Ministry of Health of the Republic of Belarus

Educational Establishment

«Vitebsk State Order of Peoples` Friendship Medical University»

Chair of Propedeutics of Internal Diseases

It predicated on methodical

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The report № \_\_\_\_\_\_

**METHODOLOGICAL GUIDELINES FOR STUDENTS N 11**

for the practical training

on Propaedeutics of Internal Diseases

for specialty 1-79 01 01 "General medicine"

2 course of medical faculty

full-time form of higher education

**THEME:** **Main clinical syndromes in respiratory diseases**

Vitebsk, 2023

**THEME:** Main clinical syndromes in respiratory diseases

**Time:** 3 academic hours.

**Purpose of the lesson**

To familiarize students with the data of subjective and objective methods of examination of patients with the following clinical syndromes accompanying respiratory diseases:

- bronchial patency disorder syndrome (bronchial obstruction).

- syndrome of increased airiness of lung tissue.

- syndrome of pulmonary tissue thickening.

- syndrome of lung cavity formation.

- atelectasis syndrome (obturation and compression).

- syndrome of fluid accumulation in pleural cavity.

- syndrome of air accumulation in the pleural cavity (pneumothorax).

- syndrome of respiratory failure (acute and chronic).

- Chronic pulmonary heart syndrome.

**Objectives of the lesson**

1. Repeat with students the technique of questioning, general examination of the patient, examination and palpation of the chest, percussion, auscultation.

2. To teach the differential diagnosis of clinical syndromes in diseases of the respiratory organs.

**Motivational characteristics of the need to study the topic of the class.**

Syndrome is a symptomcomplex of subjective and objective examinations accompanying various lung diseases. The concepts of "syndrome" and "disease" should be differentiated. A physician of any specialty must master perfectly the technique of making a diagnosis using syndromic diagnosis.

**Questions for classroom knowledge control.**

1. Bronchial permeability disorder syndrome.

2. Syndrome of increased airiness of lung tissue.

3. Syndrome of thickened pleural leaflets.

4. Chronic pulmonary heart syndrome.

5. Pulmonary thickening syndrome.

6. Cavity formation syndrome in the lung.

7. Syndrome of atelectasis (obturation and compression).

8. Syndrome of fluid accumulation in the pleural cavity.

9. Syndrome of air accumulation in the pleural cavity (pneumothorax).

10. Respiratory failure syndrome.

**Information block of the topic**

**Bronchial obstruction syndrome**

It is a sign of obstructive bronchitis, chronic obstructive pulmonary disease, bronchial asthma. In long-term course (development of emphysema) it is combined with signs of syndrome of increased lung airiness.

*Complaints:* dyspnea of expiratory nature, increasing with strain, cough with poorly separated viscous sputum.

Objective pulmonary examination data are more often caused by concomitant syndrome of increased lung airiness.

*General survey*: diffuse cyanosis, forced posture - sitting with hands resting on the edge of the bed, table.

*Static survey of the chest* (syndrome of increased airiness of the lungs): increased volume of the chest, emphysematous type of the chest. Smoothing of intercostal spaces, bulging of supra- and subclavian fossae.

*Dynamic survey of the chest*: participation of the accessory muscles of the thorax in breathing, decreased respiratory mobility of the thorax.

*Chest palpation:* chest stiffness, decreased vocal tremor (syndrome of increased lung airiness).

*Lung percussion:* boxy sound (syndrome of increased airiness of the lungs).

*Lung auscultation:* vesicular breathing with prolonged exhalation, scattered dry whistling rales.

*Bronchophony* is attenuated (syndrome of increased lung airiness).

*X-ray examination of the lungs:* lucidity of the lung fields (syndrome of increased lung airiness).

*Spirography:* decreased forced expiratory volume in the first second (FEF1), Tiffeneau index less than 70%, normal vital capacity (VCL).

**Syndrome of increased lung airiness**

Characteristic of emphysema, obstructive lung diseases (obstructive bronchitis, chronic obstructive pulmonary disease, bronchial asthma). It is caused by loss of elasticity of alveolar tissue, increased amount of air in alveoli. It is combined with symptoms of bronchial obstruction syndrome.

*Complaints:* dyspnea of expiratory or mixed nature, intensifying with exertion.

*General survey:* diffuse cyanosis with violet tint, swelling of cervical veins.

