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**INTERNAL DISEASES PROPEDEUTICS**

**PART I**

DIAGNOSTICS OF PULMONARY DISEASES

Textbook of Medicine for medicine faculty students

**Ulyanovsk, 2016**

**Reviewers:**

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This publication is the first part of "Internal diseases propedeutics", which main goal is the practical assistance for students in the development of the fundamentals of clinical diagnosis of diseases of the respiratory system. It contains a description of the main methods of laboratory and instrumental diagnostic tests of diseases of the respiratory system. The publication is illustrated with charts, drawings and tables. The textbook is intended for students of medical universities.

**THE CONTENTS OF A TEXT BOOK**

**Questioning and examination of patients with diseases of the lungs.**

**Main complains of patients with diseases of the lungs.**

Shortness of breath, breathlessness (dyspnoe): patients subjective feeling of lack of air; causes: stenosis of the larynx or (and) trachea, bronchial asthma, chronic obstructive pulmonary disease, lung cancer (obstruction of the air passage in the respiratory tract), pneumonia, tuberculosis, pulmonary infarction, pulmonary fibrosis, obstructive pulmonary atelectasis, cardiac asthma (decreases the airiness of the lungs), pneumothorax, hydrothorax (a decrease in respiratory surface of the lung due to the accumulation of air or fluid in the pleural cavity), pulmonary and respiratory failure. Types of dyspnea: inspiratory (inhale is difficult), expiratory (difficulty exhaling), mixed, physiological and pathological.

Cough (tussis) - definition: a reflex act when a cluster of airway pathologic discharge or ingress of foreign bodies; laryngitis, pleuritis, tracheitis, pneumonia; causes: chronic obstructive pulmonary disease, bronchial asthma, multiple bronchiectasis, lung abscess, tuberculosis, tumors.

 Types of cough: cough without sputum, cough with sputum expectoration, morning cough (bronchitis, bronchiectasis, lung abscess, and cavernous tuberculosis of the lungs), night (tuberculosis, lymphogranulomatosis, malignant neoplasm), evening (sometimes after pneumonia), persistent and periodic cough, barking (whooping cough, compression of the trachea by a goiter or tumor, lesions of the larynx with swelling of the vocal cords), cough with sputum "full mouth" (abscess of the lungs), cough with release of large quantities of sputum (abscess and bronchiectasis).

**Sputum**

Information should be obtained about its quantity, colour (white, grey, black, pink, yellow or green), viscosity (serous or tacky), taste and odour (Table 1).

**Table 1.** Characteristics of sputum

| Sputum | Condition |
| --- | --- |
| Mucoid, excessive quantities | Chronic bronchitis |
| Mucopurulent or purulent (yellow or green) | Infection - acute or chronic bronchitis |
| Excessive in early mornings, or at change of posture, purulent | Bronchiectasia |
| Black | Cigarette or atmospheric smoke, coal-miner's sputum |
| Pink, frothy | Acute pulmonary oedema |
| Rusty | Lobar pneumonia |
| Blood-stained | Acute bronchitis, tuberculosis, neoplasia |
| Viscous with plugs | Asthmatic pulmonary eosinophilia |

A change of colour from white to green or yellow suggests the onset of infections in patients with chronic bronchitis. A pink and frothy sputum associated with breathlessness is commonly encountered in pulmonary oedema.

Hemoptysis (haemoptoe) – definition: the allocation of blood with sputum during cough; causes: viral pneumonia, abscess and gangrene of lungs, bronchiectasis, tuberculosis, lung cancer, actinomycosis and ascariasis, mitral stenosis, mitral insufficiency, cardiac asthma, pulmonary embolism, infarction of the lungs. Features of hemoptysis in certain diseases: scarlet blood (tuberculosis, Central bronchogenic cancer, bronchiectasis, ascariasis and actinomycosis of the lung, pulmonary infarction in the first 2-3 days; "rusty" sputum (pneumonia in stage 2).

Chest pain characteristics: location, nature, intensity, duration, irradiation, connection with the act of breathing, cough and body positions. Causes of pain in the chest, and in certain diseases: superficial pain in the chest wall, worse on breathing, coughing, sudden movements of the trunk, or in a position on the sick side (trauma, erysipelas, herpes zoster, myalgia, myositis, thoracic osteochondrosis with radicular syndrome, fractures and periostitis of the ribs, the tumor metastasis to the ribs and the pleura); in lesions of the lung and pleura (dry pleurisy, pneumonia, abscess, tuberculosis, infarction, metastasis of tumors to the pleura or the tumor of the pleura, traumatic or spontaneous pneumothorax); other disorders (subphrenic abscess, acute pancreatitis).

Risk factors of respiratory diseases: hypothermia, Smoking, atmospheric and occupational pollutants, pathology of internal organs, burdened heredity (asthma, Kartagener disease, cystic fibrosis, Oncology, primary emphysema, etc.), trauma, aspiration, contact with tuberculosis patients, reducing the reactivity of the immune system, the season of epidemics of viral or other infection.

**General inspection**

On general examination, there may be clues to the underlying disease:

Cachexia may occur in malignant disease, and in severe chronic lung disease, including fibrosis, infection and emphysema. Cachexia may occur in a number of severe disorders, including chronic lung disease such as pulmonary fibrosis, tuberculosis and emphysema, malignant disease, including bronchial carcinoma, and systemic infection, especially with HIV («slim disease»). Note the obvious signs ofweight loss, fever, with widespread muscle and soft-tissues wasting.

Cyanosis - best seen in the lips, tongue, buccal mucosa and fingers - indicates significant desatura-tion of circulating haemoglobin. Cyanosis is a fundamental sign of cardiorespiratory disorders and suggests capillary oxygen desaturation of 85% or lower.

A herpetic eruption on and around the lips is sometimes seen in a patient with a respiratory infection.

**Coal dust tattoos** may be seen on the face, though these are more often seen on the arms, as an occupational legacy in a patient with pulmonary fibrosis.

Nasal polyps frequently occur in patients with an atopic background and in those with cystic fibrosis.

Eczema is often found in conjugation with hay fever and asthma.

The oscillations of the jugular venous pulse are difficult to interpret in patients with chronic airways obstruction who generate a high intrathoracic pressure to drive the air out through the narrowed bronchi. However, static engorgement of the neck veins is an important sign of obstruction of the superior vena cava, usually caused by mediastinal malignancy.

«Nicotine» stained fingers occur in heavy smokers, and typical pigmented scars may occur in coal miners; in association with finger clubbing both signs have an ominous significance, suggesting underlying bronchial carcinoma, pulmonary fibrosis, bronchiectasia or chronic sepsis.

Finger clubbing is frequently present in a number of conditions, especially bronchial carcino ma (occasionally with a pleural fibrinoma), and in those with chronic purulent conditions such as bronchiectasia, lung abscess and empyema (Application. Figure 1)

**Examination of the chest.**

Patients having respiratory system diseases may present the following problems: chest pain, cough, dyspnea, asphyxiation. Pains caused by respiratory apparatus lesion depend on pulmonary pleura involvement. If the process is confined to lungs only, no pain can be registered since the lung tissue has no pain receptors. Thus, pain can accompany any lung process provided it spread as far as pleura. Pleural pains are characterized by the following features: they are of shooting character, not of radiating nature, and are usually aggravated or detected only at the maximum of inhale or while coughing and sneezing, that is, when pleural leaves overlap. It is important to specially note some specific character of pains arising due to diaphragmatic pleurisy. They are peculiar in having ability to spread to jugular region via phrenic nerve. On the other hand, these pains radiate to abdominal cavity and can

be mistaken for abdominal diseases. Pleural pains should be distinguished from other kinds that can arise in the thorax region: caused by thorax diseases: intercostal muscles myosites, intercostal nerves pleurisy and nerve root compression (osteochondrosis), rib injuries (fractures, fissures, etc.); pleural pains, cardiac and vascular pains (angina pectoris, myocardial infarction, aortitis, etc.); reflex pains (cholecystitis, diaphragmatic hernia, ulcer, appendicitis). The second characteristic complaint is cough which categorizes according to pathogenesis: pulmonary, reflex, central; according to duration: permanent, occasional; according to timbre: barking, hoarse, noiseless, etc.; according to character: dry, productive (nature, smell, amount, period of expectoration). The third characteristic patient complaint is dyspnea. It categorizes: depending on breathing stages: inspiratory, expiratory, mixed; accordingto pathogenesis: pulmonary, cardiac, anemic, etc. Asthma is an attack-like abrupt dyspnea. It occurs not only with lung diseases (bronchial asthma), but also with a number of other diseased states: bronchial, cardiac, mixed, cerebral, and hysteric types.

On static examination, thorax shape characteristics are described. There exist three normal types of thorax: asthenic chest, normosthenic (athletic) type, and hypersthenic chest. Its pathologic shapes are paralytic chest, emphysematous (barrel) chest, rickets breast, funnel breast, kyphoscoliotic chest. It is necessary to explain here the notions of scoliosis, lordosis, and kyphosis. On static examination of thorax, there can also be detected distortion in terms of restriction or enlargement of one side. Examples can be given of the restriction of one side of the chest when having pulmonary fibrosis, and enlargement with exudative pleurisy. Dynamic inspection allows to evaluate the extent of thorax share in the breathing process, lagging of one side, etc. It also allows to characterize breathing process. Palpation of chest determines: chest elasticity or resistance, tenderness areas, vocal or tactile fremitus (fremitus pectoralis).

Normal shape of the chest: normosthenic (truncated cone shape, transverse size is larger than the Antero-posterior, supra - and infraclavicular fossa are moderately, clearly defines the angle of Louis, the epigastric angle is sharp , the edges are

conisholme direction, the blades are tightly adjacent to the body, thoracic abdominal equal; hypersthenic (cylinder in shape, the transverse dimension close to the Antero-posterior, supra - and infraclavicular fossa are virtually absent, significantly pronounced angle of Louis, the epigastric angle is obtuse , the ribs are horizontal, the intercostal spaces are reduced, the vanes merge with the trunk, thoracic abdominal less; asthenic (long and narrow in shape, reduced transverse and anteroposterior dimensions, above and subclavial fossa pronounced, the angle of Louis is missing, the epigastric angle is sharp, the edges are close to the vertical direction and "wing" the scapula, thoracic and more abdominal.

Pathological shapes of the chest: emphysematous (with emphysema: barrel shape, expansion of intercostal spaces, clearly defined the angle of Louis, the epigastric angle is obtuse, the ribs are almost horizontal direction, the breath is actively involved auxiliary respiratory muscles (sternokleidomastoid, trapezius, etc. with indrawing of the intercostal spaces), lungs are in the phase of constant inhalation and exhalation is much difficult), paralytic (for a total asthenia, disease Marfan's tuberculosis: symptoms asthenic chest + atrophy of the muscles of the chest, an asymmetric location of the clavicle, and the unequal retraction of the supraclavicular fossae, shoulder blades at different levels and meshayutsya when breathing asynchronously), rachitic (synonym-keel: increased anteroposterior size due to the keeled sternum, "rachitic rosary" in the transition region from the rib cartilage into bone) (Application pic. 2.), funnel (due to a congenital anomaly of the sternum: the chest has a funnel-shaped indentation in the lower 1/3 of the sternum) (Application, pic. 3), navicular (in syringomyelia: thoracic has indentation in the middle to upper parts of the sternum).

Causes and types of spinal deformities: trauma, tuberculosis of the spine, ankylosing spondylitis, etc. Scoliosis (curvature in the lateral direction), kyphosis (backward curvature), lordosis (forward curvature), kyphoscoliosis – combination of scoliosis and kyphosis. Kyphosis results in anterior concavity of thoracic spine and thereby leads to shortening of the chest. Kyphosis is frequently seen in erdely people with osteoporosis, chronic obstructive airways disease.

The cause of reduced 1/2 chest: pleural adhesions, pulmonary fibrosis, lung carnification, pulmonary infarction,lung abscess, tuberculosis, pneumonectomy or lobectomy, obstructive atelectasis.

The reasons for the increase of 1/2 chest: fluid in the pleural cavity, a pneumothorax (the flattening and bulging of the intercostal spaces, asymmetry of the clavicles and the shoulder blades, lag 1/2 of the chest during breathing).

Physiological types of breathing: thoracic (mostly in men), abdominal (more common in women) and mixed. The breathing rate should be counted when the patient is not conscious of it it can be done during the earlier part of the inspection/ The normal rate is between 14 and 18 breaths a minute. In opiate or barbiturate poisoning this may fall to below eight breaths a minute (bradypnoe) whereas in acute bronchopneumonia the rate may exceed 40 a minute (tahypnoe). The relationship between inspiration and expiration should be determined. Normally, the inspiration is active and longer whereas expiration is shorter and accomplished by the passive recoil of the lungs. In small airways obstruction the expiration becomes active and prolonged, due to a greater pressure gradient from small to major airways.

The deep inspiration and shorter expiration which follows immediately gives the respiration its normal **rhythm. Shallow breathing** with short inspiration and expiration occurs either when breathing is restricted (e.g. obesity, pulmonary fibrosis) or is painful as in chest wall disease and pleurisy, or in anxiety states. **Kussmaul breathing** with deep inspiration and expiration typically occurs in metabolic acidosis (e.g. diabetic ketoac-idosis, renal failure, methyl alcohol poisoning, etc.). **Cheyne-Stokes breathing** comprises periods of apnea alternating with a gradual resumption of respiration with increasing depth which then declines to another period of cessation of breathing. This pattern of breathing is also termed periodic or cyclical breathing and occurs in advanced cardiac and respiratory failure, narcotic drug poisoning and in cerebrovascular disease (Fig. 1). **Pursed lip breathing** is a sign of severe small airways obstruction, as can be found in asthma and emphysema, but it also occurs occasionally in left heart failure. It is an attempt by the patient to create

an effective pressure gradient, by narrowing the outlet, to drive the air during expiration through the diseased airways.
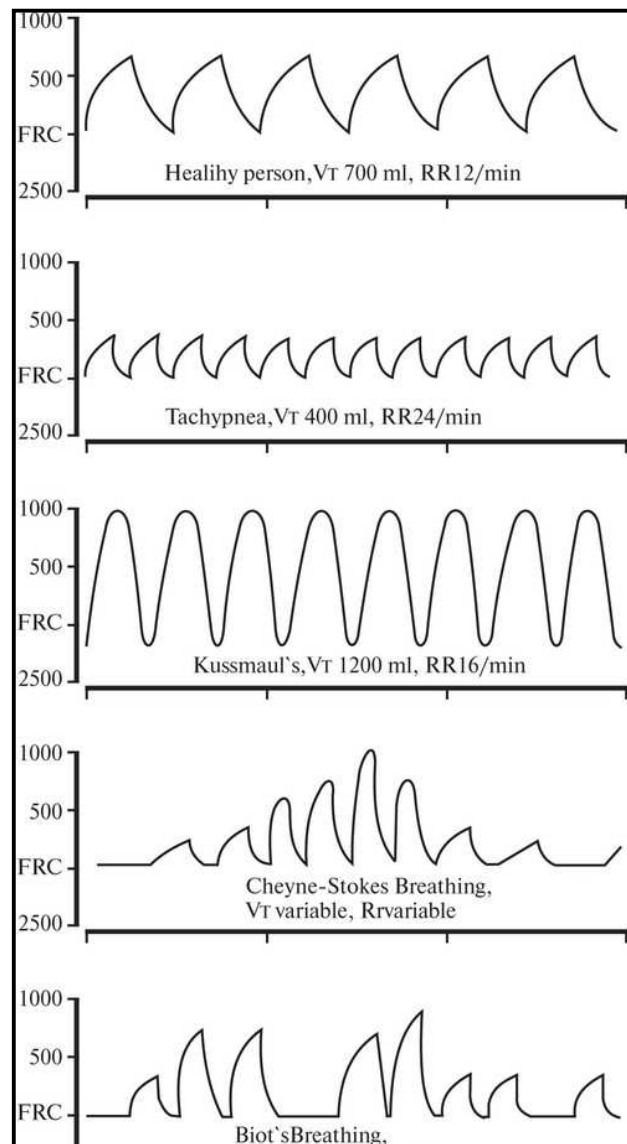


Figure 1. Pathological types of the rhythm and depth of breathing.

Pathological the rhythm and depth of breathing: the breath of Grocka (a type of periodic breathing with undulating respiration without respiratory pauses), breathing Cheyne-Stokes (periodic breathing variation: wave-like breathing + breathing pauses), Biota breathing (deep and rhythmic respiratory movements equal in amplitude + breathing pauses); Kussmaul breathing (deep, noisy breathing), abnormal shallow breathing.

**Palpation.**

General palpation. The purpose of the general palpation is to detect the location of the chest pain.

Resistance (expansion) of the chest - opposite the property of elasticity; causes: emphysema of the lungs, ossification of ribs in the elderly, fluid in the pleural cavity, tumors of the pleura

Vocal fremitus – carrying out oscillatory movements of the vocal cords in the bronchi at the surface of the chest.  Vocal fremitus is determined by palpation  of the chest.  You can test the vocal fremitus by placing palms or more sensitive ulnar border of your hand on the chest while the patient repeats «ninety nine» in a deep clear voice. The corresponding areas on the chest must be tested simultaneously by both palms in symmetrical areas. Vocal fremitus is increased through a consolidated lung (lobar pneumonia) and decreased when the corre sponding bronchi are obstructed, or if there is a pleural effusion. It is useful in distinguishing consolidation from pleural effusion, both of which produce a dull note on percussion.

Causes physiological increase voice fremitus: over upper lobes of the lungs compared to the lower, in men with a low voice, at astenikov with a thin rib cage; Causes physiological weakening voice fremitus: increase of subcutaneous tissue in women and children with high tone of voice, over the lower lung lobes than the upper.

The causes for pathological voice fremitus increase: the inflammatory syndrome seal lung tissue, compression atelectasis, pneumothorax communicating with a bronchus, the air cavity in the lung communicating with the bronchus. The causes of pathological weakening voice fremitus: obesity, hydrothorax, pneumothorax is not communicating with a bronchus, obstructive atelectasis.

**Lungs percussion data in norm and pathology**

Percussion in its modern modification was proposed by Viennese physician Leopold Auenbrugger in 1761. They distinguish the following peculiarities of percussion note: loudness, i.e. amplitude of oscillation, duration, pitch, and tympanic component.  Loudness and duration of percussion note is relating to density of underlying tissues. On comparative percussion the percussion note over the lung may change towards either tympanic and flat note.

Percussion of the lungs — is applied to the chest percussion beats leading subject authorities in oscillatory motion whose physical characteristics (duration of sound vibrations, their frequency, amplitude and timbre coloration) depend on the density of the body, elasticity its structure and moisture content of the air. There are the following methods of percussion: a) direct percussion (Auenbrugger, F. G. Yanovsky and V. P. Obraztsov); b) indirect percussion using pleximeter and mallet percussion finger by finger (P. Pirri, 1827; G. I. Sokolsky, 1835).

The palm of the left hand is placed on the surface of the body, fingers spaced slightly apart, the middle finger plays the role of plessimeter (the site of application of percussion blow to the finger-plessimeter — in the middle of an average or first phalanx). Right wrist bent for applying a percussion blow is placed parallel the left hand at a distance of 1-2 cm between the finger-plessimeter and finger-hammer.

The stroke shold be delivered from the wrist and finger joints to give you control over the force of the blow and over the precision of the site where it lands.

You should pay attention to the fact that the blow should be  abrupt, perpendicular to the finger-plessimeter, finger-the -hammer should not be committed at the finger-plessimeter. For percussion at one point cause two of the same percussion of impact in a short time interval, after which the finger-plessimeter move to a new location.

One of the main advantages of this method of percussion consists in the possibility to dose the force of percussion blow in a wide range, so this method can be used for both comparative and topographic percussion.

Dull percussion sound — small amplitude (volume), duration and relatively high frequency. Tympanic sound — loud, long and relatively low frequency. Clear pulmonary sound — loud, long and also relatively low frequency.

 Clear pulmonary sound, defined a healthy person, is characterized by a rich tonal colouring, which is caused by vibrations of elastic structures of the lung tissue. The propagation of sound vibrations with a quiet percussion (1) — about 3-4 cm, with the average percussion force (2) — 5-6 cm, while conducting a loud percussion

(3) — 7-8 cm. In quiet (threshold) percussion sound waves penetrate deep into tissues 2-3 cm.

As the standard of the absolutely dull sound is the sound, which is determined by percussion of the thigh muscles (femoral sound). The tympanic sound is a sound that can only be detected by percussion of the abdominal cavity and space of Traube. The standard of the clear pulmonary sound is the sound, which will be determined during percussion of the axillary and subscapular areas in a healthy person. The standard hyperresonant (tympanic) sound is the sound that appears when the percussion cushion.

General rules of percussion of the lungs

1. Position of patient and physician should be comfortable to study.

2. Finger-plessimeter pressed tightly to the skin.

3. Finger-the hammer perpendicular to the finger-plessimeter.

4. Right-hand parallel the left (wrist joints placed one above the other).

5. 2 applied percussion blows are delivered through short time intervals.

6. Hand movements are carried out only in the wrist joint.

7. The doctor's hands should be warm.

Distinguish between comparative and topographic percussion of lungs.

Comparative percussion of the lungs is used to determine the nature of pathological changes in the lungs and pleural cavity and used for the diagnosis of bronchopulmonary syndromes.

Technique of comparative percussion is:

1. Conduct a comparison of the nature of percussion sounds obtained over symmetrical areas of the chest.

2. Cause "rebounding" of percussion blows of medium strength. The volume of the percussion sound can change depending on the thickness of the subcutaneous tissue, the degree of development of muscles, the depth of location of the pathological process and other reasons.

3. Percussion is carried out on the intercostal spaces.

To percuss the front of the chest, you should start by percussing over the clavicle on one side, then on the other side, and then percuss on each ribspace and compare the note elicited over the corresponding note on the other side.Percussion is carried out 1 finger phalanx of plessimeter, because anatomically this is the most convenient. Then put the direct percussion blows to the collarbone, using it as plessimeter (Application Fig.4.).

Further percuss in the first, second and third right and left intercostal spaces at the level of the midclavicular line. Below level III intercostal space on the left cardiac dullness, so further research is carried out in the pits of Maranham. The patient stands or sits, arms lowered along the torso, muscles tense, breathing smooth and shallow. The doctor performs the percussion, usually standing to the right of the patient. Finger-plessimeter is parallel to the ribs, but it is tightly pressed against the patient's body.

For percussion axillary region finger-plessimeter put vertically in the upper part of the right, and then left arm. The doctor is beside the patient, opposite the axillary region. Then comparative percussion is carried out by comparing the percussion blows in the third intercostal space of the axillary region on the right and left, and then the percussion continue in the fourth intercostal space of the axillary region on the right and left. The doctor is in front of the patient.

When performing comparative percussion on the posterior surface of the chest at the beginning percuss suprascapular region, the finger-plessimeter set slightly above the spine of the scapula and parallel to it, percussion is applied consistently blows right and left with the patient standing with his hands at his sides, muscles tense. Then percuss "alarm" zones and interscapular region. Finger-plessimeter is parallel to the spine at the edge of the blades, sequentially from right to left. Hands patient is asked to cross on his chest, putting hands on shoulders, with the blades of the supplies are provided, expanding the interscapular space. Further percuss subscapular area (Application Fig.5).. Finger-plessimeter is placed horizontally below the angle of the scapula, alternately right and left. The arms of the patient are lowered along the body, the muscles are relaxed. In detail, the technique

of comparative percussion of the lungs is represented on the website UlSU in the form of a training video http://www.ulsu.ru/com/chairs/pii/stud/Uchebnie_filmi/), as well as at the following address: http://youtu.be/Z9j26eDV5eA

The clinical significance of comparative percussion of lungs: Percussion the clear pulmonary sound is heard in a healthy person over the lungs with unchanged pulmonary tissue. Characteristic sound: loud, long and of low frequency caused by fluctuations in the unmodified elastic structures of the lung tissue. The standard is sound, as determined by percussion in the axillary and subscapular areas in a healthy person.

