R. N. Yuldashev

PROPEDEUTICS OF INTERNAL DISEASES

Andizhan - 2022

This book has been adopted as a textbook for undergraduate students of medical universities by Ministry of Higher and Secondary special Education and recommended for publication.

Textbook covers basic principles of internal diseases by discipline of Propedeutics of internal diseases. Regarded chosen for medical examination of respiratory, cardiovascular, gastrointestinal, urogenital and connective tissue system to reviele pathological changes of them in it.

This book expound inquiring, inspection, palpation, percussion, auscultation of these systems with giving initial information about classification, etiology, pathogenesis, clinical picture, diagnosis and differential treatment of selected nosology.

Revealed themes of internal diseases give students opportunities orientating on studied theme of Propedeutics of internal diseases correctly and easy. Educational book is proposed for teaching students of medical institutes in discipline of Propedeutics of internal diseases.

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Propedeutics of internal diseases : Syndromes and diseases; *textbook for English learning Students of higher medical institutions ;*

R. N. Yuldashev, – Andizhan: 2022

The textbook focuses on syndromes and diseases of respiratory, cardiovascular, digestive, kidneys and urinary, blood and endocrine system organs.

The textbook is intended for medical students, intern – doctors and doctors of general practice.

R. N. Yuldashev

Introduction

This subject was written for 2 nd and 3rd year students who studies at the medical institutions.

The subject of propaedeuties of internal diseases studies the causes of different diseases of internel organs, the development of the disease, it's manifastation, course, diagnosis, treatment and the methods of prevention.

The subject of propaedeuties of internal diseases considers the respiratory system, cardiovascular system, gaster, intestine, gall-bladder, kidneys, the urinary system, the diseases of joints and conjuctive tissue.

This Subject is being studied at the 3 therapeutic chairs of medical institutes.

To write this book we have used books of O.Kovalyova and E. Kosimov.

Of course this book can have some short comings. That's why we will take your opinions and thank you for them.

The servies of the world scientists and Central Asia in developing of the studing of propaedeutics of internal diseases.

The studiing of the internal diseases has a long history. It's founder is the greek physician Gippokrat who lived in the 5th century B.C.

Gippokrat paid attention to the learning of behaviour of the patient, looking after attentivly, questioning in order, examination the organs. Body skin and the state of mucous membrane. Because in that period to diagnose different diseases the doctor could only rely on his sensitive organs.

Gippokrat by saying "Where the human is loved there the medicine is respected" placed the human factor to the 1st stage.

Another physician of ancient period Galen (Jalinus) supplemented the Gippokrats studies with his words like: "The nidus is very important for developing diseases".

At the end of the XVIII century were entering the new methods of diagnose and treatment. For example in the XVIII centure Selsiy created the thermometer that can measure the temperature. Auenbrugger offered to diagnose diseases by percussion, the Franch doctor Laenneck inculcate in practice the method of auscultation.

Also was created the instrument that can measure the blood pressure.

At the end of the XIX centure were founded the x-ray examination. By it we can establish a lot of pathologic processes in the human organism. Subsequently we began to use ultrasound examinations that can help to diagnose the heart, lungs, digestive system and brain diseases.

We mention Asian and world famous scientists whose deeds were valuable for the development of medicine.

In Central Asia the medicine has own long history. It was developing gradually during years, century and got the status of

scientific medicine in 900 years. In that moment were developing many famous doctors and scientists in Central Asia.

One of them is Abu Bakr ar-Roziy. He was born and grown in the city of Ray. Iran and some period lived in Bukhara. He from his childhood was interested in literature, art, mathematics, astronomy, chemistry and philosophy.

To medicine he interested when he was 30. But quickly he became a famous doctor. And he knew the books of Gippokrat, Galen, Karneliya and Sels.

Abu Bakr ar – Roziy wrote about medicine-56, nature-33, logic-7, mathematics and astronomy – 10 and a lot of other books.

The Ar-Roziy's book named "Case history" deserved a great attention and praise. Ar-Roziy considered that the function of medicine not only treatment of the diseases and prophylaxis too. He gave attention to the preventive measures from that period. Ar-Roziy knew surgery, pediatries, gerontology,otoloryngology, ophtalmology and neurological diseases. That's why we can fully call him a doctor of general practice. Ar-Roziy died in the city of Ray in 925.

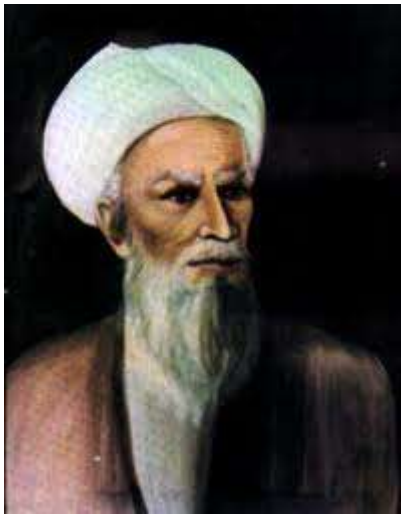
Abu Ali ibn Sino is the pride of Central Asia and one of the greatest scientists. Besides medicine he was occupied with mathematics, logic and philosophy. He was bornin bukhara in the village of Afshana in 980 and got his education in Bukhara.

Because of his perfect memory and quick wits he obtained a lot of knowledge very quickly. He had already learnt the Koran by heart when he was 10. Later, when he gaining new knowledge, curing people finding the reasons of many diseases writing works on medicine and bringing this knowledge to upcoming generation.

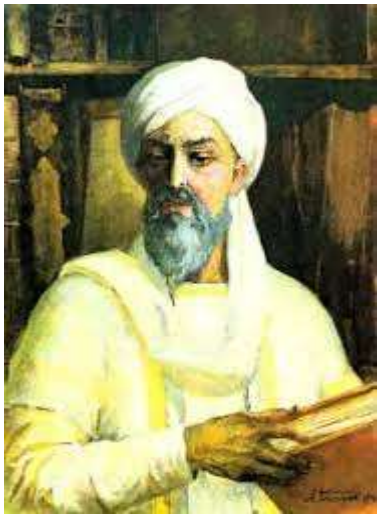
Abu Ali ibn Sino traveled to many cities as a visiter and died from serious infectious disease on June 18, 1037 in the city of Isfakhan. The number of works that Abu Ali ibn Sino had written exceeds 450, but only 160 out of them reached our hands. One of his main books is "Konun at-Tib". This works consists of 5 big parts containing the reason of diseases, hygiene, medication and a lot of other information related to medicine. His other books such as

“Shifo kitobi”, “Insof kitobi”, “Hojat kitobi”, “Donishnoma”, “Arab tili kitobi”, “Metafizika” had played the main role in the development of many sciences.

Abu Ali ibn Sino is well –known in Europe by the name Avicenna. The naturalist Karl Linney named a type of plant “Avicenna” in honour of him. To sum up we can say that Abu Ali ibn Sino was an encyclopedic scholar whose contribution to world civilization was incomparable.



Abu Bakr ar – Roziy



Abu Ali ibn Sino

Matvey Yakovlevich Mudrov (1776-1831). He is one of the greatest scientist of XIX centure, professor of moscow University. He firstly put the clinical examination in order and used it in practice. He paid attention not only to the drugs; but and to widely using natural things and keepong to a diet, advised to treat diseases according to the condition of the patient, insisted the importance of psychotheraphy. Matvey Yakovlevich Mudrov died during struggle against plague in Peterburg.

Sergey Petrovich Botkin was a professor of military – medical Academy in Peterburg. The developing of the physiological way in medicine connected with his name the fully described the infectious disease –icberus, that’s why now we call it by his name.

Except that he found the place where the pain can spread in gall-stone disease. He paid attention to the mechanism of drugcs to the organism. He considered that women are more needed to the

medical institutions. That's why to prepare medical workers he created the society named "Valorous nurses". Nowadays the biggest hospitals in Moscow and Peterburg are named by his name.

Matvey Yakovlevich Mudrov

Sergey Petrovich Botkin

Umidova Zulfiya Ibragimova was born in 1896. In 1922 she graduated the medical faculty of the Tashkent State University. In 1938 she achieved the status of candidate of medical sciences and in 1946 defended a thesis for a Doctor's degree. Her scientific works were dedicated to a cardiovascular diseases in hot climate.

Umidova Zulfiya Ibragimova was an author of 3 monographies and more than 80 scientific articles. Under her quidance prepared 3 doctors of Philosophy and about 30 candidates of science.

Ismoilov Nosir Ismoilovich. In 1929 he graduated a medical faculty of the Central Asia State University. In 1935 he achieved the status of candidate of medical scienses and in 1947 defended a thesis for a Doctor's degree. In 1942 -1971 years he worked as a head of the chair of propaedeuties of internal diseases at the Tashkent State Medical Institute.

He is a Honoured Science worker in Uzbekistan. He is an author of 3 monographies and more then 100 scientific articles. Under his quidance were prepared 3 doctors of Philosophy and 16 candidates of science.

Askarov Akbar Askarovich was one of the first students of medical faculty of the Central Asia State University. In 1947 he defended a thesis for a Doctor's degree named "Ulceraus colitis in Uzbekistan".

He fully learned aetiology, pathogenesis and clinics of the gastro –intestinal diseases. He devised the methods of treatment of the gastro-intestinal diseases by diet.

We can call him a father of a clinic gastroenterology in Uzbekistan. He is an author of 10- monographies, more than 200 scientific articles, Latin- Lizhek normal anatomy and Russian – Uzbek medical dictionaries.

Askarov Akbar Askarovich was one of the author who translated the Abu Ali ibn Sino's book "Kitob at-tib". Under his quidance were prepared 10 doctors of philosophy and 36 candidates of science.

Tursunov Sayfiddin Yunusovich (1939. 28.02) a therapeutist, scientific statesman of Uzbekistan, Doctor of Medical Sciences (1981y), professor (1983y). He graduated the ASMI in 1962. In 1962-1964 years he was intern of the chair of the prapaedeuties of internal diseases, in 1964-1968years an assistant, in 1968 -1970 years head of the course, in 1975-1978 years head of the chair. His scientific work was on the studiing the causes of cardio – vaskular diseases and preventing them.

Xujamberdiev Mamazoir Axmedovich –doctor of Medical Sciences, professor. He has 10 textbooks, 14 monographs, 6 handbooks, 600 articles and more than 200 other publishes. He had prepared 6 doctors and 12 candidate of medical sciences on speciality of the therapy and cardiology. He is an initiator of the introduction

of new techniques.

Kovalyova Olga Nikolaevna – Doctor of Medical Sciences, Professor Head of the chair prapaedeuties of internal diseases of Kharkov National Medical Academy of Ukraine.

Z.U.Umidova (Zulfiya Ibragimovna Umidova) She devoted her life for developing the medicine of Uzbekistan. She created great inventions with famous scientists like A. N.Krukov, N.U.Ragoza, M.I.Slonim and I.A. Kassirskiy.

Saidjalol Bahromov - doctor of medical science, professor, Honoured academic of the Uzbekistan and Russian Fedederation. He is the founder of haematology and transfusiology in Uzbekistan.

Abdulla Ubaydullayev - doctor of medical science, professor, Honoured academic of the Uzbekistan. He was a head of the scientific research institute of pulmonology and phthisiology in Uzbekistan. He was the famous scientist.

Mamasoliev Nematjon Solievich, MD, professor, academician of the Academy of Sciences of the Republic of Uzbekistan, head of the Department of Advanced Training and Retraining of Doctors of Andijan State Medical Institute. He is the author of more than 3500 published books, textbooks, educational manuals, monographs and many scientific articles during his 48-year career. He prepared and trained more than 10 doctors of science, more than 60 candidates of science.

Salokhiddinov Zukhriddin Salokhiddinovich, MD, professor, head of the Department of Training of Family Doctors No.1. He is the author of more than 1000 scientific publications. He prepared and trained a number of doctors of science and about 20 candidates of science.

GENERAL PART

Chapter 1. INTERNAL MEDICINE

GENERAL CONCEPTS

Internal medicine is the major and one of the most important parts of medical practice. It is the science dealing with diseases of the internal organs, which are the most common. The study of the internal disease is leading subject of medical training.

The World Health Organization (WHO) idealistically defined health in 1946 as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity". This is a definition for a conception of health that literally encompasses every element of human happiness.

On this basis, health for person means being able to function as a member of society, to create material values, as well as freedom from physical ailments, loving and being loved, finding meaning and purpose in life leading to a sense of hope and joy, forgiving and accepting forgiveness

The most important and essential aim of practical medicine is the prevention and treatment of disease, and the alleviation of the patient's suffering. If we assume that the first aim of a physician is to reveal the disease we must than define what a disease is.

What causes a disease, and what is actually a disease?

What must we investigate and learn? We all know in general terms what a disease is, but this does not mean that the disease has been understood. The general concept of disease, its essence and genesis, have undergone significant changes during the progress of medicine.

Health and disease are opposite yet interdependent forms of the organism's functioning in the physical and social environment.

Disease is not only an anatomical or functional disorder caused by a pathogenic or an extraordinary stimulus; neither is it their total; it is rather a whole set of changes which arise from affections. Changes in the body in response to stimuli or damage are considered as a reaction. This is manifested by functional and morphological processes.

The first essential sign of a disease is damage caused to the organism (damage to its integrity, its "breakdown", structural damage, functional disorder, the absence of enzymes or other biologically active substances, insufficient homeostasis and etc.)

Another important sign of a disease is the body's response to various affections.

Disease is a response of the body to its damage. It is understood that the entire body responds to damage; a local affection causes a response in the entire body, since the response is the complicated result of activity of many system of the living body.

It should be remembered that disease is a general response of the body, which is regulated by the nervous and humoral systems. In every disease the entire body is involved. For example the main site of affection in pneumonia is the lung, but the cardiovascular system and many other organs of the body are involved simultaneously.

The following causes of disease are distinguished:

1) mechanical (closed and open injuries, concussion etc.);
2) physical (high or low temperature, electric current, light);
3) chemical (spoiled foods, poisons);
4) biological (microorganisms);
5) psychogenic;
6) genetic (hereditary).

Most diseases develop as a result of the combined action of many factors. Congenital and acquired specific features and properties of the body are important in etiology of disease. Sometimes a disease develops suddenly and lasts a comparatively short time. Such diseases are called acute. Chronic diseases are characterized by prolonged course and may exacerbate periodically.

Thus the health cannot be defined apart from its opposite – disease, and disease affect all dimensions of the person. It includes the physical, psychosocial and spiritual dimensions of the person.

Definition of disease is based on the initial and most general signs; these signs are of major importance for us. Needless to say that disease in man is not only a biological but also a social phenomenon; there is not only a somatic, but also a psychic suffering.

BASIS OF CLINICAL DIAGNOSTICS

Successful treatment becomes only possible with correct diagnosis, identification of the cause of the disease, specific signs and course of the disease.

Propedeutics (Gk *propos* – introduction) **to internal diseases** is introduction to internal medicine, the science about methods of clinical examination of the patient and diagnosis basing. The course of propedeutics to internal diseases includes diagnostics, semiology or symptomatology, and medical deontology.

Diagnostics (Gk *dia* – through, *gnosis* – knowledge) is the science of methods by which disease is identified. Diagnostics consists of clinical examination of the patient and diagnosis basing (explanation) (Fig. 1.1).

The result of clinical examination is the diagnosis of the disease. **Diagnosis** is a short conclusion of a doctor about the essence of the disease and the patient condition, expressed in terms of modern medical science, and resulted from the examination of the patient.

The following types of the diagnosis are distinguished.

Preliminary diagnosis is based on the patient's present complaints, the history of the present disease (anamnesis morbi), the past history (anamnesis

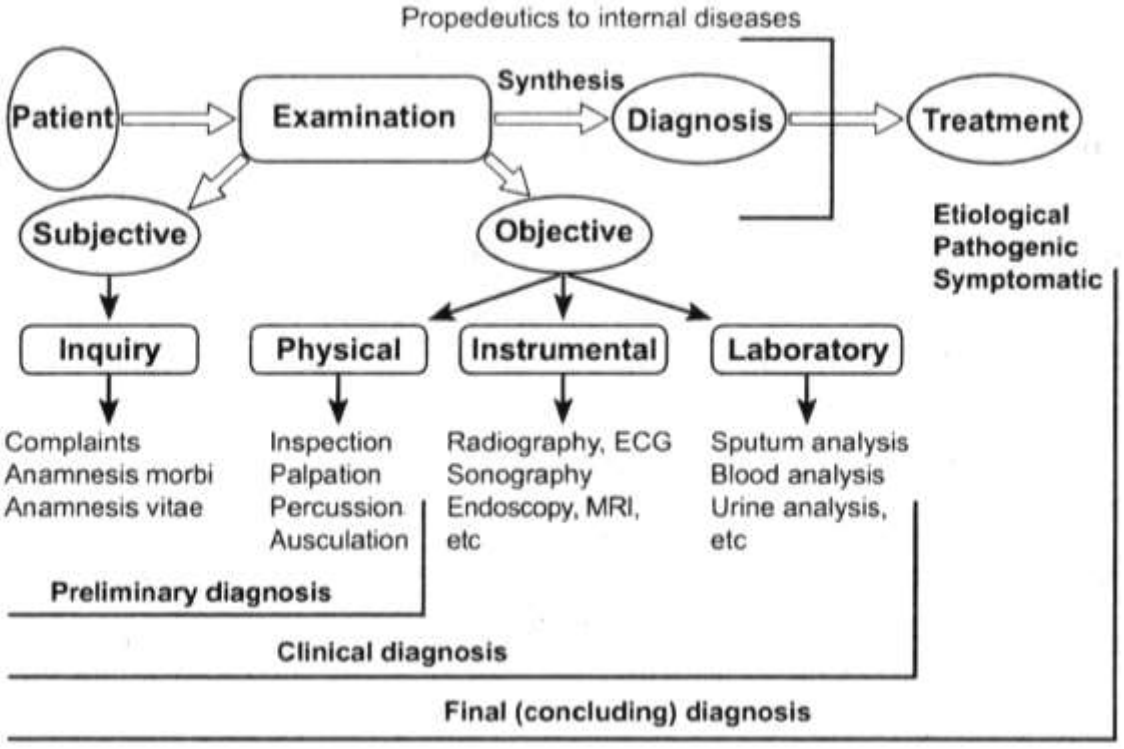


Fig. 1.1. The algorithm of a clinical examination.

vitae), and physical examination data – inspection, palpation, percussion, and auscultation.

Clinical diagnosis is based on the subjective examination of the patient (inquiry: complaints, anamnesis morbi and vitae), and objective examination: physical, instrumental, and laboratory.

Final diagnosis is based on the subjective, objective examination, and treatment.

Diagnosis has the following structure:

1. The main disease,
2. Complication of the main disease,
3. Concomitant (concurrent) disease.

Semiology *(*Gk *semion* – sign*)* ***or symptomatology.*** In order to diagnose correct it is necessary to determine the signs of the disease and pathological changes in the patient's organism.

A healthy individual does not feel any unpleasant sensations. Such abnormal phenomena as pain, dizziness, nausea, vomiting, etc, occurring in sick persons are called **signs** or **symptoms** of the disease (Gk *symptoma* – that which happens).

Subjective and objective symptoms are differentiated.

Subjective symptoms are those that cannot be found on examination of the patient – pain, dizziness, nausea, etc. These are subjective sensation of the patient, which reflects however objective changes in the organism.

Objective symptoms are those that can be found on examination of the patient – cyanosis, jaundice, enlarged internal organs, tachycardia, etc.

The symptoms are divided into *pathological symptoms* (pain, elevated temperature) and *compensatory symptoms* (tachycardia, tachypnoea, hypertrophy).

According significance symptoms can be *specific* or *pathognomic* ('triple rhythm' in mitral stenosis, crepitation – typical sign of the pulmonary tissue damage), *nonspecific* (headache, fever, nausea), and *relative* (diastolic murmur in mitral stenosis or aortic regurgitation).

According to time symptoms are *early* (pain in the heart in myocardial infarction, elevated temperature in pneumonia) or *late* (pleural friction sound, elevated temperature in myocardial infarction).

According to prognosis symptoms are subdivided into *favorable* (pleural friction sound in effusive pleurisy) and *unfavorable* (gallop rhythm in myocardial infarction).

Syndrome is defined as combination of symptoms that are interrelated and give rise to one another.

Syndromes can be as independent disease: Cushing, Reino, WPW (Wolf-Parkinson-White), CLC (Clerk-Levy-Critesko), etc, or as a part of disease: in peptic ulcer disease – painful, dyspeptic syndrome can occur, in hypertension – asthenic, cardiac syndrome.

Symptomocomlex is defined as symptoms that are not interrelated and do not give rise to one another.

DEONTOLOGY

Members of the medical professions are repeatedly faced by ethical dilemmas in the course of their normal working lives. Although ethics has always been viewed as a branch of philosophy, its all-embracing practical nature links it with many other areas of study, including biology, economic, history, politics, and sociology. But now ethics often refer to judgments and principles with an extended and intensive opportunity to review and update theirs approach to the analysis of key "medico-moral" issues. Practicing professionals at different stage in their career deal with moral questions at all levels. Physicians have always made ethical decisions in their day-to-day practice of medicine. The technical advances that have enabled the medical sciences expand diagnostic skills proliferate treatment alternatives, and aid to prolongation of physical life have also made ethical decision problems making in medicine more complex by creating moral dilemmas. Moral philosophy should become the basic science for the art of medicine.

Deontology is a Greek origin derivative from word: *deon* – duty and *logos* – science. Deontological ethics – is theories that place special emphasis on the relationship between duty and the morality of human actions. Deontology consequently focuses on logic and ethics. No attempt is made in such theories to explicate specific moral obligations.

The simple implication of medical deontology merely means – the science of the duties and rights of the doctor in relation to his patients.

Deontology relevant to the medicine may also be defined as the set of rules and principles of medical ethics.

The concepts of morals and professional duties of practitioners have changed during the centuries, depending on the social, economic and class relationships, the political structure of the state, the level of civilization, national culture, religious traditions, and many other factors.

The Greek medical schools greatly contributed to the development of the deontology.

Hyppocrates (b. c. 460 BC, island of Cos, Greece – d. c. 377, Larissa, Thessaly), is Greek physician of antiquity who is traditionally regarded as the father of medicine.

The well famous physician of ancient Greece Hyppocrates is the founder of diagnostics. Some of the general principles used at his time are still valuable for modern medicine.

Hyppocrates is created the oath (swear). It's the rules for moral behavior of doctor.

Oath is the ethical code attributed to the ancient Greek physician Hyppocrates, adopted as a guide to conduct by the medical profession throughout the ages and still used in the graduation ceremonies of many medical schools. Although little is known of the life of Hyppocrates – or, indeed, if he was the only practitioner of the time using this name – a body of manuscripts, called the Hyppocratic Collection (Corpus Hyppocraticum), survived until modern times. In addition to containing information on medical matters, the collection embodied a code of principles for the teachers of medicine and for their students. This code, or a fragment of it, has been handed down in various versions through generations of physicians as the Hyppocratic oath.

The text of the oath itself is divided into two major sections. The first sets out the obligations of the physician to students of medicine and the duties of pupil to teacher. In the second section the physician pledges to prescribe only beneficial treatments, according to his abilities and judgment; to refrain from causing harm or hurt, and to live an exemplary personal and professional life.

In conclusion, the ideas of Hyppocratic Medicine have a transcultural and universal significance.

The fundamental reason why Hyppocratic medicine can speak timelessly and relevantly to us today is because our embodied humanity is the

same now as it was in ancient Greece. Hence, the activity of healing and caring for the sick is essentially the same; it cannot change. Nor can the desire of the sick to get well. The basic understanding of our being healthy as a natural condition of our embodied selves working well is also the same. Clearly human beings have not invented animal or human health. Whatever our difficulties in understanding people of other times, other languages and other cultures. We are all products of similar natural processes and we all live out our lives under the same skies and this gives us at least enough in common to recognizes each other's reality: "the health of my patient", an individual human being. Aristotle with his profound sense both of the general and of the concrete says in this respect: "what the doctor appears to consider is not even health universally, but the human being's health and even more than that, presumably, this human being's health, since it is particular patients that he heals".

The aim of medicine is to expand diagnostic skills, proliferate treatment alternatives, prolongation of physical life, and final purpose of which is the complete recovery of the patients.

Moral philosophy should become the basic science for the art of medicine.

Thus medicine's central concern is to heal, care for and comfort the sick, within the context of an unconditional respect for the dignity of each human being.

At any time, the society elaborated the set of requirement towards the medicine in generally and physician as professional.

These requirements include professional competence and compassion, which are connected with ethical and moral duties.

Professional competence of the physician consists from the good theoretical knowledge, perfect diagnostic skills, and knowledge of pharmacotherapy. A good physician should know all recent advances in medicine, follow all periodicals and other publications in his profession, attend meetings of medical societies, conferences, take active part of them, and should also be acquainted properly with the problems of the neighboring medical specialties.

The doctors should constant drive to improve their knowledge and skills. But these scientific skills are not enough to define professional com-

petence. Competence must include other skills, less easily testable on multichoice test, but nonetheless objective.

More important, many decisions in medicine, especially moral decisions, involve subtle from which great benefit and harm can come to patients.

Compassion must sometimes involve feeling. Compassion involves responding to suffering and although a virtue for everyone, it's a special virtue for physicians who see great suffering. Suffering and unwanted pain are the ultimate enemies and the first steps in defeating them are compassion and virtues.

Something is wrong with the physician who never feels concern for anyone, just as a person who never cares about anyone is morally suspicious. As a kind of desire, compassion involves wants to end suffering.

As a disposition to act, compassion involves activating to end suffering. Compassion is a good partly because lonely humans, cut off from others by disease need others to understand them. But compassion is good also because people who see suffering in others are motivated to end it.

Compassion by its nature is not impartial response to each patient on the level of emotional sympathy; real compassion involves understanding. Compassion can't be the same for all patients. Each patient is indeed unique. The doctor must treat the patient rather than the disease.

The doctor may be a good diagnostician and be quite exacting in his prescription but all his talents are useless if he is unable to conquer the patient's soul.

Compassion for the patient is not a formal duty but a genuine sympathy for the patient and the desire to help him.

And the patient should feel this compassion whenever he is in contact with medical personnel, beginning with the nurse and secondary personnel and ending with his "savoir", the physician.

The task of medical staff is first to calm the patient, and to remove his fears and anxiety for the outcome of the disease, then to persuade the patient that the outcome will be favorable and to strength his will power, which is necessary for a successful fight against the disease.

Chapter 2. CLINICAL EXAMINATION METHODS

SUBJECTIVE EXAMINATION

Inquiry

Inquiry is used in everyday observation of the patient, and it is very important to have good interview technique. Sometimes information obtained during interview is sufficient to correct preliminary conclusion.

The ability to elicit an accurate history from the patient is crucial. It is the history, which provides the basis for priorities in the clinical examination and subsequent investigation, and management. The style of obtaining a history leads to the therapeutic alliance between doctor and patient – so essential for establishing trust and satisfaction.

It should be remembered that some people are naturally better communicators that others. From the patient's perspective the most important component of the clinical examination is the explanation. Clinicians who are courteous and patient, appear interested, encourage patients and relatives to ask questions and to spend time explaining situation in a way, which is understood, are judged to be 'good doctor', irrespective of their attributes.

Inquiry includes following aspects: general information (passport part) – name, date of birth, age, address, occupation, etc; patient's present complaints; history of the present disease (anamnesis morbi); and past history (anamnesis vitae).

Present Complaints

It is important to establish the patient's presenting complaint or complaints. The patient needs to understand what is being said. Generally speaking, technical terms are best avoided. The public is becoming increasingly informed through the Internet and mass media, but their use of medical jargon does not necessarily mean they understand the terms. Similarly there are terms such as 'shock', 'nervous breakdown', and 'gastric flu', which need to be clarified, if used by the patient.

The presenting complaint, as described by the patient, is the body of the history. The main complaints, those are most pronounced and determine

the clinic of the disease, should be first detected. As a rule, the patient firstly describes the main complaints. However, sometimes the patient complains of unimportant signs, and only additional questioning helps to evaluate the main complaints. Detail description of each complaint should be given according to definite plan: location, intensity, character, course, duration, frequency, radiation, associated symptoms, cause of onset, aggravating factors, and relieving factors.

The patient should be questioning then according to special scheme: ***general condition*** (weakness, fever, skin itching, sweetness, changes of body weight), and then on ***organs and systems:*** *nervous system* (work capacity, mood, memory, attention, sleep, headache, dizziness, etc), *senses organs* (vision, hearing, etc), *respiratory system* (voice changes, pain in the chest during breathing, breathlessness, asthma, cough, expectoration of sputum and blood), *cardiovascular system* (pain in the heart, breathlessness, attacks of suffocation, palpitation, intermissions, edema), *digestive system* (appetite, thirst, swallowing, nausea, vomiting, epigastric pain, defecation, etc), *urinary system* (pain, urination, urine character), *locomotors system* (pain in the joints, muscles, etc). Inquiry should be started from that system on which are the main complaints.

Anamnesis Morbi

Anamnesis morbi or history of the present disease includes obtaining of following information: the time of disease onset (acute or gradual), the cause (if known), the first symptoms and their character, previous examination and results (if any), and treatment and results (if any).

Anamnesis morbi includes data concerning onset and progresses of the present disease until the present. The patient should be asked about the first signs of the disease and their dynamics, about exacerbations, remissions and their duration, about possible previous examinations and treatment and their results in chronological order. And finally, the cause of the present hospitalization should be noted (exacerbation, verification of the diagnosis, etc).

Correctly collected anamnesis morbi can be helpful in identifying of the present disease, because most of them have definite course; one symptom followed another in specific order and quite frequent the present complaints differ from the initial manifestation of the disease.

Anamnesis Vitae

Anamnesis vitae is past medical history of the patient in infancy, childhood, adolescence, and adult. The information obtained during collecting of the anamnesis vitae is very important for understanding of character, cause, and conditions of the disease onset.

The past history involves *biographical data*: place of the birth (endemic diseases), the age of the parents, living conditions in childhood, education, profession, occupation, etc. Then the patient should be asked about his *past diseases* in childhood, adolescent, and adult (tuberculosis, cardiovascular, nervous, psychiatric, endocrine diseases, etc), and also about possible traumas or operation.

Habits. Because of the extent to which smoking cigarettes, drinking alcohol, and narcotics contribute to disease, inquiries into these habits if often necessary. Patients tend to be defensive and are quite likely to deny or minimize their substance use. If here are clinical grounds for suspecting misuse, questioning has to be tactful but firm and persistent. It is important to determine whether the patient is a smoker, an ex-smoker or a lifelong non-smoker. If the patient smokes, the following information is required: form (cigarettes, cigars or pipe); quantity (number of cigarettes/cigars or amount of pipe tobacco per day), and duration. If the patient is an ex-smoker, the length of time since the practice ceased should be noted. In smokers, the possibility of tobacco-related disease should be considered (cerebrovascular disease, tobacco amblyopia, carcinoma of the mouth, lung cancer, chronic obstructive pulmonary disease, ischemic heart disease, peptic ulceration, peripheral vascular disease).

It is often necessary to ask whether the patient teetotal or drinks alcohol. If he does then how much alcohol, frequency and what exactly (vine, beer, vodka) he drinks.

Family history. Information about the health of the patient's relatives (parents, sisters, brothers) is very important because some diseases are caused by hereditary factors. However it should be noted that predisposition to some disease not always provoke its development. Pathological heredity as a rule manifests under harmful environmental conditions (hypertension, atherosclerosis, cholelithiasis, etc). Inherited character of the diseases can vary widely. Some inherited disease may observed only in one member of

the family, or in offspring after several generation, or in family members of one sex (hemophilia).

Social history should include basic information about occupation and domestic arrangements. It is important to determine labor conditions, exposure to hazard, e.g. chemicals, mechanical (noise, vibration, high or low temperature, etc), foreign travels, and accidents. It is necessary to ask the patient about his living condition: type of home, size, owned or rented, illumination, if it is damp or dry, hygienic conditions, etc. The patient should be also asked about the nutrition: regularity, quantity (under eating, overeating), character of food (is the diet sufficient rich in vegetables and fruits, is there meat or fatty food abuse or salt abuse, etc).

Allergological history. It is necessary to determine in the patient and his relatives possible allergic reactions to various food (strawberry, eggs, crabs, etc), cosmetics, odor, etc. It is important to reveal whether the patient has ever experienced an adverse reaction to a drug and, if so, to record the information prominently so that it is immediately obvious at any future presentation. Allergic reactions are quite varied: from vasomotor rhinitis, Quincke's edema to even anaphylactic shock. Failure to obtain and record an adverse reaction therefore may lead to serious illness or even death.

OBJECTIVE EXAMINATION

Objective examination of the patient is helpful to obtain information about *status praesens* – condition of the entire body and of the internal organs.

Examinations methods are divided into the main and auxiliary.

The main methods include systemic inquiry and physical examination. The time-honored sequence of physical examination is inspection, palpation, percussion and auscultation. These have to be integrated into the examination and can be altered as deemed necessary. For example, it may be advisable to listen to bowel sounds before palpating the abdomen, or percuss liver before it's palpating. Although the tendency is to teach the technique of physical examination system by system, in practice these require to be integrated because this approach is less tiring to the patient.

The auxiliary methods include instrumental and laboratory methods of examination, which will be discussed in the special part. These methods are called auxiliary because only after use of the main methods physician decides which instrumental or laboratory method is necessary to verify preliminary diagnosis. Other specialists conduct the auxiliary examination.

General inspection

General patient's condition may be good, satisfactory, moderate grave, grave, extremely grave. The criteria's of patient's condition are the following clinical features: consciousness, posture, gait, the facial expression, weight, and mental condition.

Good patient's condition is characterized by clear consciousness, active posture, free gait, sensible facial expression, sufficient weight, and good mood. This condition occurs in patients with remission of chronic disease favorable course of a disease, or during recovery.

Satisfactory patient's condition (*status morboacili*) is characterized by clear consciousness, active or active with restriction posture, free or partial deranged (specific) gait, sensible facial expression, and adequate mental reaction. This condition occurs in patients with remission of prolong chronic disease, or during recovery from acute disease.

Moderate condition (*status ingravescens*) is characterized by deranged consciousness, alteration of facial expression and posture (forced), uncertain gait, partial deranged mental state and may be observed in patients with recurrence of chronic disease, acute diseases, or due to the traumas and poisoning.

Grave condition (*status morbogravi*) is characterized by disorders of practically all clinical features: deranged consciousness, changed facial expression (fear, suffer, hopelessness, indifference). The patients have forced or passive posture, loss of weight, edema, and inadequate mental state. Grave condition is observed in patients with infections and oncological diseases, heart failure, disorders of renal, liver functions, abnormalities of nervous and endocrine systems, after operations, traumas.

Extremely grave condition (*status gravissimus*) is characterized by unconsciousness, passive posture, and indifferent facial expression and observes in the patient with coma, shock, and agony.

Consciousness (*sensorium*) may be clear or deranged. The criteria's of consciousness condition are the following features: orientation to the surroundings, adequate answers, concentrated attention, reflexes, and pupil reaction on light.

Clear consciousness (*sensorium lucidum*) is characterized by adequate behavior, correct orientation to the surroundings, timely answer to the question, and preservation of all reflexes.

The deranged consciousness develops due to the different causes: disorders of cerebral or cardiac circulation; endogenic and exogenic intoxication; infectious affections; hormonal, mineral, metabolic abnormalities; and traumas of the brain.

The deranged consciousness is divided into two groups – depressed (stupor, sopor, coma) (Tab. 2.1) and excited consciousness (irritative disorder, delirium).

Tab. 2.1. ***The forms of depressed consciousness***

Forms	Definition	State
Cloudiness	Disorientation in space, indifferent, the answers adequate, but delayed, reflexes are present	Intoxication, infectious diseases
Stupor	Disorientation in space, surroundings, the answers inadequate and delayed, reflexes are present	Contusion, intoxication, mental diseases
Sopor	Disorientation in time, space, surroundings, own personality. Pathological deep sleep from which patient wake up only for short periods of time when called loudly or roused by an external stimulus, reflexes are present, but delayed	High temperature intoxication, infectious diseases, poisoning
Coma	Unconsciousness with absence of response to external stimuli, absence of reflexes, deranged vital function	Stroke, poisoning, metabolic, hormonal disorders

The following forms of coma are most common.

Coma due to the disorders of cerebral or cardiac circulation:

– *apoplexic coma* resulting from stroke, thromboembolism of cerebral vessels is characterized by asymmetric face, noisy, deep breathing, narrowed pupil. Duration from several hours to several days;

– *syncope* – transient loss of consciousness (1–10 min) resulted from acute ischemia of brain due to aortic valve disease, acute hemorrhage. Recurrent syncope or "Adams-Stokes attacks" may occur in patients with episodes of ventricular asystole, which complicates complete (third degree) atrioventricular block or Mobitz type II (second degree) atrioventricular block, recurrent sinoatrial block. A typical episode is characterized by a sudden loss of consciousness, which frequently appears without warning and may result in a fall. Convulsions (due to cerebral ischemia) can occur if there is prolonged asystole. There is pallor and deathlike appearance during the attack. In contrast to epilepsy, recovery is rapid.

Endotoxic coma: *diabetic coma* occurs in patients with diabetes mellitus due to metabolic carbohydrate and lipid disorders. The biochemical basis of the diabetic coma is hyperglycemia and ketoacidosis, but these parameters are not always necessarily closely correlated. Even moderate hyperglycemia may be associated with life-threatening acidosis. The rise in hydrogen ion concentration in the arterial blood alters pulmonary ventilation and "air hunger" is observed clinically. Coma may occur with extreme hyperglycemia and dehydration but no ketoacidosis so called hyperosmolar diabetic coma. The causes of coma with ketoacidosis: too little or no insulin; an infection; digestive disturbance. The duration of prodromal period are several days. The symptoms of ketosis; abdominal pain and vomiting, dry skin and tongue, noisy, deep breathing (Kussmaul's respiration), the pupils are dilated, acetone smell from mouth and urine, weak pulse, low blood pressure, diminished reflexes and finally loss of consciousness.

Hypoglycemic coma occurs in patients with diabetes mellitus commonly treated with insulin due to sudden decreasing of a blood glucose concentration less than 2.5 mmol/l. The causes of hypoglycemia: poorly designed insulin regime; missed, delayed or inadequate meal; alcohol; other endocrine disorder. Symptoms of hypoglycemia: sweating, feeling of hun-

ger, anxiety, trembling, accelerated heart beat, speech difficulty, confusion, inability to concentrate and finally loss of consciousness (Davidson's "Principles and Practice of Medicine", 1995).

Tab. 2.2. ***Difference in coma due to hypoglycemia and ketoacidosis in insulin depended diabetes mellitus***

	Hypoglycemic coma	**Coma with ketosis**
History	No food, too much insulin, unaccustomed exercise	Too little or no insulin, an infection; digestive disturbance
Onset	In good previous health; related to last insulin injection in some min.	Ill-health for several days
Biochemical bases	Hypoglycemia less than 2.5 mmol/l	Ketoacidosis, hyperglycemia and increased osmolarity of plasma
Complaints (before coma)	Feeling of hungry Fatigue	Vomiting Abdominal pain
Signs	Moist skin and tongue Shallow or normal breathing Trembling Full pulse Normal or raised SBP Brisk reflexes	Dry skin and tongue "Air hunger" Kussmaul's respiration Weak pulse Low BP Diminished reflexes
Urine	No ketonuria No glucosuria, if bladder recently employed	Ketonuria Glucosuria
Blood	Hypoglycemia, normal plasma bicarbonate	Hyperglycemia, reduced plasma bicarbonate

Hepatic coma develops in patients with acute and subacute dystrophy and necrosis of the liver parenchyma (acute viral hepatitis, acute drug-in-

duced liver disease); in the patient with final stage of liver cirrhosis related to disorders of bilirubin, protein, and carbohydrate metabolism. The main cardinal manifestation of hepatic failure results from cerebral disturbance – encephalopathy. Cerebral edema and toxicity (intoxication) are important factors. The earliest features are poor concentration progressing through behavioral abnormalities such as restlessness, aggressive outbursts and mania to drowsiness and coma. Confusion, disorientations, inversion of sleep rhythm, slurred speech and convulsion may occur. An unequal or abnormally reaction pupils, hyperventilation, local or general myoclonus tremor of the extended hands are characteristics. More general symptoms include weakness, nausea, vomiting, right hypochondrial pain, profuse sweating.

Examination shows jaundice, splenomegaly, and enlargement of liver. Liver later becomes impalpable; disappearance of hepatic dullness on percussion indicates a bad prognosis.

Uremic coma develops slowly in patients with congenital and inherited renal diseases, glomerular and interstitial diseases, obstructive uropathy, as complication of vascular systemic diseases, in condition which destroyed the normal structure and function of the kidney, acute and chronic renal failure develops. Disturbance in water, electrolyte and acid-base balance contribute to the clinical picture in patients because these substances present in abnormal concentration in the plasma has been suspected of being a "uremic toxin". As a rule, uremia occurs in acute toxic and terminal stages of chronic diseases of the kidneys. Symptoms of uremia: generalized weakness and lack of energy, anorexia, nausea and vomiting, particularly in mornings, breathlessness on exertion, headaches, visual disturbances, pallor, generalized myopathy, muscle cramps, neuropathy. In case of impaired renal function and progression of uremia develops loss of consciousness.

Exotoxic coma is relevant to acute poisoning. Poisoning substances may give rise to primary toxic effects, which may result in organ damage of a nonspecific type.

The organ damage may then lead to respiratory or metabolic disturbance or a combination of these, hence to a variety of clinical features.

The Edinburgh Coma Classification was proposed in order to assess the complex neurological abnormalities (Tab. 2.3).

Tab. 2.3. ***Edinburgh coma classification: impaired levels of consciousness in patients with acute poisoning***

Grade of severity	Definition
0	Fully conscious
1	Drowsy bat responsive to vocal command
2	Unconscious but responsive to minimal painful stimuli
3	Unconscious but just responsive to strong painful stimuli
4	Unconscious with no response to stimuli

The forms of exited consciousness

Twilight state is characterized by disorientation in surroundings, loss of memory (amnesia), patient is exited, has pathologically high spirits, is anxious, sometimes even aggressive. This state may observe in patients with epilepsy.

Delirium is characterized with visual and acoustic hallucinations, inadequacy of emotions, anxiety, intermittent thinking. There are some kinds of delirium: alcoholic (delirium tremens), infection, senile, traumatic, pharmacogenic, epileptic.

Posture of the patients may be active, active with restriction, passive, and forced (Tab. 2.4).

Active posture with restriction may observe in patients, which suffer with pain due to the affection of joints. The patients feel pain during walking that can limit their activity.

Tab. 2.4. ***Posture of the patients***

Forms	Definition	Pathological state
Active	Patient has ability to walk, stand, to change his posture	Mild disease Onset of a grave disease
Passive	Patient is motionless, he lies, his head and the limbs hand down	Unconscious state
Forced	Forced posture assumed by the patient to relieve or remove pain, cough, dyspnea, or other signs of disease	Grave disease

Forced posture differs relevant to the process, which cause specific patients position.

Standing upright position is observed in the patient with attacks of angina pectoris (sudden and severe pain in the retrosternal region) feeling of fear, absence of air. The patients have to stop, after rest the pain disappears.

Standing upright position is observed also in patients with atheromatous peripheral vascular disease. The commonest presenting symptom is intermittent claudication – a discomfort or pain in the calves or buttocks, which comes on with walking and makes patient stop. The unpleasant feeling disappears with rest. This stopping may repeat every 100–200 m of walking at the onset of disease. Later with progression of atherosclerotic process this interval become shorten and accompanies another signs: coldness of the feet or lower limbs, discoloration due to the peripheral cyanosis, diminished or absent peripheral pulse;

Sitting position – orthopnea (Gk *orthos* – straight, *pnoe* – breath) – the severe stage of short breath: circulatory insufficiency, attacks of bronchial asthma, cardiac asthma; narrowing bronchitis due to inspiration of the foreign body, tumor bronchitis, spasm of bronchi; decreased breath surface in patients with pneumonia, lung tumor, pneumothorax, effusive pleurisy. This position improves the patient's state; due to decreased volume of circulating blood dyspnea becomes less expressed.

- Sitting position fixing the shoulder girdle is characteristic of bronchial asthma attack. This position assists the accessory muscles and diaphragm to take part in respiration, thus promotes chest widening during inspiration and improves patient's condition.
- Sitting position with legs hanging down from the bed is due to the acute left ventricular heart failure – cardiac asthma. The patient's condition improved according to the distribution circulating blood from the lesser circulation to the low limbs, decreasing blood pressure in the pulmonary artery, increasing cerebral circulation.
- Sitting position and inclines forward may observe in the patient with pericarditis, which produce a pericardiac effusion resulted restriction a diastolic heart function.

The supine posture is characteristic of strong pain in the abdomen – acute appendicitis, acute cholecystitis, and perforated ulcer of stomach or

duodenum. Sometimes patient bends the leg in knee joint for decreasing marked strain of muscle of the abdominal wall.

The supine posture with complete immobility is observed in patients with acute rheumatic polyarthritis due to the severe pain; patients with scleroderma and patients with severe fatigue.

The position lying on the side with head thrown back and the thighs and legs flexed on the abdomen is characteristic of cerebrospinal meningitis due to the rigidity and contraction of the muscles at the neck and trunk of varying degree. The back is usually slightly arched and there is a board-like abdominal wall.

Opistotonus is characteristic of paralytic rabies. The patient has forced supine position, under extreme stimuli occurs tetanic cramp of long back muscle and back is arched with 2–3 points touching with bed: back of the head, pelvis and heel. This position may observe in meningitis, epilepsy, some poisoning.

The position "lying with raising of head end of the bed" is characteristic patients with chronic heart failure.

The prone position (lying with face down) is characteristic of patients with tumor of pancreas, gastric ulcer (in the posterior wall of the stomach is affected), acute thrombosis of lien vein, trauma and tuberculosis of spine, trophic ulcer, placed on the skin of back and buttock.

The forced posture on the side:

- *on the affected side* lie the patients with the lung, pleura diseases. The patients with dry pleurisy prefer to lie on the affected side because the limitation of the pleural layers movement relieves the pain. The patients with pneumonia, massive lung tumor, effusive pleurisy prefers to lie on the affected side for decreasing dyspnea resulted decline pressure and hyperventilation of healthy lung;
- *on the healthy side* often lie the patients with fractured ribs, intercostal neuralgia, herpes zoster, because pain intensifies if the affected side is pressed against bed.

The forced "knee-elbow position" with bend trunk forward may observe in patients with effusive pericarditis.

The state of restless, anxiety, occur in the patient due to the urinary tract calculi and nephrocalcinosis. When a stone becomes impacted in the

ureter, an attack of renal colic develops. The patient suddenly aware of pain in the loin, which soon radiate rounds the flank to the groin in the sensory distribution of the first lumbar nerve. The pain steadily increases in intensity to reach a maximum in a few minutes. The patient is restless, and generally tries unsuccessfully to obtain relief by changing position and by pacing the room. There is pallor, and often vomiting.

Gait – combination of the pose and movement during walking. Gait depends from the state of nervous system, connective tissue, muscles, joint and bones.

Gait of the healthy person is firm, free, and straight. There are some specific gaits according to the pathological processes:

- *spastic gait* is characterized by small step with difficulties during bend of limb in knee and hell clinging due to the a pyramidal tract lesion;
- *paretic gait* is characterized by slow movement with difficulties walking due to the development of flexor spasm and contractures in the limbs resulted from the tumors, trauma and other forms of spinal compression, degeneration of the cord. In severe cases the patient loss of ability to walk and becomes immobility. Another clinical signs of spinal compression: the loss of sensation disorders of urination and stools;
- *hemiplegic/circumductive gait* is characterized by abundance (superfluous) leg draw aside and the arm from the same side bond to the trunk due to the increased muscle tone resulted from central hemiparesis;
- *doll's/puppet gait* is observed in patients with Parkinsonism, which includes three main components: tremor, muscular rigidity and hypokinesis. Tremor may affect the legs, mouth, and tongue, head. Many patients have difficulty in initiating rapid fine movement, and slowness of gait and difficulty with tasks such as fastening buttons or writing. Rigidity of muscular tone causes stiffness and flexed posture;
- *peroneal gait*, stoppage is characterized by high climb of leg, sharp drawing; it due to the muscular atony in patients with damage of femur nerve;
- *cerebellar gait*, wobbly/tottering/reeling gait is characterized by incoordination of ipsilateral limbs: decomposition of movements, impaired alternating movements; loss of balance: broad based gait, leaning towards of lesion; hypotonia of limbs; head tremor and may observe in patients with damage of cerebellum, alcohol abuse;

- *ataxic gait* – (origin from Greek *ataktos* – confused) is characterized by high rising of climb, reach the floor, limb continue to search fulcrum. This gait is due the discoordination resulted from affection of sensory system components (posterior column of spinal cord and peripheral nerves). Ataxic gait is observed in patients with polyneuritis, hereditary ataxia's, neurosyphilis;
- *gait as "a duck"* – is characterized by small, slow step with compensatory inclination trunk to the opposite side due to the hypotonia of pelvis muscle in patients with myopathy, a dislocation of femur, aseptic necrosis of femur, osteomyolitis;
- *gait with forced movements* femur nerve fibula neuritis is observed in patients with child central paralysis;
- *retarded gait* is characterized by small snuffle step with uncertain and uncoordinative movement of arms due to the pronounced cerebral atherosclerosis;
- *"proud" gait* is characterized by putting trunk backward for support balance relevant to pregnancy, ascitis, or great tumor of abdominal cavity

Habitus. The concept of habitus includes the body-build, height and body weight. In addition to general inspection it is necessary, to perform some anthropometry measurement.

Height. The main anthropometric signs are the man's height and weight, which depend on the ethnic factors. The normal height of males varies from 165 to 180 cm, females 155–170 cm. Deviations on either side are connected with endocrine dysfunction.

Dwarfism may be due to hypofunction of the anterior lobe of the pituitary (nanism) or of the thyroid gland (cretinism).

Gigantism can be due to dysfunction of the anterior lobe of the pituitary or hypofunction of the sex glands.

Patients height and the length of his trunk are important for the assessment of both his physical grow and proportions of his separate parts, which can be upset in some congenital diseases and disease acquired in childhood.

Weight is measured on a special medical balance weight should be done in the morning. Whenever possible the patient should be with no clothing. In order to follow changes the patient's weight repeated weightings should be done in the same conditions.

Body Mass Index (BMI) was proposed in order to assess the weight in adults. BMI may calculate using formula:

$$BMI = \frac{weight\,(kg)}{height\,(m^2)}$$

According to the BMI it is possible to reveal the overweight and obesity (Tab. 2.5).

Tab. 2.5. *WHO classification of overweight and obesity in adults*

Category	Body Mass Index (BMI), kg/m^2
Underweight	< 18.5
Normal weight	18.5–24.9
Overweight	≥25.0
Pre-obese	25.0–29.9
Obese class I	30.0–34.9
Obese class II	35.0–39.9
Obese class III	≥40.0

Gain of weight may be in persons without weight control, having eating habits with increased intake of carbohydrates, saturated fat and alcohol. Endocrine disorders are potential contributors to obesity (Cushing's syndrome, hypofunction of thyroid gland, diabetes mellitus I type).

Note: The BMI values are independent of age and sex. BMI, however, may not correspond to the same degree of adiposity between populations, in part because of different body proportions.

Loss of weight is observed in persons during starvation, in patients with grave diseases, oncology pathology, endocrine dysfunction.

Body-build is determined by morphological bodily features and divided into two groups:

a) correct habitus with a well proportioned make up of the part of the body: trunk, head, limbs without deformity;

b) incorrect habitus with different deformity and disproportion of the trunk, limbs, chest, and abdomen.

Face of the patient

Face in patient with pathology of respiratory system:

- *facies pneumonica* – one-sided blush on the same cheek as affected lung, cyanosis, often herpes on the lips and nose;
- *facies tuberculous* – exhausted, pale face with blush localized on the cheeks, "burning eyes", dry lips, excited countenance, half open mouth;
- *facies asthmatica* – pale, cyanotic face, sweating, cool extremities, an unproductive cough, accelerated breathing rate;
- *facies adenoidea* – half open or full open mouth, loose-hanging lower lip, noisy breathing.

Face in patient with pathology of cardiovascular system:

- *facies aortale* – pale skin, rhythmical movements of the head, simultaneously with aortic regurgitation (Musset's symptom);
- *facies mitrale* – the patient looks younger his age, face with blush, localized on the cheeks, cyanotic color of the tip nose, ears, dyspnea. The face is observed in patients with mitral stenosis;
- *facies Corvisara*, facies cardiaca – is characteristic of heart failure. The face is edematous, pale, and yellowish with a cyanotic hue. The mouth is always half open, the lips are cyanotic, the yeas are dull;
- *facies plethorica* – hyperemic and cyanotic skin, puffy face due to the excessive circulated blood in patients, plethora with hypertensive crises.

Face in patient with endocrine pathology:

- *facies acromegalica* – due to the hyperproduction of growth hormone by anterior lobe of hypophysis. The disease is characterized by disproportional growth of all bones, soft tissues, and internal organs. There are enlarged superciliary arches, zygomatic bones, ears, auricles nose, lips, tongue, growth and putting forward of low jaw (prognotism). The head is elongated, and the facial features appear generally coarsened. The hand wrists, feet, heel bones also enlarge;
- *facies in patients with Cushing's syndrome* due to the increased excessive cortisone production in patients with adrenal tumor or prolonged glucocorticoid administration is characterized by round or "moon-like" face, plethora, red cheeks. Excessive hair growth may be present in the mustache and sideburn areas and on the chin (hirsutism in women). The additional clinical obesity, diabetes and increased bruisability;

– *facies myxoedemica* in patient with severe hypothyroidism (myxedema) due to the thyroid hypofunction has a dull, puffy face, with purplish lips and malar flush. The edema often particularly pronounced around the eyes does not pit with pressure. The hair and eyebrows are dry, coarse, the hair is thinned or absent on the outward portions of the eyebrows. The face is pallor due to the vasoconstriction and anemia. The presence of a blush on a pale face resembles the appearance of a doll. The patients have often drowsiness and diminished intellect (if the disease begin from childhood);
– *facies basedovica* are observed in patients with hyperthyroidism which results from exposure of the body tissues to excess circulating levels of free thyroid hormones. The face is lively with widened eye slits and abnormally sparkling eyes: the eyes are protruded (exophtalmus). The face looks as if frightened;
– *facies in patients with hypogonadism* is characterized by dry skin, wrinkled, absence of hair in men, thin eyebrows, looks as "baked apple".

Face in patient with pathology of kidney:

– *facies nefritica* – the face is edematous and often pale. Swelling usually appears first around the eyes and in the morning. The eyes may become slit like when edema is pronounced.

Face in patient with pathology of nervous system:

– *facies amimica*, Parkinson's mask in patients with blunts expression. A mask like, amimic face may result, with decreased blanking and a characteristic stare. Since the neck and upper trunk tend to flex forward, the patient seems to peer upward toward the observer. Facial skin becomes oily and drooling may occur. This face is observed in patients with cerebral atherosclerosis;
– *facies myopathica* – with half-open month, without wrangle on the forehead, amimic, halt open eyes is characteristic of progressive myopathy;
– *risus sardonicus* with a resemblance of a grieve occurs in tetanus patients: the mouth widens as in laughter, while the skin folds on the forehead express grief;
– *facies asymmetrica* – asymmetries movements of facial muscles of central or peripheral facial neuritis.

Face in patient with infectious pathologies:

– *facies fibrilis* is characterized hyperemic skin, sparkling eyes and excited expression;

- *facies* in patients with *louse-borne typhus*: general hyperemia, the sclera is injected ("rabbit eye");
- *facies* in patients with *typhoid fever*: slightly icteric yellow color;
- *facies* in patients with *meningitis*: the countenance of the read, anisocoria (different size of pupils), ptosis;
- *facies* in patients with *cholera* – frequent blinking, changing grimace, disorderly (irregular)mobility of face;
- *facies leontina* with nodular thickening of the skin under the eyes and over the eyebrows, with flattered nose is observed in leprosy;
- *facies* in patients with *parotitis* (mumps) swelling of parotid glands, which are visible above the angles of the jaw. At first it may be unilateral swelling, gradually become bilateral swelling due to the parotid gland enlargement;
- *facies* in patients with *whooping cough*: puffy face with edematous eyelids, conjunctiva hemorrhage, constant tears.

Face in patient with pathology of blood system:

- *facies anemic* – very pale, with greenish tint in patients with iron deficiency anemia;
- *facies* as a *"wax-doll"*: very pale with yellowish tint and seemingly translucent skin.

Face in patient with pathology of digestive system:

- *facies Hyppocratica*: sunken eyes, pinched nose, deadly livid and cyanotic skin, which is sometimes covered with large drops of cold sweat. This face is specific for the collapse due to the grave disease of abdominal organs, accompanied by peritonitis (rupture of gall blander, perforated ulcer of the stomach or duodenum).

Face in patient with another pathology:

- *facies cachectica* – very pale, with earth like tint, pinched nose. Patient loss weight significantly. These features are observed in patients with malignant tumor of digestive system;
- *facies potatorice* – hyperemic skin, especially nose, cyanosis of the nose, lips, cheek, observed on alcohol abuse;
- *Stokes collar* – means edematous neck associated with edematous face due to the compression of lymph ducts and veins with enlarged mediastinal lymph nodes, tumor of mediastinum, adhesive mediastinopericarditis, excessive effusion in the pleural and pericardial cavity.

Skin. Examination of the skin should be performed using the general inspection and palpation. During the inspection of the skin attention should be paid to the color; eruption of the skin; turgor and elasticity (visual and palpative methods); moisture of the skin (visual and palpative methods); edema; temperature of the skin, subcutaneous veins.

Color of the skin

In healthy person skin has corporeal color (*cutis coloris somatici*), without eruption, moderate moisture and elasticity, preserved turgor. The color of the skin depends of the amount and quality of pigments, the blood circulation, and chemical content of blood and on the thickness of the skin. In order to assess correctly the color of the skin, the patient should be inspected in daylight.

There are following pathological changes of the skin color: pale, red, cyanotic, yellow, and bronze. The pale and red color of the skin related to the thickness, blood circulation, innervation and may be transient character in physiological condition (fear, high and low temperature of the air). The yellow, cyanotic and bronze color of the skin are due to the changing of the chemical blood content and are observed only in pathological condition except physiological jaundice at newborn.

Pale color of the skin (*cutis pallide*) may be physiological and pathological.

Transient physiological pallid skin pear is due to the vasomotor reaction of central (fear) and peripheral origin (effect of low temperature).

Constant physiological pallid skin is observed in patients with thick skin, insufficient development of subcutaneous vessels.

Pathological pallid skin is connected with amount and quality of blood. The changing quality of blood is observed in patients with decreased number of erythrocytes and/or hemoglobin content in a blood unit volume, classified as anemia, which accompanied such diseases as hemoblastosis, different types of anemia; acute and chronic infections with hemolysis (malaria, sepsis) and chronic toxicity.

The causes of altered amount of blood:

absolute decreasing of blood amount due to the external hemorrhage: (traumas, burns, wounds) and internal hemorrhage: (gastro-intestinal, rupture of spleen, extra-uterine, hemorrhoid, dilated esophageal veins);

relative decreasing of blood amount due to the vascular spasms and accumulation of blood in dilated vessels, narrowing of vessels by renal and cardiac edema; insufficient blood filling of vessels in patients with aortic regurgitation.

The doctor should pay attention on the duration of development such symptoms as: sudden sharp pallor skin, which accompanied with syncope, sharp diminishing blood pressure, and low pulse. These clinical features indicate to the threaten life acute internal bleeding, which require emergency help.

In certain forms of anemia the skin is specifically pallid: characteristic yellowish tint occurs in Addison-Biermer anemia, hemolytic anemia; greenish tint – in iron deficiency anemia (chlorosis).

Specific earth-like tint occurs in malignant tumor; brown or ash-colored – in malaria; "cafe an lait" (coffee with milk) in infectious endocarditis. Pale, yellow and cyanotic color of the skin is typical to congestive heart failure.

Red color of the skin (*cutis rubra s. erythema*) may be local or diffuse related to the quality of blood, circulation and innervation, thickness of the skin.

Red color of the skin can be of *physiological* origin in persons who are permanently exposed to high temperature; sunshine; with superficial location of skin vessels; excitement.

Pathological red color may be transient in fever. In patient with acute pneumonia the redness is located on checks, more pronounced on the side of the affected lung. *Local erythema* as two-sided blush is characteristic of mitral stenosis ("mitral butterfly" with cyanotic tint), lupus hemoglobin concentration erythematous ("lupus bufferfly") and tuberculosis.

Constant diffuse erythema is observed in polycythemia (erythremia), and explained by excessive production by bone marrow erythroid precursor and consequently the erythrocyte count, hemoglobin concentration increases in peripheral blood.

Cyanosis (*cutis cyanotica*) may be due to the changing the quality of blood – (accumulation of the carbon dioxide and reduced restored hemoglobin) and venous congestion.

There are three forms of cyanosis: central or diffuse, peripheral or acrocyanosis and local one.

Central or diffuse cyanosis (*cyanosis diffuse*) may be observed in such pathological states as:

- chronic lung diseases (chronic bronchitis, acute pneumonia, emphysema, pneumosclerosis, bronchial asthma, atelectasis, thromboembolism of the pulmonary artery);
- poisoning of the hemolytic substances;
- congenital heart disease.

Peripheral or acrocyanosis is observed in patients with congestive heart failure. The blue color appears in the lips, cheeks, ear auricles, tip of the nose, and fingers.

Local cyanosis (*cyanosis localis*) is observed in patients with thrombosis of artery or vein.

Yellow skin and mucosa (*cutis icterica, s. icterus*) can be due to increased concentration of bilirubin in the blood (bilirubinemia) and accumulation it in the tissue and skin. Initial and moderate yellow skin is named subicterus, pronounced yellow color defines as jaundice. Physiological, pathological and exogenic jaundice are differentiated.

Physiological jaundice is observed in newborn at first 5–7 days and resulted from hemolysis of excessive erythrocyte amount during transition to external respiration.

Pathological jaundice are divided into three types according to their etiology:

- ***hemolytic or suprahepatic jaundice*** (*icterus colore citricoluteo s. icterus suprahepatica*) is characterized by lemon-yellow tint due to the excessive hemolysis of erythrocytes in the cells of the reticulohistocytic system (spleen, liver, bone marrow). Hemoglobin brakes down to the globin and haem. Bilirubin is formed from the released haem and accumulate in blood observe in malaria, sepsis, poisoning. The jaundice of hemolytic substances, inherited or acquired hemolytic anemia;
- ***parenchymatous or hepatic jaundice*** (*icterus colore rubiginoso s. icterus hepatica*) is characterized by orange-yellow tint due to the damage of hepatocytes and disorders of their function (inversion of unbound bilirubin to bound), observe in acute and chronic hepatitis, poisoning;
- ***obstructive or subhepatic jaundice*** (*icterus colore luteoviridi s. icterus infrahepatica*) is characterized by greenish-yellow tint due to the accumu-

lation of bilirubin (the product of gradual oxidation of bilirubin) resulted from partial or complete obstruction of the common bile duct in patients with stones in the gall bladder, cancer of the head of the pancreas, cancer of the major duodenal papilla.

Exogenic jaundice or xanthosis related with prolonged using of carotin (carrots), oranges, tangerines and administration of ethacridine lactate (rivanol), picric acid. This jaundice defines as false one and differs from true on some signs (Tab. 2.6).

Tab. 2.6. ***Difference between true and false jaundice***

Signs	Jaundice	
	True	False
Location of the yellow color	– sclera – low surface of the tongue – soft palate – palms – soles – entire skin	– palms – soles
Itching of the skin	Present	Absent
Scratching of the skin	Present	Absent

In some cases the skin becomes yellow pallid due to hemorrhage from varicose esophageal or hemorrhoid vein in portal cirrhosis of the liver.

Brown or bronze skin can observe in physiological and pathological condition.

Physiological brown color is of transient character and observes during prolonged exposure of sunshine (gelioxanthosis) and in pregnancy (as a separate brown points).

Pathological brown or bronze color is observed in patients with Addison's disease or bronze disease resulted from the adrenal insufficiency in patients with hypofunction of adrenal gland. There is specific consequence in appearance bronze skin color. First open parts of the body: face, neck, arms – "symptom of gloves", later bronze color appears in the site of excessive pressure, next – all skin, except palms and soils. Pathological brown or bronze

color is characteristic of hemochromatosis (bronzed diabetes or pigmentary cirrhosis of the liver). The disease is associated with inherited disorder of iron metabolism, excessive absorption of iron in the intestine and accumulation of hemosiderin in various tissues and organs, in the first instance in the liver and pancreas with development of cirrhosis and symptoms of diabetes.

Local hyperpigmentation (*chloasmus*) 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.

Grayish ("dirty") color on the open parts of the body (argyria) due to the administration the silver drug for a long time.

Depigmentation (*depigmintatio*) occurs as a symmetrical white spots on the face, trunk, limbs (*vitiligo*), small white foci after syphilis – leukoderma, complete absence of pigment in the skin – *albinismus*.

Eruption of the skin

Herpetic lesions (*herpes*) are small vesicles 0,5 to 1 cm in size, filled with transparent fluid and located near the trigeminus nerve (*herpes labialis, nasalis*), intercostal nerve (*herpes intercostalis*), severe pain appears for some day before and during appearance of herpes zoster. Herpetic lesions (*herpes simple*) appear on the chin, forehead cheeks and ears in patients with pneumonia, malaria, influenza, and meningitis.

Hemorrhage lesions are of different forms:

- petechia – small pointed hemorrhages;
- ecchymoses – large black and blue spots, a large extravasation of blood into the skin;
- purpura (hemopurpura) – red spots of different size, vary of color from red to yellow-greenish;
- hematoma – a swelling from gross bleeding.

The main causes of the hemorrhage lesions:

- trauma with damage of vessel;
- disease of the blood (Werlhoff's disease, hemophilia, acute leukemia, B_{12}-deficiency anemia);
- disease of liver (obstructive jaundice);
- infections disease, accompanied by capillarotoxicosis;
- deficiency of vitamins C and K.

Roseola is a rash-like eruption of 2–3 mm patches, which disappears when pressed and explained by local dilation of the vessels. Roseola is a characteristic signs of some infections such as typhoid fever, para-typhus, louse-borne typhus and syphilis.

Erythema – large red spot with distinctly outlined margins slightly elevated under skin due to the dilation of the vessel resulted from allergic or inflammatory process. Erythema develops in some persons hypersensitive to strawberries, crabs, and eggs and in patients with erysipelas sepsis, erythema nodosum at rheumatic fever. Sometimes erythema may develop after administration of some drugs.

Erythema nodosum – painful, palpable, dusky blue-red nodules are most commonly seen on the shins. Malaise, fever and joint pains are common. The lesions resolve slowly over a month leaving bruise-like marks in their wake. This characteristic reaction is due to a vasculitis in the deep dermis and subcutaneous fat. Erythema nodosum may be caused by infections, systemic disease, and administration of some drugs.

Weals (*urticaria, nettle rash*) – red round itching lesions elevated under skin, which appear as allergic reaction on some food, chemical substance, drugs.

Teleangioectasia – dark-red spots on the skin and mucosa, the visible dilation of small subcutaneous blood vessels, as a rule on the upper part of the trunk, disappeared after pressing due to the excessive production of estragens in patients with portal liver cirrhosis.

Ulcer (*ulcus*) is damage of the skin and subcutaneous tissue with retarded healing process. According to the etiology there are some groups of ulcer:

- exogenic factors (mechanical, chemical, radiation);
- longstanding administration of glucocorticosteroid drugs – ulcus steroideum;
- disorders of skin trophicity (ulcus trophicum) due to the diabetes mellitus, heart failure, endoarteritis, atherosclerosis of the peripheral arteries, thromboflebitis;
- abnormalities of blood – sickle cell disease, spherocytosis, cryoglobulinemia;
- neuropathy – leprosy, syphilis.

Pustula – a visible accumulation of the pus in the skin.

Abscess – a localized collection of pus in the cavity more than 1 cm in diameter.

Acne vulgaris – lesions, which are limited to the face, shoulders, upper chest and back. Seborrhea (greasy skin) is often present. Acne vulgaris is the chronic inflammation and blocked pilosebaceous follicles and observed in teenagers.

Decubitus – necrosis of the soft tissues due to the ischemia, longstanding mechanical pressure in the in patients with grave diseases.

Scars – the result of healing, in which normal structure are permanently replaced by fibrous tissue. Scars are formed from the connective tissue after inflammatory process and indicate to the previous trauma, burns, operation, infections (tuberculosis, German measles). Scars on the skin of the abdomen and the hips remain after pregnancy (striae gravidarum) due to the over stretching of the skin. Stria – a streak-like, linear, atrophic, pink, purple or white lesions of the skin due to change in the connective tissue. Pink line scars on the abdomen, hips, shoulders are specific for Itsenko-Cushing disease. Stellar scars, tightly connected with under lying tissue are characteristic of syphilitic affections.

Turgor and elasticity of the skin

Turgor (turgor) of the tissue depends on the blood circulation, innervation and metabolism, development of the subcutaneous tissue. Elasticity means flexibility of the skin. Elasticity and turgor can be determined by pressing a fold of skin on the extensor surface of the arm between the thumb and the forefinger. The fold disappears quickly on normal skin when the pressure is released. In cases with decreased turgor, the fold persists for a long period of time. Diagnostic meaning of the diminished turgor: oncology pathology (cachexia); stenosis of the esophagus or pylorus; endocrine pathology (Addison's disease, Simond's disease); infections with dehydration (cholera, dysentery).

Moisture of the skin in normal condition is moderate. Both in physiological and pathological state the skin may be dry or moist (Tab. 2.7).

Tab. 2.7. ***Moisture of skin: the reasons of alteration***

Causes	Dry skin (xeroderma)	Moist skin (hyperhydrosis)
Physiological	Effect of medications: atropine, euphillin	High temperature excessive physical exercise excitement
Pathological	– longstanding vomiting (stenosis of the pylorus, toxicity of pregnancy) – diarrhea (cholera, dysentery) – excessive destruction of tissue (malignant tumor, grave infections) – hypofunction of the thyroid gland (myxedema)	– peritonitis, myocardial infarction, renal and hepatic colic – sharp decreasing of blood pressure (syncope, collapse, shock) – grave infection with high temperature of the body (tuberculosis, influenza, brucellez, pneumonia) – disease of the blood (lymphogranulomatosis, acute leukemia) – hyperfunction of the thyroid gland (thyrotoxicosis), hypoglycemic state

The skin derivates

Nails (Gr *unguis, onychia*) in normal condition are smooth, moderate prominent, of pale pink color with mat surface.

The following pathological changes of nails are of great diagnostic meaning:

– *Nails in* a form of "*watch glass*" (*Hippocratic nails*). Digital clubbing in its most gross form is seen as a bulbous swelling of the tip of the finger or toe. The normal angle between the proximal part of the nail and the skin is lost. Hyppocratic nails appear in the patients with affection of such systems:
  - respiratory – bronchogenic carcinoma, suppurative lung disease,
  - cardiac – congenital heart disease, bacterial endocarditis,
  - other – inflammatory bowel disease, biliary cirrhosis, thyrotoxicosis.

- *Hypertrophic nails* (scleronychia) looks like flattened and thickened in acromegaly;
- *Spoon-shaped nails* (koilonychia) are characterized by altered forms as curved inside with marked transverse folds. Such form of nails is observed in patients with iron deficiency anemia due to the dystrophic process. Specific lines are transverse groves, which appear at the same time on all nails a few weeks after acute illness, and which move out to the free margins as the nails grow;
- *Onychia diabetica* is characterized by hemorrhage, flat and brittle, with parts of hyperpigmentation and observed in diabetes mellitus;
- *"half and half" nails* (white proximally and red-brown distally) are seen in some patients with renal failure.

Hair. Type of hair growth corresponds to the age and sex. If the type of hair growth does not correspond to sex it indicates the endocrine disorder:

- male-type hair growth in women (hirsutism, hypertrichosis);
- abnormally excessive hair growth may be present in the mustache and sideburn areas and on the chin due to the increased adrenal hormone production of Cushing's syndrome;
- Deficient hair growth is characteristic of myxedema, liver cirrhosis, congenital disorders of sex glands;
- Absence of the hair on the outward portions of the eyebrow is characteristic of myxedema;
- Baldness may be general, partial, cellular;
- Premature baldness due to the exposure of radiation;
- Partial or cellular baldness in congenital hypothyreosis;
- General baldness (alopecia) in syphilis.

Edema may be caused by penetration of fluid through the capillary walls and its accumulation in tissues. According to the pathogenic and location factors, edema may by general and local.

General edema associated with disease of the heart, kidneys, and endocrine disorders is characterized by symmetrical localization in some regions of the body or general overspreading of edema throughout the entire body.

There are such kinds of general edema:

- *Congestive* (oedema congestivum) due to the heart failure, resulted from decreased pump myocardial function;

- *renal* (oedema renalis) due to the decreased oncotic pressure, hypoproteinemia, excessive secretion of aldosterone and increased reabsorption of water. Renal edema is observed in glomerulonephritis, nephrotic syndrome;
- *cachectic* (oedema cachecticum) – due to the starvation, cachexia, hypoproteunemia, decreased oncotic pressure in patients with oncology pathology;
- myxoedematous (oedema hypothireoidum) is caused by accumulation in the skin and subcutaneous tissue the specific substances, which contents mucopolysaccharide.

Local edema is a result of some local disorders in the blood or lymph circulation; inflammation; allergic process:

- *local congestive edema* is usually associated with thrombosis of the veins, compression of the veins by tumor or enlarged lymph nodes;
- *inflammatory edema* (oedema inflammatorium) is observed in erysipelas, rheumatic polyarthritis, rheumatoid arthritis;
- *angioneurotive edema* (oedema angioneuroticum) Quincke's edema as a result of allergic reaction.

If edema is generalized fluid may accumulate in the body's cavities: in the abdomen (*ascitis*), in pleural cavity (*hydrothorax*), in pericardium (*hydropericardium*).

General edema overspread throughout the entire body is named *anasarca.*

Subcutaneous fat depends on the sex, age, character of nutrition, functional condition of endocrine and nervous systems. In order to assess the degrees of subcutaneous fat you should take a fold of the skin and fat over Traube's cavity and measure the thickness.

In normosthenic person this size is 1.5–2 cm. The thickness of the skin wrinkle more than 2 cm reflects the excessive accumulation of subcutaneous fat, less than 1.5 cm – deficiency, and less than 0.5 cm is the sign of cachexia.

Excessive accumulation of fat in the cells and tissue is defined as *obesity* (adipositas).

The fat can be distributed in tissue uniformly or deposited in only certain parts of the body. According to the peculiarities of fat distribution two types of obesity are distinguished:

- *ginoid type* of obesity is characterized by uniformly fat distribution with more pronounced accumulation at the buttock and hip. Another terms of this type of obesity: peripheral, buttock-thigh;

– *android type* of obesity is characterized by accumulation of fat mainly at the upper part of the body, abdomen, and completely absence of fat at the buttock and legs. Another terms obesity: abdominal, central, male obesity.

There are additional criteria of the abdominal obesity: 1) waist to hip ratio more than 0.90 (male), more than 0.85 (female); 2) waist circumference more than 102 cm (male) and more than 88 cm (female).

The main reasons of obesity: overfeeding, hypodynamia, endocrine disorders of pituitary gland, sex glands (adiposogenital obesity), thyroid gland (hypothyroidism), adrenal gland (Cushing's syndrome).

Insufficient accumulation of fat may result from constitutional factor (asthenic type), diseases of digestive system.

Emaciation (macies) is divided into three groups: loss of weight (demetritio), disturbances of weight (dysthrophia) and sharp and excessive loss of weight (cachexia).

Pronounced loss of weight may occur in patients with prolonged intoxication, chronic infections, malignant tumor, some endocrine disorders (hyperthyroidism, diabetes mellitus, Addison's disease, hypophysial cachexia – Simmond's disease.

Lymph nodes may reveal during general inspection and using palpation. Regional lymph nodes include: occipital, auricular, posterior cervical, superior cervical, submandibular, supraclavicular, subclavicular, axillary, cubital, inguinal, popliteal. The examination of lymph nodes is performed by simple inspection and superficial palpation of the symmetrical region following the certain consequence: location, size, consistency, pain, mobility, and color of the skin over the lymph nodes. Normal lymph nodes cannot be detected visually or by palpation.

The main causes of the enlargement of the lymph nodes:

– infections (tuberculosis, AIDS, brucellosis, infectious mononucleosis, tularemia, plague;
– inflammation (local or generalized);
– diseases of the blood (leukemia, lymphogranulomatosis);
– lymphatic metastatic spread.

Diagnostic meaning of the pathological lymph nodes:

– inflammatory lymph nodes – different size, soft, elasticity, painful, with smooth surface, movable, reddening of the overlying skin. Cervical lymphadenit occurs in tuberculosis, which characterized by enlargements

of lymph nodes with purulent foci with subsequent formation of fistulae and immobile cicatrices;
- the diseases of the blood – systemic symmetrical enlargement of the peripheral lymph nodes and mediastinal, mesenterial ones. The nodes are firm and tender; their surface is rough, mobile, developed lymph nodes conglomerates. The nodes fuse together but do not separate;
- lymphatic metastatic spread – as a rules local enlargement of lymph node, which are hard, rough, palpation is painless;
- Virchov's gland (glandula Virchov's) – metastatic tumor in the supraclavicular lymph nodes, commonly at left in patient with cancer of stomach;

Tab. 2.8. ***Diagnostic meaning of enlarged lymph nodes localization.***

Localization	Diseases
Occipital	Measles erythema
Cervical	Chronic tonsillitis, tuberculosis, infectious mononucleosis, chronic lympholeucosis, lymphogranulomatosis, lymphosarcoma
Axillary	Purulent process at the hands, cancer of the breast
Inguinal	Purulent process at the leg, venereal disease
Cubital (local)	Syphilis

Muscular system. The main methods of examination are inspection and palpation. During examination of the muscular system doctor should assess: the level of development, sex and age correspondence, tenderness, muscular tonus, and evidence of cramps.

In normal condition the muscular system develops corresponding to sex and age, the muscular tonus is present, the muscles are painless, cramps and atrophy are absent.

Disorders of voluntary muscles include:
- muscular dystrophy;
- metabolic and endocrine myopathy;
- congenital myopathy;
- toxic myopathy;
- disorders of the neuromuscular junction.

Muscular dystrophy is a group of hereditary disorders characterized by progressive degeneration of a group of muscles with symmetrical wasting and weakness, tendon reflexes are preserved.

Metabolic causes of muscular weakness are hypokaliemia, hyperkaliemia, hypocalcaemia, and hypercalcaemia.

Endocrine causes of muscular weakness are hyperthyroidism, hypothyroidism, Cushing's syndrome, and Addison's disease.

In patients with chronic longstanding diseases (severe infections, intoxication, endocrine and oncology diseases) the generalized atrophy of the muscular system are observed. The atrophy may be revealed: 1) visually, by comparison the symmetrical muscular groups, 2) by antropometry with measurement the circumference of muscle of both extremities, and 3) by palpation with checking up the muscular tonus and strength. The forms of muscular tonus: hypotension, atony, hypertension, and muscular rigidity.

The local atrophy and atony occurs in patients with the disorders of nervous, muscular systems, with the diseases of connective tissues, joint and bones. The hypotonia and atony are observed in patients with progressive myopathy, myasthenia. In patients with Cushing's syndrome the loss of protein in muscle leads to a proximal myopathy (difficult or impossible to get up from a squatting position). Muscle weakness, proximal and bulbar myophaty, periodic paralysis is the clinical features of hyperthyroidism. Muscular occipital rigidity is characteristic sign of meningitis.

Cramps (spasmus) is defined as sharp involuntary spasms of muscles. The clonic and tetany (tonic) cramps are distinguished.

The tetany cramps (*spasmus tonicus*) is characterized by contraction of muscles (from some minutes till some hours and even days). There is an increased excitability of peripheral nerve due either to low serum calcium or to alkalosis. Magnesium depletion should also be considered as a possible contributing factor. Causes of tetany: due to the hypocalcaemia – malabsorption, osteomalacia, hypoparathyroidism, acute pancreatitis, chronic renal failure; due to alkalosis – repeated vomiting, hyperventilation, primary hyperaldosteronism. In children with deficiency of vitamin D is a reduction of ionized plasma calcium and infantile rickets (rachitic) tetany may result with spasm of the hands and feet and of the vocal cords. The cramps are observed in patients with meningitis (tetanus, opistotonus, risus sardonicus),

hypoparathyroidism ("obstetrician's hand", accotisher's hand, opistotonus, tetany, trismus, lockjaw, excessive excitability, "fish's mouth").

Tetanus – the most important early symptom is trismus – spasm of the masseter, which causes difficulty in opening the mouth and in masticating, hence the name "lock-jaw". This tonic rigidity spreads to involve the muscles of the- face, neck, and trunk. Contraction of the frontalis and the muscles at the angles of the mouth gives rise to the "risus sardonicus". There is rigidity of the muscles at the neck and trunk of varying degree. The back is usually slightly arched and there is a board-like abdominal wall.

The clonic cramps (*spasmus clonicus*) is characterized by fine tremor and observes in patients with hyperthyroidism (finger tremor), nervous diseases, and some poisoning.

Paralysis – loss of muscle ability to contraction. The complete paralysis (plegia) and incomplete paralysis (paresis) are distinguished. The reasons of the damages of motor centers in brain and spine, motor nerves due to the stroke, traumas, compression by the tumor. May be paralyses of one half of the body – spastic (central, pyramidal paralysis or hemiplegia), paralysis of upper extremities (upper paraplegia), paralysis of the upper and lower extremities (tetraplegia), paralysis of an extremity (monoplegia).

Bones system. The main methods of examination of bones system are inspection and palpation. The attention should be paid to the development of the skeleton, correspondence to the age and sex, the presence of visible deformities (fracture, curvature, enlargement). The palpation is useful for detection of insignificant deformity and tenderness.

Skeletal abnormalities are characterized by short or high stature and abnormal body proportion.

Short stature may be observed in the patient with impaired growth velocity due to the endocrine pathology: isolated growth hormone deficiency, panhypopituitarism (hypophysical dwarfism, hypophysial nanism, hypophysial infantilism), primary hypothyroidism (cretinism), and Cushing's syndrome. There are another conditions for short stature: chromosomal abnormalities (Cherechewski-Turner syndrome), malabsorption (coeliac, Chrohn's disease, colitis), systemic chronic illness (asthma, cardiac, renal disease), malnutrition, and psychosocial deprivation.

Tab. 2.9. ***The common causes of the bones system abnormalities***

Pathology	Examples
Endocrine pathology	Acromegaly, achondroplasia, hypopituitarism, hypothyroidism, Cushing's syndrome
Infections	Tuberculosis, syphilis
Hypo- and avitaminosis	Rickets (Rachitis)
Diseases of the blood system	Leukemia, congenital hemolytic anemia, lymphogranulomatosis, polycythaemia, multiple myeloma
Oncology	Sarcoma, metastatic spread
Congenital pathology	Deformity of the thorax, cleft lip, Cherechewski-Turner syndrome, Marfan's syndrome, Klinefelter's and Edwards syndrome, mucopolysaccharidoses, Ehlers-Danlos syndrom

High stature is cased by endocrine dysfunction (acromegaly) or chromosomal abnormalities (Marfan's syndrome, Kleinefelter's syndrome). As a rules the short or high stature are accompanied by abnormal body proportion and dysplasia.

Joints system. Examination of the joints is performed in such consequence: symmetrical joints of the upper extremities, symmetrical joints of the low extremities, head, neck and spine. Attention should be paid to their shape, configuration, swelling and hyperemia. Using palpation you should reveal possible pain, fluid in the join cavity, and crackle.

In clinical practice it is important to assess joints movements. Two kinds of movement are distinguished: active movement, which is fulfilled by patient according to the doctor's instruction and passive movement, which perform doctor. Simultaneous restriction active and passive movement suggests the affection of the joints (rheumatoid arthritis, rheumatic polyarthritis). Restriction of active, but preservation of passive movements is observed in patients with coma and during disorders of local joint circulation and innervation.

Affection of the **joints** develops due to the **inflammatory diseases** (tuberculosis, gonorrhea, rheumatic fever, rheumatic arthritis), metabolic disorders of uric acid (gout), and psoriasis.

The *arthritis of rheumatic fever* is often symmetrical, affecting large joints with acute painful inflammation, which characteristically "flits" from joint to joint (a migratory polyarthralgia). The joints affected include those of the limbs, spine, and sometimes temporomandibular and costoclavicular joints.

In the *rheumatoid arthritis* appears the pain on movement of joints, morning stiffness and symmetrical swelling of small joints of the fingers and the toes. Swelling of the proximal, but not the distal interphalangeal joints gives the finger a "spindled" appearance. As the disease progresses there is tendency for it to spread to involve the wrists, elbows, shoulders, knees, and ankles. The hip joints become involved only in the more severely affection, but neck pain and stiffness from cervical spine diseases is common. As the disease advances, muscle atrophy, tendon sheath and joints destruction result in limitation of joint motion, subluxation and deformities.

Gonoccocal arthritis damage commonly knee in a form of monoarthritis. Sometimes may be polyarthritis with asymmetrical joint involvement and acute, or subacute emigratory polyarthralgia.

Tuberculosis of the joints is usually secondary to an established focus in the lungs or kidneys. Clinical features include pain, stiffness, swelling and restriction of movement of single large joint associated with anorexia, weight loss and night sweat.

Syphilitic arthritis may be associated with painful paraarticular swelling due to the epiphyseal involvement soon after birth – congenital syphilis. Acquired secondary syphilis may be referred to the migrating polyarthralgia.

Brucellosis is associated with polyarthralgia or transient polyarthritis. Chronic bursitis and osteomyelitis may also observe.

Systemic lupus erythematosus leads to the transient and migratory or a more persistent polyarthritis. Chronic inflammatory arthritis and tendosynovitis may cause deformities and contractures.

Psoriatic arthritis is a secondary inflammatory arthritis found in patients with psoriasis. An inflammatory arthritis affecting the distal interphalangeal joints is the most characteristic form of psoriatic arthropathy and associated with nail changes.

Reiter's disease – is the triad of non-specific urethritis, conjunctivitis and arthritis that follows bacterial dysentery or exposure to sexually transmitted infections. The clinical features include the affection of large or small joints of the lower limbs, sometimes monoarthritis of a knee or an asymmetrical inflammatory arthritis of some interphalangeal joints. The skin lesions can vary from faint muscles, vesicles and pustules on the hands and feet to marked hyperkeratosis with plague – like lesions spreading to the scalp and trunk. These may be associated with severe nail dystrophy and massive hyperkeratosis.

Examination of the spine

In normal condition there are four physiological curvature of the spine:

- cervical curvature of the spine column with forward convexity – lordosis;
- thoracic backward curvature of the spinal column – kyphosis;
- lumbar forward curvature – lordosis;
- pelvic kyphosis.

The deformity of the spine is observed as pathological *lordosis*, pathological *kyphosis*, lateral curvature of the spinal column – *scoliosis*, and curvature of the spinal column with lateral and backward convexity – *kyphoscoliosis*.

The form of spine is defined by constitution, gait, muscles development and depend on environmental factors.

Head. During inspection of the head you should pay attention on the size, shape, position, movement and state of eyes, nose, mouth, tongue, and teeth.

Head size may be normal, enlarged (macrochephalia) and small (microcephalia). Microcephalia reflects small size of brain and cerebral skull.

Macrocephalia is observed in such pathological state:

- hydrocephalia (hydrocephalus) – inherited disorder characterized by enlargement of the head, dilation of the brain ventricles due to the disturbance flowing away the spinebrain fluid and increasing of intracranial pressure resulted in neurological symptoms and mental retardation;
- achondroplasia is characterized by large skull with prominent back of the head, disproportion of brain and facial parts of skull, nose as a saddle, short stature, dwarf, disproportion of the trunk due to the short limbs, cervical lordosis;
- cerebral gigantism (syndrom sotos) may reveal after birth. The newborn has great weight and length (more than 3.9 kg and 55 cm) and accelerated growth

in first years of life. Acromegalic features – macrocephalia, with prominent frontal hillock, enlarged foot and hand, scoliosis, and syndactilia;

- toxoplasmosis syndrome occurs in case of evidence of infection at mother and transmission it to the child during development of pregnancy. Congenital toxoplasmosis is characterized by hydrocephalia, sometimes microcephalia, affection of eyes, cyanosis, jaundice, and hepatosplenomegalia.

Microcephalia – small head, brain weight, mental retardation. There are neurological disorders: athonia, spastic paralysis, cramps. Two forms of microcephalia are distinguish: primary with autosomal-recessive inheritance and secondary due to the organic brain damage different etiology (birth trauma, toxoplasmosis, cytomegalia, intrauterus hypoxia, exposure of radiation on fetus). Microcephalia is often accompanied the gene and chromosomal syndrome:

Head Shape

A "square" head with "bossing" of the frontal and parietal bones and delayed closure of the anterior fontanelle in childhood is characteristic of rickets. May be craniotabes – small round unossified areas in the membranous bones of the skull.

"Bossing" of the skull, prominent malar bones and protuberant teeth are development in sickle-cell anemia.

A head bossing due to the bone marrow hyperplasia appears in child with beta-thalassemia. The skull radiograph shows a "hair on end" appearance and general widening of the medullary spaces, which may interfere with the development of the paranasal sinuses;

A "triangle" head develops due to the intrauterus craniosynostosis, closing the skull fissura, elevation of intracerebral pressure. The characteristically features: high forehead, exophtalmus, ptosis, and nose as beak;

Local osteomalacia of skull bone is characteristic of multiple myeloma (disease of blood);

A "square" head, flattened on top, with prominent frontal tubers, can indicate congenital syphilis.

Acrocephalia – tower-like shape of skull accompanied with polydactily and syndactily is the feature of the congenital pathology – acrocephalopolysandactyly (Carpenter's syndrome).

Craniosynestosis – premature closing of skull fissure limited the skull growing, resulted to bone deformity.

Position of the head depends on state of spine and nervous system:

- immovable head is observed at ankylosing spondyloarthritis, Bekhterev-Strumpal-Mari's disease, verterbal osteochondrosis, myositis, myopathy, fusion of cervical vertebral (Klippel-Fell's disease cervical ribs);
- torticollis (stiff-neck) – turning head in one side may be acquired and occur due to the myositis. More commonly the reason are congenital muscular torticollis (Grisel's disease), spastic torticollis;
- neck stiffness with head throw back is specific for meningitis or tetanus.

***Movement of the head*:**

Involuntary shaking head associated with tremor of the hands occurs at patients with Parkinson's disease;

Non-rhythmic tremor of the head is the symptom of chorea, st. Vitus's dance;

Shaking head synchronous with heart function (with pulse wave a head throw back) named as Musset's sign is observed at aortic regurgitation

Eyes

Inspection of the eyes can reveal some essential diagnostic signs. It is necessary to exam eyelids, eye slit, eyeball, sclera, cornea of the eye, pupils.

Eyelids:

- swellowing and pigmentation is the sign of dermatomyositis;
- edema of the eyelids is the first indication of the nephritis;
- narrowing of the eye slit occurs in myxedema and general edema (anasarca);
- dark eyelids are the characteristic of Addison's disease and diffuse toxic goiter;
- xanthomas at eyelids indicate deranged cholesterol metabolism – atherosclerosis, liver cirrosis, cholestatic jaundice;
- persistent drooping of the upper eyelids (ptosis) may be congenital or acquired origin. Ptosis congenital has autosomal dominant inheritance, often one-side character. Sometimes ptosis is one of the multiple symptoms congenital etiology – myotonic dystrophy, Roberts syndrome, ptosis intermittent character or diplopia – the symptom of myastenia gravis. Acquired ptosis observes at botulism, syphilis, hemiparesis. Unilateral ptosis accompanied with recession of the eyeball (enophtalmos), pupil narrowing, described as Klod-Bernard-Horner syndrome observe at affection of cervical pupil sympathetic innervatiom part sympathetic spinal

tract. Lagophtalmus – incomplete closing of eyelids is the sign of congenital pathology.

Eye slit depends on the eyeball and eyelid position. Narrowing of the eye slit may be observed in acute glomerulonephritis, Quincke's edema, myxoedema, peritonitis, and congenital pathology. Widening of the eye slit may be observed in thyrotoxicosis, retrabulbar abscesses. Asymmetry of the eye slit may be at unilateral ptosis, tumor of the brain.

Hyperthelorism – diminished distance between auter angle of eyehole.

Epicanthus (canthus the angle of the eye slit) – vertical skin fold at the outer angle of the eye slit. Hyperthelorism, and epicantus are the signs of congenital pathology – craniocarpotarsal dysplasia, wich characterized by facial abnormalities: epicanthus, hypothelorism, enophtalmus, squint, small nose, deviation of finger. *Telecantus* – lateral deviation of the eye slit.

Sclera, conjunctiva and cornea of the eye

- blue sclera or syndrome of "blue eyes" may observed at congenital pathology: ostegenesis imperfacta ("blue eyes", fragile bones and multiple fracture of limbs, ribs and clavicles), focal dermal hypoplasia (thin partial skin, "blue eyes"), Rieger syndrome ("blue eyes", aniridia, kataracta, strabismus);
- yellow sclera is early sign of jaundice;
- bleeding into the conjunctive and sclera at bacterial endocarditis, epilepsy, deficiency of vitamin C;
- red "as a rabbit" conjunctivae ocular injection at typhus;
- single brown spot at conjunctiva may observe at Addison's disease.

Pupil: examination of the size, shape, reaction to convergence, light, accommodation.

Myosis – papillary constriction is observed in uremia, intracranial hemorrhages, brain tumor, neurosyphilis, typhus, chronic poisoning. In persons with morphine abuse – point-like pupil are typical.

Mydriasis– papillary dilation is observed in patients with coma (except uremic and apoplectic), syphilis, sometimes at aortic aneurysm.

Anisocoria – asymmetrical pupils is observed in syphilis, Argyll Robertson's syndrome.

Pupilla pulsation of the pupil in synchronism with the cardiac beat and pulse (Landolphys symptom) is characteristic of aortic regurgitation.

Reaction to light is characterized by constriction of the pupil during exposure of light, and pupil dilation during absence of light. Reaction pupil to light is useful for diagnostics of nervous system damage, syphilis, unconsciousness, poisoning of atropine.

Papillary abnormalities, described by Argyll Robertson's may accompany any neurosyphilitic syndrome. The pupils are small and irregular, and may react to convergence but not directly to light.

Mouth

When inspecting the mouth attention should be paid to its size, shape, symmetry of the angles, forms and color of the lips and mouth mucosa. It is necessary to exam teeth, tongue, and gums.

Mouth shape in pathological condition may be in a form of macrostomia and microstomia.

Macrostomia is a result of congenital pathology. Microstomia has inhereted origin and may be acquired (mouth in patients with sclerodermia and hypoparathyroidism).

Asymmetry of the mouth angles observed in local inflammatory process and in patients with lesions, affecting the trygeminus system; paralyses of the facial nerve; stroke with such clinical feature: unilateral loss of nasolabial fold, mouth angle is lowered on the affected side and saliva may droop from it.

Constant half-open mouth is observed in acromegaly, Daun's disease, congenital hypothyroidism, severe dyspnea, and paralyses of the facial nerve.

Contracted lips and closed mouth are the characteristic of exlampsia, tetanus, and rabies.

"Risus sardonicus" with a semblance of a grin occurs in tetanus patients: the mouth widens as in laughter while the skin folds on the forehead express grief.

Lips

Color of the lips

Cyanotic color of the lips is characteristic of mitral stenosis, heart failure.

Hyperemic lips observe in high temperature, inflammatory processes.

Pale lips occur in acute and chronic bleeding, oncological process, leukemia, hypo- and aplastic anemia.

Cavity of the mouth

When inspecting the cavity of the mouth attention should be paid to the color of the mucosa, presence of eruption, ulcer, erosions, and form of palate.

Color of the mucous membrane

Yellowish color is characteristic of true jaundice; appearance of yellowish soft palate precedes the yellowish sclera and is an early sign of virus hepatitis.

Pale color with swelling is the specific sign of anemia different etiology.

Hyperemic mucous membrane with swelling is characteristic of stomatitis.

Koplik's spot (Koplik's enanthema) appear at the second day in catarrhal stage of measles. Koplik's spots are small white on the mucous membrane of the mouth surrounded by a narrow zone of inflammation.

Single brown spots are characteristics of Addison's disease.

Hemorrhages, aphthae, ulcers, herpes labials, sore throats are common signs of acute leukemia.

Hemorrhage is observed in patients with congenital or acquired bleeding disorders.

Aphtous ulceration characterized by superficial ulcers in the mouth, which are often multiple. Severe aphtous ulceration may be in association with Crohn's disease, ulcerative colitis, and celiac disease.

Candidosis is a fungal infection and classified either as superficial (skin or mucous membrane) or systemic. In mouth appears white patches on tongue and buccal mucosa, and may enlarge and coalesce to form an easily detached membrane; there is a little surrounding inflammation.

Gum, gingiva

Marked changes in the gums can be observed in some disease and poisoning.

Ulcerative-necrotic gingivitis with hemorrhage from the mouth may be in leukemia.

Inflammation of gingival mucous membrane, stomatorrhagia, painful gums are observed in hypovitaminosis C – scorbutic gingivitis.

Mercuric gingivitis is characterized by desquamate and atrophic processes of the mucous membrane.

Diabetic gingivitis is diffuse inflammation with gangrenous complications.

Lead gingivitis is characterized by blue punctate line on the gum margins adjoin the teeth and injury of the gum.

Tongue

In health the tongue is moist with only slight white fur on the dorsum. The papillae are readily seen.

With inspecting the tongue attention should be paid to its shape and size, surface, movement, color, and the state of papillae.

Shape and size

Macroglossia – enlargement of the tongue is a sign of congenital pathology (Daun's disease; Beckwith-Wiedeman syndrome – macroglossia, visceromegalia, ompalocele; glycogenosi type II – macroglossia, cardiomegalia, myotonia; cerebral gigantism). Macroglossia appears on development of acromegaly, hypothyroidism. Moderate macroglossia may appear in the patients with fever, gastrointestinal diseases.

Microglossia – decreased tongue observe in the patients with cholera, typhus, starvation, and vitamin B_{12}-deficiency.

Unilateral atrophia of the tongue occurs at pyramidal tract lesion.

Flat tongue due to the atrophy of it base resulted from ulcerative stomatitis and scarification of the soft palate and pharynx in secondary syphilis.

Fur of the tongue in pathological conditions has diagnostic significance:

- Coated (furred) tongue is observed at gastritis, peritonitis, colitis, fever, infections (hepatitis), and pneumonia;
- Coated in the center and at the base but clear the tip and margins of the tongue is typical to typhoid fever. Additional diagnostic meaning is a fur character and color:
- White fur observe at typhus, pneumonia and peritonitis;
- White-gray fur observe at gastritis, virus hepatitis, and some infections;
- White-dirty or yellow fur – at peritonitis;
- White-blue – in rheumatic polyarthritis;
- White-yellow fur in those who smoke excessively;
- Crimson-red (strawberry/raspberry) tongue observe in scarlet fever;
- Dry tongue is an indication of dehydration with followed formation of erosion hemorrhage and observed in peritonitis, and severe infections. Dryness of the mouth (xerostomia) may be caused by anticholinergic or antidepressant drugs; bur commonly it is due to anxiety.
- Glossitis may be a prominent feature of stomatitis resulting from nutrition deficiency and overdose of antibiotics.

Surface of the tongue

Atrophy of tongue papilla cause smooth (as if polished) crimson tongue, Hunter's glossitis, which may observe in the patients with B_{12}-deficiency anemia.

The glassy tongue is characteristic of gastric cancer, pellagra, and sprue.

The local thickening of the tongue with chronic migrating superficial glossitis named as geographical tongue is found in the patients with hyperacidity of gastric juice.

Grooved (fluted) tongue, with multiple wrinkles is characteristic of acromegaly.

There are some patches, ulceration at the mouth.

Leucoplakia is white, firm, smooth patches beginning at the side of the tongue and later spreading over the dorsum. In the early stages the tongue is not painful but later fissures split the patches with tenderness. Hairy leucoplakia occurs in AIDS.

Syphilis may present as a painful solitary ulcer usually on the tongue or palate.

Neck

During inspection of the neck attention should be paid to the shape and size, symmetry, skin color, presence of scars and visible pulsation.

Changing of *neck shape and size* depend on constitutional type, the state of lymph nodes, thyroid gland, cervical column and muscles development.

Short thick neck is observed in hypersthenic persons. In pathological condition such neck may be in patients with lung emphysema, obesity, hypothyreoidism, Cushing's disease, and pronounced enlargement of thyroid gland.

Long slim neck with prominent cartilage is observed in asthenic persons. In pathology suck neck may be in patients with disorders of pituitary (sex) gland, starvation, cachexia.

Disorders of head movement are due to the osteochondrosis of cervical column, rigidity of neck (occipital) muscles, presence of scars, enlargement of lymph nodes, thyroid gland, appearance of tumor.

Skin color changes at the neck region can indicates such pathology:

- pigmentation with outlined border is observed in Addison's disease;
- multiple pound white sport (syphilitic leucoderma) as a neclace (collar Veneris) in syphilis;
- scars at neck indicate the previous tuberculous lymphadenitis.

Prerugium-syndrom – a skin fold placed on the side neck surface from mastoid process is a sign of Shereshevsky-Turner syndrome.

Pulsation of the carotid artery (carotid shudder, saltus carotidum) appears due to the changing of blood pressure and filling of arteries during systole and diastole in patients with aortic regurgitation, hyperthyroidism, and fever.

Swollen and pulsation of jugular veins is explained by difficulty of blood flow to right atrium in tricuspid regurgitation, pericarditis, and chronic lung diseases.

Thyroid gland placed on anterior surface of cricoid cartilage. In normal condition in healthy persons thyroid gland is impalpable. The thyroid isthmus is often but not always palpable. The lobes are more lateral, than isthmus and harder to feel.

Goiter is a general term for an enlarged thyroid gland and observed in Basedovica disease (hyperthyroidism), autoimmune thyroiditis (Hashimoto goiter) and endemic goiter.

The diffuse enlargement of thyroid gland is observed in hyperthyroidism; asymmetric separate nodes with unequal surface characterize cancer of the thyroid gland.

Palpation

Palpation (L *palpare* – to touch gently) is the method of clinical examination, which is known from the ancient time. Despite the wide use of modern instrumental methods of examination, palpation remains one of the main and important technique of the internal organs diseases diagnostics.

Palpation is used to determine elasticity and dryness of the skin, to assess condition of the subcutaneous fat, to detect edema. The size, consistency, tenderness, and mobility of the peripheral lymph nodes it is possible to determine by the help of the palpation. Muscles development, the size of the joints, their tenderness or possible swelling, deformities, presence of fluid in the articular capsule can also be revealed by palpation.

Palpation is widely used in examination of the chest to detect its elasticity, tenderness, and vocal fremitus.

Apex beat location, and its properties; presence of the chest thrill ('cat's purr' symptom) can be detected in palpation of the precordium.

Palpation is very important in examination of the abdominal organs: intestine, liver, gall bladder, spleen, etc.

Palpation technique differs depend on object and task of the examination. Surface and deep palpation are differentiated. Surface or tentative palpation is done by the tip of the fingers very gently in order to reveal tenderness, muscular strain of the abdomen, for example. Deep or sliding palpation developed by Obraztsov-Strazhesko is used to examine deep located abdominal organs. Penetrative palpation is a variant of the deep one. Palpation can be done by one hand, or by two hands – so-called bimanual palpation.

Palpation technique of the chest, the heart region, abdomen, kidneys will be described in detail in relevant chapters.

Percussion

Percussion (L *percutere* – to strike through) is method of physical examination, which was proposed by an Austrian physician Leopold Auenbrugger in 1761.

Tapping various part of the body sets underlying tissues into motion, producing audible sounds. Percussion helps to determine whether the underlying tissues are air-filled, fluid-filled, or solid.

Technique of the percussion for the right-handed person. Immediate and mediate percussion are distinguished. In immediate percussion, proposed by Obraztsov, the examined surface is tapped by plexor (hammer) – the tip of the index finger of the right hand. The obtained sound is not intense, therefore, the index finger may be first held by the side of the middle finger and then release to make sound louder. This method is rare used today.

The sound obtained in mediate percussion is loud and distinct. The key points of the mediate percussion are following:

1. Place your left hand on the examined surface;
2. Press the second phalanx of the middle finger (the pleximeter finger) tightly on the surface to be percussed;
3. Slightly flex middle finger of your right hand (the plexor finger);
4. With a quick and short wrist motion (without involving the forearm), strike the pleximeter finger with the plexor finger;
5. Striking intensity should be uniform, and directed perpendicularly to the pleximeter finger. (A short fingernail is required).

Loud, light, and lightest percussion are differentiated. *Loud* percussion is used to examine deep located organs (the vibrations reach a depth of 4–7 cm), *light* percussion – for examining superficial organs, their size and borders (the vibrations reach a depth of 2–4 cm). The *lightest* percussion technique is used to outline the borders of the absolute cardiac dullness.

Comparative and topographic percussion are distinguished. The aim of comparative percussion is to compare the sounds on the symmetrical parts of the body. Topographic percussion is used to detect the borders, size, and shape of the internal organs.

Auscultation

Auscultation (L *auscultare* – to listen) means listening the sound inside the body. Auscultation was first proposed by French physician Laënnec in 1816; in 1819 this method was described and introduced into medical practice. Laënnec also proposed an instrument, called stethoscope that uses for auscultation. He described and named almost all auscultative sounds: bronchial and vesicular breathing sounds, crepitation, heart murmurs, and confirmed clinical significance of auscultation by checking its results during section.

Auscultation can be direct – by ear, or indirect – using stethoscope. The binaural stethoscope is now widely used. It is closed acoustic system where air serves as a transmitting medium for sounds. Phonendoscope differ from simple stethoscope is that it have a membrane covering the bell, which intensifies auscultative sounds.

Auscultation is one of the most important diagnostic technique for examining the lungs, heart and vessels, blood pressure measuring by Korotkoff's method. Auscultation is used in obstetrical practice, for the study of digestive organs.

The key points of auscultation are following:

1. The room should be silent and warm;
2. The patient should be undressed (clothing produce additional friction);
3. The skin should be hairless (hair produce additional friction) or hair may be wetted with water;
4. The bell of the stethoscope should held the thumb the forefinger, and pressed firmly and uniformly to the patient's skin (however, not too strong);
5. The posture of the patient is different that depend on the examined organs.

SPECIAL PART

Chapter 3. RESPIRATORY SYSTEM

FUNCTIONAL AND CLINICAL ANATOMY

The *respiratory organs* consist of the **upper respiratory tract**: which includes the nose, pharynx, larynx, and the **lower respiratory tract** – trachea, bronchi, and lungs (Fig.3.1).

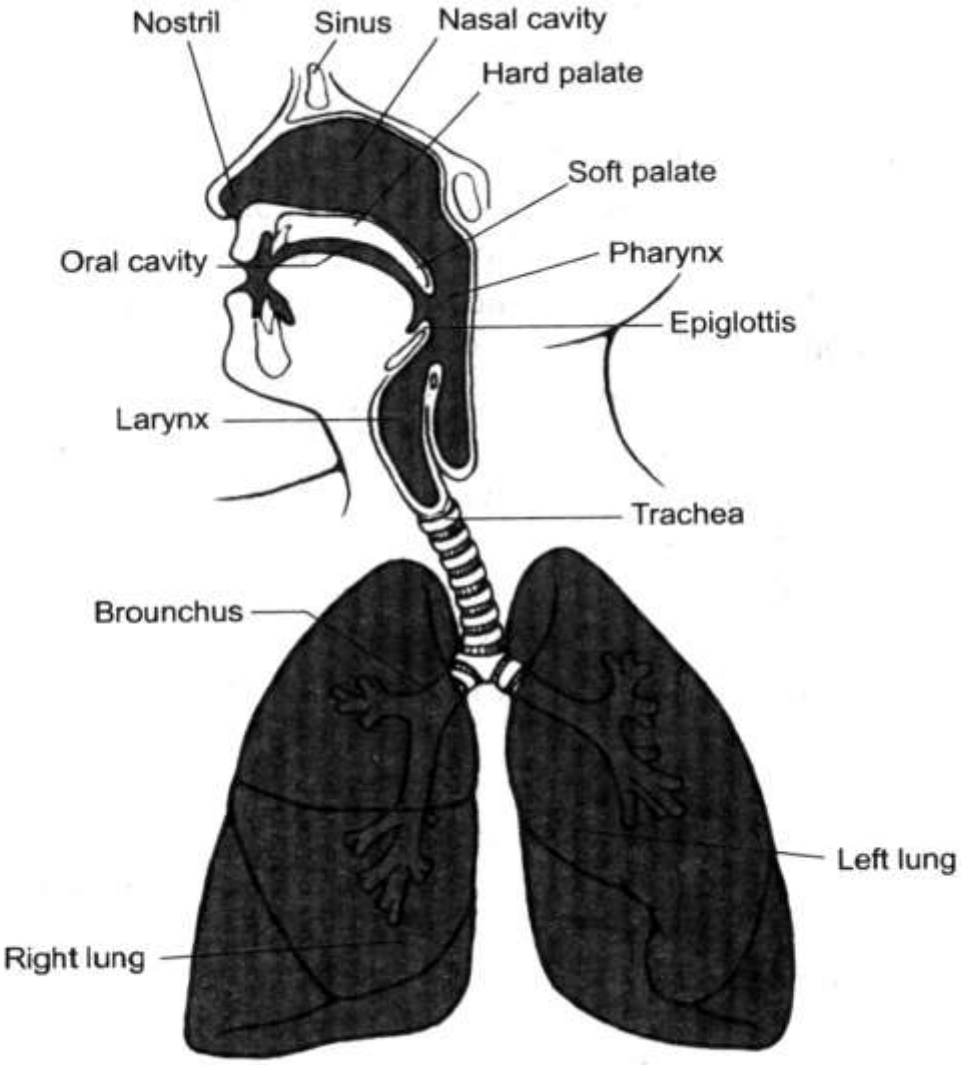


Fig. 3.1. Respiratory organs.

The **nose** consist of the **external nose** – the projection on the face with two openings (nostrils or anterior nares) – and the **nasal cavity,** which is the internal part which is divided into right and left halves by the nasal septum. Each half of the nasal cavity communicates with the **paranasal air sinuses** via small opening, and by a large opening at the back (the posterior naris or choana) with the pharynx.

The **pharynx** is a muscular tube about 12 cm long, which extends down from the base of the skull, consisting of nasal (the **nasopharynx**), oral (the **oropharynx**), and laryngeal parts (the **laryngopharynx**). The nasal part, into which the nasal cavities open, belongs exclusively to the respiratory tract, but the oral and laryngeal parts, commonly called the throat, belong to both the respiratory and alimentary tracts.

At its lower end, the **laryngopharynx** opens into two structures: the larynx anteriorly, and the oesophagus directly below. The **larynx**, which is the organ of speech, opens off from the front of the laryngopharynx and contains the **vocal folds** (or **vocal cords**) whose movements produce sounds.

At the level of C6 vertebra, the lower end of the larynx continues into **trachea** (**windpipe**), which passes from the neck into the upper thorax. It is muscular tube about 10 cm long, who's contain U-shaped strips of cartilage (called "rings"), which keep lumen continuously open.

Just below the level of T4 vertebra, the trachea divides into the right and left main or **principal bronchi**, which enter the lungs.

The **left lung** is divided into *two* **lobes** (upper and lower) by an oblique fissure, and is somewhat smaller than the right lung because the heart bulges towards the left.

The **right lung** is divided into *three* **lobes** (upper, middle, and lower) by oblique and transverse fissures.

Within the lungs, the main bronchi divide to form **lobar bronchi,** one for each lobe, and each in turn divides into **segmental bronchi.** Further subdivision results in a profusion of smaller tubes (**bronchioles**) that eventually open into the microscopically small air sacs (**alveoli**) through whose thin walls gaseous exchange can occur with the plasma and red cells of the blood in adjacent capillaries (Fig. 3.2). Due to its extensive branching pattern, the system of bronchi and bronchioles within each lung if often referred to as the **bronchial tree.**

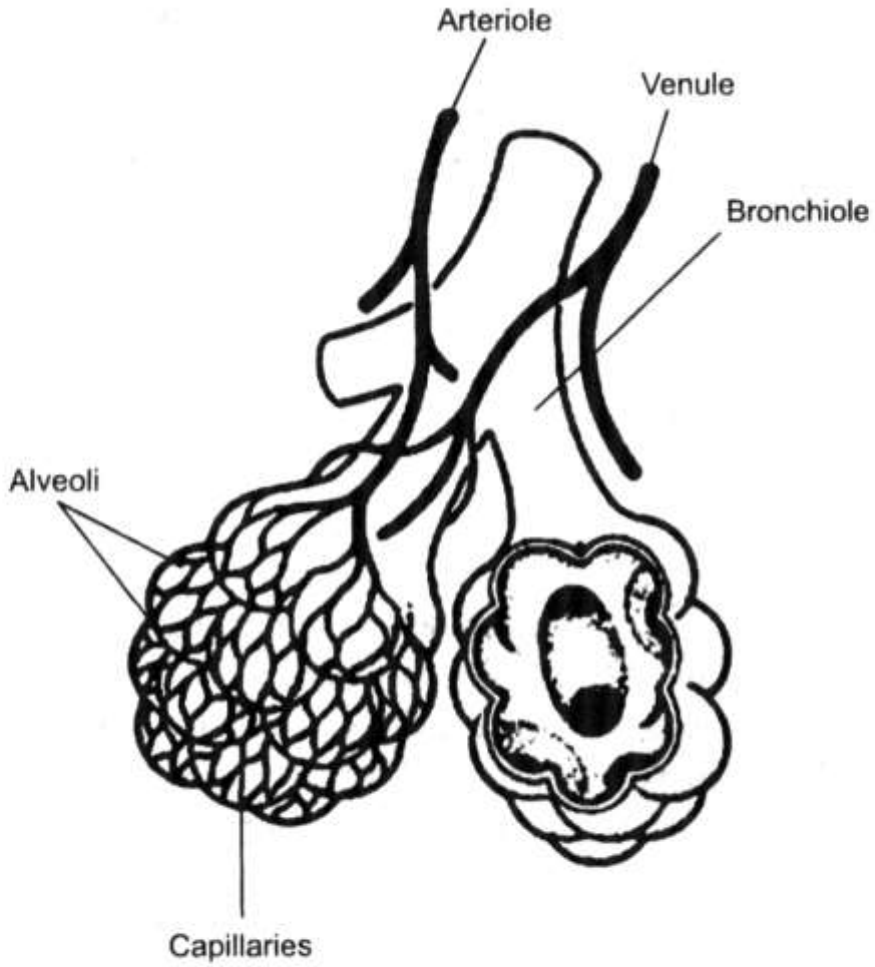


Fig. 3.2. Diagrammatic representation of bronchioles, alveoli and blood capillaries.

Topographic regions and lines of the chest. To describe an abnormality on the chest, you need to locate it in two dimensions: along vertical axis and around the circumference of the chest.

To locate vertically, you must be able to number the ribs and interspaces accurately (Fig. 3.3).

Note that an interspace between two ribs is numbered by the rib above it.

As the 1st rib is covered by clavicle, the 1st interspace is below it. From here, using two fingers, you can "walk down the interspaces". Do not try to count interspaces along the lower edge of the sternum; the ribs here are too close together. Note that the costal cartilages of only the first seven ribs articulate with the sternum. Those of the 8th, 9th, and 10th ribs articulate

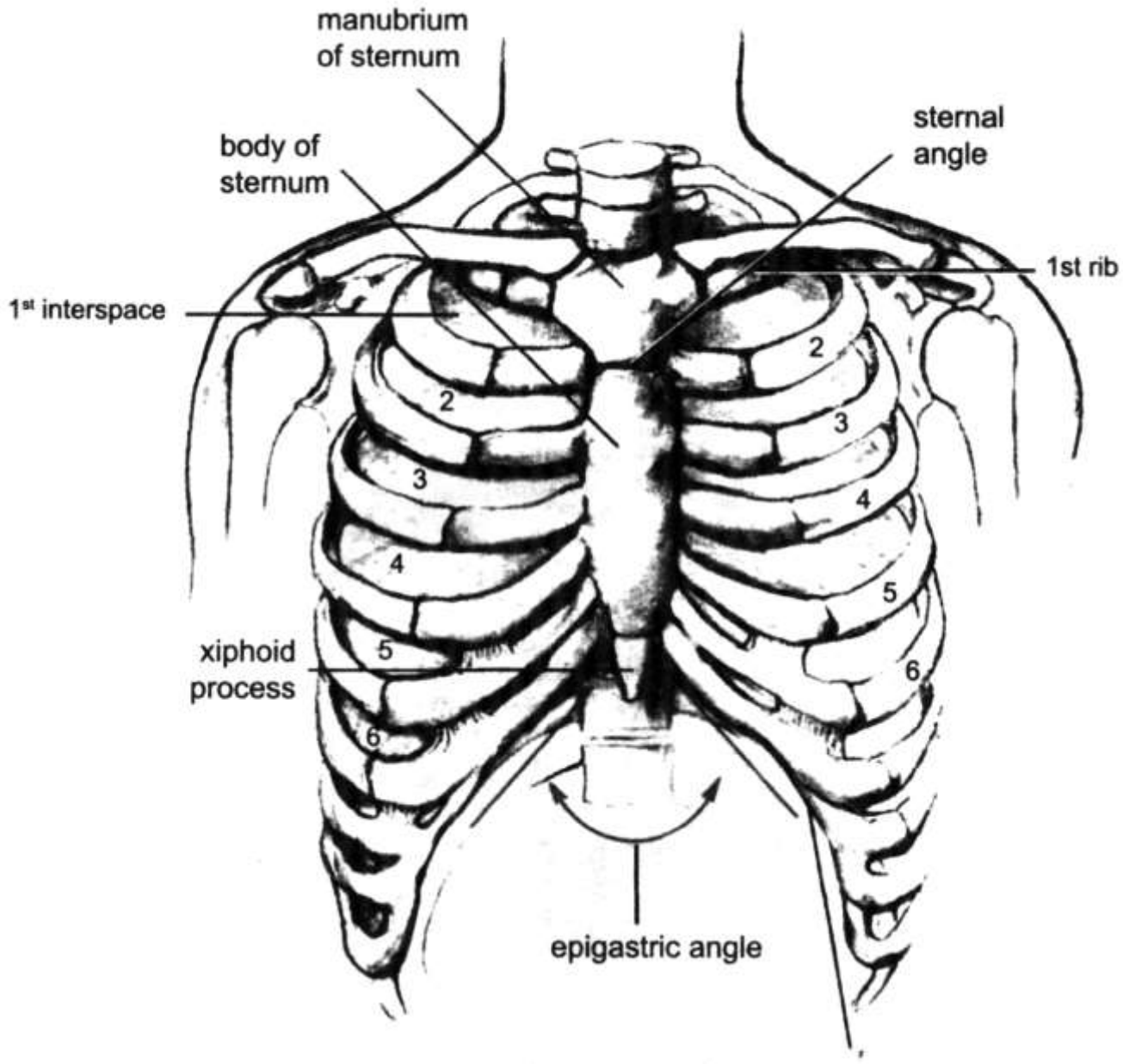


Fig. 3.3. Anatomy of the chest wall.
Anterior view.

instead with the costal cartilages just above t hem. The 11th and 12th ribs, the so-called floating ribs, have no anterior attachments. The cartilaginous tip of the 11th rib can usually be felt laterally, and the 12th rib may be felt posteriorly. Costal cartilages are not distinguishable from ribs by palpation.

When estimating location posteriorly, remember that the inferior angle of the scapula usually lies at the level of the 7th rib or interspace (Fig. 3.4.).

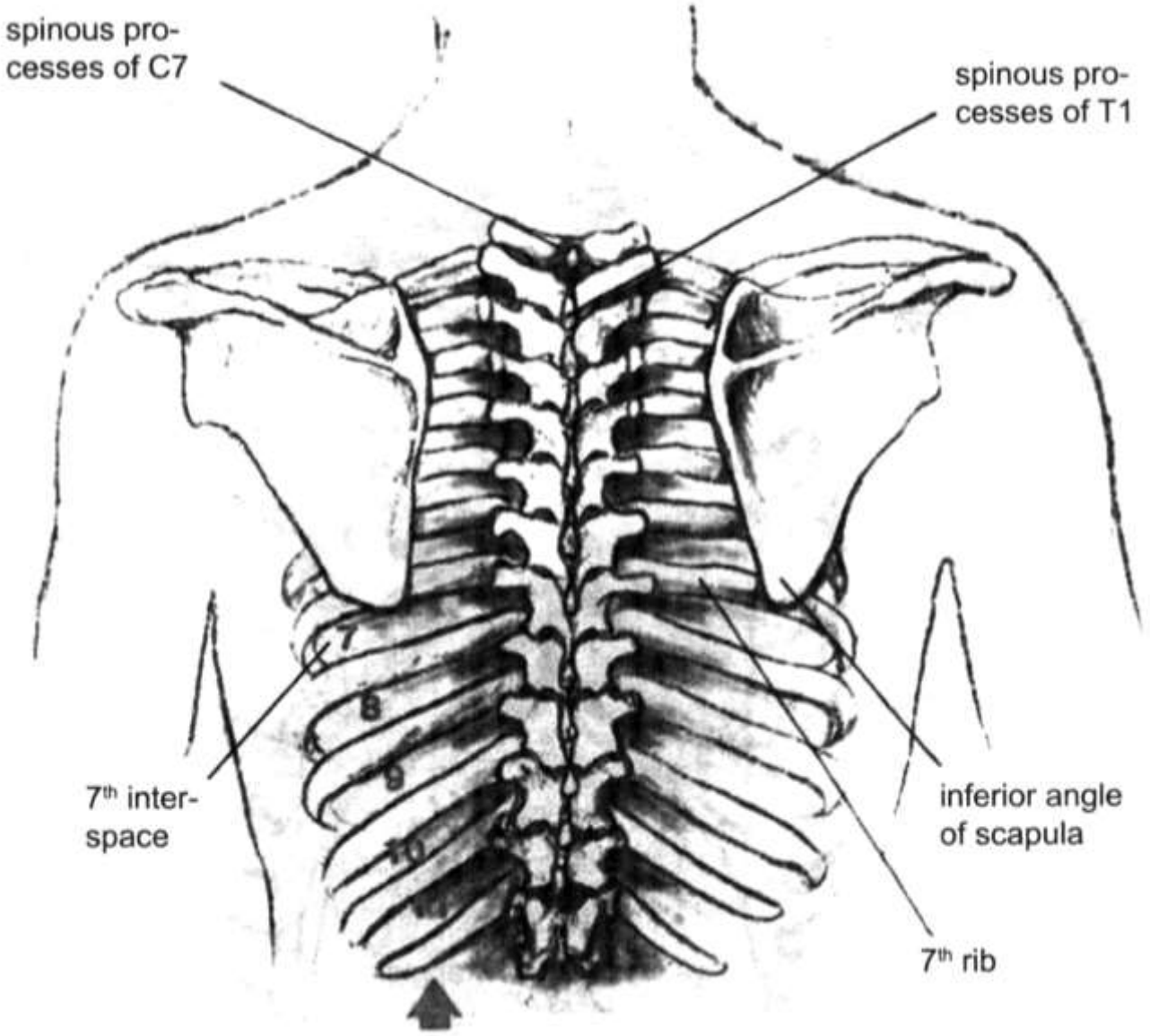


Fig. 3.4. Anatomy of the chest wall. Posterior view.

Findings may also be located according to their relationship to the spinous processes of the vertebrae. When a patient flexes the neck forward, the prominent process is usually that of the 7th cervical. When two processes equally prominent, they are the 7th cervical and 1st thoracic. The processes below them can often be felt and counted, especially when the spine is flexed. The 12th rib gives you another possible starting point for counting the ribs and interspaces. This especially useful in locating findings on the lower posterior chest.

To locate findings around the circumference of the chest, use a topographic vertical lines. The median (or midsternal) and vertebral lines are precise; the others are estimated. The midclavicular line drops vertically from the midpoint of the clavicle. To find it, you must identify both ends of the clavicle accurately (Fig. 3.5).

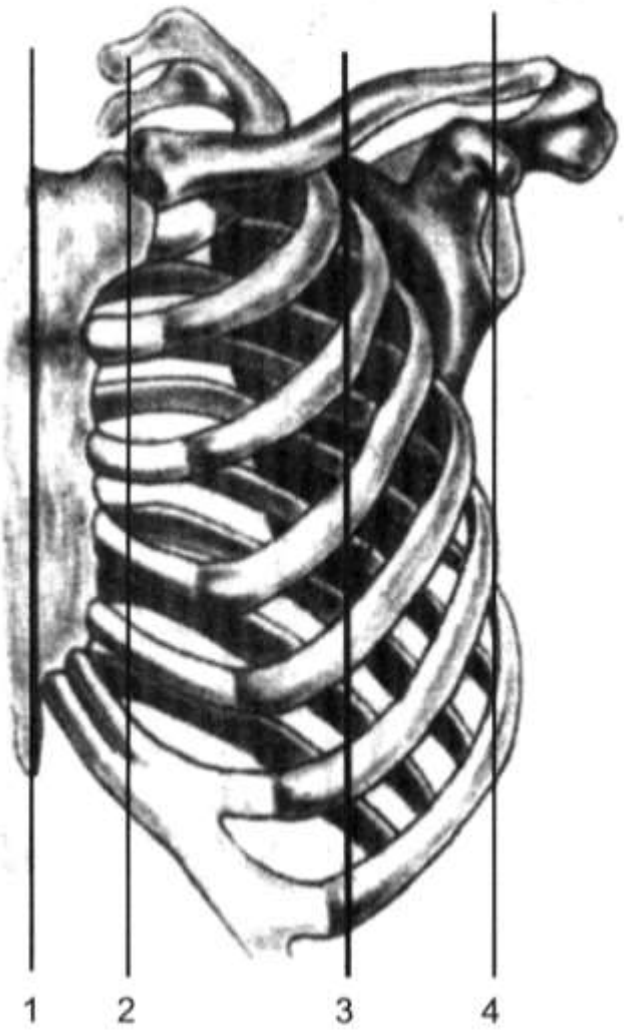


Fig. 3.5. Topographic lines.
Anterior view.
1 – median line, 2 – left parasternal line, 3 – left midclavicular line,
4 – left anterior axillary line

The anterior and posterior axillary lines drop vertically from the anterior and posterior axillary folds (the muscles that border the axilla). The midaxillary line drops from the apex of axilla (Fig. 3.6).

Fig. 3.6. Topographic lines.
Right anterior oblique view.
1 – midaxillary line, 2 – anterior axillary line,
3 – posterior axillary line

Posteriorly, the vertebral line follows the spinal processes of the vertebra. Paraspinal lines drop along vertebra; each scapular line drops from the inferior angle of the scapular (Fig. 3.7).

