large medical and social problem. These diseases occupy one of leading places in terms of morbidity and mortality. Specialists of the Institute of Pulmonology and Phthisiatry Sciences of Ukraine believe that about 7% of the population suffer from COPD. It should be noted that about 60% of patients with COPD is disabled group 2 with an average life expectancy of 5.5 years after the discovery of the disease.

Learning Objectives:

- teach students to recognize the basic signs and symptoms of chronic bronchitis and emphysema of the lungs;
- acquaint students with physical methods of research in COPD;
- provide students with the research methods used for diagnosis of COPD; indications and contraindications for their conduct; methods of their implementation; Diagnostic value of each of them;
- teach students to interpret the results of studies;
- teach students to recognize and diagnose complications of chronic bronchitis;
- teach students to prescribe treatment for COPD.

What the student should know?

- definition and incidence of COPD;
- etiological factors of COPD;
- COPD pathogenesis of chronic bronchitis;
- main clinical symptoms of COPD;
- General anxiety and symptoms of chronic bronchitis;
- Physical symptoms of COPD;
- methods of physical examination of patients with COPD;
- COPD diagnosis;
- diagnostic capabilities peak flow metria of chronic bronchitis, indications, contraindications;
- radiological diagnostic methods COPD;
- complications of chronic bronchitis;
- COPD treatment (change of lifestyle, nutrition, drug therapy).

What the student should be able to?

- remove the main clinical and physical symptoms in COPD;
- interpret results of biochemical and immunological studies;
- interpret peakfluometria data;
- interpret data spirography, research related flow / volume.
- interpret data of radiological diagnostic methods in COPD;
- prescribe treatment for patients with COPD.

Practical skills the students must learn^

- external examination of the patient
- examination of the chest;
- percussion;
- auscultation of the lungs.

CONTENTS THEME:

Chronic obstructive pulmonary disease (COPD) - a primary chronic inflammatory disease primarily involving the distal airways and lung parenchyma, characterized by the formation of emphysema, airflow restrictions on development are not fully reverse or irreversible airflow obstruction caused by productive and persistent non-specific inflammatory response.

Chronic obstructive bronchitis - a disease characterized by diffuse inflammation of the bronchi non-allergic, leading to progressive violations of pulmonary ventilation and gas exchange for the obstructive type and appears cough, dyspnea and sputum release that are not associated with damage to other organs and systems. It should be noted that and still has not lost its significance as determining the condition of chronic bronchitis, accompanied by cough with sputum release for at least 3 months in the last 2 years. The prevalence of COPD used in European countries ranges from 10 to 20%. The highest incidence is in England. At COPD affects about 14 - 20% of adult men and 3 - 8% of women. COPD refers to the main causes of temporary disability of working in Europe, ranks fifth among all causes of death, second only to diseases of the cardiovascular system, oncology and death from accidents. At the rate of a COPD is an important social and medical problem, and considered, along with cholesterol "disease Century" due to the high prevalence, morbidity steady growth and huge economic losses, which brings to society.

Emphysema of lungs - A disease characterized by swelling, enlargement (from the Greek Emphysema - swelling) of the lungs and is caused by hyperextension or destruction of the alveoli. All the definition of EL, which appear in modern literature, based on the opinion of the American Thoracic Society (1962), which states that the EL - anatomical alteration of the lungs that is characterized by abnormal enlargement of air spaces that are more distal than the terminal bronchioles and accompanied by destructive changes of the alveolar walls. This definition was adopted further WHO and received international recognition. EL prevalence is more than 4%, and the results of autopsies it is registered in 60% of deaths in men and 30% women.

Morphological changes in COPD.

The central airways (bronchi, bronchioles> 2- 4 mm in diameter)

1. mucus hypersecretion (stimulation of inflammatory mucous glands, increasing the number of goblet cells), squamous metaplasia ciliated epithelium; epithelial dysfunction, mucus hypersecretion, violation of mucociliary clearance; increasing the number of smooth muscle and connective tissue of the bronchi, degeneration of cartilage bronchial wall.

2. Changes in the central airways cause the symptoms of chronic cough and sputum production.

3. Changes in the central airways may exist alone or in combination with changes in peripheral airways and lung parenchyma.

Peripheral respiratory tract (small bronchi and bronchioles <2 mm in diameter)

1. The accelerated decline in lung function in COPD correlates with inflammatory changes in peripheral airways.

2. mucus hypersecretion, goblet and squamous metaplasia of the epithelium, swelling of the mucous airways; mucociliary dysfunction; swelling of the bronchial mucosa.

3. The most characteristic lesion is a narrowing of peripheral airways due to bronchial wall repair of tissue remodeling. with damage to the bronchial wall structure, with the inclusion of collagen and the formation of scar tissue that narrows the lumen of the bronchus and causes bronchial fixed.

4. Peripheral airways - a place the largest and most bronchial resistance in COPD.

Parenchyma of lungs (pulmonary gas exchange surface - respiratory bronchioles and alveoli and pulmonary capillary system).

1. The destruction of alveolar wall apoptosis of epithelial and endothelial cells.

2. Central lobular emphysema with expansion and destruction of respiratory bronchioles resulting from severe smoking.

3. Pan-acinar emphysema with alveolar destruction and expansion moves, bags and respiratory bronchioles is rare, due to shortage of α-1 antitrypsin.

4. The main mechanism of lung parenchymal destruction at central lobular pan-acinar and emphysema - an imbalance of proteases and antiproteases and oxidative stress.

The vessels of the lungs

1. Endothelial dysfunction of pulmonary arteries, which is due to the influence smoking and inflammation are common already in the early stages of COPD.

2. Restructuring vessels of the lungs due to endothelial dysfunction, intima thickening, increased smooth muscle of blood vessels, vascular wall infiltration of inflammatory cells, including macrophages and T lymphocytes (CD 8+). With the progression and severity of COPD - further increase the number of muscle mass vessels, proteoglycans and collagen deposition, further thickening of the vessel wall, may develop emphysematous destruction capillary bed.

3. Structural changes in the vessels correlate with increased pressure in the pulmonary vessels, first during exercise and later - alone.

Risk factors for COPD. *External risk factors:*

1. Long-term smoking (smoking index - 10-20 pack-years), passive smoking.

2. Industrial and domestic emissions (air pollutants, gases and steam, chemicals, bio-organic fuel combustion).

3. Infections (children of infection from severe course, respiratory infections, HIV).

4. Low socioeconomic status (restriction of food, overcrowding, hypothermia, bad habits).

Internal risk factors:

1. Genetically caused (hereditary deficiency of α- 1-antitrypsin).

2. Hyperreactivity bronchi (associated with long-term smoking, concomitant asthma).

3. Incomplete development of the lungs (complications during pregnancy of aggravating circumstances in childhood).

The pathogenesis of COPD.

1. Chronic inflammation of airway, parenchyma and blood vessels of the lungs.

2. Imbalance systems protease / antiproteases in the lungs.

3. oxidative stress (an imbalance of the system oxidants / antioxidants increase of oxidants).

4. The cells of inflammation - increased content and activity of neutrophils, macrophages, T lymphocytes (especially CD 8+), eosinophils (in some patients, particularly during exacerbations), dysregulation of epithelial cells with increased synthesis of inflammatory mediators.

5. Mediators of inflammation - leukotriene B4 (LTB 4), interleukin 8 (IL - 8), interleukin 1β (IL -1β), interleukin 6 (IL -6), tumor necrosis factor α (TNFα), endoteliy-1, substance P , vasoactive intestinal peptide (VIP), neutrophilic elastase, matrix metalloproteinaze (MMPs), cathepsin and others.

6. Airflow limitation in COPD the airways caused by a combination of bronchial lesions (COPD) and the destruction of lung parenchyma (EL), the ratio of which varies individually. Remodeling and bronchoconstriction, destruction of alveolar membranes, reduced lung elastic recoil reduces keep the airways open during exhalation.

Pathophysiology of COPD. 1. Mucus hypersecretion (due to increased mucous glands stimulation of inflammatory mediators - leukotrienes, proteinazes, neyropeptydazes) and increased number of goblet cells.

2. Squamous hypoplastic ciliary epithelium (leads to disruption of mucociliary clearance mechanisms).

Both of these mechanisms are the first in the development of COPD and may be long before the development of other pathological manifestations.

3. Restricting airflow in the bronchi - Fatal initially (by: remodulation - fibrosis and narrowing mainly small airways - the bronchi and bronchioles diameter <2 mm, causing fixed

airway obstruction, loss of lung elastic recoil due to alveolar destruction, destruction alveolar frame that supports the open state of the airways), with little feedback component (caused by mechanical obstruction of bronchial mucus, inflammatory cells, exudate plasma contraction of smooth muscle of peripheral and central bronchi, dynamic hyperinflation during exercise).