*Static survey of the chest:* barrel-shaped (emphysematous) chest - increased volume, flattening, widening of intercostal spaces, bulging of supra- and subclavian fossae.

*Dynamic survey of the chest:* involvement of auxiliary muscles of the thorax in breathing, decrease in respiratory mobility of the thorax.

*Chest palpation:* chest stiffness, decreased vocal tremor.

*Lung percussion:* boxy sound.

*Lung auscultation:* weakened vesicular breathing. Bronchophony is attenuated. As a sign of bronchial obstruction, vesicular breathing with prolonged exhalation, scattered whistling rales.

*X-ray examination of the lungs:* luminescence of the lung field.

*Spirography:* mixed type of pulmonary ventilation function disorders, when restriction is combined with obstruction - reduced vital capacity (VCL), decreased forced expiratory volume in the first second (FEF1), Tiffeneau index can be equal or over 70%.

*Pulmonary thickening syndrome*

Pulmonary tissue thickening occurs during inflammatory process in the lungs (pneumonia), saturation of alveoli with blood (infarct-pneumonia in pulmonary artery thromboembolism), replacement of alveolar tissue with connective tissue elements (pneumosclerosis). Clinical signs of pulmonary tissue thickening syndrome are caused by disconnection of the lung area from breathing. The severity of symptoms depends on the volume of the non-functioning lung.

*Complaints:* dyspnea of mixed nature, intensifying with exertion.

*General survey:* diffuse cyanosis.

*Static survey of the chest*: reduction of the affected half of the chest.

*Dynamic survey of the chest*: lagging of the affected half of the thorax when breathing.

*Chest palpation:* decreased elasticity, increased vocal trembling in the affected half of the chest.

*Lung percussion:* dull or dull sound over the affected part of the lung.

*Lung auscultation:* weakening of vesicular breathing or its replacement by pathological infiltrative bronchial breathing over the affected part of the lung, crepitation, moist fine, medium, large bubble rales in a limited area over the affected lung. Bronchophony over the thickened lung is intensified.

*X-ray examination of the lungs:* shading of the lung area with indistinct contours of different sizes depending on the volume of the affected lung area.

*Spirography:* Restrictive impairment of lung ventilation function - decreased vital capacity (VCL) with normal forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

**Lung atelectasis syndrome**

Lung atelectasis is the collapse of the lung or part of it. Compression atelectasis develops as a result of lung compression from the outside (fluid, air in the pleural cavity); obstructive atelectasis - as a result of cessation of air flow due to bronchial obstruction (tumor, foreign body). Clinical signs of atelectasis syndrome are caused by disconnection of a lung part from breathing. The severity of symptoms depends on the volume of the non-functioning lung.

In compression atelectasis, the lung may be compressed by fluid located in the rib-diaphragmatic sinus. The lower lateral parts of the lung are compressed towards the root.

If the lung is compressed by air getting into the pleural cavity, the upper lateral parts or the whole lung are collapsed towards the root.

There are signs of atelectasis syndrome in the projection of the area of the collapsed lung. Changes in other parts of the chest are determined by air or fluid in pleural cavity.

*Complaints*: dyspnea of mixed character, intensifying on exertion.

*General survey:* diffuse cyanosis.

Thoracic examination findings depend on the type of atelectasis.

**Compression atelectasis.**

*Static survey of the chest:* half of the chest may be enlarged due to fluid or air in the pleural cavity.

*Dynamic thoracic examination:* lagging of the affected half of the thorax while breathing.

*Chest palpation:* decreased elasticity, increased vocal trembling above the exudate level or at the root zone in case of pneumothorax;

*Lung percussion:* dull pulmonary sound or blunt sound over the collapsed part of the lung.

*Lung auscultation:* weakening of vesicular breathing, appearance of pathological bronchial breathing above the level of exudate or in the root zone - with pneumothorax.

Bronchophony is increased above the level of exudate or in the root zone - with pneumothorax.

*X-ray examination of the lungs:* shading in the projection of the collapsed lung, absence of lung pattern in the lateral parts of the chest - for pneumothorax, fluid level - for pleurisy.

*Spirography:* restrictive ventilatory function disorder - decreased vital capacity of lungs (VCL) with normal volume of forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

**Obturation atelectasis.**

*Static chest examination*: in obturative atelectasis, reduction of the affected half of the thorax.

*Dynamic chest examination*: lagging of the affected half of the thorax when breathing.

*Chest palpation*: decreased elasticity, weakened vocal tremor in the affected half of the chest.