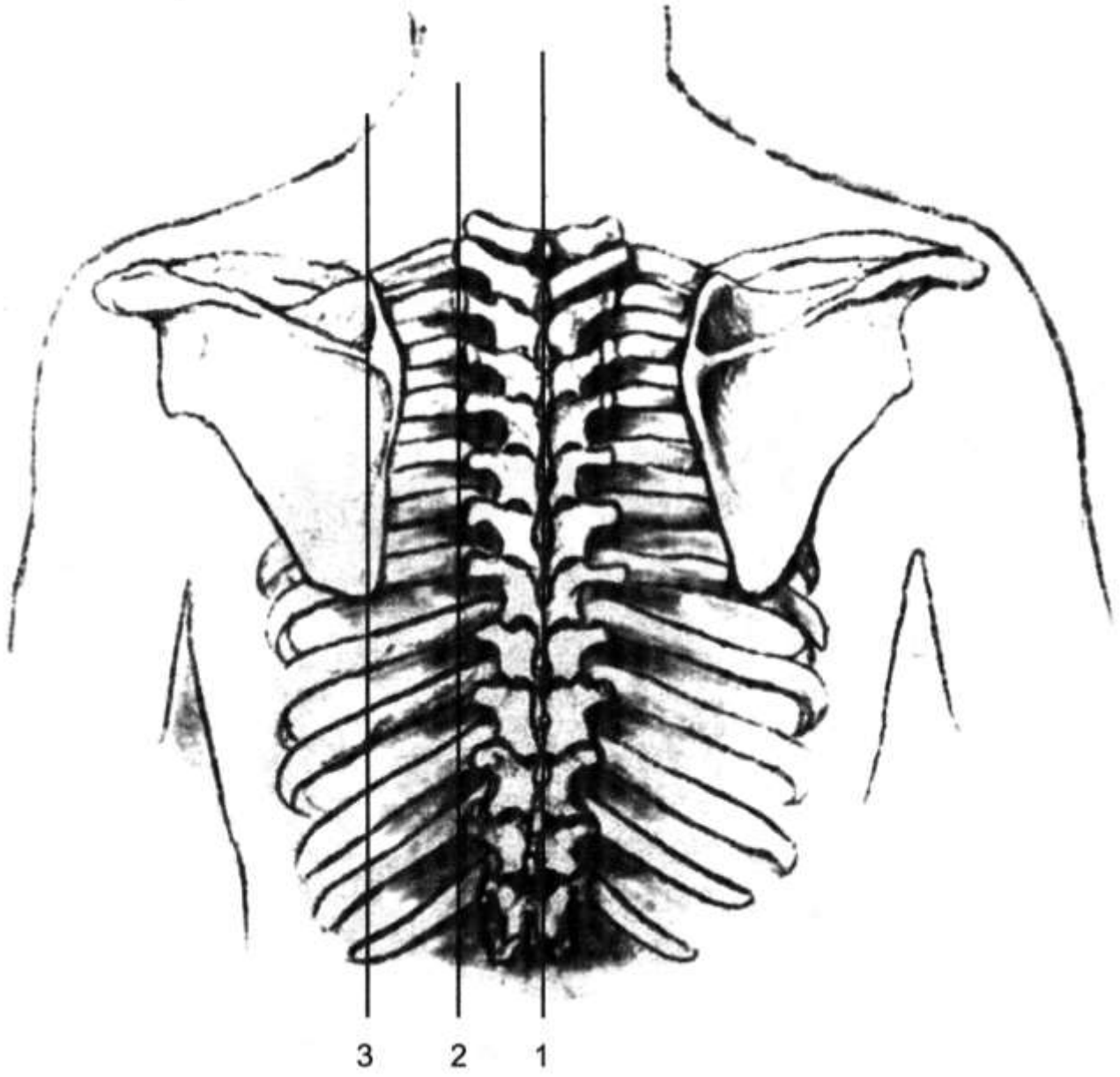


Fig. 3.7. Topographic lines.
Posterior view.
1 – vertebral line, 2 – paraspinal line, 3 – scapular line

The lungs lobes and fissures can be outlined mentally on the chest wall. Anteriorly, the apex of the each lung rises about 2 cm to 4 cm above the inner third of the clavicle (Fig. 3.8). The lower border of the lung passes the 6^{th} rib at the midclavicular line and 8^{th} rib at the midaxillary line.

Posteriorly, the lower border of the lung lies at about the level of the 11^{th} thoracic spinous process at the paraspinal line. On inspiration it descends (Fig.3.9).

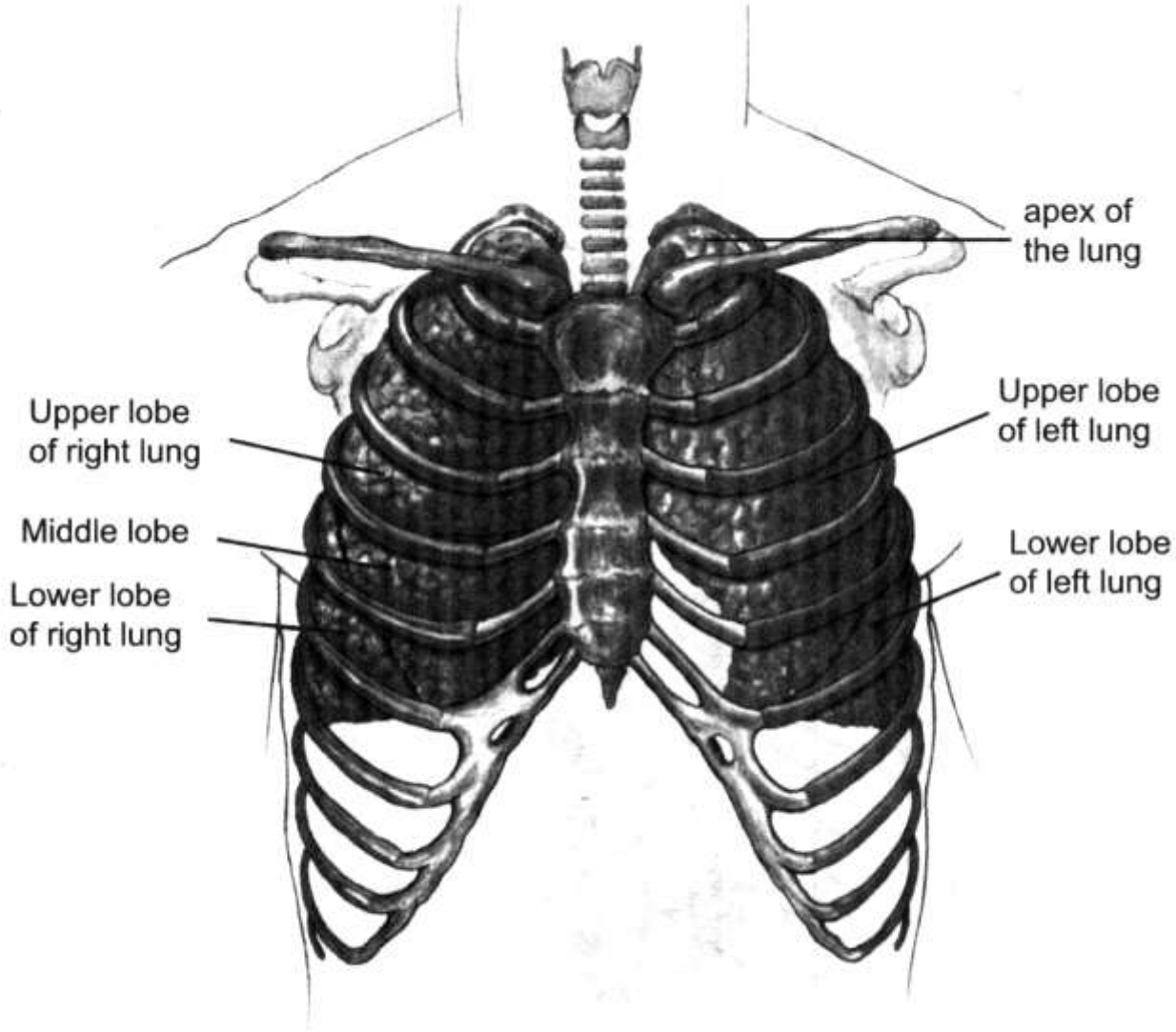


Fig. 3.8. Projection of the lungs on the chest wall.
Anterior view.

You should usually locate your pulmonary findings in external terms, such as these:

- ***Supraclavicular region*** – above clavicles;
- ***Infraclavicular region*** – below clavicle;
- ***Suprascapular region*** – above scapulae;
- ***Interscapular region*** – between the scapulae;
- ***Infrascapular region*** – below scapular;
- ***Bases of the lungs*** – the lowermost points;
- ***Upper, middle, and lower lungs fields***.

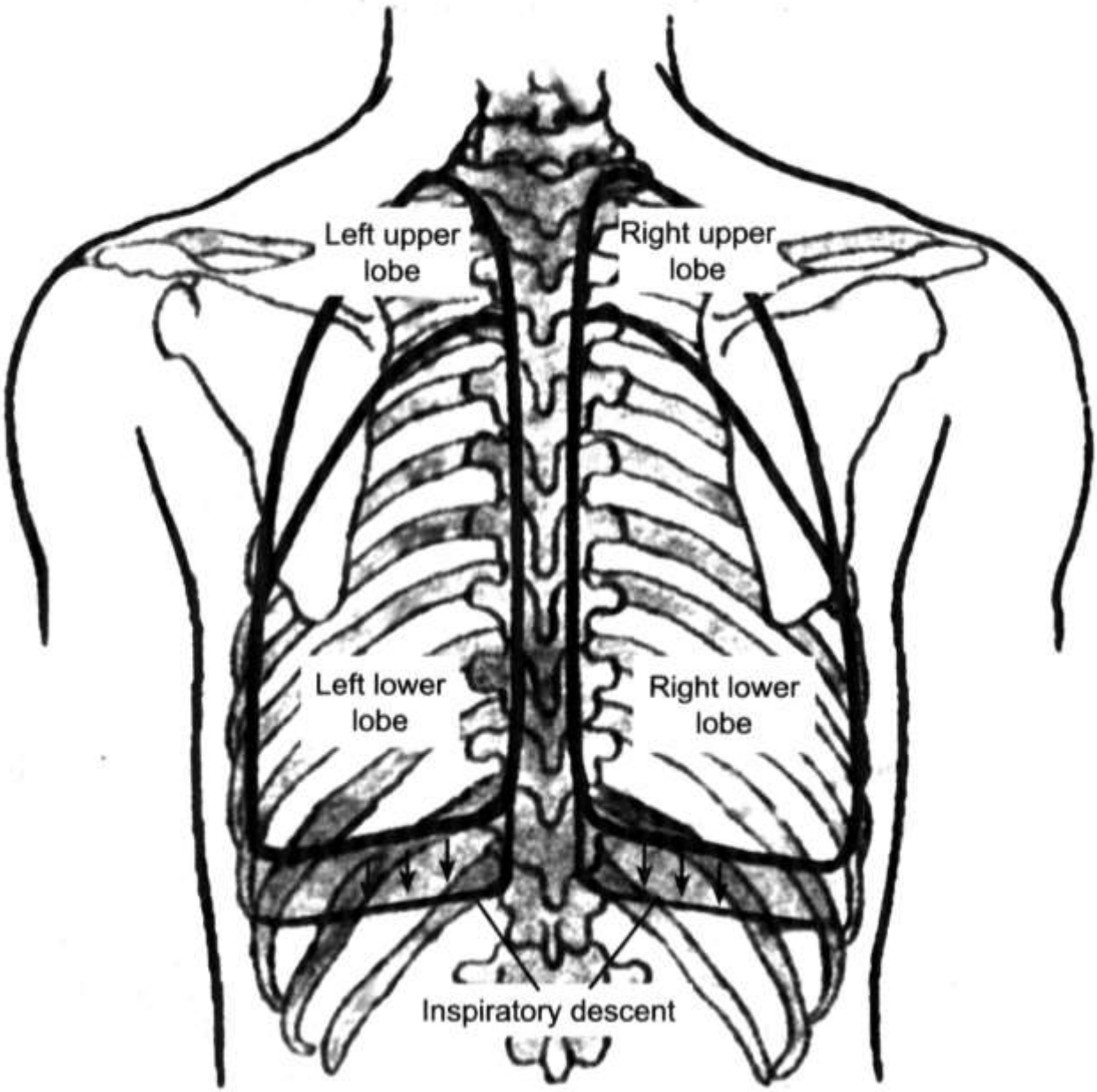


Fig. 3.9. Projection of the lungs on the chest wall. Posterior view.

The pleurae are serous membranes that cover the outer surface of each lung (*visceral pleura*) and also line the inner thorax and upper surface of the diaphragm (*parietal pleura*). Their smooth opposing surfaces, lubricated by pleural fluid, allow the lungs to move easily within the thorax during inspiration and expiration. The *pleural space* is the potential space between visceral and parietal pleura.

Inquiry

The main complaints of the patients with disease of the respiratory system are: dyspnea (breathlessness), cough, and chest pain.

Dyspnea is determined as an abnormally uncomfortable awareness of breathing. Patients use a large number of verbal expressions to describe these uncomfortable sensations, such as 'breathlessness', 'short of breath', 'out of breath', 'cannot get enough air', 'air does not go all the way down', 'smothering feeling or tightness or tiredness in the chest', a 'choking sensation', or even more colloquially 'puffed'.

The clinical analysis of dyspnea. All normal subjects will have experienced dyspnea on heavy exertion – *physiological dyspnea. Pathological dyspnea* is the same sensation occurring at lower workloads or at rest, and includes a perception that the awarness of breathing is unpleasant and/or inappropriate to the situation. The gradation of dyspnea may usefully be based on the amount of physical exertion required to produce the sensation. In assessing the severity of dyspnea, it is important to obtain a clear understanding of the patient's general physical condition, work history, and recreational habits. For example, the development of dyspnea in a trained runner upon running 3 km may signify a much more serious disturbance than a similar degree of breathlessness in a sedentary person upon running a fraction of this distance.

Dyspnea in its manifestation can be subjective, objective, and mixed. By *subjective dyspnea* is understood the subjective feeling of difficult breathing. *Objective dyspnea* is characterized by changes in respiration rate, depth, or rhythm, and also the duration of inspiration and expiration. Respiratory diseases are often accompanied by *mixed dyspnea.*

Dyspnea is possible with normal, rapid breathing (*tachypnea*), and slow rate of breathing (*bradypnea*).

Breathlessness is difficult to describe. Most patients can go no further than saying that they are 'short of breath'. Three types of *dyspnea quality* are differentiated by the prevalent breathing phase: *inspiratory dyspnea* (more difficult to breath in than out), expiratory dyspnea (more difficult to breath out than in), and *mixed dyspnea* when both inspiration and expiration phases become difficult.

Of the greatest value in separating out conditions likely to be associated with breathlessness is noting its rate of onset. There are five categories (Tab.3.1).

Tab. 3.1. ***Conditions causing dyspnea classified by rate of onset***

№	Categories	Causes
1.	**Dramatically sudden**: over minutes	Pneumothorax Pulmonary embolism Pulmonary edema
2.	**Acute**: over hours	Pneumonia Acute pulmonary infiltrations, e.g. allergic alveolitis Asthma Left ventricular failure
3.	**Subacute**: over weeks	Pleural effusion Bronchogenic carcinoma Subacute pulmonary infiltrations, e.g. sarcoidosis
4.	**Chronic**: over month or years	Chronic airflow obstruction Diffuse fibrosing conditions Chronic non-pulmonary causes, e.g. anemia, hyperthyroidism
5.	**Intermittent**: Episodic breathlessness	Asthma Left ventricular failure

Dyspnea may be of *dramatic onset* (over minutes), *acute onset* (over hours), *subacute onset* (over weeks), or *chronic onset* (over month or years), or it may be *intermittent*. The subdivision is not rigid.

The symptom of dyspnea is related to a process such as obstruction of the airways or to conditions that are associated with decreased vital lung capacity and ventilation.

Obstruction to airflow can be present anywhere from the extrathoracic airways out to the small airways in the periphery of the lung. A mechanical obstruction of the upper respiratory ducts (larynx, trachea) complicates and

slows down passage of the air into alveoli and causes inspiratory dyspnea. When the trachea and large bronchus are sharply contacted, both inspiration and expiration become difficult and noisy.

Obstruction of infrathoracic airways can occur acutely and intermittently or can be present chronically with worsening during respiratory infection. Narrowed lumen in the fine bronchi and bronchioles due to the inflammatory edema and swelling of their mucosa, or else spasm in the smooth muscles (bronchial asthma), interferes with normal air passage from the alveoli and expiratory dyspnea thus develops.

Dyspnea as an isolated symptom is likely to be due to diffuse parenchymal lung disease such as emphysema or diffuse fibrosis, pulmonary hypertension, or to extrathoracic conditions. The patients with predominant emphysema is characterized by many years of exertional dyspnea progressing to dyspnea at rest.

Heavy dyspnea, often followed by asphyxia is called suffocation. Asphyxia arising as sudden attack is asthma. In bronchial asthma the expiration is difficult, long, and noisy as a result of spasm of small bronchi. Bronchial asthma should be differentiated from cardiac asthma, which is secondary to left ventricular failure and is often accompanied by pulmonary edema with very difficult inspiration.

Cough is a defensive reflex designed to clear and protect the lower respiratory tract. The cough reflex can be initiated by stimulation of irritant receptors in the larynx, trachea, and major bronchi. These receptors respond to mechanical irritation by intraluminal material such as mucus, dust, or foreign bodies, and to chemical irritation by fumes and toxic gases. Mechanical events within the thorax, such as sudden and large changes in airway caliber or lung collapse, can also stimulate cough receptors.

The clinical analysis of cough. Any disorder resulting in inflammation, constriction, infiltration, or compression of airways can be associated with cough. Acute viral upper respiratory tract infection affecting pharynx, larynx, or postnasal space is the most common cause of short-lived cough over all ages. Asthma is the next common cause at all ages with chronic upper or lower respiratory tract infection also important. Drug therapy must not be forgotten because of potential for cure. Both β-blockers and angiotensin-converting enzyme inhibitors can cause an irritating and persistent cough.

The clinical description of cough relies to its character, its timing, and whether or not there is expectoration.

A cough may fail to produce expectoration; such type of cough is called *dry*. The cough productive of sputum can be described as *moist*. Some diseases are accompanied only by dry cough: laryngitis, dry pleurisy or compression of the main bronchi by the bifurcation lymph nodes (tuberculosis, lymphogranulomatosis, tumor metastases, etc.). Some disease, such as bronchitis, pulmonary tuberculosis, pneumosclerosis, abscess, or bronchogenic tumor of the lungs, can be first attended by dry cough, which will then turn into the moist cough.

A dry cough with irritative barking quality, short and often repeated, is heard in inflammatory conditions of pharynx, tracheobronchitis, and early pneumonia. With laryngitis the sound is harsh and hoarse ('croup'). The long inspiratory sound that gives whooping cough is also produced by tracheal and laryngeal inflammation. Abductor paralysis of the vocal cords creates a cough that is prolonged and lowing like the sound of cattle, and hence is described as 'bovine'.

Certain aspects of the timing of coughing may give useful diagnostic clues. Cough with expectoration on rising in the morning is characteristic of chronic bronchitis *('morning cough')*, although it may also be reported in asthmatics. Patients with pneumonia and acute bronchitis may complain of cough attacks during the entire day, but attacks may intensify by night *('evening cough')*. *'Night cough'* is characteristic of tuberculosis, lymphogranulomatosis, or tumor. A bout of coughing with food or when lying down after a meal points to esophageal, pharyngeal, or neuromuscular disease, causing aspiration into the lung. Changes of posture can also set off coughing in the bronchiectasis.

Cough may be *periodic* or *permanent*. Periodic cough occurs more frequently. Such cough is characteristic of influenza, pneumonia, pulmonary tuberculosis, and chronic bronchitis. Permanent cough occurs in laryngitis, acute bronchitis, bronchogenic tumor of the lungs, and in certain forms of tuberculosis.

Phlegm, the secretions of the lower respiratory tract, is admixed with nasal and pharyngeal secretions as well as saliva to give expectorated *sputum*. If the patient complains of cough with sputum you should try to determine:

- the amount of sputum during one fit and during entire day;
- the time of day when sputum is expectorated;
- posture of the patient at which cough is provoked;
- properties of the sputum (the color, odour, etc.).

In the patients with chronic bronchitis, bronchiectasis, lung abscess, and cavernous tuberculosis of the lungs, the sputum accumulates during the night sleep in the lungs and bronchi. When the patient gets up in the morning, the sputum moves to stimulate the reflex zones of the bronchial mucosa and cause cough and expectoration of sputum. The amount of the morning sputum is two thirds of the entire daily expectoration. The daily amount of sputum may vary from 10–15 ml to 2 liters. In unilateral bronchiectasis, sputum may be better expectorated when the patient lies on the healthy side.

Hemoptysis defined as the expectoration of blood from the respiratory tract.

The clinical analysis of the hemoptysis. In the assessment of the hemoptysis it is important to establish first that the blood-stained material has come from the chest and not from the gastrointestinal tract. Hemoptysis is produced with a "cough" not a "retch". Accompanied features of an appropriate disease are usually present, but it is worth remembering that in hemoptysis there is usually froth due to admixed air, and the blood is bright red not dark brown. Gastric contents should be acid, bronchial contents should be alkaline.

The most common site of bleeding is the airways, i.e., tracheobronchial tree, which can be affected by inflammation (acute or chronic bronchitis, bronchiectasis) or by neoplasm (bronchogenic carcinoma, or bronchial carcinoid tumor). Blood originated from the pulmonary parenchyma can be either from a localized source, such as an infection (pneumonia, lung abscess, tuberculosis), or from a process diffusely affecting the parenchyma. Disorders primarily affecting the pulmonary vasculature include pulmonary embolic disease and those conditions associated with elevated pulmonary venous and capillaries pressure, such as mitral stenosis or left ventricular failure.

Hemoptysis that is described as blood-streaking of mucopurulent or purulent sputum often suggest bronchitis. Chronic production of sputum with a recent change in quantity or appearance favors an acute exacerba-

tion of chronic bronchitis. It is unwise to attribute hemoptysis simply to bronchitis or infection. In bronchiectasis, however, hemoptysis not uncommonly mixed with mucopurulent sputum. In the early stages of pneumococcal pneumonia a 'rusty' staining of mucoid sputum is quite characteristic, a putrid smell to the sputum raises the possibility of lung abscess. In tuberculosis frank blood in otherwise mucoid sputum is well recognized. Heamoptysis following the acute onset of pleuritic chest pain and dyspnea is suggesting the pulmonary embolism. In bronchial neoplasia there may be streaking of the sputum with blood or more substantial bleeding with clots, often observed daily. In general context, it may necessary to consider thoracic trauma, endometriosis, or a blood coagulation disorder as cause of hemoptysis.

Chest pain. The greater part of the lower respiratory tract is insensitive to pain. Most parenchymal lung disorders proceed to an advanced state without pain. However, the parietal pleura, is exquisitely sensitive to painful stimuli and unpleasant sensations can arise from the tracheobronchial tree.

The clinical analysis of the chest pain. Typical pleural pain has a sharp stabbing and knife-like character in pleurisy and is accentuated by respiratory movement. Hence it is aggravated by respiration and coughing thus leading to rapid shallow breathing and a suppressed cough. The pain is more likely to be due to stretching of the inflamed parietal pleura and can be relieved by splinting the chest wall. If pleurisy progresses to pleural effusion, the sharp pain largely disappears and is replaced by a dull and more constant ache or heaviness, quantitatively roughly proportional to the amount of fluid. Most conditions giving rise to pleuritic pain are acute and inflammatory in origin: either infective when there is usually associated pneumonia (pleurisy is particularly common in pneumococcal pneumonia) or infarctive as pulmonary embolism. Chest-wall pain can mimic pleurisy, and conditions in the chest wall provide its important differentials. Pain due to strain or tearing of thoracic muscles can quite sharp, and since it may be caused by coughing and may cause shallow respiration, it can easily be confused with pleurisy. However, there is always local tenderness over affected muscle and none of the ancillary investigations for pleurisy prove positive. Patients with

persistent cough or distressing breathlessness, particularly due to asthma, may complain of muscular pain around the lower thorax. Sensations arising from the tracheobronchial tree are less easy to characterize as painful, although some are exceedingly unpleasant. Tracheal inflammation, as infective tracheobronchitis or following the inhalation of toxic vapours, cause a raw painful sensation retrosternally. Finally, the mediastinal structures of the thorax are responsible for a multitude of pains. Myocardial ischemia, pericarditis, aneurysm, esophagitis, and referred abdominal pain will all need to be considered in the differential diagnosis of central chest pain. Most have distinctive features that will rarely lead to confusion with the few central pulmonary lesions likely to give mediastinal pain.

Inspection of the Chest

The patient should be better examined in standing or sitting position with the chest being naked. Illumination of the body should be uniform. Examine the chest in an orderly fashion. Note the shape of the chest, its symmetry *(static inspection)*, type of respiration, participation of the chest wall in breathing act, respiration rate, depth and rhythm *(dynamic inspection)*.

Static inspection

The shape of the chest. Normal and pathological shapes of the chest are distinguished.

Normal shapes of the chest. As three types of the constitution are existed, the chest has different shape according to these constitutional types. There are three normal shapes of the chest: normosthenic, hypersthenic, and asthenic chest.

Normosthenic chest: the shoulders are under the right angle to the neck, supra- and infraclavicular fossae feebly expressed, the ribs are moderately inclined, the interspaces are visible, but moderate expressed, epigastric angle is near 90°, the lateral diameter is larger than anteroposterior, shoulder blades closely fits to the chest and are on the same level. The thorax is about the same height as abdominal part of the trunk (Fig. 3.10).

Hypersthenic chest: the shoulders are wide and the neck is short, supra- and infraclavicular fossae are absent (level with the chest), direction of the ribs are nearly horizontal, the interspaces are narrow and slightly expressed, epigastric angle exceeds 90°, the lateral diameter is about the same as anteroposterior, the chest has form of a cylinder, the shoulder blades closely fit to the chest. The thorax is smaller than abdominal part of the trunk (Fig. 3.11).

Asthenic chest: the shoulders are sloping and are under the dull angle to the neck, clavicles are well visible, supra- and infraclavicular fossae are distinctly pronounced, the ribs direct downward abruptly, more vertical at sides, the 10th ribs are not attached to the costal arch (*costa decima fluctuens*), the interspaces are wide and pronounced, epigastric angle is less than 90°, both lateral and anteroposterior diameter are smaller than normal, the chest narrow and elongated, the shoulder blades are separate from the chest (*scapula alatae*) and their angles are well visible. The muscles of the shoulder girdle are underdeveloped. The thorax is longer than abdominal part of the trunk (Fig. 3.12).

N.B. Palpation is used to confirm the observed patterns of the epigastric angle. In order to determine epigastric angle place your thumbs along the cos-

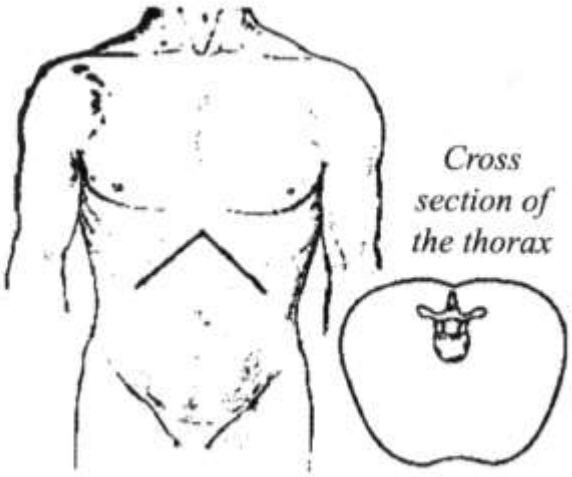


Fig. 3.10. Normosthenic chest.

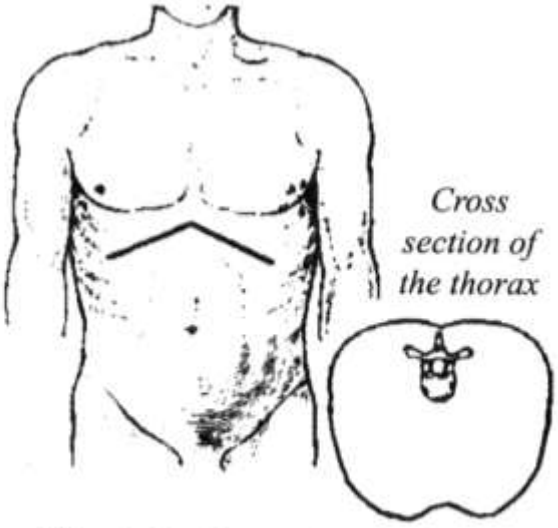


Fig. 3.11. Hypersthenic chest.

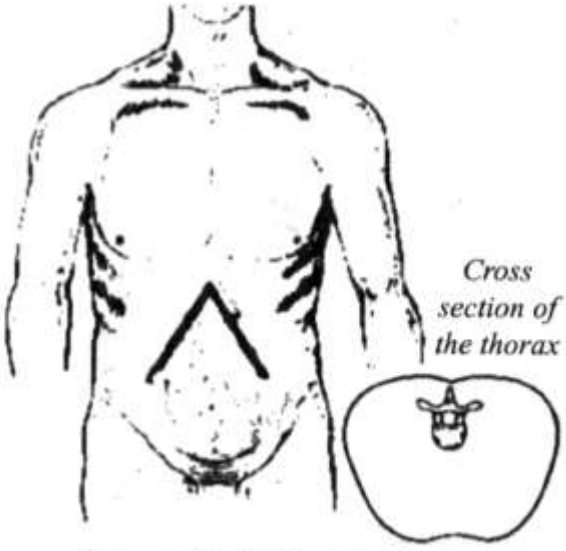


Fig. 3.12. Asthenic chest.

tal arch, their tips resting against xiphoid process. Your hands should be along the lateral rib cage.

Pathological shapes of the chest can be caused either by chronic diseases of the lungs and pleura (emphysematous, paralytic chest), or by pathology of the thorax costal skeleton (rachitic, funnel, and foveated chest), or by various deformities of the spine (scoliosis, kyphosis, lordosis, kyphoscoliosis).

Pathological shapes of the chest caused by chronic pulmonary diseases.

Emphysematous or barrel chest resembles hypersthenic in its shape. Supra- and infraclavicular fossae are absent or even protruded, the ribs are horizontal, the interspaces are enlarged, epigastric angle is more than 90°, the chest wall is prominent, chest has an increased anteroposterior diameter and that is why chest has a barrel-like configuration (Fig. 3.13).

Emphysematous chest observes in emphysema of the lungs. The lungs seem to be as if at the inspiration phase due to decreased elasticity of the lungs. The volume of the lungs increases, the natural expiration become difficult (expiratory dyspnea). Accessory respiratory muscles active participate in the breathing act, especially *m. sternocleidomastoideus and m. trapezius*. Elevation and lowering of the entire chest during breathing become evident. A barrel shape is normal during infancy, and often accompanies normal aging.

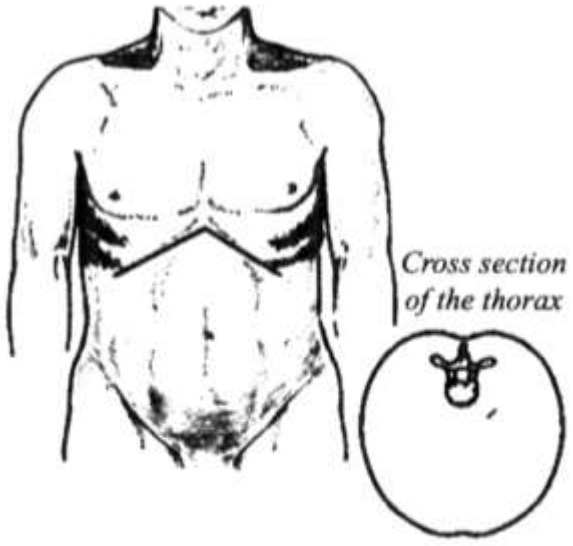


Fig. 3.13. Emphysematous or barrel chest.

Paralytic chest. The same signs that peculiar to the asthenic chest but more pronounced characterize paralytic chest. The shoulders are sloping, clavicles are asymmetrical and pronounced, supra- and infraclavicular fossea depresses, the ribs are vertical, the interspaces are wide and depressed, marked atrophy of the chest muscles, epigastric angle is less than 90°, the shoulder blades are not on the same level, and their movement during breathing are asynchronous (Fig. 3.14).

Paralytic chest is found in emaciated patients, in general asthenia, constitutional underdevelopment and in the patients with long-standing pulmonary pleural diseases, such as pulmonary tuberculosis and pneumosclerosis. Growing of the connective tissue contracts the lungs and diminished their size due to progressive chronic inflammation.

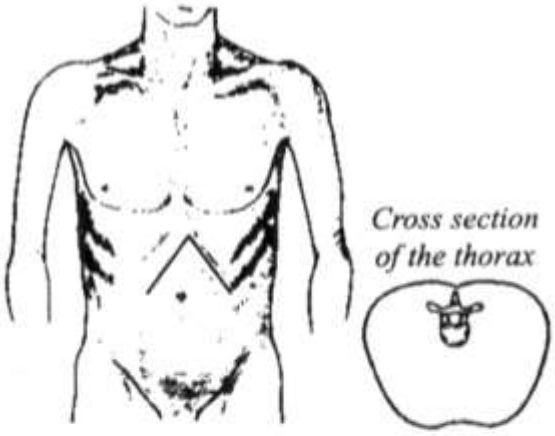


Fig. 3.14. Paralytic chest.

Pathological shapes of the chest caused by pathology of the thorax costal skeleton.

Rachitic or pigeon chest *(Pectus Carinatum)* is a result of abnormal skeleton formation in childhood in the patients with rachitis. In a pigeon chest, the sternum is displaced anteriorly, increasing anteroposterior diameter, resembling the keel of the boat. The ribs meet at an acute angle at the sternum, the costal cartilages thicken like beads at points of their transition to bones (rachitic beads) (Fig. 3.15).

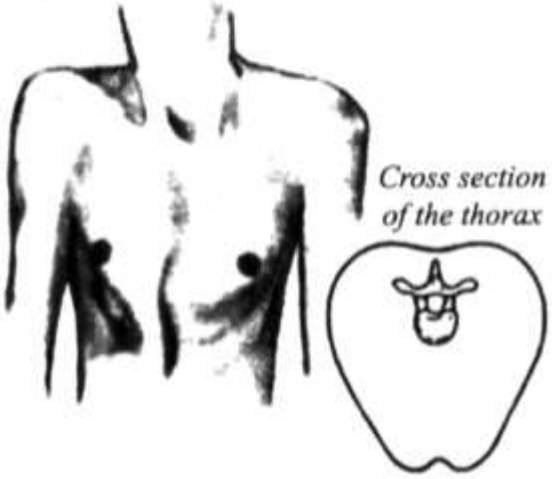


Fig. 3.15. Rachitic or pigeon chest *(Pectus Carinatum).*

Funnel chest *(Pectus Excavatum)* is characterized by a depression in the lower portion of the sternum near the xiphoid process. This deformity can be the result of abnormal development of the sternum or prolonged compressing effect. But exact cause is now unknown. In older times such shape of the chest was found in shoemaker adolescents, and was explained by permanent pressure of the chest against the shoe. Therefore, the funnel chest is also called 'cobbler chest' (Fig. 3.16). Compression of the heart and great vessels may cause murmurs.

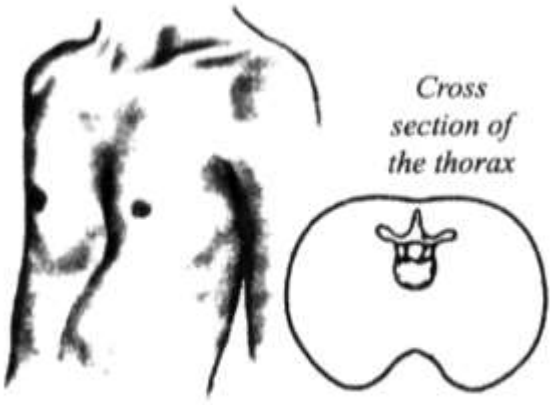


Fig. 3.16. Funnel chest *(Pectus Excavatum).*

Foveated chest is characterized by vertical depression on the upper and middle parts of the anterior surface of the chest. This deformity arises in syringomyelia, a rare disease of the spinal cord.

Pathological shapes of the chest caused by various deformities of the spin as a result of injuries, tuberculosis of the spine, rheumatoid arthritis, etc.

Four types of spine deformities are distinguished:

Scoliosis – lateral curvature of the spine, is most common. It develops in schoolchildren due to bad habitual posture.

Kyphosis – backward curvature of the spine with formation of the gibbus, occurs less frequently.

Lordosis – forward curvature of the spine, generally in the lumber region, occurs in rare cases.

Kyphoscoliosis – combination of the lateral and backward curvature of the spine (Fig. 3.17).

Kyphosis, lordosis, and kyphoscoliosis change physiological position of thoracic organs and thus interfere with their normal function.

The symmetry of the chest. The right and left sides of the normal chest are symmetrical, the clavicles and shoulder blades are on the same level, the supra- and infraclavicular fossae and interspaces equally pronounced on both sides. One-sided enlargement or diminutions of the chest (asymmetry) due to the pulmonary and pleural diseases are of great diagnostic value. These changes can transient or permanent.

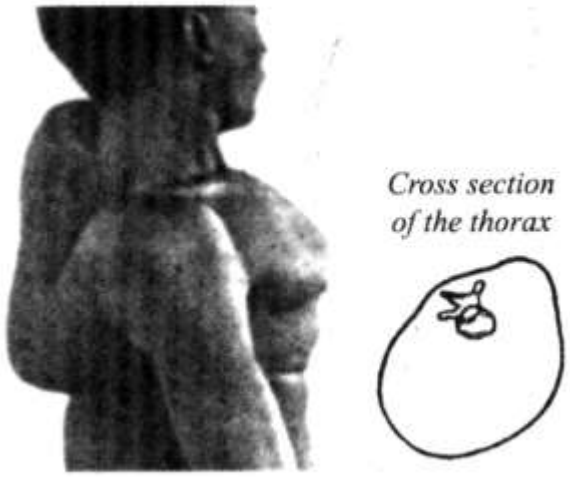


Fig. 3.17. Thoracic kyphoscoliosis.

Enlarged volume of one half of the chest observes in accumulation of considerable amount of fluid (exudates, transudate, blood, pus) in the pleural cavity, or in penetration of air inside the chest in injuries (pneumothorax).

During examination of the enlarged part of the chest you can see asymmetry of the clavicles; leveling or protrusion of the interspaces, and

they more wide; the distance between nipple and median line, and from inside edge of scapula to the spine on affected side is larger than on healthy one. Enlarged part of the chest lags in the breathing act. The patient slightly bends to the affected side of the chest. The chest assumes normal symmetrical shape after the fluid or air is removed from the pleural cavity.

Decreased volume of the one part of the chest observes in:

- contraction of a considerable portion of the lung due to the growth of connective tissue – pneumosclerosis, after acute or chronic inflammatory processes, such as acute pneumonia (with subsequent carnification of the lung), lung infarction, pulmonary abscess, tuberculosis;
- pleural adhesion or contraction of the pleural membranes after resorption of fluid;
- obstructive athelectasis;
- resection of a part or the entire lung.

During examination of the decreased part of the chest you can see that the shoulder and clavicle lowers, supra- and infraclavicular fossae are more depressed, the interspaces are decreased in size or invisible, the nipple is nearer to the sternum as compared with healthy side, and the scapula lowers. The respiratory movement of clavicle and scapula become slower and limited on affected side.

Dynamic inspection

In dynamic inspection of the chest the pattern of breathing (type of respiration, participation of the chest wall in breathing act, respiration rate, depth and rhythm) must be observed.

Breathing in and out (*inspiration* and *expiration*, together called *respiration* or *external respiration*) is essential for taking O_2 and getting rid of CO_2. Respiration is largely an automatic act, controlled in the brain system and mediated by the muscles of respiration. The *main respiratory muscles* – diaphragm, intercostals muscles, and partly the abdominal wall muscles are normally used for this propose. The *accessory muscles – mm. sternocleidomastoideus, trapezius, pectoralis major et minor*, etc., join the respiratory effort in pathological conditions, associated with difficult breathing.

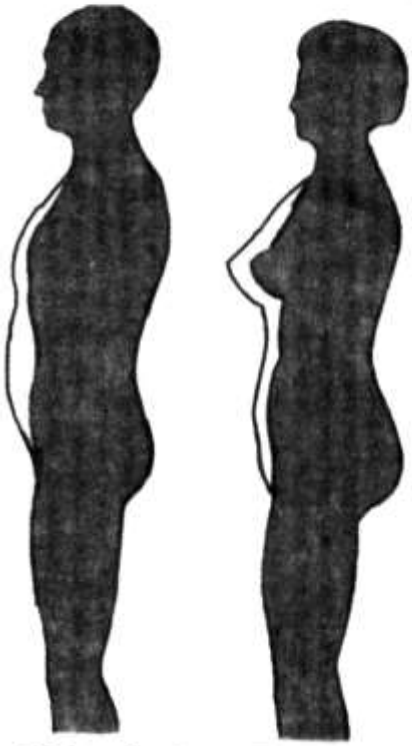
Respiration type. Respiration can be thoracic, abdominal or mixed type.

Thoracic (costal) respiration. Mainly the intercostals muscles carry out respiratory movements. In *inspiration* the intercostals muscles contract

and elevate the ribs, these movements increase the internal capacity of the lungs. As the thoracic wall expands, the lungs also expand and draw in air. In *expiration*, the thoracic capacity decreases as the inspiratory muscles relax – the lungs then shrink by their own elasticity. This type of breathing is known as costal and is mostly characteristic of women (Fig. 3.18).

Abdominal respiration. The diaphragm is the primary muscle in this type of respiration. In *inspiration* the diaphragm contracts, descends in the chest and enlarges the thoracic cavity. The thoracic enlargement decreases intrathoracic pressure, draws air through the tracheobronchial tree into the alveoli, and expands the lungs. At the same time it compresses the abdominal contents, pushing the abdominal wall outward. In expiration the chest wall and lungs recoil, the diaphragm rises passively, air flows outward, and the chest and abdomen return to their initial position (Fig. 3.18). This type of breathing is also called diaphragmatic and is mostly characteristic of men.

Mixed respiration. The diaphragm and the intercostals muscles simultaneously carry out respiratory movements. This type of respiration observes in the aged persons and some pulmonary and digestive diseases.



Abdominal **Thoracic**

Fig. 3.18. Types of respiration.

In women mixed respiration occurs in dry pleurisy, pleural adhesion, myosytis, thoracic radiculitis, and lung emphysema.

In men mixed respiration occurs in persons with underdeveloped diaphragmatic muscle, diaphragmatitis, acute cholecystitis, perforating ulcer.

Participation of the chest wall in breathing act. In observing respiratory movement, particular attention must be paid to expansion. Poor movement of the chest on one side only always indicates pathology on that side. One part of the chest lags in the breathing act in inflammatory infiltration of extensive part of the lung, dry pleurisy, hydrothorax, pneumothorax, ribs fractures, intercostals neuralgia, and myositis. In paralysis or paresis respiratory excursion on the corresponding part is limited.

Respiration rate. In order to determine respiratory rate count the movement of the chest or abdominal wall, with patient being unaware of the procedure.

The repeated cycles of inspiration followed by expiration (*respiratory cycle*) occur in adults at rest about 16–20 times per minute (the *respiratory rate*), with inspiration lasting approximately 2 seconds and expiration 3 seconds (Fig. 3.19).

The respiration rate in newborn is 40–45 per minute, this rate gradually decreasing with age. During night sleep respiratory rate decreases to

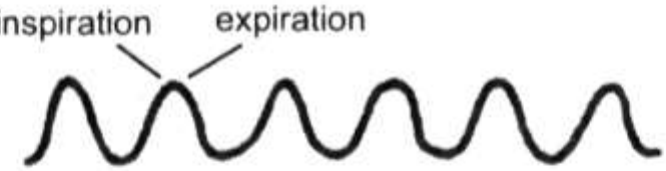


Fig 3.19. Normal breathing.

12–14 per minute, and increases in physical and emotional exertion, and after heavy meals.

Pathological rapid breathing above 20 per minute is called **tachypnea** (Fig. 3.20).

Fig. 3.20. Tachypnea – rapid shallow breathing.

Tachypnea has a number of causes:

- conditions associated with decreased respiratory surface of the lungs: inflammation, tuberculosis, compressive atelectasis (hydrothorax, pneumothorax, mediastinal tumor), obstructive atelectesis, pulmonary emphysema, and pulmonary edema;
- narrowing of the small bronchi due to spasm or diffuse inflammation of their mucosa (bronchiolitis), which interfere normal air passage into alveoli;
- shallow respiration as a result of difficult contractions of the respiratory muscles in acute pain (dry pleurisy, acute myositis, intercostals neuralgia,

rib fracture) and in elevated abdominal pressure and high diaphragm level (ascitis, meteorism, late pregnancy).

Pathological slow breathing below 16 per minute is called **bradypnea** (Fig. 3.21).

Fig. 3.21. Bradypnea – slow breathing.

Bradypnea may be secondary to such causes as increased intracranial pressure (cerebral tumor, hemorrhage, meningitis, brain edema) due to inhibition of the respiratory center, and also due to the toxic effect on respiratory center in uremia, diabetic or hepatic coma, and drug-induced respiratory depression.

Respiration depth. The volume of the inhaled and exhaled air at rest in adults varies from 300 to 900 ml (500 ml on the average). Depending on depth, breathing can be shallow or deep.

Shallow respiration is characterized by short inspiratory and expiratory phases. Shallow breathing is usually rapid (Fig. 3.20). In some cases, however, shallow respiration can be slow due to inhibition of the respiratory center, pronounced pulmonary emphysema, and sharp narrowing of the vocal slit or trachea.

Deep respiration is characterized by elongation of the inspiratory and expiratory phases. As a rule, deep respiration is slow (Fig. 3. 21). Rapid deep breathing has several causes, including exercise, anxiety, fever, anemia, and metabolic acidosis. Deep rapid breathing, with marked respiratory movements, accompanied by noisy sound is called *Kussmaul respiration* (Fig. 3.22). This type of breathing observes in the comatose patients due to metabolic acidosis.

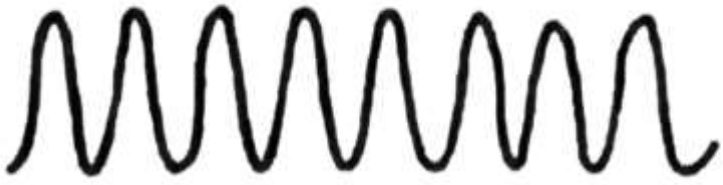


Fig. 3.22. Kussmaul respiration – deep rapid breathing (*Hyperpnea, Hyperventilation*).

Respiration rhythm. A normal rhythm of breathing is controlled by groups of nerve cells in the brainstem, called the *respiratory center*. These nerve cells send impulses down the spinal cord to act on the spinal nerve fibers that supply the diaphragm and intercostals muscles.

Respiration of a healthy person is rhythmic, and characterized by uniform depth, and approximately equal duration of inspiratory and expiratory phases (Fig. 3.19). In depression of the respiratory center breathing becomes arrhythmic. Periods of breathing alternate with readily detectable elongation of respiratory pause from few seconds to a minute or with apnea (temporary arrest of breathing) and also respiration may be of different depth. Such type of respiration is called *periodic* and includes Cheyne-Stokes respiration, Grocco's respiration, and Biot's respiration.

Cheyne-Stokes respiration (Fig. 3.23). Noiseless shallow respiration quickly deepens, becomes noisy to attain its maximum at the 5–7 inspirations and slows down gradually. Such periods alternate with periods of apnea (from few second to a minute), during which patient can loses orientation in surroundings or even faints to recover from unconsciousness after respiration restores.

Fig. 3.23. Cheyne-Stokes respiration.

Children and aged people normally may show Cheyne-Stokes respiration in sleep. Other causes include heart failure, uremia, drug-induced respiratory depression, and brain damage (acute or chronic failure of the cerebral circulation, cerebral hypoxia, meningitis).

Grocco's respiration resembles Cheyne-Stokes respiration except that shallow respiration occurs instead of apnea (Fig.3.24).

Fig. 3.24. Grocco's respiration.

Grocco's respiration is caused probably by early stages of the same conditions as Cheyne-Stokes respiration.

Biot's respiration (Fig. 3.25). In this type of breathing deep rhythmic respiration alternate with apnea (from few seconds to half minute). Causes include respiratory depression and brain damage (meningitis, agony with disorders of cerebral circulation).

Fig. 3.25. Biot's respiration.

Palpation of the Chest

Palpation of the chest has three potential uses:

1. identification of the tender areas;
2. assessment of elasticity of the chest;
3. assessment of tactile fremitus.

Identification of tender areas. Carefully palpate from the 1st interspaces on the anterior chest (5 steps), then along midaxillary lines (3 steps), and along the spine on the posterior chest (10 steps).

In rib fracture pain is localized over a limited area, careful displacement of bone fractures attends by a specific sound (crunch). Intercostals tenderness commonly has musculoskeletal origin. Such pain is called superficial, it intensifies during deep breathing, and when the patient bends or lies on the affected side.

Assessment of elasticity of the chest. Assess elasticity by pressing the chest in anteroposterior and lateral directions. Place your hands parallel: right on the middle of the sternum, left – on the spine and press the chest. Then by both hands press the chest in lateral direction.

The chest of a healthy person is elastic, and yields under the pressure. Rigidity of the chest indicates presence of fluid in the pleural cavity or pleural tumor, and pulmonary emphysema. In aged persons the chest become rigid due to ossification of the costal cartilages.

Assessment of tactile fremitus *(vocal fremitus, s. pectoralis)*. Fremitus refers to the palpable vibrations transmitted through the bronchopulmonary

tree to the chest wall when the patient speaks. Ask the patient to repeat the words "ninety-nine" or "one-one-one". If fremitus is faint, ask the patient to speak more loudly or in a deeper voice.

Palpate and compare symmetrical areas of the chest, using the palms of your both hands simultaneously.

Firstly, place your hands on the patient's shoulder over the lungs apices projection, then in the infraclavicular regions, and axillary regions, using the vibratory sensitivity of your hands to detect fremitus (Fig. 3.26).

Posteriorly, you should assess vocal fremitus in the supra-, inter-, and infrascapular regions (Fig. 3.27).

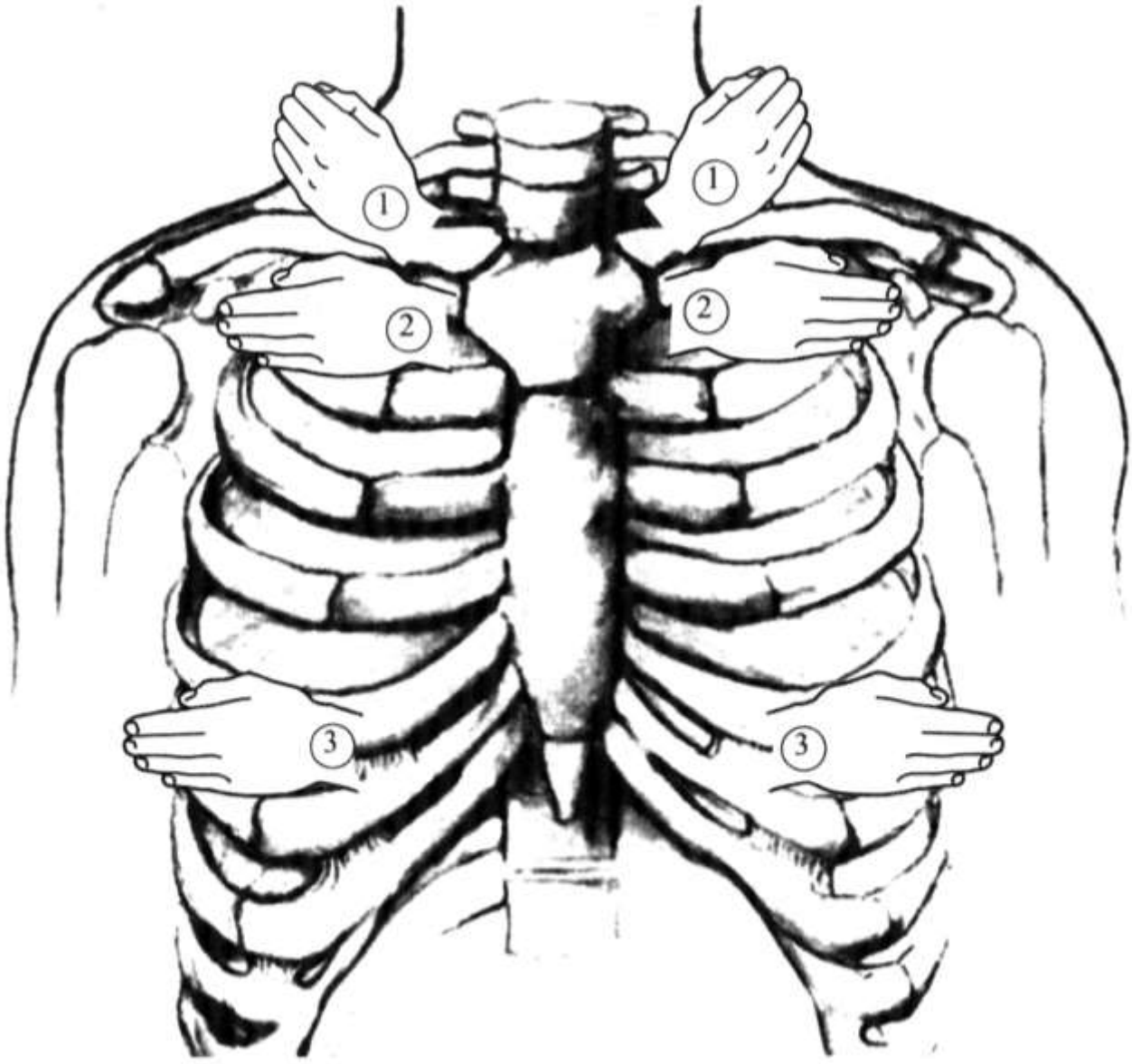


Fig. 3.26. Assessment of tactile fremitus. Anterior view.

Identify and locate any areas of increased, decreased or absent fremitus.

Tactile fremitus is typically more prominent in men than in women and children; in the upper lung fields than in the lower one; and more prominent on the right side (voice transmission is better through the shorter right main bronchus) than on the left.

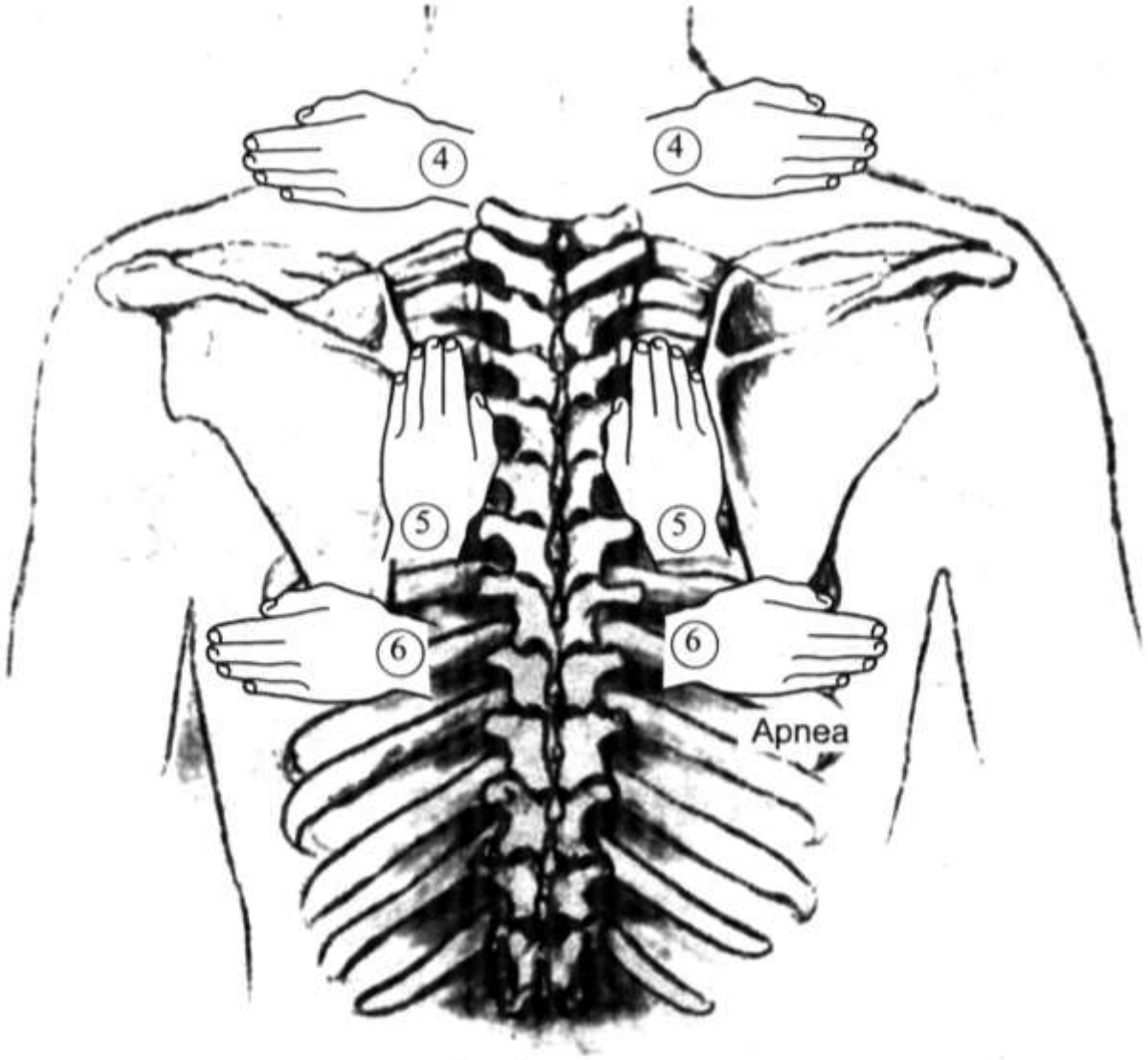


Fig. 3.27. Assessment of tactile fremitus.
Posterior view.

Vocal fremitus is increased in consolidation of the pulmonary tissue (lobar pneumonia, lungs infarction, pulmonary tumor, tuberculosis, compressive atelectasis), and also in the presence of an air cavity communicated with bronchus.

Tactile fremitus is decreased when the voice is soft in emaciated patients or when the conduction of vibrations from the larynx to the surface of the chest is impeded. Causes include separation of the lung by moderate amount of fluid (pleural effusion) or air (pneumothorax), by fibrosis (pleural thickening); obstructive atelectasis; and also a very thick chest wall (edema, subcutaneous fat).

Tactile fremitus can be absent when significant amount of fluid or air are accumulated in the pleural cavity.

Percussion of the Lungs

Two types of percussion of the lungs – comparative and topographic – are existed.

Comparative percussion of the lungs

The *task* of comparative percussion is to compare percussion sounds over the lungs on the opposite parts of the chest, and also on neighboring areas on the one side.

The technique of comparative percussion. The patient should be in a comfortable posture and relaxed. The best position is standing or sitting. Patients with grave diseases should be percussed in the lying posture. The room should be warm and protected from external noise.

Percussion consists of setting up vibrations in the chest wall by means of a sharp tap. The middle finger of the left hand (pleximeter finger) is placed in close contact with the chest wall in the intercostals space. A firm sharp tap is then made by the middle finger of the right hand (plexor finger) kept at right angles to the pleximeter finger. Loud percussion (with a normal force of taping) is used.

All areas of the chest are percussed, that is, the front, both axillary regions, and back.

In *anterior percussion*, place pleximeter finger parallel to the clavicle in the right, in the left supraclavicular regions, and then along midclavicular line. On the left side percussion is carried out only to the 3rd interspace, because underlying heart below this level changes percussion sound (Fig. 3.28).

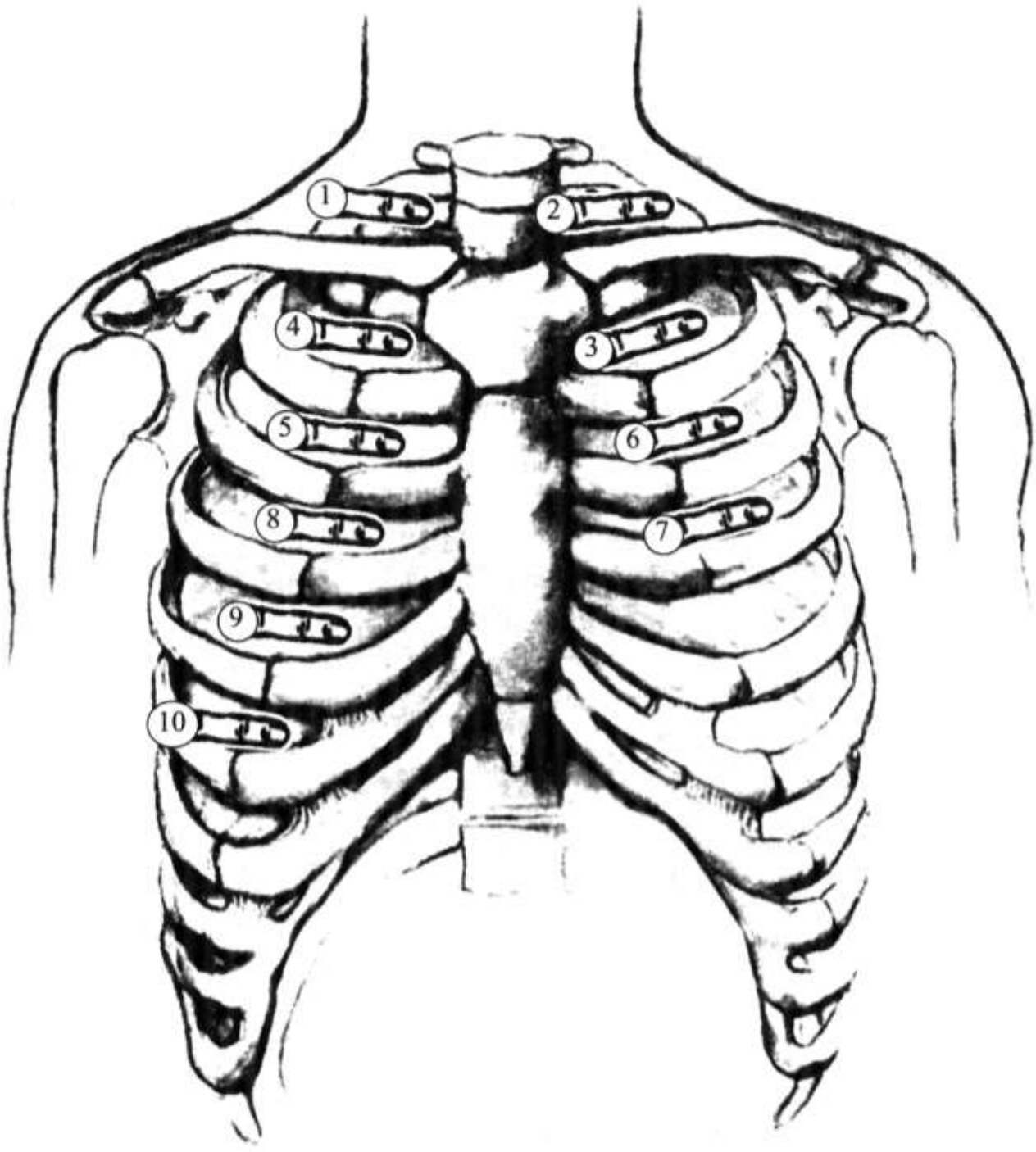


Fig. 3.28. Comparative percussion of the lungs.
Anterior view.

Axillary regions are percussed with the patient's arms folded at the back of the head. Percuss lateral chest along midaxillary line, starting from the right side (Fig. 3. 29).

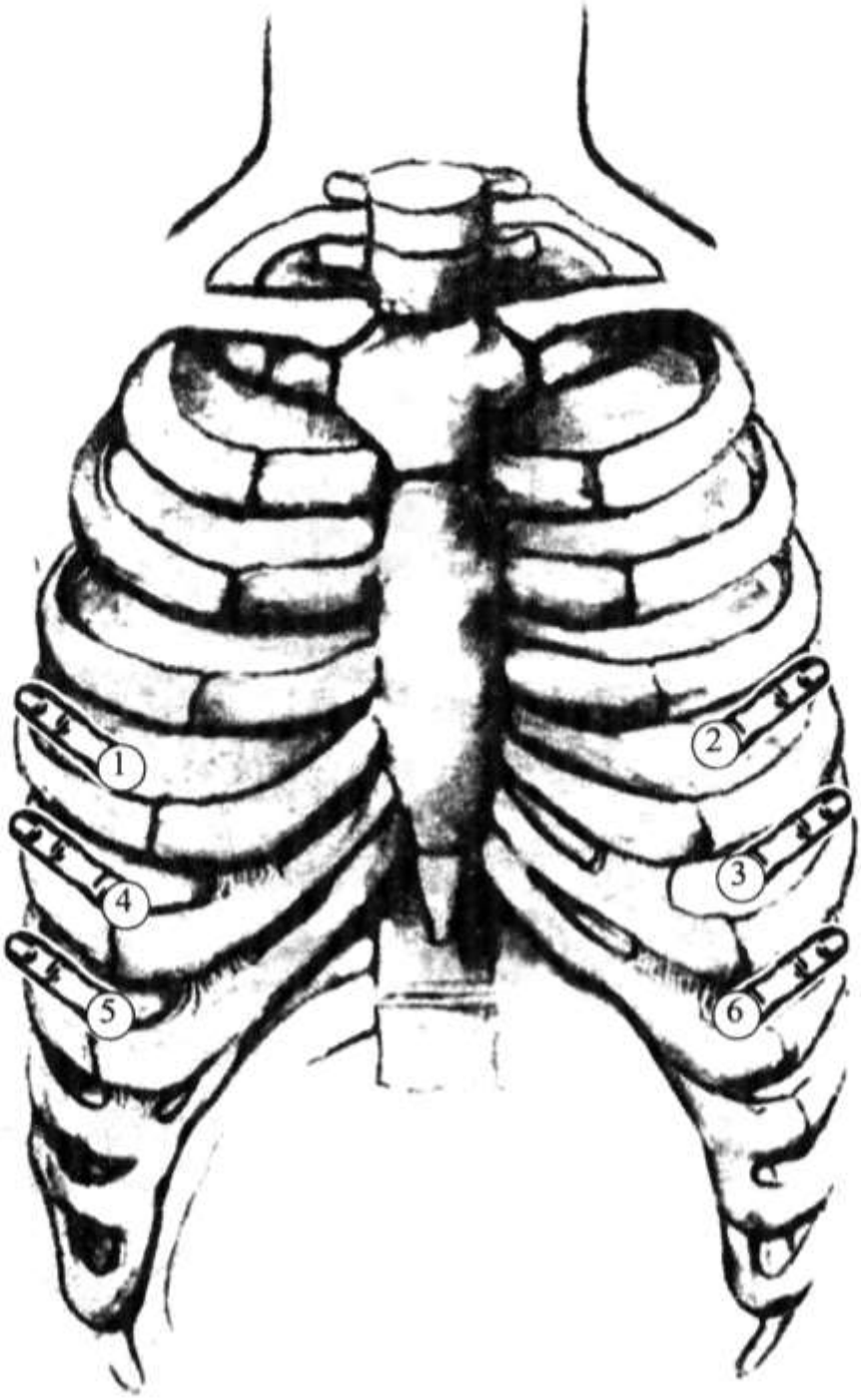


Fig 3.29. Comparative percussion of the lungs. Axillary regions.

When percussing *posteriorly* the patient keeps both arms crossed in front of the chest to move scapulae anteriorly. Place pleximeter finger in the suprascapular regions horizontally, in the interscapular regions vertically, and in infrascapular again horizontally (Fig. 3.30).

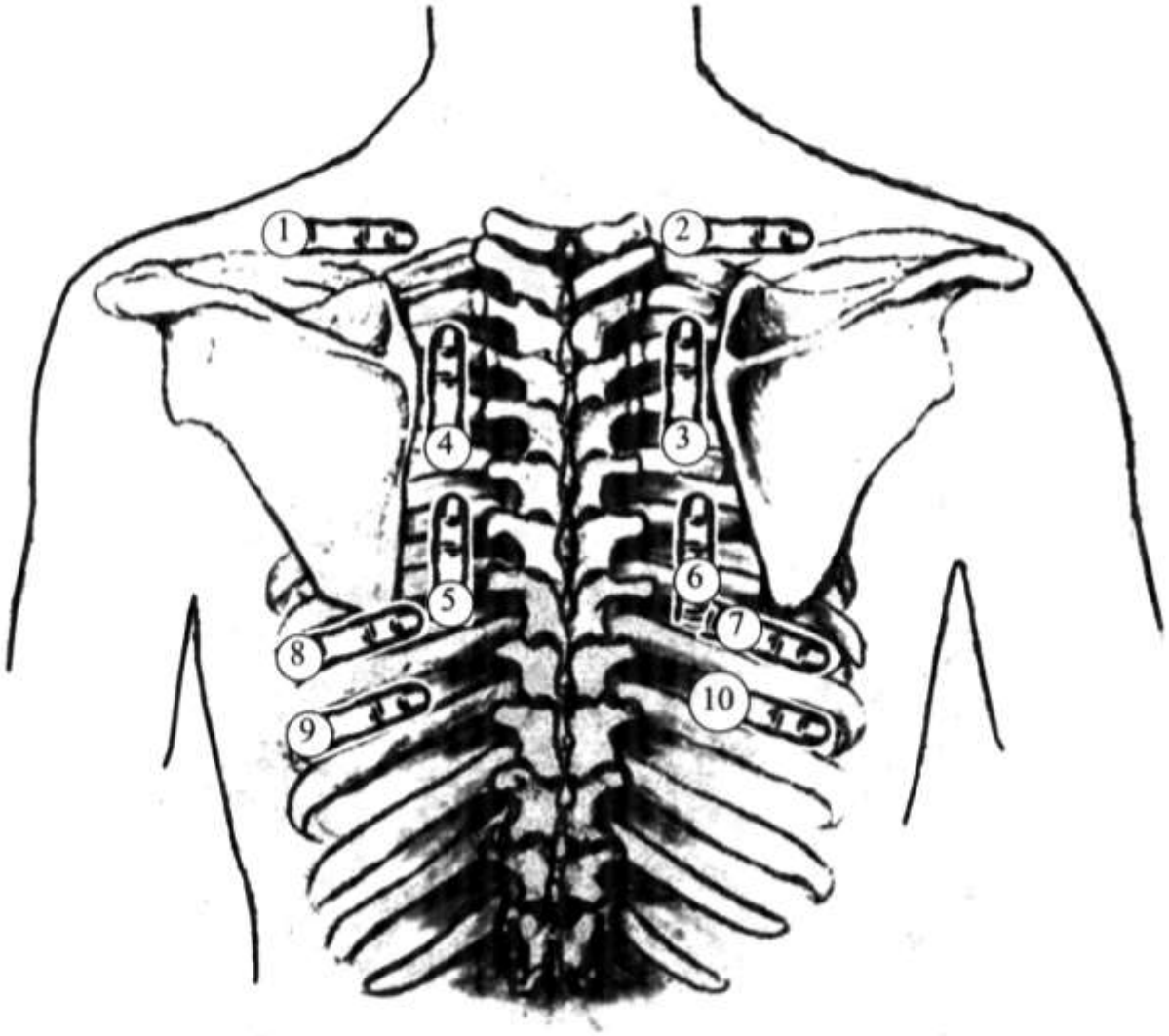


Fig. 3.30. Comparative percussion of the lungs.
Posterior view.

Percussion notes can usually be distinguished in their basic qualities of sound: intensity, pitch, and duration.

Sounds may be loud and soft in *intensity*, depending on amount of air in the underlying organs (Fig. 3.31). Sound is loud over airiness organs, and soft over airless organs.

Fig. 3.31. Intensity of the percussion sound.

The *pitch* of the sound depends on vibration frequency. Low pitched and high pitched sounds are distinguished (Fig. 3.32).

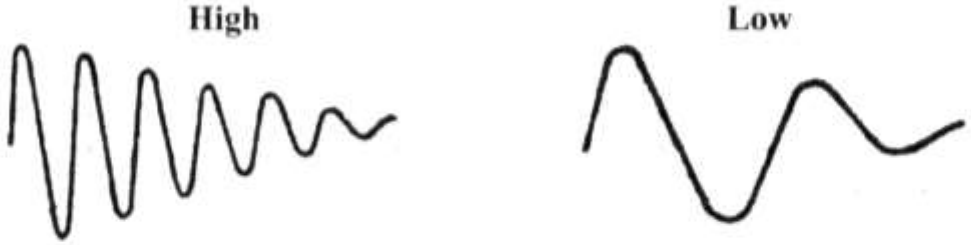


Fig. 3.32. Pitch of the percussion sound.

Percussion sounds may be of long and short *duration* (Fig. 3.33).

Fig. 3.33. Duration of the percussion sound.

It should be remembered that these qualities could be not equal in healthy person on symmetrical part of the chest (Tab. 3.2).

The air-containing lung tissue will give a clear pulmonary sound (resonance) in percussion. Comparative percussion helps to determine whether the underlying tissues are air-filled, fluid-filled, or solid. The common cause of percussion notes changes include:

- decreased airiness of the pulmonary tissue or full absence of air in a part of the lung;
- increased airiness of the pulmonary tissue;
- pleural accumulation of fluid;
- pleural accumulation of air.

Tab. 3. 2. ***Physiological changes of the percussion sounds over the lungs.***

Location	Changes of qualities	Cause
Over the right upper lobe as compared with left one	Softer and shorter	Left upper lobe locates below the right due to the shorter right main bronchus
2nd and 3rd interspaces to the left of the sternum	Softer and shorter	Close location of the heart
Over the upper lobes of the lungs as compared with lower lobes	Softer and shorter	Smaller amount of pulmonary tissue
Right axillary region as compared with left one	Softer and shorter	Close location of the liver
Left axillary region	Louder and higher (with tympanic character)	Resonant effect of the stomach

Tab. 3.3. ***Characteristics of the percussion sounds.***

Sound	Relative intensity	Relative pitch	Relative duration	Example location	Pathological examples
Clear pulmonary (resonance)	Loud	Low	Long	Normal lungs	–
Intermediate	Softer	Higher	Shorter	Heart covered by the lungs	Decreased airiness of the pulmonary tissue
Bandbox (hyper-resonance)	Very loud	Lower	Longer	None normally	Increased airiness of the pulmonary tissue
Dullness	Soft (Medium)	High (Medium)	Short	Liver (airless organs)	Consolidation of the pulmonary tissue, fluid

Tympany	Loud	High (with music tembre)	Long	Airiness organs	Large pneumothorax, cavity filled with air
Metallic				None normally	Large cavity
Cracked-pot				None normally	Superficial cavity

In decreased amount of air in the lungs clear pulmonary sound becomes duller, that is **intermediate**. Causes include:

1. lobar pneumonia initial stage, when alveoli in addition to air contain also a small amount of fluid, or when air-containing tissue alternates with consolidations;
2. pneumosclerosis, fibrous-focal tuberculosis;
3. pulmonary edema due to the left ventricular failure;
4. compressive atelectasis (above fluid level);
5. pleural adhesion or obliteration, which interferes with normal distension of the lungs during inspiration;
6. obstructive atelectasis due to gradual resorption of air from the lungs below obstruction.

Dullness replaces resonance when solid tissue replaces air-containing lungs in such conditions as:

1. acute lobar pneumonia (consolidation stage), when the alveoli are filled with the exudates;
2. formation in the lung of a large cavity, which is filled with inflammatory fluid (sputum, pus, ets);
3. pulmonary tumor (airless tissue);
4. dullness also heard when fluid occupies the pleural space (over fluid): pleural accumulation of serous fluid (pleural effusion), blood (hemothorax), or pus (empyema).

Generalized hyperresonance (**bandbox sound**) may be heard over the hyper inflated lungs of emphysema.

Unilateral **tympany** suggests a large pneumothorax or possibly a large smooth-wall air-filled cavity (bulla) communicated with bronchus (pul-

monary abscess, tubercular cavern). *Metallic percussion sound*: tympanic sound resembling a stroke on a metal may be heard over a large (6–8 cm in diameter) air-filled bulla in the lungs. *Cracked-pot percussion sound* (soft, resembles that of a cracked pot) may be heard over a large superficial cavity communicated with the bronchus through the narrow slit.

Topographic percussion of the lungs

Topographic percussion has following potential uses:

- determination of the upper borders (apices) of the lungs;
- determination of the lower borders of the lungs;
- determination of the excursion of the lower borders of the lungs.

Determine the **upper borders of the lungs** both anteriorly and posteriorly. To assess location of the lung apex *anteriorly*, place pleximeter finger parallel to the clavicle and move it gradually upwards and medially to dullness on the right side then on left one (Fig. 3.34). Normally the upper level of the lung apices is 3–4 cm above clavicle.

In order to determine the upper borders of the lungs apices *posteriorly*, place pleximeter finger parallel to the scapular spine and move it gradually upwards to the point located 3–4 cm laterally to the spinous process of the 7th cervical vertebra (C7). Normally, the upper level of the lungs apices is about at the level of the C7.

The upper borders of the lungs can vary depending on the amount of air in the apices. In increased airiness of the pulmonary tissue (emphysema, attack of the bronchial asthma) the apices increased in size and move upward. In decreased airiness of the pulmonary tissue (presence of connective tissue in the lungs as a result of inflammation in tuberculosis or pneumonia) the apices decreased in size and move downward.

In order to determine the **lower lungs borders** percussion is carried out along topographic lines (Fig. 3.5; Fig. 3.6; Fig. 3.7). First determine the lower border of the *right lung* anteriorly from second interspace along parasternal and midclavicular lines (Fig. 3.34). Lateral percussion starts from the axillary fossa along anterior axillary, midaxillary, and posterior axillary lines (Fig. 3.35). The patient should put his hands behind the back of the head.

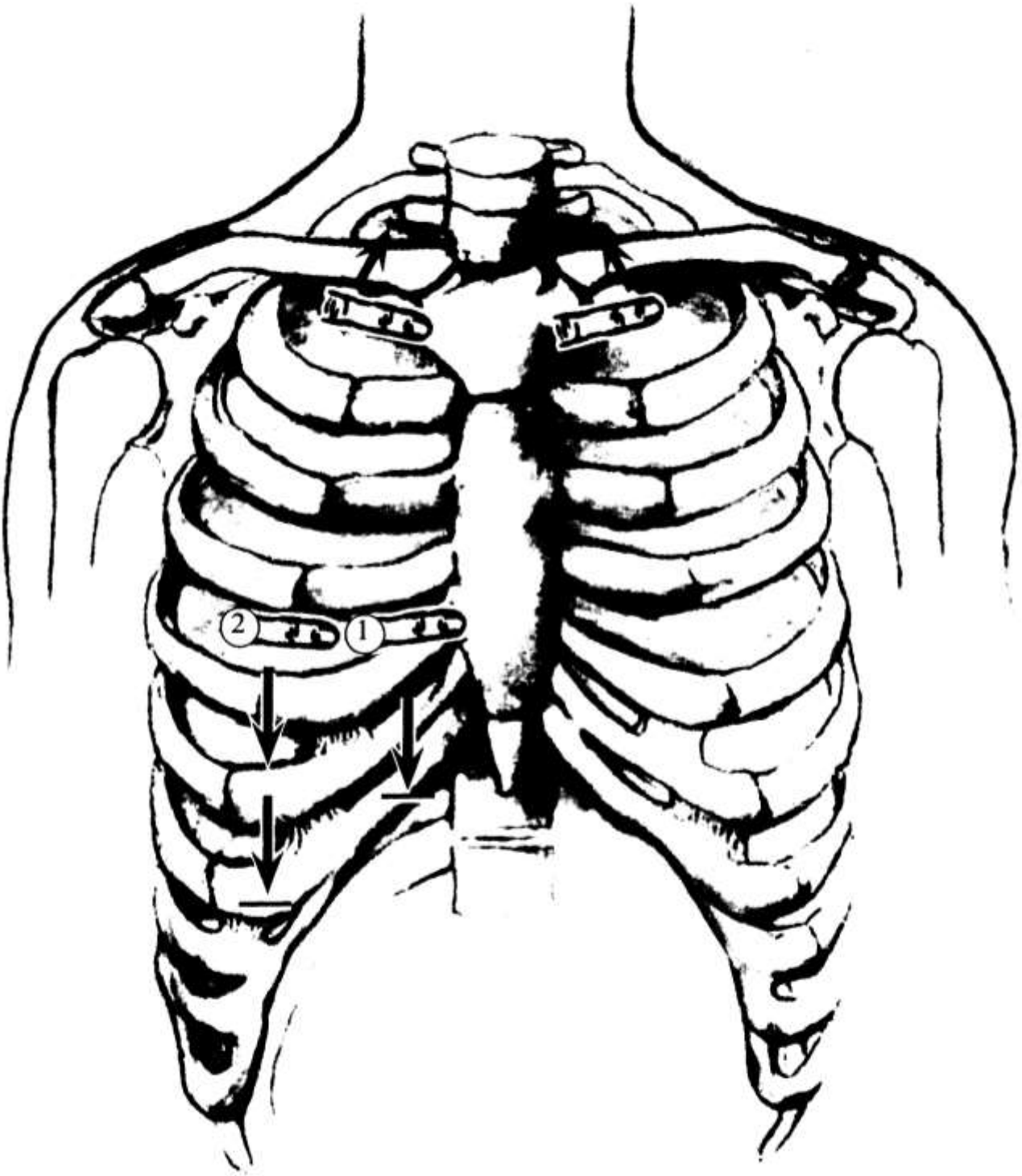


Fig. 3.34. Topographic percussion of the lungs. Anterior view.

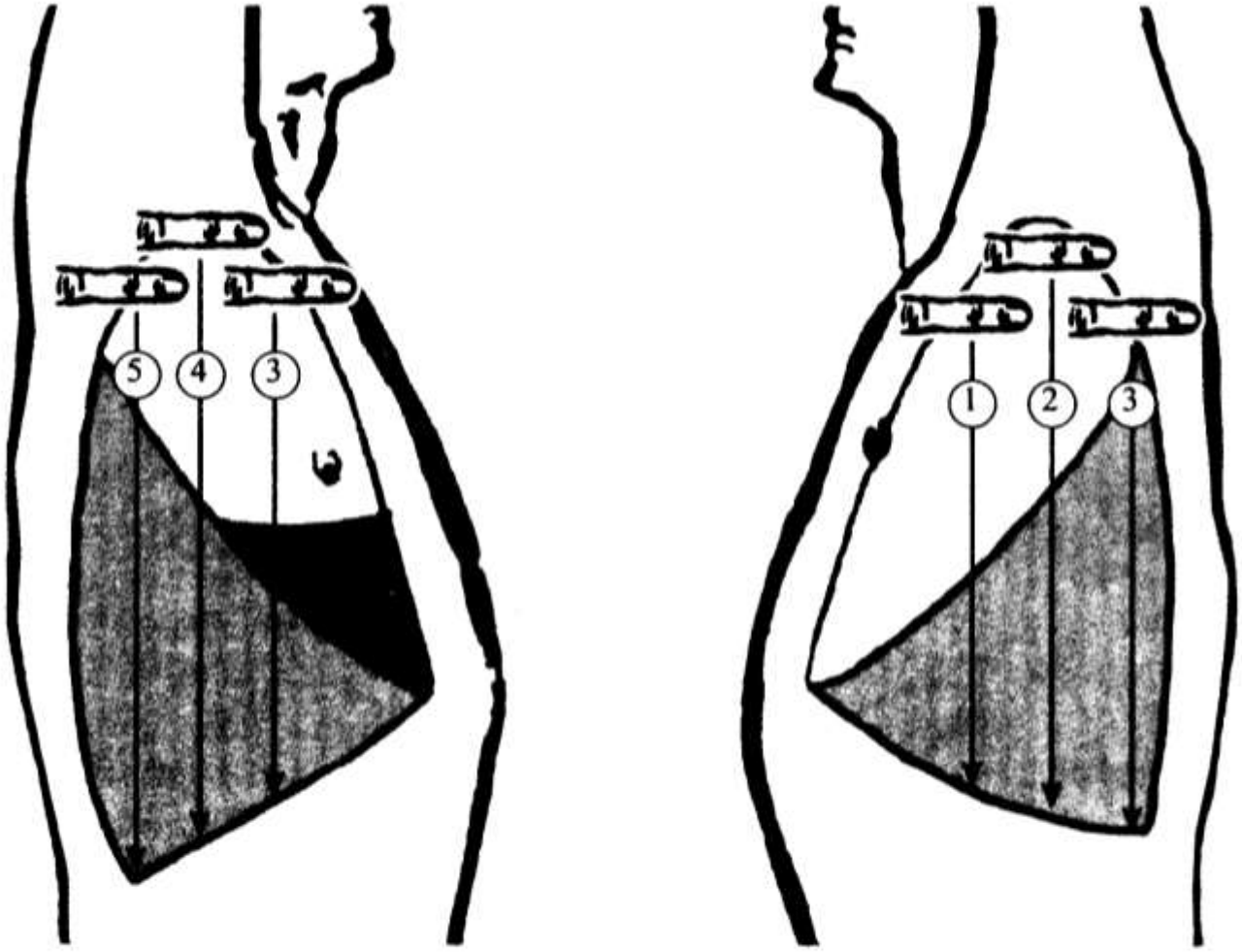


Fig. 3.35. Topographic percussion of the lungs.
Lateral view.

To outline the lower border of the right lung posteriorly, percuss from the 7th interspace (below scapular angle, which ends at the level of the 7th rib) along scapular and paraspinal lines (Fig. 3.36).

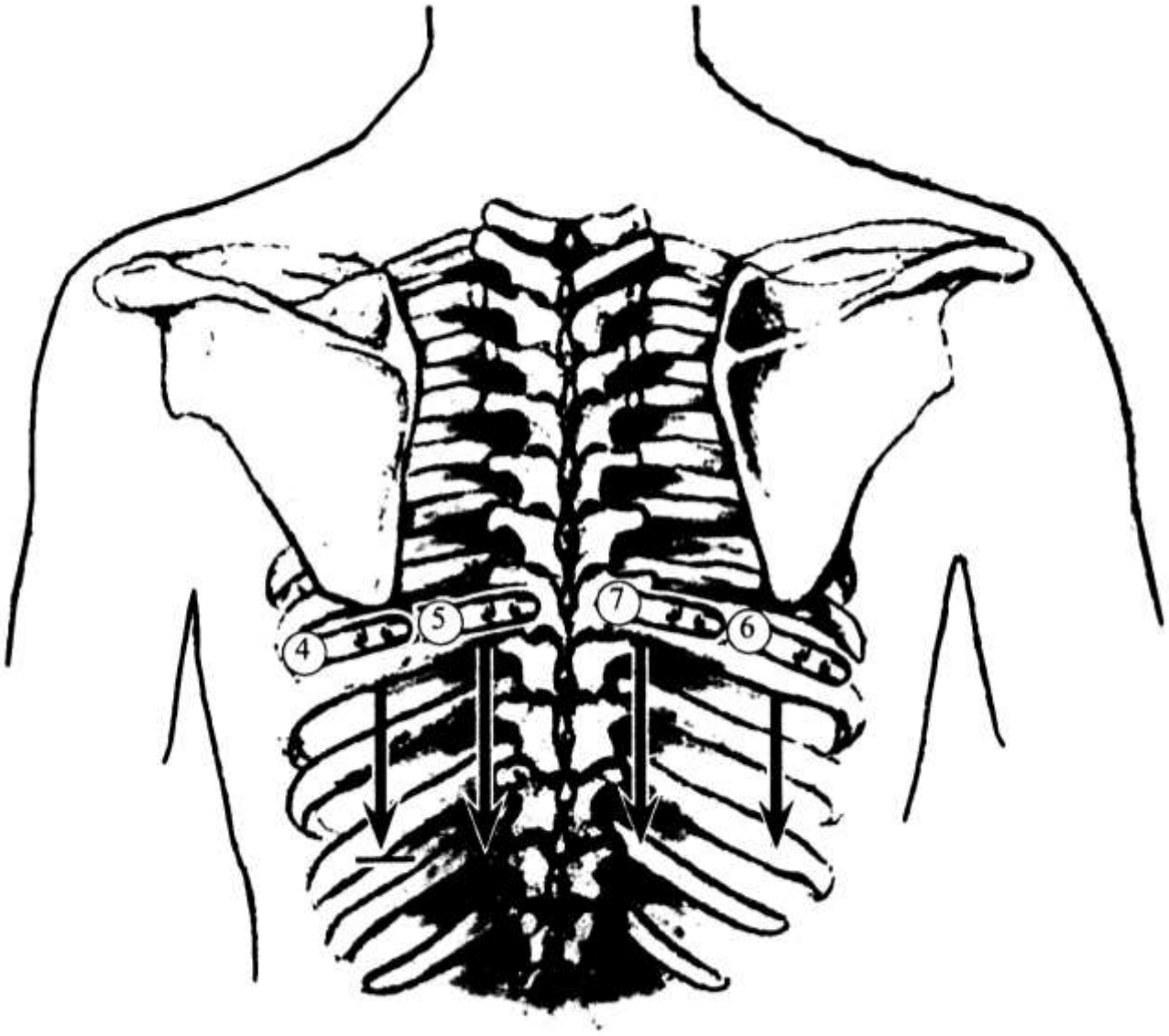


Fig. 3.36. Topographic percussion. Posterior view.

Right lung lower edge is at the point of transition of clear pulmonary sound to dullness – so-called lung-liver border (Fig.3.37; Fig.3.38).

The lower edge of the *left lung* is not determined anteriorly because of presence of the heart (Fig.3.37). Identify the lower border of the left lung only laterally along axillary lines (Fig. 3.35), and posteriorly, along scapular and paraspinal lines (Fig. 3.36). Along anterior and midaxillary lines the lower border of the left lung is at the site of transition of resonance to tympany (Traube's space) (Fig.3.37; Fig.3.38).

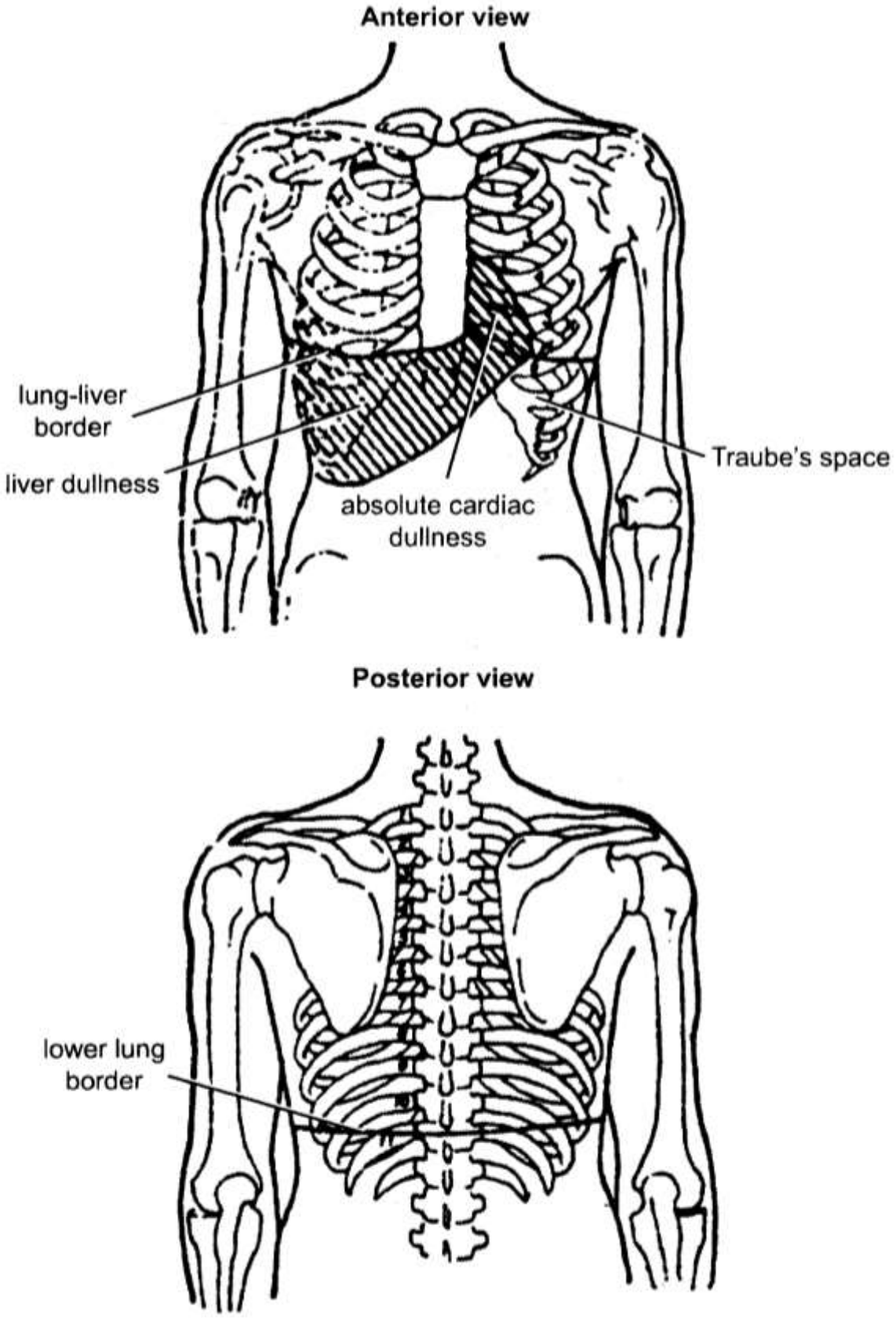


Fig. 3.37. The normal lower borders of the lungs.

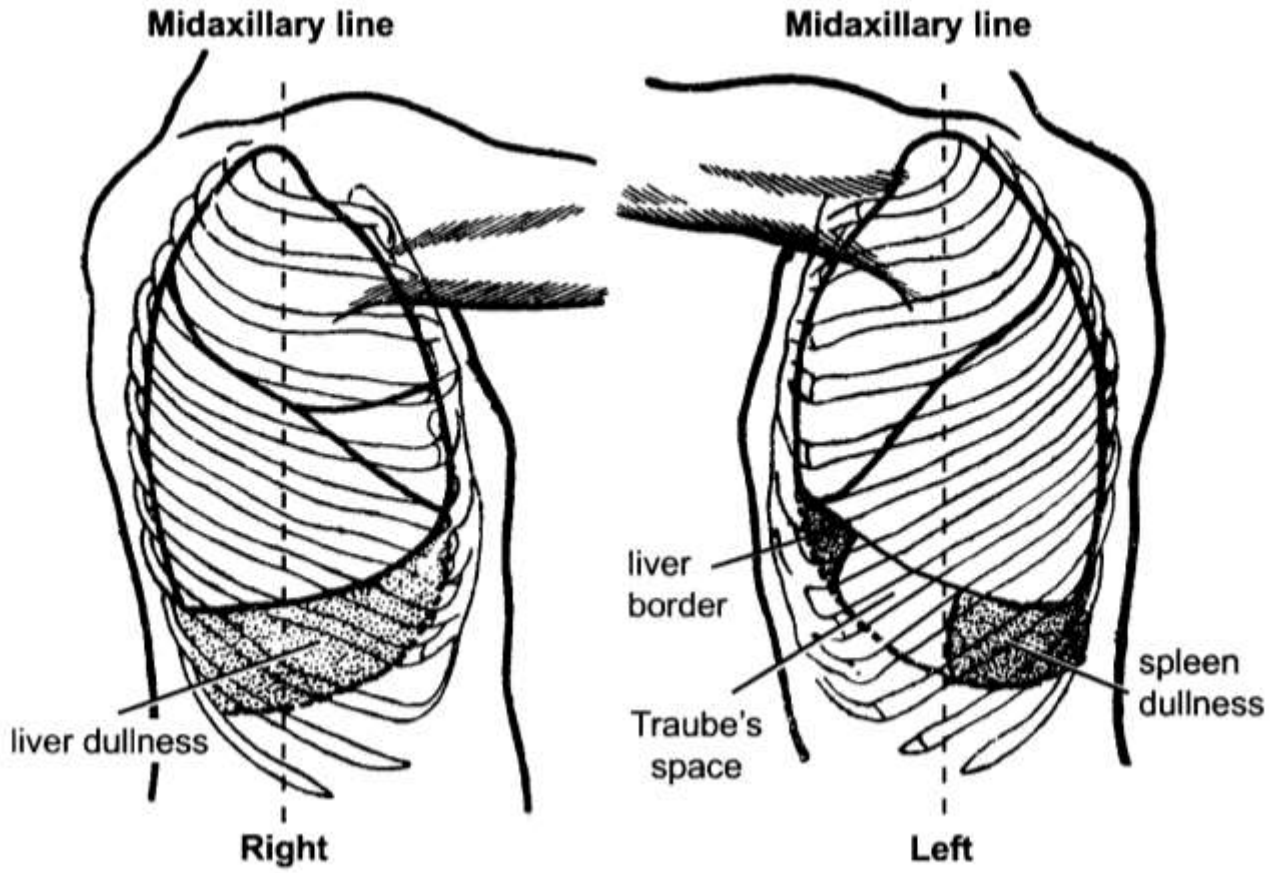


Fig. 3.38. The normal lower borders of the lungs.
Lateral view.

The normal lower borders of the lungs are represented in Tab. 3.4.

Tab. 3.4. ***Lower borders of the lungs in normosthenic persons.***

Topographic lines	Right lung	Left lung
Parasternal	5th interspace	–
Midclavicular	6th interspace	–
Anterior axillary	7th interspace	7th interspace
Midaxillary	8th interspace	8th interspace
Posterior axillary	9th interspace	9th interspace
Scapular	10th interspace	10th interspace
Paraspinal	Spinous process of T11	Spinous process of T11

Displacement of the lower border of the lung can be bilateral or unilateral.

Bilateral lowering of the lower lungs edges is observed in:

- asthenic persons;
- in acute dilation of the lungs (attack of bronchial asthma);
- in chronic dilation of the lungs (emphysema).

Unilateral lowering of the lower lung edge is observed in:

- compensatory emphysema of one lung with inactivation of the other (pleural effusion, hydrothorax, pneumothorax, hemiparesis of the diaphragm).

Bilateral elevation of the lower lungs edges is observed in high diaphragm level:

- in hypersthenic persons;
- temporary in late pregnancy;
- ascitis;
- meteorism;
- presence of air in abdomen due to acute perforation of gastric or duodenal ulcer.

Unilateral elevation of the lower lung edge is observed in:

- pneumosclerosis;
- obstructive atelectasis;
- compressive atelectasis;
- marked enlargement of the liver or the spleen.

Respiratory excursion may be estimated by noting the distance between the levels of lower pulmonary borders on full expiration and full inspiration.

Identify respiratory mobility by right midclavicular, midaxillary, and scapular lines, and also by left midaxillary and scapular lines (Tab. 3.5).

Respiratory mobility of the lower border of the lungs is decreased in inflammatory processes, decreased elasticity of the pulmonary tissue (emphysema), pleural effusion, adhesion or obstruction.

Tab. 3.5. ***Respiratory excursion of the lower border of normal lungs in cm.***

Topographic lines	Right lung			Left lung		
	Inspiration	Expiration	Total	Inspiration	Expiration	Total
Midclavicular	2–3	2–3	**4–6**	–	–	–
Midaxillary	3–4	3–4	**6–8**	3–4	3–4	**6–8**
Scapular	2–3	2–3	**4–6**	2–3	2–3	**4–6**

Auscultation of the Lungs

Auscultation of the lungs is the most importing examining technique for assessing airflow through the tracheobronchial tree.

Auscultation involves:

1. listening the sounds generated by breathing – breath sounds (respiratory sounds);
2. listening for any adventitious (added) sounds.

Auscultation technique. Listen to the respiratory sounds with the diaphragm of a stethoscope after instructing the patient to breath deeply through a nose with close mouth. Be alert for patient discomfort due to hyperventilation (light headedness, faintness), and allow the patient to rest as need.

As auscultation is also comparative method, use the pattern suggested for comparative percussion, moving stethoscope from one side to the other and comparing symmetrical areas of the lungs. Listen to at least one full breath in each location.

Listen to the breath sounds in supra-, infraclavicular regions, and them move stethoscope downward. In the left 2nd and 3rd interspaces place stethoscope more laterally, as compared with percussion, in order to round the heart (Fig. 3.39).

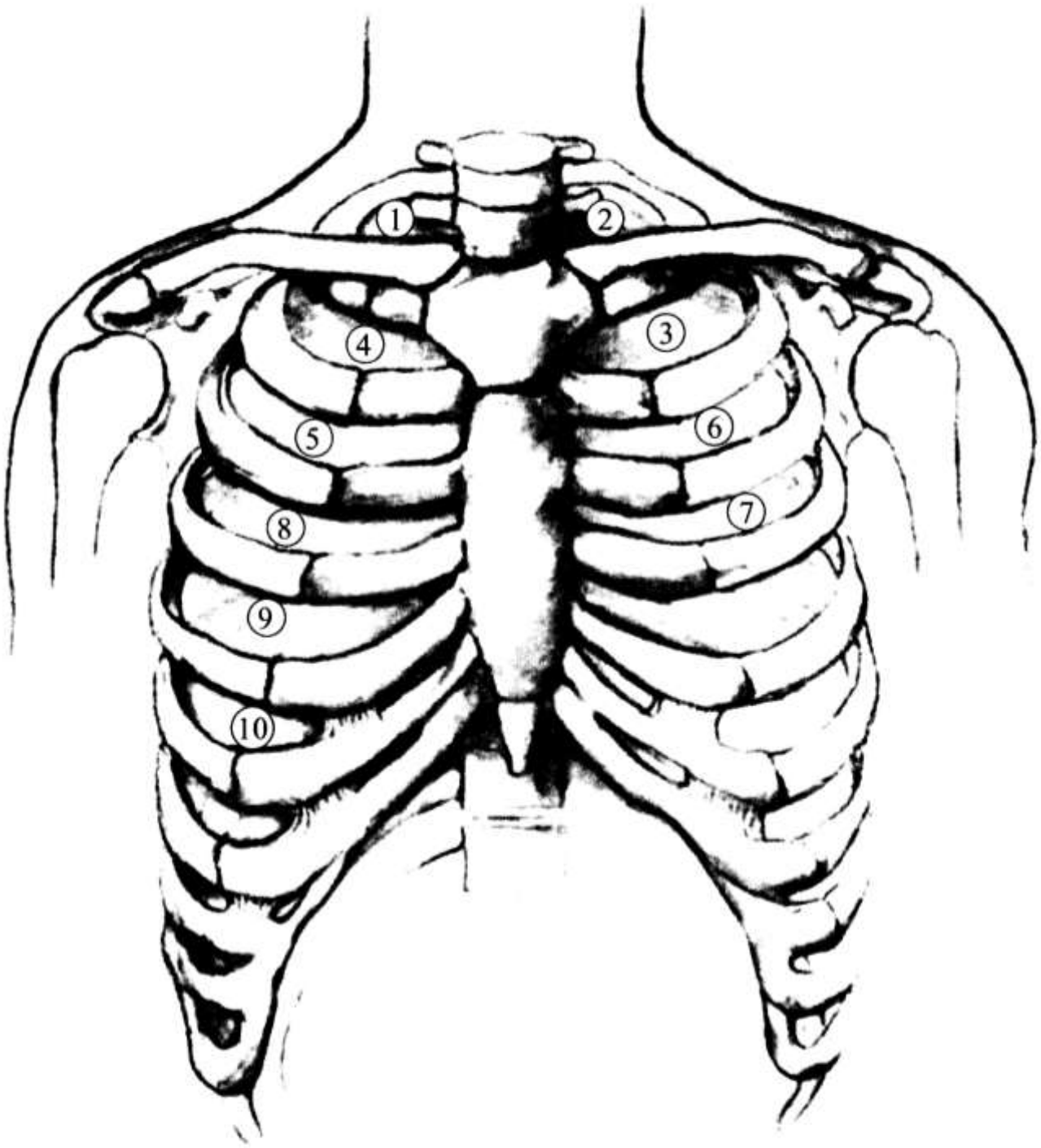


Fig. 3.39. Auscultation of the lungs.
Anterior view.

The lungs are then auscultated in the axillary regions with the patient's hands on the back of the head (Fig. 3.40).

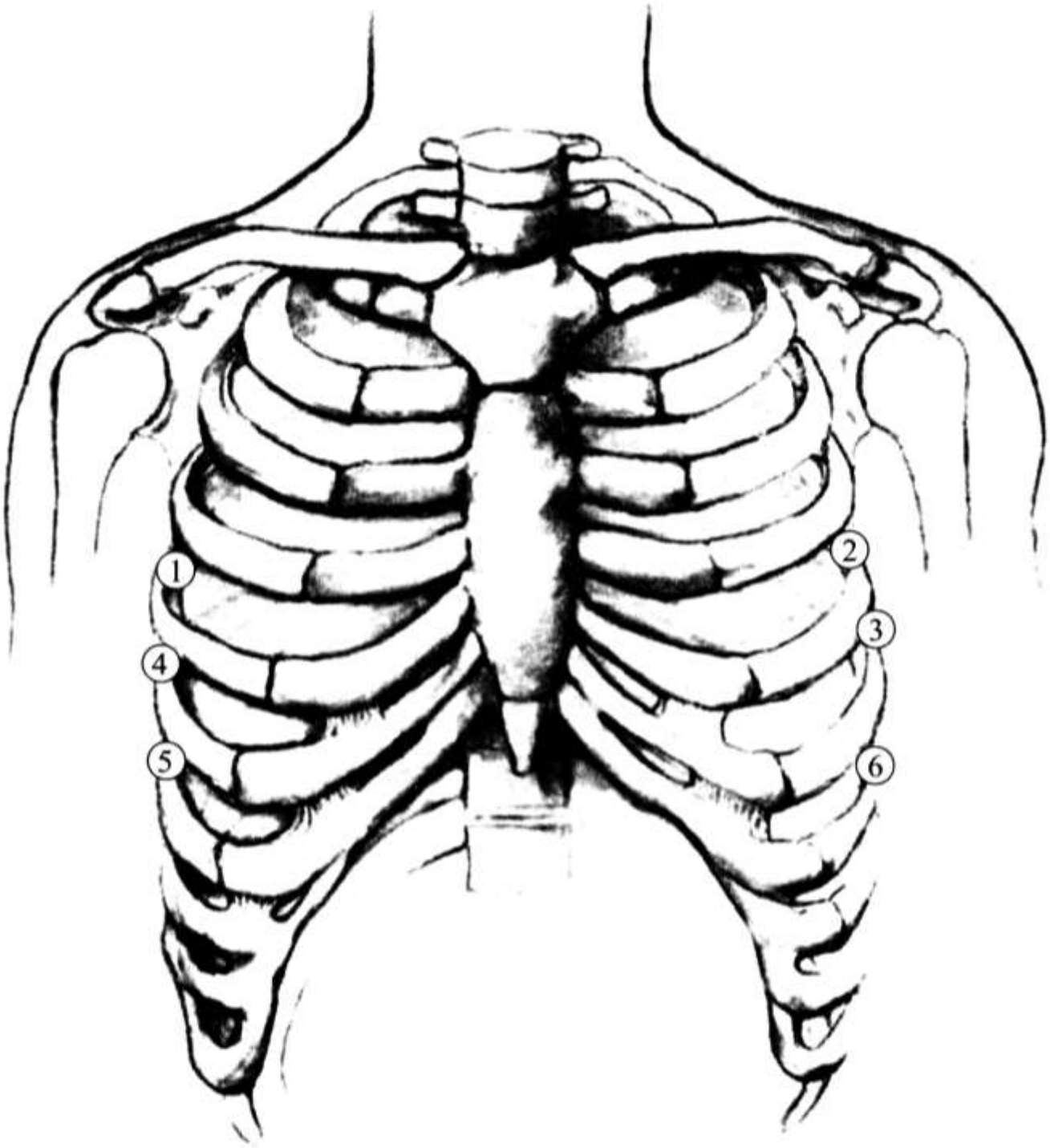


Fig. 3.40. Auscultation of the lungs in axillary regions.

Listen to the breath sounds posteriorly in supra-, inter-, and infrascapular regions (Fig. 3.41). Ask the patient to cross his arms on the chest to move scapular from the spine.

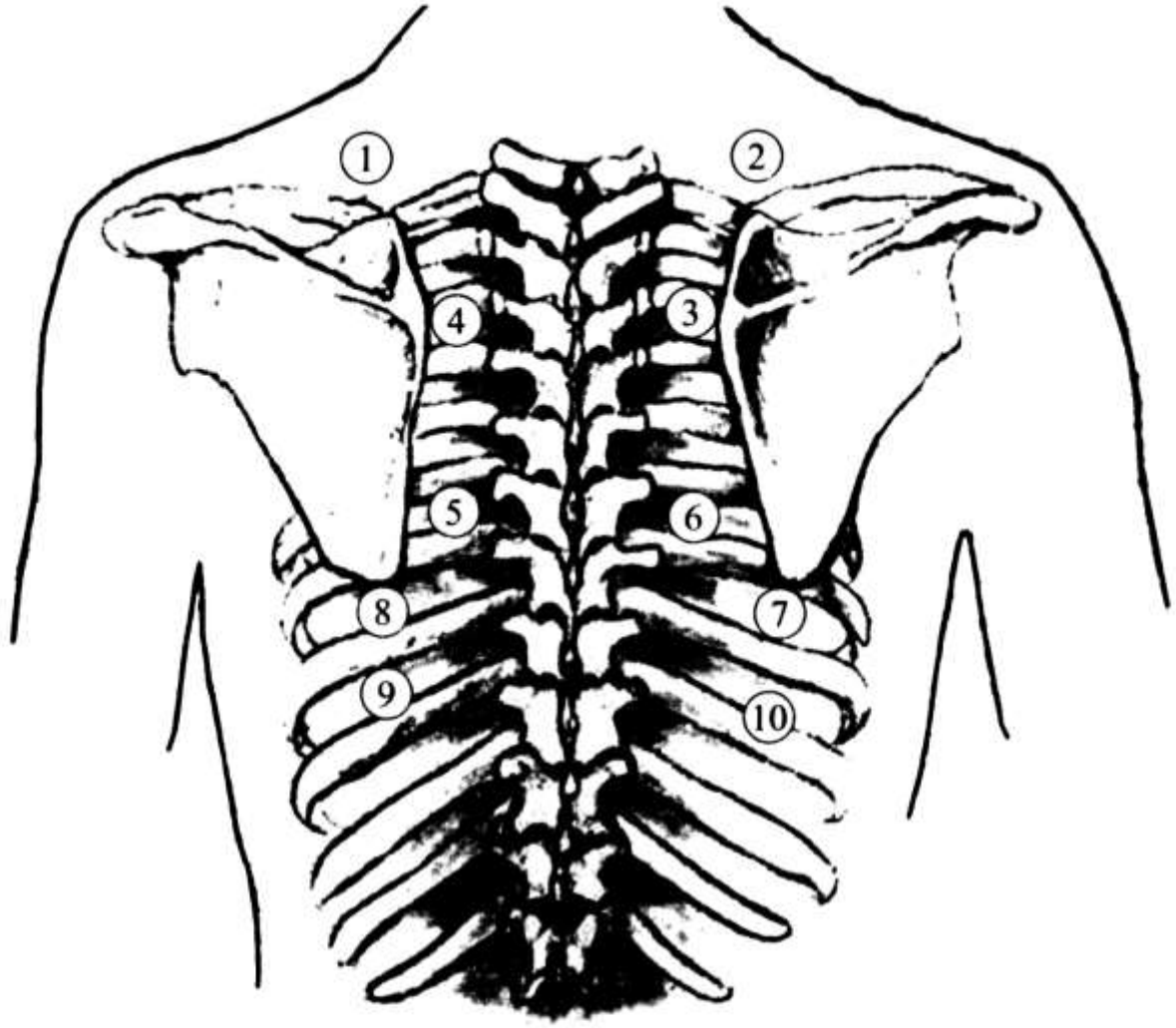


Fig. 3.41. Auscultations of the lungs. Posterior view.

Two types of sound can be heard coming from the lungs: the **main respiratory sounds** (breath sounds) and **adventitious** (added) **sounds** (Tab. 3.6). Breath sounds may be normal or abnormal, added sounds are always abnormal.

Tab. 3.6. ***Lungs sounds.***

Lung sounds	
Main respiratory (breath) sounds	**Adventitious** (added) sounds
• vesicular (alveolar) breath sounds • bronchial (laryngotracheal) breath sounds	• rales • crepitation • pleural friction sounds

The main respiratory sounds (breath sounds). Normal breath sounds have been classified into two categories: vesicular and bronchial, according to their intensity, their pitch, and the relative duration of their inspiratory and expiratory phases.

Vesicular breath sounds are soft, low pitched, and are heard through inspiration, continue about one third of way through expiration (Fig. 3.42).

Fig. 3.42. Vesicular breath sound.

Breath sounds known as vesicular breathing are generated by vibration of the alveolar walls due to airflow in inspiration. A long soft (blowing) noise gradually increases and is heard through inspiration. Alveolar walls still vibrate during initial stage of expiration to give shorter expiratory sound during about one third of the expiration phase. Vesicular breathing is also therefore called – *alveolar breathing*.

Vesicular breath sounds are heard normally over most of both lungs. It should be remembered however that intensity of vesicular breathing is differ over healthy lungs (Tab. 3.7).

Tab. 3.7. ***Physiological difference of the vesicular breath sounds.***

Intensity	Location	Cause
More loud	• below the 2nd rib, laterally of the parasternal line; • axillary regions; • below scapular angle	Largest masses of the pulmonary tissue
Longer and louder	• over the right lung as compared with left one	Better conduction by the right main bronchus, which is shorter and wider
Less loud	• lung apices; • lowermost lungs parts	Smallest masses of the pulmonary tissue

Vesicular breath sounds can vary for both physiological and pathological causes.

Physiological changes of the vesicular breathing always involve both part of the chest, and breath sounds are equally changes at the symmetrical points of the chest (Tab. 3.8).

Tab. 3.8. ***Physiological changes of the vesicular breath sounds.***

Decreased vesicular breathing	**Increased** vesicular breathing
Thick chest wall: • excessively developed muscles or • subcutaneous fat	Thin chest wall: • underdeveloped muscles or • subcutaneous fat in children (good elasticity of the alveoli). This type of breathing is called '*puerile*' (L *puer* – child) During exercise

Pathologic changes of the vesicular breathing can be a result of following causes:

1. *abnormal generation of breath sounds*, which depend on amount of intact alveoli, properties of their walls, and amount of air contained in them;
2. *abnormal transmission of the breath sounds* from the vibrating elastic elements of the pulmonary tissue to the surface of the chest.

Abnormalities in vesicular breath sounds may be unilateral, bilateral, or only over a limited area of the lung. Vesicular breathing can be decreased or inaudible, and increased (Fig. 3.43).

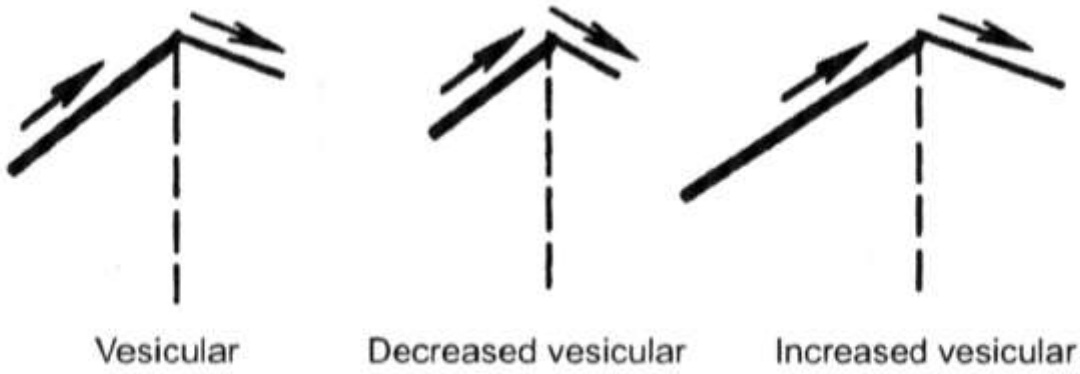


Fig. 3.43. Vesicular breath sounds and their changes.

Pathologically decreased vesicular breathing observes in:

I. abnormal generation of breath sounds occurs in:

- *pulmonary emphysema,* when the number of the alveoli significantly diminished. The remaining alveoli are no longer elastic, their walls become incapable of quick distension, and do not give sufficiently strong vibration;
- *initial stage of acute lobar pneumonia* due to inflammation and swelling of alveolar walls and decreased their vibrations. Vesicular breath sounds becomes *inaudible* during the *second stage of acute pneumonia*, when alveoli of affected lobe are filled with effusion;
- *obstructive atelectasis*, when airflow is decreased (over atelectasis zone). In complete obstruction breath sounds are inaudible;
- *compressive atelectasis,* when alveoli are compressed, and airflow in them is decreased;
- *inflammation of the respiratory muscles, intercostals nerves, rib fracture, muscular weakness* as a result of marked weak inspiration.

II. abnormal transmission of breath sounds results from:

- thickening of the pleural layers;
- pleural effusion;
- pneumothorax.

Pathologically increased vesicular breathing occurs when air flows at increased speed through narrowed airways (inflammatory edema of the mucosa, bronchospasm) in bronchitis and bronchial asthma. This increase in speed increases turbulence, the amount of noise made, and expiration become louder and longer.

Deeper vesicular breathing when inspiration and expiration are intensified is called **harsh**. This type of increased vesicular breathing can observe in bronchitis as a result of marked and nonuniform narrowing of small bronchi and bronchioles due to inflammatory edema of their mucosa.

Interrupted or cogwheel respiration is characterized by short jerky inspiratory efforts interrupted by short pauses between them (Fig. 3.44).

Fig. 3.44. Interrupted or cogwheel respiration.

Such type of respiration can be observed in non-uniform contraction of the respiratory muscles, when you listen patient in cold room, in nervous trembling, and sometimes in children during crying. Cogwheel respiration over limited area indicates difficult airflow from small bronchi to the alveoli, and also uneven unfolding of the alveoli. Interrupted breathing indicates pathology in fine bronchi and is more frequently heard over lungs apices during their tubercular infiltration.

Fig. 3.45. Bronchial breath sound.

Bronchial breath sounds are loud, harsh, high in pitch, and expiratory sound last longer than inspiratory one (Fig. 3.45).

Bronchial breath sounds are generated by turbulent airflow in the larynx and the trachea when air passes through the vocal slit. Since the vocal slit is narrower during expiration, expiratory sounds are louder, harsher, and longer. This type of breath sounds is also called *laryngotracheal*.

Bronchial breathing is heard normally over the larynx, the trachea in the neck, and at the site of projection of the tracheal bifurcation (anteriorly over manubrium, and posteriorly in the interscapular region at the level of T3 and T4 spinous processes) (Fig. 3.46).

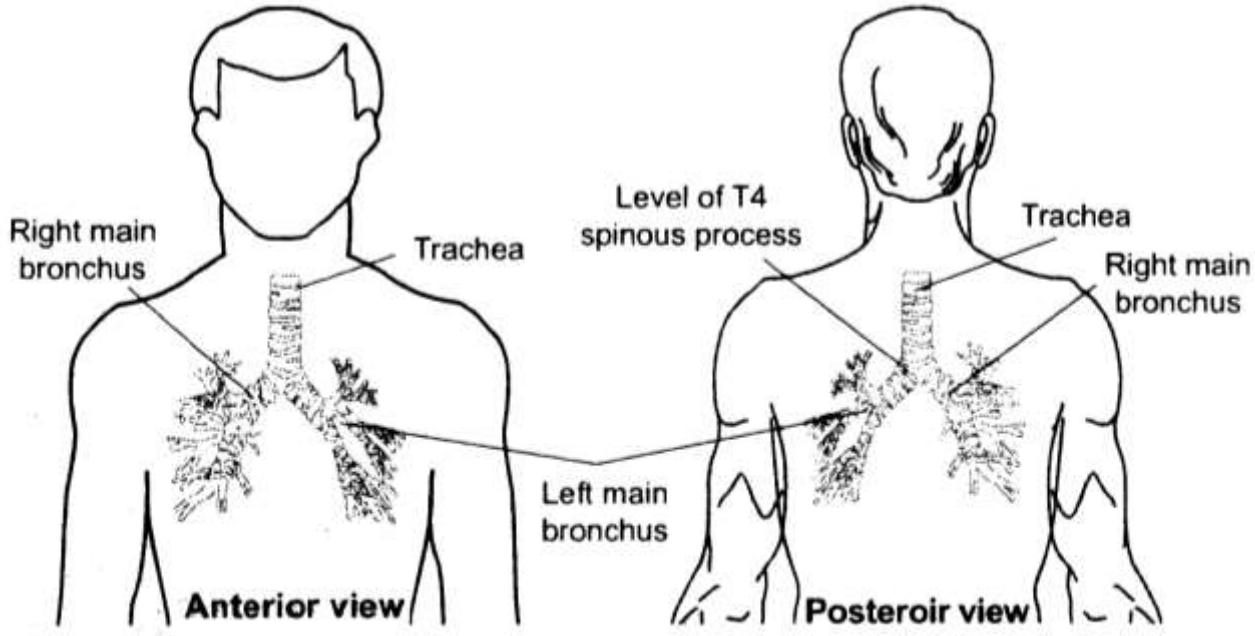


Fig. 3.46. Trachea and main bronchi projection on the chest.

Bronchial breath sounds are inaudible over the lungs because bronchi are covered by air-containing 'pillow' of the pulmonary tissue.

If bronchial breathing is heard over the lungs, suspect that air-filled lung has been replaced by fluid-filled or solid lung tissue, which conducts sounds better. This is so-called pathological bronchial breathing.

Pathological bronchial breathing is observed in ***consolidation of the pulmonary tissue*** in:

- acute lobar pneumonia, tuberculosis (when alveoli are filled with effusion);
- *lung infarction* (when the alveoli are filled with blood);
- *lung tumor* (airiness tissue);
- *compressive atelectasis* (when alveoli are compressed completely by pleural air or fluid);
- *pneumosclerosis, carnification of the lung lobe* (airless connective tissue replace airiness lung tissue);
- in formation of an ***empty cavity*** in the lung communicated with a large bronchus:
- pulmonary abscess;
- cavernous tuberculosis;
- disintegrated tumor;
- *disintegrated lung infarction*;
- seldom *opened echinococous*.

Solid pulmonary tissue round the cavity transmits the breath sounds better, and the sounds are intensified in the resonant cavity.

Amphoric respiration is heard in the presence of a large smooth-wall cavity (not less than 5–6 cm in diameter) communicated with a large bronchus and situated superficially. A strong resonance causes additional high overtones, which alter the main tone of the bronchial breath sounds. Blowing over the mouth of an empty glass or clay jar can produce such sounds. This altered bronchial breathing is therefore called amphoric (GK *amphoreus* – jar).

Bronchovesicular or mixed breathing is intermediate in intensity and pitch, inspiratory and expiratory sounds are about equal (inspiratory sounds is characteristic of vesicular breathing, expiratory of bronchial breathing) (Fig. 3.47).

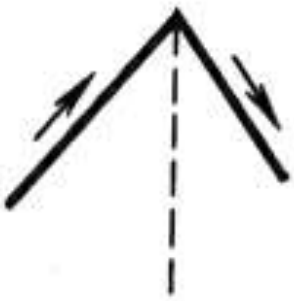


Fig. 3.47. Broncho-vesicular breathing.

Such type of breath sounds are heard when solid lung tissue locates deep or far from one another.

The characteristics of the main respiratory sounds are summarized in the Tab. 3.9.

Adventitious (added) sounds. Three types of adventitious sounds can be heard in pulmonary pathology: rales, crepitation, and pleural friction sound.

Rales are generated in bronchi and bronchioles. Dry and moist rales are distinguished.

Dry rales can be caused by narrowing of airways, or by presence of viscous sputum in them (Fig. 3.48).

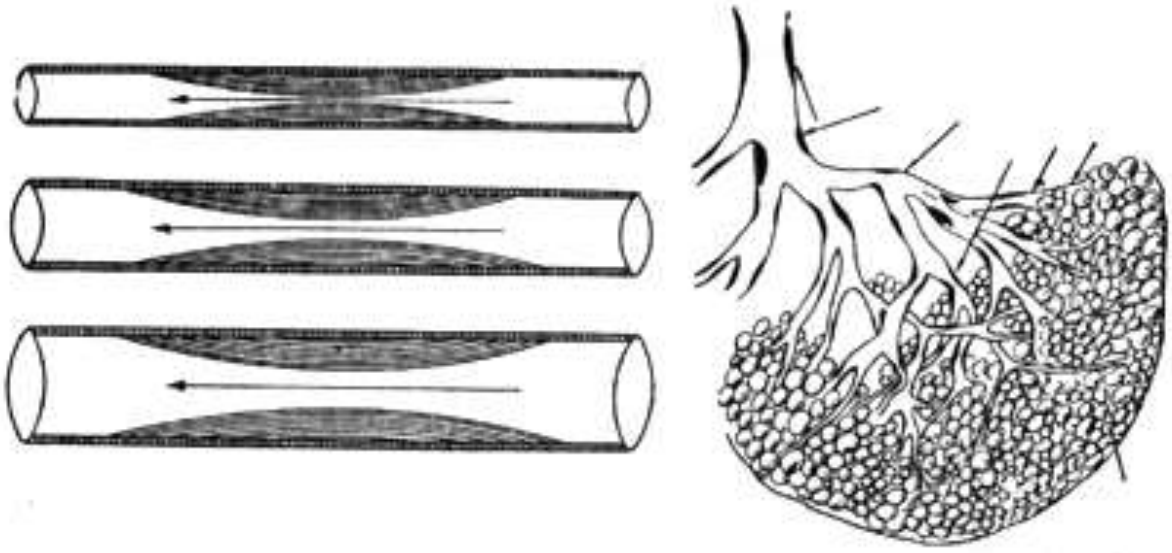


Fig. 3.48. Mechanism and site of dry rales generation.

Dry rales are continuous musical sounds, persist throughout the respiratory cycle, and vary greatly in their character, pitch, and intensity. Depending on character and pitch, dry rales are divided into sibilant and sonorous rales.

Sibilant rales (wheezes) are relatively high pitched, whistling sounds (Fig. 3.49).

Tab. 3.9. ***The characteristics of the main respiratory sounds.***

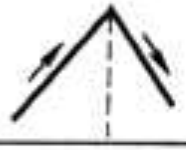
Sound	Duration	Intensity of the expiratory sound	Pitch of the expiratory sound	Example location	Pathologic example
Vesicular 	Inspiratory sounds last longer than expiratory one	Soft	Low	Over most of both lungs	–
Decreased vesicular 	Inspiratory and expiratory sounds last shorter	Softer	Low	None normally	Emphysema, acute pneumonia, obstructive atelectasis, muscular weakness, hydrothorax, pneumothorax
Increased vesicular 	Inspiratory and expiratory sounds last longer	Louder	Low	None normally	Bronchial asthma, bronchitis
Cogwheel 	Interrupted inspiration	Relatively soft	Relatively low	Cold room, nervous trembling	Diseases of the respiratory muscles, pathology in fine bronchi (tuberculosis)
Bronchial 	Expiratory sounds last longer than inspiratory one	Loud	Relatively high	Over the larynx, the trachea, manubrium, interscapular region (at the level of T3, T4)	Over the lungs in consolidation of the pulmonary tissue (acute lobar pneumonia, tuberculosis, lung infarction, compressive atelectasis), cavity in the lungs (abscess, caverna)
Broncho-vesicular 	Inspiratory sounds and expiratory sounds are about equal	Intermediate	Intermediate		Deep location of the solid lung tissue

Fig. 3.49. Sibilant rales.