Dull percussion sound – quiet, vague and high-frequency sound. Is formed over the area of the lung containing less air than in norm or above the liquid. Causes and anatomical localization of physiological shortening of percussion sound: increase the thickness of the pulmonary layer over the right top of the shorter right bronchus; in patient with muscled, in 2-3 intercostal space to the left due to the proximity of the heart, over the upper lobes of both lungs, in the right axillary region due to the proximity of the liver.

### Table 2. Lung comparative percussion abnormalities

| Into dull or flat | Into tympanic |
|---|---|
| Infiltration of the lung parenchyma | Emphysema |
| Exudative pleuritis and hydrothorax | Empty pulmonary cavity (lung abscess) |
| Pleura thickening (adhesions) | Large bronchiectasis |
| Obturative atelectasis | Pneumothorax |

**Topographic percussion**

In order to determine the exact size of the various organs or to differentiate the borders of two organs that lie adjacent to one another, they must be of different densities. Thus, by percussion it is easy to determine where the lung ends and the heart begins because of the different densities of these organs. However, it is extremely difficult to differentiate between heart and liver dullness, or between the dullness of pleural effusion and liver dullness since the densities so closely approximate one another.

The normal limits of pulmonary resonance correspond accurately to the anatomic boundaries of the lung. With light percussion the inferior limits of the lung are found at the level of the sixth rib in the mediclavicular line, the eighth rib in the midaxillary line and the tenth rib in the scapular line (Application Fig 6-8.).

**Table 3.**

**Lungs topographic percussion abnormalities (lower lung borders).**

| Elevation | Depression |
|-----------|------------|
| Shrinking of the lung | Emphysema |
| Thickening of pleura | Asthma |
| Pneumothorax (false depression) | Chronic pulmonary congestion |
| Exudative pleuritis and hydrothorax | High diaphragm |
|  | Flatulence(meteorism) |

The lower limit of pulmonary resonance should in all instances be examined by percussion during both forced inspiration and expiration; normally the difference in space between these two extremes measures 3 to 4 cm. This space represents the complemental pleural space, and by this means the degree of respiratory mobility is attained. This respiratory mobility is diminished or absent in diseases of the lung such as emphysema, pleural diaphragmatic adhesions and conditions that interfere with movement of the diaphragm (Fig.2,3).

**Fig. 2.** Measuring the respiratory mobility of the right lung at midclavicular line
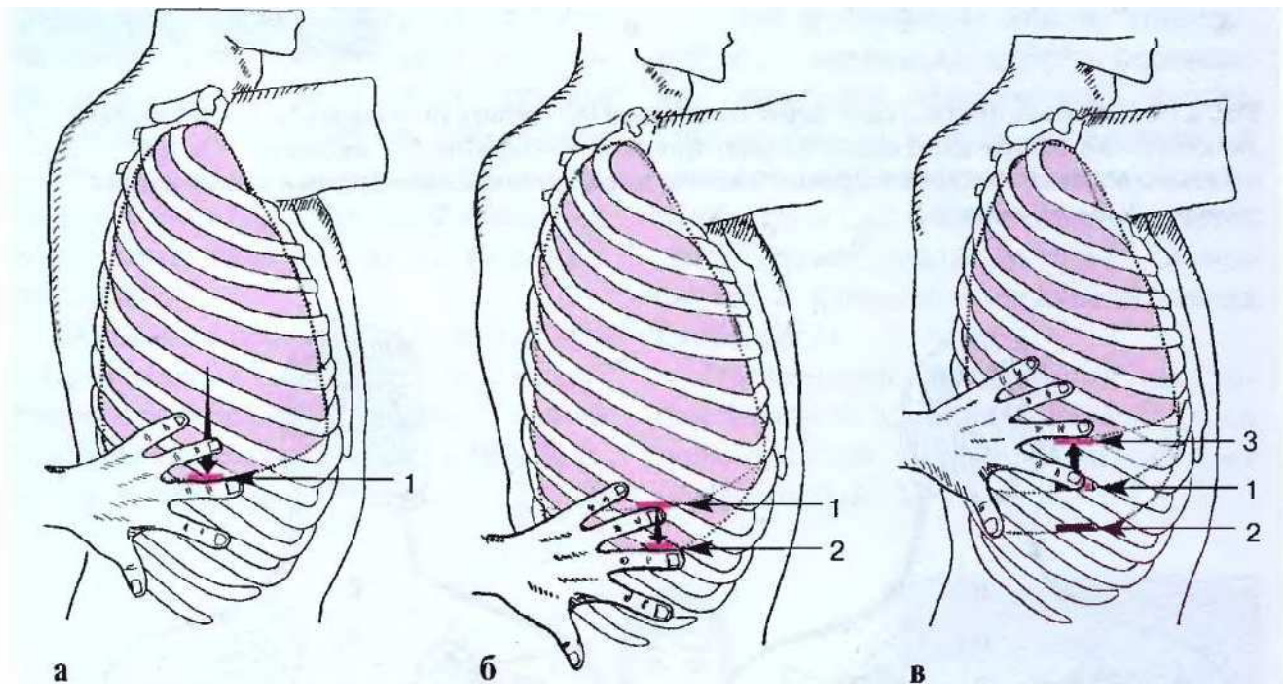(From Ivashkin V.T., Okhlobystin A.V., 2014)



**Fig. 3.** Measuring the respiratory mobility of the right lung

at posterior axillary line

On the left side near the lower costal margin, a tympanitic area, called Traube's semilunar space, is encountered. This area is bounded above by the lower border of the left lung, below by the spleen, internally by the left lobe of the liver and externally by the costal margins. It contains the fundus of the stomach, and the tympanitic note obtained by percussion is occasioned by the air content of the

stomach. When the stomach is filled with food, the tympanitic note is decreased or disappears, as it also does in cases of pericardial effusion and left pleural effusions.

**Lungs auscultation data in norm and pathology**

Auscultation is objective diagnostic method included listening to sound phenomena arising in organs. This method was proposed by Laennec in 1819. The sound of breathing originates somewhere between the pharynx and smaller bronchi, although its exact source remains under study. Respiratory sounds are transmitted through the lungs and chest wall. The tissues through which they pass, however, filter out their higherpitched components. What you hear over most of the lungs are soft, relatively low-pitched sounds that last through inspiration and fade out of your range of hearing relatively early in expiration. Such sounds have been termed vesicular breath sounds. According Laennec, a soft blowing murmur resembling the sound "f-f" is caused by vibration of extending elastic alveolar walls, heard during the whole inhalation. In the first third of exhalation the vibrations of collapsing alveoli walls are still significant and may be heard, and during the last two thirds of exhalation collapse of the alveoli is silent (Application Fig.10).

When you listen near the trachea – over the manubrium or between the scapulae on the level of Th2-4, for example—your stethoscope is close enough to the source of the breath sounds so that little filtration occurs. Here the breath sounds are louder and higher in pitch. This difference is most noticeable during expiration, and you can hear relatively high-pitched breath sounds throughout expiration. –laryngotracheal breath sounds. Harsh and loud respiratory murmur of laryngotracheal respiration, resembling the sound "H-H" is caused by turbulent air flow and associated vibrations of adjacent dense tissues (Application Fig.11).

**Quantitative changes of vesicular breath sound:**

| enhancement: | diminishing: |
| --- | --- |
| Thin chest wall | Thick chest wall |
| Puerile breathing in children | Shallow breathing in weak |

| Vicarious hyperventilation | patients |
| | Narrowing of airways |
| | Thickening of pleura |
| | Emphysema |
| | Presence of small consolidation foci |
| | Fluid or air in pleural cavity |

Qualitative changes of vesicular breath sound: harsh breath sound occurs in bronchitis due to airflow via unevenly narrow and thickened bronchi <mark>(Application Fig. 12)</mark>.

It is more rough and raspy; saccadic (interrupted) breath sound occurs in nervous and shivering patients.

They distinguish the following variants of pathological bronchial breath sound: infiltrative (inflammation, tumour, infarction etc.), compressive (in exudative pleuritis above the fluid border), cavitary (in presence of large pulmonary cavities) – amphoric (<mark>Application Fig. 13</mark>).

Mixed (bronchovesicular) breath sound appears in focal inflammatory pulmonary consolidation (focal pneumonia). Weak bronchial respiration is transmitted to the lung surface in the projection area of a small focus of consolidated pulmonary tissue. The unchanged alveoli surrounding this focus induce vesicular respiratory murmur <mark>(Application Fig. 14)</mark>.

Breath sounds are diminished or absent over thickened pleura, pleural effusion, pneumothorax or whenever there is fibrosis, collapse or infection in the underlying lung <mark>(Application Fig. 15)</mark>. Some people mistakenly state that air entry is diminished into such an area where they do not hear any breath sounds. It should be appreciated that breath sounds are not generated in the alveoli but in the major air passages and conducted through the intervening lungs and pleura to the stethoscope.

**Adventitious sounds.**

**Late inspiratory (fine) crackles.** The opening up of multiple collapsed alveoli produces discontinuous, non-musical crackling, clicking or bubbling sounds during the middle or late phase of inspiration, sometimes spilling over the early part of expiration. The cause of late inspiratory crackles is associated with the appearance in the alveoli a small amount of viscous secretions (transudate, exudate, blood) (Application Fig. 16).These sounds can be imitated by rubbing together a few hairs between the thumb and finger in front of your ear.

Ispiratory crackles are characteristically heard over the lung bases in left heart failure and fibrosing alveolitis, but they may be restricted to one area in lobar pneumonia and localized fibrosis, and over the apex in tuberculosis.

**Inspiratory and expiratory (coarse) crackles** occur during inspiration and expiration, when air passes through the above pathological liquid, forming bubbles that, bursting, give a characteristic sound. Inspiratory and expiratory crackles depend on the size of the lumen (caliber) of the bronchi: there are small, medium and major "buble" crackles (Application Fig.17). As stated above, inspiratory crackles do not occur at random but appear in the same sequence from breath to breath, suggesting that pressure and volume changes determine their occurrence. However, in the late stages of pulmonary oedema and in inflammatory conditions of bronchi, the larger airways may be flooded with oedema fluid or bronchial secretions, and then the crackles will be heard during both phases of respiration. In these cases the crackles appear at random and are modified by coughing.

If crackles in the bronchi, lung tissue surrounded by a slightly changed, which somewhat dampens the sounds, the crackles are heard muted or not euphonic.

**Wheezes.** Wheezes are high-pitched sounds which can be heard without the stethoscope especially at the end of expiration. Polyphonic wheezes consist of a cluster of continuous musical noises and are caused by high velocity of air flow through narrowed small bronchi in asthma, emphysema and chronic bronchitis. Healthy subjects can generate polyphonic wheezes towards the end of a forced expiration as the bronchi are compressed and the velocity of air is increased. In

diffuse airways obstruction due to asthma and chronic bronchitis, wheezes occur at submaximal respiration and may occur even at tidal breathing. In severe airways obstruction, there may be a paradoxical absence of wheezes as the air flow through the maximally narrowed bronchi is very slow. The absence of wheezes under such circumstances is an ominous sign.

Bass wheezing – occurs in large and medium bronchi, the trachea. Auscultation characteristics: low sounds "musical" character, like the humming or buzzing. Better heard on inspiration and fickle (especially when coughing). Diagnostic value: tracheitis, bronchitis (Application Fig. 18).

Whistling wheezing –occurs in the smaller bronchi and bronchioles due to narrowing of the lumen due to the presence of parietal-phlegm, thickening of the bronchial mucosa and or bronchospasm. Feature: a prolonged time of high-frequency, high squealing sounds and "musical tone" similar to a whistle. Better are heard on forced expiration, coughing little change. Observed in bronchitis, bronchoobstructive syndrome (Application Fig. 19).

**Pleural crackles (rub).** These are coarse, non-musical sounds and are heard at some point during both phases of respiration. Pleural crackles are localized to a small area of the chest. They can be imitated by scratching the scalp while the corresponding ear is blocked. The sound is caused by the two inflamed surfaces of the pleura rubbing against each other during respiration and disappears when sufficient fluid accumulates to separate the two layers of the pleura (Application Fig. 20). A pleural rub may be confused with a monophonic wheeze or lung crackles, but it is usually confined to one area, has a fixed relation to **both** phases of respiration, and is not changed by coughing. Sometimes a pleural rub is palpable.

**Internet links:**

1. Bronchial and vesicular breathing:

http://www.youtube.com/watch?v=nhUT5BfAFic&feature=BFa&list=PL9E04387C1A0FBF82

2. Wheezes on the exhale:

http://www.youtube.com/watch?v=YG0-

ukhU1xE&feature=BFa&list=PL9E04387C1A0FBF82

3. Pleural crackles (rub):

http://www.youtube.com/watch?v=t2QE0O_exAQ&feature=BFa&list=PL9E04387C

1A0FBF82

4. Respiratory sounds:

http://www.youtube.com/watch?v=MzTcy6M3poM&feature=related

5. Auscultation of the lungs (1979):

http://www.youtube.com/watch?v=f0pSlN5j_v8


**Pulmonary syndromes.**

Pulmonary consolidation syndrome.

The essence of pulmonary consolidation syndrome is significant decrease or complete absence of lung parenchyma airiness on more or less widespread area (segment, lobe, few lobes simultaneously). This is one of the most frequent syndromes in pulmonary pathology.

*Causes*. They distinguish the following causes of pulmonary consolidation:

1. Inflammatory infiltration (e.g. pneumonia focus, as well as specially defining tuberculous infiltration with inclination to caseous abscess).

2. Pulmonary infarction due to thromboembolism or local vascular thrombosis.

3. Atelectasis and hypoventilation:

   -obturative atelectasis (segmental or lobar);

   -compressive atelectasis (pulmonary [lung] collapse);

   -hypoventilation (e.g. middle lobe hypoventilation due to reduction of middle lobar bronchus patency owing to bronchopulmonary lymph nodes, fibrous tissue; as is well known, middle lobar bronchus incompletely ventilates middle lobe in norm).

4. Lung tumour.

5. Congestive heart failure (blood congestion in lower pulmonary parts).

*Location.* Consolidation focus might have different location (lower, upper parts, middle lobe), that have differential diagnostic meaning. There is defined subpleural

location accompanied, as a rule, by visceral and then parietal pleura involvement. Consolidation may sharply arise (acute pneumonia, lung infarction) or develop gradually (tumour, atelectasis).

Inflammatory infiltration

*Signs.*

Cough

Sputum

Pains enhancing in deep inhalation particularly in subpleural location of consolidation focus.

Asymmetric chest motions in respiration. In large consolidation focus and its superficial location it may be discovered bulging and lag in motion of this part of chest during respiration.

Increased vocal fremitus in consolidation area.

Dull or flat percussion note.

Vesicular breath sound changes into bronchial breath sound, bronchophony increase (Application Fig.21).

At the initial and resolution stages of pneumonia when there is a little amount of exudates in alveoli and they are stretched in air coming in, diminished vesicular breath sound and fine (late inspiratory) crackles (crepitation) are listened above infiltration area. At the height stage of pneumonia alveoli are filled up of exudates, so, vesicular breath sound is replaced by bronchial.

Heterogeneous coarse (inspiratory and expiratory) crackles are heard because of frequent involvement of bronchi in inflammatory process. Revealing of consonant moist fine bubbling rales has particular diagnostic meaning because it witnesses about presence of infiltration zones, increasing sound transmission, around small bronchi.

X-ray allows to obtain a notion about focus shape and size. Consolidation focus of lung parenchyma looks like local shading (Fig.4).

ЛЕВОСТОРОННЯЯ ПНЕВМОНИЯ (С-М ДОЛЕВОГО УПЛОТНЕНИЯ Л-Х), ЦК МСЧ

Fig.4. Consolidation focus (inflammatory infiltration) of lung parenchyma (left lower lobar)

Pleural rub is defined in subpleural situation of infiltration or tumour and in pulmonary infarction.

*Lobar pneumonia: initial stage*

Morphology. Congestion stage - extensive serous exudation, vascular engorgement, rapid bacterial proliferation.

Inspection. An increased respiratory rate is usually evident. Pain is a frequent accompaniment, and with it the involved side shows a lag of respiratory motion.

Palpation. Palpation confirms the findings on inspection. Tactile fremitus is normal or even slightly decreased, and a pleural friction rub may be present.

Percussion. Impaired resonance may be elicited with light percussion. This finding is extremely important.

Auscultation. Although the breath sounds may be diminished, expiration is prolonged and crepitation *(crepitus indux)* is heard. With pleural involvement, a pleural friction sound is determined.

*Lobar pneumonia: stage of consolidation*

Morphology. Red hepatization stage - airspaces are filled with PMN cells, vascular congestion, extravasation of RBC. Grey hepatization stage - accumulation of fibrin, inflammatory WBCs and RBCs in various stages of disintegration, alveolar spaces filled with inflammatory exudate.

Complaints. Coughing may be associated with a sharp pain in the affected side. Mucoid sputum becomes rusty brown (prune juice color).

General inspection. Cyanosis of the lips and fingers. When the fever is high, the face may be flushed. The patient's nostrils dilate on inspiration, and expiration is often grunting.

Inspection. Dyspnea is invariably present. Respiratory movements are generally decreased on the affected side.

Palpation. Diminished respiratory excursions, a pleural friction rub may be felt. Tactile fremitus is increased.

Percussion. Dullness.

Auscultation. Bronchial breathing, bronchophony, pectoriloquy and whispered bronchophony are evident with consolidation provided the bronchus to the involved area is open. Rales are less numerous and distinct than in the stages of engorgement or resolution.

*Lobar pneumonia: stage of resolution*

Morphology. Resolution stage - resorption of the exudate.

Inspection. The patient looks more comfortable and the cyanosis disappears. The dyspnea disappears and the affected lung begins to expand again.

Palpation. The previously increased tactile fremi-tus becomes less marked and gradually findings become normal.

Percussion. The dullness gradually disappears and normal resonance returns.

Auscultation. The bronchial breathing is gradually replaced by bronchovesicular breathing and later by normal vesicular breathing. Crepitation reappears (crepitus redux). Small and large moist rales are heard in increasing numbers.

Compressive atelectasis (pulmonary [lung] collapse) syndrome.

*Causes:*

- pneumothorax
- pleural effusion syndrome

*Sings:*

- Dyspnea.
- Asymmetric chest motions in respiration.
- Increased vocal fremitus in consolidation area.
- The tympanic percussion note is defined at the initial stage of compressive atelectasis development when alveoli still contain air and communication with adductor bronchus is kept. Further, upon complete air resorption percussion note becomes flat.
- Vesicular breath sound changes into bronchial breath sound, bronchophony increase: patent bronchus passes bronchial breath sound extending on periphery through consolidated drawn in pulmonary area (in the case of compressive atelectasis, e.g. lung compression from outside).
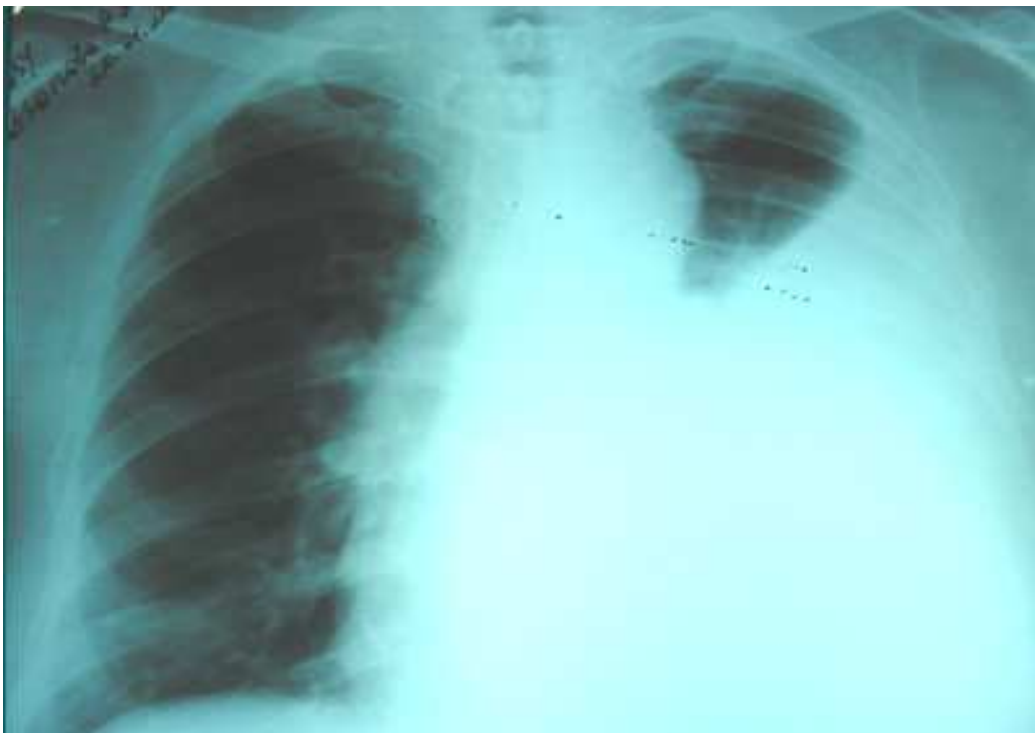


Fig.5. Compressive atelectasis at the left side (lung compression from outside by the fluid – exudates).

Obturative atelectasis (segmental or lobar).

*Causes:*

- closure of airing bronchus lumen by endobronchial tumour, foreign body, compression from outside

*Sings:*

Dyspnea.

Asymmetric chest motions in respiration.

Weakened or disappearance of vocal fremitus in consolidation area.

At the initial stage of atelectasis (hypoventilation stage) when a small amount of aired alveoli in the collapsed area is still kept, diminished vesicular breath sound may be defined. In obturative atelectasis at the complete bronchus closure stage no breath sounds are heard above airless zone. No breath sounds are also heard above tumour. Bronchophony reveals sound transmission increase above pulmonary consolidation area. (Application Fig.22)

Pulmonary cavity syndrome

This syndrome is connected with presence of cavities with dense and smooth walls, not rarely surrounded with infiltrate or fibrous tissue (cavern, abscess, cyst). Symptomatology in every concrete case depends on many conditions:

- Cavity size
- Depth of its location
- Cavity contents: air only (empty cavity), air with some amount of fluid (e.g. air and exudates).
- Cavity communication with respiratory tract (via drainage bronchus) or isolated cavity.

*Causes*

1. Disintegrating (with emptying) lung infiltrate:
   — pneumonia complicated by an abscess;

— pulmonary infarction complicated by an abscess;

— tuberculosis (cavern);

— granulomatous focus (necrotizing respiratory [Wegener's] granulomatosis).

2. Cysts (congenital and acquired).

*Signs.*

Decreased vocal fremitus is characreristic for large superficially located and isolated cavities beyond dependence on their contents.

If cavity communicates with bronchus and even, if partially contains air, there is increase vocal fremitus and tympanitic shade to percussion.

Above cavity filled with fluid there is dullness or flatness to percussion (much as pulmonary consolidation syndrome).

Above empty cavity there is tympanic percussion sound.