4. Excessive swelling of the lungs (because of hardening of lung tissue, the premature closure of airways worsens lung emptying, increasing functional residual capacity of the lungs, worsening respiratory muscle function and coordination against the backdrop saved or reduced contractility of diaphragm decreases inspiration is stated capacity.

5. Violation of gas exchange (peripheral bronchial obstruction, parenchymal destruction, pulmonary vascular abnormalities reduce the lung's ability to adequate gas exchange is disturbed relationship ventilation / perfusion, hypoxemia develops later - hypercapnia. Hypoxemia in the early stages when there physical loads later - alone).

6. Pulmonary hypertension (developed in the later stages of COPD, with IV stage - very severe COPD, usually after the development of severe hypoxemia (Rao_2 <8.0 CRA or 60 mm Hg. Art.) and often hypercapnia in the background. This fundamental COPD complications of the cardiovascular system, is associated with the development of pulmonary heart disease and a poor prognosis. Factors leading to the development of pulmonary hypertension in patients with COPD, hypoxic vasoconstriction, remodeling of pulmonary arterioles, intima's hyperplasia, hypertrophy / hyperplasia of smooth muscle; destruction of pulmonary capillaries due to emphysema, which subsequently leads to increased pressure in the pulmonary vessels required to Perfusion gases through the wall of blood vessels).

7. Pulmonary heart - right ventricular hypertrophy due to dysfunction and / or structure of the lungs, except states where violations of state is the result of lung disease that primarily affects the left departments (congestive heart failure). Lack of right heart associated with venous stasis and thrombosis, which may lead to pulmonary embolism, which further deteriorate the pulmonary circulation.

8. Systemic effects - extrapulmonary disorders. COPD, particularly in patients with severe disease, causes systemic effects, which greatly aggravate the patient's condition and initiate the development of associated diseases and worsen the prognosis regarding survival. So, are typical:

- Cachexia loss of fat mass;
- The loss of skeletal muscle and its weakness;
- Osteoporosis;
- Depression;
- Anemia;
- Increased risk of cardiovascular disease.

System components COPD are the result of chronic inflammation with activation of inflammatory cells, abnormal concentrations of circulating cytokines, and increased levels of inflammatory mediators. An important pathogenetic role of oxidative stress, tissue hypoxia and other metabolic disorders, especially in low physical activity, sedentary lifestyles is characteristic of patients with COPD.

Classification of COPD. COPD is divided into 4 stages according to the severity of the disease, which is determined by a survey of patients in clinically stable period in the absence of exacerbation. This approach allows the patient to develop a plan with COPD basic treatment that must consistently perform. In determining the stages of COPD accounted severity of clinical signs of disease and functional characteristics of bronchial obstruction syndrome.

Signs stage (severity) of COPD

and stage degree severity course COPD	Symptoms of COPD
And easy	- FEV $_1$ / FVC <70% - $_{FEV1>}$ 80% of the relevant - Usually, but not always, chronic cough, sputum selection
II, moderate	- FEV $_1$ / FVC <70% - 50% $_{<FEV1}$ <80% of the relevant - Symptoms progress, there is shortness of breath during exercise and during exacerbations
III, hard	- FEV $_1$ / FVC <70% - 30% $_{<FEV1}$ <50% of the relevant - Increasing shortness of breath, recurrent exacerbation, which affects the quality of life of patients
IV, very hard	- FEV $_1$ / FVC <70% * $_{FEV1}$ <30% of the appropriate or FEV $_1$<50% due to the presence of chronic pulmonary disease - Further progression of symptoms, quality of life significantly impaired, exacerbation can be life-threatening

Note. * - If very severe course of COPD and a significant decrease in FVC ratio of FEV $_1$ / FVC increases the diagnostic value of losing.

Clinical classification of chronic bronchitis.

I. Forms: Simple, purulent, obstructive, purulent-obstructive

II. The disease currence: latent, with rare aggravation, with often aggravation, recidivous currence

III. The phases of the process: with aggravation, remission;

IV. Complications: emphysema of lung, blood spitting, lung failure;

Secondary pulmonary hypertension: a) transient phase, b) stable stage without circulatory failure c) stable stage of heart failure

EL Classification by Putov NV (1984)

1. Pathogenesis:

a) primary (idiopathic, essential), which occurs in the intact lung;

b) secondary, which occurs against other lung diseases.

2. prevalence:

a) diffuse;

b) localized.

3. The morphological features:

a) panatsynar (panlobular) - with the defeat of a acinus;

b) central lobular - with damage to the central part of acinus;

c) peryatsynar (perilobular) - acinus with damage to the periphery;

d) non-reginal (localized at the scars and c);

e) Bullous - with a large number Boulevard - emphysematous cavities with a diameter greater than 1 cm.

4. Specific forms:

a) congenital partial EL;

b) –Mac-Laeod Syndrome.

In this isolated *interstitial* (when air enters the lungs stroma peribronhial, perylobular) and *alveolar* (when excess air is in the alveoli) EL. There are *acute* obstructive emphysema, which occurs in asthma and *chronic.* In general involutive changes develope *nonobstructive*

senile emphysema. It also belongs to the so-called nonobstructive *vicariously in behalf emphysema,* characterized by the expansion of the lung tissue remaining after removal of part or parts of the lungs.

Classification of EL by Serkova V.K., M.A. Stanislavchuk, Monastursky Y.I. (2005)

1. Only the extensions:

a) indiscriminate distribution (compensatory emphysema; emphysema associated with partial obstruction of the main bronchus)

b) selective distribution, mainly affecting the respiratory bronchioles (pulmonary emphysema caused by dust).

2. The destruction of the walls of airspace:

a) indiscriminate distribution (pan-acinar destructive emphysema);

b) selective distribution, mainly affecting the respiratory bronchioles (central lobular emphysema);

c) uneven distribution (uneven emphysema).

International Classification of Diseases 10 th revision

J40. Bronchitis, not specified as acute or chronic

Note: unspecified bronchitis in children aged under 15 can be considered as acute and so entered in section J20.

Excludes: bronchitis:

• allergic NOS (J45.0)

• asthmatic NOS (J45.9)

• caused by the chemicals we (acute) (J68.0)

J41. Simple and mucopurulent chronic bronchitis Excludes: chronic bronchitis:

• NOS (J42)

• obstructive (J44.-)

J41.0 Simple chronic bronchitis

J41.1 mucopurulent chronic bronchitis

J41.8 Mixed, simple and mucopurulent chronic bronchitis

J42. Unspecified chronic bronchitis

Excludes: chronic (a):

• asthmatic bronchitis (J44.-)

• bronchitis:

• Simple and mucopurulent (J41.-)

• with airflow obstruction (J44.-)

• emphysematous bronchitis (J44.-)

• obstructive pulmonary disease NOS (J44.9)

J43. Emphysema

Excludes: emphysema:

• compensatory (J98.3)

• caused by chemicals, gases, or vapors (J68.4)

• interstitial (J98.2)

• newborn (R25.0)

• mediastinal (J98.2)

• surgery (subcutaneous) (T81.8)

• traumatic subcutaneous (T79.7)

• chronic (obstructive) bronchitis] (J44.-)

• emphysematous (obstructive) bronchitis (J44.-)

J43.0 syndrome McLeod

Panlobular emphysema J43.1

J43.2 Emphysema central lobular

J43.8 Other emphysema

J43.9 Emphysema, unspecified

J44. Other chronic obstructive pulmonary disease
Includes: chronic:
• bronchitis:
• asthmatic (obstructive)
• emphysematous
• bronchitis of:
• airflow obstruction
• emphysema
• obstructive (a):
• asthma
• bronchitis
• bronchitis
Excludes: asthma (J45.-)
asthmatic bronchitis NOS (J45.9)
bronhoektasia (J47)
chronic:
• bronchitis:
• NOS (J42)
• Simple and mucopurulent (J41.-)
• tracheitis (J42)
• bronchitis (J42)
• emphysema (J43.-)
• lung disease, caused by external agents (J60-J70)

Clinical manifestations of COPD.

A. **Chronic cough** *:* 1) is usually the first symptom of COPD in that is before shortness of breath; 2) may initially occur occasionally, over time - daily worries; 3) are more concerned about the day, at least - at night; can be counter-productive, without providing specimens; in some cases may be absent.

B. bold sputum - usually in small numbers, mucus after coughing.

C. Shortness of breath - progressive (gradually enhanced over the years); persistent (worried sick day); occurs or worsens during exercise, causing his poor tolerance; in the future there alone and significantly limits livelihoods; increases during respiratory infections; can be defined as patients' need for additional efforts at breath "," breathing discomfort, "" chest compression, "shortness of breath.

D. The presence of a history of risk factors, especially: smoking, exposure to industrial dust and chemicals, kitchen smoke, combustion products bioorganic fuel.

E. In severe COPD may be weight loss , anorexia; osteoporosis; depression and / or anxiety, anxiety (systemic consequences of the disease).