Sibilant rales signify obstruction in small bronchi in:

- bronchial asthma (total bronchospasm during attack);
- *bronchitis* (non-uniform swelling of the bronchial mucosa due to inflammation, or viscous sputum narrows the lumen of bronchi);
- *tuberculosis or tumor of bronchus* (localized constriction of the bronchus. Limited dry rales over apex of the lung can suggest early symptom of tuberculosis).

Sonorous rales (rhonchi) are relatively low pitched, sonoring sounds (Fig. 3.50).

Fig. 3.50. Sonorous rales.

Sonorous rales are generated by vibration of the viscous secretions or in widespread obstruction of medium and large bronchus. The most common cause of sonorous rales is bronchitis. They may be also heard in bronchial asthma, tuberculosis, and bronchocarcinoma.

Intensity and transmission of the dry rales depends on the size and depth of the affected bronchi. In localized affection of medium and large bronchus insignificant amount of low pitched and soft rales is heard. Widespread bronchi inflammation or bronchospasm in asthma attack both sibilant and sonorous rales of different tone and intensity are heard. Such rales can be heard at a distance during expiration. If dry rales are caused by accumulation of the viscous secretions in the lumen of bronchi, they can be altered by coughing or deep inspiration to shift mucus.

Moist rales (clackles) are generated in bronchi and cavities in the lungs in the presence of liquid secretions (sputum, congestive fluid, blood).

Airflow in liquid-containing bronchi causes formation of air bubbles, which break to produce specific cracking sound. Similar sound can be heard

when bubbling air through the water using small tube (Fig. 3.51). Such sounds are called bubbling or moist rales.

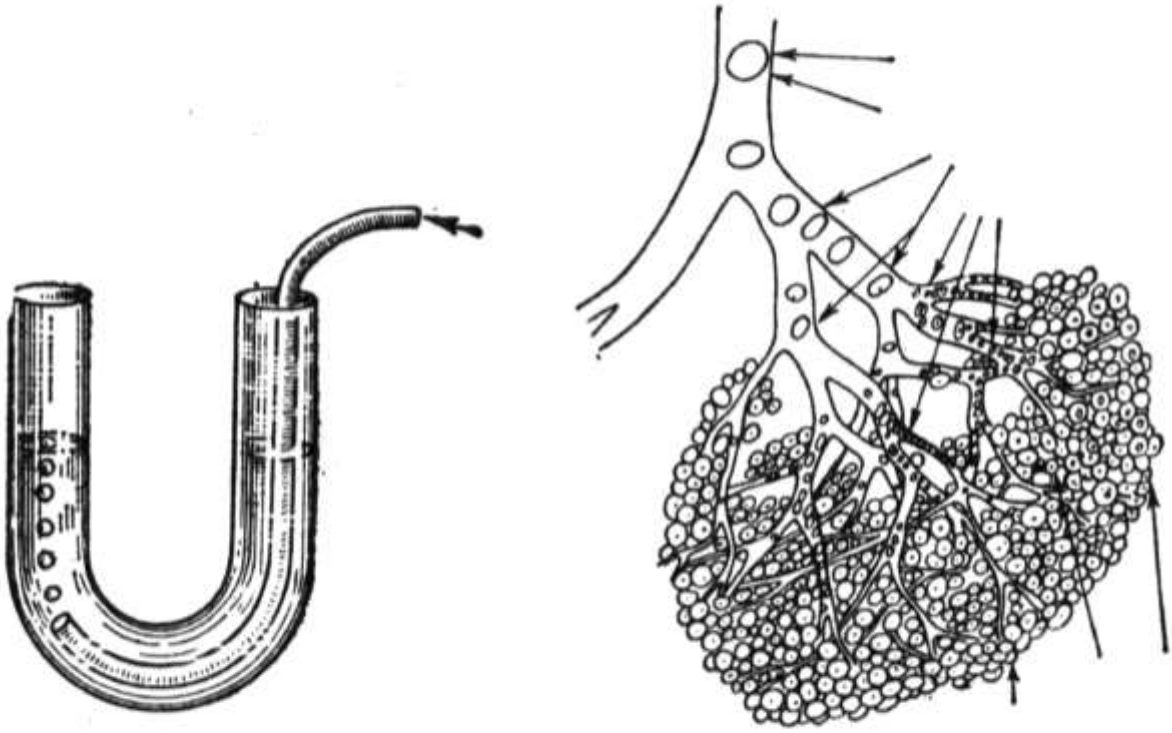


Fig. 3.51. Mechanism and site of moist rales generation.

Moist rales are discontininuous sounds, intermittent, nonmusical, and brief. Moist rales are heard throughout the respiratory cycle, but as speed of airflow in inspiration is higher, rales are somewhat louder during inspiration.

Moist rales are subdivided into fine, medium, and coarse bubbling rales depend on the caliber of bronchi where they are originated.

Fine bubbling rales (. . . . .) generate in small bronchi and bronchioles. These rales are soft, high-pitched, and very brief.

Medium bubbling rales (. . . . .) originate in bronchi of medium caliber. They are somewhat louder, and not so brief.

Coarse bubbling rales (• • • • • • •) produce in bronchi of large caliber, large bronchiectasis, and also in fluid-containing lung cavity communicated with large bronchus (Fig. 3.52). Coarse bubbling rales are loud, low-pitched, and longer.

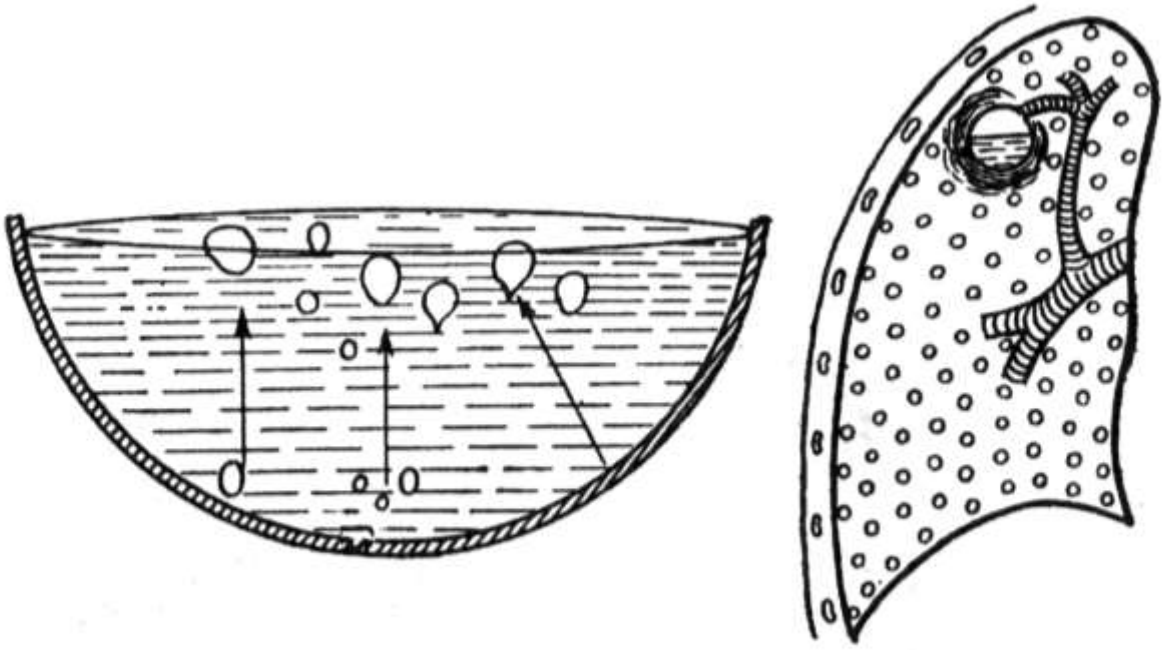


Fig. 3.52. Mechanism of the coarse bubbling rales generation over the cavity.

Moist rales are classified into consonating and non-consonating rales.

Consonating rales are heard when liquid-containing bronchi or cavity are surrounded by solid lung tissue. The cavity itself act as resonator to intensify loudness of rales.

Non-consonating rales are heard in bronchitis or acute pulmonary edema caused by left ventricular failure, when intensity of rales produced are dampened by air-containing lung tissue.

The most common causes of the moist rales include:

- acute and chronic bronchitis (bilateral, symmetrical, of various caliber, non-consonating rales);
- *bronchopneumonia* (consonating rales);
- *bronchiectasis* (of various caliber, over limited area, non-consonating rales);
- *cavity in the lungs* (coarse bubbling, over limited area, as rule over lung apices, consonating rales);
- *pulmonary edema* due to the left ventricular failure (bilateral symmetrical, of different caliber, non-consonating rales).

Crepitation is generated in alveoli, when they contain small amount of liquid secretion. During expiration alveoli stick together as a result of fluid presence. During inspiration alveolar walls separate with difficulty only

at the end of inspiration to produce late inspiratory slight cracking sound (Fig. 3.53). Crepitation somewhat resembles sound produced by rubbing a lock of hair near the ear.

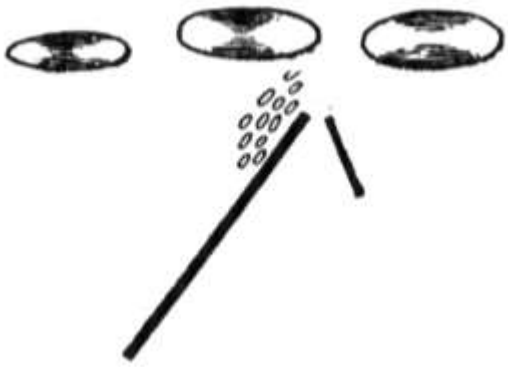


Fig. 3.53. Mechanism of crepitation.

Temporary crepitation in first deep inspiration can be heard in the patients with grave cardiovascular and infectious diseases, in aged persons, especially so if the patient was in lying posture before auscultation.

Relatively constant crepitation can be due to:

- a c u t e l o b a r pneumonia at the initial and final stages (insignificant amount of exudates at the initial stage causes so-called indux crepitation – quit, remote sound. During next stage of disease alveoli are overfilled with inflammatory fluid and crepitation therefore disappears. At the final stage due to resolution of exudates, loud, as near the ear, crackling sound – redux crepitation is heard again);
- *pulmonary tuberculosis* (in small amount of inflammatory fluid in alveoli);
- *lung infarction* (in small amount of blood in alveoli);
- *congestive heart failure* (in small amount of congestive fluid in alveoli);
- *compressive atelectasis* (alveoli are compressed by pleural air or fluid, and separate therefore with difficulty).

Pleural friction sound (pleural rub, friction rub) is diagnostic added sound of pleurisy.

The smooth surfaces of visceral and parietal pleura lubricated by pleural fluid, allow pleura to move easily and noiseless during breathing.

Adventitious sound known as pleural friction sound, generates as a result of *decreased amount of pleural fluid* in dry pleurisy due to dehydrotation:

- intestinal infections (cholera, dysentery);
- profuse bleeding;
- profuse diarrhea;
- profuse vomiting;

or cicatrices, commissures, bands between pleural layers at the focus of inflammation, or when fibrin deposits on inflamed *pleura* to make it *surface rough* in:

- pleuropneumonia;
- rheumatic pleurisy;
- pleural tuberculosis;
- tumor;
- effusive pleurisy at the period of rapid resorption of exudates.

Pleural friction rub is heard throughout inspiration and expiration, and is differentiated by intensity, location and duration. A soft friction rub in early dry pleurisy may be mistaken for crepitation or fine bubbling rales but is not altered by coughing as rales; it can be louder by pressure with stethoscope. During rapid resorption of pleural effusion, pleural rub becomes louder, and more intense. Such sound is so rough that can be felt even during palpation.

Pleural friction sound is best heard at the lung bases due to better respiratory mobility, and rarely at the lung apices (tuberculosis with involvement of the pleura, for example). Duration of the friction rub varies in different diseases. Periodic pleural friction sound is typical to rheumatic pleurisy. It is heard a few hours, temporary disappears, and then appears again. In dry pleurisy of tuberculosis etiology and pleurisy with effusion at resorption stage, pleural rub is heard for a week and over. Longstanding, for years after pleurisy, friction rub can be sometimes heard due to significant roughness of the pleural surfaces.

Characteristics of added sounds are summarized in Tab. 3.10.

Tab. 3.10. ***Differential diagnosis of adventitious sounds.***

Signs	**Dry rales**	**Moist rales**	**Crepitation**	**Pleural friction sound**
Relation to the respiratory phases	Best heard during expiration	Best heard during inspiration	Heard at the end of inspiration	Heard throughout respiratory cycle
Change during cough	Decrease or change character	Decrease or disappears	Without changes	Without changes

Pressure with the stethoscope	Without changes	Without changes	Without changes	Increase
Breathing movement with close nose and mouth	Absent	Absent	Absent	Only this sound is heard

Insrumental and Laboratory Methods

Diagnostic procedures for assessing the patients with suspected or known respiratory system disease include imagine studies, technique for obtaining biological specimens, and method used to characterize the functional changes developing as a result of disease.

Imagine studies

Imagine studies used to examine the patients with disorders of the respiratory system include:

- Roentgenoscopy
- Roentgenography (radiography)
- Fluorography
- Computed tomography
- Magnetic resonance imaging
- Scintigraphic imaging
- Bronchography
- Pulmonary angiography
- Ultrasound examination

Roentgenoscopy is the most common method for assessing relative lungs translucency, and for the diagnostic evaluation of disease involving the pulmonary parenchyma (consolidation of the pulmonary tissue, pneumosclerosis, tumor), the pleura (pleural fluid or air, pleural adhesions), and, to a lesser extent, the airways. Presence of the cavity in the lungs can also be determined roentgenoscopy.

Roentgenography (radiography, x-rays). Routine chest radiography generally includes both posteroanterior and lateral views, and used for film

recording – radiograph. The detail that can be seen on radiograph allows better recognition of parenchymal and airway diseases (indistinct focal consolidations, bronchovascular pattern, etc.).

Fluorography – a variant of radiography, is a convenient method for screening the population. The image in fluorography is made on a role film of a small size.

Computed tomography is cross-sectional scanning of the chest. This technique is more sensitive than plain radiography in detecting respiratory abnormalities. Computed tomography makes possible to distinguish more accurate tumors, small indurations, cavities and caverns in the lungs. This method is far better than radiographic studies at characterizing tissue density, distinguishing subtle differences in density between adjacent structures, and providing accurate size assessment of lesions. The use of computed tomographic scanning of the chest is very useful as a means of gathering quantitative information about specific radiographic findings.

Magnetic resonance imaging provides a less detailed view of the pulmonary parenchyma as well as poor spatial resolution. However, magnetic resonance imaging offers several advantages over computed tomography in certain clinical settings: for imaging abnormalities near the lung apex, the spine, and the thoracoabdominal junction. Vascular structures can be distinguished from nonvascular without the need of contrast.

Scintigraphic imaging. Administered radioactive isotopes allow the lungs to be imaged with a gamma camera. The most common use of such method is ventilation-perfusion lung scanning performed for detection of pulmonary embolism. Radioactive isotopes can be injected intravenously; albumin macroaggregates labeled with technetium 99m is used for this purposes, or inhaled – radiolabeled xenon gas. When injected intravenously, the distribution of the trapped radioisotope follows the distribution of blood flow. When inhaled, radioisotopes can be used to demonstrate the distribution of ventilation.

Bronchography is an integral part of the diagnosis evaluation of diseases of bronchi. The standard technique requires the injection of contrast medium, usually iodolipol, into the bronchi lumen. This may be done through a catheter passed via the nose or mouth through the anaesthetized larynx. Then radiographs are taken, that give a distinct patterns of the bron-

chial tree. This procedure is of particular importance to the evaluation of bronchiectasis, abscesses, caverns in the lungs, and compression of the bronchi by tumor.

Pulmonary angiography. The technique of the pulmonary angiography requires the injection of radiopaque contrast medium into the pulmonary artery through a previously threaded catheter. Radiographs are taken, on which the pulmonary arterial system can be visualized. Pulmonary angiography in pulmonary embolism demonstrates the consequences of an intravascular clot (a defect in the lumen of a vessel, or abrupt termination of the vessels). Suspected pulmonary arteriovenous malformation can be also visualized by this method.

Ultrasound examination generally is not useful for evaluation of parenchyma of the lungs due to physical properties of the ultrasound waves: ultrasound energy is rapidly dissipated in air-containing pulmonary tissue. However, it is helpful in the detection and localization of pleural fluid and therefore is often used as a guide to placement of a needle for sampling of the liquid in thoracentesis.

Techniques for obtaining biologic specimens.

Techniques for obtaining biologic specimens, some of which involve direct visualization of the part of the respiratory system, include

- Collection of the sputum
- Thoracentesis
- Bronchoscopy

Collection of the sputum

Sputum is pathological secretion expectorated from the respiratory tract. Sputum should be collected after thorough mouth and throat rinsing in the morning hours before breakfast. To collect sputum for more than 12 hours is not expedience because long-standing storage leads to rapid flora multiplying and autolysis of the formed elements.

Sputum Analysis

Clinical sputum analysis includes: macroscopic, microscopic, and bacterioscopic studies.

Macroscopic study

In macroscopic study amount, character, color, consistence, and admixture in the sputum are assessed.

Amount of the sputum

Daily amount and amount of separate portions of the sputum depends on the character of the diseases from one side, and from the patient ability to expectorate from other one.

Scarce amount of sputum observes in the patients with inflammation of the respiratory tract: in laryngitis, trachitis, at initial stage of acute bronchitis, bronchial asthma out of attack, and in bronchopneumonia.

Ample amount of sputum (from 0.5 to 2 liters) secrete from the cavity in the lungs, in bronchus (bronchiectasis, pulmonary abscess), or in pulmonary edema due to significant transudate in bronchi.

Significant amount of purulent sputum may forms layers on standing. Two-layers (pus and plasma) sputum is typical to pulmonary abscess, three-layers (pus, plasma, and upward mucus) – to bronchiectasis, pulmonary tuberculosis (in cavern presence).

Character of the sputum

Character of the sputum is determined by its composition: mucus, pus, blood, and serous fluid.

Mucous sputum consists of mucus – product of mucous glands. Such sputum is produced in acute bronchitis, at the peak of bronchial asthma attack.

Mucopurulent sputum is mixture of mucus and pus, moreover mucus is predominant part, and pus in a form of traces or small bundles is observed. Mucopurulent sputum can be obtained in chronic bronchitis, trachitis, bronchopneumonia, and tuberculosis.

Puromucous sputum contains pus and mucus; pus is predominant part of the sample. Such sputum arises in chronic bronchitis, bronchiectasis, pulmonary abscess, etc.

Purulent sputum without mucus admixture appears in opened to the bronchus pulmonary abscess, in rupture of the pleural empyema to the bronchus lumen.

Mucous-bloody sputum consists mainly of mucus with streaks of blood, and can be produced in inflammation of upper respiratory ducts, pneumonia, lung infarction, congestion in the pulmonary circulation, and bronchogenic tumor.

Mucopurulent bloody sputum contains uniform mixed mucus, blood and pus. Such sputum arises in tuberculosis, bronchiectasis, actinomycosis of the lungs, and bronchogenic tumor.

Bloody sputum observes in pulmonary hemorrhage: tuberculosis, wounds of the lungs, actinomycosis, and bronchogenic tumor).

Serous sputum is plasma of the blood that passes to the bronchi in edema of the lungs.

Serous bloodstained foamy sputum is characteristic of pulmonary edema, when not only plasma, but also erythrocytes penetrate from pulmonary alveoli to the bronchi.

Color of the sputum

Color of the sputum depends on its character, and also by inspirited particles. Predominance of one of substrates gives sputum corresponding hue.

Mucous sputum is usually *colorless*, *transparent*, and *glass-like*.

Mucopurulent sputum is *glass-like with yellow tint* as its main component is mucus, on the background of which pus traces is observed.

Puromucous sputum is *yellow-greenish* due to predominance of pus.

Purulent sputum is *greenish-yellow* due to the pus.

Mucous-bloody sputum is *glass-like* (due to predominance of mucus) with pink or rusty tint (due to the presence of changed or unchanged blood pigment – hematin). *Rusty sputum* is characteristic of acute lobar pneumonia, when blood is not expectorated immediately from the respiratory tract and stays there for sometimes. The hemoglobin converts into hemosiderin to give a rusty hue to the sputum.

Mucopurulent bloody sputum is *glass-like* (predominance of mucus), with *yellow traces* (pus), with *red* color *streaks* (fresh blood) or rusty hue (changed blood pigment).

Bloody sputum is of *red* color. Peculiarity of the pulmonary hemorrhage is the presence foamy secretions due to the air bubbles.

Serous sputum is transparent-yellow (color of penetrated blood plasma), and foamy.

Sputum containing foreign admixtures has color of these admixtures: white in millers, black – in miners, blue in inspiration of ultramarine paint, etc.

Consistency of the sputum

Consistency tightly connected with sputum character and may be tenacious, thick, and liquid.

Tenacity of the sputum depends on the presence of mucus and amount of it. For example, in bronchial asthma, acute and chronic bronchitis, bronchopneumonia consistency of the sputum is tenacious.

Thickness of the sputum is caused by the presence of the large amount of the formed elements – leucocytes, various epithelium cells (bronchiectasis, chronic bronchitis, pulmonary abscess, and tuberculosis).

Liquid sputum can be in large it amount, when the plasma is significant composing component (pulmonary hemorrhage, pulmonary edema).

Odor of the sputum

Fresh sputum is usually odorless. Unpleasant smell can appears in protracted conservation of the sputum. Foul odor of freshly expectorated sputum can be caused by it retaining in bronchi and cavities in the lungs due to putrefactive decomposition of proteins. Unpleasant odor sputum can be had in chronic bronchitis with bad bronchi drainage, strong smell – in bronchiectasis, pulmonary abscess, sometimes in tuberculosis, in malignant tumor with necrosis, fetid (putrid) odor is characteristic of tissue decomposition – gangrene.

Admixture

The following elements can be seen in the sputum by an unaided eye:

- Curschmann spirals – has diagnostic significance in bronchial asthma;
- *Fibrin clots* –has significance in fibrinous bronchitis, and rarely in lobar pneumonia;
- *Lentil or rice-like bodies (Koch's lens)* – observe in sputum in cavernous tuberculosis;
- *Purulent plugs (Dittrich's plugs)* – occurring in bronchiectasis, gangrene, chronic abscess, and fetid bronchitis.
- *Diphtherias films*;
- *Necrotic pieces of the lungs* – observes in pulmonary gangrene and abscess;
- *Pieces of the pulmonary tumor*;
- *Actinomycete;*
- *Lime grains* – in decomposition of old tubercular foci;
- *Echinococcus bubbles* – observe in sputum in rupture of echinococcus cyst in the lung and expectoration of plentiful amount of colorless transparent fluid;
- Foreign bodies.

Physical properties of the sputum revealed in macroscopic examination are summarized in Tab. 3.11.

Tab. 3.11. ***Physical properties of the sputum.***

Character	Consis-tency	Color	Odor	Layer ness	Disease
Mucous	Tenacious	Glass-like	Odorless	Absent	Acute bronchitis, at the peak of the bronchial asthma attack
Muco-puru-lent	Tenacious thick	Glass-like with yellow traces	Odorless	Absent	Chronic bronchitis, trachitis, broncho-pneu-monia, tuberculosis
Puro-mu-cous	Thick, tena-cious	Yellow-greenish	Unpleasant	Three layers	Chronic bronchitis, bronchiectasis, pulmonary abscess
Purulent	Thick	Greenish-yellow	Sharp un-pleasant	Two lay-ers	Pulmonary abscess, gangrene of the lungs
Mucous bloody	Viscous, tenacious	Rusty, glass-like, reddish	Odorless or without unpleasant odor	Absent	Inflammation of the upper respiratory ducts, pneumonia, broncho-genic tumor, lung in-farction, congestion in the lesser circulation
Muco-puru-lent bloody	Tenacious thick	Glass-like, reddish with puru-lent traces	Unpleasant, putre-factive	In large amount three layers	Tuberculosis, bronchi-ectasis, bronchogenic tumor
Bloody	Liquid, foamy	Red	Odorless	Absent	Pulmonary hemor-rhage: in tuberculosis, wounds, lung tumor
Serous	Foamy, liq-uid, sticky	Transparent yellowish	Odorless	Absent	Pulmonary edema

Microscopic study

Sputum elements revealed in microscopic study can be divided into three main groups: cellular, fibrous, and crystal formations (Tab. 3.12).

Tab. 3.12. ***Sputum elements in microscopic study.***

Cellular elements	Fibrous elements	Crystal elements
• Epithelium cells (squamous, columnar) • Macrophages • Leucocytes, (eosinophils) • Erythrocytes • Tumor cells	• Curschmann spirals • Elastic fibbers • Fibrin fibers	• Charcot-Leyden crystals • Crystals of hematoidin • Crystals of cholesterol • Crystals of fatty acid

Cellular elements

Squamous epithelium – is epithelium of mucous membrane of mouth cavity, nasopharynx, larynx, and vocal chords. Single cells of squamous epithelium are always observed in sputum, and have no diagnostic significance.

Columnar ciliated epithelium – is epithelium of bronchi and trachea mucous membrane. It is contained in small quantity in any sputum, but its large amount is found in acute bronchitis, in bronchial asthma attack, and in acute infections of upper respiratory tract.

Alveolar macrophages. Insignificant quantities of alveolar macrophages are present in any sputum, large amount – in various inflammatory processes of bronchi and pulmonary tissue: pneumonia, bronchitis, and professional diseases of the lungs. Siderophages arise in the sputum of the patients with congestion in the pulmonary circulation, especially in mitral stenosis; in lung infarction, acute lobar pneumonia.

Leucocytes observe in any sputum; in mucous – single, and in purulent – all microscope vision area. Their large amount is characteristic of inflammatory and especially purulent process. Sometimes among leucocytes eosinophils can be identified. *Eosinophils* are the large leucocytes with uniform large lustrous grains. Eosinophils presence in the sputum suggest bronchial asthma or chronic bronchitis with asthma component.

Erythrocytes Single erythrocytes can be visible at any sputum; in large quantity observed in bloody sputum: pulmonary hemorrhage, lung infarction, congestion in the pulmonary circulation, etc.

Malignant tumor cells. Sputum with such cells is underwent then special cytological study. Tumor cells are found in the sputum especially when tumor degrades or growth endobronchially.

Fibrous elements

Curschmann spirals – are found in the sputum of patients with respiratory pathology accompanied by bronchospasm: bronchial asthma, bronchitis with asthmatic component, bronchial tumor.

Elastic fibers presence in the sputum indicates degradation of the pulmonary tissue: in tuberculosis, pulmonary abscess, and tumor.

Fibrin fibers – are found in fibrinous bronchitis, tuberculosis, actinomycosis, and lobar pneumonia.

Crystal elements

Charcot-Leyden crystals. Presence of Charcot-Leyden crystals in the sputum is characteristic of the bronchial asthma even not in attack, and between attacks period. Less frequently they can be observed in the sputum of patients with eosinophilic bronchitis, lobar pneumonia, and bronchitis.

Hematoidin crystals. These crystals are the product of hemoglobin degradation, and are formed in hemorrhage, and necrosis tissue.

Cholesterol crystals – observed in the sputum of the patients with tuberculosis, tumor, pulmonary abscess, etc.

Fatty acid crystals – are frequently found in purulent sputum (Dittrich's plugs), produced in sputum congestion in the cavity (abscess, bronchiectasis).

Bacterioscopic study

Tuberculosis mycobacteria presence in the sputum indicates tuberculosis.

Pneumococcus, streptococcus, staphylococcus, Pfeiffer's bacillus – all these microorganisms occur in small amount in the sputum of the respiratory ducts of healthy persons and only become pathogenic under the certain unfavorable condition to cause pneumonia, lung abscess, bronchitis.

Microbes, their virulence and drug-resistance can be identifying by bacterioscopic study.

Sputum analysis in selected respiratory pathology is represented in Tab. 3.13.

Tab. 3.13. ***Sputum analysis in selected respiratory pathology.***

Disease	Sputum amount	Sputum character	Macroscopic study	Microscopic study
Acute bronchitis	Scarce, in later stages – large amount	Mucous, mucous-purulent	–	Columnar epithelium, leucocytes- moderate amount, in long-standing course – macrophages
Chronic bronchitis	Various	Mucous-purulent, mucous-purulent bloody	–	Leucocytes – large amount; erythrocytes, macrophages
Bronchi-ectasis	Ample (morning)	Puro-mucous three-layers	Dittrich's plugs	Leucocytes – many; fatty acids, hematoidin, cholesterol crystals
Bronchial asthma	Scare	Mucous	Curschmann spirals	Columnar epithelium, Charcot-Leyden crystals, eosinophils,
Lobar pneumonia	Scare initially, ample – later	Sticky, rusty initially, later mucous-purulent	Fibrin clots, changed blood	Macrophages, leucocytes, erythrocytes, hematoidin crystals, pneumococcus
Pulmonary abscess	Ample in secrete to bronchus	Purulent with foul odor	Necrotic pieces of the lung tissue	Leucocytes, elastic fibers, fatty acid, hematoidin, cholesterol, crystals,
Pulmonary tuberculosis	Various	Mucous-purulent, sometimes with blood	"Koch's lens" in cavern presence	Tuberculosis mycobacteria; elastic fibers, and various crystals
Broncho-pulmonary tumor	Various	Mucous-bloody, mucous-purulent bloody	Tissue pieces in ample sputum in tumor degradation	Atypical cells

Thoracentesis (pleurocentesis) is performed to sampling of pleural fluid for diagnostic purposes; in the case of a large effusion to remove fluid from the pleural cavity; and, whenever necessary, to administer drugs.

Technique. The patient should sit facing the chair back with arms crossed on the chest. The puncture is done in posterior axillary line at the preliminary determined by percussion point of maximum dullness – usually 7th or 8th interspaces at the upper edge of the underlying rib (at the lower edge intercostals vessels are located). Previous the place of the puncture is treated with alcohol iodine and then anesthetized. Sampling is obtained by 10 ml syringe with a thick and long needle. For diagnostic purposes 50–150 ml of fluid is taken, and then puncture site after needle removing is treated with a 5 % iodine solution.

Study of the Pleural Fluid

Diagnostic sampling allows the collection of liquid for macroscopic, chemical, microscopic, and bacteriologic studies.

Macroscopic study

In macroscopic study character, color, consistency, and relative density of the pleural fluid are assessed.

Character. The pleural fluid is divided into two large groups transudates and exudates.

Transudates – are non-inflammatory fluid that occurs in disorders of lymph and blood circulation in the lungs (for example in heart failure).

Exudates – are of inflammatory character, and occur in inflammatory affection of the pleura. Exudates can be:

- *Serous* and *serofibrinous* in exudative pleurisy, rheumatic pleurisy;
- *Seropurulent* and *purulent* in bacterial pleurisy;
- *Hemorrhagic* more frequent in traumatic pleura affection, less frequently in infarction of the lungs, and tuberculosis;
- *Chylous* in congestion of lymph or destruction of the thoracic duct by a tumor or an injury;
- *Chylous-like* in chronic inflammation of serous membrane as a result of cellular degradation with fatty degeneration;
- *Putrefactive* in wounds associated with putrefactive flora.

Transparency of pleural fluid depends on its character. Transudates and serous exudates are transparent and slightly opalescent. Another exudates in most cases are turbid that can be caused by abundance of leucocytes (seropurulent, purulent), erythrocytes (hemorrhagic), fat drops (chylous), and cellular detritus (chylous-like).

Color of the pleural fluid is also depends on its character. Transudates have pale yellow color, serous exudates – from pale to golden yellow, in jaundice – deep yellow. Purulent and putrefactive effusions are of grayish-white or greenish-yellow color; in blood admixture they can be reddish or more frequent – grayish-brown. The color of hemorrhagic exudates varies from pink to dark red or even brown depending on amount of blood in the fluid, and also on the time of its retention in pleural cavity. Chylous exudates resemble thin milk.

Consistency of pleural fluid in transudates and exudates is usually liquid. Only purulent exudates are thick and cream-like. In old encapsulated empyema the pus can be of puree consistency with grains and fibrin flakes.

Odor. The pleural fluid is a rule odorless. Only putrefactive exudates have unpleasant, offensive smell due to decomposition of protein by anaerobic enzymes.

Relative density of the pleural fluid is determined by urometer. Relative density of transudates is less than of exudates. Relative density of transudates varies from 1005 to 1015 g/cm^2; relative density of exudates is usually higher than 1015 g/cm^2 (1018–1022).

Chemical study

Protein level in the pleural fluid is assessed by refractometer. The relative density and protein contents are the main criteria that allow the effusion to be classified as either exudative or transudative. Protein content in transudates is 5–25 g/l (0.5–2.5 %), in exudates – more than 30 g/l (3–8 %). Qualitative protein content is also of great diagnostic significance for differentiation between transudates and exudates.

Correlation of protein fractions of exudates is about the same as of blood serum; albumin-globulin ratio is 0.5:2; the fibrinogen contents is lower than that of blood (0.05–0.1 %) but its quantity is sufficient to clot spontaneously.

In transudates albumin-globulin ratio is 2.5:4; albumin prevail while fibrinogen is absent or almost absent (therefore transudates does not clot).

Rivalta's reaction was proposed for differentiation between transudates and exudates. In a cylinder filled with 100–150 ml of distillated water and 2–3 drops of acetic acid, 1–2 drops of the punctate are added. Exudates drop cause turbidity in a form of white cloud (or like cigarette smoke), which

sinks to the bottom of a cylinder (positive reaction). Transudates drops or do not leave a cloudy trace, or it can be insignificant and quickly disappears (negative reaction).

Lucaerini test. To 2 ml of 3 % hydrogen peroxide solution placed on a watch glass (against a black background) one drop of punctate is added. Exudates drop leaves opalescence turbidity (positive reaction); transudates drop cause no turbidity (negative reaction). In both reactions the cause of turbidity is the presence of seromucin – mucopolysaccharide complex in exudates. In transudates seromucin is absent.

Microscopic study

Microscopy allows study cellular composition of the pleural precipitate obtained by centrifuging. A native preparation before staining is recommended to study.

Native preparation

Study of the native preparation allows assessing quantity of cellular elements, qualitative content of precipitate, presence of suspected atypical cells, etc. In native preparation the following elements can be revealed.

Erythrocytes in small quantity can be present in any pleural fluid because of puncturing of the tissues. In transudates and serous exudates insignificant amount of erythrocytes is detected; in hemorrhagic exudates in patients with tumor, infarction of the lung, injuries, hemorrhagic diathesis they usually covered all vision area.

Leucocytes in a small quantity (to 15 in vision field) are revealed in transudates and in a large amount – in fluid of inflammatory genesis (especially in purulent exudates). Qualitative content of leucocytes are assessed in stained preparations.

Mesothelium cells are recognized by their large size (to 50 mcm). Transudates contain significant amount of mesothelium cells. They also can be determined in exudates in canceromatosis, and sometimes in tuberculosis.

Tumor cells. Exudates sometimes contain cells suspected for tumor according to absence of distinct cellular borders, polymorphism of their size and shape. The nature of tumor cells is difficult to assess in native preparation.

Stained preparation

Cytological picture of the pleural fluid is different and depend on character, etiology and duration of liquid presence. In stained preparation the following cellular elements are differentiated.

Neutrophils are present in exudates of any etiology. In serous exudates of tubercular or rheumatic etiology they are found in significant amount at initial stage of exudates development (approximately during first 10 days), and then their amount gradually decreases – replaced by lymphocytes. Long-standing neutrophilia indicates grave course of disease; appearance of predominant amount of neutrophils is a sign of transition of serous exudates to purulent. In purulent exudates neutrophils are prevalent cells.

Lymphocytes are obligatory elements of any exudates. They are predominant in cytological picture of serous exudates at a peak of clinical manifestation (80–90 % of all leucocytes).

Eosinophils are contained sometimes in serous and hemorrhagic exudates of various etiology: in rheumatic, tubercular, tumor exudates, composing 20–80 % of all cellular elements.

Macrophages resemble morphologically monocytes, but differ from they by the presence in the cytoplasm of phagocytosis products.

Mesothelial cells are always present in transudates, at initial stage and at the period of reparation of exudates, in significant amount in canceromatosis of serous membrane. In long-standing and sometimes in acute pleural affections and also in transudates coarse vacuolized mesothelial cells acquire many properties of blastoma cells that can be lead to mistakes.

Malignant cells. It is very difficult to differentiate between tumor and mesothelial cells. Luminescent microscopy helps in this situation: when stained with rhodamine, acridine orange or some other fluorochromes, tumor cells luminescence differently than normal cells.

Bacterioscopic study

Transudates as a rule are sterile in microbiological studies, but they can be infected during repeated thoracentesis.

Exudates may be sterile, for example in rheumatic pneumonia, tumor of the lung, and lymphosarcoma. Bacterioscopy of serous exudates in tuberculosis rare gives positive results. More effective method for tuberculosis mycobacteria detection is inoculation to guinea pigs. In pleurisy caused by pyogenic flora the bacteria can be detected in Gram-stained smears. Pneumococcus, streptococcus, staphylococcus, enterococcus, Klebsiella organisms, Pfeiffer's bacillus, colibacillus can be found in bacterioscopic study. Microbes are tested for antibiotics sensitivity in order to prescribe a correct treatment.

Characteristics of the pleural fluid obtained in thoracentesis are represented in Tab. 3.14.

Tab. 3.14. ***Pleural fluid characteristics.***

Character	Macroscopic study					Chemical study		Microscopic study
	Transparency	Color	Consistency	Odor	Relative density	Protein level	Rivalta's reaction	
Transudate	Transparent	Light yellow	Liquid		Less than 1015	Less than 30g/l	Negative	Mesothelial cells, small amount of erythrocytes and leucocytes
Exudates:					More than 1015	More than 30g/l	Positive	
Serous	Transparent	Light, golden or deep yellow	Liquid					Small amount of cellular elements, predominance of lymphocytes
Seropurulent	Transition from transparent to turbid	yellow green	Liquid					Amount of neutrophils increases
Purulent	turbid	Gray-white, green-yellow	Thick					Many neutrophils, cellular degradation, detrit, cholesterol crystals, microflora
Hemorrhagic	turbid	Pink, dark red or brown	Liquid					Many erythrocytes in infection neutrophils
Cholesterol	turbid	Yellow with brown tint	Thick					Cholesterol crystals, cellular degradation
Chyleous	turbid	Milk-like	Liquid					Fatty drops, many erythrocytes lymphocyte
Chyleous-like	turbid	Milk-like	Liquid					Larger fatty drops, large amount of fatty-degradated cells
Putrefactive	turbid	Gray white, green yellow	Thick puree-like	Foul				

Endoscopic studies include bronchoscopy and thoracoscopy.

Bronchoscopy is used to direct visualization of the tracheobronchial tree. Bronchoscopy is now performed almost exclusively with flexible fiber-optic bronchoscope. The upper airways mucosa is preliminary anaesthetized by 1–3 % dicaine solution. The bronchoscope is passed through the nose, between the vocal cords, and into the trachea. The ability to flex the scope makes it possible to visualize virtually all airways to the level of subsegmental bronchi. Samples from airway lesions can be taken by biopsy. Using a small brush or biopsy forceps at the end of a long cable inserted through a channel of the bronchoscope, allows recovery of cellular material or tissue for analysis by standard cytological and histopathological methods. Photography can also be made whenever necessary. The bronchoscopist is able to identify endobronchial pathology including erosions and ulcers of the bronchial mucosa, tumors, granulomas, bronchitis, foreign bodies, and sites of bleeding. The bronchoscope may provide the opportunity for treatment as well as diagnosis. For example, an aspirated foreign body may be retrieved with an instrument passed through the scope, and bleeding may be controlled with a ballon catheter similarly introduced. Bronchoscopy is used for extracting polyps, treating bronchiectasis, and centrally located abscesses of the lungs.

Thoracoscopy. Recent advances in video technology have allowed the development of thoracoscopy for examination of the visceral and parietal pleura, and for severance of pleural adhesion bands that may interfere with placing artificial pneumothorax. This procedure, done under general anesthesia, involves the passage of a rigid scope with a distal lens through a trocar inserted into the pleural space through separate small intercostals incisions.

Methods for functional studies

Methods used to characterize the functional changes developing as a result of disease are very important for an integrated examination of the patients. It is unusual for a specific lung function test to diagnose a disease. At best, a series of tests may place a lung disorder into one of several categories and when other features such as history, physical examination, radiology and pathology are added to the equation, a possible diagnosis is considered.

The main uses of lung function testing are to help define more clearly the type, character and degree of respiratory failure, and to measure serially natural progression (or regression with therapy) of functional disorder.

Tests of ventilatory function. Various indices are used to assess lung ventilation. Their size and relationship to each other give clues to underlying functional disorder. How normal a volume is will depend on what we predict it should be for that person's height, weight, sex, and age.

Figure 3. 54 shows how the total lung capacity is broken down into its various volumes.

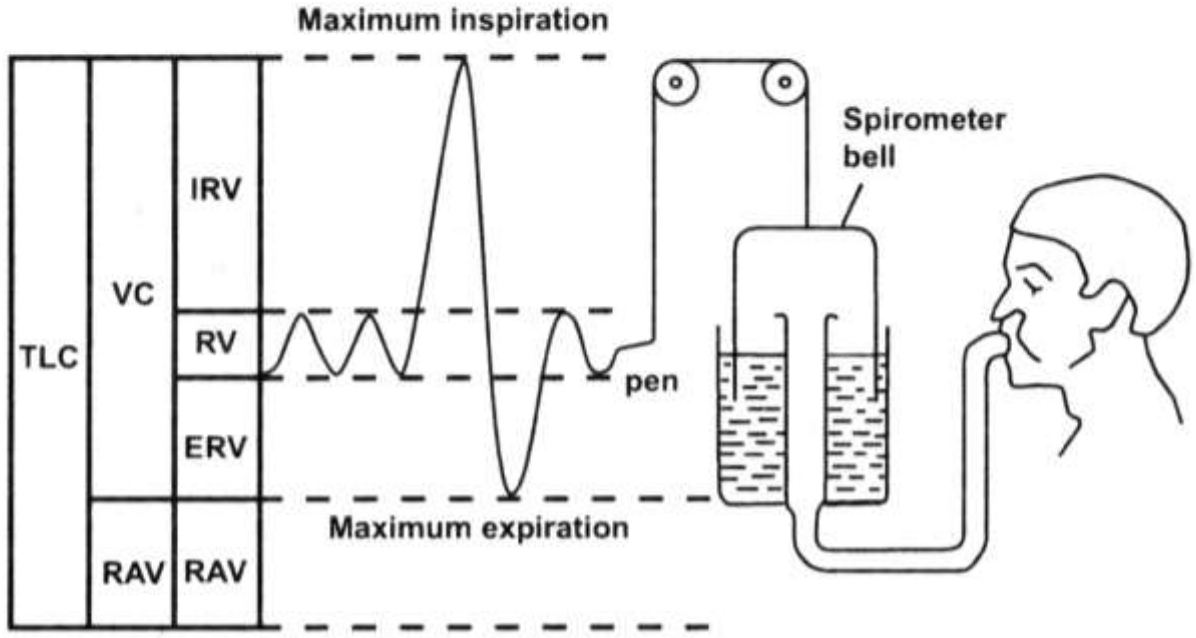


Fig. 3.54. The subdivision of the total lung capacity (TLC) with spirometric recording.
RV – respiratory volume, ERV – expiratory reserve volume, IRV – inspiratory reserve volume, RAV – residual air volume, VC – vital capacity.

The *respiratory volume* (RV) or tidal volume is the total air volume of each normal resting breath (inspiration and expiration). RV varies from 300 to 900 ml; 500 ml on the average. It consists of two parts:

1. Alveolar volume: the volume of gas, which reaches the alveoli – the volume of alveolar ventilation;
2. Dead space volume (about 150 ml): the volume of gas, which passes the lips and is present in the larynx, trachea, and bronchi, but does not take part in gas exchange. However, the air of the dead space is mixed with the inspired air to warm and moisten it, which makes it physiologically important.

The *expiratory reserve volume* (ERV) is the volume of air that can be expired after normal expiration – 1500–2000 ml.

The *inspiratory reserve volume* (IRV) is the volume of air that can be inspired after normal inspiration – 1500–2000 ml.

The *vital capacity* (VC) is the largest volume that can be expired after full inspiration – 3700 ml on average.

The *residual air volume* (RAV) is the volume of air that remains in the lungs after maximum expiration – 1000–1500 ml.

The *total lung capacity* (TLC) can be derived by adding RV, ERV, IRV, and RAV. It is about 5000–6000 ml.

Studies of the respiratory volumes allow assessing ability of the respiratory failure compensation at the expense of reserve inspiratory and expiratory volumes. All these volumes, apart from RV, can be measured by spirometer. Spirography gives more reliable information on respiratory volumes. It can be used to measure additional ventilation characteristics such as minute volume, maximum lung ventilation, respiratory reserve, and volume of lung ventilation.

The *minute volume* (MV) is the volume of gas, which passes the lips in one minute. It can be calculated by multiplying RV by the respiratory rate (frequency, f): MV = f × RV. It is about 5000 ml on the average.

The *maximum lung ventilation* (MLV) is the amount of air that can be handed by the lungs by maximum efforts of the respiratory system. MLV is determined during deepest breathing at the rate of 50 per minute by spirometer; normally – 80–200 l/ml.

The *respiratory reserve* (RR) may be calculated by the formula: RR = = MLV – MV. Normally RR exceeds the MV by at least 15–20 times; RR is 85 % of MLV (in respiratory failure 60% and lower). This value reflects ability of healthy person in considerable load, or of patients with pathology of the respiratory system to compensate significant insufficiency by increasing of minute respiratory volume.

The study of mechanics of the respiratory act allows to evaluate changes in the inspiration and expiration correlation, breath efforts at various respiratory phases, etc.

The *forced expiratory vital capacity* (FEVC) is determined according to Votchal-Tiffeneau during maximum fast, forced expiration. FEVC is 8–11 % (100–300 ml) lower than VC in healthy persons.

The *forced inspiratory vital capacity* is assesses during maximum fast forced inspiration.

Pneumotachymetry, pneumotachygraphy – methods of speed and pressure measuring at various phases of the breathing by pneumotachygraph. Pneumotachygraphy allows to determined volumetric rate of the airflow during inspiration and expiration (normally in rest breathing it is about 300–500 ml/s; in forced – 5000–8000 ml/s), duration of the respiratory cycle phases, MV, alveolar pressure, airways resistance, elasticity or distensibility or stiffness of the lungs and chest, and some other indices.

Tests for respiratory failure.

Determination of *oxygen consumption and oxygen deficit* is carried out by spirography with a closed CO_2 absorption system. Obtained spirogram compared then with spirogram that records with apparatus filled with O_2.

Ergospirography is the method, which allow assessing reserves of the respiratory system. Oxygen consumption and deficit is detected by spirography in the patient at rest and during exercise on ergometer.

Measurement of blood gases

Gas composition of blood samples obtained from warmed up finger is measured on a Van-Slike apparatus. The following is determined:

1. O_2 content in units of volume;
2. oxygen capacity of the blood (the amount of O_2 that can bound by a blood unit);
3. percentage of O_2 saturation of the blood (95 % in norm);
4. partial pressure of O_2 in the blood (90–100 mm Hg in norm);
5. CO_2 content in arterial blood (about 48 % v/v);
6. partial pressure of CO_2 (about 40 mm Hg in norm).

Chapter 4. CARDIOVASCULAR SYSTEM

FUNCTIONAL AND CLINICAL ANATOMY

The **cardiovascular system** consists of the **heart** and **blood vessels**, and contains **blood** that circulates through them.

The function of the heart is to pump sufficient oxygenated blood containing nutrients, metabolites, and hormones to meet moment-to-moment metabolic needs and preserve a constant internal milieu.

Heart, Arteries and Veins

The **heart** is a four-chambered muscular pump that causes blood to move through the vessels to all parts of the body, and then to return to the heart (Fig.4.1). The **chambers** of the heart are the **left atrium** and **right**

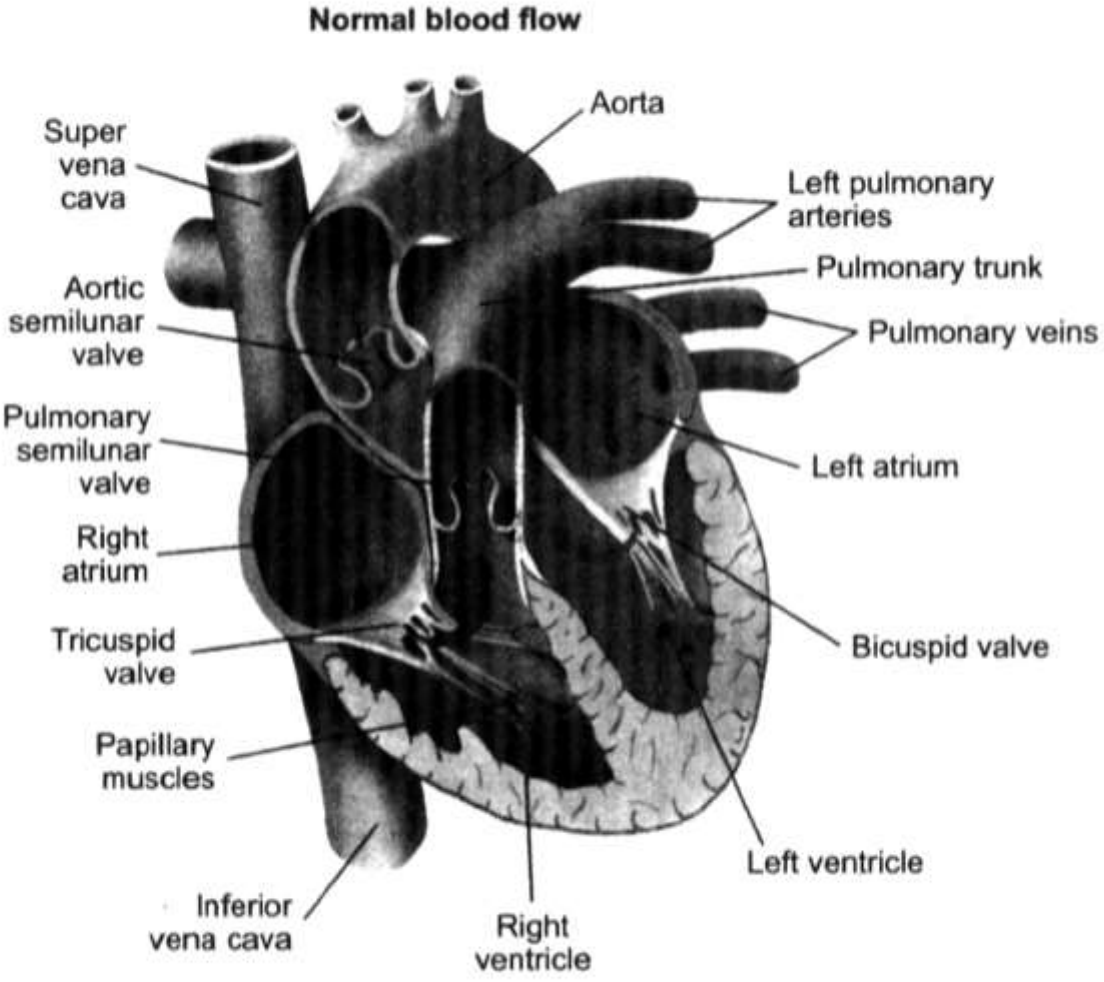


Fig. 4.1. Diagrammatic representation of the heart and great vessels. The arrows indicate the direction of the blood flow.

atrium and the **left ventricle** and **right ventricle**. The left atrium and left ventricle communicate with one another through the **mitral (bicuspid) valve**, and the right atrium and right ventricle through tricuspid valve. (The two atria do not communicate with one another, nor do the two ventricles).

The vessels are called **arteries** or **veins**, according to the direction of blood flow within them: arteries conduct blood *away from* the heart and veins return blood *to* the heart. The vessels that connect the smallest arteries (which called **arterioles**) to the smallest veins (called **venules**) are the blood capillaries. It is only through capillaries that substances can diffuse out of or into the blood; all the other vessels are simply conducting tubes leading to or from the networks of capillaries.

In general, arteries contain blood with a high oxygen content (*oxygenated blood*), and the pumping action of the heart delivers it to all parts of the body. On the other hand, veins return blood to the heart; in most cases this blood has given up a significant proportion of its oxygen and is therefore called *deoxygenated blood* (although it has not, in fact, lost all of its oxygen), and it has also gained a good deal of carbon dioxide.

There is ***one important exception*** to this description of the content of arteries and veins: the *pulmonary arteries* take deoxygenated blood to the lungs so that it can take up oxygen and give off carbon dioxide, while the *pulmonary veins* return the newly oxygenated blood to the heart. Thus, the circuit that carries blood to the lungs and back to the heart – the **pulmonary or lesser circulation**, from the right ventricle to left atrium – is distinct from that which circulates blood to all other parts of the body – the **systemic or greater circulation**, from the left ventricle to the right atrium (Fig. 4.2).

Not only is the content of the arteries and veins different in the two circuits, the circuits themselves are actually separate from one another.

The two atria thus received blood from veins, and the two ventricles pump blood out into arteries. The **superior** and **inferior vena cavae** enter the top and bottom of the *right atrium* respectively, and the four **pulmonary veins** (two on each side) enter the *left atrium*. The **aorta** leaves the top of the *left ventricle*, and **pulmonary trunk** (which divides into the right and left pulmonary arteries) leaves the top of the *right ventricle*. All the above-mentioned vessels are commonly called the 'great vessels' of the heart.

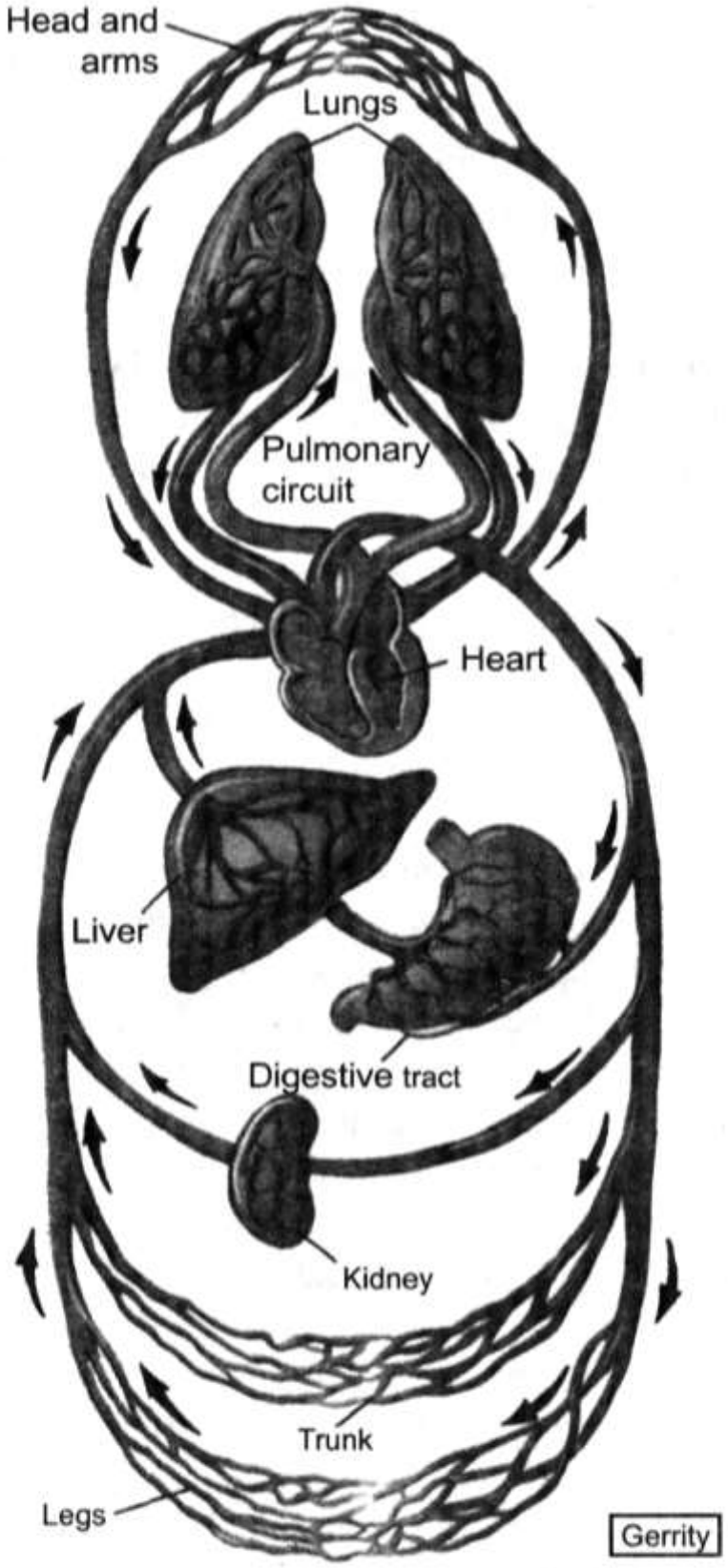


Fig. 4.2. Diagrammatic representation of the cardiovascular system:
1 – pulmonary or lesser circulation, 2 – systemic or greater circulation.

Cardiac Cycle

In the pumping action of the heart, both atria contracts together to propel the blood into the ventricles, and then the ventricles contract to force blood out. The sequence of atrial and ventricular contraction – **the cardiac cycle** – normally takes about 0.8 seconds, giving the heart rate of over 70 beats per minute. The term **systole** (Greek for 'contraction') is the medical word for the contraction of an atrium or ventricle, and **diastole** (Greek 'dilation') means the return of the chamber to its resting size. Atrial systole occupies the first 0.1 seconds of the circle, and accompanied by ventricular diastole; this is followed, over the next 0.3 seconds, by ventricular systole with atrial diastole; in the remaining 0.4 seconds, all the chambers are in a diastolic phase.

On the *left* side of the heart, the mitral valve opens during atrial systole but is closed at all other times of cycle to prevent the oxygenated blood from returning to the left atrium. During ventricular systole, blood passes out of the left ventricle into the aorta through the aortic valve, whose cusps are so arranged that they prevent regurgitation into the ventricle when contraction ceases – the back pressure of the blood in the aorta forces them to close. The rhythmic contractions of the left ventricle cause waves of increased pressure within arteries, and pulsations so caused can be felt in some arteries that are sufficiently near to the skin's surface. The commonest site for 'feeling the pulse' is the radial artery at the front of the wrist.

On the *right* side of the heart, the tricuspid valve opens during atrial systole but is otherwise closed prevent the deoxygenated blood from returning to the right atrium. During ventricular systole, blood passes out into the pulmonary trunk through the pulmonary valve, whose cusps, like those of the aortic valve, are closed by the backpressure of blood as ventricular contraction ceases. The pressure in the pulmonary system is much lower than in systemic system, and, because the pulmonary vessels are deep in the thorax, there is no way to feeling a 'pulmonary pulse'.

Although in theory the words systole and diastole apply respectively to contraction and relaxation of either atria or the ventricles, in practice these terms often refer to activity in the ventricles alone, since ventricular contraction is much more powerful than atrial contraction and rends to overshadow activity in the atria. Thus systole, if used alone, should be taken to mean the phase of ventricular contraction, and diastole to mean ventricular relaxation.

When the heart chambers contract, they do not completely empty. Each ventricle, for example, holds about 120–130 ml of blood when full, and ejects about 70 ml during ventricular systole, leaving about 50–60 ml in the ventricle. The volume ejected by each ventricle (about 70 ml) is the **stroke volume**; with the heart rate of over 70 beats per minute, over 5 liters of blood are pumped out of each ventricle every minute – this is **cardiac output**. Since the right ventricle pumps blood into the pulmonary circuit, and the left into systemic circuit, cardiac output can be thought of as volume of blood flowing through either the pulmonary or systemic circuit every minute. These two volumes may differ from one another slightly at any given moment, but, over time, they should be equal. (Note that cardiac output refers to the volume pumped out be *either* the right *or* the left ventricle, not the total amount pumped by both ventricles.) The cardiac output will vary with changes in stroke volume or heart rate – an increase in either will increase the output, and decrease in either will decrease output.

Control of the Heart Rate

Specialized nerve receptors – **baroreceptors** – in the walls of the internal carotids arteries (the carotid sinuses) and the arch of the aorta constantly monitor the blood pressure. Other similar receptors – called **chemoreceptors** – in the carotid and aortic bodies are mainly concerned with monitoring the amount of oxygen in the blood. Through their nerve supplies by the vagus and glossopharyngeal nerves, information from all of these receptors reaches the reticular formation in the brainstem, so that any necessary changes in heart rate can be made. For example, with exercise when more oxygen is required than at rest, the heart rate increases to increase blood supply and therefore the oxygen supply; after a sudden hemorrhage with low blood pressure, the heart rate increases to compensate for the reduce pressure and keep up an adequate blood supply to the tissues.

METHODS OF EXAMINATION

The establishment of a correct and complete cardiac diagnosis often requires the use of different methods of examination: inquiry, physical examination (general inspection, inspection of the heart region, palpation, and auscultation), noninvasive instrumental examination (electrocardiography, chest roentgenogram, echocardiography, radionuclide and others noninva-

sive imaging techniques), and occasionally specialized invasive examinations, i.e., cardiac catheterization, angiography, and coronary arteriography.

Inquiry

The symptoms caused by heart disease result most commonly from myocardial ischemia, from disturbance of the contraction and/or relaxation of the myocardium, from obstruction to blood flow, or from an abnormal cardiac rhythm or rate.

Tab. 4.1. ***Complaints in the cardiovascular diseases***

Specific	*Nonspecific*
• Pain in the heart region • Intermissions • Palpitation • Dyspnea • Asphyxia • Cough • Hemoptysis • Edema • Syncope	• Fever • Sweatiness • Weight loss • Fatigue • Headache • Dizziness • Sleeplessness • Deranged vision and hearing • Voice changes • Dysphagia • Dyspepsia • Thirst • Pain in the abdomen • Pain in the joints

Specific complaints

Pain in the heart region. Pain in the heart region is one of the most frequent complaint of the patients in the internal diseases clinic. A correct estimation of the patient sensations, when the pain disturbs him – is one of the diagnosis stage. On this stage doctor should form further diagnostic and curative tactics, and in some cases the patient life depends on this tactic. Therefore, at first stage of the diagnostic search is very important to obtain maximum information about the pain. A good history remains the first and often most important approach to diagnosis. Nowhere is this truer than in

diagnosing the cause of chest pain, when the history may be the overriding contribution to establishing the diagnosis. History-taking skills graduate from straight reportage of the patient's story, to what has been called the clinician's 'art' but is in fact attention to detail against a carefully ordered personal database of accumulated experience. History taking is an active process. It involves matching the patient's symptoms to the archetype of a particular cause of chest pain. Obtaining a complete and accurate picture of the patient's symptoms requires the establishment of rapport and, contrary to conventional teaching, judicious direct questioning: specific promptings are generally necessary both to check details and to confirm common understanding of the words used to described the pain. Archetypes are learned from textbook descriptions but they need to be clothed and honed with experience the patient's symptoms should be matched both positively against the archetype of the likely diagnosis, and negatively against the archetypes of differential diagnosis to be excluded. It is often helpful to ask the patient to recall the very first episode of the pain in question: this may be more typical of the archetype.

Diagnostic approach to the patients with pain in the heart region

1. *Location*: retrosternal, in the apex region, to the left of the sternum…
2. *Intensity*: severe, rather intense, moderate, mild…
3. *Character*: a) superficial or profound ("deep");
   b) type of the pain: squeezing, pressing, stabbing, piercing, burning, boring, gnawing, feeling of tightness, shooting;
4. *Frequency*: seldom, every day, every week, several times a day (to indicate how many times);
5. *Duration*: transitory, constant, intermittent, attacks of pain (to indicate in seconds, minutes, hours);
6. *Radiation*: to the left shoulder, left arm, left shoulder-blade, left supraclavicular and subclavicular region, to the back, interscapular region, to the left of the neck, lower jaw, to the epigastric region, to the right half of the chest;
7. *Associated features*: morbid fear of death, palpitation, intermissions, dyspnea, weakness, trembling in the body, cramps, feeling of air deficit, dizziness, excessive urination;
8. *Provocation*: during insignificant physical exertion – during walk: quick, ordinary, slow; ascending the stairs or hill; frosty day; in going out of

doors in 10–20 minutes; emotional factors; excessive meal; after alcohol use, smoking; in considerable physical loading; without visible cause.

9. *Relieving conditions:* is abated by nitroglycerin (how many tablets a day, pain relieve at once, in few seconds, in few minutes); at rest; changing position; physical or emotional exertion; talking; is abated by analgetics.

The main diseases of the heart and great vessels, which can cause pain in the heart region, are represented in Table 4.2.

Tab. 4.2. ***Pain in the heart region. The main causes.***

Diseases of the heart and pericardium	Diseases of the great vessels
Angina pectoris Myocardial infarction Pericarditis Myocarditis Coronaritis Heart valvular diseases Cardiomyopathy Cardiosclerosis	Dissecting aortic aneurysm Tromboembolism of the pulmonary artery and it's branches Inflammatory and degenerative affection of the aorta

A number of key characteristic help to distinguish cardiac pain from other causes (Tab. 4.3).

Tab. 4.3. ***Differential diagnosis of pain in the heart region from history.***

Pain features	Disease
Retrosternal, constricting, feeling of heaviness, from few seconds to 15 min, radiate to the left arm, scapula, jaws, the neck, associated with morbid fear of death, comes on with exertion, is relieved by rest, is relieved by nitrates.	Favors angina pectoris (ischemic pain)
Pain as above but prolonged, continuous pain > 20–30 min, more severe, tight or burning, resist at rest, and does not respond to nitrates.	Favors myocardial infarction

Retrosternal, extremely severe, sharp and tearing, piercing, radiate to the spinal column, moves gradually along coarse of the aorta, associated with collapse, syncope, cyanosis, with very sudden onset.	Consider aortic dissection
Middle of the sternum or heart apex or entire heart region, stabbing, shooting, feeling of heaviness, persist several days or may arise in attack during inspiration, coughing, radiate to the left scapular, the neck, epigastric region, left arm, varies in intensity with movements, the phase of respiration, and under the pressure of stetoscope.	Consider pericarditis
Behind manubrium sterni, permanent, does not respond to exertion	Consider aortitis
Very variable in site and intensity, may vary with posture or movement, very commonly accompanied by local tenderness over the rib or costal cartilage.	Consider musculo-sceletal cause

Chest pain is one of the most common of symptoms and one of the most testing of the clinician's diagnostic skills. The range of its causes, from the trivial, self-limiting behind to the serious and life threatening, independent of severity, emphasizes the importance of establishing the diagnosis. This is turn emphasizes the importance of the history.

Intermissions (escaped beats) are due to disorders of the cardiac rhythm. Patients describe it as a feeling of "disordered activity" of the heart, "stroke of the heart of various strength", "sinking" or "stoppage" of the heart. Intermissions may develop during exertion or at rest, and may intensified in special posture of the patient. Intermissions are clinical sign of atrial fibrillation, extrasystoles and heart blocks.

Atrial fibrillation. In this arrhythmia the atria beat rapidly, chaotically and ineffectively; the ventricles respond at irregular intervals giving the characteristic "irregularly irregular" pulse. The onset of atrial fibrillation can cause palpitation and may precipitate or aggravate cardiac failure in patients with mitral stenosis or poor left ventricular function. Nevertheless,

atrial fibrillation is often asymptomatic, particularly in the elderly. Common causes are listed in the Table 4.4.

Tab. 4.4. ***Common causes of atrial fibrillation***

• Coronary heart disease
• Valvular heart disease (especially mitral valve disease)
• Hypertension
• Thyrotoxicosis
• Sinoatrial disease
• Alcohol
• Cardiomypathy
• Congenital heart disease
• Pulmonary embolism
• Pericardial disease
• Pneumonia

Premature beats (extrasystoles) are frequently found in healthy subjects (emotional exertion, in heavy smokers, in coffee, strong tee, and alcohol abuse), and their prevalence increases with age. Extrasystoles can occur in hyperthyroidism, menopause, digitalis and adrenaline toxicity as well as by reflex in the diseases of the abdominal organs. Premature beats are common in patients with coronary heart disease (especially in acute myocardial infarction), hypertension, rheumatic heart valvular disease, myocarditis, and heart failure. Patients with premature cardiac contraction feel their heart missing a beat (escape beat) with subsequent strong stroke.

Heart block is delay or interrupt of the electrical impulse passage into any part of the conduction system of the heart. Feeling of periodic missing of the heartbeat observes in the patients with sinoatrial block and complete atrioventricular block. Block may develop in inflammatory, dystrophic, and sclerotic affections of the myocardium, atherosclerosis, coronary heart disease, and in drug toxicity (digitalis, quinidine, atropine, salicylates).

Palpitation is subjective feeling of accelerated and intensified heart contractions onto the chest wall. This is a symptom experienced by most people at some time in their lives. Heart palpitation is clinical sign of tachycardia. A

provisional diagnosis can usually be made on the basis of a careful and thorough history (Fig. 4.3). However, in order to make a definitive diagnosis it may necessary to obtain an ECG recording during an attack of typical palpitation. Palpitation is a symptom, mainly, of organic affection of the cardiovascular system. In this case, it has lingering character, more expressive without any external causes, and moreover, accompanied by the pain in the heart region, disorders of the heart rate, feeling of compression in the chest, feeling of fear, stoppage of breathing, headache, noise in the ears, and "net" before eyes.

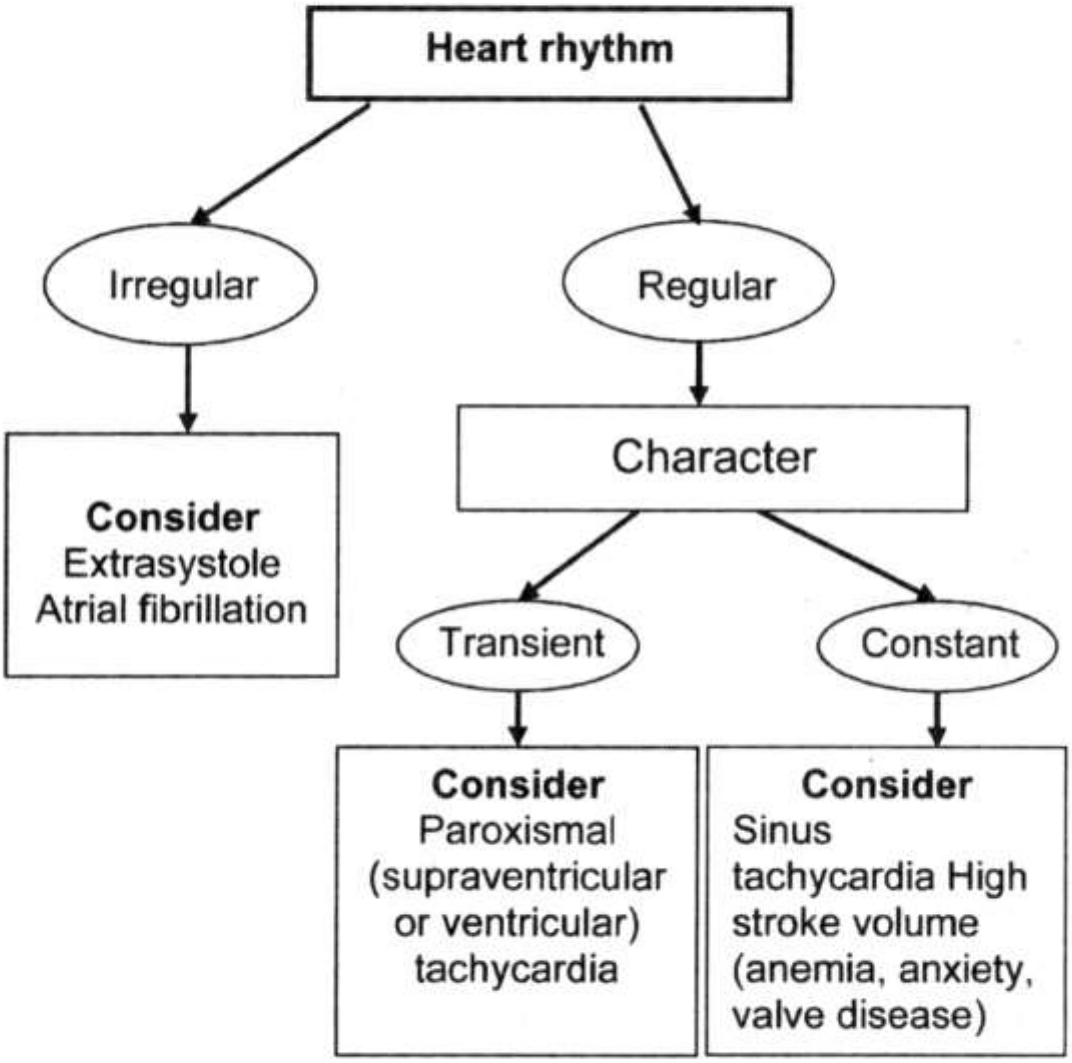


Fig. 4.3. Differential approach to the diagnosis of palpitation.

Accurate assessment of palpitation requires an exact description of the sensation and it is often helpful to ask patients to explain their symptoms by taping out the heartbeat on their chest or a tabletop.

Clinical variants of palpitation

Palpitation periodic and transitory occurs in healthy persons after intensive physical exertion, during running, after emotional stress, strong tea, alcohol, smoking and coffee abuse. It can be provoked by some pharmacological preparations: adrenaline, caffeine, atropine sulphate. Such palpitation is physiologic, and is due to increased chronotropic activity of sympathetic nervous system on the heart and decreased chronotropic influence of vagus nerve.

Attacks of palpitation arise, mainly, as a result of organic affection of the myocardium in coronary heart disease, rheumocarditis, pericarditis, heart valvular disease, cardiac tumor, myocardiopathy, mitral valve prolapse, and ventricular preexitation syndromes (WPW, CLC).

Attacks of palpitation with the heart rate over 160 per minute – paroxysmal tachycardia. This is a sudden acceleration of the cardiac rate (to 180–240 beats per minute). Attack of paroxysmal tachycardia may last from several seconds to a few days, and terminate just as unexpectedly as it begins. During an attack the patient feels strong palpitation, discomfort, feeling of compression in the chest, squeezing pain in the heart region, dyspnea, lack of air, dizziness, and weakness. Paroxysmal tachycardia arises in the patients with organic affection of the heart: in myocardial infarction, heart valvular diseases, and in cardiosclerosis. It may occur in subjects with increased nervous excitability in the absence of pronounced affection of the heart muscle.

Palpitation periodic, of not long duration, appeared regular after moderate physical activity, is a symptom of the heart failure. In increased pressure in the lesser circulation, elevated pressure in the orifice of the vena cava by reflex through the sympathetic nerve accelerates cardiac rate (Bainbrigde reflex) to unload lesser circulation.

Palpitation often develops as a reflex in diseases of some internal organs: in disease of the central nervous system, neurosis, endocrine pathology (thyrotoxicosis), in fever, anemia, hypotension, and in many infectious diseases (Tab. 4.5).

Tab. 4.5. ***Palpitation causes***

Physiological	Pathological	
	Cardiac diseases	Noncardiac causes
Physical exertion Emotional exertion Strong tea, coffee, alcohol Drugs (adrenaline, caffeine, atropine sulfate)	Coronary heart disease Rheumocarditis Pericarditis Heart valvular disease Cardiac tumor Myocardiopathy Mitral valve prolapse Ventricular preexcitation syndromes (Wolf-Parkinson-White Syndrome, Clerk-Levi-Critesko Synrome)	Diseases of the central nervous system, neurosis Endocrine pathology Digestive diseases Fever

Dyspnea (breathlessness). The term "dyspnea" is derived from the Greek roots *dys* (difficult, painful) and *pnoia* (breathing). Breathlessness or dyspnea is disorder of the respiratory ventilation of the lungs, manifested by unreasonably accelerated and intensified breathing. Patients describe dyspnea as 'the sensation of difficult, labored, uncomfortable breathing', as 'distressing feeling of air deficit', and as 'the consciousness of the necessity for increased respiratory effort'. Often dyspnea accompanied by the feeling of the fear and alarm, and by others unpleasant feelings.

Dyspnea is a cardinal symptom of left heart failure and occurs in many others cardiovascular conditions.

Different types of dyspnea can be distinguished in clinical practice, although they often coexist.

1. *Exertional dyspnea:* this may be graded according to the revised New York Heart Association scale (Tab. 4.6). It occurs in the patients with left heart failure (left ventricular failure acute and chronic, mitral valve disease), atrial myxoma, congenital heart disease, pulmonary vascular disease (pulmonary embolism, acute and chronic other causes of pulmonary hypertension), angina equivalent, lung disease, upper airways obstruction, fluid overload, anemia, obesity, and psychogenic exertional dyspnea

may observes. The severity of cardiac disease may be underestimated if the patient's physical activities are restricted for any other reason – sedentary habit, intermittent claudication, or arthritis.

Tab. 4.6. ***Heart failure. The New York Heart Association scale***

Class I	Patient with cardiac disease but without resulting limitation of physical activity; ordinary physical activity does not cause undue dyspnea (or fatigue, palpitation, or anginal pain).
Class II	Patient with cardiac disease resulting in slight limitation of physical activity; they are comfortable at rest; ordinary physical activity results in dyspnea (or fatigue, palpitation, or anginal pain).
Class III	Patient with cardiac disease resulting in marked limitation of physical activity; they are comfortable at rest; less than ordinary physical activity causes dyspnea (or fatigue, palpitation, or anginal pain).
Class IV	Patient with cardiac disease resulting in inability to carry on any physical activity without discomfort; symptoms of dyspnea (or of angina) may be present even at rest; if any physical activity is undertaken, discomfort increased.

2. *Orthopnea*: dyspnea worse when lying flat than when sitting up or standing is common in the patients with left ventricular failure, lung diseases, and diaphragmatic weakness.
3. *Paroxysmal nocturnal dyspnea:* acute dyspnea waking the patient from sleep. Characteristically the patient sits or stands up, and may throw open the windows for air. Paroxysmal nocturnal dyspnea can be crudely graded by the number of pillows that patient uses to prop himself up to allow uninterrupted sleep. Is found in left ventricular failure, nocturnal asthma, and gastro-esophageal reflux with aspiration.
4. *Acute dyspnea at rest:* this is uncommon. It may complicate myocardial infarction, severe arrhythmias (supraventricular or ventricular tachycardia) or number of other catastrophic events: acute dissection of the aortic root, mitral chordal or papillary muscle rupture, large pulmonary embo-

lism, mitral obstruction by left atrial ball thrombus or left atrial myxoma, obstruction or dehiscence of an artificial valve, infundibular spasm of Fallot's tetralogy, asthma, pneumothorax, aspiration/inhaled foreign body, metabolic acidosis, and massive hemorrhage.

Other respiratory symptoms may occur with dyspnea in cardiovascular disease:

1. *Acute pulmonary edema*: acute severe dyspnea accompanied by cough producing copious white or pale pink frothy sputum. There is usually cyanosis, sweating, tachycardia, and raised systemic blood pressure. Dyspnea with copious pale pink frothy sputum also occurs in the rare condition of alveolar cell carcinoma of the lung.
2. *Dry cough*: a persistent dry cough may occur in chronic left heart failure, particularly after exercise and when lying flat in bed at night. A dry cough may persist for about half an hour after an episode of paroxysmal nocturnal dyspnea. Treatment with angiotensin-converting enzyme inhibitors sometimes causes troublesome cough.
3. *Hemoptysis:* coughing large amounts of blood is a dramatic symptom and has many causes. Small hemoptysis occur in severe mitral stenosis and occasionally in severe left ventricular failure. Massive or exsanguinating hemoptysis may occur with rupture of a thoracic aortic aneurysm, pulmonary artery aneurysm, or arteriovenous malformation.
4. *Irregular respiration:* Cheyne-Stokes periodic respiration is well known to occur in advanced cardiac failure, but is uncommon. Cyclical variation in ventilation without frank apnoeic phases is relatively common during sleep in moderate and severe heart failure.

Cardiac dyspnea is caused by upset gas exchange and accumulation of underoxidized metabolites in the blood, which stimulate the respiratory center to accelerate and deepen respiration.

Especially pronounced disorders in gas exchange arise in blood congestion in the lesser circulation, when the respiratory surface and respiratory excursion of the lungs decrease.

Asphyxia is attack of grave dyspnea that occurs due to acute congestion in the lungs and upset of gas exchange in acute left ventricular failure, and observes in the patients with myocardial infarction, aortic stenosis and regurgitation, and in essential hypertension.

Attacks of asphyxia, which are known as cardiac asthma, arise suddenly at rest or soon after physical or emotional stress, and usually during night sleep. This can be explained by an increased vagus tonus during sleep, which causes narrowing of the coronary arteries and thus impairs nutrition of the myocardium. During an attack of cardiac asthma in patients appears feeling of intense pressure in the chest, acute lack of air; the patient suffocates, catches the air by the mouth, marked weakness develops, and appears cold sweat. The skin becomes pallid and cyanotic. The face of the patient, not infrequently, expresses the fear and suffering. Respiration becomes superficial and accelerated, inspiratory dyspnea develops. The patient become coughing and expectorated tenacious sputum. During an attack of cardiac asthma the patient has to assume forced position – orthopnea, or stands up.

If congestion in the lesser circulation progresses, edema of the lungs develops. The feeling of suffocation and cough intensify still more, respiration becomes stertorous, ample foaming sputum with traces of blood (pink or red) is expectorated. Edema of the lungs requires prompt and energetic measure to be taken to prevent possible death of the patient.

Cough in the patients with cardiovascular diseases is due to congestion in the lesser circulation. Cough, as a rule, at first dry, arises during exertion, and particularly in the lying posture of the patient. In prolonged congestion cough is with sputum.

Symptomatic features of cardiac cough are represented in Tab. 4.7.

Hemoptysis. Coughing up blood is an alarming symptom and nearly always brings the patient to the doctor. Hemoptysis in cardiac pathology is mostly due to congestion in the lesser circulation and rupture of fine bronchial vessels during coughing (Tab. 4.8).

Tab. 4.7. ***Symptomatic features in the differential diagnosis of cardiac cough.***

Cough features	Causes of cough	Disease
Periodic, dry, persistent, sonorous, comes on with exertion, at rest, in the lying position, at night	Congestion in the lesser circulation, increases of bronchial secretion	Chronic heart failure
Periodic with insignificant bloody sputum, comes on with exertion, in lying position	Significant hypertension in the lesser circulation	Mitral stenosis
In attacks, dry, mainly at night, comes on directly before the beginning of the night sleep or in 1–2 hours of staying in the bed. In the morning the cough resumes, but slightly of lesser intensity, after expectorating of the mucus sputum the condition of the patient relieves	Aggravation of the septic process and spreading of infection to the upper respiratory tract	Long-standing septic endocarditis
Dry, transitory, sharp rending, accompanied by sensation of pain in the heart	Irritation of the pleural coughing zone	Pericarditis
Strong, sonorous, dry, barking, and dull.	Pressure of enlarged great vessels on bronchi and trachea	Aortic or pulmonary aneurysm

Tab. 4.8. ***Symptomatic features in the differential diagnosis of hemoptysis in cardiovascular pathology.***

Hemoptysis features	Causes of hemoptysis	Disease
In a form of streaks of the blood in the sputum	Congestion in the lesser circulation, rupture of fine bronchial vessels during coughing	Mitral stenosis
In a form of ample foaming sputum with traces of blood (pink or red)	Sudden significant pressure elevation in the lesser circulation, erythrocytes diapedesis through the vessels walls into respiratory tract	Acute left ventricular failure (pulmonary edema)
Traces of blood in a form of streaks or clots	Pulmonary hypertension, erythrocytes diapedesis through the vessels walls into respiratory tract	Pulmonary thromboembolism Pulmonary infarction
Traces of blood or bleeding	Break of the aortic aneurysm to the bronchi, trachea, lungs	Aortic aneurysm dissection
In a form of streaks of blood in insignificant mucus sputum	Disorders of vessels penetrability, erythrocytes diapedesis	In elderly patients with atherosclerosis of pulmonary and bronchial arteries

Edema. Patients with cardiac pathology quite often complain of edema, which initially arises in the evening on the foot, has ascending character and resolves during night. Accumulation of fluid in the abdominal cavity (ascitis) causes sensation of heaviness in the abdomen and its enlargement. The patient can feel heaviness in the right hypochondrium due to congestion in the systemic circulation and enlargement of the liver.

Syncope – is sudden loss of consciousness. Cardiac syncope is caused by a sudden drop in cardiac output and recoverable loss of adequate blood

supply to the brain (cerebral ischemia) due to an arrhythmic or a mechanical problem (Tab.4.9)

Tab. 4.9. ***Common causes of cardiac syncope.***

Arrhythmia	Bradycardia (especially sick sinus syndrome, complete atrio-ventricular block) Tachycardia (especially ventricular tachycardia) Ventricular preexitation syndromes (WPW, CLC) Atrial fibrillation and flutter
Mechanical	Ischemic left ventricular dysfunction Aortic stenosis Mitral valve prolapse Hypertrophic obstructive cardiomyopathy

A faint is often preceded by a brief feeling of "lightheadness"; vision then darkens and there may be ringing in the ears.

Vasovagal syncope may be provoked by some emotionally charged event (e.g. venopuncture) and almost always occurs from the standing position.

Cardiac syncope may be provoked by exertion (e.g. with severe aortic stenosis) or occur completely "out of the blue" (as in heart block). The loss of consciousness is brief, and the patient recovers quickly as long as he or she has assumed the horizontal position.

In vasovagal syncope the loss of consciousness is gradual and rarely associated with injury. There is no amnesia for events that occur after regaining awareness. During a syncopal attack incontinence of urine can occur and there may be some stiffening of the limbs and even some brief twitching of the limbs, but tongue-biting never occurs.

Whenever possible, an accurate description of syncope should be obtained from the patient and a witness. A careful history will often reveal the cause of syncope without recourse to complex and expensive investigations (Tab. 4.10).

Tab. 4.10. ***Typical features of cardiac and neurogenic syncope.***

Features	Cardiac syncope	Neurogenic syncope
Premonitory symptoms	Lightheadness Palpitation Chest pain Breathlessness	Headache Confusion Hyperexcitability Olfactory hallucinations "Aura"
Unconscious period	Extreme "death-like" pallor	Prolonged (> 1 min) unconsciousness Motor seizure activity* Tongue-biting Urinary incontinence
Recovery	Rapid recovery (<1min) Flushing	Prolonged confusion (> 5 min) Headache Focal neurological signs

* NB. Cardiac syncope can also cause convulsions by inducing cerebral anoxia.

Nonspecific complaints

Fever, sweatiness – are the result of infectious-allergic diseases: bacterial endocarditis, rheumatism.

Weight loss. Weight loss may occur as a consequence of heart disease. Long-standing severe congestive heart failure is commonly accompanied by a loss of total body fat and lean body mass, principally skeletal muscle, which in its most severe form is described by term *cardiac cachexia.* This generally implies a fall in total body mass of 20 per cent below predicted. The term is not generally used to include those conditions in which cachexia is due to infection involving the heart such as endocarditis. Cachexia *per se* may lead to cardiac atrophy with further deterioration in cardiac function. Patients with cardiac cachexia generally complain of effort intolerance, muscular weakness, and fatigue.

The mechanism of the muscle wasting is a reduction of protein synthesis, coupled with an increase in the rate of breakdown protein in skeletal muscle. Various factors have been postulated to explain the mechanism of cardiac cachexia (Fig. 4.4).

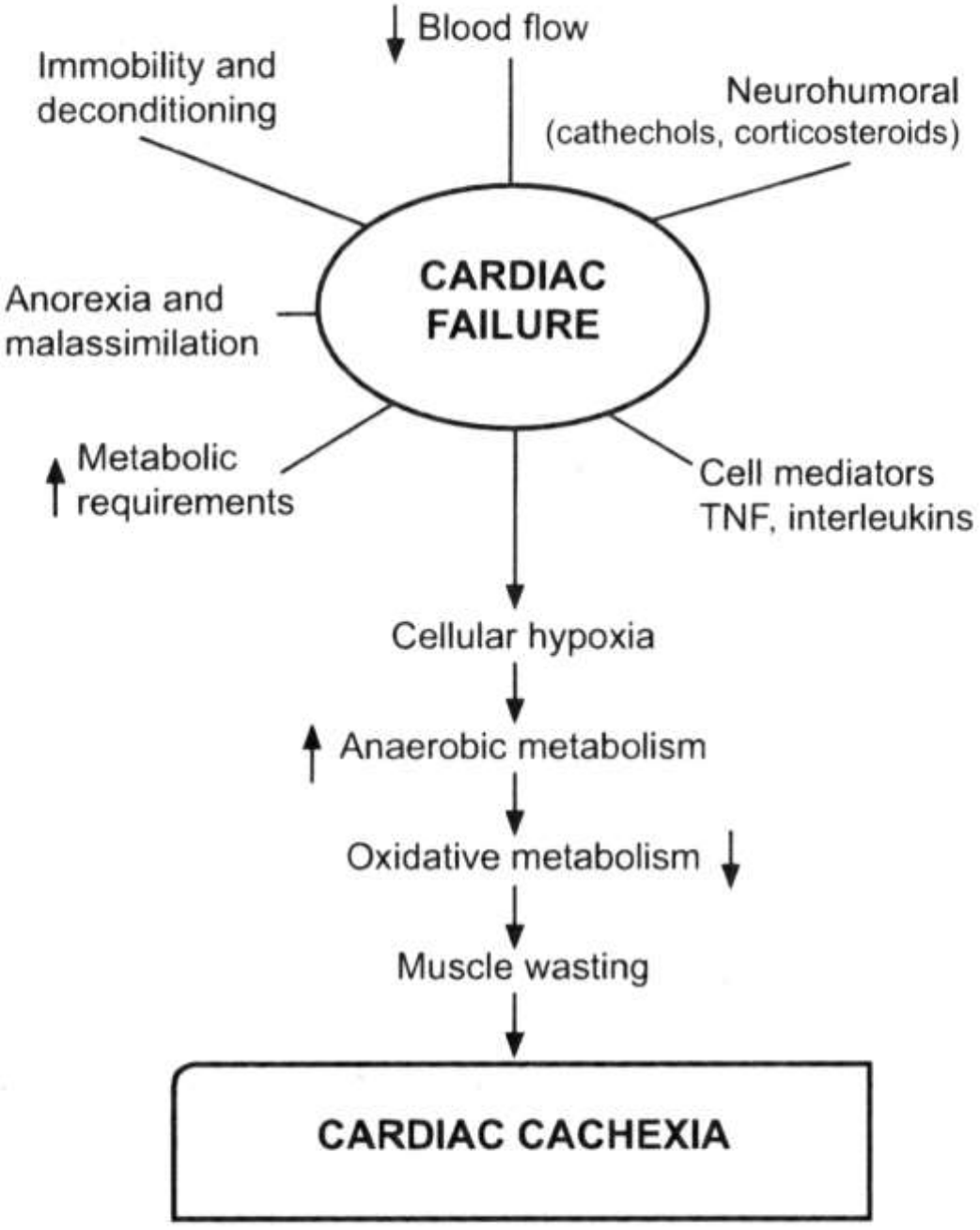


Fig. 4.4. Pathogenesis of cardiac cachexia.

Reduced nutrient limb blood flow exists in severe cardiac failure as a result of diminished cardiac output, neurohumoral compensatory mechanisms,

and increased vascular stiffness, and this may supply tissues inadequately with the substrates necessary for normal protein turnover and growth.

Relative immobility has been shown to produce wasting and reduced protein synthesis in skeletal muscle.

Anorexia is commonly present in patients with cardiac cachexia. When central venous pressure is chronically elevated the resultant increased back-pressure on splanchnic vessels and lymphatics may result in malabsorption of nutrients. Hepatic congestion, hypoxia, and drug toxicity may all result in delayed emptying and hypomotility of the gut, and these factors can also contribute to malassimilation of nutrients and, consequently, an inadequate energy supply to maintain tissue growth. If anorexia is severe, skeletal muscle may be utilized as an energy source. Protein losing enteropathy and steatorrhoea may also occur.

Sympathic nervous system activity is increased in heart failure, and neurohumoral compensatory mechanisms results in elevated catecholamine and corticosteroid secretion. Both have been shown experimentally to result in increased breakdown of skeletal muscle and are associated with type IIb fibre atrophy.

Cellular hypoxia is likely to play a central role in the pathogenesis of cardiac cachexia.

Fatigue. The sensation of fatigue is a non-specific symptom of many cardiac and non-cardiac diseases. It limits the exercise capacity of patients and impairs the quality of life. An explicit definition of fatigue is elusive: it may be defined as 'weariness of mind and body following exertion, associated with a desire for rest and a disinclination or inability to make further effort'. Fatigue can be normal after heavy exertion – pathological fatigue is the same sensation occurring at lower workloads or at rest. Fatigue ranges from slight lassitude to exhaustion.

Fatigue is a cardinal symptom of heart failure but is very non-specific. In obtaining the history two clinical types of fatigue should be distinguished:

1. *Local fatigue*: tiredness of particular muscle groups during exercise, often with local muscular discomfort. The muscles may be described as feeling heavy or stiff. Local fatigue of the leg muscles, and in particular of the quadriceps, is a common symptom limiting cycle exercise in normal subjects.
2. *General fatigue*: tiredness of all parts of the body during exercise. General fatigue is common symptom limiting treadmill exercise in normal subjects.

Almost any type of cardiac disease may cause fatigue, and as in normal subjects the distinction between local and general fatigue often depends upon whether the exercise involves predominantly restricted muscle groups (as in cycling) or all the limb and trunk muscles (as in treadmill exercise). However, the differential diagnosis of fatigue differs for the local and generalized forms (Tab. 4.11).

The exercise causing fatigue should be related to the patient's age and previous level of physical fitness. Fatigue includes lassitude – a sensation of weariness at rest – but should be distinguished from malaise, which denotes an indefinite sensation of discomfort and uneasiness.

Fatigue is often regarded as a symptom of heart failure, but also common in patients with a diagnosis of any serious cardiac disease in whom cardiac function is objectively unimpaired. In such cases there is probably a large psychological component in the symptom. The pathophysiology of fatigue is complex and not well understood.

Tab. 4.11. ***Causes of fatigue.***

Clinical types	Cardiovascular causes	Other causes
Local fatigue	Heart failure – all cases	Localized neuromuscular disease – poliomyelitis, myophathy Local vascular diseases
General fatigue	Heart failure – all cases Some cardiovascular drugs β–Blockers α-Methyldopa	Lung diseases Psychogenic – depression, myalgic encephalomyelitis Obesity Anemia Chronic renal diseases Chronic liver diseases Generalized neuromuscular diseases – following stroke, myopathis Hypokaliemia, hyponatriemia

Headache, dizziness, insomnia, deranged vision and hearing arise in arterial hypertension, heart valvular diseases: aortic stenosis, aortic regurgitation, and heart failure.

Voice changes observe in the patients with aortic aneurysm dissection, heart valvular diseases, which are accompanied by left atrium and ventricular hypertrophy. In this diseases patients complain on hoarseness, husky voice or even complete absence of voice as a result of compression or paralysis of recurrent nerve.

Dysphagia is caused by pressure of enlarged left atrium on esophagus, enlarged left ventricle in aortic valvular diseases and aneurysm.

Thirst observes in the patients with circulatory failure.

Dyspepsia – poor appetite, anorexia, vomiting, meteorism in the patients with cardiovascular pathology are caused by right ventricular failure, disordered circulation in the organs of the abdominal cavity.

Pain in the abdomen arises in different pathological conditions of cardiovascular system:

- Abdominal form of myocardial infarction
- Acute and chronic right ventricular failure
- Aneurysm of abdominal aorta
- Atherosclerosis of mesenterial arteries
- Thromboembolism of mesenterial arteries

Diuresis changes. Decreasing of diurnal diuresis (*oliguria*) and predominance of night diuresis (*nycturia*) are the features of the heart failure development.

Pain in the joints – symptom of rheumatic polyarthritis, typical in acute phase of rheumatism.

General Inspection

General condition depends on severity of the disease. Condition is satisfactory in the patients with cardiovascular pathology in compensation stage. Condition becomes worse in progression of pathological process and associated with complications.

Posture of the cardiac patients may be active, passive or forced. Active posture is in patients with heart valvular diseases, arterial hyperten-

sion, and coronary heart disease without signs of the heart failure. Passive posture – horizontal with low head of the bed is observed in the patients with acute vascular failure. In some cardiac diseases patients assume forced posture (Tab. 4.12).

Tab. 4.12. ***Forced posture of the patients in cardiovascular diseases.***

Posture	Pathological condition	Pathophysiological mechanisms
Upright	Attack of angina pectoris	Tissue oxygen demand reduce at rest, decreased myocardial ischemia
On the right side with high head of the bed	Chronic heart failure of II degree	Re-distribution of blood into the low extremities, reducing of circulating blood volume, decreasing of venous pressure in the lesser circulation, improvement of gas exchange in the "alveoli-pulmonary capillaries" system, displacement of ascitis fluid
Orthopnea	Acute left ventricular failure, chronic heart failure of II–III degree	
Sitting posture bending forward	Dry pericarditis	Pericardial layers presses to one another, reduce their movement that decrease irritation of pain receptors in pericardium
Knee-elbow posture	Effusive pericarditis	Improvement of diastolic cardiac function

Consciousness of the patients with various cardiovascular diseases is clear. Significant hypoxia, as a result of acute and chronic heart failure, is accompanied by consciousness disorders in a form of *stupor* or *sopor*.

Skin and visible mucosa color changes are of important diagnostic significance (Tab. 4.13).

Tab. 4.13. ***Changes of skin and visible mucosa color in cardiovascular pathology.***

Color	Pathological conditions	Mechanisms
Cyanosis	Mitral valvular diseases Acute and chronic left ventricular failure	Secondary pulmonary hypertension
	Thromboembolism of the pulmonary artery	Restricted pulmonary circulation
	Congenital heart diseases	Artery-venous blood shunting
	Aerz's syndrom	Primary pulmonary hypertension
Constant pallor	Aortic stenosis	Low stroke volume
	Aortic regurgitation	Low diastolic pressure
Transitory pallor	Acute vascular failure	Low stroke volume, peripheral vascular spasms, re-distribution of blood
Growing pallor	Aortic aneurysm dissection	Bleeding, shock
Pallor with yellowish tint (coffee with milk)	Infectious endocarditis	Anemia increase hemolysis of erythrocytes
Jaundice	Chronic right ventricular failure Infectious endocarditis	Cardiac liver cirrhosis, infectious-toxic hepatitis
Jaundice with acrocyanosis	Total heart failure	Cardiac liver cirrhosis, slow peripheral circulation

Inspection of the face and the neck.

'Facies mitrale' is characterized by cyanotic blush on the cheeks, cyanotic lips, tip of the nose, ears, young-looking, observes in the patients with mitral stenosis.

Face of the patient with aortic regurgitation is pale, rhythmic movements of the head, synchronous with carotid arteries pulsation – Musset's symptom is observed.

'Corvisart's face' observes in patients with severe heart failure. The face is edematous, pale yellowish with cyanotic tint, the eyes are dull and eyelids are sticky, always open mouth, cyanotic lips.

Excitement, fear of death, suffering expression of the face is typical to the patients with acute left ventricular failure.

In myocardial infarction complicated by cardiogenic shock the face of the patient is pale with cyanotic hue, covered by cold sweat.

'Stokes' collar' – marked dilation of neck veins, edema of the neck, head, shoulders. These signs arise as a result of compression of superior vena cava by aortic aneurysm, tumor of mediastinum, and enlarged mediastinal lymph nodes.

Cardiac edema. Right heart failure produces a high jugular venous pressure, with hepatic congestion and dependent peripheral edema. Edema is caused by penetration of fluid through the capillary walls and its accumulation in tissues. Cardiac edema can first be latent. Retention of fluid in the body does not immediately cause visible edema but provokes a rapid gain in the patient's weight and his decreased urination. Edema becomes visible in the first instance in the malleolus region, on the dorsal side of the foot, shins (if the patient sits or stands), and in sacral region (if the patient keeps bed). Edema first develops only in the evening and resolves during the night sleep. If the heart failure progresses, edema increases, and transudate may accumulate in the body's cavities: in the abdominal cavity (*ascitis*), pleural cavity (*hydrothorax*), and in the pericardium (*hydropericardium*). General distribution of edema throughout the entire body is called *anasarca*.

There are following methods of edema revelation: inspection, palpation, patient weighing, and diuresis control (Tab. 4.14).

For revelation of cavities edema percussion, auscultation, X-ray and ultrasound examination methods are also used.

Tab. 4.14. ***Methods of edema revelation.***

Method	Features
Inspection	Swollen glossy skin. The specific relief features of the edema-affected parts of the body disappear due to the leveling of all irregularities on the body surface. Stretched and tense skin appears transparent, and is especially transparent on loose subcutaneous tissues (the eyelids, the scrotum, etc.)
Palpation	When the pressed by the finger, the edematous skin overlying bones (external surface of the leg, malleolus, loin, etc) remains depressed for 1–2 minutes after the pressure is released
Weighing of the patient	Gain of the body mass
Diuresis control	The amount of intake fluid exceeds the amount of urine

It should be remembered that general edema could be caused not only by cardiac pathology, but also by renal diseases (Tab. 4.15), hypofunction of the thyroid gland, and by long-standing starvation. Considerable edema of low limbs, accompanied by cyanosis of the low part of the body, dilated venous network in the navel region, ascitis that are caused by obstruction to blood flow in vena cava inferior trunk are classified as vena cava inferior syndrome.

Tab. 4.15. ***Symptomatic features in the differential diagnosis of cardiac and renal edema.***

Features	**Cardiac edema**	Renal edema
Location, character	Ascending character, starts from low extremities and spread upward	Descending character, starts from the face and spread downward
Time of arising	More pronounced in the evening	More pronounced in the morning
Color of the skin	Cyanotic	Pallor
Temperature of the skin	Cold	Warm

Skeletal and muscular system. Marfan's syndrome is characterized by affection of the aorta in a form of aneurysm, coarctation, regurgitation and others congenital heart valvular diseases. Phenotype of the patients – tall, long narrow limbs, arachnodactyly, kyphoscoliosis, deformation of the sternum, and hypermobility of the joints.

Drum-stick (Hippocratic) fingers – clubbing of the terminal phalanges of the fingers and toes, nails in a form of 'hour glass' – are characteristic of congenital heart valvular diseases, subacute septic endocarditis, and chronic cor pulmonale.

In aortic coarctation disproportion of the muscular system of upper and low limbs are observed: muscles of upper limbs are hypertrophied, and on the other hand, muscles of low limbs are relatively hypotrophied.

Inspection of the Heart Region

Examination plan:

1. Presence of deformation of the chest in the heart region
   a) cardiac "humpback",
   b) effusive pericarditis;
2. Presence of the apex beat;
3. Presence of the pathological pulsation in the heart region;
4. Presence of the remoted pathological pulsation.

Tab. 4.16. ***Inspection of the heart region.***

Symptoms description	Arising conditions	States
1	2	3
Cardiac "humpback" – constant, diffuse bulging of the area over the heart	Enlargement of the heart chambers in childhood, when the chest is liable to changes	1. Congenital heart valvular diseases. 2. Heart valvular diseases acquired in childhood
Temporary, diffuse and general protrusion of the cardiac region and leveling of the costal interspaces	Effusion in the pericardium cavity	Effusive pericarditis
Apex beat – limited rhythmic pulsation in the site of projection of the heart apex, synchronous to the left ventricle contraction	The thrust of the heart apex against chest wall	Observed in healthy persons with moderately developed subcutaneous fat and wide intercostals spaces

1	2	3
Negative apex beat – precordial depression during systole.	Adhesion of the parietal and visceral layers of the pericardium.	Adhesive pericarditis, mediastinopericarditis
Cardiac beat – spread pulsation in the III-IV interspaces along left edge of the sternum with synchronous pulsation in the epigastric region	Dilation and hypertrophy of the right ventricle	Mitral valvular diseases, tricuspid regurgitation, chronic cor pulmonale
Systolic depression and diastolic bulging of the chest in the III-IV interspaces along left edge of the sternum	Decreased volume of the right ventricle during systole and considerable enlargement of it during diastole	Tricuspid regurgitation
Weak restricted pulsation in the III-IV interspaces somewhat laterally from the left sternal edge	Presence of the bulging in the ventricular wall after myocardial infarction	Aneurysm of the left ventricular anterior wall
Pulsated bulging in the jugular fossae	Dilation of the aortic arch	Aneurysm of the aortic arch
Pulsation in the II interspace to the right of the sternum edge	Dilation of the ascending part of the aorta	Aneurysm of the ascending part of the aorta, aortic regurgitation, syphilitic mesoartritis
Pulsation in the II interspace to the left of the sternum edge	Pulmonary hypertension, poststenotic dilation of the pulmonary artery	Mitral stenosis
Epigastric pulsation, which increased in deep inspiration	Hypertrophy and dilation of the right ventricle	Mitral valvular diseases, tricuspid regurgitation, chronic cor pulmonale
Epigastric pulsation, which decreased in deep inspiration	Pulsation of the abdominal aorta	In healthy persons with underdeveloped subcutaneous fat, enteroptosis, aneurysm of the abdominal aorta

Palpation of the Heart Region

Examination plan:

1. Estimation of location and properties of the apex beat;

2. Determination of the "cat's purr" symptom presence;
3. Palpation of pulsated liver.

Palpation of the apex beat

Technique. Place the palm of your right hand on the chest about at the level of and parallel to the 3rd – 6th ribs. Flex the terminal phalanges of three fingers and slide them medially along the interspaces until the moderately pressing fingers feel the movement of the heart apex. If the apex beat is diffuse, extreme left and lower point is considered to be the point of the apex beat.

Location. A normal apex beat is found in the 5th intercostal space 1–1,5 centimeters toward to the sternum from the left midclavicular line (Fig. 4.5a). If the patient slightly leans forward or during deep expiration you can better detect apex beat, because in these positions the heart presses closer to the chest wall. When the patient lies on his left side, the beat is displaced 3–4 cm to the left, and on right side – 1–1.5 cm to the right. In about one third of cases the apex beat is impalpable: covered by rib.

Displacement of the apex beat may depend on noncardiac and cardiac causes (Tab. 4.17).

Tab. 4.17. ***Causes of the apex beat displacement.***

Physiological	Pathological	
	Noncardiac	Cardiac
Respiration phases Position on the left, right side, lying, standing position Constitutional types	Changes of pressure in the chest and diaphragm level Changes of pressure in the pleural cavities Tumor of the lungs and mediastinum	Changes of the heart chambers size

In changes of the heart chambers size there may be different variants of the apex beat displacement (Fig. 4.5). In left ventricular hypertrophy apex beat is displaced outward (Fig. 4.5b), in dilation of left ventricular cavity apex beat is displaced downward (Fig. 4.5c), in combination of hypertrophy and dilation apex beat is displaced outward and downward (Fig. 4.5d).

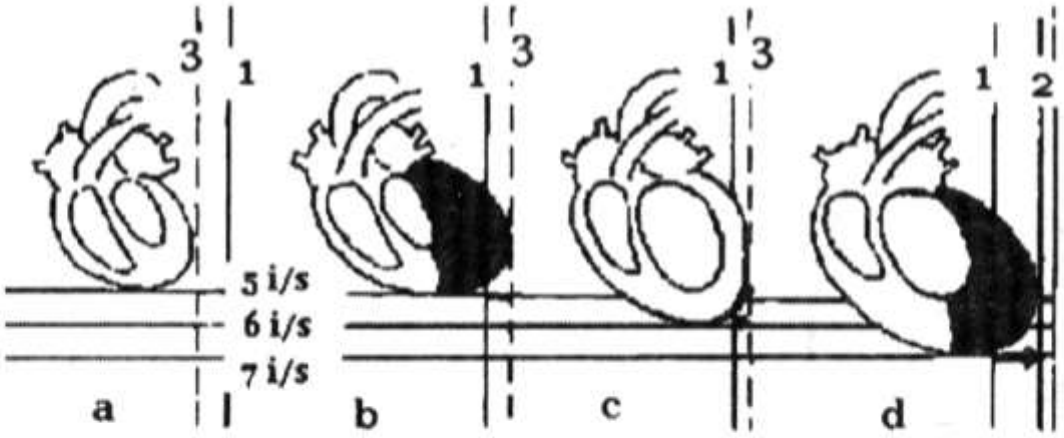


Fig. 4.5. Cardiac causes of the apex beat displacement
a) norm; b) left ventricular hypertrophy; c) left ventricular dilation;
d) left ventricular hypertrophy and dilation;
1 – left midclavicular line; 2 – left anterior axillary line; 3 – line of displacement.

Tab. 4.18. ***Diagnostic significance of the apex beat displacement.***

Apex beat is displaced outward 	Hypertrophy of the left ventricle: mitral regurgitation, aortic stenosis, essential hypertension, atherosclerotic cardiosclerosis, hypertrophic cardiomyopathy. Hypertrophy and dilation of the right ventricle: mitral stenosis, tricuspid regurgitation, cor pulmonale. Extracardiac causes: right-sided effusive pleurisy, hydrothorax, left-sided obstructive atelectasis
Apex beat is displaced outward and downward 	Considerable hypertrophy and dilation of the left ventricle: aortic regurgitation, considerable dilation of the left ventricle – dilative myocardiopathy

Apex beat is displaced outward and upward 	Elevated pressure in abdominal cavity, high diaphragm level: ascitis, meteorism, pregnancy, hepatomegaly
Apex beat is displaced inward 	Right-sided obstructive atelectasis
Apex beat is displaced inward and downward 	Low diaphragm level: asthenic constitution, visceroptosis

Apex beat properties: area, height, strength (or resistance).

A r e a of normal apex beat is near 2 cm^2. Different physiological or pathological conditions can cause diffuse (Tab. 4.19a) or restricted (Tab. 4.19b) apex beat.

Table 4.19a. ***Causes of the diffuse apex beat***

	Causes
Physiological	Deep inspiration, pregnancy, in subjects with thin chest wall, wide intercostal spaces (asthenic chest)
Pathological	Tumor of mediastinum, high diaphragm level (ascitis, meteorism), sclerotic affection of the lower border of the left lung, hypertrophy and dilation of the left ventricle (aortic regurgitation, aortic stenosis, arterial hypertension in myogenic dilation stage)

Table 4.19b. ***Causes of restricted apex beat***

	Causes
Physiological	Deep expiration Low diaphragm level
Pathological	Pulmonary emphysema Left-sided effusive pleurisy* Left-sided pneumothorax* Effusive pericarditis*

* in considerable accumulation of fluid or air apex beat is impalpable.

H e i g h t of the apex beat is the amplitude of vibration of the chest wall (Fig. 4.6).

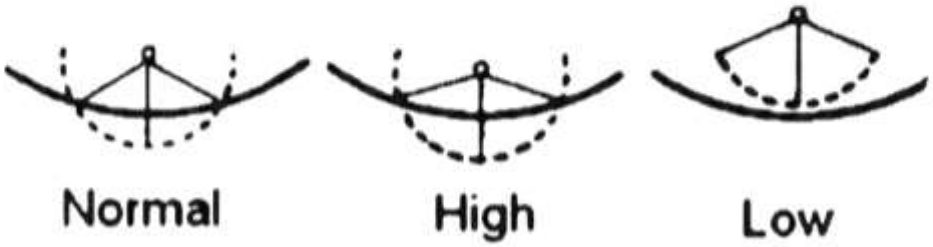


Fig. 4.6. Height of the apex beat.
Chest wall Amplitude of the apex beat

Usually, the height varies with the area. High and low apex beats are differentiated (Tab. 4.20a, 4.20b).

Tab. 4.20a. ***Physiological causes of the apex beat height changes.***

Properties	
High	Low
Physical exertion Emotional exertion	Obesity Overdeveloped muscles

Tab. 4.20b. ***Pathological causes of the apex beat height changes.***

Causes	Properties	
	High	Low
Noncardiac	Tumor of posterior mediastinum Diffuse toxic goiter (Basedow's disease) Fever	Pulmonary emphysema Effusive pleurisy Pneumothorax
Cardiac	Left ventricular hypertrophy	Effusive pericarditis

S t r e n g t h of the apex beat is determined by resistance of the heart apex to palpated fingers during systole.

Strong or resistant apex beat – sign of the left ventricular hypertrophy in aortic valvular diseases, arterial hypertension, and mitral regurgitation.

Weak apex beat is determined in pulmonary emphysema, obesity, left-sided effusive pleurisy, effusive pericarditis (in small amount of fluid).

Dome-like apex beat in VI–VII intercostals spaces on left anterior or midaxillary line, diffuse, high, strong is determined in aortic regurgitation.

Cat's purr symptom is thrill of the chest wall in the heart region, low vibrating murmur, which resembles purring of the cat. This sign is of great diagnostic significance. The cat's purr symptom is palpatory equivalent of cardiac murmur in organic heart valvular diseases (Fig. 4.7).

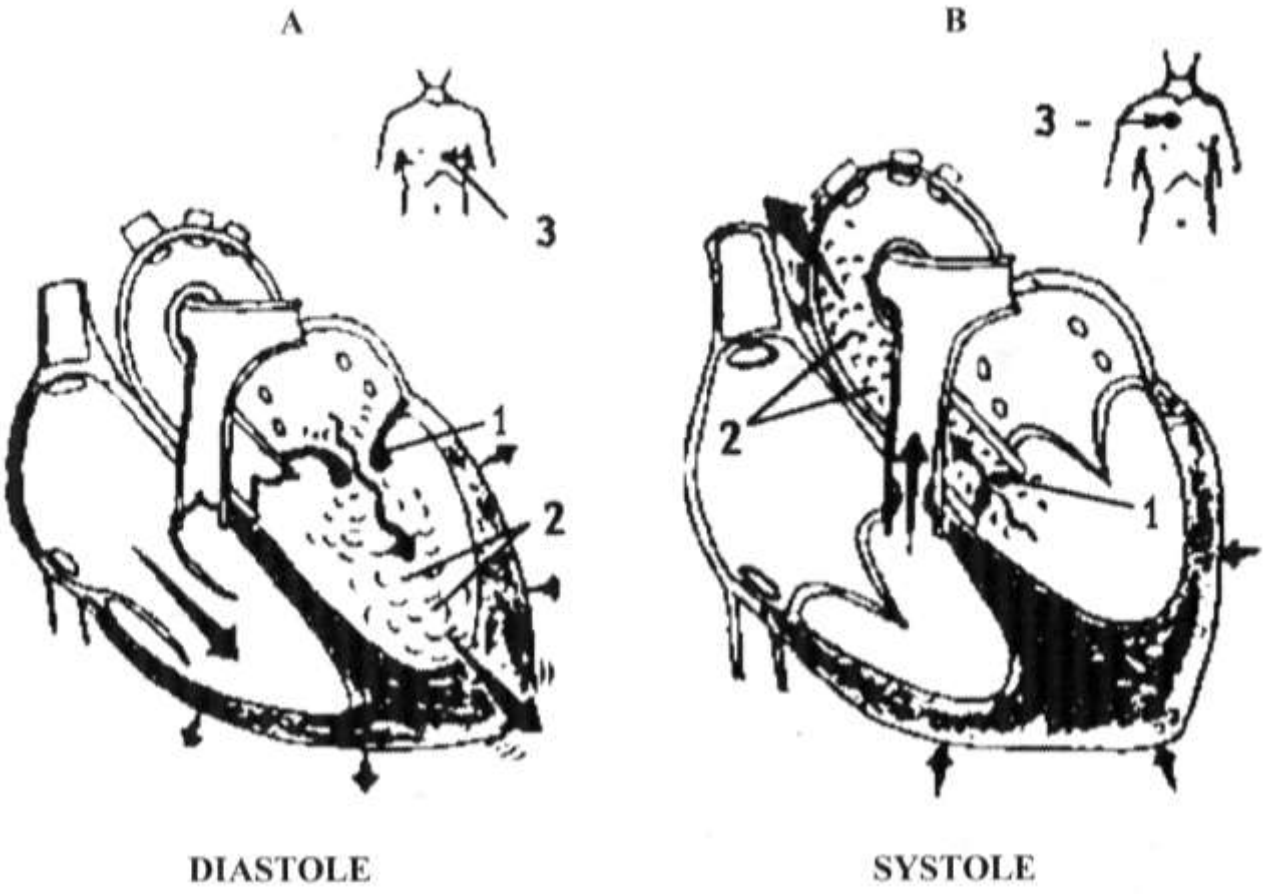


Fig. 4.7. Cat's purr symptom in cardiac pathology (Strutinsky A.V., et al, 1997):
A – *mitral stenosis;* B – *aortic stenosis;*
1 – narrowing of the valve orifice; 2 – vortex blood flow;
3 – location of systolic and diastolic thrill.

Depend on cardiac cycle phases systolic and diastolic thrill are differentiated (Fig. 4.8).

Palpation of the pulsated liver. True and transmitted pulsations are distinguished. True pulsation is observed in tricuspid regurgitation due to regurgitation of blood from the right ventricle to the right atrium during the systole, overfilling of the vena cava superior and liver veins. Therefore liver is enlarged during systole and positive venous pulse is determined. Transmitted pulsation is characterized by movement of the liver in the one direction, and is caused by transmission of heart contractions.

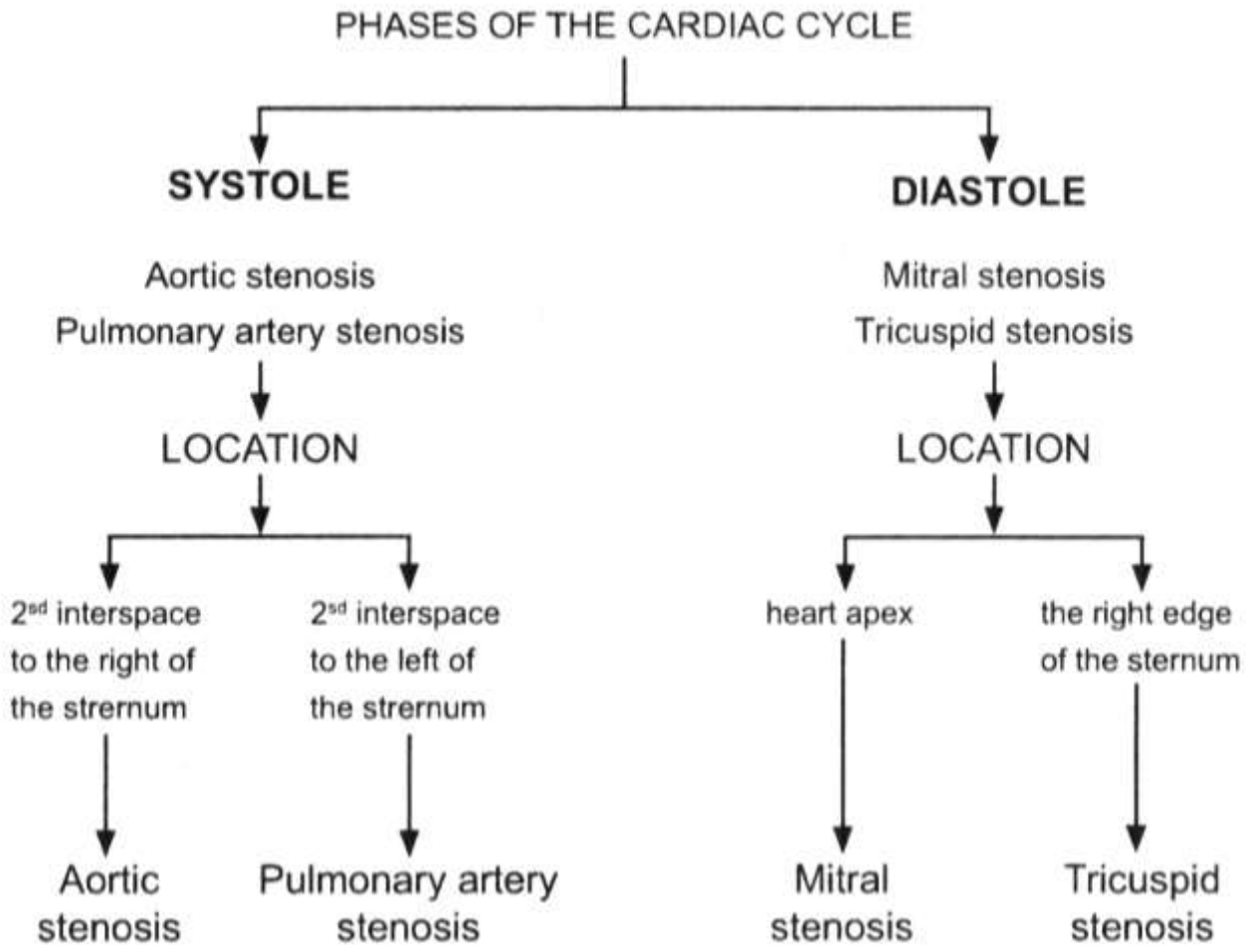


Fig. 4.8. Cat's purr symptom.

Percussion of the Heart

Examination plan:

1. Borders of the relative cardiac dullness;
2. Borders of the absolute cardiac dullness;
3. Transverse length of the heart;
4. Borders of the vascular bundle;
5. Configuration of the heart.

Determination of the size, position, and shape of the heart is based on the distinction between percussion sounds (Fig. 4.9). Being the airless organ, the heart gives dull percussion sound. But since it is partly covered by the lungs on its sides, the sound here is intermediate. The heart is surrounded by the lungs, which give clear pulmonary sound in percussion.

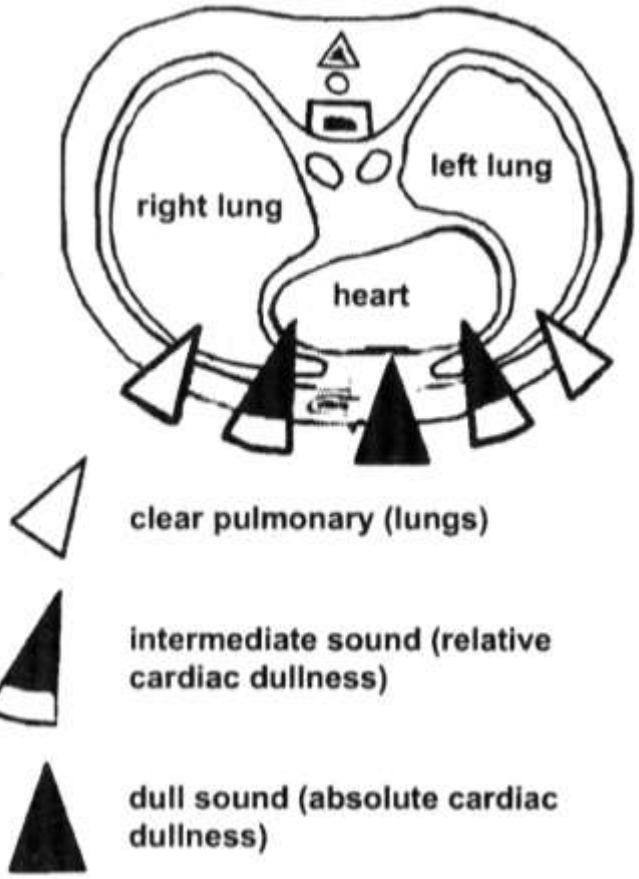


Fig. 4.9. Principle of the cardiac borders determination.

Projection of the heart chambers onto the chest is represented on the Figure 4.10.

The right contour of the heart is formed by the right atrium at the bottom and by the superior vena cava to the upper edge of the 3rd rib. The left contour is formed by the arch of the aorta, pulmonary trunk, auricle of the left atrium, and downward by the narrow strip of the left ventricle.

Relative cardiac dullness – is the projection of its anterior surface onto the chest. The relative cardiac dullness corresponds to the true borders of the heart.

Technique. Percussion can be done with the patient in both upright and lying position. It should, however, be remembered that the area of cardiac dullness in the vertical position is smaller than in horizontal. This is due to mobility of the heart and the displacement of the diaphragm as the patient change posture. The percussion stroke should be of medium strength.

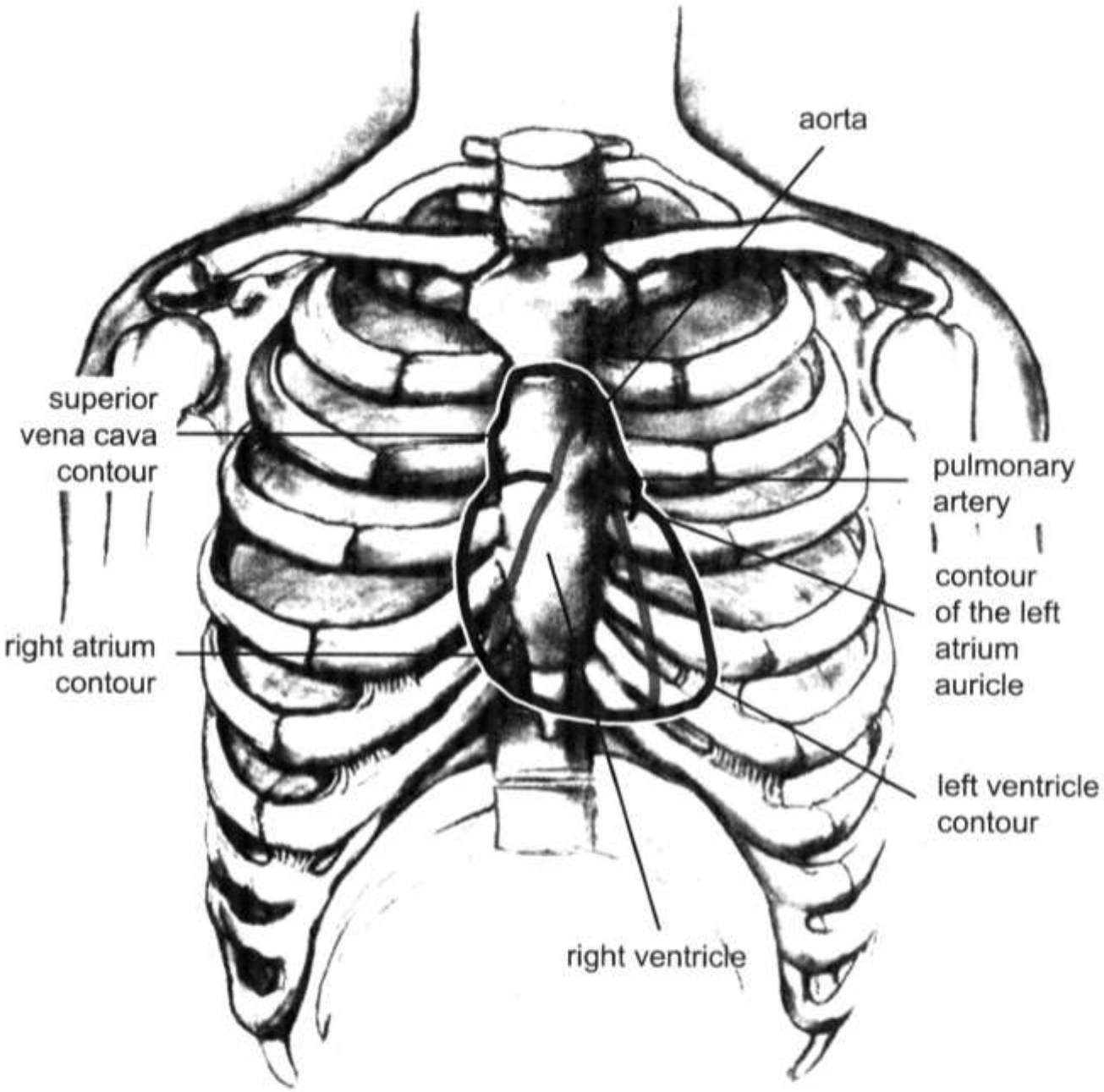


Fig. 4.10. Projection of the heart chambers onto the chest.