Above isolated cavity no breath sounds are heard.

If a cavity communicates with drainage bronchus auscultated bronchial sounds (breath sounds are easily transmitted from glottis along respiratory tract) due to the sound resonance in cavity may acquire metallic shade (resemble a sound of blow on metallic object). *Metallic breath sounds* should be distinguished from *amphoric* (also appeared above cavities with very smooth walls) – the variant of bronchial sounds, differed from usual with musical shade (appears due to resonance of smooth cavity walls). Sounds resembling amphoric breath sounds may be simulated to wind over neck of empty bottle.

Cavity partially containing a fluid not rarely may be issue of moist bubbling rales which, as a rule, are consonant because their transmission is enhanced by surrounding consolidated infiltrated tissues.

Independent stenotic noise which increases bronchial breath sounds, may be heard above the place of cavity connection with drainage bronchus.

*X-ray changes*. More often pulmonary cavities are exactly discovered in the course of X-ray examination (Fig. 6).

CAT allows to detect specific plural small cavities (cysts) forming at the late stage of fibrosing alveolitis (―honeycomb lung).

It is necessary to point that all mentioned signs characterized the pulmonary cavity syndrome are very dynamic as long as staging has place in cavity development. Dynamics of signs is particularly demonstrative in the course of lung abscess: fluid accumulation changes on complete or partial emptying and is accompanied by appropriate symptomatic changes.
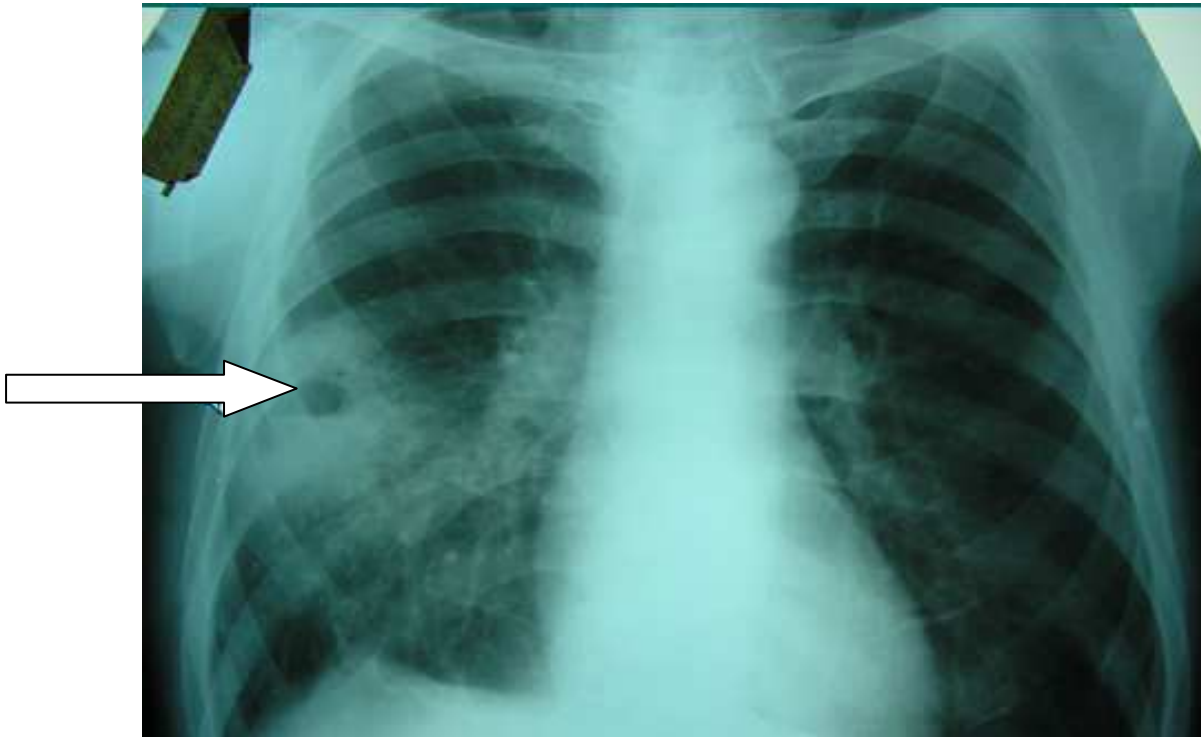


Fig.6. Pulmonary cavity syndrome (lung abscess at the right side).

Pleural effusion syndrome

*Hydrothorax* is the accumulation of increased amount of liquid in pleural cavity. Liquid contents depends on pathologic process character, its stage and intensity. They distinguish exudate and transudate due to liquid contents. Pus (in this case they say *pyothorax* or *empyema*) and blood (*hemothorax*) may also gather in pleural cavity. Effusion may have mixed character.

*Causes.*

1. Essential pleura lesion:

— Inflammation (pleuritis) with exudates production, that may be caused by pathogens as well as immune mechanisms (nonspecific inflammation as manifestation of rheumatic fever, systemic lupus erythematosus and others).

— Tuberculosis: more often para-tubercular nonspecific exudative pleural reaction appears, rarely — proper tubercular pleura affection.

— Pleural tumour (e.g. mesothelioma) or pleural metastases.

   2.  Suppurative processes, including septicemia.

   3.   Pus (or blood) drain from adjacent foci in pulmonary tissue.

   4.  Trauma (wounds) of the thorax.

*Signs*

Fluid in pleural cavity squeezes the lung resulting in *compressive atelectasis* formation and **dyspnea** appearance.

A large liquid amount is accompanied by smoothing of intercostal spaces, protrusion of affected chest side, and its lag in motion on respiration.

Vocal fremitus over the liquid is sharply decreased down to absence.

On comparative percussion in fluid accumulation projection area dull or flat percussion note is defined. Above the upper border of liquid badly ventilated squeezed lung is situated near air containing bronchi, so according to the law of compressive atelectasis it gives dull-tympanitic shade of percussion note.

On topographic percussion peculiarities of dullness upper boundary (which may have various direction due to fluid contents) and also significant restriction on diaphragm excursions on affected side are revealed.

— In case of inflammation (exudate) upper dullness border has the appearance of the curve (Ellis-Damuazo-Sokolov's curve) with the apex along axillary lines, that is characteristic of irregular fluid level elevation.

— Transudate is characterized by more horizontal level of dullness (Fig.7).

Fig.7. Transudate at the right side.

On auscultation above the dullness zone sharp decrease down to absence of vesicular breath sounds, and over this zone – diminished vesicular breath sounds are listened.

— In oblique direction of upper dullness border (e.g. in exudative pleurisy) a part of more squeezed lung (near the spine) is adjacent to large bronchi therefore area of dull-tympanic percussion note and listened bronchial breath sounds – Garlend's triangle is formed. It is limited by upper dullness border above the fluid on below, spine – from one side, perpendicular on the spine, dropped from the crown of the upper dullness border – on top.

- Sometimes in exudative pleurisy one more small area, adjacent to the spine with lower part of dullness zone on the healthy side of the chest, where due to aorta shifting dullness to percussion and no breath sounds are detected – Rauhfus-Grocco's triangle, is marked. It has right-angled triangle form, legs of which are the spine (below the exudate's level) and lower border of the healthy lung and hypotenuse is the continuation of Ellis-Damuazo-Sokolov's curve on the healthy side (Fig.8, 9).
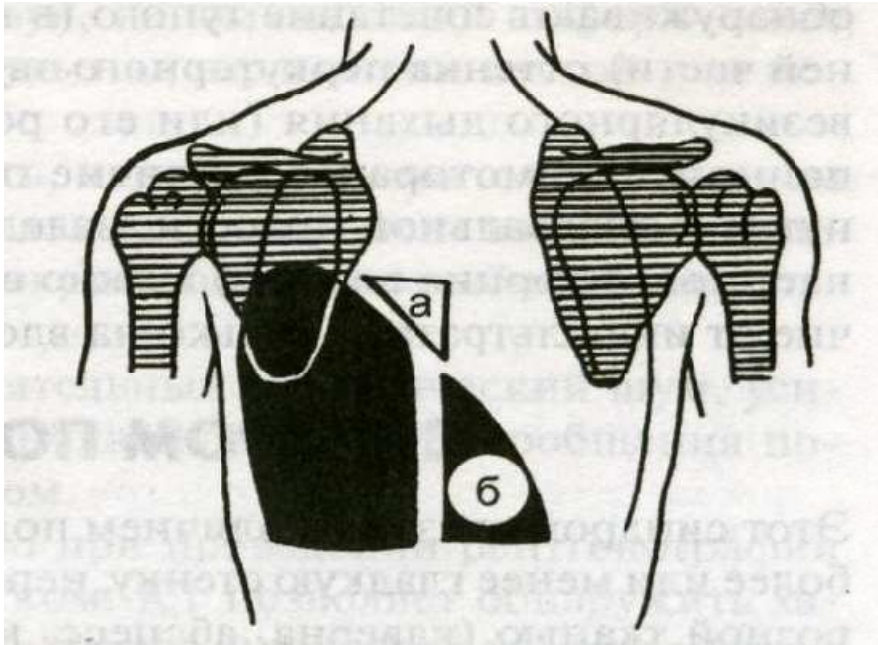
Fig. 8. Garlend's triangle (a) and Rauhfus-Grocco's triangle (б) in exudative pleurisy.
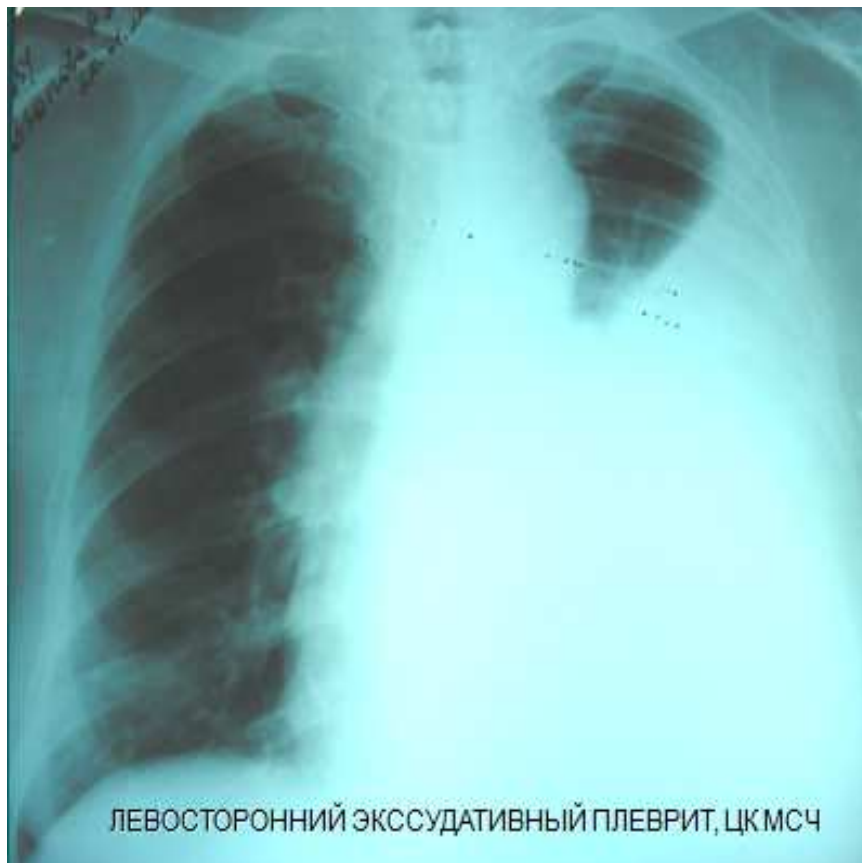


Fig.9. Exudative pleurisy

<u>Syndrome of air in pleural cavity (pnemothorax)</u>

The findings in pneumothorax depend on the size of the pleural airspace. Motion may be normal or diminished, vocal fremitus may be decreased to absent, the percussion note is usually normal but may be more resonant than over the contralateral lung, and breath sounds and bronchophony are decreased to absent.

**Pneumothorax**

*Spontaneous pneumothorax* develops in the absence of any trauma to the chest. When no obvious diseases of the lung are present, a spontaneous pneumothorax is considered to be primary. In contrast, secondary spontaneous pneumothorax develops as a complication of a wide variety of diseases of the airways and lungs.

Primary spontaneous pneumothorax is predominantly a disease of young males and is six times more common in men than in women. It results from the rupture of small apical sub- pleural emphysematous cysts that either are congenital or are caused by bronchiolar inflammation and obstruction. Primary spontaneous pneumothorax is more likely to occur in athenics. It has been suggested that in athenics, the pleural pressure is more subatmospheric at the apex and as a result apical alveoli are more greatly distended. This may play a role in cysts formation in those who are congenitally predisposed. Cigarette smoking also increases the probability of primary spontaneous pneumothorax.

Primary spontaneous pneumothorax is not precipitated by exertion. It usually occurs when the patient is at rest and only infrequently develops during exercise. **Chest pain** and **dyspnea** are the most common symptoms, and only rarely are both these symptoms absent. The chest pain is sudden in onset and pleuritic in nature; shoulder pain reflects irritation of the diaphragmatic pleura. Compression and collapse of the lung by a pneumothorax causes **cough** in over half the patients.

The characteristic findings on **physical examination** include impaired expansion of the involved hemithorax, a tympanic percussion note, and diminished or absent fremitus and breath sounds.

Marked respiratory distress with cyanosis, tachycardia, and hypotension signals *a tension pneumothorax.*

The **diagnosis** is made by identifying a visceral pleural line on the chest radiograph (Fig.10).



Fig.10. Right-sided pneumothorax in an adult woman with asthma.

Spontaneous pneumothorax is also a common complication in patients with underlying lung diseases, most commonly chronic obstructive pulmonary disease.

Patients with underlying lung disease in whom *a secondary spontaneous pneumothorax* develops tend to have severe symptoms and gas-exchange abnormalities. Most complain of **shortness of breath** and **chest pain**, but the shortness of breath is often out of proportion to the size of the pneumothorax. Severe hypoxemia, cyanosis, and hypotension can occur. The mortality may be as high as 15 percent. The clinical diagnosis is often difficult in the patient with severe chronic obstructive pulmonary disease who may have overinflated lungs, decreased breath sounds, and hyperresonance to percussion. A chest radiograph is required to establish the diagnosis. This can sometimes be difficult in the presence of marked emphysema

or bullous disease. Under these circumstances, the diagnosis of pneumothorax should be made only if a visceral pleural line can be demonstrated.

**Traumatic pneumothorax** is most often due to penetrating chest trauma, but it also can occur with closed chest trauma consequent to alveolar rupture from thoracic compression, fracture of a bronchus, esophageal rupture, or rib fractures that lacerate the pleura. Tube thoracostomy is required to evacuate air and blood from the pleural space.

**Jatrogenic Pneumothorax.** Pneumothorax is also a common complication of central venous line insertion, thoracentesis, pleural biopsy, percutaneous needle aspiration of the lung, and transbronchial lung biopsy.

**Tension Pneumothorax.** When the pressure in a pneumothorax exceeds atmospheric pressure, a tension pneumothorax is said to exist. It most commonly occurs during mechanical ventilation or cardiopulmonary resuscitation, but it may complicate any type of spontaneous or traumatic pneumothorax. Characteristic findings on chest radiogram include a shift of the mediastmum away from the pneumothorax and ipsilateral depression of the diaphragm. Since tension pneumothorax is a medical emergency, the diagnosis must be made clinically. Treatment cannot be delayed until a chest radiograph is obtained. Once the presence of a tension pneumothorax is confirmed, a chest tube should be inserted immediately.

---

Hyperinflated lung syndrome (emphysema)

Emphysema is characterized by two features.

Anatomically, it is defined as an abnormal enlargement of the air spaces distal to the terminal bronchioles, accompanied by destructive changes in the alveolar walls.

Physiologically, it is characterized by a loss of elastic recoil and thus an increased lung compliance.

The degree of airways obstruction in patients with COPD correlates most closely with the severity of emphysema, and patients who have significant functional impairment usually have at least a moderate degree of emphysema.

The diagnosis of emphysema is usually inferred from the clinical and laboratory findings.

When examining the chest emphysematous type of chest is determined: barrel shape, expansion of intercostal spaces, clearly defined the angle of Louis, the epigastric angle is obtuse, the ribs are almost horizontal direction, the breath is actively involved auxiliary respiratory muscles (sternokleidomastoid, trapezius, etc. with indrawing of the intercostal spaces), lungs are in the phase of constant inhalation and exhalation is much difficult).

Vocal fremitus is decreased.

Percussion sound is hyperresonant.

On auscultation the diminished vesicular breath sounds are determined. In patients with COPD wheezes may also be determined.
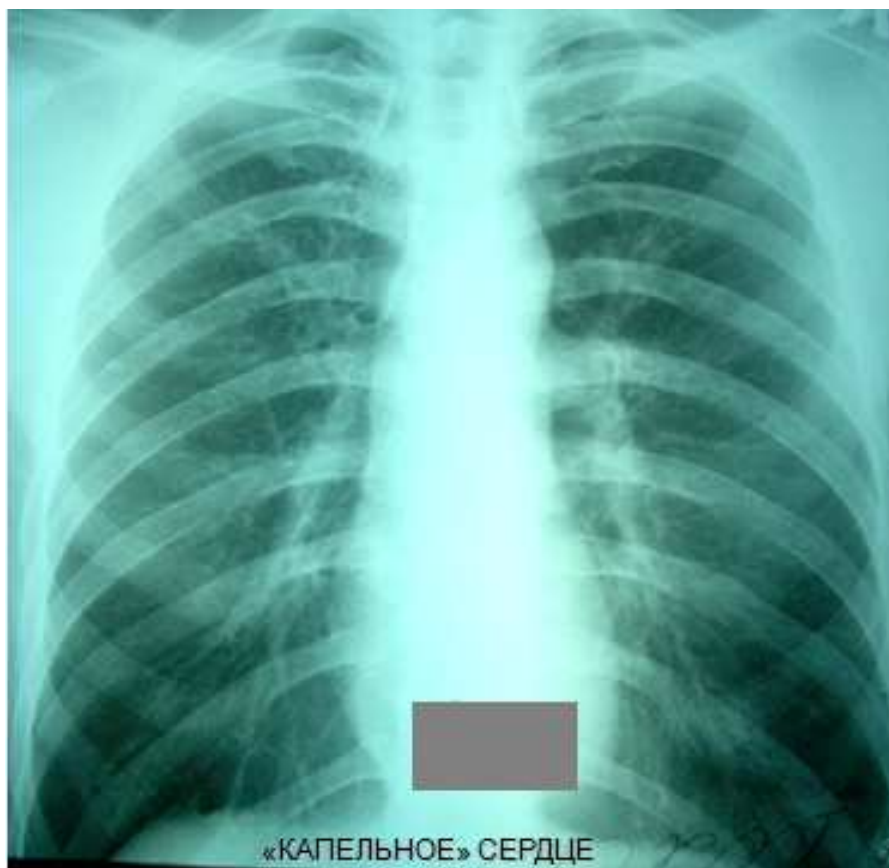


Fig.11 Emphysema and "drip" heart

Chest roentgenograms demonstrate hyperinflation with depressed diaphragms, increased anteroposterior diameter, and widened retrosternal air space. These findings, however, are seen whenever hyperinfiation is present, and more specific

features in emphysema include attenuation of the pulmonary vasculature. The one finding that correlates well with the anatomic presence of emphysema is a reduction in diffusing capacity because of the loss of alveolar capillaries.

**A list of the main instrumental and laboratory methods of examination of respiratory system**

Chest x-ray

Computed tomography of chest organs

Bronchoscopy

Spirometry and peakflowmetry

The study of lung diffusion capacity (transfer factor)

Measurement of airway resistance (the method of "short-term interruption of the air flow" or by the method of oscillations

Capnography

Bodyplethismography

Pulse oximetry, including monitoring of blood oxygenation

microtechnique Astrup (research arterializing gas composition of blood and acid-base balance)

Blood tests

Sputum tests

sputum on flora and sensitivity to antibiotics

examination of sputum for Mycobacterium tuberculosis and atypical cells

blood test for tumor markers (Cyfra 21-1)

Fiberoptic bronchoscopy is used for diagnostic and therapeutic purposes (see Table 4)

Diagnostic bronchoscopy allows visually to estimate respiratory tract peculiarities from glottis to subsegmental bronchi, to obtain samples of content of respiratory tract on different levels for bacteriological and cytological examination, to perform bronchopulmonary lavage with subsequent sampling of received fluid. Using bronchoscope it is possible to perform puncture biopsy of bronchial mucous and tansbronchial biopsy of adjacent tissues (lymph node, lung parenchyma).

Bronchoscopy is used for therapeutic purposes, for example, for bronchopulmonary lavage and local introduction of antibacterial drugs in bronchiectasis (bronchi sanation), dilution and aspiration of mucus from corked bronchi lumen in intractable asthmatic onset (particularly in presence of so-called ―dumb lung) and removal of foreign bodies.

Absolute contraindications: unstable angina, acute myocardial infarction, acute stroke, pulmonary heart and cardiovascular failure article III, life-threatening arrhythmias, severe hypoxemia, absence due to mental illness the patient contact the doctor with the patient.

**Table 4.**

**Indications for bronchoscopy**

| Indications for bronchoscopy | Purposes |
| --- | --- |
| Hemoptysis | Determination of source of bleeding and hemostasis. |
| Chronic cough without visible reason. | Detection of possible intrabronchial tumour invisible on X-ray. |
| Delayed pneumonia resolution | Exception of local bronchial obstruction |
| Atelectasis | Determination of its cause. |
| Cancer of lung | Biopsy, estimation of operable status |
| Abscess | Exception of bronchial obstruction, receiving of material for bacteriological examination and cavity drainage improvement |
| Bronchiectasis | Bronchial lavage, introduction of medications (antibiotics, for example) |
| Dumb lung | Dilution and aspiration of mucus |
| Foreign body | Removal |

Bronchoscopy plays a central role in the diagnosis of tumors of the tracheobronchial localisation. central lung cancer when the tumor arises from the mucosa of the bronchi of the 1st, 2nd and 3rd orders, which are radiographically the

root of the lung. On the nature of growth of central cancer is divided into endobronchial and peribronchial, in turn, is divided into endobronchial exophytic (nodular form) and endophytic (flat infiltrations and ulcers). During the bronchoscopic studies the diagnosis of lung cancer is based on the direct and indirect endoscopic signs. Direct signs include the presence of the tumor is exophytic or endophytic (Application Fig. 23, 24). Exophytic tumour has a hemispherical or semi-oval shape, rough surface, grayish-red, wide base. Partially or completely occlusive the lumen of the bronchus.

It is difficult to make a differential diagnosis between a stenosis of bronchus inflammatory and neoplastic etiologies in cases of infiltrative tumor growth. Diagnostic difficulties help to resolve forceps and brush biopsy.

Ulcerative form of cancer is an ulcer of irregular shape, with fuzzy edges and uneven bottom, covered with fibrinous coating (Application Fig. 25). Biopsies are taken with the tongs from the edges of the ulceration.

When foreign bodies of the trachea the cough is paroxysmal in nature. On bronchoscopic examination of patients is usually direct, as a rule, with other diagnoses: pneumonia, lung cancer, hemoptysis of unclear etiology, bronchial asthma (Application Fig. 26).

It is necessary to emphasize the bronchoscopy need for patients with hemoptysis (especially repetitive) or bronchial [pulmonary] hemorrhage since it allows to determine source of bleeding (trachea, bronchi, parenchyma) and its cause (bronchiectasis, tumour, tuberculosis).