*Lung percussion*: muffled lung sound or blunt sound over the collapsed part of the lung.

*Lung auscultation*: no breath sounds over the collapsed portion of the lung. No bronchophony.

*X-ray examination of the lungs:* shading of the lung, absence of lung pattern.

*Spirography:* Restrictive ventilatory function disorder - decreased vital capacity (VCL) with normal forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

**Syndrome of fluid accumulation in the pleural cavity (hydrothorax)**

Pleural effusion, hydrothorax is an excessive accumulation of fluid in the pleural cavity. Transudate is a consequence of violation of interaction between hydrostatic and oncotic blood pressure in capillaries (congestive heart failure, hypoalbuminemia in nephrotic syndrome, liver failure) or lymphatic outflow obstruction. Exudate is the result of violation of the permeability of vessels and pleura itself (inflammatory, tumorous affection of pleura - pleurisy). The causes of pleuritis - bacterial and viral infection, tuberculosis, pulmonary infarction, systemic connective tissue diseases (systemic lupus erythematosus, rheumatoid arthritis), subdiaphragmatic abscess, acute pancreatitis. Pleural empyema - purulent pleurisy.

Occurrence of ventilation disorders and objective signs in pleural effusion are associated with compression of the adjacent part of the lung. Signs of the syndrome of fluid accumulation in pleural cavity are combined with signs of lung collapse (atelectasis syndrome) (Fig. 1.1).

*Complaints:* dyspnea of mixed nature, intensifying with exertion, heaviness, chest pain, intensifying with deep breathing, dry cough.

*General examination:* diffuse cyanosis.

*Static examination of the chest:* enlargement of the affected half of the chest. Smoothing of intercostal spaces.

*Dynamic chest examination:* lagging of the affected half of the thorax when breathing.

*Chest palpation:* decreased elasticity, weakened vocal tremor in the affected half of the chest (in the fluid projection - over the lower lateral parts), above - increased vocal tremor over the collapsed part of the lung (area of atelectasis).

*Lung percussion:* blunt sound over the fluid with the highest level of elevation of the lower edge of the lung along the axillary lines, above - blunt tympanic sound over the collapsed part of the lung (atelectasis area).

*Lung auscultation:* weakened or absent vesicular breathing in exudate projection (above the lower lateral parts), bronchial breathing (compression-atelectatic) - above, in the area of lung atelectasis. Bronchophony is weakened or absent in fluid projection.

*X-ray examination of the lungs:* homogeneous shadowing of the rib-diaphragmatic angle with a concave oblique upper border (pleurisy). Displacement of mediastinum to the healthy side.

*Spirography:* impaired pulmonary ventilation function of restrictive type - decreased vital capacity (VCL) with normal forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

Analysis of pleural contents:

Transudate - consistency liquid, low specific gravity (1.008-1.015), protein content less than 3% (30 g/l), Rivalt's test negative, leukocytes in small numbers (up to 15-20 in the field of view, <1-109/l), (transudate of cardiac and renal origin);

Exudate - consistency of pleural fluid is semi-liquid or thick, specific gravity more than 1.015, protein content above 3% (30 g/l), Rivalt's test positive, leukocytes in large numbers (>1-109/l).

**Syndrome of air accumulation in the pleural cavity (pneumothorax)**

Pneumothorax is a pathological accumulation of gas in the pleural cavity, leading to collapse of the lung (spontaneous, secondary to bronchopulmonary diseases, trauma, artificial ventilation).

Occurrence of ventilation disorders and objective signs of pneumothorax with lung collapse and mediastinum displacement to the opposite side. Signs of air accumulation syndrome in pleural cavity are combined with signs of lung collapse (atelectasis syndrome).

*Complaints:* suddenly appeared dyspnea of mixed character, intensifying with exertion, pain in the chest, intensifying with breathing, dry cough.

*General examination:* diffuse cyanosis.

*Static examination of the chest:* enlargement of the affected half of the chest. Smoothing of intercostal spaces.

*Dynamic chest examination:* lagging of the affected half of the thorax when breathing.

*Chest palpation*: decreased elasticity, no vocal trembling in the affected half of the chest.

*Lung percussion:* tympanic sound in the affected half of the chest.

*Lung auscultation:* weakened or absent vesicular breathing. No bronchophony.

*X-ray examination of the lungs:* homogeneous luminescence of the lung field, closer to the root - thickened shadow of the compressed lung.