In order to determine the borders of the relative cardiac dullness the remotest points of cardiac contour are first found on the right, then at the top, and finally on the left (Fig. 4.11).

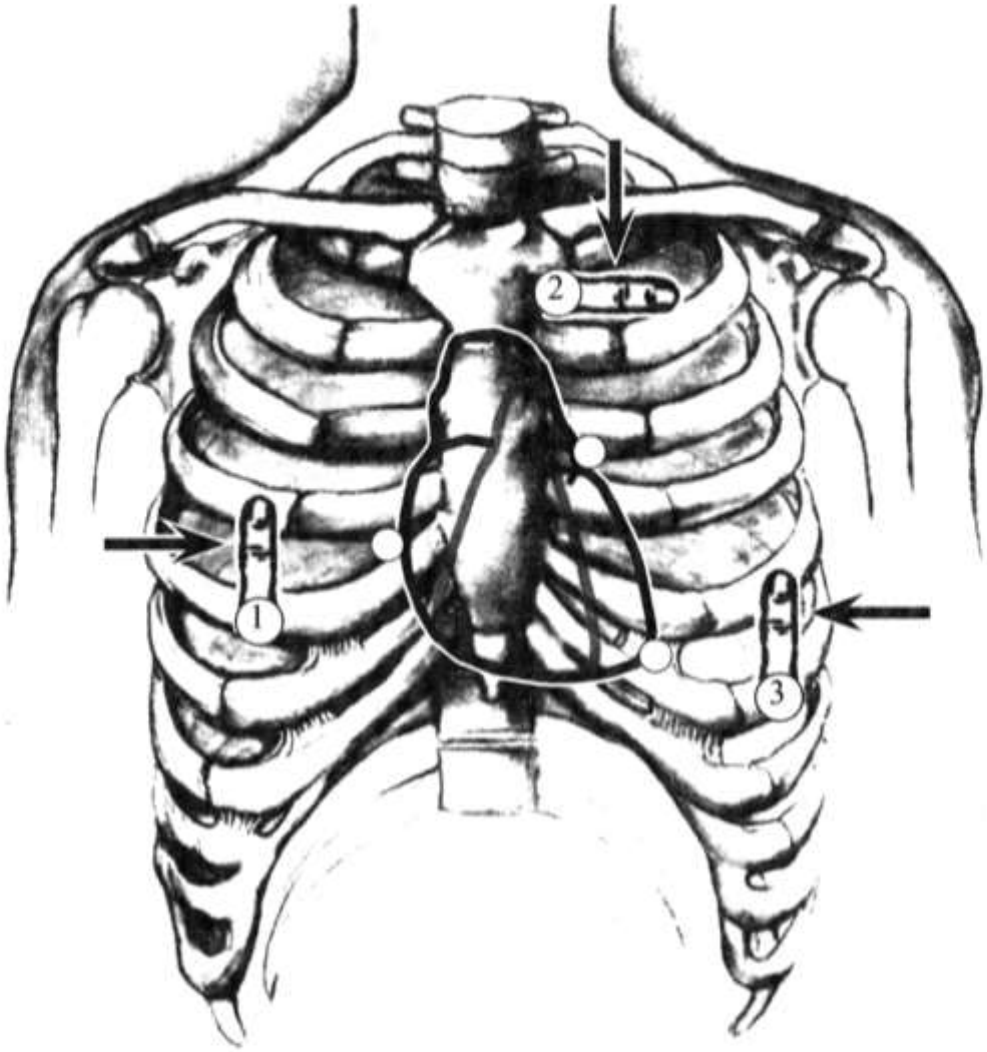


Fig. 4.11. Position of the pleximeter-finger during outlining the borders of the relative cardiac dullness.

The right border. As known the position of the heart depends on the diaphragm level, which is indicated by the lower border of the lung. The lower border of the right lung by loud percussion is, therefore, first determined in the midclavicular line (normally at the level of the 5th interspace). Then move your pleximeter-finger one interspace above, place it parallel to the sternum and change the percussion technique – medium strength percussion stroke. Continue percussion by moving the pleximeter-finger along the interspace toward the heart until the sound change. The right border of the relative cardiac dullness is marked by the edge of the finger directed to the more clear sound. The normal right border of the relative cardiac dullness is in the 4th intercostal space 1 cm outward from the right edge of the sternum (Tab. 4.21).

Tab. 4.21. ***Borders of the relative cardiac dullness.***

Borders	Location	Formed by
Right	4th interspace 1 cm laterally of the right edge of the sternum	Right atrium
Upper	3rd interspace in the left parasternal line	Cone of the pulmonary artery, the auricle of the left atrium
Left	5th interspace 1.5 cm medially of the left midclavicular line	Left ventricle

The upper border. In order to determine the upper border of the relative cardiac dullness place pleximeter-finger in the 1st intercostal space in the left parasternal line and move it downward until the percussion sound change. The normal upper border of the relative cardiac dullness is in the 3rd intercostal space in the left parasternal line.

The left border is determined in the interspace, where the apex beat is palpated. Place your pleximeter-finger laterally in this intercostal space parallel to the sought border and move it toward the sternum. If the apex beat is impalpable you should start percussion in the 5th intercostal space from the left anterior axillary line. The left border of the relative cardiac dullness is in the 5th intercostal space 1.5 cm medially of the left midclavicular line.

The borders of the relative cardiac dullness can be modified by physiological and pathological factors (Tab. 4.22).

Tab. 4.22. ***Displacement of the relative cardiac dullness borders.***
Physiological and pathological causes.

Extracardiac		Cardiac
Physiological	Pathological	
Position of the body Constitutional types Diaphragm level (pregnancy)	Pulmonary pathology Fluid, air in the pleural cavity Diaphragm level (ascitis)	Changes of the heart chambers size and volume

There are different clinical variants either isolated and combined displacement of the relative cardiac dullness borders (Tab. 4.23).

Tab. 4.23. ***Clinical variants of the relative cardiac dullness borders displacement.***

Heart borders displacement	Extracardiac causes	Changes of the heart chambers size and volume	
		Conditions	Clinical variants
To the right	*Left-sided* pneumothorax, effusive pleurisy,hydrothorax. *Right-sided* obstructive atelectasis	Dilation of the right ventricle Dilation of the right atrium and ventricle	Pulmonary artery stenosis Tricuspid stenosis, chronic pulmonary diseases (cor pulmonale)
To the right and upward		Dilation of the right ventricle and left atrium	Mitral stenosis
Upward and to the left		Dilation of the left atrium and ventricle, protrusion of the pulmonary artery cone	Mitral regurgitation
To the left	*Right-sided* pneumothorax, effusive pleurisy, hydrothorax. *Left-sided* obstructive atelectasis	Dilation of the right ventricle Hypertrophy and dilation of the left ventricle	Mitral stenosis Aortic stenosis, arterial hypertension, atherosclerotic cardiosclerosis
To the left and downward		Dilation of the left ventricle	Aortic regurgitation

Absolute cardiac dullness is the projection of the anterior surface of the heart, which is not covered by the lungs onto the chest. Absolute cardiac dullness is formed by the right ventricle.

Technique. The right border of the absolute cardiac dullness is first elicited. Place your pleximeter-finger on the right border of the relative cardiac dullness parallel to the sternum, and using light percussion stroke move it medially to dullness (Fig. 4.12).

To determine the upper border place pleximeter-finger on the upper border of the relative cardiac dullness and move downward to dullness.

To outline the left border place pleximeter-finger slightly outside the left border of the relative cardiac dullness and move medially.

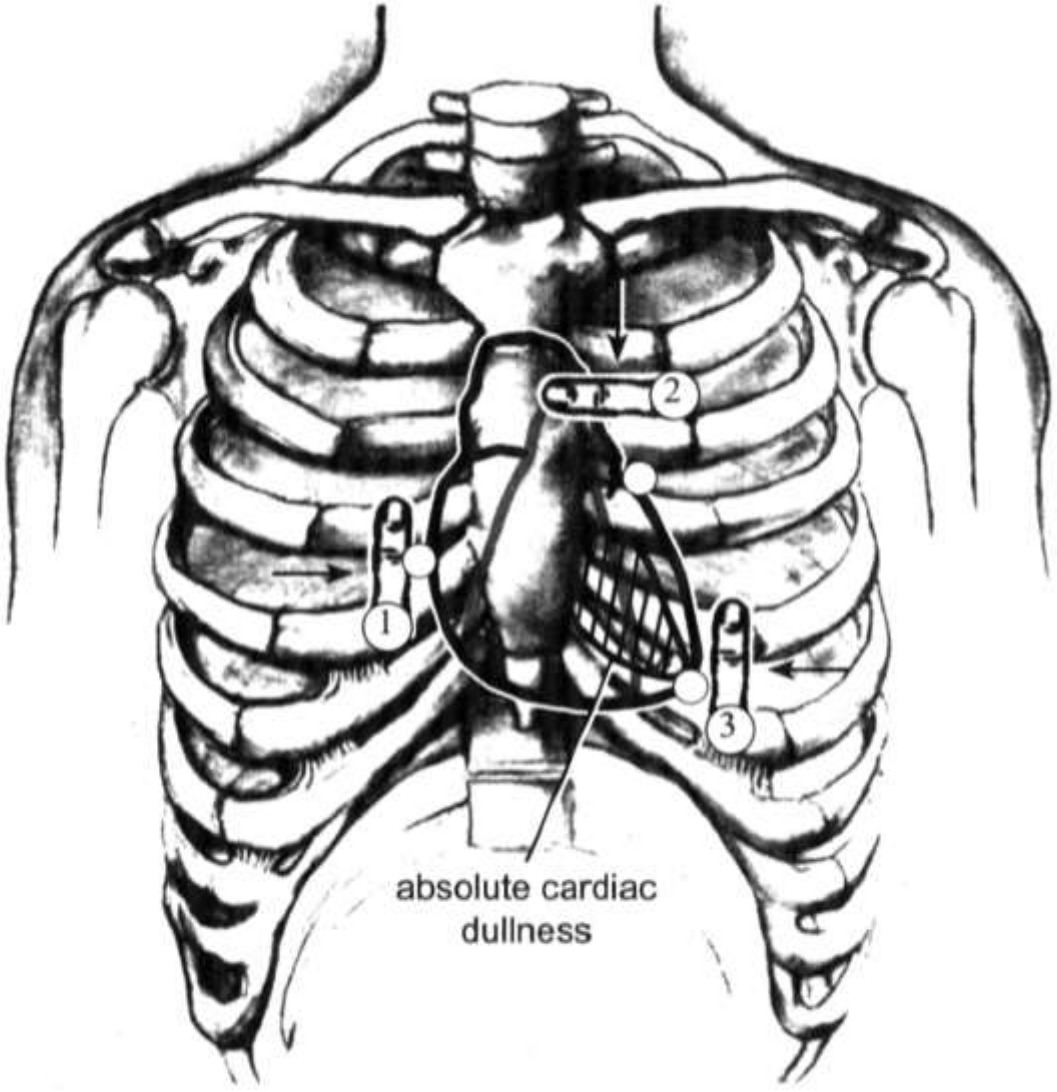


Fig. 4.12. Position of the pleximeter-finger during outlining the borders of the relative cardiac dullness.

Normal borders of the absolute cardiac dullness:

The right – along the left edge of the sternum from 4th to 6th rib;

The upper – lower edge of the 4th rib along left parasternal line;

The left – 5th intercostal space 0.5 cm medially of the left border of the relative cardiac dullness.

Tab. 4.24. ***Changes of absolute cardiac dullness area***

Decreasing	Increasing
• Low diaphragm level • Pulmonary emphysema • Left-sided pneumothorax	• Pregnancy • High diaphragm level (ascitis, meteorism) • Tumor of mediastinum • Dilation, hypertrophy of the right ventricle

Transverse length of the heart is the sum of distance from the right border of the relative cardiac dullness to the anterior median line (3–4 cm) and from the left border of the relative cardiac dullness to the median line (8–9 cm). The transverse length is measured by a measuring tape, and normally is 11–13 cm (Fig. 4.13).

Enlargement of the cardiac transverse length is observed in hypertrophy and dilation of the heart chambers.

The borders of the vascular bundle are determined by light percussion in the 2nd intercostal space from midclavicular line to the right and left toward the sternum. The borders of the vascular bundle are normally found along the edges of the sternum. The normal width of the vascular bundle is 4–6 cm (Fig. 4.13).

The width of the vascular bundle is increased in:

- Dilation of the pulmonary artery in elevated pressure in it;
- Aortic aneurysm;
- Syphilitic mesoaortitis.

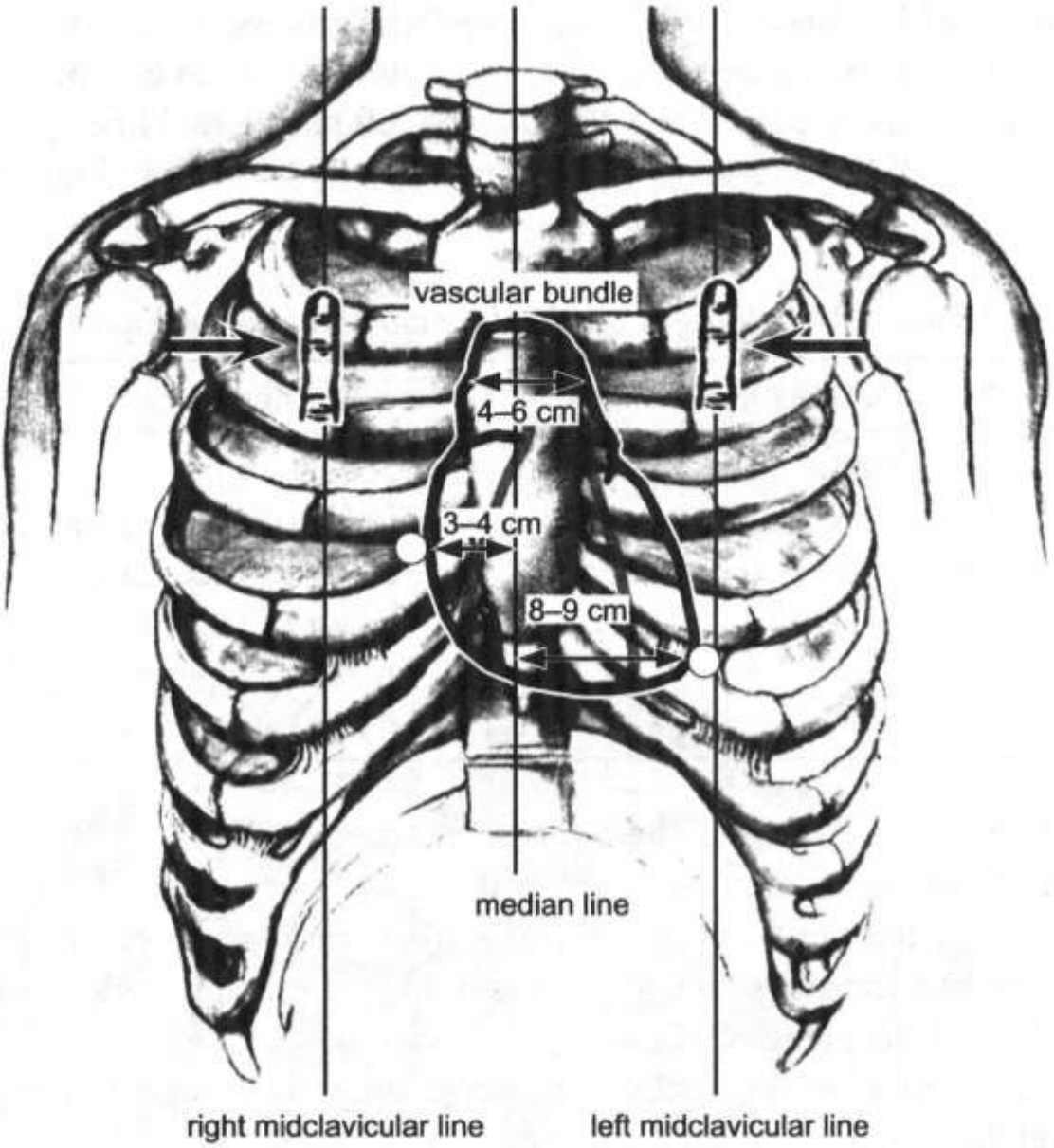


Fig. 4.13. Transverse length of the heart and the borders of the vascular bundle.

Configuration of the heart can be determined by percussion in the 2nd, 3rd, 4th intercostal spaces on the right and 2nd, 3rd, 4th, 5th intercostal spaces on the left. The pleximeter-finger is moved parallel to sought border. The elicited points are marked on the patient's skin and connected by a line.

Normal configuration of the heart:

Right contour: 2nd intercostal space along right sternal edge,
3rd intercostal space along right sternal edge,
4th intercostal space 1 cm laterally of right sternal edge;

Left contour: 2nd intercostal space along left sternal edge;
3rd intercostal space along left parasternal line;
4th and 5th intercostal spaces 1,5 cm medially of left midclavicular line.

The angle formed by the bundle of the great vessels and left ventricle is called *waist of the heart*. In normal configuration of the heart this angle is dull (Fig. 4.14a).

In pathological conditions mitral (Fig. 4.14b), aortic (Fig. 4.14c), and trapezium (Fig. 4.14d) configurations of the heart are distinguished.

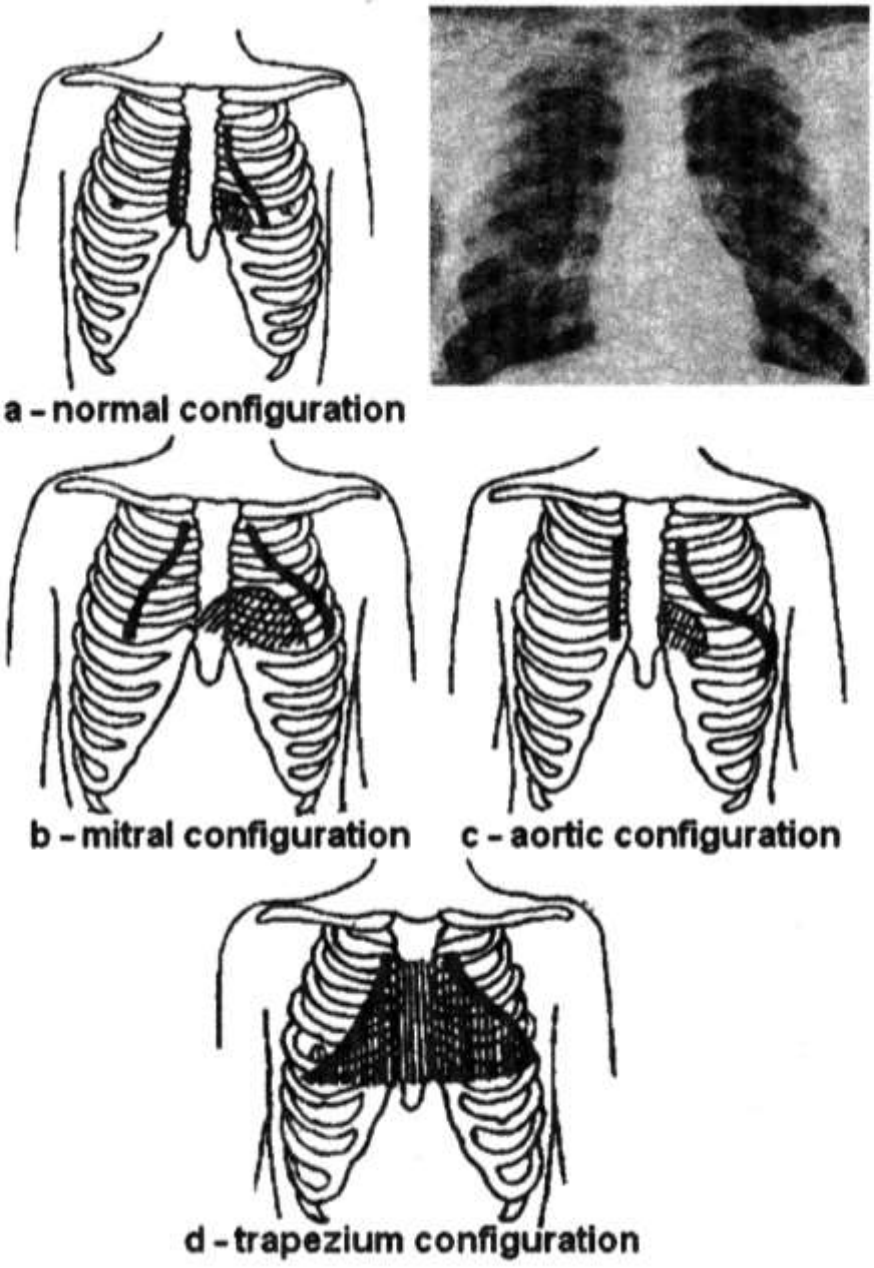
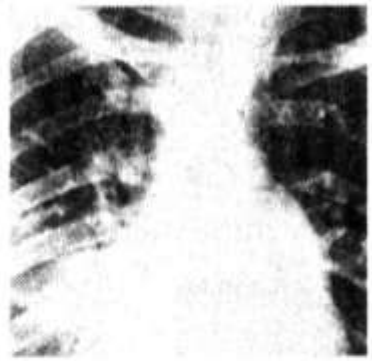
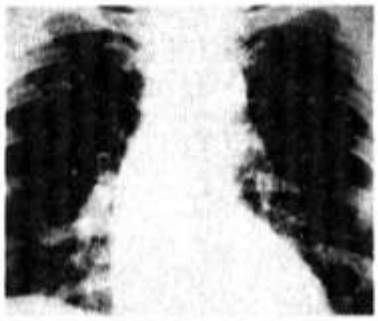
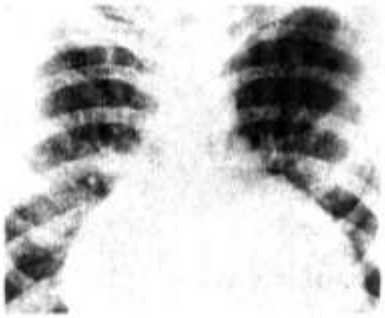
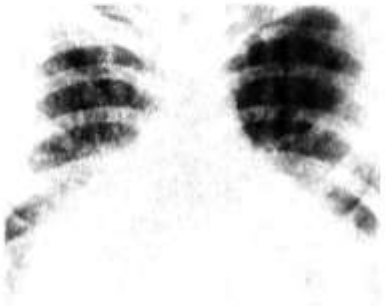


Fig 4.14. Configuration of the heart.

Tab. 4.25. ***Pathological configurations of the heart.***

Configuration	Features	Conditions	Pathological state
Mitral 	Protrusion of the upper part of the left contour, indistinct or protruded waist of the heart	Dilation of the left atrium and blood pressure elevation in the pulmonary artery	Mitral stenosis and regurgitation
Aortic 	Protrusion of the lower part of the left contour, pronounced waist of the heart	Dilation of the left ventricle	Aortic stenosis and regurgitation
Trapezium 	Symmetrical protrusion of both cardiac contours	Transudate or exudate in the pericardium	Effusive pericarditis, hydropericardium
Cor bovinum 	Protrusion of all cardiac contours	Myogenic dilation of both ventricles	Dilative cardiomyopathy

Auscultation of the Heart

Auscultation of the heart – is objective method based on listening a noise within the heart during cardiac cycle.

Examination plan:

1. Heart rhythm;
2. Heart rate;
3. Heart sounds (loudness, timbre);
4. Presence of the splitting and additional sounds;
5. Presence of the heart murmurs.

Technique. To obtain the most information from cardiac auscultation and to assess correctly the findings, it is necessary to know the sites of valves projection on the chest wall and listening points of the heart.

Since the sites of the valves projection on the chest are very close to one another (see Fig. 4.15), it is difficult to assess which valve is affected if listen them in the points of their actual projection. Therefore the heart sounds are auscultated in the certain listening points where sounds of each valve can be better heard (see Fig. 4.16, Tab. 4.26).

Auscultation should be performed in the order of decreasing frequency of valves affection: 1 – mitral valve, 2 – aortic valve, 3 – pulmonary valve, 4 – tricuspid valve. The fifth listening point to the left of the sternum at the

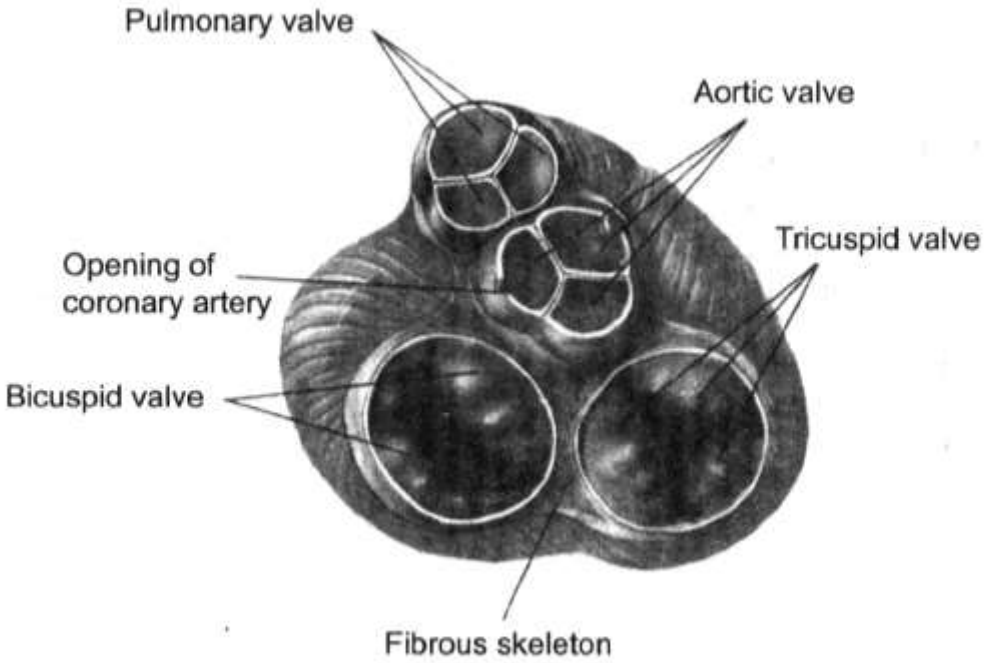


Fig. 4.15. Heart in systole: viewed from base with atria removed.

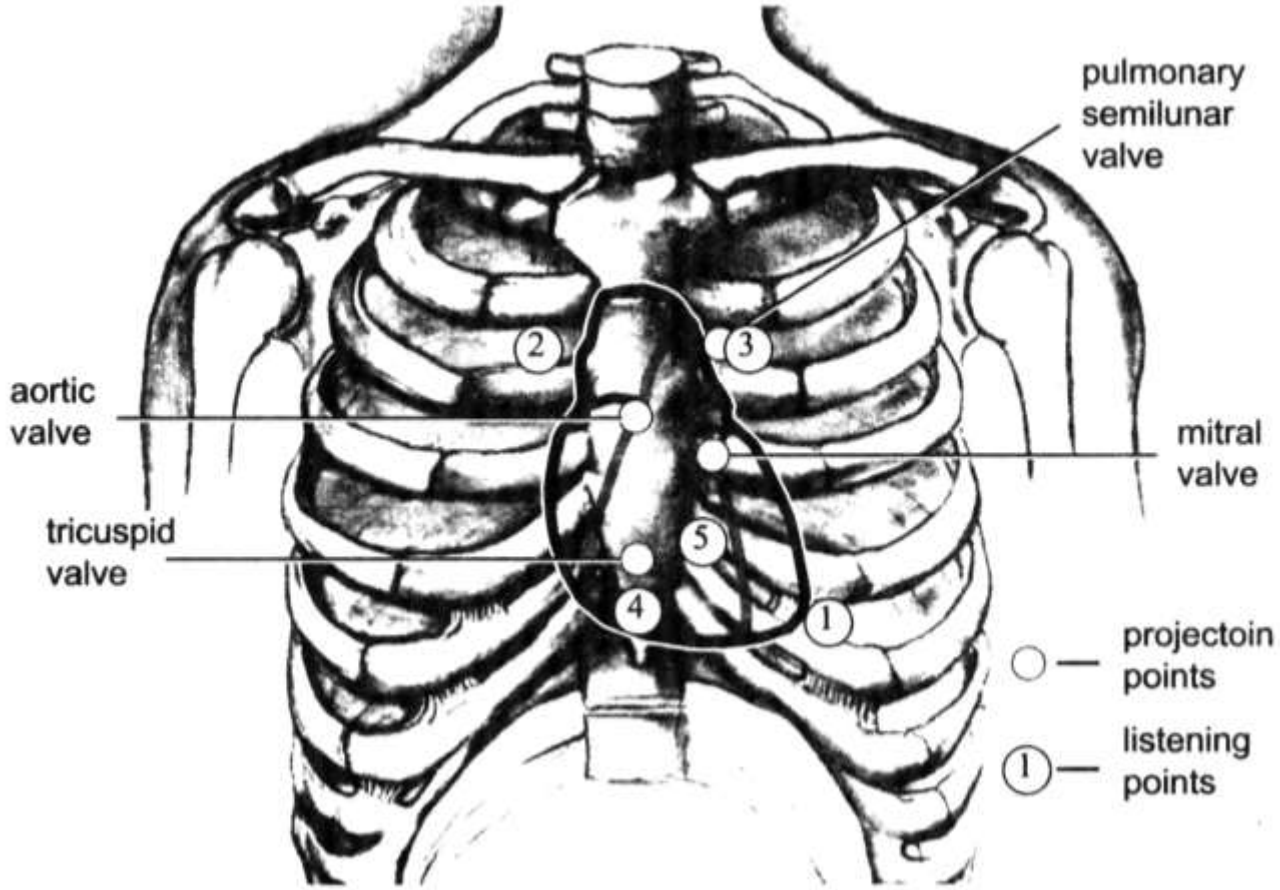


Fig. 4.16. Projection of the heart valves on the chest wall and standard listening points of the heart.

3rd and 4th costosternal articulation – so-called Botkin-Erb's point, was proposed to assess aortic valve sound.

Tab. 4.26. ***Projection of the heart valves on the chest wall and standard listening points of the heart.***

Valve	Mitral	Aortic	Pulmonary	Tricuspid
Site of projection	To the left of the sternum at the level of the 3rd costosternal articulation	In the middle of the sternum at the level of the 3rd costosternal articulation	In the 2nd intercostal space 1–1.5 cm to the left of the sternum	On the sternum midway between 3rd left and 5th right costosternal articulation

Listening point	Heart apex	2nd intercostal space to the right of the sternum	2nd intercostal space to the left of the sternum	Base of the xiphoid process

Cardiac auscultation is performed using a stethoscope in a quiet room to avoid the distracting noises of normal activity. Heart sounds are better heard if the patient keeps breath for short period so that the respiratory sounds should not interfere with auscultation of the heart. The mitral valve sound in its pathology is better heard when the patient lies on the left side; aortic valve sound – in the upright posture of the patient or when he lies on the right side.

Normal heart sounds. The noise produced by a working heart is called heart sounds. In auscultation two sounds can well heard in healthy subjects: the first sound (S_1), which is produced during systole, and the second sound (S_2), which occur during diastole (Fig. 4.17)

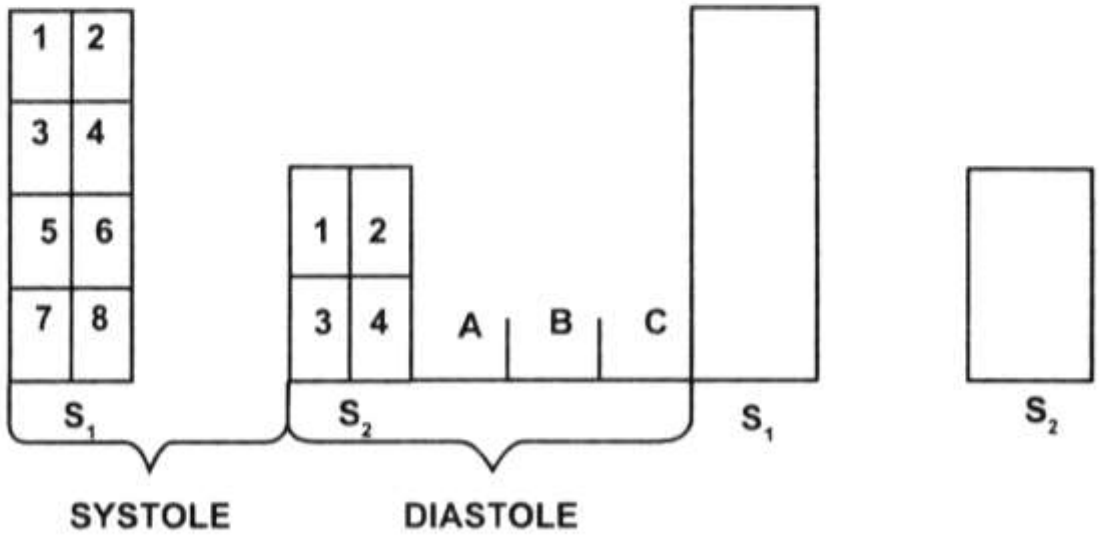


Fig. 4.17. S1 and S2 components: S1 components: *1, 2 – atrial; 3, 4 – valvular; 5, 6 – muscular; 7, 8 – vascular; S2 components: 1, 2 – valvular; 3, 4 – vascular. A – protodiastole, B – mesodiastole, C – presystole.*

S_1 consists of four pair components: **atrial component**: 1 – tension and contraction of the right atrium, 2 – tension and contraction of the left atri-

um; **valvular component**: 3 – closure and vibration of mitral valve cusps, 4 – closure and vibration of tricuspid valve cusps; **muscular component**: 5 – isometric tension and contraction of the right ventricle, 6 – isometric tension and contraction of the left ventricle; **vascular component**: 7 – vibration of the initial portion of the aorta, 8 – vibration of the initial portion of the pulmonary trunk.

S_2 consists of two pair components: **valvular component**: 1 – closure and vibration of the aortic valve cusps, 2 – closure and vibration of the pulmonary valve cusps; **vascular component**: 3 – vibration of the aortic walls, 4 – vibration of pulmonary trunk walls.

The *first heart sound*, a dull, prolonged 'lub' marks the onset of the ventricular systole. The *second heart sound*, a short, sharp 'dup' occurs at the beginning of ventricular diastole. Diastole is subdivided in protodiastole, mesodiastole, and presystole. Both heart sounds can be heard over precordium, but their intensity changes depend on nearness of valves that take part in formation of S_1 or S_2.

In rhythmic heart activity S_1 and S_2 can be differentiate according following signs (Tab. 4.27).

Tab. 4.27. ***Differential signs of S_1 and S_2.***

Main sign	First sound	Second sound
Listening point	Heart apex	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 apex beat	Synchronous	Follows the apex beat
Relation to carotid pulse	Synchronous	Asynchronous

A weak, low-pitched, dull *third sound* (S_3) is sometimes heard and is thought to be caused by vibration of the walls of the ventricles when they are suddenly distended by blood from atria (passive rapid filling), occurs

0.12–0.15 s after the onset of S_2. The third sound is heard most clearly at the apex of the heart with the bell of a stethoscope; it may be normal in children, adolescents, or very thin adults, or in patients with high cardiac output.

The *fourth heart sound* (S_4) is a low-pitched, presystolic sound produced in the ventricle during ventricular filling; it is associated with an effective atrial contraction and is best heard with the bell piece of the stethoscope.

Cardiac rhythm. In healthy subjects S_1 and S_2, S_2 and S_1 follow one another at regular intervals: the heart activity is said to be rhythmic or regular. When the cardiac activity is arrhythmic, the heart sounds follow at irregular intervals.

Heart rate (HR) in normal conditions is 60-80 beats per minute. Acceleration of the heart rate to more than 90 beats per minute is called *tachycardia*. A heart rate less than 60 beat per minute is called *bradycardia*.

In **heart sounds analysis** their loudness and timbre should be assessed. Loudness of the heart sounds depends on the point of auscultation (Tab. 4.26, Fig. 4.18).

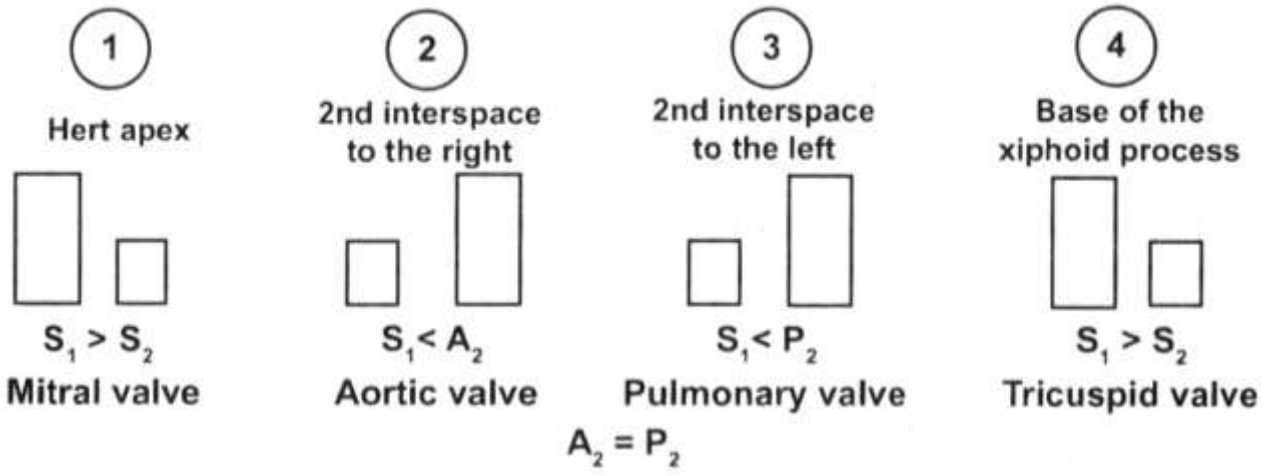


Fig. 4.18. Relation of the heart sound loudness in the listening points.

In the first and fourth listening points first heart sound is louder than second one $S_1 > S_2$, in the second and third – second heart sound is louder than the first $S_1 < S_2$, the second sound over aorta and pulmonary artery is of the same loudness $A_2 = P_2$.

Loudness of the heart sounds depends on several factors (Tab. 4.28).

Tab. 4.28. ***Loudness of the heart sounds.***

Influenced factors.

Factors	Sounds		
	S_1	S_2	S_1+S_2
1. Listening point	+	+	
2. Condition of atrioventricular valves	+		
3. Ventricular contractility	+	+	+
4. Volume of ventricular filling by blood	+		
5. Velocity of ventricular contraction	+		
6. Condition of semilunar valves of the aorta and pulmonary artery		+	
7. Condition of initial portion of the aorta		+	
8. Pressure in the lesser and greater circulation		+	
9. Properties of the sound wave transmission			+
10. Properties of the nearest to the heart organs			+

The loudness of the heart sounds can be changed in several physiological and pathological conditions. Loudness of one or both heart sounds may increase or decrease.

Tab. 4.29. ***Both heart sounds decreasing*** (in all listening points).

CAUSES			
Extracardiac		Cardiac	
Physiological	Pathological	Primary	Secondary
Excessive muscles development	Obesity Swelling of the chest wall Pulmonary emphysema Effusive pericarditis Effusive left-sided pleurisy	Myocarditis Myocardiosclerosis Myocardial infarction Myocardiopathy	Collapse Shock

Tab. 4.30. ***Both heart sounds increasing***
(in all listening points).

CAUSES	
Physiological	Pathological
Thin chest wall Nervous excitement Hard physical exertion	Thyrotoxicosis Anemia Wrinkled pulmonary edges Inflammatory consolidation of pulmonary edges Fever

Changes in only one heart sound are very important diagnostically.

Tab. 4.31. ***Increased loudness of the first heart sound at the heart apex.***

Causes	Mechanism
Mitral stenosis Tachycardia Left ventricular extrasystole Complete atrioventricular block in synchronous contraction of atria and ventricles – 'pistol-shot' sound according Strazhesko	Not adequate filling of the left ventricular cavity during diastole, quick and intense contraction of the myocardium

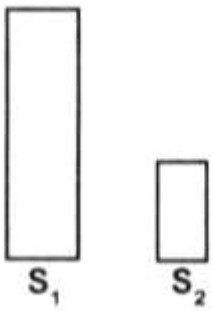


Fig. 4.19. Heart apex.
S_1 more than 1,5 times louder than S_2

Tab. 4.32. ***Decreased loudness of the first heart sound at the heart apex.***

Causes	Mechanism
Mitral regurgitation Aortic regurgitation Aortic stenosis	Anatomic abnormalities of the valve Absence of closed valve period Overfilling of the left ventricular cavity

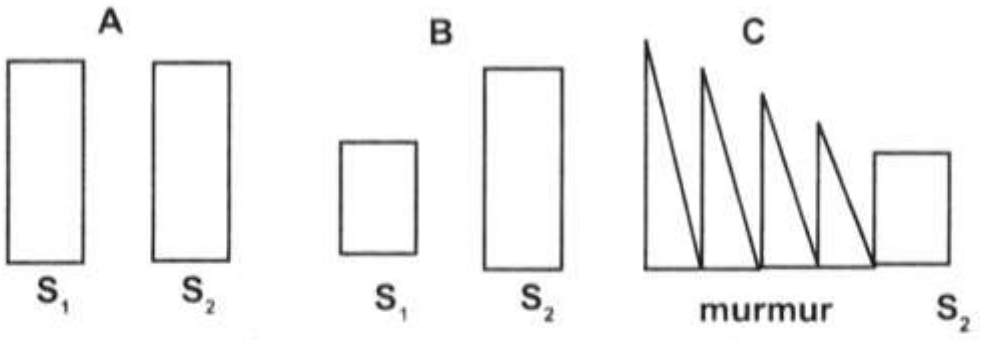


Fig. 4.20. Heart apex.
A – the first heart sound is of the same loudness as the second heart sound;
B – the first heart sound is less loud than the second;
C – systolic murmur instead of the first heart sound is heard.

Tab. 4.33. ***Different loudness of the first heart sound at the heart apex.***

Causes	Mechanism
Complete heart block Atrial fibrillation Extrasystolic arrhythmia Ventricular flutter	Different ventricular filling in each cardiac cycle

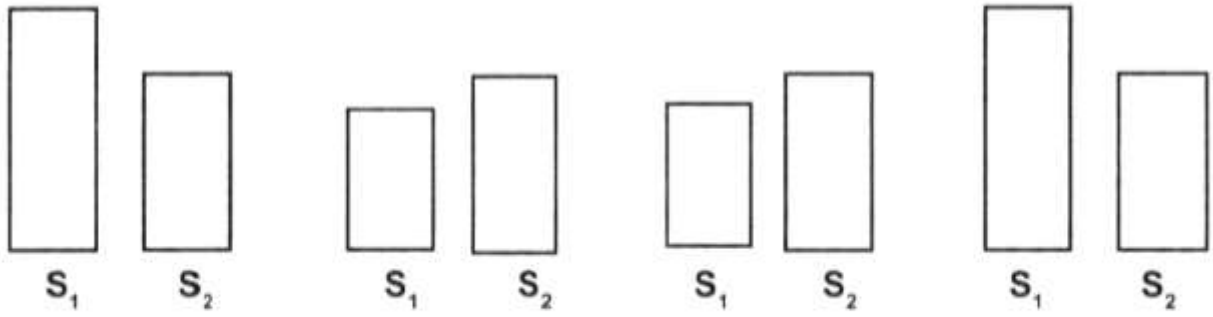


Fig 4.21. Heart apex. The first heart sound is not of the same intensity in the different cycles.

Tab. 2.34. ***Accentuated second heart sound over aorta.***

Causes		Mechanism
Physiological	Pathological	
Emotional exertion Physical exertion	Essential hypertension Symptomatic hypertension Aortic atherosclerosis Syphilitic mesoaortitis	Pressure elevation in the greater circulation, decreased elasticity of the aorta

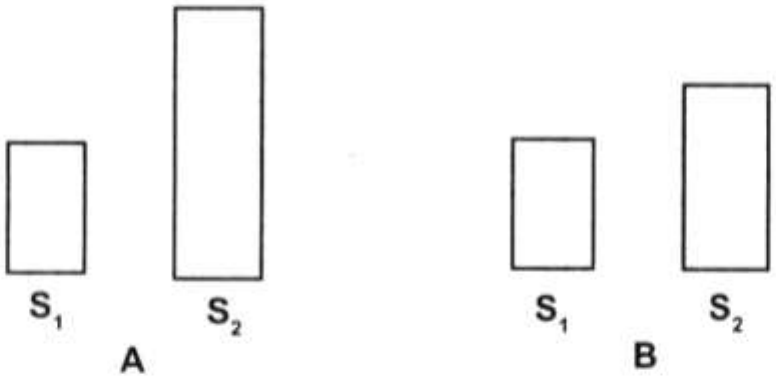


Fig. 4.22. Second intercostal space to the right (A), and to the left (B). The second sound over aorta is louder than over pulmonary artery.

Tab. 4.35. ***Decreased second sound over aorta.***

Causes	Mechanism
Aortic regurgitation (A) Aortic stenosis (B)	Anatomic changes of valve (A) Low pressure in the aorta at the beginning of the diastole (B)

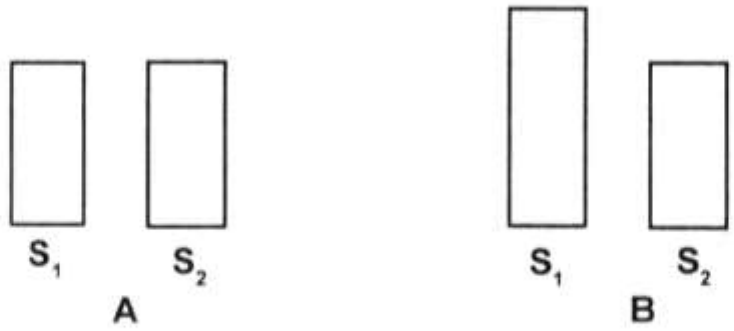


Fig. 4.23. Second intercostal space to the right. Loudness of the second heart sound is the same as the first heart sound (A), the second heart sound loudness is less than the first one (B).

Tab. 4.36. ***Accentuated second heart sound over pulmonary artery.***

Causes		Mechanism
Physiological	Pathological	
In children Thin chest wall	Mitral valvular diseases Diseases of the broncho-pulmonary system Adhesion of the pleural layers Kyphoskoliotic chest	Pressure elevation in the pulmonary circulation

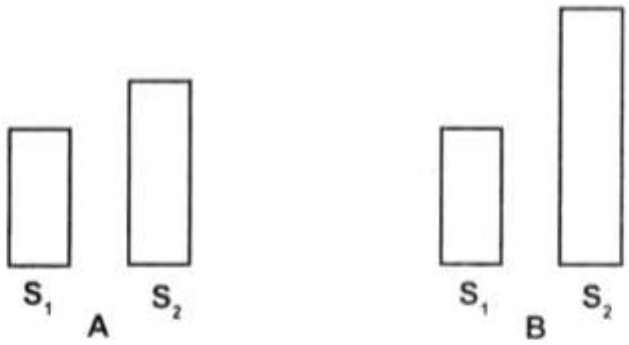


Fig. 4.24. Second intercostal space to the right (A) and to the left (B). S_2 over pulmonary artery is louder than over aorta.

Tab. 4.37. ***Decreased second sound over pulmonary artery.***

Causes	Mechanism
Pulmonary artery stenosis Pulmonary regurgitation	Anatomical valve changes Low pressure in the pulmonary artery before diastole onset

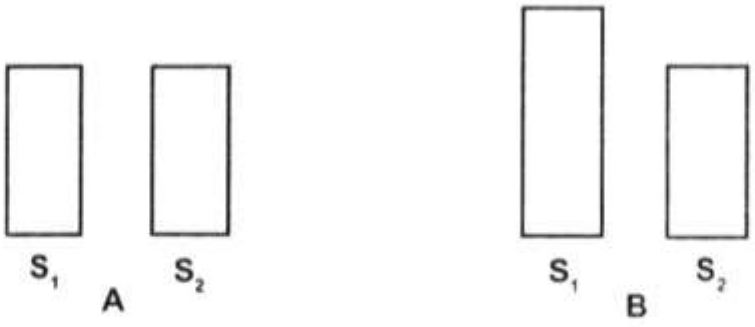


Fig. 4.25. Second intercostal space to the left.
Loudness of the second heart sound is the same as the first heart sound (A), the second heart sound loudness is less than the first one (B).

Tab. 4.38. ***Decreased loudness of the first heart sound at the base of the sternum.***

Causes	Mechanism
Tricuspid regurgitation	Anatomic changes of the valve Absence of closed valves period Overfilling of the right ventricular cavity

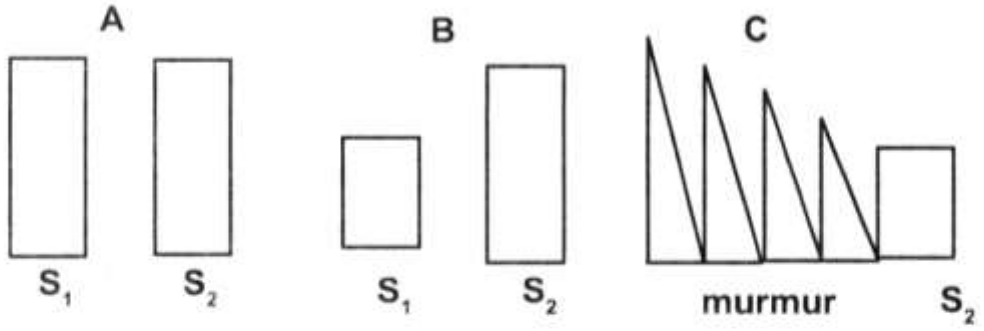


Fig. 4.26. Base of the sternum.
A – the first heart sound is of the same loudness as the second heart sound;
B – the first heart sound is less loud than the second;
B – systolic murmur instead of the first heart sound is heard.

Reduplication and splitting of the heart sounds may be revealed in auscultation, which are caused by asynchronous work of right and left chambers of the heart.

Reduplication – two short sounds follow one another are heard instead S_1 or S_2.

Splitting – two short sounds follow one another at a short interval, and therefore they are not perceived as two separate sounds (Fig. 4.27).

Splitting of the two high-pitched components of S_1 by 10–30 ms is a normal phenomenon, which is recorded by phonocardiography. The third component of S_1 is attributed to mitral valve closure, and the fourth to tricuspid valve closure. Widening of the interval between these two components is heard as S_1 splitting or reduplication at the heart apex or at the base of the xiphoid process.

Physiological splitting of S_1 is heard in the upright position of the patient during very deep expiration, when the blood delivers to the left atrium

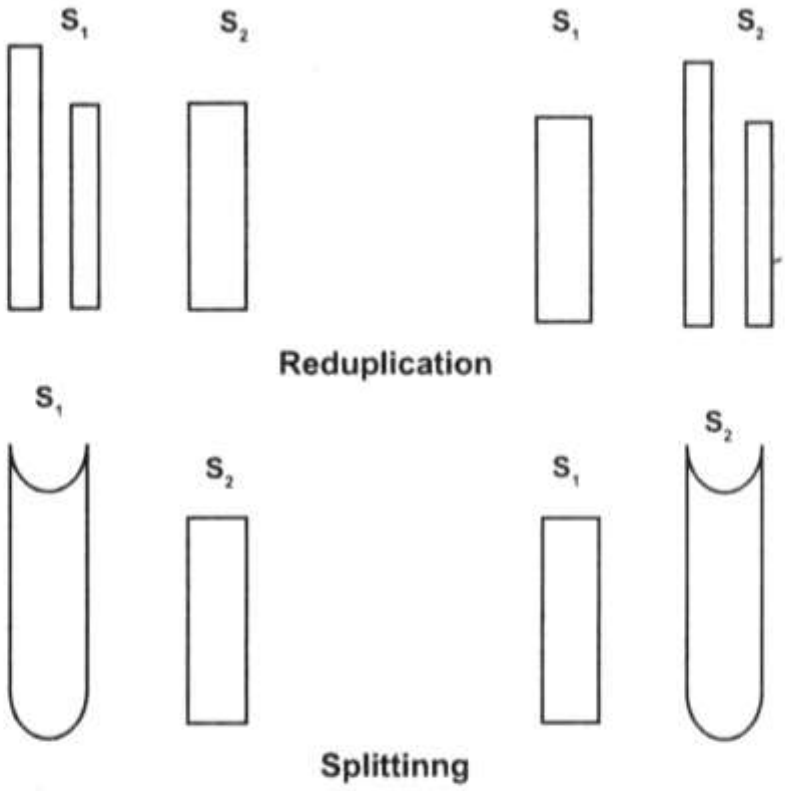


Fig. 4.27. Reduplication and splitting of S_1 and S_2.

with a greater force to prevent the closure of the mitral valve. The valvular component of the left ventricle is therefore splits and is perceived as a separate sound.

Pathological splitting of S_1 is due to:

- Sclerosis of the initial part of the aorta;
- Decreased left ventricular contractility in hypertension, nephritis leads to asynchronous contraction of the ventricles;
- Aortic regurgitation ("interrupted contraction of the left ventricle – Obraztsovs' bisystolia");
- Complete right bundle branch block and resulting delay in onset of the right ventricular systole.

Splitting of S_2 occurs more frequently than S_1.

Physiological splitting of S_2 into audibly distinct aortic (A_2) and pulmonic (P_2) components is due to a normal physiological cause: respiration. Normally, the aortic valve closes just before the pulmonary valve, but they

are so close together that the sound is a uniform and instantaneous S_2. When a person takes in a deep breath, the decrease in intrathoracic pressure causes an increase in venous return. This causes the right atrium and ventricle to fill slightly more than normal, and it takes the ventricle slightly longer during systole to eject this extra blood. This delay in ejection forces the pulmonary valve to stay open a bit longer than usual, and the normally small difference between aortic and pulmonary valve closure becomes noticeable as a split S_2 at the heart base.

Pathological splitting of S_2 may be due to many causes: delayed activation of the right ventricle in right bundle branch block, left ventricular ectopic beats, a left ventricular pacemaker; or delayed pulmonic valve closure because of right ventricular volume overload associated with right ventricular failure.

Pathological splitting of S_2 occurs in:

- Mitral stenosis – delayed pulmonic valve closure because of right ventricular volume overload, and prolongation of the right ventricular ejection;
- Pulmonary stenosis or pulmonary embolism is characterized by prolongation of the right ventricular systolic ejection period and thus delay closure of the pulmonic valve;
- Shortening of the left ventricular systole and early aortic valve closure occurring with mitral regurgitation because blood passes in two direction – into aorta and in the left atrium, also may produce splitting of S_2;
- In the patients with a ventricular septal defect blood ejected into aorta and throughout the defect to the right ventricle, left ventricular systole is thus shortened, and occurs splitting of the S_2 a result early aortic component of S_2.
- An atrial septal defect leads to increased diastolic filling of the right ventricle and early aortic valve closure.

A delay in aortic valve closure causing P_2 to precede A_2 results in so-called reversed (paradoxic) splitting of S_2. The most common causes of reversed splitting of S_2 are left bundle branch block and delayed excitation of the left ventricle from a right ventricle ectopic beat. Mechanical prolongation of the left ventricular systole, resulting in reversed splitting of S_2, also may be caused by severe aortic outflow obstruction, a large aorta-to-pulmonary artery shunt, systolic hypertension, and coronary heart disease or cardiomyopathy with left ventricular failure.

Three-sound rhythms, caused by appearance of additional sounds

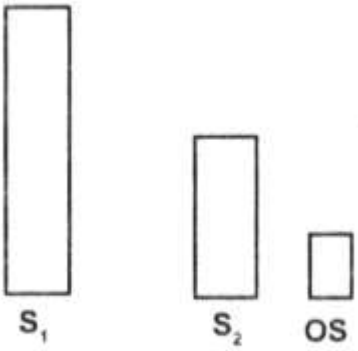


Fig. 4.28. Triple rhythm.

Triple rhythm is three-sound rhythm, which is heard at the heart apex in the patient with mitral stenosis (Fig. 4.28).

Triple rhythm consists of loud (snapping) S_1, normal S_2 and additional sound, which is heard 0.07–0.13 s following S_2, and termed OS (opening snap). The cusps of the normal mitral valve open noiseless; they are freely forced back by the blood flow ejected from the atria to the ventricle. In mitral stenosis blood thrusts against the sclerosed valve, cusps of which cannot freely move, to produce OS. The opening snap is a brief, high-pitched, early diastolic sound. This phenomenon is of considerable diagnostic value because it is heard only in the mitral stenosis.

Gallop rhythm.

Three-sound rhythm of a peculiar acoustic character, termed gallop rhythm (*bruit de galop* or *rythme de galop* according to Laubry and Pezzi), is also of considerable diagnostic value. The sounds of gallop rhythm are usually soft and low, resemble the galloping of a horse, and are best heard in direct auscultation. Gallop rhythm is heard as three separate audibly distinct sounds in approximately equal intervals.

Gallop rhythm is classified as presystolic (at the end of diastole), protodiastolic (at the beginning of diastole), and mesodiastolic (at the middle of the diastole) depend on the time of appearance of the extra sound in diastole.

Presystolic gallop rhythm occurs due to delayed atrioventricular conduction, when atrial systole is separated from the ventricular systole by a longer than normal period, and is heard as separate sound (Fig. 4.29).

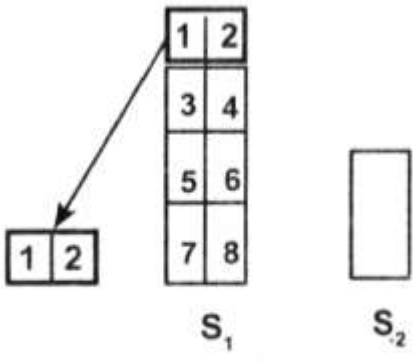


Fig. 4.29. Presystolic gallop rhythm.

Three-sound rhythm at the heart apex, in which S_1 is decreased, S_2 is normal, and the first sound is weakest – is presystolic gallop rhythm.

Presystolic gallop rhythm is heard in the patients with:

- Rheumocarditis;
- Cardiosclerosis;
- Essential hypertension;
- Chronic nephritis with arterial hypertension syndrome;
- Toxic and infectious affection of the myocardium.

Protodiastolic gallop rhythm is caused by appearance of pathological additional sound 0.12–0.02 s after S_2 as a result of considerably decreased tone of the ventricular myocardium (Fig. 4.30). Ventricles distended quickly during their filling with blood at the beginning of the diastole and the vibrations of myocardium thus generated are audible as an extra sound.

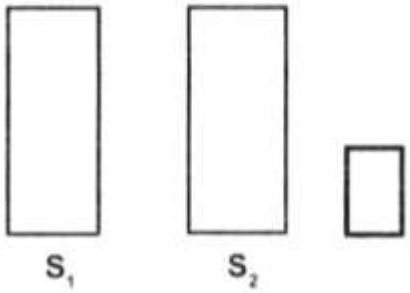


Fig. 4.30. Protodiastolic gallop rhythm.

Three-sound rhythm at the heart apex, in which S_1 is decreased, and the third sound is weakest – is protodiastolic gallop rhythm. This auscultation phenomenon is observed in the patients with:

- Acute and chronic myocarditis;
- Myocardiosclerosis;
- Heart failure;
- Toxicosis;
- Thyrotoxicosis;
- Anemias.

Mesodiastolic (summation) gallop rhythm arises in severe dystrophic affection of the myocardium in the patients with myocardial infarction, essential hypertension, heart valvular diseases, myocarditis and chronic nephritis. Mesodiastolic gallop rhythm is characterized by appearance of the additional sound in the middle of diastole caused by increase intensity of the S_3 and S_4, which are heard as one gallop sound.

Systolic clicks – auscultation phenomenon, which denote prolapse of one or both cusps of the mitral valve. They also may be caused by tricuspid valve prolapse. Auscultation symptomatic may be very different: systolic clicks may be single or multiple, they may occur at any time in systole with or without a late systolic murmur. Typical peculiarity – changes of the auscul-

tation data depend on position of the patient and exercise test. If the patient squat click and murmur slightly delayed; in the upright posture click and murmur are closer to S_1 (Fig. 4.31).

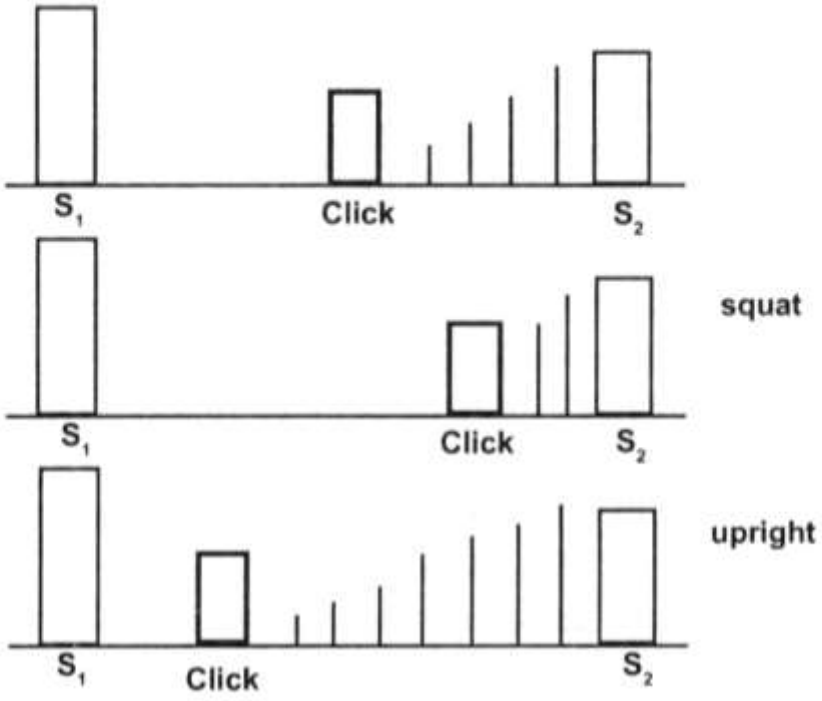


Fig. 4.31. Systolic clicks.

Pericardial knock – high-pitched sound occurs 0.01–0.06 s after S_2 in the patients with constrictive pericarditis due to vibration of the adherent pericardium in abrupt dilation of the ventricle at the beginning of diastole. Pericardial knock is better heard at the heart apex or medially toward to xiphoid.

Embryocardial or pendulum rhythm occurs in severe heart failure, attacks of paroxysmal tachycardia, high fever, etc. Tachycardia makes diastolic pause almost as short as the systolic one. A peculiar auscultative picture, in which heart sounds are similar in intensity, resembles foetal rhythm is termed embryocardia.

Cardiac murmurs

In addition to the normal heart sounds, abnormal sounds known as murmurs may be heard in auscultation. Cardiac murmurs may both endocardiac and exocardiac. *Endocardiac* murmurs occur in dysfunction of the intact valves – *functional murmurs* or in anatomical changes in the structure of the heart valves – *organic murmurs.*

Organic cardiac murmurs.

When a valve is stenotic or damaged, the abnormal turbulent flow of blood produces a murmur, which can be heard during the normally quiet times of systole or diastole.

The *mechanisms* of cardiac murmurs can be explained by the physics laws concerning the flow of liquids in tubes (Fig. 4.32).

Such condition as liquid flowing through a partially narrowed portion of the tube (Fig. 4.32.a) can cause turbulent flow. The intensity of noise depends on the extent of narrowing: the narrower lumen of the tube, the more intense noise. In significant narrowing of the tube, noise may weaken or even disappears. Liquid flowing from a smaller portion of the tube to a larger one can also cause vortex movement (Fig. 4.32b). Murmur can be caused by blood flow in the vascular lumen partially obstructed by atherosclerotic plaque or thrombus (Fig. 4.32.c).

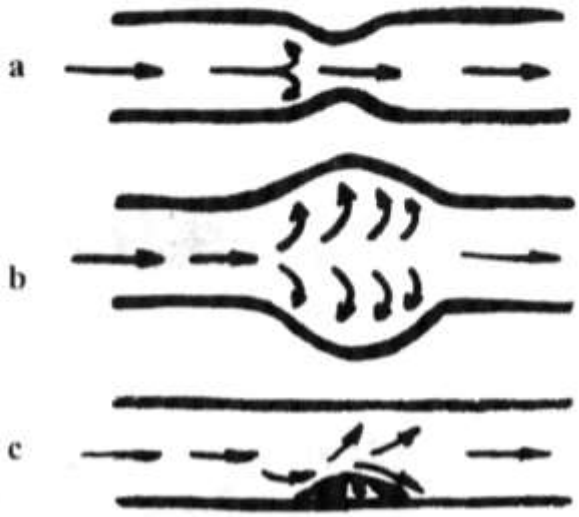


Fig. 4.32. Mechanisms of murmurs.

Following characteristics used to describe cardiac murmurs are timing, intensity, pitch, quality, configuration, duration, location and radiation.

Murmurs are defined in terms of their *timing* within the cardiac cycle. ***Systolic murmur*** terminates between S_1 and S_2 or begins instead of significantly decreased S_1. ***Diastolic murmur*** begins with or after S_2 and terminates at or before the subsequent S_1.

The *intensity* of the murmurs is graded according to the Levine scale:

- **Grade I** – Lowest intensity, difficult to hear even by expert listeners
- **Grade II** – Low intensity, but usually audible by all listeners
- **Grade III** – Medium intensity, easy to hear even be inexperienced listeners, but without a palpable thrill
- **Grade IV** – Medium intensity with a palpable thrill
- **Grade V** – Loud intensity with a palpable thrill. Audible even with the stethoscope placed on the chest with the edge of the diaphragm
- **Grade VI** – Loudest intensity with a palpable thrill. Audible even with stethoscope raised above the chest.

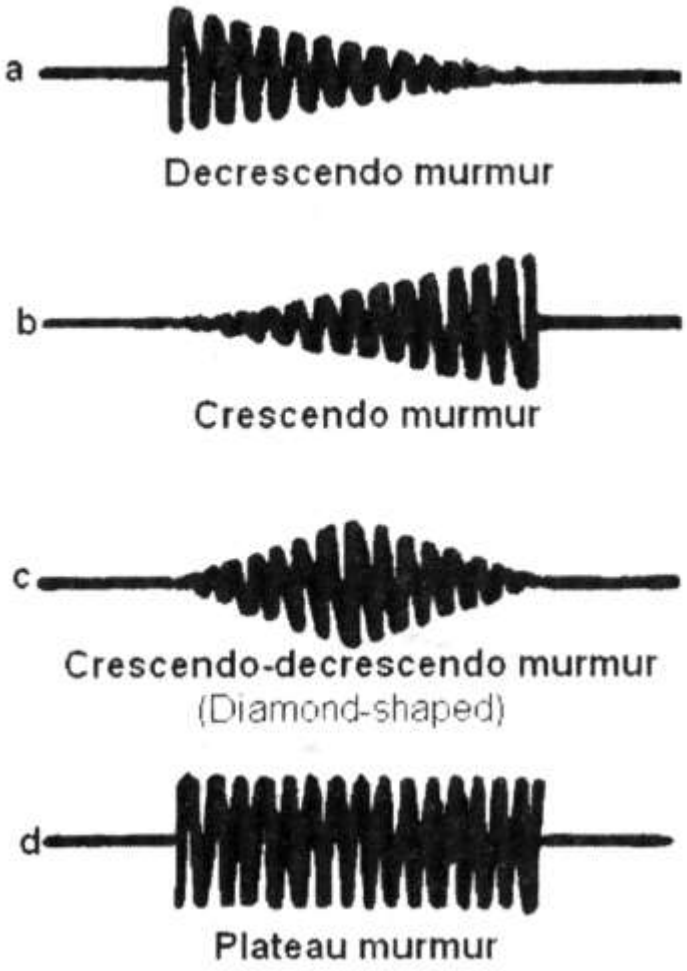


Fig. 4.33. Configuration of the cardiac murmurs.

A cardiac murmur's *pitch* varies from high to low.

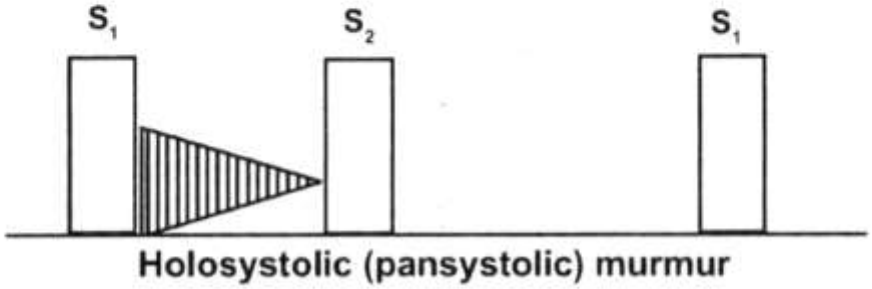
Common descriptive terms of a murmur's *quality* include rumbling, blowing, machinery, scratchy, harsh, rough, squeaky, or musical.

The configuration of murmur is defined by changes in their intensity during systole and diastole as recorded on a phonocardiogram (Fig. 4. 33).

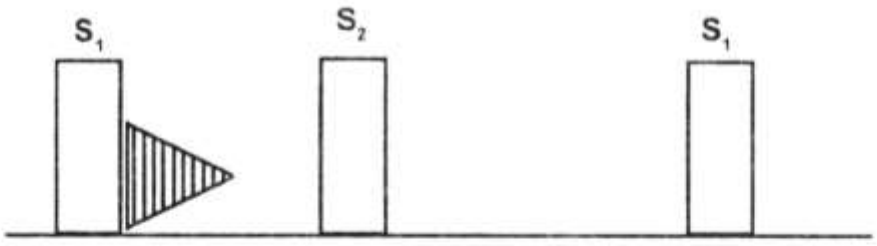
A decrescendo murmur gradually decreases in intensity (Fig. 4.33.a), a crescendo murmur gradually increases in intensity (Fig. 4.33.b), a crescendo-decrescendo murmur (a diamond-shaped) first increases in intensity, and then decreases in intensity (Fig. 4.33.c), and a plateau murmur is equal in intensity throughout the murmur (Fig. 4.33.d).

A murmur's *duration* can be of different length (Fig. 4.34).

SYSTOLIC HEART MURMURS

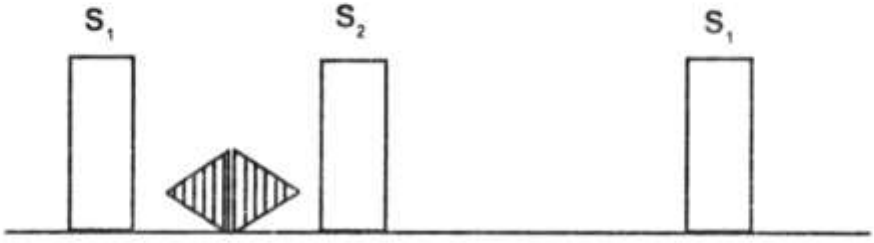


begin with S_1 and continue through all systole to S_2.



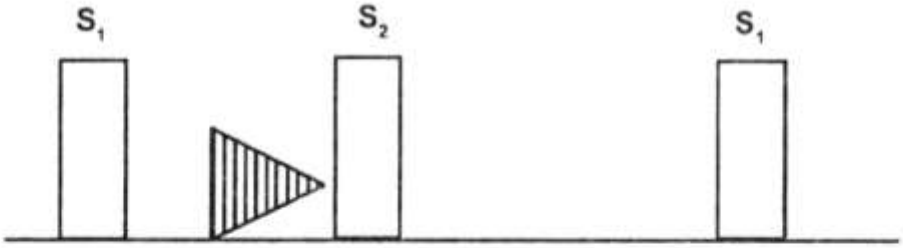
Early systolic murmur

begins with S_1 and extend for a variable period of time, ending well before S_2.



Midsystolic murmur

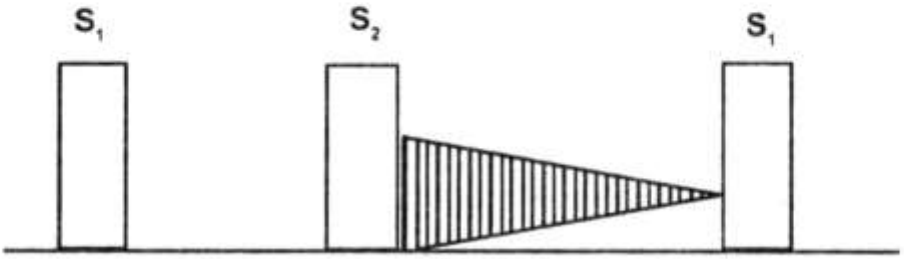
begins at a short interval following S_1, end before S_2, and are usually crescendo-decrescendo in configuration.



Late systolic murmur

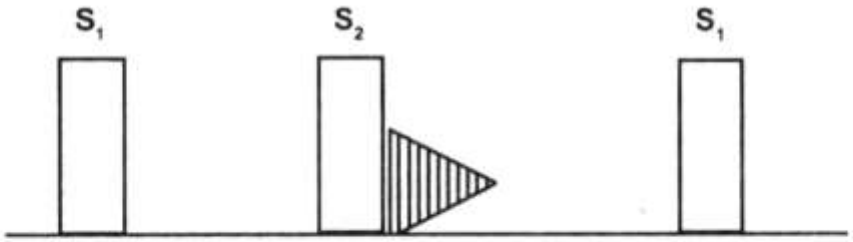
begins well after the onset of ejection that is at the end of systole.

DIASTOLIC HEART MURMURS



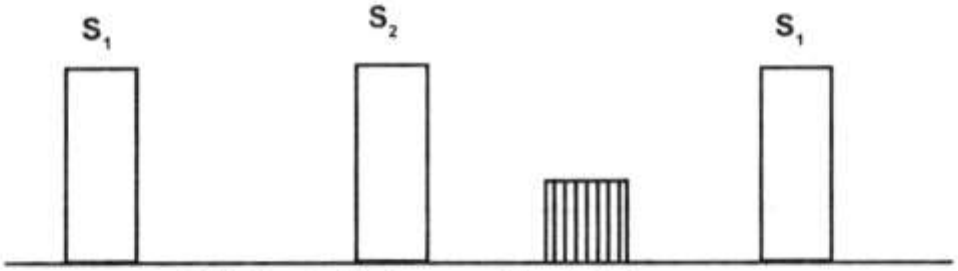
Holodiastolic (pandiastolic) murmur

begins after S_2 and continue through all diastole to S_1.



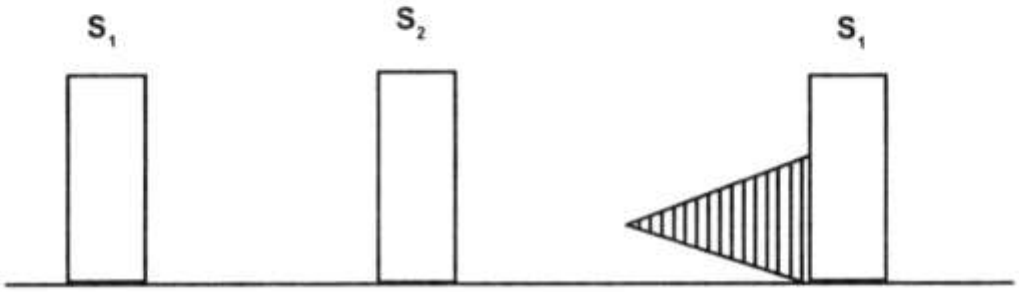
Early diastolic (protodiastolic) murmur

begins with S_2 and ends well before S_1,
usually decrescendo in configuration.



Middiastolic (mesodiastolic) murmur

begins at a short interval following S_2, end before S_1



Presystolic murmur

begins at the end of diastole, usually crescendo in configuration.

Fig. 4.34. Duration of a cardiac murmurs.

Location. Cardiac murmurs may not be not audible over all areas of the chest, and it is important to note where it is heard best and where it radiate to.

The location on the chest wall where the murmur is best heard and the areas to which it radiates can be helpful in identifying the cardiac structure from which the murmur originates (Tab. 4.39).

Tab. 4.39. ***Best auscultatory areas of a cardiac murmurs. Topographic classification of murmurs.***

Auscultatory areas	Murmur	Heart valvular disease
Heart apex	Systolic Diastolic	Mitral regurgitation Mitral stenosis
Second intercostal space at the right sternal edge	Systolic Diastolic	Aortic stenosis Aortic regurgitation
Second intercostal space at the left sternal edge	Systolic Diastolic	Pulmonary stenosis Pulmonary regurgitation
Base of the xiphoid	Systolic Diastolic	Tricuspid regurgitation Tricuspid stenosis

Tab. 4.40. ***Auscultatory areas and radiation of murmurs in heart valvular diseases.***

Heart valvular disease	Murmur	Auscultatory areas	Radiation areas
Mitral regurgitation	Systolic	Heart apex	Axillary region
Mitral stenosis	Diastolic	Heart apex	No radiation
Aortic regurgitation	Diastolic	Second intercostal space at the right sternal edge	Botkin-Erb's point, sometimes heart apex
Aortic stenosis	Systolic	Second intercostal space at the right sternal edge	Subclavian, carotid arteries, interscapular region

Radiation. Some cardiac murmurs may be heard not only in standard auscultatory areas but also transmitted in the direction of blood flow. This phenomenon is known as radiation (Fig. 4.35).

Murmurs radiate in either a forward (*ejection murmurs*) or backward direction (*regurgitation murmurs*).

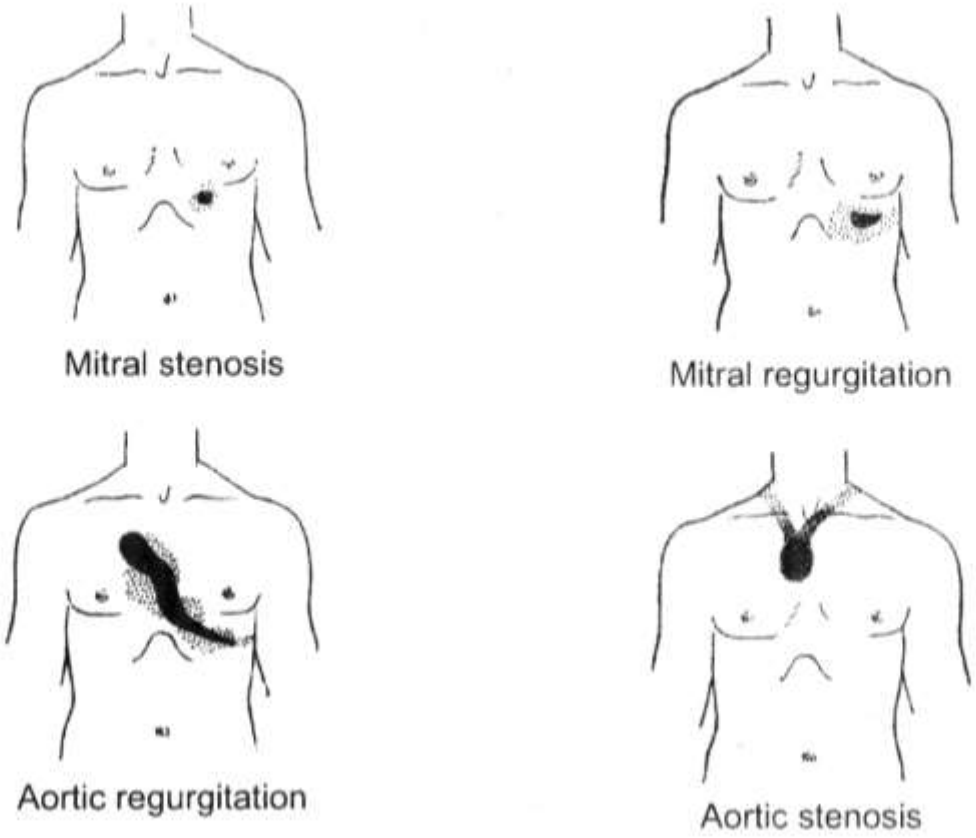


Fig. 4.35. Murmur's radiation in heart valvular diseases.

Systolic murmurs

Aortic stenosis. One of the most frequent pathologic systolic murmurs is due to aortic stenosis. The murmur of aortic stenosis heard best over "aortic area", second intercostal space along right sternal border, with radiation into the neck, along carotid arteries, into the interscapular region (ejection murmur). The intensity of murmur varies directly with the cardiac output. It has a harsh quality, are usually crescendo-decrescendo in configuration (as the velocity of ejection increases, the murmur gets stronger, and as ejection declines, its diminished), is typically midsystolic murmur (starts shortly after S1, when the left ventricular pressure becomes enough to open aortic valve; ends before left ventricular pressure falls enough to permit closure of the aortic leaflets).

Pulmonary stenosis. The murmur of pulmonary stenosis is heard best in the "pulmonic area", second intercostal space along the left sternal border. The murmur can be heard radiating into the neck or the back (ejection murmur), has a harsh quality, a crescendo-decrescendo shape, and midsystolic duration.

Mitral regurgitation. Systolic murmur in mitral regurgitation is best heard at the heart apex, with radiation into the left axilla (regurgitant murmur). The quality of murmur is usually described as blowing, frequency – as high-pitched, the configuration of murmur may vary considerably, and its duration is holosystolic.

Tricuspid regurgitation. The holosystolic murmur of tricuspid regurgitation is best heard at the base of the sternum, generally softer than that of mitral regurgitation, and frequently increases during inspiration.

Diastolic murmurs

Aortic regurgitation. The murmur of aortic regurgitation best heard in the second intercostal space along left sternal edge, it widely radiates along the left sternal border (Botkin-Erb's point) and to be well transmitted to the heart apex (regurgitant murmur). This murmur is usually characterized as blowing, generally high-pitched, decrescendo (since there is progressive decline in the volume of regurgitation during diastole), and early diastolic murmur. In severe regurgitation, it may be holodiastolic. The soft, rumbling, low-pitched, mid- to late diastolic murmur at the heart apex (Austin Flint murmur) may be detected in severe aortic regurgitation. It is thought to be due to a functional mitral stenosis, as the backflow blood from the aorta presses on the mitral valve, slightly occluding the flow from the left atrium.

Pulmonary regurgitation. The murmur of pulmonary regurgitration is best heard in the second intercostal space to the left of the sternum, with radiation along left sternal edge (regurgitant murmur), high-pitched, decrescendo, early diastolic murmur. The diastolic murmur of pulmonary regurgitation without pulmonary hypertension is softer, and low- to medium-pitched.

In mitral stenosis functional early diastolic, high-pitched, with a decrescendo quality murmur is heard over the pulmonic area. This murmur, known as *Graham Steel murmur*, begins with accentuated S_2, and is caused by dilation of the pulmonary artery due to significant pulmonary hypertension.

Mitral stenosis. The murmur of mitral stenosis is best heard at the heart apex with a little radiation. It is usually described as low-pitched, rum-

bling, characteristically follows OS, and can be heard best with the patient in the left lateral decubitus position. The murmur is nearly holodiastolic with presystolic accentuation, or presystolic crescendo, or early diastolic (protodiastolic) decrescendo.

Tricuspid stenosis. The diastolic murmur associated with tricuspid stenosis is localized to a relatively limited area over the xiphoid, low-pitched, rumbling, and like most right-sided events, may be stronger during inspiration.

Study of Arterial Pulse

Pulse [L, *pulsare,* to beat] is the regular, recurrent expansion and contraction of an artery produced by waves of pressure caused by the ejection of blood from the left ventricle of the heart as it contracts. The phenomenon is easily detected on superficial arteries, such as the radial, brachial, carotid, and femoral arteries, and corresponds to each beat of the heart.

Palpation of the pulse. Technique. Pulse is commonly studied on the radial artery between the styloid process of the radial bone and the tendon of the internal radial muscle.

The carotid pulse is best examined with the sternocleidomastoid muscle relaxed and with the head rotated slightly toward the examiner. Palpate the carotid pulse with the patient lying on a bed or couch. Never compress both carotids simultaneously. Use the left thumb for the right carotid and vise versa. Place the tip of the thumb between the larynx and the anterior border of the sternocleidomastoid muscle. Press the thumb gently backwards to feel the pulse. In palpating the brachial arterial pulse, the examiner can support the subject's relaxed elbow with the arm while compressing the brachial pulse with the thumb with the fingers cupped round the back of the elbow. Feel just medial to the tendon of the biceps muscle to detect the pulse.

The usual technique is to compress the artery with the fingers until the maximum pulse is sensed. Varying degrees of pressure should then be applied while concentrating on the separate phases of the pulse wave.

This method is used for assessing the following characteristics of the pulse: symmetry, rhythm, rate, correlation of the pulse and heart rate, tension, filling (volume), size, speed, and pulse waves shape (Tab. 4.41).

Tab. 4.41. ***Examination of the arterial pulse.***

Examination sequence	Factors that cause pulse characteristics	Norm	Pathology
Symmetry on the pair arteries	Degree of arteries filling	Symmetric	Asymmetrical *p. differens*
Rhythm	Cardiac activity	Rhythmic *p. regularis*	Arrhythmic *p. irregularis*
Rate (PR)	Heart rate (HR)	60–80 beats per minute (b.p.m.)	Frequent – *p. frequens,* Rare – *p. rarus*
Correlation between PR and HR	Contractile ability of the heart	HR = PR	HR>PR – pulse deficit *p. dificiens*
Tension	BP level in the greater circulation	Sufficient tension	Soft (BP low) –*p. mollis* Firm (BP high)–*p. durus*
Volume	Volume of circulating blood	Sufficient volume	Large volume -*p. plenus* Empty or low volume – *p. vacuus*
Size	Stroke volume, arteries filling	Moderate size	Large – *p. magnus* High – *p. altus* Small – *p. parvus* Thready – *p. filiformis*
Speed	Speed of pressure changes in the arteries	Moderate speed	Fast – *p. celer* Slow – *p. tardus*
Shape	Stroke volume, velocity of the BP changes during systole and diastole	Moderate size and velosity, uniform – *p. aequalis*	Fast (*p. celer)* and high (*p. altus)* Slow (*p. tardus*) and small (*p. parvus*) Nonuniform–*p. inaequalis* Variable – *p. alternans*

Normal findings. Pulse is symmetrical (pulsus differens is absent), rhythmic, pulse rate is between 60–80 beats per minute, pulse deficit is absent, pulse is of sufficient pressure and volume, of moderate speed and size.

Symmetry. The pulse should be taken in definite order. As the pulse may be different on different arms, you should first palpate simultaneously on both radial arteries. Apply four fingers over radial pulse at the wrists. Use the pulp of the fingers to assess the symmetry of the pulse. If the pulse waves are equal on both hands, you continue palpation on either hand. If the pulse waves are different, further study should be carried on that arm where the pulse waves are more pronounced.

The pulse waves on the both hands normally should be equal. If the pulse waves on the one hand are smaller or lag in time is said to be *pulsus differens* is present. It occurs in compression of corresponding artery (peripheral, brachial, or subclavian arteries) or by scar, tumor, enlarged lymph node, or by inflammatory infiltration. Pulsus differens may also be caused by compression of large arteries by aortic aneurism, mediastinal tumor, retrosternal goiter, or by markedly enlarged left atrium.

Rhythm. Pulse rhythm is reflection of the heart rhythm. The normal rhythm of the heart is called sinus rhythm because it originates from the sinoatrial node. Normally pulse is rhythmic or regular. When the pulse is irregular, it is important to identify the nature of the irregularity and to determine whether it is present all-time or only intermittently.

Sinus rhythm is seldom completely regular, because the heart speeds up during inspiration. This sinus arrhythmia is most obvious in children, young people and athletes.

An occasional irregularity is commonly caused by ectopic beats or extrasystoles, which can be atrial or ventricular in origin.

Atrial fibrillation causes a totally random heart rhythm leading to a pulse, which is irregular in both timing and volume. This is often described as an 'irregularly irregular' rhythm.

Arrhythmic pulse can also be caused by atrial flutter with variable response and atrioventricular block (AV block).

The most common causes of an irregular pulse are listed in Table 4.42.

Tab. 4.42. ***Causes of an irregular pulse.***
Pulsus irregularis.

- Sinus arrhythmia
- Atrial extrasystoles
- Ventricular extrasystole
- Atrial fibrillation
- Atrial flutter with variable response
- Atrioventricular block

Pulse rate. A normal resting pulse rate is from 60 to 80 beats per minute in an adult. If the pulse is regular, count the pulse for 15 seconds and multiply by four to obtain pulse rate in beats per minute. If the pulse is irregular, you should count the pulse rate not less than 1 minute.

In accelerated heart rate to more than 90 beats per minute – *tachycardia*, the pulse rate increases accordingly so-called – *p. tachus* or *p. frequens*.

Frequent pulse can occur in healthy persons in physical and emotional exertion, in coffee, strong tee, alcohol, hot food and drinks, pepper, garlic intake, and smoking.

Compensatory tachycardia arises in heart failure, hypotension, anemia, bleeding, collapse, traumatic and postoperative shock, and in neurosis. Tachycardia is also frequent symptom of myocarditis and heart defects.

In infectious diseases, in elevation of temperature at 1° pulse rate accelerates at 10 beats, except meningitis and typhoid fever. Some medication (sympathomimetics, vasodilators) can also cause acceleration of the pulse rate.

The causes of pulse rate acceleration are shown in Table 4.43.

Bradycardia is defined as heart rate of < 60 beats per minute, in such case *p. rarus* or *p. bradus* is found. An important cause of bradycardia is complete heart block (3 rd degree atriventricular block) when the heart rhythm is regular and the rate is often less than 40 b.p.m. This may be intermittent and cause the patient to lose consciousness (Adams-Stokes attacks). The most common causes of bradycardia are listed in Tab. 4.44.

Tab. 4.43. ***Causes of frequent pulse.***

Tachycardia, HR>90/min – ***pulsus freguens*** or ***pulsus tachus***	
Sinus tachycardia	Arrhythmia
Physiological Exercise Excitement/anxiety Coffee Strong tee Alcohol Smoking Pepper, garlic During inspiration **Compensatory** Heart failure Myocarditis Heart defects Hypotension Anemia Bleeding Collapse, shock Neurosis Infections, intoxication Fever Tuberculosis Hyperthyroidism Tumor **Medication** Sympathomimetics Vasodilators	Atrial fibrillation Atrial flutter Supraventricular tachycardia Ventricular tachycardia

Tab. 4.44. ***Causes of rare pulse.***

Bradycardia, HR<60/min – ***pulsus rarus*** or ***pulsus bradus***	
Sinus bradycardia	Arrhythmia
Physiological Congenital bradycardia Sleep Lying posture After food intake During expiration Athletic training **Increased vagus tone** Jaundice Meningitis Cerebral tumor, edema Cerebral hemorrhage Hypertension Hypothyroidism **By reflex in irritation of the peritoneum** Appendicitis Cholecystitis Peptic ulcer disease **Medication** Beta-blockers Digoxin Verapamil, diltiazem	Sick sinus syndrome Second degree AV block Third degree AV block

Pulse deficit is difference between the actual heart rate and the rate palpable at the radial pulse. You should calculate heart rate, for example 85 beats per minute, then pulse rate – 75 beats per minute. 85–75=10. Pulse deficit is 10 beats per minute.

The most common causes of pulse deficit are extrasystoles and atrial fibrillation. Frequently the pulse waves produced by ectopic beat are too weak to be felt at the wrist. In atrial fibrillation, the delay between some ventricular contractions may too short to allow proper filling, leading to a reduced stroke volume and an impalpable pulse. This may produce a considerable pulse deficit.

Pulse tension is defined as the force that should be applied to compress pulsating artery completely. Pulse tension depends on blood pressure level. If blood pressure level is normal, a moderate force is applied to compress the radial artery, and pulse is said to be of moderate tension.

According to the pulse tension you can estimate indirectly BP level: in high BP the pulse is firm – *p. durus*, in low BP the pulse is soft – *p.mollis*.

Pulse volume depends on the arteries filling by blood. The full pulse – *p. plenus*, and empty pulse – *p. vacuus* are differentiated.

The full pulse *(p. plenus)* is found in the normal stroke volume, when the arteries are sufficiently filled with blood.

A large volume pulse may be due to aortic regurgitation or vasodilatation. Exercise, emotion, heat and pregnancy are physiological factors causing vasodilatation, which is also, occurs with fever, thyrotoxicosis, anemia, or drug therapy.

In decreased pulse volume, the pulse is said to be empty – *p. vacuus*. The low volume pulse is found in patients with decreased circulating blood volume (bleeding, vomiting, diarrhea, profuse sweating), re-distribution of blood in circulatory system – collapse, and in patients with reduced stroke volume due to heart failure or peripheral vascular diseases.

Pulse size implies its tension and volume. The pulse of large volume and tension is defined as large pulse – *p. magnus*. The pulse of small volume and tension is called small pulse – *p.parvus*. Considerably weak pulse is called thready or *p. filiformis*. It found in shock, acute heat failure, and profuse bleeding.

Pulse wave form. The vascular wall pulsation is recorded as a curve – sphygmogram. Normal sphygmogram has a sharp upstroke – the *anacrotic wave*, a peak of the curve, *dicrotic wave* (repulsion of the blood from the closed aortic valve during early diastole), and a gradually declining upstroke – the *catacrotic wave* (Fig. 4.36).

Fig. 4.36. Normal sphygmogram.
1 – anacrotic wave, 2 – discrotic wave, 3 – catacrotic wave

In aortic regurgitation *p. altus, p. celer et p. saliens* is found (Fig. 4.37). The pulse wave upstroke rises rapidly and forcefully due to increased stroke volume and systolic pressure, while during diastole rapid decline in pressure follows due to regurgitation of the blood to the left ventricle. This pulse is less characteristic of thyrotoxicosis, anemia, and excitement.

Fig. 4.37. Shygmogram in aortic regurgitation – *p. altus, p. celer et p. saliens.*

In aortic stenosis *p. rarus*, *p. tardus* et *p. parvus* is found due to slow rise and decline in pressure in the arterial system (Fig. 4.38).

Fig. 4.38. Shygmogram in aortic stenosis – *p. rarus, p. tardus* et *p. parvus.*

P. bisferiens, which has two systolic peaks separated by a distinct mid-systolic dip, is characteristic of combined aortic stenosis and regurgitation, and of hypertrophic cardiomyopathy (Fig. 4.39).

Fig. 4.39. Pulsus bisferiens.

P. altenrans is rare and describes alternate variations in the size of the pulse waves (Fig.4.40). The pulse rhythm is regular. It is a sign of severe myocardial disease and heart failure.

Fig. 4.40. Pulsus alternans.

P. dicroticus – describes as pulse with dicrotic wave appearance in palpation. In most normal persons a dicrotic wave is not palpable. The dicrotic pulse has two palpable waves, one in systole and one in diastole (Fig.4.41). It occurs most frequently in patients with a very low stroke volume, particularly in those with dilated cardiomyopathy.

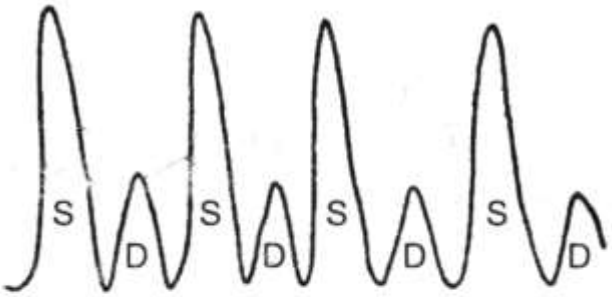


Fig. 4.41. Pulsus dicroticus.

Pulsus paradoxus describes a pulse that increases in volume on expiration and decreases in volume in inspiration. It occurs with severe airways obstruction and pericardial tamponade, due to a tense pericardial effusion.

Simultaneous palpation of the radial and femoral arterial pulses, which normally are virtually coincident, is important to rule out *aortic coarctation*. Coarctation is congenital narrowing of the aorta usually situated just after the left subclavian artery. In children with this condition the upper limb pulses are normal, while all lower limb pulses are reduced or impalpable. In adults, coarctation usually presents with hypertension, and although the femoral pulses are usually palpable owing to the development of collaterals they are diminished in volume and delayed with respect to the radial pulse.

Study of Venous Pulse

Examination of the internal jugular vein provides valuable information regarding the right atrial pressure and the character of the venous waveform.

There are no valves between the right atrium and the internal jugular vein. It follows that the degree of distension in the vein equates to the

pressure in the right atrium and the venous waveform provides information about cardiac function.

The internal jugular vein enters the neck behind the mastoid process. It runs deep to the sternomastoid muscle before entering the thorax between its sternal and clavicular heads. Because of its deep position the internal jugular vein can only be examined when the neck muscles are relaxed. Only a diffuse pulsation can be seen and the vein is not visible. The external jugular vein is visible but it is not routinely examined because it is prone to kinking and partial obstruction as it traverses the deep fascia of the neck.

Jugular venous pressure. The normal mean right atrial pressure is < 7 mmHg. Since the sternal angle is approximately 5 cm above the right atrium, the normal jugular venous pulse should extend no more than 4 cm above sternal angle. When a normal subject sits upright the pulse is hidden behind the clavicle and sternum. When reclining at 45 degrees, the top of the pulsation is normally just at the level of the clavicle. If it cannot be seen, then a normal right atrial pressure can be confirmed by applying firm pressure over the center of the abdomen for 5–10 seconds (abdominojugular reflux). This increases the venous return and normally leads to a transient increase in the right atrial pressure of 1–3 cm, which is reflected in the height of the jugular venous pulse.

Phlebography is the recording of the venous pulse. The normal jugular venous pulse ***waveform*** comprises two peaks, which helps to distinguish the vein from the carotid artery (Tab. 4.45).

Tab. 4.45. ***Differences between carotid and jugular pulsation.***

Carotid	Jugular
• Rapid outward movement	• Rapid inward movement
• One peak per heart beat	• Two peaks per heartbeat (in sinus rhythm)
• Palpable	• Impalpable
• Pulsation unaffected by pressure at the root of the neck	• Pulsation diminished by pressure at the root of the neck
• Independent of respiration	• Height of pulsation varies with respiration
• Independent of position	• Varies with position of patient
• Independent of abdominal pressure	• Rises with abdominal pressure

The first, or 'a' wave coincides with right atrial contraction; the venae cavae are overfilled due to delayed their emptying. The second positive wave, 'c' wave, comprises a transmitted pulsation from the carotid artery, and associated with ventricular systole. Next negative wave, 'x' wave, is due to atrial relaxation. The 'v' wave is caused by atrial filling during ventricular systole, when tricuspid valve is closed. Wave 'v' is followed by wave 'y', which begins with opening of tricuspid valve and right ventricular filling (Fig. 4.42).

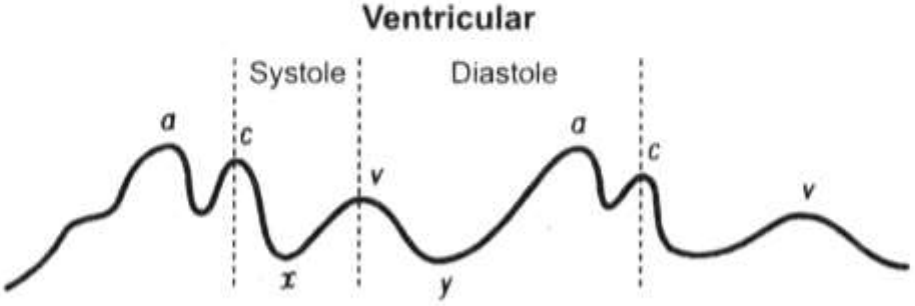


Fig. 4.42. Normal phlebogram.

Normally the jugular venous pressure falls on inspiration because fall in intrathoracic pressure is transmitted to the right atrium.

Examination sequence

- Position of the patient reclining supine at 45 degrees in good light.
- Ensure that the neck muscles are relaxed by resting the back of the head on a pillow.
- Inspect by looking across the neck from the side of the patient.
- Identify the internal jugular pulsation, if necessary, by means of abdominojugular reflux.
- Measure the vertical height in centimeters between the top of the venous pulsation and the sternal angle to give the venous pressure.
- If necessary, readjust the position of the patient until waveform is clearly visible.
- Now identify the pattern of pulsation and note any abnormalities.

In simultaneous phlebogram and sphygmogram recording, the maximal peak of sphygmogram corresponds to negative 'x' wave of phlebogram, because atrial filling begins during ventricular systole and blood ejection into arterial system. Therefore, normal venous pulse is also called *negative venous pulse*.

Raised jugular venous pressure. Heart failure is the commonest cause of a raised jugular venous pressure (Tab. 4.46). In major pulmonary embolism the jugular venous pressure may be so elevated that it is missed in a semirecumbent position. In pericardial constriction the jugular venous pressure is elevated and there is a characteristic paradoxical rise on inspiration (Kussmaul's sign). Serial estimation of the jugular venous pressure give valuable information regarding response to treatment: a reduction in the pressure as result of effective diuretic therapy in the patients with heart failure.

The waves of phlebogram can increase, decrease or disappears in pathology. Abnormalities of venous pulsation also given in Table 4.46.

Prominent 'a' waves are seen in any condition that restricts blood flow from the right atrium to the right ventricle: pulmonary hypertension and, rarely, tricuspid stenosis (Fig. 4.43).

Tab. 4.46. ***Abnormalities of the jugular venous pulse***

Condition	Abnormalities
Heart failure	Elevation, sustained abdominojugular reflux
Pulmonary embolism	Elevation
Pericardial effusion	Elevation, prominent 'y' descent
Pericardial constriction	Elevation, Kussmaul's sign
Superior vena caval obstruction	Elevation, loss of pulsation
Atrial fibrillation	Absent 'a' wave
Tricuspid stenosis	Giant 'a' wave
Tricuspid regurgitation	Giant 'v' wave
Complete heart (AV) block	'Cannon' waves

A prominent 'v' wave is characteristic of tricuspid regurgitation (Fig. 4.44). When severe this may be associated with pulsation of the liver.

Fig. 4.43. Phlebogram in tricuspid stenosis.

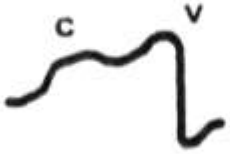


Fig. 4.44. Phlebogram in tricuspid regurgitation.

In atrial fibrillation there is no atrial systole. This result in the loss of the 'a' wave and the characteristic double impulse (Fig. 4 45).

Cannon waves are giant 'a' waves occurring when the right atrium contracts against a closed tricuspid valve (Fig. 4.46). They are seen as impulses shooting up the neck. Cannon waves occur with complete heart block and in both ventricular and supraventricular tachycardias.

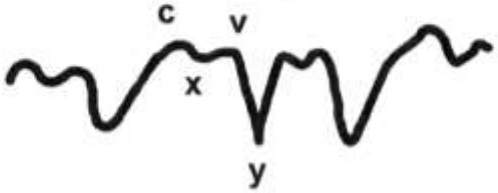


Fig. 4.45. Phlebogram in atrial fibrillation.

Fig. 4.46. Phlebogram in complete AV block.

Blood Pressure Measurement

Blood pressure (BP) is a hemodynamic variable dependent on cardiac output and total peripheral resistance.

Blood pressure is generally measured by the indirect method, using a mercury sphygmomanometer. N. Korotkoff proposed this method in 1905. In 1953 World Health Organization (WHO) introduced the Korotkoff's method as a classical world standard for measure BP.

Semi-automatic and automatic devices for blood pressure measurement at home and for prolonged (24 h and longer) ambulatory blood pressure monitoring are now available. It should be stressed that all of these devices should be tested for accuracy and reliability against standard methods according to strict protocol. Ambulatory blood pressure monitoring is an interesting research technique, which is used to investigate blood pressure variability, behavioral influences on arterial pressure and the time-course of the effects on antihypertensive therapy. It is also used, as are home blood pressure readings, to provide a supplementary source of information for diagnostic and therapeutic decisions.

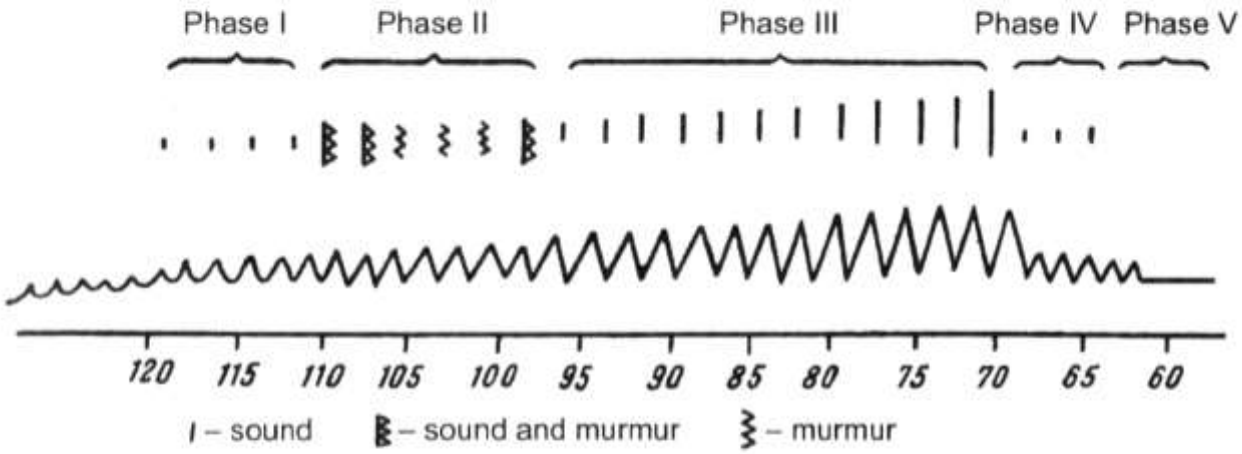
Attention to detail is needed for accurate blood pressure measurement. It is important to use the correct size of cuff. A cuff for adults must have a bladder 13–15 cm wide and 30–35 cm long so as to encircle the average arm. Larger cuff are needed for fat arms and smaller cuffs for children.

Examination sequence

- Before measurement commences the patient should be seated for several minutes in a quiet room.

- The chair should provide comfortable back support.
- The arm muscles should be relaxed.
- Support the arm comfortably with cubital fossa at about heart level (fourth intercostal space).
- Apply a cuff of suitable size to the exposed upper arm. Care should be taken to avoid tight sleeves.
- Identify brachial pulse.
- Inflate the cuff rapidly until the manometer reading is about 30 mmHg above the level at which pulse disappears.
- Deflate the cuff slowly at approximately 2 mmHg until regular heart sounds (called Korotkoff sounds) can be just heard (Fig. 4.47). This is the systolic blood pressure (SBP).
- Continue to deflate the cuff slowly until the sounds disappear – diastolic blood pressure (DBP).

Blood pressure may also be measured with the subject supine and standing, and in each position the arm should be supported at the heart level.



Phase I	The pressure at which the sounds are first heard is SBP
Phase II	The sounds and murmur are heard
Phase III	Loud sounds increased in intensity are heard
Phase IV	Intensity of the sounds decreased sharply
Phase V	The pressure at which the sounds disappear is DBP

Fig. 4.47. Phases of Korotkoff sounds.

Normal findings

The pressure at which the sounds are first heard is the *systolic blood pressure* (phase I).

The pressure at which the sounds disappear is *diastolic blood pressure* (phase V). Phase V may not be heard (the sounds not disappears), the phase IV (muffling of sounds) is taken as diastolic blood pressure in these cases.

The difference between SBP and DBP is defined as *pulse pressure*. Normally pulse pressure is 40–50 mmHg.

Blood pressure varies with excitement, stress and environment. Repeated measurements are required before a patient should be identified as hypertensive. SBP and DBP should be measured at least twice over a period of no less than 3 min; both should be recorded and the mean value for both should be used. It is also recommended that, on the first visit, the blood pressure should be measured on both arms. Measurement with the subject in the standing position should also be performed when postural hypotension is suspected and in the elderly, in whom this condition may be more common.

In some patient blood pressure is elevated in the presence of a doctor but falls when the subject leaves the medical environment – so-called 'white-coat hypertension' or 'effect'. Measurement by trained non-medical staff may reduce white-coat effect. Ambulatory blood pressure monitoring helps to distinguished these patients from those with true sustained hypertension.

Common abnormalities

Hypertension

Hypertension is an increase in total peripheral resistance due to arterioral vasoconstriction and wall thickening, leading to raised systemic pressure. According to World Health Organization and International Society of Hypertension (WHO/ISH) definition in 1999 hypertension exists when in an adults SBP is 140 mmHg and more, and DBP is 90 mmHg and more. Two classifications of hypertension by blood pressure level (Tab. 4.47) and by extent of organ damage (Tab. 4.48) were published by WHO/ISH.

Tab. 4.47. ***Classification of hypertension by blood pressure level (WHO/ISH, 2003).***

	SBP (mmHg)	**DBP** (mmHg)
Optimal	< 120	< 80
Normotension	< 130	< 85
High normal	130–139	85–89
Hypertension:		
I degree (mild hypertension)	140–159	90–99
II degree (moderate hypertension)	160–179	100–109
III degree (severe hypertension)	≥ 180	≥110
Isolated systolic hypertension	≥ 140	< 90

Tab. 4.48. ***Classification of hypertension by extent of organ damage.***

Stage I	*No objective signs of organic changes*
Stage II	*At least one of the following signs of organ involvement without symptoms or dysfunction* Left ventricular hypertrophy (radiogram, electrocardiogram, echocardiogram) Generalized and focal narrowing of the retinal arteries Proteinuria and/or slight elevation of plasma creatinine concentration (1.2–2.0 mg/dl or to 177 mmol/l Ultrasound or radiological evidence of atherosclerotic plaque (carotid arteries, aorta, iliac and femoral arteries)
Stage III	*Both symptoms and signs have appeared as result of organ damage*. These include: Heart Myocardial infarction Heart failure Brain Stroke Transient ischaemic attack

	Hypertensive encephalopathy Vascular demension Optic fundi Retinal hemorrhages and exudates with or without papilloedema Kidney Plasma creatinine concentration > 2.0 mg/dl or > 177 mmol/l Vessels Dissecting aneurysm Symptomatic arterial occlusive disease

Hypertension may be essential or primary and secondary. The causes of secondary hypertension are outlined in Table 4.49.

Tab. 4.49. ***Clasification of secondary hypertension.***

I. Renal
1. Damage of renal parenchyma and tissue: • glomerulonephritis (acute and chronic) • pyelonephritis (chrronic) • diabetic glomerulosclerosis • nephrolithiasis • polycystic kidney disease • hypo- or dysplasia of kidney • renal tuberculosis • renal tumor • renin-secreting renal tumor • renal transplantation • chronic renal failure • rheumatoid arthritis • systemic lupus erythematosus • scleroderma 2. Renovascular (vasorenal): • fibromuscular dysplasia and hypertrophy • aortitis

• atherosclerotic damage of renal arteries • congenital renal artery disease
II. Hemodynamic
1. Coarctation of the aorta 2. Hypertension in aortic atherosclerosis 3. Arterio-venous fistula (arterial shunt), various congenital defects 4. Hyperkinetic syndrome (aortic regurgitation, congestive heart failure) 5. Complete AV block 6. Cor pulmonale
III. Endocrine
1. Phaeochromocytoma 2. Primary aldosteronism 3. Cushing's symdrome
4. Hyperthyroidism 5. Hyperparathyroidism 6. Acromegaly 7. Adrenogenital syndrome 8. Primary renin hyperproduction 9. Endotelin-secreting tumors
IV. Neurogenic
1. Cerebral tumor 2. Cysts after stroke 3. Meningitis, traumatic encephalitis, diencephal syndrome 4. Carbon dioxide retention (pulmonary emphysema, bronchial asthma, pneumosclerosis, sleep apnoe syndrome)
V. Blood system disease
1. Polycythemia rubra vera
VI. Exogenic
1. Fluorine and chlorine poisoning 2. Drug-induced problems: estrogens, cyclosporin, oral corticosteroids (systemic or topical), oral contraceptives, narcotics.

Hypotension

Arterial hypotension is defined as SBP < 100 mmHg and DBP < 60 mmHg. Hypotension can be physiological and pathological.

Physiological hypotension is caused commonly by constitutional and inherited factors. It occurs in asthenic persons, in athletes, and sometimes in healthy persons during usual exertion and is not accompanied by any complaints and pathological changes in the organism. Orthostatic hypotension (drop of BP in upright position) is typical for the subjects with constitutional hypotension. This BP liability can cause unconsciousness states in changes of body position, especially in the morning in getting out of bed, or in long standing.

Pathological hypotension can be the result of circulatory failure (of cardiac or peripheral origin) and endocrine pathology.

Hypotension due to altered circulation is divided into three forms:

1. *Cardiogenic hypotension* (myocardial infarction, heart failure, aortic stenosis, pulmonary vessels embolism);
2. *Hypotension due to dilation of peripheral vessels* (infections: pneumonia, peritonitis, septic processes; intoxication: antihypertensive drugs, barbiturates; anaphylactic reactions; hyper reflex mechanism: psychogenic hypotension);
3. *Hypotension caused by decreased blood volume* (dehydration, hemorrhage that leads to collapse, shock).

Arterial hypotension is found in such endocrine pathology as Addison's disease, and hypothyroidism.

According to course acute and chronic arterial hypotension is differentiated. *Acute* arterial hypotension (collapse, shock) occurs as a result of acute blood loss, poisoning by medicines and industrial toxins, in emotional stress (vasovagal syncope), sudden redistribution of the circulating blood volume (in rapid evacuation of ascitic fluid in paracentesis, or pleural fluid in thoracentesis). Acute vascular failure quite often complicates various infectious diseases: pneumonia, influenza, typhus, dysentery, etc.). Acute hypotension can be of reflex or pain origin (syncope in injections, collapse or shock in myocardial infarction), or in intense physical exertion.

Chronic pathological hypotension is divided into primary or essential (increased parasympathetic nervous system activity, dysfunction of vegeta-

tive centers of vasomotor regulation that lead to decreased peripheral vascular resistance) and secondary or symptomatic (professional allergy, chronic intoxications).

Instrumental Methods

Clinical electrocardiography

Basic Physiological Principles

Electrocardiography (ECG) is a simple, useful, and practical diagnostic test. The ECG should be interpreted with knowledge of the entire clinical picture and must never be the sole basis for judging a patient's cardiac status.

Abnormalities of cardiac function and structure can occur without changes of the ECG. Similarly, ECG changes may occur without structural and functional abnormality of the heart. Many noncardiac factors alter the appearance of the ECG. Some of these factors are: skin resistance, the heterogeneity of tissue conduction, polarization, thickness of the chest wall, distance of the chest wall from the heart, position of the heart within thoracic cage, skeletal muscle tremors, electrical interference, technical problems concerned with a proper method of taking ECGs (particularly incorrect standardization), and improper functioning of the ECG instrument. Drugs, infections, pulmonary emboli, pain, fear, exercise, shock, and blood electrolytes alter the ECG. Thus, it is important to remember that the electrical forces of the heart that are recorded are influenced by various extrinsic factors.

To understand electrocardiography, one must know certain electrophysiologic principles including automaticity, conduction, refractoriness, depolarization, repolarization, and reentry.

The primary function of the heart is to contract so that it can serve its role as a pump. Each contraction is preceded by an electrical stimulus. When a patient has a regular sinus rhythm, the rhythm is initiated by the discharge of the sinus node. This electrical impulse is conducted through the right atrium, into the left atrium and into the ventricles through specialized conduction tissue. These specialized conduction pathways allow the heart to be electrically activated in a predictable manner (Fig. 4.48).

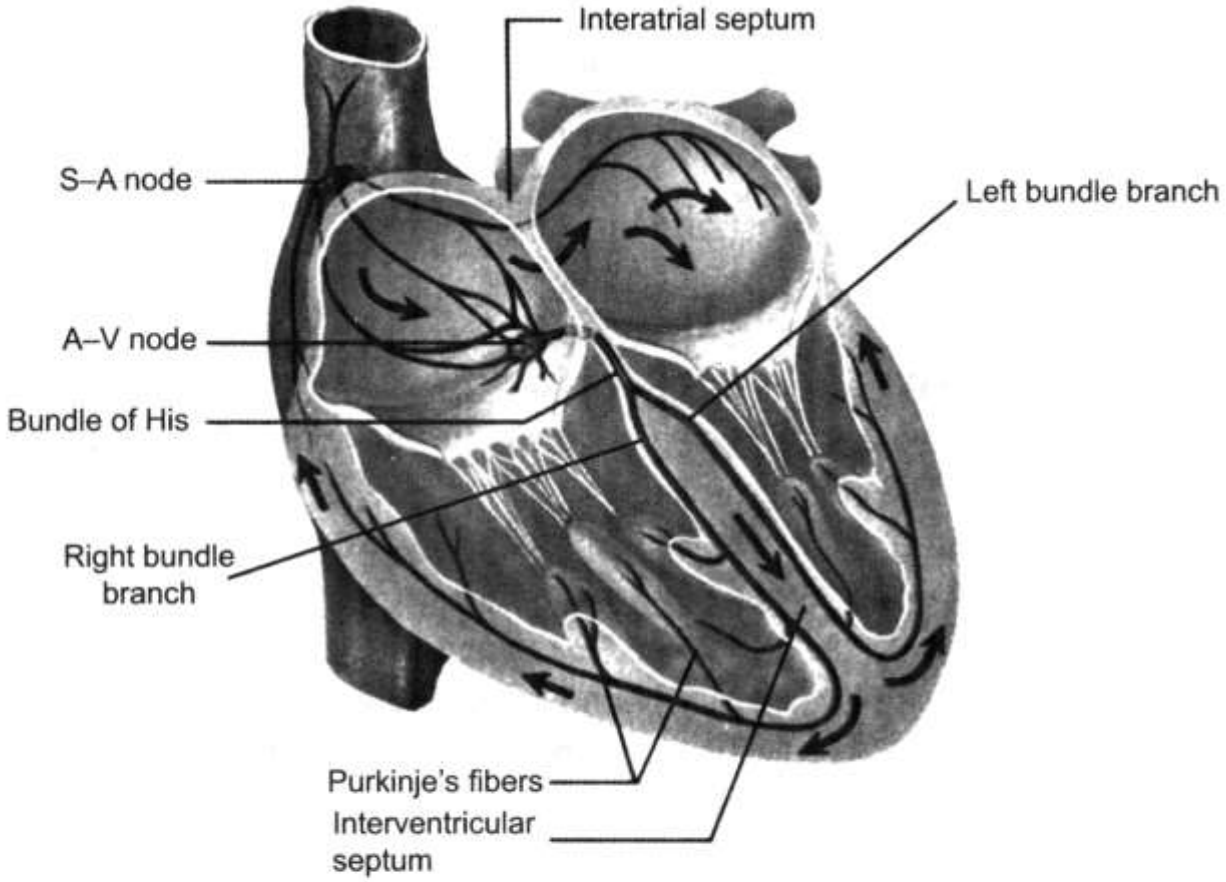


Fig. 4.48. Schematic representation of distribution of the specialized conductive tissues in the atria and ventricles.

Cardiac Conduction System

The **sinoatrial (SA)** node, preferably termed the sinus node, is the primary pacemaker of the heart. In adult it is approximately 10 to 20 mm long and is elliptical, and lying in the right side of the right atrium near the root of the superior vena cava.

The impulse from the sinus node passes to the atrioventricular (AV) node by special pathways There are three well-described internodal pathways: anterior that divide into two branches, one of which goes to the left atrium as Bachmann's bundle, middle internodal tract (Wenckebach's bundle), and posterior (Torel's bundle) tract. The anterior and middle pathways are appreciably shorter than posterior.

Other electrical pathways may join the atria and ventricles. These are the Kent's bundle, the James' bundle, and paraspecific septal fibers of Ma-

haim. These are of clinical importance because they form either the reentrant pathway or primary pathway for paroxysmal reentrant arrhythmias.

Bundles of Kent – Right and left accessory pathways, which directly connect the right and left atria to their respective ventricles (preexcitation).

James Tracts. – These pathways connect the atria with the lower AV node or bundle of His.

Mahaim Fibers. – Other anomalous pathways connecting the atria and ventricles are the paraspecific septal fibers of Mahaim, providing direct connections between the lower AV node, the His' bundle, and bundle branches with the ventricular septum.

The **AV node** is smaller than the sinus nodes, and measuring approximately 3 mm by 6 mm. It is an elliptical structure located in the endocardial surface of the right side of the interatrial septum. The AV node is divided into three electrophysiologic parts according to the action potentials and the response to electrical and chemical stimulation demonstrated by the cells in the three regions: 1 – the upper junctional area (AN region), (2) the middle nodal area (N region) and (3) the lower junctional area (NH region).

Common Bundle of His. – The bundle of His is an extension of the tail of the AV node. It is approximately 2 cm long in adult, but its size varies considerably. It is located under the endocardial surface of the right side of the interatrial septum and extends downward and leftward to enter the interventricular septum.

The bundle of His divides into right and left branches. ***The right bundle*** extends down the right side of the ventricular septum, until it reaches the apex of the right ventricle. There it divides into the small arborizations of the Purkinje system that spread over the endocardial surface of the right ventricular wall and right ventricular surface of the interventricular septum. The right bundle is close to the endocardial surface along two thirds of its length, and is therefore vulnerable to slight intracavitary pressure change. Increases in right ventricular intracavitary pressure that occur in acute cor pulmonale, stretching of the myocardium in heart failure or mechanical pressure on the septum occurring with right heart catheterization may cause right bundle-branch block.

The main trunk of ***the left bundle branch*** is narrow in its origin. It widens prior to branching into the *anterior* (superior) division and the *pos-*

terior (inferior) division. The anterior division is thin and long, lying below the aortic valve in the left ventricular outflow tract. The posterior division is must thicker and shorter, lying mostly in the left ventricular inflow tract. Each division terminates in the respective papillary muscles of the mitral valve. The divisions are now termed fascicles. Each major fascicle is responsible for activation of a portion of the septum and its respective portion of the ventricular Purkinje's network.

Thus, cardiac conduction system consists of the sinoatrial (SA) or sinus node, internodal tracts (anterior, middle, posterior), atrioventricular (AV) node, bundle of His (the right bundle, and the left bundle).

Cardiac automaticity function

Automaticity is the heart ability to discharge electrical impulse without external stimulus, and is characteristic of the entire conduction system. Contractile myocardium has no automaticity function.

The sinus node has the highest automaticity and is therefore the pacemaker of the cardiac rhythm. In the adult the usual resting discharge rate of sinus is 60 to 100 impulses per minutes. The impulses from the SA node are transmitted by internodal tracts to the AV node at a rate of 0.8–1 m/s. This rate sharply decreases in the region of the AV node to 0.05 m/s, and the atrial systole therefore ends earlier than excitation spreads over onto the myocardium of the ventricles to cause their contraction. Impulse passes from AV node through the bundle of His at a higher rate (1–1.5 m/s), while the rate of impulse transmission in Purkinje's fibers is as high as 3–4 m/s. Excitation is the triggering mechanism for the heart contraction. During the heart contraction, and immediately after systole, the cardiac muscle is absolutely refractory; then its excitability gradually restores.

Automaticity is characteristic of the entire conduction system of the heart, but in normal conditions it is inhibited by the high activity of the SA node, which is the automaticity center of the first order. If SA node is affected, or transmission of excitation to AV node is impaired, the zone of transition of AV node to the common bundle of His becomes the pacemaker (the second-order automaticity center). Impulses are generated here at a rate of 40 to 50 per minute. Lower part of the bundle of His and its branches, and Purkinje fibers are the automaticity center of the third order, but the rate of the cardiac rhythm then slow down to 20–30 per minute.

Cardiac conductivity function

Conductivity is a property of the myocardium that allows the transmission of electrical impulse from one cell to another, and is characteristic and of specialized conduction system, and of contractile myocardium. But by muscle cell-to-cell conduction is significantly slower than by specializes conduction pathways.

The sinus node impulse reached the AV node via the special internodal tracts, chiefly the anterior and middle nodal tracts, and later via the posterior internodal tract, which takes a longer route. Most impulses reach the left atrium via Bachmann's bundle, the major internodal tract. Excitation spreads by these internodal tracts 2–3 times faster than by atrial myocardium. General direction of the excitation wave is downward and slightly to the left. Initially, excitation occurs in the right atrium, than spreads in the left atrium, and at the end only left atrium activates.

Conduction is delayed at the AV node. The excitation wave than passes to the bundle of His, proceeding along its main right and left branches to the Purkinje system. The Purkinje fibers penetrate the myocardium from endocardium toward epicardium. Since conduction is about three to five times faster in the Purkinje system than by muscle cell-to-cell conduction, the endocardial portion of the myocardium is activated more quickly than the subepicardial portion. Activation of the ventricular musculature occurs initially at the middle third of the left ventricular side of the interventricular septum, than the excitation wave spreads to the right and downward to lower part of the ventricular septum, to the apex, walls of the right ventricle, than to the left ventricle. The basal parts of the right ventricle, interventricular septum, and of the left ventricle are activated finally.

In general, the activation pathway is from the endocardial to the epicardial surface.

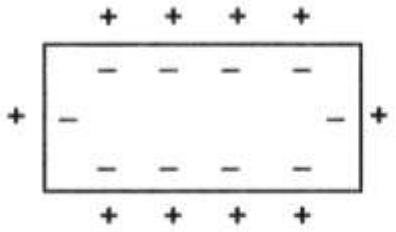
Depolarization, repolarization. Refractoriness

The transmission of an electric impulse from one muscle cell to another is dependent on depolarization, repolarization, and refractoriness.

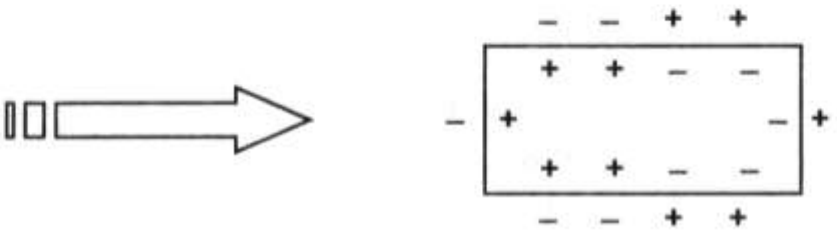
During excitation, physicochemical properties of the cell membranes and ionic composition of the intercellular and intracellular fluid change to cause the electrical current generation.

As the electrical impulse moves across the cells of the myocardium, the polarity (negative or positive charge) of the cells is changed.