At *COPD* is a syndrome *of chronic bronchial obstruction* - number of shocks cough increases significantly decreases their strength (determined by the strength of the flow of air with his hand raised to the mouth of the patient). When coughing patient is severely strained, neck veins swell, the skin of the chest and face red, but released a small amount of sputum. Most often unproductive cough is in morning. During the day, improving bronchial permeability, drainage of mucus increases - the patient "coughts." Unproductive cough does not perform drainage functions, but promotes emphysema and bronhoektase and so should is inhibited drugs. In the later stages of the disease because of the death reflex zones cough reflex may become extinguished, and the cough persists even in the presence of a large number of purulent sputum. In such cases, should be encouraged to cough complete drainage of the bronchi.

The syndrome of bronchial obstruction breathlessness occurs imperceptibly, slowly, but then steadily progressing. In 25% of patients with COPD dyspnea long time the only symptom. The severity of dyspnea is variable, depending on weather and other factors -

temperature, humidity, atmospheric pressure, smells, etc. Recurrent bronchial obstruction in the COPD must be differentiated from bronchial mucus transient obturation morning, manifested feeling shortness of breath and cough disappears after sputum.

Bronchospasmodic syndrome is characterized by its own specific version of coughing, which occurs paroxysmal, often for no apparent reason, has character takes 30-40 minutes has typical circadian rhythm (evening, night), eliminating the intake bronhospazmol i tics. This cough is often accompanied by breathing "of the whistle "and a sense of wheezing and breathlessness.

Lung failure is often accompanied by symptoms hyperkapniae. Reflex expansion of cerebral vessels appears headache that is worse at night against the backdrop hypeventilation, arrhythmia sleep - daytime sleepiness and insomnia at night, anorexia, tremor or close up to the court. Severity of impaired consciousness is variable - from reduced ability concentrate attention in to confusion, stupor and coma. Expanding brain blood vessels can lead to intracranious hypertension, nipple swelling of the optic nerve. Vasodilation vessels of the skin manifests face and neck flushing, increased sweating.

The syndrome of intoxication accompanied by severe exacerbation of chronic bronchitis, manifested general malaise, chills or sweating, especially at night (a symptom of "wet bag"), drowsiness, tachycardiasi tsiey at normal or low-grade fever.

The main symptom of EL is *expiratory dyspnea* which occurs first during exercise and later at rest. Dyspnea worse when weather changes in autumn and winter, colds, bronchitis during exacerbations during an attack of coughing. Sigh patients elongated, often reminds chug, but weakened so that the patient can not always blow out the flame. *Coughing* often has paroxysmal character, with little sputum, accompanied by a slightvwhistling.

In clinical practice, there are three degrees EL:

1 - not determined the absolute heart dullness, the lower limit of light is not changed, but the tour of lung edges in l . Axyllaris media is reduced to 4 cm ;

2 - the omission of the lower boundary lung on one edge and limit tours to 2 cm ;

3 - the lower limit of light is shifted down by 2 ribs tour of lung edges missing .

EL clinical picture depends on the presence and severity of respiratory failure and the degree of hypoxemia.

Physical examination. Physical symptoms usually appear with severe COPD. The most common physical signs in patients with COPD and severe course is central cyanosis, cyanosis of mucous membranes; box-like big chest, flattening the dome of the diaphragm, breathing part in supporting muscles, reduce cardiac dullness on percussion, expanding xifosternalic angle; increase in respiratory rate (> 20 / min.), breathing depth reduction; prolonged exhalation; patients exhale everywhere closed lips (to slow exhale and improve emptying of the lungs); auscultation - easing respiratory noise, whistling wheezing during quiet breathing.

In case of *EL* chest becomes box-likes or bell-likes forms: anter-dorsal chest size increases for developing thoracic spine, ribs become horizontal direction costales angle is obtuse. Supraclavicular holes explode neck becomes shorter. Respiratory excursion and reduced chest, accompanied by a noticeable tension auxiliary respiratory muscles, drawing intercostal spaces.

Peripheral osteodystrofiya (a symptom of "drumsticks") observed in chronic bronchitis is rare.

Percussion changes determined by the presence emfizema of lungs. Percussion sound gets boxed shade in combination with pneumosclerosis there is a "mosaic" pattern percussion - blunting duty areas and areas with a box sound. The lower boundary lung lowered sharply reduced their mobility. Because of "spreading" medial edges of the lungs the size of cardiac borders reduced, relative cardiac thickness boundaries become indistinct character.

In auskultation the earliest sign of bronchial patency is *lengthening the exhalation* - the normal ratio of the length of inhalation and exhalation is 1: 1.1 - 1: 1.2. Often defined breathing

hard, which is unequal, "gritty" kind. Emfizema lungs leads to a weakening of breath ("Watt breathing").

A frequent symptom auscultation COPD is *wheezing* , toness which depends on caliber bronchial lesions that they caused. The smaller bronchi caliber affected, the higher tone wheezes. Any damage to the small bronchi, accompanied by accumulation of viscous mucus in them, their lumen narrowed due to bronchospasm, wheezing with whistling appear high tonalnost. The primary lesion of the bronchi medium caliber manifested wheezing and a buzzing. In large bronchi having low bass wheezing. Since the air flow rate, which determines the sound of wheezing, breathing in certain phases in different caliber bronchi, wheezing low-frequensy amplified in the inspiratory phase, high - a phase of exhalation.

Reveal hidden signs of bronchial obstruction (extension of the trachea and wheezing with whistling at the height of the trachea) may auscultation of the patient in a horizontal position and with forced breathing. Dry and wet wheezing differ instability may disappear after coughing.

Wet wheezing occur in the presence of liquid mucus in the lumen of the bronchi. Character wet wheezing determined caliber bronchi. Big bubble wet wheezing listen or in the projection of large bronchi, or over cavities in the lungs bronchiectasis. The presence of wheezing wet big bubble peripheral regions of the lungs with no major bronchi, bronchiectasis is a reliable sign. Middle bubble wheezing characteristic cylindrical bronchiectasis.

In examination *of the cardiovascular system* may show abnormal pulsation in epihastric caused or right ventricular hypertrophy, or positional displacement due to severe heart emfizema of lungs. Pulse is often full, jumping . In 25% of patients with chronic bronchitis appears pulmonohenna arterial hypertension caused by resistant hyperkapnia. Cardiac be muted (emfisematic weakening), in pulmonary hypertension listen accent II tone of the pulmonary artery. In chronic pulmonary heart disease with right ventricular failure can listen to gallop over the xiphoid process.

In severe EL there is a positional shift liver down. This liver painless, elastic, not relieved by diuretyks, not combined with edema.

Two characteristic phenotypes of patients with COPD

Ppink puffe. Emphysematous type - benefit emphysema

- Mostly dyspnea relatively constant
- Skin pink-gray hue
- Patients often asthenic physique
- Body weight decreased
- Cough after joining of shortness of breath, dry
- Sputum was released
- Chest increased in volume
- Breathing weakened
- Rattling absent in the lungs
- hypoxia at rest is not observed, but exercise tolerance in them is much reduced
- Often they occupy forced posture with tilt forward and fixation of the shoulder girdle
- exhalation made by lips
- Sometimes, usually in the later stages of emerging pulmonary heart
- Pulmonary hypertension expressed no or minimal

"The blue and puffy» (blue bloater). Bronchitic type- advantage bronchitis

- Mostly cough
- Piknic constitution, the tendency to be overweight
- Diffuse cyanosis at rest
- Shortness of breath moderate, increasing with exacerbation
- Swelling of legs
- Rattling in the lower lung
- Strengthening II tone of the pulmonary artery
- Early development of chronic pulmonary heart

• Inter acinar emphysema

Additional methods of research in COPD. *1. Radiographic signs .* When X-ray - the lungs of large volume, low standing diaphragm, narrow shadow of the heart, increased retrosternal airspace, sometimes emphysematous were determined.

2. Investigation of respiratory function (ERF). Spirometry is mandatory for diagnosis of COPD, it is needed and to assess the seriousness of the disease and for periodic monitoring to assess disease progression.

To detect the disease early on spirometry preferably carried out with complaints of chronic cough and sputum production even in the absence of dyspnea.

Spirometry to measure, the maximum volume of air exhaled during forced exhalation from the point of maximal inspiration is stated (FVC); the volume of air exhaled in the first second of this maneuver (forced expiratory volume in the first second, FEV_1); the ratio of these two measurements (FEV_1 / FVC); peak expiratory volume velocity $NEPs_{view}$ and instant expiratory volume at different levels of FVC (MOS25, 50, 75, 25-75). The resulting spirometric parameters evaluated by comparison with appropriate for each patient, which is calculated based on age, height, sex and race of the patient.