Also there are used thoracoscopy (inspection of the pleural sheets) and mediastinoscopy (inspection of anterior mediastinum). One of the basic purposes of these investigations is to receive biopsy material.

**Blood gases**
The presence of respiratory failure may be suspected by the signs of central cyanosis. It is important to define the type and extent of failure of oxygenation and this is best done by measurement of arterial blood gas tensions ($PaO_2$ and $PaCO_2$), oxygen saturation ($SaO_2$) and pH.

**The study of gas composition and blood acid-base status (Astrup microtechnique).**

Normally, in a healthy person the constancy of pH (7,4; 7,35-7,45) is maintained by buffer systems of blood and lung and kidney (Shmidt R., Tevs G., 1996). Equilibrium in acid-base status is determined by ratio of hydrogen (H+) and hydroxyl (OH-) ions. With increasing concentration of H+ ions oxidizes the blood, while increasing OH - ions oxalacetate. Bicarbonate buffer system regulates the buffering capacity of the blood due to the change $PaCO_2$ - voltage of carbon dioxide in the blood (level $PaCO_2$ depends on lung ventilation and at the same time affects it). Adjusting the tension of $CO_2$ in blood, respiratory system facilitates effective buffer system in General.

Important buffer parameters:

1. BB - the sum of all the buffer bases of the blood ≈ 48 mmol/L. This value does not change when the shifts $PaCO_2$;

2. BE - the excess of bases (shows how the concentration of buffer bases is deviated from the normal value (≈ 48 mmol/l). The normal value of VE varies from -2.5 to +2.5 mmol/L. the Level BE > -5 mmol/l – metabolic acidosis.<5 mmol/l is characteristic of metabolic alkalosis.

3. SB - standard bicarbonate. SB ≈ 24 mmol/l. SB corresponds to the content of bicarbonate in the plasma of fully oxygenated and equilibrated with a gas mixture (PCO2 = 40 mm Hg) at 37 degrees Celsius.

For the study of gas composition of blood and acid-base status is used microtechnique Astrup. Analyzed blood obtained by puncture of the scarifier terminal phalanx of one finger, pre-warmed for 10 minutes at a temperature of about 40 ° C and treated with alcohol. Portion blood type in special capillaries that are on both sides immediately after the blood sample are isolated from the air "plugs". Oxygen tension is measured polarographically (Shmidt R., Tevs G., 1996).

Define:

1. indicators in acid-base status: pH, BE, BB, SB, etc.;

2. indicators of gas composition of blood arterialization: partial tension of oxygen $PaO_2$ (normal > 80 mm Hg) partial tension of carbon dioxide $PaCO_2$ (normal 35-45 mm Hg), the saturation of hemoglobin by oxygen $SaO_2$ (normal > 95%).

The main types of violations arterializing gas composition of blood and acid-base balance:

1. Arterial hypoxemia ($PaO_2$ <80 mm Hg). At persons of young age PaO2 is about 95 mm Hg to 40 years it is reduced to 80 mm Hg and 70 years approximately 70 mm Hg. In the clinic chronic respiratory diseases are the most frequent cause of arterial hypoxemia is the development of chronic respiratory insufficiency (IR) in COPD patients

2. Arterial hypocapnia ($PaO_2$ < 35 mm Hg). Characteristic of the early stages IR when arterial hypoxemia is compensated by the increased pulmonary ventilation, resulting in lower $PaCO_2$ and development of **respiratory alkalosis**.

3. Arterial hypercapnia (PaCO2 > 45 mm Hg). A characteristic feature of the later stages IR when the removal of carbon dioxide through the alveolo-capillary membrane is disturbed. It is often noted **respiratory acidosis** is an important sign of severity IR.

4. **Respiratory alkalosis** (PaCO2 < 35 mm Hg, BE = ± 2.5 mmol/l).

5. **Respiratory acidosis** (PaCO2 > 45 mm Hg, BE = ± 2.5 mmol/l)

6. **Metabolic alkalosis** (PaCO2 = 35-45 mm Hg BE > +5 mmol/l) is atypical for a primary lesion of the lung.

7. **Metabolic acidosis** (PaCO2 = 35-45 mm Hg, BE < -5 mmol/l) - metabolic acidosis atypical for a primary lesion of the lung.

The study of gas composition and blood acid-base balance is a powerful technique for objectifying the severity of chronic respiratory failure. To determine blood gas composition in COPD patients is recommended when the level of oxygen saturation of hemoglobin ($SpO_2$) by pulse oximetry data < 92% and $FEV_1$ <50%.

The response to drugs and the therapeutic response to oxygen can then be monitored easily. Haemoglobin saturation reflects oxygen carriage by the blood and

thus the adequacy of tissue oxygenation (if perfusion is satisfactory) and the requirement for oxygen therapy. This can be measured noninvasively by pulse oximetry. Normally haemoglobin saturation is 95-99%. There is a type of pulse oximetry providing 12-24 hours monitoring of oxygen saturation ($SaO_2$) during night sleep (Fig.12). It is important for diagnosis of night hypoxemia in COPD patients and obstructive apnea.
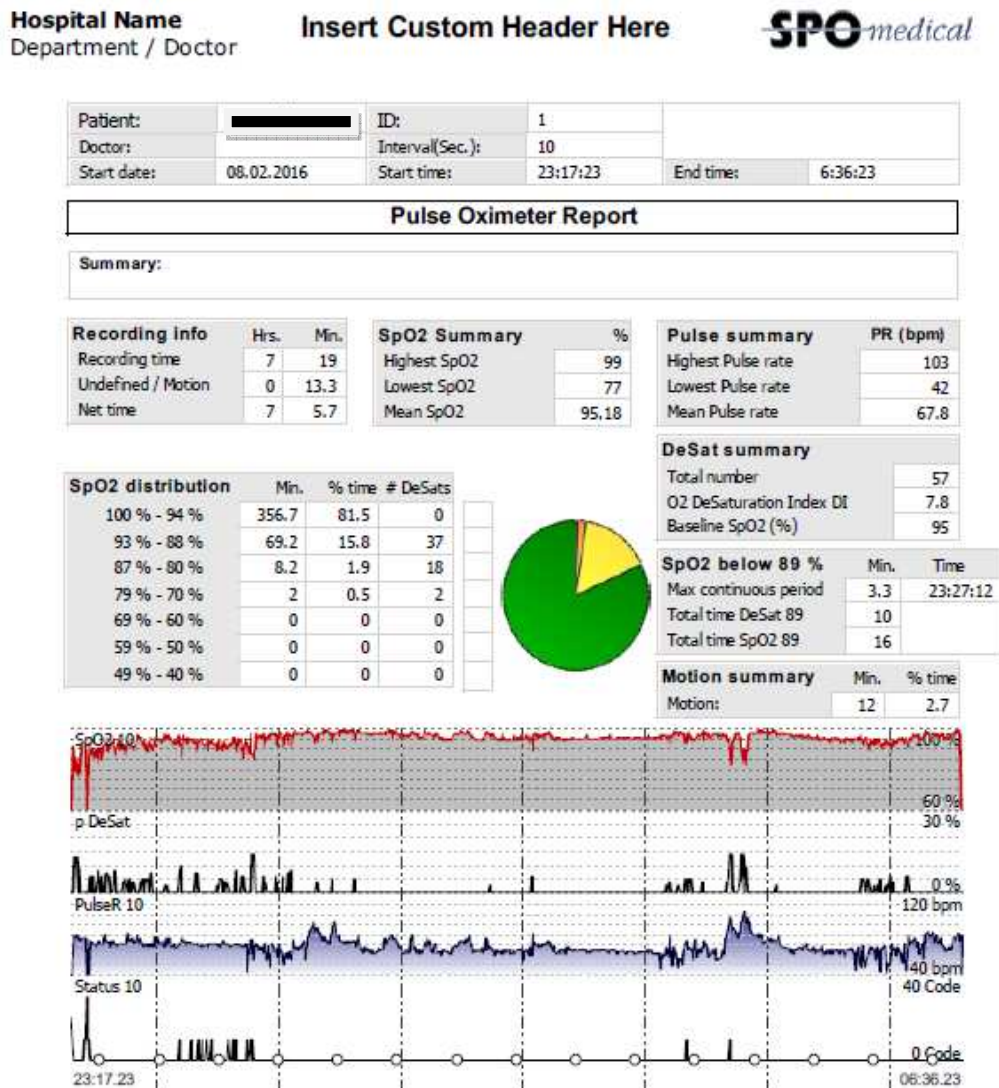


Fig.12. Night sleep monitoring of oxygen saturation ($SaO_2$) in COPD patient (our own observation).

Decrease of oxygen saturation of hemoglobin min SpO2 decreased to 77%, SpO2max - 99%, SpO2 medium – 95,18%, total number of desaturation during night sleep – 57.

There is a type of pulse oximetry providing monitoring of oxygen saturation ($SaO_2$) during physical exercises, for example 6 minutes walking test (6MWT) (Fig.13).
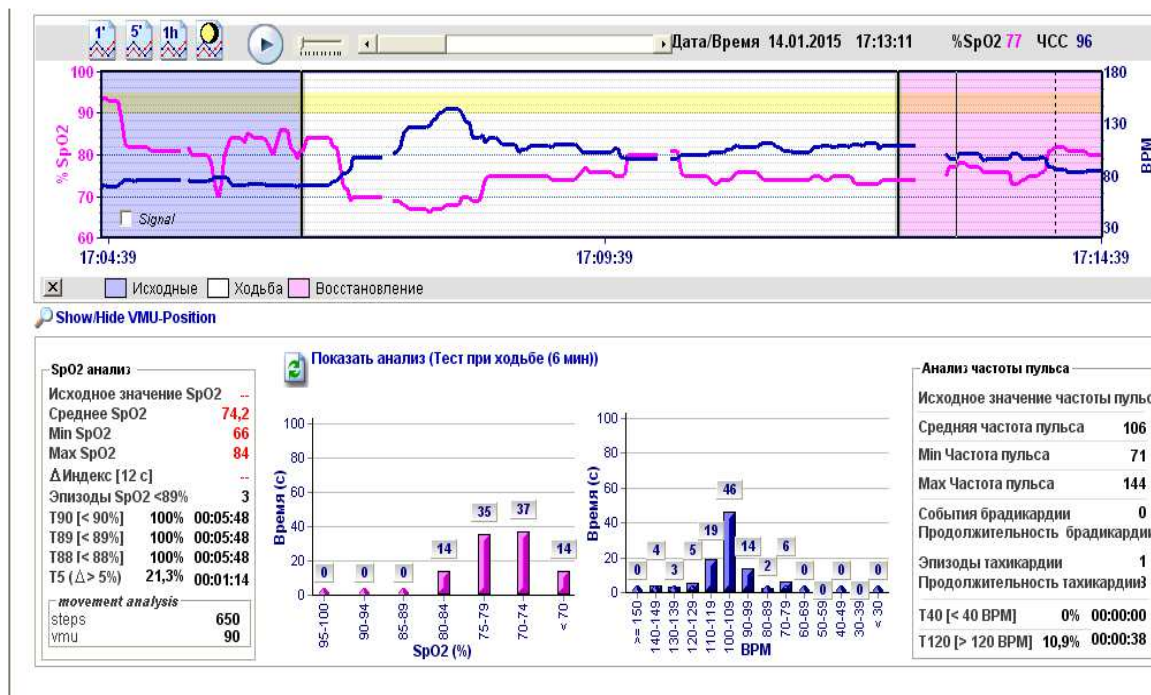


Fig.13. Monitoring of oxygen saturation ($SaO_2$) during 6 minutes walking test (our own observation).

When conducting a test with 6 minute walk of marked reduction in distance travelled (63% of theoretical), decrease of oxygen saturation of hemoglobin and hemodynamics: min SpO2 decreased to 66%, SpO2max - 84%, SpO2 medium - 74,2%, range SpO2 shifted in ranges of less than 84% (SpO2 in the range of 95-100% 94-90% is not registered, SpO2 in the range of 80-84% - was 14% of the recording time, SpO2 in the range of 75-79% - 35%, SpO2 in the range of 70-74% - 37% and SpO2 in the range < 70 – 14% of the time entries); index T5($\Delta \geq 5\%$) has doubled, reaching 21.3 per cent. Cardio-vascular system also highlights the negative trends of heart rate: the heart rate medium 106 beats/min, min heart rate is 71 BPM, max heart rate - 144 beats/min, recorded one episode of tachycardia duration of 10.8% of the recording time.

**Pulmonary function testing** includes both simple **spirometry**

*Static lung volumes* (see Fig. 14) reflect the elastic properties of the lungs and chest wall.
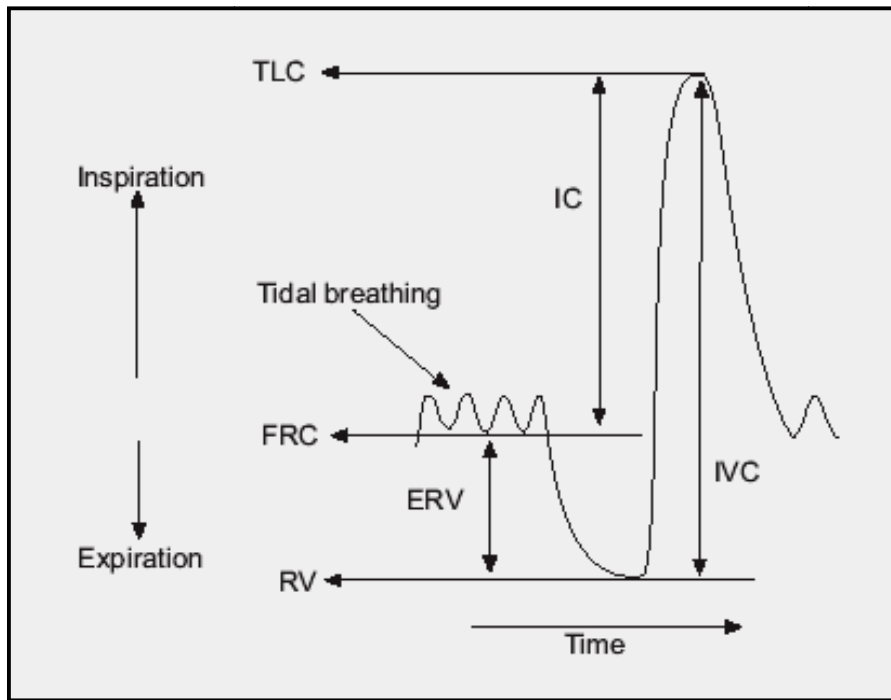
Fig. 14. Static lung volumes  ATS/ERS Task Force: Standartisation of lung function testing, 2005
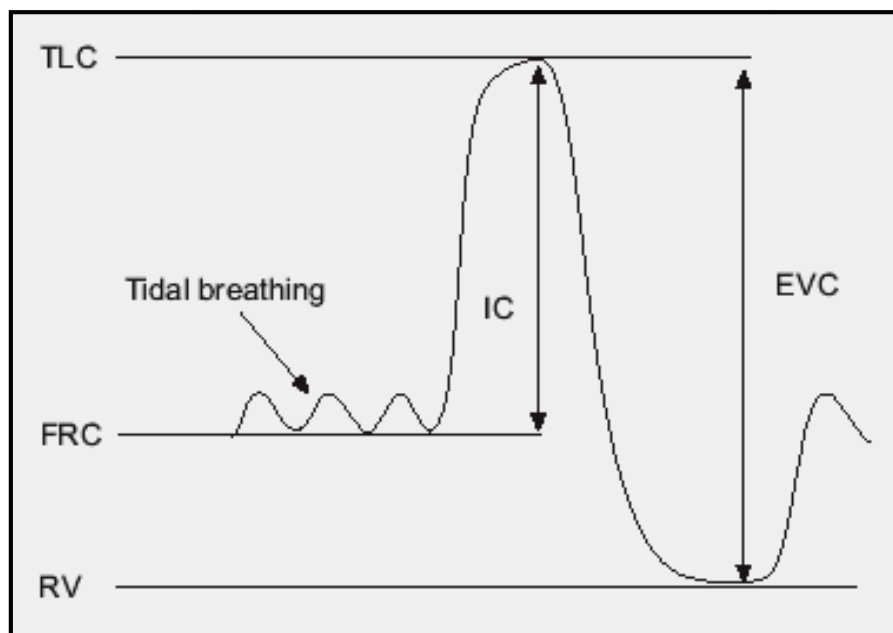


Fig. 15. Static lung volumes  ATS/ERS Task Force: Standartisation of lung function testing, 2005

*Vital capacity* (VC or "slow VC") is the maximum volume of air that can be expired slowly after a full inspiratory effort. Simple to perform, it is one of the most valuable measurements of pulmonary function. Because VC decreases as **a**

**restrictive lung disorde**r (eg, pulmonary edema, interstitial fibrosis) worsens, it can be used along with the diffusing capacity to follow the course of such a disorder and its response to therapy. The VC also reflects the strength of the respiratory muscles and is often used to monitor the course of neuromuscular disorders.

*Forced vital capacity* (FVC), similar to VC, is the volume of air expired with maximal force. It is usually measured along with expiratory flow rates in simple spirometry (see Dynamic Lung Volumes and Flow Rates, below). The VC can be considerably greater than the FVC in patients with **airway obstruction**. During the FVC maneuver, terminal airways can close prematurely (ie, before the true residual volume is reached), trapping gas distally and preventing its measurement by the spirometer.

*Total lung capacity* (TLC) is the total volume of air within the chest after a maximum inspiration.

*Functional residual capacity* (FRC) is the volume of air in the lungs at the end of a normal expiration when all respiratory muscles are relaxed. Physiologically, it is the most important lung volume because it approximates the normal tidal breathing range. Outward elastic recoil forces of the chest wall tend to increase lung volume but are balanced by the inward elastic recoil of the lungs, which tends to reduce it; these forces are normally equal and opposite at about 40% of TLC. Loss of lung elastic recoil in **emphysema** increases FRC. Conversely, the increased lung stiffness in pulmonary edema, interstitial fibrosis, and other **restrictive disorders** decreases FRC. Kyphoscoliosis leads to a decrease in FRC and in other lung volumes because a stiff, noncompliant chest wall restricts lung expansion.

*Inspiratory capacity* is the difference between TLC and FRC.

The FRC has two components:

*residual volume* (RV), the volume of air remaining in the lungs at the end of a maximal expiration, and

*expiratory reserve volume* (ERV); ERV = FRC - RV.

The RV normally accounts for about 25% of TLC (see Fig. 15).

*Dynamic lung volumes* reflect the caliber and integrity of the airways. Spirometry (see Fig. 16) records lung volume against time during an FVC maneuver.

*Forced expiratory volume in 1 sec* (FEV1) is the volume of air forcefully expired during the first second after a full breath and normally accounts for > 75% of the FVC. This value is recorded both as an absolute value and as a percentage of the FVC (FEV1 %FVC).

*The mean forced expiratory flow* during the middle half of the FVC (FEF25-75%) is the slope of the line that intersects the spirographic tracing at 25% and 75% of the FVC. The FEF25-75% is less effort-dependent than the FEV1 and is a more sensitive indicator of early airway obstruction.
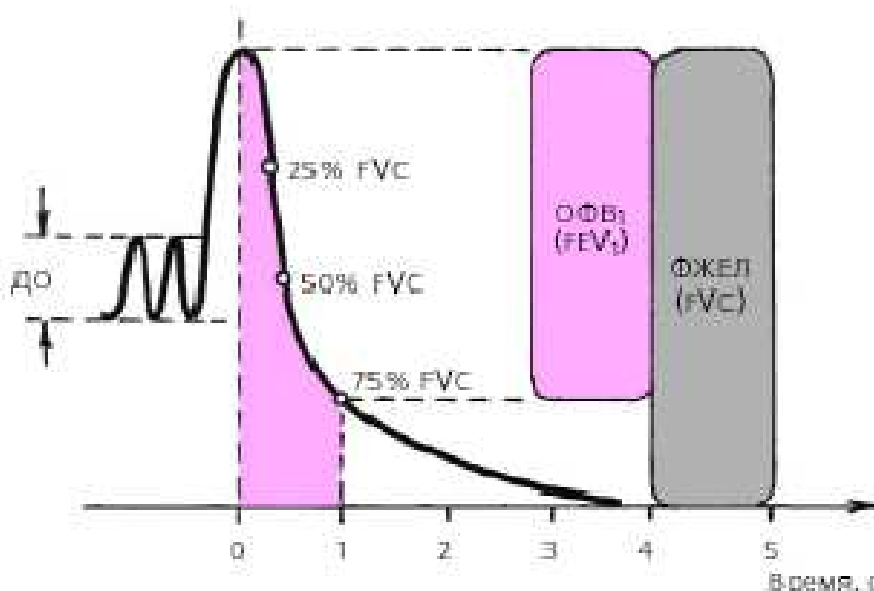


Fig.16. Dynamic lung volumes.

(From Roitberg G.E., Strutynskyi A.V., 2001).

Prolongation of expiratory flow rates is increased by bronchospasm (in asthma), impacted secretions (in bronchitis), and loss of lung elastic recoil (in emphysema). In fixed obstruction of the upper airway, flow is limited by the caliber of the narrowed segment rather than by dynamic compression, resulting in equal reduction of inspiratory and expiratory flow rates.

In restrictive lung disorders, increased tissue elastic recoil tends to maintain the caliber of the larger airways so that at comparable lung volumes, flow rates are often higher than normal.

Retesting pulmonary function after the patient inhales a bronchodilator aerosol (eg, albuterol, ipratropium) provides information about the reversibility of an obstructive process (ie, the asthmatic component). Improvement in FVC or $FEV_1(L)$ of > 15 to 20% is usually considered a significant response (Fig.17). In patients with airway obstruction, absence of a response to a single exposure to a bronchodilator, however, does not preclude a beneficial response to maintenance therapy. In bronchoprovocation testing, a significant decrease in flow rates after inhaling methacholine (a cholinergic drug) may indicate asthma.
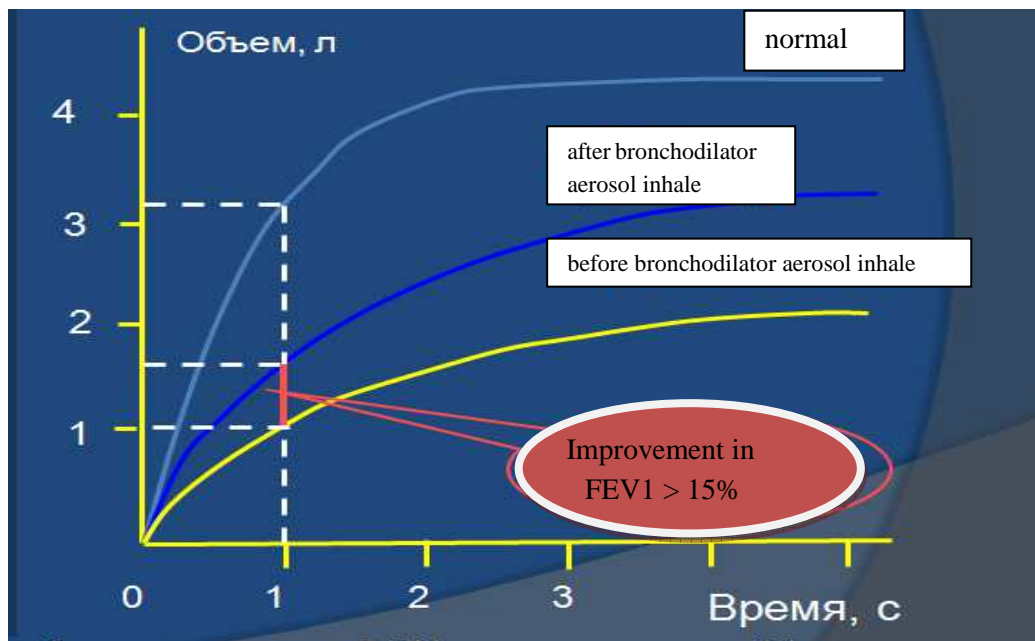


Fig.17. $FEV_1$ before and after bronchodilator aerosol inhale.

