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УО «Витебский государственный медицинский университет»

ПРОПЕДЕВТИКА ВНУТРЕННИХ БОЛЕЗНЕЙ

Часть I: Обследование органов дыхания и сердечно-сосудистой системы

PROPAEDEUTICS OF INTERNAL DISEASES

Part I: Examination of Respiratory and Cardiovascular Systems

учебное пособие

Допущено Министерством образования Республики Беларусь
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Предназначается для студентов 2- и 3-го курсов, изучающих пропедевтику внутренних болезней на английском языке.

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CONTENTS

Dedicated to the Teacher – professor N.E. Fedorov, Head of the Department of Propaedeutics of Internal Medicine of the Vitebsk Medical University (1987-2002)

PREFACE

The textbook “Propaedeutics of Internal Diseases. Part I: Examination of Respiratory and Cardiovascular systems” is designed for 2-d and 3-d year students for the training through the mediation of the English language. The textbook contains the following sections of the educational discipline “Propaedeutics of Internal Diseases”: subjective and objective examination of a patient’s clinic of internal diseases, respiratory and circulatory system examination.

The textbook corresponds to the basic educational thematic parts of Propaedeutics of Internal Diseases, according to Standard Educational Program of Propaedeutics of Internal Diseases approved by the Ministry of Public Health of the Republic of Belarus from 4th September 2014., registration № ТД-L.399/тип. and the syllabus of internal diseases propaedeutics for students of the medical faculty approved by Vitebsk State Medical University in 2014. The textbook can be also useful for teachers of therapeutic chairs of medical universities in preparations for classes as well as for senior students, post-graduates and clinical residents.

The main provisions of the textbook are presented in accordance with the Prof. N.E. Fedorov’s methodological approaches to teaching Propaedeutics of Internal Diseases. The reference sources are selected regarding a modern level of internal medicine development. We ask to send all critical remarks and wishes to the Chair of Propaedeutics of Internal Diseases of Vitebsk State Medical University.

The author is extremely grateful for the constant support of the head of Propaedeutics of Internal Diseases Chair of Vitebsk State Medical University, Prof. G.I. Yupatau, associate professors O.V. Dragoon, M.S. Drozdova, L.V. Soboleva, V.T. Valui, senior teachers I.V. Arbatskaya, L.A. Efremova, I.N. Pavlova, who have provided very helpful advices and contributed in many ways assistance with the text-pictures of this book.

List of Main Abbreviations

AF - atrial fibrillation
AV block - atrioventricular block
AV node (AV junction) - atrioventricular node, or atrioventricular junction
BMI - body mass index
BNP - B-type (brain) Natriuretic Peptide
BP - blood pressure
CK-MB - creatine phosphokinase MB-fraction
COPD - chronic obstructive pulmonary disease
CRP - C-reactive protein
CT - computed tomography
DBP - diastolic blood pressure
ECG - electrocardiography
EchoCG - echocardiography
ERV - expiratory reserve volume
FEF - forced expiratory flow
FEV1 (or FVC1) - forced expiratory volume after first second of expiration
FEVC (or FVC) - forced expiratory vital capacity
GCS - Glasgow Coma Scale
HDL - high density lipoprotein
ICD-10 - 10th revision of the International Statistical Classification of Dis-eases and Related Health Problems
IRV - inspiratory reserve volume
LDH1- lactic dehydrogenase first enzyme
LDL - low density lipoproteins
MAS - Morgagni-Adams-Stokes syndrome
MUAMC - mid upper arm muscle circumference
MV - minute volume of respiration
MVL - maximum ventilation of lungs
6MWT - 6 minute walk test
NYHA - New York Heart Association
PCG - phonocardiography
RR - respiration rate
RV - respiratory volume
S1 - first heart sound
S2 - second heart sound
S3 - third heart sound
S4 - fourth heart sound
SBP - systolic blood pressure
VC - vital capacity
WHR - waist-to-hip ratio

Unit I. Subjective and Objective Examination of a Patient

CHAPTER 1. Subjective Examination (Inquiry) and its Role in making the Diagnosis

Objectives: to enable students to learn –

1) Subject and purposes of the academic discipline "Propaedeutics of internal diseases";
2) general plan of clinical examination and taking a case history;
3) rules and technique of subjective examination (inquiry of a patient);
4) diagnostic value of questioning complaints, general anamnesis, present disease history (anamnesis morbi), past life history (anamnesis vitae).

1.1. Subject and problems of Propaedeutics of Internal Diseases

Propaedeutics of the internal diseases is the academic discipline, containing systematized scientific knowledge and techniques for the examination of a healthy man and a patient with internal organs pathology, and basics of etiology, pathogenesis and clinical manifestations of the most widespread internal organs diseases. The subject of propaedeutics of internal diseases is the introduction to diagnosis and therapy of internal organs diseases.

The purposes of academic discipline "Propaedeutics of Internal Diseases" consist of acquiring scientific knowledge and practical skills development by students in techniques of the examination of a healthy man and a patient with internal organs pathology; symptoms, signs and syndromes of the most widespread internal organs diseases; techniques of diagnosis, and writing a case history.

Clinical examination of a patient requires adherence to **medical ethics and medical deontology**.

Medical ethics is a system of moral principles that apply values to the practice of clinical medicine and in scientific research. Medical ethics is based on a set of values that professionals can refer to in the case of any confusion or conflict. These values include the respect for autonomy, non-maleficence, beneficence, and justice. Medical ethics is the theoretical basis of a deontology. The deontology in a literal translation means the doctrine about a duty.

Medical deontology means a complex of ethical standards, principles by which medical profession is guided, including medical confidentiality, the medical worker's responsibility for the life and health of the patient, and relationships of medical workers to each other (T. L. Beauchamp, 2013).

***The basic principles of medical ethics and medical deontology*:**

- medical personnel should avoid doing harm to their patients in any situation;
- medical personnel, including medical students, should approach patients with consideration, respecting their personal dignity, right to intimacy and privacy, and should perform all diagnostic, therapeutic and preventive procedures with due exactitude and sharing the required time;
- medical personnel, including medical students, has the duty to maintain confidentiality. The information obtained in the course of medical professional duties concerning the patient and his/her background is to be kept confidential. The death of the patient does not release the physician from the duty of maintaining confidentiality;
- it is the duty of every medical profession and medical student to continually update and develop professional knowledge and skills as well as to share them with co-workers.

The main definitions for an understanding of the academic discipline "Propaedeutics of Internal Diseases": health, disease, sign, symptom, syndrome, diagnosis.

Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.

Disease - any harmful deviation from the normal structural or functional state of an organism, generally associated with certain signs and symptoms and differing in nature from physical injury. A diseased organism commonly exhibits signs or symptoms indicative of its abnormal state (Encyclopædia Britannica, 2020). Diseases usually are indicated by signs and symptoms.

A sign is defined as an objective manifestation of disease, for example, jaundice or enlarged liver that can be determined by a physician during the physical examination of a patient.

A symptom is subjective evidence of disease reported by the patient. Such sensations as, for example, pain or nausea experienced by the patient are referred to symptoms. They can reflect objective changes in the patient's body. *Symptoms and signs are often nonspecific, but certain combinations can be suggestive of certain diagnoses*. In other cases, they are specific even to the point of being *pathognomonic* (characteristic for a particular disease) *symptoms and signs*.

A syndrome is the association of several medical signs, symptoms, or other characteristics that often occur together, and characterize a particular abnormality or condition. Some syndromes, such as Down syndrome, have only one cause. Others, such as syndrome of pulmonary heart, have multiple possible causes. Even in syndromes without known etiology, the presence of the associated symptoms and signs usually leads the physician to diagnostic hypothesis that there exists an unknown underlying cause.

Diagnosis is the process of determining the nature of a disease or disorder and distinguishing it from other possible conditions. The term comes from the Greek *gnosis*, meaning knowledge.

Clinical diagnosis combines taking the patient's health history, physical examination, and laboratory-instrumental examinations. Examinations include three main stages - *subjective (interview, or inquiry, taking anamnesis), objective (physical) and laboratory-instrumental examination.*

1.2. General plan of clinical examination and a case history

Common scheme of a case history corresponds to the general plan of clinical examination (Table. 1-1).

Table 1-1. Plan of clinical examination

<table>
<tr><td colspan="4">I. Subjective Examination
(Interview, inquiry, taking history, or anamnesis)</td></tr>
<tr><td>Complaints</td><td>Review of systems (anamnesis communis, status functionalis)</td><td>History of present disease (anamnesis morbi)</td><td>History of past life (anamnesis vitae)</td></tr>
<tr><td colspan="4">Primary diagnostic hypothesis</td></tr>
<tr><td colspan="4">II. Objective Examination (Physical examination)</td></tr>
<tr><td>Survey (inspection)</td><td>Palpation</td><td>Percussion</td><td>Auscultation</td></tr>
<tr><td colspan="4">Provisional diagnosis</td></tr>
<tr><td colspan="4">III. Laboratory and instrumental examination
(X-ray, radiological, ultrasound, ECG, immunology, etc)</td></tr>
<tr><td colspan="4">Clinical diagnosis</td></tr>
</table>

Common scheme of a case history

1. Passport data: surname, name, patronymic; sex, age, home address, place of employment, the diagnosis when admitted to hospital, the clinical diagnosis.
2. Inquiry:

a) Present complaints of the patient;

b) Status functionalis, review of systems, or general anamnesis;

c) Anamnesis morbi, or history of a present disease;

d) Anamnesis vitae, or life history, or past history;

3. Objective examination of the patient's condition at the present

time (status praesens):

a) general survey, survey of the head and the neck;

b) respiratory system (survey, palpation, percussion of the chest, auscultation of the lungs);

c) cardiovascular system (survey; palpation, percussion and auscultation of the heart and the large blood vessels);

d) system of digestion (examination of an oral cavity, examination of the abdomen in the vertical and horizontal position, including auscultation, percussion, and palpation of the abdomen);

e) genitourinary system (survey of the lumbar region and external genetalia, percussion and palpation of the kidneys and the urinary bladder, ureteric points);

f) nervous system.

4. Substantiation of the provisional diagnosis.

5. Plan of additional examination and treatment of the patient.

According to the provisional diagnosis the proper plan of the treatment and additional laboratory and instrumental methods of research, as well as medical consultations are prescribed.

6. Results of laboratory and instrumental investigations.

7. Conclusions of a consulting physician.

8. Clinical diagnosis and its substantiation.

Clinical diagnosis is formulated as a short-form statement of the disease based on analysis of the specific complaints of the patient, the data of an anamnesis, the data of survey and physical examination, and the data of laboratory and instrumental methods of research**.**

The clinical diagnosis indicates the underlying disease, including the degree of severity, the stage of disease, its functional class, the failure degree of an organ or system of organs, its complications and concurrent diseases.

Diagnosis is thus, a dynamic process to be developed and completed by the analysis of the continuing variations in the patient's condition. The study of these changes is another test for correctness of the primary diagnosis.

9. Medical diary.

A diary in a case history is filled every day and reflects dynamics of patients' state for the running day and efficacy of the prescribed medical actions. The diary of a patient in a serious state should be recorded every 2-4 hours with a precise statement of all medical actions and their results, description of new symptoms and substantiation of new prescriptions. Body temperature, a pulse rate, a respirations rate, stool and diuresis are marked daily in the diary.

10. Epicrisis

The case history routinely is finished by an epicrisis. The epicrisis

is a briefly described summary of the basic complaints of the patient, the history of his/her disease, objective data, basic laboratory and instrumental studies, the diagnostic resume, the course of the disease during the observation, the treatment and its results, the discharge recommendations for further treatment and regimen, and for a place of work.

1.3. Subjective examination (inquiry)

1.3.1. Approach to a medical interview

A clinical examination begins with the questioning of the patient (*inquiry, taking anamnesis*). Anamnesis is the most universal diagnostic tool for physicians. Inquiry is also one of the most complicated clinical skills.

The patient's history is of a greater diagnostic value than a physical examination or the laboratory test results. About two-thirds of the diagnoses can be made only on the basis of the medical history, despite the technological achievements of the modern hospital. Careful taking anamnesis makes further physical and laboratory-instrumental examinations of the patient more effective.

Getting started with medical interview*:*

A conversation between the doctor and the patient begins with an acquaintance. It is necessary to express the doctor's respect and attention to the patient as a unique person. Taking anamnesis should begin with a presentation that includes the names, the purpose and the timing of the medical interview. For example: *Hi, Mrs. Miller, my name is James Brown. I'm a second-year medical student. I'll talk to you for about 30 minutes to find out what issues are bothering you.*

The first questions are usually as follows, as a rule: *what problems brought you to the hospital (polyclinic) today? what's been bothering you lately?*

It is necessary to clarify whether there are any other problems with the patient's health. If the list of patient's problems is very long, it is necessary to ask what is bothering him/her the most?

The patient is given the opportunity to speak freely. Then the physician takes the issues into his own hands and discovers to ask in details every complaint of the main symptoms of each of the systems of the internal organs (respiratory, blood circulation, digestion, urinary systems, etc.).

It is necessary to evaluate objectively the intellectual level of each patient to be sure what the patient meant by each complaint. If you doubt that the patient correctly understands what he/her is asked, it is better to formulate a question again in a simpler form.

The most important features of a symptom are the time of its appearance, duration, localization, nature (pain, vomiting, cough, etc.), fre-

quency, causes (according to the patient), any provoking, enhancing or facilitating factors associated with their manifestations.

Follow-up Questions:

There is no single best way to question a patient. Successful interviewing requires avoiding medical terminology and making use of a descriptive language that is familiar to patients. There are several extensive questions which are applicable to any complaint. These include:

1. Duration: *"How long has this condition lasted? Is it similar to a past problem? If so, what was done at that time?"*

2. Location/Radiation: *"Is the symptom (e.g. pain) located in a specific place? Has this changed over time? If the symptom is not local, "does it radiate to a specific area of the body?"*

3. Severity/Character: *"How bothersome is this problem? Does it interfere with your daily activities? Does it keep you up at night?"* Try to have them objectively rate the problem. If they are describing a pain, ask them to rate it from 1 to 10 with 10 being the worse pain of their life, though first find out what they are using for comparison (e.g. childbirth, a broken limb, etc.). Furthermore, ask them to describe the symptom in terms which they are already familiar with. When describing a pain, ask if it is like anything else that they have felt in the past. *"Knife-like? A sensation of pressure? A toothache?"*

If it affects their activity level, determine to what degree this occurs**.** For example, if they complain of shortness of breath with walking, *"how many blocks can you walk? How does this compare with 6 months ago?"*

4. Ask if they have tried any therapeutic maneuvers? *"If so, what's made it better (or worse)?"*

5. Pace of illness: *"Is the problem getting better, worse, or staying the same? If it is changing, what has been the rate of the change?"*

6. *Are there any associated symptoms?* Over times the patient notices other things that have popped up around the same time as the dominant problem. These tend to be related.

7. What does a patient think the problem is and/or what is the patient worried it might be?

8. Why today? This is particularly relevant when a patient chooses to mention the symptoms/complaints that appear to be long standing. *Is there anything different today, than any other day when this problem has been present?*

The content of subsequent questions will depend both on what you uncover and your knowledge base/understanding of the patients and their illnesses. If, for example, the patient's initial complaint was a chest pain you might have uncovered by using the above questions:

The pain behind the breast bone began 1 month ago and only occurs with a physical activity. It is paroxysmal, often pressing or compressing,

accompanied by a feeling of fear of death. The duration of the pain is 3-5 minutes, rarely 15-20 minutes. The pain often radiates to the left shoulder, left arm, scapula, less often to the neck, lower jaw, interscapular space, sometimes to the epigastric region. It is associated with a physical exertion, lifting arterial pressure, and a psycho-emotional stress. It stops when a physical activity stops or immediately after taking nitroglycerin.

Questioning the patient provides a lot of information. With additional experience, exposure, and knowledge a physician will learn the appropriate settings for particular lines of questioning. When physicians obtain a history, they are continually generating differential diagnoses in their minds, allowing the patient's answers to direct the logical use of additional questions. With each step, the list of probable diagnoses is pared down until a few likely choices are left from what was once a long list of possibilities.

1.3.2. History taking (Inquiry)

An inquiry is taken in the following order:

1. Present complaints – the inquiry of the patient about complaints, about his/her sensations, experiences (Table 1-2). There are *chief (main) and minor (secondary) complaints*.

The ***chief (major, or main) complaints*** - the one or more symptoms causing the patient to seek medical care. The chief complaints are stated in details (see Subsection 1.3.1. Approach to medical interview).

The minor (***secondary) complaints*** are not related to the underlying disease (caused by another disease that does not currently play the main or leading role). If the patient's complaints are referred to any system of organs, then a patient is questioned in details about this system according to the scheme mentioned below.

Table 1-2. Complaints

Complaints		Complaints	
Chief (major, or main)	Minor (secondary)	Active	Identified
The major complaint, as a rule, coincides to seek a medical aid			
Details of complaints: *chronology, bodily location, quality, quantity, setting, any aggravating or alleviating factors, and associated manifestations*			

2. Status functionalis, review of the systems, or general anamnesis – the inquiry about a general state of the patient, major organs and systems of his/her organism at present and at the time immediately previous to the disease.

This interrogation is performed according to the following scheme:

General state of the patient: delicacy, malaise, loss of weight, fever,

diaphoresis (sweating), edemas, condition of skin (itching, skin rash).

State of the musculoskeletal system: any bone or joint pain accompanied by joint swelling or tenderness, aggravating and relieving factors for the pain and any positive family history for a joint disease.

State of the respiratory system: respiration by a nose, dyspnea (shortness of breath), coughing, dyspnea, pains in the chest.

State of the cardiovascular (circulatory) system: dyspnea, pains in the heart area, edemas, the heart palpitations, headache, elevated blood pressure.

State of the alimentary (digestive) system: appetite, swallowing, eructation, a heartburn, vomiting, meteorism, pains, stool (defecation).

State of the genitourinary system: frequency in urination, diuresis volume, pain with micturition (dysuria), urine color, any urethral discharge, altered bladder control like urgency in urination or incontinence, menstruation and sexual activity.

State of the endocrine system (weight loss, polydipsia, polyuria, increased appetite and irritability).

State of the nervous system: headache, giddiness, sleeplessness, vision, audition, sense of smell (olfaction), taste. Cranial nerves symptoms - vision (amaurosis), diplopia, facial numbness, deafness, oropharyngial dysphagia, limb motor or sensory symptoms and loss of coordination.

3. Anamnesis morbi, or History of present illness – the inquiry about the present disease, onset of the disease and the subsequent course to the present day, i.e. the day of the research of the patient, an anamnesis of disease.

Exact answers should be obtained from the patient concerning the following aspects of his present disease (*anamnesis morbi*):

(1) onset of the disease;

(2) first symptoms character;

(3) course of the disease;

(4) examinations and their results;

(5) treatment and its efficacy.

The answers to these questions may give the physician the necessary information on the present disease.

The history of the disease should include the information concerning the onset of the disease and its development until present. The patient's general condition before the disease should first be determined and the causes that might have provoked the disease be established wherever possible. The patient should be questioned in detail about the first signs of the disease and the chronology of their development (dynamics), about relapses or exacerbations, remissions and their duration. If the patient was examined during an exacerbation of the disease by some other physician, the results should be studied. The results of the previous examinations and treatment are important (therapy with cardiac glycosides, vasodilators, di-

uretics, antibiotics, hormones, etc.). Motives for hospitalization should also be determined (exacerbation of the disease, verification of the diagnosis, etc.).

4. Anamnesis vitae, or Past life history – the inquiry about previous life of the patient (Table 1-3). The past history is often very important for establishing the character, the cause, and conditions for the onset of the disease.

Table 1-3. Past life history (anamnesis vitae)

1	Conditions in which the patient lived and developed: *place of birth, development in childhood and adolescence, education, military service*
2	Medical anamnesis: *past diseases, surgery operations, blood transfusions, allergy and pharmacology anamnesis, obstetrical, epidemiological, and hereditary history*
3	Social anamnesis: *housing and living conditions, unfavourable labour conditions and industrial hazards, professional, and expert-labour history*
4	Bad habits: *tobacco smoking, consumption of alcohol beverage, narcotic drugs, sleeping pills and sedatives, strong tea and coffee, etc. (at what age, how much, how often and how much?)*
5	Risk factors: *behavioural, physiological, demographic, environmental, genetic factors*

Anamnesis vitae is a history or a medical biography of the patient in every period of his/her life (infancy, childhood, adolescence, and maturity). Collecting the anamnesis begins with the *general biographical information.*

Social history (SH) includes the information about housing and living conditions; flat (house), heating, house, running water, sewerage, its area, on which floor the apartment, and how many family members live in the apartment, etc. Characteristics of nutrition are important: regularity and frequency of food intake, its usefulness, dry and hasty eating, addiction to any food. Social history includes also information about the patient's education, profession, way of life, financial status, relationships in the family and with friends.

Unfavourable labour conditions and industrial hazards are important. For example, some harmful dusts may cause bronchial asthma and chronic diseases of the bronchi and lungs.

For adult males important question is about military service, if patient had not served - whether it is related to any health problem.

Past diseases are also important, including childhood and adult ill-

nesses. Some infectious childhood diseases, such as measles or scarlet fever, do not recur because of acquired immunity, while, other diseases, tend to recur. Acute rheumatic fever or diphtheria often provokes heart diseases. Separately each patient must be asked, whether he (she) was sick of rheumatism, tuberculosis, virus hepatitis, oncologic and venereal diseases, whether mental diseases, alcoholism were in the patient's family.

Surgery history includes questions about the passed operations, traumas.

Transfusiology anamnesis – questions about passed hemotransfusion and transfusion of blood substitutes, the complication of transfusions.

Familial and sexual history. Questioning under this section should be confidential, without the presence of other patients. It turns out the marital status (at what age married or married), the composition of the family and the health of its members. In men, the time of puberty (the appearance of a mustache, beard, the beginning of the emission), especially sexual life are turned out. In women, the state of the menstrual cycle (the time of the first menstruation, when it started, their duration, intensity, pain, time of menopause), pregnancy and childbirth, their course, abortion and their complications, miscarriages are turned out.

Obstetrical (gynecological) history contains questions for female patients – pregnancy and their course, delivery, abortions and their complications, transferred gynecologic diseases, health of the husband.

Hereditary history. Health of the parents, sisters or brothers is often informative. If they died, you should find out at what age and from what disease it happened. Did the parents or other relatives suffer from the same disease as the patient? By comparing the pathology of the patient with diseases of his/her relatives, the physician can make a conclusion on the role of the hereditary factors in the development or the origin of the disease.

Bad habits. This section of the history is also desirable to collect without witnesses in view of the delicacy of the questions. Information about smoking – how long and what smokes, the number of cigarettes smoked or cigarettes per day. Consumption of alcohol drinks (from what age, what, how often and in what quantity?); narcotic drugs (morphine, opium, cocaine, codeine, etc.), sleeping pills and sedatives, strong tea and coffee.

Pharmacologic history – questions about regular and short-term taking medications (including those prescribed by doctors, and others obtained over-the-counter or alternative medicine), and adverse effects of medications.

Allergy anamnesis is very important. Some patients (and even healthy people) often develop a pathologically heightened (or an inverted) response of the immune system (allergy), and this factor is essential in the pathogenesis of certain diseases of internal organs. It is necessary therefore to collect an allergy anamnesis, that is determined whether

the patient or his/her relatives had allergic reactions to various foods, because strawberry, eggs, canned crabs, and other foods may frequently act as an allergen as well as some medicinal preparations, perfumes, and pollen. Allergic reactions are quite varied: from vasomotor rhinitis, nettle rash or Quincke's edema to an anaphylactic shock.

Epidemiological anamnesis has a special value to diagnosis of the infectious diseases and includes such parts as:

- business trips and tours in districts, unsuccessful in the attitude of any infection contamination;
- place of employment, occupation (profession of the patient) (the milk-maid, the worker of a meat-packing plant, veterinary workers);
- contact with infectious patients (AIDS; malaria, typhus, acute fever, diarrhea, etc.);
- intake of not fresh and poor quality foodstuff, raw water from reservoirs;
- beginning of similar diseases simultaneously among surrounding people;
- past contagious diseases and vaccinations.

Expert-labour anamnesis includes questions about:

- temporary disability for work are estimated on a number of cases of illness and days of disability (duration of a sick-list) within the last 12 months;
- permanent disability for work are estimated on presence of disability group, its causes and duration of it;
- professional labour activity – whom and where the patient worked, the experience of work in the basic profession;
- character of labour conditions – a degree of a psychological strain and heaviness of a physical stress, a regimen of work, working shifts, etc.;
- sanitary-and-hygienic working conditions – temperature, humidity, (a dust content, a gassed condition, vibration, hum, illuminating intensity, presence of toxicants, radiations and other industrial hazards, etc.

Risk factor is a characteristic, condition, or behaviour that increases the likelihood of getting a disease or injury. Risk factors can be:

- behavioural – nutritional choices, physical inactivity, not having certain vaccinations, unprotected sex, etc.;
- physiological – overweight, high blood pressure, high blood cholesterol, high blood glucose etc.;
- demographic – age, gender, population subgroups, such as occupation, religion, or income;
- environmental – access to clean water and sanitation, risks in the

workplace, air pollution, social settings;

- genetic – risk factors are based on an individual's genes for some diseases, such as cystic fibrosis and muscular dystrophy, come entirely from an individual's 'genetic make-up'. Many other diseases, such as asthma or diabetes, reflect the interaction between the genes of the individual and environmental factors. Other diseases, like sickle cell anaemia, are more prevalent in certain population subgroups.

1.4. Primary diagnostic hypothesis (initial diagnosis)

This is a first diagnostic stage that distinguishes cardinal symptoms of the disease revealed by means of the inquiry of the patient. According to complaints and anamnesis data of the patient, the conclusion of probable character of pathological process and localization of the affected organ (or the affected system are substantiated.

You may then focus your exam on the search for physical signs that would lend support to your diagnostic hypothesis and help direct you in the rational use of adjuvant testing.

1.5. The key points for the theme "Subjective Examination (Inquiry) and its Role in making the Diagnosis"

Propedeutics of internal diseases includes the techniques of the examination of a healthy man and a patient with the internal organs pathology; symptoms, signs and syndromes of the most widespread internal organs diseases; techniques of diagnosis, and writing a case history.

Clinical examinations includes three main stages - subjective, objective and laboratory-instrumental examination. Common scheme of a case history corresponds to the general plan of the clinical examination.

Clinical examination of a patient requires adherence to the basic principles of medical ethics.

Diagnosis in medicine is the determination of the nature of a disease. Clinical diagnosis combines taking the patient's anamnesis, physical examination, and laboratory-instrumental examinations.

Subjective examination (interview, or inquiry, taking anamnesis) includes present complaints, status functionalis (or review of systems, general anamnesis, anamnesis communis), history of present illness (anamnesis morbi), past history (anamnesis vitae, past life history).

Present complaints is the inquiry of a patient about his/her complaints, sensations and experiences at present.

Status functionalis (review of systems, or general anamnesis) is the inquiry about a general state of the patient, and about functioning major organs and systems of the body at present and in the time immediately previous to the disease.

Anamnesis morbi (or history of the present disease) is the inquiry about

the present disease, about its beginning and the subsequent course to the present day, i.e. day of the patient's examination.

Anamnesis vitae (past life history) is the inquiry about the previous life of the patient from a medical point of view: conditions in which the patient lived and developed, medical anamnesis (past diseases, surgery operations, allergy reactions, etc.), hereditary history, bad habits.

Primary diagnostic hypothesis (initial diagnosis) is a first diagnostic stage that distinguishes cardinal symptoms of the disease revealed by means of the inquiry of the patient.

1.6. Assessment tests on the theme "Subjective Examination (Inquiry) and its Role in making the Diagnosis"

Choose the correct answers on the following tests:

1. Subjects of propedeutics of internal diseases are:
1. diagnosis of internal diseases;
2. taking anamnesis;
3. symptoms of internal diseases;
4. proper physical examination of a patient;
5. treatment of internal diseases.

2. General plan of inquiry includes the following parts:
1. present complaints;
2. general anamnesis;
3. history of present illness;
4. anamnesis vitae;
5. status praesens.

3. Basic parts of the clinical examination of patients:
1. subjective examination;
2. objective examination;
3. laboratory-instrumental examination;
4. inspection;
5. auscultation.

4. Subjective examination of a patient includes the following parts:
1. present complaints;
2. anamnesis communis;
3. anamnesis morbi;
4. anamnesis vitae;
5. general inspection;
6. local inspection.

5. Subjective symptoms are:

1. perceptible just to the patient;
2. perceptible to others than the patient;
3. signs of the disease revealed by a physician;
4. data of laboratory tests;
5. data of general and local inspection.

6. Objective symptoms are:
1. pain;
2. vertigo;
3. nausea;
4. jaundice;
5. edema.

7. Objective symptoms are:
1. perceptible to others than the patient;
2 perceptible just to the patient;
3. signs of the disease experienced by a patient;
4. data of instrumental tests;
5. data of anamnesis morbi.

8. Objective symptoms are:
1. pallor;
2. rapid pulse;
3. headache;
4. itching;
5. pain.

9. Taking general anamnesis includes the following parts of interrogation:
1. general state of the patient;
2. state of respiratory, cardiovascular, digestive, urinary and nervous systems;
3. anamnesis morbi;
4. anamnesis vitae;
5. status praesens.

10. Taking anamnesis morbi includes the following questions about:
1. time of the disease onset;
2. first symptoms and course of the disease;
3. general state of the patient;
4. past diseases;
5. bad habits.

11. The case report section that contains the information about the passed infectious diseases:

1. anamnesis vitae;
2. passport data;
3. main complaints and its detailing;
4. anamnesis communis;
5. anamnesis morbi.

12. Taking anamnesis vitae includes the following parts:
1. general biographical data;
2. labour conditions and industrial hazards;
3. social history;
4. allergy anamnesis;
5. hereditary anamnesis;
6. transfusiology anamnesis.

13. Social history includes the information about:
1. housing and living conditions of the patient;
2. occupational and recreational aspects of the patient's personal life;
3. hereditary diseases in relatives of the patient;
4. passed infectious diseases;
5. bad habits.

14. Infectious-epidemiological anamnesis includes the information about:
1. passed infectious diseases;
2. contact with infectious patients;
3. possibility of infection by a contact with a sick animal;
4. visits to epidemiologically dangerous regions;
5. passed vaccination;
6. housing and living conditions of the patient.

15. Most important bad habits in taking anamnesis vitae:
1. alcohol intake;
2. tobacco smoking;
3. hypodynamy;
4. overeating;
5. fidgeting;
6. gossips.

16. Objective examination consists of the following parts:
1. physical examination;
2. laboratory and instrumental examination;
3. main complaints and its detailing;
4. anamnesis communis;
5. anamnesis morbi.

17. Physical examination includes:
1. general inspection (survey);
2. assessment of general state of patient;)
3. special (local) inspection;
4. palpation, percussion and auscultation of respiratory, cardiovascular, digestive, and urinary systems;
5. X-ray and laboratory tests.

18. Medical deontology includes the problems of:
1. medical worker's responsibility for the life and health of the patient;
2. medical confidentiality;
3. relationships of medical workers to each other;
4. diagnosis of diseases;
5. treatment of patients.

19. Allergy anamnesis includes:
1. past allergy diseases;
2. past allergy reactions to food, medications, pollens, dusts, cosmetics, etc.;
3. familial predisposition for allergy reactions;
4. skin rash and itching;
5. passed infectious diseases.

20. Transfusiology anamnesis includes:
1. past transfusions of blood and blood substitutes;
2. adverse reactions after blood and blood substitutes transfusions;
3. past allergy diseases;
4. hereditary diseases in relatives of the patient;
5. past infectious diseases;
6. past surgery operations.

21. Medical diary contains:
1. new symptoms;
2. body temperature;
3. pulse rate;
4. blood pressure;
5. diuresis.

22. Medical diary of a patient in a serious state should be recorded:
1. once a day;
2. once a week;
3. regularly in two days;
4. two times a day;

5. every 2-4 hours.

23. Epicrisis of the case history includes:
1. basic complaints of the patient;
2. basic objective data;
3. course of the disease;
4. summary of the laboratory study and passed treatment;
5. recommendations for further rehabilitation after a discharge from the hospital.

24. Clinical diagnosis is supported by:
1. objective data of examination exclusively;
2. subjective dataexclusively;
3. laboratory and instrumental dataexclusively;
4. passed diseases;
5. subjective, objective and laboratory-instrumental data of examination.

CHAPTER 2. Objective Examination of a Patient. General Inspection (Survey)

Objectives: to enable students to learn –

1) Subject and purposesof the objective examination of a patient;
2) Purposes and rules ofa general survey (general inspection), andinterpretation of results;
3) Assessment of a consciousness state of the patient;
4) Evaluation and clinical significance of the patient's body-build (constitution) type;
5) Technique of evaluation and diagnostic value of the skin and the visible mucous membranes;
6) Rules and diagnostic value of the muscles, bones and jointsexamination;
7) Rules and diagnostic value of thermometry;
8) Assessment of a general state of the patient.

2.1. Objective examination

Another stage of examination is *anobjective examination* of the patient's condition at the present time (*status praesens*).

Objective examinationincludes two stages:

(1) physical examination includes a general and a local survey (inspection), palpation, percussion, and auscultation;

(2) additional methods of an objective examination include variable measuring (anthropometry, blood pressure, and others), laboratory (clinical,

biochemical, histological, microbiological, immunological, etc.), and instrumental (endoscopy, X-ray, radiology, ultrasound, functional and others) studies.

Objective examination reveals changes in the patient's body and deviations froma normal structure and a function of various organs that could not be sensed by the patient himself /herself.

Physical examination is the process of evaluating objective anatomic findings through the use of observation, palpation, percussion, and auscultation. The information obtained must be fully integrated with the patient's history and pathophysiology. Moreover, it is a unique situation in which both a patient and a physician understand that the interaction is intended to be diagnostic and therapeutic. The physical examination, thoughtfully performed, should yield 20% of the data necessary for the patient's diagnosis and management.

2.2. Approach to a general survey

Technique of a general survey (general inspection).

The inspection often precedes taking anamnesis, and begins with the first sight of the doctor on the patient. The examination data, combined with the questioning data, make it possible to construct a sufficiently substantiated diagnostic hypothesis.

The examination of the patient is the simplest and most natural method of the research. To obtain reliable results, it requires compliance with certain rules: lighting, in which the inspection is performed, inspection technique, inspection plan.

General inspection must be carried out with scattered daylight or white artificial light. Lighting can be direct or lateral. The patient should be wholly or sometimes partially exposed.

Inspectioncan be divided into a***general survey***to start the study, and a***local survey***of the parts of the body, organs and systems (chest, heart area and large blood vessels, abdomen, and others).

Assessment of consciousness (mental state) is the first part of general survey of the patient, and begins as rule simultaneously with the patient's inquiry (Table 2-1).As a result of the general survey, the general condition of the patient is determined by a number of signs: state of consciousness, position in bed, constitutional type and nutritional status of the patient, posture, gait, habitus. General survey includes necessarily the examination (inspection and palpation) of thyroid gland and lymphatic nodes, and taking body temperature. In addition, its assessment takes into account the functional indexes of the cardiovascular system, respiratory and other systems, as well as the results of laboratory and instrumental methods of examination.

Table 2-1. General survey plan

General survey includes assessment of:
(1) Consciousness (mental state); (2) Posture (position) of the patient; (3) Habitus, including body-build (constitution) type, height, and body weight, facial appearance; (4) Skin and mucosa examination; (5) Lymph nodes examination; (6) Thyroid gland examination; (7) Musculoskeletal system examination; (8) Body temperature; (9) General condition of the patient.

2.3. Consciousness (mental state) assessment

Assessment of consciousness (mental state) begins simultaneously with the patient's inquiry. Use the following headings:

- Appearance and behavior of the patient;
- Speech;
- Mood;
- Thought content;
- Abnormal beliefs;
- Abnormal experiences;
- Cognitive state;
- Intelligence;
- Insight and rapport;
- Specific tests of cerebral function.

State of consciousness (mental state)is evaluated on the following levels:

*Alertness*is characterized by the state of wakefulness and awareness in which a patient correctly orients in environment, time and his/her own personality, or by normal sleep from which he/she can be light awake.

Lethargy includes a severe drowsiness in which the patient can be aroused by moderate stimuli and then drift back to sleep.

Obtundation is a state of stun, when the patient is poorly oriented in the environment and sluggish, and not quite clearly with a great difficulty answers the questions.

*Stupor*is a non-responsive state, from which the patient may be derived only a loud shout or mechanical stimulus, but then returns to his/her previous state.

*Coma*is characterized by a complete loss of consciousness, lack of reflexes, possibly involuntary urination and defecation. Only the functions

of breathing and blood circulation are preserved. Coma occurs in diabetes (diabetic comas), renal failure (uremic coma), liver encephalopathy (hepatic coma), respiratory failure (hypoxic coma), cerebrovascular disorders (cerebral coma).

Normally, a person has a state of alertness (clear consciousness).

The ***Glasgow Coma Scale or GCS***is aneurologic scale that aims to give a reliable, objective way of recording the conscious state of a person for initial as well as subsequent assessment(Table 2-2) (Teasdale G, Jennett B., 1974). A patient is assessed against the criteria of the scale, and the resulting points give a patient score between 3 (indicating deep unconsciousness) and either 14 (original scale) or 15 (the more widely used modified or revised scale). GCS was initially used to assess a level of consciousness after head injury, and the scale is now used to all acute medical and trauma patients. In hospitals it is also used in monitoring chronic patients inan intensive care.

***The GCS measures the following function*:**eye opening, verbal response, and motor response.

Glasgow Coma Scale classifies a brain injury as:

- ***Severe, with GCS < 8–9 points***
- ***Moderate, GCS - 9–12 points***
- ***Minor, GCS ≥ 13 points.***

Table 2-2.Glasgow Coma Scale

Eye Opening (E)	Verbal Response (V)	MotorResponse (M)
4 = spontaneous 3 = to sound 2 = to pressure 1 = none NT = not testable	5 = orientated 4 = confused 3 = words, but not coherent 2 = sounds, but no words 1 = none NT = not testable	6 = obeys a command 5 = localizing 4 = normal flexion 3 = abnormal flexion 2 = extension 1 = none NT = not testable
Total=E+V+M		

*Agitated mental disorders*may also developcommonly in diffuse brain lesions, for example, against the background of intoxication (alcohol, lobar pneumonia, etc.). They are characterized by pathological excitation of the central nervous system, delirium and hallucinations. Hallucinations are false, inadequate perception of reality by the senses. Patients see, hear, feel what is not real. There are visual, auditory and tactile hallucinations. Delirium is hallucinatory confusion with a predominance of true visual hallucinations and illusions, imaginative delusion.

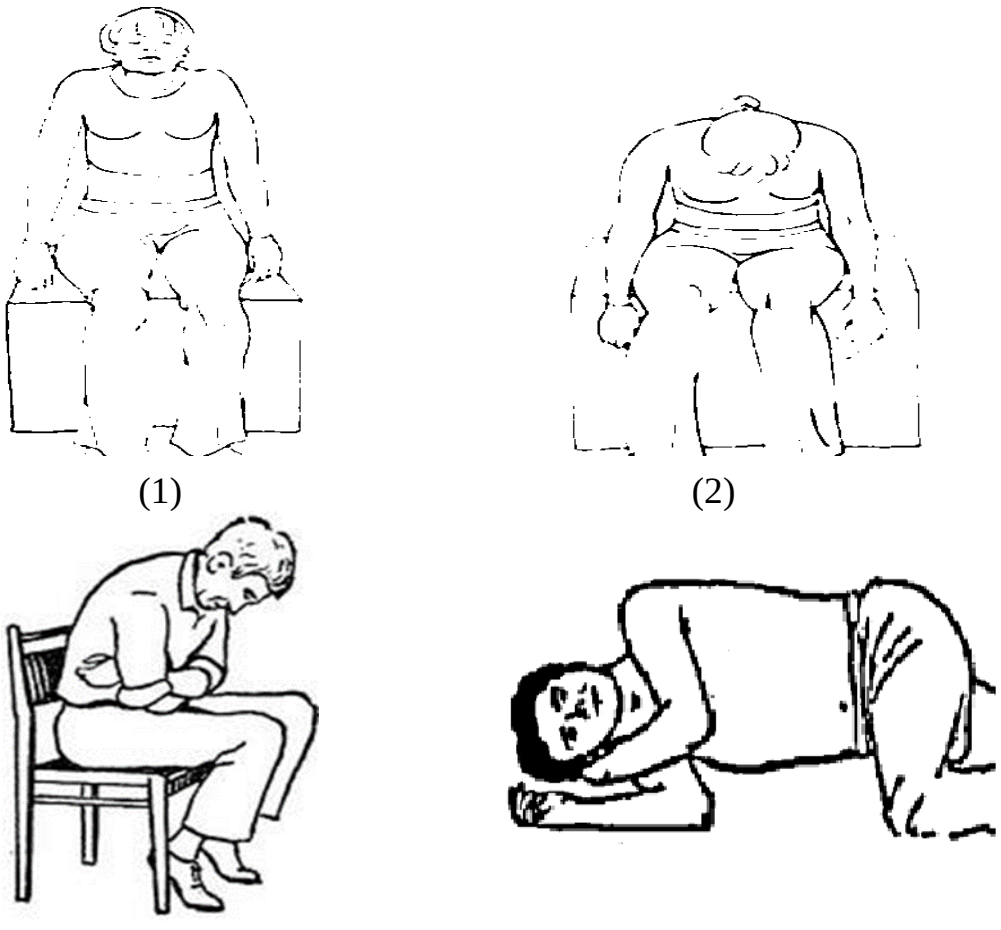
2.4. Assessment of the patient's position (posture)

A patient's position may be active, passive and forced.

Normally, a person has an active position - the most natural under these conditions, easily changed depending on the desire. In most cases, the patient's position also remains active. If it is impossible to get out of bed, the situation is assessed as active in bed when the patient could change it.

In the *passive position*, a patient cannot spontaneously change it because of the severity of the condition (with loss of consciousness, tetraparesis, fracture of the spine).

Forced position is a certain position that takes the patient to facilitate his state of health. *Forced position of the patient may be vertical (sitting, standing) and horizontal (recumbent, lying down)*(Fig.2-1).



(1) (2)

(3) (4)

Fig. 2-1. Variants of forced position:

1) the orthopnea with support on hands in attack of bronchial asthma;
2) the orthopnea with freely hands in the left ventricle heart failure;
3) the position leaning forward to the affected side of the chest and pressing hands to the sore place in dry pleurisy;
4) the lateral recumbent position with bent knees in the stomach peptic ulcer.

A forced sitting position (*orthopnea*) occurs when patients have dyspnea in case of the heart failure and the acute lung diseases (bronchial asthma attack, pneumothorax). The patient in dry pleurisy takes the forced sitting position leaning to the affected side of the chest and pressing his/her hand to the sore place is typical. A lying (recumbent) forced position can be on the back, on the stomach, on one or another side. Forced position on the back is observed in the onset of severe abdominal pain (in appendicitis, peritonitis). A position lying on one side (lateral recumbent position) with bent knees is a characteristic in the peptic ulcer of stomach ulcer with a pain syndrome. A forced position on the abdomen is observed in the diaphragmatic pleurisy, tuberculosis of the spine, solaritis (inflammation of the solar plexus). A position on the affected side of the chest is most often in patients with lobar pneumonia, pleurisy, suppurative process in the lungs.

The patient stops and freezes with a painful attack of angina pectoris (a "symptom of the shop-window") stopping physical activity.

2.5. Habitus

The concept of habitus (general appearance of a patient according the medical pointofview) includes ***body-build type, constitution, height, and body weight.***

2.5.1.Body-build typeand constitution of the patient

Body-build type **-** properties and characteristics of the body parts, as well as features of the development of bones, fat and muscle tissue.

Constitution (somatotype) of a person is not only the generalized morphological and functional characteristic of an individual, and reflects the features of the body-build, as well as psychophysiological, metabolic differences, and predisposition to the certain diseases.

The body-build of a person changes throughout his/her life, while the constitution is genetically determined, and is his/her constant characteristic from birth to death.

There is a certain correlation between the external forms of the body and its internal structure, as well as between the physiological and morphological properties of the body.

Classification adopted by M. Chernorutsky and W. Sheldon differentiates between the following ***three main constitution types: asthenic*** *(ectomorph, oligomorph****),*** ***hypersthenic*** *(endomorph,brachymorph****),*** ***and normosthenic*** *(mesomorph)*.

A well proportioned body-buildis characterized by normal weight and height that roughly equals to the fingertip-to-fingertip measurement of the outstretched arms, and twice the leg length from pubis to heelin.

Assessment of a body-build type of the patient is based on a body mass index (a ratio of height and weight), lengths of limbs, shape of the head,

neck, and chest, development of muscle mass and subcutaneous fat tissue (Fig.2-2).

Hypersthenic(endomorph)type characteristics are transverse dimensions over prevail longitudinal ones, wide and short body, medium or short height, often increased weight, strong muscles, wide shoulders, relatively short limbs, rounded head, short and thick neck, wide chest, evenly protruding abdomen. These individuals have slightly increased function of gonads (sex glands) and reduced – of thyroid gland. More often, there is a violation of lipid metabolism, a tendency to arterial hypertension and coronary artery disease, gallstones and urolithiasis.

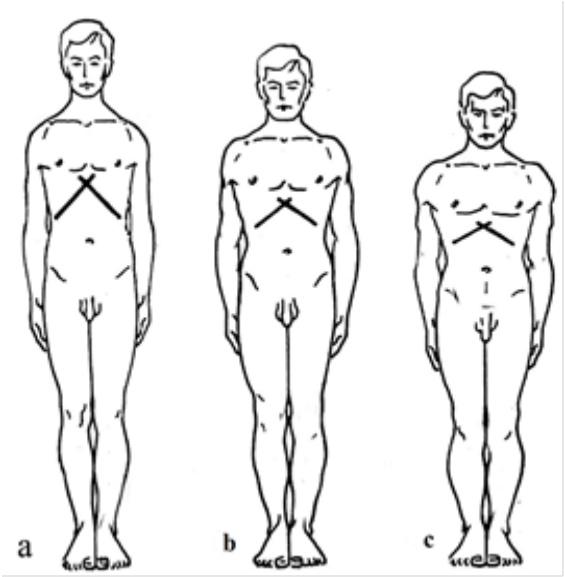


Fig. 2-2. Typesofbody-build
(a) asthenic (ectomorph); b) normosthenic (mesomorph);
c) hypersthenic (endomorph)

Asthenic (ectomorph) type has a narrow and elongated body, above average height, thin and narrow shoulders, long limbs, oblong head, elongated and thin neck and chest, weak muscles. Abdomen isdrawn in the upper part, and is protruding slightly in the lower one. Often there is a lowering kidneys, liver, and stomach (visceroptosis, or enteroptosis).These people are easily excitable. They may be with an increase in the thyroid function, and some decrease in the function of the gonads. Asthenic type predisposes to the diseases of the lungs and gastrointestinal tract.

Normosthenic (mesomorph) type occupies an intermediate position between the hypersthenic and asthenic constitutional types. The normosthenic type is a proportionaly built person with well-developed muscles, broad shoulders, a convex chest, a small elastic abdomen and an average length of limbs. Normosthenic type people are energetic, confident in their abilities. They have a tendency to the diseases of the upper respiratory tract, musculoskeletal system, neuralgia, and coronary arteriosclerosis.

2.5.2. Nutritional state

Body Mass Index (BMI) is a simple index of weight-for-height that is commonly used to classify underweight, overweight and obesity in adults. It is defined as the weight in kilograms divided by the square of the height in meters (kg/m^2).BMI values are age-independent and the same for both sexes (Table 2-3) (WHO, 1995).

Waist–hip ratio or waist-to-hip ratio (WHR) is the ratio of the circumference of the waist to that of the hips. This is calculated as waist measurement divided by hip measurement (W/H). WHR is used as a measurement of obesity, which in turn is a possible indicator of other more serious health conditions. The abdominal obesity is defined as a waist–hip ratio above 0.90 for males and above 0.85 for females, or a body mass index (BMI) above 30.0. Women with waist–hip ratios of more than 0,8 and men with more than 1.0 are at an increased health risk because of their fat distribution.

Table 2-3. The International Classification of adult underweight, overweight and obesity according to BMI(WHO, 1995)

Category	Body Mass Index (kg/m^2)
Underweight	<18.50
Veryseverelyunderweight	<16.00
Severelyunderweight	16.00 – 16.99
Mildunderweight	17.00 – 18.49
Normal (healthy weight)	18.50-24.99
Overweight (Pre-obese)	25.0-29.99
Obese	≥30.00
Obese Class I (Moderately obese)	30.00-34.99
Obese Class II (Severely obese)	35.00-39.99
Obese Class III (Very severely obese)	≥40.00

Obese and pre-obese patients have higher proportion of the body fat and an increased risk for cardiovascular diseases (ischemic heart diseases, arterial hypertension, hemorrhagic stroke) and diabetes mellitus. A history of weight gain or loss can be checked by observation, remembering that fluid retention (*edema*) will increase weight. BMI may be higher than norm in case of edema, and in sportsmen and in laboring workers because of enlarged skeletal muscles.

Underweight might be secondary to or symptomatic of an underlying disease (malignancies, tuberculosis, AIDS, hyperthyroidism, renal and hepatic failure). Obvious weight loss, even when food intake has been increased, is a feature of thyrotoxicosis and diabetes mellitus. Psychogenic loss of appetite usually affecting girls (*anorexia nervosa*) causes extreme emaciation while physical activity remains unimpaired.Unexplained weight loss may require a

careful medical examination.Severely underweight individuals may have a poor physical condition and a weak immune system, leading them to severe infection diseases. It can also cause anemia and osteoporosis.

2.5.3. Facial appearance

During general survey, it is important to assess the expression of the patient's face.The expression, and particularly the eyes, indicates real feelings better than words. Some diseases, for example Parkinson's disease, depression, hypothyroidism, thyrotoxicosis, acromegaly, produce characteristic facial appearances.A facial expression is an important diagnostic featurein a number of diseases. Among the most common are the following:

"Mitral face" (*facies mitralis*) is the characteristic of patients in mitral stenosis: red-cyanotic “blush” cheeks against the background of pallor skin of face, and cyanosis of the lips, nose and ears;

"Corvisart's face " (*facies Corvisari*) - a sign of severe chronic heart failure: yellow-pale facial skin with a blue tint, puffy, dull eyes, cyanosis of the lips, half-open mouth, and pronounced dyspnea;

Face in Itsenko-Cushing syndrome (hypercorticism): round, moon-shaped, red skin, shiny face, and hirsutism (growth of facial hair in women);

"Facies Basedovica" in a person with *Graves disease* (thyrotoxicosis, or hyperthyroidism): lively, rich by facial expressions the face, marked exophthalmos (protrusion of eyes); eyes are glittering and express fright or surprise, sometimes "frozen horror";

Myxedamatous face in myxedema (hypothyroidism): blunt, puffy, with sluggish facial expressions, edematous, an indifferent look, narrow eyes slit;

"Facies acromegalica" – is the face of patients in acromegaly (an increased production of growth hormone of the anterior pituitary gland): a sharply increased size of the nose, lips, brow, mandible, tongue;

"Facies nephritica" is the face in the kidney diseases: pale, puffy, swelling of the eyelids, "bags" under the eyes;

Face in tetanus (risussardonicus): a violent, "sardonic smile" - lips stretched in a smile, and the forehead creases, as if sorrow;

"Facies Hippocratica" - is typical for patients in peritonitis (inflammation of the peritoneum) or agonal condition: pale with a blue tint, sharpened cheekbones and nose, sunken eyes, expression of suffering, drops of sweat on the forehead;

"Facies febrilis" is a face in lobar pneumonia and high fever - one-sided redness on the side of the inflamed lung, the wings of the nose participate in the act of breathing, the eyes are shiny, the expression is agitated, and herpetic eruptions may be;

"Facies phthisica" is a face in pulmonary tuberculosis: a pale, thin face with a bright blush on the cheeks, shiny eyes;

Face in chronic alcoholism - red with dilated veins on the cheeks and nose, "empty" look.

*Parotid swelling*in the region of the parotid glands(lies wedged between the sternocleidomastoid and the mandible)are obvious on inspection of the face. It is typical in acute infectious parotitis and Syogren's syndrome.

The ***cheeks*** give information regarding the patient's health: in anemia and hypopituitarism they are pale; in the nephrotic syndrome they are pale and puffy; in cases of mitral stenosis there is sometimes a bright circumscribed flush over the malar bones; in many persons who lead an open-air life they are red and highly coloured; in a congestive heart failure they may also be highly coloured, but the colour is of a bluish tint which cannot be mistaken for the red cheeks of weather-beaten people.

2.6. Skin and mucosa examination

A study of the skin and mucous membranes (conjunctiva, oral mucosa) is produced in parallel, assessed:

- colour,
- humidity (moistness),
- elasticity (turgor),
- temperature,
- subcutaneous fat,
- presence of edema, focal lesions (rashes, scars),
- dermal appendages (hair and nails).

The colour of the skin and mucous membranes in individuals of the Caucasian (Europoid) race is estimated as pale-pink. It depends on the thickness of the skin, transparency, blood filling, and the amount of pigment.

The skin is felt dry or slightly moist, warm, smooth, elastic (collected by two fingers fold quickly straightens). Mucous membranes should be moist, their surface smooth. Constitutional pale skin is combined with normal pale pink color of mucous membranes. With anemia, the skin and mucous membranes become pale.

Pale skincolor is associated with insufficient filling of the cutaneous blood vessels (due to spasm of blood vessels of the skin or emptying them in massive bleeding, collapse, and acute vascular insufficiency). It is observed in anemia, kidney disease, aortic heart valves diseases. In B_{12}-(folate)-deficiency anemia, pale skin becomes yellowish, in iron-deficiency anemia - greenish, in cancer patients - earthy, malaria - ash or brown, in infective endocarditis - the color of "coffee with milk".

In anemia, the pallor of the skin is combined with the pale color of the mucous membranes (conjunctiva of the eyes, soft and hard palate, gums, tongue).

Pronounced pallor of the skin occurs in the development of subcutaneous edema, squeezing the capillary network and pushing it away from the surface of the skin (in renal origin of edema).

Yellow colorof the skin andmucous membranes - *jaundice (icterus*). Icterus firstly appears on the mucous membranes of the hard palate and eyes with an increased bilirubin in the blood up to 30 mcmol/l), and only at a higher level of bilirubin – on the skin. Inspection for discovering icterus is carried out only in daylight.

Cause of jaundice is an accumulation of bile pigments in the skin and mucuos membranes due to (1) *posthepatic (mechanic, obstructive) jaundice* as a result of blockage of the common bile duct (by stone in gallstone disease, by cancer of the pancreas head), (2) *hepatic (parenchymatous) jaundice* as a result of disordered bilirubin metabolism and secretion in hepatitis, cirrhosis of the liver; (3) or in *prehepatic (hemolytic jaundice)* as a result of hemolysis (destruction) of red blood cells in hemolytic anemia.

A pale lemon-yellow tint is a characteristic of prehepatic jaundice. There is a dark-yellow or orange tintin hepatic jaundice. In posthepatic jaundice there are green tint and scratch marks from itching evoked by bile salts.

In rare cases, yellowness may be due to carotenemia (elevated blood serum concentration of vitamin A). A yellow skin color can be the result of taking in large doses of certain drugs (quinine, etc.), as well as food products (carrots, citrus). However, the sclera of the eyes are not stained in these conditions.

Cyanosis (blue shade of skin and mucous membranes) may be diffuse and local.

Central (or diffuse) cyanosis depends on insufficient arterialization of the blood in diseases of the lungs. In respiratory failure, mild cyanosis appears only with exercise. Permanent cyanosis with a purple tint (due to compensatory erythrocytosis) is characteristic of respiratory failure of II-III degree. It is evident from the moderate cyanosis of face and upper part of the chest up to the diffuse cyanosis.

Peripheral cyanosis (acrocyanosis) is associated with impaired blood circulation in heart failure. It is detected on the tip of the nose, ears, fingers, lips. The skin in these places is cold to the touch.

Local limited cyanosis appears on one part of the body: on the face and neck – in a mediastinal tumor, on one limb – in thrombosis of the corresponding vein).

Red skin color (hyperemia) can occur under the influence of mental excitement, excessively high air temperature, fever, alcohol intake, carbon monoxide poisoning. The face is hyperemic in patients with arterial hypertension. In chronic alcoholism, it is constantly marked crimson-red color due to the persistent dilation of the capillary network, especially on the back and tip of the nose, and cheeks.

In *polycythemia* (a disease characterized by high levels of red blood cells and hemoglobin), the face is red, with a cherry tint;the blood vessels of the eyes conjunctiva are dilated.

Pigmentationis most commonly racial. The pigmentation of *Addison's disease (adrenal insufficiency)* affects the buccal mucous membranes as well as exposed skin and parts subject to the friction. Dark red or brown skin is characteristic of adrenal insufficiency. Hyperpigmentation of the breast nipples and the areola in women, pigmented patches on the face and the white line on the abdomen are signs of pregnancy. Foci of depigmentation of the skin (*vitiligo*) also occur.

Turgor (elasticity) of the skin depends on: the degree of development of fatty tissue, moisture content, blood supply, the presence of elastic fibers. Preserved elasticity (turgor) of the skin is defined when taken by fingers a skin fold is quickly smoothed. Skin turgor decreases in elderly persons (over 60 years), with a sharp depletion, dehydration (vomiting, diarrhea), circulatory disorders.

Moistness of skin is detected on the touch. High moistness is physiological in the summer in the heat, with increased muscle work, excitement) and pathological with severe pain, attacks of suffocation, fever, severe intoxication, thyrotoxicosis, tuberculosis, lymphogranulomatosis, heart failure).

Dry skin is noted with the loss of a large amount of fluid (in vomiting, diarrhea, diabetes mellitus and diabetes insipidus, myxedema, scleroderma, chronic nephritis).

Hair. Violation of hair growth often indicates a pathology of the function of the genitalia and other endocrine glands. Deficient hair growth or absence of hair (alopecia) is characteristic of myxedema, liver cirrhosis (hypothyroidism), eunuchoidism, and infantilism. Hair growth by a male type (hirsutism) is observed in women with Cushing's disease and adrenal tumors. Hair is also affected in some skin diseases.

Nails. Normal nails are smooth and pink. Thin, brittle, flaking nails, with depressions, transverse and longitudinal striations on them are observed in iron-deficiency and vitamin B12 –deficiency anemias, hypo- and hyperthyroidism. Spoon-like deformation of nails (*koilonychia*) is typical of severe iron-deficiency anemia. Nails are deformed in the form of “watch glasses” (or *nail clubbing*)in chronic suppurative lung diseases (abscesses, bronchiectasis) and in chronic circulatory failure.

Development of subcutaneous fat layer may be normal, increased or decreased. The fat layer can be distributed equally, or its deposition occurs only in certain areas. The thickness of the subcutaneous fat layer (degree of fatness) can be judged by palpation or caliper measuring skin-fold.

For these purposes, two fingers of the hand take a fold of theskin with subcutaneous tissue on the outer edge of the rectus abdominis muscle at the level of the navel, the lateral surface of the shoulder or at the angle of the shoulder blade and measure its thickness with a caliper. Traditionally, the thickness of the skin fold should be within 2 cm, the thickness less than 1 cm is regarded as a decrease, and more than 2 cm – as an increase in the devel-

opment of the subcutaneous fat layer.

Modern skinfold estimation methods are based on a***skinfold test***, also known as a*pinch test*, whereby apinchof theskin is precisely measured bycalipersat several standardized points on the body to determine thesubcutaneousfat layer thickness (Fig. 2-3).Triceps skinfold is the most common place for measuringskinfold thickness.

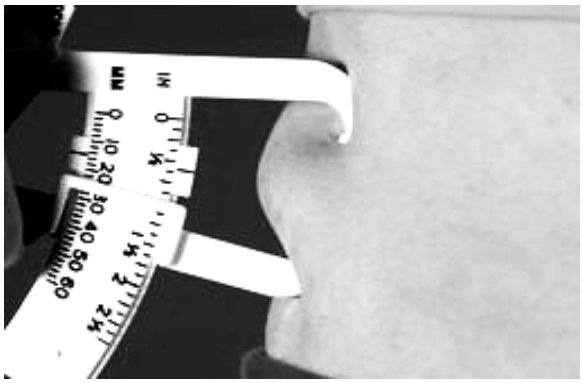


Fig. 2-3. Skin measurement by caliper

Triceps Skinfold: Along the midline on the back of the triceps of the right arm, determine the midpoint located between the top of the acromial process (top of the shoulder) to the bottom of the olecranon process of the ulna (elbow). Pinch the skin so that the fold is running vertically.

The TSF varies from 0.5 to 2.5 cm (average, 1.2 cm) in healthy adult males and from 1.2 to 3.4 cm (average, 2.0 cm) in healthy adult females. A patient whose TSF < of the norm is considered to have depleted body fat stores; one whose TSF is 100% above the norm is considered obese.

*Pectoral (Chest) skinfold:*Using a line from the fold of the axillary (armpit) to the nipple, determine the midpoint. Pinch the skin with the fold running in the same direction of the line.

Abdominal Skinfold: Select a site on the right side about 1 inch (2.5 cm) lateral from and 0.5 inch (1.3 cm) below the umbilicus. Lift a horizontal fold of the skin for the measurement

Suprailiac Skinfold: Determine the midaxillary line and palpate for the iliac crest (top of the hip bone). Grasp the skin that follows the natural fold which will follow a line of approximately from the suprailiac to the umbilicus (bellybutton), an angle of approximately 30 degrees.

*Thigh Skinfold:*Determine the midline of the front of the thigh and measure midway between the inguinal crease (the natural crease between the thigh and the hip which is at an approximate 45 degrees) and the top of the patella (kneecap). Grasp a vertical fold of the skin for the measurement.

Increased development of the subcutaneous fat is mentioned in various forms of obesity (alimentary-exogenous, endocrine, etc.). Insufficient development of subcutaneous tissue is due to the constitutional features of the

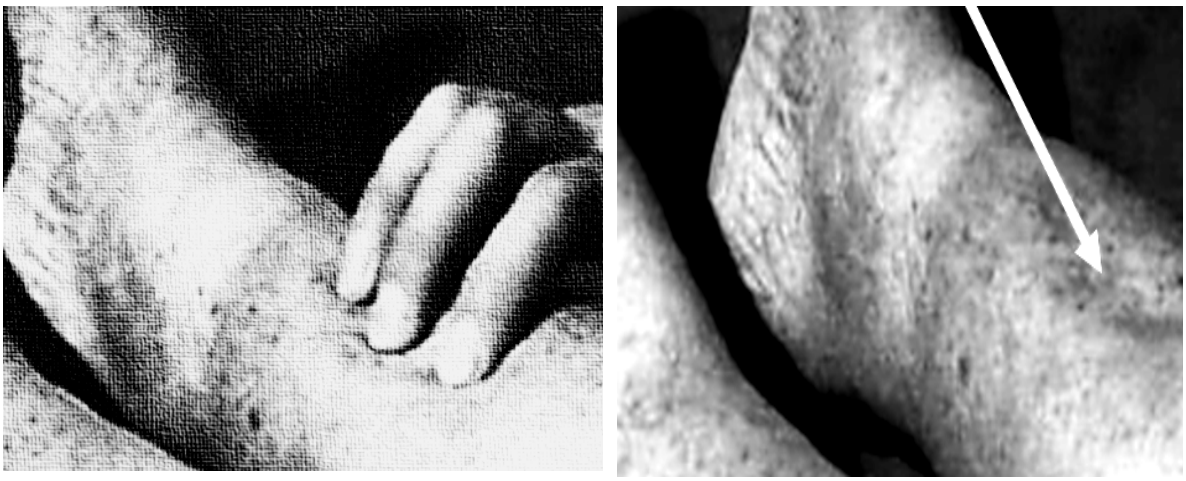
body (asthenic type), malnutrition, and dysfunction of the digestive system. The extreme degree of malnutrition is called cachexia. It is observed in advanced forms of tuberculosis, malignant tumors.

Edemais an accumulation of the non-inflammatory fluid (transudate) in tissues and interstitial spaces. *Edema may be local and generalized*.

Local edema may be associated with difficulty in the outflow of the venous blood during compression or thrombosis of the vein. "*Angioedema*" is caused by violation of vegetative innervation and capillary permeability.

Generalized edema develops due to the venous congestion ("warm" edema) or disordered the colloid-osmotic equilibrium (renal and hungry edema). Edema, and on the face, and especially paraorbital, clearly indicate renal pathology. On the legs edema often appear in heart failure. In severe cases, edematous fluid accumulates in serous cavities: pleural (*hydrothorax*), pericardial cavity (*hydropericardium*), abdominal cavity (*ascites*).

Anasarca is a generalized edema in combination with accumulation of fluid in serous cavities.Edema can be recognized by the pallid and glossy appearance of the skin over the swollen part, by its doughy feel, and by the fact that it pits on finger pressure (Fig. 2-4).



(1) (2)

Fig. 2-4. Identification of edema on the tibia:
(1) pressure ;(2) pitting at the site of the pressure (see arrow)

In bed-bound patients edema often appears first over the sacrum. To recognize pitting edema, it is important to press firmly and for a sustained period, and the "pit" is as easily felt as seen. The edema of lymphatic obstruction and in hypothyroidism (*myxedema*) donot pit on pressure.

Subcutaneous emphysema is uncommon, but if present can be recognized by the crackling sensation produced by lightly compressing the part affected.Subcutaneous emphysema occurs when air gets into tissues under the skin. This most often occurs in the skin covering the chest wall or neck, but

can also occur in other parts of the body.Subcutaneous emphysema can result from a spontaneous pneumothorax,an injury to the thoracic cavity, sinus cavities, facial bones, barotrauma.

2.7. Examination of the lymph nodes and the thyroid gland

Generallynormal ***lymph nodes***are not detected visually or by palpationin adults.As an exception in healthy adults, single node ofaxillary, cervical, submandibularlymph node groups may be palpated no larger than 1 cm, mobile, painless and not soldered together.

Lymphadenopathy – is enlargement of lymph nodes. Lymph nodes as one of the components of the peripheral immune system increase in a variety of infections, malignancy, autoimmune and metabolic disorders.

Diagnostic features of lymphadenopathy are patient's age, physical characteristics and location of the lymph node, clinical features, combined with lymphadenopathy.In children, the lymph nodes respond by hyperplasia to the slightest stimulus.Lymphadenopathy of adults under the age of 30 years is benign in 80% of cases and after 50 years - benign in 40% of cases.

The method of lymph nodespalpation is described in the textbook “Propedeutics of Internal Diseases: Part II. Unit VII. Blood System Examination”).

The ***thyroid gland*** normally lies just caudal to the thyroid cartilage in the anterior neck. This location allows an examiner to inspect and palpate this bilobed structure. *Normally, thyroid gland volume does not pre-exceed the volume of the distal phalanx of the thumb of a patient.*

Enlarged thyroid gland is defined by the term *goiter*.Enlargement of the thyroid gland most commonly results from increased pituitary secretion of TSH (thyrotropin)or lymphocyte production of TSH-like immunoglobulins. In addition, a number of inflammatory, infiltrative, and neoplastic diseases can cause goiter. Physical examination enables the clinician to differentiate among these possibilities.

The methods of the thyroid glandpalpation is described in in textbook “Propedeutics of Internal Diseases: Part II. Unit VIII. Endocrine System Examination).

2.8. Musculoskeletal system examination

The musculoskeletal system includes the muscles, bones, joints, and soft tissue structures such as tendons and ligaments.

2.8.1. Muscular system

Study of muscles includes:

- estimation of the shape, volume and consistence of muscles;

- detection of pain at palpation;

- estimation of muscle tone and strength.

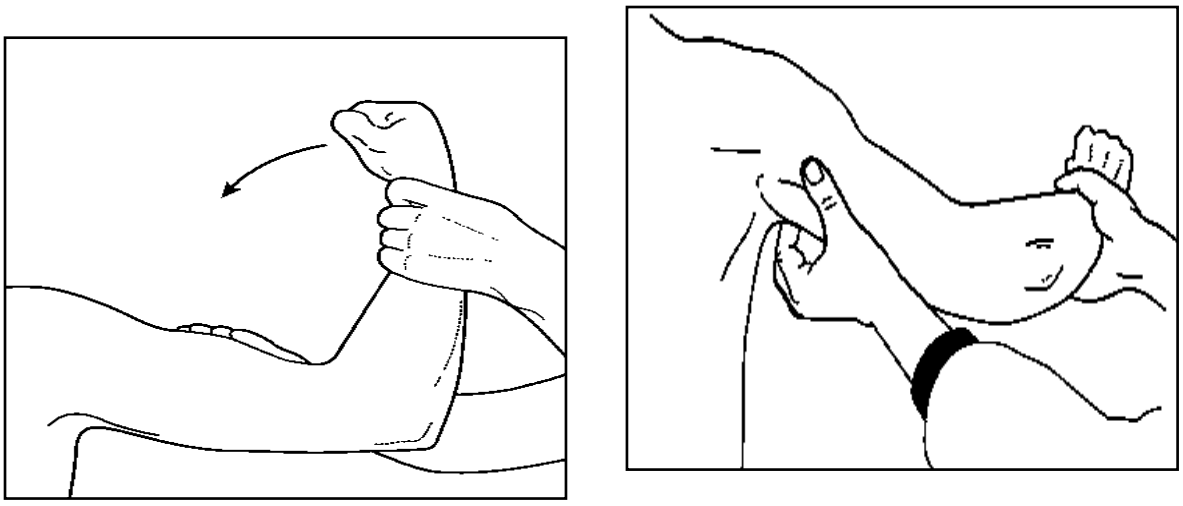
Remember that although muscle wasting may be due to the*primary*

muscle disease (e.g. polymyositis), it is more commonly secondary to *disuse*, perhaps because of a painful joint, or to *neuropathy* due to the nerve root compression or peripheral neuropathy.

Muscles may be developed well or weakly, their tone – normal, high or low. They may be painful, often with tonic or clonic convulsions. Well-developed muscles present in people engaged in physical labor, sports. Malnourished and seriously ill patients have severe atrophy of muscles. Unilateral muscle atrophy develops after injuries of the limbs, and especially in lesions of the nerves. To detect unilateral muscle atrophy, it is necessary to measure the volume of healthy and affected limbs at the same level in centimeters.

Wasted or atrophic muscles are not only smaller, but softer and more flabby than normal when they are contracted. When muscular wasting is accompanied by fibrosis, as in muscular dystrophy, polymyositis or eosinophilic myositis, the muscles feel hard and inelastic.

Muscular tone refers to the state of muscle tension or contraction. Increased tone is called *hypertonia* and reduced tone *hypotonia*. In *spasticity* tone is increased in proportion to the speed of passive stretch, whereas *rigidity* is an increase in tone at rest. Tone is assessed by taking a limb and moving it passively back and forth at different rates. By giving passive movements to all joints one after another and by palpating the muscles, it is possilbe to determine their tone (Fig.2-5). Important joint tests include pronation and supination at the wrist, and flexion and extension at the elbow and knee.



(A) (B)

Figure 2-5. Testing muscles: (A) the biceps; (B) the triceps.

The *strength of the muscles* of the arms is examined with the help of dynamometer or asking the patient to squeeze the doctor's hands simultaneously with both hands, and the difference in the strength of their pressure

determine the weaker muscles.

In the study of the shoulder flexors, the patient flexes the arm in the elbow joint and holds it, and the doctor tries to straighten it. The strength of the resistance on the affected side will be weaker.

For the study of shoulder extensors, the doctor tries to bend the patient's arm, which is bent in the elbow joint, held by him in this position. The strength of leg muscles is determined in the same way.

*Mid upper arm muscle circumference (MUAMC)*is used to estimate lean body muscle mass. It is derived from the TSF and the midarm circumference, which is measured at the same site as the TSF, with the patient's right arm in a relaxed position. The average midarm circumference (MC) is about 32 ± 5 cm for males and 28 ± 6 cm for females.Mid upper arm muscle circumference is calculated according to the formula:

$$\text{MUAMC (cm)} = \text{MC} - (\pi \times \text{TSF}).$$

Normal mid upper arm muscle circumference is in average is 25.5 cm in male and 23.0 cm in female. Body muscle mass depletion presents if mid upper arm muscle circumference <15.0 cm in male and <14.0 cm in female.

2.8.2. Bones and joints examination

Study of bones and joints includes:

- shape assessment of the bones, joints, spine,

- detection of pain at palpation,

- estimation of the volume and pain at physiological movements of the joints.

Normally, bones and joints have the regular symmetrical shape, the spine - the physiological curves (cervical and lumbar lordosis, thoracic kyphosis). The skin over them is pale pink, warm to the touch. Active and passive movements of the joints are not restricted in full volume, painless.

The hands of a healthy person are straight. The hands outstretched with palms turned up touching little fingers do not touch the hands in the region of the elbow. The legs of a normal shape while standing are touching in heels, internal ankles, calves, the entire inner surface of the thighs.

Vertebral column. The spine has four physiological curvatures: in the cervical and lumbar parts - convexity forward (cervical and lumbar lordosis), in the thoracic and sacrum parts - convexity back (thoracic kyphosis).

When examining the spine, it is necessary to pay attention to the presence of pathological deformities of it, mobility during flexion and extension, lateral movements, pains of the vertebrae.

A hump with bulging posteriorly (pathological kyphosis) can develop as a result of rickets, congenital dysplasia of the vertebrae, tuberculosis process. In pathologic curvatures of the spine, lordosis develops anteriorly, scoliosis aside. It is possible a combined lesion - *kyphoscoliosis*.

Bones.It is necessary to pay attention to bones shape (curvature, deformation), surface and painfulness. Curvature and deformation of bones occurs as a result of rickets, syphilis, osteomyelitis, and may be a manifestation of such diseases as osteochondrodystrophy, osteochondropathy, poorly consolidated fractures of bones. In patients with blood diseases, pain is noted when tapping in the sternum, ribs, tibia bone. Systemic bone loss is observed in multiple myeloma.

In chronic diseases of lungs (bronchiectasis, abscesses, tuberculosis), congenital heart diseases, infective endocarditis, the fingers of arms and feet acquire the appearance of "drumsticks" (thickening of the terminal phalanges), while the nails form the *"nail clubbing", or "watch glass" - the Pierre-Marie- Bamberger syndrome, or Hippocratic fingers* (Fig. 2-6).

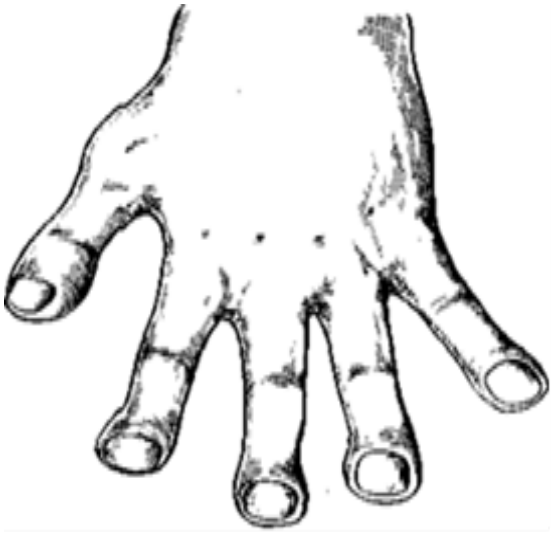


Fig. 2-6. Nail clubbing.

Joints.Changes in the musculoskeletal system are related primarily to the joints in the clinic of internal diseases.

These are signs of inflammation in joints: pain, local fever, redness, swelling, deformation and dysfunction.A disease may involve only one joint or a portion of the bone system (spinal tuberculosis).

Active and passive mobility is examinedfor evaluation of a jointfunctional state (Fig. 2-7).

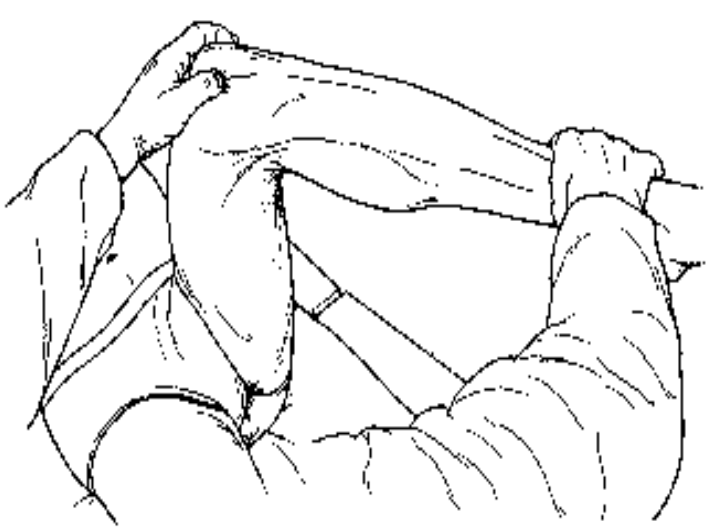


Fig. 2-7. Examination of passive mobility of the knee joint.

To determine the active joint mobility, the patient is asked to perform physiological movements in this joint. To assess passive joint mobility, a doctor sets his arms distal and proximal of the joint and himself performs movements in the joint. In both cases, the volume of movements and its tenderness are estimated.

*Arthrosis (loss of the articular surfaces of bones)*is characterized by limitation of active and passive movements.Reducing the active mobility only with the preservation of more volume passive movements is observed in arthritis, and periarthritis.

2.9. Thermometry

Depending on the measurement technique, there are 1) *contact thermometers*: electronic (digital) touch thermometers, chemical - disposable single-phase thermometers in the form of a color indicator strip, liquid crystal thermometers, liquid - mercury and gallium (10 minutes in armpit), 2) and *non-contactthermometers* (infrared - ear, frontal thermometer).Since 2011 year, mercury thermometers in European Union countries had not been used due to the high toxicity of mercury (if broken).

Thermometry is usually carried out in axillary space. Before placing the thermometer in the armpit, the skin would be wiped from sweat, as the liquid does not conduct heat from the skin to the thermometer, and the evaporation of sweat takes away some of the heat. The patient's hand must be tightly pressed against the chest in order to protect the temperature measurement area from the influence of the ambient air. *The duration of the temperature measurement in an axillary space is 10 minuteswhen applied the mercury or gallium thermometer,theliquid crystal thermometers - 3 minutes, an electronic touch thermometers – more than 30 seconds up to 5 minutes depending on the type of thermometer.* As soon as the temperature scale sign ° C or ° F on the display of the electronic thermometers stops flashing, the

measurement is completed. A sound signal (beep) also indicates the end of the measurement. The value of themeasured temperature is recorded.

The body temperature is measured, as a rule, 2 times a day: in the morning (from 6-00 to 8-00) and in the evening (from 17-00 to 19-00). With fever, there is a need for more frequent measurement of body temperature (every 2-3 hours).

On the basis of the multiple measurements of the temperature during the day, carried out for several days, *temperature curves* are obtained. The temperature curve of each patient is marked in a special temperature chart of his/her medical case history.

It is necessary to have a more frequent measurement of body temperature in fever (every 2-3 hours).The thermometer readings should be registered on a temperature chart for several days where the morning and the evening temperature is designated by dots. The dots are then interconnected to give a temperature curve which is characteristic of many specific diseases.

The temperature of a healthy person, measured in the armpit, varies normally within 36.0-36.9ºC.Depending on certain conditions, there may be physiological fluctuations in body temperature. Normal body temperature is measured in the rectum, vagina, inguinal fold, and oral cavity is 0.2-0.4 degrees higher than in the armpit.

During digestion, physical stress the temperature is increased by a few tenths of a degree.Among normal individuals, mean daily temperature can differ by 0.5°C (0.9°F), and daily variations can be as much as 0.25 to 0.5°C. The temperature slightly rises after meals and physical strain.In women, the body temperature is determined by the phases of the menstrual cycle: during ovulation it is increased by 0.6-0.8°C.

In children whose metabolic reactions are more intense, and the mechanisms of thermoregulation are still imperfect, there is a higher body temperature than in adults. In newborns, it reaches 37.2°C in the armpit.

Hypothermiais lowering body temperature by 1-2°C. It can be observed in severe circulatory failure, after heavy blood loss, with collapse, myxedema, cachexia.

Fever is a body temperature higher 37°C, measured in the armpit.

Types of fever according to the level of increased temperature:

- *subfebrile fever (37- 38°C),*
- *moderate fever (38-39°C),*
- *high fever (39-40°C), excessively high (40-41C),*
- *hyperpyretic (> 41°C) fever.*

Types of fever according to the character of temperature fluctuations are (Fig. 2-8):

- Constant, or continued, fever *(febris continua)* - is characterized by a high temperature; for 7-10 days (39-40ºC), the fluctuations between the

morning and the evening temperatures do not exceed 1C, typical in lobar pneumonia, typhoid fever (Fig. 2-7a,);

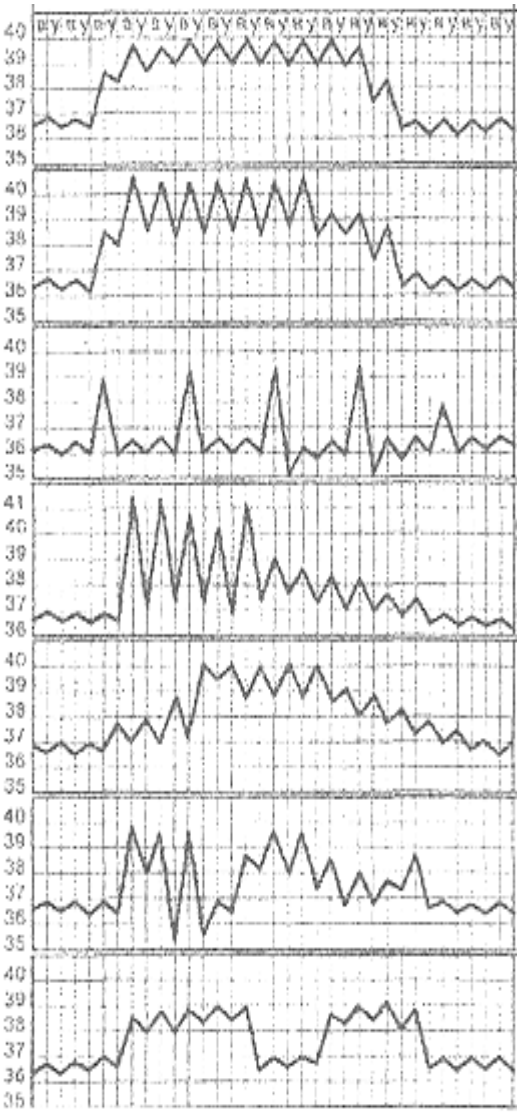
Remittent fever (*febrisremittens*) - daily fluctuations in temperature up to 2-3C, and the morning temperature does not reach the norm, typical in purulent diseases, pneumonia (Fig. 2-7b);

- Intermittent fever (*febrisintermittens*) - difference between the morning and the evening temperatures up to 2-3C, but in contrast to remittent fever the morning temperature is always below 37C, typical in malaria (Fig. 2-7c);

- Septic, or hectic, fever (*febrishectica*) - fluctuations in temperature reach 2-4C; it is accompanied by chills and profuse sweating, typical in sepsis, severe course of tuberculosis (Fig. 2-7d);

- Recurrent fever (*febrisrecurrens*) - body temperature increases for a few days, then period of temperature normalization, and further a new rise is noted, typical in recurrent typhus (Fig. 2-7g);

- Wave-like, or undulating, fever (*febrisundulans*) - is characterized by by a gradual daily rise in temperature, and then by the same gradual descent, after which in a few days it begins to rise again, typical in lymphogranulomatosis, brucellosis).



(a) Febris continua

(b) Febrisremittens

(c) Febrisintermittens

(d) Febrishectica

(e) Febris inverse

(f) Febrisatypica

(g) Febrisrecurrens

Fig. 2-8. Types of fever.

In its development, fever has three stages(Fig. 2-8):

Stage I (stadium incrementi) - a gradual rise, accompanied by sharp chills, blue lips, headache, poor health.

Stage II (stadium fastigii) - maximum fever, headache, dry mouth, hyperemia of the face, skin, delirium, hallucination.

Stage III (stadium decrementi) - lowering temperature. There are critical (sharp) or lytic (gradual) temperature drop.

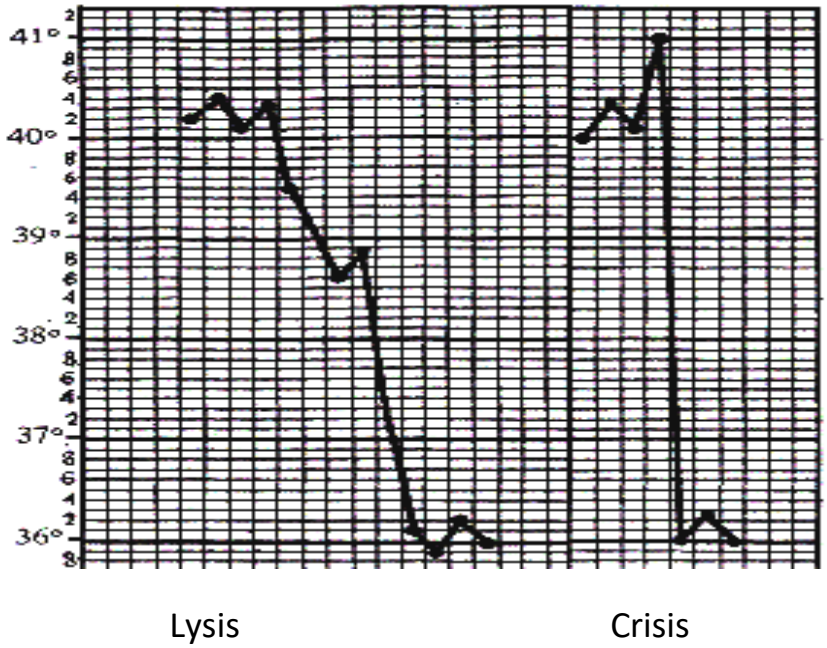


Fig. 2-8. Course of fever.

2.10. General condition of a patient

The state of the patient's health and general condition of the patient are not the same. *General condition* of a patient canbeestimatedinthe following degrees of assessment*: satisfactory, moderate severity, and severe (serious) condition* (Table. 2-4).

Assessment of the general condition of the patient is based on such objective criteria as:

- state of consciousness (mental state),
- position of the patient,
- -habitus bearing, gait, a look, state of feeding,
- body temperature,
- parameters of activity of cardiovascular system (pulse, heart rate, BP),
- parameters of activity respiratory organs (rate and character of respiration),
- functions of excretory system (especially 24-hours volume urination).

The severity of the patient's condition is determined first of all by the state of consciousness, positions in bed and changing the vital signs (pulse, heart and respiratory rate, BP, body temperature, 24-hours volume urination).

Table 2-4.Assessment of the general condition

Assessment of the general condition	Characteristics of general condition
Satisfactory condition	Patient is conscious and in active position but may be uncomfortable. Vital signs (body temperature, pulse rate, respiration rate, blood pressure, daily diuresis) are normal or stable
Moderate severity condition	Patient is seriously ill. Clear (*alertness*) or deranged consciousness (lethargy), active or forced position, moderate nutrition disorders may be. Vital signs may be changed (body temperature, pulse rate, respiration rate, blood pressure, daily diuresis)
Severe (serious) condition	Patient may be unconscious (*coma*) or in deranged consciousness (*obtundation*, *stupor*). Forced or passive position, nutrition disorders may be. Vital signs are unstable and changed significantly (body temperature, pulse rate, respiration rate, blood pressure, daily diuresis)

2.11. The key points of the theme "Objective Examination of a Patient. General Inspection (Survey)"

Objective examination includes two stages:

(1) physical examination includes a general and a local survey (inspection), palpation, percussion, and auscultation; and

(2) additional methods of objective examination include variable measuring (anthropometry, blood pressure, and others), laboratory (clinical, biochemical, histological, microbiological, immunological, etc.), and instrumental (endoscopy, X-ray, radiology, ultrasound, functional and others) studies.

Survey (inspection) *can be divided into* ***a general survey****, which is made at the beginning of the study, and* ***a local survey*** *of parts of the body, organs and systems (chest, heart area and large blood vessels, abdomen, and others).*

Assessment of consciousness (mental state) is the first part of general survey of the patient, and begins simultaneously with the patient's inquiry. A general survey includes necessarily the examination (inspection and palpation) of the thyroid gland and the lymphatic nodes, and taking body temperature.