*Spirography:* Restrictive ventilatory function disorder - decreased vital capacity of the lungs (VCL) with normal (SPH1), Tiffeneau index >70%.

**Syndrome of thickening pleural membranes**

Characteristic of dry pleurisy.

*Complaints:* dyspnea of mixed character, intensifying on exertion, pain in the affected part of the chest, intensifying with deep breathing, dry cough.

*General examination:* forced position on the painful side to reduce pain.

*Static examination of the chest:* the affected half of the chest may be reduced.

*Dynamic thoracic examination:* lagging of the affected half of the thorax when breathing.

*Chest palpation:* decreased elasticity, soreness, voice tremor, weakened in the affected half of the chest.

*Lung percussion:* muffled pulmonary sound over the affected area.

*Lung auscultation:* weakened vesicular breathing, pleural friction noise in a limited area. Bronchophony is attenuated over the affected area.

*X-ray examination of the lungs*: restricted movement of the diaphragm on the diseased side.

*Spirography*: normal or restrictive ventilatory function disorder - decreased vital capacity (VCL) with normal forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

**Lung cavity syndrome**

Cavities are most often formed in the place of an already existing infiltrate (lung gangrene, abscess, tuberculosis). Because of this, signs characteristic of lung thickening syndrome are present.

Identification of signs of a cavity in the lung is possible if the cavity meets certain characteristics: diameter at least 4 cm, location close to the chest wall, communication with the bronchus, contains air and has smooth walls. In other cases cavities may not be revealed by objective examination, but only by X-ray or CT scanning of the lungs.

*Complaints:* dyspnea of mixed character, intensifying at loading, at first dry, then productive cough with considerable quantity of mucopurulent, hemorrhagic sputum (sometimes sputum goes "full mouth").

*General examination:* diffuse cyanosis.

*Static examination of the chest*: reduction of the affected half of the chest.

*Dynamic chest examination:* lagging of the affected half of the thorax when breathing.

*Chest palpation:* decreased elasticity, increased (with a large surrounding infiltrate) or decreased (with a large air-containing cavity) vocal tremor in the affected half of the chest.

*Lung percussion*: blunted and tampanic sound, with metallic tinge (smooth-walled cavity), "cracked pot" sound over the cavity communicating with the bronchus.

*Lung auscultation:* weakening of vesicular breathing, appearance of pathological cavity bronchial breathing, moist medium- and large-bellied rales in a limited area above the affected lung. Bronchophony is intensified (with large surrounding infiltrate).

*X-ray examination of the lungs*: oval or rounded cavity, possibly with horizontal level of uniform shading (liquid content), better defined in lateral view.

Spirography: Restrictive impairment of lung ventilation function - decreased vital capacity (VCL) with normal forced expiratory volume in the first second (FEF1), Tiffeneau index >70%.

**Syndrome of respiratory insufficiency**

*Definition: Respiratory failure (insufficiency****)*** is a condition in which normal oxygenation of blood flowing through the lungs is not achieved, and adequate excretion of carbon dioxide from the body is not provided.

***Etiology.***It is distinguished primary *(pulmogenic) and secondary (non- pulmogenic) respiratory failure*.

*Primary (pulmogenic) respiratory failure* occurs in the diseases of the respiratory apparatus (lungs, airways, pulmonary circulation, respiratory muscles and chest).

*Secondary (non-pulmonogenic) respiratory failure* occurs in diseases of the organs and systems that are not included in anatomical and physiological complex of an external respiratory apparatus (with the damage to the brain or the spinal cord, renal and hepatic insufficiency, sepsis, peritonitis).

The *respiratory failure is developed* *most often due to defeat of bronchi and respiratory apparatus of the lungs, deformation of chest, the defeat of the respiratory muscles and circulatory disorders in the pulmonary circulation.*

***Classification of Respiratory Insufficiency***

1. Etiology: (a) Primary - due to the respiratory diseases; (b) Secondary - due to other pathology (chest trauma, kyphoscoliosis, intoxica- tions, etc.). 2. Course - (a) Acute, (b) Chronic. 3. Type of external respiration dysfunction: (a) restrictive, (b) оbstructive, (c) мixed. 4. Degree of respiratory insufficiency:

I (compensated), II (subcompensated), III (decompensated).