*Maximal voluntary ventilation* (MVV) is determined by encouraging the patient to breathe at maximal tidal volume and respiratory rate for 12 sec; the volume of air expired is expressed in L/min. The MVV generally parallels the FEV1 and can be used to test internal consistency and estimate patient cooperation. The MVV can be estimated from the spirogram by multiplying the FEV1(L)x40.

When the MVV is disproportionately low in a patient who seems to be cooperating, neuromuscular weakness should be suspected. Except in advanced neuromuscular disease, most patients can generate fairly good single-breath efforts (eg, FVC). Because the MVV is much more demanding, it can reveal the diminished reserves of weak respiratory muscles. The MVV decreases progressively with

increasing weakness of the respiratory muscles and, along with maximum inspiratory and expiratory pressures (see below), may be the only demonstrable pulmonary function abnormality in patients with moderately severe neuromuscular disease.

The MVV is important preoperatively because it reflects the severity of airway obstruction as well as the patient's respiratory reserves, muscle strength, and motivation.

*The flow-volume loop* is generated by continuously recording flow and volume with an electronic spirometer during a forced inspiratory and expiratory VC maneuver.

The shape of the loop reflects the status of the lung volumes and airways throughout the respiratory cycle. Characteristic changes occur in restrictive and in obstructive disorders. The loop is especially helpful in detecting laryngeal and tracheal lesions. It can distinguish between fixed obstruction (eg, tracheal stenosis) and variable obstruction (eg, tracheomalacia, vocal cord paralysis) of the upper airway. Fig. 18. illustrates some characteristic flow-volume loop abnormalities.
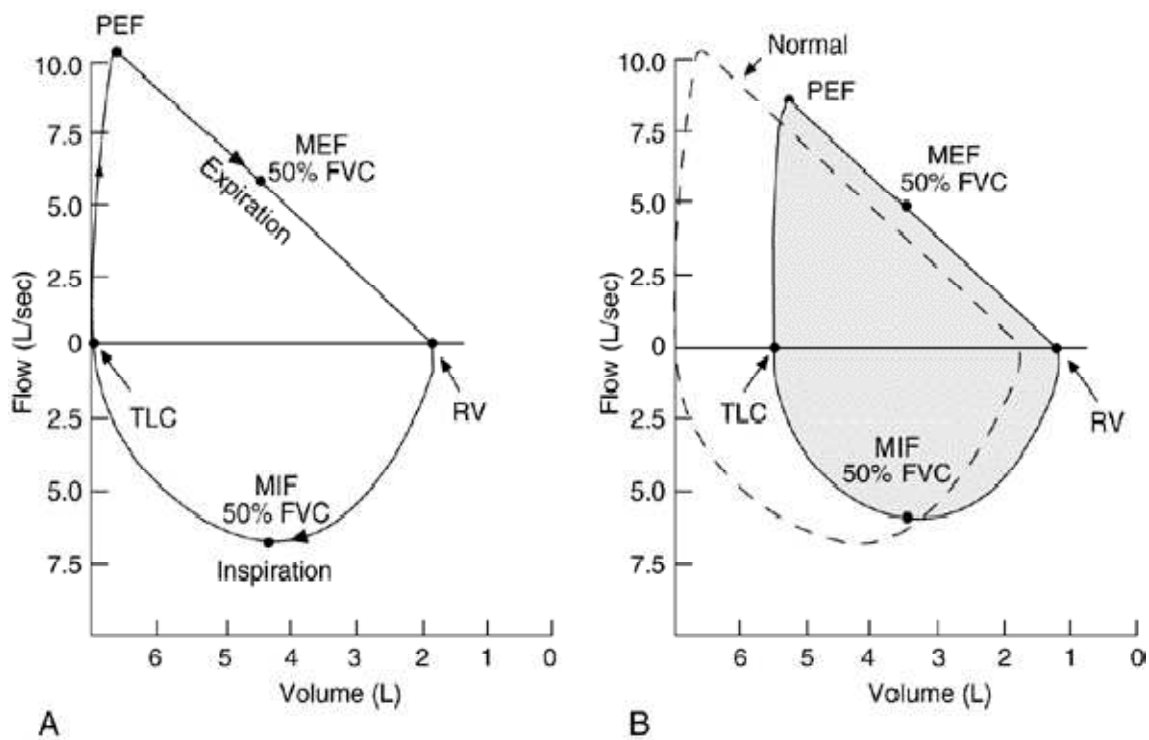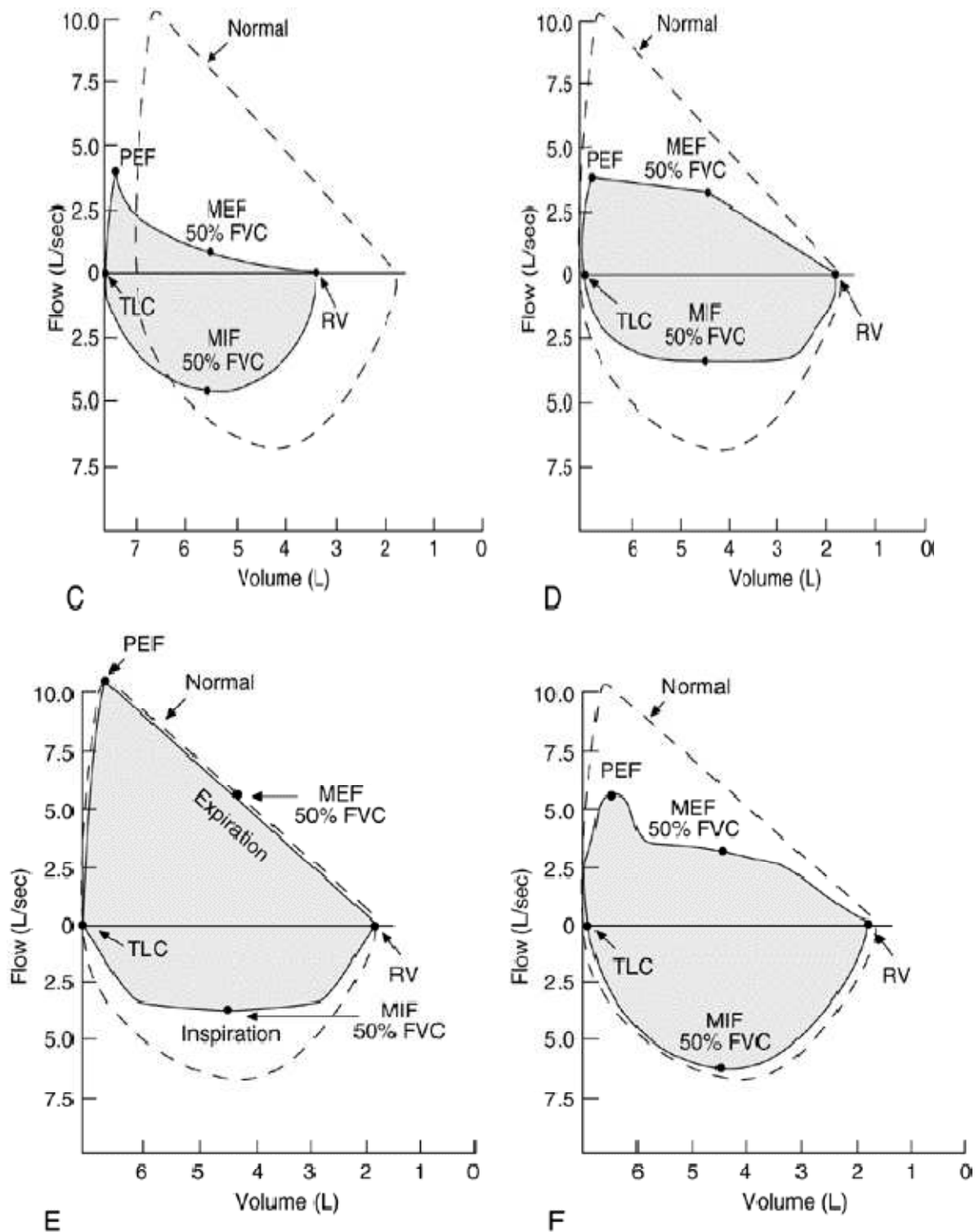
Fig.18. Flow-volume loops.

(**A**) Normal. Inspiratory limb of loop is symmetric and convex. Expiratory limb is linear. Flow rates at midpoint of VC are often measured. MIF 50%FVC is > MEF 50%FVC because of dynamic compression of the airways. Peak expiratory flow is sometimes used to estimate degree of airway obstruction but is very dependent on patient effort. Expiratory flow rates over lower 50% of FVC (ie, approaching RV) are sensitive indicators of small airways status. (**B**) Restrictive disease (eg, sarcoidosis, kyphoscoliosis). Configuration of loop is narrowed because of diminished lung volumes, but shape is basically as in (A). Flow rates are normal (actually greater than normal at

comparable lung volumes because increased elastic recoil of lungs and/or chest wall holds airways open). (**C**) COPD, asthma. Though all flow rates are diminished, expiratory prolongation predominates, and MEF is < MIF. (**D**) Fixed obstruction of upper airway (eg, tracheal stenosis, bilateral vocal cord paralysis, goiter). Top and bottom of loop are flattened so that the configuration approaches that of a rectangle. The fixed obstruction limits flow equally during inspiration and expiration, and MEF = MIF. (**E**) Variable extrathoracic obstruction (eg, vocal cord paralysis). When a single vocal cord is paralyzed, it moves passively in accordance with pressure gradients across the glottis. During a forced inspiration, it is drawn inward, resulting in a plateau of decreased inspiratory flow. During a forced expiration, it is passively blown aside and expiratory flow is unimpaired, ie, MIF 50%FVC is < MEF 50%FVC. (**F**) Variable intrathoracic obstruction (eg, tracheomalacia). During a forced inspiration, negative pleural pressure holds the "floppy" trachea open. With forced expiration, the loss of structural support results in narrowing of the trachea and a plateau of diminished flow (a brief period of maintained flow is seen before airway compression occurs). (From Oslopov B.N, Sadykova A.R., Karamysheva I.V. Introduction to internal diseases. Manual. Part V.2nd edition., 2008).

***Ordering Pulmonary Function Tests.*** As a general preoperative screen, determination of the FVC, FEV1, FEV1 %FVC, and MVV usually suffices. Testing should be performed before chest or abdominal surgery in smokers > 40 yr old and in patients with respiratory symptoms. In patients with suspected laryngeal or tracheal disorders, a flow-volume loop should be requested. If weakness of the respiratory muscles is suspected, the MVV, MIP, MEP, and VC are the appropriate tests.

A complete set of pulmonary function tests should be requested when the clinical picture does not coincide with the data obtained by simple spirometry or when more complete characterization of an abnormal pulmonary process is desired. A complete set includes determination of static and dynamic lung volumes, DLCO, flow-volume loop, MVV, MIP, and MEP. However, extensive testing is tiring, time-consuming, expensive, and unnecessary for adequate clinical assessment of most patients. Periodic determinations of VC and DLCO usually suffice to monitor patients with interstitial lung disease.

Table 5 is intended as general guidelines for interpreting pulmonary function tests.

Table 5

## Restrictive lung disease

| Impairment | None | Mild | Moderate | Severe | Very Severe |
|---|---|---|---|---|---|
| VC (%predicted) | >80 | 60-80 | 50-60 | 35-50 | <35 |
| $FEV_1/FVC$ | >75 | >75 | >75 | >75 | >75 |
| MVV | >80 | >80 | >80 | 60-80 | <60 |
| RV(%predicted) | 80-120 | 80-120 | 70-80 | 60-70 | <60 |

## Obstructive lung diseases

| Impairment | None | Mild | Moderate | Severe | Very Severe |
|---|---|---|---|---|---|
| VC (%predicted) | >80 | >80 | >80 | ↓ | ↓↓ |
| $FEV_1/FVC$ | >75 | 60-75 | 40-60 | <40 | <40 |
| MVV | >80 | 65-80 | 45-65 | 30-45 | <30 |
| RV(%predicted) | 80-120 | 120-150 | 150-175 | >200 | >200 |

In the home for monitoring of lung function is recommended to determine the peak expiratory flow (PEF) using the peak flow. This method belongs to an early screening-diagnosis of bronchial obstruction – one of the most important biomarkers of asthma and COPD. The below-mentioned patient after necessary training the health worker is able to apply independently for objective control of their condition and timely treatment to the doctor in case of its functional state. Note, however, that the below-mentioned will never be able to replace spirometry, because peak flow can be used to identify only the peak expiratory flow (PEF). Method of measurement of PEF:

Measurement of PEF is once in the morning before bronchodilator. The procedure is repeated three times and the highest peak expiratory flow (PEF) is recorded. This can be compared with a nomogram that shows the patient's sex, age and weight, and plotted on a chart to show the progress or response to treatment. Measurements should be made 2-3 weeks (minimum 1 week). If the measurement result is classified as proper or best for the period of measurement the indicator PEF

(the so-called "index PEF-variability or PSV-lability respiratory tract") <80%, you should change basic therapy (Fig. 19). (GINA, 2002)
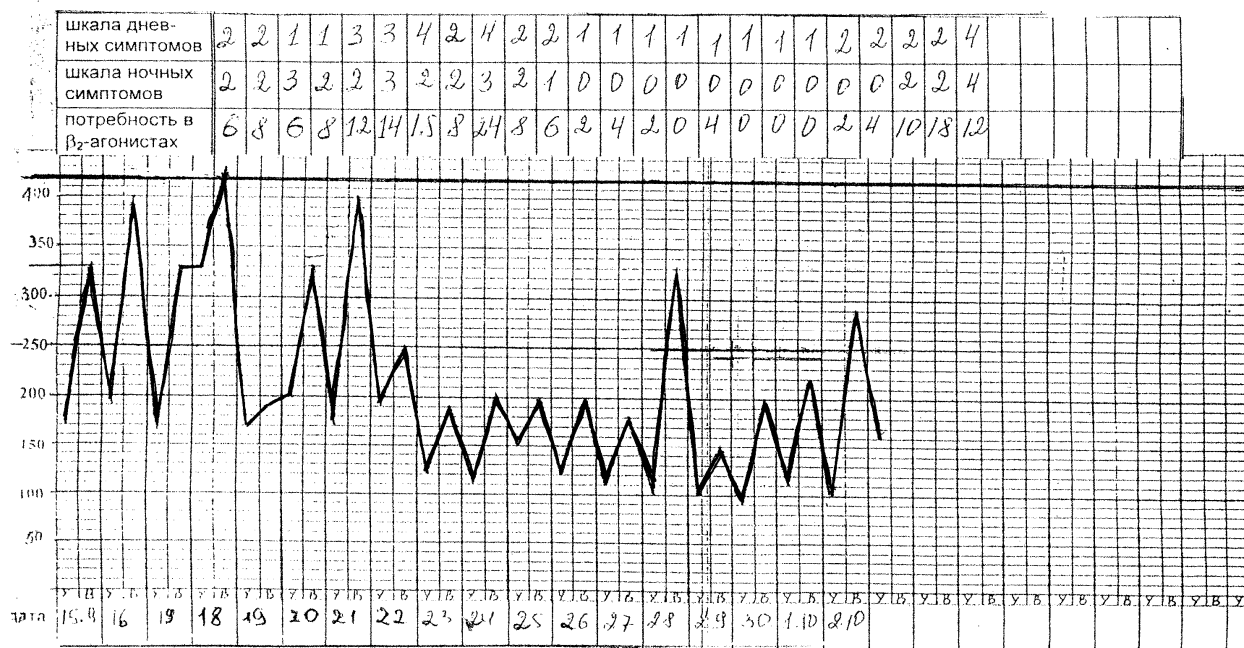


A.
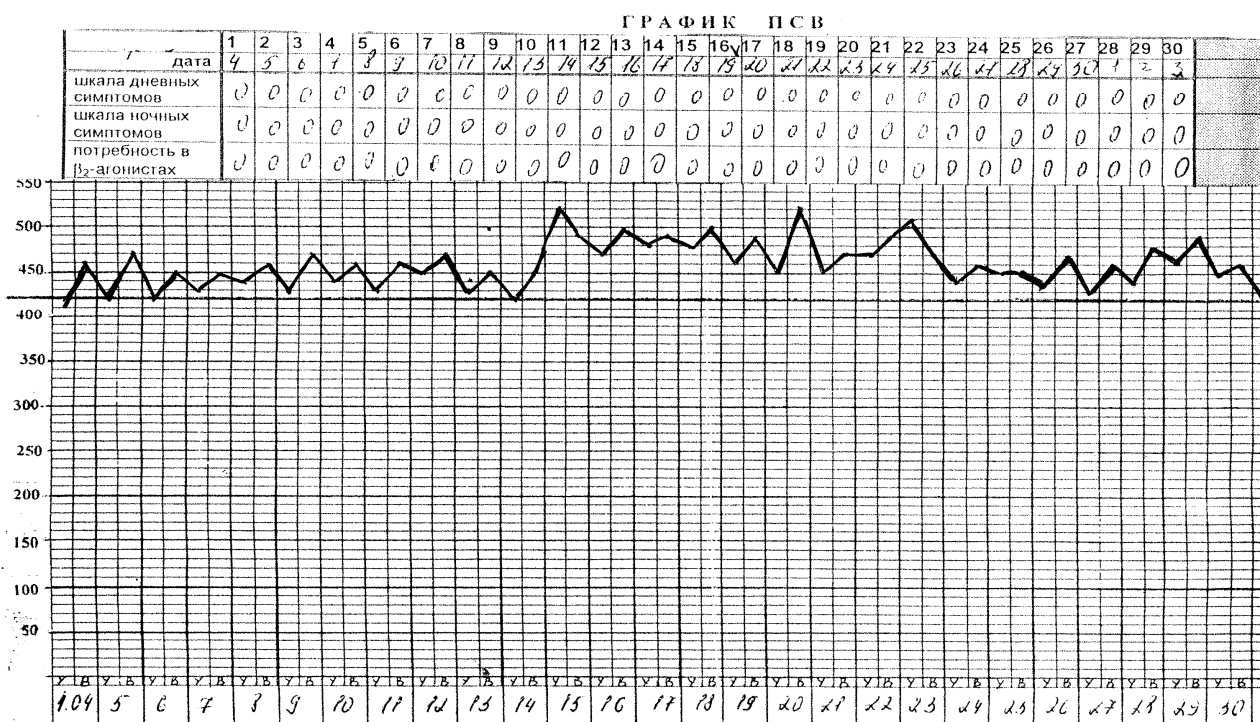


B.

Fig.19. Monitoring of PEF.

A. Initial level of PEF is low (55%), index PEF-variability is very high (60%, Normally -20%).
B. After change of basic therapy PEF is 110%, index PEF-variability is 7%.

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Airflow obstruction syndrome

Airflow obstruction syndrome — syndrome of bronchial patency alteration, in chronic course of disease manifested by severe productive, rarely non-productive cough and also emphysema development. In acute bronchial obstruction signs of acute respiratory insufficiency occur, that is assessed as emergency situation.

*Causes.* In airflow obstruction syndrome the appearing changes are concerned predominantly small bronchi. Inflammation and edema of bronchial mucous (chronic bronchitis, allergic reactions), bronchospasm, usually with edema of bronchial mucous (e.g. bronchial asthma) are the most frequent causes, rarely – diffuse peribronchial fibrosis, compressing the bronchi from the outside, and also mechanical bronchi compression from the outside in emphysema (on expiration intraalveolar pressure increases resulting in small bronchi collapse).

*Symptoms and signs.* The main symptoms are cough, expiratory dyspnea. In lingering course of airflow obstruction syndrome it is necessary to mark the particular clinical meaning of cough not only as the symptom and sign of bronchi disorder but as factor, which worsened pulmonary parenchyma lesion itself.

There are few outward manifestations of abnormality in patients with mild airflow obstruction. However, as the process becomes more severe, the patient's distress becomes evident from

- the labored breathing,
- the use of the accessory muscles on respiration,
- inspiratory retraction of the supraclavicular fossae and lower interspaces, and
- the positioning of the chest near total lung capacity.

When the patient is asked to empty the lungs forcibly and completely, it is evident that expiration is prolonged and difficult, with pulmonary emptying incomplete. The degree of expiratory slowing may be estimated by measuring the forced expiratory time (which normally measures 4 seconds or less), with a watch and a stethoscope. Auscultation over the larynx permits accurate determination of the

end of expiration. Sounds are audible at this site at the low airflows occurring near residual volume, when breath sounds are no longer audible over the lungs.

On auscultation: harsh vesicular breath sounds with prolonged expiration, wheezes and rhonchi, which presence gives an opportunity to define the level of obstruction. Abnormality of inhalation and exhalation proportions and rough prolonged exhalation appearance is the significant auscultative index of bronchial obstruction. It must be remembered that when airway obstruction is very severe, wheezes may completely disappear, usually with a marked decrease in the intensity of breath sounds. The reappearance of wheezes indicates the response of the patient to treatment, with diminution in the severity of airway obstruction.

In prominent acute bronchial obstruction the picture of —silent lung appears, when the bronchial patency is altered so much that breath sounds aren't listened at all.

Functional tests: $FEV_1$ <80 % predicted, $FEV_1$ / $FVC$<70% predicted and FEF25-75% decrease.

Syndrome of hyperinflated lung closely linked with airflow obstruction syndrome is discussed below.

Respiratory deficiency syndrome.

Diagnostics of respiratory insufficiency presence is an important and mandatory moment in estimation of respiratory organs pathology. Respiratory insufficiency (RI) is a condition when maintenance normal gas composition of arterial blood is not supported or supported due to abnormal (heavy) work of external respiration apparatus that leads to decrease of organism functional capacities.

Maintenance of normal gas exchange in the lungs is possible, as it was already stated, only on condition of sharp interconnection of three components:

1. ventilation
2. gases diffusion through alveolocapillary membrane and
3. capillary blood perfusion in the lungs.

That is why any pathologic processes in the organism or unfavorable environment factors (for example, decrease of oxygen partial pressure in atmospheric air) that influence at least one of these components, may be the reasons of RI.

Two RI groups are distinguished:

group I with predominant lesion of extrapulmonary mechanisms;

group II with predominant lesion of pulmonary mechanisms: ventilation, perfusion and alveolocapillary gases diffusion.

*Main reasons and mechanisms of respiratory insufficiency, characteristic of dyspnea*. The following pathologic conditions may be included in group I of RI:

1. disturbance of central regulation of respiration (traumatic, metabolic, circulatory, toxic, infectious and other brain lesions);

2. respiratory muscles lesion (trauma, intoxication, myalgia, myodystrophy, etc.) or peripheral nerves lesion (poliomyelitis, polyradiculoneuritis, tetanus);

3. chest lesion (kyphoscoliosis, deformations, trauma, etc.). Group II of RI includes the following pathologic conditions:

4. obstruction of large respiratory tracts (tumor, foreign body, dyskinesia of membranous part of the trachea);

5. obstruction of small respiratory tracts (bronchial asthma, bronchiolitis);

6. disturbance of alveolar tissue restriction (interstitial edema, pleurisy, pneumothorax, hydrothorax, etc.);

7. reduction of pulmonary tissue (massive inflammation, lung resection, atelectases);

8. alveolocapillary membrane thickening (interstitial edema, pulmonary tissue inflammation, pulmonary fibrosis, etc.);

9. pulmonary circulation lesions (blood congestion in the lesser circulation circle in left ventricular cardiac failure, hypovolemia, etc.);

10. disturbance of ventilation - perfusion proportions (chronic obstructive bronchitis, pneumonia, pulmonary artery branches thromboembolism, etc.).