Nutritional state is estimated by a body mass index (BMI). In addi-

tion, it is necessary to determine the *circumference of waist and hips*, which allows you to estimate the number of visceral fat by a waist-to-hip ratio (WHR).

General condition (general state) of the patient is determined as a result of the general survey by a number offeatures that include:

- state of consciousness;
- position of the patient;
- habitus;
- gait;
- face;
- state of the skin,visible mucosa, andsubcutaneous tissue;
- constitutional type and nutritional state;
- musculoskeletal system;
- vital signs, such as body temperature, heart rate, blood pressure, respiratory rate, daily diuresis.

The severity of the patient's condition is determined by, first of all, the state of consciousness, position and changes of vital signs.

*General condition of a patient can be estimated in the following degrees:****satisfactory, moderate severity, and severe (serious) condition***.

2.12. Assessment tests on the theme "Objective Examination of a Patient. General Inspection (Survey)"

1. Objective examination consists of the following parts:

1. physical examination;
2. laboratory and instrumental examination;
3. main complaints and its detailing;
4. anamnesis communis;
5. anamnesis morbi.

2. Physical examination includes:

1. general inspection (survey);
2. assessment of a general condition of a patient;
3. special (local) inspection;
4. palpation, percussion and auscultation of respiratory, cardiovascular, digestive, and urinary systems;
5. X-ray and laboratory tests.

3. Variants of mental state assessment:

1. alertness;
2. stupor;
3. obtundation;
4. coma;

5. amnesia.

4. Normal weight according to BMI includes:

1. 18.5-24.9 kg/m^2;
2. 35.0-40.0 kg/m^2;
3. 25.0-30.0 kg/m^2;
4. 30.0-35.0 kg/m^2;
5. 16.0-20.0 kg/m^2.

5. Specify the types of patient position:

1. active;
2. passive;
3. force;
4. vertical;
5. horizontal.

6. Cyanosis is characteristic of:

1. respiratory failure;
2. circulatory failure;
3. digestive system diseases;
4. uremia;
5. anemias.

7. Enlargement of lymph nodes occurs in:

1. local inflammation;
2. metastasis of malignant tumor;
3. diseases of the hematopoietic system;
4. infectious diseases;
5. autoimmune diseases.

8. Degree of the thyroid gland hyperplasia (according to WHO):

1. 0 – absence of a goiter;
2. I – the invisible goiter, but the goiter is defined as a lateral lobe with a volume greater than the thumb of the individual being examined;
3. II – the visible and palpable goiter;
4. III – the goiter is visible from a distance;
5. III - the goiter is visible from a distance>7-10 m.

9. Study of bones and joints includes the examination of:

1. shape of the bones, joints, and spine;
2. pain at palpation;
3. estimation of the volume and pain at physiological movements of the joints;
4. skin temperature around bones and joints;

5.edema around bones and joints.

10. Study of muscles includes the examination of:
1. volume and consistence of muscles;
2. pain at palpation;
3. muscle tone and force;
4. skin temperature around muscles;
5.passive and active mobility.

11. Thermometry of adult patients is performed in:
1. axillary space;
2. oral cavity;
3. rectum;
4. inguinal fold;
5. ear.

12. Types of fever according to level of the body temperature:
1. subferile – 37.0-38.0 °C;
2. moderate – 38.1-39.0 °C;
3. high – 39.1-41.0 °C;
4. hyperperetic - >41.0°C;
5. critical > 42.0°C.

13. Types of temperature curve:
1. persistent;
2. hectic;
3. remittent;
4. intermittent;
5.repeating.

14. Duration of the body thermometry by mercury thermometer
1. 10 minutes;
2. 3-5 minutes;
3. 10-15 minutes ;
4. up to 30 minutes;
5. 2-3 minutes.

15. Specify conditions, which can lead to physiological elevation of the body temperature:
1. muscular effort;
2. food intake;
3. emotional stress;
4. sleep;

5. infectious diseases.

16. Persistent fever is characterized by:
1. daily temperature fluctuations within 1˚C;
2. daily temperature fluctuations within 2˚-3˚C;
3. daily temperature fluctuations more than 1˚C;
4. raising the temperature to above 39˚ and subsequent rapid decrease to the normal;
5. daily temperature fluctuations within 3˚-4˚C.

17. Hectic fever is characterized by:
1. daily temperature fluctuations within 2˚-4˚;
2. daily temperature fluctuations within 1˚C;
3. daily temperature fluctuations more than 1˚C;
4. raising the temperature to above 39˚ and subsequent rapid decrease to the normal;'
5. daily temperature fluctuations absent.

18. General condition of a patient can be estimated in the following degrees of assessment:
1. severe condition;
2. state of moderate severity;
3. satisfactory state of the patient;
4. critical condition;
5. state of clinical death.

19. Assessment of the general condition of the patient is based on such criteria as:
1. state of consciousness;
2. position of the patient;
3. state of feeding;
4. body temperature;
5. parameters of activity of cardiovascular, respiratory and excretory systems.

20. Vital signs are important for assessment of general condition of the patient:
1. pulse rate;
2. respiration rate;
3. blood pressure;
4. volume of daily diuresis;
5. body temperature;
6. colour of skin and visible mucosa.

21.What are characteristics of a satisfactorycondition of a patient?

1. clear consciousness (alertness);
2. stupor;
3. normal body temperature;
4. active position;
5. forced position.

22.What are characteristics of a moderate severity condition of a patient?

1. clear consciousness;
2. stupor or obtundation;
3. active or forced position;
4. passive position;
5. vital signs (body temperature, pulse rate, respiration rate, blood pressure, daily diuresis) are changed/

23. What are characteristics of a severecondition ofa patient?

1. coma;
2.stupor;
3. passive position;
4.forced position;
5. vital signs(body temperature, pulse rate, respiration rate, blood pressure, daily diuresis) are not stable and changed significantly.

Unit II. Respiratory System Examination

Chapter 3. Subjective and Objective Examination of Patients with Respiratory System Diseases

Objectives: to enable students to learn –

1) subjective examination (inquiry) of patients with the respiratory system diseases and interpretation of the obtained results;
2) technique of a general survey in the respiratory system diseases and its diagnostic value;
3) technique and diagnostic value of a static and a dynamic inspection of the chest;
4) technique and diagnostic value of the chest palpation in the respiratory system diseases.

3.1. Subjective respiratory examination (inquiry)

Complaints

The main complaints typical of the respiratory system are dyspnea, cough, bloody expectorations, and pain in the chest (Table 3-1).

Dyspnea (*shortness of breath, breathlessness*) can be subjective, objective, or subjective and objective simultaneously. S*ubjective dyspnea* is the feeling of difficult breathing. *Objective dyspnea* is determined by the objective methods of the examination; and it is characterized by changes in the frequency, depth, rhythm of breathing, as well as the duration of inspiration or expiration.

Diseases of the respiratory system are often accompanied by mixed (i.e. subjective and objective) dyspnea. It is often associated with rapid breathing (*tachypnea*). These symptoms occur in pneumonia, bronchogenic cancer, and in tuberculosis. Cases with purely subjective dyspnea (in hysteria, thoracic radiculitis) or purely objective dyspnea (in pulmonary emphysema or pleural obliteration) occur less frequently. Dyspnea is possible with both a normal and a slow rate of breathing (*bradypnea*). Three types of dyspnea are differentiated by the prevalent breathing phase: *inspiratory dyspnea, expiratory dyspnea and mixed dyspnea*, when both expiration and inspiration become difficult.

Dyspnea may be physiological (caused by heavy exercise) and pathological (associated with diseases of the respiratory, cardiovascular and hemopoietic systems, and poisoning). Pathologic dyspnea associated with the respiratory pathology may have various etiology: due to the lung compression by a liquid or an air accumulated in the pleural cavity, a decreased pneumatization of the lung in pneumonia, an atelectasis, an infarction or a decreased elasticity of the lungs.

Table 3-1. Typical complaints in respiratory system diseases

Complaints	Characteristics	Causes
Cough	Non-productive (dry)	- viral infections; - “post-nasal drip” – in rhinosinusitis; - interstitial lung diseases; - tumors; - allergic diseases; - an increase of bronchopulmonary lymphatic nodes; - hypotensive medications – ACE (angiotensin converting enzyme) inhibitors
	Productive (wet) cough with: 1) Mucoid (grey, white) sputum	- chronic obstructive pulmonary disease (COPD); - bronchial asthma; - upper airways diseases (in pharyngitis, laryngitis, tracheitis – small volume of mucus sputum)
	2) Purulent (yellow, green) sputum	- infectious – pneumonia, bronchiectasis and abscess; - cystic fibrosis
	3) Serous (clear, foamy, can be pink) sputum	- pulmonary edema
	4) Hemorrhagic sputum	- malignancy; - pulmonary embolism; - clotting blood disorders; - tuberculosis; - infections; - congestive heart failure; - traumas of chest
Chest pain	At coughing and deep inspiration	- dry (fibrinous) pleurisy; - lobar pneumonia (pleuropneumonia); - pulmonary embolism; - pneumothorax - traumas of chest
Dyspnea	Inspiratory	- obstruction of the extra-thoracic airways such as the larynx or trachea, e.g. bilateral laryngeal paralysis
	Expiratory	- chronic obstructive pulmonary disease (COPD) and bronchial asthma
	Mixed	- obstruction of the upper airways

Cough may indicate the presence of a lung disease, but cough per se is not useful for the differential diagnosis. Presence of sputum accompanying the cough often suggests airway disease (in asthma, chronic bronchitis, or bronchiectasis).

By nature, there are types of cough: dry without sputum and wet-with sputum (productive).

Sputum is a the respiratory tract mucous discharge, and expectorated by coughing. Sputum is always a pathological phenomenon.

The consistency of sputum depends on the content of mucus: the more mucus, the more dense and viscous sputum.

The character of the sputum can be different: mucoid, serous, purulent, mucopurulent, seropurulent and bloody (hemorrhagic).

In the presence of sputum, it is necessary to find out its amount during the day, at what time of the day and in what position the patient has better expectoration, the nature of sputum, its color, smell, and consistency.

By time of appearance, there are several typical types of cough:

- morning cough - in chronic inflammation of the upper respiratory tract (nose, nasopharynx, nasal cavity, larynx, trachea). Smokers that cough is called " cough when washing";

- evening cough -in bronchitis, pneumonia;

- nocturnal cough - because of night increase of *n. vagus* tone, and in case of enlargement of the bronchopulmonary lymphatic nodes, tuberculosis of the lungs.

Cough may occur under certain conditions or be accompanied by certain phenomena.

Cough occurs when changing the position of the body in case of cavities in the lungs (bronchiectasis, tuberculous caverns, abscess, lung gangrene).

Cough that occurs after eating, especially in the presence of particles of food intake, indicates fistula of esophagus penetrating into trachea or bronchus (in esophageal cancer, ulceration and rupture of the respiratory tract).

Cough, accompanied by the release of a large amount of sputum (sputum by "full mouth"), is caused by emptying the cavities in the lungs (in abscess).

Hemoptysis is expectoration of blood with sputum during cough. Hemoptysis can originate from the disease of the airways, the pulmonary parenchyma, or the vasculature. The diseases of the airways can be inflammatory (acute or chronic bronchitis, bronchiectasis, or cystic fibrosis) or neoplastic (bronchogenic carcinoma or bronchial carcinoid tumors). Parenchymal diseases causing hemoptysis may be either localized (pneumonia, lung abscess, tuberculosis, or infection with *Aspergillus)* or diffuse (*Goodpasture's syndrome*, idiopathic pulmonary hemosiderosis). Vascular diseases potentially associated with hemoptysis include pulmonary

thromboembolic disease and pulmonary arteriovenous malformations.

Chest pain caused by diseases of the respiratory system usually originates from involvement of the parietal pleura. As a result, the pain is accentuated by respiratory motion and is often referred to as *pleuritic*. Common examples include primary pleural disorders, such as neoplasm or inflammatory disorders involving the pleura, or pulmonary parenchymal disorders that extend to the pleural surface, such as pneumonia or pulmonary infarction. *Pain* in the chest is classified by its location, origin, character, intensity, duration, and irradiation, by its connection with the respiratory movements, cough, and the posture. Pain may arise during the development of a pathological condition in the thoracic wall, the heart, and the aorta, and in diseases of the abdominal organs (by irradiation).

Pain may develop in injury of the skin (trauma, erysipelas, herpes zoster, etc.), muscles (trauma, myositis), intervertebral nerves (thoracic radiculitis in spondylarthrosis), ribs and costal pleura (metastases of the tumour, fractured bones, periostitis).

Pleural pain has a distinct localization in the chest, often in the lateral parts ("pain in the side"). If the diaphragmatic pleura is affected, pain is felt in the abdomen.

A characteristic feature of the pleural pain is its intensification during inspiration, especially when coughing and deep breathing (as a result of which the patient tries to breathe superficially).

In dry pleurisy, pain is a result of friction of inflamed pleural membranes against each other. With exudative pleurisy, pain is usually observed only in the initial period of the disease, then it transforms into a feeling of heaviness in the side.

Anamnesis

Present disease history (*anamnesis morbi*) in respiratory system pathology often gives sufficient recognition of the disease. Essential is the beginning of the disease and its course: sudden with the rapid development of symptoms - in lobar pneumonia, gradual with increasing shortness of breath - in exudative pleurisy.

It is important to know the circumstances preceding the onset of the disease: influenza, rapid cooling, contact with infectious patients, trauma with a fracture of a major bone or abdominal surgery.

In *past life history (anamnesis vitae),* past diseases can have a significant impaction, for example: recurring pneumonias in bronchiectatic disease. Home and working conditions (poor ventilation, lack of light, a small volume of air in the room) affects the proper function of the respiratory system, creates predisposition to lung diseases.

Information about risk factors for a lung disease should be explicitly explored to assure a complete basis of historic data. A history of current and past smoking, especially of cigarettes, should be sought from all patients.

The smoking history should include the number of years of smoking, the intensity (i.e., number of packs per day), and, if the patient no longer smokes, the interval since smoking cessation. The risk of lung cancer falls progressively with the interval following discontinuation of smoking, and loss of lung function above the expected age-related decline ceases with the discontinuation of smoking. The patient may have been exposed to other inhaled agents associated with the lung disease, which act either via direct toxicity or through immune mechanisms. Important agents include the inorganic dusts associated with pneumoconiosis (especially asbestos and silica dusts) and organic antigens associated with hypersensitivity pneumonitis (especially antigens from molds and animal proteins). Bronchial asthma, which is more common in women than men, is often exacerbated by exposure to environmental allergens (dust mites, pet dander, or cockroach allergens in the house or allergens in the outdoor environment such as pollen and ragweed) or may be caused by occupational exposures (diisocyanates).

Family history is important for evaluating diseases that have a genetic component. These include disorders such as cystic fibrosis, α-antitrypsin deficiency, and asthma.

3.2. The respiratory system inspection

General survey is started by studying a state of the patients's consciousness and position. In connection with a hypoxia of a brain in respiratory failure all kinds of disordered consciousness can be observed: obtundation, stupor, hypoxemic coma, hallucinations.

The ***forced lateral recumbent position (lateral decubitus)*** on the affected side of the chest is accepted by patients in pneumonia, tuberculosis, exudative and dry pleurisy, pulmonary abscess or gangrene, bronchiectases (Table 3-2).

The ***forced sitting position*** *(orthopnea)* is connected mainly to dyspnea (in pneumothorax, an attack of bronchial asthma, emphysema, stenosis of a larynx). In sharp degrees of dyspnea the patients put arms on knees, on edges of a bed, a seat of a chair or the handle of an armchair fixing thus a shoulder girdle and starting auxiliary respiratory muscles.

A characteristic face expression presents in an acute stage of pneumonia: it is a little reddened and edematous (*facies febrilis*), restless, with a suffering expression, with running over at coughing (in view of its tenderness) a grimace, with motility of wings of a nose (owing to a short breathlessness), with typical blisters of herpes on lips of the mouth and wings of a nose.

In presence of a respiratory failure, ***diffuse, or central, cyanosis*** is observed in various degrees of blueness from moderate cyanosis of the face up to diffuse cyanosis with a crimson shade due to a hyperglobulinemia.

Typical changes of fingers of arms are observed in prolonged suppurative processes in the lungs (abscesses and gangrene), emphysema,

tumours of mediastinum, bronchoectatic disease. There are clubbing distal phalanges of fingers with a loss of the nail bed angle. In the typical cases distal phalanges are represented ***drumstick (clubbed, Hippocratic) fingers***. Nails become convex and get looking alike ***"watch glass, or nail clubbing"***.

Table 3-2. General survey in the respiratory system diseases

Signs	Characteristics	Causes
Forced position	forced sitting position (orthopnea) – in severe dyspnea with support on hands	-chronic obstructive pulmonary disease (COPD); - emphysema; - bronchial asthma; -may be in pneumothorax and stenosis of larynx
	forced lateral recumbent position (lateral decubitus) on the affected side of the chest	- exudative and dry pleurisy; - pulmonary abscess; - bronchiectases; - may be in pleuropneumonia and tuberculosis
Cyanosis	Diffuse (total) or central (face, neck, and upper part of chest) blueness of the skin and visible mucosa	- respiratory failure; - syndrome of pulmonary heart; - pulmonary embolism; - congenital heart diseases; - hemoglobin anomalies; - polycythemia
Hyppocratic ("drum-stick") fingers	Clubbing of the fingers distal finger phalanges with loss of the nail bed angle ("watch-glass nails", or "nail clubbing")	Conditions of chronic hypoxemia in: - prolonged suppurative processes in the lungs (abscesses); - COPD, emphysema; - tumours of mediastinum and lungs; - bronchoectatic disease; - chronic circulatory failure; - chronic anemia

3.3. Chest inspection

Examination of the chest is carried out in a standing or sitting position of the patient with the body naked to the waist.

Static and dynamic inspection of chest are performed.

3.3.1. Static chest inspection

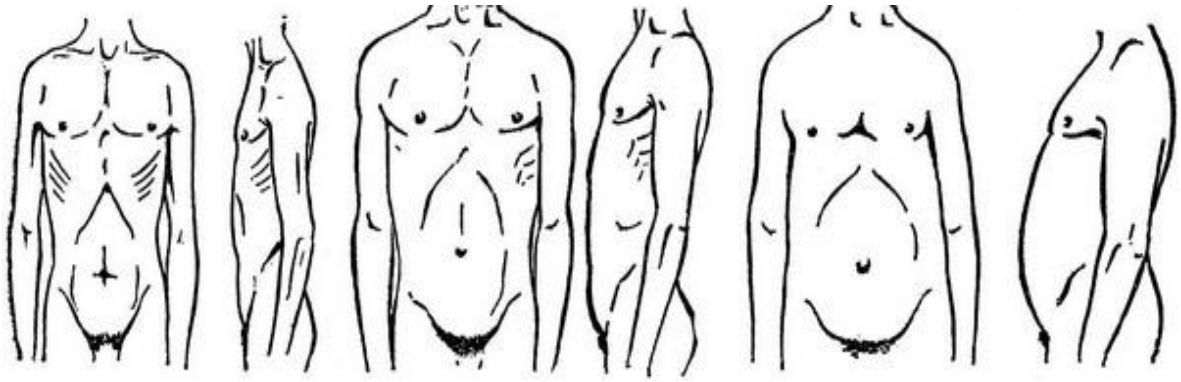
Static survey estimates the shape of the chest at quiet respiration.

Static survey examines thorax at quiet respiration, including characteristics of supraclavicular and infraclavicular fossae (pronounced, smooth or bulges), position of clavicles, ribs (oblique, horizontal), condition of intercostal spaces, size of epigastric angle, and the angle of Louis (angulus Ludowici), position of scapulae blades, assessment of symmetry and sizes of the chest (ratio of anteroposterior to transverse dimensions, thoracic index). According to these features, we determine a form of the chest.

The chest form may be normal or pathological.

Normal chest is symmetrical; nipples, clavicles and shoulder blades are on the same level. ***There are three forms of normal chest: asthenic, hypersthenic, and normosthenic.***

Asthenic chest is *flat and elongated*. *Asthenic chest is a characteristic of the ectomorph (asthenic) body type.* An anteroposterior size is less than 0.6 of the transverse one. An acute epigastric angle is less than 90º. Ribs direction is oblique nearly vertical at the lateral surface of the chest. Intercostal spaces are wide. Supraclavicular and infraclavicular fossa are expressed. Scapulae are spaced from the chest, their edges are clearly visible, prominent *("wing-shaped shoulder blades"*, or *scapulae alatae*). The free anterior end of the X rib (*costa decima fluctuans*) is not joined to the inferior edge of the costal arch. Asthenic chest is characterized by a relatively weak muscle development (Fig. 3-1, 3-2, Table 3-3).



(1) (2) (3)

Fig. 3-1. Normal types of the chest:
(1) asthenic (ectomorph) chest; (2) normosthenic (mesomorph) chest;
(3) hypersthenic (endomorph) chest.

Hypersthenic chest is cylindrical. *Hypersthenic chest is a characteristic of endomorph (hypersthenic) body type*. Its anterior-posterior size is over 0.75 of a transverse size. Epigastric angle is wide more than 90º. Ribs are almost horizontal, intercostal spaces are very narrow.

Supraclavicular and infraclavicular fossas are smooth. Scapulae are tight to the chest, sometimes almost invisible. Muscles of the shoulder and chest are well developed.

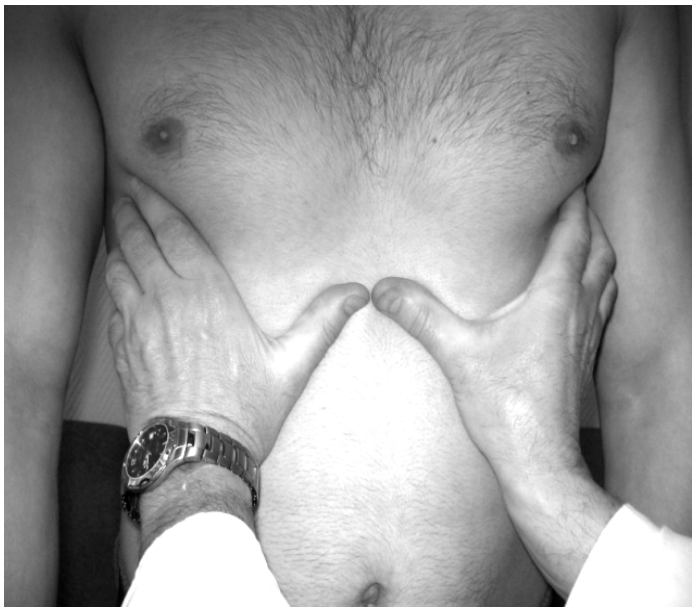


Fig. 3-2. Determination of the epigastric angle.

Table 3-3. The chest inspection

Static inspection of the chest	Dynamic inspection of chest
Normal chest shape: - normosthenic (mesomorph); - asthenic (ectomorph); - hypersthenic (endomorph)	*Type of respiration:* - thoracic (costal); - abdominal (diaphragmatic); - mixed
Pathologic chest shape: - emphysematous (barrel-like); - rachitic chest (keeled, or pigeon); - paralytic; - funnel (foveated); - kyphoscoliotic chest	*Symmetricity of respiratory movements* - lag when breathing – in pleurisy, presence of liquid or gas in pleural cavity *Respiration rate, depth and rhythm of respiration, chest excursion measuring*
Asymmetry of chest: - *bulging* – in presence of fluid in the pleural cavity; - *retraction* – in pneumofibrosis; pleural adhesions, obturator atelectasis, and passed surgery operation on lung	*Abnormal breathing patterns:* - undulant Grocco's respiration; - Cheyne-Stokes respiration; - Biot's respiration; - Kussmaul's respiration

Normosthenic chest is conical. Normosthenic chest is a characteristic of mesomorph (normosthenic) body type. An anteroposterior size is shorter than the transverse one, from it - 0.65-0.75. An epigastric angle is about 90º. Ribs are directed somewhat obliquely downwards. Intercostal spaces are expressed and visible in the lower lateral parts of the chest, where the muscles are less developed. Supraclavicular fossa is expressed, the subclavian – smooth. Scapulae with arms are tightly against the back of the chest. Chest and shoulder muscles are well developed.

Asymmetric changes in the chest: retraction (retraction) of one half of the chest (part of it) or protrusion.

The causes of the unilateral deformation of the chest can be the diseases of the lungs, pleura, heart and blood vessels.

Retraction of one side or a separate area of chest indicates a decrease in the volume of one lung, obliteration of the pleural cavity as a result of past inflammatory process (tuberculosis, bronchiectasis; lung abscess, atelectasis, lung resection).

Protrusion or enlargement of one side of the chest are caused by air and/or fluid accumulation in the pleural cavity (hydrothorax, pneumothorax).

Pathological forms of the chest

Emphysematous (barrel-like) chest resembles a hypersthenic. Hence, intercostal spaces, unlike hypersthenic, are wide. Supra- and subclavian fossa are smoothed or inflated due to increased airiness of the lung apex (Fig. 3-3). The thoracic index is sometimes greater than 1.0 by increasing the anterior-posterior size. The chest resembles a barrel. It occurs in patients with emphysema of the lungs, in which the elasticity of the lung tissue decreases, its airiness increases.

Paralytic chest resembles a modified asthenic chest. It has a reduced anterior-posterior size. The chest is flat. It happens in severely malnourished people and in patients with long-term pulmonary tuberculosis. In these cases, lungs shrinks in size. It can be often asymmetric (one half is smaller than the other).

Rachitic (pigeon, or keeled) chest is characterized by protruding sternum with increase of an anteroposterior size over the transverse one. It is developed in childhood due to vitamin D deficiency.

Funnel (foveated) chest is characterized by depression in a lower part of the sternum. It is a result of congenital abnormalities or from prolonged pressure on the sternum *(*"*shoemaker's chest*").

Scaphoid chest is characterized by depression mainly in the upper and middle part of the sternum (*a boatshaped chest*). Cause is syringomyelia (a rare disease of the spinal cord).

Kyphoscoliotic chest develops as a result of kyphoscoliosis, a combination of curvature of the spine to the side and posteriorly.

These pathological forms of the chest lead to the impaired lung and heart function. Such patients often suffer from bronchitis, pneumonia; they develop early respiratory and heart failure.

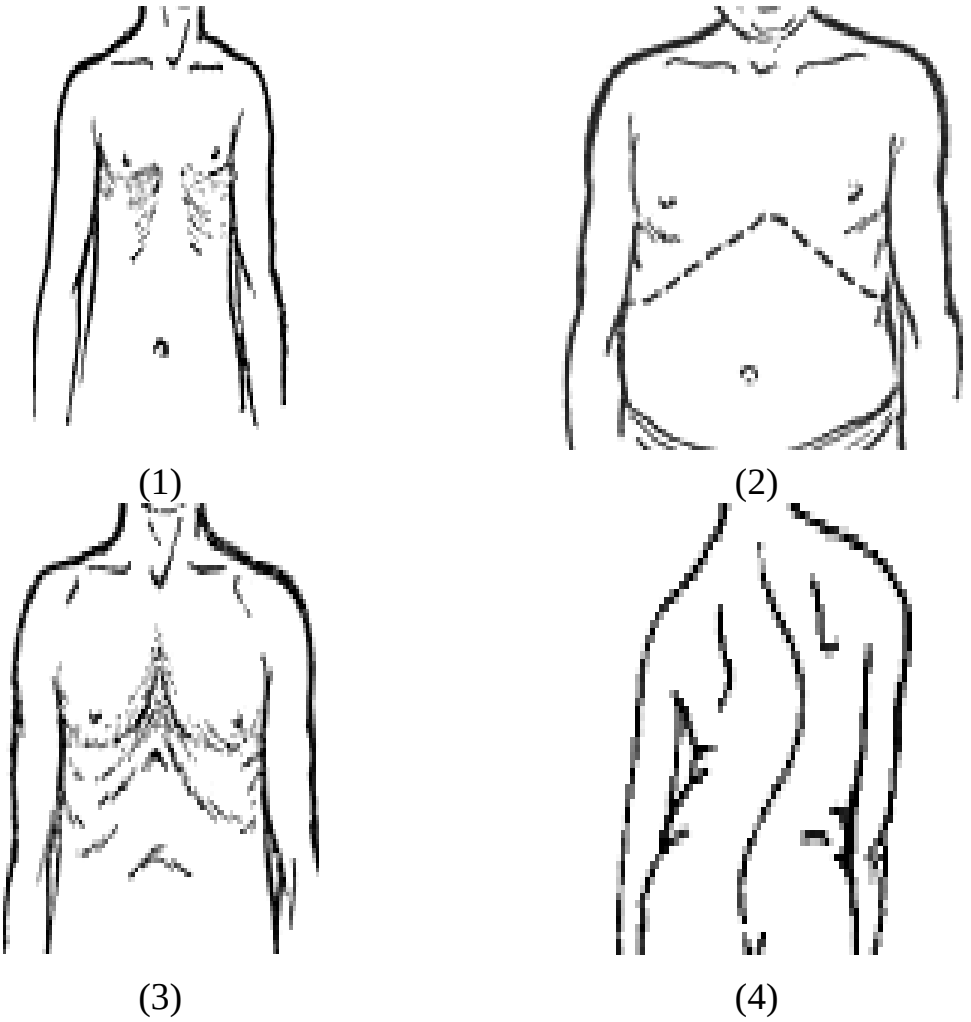


Fig. 3-3. Pathologic types of chest:
1 - rachitic chest; 2 - emphysematous chest; 3 - funnel chest;
4 - kyphoscoliotic chest (back view).

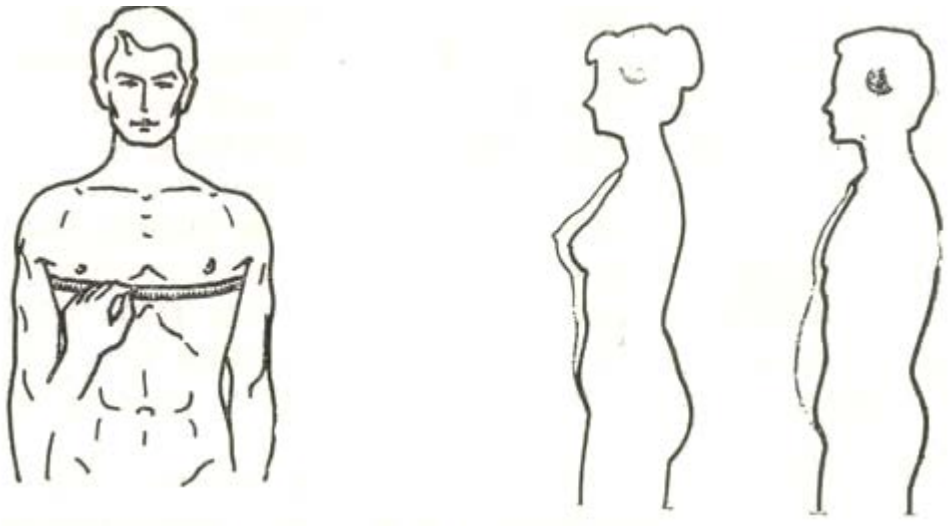
3.3.2. Dynamic chest inspection

Respiratory movements of the chest should be examined during *dynamic inspection of the chest.* In physiological conditions, they are performed by the contraction of the main respiratory muscles: intercostal muscles, muscles of the diaphragm, and partly the abdominal wall muscles. The so-called accessory respiratory muscles (*mm. sternocleidomastoideus, trapezius, pectoralis major et minor, etc*.) are actively involved in the respiratory movements in pathological conditions associated with difficult breathing.

The type, symmetricity, frequency, depth and rhythm of respiration can be determined by carefully observing the chest and the abdomen.

Respiration can be thoracic (costal), abdominal, or mixed types.

Thoracic (costal) respiration. Respiratory movements are carried out mainly by the contraction of the intercostal muscles. The chest markedly broadens and slightly rises during the inspiration, while during the expiration it narrows and slightly lowers. This type of respiration is known also as costal and is mostly characteristic of women (Fig. 3-4). A pathologic costal respiration occurs sometimes in cases of diaphragmatitis (an inflammation of the diaphragm), acute cholecystitis, perforating ulcer of the stomach or the duodenum; it is carried out only by the intercostal muscles.



(1) (2)

Fig. 3-4. Chest excursion measuring and types of respiration:
1 - thoracic; 2 - abdominal

Abdominal (diaphragmatic) respiration. Respiration is mainly accomplished by the diaphragmatic muscles; during the inspiration phase the diaphragm contracts and lowers to increase rarefaction in the chest and to suck in air into the lungs. The intraabdominal pressure increases accordingly to displace of the abdominal wall. During expiration the muscles are relaxed, the diaphragm rises, and the abdominal wall returns to the initial position. This type of respiration is also called diaphragmatic and is mostly characteristic of men. A pathological abdominal respiration is temporarily carried out by the diaphragmatic muscles exclusively in case of extensive pleural adhesions, lung emphysema, and in strong pain in the chest due to acute inflammation of the intercostal muscles or nerves.

Mixed respiration. The respiratory movements are carried out simultaneously by the diaphragm and the intercostal muscles. In physiological conditions this respiration sometimes occurs in the aged persons, and in some pathological conditions of the respiratory apparatus and the abdominal organs. For example, the contractile activity of the intercostal muscles decreases and the respiratory movements are carried out by the accessory movements of the

diaphragm in women with dry pleurisy, pleural adhesion, myositis, and the thoracic radiculitis. Mixed respiration occurs in men with underdeveloped diaphragmatic muscles, and in diaphragmatitis (an inflammation of the diaphragm), acute cholecystitis, perforating ulcer of the stomach or the duodenum.

Respiratory chest movements are symmetrical in the norm. If they diminished, that is likely to be the side on which there is an abnormality. Unilateral lag when breathing presents in pleurisy, presence of liquid or gas in pleural cavity, pleural adhesions, pathology of the pulmonary tissue - pneumofibrosis; obturator atelectasis, and passed surgery operation on the lung. Intercostal recession - a drawing-in of the intercostal spaces with inspiration - may indicate severe upper airways obstruction, as in laryngeal disease, or tumours of the trachea. In COPD the lower ribs often move inwards on inspiration instead of the normal outwards movement.

Depth of respiration is measured by *chest excursion* (See Fig. 3-4.). *Chest excursion is the difference between the thorax circumference in a state of maximum inspiration and exhalation*. A centimeter tape is applied so that it passes under the inferior corners of the blades and the front in men - on the lower segment of the nipple, in women - on the breast at the place attachment of IV ribs to the sternum.

Normally, the circumference of the chest in men is 88-92 cm, in women - 83-85 cm. Excursion, depending upon its volume and height of the patient, is ranged respectively 6-8 cm and 3-6 cm. In persons regularly engaged in exercise and sport, chest excursion can reach 12-15 cm.

Respiratory rate. Respiratory rate is determined by counting chest movements or abdominal wall during a minute. It is necessary to divert the attention of the patient by palpation of the radial artery pulse, to avoid possible second mental influence.

At rest, normal respiration rate is 16-20 per minute. In case of physical exertion, emotional stress, after eating, respiration rate increases.

Abnormal rapid respiration (tachypnea) occurs in:

- narrowing the lumen of small bronchi (bronchospasm);
- reducing respiratory surface of the lungs in pneumonia, pulmonary atelectasis, infarction of the lungs;
- sharp pain in the chest (dry pleurisy, broken ribs, myositis).

Pathological slow respiration (bradypnea) occurs when a brain respiratory center is depressed (cerebral hemorrhage, brain edema, brain tumor, exposure to the respiratory center of toxic substances).

Abnormal breathing patterns

Respiration may be deep or shallow. Depth of breathing is inversely proportional to the respiration rate, rapid breathing is superficial; slow breathing usually deep. The exception to this rule may be *stenotic respiration* (in narrowing of the upper respiratory tract as a result of edema, tumor, foreign body, etc.), which is both rare, but at the same time superficial.

Deep, noisy *Kussmaul's respiration* may be frequent at the same time ("the breathing of a hunted animal"). Kussmaul's respiration in decompensated acidosis (diabetic hyper-glycemic-hyperketonemic coma, uremic coma) (Fig. 3-5).

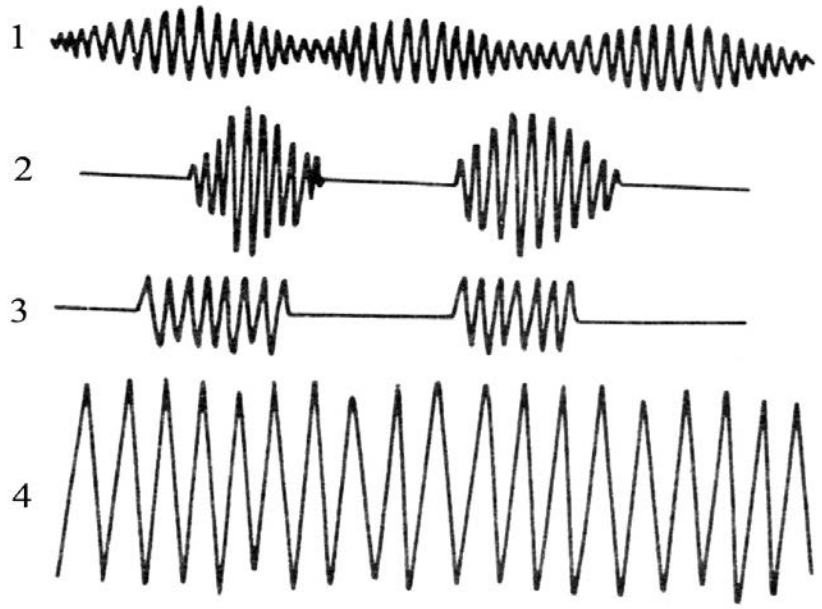


Fig. 3-5. Abnormal breathing patterns:
1 - undulant Grocco's respiration; 2 - Cheyne-Stokes respiration; 3 - Biot's respiration; 4 - Kussmaul's respiration.

Rhythm of respiration. Normal breathing is rhythmical. Depression of a brain respiratory center may generate the following breathing patterns: Biot's, Cheyne-Stokes, Grocco's respiration.

Biot's respiration is characterized by rhythmic deep respiratory movements, which alternate with periodic respiratory pauses. In this case, amplitude of the respiratory movements is the same. It happens in inflammatory diseases of the brain and brain membranes (meningitis, encephalitis).

Cheyne-Stokes respiration is characterized by q prolonged respiratory pause of apnea (up to 1 minute) firstly, following shallow breathing, which gradually increases in depth and reaches a maximum at 5-7-th inspiration. Then breathing decreases again until it pauses. This type of respiration is observed in acute insufficiency of cerebral circulation (brain stroke).

Undulant (wavy) respiration, or Grocco's respiration. It is considered by many as a prelude to Cheyne-Stokes respiration. Unlike the latter, in Grocco's respiration periods of complete apnea does not occur, it periodically becomes only very superficial.

Dissociated respiration of Grocco-Frugoni. It occurs as a result of a deep disorder of the respiratory muscles synchronicity (intercostal muscles and diaphragm) due to a severe depression of the brain respiratory center. It

can be stated that the upper half of the chest is in the phase of inspiration, while the lower part due to the reduction of the diaphragm is in the phase of expiration.

3.4. Chest palpation

Chest palpation purposes are:

- to specify the place and degree of severity of the pain;
- to determine resistance and elasticity of the chest;
- to determine tactile fremitus (*syn*. vocal fremitus, fremitus pectoralis, or literal translation from Russian "голосовое дрожание" -"voice tremor");
- identify the friction of the pleural membranes ("friction fremitus").

To specify the exact localization of changes on the chest conditionally identification horizontal and vertical lines are allocated.

Horizontal lines are placed along the ribs and intercostal spaces. Count of the ribs anteriorly are starting with I ribs (in the majority of people it is located under the clavicle). It is simply defined with the connection of manubrium and the body of the sternum (*angulus Ludovici, or angle of Louis*) - the II rib is attached to the sternum at this level.

Posteriorly it is guided by the spinous processes of the vertebrae - easily palpable spinous process of VII cervical vertebra (*processus prominens*). VII rib is palpated under the lower angle of the scapula when lowered hands.

Vertical lines are measured at the hands of the patient and descended divided as follows (Fig. 3-6):

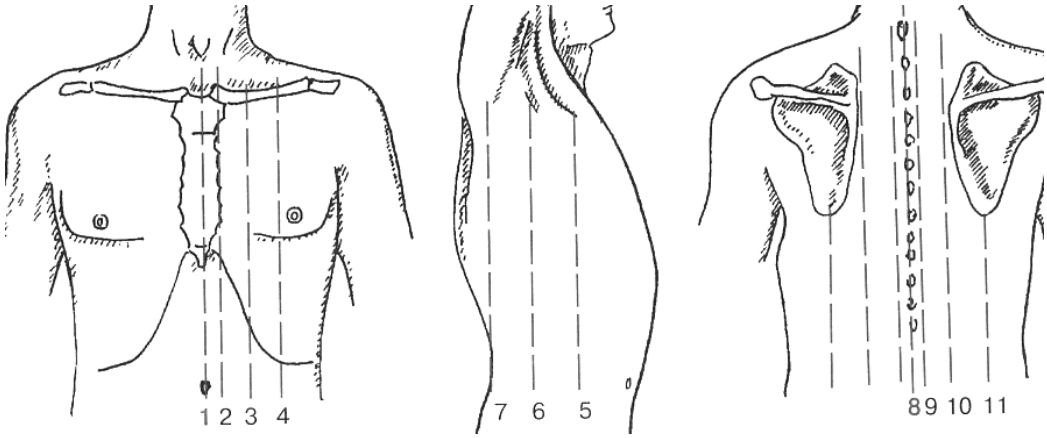


Fig. 3-6. Topographic vertical lines on the chest:

1 - mediana anterior, 2 – sternalis dextra et sinistra, 3 - parasternalis dextra et sinistra, 4 - medioclavicularis dextra et sinistra, 5 – axillaris anterior dextra et sinistra, 6 - axillaris media dextra et sinistra, 7 - axillaris posterior dextra et sinistra, 8 - mediana posterior, 9 – vertebralis dextra et sinistra, 10 – paravertebralis dextra et sinistra, 11 - scapularis dextra et sinistra.

Lin. mediana anterior goes from top to bottom in the middle of the sternum from the lowest point of the sternal notch. Lin. sternalis dextra et sinistra are respectively on the right and left of the sternum. Lin. parasternalis dextr. et sin. are located between lin. medioclavicularis and sternalis. Lin. medioclavicularis dextr. et sin. start from the middle of the clavicle. Lin. axillaris anterior et posterior dextr. et sin. are directed vertically down, respectively, at the anterior and posterior edges of the armpits. Lin. axillaris media dextr. et sin. go straight down at the middle of the armpits. Lin. scapularis dextr. et sin. pass through the inferior angle of the scapula. Lin. paravertebralis dextr. et sin. are midway between lin. vertebralis and scapularis. Lin. vertebralis dextr. et sin. are on the edge of the spine. Lin. mediana posterior passes on the spinous processes of the vertebrae.

3.4.1. Palpation of the chest in order to identify the pain points

It is performed with the fingertips on symmetrical areas, pressing on the chest in a certain sequence: start with the anterior surface – at supraclavicular and infraclavicular regions, and then – the major pectoral muscles, on lateral-inferior parts of the chest – by axillary lines from the top down; on the back – supraspinal areas (above the spina of the scapula), moving to the interscapular region, subscapular area, and posterior-lateral parts of the chest.

Deep, or pleural, pain in dry pleurisy increases sharply with deep breathing, coughing, when benting the chest healthy side. This is due to the increased lung excursions and increased frictions of the pleura. Pleural pain is reduced if the immobilized chest, at pressing the both chest sides by hands (the chest excursion is limited). Pleural pain is often one-sided (Table 3-4).

3.4.2. Resistance (elasticity) of the chest

Determination of *resistance (elasticity) of the chest* is performed by its compression in two perpendicular directions, as well as palpation of the intercostal spaces.

Palm of the right hand is established in the middle of the sternum, the left palm - in the interscapular space at the same level (Fig.3-7). Then palms are placed on axillary lines at lateral-inferior parts of the chest. When you press on the chest and intercostal spaces, they seem to spring up.

As a rule, the chest is elastic.

With age, the elasticity of the chest decreases, and it becomes malleable, rigid (stiffness – increased resistance). In such cases, when the chest is compressed, there is an increased resistance. The *rigidity of the chest* depends on elasticity of ribs cartilages, thickness of the chest wall, and intrathoracic pressure.

The chest is rigid in pulmonary emphysema, hydrothorax, pleurisy with effusion, ossification of the costal cartilages, after fractures of the ribs. Physiological increased rigidity presents in a hypersthenic type of the chest in comparison with the asthenic chest.

Table 3-4. Palpation of the chest

Purposes	Characteristics	Causes
Allocation of the pain in the chest	at intercostal spaces, increasing at cough and deep inspiration	herpes zoster, intercostal neuralgia, dry pleurisy
	at ribs palpation	fractures of ribs, osteomyelitis, malignancy of ribs
	at chest muscles palpation	myositis, fibromyalgia, myopathy
Resistance of the chest (opposite – chest elasticity)	increased resistance	presence of liquid or gas in pleural cavity, pulmonary emphysema, hypersthenic chest, obesity, aged patients
	decreased resistance (elastic chest)	asthenic chest, children, females
Tactile fremitus (vocal fremitus)	increased	consolidation of pulmonary tissue (in pneumonia, pneumofibrosis, tuberculosis), air cavity communicated with the bronchus, asthenic chest
	decreased	- presence of liquid or gas in pleural cavity; - pulmonary emphysema, COPD; - hypersthenic chest; - obesity; children; females

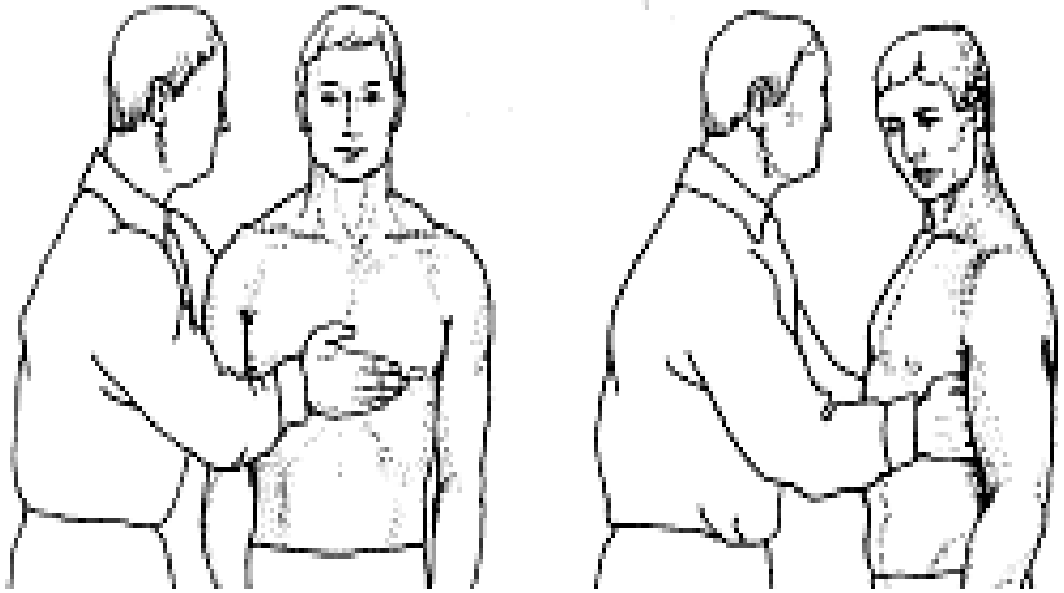


Fig. 3-7. Examination of the chest elasticity

3.4.3. Tactile fremitus (vocal fremitus)

Tactile fremitus is a vibration of the chest caused by the vibration of the vocal cords, followed by air fluctuations in the trachea, bronchi and alveoli.

To determine the tactile fremitus, the palms of the hands are put on symmetrical areas of the chest, tightly pressing them to the chest, and asked to say loudly the words "ninety-nine", or “aa”, or “blue moon” in English tradition; or words containing the sound of the letter "Р” - “тридцать три” in Russian tradition. The air oscillation that occurs in the vocal slit is transmitted through the bronchi to the chest wall.

Position of the patient.

When examining on the anterior surface of the chest, the patient's hands are lowered along the trunk. In the study on the lateral surface – the patient should have his hands behind his head. When examining a posterior surface of the chest the patient should cross hands to widen the interscapular space. The physician should be in front of the patient at examination of the anterior and lateral surfaces of the chest, and at the posterior surface – behind him/her.

Sequence of tactile fremitus examination:

Start at supraclavicular fossa on both sides, placing the palm vertically - then the I and II intercostal spaces on both sides from the sternum to the midclavicular lines. Compare the left and right of tactile fremitus to the upper border of the heart.

On the left below the III rib, of tactile fremitus on the anterior surface is not determined.

On the right below III rib, examiner compares upper and lower parts. The palm is placed horizontally, the fingers pointing to the axillary lines.

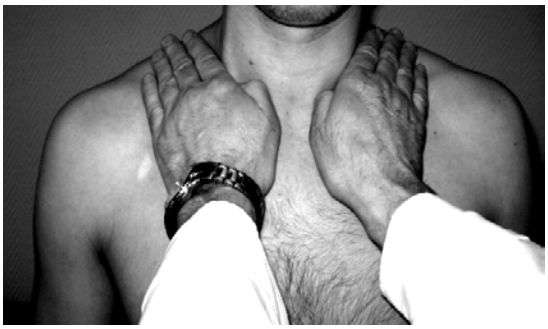
Next, the of tactile fremitus is determined on the lateral surface from the axillary fossa down on both sides.

On a posterior surface, the of tactile fremitus is determined starting supraspinal areas (above spina of the scapula). From the supraspinal areas to the angle of the scapula, the palms are set vertically on two lines: l. scapularis and l. paravertebralis. Below the angle of the scapula, the palms are located along the intercostal spaces (Fig. 3-8).

Normal tactile fremitus conducts equally in symmetric points. It is somewhat more pronounced in the upper parts of the chest, especially above the lung apex.

Tactile fremitus in normal conditions depends on the voice timbre and the chest thickness. The lower the timbre, the better is the transmission. The tactile fremitus is better transmitted with a thin chest wall (in asthenic chest), and in males.

Tactile fremitus may not be determined in females, and children because of the high timbre of the voice.



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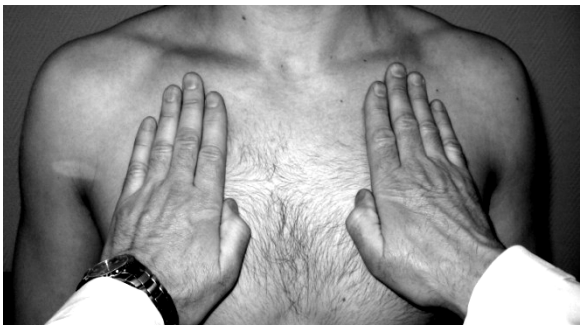

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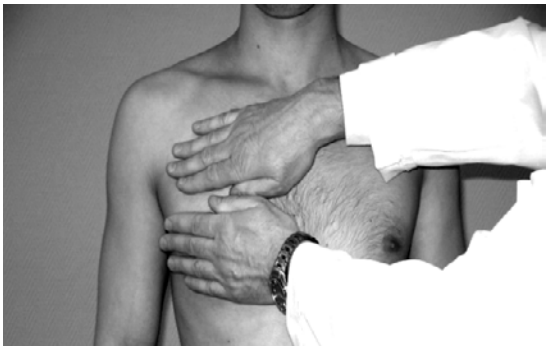

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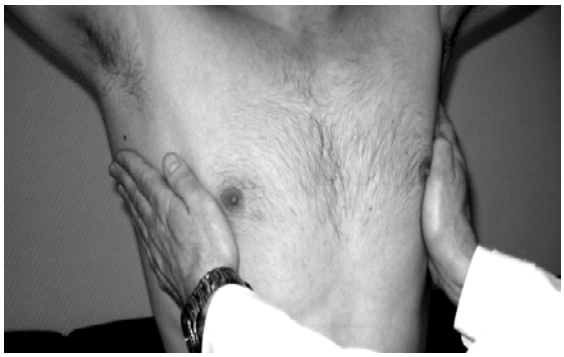

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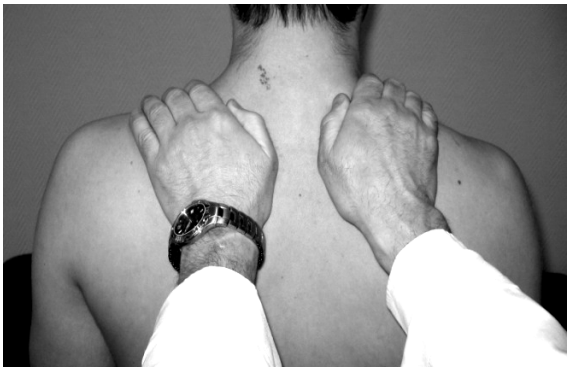

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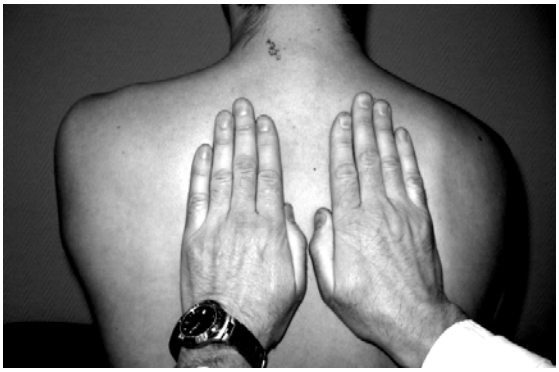

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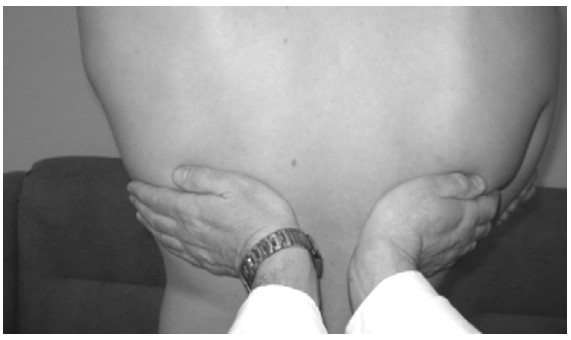

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Fig.3-8. Examination of the tactile fremitus (vocal fremitus):
1 - above the clavicle; 2 - under the clavicle; 3 - at the anterior right part of chest; 4 - at the lateral parts of the chest; 5 - over the scapulae; 6 - between the scapulae; 7 - under the scapulae.

Weakened tactile fremitus until the complete disappearance is de-

scribed in obesity, physiologically in the hypersthenic chest. Weakened tactile fremitus is observed in a number of diseases: hydrothorax, pneumothorax, emphysema, foreign body in large bronchus. If the bronchus is obstructed with a lump of mucus, then after coughing up sputum, tactile fremitus again manifests itself quite clearly.

Increased tactile fremitus is observed in the consolidation of pulmonary tissue (pneumonia, pulmonary infarction, compression atelectasis), in presence of a lung cavity, communicating with the bronchus (tuberculous cavern, lung abscess). Around the cavity, there is usually a reactive inflammatory process leading to compaction of pulmonary tissue, and the cavity itself resonates well.

Palpation of the chest can also determine ***friction of pleural membranes* (*friction fremitus*)** in fibrinous (dry) pleurisy, and crepitation (crackles) of air bubbles in the subcutaneous tissue due to ***subcutaneous emphysema*** caused by pneumothorax.

3.5. The key points on the theme "Subjective and Objective Examination of Patients with Respiratory System"

The main complaints typical for respiratory system diseases are dyspnea, cough, bloody expectorations (hemoptysis), and pain in the chest. *The main types of dyspnea by nature are inspiratory, expiratory, mixed. There are types of cough: dry, without sputum and wet with sputum (productive).*

General examination objectively estimates general condition of the patient, as well as studies of the features of functioning the respiratory system. In a respiratory failure, all kinds of violations of consciousness can be observed: obtundation, stupor, hypoxemic coma, hallucinations. ***Forced lateral recumbent position*** is taken by patients with lobar pneumonia, exudative and dry pleurisy, abscess or gangrene of the lungs, bronchiectasis. ***Forced sitting position*** (orthopnea) is associated with dyspnea (pneumothorax, asthma attack, emphysema, laryngeal stenosis). ***Central (diffuse) cyanosis*** is a sign of respiratory failure in lung diseases. ***Hippocratic (drumstick) fingers*** - with loss of the nail bed angle ("***watch glass nails***", ***or "nail clubbing"***) are typical changes during long suppurative processes in the lung (abscess, gangrene), emphysema, mediastinal tumor, bronchiectasis.

Static survey assesses the shape of chest. *Normal thorax, regardless of shape, has the same size of the two halves.* There are three normal forms of the chest: normosthenic (mesomorph), asthenic (ectomorph) and hypersthenic (endomorph). Pathological types of chest: emphysematous (barrel-like), rachitic (keeled, or pigeon), paralytic, funnel (foveated), and kyphoscoliotic chest.

Dynamic survey of chest evaluates symmetricity of respiratory chest

movements, type of respiration; rhythm and depth of respiration; and respiratory rate. *Respiratory chest movements are symmetrical in the norm.*

Chest palpation examines localization and degree of pain severity; resistance and elasticity of the chest; and tactile fremitus (vocal fremitus).

As a rule, the chest is elastic. *Rigidity of chest depends on elasticity of ribs cartilages, thickness of chest wall, and intrathoracic pressure*. With age, elasticity of chest decreases, and it becomes rigid. The chest is rigid in pulmonary emphysema, hydrothorax, pleurisy with effusion, ossification of the costal cartilages, after fractures of ribs. Physiological increased rigidity presents in hypersthenic type of chest in comparison with asthenic chest.

Normal tactile fremitus (vocal fremitus) conducts equally in symmetric points.

Weakened tactile fremitus until the complete disappearance is described in obesity, and physiologically in hypersthenic chest. Weakened tactile fremitus is observed in a number of diseases: hydrothorax, pneumothorax, emphysema, foreign body in large bronchus.

Increased tactile fremitus is observed in consolidation of pulmonary tissue (pneumonia, pulmonary infarction, compression atelectasis), in presence of a lung cavity, communicating with the bronchus (tuberculous cavern, lung abscess).

3.6. Assessment tests on the theme "Subjective and Objective Examination of Patients with Respiratory System"

1. Characteristic symptoms of diseases of the lungs:
1. cough;
2. dyspnea;
3. asphyxia;
4. heartburn;
5. dysphagia.

2. Normal respiratory rate per minute:
1. 16 – 20;
2. 12 – 14;
3. 19 – 21;
4. more than 20;
5. more than 30.

3. Dyspnea with impaired respiratory rhythm includes:
1. Kussmaul’s respiration;
2. Biot’s respiration;
3. Cheyne-Stokes respiration;
4. Grocco’s (undulating) respiration;

5. Cellular respiration.

4. Cheyne-Stokes respiration is characterized by:
1. gradual increase and gradual extinction of breath;
2. periods of apnea;
3. short expiration;
4. short inspiration;
5. long expiration.

5. Characteristics of inspiratory dyspnea:
1. difficult inspiration;
2. difficult expiration;
3. difficult inspiration and expiration;
4. Cheyne-Stokes respiration;
5. Grocco's (undulating) respiration.

6. Characteristics of expiratory dyspnea:
1. difficult expiration;
2. difficult inspiration;
3. difficult inspiration and expiration;
4. bradycardia;
5. Kussmaul's respiration.

7. Characteristics of mixed dyspnea:
1. difficult inspiration and expiration;
2. difficult inspiration;
3. difficult expiration;
4. bradycardia;
5. Kussmaul's respiration.

8. Central cyanosis is characteristic of:
1. respiratory failure;
2. circulatory failure;
3. digestive system diseases;
4. uremia;
5. disorders of brain circulation.

9. Diffuse cyanosis is characteristic of:
1. respiratory failure;
2. circulatory failure;
3. digestive system diseases;
4. uremia;
5. anemias,

10. Peripheral cyanosis is characteristic of:
1. circulatory respiratory failure;
2. right ventricle failure;
3. circulatory failure;
4. left ventricle failure;
5. uremia,

11. Dyspnea with disorders of respiratory rhythm includes:
1. Kussmaul's respiration;
2. Blot's respiration;
3. Cheyne-Stokes' respiration;
4. vesicular respiration;
5. amphoric respiration.

12. Criteria of assessment of the chest shape are:
1. size of the epigastric angle;
2. contours of scapulas;
3. degree of manifestation of supra-and subclavicular fosses;
4. interrelation between anteroposterior and transversal dimensions of the chest;
5. width and angle of the ribs and intercostal spaces on the lateral surface of the chest.

13. It is typically of a normosthenic form of the chest:
1. epigastric angle is equal to 90 degrees;
2. supraclavicular fosses are well-marked, subclavicular fosses are smooth;
3. width of ribs is 1-1.5 cm, intercostal spaces are equal to 1-1.5 cm;
4. width of ribs is 2.5-3 cm, intercostal spaces are equal to 0.5-1 cm;
5. epigastric angle is less than 90 degrees.

14. It is typically of an asthenic form of the chest:
1. supra-and subclavicular fosses are well-marked;
2. epigastric angle is less than 70 degrees;
3. scapulae lie down not closely to the chest;
4. epigastric angle is equal to 90 degrees;
5. scapulae contours lie down closely to the chest.

15. It is typically for hypersthenic form of the chest:
1. epigastric angle is more than 90 degrees;
2. supra-and subclavicular fosses are smooth;
3. width of ribs is 2.0-2.5 cm, intercostal spaces are 0.5-1 cm;

4. ribs go almost horizontally;
5. width of ribs is 0.5-1 cm, intercostal spaces are 2-2.5 cm.

16. Pathological types of the chest are:
1. emphysematous chest;
2. paralytic chest;
3. foveated chest;
4. funnel-shaped chest;
5. kyphoscoliotic chest.

17. It is typically to the emphysematous form of the chest:
1. epigastric angle is more than 90 degrees;
2. ribs are wide, intercostal spaces are narrow;
3. interrelation between anteroposterior and transversal dimensions of thorax is about 1.0;
4. epigastric angle is less than 90 degrees;
5. interrelation between anteroposterior and transversal dimensions of thorax is less than 0.55.

18. It is typically to the paralytic form of chest:
1. epigastric angle is less than 90 degrees;
2. ribs are narrow, intercostal spaces are wide;
3. interrelation between anteroposterior and transversal dimensions of thorax are less than 0.55;
4. ribs are wide, intercostal spaces are narrow;
5. interrelation between anteroposterior and transversal dimensions of thorax is more than 1,0.

19. Enlargement of the one half of the chest is observed in:
1. accumulation of fluid in a pleural cavity;
2. accumulation of air in a pleural cavity;
3. fibrous changes in the lungs;
4. emphysema of the lungs;
5. presence of a cavity in the lung.

20. Palpation of chest is used for:
1. assessment of tactile fremitus (vocal fremitus);
2. assessment of resistance of the chest;
3. determination the localization of tender area of the chest;
4. assessment of inferior border of the lungs;
5. assessment of height of the lung apexes.

21. Increase of the tactile fremitus (vocal fremitus) is observed in:

1. infarct-pneumonia;
2. pneumosclerosis;
3. second stage of a lobar (croupous) pneumonia;
4. increased air filling of the lung (pulmonary emphysema);
5. accumulation of air in a plural cavity (pneumothorax).

22. Weakening the tactile fremitus (vocal fremitus) is observed in:
1. accumulation of air in pleural cavities (pneumothorax);
2. accumulation of fluid in a pleural cavity;
3. obturation atelectasis of a lung lobe;
4. increased air filling of lung (pulmonary emphysema);
5. pneumosclerosis.

23. What are characteristics of chest in pneumothorax?
1. increased volume of the affected part of the chest;
2. decreased respiratory excursions of the affected part of the chest;
3. subcutaneous emphysema;
4. increased rigidity of the chest;
5. increased tactile fremitus.

24. What are characteristics of the chest in hydrothorax?
1. increased volume of the affected part of the chest;
2. decreased respiratory excursions of the affected part of the chest;
3. forced position on the affected part of the chest;
4. increased rigidity of the chest;
5. weakened tactile fremitus.

25. What are characteristics of the emphysematous chest at palpation?
1. increased tactile fremitus;
2. symmetrically decreased respiratory excursions of the chest;
3. forced position on affected part of the chest;
4. increased rigidity of the chest;
5. weakened tactile fremitus (vocal fremitus).

Chapter 4. Percussion of the Lungs

Goals: to enable students to learn –

1) physical bases and technique of percussion;
2) types of percussion sound and its diagnostic value;
3) technique and diagnostic value of comparative percussion of the lungs;
4) technique and diagnostic value of topographic percussion of the lungs.

4.1. Percussion: physical bases and technique

Percussion is a diagnostic procedure designed to determine the density of a body part by the sound produced by tapping the surface with the *finger-hammer* (*plexor)*; performed primarily over the chest to determine the presence of the normal air content in the pulmonary tissue and over the abdomen, to evaluate the air in the loops of intestine, and borders of solid organs such as (the heart, the liver and the spleen**).**

Technique of percussion

By the technique, there are two types of percussion – **mediate (pleximetric) and direct (immediate) percussion** (Fig. 4-1).

Historically, ***mediate (pleximetric) percussion*** was performed by tapping on the object applied to the surface of the patient's body - a *pleximeter*, a plane plate made of metal or wood. Tapping on the pleximeter was performed by a *plexor* - percussion hammer.

The role of the pleximeter is now played by the middle phalange of II or III finger applied to the surface of the body. As a hammer at a mediate percussion, the slightly flexed index finger (*plexor*) of the other arm is used *("finger by finger" percussion*).

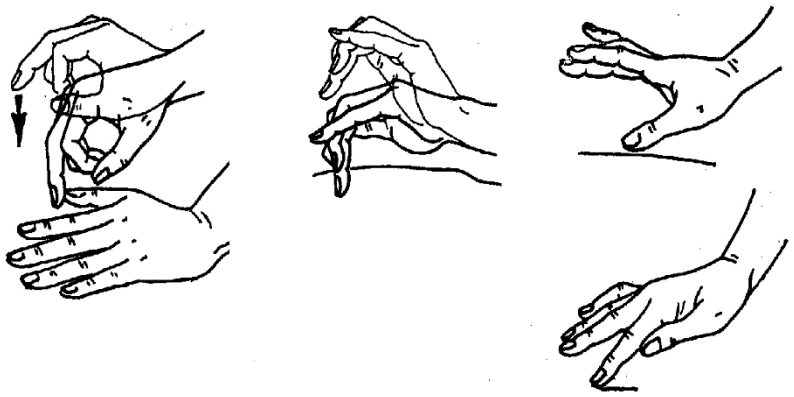

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Fig. 4-1. Types of percussion (O.G. Dovgyallo, 1986):

(1) mediate (pleximetric) percussion (“finger by finger”), (2) direct percussion by F.G. Yanovsky, (3) direct percussion by V.P. Obraztsov.

Direct (immediate) percussion is performed by tapping with distal phalanges of one or few flexed fingers directly on the surface of the patient's body (technique by V.P. Obraztsov or F.G. Janowski).

Direct percussion by Yanovsky is tapping the tip of distal phalange of the bent index or the middle finger on the surface of the patient's body. It is used for percussion on protruding dense formations (clavicle, ribs).

Direct percussion by Obraztsov is conducted by tapping to the chest with the palmar surface of the distal phalange of the index finger of the right hand. To increase the force of the stroke, an ulnar edge of the index finger is how to be hooked over the radial edge of the middle finger. It is waving the arm, not reaching the surface of the body 3 cm, the arm is suspended, the index finger jumps off the middle finger, and the distal phalange hits the chest.

Rules of mediate (pleximetric, "finger by finger") percussion

1. The room should be warm and quiet. The hands of the examiner should be warm to avoid unpleasant sensations in the patient and reflex muscle tension.

2. As pleximeter III (middle) of II (index) fingers of the left hand are used. Tightly, but without strong pressure, the finger-pleximeter is pressed to the chest surface. Thus, the force of the percussion is directed mainly deep into. The pleximeter should be located in the intercostal space and not be displaced on the ribs, as the bone increases the area of propagation of oscillations to the neighboring areas.

3. Percussion strokes are performed by the distal phalange of the middle finger of the right arm strictly perpendicular to the finger-pleximeter. The place of the contact is along the middle phalange or distal interphalangeal joint of this finger.

4. The percussion finger middle or distal phalanges should be bent at a right angle.

5. Percussion should always be of the same strength, short and jerky: the finger-flexors should jump off the pleximeter. To do this, it is recommended to strike with bending movements only in the wrist joint, with the immobility of the shoulder and elbow joints. A series of the two percussion strikes is applied.

6. Percussion of the lungs is usually carried out in a vertical position of a patient. The position of the patient's hands depends on which surface of the body the study is performed. When performing the percussion on the anterior surface of the chest, the patient's hands are lowered along the body. In the study on the lateral surface - the patient should put his hands behind his head. At the percussion on the back of the chest, the patient should cross his/her hands on the chest for widening interscapular space.

A physician at percussion on the anterior and lateral surfaces of the chest should be in front of the patient, and on the back – behind the patient.

Strength of percussion strike

According to the purposes, we can use *lightest (quietest, weak, threshold), medium strength (quite, light) and strong (loud) percussion*. Distribution of the acoustic waves in the depth and on the surface of the tissues, and percussion sound intensity depend on the strength of the percussion stroke.

Sphere of percussion is the area of the tissue coming to oscillate under the influence of the percussion strike (Fig. 4-3). The stronger the percussion strike will be, the greater the sphere of percussion is. The depth of the percussion sphere at: weak percussion is 2-3 cm, medium percussion – 4-5 cm, loud percussion – not more than 7 cm. Percussion does not find pathological changes in the tissue located at great depth (more than 7cm).

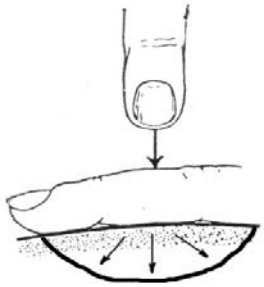

Fig. 4-3. Sphere of percussion

Basic types of the percussion sound

Resonant sound *(or literal translation from Russian "ясный лёгочный звук"- clear pulmonary sound)* is heard at percussion over the unaltered lungs. Characteristics of resonant sound depend on the presence of air in the organ and the elasticity of the pulmonary tissue. Resonant sound is a long, low, loud. The strength and height of pulmonary sounds vary depending on the physiological conditions - the age, characteristics of the chest, muscles development, and thickness of the subcutaneous layer (Fig. 4-2).

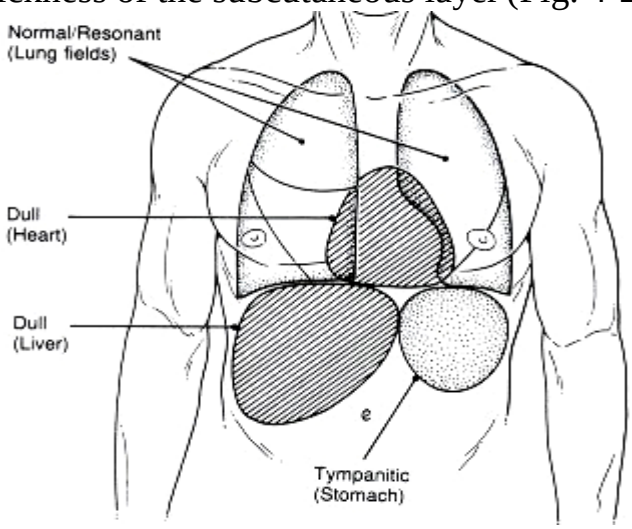

Fig. 4-2. Normal location of percussion sounds.

Dull sound presents in the norm at percussion of solid organs and tissues - heart, liver, spleen, large muscles and bones (not covered by lungs). It is short, soft sound of a high timbre. *Hyporesonant kind of the dull sound* presents above the solid organs (liver, heart) in areas covered by the thin layer of the pulmonary tissue. Under these conditions the sound becomes a lightly quieter, shorter, and higher with a comparison of the normal resonant sound.

Tympanic sound is heard above the cavity containing the air. It is the longest, lowest, loudest sound with a musical tint. In the norm, tympanic sound presents above abdomen (loops intestines and gas buble of the stomach), and only at the one place of the chest in the "*semilunar space of Traube*" (the inferior-lateral part of the left side of the chest). This space is placed between superiorly - inferior edge of the left lung, on the right – left edge of the liver, on the left – spleen, and below – costal arch. Here, the gastric fundus with gas bubble applies to the chest wall that explains the tympanic sound.

4.2. Comparative percussion of the lungs

Comparative percussion is performed with a comparison of the percussion sound (resonance) on the symmetrical points of the appropriate topographical lines of the chest (Table 4-1, Fig. 4-4).

Table 4-1. Topographical lines of the chest used for percussion of lungs

Topographical lines	Position
Lin. parasternalis dextr. et sin. (parasternal right [left] line)	exactly in the middle between the media-clavicular and the sternal lines
Lin. medioclavicularis dextr. et sin. (mediaclavicular/midclavicular right [left] line, mammary line)	from the middle of the clavicle and perpendicularly downwards
Lin. axillaris anterior et posterior dextr. et sin. (anterior/posterior axillary right [left] line)	accordingly on the anterior and posterior edges of the axillary space
Lin. axillaris media dextr. et sin. (mid-axillary right [left] line)	downwards from the middle of the axillary space
Lin. scapularis dextr. et sin. (scapular right [left] line)	at the inferior scapular angle
Lin. paravertebralis dextr. et sin. (paravertebral right [left] line)	in the middle between the posterior median and scapular lines

Normally, the same resonant sound is determined above symmetrical areas of the chest. Any asymmetry of percussion sounds often indicates a pathological process in the lungs. Comparative percussion allows to identify

these deviations. *Light percussion is used to examine the surface areas of the lungs, medium strength (louder) percussion - to search for deeper located lesions.*

Comparative percussion is performed on proper topographic lines of the chest (Fig. 4-4). A physician compares a percussion sound, first on the right, then on the left on the symmetric points of the topographic lines from top to bottom. In case of pathology of the right lung or the right part of the chest, the percussion sound on the points of the healthy left side is compared firstly with the right ones.

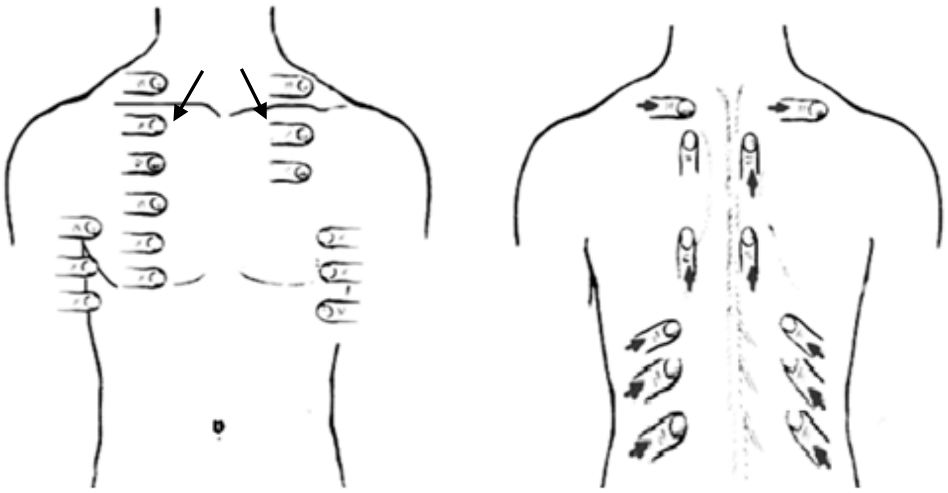

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Fig. 4-4. Comparative percussion of lungs:
(1) – anterior side, arrows indicate percussion site on clavicles;
(2) – posterior side, arrows indicate position of the finger-pleximeter.

Comparative percussion on the anterior surface starts on lung's apexes. The finger-pleximeter is alternately located at supraclavicular fossae. Then strikes are directly applied to clavicles (by immediate percussion), further - at I and II intercostal spaces on both sides (“finger by finger” percussion). On midclavicular and parasternal lines, comparative percussion is done to II interspace on the left side, since the level of the detected cardiac dullness (Fig. 4-5, 4-6, 4-7). Further percussion continues below III rib only on the right side. Here a physician alternately compares a percussion sound of overlying intercostal space with the underlying ones. The percussion sound may be louder above the left lung apex as its volume is more than the right lung apex.

Comparative percussion on axillary lines starts from the axillary fossae, the finger-pleximeter is placed collateral to the ribs. During the percussion on the axillary lines, the patient's hands should be crossed over the head. Normally dull sound is revealed due to the closeness of the liver dullness inferiorly on the right axillary line, on the left at the same level – a tympanic sound, as here it is close to the space of Traube. When performing comparative percussion along the scapular lines, the patient's hands should be

crossed on the chest, while the scapulae move apart and the interscapular space is widened. A finger-pleximeter in supraspinal areas must be in a horizontal position, at the interscapular region - vertically, below the inferior angles of the scapulae -again horizontal ones (Fig. 4-8, 4-9).

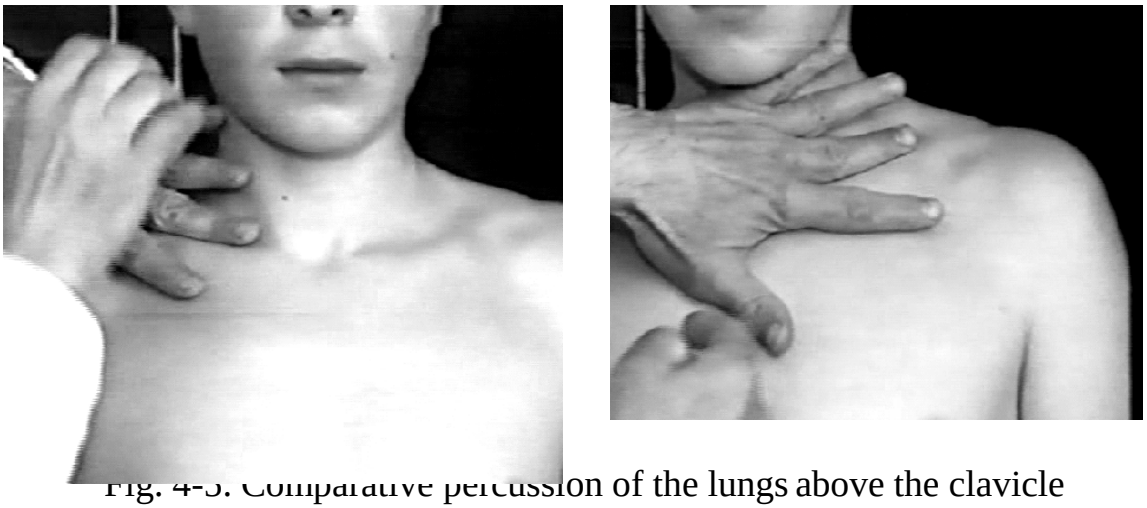

Fig. 4-5. Comparative percussion of the lungs above the clavicle

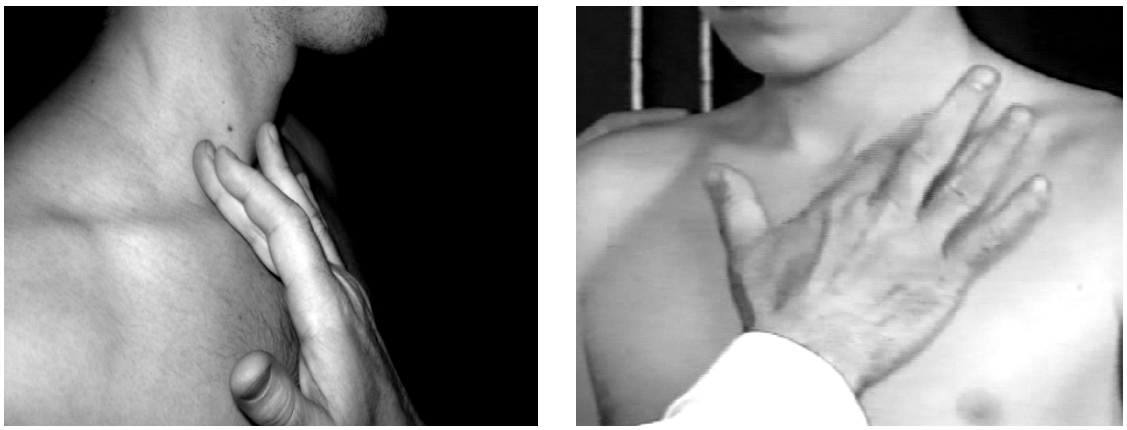

Fig. 4-6. Comparative percussion of the lungs on the clavicle (by the method of Obraztsov)

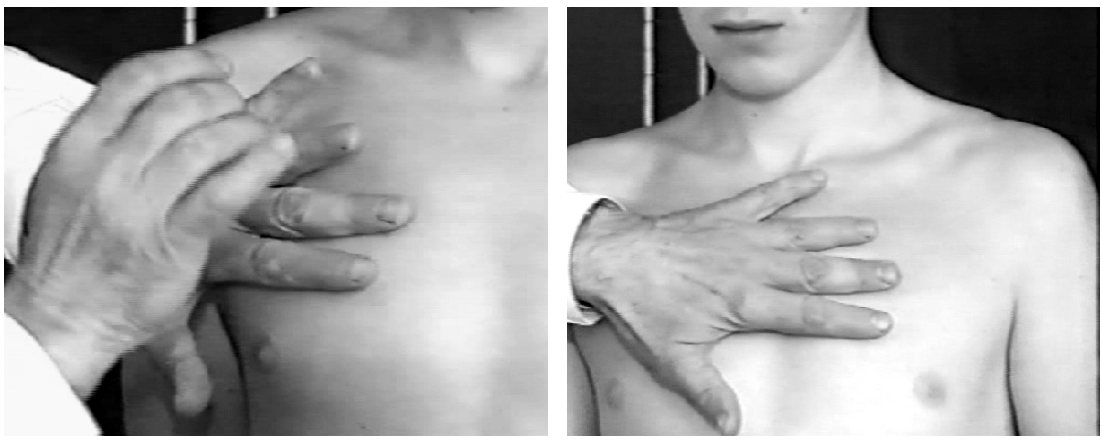

Fig. 4-7. Comparative percussion of the lungs below the clavicle at II interspace.

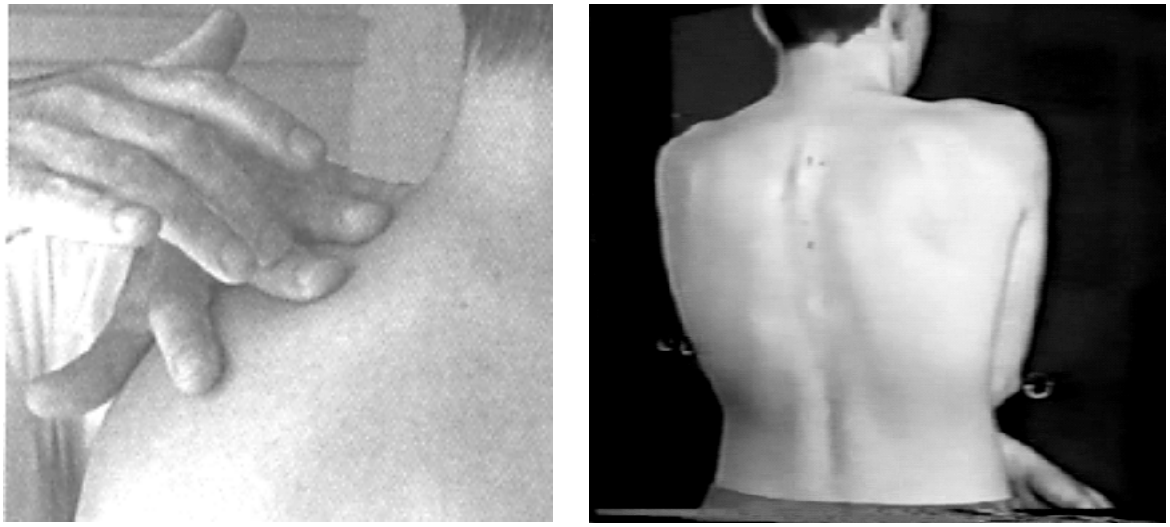

Fig. 4-8. Comparative percussion of the lungs posteriorly (percussion above the scapula spine, position of the patient´s hands).

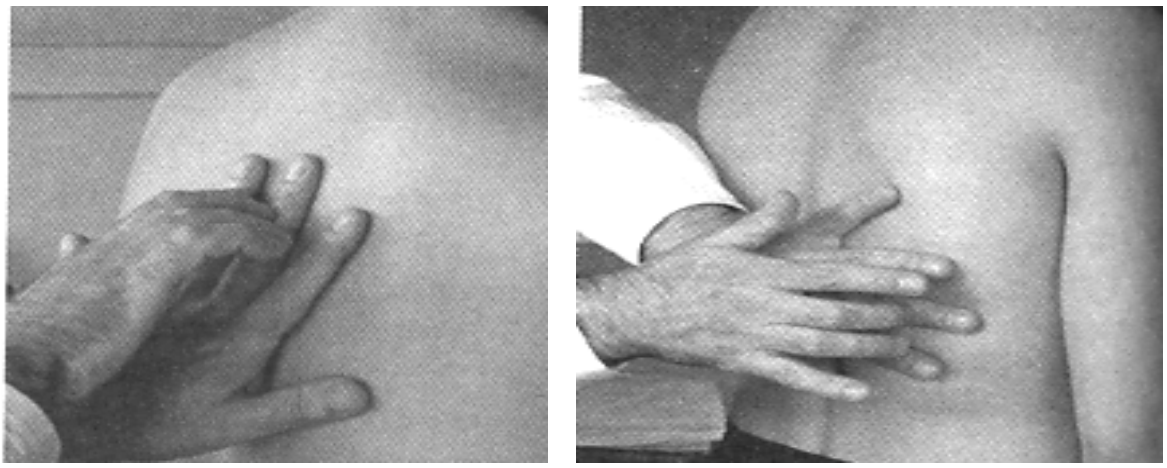

Fig. 4-9. Comparative percussion of the lungs posteriorly (percussion between the between the shoulder blades, and below the angle of the scapula)

In pathology, the following sounds can be detected above the lungs:

1) Dull sound is defined in the complete disappearance of air from the lung in II and III stage of lobar pneumonia (stages of the *red and grey hepatization*), in lung tumors of a large size, acute lung abscess before opening, accumulation of fluid in the pleural cavity (exudate or transudate). The dull sound is high in pitch, quiet and short. The *hyporesonant kind of the dull sound* is detected in case of decreased airiness of a limited part or in the whole lung: focal pneumosclerosis (replacement of the pulmonary by connective tissue); inflammatory infiltration of the pulmonary tissue – focal pneumonia, in I and IV stages (stages of *congestion and resolution*) of lobar pneumonia; pulmonary edema in left ventricular heart failure; compression of

the pulmonary tissue with the pleural fluid (compressive atelectasis); complete obstruction of large bronchus and gradual air resorption in the lung (obturator atelectasis) (Table 4-2):

2) Flat sound (stony flat sound, or stony dull sound) is an extremely soft, high and short sound. It presents in the large pleural effusion, and normally over solid areas such as the large bones. The upper limit of dullness in the form of an oblique line (*Damoiseau's curve*) presents in exudative pleurisy (Fig. 4-10).

Table 4-2. Diagnostic value of percussion sounds of the lungs

Percussion sound	Norm	Pathology
Resonant sound	Lungs	-
Dull sound	Liver, heart, femur	Lung infiltration, atelectasis, fluid in the pleural cavity
Flat sound (stony flat sound)	Over solid areas such as large bones	Large volume of fluid in the pleural cavity
Tympanic sound	Traube's semilunar space, gas in stomach and intestines	Air cavity in the lung connected with bronchus, pneumothorax
Hyperresonant sound	-	Pulmonary emphysema

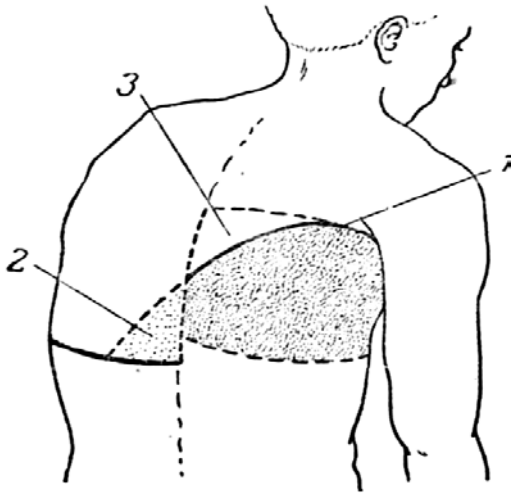

Fig. 4-10. Percussion data in exudative pleurisy:
1 - Damoiseau's curve; 2 - Rauchfuss-Grocco's triangle; 3 – Garland's triangle.