5. Stage: (a) latent pulmonary, (b) pronounced pulmonary, (c) cardiopulmonary insufficiency.

**Clinical and pathogenetic forms of respiratory failure:** *1*. *Obstructive respiratory failure. 2. Restrictive respiratory failure. 3. Mixed respiratory failure*.

*Obstructive respiratory failure* (obstruction) is due to narrowing the airways (in acute obstructive bronchitis, chronic obstructive pulmonary disease, bronchial asthma).

*Restrictive respiratory failure* (restriction) is associated with a decrease in the respiratory surface of the lung due to changes in the alveolar tissue (in pneumonia, atelectasis, lung destruction, and pneumosclerosis).

*Mixed respiratory failure* is most often the result of a combination of several of the above forms.

***Obstructive type*** of respiratory failure is characterized by difficulty in passing the air through the bronchi. It is observed in patients with acute obstructive bronchitis, bronchiolitis, COPD, with an attack of bronchial asthma.

*Clinical picture* *includes* *bronchial obstruction syndrome and the syndrome of increased airiness of the lung tissue (pulmonary emphysema).*

Inspection of the patient shows barrel-chest, involvement of accessory muscles, expiratory dyspnea. Chest palpation may reveal decreased tactile fremitus, and increased rigidity of chest may occur. Percussion may detect a hyper-resonant sound (in case of emphysema) and widening the Kroenig’s fields, increasing height of the lung apexes, and lowering inferior borders of the lungs.

Harsh vesicular breathing with prolonged expiration and wheezes are auscultated. Bronchophony is weakened.

*The X-ray picture of the lungs* shows an increased bronchovascular pattern or/and an increased translucency of the lung.

*Spirography*: marked decrease in expiratory forced vital capacity of the lungs (FVC), forced expiratory volume per second (FEV1), FEV1<70% of FVC; and a slight decrease in the VC. Peak flowmetrydetects diminished PEF (peak expiratory flow rate) <80% of normal PEF.

***Restrictive type of respiratory failure***is observed with the restriction of the pulmonary tissue to expansion (in pneumosclerosis, pleural adhesions, kyphoscoliosis), decrease in the respiratory surface of the lungs (pneumonia, exudative pleurisy, hydrothorax, pneumothorax).

*Clinical picture* *may include such syndromes as focal consolidation of the pulmonary tissue, atelectasis, thickening of pleural membranes, accumulation of the air and fluid in the pleural cavity.* Inspection of the patient shows the asymmetric chest, a decrease of the respiratory mobility of the chest, inspiratory or mixed dyspnea. Diminished vesicular breathing or pathologic bronchial respiration, fine crackles, pleural rub may be heard.

*Spirography* shows a marked decrease of the vital capacity of the lungs (VC); a slight decrease in the FVC may occur; forced expiratory volume per second FEV1>75% of FVC. Peak flow metry indexes are normal.

***Mixed, or combined, type of respiratory insufficiency*** includes the signs of the two previous disorders, often with prevalence of one of them; this type of disorder occurs in long-term diseases of lungs and heart.

**Stages of respiratory insufficiency** in chronic diseases of lungs reflect the changes occurring during the progress of the disease. Stages of *latent pulmonary, pronounced pulmonary, and cardiopulmonary insufficiency* are normally differentiated.

**Three degrees of respiratory insufficiency** are also distinguished. The degrees of respiratory insufficiency reflect the gravity of the disease at a given moment.

*I degree* – dyspnea when effort is available earlier, cyanosis is absent, light hypoxemia presents. In arterial blood HbО2 (oxyhemoglobin) – 80–96%; РаО2 (partial pressure of oxygen) – 100–70 mm Hg; РаСО2 (partial pressure of carbon dioxide) – 40–50 mm Hg. VC is normal or ≥ 70 % of the predicted VC.

MVL ≥ 60% of the predicted MVL.

*II degree* - dyspnea at usual load, obvious cyanosis, moderate hypoxemia.

In arterial blood HbО2 - 80–60%; РаО2 – 70–50 mm Hg; РаСО2 – 50 mm Hg.

VC is ≥ 50 % of the predicted VC. MVL ≥ 40% of the predicted MVL.

*III degree* - dyspnea at rest, pronounced cyanosis, severe hypoxemia. In arterial blood HbО2 < 60%; РаО2 < 50 mm Hg; РаСО2 > 70 mm Hg. VC is < 50 % of the predicted VC. MVL < 40 % of the predicted MVL.