Two forms of RI are distinguished depending on predominant lesion of three respiratory system components (ventilation, perfusion and diffusion).

In *ventilation form of RI* external respiration lesion prevails which is accompanied by development of hypoxemia as well as hypercapnia.

In the so-called *parenchymatous form of RI* disturbances of gases diffusion, capillary blood perfusion or perfusion - ventilation proportions prevail. This form of RI leads to development of hypoxemia whereas hypercapnia is not usually observed.

Attention should be paid to the fact that the majority of pulmonary pathologic processes are accompanied by disturbance of several gas exchange mechanisms. For example, in pneumonia restriction lesions are mainly observed, obstructive lesions are somewhat less frequent, gases diffusion through alveolocapillary membrane decreases, number of functioning alveoli lessens, etc.

In chronic obstructive bronchitis alongside with pronounced obstructive lesions disturbances of ventilation-perfusion proportions are observed due to significant unevenness of pulmonary ventilation, etc.

*Dyspnea* caused by respiratory center irritation and having a very variable character is the most important symptom of RI. The type of dyspnea may be more distinctly defined in small respiratory tracts obstruction (expiratory dyspnea) and in restrictive disturbances (inspiratory dyspnea).

The traditional principle clinical classification RI is its division into degrees of severity depending on the tolerance to physical activity and severity of dyspnea (by A. G. Dembo, 1957; Schick, L. L., Kanaev N. N., 1980). Depending on the level of physical activity in which there is shortness of breath, in our country it is customary to distinguish three degrees of severity RI. When I grade dyspnea occurs with increased physical activity; at II degree of dyspnea observed in moderate daily physical activity; for III the severity of chronic pulmonary disease characterized by shortness of breath at rest.

Almost all existing definitions of RI clearly indicate a gas composition of arterial blood as a possible indicator of the degree days. The division of chronic respiratory insufficiency in severity depends on the levels of the two most important indicators of gas composition of arterial blood – $PaO_2$ and $SaO_2$ (saturation of

hemoglobin with oxygen). This approach to the classification of the severity RI presented in table 6. (Avdeev S. N., 2004).

Table 6.

Classification of severity of respiratory insufficiency (S. N. Avdeev, 2004)

| Degree of RI | $P_aO_2$, мм Hg | $SaO_2$, % |
|---|---|---|
| Norm | >80 | >95 |
| I | 60–79 | 90–94 |
| II | 40–59 | 75–89 |
| III | <40 | <75 |

PaO2 – partial tension of oxygen in arterial blood; SaO2 – saturation of arterial blood hemoglobin with oxygen

***Main manifestations of respiratory insufficiency***. Among a large number of respiratory insufficiency signs the following are most significant in clinical practice:

1. dyspnea;

2. central (diffuse) cyanosis;

3. enhanced work of respiratory muscles;

4. intensification of circulation (tachycardia, minute volume increase);

5. change of respiratory volumes and capacities.

In restrictive RI VC and MPV predominantly decrease, FVC1 is slightly changed, and in obstructive RI FVC1 and MPV significantly decrease. In practice combined RI is often met where pulmonary tissue elasticity disturbances as well as respiratory tracts passage lesions are observed.

**Blood tests**

Venous blood samples taken for automated blood counts may provide major clues or confirmation of a suspected respiratory disease. A high haemoglobin concentration may be a reflection of polycythaemia, either primary or secondary and a low haemoglobin may cause breathlessness. The total white cell count may be

elevated in a range of acute bacterial infections and its subsequent fall is a reflection of successful therapy. Normal or low white cell counts are found in mycoplasma or viral infections. Eosinophilia suggests an allergic component or parasitic infection.

A range of serological tests that depend on agglutination, precipitation and complement fixation provide evidence of the presence in the patient's serum of specific antibodies against viral, bacterial, fungal, protozoal and helminthic infections. Samples of blood should be tested on admission and repeated after 10- 14 days to detect a rising titer.

Radioallergoabsorbent tests (RASTs) on venous blood are an alternative to skin-prick tests as a method of identifying specific IgE antibodies.

### Sputum test

A **sputum culture** is a test to detect and identify bacteria or fungi that infect the lungs or breathing passages. Sputum is a thick fluid produced in the lungs and in the adjacent airways. A sample of sputum is placed in a sterile container and sent to the laboratory for testing. Sampling may be performed by sputum being expectorated (produced by coughing), induced (saline is sprayed in the lungs to induce sputum production), or taken via an endotracheal tube with a protected specimen brush (commonly used on patients on respirators) in an intensive care setting. For selected organisms such as Cytomegalovirus or "Pneumocystis jiroveci" in specific clinical settings (immunocompromised patients) a bronchoalveolar lavage might be taken by an experienced pneumologist. If no bacteria or fungi grow, the culture is negative. If organisms that can cause the infection (Pathogenicity organisms) grow, the culture is positive. The type of bacterium or fungus is identified by microscopy, colony morphology and biochemical tests of bacterial growth.

If bacteria or fungi that can cause infection grow in the culture, other tests can determine which antimicrobial agent will most effectively treat the infection. This is called susceptibility or sensitivity testing.

*Bacterial culture*

A portion of the sputum is smeared on a microscope slide for a Gram stain. Another portion is spread over the surface ofseveral different types of culture plates, and placed in an incubator at body temperature for one to two days.

A Gram stain is done by staining the slide with purple and red stains, then examining it under a microscope. Gramstaining checks that the specimen does not contain saliva or material from the mouth. If many epithelial (skin) cells andfew white blood cells are seen, the specimen is not pure sputum and is not adequate for culture. Depending on laboratorypolicy, the specimen may be rejected and a new specimen requested. If many white blood cells and bacteria of one typeare seen, this is an early confirmation of infection. The color of stain picked up by the bacteria (purple or red), their shape(such as round or rectangular), and their size provide valuable clues as to their identity and helps the physician predictwhat antibiotics might work best before the entire test is completed. Bacteria that stain purple are called gram-positive; those that stain red are called gram-negative.

During incubation, bacteria present in the sputum sample multiply and will appear on the plates as visible colonies. Thebacteria are identified by the appearance of their colonies, by the results of biochemical tests, and through a Gram stainof part of a colony.

A sensitivity test, also called antibiotic susceptibility test, is also done. The bacteria are tested against differentantibiotics to determine which will treat the infection by killing the bacteria.

The initial result of the Gram stain is available the same day, or in less than an hour if requested by the physician. Anearly report, known as a preliminary report, is usually available after one day. This report will tell if any bacteria have beenfound yet, and if so, their Gram stain appearance—for example, a gram-negative rod, or a gram-positive cocci. The finalreport, usually available in one to three days, includes complete identification and an estimate of the quantity of thebacteria and a list of the antibiotics to which they are sensitive.

### *Fungal culture*

To look for mold or yeast, a fungal culture is done. The sputum sample is spread on special culture plates that willencourage the growth of mold and yeast. Different b iochemical tests and stains are used to identify molds and yeast.Cultures for fungi ma y take several weeks.

### *Viral culture*

Viruses are a common cause of pneumonia. For a viral culture, sputum is mixed with commercially
prepared animal cellsin a test tube. Characteristic changes to the cells caused by the g rowing virus help identify the virus. The time tocomplete a viral culture varies with th e type of virus. It may take from several days to several weeks.

### *Special procedures*

**Tuberculosis** is caused by a slowgrowing bacteria called *Mycobacterium tuberc ulosis*. Because it does not easily growusing routine culture methods, special procedu res are used to grow and identify this bacteria. When a sputum sample fortuberculosis first comes into the laboratory, a small portion of the sputum is smeared on a microsc ope slide and stainedwith a special stain, called an acid
fast stain. The stained sputum is examined under a microscope for tuberculosisorgani sms, which pickup the stain, making them visible. This smear is a rapid screen for the organism, and allows thephysician to receive a preliminary report within 24 hours.

To culture for tuberculosis, portions of the sputum are spread on and placed into special culture plates and tubes of broththat promote the growth of the organism. Gro wth in broth is faster than growth on culture plates. Instruments are availablethat can detect growth in broth, speeding the process even further. Growth and identification may take two to four weeks.

Other microorganisms that cause various types of lower respiratory tract infectio ns also require special cultureprocedures to grow and identify. *Mycoplasma pneumon ia* causes a mild to moderate form of pneumonia, commonly calledwalking pneumoni

a; *Bordetella pertussis* causes <u>whooping cough</u>; *Legionella pneumophila*, Legionnaire's disease;*Chlamydia pneumoniae*, an at ypical pneumonia; and *Chlamydia psittaci*, <u>parrot fever</u>.

*Pneumocystis carinii* causes pneumonia in people with weakened immune syste ms, such as people with <u>AIDS</u>. Thisorganism does not grow in culture. Special stains are done on sputum when pneumonia caused by this organism issuspected. The diagosis is based on the results of these stains, the patient's symptoms, and medical histor y.

Sputum culture is also called sputum culture and sensitivity.

It is possible that sputum cultures will eventually be replaced in the diagnosis of tuberculosis by newer moleculartechniques. These advanced methods speed the diagn ostic process as well as improve its accuracy. As of late 2002, fourmolecular techniqu es are increasingly used in laboratories around the world to diagnose TB. They includ e polymerasechain reaction to detect mycobacterial DNA in patient specimens; nuclei c acid probes to identify mycobacteria in culture;restriction fragment length polymor phism analysis to compare different strains of TB for epidemiological studies; andgen etic-based susceptibility testing to identify drug-resistant strains of mycobacteria.

**QUESTIONS FOR TEST CONTROL TO ENGAGE THE THEME "QUESTIONING, GENERAL EXAMINATION, INSPECTION AND PALPATION OF THE PATIENT WITH RESPIRATORY DISEASES":**

1. GIVE THE NAME OF THE NEXT FORM OF THE CHEST:
Thorax elongated, narrow, flat. Over - and subclavian fossae are distinctly pronounced. The epigastric angle is acute. The ribs in the lateral parts directed more vertically. The muscles are poorly developed.
a) paralytic
b) emphysematous
c<u>) asthenic</u>
d) hypersthenic
e) normasthenic

2. GIVE THE NAME OF THE NEXT FORM OF THE CHEST:

Increasing the transverse and anteroposterior size of the chest, short neck. Supraclavicular fossa smooth. The epigastric angle is obtuse. The direction of the horizontal ribs. Intercostal spaces are wide, breathing in zadanija departments indicated their retraction. The scapula firmly against the thorax. The breath is actively involved supporting musculature

a) paralytic

b) emphysematous

c) asthenic

d) hypersthenic

e) normasthenic

3. GIVE THE NAME OF THE NEXT FORM OF THE CHEST:

Wide but short ribcage. The ratio of anterior–posterior to lateral size of about 1.0. Supraclavicular fossa smooth. The epigastric angle is obtuse. The direction of the ribs are almost horizontal. Intercostal spaces narrow, their retractions when breathing is not marked. The scapula firmly against the thorax.

a) paralytic

b) emphysematous

c) asthenic

d) hypersthenic

e) normasthenic

4. GIVE THE NAME OF THE NEXT FORM OF THE CHEST:

Thorax elongated, narrow and flat. The ratio of anterior–posterior and lateral sizes close to 0.5. Over - and subclavian fossae are distinctly pronounced, but is located asymmetrically. The epigastric angle is acute. The ribs are placed obliquely. Intercostal spaces are wide Pronounced atrophy of the muscles of the chest.

a) paralytic

b) emphysematous

c) asthenic

d) hypersthenic

e) normasthenic

5. GIVE THE NAME OF THE NEXT FORM OF THE CHEST:

When inspecting the thorax, the ratio of anteroposterior to transverse size of about 0.7. Supraclavicular fossa is weakly developed. An epigastric angle of a straight line. The ribs in the lateral parts have moderately oblique direction. The scapula firmly

against the thorax.

a) paralytic

b) emphysematous

c) asthenic

d) hypersthenic

e) normasthenic

6. WHAT IS THE NAME OF SPINAL CURVATURE FORWARD?

a) <u>lordosis</u>

b) scoliosis

c) kyphosis

d) kyphoscoliosis

7. WHAT IS THE NAME OF SPINAL CURVATURE IN A LATERAL DIRECTION?

a) lordosis

b) <u>scoliosis</u>

c) kyphosis

d) kyphoscoliosis

8. HOW IS CALLED THE CURVATURE OF THE SPINE AGO WITH THE FORMATION OF THE HUMP?

a) lordosis

b) scoliosis

c) <u>kyphosis</u>

d) kyphoscoliosis

9. WHAT IS THE NAME OF SPINAL CURVATURE IN A LATERAL DIRECTION AND BACK?

a) lordosis

b) scoliosis

c) kyphosis

d) <u>kyphoscoliosis</u>

10. SPECIFY THE MOST TYPICAL CHANGES OF THORAX IN PATIENTS WITH PNEUMOTHORAX:

a) decrease half of thorax, its retraction and backlog in breath

b) the gap in breathing and the increase of the affected part of the chest

C) only the lag in the breathing of the affected part of the chest

g) hypersthenic chest

d) an increase in anteroposterior and transverse dimensions of the thorax symmetric, retractions of intercostal spaces in inferolateral departments on both sides when breathing, bulging supraclavicular fossae, epigastric angle > 90 degrees

## 11. SPECIFY THE MOST TYPICAL CHANGES OF THORAX IN PATIENTS WITH EMPHYSEMA:

a) decrease half of thorax, its retraction and backlog in breath

b) the gap in breathing and the increase of the affected part of the chest and smoothing
intercostal spaces

C) only the lag in the breathing of the affected part of the chest

g) hypersthenic chest

d) an increase in anteroposterior and transverse dimensions of the thorax symmetric, retractions of intercostal spaces in inferolateral departments on both sides when breathing, bulging supraclavicular fossae, epigastric angle > 90 degrees

## 12. SPECIFY THE MOST TYPICAL CHANGES OF A THORAX FOR PATIENTS WITH OBTURATIVE ATELECTASIS:

a) decrease half of thorax, its retraction and backlog in breath

b) the gap in breathing and the increase of the affected part of the chest and smoothing
intercostal spaces

C) only the lag in the breathing of the affected part of the chest

g) hypersthenic chest

d) an increase in anteroposterior and transverse dimensions of the chest are symmetrical,
retractions of intercostal spaces in inferolateral departments on both sides when breathing, bulging supraclavicular fossae, epigastric angle > 90 degrees

## 13. SPECIFY THE MOST TYPICAL CHANGES OF A THORAX FOR PATIENTS WITH HYDROTHORAX:

a) decrease half of thorax, its retraction and backlog in breath

b) the gap in breathing and the increase of the affected part of the chest and smoothing of intercostal spaces

C) only the lag in the breathing of the affected part of the chest

g) hypersthenic chest

d) an increase in anteroposterior and transverse dimensions of the thorax symmetric, retractions of intercostal spaces in inferolateral departments on both sides when breathing, bulging supraclavicular fossae, epigastric angle > 90 degrees

14. DETERMINE the type of BREATHING: the patient is a feeling of incompleteness or obstruction of the exhalation; the exhalation with the participation of auxiliary muscles.

a) respiration of Cheyne-Stokes

b) expiratory dyspnea

b) stridor

d) inspiratory dyspnea

d) Kussmaul breathing

15. DETERMINE the type of BREATHING: Periodic breathing with changing amplitude of the respiratory movements and long periods of apnea.

a) respiration of Cheyne-Stokes

b) expiratory dyspnea

b) stridor

d) inspiratory dyspnea

d) Kussmaul breathing

16. DETERMINE the type of BREATHING: Deep noisy breath in patient in a coma

a) respiration of Cheyne-Stokes

b) expiratory dyspnea

c) stridor

d) inspiratory dyspnea

e) Kussmaul breathing

17. DETERMINE THE TYPE OF BREATHING: THE patient dramatically difficult to inhale and exhale; jugular veins swollen. Inspiratory and expiratory breathing is noisy.

a) respiration of Cheyne-Stokes

b) expiratory dyspnea

c) stridor

d) inspiratory dyspnea

e) Kussmaul breathing

18. HOW TO CHANGE THE VOCAL FREMITUS IN  SYNDROME OF AIR CAVITY?

a) no change

b) weakening of one side

c) increase on both sides

d) the weakening of both sides

e) increase one side

19. HOW TO CHANGE VOCAL FREMITUS IN EMPHYSEMA?

a) no change

b) weakening of one side

b) increase on both sides

d) the weakening of both sides

d) increase one side

20. HOW TO CHANGE VOCAL FREMITUS IN CANCER OF THE LARGE BRONCHUS WITH THE OBTURATIVE ATELECTASIS?

a) no change

b) weakening of one side

b) increase on both sides

d) the weakening of both sides

d) increase one side

21. HOW TO CHANGE VOCAL FREMITUS  IN THE PLEURAL EFFUSION SYNDROME?

a) no change

b) weakening of one side

b) increase on both sides

d) the weakening of both sides

d) increase one side

22. HOW TO CHANGE VOCAL FREMITUS  IN  LOBAR PNEUMONIA?

a) no change

b) weakening of one side

b) increase on both sides

d) the weakening of both sides

<u>d) increase one side</u>

23. HOW TO CHANGE VOCAL FREMITUS WITH PNEUMOTHORAX, NOT COMMUNICATING WITH THE BRONCHUS?

a) no change

b) weakening of one side

b) the gain on both sides

d) the weakening of both sides

d) increase one side

**QUESTIONS FOR TEST CONTROL TO ENGAGE THE THEME "COMPARATIVE PERCUSSION OF THE LUNGS. PULMONARY SYNDROMES":**

1. WHAT ARE THE 2 TYPES OF PERCUSSION SOUNDS APPEAR WHEN THE PLEURAL EFFUSE SYNDROME?

<u>a. dull sound</u>

b. clear lung sounds

c. tympanic sound

d. dullness with tympanic shade

e. hyperresonant sound

2. WHAT PERCUSSION SOUND IS CHARACTERISTIC OF OBTURATIVE ATELECTASIS?

<u>a. dull sound</u>

b. clear lung sounds

c. tympanic sound

d. dulled-tympanic sound

e.  hyperresonant sound sound

 3. WHAT PERCUSSION SOUND OCCURS OVER A LARGE CAVITY IN THE LUNG?

 a. femoral sound

 b. clear lung sounds

c. tympanic (metal) sound

d. dullness with tympanic shade

e. hyperresonant sound

4. WHAT PERCUSSION SOUND IS CHARACTERISTIC FOR INFLAMMATORY INFILTRATION OF LUNG TISSUE ( HEIGHT STAGE OF PNEUMONIA)?

a. dull (femoral) sound

b. clear lung sounds

c. tympanic sound

d. dullness with tympanic shade

e. hyperresonant sound

5. WHAT PERCUSSION SOUND IS GENERATED AT THE INITIAL STAGE OF THE INFLAMMATORY INFILTRATION OF LUNG TISSUE (STAGE 1 OF PNEUMONIA)?

a. absolutely dull (femoral)

b. clear lung sounds

c. tympanic sound

d. dull sound

e. hyperresonant sound

6. WHAT PERCUSSION SOUND ABOVE THE PLEURAL THICKENING?

a. dull sound

b. clear lung sounds

c. tympanic sound

d. dullness with tympanic shade

e. hyperresonant sound

7. WHAT PERCUSSION SOUND IS CHARACTERISTIC FOR COMPRESSION ATELECTASIS?

a. dull sound

b. clear lung sounds

c. tympanic sound

d. dulled-tympanic sound

e. hyperresonant sound

8. WHAT PERCUSSION SOUND IS CHARACTERISTIC FOR EMPHYSEMA?

a. dull sound

b. clear lung sounds

c. tympanic sound

d. dullness with tympanic shade

e. hyperresonant sound

9. WHAT PERCUSSION SOUND IS CHARACTERISTIC FOR PNEUMOTHORAX?

a. dull (femoral) or blunt sound

b. clear lung sounds

c. tympanic sound

d. dullness with tympanic shade

e. hyperresonant sound

10. WHAT PERCUSSION SOUND IS CHARACTERISTIC FOR THE FINAL STAGE OF THE INFLAMMATORY INFILTRATION  OF LUNG (RESOLUTION STAGE OF PNEUMONIA)?

a. dull (femoral)

b. clear lung sounds

c. tympanic sound

d. blunt sound

e. hyperresonant sound

11. WHAT SYNDROMES ARE CHARACTERIZED BY "SHORTENING" OF PERCUSSION SOUNDS?

a. the syndrome of inflamantory of  lung tissue

b. the syndrome of pneumothorax not communicating with a bronchus

c. syndrome of hydrothorax

d. the syndrome of pneumothorax communicating with a bronchus

e. syndrome of the air cavity in the lung

f. syndrome of compression atelectasis

g. the syndrome of obturative atelectasis

h. the syndrome of pulmonary fibrosis

i. the syndrome of emphysema

12.  WHAT SYNDROMES ARE CHARACTERIZED BY TYMPANIC PERCUSSION SOUND?

a. the inflamantory infiltration syndrome

b. the syndrome of pneumothorax not communicating with a bronchus

c. syndrome of hydrothorax

d. the syndrome of pneumothorax communicating with a bronchus

e. syndrome of the air cavity in the lung

f. the syndrome of obturative atelectasis

g. the syndrome of pulmonary fibrosis

h. the syndrome of emphysema


13. WHAT SYNDROME IS CHARACTERIZED BY DULL-TYMPANIC PERCUSSION SOUND?

a. the inflamantory infiltration syndrome

b. the syndrome of pneumothorax not communicating with a bronchus

c. syndrome of hydrothorax

d. the syndrome of pneumothorax communicating with a bronchus

e. the syndrome of compressive  atelectasis

f. the syndrome of obturative atelectasis

g. the syndrome of pulmonary fibrosis

h. the syndrome of emphysema


14. FOR ANY OF THE SYNDROMES NOT CHARACTERISTIC METALLIC PERCUSSION SOUND?

a. the inflamantory infiltration syndrome

b. the syndrome of pneumothorax not communicating with a bronchus

c. syndrome of hydrothorax

d. the syndrome of pneumothorax communicating with a bronchus

e. the syndrome of compressive  atelectasis

f. the syndrome of obturative atelectasis

g. the large air cavity syndrome

h. the syndrome of emphysema


15. IN CASE OF IDENTIFICATION  THE INCREASE OF VOICE FREMITUS OVER THE AFFECTED SIDE OF THE CHEST WHAT PERCUSSION SOUND CAN I EXPECT?


a. dull percussion sound

b. blunt percussion sound

c. dull-tympanic percussion sound

d. tympanic percussion sound

e. metal percussion sound

f. hyperresonanted percussion sound


16. IN CASE OF IDENTIFICATION THE WEAKENED VOICE FREMITUS OVER THE AFFECTED SIDE OF THE CHEST WHAT PERCUSSION SOUND CAN I EXPECT?