The highest level of a stony dull sound on the posterior axillary line, and the lowest – on the paravertebral line. With large effusions, two triangles are conventionally isolated: *Garland's triangle* (it is formed by Damoiseau's

curve and the spine, this is the area of compression atelectasis of the lung with a dulled tympanic percussion sound above it) and *Rauchfuss-Grocco's triangle* (due to the displacement of the of mediastinal organs to the healthy side with a dull sound above it)

(3) Tympanic (from Greek tympanon – drum) sound - occurs in the presence of a large, smooth-walled air-containing cavity with a diameter of at least 5 cm. This sound is very loud, long, reminiscent of the sound of the drum. In height, it can be high (with a cavity of a small size) or low (with large cavities). According to its characteristics, it is close to the musical tone, as it is the result of the oscillation of a homogeneous substrate in the cavity – air. This sound happens when the lung abscess after opening, tuberculous cavity, in presence of air in the pleural cavity (pneumothorax).

Sometimes, in the presence of a large surface-located smooth cavity the sound can resemble a blow to the metal. This kind of tympanic sound is called a ***metallic percussion sound***. Another type of the sound is the so-called ***cracked pot sound***. It occurs in the presence of a lung cavity, communicating with the bronchus through a very narrow, slit-like hole. Since the tone of the walls of such a cavity is reduced, with the percussion a kind of rattling sound occurs that is like reminiscent of the sound when hitting a cracked pot.

4) Hyperresonant sound *(or literal translation from Russian "коробочный звук"- box, or bandbox, sound)* is detected in increasing the airiness of the lung tissue in pulmonary emphysema. This also reduces the elasticity of the lung tissue. When comparing with a resonant sound in emphysema the percussion sound becomes even lower, louder and longer, acquires a tympanic timbre. It resembles the sound of tapping an empty box.

4.3. Topographic percussion of the lungs

Topographic percussion of the lungs is used to determine the borders of the lungs, width of the lung's apices (Kroenig's area), and the mobility of the inferior edge of the lungs.

Rules of topographic percussion

1) Topographic percussion is always performed *from a resonant sound in the direction of the dull sound*.

2) The *weak (light) percussion* is used.

3) The percussion is conducted in a vertical position of a patient. If unable to comply with this rule a patient can be in horizontal position. The breath of the patient should be superficial.

4) The doctor sits or stands in front of the patient when percussion is performed on the anterior and lateral surfaces of the chest, and behind the patient - when percussion is performed on the posterior surface of the chest.

5) The *border of the lung is marked on that side of a finger-pleximeter, which is reversed to a more resonant sound.*

Topographic percussion of the superior border of the lungs

To determine the ***height of the lung apex (superior border of the lung)*** anteriorly, the finger-pleximeter is placed in the supraclavicular fossa parallel to the clavicle, and in the course of percussion it is shifted upwards and medially along the *m. scalenus media* (or in other words along posterior edge of *m. sternocleidomastoideus* to *processus mastoideus*) to dullness (Fig. 4-11). ***Normally, the height of the lung apex anteriorly is 3-4 cm above the clavicle, while the left apex is often 0.5 – 1 cm above the right one.***

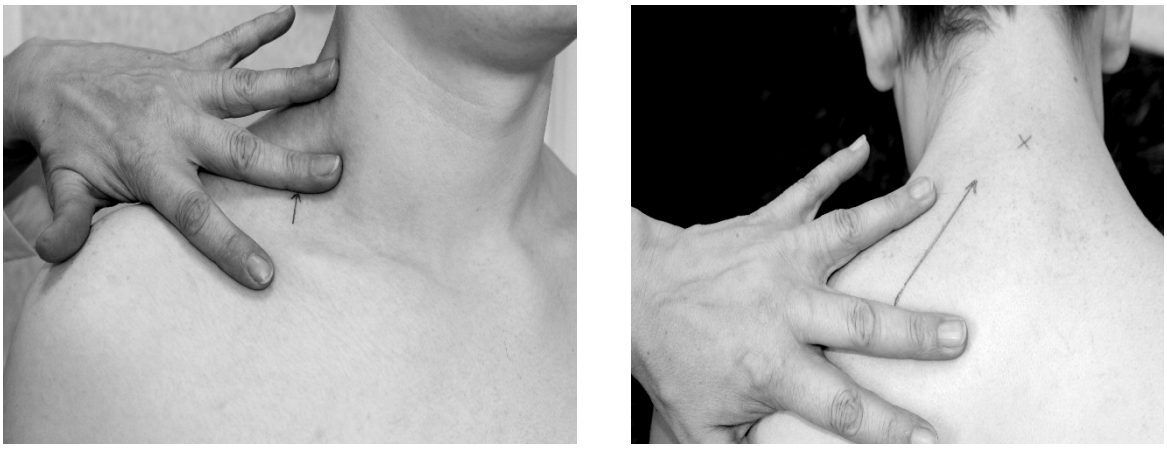

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Fig. 4-11.Topographic percussion of the superior border of the lung (height of the lung apices): 1- anterior side, 2 – posterior side.

When determining the height of the lung apex on the back, the finger-pleximeter is placed collateral spina scapula, and moved up and medially in direction to the spinous process of VII cervical vertebrae (C7). Normally, the ***lung apex posteriorly is determined on the level of spinous process of C7.*** On the right, the lung apex is usually 1 cm lower, as the right bronchus of the superior lobe is narrower, and in addition, the muscles of the shoulder girdle are more developed on this side.

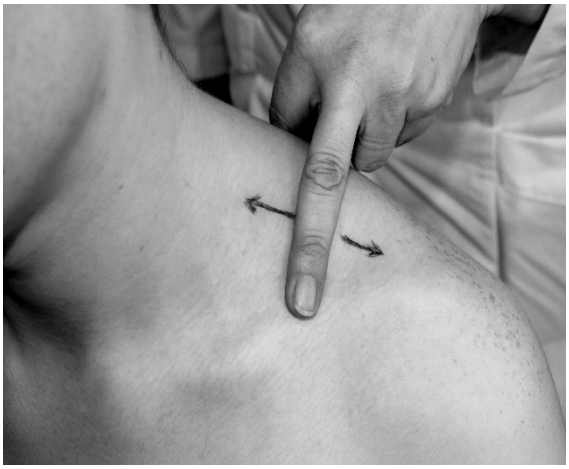
Width of the lung apex, or Kroenig's area, is determined by percussion on the anterior edge of the trapezius muscle. The finger-pleximeter is applied at the middle of trapezius muscle perpendicular to its anterior edge, and then is moved in medial and lateral directions to a dull sound (Fig. 4-12). ***Normally, Kroenig's area width is 4-7 cm, at the right side 1-1.5 cm less, but may vary depending on the type of constitution from 3 to 8 cm.***

The lung apex is widened and elevated often in pulmonary emphysema. The lung apex width and height are decreased in consolidation of the pulmonary tissue (tuberculosis, cancer, pneumosclerosis), and in obturator atelectasis of the superior lobe of the lung.

Topographic percussion of the inferior border of the lungs

Percussion of the ***inferior border of lungs*** is carried out from the top

to bottom along the symmetrical topographic lines on the left and right. However, on the left it is usually not determined by the two lines – parasternal and midclavicular lines. In the first case, this is due to the fact that at III rib on the left the border of the relative cardiac dullness locates, and, thus, this level does not reflect the true border of the lung. With regard to the midclavicular line, the definition of the inferior border of the lung it is difficult because of the tympanic sound over the *space of Traube* (gas bubble in the body of the stomach). When determining the inferior borders of the lungs, the finger-pleximeter put in the intercostal spaces parallel ribs, moving it down to the dull sound. The latter is formed during the transition from the inferior edge of the lung to the diaphragm and hepatic dullness. The mark of the border is carried out on the edge of the finger, facing the resonant sound.



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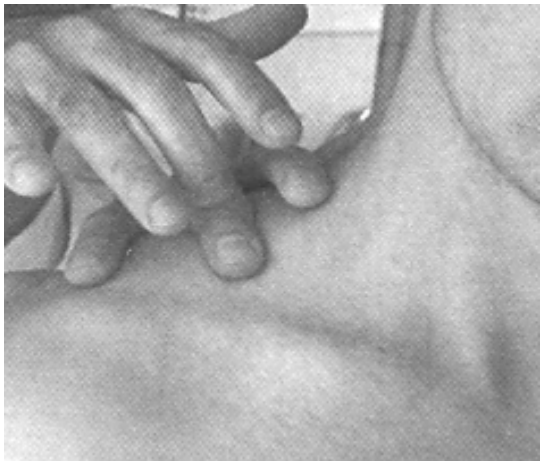

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Fig. 4-12.Topographic percussion of the Kroenig's area:
1 – start point and direction of percussion, 2 - percussion at the anterior edge of m. trapezius.

Since the percussion is conducted on intercostal spaces, then to clarify the border of the lungs it is necessary to recheck it as well as along the ribs. ***For more precise determination of the inferior border on l. parasternalis dextra, direct percussion according to Obraztsov is carried out on two superposed ribs above the dullness (Fig. 4-13).*** The upper (control) rib lies obviously above the pulmonary tissue and serves as the control, it generates a resonant sound. Next, the second (test) rib lied directly above a dull sound is percussing. If at percussion of this rib, the same sound is obtained as above overlying control rib, it means the border lays on the inferior edge of this rib. If the sound on the lower rib is a little dull (hyporesonant), the liver lies under this rib, and the border of the lung will be marked on the superior edge of the upper rib that most often occurs. In the normosthenic chest, the inferior border of the lungs is at the following location (Table 4-3).

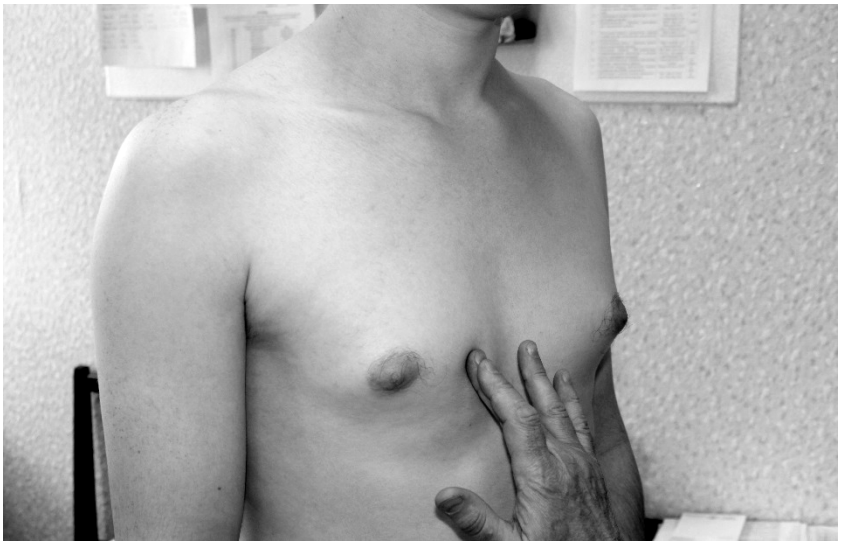


Fig. 4-13. Specification of the inferior border of the right lung parasternal line by percussion according to Obraztsov

Table 4-3. Normal position if the inferior border of the lungs

Topographical lines	Position of lower border of the lungs
Lin. parasternalis dextr.	the upper edge of 6-th rib
Lin. medioclavicularis dextr.	the inferior edge of 6-th rib
Lin. axillaris anterior dextra et sinistra	the inferior edge of 7-th ribs
Lin. axillaris media dextr. et sin.	the inferior edge of 8-th ribs
Lin. axillaris posterior dextr. et sin.	the inferior edge of 9-th ribs
Lin. scapularis dextr. et sin.	the inferior edge of 10 ribs
Lin. paravertebralis dextr. et sin.	the level of the spinous process of the 11-th thoracic vertebra

Lowering inferior border of lungs is observed in (Table 4-4):

- low standing of the diaphragm with a sharp lowering abdominal organs (enteroptosis);
- emphysema;
- pneumothorax.

Elevation of inferior border of lungs occurs in:

- Inflammatory processes in the lung tissue (lobar pneumonia, infiltrative pulmonary tuberculosis, abscess and gangrene of the lung, when the cavity is filled with purulent contents, pulmonary infarction, lung tumor of a large size);

- Fluid accumulation in the pleural cavity (exudative pleurisy, hydrothorax). The inferior border of the lung on the affected side corresponds to the oblique Damoiseau's curve;
- Obstructive atelectasis and pulmonary fibrosis;
- Bronchiectasis filled with a secret or surrounded by a massive layer of the infiltrated lung tissue;
- High intra-abdominal pressure caused by accumulation of fluid in the abdominal cavity (ascites), gas in the intestine (meteorism), excessive deposition of fat, pregnancy, which leads to a high standing of the diaphragm.

Table 4-4. Diagnostic value of changes in position of the lungs borders

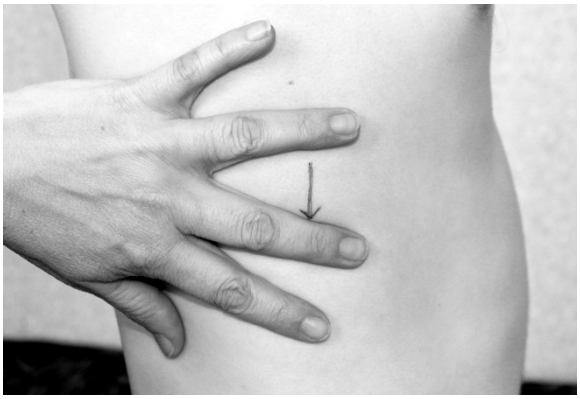
Changes in position of the lungs borders	Causes
Lowering inferior borders	1. Low position of diaphragm (splanchnoptosis, asthenics), 2. Pulmonary emphysema, 3. Accumulation of air in pleural cavity.
Elevation of inferior borders	1. High position of diaphragm (hypersthenics, pregnancy, obesity, meteorism, ascites, acute perforation of gastric or duodenal ulcer), 2. Contraction (or cicatrization) of inferior lobes of the lungs in inferior lobe pneumonia, pneumosclerosis, obturator atelectasis, 3. Significant enlargement of the liver or spleen (hepatosplenomegaly).
Lowering superior borders	Atelectasis or pulmonary fibrosis of superior lobes of the lungs (due to pneumonia, tuberculosis, and cancer)
Elevation of superior borders	Pulmonary emphysema, air accumulation in pleural cavity

Active respiratory mobility of the inferior edge of the lung is determined by the percussion of the inferior border of the lung on a deep inspiration and a deep expiration on the right side by 3 lines (l. medioclavicularis, l. axillaris media, l. scapularis) and 2 lines on the left side (l. axillaris media, l. scapularis). The normal mobility of the inferior border of the lungs is described in Table 4-5. *First point* is marked at inferior border of the lung determined by these lines at the quiet respiration (Fig. 4-14). Then the patient is asked to inspirate completely and to hold breath, and percussion is continued by moving the pleximeter downwards to complete dullness, where the second point should be marked by dermograph at the upper edge of the pleximeter-finger. Next, the patient is then asked to breathe out a

maximum air from the lungs and to hold breath again. The percussion is continued in the downward direction from a starting point until the clear vesicular resonance disappears. The third dermographic point should be marked at the point where the dull sound is heard. The distance between the extreme second and third points is measured. It corresponds to the active respiratory mobility.

Table 4-5. Active respiratory mobility of the inferior edge of the normal lungs

Topographical lines	Active respiratory mobility(cm)
Lin. medioclavicularis dextr.	4-6
Lin. axillaris media dextr. et sin.	6-8
Lin. scapularis dextr. et sin.	4-6



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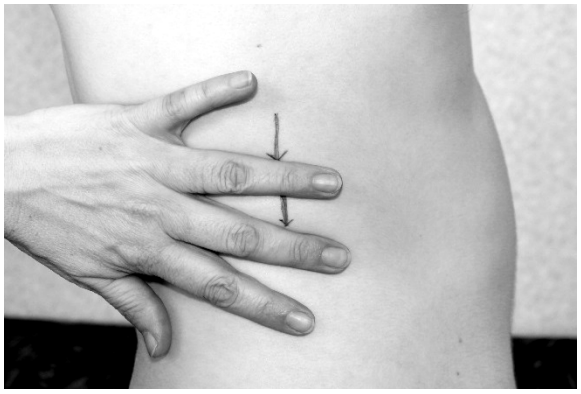

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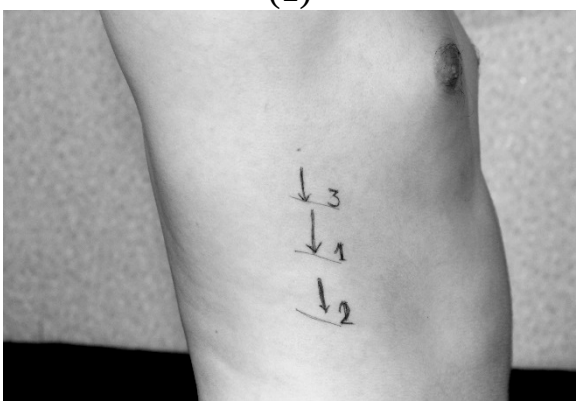


(3)

Fig. 4-14. Percussion of the active respiratory mobility of the inferior border of the lung on *l. axillaris media dextr*. :

(1) delimitation of the inferior border during quiet respiration,

(2) delimitation of the inferior border at maximum inspiration,

(3) delimitation of the inferior border at maximum expiration.

Normally, the mobility of the inferior edge of the lung on the right midclavicular and scapular lines is 4-6 cm (2-3 cm for inspiration and

expiration), for midaxillary lines - 8 cm (3-4 cm for inspiration and expiration).

Active respiratory mobility of the inferior edge of the lungs is reduced in pneumonia, pulmonary edema, emphysema, pneumosclerosis, pleurisy, accumulation of fluid and air in pleural cavity, in pleural adhesion or obliteration of pleural cavities, high position of diaphragm (in obesity, meteorism, ascites, pregnancy).

Passive mobility of the inferior edge of the lungs is examined at the change of the patient's body position. When changing a position from vertical to horizontal, the inferior border of lungs is lowered about 2 cm, and the inferior border of the right lung lying on the left side can drop to 3-4 cm. Passive mobility of the inferior edge of the lung can be reduced in pleural adhesions or obliterations of the pleural cavities after passed pleurisy.

4.4. The key points on the theme "Percussion of the Lungs"

Basic Types of Percussion sound:

Resonant sound is heard at percussion over the unaltered lungs. Resonant sound is a long, low, and loud.

Dull sound presents in the norm at the percussion of the solid organs and tissues not covered by the lungs (heart, liver, spleen, large muscles, and bones). It is a short, soft sound of high timbre. *Hyporesonant kind of the dull sound* presents above solid organs (liver, heart) in the areas covered by the thin layer of the pulmonary tissue.

Flat sound is an extremely soft, high and short dull sound (*stony flat sound*). It presents normally over the solid areas such as large bones.

Tympanic sound is heard above the cavity containing air. It is the longest, lowest, loudest sound with a musical tint. In norm, tympanic sound presents above the abdomen, and only at the one place of the chest in "semilunar space of Traube".

Comparative percussion is performed with a comparison of the percussion sound (resonance) on the symmetrical points of the appropriate topographical lines of the chest.

Normally, the same resonant sound is determined above the symmetrical areas of the chest. Any asymmetry of the percussion sounds often indicates a pathological process in the lungs.

Weak (light) percussion is used to examine the surface areas of the lungs, *medium strength (louder) percussion* - to search for deeper located lesions.

In pathology, the following sounds can be above the lungs:

Dull sound is defined in the complete disappearance of theair from the lung - at II and III stages of lobar pneumonia, in the lung tumors, acute lung abscess before opening, accumulation of fluid in the pleural cavity. *Hyporesonant kind of the dull sound* is detected the decreased airiness of a

limited part of the lung: focal pneumosclerosis, focal pneumonia, at I and IV stages of lobar pneumonia, atelectasis.

Flat sound (stony flat sound, or stony dull sound) presents in the large pleural effusion. *Hyperresonant sound* is detected in increasing airiness of the lung tissue (in pulmonary emphysema). *Tympanic sound* is heard above the cavity containing air (in pneumothorax, drained lung abscess).

Topographic percussion is used to determine the borders of the lungs, width of lung's apices (*Kroenig's area*), and mobility of the inferior edge of the lungs. Topographic percussion is always performed from a resonant sound in direction of a dull sound. *The weak (light) percussion is used*.

Widening borders of lungs (elevation of superior border, and lowering inferior border) is observed in pulmonary emphysema, air accumulation in pleural cavity, and a low position of the diaphragm (for inferior border).

Restriction of lung borders (lowering superior border, and elevation of the inferior border) is found in contraction (or cicatrization) of the lung lobes in pneumonia, pneumosclerosis, obturator atelectasis, and fluid accumulation in the pleural cavity and high position of the diaphragm (for inferior border).

Active respiratory mobility of inferior edge of the lungs is reduced in pneumonia, pulmonary edema, emphysema, pneumosclerosis, pleurisy, accumulation of fluid and air in pleural cavity, in pleural adhesion or obliteration of pleural cavities, high position of diaphragm (in obesity, meteorism, ascites, pregnancy).

4.5. Assessment tests on the theme "Percussion of the lungs"

1. Comparative percussion of lungs is used for detection of:
1. pathological foci in the lungs;
2. the width of Kroenig's area;
3. respiratory excursions;
4. borders of the lungs;
5. all answers are true.

2. Normal sound of lungs apexes percussion:
1. resonant;
2. tympanic;
3. dull;
4. hyperresonant;
5. flat.

3. Rules of comparative percussion are the following:
1. percussion by intercostal spaces;
2. beginning with an anterior surface of the chest;
3. percussion strictly on symmetric fields of the chest;
4. quiet or medium percussion;

5. lightest percussion.

4. Rules of topographic percussion are the following:
1. percussion by intercostal spaces;
2. beginning with an anterior surface of the chest;
3. quiet percussion;
4. percussion strictly on symmetric fields of the chest;
5. loud or medium percussion.

5. Elevation of the height of the lung apex presents in:
1. pulmonary emphysema;
2. pneumothorax;
3. superior lobe pneumonia;
4. superior lobe obturator atelectasis;
5. tumor of superior lobe of the lung.

6. Lowering the height of lung apex presents in:
1. superior lobe pneumonia;
2. superior lobe obturator atelectasis;
3. tumor of the superior lobe of the lung;
4. pulmonary emphysema;
5. pneumothorax.

7. Widening Kroenig's area presents in:
1. pulmonary emphysema;
2. pneumothorax;
3. superior lobe pneumonia;
4. superior lobe obturator atelectasis;
5. tumor of superior lobe of the lung.

8. Elevation of inferior border of the lung presents in:
1. inferior lobe pneumonia;
2. inferior lobe obturator atelectasis;
3. exudative pleurisy;
4. hydrothorax;
5. pulmonary emphysema.

9. Lowering inferior border of the lung presents in:
1. pulmonary emphysema;
2. visceroptosis;
3. exudative pleurisy;
4. hydrothorax;
5. inferior lobe obturator atelectasis.

10. **Normal height of the lungs apex anteriorly is:**
1. 3-4 cm above clavicles;
2. at the level of a clavicle;
3. 1 cm above clavicle;
4. 8 cm above clavicle;
5. 1 cm below clavicle.

11. **Normal width of Kroenig's area is:**
1. 3-4 cm;
2. 4-7 cm;
3. 6-8 cm;
4. 1-3 cm;
5. 7-9 cm.

12. **Normal position of the inferior border of the lung on the parasternal right line is:**
1. inferior edge of V rib;
2. inferior edge of VI rib;
3. superior edge of V rib;
4. superior edge of VI rib;
5. inferior edge of VII rib.

13. Normal position of the inferior border of lung on the midclavicular right line is:
1. inferior edge of VI rib;
2. superior edge of VI rib;
3. superior edge of V rib;
4. inferior edge of V rib;
5. inferior edge of VII rib.

14. **Normal position of the inferior border of the lung on the anterior axillary lines is:**
1. superior edge of VII rib;
2. inferior edge of VI rib;
3. inferior edge of VII rib;
4. superior edge of VI rib;
5. inferior edge of VIII rib.

15. **Normal position of the inferior border of the lung on the midaxillary lines is:**
1. inferior edge of VIII rib;
2. inferior edge of VII rib;
3. superior edge of VIII rib;

4. superior edge of VII rib;
5. inferior edge of IX rib.

16. **Normal position of the inferior border of the lung on the posterior axillary line is:**
1. superior edge of VIII rib;
2. inferior edge of VIII rib;
3. superior edge of IX rib;
4. inferior edge of IX rib;
5. inferior edge of X rib.

17. **Normal position of the inferior border of the lung on the scapular line is:**
1. inferior edge of X rib;
2. inferior edge of IX rib;
3. superior edge of X rib;
4. superior edge of IX rib;
5. at the level of spinous process of XI thoracic vertebra.

18. **Restriction of Kroenig's area presents in:**
1. superior lobe pneumonia;
2. superior lobe obturator atelectasis;
3. tumor of superior lobe of the lung;
4. pulmonary emphysema;
5. pneumothorax.

19. **Active respiratory mobility of the inferior border of the lung on the right midclavicular line in norm is:**
1. 4-6 cm;
2. 7-8 cm;
3. 3-5 cm;
4. 2-4 cm;
5. 6-9 cm.

20. **Restriction of active respiratory mobility of the inferior borders of the lung presents in:**
1. inferior lobe pneumonia;
2. inferior lobe obturator atelectasis;
3. dry pleurisy;
4. pulmonary emphysema;
5. congested lungs.

Chapter 5. Auscultation of the Lungs

Goals: to enable students to learn –

1) physical bases and rules of the lung auscultation;
2) Concept of main and adventitious (additional) breath sounds, the mechanism of their genesis, and their diagnostic value;
3) Character of the main breath sounds in norm and in pathology, diagnostic value;
4) auscultation rules, classification, diagnostic value and differentiation of adventitious breath sounds rhonchi, wheezes, coarse crackles, fine crackles, and pleural rub);
5) technique and diagnostic value of bronchophony.

5.1. Method of Auscultation

Auscultation (Latin *auscultare* listen) is a method of examination based on listening to the sounds inside the body. The founder of the method of auscultation was the French doctor Rene Laennec. In 1816-1819, he described and implemented an auscultation of the lungs in medical practice. At the same time, Laennec invented the first stethoscope. He first confirmed the diagnostic value of auscultation compared its data and the results of autopsies of deceased patients. Thanks to the work of Laennec, auscultation has become a mandatory method of clinical diagnosis worldwide.

Auscultation method is differentiated in:

direct auscultation - produced by applying the ear to the patient's body,

mediated auscultation - made with a special tube - stethophonendoscope (from the Greek words stethos – chest, phone - sound, scopeo – look).

Stethophonendoscope is a device that intensifies auscultatory sounds. It consists of a bell (*stethoscope*) and a flat chamber closed at its contact with the skin by a thin plastic diaphragm (*phonendoscope*). A phonendoscope is most useful in picking up low pitched sounds from the respiratory tract. The bell stethoscope is more suited to picking up the higher pitched heart sounds.

Auscultation rules

Special rules should be followed during auscultation:

1. The room should be quiet and warm.
2. The skin at places of auscultation should be hairless, or hairs on the chest should be moistened by a cotton with warm water, because hairs produce additional friction sounds that interferes with differentiation and interpretation of the sounds.
3. The lungs are listened to in a vertical position of the patient (standing or sitting), in a serious condition the patient can be listened to in a supine position.
4. Auscultation of the lungs, as well as percussion should be comparative. *The sequence of lungs auscultation* is the same as in the compara-

tive percussion (see Chapter 4. Fig. 4-4). Auscultation is performed in both phases of breathing (inspiration and expiration) on strictly symmetric parts of the chest, with a uniform depth of breathing.

5. Listening to the lungs, unlike percussion, is carried out not along topographic lines, but on the areas starting with the supraclavicular areas (the area of the tops of the lungs), then the area of the pectoralis major muscles and the lower lateral parts of the anterior surface of the chest. When listening to the axillary areas the patient rises hands behind his/her head, then the physician listens to the side surfaces of the chest. On the back surface of the chest, auscultation of the lungs begins with suprascapular regions (the projection of the tops of the lungs behind it) then to the interscapular region, for this the patient should cross his/her arms. Then the physician listens to the area below the corners of the scapular bones and lower lateral sections.

6. The auscultation is carried out at least 2-3 points in every area, so as to assess an auscultation picture at one point is impossible, then just hold on auscultation symmetrical area of the opposite side.

7. At first, the main breath sounds are analyzed, while the patient's breathing should be through a half-opened mouth and a medium depth.

8. Then ask the patient to breathe deeply and through the mouth, with a better identified side of the breath sounds. For the same purpose, if necessary, ask the patient to cough, exhale quickly and sharply.

All auscultative phenomena on the part of the respiratory organs can be divided into main (basic) and adventitious (additional) breath sounds (Table 5-1).

Table 5-1. Main breath sounds in norm

Type of sound	Mechanism of genesis	Sound characteristics	Duration of sound	Place of auscultation
Vesicular (alveolar) breathing	vibration of alveolar walls during their filling with air in inspiration	simulate the sound *“F”*	entire inspiration and first third of expiration	over pulmonary tissue
Bronchial (laryngotracheal) breathing	arise in the larynx and the trachea as air passes through the vocal slits	resembles the sound of Russian letter *"X"*	length and intensity of inspiration and expiration are the same	over the larynx, the trachea, and at points of projection of the tracheal bifurcation

Main breath sounds:

- *vesicular breathing;*
- *bronchial breathing.*

Adventitious (additional) breath sounds:

- *rhonchi;*
- *wheezes;*
- *coarse crackles;*
- *fine crackles;*
- *pleural rub.*

In auscultation, it is necessary to first pay attention to the main breath sounds, determine the type of breathing, its intensity, and then proceed to the analysis of adventitious (additional) breath sounds.

5.2. Vesicular (alveolar) breathing

Vesicular breathing is normally heard above the entire surface of the lungs during inspiration and in the first third of the expiration. It is a result of the alveolar walls vibration at the time of inspiration when filling alveoli with air and alveoli emptying at the beginning of expiration. When a human person expires, these oscillations rapidly decay, as the tension of the alveolar walls decreases. Vesicular breathing is perceived as a soft, blowing noise, like the sound "F".

The strength of vesicular breathing depends on:

- the elastic properties of the lung tissue (the walls of the alveoli);
- a number of alveoli involved in breathing per unit volume;
- a rate of filling of the alveoli with the air;
- duration of inhalation and discharge;
- changes in the chest wall, pleural leaves and pleural cavity;
- patency of the bronchi.

In healthy lungs, physiological vesicular breathing as a rule is changed symmetrically in all places of auscultation.

In pathological conditions, vesicular breathing can change simultaneously in both lungs, in one of them or in a limited area of the lung.

Alterations of vesicular breathing*:*

- *quantitative - increasing and weakening breathing;*
- *qualitative - harsh, prolonged expiration, saccadic breathing.*

Physiological increase of vesicular breathing is observed in heavy physical load, and in children (*puerile breathing*) (Table 5-2).

Increased vesicular breathing is heard over healthy areas of the lungs, located next to the pathologically altered. The gain is determined over a healthy lung when you turn off from the breathing of the other half.

Physiological weakening of vesicular breathing is observed in obesity, and in well-developed chest muscles.

Pathological weakened vesicular breathing presents due to a loss of elasticity and alveoli wall destruction (in emphysema of the lungs), a decreased number of ventilating alveoli when they are filled with exudate (in focal pneumonia, initial and final stages of lobar pneumonia), alveoli collapse (compression atelectasis), filling the pleural cavity with liquid, air (hydrothorax, pneumothorax), in decreased depth of breathing due to the pain (fractures of ribs, intercostal neuralgia).

Table 5-2. Alterations of vesicular breathing

Alterations	Characteristics	Causes
Physiological weakening	sound volume reduction	thick chest wall (excessively developed muscles or obesity)
Physiological intensification	sound volume increase	thin chest wall, good elasticity of the alveoli, children *"puerile breathing"*, physical exercise, hyperthermia
Pathological weakening	sound volume reduction	diminished ventilation of alveoli (atrophy of alveoli, inflammation, degradation of elastic fibers, swelling, external compression of alveoli); bronchial obstruction of sound conduction
Abnormally increased	expiration becomes louder and longer	obstruction of small bronchi (inflammatory edema of the mucosa, bronchospasm, hypersecretion of viscid mucus)
Harsh (coarse) breathing	increase during both breathing phases	narrowing small bronchi and bronchioles (inflammatory edema of the mucosa, bronchospasm)
Interrupted (saccadic) breathing	inspiration is interrupted by short pauses	in a cold room, nervous trembling patients, or diseases of the respiratory muscles, traumas of the chest wall; locally – in pathology of small bronchi

Pathological increased vesicular breathing can be observed on the healthy side when the affected lung is switched off from breathing.

Increased vesicular breathing with prolonged expiration is heard in the unexpressed narrowing of the lumen of small bronchi, with edema of their mucosa or bronchospasm.

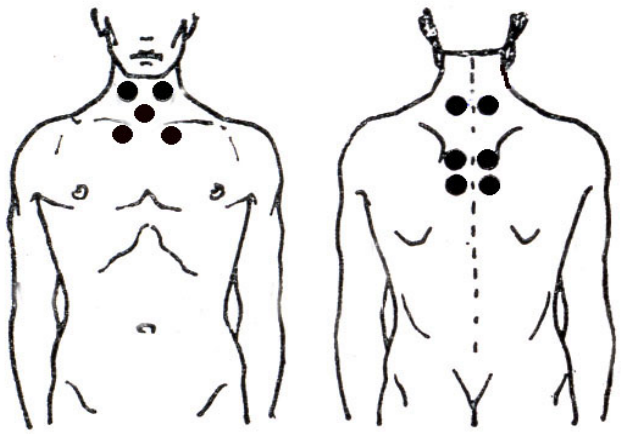
Harsh (coarse) breathing is observed with uneven narrowing of the bronchial lumen in bronchitis and focal pneumonia. In tone, it is a sharp and coarse, wheezing and with increased duration of expiration.

Another type of vesicular breathing is *saccadic breathing*. This is intermittent breathing (2-3 intermittent sounds on the inspiration), and the expiration is not changed. It occurs in healthy people with uneven contraction of the respiratory muscles (hypothermia, nervous tremor). With focal pulmonary tuberculosis, it can occur in a limited area of the lung due to the difficulty of passing air through the small bronchi and bronchioles and non-simultaneous expansion of the pulmonary tissue.

5.3. Bronchial (laryngotracheal) breathing

Physiological bronchial breathing

Breath sounds known as bronchial (laryngotracheal) breathing arise in the larynx and trachea when air passes through the vocal slits. In this case, turbulent air flows (swirls). This breathing is heard normally above the larynx and trachea on anterior surface of the neck and lower to the manubrium sterni and posteriorly from C7 to interscapular space at the level of III and IV thoracic vertebrae (Fig. 5-1, Table 5-1). Bronchial breathing is loud, and its expiration length is equal to expiration, and there is a characteristic pause between them its sound resembles the sound of the Russian letter "X". Normally, bronchial breathing is not carried on lung fields, as healthy lung tissue suppresses these fluctuations. If this breathing is carried out on the chest wall, it is called *pathological bronchial breathing*.



1 2

Fig. 5-1. Places of listening to bronchial breathing:
1) anterior side; 2) posterior side.

Pathological bronchial breathing

Depending the origin there are ***three types of pathological bronchial breathing:***

1) ***Infiltrative type*** arises in consolidation of a pulmonary tissue [(II and III stages (the “red and grey hepatization” stages) of lobar pneumonia, infarction of lungs, tuberculosis];

2*)* ***Atelectatic type*** - it is observed in compression atelectasis (if exudative pleurisy of 1.5-3 liters), and is auscultated at the vertebral column on a superior border of a dullness where there is a compressed lung; it rarely passes for *lin. axillaries anterior.* It presents at the *Garland triangle* (See Chapter 4. Fig. 4-10);

3) ***Cavitary type*** is auscultated above superficially posed smoothbore lumen of the big diameter connected with a bronchus (an abscess, a tubercular cavern, bronchiectasias with an appreciable distention of bronchi).

Amphoric breathing is a subtype of the pathological cavitary bronchial breathing. It occurs in the presence of a light smooth-walled air-containing cavity (lung abscess after opening, tuberculous cavity), communicating with the bronchus. It is heard in both phases of breathing and resembles a booming sound that occurs when air is blown into an empty vessel. This breathing occurs due to resonance phenomena in the pathological cavity. Note that for the occurrence of amphoric breathing, the diameter of the cavity should be at least 5 *cm.*

Metallic breathing is a subtype of the pathological cavitary bronchial breathing, which occurs if an open pneumothorax. It is very loud, high timbre and resembles a sound when struck by a metal. The same breath can be with large, smooth, surface-located cavities in the lungs.

Stenotic breathing is observed with narrowing of the larynx or trachea (tumor, foreign body in the larynx, laryngeal edema). It is heard in the place of narrowing, but can be heard without a stethoscope, at a distance from the patient (*stridor breathing*). This is a moaning breath with a sharply elongated inspiration. At the same time, it is shallow because of the small flow of the air into the lungs.

5.4. Adventitious breath sounds

Adventitious sounds are rhonchi, wheezes, coarse crackles, fine crackles, and pleural rub (Table 5-3).

5.4.1. Rhonchi and wheezes

Rhonchi and wheezes *are adventitious breath sounds formed in the bronchi. The late medical term for them is "dry rales" (the literal translation from Russian "сухие хрипы"). They are a continuous sound (≥250 msec), musical, prolonged, heard in the inspiration and the expiration, but not necessarily persisting throughout the respiratory cycle, like dashes in time.*

Rhonchi and wheezes result from uneven swelling of mucosa in bronchial inflammation and narrowing of the bronchial lumen, with vibrations of viscous sputum in the flowing air in the lumen of the bronchi.

Rhonchi *(late term - dry buzzing rales)* occur in the large and medium bronchi. Rhonchi are buzzing, low-pitched sounds with a snoring quality. The mechanism of their formation is similar to the appearance of harsh vesicular breathing. Their formation is due to the oscillation of sputum, located in the

bronchial lumen. They are heard on the background of harsh vesicular breathing (in non-obstructive bronchitis).

Table 5-3. Adventitious breath sounds and differences between them

Characteristics	Rhonchi	Wheezes	Coarse crackles	Fine crackles	Pleural rub
Late term	Dry buzzing rales	Dry whistling rales	Wet (moist) small-bubbling rales	Crepitation	Pleural rub friction murmur
Medical terms in Russian	Сухие басовые (жужжащие) хрипы	Сухие свистящие хрипы	Влажные мелкопузырчатые хрипы	Крепитация	Шум трения плевры
Place of formation	large and medium bronchi	Small bronchi	bronchi	alveoli	pleural membranes
Condition of formation	viscid secret in bronchial lumen, edema of bronchial mucosa, bronchospasm		liquid secret in bronchial lumen	liquid secret and air in al-veoli lumen	fibrin secret on surface of pleural membranes, and pleural adhesions
Sounds	continuous, buzzing, low-pitched with snoring quality	continuous, wheezing, high-pitched with hissing or shrill quality	somewhat louder, lower in pitch, brief crackles	soft, high-pitched, very brief crackles	crackles
Relation to breathing phases	at inspiration and expiration	at inspiration and expiration	at inspiration (better) and expiration	at inspiration	at inspiration and expiration
After coughing	change of the character	change of the character	change of the character	without change of the character	without change of the character
Acoustic characteristics	frequently variated sounds	frequently variated sounds	frequently variated sounds	uniform sounds	variated sounds
Under stethoscope pressing	no acoustic enhancement	no acoustic enhancement	no acoustic enhancement	no acoustic enhancement	acoustic enhancement

Wheezes *(late term - dry whistling rales)* occur in the small bronchi due to the increase in the air velocity when their lumen is narrowed. The increase in air friction leads to the appearance of an additional sound – wheezes. *They are continuous, wheezing, whistling sounds, high-pitched with a hissing or shrill quality*. *Wheezes* are heard against the background of vesicular breathing with an elongated expiration in COPD, obstructive bronchitis, bronchial asthma.

Intensity of wheezes can be different: with a slight narrowing of the bronchial lumen, they are heard in the form of a gentle quiet whistle, which is only at the end of inspiration and expiration. With a diffuse bronchial spasm, wheezes are heard at a distance from the patient (bronchial asthma).

Depending on the degree of inflammatory changes and the nature of the main process, *rhonchi and wheezes can be detected in a limited area of the lung (pneumonia) or diffusely over its entire surface (bronchitis).*

The number of rhonchi and wheezes can change dramatically (increase or decrease, or disappear) under the influence of cough, repeated deep breaths.

To identify the constancy of rhonchi and wheezes, ask the patient to cough after a deep breath. Rhonchi after coughing changes and may disappear due to the movement of sputum when coughing into the larger bronchus. Wheezes are more constant: when coughing, they may even increase due to an increase in air velocity during a coughing jolt and increased friction against the wall of the bronchus.

5.4.2. Crackles (coarse crackles, fine crackles, and pleural rub)

Crackles ***are discontinuous, intermittent, nonmusical, brief sounds, like dots in time. According to mechanisms of origin, basic types of crackles include coarse crackles, fine crackles, and pleural rub.***

Coarse crackles

Coarse crackles (*the late term – small-bubbling wet rales, the literal translation from Russian "влажные мелкопузырчатые хрипы"***)** occurs when air passes through the liquid sputum or other liquid secret, which accumulates in the lumen of small caliber bronchi. This creates sound like somewhat louder, lower in pitch, brief crackles (20–30 msec). The coarse crackles are better heard in the inspiration phase, because the speed of air movement in the bronchi will be greater. A cough changes coarse crackles. They can increase or disappear.

The coarse crackles present in bronchitis, and if the bronchi are surrounded by a dense tissue (in pneumosclerosis, focal pneumonia).

Russian-language medical literature uses also terms "влажные средне- и крупнопузырчатые хрипы" (the literal translation - *medium- and large bubbling wet rales),* and terms "звучные и незвучные влажные хрипы" *(the literal translation - consonating and non-consonating wet rales).*

Medium-bubbling wet rales occur in the medium caliber bronchi, and the *large-bubbling wet rales* occur in large bronchi and cavities, when air passes through the liquid in their lumen.

The consonating moist rales are very loud crackles if the bronchi are surrounded by a dense tissue (in pneumosclerosis, focal pneumonia). In addition, they can occur in cavities.

Non-consonating moist rales are heard worse, they are deaf and quiet, and resembles a rupture of the soup bubbles. The most often non-consonating rales are a direct sign of bronchitis.

Fine crackles

Fine crackles (*the late term - crepitation*) is the sound that occurs when a large number of alveoli are sticking out, it is heard only at inspiration. The condition for occurrence of the fine crackles is the presence of a small amount of exudate or other liquid in the alveoli lumen, and reducing amount of surfactant. In this case, at expiration alveoli walls stick together, and on inspiration - they stick out. The sounds of the fine crackles are similar to the coarse craclkes, but they are heard only on inspiration. A cough does not change the fine crackles (see Table 5-3).

There are the *inflammatory fine crackles* due to the accumulation of exudate in the alveoli [I and IV stages (the congestion and resolution stages) of lobar pneumonia, focal pneumonia]; and the *long-term fine crackles* due to the congested fluid in the lumen of the alveoli when the left ventricular heart failure; and the *short-time fine crackles* due to a reduction in a tone of the walls of the alveoli and even collapsing them. Short-time fine crackles happens in severe, weakened patients, as well as in the elderly. The peculiarity of this fine crackles that they are not stable, transient: after a few deep breaths alveoli wall are straightened, and fine crackles disappears.

The *loud constant fine crackles* are auscultated in focal pneumosclerosis (passed pneumonia, infiltrative tuberculosis).

Pleural rub

The ***pleural rub*** *(the late term - pleural rub friction murmur) is heard on the inspiration and expiration.* It is heard close to the ear, it does not disappear and does not change its location when coughing, and it increases when pressed by a phonendoscope on the chest. The pleural rub is heard when its membranes become roughened (in deposition of fibrin filaments in dry pleurisy, adhesions between pleural membranes, uremia) (see Table 5-3).

To differentiate it from the other crackles, the place of the appearance of pleural rub is listened to when imitating respiratory movements (the mouth is closed, and the nose is pressed with fingers, the patient retracts and protrudes the stomach). The pleural rub persists with the disappearance of other breath sounds.

Pleuropericardial rub (murmur) is the sound of pleura friction in close proximity to the heart (when involved in inflammation of the pleura directly adjacent to the heart) coincides with the respiratory movements and the heart contractions.

5.5. Bronchophony

***Bronchophony** is the auscultation of the whisper speech transfer to the surface of the chest.*

The sound from the larynx transfers on the chest wall by an air column of the trachea and bronchi through the layer of the alveoli.

To listen to the bronchophony, the patient whispers words containing sizzling sounds (*in Russian, for example, "чашка чая"*). In English, the numbers *"ninety-nine"* or *"sixty-six"* are traditionally mentioned (Table 5-4). However, the translation "ninety-nine", has fewer vowels and is less effective in evoking the phenomenon. Better phrases in English include "*toy boat*", "*Scooby Doo*", and "*blue balloons*".

To identify bronchophony, an examiner should listen to the entire surface of the chest by comparing with the sound on the symmetrical areas, as in the auscultation of the breath sounds. In a healthy person, the same quiet rustle is heard at symmetrical points. Normally, the sound of the patient's voice becomes less distinct as the auscultation moves peripherally from projection of the larynx and trachea.

Intensified bronchophony is the phenomenon of the patient's voice remaining loud at the periphery of the lungs or sounding louder than usual over a distinct. Intensified bronchophony (spoken words are heard) is observed in the consolidation of pulmonary tissue, which better conducts acoustic waves (in pneumonia), in the area of compression atelectasis and the cavity in the lung, communicating with bronchus.

Bronchophony reveals even small foci of consolidation of the pulmonary tissue. Bronchophony is already intensified, when other methods of investigation cannot reveal minimal changes (tactile fremitus is not amplified, vesicular breathing is little changed, pathological bronchial breathing and adventitious breath sounds are absent).

Bronchophony is weakened in the presence of fluid (hydrothorax, exudative pleurisy) or large amounts of air in pleural cavity (emphysema, pneumothorax).

Other tool used in auscultation includes listening for ***egophony.*** While listening to the lungs with a stethoscope, the patient is asked to pronounce the long-"E" vowel sound. Stethoscopic auscultation of a clear lung field during this articulation will detect a sound matching that received through normal hearing; that is, the sound articulated by the patient will be clearly transmitted through the lung field and heard unchanged by stethoscope. *When the lung field is consolidated, this finding is referred as the "E" to "A" transition."*

Table 5-4. Bronchophony and egophony

Technique	Patient says	Auscultation		Diagnostic value
		normal	abnormal	
Broncho-phony	whispered speech: in Russian *”чашка чая”,* or in English *"ni-nety-nine"* or *"six-ty-six"*	muffed, faint, in-distinct sounds	clear and distinct sounds	- consolidation of lung tissue (in pneumonia, tu-berculosis, pneu-mo-fibrosis, etc.); - bronchiectasis; - compressed atelectasis; - air cavity in pulmonary tissue
Egophony	*”eeeeeee”* loudly	clear sounds ”eeeeeee”	change sounds to ”aaaaaaa”	

5.6. The key points for the theme ''Auscultation of the Lungs''

Main breath sounds *are heard in normal and pathologic conditions, and include vesicular and bronchial breathing.*

Vesicular breathing is normally heard above the lungs during inspiration and in the first third of the expiration. It is a result of the alveolar walls vibration.

Abnormally increased harsh (coarse) vesicular breathing is auscultated in bronchitis and bronchial asthma (due to inflammatory edema of mucosa, bronchospasm, and viscid sputum in bronchial lumen). ***Pathological weakened vesicular breathing*** is auscultated in pneumonia, pulmonary emphysema and edema, compression atelectasis (due to diminished alveoli ventilation).

Physiological bronchial (laryngotracheal) breathing arises as air passes through the vocal slits, and heard over larynx, trachea, and tracheal bifurcation. ***Pathological bronchial breathing includes three types:*** 1) ***Infiltrative type*** arises in consolidation of a pulmonary tissue (II and III stages of lobar pneumonia, lung infarction, tuberculosis); 2) ***Atelectatic type*** is observed in compression atelectasis (by fluid in pleural cavity); 3) ***Cavitary type*** is auscultated above superficial smoothbore lumen air cavity connected with a bronchus (in pulmonary abscess, a tubercular cavern, bronchiectasis).

Adventitious (additional) breath sounds *are pathologic, and include* ***wheezes, rhonchi and coarse crackles*** *(formed in bronchi),* ***fine crackles*** *(formed in alveoli), and* ***pleural rub***.

Wheezes, rhonchi and coarse crackles are heard at inspiration and expiration. There are *w****heezes and rhonchi*** if viscid secret in bronchial lumen, and ***coarse crackles*** if liquid secret in bronchial lumen.

Rhonchi are formed in large and medium lumen bronchi - in non-obstructive bronchitis, and ***wheezes,*** formed in small bronchi - in COPD, obstructive bronchitis, bronchial asthma.

Coarse crackles occur if liquid secret in the lumen of small caliber bronchi in bronchitis and focal pneumonia/

Fine crackles are heard only on inspiration if liquid secret presents in alveoli (I and IV stages of lobar pneumonia, focal pneumonia, congested lungs, infiltrative tuberculosis), and in focal pneumosclerosis.

Pleural rub is heard close to the ear at inspiration and expiration when pleural membranes become roughened (in dry pleurisy, adhesions between pleural membranes, uremia).

Bronchophony is the auscultation of the whisper speech transfer to the surface of the chest. ***Pathological intensified bronchophony*** is heard in consolidation of the lung tissue (in pneumonia, tuberculosis, pneumofibrosis, etc.), bronchiectasis; compressed atelectasis; and the air cavity in the pulmonary tissue, communicating with bronchus. ***Weakened bronchophony*** is heard in the presence of fluid (hydrothorax, exudative pleurisy) or large amounts of air in the pleural cavity (emphysema, pneumothorax).

5.7. Assessment tests on the theme ''Auscultation of the Lungs''

1. Basic breath sounds include:
1. vesicular breathing;
2. bronchial breathing;
3. rhonchi;
4. fine crackles;
5. pleural rub.

2. Adventitious breath sounds include:
1. wheezes;
2. fine crackles;
3. pleural rub;
4. vesicular breathing;
-5. bronchial breathing.

3. Qualitative changes of vesicular breathing are:
1. harsh vesicular breathing;
2. vesicular breathing with prolonged expiration;
3. increased vesicular breathing;
4. weakened vesicular breathing;
5. bronchial breathing.

4. Inspiration is prolonged in:

1. narrowing of the larynx or the trachea lumen;
2. disorders of the medium lumen bronchi or stricture of bronchi;
3. bronchial obstruction;
4. cavity of the lung connected with a bronchus by fine-bored narrow opening;
5. increased air-filling of the pulmonary tissue (pulmonary emphysema).

5. The harsh (coarse) vesicular breathing is observed in:
1. bronchitis;
2. pneumonia;
3. accumulation of fluid in the pleural cavity;
4. accumulation of air in the pleural cavity;
5. increased air-filling of the pulmonary tissue.

6. Quantitative changes of vesicular breathing are:
1. increased vesicular breathing;
2. weakened vesicular breathing;
3. harsh (coarse) vesicular breathing;
4. vesicular breathing with prolonged expiration;
5. saccadic breathing.

7. Pathological bronchial breathing is observed in:
1. croupous (lobar) pneumonia;
2. compressive atelectasis of the lung;
3. cavity in the lung connected with bronchus;
4. large bronchiectasis;
5. bronchitis.

8. Bronchophony is intensified in:
1. pulmonary tissue infiltration;
2. compressive atelectasis of the lung;
3. cavity in the lung connected with bronchus;
4. large bronchiectasis;
5. bronchitis.

9. Bronchophony is not determined in:
1. accumulation of air in the pleural cavity (pneumothorax);
2. accumulation of fluid in the pleural cavity (exudative pleurisy);
3. increased air filling of the lung (emphysema);
4. pneumosclerosis;
5. obturator atelectasis of the lung lobe.

10. An important auscultative sign of smooth-wall air cavity in the lung is:

1. "amphoric" bronchial breathing;
2. weakened vesicular breathing;
3. rhonchi;
4. coarse crackles;
5. fine crackles.

11. The basic auscultative sign in diagnostics of the dry pleurisy is:

1. pleural rub;
2. bronchial breathing;
3. fine crackles;
4. coarse crackles;
5. rhonchi.

12. It is typically of the pleural friction rub:

1. it is auscultated in time of inspiration and expiration;
2. it does not variate after coughing;
3. it is better auscultated at imitation of the breathing;
4. it is increased by pressing a stethoscope and at inclination of a trunk forward;
5. it is auscultated only in time of inspiration.

13. Causes of the coarse crackles include:

1. accumulation of fluid sputum in lumen of small caliber bronchi;
2. bronchospasm;
3. inflammatory swelling bronchial mucosa;
4. development of pulmonary fibrosis;
5. exudate in alveoli.

14. Causes of appearance of wheezes are:

1. spasm of fine bronchi;
2. swelling of bronchial mucosa in inflammation;
3. accumulation of viscid sputum in lumen of bronchi;
4. exudate in alveoli;
5. development of fibrosis in pulmonary tissue.

15. It is typically of the fine crackles:

1. it is auscultated in time of inspiration and expiration;
2. it does not variated after coughing;
3. it is better auscultated at imitation of breathing;
4. it is increased by pressing of a stethoscope and at inclination of a trunk forward;

5. it is auscultated just in the time of inspiration.

16. Vesicular breathing is auscultated at the ventilation of:

1. larynx;
2. trachea;
3. bronchi;
4. respiratory tubes;
5. alveoli.

17. Bronchial breathing is auscultated at the ventilation of:

1. larynx;
2. trachea;
3. main bronchi;
4. respiratory tubes;
5. alveoli.

18. Vesicular breathing with prolonged expiration presents in:

1. narrowing of the larynx or the trachea lumen;
2. disorders of the medium bronchi patency;
3. spasm or stricture of the bronchi;
4. cavity of the lung connected with the bronchus by fine-bored narrow opening;
5. increased air-filling of the pulmonary tissue (pulmonary emphysema).

19. Vesicular breathing is auscultated in norm:

1. on anterior surface of the neck;
2. on parasternal lines from supraclavicular spaces to III-IV interspaces;
3. symmetrically on the chest surface excluding area of the heart, large bones, projection of trachea and main bronchi;
4. during inspiration;
5. during whole expiration;
6. during first third of the expiration.

20. Bronchial breathing is auscultated in the norm:

1. on anterior surface of the neck;
2. on parasternal lines from supraclavicular spaces to III-IV interspaces;
3. symmetrically on the chest surface excluding area of the heart, large bones, projection of trachea and main bronchi;
4. during inspiration;
5. during whole expiration;

6. during first third of the expiration.

21. Characteristics of rhonchi and wheezes are:
1. buzzing or wheezing sounds;
2. crackles sound;
3. at inspiration and expiration;
4. at inspiration alone;
5. changes after coughing.

22. Characteristics of the coarse crackles are:
1. buzzing or wheezing sounds;
2. crackles sound;
3. at inspiration and expiration;
4. at inspiration alone;
5. changes after coughing.

23. Characteristics of fine crackles are:
1. buzzing or wheezing sounds;
2. crackles sound;
3. at inspiration and expiration;
4. at inspiration alone;
5. changes after coughing.

Chapter 6. Laboratory-Instrumental Examination of the Respiratory System

Goals: to enable students to learn –
1) diagnostic value of the instrumental methods of the respiratory examination (spirography, peak flow metry, X-ray, and other chest imaging methods);
2) diagnostic value of the laboratory study of sputum, and the pleural fluid.

6.1. X-ray (roentgenography) and other chest imaging methods

The X-ray (roentgenography) method, or chest radiography, is often the initial diagnostic study performed to evaluate patients with respiratory symptoms, but it can also provide the initial evidence of the disease in patients who are free of symptoms. X-ray can find one or more nodules or masses in the lung when chest radiography is performed for a reason other than the evaluation of the respiratory symptoms.

A number of diagnostic possibilities are often suggested by the radiographic pattern. *A localized region of opacification involving the pulmonary parenchyma can be described as a nodule (usually <6 cm in the*

diameter), a mass (usually >6 cm in the diameter), or an infiltrate. A diffuse disease with increased opacification is usually characterized as having an alveolar, an interstitial, or a nodular pattern. In contrast, increased radiolucency can be localized, as seen with a cyst or bulla, or generalized, as occurs with pulmonary emphysema. The chest radiography is also particularly useful for the detection of a pleural disease, especially when the air or the fluid accumulate in the pleural cavity. An abnormal appearance of the hila and/or the mediastinum can suggest a mass or enlargement of the lymph nodes.

Other chest imaging methods

Bronchography is an additional method of the x-ray examination of the trachea and the bronchi by contrasting them for diagnosis of bronchiectasis and malignant tumors.

Computed tomography (CT) is indicated for the diagnosis of a small pleural effusion, and in suspicion of tumors of the lung, mediastinum, pleura, and to determine the exact location and spread of inflammatory changes in the chest in the complicated diagnostic cases.

Radionuclide scanning (scintigraphy) and angiography of the pulmonary circulation are indicated for evaluation of the pulmonary embolism.

Ultrasound is helpful in the detection of pleural effusion and is often used as a guide to the placement of a needle for sampling of pleural liquid (i.e., for thoracocentesis).

Bronchoscopy is the process of a direct visualization of the tracheobronchial tree by a flexible fiberoptic bronchoscope. Bronchoscopy is able to identify an endobronchial pathology, including tumors, granulomas, bronchitis, foreign bodies, and the sites of bleeding. The samples from the airway lesions can be taken by several methods, including washing, brushing, and biopsy.

6.2. Examination of the lung ventilation (spirography)

Spirography (spirometry) is a method of recording the lung volumes during breathing maneuvers in time. The aim of the study is to diagnose the type and degree of pulmonary ventilation disorders based on the analysis of the quantitative and qualitative changes in spirographic indicators.

Static volumes and capacities (capacity includes several volumes) characterize the elastic properties of the lungs and the chest.

Dynamic indicators, which are recorded in quiet breathing and in forced expiration, reflect mainly the state of the respiratory tract (bronchi).

The values obtained in the study are compared with the proper values (for the age of 25-70 years). Proper values depend on the age, height, gender of the patient. The criterion of pathological changes in ventilation indicators is the deviation of the data obtained by the spirometry from the proper values, expressed as a percentage.

The type of respiratory dysfunction (respiratory failure) depends on the list of the reduced indicators.

Basic static volumes and capacities of spirometry

When spirometry in quiet breathing mode, the respiratory volume (RV), respiration rate (RR) are recorded, and the minute volume of respiration (MV) of rest is calculated (Fig. 6-1).

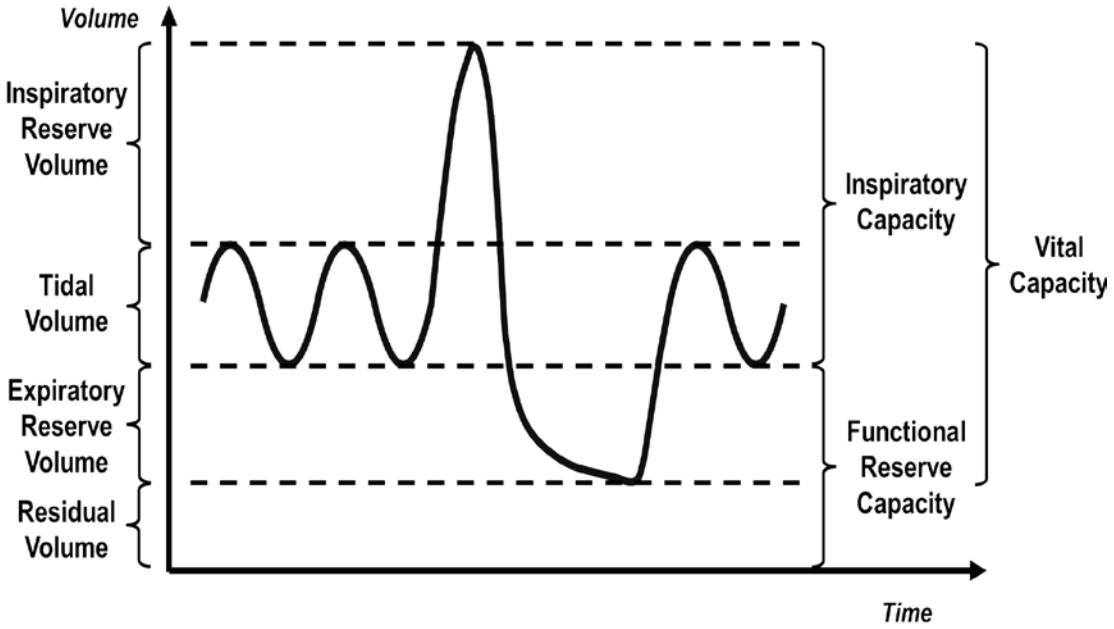


Fig. 6-1. Respiratory volumes

These indicators characterize the intensity of ventilation at the time of the study and do not characterize the condition of the lungs. Their changes are the same in healthy people and patients with a lung disease.

Minute volume of respiration (MV, in Russian – минутный объем вентиляции, МВ) is the total ventilation during quiet breathing per minute (6-8 liters).

Respiratory volume (RV, in Russian – дыхательный объем, ДО) – the volume of air entering the lungs for one quiet breath (500 – 800 ml).

Inspiratory reserve volume (IRV, in Russian – резервный объем вдоха, РОвд) – the maximum volume that can be inhaled after a quiet inspiration.

Expiratory reserve volume (ERV, in Russian – резервный объем выдоха, РОвыд) – the maximum volume of the air that can be exhaled after a quiet expiration.

Vital capacity (VC, in Russian – жизненная ёмкость легких, ЖЕЛ) – the maximum volume of air that can be exhaled after maximum deep inspiration (VC=RV+IRV+ERV), 3.7-4.0 liters on average.

Dynamic ventilation tests

These tests help study lung ventilation and its reserves, which are important when heavy work is done, or there are respiratory diseases. The study of this mechanics is necessary for determining the changes in the inspiration to

to expiration ratio, the respiratory efforts at various respiratory phases, and other indices. To assess the ventilation capacity of the lungs, tests are used to identify the maximum volume and speed of breathing.

Forced expiratory vital capacity (FEVC, or FVC, in Russian – форсированная жизненная ёмкость легких, ФЖЕЛ) - the maximum volume of the air that can be exhaled during forced exhalation after maximum inhalation (Fig. 6-2). *In healthy persons, forced expiratory vital capacity (FEVC) is not less than 80% of the proper FEVC predicted on the basis of age, sex, and height. In obstructive respiratory diseases (COPD, bronchial asthma), the FVC is usually lower than VC.*

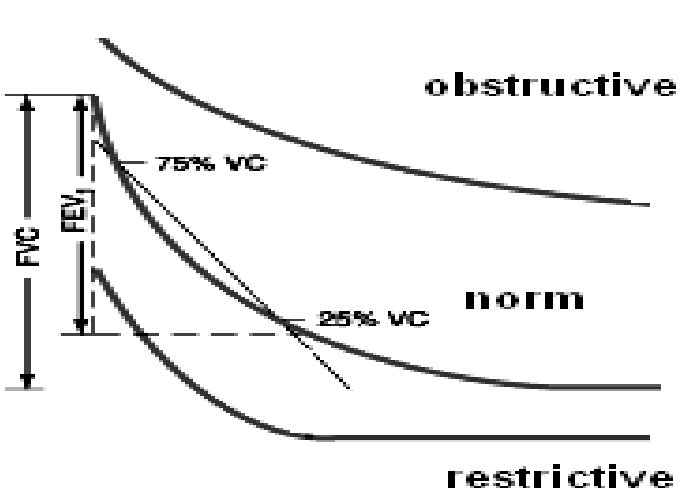


Fig. 6-2. The curves of forced vital capacity (FVC) in the norm and in obstructive and restrictive respiratory failure.

Forced expiratory volume for the first second of expiration (FEV1, or FVC1, in Russian – объем форсированного выдоха за первую секунду, ОФВ1) is easily measured and accurately predicted on the basis of age, sex, and height.

Tiffeneau index (in Russian – индекс Тиффно) - the ratio FEV1/VC (or FEV1/FVC), expressed as a percentage. The ratio of FEV1 to forced vital capacity (FEV1/FVC, or index of Tiffeneau) normally exceeds 0.75 (75%). *In obstructive lung diseases, the expiratory flow is slow, and FVC, FEV1, and Tiffeneau index are lower*.

Maximum ventilation of the lungs (MVL, in Russian – максимальная вентиляция легких, МВЛ) – the maximum amount of the air that the patient can ventilate for 1 minute. The value of MVL is determined with the maximum deep and frequent breathing for 12 seconds. Then recalculate the resulting volume for 1 minute. This indicator depends on the patency of the bronchi and the training of the respiratory muscles. It can be used as an additional indicator of the

severity of bronchial obstruction. It is more often used in assessing the state of the lung ventilation capacity and fitness in athletes.

Pneumotachymetry and peak flow metry are the techniques of recording the flow (volumetric velocity) of the air at quiet breathing and forced exhalation ("flow-volume" curve). Modern spirometers allow to produce spirometry and pneumotachometry and to determine a number of indicators of the lung ventilation.

Forced expiratory flow (FEF, or MEF; in Russian – максимальная объёмная скорость, МОС) is the flow (or speed) of the air coming out at forced expiration. It can be given at discrete times, generally defined by what fraction remains of the forced vital capacity (FVC). The usual intervals are 25%, 50% and 75% (FEF25, FEF50 and FEF75), or 25% and 50% of FVC.

Forced expiratory flow at 25 and 75% of the pulmonary volume (FEF25-75%) is defined as the mean forced expiratory flow during the middle half of the FVC and measures average flow rates on an FVC segment that includes flow from medium-to-small airways. Average ranges of FEF25-75% in the healthy population depend mainly on sex and age. FEF25-75% varies significantly in healthy subjects and is considered abnormal when it reaches values <65% of predicted. These measurements are very sensitive tests for obstructive disorders of ventilation, such as in bronchial asthma, chronic obstructive pulmonary disease.

The peak expiratory flow (PEF, or peak expiratory flow rate, PEFR) is a is the maximal rate that a person can exhale during a short maximal expiratory effort after a full inspiration. A peak flow meter (a small, hand-held device used to monitor a person's ability to breathe out air) evaluates PEF. It measures the airflow through the bronchi and thus the degree of obstruction in the airways if the peak expiratory flow (PEF) is less than 80% of a predicted value according the sex and the age of the patient.

Variants of disorders in ventilation function of the lungs (respiratory failure)

There are three types of the respiratory failure

- ***obstructive type*** (impaired patency of the respiratory tract),
- ***restrictive type*** (insufficient expansion of the lung parenchyma),
- ***mixed type***.

In assessing the spirogram, the determination of the type of the respiratory failure begins with an assessment of the decrease in FEV1.

Obstructive type of respiratory failure is characterized by the difficulty of exhalation: decrease in FVC, FEV1, decrease in the index of Tiffeneau, and little changed VC (in obstructive bronchitis, chronic obstructive pulmonary disease, bronchial asthma) (see Fig. 6-2).

The airflow obstruction is usually determined by forced expiratory spirometry - the recording of exhaled volume against the time during a maximal expiration. Normally, a full forced expiration takes between 3 and 4 sec, but

when the airflow is obstructed, it takes up to 15 or even 20 sec and may be limited by breath-holding time. The ratio of FEV1 to forced vital capacity (FEV1/FVC, or *index of Tiffeneau*) normally exceeds 0.75 (75%), in bronchial obstruction FEV1/FVC <0.7 (70%) (Table 6-1).

Table 6-1. Functional indexes of external respiration

Index	Norm	Types of disordered lung ventilation		
		obstructive	restrictive	mixed
Vital capacity (VC, % of proper VC)	≥ 80%	≥ 80%	<80%	<80%
Forced expiratory vital capacity (FEVC, % of proper FEVC)	≥ 80%	<80%	≥ 80%	<80%
Tiffeneau index (FEV_1/ FEVC)	>75%	<70%	>75%	<70%

Inhalation test with Salbutamol (*sympathomimetic bronchodilato*r) allows evaluating the reversibility of the bronchial obstruction. The scheme of the inhalation test consists of an initial study of the function of external respiration /FVC, FEV1/ with the construction of the flow-volume forced expiratory curve. After 30 minutes after the inhalation of bronchodilating medication, the FVC is re-examined and the patient's spirogram is evaluated. Its results allow us to highlight bronchial obstruction:

- *reversible obstruction*: increase in FEV1 ≥12% from the initial value;
- *partially reversible obstruction*: increase in FEV1 by 6-11% of the initial value;
- *irreversible obstruction*: increase of FEV1 ≤5% of the initial value.

In patients with bronchial asthma it is a reversible obstruction, inhalation of salbutamol improves expiratory airflow quickly after the inhalation. Patients with chronic obstructive pulmonary disease (COPD) react to sympathomimetic inhalation weaker.

Restrictive type of respiratory failure is observed in restriction of the pulmonary tissue to expansion and reduction (in pneumosclerosis, pneumofibrosis, pleural adhesions, kyphoscoliosis), decrease in the respiratory surface of the lungs (in pneumonia, exudative pleurisy, hydrothorax, pneumothorax). A restrictive variant of the respiratory failure is characterized by a proportional decrease in most lung volumes - RV, IRV, ERV, VC, and normal FEV1 (see Fig. 6-2). Tiffeneau index is 70% or more of the norm due to a significant reduction of the VC.

The mixed type of respiratory failure combines the features of both previous types.

6.3. Laboratory examination of sputum

Sputum is a pathological secret expectorated by coughing from the airways. Sputum is collected for study after a thorough rinse of oral cavity in cup of Petri in the morning (before meals). The laboratory examination of sputum includes physical, chemical, microscopic, bacterioscopic, and if necessary, bacteriological and cytological studies.

Character of sputum is determined by its composition: mucus, serous fluid, fibrin, pus or blood. Depending on the predominance of one or another of the components of the sputum, its character may be *mucous, serous, purulent and hemorrhagic (bloody); or combined - mucopurulent, muco-purulent-hemorrhagic, sero-purulent, sero-purulent- hemorrhagic.*

Color of sputum depends on the nature of its constituent elements and inhaled particles that can stain sputum. The yellowish-greenish color of sputum is due to the content of pus in it; red, rusty or brownish - an admixture of blood or products of its degradation. Black or gray colour is given sputum by coal and dust, white - flour dust.

Amount of sputum. A small amount of sputum is released in the inflammation of the respiratory tract (in laryngitis, tracheitis, initial stage of acute bronchitis, bronchial asthma outside the attack, bronchopneumonia).

Abundant sputum (from 0.3 to 1 l) is usually released from the cavities in the pulmonary tissue and bronchi (in bronchiectasis, lung abscess), with leakage in the bronchi of a large amount of blood plasma (in pulmonary edema).

Consistency of sputum depends on the composition of sputum. Mucus and fibrin are dominated in viscous sputum, in the liquid sputum - serous exudate, in semi-liquid sputum - serous exudate mixed with mucus or pus.

Chemical reaction of sputum is usually alkaline. Chemical reaction of the sputum becomes acid in the presence of gastric juice. This helps differentiate between bloody coughing and hematemesis (bloody vomiting with aspiration of gastric contents in airways and subsequent cough).

Character of sputum may be (Table 6-2):

- *mucoid sputum* - colourless, liquid; blood-stained may be (in acute bronchitis; lung infarction, pulmonary congestion);
- *serous sputum* - colourless, liquid, and foamy; blood-stained may be (in pulmonary edema);
- *mucopurulent sputum* - yellow or greenish and tenacious, streaks of blood may be (in acute and chronic bronchitis, tbc, bronchectasis, focal pneumonia);
- *purulent sputum* - semiliquid, greenish-yellow; streaks of blood, two layers and foul odour may be (in the opened lung abscess);
- *bloody sputum* contains red liquid blood (if pulmonary hemorrhage in tbc, cancer, etc.);
- *rusty sputum* - semiliquid, red-brown dark (in the lobar pneumonia);

- *three (two)-layer sputum* - sputum stratification is observed when it is isolated from the large cavities in the lungs (in abscess, gangrene, large bronchiectasis). When standing, sputum is divided into two layers: the lower (denser) - consists of pus, cell and tissue detritus; the upper layer - of serous fluid. Sometimes on the surface of the liquid layer, a third foam-layer is revealed.

Table 6-2. Types of sputum

Type of sputum	General properties, colour, and consistency of the sputum	Cause
Mucoid	colourless, liquid; blood-stained may be	acute bronchitis, lung's infarction, pulmonary congestion
Serous	colourless, liquid, and foamy; blood-stained may be	pulmonary edema
Muco-purulent	yellow or greenish and tenacious; streaks of blood may be	chronic bronchitis, tbc, bronchiectasis, etc.
Purulent	Semiliquid, greenish-yellow; streaks of blood, two layers and foul smell may be	abscess of lungs
Bloody sputum	Sputum contains red liquid blood	pulmonary hemorrhage (tbc, cancer, etc.)
Rusty	Semiliquid, red-brown dark	lobar pneumonia
Three-layer sputum	The upper layer is mucopurulent, the middle - serous, and the lower is pus; foul smell presents	chronic purulent processes, gangrene

Components of the sputum visible by unaided air:

- *Curschmann spirals* white twisted corkscrew spirals (in bronchial asthma); - small dense twisted threads (in bronchial asthma);

- *fibrin clots* - whitish and reddish branching elastic formations (in fibrinous bronchitis, pneumonia);

- *Dittrich's plugs* - whitish-yellow lamps of soft consistency the size of a pinhead and a sharp stinking smell (in gangrene, lung abscess) and consist of tissue degradation products bacteria and fatty acid crystals, having offensive odour on pressing (in gangrene, chronic abscess, chronic bronchitis);

- *lentils of Koch* (“rice corpuscles:) - greenish-yellow corpuscles curd consistency, small size, consisting of cellular detritus, Mycobacteria tuberculosis and elastic fibers (in cavernous pulmonary tuberculosis);

- *lime grains* - lime (calcium oxide) (if decomposition of old tuberculosis foci);

- *Actinomycete druses* - yellow formations resembling coarse flour; pieces of necrotizing tissue, food remains (in pulmonary actinomycosis, decomposition of cancer);

- *diphtheritic films of pharynx and nasopharynx* - grayish scraps, consisting of fibrin and necrotic cells.

Microscopic study of sputum

Microscopic examination of the sputum is performed in fresh unpainted (native) and in fixed stained preparations. *Elements of the sputum are detected in the native preparation: cells, fibers, crystals.*

Knowledge of the appearance and quality of the sputum specimen obtained is especially important when one is interested in Gram's staining and culture. Because sputum consists mainly of secretions from the tracheobronchial tree rather than the upper airway, the finding of alveolar macrophages and other inflammatory cells is consistent with a lower respiratory tract origin of the sample, whereas the presence of squamous epithelial cells in a "sputum" sample indicates contamination by secretions from the upper airways.

Besides processing for routine bacterial pathogens by Gram's staining and culture, sputum can be processed for a variety of other pathogens, including staining and culture for mycobacteria or fungi, culture for viruses, and staining for *P. carinii*.

Diagnostic value of microscopic elements of the sputum:

(1) Cellular elements:

- *leucocytes* - in native preparations at large magnification (in inflammatory, especially purulent processes);

- *eosinophils* - in the native preparation by their uniform large lustrous grains; or by staining (in bronchial asthma, allergy process);

- *erythrocytes* – in decomposition of the lung tissue, pneumonia, lung congestion, lung infarction;

- *epithelium* - columnar ciliated epithelium in any sputum, if large amounts - in bronchitis, bronchial asthma, etc.;

- *alveolar macrophages* are large cells of round shape with the presence of dark brown inclusions in the cytoplasm. These cells are found in various inflammatory processes of the bronchi and the lungs (in bronchitis, pneumonia, pneumoconiosis);

- *siderophages, or "cells of the heart defects"*, are alveolar macrophages containing hemosiderin in their cytoplasm in the form of golden-yellow inclusions. These cells present in pulmonary circulation congestion (often in mitral stenosis), and pulmonary infarction.

- *malignant tumour cells* - large and disfigured cells with large (several) nuclei;

- *Candida albicans* - yeast-like cells and branching mycelium in native preparation (in prolonged antibiotic therapy, candidosis);

- *Actinomycetes* - It separated from small yellow compact grains (drusen), Gr-stained (in pulmonary actinomycosis, decomposition of cancer);

- *bacteria* - acid-resisiting mycobacterium (in tuberculosis), Gram-positive and Gram-negativebacteria (in bronchitis, pneumonia); anaerobe (in abscess, gangrene); *Pneumocystis carinii* (AIDS or other serious immunodeficiency state).

(2) Fibres

- *Curschmann spirals* - compacted, twisted into a spiral mucous formation, consisting of an axial filament surrounded by a mass of spirally coiled thin fibrous formations (in bronchial asthma, COPD);

- *elastic fibres* - fine formations of two dichotomically branching filaments of the uniform thickness (in decomposition of the lung tissue in tuberculosis, cancer and abscess);

(3) Crystals

- *Charcot-Leyden crystals* - colourless octahedric of various size crystals (decomposition of eosinophils in bronchial asthma, old sputum);

- *crystals of hematoidin* - rhombic or needle-shaped brown-yellow formation (in pulmonary hemorrhage).

6.4. Study of the pleural fluid

***Pleurocentesis* (*thoracocentesis, pleural puncture*)** is a puncture of the chest wall for extraction of *pleural fluid* (Fig. 6-3).

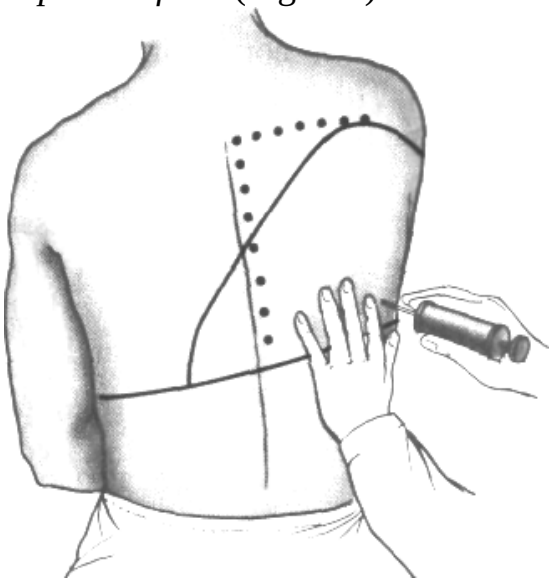


Fig. 6-3. Pleurocentesis

Pleurocentesis is used: (1) to take samples of the pleural fluid for diagnostic studies, (2) to remove fluids from the pleural cavity, and, whenever

necessary, to administer medicinal preparations. The chest is punctured as rule on the posterior axillary or scapular line at the point of maximum dullness, usually at the VII or VIII intercostal space). The puncture is made at the upper edge performed on the superior edge of underlying rib to prevent an injury of intercostals nerves and blood vessels.

Macroscopic examination determines the character, color, and transparency of fluid. *By character, pleural fluids are divided into two large groups –* ***transudates and exudates***.

Transudates (non-inflammatory fluids) are formed with an increase in venous pressure (right ventricular heart failure), a decrease in oncotic pressure in the blood vessels (diseases with hypoproteinemia: nephrotic syndrome, severe liver damage, cachexia), a violation of electrolyte metabolism, mainly an increase in sodium concentration (hemodynamic heart failure, nephrotic syndrome), an increase in aldosterone production and some other conditions. Transudate is characterized by clear and slightly opalescent, liquid, pale yellow appearance. Microscopy study detects many mesothelium cells, less blood cell. Transudate is not inflammatory fluid, it is typical to heart failure, and liver cirrhosis. Transudates used for microbiological studies are as a rule sterile but they can be infected during repeated paracenteses.

Exudates (inflammatory fluids) are serous and serous-fibrinous (with exudative pleurisy of tuberculosis etiology, rheumatic pleurisy), hemorrhagic (most often with malignant neoplasms and traumatic lesions of the pleura, less often with lung infarction, acute pancreatitis, hemorrhagic diathesis, tuberculosis), hilous (with difficulty lymph flow through the thoracic duct due to compression of the tumor, increased lymph nodes, as well as the rupture caused by injury or tumor), putrid (upon accession of putrefactive flora).

Colour and transparency of the pleural fluid depends on their nature. Transudates and serous exudates have a light yellow color, transparent; other types of exudates in most cases are cloudy, of different colors.

Types of exudate:

Serous exudate is characterized by clear and slightly opalescent, liquid, yellow – golden yellow appearance. Microscopy study detects abundance of leucocytes. It may be in various etiology of pleurisy.

Purulent exudate is thick, cloudy, yellowish-green liquid (in empyema of the pleura). Microscopy - rich in neutrophils, and bacteria (Gram stain). It is typical of suppurative infection of the pleural cavity (empyema).

Putrid exudate is grayish-green, cloudy, with a very unpleasant putrid smell. It is observed in the lung gangrene with a breakthrough in the pleural cavity or the addition of putrid anaerobic microflora in pleurisy.

Hemorrhagic exudate is a cloudy liquid of brownish-brown or red color Microscopy study detects abundance of red blood cell; malignant cells and *Mycobacteria tuberculosis* may be. Hemorrhagic effusion is typical of

tuberculosis and tumors of pleura, hence it may be in various etiology of pleurisy.

Chylous fluid is a milky turbid liquid with a large amount of fat, which readily settles and forms the top layer of cream-looking (in lymphostasis, traumas of the major lymphatic vessels and the flow of their contents into the pleural cavity). Microscopy study shows fat drops (or cell detritus).

Cholesterol exudate is a thick opalescent liquid with a yellowish-coffee tint, flakes of cholesterol crystals. Opacity disappears in the presence of ether. This effusion may be due to fatty degeneration of cells (in old encapsulated pleurisy).

Physicochemical studies of the pleural fluid can differentiate exudates and transudates (Table 6-3).

Table 6-3. Differentiations between exudate and transudate

Characteristics	Exudate	Transudate
Light's criteria (≥ 1 of the following 3):		
LDH (lactate dehydrogenase)	> 2/3 ULN *	<2/3 ULN *
Pleural fluid/serum ratio LDH	≥0,6	<0,6
Pleural fluid/serum protein ratio	≥0,5	<0,5
Additional criteria:		
Specific gravity	>1,018 kg/l	<1,015 kg/l
Protein, g/l	>30	<20
Rivalta's test	positive	negative
Glucose	<3,33 mmol/l	>3,33 mmol/l
Pleural fluid/serum cholesterol ratio count	>0,3	<0,3
Erythrocyte (RBC) count	variable	< 1000/mL
Leucocyte (WBC) count	> 1000/mL	< 1000/mL
Leucocyte (in area of vision)	up to 15	>15

*Note: *- ULN = upper limit of normal serum LDH [200 U/l (1,6 g/l)]*

The ***Light's criteria*** are the international standard for recognizing differences between the pleural exudate and transudate (R.W. Light, 1972). When Light criteria are used, the *protein and LDH of the blood* should be measured as close as possible in time with the pleural fluid taken during thoracentesis.

Relative density of the cavity fluids is determined using a urometer. Transudates have a relative density than exudates. Relative density of transudate ranges from 1005 to 1015; relative density of exudates typically above 1015.

Protein content in the transudate is less than 3% (30 g/l), in the exudate - more than 3% (30 g/l). The difference in the amount of protein can be used to distinguish transudate from exudate.

Rivalta's test is based on the sedimentation of seromucin (a substance of globulins nature) by a weak solution of acetic acid. Seromucin presents in the pleural fluid of an inflammatory nature and is not in the transudate. Rivalta's test is negative in transudate, positive - in exudate.

Microscopy of pleural fluid

Erythrocytes are often found in the pleural fluid due to traumatic blood impurity at the time of puncture. Their number is small (up to 10 in the field of view). Hemorrhagic exudates contain a very large number of erythrocytes, they cover the entire field of view of the microscope (in lung tumor).

Leukocytes in a small amount (up to 15 - 20 in the field of view) are found in transudates and in large quantities (cover the entire field of view of the microscope) - in exudates (in purulent exudate). The qualitative composition of leukocytes (the ratio of their individual species) is studied in stained preparations.

Mesothelial cells are recognized by a large size (up to 25-50 mcm). They are found in transudates of cardiac and renal origin, in exudates of the tumor nature (in a small amount).

Tumor cells have a pronounced polymorphism of size and shape.

Bacterioscopic and bacteriological examination of the pleural fluid is similar to the study of sputum. *Mycobacterium tuberculosis* in bacterioscopic examination of exudate is detected very rarely. It is a more efficient culture of the pleural effusion.

6.5. The key points on the theme ''Laboratory-Instrumental Examination of the Respiratory System''

Diagnostic value of chest radiography (X-ray). A localized opacification (shadow) in the lungs can be described as a nodule (usually <6 cm in diameter), a mass (usually >6 cm in diameter), or an infiltrate. This reflects the decrease or absence of the air in the pulmonary parenchyma (pneumonia, tuberculosis, tumors, undisclosed lung abscess, etc.).

On the contrary, an increased airiness may be localized, as seen in the lung cyst or bull, or generalized, as occurs in emphysema of the lungs. The chest radiography is also particularly useful for the detection of a pleural disease, especially when the air or the fluid accumulate in the pleural cavity.

Diagnostic value of spirography (spirometry). The aim of the study is to diagnose the type and degree of pulmonary ventilation disorders.

Obstructive type of respiratory failure is characterized by difficulty of exhalation: decrease in FVC, FEV1, and in the index of Tiffeneau, and little changed VC (in obstructive bronchitis, emphysema, COPD, bronchial asthma).

Restrictive variant of respiratory failure is characterized by a decrease in VC, and normal FEV1. It is observed in restriction of pulmonary tissue to expansion and reduction (in pneumosclerosis, atelectasis, pleural adhesions, kyphoscoliosis), decrease in the respiratory surface of the lungs (in pneumonia, exudative pleurisy, hydrothorax, pneumothorax). *A mixed type of respiratory failure* combines the features of both previous types.

Diagnostic value of sputum examination. By character, sputum may be *mucoid sputum* (in acute bronchitis; lung infarction, pulmonary congestion); *serous sputum* (in pulmonary edema); *mucopurulent sputum* (in acute and chronic bronchitis, bronchiectasis, focal pneumonia); *purulent sputum* (in opened lung abscess); *bloody (hemorrhagic) sputum* (if pulmonary hemorrhage in tbc, cancer, etc.); *rusty sputum* (in lobar pneumonia); *three (two)-layer sputum* (in pulmonary abscess, gangrene, large bronchiectasis). In bronchial asthma, sputum contains *eosinophils and Charcot-Leyden crystals. Curschmann spirals* is a mass of spirally coiled thin fibrous formations (in bronchial asthma, chronic bronchitis). *Elastic fibres* are typical in decomposition of the lung tissue in tuberculosis, cancer and abscess. Sputum contains *bacteria* - acid-resisting mycobacterium (in tuberculosis), Gram-positive and Gram-negative bacteria (in bronchitis, pneumonia); anaerobe (in abscess, gangrene).

Diagnostic value of pleural fluid examination. By character, *pleural fluids are divided into two large groups – transudates and exudates.* ***Transudates (non-inflammatory fluids)*** are formed in a right ventricular heart failure, diseases with hypoproteinemia (nephrotic syndrome, severe liver damage, cachexia), a violation of electrolyte metabolism, mainly an increase in sodium concentration. **Exudates are** *inflammatory fluids in exudative pleurisy of variable etiology* (bacterial, tuberculosis, viral, rheumatic, traumatic, malignant pleurisy; with lung infarction, acute pancreatitis, hemorrhagic diathesis, and others). Pleural exudates are differentiated from transudates by international *Light's criteria* (based on ratio of protein and LDH in pleural effusion compared to blood serum levels), and additional criteria such as physical (relative density of exudates typically above 1015) and chemical tests (protein content in in the exudate - more than 3%; positive Rivalta's test).