For patients with COPD *mild to moderate severity* characterized as a slight decrease in FEV_1 and FVC. The degree of spirometric disorders reflects the severity of COPD. The values after administration of bronchodilators FEV_1 <80% against the ratio of FEV_1 / FVC <70% confirms the presence of airway limitation that is not fully reversible. Value FEV_1 / FVC is a sensitive indicator of the presence of airflow obstruction, and its decrease <70% believe an early sign of airway restriction in patients with FEV_1 is normal (> 80% predicted).

Peak expiratory volume ($NEPs_{type}$) is used to determine airway limitation, but in COPD correlation between $NEPs_{view}$ and FEV_1 - low. Determination of $NEP_{species}$ may lead to an underestimation of the degree of bronchial obstruction in these patients. If you can not hold spirometric study, the presence of lengthening forced expiratory 6 seconds is rough but useful definition - predictor reduction ratio of FEV_1 / FVC <50%. At the same time, a significant reduction in FVC in parallel with a decrease in FEV_1 in the case of severe course of COPD slightly reduces the diagnostic value ratio of FEV_1 / FVC.

To the differential diagnosis of asthma and to determine the best indicator FDD personally for each patient test is done to determine the reversibility of bronchial obstruction in a sample of bronchodilators.

Method of test: before the study, patients must refrain from taking short-acting bronchodilators - at least 6 hours, extended release - 12 hours, long-acting anticholinergics - 24 hours. Spirometry (research FEV_1) is performed before and 15 minutes after inhalation of large volume spacer through 400 mg β_2 agonists short-acting or 30-45 minutes after inhalation of 80 mg of short-acting anticholinergics or combined bronchodilators (ipratropium bromide + salbutamol, ipratropium bromide + fenoterol). The increase in FEV_1 at 12% of initial value + increase it in absolute values $\geq$ 200 ml indicates the presence of bronchial obstruction reversibility.

With the progression of the disease increases bronchial obstruction, increasing overall bronchial resistance (R_{tot}), occurs and increases expiratory swelling of the lungs, developing air traps in the lungs, resulting from the loss of elastic recoil of the lungs and the collapse of airways redistributed structure of total lung capacity (VLC): increases functional residual capacity of the lungs (FVLC), pulmonary residual volume (RV) increases the ratio of RV / VLC; reduced capacity of inspiration is stated (There $_{WA}$) and backup volume inspiration is stated (PO_{WA}). For the purpose of comprehensive assessment of these indicators should conduct more complete and informative research - *general bodipletyzmography* . In difficult diagnostic cases and to address the issue of surgeries performed measurements of the diffusion capacity of the lungs. In the later stages of COPD significantly increased pulmonary residual volume (RV) in the structure of total lung capacity (VLC) and the ratio of RV / VLC is 40%. This increases the disproportionate ratio of ventilation / perfusion (V / Q), which leads to disruption of gas exchange.

In order to monitor the progression of COPD, evaluating the effectiveness of remedial measures applied in specific patient spirometry performed annually. So, if healthy annual decline in FEV_1 <30 ml, in patients with COPD - 30-60 ml or more.

3 . *Measurement of arterial blood gases* performed in patients with FEV_1 <40% due, or in the presence of clinical manifestations of lung failure, right heart failure. Pulmonary insufficiency Ra0 point $_2$<8.0 Kra (60 mm Hg. In.) With / without $RaS0_2$ > 6.7 Kra (50 mm Hg. Art.) In arterial blood. *Assessment of pulmonary hemodynamics* - is important only in the development of lung failure.

Diagnosis COPD. By binding methods include *research FDD, sputum cytology, X-ray of chest,* and additional (indication) - *microbiological examination of specimens, determination of blood gas composition, immunological, exercise testing.*

1. Hemography. In the clinical analysis of blood in patients with COPD may be secondary polycythemia with increasing hematocrit and increased hemoglobin levels that compensate for a chronic condition in patients with respiratory failure. Sometimes polycythemia masked proportionate increase in plasma volume. Erythrocytosis is fixing or reducing ESR, blocking leukocyte reaction. Many patients with COPD determined eosenophylia and varying degrees expression. In marked exacerbation of the disease can occur leukocytosis.

2. Protein fractions of blood serum. In the biochemical analysis of blood serum shows signs of active inflammation, increased levels haptohlob i well, with cialic acids seromucoid, C-reactive peptide, α-2-globules, at least - α-1 - and γ-globules , decreases album i n globules i new factor. The activity of inflammation in the bronchi responsible way increase the concentration c i Alov acids, CRP, α-2-globules i electrons in plasma.

3. Investigation of specimens. For this and mikroskopiy judge of character and sputum inflammation in the bronchi. Mucous and muco-purulent sputum revealed at catarrhal bronchitis. When purulent sputum purulent bronchitis nature neutrofil contains a large number of white blood cells, macrophages, cells of the bronchial epithelium.

Indirect signs of obstructive syndrome and bronchospasm have pus and mucus plugs, bronchial casts. In liquid form fibrotic chronic bronchitis sputum are "bronchial tree."

Typical change of physical and chemical properties of sputum test . At COPD morning sputum is alkaline, daily - an acidic or neutral. Deviations from the optimal values of viscosity and phlegm elasticity is slowing mucociliar transport requires mukolitics. Typically, mucous sputum elasticity has high and low viscosity, purulent - low elasticity and high viscosity. The activity of inflammation in the bronchi is in line with the increase of sialic acids, total protein, Ig A DNA in the sputum.

To establish the etiological diagnosis (determine the infectious agent) is used *microbiology of sputum* or bronchial washings, bronchial aspirate from, rarely - strokes of the larynx or pharynx. Terms of sputum sampling for the study: in the morning, before the cough, the patient cleans teeth and rinse your mouth three times with boiled water. Then sputum collected in a sterile jar and sown on nutrient media within 1-2 hours after collection.

4. Immunological research. Perhaps imunological determine the nature of the pathogen by paired sera: identification of a sharp increase in the number of antibacterial antibodies and bacterial antigens in serum and early aggravation 1-2 weeks.

5. X-ray examination. Radiographic changes occur not more than 30% of patients with chronic bronchitis, usually with many years of illness. Uncomplicated bronchitis has no specific radiological symptoms. Possible complications accurate radiological diagnosis of chronic bronchitis: pneumosclerosis, emphysema lungs, pulmonary hypertension, chronic pulmonary heart.

- *Net fibrosis* radiographically evident pulmonary picture enhancement (increased number of elements pulmonary figure per unit area of the lung field), diffuse cellular net-form or deformation of lung pattern. These changes caused peribronhial sclerosis, multiple sclerosis intra-acinar, intra-lobular and intra-segment partitions. Net-form deformation of lung pattern typical of COPD with lesions of small bronchi. Yellow macroporous deformation of lung pattern

may be due to bronchiectasis. Fibrosis more pronounced in the basal parts of lungs. Typical reduce the difference in transparency lungs on inhalation and exhalation.

- *Pulmonary hypertension* is characterized by a decrease in caliber of small peripheral vessels as a result of general - vascular spasm against the background of the expansion of major branches of the pulmonary artery - a symptom of "jump cal i Bru". Expanding the descending branch of the right pulmonary artery - more than 16- 18 cm . Cone pulmonary artery bursts.

6. Bronchography. Bronchographic study can detect signs of chronic bronchitis in 97% of patients. The accumulation of mucus in the bronchi leads to regional bronchial contrast, the occurrence of defects filling curves internal circuits, fragmented fill the bronchial tubes, reducing the number of lateral branches of the bronchi and breakage of the blunt end of the peripheral ends - a symptom of "chopped off bitch", occurs in 80% of patients. Bronchospasm manifested in the uneven bronhography decrease bronchial lumen - a symptom of "threads of similar bronchi."

In the lower regions of the bronchial tubes are often on one Oki bronchiectasis. Signs of peripheral bronchi is bronchiectasis ("lake") diameter round shadow 1- 3 mm at the end of the small bronchial branches. Bronchographic signs EL is sliding peripheral branches of the bronchi, accompanied by an increase in small corners branching bronchi.

7. Studies of lung function. For monitoring of respiratory function in the home is recommended to determine *peak expiratory flow rate* using portable cutlery - *pikfluorometrias.*

For diagnosis, choice of adequate treatment, prognosis of the disease is to find out whether the patient's bronchial patency how reverse these changes and that their mechanism - these changes are adrenergic effects.

Traheobronhial tree on anatomical and physiological features are divided into three "floors": out-chest respiratory tract, central parts extra-chest airways (bronchi large order of 1-4 and 5-7 small bronchi order), peripheral extra-chest airways (8-10 bronchi order bronchus i hen 11-16 order).

According to the release *several options obstructive syndrome.*

1. At the level of airways - fixed or valve stenosis of the trachea.

2. At the level of the central department intra-chest respiratory tract - the trachea and large dyskineziya bronchial inflammatory or spastic narrowing of the large bronchi.