a. dull or blunt percussion sound

b. dulled-tympanic percussion sound

c. tympanic percussion sound

d. metal percussion sound

e. hyperresonanted percussion sound


17. WHAT SYNDROME IS CHARACTERIZED BY HYPERRESONANTED PERCUSSION SOUND?

a. the inflamantory infiltration syndrome

b. pneumothorax, not communicating with a bronchus

c. the syndrome of hydrothorax

d. syndrome pneumothorax communicating with a bronchus

e. the syndrome of large smooth-walled cavities in the lung

f. the syndrome of compression atelectasis

g. the syndrome of obturative atelectasis

h. emphisema

18. WHAT SYNDROMES ARE CHARACTERIZED BY A DULL OR BLUNT PERCUSSION SOUND?

a. the inflamantory infiltration syndrome

b. pneumothorax, not communicating with a bronchus

c. the syndrome of hydrothorax

d. syndrome pneumothorax communicating with a bronchus

e. the syndrome of large smooth-walled cavities in the lung

f. the syndrome of compression atelectasis

g. the syndrome of obturative atelectasis

h. emphisema

19. IN THE CASE OF "ABSENCE" IN A PATIENT VOICE FREMITUS OVER THE AFFECTED SIDE OF THE CHEST, WHAT PERCUSSION SOUND CAN I EXPECT?

a. dull percussion sound

b. dulled-tympanic percussion sound

c. tympanic percussion sound

d. metal percussion sound

e. hyperresonanted percussion sound


20. CHEK 4 SPECIFIC CHARACTERISTIC FEATURE OF THE SYNDROME OF COMPRESSION ATELECTASIS:

a. the weakening voice fremitus

b. respiratory depression

c.  pathological bronchial breath sounds

d. increase voice fremitus

e. tympanic percussion sound

f. clear lung sounds

g. dulled-tympanic percussion sound

h. croase crackles

i. wheezes

k. crepitus (fine crackles)

l. pleural rub


21. SPECIFY 5 FEATURES, NOT CHARACTERISTIC FOR SYNDROME OF OBTURATIVE ATELECTASIS:

a. pathological bronchial breath sounds

b. the weakening voice fremitus

c. increase voice fremitus

d. tympanic percussion sound

e. deminished vesicular breath sounds

f. weakening of bronchophony

h. the increase affected half of the thorax

i. pleural rub

22. 5 SPECIFIC CHARACTERISTICS COMMON TO THE SYNDROME OF EMPHYSEMA:

a. the weakening voice fremitus

b. deminished vesicular breath sounds

c. pathological bronchial breath sounds

e. increase voice  fremitus

f. hyperresonant percussion sound

g. funnel chest

h. barrel chest

i. pathological decrease in lung volume

j. pathological increase in lung volume


23. SPECIFIC CHARACTERISTICS COMMON TO THE PLEURAL EFFUSION SYNDROME:

a. the symmetry of participation in the breathing of both halves of the chest

b. the lag in the breathing of the affected part of the chest

c. the weakening voice fremitus

d. the weakening or disappearance of breath

e. pathological bronchial breath sounds

f. tympanic percussion sound

g. blunt or dull percussion sound

h. pleural  rub

24. 4 SPECIFIC  SYMPTOMS, NOT CHARACTERISTIC FOR SYNDROME OF PNEUMOTHORAX THAT ARE NOT COMMUNICATING WITH A BRONCHUS:

a. the appearance of pathological bronchial breathing

b. the weakening voice fremitus

c. tympanic percussion sound

d. deminished vesicular breath sounds

e. strengthen voice  fremitus

f. dull percussion sound

g. the symmetry of participation in the breathing of both halves of the chest

h. the lag in the breathing of the affected part of the chest


25. 4 SPECIFIC  CHARACTERISTIC FEATURES OF THE INFLAMMATORY

INFILTRATION SYNDROME:
a. the weakening voice fremitus
b. pathological bronchial breathing
c. increased voice  fremitus
d. tympanic percussion sound
e. hyperresonanted percussion sound
f. wheezes
g. the symmetry of participation in the breathing of both halves of the chest
h. the lag in the breathing of the affected part of the chest
i. fine or croake crackles


26. 3 INDICATE SYMPTOM, NOT CHARACTERISTIC FOR SYNDROME OF THE AIR CAVITY connecting with bronchus:
a. the weakening voice fremitus
b. the appearance of pathological bronchial breathing
c. increased voice  fremitus
d. tympanic percussion sound
e. dull percussion sound
f. crackles
h. pleural  rub


27. SPECIFY 5 FEATURES NOT CHARACTERISTIC FOR THE SYNDROME OF EMPHYSEMA:
a. bronchial obstruction by tumor or foreign body
b. pathological decrease in elastic properties of lungs with the development of "air traps" and pathological lung hyperinflation
c. appearance in the pleural cavity fluid
d. appearance in the pleural cavity air
e. the appearance of air in the lung cavity in the case of the emptying of lung abscess or tuberculosis caverns
f. inflammatory infiltration syndrome

28. WHAT ARE 5 SYNDROMES CHARACTERIZED BY PATHOLOGICAL BRONCHIAL BREATH SOUNDS?:
a. syndrome of  pleural effusion
b. the syndrome of pneumothorax communicating with a bronchus
c. pneumothorax, not communicating with a bronchus

d. the syndrome of obstructive atelectasis

e. the syndrome of compression atelectasis

f. the syndrome of the air cavity

g. the syndrome of pulmonary fibrosis

h. syndrome emphysema

i. inflammatory infiltration syndrome


28. WHAT ARE 2 SYNDROMES CHARACTERIZED BY FINE CRACKLES?:

a. syndrome of pleural effusion

b. pneumothorax

c. syndrome of compression atelectasis

d. the syndrome of the air cavity

e. the syndrome of emphysema

f. the inflamatory infiltration syndrome


29. WHAT ARE THE CAUSE OF COMPRESSION ATELECTASIS:

a. obturation (closing of the lumen) of the bronchus by tumor or foreign body

b. pneumonia

c. the pleural effusion syndrome

d.decrease in the elastic properties of the small bronchi and bronchioles with the development of "expiratory closure of the small airways"

e. infiltrative tuberculosis

f. partial replacement of the alveoli by connective tissue


30. SPECIFY THE CAUSE OF OBTURATIVE ATELECTASIS:

a. the pleural effusion syndrome

b. pneumothorax

c. the obstruction of the bronchus by tumor or foreign body

d. pneumonia

e. infiltrative tuberculosis

f. "air trap" due to "expiratory closure of the small airways"

g. partial replacement of the alveoli by connective tissue


31. SPECIFY 2 CAUSES OF INFLAMMATORY INFILTRATION SYNDROME:

a. the pleural effusion syndrome

b. infiltrative tuberculosis

c. pneumothorax

d. the obturation (closing of the lumen) of the bronchus by tumor or foreign body

e. pneumonia

f. air trap due to the "expiratory closure of the small Airways"

32. SPECIFY 2 CAUSES OF THE SYNDROME OF THE AIR CAVITY:

a. the pleural effusion syndrome

b. pneumothorax

c. obturation (closing of the lumen) of the bronchus by tumor or foreign body

d. pneumonia

e. infiltrative tuberculosis

f. tuberculous cavity

g. air trap due to the "expiratory closure of the small Airways"

h. partial replacement of the alveoli by connective tissue

i. lung abscess


33. 2 FEATURES OF BRONCHIAL OBSTRUCTION ACCORDING TO THE SPIROMETRY

a. the decline of VC

b. reduction FEV1/FVC<70%

c. the decline of FEV1<80% from proper

d. FEV1/FVC>70%


34. 2 FEATURES OF RESTRICTIVE DISORDERS OF RESPIRATORY FUNCTION ACCORDING TO SPIROMETRY

a. the decline of VC

b. reduction FEV1/FVC<70%

c. the decline of FEV1<80% from proper

d. FEV1/FVC>70%


35. CHOOSE THE CORRECT SYMPTOM OF RESPIRATORY ALKALOSIS

a. PaCO2 > 35 mm Hg, BE = ± 2.5 mmol/l

b. PaCO2 > 45 mm Hg, BE = ± 2.5 mmol/l

36. CHOOSE THE CORRECT SYMPTOM OF RESPIRATORY ACIDOSIS

a. PaCO2 < 35 mm Hg, BE = ± 2.5 mmol/l

b. PaCO2 >45 mm Hg, BE = ± 2.5 mmol/l


37. SPECIFY 3 FEATURES OF RESPIRATORY INSUFFICIENCY  (RI) STAGE I.

a. signs of  lung diseases that can cause RI

b. shortness of breath during physical exertion exceeding the everyday

c. shortness of breath with everyday physical activity

d. shortness of breath at rest

e. optional features of pulmonary hypertension and RI

f. obligate signs of pulmonary hypertension and RI


38. A SIGN OF HYPOXEMIA IS

a. PaO2<80 mm Hg

b. PaCO2<45 mm Hg

c. SaO2<95%


39. A SYMPTOM OF HYPERCAPNIA IS

a. PaO2<35 mm Hg

b. PaCO2>45 mm Hg


40. CURSHMANN'S SPIRALS IN SPUTUM ARE DETECTED IN:

a) lung abscess;

b) crupous pneumonia;

c) chronic bronchitis;

d) asthma;

e) bronchiectasis.


41. ELASTIC FIBERS IN SPUTUM ARE DETECTED IN:

a) dry pleuritis;

b) crupous pneumonia;

c) asthma;

d) emphysema;

e) lung abscess.

Fig. 1. Finger clubbing.



Fig. 2. Rachitic type of chest shape.

Fig. 3. Navicular type of chest shape.
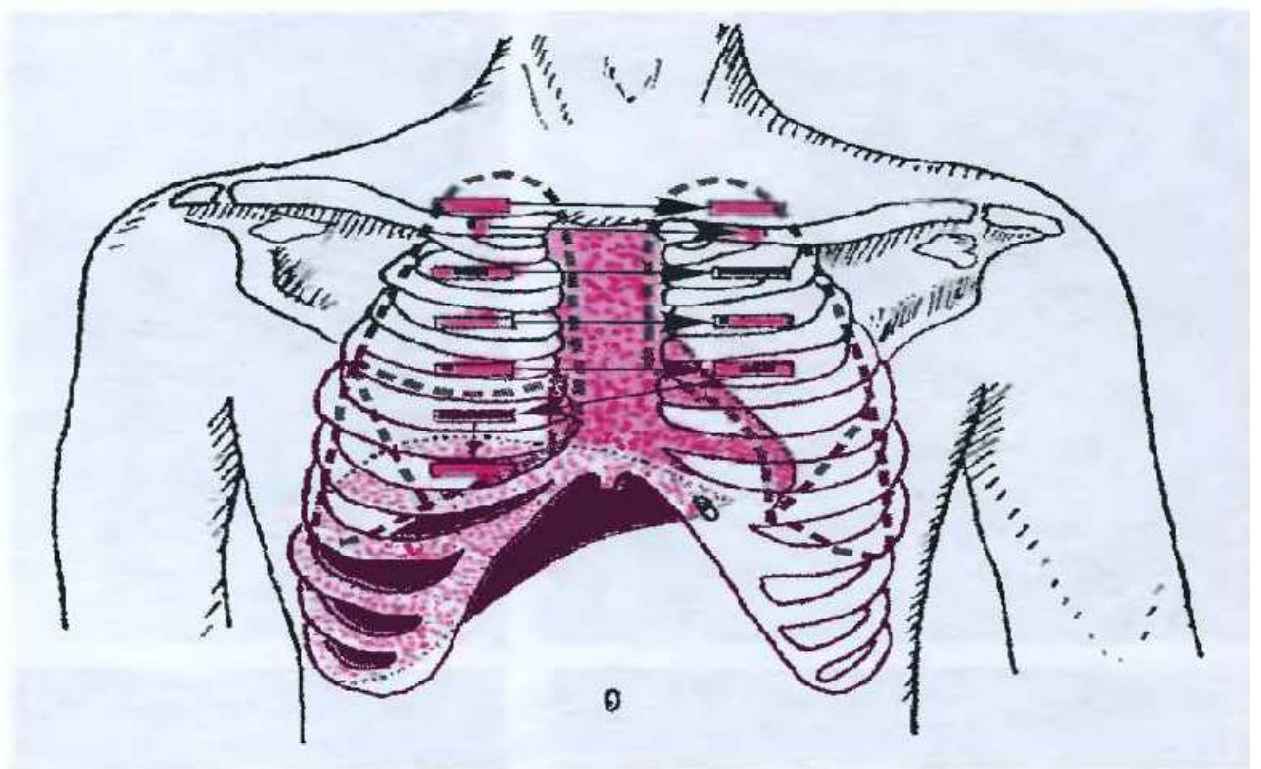


Fig.4. Consequence of percuss of the anterior side of the chest.
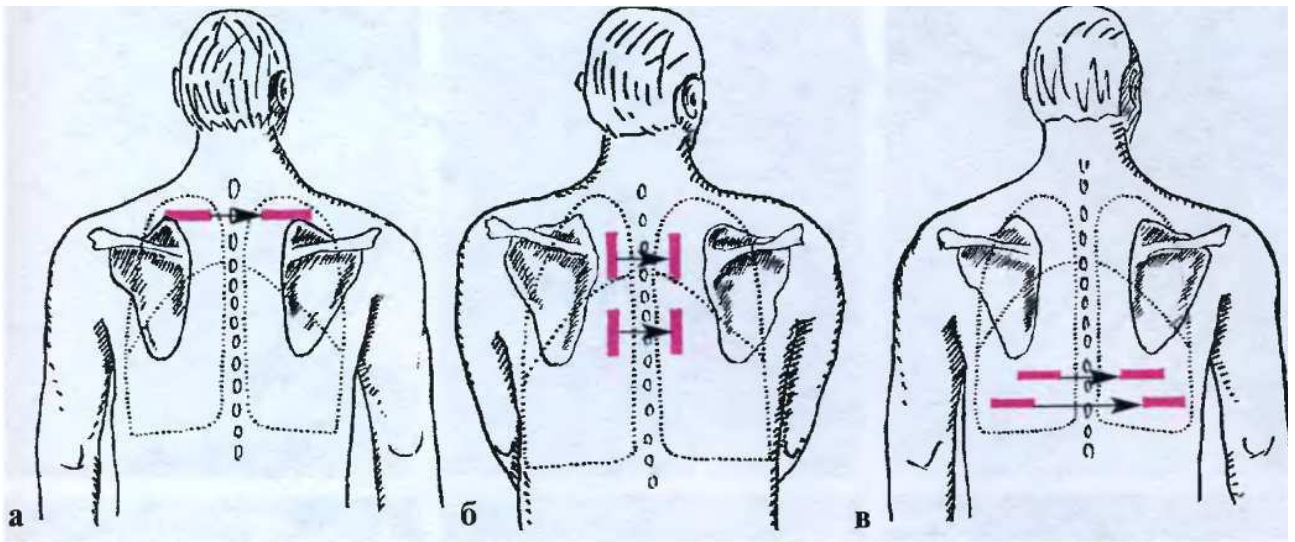
Fig.5. Consequence of percuss of the posterior side of the chest.

a. suprascapular region; b. intrscapular region; в. subscapular region
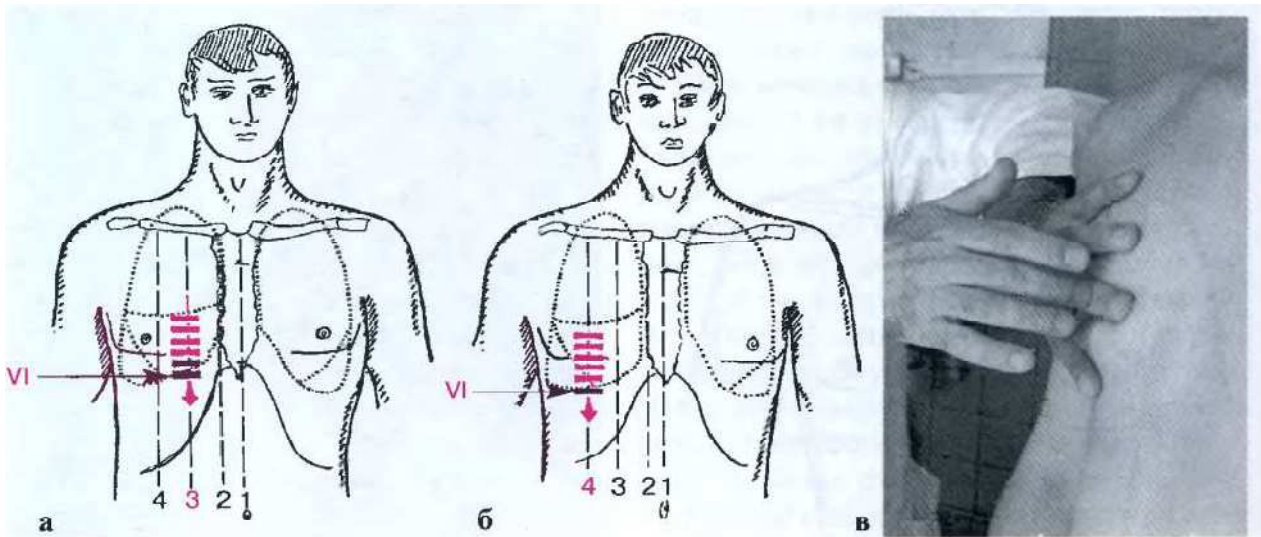


Fig. 6. Percuss of lower border of the right lung  (the anterior side of the chest).

a.  parasternal line; b. mediaclavicular line
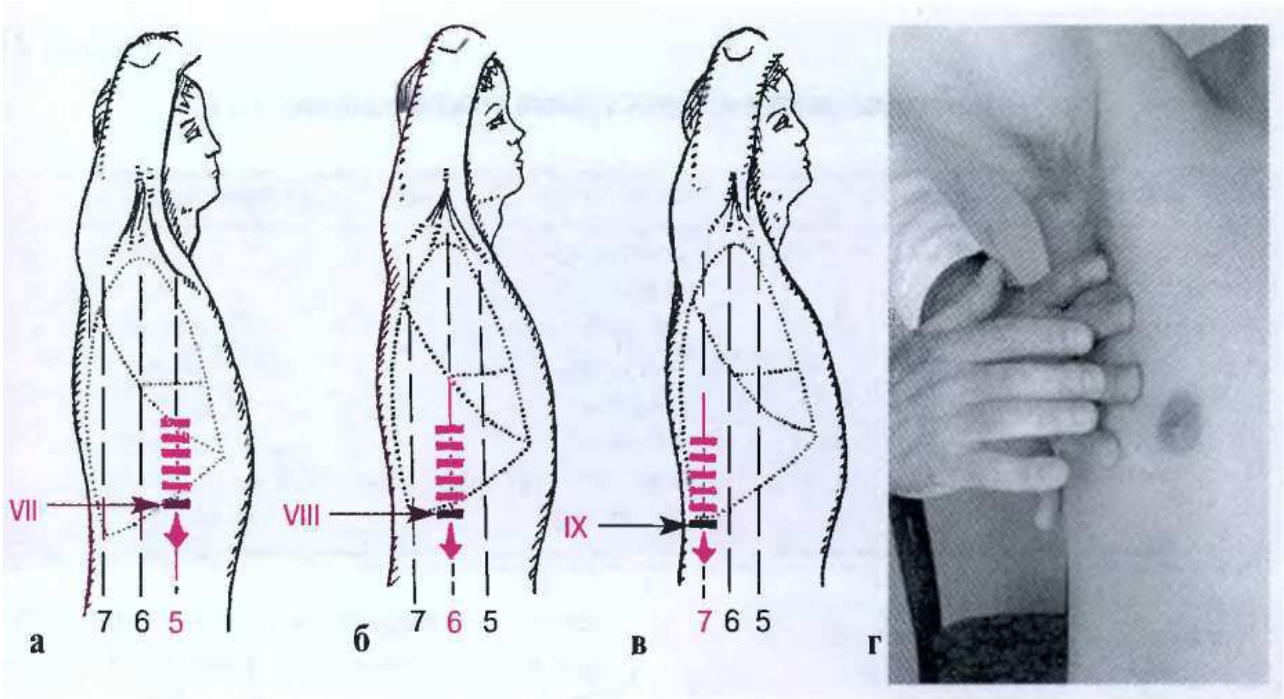
Fig.7. Percuss of lower border of the right lung at (a) anterior (b) medium (в) posterior axillaries lines.
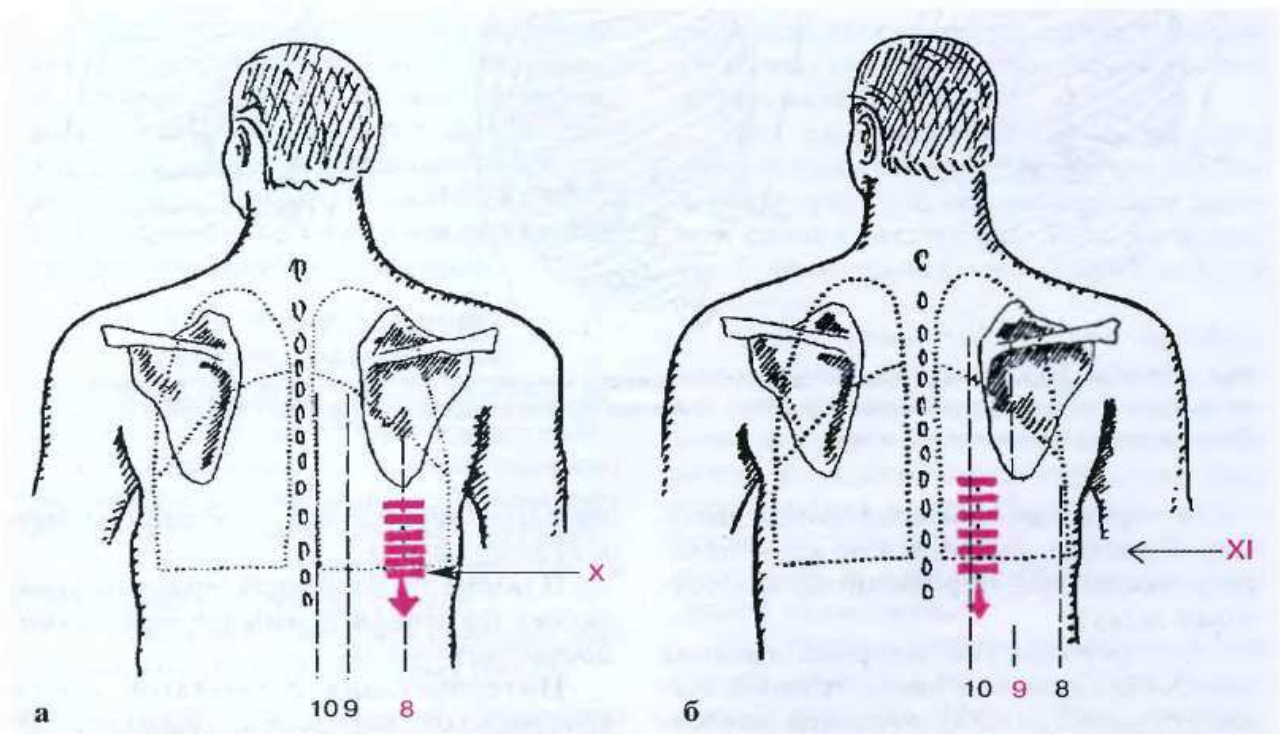


Fig.8. Percuss of lower border of the right lung at (a) scapulars (b) paravertebralis lines.
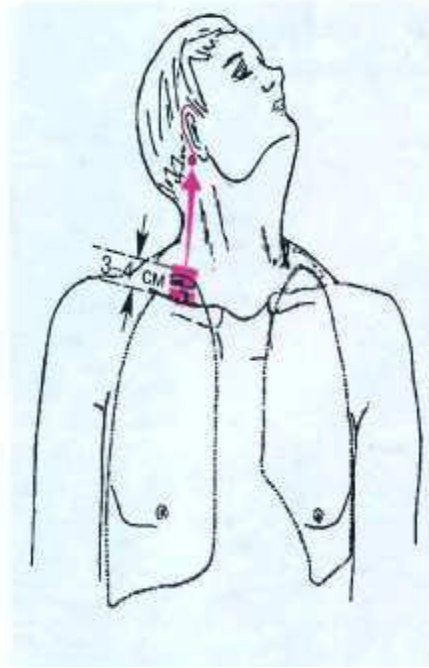
A.
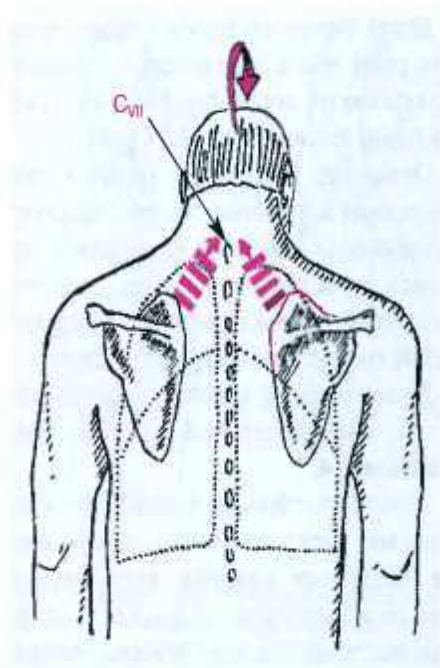


B.