6.6. Assessment tests on the theme ''Laboratory-Instrumental Examination of the Respiratory System''

1. Sputum analysis includes the examination of:

1. physical characteristics;
2. chemical characteristics;
3. macroscopic characteristics by an unaided eye;

4. microscopy study;
5. bacterioscopy.

2. What is true for mucoid sputum?
1. colourless;
2. red or pink;
3. liquid;
4. characteristic in acute bronchitis;
5. characteristic in pneumonia.

3. **What is true for purulent sputum?**
1. yellow or greenish;
2. red or pink;
3. liquid;
4. semiliquid, tenacious;
5. characteristic in lung abscess;
6. characteristic in acute bronchitis.

4. **What is true for hemorrhagic sputum?**
1. yellow or greenish;
2. red;
3. liquid or semiliquid;
4. viscid;
5. characteristic in tuberculosis and cancer;
6. characteristic in pneumonia.

5. **What is typical for microscopy of sputum in bronchial asthma?**
1. erythrocytes;
2. eosinophils;
3. Curschmann spirals;
4. Dittrich's plugs;
5. elastic fibres;
6. Charcot-Leyden crystals.

6. **What is typical for microscopy of sputum in pulmonary abscess and gangrene?**
1. purulent sputum;
2. three-layer sputum;
3. Curschmann spirals;
4. Dittrich's plugs;
5. elastic fibres.

7. Thoracocentesis is used:

1. to take samples of the pleural fluid for diagnostic studies;
2. to remove fluid from the pleural cavity;
3. If necessary, to administer medicinal preparations into the pleural cavity;
4. to evacuate pericardium fluid;
5. to take samples of ascitic fluid.

8. **Pleural fluid study includes:**
1. organoleptic analysis;
2. macroscopic analysis;
3. physicochemical analysis;
4. microscopic analysis;
5. microbiological analysis.

9. Specific tests of transudate:
1. relative density below 1015;
2. protein less than 3%;
3. negative Rivalta's test ;
4. relative density more than 1015;
5. protein more than 3%.

10. Specific tests of exudate:
1. relative density more than 1015;
2. protein more than 3%;
3. negative Rivalta's test;
4. relative density below 1015;
5. protein less than 3%.

11.Serous exudate is characterized by:
1. clear and liquid;
2. cloudy and semiliquid;
3. yellow – golden colour;
4. microscopy study detects abundance of leucocytes;
5. microscopy study detects abundance of eosinophils.

12. Purulent exudate is characterized by:
1. clear and liquid;
2. grayish or greenish-yellow;
3. yellow – golden colour;
4. microscopy study detects rich in neutrophils, and bacteria (Gram- stain);
5. microscopy study detects abundance of eosinophils.

13. Hemorrhagic exudate is characterized by:
1. pink to dark red;

2. grayish or greenish-yellow;
3. yellow – golden colour;
4. microscopy study detects of red blood cells;
5. malignant cells and Micobacteria tuberculosis may be.

14. The patient has made maximal inspiration and expiration. What volumes are in hi/her expired air?
1. inspiratory reserve volume (IRV);
2. expiratory reserve volume (ERV);
3. respiratory volume (RV);
4. vital capacity (VC);
5. residual air volume (RAV).

15. Index of Tiffeneau is:
1. ratio of forced expiratory vital capacity for 1-th second FEVC1 and forced expiratory vital capacity (FEVC);
2. ratio of forced expiratory vital capacity for 1-th second ($FEVC_1$) and maximum-lung ventilation (MLV);
3. ratio of forced expiratory vital capacity (FEVC) and vital capacity (VC);
4. ratio of forced expiratory vital capacity for 1-th second (FEVC1) and vital capacity (VC);
5. ratio of vital capacity (VC) and maximum-lung ventilation (MLV).

16. What parameter of spirogram is variated in a respiratory failure of the obstructive type?
1. forced expiratory vital capacity for 1-th second (FEVC1);
2. residual air volume (RAV);
3. total lung capacity (TLC);
4. maximum lung ventilation (MLV);
5. vital capacity (VC).

17. What parameter of spirogram is variated in a respiratory failure of the restrictive type?
1. total lung capacity (TLC);
2. maximum lung ventilation (MLV);
3. vital capacity (VC);
4. residual air volume (RAV);
5. forced expiratory vital capacity for 1-th second (FEVC1).

18. Vital capacity consists of such volumes of the lungs as:
1. inspiratory reserve volume (IRV);
2. expiratory reserve volume (ERV);
3. respiratory volume (RV);

4. residual air volume (RAV);
5. forced expiratory vital capacity for 1-th second (FEVC1).

19. What parameter of spirogram is variated in a respiratory failure of the mixed type?
1. forced expiratory vital capacity for 1-th second (FEVC1);
2. forced expiratory vital capacity (FEVC);
3. total lung capacity (TLC);
4. maximum lung ventilation (MLV);
5. vital capacity (VC).

20. Decrease of vital capacity (VC) is typical in:
1. respiratory failure of restrictive type;
2. respiratory failure of obstructive type;
3. lobar pneumonia;
4. pulmonary emphysema;
5. atelectasis.

21. Decrease of forced expiratory vital capacity (FEVC**) is typical in:**
1. respiratory failure of restrictive type;
2. respiratory failure of obstructive type;
3. lobar pneumonia;
4. pulmonary emphysema;
5. bronchial asthma.

22. Decrease of Index of Tiffeneau is typical in:
1. respiratory failure of restrictive type;
2. respiratory failure of obstructive type;
3. chronic bronchitis;
4. pulmonary emphysema;
5. bronchial asthma.

23. Reversible bronchial obstruction characteristics by inhalation Salbutamol test:
1. increase of FEV1 ≥12% from the initial value;
2. increase of FEV1 by > 6-11% from the initial value;
3. increase of FEV1 ≤5% from the initial value;
4. increase of VC> 7% from the initial value;
5. increase of MVL> 11% from the initial value.

24. Reversible bronchial obstruction is characteristic of:
1. obstructive bronchitis
2. bronchial asthma

3. pulmonary emphysema
4. pneumonia
5. pleurisy

25. **The Light's criteria for recognizing differences between the pleural exudate and transudate include:**
1. the exudate LDH (lactate dehydrogenase) > 2/3 upper limit of the normal serum LDH;
2. pleural fluid/serum ratio LDH ≥0,6 in case of an exudate;
3. pleural fluid/serum protein ratio ≥0,5 in case of an exudate;
4. pleural fluid/serum protein ratio <0,5 in case of an exudate;
5. pleural fluid/serum ratio LDH <0,6 in case of an exudate.

Chapter 7. Basic Clinical Syndromes of the Respiratory System Diseases

Goals: to enable students to learn:
clinical symptoms and signs, laboratory and instrumental diagnosis, and diagnostic value of basic clinical syndromes of respiratory system diseases.

7.1. Syndromes of the pulmonary tissue pathology

7.1.1. Syndrome of focal consolidation of the pulmonary tissue

Causes: Consolidation of the pulmonary tissue occurs during the inflammatory process in the lungs (in pneumonia), impregnation of the alveoli with blood (in pulmonary embolism), replacement of the alveolar tissue with connective tissue elements (pneumosclerosis) (Table 7-1).

Clinical signs of pulmonary tissue consolidation syndrome are due to the shutdown of the lung area from breathing. The severity of the symptoms depends on the volume of the non-functioning lung tissue.

Complaints: mixed dyspnea increasing under physical load.

General survey: diffuse cyanosis.

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

Dynamic chest survey: lagging of the affected half of the chest while breathing.

Palpation of the chest: decreased elasticity, increased tactile fremitus on the affected half of the chest.

Percussion of the lungs: a dull sound over the affected part of the lung.

Auscultation of the lungs: weakening of the vesicular breathing or replacing it with the bronchial breathing over the affected area of the lung, the fine crackles and/or coarse crackles in a limited area over the affected lung. Bronchophony intensifies over the focus of consolidation.

Chest X-ray: shadows of different sizes depending on the volume of the affected area of the lung.

Table 7-1. Syndromes of pulmonary tissue pathology

Syndrome	Normal condition	Focal consolidation of pulmonary tissue	Air cavity in pulmonary tissue	Increased airiness of the lungs (emphysema)
Causes	-	pneumonia, lung's infarction, tumour, pneumosclerosis	abscess, tuberculosis cavern, degradation of the lung tumour	pulmonary emphysema
Complaints	absent	dyspnea	cough with putrid sputum	expiratory dyspnea
Survey of chest	Symmetric	lagging of the affected side during breathing	unilateral thoracic lagging	barrel-like chest, additional respiratory muscles force
Palpation of chest	fremitus, rigidity – moderate	increased tactile fremitus	increased tactile fremitus	increased tactile fremitus, decreased rigidity
Percussion	resonant sound	dull sound	tympanic or metallic sound	hyperresonant sound, decreased respiratory mobility of lung borders
Main breath sounds	vesicular breathing	bronchial breathing, decreased vesicular breathing	bronchial (amphoric) breathing	decreased vesicular respiration with prolonged expiration
Adventitious sounds	absent	coarse and/or fine crackles	coarse crackles may occur	wheezes may occur
Bronchophony	absent	increased	increased	decreased
X-ray	without changes	focal shadows (nodule) or infiltrate	air cavity with a horizontal liquid level	increased translucency of a lung pattern

7.1.2. Syndrome of cavity in the lung

Causes: This syndrome may be after opening an acute abscess, chronic abscess, tuberculosis cavern, large bronchiectasis, destruction of tumor. In the lung, there is an air-containing cavity, communicating with the bronchus.

Complaints: cough with the separation of a large amount of sputum (200-400 ml or more per day) with purulent or mucopurulent character often with an admixture of blood. Cough depends on the position of the patient – the cough increases, and a large amount of sputum is expectorated at a certain position of the patient (on a healthy side, often with a lowered head – "postural pose").

Survey and palpation of the chest: the lag of the "sick" half of the chest in the act of breathing; tactile fremitus is increased.

Percussion: a tympanic percussion sound. If the cavity of the regular shape, has smooth walls and the surface – a metallic sound is located.

Auscultation: decreased vesicular breathing, a pathologic bronchial breathing; the coarse crackles (medium- and large- bubbling consonating wet rales) above limited part of the lung. Bronchophony is enhanced.

X-ray examination of the lungs: in abscess, a cavity (enlightenment) is often found with a horizontal level of fluid, and in tuberculous cavern, a ring – shaped shadow.

7.1.3. Syndrome of increased airiness (emphysema) of the lungs

Emphysema is defined anatomically as a permanent and destructive enlargement of airspaces distal to the terminal bronchioles without obvious fibrosis and with a loss of normal architecture. Due to the loss of elasticity of alveolar tissue, the over distended alveoli contain an increased amount of air.

Causes. The most common causes of emphysema of the lungs are obstructive bronchitis, recurrent pneumonia, long-term bronchial asthma, occupational lung diseases, smoking, alpha-1 antitrypsin deficiency, etc.

Pulmonary emphysema occurs in mechanical overdistension of the lungs (in musicians playing woodwind and brass instruments) or in heavy physical load associated with retention of breath. Advanced age is another predisposing factor.

Complaints. The patient mainly complains of dyspnea, which at the onset of the disease may only develop during exercise but later it occurs at rest.

Survey and palpation of chest. Dyspnea is usually expiratory: a healthy person expires air whereas the patient with emphysema presses it out from the chest with an physical effort. The intrathoracic pressure increases during expiration and the neck veins therefore become swollen. In long-term disease, the chest becomes barrel-shaped. Supraclavicular fossae are usually leveled or protruded over the clavicles. The tissues under the clavicles may protrude as well. During inspiration, the rigid chest seems to rise due to the contraction of the accessory muscles. Tactile fremitus is diminished.

Percussion. Hyperresonant sound can be heard on percussion. Topographic percussion demonstrates lowering and restriction of active respiratory mobility of inferior borders, and elevation of superior borders of the lungs. Kroenig's areas are expanded.

Auscultation. Diminished vesicular breathing is heard on auscultation (the sounds are especially weakened in grave cases). In the presence of concurrent bronchitis, diffuse rhonchi and wheezes are heard.

The *X-ray picture* of the lungs is especially translucent. Inferior borders of the lungs are lowered. Restricted respiratory mobility of the diaphragm presents. The X-ray picture of the lungs has increased bronchovascular pattern or/and increased translucency of lung.

Spirography shows an accentuated decrease in FEVC, FEV1, and Tiffeneau index.

7.1.4. Syndromes of obturator and compressive atelectasis

Atelectasis is a shrunken, airless condition affecting all or part of the lung. According to a pathogenic mechanism, *atelectasis may be due to obturation of bronchi and compression*.

In *obturator (syn. obstructive, or occlusive) atelectasis*, the main cause of acute or chronic atelectasis is intraluminal bronchial obstruction, often due to the plugs of tenacious bronchial exudates, endobronchial tumors, granulomas, or foreign bodies, and bronchial strictures.

Compressive atelectasis may be due to external bronchial compression by enlarged lymph nodes, tumor, or aneurysm; external pulmonary compression by pleural fluid or gas (eg, due to the pleural effusion or pneumothorax), and surfactant deficiency (Table 7-2).

Atelectasis may be acute or chronic. In chronic atelectasis, the affected area is often composed of a complex mixture of airlessness, infection, bronchiectasis, destruction, and fibrosis.

Clinical picture of atelectasis. Symptoms and signs depend on how rapidly the bronchus is occluded, how much of the lung is affected, and whether infection is present. Rapid occlusion with a massive collapse, particularly with infection, causes pain on the affected side, sudden onset of dyspnea and cyanosis, a drop in BP, tachycardia, elevated temperature, and sometimes shock. Chest wall excursion at the affected side is reduced or absent. Chest percussion reveals a dull sound, and limitation of the respiratory mobility of an inferior border at the affected part of the lung.

Gradual development of atelectasis may occur without visible signs. Physical and radiological data depend on the type of atelectasis.

In ***obturator atelectasis,*** survey of the chest reveals a decrease of the affected side, unilateral thoracic lagging during respiration, and retraction of the ribs at the affected side of the chest. Tactile fremitus is weakened. Auscultation detects decrease or absence of vesicular breathing and bronchophony. The X-ray of the lungs in obturator atelectasis: shadow and absence of a pulmonary pattern

in the affected part of the lung, decreased volume of the lung, and shift of mediastinum in the affected side.

Table 7-2. Syndromes of obturator (obstructive) and compressive atelectasis

Syndrome	Obturator (obstructive) atelectasis	Compressive atelectasis
Causes	plugs of tenacious sputum, endobronchial tumors, scars, foreign bodies	enlarged lymph nodes, tumour, aorta aneurysm; pleural effusion or pneumothorax
Complaints	dyspnea, cough, and pain may occur	dyspnea
Survey of chest	decreased affected side of the chest, unilateral thoracic lagging during respiration, retraction of ribs	asymmetric chest, increased affected side of the chest, unilateral thoracic lagging during respiration may occur
Palpation	decreased tactile fremitus	increased tactile fremitus
Percussion	dull sound, decreased respiratory mobility of the lung borders	dull sound, decreased respiratory mobility of the lung borders
Main breath sounds	decreased vesicular breathing	pathologic bronchial breathing
Adventitious sounds and bronchophony	decreased bronchophony	increased bronchophony, fine crackles
X-ray	elevated diaphragm; deviated mediastinum to the affected lung	shadow of the collapsed part of the lung toward the root, deviated mediastinum to the non-affected lung

In ***compressive atelectasis*** survey of chest may reveals an increased volume and flatness of intercostal spaces at the affected side of the chest, unilateral thoracic lagging during respiration. Tactile fremitus is increased. Auscultation over the compressive atelectasis area detects bronchial breathing. If air presents in partly compressed alveoli, fine crackles may occur. Bronchophony may be increased. X-ray of the lungs in compression atelectasis: shadow in projection of the collapsed lung, absence of a pulmonary pattern in the lateral parts of the

chest – in pneumothorax, pleural fluid – in exudative pleurisy, and shift of mediastinum to the non-affected side.

Diagnosis of atelectasis is usually made from clinical findings plus x-ray evidence of a diminished lung size (indicated by the retracted ribs; elevated diaphragm; deviated trachea, heart, and mediastinum to the affected side; and overdistention of the unaffected lung) and of a solid, airless area. If only a segment is affected, the shadow is triangular, with its apex toward the hilum. When small areas are affected, surrounding tissue distention causes them to appear curiously discoid, particularly in subsegmental lower lobe atelectasis. An entire lobe may be affected (lobar atelectasis). As the lobe loses air, the interlobar fissures become displaced, and the lobe becomes more densely opacified as the bronchi, blood vessels, and lymphatics are crowded together. Exact X-ray findings depend which lobe is affected and how other structures compensate for the volume loss. Posterior-anterior and lateral views aid in the diagnosis.

The *middle lobe syndrome* is usually recognized by the characterisitic x-ray findings: on posterior-anterior view, subtle obliteration of the right lateral heart border and, on the lateral view, the triangular or rectangular shadow running from the posterior cardiac border to the anterior chest wall.

The cause of an obstruction should always be found regardless of the patient's age. With a fibrobronchoscopy, lobar as well as segmental and subsegmental divisions can be seen. Chest CT can help clarify the mechanism of collapse; an experienced interpreter can distinguish among the causes of atelectasis: endobronchial obstruction, compression due to intrapleural fluid or air, and scars resulting from chronic inflammation.

7.2. Syndromes of pleural cavity pathology

7.2.1. Syndrome of thickening pleural membranes

Syndrome of thickening pleural membranes is the pathology of the respiratory system characterized by fibrinous exudation without an effusion, resulting in adhesion between the opposing surfaces of the pleural membranes, and increase of their thickness.

Causes of this syndrome are dry pleurisy due to infections (tuberculosis, bacterial infection, fungus, viral infection); dissemination of the tumor cells to pleura; reactive pleurisies in uremia (severe renal failure); and it may be in dehydration (profuse bleeding, vomiting, diarrhea) (Table 7-3).

Complaints: mixed dyspnea increasing with physical load, pain in the affected half of the chest, increasing with deep breath, dry cough.

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

Static chest examination: there may be a reduction in the affected half of the chest.

Dynamic chest examination: lagging of the affected half of the chest while breathing.

Table 7-3. Syndromes of pleural cavity pathology

Syndrome	Thickening of pleural membranes	Fluid in the pleural cavity	Air accumulation in the pleural cavity
Causes	dry pleurisy, pleural adhesions	exudative pleurisy, hydrothorax	pneumothorax
Complaints	chest pain at inspiration and cough, dyspnea	dyspnea	chest pain at inspiration and cough, dyspnea
Survey of chest	lagging of the affected side of chest, superficial breathing, forced position on the affected side	asymmetric chest, enlargement of affected side, unilateral thoracic lagging during breathing	asymmetric chest, enlargement of affected side, unilateral thoracic lagging during breathing
Palpation	local palpatory tenderness, decreased fremitus	decrease or absence of fremitus; increased rigity of chest	decreased or absence of fremitus, increased rigity of chest
Percussion	hyporesonant or dull sound maybe, decreased respiratory mobility of the lung border	stony dull (flat) sound, decreased respiratory mobility of the lung border	tympanic sound, decreased respiratory mobility of the lung border
Main breath sounds	decreased vesicular breathing	decrease or absence of vesicular breathing	decrease or absence of vesicular breathing
Adventitious sounds and bronchophony	pleural rub, decreased bronchophony	decrease or absence of bronchophony, pleural rub	decrease or absence bronchophony
X-ray	limited mobility of the diaphragm	homogenous shadow in the area of the fluid, deviated mediastinum to the non-affected lung	light pulmonary field without pulmonary pattern; a shadow of the collapsed lung toward the root

Palpation of chest: decreased elasticity, painfulness, tactile fremitus is weakened on the affected half of the chest.

Percussion of lungs: a hyporesonant or dull sound over the affected area.

Auscultation of lungs: weakening vesicular breathing, pleural rub in a limited area. Bronchophony is weakened over the affected area.

X-ray of lungs: restriction of the diaphragm movement on the affected side.

7.2.2. Syndrome of fluid accumulation in the pleural cavity

Causes: It is observed in exudative pleurisy, hydrothorax.

Complaints: dyspnea and a feeling of heaviness in the chest on the affected side.

Survey and palpation of the chest: the affected half of the chest is enlarged and lags in the act of respiration. There are smooth or even protruded intercostal spaces of the lower part of the chest in case of a large accumulation of the pleural fluid. Tactile fremitus is sharply weakened or not detected.

Percussion: a stony dull (flat) sound above the pleural fluid. In exudative pleurisy, it is possible to determine the upper limit of dullness in the form of an oblique line (*Damoiseau's curve*) with the highest level on the posterior axillary line and the lowest level – on the paravertebral line (See Chapter 4. Fig. 4-10).

Auscultation: vesicular breathing and bronchophony are sharply weakened or absent.

X-ray examination of the lungs: shadow in the area of fluid accumulation with a clear upper border, which in exudative pleurisy coincides with the Damoiseau's curve, and in hydrothorax it is more horizontal. In a large accumulation of fluid, the mediastinal organs shift to the "healthy" side.

7.2.3. Syndrome of air accumulation in the pleural cavity

Syndrome of air accumulation in the pleural cavity (pneumothorax) is defined as the presence of air or gas in the pleural cavity (i.e., in the space between visceral and parietal pleura of the lung), which can impair oxygenation and/or ventilation.

Causes: a rupture of air cavity in the lung tissue (abscess or tuberculosis cavern) resulting in communication between the pleural cavity and the bronchi, and traumas of the chest. Pneumothorax may be in many types of underlying lung diseases, including chronic obstructive pulmonary disease (COPD), emphysema of the lungs, cystic fibrosis and pneumonia**.** The damaged lung tissue is more likely to collapse.

A severe type of pneumothorax can occur in people who need mechanical assistance to breathe. The artificial ventilation can create an imbalance of air pressure within the chest. The lung may collapse completely.

Clinical picture. The main symptoms of a pneumothorax are a sudden chest pain and dyspnea.

Survey and palpation of the chest: the affected half of the chest is enlarged, and lags in respiration; smooth intercostal spaces. Tactile fremitus is decreased or absent.

Percussion: tympanic sound.

Auscultation: vesicular breathing and bronchophony are weakened, or are not conducted.

X-ray of lungs: above air pleural cavity – a transparent field without a pulmonary pattern, and closer to the mediastinum – a shadow of the collapsed lung.

7.3. Syndrome of bronchial obstruction

Syndrome of bronchial obstruction is a set of the symptoms related to the reduced maximum expiratory flow during forced expiration. Bronchial obstruction may be chronic and persistent (in emphysema) or episodic (in bronchial asthma), and recurrent (chronic bronchitis) (Table 7-4).

Table 7-4. Syndrome of bronchial obstruction

Chest examination	Bronchial obstruction
Cause	bronchitis, bronchial asthma, pulmonary emphysema
Complaints	expiratory dyspnea, cough
Survey of chest	barrel-like (emphysematous) chest, and accessory respiratory muscles force may occur
Palpation	decreased tactile fremitus
Percussion	a hyperresonant sound, decreased respiratory mobility of the lung borders may occur
Main breath sounds	decreased harsh vesicular breathing, prolonged expiration
Adventitious sounds, bronchophony	wheezes, decreased bronchophony
X-ray	an increased bronchovascular pattern and/or an increased translucency of the lung pattern
Spirography	a decrease in FEVC, FEV1 and the Tiffeneau index

Air flow limitation and increased airways resistance may be caused by:

- the loss of the elastic recoil driving passive expiration due to the emphysema,
- an increased collapsibility of small airways through the loss of a radial traction on the airways,

- increased resistance due to the intrinsic narrowing of small airways (because of edema and inflammation of mucous membrane, bronchospasm, and viscous sputum).

Clinical picture. The patient mainly complains of expiratory dyspnea and cough with viscid sputum. Inspection reveals barrel (emphysematous) chest in chronic bronchial obstruction, and accessory respiratory muscles force strain may occur. Chest palpation detects decreased tactile fremitus, and increased rigidity of chest may be. Percussion may detect a hyperresonant sound (in case of emphysema) and a restricted respiratory mobility of the inferior border of the lungs.

Auscultation of lungs: a harsh vesicular breathing with extended expiration, diffuse wheezes. Bronchophony is weakened (in case of emphysema).

X-ray of the lungs: an increased translucency of the pulmonary fields (in case of emphysema) and/or an increased bronchovascular pattern.

Spirography shows a significant decrease in FEVC, FEV1 and the Tiffeneau index*;* VC may be normal or slightly decreased.

7.4. Syndrome of respiratory insufficiency

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

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

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

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

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

Classification of Respiratory Insufficiency

1. Etiology:

(a) Primary - due to the respiratory diseases;
(b) Secondary - due to other pathology (chest trauma, kyphoscoliosis, intoxications, etc.).

2. Course - (a) Acute, (b) Chronic.
3. Type of external respiration dysfunction:

(a) restrictive, (b) obstructive, (c) mixed.

4. Degree of respiratory insufficiency:

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

5. Stage:

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

Clinical and pathogenetic forms of respiratory failure:

1. Obstructive respiratory failure.

2. Restrictive respiratory failure.

3. Mixed respiratory failure.

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

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

Mixed respiratory failure is most often the result of a combination of several of the above forms (Table 7-5).

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

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

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

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

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

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

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

Clinical picture may include such syndromes as focal consolidation of the pulmonary tissue, atelectasis, thickening of pleural membranes, accumulation of the air and fluid in the pleural cavity. Inspection of the patient shows the asymmetric chest, a decrease of the respiratory mobility of the chest, inspiratory or

mixed dyspnea. Diminished vesicular breathing or pathologic bronchial respiration, fine crackles, pleural rub may be heard.

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

Table 7-5. Types of respiratory failure

Characteristics	Restrictive type	Obstructive type	Mixed (combined) type
Cause	lobar pneumonia, pneumosclerosis, pleurisy, pneumothorax, atelectasis, pathologic chest	chronic obstructive pulmonary disease (bronchitis, emphysema), bronchial asthma	combination of restrictive and obstructive types causes
Pathology mechanism	limited ability of the lungs to expand and to collapse	difficult passage of air through bronchi	combination of the two previous mechanisms
Survey	asymmetric chest, decreased respiratory mobility, inspiratory or mixed dyspnea	barrel-like chest, involvement of accessory muscles, expiratory dyspnea	combination of the signs of two previous types
Breath sounds	decreased vesicular breathing and/or pathologic bronchial breathing; fine crackles, pleural rub may be	harsh vesicular breathing with prolonged expiration; wheezes	combination of the signs of two previous types
Spirogram	*VC<80% of proper VC, ** FEV1 > 75% of ***FVC	VC - decreased insignificantly, FEV1 < 70% of FVC	VC<80% of proper VC, FEV1 < 70% of FVC

Note: *VC – vital capacity; **FEV1 – forced expiratory volume of the first second; FVC - forced vital capacity.

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

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

Three degrees of respiratory insufficiency are also distinguished (Table 7-6). The degrees of respiratory insufficiency reflect the gravity of the disease at a given moment.

I degree – dyspnea when effort is available earlier, cyanosis is absent, light hypoxemia presents. In arterial blood HbO_2 (oxyhemoglobin) – 80–96%; PaO_2 (partial pressure of oxygen) – 100–70 mm Hg; $PaCO_2$ (partial pressure of carbon dioxide) – 40–50 mm Hg. VC is normal or ≥ 70 % of the predicted VC. MVL ≥ 60% of the predicted MVL.

II degree - dyspnea at usual load, obvious cyanosis, moderate hypoxemia. In arterial blood HbO_2 - 80–60%; PaO_2 – 70–50 mm Hg; $PaCO_2$ – 50 mm Hg. VC is ≥ 50 % of the predicted VC. MVL ≥ 40% of the predicted MVL.

III degree - dyspnea at rest, pronounced cyanosis, severe hypoxemia. In arterial blood HbO_2 < 60%; PaO_2 < 50 mm Hg; $PaCO_2$ > 70 mm Hg. VC is < 50 % of the predicted VC. MVL < 40 % of the predicted MVL.

Table 7-6. Degrees of respiratory insufficiency

Characteristics	I degree	II degree	III degree
Dyspnea	at moderate or significant physical load	during light exercise	at rest
Cyanosis	absent	during light exercise	persistent
[1] VC (% of proper VC)	≥70	≥50	<50
[2] MVL (% of proper MVL)	≥60	≥40	<40
[3] HbO_2 (%)	93–98	86–92	<85
[4] PaO_2 mm Hg	100–85	75–85	<75
[5] $PaCO_2$ mm Hg	40–50	50-70	>70

Note: [1] - vital capacity; [2] – maximal ventilation of the lungs; [3] - oxyhemoglobin, [4] – partial pressure of oxygen, [5] - partial pressure of carbon dioxide of arterial blood.

7.5. The key points on the theme "Basic Clinical Syndromes of Respiratory System Diseases"

Syndrome of focal consolidation of the pulmonary tissue. The main diseases are pneumonia, tuberculosis, lung cancer. Complaints depend on the underlying disease: wet cough prevails; hemoptysis, mixed dyspnea, and fever may occur. Objectively: a dull percussion sound, weakened vesicular and pathological bronchial breathing, fine crackles. Chest X-ray - shadows (nodes, infiltrates) in the lungs.

Syndrome of cavity in the lung. The main diseases are abscess, cavernous tuberculosis, destruction of tumor. Complaints - dry cough (closed cavity), cough with purulent sputum (opened cavity), hemoptysis. Objectively: a dull percussion sound (cavity with pus), a tympanic sound (cavity with air), pathological bronchial breathing may occur. Chest X-ray - cavity in the lung.

Atelectasis syndrome. ***Obturator (obstructive) atelectasis*** is associated with the closure of the bronchial lumen (tumor, foreign body). ***Compressive atelectasis*** is associated with compression of the lung from the outside (exudative pleurisy, pneumothorax). Clinical picture – dyspnea, cough. Objectively – a dull percussion sound, pathological bronchial breathing, absence of vesicular respiration. Chest x-ray: obturator atelectasis – infiltration of the lung area, mediastinum displacement to the affected side; compression atelectasis – reduction of the lung in size, mediastinum displacement to the healthy side.

Syndrome of the increased airiness of the lung (emphysema). The main disease is COPD. Complaints - constant expiratory dyspnea. Objectively: barrel-like chest, a hyperresonant percussion sound, weakened vesicular breathing. Chest X-ray - increased transparency of the pulmonary tissue.

Syndrome of fluid accumulation in the pleural cavity. The main diseases are exudative pleurisy, hydrothorax. Complaints - dyspnea. Objectively: asymmetry of the chest and the lag of the affected side in respiration, a stony dull percussion sound above fluid, vesicular breathing is weakened or absent. Chest X-ray - a homogeneous shadow that displaces the mediastinum in the healthy side.

Syndrome of air accumulation in the pleural cavity (pneumothorax). The main diseases are emphysema, tuberculosis, chest trauma. Complaints – dyspnea, chest pain; hemoptysis may occur (in traumatic pneumothorax). Objective data – a tympanic sound over the air, absence of vesicular breathing. Chest X-ray: absence of a pulmonary pattern in the area of air accumulation, lung collapse.

Syndrome of pleural membrane thickening. The main disease – dry pleurisy. Complaints: pain in the affected half of the chest, increasing with deep breathing, dry cough. Objective data - pleural rub in a limited area.

Syndrome of bronchial obstruction. The main diseases are COPD, bronchial asthma. Complaints - expiratory dyspnea, attacks of suffocation, paroxysmal cough. Objectively: a hyperresonant sound at percussion, hoarse vesicular

breathing with prolonged expiration, wheezes. Spirography – reduction of FVC, FEV1.

Syndrome of respiratory insufficiency. Causes – acute and chronic respiratory system diseases. Clinical picture – dyspnea of various types up to suffocation, central or diffuse cyanosis, tachycardia, clubbing nails (with a long course). Objective data correspond to the disease that caused a respiratory failure. Pulse oximetry – reduction of oxygen saturation below 95%. Spirography – reduction of VC, FVC, FEV1 (according to the type of respiratory failure).

7.6. Assessment tests on the theme "Basic Clinical Syndromes of Respiratory System Diseases"

1. Syndrome of restrictive type of respiratory failure presents in:

1. exudative pleurisy;
2. focal pneumonia;
3. lobar pneumonia;
4. chronic bronchitis;
5. bronchial asthma.

2. Syndrome of obstructive type of respiratory failure presents in:

1. chronic obstructive pulmonary disease (COPD);
2. bronchial asthma;
3. pleurisy with effusion ;
4. focal pneumonia;
5. lobar pneumonia.

3. Clinical signs of bronchial obstruction syndrome:

1. expiratory dyspnea;
2. forced sitting position with support on hands;
3. vesicular respiration with prolonged expiration;
4. diffuse wheezing rales;
5. inspiratory dyspnea.

4. Syndrome of increased airiness of the lungs is characteristic in:

1. emphysema of lungs;
2. pneumothorax;
3. cavity in lung connected to a bronchus;
4. lobar pneumonia;
5. focal pneumonia.

5. An auscultative sign connected with the syndrome of focal consolidation of pulmonary tissue is:

1. bronchial breathing;

2. vesicular breathing;
3. indeterminate respiration;
4. "silent" lung;
5. harsh (coarse) respiration.

6. Survey of the patient with the syndrome of fluid accumulation in pleural cavity detects:
1. lagging of the one side of the chest at respiration;
2. protrusion of affected side half of the chest;
3. barrel-shaped chest;
4. retraction of intercostal spaces at inspiration;
5. compelled position of patient (orthopnea).

7. Characteristics of fluid in pleural cavity are:
1. mediastinal displacement in the opposite direction from affected side of the chest;
2. weakening of respiratory sounds;
3. intensifying of respiratory sounds;
4. retraction of intercostal spaces on the side of affection;
5. tympanic sound.

8. Which parameter of spirogram is diagnostic in the syndrome of the obstructive respiratory failure?
1. forced expiratory volume for 1-th second (FEV1);
2. residual air volume (RAV);
3. total lung capacity (TLC);
4. maximum lung ventilation (MLV);
5. vital capacity (VC).

9. Which parameters of spirogram are decreased in the syndrome of the restrictive respiratory failure?
1. total lung capacity (TLC);
2. maximum lung ventilation (MLV);
3. vital capacity (VC);
4. residual air volume (RAV);
5. forced expiratory volume for 1-th second (FEV1).

10. Which parameters of spirogram are decreased in the syndrome of the mixed respiratory failure?
1. total lung capacity (TLC);
2. maximum lung ventilation (MLV);
3. vital capacity (VC);
4. residual air volume (RAV);

5. forced expiratory volume for 1-th second (FEV1).

11. Increase of tactile fremitus is observed in:
1. syndrome of focal consolidation of pulmonary tissue;
2. syndrome of bronchial obstruction;
3. syndrome of obturator atelectasis;
4. syndrome of increased airness of lung (pulmonary emphysema);
5. syndrome of fluid accumulation in a pleural cavity.

12. Weakening tactile fremitus is observed in:
1. syndrome of air accumulation in pleural cavities (pneumothorax);
2. syndrome of fluid accumulation in a pleural cavity;
3. syndrome of obturator atelectasis;
4. syndrome of increased airness of lung (pulmonary emphysema);
5. syndrome of focal consolidation of pulmonary tissue.

13. Elevation of the height of lung apex presents in:
1. syndrome of increased airness of lung (pulmonary emphysema;
2. syndrome of air accumulation in pleural cavities (pneumothorax);
3. syndrome of focal consolidation of pulmonary tissue ;
4. syndrome of obturator atelectasis;
5. syndrome of pleural membranes thickening.

14. Pathological bronchial respiration is observed in the syndrome of:
1. focal consolidation of pulmonary tissue;
2. compressive atelectasis of lung;
3. cavity in lung connected with bronchus;
4. air accumulation in pleural cavities (pneumothorax);
5. bronchial obstruction.

15. Bronchophony is intensified in the syndrome of:
1. focal consolidation of pulmonary tissue;
2. compressive atelectasis of lung;
3. bronchial obstruction;
4. increased air-filling of lung (emphysema);
5. accumulation of air in pleural cavities (pneumothorax).

16. Bronchophony is not determined in the syndrome of:
1. accumulation of air in pleural cavity (pneumothorax);
2. accumulation of fluid in pleural cavity (exudative pleurisy);
3. increased air-filling of lung (emphysema);
4. focal consolidation of pulmonary tissue;
5. obturator atelectasis of a lung lobe.

17. An important auscultative sign of the syndrome of the air cavity in the lung is:
1. "amphoric" bronchial breathing;
2. weakened vesicular breathing;
3. wheezes;
4. coarse crackles;
5. fine crackles.

18. The basic auscultative sign in diagnosis of the pleural membrane thickening syndrome is:
1. pleural rub;
2. bronchial breathing;
3. fine crackles;
4. coarse crackles;
5. rhonchi.

19. Causes of the syndrome of focal consolidation of the pulmonary tissue:
1. lobar pneumonia;
2. pneumofibrosis;
3. tumour of lungs;
4. exudative pleurisy;
5. pulmonary emphysema.

20. Causes of the syndrome of bronchial obstruction:
1. chronic bronchitis;
2. pneumofibrosis;
3. bronchial asthma;
4. lobar pneumonia;
5. pulmonary emphysema.

Unit III. Cardiovascular System Examination

Chapter 8. Subjective and Objective Examination of Patients with Diseases of the Cardiovascular System

Goals: to enable students to learn -

1) subjective examination (inquiry) of the patients with the cardiovascular system diseases and the interpretation of the obtained results;
2) technique of a general survey in the cardiovascular system diseases and its diagnostic value;
3) technique and diagnostic value of a survey and palpation of the heart and greater vessels area;
4) technique and a diagnostic value of arterial, venous and "capillary" pulse examination;
5) technique of measuring and diagnostic value of arterial (blood) pressure.

8.1. Subjective examination (inquiry) of patients with diseases of the cardiovascular system

Complaints

Dyspnea. *Dyspnea* (shortness of breath) is a difficulty breathing with change by its frequency, depth and rhythm, manifested by the feeling of the lack of air. Dyspnea is the early and constant manifestation of the cardiac failure, which should be distinguished from dyspnea in the lung diseases. Dyspnea due to heart diseases is characterized by a violation of breathing in both phases (inspiration and expiration) – *mixed dyspnea*. Inspiratory dyspnea is less often in the cardiac failure.

Cardiac dyspnea increases with a physical activity as well as in a horizontal position of the patient. The difficulty of the respiratory muscles and diaphragm movements is typical as well as an increasing volume of the circulating blood. Therefore, patients with a heart failure tend to take a sitting or semi-sitting position. In severe cases, patients spend all day in a ***forced sitting position (orthopnea)*** (Table 8-1).

Cardiac asthma is attacks of quickly progressing and very severe dyspnea due to diseases of the heart, in which the patient literally suffocates up to asphyxia.

Chest pain is a significant and frequent symptom of the heart diseases. It is extremely important to identify a coronary pain, the basis of which is a chronic or acute coronary blood flow disorder due to atherosclerosis and / or coronary artery thrombosis, leading to ischemia and myocardial necrosis.

Table 8-1. Typical complaints in the cardiovascular system diseases

Complaints	Characteristics	Causes
Dyspnea	- mixed (inspiratory-expiratory); - occurs with physical activity; - combines with orthopnea at the rest	1. Hypertension and congestion in pulmonary circulation because: - left ventricle failure; - heart valves diseases. 2. Pulmonary artery embolism 3. Heart arrhythmias
Pain in the heart region	1. Coronary (ischemic) pain: - behind sternum; - pressing or squeezing, - at a physical load; - short time (5-10 minutes typically); - relieved by nitroglycerin, stop of physical exertion	Chronic or acute disturbance of the coronary blood flow due to atherosclerosis and / or thrombosis of the coronary arteries, leading to ischemia and necrosis of the myocardium
	2. Non-coronary pain (cardialgia): - stabbing or burning, - variable from one episode to another; - variable in time (minutes - hours - days) and position, more frequently at the heart apex; - not related to a physical load; - no response to nitroglycerin	1. Cardiac causes: myocarditis; pericarditis, dyshormonal cardiomyopathy
		2. Outcardiac causes: myalgia, osteochondrosis, intercostal neuralgia, rib diseases, alimentary system diseases, pulmonary pathologies, depression, and alcohol dependence
Edema	- from inferior extremities; - accumulation of fluid in serous cavities (ascites, hydrothorax, hydropericardium)	- right ventricle failure; - pulmonary hypertension; - dilated cardiomyopathy; - myocarditis
Heart palpitations and intermissions	- patient's feelings of accelerated and intensified heart contractions; and escaped beats	- extrasystoles; - atrial fibrillation; - paroxysmal tachycardia; - sinoatrial and atrioventricular blocks of II degree
Cough and hemoptysis	Cough - usually dry together with dyspnea, and as a rule, under a physical load or in a horizontal position	- left ventricular heart failure; - pulmonary hypertension; - aortic aneurysm; - pulmonary embolism with development of infarct-pneumonia

Such a pain is called ***angina pectoris, or stenocardia, or anginous pain.*** The pain lasts typically from a few seconds to 2-5 minutes, rarely up to 20-30 minutes.

Anginous pains usually occur as attacks of squeezing, pressing, or burning pain behind the middle and the inferior parts of sternum, often during exercise, but sometimes at rest. With a significant duration and intensity of anginous pain, they are regarded as manifestations of acute coronary syndrome (ACS) or myocardial infarction (MI).

The most important characteristic of angina pectoris is the disappearance or distinct reduction of the pain after sublingual administration of nitroglycerin or cessation of a physical activity. Among other serious causes of the chest pain, there are dissecting aneurysm of the thoracic aorta, thromboembolism of the pulmonary artery branches, pericarditis.

Heart palpitation and intermissions

Heart palpitation is a subjective feeling of strengthening and increasing heart beats, may be constant or occur paroxysmally. As a rule, the basis of this feeling is a true increase in a heart rate (sinus tachycardia or various heart rhythm disorders); sometimes such complaints are made by patients.

The feeling of *intermissions* (or *interruptions, or escaped beats) in the heart* is accompanied by a sense of fading, "somersaults", and cardiac arrest, and is usually associated with a violation of the heart rhythm or heart blocks.

Cough and hemoptysis

Cough (Lat. tussis) in cardiac patients occurs due to congestion in the pulmonary circulation with the left ventricular heart failure or mitral stenosis. Among other "heart" causes of cough – thromboembolism of the pulmonary artery branches. Cough in the heart failure is generally dry, appears with dyspnea, usually under physical load or in a horizontal position. Cough can be productive sometimes combined with hemoptysis.

Coughing with blood (Lat. hemoptysis or, pneumorrhagia) is characterized by the presence of blood in the sputum. In cardiac patients, hemoptysis in most cases is due to congestion in the small circle of the circulation and the release of red blood cells through the capillary wall.

Edema.

Edema on the inferior extremities is a common complaint with the development of heart failure. It is necessary to clarify the predominant localization of edema, the time of their appearance (in the morning, at the end of the day), the relationship with fluid intake, a physical activity. Edema in case of the heart failure is characterized by swelling in the inferior extremities mainly by the end of the day to the evening, rapidly increasing with intake of large amounts of liquid and salt food, physical overload, after a long stay in an upright position. This is due to venous congestion in the lower extremities and the right ventricular heart failure**.**

As the heart failure progresses, swelling is spreading on tibia, thighs, scrotum, and loin. A significant accumulation of fluid in the tissues, especially in the subcutaneous tissue (***anasarca***), usually accompanied by the accumulation of fluid in the pleural cavities (***hydrothorax***), in the pericardium cavity (***hydropericardium***), the abdominal cavity (***ascites***).

Other complaints

Patients may have additional complaints: *weakness, fatigue, sweating, and sleep disorder*. All these complaints are due to developing hypoxemia against the background of the heart failure.

Headache is a characteristic complaint in arterial hypertension and arterial hypotension. In the first case, the pain is localized typically in the occipital-parietal region, in the second – in the temporal-parietal region. One of the most common cause of a headache is a cerebral ischemia due to atherosclerosis of the brain blood vessels.

Dizziness, noise in the head are characteristic of arterial hypertension, and cerebral atherosclerosis.

Complaints of a dyspeptic character (nausea, vomiting, decreased appetite, diarrhea or constipation). They occur against the background of the right ventricular heart failure with venous congestion in the system of *Vena cava inferior*. In this case, there are often *pains and/or heaviness in the right hypochondrium* because of the acute enlargement of the liver due to the venous congestion.

Insomnia, excitement, sometimes psychotic disorders (delirium, hallucinations) are often noted in patients with a severe heart failure on the background of severe hypoxemia.

Anamnesis

When taking a *history of present disease (anamnesis morbi)*, it is important to fix the time of the occurrence of the symptoms of the heart damage, their relationship with previous infections (myocarditis, acute rheumatic fever, infectious endocarditis), other diseases (anemia, thyrotoxicosis), age, physical and emotional strain.

From the *past life history* (*anamnesis vitae*) of the patient, a physician pays attention to the risk factors of heart diseases: adverse living and working conditions (stay in a damp room, nervous and mental stress, hypodynamic lifestyle, overeating), bad habits (smoking, etc.). It is important to pay attention to the course of pregnancy, childbirth, and menopause.

8.2. General survey

A general survey of a patient with a disease of the cardiovascular system includes determining his/her position in bed, facial expressions, color of the skin and mucous membranes, the presence of edema (see Chapter 2. Objective examination of a patient. General inspection (survey). The most informative significant signs in cardiac diseases are given here (Table 8-2).

Depression of consciousness in a severe cardiac patient can be considered as one of the reliable signs of a severe heart failure (shock), indicating hypoperfusion (poor blood supply) of the brain.

The doctor would pay attention to a *position of patients*. Patients with a severe heart failure sit with their legs down. In acute vascular insufficiency, patients lie on a bed with a low headboard, avoiding movements. *Orthopnea* (sitting or semi-sitting position on the bed with raised headboard) is a characteristic symptom of the heart failure. The sitting position leaning forward is typical in exudative pericarditis.

Table 8-2. General survey in the cardiovascular system diseases

Signs	Characteristics	Causes
Forced position	sitting or semi-sitting position (orthopnea) in bed with raised head, without support on hands	a heart failure
	sitting position with forward inclination	- exudative pericarditis
Acrocyanosis	peripheral (fingers and toes, the tip of the nose, the lips, and the ear lobes) blueness of the skin and visible mucosa	- a heart failure; - congenital heart diseases; - heart valves diseases
"Facies mitralis"	violet-red colour of the patient's cheeks, mildly cyanotic colour of the lips, nose	mitral stenosis
Other changes of the skin	paleness	-aortal valves diseases, -arterial hypotension
	hyperemia (redness)	- arterial hypertension - fever
	colour of “milk-and- coffee” with hemorrhagic spot	infective endocarditis
Edema	- from inferior extremities; - especially in the evening; - accumulation of fluid in serous cavities (ascites, hydrothorax)	- right ventricle failure; - pulmonary hypertension; - dilated cardiomyopathy; - myocarditis
Hippocratic (“drum-stick”) fingers, or nail clubbing	clubbing of the fingers distal finger phalanges with loss of the nail bed angle (“watch-glass” nails)	condition of chronic hypoxemia in the chronic heart failure

Examination of the skin. The most common cardiac symptom on the part of the skin is its peripheral blueness (*acrocyanosis*) in the heart failure. Acrocyanosis occurs as a result of slowing blood flow and greater oxygen return to the surrounding tissue. In these cases, the venous blood concentration of carboxyhemoglobin is more than in norm.

Characteristic features of acrocyanosis:

- localization at the tip of the nose, lips, hands, feet;
- the intensity of cyanosis can increase with exercise: from light bluish color to a expressed black and blue color;
- the skin in the area of acrocyanosis is cold to the touch.

A face examination may provide additional diagnostic data. Most typical:

- *mitral face* (*facies mitralis*) in patients suffering from mitral stenosis: against the background of pallor, cyanosis of the lips, cheeks, tip of the nose, earlobes is clearly manifested;

- *face of Corvisart* (*facies Corvisari*) in patients with severe heart failure: a lean, pale with cyanotic icteric tinge face, opened mouth, dyspnea at rest, orthopnea.

It is much less often, a jaundice is associated *with venous congestion in the liver* in the right ventricular failure.

A kind of *jaundice combined with pallor* develops sometimes in subacute infective endocarditis: the skin has a shade of *"coffee with milk"*. Tiny embolisms in the capillaries of the skin and especially the capillaries of the conjunctiva in endocarditis give spot hemorrhages.

Edema of subcutaneous tissue in patients with a heart disease often indicates the presence of a heart failure (see Chapter 2. Fig. 2-4). It is very important to assess if an edema is symmetric or not. The predominant edema of one of the lower limbs leads to suspicion of deep vein thrombosis of the limb with the risk of thromboembolism of the pulmonary artery.

Signs of edema of cardiac origin:

- it appears on symmetrical areas, when walking patients - first on the lower extremities (feet, ankles) during the day time, especially in the evening (a symptom of "tight shoes"), and disappears during the night time towards the morning;
- edemas change its position: at the patient with swelling of the lower limbs, laid on the bed, swelling goes to the waist; at the patient lying on his side - on the side and the corresponding hands and feet;
- at palpation, the swelling of the heart is initially mild; but prolonged edema, especially on the legs, becomes dense;
- skin in the places of edema is cyanotic;
- edematous skin is tense, smooth, subcutaneous tissue ruptures can be under the skin;

- at extreme degrees, bubbles are formed, which can burst, and edematous fluid is released from them;
- after edema, the upper layer of the skin is dry, wrinkled and easily exfoliated;
- in addition to edema of subcutaneous tissue, edema develops in parenchymal organs - liver, kidneys, gastrointestinal tract (congested liver enlargement, intestinal dysfunction);
- rising up, edematous fluid accumulates in the abdominal cavity (*ascites*), then in the chest cavity (*hydrothorax*) and in the pericardial cavity (*hydropericardium*).

Changes in the type of "drum-stick (Hippocratic) fingers (nail clubbing) (see Chapter 2. Fig. 2-6) sometimes are detected in infective endocarditis and heart valve diseases, are not specific as occurs in pulmonary and some other diseases (chronic anemia).

8.3. Survey of the heart region and the peripheral blood vessels

8.3.1. Survey of the heart region and greater blood vessels

Survey of the heart region is carried out primarily to identify apex and cardiac impulses, and other pathological pulsations.

*"**Cardiac hump**"* is uniform protrusion of the chest wall to the left of the sternum. It is formed during the development of a heart valve disease in early childhood as a result of the ribs deformation by increased contractions of the hypertrophied heart.

Apical impulse is a limited rhythmic pulsation of the left ventricle apex observed normally in V-th interspace inside of the *linea medioclavicularis sinistra*. In 30% of healthy people, apical impulse is not determined.

Apical impulse cannot be detected during the inspection:

- in hypersthenics, due to narrow intercostal spaces;
- in people with a strongly developed muscles of the chest;
- in case of weak heart contractions;
- -if the apical impulse is behind the rib.

With a pathological enlargement of the left ventricle, the apical impulse can shift to the left and down. With deep inspiration, the apex descends slightly, while exhaling - rises. In a high standing diaphragm (ascites, flatulence, pregnancy), it moves up and to the left, with a low standing diaphragm (emphysema of the lungs, asthenics) – down and inside (to the right). In increasing pressure in one of the pleural cavities (exudative pleurisy, pneumothorax), the apical impulse shifts in the opposite direction.

Positive apical impulse is a visible protrusion of the chest wall tissues during the systole. It is revealed in norm and in case of the heart diseases.

Negative apical impulse is a visible retraction of the chest tissues during the systole. It occurs because of adhesions of both pericardium sheets or between external sheet of pericardium with the chest wall and pleura (adhesive pericarditis).

The *false negative (pseudonegative) apical impulse* can be observed at asthenics, in case the range of an apical beat settles down opposite to the rib. It is a systolic intrusion of a thorax wall to the right and higher of a place of the routine localization of an apical impulse that it is inaccurately possible to accept for a negative apical beat.

Cardiac impulse is a pulsation observed sometimes synchronous with the apical impulse at the IV-V-th interspaces near the left edge of the sternum. It is due to the pulsation of the enlarged right ventricle with projection to the left of the sternum. Normally, cardiac impulse is absent as a rule, except occasionally in young people with a thin chest wall.

Other pulsations at the heart and greater vessels region

Epigastric pulsation of hypertrophied right ventricle may be in pulmonary heart, mitral stenosis, tricuspid valve insufficiency. This pulsation may be *visible directly below xiphoid process*, and is enhanced by deep inspiration, and in a vertical position.

Pulsation of abdominal aorta can be observed in asthenics, *at epigastrium and mesogastrium, slightly to the left of midline*, the best – in the time of expiration in a horizontal position of the patient. *Prominent pulsation of abdominal aorta* is visible in aneurysm of the abdominal aorta or in the presence of a tumor prior to the aorta, transmitting its vibrations.

Pulsations in the right hypochondrium (area of the liver projection) can be visible as the *true or transmitting pulsation of the liver*.

True pulsation of the liver is a result of changes in its blood supply. True pulsation of the liver (alternating increasing and decreasing its volume) is observed in tricuspid valve insufficiency. This is venous pulsation of the liver.

Transmitting pulsation of the liver are motions of the liver in the one direction when transmitting heartbeats to it. It is linked to the transfer pulsation of the hypertrophied and dilated right ventricle contractions (in pulmonary heart, mitral stenosis, tricuspid valve insufficiency).

At examination of the neck, "*Corrigan's pulse, or hyperkinetic pulse*" can be observed. This is an increased (in amplitude and in velocity) pulsation of the carotid arteries, the characteristic of the aortic valve insufficiency.

8.3.2. Survey of the peripheral blood vessels

The so-called *capillary pulse* (*Quincke's pulse) - periodic redness (in systole) and paleness (in the diastolic phase) of the nail bed due to arterioles pulsation* (Fig. 8-1). It appears in young adults with a high temperature, after applying heat procedures.

Pathological capillary pulse is possible to identify visually in the aortic valve insufficiency, and sometimes in thyrotoxicosis. To identify it, lightly press the end of the nail, below in the middle of it a small white spot appears at each pulse stroke; it will expand and then shrink. It can also be identified as a discoloration hyperemic spot obtained on the mucosa of the lips while pressing on them with a glass slide.

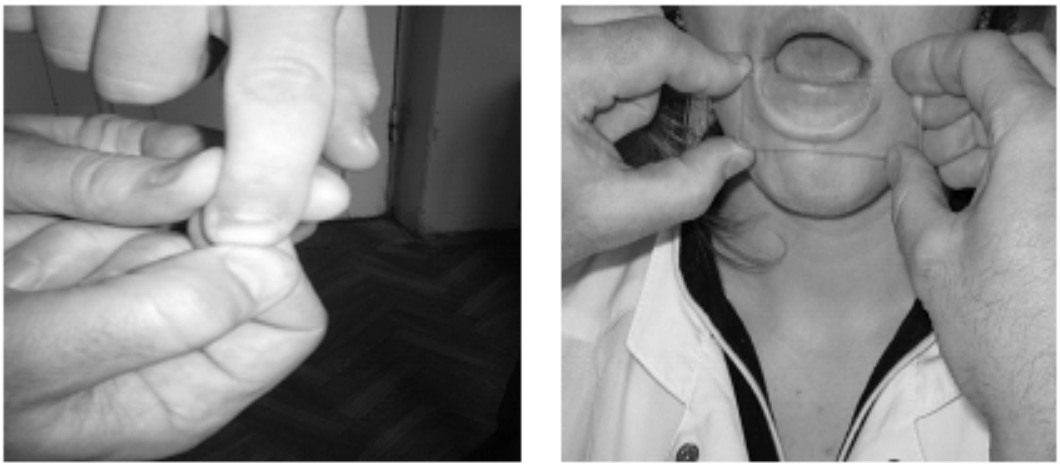
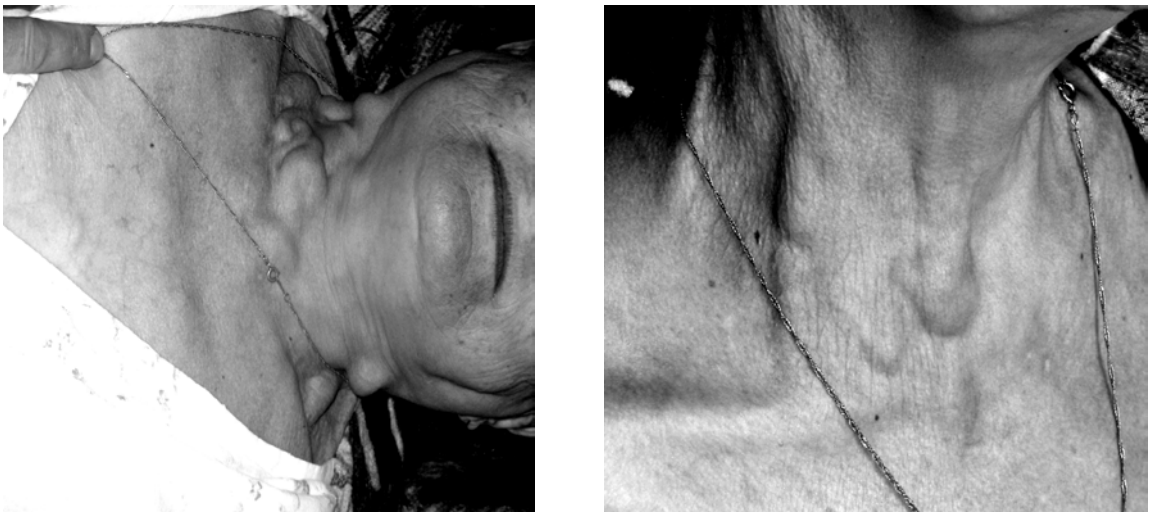


Fig. 8-1. Examination of the capillary pulse.

Dilated neck veins present in affection of the right heart chambers, and external compression of *vena cava superior* (adhesive pericarditis, pulmonary emphysema, pneumothorax, compression of tumor or mediastinal aortic aneurysm) or in occlusion of it by a thrombus (Fig. 8-2).



(1) (2)

Fig. 8-2. Dilation of the neck veins:
(1) in horizontal position; (2) in vertical position.

The expressed edema of the face and neck and the jugular veins dilation (*collar of Stokes*) occurs during *vena cava superior compression* by mediastinal tumors, or aortic aneurysm.

The difficult venous blood flow by the *vena cava inferior* results in edema and veins dilation of the inferior extremities (Fig. 8-3), and the veins of the lateral part of the abdominal wall (collaterals to *vena cava superior*).

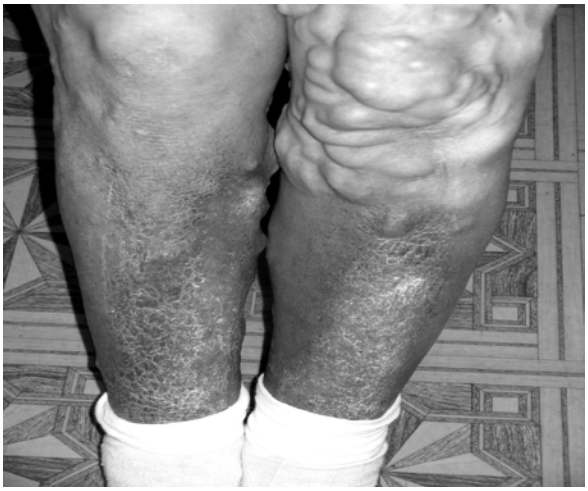


Fig. 8-3. Varicose veins of the inferior extremities

Venous hypertension in the system of *vena portae* and its branches results in the dilation of the superficial veins on abdominal surrounding umbilicus (so called *caput medusa*e – venous collaterals to *vena cava superior et inferior*).

Venous pulsations

Negative venous pulse is observed at the neck, when during the systolic expansion of the carotid artery, the jugular vein is reduced. The activity of the heart causes a rhythmic slowing of the blood flow in jugular veins during the atrial systole, and the acceleration of it during the atrial diastole coincides with the ventricular systole. Slowing of the blood flow leads to some neck veins swelling, and accelerating of the blood flow leads to a decrease in vein swelling.

In a healthy person, a *small pulsation of the jugular veins (negative venous pulse)* can be seen just in the horizontal position, in the vertical position it disappears. The pulsation is best visible when the muscles of the neck are relaxing, and the direction of the light is tangentially to the skin surface.

Positive venous pulse is observed when the veins expand simultaneously with the appearance of an arterial pulse wave. It is detected on the jugular veins in case of the tricuspid valve insufficiency, when in the ventricular systole; the reverse wave of the blood from the right ventricle to the right atrium delays the outflow of the blood from the veins to the atrium.

8.4. Palpation of the heart area

Palpation of the heart area enables:

- *to detect the presence of a cardiac impulse;*
- *to determine the apical impulse and its properties;*
- *to identify the thrill of chest wall in the heart area.*

8.4.1. Cardiac impulse palpation

Cardiac impulse is a fluctuation of the chest in the place of the direct application of the heart to it, not covered by the lungs, due to the contraction of the ventricles.

Cardiac impulse is palpated with the entire palpable surface of the right hand in the IV-V intercostal space to the left of the sternum or in the epigastric region under the xiphoid process of the sternum at the height of deep inspiration (Fig. 8-4). Cardiac impulse is palpable in case of the right ventricle hypertrophy and dilation.

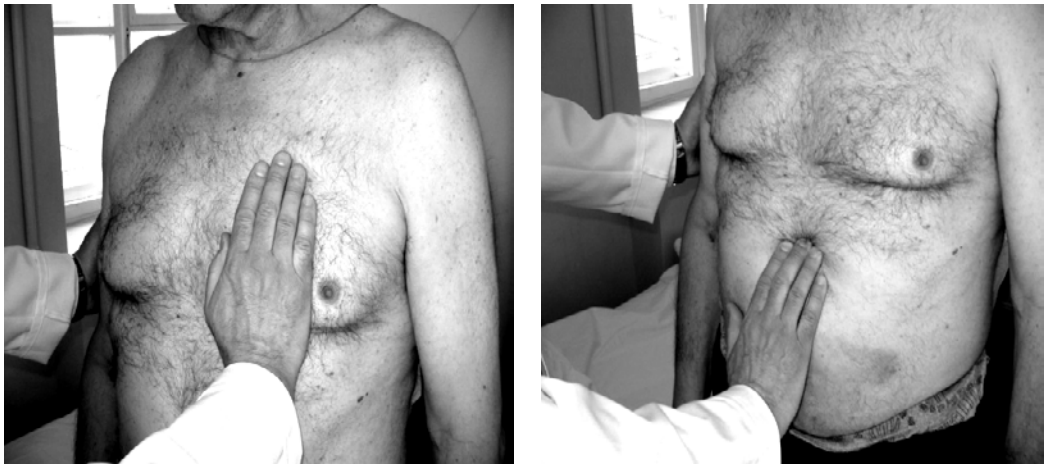


Fig. 8-4. Palpation of the cardiac impulse to the left of the sternum and in the epigastric region

8.4.2. Apical impulse palpation

Apical impulse is the fluctuation of the tissues of the chest wall in the region adjoining the heart apex, synchronous with the systole of the ventricles.

Sometimes apical impulse is not palpated. In such cases, it is recommended to slightly tilt the patient anteriorly, or ask the patient to perform several squats. You can put the patient on the left side, the apical impulse is shifted by 2 cm the outside, but it is better palpable.

Palpation can determine properties of the apical impulse:

• *localization,*

• *area,*

• *height,*

• *resistance.*

Localization of the apical impulse

To determine the apical impulse, the physician-examiner places the palm of the right hand on the heart area so that it is located in the zone from the III to the VI rib - vertically, and from the left sternal line to the anterior axillary – horizontally. Fingers are turned to the axillary lines (Fig. 8-5).

In women with the developed breasts, it is initially necessary to lift the left breast up and to the right.

The localization of the apical impulse is determined by one finger, index or middle, moving it from the outside inwards along the intercostal space to the outer pulsation point.

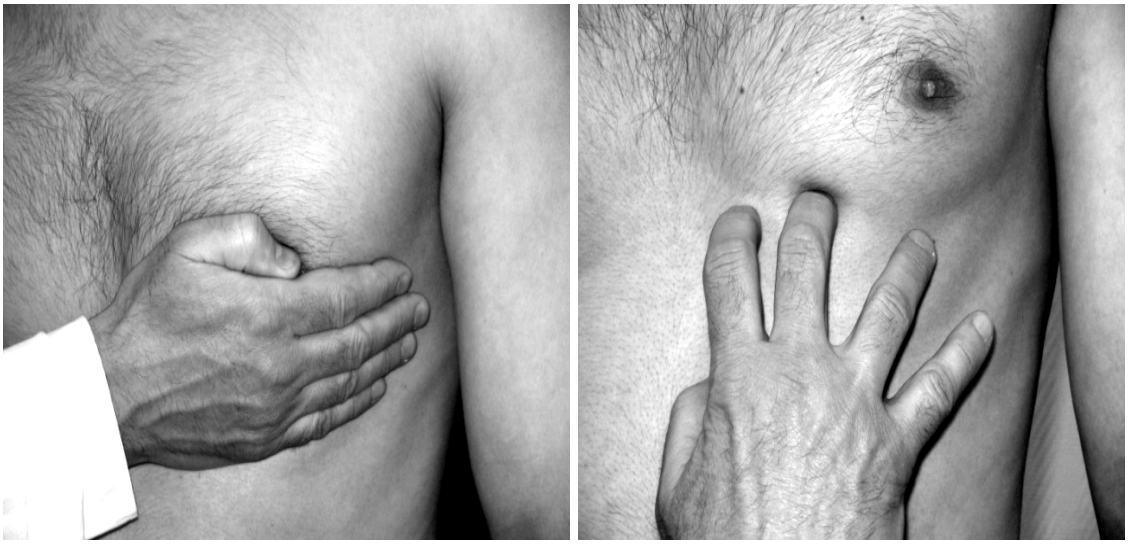


Fig. 8-5. Palpation of the apical impulse localization

Normally, the apical impulse localization is 1.5 cm inside of the left midclavicular line in the V intercostal space (Table 8-3). Sometimes the apical impulse is palpated not in one intercostal space, but in two or even three. *The localization is the outer and lower extreme point of the apical impulse.*

With a deep inspiration, the apical impulse somewhat descends, with a deep expiration – rises. This is a *physiological shift of the apical impulse.*

Diseases, accompanied by hypertrophy of the left ventricle, lead to a shift of the apical impulse to the left midclavicular line. With dilatation of the left ventricle, the apical impulse shifts outward from the midclaviular line.

The defects of the aortic valve (stenosis and insufficiency) displace the apical impulse to the left and down, in case of insufficiency of the mitral valve, arterial hypertension - to the left.

Extra-cardiac causes, leading to a change in the localization of the apical impulse: With a high standing diaphragm (ascites, flatulence, pregnancy), it moves up and to the left, with a low standing diaphragm (emphysema

of the lungs, asthenics) – down and inside (to the right). With increasing pressure in one of the pleural cavities (exudative pleurisy, pneumothorax), the apical impulse shifts in the opposite direction, and with wrinkling processes in the lung – towards the pathological focus.

The apical impulse is not palpable normally in 1/3 of the patients, as it can be closed by the rib. The apical impulse is determined on the right in congenital dextrocardia. In exudative pericarditis, the left-sided hydrothorax and pneumothorax, the apical impulse is not palpated.

Table 8-3. Characteristics of the apical impulse

Property	Norm	Alterations
Localization	V interspace, 1-1,5 cm toward the sternum from the left midclavicular line	down- and left shift – in the left ventricle hypertrophy
		left shift – in the left and/or right ventricle hypertrophy
		upward shift – in high position of the diaphragm
		down- and right shift – low position of the diaphragm
Area (width)	1-2 cm^2	diffuse apical impulse (>2 cm^2)– in the left ventricle hypertrophy, pneumosclerosis, in asthenics)
		restricted apical impulse (< 1 cm^2) - in pleurisy, pericarditis, adhesions, emphysema, hypersthenics, the low position of the diaphragm
Height	Moderate (depending on the contractile strength of the heart)	high apical impulse – in asthenics, physical exercises, emotional stress, fever, thyrotoxicosis, left ventricle hypertrophy; low apical impulse – in hypersthenics, obesity, pulmonary emphysema, left-sided exudative pleurisy, dilatation of the left ventricle

Resistance	Moderate (depends on thickness of the chest wall, the strength of contractions and density of the left ventricle	resistant apical impulse - in the left ventricle hypertrophy)

Area (width) of the apical impulse.

The area of the apical impulse is determined after finding its localization. The middle finger of the right hand is perpendicular to the surface of the chest at the external pulsation point, and the index finger is displaced to the inside along the intercostal space until the pulsation disappears. The distance between the two marks – the width (area) of the apical impulse (Fig. 8-6.).

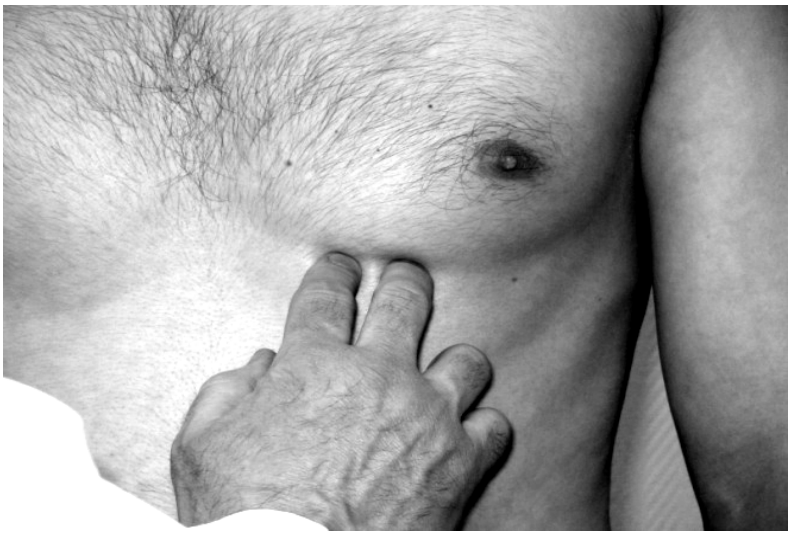


Fig. 8-6. Palpation of the apical impulse width (area)

The width of the apical impulse is normally 1-2 cm. If the apical impulse is localized in one intercostal space, and the distance between the two edges is 1 cm, then the area of the apical impulse can be calculated by multiplying its width by 1 cm. The normal apical impulse area is of 1-2 cm^2.

Restricted (limited) apical impulse - area less than 1cm^2.

The restricted apical impulse occurs when a smaller surface of the heart adjoins to the anterior chest wall - in narrow intercostal spaces, the hypersthenic body-build type, during a deep inspiration. A pathological restricted apical impulse occurs in obesity, emphysema of the lungs, left-sided exudative pleurisy.

Diffuse apical impulse - area more than 2 cm^2.

The diffuse apical impulse occurs when a larger surface of the heart adjoins to the anterior chest wall - in the asthenic body-build type, in wide intercostal spaces, during expiration. A pathological increase in the area of the apical impulse is detected in hypertrophy and dilatation of the left ven-

tricle (insufficiency of the mitral valve, insufficiency of the aortic valve, arterial hypertension).

Height of the apical impulse

For the definition of the apical impulse height, the 3-rd middle finger is displaced perpendicularly to a maximal pulsation place of the apical impulse area (Fig. 8-7).

It is *a moderate height of the apical impulse* if the finger feels a pressing mild tissues of the distal phalanx. It is a *high apical impulse* if the distal phalanx moves synchronously with a pulsation. It is a *low apical impulse* if a weak sensation of a pulsation without pressing of the mild tissues of the phalanx.

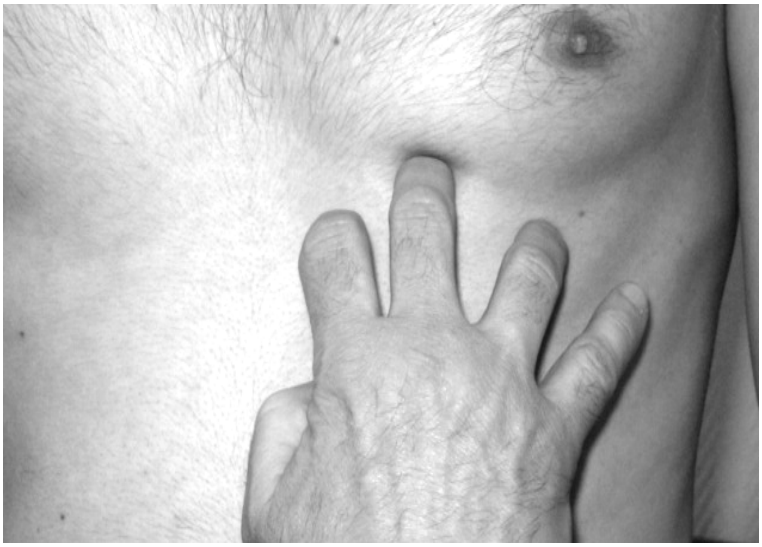


Fig. 8-7. Palpation of the apical impulse height and resistance

High or low apical impulse may be in pathology, and sometimes in norm. The height varies with the area of the apical impulse. High apical impulse is usually diffuse, and low apical impulse is restricted.

Normally, it is moderate height of the apical impulse.

The height of the apical impulse depends on the strength of the contraction of the left ventricle, thickness of the chest wall, and width of interspaces.

The *physiological high apical impulse* occurs in the asthenic body-build type, wide intercostal spaces, in physical and emotional stress. The p*athological high apical impulse* occurs in fever, thyrotoxicosis; in hypertrophy of the left ventricle (mitral insufficiency, aortic valve insufficiency, aortic stenosis, arterial hypertension).

The *physiological low apical impulse* occurs in the healthy hypersthenic body-build type, with narrow intercostal spaces, during expiration.

The *pathological low apical impulse* may be in: (1) extracardiac diseases (obesity, emphysema, left-sided pleural effusion); (2) dilation of the left ventricle (mitral insufficiency, aortic valve, reducing the tone of the left ventricular myocardium in myocarditis, myocardial dystrophy, and myocardial infarction).

Resistance of the apical impulse

Resistance of the apical impulse is estimated by resistance that is felt by the palpating finger at the attempt to prevent the pulsations of the apical impulse. To do this, the middle finger of the right arm, located perpendicular to the surface of the chest at the place of the maximum pulsation, is pressed against the chest (see Fig. 8-7).

Moderately resistant apical impulse is a pulsation that can be suppressed with a certain effort.

Resistant apical impulse is not possible to suppress.

Non-resistant apical impulse means that a slight pressure stops pulsation of the chest wall in the area of the apical impulse.

Normal resistance of the apical impulse is moderate.

The *resistant apical impulse* occurs in hypertrophy of the left ventricle (mitral valve insufficiency, aortal valve insufficiency and stenosis, arterial hypertension).

Its resistance decreases in dystrophy and dilation of the left ventricle.

With the severe hypertrophy of the left ventricle, the apical impulse becomes diffuse, high, resistant and, when palpated, gives a feeling of a dense elastic dome ("*dome-shaped" apical impulse*). Such apical impulse happens in the aortic valve insufficiency.

8.4.3. Palpation of *"thrills"*

Chest thrill ["cat's purring" – literal translation from Russian ("кошачье мурлыканье")] is noted above the apex of the heart during diastole in mitral stenosis and over the aorta during systole in aortic stenosis. Chest thrill isa result of the vortex blood flow through the narrowed opening of the mitral or aortic valves. It resembles the feelings of "thrills" at palpation.

There are systolic and diastolic variants of thrillы depending on which phase of the cardiac activity it appears.

Diastolic thrill is palpated by the palm put on the apex of the heart during the diastole (in mitral stenosis).

Systolic thrill is palpated by the fingers of the palm applied at the II-d intercostal space near the right edge (in aortic valves stenosis) or at the left edge of sternum (in the pulmonary artery valves stenosis, and the opened ductus arteriosus).

8.5. Arterial pulse palpation

Arterial pulse (pulsus) is called rhythmic oscillations of the artery wall, due to the changes in their blood filling as a result of heart contractions. In clinic of internal diseases, radial artery pulse is usually examined like a part of an assessment of the general condition and cardiovascular system functional abilities of patients.

Following properties of pulse are examined by palpation:

- ***similarity (uniformity, equality) of the pulse on both radial arteries;***
- ***rhythm of the pulse,***
- ***frequency (pulse rate),***
- ***arterial wall elasticity,***
- ***filling radial artery pulse,***
- ***resistance (tension, strain) of the pulse,***
- ***size of the pulse,***
- ***deficiency of the pulse.***

Palpation of the pulse begins with a study of the pulse on both hands.

To palpate the pulse on the radial artery, the thumb is placed on the back surface of the arm and forearm, and the remaining fingers – on the place where the artery passes. On the artery it is best to impose 2-3 fingers (one finger is difficult to find an artery and determine the characteristics of the pulse). The doctor always takes the right hand of the patient with his left hand, and the left hand with his right hand.

In norm, a radial artery pulse is identical (symmetrical, equal, uniform) on both arms. If the pulse is the symmetrical on both arms, the study of its characteristics is carried out on one hand. At the presence of a various pulse on both hands, its characteristics are examined on the side where they are expressed better (Fig. 8-8).

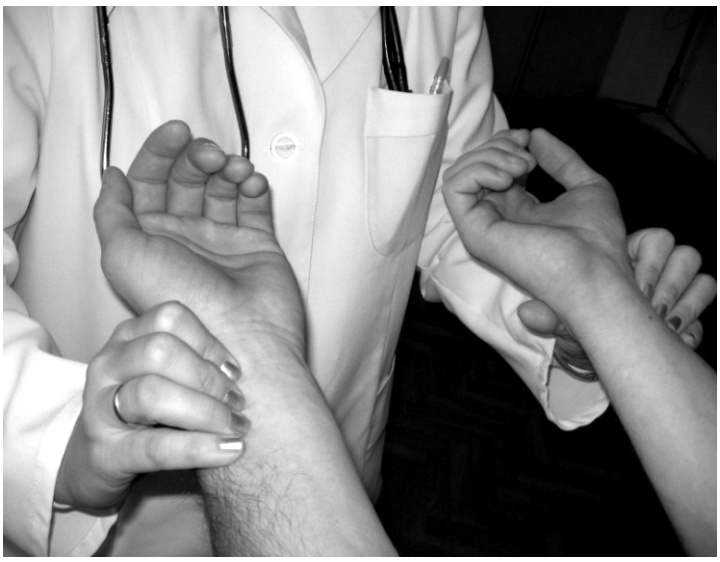


Fig. 8-8. Palpation of similarity (uniformity) of the radial artery pulse

The pulse on the radial arteries can be asymmetrical (*pulsus differens*). The pathological processes (unilateral abnormalities of the structure and position of the peripheral arteries, arterial compression by tumors, scars, enlarged lymph nodes, aortic aneurysm and its branches, mediastinal, retrosternal localization of goiter) can deform the arterial vessel in the path of the pulse wave. Unilateral reduction of filling the pulse appears.

Sign of Popov and Savelyev is described as *pulsus differens* with lowering amplitude pulsation of the left radial artery in mitral stenosis, because compression of the left subclavian artery by the dilated left atrium.

After definition of *similarity (uniformity) of the pulse* on both hands, a physician determines *rhythm of the pulse*. For the definition of rhythm of the pulse, the 2-, 3-d, and 4-th fingers of a palpating arm are positioned on a radial artery, and the 1-t finger on a forward surface of a forearm from the back side. If pulsations follow one after another through identical intervals, the pulse is rhythmical (*pulsus regularis, s. rhythmicus*). The arrhythmical pulse (*pulsus irregularis, s. arrhythmicus*) is characterized by the irregular intervals between the pulse waves (in heart arrhythmia).

Normal pulse is rhythmic (regular).

For the definition of a *pulse rate,* a physician puts three fingers of a palpating arm (2-, 3-d, 4-th) on the radial arteria and counts the number of pulse strokes for 30 s, and the received number multiply accordingly by 2 (at the rhythmical pulse). At the arrhythmic pulse, the rate is counted inventory within 1 minute.

Normal frequency of the pulse rate is 60-90 in 1 minute.

A pulse rate more than 90 for 1 minute pulse refers to a frequent pulse (*pulsus frequens*). The decrease of a pulse rate less than 60 for 1 minute refers to infrequent pulse (*pulsus rarus*).

Some types of arrhythmias are relatively easy to caught by palpation:

- *Respiratory arrhythmia* – a heart rate during breathing movements that quickens (during inspiration), then slows down (during exhalation). When the breath is held, this type of arrhythmia eliminates;

- *Extrasystoles (ectopic beats)* – the pulse waves are smaller in size, appear earlier than usual time (premature contractions), after which a long pause (a *compensatory pause*) follows;

- *Atrial fibrillation* – an arrhythmic pulse, some of its waves of different sizes; and the *deficiency of pulse (see low) may occur*;

- *Paroxysmal tachycardia* - begins as a sudden attack and ends a pulse frequency of up to 140 beats per minute, which is not the case with other rhythm disturbances;

- *II-degree of atrioventricular block and sinoatrial blocks – arrhythmic* pulse with periodical missing each third or fourth pulse waves;

- *III-degree of atrioventricular block* - very rare (less than 40 beats), regular, and is not an accelerated pulse.

Elasticity of the vascular wall

To determine the state of the radial artery wall, three fingers of the palpating hand (second, third, fourth) are placed on it. Initially 2-d finger squeezes the artery until the cessation of the reverse current of the blood from the blood vessels of the brush, and then a 4-th finger squeezes it until the termination of the passage of the pulse wave. The 3-d finger lies freely on the artery and slides along the vessel wall (Fig. 8-9).

Normally, the arterial wall is soft, elastic, smooth. Atherosclerotic arteries under a seal's third finger is felt as a tight rough convoluted tube.

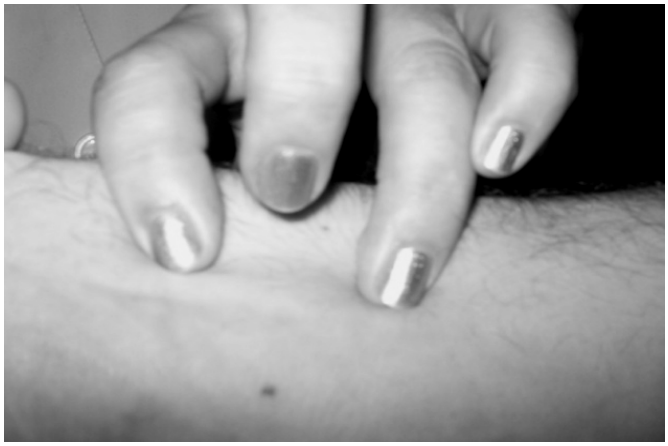


Fig. 8-9. Palpation of elasticity of the radial artery wall

For the definition of the *filling (volume) of the pulse* three fingers of a palpating arm (the 2-, 3-d, 4-th) are placed on *a. radialis*. In the beginning, the 2-nd finger squeezes a. radialis up to the arrest of a reversed current of a blood from the vessels of a hand, and then the 4-th finger squeezes out the blood from the vessel and squeeze it up to the arrest of the transit of the pulse wave. The 3-rd finger freely lies on an empty arteria. The 4-th finger is raised with the pulse wave. The pulse wave passing under the 3-rd finger raises it and hits about the 2-nd finger. The filling of the pulse is assessed on the feelings of the arterial wall elevation by 3-d finger (Fig. 8-10).

The normal pulse is of the satisfactory filling, while there is the pressure of the soft tissues of the finger, without lifting it.

Full pulse (pulsus plenus) – there is a fluctuation of the palpating finger. A full pulse occurs in athletes during sports meets and physical activities.

Empty pulse (pulsus vacuus) – pulse waves without the feeling of pressure on the soft tissues of the palpating finger. The empty pulse occurs in arterial hypotension, acute heart insufficiency.

Resistance of the pulse, or tension (strain) of the pulse

Pulse resistance depends on the level of systolic blood pressure and vascular wall tone. For definition of strain of pulse the 2-, 3-d, and 4-th fingers of a palpating arm squeeze the artery until the complete cessation of pulsation (Fig. 8-11). The degree of pulse resistance is assessed by the force that is necessary to compress the artery.

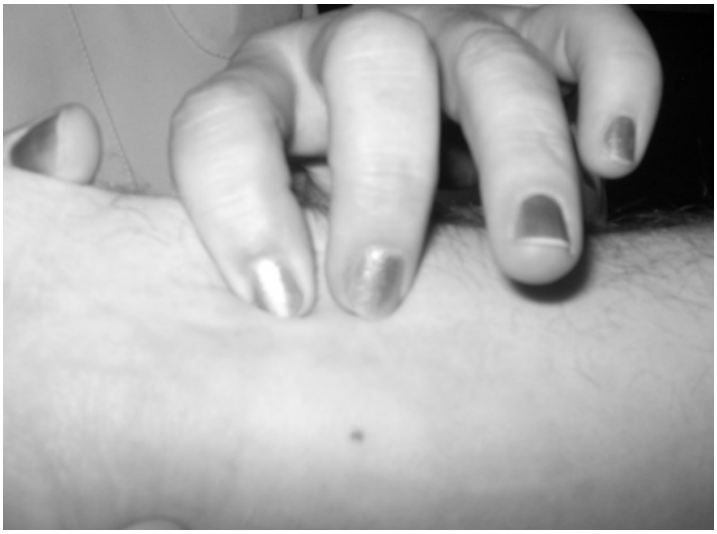


Fig. 8-10. Palpation of the filling radial artery pulse

Normal pulse is of satisfactory resistance.

The solid (dense) pulse (pulsus durus) is observed with strong compression of the artery when the arterial pulsation is not suppressed. The solid pulse occurs in arterial hypertension, atherosclerosis of the arteries.

The soft (mild) pulse (pulsus mollis) is a minimal effort required to suppress the pulsation. The soft pulse occurs in arterial hypotension, acute bleeding, mitral stenosis, mitral valve insufficiency, aortic stenosis.

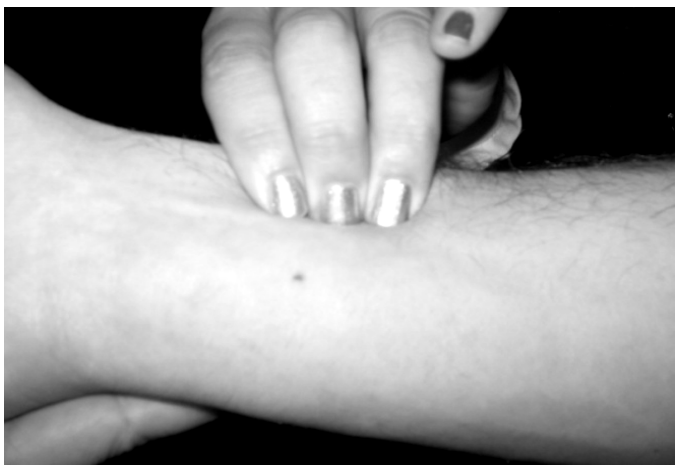


Fig. 8-11. Palpation of the radial artery pulse tension (strain of the pulse)

Size of the pulse is an integral characteristic depending on the filling and resistance of pulse, elastic properties of the arterial wall, and the difference between systolic and diastolic blood pressure (BP). If the patient has also increased the left ventricle output and the significant difference between systolic and diastolic BP, and a decreased vascular tone, the pulse on the peripheral arteries is perceived as a large (*pulsus magnus, or pulsus altus*). This pulse occurs in the aortic valve insufficiency, thyrotoxicosis, and fever.

Opposite while reducing the peripheral circulation, a small difference between systolic and diastolic BP, there is a small size of the pulse (*pulsus parvus*). This pulse is noted in aortic stenosis, tachycardia, acute heart failure.

The extremely small *thready-like pulse (pulsus filiformis)* occurs in shock, massive blood loss, when at the same time reduce presents in filling and resistance of pulse reduce, and the systolic BP.

Deficiency of the pulse is a difference between the rate of the pulse waves on the peripheral artery and the rate of the heart beats. The deficiency of the pulse is found by a palpatory-auscultative method. For the deficiency of the pulse, the heart rate does not correspond with the pulse rate on the radial artery.

There are two methods for the assessment of the deficiency of the pulse:

(1) It is performed by one physician. The doctor simultaneously counts a heart rate during the auscultation at the apical impulse, and palpates the radial artery pulse by the other arm and counts the pulse rate during 1 minute ;

(2) It is performed by two physicians. The first physician auscultates the heart and counts the heart rate within 1 minute, and the second physician counts the pulse rate on the radial artery at the same time.