3. At the level intrathoracik peripheral regions of the respiratory tract - isolated obstruction of small airways , EL.

For this and spirographic research conducted differential diagnosis of chronic nonobstructive bronchitis and COPD .

Chronic non-obstructive bronchitis is characterized by normal levels VC, aerodynamic breathing resistance, FEV_1 and $NEPs_{view}$.

COPD appears irreversible syndrome of bronchial patency of steady decline in FEV_1 and increased breathing resistance aerodinamic. In addition to detection of bronchial obstruction i th, in various pharmacological tests determine the return and irreversible component of bronchial obstruction.

8. Research the blood gas and acid-base status.

Gas composition of blood and acid-base status characterize the severity of disease, determine the main directions corection violations homeostasis. The easiest method for diagnosing arterial hypeksemia is a sample of oxygen - after inhalating severity of cyanosis the patient is reduced. For patients with COPD typical hyperkapniaeiya due alveolar hypeventilation. Hyperkapniaeia inevitably leads to respiratory acidosis.

A more accurate method, while specifying the following parameters: pH, carbon dioxide tension, oxygen tension, excess or shortage of buffer of bases in blood. Respiratory acidosis with a decrease in blood pH decreases the affinity for oxygen and oxygen saturation of arterial blood. Promotes arterial hypeksemiyi at COPD and violations of ventilation-related perfuziyn, shunting blood to the lungs from right to left.

9 . Endoscopy bronchi. Bronchoscope i am not a mandatory method of research at COPD. Typically, it resorted to the presence of symptoms is not characteristic of bronchitis - hemoptysis, severe astenization. Bronhoskopia to determine the morphological form endobronhitis (catarrhal, purulent, atrophic, hypertrophic, fibrotic-ulcerative, granulating, hemorrhagic) and the nature of the secret and the state intracartilagous intervals. When endoscopic examination receive information about the functional status of the bronchial tree, as the severity f trachea-bronchial dyskinesia, the presence of static bronchi retraction. During the bronchoscopia receiving washings or aspirate for microbes and cytology.

10. Electrocardiography. Electrocardiography research necessary to detect secondary pulmonary hypertension. The most frequently observed following signs of right ventricular hypertrophy.

1. Severe electrical axis deviation to the right heart.
2. Shifting the transition zone R / S left to V 5-6.
3. S-type ECG.
4. T Inversion in leads V 1-4 due to severe hypoxemia and hypercapnia with significant right ventricular failure.
5. The most information criteria - rate R / S under 2.5 when recording esophageal ECG leads at the stomach.

11. Echocardiography is of secondary importance, can detect hypertrophy and dilatation of the right ventricle, paradoxical motion of the interventricular septum, increased the estimated value of the average pressure in the pulmonary artery. This sensor is advisable to place in the right upper quadrant.

EL diagnosis. Patients with EL subject of respiratory failure occurs *secondary polycythemia, increased hemoglobin levels, reduced ESR.* If bronchopulmonary infection in the blood revealed leukocytosis and increased ESR. Serum observed *deficiency of α -1-antytrypsinu* . In the *X-ray* revealed a syndrome of limited or widespread enlightenment, increased transparency of the lungs, low standing diaphragm, limiting its respiratory excursions, pulmonary vascular relaxation picture. EL considered pathognomonic for local or diffuse peripheral devascularisation. Heart shadow narrowed, elongated - "drip heart." *Bronchoscopy* provides an opportunity to find out the degree of bronchial inflammation with concomitant bronchitis. *Spirographic study* allows to study increasing volume of air, reducing the VC. When EL increased breathing resistance, reduced the maximum rate of exhalation. *Computed tomography* demonstrates hyper airness of lung tissue, same of vascular pattern, the presence Boulevard. *The ECG* often observed deviation of electrical axis of the heart to the right, the signs of right ventricular hypertrophy, left bundle branch block. Signs of pulmonary hypertension in the *echocardiography* believe hypertrophy of the right ventricle wall (more than 5 mm), increasing the amplitude of motion, dilatation of the right ventricle (more than 25 mm provided increasing ratio of the size of the right and left ventricles of more than 0.5).

Complications COPD.

-chronic pulmonary insufficiency;
-chronic pulmonary heart;
-Gentle recurrent infection respiratory tract - acute pneumonia
-obstructive pulmonary emphysema, fibrosis;
-spontaneous pneumothorax

Differential diagnosis of COPD. In conducting the differential diagnosis of COPD is most often necessary to exclude *asthma* . A also differential diagnosis of COPD with chronic nonobstructive bronchitis, bronchiectasis, cystic fibrosis, tuberculosis, diseases of the cardiovascular system (chronic heart failure), accompanied by shortness of breath, obliterative bronchiolitis, diffuse panbronhiolitis.

On the basis of functional respiratory study, clinical course excludes *chronic nenobstruktive*, the specific nature of the obstruction, which is caused by *cystic fibrosis* .

Cystic fibrosis (CF) - the most common monogenic hereditary disease with autosomal recessive mode of inheritance, which can be characterized as universal endocrinopathias. The main manifestations of CF is a chronic obstructive process in the airways, accompanied by recurrent bacterial infection, violation of the digestive system to the lack of exocrine pancreatic function, high content of electrolytes in sweat and obstructive azoospermia in men due to congenital agenesis of the ejaculatory ducts.

The etiology and pathogenesis of CF **.** The cause of the characteristic pathological changes in the patient's CF is the presence of mutations in both alleles of the gene localized on the long arm of chromosome 7. This gene controls the synthesis of the protein cystic fibrosis transmembrane regulator (CFTR), which functions as an adjustable cyclic adenosine monophosphate chlorine feed on the apical surface of epithelial cells. Violation transport of chloride ions across the apical membrane of epithelial cells due to mutations of the gene CFTR increases the reabsorption of sodium cells, changing electrolyte composition and dehydrate secret exocrine glands, and is the cause of the pathophysiological processes in the development of basic and clinical manifestations.

Classification MB. 1. CF with pancreatic insufficiency. 2. CF without pancreatic insufficiency in t. H. Primary genital form of congenital bilateral aplasia ejaculatory ducts (VBASP). 3. Atypical forms MW (for atypical forms of CF include cases of chronic respiratory disease of varying severity of CF symptoms characteristic (or the only clinical symptom MW) in the case of normal exocrine pancreatic function and normal (<40 mEq / L) or bordering on normal sweat chloride levels. *Diagnosis MB.* The diagnosis of CF is considered significant in the case of two criteria (at least one of the positions). *criteria for diagnosis joints MV:* 1. one or more typical change in phenotype or disease CF brothers or sisters (family history) plus 2. Increased concentration of sweat chloride results by 2 -x or more studies using pilokarpin for gibbons and Cook or identification of two mutations in the CFTR gene. Characteristic CF phenotype changes that have diagnostic value.

1. Chronic respiratory disease, which manifests as:

a) chronic cough with viscous sputum;

b) persistent colonization / infection of the airways typical CF pathogenic microorganisms (Staphylococcus aureus, and Pseudomonas aeruginosa);

c) persistent changes on radiographs of the thoracic cage for a (eg, bronchiectasis, atelectasis, infiltrates, hyperinflation);

d) airway obstruction, which shows intermittent whistling wheezing and breathing;

e) nasal polyps; sinusitis or radiographic changes in the paranasal sinuses;

e) deformity of the distal phalanges as "drumsticks."

2. Changes in the digestive system and nutritional status, namely:

a) in the gut - mekonial ileus, a syndrome of obstruction distal small intestine (the equivalent mekonial ileus), rectal prolapse;

b) in the pancreas - pancreatic insufficiency with typical changes stools, recurrent pancreatitis;

c) in the liver - clinical or histological signs of focal biliary cirrhosis or cirrhosis multi-lobular;

d) violation nutritional status - displays insufficient assimilation of food components (lack of weight and body length regarding appropriate age);

e) hypoproteinemia with edema and anemia, secondary deficiency of fat-soluble vitamins.

3. The syndrome of acute salt loss, chronic metabolic alkalosis.

4. obstructive azoospermia in men is associated with congenital bilateral aplasia ejaculatory ducts.

The main *criterion for the diagnosis of CF* recognized the results of clinical diagnosis and paraclinical research methods.

Family history. Individuals who have (or had) brothers or sisters of patients with CF, there is a risk of 25% to be too patient. Therefore, all siblings with CF to exclude the diagnosis to be meticulous clinical examination, carrying out sweat tests and molecular diagnostics. 2. The sweat test. This test is the "gold standard" in the diagnosis of CF. The classic method for Gibson Cook is to determine the concentration of chloride ions and sodium (or only chlorine) in portions sweat obtained exclusively to standard iontophoresis with pilocarpine. A positive sweat test is considered at a concentration of chloride than 60 mEq / L questionable - at 40 - 60 mEq / L, negative - at 40 mEq / L or less. Positive sweat test in most cases, if there is at least one clinical manifestation MoU confirms the diagnosis. But negative sweat test does not mean the absence of CF patients.