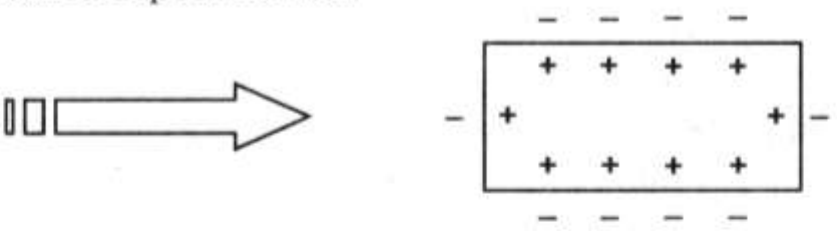
The resting cell outside has positive charge:



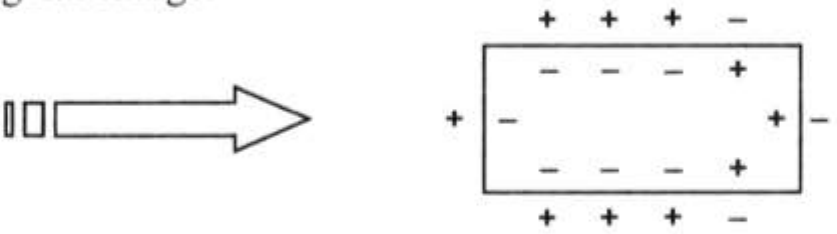
The electrical impulse carries a positive charge into the cell, changing the polarity:



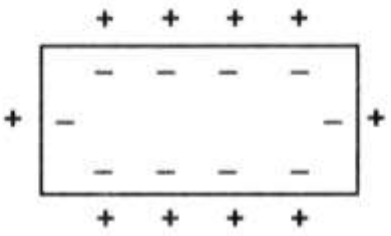
This is called depolarization:



It is followed by a continuing wave of repolarization that restores the cell to its original charge:



The cell is then ready to receive another stimulus.



During depolarization and early repolarization, the myocardial cell is completely refractory to further electrical stimulation, and excitability is zero; that is, no stimulus will produce response.

Refractoriness is the property of conductive and heart muscle tissue that prevents it from responding to a stimulus until it has appropriately repolarized. This prevents the muscle from sustaining a titanic contraction. Refractoriness is rate-dependent. The slower the rate and the longer the cycle, the longer refractory period.

Recording ECG. Development of electrocardiography is connected with the name of a Dutch physiologist Einthoven, who was the first in 1903 to record biocurrents of the heart by a string galvanometer. He developed some theoretical and practical principles of electrocardiography.

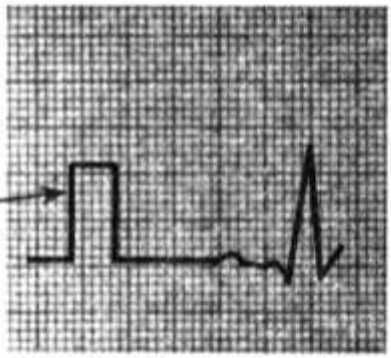
A modern **electrocardiograph** is actually a voltage-measuring instrument. It includes the following parts:

1. The sensitive elements, electrodes, which are attached to the body of the patient to pick up the potential differences that arise during excitation of the heart muscle, and lead wires;
2. Amplifiers, which amplify the minutest voltage of electromotive force (1–2 mV) to the level that can be recorded;
3. A galvanometer to measure the voltage;
4. A recording instrument, including a traction mechanism and a time marker;
5. A power unit.

Operating principle. Fluctuations in the potential difference that arise during the excitation of the heart muscle are sensed by the electrodes attached to the patient's body and transmitted to the apparatus. The input voltage is extremely low, and it is therefore amplified 600–700 times. Since the magnitude and direction of the electromotive force incessantly change during the cardiac cycle, the galvanometer pointer shows variations in the voltage a recording device draws a curve on a moving paper to show graphically these fluctuations. The recording principle differs with various instruments. Electrocardiographs are popular in which the fluctuations are recorded on a moving paper during the measurement. These are ink-writing instruments, which draw curves on paper. There are electrocardiographs in which special heat-sensitive paper is used. Dark paper is coated with heart-sensitive layers of paraffin and chalk; a hot stylus, which removes paraffin from the colored supporting paper, does the record.

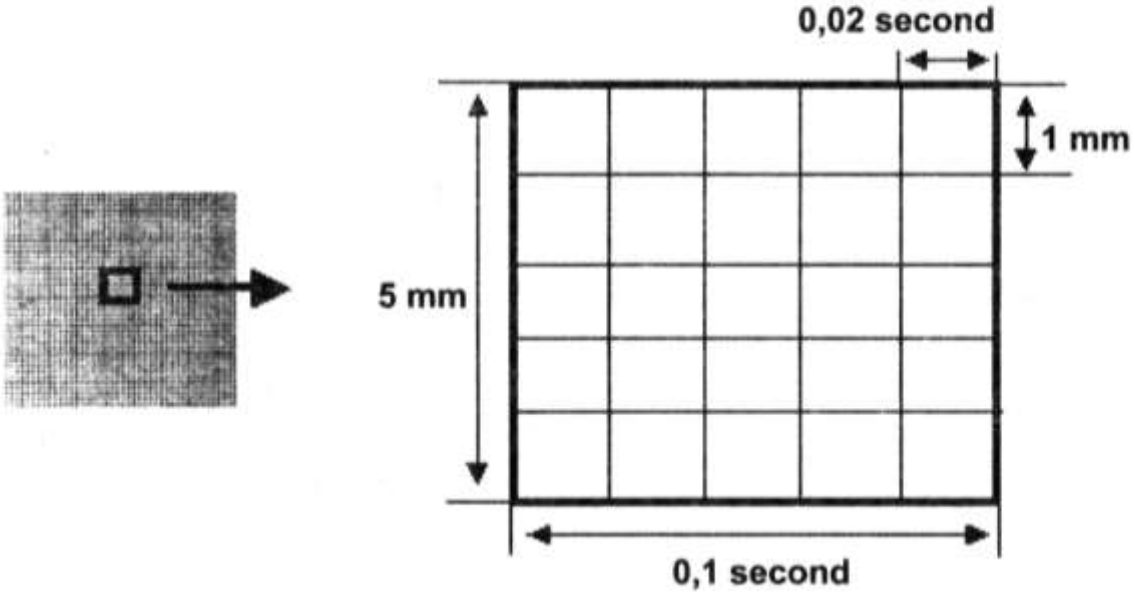
Whichever instrument is used, the sensitivity of the galvanometer is so selected that the voltage of 1 mV causes 1-cm deviation of the recording device. The sensitivity and amplification of the apparatus should be checked before recording electrocardiograms. To that end, a standardization voltage of 1 mV should be used, and this caused a 1-cm deviation of the stylus.

A normal one-millivolt curve
looks like a square wave.
The height of the vertical lines is 1 cm.

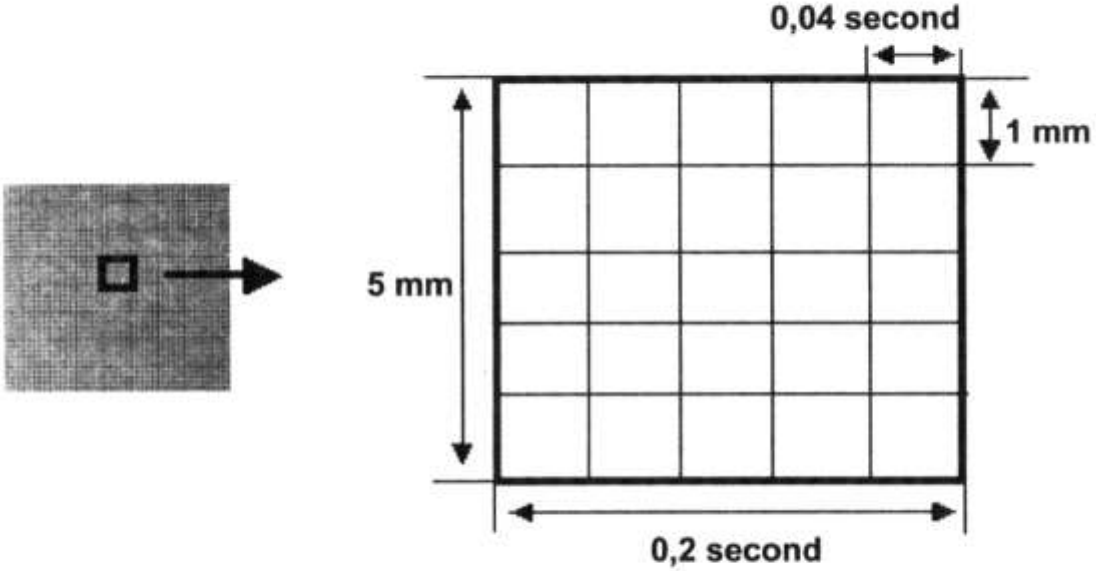


The tape may move at various speeds, from 25 to 100 mm/s, but the speed of 50 mm/s is usually preferred. Once the speed of the tape is known, it is easy to calculate the duration of the separate elements of the ECG. Waves amplitude is measured in mm, duration – in seconds.

ECG paper. The paper graduated in millimeters is used to record ECG. If an ECG is recorded at a speed of 50 mm/s, one small block represents 0.02 second on the horizontal line and 1 mm on the vertical line. Since a large block is five small blocks wide and five high, each large block represents 0.1 second (horizontal) and 5 mm (vertical).



If an ECG is recorded at a speed of 25 mm/s, one small block represents 0.04 second on the horizontal line and 1 mm on the vertical line. A large block represents 0.2 second (horizontal) and 5 mm (vertical).



The ECG leads

To record a routine ECG, 12 leads are used in clinical practice: 3 standard bipolar limb leads, 3 augmented unipolar limb leads, 6 chest leads.

Standard Bipolar Limb Leads

Standard leads proposed by Einthoven in 1913 y., record potential difference between two points of the body. The electrodes are placed on the right arm, the left arm, the left leg, and the fourth electrode on the right leg is connected with the earth wire.

Lead I is obtained by connecting the right arm (–) and left arm (+) electrodes.

Lead II is obtained by connecting the right arm (–) and left leg (+) electrodes.

Lead III is obtained by connecting the left arm (–) and left leg (+) electrodes.

Marks (+) and (–) indicate electrode connection to the positive or negative galvanometer pole (Fig. 4.49).

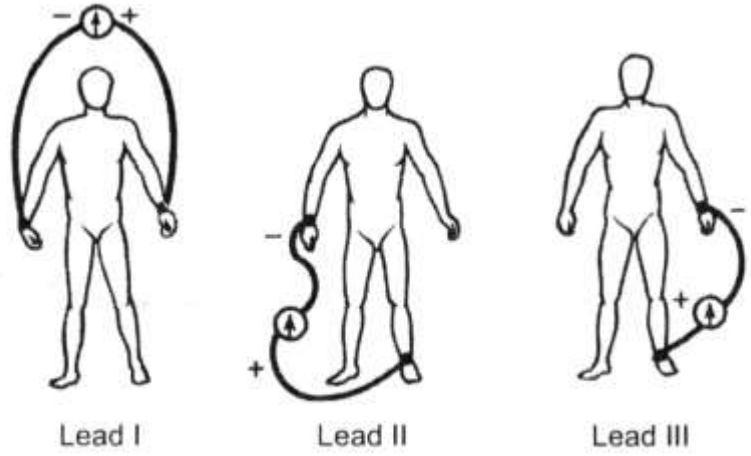


Fig. 4.49. Electrode placement in the standard leads technique.

Augmented Unipolar Limb Leads

Goldberger proposed augmented unipolar leads in 1942 year. Before Goldberger proposition unipolar limb leads by Wilson were taken with the exploring electrode connected to the designated extremity (right arm, left arm, left leg) and the indifferent electrode (central terminal) connected to all three limbs, including the extremity being explored. It was observed that the final deflections obtained with the Wilson central terminal system in the limb leads were generally of very low voltage, making interpretation difficult. Coldberger modified or "augmented Wilson's extremity lead, increasing the amplitude of the deflections by 50 %. He proposed to exclude from the electrode connection the electrode of the extremity being explored. The letter "a" is used to designate the augmented lead. Three augmented unipolar leads are distinguished (Fig. 4.50):

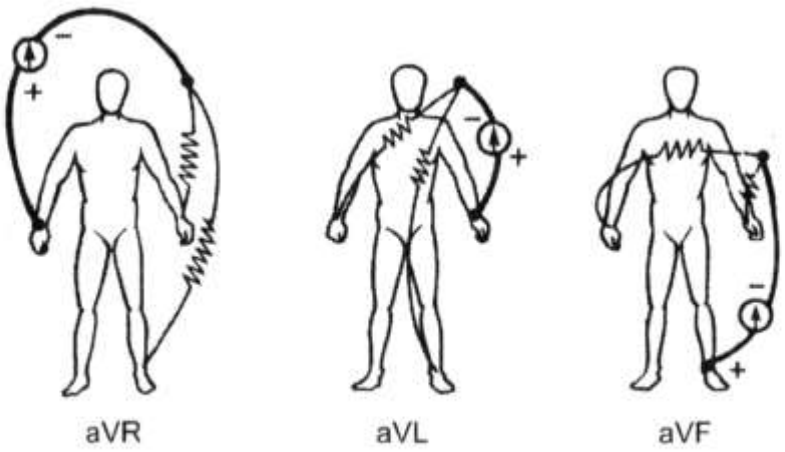


Fig. 4.50. Electrode placement in the augmented leads technique.

aVR: augmented unipolar right arm (+) lead, the central (–) terminal is connection of the right arm and left leg electrodes;

aVL: augmented unipolar left arm (+) lead, the central (–) terminal is connection of right arm and left leg electrodes;

aVF: augmented unipolar left leg (+) lead, the central terminal is connection of right arm and left arm electrodes.

Unipolar Chest Leads

Wilson proposed unipolar chest leads in 1934 year, are designated by the single capital letter V followed by a subscript numeral that represents the location of the active electrode on the precordium. The negative Wilson electrode is formed by connection of right arm, left arm, left leg electrodes.

In the 1938 the American and British Heart Associations appointed committees to standardize the nomenclature of precordial leads. The positions of the chest leads selected are (Fig. 4.51):

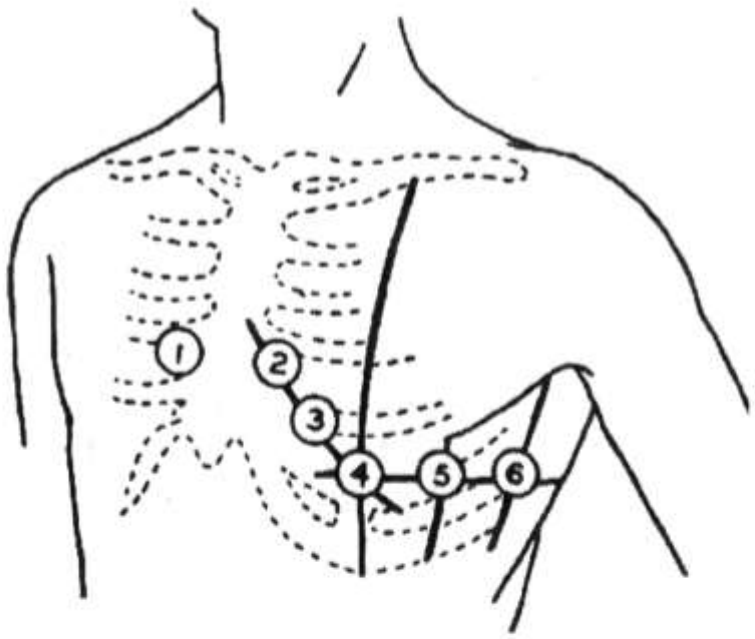


Fig. 4.51. Electrode placement in chest leads technique.

V_1: fourth intercostal space, right sternal border
V_2: fourth intercostal space, left sternal border
V_3: midway between V_2 and V_4 on a line jointing these two points
V_4: interspace in which apex is located (fifth or sixth); midclavicular line
V_5: anterior left axillary line; on the same level with V_4
V_6: left midaxillary line; on the same level with V_4 and V_5

The most important leads to remember in relation to the anatomy of the heart are:

Left leads	Right leads
Lead I	Lead III
Lead II confirms alterations in lead I or lead III, therefore:	
Leads I, II	Leads III, II
aVL	aVF, aVR
V4, V5, V6	V1, V2
V3 – transition zone between right and left sides of the heart	

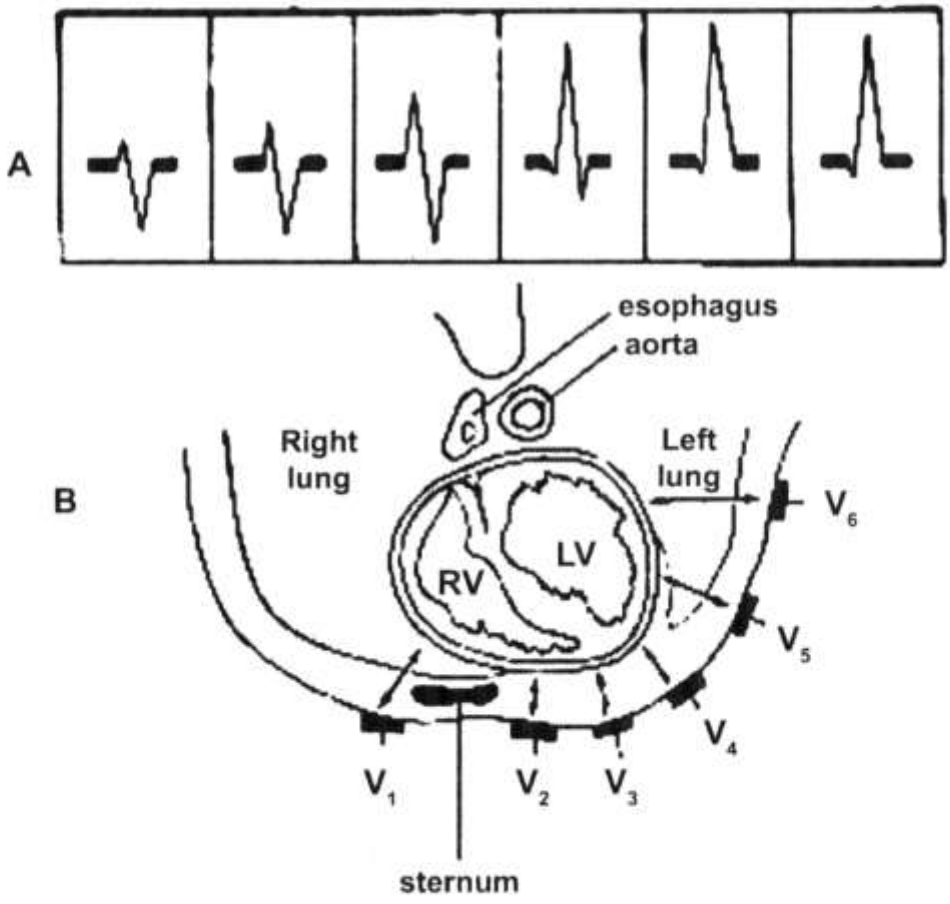


Fig. 4.52. A, the QRS changes commonly seen in leads V_1 to V_6 in a normal subject.
B, cross-section of the heart in the thoracic cage.
Note the relation of chest electrodes to the anatomy of the heart.

Basic ECG Principles

Einthoven Triangle

The Einthoven equilateral triangle concept supposes that the left arm, right arm, and left leg form the apexes of an equilateral triangle, while the heart, an electrical point, is assumed to be the center of the triangle. At any given moment of the cardiac cycle, the electromotive forces generated by the heart may be projected to the sides of the triangle. The sides of the triangle are the analogous to the three standard limb leads and are called lead axes (Fig. 4.53).

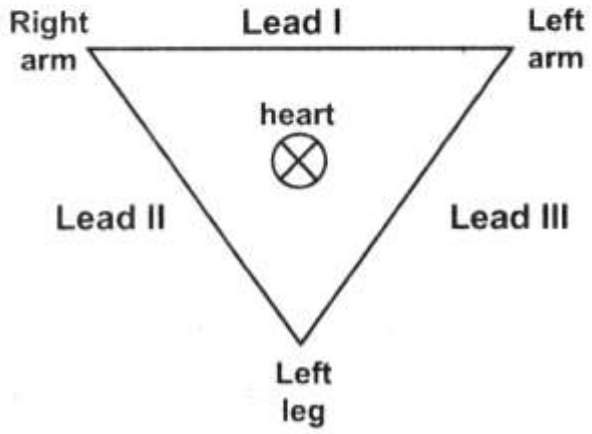


Fig. 4.53. The equilateral triangle of Einthoven, formed by leads I, II, and III.

The three standard leads have been arbitrarily established so that in lead I the right arm is negative and the left arm is positive, in lead II the right arm is negative and the left leg is positive, and in lead III the left arm is negative and the left leg is positive. These polarities are transported onto the Einthoven triangle.

The perpendicular from the center of the triangle divides each lead axis into two parts – positive turned to the positive limb and negative turned to the negative limb.

A single vector, called cardiac vector, which has magnitude, direction, and sense, represents depolarization and repolarization.

A vector projections that fall on the positive half of a lead axis appear as a positive deflections on that lead in the ECG, and on the negative half – as a negative deflection.

A vector force parallel to a given lead axis will project a large deflection in that lead; a vector perpendicular to a lead axis will project a small or biphasic complex in that lead (Fig. 4.54).

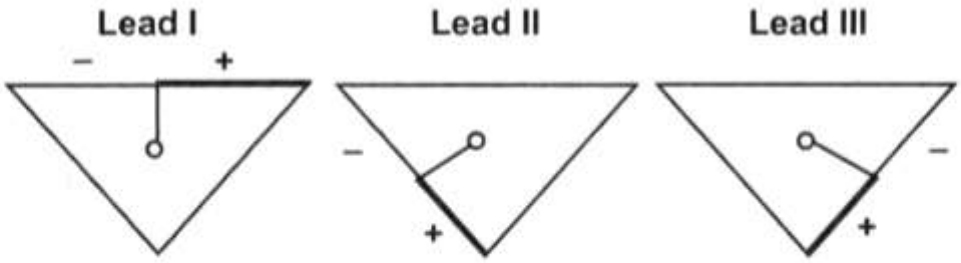


Fig. 4.54. Polarity of standard leads axes.

Triaxial and Hexaxial Frontal Plane Lead Reference System

When three sides of the triangle (leads I, II, and III) are transported so that their centers are superimposed on one another, the triaxial reference system described by Bayley is formed (Fig. 4.55).

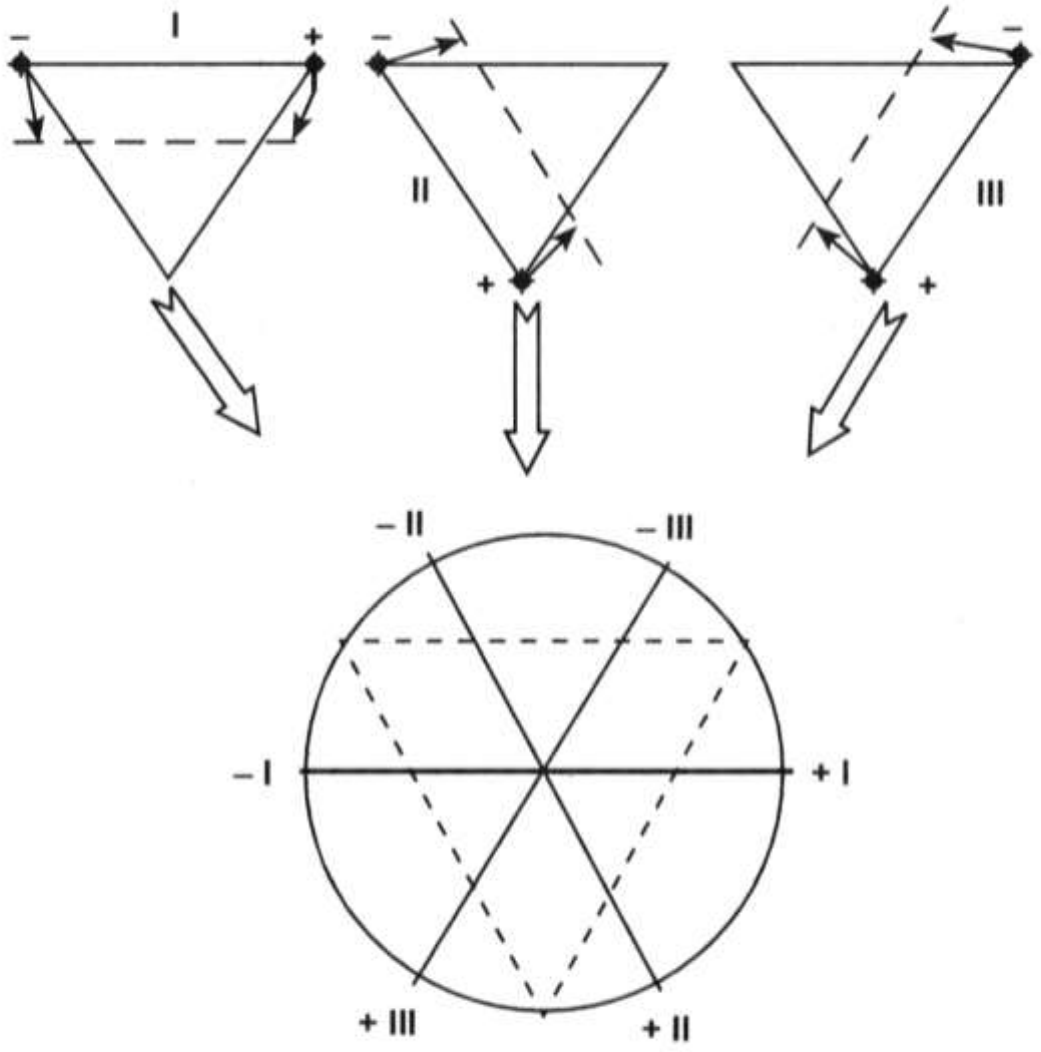


Fig. 4.55. The triaxial reference system of Bayley, produced by transposing the three sides of the triangle (leads I, II, and III) to a common central point.

The hexaxial reference system is produced by adding the augmented unipolar limb lead axes to the triaxial system (Fig. 4.56).

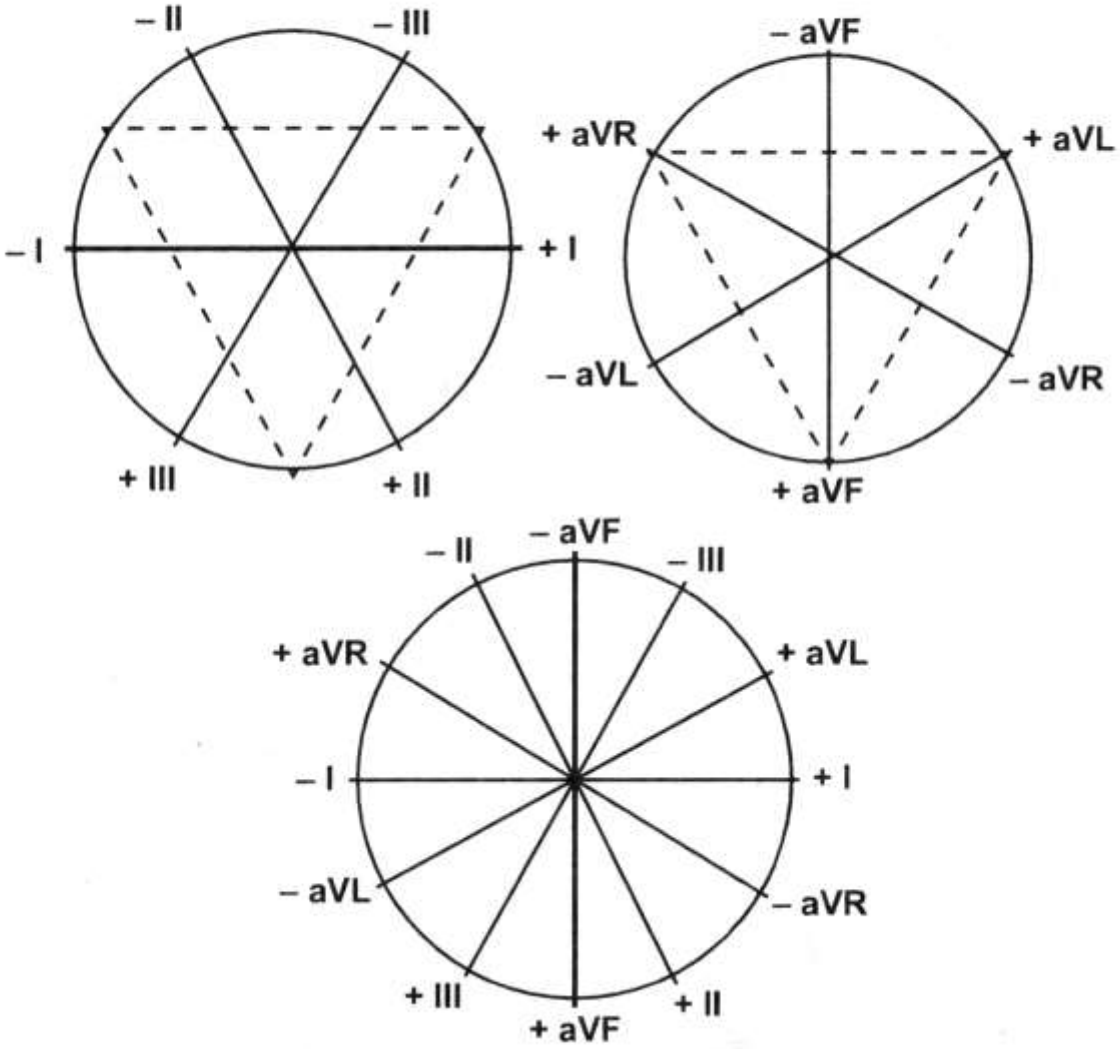


Fig. 4.56. The hexaxial reference system, produced by adding the augmented unipolar limb lead axes to the triaxial system.

The Electrical Axis of the Heart

The electrical axis of the heart coincides with its anatomical axis, and lies through the interventricular septum.

Einthoven's triangle and hexaxial reference system are useful for determining the electrical axis of the heart.

The basic principle is that an electrical force parallel to a given lead axis will record its largest deflection in that ECG lead; an electrical force perpendicular to a lead axis will record a small complex in that ECG lead.

If the cardiac vector is directed toward the positive pole of lead axis the deflections are positive in a given lead; if the cardiac vector is directed toward the negative pole of lead axis the deflections are negative in a given lead.

The electrical axis of the heart is determined by the shape of ventricular complexes in standard leads.

The relation between the electrical axis and the magnitude of the QRS complexes in standard leads is described by Einthoven triangle. The magnitude and direction of the electromotive force are designated by the A–B arrow.

If vertical lines are drawn from the ends of this arrow to the sides of the triangle, the difference of potentials recorded in each lead can be obtained.

In the normal position of the heart the direction of electrical axis is downward and leftward, therefore the highest R wave will be recorded in the lead II, the lowest – in the lead III (Fig. 4.57).

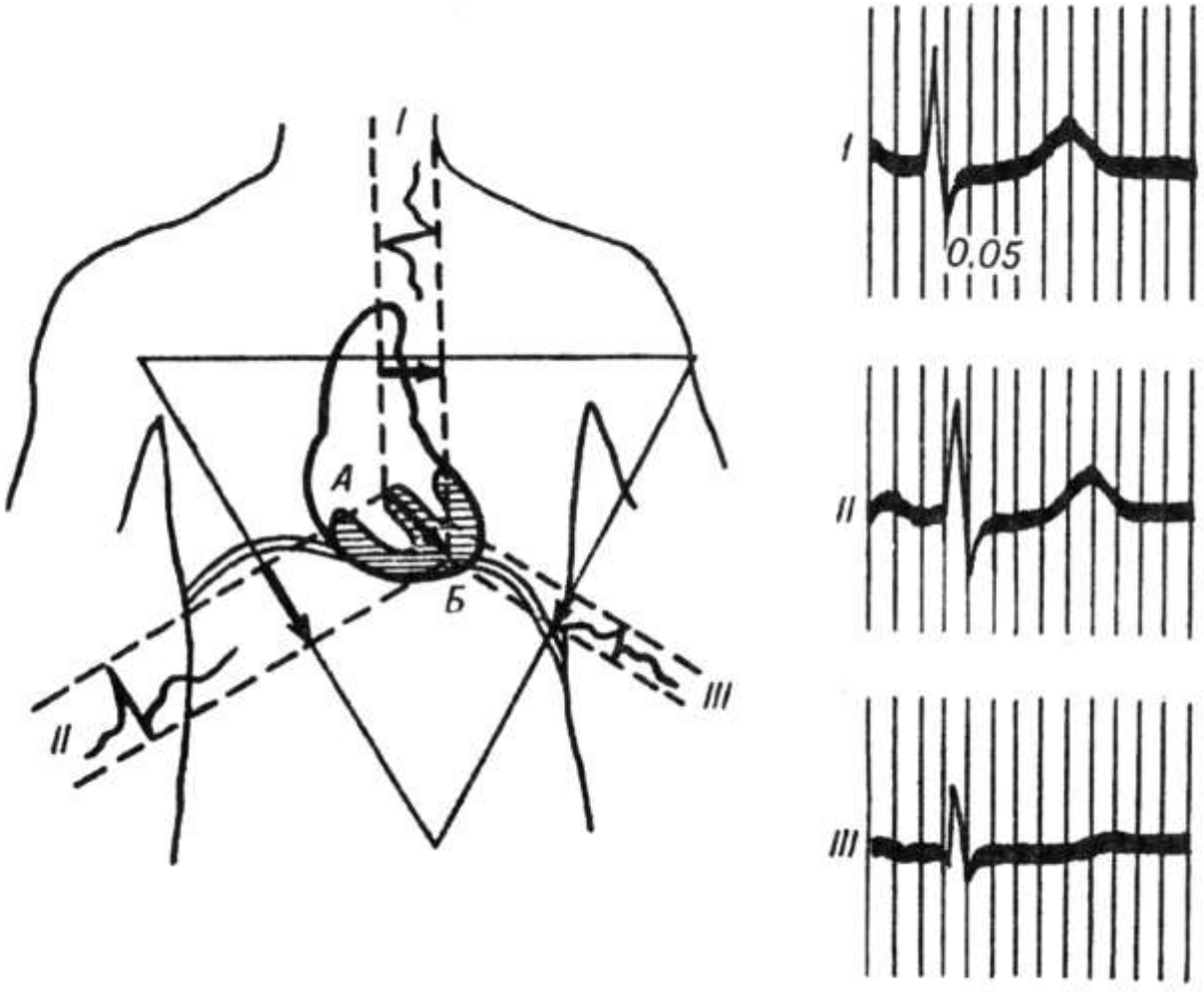


Fig. 4.57. Normal position of the heart electrical axis.

The direction of electrical axis of the heart changes depends on the position of the heart in the thoracic cage.

Values of R wave amplitude in normal position of the electrical axis can be shown as follows: $R_{II} > R_{I} > R_{III}$, or it can be calculated that amplitude of the R wave in lead II is equal to the algebraic sum of R in leads I and III: $R_{II} = R_{I} + R_{III}$.

In high diaphragm level (hypersthenic persons) the heart assumes horizontal position (so-called "lying heart"), and electrical axis of the heart deviates to the left or horizontal become more parallel to the lead I. (Fig. 4.58).

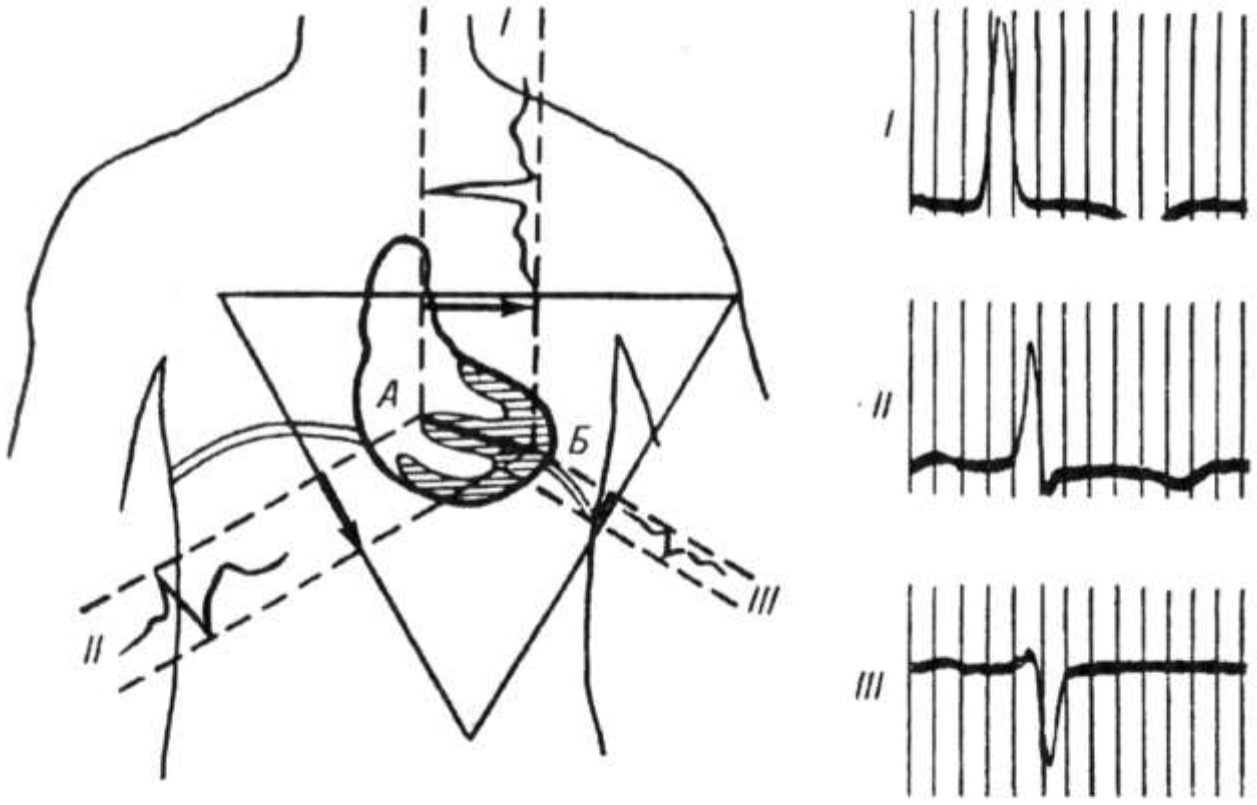


Fig. 4.58. Horizontal position of the heart electrical axis.

Therefore, the highest R wave in horizontal position of the heart is recorded in the lead I: $R_{I} > R_{II} > R_{III}$.

In low diaphragm level (asthenic subjects) the position of the heart is vertical (so-called "drop heart"), and electrical axis of the heart deviates to the right or vertical, that is, more parallel to the lead III (Fig. 4.59).

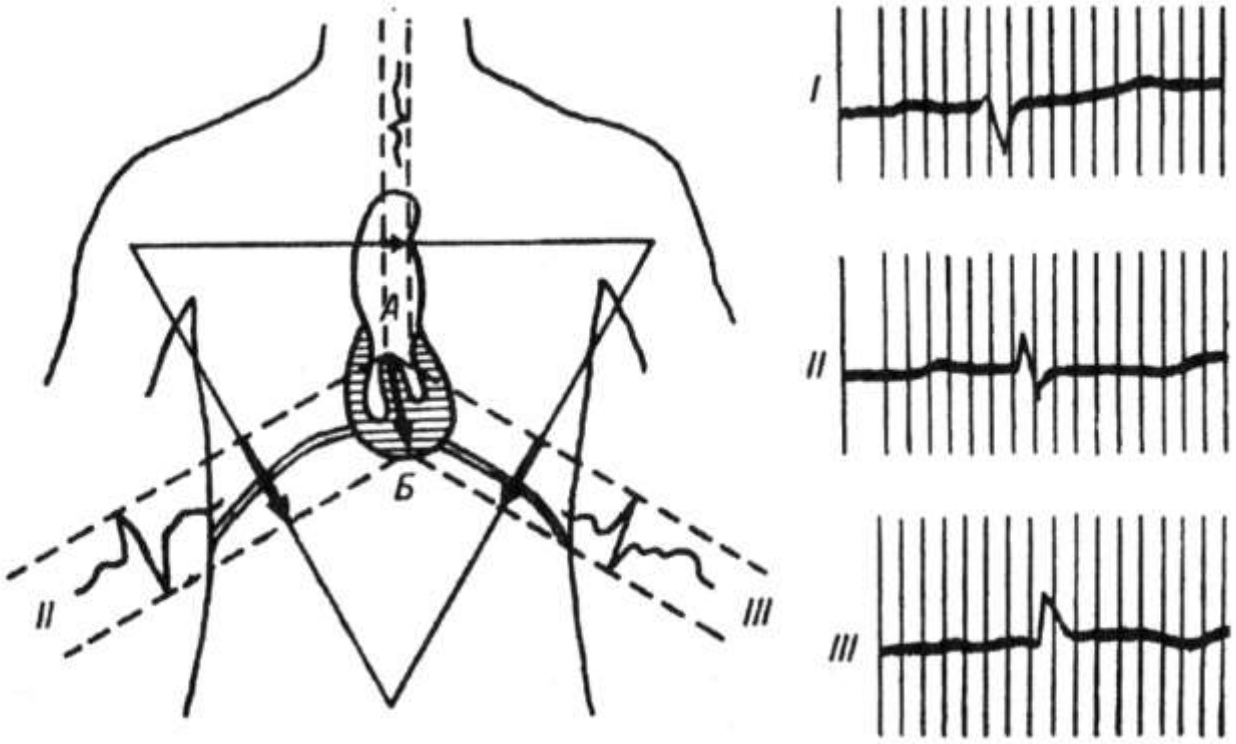


Fig. 4.59. Vertical position of the heart electrical axis.

The highest R wave is therefore recorded in lead III: $R_{III} > R_{II} > R_{I}$

The Electrocardiogram. The electrocardiogram is simply a graphic representation of the electrical forces produced by the heart (Fig. 4.60).

Electrical activation of the heart is initiated by the SA node; however, this does not cause an electrical deflection on the surface ECG.

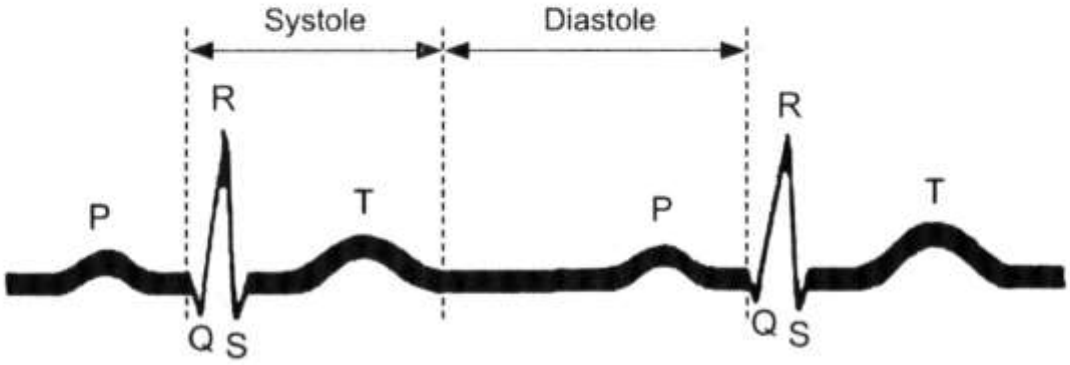


Fig. 4.60. Normal electrocardiogram: sinus rhythm.

P wave

The P wave is the first upward deflection and is the graphic representation of the electrical activation of the atria. As impulse at first cause excitation of the right atrium and than the left atrium, the ascending portion of P wave reflects depolarization of the right atrium, and descending portion reflects depolarization of the left atrium (Fig. 4.61).

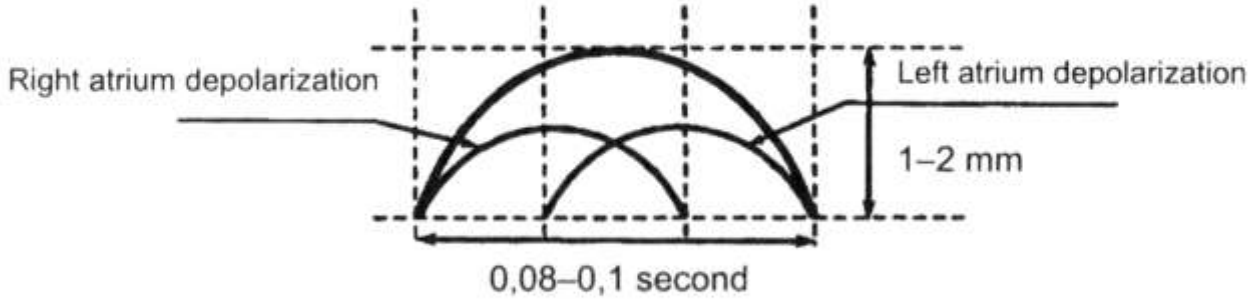


Fig. 4.61. The normal P wave formation.

1. The normal P wave duration is 0.08 – 0.10 second, amplitude – 1–2 mm.
2. The normal P wave is upright in the I, II, aVF, $V_2 - V_6$ leads.
3. P wave may be upright, two-phased in III, aVL, V_1 leads, and sometimes even inverted in the III and aVL leads.
4. P wave is always inverted in the aVR lead.

P–Q (or R) interval measured from the beginning of the P wave to the onset of the Q (or R) wave includes activation in atria, the AV node, the His' bundle, bundle branches, and the Purkinje network. The normal P–Q duration is 0.12–0.2 second. The P – Q segment, measured from the end of the P wave to the onset of the Q wave, represents mostly the delay in activation as the impulse passes through the AV node and His' bundle (Fig. 4.62).

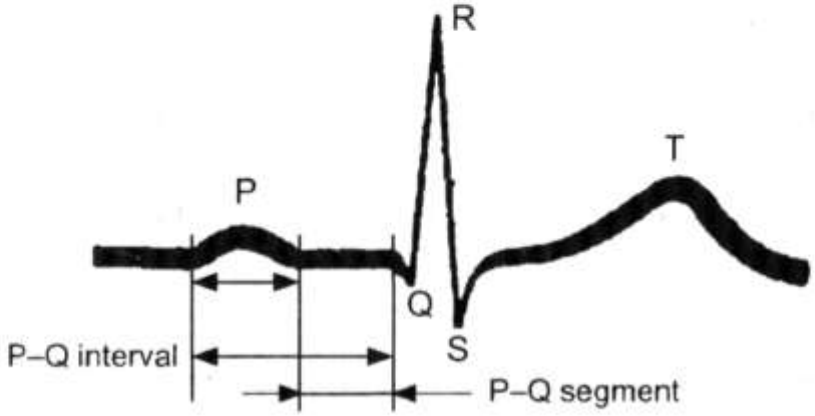


Fig. 4.62. The P – Q segment and P – Q interval.

Ventricular complex QRST

Q wave represents depolarization of the interventricular septum.

1. The normal Q wave amplitude in all leads except aVR is not more than ¼ of the R wave amplitude, and its duration is 0.03 second.
2. The normal Q wave is inverted in I, II, III, aVL, aVF, V_4–V_6 leads.
3. Q wave may be deep and wide in the aVR lead, or even QS complex may register.

R wave represents the process of depolarization of the ventricles.

1. The normal R wave amplitude is 5–15 mm.
2. The normal R wave is recorded in all standard and augmented limb leads. In the aVR lead R wave may be low or even absent.
3. In the chest leads R wave amplitude increases from V_1 to V_4, and than slightly decreases in V_5 and V_6 leads.
4. $R_{V1,V2}$ reflects activation of the interventricular septum, and R wave in V_4, V_5, V_6 leads activation of the right and left ventricles.

S wave formation on the ECG reflects depolarization of the basal parts of interventricular septum of right and left ventricles.

1. The normal S wave is inverted, its amplitude in the different electrocardiographic lead is within the large ranges, not exceed 20 mm (2.5 mm on the average).
2. In the limb lids S wave amplitude is low, except aVR lead, in the normal position of the heart in the chest.
3. In the chest leads S wave amplitude decreases from V_1, V_2 to V_4, and in V_5, V_6 leads S wave amplitude is very low or S wave may be even absent.
4. Equal S wave and R wave amplitude in the chest leads are usually in the V_3 ("transition zone") or (rarer) between V_2 and V_3 or V_3 and V_4.

The QRS interval, measured from the beginning of the Q wave to the end of the S wave, represents the process of depolarization of the ventricles.

The normal duration of the QRS interval is 0.06–0.1 sec., this time corresponds to the intraventricular conduction (Fig. 4.63).

The ST segment represents the period when all parts of the ventricles are in the depolarized state. The ST segment duration depends on the heart rate (Fig. 4.63).

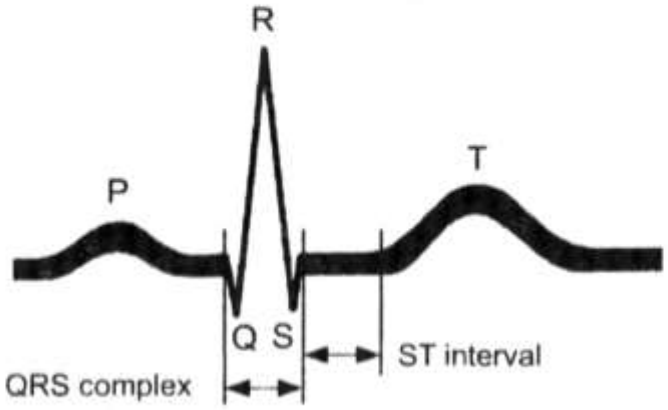


Fig 4.63. Ventricular complex QRS and ST segment on the normal ECG.

T wave represents repolarization of both ventricles. The normal T wave is asymmetric: the gradual ascent converts into a rounded summit, which is followed by abrupt descent.

1. The normal T wave duration is 0.12–0.16 second, amplitude – 2.5–6 mm.
2. The normal T wave is always upright in I, II, aVF, V_2–V_6, $T_I > T_{III}$, and $T_{V6} > T_{V1}$ leads.
3. T wave may be upright, two-phased or inverted in III, aVL and V_1 leads.
4. The normal T wave is always inverted in aVR lead.

Q–T interval (QRST complex), measured from the beginning of the Q (or R) wave to the end of the T wave, represents electrical ventricular systole. Its duration depends on the cardiac rate. The Q–T interval in women is longer than in man. For example, at the rate of 60–80 beats per minute, Q–T duration is 0.32–0.37 second in men, and 0.35–0.4 in women (Fig. 4.5).

T–P interval, measured from the end of the T wave to the beginning of the P wave, represents electrical diastole of the heart. Its duration depends on the heart rate (Fig. 4.64).

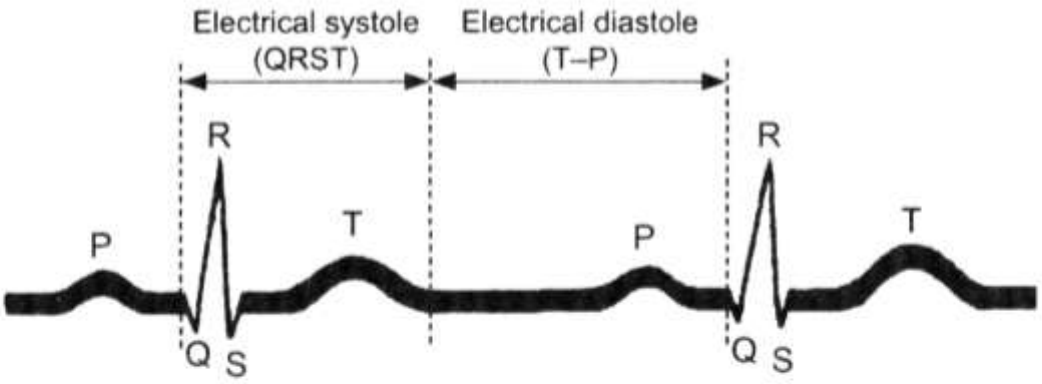


Fig. 4.64. Electrical systole and diastole on the normal ECG.

In each cardiac cycle there are two electrical processes: depolarization and repolarization. Depolarization is an electrochemical phenomenon that occurs rapidly. Repolarization is an electrochemical phenomenon of energy restoration that occurs more slowly. Therefore, QRS complex (ventricular depolarization) is of short duration, but the T wave (ventricular repolarization) is of long duration (Fig. 4.65).

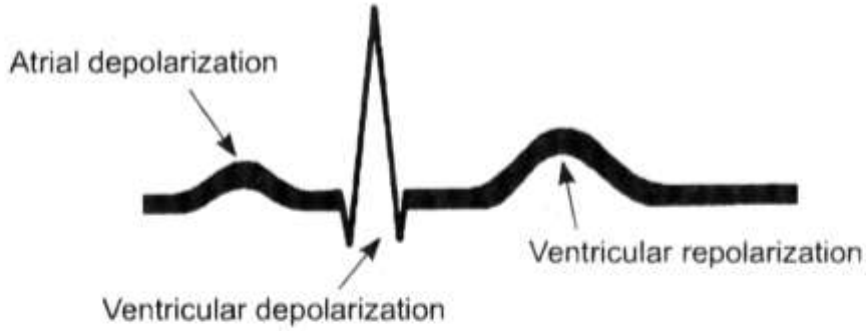


Fig. 4.65. The depolarization and repolarization portions on the normal ECG.

Interpretation of the ECG

1. ***Determination of the Cardiac Rhythm Regularity.*** The R–R intervals in regular rhythm should be equal. Its fluctuation normally does not exceed 0.1 second. Greater variations in the R–R intervals duration indicate irregular cardiac rhythm.
2. ***Calculation of the Heart Rate.*** In regular cardiac rhythm the heart rate is determined by formula: $HR = \frac{60}{R-R}$; where 60 is a number of seconds in minute, R–R – duration of the R–R intervals in seconds.

3. ***Measurement of the ECG Amplitude.*** The R waves amplitude are measured in standard leads. If the amplitude of the highest R wave does not exceed 5 mm, or the sum of the R waves amplitude in the three standard leads is less than 15 mm the ECG voltage is considered decreased.
4. ***Determination of the Cardiac Rhythm Pacemaker Site.*** The normal sinus rhythm is characterized by upright P wave in lead I, II, aVF following by QRS complex. The P waves configuration is equal in each lead.
5. ***Estimation of the Conductivity.*** The P wave duration indicates time of atria activation, the P–Q interval and the QRS complex duration – ventricular activation. Increased duration of these waves and intervals indicates slowed impulse conduction in corresponding part of the cardiac conduction system.
6. ***Determination of the Electrical Axis of the Heart.***
7. ***Measurement of the duration and amplitude of the ECG waves and intervals.***
8. ***ECG conclusion.*** In the ECG conclusion it is necessary to note following:
   a) The cardiac rhythm pacemaker (sinus or nonsinus rhythm);
   b) Regularity of the cardiac rhythm (regular or irregular);
   c) The heart rate;
   d) Position of the electrical axis of the heart;
   e) Presence of the four ECG syndromes: arrhythmias, abnormalities of conductivity, atrial and ventricular hypertrophy, myocardial damage (ischemia, injury, necrosis, scar).

Diagnostic Electrocardiographic Signs of Atrial and Ventricular Hypertrophy

Left Atrial Enlargement

Left atrial enlargement is now seen most commonly in the patients with mitral valvular diseases, aortic insufficiency, and in systemic hypertension.

With the left atrium enlargement, the frontal plane P vector is oriented more horizontally and is of longer duration because the left atrial activation is prolonged. Therefore, the P wave duration is greater than 0,1 second, and amplitude of the left atrial phase of P wave increases that can cause splitting of this wave (Fig. 4.66).

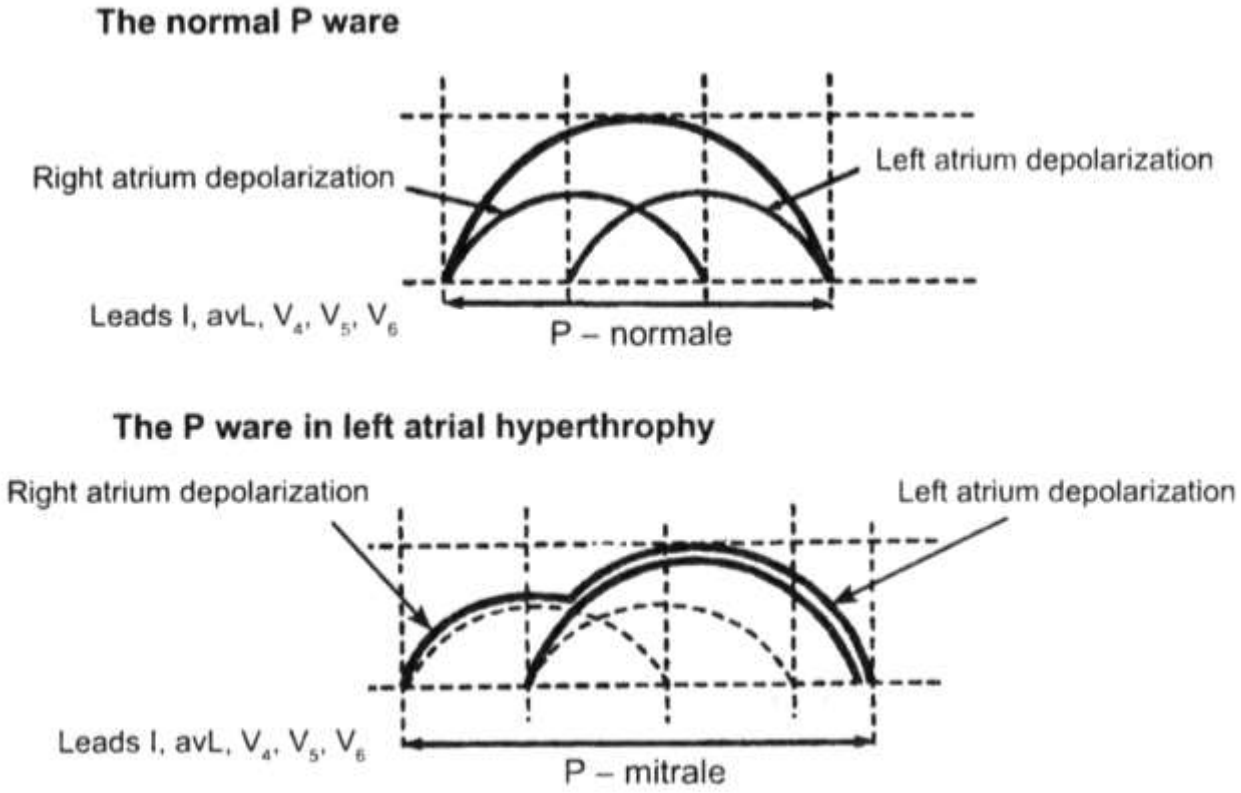


Fig. 4.66. Left atrial enlargement.
Note increased duration and amplitude of left atrial phase of the P wave.

The left atrial enlargement occurred most commonly in the patients with mitral stenosis. For this reason, this type of the P wave morphology if often termed **P – mitrale** (Fig. 4.67).

Various indices have been established for the diagnosis of left atrial enlargement. The Macruz index measures the ratio between the duration of the P wave and P – R segment. In normal persons this is between 1.0 and 1.6. in the left atrial enlargement, the P wave duration increases but P – R interval tends to remain constant. Thus Macruz index becomes larger than 1.6.

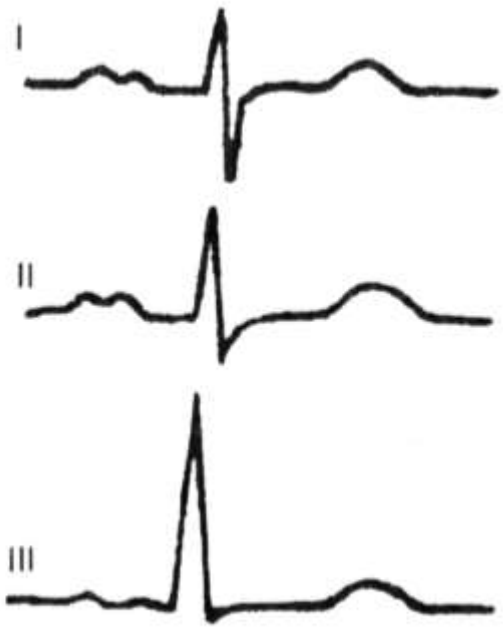


Fig. 4.67. P – mitrale.

Diagnostic Electrocardiographic Signs of Left Atrial Enlargement

1. High-amplitude and two-peaked P wave in leads I, II, aVL, V_4, V_5, V_6.
2. In lead V_1 (rarer in V_2) P wave is initially positive and terminally negative or negative P wave in V_1 is formated.
3. In lead III negative or two-phased (+ –) P wave (inconstant sign).
4. The P wave duration is more than 0,1 second.

Right Atrium Enlargement

Right atrium hypertrophy usually observes in diseases, which are accompanied by hypertension in the lesser circulation. Right atrial enlargement causes the frontal plane P wave axis to shift vertically. Since the right atrium is the first to be activated, prolongation of its activation time as a result of enlargement does not cause widening of the P wave, but only increased amplitude (Fig. 4.68).

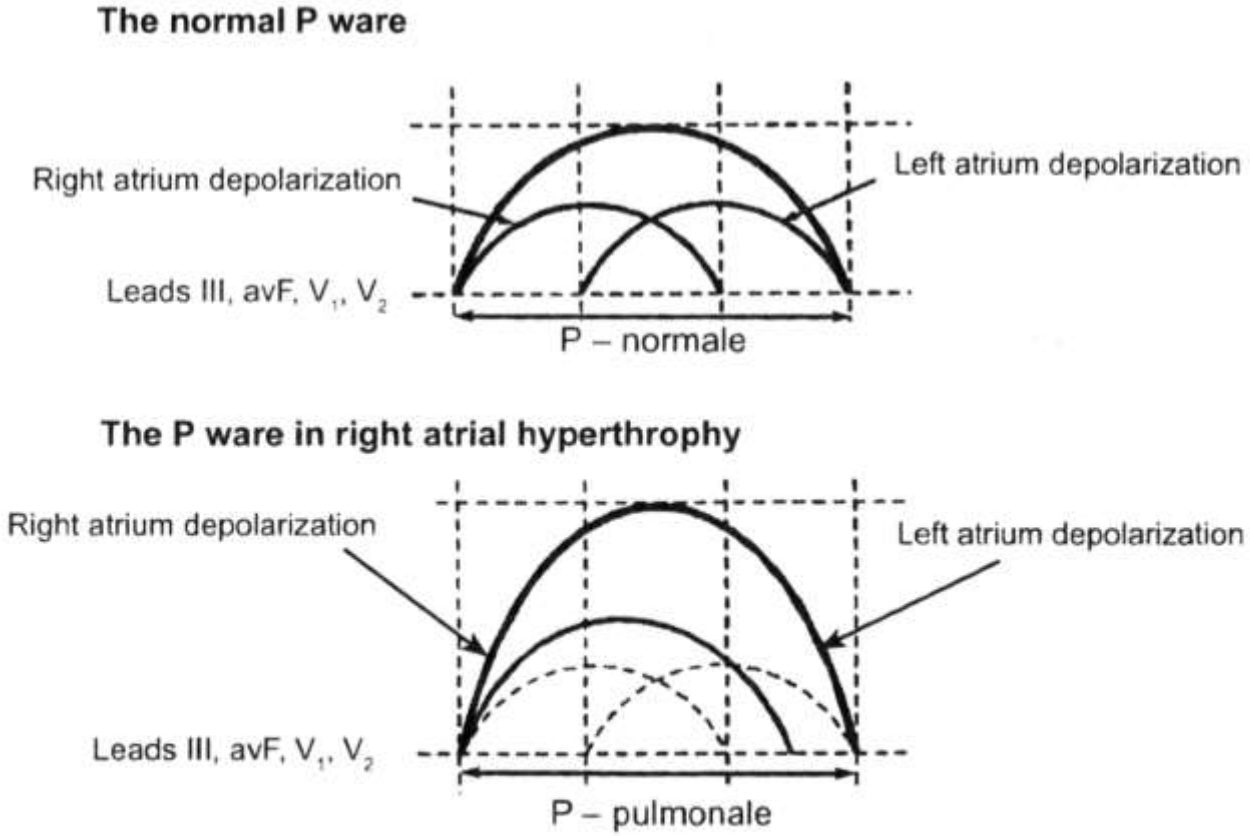


Fig. 4.68. Right atrial enlargement.
Note increased amplitude of right atrial phase of the P wave.

Diagnostic Electrocardiographic Signs of Right Atrial Enlargement

1. High-amplitude, peaked P wave more than 2 mm, higher than ¼ of the R wave amplitude in leads II, III, aVF.
2. Positive, peaked P wave (or its initial right atrial phase) in leads V_1, V_2.
3. Low-amplitude P wave in leads I, II, aVL, V_4, V_5, V_6, in lead aVL it may be negative (inconstant sign).
4. The P wave duration is not more than 0,1 second.

Since right atrial enlargement is often due to pulmonary diseases, the terms **P – pulmonale** is used to describe this P wave morphology (Fig. 4.69).

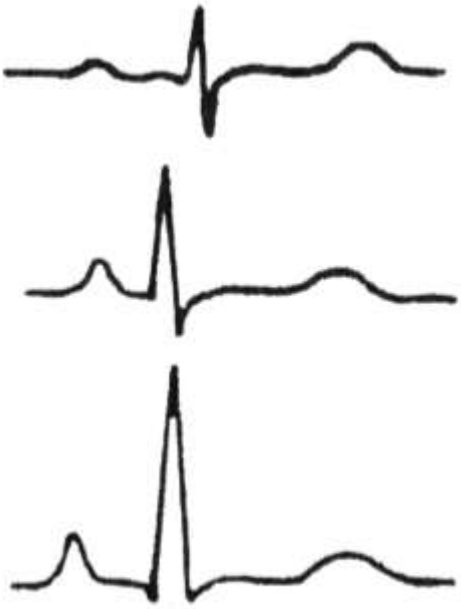


Fig. 4.69. P – pulmonale.

Left Ventricular Hypertrophy

Left ventricular hypertrophy is initially a useful compensatory process that represents an adaptation to chronic hemodynamic overload. However, left ventricular hypertrophy is also the first step toward the development of overt clinical diseases such as congestive heart failure, cardiac arrhythmias, and coronary heart disease.

Diagnostic Electrocardiographic Signs of Left Ventricular Hypertrophy

1. Increased voltage of QRS deflection. In the presence of ventricular hypertrophy the increased magnitude of the left ventricular forces from the hypertrophied left wall results in increased magnitude of the main QRS vector. The increased QRS vector if therefore oriented more posteriorly, superiorly, and to the left than normally, and projected on the positive half of the axes of leads I, aVL, V_4, V_5, V_6, and on negative half of the axes of leads III, aVF, V_1, V_2, V_3. Therefore, the left leads **I, aVL, V_4, V_5, V_6** show **high-amplitude R waves** and the right leads **III, aVF, V_1, V_2, V_3** show a **deep S wave.**
2. Increased duration of the QRS complex as a consequence of the increased muscle mass, the activation wave must travel a longer than normal course,

and the **QRS complex is widened to 0,12 second**. However, it may also be 0,10 second or less and still be compatible with the diagnosis of the left ventricular enlargement.

3. ST segment and T wave changes. **The ST segment is depressed and the T wave is inverted in the leads I, II, aVL, V_4, V_5, V_6.** In the right precordial leads (V_1, V_2) the T wave is upright and the ST segment may be slightly elevated. ST segment and T wave changes are result from the altered ventricular depolarization and repolarization processes in the presence of left ventricular hypertrophy. The ST segment is normally isoelectric for the following reason. Repolarization begins shortly before ventricular activation is completed; however the potential of early repolarization does not reach sufficient magnitude at the completion of ventricular activation to be recordable. When the repolarization forces reach sufficient potential to be registered, the T wave is inscribed. In the left ventricular hypertrophy, left ventricular activation is prolonged; hence, by the time ventricular activation is completed, the potential of repolarization is of sufficient magnitude to produce a deviation of the ST segment. There are two possible explanations for the inverted T wave: 1 – the T wave chance may be primary, due to myocardial ischemia resulting from the relative disproportion between the increased ventricular muscle mass and the available blood supply; or 2 – the changes may be secondary. Since activation of the left ventricle is prolonged, and the direction of repolarization is reversed (endocardium toward epicardium rather than normal direction of epicardium to endocardium), the overall balance of T forces in the hypertrophied ventricle causes the T vector to be oriented away from the QRS vectors to produce inverted T wave.
4. **Left axis deviation**. In the left ventricular hypertrophy electrical axis of the heart have a leftward orientation because the left ventricular mass is greatly increased and there may be an anatomical change in the position of the heart. **$R_I \geq 15$ mm, $R_{aVL} \geq 11$ mm, or $R_I + S_{III} > 25$ mm.**
5. **The transition zone is displaced to the right (V_2)**. In marked hypertrophy, the enlarged left ventricle as moves a little the right ventricle to the right, and heart turns around its vertical axis against clock hand.
6. **The deep S wave is preserved in lead III during deep inspiration**. During deep inspiration the diaphragm lowers, and if axis deviation is positional, deep S wave disappears because the heart assumes normal po-

sition. And, if axis deviation is caused by enlarged left ventricle, deep S wave preserves in lead III during deep inspiration.

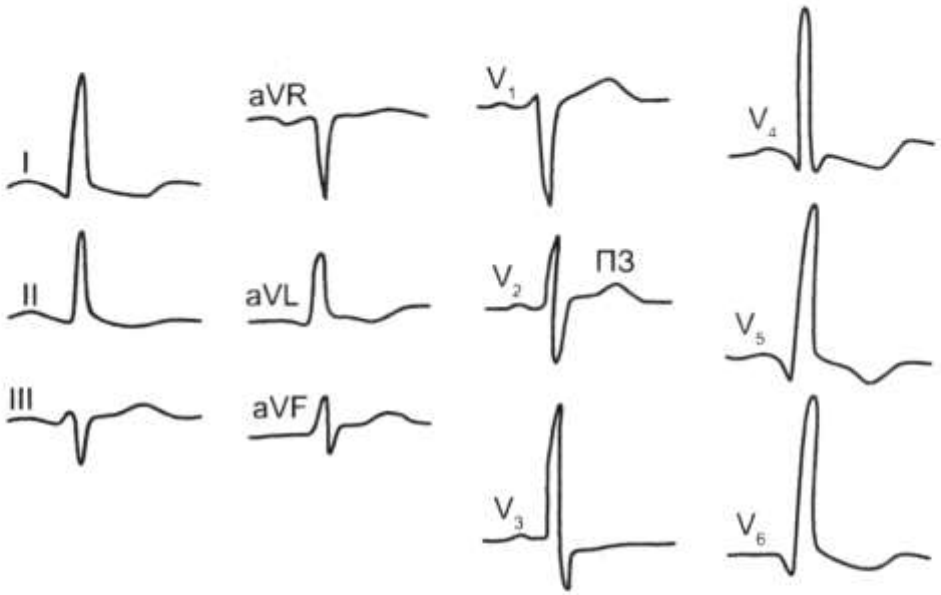


Fig. 4.70. Left ventricular hypertrophy.

Right Ventricular Hypertrophy

Right ventricular hypertrophy is initially a compensatory process that represents an adaptation to longstanding hemodynamic overload in the patients with mitral stenosis, and in chronic pulmonary diseases, which are accompanied by hypertension in the lesser circulation.

Diagnostic Electrocardiographic Signs of Right Ventricular Hypertrophy

1. **Right axis deviation.**
2. The right leads **III, aVF, V_1, V_2, V_3** show **high-amplitude R waves** and the left leads **I, aVL, V_4, V_5, V_6** show a **deep S wave.** $R_{V1} \geq 7$ mm or $R_{V1} + S_{V5,6} \geq 10{,}5$ mm.
3. Duration of the QRS complex. In contrast to the pattern in left ventricular hypertrophy, the QRS duration seldom is prolonged (to 0,12 second), because even with hypertrophy, the thickness of the right ventricle does not exceed that of the left.
4. In leads **III, aVF, V_1, V_2, V_3 the ST segment** may be **depressed** and the **T wave inverted** over the hypertrophied right ventricle. When ST–T wave changes are present, it is often indicate of more severe right ventricular hypertrophy.

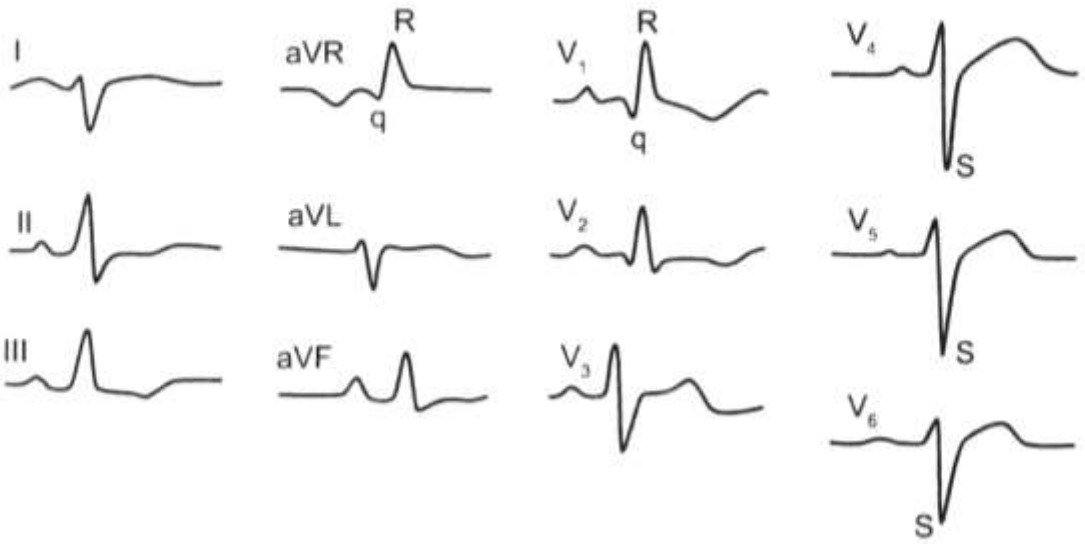


Fig. 4.71. Right ventricular hypertrophy.

5. **The transition zone is displaced to the left (V_5–V_6).** In the right ventricular the muscle mass is increased, and the heart tends to rotate on its longitudinal axis in a clockwise manner, so that the right ventricle becomes more anterior and the left ventricle rotates posteriorly. The septum rotates similarly, becoming more parallel to the frontal plane of the body.

Coronary (Ischemic) Heart Disease

The term coronary heart disease includes such diseases as angina pectoris, myocardial infarction, and coronary cardiosclerosis.

The most frequent cause of the **angina pectoris** is atherosclerosis of the heart coronary arteries. Its main clinical symptoms are attacks of retrosternal pain due to acute but transient disorder in the coronary circulation.

Myocardial infarction is formation of a necrotic focus in the heart muscle due to upset coronary circulation.

Cardiosclerosis is the disease of the myocardium caused by developing fibroid elements in the heart muscle. Atherosclerotic and myocarditic cardioscleroses are distinguished. The later may result from any myocarditis. Atherosclerotic cardiosclerosis is the result of atherosclerosis of the coronary arteries. Myocardial infarction that ends in the formation of scars becomes the cause of focal post-infarction cardiosclerosis.

ECG signs of the coronary heart disease

If the coronary artery is occluded the involved heart muscle progresses in sequence through three stages of damage toward infarction. Each stage is associated with electrical changes.

Ischemia develops in conditions when the insufficient amount of blood is delivered to the heart muscle through the coronary arteries, and the myocardium does not receive the necessary amount of oxygen. Ischemic damage of the myocardium alters the sequence of ventricular activation and affects the repolarization or the recovery process of the heart. These changes alter the electrical field of the heart, resulting in T wave modifications. Ischemia is recognized by symmetrically inverted T wave. Ischemia is a reversible process unassociated with histological changes (Fig. 4.72).

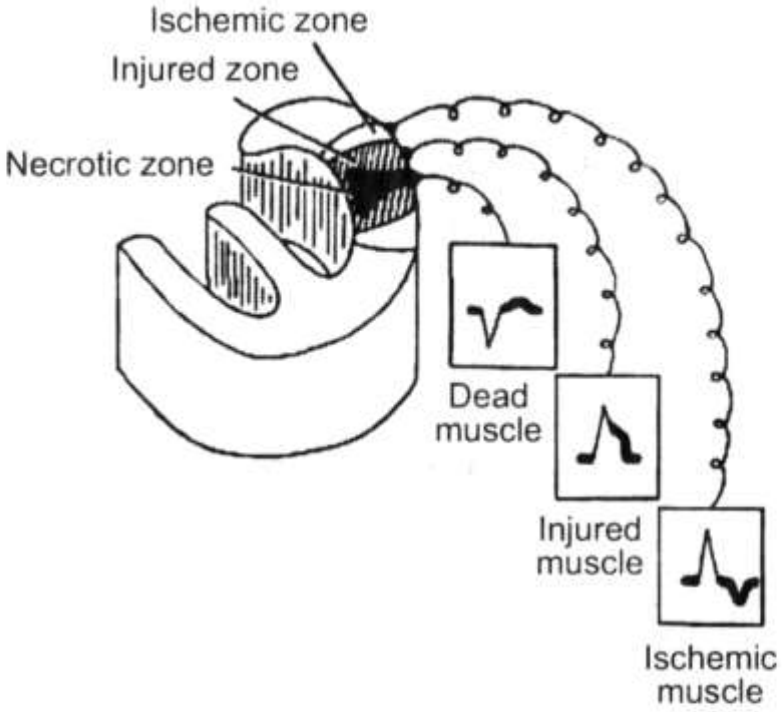


Fig. 4.72. Ischemic, injured, and infracted zones with their respective electrical patterns.

Injury is recognized by ST segment elevation. These changes are caused by persisted ischemia, which lead to more significant alteration of the repolarization processes. The injury patterns are also reversible.

If a deficient blood supply persists injured muscle progresses to necrosis.

Necrosis or infarction. With infarction, "a dead zone" appears and become electrically inactive. Dead or necrotic muscle tissue is inexcitable and

incapable of producing an action potential. Such tissue acts as a passive conductor of the potential forces generated in viable areas of the myocardium. In the event of myocardial death, a Q wave appears and the R wave deflection decreases in amplitude or disappears. The depth of the Q wave is directly proportional to the relative thickness of the dead zone, and the height of the R wave is directly proportional to the amount of living tissue that escapes death. Surrounding the area of acute infarction are zones of injured muscle and ischemic muscle. These adjacent zones are transient, and either eventual progression to the stage of necrosis or recovery occurs, depending on the collateral circulation.

Diagnostic electrocardiographic signs of myocardial infarction

1. Decreased R wave amplitude or its absence in the leads facing the necrotic myocardium. $(R_I + R_{II} + R_{III}) : 3$ = less than 5 mm.
2. Deep and wide Q wave, deeper than ¼ of the R wave amplitude, and wider than 0.03 second.
3. Pathological T wave – high ischemic or inverted (++, + –, – –, – +).
4. ST segment changes.
5. Discordance of ST segment and T wave in opposite leads. That is, ST elevation in the lead I and depression in the lead III; T wave upright in the lead I and inverted in the lead III. Concordance of ST segment and T wave in opposite leads is an ECG sign of the angina pectoris.

Evolution of myocardial infarction

The clinical myocardial injury pattern does not remain stationary, but changes. Evolutionary ECG alterations take place over a period of hours, days, weeks, month, or even years. The series of changes has been arbitrarily separated into stages (Fig. 4.73).

Stages of the myocardial infarction

Stage 1 – *acute*: abnormal Q wave, elevated ST segment, and inverted T wave.

Stage 2 – *evolutionary*: deep Q wave, ST segment isoelectric, and inverted symmetrical T wave.

Stage 3 – *recovery*: deep Q wave, ST isoelectric, and T wave returning to normal.

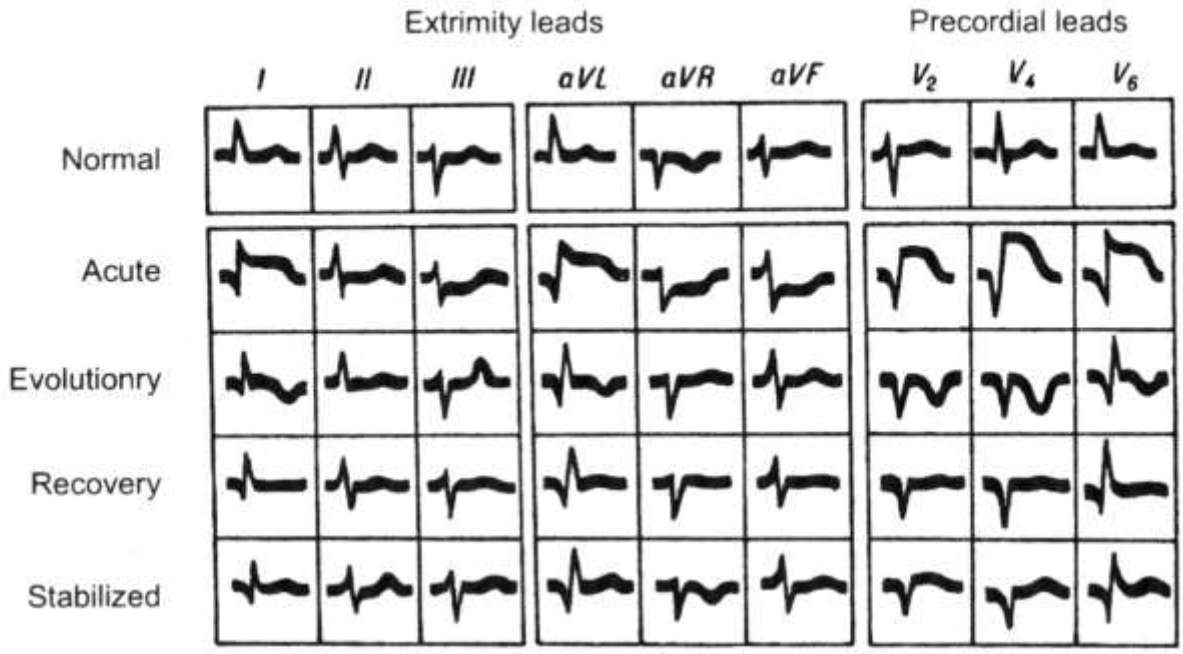


Fig 4.73. Evolution of myocardial infarction.

Stage 4 – *stabilized*: T waves normal, only evidence of old infarction is the deep Q wave.

Location of the myocardial infarction

Leads I, II, aVL – anterior infarction.

Leads III, II, aVF – posterior infarction.

V_1, V_2 – septal infarction.
V_3, V_4 – apical infarction.
V_5, V_6 – lateral infarction.

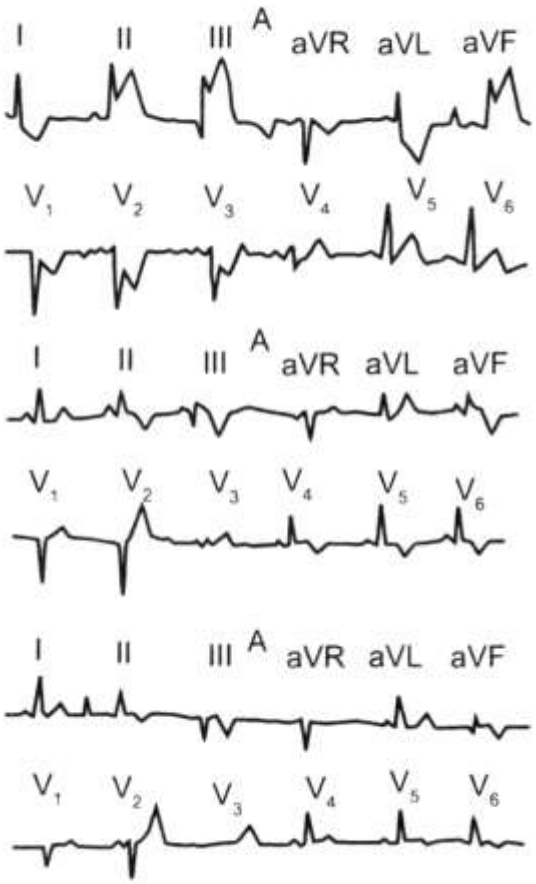


Fig. 4.74. Acute posterior myocardial infarction.

A, 12 hours after onset of pain. Markedly elevated S-T segment in leads II, III, and aVF; depressed ST in I, aVL, and V_1 through V_4.

B, 24 hours later. Less marked ST changes; deeply inverted T waves; Q

wave in II, III, and aVF; ST elevation from V_4 to V_6, and T wave inversion in these leads demonstrate lateral wall involvement with the infarction. The tall, peaked T wave in lead V_2 probably represents posterior ischemia.

C, 5 days later, shows evolutionary changes.

Cardiac Arrhythmias

Cardiac arrhythmias are diagnosed by noting changes in the regularity and the rate of the heart beats (Tab. 4.50). The three electrophysiologic properties of the heart are most intimately related to cardiac arrhythmias are automaticity, excitability, and conductivity.

Tab. 4.50. ***ECG signs of normal sinus rhythm and cardiac arrhythmias.***

Normal Sinus Rhythm	Cardiac Arrhythmias
1. The SA node is pacemaker. Heart rate: 60–80 beats per minute.	1. 60 > Heart rate > 90 beats per 140minute.
2. Regular cardiac rhythm: the R–R intervals are approximately equal (±10 %).	2. Irregular cardiac rhythm: the R–R intervals vary significantly (>10 %).
3. The P waves originate from SA node: most prominent upright R waves in leads II, III, aVF, may be inverted in aVR, or biphasic in leads V_1, V_2.	3. The P waves originate from a site outside SA node: changes of the P waves configuration (inverted, biphasic ±).
4. The relationship of the P wave and the QRS complex: all P waves are followed by QRS complex.	4. Changes of the relationship of the P wave and QRS complex.
5. The conduction to the ventricles is 1:1 (equal amount of P waves and QRS complexes).	5. The P waves amount are larger than QRS complexes.
6. Constant and normal duration of P wave, P–Q interval, QRS complex.	6. Changed the P wave, P–Q interval, and QRS complex duration.

Cardiac Arrhythmias

I. ***Abnormalities of the Impulse Formation***

A. *Altered Automaticity of the Sinoatrial Node (Nomotopic Arrhythmias).*

1. Sinus Tachycardia
2. Sinus Bradycardia
3. Sinus Arrhythmia
4. Sick Sinus Syndrome

B. *Increased Automaticity of an Ectopic Pacemaker*

1. Atrial Rhythm
2. Junctional (AV) Rhythm
3. Ventricular or Idioventricular Rhythm
4. Wandering pacemaker

C. *Ectopic (heterotopic) Arrhythmias caused by increased excitability of the myocardium*

1. Premature heart beat (contractions)
2. Paroxysmal Tachycardias
3. Atrial and Ventricular Flutter and Fibrilation.

II. ***Abnormalities of Conduction***

1. Sinoatrial Block
2. Atrioventricular Block
3. Intraventricular Blocks (His Bundle-Branch Blocks)
4. Ventricular Preexcitation Syndromes:
   - Wolff-Parkinson-White syndrome (WPW syndrome)
   - Shorted P-Q interval syndrome or Clerk-Levy-Critesco syndrome (CLC syndrome)

Abnormalities of the impulse formation

Altered automaticity of the sinoatrial node (nomotopic arrhythmias).

Sinus Tachycardia

In the sinus tachycardia in the adults, impulses are initiated in the SA node at a rate from 90 to 180 beats per minute (Fig. 4.75). Sinus tachycardia

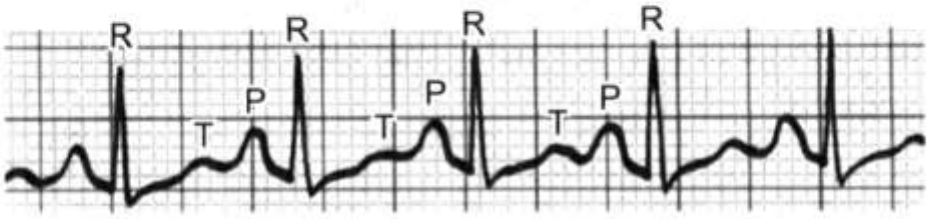


Fig. 4.75. Sinus tachycardia.

even at a very high rates is gradual, not sudden, in onset; if followed over a sufficient time, it will exhibit changes in rate of more than 10 beats per minute.

It may be seen in physical and emotional exertion, during meals, in alcohol, smoking, coffee, and tee abuse. Pathological conditions such as fever, sepsis, electrolyte disturbances, hemorrhage, shock, hyperthyroidism, cardiovascular diseases (myocarditis, heart valvular diseases, heart failure), and chronic pulmonary diseases as well as drugs such as atropine, epinephrine, and isoproterenol can produce sinus tachycardia.

Clinical symptoms of sinus tachycardia are heart palpitation and accelerated pulse.

Diagnostic ECG signs of sinus tachycardia

1. Decreased duration of the R–R intervals, increased heart rate to 90–180 per minute.
2. Regular sinus rhythm: the P waves are easily seen, upright in the leads I, II, aVF, V_4, V_5, V_6. The conduction to the ventricles is 1:1, the P–P and R-R intervals are constant.
3. The P–Q interval is 0.12 second or more.
4. The QRS complexes are normal.

Sinus Bradycardia

Sinus bradycardia is rhythm in which the ventricular rate customarily is less than 60 beats per minute (Fig. 4.76).

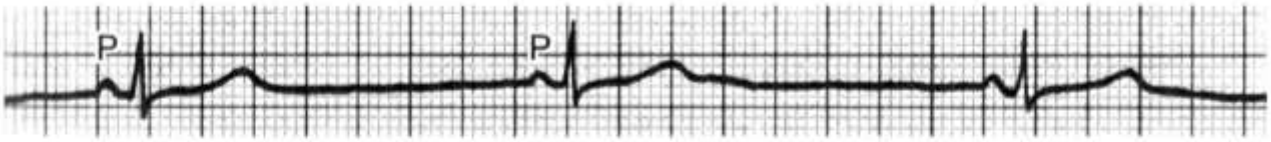


Fig. 4.76. Sinus bradycardia.

Sinus bradycardia is commonly seen in athletes, particularly marathon runners (HR accelerates during exercise), during night sleep, in stoppage of the breathing, and often in the aged. It may also occur with acute myocardial infarction, hypothyroidism, increased intracranial pressure, toxicosis, and with administration of drugs (digitalis, cardiac glycosides, β-adrenoblockers). Extreme sinus bradycardia may be caused by increased parasympathetic tone of any cause. Clinical signs of significant bradycardia (less than 40 beats per minute) are nausea, dizziness, syncope due to cerebral ischemia, and slow pulse rate (p. rarus, p. bradus).

Diagnostic ECG signs of sinus bradycardia

The impulse is initiated in the sinus node and spreads in normal sequence through the conducting system of the ventricles.

1. Increased duration of the R–R intervals, slow heart rate less than 60 beats per minute.
2. Regular sinus rhythm: the P waves are easily seen, upright in the leads I, II, aVF, V_4, V_5, V_6. The conduction to the ventricles is 1:1, the P–P and R–R intervals are constant.
3. The P–Q interval is between 0.12 and 0.20 second, although at very slow rates it may be slightly longer.
4. The QRS complexes are normal.

Sinus Arrhythmia

In sinus arrhythmia, the impulse is initiated in the sinus node, but the rate varies with respiration. During inspiration the rate increases; during expiration the rate slows (Fig. 4 77).

Fig. 4.77. Sinus arrhythmia, showing acceleration of heart rate with inspiration, and slowing with expiration.

Sinus (respiratory or juvenile) arrhythmia may be seen in children and adolescents. It may be caused by irregular impulse discharge from SA node and (or) changes of heart filling by blood during respiration.

Pathological conditions such as neurocirculatory dystonia by cardiac type, rheumocarditis as well as some infectious diseases can produce sinus arrhythmia. There are no clinical signs of sinus arrhythmia.

Diagnostic ECG signs of sinus arrhythmia

1. The P–P and R–R intervals vary significantly and rhythmically in such manner that the R–R intervals gradually shorten and lengthen with the respiratory cycles. The R–R intervals length may vary by 0.16 second or more.
2. Regular sinus rhythm: the P waves are upright in the leads I, II, aVF, V_4, V_5, V_6. The conduction to the ventricles is 1:1.
3. The P–Q intervals are constant.
5. The QRS complexes are normal.

Sick Sinus Syndrome

The term "Sick sinus syndrome" described in 1960 by American cardiologist Town, attributes the symptoms to the slow rate resulting from failure of impulse formation in the SA node or its conduction to the AV node.

Sick sinus syndrome includes a number of arrhythmias such as SA block with or without an ectopic or "escape" rhythms, marked bradycardia, atrial fibrillation, and attacks of tachycardia (bradycardia-tachycardia syndrome) (Fig. 4.78).

Such diseases as coronary heart disease, atherosclerosis, myocarditis, cardiomyopathy, cardiac or renal amyloidosis, diabetes mellitus, and toxicosis can caused sick sinus syndrome.

Clinical symptoms may be due to a very slow (dizziness, syncope) or a very rapid heartbeat (heart palpitation).

Diagnostic ECG signs of sick sinus syndrome

1. Constant bradycardia.
2. Periodic ectopic rhythms.
3. SA block.
4. Bradycardia-tachycardia syndrome.

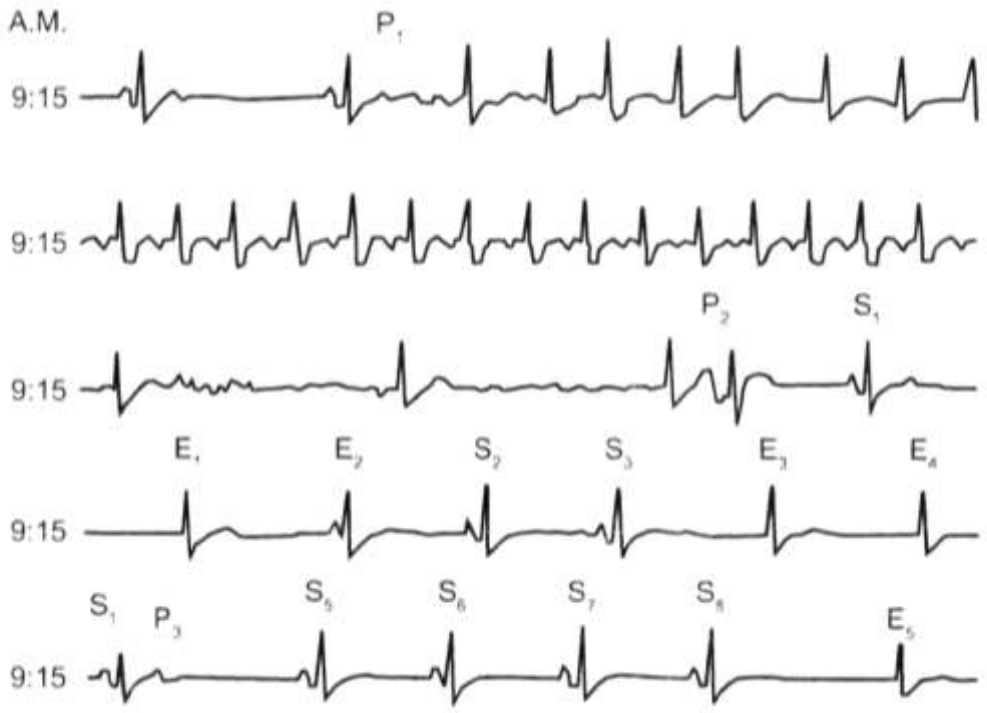


Fig. 4.78. Sick sinus syndrome (monitor leads).

Note particularly, the alternating tachycardia and bradycardia, which occurred spontaneously. In the top strip, taken at 9:15, sudden onset of atrial fibrillation occurred when the ectopic atrial beat (P_1) appeared. In the second strip, taken at 9:20, the rhythm is atrial flutter with 2:1 block. In the following three continuous strips, taken at 9:30, the rhythm is initially atrial fibrillation with very slow escape rhythm, probably lower junctional in origin; succeeded by a conducted atrial premature contraction (P_2), a sinus beat (S_1), then two escape beats (E_1 and E_2); two sinus beats (S_2, S_3), two escape beats (E_3, E_4) followed by a sinus beat (S_4), a blocked atrial premature contraction (P_3), since the expected escape beat does not occur on time; the last sinus beats (S_5 through S_8) with an escape beat (E_5) that occurred at the expected time.

Increased Automaticity of an Ectopic Pacemaker

Arrhythmias occurring from an ectopic pacemaker may be active or passive.

Active arrhythmias develop because of increased automaticity of an ectopic pacemaker.

Passive arrhythmias occur because an established pacemaker fails to discharge at its usual rate, allowing a slow subsidiary pacemaker to emerge. Passive rhythms protect the heart from a long asystole period.

Atrial, junctional and ventricular rhythms are distinguished.

Atrial Rhythm

In atrial rhythm the pacemaker lies within the atria resulting in abnormal direction of their depolarization. The direction of ventricular activation is normal (Fig. 4.79).

Diagnostic ECG signs of the atrial rhythm

1. Heart rate is from 60 to 90 beats per minute.
2. Inverted P wave in leads II, III, aVF within an upright P wave in lead aVR or biphasic P wave depend on the site of the pacemaker.
3. The P–Q interval is normal.
4. The QRS configuration is normal.

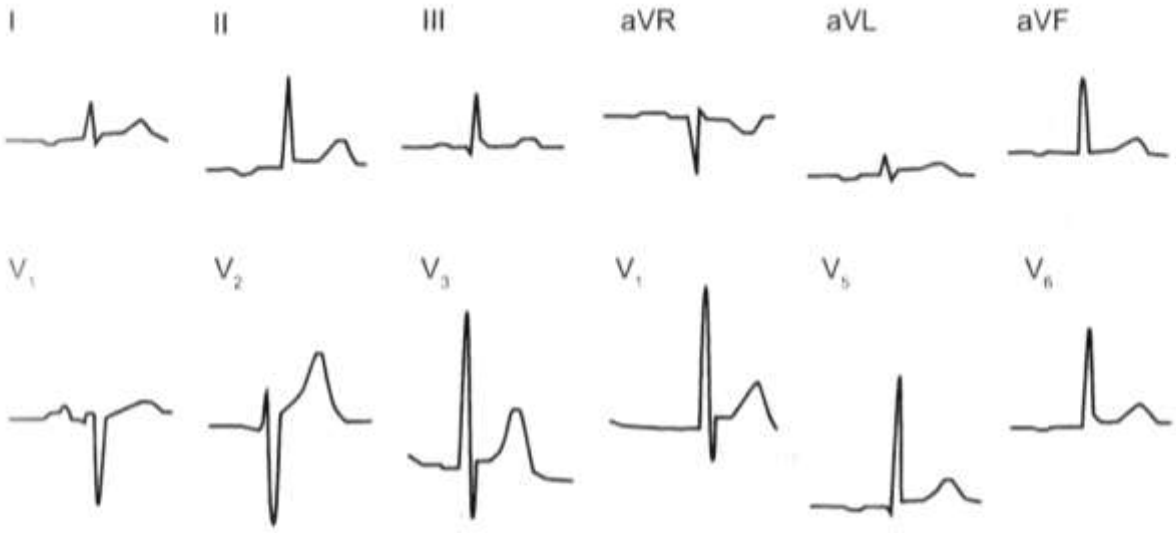


Fig. 4.79. Left atrial rhythm.

Note the negative P waves in leads I and V_6 and the "dome and dart" P waves in V_1. The dome-and-dart P wave is characterized by an initial smooth, slowly rising positive wave punctuated by high amplitude, spiked, terminal positive component.

Junctional (AV) Rhythm

A junctional rhythm is found in acute rheumatic carditis, acute myocardial infarction, and with digitalis intoxication.

If the junctional pacemaker depolarized the atria in retrograde fashion, an inverted P wave may be seen. The QRS complex occurs after a longer pause than normal. The QRS complex is usually normal, although it may show slight aberration (Fig. 4.80).

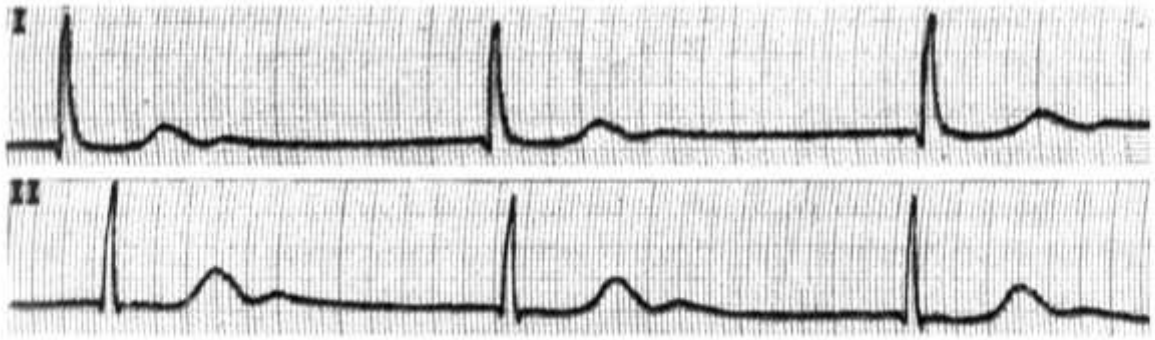


Fig. 4.80. Junctional rhythm. The rhythm is regular.
No P waves are visible.

Diagnostic ECG signs of junctional rhythm

1. Heart rate is 40–60 beats per minute.
2. An inverted P wave in leads II, III, and aVF may be seen just preceding or following the QRS complexes; sometimes no P wave is seen.
3. The P–Q interval duration is more than 0.2 second.
4. The QRS complex is usually normal.

Ventricular or Idioventricular Rhythm

Occasionally, the sinus pacemaker is suppressed, and the junctional pacemaker fails to respond. Under these circumstances, an idioventricular pacemaker may discharge to maintain cardiac function. When such beats occur in succession, an idioventricular rhythm is initiated.

Idioventricular rhythm most commonly observes in complete AV block.

The course of ventricular activation is abnormal, and QRS complex is therefore wide and of abnormal configuration. The sinus P wave may be seen, but it has no relationship to the QRS complex.

Diagnostic ECG signs of ventricular rhythm

1. Heart rate is less than 40 beats per minute.
2. The sinus P wave or its absence. There is no relationship of P wave to the QRS complex.
3. Wide and deformed QRS complex.

Wandering Pacemaker

The term wandering pacemaker refers to conditions where in the primary pacemaker of the heart is presumed to move from site to site in the atrium. This may vary from areas near the SA node to areas near AV node. The rhythm is only slightly irregular and the rate is essentially unchanged. The P wave morphology changes from beat to beat, varying from peaked to flat to slightly inverted (Fig. 4.81).

Diagnostic ECG signs of the wandering pacemaker

1. The P wave configuration changes from cycle to cycle.
2. Changes of the P–Q interval duration depend on the site of pacemaker.
3. The cardiac rhythm is slightly irregular, different R–R intervals.
4. The QRS complex morphology is normal.

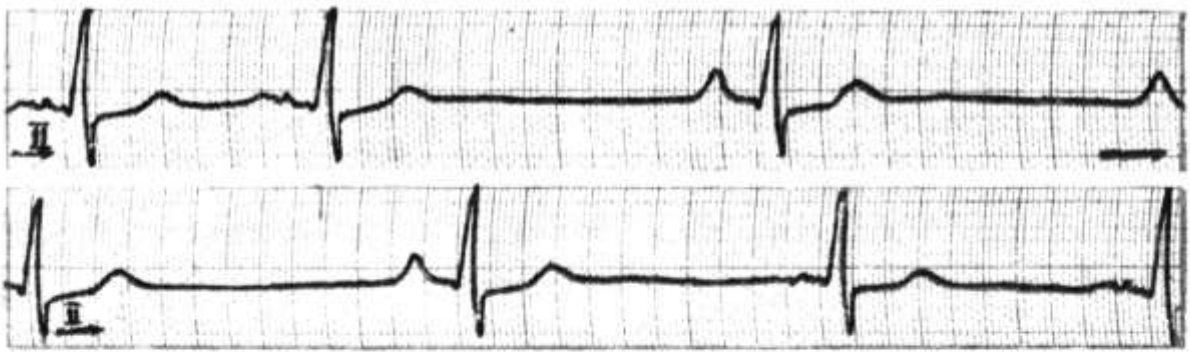


Fig. 4.81. Wandering atrial pacemaker.
A, note the different contour of the P waves (arrows) with constant P–Q intervals; **B,** note the varying P–Q intervals with changing contour of the P waves. The 5th arrow points to a P wave that is not visible because the QRS complex is superimposed on it.

Ectopic Arrhythmias Caused by Increased Excitability of the Myocardium.

Ectopic arrhythmias may be due to increased automaticity of an ectopic pacemaker or reentry mechanism. Reentry or circus mechanisms occur when an impulse returns to its site of origin and reenters the conduction pathway in its nonerefractory state (Fig. 4.82). For reentry to occur, an impulse must enter an area of myocardium and divide into two branches. One branch must be in its refractory state. The impulse cannot pass through this refractory branch; however, through circuitous paths, the impulse returns to its point of origin and reenters the same nonrefractory pathway. Reentry requires a slight prolongation of the refractory period in some area of the heart, so the impulse is blocked in one direction but allowed to gain access from another direction.

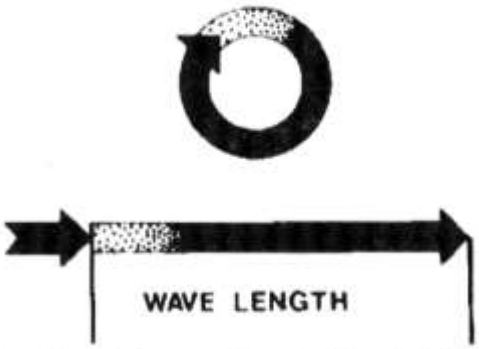


Fig. 4.82. Schematic drawing of a reentrant circuit. The black area represents tissue, which is in its unexcitable phase, whereas the dotted area indicates relatively refractory tissue. No excitable gap exists between head and tail of the excitation wave.

An ectopic impulse may arise in the atria, junctional tissue, or ventricles. Junctional premature contractions are the least common; atrial and ventricular premature beats are quite common.

Premature heart contractions can observe in normal individuals: emotional exertion, in heavy smokers, in coffee, strong tee, and alcohol abuse. Coronary heart disease (most commonly acute myocardial infarction), hypertension, rheumatic heart valvular diseases, myocarditis, congestive heart failure cause premature contractions as well as by reflex in the diseases of the abdominal organs premature beats can arise. Premature heartbeats can also occur in hyperthyroidism, menopause, and digitalis intoxication.

Clinical signs: Patients with premature cardiac contractions can feel their heart missing a beat (escape beat) and subsequent strong stroke. In auscultation premature beat with a specific loud first sound is heard. Study of the arterial pulse reveals premature weak pulse wave following by long pause. If premature beat is close to the normal one the pulse wave does not reach radial artery causing pulse deficit.

Premature Atrial Contraction

Atrial premature contractions (APCs) may arise from either atrium. A P wave is always present and occurs earlier than the normal sinus P wave. The premature P wave varies in configuration depending on its site of origin or reentrant mechanism. If the impulse originates in or near the SA node and depolarization follows the usual course, the P wave will resemble the normal P wave. If the ectopic atrial site is near the AV node, atrial activation will occur retrogradely and will be directed toward the SA node, giving rise to an inverted P wave (Fig. 4.83).

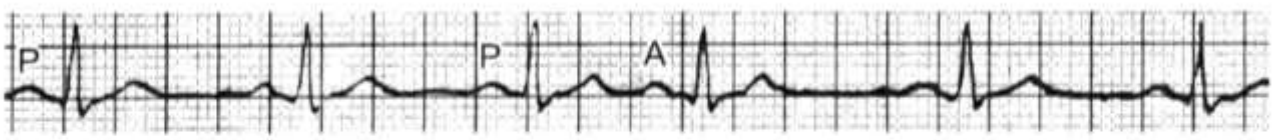


Fig. 4.83. Atrial premature contraction (A).
The P wave (A) occurs early, it is of different configuration; the QRS-T wave following it is normal appearance, and the compensatory pause is incomplete.

After an APC, there is a pause before the next sinus beat. This is usually incomplete compensatory pause (Fig. 4.84).

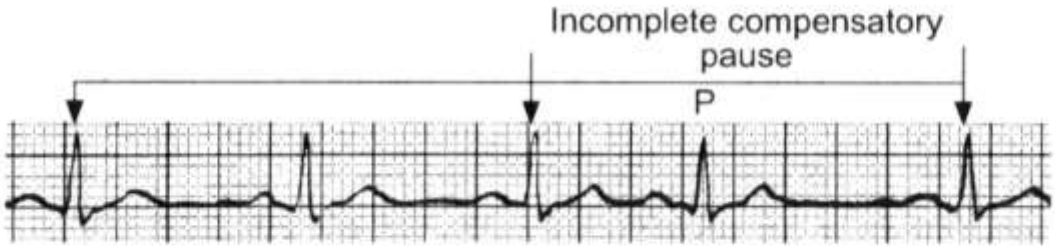


Fig. 4.84. Mechanism of compensatory pause following atrial extrasystole.

The atrial premature contraction not only activates the ventricles but also is able to spread retrograde direction and to discharge the sinus node prematurely. The premature discharge of the sinus pacemaker causes transient depression of its rhythmicity so that the sinus cycle immediately following the ARC is slightly longer than the usual sinus cycle. However, the lengthening of the postectopic sinus cycle is usually less in amount than the preceding cycle is shortened by the atrial premature beat. Consequently, the interval between the last R wave preceding the APC and the first R wave following the extrasystole is less than two cycle lengths of the sinus rhythm – in other words, the postectopic pause is incomplete compensatory (Fig 4.86).

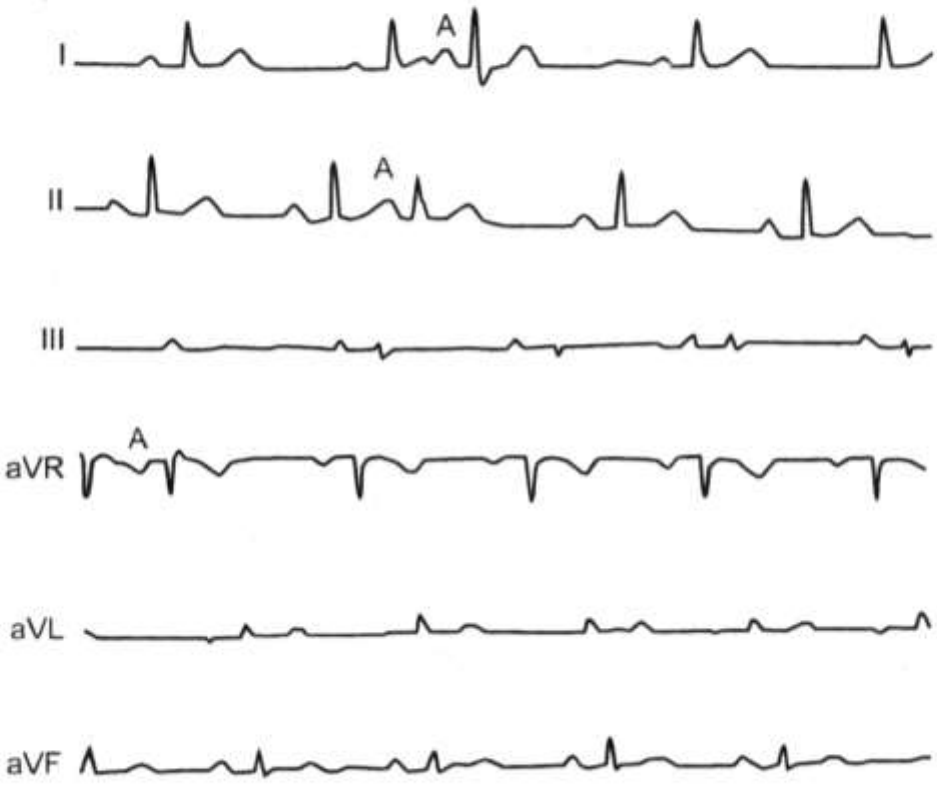


Fig. 4.85. Premature atrial contractions (A).

ECG signs of atrial premature contractions

1. Premature appearance of the P wave following by the QRS complex.
2. Changes of the P wave polarity depend on the site of the ectopic focus.
3. Normal configuration of the QRST complex.
4. Presence of the incomplete compensatory pause after the atrial premature contraction.

Junctional Premature Contractions.

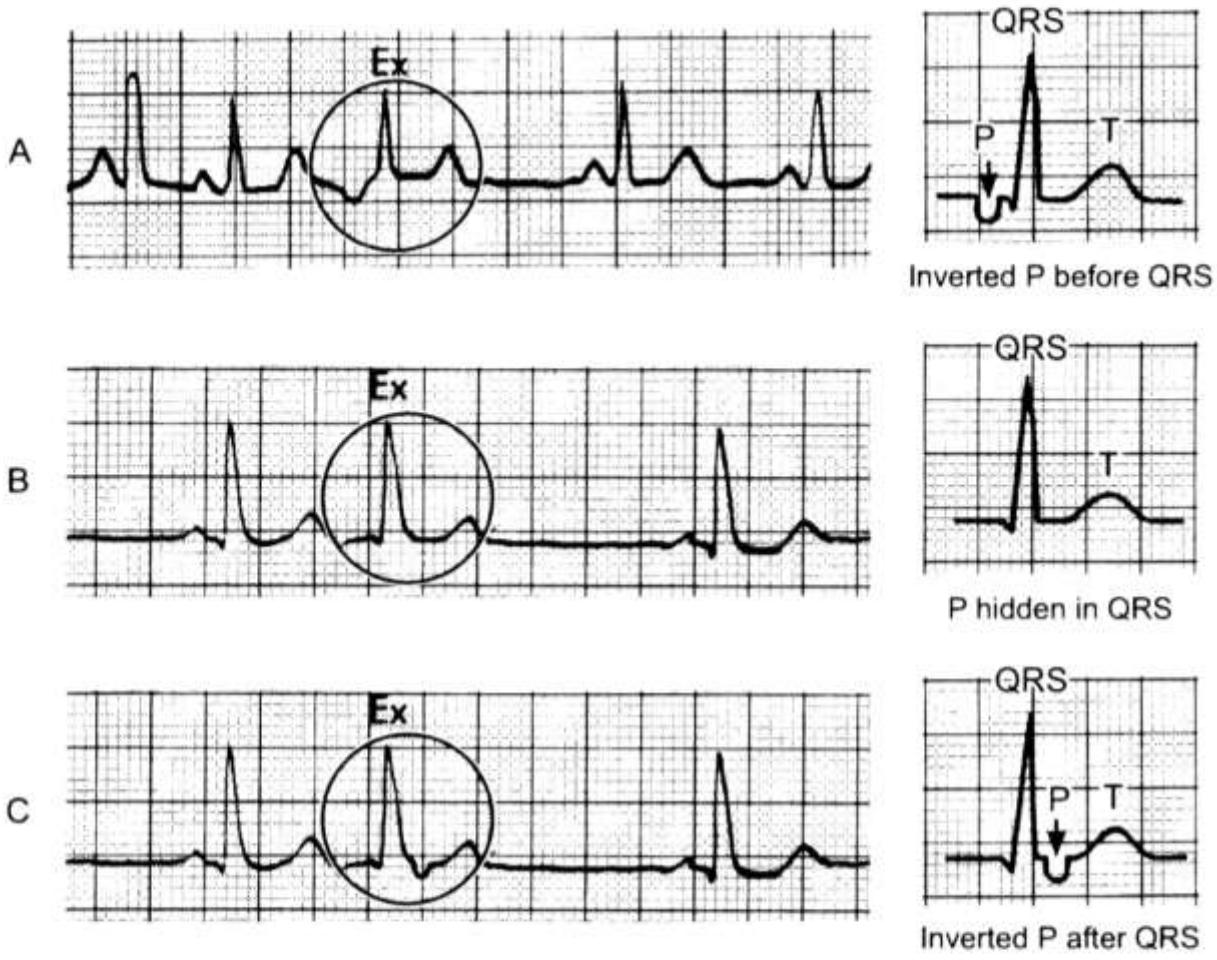


Fig. 4.86. Junctional premature contractions.
A – an upper nodal extrasystole; the P wave is inverted before QRS complex, the QRS complex resembles the others, and the compensatory pause is incomplete.
B – a midnodal extrasystole; the P wave cannot be identified, the QRS complex resembles the others, and the compensatory pause is incomplete.
C- a lower nodal extrasystole; the P wave is inverted after QRS complex, the QRS complex resembles the others, and the compensatory pause is incomplete.

In junctional premature contraction the pacemaker is in the AV node. The P wave may or not may be identified. The QRS complex has the same morphology as the QRS complex of the normal sinus complex in the same lead because ventricles are activated by usual pathways. When there is retrograde conduction into the atria, the inverted P waves are present, which occur before, after, or within the QRS complex. If it occurs simultaneously with the ORS complex, it may be buried in the QRS complex and not visible, although it may deform the complex somewhat. The atrial impulse is normally delayed in the junctional tissues, a delay, which accounts for most of the duration of the P–Q interval. If no delay in retrograde or antegrade conduction occurs, then the ectopic focus in the junctional tissue is located above the site of delay, and the inverted P waves will precede the QRS complex (formerly called an upper nodal extrasystole). If the ectopic focus is below the site of junctional delay, retrograde atrial activation will be delayed and the P wave will coincide with the QRS complex (formerly called a midnodal extrasystole). The P way may follow the QRS, (formerly called a lower nodal extrasystole) (Fig. 4.86).

ECG signs of the junctional premature contractions.

1. Premature appearance of the normal QRS complex.
2. Inverted P wave before, after, or within the QRS complex.
3. Presence of incomplete compensatory pause.

Ventricular Premature Contractions.

As the name denotes, premature ventricular contractions (PVCs) originate in the ventricles below the AV node. Because the PVCs do not follow the normal conduction path in the ventricles, they show a bizarre QRS configuration on the ECG. It is characterized by being wide, slurred, and notched. An ectopic impulse arising near the bundle of His may produce a relatively normal-appearing QRS. The QRS complexes of PVCs are not preceded by P wave because impulse cannot passes retrogradely through the AV node to cause atrial activation. The T wave is usually opposite in direction to the main deflection of the QRS. Usually the sinus node maintains control of the atria and sinus-initiated P wave may occur in the T wave of the premature ventricular beat. Since the ventricle is refractory at this time, there is no conduction to the ventricle from this sinus impulse, and a com-

pensatory pause occurs. Since there has been no premature discharge of the sinus node and the sinus rhythm is undisturbed, the interval between the normally conducted beats that surround the premature ventricular beat is exactly twice the normal R–R interval. This is termed a complete compensatory pause (Fig. 4.87).

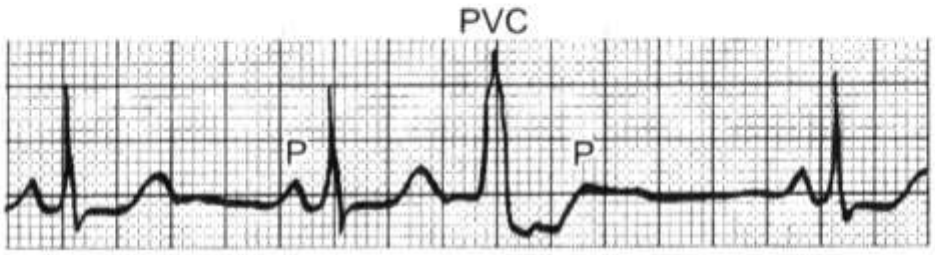


Fig. 4.87. Ventricular premature contraction (PVC).
The QRS–T complex has a bizarre shape, being wide and slurred; the P wave following occurs at the proper interval after the preceding P wave; the compensatory pause is complete.

Occasionally the atrial depolarization occurring during the ventricular premature contraction does not succeed in traversing the AV node and succeeds in the depolarizing the ventricle. Under these circumstances, there is no compensatory pause. The PVC does not interrupt the sinus rhythm. This type of PVC is called interpolated PVC (Fig. 4.88).

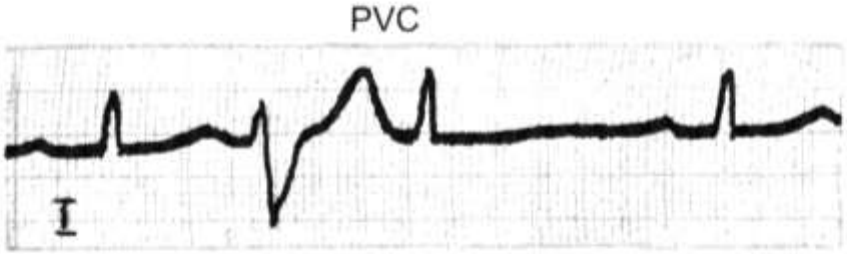


Fig. 4.88. Interpolated premature ventricular contraction (PVC), occurring between two normal beats without compensatory pause.

If the ectopic impulse originates in the right ventricle, the impulse spreads slowly from the right ventricle to the left, producing high amplitude R wave in leads I, aVL, V_4, V_5, V_6 and deep S wave in leads III, aVF, V_1, V_2, V_3. An impulse originating in the left ventricle spreads slowly to the right ventricle to produce high amplitude R wave in leads III, aVF, V_1, V_2, V_3 and deep S wave in leads I, aVL, V_4, V_5, V_6.

ECG signs of PVCs.

1. Premature appearance of significantly wide and distorted QRS complex.
2. The S–T segment and T wave of PVC opposite in direction to the main deflection of the QRS complex.
3. The QRS complexes of PVCs are not preceded by a P wave.
4. Presence of the incomplete compensatory pause after PVCs.

PVCs from the same site have the same configuration in the same lead, and is called – ***unifocal PVCs*** (Fig. 4.89).

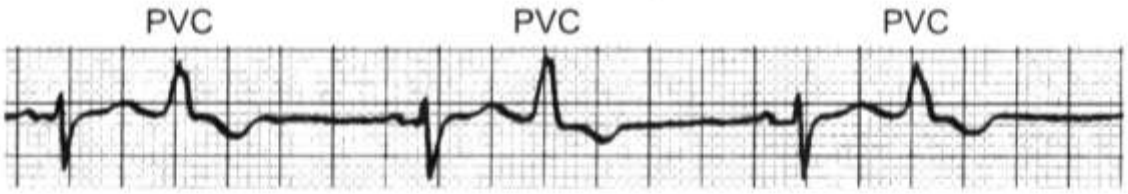


Fig. 4.89. Unifocal premature ventricular contractions, bigeminy is present. Note the same configuration of PVCs.

PVCs that originate from the different site have different configuration when recorded in the same lead, and is called – ***multifocal PVCs*** (Fig. 4.90).

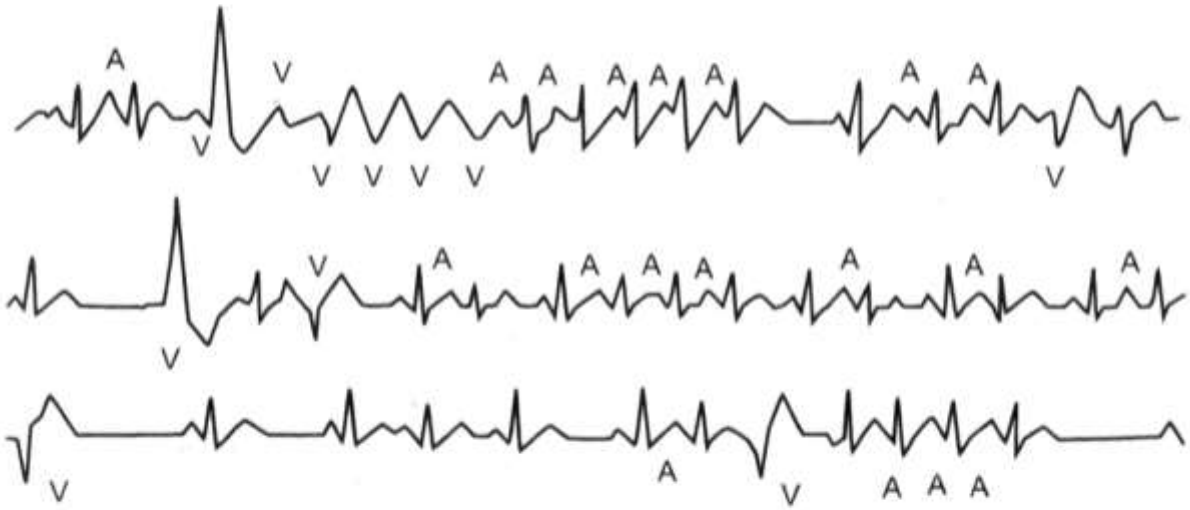


Fig. 4.90. Multifocal premature ventricular contractions (V). Continuous strip of lead II show premature beats from multiple foci. Not also multifocal atrial baets (A)

Ventricular ***bigeminy*** occurs when PVCs alternate with normal contractions (Fig. 4.89).

Trigeminy is a term applied to a grouping of the PVCs in runs of three, such as two normal beats followed by a PVC (Fig. 4.90), or a normal beat is followed by two PVCs (Fig. 4.91).

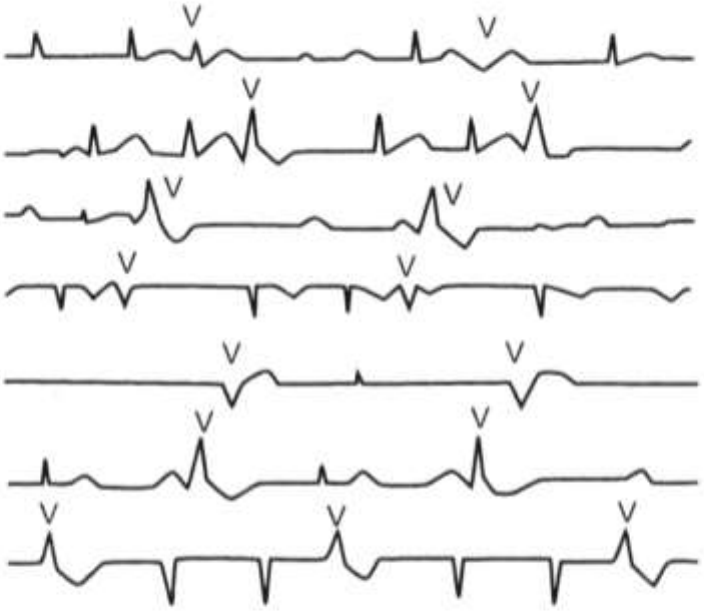


Fig. 4.91. Ventricular trigeminy. Premature ventricular contractions (V) from a single focus occurring regularly after two normal beats.

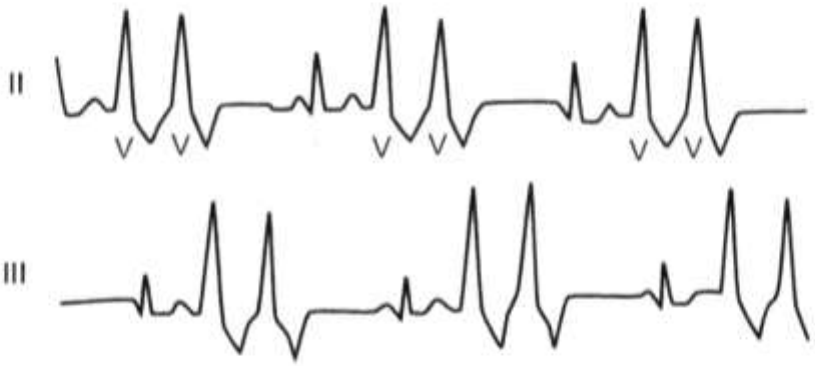


Fig. 4.92. Ventricular trigeminy. Sinus rhythm with frequent ventricular premature contractions, producing a trigeminal rhythm consisting of one sinus beat followed by two consecutive ventricular premature beats.