Deficiency of the pulse is typical of atrial fibrillation.

8.6. Measuring arterial pressure (taking blood pressure, taking BP)

To measure blood pressure (BP), there are widely used 2 indirect (non-invasive) methods: a manual method and an oscillometric method. Indirect methods are based on collapsing the artery by an external cuff.

The ***manual method*** was developed in the St. Petersburg clinic of M. V. Yanovsky by the Russian surgeon N. S. Korotkoff in 1905. To measure the blood pressure, a simple device consisting of a mechanical manometer, a cuff with a rubber bubble and a phonendoscope is used. The method is based on full compression of the cuff of the brachial artery and listening to the tones arising from the slow release of air from the cuff (Korotkoff tones). The manual blood pressure cuffs have either mercury or aneroid sphygmomanometers (Fig. 8-12, Table 8-4).

The ***oscillometric method*** can be performed using the manual cuff with an automated oscillometric device. The oscillometric devices obtain the pres-

sure measurement by detecting oscillations on the lateral walls of the occluded artery as the cuff is deflated. Automated oscillometric blood pressure measuring devices are now more commonly used due to their ease of use and availability.

Table 8-4. Phases of Korotkoff tones (sounds)

Phases	Characteristics
I phase	Tones appear and grow when blowing off the cuff. Systolic BP is determined by the first tone (sound).
II phase	Appearance of noise in the further deflation of the cuff
III phase	The sound grows and resembles a crunch
IV phase	Sharp muted soft "blowing" sound, turning into tones[1].
V phase	Disappearance of the last tone. Diastolic BP is determined by the last tone (sound)[2]

Note: 1 - IV phase correlates as alternate measure of diastolic BP. IV phase is used for registration the diastolic BP if the pressure upon appearance of phase IV is ≥10 mmHg than the pressure at phase V.

2- It is the standard level of diastolic BP.

(1) (2) (3)

Fig. 8-12. Types of sphygmomanometers:
1 - aneroid, 2- mercury, 3 - electronic oscillometric

Rules of taking BP

Step I. Properly prepare the patient:

1. Have the patient relax, sitting in a chair (feet on floor, back supported) for >5 min. 2. The patient should avoid caffeine, exercise, and smoking for at least 30 min before measurement. 3. Ensure patient has emptied

his/her bladder. 4. Neither the patient nor the observer should talk during the rest period or during the measurement. 5. Remove all clothing covering the location of cuff placement. 6. Measurements made while the patient is sitting or lying on an examining table do not fulfill these criteria.

Step II. Use proper technique for BP measurements:

1. Use a BP measurement device that has been validated, and ensure that the device is calibrated periodically. 2. Support the patient's arm (eg, resting on a desk) (Fig. 8-13, 8-14). 3. Position the middle of the cuff on the patient's upper arm at the level of the right atrium (the midpoint of the sternum). 4. Use the correct cuff size, such that the bladder encircles 80% of the arm, and note if a larger- or smaller-than-normal cuff size is used. 5. Either the stethoscope diaphragm or bell may be used for the auscultatory readings.

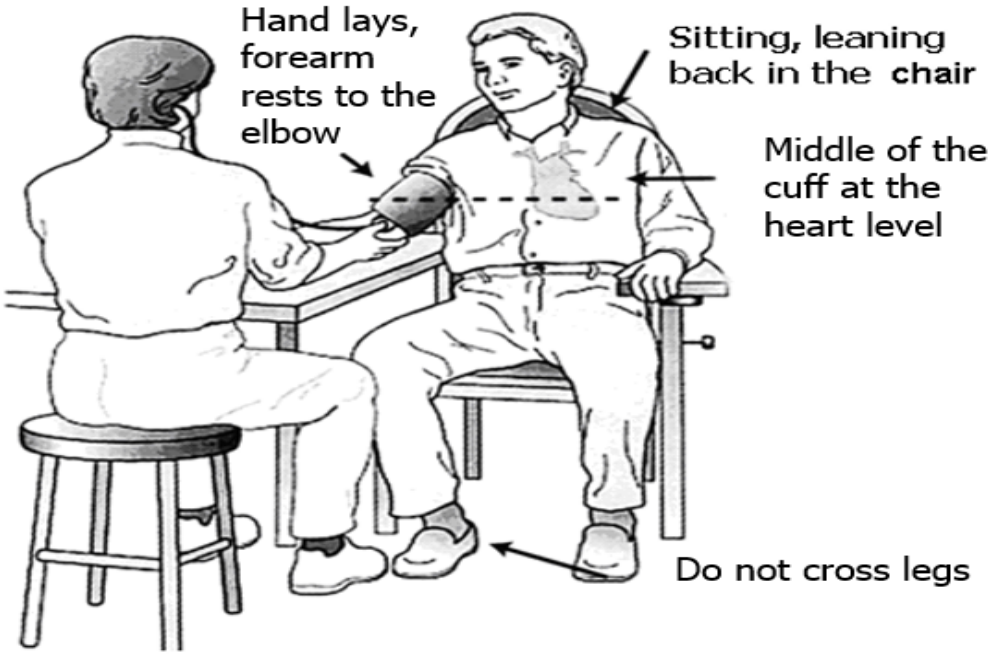


Fig. 8-13. Position of the patient during the blood pressure measurement

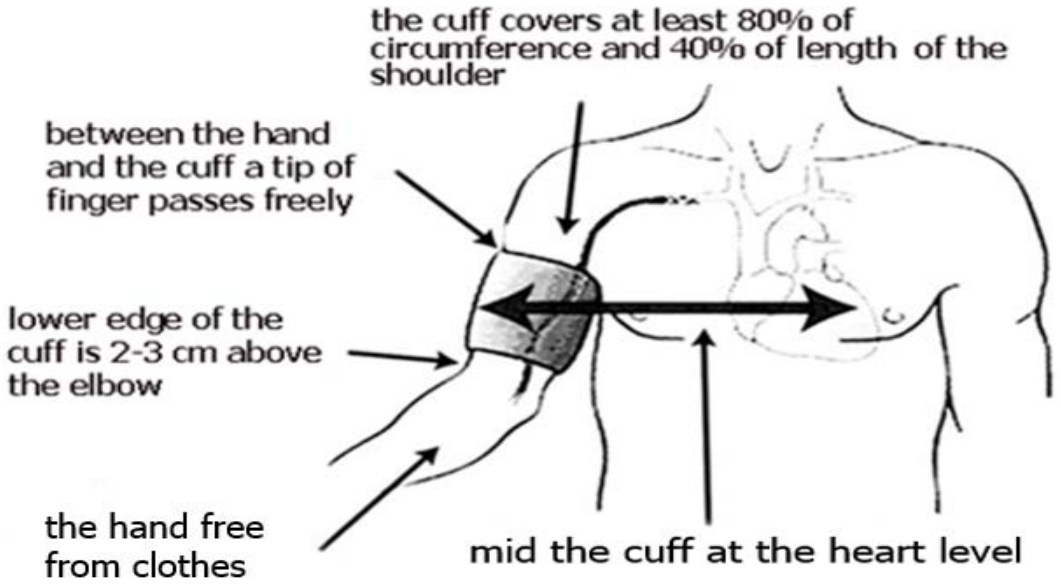


Fig. 8-14. Position of the cuff during the blood pressure measurement

Step III. Take the proper measurements needed for diagnosis and treatment of elevated BP:

1. At the first visit, record BP in both arms. Use the arm that gives the higher reading for subsequent readings. 2. Separate repeated measurements by 1–2 min. 3. For auscultatory determinations, use a palpated estimate of radial pulse obliteration pressure to estimate SBP (systolic BP). Inflate the cuff 20– 30 mm Hg above this level for an auscultatory determination of the BP level. 4. For auscultatory readings, deflate the cuff pressure 2 mm Hg per second, and listen for Korotkoff sounds.

Step IV. Properly document accurate BP readings:

1. Record systolic blood pressure (SBP) and diastolic blood pressure (DBP). If using the auscultatory technique, record SBP and DBP as onset of the first Korotkoff sound and disappearance of all Korotkoff sounds, respectively, using the nearest even number.

The phenomenon of "infinite tone" is the continuation of tones to the zero scale of the manometer. It is observed in patients with a large cardiac out-put (in pregnant women, with fever, anemia, aortic insufficiency). In this case, the value of DBP corresponds to the start of phase IV Korotkoff sounds.

2. Note the time of most recent BP medication taken before measurements.

Step V. Average the readings:

Use an average of ≥2 readings obtained on ≥2 occasions to estimate the individual’s level of BP.

BP measurement is performed at least 2 times at the intervals of not less than one minute. If the difference in blood pressure more than 5 mm Hg

or in severe arrhythmia the extra dimension needs. Estimate the average value of the last two measurements.

Step VI. Provide BP readings to patient:

Provide patients the SBP/DBP readings both verbally and in writing.

Diagnostic value of blood pressure

According to classification of the BP levels (WHO, 1999; European Society of Cardiology (ESC) and European Society of Hypertension (ESH, 2018) there are the following categories of BP (Table 8-5):

According American College of Cardiology/American Heart Association (ACC/AHA) guidelines (2017) in adults, BP is categorized into 4 levels on the basis of average BP measured in a healthcare setting (office pressures): normal, elevated, and stage 1 or 2 hypertension (Table 8-6). Both the classifications above are based on the average of two or more readings taken at each of two or more visits after initial screening.

BP may be physiologically elevated in healthy people in physical and emotional strain. *Basal BP* is taken in the morning, after night sleep, on an empty stomach.

Table 8-5. Categories of blood pressure (WHO, 1999; ESC/ESH, 2018)

Categories of BP	SBP		DBP
Normal BP:	100-139 mm Hg	and	60-89
Optimal	<120 mm Hg	and	<80 mm Hg
Normal	120 – 129 mm Hg	and/or	80-84 mm Hg
High normal	130-139	and/or	85-89 mm Hg
Hypertension:	≥140 mm Hg	and/or	≥90 mm Hg
I degree	140-159 mm Hg	and/or	90-99 mm Hg
II degree	160-179 mm Hg	and/or	100-109 mm Hg
III degree	≥180 mm Hg	and/or	≥110 mm Hg
Isolated systolic /diastolic arterial hypertension	≥140 mm Hg	or	≥90 mm Hg
Arterial hypotension	<100 mm Hg (males) <95 mm Hg (females)	and	<60 mm Hg

Table 8-6. Categories of blood pressure (ACC/AHA, 2017)

Categories of BP	SBP		DBP
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120 – 129 mm Hg	and	<80 mm Hg
Hypertension			

Stage 1	130-139 mm Hg	or	80-89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

Systolic BP is over 139 mm Hg is called *systolic arterial hypertension*. *Isolated systolic arterial hypertension* occurs in the aortal valve insufficiency, aortae atherosclerosis, and thyrotoxicosis. Diastolic pressure is over 89 mm Hg is called *diastolic arterial hypertension*. *Isolated diastolic arterial hypertension* may be in renal diseases, post-infarction myocardiosclerosis, high sodium diet. Combined elevation both systolic and diastolic BP is the most typical situation in clinical practice: essential arterial hypertension is near 95% of a total number of arterial hypertension.

Arterial hypotension is typical in acute circulatory failure (collapse, shock), adrenal glands insufficiency, autonomic nervous system dysfunction. Mild physiological arterial hypotension (not less than 80/60 mm Hg) occurs sometimes in healthy adults at the rest, but at physical and emotional strain BP elevates at them.

Features of blood pressure measurement

The first visit BP measurement is performed on both hands, the difference in this case must be no more than 10 mm Hg. The measurement is carried out in the future on the non-working arm. It is impossible to measure the BP on the hand by a mastectomy, paralyzed or weaker hand after a brain stroke.

When expressed *asymmetry of BP* the higher value is more representative, and the hand that has the higher BP must be used for subsequent measurement of the blood pressure.

Measure the BP while the patient is in a standing position especially in the event that the patient experiences dizziness on standing. If a fall of 20 or more millimeters of mercury occurs in systolic BP or 10 or more in diastolic BP, the patient is considered to have an *orthostatic hypotension*, an abnormally great fall in blood pressure with standing.

The elderly and people with diabetes mellitus need to measure BP in a lying position and standing, as they have a high probability of orthostatic hypotension (decrease in arterial pressure, duration of more than 3 minutes).

Pseudohypertension in the elderly and *Osler's sign of pseudohypertension* is a falsely elevated BP reading obtained through sphygmomanometry due to the calcified blood vessels which do not compress with pressure. There is normal BP when it is measured from within the artery. This condition however is associated with a significant cardiovascular disease risk. Because the stiffened arterial walls of arteriosclerosis do not compress with pressure normally, the BP reading is theoretically higher than the true intra-arterial measurement.

To perform the *Osler's sign* test, a physician first inflates the BP cuff above systolic pressure to obliterate the radial pulse. Then the physician attempts to palpate the radial artery, there is a positive test if it remains palpable as a firm "tube".

Blood pressure on the thigh is determined similarly to the measurement on the shoulder. BP is measured at the thigh must be higher than the pressure measured at the shoulder. The ratio decreases with coarctation of the aorta.

Pregnant women need to measure BP in the reclining position. When receiving high numbers of BP at the doctor's office, home independent measurements help to identify the "white coat effect" in 30% of pregnant women (emotional reaction to the medical measure).

White coat hypertension, more commonly known as *white coat syndrome* (close term "*office hypertension*") is a phenomenon in which patients exhibit a BP level above the normal range in a clinical setting, though they do not exhibit it in other settings. *White coat hypertension* can be defined as the presence of a defined hypertensive average blood pressure in a clinic setting, although it is not present when the patient is at home.

8.7. The key points on the theme "Subjective and Objective Examination of Patients with Diseases of the Cardiovascular System"

The main complaints are most typical for the cardiovascular system diseases: *mixed dyspnea* in circulatory failure that increases with physical exercise and in a horizontal position of the patient; *pressing chest pain* behind sternum in ischemic heart disease; *heart palpitations and intermissions* in cardiac arrhythmia, *edema on the inferior extremities* (in a heart failure), *cough* (*usually) dry and hemoptysis* together with dyspnea, and as a rule, under load or in a horizontal position.

Patients may have additional complaints on the background of the heart failure or arterial hypertension: *weakness, fatigue, sweating, and sleep disorder, headache, dizziness, noise in the head.*

General survey in circulatory failure demonstrates typically a sitting or semi-sitting position (*orthopnea*), *acrocyanosis*, and *symmetrical edema on the lower extremities*. *Paleness of the skin* is typical in the arterial hypotension, *hyperemia* (redness) of the skin – in arterial hypertension. The skin *colour of "milk-and- coffee"* with hemorrhagic spots may occur in infective endocarditis.

Survey and palpation of the heart area can examine ***apical impulse***, formed by the left ventricle contractions. Normally, the apical impulse localization is 1.5 cm inside of the left midclavicular line in the fifth intercostal space. Down- and left shift of the apical impulse presents in the left ventricle hypertrophy.

Cardiac impulse is a pathological pulsation of the enlarged right ventricle. It is observed sometimes synchronous with the apical impulse at the IV-V-th interspaces near the left edge of the sternum.

Chest thrill is palpated above the apex of the heart during diastole in mitral stenosis and over the aorta during systole in aortic stenosis.

Normally, the radial artery pulse is symmetrical on both arms, rhythmic, 60-90 in 1 minute, of satisfactory filling and resistance.

Pathological changes of the pulse: asymmetrical pulse *(pulsus differens)* - unilateral abnormalities of the peripheral arteries*;* arrhythmical pulse *(pulsus irregularis, s. arrhythmicus)* – in a heart arrhythmia*;* large pulse when increased filling and resistanse (*pulsus magnus, or pulsus altus)* – in the aortic valve insufficiency, thyrotoxicosis, and fever*;* a small size of the pulse *(pulsus parvus)* – in the aortic valve stenosis, tachycardia, acute heart failure*;* extremely small *thready-like pulse (pulsus filiformis)* – in shock and a massive blood loss*; deficiency of* pulse (difference between the rate of the pulse and the rate of heart beats) – *in atrial fibrillation.*

According to classification of the BP levels (WHO, 1999; ESC/ESH, 2018): *normal blood pressure* - 100/60 - 139/89 mm Hg*; arterial hypertension* - 140/90 mm Hg and over*; arterial hypotension* - less than 100/60 mm Hg.

8.8. Assessment tests on the theme "Subjective and Objective Examination of Patients with Diseases of the Cardiovascular System"

1. The symptoms of dyspnea in cardiovascular disease are:
1. intensification in a horizontal position;
2. intensification at physical exercises;
3. decrease upright;
4. decrease in a horizontal position;
5. intensification upright.

2. Anasarca is:
1. swelling of the entire body with the simultaneous accumulation of fluid in the serous cavities;
2. swelling feet and ankles;
3. loss of weight;
4. severe bleeding;
5. exudate in serous cavities.

3. Main complaints in cardiac arrhythmias are:
1. interruptions in the heart, palpitations;
2. feelings of fear, heart fading;
3. chest pains;
4. dyspnea;

5. weakness, dizziness.

4. The patient complains of intensive pressing pain behind the sternum. The pain appeared on the background of physical exercises. Pain lasts 50-60 minutes and is not stopped by Nitroglycerinum. What is it with the patient?
1. myocardial infarction;
2. myocarditis;
3. angina pectoris (stenocardia);
4. pleurisy;
5. thoracalgia.

5. Moderate pressing pains behind sternum appeared at the time of the physical exercises. Pains last 5-10 minutes and are stopped by Nitroglycerinum. What is it with the patient?
1. angina pectoris (stenocardia);
2. myocardial infarction;
3. myocarditis;
4. esophagitis;
5. myositis.

6. Patient has visible pulsation in epigastric region, it is closer to xiphoid process and better visible in a vertical position and intensifies at deep inspiration. What is it?
1. pulsation of abdominal aorta;
2. pulsation of liver;
3. pulsation of right ventricle;
4. pulsation of right atrium;
5. pulsation of vena cava inferior.

7. Forced position of a patient ("orthopnea") is typical in:
1. angina pectoris (stenocardia);
2. arterial hypertension;
3. left ventricle heart failure;
4. respiratory failure of obstructive type;
5. arterial hypotension,

8. Normal localization of the apical impulse is:
1. V-th intercostal space;
2. 1-1.5 cm outwards from the left midclavicular line;
3. VI-th intercostal space;
4. VII-th intercostal space;
5. 1-1.5 cm inside from the left midclavicular line.

9. Normal area (width) of apical impulse is:
1. 3-4 sq. cm;
2. 1-2 sq. cm
3. 2.5-3.5 sq. cm;
4. 0.5-1.5 sq. cm;
5.4-6 sq. cm.

10. Normal height of apical impulse is:
1. moderate (satisfactory);
2. high;
3. low;
4. 0.5-1.5 cm;
5. 2-3 cm.

11. The normal heart rate (in adults) is:
1. 60-90 per minute;
2. less than 60 beats per minute;
3. over 90 per minute;
4. 50-80 per minute;
5. 72 per minute.

12. Pulse properties determined by palpation include:
1. filling (volume);
2. rhythm;
3. resistance;
4. frequency;
5. size.

13. Mild pulse (pulsus mollis) occurs in:
1. arterial hypotension
2. acute bleeding
3. mitral stenosis
4. arterial hypertension
5. atherosclerosis of peripheral blood vessels

14. Small pulse characteristic is:
1. small filling and resistance;
2. satisfactory filling and resistance;
3. the pulse filling is determined with difficulty;
4. the pulse filling is not detected.

15. “Pulsus differens” is:
1. difference between heart beats and arterial pulsation;

2. difference between arterial and venous pulsation;
3. difference between filling amplitude of the left and right radial arteries pulsations;
4. difference between a pulse rate on the left and right radial arteries;
5. varying pulse rate and filling and resistance of the pulse on the left and right and left radial arteries.

16. Normal levels of the BP is:
1. 100/60-139/89 mm Hg;
2. 160/100-179/109 mm Hg;
3. 140/90-159/99 mm Hg;
4. <180/110 mm Hg;
5. 120/80 mm Hg exclusively.

17. Pulse pressure is:
1. maximum systolic blood pressure;
2. minimum systolic pressure;
3. maximum diastolic pressure;
4. minimum diastolic pressure;
5. difference between systolic and diastolic pressure.

18. Blood pressure should be measured:
1. 1-2 hours after meal;
2. 1 hour after drinking coffee;
3. 1 hour after smoking;
4. 15 min after physical exercise;
5. all these factors do not matter.

19. Blood pressure should be measured:
1. on the right hand;
2. on both hands;
3. on the hand by side of mastectomy;
4. on paralyzed or weak hand after brain stroke;
5. on non-working hand.

20. To identify orthostatic hypotension, blood pressure is measured necessarily supine and standing in:
1. elderly patients;
2. patients with gastric ulcer;
3. patients with diabetes mellitus;
4. pregnant women;
5. COPD.

Chapter 9. Percussion of the Heart

Goals: to enable students to learn -

1) purposes, rules and diagnostic value of heart percussion;
2) position of the relative heart dullness borders in the norm and in pathology;
3) types of the heart configurations, technique of heart contours percussion, and their diagnostic value;
4) width of vascular bundle of the heart: technique of percussion, and diagnostic value;
5) transverse length of the relative heart dullness: technique of percussion, and diagnostic value;
6) borders of absolute heart dullness in the norm and in pathology: technique, and diagnostic value.

9.1. Conception of heart (cardiac) dullness

Percussion is used to determine sizes, position and configuration (shape) of a heart.

Absolute heart dullness is the part anterior surface of the heart, which is directly adjacent to the chest wall, gives a completely dull sound at percussion. It is formed by part of the anterior wall of the right ventricle.

Relative heart dullness is the anterior surface of the heart, covered by the lungs. It gives a hyporesonant kind of dull sound at percussion (Fig. 9-1).

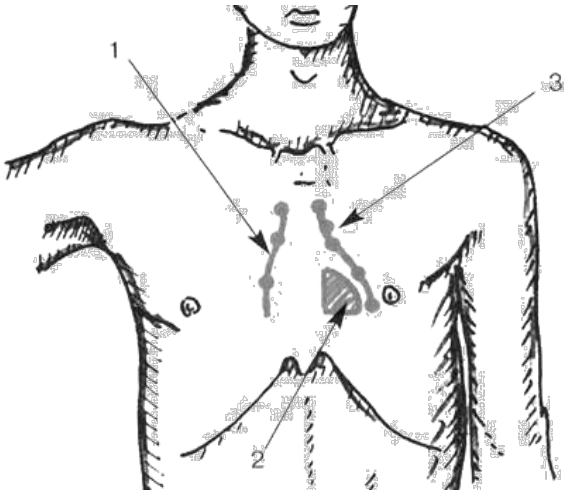


Fig. 9-1. Relative and absolute dullness of the heart:
(1) right contour of relative dullness of the heart; (2) area of the absolute dullness of the heart; (3) left contour of relative dullness of the heart

Rules of the heart percussion:

1. Percussion is performed in most cases in a vertical position of the patient, with the arms lowered downwards, at impossibility of keeping this rule

it is possible to do percussion in a horizontal position.

2. The doctor can sit or stand to the right of the patient at the time of percussion.

3. Respiration of the patient should be superficial.

4. The finger-plexímeter (3-d finger of the left arm) must be densely applied to intercostals spaces. The finger-plexímeter is always parallel to the supposed border percussing strictly by intercostal spaces.

5. Percussion is conducted from a resonant sound to the hyporesonant sound (for relative heart dullness) or the dull sound (for absolute dullness) depending on the purpose of percussion (that is from the lungs to the heart).

6. The revealed border of the heart dullness is marked on outside edge of the finger-plexímeter inverted to a louder percussion sound.

7. The strength of percussion stroke depends on the purpose of percussion: *at delimitation of relative heart dullness, the medium (quiet, or light) percussion is used; at delimitation of absolute heart dullness - the quietest percussion.*

8. The sequence of percussion:

1) *Delimitation of relative heart dullness,*
2) *Delimitation of heart configuration*
3) *Delimitation of transverse length of the relative heart dullness,*
4) *Delimitation of size of the heart vascular bundle,*
5) *Delimitation of absolute dullness of the heart.*

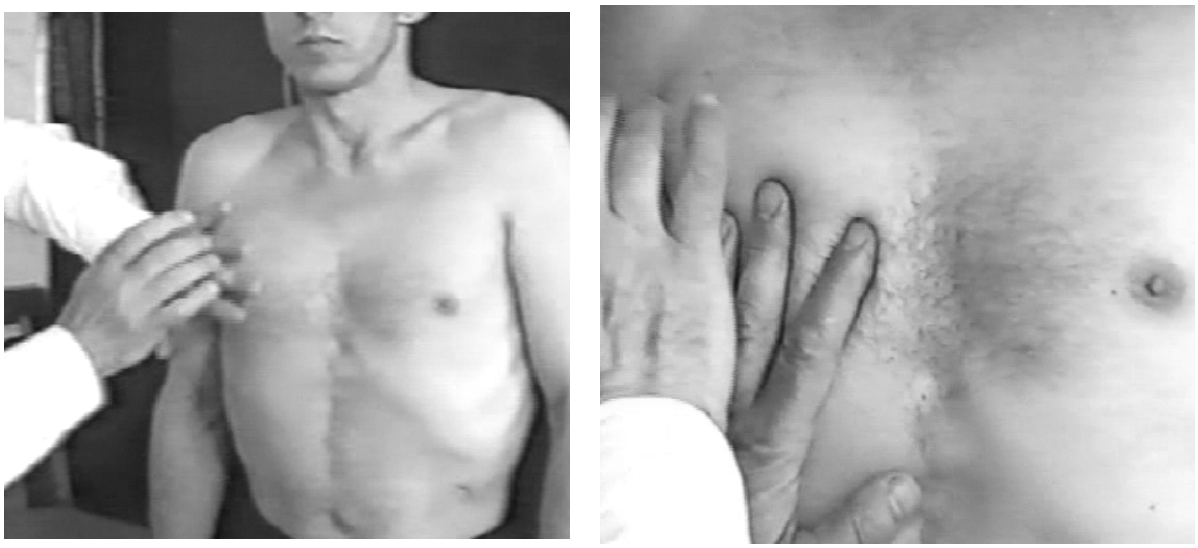
9.2. Delimitation of relative heart dullness

It is distinguished right, left and superior borders of relative dullness of the heart.

Percussion of the right border and the right contour of the relative heart dullness

At the beginning, a physician-examiner does percussion of the inferior border of the right lung by the midclavicular line [the same determining position of the cupula (dome) of diaphragm] *to avoid from the liver dull sound on two interspaces upper*.

For this purpose, the finger-plexímeter is applied at the level of II intercostal space on the right midclavicular line, and percussion is performed strictly on intercostals spaces downwards by quiet percussion before change of resonant sound on a dull sound. The mark is made on the edge of the finger-plexímeter inverted to a side of clear pulmonary sound (Fig. 9-2), in norm at the VI intercostal space (inferior edge of VI rib).



(1) (2)

Fig. 9-2. Percussion of the right border of the relative heart dullness: (1) 1-t step – percussion of the inferior border of the lung on the right midclavicular line; (2) 2-d step – percussion of the right border of the relative heart dullness

Next, the right border of relative dullness of the heart can be defined on two interspaces upper at the IV intercostal space. The finger-pleximeter is a vertical and is moving from the right midclavicular line in the direction to the sternum with light percussion to transmission from clear pulmonary to hyporesonant kind of dull sound. *The border is marked on the side of resonant sound, in the norm 1 cm externally of the right edge of the sternum at the IV interspace* (Table 9-1). In case of a change of height of standing of a diaphragm the rules of percussion for the definition of the right border of relative dullness are not variated.

In order to the *definition of the right contour of the heart* the finger - pleximeter is located in the III and II intercostal spaces at the level of midclavicular line parallel to the sternum (*parallel to a finding border of the heart in this intercostals space*). Quiet percussion is made in direction to the sternum with light percussion to transmission from clear pulmonary to a hyporesonant kind of dull sound.

Table 9-1. Normal position of relative heart dullness

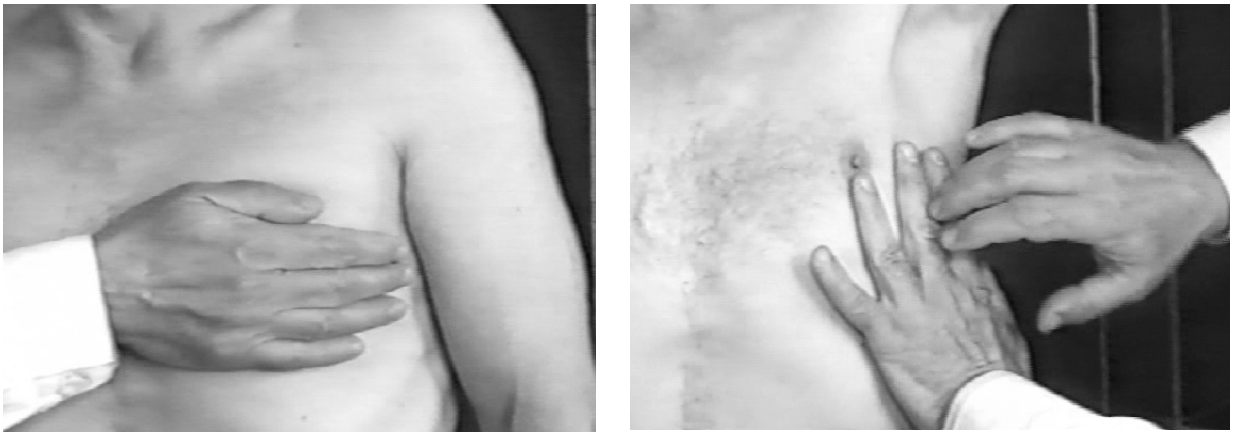
Border/ Countour	Position	Anatomical structure
Right – IV-th interspace	1 cm laterally of the right edge of the sternum	right atrium
Right – II-d - III-d interspaces	0.5 - 1cm laterally of the right edge of the sternum	superior vena cava and an ascending aortic arch
Left – V-th	1-1.5 cm medially of the left	left ventricle

interspace	midclavicular line	
Left – IV-th interspace	more medially than in V-th interspace	left ventricle
Left – III-th interspace	on the middle between mid-clavicular and parasternal lines	left auricle
Left – II-th interspace	0.5 - 1cm laterally of the left edge of the sternum	left part of an aorta arch and a pulmonary trunk
Superior	on the upper edge of III-d rib at the left parasternal line	cone of a pulmonary artery and left auricle

Further, the points received at a percussion in the IV, III, and II intercostal spaces are connected among themselves to the representation of the right contour of the heart. *The right contour of the heart is formed at the II intercostal space by superior vena cava and ascending aortic arch, and at the III-IV intercostal spaces by the right atrium.*

Percussion of the left border and the left contour of the relative heart dullness

The left border of the relative heart dullness would be examined in the interspace of the apical impulse localization (normally at V interspace). That is why, a previous step before the percussion is palpation of the apical impulse. Percussion starts from the anterior axillary line in the direction to the sternum with the finger-pleximeter, applied to the interspace vertically, a collateral finding border (Fig. 9-3). The l*eft border of the relative heart dullness is placed normally at V intercostal space on 1-1.5 cm medially from the midclavicular line (see Table 9-1).*



(1) (2)

Fig. 9-3. Percussion of the left border of the relative heart dullness: 1) 1-t step – apical impulse palpation; (2) 2-d step – percussion of the left border.

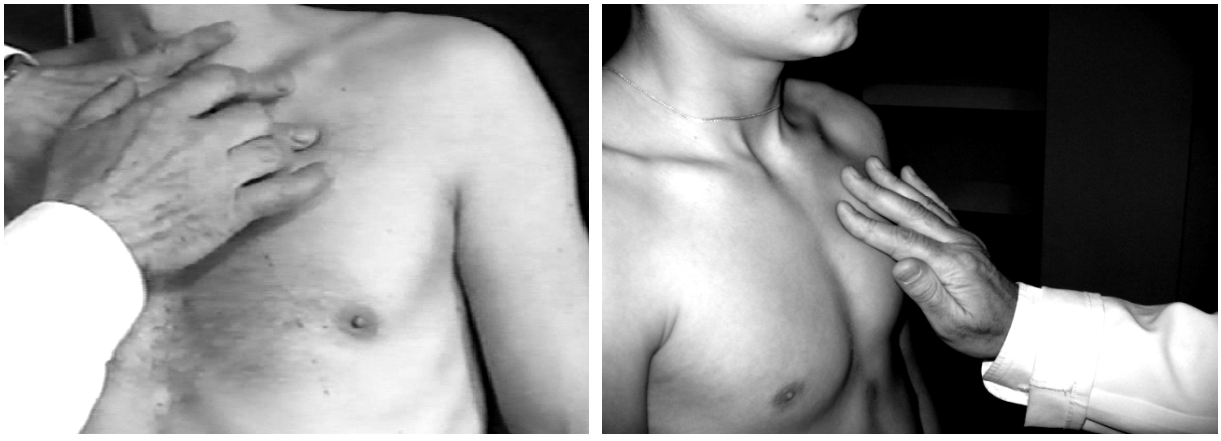
In order to the *definition of the left contour of the relative heart dullness*, the finger-pleximeter is raised on one intercostals space above, the finger-pleximeter position in the IV intercostal space is parallel to the sternum at the level of the anterior axillary line, and the percussion is performed before the change of a resonant sound on a hyporesonant kind of the dull sound. The point is marked from the side of the resonant sound. Percussion in the III intercostal space is performed by the same rules. Later the left border of the heart vascular bundle in the II intercostal space is defined by the percussion from the midclavicular line to the sternum before the change of the resonant sound on the hyporesonant sound. The points received by means of percussion in the V, IV, III, and II intercostal spaces are connected representing the left contour of the heart.

The left contour of the heart is formed in the II intercostal space by the left part of the aorta arch and the pulmonary trunk, at the III intercostal space – the left auricle, and lower (IV-V interspaces) – the left ventricle.

Percussion of the superior border of the relative heart dullness

Superior border of the relative heart dullness is examined on the left parasternal line. The finger-pleximeter applied perpendicular to the sternum is moving down with a quiet percussion from I intercostal space (Fig. 9-4). Normally, a hyporesonant kind of the dull sound should be found in III interspace.

For accurate determination of the superior border, the direct percussion (Obraztcov method) is performed on two overlying ribs above a hyporesonant sound (first – the 2-d control rib, then – the 3-d test rib). If the percussion by the ribs yields an identical note, the border is placed on the inferior edge of the lower (the 3-d) rib. If the hyporesonant percussion sound is found above the lower rib, the superior border is defined on the upper edge of this rib.



(1) (2)

Fig. 9-4. Percussion of the superior border of the relative heart dullness:

(1) 1-t step – percussion by interspaces on the left parasternal line; (2) 2-d step – immediate percussion by two ribs overlying the dulling interspace.

The normal superior border of the relative heart dullness is located at the level of the upper edge of the 3-d rib and is formed by a cone of the pulmonary artery and the left atrium.

Enlargement of the relative heart dullness is observed in:

- an elevated position of the diaphragm (in hypersthenic body-build type, meteorism, ascites, pregnancy);
- in hypertrophy and dilatation of the right auricle and the right ventricle (in stenosis and insufficiency of tricuspid valve, stenosis of ostium of the pulmonary artery, sclerosis of the pulmonary artery, the development of the pulmonary heart, mitral stenosis) the borders of the heart are displaced to the right;
- as a result of the hypertrophy and dilatation of the left ventricle (in arterial hypertension, aortal valves insufficiency and stenosis, aneurysm of the left ventricle) the borders of the heart are displaced to the left;
- as a result of the hypertrophy of the left atrium (mitral stenosis and insufficiency of the mitral valve) the borders of the heart are displaced upwards;
- as a result of combined heart valves diseases the enlarging of the dimensions of the heart is observed in all directions.

Restriction of the relative heart dullness is observed in**:**

- phrenoptosis (descent position of a diaphragm in asthenic constitution, at the general enteroptosis);
- pulmonary emphysema.

9.3. Heart configuration

Heart configuration is formed by the right and the left contours of the relative heart dullness. The h*eart configuration* can be determined by percussion of the right contour of the relative heart dullness in 4-th, 3- and 2-d intercostal spaces, and percussion of the left contour of the relative hart dullness in the 5-th, 4-th, 3- and 2-d intercostal spaces. The heart configuration is changed in pathology (Table 9-2).

Table 9-2. Types of the heart configuration

Type	Characteristics	Causes
Normal configuration	normal position of the right and the left contours of the relative heart dullness	
	narrowing of contours of relative heart dullness at the 3-d interspace (“waist of heart”)	
Aortic	significant angle formed by vascular	aortic insufficiency;

configuration	bundle	aortic stenosis
	hypertrophy and dilation of the left ventricle	
Mitral configuration	smooth “waist of heart” because of enlarged left atrium	mitral insufficiency; mitral stenosis
Trapezoidal (triangle) configuration	considerable widening of the right and left borders of the relative heart dullness	exudative pericarditis; hydrothorax
	borders of absolute and relative dullness are almost indistinguishable	
Spheroidal configuration ("cor bovinum“)	enlargement of the relative heart in all directions	combined heart valves diseases; congenital heart disease; dilated cardiomyopathy; diffuse cardiosclerosis

Normal heart configuration is characterized by a normal position of the right and left contours of the relative heart dullness and narrowing of the contours of relative heart dullness at the III-d interspace (“*waist of heart*”) (see Fig. 9-1).

Mitral configuration is determined in the mitral valve disease (mitral valve stenosis and insufficiency) by increasing the left atrium and smoothing the “waist of the heart”.

The heart accepts *aortic configuration* in defects of the aortic valve, and also at the expressed arterial hypertension, at the expense of the enlarged left ventricle, and underlining of the “waist of heart”.

Trapezoidal (triangle) configuration appears as a result of accumulation of large amounts of fluid in the pericardium.

Spheroidal configuration ("cor bovinum", or “bull's heart”) is a sharp shift in the borders of the heart in all directions. This happens at increase of all parts of the heart (combined heart valves disease, ischemic heart disease).

9.4. Transverse length of the relative heart dullness and width of the vascular bundle of the heart

Setting the borders of the relative heart dullness, *transverse length of the heart* is measured. To do this, from the extreme points of the right and left borders of the relative cardiac dullness, the perpendiculars are lowered to the anterior median line and measured with a centimeter tape (Fig. 9-5). Normally, the right perpendicular is 3-4 cm, the left-8-9 cm. Thus, the total transverse size of the relative cardiac dullness in the norm is 11-13cm.

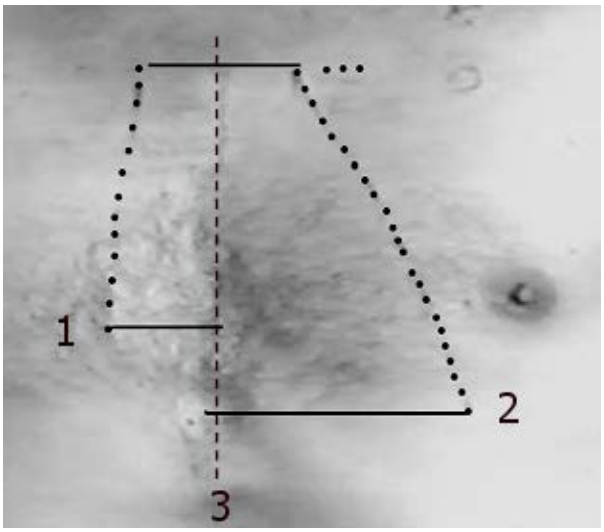
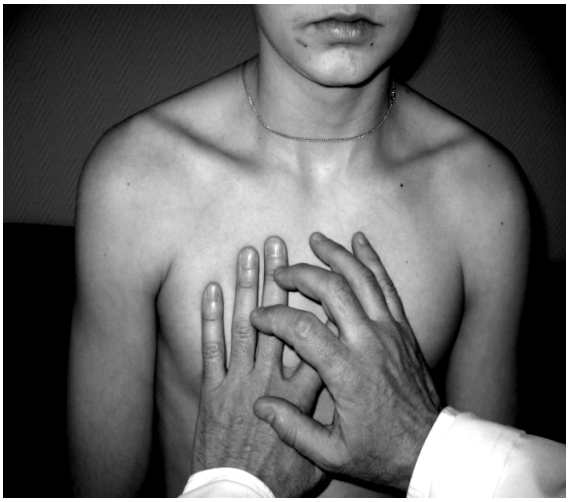
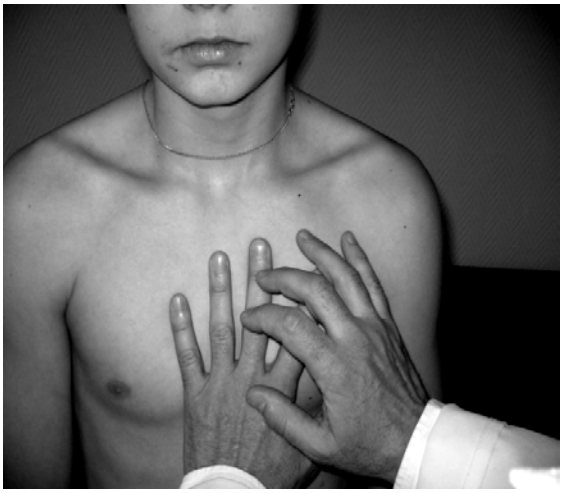


Fig. 9-5. Measuring transverse length of the relative heart dullness:
(1)- right border of the relative heart dullness in the 4-th intercostal space;
(2) – left border of the relative heart dullness in the 5-th intercostal space;
(3) – l. mediana anterior.

Vascular bundle of the heart is formed: on the – by the cava vein and an ascending part of the aortic arch, on the left – by the pulmonary artery and a part of the aortic arch. Percussion is produced in the II intercostal space on the right and left in the direction from the midclavicular line to the sternum, using a quiet percussion. When percussion gives a hyporesonant kind of the dull sound, a mark is made on the outer edge of the finger-pleximeter (Fig. 9-6).



(1)



(2)

Fig. 9-6. Percussion of the width of a vascular bundle of the heart:
(1) - on the right; (2) – on the left.

The right and left borders of the vascular bundle are located at the edges of the sternum. *Normally*, the *width of the vascular bundle of the heart is 4-6 cm*. Expansion of the borders may occur in widening the aorta, pulmonary artery, tumors of the mediastinum.

9.5. Delimitation of absolute (superficial) heart dullness

Absolute (superficial) heart dullness is the part of the anterior wall of the right ventricle heart which is not covered normally by the lungs. Percussion of the anterior wall of the heart not covered by the lungs area produces *the dull sound* and reveals the *absolute heart dullness* (Table 9-3).

Table 9-3. Normal position of absolute heart dullness

Border	Position	Anatomical structure
Right - 4-th interspace	at the left edge of the sternum	right ventricle
Left – 5-th interspace	1.5-2 cm medially of the left midclavicular line	right ventricle
Superior	on the lower edge of 4-d rib at the left parasternal line	right ventricle

To determine the boundaries of absolute dullness of the heart, the percussion of the corresponding border of the relative dullness of the heart (from resonant to hyporesonant kind of dull sound) is carried out previously. Then, it is necessary to continue the quietest percussion until a dull sound is achieved (Table 9-3): *in the norm for the right border - at the left edge of sternum in IV interspace, the left border - 1.5-2 cm inside of midclvicular line in V interspace, superior border - on left parasternal line at the IV interspace.*

For more precise determination of the superior border, the direct percussion (Obraztcov method) is performed on two overlying ribs above the dull sound (first – the III control rib, then - the IV test rib). If percussion by ribs yields an identical note, the border is placed on the inferior edge of a lower (the 4-th) rib. If the dull percussion sound is found above a lower rib, the superior border is defined on the upper edge of the 4-th rib. *In norm, the superior border of the absolute dullness of the heart settles down at the level of the inferior edge of the IV rib.*

All borders of absolute cardiac dullness correspond to the right ventricle in healthy people.

Increased size of the absolute cardiac dullness is observed in a deep expiration, an upper part of the body inclination forward, high position of the diaphragm (in the hypersthenic body-build type, meteorism, ascites, pregnancy),

lung diseases (tumour of the posterior mediastinum, an inferior lobes pneumosclerosis, the left-sided pleural effusion), exudative pericarditis, expressed hypertrophy and dilatation of the right ventricle.

Reduced size of the absolute heart dullness occurs in deep inspiration, a low position of the diaphragm (in asthenic body-build type, visceroptosis), lung diseases (emphysema, bronchial asthma attack, left-sided pneumothorax), air accumulation in the pericardial cavity (pneumopericardium), subcutaneous emphysema in the heart region.

9.6. The key points on the theme “Percussion of the Heart”

Relative dullness of the heart is the anterior surface of the heart, covered by the lungs. It gives a hyporesonant kind of dull sound at percussion.

Right contour of the relative heart dullness is formed bottom to top by the right atrium at the IV intercostal space in the norm (it is the **right border of the relative heart dullness**), III intercostal space - right atrium as well, II intercostal space - superior vena cava and ascending aortic arch. The ***left contour of the relative heart dullness*** is formed bottom to top by left ventricle at the V interspace in the norm (it is the ***left border of relative heart dullness***), IV interspace - left ventricle as well, III interspace - left auricle, II interspace - left part of aorta arch and pulmonary trunk. The ***superior border of relative heart dullness*** is formed by a cone of pulmonary artery and left atrium, and is located at the upper edge of the III rib.

Enlargement of the relative heart dullness is observed in an elevated position of the diaphragm (in hypersthenic body-build type, meteorism, ascites, pregnancy); in hypertrophy and dilatation of heart chambers (ventricles and atriums); in diffuse pneumosclerosis. ***Restriction of the relative dullness of the heart*** is observed in phrenoptosis (descent position of a diaphragm in asthenic body-build type, at the general enteroptosis); pulmonary emphysema.

Heart configuration is formed by the right and the left contours of the relative heart dullness. ***Normal heart configuration*** is characterized by a normal position of the right and the left contours of the relative heart dullness and narrowing of contours of the relative heart dullness at the III-d interspace (“waist of heart”).

Mitral configuration is determined in mitral valve diseases (mitral valve stenosis and insufficiency) by increasing the left atrium and smoothing the («waist of heart»). ***Aortic configuration*** presents in aortic valve diseases at the expense of increase of the left ventricle, and underlining of the “waist of heart”.

Trapezoidal configuration becomes as a result of accumulation of large amounts of fluid in the pericardium. ***Spheroidal configuration ("cor bovinum", or bull's heart)*** is a sharp shift in the borders of the heart in all directions (in combined heart valves disease, ischemic heart disease).

Absolute dullness of the heart is the part anterior surface of the heart, which is directly adjacent to the chest wall, gives a completely dull sound at

percussion. It is formed by part of the anterior wall of the right ventricle.

An increased size of the absolute heart dullness is observed in deep expiration, upper body inclination forward, high position of the diaphragm (in hypersthenic body-build type, meteorism, ascites, pregnancy), lung diseases (tumour of the posterior mediastinum, inferior lobes pneumosclerosis, left-sided pleural effusion), exudative pericarditis, and in expressed hypertrophy and dilatation of the right ventricle.

A reduced size of the absolute heart dullness occurs in deep inspiration, a low position of the diaphragm (in asthenic body-build type, visceroptosis), lung diseases (emphysema, bronchial asthma attack, left-sided pneumothorax), air accumulation in the pericardial cavity (pneumopericardium), subcutaneous emphysema in the heart region.

9.7. Assessment tests on the theme "Percussion of the Heart"

1. Superior border of the heart relative dullness is on the level of:

1. superior edge of III rib on the left parasternal line;
2. inferior edge of III rib on the left midclavicular line;
3. superior edge of III rib on the right parasternal line;
4. inferior edge of IV rib on the left midclavicular line;
5. at the level of sternal angle on the left parasternal line.

2. Right border of the relative dullness of the heart is:

1. right edge of the sternum;
2. 1.0 cm outward from right edge of the sternum;
3. 1.0-1,5 cm outward from right edge of the sternum;
4. left edge of the sternum;
5. 0.5-1.0 cm outward from left edge of sternum.

3. Left border of the relative heart dullness in the V intercostal space is:

1. 1.0 cm outward from the left outward line;
2. 2.0 cm outward from the left midclavicular line;
3. 2.5 cm inside from the left midclavicular line;
4. left midclavicular line;
5. 1.0-1.5 cm inside from left midclavicular line.

4. Width of vascular bundle of heart in II-d intercostal space is:

1. 5-7 cm;
2. 4-6 cm;
3. 3-5 cm;
4. 1-4 cm;
5. 6-9 cm.

5. Transverse length of relative heart dullness:

1. 11-16 cm;
2. 7-8 cm;
3. 3-5 cm;
4. 10-14 cm;
5. 6-9 cm.

6. Widening the transverse length of the relative heart dullness presents in:
1. right atrium hypertrophy;
2. right ventricle hypertrophy;
3. left ventricle hypertrophy;
4. elevation of diaphragm;
5. left atrium hypertrophy.

7. Restriction of the transverse length of the relative heart dullness presents in:
1. pulmonary emphysema;
2. pneumothorax;
3. elevation of diaphragm;
4. lowering diaphragm;
5. left atrium hypertrophy.

8. Parts of the right contour of the relative heart dullness are:
1. right atrium;
2. vena cava superior;
3. ascending aorta;
4. right ventricle;
5. pulmonary artery.

9. Parts of the left contour of the relative heart dullness are:
1. left atrium;
2. left ventricle;
4. right ventricle;
5. ascending aorta;
5. pulmonary artery.

10. Pathologic types of the heart configuration are:
1. aortic;
2. mitral;
3. trapezoidal;
4. spherical;
5. pulmonary.

11. Absolute heart dullness is formed by:
1. right atrium;
2. right ventricle;
3. left ventricle;
4. left atrium;
5. aorta.

12. Right shift of the right border of the relative heart dullness presents in:
1. right atrium hypertrophy;
2. cor pulmonale;
3. tricuspid valve insufficiency;
4. tricuspid valve stenosis;
5. aneurism of ascending aorta.

13. Left shift of the left border of the relative heart dullness presents in:
1. mitral valve stenosis;
2. right atrium hypertrophy;
3. left ventricle hypertrophy;
4. arterial hypertension;
5. mitral valve insufficiency.

14. Elevation of the superior border of the relative heart dullness presents in:
1. aortal valve insufficiency;
2. aortal valve stenosis;
3. mitral valve stenosis;
4. arterial hypertension;
5. left atrium hypertrophy.

15. Widening the width of the vascular bundle of the heart in II-d intercostal space presents in:
1. aneurism of ascending aorta;
2. diffuse pneumosclerosis;
3. pulmonary emphysema;
4. tumor of superior part of mediastinum;
5. right atrium hypertrophy.

16. Restriction of the width of the vascular bundle width of the heart in II-d intercostal space presents in:
1. pulmonary emphysema;
2. diffuse pneumosclerosis;
3. pneumothorax;

4. aneurism of ascending aorta;
5. pulmonary hypertension.

17. Superior border of the absolute heart dullness on the left parasternal line is:
1. superior edge of III rib;
2. inferior edge of III rib;
3. superior edge of IV rib;
4. inferior edge of IV rib;
5. superior edge of V rib.

18. Right border of the absolute heart dullness in V intercostal space is:
1. right edge of sternum;
2. 0.5-1.0 cm from right edge of sternum;
3. 1.0-1.5 cm from right edge of sternum;
4. left edge of sternum;
5. 0.5-1.0 cm from left edge of sternum.

19. Left border of the absolute heart dullness in V intercostal space is:
1. left midclavicular line;
2. 1.0-1.5 cm inside from left midclavicular line;
3. 1.5-2.0 cm externally from left midclavicular line;
4. 2.5-3.0 cm inside from left midclavicular line;
5. 0.5-1.0 cm externally from left midclavicular line.

20. Widening the absolute heart dullness borders presents in:
1. right atrium hypertrophy;
2. right ventricle hypertrophy;
3. diffuse pneumosclerosis;
4. left ventricle hypertrophy;
5. hydropericardium.

Chapter 10. Auscultation of the Heart

Goals: to enable students to learn –

1) anatomical and physiological bases of the heart auscultation, components of the heart sounds formation;
2) purposes and rules of the heart auscultation, differentiation between I and II heart sounds;
3) diagnostic value of physiological and pathological changes of the heart sounds;
4) diagnostic value of pathological heart rhythms ("gallop rhythm"), opening snap, splitting and duplication of the heart sounds;
5) causes, classification, rules of auscultation and diagnostic value of heart murmurs;
6) differentiation between heart sounds and murmurs, and between organic and functional murmurs;
7) vascular and extracardiac murmurs: technique of auscultation and diagnostic value;
8) auscultation of arteries and veins: technique and diagnostic value.

10.1. Anatomical and physiological bases of the heart auscultation

There are recorded four heart sounds at all humans when conducting phonocardiography.

Two heart sounds (the first and the second) are determined at auscultation in healthy individuals.

The **first sound (S1)** consists of the sound phenomena that occur in the heart during systole, so it is called a **systolic sound**.

The first (systolic) sound is produced by *three components*:

• ***valve component*** – vibration of the intense closed valves of bicuspid (mitral) and tricuspid valves,

• ***muscular component*** – oscillations of the tense muscles of the ventricles during their contraction,

• ***vascular component*** – oscillations of the walls of the proximal parts of the aorta and pulmonary artery when blood enters them from the ventricles.

The **second sound (S2)** consists of *two components*: valve and vascular.

- ***valve component*** is due to the *vibration of the closed semilunar valves of the aorta and pulmonary artery*. Its appearance coincides with the beginning of diastole, so it is called a ***diastolic sound;***
- **vascular component** of II sound is formed because of the proximal parts of the aorta and the pulmonary trunk oscillate at the same time with closing semilunar valves.

In conditions of the pathology, you can listen to the third and fourth heart sounds.

The **third sound (S3**, **protodiastolic sound**) occurs at the beginning of

the diastole (protodiastole) as the sound of the vibrations of the atonic walls of the ventricles (mainly left), when the first portion of the blood hit the ventricles at the beginning of the diastole.

The **fourth sound (S4, presystolic sound)** is due to the reduction of the myocardium of the atria and the impact of the main mass of the blood on the walls of the ventricles in the late diastole (presystole).

Projection of the heart valves

For the correct interpretation of the sound picture of the heart, it is necessary to know the location of the projection of the heart valves on the anterior thoracic wall (Fig. 10-1).

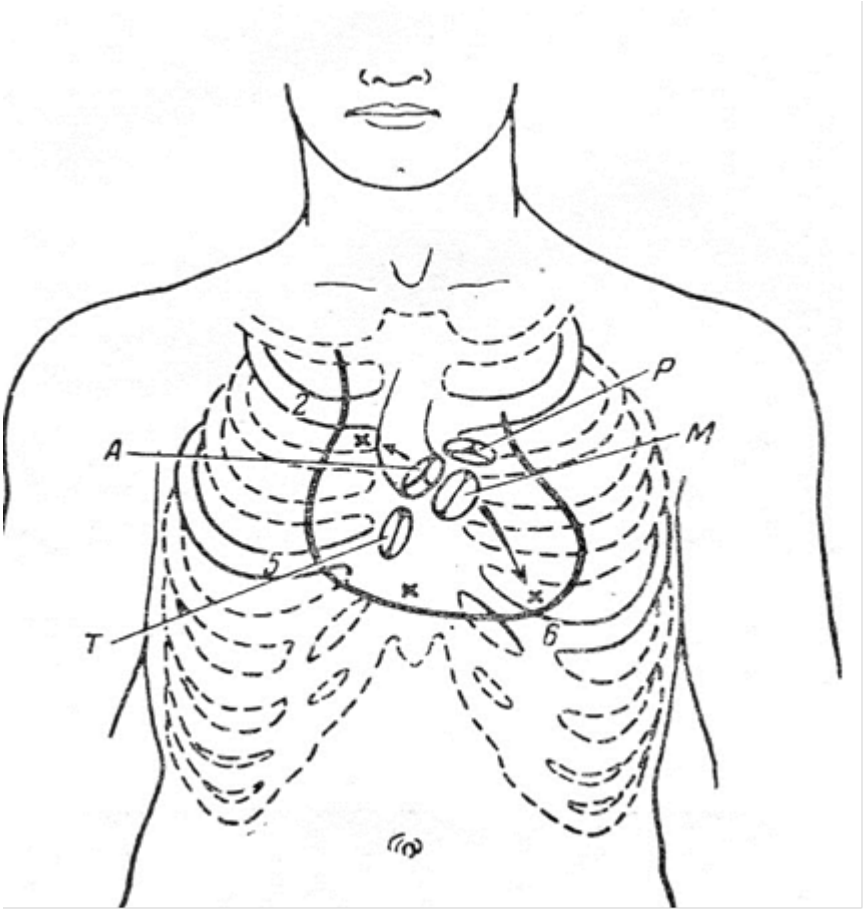


Fig. 10-1. Projection of the heart valves on the chest wall: M - mitral (bicuspid) valve; A - aortic valve; P - pulmonary valve; T - tricuspid valve

Mitral (bicuspid) valve is projected at the point of the attachment of the 3-d left rib cartilage to the sternum.

Aortic valve is at the middle of the sternum at the level of the 3-d costal cartilage.

Pulmonary valve – at the II-d intercostal space on the left 1-1.5 cm from the left edge of the sternum.

Tricuspid valve – at the middle of the line connecting the points of at-

tachment to the sternum of cartilage of the III-d left and V-th right ribs.

Projections of the valves are close to each other, so when listening to in these areas we get a sound picture of several valves (at least two). Therefore, empirically selected point of the best listening to the sounds of the heart (tones and noises), arising on each valve.

Auscultation points do not coincide with the anatomical projection of heart valves on the anterior chest wall.

The best auscultation areas (points) of the heart valves (Fig. 10-2):

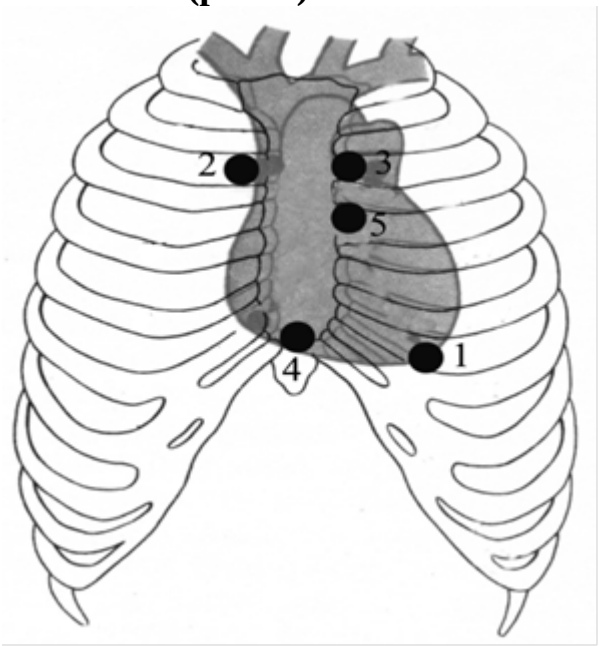


Fig. 10-2. Points of the heart auscultation:
1 - mitral (bicuspid) valve; 2 - aortic valve; 3 - pulmonary valve; 4 - tricuspid valve; 5 - Botkin-Erb's point

(1) *area of the apical impulse* - the best point for auscultation of the mitral valves because the vibrations are transmitted by the firm muscle of the left ventricle during systole;

(2) *II-d intercostal space to the right of the sternum* - the best point for auscultation of the aortic valves where the aorta is the nearest to the anterior chest wall;

(3) *II-d intercostal space, to the left of the sternum* - the best point for auscultation of the pulmonary trunk valves where the anatomical projection these valves onto the chest;

(4) *lower part of the sternum near its junction with the xiphoid process* (the right-ventricular area) - the best point for auscultation of the tricuspid valves;

(5) so-called *Botkin-Erb's point* to the left of the sternum at the III-d and IV-th costosternal joints - for the heart sounds which are associated with closing the aortic and the mitral valves or which can be heard during their affections.

10.2. Rules of the auscultation of the heart

Auscultation of the heart can be carried out using a stethoscope (***mediated auscultation***), as well as pressing the auricle in the projection of the heart (***direct auscultation***).

1. When auscultating the doctor is located on the right side of the patient, so that it is convenient to apply a stethoscope to the places of listening.

2. ***Auscultation is performed in the vertical and horizontal (supine and left side) positions of the patient.***

3. Auscultation sequence: mitral valve, aortic valve, pulmonary valve, tricuspid valve and Botkin-Erb's point (according to the incidence of the heart valves pathology).

4. ***Auscultation is performed before and after the exercise (if the state of the subject allows).*** To do this, the patient is offered to do a few squatting, to climb the stairs, to walk around the ward. This technique makes it possible to distinguish the changes in the auscultative pattern of the organic and functional nature.

5. The patient is offered ***to hold his breath*** after a full expiration to eliminate sound phenomena from the lungs. Auscultation of the heart is also carried out when the breath is held after a deep inspiration.

The first stage of auscultation should always be analytical, dividing the auscultative symptoms into fragments. First, you need to focus on the heart sounds (on the first, then - on the second), then – on the systolic and diastolic pauses.

10.3. Heart sounds

Two heart sounds (systolic and diastolic) are heard well in healthy individuals.

The differences of the first (S1) and second (S2) sounds (Table 10-1):

1. S1 is separated from S2 by a short pause (systole), and the S2 is followed by a longer pause (diastole).

2. Normally, S1 is heard louder than S2 at the apex of the heart (mitral valve) and at the lower third of the sternum (tricuspid valve).

3. S2 is louder than S1 heard at the base of the heart (in the II-d intercostal space to the right and left of the sternum). S2 should be the same volume at these points.

4. S1 is longer and has a lower timbre than S2.

5. S1 coincides with the apical impulse and pulse on the carotid artery. The pulse on the radial artery appears later than S1.

Table 10-1. Differentiation between the first and the second heart sounds

Characteristics	I sound	II sound
Best auscultation area	Apical impulse	Heart base
Relation to cardiac pause	Follows the long pause	Follows the short pause
Duration	0.09-0.12 s	0.05-0.07 s
Relation to apical impulse	Synchronous	Follows the apical impulse
Relation to carotid pulse	Synchronous	Asynchronous

The third (S3) and fourth (S4) heart sounds are heard in pathologic conditions. S3 (protodiastolic sound) is heard on the apical impulse or a few inside of it, better with direct auscultation in the supine position. It can be defined as the shaking of the chest. S4 (presystolic sound) is heard in late diastole (presystole).

10.4. Changes of heart sounds

Changes of heart sounds can be:

- **quantitative:** strengthening (increased intensity), weakening, splitting, or duplication**;**
- **qualitative:** timbre (flapping, dull, metal, velvet), duration;
- **appearance of additional sounds** (“opening snap”, “gallop” rhythm**).**

10.4.1. Quantitative changes of the heart sounds

Increased intensity (amplitude, or loudness) of S1 at the apical impulse is most often found in tachycardia (by reduced filling ventricles with the blood). The most pronounced increase of S1 in the narrowing of the left atrioventricular orifice (mitral stenosis) (Table 10-2). This is due to faster contraction of the left ventricle myocardium due to a decreased blood filling through the narrowed mitral valve orifice during the previous diastole.

Increased intensity of S2 at the II-d intercostal space on the right (S2 accent on the aorta) is observed with an elevation of blood pressure in the greater circulation (arterial hypertension), as well as in increased density of the wall and valves of the aorta (atherosclerosis).

The accent of the S2 is determined by comparing its intensity and timbre on the aorta and pulmonary artery**.**

Increased intensity of S2 at the II-d intercostal space on the left (its S2 accent on the pulmonary artery) indicates an elevation of pressure in the pulmonary circulation (pulmonary hypertension). This can be observed in heart diseases (mitral valves defects, left ventricular failure), as well as in a number of respiratory diseases (emphysema, pneumosclerosis).

Table 10-2. Causes of the heart sounds intensity changes

Sounds intensity	Both sounds	I sound	II sound
Increase of intensity	- physical and emotional strain; - thyrotoxicosis; - thin chest wall; - pneumosclerosis; - blood viscosity decrease (anemia)	- mitral stenosis; - tricuspid stenosis; - during extrasystole	- in essential hypertension; - atherosclerosis of aorta; - mitral valves diseases; - pulmonary heart
Decrease of intensity	- in decreased myocardial contractility (myocarditis, cardiosclerosis, collapse); - pericardial cavity; - obesity; - pulmonary emphysema; - fluid accumulation in the left pleural cavity	- mitral, tricuspid and aortic valve insufficiency, - aortic stenosis; - diffuse affections of the myocardium (due to dystrophy, cardiosclerosis or myocarditis)	-in aortic and pulmonary valve a diseases; - arterial hypotension in greater and lesser circulation

Weakening of S1 at the apical impulse can be observed in inflammatory processes in the myocardium (myocarditis), cardiosclerosis, with the defeat of the valve apparatus (insufficiency of the bicuspid valves). This is due to the weakening of the muscle (the force of the ventricular contraction), valves (decrease in the amplitude of the vibration of the valves, the surface of the closing of the valves) components of the S1.

Weakening of S2 on the aorta is possible with aortic defects (aortic valve insufficiency, aortic stenosis). **Weakening of S2 on the pulmonary artery** occurs with valve insufficiency or narrowing of the orifice (stenosis) of the pulmonary artery. The mechanism of these changes is associated with destruction of the cusps of the valves are in their insufficiency, a decrease in the power collapse and oscillations of the valves during the stenosis of the semilunar valves.

Sound reduplication - when auscultating the heart, instead of one of the sounds, two short sounds are heard, following each other through a short period of time. **Sound splitting** – if the difference in the time of occurrence of these components is insignificant and does not create the impression of a split. Thus, there is no fundamental qualitative difference between splitting and duplication of the sounds there is only some quantitative difference (Fig. 10-3).

Splitting and reduplication of the sounds appear at the non-simultaneous occurrence of the sound components: non-simultaneous closure of the atrioventricular valves leads to a split of the S1 non-simultaneous closure of the semilunar valves of the aorta and pulmonary artery – to a split of the S2.

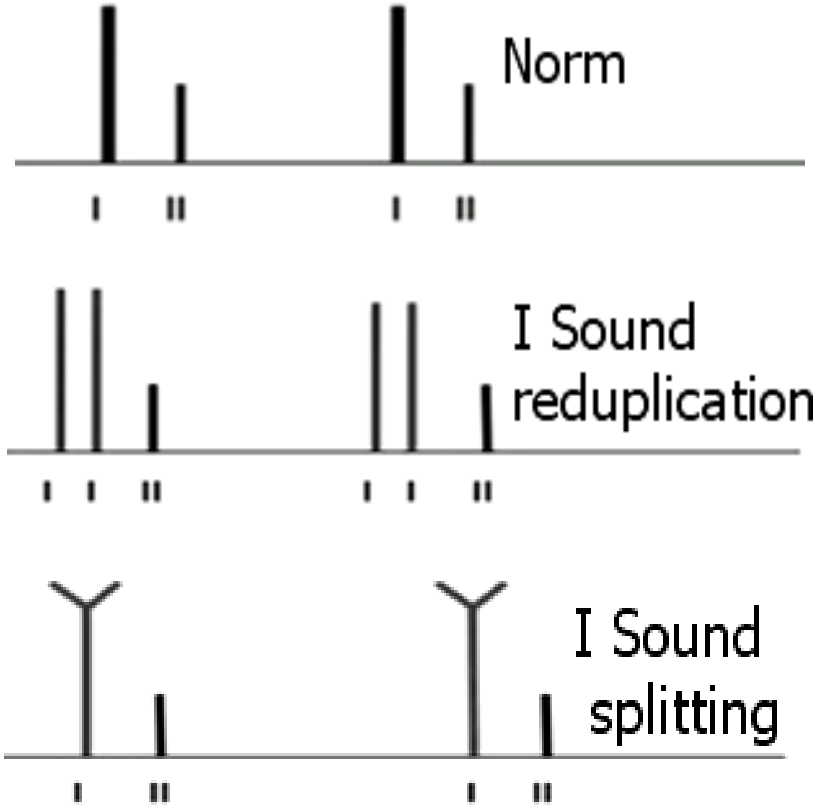


Fig. 10-3. Reduplication or splitting of the first sound:
I – first sound, II – second sound

Physiological reduplication of S1 may depend on the non-simultaneous closure of the bicuspid and tricuspid valves as a result of changes in chest pressure in different phases of the respiratory cycle: during a very deep exhalation due to the increased pressure in the chest, blood enters the left atrium with greater force and slows down the closure of the mitral valve.

Pathological reduplication of S1 can be observed in the violation of the intraventricular conduction (along the His bundle branches) that result in lengthening the duration of the myocardial contraction of one of the ventricles (in the systole).

Reduplication of S2 occurs more often than reduplication of S1, and it is due to the non-simultaneous closure of the semilunar valves of the aorta and pulmonary artery because a significant increase in pressure in the greater or pulmonary circulation. *Reduplication and splitting of S2 can be physiological and pathological.*

Physiological reduplication of S2 is heard only at the base of the heart during inhalation and exhalation or during exercise.

At the end of a deep inspiration with the expansion of the chest, the pressure in it decreases, the blood is somewhat delayed in the dilated vessels of the pulmonary circulation, and therefore in a smaller amount enters the left atrium. The left ventricle, due to less blood filling, finishes the systole before the right one, and the closure of the aortic valve precedes the closure of the pulmonary valve. The closure of the pulmonary valve at the same time is delayed due to increased blood filling of the right ventricle and elongation of its systole.

During the expiration, the opposite conditions present: with increasing pressure in the chest, the blood, as if squeezing out of the vessels of the pulmonary circulation, enters the left half of the heart in greater quantities. The systole of the left ventricle and the beginning of its diastole occurs later than the right.

Pathological reduplication of S2 in the second intercostal space on the left appears with mitral stenosis. At the stage of hypertrophy and expansion (dilatation) of the right ventricle, its blood filling is increased. As a result, the right ventricle overflowing with blood finishes the systole later than the left. Therefore, the pulmonary valve closes later than the aortal valve.

Reduplication or splitting of S2 can be heard with mitral valve insufficiency. An additional volume of blood circulating between the left parts of the heart with this defect leads to an elongation of the left ventricular systole. In this case, diastole of the left ventricle begins later than the right. As a result, the aortic valve closes later than the pulmonary valve.

10.4.2. Qualitative changes of the heart sounds

Changes of the heart sounds timbre is acquisition of special tint (soft, dull, sonorous, metallic). In this case, the heart sounds volume may not change, increase or decrease simultaneously with the change of sound timbre.

The **flapping S1 at the apical impulse** is a characteristic of mitral stenosis. Flapping timbre of S1 is the result of oscillations of sclerotic modified cusps of the mitral valve with reduced blood filling of the left ventricle in the diastolic phase. The flapping S1 resembles the sound of the flag flapping in the wind, or the sound coming from the children's clapper. It is combined with S1 volume increase.

The dull (muffed, soft) S1 is characterized by a decrease in volume and a muffled sound. It is auscultated with diffuse lesions of the myocardium (myocarditis, myocardial dystrophy, ischemic heart disease).

Metallic timbre of S2 is heard above the aorta with atherosclerotic changes in its wall. “Metallic” S2 may be increased intensity (S2 accent).

Gun I sound (described by N.D. Strazhesko) is due to accidental simultaneous contraction of atria and ventricles during complete (III-d degree) atrioventricular blockage. It is heard like a loud tone in the background of the rare dull sounds.

Velvet sound (of Dmitrenko) is observed in active rheumatic myocarditis in children, and it is characterized by a special softness. This is kind of the first sound (S1) on timbre reminiscent of the sound of the drum stick beat on the taut velvet (described by L.F. Dmitrenko).

10.5. Pathologic triple rhythms

Opening snap

Opening snap is an additional high-frequency sound immediately after S2 that is heard at the apical impulse in mitral stenosis.

In mitral stenosis, the deformed and sclerosed mitral valve opens sharply under the pressure of a jet of blood from the overfilled left atrium. This leads to ***opening snap*** (or “click of mitral valve opening”) immediately in 0.03-0.14 second after S2.

The additional sound together with the intensive flapping S1 and normal S2 form a melody resembling the cry of a quail (Fig. 10-4). Hence the name of this sound phenomenon in Russian “ритм перепела” (literal translation in English - "rhythm of quail "). The distribution area of this triple rhythm is the apical impulse and upwards.

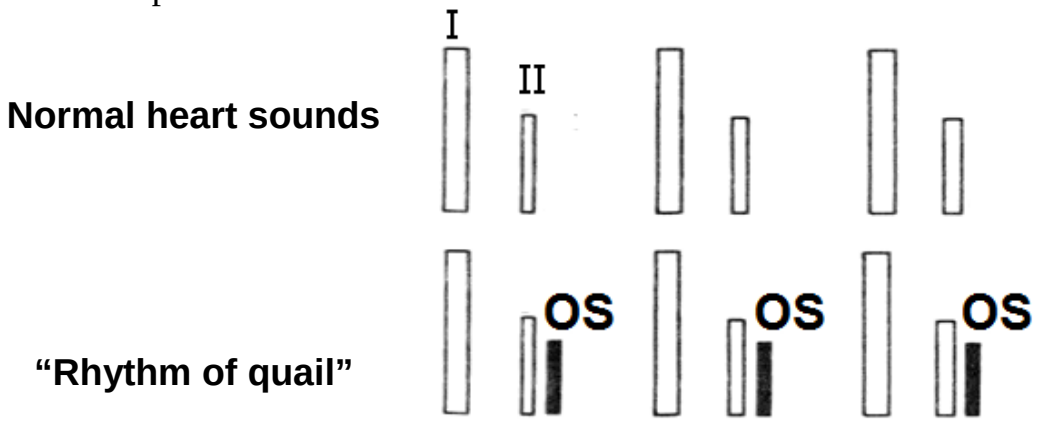


Fig. 10-4. Opening snap and “rhythm of quail”:
I – first sound, II – second sound, OS – opening snap

Gallop rhythm

"Gallop rhythm" is a three-membered rhythm, reminiscent of the trampling of a galloping horse, it is heard in severe myocardial lesions. It is figuratively called the "cry of the heart for help". The "gallop rhythm" is a due to a significant weakening of the contractile ability of myocardium of the ventricles (predominantly the left) and by overstraining myocardium of the atria (mostly left).

"Gallop rhythm" includes weakened dull S1 and S2 in combination with pathological enhanced S3 or S4.

"Gallop rhythm" is heard at the apical impulse with a stethoscope and directly by the ear (together with the sound, a soft push is perceived, transmitted

from the heart to the chest in the diastole phase). The conditions of listening is improved in position on the left side.

It is distinguished between presystolic, protodiastolic, mesodiastolic, summation, and systolic "gallop rhythm" (depending on the phase of the cardiac cycle during which there is abnormal additional sound).

Protodiastolic "gallop rhythm": the S1 and S2 are weakened, the pathological enhancement of S3 in protodiastole is a consequence of the rapid stretching of the atonic walls of the left ventricle when the first portion of blood enters them. The sound of the ventricular myocardium is reduced in myocarditis, myocardial infarction, myocardiodystrophy (Fig. 10-5).

Presystolic "gallop rhythm" is due to the appearance of a pathological enhancement of S4 at the end of the diastole due to the constriction of the hypertrophied and dilated left atrium and the maximum stretching of the atonic walls of the ventricles into the presystole. It can be auscultated in mitral stenosis, severe hypertension, myocarditis, myocardial infarction, myocardiodystrophy.

Mesodiastolic "gallop rhythm" is the triple rhythm, in which pathological S3 or S4 occurs in mid-diastole with severe tachycardia, accompanied by shortening of the diastole. The rest of the mechanism is the same as protodiastolic or presystolic "gallop rhythm".

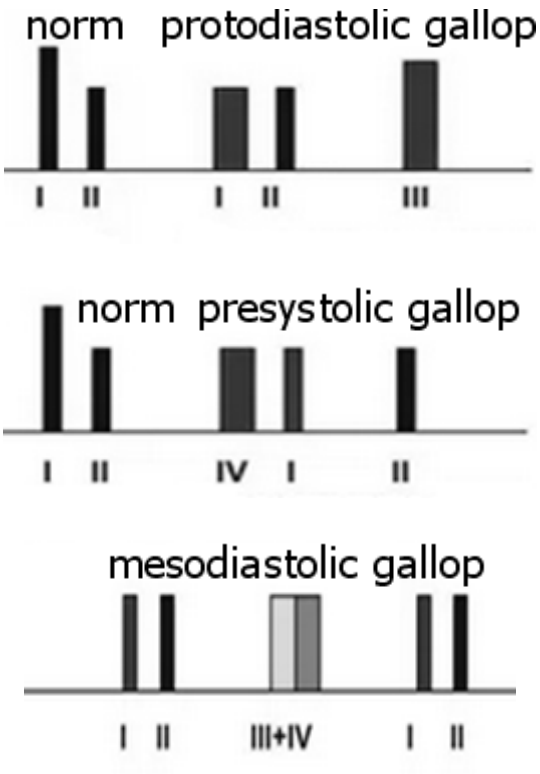


Fig. 10-5. Variants of the gallop rhythm:
I – first sound, II – second sound, III - abnormally increased third (protodiastolic) sound, IV – abnormally increased fourth (presystolic) sound

Summation "gallop rhythm" is the three-membered rhythm due to the summation of the S3 and S4. With severe myocardial damage, the muscle of the left ventricle loses its tone so much that it does not react to the first portion of blood from the atria, since there is a residual portion of the blood in the left ventricle. Stretching of its walls (lagging S3) occurs when at the end of the diastole before the systole, there is a contraction of the atria, and all the blood from the left atrium enters the left ventricle (S4). Therefore, the S3 and S4 are summed up – the summation "gallop rhythm".

Systolic "gallop rhythm" consists of a bifurcated S1 and normal S2. Bifurcation of the S1 is associated with the presence of a complete blockage of one of the His bundle branches in severe myocardial lesions (myocarditis, myocardial infarction).

10.6. Heart murmurs auscultation

Physical and hemodynamic bases of heart murmurs

In pathology and often in healthy people, murmurs are auscultated in addition to the heart sounds.

Differences between heart sounds and murmurs: Heart sounds are heard as a single short sound, having a clear beginning and end. A heart murmur is perceived as a long-lasting sound of various nature.

The conditions of murmurs occurrence:

• narrowing of the orifice through which the blood flows,

• increased blood flow rate,

• pathological blood flows in the presence of a defect in the heart septum and the presence of a hole in non-closed cusps of the valve,

• appearance of turbulent blood flows in violation of the myocardium contractility,

• reducing blood viscosity (in anemia).

Classification of murmurs

Systolic murmurs occur during systole between S1 and S2, **diastolic murmurs** occur during diastole between S2 and S1 (Table 10-3). Systolic murmur coincides with the apical impulse and pulse on the carotid artery, and diastolic – with a long pause in the heart.

Murmurs can be soft, blowing, rough, scratching, musical. They can be short, long, quiet and loud.

The murmur volume changes in proportion to the speed of the blood flow that makes the murmur. Accordingly, they decrease and increase.

A systolic murmur is usually decreasing. Systolic murmurs can occupy part of the systole or be listened to throughout the systole. Systolic murmurs can occur in heart valve defects, violations of greater vessels (aorta, pulmonary artery) and in the unchanged heart with functional disorders.

Table 10-3. Classification of cardiac murmurs

Criteria of classification	Classification groups
Cause	Endocardiac and extracardiac murmurs (pleuropericardial/ cardiopulmonary and pericardial friction murmurs)
Changes of the structure of the heart	Organic and functional (innocent) murmurs
Time of appearance	- Systolic murmurs - Diastolic murmurs (protodiastolic, mesodiastolic, presystolic murmurs)
Relation to the course of the blood flow	Ejection and regurgitation (regurgitant) murmurs
Amplitude of the murmur	High and low amplitude murmurs
Oscillation frequency of the murmur noise	High-pitched and low-pitched murmurs
Character of the murmur noise	Faint (weak), soft, blowing, coarse, rough, grating or grazing sounds; musical murmurs
Changes of the intensity of the noise with the phase of the heart activity	Decrescendo (decreasing), crescendo (increasing, growing), and crescendo-decrescendo (diamond-shaped) murmurs

Ejection (expulsion) murmurs occur during the normal direction of blood flow through the valve orifice. **Regurgitation (return) murmurs** appear at retrograde unusual movement of blood through the heart valves.

There are three types of diastolic murmur, depending on the phase of diastole, when this murmur occurs.

Protodiastolic murmur is heard at the very beginning of the diastole, immediately after the S2, decreasing.

Mesodiastolic murmur occurs in mid-diastole, in 0.1-0.2 sec. after the S2, increasing-decreasing (fusiform).

Presystolic murmur - at the end of the diastole, before the S1, increasing (with presystolic amplification). Diastolic murmur may occupy the entire diastole (pandiastolic murmur).

Diastolic murmurs occur only in the heart valves defects.

By the nature of the occurrence of heart murmurs can be divided into two groups:

• **intracardiac murmurs** - due to the structural and functional features of the intracardiac structures (valves, myocardium);

• **extracardiac murmurs** - occurring outside the heart (pericardium, adjacent to the heart areas of the lungs, blood vessels).

Properties of murmurs

The following characteristics of murmurs are examined:

1. Determine the phase of the heart in which the murmur is heard: systolic murmur (the first half, mesosystolic, the second half, and total - pansystolic), diastolic murmur (presystolic, mediastolic, protodiastolic, total - pandiastolic) (Fig. 10-6);

2. To determine the intensity of the murmur;

3. Determine the shape of the murmur:

- murmurs may become weaker (decrescendo, or decreasing) or louder (crescendo, or increasing), and crescendo-decrescendo (increasing- decreasing, or rhomboid, or diamond-shaped), ribbon-shaped (a murmur takes the entire phase from one tone to another).

4. To determine the best place of the murmur auscultation, consistently listening to the entire area of the heart. The best point of auscultation shows which heart chamber or valve affection results in the murmur origin.

5. Determine the murmur irradiation: whether the noise is carried out for the contour of the heart or not, the place of the noise.

6. Determine the effect of breathing phases, position of the patient, and physical load on the characteristics of the murmur.

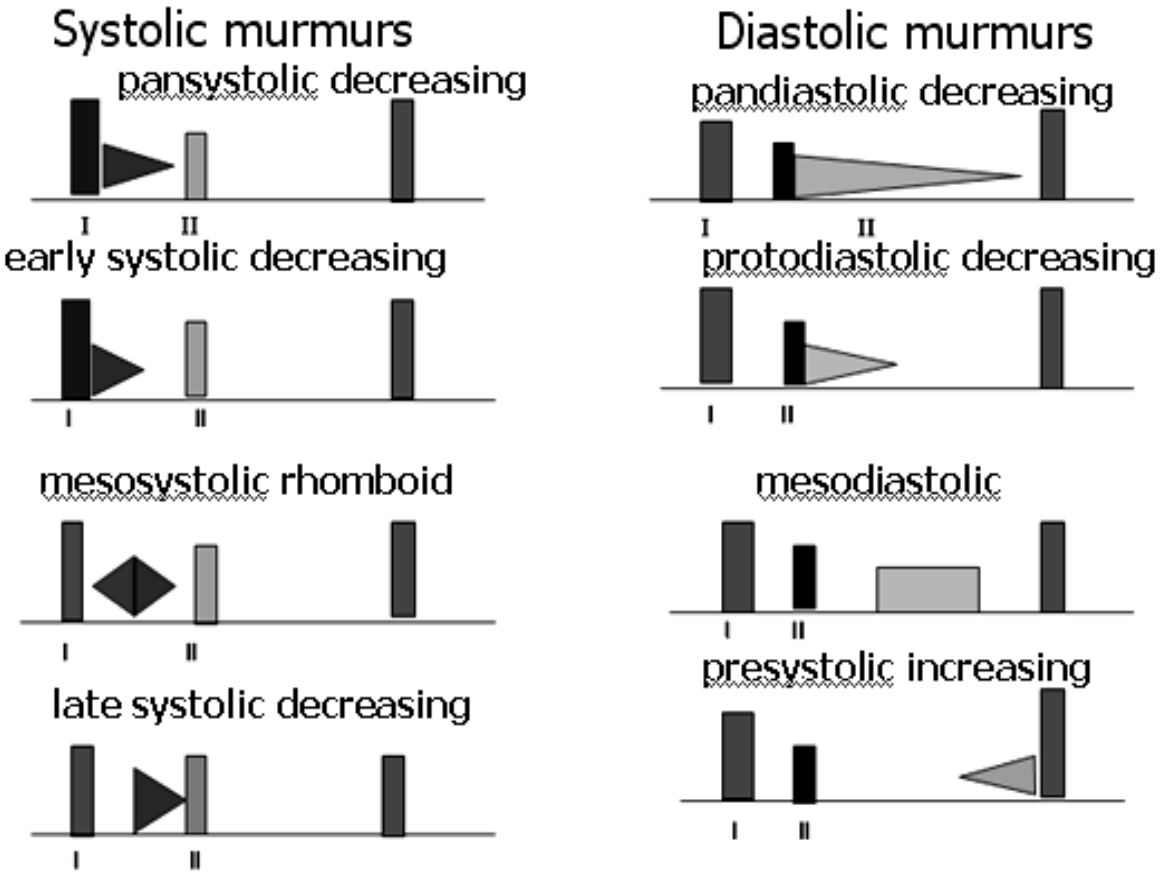


Fig.10-6. Variants of murmurs according relation to systole or diastole and intensity of the noise

10.7. Differentiation of heart murmurs

Intracardiac murmurs may be:
I. Organic murmurs -
1) muscular;
2) valvular;
3) vascular.
II. Functional (innocent) murmurs.

Organic intracardiac murmurs indicate anatomical lesions of the valves or the orifices they close, changes in the heart muscle.

Organic muscular murmurs are caused by the damage to myocardium (in myocarditis, myocardiodystrophy, myocardial infarction, cardiosclerosis) due to a violation of the geometry of the ventricular myocardium contraction. It is heard at the apical impulse, at the base of the xiphoid process, at the Botkin-Erb's point. Always systolic murmurs are not held at other points.

Organic valvular murmurs occur as a result of the damage to the heart valves: with incomplete closure of their valves during the closure of the corresponding orifice (valve insufficiency), or with narrowing of the lumen of the latter (stenosis of the mouth of the vessel). The best listening point for a valve noise is the listening point of the respective valve.

Organic vascular murmur appears in the defeat of the initial parts of the large vessels (aorta aneurysm, aorta and pulmonary artery sclerosis).

Vascular systolic murmur can occur in sclerosis of the ascending aorta. It is auscultated in the II-d intercostal space on the right, and it is due to the relative narrowing of the aortic lumen (due to the mismatch of the lumen of the aortic mouth and its ascending part). This murmur increases with raised hands (symptom of Sirotinin-Kukoverov). Other features – as with valvular aortic stenosis.

10.7.1. Differentiation of organic and functional murmurs

Functional (*innocent, physiologic*) murmurs are heard in the absence of the organic damage to the muscular, valvular and vascular apparatus of the heart.

Causes of functional murmurs:

• acceleration of blood flow (tachycardia),

• reduction of blood viscosity (anemia),

• papillary muscle dysfunction (increased or decreased tone).

Differences between functional and organic murmurs (Table 10-4):

• functional murmurs are unstable, can change, arise, and disappear at different positions of the body, after physical activity, in different phases of respiration;

• functional murmurs are heard on the background of unmodified or functionally changed heart sounds;

• functional murmurs are heard in the systole at a limited area (most often at the apical impulse and on the pulmonary artery);

• functional murmurs are not held at other points, have low volume and soft timbre.

Table 10-4. Differentiation between the organic and the functional murmurs

Properties	Organic murmurs	Functional murmurs
Relation to systole and diastole	systolic, diastolic	systolic (in most of cases)
Relation to changes of posture, active respiration, exercises	persistent, increase may be	transient, and may arise and disappear
Best point of auscultation	depending on the affected heart valve	pulmonary trunk and less frequently over the apical impulse
Relation to the phase of the heart activity	persistent during the entire systole, and at diastole	transient and are rarely heard during the entire systole
Character of murmur	more coarse, rough, with musical timbre	soft and blowing
Irradiation of murmur	presents	no radiation

In case of insufficiency of the aortic valves (the main point of auscultation – the second intercostal space to the right of the edge of the sternum) at the apex of the heart, functional diastolic (presystolic) ***Flint 's murmur*** can be heard. It is formed due to the rapid reverse flow of blood to the left ventricle from the aorta, which leads to the elevation of the mitral valve cusps and the emergence of a transient narrowing of the left atrioventricular opening.