The question of the presence *of bronchiectasis* is decided based on the reverse a history of infections, release of large amounts of purulent sputum, bronchodilation, thinning the walls of the bronchi (according to X-ray study, CT). For *tuberculosis* characteristic of a typical X-ray picture, microbiological confirmation of disease. *Chronic heart failure* is manifested by small crispy wheezing on auscultation, the X-ray - enlarged heart, pulmonary edema, restrictive changes on the background of the stored airway in the study FDD. For *obliterative bronchiolitis* typical start at a young age; develops mainly in property; rheumatoid arthritis, the effect of smoke in history; areas of low density during expiration on computed tomography (CT). *Diffuse pan-bronhiolitis* affects mostly women and smoking; presence in most of them chronic sinusitis; diffuse small central lobular nodular opacity and hyperinflation in the X-ray study and CT high resolution. Unlike COPD for *asthma* typical onset in early childhood, often as a child, concomitant allergic pathology upper respiratory tract, skin; family history of asthma, spontaneous lability of clinical symptoms and bronchial obstruction, resulting in a significant daily variability of FEV_1 and $NEPs_{view}$. Reversibility of bronchial obstruction in response to β_2 agonists and pronounced effect of long-term anti-inflammatory therapy steroids characteristic of asthma. While the response to these drugs in COPD are generally less pronounced.

Treatment of COPD. Pharmacotherapy assigned to prevent and control symptoms; reducing the frequency and severity of exacerbations, prevent the development of complications of the disease and reduce their severity, improve physical endurance; improve overall health and quality of life of the patient; reduce mortality.

Basic principles of treatment of patients with COPD:

- Gradually increasing the intensity of treatment depending on the severity of the disease;
- Regularity, continuity of basic therapy according to the severity of the disease;
- The variability of individual response to treatment determines the need for careful and regular monitoring of clinical and functional signs of the disease.

Therapeutic modes customized for the individual patient with COPD is caused by:

- Severity of symptoms;
- Degree of violations FDD;
- The frequency and severity of exacerbations;
- The presence of complications of COPD;
- The presence of pulmonary disease;
- The presence and severity of comorbidities;
- General health.

In the treatment of COPD *preferred route of administration is inhalation drugs* - bronchodilators, inhaled corticosteroids, combination therapies. Benefits inhalation following: the active ingredient is delivered directly into the airways, creating a locally high concentrations when using lower doses to minimize systemic side effects; no response from the gastrointestinal tract and elements of drug interactions. The effectiveness of this route of administration to a large extent depends on the possession patient inhalation technique. Patients with COPD may have problems with coordination, they may have difficulty using MDI (GAI). Therefore, the appointment of inhaled forms of medication necessary to ensure the correctness of technology application and ix periodically check it on during the treatment period.

With the appointment of high doses and for the improvement of inhalation technique is recommended to use a large volume spacer (much lower oropharyngeal deposits drugs, which reduces the local adverse effects with inhaled corticosteroids (ICS) - oral candidiasis, dysphonia - and systemic effects with β_2 agonists by reducing drug absorption from the gastrointestinal mucosa, the use of spacers lead to 2 - 4 fold increase depozytsiyi drug in the lungs compared to the traffic police. The use of nebulizers allows the use of high doses of therapeutic and receive answers within a short period of time, as well as the simultaneous oxygen in the circuit (if necessary).

1. **bronchodilators**

- Improve bronchial patency, changing the tone of smooth muscles of the bronchi;
- Improve emptying of the lungs, reducing hyperventilation at rest and during exercise;
- Improve physical endurance;
- Take center stage in the symptomatic treatment of patients with COPD;
- Appointed as regularly as the standard treatment to prevent or reduce persistent symptoms, and "if necessary" to relieve some acute symptoms;
- Prevail inhaled bronchodilators form.

Choose between inhaled bronchodilators - β_2 agonists short (salbutamol, fenoterol) and long-acting (salmeterol, formoterol) and short anticholinergics (ipratropium) and long-acting (tiotropium), or a combination depends on:

- Their presence;
- The severity of the disease;
- Individual answers to reduce the symptoms of side effects.

Inhaled β_2 agonists, short acting (salbutamol, fenoterol) have a relatively rapid onset of bronchodilator effect which is dose-dependent and lasts over 4-6 hours.

Long-acting inhaled β_2 agonists (salmeterol, formoterol) result in a stronger and sustainable effect, have some anti-inflammatory action, working over 12 hours or more, with a faster onset of action of formoterol.

Inhaled short-acting anticholinergics (ipratropium) is characterized by a dose-dependent effect with a slower onset and longer duration of action than β_2 agonists of short action.

Long-term action anticholinergics (tiotropium) and lasted selectively binds to the M_3 and M_1 –holinoreceptor works over 24 hours or more, causing constant, much stronger effect than ipratropium, has some anti-inflammatory effect, has high safety and well tolerated by patients .

The combination bronchodilator drugs with different mechanisms of action and duration (β_2 agonists and cholinolytics) makes it possible to raise the degree of bronchodilation, get a steady improvement in FEV_1 and reduce hyperventilation lungs than with each bronchodilators alone. This decreases the chance of side effects, tachyphylaxis in long-term treatment compared with the use of high doses of one of bronchodilators.

Long-term use of long-acting bronchodilators (β_2-agonist agents or anticholinergics), or a combined destination depending on the severity of COPD:

- A positive effect on respiratory function (improves bronchial permeability, reduces hyperventilation lungs, optimize the structure of total lung capacity);
- Reduces shortness of breath - the most onerous symptom in patients with COPD;
- Increases physical endurance;
- Improves overall health and quality of life patients;
- Reduces the number of exacerbations and hospitalizations.

Treatment of long-acting bronchodilators is more effective and convenient than short-acting bronchodilators therapy, but something more valuable.

Theophylline are less pronounced bronchodilator effect of potentially toxic metabolism characterized by variable under certain conditions, concomitant diseases and concomitant administration of other medicines.

At the same time, in addition to possible additional bronchodilation, theophylline cause some anti-inflammatory effect, increase the strength of the respiratory muscles, improve COPD patients reduced sensitivity to oxidative stress conditions to corticosteroids.

Thus, *theophylline bronchodilators are the second choice* and can to improve the effectiveness of treatment added to the pre-assigned first choice bronchodilators (β_2 agonists and / or cholinolytics) in severe and very severe COPD, or intended as an alternative to the impossibility of inhalation bronchodilators therapy.

2. Glucocorticosteroids

The role of glucocorticosteroids (GCS) in COPD are much less distinct than in bronchial asthma. In the basic treatment of COPD appointed *inhaled corticosteroids* for specific indications. Oral corticosteroids (ACS) recommended to prescribe only in exacerbations of COPD.

Oral steroids in the short course (2 weeks at a dose of 30 mg) are weak predictors of future long-term action of inhaled corticosteroids purpose and not used for the purpose of trial therapy in patients with III and IV stages of COPD.

Long-term appointment ACS in the basic treatment of COPD is not recommended because of the lack of available benefits, adverse systemic effects and side effects of this therapy (steroid myopathy, muscle weakness, loss of functional capacity, pulmonary insufficiency).

However , *inhaled corticosteroids* are prescribed long-term basic treatment of COPD (patients III , IV stages of disease in FEV_1 <50% adequate, frequent (3 or more in the last three years) exacerbations). This decreases the frequency of severe exacerbations, hospitalizations, improves overall health and quality of life, reduced mortality due to all causes in COPD.

The combination of inhaled corticosteroids and β_2 agonists with prolonged action is more effective than each component separately.

Proposed 6-12 -week trial course with ICS to identify COPD patients who may benefit due to long-term inhalation corticosteroid therapy. However, ICS not intended as monotherapy (central role bronchodilators) do not alter the gradual progression of bronchial obstruction in COPD time.

Scheme pharmacotherapy of COPD patients depending on the stage of disease severity

<table>
<tr><th>And the stage,
easy course</th><th>II stage,
moderate course</th><th>III stage,
severe course</th><th>IV stage
very severe course</th></tr>
<tr><td colspan="4">Avoid risk factors, stop smoking, influenza vaccination short-acting bronchodilators Assign if necessary, on request</td></tr>
<tr><td rowspan="3"></td><td colspan="3">Planning Add 1 or 2 bronchodilators of prolonged action + rehabilitation</td></tr>
<tr><td rowspan="2"></td><td colspan="2">Add inhaled corticosteroids with frequent exacerbations</td></tr>
<tr><td></td><td>Chronic lung

failure (HLN)
add long-term O_2 -
therapy.
Consider
surgery</td></tr>
</table>

In each of the stages of the disease requires patient education about the nature of the disease; risk factors; Diagnostic features; monitoring; opportunities, strategies and tactics of treatment, consequences and prognosis.