**Syndrome of pulmonary heart (Cor Pulmonale)**

*Definition:* ***Сor Pulmonale (Pulmonary heart,*** I26-J28 according to IСD-X) is a syndrome of the right ventricular hypertrophy and dilatation secondary to the diseases of the lungs that produces pulmonary artery hypertension.

*Etiology and pathogenesis:* Cor pulmonale is usually caused by COPD (chronic bronchitis, emphysema), extensive loss of the lung tissue from surgery or trauma, etc.; pulmonary emboli; acute pneumonia, other acute respiratory infections; primary pulmonary hypertension; pulmonary venoocclusive disease, scleroderma, diseases leading to diffuse pneumosclerosis (or pneumofibrosis); kyphoscoliosis and others types of the pathologic chest; obesity with alveolar hypoventilation; neuromuscular diseases involving respiratory muscles, and idiopathic alveolar hypoventilation.

Cor pulmonale does not refer to a congenital heart disease, or acquired valvular heart disease. It is usually chronic but may be acute and reversible.

Cor pulmonale is directly caused by alterations in the pulmonary circulation that lead to pulmonary arterial hypertension, thereby, increasing the mechanical load on RV emptying (after load). However, the most important mechanism leading to pulmonary hypertension is alveolar hypoxia, which results from localized inadequate ventilation of well-perfused alveoli or from a generalized decrease in the alveolar ventilation. Alveolar hypoxia is a potent stimulus of pulmonary vasoconstriction. Hypercapnic acidosis augments the pulmonary vasoconstriction. During chronic hypoxia, pulmonary hypertension may be intensified by increased blood viscosity arising from secondary erythrocytosis.

*Clinical picture*

Pulmonary heart is asymptomatic at an early stage, although patients usually have pronounced manifestations of the underlying lung disease (e.g., dyspnea, productive cough, and fatigue during the exercise). Dyspnea limits the patient's ability in the minor stresses of daily living. There is frequently a history of emergency hospital admissions because of respiratory infection, sometimes necessitating mechanical ventilation. Hypoxia due to hypoventilation is usually worse at night. Exertional dyspnea is the most common symptom of pulmonary hypertension. Some patients suffer syncope or fatigue on exertion, and substernal anginal pain is common.

*Survey* of the patient reveals cyanosis (warm), distended jugular veins, enlargement of abdomen (due to hepatomegaly and ascites), edema, and positive venous pulse may occur.

*Heart palpation* detects cardiac impulse and epigastric pulsation of the right ventricle.

*Percussion* may reveal widening relative heart dullness to the right (emphysema can mask it).

At *auscultation* accent of II sound above pulmonary artery; systolic murmur of tricuspid insufficiency, diastolic murmur of pulmonary artery insufficiency, and gallop rhythm may be heard.

*X-ray examination* discovers right ventricle and proximal pulmonary artery enlargement with distal arterial attenuation, signs of emphysema.

*ECG:* evidence of right ventricle and right atrium hypertrophy - dextrogram, “P pulmonale” – high (>2,5 mm) acute Р in II, III, AVF and right chest leads V1-2; high R wave ≥ 7 mm appears in V1-2, deep S wave in V4-6).

*Echocardiography* may detect the signs of right ventricle enlargement and dysfunction, and pulmonary artery hypertension. Diagnosis of pulmonary hypertension may require right heart catheterization.

*Diagnosis of pulmonary heart* is based on detection of right ventricle hypertrophy secondary to the diseases of the lungs.

**Literature:**

1. Ivashkin V.T., Okhlobystin A.V. Internal diseases propedeutics: Textbook. – Moskow: Geotar-Media, 2006. – 176 p.
2. Немцов, Л.М. Special propedeutics of internal diseases: Lecture course (Частная пропедевтика внутренних болезней: Курс лекций (на английском языке)/ Л.М. Немцов. – Витебск: ВГМУ, 2016. – 318 с.
3. Немцов, Л.М. Пропедевтика внутренних болезней, Часть I = Propaedeutics of internal diseases, Part I: учебн.–метод. пособие (на английском языке) / Л.М. Немцов. – Витебск : ВГМУ, 2020. – 297 с.
4. Пронько, Т.П. Диагностика основных заболеваний внутренних органов = Diagnostics of main internal diseases : курс лекций для студентов фак. иностр. учащихся, обучающихся на англ. яз. / Т.П. Пронько, А.В. Пырочкин. - Гродно : ГрГМУ, 2013. - 243 с.

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