Systolic functional murmur is auscultated at the apical impulse when the prolapse of mitral valve in connection with the bending of the valve cusps into the atrium, the discharge edges of the valves from each other with the formation of a gap.

10.7.2. Differentiation of most common organic murmurs

Organic valvular murmurs occur as a result of the damage to the heart valves: with incomplete closure of valves orifice (valve insufficiency), or with narrowing of the lumen of the latter (valve stenosis). The best listening point for valve murmur is the standard auscultation point of the respective valve.

Valvular murmurs in acquired heart valves diseases:

• **systolic murmur** – in case of insufficiency of bicuspid and tricuspid valves, stenosis of semilunar valves of aorta and pulmonary artery;

• **diastolic murmur** – in stenosis of bicuspid and tricuspid valves, insufficiency of semilunar valves of aorta and pulmonary artery.

In case of ***combined defect of one valve***, i.e. insufficiency of the valve and narrowing of the opening valve (stenosis), two murmurs – systolic and diastolic - are heard above this valve.

With ***combined defects of several valves***, murmurs are heard at several appropriate auscultation points.

Murmurs in heart valves disease are conducted:

• by blood flow,

• in the direction of greater cavity,

• along contracted part of myocardium attached to the affected valve.

The sound volume of the murmur at the point of conduction is reduced in proportion to the square of the distance from the main point of auscultation.

Systolic apex murmur

In case of mitral valve insufficiency, the point of the highest volume of systolic murmur corresponds to the place of the best listening of the valve at the apical impulse. The murmur is conducted in the direction of the left atrium (left II–III-d intercostal spaces), as well as in the left axillary region. This murmur becomes clearer when the patient holds breath while lying on the left side (Table 10-5).

Diastolic apex murmur

Diastolic murmur in mitral stenosis is heard at the apical impulse and Botkin-Erb's point. Depending on the characteristics of the heart rate, this diastolic murmur may have the following signs: with a sinus rhythm, diastolic murmur has the greatest amplitude in the presystole, and with atrial fibrillation – in protodiastole. This murmur is better auscultated in the vertical position of the patient with breath holding at expiration.

Systolic murmur at the II-d interspace by the right edge of the sternum

Systolic murmur at the aortic stenosis has the highest volume in the II-d intercostal space to the right of the sternum, and it is also heard at the Botkin-Erb's point. The murmur is conducted to the apical impulse and by the blood flow to carotid and subclavian arteries. It can be heard at the interscapular space. It is amplified in the supine position, and with breath holding at expiration. This murmur has musical or rough timbre, scraping (increasing-decreasing) character.

Diastolic murmur at the II-d interspace by the right edge of the sternum

Diastolic murmur in case of aortic valve insufficiency is heard in the II-d intercostal space to the right of the sternum, and is conducted by blood flow to the apical impulse point. Often, this murmur is auscultated better at Botkin-Erb's point in the vertical position of the patient. It has a blowing, gentle timbre.

Systolic murmur at the II-d interspace by the left edge of the sternum

Systolic murmur in the II-d intercostal space to the left of the sternum edge is heard in pulmonary artery stenosis. The mechanism formation of this murmur is similar to systolic murmur in aortic stenosis. It can be conducted to the Botkin-Erb's point, to the base of xiphoid process. This murmur is better auscultated in the horizontal position of the patient.

Table 10-5. Characteristics of organic heart murmurs

Heart Valves Disease	Localization of the murmur	Best position for auscultation	Relation to cardiac phases	Character of murmur	Irradiation of murmur to
Mitral insufficiency	apical impulse	horizontal, recumbent on the left side	pansystolic	descending	left axillary area, II-III interspaces to the left of sternum
Mitral stenosis	apical impulse	vertical	protodiastolic, presystolic, pandiastolic	descending, ascending	no typical irradiation
Aortal insufficiency	II interspace to the right of sternum, Botkin-Erb point	vertical	pandiastolic	blow, descending	apical impulse
Aortal stenosis	II interspace to the right of sternum	horizontal	pansystolic	rough, “diamond-shape”	onto the carotids, interscapular space
Tricuspid insufficiency	xiphoid process, III-IV interspaces to the right of sternum	horizontal, at the height of inspiration	pansystolic	descending	no typical irradiation

Diastolic murmur at the II-d interspace by the left edge of the sternum

Diastolic murmur at the II-d interspace by the left edge of the sternum is characteristic of pulmonary artery valves insufficiency. The mechanism formation of this murmur is similar to diastolic murmur in aortic insufficiency.

Systolic murmur at the base of xiphoid process

In case of tricuspid valve insufficiency, systolic murmur is better heard at the base of xiphoid process of the sternum. From here, it is conducted up and to

the right towards the right atrium. The murmur is amplified in the horizontal position of the patient and breath holding at a height of inspiration.

Diastolic murmur at the base of xiphoid process

Diastolic murmur above the xiphoid process is characteristic of tricuspid stenosis. The mechanism formation of this murmur is similar to diastolic murmur in bicuspid (mitral) valve stenosis.

Organic murmurs can be heard in congenital heart disease. The most common congenital heart diseases include defect of intraatrial septum - patent (opening) foramen ovale, defect of interventricular septum, patent ductus arteriosus (Botallo's duct).

With a *defect of the intraatrial septum (patent foramen ovale)*, the presence of systolic and diastolic murmurs is characteristic, having a maximum of audibility in the region of attachment of the left III-d rib to the sternum.

In case of a *defect of the intraventricular septum* – the scratching murmur is heard on the left edge of the sternum at the level of III-d or IV-th intercostal spaces, and it is conducted to interscapular space.

In case of *patent ductus arteriosus (Botallo's duct)*, when the aorta is connected to the pulmonary artery, systolic murmur (sometimes diastolic) is heard at the left II-d intercostal space. It is heard (to a lesser extent) and above the aorta, and is conducted in the interscapular region closer to the spine and on the carotid arteries. This murmur is combined with the accented S2 on the pulmonary artery.

10.8. Extracardiac murmurs

Extracardiac murmurs include:

- ***pericardial friction murmur,***
- ***pleuropericardial murmur,***
- ***cardiopulmonary murmur.***

Pericardial friction murmur occurs in inflammatory processes in pericardium. It is heard in both phases of cardiac activity – in systole and diastole. It is better revealed at the area of absolute dullness of the heart when tilting forward on expiration. Pericardial friction murmur is characterized by instability of murmur and different timbre color.

Pleuropericardial murmur occurs in an inflammatory process of pleura adjacent to pericardium. It resembles pericardial friction murmur, but different – pleuropericardial murmur increases on inspiration and expiration, and when breath is delayed - decreases or disappears, i.e., it is essentially a pleural friction murmur. Pleuropericardial murmur is heard on the left contour of the heart relative dullness.

Cardiopulmonary murmur is a vesicular respiration listened to on edge of the heart in connection with expansion of the lung with a decrease in volume of ventricles during systole.

10.9. Auscultation of blood vessels

Auscultation of arteries

With auscultation of arteries, it is impossible to squeeze the vessel with a phonendoscope. When compression, a stenotic murmur occurs.

Listening to ***carotid arteries*** is carried out along the medial edge of *m. sternocleidomastoideus* at the level of thyroid cartilage.

Subclavian artery is listened to externally from the junction of the upper edge of clavicle and the lateral leg of *m. sternocleidomastoideus,* or at the lateral third of subclavian fossa.

Femoral artery is auscultated below the middle of *ligamentum Pouparti* with the hip turned outwards in a horizontal position of the patient.

For auscultation of ***abdominal aorta***, the bell of phonendoscope is installed in the umbilical region.

Under normal conditions, you can listen to two quiet sounds with auscultation of the arteries located close to the heart (carotid and subclavian). The first sound (S1) occurs as a result of tension of the arterial wall during ventricular systole, and the second (S2) is carried out from closing the valves of the aorta during diastole. When auscultation of an artery, which is located far from the heart, sounds are not heard. Sometimes S1 can be heard on the femoral artery.

Two sounds appear on peripheral large arteries in the left ventricular hypertrophy, hyperthyroidism, falling tonus of arteries in severe infectious diseases.

In case of aortic valve insufficiency, on femoral and other major arteries (brachial, abdominal aorta), two sounds (***double sound of Traube***) are heard.

In a patient with aortic valve insufficiency with a light compression of femoral artery by phonendoscope, two murmurs can be heard – in systole and in diastole (***double Vinogradov-Durozier murmur***). The first of murmurs is stronger. Appearance of systolic murmur is associated with a significant acceleration of the blood flow into systole due to release of an increased amount of blood from left ventricle; diastolic murmur - with a slowdown in the blood flow into diastole due to blood regurgitation from ascending part of aortic arch to the left ventricle.

Conducted systolic murmur on the carotid arteries is auscultated in aortal valve stenosis, aneurysm of ascending portion and the arch of the aorta.

On carotid and subclavian arteries, systolic murmur can be heard in anemia, which is associated with an increase in speed of blood flow and decrease of blood viscosity.

In thyrotoxicosis, murmur can be heard over the thyroid gland due to increased blood flow through its vessels.

Auscultation of veins

On jugular veins (often right), systolic-diastolic blowing or buzzing murmur (***nun`s murmur, or venous hum; or humming-top murmur***) may appear. It

occurs in anemia due to the increased blood flow rate and reduced blood viscosity. The stethoscope is mounted on the neck between medial legs of m. sternocleidomastoideus (above sternal end of clavicle) at vertical position of the patient. The murmur increases with a deep breath or turn the head to the left (opposite) side.

10.10. The key points on the theme "Auscultation of the Heart"

1. Goals of the heart auscultation:
- heart rate detection;
- listening to heart sounds;
- listening to heart murmurs.

2. Auscultation of the heart in healthy individuals determines two sounds - S1 (systolic) and S2 (diastolic). In pathologic conditions, you can listen to third and fourth sounds - S3 (protodiastolic) and S4 (presystolic).

3. Auscultation points differ significantly from the valves projection on thoracic wall. Listening sequence: mitral valve, aortic valve, pulmonary valve, tricuspid valve and Botkin-Erb's point.

4. Assessment of S1 is conducted on apical impulse. S1 coincides with the apical impulse and pulsation of the carotid arteries. S2 is evaluated on the basis of the heart.

5. Changes of heart sounds may be: 1) quantitative - strengthening (increased intensity), weakening, splitting, or duplication; 2) qualitative - timbre (flapping, dull, metal, velvet), duration; 3) appearance of additional sounds - (opening snap, "gallop rhythm").

6. Physiological changes of heart sounds depend on thickness of chest wall, heart rate and physical load. Pathological changes of S1 are due to disorders of atrioventricular valves and ventricular myocardium. Pathological changes of S2 are caused by disorders of semilunar valves, walls of aorta and pulmonary artery, and blood pressure in greater and pulmonary circulation.

7. Heart murmurs causes are:

(1) incomplete opening of the heart valve (stenosis);

(2) incomplete closure of the valve (valves insufficiency) that results in a reverse blood flow (regurgitation);

(3) presence of an abnormal hole (defect of interventricular or intraatrial septum);

(4) increasing speed of blood flow;

(5) reduction of blood viscosity (in anemia).

8. When auscultation of murmurs, it is determined:

1) phase of cardiac cycle (systolic or diastolic murmur);

2) properties of the murmur - character, strength, duration. intensity;

3) localization of the murmur (the place of best listening);

4) direction of murmur irradiation.

9. Differences of functional murmurs from organic murmurs:

1) the heart valve is not changed,

2) no other signs of valve lesions (enlargement of the heart, changing heart sounds);

3) inconstant, appear and disappear during change of personal body positions, physical activity, in different phases of the respiratory cycle;

4) most often systolic, heard over pulmonary trunk a apical impulse;

5) short, soft, blowing, at the limited site.

10.11. Assessment tests on the theme "Auscultation of the Heart"

1. Components of I cardiac sound are:

1. muscle;
2. valves;
3. vascular;
4. atrial;
5. venous.

2. Components of II cardiac sound are:

1. valves;
2. vascular;
3. musclular;
4. atrial;
5. ventricular.

3. Heart sounds in mitral stenosis:

1. I clapping sound;
2. II sound is not changed;
3. opening snap of mitral valve;
4. I sound not changed;
5. II sound is amplified.

4. Opening snap is auscultated in:

1. tricuspid valve insufficiency;
2. mitral valve insufficiency;
3. aortic stenosis;
4. aortic insufficiency;
5. mitral stenosis.

5. What diseases does the "gallop rhythm" (pathological III and IV heart sounds) occur in?

1. rheumatic carditis;
2. mitral stenosis;
3. myositis;
4. myocardial infarction;

5. myocarditis.

6. "Gallop" rhythm can be depending on the time of additional diastolic sound:
1. presystolic;
2. asystolic;
3. prediastolic;
4. protodiastolic;
5. mesodiastolic.

7. Heart auscultation should be performed in the following order:
1. apical impulse - - II-d intercostal space to the left of sternum - II-d intercostal space to the right of sternum - the base of xiphoid process – Botkin-Erb's point;
2. apical impulse – II-d intercostals space to the right of sternum – II-d intercostals space to the left of sternum – the base of xiphoid process – Botkin-Erb's point;
3. apical impulse - the base of xiphoid process - II-d intercostal space to the right of sternum - II-d intercostal space to the left of sternum –- Botkin-Erb's point;
4. II-d intercostal space to the left of sternum - II-d intercostal space to the right of sternum - apical impulse - the base of xiphoid process.

8. Point of mitral valve auscultation:
1. apical impulse;
2. Botkin-Erb's point;
3. base of xiphoid process;
4. II-d intercostal space to the left of sternum;
5. II-d intercostal space to the right of sternum.

9. Point of aorta valve auscultation:
1. apical impulse;
2. Botkin-Erb's point;
3. base of xiphoid process;
4. II-d intercostal space to the left of sternum;
5. II-d intercostal space to the right of sternum.

10. Differentiation between I and II heart sounds includes:
1. the best auscultation point of I sound is the apical impulse;
2. the best auscultation point of II sound is the heart base;
3. I sound follows a short pause, II sound follows a long pause;
4. I sound follows a long pause, II sound follows a short pause;
5. I sound follows apical impulse;

6. I sound is synchronous apical impulse.

11. Increased volume of both I and II heart sounds presents in:
1. tachycardia;
2. thin chest wall;
3. pneumosclerosis;
4. blood viscosity decrease (anemia);
5. mitral stenosis.

12. Increased volume of I heart sound presents in:
1. tricuspid stenosis;
2. during extrasystole;
3. mitral stenosis;
4. arterial hypertension;
5. bradycardia.

13. Increased volume of II heart sound presents in:
1. pulmonary hypertension;
2. arterial hypertension;
3. mitral stenosis;
4. atherosclerosis of aorta;
5. during extrasystole.

14. Diastolic murmurs are better auscultated:
1. in a vertical position;
2. in a horizontal position;
3. during physical exercises;
4. at inspiration;
5. at a horizontal position turning on the left side.

15. Systolic murmurs are better auscultated:
1. in vertical position;
2. in horizontal position;
3. during a pause after expiration at forward inclination;
4. at inspiration;
5. during physical exercise stress.

16. What are typical for the pericardium friction rub?
1. it is intensified by pressing by a stethoscope and at forward inclination of a trunk;
2. it is better auscultated at imitation of respiration;
3. it is auscultated in systole and diastole;
4. it is auscultated above absolute heart dullness;

5. it is weakly conducted from the place of the formation.

17. Mechanisms of heart murmurs are:
1. turbulent blood flow due to luminal narrowing;
2. turbulent blood flow due to luminal dilatation;
3. turbulent blood flow due to other the obstructions;
4. arterial hypertension;
5. increased blood flow velocity.

18. Kinds of heart murmurs according to the phases of the cardiac activity:
1. systolic;
2. protodiastolic;
3. mesodiastolic;
4. presystolic;
5. pansystolic.

19. Causes of functional murmurs:
1. increased rate of blood flow;
2. decreased blood viscosity;
3. disordered tonicity of myocardium due to autonomous dysfunction;
4. relative insufficiency of heart valves due to dilation of heart chambers and fibrous rings of the atrioventricular orifices;
5. turbulent blood flow due to luminal narrowing or dilation;

20. Differentiation between the organic and functional murmurs includes:
1. organic murmurs are systolic and diastolic;
2. functional murmurs are systolic usually;
3. after physical exercises a functional murmur increases, an organic murmur decreases;
4. after physical exercises an organic murmur increases, a functional murmur decreases;
5. best points of auscultation of functional murmur depend on affected heart valves.

21. Organic murmur in mitral valve insufficiency is characterized by:
1. localization on the base of xiphoid process;
2. localization on the apical impulse;
3. best position for auscultation is recumbent on the left side;
4. pansystolic descending murmur;
5. irradiation to the left axillary space;
6. irradiation on carotid artery.

22. Organic murmur in the mitral valve stenosis is characterized by:

1. localization on the apical impulse;
2. best position for auscultation is recumbent on the left side;
3. pandiastolic, or protodiastolic, or presystolic;
4. systolic;
5. irradiation to the left axillary space.

CHAPTER 11. Electrocardiography

Goals: to enable students to learn –

1) anatomical and physiological bases of ECG (electrocardiography);
2) algorithm of interpreting ECG;
3) ECG signs of heart chambers hypertrophy;
4) ECG diagnosis in IHD (ischemic heart disease);
5) ECG diagnosis of sinus node heart arrhythmias and blocks.

11.1. Electrophysiological bases of ECG

Electrocardiography is a method of graphic registration of electrical processes occurring in the heart during its activity.

Electrophysiological functions of the heart:

- ***Automaticity*** – function of pacemaker cells to produce spontaneously the action potential (transient depolarization);
- ***Conduction*** - capability to impulse propagation through cardiac tissues;
- ***Excitability*** – capability to become excited under the influence of impulses;
- ***Refractoriness*** is a property of cardiac cells that defines the period of recovery that cells require before they can be reexcited by a stimulus;
- ***Contractility*** – capability of myocardium to contract in response to excitement.

Cardiac conduction system

The depolarization stimulus for the normal heartbeat originates in the ***sinoatrial (SA) node,*** **or** ***sinus node***, a collection of ***pacemaker cells***. These cells fire spontaneously; that is, they exhibit *automaticity*. ***Pacemaker cells exhibit automaticity in all departments of conduction system: I- sinus node (SA), II - AV junction (and atrial fibres) and AV (atrioventricular) node and His-bundle), III- His-bundle branches, Purkinje fibers*** (Fig.11-1).

The first phase of cardiac electrical activation is the spread of the depolarization wave through the right and left atria, followed by atrial contraction. Next, the impulse stimulates pacemaker and specialized conduction tissues in the atrioventricular (AV) nodal and His-bundle areas; together, these two regions constitute the AV junction. The bundle of His bifurcates into two main branches, the right and the left bundles, which rapidly transmit depolarization wavefronts to myocardium of ventricles by way of Purkinje fibers. The main left bundle bifurcates into two primary subdivisions, a left

anterior fascicle and a left posterior fascicle. The depolarization wave fronts then spread through the ventricular wall, from endocardium to epicardium, triggering ventricular contraction. Ventricular depolarization can be divided into two major phases, each represented by a vector. The first phase denotes depolarization of the ventricular septum, beginning on the left side and spreading to the right. Simultaneous depolarization of the left and right ventricles (LV and RV) constitutes the second phase.

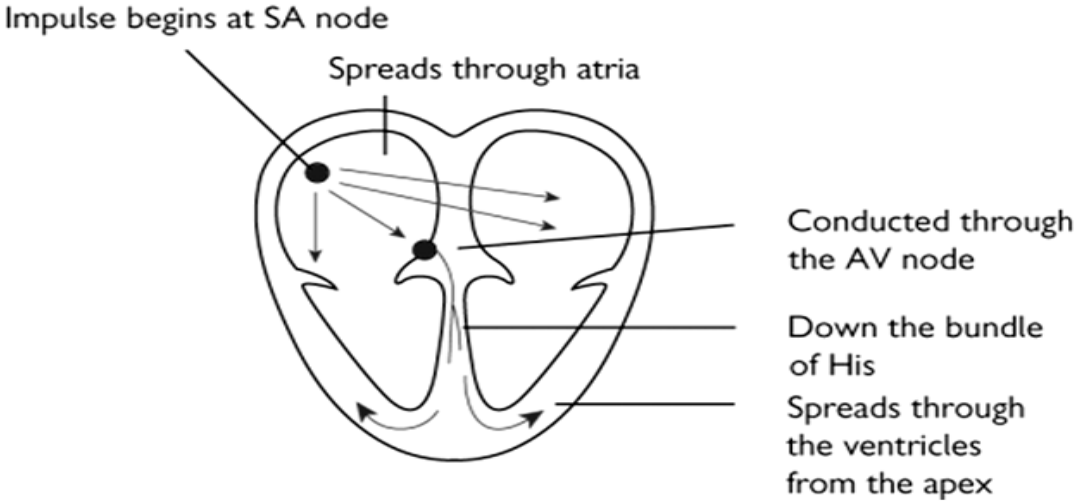


Fig. 11-1. Cardiac conduction system:

SA – sinoatrial node, AV – atrioventricular node

11.2. ECG registration

ECG registration is carried out with the help of special devices (electrocardiographs), which convert electrical oscillations into mechanical ones. Biopotentials are taken by means of electrodes applied to different areas (leads) (Fig. 11-2, Table 11-1).

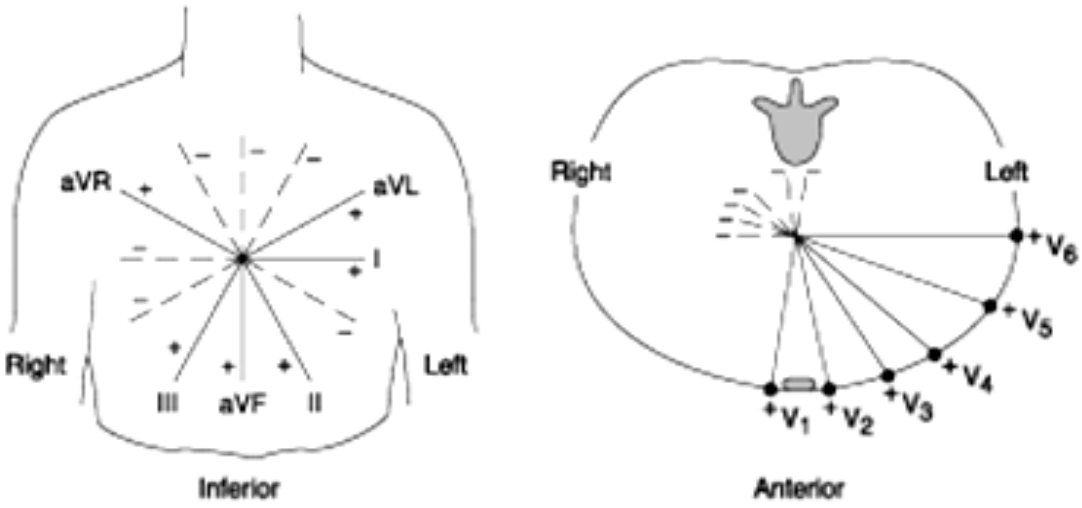


Fig. 11-2. ECG-leads

Table 11-1. Basic leads of ECG

Leads	Position of an electrode	Projection of heart chambers
Standard leads I	Right arm - left arm	Anterior wall of the left ventricle
II	Right arm - left foot	-/-
III	Left foot - left arm	Posterior wall of the left ventricle and the right ventricle
Augmented leads aVL	Left arm	Anterior wall of the left ventricle
aVR	Right arm	-/-
aVF	Left foot	Posterior wall of the left ventricle and the right ventricle
Chest leads V1	right sternal edge- the 4th intercostal space	Anterior wall of the right and the left ventricle
V2	left sternal edge - the 4th interspace	Anterior part of the interventricular septum
V3	between V2 and V4	Anterior wall of the left ventricle to apex
V4	left midclavicular line – the 5th interspace	Apex of the left ventricle
V5	left anterior axillary line – the 5th interspace	Lateral wall of the left ventricle
V6	left midaxillary line – the 5th interspace	Lateral wall of the left ventricle

The six frontal plane and six horizontal plane leads provide a three-dimensional representation of cardiac electrical activity. The frontal plane leads – standard (I, II, III) and augmented leads (aVR, aVF, aVL)**.** The horizontal plane leads – chest leads (V1-V6).

11.3. Normal ECG

Basic ECG waves and intervals

Normal ECG consists of a number of waves, segments and intervals reflecting the spread of excitation in myocardium. ECG interval is the interval from the beginning of one wave to the beginning of another wave. ECG segment is the interval from the end of one wave to the beginning of the next wave.

The waves are denoted by the Latin letters P, Q, R, S, T and U (Table 11-2, Fig. 11-3). Some of the waves are directed upward (R, U - always; P and T -

mainly) and are therefore considered as positive (+), some teeth (Q and S - always, P and T – in some cases) are directed downward and therefore considered negative (–). The analysis of waves is carried out according to their size (amplitude) and width.

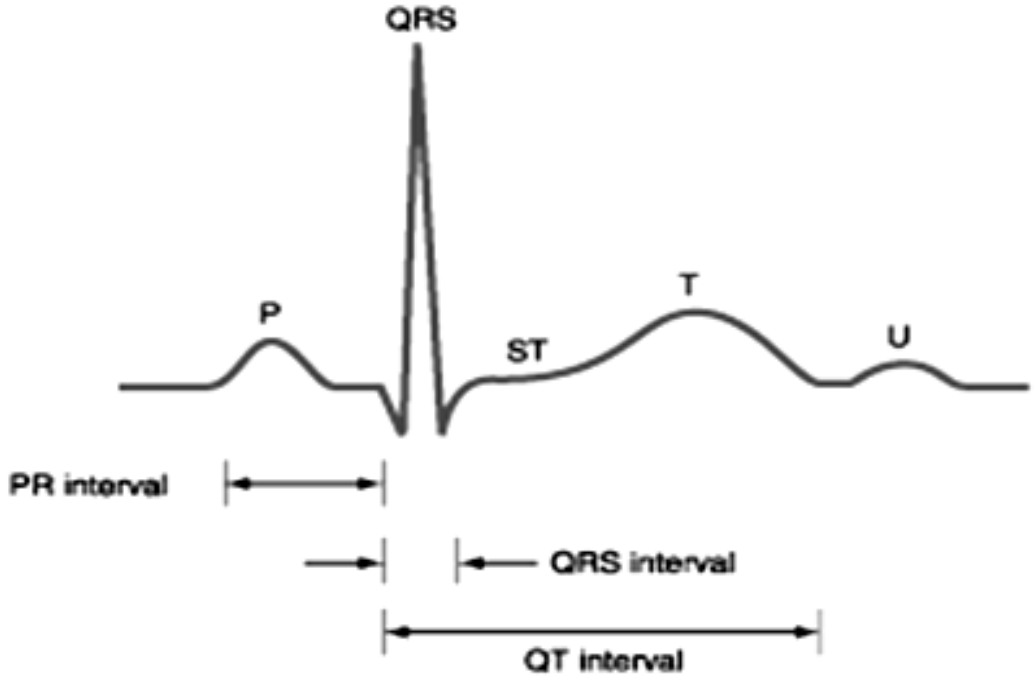


Fig. 11-3. Basic waves and intervals of normal ECG

Table 11-2.Waves and intervals of normal ECG

Waves Intervals	Width (s)	Height (mm)	Other characteristics
P	≤0.10	≤2-2.5	(+) I, II, aVF, (-) aVR may be (±) III, aVL, V_{1-2}
PQ	0.12—0.20	-	isoline
QRS	0.06—0.10	≥5 all leads; ≥ 10 chest leads	Q (<0.04 s, ≤2 mm) except aVR, V_{1-2} ; transition zone (R wave = S wave) between V_2 –V_4
ST	-	-	isoline (as rule); may be - (+) 1 or (-) 0.5 mm in aVL, aVF. aVR; oblique (+) 3 mm in V_{1-6}
T	-	≤1/4-1/2 R	(+) I, II, V_3—V_6; (-) aVR, V_1; may be(±) III, aVL, aVF, V_{1-2}
QT	0.30—0.46 correlates HR	-	QT = K/√RR (<0.46-0.47 s, K=0.37 for men and 0.39 for women)

Generation of ECG waves and intervals: P – depolarization of atriums, QRS - depolarization of ventricles, ST, T, U - repolarization of ventricles. The QRS-T complex corresponds to different phases of electrical systole of the heart.

P wave reflects the passage of excitation by the atria. Normally P wave is always positive in I, II, aVF, V2 – V6, and P wave is always negative in aVR. Amplitude of P wave does not exceed 0.25 mm, duration – 0.11 seconds.

P-Q interval is measured from the beginning P wave to the beginning of the QRS complex. It reflects the duration of conduction by the atria to an atrioventricular node. The duration of P-Q interval is from 0.12 to 0.20 seconds.

Ventricular QRST complex

QRS complex reflects the process of propagation and extinction of excitation (depolarization) in ventricular myocardium. Its duration does not exceed 0.1 sec.

Q wave is normally a negative wave recorded in all standard, augmented and thoracic V4 – V6 leads. Amplitude of the Q wave does not exceed 1⁄4 of the height of the proper R wave, the duration is not more 0.03 seconds. With a greater amplitude of the Q wave, it is said about "pathological Q wave".

R wave can be registered in all standard and augmented leads. In thoracic leads, amplitude of the R wave increases from V1 to V4, and it decreases slightly in V5 and V6.

Normal the **S wave** amplitude may vary in the range of 20 mm. In thoracic leads, the S wave gradually decreases from V1 to V4, and it may be absent in V5 and V6. Equality of the R and S waves in thoracic leads ("transition zone") is usually recorded in V3 (less often in V4).

RS-T segment - interval from end of the wave R (or S) to the beginning of the wave T. It corresponds to the period of complete excitation of both ventricles. RS-T segment is normally located on isoelectric line (± 0.5 mm) in the leads from the limbs. Normally, in V1-V3 leads, displacement of this segment is not more than 2 mm up, and in V4 - V6 – down is not more than 0.5 mm.

The T wave reflects the process of ventricular repolarization. Normally, the T wave is always positive in standard, aVF, V2 - V6 leads. The T wave is always negative in aVR and can be positive, two-phase, negative - in III, aVL, V1 leads.

Q-T interval is measured from the beginning of Q wave (or R in the absence of Q) to end of T wave. This interval displays **electrical systole of ventricles**: their depolarization (QRS complex), and repolarization (T wave) (Fig. 11-4).

Normal QT equals 340-450 msec for men and 340-470 msec for women. The duration of Q-T depends on the heart rate. Q-T prolongation is associated with an increased risk of fatal arrhythmia - ventricular tachycardia. Q-T

shortening is a group of very rare hereditary syndromes that also lead to a sudden death due to the paroxysmal ventricular arrhythmias.

In some cases, a small positive **wave U** is registered after the T wave in 0.02 – 0.04 seconds. The wave U is a reflection of the trace potential of excitability of ventricular myocardium after its systole. It has no great diagnostic value.

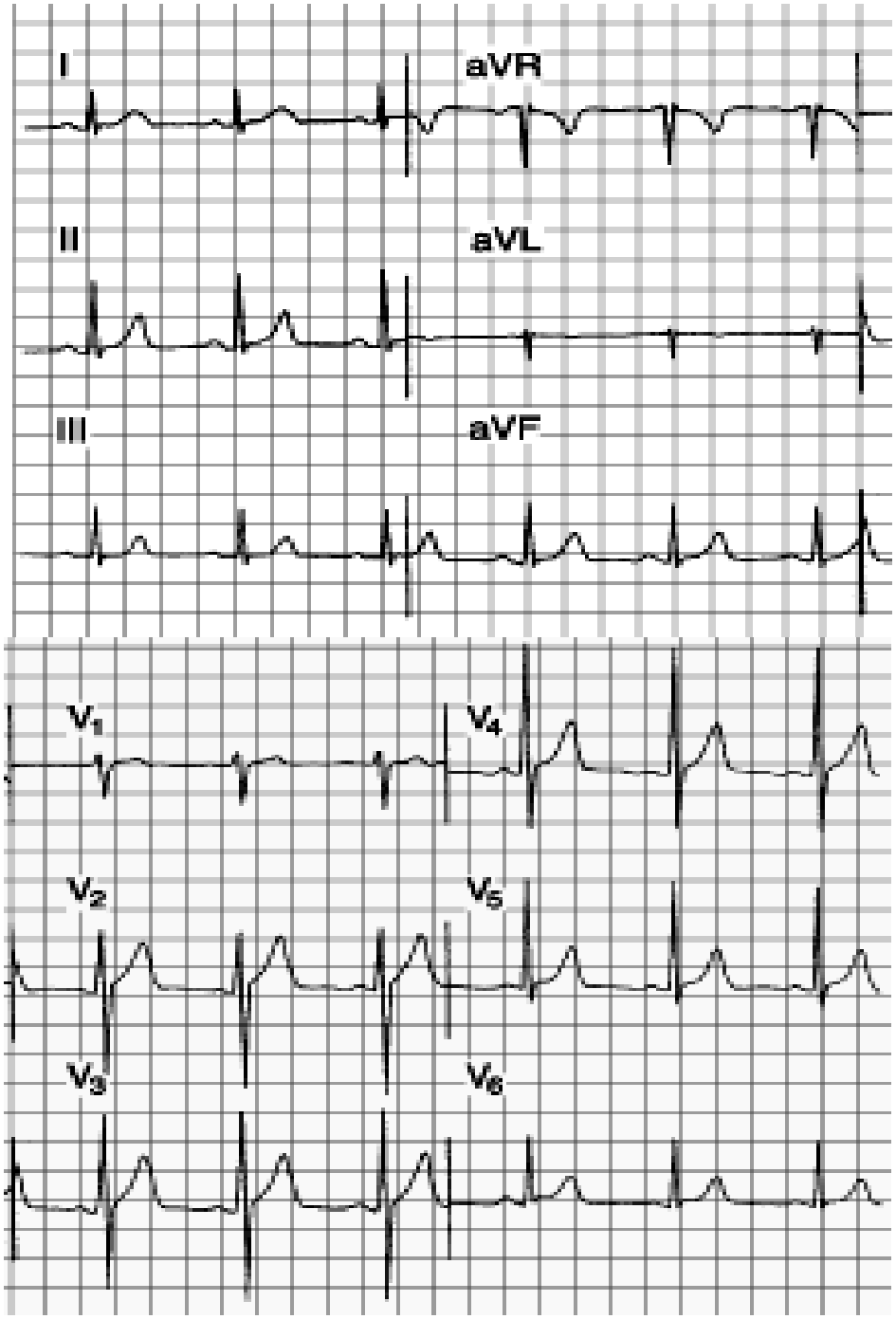


Fig. 11-4. Normal ECG of the healthy person:

Sinus rhythm is present with a heart rate of 75 per minute. PR interval is 0.16 s; QRS interval (duration) is 0.08 s; QT interval is 0.36 s; the mean QRS axis is about +70°, transition zone V3.

A horizontal isoelectric line (**interval T – P**) is normally recorded after the T or the U wave, if any, until the P wave of the next cardiac cycle. Interval T – P corresponds to **electrical diastole** of the heart.

11.4. Interpretation of ECG

At the beginning of the interpretation of ECG, technical conditions of tape recording must be defined (voltage of the ECG and speed of a tape). The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis. It is important for correct estimation of a heart rate, amplitude and duration of ECG waves and intervals.

The sequence of ECG analysis:

1. **Voltage of the ECG** is estimated in compliance with a standard size of 1 mv =10 mm.

2. **Speed of tape**. If the speed of the tape is 50 mm/sec – 0.02 s in 1 mm of tape (width of QRS=3-4 mm). If the speed of the tape 25 mm/sec – 0.04 s in 1 mm of tape (width of QRS=1-2 mm) (Fig. 11-5).

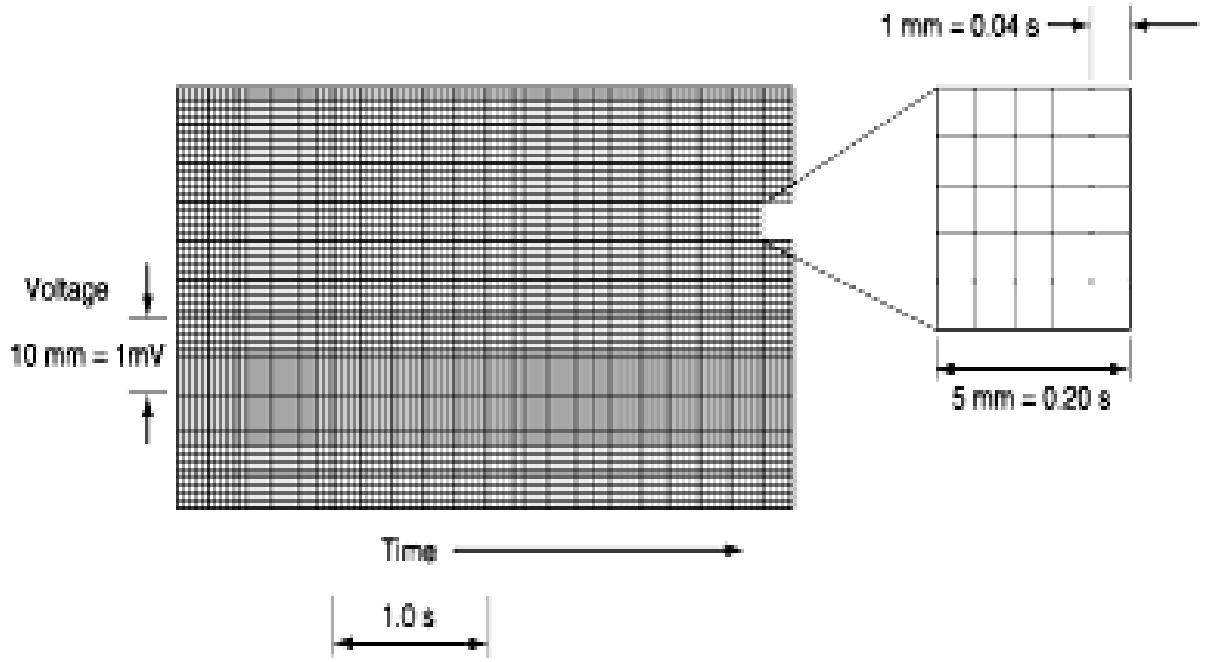


Fig. 11-5. The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis

3. **Regularity and pacemaker of the cardiac rhythm**

Regularity of cardiac contractions is estimated in comparison of the duration of the intervals R-R between the cardiac cycles in the corresponding lead. **Regular rhythm** is diagnosed if the duration of R-R intervals is the same.

Sinus rhythm signs: the P-wave is positive in II standard lead and corresponds (previous) to complexes QRS.

4. **Heart rate (HR)**

$$HR = \frac{60 \text{ (seconds in 1 minutes)} \times 25 \text{ (or 50) (mm/sec)}}{[R\text{-}R] \text{ (mm)}},$$

where HR – heart rate;

R-R - the distance between the two nearest R waves (R-R interval);

25 (or 50) (mm/sec) - speed of tape recording.

If an irregular heart rhythm, an average heart rate is determined, or is specified a minimum and maximum frequency of the heart contractions. Other variant of HR determination - multiply by 20 times (×20) the number of R-R intervals during 3 seconds (Fig. 11-6).

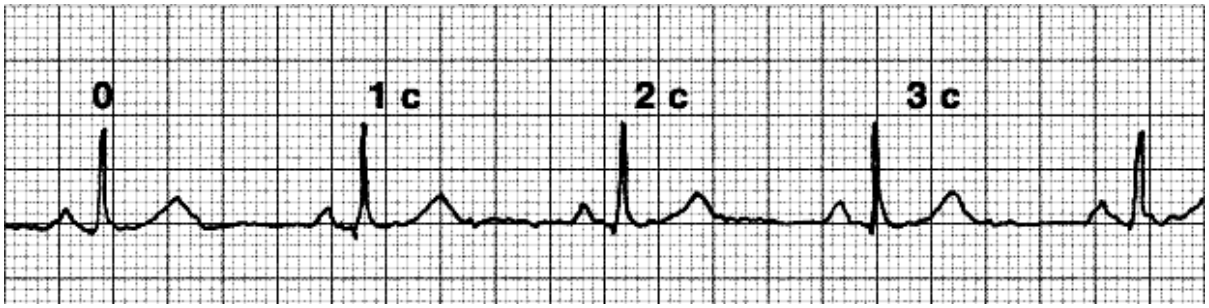


Fig. 11-6. HR determination: the number of R-R intervals during 3 seconds is multiplied in 20 times (×20)

5. **Analysis of myocardial conduction depending duration of***:*

- P wave (≤0.10-0.12 s) – intraatrial conduction;
- PQ interval (0.12-0.20 s) – conduction in atrioventricular node;
- QRS complex (0.06-0.10 s) - His bundle (intraventricular) conduction.

6. **Determination of the position of electrical axis of the heart**

Electrical axis of the heart is a direction of the electromotive force of the heart over a period of depolarization (the time of registration of the complex QRS). The angle α is formed by electrical axis of the heart with I standard lead (Fig. 11-7, 11-8).

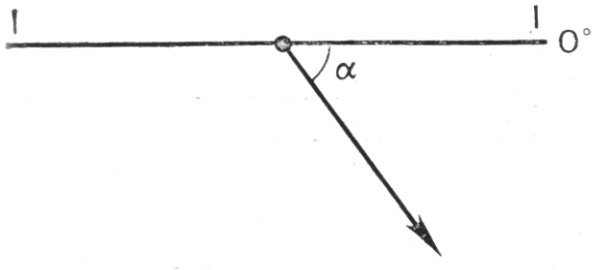


Fig. 11-7. Determination of the angle α.

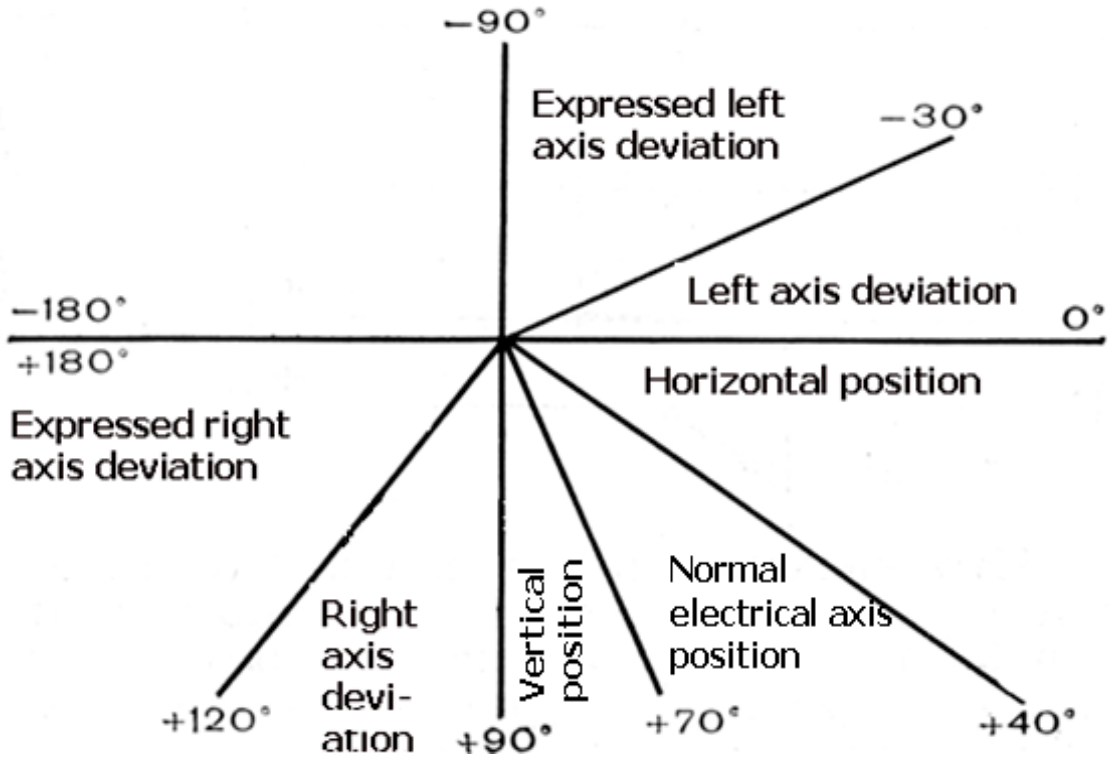


Fig. 11-8. Determination of the electrical axis of the heart

Variants of electric axis of the heart:

- **normal electrical axis** - angle α equals 40° - 70°, the wave RII highest, the ratio of waves RII> RI> RIII;
- **horisontal electrical axis,** or **levogram** - angle α equals 0°- 40°, the wave RI highest, the ratio of waves RI> RII> RIII;
- **vertical electrical axis°, *or* dextrogram** – angle α equals 70°- 90°, the wave RIII highest, the ratio of waves RIII> RII> RI.

Horizontal, vertical position and normal electrical axis of the heart are observed in healthy people.

The **right axis deviation** (angle α> + 90°) and expressed right axis deviation (angle α> + 120°, the ratio of waves: RIII> RII> RI, SI> RI) present in right ventricle hypertrophy and dilation, right His bundle brunch block, pulmonary heart, tricuspid and pulmonary artery valves diseases.

The **left axis deviation** (angle α <0) and expressed left axis deviation (α <-30°, the ratio of waves: RI> RII> RIII, SIII> RIII) present in left ventricle hypertrophy and dilation, left His bundle brunch block, aortal valves diseases.

7. Analysis of ECG waves and intervals

The duration, amplitude and shape of ECG waves are assessed as well as the position of relative to the isoelectric line for all intervals and segments of ECG (see Table 11-2). A heart rate can be estimated in the intervals between ECG complexes at a certain speed of movement of a cardiograph tape, and in the intervals between the waves - duration of the phases of cardiac activity and

the state of intra-cardiac conduction. By voltage, i.e. the amplitude, of distinct ECG waves in proper leads, you can judge on the electrical activity of certain parts of the heart and, above all, on the mass of their myocardium.

8. Electrocardiographic conclusion is based on the analysis of all waves, intervals and segments with an obligatory account of the clinical data.

11.5. ECG in hypertrophy of atriums

Atrium hypertrophy is detected by change of the P wave (Fig. 11-9, 11-10).

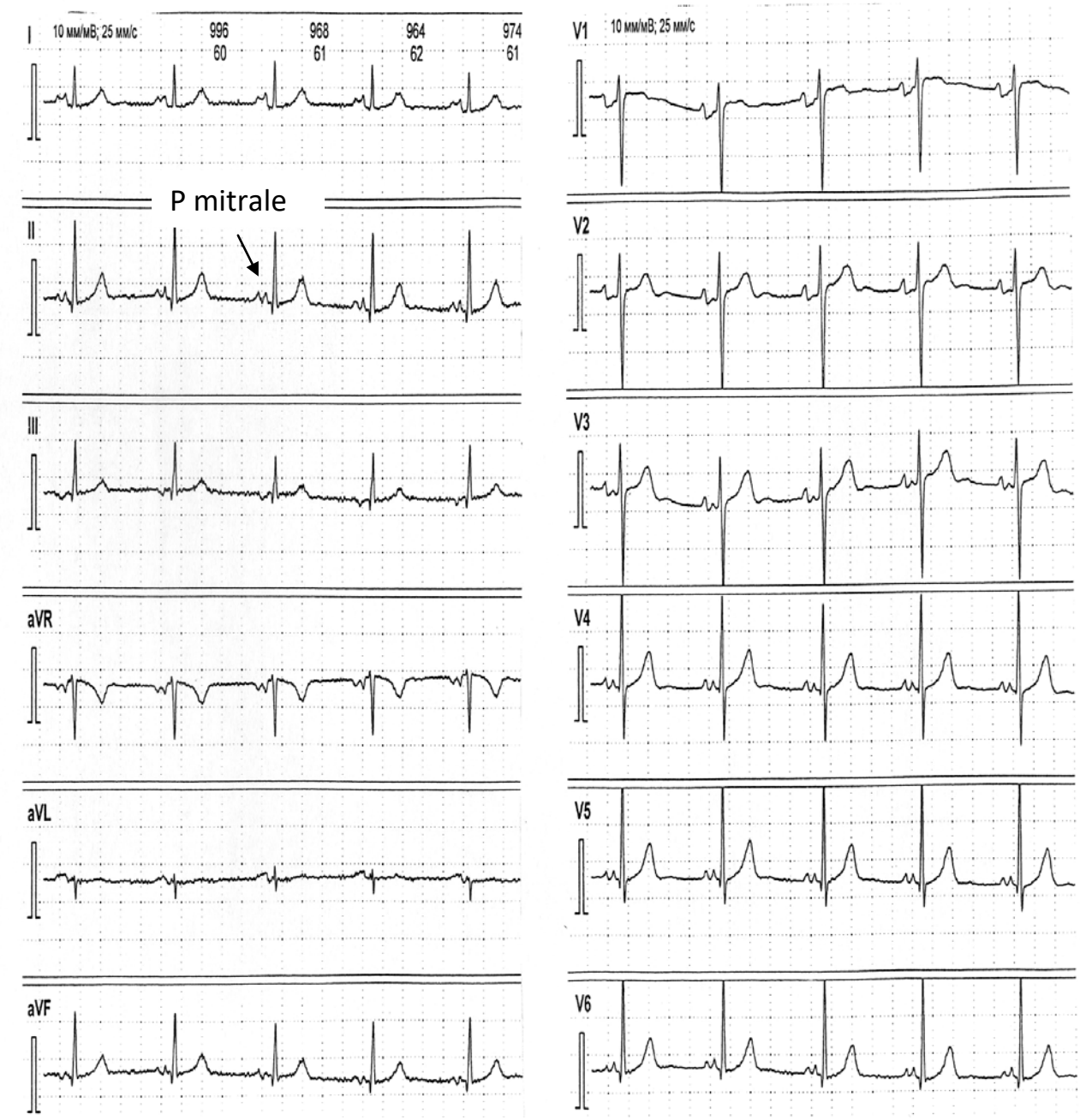


Fig. 11-9. ECG in hypertrophy of the left atrium:
the P wave is over 0.11 seconds, two-humped (*P-mitrale*) in I, II, left chest leads V_{5-6}; biphasic or negative (>1 mm) P in right chest leads V1-V2

Left atrium hypertrophy is detected by "**P mitrale**": wide (>0.1 s), splitted P-wave in I, II, AVL, left chest leads (V5-6); flat or negative P in III, biphasic or negative (>1 mm) P in V1. Left atrium hypertrophy is typical in mitral valves diseases (mitral stenosis and mitral incompetence).

Right atrium hypertrophy is detected by "**P pulmonale**": high (>2.5 mm) acute P in II, III, AVF and right chest leads (V1-2). Right atrium hypertrophy is typical in chronic pulmonary diseases (pulmonary heart) and tricuspid valves incompetence.

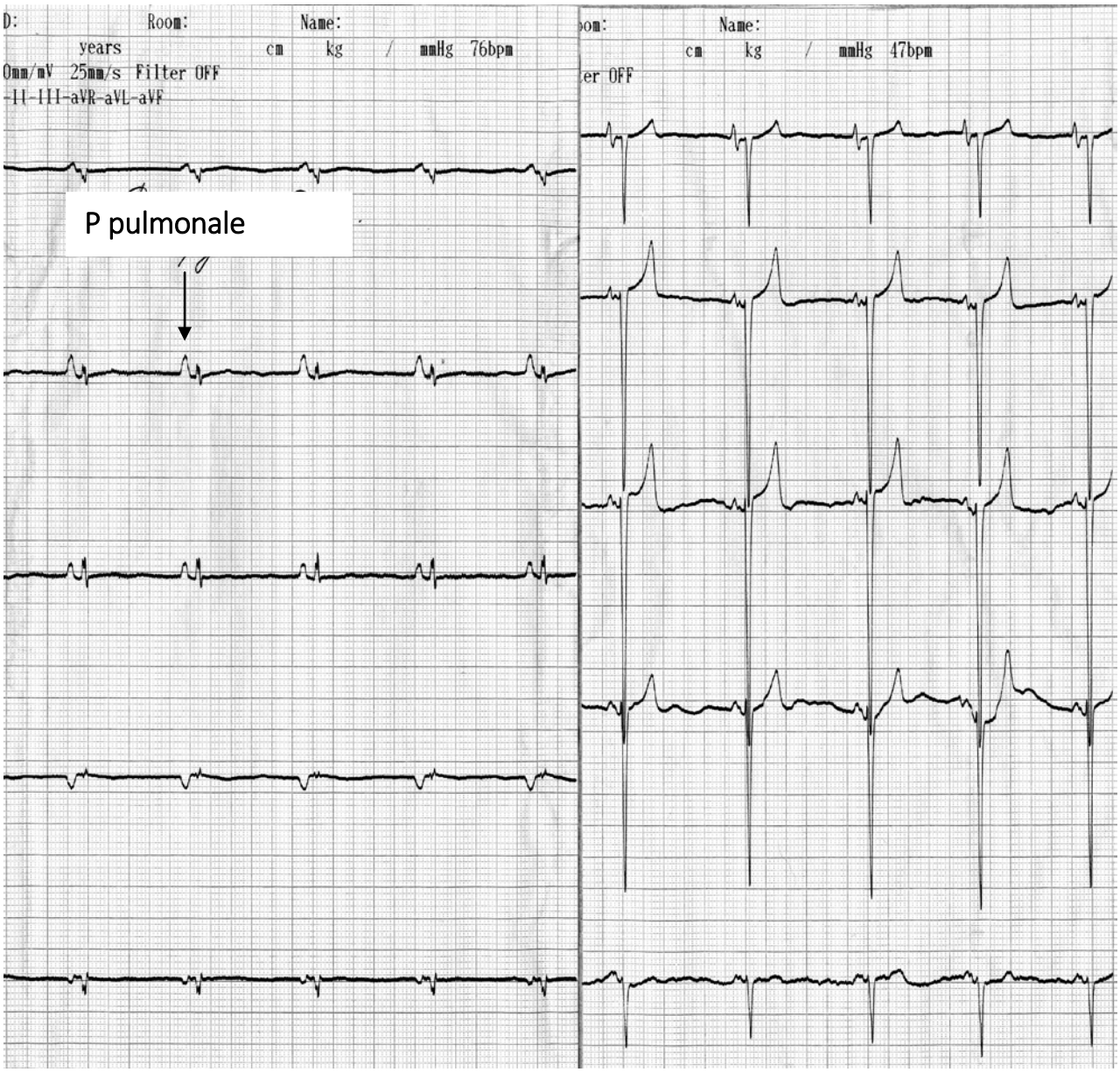


Fig. 11-10. ECG in the right atrium and right ventricle hypertrophy: the high P wave (*P-pulmonale)* > 2.5 mm in II standard lead (ECG-sign of right atrium hypertrophy) and ECG-signs of the right ventricle (right axis deviation - RIII> RII> RI, a deep S wave in leads I, αVL, left chest leads V5-V6)

11.6. ECG in hypertrophy of ventricles

Hypertrophy of ventricles is determined mainly by changes of ventricular complex QRS, the changes in the S and R waves are the reverse.

ECG signs of left ventricle hypertrophy:

- **the most important ECG sign of left ventricle hypertrophy is increase in amplitude of R $_{V5-6}$>R $_{V4}$** (Fig. 11-11);
- amplitude R $_{V5-6}$>25 mm;
- similar changes in R I, aVL;
- deepening of S $_{V1-V2}$ ≥10-25 mm;
- transition zone shift (R=S) to right chest leads (from V3 to V1-2);

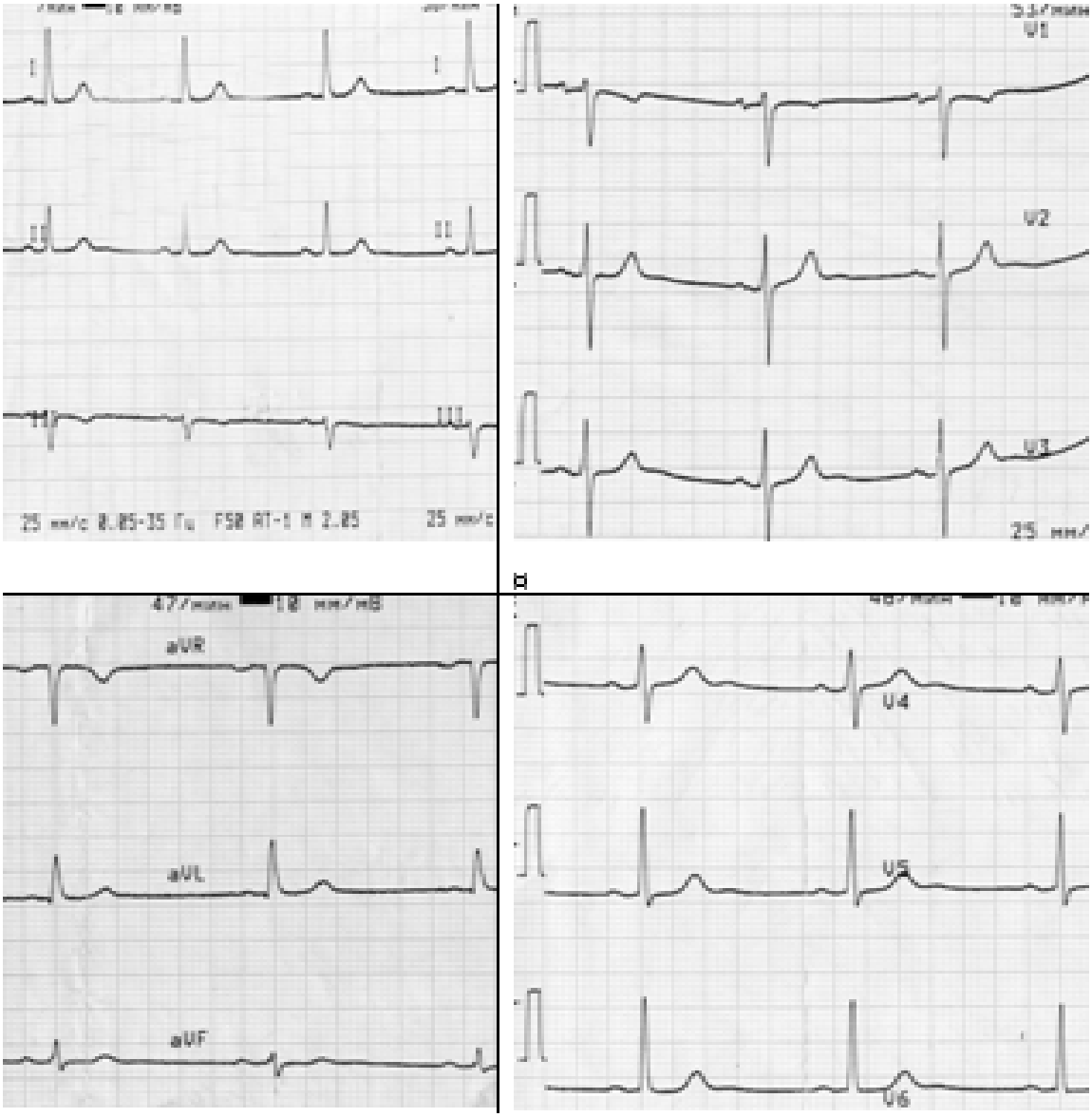


Fig. 11-11. ECG in the left ventricle hypertrophy:
high waves *R* $_{V5-6}$>R $_{V4}$; deep S wave in V_{1-2}, levogram (RI> RII> RIII)

- levogram RI> RII> RIII
- downward shift of ST segment and formation of a two-phase or negative T wave in I , aVL, V5 and V6 leads;
- sum of amplitude S_{V1} + R_{V5} (or R_{V6})≥35 mm.

ECG signs of right ventricle hypertrophy:

- **the most important ECG sign of the right ventricle hypertrophy is an increase in the amplitude of R_{V1-2} wave ≥ 7 mm (Fig. 11-12)**;
- deep wave $S_{I, \alpha VL, V4-6'}$;
- high R wave in leads III, aVF;
- dextrogram (RIII > RII > RI);
- the shift of the transition zone (R=S) to the left chest leads (from V3 to V5-6);
- the negative wave T and ST segment shift down in V1-2, III, aVF leads.

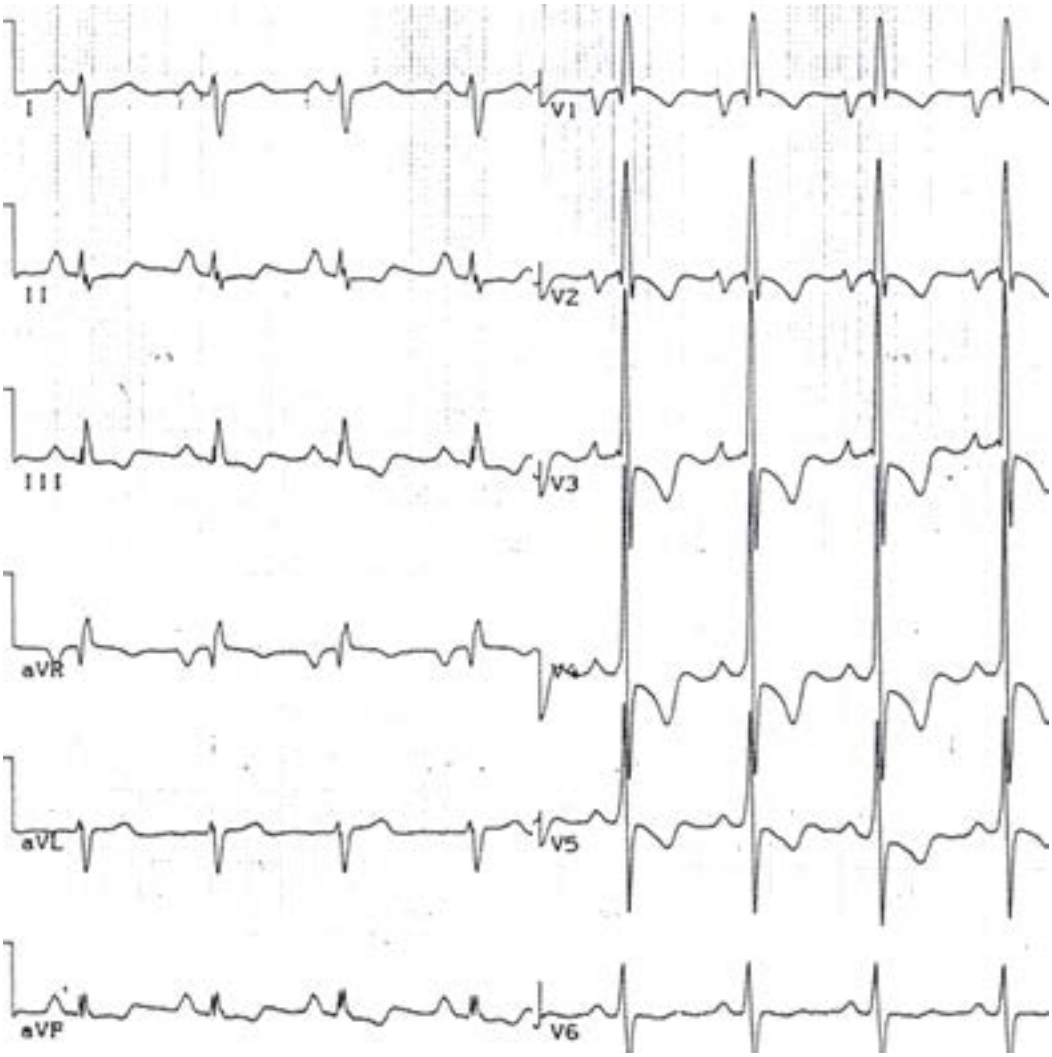


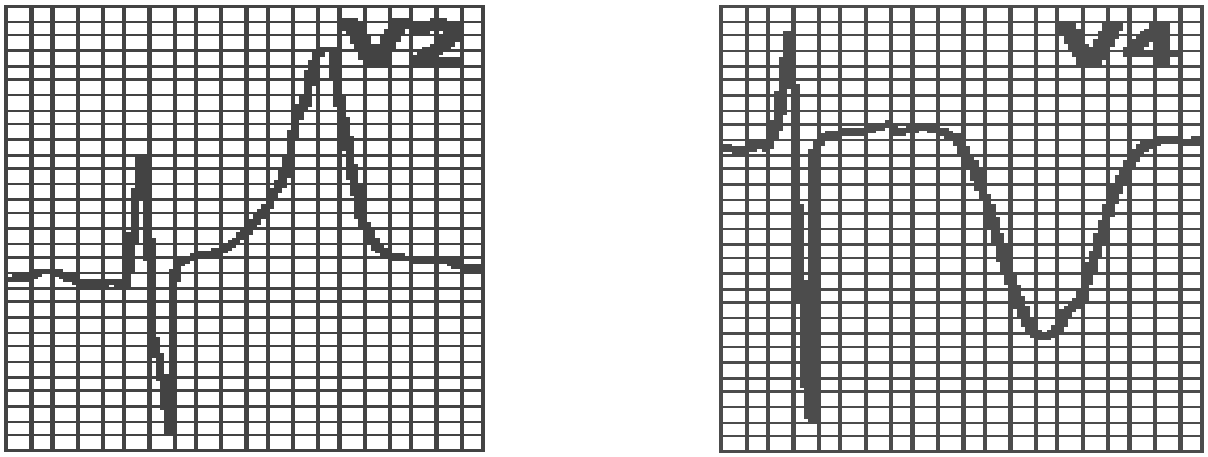
Fig. 11-12. ECG in the right atrium and right ventricle hypertrophy: The high P wave (*P-pulmonale)* > 2.5 mm in II standard lead; high R wave ≥ 7 mm in V_{1-2}; deep S wave in leads I, αVL, V5-V6; dextrogram (RIII> RII> RI)

11.7. ECG in the Ischemic Heart Disease (IHD)

Ischemia is a short-term disturbance of the blood supply to certain areas of the myocardium. Under the influence of the ischemia processes of repolarization are retarded.

ECG signs of myocardial ischemia are various changes in the shape and polarity of the T wave and ST- interval. The T wave may become high, wide and symmetrical (the ***positive "coronary" T wave***), negative symmetrical with a pointed apex (the ***negative "coronary" T wave***), two-phase (+ ─ or ─ +). Depending in which ECG-leads, the T- wave changes appear, a doctor can judge the localization of ischemia.

Subendocardial ischemia is detected by symmetrical acute high T-wave in overlying leads (>6 mm in standard and augmented leads, >8-10 mm in chest leads). Transmural or epicardial ischemia is detected by symmetrical acute deep T-wave (Fig. 11-13).



(1) (2)

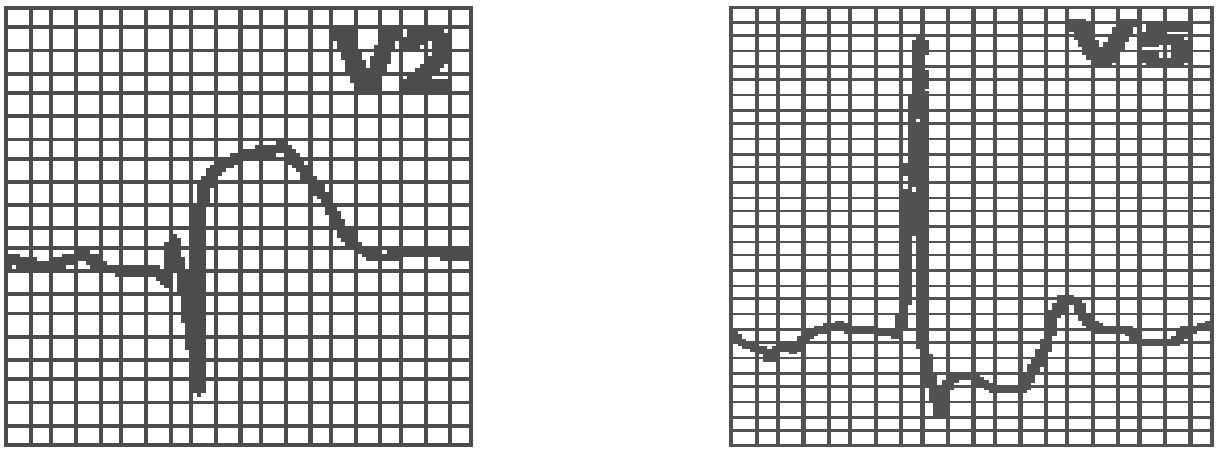
Fig. 11-13. Ischemic changes of the T-wave:
(1) positive "coronary" T wave in subendocardial ischemia;
(2) negative "coronary" T wave in transmural or epicardial ischemia

Severe acute ischemia of myocardium causes a current **ischemic damage of myocardium** (Fig. 11-14). With more prolonged circulatory disorders of this or that portion of the myocardium, it develops ischemic damage. The reversible morphologic damage is manifested by disturbances of the myocardium excitation and repolarization.

The main feature of the ECG in ischemic damage of myocardium is the displacement of the RS-T segment above (elevation) or below (depression) the isoline:

Subendocardial ischemic damage *– overlying leads record the ST depression.*

Transmural or epicardial ischemic damage *– the ST elevations and sometimes, at the earliest stages of ischemia, tall, positive the so-called hyperacute T waves over the ischemic zone.*



(1) (2)

Fig. 11-14. Ischemic myocardium injury is detected by the ST-interval:
(1) transmural or epicardial injury - convexing elevation ST;
(2) subendocardial injury – horizontal or concaving depression ST.

ECG in myocardial infarction

Myocardium infarction is characterized by the irreversible changes in myocardial cells, their necrosis. Dead cells do not conduct electrical impulses, and do not participate in the process of excitation, which manifests itself as a violation of the process of ventricular depolarization.

Electrocardiographic examination establishes the presence of myocardial infarction and its localization, the size of affected myocardium, and the stage of process. ***Three zones of myocardial damage in acute myocardial infarction can be detected by ECG: (1) necrotic zone, (2) ischemic myocardium damage zone, (3) and the zone of ischemia.***

Myocardial necrosis is detected by pathologic Q-wave:

pathologic Q-wave is characterized by width≥0.04 s (in V_{4-6} >0.025 s), depth>2 mm or>1/4 R-wave (in V_{4-6} >15%R) (Fig. 11-15).

Ischemic myocardium damage is detected by ST-interval changes:

- *Transmural or epicardial damage – convexing elevation ST with transmission in T-wave (Fig. 11-14);*
- *Subendocardial damage – horizontal or concaving depression ST.*

Ischemia of myocardium is detected by T-wave*:*

- *Subendocardial ischemia – symmetrical acute high T-wave in overlying leads (>6 mm in standard and augmented leads, >8-10 mm in chest leads) (Fig. 11-13);*
- *Transmural or epicardial ischemia - symmetrical acute deep T-wave.*

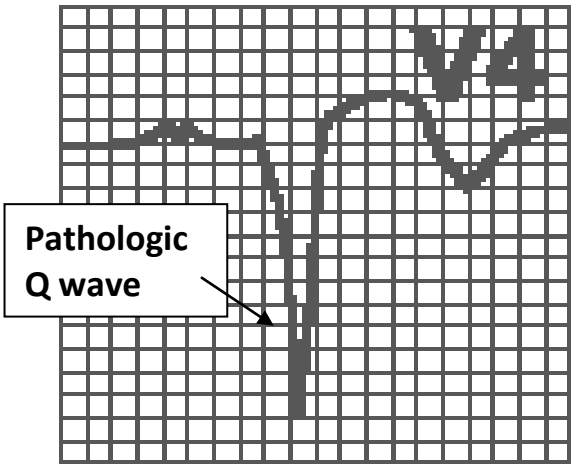


Fig. 11-15. Myocardial necrosis is detected by the pathological Q-wave

Stages of myocardial infarction

Stage of damage*, or (up to 6 hours).* The *S-T* segment and *T* wave change during the first hours of the disease. ST segment rises above the isoelectric line to form a convexing arch, and merges with the positive "coronary" T wave in monophasic curve. R wave is reduced or pathological wave Q apprears: ventricular complexes takes shape *QR or Qr* when non-transmural infarction and QS when transmural infarction (Table 11-3, Fig. 11-16).

Table 11-3. Stages of myocardial infarction

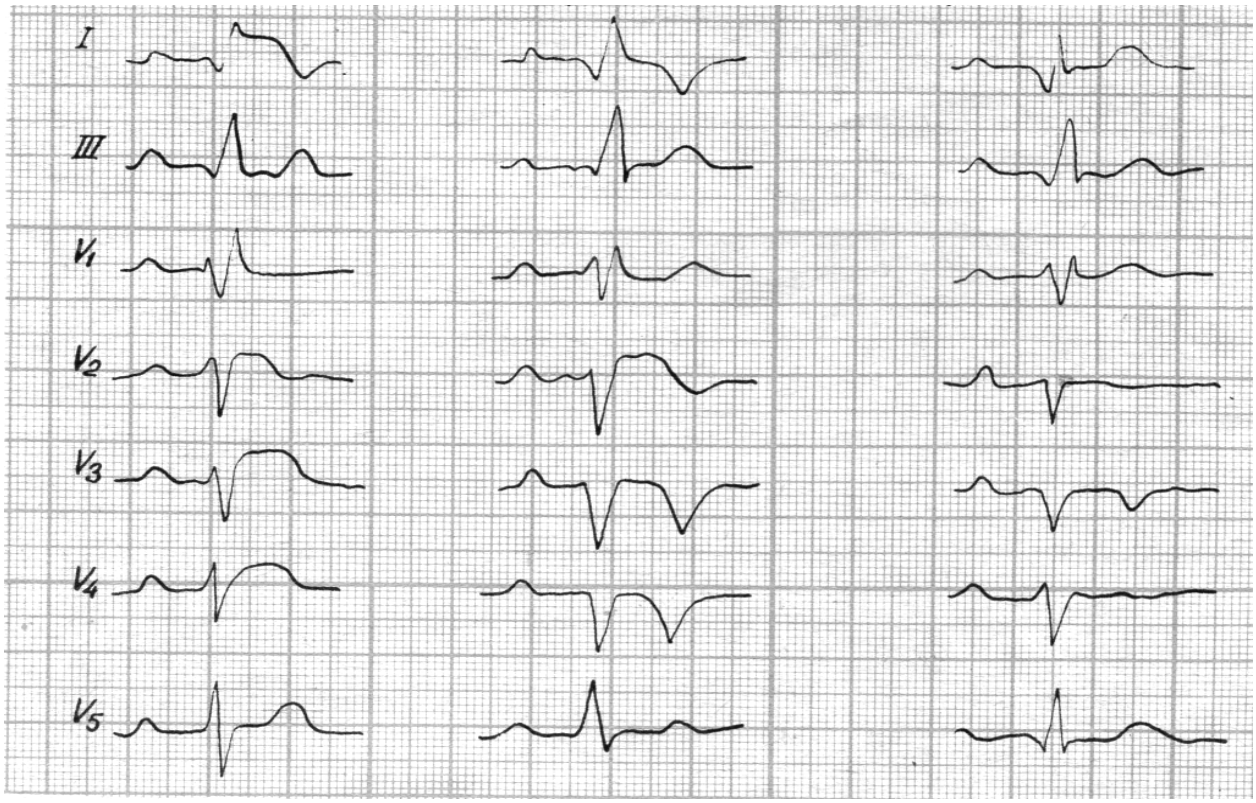
Stage	Changes of ventricular complex	Duration of changes
Ischemic Damage	S-T elevation in form a convex arch coincide with the T wave (monophase curve)	up to 6 hours from the beginning of infarction
Acute	Deep Q, small R-wave, negative T – wave begin be differentiated	up to 7 days from the beginning of infarction
Subacute	Deep Q; S-T returns on an isolectric line; negative ischemic (symmetrical) T	7-28 days from the beginning of infarction
Reduction (cicatrization)	Penetrating and widened Q, negative T	over 29 days from the beginning of infarction

Acute stage *(up to 7 days)* - the depth the of Q wave increases (the zone of myocardial necrosis), the ST segment is approaching isoelectric line (a reducing ischemic damage zone), there is a negative symmetrical "coronary" wave T (the zone of ischemia increases).

Subacute stage (*7-28 days from the beginning of infarction*) - the zone of ischemic damage disappears due to the recovery (transition to a state of ischemia) and loss of the myocardial fibers: ST-segment returns to the isoelectric line, negative symmetrical "coronary" wave T is saved or even increased. Pathological Q wave persists.

Reduction (cicatrization) stage (*over 29 days from the beginning of infarction*). Scar tissue electrophysiologically behaves just as necrotic zone does not create electromotive force, the damaged area of ischemia disappears: preserved pathological wave Q, ST segment on the isoelectric line, wave T becomes slightly negative or positive smoothed.

The initial shape of ECG can be restored during a cicatrization stage, or the changes may remain for the rest of life.



(1) (2) (3)

Fig. 11-16. ECG in stages of myocardium infarction:
(1) – initial (damage) stage; (2) - acute stage; (3) - reduction (cicatrization) stage

Topical diagnosis of myocardial infarction

Depending on localization of infarction, changes of the ventricular complex are observed in the corresponding leads (Table 11-4; Fig. 11-17, 11-18).

Table 11-4.Topical diagnosis of myocardial infarction

Pathological changes of QRST	Localization of myocardial infarction
V1-4, I, aVL	Anterior wall of LV
V1-2, and disappearance of septum Q in leads V5-6	Anterior part of interventricular septum
I, aVL, V5-6	Lateral wall of LV
II, III , aVF	Posterior (inferior diaphragmatic) wall of LV
V7-V9, and high R in V1-2	Posterior (superior basal) wall of LV

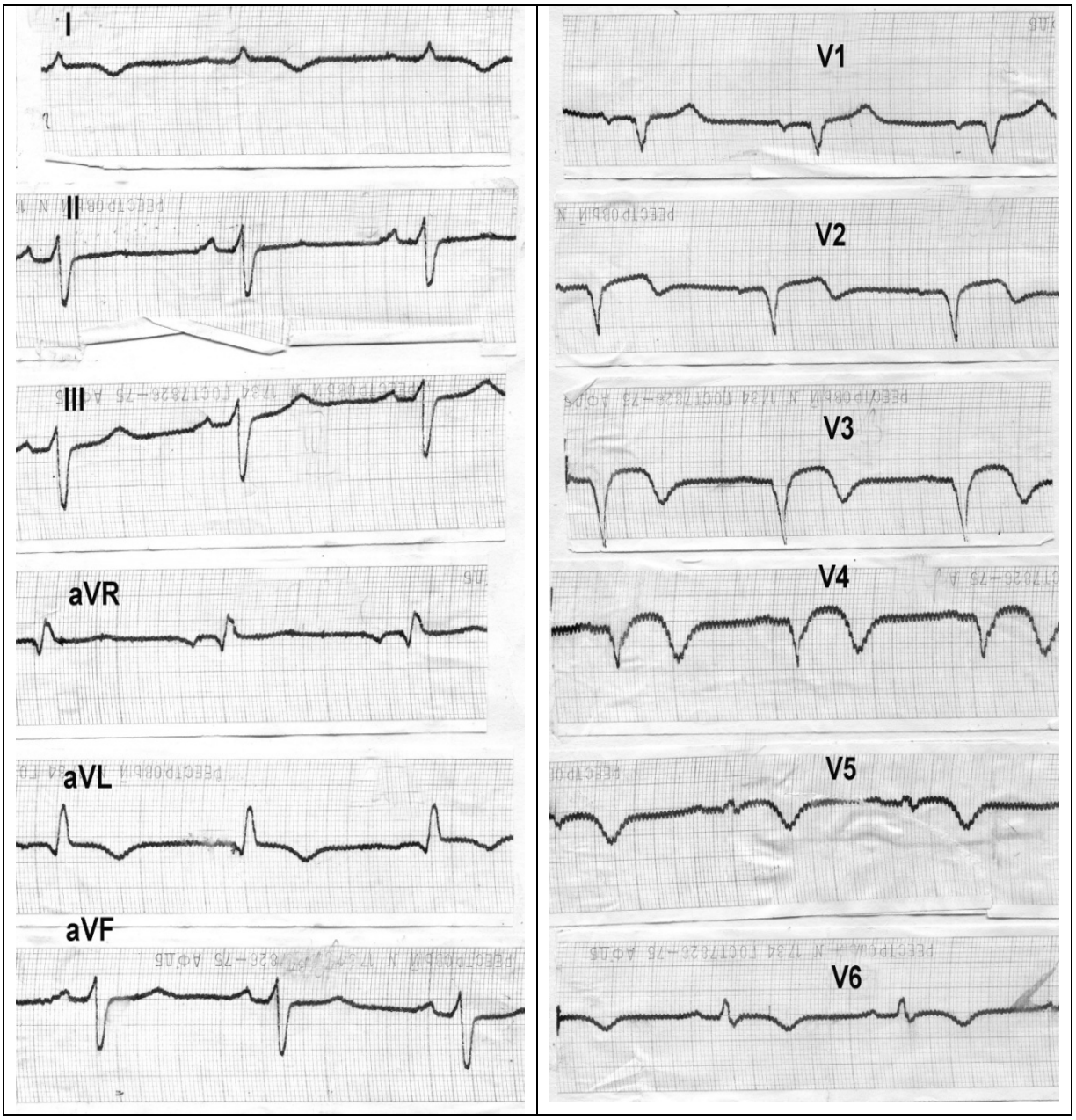


Fig. 11-17. Acute myocardial infarction of the anterior wall and the anterior part of interseptum (I, αVL, V1-V3) with the transition to the apex (V4) and lateral wall (V5-6) of the left ventricle

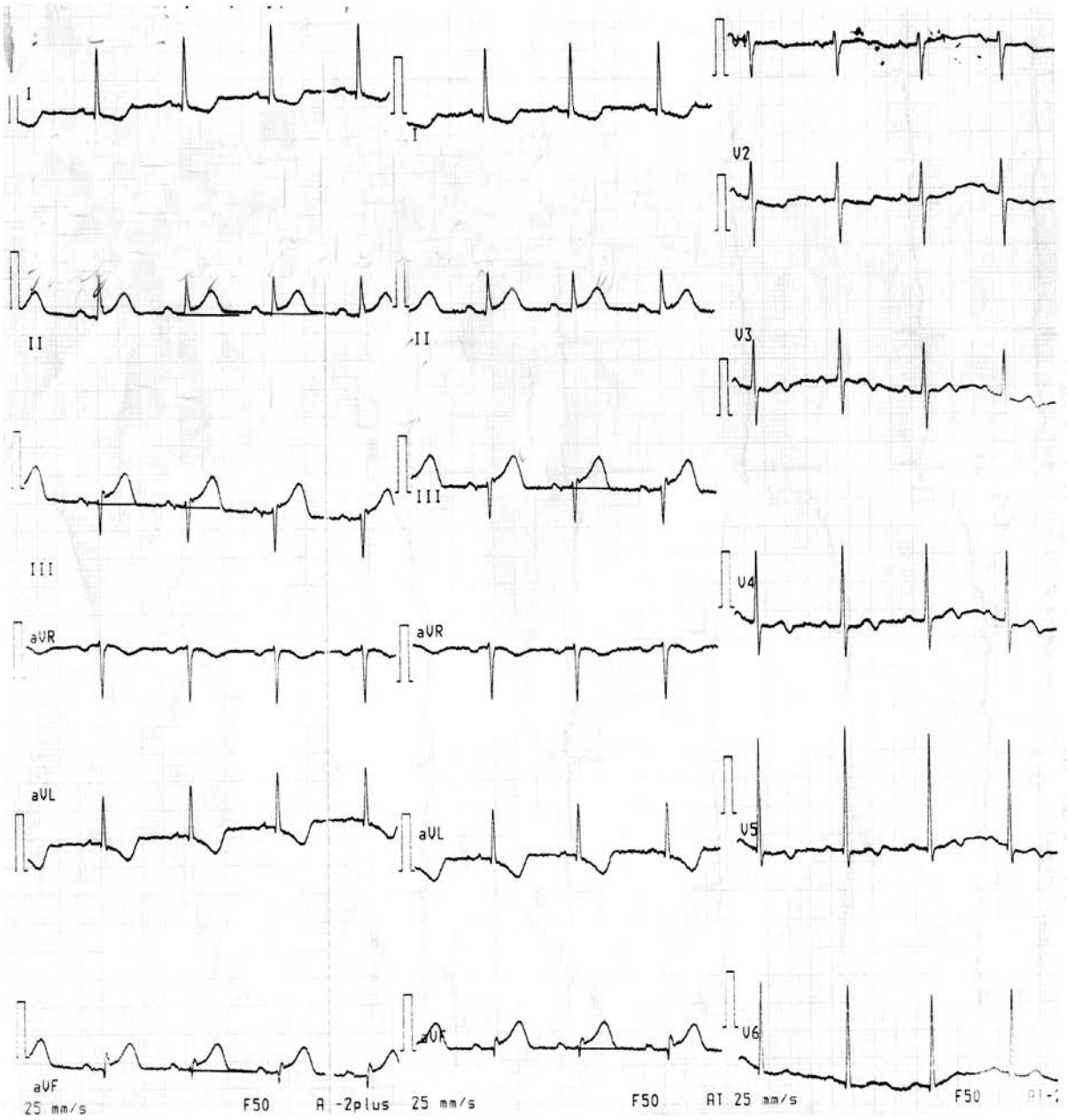


Fig. 11-18. Acute posterior (inferior diaphragmatic) myocardial infarction (II, III, αVF)

Variability of ECG patterns with acute myocardial ischemia:

- *Non–infarction subendocardial ischemia* – transient ST depressions;
- *Non–infarction transmural ischemia* - transient ST elevation or paradoxical T-wave normalization, some times followed by T-waves inversions;
- *Non–Q-wave (non-ST elevation) infarction* - ST depressions or T-inversions without Q-wave (Fig. 11-19)*;*
- -*Non–Q-wave (ST elevation) infarction* - ST-elevations followed by T-waves inversions;

- *Q-wave – infarction* - Q-wave with hyperacute T-waves/ST-elevations followed by T-waves inversions.

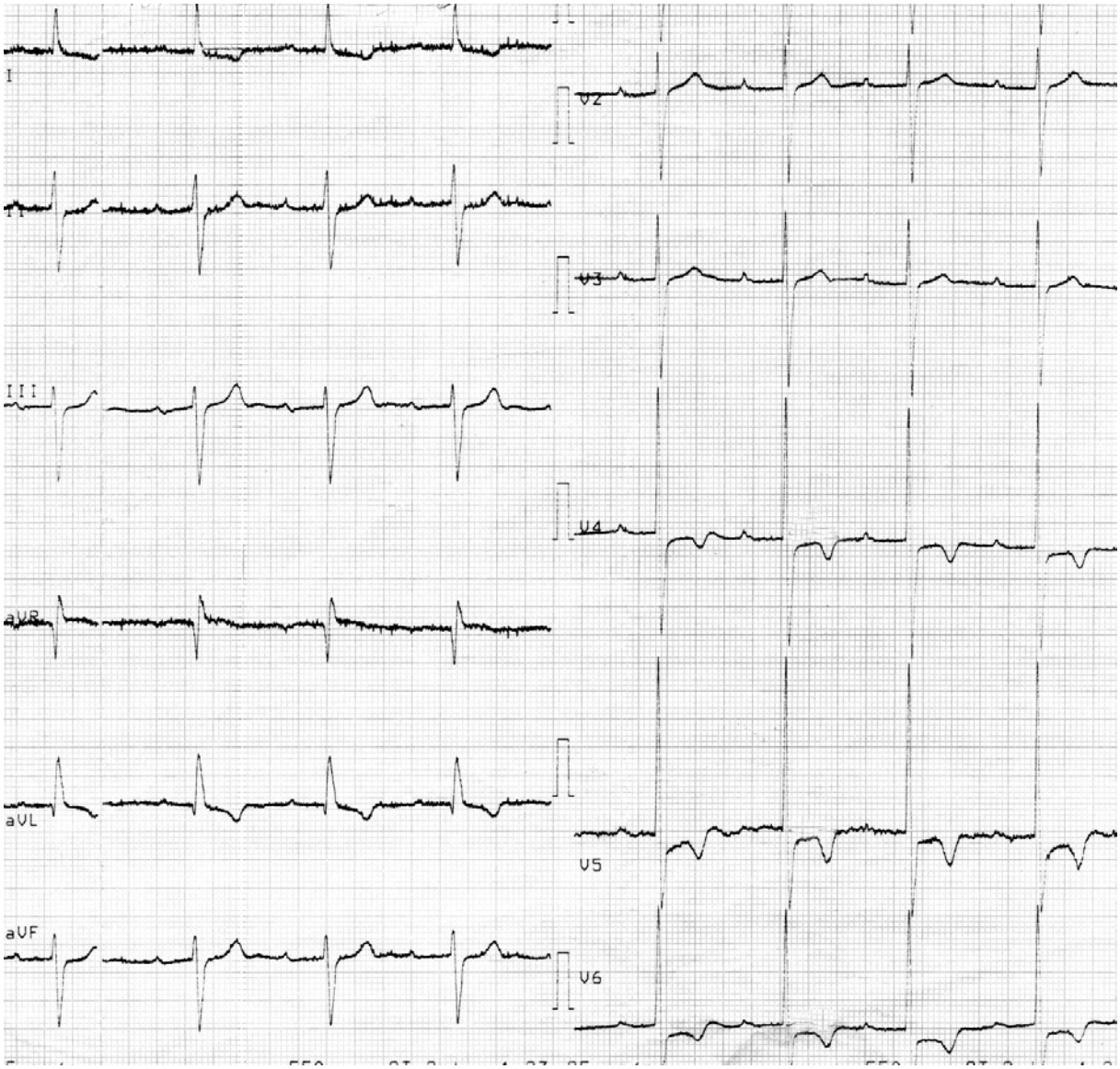


Fig. 11-19. Acute non–Q-wave (non-ST elevation) infarction of the apex and the lateral wall of the left ventricle (horizontal displacement of the ST segment and negative “coronary” T wave in I, αVL, V4-6), atrioventricular block of I degree (interval PQ 0.4 seconds)

Exercise stress ECG testing:

Because the diagnosis of angina pectoris is usually primarily based on the patient's history, exercise testing in a patient with typical symptoms is generally used to determine a functional and ECG response to a graded stress (for the exercise stress testing using radionuclide imaging; for exercise testing in asymptomatic persons to determine fitness for exercise programs, see below).