Regardless of the stage of the disease to avoid risk factors, stop smoking, make annual flu vaccination.

In *stage I* disease in patients with few in number or intermittent symptoms prescribe inhaled short-acting bronchodilators as needed to control attacks of breathlessness and

coughing. To this end appoint salbutamol or fenoterol or ipratropium or ipratropium + salbutamol or ipratropium + fenoterol. If inhaled bronchodilators are missing or can not use them, you should consider the purpose of prolonged theophylline.

Patients *II , III , IV stages of* disease rates are reduced bronchial obstruction, the symptoms are not controlled by inhaled bronchodilators purpose only short-acting needed. It is recommended to supplement treatment to basis through regular use of one or more of inhaled long-acting bronchodilators. Appointed by drugs of different pharmacological groups (salmeterol or formoterol or tiotropium or salmeterol + tiotropium, formoterol or tiotropium +).

If necessary to achieve a more strict control of symptoms may have an additional purpose long-acting theophylline.

In the absence of the possibility of regular use of inhaled long-acting bronchodilators can be replaced using them (but less effective) inhaled short-acting bronchodilators on a regular basis to basis.

Patients II , III **,** IV stages of COPD receiving regular treatment with inhaled bronchodilators basic short or extended release may also use inhaled short-acting bronchodilators as needed.

Since II stage of the disease is set to rehabilitation patients.

Patients III , IV stages of disease postbronhodilatating FEV_1 <50% of appropriate and history of frequent exacerbations in addition to bronchodilators assigned a basic regular treatment with inhaled glucocorticosteroids (beclomethasone, budesonide, flutikazon, mometasone) in moderate and high doses. You may use the combined dosage forms (X + β_2 agonists long-acting).

Regular, basic treatment of oral, systemic glucocorticosteroids in COPD must be excluded.

In the IV stage of the disease in the presence of chronic pulmonary disease added to the long-term *oxygen therapy.*

In recent years, in order to improve the treatment of patients with COPD conducted research on the possibility of the clinic blockers leykotriyen receptors tumor nekrotic factor protease inhibitors, antioxidants (retinoids) antagonists tahikininu (help reduce hypersecretion of mucus) inhibitors, neuropeptides, substitution therapy α - 1 antytrypsinom, lung transplantation.

3. Other pharmacological treatment:

- *Anti-inflammatory agents steroidal action* (fenspirid) appointed under mild exacerbations and as part of standard treatment within 2-5 months after exacerbation of COPD.
- *Influenza vaccination* can reduce the severity of exacerbation and mortality of patients with COPD. Vaccination is held annually 1 (fall) or 2 (autumn and winter) times a year.
- *A- 1-antitrypsin replacing therapy* can be used in patients younger in case of severe hereditary deficiency α- 1-antitrypsin and verified emphysema. It is not recommended for patients with COPD is not associated with deficiency of α- 1-antitrypsin.
- *Antioxidants:* N -atsetyltsysteyin reduces the frequency of exacerbations. It is recommended in patients with frequent exacerbations, severe history of smoking.
- *Antibiotics are indicated if proven infectious exacerbation of COPD.*

When antibiotic therapy preference should be given antibiotics that are highly active in vitro against major probable pathogens exacerbation of COPD and low (10%) acquired resistance of these pathogens in the population, form a high concentration in the lining of the bronchi and bronchial secretions, as well as proven high clinical efficacy and safety of the results of controlled studies.

When selecting antibacterial therapy should be guided by criteria such as age of the patient, the frequency of exacerbations in the last year, the presence of comorbidity and the level of FEV_1 .

Treatment EL. Treatment of EL is very limited, since almost impossible to influence the morphological changes in the alveoli and small bronchi. In this regard conducted symptomatic

therapy. It aims to combat bronchitis, pneumonia, respiratory symptoms and heart failure. Treatment is based on the following principles:

-eliminate factors that cause the development or progression of chronic bronchitis and EL;

-antibiotic therapy for signs of bronchopulmonary infection; prescribe broad-spectrum, indicative of the course of treatment 7-10 days;

-the use of bronchodilator drugs (methylxanthines short and long-acting, cholinolytics, β_2 agonists);

-EL treatment complications (pulmonary hypertension, chronic pulmonary heart disease, respiratory failure);

-surgical treatment in the presence of huge Boulevard that compress the functional areas of the lungs.

Therapeutic exercise and physical therapy is an important step therapy of EL. Recommend complexes breathing exercises aimed at increasing the mobility of the ribs, spine, diaphragm, chest muscle strength; exercises to develop skills of complete breathing with prolonged exhalation into account provided that the degree of respiratory failure. Shown vibrating chest. Patients EL, especially in the presence of chronic bronchitis during remission spa treatment at resorts with mountain and seaside climate.

Prevention EL. Equally important in preventing the emergence of EL has the correct treatment of acute inflammation in the broncho-pulmonary system, exacerbation of chronic bronchitis, asthma, sanitation inflammatory lesions in the nasopharynx, the elimination of occupational hazards, hardening of the body **.**

Control of initial level of knowledge

" Chronic obstructive pulmonary disease "

1. The most common etiological factors of chronic obstructive bronchitis are all except:

A. Physical.
B. Chemicals.
C. Infectious.
D. allergic.
E. stressful.

2. For the occurrence of exacerbation of chronic obstructive bronchitis matter:

A. Tabacco vape during smoking .
B. pollutants of industrial origin .
C. Regular alcohol consumption .
D. Climatic-weather factors.
E. Infectious factors.

3. The leading symptom of chronic bronchitis with a primary lesion of the mucous membrane of the large bronchi are:

A. Severe dry cough.
B. cough with phlegm.
C. Shortness of breath.
D. Pain in the chest.
E. Sore throat.

4. The clinical manifestation s of chronic obstructive bronchitis are all, except one:

A. coughing.
B. bold small amounts of sputum .
C. shortness of breath.
D. sputum 200 ml.
E. respiratory failure.

5. The main symptom of chronic bronchitis, which flows from the primary lesion of the mucous membrane of small bronchi are:

A. Strong dry cough.
B. cough with phlegm.
C. Shortness of breath.
D. Pain in the chest.
E. edema of the lower extremities.

6. The vital capacity (VC) is all of the above, except one:

A. Capacity inspiration.
B. Reserve about ' cap expiration.
C. Respiratory about ' cap.
D. The remaining about ' cap.
E. Functional residual capacity of the lungs.

7. The most common physical signs in patients with COPD (severe course) are all except:

A. barrel chest.
B. Vesicular breathing.
C. Weakening of respiratory noise.
D. Prolonged expiration.
E. Distant wheezing.

8. expectorants, which also has antiseptic properties are:

A. Grass Thermopsidis.
B. Bromhexine.
C. Mukaltyn.
D. herb thyme.
E. Acetylcysteine

9. In appointing patients with chronic bronchitis antibacterial agents, dose selection and input should be considered:

A. The nature flora tracheobronchial secretions.
B. microflora sensitivity to chemotherapeutic drugs.
C. tolerability of the drug to patients.
D. The concentration of the selected product, you must create a bronchial mucus.
E. All of the above.

10. Chronic obstructive bronchitis treatment the patient should be made:

A. continuously.
B. exacerbation of the disease.
C. exacerbation of the disease and as prevention courses.
D. Only outpatients.
E. Only permanently.

Control of final level of knowledge

1. Dry cough in COPD is caused by:

A. inflammation of the lining of large bronchi.
B. inflammation of the mucous membrane of small bronchi.
C. Hypersensitivity reflex zones of the mucous membrane of large bronchi.
D. hypertrophy of bronchial mucosa.
E. atrophy of the bronchial mucosa.

2. chronic obstructive bronchitis auscultative characterized by the following data:

A. Wet wheezing.
B. The sharp weakening of vesicular breathing .
C. breathing hard and dry whistling wheezing.
D. Amphora breathing .
E. Prolonged inhalation .

3. sputum in COPD are:

A. basophilic leukocytes .
B. The cells of the bronchial epithelium, leucocytes.
C. Macrophages .
D. Atypical OJ and cells .
E. A large number of eosinophils.

4. Complications COPD is all of these, except:
A. hemoptysis.
B. pneumonia.
C. respiratory failure.
D. Spontaneous pneumothorax and thromboembolism.
E. chronic pulmonary heart.

5. indication for corticosteroid therapy in COPD are:
A. Severe clinical disease.
B. The ineffectiveness of treatment by bronchospasmolytic means.
C. Intolerance to bronchospasmolytic means.
D. The presence of III-IV severity.
E. All of the above.

6. The product which improves expectoration of sputum by increasing its turnover (decrease sticking) due to stimulation of lung surfaktant system include:
A. Trypsin.
B. Acetylcysteine.
C. Bromhexine.
D. Mukaltyn.
E. Hedelyks.