Group PVCs occur in groups of two or three.

Premature ventricular contractions are especially dangerous when they:

1. Occur more frequently than one in 10 beats.
2. Occur in groups of two or more.

3. Are multifocal: Several ventricular sites are irritable.
4. Occur on or near the T wave. At this time in the cycle, the conduction tissue is partially repolarized and may respond in an erratic manner. Some cells respond immediately and other later, causing intermittent depolarization and triggering ventricular fibrillation.

Paroxysmal Tachycardias

Depend on ectopic site location atrial, junctional, and ventricular paroxysmal tachycardias are distinguished. Conventionally, however, paroxysmal atrial and junctional tachycardias refer to supraventricular paroxysmal tachycardias.

Supraventricular paroxysmal tachycardia

Supraventricular paroxysmal tachycardia is an arrhythmia characterized by abrupt onset and cessation. The ectopic impulse is initiated in either atrium or junctional tissue, and the rate of discharge is usually 140 to 220 beats per minute. The ventricular response is 1:1, with normal-appearing QRS complex. When the impulse arises in the atria, the QRS complex is preceded by abnormal P wave and the rhythm is termed paroxysmal atrial tachycardia. When the ectopic focus or reentry site lies in the junctional tissue, the QRS complex may precede, follow, or occur simultaneously with the P waves. The P waves, when seen, are produced by retrograde conduction, and are usually inverted. This rhythm is called paroxysmal AV or junctional tachycardia. In many cases of paroxysmal atrial or junctional tachycardia, the P waves are not well seen, and the general term supraventricular is used (Fig 4.93).

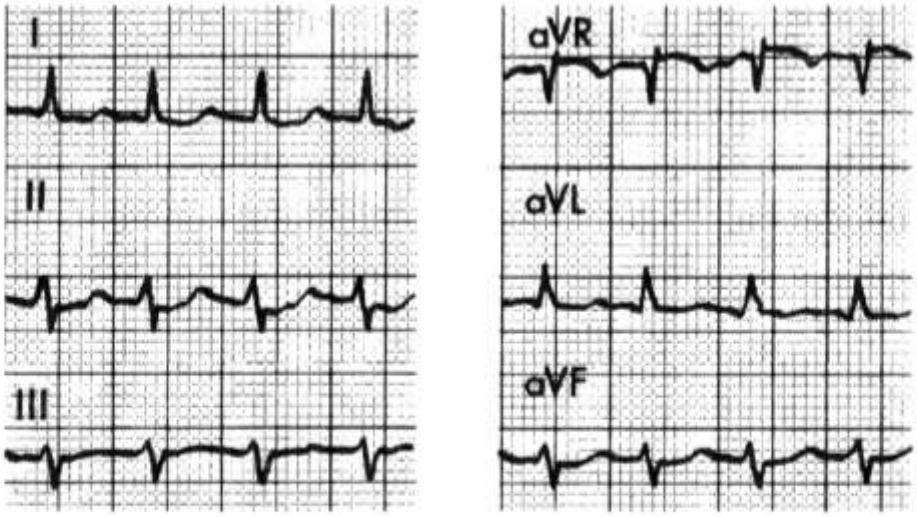


Fig. 4.93. Supraventricular tachycardia.
The P and T waves are superimposed.

ECG signs of paroxysmal supraventricular tachycardia.

1. Sudden acceleration of heart rate 140 to 220 beats per minute.
2. The QRS complexes configuration is normal.
3. Inverted P wave before, after, or simultaneously with QRS complex.

Paroxysmal Ventricular Tachycardia

Ventricular tachycardia is a cardiac arrhythmia originating in the ventricles at a rate of 100 beats per minute or faster. A minimum of three consecutive beats is required to establish this diagnosis. Paroxysmal ventricular tachycardia starts and stops abruptly; it is usually regular rhythm, but may show slight irregularities of ventricular response (Fig. 4.94).

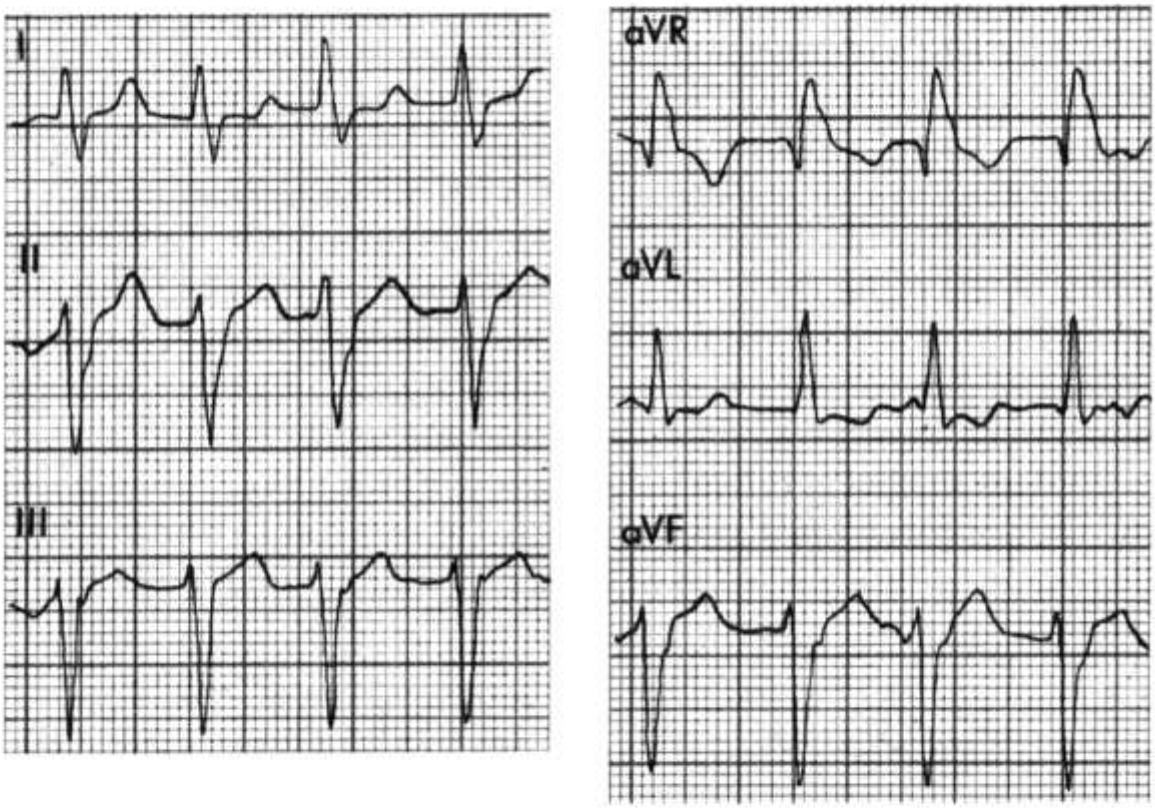


Fig. 4.94. Paroxysmal ventricular tachycardia.

ECG signs of paroxysmal ventricular tachycardia.

1. Abrupt starts and stops of the heart rate acceleration 100 to 220 beats per minute in regular rhythm.
2. Deformation and broadening of the QRS complex with opposite to the main QRS deflection S-T segment and T wave.
3. AV dissociation (independent atrial and ventricular rhythms).

Ventricular Flutter and Fibrillation.

Ventricular flutter is characterized by fairy regular, oscillating waves of large amplitude with no isoelectric interval, and a rate between 200–300 cycles per minute. In ***ventricular fibrillation***, electrical and mechanical activity of the heart is totally disorganized. There is no cardiac output; clinical cardiac arrest is present. The ECG is bizarre, with complete absence of the characteristic P, QRS, and T waves; it shows irregular waves of varying amplitude and shape occurring 250 to 500 times per minute (Fig. 4.95). Both rhythms usually result in death unless they are quickly converted by drugs or electrical defibrillation.

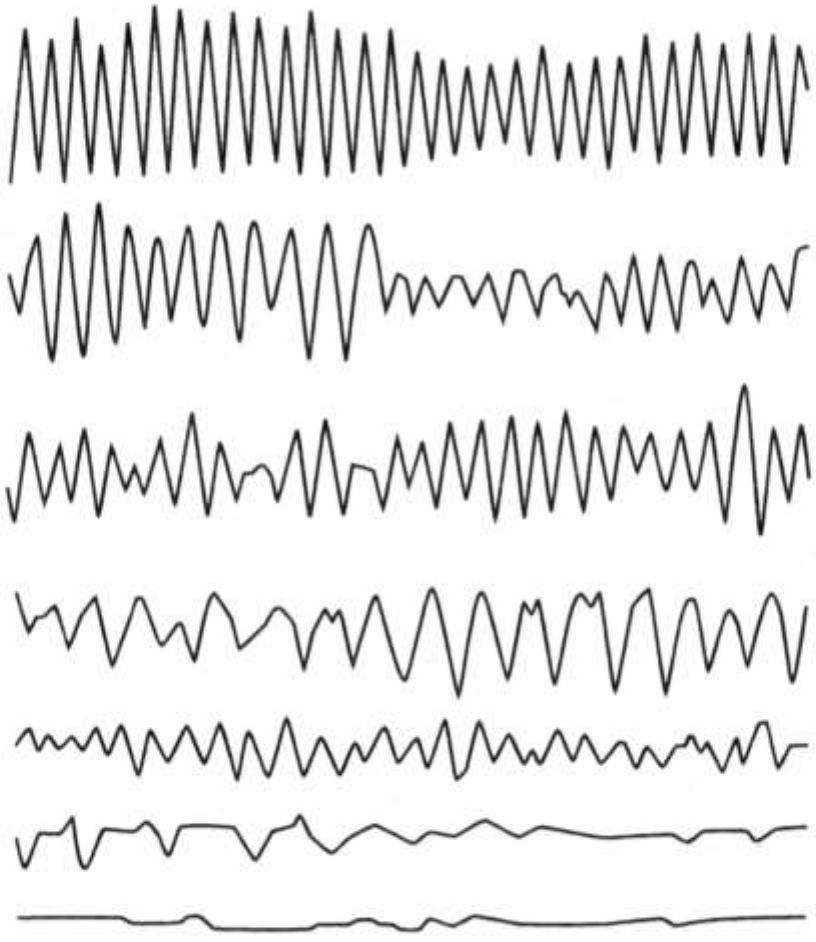


Fig. 4.95. Dying heart.
Continuous strip of lead II. Note onset in top lead episode of ventricular flutter with regular oscillatory waves (no isoelectric baseline or T waves identified) changing to irregular undulating waves characteristic of ventricular fibrillation, ending with a straight line.

Atrial Flutter

Atrial flutter may result from a rapid series of impulses arising from a single ectopic focus, a series of impulses from multiple ectopic atrial foci, or a circus movement. The atrial rate is usually between 240 and 350 beats per minute.

The ECG in atrial flutter is usually characterized by a uniformly appearing series of flutter waves (F waves), often described as resembling a picket fence, or as having a saw-toothed appearance. These waves are usually best seen in leads II, III, aVF, and V_1. There is a sharp upstroke with a more gradual down-stroke, and no isoelectric interval exists between the waves (Fig. 4.96).

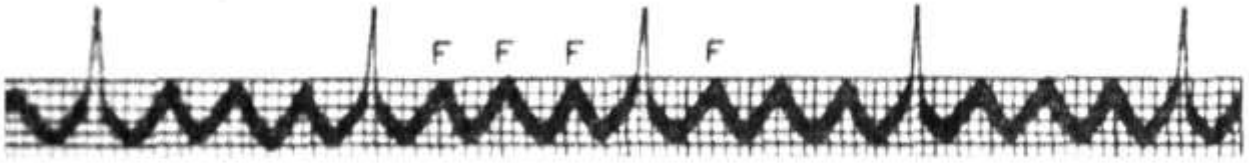


Fig. 4.96. Atrial flutter with 3:1 conduction. The atrial rate is 300 beats per minute and the ventricular rate is 75 beats per minute. Note the saw-toothed baseline due to the flutter waves.

At rest, the AV node will not allow more than 150–180 impulses per minute to pass. Therefore, when atrial flutter is present in its untreated state, the conduction to the ventricles is blocked (2:1, 3:1, 4:1 ets).

ECG signs of atrial flutter.

1. Presence of rapid (150–180 per minute) regular, uniform atrial F waves, which are best seen in leads II, III, aVF, V_1 (Fig 4.99)
2. Regular ventricular rhythm with equal R–R intervals.
3. The QRS complex is usually normal in configuration.
4. The conduction to the ventricles is 2:1 or 3:1, or 4:1 ets.

Atrial Fibrillation

In atrial fibrillation the atria are beating very rapidly and irregularly. The rate is between 350 and 700 beats per minute. Most of these impulses are blocked in the AV node, so the ventricular rate is much slower, but totally irregular. Many impulses reach the junctional tissue while it is still refractory from preceding impulses. Actually, some impulses may not reach the junctional tissue at all, and the impulses that do arrive and conduct to the ventricle do so quite irregularity, giving rise to rapid and very irregular ven-

tricular response. Usually the ventricular rate is between 110 and 160. There are no definite P waves; there are, instead, continuous irregular undulations of the baseline of varying amplitude, spacing, and contour. These are known as fibrillation waves (f waves). The QRS complexes have relatively normal configuration (Fig. 4.97).

Fig. 4.97. Atrial fibrillation with an irregular ventricular beat. Note that the normal contractions of the atria replaced by irregular oscillations.

ECG signs of atrial fibrillation.

1. Instead P wave irregular undulations of varying shape and amplitude (f waves), which are best seen in leads II, III, aVF, V_1.
2. Irregular ventricular rhythm with different R–R intervals.
3. The QRS complexes have relatively normal configuration.

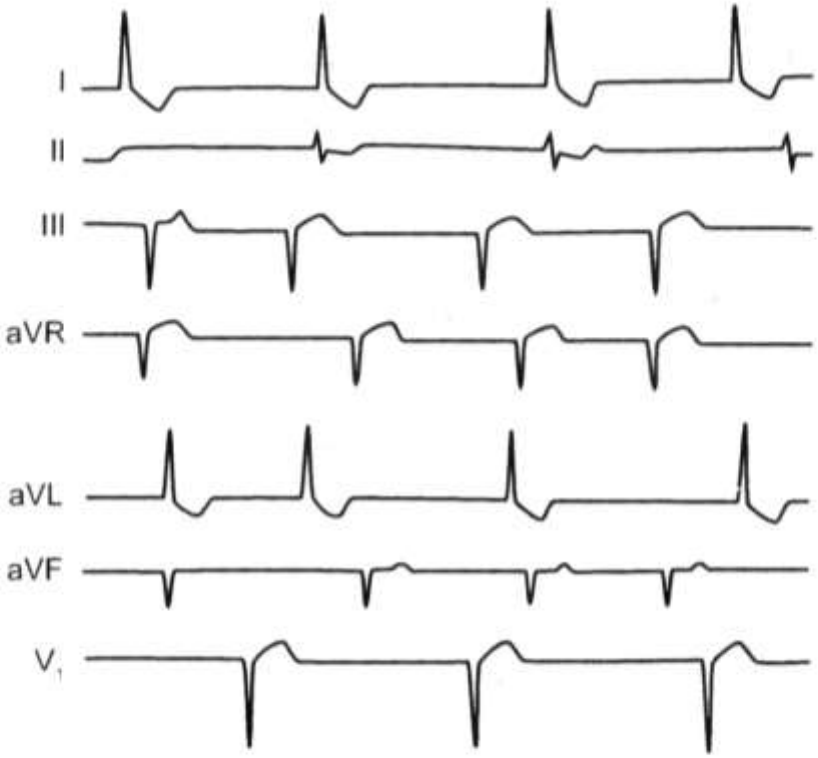


Fig. 4.98. Atrial fibrillation with a slow, irregular ventricular rate of 42 to 70 beats per minute. Note the fairly continuous undulating fibrillary waves, varying in size, shape, and spacing.

Abnormalities of Conduction. Heart Blocks

Heart block is an electrocardiographic diagnosis arising from situations that delay or interrupt the passage of an electrical impulse into any part of conduction system of the heart. Incomplete block indicates defect, functional or organic, in the conduction system that slows, but does not interrupt, the transmission of the impulses. Complete heart block indicates defect in the conduction system that interrupt the transmission of the electrical impulses.

Depending on the site of conduction abnormalities the following heart blocks are distinguished:

1. Sinoatrial Block.
2. Atrioventricular Block.
3. Intraventricular Block.

Sinoatrial Block

Sinoatrial (SA) block indicates failure of the sinus node to form impulses or impaired conduction of the impulse from sinus node to the atrial myocardium. Since the sinus impulse itself produces no ECG deflection, a sinus block is indirectly diagnosed by the absence of one or more expected P wave with the associated QRS complex (Fig. 4.99).

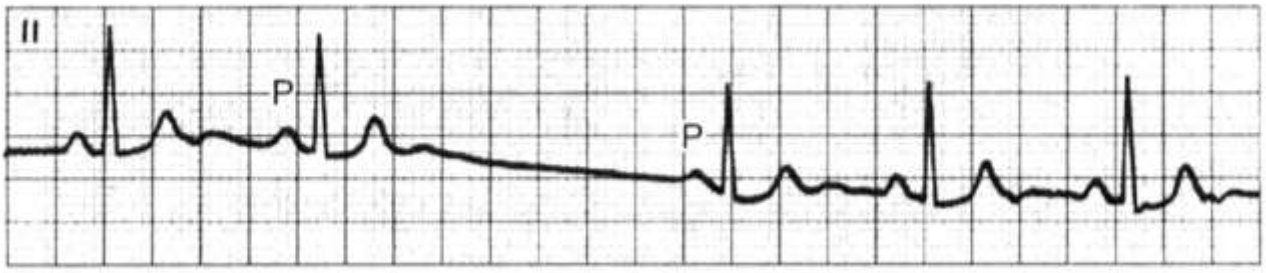


Fig. 4.99. Sinoatrial block.
The normal impulse is formed within the SA node, but is not conducted to the atrium. The regular sinus rhythm is present, after which there is a pause during which no P-QRS-T complex occurs. The pause is double the R–R interval of the beats displaying sinus rhythm.

Sinus block occur most frequently in patients with increased vagal tone and often during acute diaphragmatic myocardial infarction. Ischemia, hemorrahage, rheumatic fever, diphtheria, other acute infections, and drug toxicity (digitalis, quinidine, atropine, salicylates) may also cause SA block.

ECG signs of the sinoatrial block.

1. Periodic missing of the separate cardiac cycle (P wave and QRST complex) in the regular sinus rhythm.
2. The pause is double the P–P or R–R interval of the beats displaying sinus rhythm.

Atrioventricular Block

Atrioventricular block (AV block) is an important and frequent cause of slow rhythms. The greatest delay in normal transmission of an impulse from the atria to the ventricles occurs in the AV junctional tissues. AV blocks observe in the patients with atherosclerosis, coronary heart disease with acute myocardial infarction, rheumocarditis, and in drug toxicity.

By tradition, AV block has been divided into three degrees of block, depending on changes in the P–Q interval and relationship between the P wave and QRS complex.

The First-Degree AV Block

In the first-degree AV block the P–Q interval is prolonged over 0.21 second, but all sinus impulses are conducted to the ventricles: every sinus beat (P wave) is followed by a ventricular complex QRS. Since the ventricles are activated in the usual manner, the QRS complex is normal in configuration (Fig. 4.100). Ordinarily, the P–Q interval is constant at a given heart rate. In the normal heart, the P–Q interval tends to shorten as the rate increases. When some forms of conduction disturbances are present, the P–Q interval lengthens as the heart rate increases.

ECG signs of the first-degree AV block.

1. Prolonged P–Q interval to more than 0.21 second.
2. The QRS complexes normal in configuration.

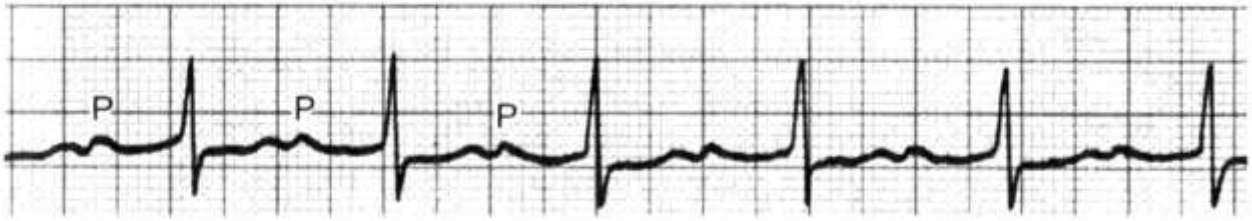


Fig. 4.100. First-degree AV block. The P–Q interval is prolonged.

First-degree AV block does not diminish cardiac output. However, it is an indicator of possible damage to junctional tissue or drug effect, especially from digitalis.

Second-Degree AV Block

In second-degree AV block, some impulses are blocked and fail to reach the ventricles (some P wave are not followed by a QRS complex). The more atrial impulses blocked from reaching the ventricles, the slower the ventricular rate. Thus, second-degree AV block often causes bradycardia.

Three types of second-degree AV block have been described: Mobitz type I, Mobitz type II, and type III.

Mobitz (Wenchenbach) type I

In type I second-degree heart block involving AV node, the P–Q interval progressively lengthens until the atrial impulse fails to conduct to the ventricles (P wave not followed by QRS complex), and then the cycle repeats. The ECG sequence starting with the first conducted beat following by the ventricular pause, and ending with the next blocked atrial beat, constitutes a Samoilov-Wenchenbach period. The basic principle of the Wenchebach phenomenon is that conduction time progressively lengthens until it is blocked for a one beat, producing a pause. Following the pause the conduction time shortens and then progressively lengthens again. Since the ventricles are activated in the usual manner, the QRS complex is normal in configuration (Fig. 4.101).

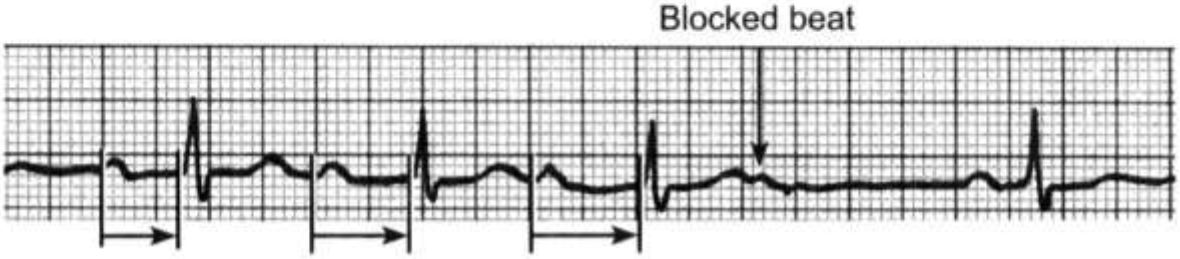


Fig. 4.101. Second-degree AV block Mobitz type I illustrating the Samoilov-Wenchenbach period. After each blocked atrial beat the P-Q interval shortens, to lengthen again progressively until the next blocked beat.

ECG signs of Mobitz type I second-degree AV block.

1. The P–Q interval is progressively prolonged until P wave is completely blocked.
2. The R–R interval is progressively shortened until the block occurs.

3. The P–Q interval after the blocked impulse is shorter than P–Q interval before the blocked impulse.
4. The R–R interval following block impulse is longer than the R–R interval preceding the blocked impulse.
5. The long interval due to the blocked impulse is less than twice the preceding R–R interval.
6. The QRS complex is normal in configuration.

Mobitz type II

In this type of block, an impulse from the atria suddenly fails to conduct to the ventricles without antecedent progressive lengthening of the P–Q interval. The P–Q intervals are constant before and after the pause (Fig. 4.102).

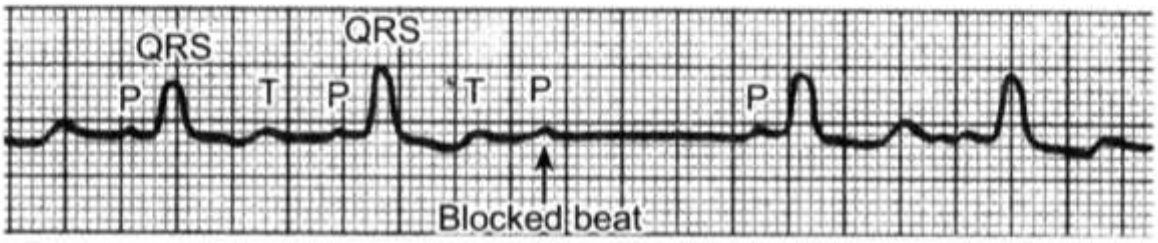


Fig. 4.102. Second-degree AV block Mobitz II illustrating blocked ventricular beat. The P–Q interval duration is constant.

ECG signs of Mobitz type II second-degree AV block.

1. The P–Q interval is constant.
2. Irregular missing of the ventricular QRST complex.

Type III second-degree AV block or incomplete AV block

Type III shows a specific ratio of blocked beat. The ratio of P waves to QRS complexes varies: 2:1, 3:1, 4:1, etc (Fig. 4.103).

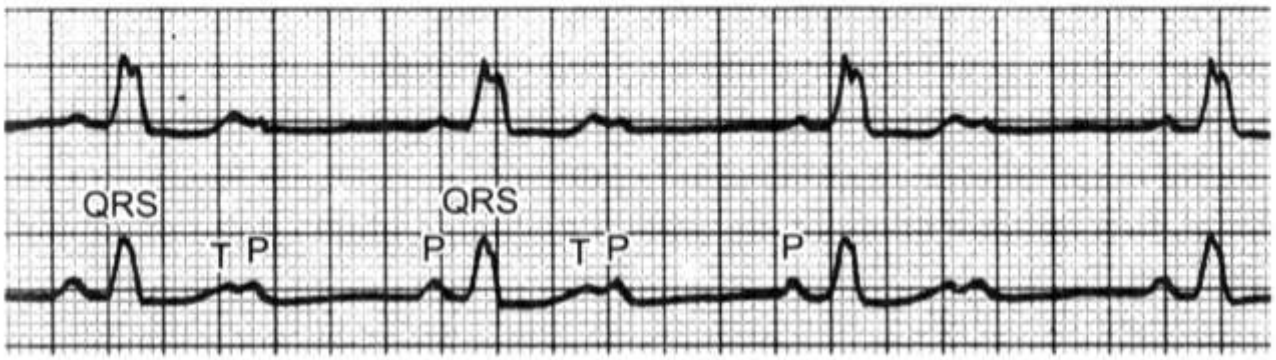


Fig. 4.103. Second-degree (incomplete) AV block type III. Group beating with block 2:1.

ECG signs of type III second-degree AV block.

1. Every second (2:1) ventricular complex is blocked, or two and more in succession (3:1, 4:1, etc).
2. The R-R intervals are regular.

Third-Degree AV block

Third-degree AV block is also called complete heart block. Inflammation, scarring, myocardial infarction, or drugs such as digitalis may cause complete heart block.

Third-degree AV block indicates a complete interruption of AV conduction. In this arrhythmia, no atrial impulses (P waves) activate the ventricles. The QRS originates from a junctional or ventricular pacemaker site. Therefore, the P waves and QRS complexes occur independently. Both the P waves and QRS complexes occur regularly, but there is no relationship between them. The atria are controlled by SA node at a rate of 70–80 per minute; the ventricular rate is 60–30 beats per minute. The lower ventricular pacemaker, the slower ventricular rate, the more bizarre the QRS complex (Fig. 4.104).

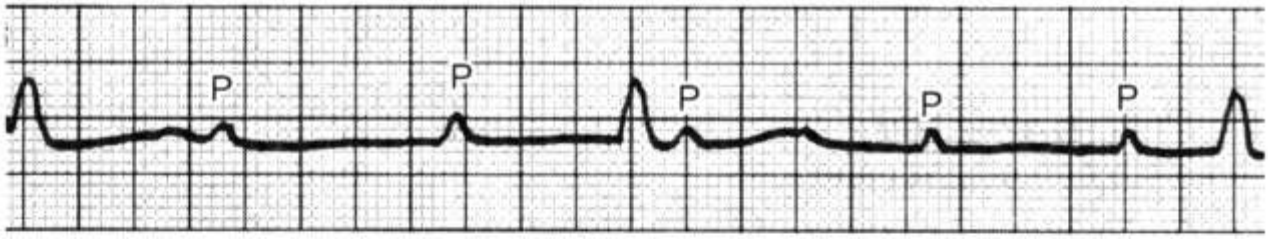


Fig. 4.104. Third-degree (complete) AV block, diagnosed as such because the regularity occurring P waves are not fixed in the ventricular cycle and the ventricular rate is regular.

ECG signs of third-degree AV block.

1. The P waves bear no relation to the QRS complexes.
2. The P wave may be before, after, or superimposed on the QRS complex or on the T wave to cause their deformation.
3. The R–R and P–P intervals are constant, but R–R intervals are longer than P–P intervals.
4. If the site of the block is high in the AV junction, the QRS complexes will be normal in configuration. The ventricular rate is not less than 45–60 beats per minute.

5. If the site of the block is below the bifurcation of the common His bundle, the QRS complex is wide and deformed because the ectopic ventricular pacemaker causes the ventricles aberrantly to be activated. The ventricular rate is not more than 40–45 beats per minute.

Abnormalities of the Ventricular Conduction.

Bundle-Branch Block.

Bundle-branch block is primarily an electrocardiographic diagnosis arising from situations that delay or interrupt the passage of an electrical impulse into, or within, the ventricles. Bundle-branch block is a condition that occurs when an electrical impulse passes through the AV node in a normal fashion, but delayed or blocked below this level, as a consequence, ventricular activation is abnormal, resulting in abnormal QRS, S–T, and T morphologies.

Bundle-branch block is an obstruction in the right or left ventricular conduction pathway (Fig. 4.105). When this occurs, the impulse travels first through the unobstructed branch and is then transmitted by nonspecialized myocardial tissue to the opposite ventricle. This aberrant pathway requires a longer time for activation of the ventricles, and the resulting QRS is greater than 0.12 second and of abnormal configuration. Origin of this beat is from the SA node. Therefore, a P wave will precede the wide QRS complex.

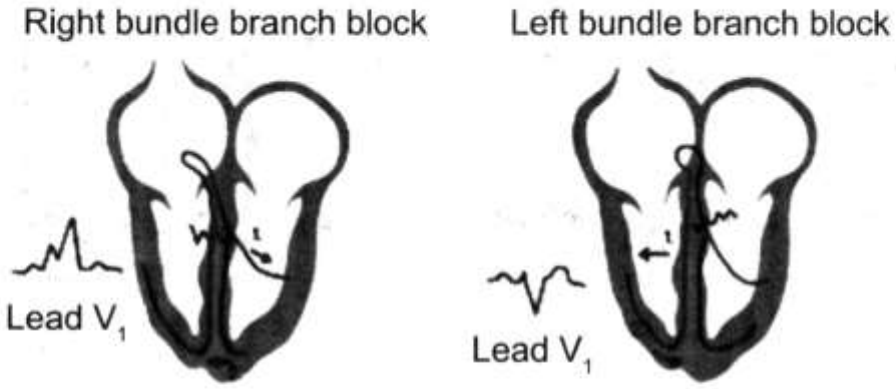


Fig. 4.105. Right and left bundle-branch block.

Right Bundle-Branch Block

When the right bundle-branch is blocked, the impulse travels first through the left ventricle. Therefore, the initial electrical activation on the left side of the heart is normal, but the right ventricle is the last portion of the heart to be activated.

ECG signs of right bundle-branch block (Fig. 4.106).

1. The M-shaped QRS complex in leads V_1, V_2 (rarer in leads III, aVF), where R'>r. R' – is produced by delayed onset of ventricular activation in a rightward direction, r – activation of the left ventricle.
2. A decidedly slurred, broad S wave, produced by the late right ventricular and septal activation, in leads V_5, V_6, I, aVL.
3. QRS widened to more than 0.12 second. The prolongation of the QRS interval is due to the delayed onset of right ventricular activation and the slow conduction in the right ventricle due to muscle cell-to-cell conduction through the septum and the right ventricular wall.
4. S–T segment and T wave changes in the leads over the right ventricle. The right precordial leads V_1, V_2 (rarer lead III) show depressed S–T segment and an inverted T wave. The course of activation is altered, with a resultant change in the course of repolarization. In general, the T wave is opposite in direction from the terminal part of the QRS complex.

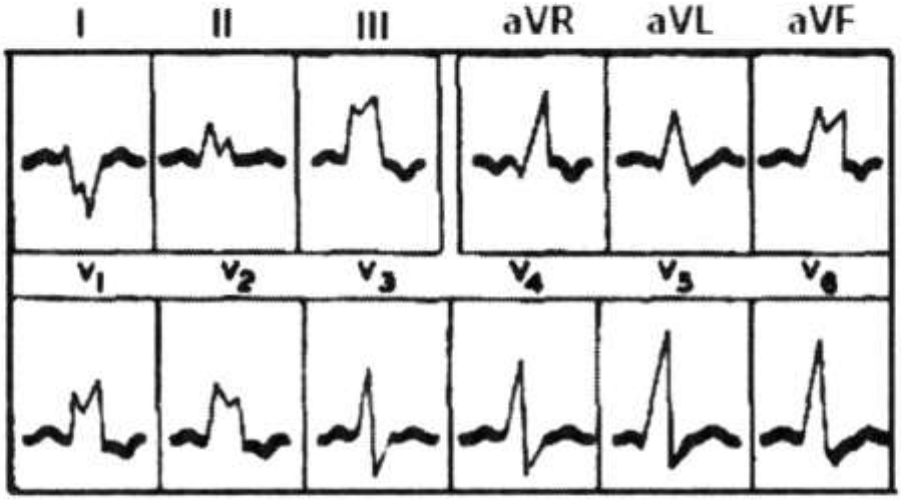


Fig. 4.106. Complete right bundle-branch block.

Left Bundle-Branch Block.

If the entire left bundle-branch is blocked, the impulse first depolarizes the right side of the heart and then through aberrant pathways – the left ventricle.

ECG signs of left bundle-branch block (Fig. 4.107).

1. Increased amplitude of ventricular complex QRS with notched or slurring peak of the R wave in leads V_5, V_6, I, II, aVL. Because left ventricular

stimulation is delayed, unopposed by right ventricular activation, the resultant potential is of greater than normal magnitude.

2. A wide and deep ventricular complex by QS type with a slurred, broad S wave in leads V_1, V_2, III, aVF.
3. QRS widened to more than 0.12 second. The late arrival of the depolarization wave at the left ventricle produces this change.
4. S–T segment and T wave changes in the leads over the left ventricle. The left precordial leads V_5, V_6, and leads I, aVL have depressed S–T segment and an inverted T wave, because abnormal depolarization results in abnormal repolarization. In general, the S-T segment and T wave are discordant (opposite) in direction from the terminal part of the QRS complex.

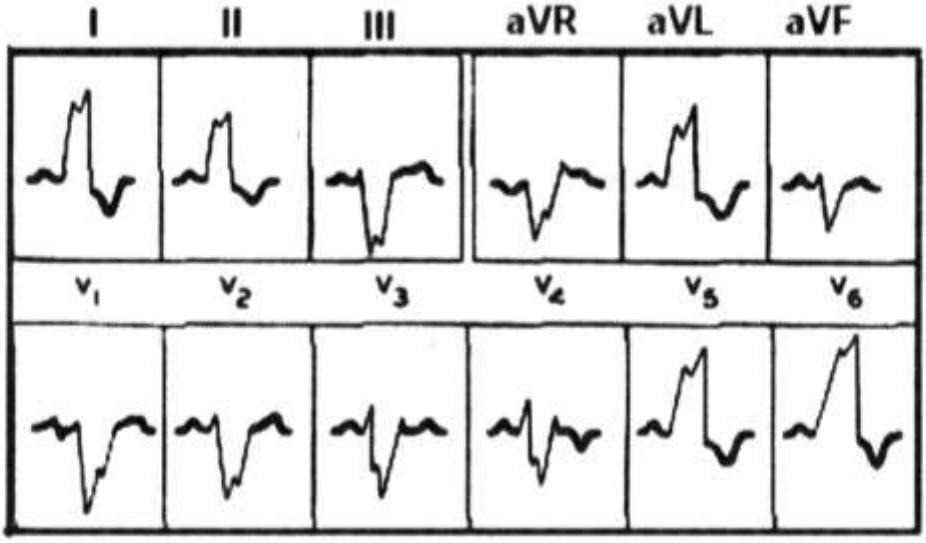


Fig. 4.107. Complete left bundle-branch block.

Ventricular Preexcitation Syndromes

Ventricular preexcitation has been defined as a conduction abnormality in which ventricular muscle is activated earlier than would be expected had the impulse reached the ventricles via the normal AV conducting system. Early activation of the ventricles occurs because of bypass tracts from atria to ventricles.

Syndrome of Shortened P–Q Interval or CLC Syndrome

The syndrome carries the names of physicians who described it in 1938. In Clerk-Levy-Critesko (CLC) syndrome, impulses enter the ventricles prematurely through specialized anomalous conduction pathway called

the fibers of James. These fibers bypass the AV node to cause ventricular preexcitation. As ventricles are activated in usual manner, the QRS complexes are of normal configuration.

ECG signs of CLC syndrome.

1. The P–Q interval duration is less than 0.12 second.
2. The QRS complexes are of normal configuration.

Wolf-Parkinson-White (WPW) Syndrome

In this syndrome, impulses enter the ventricles prematurely through specialized anomalous conduction pathways called the bundles of Kent. These bundles are formed outside the conduction system. The Bundle of Kent connects the conduction system of atria to either ventricle, bypassing the AV node. The impulses do not travel through the AV node and thus avoid the normal conduction delay that occurs there.

ECG signs of WPW syndrome.

1. A short P–Q interval (less than 0.12 second).
2. A slurred upstroke on the QRS complex (delta wave).
3. A wide QRS complex (greater than 0.10 second).
4. Secondary S–T and T wave changes.

Echocardiography

Echocardiography is defined as the transmission ultrasound through the heart with detection of the returning echoes.

Cardiac ultrasound scanners have improved to the point that diagnostic images can be obtained in nearly every patient. The commonest indication is the assessment of the left ventricular function. In addition to imaging cardiac structure, blood flow can be displayed (Doppler echocardiography) and quantified. Most scans are performed through the anterior wall of the chest but when high-resolution images of posterior structures (left atrium or descending aorta) are required transesophageal imaging is employed. This can be carried out safely in outpatients using topical anesthesia and intravenous sedation.

Echocardiography includes three interrelated modalities: two-dimensional echocardiography, M-mode echocardiography, and Doppler echocardiography.

Two-dimensional and M-mode echocardiography

From following standard positions of the transducer examination is performed (Fig. 4.108):

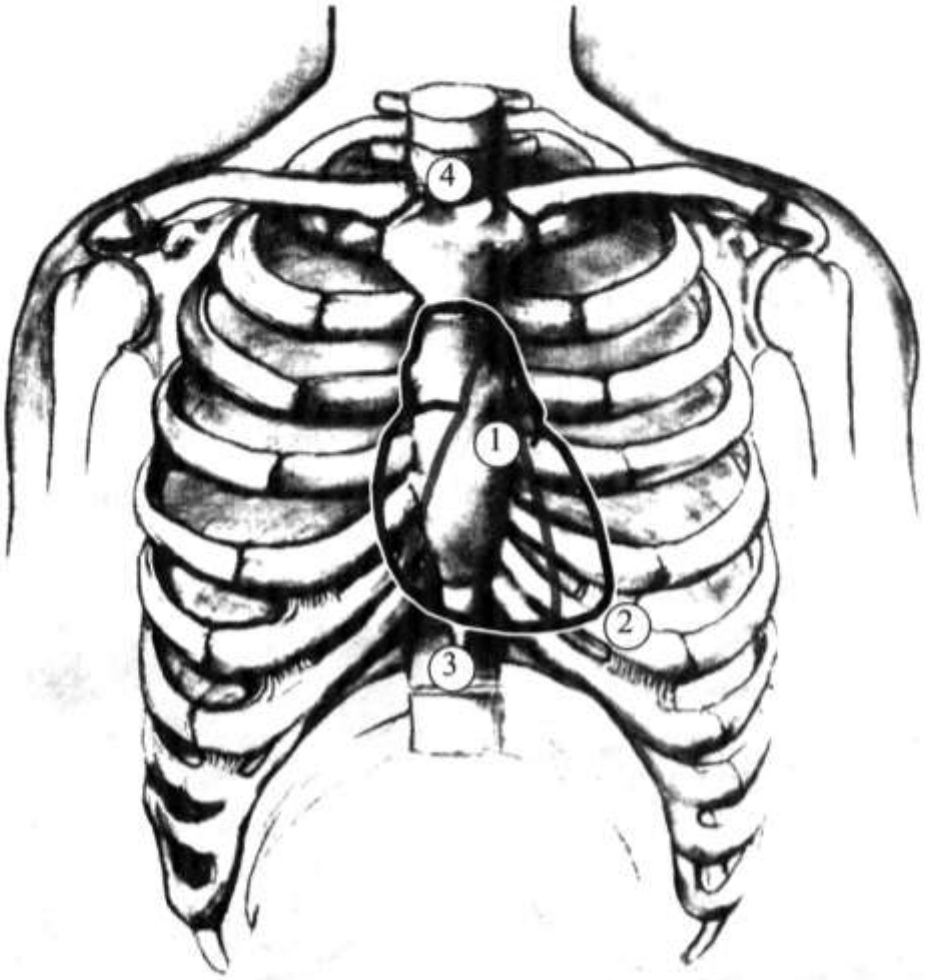


Fig. 4.108. Diagram of the transducer standard positions.

Parasternal – the transducer is placed in the third or fourth intercostal space at the left side of the sternum;

Apical – the transducer is placed at the heart apex in cranial direction, the patient being in left lateral position;

Subcostal – the transducer is placed below the xiphoid of the sternum;

Suprasternal – the transducer is placed in the suprasternal notch.

From each standard position, the heart is examined in several directions: ***long axis view*** (parallel to the long axis of the heart) and ***short axis view*** (perpendicular to the long axis of the heart).

Initial echocardiographic examination begins with two-dimensional study, conducting corresponding measurements, and choice optimal orientation of the transducer at various levels for M-mode study. Then examination continues in next standard position.

Parasternal long axis view

Two-dimensional view. Parasternal long axis view is used mainly for study of the left heart chambers: the shape and the size of the left ventricle, left ventricle wall motions, thickness and motion of the interventricular septum, ventricular outflow tract, aortic root, left atrium, mitral and aortic valves apparatus. The left ventricular apex is not seen in this view, and therefore it is not useful for assessment of ventricular volumes.

In this position the transducer is placed in third or fourth intercostal space at the left side of the sternum perpendicular to the chest surface. The marker of the transducer points cephalad towards right shoulder.

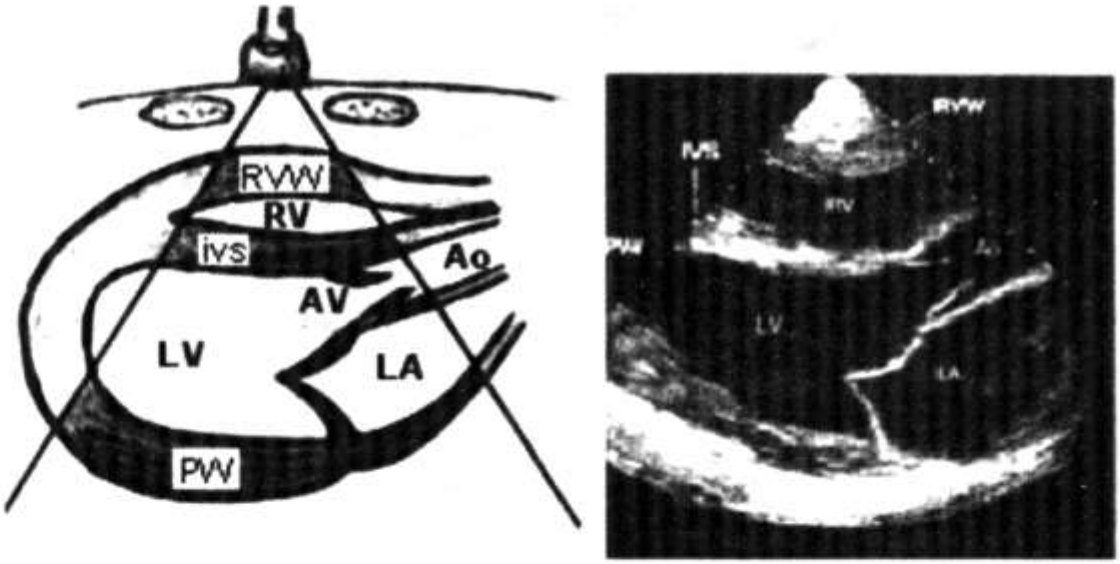


Fig. 4.109. Two-dimensional parasternal long axis view in systole.

The following intracardiac structures are excellent visualized (Fig. 4.109): anterior wall of the right ventricle (RVW), right ventricular cavity (RV), the interventricular septum (IVS) is in anatomic continuity with the anterior wall of the aorta, part of the left ventricular cavity (LV), except apex area, aorta (Ao) and aortic valve, mitral valve (MV) (the large anterior mitral leaflet is in anatomic continuity as posterior wall of aorta; the small posterior leaflets originate at the atrioventricular groove), left atrium (LA),

and the posterior wall (PW) of the left ventricle is in continuity as left atrium.

M-mode view. M-mode study is commonly conducted from the left parasternal long axis view (Fig. 4.110). The ultrasonic beam should be maximal perpendicularly directed towards the area to be examined by M-mode at the level of the leaflets of the aortic valve and left atrium (1), at the level of the leaflets of the mitral valve (2), and at the level of chordae (3).

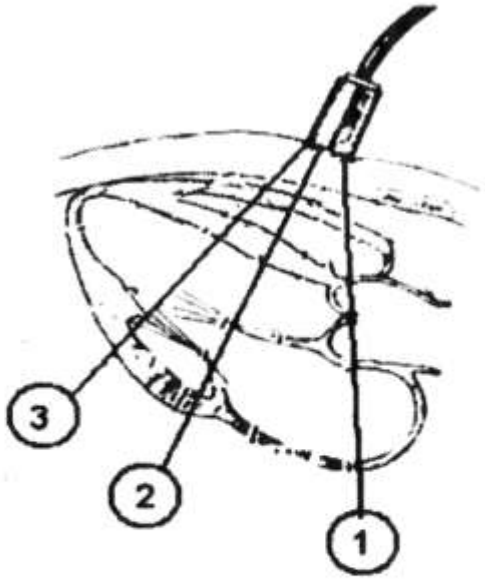


Fig. 4.110. Diagram of the M-mode examination with transducer oriented at various levels.

M-mode study at the level of the aorta and left atrium

Transition from the two-dimension view to M-mode gives view of aorta, aortic valve, and left atrium (Fig. 4.111). The following structures are visualized: anterior right ventricular wall (RVW), right ventricular cavity (RV), aorta (Ao), left atrial cavity (LA), and posterior wall of the left atrium.

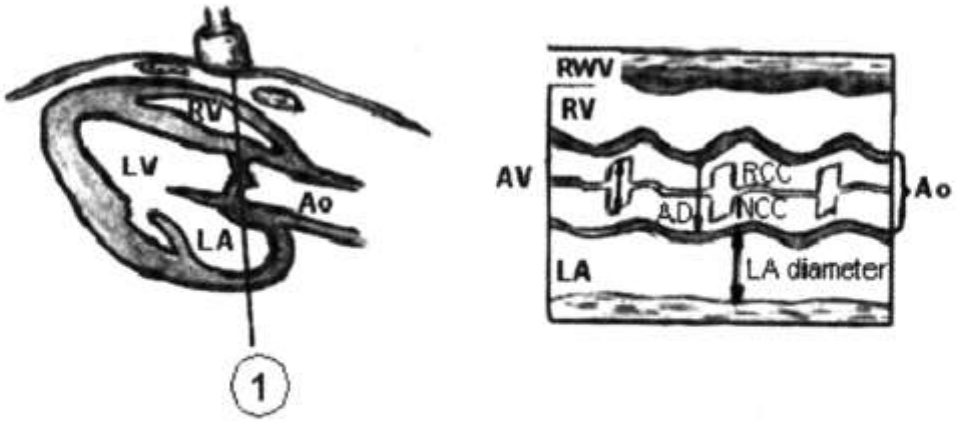


Fig. 4.111. M-mode echocardiogram at the level of the aortic valve cusps.

Anterior and posterior aortic walls are seen as a parallel undulating 2–3mm lines. They move toward transducer (anteriorly) and parallel to each other in systole and posteriorly in diastole and give a characteristic appear-

ance. Amplitude of their movements normally is 7–10 mm. The aortic diameter (AD) is measured as a distance between outside surface of its anterior wall and inside surface of posterior wall (between the anterior edges of two aortic walls) in end diastole. Normally the diameter of the aorta is 20–36 mm. Within the aortic walls movements of the aortic valve cusps are seen. The anterior or right coronary cusp (RCC) and posterior or noncoronary cusp (NCC) are commonly recorded. The middle or left coronary cusp, being parallel to the beam is not commonly seen. During systole the aortic valve cusps separate to form a box-like structure, and during diastole – close up. Amplitude of the aortic valve cusps opening at the beginning of left ventricular systole exceeds 18 mm normally.

The left atrial posterior wall moves in this position during the ventricular systole approximately 8–10 mm downward. The left atrial diameter measurement is done at end systole at the period of maximal movement of posterior aortic wall upward between the outward surface of the posterior aortic wall and endocardial surface of the left atrial wall. Normal diameter of the left atrium is 19–33 mm.

M-mode study at the level of the mitral valve

When ultrasonic beam directed toward the mitral valve cusps the following cardiac structures are visualized on the monitor (Fig. 4.112): anterior wall of the right ventricle (RWV), right ventricular cavity (RV), interventricular septum (IVS), left ventricular cavity (LV), within LV anterior (aML) and posterior (pML) mitral valve leaflets, and left ventricular posterior wall.

This position allows assessing structure and character of the mitral valve movements. The anterior leaflet of the mitral valve shows characteristic "M" pattern motion and the posterior leaflet of the mitral valve shows characteristic "W" pattern motion during diastole. During systole, the anterior leaflet lies close to the posterior leaflet. The curve of the anterior leaflet movement is analyzed. Point 'C' corresponds to complete closure of the mitral valve at the beginning of the left ventricular systole, point 'D' – to opening of the mitral valve leaflets at the beginning of the diastole. Left ventricular systole and complete closure of the mitral valve is designated as C–D interval. From 'D' point, diastole starts and anterior leaflet moves anteriorly toward the septum to reach the point 'E'. D–E interval reflects separation of the mitral valve leaflets during the phase of quick filling of the

left ventricle. During middiastole, as the flow falls off, the anterior leaflet shows a downward motion to a point defined 'F'. Incomplete closure of the mitral valve leaflets during the phase of slow left ventricular filling is seen as 'E–F' interval. As a result of left atrial systole, the leaflets moves again anteriorly to cause 'A' wave.

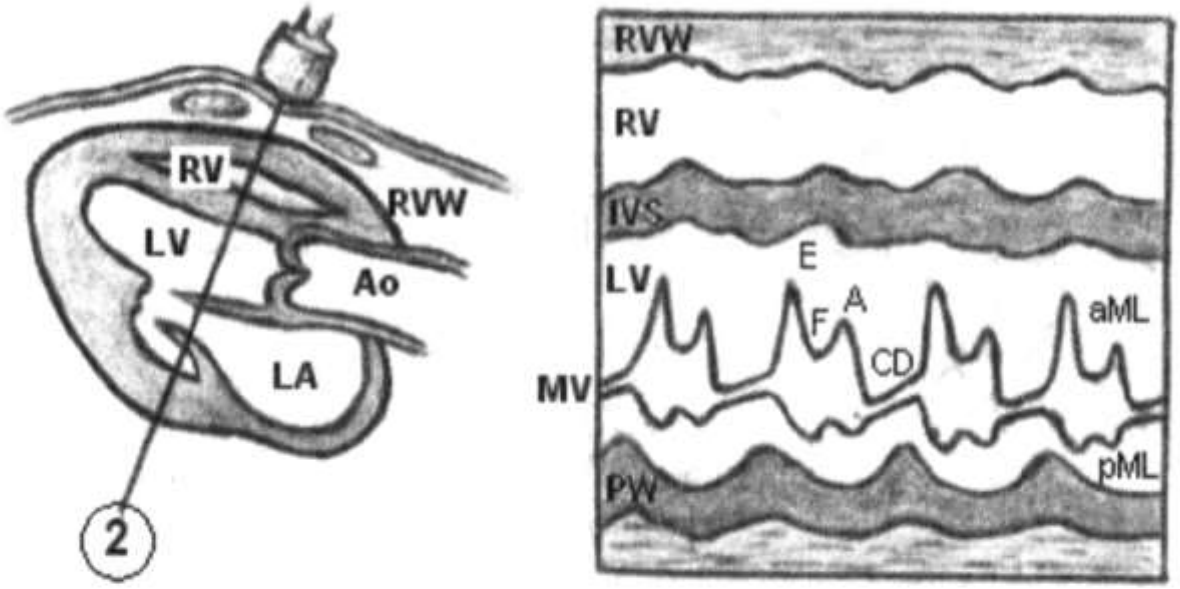


Fig. 4.112. M-mode echocardiogram at the level of the mitral valve cusps.

M-mode study at the level of papillary muscles

In this view right ventricular wall (RVW), right ventricular cavity (RV), interventricular septum (IVS), left ventricular cavity (LV), and posterior wall of the left ventricle (PW) are visualized (Fig. 4.113). M-mode echocardiography at this level shows changes of systolic and diastolic size of the left ventricular cavity, thickness of the interventricular septum, and posterior left ventricular wall.

The left ventricle cavity is seen between interventricular septum and left ventricular posterior wall. Both these walls become thicker and move towards to each other during systole to decrease diameter of the left ventricular cavity. It is so-called end systolic diameter (ESD), normally – 22–38 mm. During diastole interventricular septum and posterior wall of the left ventricle become thinner and move away from each other to cause increasing of the left ventricular cavity. It is so-called end diastolic diameter (EDD), normally – 38–56 mm.

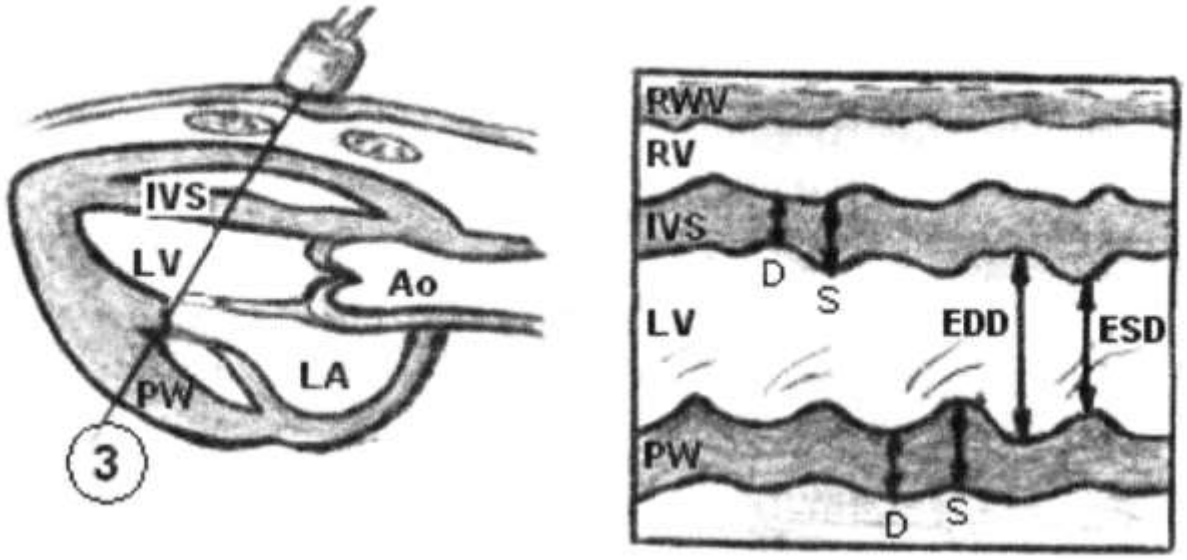


Fig. 4.113. M-mode echocardiogram at the level of the papillary muscles.

In this transducer position interventricular septal thickness is measured as distance between the anterior right septal echo and the anterior left septal echo at the end of diastole (normally 7–10 mm), and at the end of systole (normally 5–6 mm). The posterior wall thickness is the distance between the anterior endocardial echo and anterior epicardial echo at the end of diastole (8–11 mm), and at end systole (8–12 mm).

Parasternal short axis view

The parasternal short axis view is obtained by placing the transducer in the III–IV intercostal space at the left side of sternum, and is rotated 90° clockwise. The short axis views are commonly obtained at the level of aortic valve, mitral valve, and left ventricle papillary muscles by tilting the transducer from the direction of the feet to upward and slight medially.

Two-dimensional echocardiographic short axis examination of the heart is unique in a sense as such view is impossible either by M-mode echocardiography or by angiography. In short axis view, the left ventricle is cut in transverse plane and is seen in circular shape of the normal heart. The right ventricle is placed anteriorly in front and to the left side of the left ventricle image.

Short axis view of the left ventricle at the aortic valve level is obtained by ultrasonic beam directed slight to the right and upward (Fig. 4.114).

The circular aorta containing the aortic valve is seen in the middle of the image. In diastole, the three aortic valve cusps to form a 'Y'-shaped fig-

ure. The anterior aortic leaflet or right coronary cusp (R) is at the top, the left coronary cusp (L) is to the right, and the non-coronary (N) or posterior cusp is visualized below. The right ventricle outflow tract (RVOT) is placed anteriorly around aorta. The pulmonary valve (PV) is seen at two o'clock position on the aortic circle and separates the main pulmonary artery from the right ventricular outflow tract. The left atrium (LA) is placed posterior to the aortic circle. Interatrial septum (IAS) is seen at seven o'clock position on the aortic circle and separates left and right atrium (RA). The tricuspid valve (TV) is positioned at ten o'clock position on the aortic circle and separates right ventricle and right atrium.

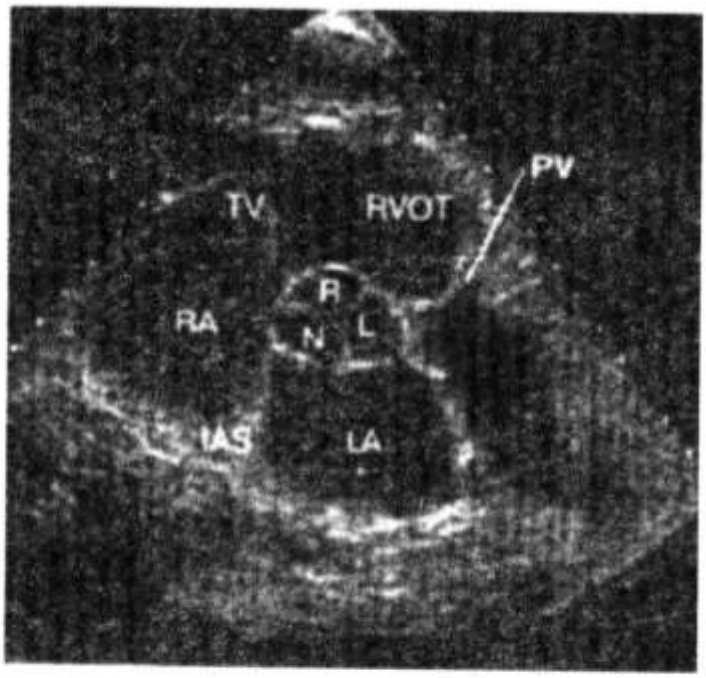


Fig. 4.114. Two-dimensional parasternal short axis view at the level of the aortic valve.

With slight change in angulation of the transducer laterally towards the left shoulder, one can visualized the right ventricular outflow tract (RVOT), pulmonary valve (PV), the total length of the main pulmonary artery (PA) up to its bifurcation into the right (RPA) and left (LPA) branches (Fig. 4.115). This position is commonly used for Doppler study of the blood flow in the pulmonary artery.

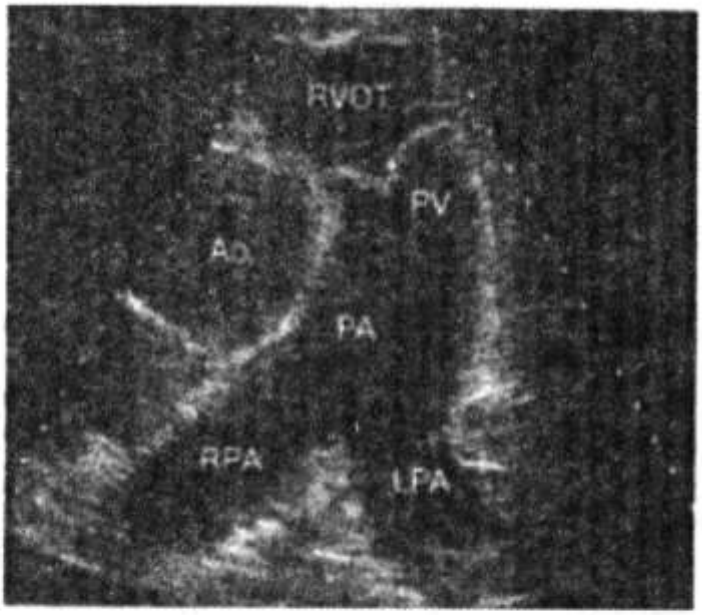


Fig. 4.115. Two-dimensional parasternal short axis view at the level of the aortic valve. The slight change in angulation of the transducer allows visualize pulmonary artery.

The short axis view at the level of the mitral valve is used

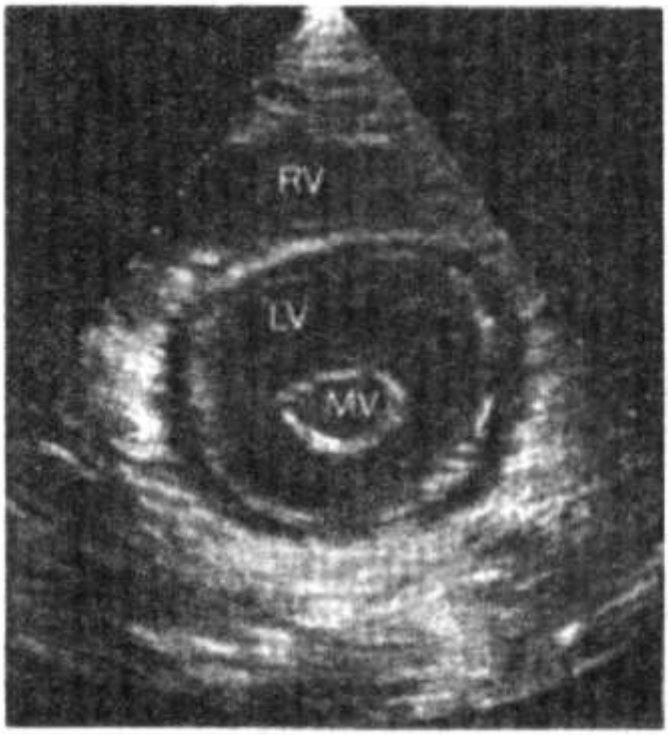


Fig. 4.116. Two-dimensional parasternal short axis view at the level of the mitral valve.

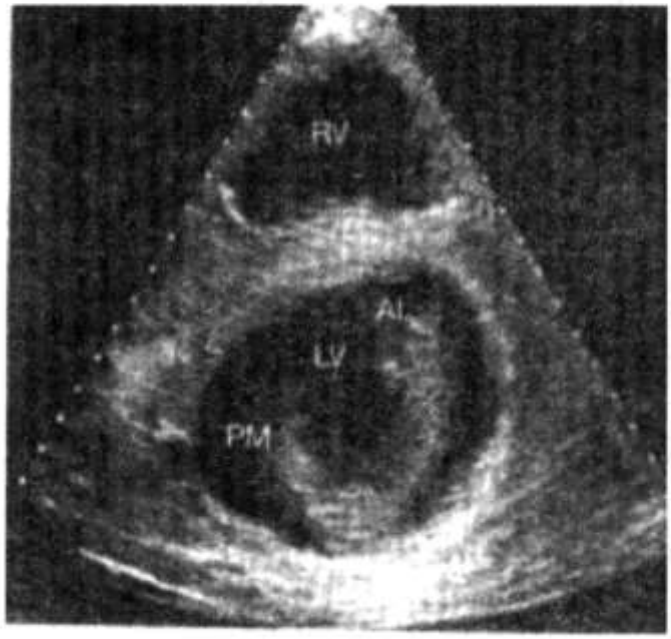


Fig. 4.117. Two-dimensional parasternal short axis view at the level of the papillary muscles.

for evaluation of the left ventricular regional wall motion abnormalities and mitral orifice area. By slightly tilting the transducer to the left, an excellent view of a circular left ventricle (LV) cavity with mitral valve (MV) in the center can be obtained (Fig. 4.116). As the mitral valve leaflets open and close during systole and diastole, it gives 'fish mouth' appearance.

The short axis view at the level of the papillary muscles is also used commonly for examination of the left ventricle. With change in angulation of the transducer slightly downward, a circular left ventricle (LV) cavity in the center of image is seen (Fig. 4.117). The posteromedial (PM) and anterolateral (AL) papillary muscles are located in the center of the left ventricular cavity. The right ventricle (RV) is placed anteriorly.

The abnormalities of the papillary muscles including thickening, carnification and rupture can be studied in this view.

Apical four-chamber view

The apical four-chamber view is obtained by placing transducer at the heart apex with the sector plane from the left

scapula to the right clavicle, the patient being in left lateral position. Section through the whole heart displays all four chambers: interventricular and interatrial septum are in the center of image, left ventricle (LV) including apex and left atrium (LA) are to the right, right ventricle (RV) and right atrium (RA) are to the left (Fig. 4.118). The mitral and tricuspid valves are seen in one plane.

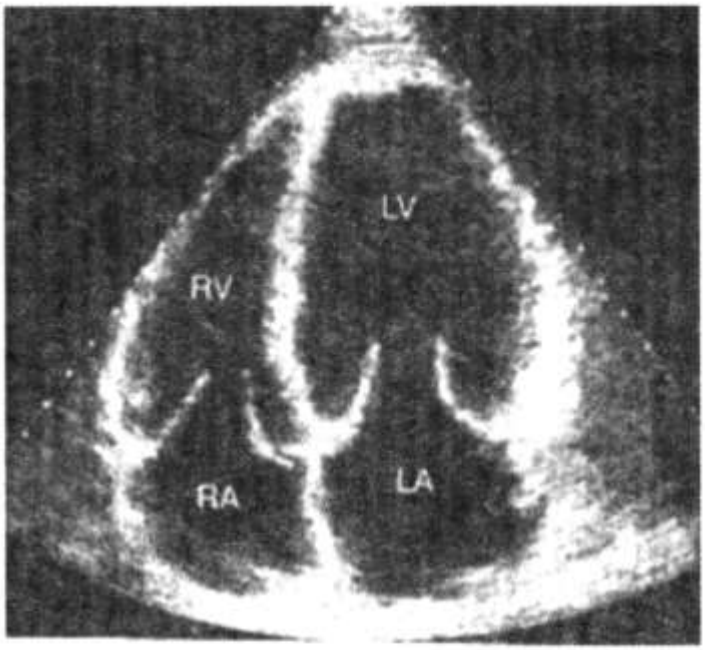


Fig. 4.118. Two-dimensional apical four-chambers view in diastole.

The apical four-chamber view is good for evaluation of the left ventricular wall including apex, comparative evaluation of left and right chambers of the heart, ventricular and atrial septa.

Apical five-chamber view

The apical five-chamber view can be obtained by slightly tilting the transducer anteriorly with an additional recording of root of aorta (Fig. 4.119). This position allows visualized not only both ventricles and atria but also left ventricular outflow tract (LVOT), aortic valve, and initial part of the ascending aorta. This view is commonly used for the Doppler study of the left ventricular outflow tract and aortic blood flow.

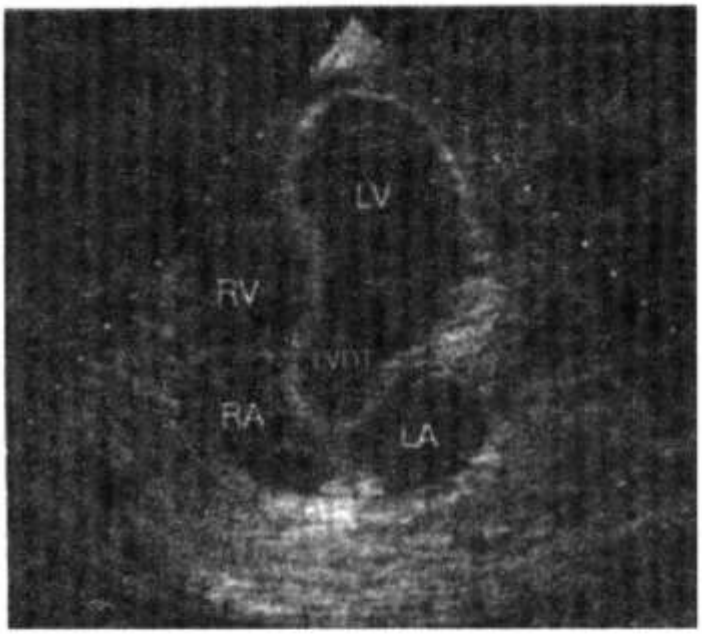


Fig. 4.119. Two-dimensional apical five-chambers view in systole.

Apical two-chamber view

In the apical two-chambers view left heart is examined left ventricle (LV), left atrium (LA), and mitral valve (Fig. 4.120). This view can be obtained by

Fig. 4.120. Two-dimensional apical two-chambers view in systole.

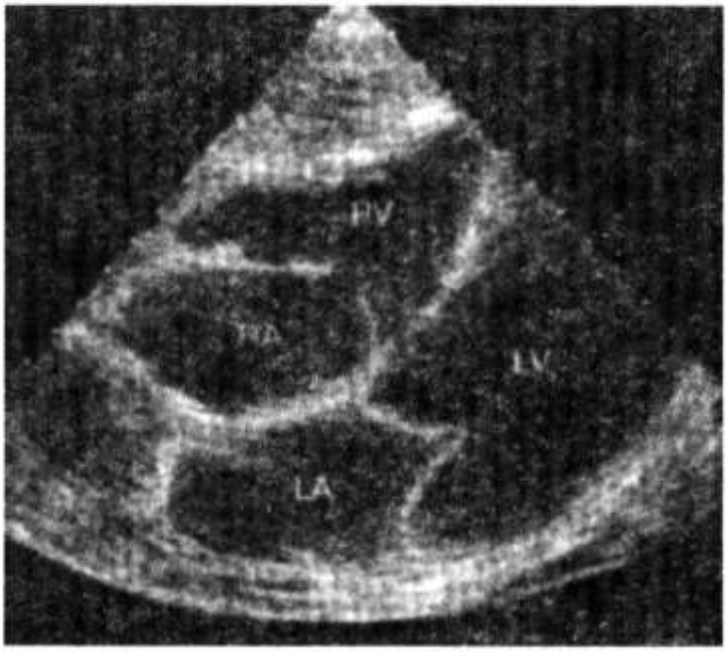


Fig. 4.121. Two-dimensional subcostal long axis view.

rotating the transducer at the heart apex counterclockwise by 45 degrees, converting apical four-chambers view into apical two-chambers view.

The subcostal view

Echocardiography in this view allows visualize inferior vena cava together with right atrium, hepatic veins and abdominal aorta. Moreover, subcostal view is particularly useful in examination of the morphological changes of the cardiac structures in children, in the patients who have pulmonary emphysema (hyperinflated lungs) and low diaphragm.

The transducer is placed below the xiphoid of the sternum in transverse sector plane. To obtain subcostal long axis view, the central ultrasonic beam is directed upward and to the left (Fig.4.121). The subcostal view is suitable for evaluation of left ventricle (LV), left atrium (LA), right ventricle (RV), right atrium (RA), mitral and tricuspid valves, interventricular and interatrial septum.

Suprasternal view

The suprasternal view gives a view of the base of the heart and great vessels: aortic arch, right branch of the pulmonary artery, and inferior vena

cava. This approach is used mainly for the Doppler echocardiography examination of the blood flow in these vessels.

A picture of the ascending and descending aorta is obtained if the tranducer is placed in the suprasternal notch. The sector plane is almost in anteroposterior direction. Below the aortic arch, pulmonary artery is seen in transverse section and below it, lies the left atrium.

Assessment of ventricular function

The assessment of the ventricular function is one of the most important and useful clinical application of echocardiography. Cardiac chamber dimensions can be accurately measured by M-mode and two-dimensional echocardiography. Measurement of the left ventricular end diastolic diameter (EDD) and end systolic diameter (ESD) can provide a reliable index of left ventricular size and performance. These dimensions can be used to estimate ventricular volumes.

Ejection fraction is the most useful single index of the left ventricular function, which correlates best with patient's outcome.

Teicholz method.

End diastolic volume (EDV): $EDV = 7\times(EDD)3/(2,4+EDD)$ (ml),
End systolic volume (ESV): $ESV = 7\times(ESD)3/(2,4+ESD)$ (ml),
Stroke volume (SV): $SV = EDV - ESV$ (ml),
Ejection fraction (EF): $EF = SV / EDV$ (%),
Minute volume (MV): $MV = SV\times HR$ (l), where HR – heart rate,
Stroke index (SI): $SI = SV / S$, where S – body surface area,
Cardiac index (CI): $CI = MV / S$, where S – body surface area.

It should be remembered however, that these M-mode measurements are not valid in ischemic heart disease patients with left ventricular segmental wall motion abnormalities, and not take into account complete geometry of the ventricular cavity.

Two-dimensional echocardiography is having considerable advantage over M-mode for quantitating volumes because of the inability to have global left ventricular measurements by M-mode technique, and 2D echo technique can analyze left ventricle in various tomographic views. Ejec-

tion fraction derived by two-dimensional echocardiographic measurements and volume estimations have been shown to have consistent relation to angiographic ejection fraction. Several geometric formulas: Simpson's rule, modified Simpson's rule, Bullet's formula have been used to calculate left ventricular volumes and ejection fraction.

Fractional shortening (%ΔS) of the left ventricle is an important and valuable index of the overall left ventricular performance. It is the difference between the end diastolic and end systolic dimensions divided by end diastolic dimension:

$$\%\Delta S = EDD - ESD / EDD\ (\%).$$

In determination of mean circumferential shortening (V_{CF}), the calculations are made in the same manner as for the fractional shortening except that ejection time is taken into consideration:

$$V_{CF} = EDD - ESD / dt \times EDD.$$

The value of fractional shortening and mean circumferential shortening in assessment of left vwentricular global function is limited in patients with segmentally diseased left ventricle, left bundle branch block, dilated right ventricle or with low echocardiographic window.

Selected echocardiographic parameters are represented in the Table 4.51.

Tab. 4.51. ***Average echocardiographic parameters in healthy adults.***

Parameter	Value
Left ventricle	
Left ventricular myocardium mass (LVMM) *Average:* Male Female *Upper limit:* Male Female	 135 g 99 g 183 g 141 g

Left ventricular myocardium mass index (LVMMI)	
Average:	
Male	71 g/m^2
Female	62 g/m^2
Upper limit:	
Male	94 g/m^2
Female	89 g/m^2
Posterior wall thickness in diastole (PWT)	0.8–1,1cm
Posterior wall systolic excursion	0.8–1.2 cm
End diastolic diameter (EDD)	3.8–5.6 cm
End systolic diameter (ESD)	2.2–3.8 cm
End diastolic volume (EDV)	110–145 ml
End systolic volume (ESV)	45–75 ml
Stroke volume (SV)	60–80 ml
Stroke index (SI)	25–34 ml/m^2
Ejection fraction (EF)	55–65 %
Minute volume (MV)	3.5–4.5 l/min
Cardiac index (CI)	1.9–2.5 l/min/m^2
Fractional shortening (%ΔS)	28–43 %
Circumferential shortening (V_{CF})	0.8–1.2 [c-1]
Right ventricle	
Wall thickness	5 mm
Diastolic diameter	0.95–2.05 cm
Size index	0.75–1.25 cm/m^2
Left atrium	
Diameter	1.9–3.3 cm
Diameter index	1.45–2.9 cm/m^2
Right atrium	
End diastolic volume:	
Decreased	Less than 20 ml
Increased	More than 100 ml
Significantly increased	More than 300 ml

Interventricular septum	
Myocardium thickness	0.7–1.0 cm
Systolic excursion	0.5–0.6 cm
Mitral valve	
Common motion excursion	19; 25; 5 mm
Diastolic cusps separation	14–0 mm
Mitral orifice area	4–6 cm^2
Mitral stenosis degrees (according to mitral orifice area):	
Insignificant	exceed 2.0 cm^2
Small	1.6–2.9 cm^2
Moderate	1.1–1.5 cm^2
Significant	0.8–1.0 cm^2
Critical	less than 0. 8 cm^2
Aorta	
Diameter	2.0–3.6 cm
Amplitude of the aortic valve opening	exceed 18 mm

Clinical applications of echocardiography

Valvular heart disease

Mitral stenosis

Echocardiographic M-mode signs:

- Considerable altered motion of the mitral valve with slow diastolic closure rate (E-F slope);
- Absence of normal 'M'-shaped configuration during diastole, pathologic "П"-shaped configuration presence;
- Anteriorly movement of posterior mitral leaflet in diastole;
- Increased size of the left atrium;
- Increased size of the right ventricle.

Echocardiographic two-dimensional signs:

- Increased thickness, calcification or fibrosis of valve leaflets;
- Doming of the mitral valve leaflets;

- Distortion in shape with opening of the valve as the tips of the leaflets are restricted in their opening;
- Decreased cross-sectional area of the mitral valve orifice;
- Enlarged left atrium;
- Enlarged right ventricle.

Mitral regurgitation

Echocardiographic M-mode and two-dimensional signs:

- Incomplete closure of the mitral valve leaflets in systole;
- Smoothed A wave;
- Enlarged and dilated left atrium;
- Enlarged and hypercontractile left ventricle;
- Increased stroke volume;
- Increased ejection fraction;
- Exaggerated septal motion;
- Enlarged cross-sectional area of the mitral valve orifice (exceeds 7 cm^2).

Mitral valve prolapse

Echocardiographic M-mode signs:

- Posterior displacement of mitral valve, especially the posterior leaflets towards left atrium in mid or late systole.

Echocardiographic two-dimensional signs:

- Bucking of one or both mitral leaflets into the left atrium during systole, demonstrable in parasternal long axis view;
- In apical four-chamber view the prolapsed leaflet is demonstrated bulging into the left atrium distinctly below the level of mitral valve annulus;
- Posterior displacement of the coaptation point of the two leaflets, thickened and redundant floppy mitral leaflets due to myxomatous degeneration, and abnormal size and contraction of the mitral annulus.

Aortic stenosis

Echocardiographic M-mode signs:

- Thickened aortic valves leaflets;
- Immobile aortic valve leaflets, incomplete closure of the aortic valve;
- Left ventricular hypertrophy.

Echocardiographic two-dimensional signs:

- Thickened leaflets, calcification can be present, and the valve is frequently seen in diastole;

- Systolic doming of the aortic leaflets. The echoes from the leaflets are not parallel to the aorta as they are in the normal aortic valve;
- Reduced separation of the leaflets and edges of leaflets curved toward the center of aorta;
- Enlarged left ventricle;
- Afterstenotic dilation of the aorta.

Aortic regurgitation

Echocardiographic signs:

- Incomplete closure of the aortic valve in diastole;
- Enlarged aortic diameter and pulsation of its wall;
- Dilated left ventricle;
- Increased excursion of the left ventricular wall and interventricular septum;
- Fine fluttering of anterior mitral leaflet, or even interventricular septum;
- Reverse doming of the mitral leaflet on two-dimensional echocardiography;
- Premature closure of the mitral valve due to high left ventricular diastolic pressure.

Tricuspid stenosis

Echocardiographic M-mode signs:

- Decreased diastolic slope;
- Thickening of valve leaflets;
- Decreased separation of the leaflets.

Echocardiographic two-dimensional signs:

- Doming of the tricuspid valve in systole, best seen in parasternal long axis view or four-chambers view;
- Thickening of the tricuspid valve leaflets;
- Restricted movements of the tricuspid valve leaflets;
- Enlarged right atrium.

Tricuspid regurgitation

Echocardiographic signs:

- Incomplete closure of the tricuspid valve leaflets in systole;
- Enlarged and dilated right atrium;
- Enlarged and dilated right ventricle;
- Exaggerated septal motion: anterior motion of interventricular septum during isovolumetric contraction in M-mode study;

- Dilated right ventricle with flattening of interventricular septum during diastole by two-dimensional echocardiography.

Pericardial diseases

Effusive pericarditis

Pericardial effusion can be seen as echo—free space around the heart, excessive movement of the outside cardiac contour, decreased amplitude of surrounding pericardium movement, and deformation of the heart chambers, shape and movement of the valves.

Constrictive pericarditis

The thickened pericardium, a flat diastolic slope of the posterior left ventricular wall, abnormal interventricular septal motion, cogwheel closure of the mitral valve, and dilated inferior vena cava can be visualized.

Cardiomyopathies

Hypertrophic cardiomyopathy

The echocardiographic features are thickening of the left ventricular wall, thickening of the right ventricular wall (rare), decreased left ventricular cavity size, dilated left atrium, asymmetric septal hypertrophy, reduced septal wall motion. With the presence of the left ventricular outflow obstruction, the systolic anterior motion of the mitral valve is seen.

Dilated (congestive) cardiomyopathy

Echocardiographic signs are dilation of all cardiac chambers, especially left ventricle, decreased thickness of the heart chambers wall, reduced left ventricular function, mitral valve and aortic valve excursion are reduced due to low cardiac output. Due to dilation of the left ventricular cavity, incomplete closure of the mitral valve can be seen (relative mitral regurgitation).

Restrictive (infiltrative) cardiomyopathy

Concentric left ventricular hypertrophy, normal or reduced left ventricular cavity, reduced systolic and diastolic function with reduced septal and posterior wall motion is frequently seen, but not specific for restrictive cardiomyopathy.

Coronary artery disease

An ischemic myocardium can be detected by studying the segmental wall motions and thickness as well as thickening of walls during diastole

and systole. Ischemic myocardium show wall motion abnormalities (hypokinesia, akinesia, dyskinesia, aneurysm) and thickening of myocardial walls. Stress or exercise echocardiography is more informative in assessing patients at early stages of coronary hear disease.

In transmural myocardial infarction, the affected muscles will show akinesia or dyskinesia with thin-walled fibrotic appearance. In subendocardial myocardial infarction, the echocardiographic examination may not show any wall motion or thickness abnormalities.

The echocardiography is very useful in assessing extent and severity of the myocardial damage, and complications like ventricular dysfunction, thrombi, ventricular septal rupture, papillary muscles rupture, and ventricular aneurysm. Left ventricular aneurysms are thin walled, scarred, frequently dilated dyskinetic segments. Apical aneurysms are most common. The aneurysm area shows systolic expansion while rest of the myocardium shows contraction with reduction of cavity size.

Arterial hypertension

Echocardiographic signs include: left ventricular hypertrophy, thickening of the interventricular septum to more than 1,1 cm; IVS/PWT ratio >1, increased LVMMI, stroke volume and ejection fraction in compensation stage. With decompensation enlarged left ventricular cavity, decreased ejection fraction, and systolic thickening of the left ventricular wall can be seen.

Chapter 5. DIGESTIVE SYSTEM

FUNCTIONAL AND CLINICAL ANATOMY

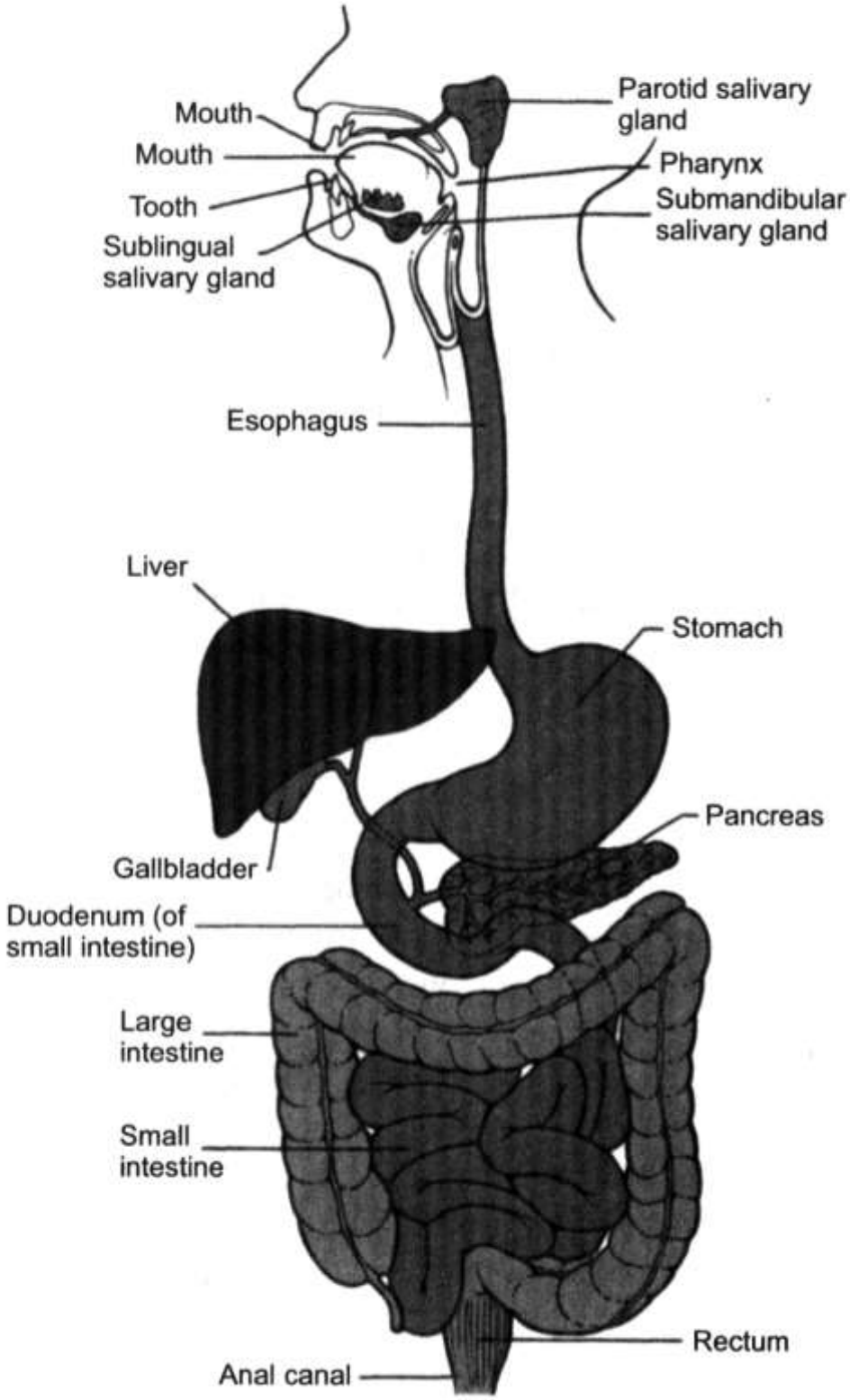


Fig. 5.1. The human digestive system.

After food is partially broken down by being chewed and mixed with saliva in the mouth, it is swallowed and enters the stomach, whose acids break it down further. As the food passes through the duodenum, secretions from the pancreas are added. Bile from the gall bladder is added in the small intestine. Digestive proteins and carbohydrates travel from the small intestine through the liver and into the bloodstream. Digestive fats move from the large intestine through the lymph system, which drains into the bloodstream via the heart. The large intestine reabsorbs excess water; the solid wastes that remain are collected in the rectum and excreted through the anus. The time that food spends in each part of the gastrointestinal tract is highly variable; the intervals shown here are averages (Fig. 5.1).

The mucosal surface of the gastrointestinal tract is composed of epithelial cells that are highly developed in their capacity for transmembrane absorption and secretion. These secretory and absorptive abilities facilitate the essential function of the digestive tract in digestion and nutrient uptake.

The intestinal surface itself also contains the distinctive M cells that serve to sample the antigenic milieu of the lumen. They overlie lymphoid aggregates (Peyer's patches). The predominance of suppressor lymphocytes in the surface epithelial layer (intraepithelial lymphocytes) suggests that damping of the body's response to the enormous number of potentially antigenic substances in the lumen is necessary to prevent the constant and unrestrained activation of immune and inflammatory processes. Conversely, the presence of large numbers of helper lymphocytes as well as other cellular effectors of immune response in the lamina propria and submucosa attests to a large armamentarium ready to respond when surface defenses have been breached. No doubt the concentration of so many immune cells capable of attracting and activating inflammatory cells predisposes to the numerous inflammatory conditions to which the gastrointestinal tract is subject.

The mucosal surface of the gastrointestinal tract is also remarkable for the very rapid turnover of the epithelial cell population. It is likely that the epithelium turns over in its entirety every 24 to 72 h. This capacity may permit rapid restitution of a functional cell population following an acute insult and may reduce the risk of malignancy through the shedding of cells affected by the many mutagens in the luminal contents. Nevertheless, this

proliferative potential creates the setting for neoplastic disorders, which are so common in the gastrointestinal tract.

In view of the important secretory and absorptive activities of mucosal surface, diseases of the gastrointestinal tract may result in clinical consequences owing to physical disruption of the mucosal layer (e.g., blood loss, fluid loss, pathogenic invasion) or to nutritional derangements caused by impaired digestion and nutrient absorption. In focal or localized disease processes the former effects predominate, whereas the latter may be especially prominent in disorders that affect extensive areas of the gastrointestinal tract in a diffuse manner.

While the essential roles of the gastrointestinal tract—the absorption of nutrients and the excretion of the products—are accomplished in large part at the luminal surface, these processes also depend on the deeper muscular layers for the coordinated propulsion of food through the lumen. The complexity of the local and distant neural and endocrine factors that contribute to the regulation of intestinal motility is only now becoming fully appreciated. Disruption of normal motility is quite common, with functional bowel complaints affecting as many as 15 per cent of adults. Alterations in frequency of bowel movements, abdominal distention, abdominal pain, and nausea, individually or in varying combinations, may result from dysmotility. In addition, structural lesions may also indirectly lead to symptoms through their impact on motility involving some or all regions of the gastrointestinal tract. These range from the direct effects of an obstructing lesion to the indirect actions of substances released by a primary mucosal disorder (e.g., inflammatory mediators such as arachidonic acid metabolites that also affect smooth-muscle activity).

Although valid unifying generalizations can be made about the gastrointestinal tract in its entirety, the spectrum of diseases affecting the system and their clinical manifestations are significantly related to the constituent organ(s) involved.

METHODS OF EXAMINATION

Inquiry

Complaints of the patients with the diseases of digestive organs are various, and depend on which part of the alimentary tract is affected (Tab. 5.1).

The most common complaints are:

- Dysphagia
- Heartburn (pyrosis)
- Regurgitation, cructio, ructus
- Nausea
- Vomiting
- Abdominal pain
- Diarrhea
- Constipation
- Gain and loss in weight
- Gastrointestinal bleeding

Dysphagia is defined as a sensation of "sticking" or obstruction of the passage of food through the mouth, pharynx, or esophagus. It should be distinguished from other symptoms related to swallowing.

Tab. 5.1. ***Overview of approach to patients with common gastrointestinal disorders.***

Site of disorder	Common symptoms	Possible physical signs
Esophagus	Dysphagia Odynophagia Heartburn, chest pain Hematemesis/melena	
Stomach	Nausea and vomiting Epigastric pain Hematemesis/melena Early satiety	Distension Tenderness Succusion splash
Pancreas	Pain Weight loss Diarrhea Steatorrhea	Jaundice

Duodenum	Pain Nausea/ vomiting Hematemesis	Tenderness Altered bowel sounds Distension
Jejunum	Pain Diarrhea	Altered bowel sounds Distension
Ileum	Pain Diarrhea	Altered bowel sounds Distension
Colon	Diarrhea Pain Blood	Tenderness Distension
Rectum	Pain urgency Hematochezia Pruritus	Tenderness

Aphagia signifies complete esophageal obstruction, which is usually due to bolus impaction and represents a medical emergency.

Difficulty in initiating a swallow occurs in disorders of the voluntary phase of swallowing. However, once initiated, swallowing is completed normally.

Odynophagia means painful swallowing. Frequently, odynophagia and dysphagia occur together.

Globus pharyngeous is the sensation of a lump lodged in the throat. However, no difficulty is encountered when swallowing is performed.

Phagophobia, meaning fear of swallowing, and *refusal to swallow* may occur in hysteria, rabies, tetanus, and pharyngeal paralysis due to fear of aspiration.

Painful inflammatory lesions that cause odynophagia also may cause refusal to swallow.

Some patients may feel the food as it goes down the esophagus. This esophageal sensitivity is not associated with the food sticking or obstruction, however.

Similarly, the *feeling of fullness in the epigastrium* that occurs after a meal or after swallowing air should not be confused with dysphagia,

The normal transport of an ingested bolus through the swallowing passage depends on the size of the ingested bolus; the luminal diameter of the swallowing passage; the peristalsis contraction; and deglutitive inhibition, including normal relaxation of upper and lower esophageal sphincters during swallowing.

Dysphagia caused by a large bolus or luminal narrowing is called *organic (or mechanical) dysphagia,* whereas dysphagia due to incoordination or weakness of peristaltic contractions or to impaired deglutitive inhibition is called *functional (or motor) dysphagia* (Fig. 5.2).

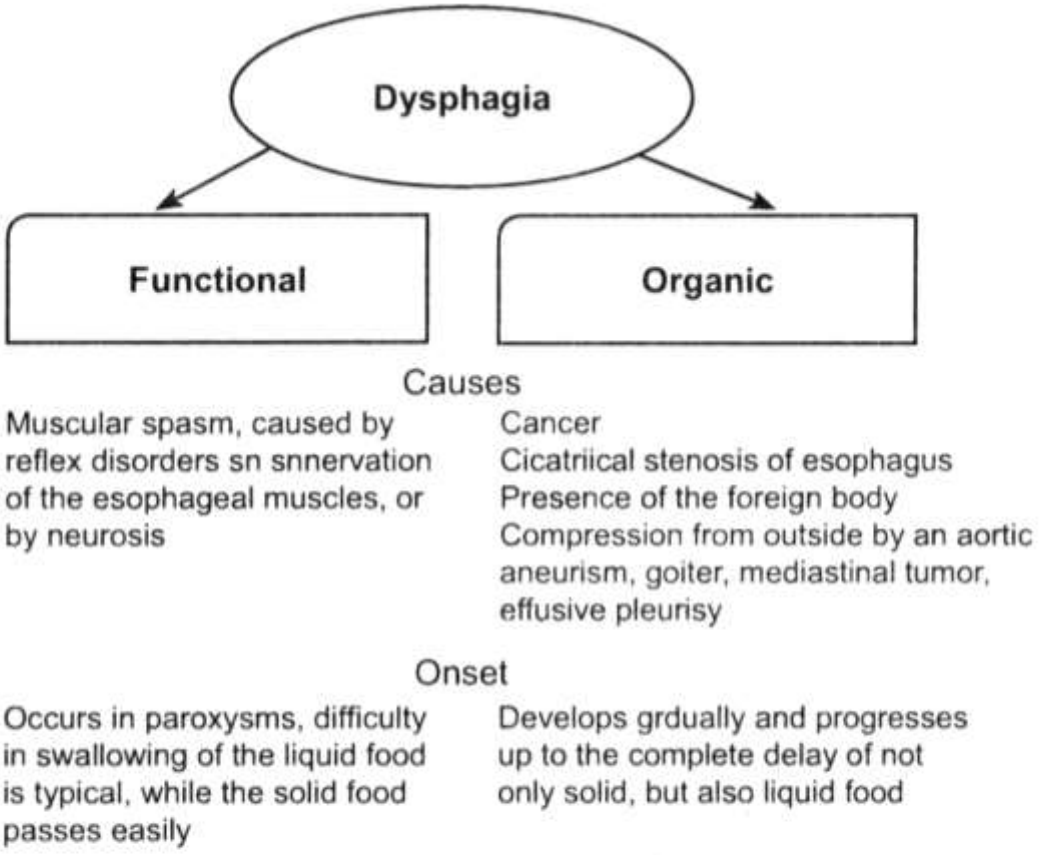


Fig. 5.2. Types of dysphagia.

Heartburn (pyrosis) is a specific burning sensation behind the sternum associated with regurgitation of gastric contents into the inferior portion of the esophagus.

Occasional heartburn is common in normal persons, but frequent and severe heartburn is generally a manifestation of esophageal dysfunction.

Heartburn may result from abnormal motor activity or distension of the esophagus, sensitivity of the esophageal mucosa to refluxed acid or bile, or esophageal mucosal inflammation (esophagitis).

Heartburn is most often associated with gastroesophageal reflux. In this setting, heartburn typically occurs after a large meal, with stooping or bending, or when the patient is supine. It may be accompanied by the spontaneous appearance in the mouth of fluid, which may be salty ("water brash"), sour (gastric contents), or bitter and green or yellow (bile). Heartburn may arise following the ingestion of certain foods (e.g., citrus fruit juices) or drugs (alcohol and aspirin). Heartburn arises also in various diseases of the alimentary tract with hyperacidity – gastritis, peptic ulcer disease, cholecystitis, hiatus hernia, and in pregnancy.

Regurgitation (crutio, ructus) is return of the part of swallowed food into the mouth due to backward movement of esophagus and stomach with open cardia without contraction of diaphragm and abdominal muscles (Tab. 5.2).

Tab. 5.2. ***Regurgitation.***

	By air *(eructation)*	By food or by gas *(regurgitatio)*	
Cause	Due to air swallowing (aerophagy) during the fast meals, heard at a distance	Motor dysfunction of the stomach with increased formation of gas due to abnormal	
		Fermentation	Putrefaction
Odor	Odorless	Odorless or smell of sour, or bitter oil, which is due to the presence of organic acids	Odor of rotten eggs, that indicates intensive degradation of proteins

Disease	Psychoneurosis Fast meals	Usually associated with hypersecretion of gastric juice and occurs during pain attacks in ulcer. Can occur in normal or insufficient secretion of the stomach in failure of the cardia, when the stomach contents are regurgitated into the esophagus	Pylorus stenosis with great distension of the stomach and significant congestion in it

Nausea is reflex act associated with irritation of the vagus nerve. Nausea denotes the feeling of an imminent desire to vomit, usually referred to the throat or epigastrium. The mechanism of nausea is unknown. Nausea often precedes or accompanies vomiting. Often accompanying severe nausea is evidence of altered autonomic (especially parasympathetic) activity, such as skin pallor, increased perspiration, hypersalivation, defecation, and, occasionally, hypotension and bradycardia (vasovagal syndrome); anorexia is also usually present. It is usually associated with diminished functional activity of the stomach (hypotonicity, hypoperistalsis, and hyposecretion) and altered small-intestinal motility (hypertonicity and reversed peristalsis of the duodenum).

The most common causes of nausea are listed in Fig. 5.3.

Vomiting (emesis) refers to the forceful oral expulsion of gastric contents. Nausea, retching, and hypersalivation frequently precede the act of vomiting, which is highly integrated sequence of involuntary visceral and somatic motor events. The stomach plays a relatively passive role in the vomiting process, the major ejection force being provided by the abdominal musculature. With relaxation of the gastric fundus and gastroesophageal sphincter, a sharp increase in intraabdominal pressure is brought about by forceful contraction of the diaphragm and abdominal wall muscles. This, together with concomitant annular contraction of the gastric pylorus, results in

the expulsion of gastric contents into the esophagus. Increased intrathoracic pressure results in the further movement of esophageal contents into the mouth. Reversal of the normal direction of esophageal peristalsis may play a role in this process. Reflex elevation of the soft palate during the vomiting at prevents the entry of the expelled material into the nasopharynx, whereas reflex closure of the glottis and inhibition of respiration help to prevent pulmonary aspiration.

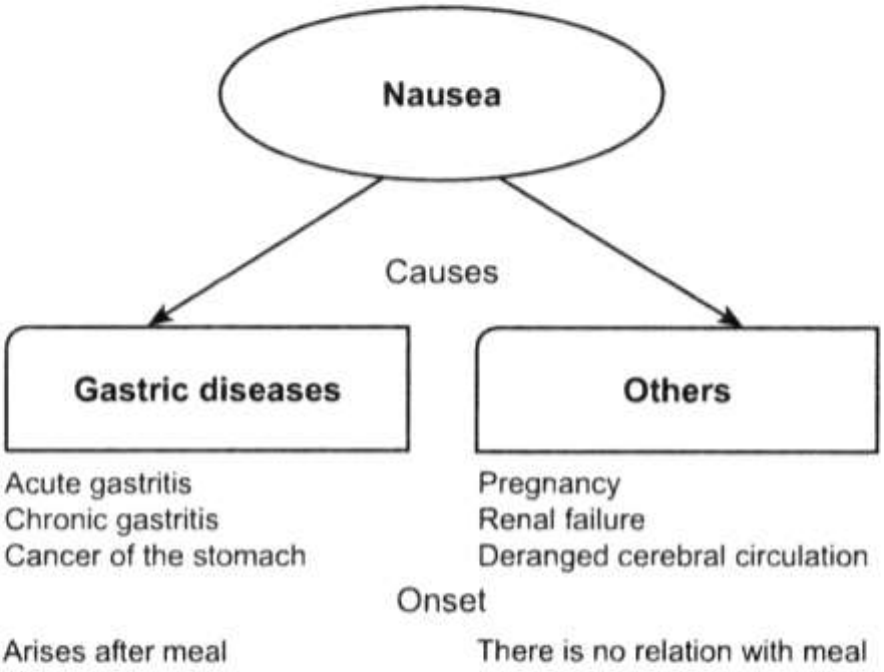


Fig. 5.3. Causes of nausea.

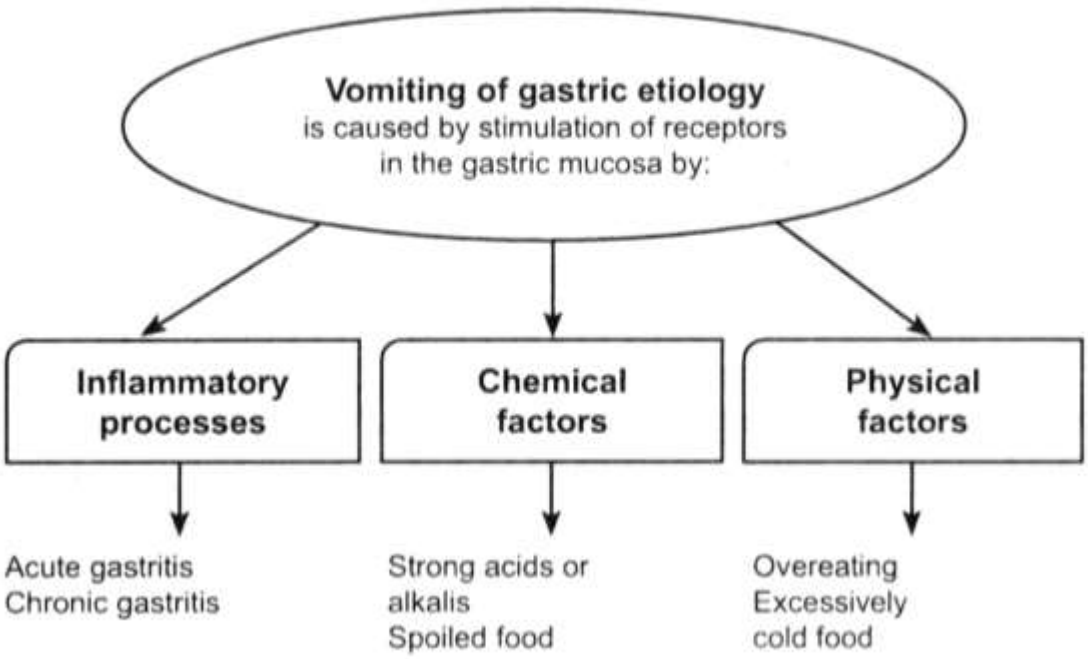
Vomiting mechanism. The act of vomiting is under the control of two functionally distinct medullary centers: **the vomiting center** in the dorsal portion of the lateral reticular formation and the **chemoreceptor trigger zone** in the area postrema of the floor of the fourth ventricle. The vomiting center controls and integrates the actual act of emesis. It receives afferent stimuli from the gastrointestinal tract and others part of the body, from higher brainstem and cortical centers, especially the labyrinthine apparatus, and from the chemoreceptor trigger zone. The important efferent pathways in vomiting are the phrenic nerves (to the diaphragm), the spinal nerves (to the intercostals and abdominal musculature), and visceral efferent fibers

in the vagus nerve (to the larynx, pharynx, esophagus, and stomach). The vomiting center is located near other medullary centers regulating respiratory, vasomotor, and autonomic functions that may be involved in the act of vomiting.

Vomiting is common manifestations of many organic and functional disorders (Tab. 5.3).

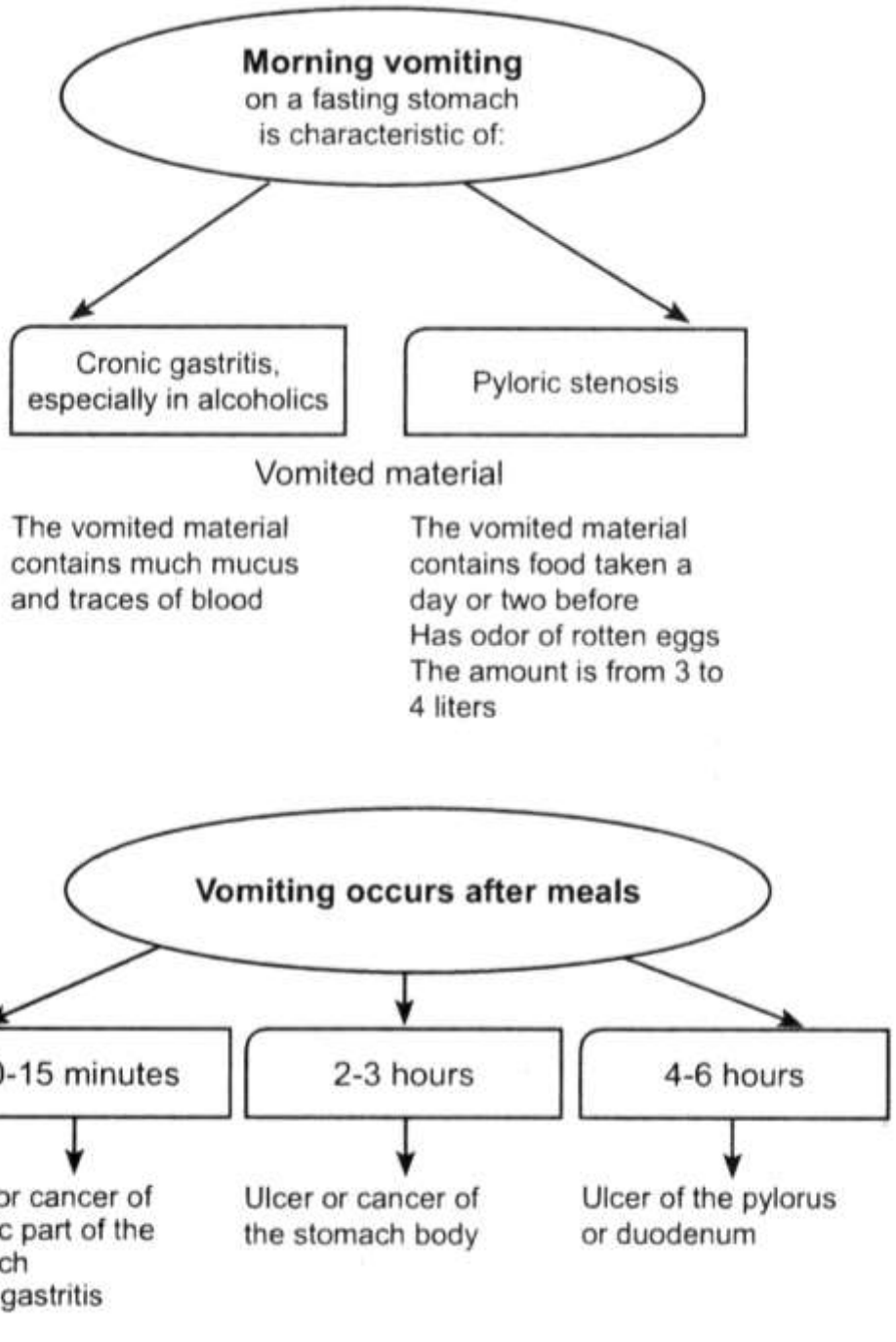
Tab. 5.5. ***Vomiting etiology.***

Central	Peripheral (of visceral etiology, reflex)	Hematogenic and toxic
Nervous • Encephalitis • Meningitis • Cerebral tumor • Stroke Sudden blood pressure elevation	• Peptic ulcer disease • Gastric tumor • Diseases of bile ducts • Pancreatitis • Myocardial infarction • Renal colic	• Liver failure • Renal failure • Diabetes mellitus

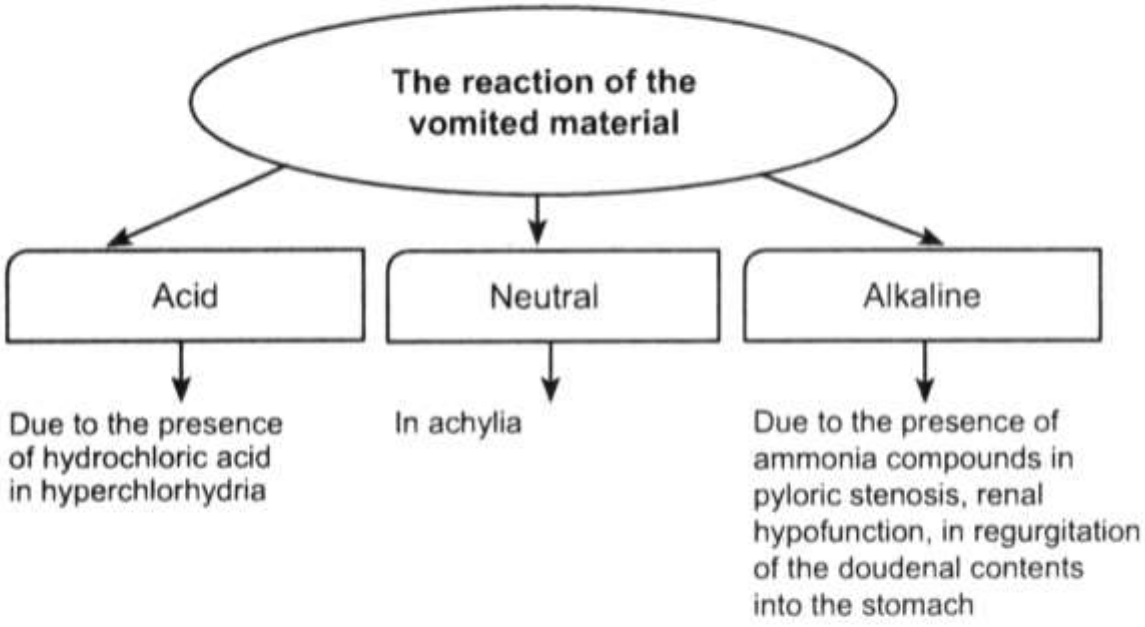


Vomiting can also be caused by difficult evacuation of the stomach due to spasms or stenosed pylorus.

If the patient complains of vomiting you should ask about the time when the vomiting arises: before or after meals, possible connection with pain, the amount and character of the vomiting material. Vomiting may attend acute gastritis, exacerbation of chronic gastritis, peptic ulcer disease, spasm and organic stenosis of pulorus, and cancer of stomach.



The **odor** of the vomit is usually acid, but it can be fetid due to putrefactive processes in the stomach, or even fecal in presence of a fecal fistula between the stomach and the transverse colon.



Abdominal pain is important and the leading symptom in diseases of digestive system.

You should ask the patient about:

1. *location* of the pain (epigastric region, right or left hypochondrium, umbilical region, etc)
2. *character* of the pain (periodical or paroxysmal (at certain time of the day); permanent or seasonal (in spring or autumn); intensity (dull, stabbing, etc)
3. *connection with meals* (fasting pain, after meal:
   early – occurring 30–40 min after meals;
   late – 90–120 min after meals;
   nocturnal, hunger pain, which is abated after taking food.)
4. *radiation* of the pain
5. *relieving factors* (vomiting, taking food or soda, spasmolytics, warmly)
6. possible *connection* between *pain* and *physical strain* (weight lifting, traffic jolting)

Pain in **peptic ulcer** is epigastric, may radiate to the back, and is of variable quality: gnawing, burning, boring, aching, pressing, or hunger like. The pain is intermittent. Duodenal ulcer is more likely than gastric ulcer can cause pain that wakes the patient at night, and occurs intermittently over a few weeks, then disappears for month, and then recurs. Food and antacids may bring relief, but not necessarily in any of these disorders and least commonly in gastric ulcer. Associated symptoms are nausea, vomiting, belching, bloating; heartburn (more common in duodenal ulcer); weight loss (more common in gastric ulcer). Gastric ulcer is more common in the older (over 50 yr), and duodenal ulcer in those from 30–60 yr.

Pain in the **cancer of the stomach** is epigastric, and of variable quality. The history of pain is typically shorter than in peptic ulcer. The pain is persistent and slowly progressive. Often food can aggravate the pain, and it not relived by food or antacids. Associated symptoms are anorexia, nausea, easy satiety, weight loss, and sometimes bleeding. Cancer of the stomach is most common in ages 50–70.

In **acute pancreatitis** the pain is epigastric, may radiate to the back or other parts of the abdomen or may be poorly localized. The quality of the pain is usually steady. Acute onset, and persistent pain is typical. Pain aggravates in lying supine posture, and relives in leaning forward with trunk flexed. Nausea, vomiting, abdominal distension, and fever accompany pain in acute pancreatitis. Often history of previous attacks and of alcohol abuse or gallstones.

Chronic pancreatitis is characterized by steady, deep epigastric pain, radiating through to the back. Pain is chronic or of recurrent course. Alcohol, heavy or fatty meals can aggravate pain, and possibly leaning forward with trunk flexed can relieve it. Symptoms of decreased pancreatic function may appear: diarrhea with fatty stools (steatorrhea) and diabetes mellitus.

Epigastric and in either upper quadrant steady and deep pain that often radiates to the back is typical to the **cancer of the pancreas**. The pain is persistent, and can relieve in leaning forward with trunk flexed. Associated symptoms are anorexia, nausea, vomiting, weight loss, jaundice, and emotional symptoms, including depression.

Sudden obstruction of the cystic duct or common bile duct by a gallstone in **biliary colic** causes epigastric or right upper quadrant steady, ach-

ing pain (not colicky) that may radiate to the right scapula and shoulder. This pain is characterized by rapid onset over a few minutes, lasts one to several hours and subsides gradually. Anorexia, nausea, vomiting, restlessness often accompany the pain in biliary colic.

In **acute cholecyctitis** due to inflammation of the gallbladder, usually from obstruction of the cystic duct by a gallstone steady, aching pain arises in right upper quadrant or upper abdominal, which may radiate to the right scapular area. Gradual onset and course longer than in biliary colic is typical. Jarring and deep breathing aggravate pain, and it is usually accompanied by anorexia, nausea, vomiting, and fever.

In **acute diverticulitis** the pain location is left lower quadrant; it may be cramping at first, but becomes steady. The pain is often a gradual onset, and accompanied by fever, and constipation. There may be initial brief diarrhea.

Acute inflammation of the appendix with distension or obstruction – **acute appendicitis** initially poorly localized *periumbilical pain* of mild character but increasing, possibly cramping is observed. It lasts roughly 4–6 hours, and associated with anorexia, nausea, possibly vomiting, which typically follow the onset of pain, and low fever. This pain followed usually by r*ight lower quadrant s*teady and more severe *pain.* Movement or cough often aggravates pain. If it subsides temporarily, suspect perforation of the appendix

Pain in the stomach (hollow organs with smooth muscles) may be: ***spastic*** (provoked by spasm), ***distensional*** (provoked by distension), or provokes by its motor dysfunction.

Diarrhea is formally defined as an increase in daily stool weight above 200 g. Typically the patients also may describe an abnormal increase in stool liquidity and frequency. Diarrhea is considered *acute* when lasting less than 7 to 14 days and *chronic* when lasting more than 2 to 3 weeks.

Acute diarrhea. The most common causes of acute diarrhea are infectious agents. Acute diarrhea also may be caused by ingested drugs or toxins, the administration of hemotherapy, resumption of enteral feeding following a prolonged fast, fecal impaction (overflow diarrhea), or particular situations, such as marathon running.

Virtually any medication can cause diarrhea, and a careful drug history should be obtained in any patient with acute diarrhea.

Other ingested toxins also must be considered, including organophosphate insecticides, mushrooms, arsenic, and even caffeine. Acute di-

verticulitis occasionally may present with diarrhea accompanied by fever and abdominal pain. In patients with acute bloody diarrhea, diagnostic considerations may include superior mesenteric arterial or venous thrombosis, ischemic or drug-induced colitis, or idiopathic inflammatory bowel disease (ulcerative colitis or Crohn's disease).

Chronic diarrhea. Diarrhea that persists for weeks or month, whether constant or intermittent, requires evaluation. Although in the majority of cases the cause will prove to be irritable bowel syndrome, diarrhea may represent a manifestation of an underlying serious illness, and a careful search for disease should be taken.

Chronic diarrhea can be categorized pathophysiologically as inflammatory, osmotic (malabsorption), secretory, due to intestinal dysmotility, or factitious.

Inflammatory diarrhea is characterized by the presence of fever, abdominal tenderness, and blood or leukocytes in the stool, with inflammatory lesions on intestinal mucosal biopsy. In some cases, hypoalbuminemia, hypoglobulinemia, and protein-losing enteropathy may be present. In addition to inflammation, the mechanism of diarrhea may also be malabsorption or increased intestinal secretion.

Osmotic diarrhea occurs when an orally ingested solute is not fully absorbed in the small intestine and thereby exerts an osmotic force that draws fluid into the intestinal lumen. The increased luminal fluid volume overwhelms the capacity of the colon for reabsorption. The nonabsorbed solute can be a maldigested or malabsorbed nutrient or drug. Clinical symptoms are usually recognized because of the malanbsorption of fat (steatorrhea) or carbohydrates. Protein or amino acid malabsorption (azotorrhea) is generally not recognized clinically unless it severe enough to cause malnutrition or the consequences of a specific deficiency in an amino acid.

Secretory diarrhea is characterized by a large volume of fecal output caused by abnormal fluid and electrolyte transport not necessarily related to the ingestion of food. Therefore, diarrhea usually persists with fasting. The term watery diarrhea is often used synonymously with secretory diarrhea. Because there is no malabsorbed solute, fecal osmolality in secretory diarrheas can be accounted for by normal ionic constituents with no fecal osmotic gap.

Altered intestinal motility. Diarrhea may be associated with disorders that affect intestinal motility. The most common of these is irritable bowel syndrome, in which diarrhea typically alternates with constipation and is associated with abdominal pain, the passage of mucus, and a sense of incomplete evacuation. Diarrhea may occasionally occur paradoxically as a result of fecal impaction or an obstructing tumor with the overflow of liquid colonic contents around the impacted stool or obstruction. A variety of neurologic diseases also may be associated with diarrhea because of altered autonomic control of bowel function. Profuse watery diarrhea, often with incontinence, may be seen in the patients with type I diabetes and is often associated with severe neuropathy, nephropathy, and retinopathy. Additional contributing factors may include bacterial overgrowth secondary to intestinal dysmotility, pancreas endocrine insufficiency, or rarely, celiac sprue.

Factitious diarrhea is self-induced by the patient and may result from intestinal infection, the addition of water or urine to the stool, or self-medication with laxatives. Patients are predominantly women with severe chronic watery diarrhea, abdominal pain, nausea and vomiting, weight loss, peripheral edema, and weakness resulting from hypokalemia. The diagnosis of factitious diarrhea should be suspected in a patient with a history of psychiatric disease or multiple previous negative evaluations for diarrhea.

Constipation is a common complaint in clinical practice because of wide range of normal bowel habits, constipation is difficult to define precisely. Most persons have at least three bowel movements per week, and constipation has been defined as a frequency of defecation of less than three times per week. However, stool frequency alone is not a sufficient criterion to use, because many constipated patients describe a normal frequency of defecation but subjective complaints of excessive straining, hard stools, lower abdominal fullness, and a sense of incomplete evacuation. Thus a combination of objective and subjective criteria must be used to define constipation.

Pathophysiologically, constipation generally results from disordered colonic transit or anorectal function as a result of a primary motility disturbance, certain drugs, or in association with a large number of systemic diseases that affect the gastrointestinal tract. Constipation of any cause may be exacerbated by chronic illnesses that lead to physical or mental impairment and result in inactivity or physical immobility. Additional contributing

factors may include a lack of fiber in the diet, generalized muscle weakness, and possibly stress and anxiety.

In the patient presenting with the recent onset of constipation, an obstructing lesion of the colon should be sought. In addition to a colonic neoplasm, other causes of colonic obstruction include strictures due to colonic ischemia, diverticular disease, or inflammatory bowel disease; foreign bodies', or anal strictures. Anal sphincter spasm due to painful hemorrhoids or fissures also may inhibit the desire to evacuate.

In the absence of an obstructing lesion of the colon, disturbed colonic motility may mimic colonic obstruction. Disruption of parasympathetic innervation to the colon as a result of injury or lesions of the lumbosacral spine or sacral nerves may produce constipation with hypomotility, colonic dilatation, decreased rectal tone and sensation, and impaired defecation. In patients with multiple sclerosis, constipation may be associated with neurogenic dysfunction of other organs. Similarly, constipation may be associated with lesions of the central nervous system caused by parkinsonism or a cerebrovascular accident.

Drugs that may lead to constipation include those with anticholinergic properties, such as antidepressants and antipsychotics, codeine and other narcotic analgesics, aluminum- or calcium-containing antacids, sucralfate, iron supplements, and calcium channel blockers. In patients with certain endocrinopathies such as hypothyroidism and diabetes mellitus, constipation is generally mild and responsive to therapy. Rarely, life-threatening megacolon occurs in patients with myxedema. Constipation is common during pregnancy, presumably as a result of altered progesterone and estrogen levels, which decrease intestinal transit. Collagen vascular diseases may be associated with constipation, which may be a particularly prominent feature of progressive systemic sclerosis, in which delayed intestinal transit results from atrophy and fibrosis of colonic smooth muscle.

Gain and loss in weight

In normal persons weight is stable over long periods because food intake is matched to energy expenditure by neural activity in the hypothalamus that provides signals to eat or to stop eating. Because the system is usually effective, either weight gain or weight loss may bring a patient to the physician. No history is complete without ascertaining whether weight has been

gained or lost and, if so, how much. In general, a change of 5 per cent of body weight or 5 kg is considered significant. However, a 5 kg weight loss may not be of importance in a 130 kg man who is trying to lose weight, while a 2 kg weight loss may well be worrisome in a person weighing 40 kg.

Weight gain is less likely to have a pathologic cause than weight loss. In most cases it is a consequence of overeating and inadequate exercise, and the diagnosis is usually simple obesity. Occasionally obesity may be the consequence of hypothyroidism, Cushing's syndrome, or hypothalamic disease such as craniopharyngioma. Workup for pathologic causes of weight gain is rarely indicated unless accompanying signs suggest an underlying cause. Sometimes weight gain is not fat but fluid, in which case the primary problems to be considered are congestive heart failure, renal failure, or cirrhosis with ascites.

Although rarely caused by disease, obesity predisposes to disease, especially diabetes mellitus but also to gall stones, degenerative joint disease, hyperlipidemia. atherosclerosis, hypertension, sleep apnea, and perhaps cancer.

Weight loss. In all studies there are patients who lose weight without discernible cause, but significant involuntary weight loss is usually a marker of serious disease. Even if no disease is found on initial evaluation, it should not be assumed that weight loss is idiopathic. The patient should be followed at regular intervals with careful repeat examination since occult illness causing weight loss may not become manifest for long periods.

There are three general mechanisms of weight loss, but more than one may be operative in the same patient: increased energy expenditure, increased loss of energy in stool or urine, and decreased food intake. Decreased intake is by far the most common mechanism; it is usually due to loss of appetite but may result from obstruction in the esophagus or stomach secondary to stricture, compressive mass, or infiltrating malignancy. The only common causes of increased metabolism are hyperthyroidism, pheochromocytoma, and major exercise programs. Loss of ingested energy generally is due to either diabetes mellitus with glycosuria or intestinal malabsorption with steatorrhea. Chronic pancreatitis in alcoholics is the most common cause of steatorrhea, but malabsorption can occur with intestinal lymphoma, celiac sprue, islet cell tumors (such as somatostatinomas or gastrinomas), radiation

injury, biliary tract obstruction, inflammatory bowel disease, and a variety of other disorders. If weight loss occurs with a history of increased food intake, the diagnosis is usually diabetes mellitus, hyperthyroidism, or malabsorption syndrome. Occasionally leukemias and lymphomas may present with weight loss in the absence of anorexia or even with increased food intake.

Under most circumstances the diagnosis of the cause of weight loss is not difficult and is revealed by history, physical examination, and routine laboratory screening. The most common causes of weight loss is given in Table 5.4.

Tab. 5.4. ***Common causes of weight loss.***

Increased energy expenditure
Hyperthyroidism
Pheochromocytoma
Extensive exercise
Increased energy loss
Diabetes mellitus
Malabsorption syndromes
Diminished food intake
Cancer
Infection (HIV, tuberculosis, endocarditis)
Hypercalcemia (malignancy, hyperparathyroidism, sarcoidosis)
Uremia
Obstructive gastrointestinal disease
Anorexia nervosa
Adrenal insufficiency (primary or secondary)
Pernicious anemia
Alzheimer's disease
Depression

Gastrointestinal bleeding. ***Hematomesis*** is defined as the vomiting of blood, and ***melena*** as the passage of stools rendered black and tarry by the presence of blood. These clinical manifestations of gastrointestinal hemorrhage suggest a proximal source of bleeding. The color of vomited blood

depends on the concentration of hydrochloric acid in the stomach and the duration of its contact with the blood. Thus, if vomiting occurs shortly after the onset of bleeding, the vomitus appears red, and later the appearance will be dark red, brown, or black. Precipitated blood clots and acid-degraded blood in the vomitus will produce a characteristic "coffee grounds" appearance when vomited. Hematemesis usually indicates bleeding proximal to the ligament of Treitz because blood entering the fit tract below the duodenum rarely enters the stomach.