The patient exercises to a predetermined goal (e.g., 80 to 90% of maximal heart rate, which can be approximated as 220 less the age in years), unless distressing cardiovascular symptoms (dyspnea, reduced endurance, fatigue, hypotension, or chest pain) supervene. The ischemic ECG response during or after exercise is characterized by a flat or downward-sloping ST segment depression>0.1 millivolts (1 mm on the ECG when properly calibrated) lasting > 0.08 sec. (Fig. 11-20).

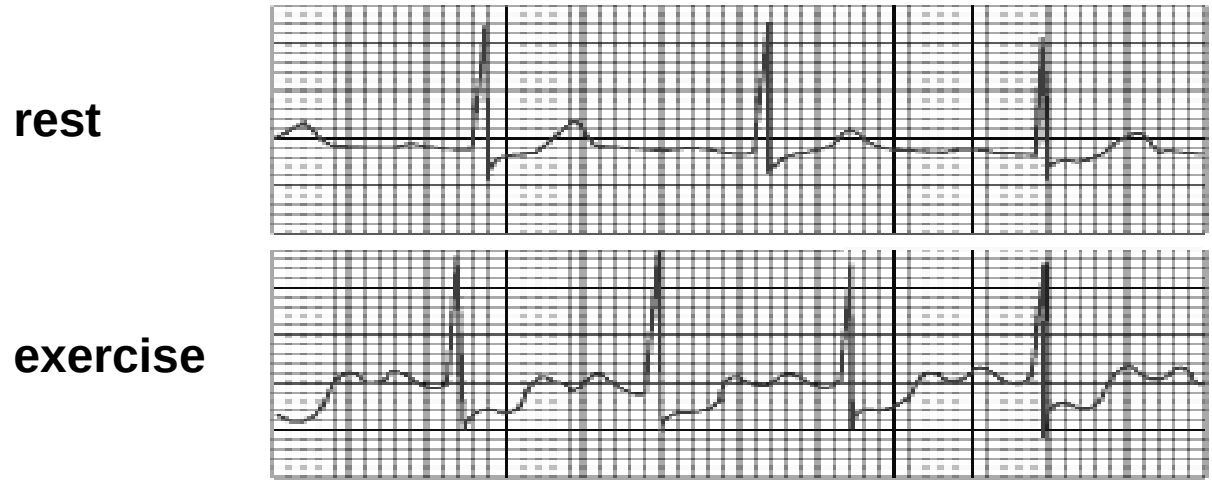


Fig. 11-20. Ischemic ECG response during and after exercise**:**
Lead V4 at rest (*top*) and after 4 1/2 min of exercise (*bottom*). There is 3 mm (0.3 mV) of horizontal ST-segment depression, indicating a positive test for ischemia

11.8. Cardiac arrhythmia – causes, classification

Arrhythmia (from *Greek* - "inconsistency, clumsiness") is a pathological condition that leads to disorders of a heart rate and rhythm, and violation of electrophysiological functions of the heart - automaticity, excitability, conduction, and contractility.

Cardiac arrhythmias are common in many organic and functional disorders of the cardiovascular system.

Causes of cardiac arrhythmias include:

(1) Affected automaticity of the sinus node (nomotopic arrhythmia);

(2) Foci of increased excitability in myocardium (ectopic arrhythmia) ;

(3) Disorders of cardiac conduction system, local conduction disorder (re-entry mechanism);

(4) Combined violation of cardiac electrophysiological functions – automaticity, excitability, conduction.

Re-entry mechanism according to the up-to-date point of view is a most common cause of cardiac arrhythmia. *Re-entry mechanism* means a pathological circuit of the impulse because of electrophysiological inhomogenity (i.e., differences in conduction and/or refractoriness with dystrophic and necrotic

changes of myocardium) in two or more regions of the heart connected with each other to form a potentially closed loop. If the impulse is looped once or twice — there is extrasystole, if three or more - paroxysmal tachycardia, or other types of arrhythmia (for example, atrial flutter or fibrillation).

Classification of cardiac rhythm disorders (arrhythmias):

1. Sinus (nomotopic) arrhythmias due disorders of sinus node automaticity;
2. Ectopic (heterotopic) arrhythmias;
3. Arrhythmias due to disordered myocardial conduction (heart blocks);
4. Combined cardiac rhythm disorders.

11.9. Sinus (nomotopic) arrhythmias

Sinus (nomotopic) arrhythmias are associated with a violation of the sinus (sinoatrial) node automatism, and include ***sinus tachycardia, sinus bradycardia, and respiratory (sinus) arrhythmia***.

Normal sinus rhythm characteristics (Fig. 11-21):

- HR (heart rate) equals 60-80 in min, regular rhythm (differences between the minimal and the maximal R-R intervals is not more than 15%);
- P wave - positive in I, II, aVF, P wave – negative in aVR, PQ ≥0.12 s.

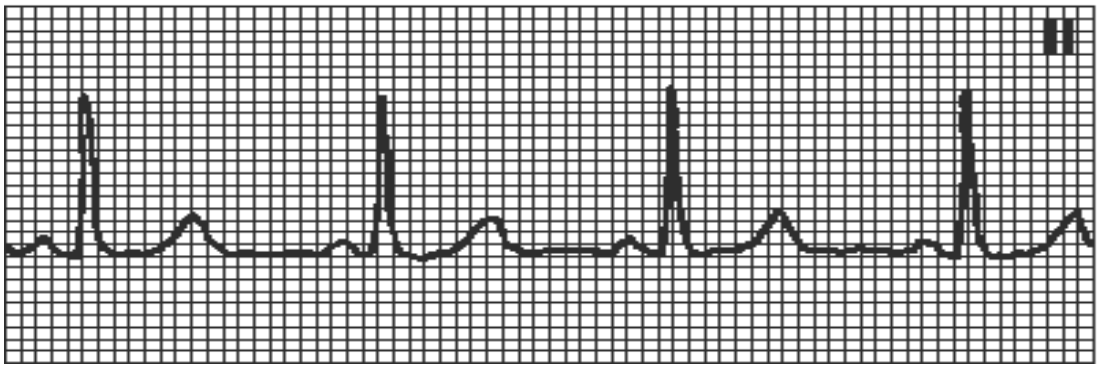


Fig 11-21. Normal sinus rhythm:
HR=60-80 in min, P (+) I, II, aVF, (-) aVR, PQ ≥0,12s

Sinus tachycardia

Sinus tachycardia is an accelerated sinus rhythm with a heart rate more than 100 per minute (in adults).

ECG signs of sinus tachycardia (Fig. 11-22):

- acceleration of a heart rate from 90-100 up to 160-180 in one minute;
- P-wave of the normal form precedes complex QRS;
- regular accelerated rhythm (all intervals R-R are identical).

Physiological sinus tachycardia occurs in response to a physical activity, stress, caffeine (strong tea, coffee, energy drinks). In these cases, sinus tachycardia is temporary and, as a rule, without unpleasant feelings.

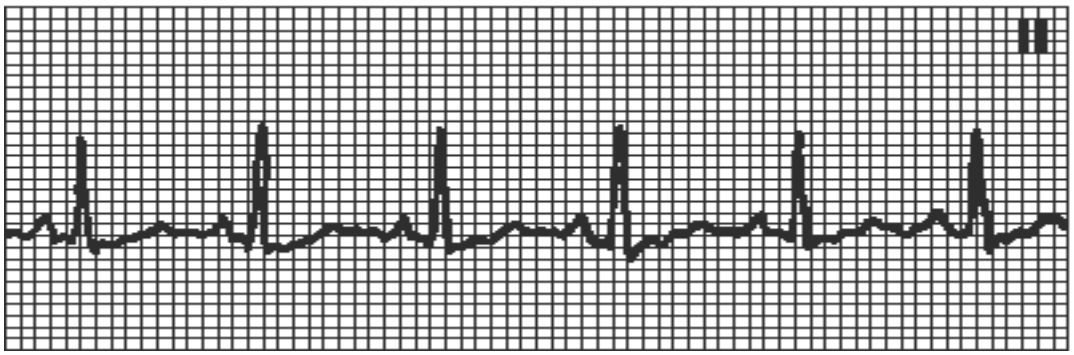


Fig. 11-22. Sinus tachycardia:
HR>90 in min, regular rhythm

Pathological sinus tachycardia remains at rest. Often, it is accompanied by unpleasant sensations of heart palpitations, a sense of lack of air. The causes of pathological sinus tachycardia may be hypoxia (hemorrhage, anemia), arterial hypotension, hypovolemia, fever (infection, inflammation), taking adrenergic drugs, thyrotoxicosis, circulatory and respiratory failure.

Sinus bradycardia

Sinus bradycardia is a slow sinus rhythm with a heart rate less than 60 per minute.

ECG signs of sinus bradycardia (Fig. 11-23)*:*

- decrease of a heart rate less than 60 in one minute;
- P-wave has a normal form;
- regular infrequent rhythm.

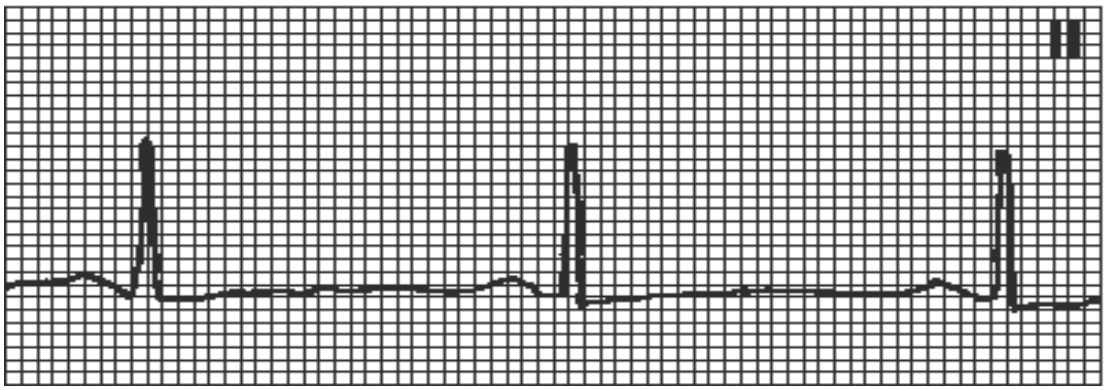


Fig. 11-23. Sinus bradycardia:
HR<60 in min, regular rhythm

Physiological sinus bradycardia is usually with a heart rate at least 45-50 per minute, and is not accompanied by unpleasant feelings. It can be observed at rest, especially in well-trained athletes and individual young healthy people.

Pathological sinus bradycardia is characterized by a significant reduction in pulse (less than 40 beats per minute). The patient may complain of weakness,

dizziness, cold sweat, fainting (syncope) due to hypoxia of brain (oxygen starvation), as there is no adequate blood supply. Causes for this condition are diverse: sclerotic changes in myocardium affecting sinus node; effects of cold; increased tonus of parasympathetic nervous system; increased intracranial pressure (brain edema, brain tumors, meningitis, cerebral hemorrhage); adverse effect of drugs (digitalis, beta-blockers, morphine); poisoning (lead, nicotine); hypothyroidism; starvation, typhoid fever, obstructive (mechanic) jaundice), etc.

Respiratory (sinus) arrhythmia

Respiratory (sinus) arrhythmia is manifested by change of R-R interval depending on the phase of respiration: interval R-R is shortened on inspiration and lengthened on expiration.

ECG signs of sinus (respiratory) arrhythmia:

- variability of P-P interval more than 0.12 s;
- a permanent shape of wave P;
- P always precedes complex QRS;
- the constant P-Q interval;
- "wavelike" change of R-R interval: in few cardiac cycles – gradually lengthening and then gradually shortening.
- disappearance of arrhythmia if ECG is registered with the patient's breathe delay.

Physiological respiratory (sinus) arrhythmia is a normal finding in young patients. It occurs due to a high activity of a parasympathetic part of an autonomic nervous system: at the moment of inspiration blood filling of atria increases, and ***Bainbridge reflex*** *(atrial reflex)* causes an increase in a heart rate; at the moment of expiration, the n. vagus is stimulated, and the heart rate decreases.

Respiratory arrhythmia is more pronounced against background of maximum n. vagus activity: for example, during night sleep, or after training in athletes. Often sinus respiratory arrhythmia is combined with bradycardia.

Pathological sinus arrhythmia. Appearance of sinus arrhythmia in an elderly patient most often indicates a pathological process, for example, the *the sick sinus (node) syndrome* (due to ischemia, myocarditis, cardiosclerosis) or effect of some medications (cholinomimetics). In these cases, change of R-R interval may not be depending on the phases of respiration.

11.10. Ectopic arrhythmias

Ectopic (heterotopic) arrhythmias occur when the increased activity of ectopic (located outside the sinus node) centers of excitation, which cause heart contractions before the sinus node impulse. Ectopic arrhythmias include extrasystole, paroxysmal tachycardia, atrial flutter and fibrillation, and ventricular flutter and fibrillation.

11.10.1. Extrasystolic arrhythmia (Ectopic beats, Extrasystoles)

Extrasystoles are premature cardiac beats resulting from an abnormal electrical focus or re-entry mechanism in the atria, AV (atrioventricular) junction and ventricles.

ECG analysis of extrasystoles includes assessment:

1. Registration of the ***extraordinary complex QRST***
2. Distance from the beginning of the previous atrioventricular complex P-QRST to beginning of extrasystole is measured, that is designated as the ***chaining interval*** of extrasystole.
3. ***Compensatory pause*** is the distance from extrasystole to the beginning of the following atrioventricular complex. ***Complete compensatory pause*** is noted equals to 2 normal R-R intervals. ***Incomplete compensatory pause –*** the sum of pre-extrasystolic and post-extrasystolic intervals is lesser than 2 normal R-R intervals.
4. Alternation of extrasystoles with normal cardiac complexes**.** ***Allorhythmia*** is a regular alternation of extrasystoles with normal cardiac complexes is often noted. It can be in form of ***bigeminy***, when extrasystole occurs after every normal heartbeat by an impulse from the sinus node, ***trigeminy*** – after two normal heart contraction, ***quadrigeminy*** - after three.
5. Quantity of extrasystole complexes at the ECG film. There *are* ***rare extrasystoles (<30 for hour) and frequent extrasystoles (>30 for hour)***. There are ***single and paired extrasystoles*** (three or more consecutive extrasystoles should be), ***monotopic*** (originating from the one source) and ***polytopic extrasystoles*** (originating from multiple ectopic foci) (Fig. 11-24, 11-25, 11-26). Polytopic extrasystoles have different configuration on the ECG.
6. Shape of extraordinary complex PQRST determines localization of extrasystole - atria, AV (atrioventricular) junction, and ventricles.

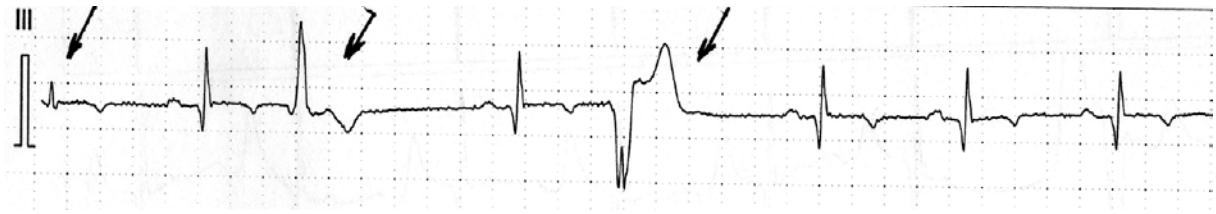


Fig. 11-24. Polytopic ventricular extrasystoles

Classification of extrasystolic arrhythmia

1. According to the origin: ***functional and organic extrasystoles***.
2. According to the site of origin: ***atrial and atrioventricular (nodal) extrasystoles (common name - supraventricular extrasystole); ventricular (left- and right-ventricular) extrasystoles*.**
3. According to the quantity of ectopic beats:

***- single, paired, frequent extrasystoles*;**

- allorhythmia - alternation of extrasystoles with sinus beats - bigeminy (1:2), trigeminy (1:3), quadrigeminy (1:4).

4. According to the quantity of ectopic foci:

- monotopic and polytopic (polymorphic) extrasystoles.

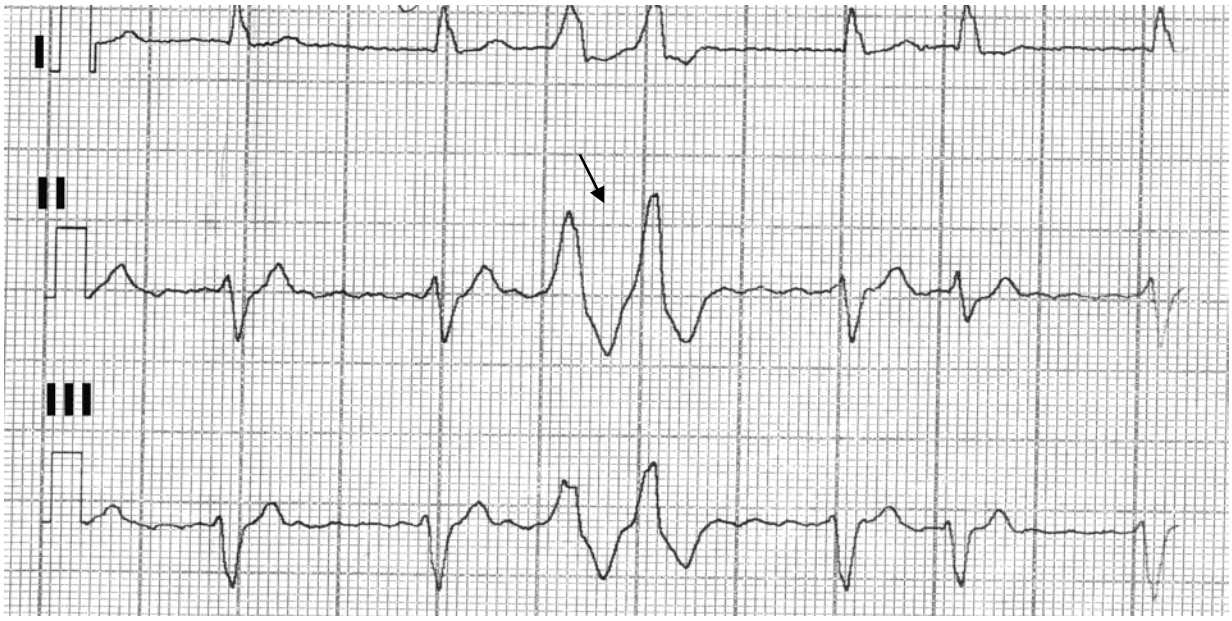


Fig. 11-25. Paired ventricular extrasystoles

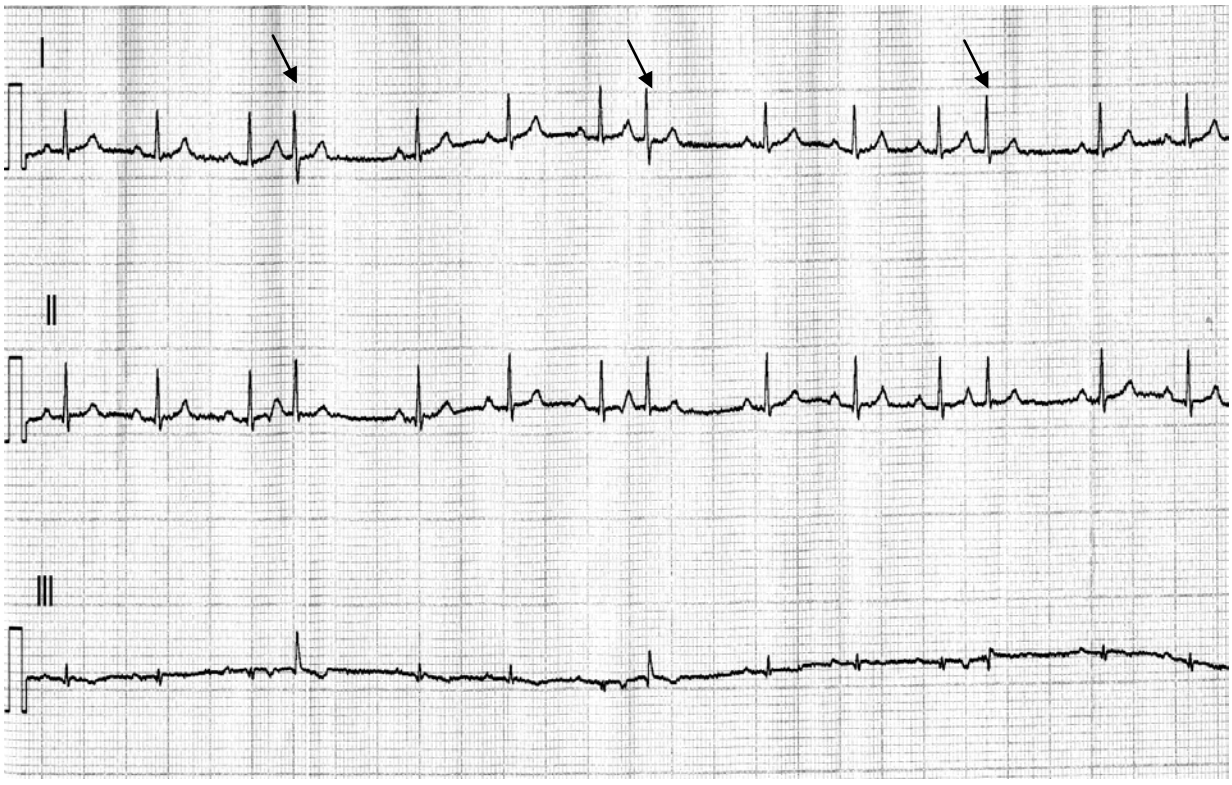


Fig. 11-26. Supraventricular extrasystoles – allorhythmia (quadrigeminy)

Functional extrasystoles are associated with various autonomic nervous system dysfunctions, emotional stress, smoking, abuse of strong tea, coffee, alcohol.

Organic extrasystoles indicate deep changes in myocardium in the form of foci of ischemia, dystrophy, necrosis or cardiosclerosis, contributing to formation of electrical inhomogeneity of myocardium. Most often extrasystoles are observed in acute myocardial infarction, coronary heart disease, arterial hypertension, rheumatic heart disease, myocarditis, chronic heart failure.

Atrial extrasystole

ECG signs of atrial extrasystole are: (Fig. 11-27)*:*

1. Registration of extraordinary complex P-QRST (can be determined by the appearance of the chaining interval);
2. P wave of normal shape;
3. Unaltered ventricular QRS complex of atrial extrasystole;
4. Incomplete compensatory pause after the extrasystole.

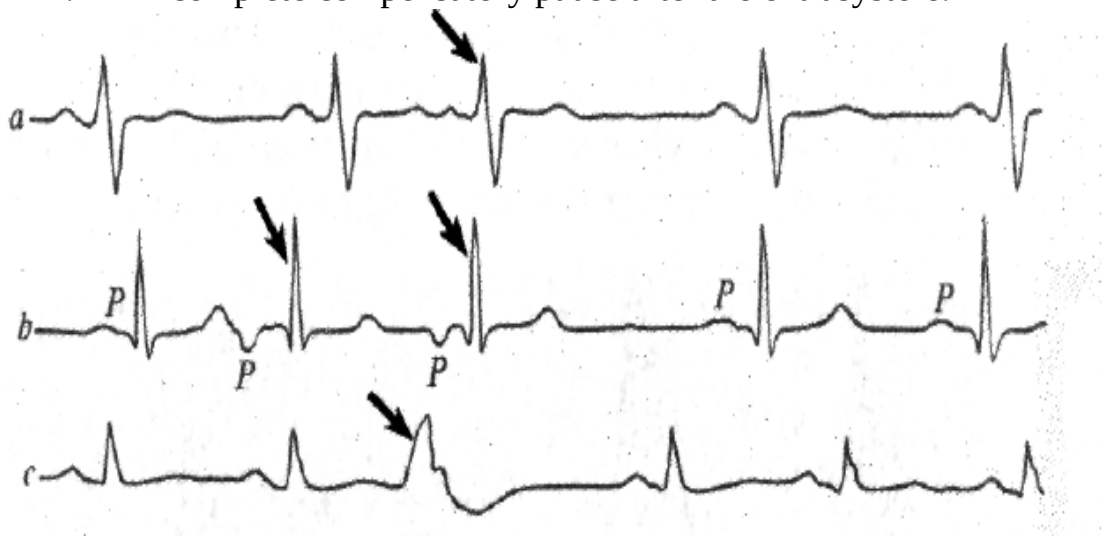


Fig. 11-27. Extrasystolic arrhythmia according to the site of the origin:
a – atrial extrasystole, b - AV-junction (atrioventricular) extrasystole, c – ventricular extrasystole

Atrioventricular (nodal) extrasystole (premature atrioventricular junctional complex)

ECG signs of atrioventricular extrasystole (Fig. 11-27 – 11-28)*:*

1. Registration of extraordinary unchanged ventricular complex QRST (can be determined by chaining interval);
2. Incomplete compensatory pause;
3. Absence of the P wave (merge P wave and QRS complex) or negative P wave after QRS complex of extrasystole; negative *P* wave - because of the retrograde atrial excitation or absence of *P* wave may be;
4. Shape of QRS is normal or slightly deformed.

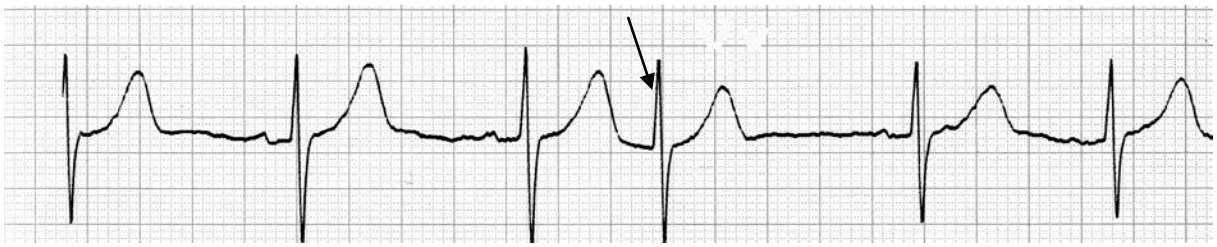


Fig. 11-28. Atrioventricular extrasystole.

Supraventricular extrasystoles (common name of atrial and atrioventricular extrasystoles) are premature electrical complexes on ECG, generated above the level of the ventricle. ***Supraventricular extrasystoles are characterized by normal or slightly deformed shape and normal length of QRS complexes.***

Ventricular extrasystole (ventricular ectopic beat) (Fig. 11-25, 11-27, 11-29):

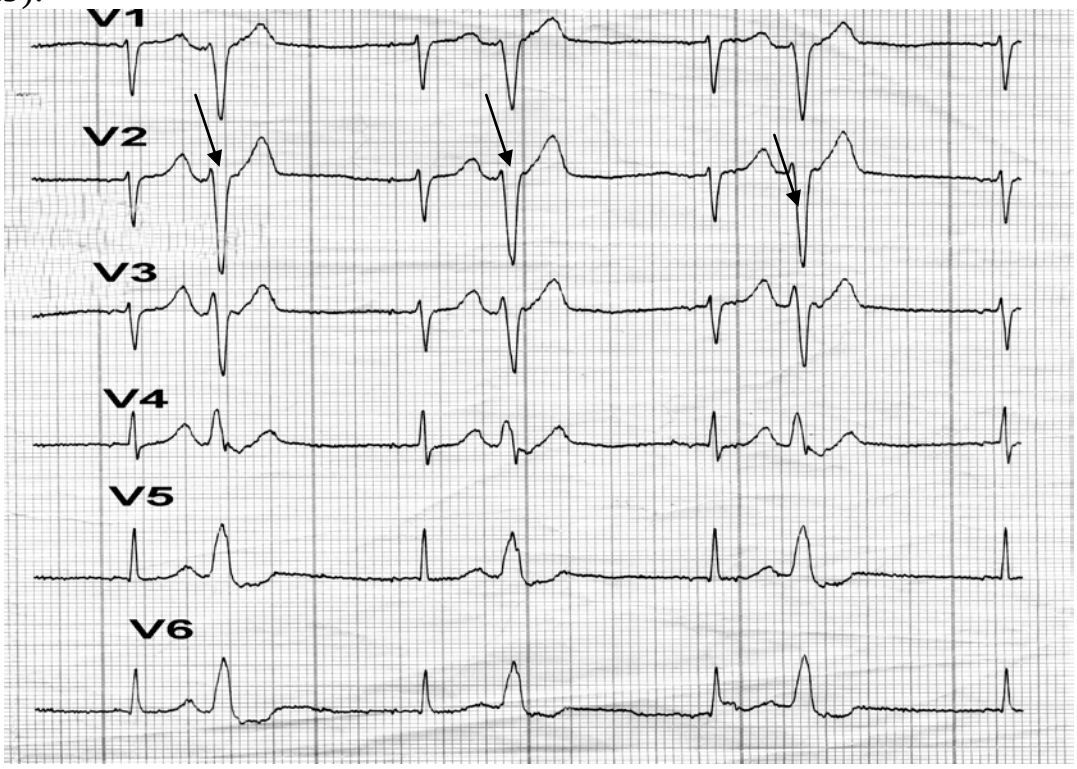


Fig. 11-29. Ventricular extrasystoles - allorhythmia (*bigeminy*)

ECG signs of ventricular extrasystole:

1. Registration of an extraordinary ventricular complex QRST, with its significant deformation and widening;

2. Absence of the P wave before the QRS complex of extrasystole;

3. Discordant displacement of the ST segment and the T wave in relation to the main wave of the QRS complex, which acquires an asymmetric two-phase shape;

4. Presence of a complete compensatory pause.

11.10.2. Paroxysmal tachycardia (PT)

Paroxysmal tachycardia is a sudden beginning, and also abruptly ending, attack of a sharp increase in the regular heart rate with a frequency of 140 to 250 in one minute, due to an abnormal ectopic impulses or re-entry mechanism in the atria, AV node and ventricles.

Classification of PT according to the site of the origin:

- atrial, atrioventricular (nodal) (common name of supraventricular) PT;
- ventricular PT.

ECG characteristics of supraventricular PT (Fig. 11-30):

1. Regular heart rate 140-250 / minute ;
2. The QRS shape is not changed as a rule (or slightly deformed);
3. The P wave – disfigured (or biphase, negative) prior to QRS in atrial PT or follows QRS in AV nodal PT, or is not differed.

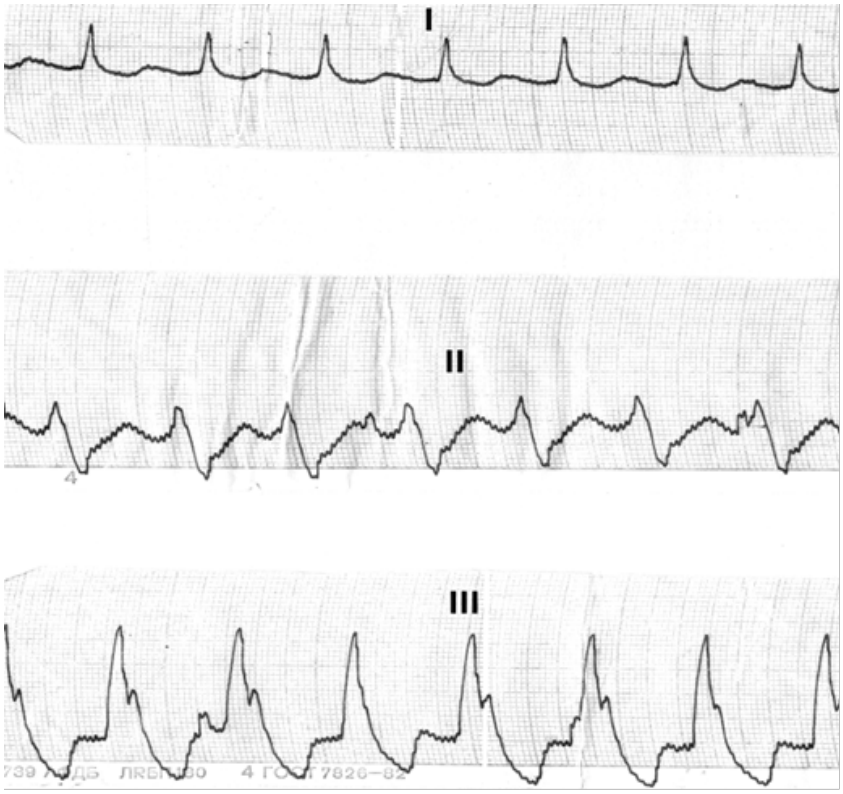


Fig. 12-30. Supraventricular paroxysmal tachycardia:
HR equals 150 per minute, QRS shape is not changed or slightly deformed

ECG signs of atrial paroxysmal tachycardia:

1. Regular heart rate 140-250 / minute;
2. Presence changed (deformed, negative, biphasic) P wave before each ventricular complex QRS;
3. Unchanged ventricular complexes QRS.

ECG signs of paroxysmal tachycardia from the atrioventricular node:

1. Registration of the heart rate 140-250 per minute, while maintaining a regular rhythm;

2. The absence of the wave P (merge P and QRS) or check the negative teeth after R of the QRS complexes;

3. Unchanged ventricular complexes QRS.

ECG signs of ventricular paroxysmal tachycardia (Fig. 11-31):

1. Regular heart rate 140-250 per minute;

2. Deformation and widening ventricular complex QRS more than 0.12 seconds;

3. Dissociation of the frequent ventricles rhythm (140-250 per minute) and the normal rhythm of the atria (P wave – from 60 to 90 per minute);

4. P waves do not correspond to ventricular complexes QRS (atrioventricular dissociation) or are not differed on ECG/

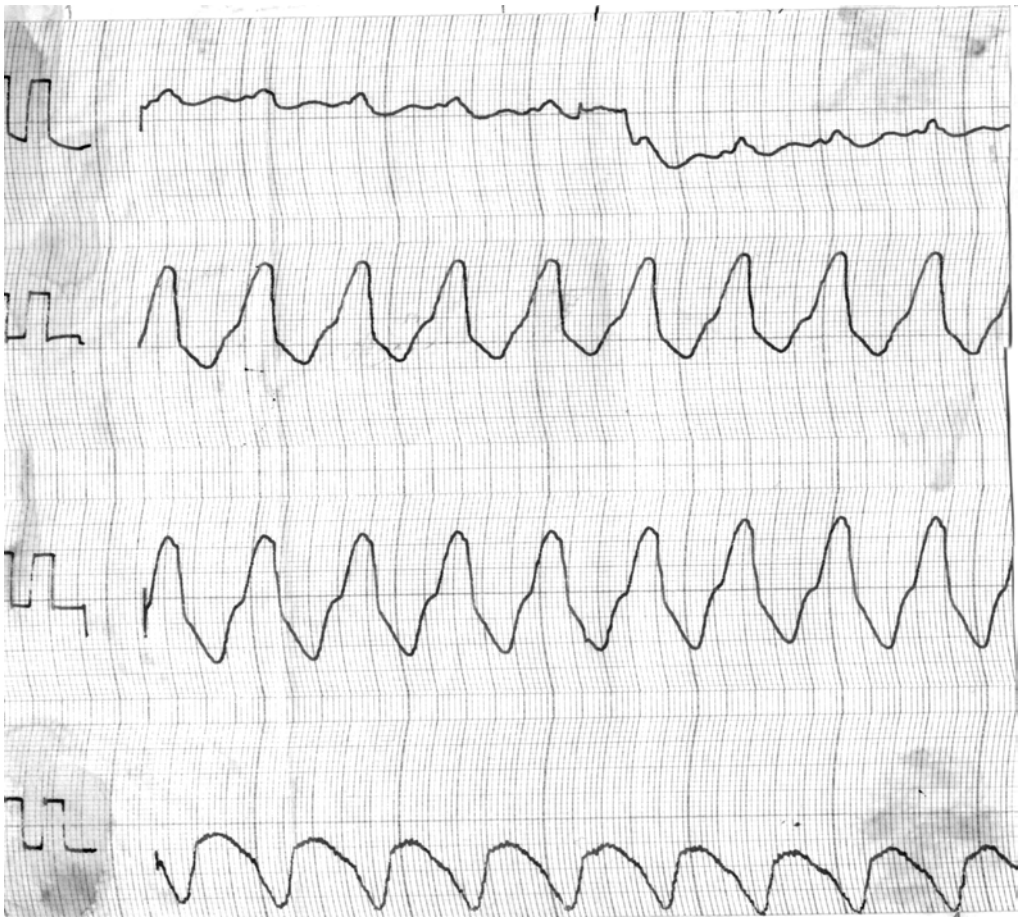


Fig. 11-31. Ventricular paroxysmal tachycardia:
HR equals 180 per minute, the QRS shape is deformed and broadened

Ventricular PT and early ventricular extrasystoles (type "R on T") and may be as predictor of ventricular flutter, ventricular fibrillation and cardiac arrest.

11.10.3. Ventricular flutter and fibrillation

Ventricular flutter usually appears as sinusoidal waves of a similar shape and amplitude with a rate between 150 and 300 per minute (Fig. 11-32).

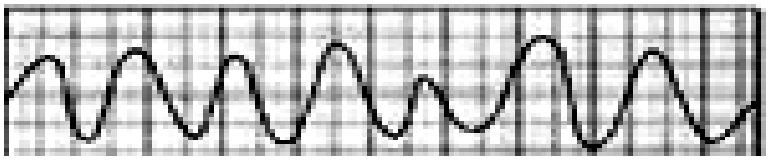


Fig. 11-32. Ventricular flutter: sinusoidal waves of similar shape and amplitude with rate 280 per minute

Ventricular fibrillation *is* a rapid irregular ventricular rhythm due to multiple irregular re-entrant waves of a variable shape and amplitude with a rate between 200 and 500 per minute associated with essentially zero cardiac output. It is a variant of cardiac arrest (Fig. 11-33).

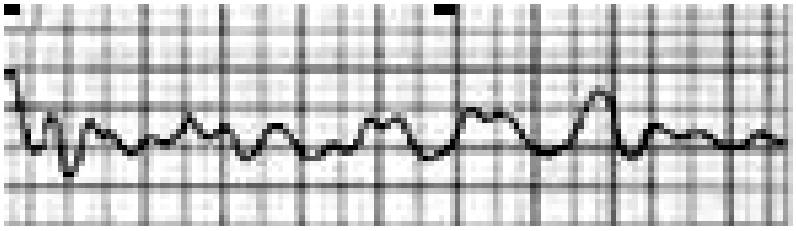


Fig. 11-33. Ventricular fibrillation: multiple irregular waves of variable shape and amplitude with rate nearly 300 per minute.

ECG signs of ventricular flutter and fibrillation (see Fig.11-32; 11-33):

1. Absence of normal ventricular QRST complexes.
2. In ventricles flutter, there are frequent (200-300 per minute) waves of the same shape and amplitude resembling a sinusoidal curve.
3. In ventricular fibrillation, there are frequent (300-500 per minute) irregular waves, different in shape and amplitude.

Ventricular flutter is observed in the preagonal and agonal states. Ventricular flutter often turns into a ventricular fibrillation. In the ventricular fibrillation, the patient is in a state of clinical death, and if no help is provided or it is not effective, biological death occurs.

11.10.4. Atrial fibrillation and atrial flutter

Atrial fibrillation and flatter *are associated with various cardiovascular diseases that contribute to the development and maintenance of arrhythmia*: arterial hypertension, ischemic heart disease, acquired heart valve diseases (most often mitral valve), congenital heart diseases (atrial septal defect, and others), dilatation cardiomyopathy, inflammation (pericarditis, myocarditis). Often, atrial

fibrillation can be detected when determining irregular heartbeats and pulse, and deficiency of pulse (pulse beats are fewer than heartbeats).

Atrial fibrillation

Atrial fibrillation (AF) is a rapid irregular atrial rhythm due to the multiple reentrant wavelets (Fig. 11-34).

ECG characteristics of atrial fibrillation:

1. P wave disappears in all ECG leads,
2. multiple small irregular f waves,
3. QRS ventricular complexes follow irregular, their shape is not changed.

4. When small-wave atrial fibrillation, "f" waves are visually absent, other characteristics of the atrial fibrillation are the same.

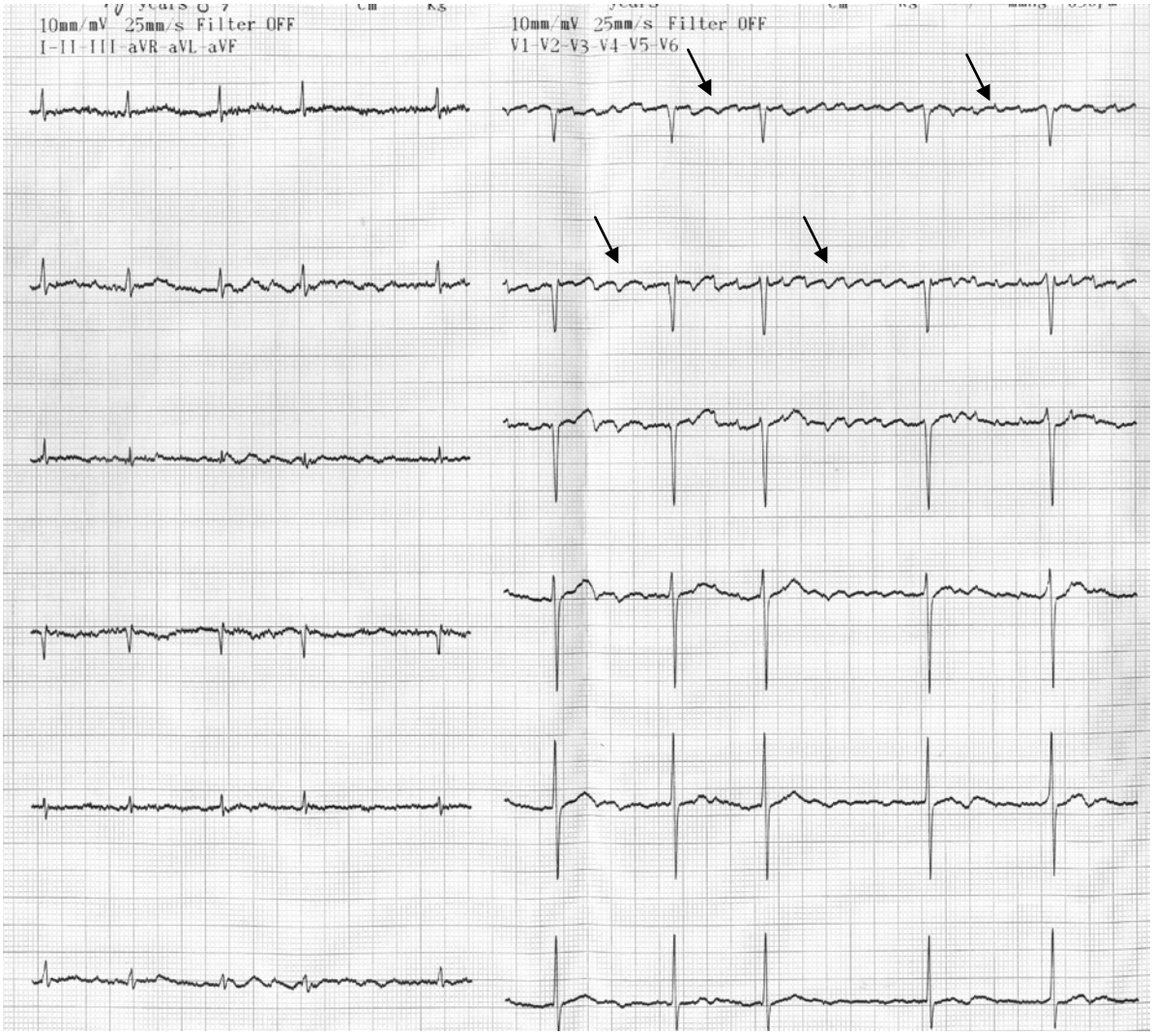


Fig. 11-34. Atrial fibrillation:

(1) *P* wave disappears in all ECG leads; (2) multiple small irregular f waves (see arrow); (3) QRS ventricular complexes follow are irregular, their shape does not change.

Classification of atrial fibrillation

1. Depending on the rate of the ventricular contractions three forms of the atrial fibrillation are distinguished*:*

- ***normosystolic form*** *– 60-100 per min;*
- ***tachysystolic form*** *–>100 per min;*
- ***bradysystolic form*** *– <60 per min.*

2. Depending on the course of atrial fibrillation:

- ***Paroxysmal AF*** – episodes of AF that terminate spontaneously or with intervention within 7 days; may recur with variable frequency 7 days*;*
- ***Persistent AF*** – episodes of continuous AF that last more than 7 days and do not self-terminate;
- ***Long-standing persistent AF*** – episodes of continuous AF that last more than 12 months;
- ***Permanent AF*** *(I48.2)* - with a prolonged course of AF, and the decision of the doctor and patient do not restore the sinus rhythm.

3. Depending on the amplitude of "f" waves:

- ***large-wave atrial fibrillation***
- ***small-wave atrial fibrillation****.*

Atrial flutter

Atrial flutter is a rapid regular atrial rhythm due to a constant well-defined macro-reentrant circuit in the right atrium (Fig. 11-35).

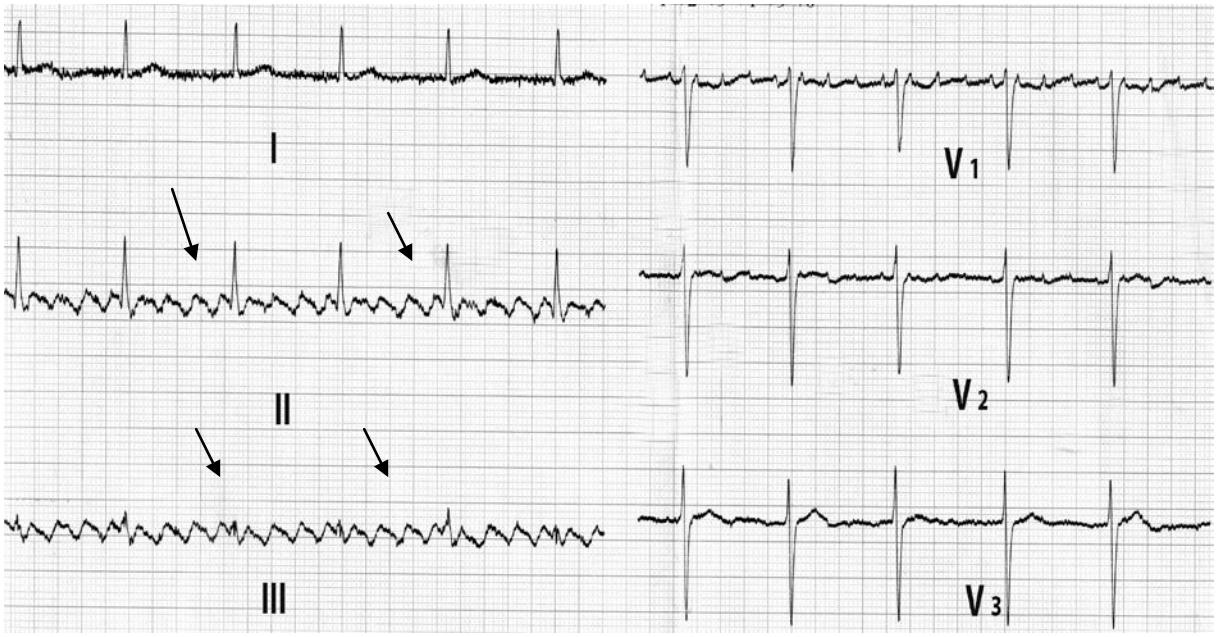


Fig 11-35. Atrial flutter (regular variant 3:1):
regular saw-tooth shape F waves (see arrow)

ECG signs of atrial flutter:

1. No P wave in all ECG leads (rhythm is not sinus);

2. Frequent (200-350 per minute), regular, sawtooth - shaped "F" waves (more often in II, III, aVF, V1, V2 leads);

3. Unchanged ventricular QRS complexes, each is preceded by a constant number of the atrial "F" waves (2:1, 3:1);

4. QRS complexes follow at regular intervals - regular variant, irregular atrial flutter occurs more rarely.

The *irregular variant of the atrial flutter* may be a little different from large-wave atrial fibrillation.

11.11. Conduction disorders (heart blocks)

Heart blocks are a delayed conduction or a complete absence of the conduction in some department of the cardiac conduction system.

Depending on the level of the block, the following ***main forms of the heart block*** are distinguished:

- ***sinoatrial block*** – impairment of the conduction between the sinus node and atria;
- ***intraatrial block*** – impairment of the conduction through the atrial myocardium;
- ***atrioventricular block*** – impairment of the conduction between the atria to the ventricles;
- ***intraventricular block (His bundle branch block)*** – impairment of the conduction through the His bundle and its branches.

11.11.1. Sinoatrial block

***ECG signs of sinoatrial block*:**

Sinoatrial block is a violation of an impulse conduction from the sinus node to the atria and ventricles with such signs as (Fig. 11-36):

1. Periodic loss of the cardiac complexes P-QRST recorded on ECG;
2. the R-R interval between registered ventricular complexes increases by 2 times.

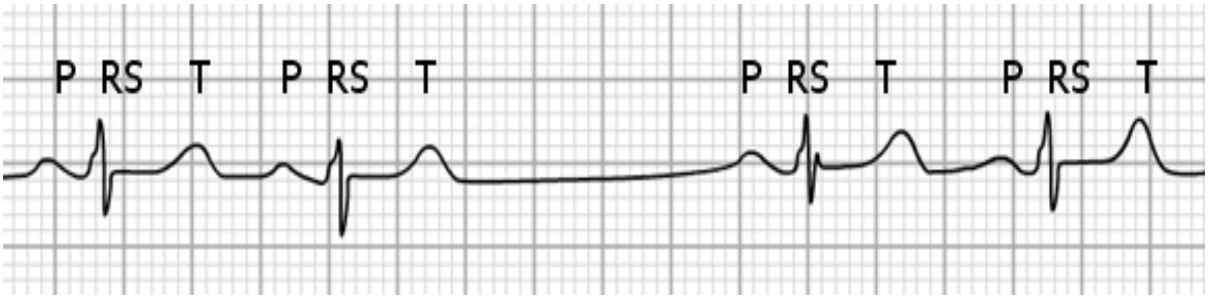


Fig. 11-36. Sinoatrial block:
periodic missing of the heart complex (PQRST)

11.11.2. Intraatrial block

Intraatrial block is a violation the of impulse conduction through the atria.

ECG signs of intra-atrial block (Fig. 11-37):

1. P waves are broadened ≥0.11 s and splitted;
2. biphase wave P_{V1}.

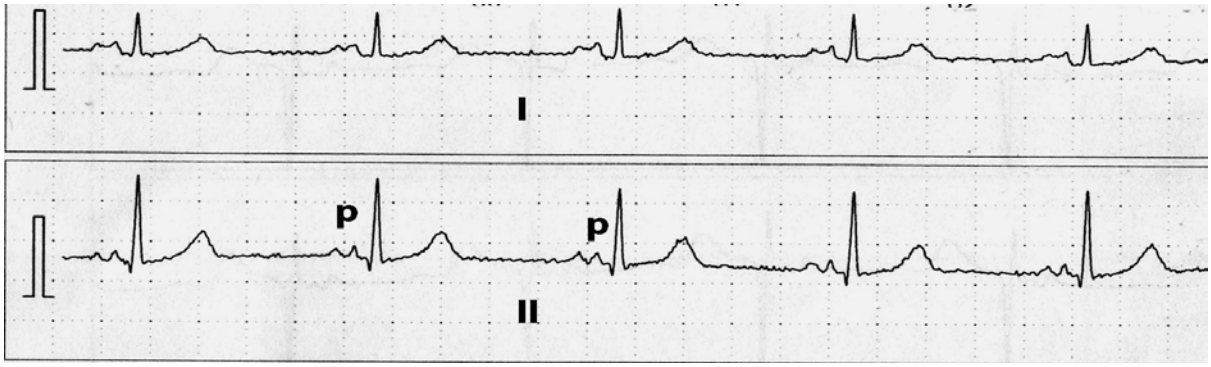


Fig. 11-37. Intra-atrial block: P waves are broadened ≥0.11 s and splitted

11.11.3. Atrioventricular block

Atrioventricular (AV) block is a violation of the impulse conduction from the atria to the ventricles, while there are 3 degrees of this block.

I degree of AV block

I degree of AV block can be revealed only electrocardiographically (Fig. 11-38). ***ECG sign of atrioventricular block of I degree*** is a permanent elongation of PQ intervals over 0.21 sec. with normal regular QRS complex.

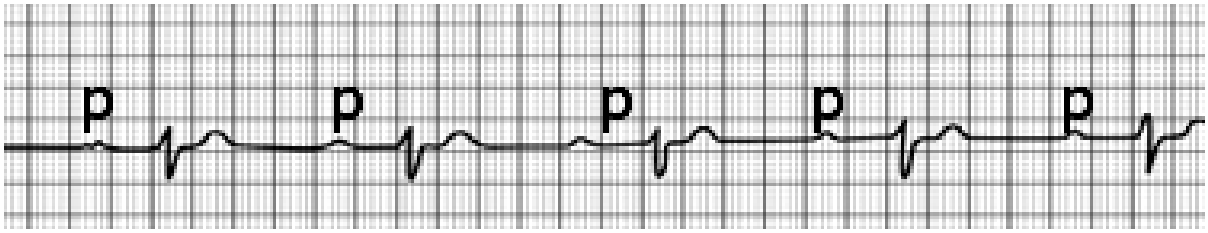


Fig. 11-38. I degree of atrioventricular block:
(1) increased *P-Q* interval > 0.21 s without missing QRS;
(2) regular heart rhythm

II degree of AV block

Atrioventricular block of II degree is a slowing the impulse conduction through atrioventricular node until its complete delay that results in periodical missing ventricular complex QRS on ECG, ventricle contractions and missing

pulse beats (which correspond to the Samoilov-Wenckebach period) in clinical picture. There are 3 types of atrioventricular block of II degree.

ECG signs of II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type) (Fig. 11-39):

1. gradual elongation PQ (Samoilov-Wenckebach periods);
2. periodically missing ventricular contractions.

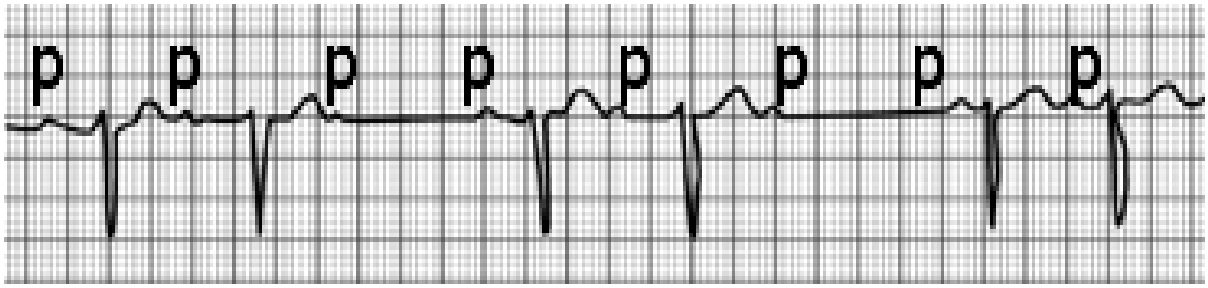


Fig. 11-39. II degree of atrioventricular block with Samoilov-Wenckebach periods (Mobitz-1 type.

ECG signs of II degree of AV block (Mobitz-2 type) (Fig. 11-40):

1. periodic missing QRS without gradual elongation PQ (2:1, 3:1);
2. PQ can be normal or a little bit prolonged.

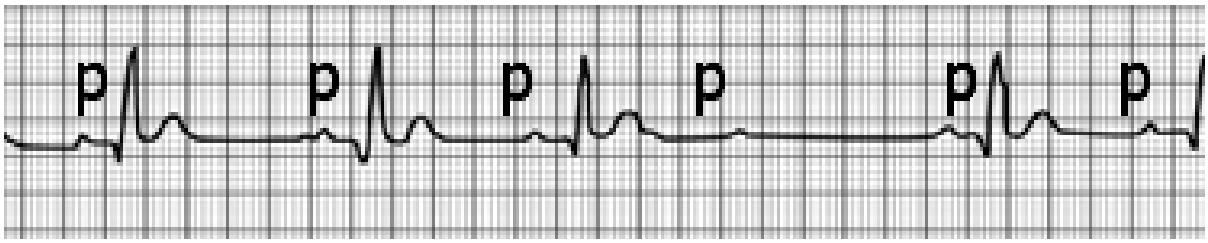


Fig. 11-40. II degree of atrioventricular block (Mobitz-2 type).

ECG signs of severe II degree atrioventricular block (Mobitz-3 type) is constantly increased PQ interval with a regular loss of every second or third ventricular complex QRS.

III degree of AV block (complete heart block)

Atrioventricular block of III degree is a complete delay impulse through atrioventricular node, there is no conduction between atriums and ventricles.

During the occurrence of the complete atrioventricular block and the associated significant reduction of the cardiac activity (a heart rate is less than 45-50 per minute) and brain blood supply, there is a different duration (from a few seconds to several minutes) loss of consciousness in some patients, accompanied by epileptoid convulsions (***syndrome of Morgagni–Adams–Stokes***).

ECG characteristics of atrioventricular block III degree (Fig. 11-41) :

1. P waves with a frequency of 60-90 per minute regardless of complexes QRS registration (can be overlap with QRS complex and T wave and after them;
2. ventricular complexes QRS are recorded with a frequency of less than 50-52 per minute;
3. intervals P-P and R-R are more often permanent, but R-R is significantly more than P-P.

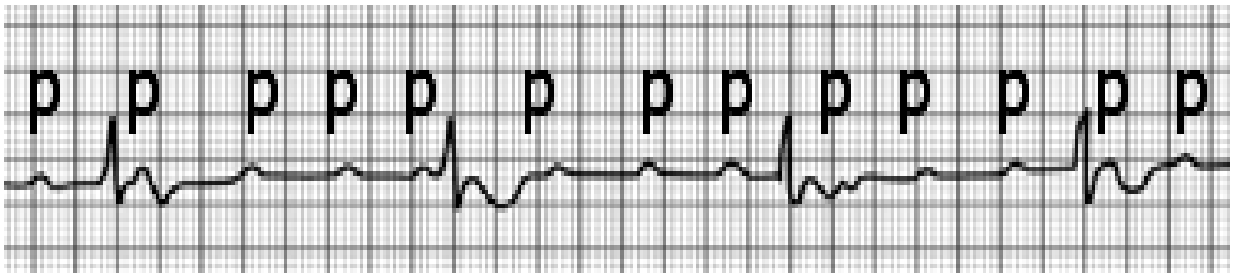


Fig. 11-41. III degree of atrioventricular block (complete heart block)

11.11.4. His bundle branch (intraventricular) blocks

Intraventricular block (His bundle branch block) is characterized by an impaired impulse conduction along the right and/or the left branches of His bundle or Purkinje fibers. This leads to an increase in the time of the excitation of the corresponding ventricle and an increase in the amplitude and duration of the QRS complex.

ECG signs of His bundle branch blocks:

- *QRS* complexes are markedly altered and widened ≥ 0.12-0.18 s and resemble complexes in the ventricular extrasystole.

ECG signs of the left His bundle branch block (Fig. 11-42):

1. wide and deformed QRS has the form of qR in I, II, V_{5-6}; rS in III, aVF, V_{1-2};
2. disconcordance of ST, T and the main wave of QRS (wide, deep S in III, aVF, V_{1-2}; depression ST and negative T in V_{5-6});
3. negative ST and T in V1-2;
4. levogram.

ECG signs of the right His bundle branch block is characterized (Fig. 11-43)*:*

1. wide QRS in III, V_{1-2} has the form of rsR, rSR, RsR´(similar to “M”) (the shape of the ventricular complexes resembles that of left-ventricular extrasystoles);
2. wide S in I, aVL,V_{5-6};
3. depression ST and negative T in V_{1-2};
4. dextrogram.

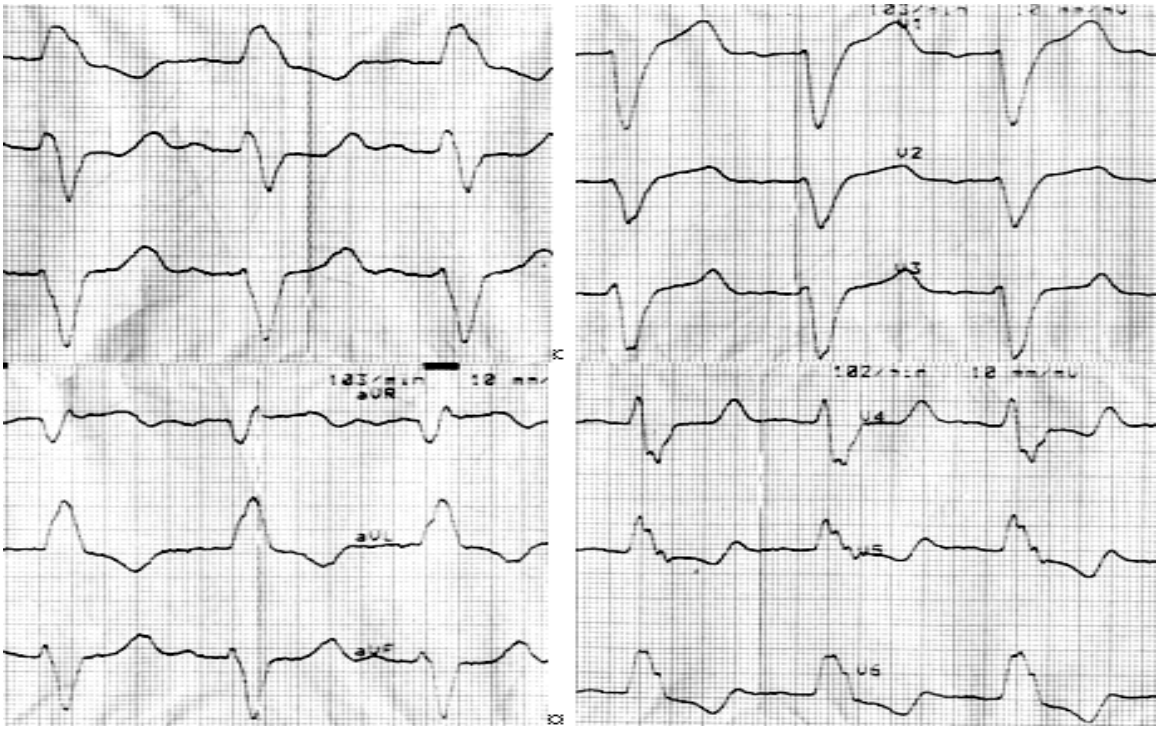


Fig. 11-42. Complete left His bundle branch block

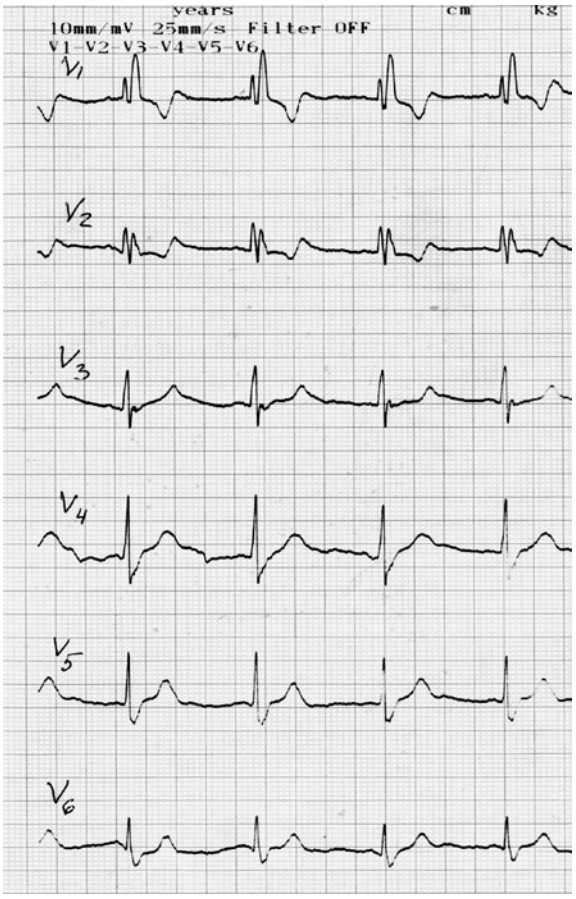


Fig. 11-43 Complete right His bundle branch block

11.12. The key points on the theme “Electrocardiography”

Standard ECG of 12 leads is the basic important method for diagnosis a large number of cardiac pathologies (Table 11-5), including heart arrhythmia and conduction disorders, atrial and ventricle hypertrophy, myocardial infarction, electrolyte disorders, etc.

Table 11-5. Interpretation of ECG changes

Component of ECG	Changes	Causes
P wave	abnormal	atrium hypertrophy, intra-atrial block, atrial extrasystole
	absence	atrial fibrillation and flutter, sinoatrial block
P-P interval	varies	sinus arrhythmia
P-R interval	elongation	I degree atrioventricular block
	varies	II degree atrioventricular block (Mobitz-1 type)
QRS complex	widening	His bundle branch block, ventricular extrasystole and paroxysmal tachycardia
Q wave	abnormal	myocardial infarction
R wave	high	ventricle hypertrophy
ST segment	depression	myocardial ischemia, His bundle branch block, ventricle hypertrophy
	elevation	myocardial infarction and ischemia, pericarditis, His bundle branch block
T wave	abnormal	myocardial ischemia, electrolyte disorders (hypo- and hyperkalemia)

11.13. Assessment tests on the theme “Electrocardiography”

1. Position of standard leads of ECG:

1. I – Right arm - left arm;
2. II – Right arm - left foot;
3. III – Left foot - left arm;
4. IV – Right foot - right arm;
5. I– Left foot - right arm.

2. Position of augmented leads of ECG:

1. aVL - left arm;
2. aVR - right arm;

3. aVF - left foot;
4. aVF - right foot;
5. aVS - xiphoid process.

3. Position of chest leads of ECG:
1. V1 – right sternal edge at the 4-th intercostal space;
2. V2 – left sternal edge at the 4-th intercostal space;
3. V3 – between V2 and V4;
4. V4 – left midclavicular line – the 5-th interspace;
5. V5 – apex beat.

4. Generation of ECG waves and intervals:
1. P – depolarization of atriums;
2. Q - repolarization of atriums;
3. QRS - depolarization of ventricles;
4. ST - repolarization of ventricles.

5. Standard size of 1 mv:
1. 1 mm;
2. 5 mm;
3. 10 mm;
4. 15 mm.

6. Standard speed of tape:
1. 5 mm/sec;
2. 10 mm/sec;
3. 25 mm/sec;
4. 50 mm/sec.

7. Sinus rhythm signs:
1. P-wave precedes, follows and superimposes complexes QRS;
2. P-wave positive in II standard lead;
3. P-wave corresponds (previous) to complexes QRS;
4. P-wave absents.

8. Analysis of myocardial conduction depending duration of:
1. P wave – intraatrial conduction;
2. PQ interval - intraventricular conduction;
3. QRS complex - intraventricular conduction;
4. T–P interval – conduction in atrioventricular node.

9. N**ormal electrical axis of the heart is characterized by:**
1. RI> RII> RIII;

2. RII> RI> RIII;
3. RIII> RII> RI;
4. V2>V1>V3.

10. Horizontal electrical axis (levogram) of the heart is characterized by:
1. RII> RI> RIII;
2. RI> RII> RIII;
3. RIII> RII> RI;
4. V1>V2>V3.

11. Vertical electrical axis (dextrogram) of the heart is characterized by:
1. RII> RI> RIII;
2. RI> RII> RIII;
3. RIII> RII> RI;
4. V3>V2>V1.

12. **ECG- signs of right atrium hypertrophy are:**
1. P > 2.5 mm in II, III, V1-V2;
2. two-humped P in I, II, V5-V6;
3. RIII> RII> RI;
4. deep S wave in leads I, αVL, V5-V6.

13. **ECG- signs of left atrium hypertrophy are:**
1. P > 2.5 mm in II, III,V1-V2;
2. two-humped P in I, II, V5- V6;
3. biphasic or negative P in V1;
4. RI> RII> RIII.

14. **ECG- signs of myocardial ischemia are:**
1. high and wide T wave;
2. negative symmetrical T wave;
3. two-phase (+ - or - +) T wave;
4. asymmetrical negative T wave;
5. pathological Q-wave.

15. ECG- signs of myocardial ischemic damage:
1. pathological Q-wave;
2. displacement of RS-T segment above or below the isoline;
3. ST depression;
4. ST elevations;
5. symmetrical negative T wave.

16. For ECG recorded in acute myocardial infarction is typically:
1. elevation of S-T interval is more than 1 mm upward from isoelectric line;
2. acuminate, symmetric and negative T-wave;
3. S-T interval is on isoelectric line;
4. increased depth of Q-wave;
5. T-wave is not changed.

17. ECG- signs in subacute stage of myocardial infarction:
1. decreased depth of Q-wave;
2. pathological Q-wave persists;
3. ST-segment returns to the isoelectric line;
4. negative symmetrical wave T.

18. ECG- signs in cicatrization stage myocardial infarction:
1. pathological wave Q persists;
2. ST segment on the isoelectric line;
3. wave T becomes slightly negative or positive smoothed;
4. decreased depth of Q-wave.

19. Pathological changes of ventricular complex (Q-wave) in myocardial infarction of anterior wall of the left ventricle in:
1. V1-4 leads;
2. I, aVL leads;
3. V5-6 leads;
4. II, III, aVF leads;
5. III, aVR leads.

20. Pathological changes of ventricular complex (Q-wave) in myocardial infarction of lateral wall of the left ventricle in:
1. V5-6 leads;
2. I, aVL leads;
3. V1-4 leads;
4. II, III, aVF leads;
5. III, aVR leads.

21. Pathological changes of ventricular complex (Q-wave) in myocardial infarction of posterior wall of the left ventricle:
1. I, aVL leads;
2. V1-2 leads;
3. V3-4, aVR leads;
4. V5-6 leads;
5. II, III, aVF leads.

22. The ischemic response during or after the exercise stress ECG testing is characterized by:
1. negative P wave;
2. pathological Q-wave;
3. flat or downward ST segment depression>1 mm lasting > 0.08 sec;
4. two-phase (+ - or - +) T wave;
5. asymmetrical negative T wave.

23. Causes of cardiac arrhythmias include:
1. affected automaticity of the sinus node;
2. foci of increased activity in the myocardium;
3. disorders of cardiac conduction system;
4. local conduction disorder (re-entry mechanism);
5. combined changes in automaticity, excitability, conduction.

24. Normal sinus rhythm characteristics:
1. HR (heart rate) equals 60-90 per minute;
2. regular heart rhythm;
3. difference between minimal and maximal R-R intervals is not more than 15%;
4. P wave - positive in I, II, aVL;
5. P wave precedes QRS.

25. ECG-signs of ventricular extrasystole are:
1. premature appearance of QRS-complex;
2. widening and deformation of QRS-complex;
3. absence of atrial P-wave;
4. direction of the main wave of QRS-complex is opposite to T-wave;
5. compensatory pause is not complete. Interval between pre-extrasystolic and post-extrasystolic R-waves is less two normal R-R-intervals.

26. ECG-signs of atrial extrasystole are:
1. premature appearance of cardiac complex;
2. P-wave is preserved, a little bit deformed;
3. shape of ventricular complex QRS is preserved;
4. compensatory pause is complete. Interval between pre-extrasystolic and post-extrasystolic R-waves is equal two normal R-R-intervals;
5. shape of ventricular complex QRS is deformed.

27. ECG-signs of atrial paroxysmal tachycardia are:
1. heart rate is more than 160 in one minute;
2. shape of ventricular complex QRS is preserved;
3. shape of ventricular complex QRS is changed;

4. distance between R-R is identical;
5. P-waves are placed before complex QRS.

Chapter 12. Laboratory-Instrumental Methods of Cardiovascular System Examination

Goals: to enable students to learn –

1) instrumental methods of examination (phonocardiogram, chest roentgenogram, echocardiogram, radionuclide and imaging techniques) in cardiovascular system diseases;
2) clinical and biochemical blood tests in cardiovascular system diseases (ischemic heart disease, rheumatic carditis, and circulatory failure).

12.1. Instrumental examination in the cardiovascular system diseases

12.1.1. Phonocardiography and computer-aided auscultation

Phonocardiography (PCG) is a diagnostic method that displays heart sounds as a graph. To register heart sounds, sensors equipped with microphones are attached to the patient's chest. Thanks to PCG it is possible to catch nuances in the change of the heart sounds and murmurs that are not auscultated by ear. PCG allows you to define III and IV sounds, which are indistinguishable to ear. ***PCG has diagnostic value especially for heart valves disease diagnosis*** (Fig. 12.1).

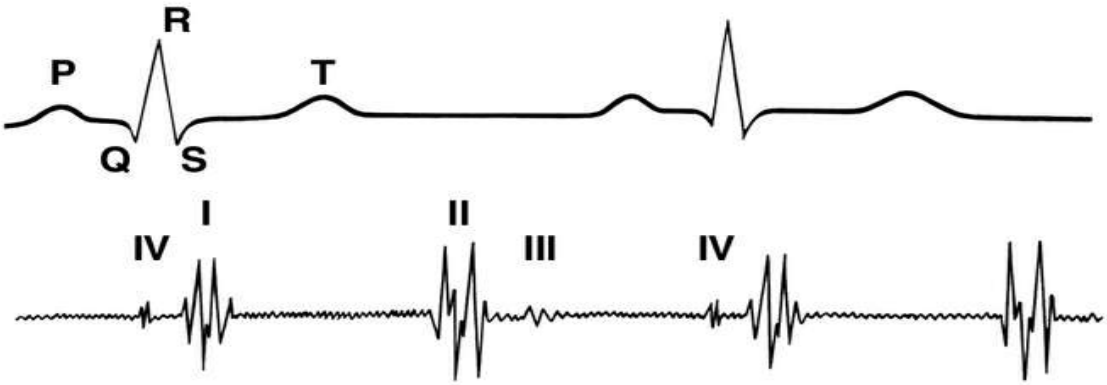
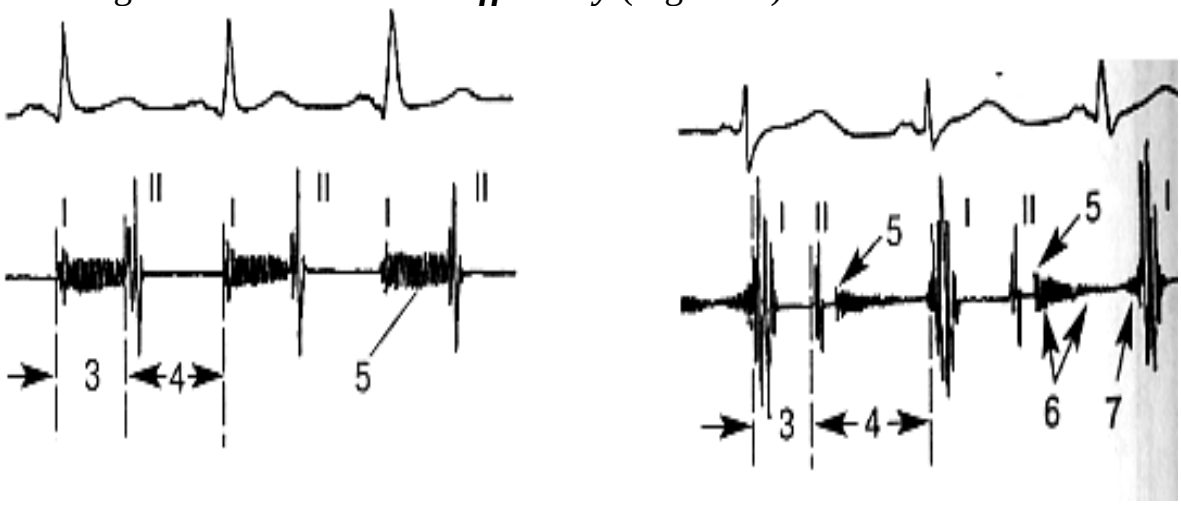


Fig. 12-1. Correlation between the phonocardiogram signal (PCG) and the electrocardiogram signal (ECG) in the norm: I, II, III, IV – heart sounds

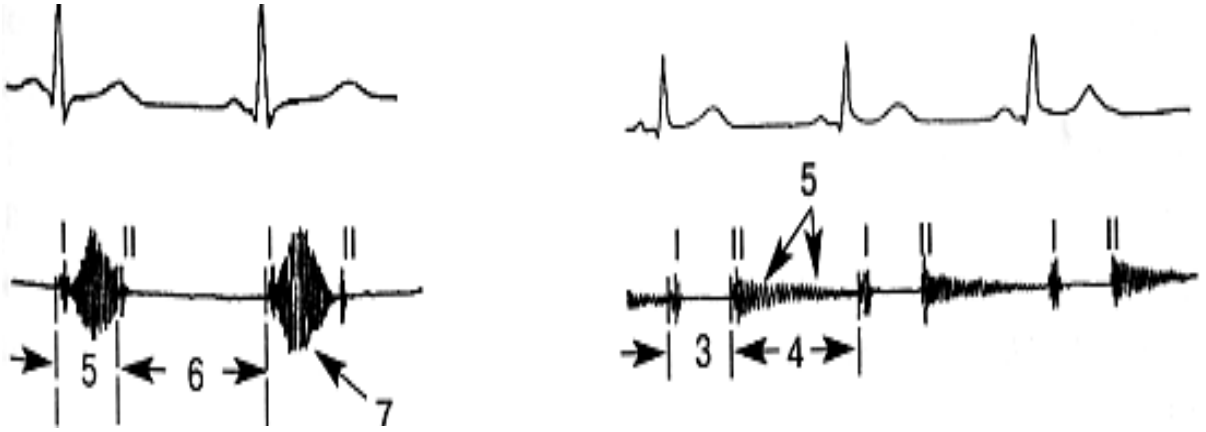
Phonocardiogram on the heart apex records a weakened I sound and pansystolic murmur ***in mitral insufficiency*** (Fig. 12-2). Phonocardiogram on the heart apex records I loud snapping sound, non-changed II sound, and III pathologic sound – “opening snap”, and protodiastolic and presystolic murmurs ***in mitral stenosis***.

Phonocardiogram on II-d interspace at the right edge of sternum demonstrates weakened I and II sounds, and “diamond shape” pansystolic murmur ***in aortal stenosis***. Phonocardiogram on II-d interspace at the right edge of sternum demonstrates weakened I and II heart sounds, and protodiastolic descending murmur ***in aortal insufficiency*** (Fig. 12-3).



(1) (2)

Fig. 12-2. Phonocardiogram on the heart apex in mitral insufficiency (1) and in mitral stenosis (2): I, II – heart sounds, 3 – pansystolic murmur, 4 – diastole, 5 - opening snap, 6 – protodiastolic murmur, 7 - presystolic murmur



(1) (2)

Fig. 12-3. Phonocardiogram on II interspace at the right sternum edge in aortal stenosis (1) and in aortal insufficiency (2): I, II – heart sounds, 5 – “diamond shape” pansystolic murmur, 6 – protodiastolic descending murmur

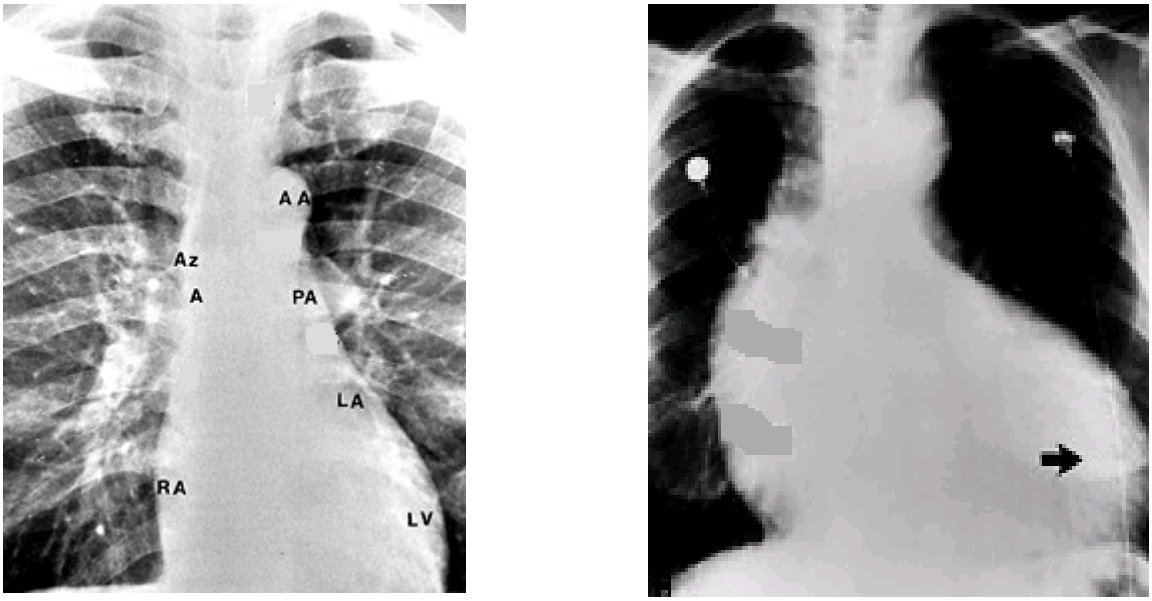
Computer-aided auscultation is currently used for auscultation by an ***electronic (digital) stethoscope*** (Fig. 12-4). An electronic stethoscope allows not only listening to the patient’s heart, but also watching the phonocardiogram directly on the display in real time, or transmitting information to a computer for later visualization of heart sounds in the form of a phonocardiogram. It allows for determination hidden diastolic or systolic murmurs of the heart. The

technology blocks unwanted background noises during auscultation, which come from the environment and from the patient's body. The electronic stethoscope has a built-in algorithm, according to which it itself identifies suspicious heart murmurs and shows them on a separate scale. Another advantage of this device over a conventional stethoscope is recording results and coping them onto a computer. Thus, the doctor can attach the record to the patient's case report and return to it later to analyze the dynamics of the disease.

Fig. 12-4. Electronic stethoscope

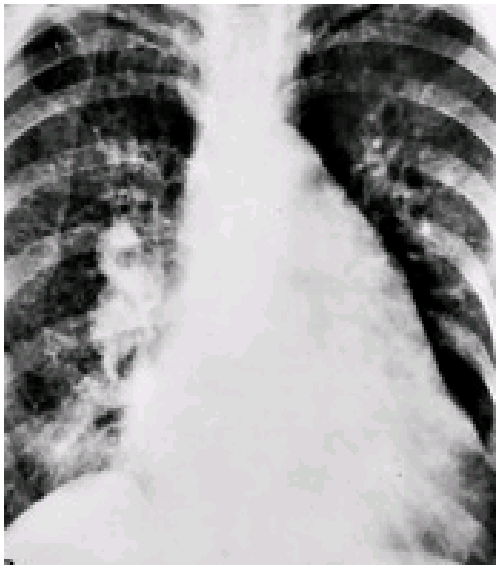
12.1.2. Chest roentgenogram

Frontal and lateral chest films should be obtained to evaluate the heart size, the heart shape and configuration, chamber analysis, and the nature of the lung fields, especially the vasculature (Fig.12-5, 12-6).

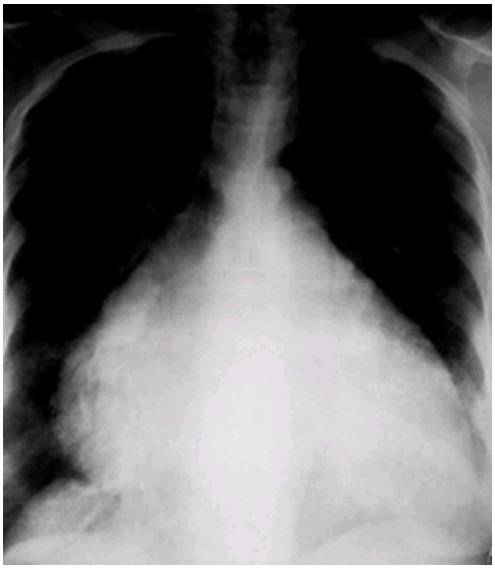


Normal heart configuration Aortal configuration in aorta stenosis

Fig. 12-5. Frontal X-ray of the heart in the norm and in aortal stenosis:
A – aorta, AA – aorta arch, Az – v. azygos, LA – left atrium, LV – left ventricle, PA – pulmonary artery, RA – right atrium



Mitral configuration in mitral stenosis



Trapezoidal configuration in exudative pericarditis

Fig. 12-6. Frontal X-ray of the heart in mitral stenosis and in exudative pericarditis

Heart size is often unequivocally normal despite a severe heart disease, especially an ischemic heart disease (IHD), and increased afterload (e.g., in aortic stenosis). Thus, measuring heart size is mainly helpful for statistical and serial studies of a patient.

Heart shape abnormalities can be difficult to interpret. Mediastinal tumors and pericardial tumors or defects are occasionally confused with abnormal chamber enlargement. *Heart contours* and *heart configuration abnormalities* can be determined by chest X-ray (see Chapter 9. Table 9-2).