7. Which drugs can not combine expectorants:
A. antibiotics.
B. Anti-cough drugs.
C. mucolytics.
D. multivitamin.
E. decongestants.

8. Complications of chronic obstructive bronchitis are:
A. Chronic pulmonary heart.
B. Pulmonary embolism.
C. emphysema.
D. mediastenitis.
E. Spontaneous pneumothorax.

9. What does Tiffno index?:
A. The total lung capacity.
B. A alveolar ventilation.
C. State of bronchial obstruction.
D. The effectiveness of ventilation.
E. Maximum oxygen consumption.

10. Which drugs are mucolytics:
A. alupent .
B. mukaltin .
C. Ephedrine.
D. Codeine.

Situational tasks .

1. Patient L., 32 years. Diagnosis: COPD exacerbation phase. Concomitant diseases - chronic gastritis. Against the background of the therapy were epigastric pain. Which drug you can link?

A. Ambroxol.
B. Loratadine.
C. Bromhexine.
D. Mukaltyn.
E. Libeksyn.

2. Patients '66 complaining of shortness of breath, which increases during exercise. OBJECTIVE: body temperature - 36,6 ° C, BH - 24 for 1 min, pulse - 90 for 1 min, blood pressure - 125/80 mm Hg "Warm" cyanosis, swelling of the neck veins during inhalation and exhalation. Swelling of the lower extremities. Above the lungs auscultated vesicular breathing weakened, scattered dry wheezing. Cardiac relaxed, on top and at Botkin point determined systolic murmur Liver speaks at 5 cm from the edge of the costal arch. Ascites. ECG: R_{v1} = 10 mm , S_{v6} = 11 mm . What pathology could most likely cause of the disease?

A. Polycystic lungs.
B. Asthma.
C. Chronic obstructive bronchitis.
D. Chronic pulmonary artery.
E. Rheumatic aortic insufficiency.

3. During the 8 years of the patient concerned cough in the morning with a discharge of a small amount of sputum, shortness of breath. Smoking more than 10 years. OBJECTIVE: cyanosis, prolongation of expiration, dry wheezing. What is the most likely diagnosis?

A. Chronic obstructive bronchitis.
V. chronic nonobstructive bronchitis.
S. Idiopathic alveolitis.
D. Bronchiectasis.
E. Asthma.

4. The woman, 33 years old, complains of bouts of dry cough, breathlessness. Ill after suffering RH 2 years ago. OBJECTIVE: BH - 16 for 1 min, pulse - 70 for 1 min, blood pressure - 130/90 mm Hg. Above the lungs percussion determined by clear lung sounds, are listened channeled scattered dry wheezing. To determine the reversibility of bronchial obstruction necessary to test:

A. From salbutamol
B. With obzidan
C. Exercise
D . With oxygen
E. From the forced expiratory

5. Patient B., 56 years old, was taken to the clinic with complaints of cough with small amounts of sputum, dyspnea on exertion, raising the temperature to 37.1 C. He considers himself sick about 7 years. Objective: diffusive warm cyanosis of skin and mucous membranes. For percussion over lungs box sound. In auscultation breathing hard, above all surface dry wheezing lungs on inhalation and exhalation. Heart: right on the limit of 1 cm outwards from the midclavicular line. Auscultation Cardiac sonorous, rhythmic. Pulse 76 beats / min. BP 110/70 mmHg Abdomen soft, painless. A blood test clinical Er - 4,7h10 ^{12} / L L - 9,8h10 ^{9} / l, C - 75% A

- 13% M - 6%. ESR - 23 mm / h. ECG: P wave in leads II and III is high, in the transition zone V chest leads. F is L - 65% Tyffno index - 40%. X-ray: increased pulmonary pattern, the expansion of the roots of the lungs. Your diagnosis?

A. Acute bronchitis
B . Chronic pneumonia in the acute stage
C . Chronic obstructive bronchitis
D . The acute focal pneumonia
E . Asthma

6. The concentration of dust in the workplace obrubnyka, 38 years, more than 10 times MDR. The patient complains of cough often dry, sometimes with little phlegm. Cough concerned for 2 - 3 years. According to medical help is not sought. Do not smoke. OBJECTIVE: hard breathing, single dry wheezing. Other organs and systems normal. Radiography of the chest, en general. blood - without deviation from the norm. Respiratory function: slight bronchial patency. What before diagnosis?

A. Chronic non-obstructive bronchitis and art.
B. Particle bronchitis
C. pneumoconiosis of II.
D. Chronic obstructive bronchitis and art.
E. Chronic bronchitis with bronchiectasis

7. Patient D., 25, complained of cough with sputum, general weakness poor appetite, fever that lasts for 2 months. Objectively: skin pale, heart rate - 80 for 1 minute, breathing hard on the lungs, dry and wet wheezing. In the blood: white blood cells, $10x10^{9}$ / L, ESR - 12 mm / h. From what method should start X-ray examination in the hospital?

A. From the X-ray.
B. With radiography
C. With fluoroscopy
D. From imaging
E. From Bronchography

8. The patient E., 30 years complains of cough mainly in the morning, with sputum cough after several aftershocks, shortness of breath during physical activity over the past 3 years. The deterioration occurred after supercooling. In history, pneumonia. OBJECTIVE: breathing hard, with lengthening of expiratory, listen to his background scattered humming wheezing. On chest radiographs - no change. Volume forced expiratory volume in 1 sec. is 70%. What is the clinical diagnosis can be set to the patient?

A. Asthma
B. Chronic obstructive bronchitis
C. Acute bronchitis
D. Chronic non-obstructive bronchitis
E. Bronchiectasis

9. sinker mine experience 10 years, complaining of shortness of breath during physical exertion, cough with little sputum, chest pain. OBJECTIVE: defined moderate cyanosis of the lips. Percussion sound of the lungs with a box shade, weakened vesicular breathing. On radiographs pulmonary drawing amplified, distorted, observed small nodules shadow size 2 - 4 mm in diameter, preferably in the middle and lower parts of the lungs. What additional data are needed to establish the professional nature of the disease?

A. Bronchoscopy
B. Complete blood count
C. ECG
D. Analysis of sputum for the presence of Mycobacterium of tuberculosis

E. Sanitary-hygienic characteristics of working conditions

10. Patient N., 33 years after hypothermia experienced general weakness, fever, cough with phlegm. OBJECTIVE: both halves of the chest symmetrically involved in breathing, percussion over lungs - lung sound auscultation - hard breathing, wheezing wet. From the heart, tones strengthened focus II ton of pulmonary artery. Which most likely diagnosis?

A. Acute bronchitis
B. chronic nonobstructive bronchitis
C. Focal pneumonia
D. Lung Cancer
E. Tuberculosis

Control questions.

1. Define COPD and COPD EL .
2. What morphological changes in COPD.
3. Call risk factors in COPD.
4. Pathogenesis and pathophysiology in COPD.
5. Etiopathogenesis at COPD.
6. Etiopathogenesis at EL.
7. Classification of COPD.
8. Clinical manifestations of COPD.
9. Degree of EL.
10. Physical data in COPD.
11. Name and Describe the phenotypes of patients with COPD.
12. Additional tests in COPD.
13. Even the study of patients with COPD.
14. Diagnosis COPD.
15. Diagnosis EL,
16. Complications COPD.
17. Differential diagnosis of COPD.
18. Treatment of patients with COPD.
19. Exacerbations of COPD and management of patients.
20. Treatment EL.
21. Rehabilitation and Prevention of COPD.

Practical tasks.

1. To provide curation of patients with COPD .
2. Assess the patient's condition and results of physical examination.
3. Fill minutes Supervision COPD patients.
4 . Dates received or interpreting laboratory studies.
5 . Give or interpreting received instrumental methods .
6. treatment.
7. Assign rehabilitation and preventive measures.

Tests answers

" Chronic obstructive pulmonary disease "

Basic knowledge

1.	E	**6.**	D
2.	E	**7.**	B
3.	B	**8.**	D
4.	D	**9.**	E
5.	C	**10**	A

The final level of knowledge

1.	C	**6.**	C
2.	C	**7.**	B
3.	B	**8.**	A
4.	D	**9.**	C
5.	E	**10**	B

Case for cottages

1.	D	**6.**	B
2.	C	**7.**	B
3.	A	**8.**	D
4.	A	**9.**	E
5.	C	**10**	B

RECOMMENDED BOOKS:

1.Clinical Pulmonology - 2016 (The Clinical Medicine Series Book 19).-343h.

2 .Pulmonary Disorders [Sect. 5, Merck manual] 2010.-123p.

3.Pulmonary Pathophysiology : The Essentials by (author) John B. West 2012 .-2(

4.Davidson's Principles and Practice of Medicine 22nd Edition .-Walker, Brian R., FRSE.-2014.-1312p.

Website of the departament: http://www. vnmed3.kharkiv.ua/,

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