Fig.9. Percuss of upper border (apex) of the right lung at (A) anterior side; (B) posterior side
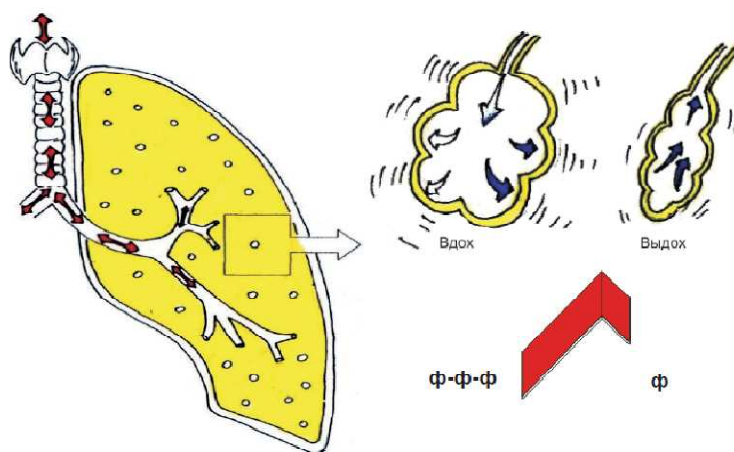
Fig. 10. The mechanism of formation  vesicular breath sounds (A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
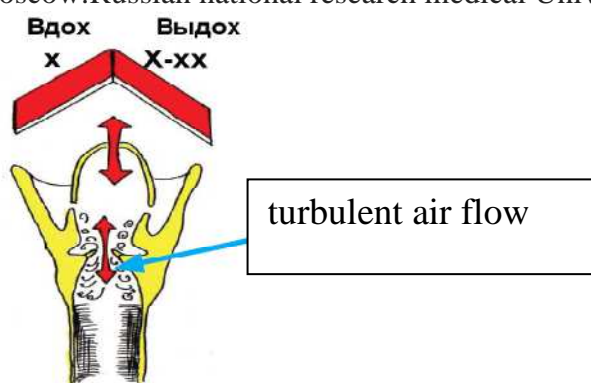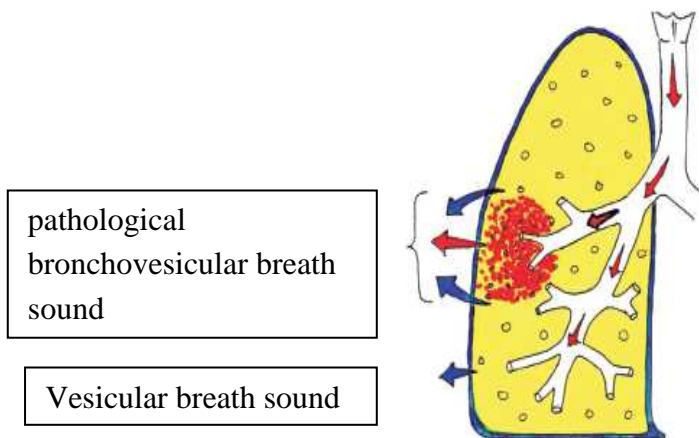


Fig. 11. The mechanism of formation  laryngotracheal breath sounds
(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
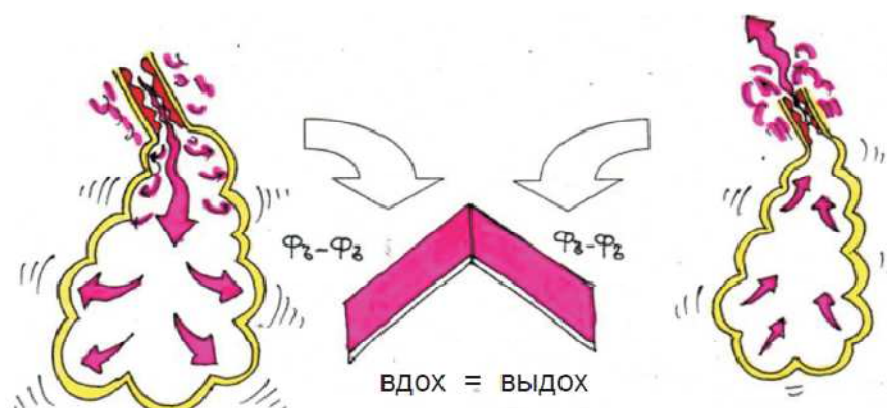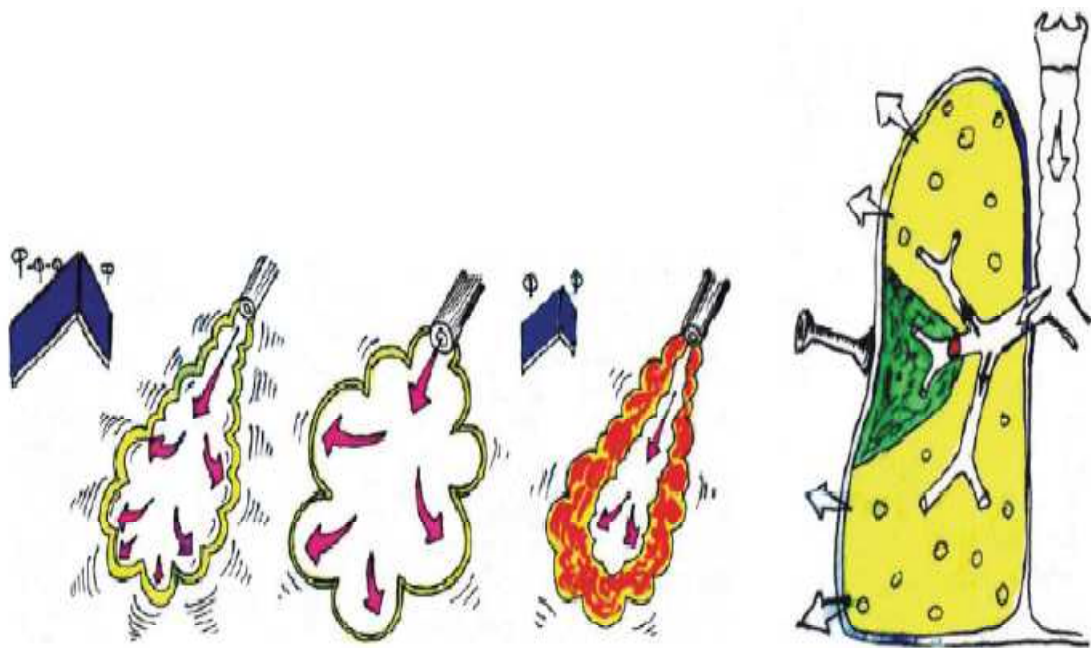


Fig. 12. The mechanism of harsh breath sound.
(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
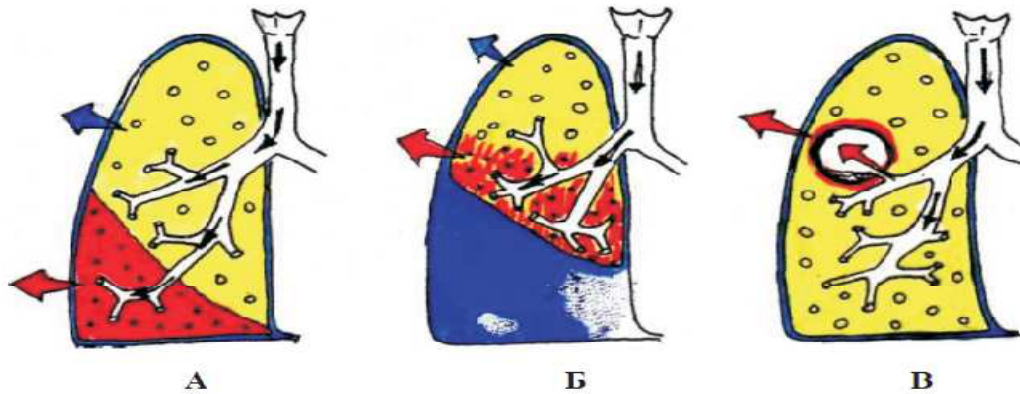
Fig. 13. The mechanism of formation  pathological bronchial breath sound:

A. infiltrative (inflammation, tumour, infarction etc.), Б. compressive (in exudative pleuritis above the fluid border), B. cavitary (in presence of large pulmonary cavities) – amphoric.

(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)



pathological bronchovesicular breath sound

Vesicular breath sound

Fig. 14. The mechanism of formation  pathological bronchovesicular breath sound. (A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)

Fig. 15. The mechanism of diminishing or absence of vesicular breath sounds.

(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
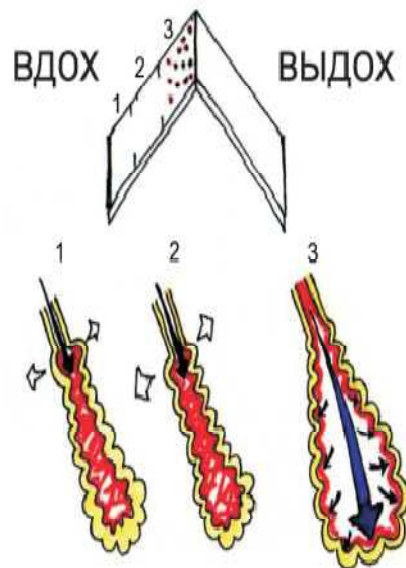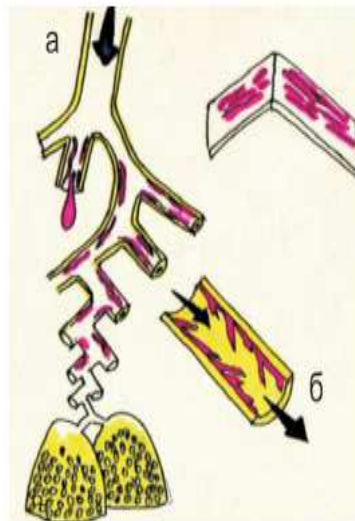


Fig. 16. The mechanism of formation of late inspiratory (fine) crackles.

(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
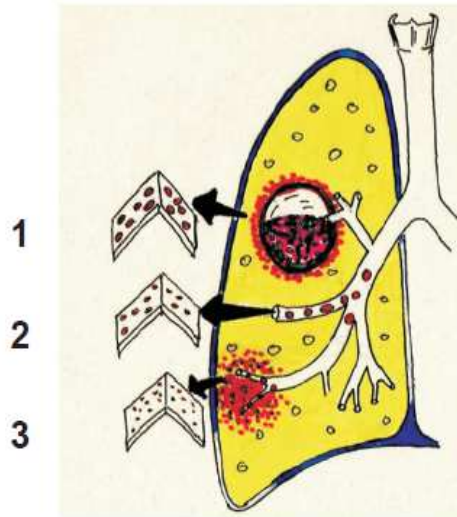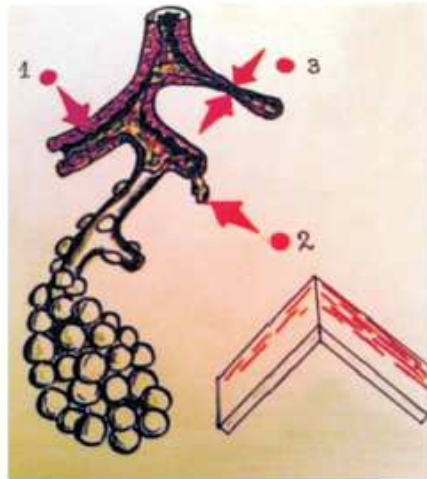
Fig. 17. The mechanism of formation of inspiratory and expiratory (croake) crackles:
1- small "buble" crackles 2- medium "buble" crackles
3- major "buble" crackles
(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)



Fig. 18. The mechanism of formation of bass wheezing.

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Fig. 19. The mechanism of formation of whistling wheezing:
1- thickening of the bronchial mucosa
2- narrowing of the lumen due to the presence of parietal-phlegm
3- bronchospasm
(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)



two inflamed surfaces of the pleura rubbing against each other during respiration
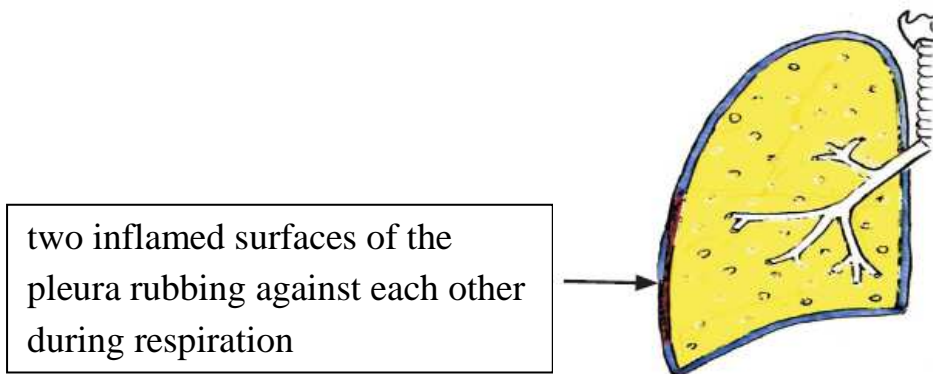
Fig. 20. The mechanism of formation of pleural crackles (rub).

(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S. A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N. And. Pirogov, 2011)
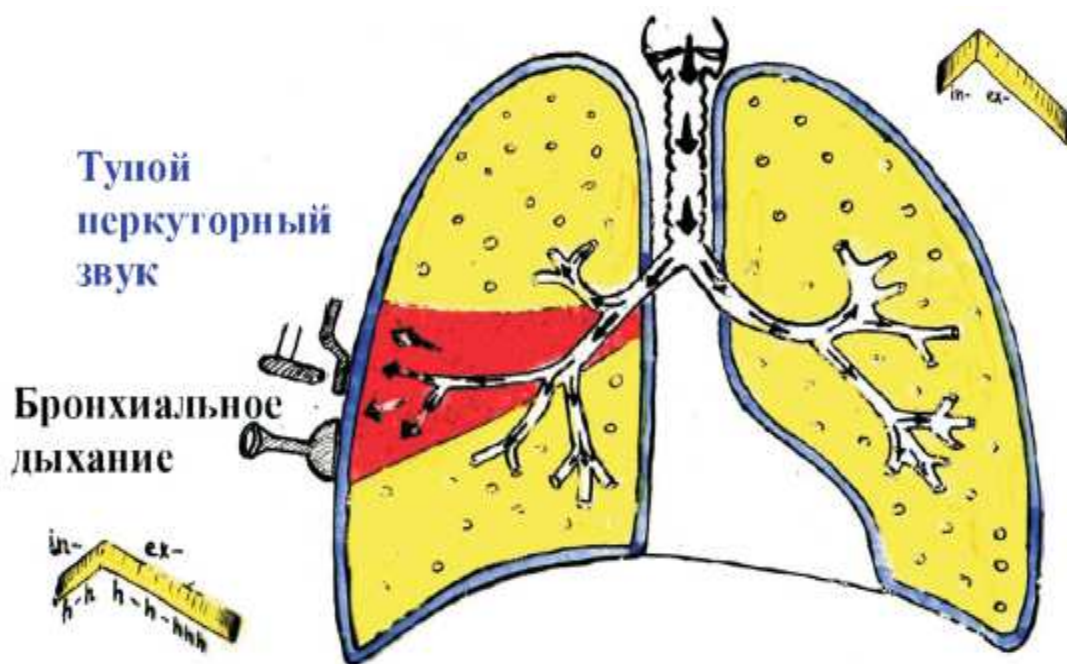
Fig. 21. The height stage of pneumonia.

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Compression atelectasis – dullness with
tympanic shade of percussion sound
Bronchial breath sounds, crepitus
Bulging of intercostal spaces

Pleural effusion
Dull percussion sound
No holding of breath

Obturative atelectasis
Retraction of intercostal spaces
Dull percussion sound
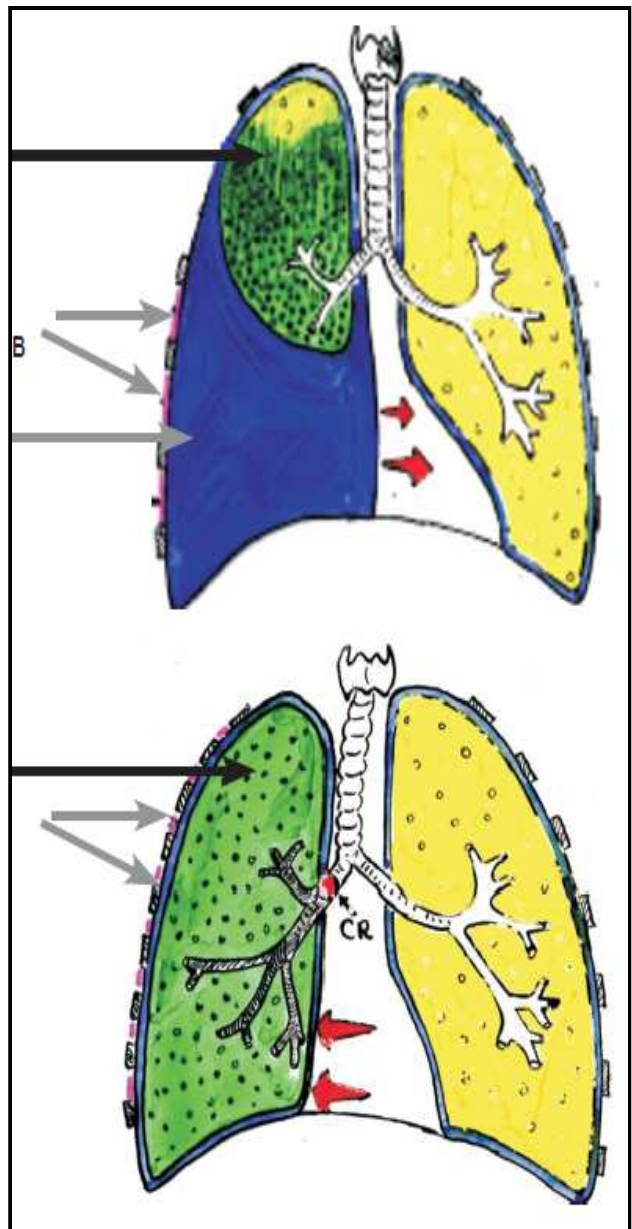The absence (diminished) vesicular breath
sounds

Fig. 22. Compression and obturative atelectasis

(A dictionary of technical terms in the discipline "Propaedeutics of internal diseases" ed. by S.
A. Melent'eva, G. Yu. Golubeva.-Moscow:Russian national research medical University them. N.
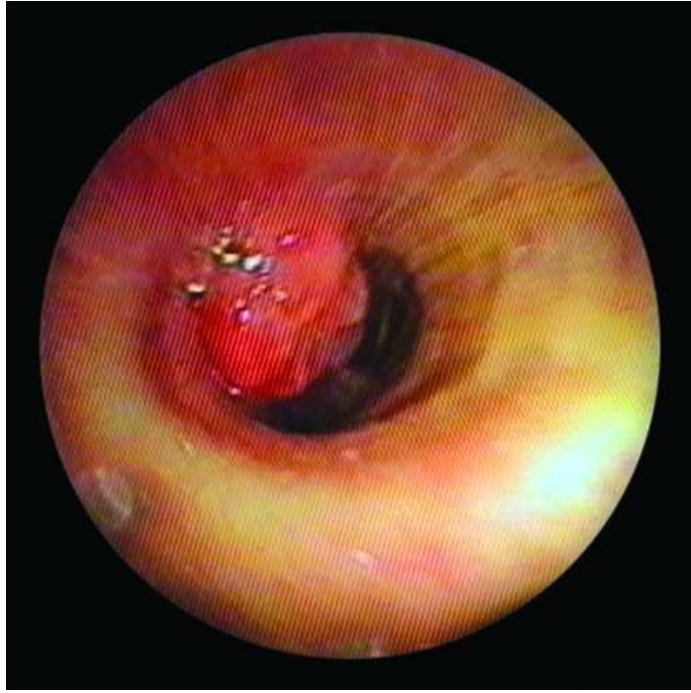And. Pirogov, 2011)

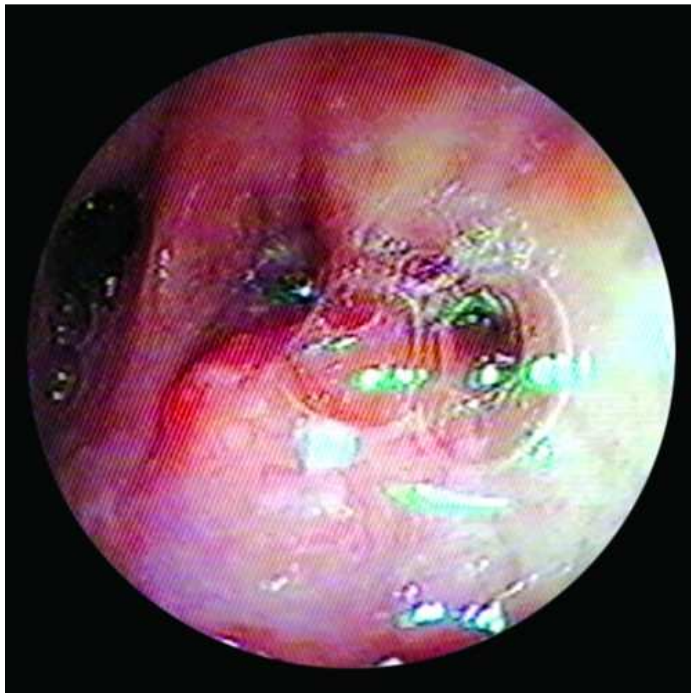Fig.23. Exophytic cancer.



Fig.24. Endophytic cancer.

Fig. 25. Ulcerative form of cancer.



Fig. 26. Foreign body of the medium bronchus.

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