While bleeding sufficient to produce hematemesis usually results in melena, less than half of patients with melena have hematemesis. Melena usually denotes bleeding from the esophagus, stomach, or duodenum, but lesions in the jejunum, ileum, and even ascending colon may occasionally cause melena provided the gastrointestinal transit time is sufficiently prolonged. Approximately 60 ml of blood is required to produce a single black stool; acute blood loss greater than this may produce melena for as long as 7 days. After the stool color returns to normal, tests for occult blood may remain positive for over a week. The black color of melena results from contact of the blood with hydrochloric acid to produce hematin. Such stools are tarry ("sticky") and have a characteristic odor. This tarry consistency is in contrast to black or dark gray stools occurring after the ingestion of iron, bismuth, or licorice. Gastrointestinal bleeding, even if detected only by positive tests for occult blood, indicates potentially serious disease and must be further investigated.

Hematochezia, the passage of red blood per rectum, generally signifies bleeding from a source distal to the ligament of Treitz. However, brisk proximal bleeding can cause hematochezia due to rapid transit.

The clinical manifestations of gastrointestinal bleeding depend on the extent and rate of hemorrhage and the presence of coincidental diseases. Blood loss of less than 500 ml is rarely associated with systemic signs; exceptions include bleeding in the elderly or in the anemic patient in whom smaller amounts of blood loss may produce hemodynamic alterations. Rapid hemorrhage of greater volume results in decreased venous return to the heart, decreased cardiac output, and increased peripheral resistance due to reflex vasoconstriction. Orthostatic hypotension greater than a change of 10 mmHg usually indicates a 20 per cent or greater reduction in blood volume. Concomitant symptoms may include lightheadedness, syncope, nausea, sweat-

ing, and thirst. When blood loss is 25 to 40 per cent of blood volume, shock frequently ensues with pronounced tachycardia and hypotension. Pallor is prominent, and the skin is cool. However, in the presence of beta-adrenergic and calcium channel blockers, these clinical signs may be blunted.

In the setting of rapid hemorrhage, the initial hematocrit may not accurately reflect the magnitude of blood loss, since equilibration with extravascular fluid and hemodilution often require over 8 h. Common laboratory findings include mild leukocytosis and thrombocytosis, which develop within 6 h after the onset of bleeding. The blood urea nitrogen (BUN) may be elevated out of proportion to the creatinine, particularly in upper gastrointestinal bleeding, due to breakdown of blood proteins to urea by intestinal bacteria as well as mild reduction in the glomerular filtration rate.

Occult bleeding, detected by card test for hemoglobin peroxidase, is an important means of finding colorectal neoplasia at earlier, potentially curable stages. Testing is advocated for patients over age 50 as a part of the yearly checkup. Multiple stools should be tested (usually two samples from three stools), and if any sample is positive, additional studies should be performed. A positive result can be due to physiologic blood loss, dietary peroxidases, undercooked meat, or any cause of upper or lower gastrointestinal bleeding. The daily ingestion of over 500 mg of vitamin C may result in a false-negative test. To limit the confounding variables, patients should be tested on a high-fiber and low-meat diet with no ingestion of nonsteroidal anti-inflammatory agents (NSAIDs) or vitamin C, although the daily low dose of aspirin (80 to 325 mg) taken to prevent cardiovascular disease generally does not lead to false-positive results.

Upper gastrointestinal bleeding. A careful history and physical examination of the oropharynx and nasal cavity should serve to exclude epistaxis or swallowed blood as a source of hematemesis or melena.

The most common causes of upper gastrointestinal hemorrhage are:

- Erosive or hemorrhagic gastropathy
- Duodenal ulcer
- Gastric ulcer
- Mallory-Weiss tear
- Varices or portal hypertensive gastropathy
- Arteriovenous malformations

Lower gastrointestinal bleeding (Tab. 5.5)

Tab. 5.5. ***Common causes of acute lower gastrointestinal bleeding*** (in order of frequency).

Under age 55	Over age 55
Anorectal disease (hemorrhoids, fissures)	Anorectal disease (hemorrhoids, fissures)
Colitis (inflammatory bowel disease)	Diverticulosis
Diverticulosis	Angiodysplasia
Polyps, cancer (hyperplastic, hamartomas)	Polyps, cancer
Angiodysplasia	Enterocolitic (ischemic, infectious, inflammatory bowel disease, radiation)

Inspection of the Abdomen

Starting from your usual standing position at the right side of the bed, inspect the abdomen. When looking at the contour of the abdomen and watching for peristalsis, it is helpful to sit or bend down so that you can view the abdomen tangentially. Illumination of the body should be uniform.

Examine the abdomen in an orderly fashion. Note the shape, size, symmetry of the abdomen, participation of the anterior abdominal wall in the breathing act, umbilicus position, expression of the subcutaneous veins, and possible presence of scars, eruptions, scratches, visible pulsation, peristalsis, and teleangioectasia.

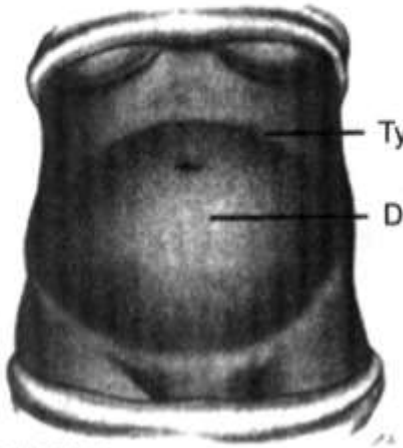
In **norm** the abdomen is of a oval shape, doesn't enlarged in size, symmetrical, anterior abdominal wall take part in the breathing act, umbilicus is retracted, pronounced venous network, scars, eruption, telangioectasia, scratches, visible pulsation and peristalsis are absent.

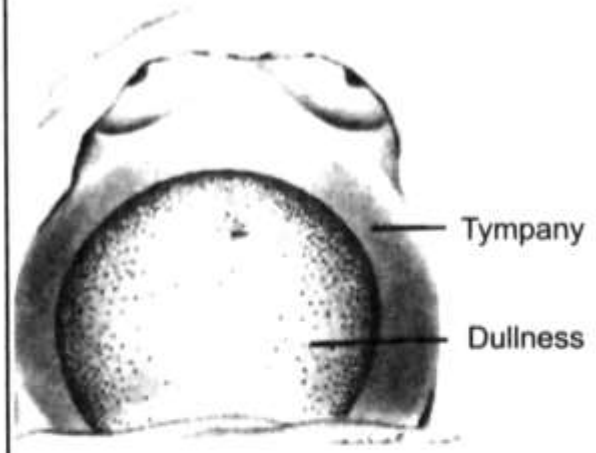
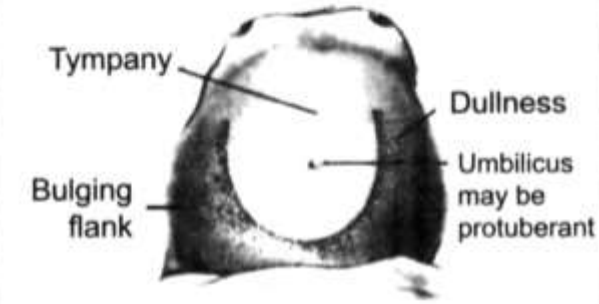
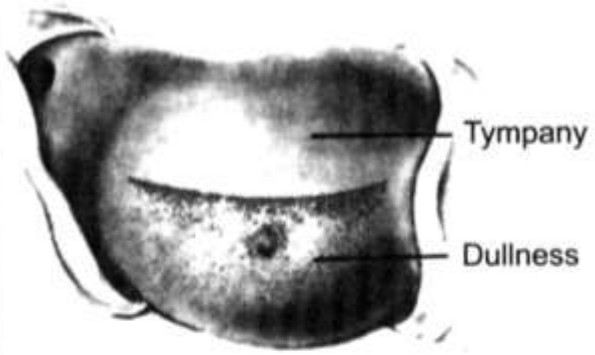
The most common causes of protuberant abdomen are pregnancy, obesity, meteorism, and ascitis (Tab. 5.6, Tab. 5.7).

Tab. 5.6. ***Differential signs of meteorism and ascitis.***

Sign	Meteorism	Ascitis
Skin	Doesn't change	"Rice-paper" (thin, shining)
Umbilicus	Retracted	Smoothed, or protruded
Fluctuation symptom	Absent	Present
Percussion	Tympanic sound	Dull sound in sloping sites

Tab. 5.7. ***Protuberant abdomens.***

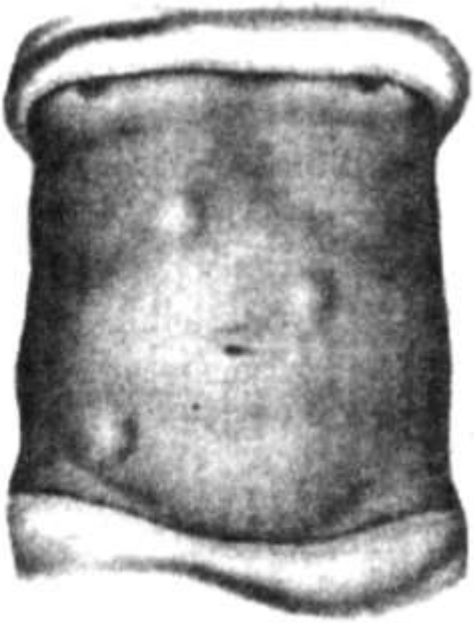
 **FAT**	Fat is the most common cause of a protuberant abdomen and is associated with generalized obesity. The abdominal wall is thick. Fat in the mesentery and omentum also contributes to abdominal size. The umbilicus may appear sunken. The percussion note is normal. An apron of fatty tissue may extend below the inguinal ligaments. Lift it to look for inflammation in the skin fold or even for a hidden hernia.
 **GAS**	Gaseous distension may be localized, as shown, or generalized. It causes a tympanitic percussion note. Increased intestinal gas production due to certain foods may cause mild distension. More serious are intestinal obstruction and adynamic (paralytic) ileus. Note the location of the distension. Distension becomes more marked in colonic than in small bowel obstruction.
 **TUMOR**	A large, solid tumor, usually rising out of the pelvis, is dull to percussion. Air-filled bowel is displaced to the periphery. Causes include ovarian tumors and uterine myomata. Occasionally, a markedly distended bladder may be mistaken for such a tumor.

 **PREGNANCY**	Pregnancy is a common cause of a pelvic "tumor". Listen for the fetal heart.
  **ASCITIC FLUID**	Ascitic fluid seeks the lowest point in the abdomen, producing bulging flanks that are dull to percussion. The umbilicus may protrude. Turn the patient onto one side to detect the shift in position of the fluid level (shifting dullness).

Localized bulges in the abdominal wall include ventral hernias (defects in the wall through which tissue protrudes) and subcutaneous tumors such as lipomas. The more common ventral hernias are umbilical, incisional, and epigastric. Rectus diastasis is also sometimes so classified. Hernias and a rectus diastasis usually become be evident when the patient raises head and shoulders from a supine position (Tab. 5.8).

Tab. 5.8. ***Localized bulges in the abdominal cavity.***

 **UMBILICAL HERNIA**	Umbilical hernias protrude through a defective umbilical ring. They are most common in infants but also occur in adults. In infants, but not in adults, they are usually close spontaneously within a year or two.
 **INCISIONAL HERNIA**	An incisional hernia protrudes through an operative scar. By palpation, note the length and width of the defect in the abdominal wall. A small defect, through which a large hernia has passed, has a greater risk of complications than a large defect.
 **EPIGASTRIC HERNIA**	An epigastric hernia is a small midline protrusion through a defect in the linea alba, somewhere between the xiphoid process and umbilicus. With the patient's head and shoulders raised (or with the patient standing), look for it, and run your finger pad down the linea alba to feel it.



LIPOMA

Lipomas are common, benign, fatty tumors usually located in the subcutaneous tissues almost anywhere in the body, including the abdominal wall. Small or large, they are usually soft and often lobulated. When your finger presses down on the edge of a lipoma, the tumor typically slips out from under it.

A rectus diastasis is a separation of the two rectus abdominis muscles, through which abdominal contents buldge to form a midline ridge when the patient raises head and shoulders. Repeated pregnancies, obesity, and chronic lung disease may predispose to it. It has no clinical consequences.

Palpation of the Abdomen

Palpation of the abdomen is the main method of physical examination of the abdominal organs along with X-ray examination. This method was first proposed by French physician Glenard. Later the Russian physicians Obraztsov and Strazhesko developed this method.

Technique. For a good abdominal examination you need good light, a relaxed patient, and full exposure of the abdomen from above the xiphoid process to the symhysis pubis. The groins should be visible, although the genitalia should be kept draped. The patient should not have a full bladder. Make the patient comfortable in a supine position; the bed should not be too soft, with a small firm pillow for the head. The patient should relax in his bed; his legs should be stretched. The patient should keep arms at the sides or folded across the chest. This position ensures relaxation of the abdominal muscles. Although patients commonly put their arms over their heads, this move should be discourages because it stretches and tightens the abdomi-

nal wall and makes palpation difficult. The ambient temperature should be comfortable for the patient, and the hands of the doctor should be warm and dry. Rubbing your hands together or running hot water over them may help to warm them. Before palpation, ask the patient to point to any areas of pain, and examine painful or tender areas last. Monitor your examination by watching the patient's face for signs of discomfort.

Surface, penetrative, and deep palpation are distinguished.

Superficial tentative oriental palpation of the abdomen

You should examine abdomen by the tips of the fingers, which should be slightly flexed, very tentative and careful to prevent muscular defense. Approach slowly and avoid quick, unexpected movements. Surface palpation conducts in topographic regions of the abdomen (Fig. 5.4).

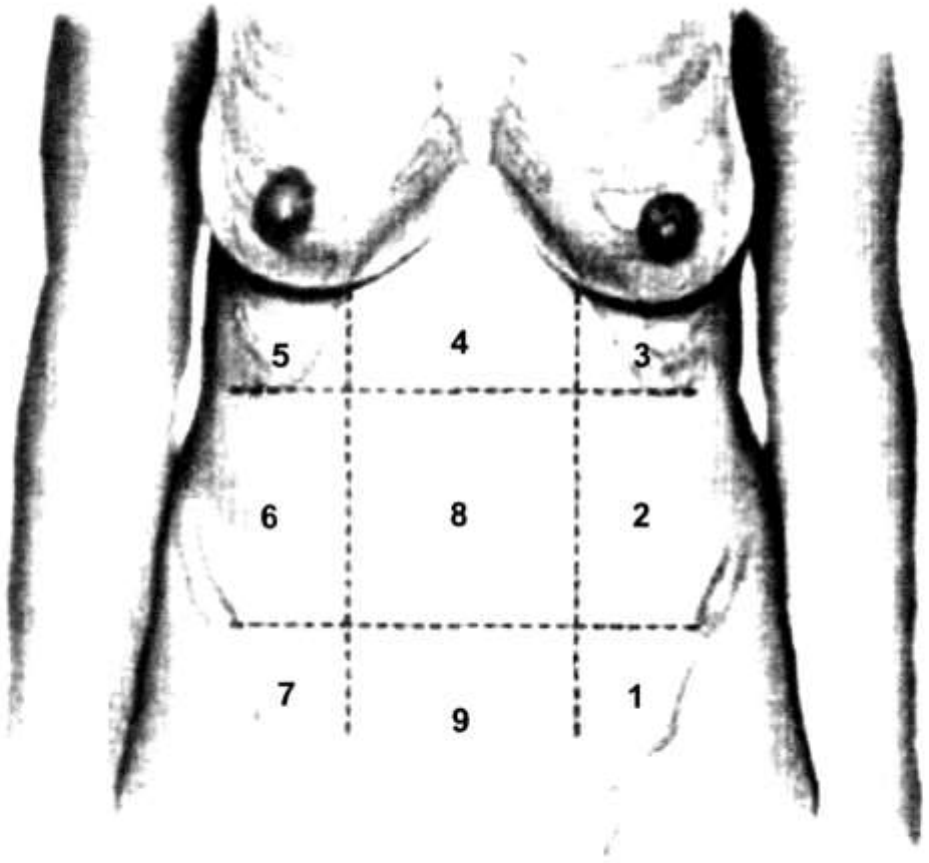


Fig. 5.4. Topographic regions of the abdomen.
1 – left inguinal, 2 – left iliac, 3 – left hypohondrium, 4 – epigastrium,
5 – right hypohondrium, 6 – right iliac, 7 – right inguinal,
8 – umbilical, 9 – suprapubical.

Keeping your hand and forearm on a horizontal plane, with fingers together and flat on the abdominal surface, palpate the abdomen with a light, gentle, dipping motion. When moving your hand from place to place, raise it just off the skin. Moving smoothly, feel in all quadrants (Fig. 5.5).

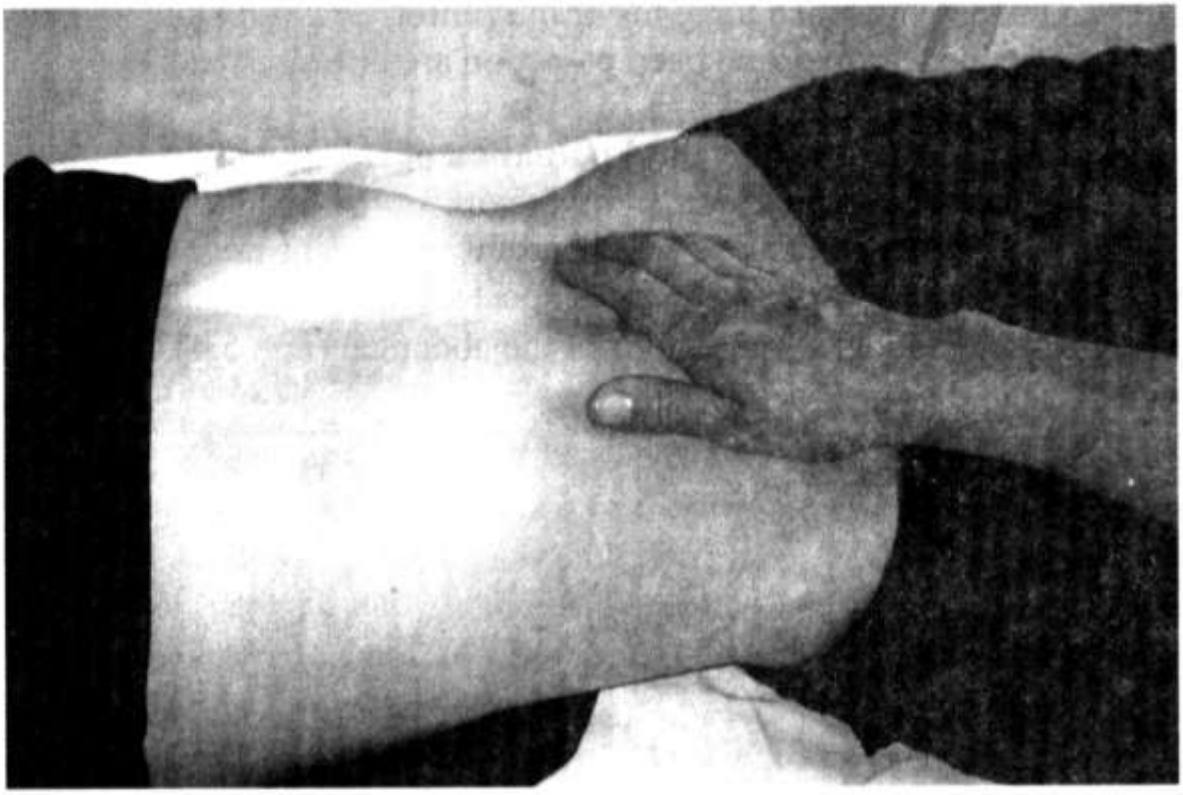


Fig. 5.5. Superficial palpation of the abdomen.

Superficial light palpation is helpful in identifying muscular resistance, abdominal tenderness, diastesis recti, and fluctuation symptom.

Identify increased resistance to your hand or any area of tenderness. If *resistance of the anterior abdominal wall* is present, try to distinguish voluntary guarding from involuntary muscular spasm. To do this try all relaxing method, feel relaxation of abdominal muBscles that normally accompanies exhalation, ask the patient to mouth-breath with jaw dropped open. Voluntary guarding usually decreases with these maneuvers. Involuntary rigidity (muscular spasm) typically persists despite these maneuvers. It indicates peritoneal inflammation.

If *abdominal tenderness* is present, you should indicate topographic region of the painful areas.

If *diastasis recti* is present you will feel separation of the two rectus abdominis muscles when you move your hand along midline.

Fluctuation symptom. While you tap one flank sharply with your fingertips, feel on the opposite flank for an impulse transmitted through the fluid. You can also ask the patient or an assistant to press the edges of both hands firmly down the midline of the abdomen. This pressure helps to stop the transmission of a wave through fat (Fig. 5.6). An easily palpable impulse suggests ascites.

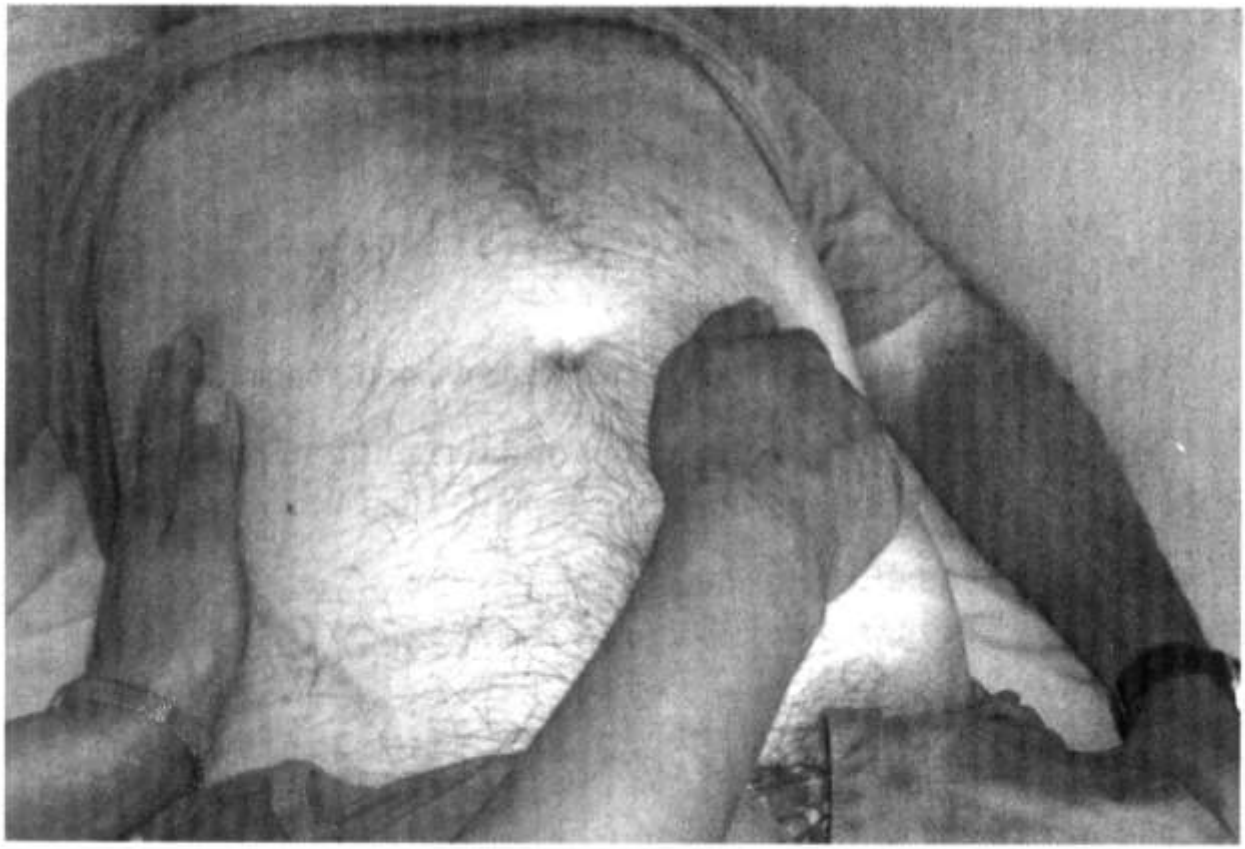


Fig. 5.6. Fluctuation symptom.

Norm. In superficial palpation the abdomen is soft, painless, diastasis recti is absent, fluctuation symptom is negative.

Penetrative palpation of the abdomen

Penetrative palpation includes revelation of tenderness points: gall bladder point (Ker point), appendix point (McBurne point), site of the duodenum bulb projection, and Shchetkin-Blumberg symptom.

Ker point – projection point of the gall bladder – places in a point of intersection of the right costal arch with the lateral edge of the right rectus abdominal muscle (Fig. 5.7).

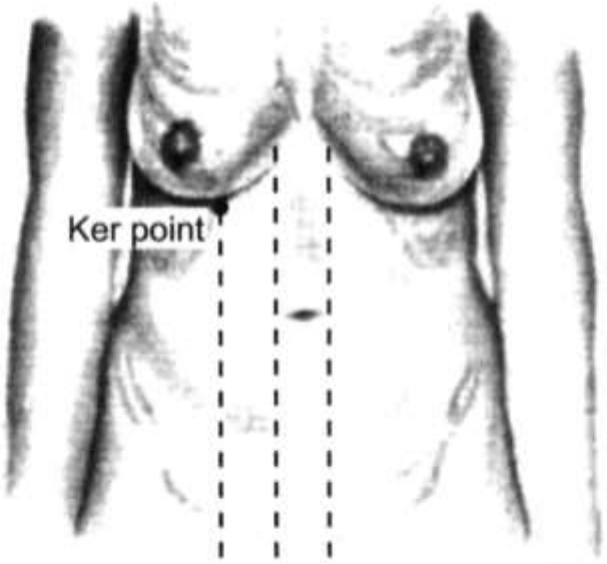


Fig. 5.7. Projection point of the gall bladder (Ker point).

McBurne point – projection point of the vermiform process – places at the border of median and outer third of the right umbilico-iliacae line (Fig. 5.8).

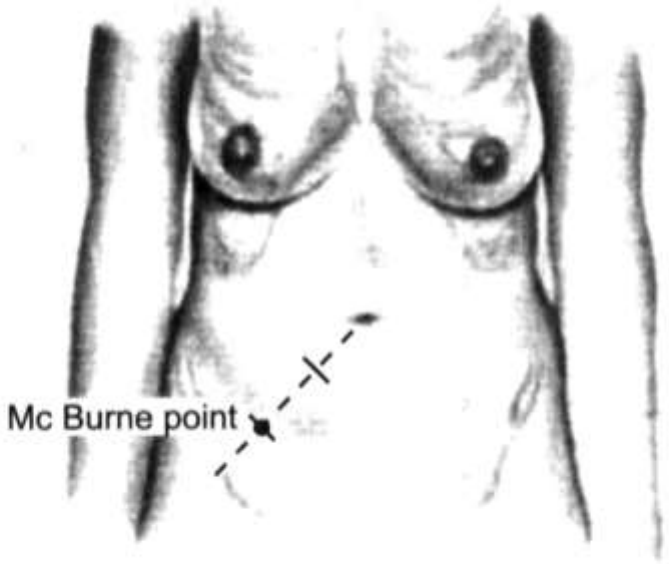


Fig. 5.8. Appendix projection point (Mc Burne point).

Duodenum bulb projection point places 2 cm from the umbilicus on the bisector of right angle formed by anterior median line and line drawing through the umbilicus (Fig. 5.9).

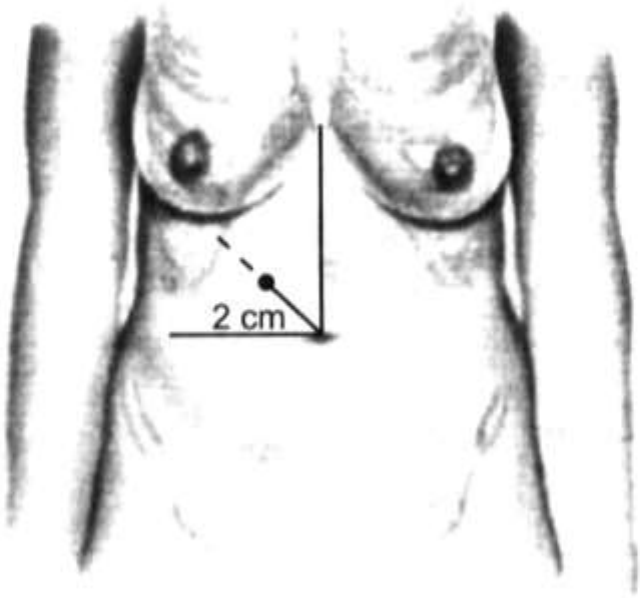


Fig. 5.9. Duodenum bulb projection point.

Shchetkin-Blumberg symptom – indicates irritation of the peritoneum. Local pressure causes strong pain, which becomes more severe when the pressure is released.

Abdominal pain and tenderness, especially when associated with muscular spasm, suggest inflammation of the parietal peritoneum. Localize it as accurately as possible. First, even before palpation, ask the patient to cough and determine where the cough produced pain. Thus guided, palpate gently with one finger to map the tender area. Pain produced by light percussion has similar localizing value. These gentle maneuvers may be all you need to establish an area of peritoneal inflammation. If not, look for rebound tenderness. Press your fingers in firmly and slowly, and them quickly withdraw them. Watch and listen to the patient for sign of pain. Ask the patient to compare which hurt more, the pressing or letting go, and to show you exactly where it hurt. Pain induced or increased by quick withdrawal constitutes rebound tenderness. It results from the rapid movement of inflamed peritoneum.

Norm. In penetrative palpation projection points of the gall bladder, duodenum bulb, and appendix are painless. Shchetkin-Blumberg symptom is negative.

Deep sliding systematic palpation of the abdomen (according to Obraztsov and Strazhesko)

Deep palpation can give you full information about the condition of the abdominal cavity and its organs, as well as their topography.

Technique. The following sequence is recommended:

1. The sigmoid
2. The caecum with the appendix
3. The ascending colon
4. The descending colon
5. The transverse colon
6. The stomach
7. The liver
8. The pancreas
9. The spleen

The palpation technique includes following steps:

- Proper position of your hands. Place the right hand flat on the anterior abdominal wall, perpendicular to the axis of the examined organ;
- Form a skin fold to facilitate further movements of the examined hand;
- Move your fingers gradually, on each expiration, into abdomen, when the abdominal wall is relaxed. Your hand thus reaches the posterior wall of the abdomen or the underlying organ;
- Last step: sliding movement of your fingertips in the direction perpendicular to the transverse axis of the examined organ.

In deep palpation you should assess location, diameter, density, the condition of surface (smooth, tubercular), tenderness, mobility, and presence or absence of rumbling sounds of examined organ.

The sigmoid. Place four fingers of the right hand together and slightly flexed perpendicularly to the axis of intestine, which runs obliquely in the left iliac region at the border of median and outer third of the linea imbilicoiliacae (Fig. 5.10).

As soon as posterior wall of the abdomen is reached, the fingers slide along intestine laterally and downward, and later the sigmoid slips from under the examining fingers. The sigmoid can be palpated in 90–95 % cases; normally over the length of 20–25 cm as a smooth firm cylinder, 2–3 cm in diameter, painless, easily displaces 3–5 cm to either side, doesn't rumble, with weak and rare peristalsis.

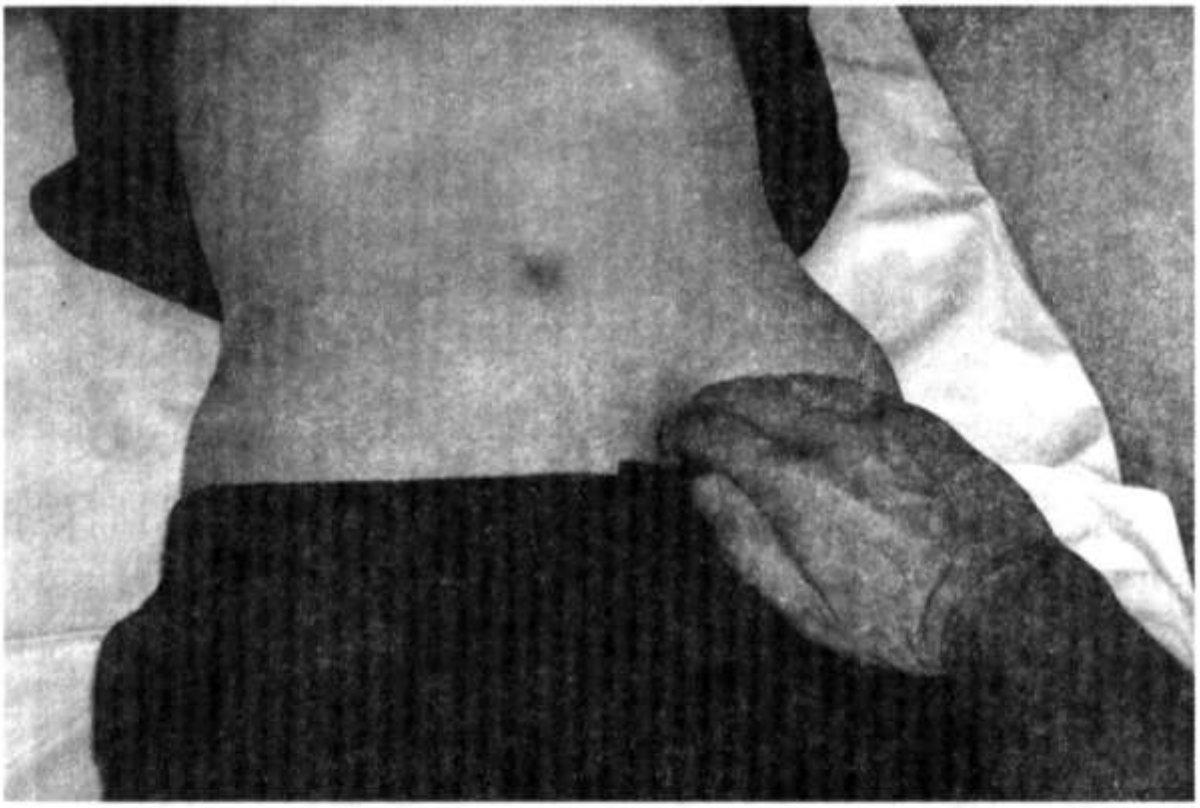


Fig. 5.10. Palpation of the sigmoid.

The caecum. Place your right hand along right umbilico-iliac line, the technique of palpation is the same (Fig. 5.11).

The caecum is situated at the border of the median and lateral third of the umbilico-iliac line (5 cm by the iliac spine). It can be palpated in 80–85 % of cases as a smooth, soft, elastic, slightly enwided downward cylinder, 3–4 cm in diameter, painless, moderate mobile (passive mobility to 2–3 cm); when pressed upon, it rumbles.

The vermiform process (appendix) can be found in 20–25% of cases above or below the terminal and of the caecum (ileum) in a form of thin,

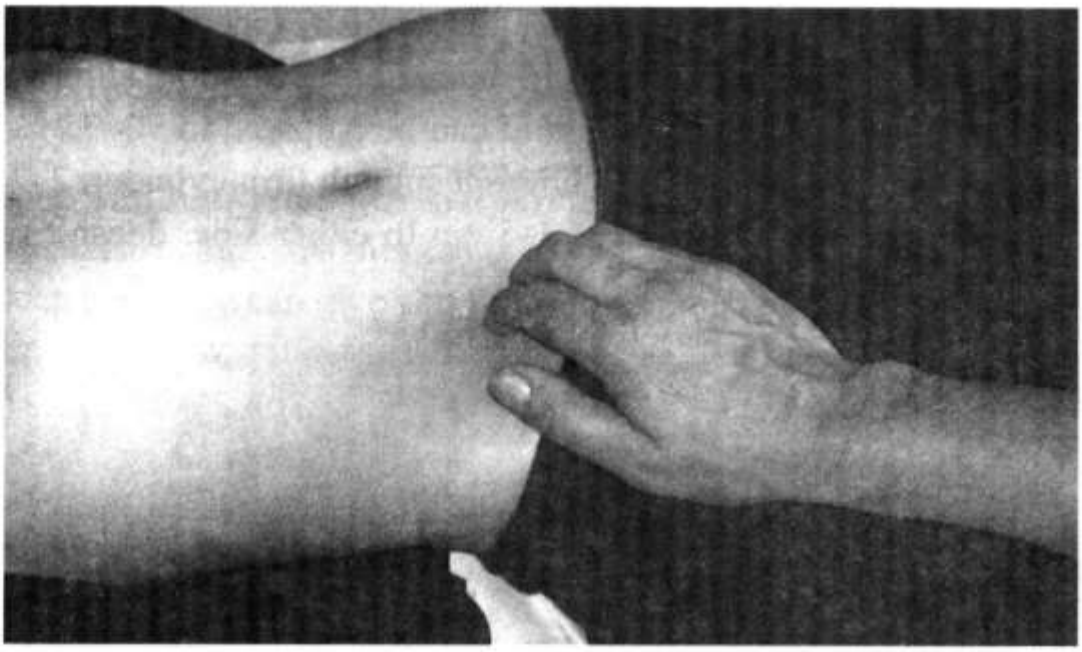


Fig. 5.11. Palpation of the caecum.

painless cylinder. During palpation appendix doesn't change consistency and doesn't rumble.

The ascending and descending colons are palpated by two hands. The left hand is placed under the right (Fig. 5.12) and then the left (Fig. 5.13)

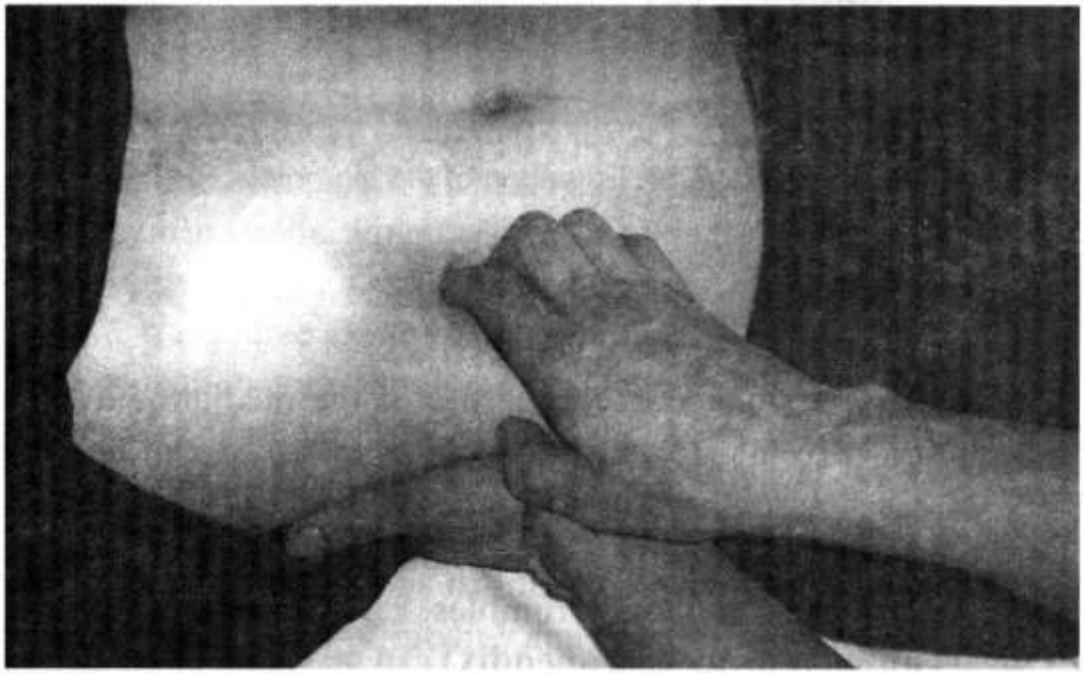


Fig. 5.12. Palpation of the ascending colon.

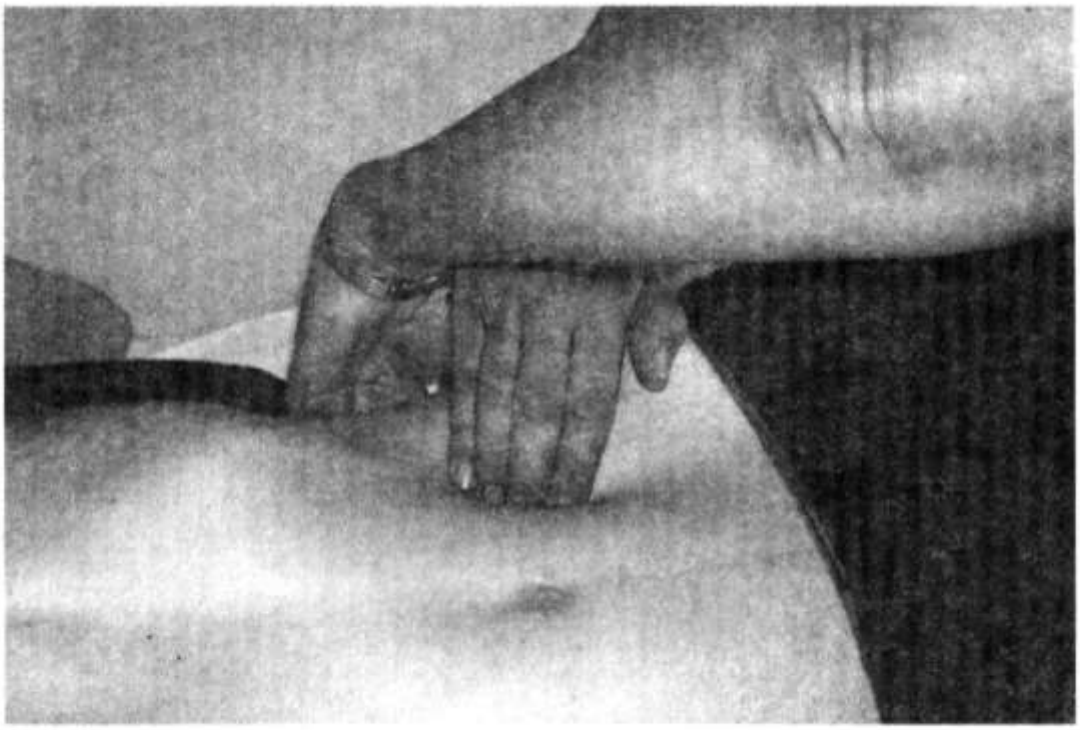


Fig. 5.13. Palpation of the descending colon.

lumbar region, while the finger of the right hand press on the anterior wall of the abdominal cavity until you feel your right and left hands meet.

The fingers then slide laterally, perpendicularly to the axis of the intestine. Ascending and descending colons are palpated, accordingly, in the right and in the left lateral flanks in a form of mobile, moderate dense, painless cylinder near 2 cm in diameter.

The transverse colon is palpated or by one hand or bimanual palpation is used (Fig. 5.14).

The right hand (or both hands) you should place on the sides of the linea alba and the skin more slightly upwards. When immerse your hands gradually during relaxation of the prelum out expiration until the posterior wall of the abdomen is felt. Once the posterior wall is reached, the examining hand should slide down to feel the intestine.

Normal transverse colon can be palpated in 60–70 % of cases, this is an arching cylinder, of moderate density, near 2.5 cm in diameter, painless, cagily movable up and down and silent.

The stomach. Pull up the skin on the abdomen and press carefully to penetrate the depth until the fingers reach the posterior wall, the stomach

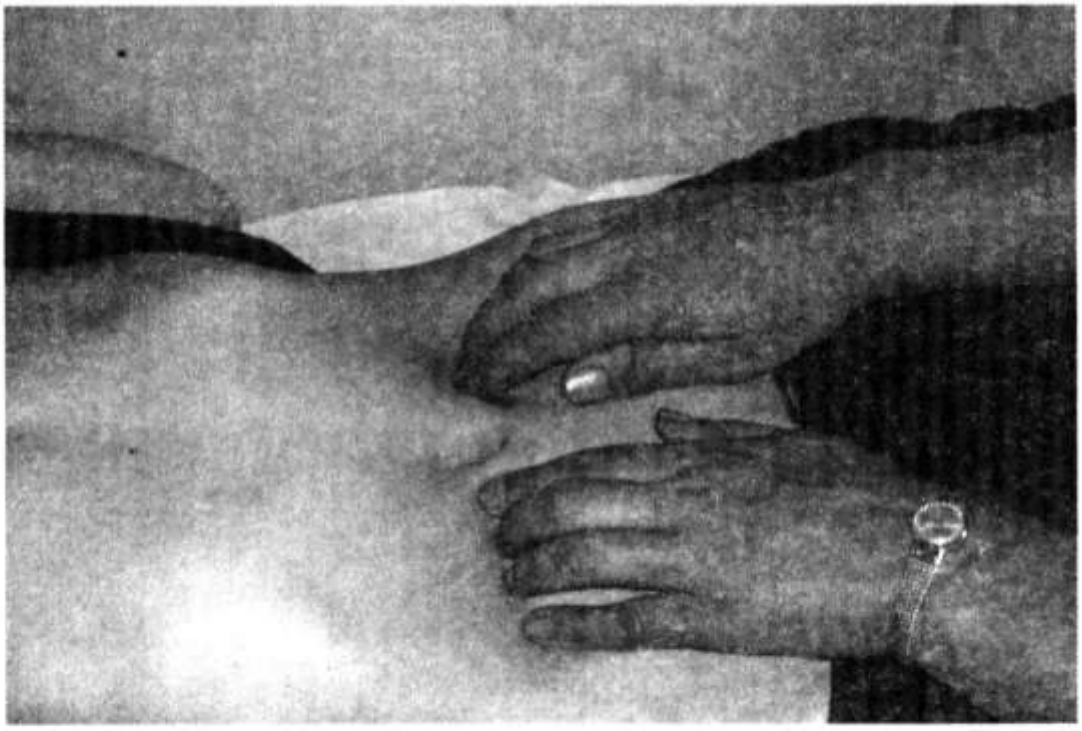


Fig. 5.14. Palpation of the transverse colon.

slips from under your fingers. The greater curvature can best of all examined by this method in 50–60 % of healthy subjects; the lesser curvature can be palpated in gastroptosis. The greater curvature is found to either side of the median line 3–4 cm above the umbilicus in male, and 1–2 cm in female in a form of smooth, soft, not mobile, and painless ridge.

The liver. Because most of the liver is sheltered by the rib cage, assessing it size by palpation is impossible. Therefore, its percussion must always accompany palpation of the liver.

Percussion of the liver has following potential uses: determination of the liver borders, assessment of its size and configuration. Percussion of the liver according M. G. Kurlov is widely used. In order to determine the size of absolute liver dullness you should identify five points by light percussion (Fig. 5.15).

Firstly, identify the upper border of absolute liver dullness in the right midclavicular line. Percuss from the 1st interspace downward toward liver dullness (1st point).

Next, starting at a umbilicus level in the right medclavicular line percuss upward toward the liver (2nd point).

Measure in centimeters the distance between these two points, normally – 9–11 cm.

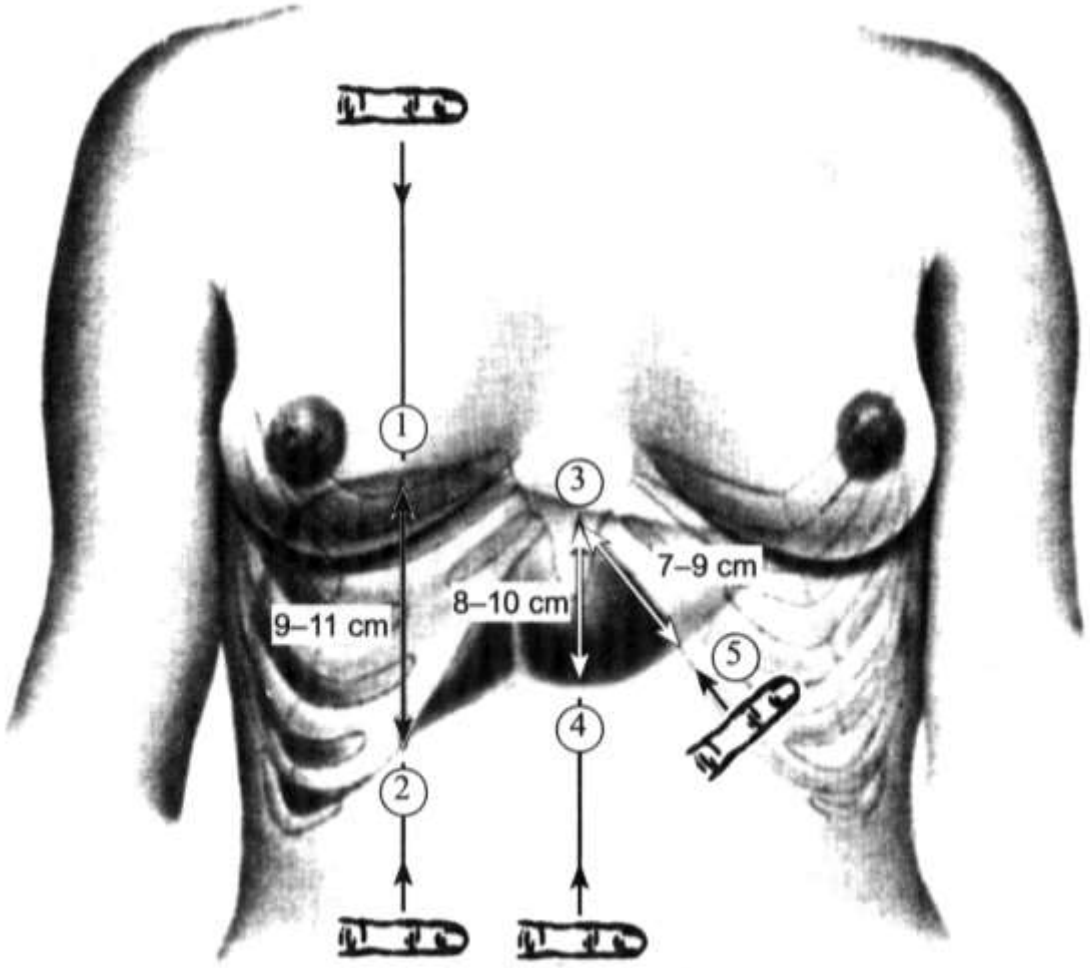


Fig. 5.15. Percussion of the liver according M. G. Kurlov.

In order to identify 3rd point, drop perpendicular from the 1st point to anterior median line (at the base of xiphoid process).

Next, identify the lower border of absolute liver dullness in anterior median line. Percuss from the umbilicus level upward toward the liver (4th point).

Measure the distance between 3rd and 4th points, normally – 8–10 cm.

Place pleximeter-finger perpendicularly to the edge of the left costal arch at the level of the 8–9th ribs and percuss toward to the 3rd point to the liver dullness.

Measure the distance between 5th and 3rd points, normally – 7–9 cm.

Diagnostic significance of the liver borders changes

The liver borders can vary depending on hepatic and extrahepatic changes.

Hepatic causes

The span of liver dullness is increased (that is upward displacement of the upper liver border and downward displacement of the lower liver border) when the liver is enlarged in considerable tumor, liver abscess, echinoccocosis.

Downward displacement of the lower liver border is caused by hepatitis, congestive liver in right ventricular failure, and acute liver failure.

Upward displacement of the lower liver border observes in atrophic stage of the liver cirrhosis.

Displacement of only upper liver border is usually caused by extrahepatic changes.

Extrahepatic causes

Upward displacement of the upper liver border can be due to consolidated right lung in pneumonia, tumor or right pleural effusion. High diaphragm level in subdiaphragmal abscess, meteorism, ascitis, pregnancy, pneumoperitoneum can also cause displacement of upper liver border upward.

Downward displacement of the upper liver border observes in low diaphragm level, pulmonary emphysema, right-sided pheumothorax, and enteroptosis.

Upward displacement of the lower liver border can be the result of decreased liver size or high diaphragm level.

Downward displacement of the lower liver border observes in low diaphragm level (asthenic constitution) or in chronic obstructive lung diseases. Liver size, however, remains normal in such cases.

Palpation of the liver. The liver lower edge, surface, consistency, and tenderness can be estimated by palpation.

You should remember that respiratory mobility of the liver is highest compared with other abdominal organs because the liver is closest to the diaphragm. Therefore, during palpation the active role belong to the liver respiratory mobility rather than to your palpating fingers.

Sit by the right side, facing the patient (Fig. 5.16). Place four fingers of your left hand behind patient, parallel to and supporting the right 11^{th} and 12^{th} ribs, use left thumb to press on the costal arch to move the liver closer to the palpating fingers, and to prevent expansion of the chest during inspiration. This stimulates greater excursions of the right cupola of the diaphragm. Place your right hand flat on the patient's right abdomen lateral to

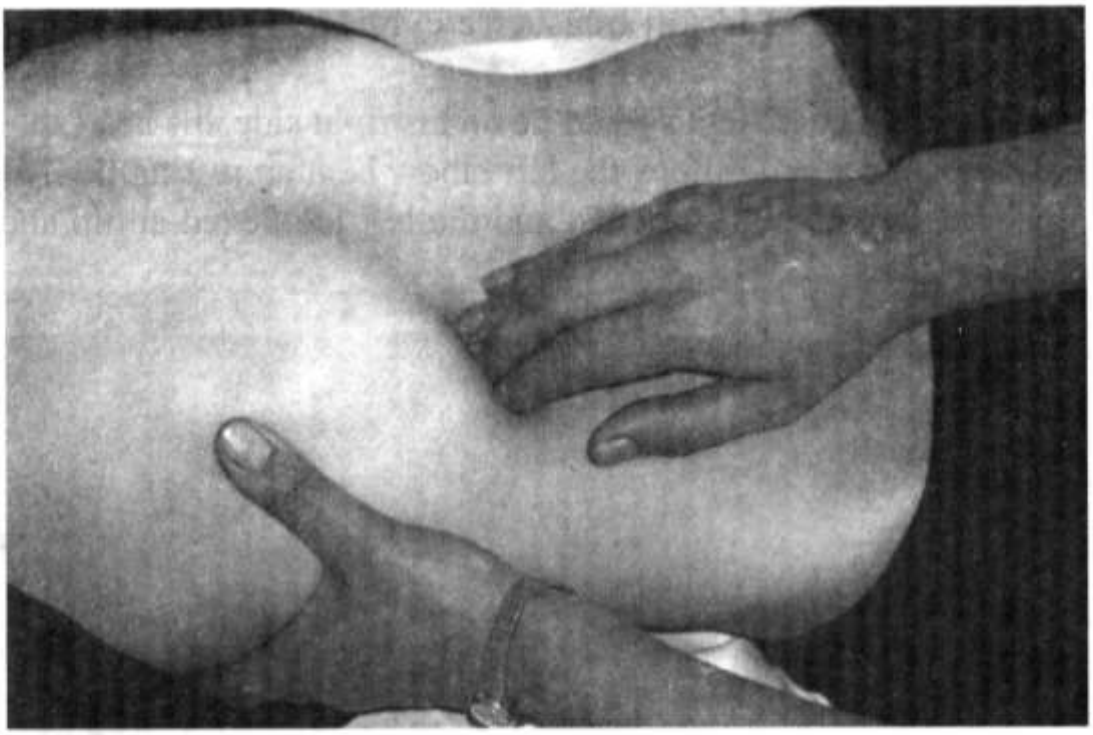


Fig. 5.16. Palpation of the liver.

the rectus muscle (in midclavicular line), with your fingertips well below the costal arch. Press gently in and up. Then ask the patient to take deep breath; the liver descends to touch the palpating fingers and then slides to bypass them. Your right hand remains motionless. The procedure is repeated several times. Try to feel the liver edge as it comes to meet your fingers. If you feel it, note surface, consistency, and any tenderness of the liver lower edge.

The normal liver can be palpated in 88 % of cases. On inspiration, the liver is palpable about 1–2 cm below the right costal margin in the midclavicular line. The edge of the normal liver is soft, sharp, tenderness; its surface is smooth.

The pancreas. Because of the deep location and soft consistency of the gland, palpation of the pancreas is very difficult. The normal pancreas can only be palpated in 4–5 % of female and 1–2 % of male with cachexia, relaxed prelum and ptosis of internal organs.

The pancreas should be palpated in the morning, with empty stomach. Place your right hand horizontally, 2–3 cm above the preliminary found lower edge of the stomach. Pull the skin upward and then press gradually right hand into abdominal cavity with each expiration. When you reach posterior wall, slide your hand downward.

A normal pancreas is soft transverse cylinder, 1.5–3 cm in diameter, immobile, and tenderness.

The spleen. The patient should lie on his right side, his head should be slightly down on a small pillow, the left elbow bent an resting freely on the chest, the right leg should stretched and the left leg flexed at hip and knee (Fig. 5.17).

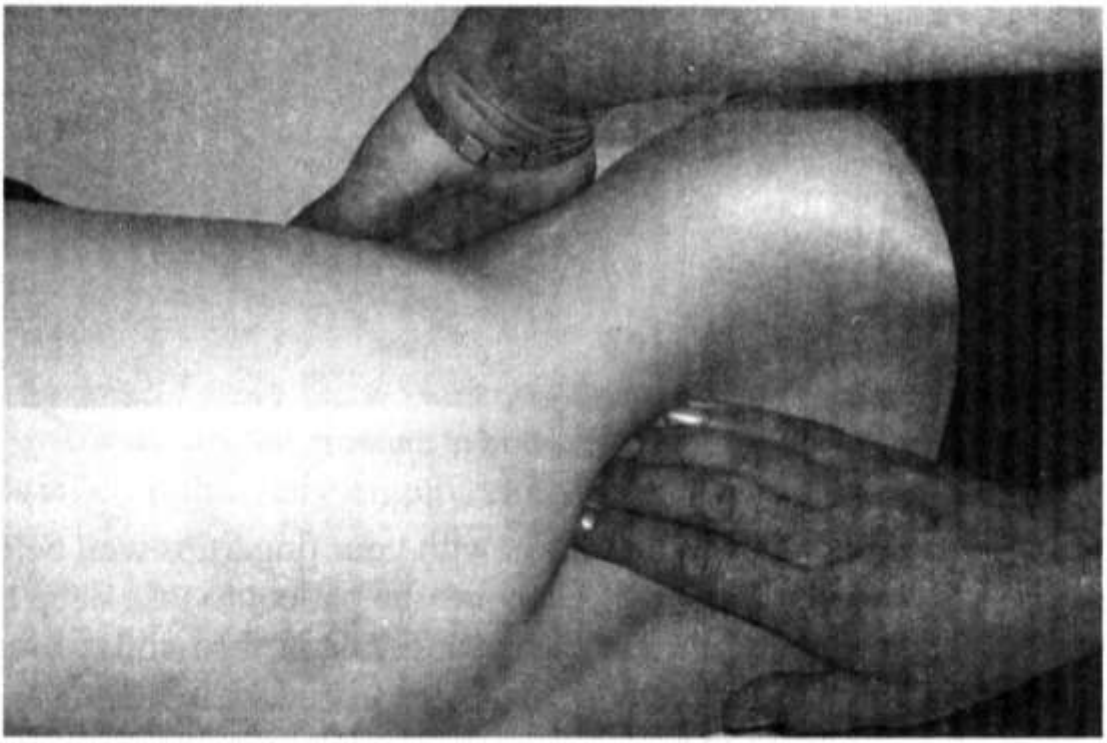


Fig. 5.17. Palpation of the spleen.

In this position the prelum is relaxed to a maximum, and gravity may bring the spleen forward and to the right into a palpable location. Sit on the right side, facing the patient. Place your left hand on the left part of the chest between 7th and 10th ribs in the axillary lines and slightly press to limit respiratory movements. With your right hand below the left costal margin, at the point of junction of the costal arch and the 10th rib, press gradually during expiration in toward the spleen. Ask the patient to take deep breath (Fig. 5.18). If the spleen is palpable, it is displaced during inspiration by the descending diaphragm to meet your fingers and to slip over them.

Try to feel the tip or edge of the spleen, note location, consistency, any tenderness, and surface.

A normal spleen is impalpable. In a small per centage of normal adults, the tip of the spleen is palpable. Causes include a low, flat diaphragm, as in

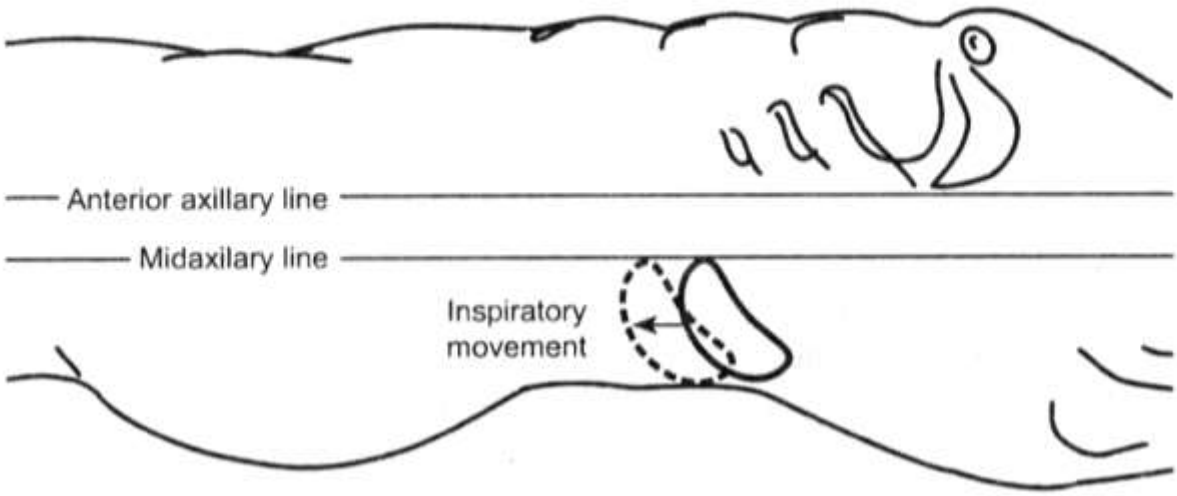


Fig. 5.18. Inspiratory movement of the spleen.

chronic obstructive pulmonary disease, and a deep inspiratory descent of the diaphragm.

Instrumental and Laboratory Methods

Imagine Studies

Radiological and imaging techniques play an important role in the diagnosis of disorders of the digestive system. These diagnostic procedures for assessing the patients with suspected or known digestive disease include:

- Roentgenography (radiography, x-rays);
- Angiography;
- Radionuclide studies;
- Computed tomography;
- Magnetic resonance imaging;
- Ultrasound examination (ultrasonography);
- Cholecyctography;

Roentgenography. Plain abdominal radiographs may provide help to obtain the required information in the majority of cases. Barium sulphate suspension continues to be widely used for routine examination of the gastrointestinal tract.

The technique allows the esophagus entire length to be visualized. The separate moments of the esophagus filling and condition of the esophageal

mucosa can be assessed. Special barium studies are sometimes indicated, such as cineradiography or videoradiography, which demonstrate peristaltic, contraction ability, and functional abnormalities of the esophagus.

The shape, size, position and mobility of the stomach; carcinomas, ulcers and ulcer scars that have a converging fold pattern are easily detected. Small lesions and slight irregularity of the mucosa can be identified.

Barium-meal examination of the small intestine is normally made following examination of esophagus, stomach and duodenum. It is indicated when disorders causing morphological changes in the small intestine, such as Crohn's disease, tuberculosis, neoplasms, radiation damage, ischemia or diverticulum, are suspected. The functional properties can be also assessed by x-ray study. Cancers of the small intestine have no specific radiological patterns.

The barium examination of the large intestine is important diagnostic procedure that gives information about its motor function, length, position, shape, and tone.

Enlargement of the liver may be identified on plain radiographs, but adds little to clinical examination. Calcification is occasionally seen in the liver and the most common causes are old granulomatous disease and hydatid cyst.

Plain radiographs are normally the initial diagnostic procedure in patients with acute symptoms of disease of the biliary tract. The plain film may show pathological calcification of the gallbladder, opaque calculi, gas in the biliary tree, or radio-opaque bile in the gallbladder.

The plain radiograph of the upper abdomen is an important investigation in patients presenting with symptoms of acute or chronic disease of the pancreas. In acute pancreatitis it may show the colon 'cut-off' sign, a sentinel loop, evidence of displacement of the stomach, or of a pancreatic abscess. Pancreatic calcification may be seen in patients with chronic pancreatitis.

The plain radiograph will usually show an enlarged spleen as is found in blood disorders of the reticuloendothelial system, infection, hepatic cirrhosis, and trauma. A single area of calcification may be seen in atherosclerosis or aneurysm of the splenic artery. Multiple calcifications occur in healed tuberculosis, phleboliths, hemangiomas, and histoplasmosis.

Angiography continues to have a role in the investigation of gastrointestinal bleeding and in the preoperative evaluation of the liver and pancreas.

Gastrointestinal angiography. Selective visceral angiography is indicated in certain patients who present with bleeding from the gastrointestinal tract. Angiography is done for two reasons in patients with acute bleeding – to locate the source of bleeding when it is unknown, and to stop the bleeding by selective infusion of drugs or embolic material into the bleeding territory.

Coeliac-axis angiography is useful in identifying hemangiomas, as they have feeding vessels of normal size but with a slow flow of contrast through the lesion. Angiography will help to differentiate hepatic tumors and may be done during computed tomography to maximize the detection of metastases, but is more commonly used to identify the exact site and blood supply of neoplasms before partial hepatectomy.

Percutaneous transhepatic cholangiography is widely used for demonstrating the bile ducts in obstructive jaundice.

Endoscopic retrograde cholangiopancreatography is frequently used to demonstrate the ducts in patients with obstructive jaundice. Endoscopic sphincterotomy can be done during the procedure, thus allowing bile-duct calculi to pass freely into the duodenum, and frequently relieving bile-duct obstruction due to calculi.

Coeliac angiography will usually show insulinomas of the pancreas, but transhepatic portal catheterization will allow samples of blood to be taken from the splenic vein to localize insulinomas if computed tomography or angiography are unsuccessful.

Radionuclide studies. In children, Meckel's diverticulum can be detected and this should be the initial radiological procedure if this condition is suspected in neonates and children. Radionuclide studies can also be used to locate the site of obscured bleeding from the gastrointestinal tract. The general anatomical location of bleeding can be identified in many patients and further investigations such as angiography or barium studies can then be made to define the site and cause of bleeding more precisely.

Ultrasonography and computerized tomography have largely replaced radionuclide studies of the liver.

The spleen can be examined by radioisotopes and this may be particularly useful in trauma or where a small splenunculus is suspected.

Computed tomography is of use in demonstrating metastatic disease and primary neoplasia of the liver. Cysts and abscesses also show well on

computed tomography, but cirrhosis may be difficult to identify with certainty. The ability of high-resolution computed tomography to detect small intrapancreatic pseudocysts, pseudocysts containing gas or solid contents, pancreatic calcification, and peripancreatic fascial thickening make it the most accurate method for evaluating pancreatitis. Accurate assessment of carcinoma of the pancreas is also possible with computerized tomography.

Magnetic resonance imaging. This technique is excellent for differentiating malignant neoplasms from benign cysts, and for providing useful information in patients with cirrhosis and with metastatic deposits. The improved sensitivity produced by the use of contrast agents, the elimination of artefacts, and the characterization of tissue both by magnetic resonance imaging and magnetic resonance spectroscopy provide a useful, radiation-free adjunct to ultrasonography and computerized tomography.

Ultrasound examination (ultrasonography). Ultrasound examination of the liver is safe, cheap, and accurate in experienced hands. Abscesses appear as black transonic areas surrounded by high-intensity echoes, whilst cysts have black transonic areas surrounded by a thin echogenic rim. Neoplasia produces areas of discontinuity in the homogeneous pattern of the liver. Most commonly, the echo amplitude is less than that of the surrounding liver, but some metastases, particularly from the colon, produce high-intensity echoes. Direct ultrasonography of the liver exposed at surgery may show lesions not visible by the normal transcutaneous technique. Cirrhosis produces a higher amplitude of echoes than does the normal liver and a large portal vein may be demonstrated. Colour-flow Doppler can help to differentiate hemangiomas from other neoplasms and is invaluable in the assessment of the portal and hepatic veins. Diagnostic biopsy of liver tumors is greatly facilitated by ultrasound control.

High-definition sector scanners provide an excellent real-time image of the gallbladder, and the intrahepatic and extrahepatic bile ducts. The small probe can be used between the ribs and allows scanning with the patient erect. The accuracy in detecting gallstones is similar to that of oral cholecystography with the added advantage that the bile ducts may be examined at the same time. Ultrasonography has replaced oral cholecystography as the method of choice for detecting biliary-tract calculi and as the initial investigation for suspected gallbladder disease. A thickened gallbladder wall is sometimes seen in acute cholecystitis. Dilated hepatic and common bile

ducts may be identified and, if the bowel is relatively free from gas, intraductal calculi or an enlarged head of pancreas may be identified.

Ultrasonography allows the measurement of size and the visualization of parenchyma of the pancreas. Acute pancreatitis, neoplasms, and pseudocysts may be identified and, if a neoplasm is diagnosed, it may be biopsied using a Chiba needle guided by ultrasound. Ultrasound is often used to investigate epigastric masses with the advantage that other organs in the region of the pancreas including the aorta, para-aortic lymph nodes, and adrenal glands may be seen. Peroperative ultrasonography may identify insulinomas not identified by other techniques. Ultrasound may be used to measure splenic size and in identifying splenic cysts.

Cholecyctography. About 80 to 85 per cent of gallstones are not radio-opaque and oral cholecystography remains the method of choice for examining the gallbladder with contrast medium to detect calculi when ultrasound is not available or is inconclusive. Abnormalities causing a change in the outline of the gallbladder such as adenomyomatosis are well demonstrated at oral cholecystography.

Endoscopy

Endoscopy not only provides the ability to visualize the esophagus, stomach, duodenum, and colon directly but also allows biopsy specimens and cytological samples to be taken.

Endoscopy is the investigation of choice for gastroduodenal disease. Gastritis (acute, chronic, superficial, atrophic), the presence of erosions is readily recognized and biopsy specimens can be obtained for histological diagnosis. This has become particularly important since the association between Helicobacter pylori and gastritis and ulcer disease has been recognized. All gastric ulcers, even if they appear benign, require biopsy and cytological brushing to exclude malignancy. Endoscopy is also essential for symptomatic patients who have had previous gastric surgery, as the postoperative stomach is notoriously difficult to examine radiologically.

Duodenal disease is always better assessed by endoscopy rather than by radiography, although the endoscopist can miss duodenal ulcers if they are just behind the rim of the pylorus. Distal duodenal biopsy specimens are being increasingly used for the diagnosis of coeliac disease.

Colonoscopy is being increasingly used for the diagnosis of colonic disease in preference to a double-contrast barium enema.

Visualization of the pancreatic duct by direct endoscopic cannulation should be done when pancreatic tumors or chronic pancreatitis are suspected. This procedure, together with ultrasound or a computerized tomographic scan and possibly a pancreatic function test, will give maximal diagnostic information.

Endoscopic visualization of the biliary tree is now the best diagnostic procedure for stones, tumors, and strictures of the bile duct and is the only reliable means of diagnosing primary sclerosing cholangitis. Furthermore, it offers the therapeutic procedures of sphincterotomy, stone withdrawal, and the insertion of stents across strictures.

Study of the gastric secretion

Study of the gastric secretion allows assessing fasting stomach function, secretory, partially evacuatory, and also acid-secretory function of the stomach.

Fractional method

Study is done in two periods: examination of nonstimulated secretion (basal secretion), and examination of stimulated secretion (after stimulants are given).

The amount of pure gastric juice aspirated during 1h is called "hour strain" of secretion (Tab. 5.9).

Tab. 5.9. ***Diagram of examination of the gastric juice by fractional method.***

Probe introducing		
Fasting sample collecting *(Normally 5–40 ml)*		
Basal secretion *(Normally 50–100 ml)*	15 min 30 min 45 min 60 min	1 hour
Histamin or pentagastrin administration		
Stimulated secretion *(Normally 100–150 ml)*	15 min 30 min 45 min 60 min	1 hour

Study of the gastric content includes assessing of its physical, chemical properties, and microscopic studies.

Physical study

The following physical properties are determined: amount, color, consistency, and admixtures.

Amount. Normally, after night fast the stomach contains from 5 to 40 ml, basal secretion is from 50 to 100 ml, and stimulated secretion is from 100 to 150 ml.

Changes of the gastric contents amount are of great diagnostic significance, and allow assess secretory and partially evacuatory function of the stomach.

Increased fasting gastric juice amount suggests constant gastric secretion (gastrochronorrhea) in high activity of parasympathetic system, in long-standing smoking abuse, ulcer disease, meal delay in the stomach, etc. Elevated "hour strain" of secretion usually suggests hypersecretion or delayed evacuation from the stomach; decreased "hour strain" – hyposecretion or accelarate evacuation.

Color. Normally, the gastric juice is colorless. In the presence of bile that may be belched into the stomach from the duodenum the juice is yellow or green. In the presence of blood, the gastric juice is red or more frequently brownish-black. In the presence of large amount of unaltered scarlet blood the procedure should be finished immediately.

Consistency. Normally, gastric juice is liquid; in the presence of mucus – tenacious and it is sometimes difficult to separate a portion from the whole mass of the juice taken.

Admixtures of bile, blood, and mucus can be present in the gastric juice. Ample mucus may suggest gastritis; supernatant mucus originates from the airways. Presence of the residues of food taken before indicates motor dysfunction of the stomach.

Chemical study

Study of the chemical properties allows assess acid-secreting function and include measurement of acidity, unbound hydrochloric acid, bound hydrochloric and lactic acid.

Total acidity is summary acidity of all acidic factors of gastric contents in physiological and pathological conditions (unbound, bound hydrochloric

acid, organic acids and acid phosphates). Total acidity level depends mainly on unbound hydrochloric acid level.

Unbound (free) hydrochloric acid is that part of acid, which are contained in the stomach in a form of dissociated hydrogen and chlorine ions.

Bound hydrochloric acid is that part of acid, which is contained in the stomach in a form of non-dissociated molecules, chemically bound with proteins.

Measurement of basal and stimulated gastric secretion is used in the clinical assessment of patients with some gastrointestinal diseases. The range of values for normal subjects is extremely broad (Tab. 5.10).

Tab. 5.10. ***Study of the gastric secretion.***

Fasting stomach content	
Amount	5–40 ml
Total acidity	Not more than 20–30 mmol/l
Unbound hydrochloric acid	To 15 mmol/l
Basal secretion	
Total amount collected by four portion in 1 hour after aspiration of fasting portion	50–100 ml
Total acidity	40–60 mmol/l
Unbound hydrochloric acid	20–40 mmol/l
Bound hydrochloric acid	10–15 mmol/l
1-h total hydrochloric acid output	1.5–5.5 mmol/h
1-h unbound hydrochloric acid output	1.0–4.0 mmol/h
Histamine stimulated secretion	
Total amount	100–150 ml
Total acidity	80–100 mmol/l
Unbound hydrochloric acid	65–85 mmol/l
Bound hydrochloric acid	10–15 mmol/l
1-h total hydrochloric acid output	8–14 mmol/h
1-h unbound hydrochloric acid output	6.5–12 mmol/h

Normally, fasting stomach contains usually no free hydrochloric acid. In can be present, but its contents should not be exceed 15 mmol/l. Presence of unbound hydrochloric acid in the fasting stomach, especially in high concentration (50–80 mmol/l) indicates duodenum ulcer or "irritated" stomach.

Basal unbound hydrochloric acid secretion is 20–40 mmol/l, total acidity is 40–60 mmol/l normally.

Stimulated secretion of unbound hydrochloric acid is 65–85 mmol/l, total acidity is 80–100 mmol/l in normal subjects.

In pathology can be observed *hyperacidity* (in the patients with peptic ulcer, chronic gastritis with acid hypersecretion), *hypoacidity* (in the patients with chronic gastritis with hyposecretion), and *unacidity* or *achlorhydria* – complete absence of hydrochloric acid after maximum dose of stimulator (atrophic gastritis associated with pernicious anemia, tumor).

To obtain more detailed information about gastric secretion function 1-h gastric acid output is measured.

Gastric output is absolute amount of hydrochloric acid secreted in certain period, and is calculated by formula or by nomogram in laboratory. Such calculations are done for each portion of the gastric juice. Then 1-h acid output is measured.

1-h acid output is absolute amount of unbound hydrochloric acid secreted in one hour, and is calculated by formula. 1-h basal acid output (BAO), stimulate submaximal acid output (SAO) or stimulate maximal acid output (MAO) are measured. 1-h acid output allows determining either HCl concentration, and amount of secreted gastric juice; and reflects true level of unbound HCl secretion. 1-h HCl output more authentically than concentration reflects condition and dynamic of acid secretion function of the stomach, especially in pathology.

Lactic acid. Gastric juice in physiological conditions contains insignificant amount of lactic acid that practically is undeterminable. In pathology it is produced as a result of vital activity of the lactobacillus in congested gastric contents in the absence of hydrochloric acid. In malignant tumor lactic acid can be produced as a product of tumor cells metabolism. Uffelmann reaction is used for qualitative determination of lactid acid.

Endogastric pH-metry.

This method has advantages over routine fractional study of the gastric contents. Acidity measurement directly in the stomach is more physiologi-

cal, as aspiration itself altered normal acid secretion. Furthermore, endogastric pH-metry allows studying acidity in the separate regions of the stomach: antrum, body, and cardia. Measurement of pH on separate levels has great diagnostic significance, especially in chronic gastritis. Registration is done in each 15 min before and after stimulation (Tab. 5.11).

Tab. 5.11. ***Assessing of the pH-metry results (according to U. Linar).***

Secretion	pH
Fasting	
Greatly acid	0.9–1.9
Medium acid	2.0–2.9
Moderate acid	3.0–4.9
Poor acid	5.0–6.9
Alkaline	7.0–8.9
Basal	
Hyperacidity	Less than 1.5
Normal acidity	1.6–2.0
Hypoacidity	2.1–5.9
Unacidity	More than 6.0
Stimulated	
Hyperacidic reaction	Less than 1.2
Normoacidic reaction	1.2–2.0
Hypoacidic reaction	2.1–3.0
Decreased reaction	3.1–5.0
Unacidic reaction	5.0 and more

Endogastric pH-metry allows obtaining more precise data concerning dynamic of gastric secretion in basal conditions and after stimulation. It is possible to assess duration and ending of secretion in response to stimulation.

Microscopic study

Microscopic studies have no particular significance, because cellular contents change greatly under influence of gastric juice acidity.

Mucus is observed in gastric juice samples of healthy subjects. Increased amount of mucus indicates inflammatory processes in mucus membrane of the stomach.

Leucocytes are altered (in a form of neutrophils nuclei) in the presence of unbound hydrochloric acid, and unaltered cells are observed in unacidic conditions. Leucocytes are placed commonly in mucus.

Squamous epithelium observes in the mucus in decreased amount or complete absence of free hydrochloric acid in the gastric contents.

Erythrocytes. Unaltered erythrocytes uncommon observe in gastric contents, as they are quickly destroyed. Small amount of erythrocytes can be caused by injury in probing or strain during vomiting, and have no diagnostic significance. Diagnostically important is ample erythrocytes that indicates the presence of peptic ulcer, tumor or erosive gastritis.

Furthermore, the food remains (muscles fibers, fat, fatty acids, cellulose) can be found that suggests evacuation dysfunction of the stomach. If congested gastric content is acid, it contains sarcinae, if acidity is absent – elastic fibers are detected

Coprological study

Faeces (stool, copros) are content of the large intestine that excreted in defecation.

Faeces of a healthy person contain 75–80 % water and 20–25 % solid residue. Solid part consists of on 1/3 of undigested food remains, on 1/3 – of remains of gastrointestinal tract secretions, and on 1/3 – of microbes, approximately 90 % of which are dead.

Stools are collected in a clean dry, better glass container. Morning faeces are usually studied as soon as possible. Especially important to examine fresh stools for the presence of protozoa and helmints. Faeces are commonly studied without of special preparation of the patient, but 2–3 days before study is recommended to avoid drugs that change character of the stool and caused functional disturbances of the gastrointestinal tract.

Coprological analysis includes macroscopic, chemical, microscopic and bacteriological studies.

Macroscopic study

This examination consists of determination of amount, consistency, shape, color, odor, presence of visible undigested food remains, pathological admixtures, and parasites in faeces.

Daily faeces amount

The amount of faeces depends on the rate of defecation, quantity and character of food, quality of the food digestion in the gastrointestinal tract, presence of pathological components (mucus, blood, pus), and water content in the stools.

The normal daily excretion is 120–250 g 1–2 times per day.

The amount of faeces and rate of defecation (1 time in 3–4 days) *decreases* in starvation, vomiting, and constipation. Stools are also meager in protein diet (meat, eggs). In dysentery as a result of increased sensitivity of rectum mucus membrane, frequent urge to defecation arises (to 20 per day), but each time small amount of stools are excreted.

The amount of faeces *increases* in ample vegetable diet, poor assimilation of food in chronic pancreatitis, amyloidosis of the intestine, chronic enteritis (to 1.5–2 kg). The amount of stools vary greatly depend on water content. In pancreatitis, enteritis, enterocolitis as a result of intensified peristalsis water not has time to absorbed in intestine to increase the amount of faeces.

Shape and consistency of faeces

The consistency and consequently the shape depend mainly on water content. Normal faeces have a cylinder shape, resembling sausage 2–4 cm in diameter, and are usually soft. It contains 70–75 % water.

In constant constipation due to excessive absorption of water the faeces are dense, or even *solid*. In spastic colitis sheep dung stools can be observed. Such solid stools contain 60 % water.

In intensified peristalsis due to insufficient absorption of the water, the faeces have no definite shape, are *gruel-like* or *liquid* in the patients with chronic colitis, chronic pancreatitis or in ample vegetable diet. More liquid consistency of the faeces is caused by ample excretion of inflammatory exudates and mucus by intestinal wall in the patients with enteritis and enterocolitis. Liquid stools contain 90–92 % water.

Sometimes the stools are of *pasty* consistency due to the presence of large quantity of fat.

In some diseases, which are accompanied by stenosis of lower part of sigmoid or rectum, or in spastic narrowing of sphincters, in normal consistency of the stools, a peculiar shape can be observed – *tape-like* or *pencil-like*.

Color of the faeces

The faeces of the healthy subject have various tint of brown color. Brown color depend the presence in the stools of stercobilin and mesobilifuscin, which are formed from the bilirubin under the influence of intestinal bacterium.

Moreover, character of the food, drugs, presence of pathological admixtures have influence on stools color. In milk food faeces are light brown, sometimes yellow; in meat diet – dark brown; in vegetable diet – greenish; in beet ingestion – reddish; in bilberry, black currants, large amount of coffee ingestion – dark.

Medical preparations can also affect color of the stools. Black color of the stools can be due to bismuth, iron; yellow-brown – due to rhubarb, Alexandrian leaf; light yellow or white – due to barium.

Very important diagnostically changes of the stools color that are caused by pathological processes in the digestive organs. In the disease of the liver and bile ducts with upset bile excretory function, the stools are *grayish-white*, clayish, or *sandy* ("acholic faeces"). In the cases of fatty stools (sprue, affection of the pancreas, amyloidosis of the intestine) their color is quite often *gray*. In intensified peristalsis, suppression of intestinal flora (for example during antibiotics therapy), the color of the faeces is *golden-yellow* due to the presence of unbound bilirubin. In significant upper gastrointestinal bleeding, the stools are *black*, tarry (melena) due to formation of sulfur compound of iron. *Red* color of the faeces suggests lower gastrointestinal bleeding (sigmoid, rectum). In typhoid fever the stools have peculiar appearance – "pea-soup stool", in cholera – "rice-water stool" is observed.

Odor of the faeces

Normally, the stools odor is unpleasant, but not strong. It depends on the presence of indol, scatol, phenol, which are formed due to bacterial degradation of proteins.

In predominance of protein food, the smell of the stool increases, in predominance of vegetable and milk food – decreases. Odor can be intensify or diminish depends on duration of stool staying in the intestine. For example, in constipation the faeces almost have no smell, in diarrhea the odor is stronger.

Especially strong, fetid odor of the stool is characteristic of putrid dyspepsia, degradation of the large intestinal tumor.

In intensification of fermentation the smell is acid due to the presence of organic acids.

Admixtures of food origin

Normally, undigested remains of vegetable food (cucumbers, salads, onion, berries, nuts, fruits), and tendons, cartilages pieces are excreted.

In significant insufficiency of gastric and pancreas digestion, or in absence of teeth, the large pieces of undigested food are observed (*lientery*).

Presence in the stools of undigested meat pieces is called *creatorrhea*. Muscular fibers presence is better examined during microscopic study. In achylia, gastroentestomy connective tissue in a form of whitish or grayish fibrous structures can be found.

Significant content of fat in the stools is called *steatorrhea*, which characterized by the appearance of mat brightness on the stool surface (solidified fat).

Pathological admixtures

Mucus. Normal stools contain insignificant amount of mucus that is practically undeterminable. Visible mucus indicates inflammation of intestine mucus membrane. If mucus is mixed with stools, it originates from the small intestine or upper part of large intestine. If mucus is found on the surface of faeces in a form of clots or bands, it originates from the lower part of the large intestine. In membranous colitis, mucus is excreted in the form of dense bands, which can resemble helmints.

Blood is also pathological admixture. Insignificant bleeding from the upper part of the gastrointestinal tract is not determined macroscopically; more significant bleeding changes the color of the stools. In bleeding from the lower part of the gastrointestinal tract, unaltered of red color blood is seen on the stool surface. Blood is easily found, when it mixed with mucus, like in dysentery and ulcerative colitis. In all doubtful cases the blood in the stool can be detected by chemical study.

Pus in the stools is found in ulcerative processes, mainly in the lower part of intestine (for example, in tuberculosis of the large intestine, dysentery, non-specific ulcer colitis, degrading tumor), or in rupture of a paraproctal abscess. Usually pus is mixed with mucus and blood, and rare present in such amount that can be detected in routine inspection of the stools. Microscopic determination of the pus is more authentically.

Concrements, which can be seen in stools, are of bilious, pancreatic or intestinal origin. *Gallstones* can be cholesterol, lime, bilirubin or mixed. They are found after biliary colic, sometimes in some days after it, and in rare cases without preliminary colic. *Pancreas calculus* is of a small size with rough surface. *Coproliths* are large in size, and are of dark-brown color.

Helmints. Such helmints as ascarides (female length is 15–45 cm, male – 15–25 cm), acanthocephala, and platyhelmints can be seen by an unaided eye in stools.

Chemical study

Reaction of the faeces is determined by moistened lithmus paper. Stool reaction depends mainly on vital activity of intestinal microflora; in predominance of fermentation, reaction is acid, in predominance putrefication – alkaline.

In healthy subjects in usual mixed nutrition both processes are balanced, and faeces reaction is neutral or poor alkaline (pH – 6.0–8.0). In protein diet the reaction shifts to alkaline (due to activation of proteolytic – putrid flora), in carbohydrates diet – to acid reaction (due to activation of fermentative flora). But the main factor that caused predominance of that or another bacterium group is the level of disintegration and assimilation of food in gastrointestinal tract. For example, in achylia, pancreatitis due to significant creatorrhea, putrid flora is activated and reaction of stool becomes alkaline. The most typical manifestations of dysbacteriosis of intestine that causes significant shift of the stool reaction are putrid (markedly alkaline reaction) and fermentative (markedly acid reaction) dyspepsia. Acid reaction of stool is found in large amount of fatty acid in it (obstructive jaundice, amyloidosis of the small intestine, etc). More detail information about intensity of fermentative processes is obtained by determining of organic acids quantity, and about intensity of putrefactive processes by determining of ammonia quantity in the faeces.

Bile pigments.

Stercobilin. Normally, the stool contains from 40 to 280 mg of stercobilin. The test for stercobilin is carried out commonly when the faeces are discolored. Absence of stercobilin in the stools (acholic stool) in obstructive jaundice is observed in complete obstruction of common bile duct (obturation by stone, tumor). Sharp decreasing or absence of stercobilin in jaundice

suggests grave parenchymatous damage of the liver. Increase stercobilin content in the stool are found in intensification of erythrocytes hemolysis (anemia).

Bilirubin. Normally, bilirubin is absent in the stools. Unbound bilirubin can be found in infant, adults in enteritis or dysbacteriosis (after antibiotics treatment).

Blood in the faeces. Determination of the blood in stools has significance for diagnostics of ulcer and tumor in gastrointestinal tract, especially if they are accompanied by insignificant bleeding that are not affected the color of the faeces (so-called latent bleeding).

Benzidine test for latent blood is commonly used. Benzidine test is very sensitive, it is possible to determine insignificant amount of blood (0.2 %) in stool. It should be remembered however that positive reaction with benzidine could be observed in ingestion of foods containing blood (meat, fish). Therefore, such foods should be excluded from the diet 2–3 days before test.

Protein. Normally, food proteins are absent in the stools, as they are almost completely split by enzymes in the absence of intensified peristalsis.

The Tribeulet-Vishnyakov test is commonly used to assess protein presence in the faeces. Soluble protein presence in stool suggests intensification of its excretion by intestinal wall in inflammatory processes, ulcerations accompanied by cell decomposition, and bleeding.

Microscopic study

Elements of food origin

Muscular fibers are usually absent normally in the stool, or can be found on a meat diet in a form of separate fibers, which have lost their striated patterns. Increased amount of muscular fibers (*creatorrhea*) in the stools indicates insufficient digestion of meat food in affected gastric digestion (unacidity, achylia, condition after gastrectomia, etc), in enzymatic insufficiency of pancreas (pancreatitis), and in intensified intestinal peristalsis.

Connective tissue is absent in the faeces of healthy person. Presence of connective tissue in a form of semitransparent fibers with indistinct contour suggests abnormal gastric digestion in unacidity condition of the stomach secretion.

Neutral fat and products of its decomposition. Normally, moderate (not more than 10 g) of ingested fat is assimilated almost complete (on 90–95 %). Thus, the remaining fat can be detected as soaps in the absence of

neutral fat. Presence of a great amount of fat in the stools (*steatorrhea*) is a result of decreased lipolytic activity of the pancreas, abnormal absorption in the small intestine, intensified peristalsis and deficient quantity of bile in intestine (fatty acids can be found in such cases).

Vegetable cellulose and starch can be detected among carbohydrates food remains in microscopy of the stools. There are two types of cellulose – digested and undigested. Normally, only undigested cellulose and small quantity of starch grains (or complete their absence) are present in faeces. In pathology digested cellulose and large amount of starch grains (*amylorrhea*) are identified in stools.

Digested cellular tissue is present in stool of the patients with gastric unacidity. In hyperacidity conditions of the stomach, in the diseases of the pancreas amylorrhea can be observed. Diseases of the small intestine accompanied by intensified peristalsis are characterized by significant amylorrhea and elevated content of cellulose in the faeces. In the diseases of large intestine, especially in affection of their upper part, the final phase of cellulose and starch digestion is altered to cause their presence in the stools.

Cellular elements – cells of intestinal epithelium, leucocytes, erythrocytes, macrophages, and malignant cells are identified in the stools, which contain mucus.

Columnar (intestinal) epithelium cells presence in large quantity indicates inflammation of mucus membrane of the large intestine.

Leucocytes. Single leucocytes can be present in the stool of healthy person. Large their accumulations in mucus are detected in ulcerative processes in the large intestine (dysentery, tuberculosis, ulcerative colitis, cancer). Leucocytes in the mucus originated from the small intestine, have time to destroy. In amoebic dysentery, ankylostomidosis, non-specific ulcer colitis, and in some helminthiases large quantity of eosinophils is observed in the stools.

Erythrocytes unaltered are seen in the faeces in bleeding from the large intestine (ulcerative affection, fissures of the anus, hemorrhoids). If the blood originates from the upper part of gastrointestinal tract, erythrocytes are decomposed and can be only detected by chemical study.

Macrophages – cells that are larger than leucocytes, contain round or oval nucleus, with various inclusions (products of phagocytosis) in cyto-

plasm. Macrophages observe in some inflammatory processes, especially in bacterial dysentery.

Malignant cells can be found in the stools in location of malignant tumor in the rectum.

Protozoa and helminthes should be better detected in freshly stool. Ova of following helminthes can be revealed: *Taenia solium, Taenia saginata, Taenia nana, Botriocephalus latus, Distomum hepaticum, Ascaris lumbricoides, Enterobius seu oxyuris vermicularis, Trichocephalus dispar*, etc. Telemann's method is more effective to evaluate the presence of helminthes ova in the stools.

Cysts of following protozoa: *Amoeba dysenteria seu histolitica, Lamblia, Balantidia* can be identified by staining with Lugol's solution.

Functional study of the liver and bile ducts

Pigment metabolism

Bilirubin. Van den Bergh test is used to measure bilirubin content in blood serum. Normally, blood bilirubin content is:

Total bilirubin – 8.5–20.5 mkmol/l,

Bound bilirubin – 2.2–5.1 mkmol/l,

Unbound bilirubin – 6.3–15.4 mkmol/l.

Elevation of total bilirubin level is observed in damage of hepatic cells of inflammatory, toxic and neoplastic genesis; obstruction of the liver ducts (obstructive jaundice); hemolytic conditions; physiological infant jaundice.

Elevation of bound bilirubin level suggests damage of the liver cells (hepatitis, liver cirrhosis), obstruction of the bile ducts, cholestasis, liver abscess, and pregnancy jaundice.

Elevation of unbound bilirubin can be caused by infectious hepatitis, hemolytic anemia, chronic hepatitis and liver cirrhosis.

Bile pigments in the urine reflect functional condition of the liver. Bilirubin and urobilinoids can be revealed in the urine.

The normal urine is practically free from bilirubin. It can contain minimal amount of only bound bilirubin; unbound is insoluble and cannot passes through the healthy renal filter.

Increased amount of bilirubin in urine is called *bilirubinuria*, and appears when bound bilirubin level in the blood elevates to 0.01–0.02 g/l ("renal threshold of bilirubin"). Bilirubinuria is an early sign of liver dysfunction, and occur in hepatic, subhepatic jaundice, and in liver cirrhosis.

Urobilinoids – *urobilin* (urobilinogens) and *stercobilin* (stercobilinogens) are derivates of the bilirubin. Normal urine always contains traces of stercobilinogen. Excretion of large amount of urobilinoids in the urine is called *urobilinuria*. Urobilinuria observes in parenchymatous damage of the liver (hepatitis, cirrhosis), hemolytic conditions (hemolytic anemia, hemoglobinuria), and in intestinal diseases (enteritis, constipation, intestinal impassability).

Protein metabolism

Determination of ***total protein*** and ***its fraction*** in the blood serum. Normally, the total protein content in the blood serum is 70–90 g/l. Total protein consists of two fractions: *albumins* and *globulins*. Total protein contains 56.5–66.5 % of albumins and 33.5–43.5 % of globulins.

Globulins are subdivided into four fractions: α_1-globulins (2.5–5 %), α_2-globulins (5.1–9.2 %), β-globulins (8.1–12.2 %), and γ-globulins (12.8–19.0 %).

Increasing of total protein level indicates chronic diseases of the liver, hyperimmunoglobulinemia, macroglobulinemia, dehydration or venous congestion.

Decreasing of the total protein level suggests grave affection of the liver with hepatic-cellular failure (liver cirrhosis, fatty liver dystrophy, chronic hepatic failure, protein loosing in gastroenteropathy, peritonitis.

Albumin levels increasing in the blood serum depend on damage degree of the liver parenchyma (chronic hepatitis, liver cirrhosis, liver cancer).

Globulins levels increasing reflect activity of chronic hepatitis and indicate immunoinflammation:

α_1-globulins (reaction of the acute phase) – acute, subacute and chronic inflammatory processes, malignant tumor;

α_2-globulins – subacute and chronic inflammatory processes, hemolysis, malignant tumor;

β-globulins – primary and secondary hyperlipoproteinemia;

γ-globulins – chronic diseases of the liver, chronic infections, autoimmune hepatitis, liver cirrhosis, chronic active hepatitis.

Globulins level decreasing observes in:

α_1-globulins – deficiency of α-antitripsine;

α_2-globulins – pancreatitis, diabetes mellitus;

β-globulins – hypobetalipoproteinemia;

γ-globulins – physiological in 3–4 month infants, congenital hypo- and ungammaglobulinemia.

Thymol test – is so-called protein sedimentation test that reflects shift of albumin-globulin fractions. Normally, thymol test is 0–5 Units. The test is always positive in virus hepatitis, toxic hepatitis, chronic hepatitis, chronic cholecyctitis, and liver cirrhosis. It is negative in obstructive jaundice.

Prothrombin index. Protrombin – is protein that is synthesized in the liver with participation of vitamin K. Not absolute its amount is measured, but in relation to standard of healthy person. Normal prothrombin index is 90–105 %. Elevation of prothrombin index occurs in obstructive jaundice, hypovitaminosis K, damage of the liver parenchyma, and intestinal dysbacteriosis.

Decreasing of the prothrombin index reflects degree of hepatic-cellular failure.

Lipid metabolism

Normally, *total cholesterol* content in the blood is less than 5.2 mmol/l.

Increasing of total cholesterol revealed in the hepatic diseases accompanied by the bile congestion in the liver (obstructive jaundice, cholestatic hepatitis, malignant tumor of the pancreas).

Decreasing of the total cholesterol in the blood arises in the liver cirrhosis, hepatic-cellular failure, and malignant tumor of the liver).

Normal β-*lipoprotein* content in the blood serum is less than 4.9 mmol/l. Increasing of the β-lipoprotein level is typical to acute hepatitis; decreasing – observes in long-standing starvation.

Study of the liver enzymes

To assess functional condition of the liver, content of enzymes in the blood serum is measured.

Aldolase normal content is 0.09–0.57 mmol/h×l. Elevated aldolase levels are found in acute hepatitis (infectious and toxic), chronic hepatitis, tumor of the liver, hemolytic anemia.

Alanine aminotransferases (ALT) normal level is 0.1–0.68 mmol/h×l. Increasing of ALT blood level are revealed in necrosis of the liver cells, acute and chronic hepatitis, cholangitis, fatty hepatic dystrophy, liver cirrhosis, obstructive jaundice, liver tumor, hemolytic conditions.

Aspartate aminotransferases (AST) normal content is 0.1–0.45 mmol/h×l. Increased AST blood levels occur in necrosis of liver cells, obstructive jaundice, acute and chronic hepatitis, fatty dystrophy of the liver.

Alkaline phosphatase normal level is 0.5–1.3 mmol/h×l according Bodanovsky or 1–3 mmol/h×l according Becey-Lauri. Increased level of alka-

line phosphatase is especially pronounced in obstructive jaundice, and can also be observed in cholangitis, tumor of the bile bladder, chronic hepatitis, liver abscess, tumor of the liver, and liver cirrhosis.

Increased activity of aldolase, ALT, AST, alkaline phosphatase suggests hepatocytes lysis and release of these enzymes in the blood.

Lactate dehydrogenase (LDG) normal blood serum content is to 3.2 mkmol/s×l. Increased LDS level in the blood is determined in hepatitis and liver tumor.

Cholinesterase level normally is 160–340 mmol/h×l. Decreased cholinesterase content is detected in hepatitis, liver cirrhosis, liver tumor, and congestive liver.

Sorbitol dehydrogenase (SDG) normal level is 0–5.6 nmol/s×l. Elevated SDG level is found in grave damage of the liver parenchyma (acute infectious hepatitis, toxic hepatitis, liver cirrhosis, liver tumor).

Ceruloplasmin normal content is 1.52–3.31 mkmol/l. increased level occur in liver cirrhosis, hepatitis, obstructive jaundice; decreased – in grave liver diseases, in Konovalov-Wilson disease (congenital disease characterized by decreased synthesis in the liver of ceruloplasmin, the copper transport protein and its increased deposition in tissues).

Combination of changes of enzyme activity gives important diagnostic information:

- Increased activity of ALT, AST, LDG, SDG and hyperbilirubunemia with predominance of bound fraction indicates mainly necrotic damage of the liver (hepatocytolisis, liver necrosis) – *cytolisis syndrome*;
- Increased activity of alkaline phosphotase in combination with hyperbilirubunemia, hypercholesterolemia, and β-lipoproteinemia is typical to cholestatic hepatitis, long-standing and grave obstructive jaundice – *cholestatic syndrome;*
- Decreased albumins, cholesterol, protrombin contents in combination with hyperbilirubinemia is characteristic of *hepatic-cellular failure syndrome*;
- Increased γ-globulins level, hyperproteinemia, elevated thymol test and immunoglobulins G and M contents are typical to immunoreactive damage of the liver – *immunoinflammatory syndrome*.

Chapter 6. URINARY SYSTEM

FUNCTIONAL AND CLINICAL ANATOMY

The urinary organs consist of the pairs of kidneys and ureters, and the single urinary bladder and urethra (Fig. 6.1). The noun 'kidney' is of Anglo-Saxon origin, but the Latin and Greek for 'kidney', 'ren' and 'nephros'.

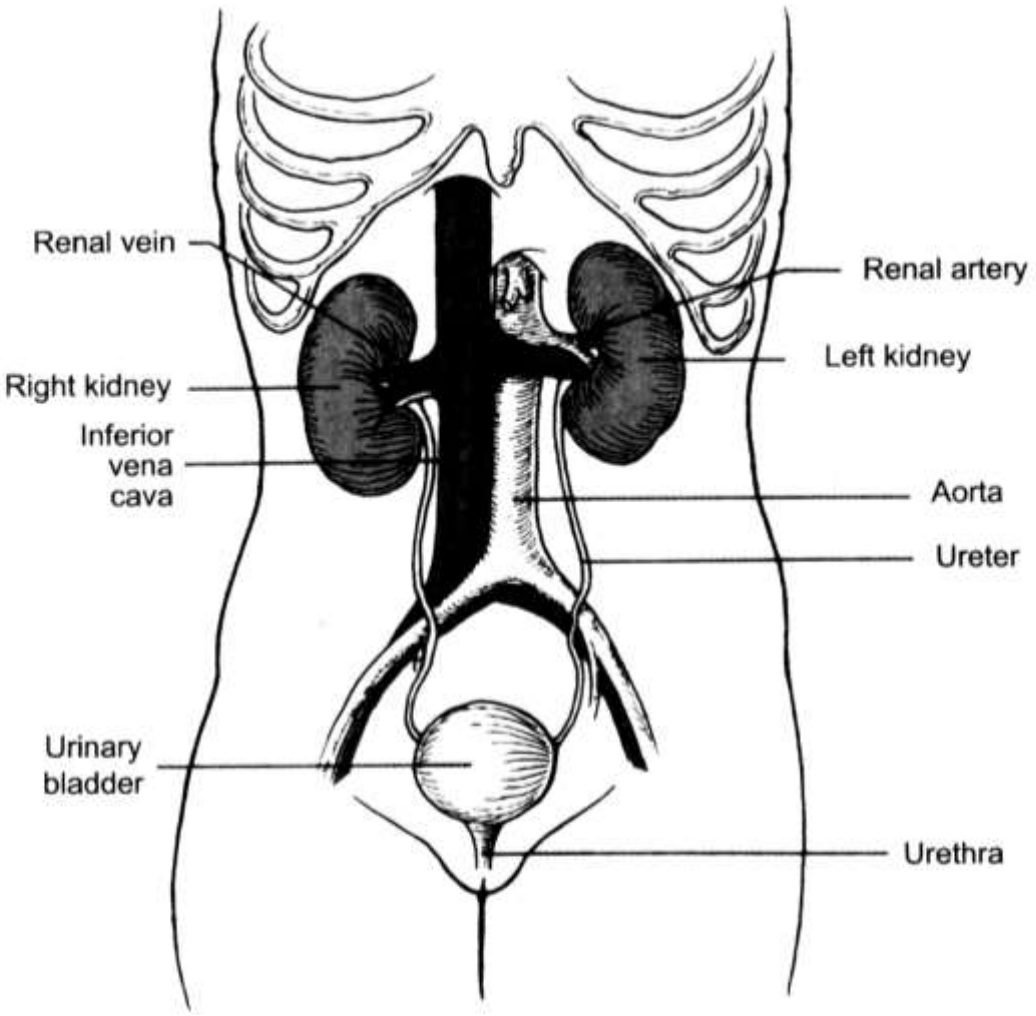


Fig. 6.1. Urinary organs.

Kidneys are excretory organs, which generate and excrete the urine. Kidney has bean-like shape (Fig. 6.2); the length of each kidney is from 10 to 12 cm, mass – from 150 to 160 g. Each kidney lies in the upper posterior

part of the abdomen extraperitoneally at the level of middle of XI thoracic vertebra to the upper edge of the III lumbar vertebra. Right kidney is placed somewhat lower than left one. The kidneys are covered by dense fibrous capsule; their surface is smooth. Two margins are distinguished: lateral margin and medial margin. The region of medial margin where the blood vessels (renal artery and vein) and ureter enter or leave is the hilus. The kidney has two extremities (or poles): superior extremity and inferior extremity.

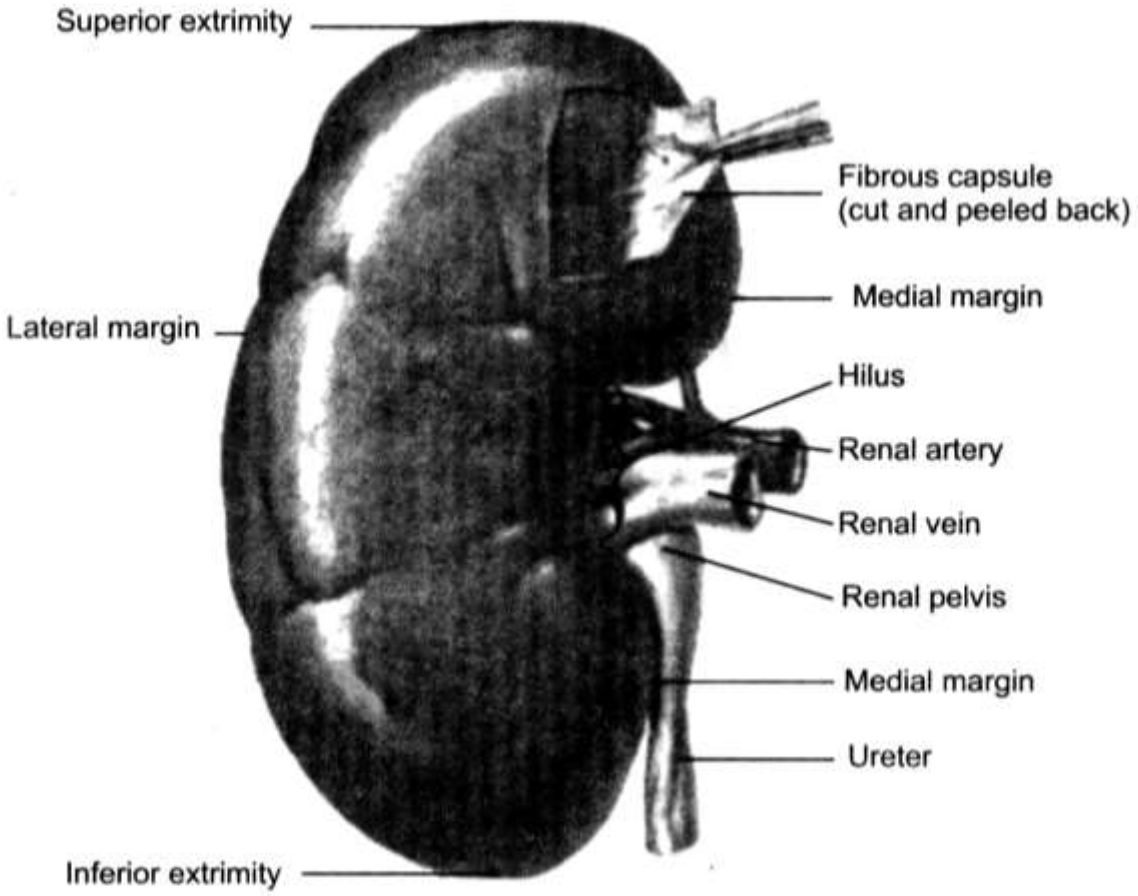


Fig. 6.2. Anterior surface of the right kidney.

Cross structure of the kidney is represented in Fig. 6.3. Two layers are well visible: cortical and medullar. Cortex in a form of renal column divides medulla in pyramids. Apex of each renal pyramid forms renal papilla that opens into renal calyces, which are united in renal pelvis that continues by ureter.

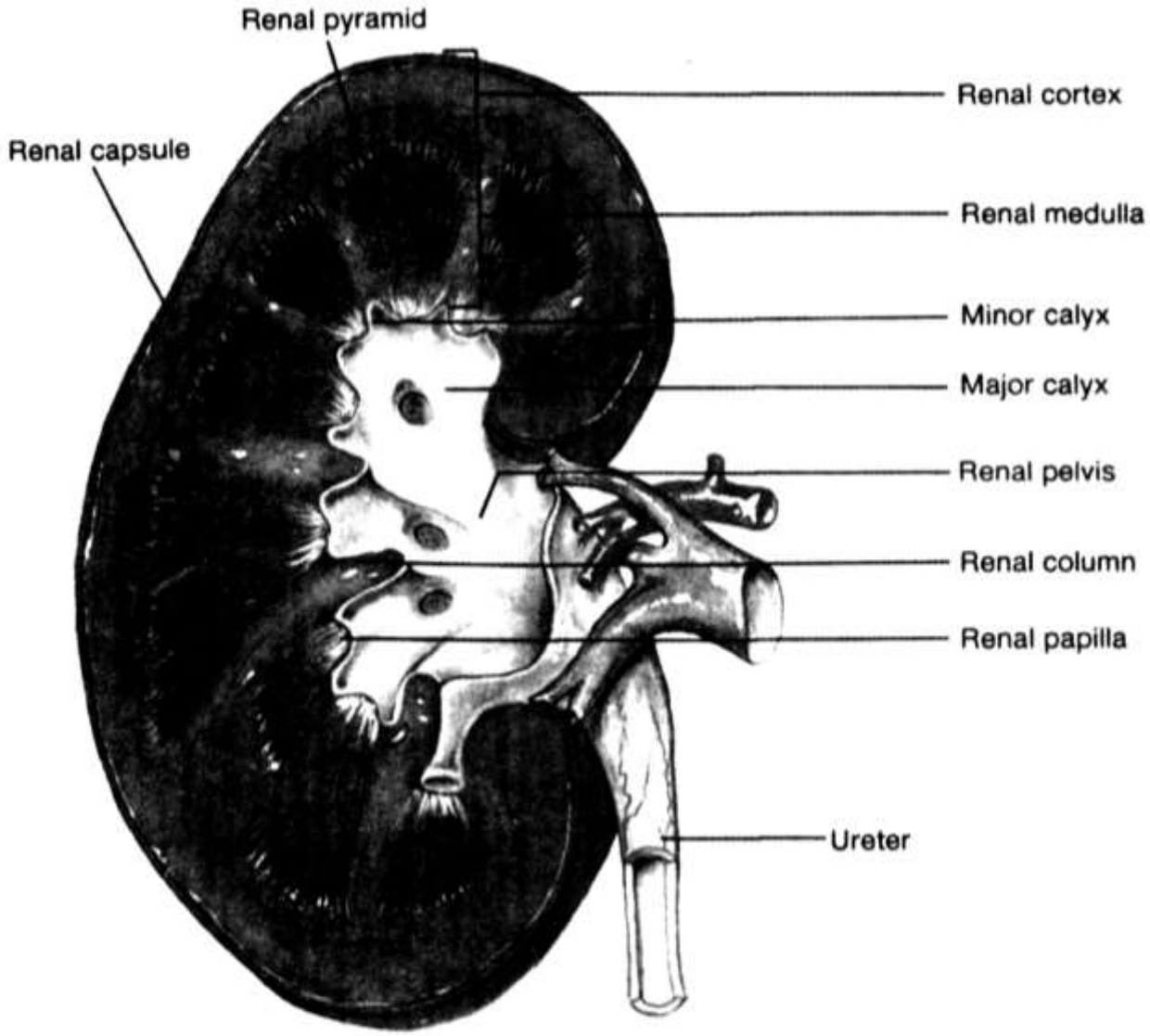


Fig. 6.3. Right kidney sectioned in several planes, exposing parenchyma and renal pelvis.

Each ***ureter*** is a muscular tube (of smooth muscle), about 25 cm long, that passes down on the posterior abdominal wall into the pelvis to enter the urinary bladder.

The ***bladder*** is a muscular storage bag (of smooth muscle) that lies in the pelvis behind the pubic symphysis – in front of the upper part of the vagina in the female or in front of the rectum in the male.

The bladder empties through the ***urethra***. Urethra is 2.5–3.5 cm long in female and runs in the front wall of the vagina and is surrounded by the external urethral sphincter to open at the external urethral meatus at the front of the vagina 2.5 cm behind the clitoris. The urethra in the male is about

16 cm long. The first 3 cm is the prostatic part; it runs through the prostate, where it is joined by the ejaculatory ducts – from here onwards the urethra is a common pathway for urine and seminal fluid. At the beginning of the prostatic part, the male urethra is surrounded by the internal urethral sphincter, which prevents regurgitation of semen into the bladder during ejaculation. The short membranous part, about 2 cm long and surrounded by the external urethral sphincter, continues into the penile part within the corpus spongiosum of the penis, to open on the glans penis at the external urethral meatus.

The functional units of the kidney are the *nephrons* (2 million in each kidney), which are delicate coiling tubules that form the urine and pass it on to the collecting tubules, from which it eventually reaches the ureters (Fig. 6.4). Each nephron is intimately associated with a glomerulus, a tuft of blood capillaries, which, during development, indents the blind end of the nephron. As blood courses through the glomerulus, fluid and many dissolved materials filter out of the capillaries and into the lumen of the nephron. The filtrate is further modified by selective absorption and secretion as it passes through the rest nephron and then finally into the collecting tubule. The formation of urine thus depends essentially on glomerular and tubular filtration.

METHODS OF EXAMINATION

Inquiry

The most common complaints of the patients with renal diseases are: pain, deranged urination (dysuria), and edema.

Pain. The patients with urinary organs diseases complain on pain of various location and character (Tab. 6.1). Location of the pain depends on the site of urinary tract affection. Pain of renal origin is usually felt in the lumbar region, pain of ureter origin – along their course, pain of bladder origin – is suprapubical.

The renal tissue is free from pain receptors. The pain in the diseases of the urinary organs can be caused by distension of the renal capsule, or by spasmodic contraction and distension of the pelvis, ureter, and urethra.

Loin pain was described by Hippocrates and remains an important symptom. The classic form is ureteric colic, often misnamed *'renal colic'*.

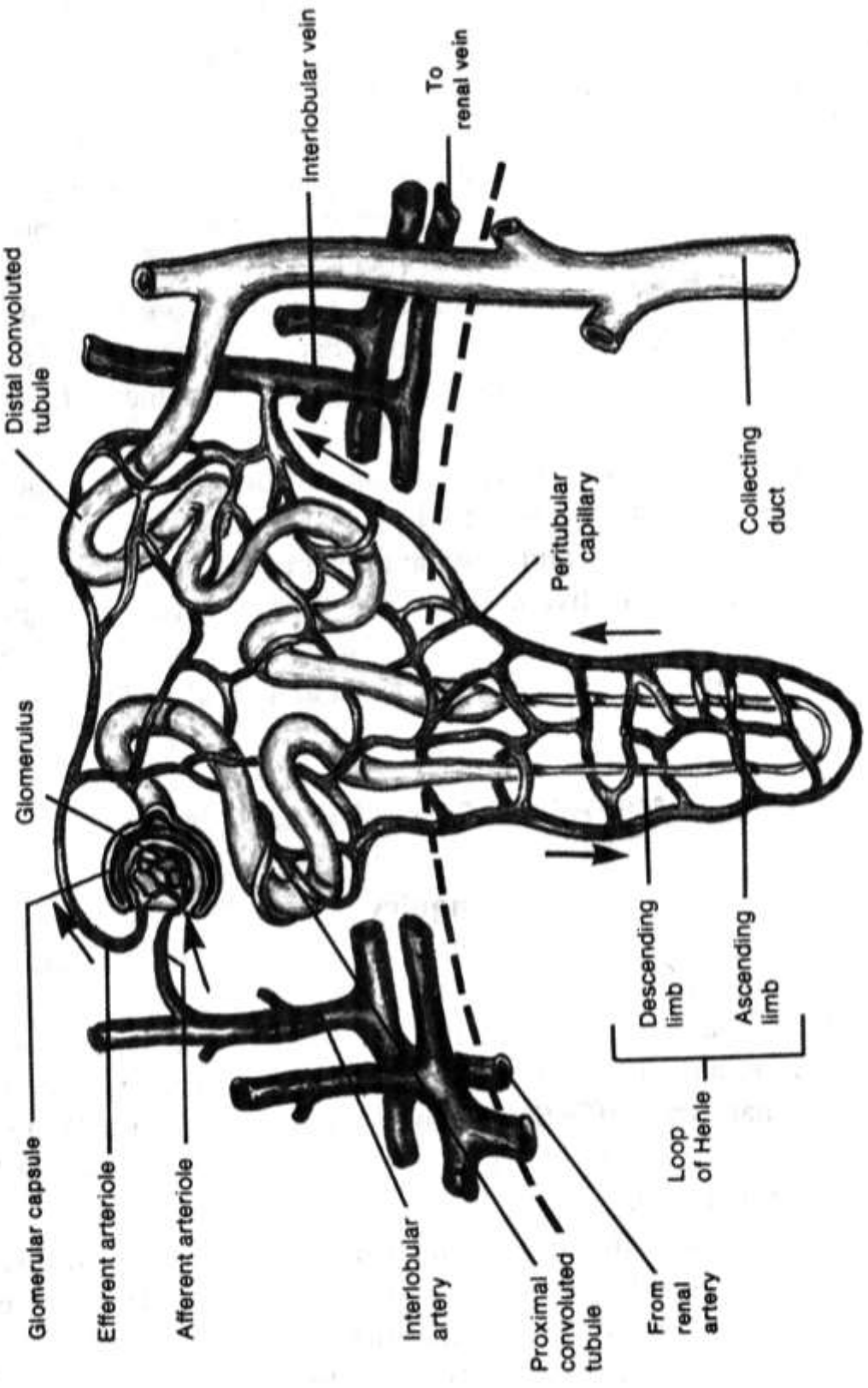


Fig. 6.4. Nephron and collecting tubule: Schema.

This is acute, usually severe, often indescribably so, and waxes and wanes in a typical colicky pattern; the suffer thrashes about, unable to find comfort, sweats with the agony, and is often pale. Pain is usual unilateral and radiates from the loin into the abdomen, and down into the testicle, labium, or upper thigh. Colic pain can be provoked by taking much liquid, jolting motion. Sudden relief may occur if the stone, blood clot, sloughed papilla, or whatever is causing the obstruction moves on, only to recur when it impacts again. Atropine sulphate, hot water bottle or warm bath can lessen or remotes renal colic.

Renal pain, as opposed to ureteric colic, is usually static, dull, constant, and felt in the loin. Sometimes there is obvious renal swelling, with or without tenderness, and heat if the infection is present. The commonest causes of this dull and boring pain are acute glomerulonephritis, renal abscess, 'congestive kidney' (in heart failure). Acute pyelonephritis will also give rise to similar unilateral pain, and a rare differential diagnosis of renal infarction by in situ thrombosis or embolism in the renal artery tree. Less severe and uncommon renal pain may be a symptom of chronic glomerulonephritis.

Tab. 6.1. ***Pain in selected urinary diseases.***

Disease	Location Radiation	Character
1	2	3
Nephrolithiasis	Loin pain, by the ureters course, more frequent unilateral, downward radiation	Periodic, intense, renal colic
Pyelonephritis	Loin pain, bilateral, without radiation	Dull, constant, increasing in intensity, accompanied by irregular fever
Renal abscess	Loin pain, unilateral	Pain and muscular tension, accompanied by fever, chills, headache, and symptoms of bacterio-toxic shock

Continue of Tab. 6.1

1	2	3
Renal infarction	Loin pain, unilateral	Occur suddenly, intense, accompanied by excretion of red urine
Nephroptosis (movable kidney)	Loin pain, unilateral, inconstant pain location	Periodic, sometimes renal colic like, intensified in upright position, in physical exertion, in jolting motion, relieved in lying posture at rest
Acute glomerulonephritis	Loin pain, bilateral, without radiation	Dull, of insignificant intensity, in some patients the pain is absent
Congestive kidney	Loin pain, bilateral, without radiation	Dull, depend on degree of edematous syndrome
Cystitis	Suprapubical, increased in palpation	Pain is provoked by urination, most intense and burning at the end of it. Imperative increasing of urination by small portions of urine
Urethritis	Urethra region	Burning pain in urethra, increasing in urination, accompanied by ample, purulent excretions from the urethra and painful and frequent erections

In the patients with cystitis the pain is provoked by urination; in stranguria – difficult and tenderness urination is typical; in urethritis – is characterized by burning pain in the urethra that arises during or after urination.

Deranged urination. Healthy person excretes approximately 1–2 liters of urine in 24 hours; urination rate is 3–6 times a 24 hours; ratio of daily

and nightly amount of excreted urine is 3:1 accordingly. Urination is free and painless.

Urine volume

The volume of urine passes varies greatly from day to day in normal individuals, under very precise stimuli, to maintain an essentially constant body fluid volume.

Polyuria describes the excretion of larger than normal volume of urine (exceed 2 liters of urine/24 h). Polyuria is most commonly caused by a habitual high fluid intake, but is also a feature of the glycosuria of diabetes mellitus and initial stage of renal failure. Less common causes include diabetes insipidus of cranial (defective secretion of arginine vasopressin – pituitary diabetes insipidus) or nephrogenic (failure of the renal tubules to respond normally to arginine vasopressin – nephrogenic diabetes insipidus) origin and hypokalaemia and hypercalcaemia (Tab. 6.2).

Tab. 6.2. ***Polyuria causes.***

Causes		
Extrarenal		Renal
Physiologic	**Pathologic**	
• High fluid intake • Neurogenic factors	• Resolution of: – edema – transudate – exudate • Diabetes mellitus • Diabetes insipidus (to 4–6 l and more)	• Chronic renal disease at initial stage of renal failure

Some patients suffering from frequent urination may complain of passing excessive amounts of urine, and it is thus important to distinguish true polyuria from frequency of micturition in such cases.

Oliguria, arbitrarily defined as the production by an adult of less than 500 ml of urine/24 h. Oliguria is normal in hot climates or where intake has been restricted. Oliguria is abnormal when the kidney is damage and unable to excrete water – as in obstruction, acute renal parenchymal disease,

or failure of renal perfusion (Tab. 6.3). In any oliguric patient the first steps in diagnosis are to establish whether the patient has had an adequate intake, has had excess extrarenal losses, is already overloaded because of failure to excrete a normal amount of intake, or has urinary tract obstruction at some level with an enlarged bladder or dilated upper tract. The composition of the urine, the state of the circulation and renal perfusion, as well as the history if available, will help in differentiated the various possibilities.

Tab. 6.3. ***Oligyria causes.***

Causes		
Extrarenal		Renal
Physiologic	**Pathologic**	
• Limited fluid intake • Loss of fluid with sweat in hot weather or during physical exertion	• Decreased cardiac output • Loss of large amount of fluid by non renal way: - pronounced sweatiness in fever, - profuse diarrhea, - intense vomiting, - bleeding • Shock, collapse	• Acute nephritis (to 200–300 ml/24h) • Nephrotic syndrome in edematous phase • Acute renal failure (hemolytic, toxic kidney) • Amyloidosis • Obstruction of the ureter, bladder, or urethra

Anuria is defined as complete absence of urine secretion and/or excretion. Anuria can be caused by damage of the renal parenchyma – *secretory anuria*, or by obstruction of urinary tract (ureters, bladder, or urethra) – *excretory or obstructive anuria* (Tab. 6.4). *Reflex anuria* can be arises in severe pain of various origins. If anuria persists several days, uraemia can be develops or even fatal outcome.

Anuria should be differentiated from ***ischuria***, when the secretion of the urine is normal but the urine is retained in the bladder because patient is unable to evacuate it. Ischuria is observed in the patients with compression or other affection of the spinal cord, and also in unconsciousness state.

Tab. 6.4. ***Anuria causes.***

Causes	
Secretory anuria	**Excretory anuria**
• Acute renal failure • Terminal stage of the chronic renal failure • Acute glomerulonephritis • Nephrosclerosis • Sepsis • Poisoning with nephrotoxic substances • Severe heart failure • Collapse • Shock • Transfusion of incompatible blood • Dehydration (profuse bleeding, vomiting, diarrhea) • By reflex in severe pain	• Mechanical obstruction of the ureters or urethra by: – stones – inflammatory edema of the mucus membrane – proliferation of the malignant tumor – prostate adenoma – prostate tumor

Nocturia (nycturia) is defined as passing of more than one-third of the total 24-h urine volume by night.

In health there is a diurnal variation in renal function, with relative retention of both water and solute by night. Normal adult excrete between 1 and 2 liters of urine in 24 hours, of which 60 to 80 % is passed by day. This diurnal rhythm may be abolished or even reversed in edematous states, in chronic renal diseases, malabsorption, adrenal insufficiency, and in some cases after head injury or renal transplantation (Tab. 6.5). It is therefore sometimes useful to record day and night urine volumes separately. It should be noted, however, that normal elderly subjects tend to pass almost the same amount of urine during the night and the day.

Tab. 6.5. ***Nocturia causes.***

Causes	
Renal	**Extrarenal**
• Chronic renal diseases at renal dysfunction (nocturia with polyuria): – chronic glomerulonephritis, final stage – chronic pyelitis – vascular nephrosclerosis, etc	• Heart failure (cardiac nycturia – nycturia with oliguria during day time) • Prostate adenoma • Diabetes insipidus

Urination frequency

Healthy individuals urinate from 3 to 6 times a day. The volume of urine passed during one micturition is from 200 ml to 300 ml. These patterns can be change depend on various physiological and pathological causes.

Pollakiuria is traditionally defined as frequent, more than 6 times a day, urination. Pollakiuria with polyuria can be observed in cold climate and in high fluid intake. It should be noted that pollakiuria is not always associated with polyuria. Pollakiuria with excretion of meager quantity of urine (oliguria) is typical to cystitis. Frequent desire to urination is also characteristic of prostatitis, urethritis, stones in the urine bladder, prostate adenoma, decreased volume of the bladder, and diuretics taking.

Ollakiuria is rare micturition that not always associated with oliguria. The commonest causes of ollakiuria are limited fluid intake, eating of much salted food, excessive sweating in hot climate or fever, and neuroreflex disorders.

Enuresis is involuntary urination without desire. Enuresis observes in organic damage of central nervous system and spinal cord, urinary tract defects, and functional disorders in children.

Dysuria is painful urination, which is frequent symptom of inflammatory diseases of urinary tract, such as cystitis and urethritis.

Isuria – urination at about equal intervals with passage of about equal amounts of urine is common symptom of chronic renal failure.

Stranguria is defined as passage of small amounts of urine (by drops). This symptom can be caused by pathological processes in the urethral

sphincter, stricture (after operative) of the bladder cervix, strangulation of stones or foreign bodies in the urethra, bladder tumor, and phimosis.

Definition and causes of urination disorders are summarized in Tab. 6.6.