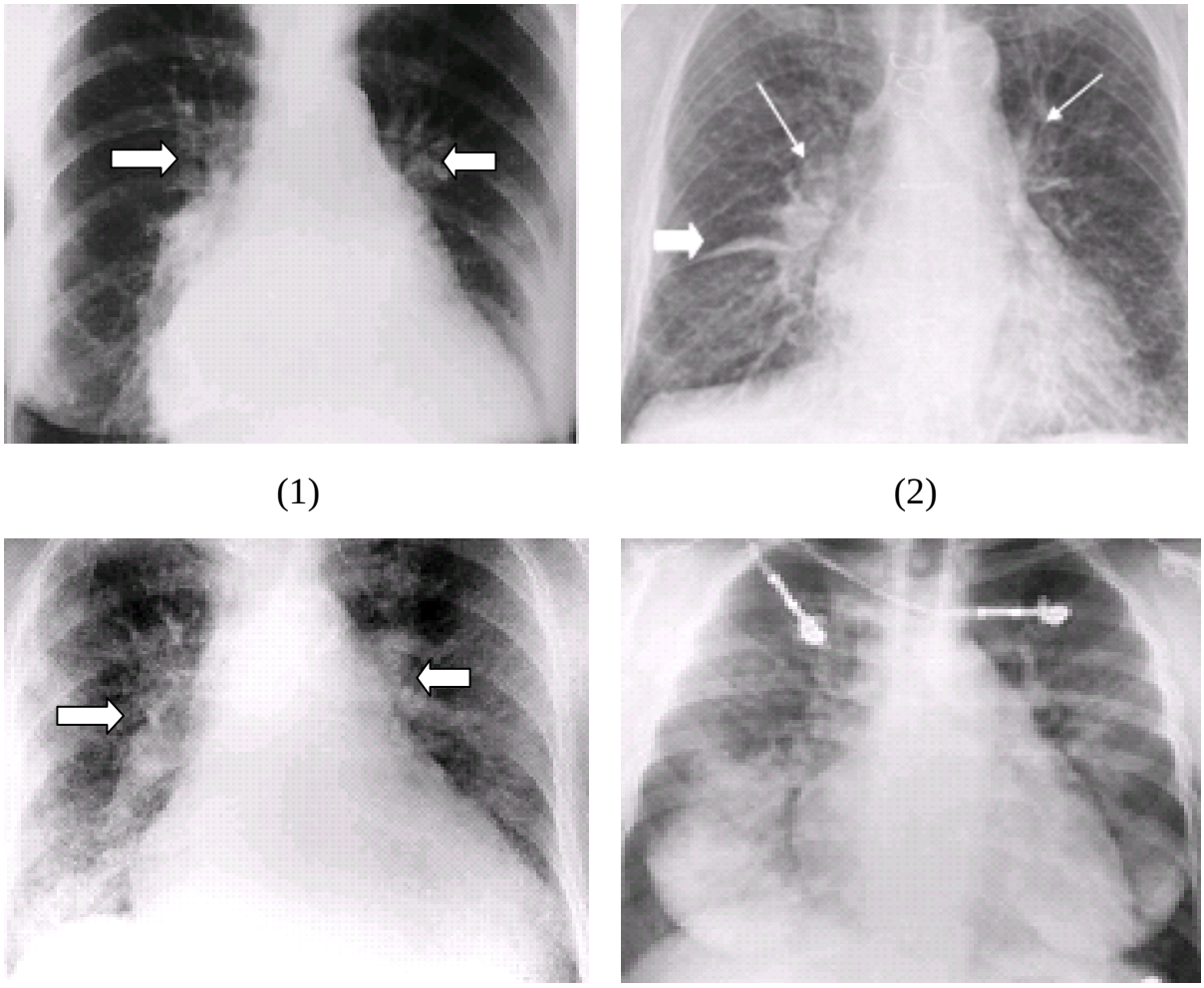
Chamber size is difficult to estimate on plain film because the chambers overlap and are covered by other structures (e.g., pericardium, mediastinal fat, diaphragm). Conventional signs of specific chamber enlargement are frequently difficult to apply and are sometimes misleading. Despite these limitations, chamber size estimation can be worthwhile.

Great vessel configurations and vascular changes in the lungs are extremely important in assessing cardiac function. In cardiac diagnosis, the appearance of the lung fields is often more helpful than the appearance of the heart (Fig. 12-7).

In *chronic heart failure*, typical X-ray characteristics of pulmonary venous hypertension are increased pulmonary vasculature in upper lobes, an increased pulmonary veins diameter, larger than the bronchi (ratio >1), and increased mediastinal width.

In *interstitial pulmonary edema* there are *Kerley lines*, peribronchial cuffing, and interlobar fissure thickens (brighter white line between the lung lobes), blood vessels become less distinct. Kerley lines are thin linear pulmonary

opacities (short - 1-2 cm) peripheral, lower lobe white lines, perpendicular and adjacent to pleura) caused by fluid or cellular infiltration into the interstitium of the lungs.



(1) (2)

(3) (4)

Fig. 12-7. Frontal chest X-ray in congestive heart failure:
(1) dilated pulmonary veins in chronic heart failure; (2) interlobar fissures thickening and Kerley lines (arrows) in interstitial phase of pulmonary edema; (3) interstitial pulmonary edema (peribronchial cuffing); (4) alveolar pulmonary edema ("bat wing" edema)

A*lveolar phase of pulmonary edem****a*** is characterized by alveolar infiltrates ("cottonwool" appearance) centrally and in basal regions ("bat-wing" or "butterfly" appearance). Acute pulmonary edema (in acute left ventricular failure) may present with diffuse white-out appearance.

12.1.3. Echocardiography

Echocardiography (EchoCG) is an ultrasound technique for diagnosing cardiovascular disorders. It is subdivided into M-mode, two-dimensional (2-D), spectral Doppler, colour Doppler, contrast, and stress echocardiography.

M-mode echocardiography is performed by directing a stationary-pulsed ultrasound beam at some portion of the heart. As the beam passes through the heart, echoes from hearts chambers and valves can be seen.

2-D (or cross-sectional) echocardiography has become the dominant echocardiographic technique. It uses pulsed, reflected ultrasound to provide spatially correct real time images of the heart, which are recorded on videotape. Four commonly used 2-D echocardiographic views can provide multiple tomographic views of the heart and great vessels and make easy diagnosis of heart valvular diseases, congenital heart diseases, and contractile dysfunction of heart in various diseases.

Spectral Doppler echocardiography uses ultrasound to record the velocity and direction of bloodstream. The spectral Doppler signal is displayed on a strip chart recorder or a videotape. Colour Doppler echocardiography is essentially 2-D Doppler echocardiography with flow encoded in color to show its direction (red is toward and blue is away from the transducer). *Doppler auscultation* using an hand-held ultrasound transducer enables the auscultation of valves movements and blood flow sounds that are undetected during cardiac examination with a conventional stethoscope.

Contrast echocardiography is an M-mode or 2-D echocardiographic examination during which contrast medium is injected into the cardiovascular circulation. Almost any liquid contrast medium that is rapidly injected into the cardiovascular space acquires microbubbles in suspension, which produce a cloud of echoes within the cardiac chambers.

Stress echocardiography is performed during or after physical or pharmacologic stress.

Main echocardiographic parameters of the heart in the norm (Fig. 12-7):
- Aorta diameter - 20-40 mm, divergence of aortic valve - not less than 15 mm;
- Pulmonary artery diameter - no more than 28 mm, a normal pulmonary artery systolic pressure at rest is 18-25 mm Hg, with a mean pulmonary pressure ranging from 12-16 mm Hg;
- Left atrium diameter at the end of systole is not more than 40 mm;
- Left ventricle end-systolic dimension - 26-40 mm, end-diastolic dimension - 40-56 mm, posterior wall thickness - 12 mm.
- Interventricular septum thickness - no more than 12 mm.
- Mitral valve cusps divergence - 22-36 mm, area of the mitral orifice - 4-6 sm^2.
- Right ventricle end-diastolic diameter - 30 mm.
- Ejection fraction - 55-75%.

Echocardiographic signs of the left ventricle hypertrophy are thickening posterior wall of the left ventricle and (or) interventricular septum more than 12 mm.

*Mitral regurgitation (insufficienc*y) is diagnosed during Doppler ultrasound registering cavity of the left atrium systolic turbulent flow, beginning after the closing of the mitral valve.

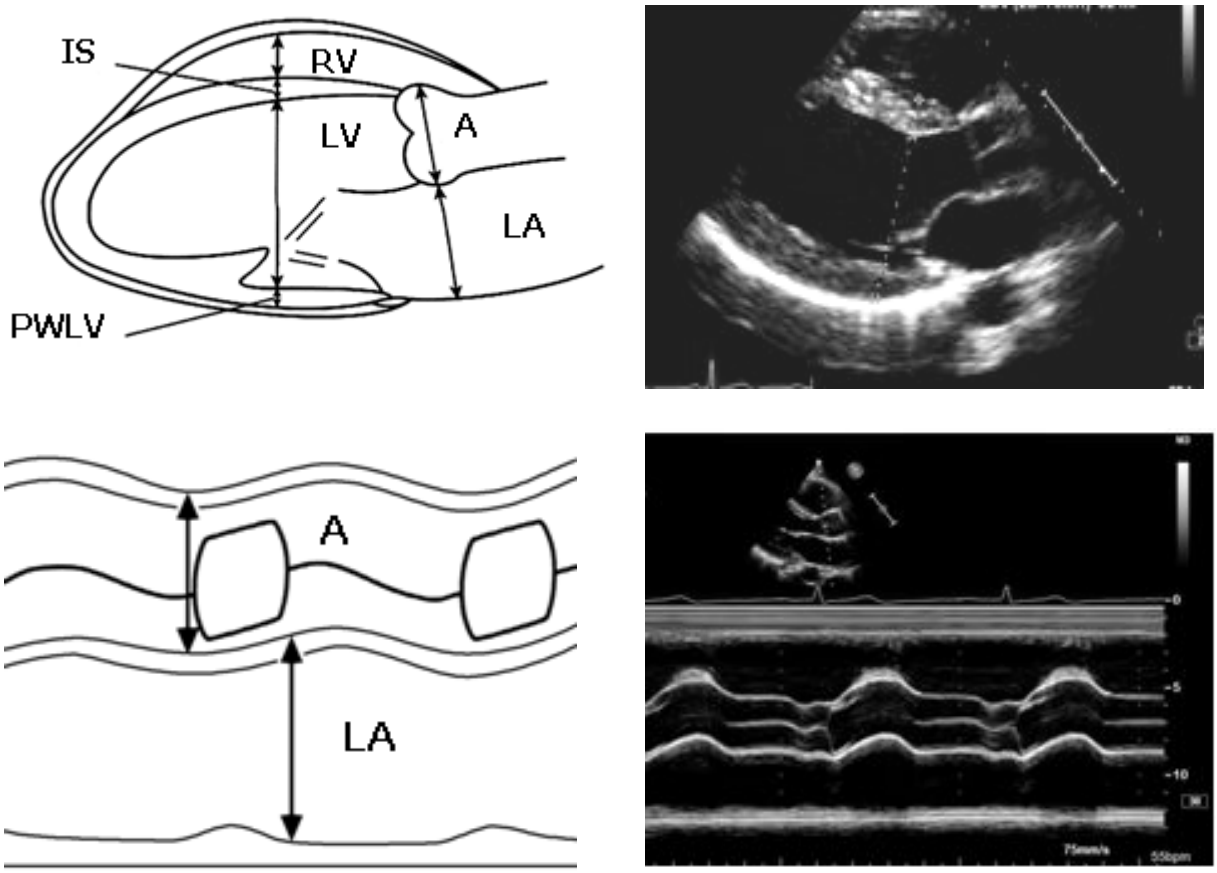


Fig. 12-7. Echocardiography determination of the basic size of the heart:
A – aorta, IS – interventricular septum, LA – left atrium, LV – left ventricle, PWLV – posterior wall of the left ventricle, RV – right ventricle.

Mitral stenosis is determined at reducing mitral orifice area less than 2.5 cm^2 and an increase in the pressure gradient transmitral diastolic flow.

Aortic regurgitation (insufficiency) is diagnosed during the registering Doppler ultrasound outflow tract of the left ventricle diastolic flow turbulent beginning after the closure of the aortic valve and ending at its opening.

Aortic stenosis is determined at reducing divergence of the aortic valve - less than 15 mm, reducing the area of the aortic opening less than 2.0 cm^2 and by increase the maximum pressure gradient between the left ventricle and the aorta more than 25 mm Hg.

Infective endocarditis. The main ultrasonographic findings is the identification of the infectious endocarditis vegetations (blood clots), which are attached to the cusps of the valves. There may be infringements of integrity of the valve cusps leaflets and chords that lead to the damaged valve incompetence.

Ischemic heart disease (IHD). In IHD, echocardiography can:

- reveal systolic and diastolic dysfunction of ventricles,
- identify areas of violation of local contractility (zones of hypo- and akinesia),

- the presence of IHD complications - heart wall aneurysm, thrombosis, left ventricular myocardial infarction, pericardial and pleural cavities effusion, ventricular septum rupture.

Pulmonary hypertension is detected by the average pulmonary artery pressure of 25 mm Hg or more.

Pericardial effusion. Normally, 20-60 ml of physiological fluid present in the pericardial cavity, wherein during echocardiography separation of pericardium membranes is determined only during systole. In exudative pericarditis and in hydropericardium more than 100 ml of liquid are accumulated that pericardial content looks like a non-echogenic space between the pericardium membranes.

12.1.4. Myocardial perfusion imaging

Myocardial perfusion imaging can be used for initial evaluation of certain patients with a chest pain (i.e., mainly those with pain of uncertain origin) to determine the functional significance of coronary artery stenosis or collateral vessels seen on angiography and to follow up procedures such as bypass surgery, transluminal angioplasty, or thrombolysis. This imaging technique can be used to estimate prognosis after acute MI because it can reveal the extent of the perfusion abnormality associated with the acute MI and the extent of scarring from previous infarcts. Myocardial perfusion imaging usually uses radioactive thallium (^{201}Tl), which behaves as potassium analog. After intravenous administration, ^{201}Tl rapidly leaves the vascular compartment and enters the cells in proportion to initial blood flow.

Radionuclide method can be helpful in diagnosing myocardial infarction. Myocardial perfusion imaging with ^{201}Tl or ^{99}Tc-sestamibi, which are distributed in proportion to myocardial blood flow and concentrated by viable myocardium reveal a defect ("cold spot") in most patients during the first few hours after development of a transmural infarction. However, although perfusion scanning is extremely sensitive, it cannot distinguish acute infarcts from chronic scars.

12.1.5. Cardiac catheterization and coronary angiography

Cardiac catheterization and coronary angiography are invasive techniques under X-ray control.

Cardiac catheterization is the passage of a catheter through peripheral arteries or veins into the chambers of the heart, pulmonary artery, coronary arteries and veins. Cardiac catheterization is used for various studies, including angiography, intravascular ultrasound, cardiac output measurement, endomyocardial biopsy. These methods allow you to determine the anatomical features of the coronary arteries and the heart, the heart function and hemodynamics, which are of a great importance for clarifying the diagnosis and choosing a treatment method. Cardiac catheterization also serves as the basis for coronary angiography.

Coronary angiography (coronarography) is an invasive method of radiographic imaging of the coronary arteries after selective intracoronary administration of a radiopaque substance. Coronarography is the most accurate and reliable way to diagnose ischemic heart disease, allowing to accurately determine the nature, the location and the degree of the coronary artery stenosis. The results of coronary angiography will help determine the method of subsequent treatment – medication therapy or surgical intervention. Coronarography determines the indications for myocardial revascularization surgery: stenting of the coronary artery or coronary artery bypass grafting, which can be performed at the same session.

12.2. Laboratory tests in cardiovascular system diseases

12.2.1. Laboratory tests in acute rheumatic fever

Common blood analysis shows moderate neutrophylic leucocytosis (with a shift to the left) in an acute rheumatic fever. ESR is always increased (by 50-70 mm/h in grave cases).

Biochemical tests include increase of C-reactive protein (CRP), α2-globulin and γ-globulin fractions, fibrinogen. The level of mucoproteins increases, and it can be revealed by a diphenylamine test (increase of seromucoid and sialic acids).

Microbiology study detects group A streptococci positive throat culture (throat swab or rapid antigen detection test).

Immunological tests discovers the increased titers of group A streptococcal antibodies (anti-streptolysin O, anti-deoxyribonuclease B, anti-hyaluronidase) in the blood serum.

12.2.2. Laboratory tests in acute myocardium infarction

Leucocytosis starts the increase in 2-3-hours, peaks on 2-d – 4-th days, and returns to normal in blood on 3-d – 10-th days of the disease. It is a result of the reactive processes which depend on the absorption of the autolysis products from the site of infarction. The larger the necrotized area, the higher is the temperature and the longer the pyretic period and leucocytosis. *ESR* begins the increase later in 2-3 days of the disease, peaks in 1 week, when leucocytosis decreases, and ESR returns to a normal from the second week of the disease.

Diagnosis of myocardial infarction depends substantially on the determination of the activity of some blood ***serum cardiac markers*** *(myocardiospecific enzymes and proteins) which are released due to necrotic changes in the myocardium.*

Cardiac markers are substances that are released into the blood when the heart is damaged (Table 12-1). Measurements of these biomarkers are used to help diagnose acute coronary syndrome (ACS) and cardiac ischemia, conditions associated with insufficient blood flow to the heart. ***Cardiac markers include myocardiospecific enzymes and proteins. Diagnostic value has elevation of***

cardiac markers (in 2 and more times than normal) accompanied clinical or ECG-signs of cardiac ischemia.

Myocardiospecific enzymes study includes the activity of the ***MB-fraction of creatine phosphokinase (CK-MB)*** and the ***first enzyme of lactic dehydrogenase (LDH_1)*** increase the first day of acute myocardium infarction.

The activity of CK-MB normalizes in 2-3 days, of aminotransferases - in 4-5 days, and of LDH_1 in 10-14 days. *CK-MB,* the myocardial component of CK, is specific in diagnostics of MI. It is found in blood within 6 h of myocardial necrosis. Levels are elevated for 36 to 48 h. Although small amounts of CK-MB are found in other tissues, elevations of CK with > 40% MB are diagnostic when associated with clinical findings suggestive of MI. Routine measurement of CK-MB on admission and 6 to 8 h for the first 24 h will confirm or reject the diagnosis. Normal CK-MB for 24 h virtually rules out MI.

Table 12-1. Cardiac Markers for the Detection of Myocardial Necrosis

Marker	Onset of increase in blood	Peaks in blood	Return to normal in blood	Advantages	Disadvantages
Myoglobin	1-2 h	4-12 h	in 24 h	Early AMI marker	Poor specificity
CK-MB	4-9 h	24 h	in 2-3 d	Routine AMI marker; detection of reperfusion	Less cardiospecific than troponins
Troponins T and I	4-9 h	12-24 h	in 7-14 d	Most cardio-specific marker; useful for detecting AMI >48 h from onset	Less effective than CK-MB for detecting reperfusion
LDH_1	12-24 h	72 h	in 10-14 d	AMI marker in case of late (in 2-3 and more days) appeal for medical aid	Less cardiospecific than troponins and CK-MB

Notes: AMI – acute myocardial infarction; h – hours, d – days, w – week.

Myoglobin and the contractile proteins ***troponin-T and troponin-I*** are also released by infarcted myocardium.

Troponin-T and troponin-I *[(Cardiac-specific troponin T (cTnT) and cardiac-specific troponin I (cTnI)]* appear to be highly sensitive markers of myocardial injury and may replace conventional CK-MB analysis in early

decisions in patients with a chest pain and nondiagnostic ECG. Cardiac-specific troponins - troponin-T and troponin-I are found in blood within 2-4 h of myocardial necrosis. Levels are elevated for 7 days. Troponins are released in some patients with unstable angina, and the activity level predicts future adverse events.

Myoglobin is released into the blood within just a few hours of the onset of acute MI. Although myoglobin is one of the first serum cardiac markers that rise above the normal range after AMI, it lacks cardiac specificity, and it is rapidly excreted in the urine, so that blood levels return to the normal range within 24 h of the onset of infarction.

12.2.3. Lipid tests

Dyslipidemia is elevation of plasma cholesterol, triglycerides, or both, or a low high-density lipoprotein level that contributes to the development of atherosclerosis. Causes may be primary (genetic) or secondary. Diagnosis is by measuring plasma levels of total cholesterol, triglycerides, and individual lipoproteins (Table 12-2).

Elevated levels of low density lipoprotein (LDL) and triglycerides, and reduced levels of high density lipoprotein (HDL) predispose to atherosclerosis. The association of total serum cholesterol and LDL cholesterol levels with the risk of IHD (ischemic heart disease) is direct and continuous. HDL levels are inversely correlated with IHD and arterial hypertension risks.

Table 12-2. Blood serum lipid tests

Test	Normal values
Total cholesterol	3.2—5.6 mmol/l
Low density lipoprotein (LDL)	1.71—3.5 mmol/l
High density lipoprotein (HDL)	>0.9 mmol/l
Triglycerids	0.41-1.8 mmol/l

Primary causes of dyslipidemia are single or multiple gene mutations that result in either overproduction or defective clearance of triglycerides and LDL cholesterol, or in underproduction or excessive clearance of HDL.

Secondary causes of dyslipidemia contribute to many cases of dyslipidemia in adults. The most important secondary cause in developed countries is a sedentary lifestyle (hypodynamia) with excessive dietary intake of easily digestible carbohydrates and saturated fat, cholesterol, and trans fats. Trans fats are polyunsaturated or monounsaturated fatty acids to which hydrogen atoms have been added; they are commonly used in many processed foods and are as atherogenic as saturated fat. Other common secondary causes include diabetes mellitus, alcohol overuse, chronic kidney disease, hypothyroidism, primary biliary cirrhosis and other cholestatic liver diseases,

and drugs, such as thiazides, β-blockers, retinoids, highly active antiretroviral agents, cyclosporine, estrogen and progestins, and glucocorticoids. Secondary causes of low levels of HDL cholesterol include cigarette smoking, anabolic steroids, HIV infection, and nephrotic syndrome.

Diabetes mellitus is an especially significant secondary cause because patients tend to have an atherogenic combination of high triglycerides; high small, dense LDL fractions; and low HDL. Patients with type 2 diabetes are especially at risk. The combination may be a consequence of obesity, poor control of diabetes, or both. Women with diabetes may be at special risk of cardiac disease from this form.

Lipid measurement should be accompanied by assessment of cardiovascular risk factors, defined as diabetes mellitus, cigarette use, arterial hypertension, family history of IHD in a male 1st-degree relative before age 55 or a female 1st-degree relative before age 65.

12.2.4. B-type Natriuretic Peptide (BNP) Blood Test

BNP *is a substance secreted from ventricles chambers of the heart in response to changes in pressure that occur when heart failure develops and worsens.* ***Blood serum BNP increases when heart failure symptoms worsen, and decreases when the heart failure condition is stable***. The BNP level in a person with heart failure – even someone whose condition is stable – is higher than in a person with normal heart function.

BNP levels below 100 pg/mL indicate no heart failure.

BNP levels of 100-300 pg/mL suggest heart failure is present.

BNP levels above 300 pg/mL indicate heart failure.

12.3. The key points on the theme "Laboratory-Instrumental Methods of Cardiovascular System Examination"

Phonocardiography (PCG) is possible to catch nuances in the change of heart sounds and murmurs that are not auscultated by ear. PCG has a diagnostic value especially for heart valves disease diagnosis (for example, mitral and aortal stenosis and incompetence).

Chest X-ray can determine pathologic changes of the heart and greater blood vessels contours, heart configuration, gives additional data about heart chamber size, and detects vascular changes in the lungs and pulmonary edema, that important for assessment of cardiac dysfunction.

Echocardiography (EchoCG) allows for assessment of the myocardium thickness and movement, and volume of heart chambers, to assume the presence of myocardial infarction, to identify fluid in a pericardial cavity. EchoCG can be used to evaluate the ability of both the systolic and the diastolic ventricular filling, that can help in the assessment of ventricular hypertrophy and heart failure. EchoCG is also used to determine structural and functional abnormalities of the heart valves; to detect the valve vegetations and intracardiac thrombi; it allows to estimate the pressure in the pulmonary artery.

Myocardial radionuclide perfusion scanning is extremely sensitive but is not specific in diagnosis of myocardial infarction.

Coronarography is the "gold standard" in diagnosis of the ischemic heart disease, because it enables to determine the exact details of the anatomical structure of the entire coronary circulation.

Changes of blood serum tests in acute rheumatic fever (ARF) and other inflammatory diseases of the circulatory system: increased levels of C-reactive protein (CRP), α2-globulin and γ-globulin fractions, fibrinogen, seromucoid and sialic acids. ***Immunological tests in ARF*** discovers increased blood serum titers of group A streptococcal antibodies (anti-streptolysin O, anti-deoxyribonuclease B, anti-hyaluronidase).

Changes of biochemical parameters in myocardial infarction: increased blood serum levels of MB-fraction of creatine phosphokinase (CK-MB), troponins T and I, myoglobin, lactate dehydrogenase (LDH), and its first isoenzyme (LDH 1).

Atherogenic dyslipidemia (association of elevated total serum cholesterol, LDL cholesterol, and triglycerides levels) is associated with the high risk of IHD (ischemic heart disease) and arterial hypertension.

Brain natriuretic peptide (BNP) is a marker for assessing the functional state of contractile potential of myocardium; it is diagnostic in the heart failure.

12.4. Assessment tests on the theme "Laboratory-Instrumental Methods of Cardiovascular System Examination"

1. Phonocardiogram characteristics on the heart apex in the mitral valve insufficiency:

1. weakened I heart sound;
2. loud snapping I heart sound;
3. weakened II heart sound;
4. pansystolic murmur;
5. protodiastolic descending murmur.

2. Phonocardiogram characteristics on the heart apex in the mitral valve stenosis:

1. weakened I heart sound;
2. loud snapping I heart sound;
3. weakened II heart sound;
4. opening snap;
5. protodiastolic and presystolic murmurs.

3. Phonocardiogram characteristics on II-d interspace at the right sternum edge in the aortal stenosis:

1. weakened I heart sound;
2. loud snapping I heart sound;

3. weakened II heart sound;
4. “diamond shape” pansystolic murmur;
5. protodiastolic and presystolic murmurs.

4. Phonocardiogram characteristics on II-d interspace at the right sternum edge in the aortal insufficiency:
1. weakened I heart sound;
2. loud snapping I heart sound;
3. weakened II heart sound;
4. “diamond shape” pansystolic murmur;
5. protodiastolic descending.

5. Pathologic types of the heart configuration are:
1. cor pulmonale;
2. cor bovinum;
3. trapezoidal;
4. aortic;
5. mitral.

6. Frontal chest X-ray characteristics in the congestive heart failure:
1. dilated pulmonary veins;
2. interlobar fissures thickening;
3. Kerley lines;
4. peribronchial cuffing;
5. infiltrative shadows on pulmonary fields.

7. Echocardiography has diagnostic value in:
1. heart failure;
2. arrhythmia and conduction disorders of the heart;
3. cardiomegaly;
4. heart valves diseases;
5. congenital heart diseases.

8. What changes of the common blood analysis are important in diagnosis of rheumatic carditis?
1. leucopenia;
2. leukocytosis;
3. left shift of the leucocyte formula;
4. right shift of the leucocyte formula;
5. increase of ESR.

9. What blood serum tests are important in diagnosis of acute rheumatic fever?

1. C-reactive protein (CRP);
2. α2-globulin and γ-globulin fractions;
3. fibrinogen;
4. MB-fraction of creatine phosphokinase;
5. anti-streptolysin O.

10. What changes of the common blood analysis are typical in myocardium infarction?
1. leucocytosis develops on the 2-d or 3-d day from onset of the disease;
2. left shift of the leucocyte formula;
3. right shift of the leucocyte formula;
4. increase of ESR develops 2-d or 3-d day from onset of the disease;
5. ESR begins elevation, and leucocytosis decreases from the second week of the disease.

11. What enzymes are increased specifically in biochemical blood analysis in acute myocardial infarction?
1. creatine phosphokinase-MB fraction (CK-MB);
2. LDG_1;
3. alpha-amilase;
4. alkaline phosphatase (AP);
5. lipase.

12. Atherogenic dyslipidemia characteristics are:
1. hypocholesterolemia;
2. hypertriglyceridemia;
3. elevation of low density lipoprotein (LDL);
4. elevation of high density lipoprotein (HDL);
5. low level of high density lipoprotein (HDL).

13. B-type Natriuretic Peptide (BNP) level is important for diagnosis in:
1. myocardial infarction;
2. brain stroke;
3. heart failure;
4. renal failure.

14 What cardiac markers are increased specifically in biochemical blood analysis in acute myocardial infarction?
1. CK-MB;
2. LDG_1;
3. troponin I;
4. troponin T;
5. myoglobin.

Chapter 13. Basic Clinical Syndromes of the Cardiovascular System Diseases

Goals: to enable students to learn –

1) clinical symptoms and laboratory-instrumental signs of basic clinical syndromes in cardiovascular system diseases;
2) acute coronary syndrome, syndromes of the heart failure, myocardium hypertrophy and dilatation of the heart chambers (cardiomegaly), heart arrhythmia and conduction system disorders, heart valves disease.

13.1. Acute coronary syndrome

Definition: ***Acute coronary syndrome (ACS)*** is a set of clinical symptoms of myocardial ischemic pain attacks (>10-15 minutes, non-responsiveness to nitroglycerin) permitting to suspect *unstable angina pectoris or acute myocardial infarction* with elevation ST or without elevation ST.

Acute coronary syndrome is caused primarily by coronary artery atherosclerosis. Most cases of ACS occur from disruption of a previously nonsevere lesion (an atherosclerotic lesion that was previously hemodynamically insignificant yet vulnerable to rupture).

Acute coronary syndrome consisting of chest pain, ischemic ST-segment and T-wave changes, elevated levels of biomarkers of myocyte injury has been shown to occur in the absence of clinical ischemic heart diseases (IHD) after emotional or physical stress.

Clinical picture of ACS

Symptoms:

Pain lasting more 10-15 minutes, which is usually described as pressure, squeezing, or a burning sensation across the precordium (behind the sternum) and may radiate to the neck, shoulder, jaw, back, upper abdomen, or either arm.

Exertional dyspnea that resolves with pain or rest;

Pain is not responsive to sublingual nitroglycerin;

Palpitations;

Diaphoresis from sympathetic discharge;

Nausea from vagal stimulation;

Decreased exercise tolerance.

Physical findings can vary from normal to any of the following:

General inspection demonstrates cool, clammy skin and diaphoresis in patients with a cardiogenic shock. Jugular venous swelling may occur;

Signs of left ventricle (LV) heart failure - mixed dyspnea, forced position orthopnea, moist rales in inferior-posterior parts of the lungs may suggest LV dysfunction, widening a left border of relative heart dullness. In addition, a third heart sound (S3) may be present, and frequently, a fourth heart sound (S4)

exists. Gallop rhythm may occur. Systolic murmur at the apex is auscultated secondary to mitral regurgitation because acute dilation of LV;

Arterial hypotension indicates ventricular dysfunction due to myocardial ischemia, infarction, or acute valvular dysfunction;

Arterial hypertension may precipitate angina pectoris or reflect elevated catecholamine levels due to anxiety or to exogenous sympathomimetic stimulation.

***ECG* remains the major diagnostic procedure of acute coronary syndrome.** Variants of ECG patterns with acute myocardial ischemia are (see Chapter 11. Fig. 11.13-.11.19; Fig. 13-1):

- non–infarction subendocardial ischemia - transient ST depressions;
- non–infarction transmural ischemia - transient ST elevation or paradoxical T-wave normalization, some times followed by T-waves inversions;
- non–Q-wave (non-ST elevation) infarction - ST depressions or T-inversions without Q-wave;
- non–Q-wave (ST elevation) infarction - ST-elevations followed by T-waves inversions;
- Q-wave–infarction - Q-wave with hyperacute T-waves/ST-elevations followed by T-waves inversions.

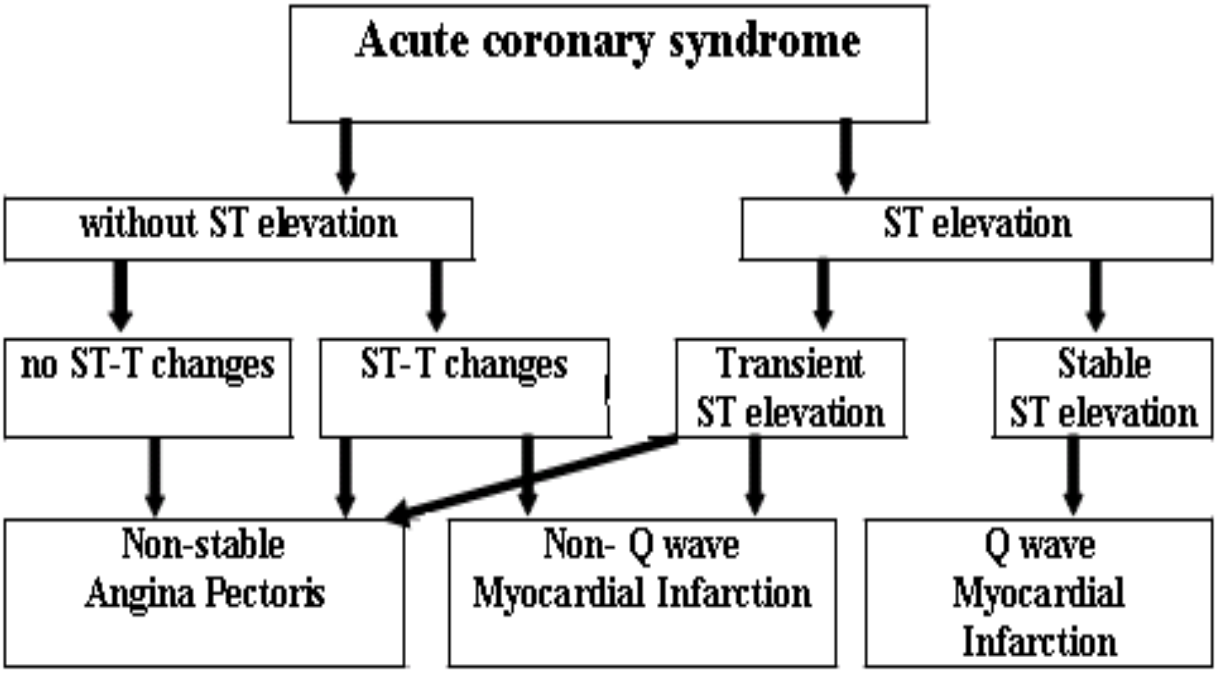


Fig. 13-1. Clinical and ECG-variants of acute coronary syndrome

Diagnosis of an acute coronary syndrome is permitted to use no more than 24-48 hours after the onset of the clinical picture. For quick and reliable differentiation unstable angina pectoris and acute myocardial infarction, the ***serum cardiac markers (CK-MB and/or cardiac-specific troponins - Troponin-***

T and Troponin-I) need be tested in all cases of acute coronary syndrome (see Chapter 12. Table 12-1). *Levels of the serum cardiac markers greater twice than the upper limit of normal confirm diagnosis of acute myocardial infarction*.

13.2. Syndromes of heart failure

Definition: ***Heart failure*** is an inability of a cardiovascular system to provide the body's need for blood (oxygen) at rest and during an exercise.

There are an *acute and chronic heart failure.*

13.2.1. Causes of heart failure

Etiology of heart failure

Heart failure is a polyetiological clinical condition:

1. *Heart failure associated with diseases that primarily affect myocardium and violate metabolism in it.* It is observed in the following types of pathology:
1) infectious, inflammatory and toxic lesions of myocardium (myocarditis, intoxication with alcohol, drugs and other poisons);
2) insufficient blood supply to myocardium (coronary atherosclerosis , anemia);
3) metabolic disorders, avitaminosis, disorders of endocrine system (thyrotoxicosis);
4) cardiomyopathy.

2. *Heart failure due to overload or overstrain of myocardium*, which occurs with pathological changes in the heart or bloodstream (heart valves disease, increased pressure in greater or lesser circle of blood circulation). Various causes lead to predominant overload of the left or the right ventricle or to an overload of the whole heart.

Pathogenesis of heart failure

Heart failure may be result as a violation of pumping function of the heart (*systolic heart failure*), as well as a violation of relaxation of ventricular myocardium (*diastolic heart failure*). In most cases of the heart failure, there is a combination of heart and vascular insufficiency.

Hemodynamic changes in circulatory insufficiency include:

- Decreased cardiac output and minute blood volume;
- Speed of blood flow decreases;
- Venous and capillary pressure increases;
- Systolic arterial pressure remains normal or decreased;
- Diastolic pressure slightly increases, and pulse pressure decreases;
- Decreased renal filtration;
- Sodium chloride and water retention;
- Abnormal gas exchange – development of hypoxemia and hypercapnia;
- Increasing a number of red blood cells (erythrocytosis) and blood viscosity.

13.2.2. Syndromes of acute heart (left- and right ventricle) failure

Definition: ***Acute heart failure*** is a rapid (a few minutes or hours) development of myocardial contractility disorders and a decrease in systolic and minute blood volumes, which is manifested by circulatory insufficiency and a sharp violation of internal organs (lungs, kidneys, brain), and poses an immediate threat to the life of the patient.

Acute heart failure (*cardiac asthma, pulmonary edema*) may also occur against the background of a long-term chronic heart failure.

Acute cardiovascular failure is characterized by the syndromes of an acute heart failure (left ventricular, right ventricular) and vascular insufficiency (collapse, shock, syncope).

Syndrome of acute left ventricular heart failure

Etiology: The main causes of the acute left ventricular failure are myocardial infarction, severe arterial hypertension (with a rapid rise in blood pressure), severe diffuse myocarditis, heart valve defects (mitral, aortic).

Clinical manifestations of the acute left ventricular heart failure may be in two variants - cardiac asthma and pulmonary edema. *The main clinical syndrome that characterizes the acute left ventricular failure is pulmonary edema*.

Pathophysiology: Its main hemodynamic factors are weakening left ventricle and acute congestion in a pulmonary circulation. Pressure in the pulmonary veins, capillaries, arterioles and capillary permeability are increased. There are seepage of blood liquid portion and swelling of the alveoli walls (*interstitial pulmonary edema*), then its appearance in the lumen of alveoli (*alveolar pulmonary edema*), violation of gas diffusion, increased platelet aggregation, and microatelectasis formation.

Interstitial pulmonary edema corresponds to the clinical picture of cardiac asthma.

Cardiac asthma

Cardiac asthma are attacks of severe dyspnea.

Cardiac asthma clinical picture: Complaints: feeling lack of air, suffocation, dry cough, heart palpitations, sleep disturbances.

General inspection: a suffering facial expression, a forced position – orthopnea; skin - moist, pale, acrocyanosis; an excited state of the patient, anxiety, puffiness of the face.

Objective examination of the respiratory system: frequent, shallow, rhythmic breathing. Palpation of the chest – weakening tactile fremitus, rigidity of the chest. Lung percussion: initially blunted tympanic sound, then dull sound in the posterior parts. Auscultation of the lungs: initially weakened vesicular breathing, then fine crackles, single rhonchi and coarse crackles.

Palpation of the heart and large blood vessels: reducing height and resistance of the apical impulse, increasing its area, and lateral shift.

Radial artery pulse - frequent, small filling, often threadlike, arrhythmic, there may be a pulse deficiency. Blood pressure – initially normal, further arterial hypotension.

Percussion of the heart: signs of an underlying disease, often widening the left and the upper borders of the relative heart dullness, and widening contours of the relative heart dullness may occur.

Auscultation of the heart: sounds are muffed, weakening S1, gallop rhythm, accented S2 on the pulmonary artery, arrhythmia may occur.

Pulmonary edema

Pulmonary edema clinical picture: Transition from interstitial pulmonary edema to alveolar edema sometimes occurs very quickly – within a few minutes. The existing symptoms are aggravated. Sometimes pulmonary edema develops without previous signs of cardiac asthma (against background of hypertensive crisis, myocardial infarction, paroxysmal ventricular tachycardia). Pulmonary edema may be accompanied by a cardiogenic shock.

Pulmonary edema is characterized by severe dyspnea, cough with pink foamy sputum, noisy gurgling breath, heart palpitations.

General inspection: impaired consciousness, forced position - orthopnea, cyanosis, swollen cervical veins, cold sweat.

Percussion of the lungs: dull sound in inferior-posterior departments.

Lung auscultation: weakened vesicular breathing, and coarse crackles (small-, medium- and large-bubbling wet rales).

Radial artery pulse - frequent, small filling and tension, thread-like, irregular, deficiency of the pulse may occur. *Blood pressure* is reduced.

Auscultation of heart: S1 and S2 are muffed; gallop rhythm, weakening S1, accent of S2 on the pulmonary artery.

X-ray of the lungs: the uncertainty of a pulmonary pattern, reducing transparency of pulmonary fields in the root zone. X-ray examination shows enlargement of the left ventricle and the atrium, distention of pulmonary veins (due to elevated pulmonary venous pressure).

In the stage of interstitial pulmonary edema, it is a characteristic sign of “peribronchial cuffing”.

Alveolar pulmonary edema: symmetrical shadows in central parts (central form of edema) such as “bat-wing”, or “butterfly” sign. The diffuse form is two-sided diffuse shadows. Focal form – limited or fused shadows of a rounded shape (in alveolar edema).

ECG-changes are characteristic of an underlying disease, signs of the overload of the left ventricle and atrium.

EchoCG: dilatation of the left atrium and ventricle, reduction of myocardial contractility, pulmonary hypertension.

Syndrome of acute right-ventricular heart failure

Etiology: Causes of acute right-ventricular failure may be pulmonary artery, tricuspid regurgitation, mitral stenosis, primary pulmonary hypertension, pulmonary artery valves stenosis.

Pathophysiological basis of this syndrome: is a pronounced venous congestion in the greater circulation.

Clinical manifestations

Complaints: heart palpitations, edema on the inferior extremities, pain in right hypochondrium, heaviness and feeling of fullness in the abdomen, dyspnea, an awareness of the fullness in the neck.

Inspection: cyanosis, edema on the legs, ascites (in advanced stages), swelling of the neck veins, a positive venous pulse.

Radial artery pulse - frequent, small filling, arrhythmic.

Heart percussion: widening the right border of relative heart dullness and transverse length of the heart.

Heart auscultation - dullness of heart sounds, systolic murmur of tricuspid regurgitation over xiphoid process, along the left sternal border, and the right-ventricle gallop rhythm may be heard in advanced stages.

Inspection and percussion of the abdomen: ascites may occur, and epigastric pulsation of the right ventricle may be visible. Liver percussion: liver enlargement and tenderness.

Palpation of the liver: painful and rounded edge, hepatomegaly may occur. *Positive symptom of J. Plesch (hepatojugular reflux, or abdominojugular reflux)* - pressure on the liver is accompanied by swelling of cervical veins; it is typical of increased venous pressure as a result of circulatory insufficiency.

ECG: signs of the right atrium and right ventricle overload, deviation of electrical axis of the heart to the right.

EchoCG: dilation of right atrium and ventricle, reduction of myocardial contractility.

Central venous pressure is dramatically increased.

13.2.3. Syndrome of chronic heart failure (CHF)

Etiology: The most common causes of CHF are ischemic heart disease and myocardial infarction, associated primarily with impaired left ventricle systolic function; other causes of CHF - dilated cardiomyopathy, chronic rheumatic heart disease, and in elderly patients - arterial hypertension and hypertensive heart primarily with development of the diastolic dysfunction of the myocardium.

Pathophysiology: From a pathophysiological point of view, it is distinguished *systolic and diastolic CHF*, however, a mixed form of CHF often occurs. Myocardial diastolic damage presents when ventricles do not properly relax and become stiff, meaning they cannot fill with the blood properly. Diastolic dysfunction usually precedes a disturbance of the systole, but

appearance of the systolic dysfunction often leads to clinical manifestations of the heart failure.

There are *right and left ventricular chronic heart failure* depending on the prevalence of congestion in the pulmonary or the greater blood circulation. Heart failure may be with a low and/or a high cardiac output. The *high-output cardiac failure* occurs in a number of diseases (thyrotoxicosis, anemia, etc.) that can significantly increase the body's need for blood and oxygen with the direct myocardium damage.

Classification of chronic heart failure

In the Republic of Belarus two classifications of chronic heart failure are used in clinical practice – clinical ***classification of chronic heart failure by N.D. Strazhesko and V.K. Vasilenko*** (Table 13-1), adopted at the XII all-Union Congress of Therapeutists in 1935, and ***functional classification of the New York Heart Association (NYHA),*** proposed in 1964 and received worldwide recognition (Table 13-2).

Table 13-1. Classification of chronic heart failure
(according to *N.D.Strazhesko* and *V.K. Vasilenko*)

H	Stage	General characteristic	Clinical signs	Morphology changes
HI	initial latent	latent heart failure	only during serious physical exercises	absent
HIIA	evident compe-nsated	pronounced heart failure, mild hemodynamic disorders	during physical exercises and partly at rest	reversible
HIIB	decom-pensa-ted	pronounced heart failure, grave hemodynamic disorders	during light physical exer-cises and at rest	partly irreversible
HIII	terminal	dystrophic stage of heart failure, grave hemodynamic and metabolic disorders and disability	persistent	irreversible

Functional classes of chronic heart failure according to NYHA (New York Heart Association, 1964):

Class I - no limitation of physical activity, no symptoms with ordinary exertion; ***Class II*** - slight limitation of physical activity, ordinary activity causes symptoms (fatigue, palpitation, dyspnea); ***Class III*** – a marked limitation of physical activity, less than ordinary activity causes symptoms, asymptomatic at

rest; ***Class IV*** - inability to carry out any physical activity without discomfort, symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.

Table 13-2. New York Heart Association (NYHA)
Functional Classification of chronic heart failure

Functional class	Symptoms
I	Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs, etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during the ordinary activity
III	Marked limitation in the activity due to the symptoms, even during less-than-ordinary activity, e.g. walking short distances. Comfortable only at rest
IV	Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients

6 minute walk test (6MWT) to evaluate the functional status of patients with chronic heart failure (CHF) : the patient walks in an acceptable fast pace when functional class 0 (FC 0) is over 550 m, FC I - 550-426 m, FC II - 425-301 m, FC III - 300-151 m, FC IV - not more than 150 m.

Clinical manifestations of chronic heart failure

Inquiry. ***General symptoms of chronic heart failure***, detected already when a patient is questioned – ***heart palpitations, dyspnea (shortness of breath), cough, edema***. In some patients – heaviness in the right hypochondrium (due to the enlargement of the liver).

General survey – ***acrocyanosis, edema, orthopnea, sometimes jaundice (in congested liver), cold hands and feet at palpation.***

Elevation of B-type Natriuretic Peptide (BNP) blood levels supports cardiac etiology of edemas (see Chapter 12. Section 12.2.4).

Cardiovascular signs of circulatory insufficiency are closely related to the dilation of the heart chambers, and relative insufficiency of atrioventricular valves: widening of the relative and absolute dullness of the heart, weak heart sounds, tachycardia, gallop rhythm sometimes, murmurs, pulse – reduction of tension and filling (*low-wave pulse*, *pulsus parvus,* until *pulsus filiformis*), dilation of pulmonary veins on chest X-ray. Chest X-ray and echocardiography can reveal dilation of the heart chambers, heart valves defects, and disorders of heart contractility (see Chapter 12. Sections12.1.2-12.1.3).

Measurement of central venous pressure – 120 mm H_2O and above.

Pulse Oximetry – decrease of O_2 saturation in the blood.

Congested lungs is characterized by congestive bronchitis and congestive pneumonia, and the development of pneumosclerosis with dyspnea and cough, sometimes with separation of a dark-red, bright red sputum (pulmonary hemorrhage may be), foamy sputum (when pulmonary edema). A survey of chest – decreased depth of respiratory movements; somewhat inflated chest (mostly in inferior parts because hydrothorax). Palpation – weakening tactile fremitus. Percussion: at first blunted tympanic sound may occur, then - shortening of percussion sound, and dull sound. Auscultation – weakening vesicular breathing or harsh vesicular breathing, intense rhonchi or coarse crackles, fine crackles in the posterior-inferior parts. Radiography of the chest – congestion in the lungs, enlargement of the heart, sluggish amplitude of the heart contractions.

Congested liver is detected by the enlargement of the liver. The patient feels heaviness or dull pain in the epigastric and right hypochondrium. Development of liver fibrosis with subsequent hepatic dysfunction and portal hypertension may be revealed by an increased density of the liver at palpation, slight jaundice, ascites, dilation of anterior abdominal wall veins, changes in liver biochemical tests (increase of serum bilirubin, AlAT, AsAT, and others).

Congested kidneys is characterized by oliguria, decrease of the daily urine, nocturia; increased specific gravity of the urine, proteinuria, erythrocyturia, casts (cylindruria) may occur in the urinalysis.

Gastro-intestinal signs in circulatory insufficiency - poor appetite, nausea, vomiting, meteorism and constipation); malnutrition (*cardiac cachexia*) may occur because of the gastrointestinal dysfunction.

Cerebrovascular dysfunction in circulatory insufficiency is characterized by a headache, dizziness, heaviness and noise in the head, sleep disturbance, depression.

Blood test: increased red blood cells (erythrocytosis) and hemoglobin, slowing ESR.

Clinical picture of stages of chronic heart failure

In *I (initial latent) stage* of circulatory insufficiency, the patient's work capacity decreases, expressed physical exertion provokes dyspnea, palpitation, and oxygen debt increases to a greater degree than that in healthy people. These symptoms subside at rest.

In *IIA (evident compensated*) *stage* the patient develops dyspnea during an ordinary exercise (e.g. in walking), and his work capacity decreases markedly. The examination reveals moderate cyanosis and edema of the legs. Congestion in the lungs is not expressed. Mild hepatomegaly may occur. The venous pressure increases. *The IIA stage of circulatory insufficiency is reversible under the action of adequate therapy.*

In the IIB (decompensated) stage, typical expressed symptoms (dyspnea) and signs of circulatory failure (pronounced cyanosis, edema, hepatomegaly,

ascites, and dysfunction of various organs) are revealed at rest and worse at a slight physical exertion. Patients are fully disabled. *The IIB (decompensated) stage is partly irreversible under the action of adequate therapy.*

In the III (terminal) stage, metabolic disorders caused by a prolonged circulatory insufficiency. The patient is extremely asthenic, and has prominent symptoms, with irreversible morphological changes in the lungs, liver, and kidneys.

Clinical picture of chronic right and left ventricle failure

There is a division of chronic heart failure on the right and left ventricular variants depending on prevalence of congestion in pulmonary or greater circulation (Table 13-3).

A clinical picture of chronic heart failure may develop in the type of total heart failure. In this case, there is congestion of blood in the veins of both pulmonary and greater circulation.

Table 13-3. Clinical variants of chronic heart failure

Right ventricle failure (in greater circulation)	Left ventricle heart failure (in pulmonary circulation)
Causes: lung emphysema, diffuse pneumosclerosis, tricuspid and pulmonary valves diseases, mitral stenosis, certain congenital heart defects	*Causes:* Arterial hypertension, ischemic heart disease, aortal and mitral valves diseases, diffuse myocarditis
Complaints: - peripheral edema (on inferior extremities); - increased volume of abdomen (ascites); - heaviness in the right hypochondrium	*Complaints:* - dyspnea (worse in a horizontal position); - attacks of night dyspnea; - cough (may be with a pink foamy sputum); - reduced tolerance to physical loads
Objective data : - peripheral cyanosis and edema; -swelling of jugular veins; - ascites; - hepatomegaly; - hydrothorax; - hepato-jugular reflux: swelling of cervical veins with the palpation of the liver	*Objective data :* - forced position - orthopnea; - acrocyanosis; - tachypnea; - non-consonating moist small-bubbling rales in the lungs; - tachycardia; - gallop rhythm

Syndrome of chronic left ventricular failure develops in many diseases attended by affections of the left ventricle (aortic incompetence, mitral failure, arterial hypertension, ischemic heart diseases, etc.).

Hemodynamic disorders usually dominated by the clinical signs of congestion in the veins of the pulmonary circulation (dyspnea, dry cough, forced position of orthopnea, suffocation under the load and at night, and fine and coarse crackles in inferior-lateral parts of the lungs).

Syndrome of chronic right ventricular failure arises in tricuspid and pulmonary valves diseases, mitral stenosis, lung emphysema, and diffuse pneumosclerosis. ***With development of right ventricular heart failure, signs of venous congestion in the greater circulation join.***

The skin is cyanotic, and sometimes becomes ictero-cyanotic. The peripheral veins, especially the neck veins, become swollen, the venous pressure increases, edema and ascites develop, and the liver is enlarged.

Primary dysfunction of one of the heart chambers may eventually cause ***total heart failure***, which is characterized by venous congestion in both the greater and lesser circulation. Moreover, chronic heart failure attended by dysfunction of the entire cardiovascular system arises in diseases affecting the myocardium (myocarditis, intoxication, ischemic heart disease, etc.). Only with primary lesion of the right ventricle or its prolonged overload (*chronic pulmonary heart*), symptoms of the isolated right ventricular chronic heart failure initially appear.

13.2.4. Syndrome of pulmonary heart (Cor Pulmonale)

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

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

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

Cor pulmonale is directly caused by alterations in the pulmonary circulation that lead to pulmonary arterial hypertension, thereby, increasing the mechanical load on RV emptying (after load). However, the most important mechanism leading to pulmonary hypertension is alveolar hypoxia, which results from localized inadequate ventilation of well-perfused alveoli or from a generalized decrease in the alveolar ventilation. Alveolar hypoxia is a potent

stimulus of pulmonary vasoconstriction. Hypercapnic acidosis augments the pulmonary vasoconstriction. During chronic hypoxia, pulmonary hypertension may be intensified by increased blood viscosity arising from secondary erythrocytosis.

Clinical picture

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

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

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

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

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

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

ECG: evidence of right ventricle and right atrium hypertrophy - dextrogram, “P pulmonale” – high (>2,5 mm) acute P in II, III, AVF and right chest leads V_{1-2}; high R wave ≥ 7 mm appears in V_{1-2}, deep S wave in V_{4-6}).

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

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

13.2.5. Syndromes of acute vascular insufficiency (syncope, collapse, shock)

Definition: ***Acute vascular insufficiency*** is acute condition that develops when a sudden decrease in circulating blood volume and deterioration of blood supply to vital organs as a result of falling vascular tone (infections, poisoning, etc.), blood loss, disorders of myocardial contractile function, etc.

Pathophysiological bases of this syndrome are a diminished volume of the blood and a diminished level of the vascular tone, and arterial and venous pressure as well. As a result, a significant part of the blood accumulates in

vessels of internal organs, and insufficient amount of the blood flows into heart, that leads to a decrease in minute blood volume and disorder of blood supply to all organs and tissues.

Causes:

(1) reflex disorders of blood vessels vasomotor innervation vessels due to irritation of serous membranes (trauma, inflammation), in myocardial infarction, embolism of the pulmonary artery, heart arrhythmias, etc;

(2) disordered vasomotor innervation of cerebral etiology (hypercapnia, acute hypoxia, psychogenic reactions);

(3) vascular paresis of toxic origin which occurs in many infections and toxicosis;

(4) diminished blood volume - loss of blood, dehydration of the body.

There are three basic clinical variants of the acute vascular insufficiency: *syncope, collapse, and shock.*

Syncope

Definition: ***Syncope (fainting)*** is a sudden and short-term manifestation of insufficient blood supply to the brain, represented in loss of consciousness and impaired sensitivity.

Predisposing factors are young asthenic people, mostly women; fatigue, excitation, fright, standing position in a non-ventilated room, decreased cardiac output, cardiac arrhythmias and conduction disorders, anemia, infectious diseases, intoxications, endocrine diseases (Addison's disease), neurologic diseases (cerebral atherosclerosis, diabetic neuropathy, syringomyelia, etc), intake of hypotensive medications (ß-blockers, including ophthalmic ß - blockers, Ca blockers, clonidine), and other drugs (digitalis), that may also cause bradyarrhythmias. Sometimes exertional (effort) syncope, swallowing syncope, postural syncope, and hyperventilation syncope may occur.

The most common pathophysiological basis for syncope is an acute decrease in cerebral blood flow (with resultant cerebral hypoxemia) secondary to decreased cardiac output. Syncope may be connected with the upset of the central nervous vasomotor innervation.

Clinical picture is characterized by abrupt, short-term and reversible state of unconsciousness and impaired sensitivity particularly when a patient suddenly changes from lying to the upright position (*orthostatic collapse*). The patient may feel a faintness, light-headedness, dizziness, confusion, or visual blurring during presyncope as the evidence of a mild to moderate reduction in cerebral blood flow. The objective examination of the patient reveals paleness of the skin and visible mucosa, cold sweat, arterial hypotension (typically > 60/40 mm Hg), decreased filling and |the tension of the pulse. Clinical manifestations of syncope last no more than 5-10 minutes and reverse spontaneously particularly at the horizontal position of the patient.

Collapse

Definition: Collapse is a sudden loss of effective blood flow due to cardiac and/or peripheral vascular factors that may reverse spontaneously or only with medical interventions.

Causes are vasodepressor syncope, transient severe bradycardia, myocardial infarction, embolism of the pulmonary artery or cardiac arrest, infections, toxicosis; diminished blood volume (loss of blood, dehydration of the body), endocrine diseases (Addison's disease).

Pathophysiology includes a decreased volume of circulating blood and reduced arterial pressure that cause ischemia of the brain and the internal organs.

Clinical picture: The patient feels a faintness, light-headedness, dizziness, confusion, or visual blurring. Syncope may be at the sudden onset of the collapse particularly when a patient suddenly changes his/her position from a horizontal to a vertical one. Objective examination of the patient reveals pallid skin, cold sweat, cold limbs, accelerated and superficial respiration, small and sometimes thready pulse, and drop of arterial pressure BP (typically < 60-70/40 mm Hg). Clinical manifestations of the collapse may last 5-10 minutes up to hours.

Shock

Definition: Shock is a cyclic clinical syndrome in which blood flow is inadequate to sustain life because of insufficient cardiac output or insufficient perfusion of the internal organs associated with arterial hypotension and oliguria.

Etiology and pathogenesis. Shock may be due to hypovolemia, vasodilation, or cardiogenic causes (poor cardiac output) or a combination. The fundamental defect in shock is a reduced perfusion of the vital tissues due (usually) to hypotension, so that oxygen delivery or uptake is inadequate for aerobic metabolism, resulting in a shift to anaerobic respiration with increased production and accumulation of lactic acid. When the shock persists, the impaired organ function is followed by irreversible cell damage and death. The degree of systemic hypotension necessary to cause a shock varies, and often it is related to a preexisting vascular disease.

Classification of shock

I. *According to etiology:*

- exogenous pain shock - traumatic (wound), electroconvulsive, burn shock;
- endogenous pain shock – cardiogenic, nephrogenic, abdominal shock;
- exogenous-endogenous painless shock – anaphylactic, bacteriemic (septic, septicemic), hemorrhagic, hypovolemic (oligemic), posttransfusion, psychogenic, toxic shock.

II. *According to the stage:* erectile, torpid stage of shock.

III. *According to the degree of severity:* mild, moderate, severe shock.

Clinical picture

Symptoms and signs of the shock may be due to the shock itself or to the underlying disease process. A clinical picture of the shock depends on stage and degree of severity.

Erectile stage of the shock is characterized by a transient clinic of the psychomotor agitation, elevation of the blood stage, tachycardia, tachypnea in first minutes or hours immediately after trauma, burn, etc.

Torpid stage of the shock is characterized by mentation disorders; lethargy, confusion, and somnolence are common. The hands and feet are cold, moist, and often cyanotic and pale. Capillary filling time is prolonged, and, in extreme cases, a bluish reticular pattern may appear over large areas. The pulse is weak and rapid unless the heart block or terminal bradycardia is present; sometimes, only femoral or carotid pulses can be felt. Tachypnea and hyperventilation are present, but apnea may be a terminal event when the respiratory center fails due to inadequate cerebral perfusion. Blood pressure taken by the cuff tends to be low (<90 mm Hg systolic) or unobtainable, but direct measurement by intra-arterial cannula often gives significantly higher values. Pulmonary complications that often coexist or develop in patients with shock must not be overlooked. This stage may last hours up to days.

Mild shock is characterized by cool extremities, diaphoresis, collapsed veins, anxiety or stupor, body temperature subnormal, systolic BP - 90-100 mm Hg, diastolic BP $\geq$60 mm Hg.

Moderate shock is characterized by cool extremities, diaphoresis, collapsed veins, tachycardia, tachypnea, oliguria <30 ml/hour, delayed reflexes, lethargy, systolic BP - 80-90 mm Hg, diastolic BP – near to 50 mm Hg.

Severe shock is characterized by cool extremities, diaphoresis, collapsed veins, hemodynamic instability, marked tachycardia, hypotension - systolic - <80 mm Hg, diastolic BP - <30 mm Hg, olyguria or anuria, mental status deterioration (coma).

Prognosis. Untreated shock is usually fatal. Even when treated, mortality from cardiogenic shock after massive myocardial infarction and from septic shock is high. Prognosis depends on the cause, preexisting or complicating illness, time between the onset and diagnosis, and adequacy of the therapy.

13.2.6. Cardiogenic shock

Definition: This is a form of the shock caused by relative or absolute reduction in cardiac output due to the left ventricular failure.

Causes of the cardiogenic shock are myocardial ischemia or myocardial infarction, myocarditis, acute mitral or aortic regurgitation, ruptured interventricular septum, pulmonary embolism, tension pneumothorax, pericardial tamponade, atrial tumor or clot, severe tachycardia or bradycardia.

This severest clinical expression of the left ventricular failure complicates acute myocardial infarction in near 20% of cases. Risk factors for the development of the cardiogenic shock include an advanced age > 65, a

depressed left ventricle ejection fraction, a large infarction, and diabetes mellitus. Typically, patients who develop a cardiogenic shock have a severe multi-vessel coronary artery disease.

Clinical picture. Cardiogenic shock should be considered as a form of severe left ventricle failure. This syndrome is characterized by marked hypotension with systolic arterial pressure of <80 mmHg and a markedly reduced cardiac output in the face of an elevated left ventricle filling. Cardiogenic shock is characterized by mental confusion, diaphoresis, and cold extremities; tachycardia (gallop rhythm may occur); marked hypotension with systolic arterial pressure of <80 mm Hg, reduced urine output (oliguria or anuria), pulmonary congestion (cardiac asthma, pulmonary edema), has a mortality of > 65%. It is most often associated with massive anterior myocardial infarction and > 50% loss of left ventricle functioning myocardium.

13.3. Syndromes of the myocardium hypertrophy and dilatation of heart chambers (cardiomegaly)

Definition. ***Hypertrophy of myocardium*** is an increase in the muscle mass of the myocardium, which in most cases is compensatory and develops with an increased particular part of the heart (ventricles or atria).

Dilatation *(dilation)* is an enlargement of one or more heart chambers, which may be compensatory, developing with an increased load on myocardium (*tonogenic dilatation*), while in others it can serve as one of the signs of decompensation and a sharp decrease in myocardial contractility (*myogenic dilatation*).

Causes of myocardial hypertrophy:

1. Increase preload.
2. Increase afterload.
3. Idiopathic hypertrophy of the myocardium (hypertrophic cardiomyopathy)

Causes of dilatation:

1. Increased preload (*tonogenic dilatation*);
2. Increased afterload (*myogenic dilatation*);
3. Acute myocardial damage (infarction, myocarditis) / myogenic dilatation.

Increased preload (tonogenic dilatation): overload by "volume" causes development of eccentric hypertrophy, characterized by enlarged heart chamber without thickening its walls (in mitral insufficiency, aortic insufficiency, insufficiency of the tricuspid valve).

Increased afterload (myogenic dilatation): overload with "resistance" induces the development of concentric hypertrophy with thickening its walls without heart chambers enlargement (in aortic stenosis, mitral stenosis, arterial hypertension, atherosclerotic and postinfarction cardiosclerosis).

Clinical picture. Complaints are not specific and due to underlying diseases (Table 13-4). Patients complain on chest pain or pressure, dyspnea, weakness, fatigue, dizziness, fainting, often after physical loads.

Table 13-4. Syndromes of heart chambers hypertrophy

Syndrome	Right atrium hypertrophy	Right ventricle hypertrophy	Left atrium hypertrophy	Left ventricle hypertrophy
Causes	Chronic diseases of the lungs (COPD, diffuse pneumofibrosis); pulmonary hypertension; pulmonary and tricuspid valves diseases		Mitral valves stenosis and incompetence	Arterial hypertension, IHD, aortal valves disease, mitral valves incompetence
Complaints	Chest pain or pressure, dyspnea, weakness, fatigue, dizziness, fainting, edema on inferior extremities		Dyspnea, cough, arrhythmia, chest pain, fainting	Fatigue, chest pain, often after exercising; palpitations, dizziness
Survey of the heart area	Cardiac and epigastric beats, swelling jugular veins may be visible		High apical impulse may occur	Left shift of apical impulse
Palpation of the heart	Cardiac and epigastric beats, may be palpable		Diastolic thrill may be in mitral stenosis	Apical impulse – left shift, wide, high, resistant
Percussion of the heart	Lateral shift of relative dullness right border	Widening of absolute dullness borders	Mitral configuration, elevation of superior border	Lateral shift of left border, aortal configuration, widening of heart transverse length
Auscultation of the heart	Systolic and/or diastolic murmurs on xiphoid	Systolic murmur on base of xiphoid	Systolic and/ or diastolic murmurs on apex	Systolic murmur on apex
X-ray of the heart	Widening heart shadow to the right		Mitral configuration, smooth “waist of the heart”	Widening heart shadow to the left, aortal configuration
ECG	P-pulmonale – high >2.5 mm in II, III, aVF	$R \geq 6$ mm in V_{1-2}, deep S in V_{5-6}	P-mitrale - wide, splitted, in I, II, aVL; biphase or negative V_{1-2}	$R_{V5-6} > R_{V4,}$ deep S wave in V_{1-2}

Notes: COPD – chronic obstructive pulmonary disease, IHD – ischemic heart disease.

Inspection and palpation of heart area show the shift of the apical impulse to the left (in the left ventricle hypertrophy) and inferiorly (in the left ventricle dilatation). Cardiac impulse (in the IV- and V-th interspaces at the left edge of sternum) and epigastric beats are visible and palpable in the right ventricle hypertrophy and dilation.

Percussion detects widening the borders of relative dullness of the heart to the left and down (in the left ventricle hypertrophy and dilation), to the right (in the right atrium hypertrophy), and widening the borders of absolute dullness of the heart (in right ventricle hypertrophy and dilation). Aortic configuration of the heart is typical in the left ventricle hypertrophy and dilatation; and mitral configuration (with widening left contour of the relative heart dullness in III-d interspace) - in the left atrium hypertrophy and dilatation.

Auscultation finds S1 weakening and functional systolic murmur of relative insufficiency of atrioventricular valves at the apex (in left ventricle hypertrophy and dilatation) and at the base of xiphoid process (in right ventricle hypertrophy and dilatation). *Gallop rhythm* (more often - protodiastolic and mesodiastolic) appears in pronounced dilatation of ventricles. Organic systolic and diastolic murmurs may be auscultated at the apex and base of xiphoid process in case of atriums hypertrophy and dilatation due to atrioventricular valves diseases (see Chapter 10. Auscultation of heart. Section 10.7.2).

ECG detects hypertrophy of atriums by changes of the P wave: right atrium hypertrophy -high acute “P-pulmonale” >2,5 mm in II, III, aVF; left atrium hypertrophy - wide, splitted “P-mitral” in I, II, aVL, and biphase or negative P wave in V_{1-2} . Hypertrophy of the ventricles is detected by changes of the ventricular QRS complex: in right ventricle hypertrophy - $R \geq 6$ mm in V_{1-2}, deep S in V_{5-6}, and dextrogram (RIII> RII> RI); left ventricle hypertrophy - $R_{V5-6} > R_{V4}$, deep S wave in V_{1-2}; and levogram (RI> RII> RIII).

Chest X-ray shows widening of the heart shadow and changes of the heart configuration (aortal configuration – left ventricle hypertrophy and dilatation; mitral configuration – in the left atrium hypertrophy).

Echocardiography substantiates syndromes of the heart chambers hypertrophy and dilatation (cardiomegaly) by changes in thickness of ventricle and atrium walls and intersepta, and increasing volume of the heart chambers.

13.4. Clinical syndrome of the heart arrhythmia and conduction system disorders

Bradyarrhythmias *(arrhythmia if heart rate <60 per minute)* arise through abnormalities of intrinsic automatic behavior or conduction, including sinus bradycardia, sinoatrial and atrio-ventricular blocks.

Tachyarrhythmias *(arrhythmia if heart rate >100 per minute)* may arise by altered automaticity, reentry, or triggered automaticity, which have been

identified electrophysiologically but can rarely be differentiated clinically. Most clinically significant tachyarrhythmias are probably due to re-entry. Tachyarrhythmias include sinus tachycardia, extrasystoles, paroxysmal tachycardia, atrial (and ventricular) flatter and fibrillation.

Common clinical symptoms of arrhythmias and heart blocks

Palpitations (awareness of the heartbeat) due to an arrhythmia may be accompanied by weakness, dyspnea, or light-headedness. Atrial or ventricular extrasystoles are often described as skipped beats, whereas atrial fibrillation is identified as an irregularity. Supraventricular or ventricular tachycardia is most often perceived as being rapid and regular and of sudden onset and termination. The onset of atrial tachyarrhythmia is often followed by the need to urinate because of increased production of atrial natriuretic factor.

Light-headedness, dizziness, and episodes of syncope (fainting) are symptomes of II degree sino-atrial and atrioventricular blocks with periodical missing heart and pulse beats.

Hemodynamic disorders are manifested by dizziness, syncope, arterial hypotension, dyspnea, acute heart failure.

Changes of rate and regularity of cardiac rhythm are detected by pulse examination (Chapter 8. Subjective and objective examination of patients with diseases of cardiovascular system . Section 8.5), heart auscultation, and ECG (Chapter 11. Electrocardiography examination. Section 11.9-11.10).

Clinical manifestations of complete heart block:

bradycardia (30-40 per min), syncope, dizziness, acute heart failure**,** postural hypotension, and breathlessness; the "gun sound" of D.N. Strazhesko is a sharply amplified S1 (when complete atrioventricular block) arising periodically in the case of coincidence in time of the systole of atria and ventricles.

Morgagni - Adams - Stokes syndrome (MAS)

Morgagni - Adams - Stokes syndrome (MAS) is an attack of consciousness loss, accompanied by breathing disorders and convulsions, resulting from acute hypoxia of the brain, due to a sudden drop in cardiac output.

Causes: sinoatrial and atrioventricular block of II-III degree. Ventricular tachycardia or fibrillation, or severe bradycardias, or asystole also may cause these symptoms in the form of MAS.

Clinical picture of MAS: The patient turns pale, feels a sudden sharp dizziness, general weakness, and loses consciousness after a few seconds (due to ventricular asystole longer than 10 seconds).

In 10-30 seconds from epy start of the attack - loss of consciousness, tonic-clonic convulsions appear, while involuntary urination and, less often, defecation occur. In 30-60 seconds, spontaneous breathing (before breathing is

either irregular, or periodic with pauses) stops, and cyanosis and eyes pupils dilatation develop.

Pulse during the attack is not determined, or it is very rare. Blood pressure is not determined. Heart sounds are not listened to, or they are very rare and weak. ECG can register sinus bradycardia, sinoatrial and atrioventricular blockade of II-III degree, and asystole.

The attack can last from 10 seconds to 4-5 minutes. If it does not last long, it may not reach convulsions. After the restoration of the cardiac activity, which can be estimated by the appearance of the pulse and heart sounds, the patient almost immediately returns to consciousness

Diagnosis of arrhythmias and heart blocks

Whereas the history and physical examination should give a working diagnosis, the ECG remains the major diagnostic procedure. The standard 12-lead ECG is crucial for the characterization and diagnosis of the various sustained tachycardias. However, it provides only a brief sample of cardiac rhythm, particularly when recorded by simultaneous multichannel recorders.

Ambulatory ECG monitoring is the most powerful method of capturing arrhythmic events, and its value is enhanced by keeping a diary of associated symptoms. ECG recorders are of many types, eg, those that log a continuous 24 hours (Holter 24 h) or those activated by the patient or by automatic detection of an arrhythmic episode. Solid-state recorders can eliminate the vagaries of mechanical tape transport systems. Ambulatory ECG monitoring is less useful when arrhythmias are infrequent. Patients with suspected life-threatening rhythm disturbances should be hospitalized for monitoring to avoid a fatal out-of-hospital event.

13.5. Syndrome of heart valves disease

Definition: Syndrome of heart valves disease includes cardiac dysfunction due to morphological and/or functional changes in one or more of its valves accompanied by disorders of heart hemodynamics, hypertrophy and dilation of heart chambers and development of circulatory insufficiency.

Causes - congenital and acquired heart valves diseases. *Congenital heart valves* include defects in the interatrial and interventricular septa, *ductus arteriosus (Botallo's duct)* and patent *foramen ovale*. Congenital heart defects may often combine with communicated greater and lesser circulation systems and stenosis of the great vessels. Moreover, the valves (bicuspid, tricuspid, aortic, and pulmonary valves) may also have congenital defects.

Causes of *acquired heart valves diseases:* rheumatic endocarditis is the main cause of the acquired heart defects; in the second place - infective endocarditis and atherosclerosis; more rarely - syphilis, traumatic injuries. Inflammatory processes occurring in the valve cusps often end in their sclerosis: deformation and shortening.

According to morphology and hemodynamic changes two types of problems can disrupt blood flow through the valves: regurgitation or stenosis.

Regurgitation is also called as a heart valve insufficiency. Regurgitation occurs when a valve does not close properly, and blood leaks backward instead of moving in the proper one-way flow.

Stenosis (narrowing) occurs when the cusps of the heart valves do not open wide enough and only a small amount of blood can flow through the valve. Both stenosis and regurgitation can coexist (*combined heart valve disease*).

According to the localization of the affected heart valves - mitral, aortic, tricuspid, pulmonary artery, and multivalvular, or complex, (if two and sometimes three valves simultaneously) heart valve disease are distinguished.

According to the condition of the blood circulation – compensated and decompensated (characterized by circulatory insufficiency) heart valve diseases are distinguished.

Clinical picture includes

(1) symptoms and signs of a heart failure – decreased working ability, heart palpitations, dizziness, episodes of collapse, dyspnea, orthopnea, peripheral cyanosis, paleness may occur, weak heart sounds, accelerated low-wave pulse;

(2) percussion signs - widening of the relative and absolute dullness of the heart;

(3) auscultation signs – stable heart murmurs, tachycardia, and pathologic triple rhythm (*gallop rhythm, opening snap*) may occur.

ECG demonstrates the signs of the heart chambers hypertrophy (Fig. 12.9-12.12).

Echocardiography detects structural changes of the affected heart valves and pathologic turbulent blood flow.

*Phonocardiogram (*PCG*)* supports the data of heart auscultation. PCG may show specific changes in heart sounds, and the appearance of murmurs (see Chapter 12. Fig. 12-1 – 12-3).

Diagnosis of acquired heart valves disorders is based mainly on typical auscultative data (heart murmurs) confirmed by echocardiography, and in some cases by heart catheterization.

13.6. The key points on the theme “Basic Clinical Syndromes of the Cardiovascular System Diseases”

Acute coronary syndrome (ACS) is a set of clinical symptoms of myocardial ischemic pain attacks suspected acute myocardial infarction with elevation ST or without elevation ST: >10-15 minutes, as pressure, squeezing, or a burning sensation behind sternum, pain and may radiate to the neck, shoulder, jaw, back, upper abdomen, or either arm, and non-responsiveness to nitroglycerin. Diagnosis of acute coronary syndrome is permitted to use not more than 24-48 hours after the onset of clinical picture. For differentiation

unstable angina pectoris and acute myocardial infarction, serum cardiac markers CK-MB and/or cardiac-specific troponins (Troponin-T and Troponin-I) need be tested in all cases of acute coronary syndrome.

Heart failure (HF) is a syndrome of ventricular dysfunction. Left ventricular failure leads to congestion in lesser circulation, development of dyspnea, cardiac asthma and pulmonary edema. Right ventricular failure leads to congestion in greater circulation, peripheral edema, acrocyanosis, hepatomegaly and accumulation of fluid in abdominal cavity. The process may involve either both ventricles, or each ventricle separately. There is a combination of heart and vascular insufficiency in most cases of cases. The diagnosis is established clinically and confirmed by the data of chest radiography, echocardiography and levels of Brain Natriuretic Peptide (BNP) in blood plasma.

Vascular insufficiency is a clinical syndrome as a result of fall in circulating blood volume and /or violations of vascular tone. It is manifested by pronounced decrease in blood pressure and filling arterial pulse, and dysfunction of vital organs (cerebral brain, heart, lungs, and kidney).

Syndromes of myocardium hypertrophy and dilatation of heart chambers (cardiomegaly) are diagnosed by widening borders of relative and absolute dullness of the heart, corresponding to the data of ECG and echocardiography. In dilatation of heart chambers, expressed cardiomegaly and heart failure develop, with auscultation, changes of the heart sounds and murmurs are determined, ECG-signs of pathological rhythm and conduction disorders may be.

Arrhythmias are clinically manifested usually by irregular pulse and arrhythmic heart sounds, confirmed by ECG data.

Heart valves disorders are diagnosed on typical auscultative data (heart murmurs, pathological heart sounds) combined with signs of hypertrophy and dilatation of the heart chambers, and confirmed by echocardiography.

13.7. Assessment tests on the theme “Basic Clinical Syndromes of the Cardiovascular System Diseases”

1. It is characterized for patients with acute coronary syndrome:

1. pressing pain in the region of the heart lasts more than 10-15 minutes;
2. pain occurs against the background of the exercise stress;
3. pain occurs at rest;
4. pain may radiate to the neck, left shoulder, jaw, back, upper abdomen;
5. pain is effectively controlled by sublingual Nitroglicerinum.

2. It is typical of the clinic of an acute coronary syndrome:

1. pressing pain behind the sternum lasts more than 30 minutes;
2. pain is not stopped by sublingual reception of Nitroglycerinum;
3. fall of arterial blood pressure and loss of consciousness;

4. pressing pain behind the sternum lasts not more than 10 minutes;
5. pain is not stopped by sublingual reception of Nitroglycerinum.

3. What biochemical blood tests are important in acute coronary syndrome?

1. creatinphosphokinase-MB fraction (CK-MB);
2. LDG_1;
3. Troponins T and I;
4. alkaline phosphatase (AP);
5. AsAT.

4. What variants of ECG patterns are typical in acute coronary syndrome?

1. transient ST depressions;
2. transient ST elevation;
3. periodical missing P waves;
4. hyperacute T-waves;
5. pathologic Q-wave with ST-elevations.

5. What are characteristics of Functional classes of a chronic heart failure according to classification of NYHA (New York Heart Association)?

1. Class I - no limitation of physical activity, no symptoms with ordinary exertion;
2. Class II - slight limitation of physical activity, ordinary activity causes symptoms;
3. Class III - marked limitation of physical activity, less than ordinary activity causes symptoms, asymptomatic at rest;
4. Class III – persistent clinical signs;
5. Class IV - inability to carry out any physical activity without discomfort, symptoms at rest.

6. What are characteristics of the chronic heart failure stages according to the classification of *N.D. Strazhesko* and *V.K. Vasilenko*?

1. HI – initial latent circulatory insufficiency, clinical signs – only during serious physical exercises, morphology changes absent;
2. HIIA - decompensated circulatory insufficiency, clinical signs – only during light physical exercise, morphology changes - partly reversible;
3. HIIB - compensated pronounced circulatory insufficiency, mild hemodynamic disorders, clinical signs - during physical exercises and partly at rest; morphology changes - reversible;
4. HIII – terminal dystrophic stage of circulatory insufficiency, grave hemodynamic and metabolic disorders; morphology changes – irreversible.

7. Clinical manifestations of chronic heart failure:
1. mixed dyspnea increases during exercises, and in the recumbent position;
2. cardiac asthma;
3. hepatomegaly;
4. peripheral cyanosis;
5. edemas at morning, mostly on the face.

8. Syndrome of the right ventricle failure characteristics:
1. causes - chronic diseases of the lungs, tricuspid and pulmonary valves diseases;
2. causes - arterial hypertension, ischemic heart disease, aortal and mitral valves diseases;
3. peripheral edema;
4. cardiac asthma;
5. hepatomegaly.

9. Syndrome of the left ventricle failure characteristics:
1. dyspnea (worse in a horizontal position);
2. cardiac asthma;
3. cough;
4. orthopnea;
5. peripheral edema.

10. Clinical variants of acute left ventricle failure:
1. syncope;
2. collapse;
3. cardiogenic shock;
4. pulmonary edema;
5. cardiac asthma.

11. Pulmonary heart (cor pulmonale) characteristics:
1. left ventricle hypertrophy;
2. right ventricle hypertrophy;
3. widening relative heart dullness to the right;
4. accent of II sound at the II-d interspace on the left edge of the sternum;
5. P-pulmonale.

12. Cardiogenic shock characteristics:
1. systolic arterial pressure of <80 mm Hg;
2. tachycardia;
3. oliguria or anuria;
4. pulmonary edema;

5. mental confusion.

13. What are typical of the right atrium hypertrophy?
1. right shift of the apical impulse;
2. lateral shift of relative heart dullness right border;
3. widening absolute heart dullness;
4. trapezoidal configuration;
5. P-pulmonale.

14. What are typical of the left atrium hypertrophy?
1. left shift of the apical impulse;
2. widening left contour of relative heart dullness;
3. lateral shift of relative heart dullness left border;
4. mitral configuration;
5. P-mitrale.

15. What are typical of the right ventricle hypertrophy?
1. right shift of the apical impulse;
2. cardiac impulse;
3. trapezoidal configuration;
4. widening absolute heart dullness ;
5. RIII> RII> RI

16. What are typical of the left ventricle hypertrophy?
1. left shift of the apical impulse;
2. expressed cardiac and epigastric beats;
3. aortal configuration;
4. widening absolute heart dullness;
5. $R_{V5-6} > R_{V4}$.

17. What are characteristics of Morgagni-Adams-Stokes syndrome?
1. bradycardia (30-40 per min);
2. loss of consciousness;
3. epileptiform convulsions;
4. “gun sound” according to D.N. Strazhesko;
5. atrioventricuar block of III degree.

18. What are typical of the heart valves disease syndrome?
1. widening borders of relative and absolute dullness of the heart;
2. stable heart murmurs;
3. signs of heart failure;
4. ECG-signs of heart chambers hypertrophy;
5. abnormalities of heart rhythm.

Appendix

Answers to the test questions for supervised students' individual work

Standards of test answers on the theme "Subjective Examination (Inquiry) and its Role in making the Diagnosis" Chapter 1

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3,4	**13**	1,2
2	1,2,3,4	**14**	1,2,3,4,5
3	1,2,3	**15**	1,2,3,4
4	1,2,3,4	**16**	1,2
5	1	**17**	1,2,3,4
6	1,2,3	**18**	1,2,3
7	1	**19**	1,2,3,4
8	1,2	**20**	1,2
9	1,2	**21**	1,2,3,4,5
10	1,2	**22**	5
11	1	**23**	1,2,3,4,5
12	1,2,3,4,5,6	**24**	5

Standards of test answers on the theme "Objective Examination of a Patient. General Inspection (Survey)" Chapter 2

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2	**13**	1,2,3,4
2	1,2,3,4	**14**	1
3	1,2,3,4	**15**	1,2,3
4	1	**16**	1
5	1,2,3	**17**	1
6	1,2	**18**	1,2,3
7	1,2,3,4,5	**19**	1,2,3,4,5
8	1,2,3,4	**20**	1,2,3,4,5
9	1,2,3,4,5	**21**	1,2,4
10	1,2,3,4	**22**	1,3,5
11	1,2,3	**23**	1,2,3,4,5
12	1,2,3,4		

Standards of test answers on the theme "Subjective and Objective Examination of Patients with Respiratory System Diseases"
Chapter 3

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3	**14**	1,2,3
2	1	**15**	1,2,3
3	1,2,3,4	**16**	1,2,3,4,5
4	1,2	**17**	1,2,3
5	1	**18**	1,2,3
6	1	**19**	1,2
7	1	**20**	1,2,3
8	1	**21**	1,2,3
9	1	**22**	1,2,3,4
10	1,2	**23**	1,2,3,4
11	1,2,3	**24**	1,2,3,4,5
12	1,2,3,4,5	**25**	2,4,5
13	1,2,3		

Standards of test answers on the theme "Percussion of the Lungs"
Chapter 4

Number of question	Variant of answers	Number of question	Variant of answers
1	1	**11**	2
2	1	**12**	4
3	1,2,3,4	**13**	1
4	1,2,3	**14**	3
5	1,2	**15**	1
6	1,2,3	**16**	4
7	1,2	**17**	1
8	1,2,3,4	**18**	1,2,3
9	1,2	**19**	1
10	1	**20**	1,2,3,4,5

Standards of test answers on the theme "Auscultation of the Lungs" Chapter 5

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2	**13**	1
2	1,2,3	**14**	1,2,3
3	1,2	**15**	2,5
4	1	**16**	5
5	1	**17**	1,2,3
6	1,2	**18**	2,3,5
7	1,2,3,4	**19**	3,4,6
8	1,2,3	**20**	1,2,4,5
9	1,2,3	**21**	1,3,5
10	1	**22**	2,3,5
11	1	**23**	2,4
12	1,2,3,4		

Standards of test answers on the theme "Laboratory-Instrumental Examination of the Respiratory System" Chapter 6

Number of question	Variant of answers	Number of question	Variant of answers
1	1,3,4,5	**14**	1,2,3,4
2	1,3,4,5	**15**	1
3	1,4,5	**16**	1
4	2,3,5	**17**	1,2,3
5	2,3,6	**18**	1,2,3
6	1,2,5	**19**	1,2,3,4,5
7	1,2,3	**20**	1,3,5
8	2,3,4,5	**21**	2,4,5
9	1,2,3	**22**	2,3,4,5
10	1,2	**23**	1
11	1,3,4	**24**	2
12	2,4	**25**	1,2,3
13	1,4,5		

Standards of test answers on the theme "Basic Clinical Syndromes of the Respiratory System Diseases" Chapter 7

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3	**11**	1
2	1,2	**12**	1,2,3,4
3	1,2,3,4	**13**	1,2
4	1	**14**	1,2,3
5	1	**15**	1,2
6	1,2	**16**	1,2,3
7	1,2	**17**	1,2
8	1	**18**	1
9	1,2,3	**19**	1,2,3
10	1,2,3,4,5	**20**	1,3,5

Standards of test answers on the theme "Subjective and Objective Examination of Patients with Diseases of the Cardiovascular System" Chapter 8

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3	**11**	1
2	1	**12**	1,2,3,4
3	1,2,3,4,5	**13**	1,2,3
4	1	**14**	1
5	1	**15**	3
6	3	**16**	1
7	3,4	**17**	5
8	1,5	**18**	1,2,3,4
9	2	**19**	2
10	1	**20**	1,3

Standards of test answers on the theme "Percussion of the Heart" Chapter 9

Number of question	Variant of answers	Number of question	Variant of answers
1	1	**11**	2
2	2	**12**	1,2,3,4
3	5	**13**	3,4,5
4	2	**14**	3,5
5	4	**15**	1,2,4

6	1,2,3,4	**16**	1,3
7	1,2,4	**17**	4
8	1,2,3	**18**	4
9	1,2,5	**19**	3
10	1,2,3,4	**20**	2,3,5

Standards of test answers on the theme "Auscultation of the Heart"

Chapter 10

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3	**12**	1,2,3
2	1,2	**13**	1,2,3,4
3	1,2,3	**14**	1
4	5	**15**	2
5	1,4,5	**16**	2,3,4,5
6	1,4,5	**17**	1,2,3,5
7	2,3	**18**	1,2,3,4,5
8	1,2	**19**	1,2,3,4
9	2,5	**20**	1,2,4
10	1,2,3,6	**21**	2,3,4,5
11	1,2,3,4,5	**22**	1,3

Standards of test answers on the theme "Electrocardiography"

Chapter 11

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3	**16**	1,2,4
2	1,2,3	**17**	2,3,4
3	1,2,3,4	**18**	2,3,4
4	1,3,4	**19**	1,2
5	3	**20**	1,2
6	3,4	**21**	5
7	2,3	**22**	3
8	1,3	**23**	1,2,3,4,5
9	2	**24**	1,2,3,4,5
10	2	**25**	1,2,3,4
11	3	**26**	1,2,3
12	1	**27**	1,2,4,5
13	2,3		
14	1,2,3		
15	2,3,4		

Standards of test answers on the theme "Laboratory-instrumental methods of circulatory system examination" Chapter 12

Number of question	Variant of answers	Number of question	Variant of answers
1	1,4	**8**	2,3,5
2	2,4,5	**9**	1,2,3,5
3	1,3,4	**10**	1,2,5
4	1,3,5	**11**	1,2
5	3,4,5	**12**	2,3,5
6	1,2,3,4,5	**13**	3
7	1,2,3,4,5	**14**	1,2,3,4

Standards of test answers on the theme "Basic Clinical Syndromes of the Cardiovascular System Diseases" Chapter 13

Number of question	Variant of answers	Number of question	Variant of answers
1	1,2,3,4	**10**	3,4,5
2	1,2,3,5	**11**	2,3,4,5
3	1,2,3	**12**	1,2,3,4,5
4	1,2,4,5	**13**	2,5
5	1,2,4,5	**14**	2,4,5
6	1,4	**15**	2,4,5
7	1,2,3,4	**16**	1,3,5
8	1,3,5	**17**	1,2,3,4,5
9	1,2,3,4	**18**	1,2,3,4,5

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