Tab. 6.6. ***Disorders of urination.***

Term	Definition	Causes
1	2	3
Polyuria	Urine volume exceed 2000 ml	High fluid intake Diabetes mellitus Renal failure initial stage
Oliguria	Urine volume is less than 500ml/24h	Hot climate Restricted fluid intake Ureter, bladder, urethra obstruction Acute renal parenchymal diseases Failure of renal perfusion
Anuria	Complete absence of urine secretion and/or excretion	Renal failure Acute glomerulonephritis Nephrosclerosis Sepsis, collapse, shock, poisoning by nephrotoxic substances Dehydration By reflex in severe pain Mechanical obstruction of the urinary tract
Ishuria	Absence of urine excretion	Damage of the spinal cord Loss of consciousness
Nocturia	Passing of more than one-third of total 24-h urine volume by night	Chronic renal diseases with renal dysfunction Heart failure Prostate adenoma Diabetes insipidus

Continue of Tab. 6.6

1	2	3
Pollakiuria	Frequent more than 6 times a day micturition	High fluid intake Cold climate Cystitis Prostatitis Urethritis Stones in the bladder Prostate adenoma Diuretics taking Decreased volume of the urine bladder
Ollakiuria	Rare micturition	Low fluid intake After much salted food Excessive sweating (hot climate, fever) Neuroreflex disorders
Enuresis	Involuntary urination without desire	Organic affection of the central nervous system and spinal cord Urinary tract defects Functional disorders in children
Dysuria	Painful urination	Cystitis Urethritis
Isuria	Urination at about equal intervals of about equal amounts of urine	Chronic renal failure
Stranguria	Passage of small amounts of urine (by drops)	Stricture (after operative) of the bladder cervix Strangulation of the stones or foreign bodies in the urethra Bladder tumor Phimosis

Edema is important and common symptom of the urinary organs diseases. Renal edema has following peculiarities. Patients complain on edema that initially arises on the face. In disease progression renal edema spreads from the face downward, up to anasarca. Edema is characteristic of acute and chronic glomerulonephritis, especially in nephrotic syndrome presence, amyloidosis, and acute renal excretory dysfunction (anuria).

Complaints concerning general condition. Patients with chronic renal diseases (glomerulonephritis, pyelonephritis) complain on *general weakness, fatigue* in development of functional disorders.

Fever can indicate infectious inflammatory affection of the kidneys and the urinary ducts, or can be the sign of the main disease, which cause damage of the kidneys. Hectic fever accompanied by chills and profuse perspiration is typical to acute pyelonephritis or aggravation of chronic pyelonephritis. High temperature (to 39–40 °C) observes in the patients with renal abscess, acute paranephritis. Subfebrile temperature (37–38 °C) can be detected in nephrolithiasis during attack of renal colic. In urinary ducts obstruction and congestion of the urine, the fever is constant. Recurrent fever is the sign of kidney tuberculosis. Insignificant elevation of body temperature arises in the patients with acute glomerulonephritis, in chronic glomerulonephritis fever, usually, is absent.

Itch of the skin occurs in the patients with severe renal failure.

Perspiration arises in the patients with renal inflammatory diseases. Considerable perspiration is common symptom of purulent destructive damage of the kidney and perirenal cellular tissue, and specific tubercular process.

Change of body mass, as a rule, weight loss may occur as a consequence of chronic renal failure, in tuberculosis and tumor of the kidneys, polyps, and in tumor of the urine bladder.

Nervous system. Patients with urinary organs diseases may complain on decreased work capacity, impaired memory and attention, deranged sleep (insomnia), headache, dizziness, flashing lights before eyes, weakness in the extremities. All these symptoms are the result of elevated blood pressure, or encephalopathy and polyneuropathy that arise as complication of the chronic renal failure. Deranged vision and hearing are due to hypertension in the patients with acute and chronic glomerulonephritis, pyelonephritis, nephropathy, and in renal vascular pathology presence.

Respiratory system. The common complaints of the patients with renal diseases are cough with insignificant amount of the sputum and dyspnea that increase in intensity corresponding to worsening of the renal function. These complaints are characteristic of chronic glomerulonephritis, pyelonephritis, and are due to the accompanied pneumonia. Pronounced breathing disorders observe in the patients with chronic renal failure, so-called "uremic lung" is formed. Patients complain on dyspnea in insignificant physical exertion, and asthma attacks. In case of uremic pleurisy, dyspnea develops quickly up to acute respiratory failure with circulatory disorders.

Cardiovascular system. Pain in the heart region, retrosternal pain, palpitation, dyspnea, and suffocation of cardiac asthma type are typical to acute glomerulonephritis, and also to other diseases, which are accompanied by elevated blood pressure. All these complaints are revealed in chronic renal failure, and are caused by metabolic disorders that lead to formation of cardiomyopathy.

Digestive system. Loss of appetite and pain in the upper part of the abdomen are early signs of renal dysfunction. Dyspeptic disorders, such as dryness and unpleasant taste in the mouth, nausea, vomiting can also be observed. At the final stage of chronic renal failure meteorism, diarrhea alternated with constipation arises that suggest enterocolitis. These symptoms are the result of uremic gastroenteropathy.

Anamnesis morbi. In acute renal diseases it is necessary to establish time of diseases onset, possible connection with previous infectious diseases, such as tonsillitis, scarlet fever, otitis, acute respiratory disease, etc. It should be note consequence and dynamic of symptoms occurring. Edema arising, blood pressure elevation, and changes of the clinical urinalysis 2–3 weeks after infectious diseases is typical to acute glomerulonephritis. Dysuria on the base of toxicosis (elevated temperature, chills) after cold is characteristic of pyelonephritis.

It is important to ask patient about possible industrial or domestic poisoning, and taking of nephrotoxic drugs (antibiotics, sulpha preparations, preparations of bismuth, silver, etc).

Chronic affection of the kidneys and urinary ducts can for a long time be latent, and are revealed occasionally. In such cases it is difficult to establish the onset of disease. Therefore the patient should be asked about

previous edema, or possible dysuria, and loin pain. In established previously disease, it should noted character of the disease course, frequency and cause of exacerbations, consequence of clinical symptoms, results of previous examination and treatment.

Anamnesis vitae. It is necessary to note previous infectious diseases (scarlet fever, influenza, etc), presence of infectious center (otitis, tonsillitis, adnexitis), and diseases with disorders of urine passage (prostate adenoma). Such diseases as diabetes mellitus, tuberculosis, collagenosis, hemoblastosis, and infectious diseases of genitals can cause renal affection. In the patients with long- standing chronic purulent pulmonary diseases, tuberculosis, osteomyelitis, rheumatic arthritis, Bekhterev's disease secondary amyloidosis of the kidneys can occur. Primary amyloidosis is of congenital character. Nephrolithiasis can be also inherited, thus it is important to know about diseases of the urinary organs and hypertension presence in the relatives of the patient.

Chronic poisoning – narcotics, smoking and alcohol abuse are accompanied by the kidneys damage.

When questioning women, it should be remember that in pregnancy difficult urine passage can observes, and frequently pyelitis and pyelonephritis occur. Women should be asked about edema and elevation of blood pressure during pregnancy – so-called nephropathy of pregnancy.

General Inspection

General condition of the patients with urinary organs pathology is usually satisfactory or of moderate gravity. In renal colic, significant toxicosis, in transition of the disease to renal failure, the condition of the patient is grave; in severe renal failure and uremic coma – extremely grave.

Consciousness of the patients is as a rule clear. Developing of uremia is accompanied by consciousness disorders: stupor, sopor, and coma.

Posture of the patients at initial stage of renal pathology is commonly active. In uremic coma passive posture of the patients is observed. In paranephritis the patient assumes forced posture: lying on affected side with flexed leg, bringing the knee to the abdomen (right leg – in right-sided paranephritis, left leg – in left-sided). During the renal colic the posture of the patient is also forced: he is restless, tosses in the bed, try to find posture that can relieve pain, groans or even cries from pain.

Skin is pale, waxy due to anemia and spasm of skin arterioles by edematous fluid. In renal failure dryness of the skin can be detected. It is possible to observe scratches on the skin in consequence of itch. The skin of the face, neck and hands may be of dirty-gray color due to urochromes formation in the skin. In uremia unpleasant smell of ammonia is felt from the mouth and skin of the patient.

Face of the renal patient has peculiarities. *Facies nephritica* – is swollen, pallid face with edematous and narrowed eye-slits.

Renal edema. Development of edema of renal origin is quite specific. Initially edema appears on the face in the morning, has descending character, and can develop very quickly (in few hours). Edema spreads on extremities, loin region, then fluid accumulates in cavities (ascitis, hydrothorax, hydropericardium), and general edema (anasarca) can arises. The skin over edema is glossy. Renal edema should be differentiated from cardiac edema (Tab. 6.7).

Tab. 6.7. ***Symptomatic features in the differential diagnosis of renal and cardiac edema.***

Features	**Renal edema**	Cardiac edema
Location, character	Descending character, starts from the face and spreads downward	Ascending character, starts from low extremities and spreads upward
Time of arising	More pronounced in the morning	More pronounced in the evening
Color of the skin	Pallor	Cyanotic
Temperature of the skin over edema	Warm	Cold

Inspection of the loin region and abdomen usually not allows determining any changes. Only in paranephritis and considerable tumor of the kidney protrusion of abdominal wall or loin on the corresponding side is detected. In overfilled bladder due to retention of urine, protrusion in suprapubical region is revealed.

Palpation

Kidneys are not usually palpable because of their posterior extraperotoneal location; anterior approach is interfering by the costal arch. Normal kidneys may be palpable in thin persons with well-relaxed prelum, and in nephroptosis. To palpate kidneys it is possible only in considerable their enlargement (at least 1.5–2 times). Causes of unilateral kidney enlargement include hydronephrosis, formation of cyst or tumor. Bilateral enlargement of the kidneys suggests polycystic diseases. The kidneys may also be palpable in cases of their displacement by tumor, or in cases with a floating kidney.

Palpation of the kidneys according to Obraztsov-Strazhesko

The patient should be in lying posture with stretched legs, and placed on the chest arms (Fig. 6.5).

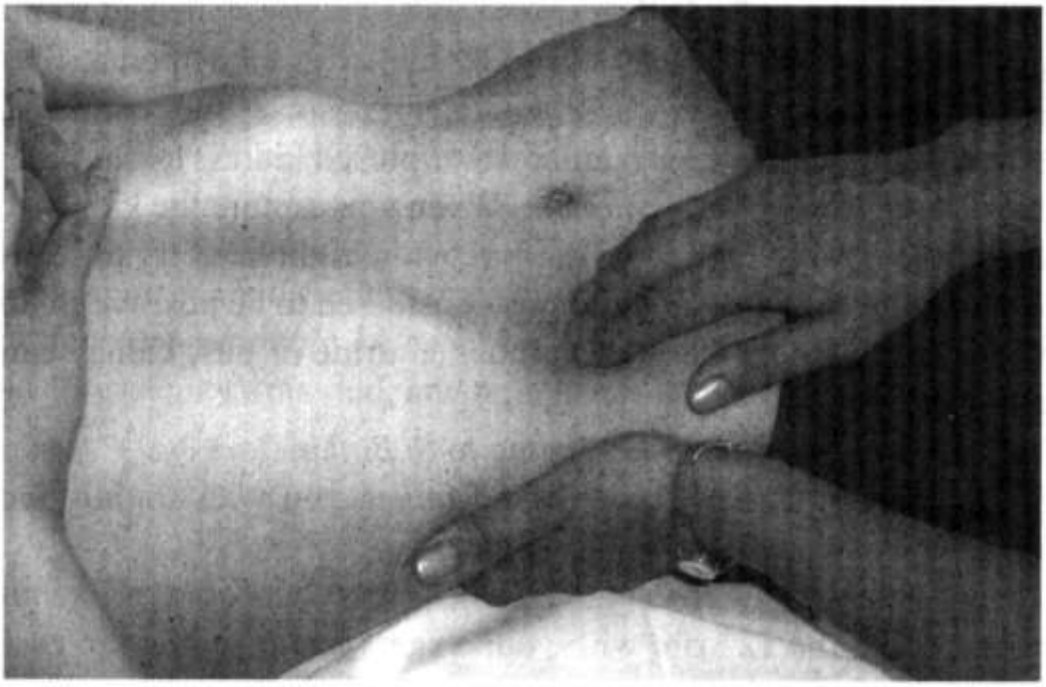


Fig. 6.5. Palpation of the right kidney in lying posture.

Sit by the right side of the patient. During palpation of the right kidney, place your left hand on the patient's loin just below and parallel to the 12th rib so that the fingertips are near the spinal column. During palpation of the left kidney move your left hand to the patient's left lumbar region. Place your right hand on the abdomen just below corresponding costal arch, lateral and parallel to the rectus abdominis muscle. Ask the patient to relax the abdominal muscles and to breath deeply and regularly. With each expiration press

by right hand firmly and deeply to reach the posterior wall, while lift your left hand, trying to displace the kidney anteriorly. Try to "capture" the kidney between your two hands. Ask the patient to breath deeply by "the abdomen". If kidney is slightly descended or enlarged, the lower pole of the kidney in inspiration reaches the fingers of your right hand to slide back into its expiratory position. Slightly press kidney toward posterior abdominal wall and slide over the anterior surface of the kidney bypassing its lower pole.

The left kidney is rarely palpable. If the kidney is palpable, describe its shape, size, surface, tenderness, consistency, and mobility.

The right kidney, if it is located more anteriorly, must be distinguished from the liver. The liver edge extends farther medially and laterally; it cannot be capture. The lower edge of the liver tends to be sharper, the lower pole of the kidney is rounded. The kidney must also be distinguished from overfilled large intestine, tumor of pararenal cellular tissue (lipoma, fibroma), gall bladder, or displaced spleen, with which it is sometimes confused.

In contrast to these organs, enlarged or ptosed kidney tosses up by ballottment – so-called *Guyon's symptom*. When you capture the kidney, by the left hand strike rapidly in the loin region, by the right hand try to feel vibration of the kidney. Guyon's symptom is considered to be positive if you feel this vibration. Enlarged, due to accumulated urine or pus, kidney can give fluctuation in palpation.

Palpation of the kidneys according to S. Botkin

Sit on a chair, patient should stand facing you with slightly inclined toward trunk (Fig. 6.6).

During palpation of the right kidney, place your left hand behind the patient parallel to the 12th rib, with your fingers just reaching the costovertebral angle. Right hand place parallel to the lateral edge of the right rectus muscle, with your fingertips just reaching the costal arch.

During palpation of the left kidney, place your right hand in the lumbar region just below and parallel to the 12th rib. Left hand place parallel to the lateral edge of the left rectus muscle.

During relaxation of abdominal muscles in expiration press by both hand as close to each other as possible. Ask the patient to inspirate deeply by "the abdomen". The kidney descends, try to capture it between your two hands and slide over the surface.

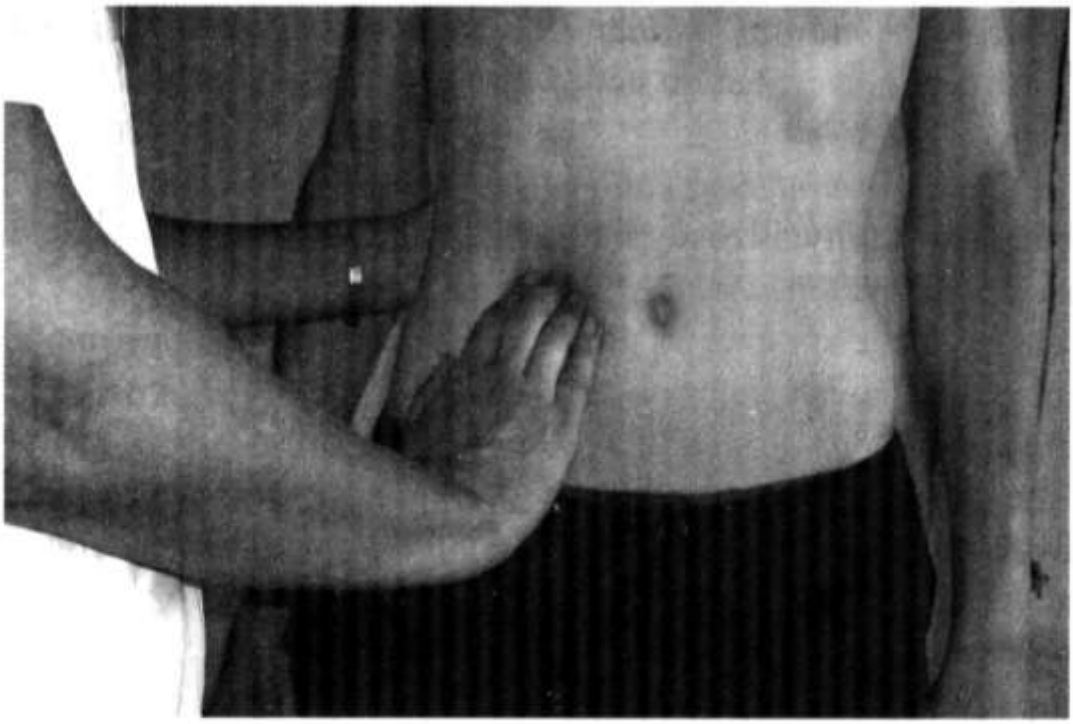


Fig. 6.6. Palpation of the right kidney in standing posture.

Palpation is used to diagnose ptosis of the kidneys. Three degrees of the nephroptosis are differentiated (Fig. 6.7):

I degree – ***palpable kidney*** (*Ren palpabilis*): the lower pole of dense elastic consistency, rounded, and tenderness is palpated;

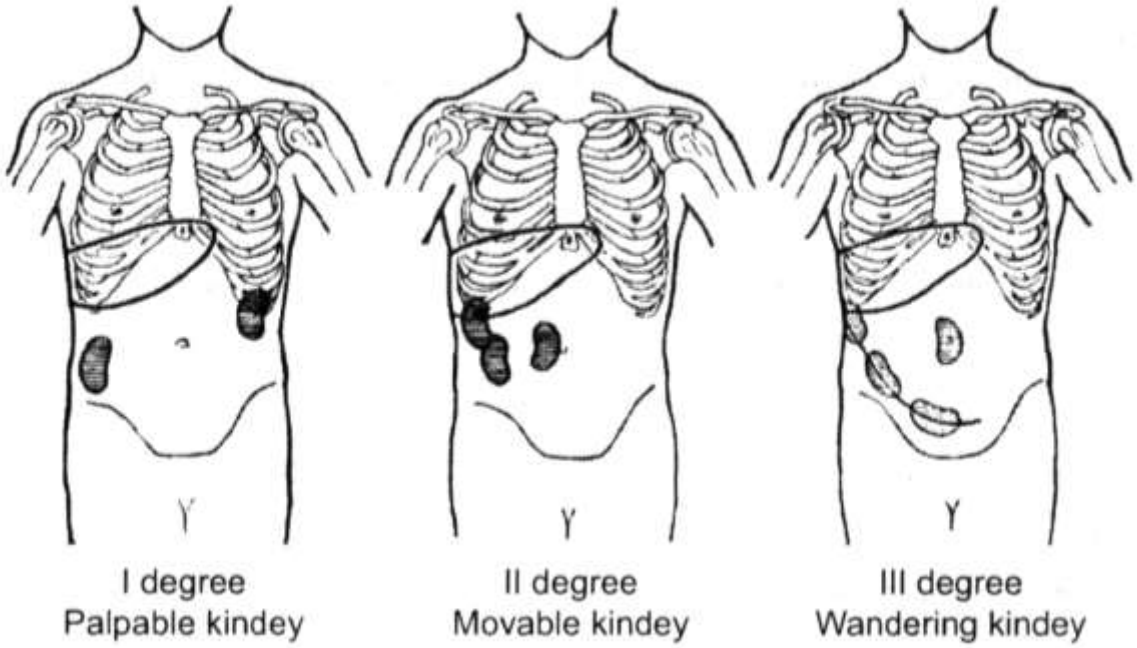


Fig. 6.7. Nep hroptosis degrees.

II degree – ***movable kidney*** (*Ren mobilis*): the entire kidney is palpated, it freely moves, but do not displaced on the opposite side of the abdominal cavity;

III degree – ***wandering kidney*** (*Ren migrans*) is palpated at any part of the abdominal cavity, is easily mobile, may displaces on the opposite side, and has normal parameters.

Enlarged due to tumor kidney is dense, tubercular, slightly mobile, and Guyon's symptom is positive.

Palpation of the urine bladder

The patient should be in the lying posture, his legs are stretched or slightly flexed in the knee, and the prelum is relaxed. Sit by the right side of the patient. Place your right hand on *linea mediana anterior* just above upper border of the urine bladder determined by percussion. By slightly flexed fingertips press deeply and slide downward to reach urine bladder.

The urine bladder can be palpated over pubic bone as an elastic fluctuating formation if it contains much urine, especially in individuals with thin abdominal wall. The upper border of significantly distended bladder can be palpated even at the umbilicus level.

In palpation you can detect following *tenderness points*:

1. ***Costovertebral point*** – the site of kidney projection, is placed in the angle between the 12th rib and the longissimus thoracis muscles;
2. ***Superior ureter point*** is placed at the lateral edge of the rectus abdominal muscle at the umbilicus level;
3. ***Inferior ureter point*** is placed at the intersection of *linea biiliaca* with the vertical line passing the *spina ossis pubis* (pubic tubercle).

Palpation of these points is of certain diagnostic importance. Normally they are painless. In pathology: in inflammatory processes of pelvis, ureters, in nephrolithiasis deep palpation of these points causes acute pain.

Percussion

Percussion of the kidneys has no diagnostic significance, because dullness over them can only be determined in pronounced enlargement of these organs. Normally, it is impossible to percuss kidneys as the intestine that gives tympany covers them.

Percussion is used in a form of tapping. Pasternatsky proposed this method mainly for establishing nephrolithiasis.

Pasternatsky's symptom

Place your left hand in the costovertebral angle and moderate strike it with the ulnar surface of the right hand palm (Fig. 6.8).

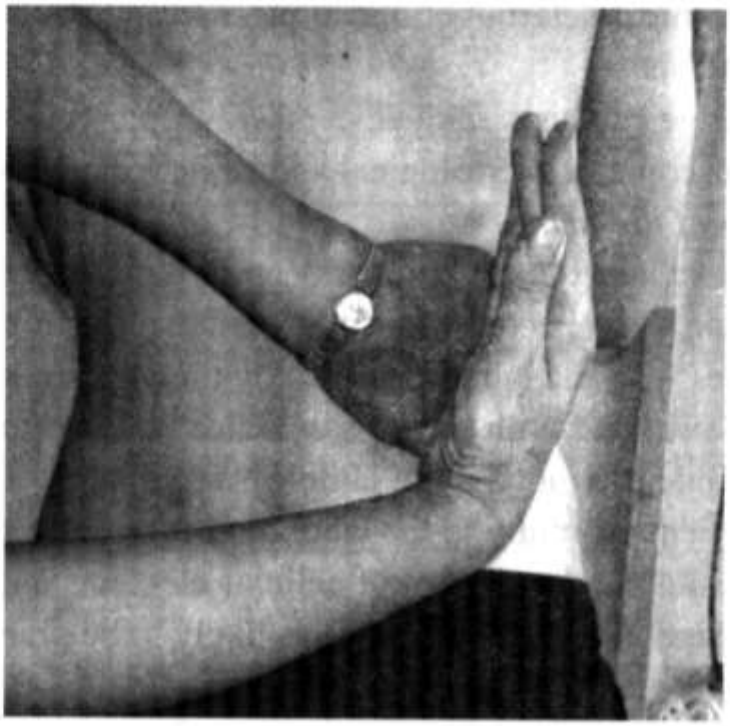


Fig. 6.8. Pasternatsky's sign.

Pasternatsky's sign is considered to be positive, if the patient feels pain in tapping. In a normal healthy person the kidneys are painless in tapping – negative Pasternatsky's sign. Pain with percussion in the costovertebral angle suggests nephrolithiasis, paranephritis, and inflammation of the pelvis. It should be remembered that this symptom might be positive in myositis and radiculitis that decrease its diagnostic value.

Percussion of the urine bladder

Place the pleximeter-finger on *l. mediana anterior* and move it from the umbilicus level downward parallel to the pubic bone. The site of tympany transition to dullness is the upper border of the urine bladder. Normally, the upper border of the bladder is under pubic bone. In accumulation of significant amount of urine in the bladder the percussion sound is dull in suprapubic region.

Laboratory Methods

The investigation of the patient with suspected renal disease usually requires assessment of both structure and function of the kidneys. Structural evaluation includes imaging studying and microscopic examination of tissue samples acquired by biopsy. Function is assessed by examination of urine and biochemical determinations of plasma samples.

Urine analysis

Clinical urine analysis includes: macroscopic (physical properties), chemical, microscopic, bacteriological and bacterioscopic studies.

Collection of the urine

Urine sample is taken after night sleep in the clear and dry container. Urine sample can be collected from males after retraction of the foreskin, and from females with the labia separated by their fingers. Ideally the genitalia should be swabbed with sterile saline but this is often impracticable. Antiseptics should be avoided if the sample is required for culture. The urine sample must be sent promptly to the laboratory to avoid growth of contaminant organisms and the dissolution of cellular elements and casts. It should be cooled in refrigerator at 4 °C if delay is likely to be greater than 2 h.

Macroscopic study

Macroscopic study includes assessment of physical properties of the urine: amount, color, cloudiness, smell, and specific gravity.

Physical properties of the urine

Amount of the urine. In healthy adult the normal amount of excreted urine is between 1000 ml and 2000 ml in 24 h (diurnal diuresis).

Polyuria is defined as a production by an adult of more than 2000 ml of urine/24h.

Olyguria is defined as a production by an adult of less than 500 ml of urine/24h.

Color of the urine depends on the presence of physiologic pigments (urochromes, urobilinoids, uroerythrin, etc) and on its concentration. The color of the normal urine varies from straw yellow to orange-yellow. Different pathological conditions of the urinary organs can cause peculiar changes of the urine color (Tab. 6.8).

Tab. 6.8. ***Clinical significance of urine color changes.***

Color	Pathological condition	Cause
Dark yellow	Congestive kidney, edema, burns, diarrhea, vomiting	High concentration of urochrome
Pale, water-like	Diabetes mellitus, diabetes insipidus	Low concentration of urochrome
Dark	Hemolytic anemia	Urobilinogenuria
Dark, almost black	Acute hemolytic kidney	Hemoglobinuria
Red	Renal colic, renal infarction	Hematuria (unaltered blood)
Appearance of "meat wastes"	Acute glomerulonephritis	Hematuria (altered blood)
Greenish-brown (beer-like)	Parenchymatous jaundice	Bilirubinuria, urobilinuria
Greenish-yellow	Obstructive jaundice	Bilirubinuria
Whitish	Fatty degeneration and decomposition of the renal tissue	Lipuria
Milky	Renal lymphostasis	Hyluria

Cloudiness of the urine. Normal, freshly excreted urine is clear. Cloudiness of the urine can be cause by the presence of salts, cellular elements (leucocytes, erythrocytes, epithelium cells), bacteria, mucus, and fats.

Smell of the urine. Normally, the urine has not strong specific smell. In bacterial decomposition on air or in urinary ducts (severe cystitis, degradation of malignant tumor) urine smells of ammonia. Peculiar "fruity" or "apple" odor of the urine is characteristic of diabetic coma or diabetes mellitus in decompensation stage. Such specific odor of the urine is a result of ketone bodies presence.

Specific gravity of the urine is proportional to concentration of dissolved in it substances: urea, uric acid, various salts, and depends not only on amount but mainly on their molecular weight.

Specific gravity is measured by urometer, normally it varies from 1.015 to 1.025. In health there is diurnal variation of the specific gravity; in morning, the most concentrated portion of the urine, it can be to 1.020–1.026.

Assessment of the specific gravity of the urine is of great diagnostic significance, because these parameter gives information about concentrating ability of the kidneys. The specific gravity can also be depends on the volume of urine excreted (Tab. 6.9).

Tab. 6.9. ***Clinical significance of specific gravity.***

Specific gravity	Extrarenal causes		Renal causes
	Physiological	**Pathological**	
Low	Polyuria	Diuretics taking Alimentary dystrophy Diabetes insipidus	Renal failure
High	Oliguria	Fluid accumulation in cavities and tissues (edema, ascitis, hydrothorax, etc) Profuse vomiting, diarrhea Diabetes mellitus	Renal glucosuria Renal amyloidosis with high proteinuria

Zimnitsky's test characterize condition of renal concentrating and excretory ability. In order to correct measure urinary concentrating ability, the patient must avoid taking much fluid.

Urine samples are collected each 3 hours in separate container with designation of time – 8 portions during 24 hours. Volume and specific gravity of the urine is measured in each portion.

The advantages of this method are:

- Possibility to measure diurnal diuresis and to detect presence of polyuria or oliguria;

- Possibility to measure separately daily and nightly diuresis and to detect presence of nycturia;
- Possibility to determine diurnal variation of the specific gravity and its maximal value.

Normally, diurnal diuresis is 1000–2000 ml, amount of urine in each portion can vary from 50 to 250 ml, daily diuresis exceeds nocturnal, and specific gravity vary from 1.010 to 1.025. If the maximal mean of specific gravity in Zimnitsky's test exceeds 1.020, renal concentrating ability is considered to be normal.

Low specific gravity in all portions is typical to renal failure.

Isosthenuria is defined as condition when osmotic concentration of urine is equal to osmotic concentration of blood plasma. Maximal osmotic concentration of urine in isosthenuria is 270–330 mmol/l, and maximal specific gravity – 1.010–1.012.

Hyposthenuria is defined as condition when maximal osmotic concentration of urine is less than osmotic concentration of blood plasma. Maximal osmotic concentration of urine in hyposthenuria is 200–250 mmol/l, and specific gravity of urine – 1.005–1.008.

Extrarenal causes of urine specific gravity changes

In diabetes mellitus, polyuria and high specific gravity of the urine (to 1.026–1.050) due to glucosuria is determined.

Diabetes insipidus and pituitary insufficiency are characterized by polyuria and low specific gravity of the urine.

Renal causes of urine specific gravity changes

In acute glomerulonephritis, nephrotic syndrome, and in congestive kidney in heart failure osmotic concentration of urine is elevated to 1200 mmol/l, specific gravity of the urine – to 1.031–1.035, that accompanied by oliguria. Hyposthenuria in normal diurnal diuresis and nicturia observe in patients with chronic glomerulonephritis, chronic pyelonephritis, and nephrosclerosis. Isosthenuria suggests complete absence of renal concentrating ability. Long standing excretion of urine with low specific gravity, monotonous means in combination with oliguria are the signs of severe chronic renal failure with unfavorable prognosis.

Chemical study

Chemical study includes assessment of reaction of the urine (urine pH), protein, glucose, ketone bodies, and bile pigments.

Reaction of the urine – urine pH can be determined calorimetrically (litmus paper and other indicators) and electrometrically. The urine reaction may vary from pH 5.0 to 7.0 – neutral or feebly acid reaction. Urine pH can be changed in both physiological and pathological conditions (Tab. 6.10).

Tab. 6.10. ***Clinical significance of urine pH changes.***

Urine reaction		
Acid	**Neutral Feebly acid**	**Alkaline**
Physiological conditions: much meat food intake *Pathological conditions:* diabetes mellitus, severe renal failure, acute nephritis, congestive kidney, tuberculosis of the kidneys, acidosis, hypokaliemic alkalosis	Norm	*Physiological conditions:* vegetable diet, at the height of digestion, ample alkaline fluid intake *Pathological conditions:* Vomiting, diarrhea, chronic infections of the urinary tracts

Protein. The normal amount of protein excreted in the urine per 24 hours is 25–75 mg that cannot be detected by routine tests. More than half of this amount consists of small molecular weight proteins or protein fragments, although albumin is the largest single component.

Proteinuria is the appearance of protein in the urine in concentration determinable by qualitative methods.

The protein content of the urine of normal individuals can rise to about 150 mg/l when the urine is concentrated.

Selective and non-selective proteinuria is distinguished. *Selective proteinuria* is characterized by the presence in the urine of low molecular weight proteins – albumin, ceruloplasmin, and transferrin. In non-selective proteinuria high molecular weight proteins – α_2-macroglobulib, β_2-lipoprotein, and γ-globulin are detected. Moreover, Bence-Jones proteins – low molecular weight proteins, can be revealed in the urine. In some pathological conditions, hemoglobin, hemosiderin, myoglobin, and Tamm-Harsfall proteins are present in the urine.

Depend on protein- amount in the urine, *microalbuminuria* – 30–300 mg/24h, and *proteinuria* (*macroalbuminuria*) – more than 300 mg/24h are distinguished.

Proteinuria can be functional and organic. *Functional proteinuria* observed in subjects without renal diseases, has transitory character, does not exceeds 1 g/24h, and are not accompanied by the other urine abnormalities. Postural (orthostatic), effort, and cold proteinuria are differentiated. Healthy adults are found to have proteinuria when up and about, but not after a period of horizontal rest. Standing position can induce significant proteinuria in a substantial proportion of people who do not otherwise show it. Proteinuria can also be observed in subjects without renal diseases after severe exercise, in fever, or on exposure to extremes of cold or heat. These findings do not imply the presence of renal disease and do not require further investigation.

Organic proteinuria can came about in three ways:

1. The glomerular filter becomes more permeable to proteins of large molecular size, as well as permitting those of small molecular weight to pass – 'glomerular' proteinuria. This is by far the commonest cause of proteinuria in clinical practice.
2. There is a marked rise in the plasma concentration of protein in circulation, so that amount filtered exceeds the reabsorptive capacity of the proximal tubule – 'overflow' proteinuria.
3. The proximal tubule is damaged so that normally reabsorbed proteins, principally of low molecular weight, pass into the urine – 'tubular' proteinuria (Tab. 6.11).

Tab. 6.11. ***Clinical significance of renal proteinuria.***

Types	Renal		
	Glomerular		**Tubular**
Character	Transitory	Constant	Transitory
Protein content	Albumin γ-globulin lisocin	Albumin Transferrin α_2-macroglobulin γ-globulin	Albumin α_2-macroglobulin β_2-microglobulin
Pathological condition	Acute infectious diseases, enteritis, colitis, trauma, burns, liver diseases, diabetic coma, cerebral stroke	Acute and chronic glomerulonephritis, renal amyloidosis, diabetic nephropathy, pyelonephritis, renal veins thrombosis, rheumatic arthritis, congestive kidney, end-stage renal diseases.	Acute tubular necrosis, interstitial nephritis, genetic tubulopathy, Fankoni syndrome

Extrarenal proteinuria is usually caused by protein admixtures (inflammatory exudates, degradated cells) in the diseases of urinary and sex ducts. Such proteinuria usually does not exceed 1g/l.

Glucose. Excretion of glucose with urine is called *glycosuria*. Glucose is freely filtered by the glomerulus and reabsorbed actively by the proximal tubule. Under normal circumstances, reabsorption is complete but if the blood glucose rises sufficiently, a plasma level is reached (the threshold) at which the transport mechanism is saturated and glucose starts to spill into the urine. Glycosuria could arise in principle in two different ways. First, if the plasma glucose concentration rises above the threshold level (around 10 mmol/l in man) the unreabsorbed glucose will appear in the urine, and this occurs in uncontrolled diabetes mellitus, the commonest clinical cause of glycosuria. Alternatively, if the tubular mechanism for reabsorption is

defective, glucose will appear in the urine even when the plasma glucose is within the normal range. This can occur as a result of an inherited abnormality in the protein mediating glucose transport across the proximal tubular cells, or as a consequence of a disease process interfering with the function of this epithelium, as in cyctinosis or tubular damage by heavy metals and other toxins.

Glycosuria can be physiological and pathological (Tab. 6.12).

Tab. 6.12. ***Clinical significance of glycosuria.***

Physiological	Pathological
Alimentary (carbohydrates diet) Following emotional stress Taking of some medicines: caffeine, corticosteroids, epinephrine	Renal diabetes as a primary disease (blood glucose is normal) Secondary renal glycosuria in chronic nephritis and nephrotic syndrome Diabetes mellitus Thyrotoxicosis Pituitary insufficiency (Itsenko-Cushing disease), Liver cirrhosis

Physiological glycosuria is usually transitory, and pathologic – constant.

Ketone bodies (acetone, acetoacetic and β_2-oxybutyric acid) are normally absent in the urine. *Ketonuria* is defined as a presence of ketone bodies in the urine. They usually occur in diabetes mellitus, carbohydrate deficit: fasting, grave toxicities, longstanding intestinal disorders, dysentery, and in postoperative period. Ketonuria is important laboratory sign of decompensation of diabetes mellitus with transformation to diabetic coma.

Bilirubin. Normal urine contains minimal quantity of the bilirubin. Increased excretion of bilirubin is pathological condition and is called – *bilirubinuria.* Bilirubinuria occurs in increased blood level of bound bilirubin more than 0.01–0.02 g/l ("renal threshold of bilirubin") in parenchymatous jaundice (acute virus, toxic, toxico-allergic hepatitis, liver cirrhosis), subhepatic jaundice (altered permeability of bile ducts due to inflammation, obstruction by stones, by tumor, or by scars).

Urobilinoids: urobilin (urobilinogens, urobilins) and stercobilin (stercobilinogens, stercobilins) are derivates of bilirubin. They are not determined separately. A large quantity of urobilinoids in urine is called *urobilinogenuria*. It occurs mainly in: parenchymatous affection of the liver (hepatitis, cirrhosis), hemolytic processes (hemolytic anemia); and in intestinal diseases (enteritis, constipation, etc).

Microscopic study

Erythrocytes. The urine of healthy person contains single erythrocytes. The presence of erythrocytes in the urine is called *hematuria*. Determination of erythrocytes in microscope vision area (more than 1000 cells in 1 ml) is defined as *microhematuria*; the color of the urine is unchanged in such cases. If erythrocytes amount is 2500 cells in 1 ml, the urine is of red color that is defined as *macrohematuria*.

Hematuria can be true (from the kidneys and urinary tract) and false (in man in prostatitis, tuberculosis and tumor of prostate, in woman of genitalia origin).

Erythrocytes in the urine can be altered and unaltered depend on their origin (Tab. 6.13).

Tab. 6.13. *Clinical significance of hematuria.*

Hematuria	
Glomerular origin	**Non glomerular origin**
Altered erythrocytes • Acute nephritis (macrohematuria) • Chronic glomerulonephritis (more pronounced during aggravation) • Renal infarction (macrohematuria) • Hypernephroma (periodic macro- and microhematuria) • Renal tuberculosis (constant microhematuria) • Congestive kidney (congestive microhematuria) **N.B.** In the presence of glomerular hematuria, the urine usually contains much protein – so-called protein-erythrocyte dissociation	*Unaltered erythrocytes* observes more frequently in urinary tract diseases: • Stones in the pelves, urinary bladder, ureters • Acute cystitis • Malignant tumors • Tuberculosis of urinary bladder or pelves • Hypertrophy of prostate

It is important diagnostically to determine location of bleeding source. A *three-glasses test* is used for this purpose. Patient urinates in three containers (Fig. 6.9).

Macrohematuria in the first portion suggests bleeding from urethra (Fig. 6.9.A), in all three portions – from kidneys or ureters (Fig. 6.9.B), and in last portion – from urine bladder (Fig. 6.9.C).

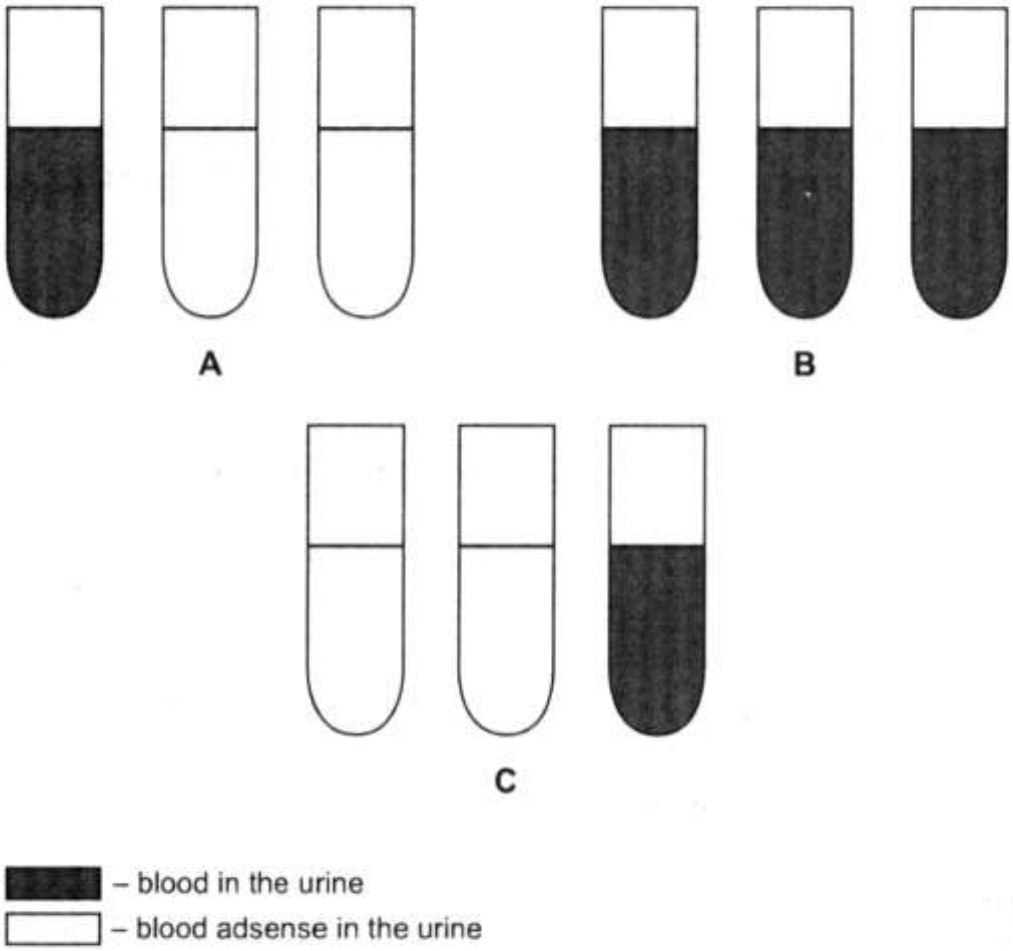


Fig. 6.9. Clinical significance of three-glasses test in hematuria.

Leucocytes are observed mainly in a form of neutrophils, and sometimes eosinophils and lymphocytes are present. Urine of healthy individuals contains small amount of leucocytes (1–2 in vision area). *Leucocyturia* is defined as elevated amount (from 5–6 to 20 cells in vision area) of leucocytes in the urine. *Pyuria* is said to be present when amount of leucocytes increases to 60–100 cells in vision field, and they are seen macroscopically.

Epithelium cells

Tubular (renal) epithelium cells are absent normally in the urine. Their presence indicates acute or chronic affection of the kidneys. They can also be detected in fever, toxicities, and in infectious diseases.

Transitional epithelium cells presence in the urine suggests inflammatory processes in the pelves or bladder.

Squamous epithelium cells originate from genitalia and urethra, and diagnostic their significance is low.

Cylinders (casts). These are cylindrical bodies formed in the lumen of the distal tubule, particularly the collecting tubule. Casts are protein copies of tubules. Appearance of cylinders in urine sediment is called *cylinduria* – the sign of organic renal diseases.

Hyaline casts are occasionally seen in the urine of normal people, particularly when it is concentrated, or after exercise. Hyaline casts appear in the urine during secondary proteinuria: febrile, congestive, orthostatic, toxic, and after administration of loop diuretics. Constant hyaline casts presence suggests proteinuria of renal genesis: glomerulonephritis, pyelonephritis, and nephropathy.

Granular casts occur in much the same situations as hyaline casts and have similar significance. They are found in the urine of normal subjects after exercise. They appear in many types of renal disease but are particularly characteristic of chronic proliferative or membranous glomerulonephritis, diabetic nephropathy, and amyloidosis.

Waxy casts presence in the urine indicates chronic diseases of the kidneys.

Erythrocytes (unaltered) casts are pathognomic of renal bleeding: nephrolithiasis, tuberculosis and tumor of the kidneys; acute process in the kidneys: acute glomerulonephritis.

Erythrocytes (altered) casts are seen in chronic glomerulonephritis.

Leucocytes casts may appear in considerable numbers during an episode of acute pyelonephritis; a few may be found in the urine in chronic pyelonephritis.

Nechiporenko's method allows counting formed elements in 1 ml of urine, normally:

- Leucocytes – to 4000;
- Erythrocytes – to 1000;
- Casts – to 200.

Crystals. Cystine crystals may be found in freshly passed urine but are found more consistently if a concentrated sample is acidified and cooled in a refrigerator, their presence is diagnostic of *cystinuria*. Oxalate crystals are common in urine from normal individuals when it has stood for an hour or two. When present in freshly passed urine, in large numbers or aggregates, they may indicate an increased liability to form oxalate stones, but firm conclusions can only be drawn if the urine is kept at 37 °C until examined on a warm-stage microscope.

Mucus. The normal urine practically contains no mucus. Commonly mucus appears in diseases of the urinary tract: urethritis, prostatitis, cystitis, and in stones presence.

It must be emphasized that although urinalysis and microscopy yield valuable information, it is possible for significant renal disease to be present without anything abnormal being detected in the urine.

Bacterioscopic study

Bacteriuria is defined as presence of bacteria in the urine. In quantity not more than 50 000 in 1 ml they may occur in the urine of healthy person. In the presence of bacteriuria, it is important to determine its degree and microorganism sensitivity to various antibiotics.

Instrumental Methods

Plain radiography of the urinary tract. A plain radiography of the kidneys, ureters, and bladder is an essential preliminary to urography. It is often possible to trace the renal outlines and measure the renal size. The left kidney is normally about 1cm longer than the right. The length should be recorded in centimetres for comparison with subsequent films. The other main function of a plain abdominal film is to detect calcification in the kidneys or radiopaque calculi in the ureters or bladder, which may be obscured in the subsequent pyelogram. Oblique views and films taken in inspiration and expiration may be necessary to confirm that suspected calculi are in kidneys. The film may yield other diagnostic information; gallstones are often detected and renal osteodystrophy or myelomatosis may be recognized in the skeleton.

Excretion urography (synonyms: intravenous pyelography; IVP; IVU). The older hyperosmolar contrast media, such as diatrizoate and me-

trizoate, are now being replaced in many centres by low osmolar, non-ionic compounds such as iopamidol and iohexol. The contrast medium is excreted almost entirely by glomerular filtration. A film at 1min after injection gives the best view of renal outlines, with contrast concentrated in the tubules (the nephrogram). Later images, from 2.5 to 30 min will show excretion of contrast into the collecting system (the pyelogram). If calyceal detail is not adequately visible the pelvis and calyces may be distended by applying compression over the lower ureters with an abdominal belt. A film immediately after release of compression will provide detail of ureteric filling. Pre- and postmicturition films of the bladder will provide information on prostatic indentation, space-occupying lesions, and the presence of bladder diverticula and will assess bladder emptying. However, in many centers ultrasound has replaced the IVU for assessment of prostatic volume, bladder capacity, and residual urine volume. Impaired renal function will cause a delayed pyelogram and extra films will need to be taken as late as 12 to 24 h in this situation or in the presence of severe obstruction.

The IVU is clearly the best choice for anatomical detail and filling defects of the collecting system, but it has been largely replaced by ultrasound and computed tomography (CT) scanning for the investigation of abnormalities of renal anatomy, and by nuclear renal scanning or angiography for the investigation of renovascular hypertension.

Retrograde pyelography/ureterography. This technique is very useful for determining the site of complete obstruction to a ureter and for visualizing the ureter distal to the obstruction. It is particularly useful in cases of poor renal function where the kidneys and ureter show poor opacification on the IVU. A cystoscopy and anesthetic is required and contrast is injected through a ureteric catheter. If an obstruction, such as sloughed papillae or epithelial tumour, is shown, then the catheter may be left in situ as a temporary drain pending surgery. Complications of the procedure include renal colic, temporary ureteric obstruction from mucosal oedema, infection, and intrarenal or extrapelvic extravasation of contrast.

Renal arteriography. The renal arteries may be opacified either by intra-arterial injection or by peripheral intravenous injection of contrast medium. The latter requires larger doses of contrast and the quality of images obtained is often not sufficient to allow accurate evaluation of renal

artery branches and intrarenal vessels. Intra-arterial injection is therefore the preferred method, but is an invasive procedure with a greater risk, including contrast nephrotoxicity, than urography, ultrasound, or nuclear renal scanning. It is performed by retrograde femoral catheterization under local anaesthesia. The narrow catheters now used have reduced the local complication rates substantially, often enabling patients to be discharged from hospital on the same day.

Contrast medium is injected rapidly into the aorta at the level of the renal arteries. Selective catheterization of the renal arteries is often required to evaluate the intrarenal vasculature, particularly in renal tumors and in assessment of living renal transplant donors. Digital techniques have now largely replaced standard angiographic imaging, allowing subtraction of superimposed tissue and contrast enhancement. Digital angiography also allows the use of 50 per cent lower doses of contrast medium, lower flow rates, and smaller catheters.

Current indications for renal arteriography are limited, as refinements of less invasive procedures such as CT scanning and ultrasound may provide appropriate information. The chief use is in the evaluation of renovascular disease. If the renal artery stenosis is amenable to transluminal angioplasty, the dilatation (or renal artery stenting) can be performed at the same time as the arteriogram. Other indications include suspected renal artery occlusion from thrombus, embolus, dissection or trauma, screening for arterial aneurysms in the diagnosis of classical polyarteritis nodosa, and when an intrarenal vascular lesion is suspected, as in persistent hematuria following a renal biopsy. In the latter situation arteriography can confirm an arteriovenous fistula or false aneurysm, and renal embolization via an endovascular catheter at the same time will prevent further bleeding. Renal angiography is always required in the surgical evaluation of the renal vasculature of potential live donors of kidney grafts. CT and magnetic resonance imaging (MRI) scanning have now largely replaced arteriography in the evaluation of renal tumors.

Renal venography. Few indications now remain for this procedure as new imaging techniques such as MRI and CT allow easier detection of renal vein thrombosis. However, an iliocavagram may be necessary to document caval extension of a renal vein thrombosis. Selective catheterization of the

renal veins for renal vein renin levels in renovascular hypertension has now largely been replaced by the more reliable and quicker technique of nuclear renal scanning.

Computed tomography (CT scanning) of the trunk displays the kidneys particularly well in contrast to the surrounding perinephric and peripelvic fat. It can reveal abnormalities of the retroperitoneal and perirenal spaces that cannot be shown with conventional techniques. Contrast media used in CT scanning are the same as those used for intravenous urography. The prime indications for CT scanning of the kidneys are to detect renal mass lesions and suspected renal trauma. Generally, simple renal cysts and polycystic renal disease can be equally well diagnosed with the cheaper technique of ultrasound. However, CT is the method of choice for the diagnosis of renal tumors, since it can confirm the solid nature of the tumor and allow determination of its local extension. Central calcification is very suggestive of a malignancy, as is contrast enhancement due to hypervascularity of the tumor. Extension of renal cell carcinoma has implications for prognosis and surgical intervention. CT is the best technique for documenting tumor thrombus in the renal veins. Lymph node involvement by tumor can be detected if the nodes are greater than about 1 cm in diameter. In renal trauma, CT scanning is clearly the best technique for demonstrating parenchymal damage, subcapsular hematoma, and perirenal urinary collections, as well as assessing damage to other organs such as the liver and spleen.

Finally, CT scanning is useful in the investigation of retroperitoneal fibrosis, which may be idiopathic or secondary to the use of drugs. The appearance of the fibrous plaque, which starts below the level of the aortic bifurcation and extends upwards, often enveloping the ureters, can usually be distinguished from lymphoma or sarcoma involving this region.

Magnetic resonance imaging (MRI) is a digital tomographic imaging system where tissue contrast depends on the manipulation of intrinsic magnetic fields. The technique employs a strong uniform magnetic field combined with transient oscillating magnetic fields to create images without the use of ionizing radiation. MRI offers very superior soft-tissue contrast and the ability to distinguish easily simple renal cysts, complex cysts, and solid renal masses. Like CT scanning, it is particularly useful in detecting tumor extension into veins. MRI angiography of the renal circulation is yet to be

assessed but may have a future role in renovascular disease. Presently, MRI is mainly indicated to clarify equivocal CT findings in renal tumors.

Ultrasonography. This is now the initial investigation for the majority of patients in renal failure and has displaced conventional radiology as the first structural investigation of the urinary tract. High-resolution real-time (moving picture) scans are standard equipment.

Tissue density is the major determinant of acoustic impedance and when sound waves are passed into tissues of widely different acoustic impedances most of the waves are reflected, for example tissue/gas 99 per cent echo, tissue/bone 70 per cent echo. Fat is highly reflective and thick subcutaneous tissues lengthen the distance from the probe to the kidney, making ultrasonography difficult and occasionally impossible in obese patients.

Ultrasound findings are independent of renal function. They can therefore be used to study patients with renal failure, measuring with considerable accuracy the shape, depth from the surface, and internal architecture of the kidney and upper urinary tract. Ultrasound generally detects the dilated calyces, pelvis, and ureter of the obstructed kidney, although in the presence of very recent obstruction the calyceal system may not have dilated sufficiently to make a firm diagnosis on ultrasound, and other investigations such as retrograde ureterography may be necessary. In unexplained renal failure, ultrasound is very useful to assess renal size and cortical thickness, with the presence of small kidneys suggesting chronic renal disease and enabling the renal physician to make appropriate decisions about whether or not renal biopsy is indicated.

Ultrasound is often helpful in identifying the cause of abnormalities detected on the IVU. It may show whether an enlarged kidney is the site of hydronephrosis, an infiltrative process, or a space-occupying lesion. It is particularly useful in deciding whether a localized swelling detected on IVU is cystic (and probably benign) or solid (and probably malignant). However, solid lesions may undergo some cystic change and therefore if a cystic lesion is not absolutely classical (unilocular, uniformly thin walls) then further investigation is indicated. This may be by CT scanning, MRI, invasive cyst puncture, or biopsy.

Ultrasonography has replaced excretion urography as the screening test for polycystic disease; the two techniques are comparable in accuracy

but ultrasonography is faster, cheaper, and devoid of the risk of contrast injection and irradiation. However, cysts and tumors below 5 mm in diameter are not detected reliably by either ultrasound or excretion urography so polycystic disease cannot be excluded with certainty below the age of about 30 years.

Perinephric lesions are readily displayed by ultrasound; it is used to detect extravasation of blood after trauma or renal biopsy, although CT scanning may provide more accurate information in this regard. Ultrasonography is the first test for radiolucent calculi; it is also used for detecting radioopaque calculi, but straight radiography remains the first investigation.

Finally, ultrasound is very useful in the assessment of complications of renal transplantation, particularly the surgical complications of extrarenal collections of blood, pus, and lymph, and in the identification of an obstructed transplant kidney. An ultrasound-guided percutaneous nephrostomy can then allow temporary decompression of the obstruction. Doppler ultrasound may have a limited role in the diagnosis of vascular rejection of the transplanted kidney, but at this stage cannot reliably distinguish acute tubular necrosis and cyclosporin nephrotoxicity from acute rejection.

Nuclear renal imaging. The value of radiolabelled tracers in the investigation of renal disease lies in the ability to obtain important information about organ function as opposed to the predominantly structural information obtained from the previously described imaging procedures. In particular, nuclear imaging of the kidneys provides the only non-invasive quantitative assessment of individual kidney function. Radionuclides (such as ^{123}I, $^{99}Tc^{m}$) are linked to compounds that depend on either glomerular filtration alone, tubular excretion, or a combination of both for excretion from the body. These compounds can therefore provide quantitative information on these functions of the kidney, in addition to dynamic images.

Chapter 7. EXAMINATION OF THE BLOOD

CLINICAL BLOOD ANALYSIS

Determination of the number of erythrocytes, leukocytes and platelet in the blood has long been fundamental procedure in hematology. Manual methods provide satisfactory measurement of the leukocytes and platelet counts, but the erythrocytes count is quite inaccurate when performed manually. Electronic automatically cell counting methods by electrical impedance and optical devices are now widely used and permit accurate enumeration of all there formed elements.

The International Committee for Standardization in Hematology has recommended that the following units be used (SI units): white cell count, "number × 10^9/l", red cell count, "number × 10^{12}/l" and platelet as "number × 10^9/l".

Erythrocytes

Normal values of erythrocytes count	
Men	5.5±1.0 × 10^{12}/l
Women	4.8±1.0 × 10^{12}/l
Infants, full-term, cord blood	5.0±1.0 × 10^{12}/l
Children, 1 year	4.4±0.8 × 10^{12}/l
Children, 10–12 years	4.7±0.7 × 10^{12}/l

Clinical significance of erythrocytes changes

The changes of erythrocytes are corresponded to the quantitative (increasing or decreasing of erythrocytes number) and qualitative (morphology cells).

Increasing of erythrocytes number is classified as **erythrocytosis**, decreasing – as **erythrocytopenia**.

Erythrocytosis

Physiological condition: newborn, excessive perspiration.

Pathological condition:

Primary: Polycythemia Vera

Secondary: ***1. Associated with hypoxia***

- cardiovascular disease, usually congenital resulting in significant venous admixture;
- pulmonary disease resulting in impaired gas perfusion, perfusion of poorly lung, pulmonary arteriovenous fistulas;
- high attitude residence;
- hypoventilation associated with obesity (Pickwickian syndrome);
- hemoglobin variant with increased affinity for oxygen;
- heavy smoking;
- methemoglobinemia (rarely).

2. Due to inappropriate erythropoietin increase in

- benign and malignant tumors of kidney, liver, central nervous system, uterus, ovary;
- renal disease hydronephrosis, Vascular impairment, cysts.

3. Associated with Adrenocortical steroids or Androgens

- adrenal hypercorticism;
- virilizing tumors;
- androgens used therapeutically.

4. Associated with chronic chemical exposure

- nitrites, sulfonamides, other substances producing methemoglobin and sulphaemoglobin;
- cobalt, shellac components, various alcohols.

5. Relative

- "stress" or "spurious" polycythemia;
- dehydration: water deprivation, vomiting;
- plasma loss: burns, enteropathy.

Erythrocytopenia:

1. Depression of bone narrow: leukemia, aplastic anemia, metastases in bone marrow.
2. Hemorrhage.

3. Hemolysis of erythrocytes.
4. Deficiency of iron.
5. Deficiency of vitamins B_{12}.
6. Hemoglobinopathy.

Hemoglobin

The International Committal for Standardization in Hematology has recommended that the following units be used (SI units): hemoglobin, "g/dl" (dl-deciliters).

Normal values of hemoglobin	
Men	15.5±2.5 g/dl
Women	14.0±2.5 g/dl
Infants, full-term, cord blood	16.5±3.0 g/dl
Children, 1 year	12.0±1.0 g/dl
Children, 10–12 years	13.0±1.5 g/dl

Clinical significance of hemoglobin changes

Decreased concentration of the hemoglobin in the blood (**oligochromemia**) observes in anemia of different etiology (as a result of hemorrhage, deficiency of iron, vitamin B_{12}, and folate, increase hemolysis of erythrocytes, etc.).

Increased concentration of hemoglobin in the blood (**hyperchromemia**) occurs in erythremia, cardio-pulmonary failure, some congenital heart defects, and usually accompanied by increased erythrocytes number.

In blood clotting relative increasing of hemoglobin concentration can be found.

Color index

Once the quantity of erythrocytes and hemoglobin in a given blood specimen is known, it is possible to calculate the hemoglobin content of each erythrocyte. There are many methods by which hemoglobin saturation can be determined. One of them is the calculation of the *color index*. This is a conventional value derived from the ratio of hemoglobin to the number

of erythrocytes. This value is found by dividing a triplet quantity of hemoglobin in grams by the first three figures expressing the quantity of erythrocytes. Normally this value is 0.85–1.1 (**normochromia**).

Clinical significance of color index changes

Hyperchromia, that is increased hemoglobin content in separate erythrocyte resulted in color index more than 1.1, depend only on increased volume of erythrocytes (*macrocytosis*), but not on increased saturation of hemoglobin.

Hyperchromia (color index 1.2–1.5) is characteristic of vitamin B_{12} deficiency anemia. Hyperchromia can observe in some chronic hemolytic and myelotoxic anemia.

Hypochromia – color index decreasing (less than 0.8) can be result of decreased erythrocytes volume (*microcytosis*) or insufficient hemoglobin saturation of normal volume erythrocytes.

Hypochromia can be caused by lack of iron, defective globin synthesis, defective porphyrin synthesis, and is found in the patients with iron deficiency anemia, anemia of chronic diseases, thalassemia, hemoglobinopathies (C, E), and sideroblastic anemia.

Normochromia, usually observed in healthy individuals, can also be found in some anemia (acute posthemorrhagic, acute hemolytic, hypo- and aplastic).

Leukocytes

Normal values of leucocytes are $4.0–9.0 \times 10^9$/l

Clinical significance of leukocytes changes

The changes of leukocytes are corresponded to the quantitative (increasing or decreasing of leukocytes number) and qualitative (morphology cells).

Increasing of leukocytes number is classified as **leukocytosis**, decreasing – as **leukocytopenia**.

Leukocytosis

Physiological condition: new born, digestive process (after taking food, rich of protein), physical exercise, pregnancy, and taking some medicine (hormones).

Pathological condition: leukemia, infectious process, inflammatory, purulent process, myocardial infarction, malignant tumor, uremia, loss of blood, shock.

Leukopenia

Physiological condition: deep sleep, starvation.

Pathological condition; bacterial, virus infectious, autoimmune disease, hypoaplastic condition (administration X-ray, chemical substances, radiation), agranulocytosis (after administration some medicine – for treatment malignant process).

The percentage of separate forms of leukocyte in peripheral smear is considered as **leukocyte formula** – it is the percentage of separate forms of blood leukocytes.

The normal value of leukocyte formula	
Leukogramma:	
Bands or stabs neutrophils	0.04–0.3 × 10^9/l (1–6 %)
Polymorphonuclear neutrophils	2.0–5.5 × 10^9/l (47–72 %)
Basophils	0–0.065 × 10^9/l (0–1 %)
Eosinophils	0.02–0.3 × 10^9/l (0.5–5 %)
Lymphocytes	1.2–3.0 × 10^9/l (19–37 %)
Monocytes	0.09–0.60 × 10^9/l (3–11 %)

Clinical significance of leukocyte formula changes

The leukocyte formula is counted in stained smears.

The changes of different forms of leukocytes have clinical significance. Increasing of neutrophils number is classified as neutrophilia, decreasing – as neutropenia.

Neutrophilia

Infections:

- pyogenic bacterial: staphylococcal, streptococcal, pneumococcal, meningococcal, gonococcal;
- non pyogenic: acute rheumatic fever, diphtheria, scarlet fever, acute poliomyelitis, cholera, herpes zoster, mycobacterial, fungal, spirochaetal, parasitic.

Metabolic disorders, due to varied causes leading to liver insufficiency, uremia, diabetes, acidosis, gout, eclampsia.

Neoplasms:

- myeloproliferative disorder: myeloid leukemia, lymphomas, polycythemia vera, myelosclerosis;
- other malignancies: carcinomas (metastatic or otherwise), sarcomas

Conditions causing cell necrosis or destruction:

- acute hemolysis;
- infarctions;
- drug intoxication: nephrotoxins, hepatotoxins;
- various drugs/chemicals implicated are phenacetin, digitalis, quinine, adrenaline, organic arsenicals, lead, mercury, carbon monoxide.

Trauma and hemorrhage:

- hemorrhage: acute hemorrhage (especially internal hemorrhage);
- trauma: operative, fractures, crush injuries, burns.

Cardiac disorders:

- paroxysmal tachycardia.

Collagen disease:

- polyarteritis nodosa;
- acute phase of rheumatoid arthritis;
- dermatomyositis.

It is necessary to account the degree of lobation of the neutrophile nuclei.

After the degree of nuclear shift in the leukocytic formula the following kinds of neutrophilic leukocytosis are distinguished:

1) with any nuclear shift – an increased number of mature segmented neutrophils on the background of leukocytosis as a whole;
2) with a hyporegenerative nuclear shift to the left – an increased number of band forms of neutrophils (above 5 %), it is characteristic of a slight course of several infections and inflammation;
3) with the regenerative nuclear shift to the left which indicates the reactive activation of granulocytopoiesis. On the background of neutrophilia and increased number of band forms metamyelocytes and sometimes individual myelocytes occur. It is characteristic of the purulent septic processes;
4) with the hyperregenerative nuclear shift to the left which reflects the excessive hyperplasia of leukopoietic tissue with disturbance of maturation

of the cells and expressed rejuvenation of the blood. The quantity of band neutrophils and metamyelocytes is strongly increased, the younger forms occur (myelocytes and even a few promyelocytes and myeloblasts). The total number of leukocytes can be increased, nonchanged or even decreased because of exhausted myelopoiesis. The absence of eosinophils (aneosinophilia) is often observed. This shift is met in adversely proceeding infectious and purulent septic processes;

5) with the degenerative nuclear shift which testifies to an inhibition of the bone marrow. On the background of leukopenia the number of band neutrophils is increased; there are a lot of destructed segmented forms, metamyelocytes are absent. It is characteristic of severe infections, endogenic intoxications, etc. In hyperproduction of pathologically changed leukocytes and disturbance of their maturation the regenerative-degenerative shift is observed. Thus leukocytosis is marked, and the number of band neutrophils, metamyelocytes and myelocytes with the attributes of degeneration is increased;
6) with the nuclear shift to the right which is characterized by occurrence of hypersegmented (5 segments) neutrophils and testifies to an inhibition of granulocytopoiesis. It is found out in radiation sickness, pernitious anemia, however can be observed in a healthy man.

In various diseases the change in the total number of leukocytes is accompanied by the occurrence in the blood of the pathologic leukocytes, which are classified into regenerative (they are found out in norm only in the bone marrow) and degenerative (destructed) forms. The attributes of degeneration are: toxicogenic granularity, vacuolisation, hypochromatosis, anisocytosis, fragmentation, picnosis and rexis of the nucleus, hypersegmentation, etc.

Leukemoid reactions – the pathologic reactions of the blood system with high leukocytosis and occurrence of immature leukocytes in the blood, which are similar to those in leukemias, but are temporary and convertible. They occur in infections, inflammatory diseases, intoxications, lymphogranulomatosis, parasitic, allergic diseases, collagenoses, infections mononucleosis, etc.

Neutropenia.

The kinds of leukopenia (neutropenia):

- distributive neutropenia is observed in shock, neuroses, inflammatory diseases, malaria. The ratio between the circulating and marginal pools of leukocytes is changed as a result of congestion of leukocytes in dilated cappilaries of the blood depots (lung, liver, intestine). This leukopenia is temporary and is usually replaced by leukocytosis;
- neutropenia owing to intensive destruction of neutrophils. It is observed: a) under influence of antibodies leukoagglutinins, which are formed in blood transfusions, under influence of some drugs, which are allergens-haptens (sulpha drugs, amidopyrin, etc.); b) in diseases accompanied by increased number of the circulating inmmune complexes (autoimmune diseases, tumors, leukemias); c) in action of toxins (the toxic forms of typhoid fever, influenza, dysentery, the extensive inflammatory processes, the poisoning with benzene, arsenic, sulphonamides); d) in the enlarged spleen (collagenoses, liver cirrhosis, hemolytic anemia, etc.);
- neutropenia owing to disturbed or inhibited leukopoiesis. It is observed in action of various toxic substances and drugs, ionising radiation, in replacement of the bone marrow by leukemic or tumor tissue, in protein and vitamin starvation, etc.

Sometimes leukopenia is manifested in the form of agranulocytosis which is characterized by a strong decrease or even absence of neutrophils and other granulocytes in the blood, when the number of granulocytes is below $0.75 \times 10^9/l$ or the total number of leukocytes is below $1.0 \times 10^9/l$.

Eosinophilia

Allergic slates: asthma, fever, exfoliative dermatitis, erythema multiforme, urticaria, food sensitivity, angioneurotic edema, serum sickness, drug allergy.

Parasitic disease: intestinal forms, tissue forms.

Skin disorders: pemphigus, dermatitis herpetiformis, psoriasis, scabies, prurigo.

Drug administration: liver extracts, penicillin, streptomycin, chlorpromazine.

Neoplasms: myeloproliferative (eosinophilic leukemia, chronic myeloid leukemia, polycythemia), others (Hodgkin's disease, multiple myeloma, metastatic and necrotic, occult tumor).

Miscellaneous: familial eosinophilia, eosinophilic syndrome, eosinophilic granulomatosis (visceral larva migrans), scarlet fever, polyarteritis nodosa, tropical eosinophilia, pernicious anemia, post splenectomy, idiopathic neutropenia, post transfusion mononucleosis.

Eosinopenia

Drug/hormone therapy: adrenocortical steroids, adrenaline, ephedrine, insulin.

Response to stress: acute infections, traumatic shock, surgical operations, severe exercise, burns, acute emotional stress, exposure to cold.

Endocrine diseases: Cushing's disease, acromegaly.

Miscellaneous: aplastic anemia.

Basophilia: chronic myeloid leukemia, myelosclerosis, polycythemia vera, hypersensitivity states, myxoedema, iron deficiency anemia (some cases), hemolytic and toxic anemia of long standing, pre-leukemia (some cases).

Basophilopenia:

- neutrophil leukocytosis or leukemoid reaction associated with infection, neoplasma, tissue necrosis, acute anemia;
- allergic conditions;
- hyperthyroidism;
- myocardial infarction;
- Cushing's syndrome;
- following prolonged corticosteroid therapy.

Monocytosis:

Infections:

- bacterial: brucellosis, sub-acute bacterial endocarditis, tuberculosis, typhoid fever, recovery stage of an acute infection;
- rickettsial: rocky mountain spotted fever, typhus;
- protozoan: malaria, kala-azar, trypanosomiasis, oriental sore;
- viral: infections mononucleosis.

Neoplasms: monocytic leukemia, carcinomatosis, Hodgkin's and other lymphomas, myeloproliferative disorders, multiple myeloma.

Collagen diseases; rheumatoid arthritis, SLE.

Miscellaneous: chronic ulcerative colitis, regional enteritis, sarcoidosis, lipid storage diseases, hemolytic anemia, hypochromic anemia, recovery from agranulocylosis.

Lymphocytosis:

- acute infections: infectious mononucleosis, pertussis lymphocytosis, mumps, chickenpox, rubella, infective hepatitis, convalescent stage of many acute infections, toxoplasmosis, influenza;
- chronic infections; brucellosis, tuberculosis, syphilis (secondary);
- endocrine disorders: thyrotoxicosis, adrenal cortical insufficiency, hypopituitarism, myasthenia gravis;
- neoplasms: non Hodgkin's lymphomas, chronic lymphatic leukemia, lymphosarcoma, multiple myeloma.

Lymphopenia:

- severe pancytopenia;
- congestive heart failure;
- adrenocorticosteroid therapy (transient).

Thrombocytes (platelets)

Their normal number is 180.0–320.0 × 10^9/l (180000–320000 per 1 μl) of blood.

Clinical significance of thrombocytes changes

If the number of thrombocytes decreases significantly (throtnhocytopenia), a tendency to hemorrhages develops. The critical figure at which hemorrhage occurs is believed to be 30 × 10^9/l (30 000 per 1 μl). Thrombocytopenia occurs in affection of the bone marrow by infectious causative agents, some medicinal preparations, ionizing radiation, and in autoimmune processes. Thrombocytosis occurs after hemorrhage, in polycythemia, and malignant tumors.

Thrombocytopenias:

- intensified destruction of thrombocytes (immune — in measles, German measles, action of sulpha drugs, chronic lymphoid leukemia, diffusive diseases of the connective tissue, etc.; in scarlet-fiver, sepsis, increased function of the mononuclear phagocytic system);
- insufficient production of thrombocytes (irradiation, poisoning by the chemical substances, metaplasia of the bone marrow, deficit of vitamin B_{12} and folic acid, hereditary disturbances);
- increased expenditure of thrombocytes (in local and generalized intravascular blood clotting);

Chapter 8

RESPIRATORY SYSTEM

There are such syndromes of the diseases of respiratory system: the syndrome of the pulmonary tissue consolidation, the syndrome of increased airiness of the pulmonary tissue, the syndrome of bronchium obstruction (bronchospastic syndrome), the syndrome of fluid accumulation in pleural cavity (hydrothorax) and the syndrome of air accumulation in pleural cavity (pneumothorax), the syndrome of the cavity in the lungs.

Syndrome of the pulmonary tissue consolidation

The syndrome of the pulmonary tissue consolidation is one of the most widespread syndromes of the respiratory system pathology. It is based on the significant decrease or full disappearing of the pulmonary tissue airiness.

Etiology:

– pulmonary tissue infiltration (pneumonia, tuberculosis);

– thromboembolia of the pulmonary artery (TEPA), lung infarction;

– pulmonary edema due to the left ventricular heart failure;

– adhesion of the pulmonary tissue (compressive atelectasis) due to the external compression by air or fluid (pneumothorax, hydrothorax);

– adhesion of the pulmonary tissue (obstructive atelectasis) due to the termination of air entrance to the lung tissue below bronchium obstruction (tumor, foreign bodies, enlargement intrathoracic lymphatic nodes);

– hypoventilation of the lung tissue through the squeezed or narrowing main bronchus (the middle lobe syndrome);

– replacement of airiness pulmonary tissue by airless connective or tumor tissue (cancer, pulmonary fibrosis).

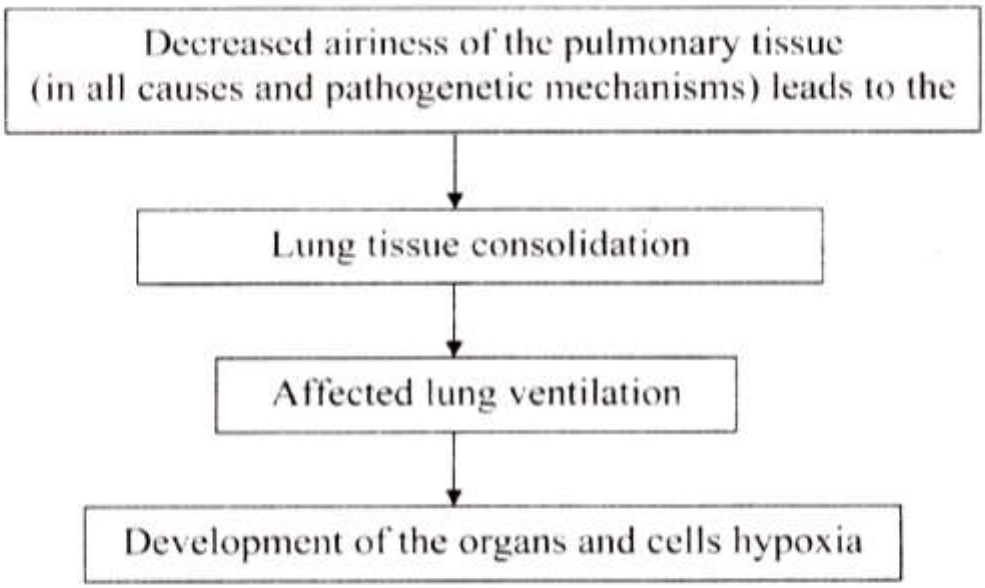
Pathogenesis

Depending on the causes there are the next mechanisms of pulmonary tissue consolidation:

– decreased airiness of the pulmonary tissue due to the consolidation and infiltration of alveolus's walls via inflammatory edema (pneumonia, tuberculosis) or due to the interstitial edema (TEPA, congestive heart failure) via increased hydrostatic pressure in lesser circulation followed with alveoli filled by exudates, transudates or blood;

– decreased airiness of the pulmonary tissue due to the abnormal alveolus distension during inspiration via the pleural affection and compression by accumulative fluid or air (compressive atelectasis) or due to the alveolus adhesion via full absence of ventilation through large bronchus (obstructive atelectasis) or due to the pulmonary tissue hypoventilation via the full or partially large or middle bronchus obturation (the middle lobe syndrome);

– decreased airiness of the pulmonary tissue due to the replacement of pulmonary tissue by progressively enlargement pathological tissue – tumor or connective tissue (cancer, pulmonary fibrosis).



Depending on the character there are the next forms of pulmonary tissue consolidation:

I. According to the localization:

- consolidation in the upper lobe (fig. 1.1);
- consolidation in the middle lobe (fig. 1.2);
- consolidation in the lower lobe (fig. 1.3).

II. According to the widespread:

- segmental (focal);
- lobar;
- diffuse (several lobes).

III. According to the development:

- rapidity (acute pneumonia, lung infarction);
- gradual (tumor).

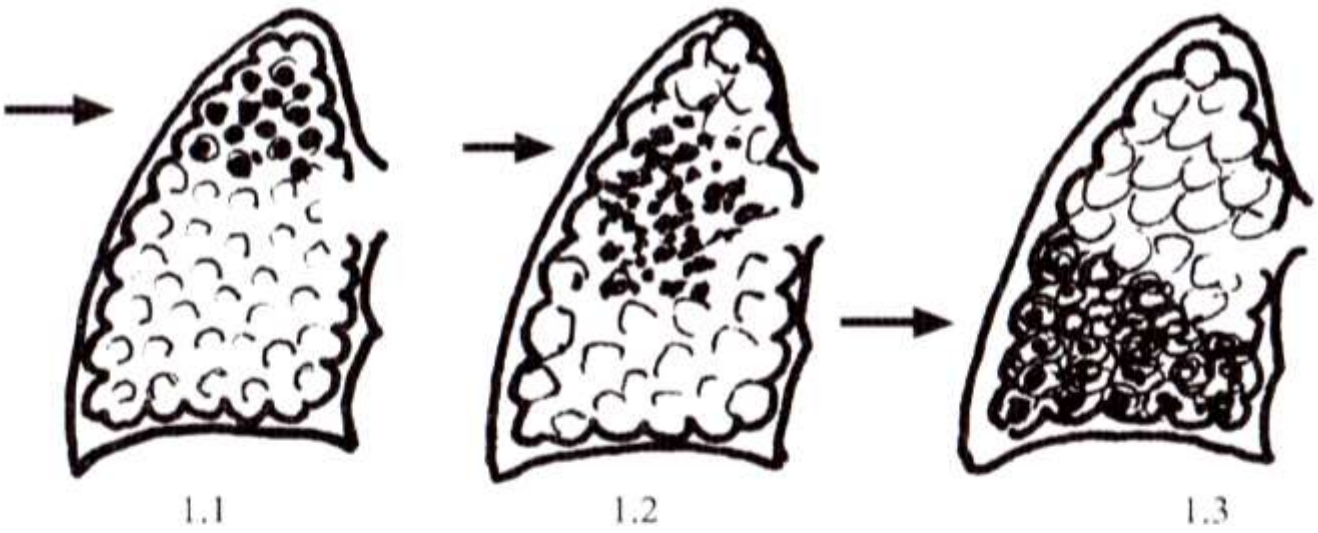


Fig. 1.1. Consolidation in the upper lobe.
Fig. 1.2. Consolidation in the middle lobe
Fig. 1.3. Consolidation in the lower lobe.

Clinical features

Decreased airiness of the pulmonary tissue lead to the impaired lung ventilation, development of tissue and cellular hypoxia that clinically manifested by dyspnea, asphyxia, cough, hemoptysis, pain in the chest during respiration.

Dyspnea – the major complaint in patients with the syndrome of decreased airiness of the pulmonary tissue. Commonly it can be observed during inspiration or has mixed character, may be periodic or permanent (pneumonia, tuberculosis, bronchocarcinoma), augmented with asphyxia advance (TEPA, pneumothorax, congenital heart failure).

Cough – may be dry or with sputum discharge, periodic or permanent, from time to time accompanied with hemoptysis (pneumonia, tuberculosis, bronchocarcinoma).

Pain in the chest – the onset of the pain is connected with the deep respiration, coughing or permanently increased in oncological pathology.

Objective examination. *General patient's condition* may be satisfactory (prodromal period, the stages of recovery or remission); may be middle grave, moderate grave, extremely grave (lobar pneumonia, TEPA, cancer). Due to the chronic hypoxia and general intoxication may be observed the deranged consciousness in form of stupor, sopor or seldom coma (tuberculosis, cancer).

The posture of the patients is frequently forced: orthopnea – in order to reduce dyspnea via decreased volume of circulating blood (lobar pneumonia, compressive or obstructive atelectasis, bronchocarcinoma) or lying on the affected side – in order to relieve the pain via limitation of the pleural layers movement (pneumonia, hydrothorax, pneumothorax, massive lung tumor).

Facies pneumonica – one-sided blush on the same cheek as affected lung is specific for lobar pneumonia; *facies tuberculous* – exhausted, pale face with blush localized on the cheeks, "burning eyes", dry lips, excited countenance, half open mouth is specific for tuberculosis.

The color of the skin is characterized by central or diffuse cyanosis due to the accumulation of the carbon dioxide and reduced restored hemoglobin.

Inspection of the chest may reveal – asymmetrical chest with one half falls in the breathing act (pneumo- or hydrothorax).

Vocal fremitus as usual increased, except obstructive atelectasis when it isn't detected on the affected side.

In comparative percussion of the lungs determinates the changes of percussion sound from intermediate to dullness:

– intermediate (softer, higher, shorter) – decreased airiness of the pulmonary tissue (lobar pneumonia initial stage, fibrous-focal tuberculosis, pulmonary edema, initial stage of compressive or obstructive atelectasis);

– dullness (soft, high, short) – consolidation of the pulmonary tissue (lobar pneumonia consolidation stage, lung infarction, the complete adhesion of the lung due to the obstruction, cancer).

In auscultation of the lungs according to the character, intensity and scattering of the pathological process can be observed decreased vesicular or bronchial breathing and adventitious respiratory sound in form of moist rales and crepitation:

– pathologically decreased vesicular breathing occurs due to the inflammation and swelling of alveolar walls and partial alveoli filled by effusion (initial and final stages of acute lobar pneumonia) or unexpected airflow decrease with alveolus adhesion (obstructive atelectasis);

– pathologically bronchial breathing occurs due to the complete consolidation of the pulmonary tissue via replacement by airless tumor or fibrosis tissue (pneumosclerosis, cancer), totally alveoli filled by effusion (second stage of acute lobar pneumonia, tuberculosis, pulmonary edema, lung infarction) or completely alveoli compressing by pleural air or fluid (compressive atelectasis);

– over the regions with pathologically decreased vesicular breathing as usual is revealed crepitation, rarely fine or medium bubbling moist rales. Most rales are revealed in the second stage of acute lobar pneumonia with pathologically bronchial breathing.

Additional methods of examination

Clinical blood analysis: due to inflammatory process – leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated Erythrocyte Sedimentation Rate (ESR); throughout due to in lesser circulation and atelectasis – secondary erythrocytosis; in tumor – anemia, accelerated ESR.

Sputum analysis: due to inflammatory process the character of the sputum is mucous-purulent or mucous-purulent bloody, in microscopic study are revealed cellular elements (columnar, ciliary, alveolar macrophages, increased amount of leukocytes and erythrocytes), fibrous elements (fibrin fibers) and presence of pneumococcus, streptococcus, staphylococcus in bacterioscopic study of the sputum; in destructive process are revealed large amount of cellular and fibrous elements, presence of mycobacterium tuberculosis or atypical cells.

Roentgenoscopy and roentgenography (X-ray examination): consolidation of the pulmonary tissue, tumor, the signs of bronchium obstruction, hydrothorax, pneumothorax.

Syndrome of increased airiness of the pulmonary tissue

The syndrome of increased airiness of the pulmonary tissue is based on the protracted enlargement of residual air volume in the lung that clinically manifests by emphysema (fig. 1.4).

Etiology:

– chronic bronchial obstruction;

– decreased of the pulmonary tissue elasticity;

– compensatory reaction on the advance of destructive process in the lung and diffuse fibrosis.

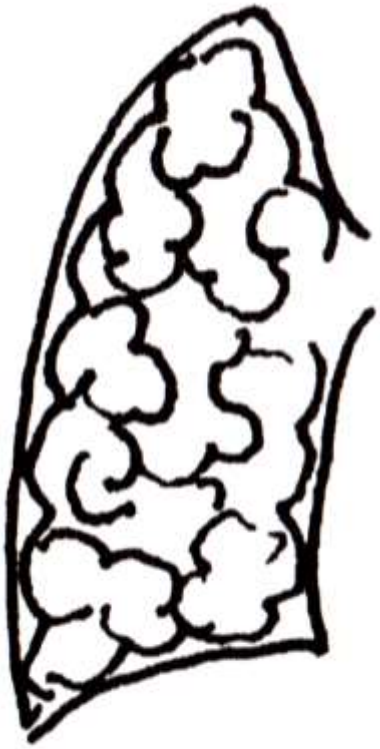
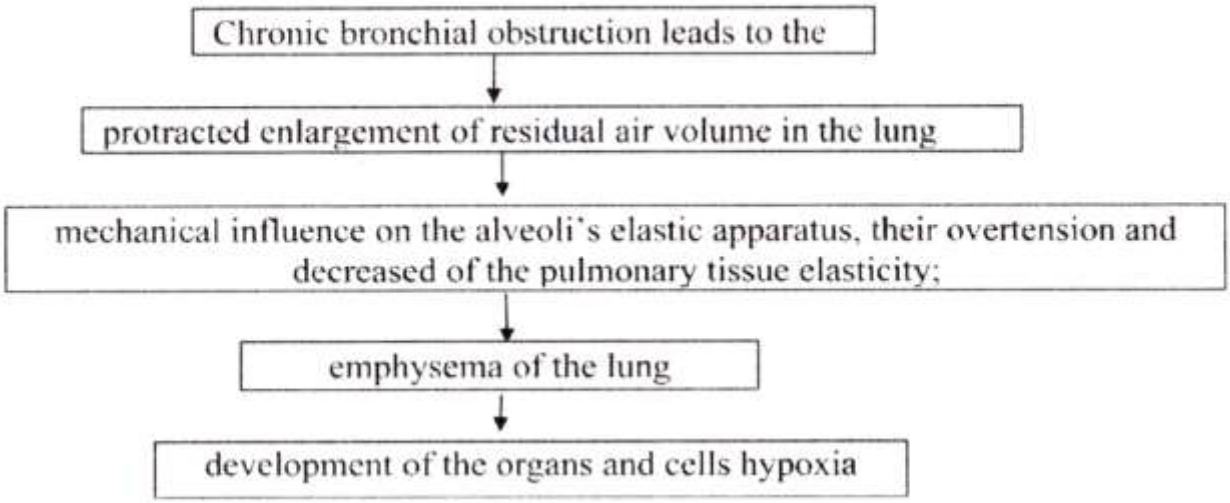


Fig. 1.4. Increased airiness of the pulmonary tissue.

Pathogenesis



Depending on the character and mechanism there are the next forms of increased airiness of the pulmonary tissue:

I. According to the widespread:

– local (one sided injury);

– diffuse (both lungs injury).

II. According to the development:

– destructive (chronic obstructive lung diseases, bronchiectatic disease);

– nondestructive (bronchial asthma).

Usually of bronchial obstruction has diffuse character, lung emphysema is most frequently bilateral process and assessed as complication of chronic lung diseases.

Clinical features

The main complaints in patients with increased airiness of the pulmonary tissue are dyspnea and cough.

Dyspnea – has expiratory or mixed character and increased during physical activity.

Cough – commonly dry and has reflex character, on destructive processes – with purulent sputum discharge.

Objective examination. *General patient's condition* may be satisfactory (early stage of the disease, the stage of remission); may be middle grave, moderate grave or grave (progression of bronchiectatic disease, destructive process in the lung, bronchial asthma attacks). Due to the acute or gradual chronic hypoxia may be observed the deranged consciousness.

The posture of the patients is frequently active. May be observed the forced posture in form of orthopnea (spasm of bronchi, attacks of bronchial asthma, decreasing the breath surface).

The color of the skin is characterized by central or diffuse cyanosis due to the accumulation of the carbon dioxide and reduced restored hemoglobin.

Inspection of the chest may reveal barrel-like (emphysematous) form of the chest with protruded supra- and subclavicular fosses, horizontal direction of the ribs, smoothed and narrow intercostals spaces, increased anteroposterior diameter. As usual the chest is symmetrical, the type of respiration is mixed or thoracic, accessory respiratory muscles active participate in the breathing act, especially m. sternocleidomastoideus and m. trapezius with evident elevation and lowering of the entire chest during breathing. May be observed tachypnea with shallow respiration depth.

Palpation of the chest. Elasticity of the chest is decreased (rigid chest), the chest is painless. Vocal fremitus is badly transmitted.

Percussion of the lungs. In comparative percussion of the lungs generalized hyperresonance (bandbox sound) may be heard over the hyperinflated lungs of emphysema. In topographic percussion of the lungs is observed bilateral lowering of the lower lungs edges, respiratory mobility of the lower borders of the lungs is decreased.

Auscultation of the lungs. In auscultation of the lungs may be observed pathologically decreased vesicular breathing and dry rales.

Additional methods of examination

Clinical blood analysis: secondary erythrocytosis; leukocytosis, neutrophilia, accelerated ESR (during progression of chronic diseases), eosinophilia (bronchial asthma).

Sputum analysis: data depends on the main disease.

X-ray examination: the signs of increased airiness of the pulmonary tissue, low diaphragm's position.

Syndrome of bronchium obstruction (bronchospastic syndrome)

Bronchospastic syndrome – the grouping of symptoms that developed due to the impaired air entrance to the pulmonary tissue through bronchus and accompanied by decreased lung's ventilation, enlargement of residual air volume in them, clinically manifests by intensive cough and resulted in emphysema.

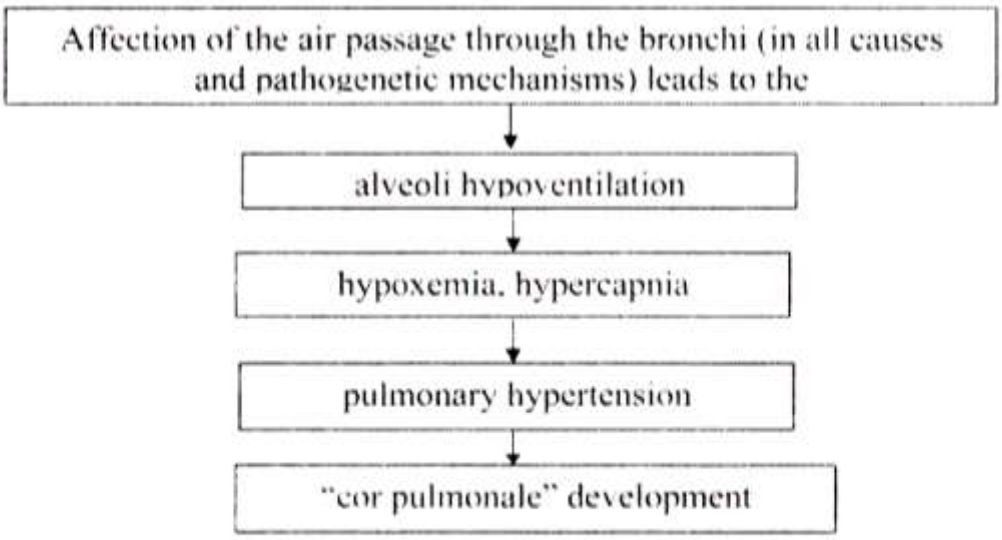
Etiology:

- spasm of the smooth muscles;
- inflammatory infiltration and edema of the tracheobronchial tree mucus;

- non-uniform swelling of the bronchial mucus due to the inflammation or viscous sputum narrows the lumen of bronchi;
- deformity of the bronchial tree;
- expiratory bronchi collapse;
- external compression of bronchi by diffuse peribronchial fibrosis.

Pathogenesis

In syndrome of bronchium obstruction at first modify air passage in small bronchi and bronchioles due to the inflammatory edema and swelling of their mucosa (chronic bronchitis), spasm in the smooth muscles (bronchial asthma) and the external compression by peribronchial diffuse fibrosis.



Clinical features

The main complaints in patients with bronchium obstruction are dyspnea and cough.

Dyspnea commonly has expiratory character, gradually increased (chronic obstructive lung diseases) and frequently transformed to periods of asthma (bronchial asthma).

Cough is commonly periodic, moist with difficult sputum expectoration that has mucous or mucopurulant character, tenacious consistency, glass-like or glass like with yellow traces color.

Objective examination. *General patient's condition* is from middle grave to grave. Due to the acute or gradual chronic hypoxia may be observed the deranged consciousness.

The posture of the patients is frequently forced in form of orthopnea – sitting position fixing the shoulder girdle in order to reduce dyspnea via assistance of accessory muscles and diaphragm to take part in respiration.

The color of the skin depends on the variant of obstruction. In chronic bronchitis observe diffuse cyanosis with peripheral edema due to the "cor pulmonale" development. In obstructive emphysema bronchi spasm occurs during expiration therefore alveolar air is a little change and inspite for constant dyspnea the skin and visible mucous cyanosis isn't specific.

The data of chest inspection, palpation and percussion include clinical features of bronchium obstruction complications: emphysematous form of the chest with accessory respiratory muscles participation in the breathing act, decreased excursion of the chest, badly transmitted vocal fremitus and generalized bandbox sound over the lungs during percussion.

Auscultation of the lungs. Auscultative data are the main specific in patients with bronchospastic syndrome: they characterized by dry rales over the pathologically increased vesicular breathing. Moreover, the particularities of the rales give possibility to evaluate the cause of the obstruction, the size and depth of the affected bronchi:

– in localized affection of medium and large bronchi insignificant amount of low pitched and soft rales are heard;

– widespread bronchi inflammation or bronchospasm in asthma attack both sibilant and sonorous rales of different tone and intensity are heard;

– accumulation of the viscous secretions in the lumen of bronchi accompanied by dry rales that can be altered by coughing or deep inspiration.

Additional methods of examination

Clinical blood analysis: secondary erythrocytosis; leukocytosis, neutrophilia, accelereted ESR (during progression of chronic diseases), eosinophilia (bronchial asthma).

Sputum analysis: the character of the sputum is mucous or muco-purulant, tenacious or tenacious thick consistency, glass-like or glass like with yellow traces color, odorless and absent of layers ness. In microscopic study are revealed columnar, ciliary epithelium, leucocytes, alveolar macrophages, eosinophils, fibrin fibers, Charcot-Leyden crystals and large amount of microorganisms (bacterial flora).

X-ray examination: augment and deformity of lung picture over increased in transparent lung tissue.

Syndrome of fluid accumulation in pleural cavity (hydrothorax)

Hydrothorax – the grouping of the symptoms that develop due to the pleura affection or general electrolyte dysbalance in the organism.

Etiology

The main causes of pleural fluid accumulation

Character of pleural fluid	Main causes	Less frequent causes
Transudates	Heart failure	Nephritic syndrome Cirrhosis Myxidema Peritoneal dialysis
Inflammatory exudates (infectious)	Parapneumonic Tuberculosis	Subdiaphragmatic abscess Viral infectious Fungus infectious
Inflammatory exudates (noninfectious)	Pulmonary artery thromboembolia	Collagenosis Pancreatitis Reaction on drugs Dresler syndrome
Tumor induced exudates	Metastasis Lymphoma	Mesothelioma Meigsa syndrome
Hemothorax	Trauma	Spontaneous (impaired hemostasis)
Chylothorax	Lymphoma Carcinoma Trauma	Lymphogranulomatosis

Pathogenesis

In hydrothorax developing the primary affection belongs to inflammatory or reactive process in pleura that accompanied by fluid accumulation in the pleural cavity from several milliliters to 1 liter or more.

Classification

Depending on the etiology, character of excudates, duration and clinic-anatomic form there are the next forms of excudative pleurisy:

I. According to the etiology:

- infection;
- non-infection.

II. According to the character of exudates:

- serous;
- seropurulent;
- purulent;
- hemorrhagic;

- cholesterol;
- putrefactive

III. According to the duration:

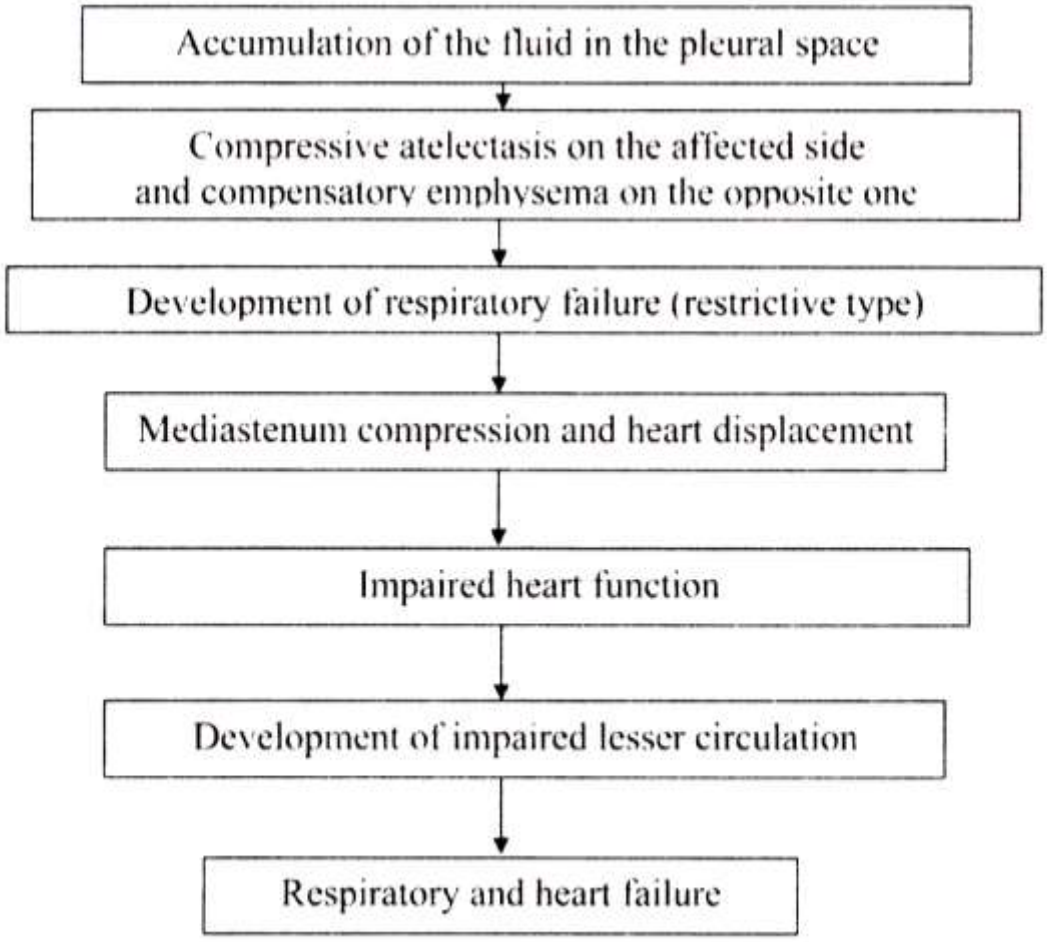
- acute;
- subacute;
- chronic.

IV. According to the clinic-anatomic form:

- diffuse;
- local.

Besides exudates in pleural cavity may accumulate uninflammatory fluid (transudate) due to the impaired electrolyte exchange (increased hydrostatic pressure in the capillaries and decreased colloid and osmotic pressure in plasma).

As result pure of proteins plasma transits through unchanged capillary's wall and accumulates in the pleural cavity (heart failure, nephritic syndrome, liver cirrhosis, alimentary dystrophia, severe anemia, mediastenum tumor, myxedema, compression of vena cava superior).



Clinical features

The main complaints in patients with syndrome of fluid accumulation in pleural cavity are: dyspnea, pain in the chest and cough; additional – weakness, loss of appetite, hyperthermia, perspiration due to the inflammatory process and general intoxication.

Dyspnea – more frequently has subacute onset and its degree depends on pleural cavity's fluid volume, the speed of fluid accumulation, grade of vital lung capacity and ventilation diminish via their compression by fluid and mediastenum displacement.

Chest pain – has a sharp stabbing and knife-like character and is accentuated by respiratory movement and coughing. Hence it is aggravated by respiration and coughing thus leading to rapid shallow breathing and suppressed cough.

If pleurisy progress to pleural effusion, the sharp pain largely disappears and is replaced by a dull and more constant ache or heaviness, quantitatively roughly proportional to the amount of fluid.

Cough – more commonly has dry character.

Objective examination. *General patient's condition* is from middle grave to extremely grave. Due to hypoxia, the inflammatory process and general intoxication may be observed the deranged consciousness.

The posture of the patients is frequently forced (lying on the affected side) in order to relieve the pain via limitation of the pleural layers movement and relieve dyspnea via decrease pressure of the fluid on mediastenum and therefore its displacement.

The color of the skin and visible mucous is characterized by diffuse cyanosis. In presence of effusion in mediastenum may be observed edematous face, swollen and pulsation of jugular veins, voice change and dysphagia.

Depending on the stage of pleural syndrome development there are the next particularities of objective examination:

I. In the initial stage of the hydrothorax there are more frequently observed the signs of dry pleurisy:

– poor movement of the affected side in respiration during dynamic inspection of the chest;

– in topographic percussion decreased of the lower lung's border respiratory mobility on the affected side;

– in auscultation of the lungs on the affected side over the region with pathologically decreased vesicular breathing is revealed pleural friction sound.

II. In stage of the fluid accumulation in pleural cavity there are observed the signs of effusive pleurisy:

– the chest is asymmetrical;

– affected half of the chest lags in the breathing act;

– vocal fremitus is badly transmitted or generally absent on the affected side (depending on pleural fluid volume);

– in comparative percussion of the lungs is determined the dull sound over the fluid. The minimum amount of the fluid that can be detected by percussion of the lungs is not less then 500 ml; determination of the dull sound from the IV rib is commensurable with 1.5:1 of fluid; from III rib – 2:1; from II rib – more then 3:1.

In presence of exudates. The upper border looks approximating a curve with a apices along posterior subribs line (the line of Damuaso-Sokolsky – fig. 1.5 a) that characterized by unevenness of fluid accumulation due to the different lung tissue compliant. In body position change the border of the dullness isn't change via visceral and parietal pleura adhesion across the exudates upper border

In occurrence of significant exudates amount in pleural cavity there are distinguish the next clinically and diagnostically zones:

– the zone of absolute dullness that accordant to exudates gathering and limited by Damuaso-Sokolsky line;

– the zone of dull sound that accordant to partial compressive atelectasis on the affected side (Garland triangle) (fig 1.5 b) and limit by spinal column, ascendant part of Damuaso-Sokolsky line and line drawing from top of Damuaso-Sokolsky line to spinal column;

– the zone of intermediate percussion sound on the health side accordantly to displaced mediastenum and joining of dull sound from exudates radiated on spinal column (Rauxfux-Grokkus triangle) (fig. 1.5 c);

– the zone of clear pulmonary sound (fig. 1.5 d) that accordant to the lung free of fluid and placed over Garland triangle and Damuaso-Sokolsky line;

– the zone of bandbox percussion sound (fig. 1.5 e) over the hyper inflated lung of emphysema on the health side.

Accordantly to distinguish zones the next auscultative records are observed:

– above the zone of absolute dullness – the breathing isn't detects or occurs pathologically decreased vesicular breathing;

– above the zone of dull sound (Garland triangle) – occurs pathologically bronchial breathing;

– above the zone of intermediate percussion *sound* (Rauxfux-Grokkus triangle) – vesicular breathing is absent via impaired sound transmition;

– over the zone of clear pulmonary sound and the zone of bandbox percussion sound (where the lungs are free of fluid) – occurs pathologically increased vesicular breathing.

In presence of transudates. The fluid's level is horizontal, more frequently bilateral and displaced throughout body's position change. The zone of dull sound (Garland triangle*)* is absent.

Moreover, in patients observed the signs of main disease that leads to hydrothorax – heart failure, renal diseases, tumor of mediastenum.

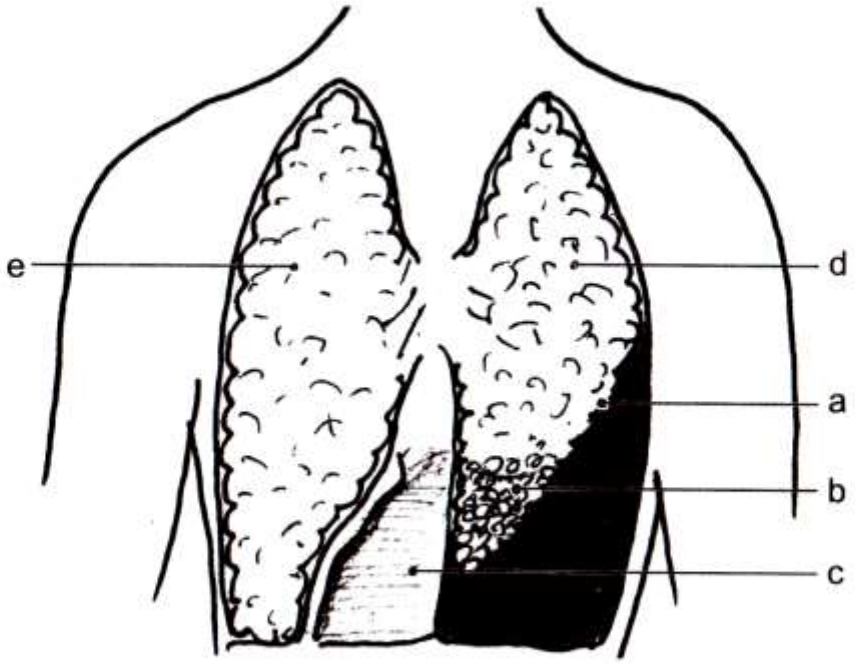


Fig. 1.5

Additional methods of examination

Clinical blood analysis: leukocytosis, neutrophilia, accelerated ESR (during progression of chronic diseases), may be anemia and secondary erythrocytosis;

Sputum analysis: more frequently sputum isn't discharge.

X-ray examination: over the fluid assessed intensive, homogenous darkening with horizontal or slating level.

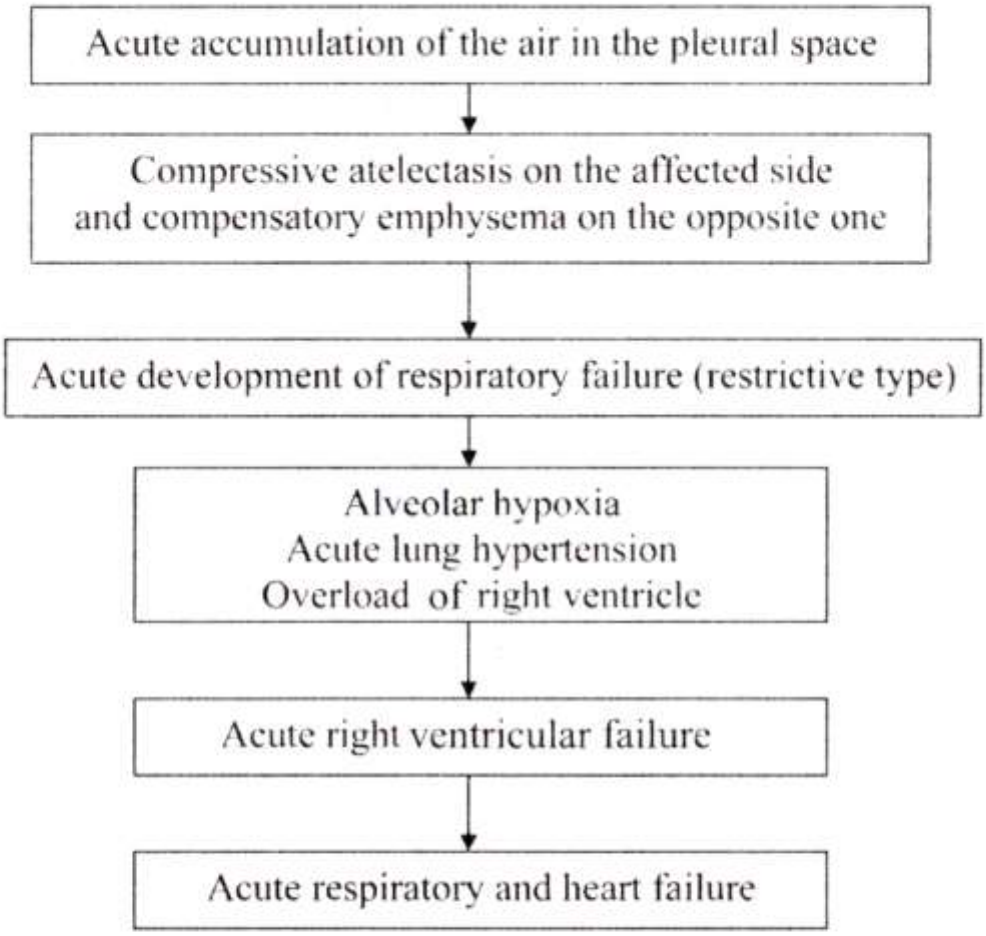
Pleural fluid analysis. Characteristics of the pleural fluid obtained in thoracentesis may be serous and serofibrinous (exudative pleurisy, rheumatic pleurisy); seropurulent (pneumonia, tuberculosis, exudative pleurisy); purulent (bacterial pleurisy); hemorrhagic (traumatic pleura affection, tuberculosis, infarction or tumor of the lungs); chyleous (congestion of the lymph or destruction of the thoracic duct by a tumor or an injury); cholesterol (chronic in flammation of the serous membrane as a result of cellular degradation with fatty degeneration); putrefactive (lung's gangrene).

Syndrome of air accumulation in pleural cavity (pneumothorax)

Pneumothorax – one of complication in the destructive lungs and pleura processes (abscess, hungrena, bronchiectasis, tuberculosis and tumor).

Pathogenesis

In pneumothorax developing the primary point belongs to the effortless lung's tissue destruction with air entrance to pleural cavity in circumstance of preceding pulmonary tissue or pleura pathology that occurs during intensive coughing, laughing, deep inspiration, extremely physical activity or trauma.



Depending on the localization, widespread and particularities of development there are distinguish the next forms of pneumothorax:

I. According to the localization:

- unilateral;
- bilateral.

II. According to the widespread:

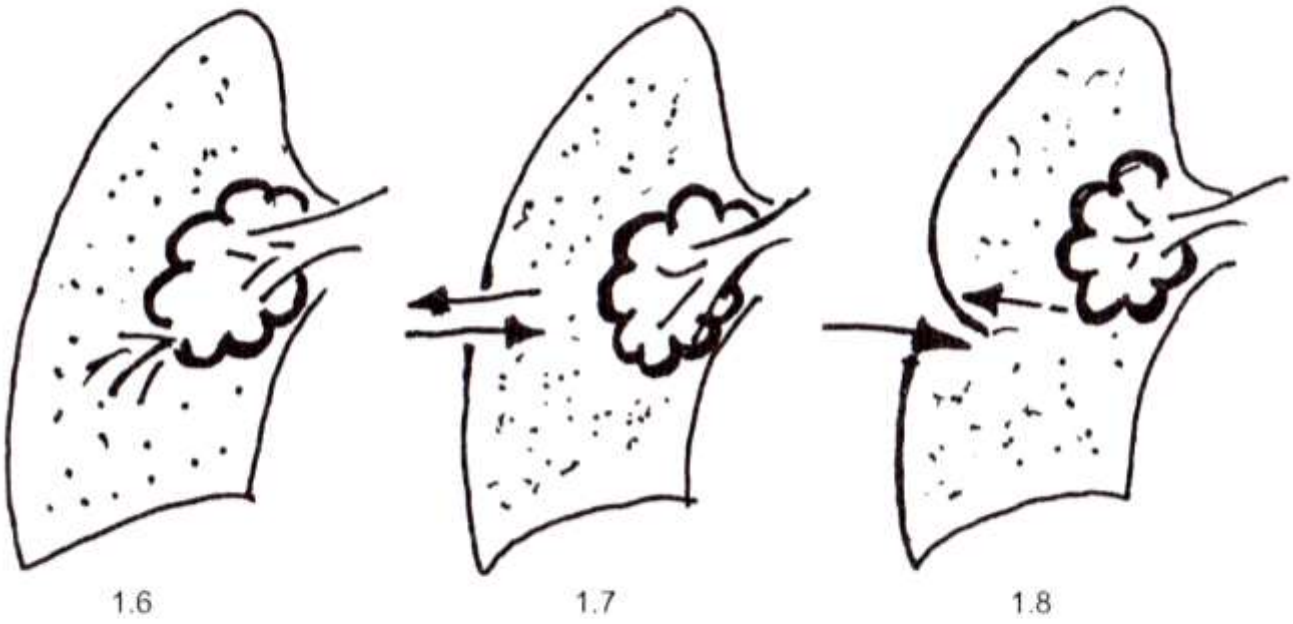
- partial;
- total.

III. According to the mechanism of development:

- closed (fig 1.6);
- opened (fig 1.7);
- valve (fig 1.8).

IV. According to the etiologic factors:

- spontaneous;
- traumatic;
- surgical;
- artificial (treatment).



1.6 1.7 1.8

Fig. 1.6. Closed pneumothorax.
Fig. 1.7. Opened pneumothorax.
Fig. 1.8. Valve pneumothorax.

Clinical features

The main complaints in patients with syndrome of air accumulation in pleural cavity are characterized by the symptoms of acute respiratory and right ventricle failure: dyspnea, pain in the chest, cough and palpitation.

Dyspnea – has acute onset, mixed character, rapidly increased and transmit to asphyxia. Its degree depends on pleural cavity's air volume, the speed of air accumulation, grade of vital lung capacity and ventilation diminish via their compression and mediastenum displacement. The most grave dyspnea assessed in valve type of pneumothorax with total lung's atelectasis.

Chest pain – has a sharp stabbing and knife-like character, localized on the affected side and may radiated to the neck, arm, epigastrium and accentuated by respiratory movement and coughing.

Cough – more commonly reflectors and has dry character via pleural receptors irritation.

Palpitation – has compensatory character due to the gradually increased hypoxia, intrathoracic pressure elevation and mediastenum organs displacement.

Objective examination. *General patient's condition* is from grave to extremely grave.

The posture of the patients is frequently forced (lying on the affected side) in order to revile the pain via limitation of the pleural layers movement and revile dyspnea via decrease pressure of the air on mediastenum and therefore its displacement.

The color of the skin and visible mucous is characterized by diffuse cyanosis.

Inspection of the chest reveal its asymmetry; enlargement of the affected side with increased distance between umbilicus and median line and from inside edge of scapula to the spine; asymmetry of the clavicles, protrusion and raise in wide of the interspaces. The type of respiration is mixed, affected side falls in breathing act, may be observed tachypnea with rapid shallow breathing.

Palpation of the chest. Elasticity of the chest is decreased (rigid chest), the chest is painful. Vocal fremitus is badly transmitted or absent on the affected side.

Percussion of the lungs. In comparative percussion of the lungs occurs unilateral tympanic or metallic percussion sound. In opened pneumothorax the cracked-pot percussion sound is observed.

In topographic percussion of the lungs is observed unilateral lowering of the lower lung's border with decreased its respiratory mobility.

Auscultation of the lungs. In auscultation of the lungs over affected side the breathing isn't determined or observed decreased vesicular breathing; over the healthy lung – pathologically increased vesicular breathing.

NB! In opened pneumothorax is typical bronchial breathing ("'metallic" – the variant of bronchial breathing with loud intensity and high pitch).

In valve type of pneumothorax is typical bronchial breathing with determination of breathing only during inspiration.

Additional methods of examination

Clinical blood analysis: without particularities

Sputum analysis: the sputum isn't discharged

X-ray examination: the signs of pneumothorax are large clear up space without lung's depiction, comprehensible border of the adhesives lung, displacement of mediastenal organs to the healthy side and low diaphragm's position.

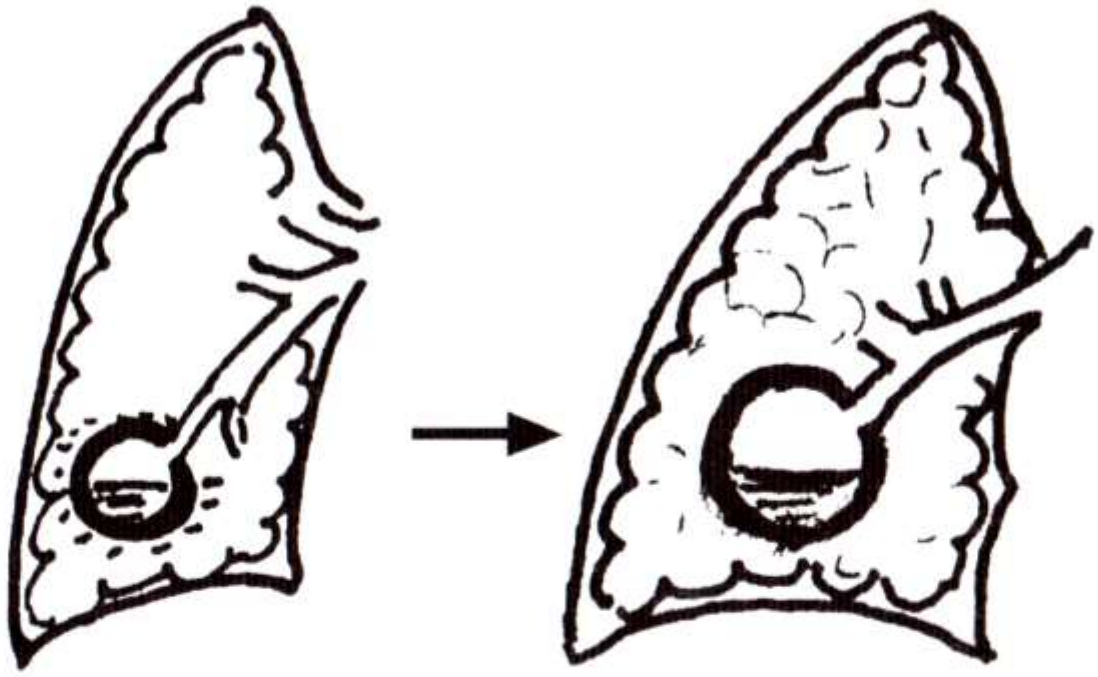
ECG – deviation of electrical axis of the heart to the right, ''P-pulmonale'', decreased R-wave in right leads.

Syndrome of the cavity in the lungs

The syndrome of the cavity in the lungs is destruction of the pulmonary tissue with solid focal wall rounded by infiltrative or fibrous bank. The cavity may be filled only by air (''empty cavity'') or contain except air quite a few amount of fluid, may be closed or communicated with bronchium.

Etiology

- chronic bronchial obstructive diseases;
- severe pneumonia;
- tuberculosis (fig 1.9);
- tumor of the lungs (fig 1.10);
- traumas of chest;
- parasite affection of the lungs;
- decreased human bodies reactivity



1.9 1.10

Fig. 1.9. Tuberculosis caverna.
Fig. 1.10. Lung abscess.

Pathogenesis

Lung's tissue destruction develops under influence of pathogenic microflora's proteolyses ferments (more frequently pathogenic hemolytic staphylococcus or mycobacterium of tuberculosis). Under condition of own bodies reactivity and immune mechanisms effort it is localized by solid capsule rounded by infiltrative or fibrous bank.

Clinical features

The clinical features of the syndrome is dynamic and depends on the stage of disease (the stage of formation or burst to the bronchi), the size, depth and character of contents (air or fluid).

During clinical examination the cavity in the lung may be revealed only in certain conditions: if superficially placed cavity more than 6 cm in diameter has smooth wall, contains the air and communicated with bronchium.

The main complaints in patients with the syndrome of the cavity in the lungs are: dyspnea, cough, hemoptysis, pain in the chest. However during the stage of cavity formation the syndrome is manifested by the symptoms of general intoxication: hyperthermia, loss of appetite, loss of weight, weakness. The typical clinic develops after cavity burst to the bronchi.

Cough – may be periodic or permanent, at the initial stage commonly dry which turn to the moist with large amount (by "full mouth") of mucopurulent, puromucous or purulent sputum that has yellow-greenish color, sharp unpleasant odor with blood traces. The time of sputums expectorated increasing with the posture of the patient particularly its changes and the time of day (increased after patients awoke in the morning).

Hemoptysis – is observed in the moment of cavities bursting to the bronchi and may be in form of red color streaks that mixed with puromucous or purulent sputum. However it may have recurrent character including development of pulmonary bleeding.

Dyspnea – commonly it has mixed character, may be periodic or permanent and amplified during physical activity due to the hypoxia and general intoxication.

Pain in the chest – the onset of the pain is patients with the syndrome of the cavity in the lungs connected with the tumor growing to the pleura and intrathoracic lymph nodes.

Objective examination. *General patient's condition* may be middle grave, moderate grave, extremely grave. Due to the chronic hypoxia and general intoxication may be observed the deranged consciousness in form of stupor, sopor or seldom coma (tuberculosis, cancer).

The posture of the patients is frequently forced: orthopnea or lying on the side.

The color of the skin is pallid with gradually growing cyanosis due to the accumulation of the carbon dioxide and reduced restored hemoglobin. In patient may be observed loss of weight till the level of cachexia. The nails are in the form of watch glasses (''Hippocratic nails'').

Inspection of the chest may reveal – dynamic asymmetric in the chest with one half lags in the breathing act.

Palpation of the chest – the elasticity of the affected chest decreased, vocal fremitus as usual increased over the cavity connecting with the bronchus and contains the air or decreased over isolated cavity independently from its contain.

In comparative percussion of the lungs over the cavity determinates:

- tympany percussion sound (over the cavity contains air);
- cracked-pot percussion sound (over superficial cavity);
- dull percussion sound (over the cavity containing fluid).

In auscultation of the lungs according to the stage of the disease, size and localization of the cavity the auscultative picture is different:

– the breathing isn't determined over the isolated cavity contains the air;

– pathologically bronchial breathing over the cavity occurs if it contains the air and connecting with bronchus;

– amphoric respiration is heard in presence of large smooth-wall cavity (not less than 5–6 cm in diameter) communicated with large bronchus and situated superficially;

– over the cavity partially contains the fluid may be revealed, rarely fine or medium bubbling moist rales.

Additional methods of examination

Clinical blood analysis: due to inflammatory process – leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated ESR; due to congestion in lesser circulation and atelectasis – secondary erythrocytosis; in tumor – anemia.

Sputum analysis: due to inflammatory process the character of the sputum is mucous-purulent or mucouspurulent bloody with unpleasant odor. In microscopic study are revealed cellular elements (columnar, ciliary, alveolar macrophages, increased amount of leukocytes and erythrocytes), fibrous elements (fibrin fibers) and presence of pneumococcus, streptococcus, staphylococcus in bacterioscopic study of the sputum; due to destructive process are revealed lured amount of cellular and fibrous elements, presence of mycobacterium tuberculosis or atypical cells.

X-ray examination: observed the cavity in the lungs.

Bronchitis

Bronchitis – inflammatory injury of bronchial tree.

Depending on the etiology, mechanism and character of duration there are distinguish the next forms of bronchitis:

1. Acute bronchitis.
2. Chronic bronchitis.

Acute bronchitis

Acute bronchitis – one of the most widespread acute diseases of respiratory tract.

Etiology:

– viral infection;

– bacterial infection;

– influence of physical and chemical factors.

Clinical features

The disease is characterized by acute onset that starts from periodic dry cough which after some days changes to the moist with mucous, mucopurulent (sometimes with blood traces) sputum discharge.

Objective examination. Results of general patient's examination and chest inspection are without particularities. Data of chest palpation and percussion of the lungs is of any changes.

In auscultation of the lungs over the regions with pathological increased vesicular breathing are revealed a lot of dry rales that can change in their intensity, localization or disappeared after coughing.

Additional methods of examination

Clinical blood analysis: the signs of inflammation (leukocytosis, shift of leukocyte formula to the left, accelerated ESR).

Sputum analysis*:* the character of the sputum is mucous or mucous-purulent (sometimes with traces of blood), scarce amount and odorless. In microscopic study observed columnar, ciliary epithelium, leucocytes and alveolar macrophages.

Chronic bronchitis

Etiology

– smoking, pollution of the environment by products of incomplete fuel substances combustion, organic and inorganic dust;
– infection (bacterial, viral, micoplasms, fungus);
– congenital occurrences in lesser circulation on heart failure;
– exposure of metabolic products on renal failure;
– result of acute bronchitis.

Classification of chronic bronchitis (by N.P. Paleev, 1990)

I. According to the character of inflammatory process:
– simple (catarrhally);
– purulent;
– muco-purulent;
– special forms: hemorrhagic and fibrinous.

II. According to the presence of bronchial obstruction:
– obstructive bronchitis (stages: I, II, III; duration: simple, moderate grave, grave);
– non obstructive bronchitis.

III. According to the level of bronchi injury:
– proximal;

– distal;
– diffuse.

IV. According to the duration:
– latently;
– with infrequent aggravations;
– with frequent aggravations;
– continuously progress.

V. According to the phases:
– progress;
– remission.

VI. According to the complications:
– emphysema of the lungs;
– hemoptysis;
– pneumonia;
– respiratory failure;
– "Cor pulmonale".

Pathogenesis

On chronic bronchitis occurs development of classic pathogenetic triad:
– hypercrinia (mucous hyperproduction);
– dyscrinia (increased sputum viscosity);
– mucostasis (overcrowding of the sputum in bronchi).

Approaching to the bronchi of infection agent leads to the sensebilization and autosensebilization of the organism.

There are the next mechanisms of the bronchial obstruction development:
– brochospasm;
– inflammatory edema and bronchial wall infiltration;
– hyper- and dyscrinia;
– hypotonic dyskinesia of large bronchi;
– collapse of small bronchi during expiration;
– mucus lays hyperplasic reaction.

Clinical features

The main complaints in patients with chronic bronchitis are moist cough, general weakness, perspiration and dyspnoea in cause of bronchium obstruction.

Cough is commonly periodic, moist with difficult sputum expectoration.

Sputum expectoration is the most important symptom of chronic bronchitis. On early stages of the disease the sputum may be mucous, tenacious consistency, glass-like, for the period of progression becomes mucopurulant or purulent. The 24-

hours amount of sputum is usually 50–70 ml, due to development of bronchiectasis significantly increase to 100–200 ml.

Dyspnea – commonly has expiratory character and its appearing indicates presence of bronchial obstruction and emphysema.

Objective examination. *General patient's condition* is usually satisfactory. On progression and complications advance general patient's condition may be from middle grave to grave. Due to gradual chronic hypoxia and intoxication possibly will be observed perspiration and subfebrile or febrile temperature.

The posture of the patients is frequently active. On progression and complications advance is forced in form of orthopnea – sitting position fixing the shoulder girdle in order to reduce dyspnea via assists the accessory muscles and diaphragm to take part in respiration.

The color of the skin and visible mucous depends on the stage and variant of obstruction. In initial stage the color of the skin and visible mucous is without any particularities. Due to the chronic bronchitis progression observe diffuse cyanosis with peripheral edema via to the "cor pulmonale" development. In obstructive emphysema bronchi spasm occurs during expiration therefore alveolar air is a little change and in spite for constant dyspnea the skin and visible mucous cyanosis isn't specific.

In purulent chronic bronchitis detect the form of the Hippocratic nails.

The data of chest inspection, palpation and percussion include clinical features of bronchium obstruction: emphysematous form of the chest with accessory respiratory muscles participation in the breathing act, decreased excursion of the chest, badly transmitted vocal fremitus and generalized bandbox sound over the lungs during percussion.

Auscultation of the lungs. Auscultative data in patients with chronic bronchitis is characterized by sibilant and sonorous dry rales of different tone and intensity over the pathologically increased vesicular breathing. In localized affection of medium and large bronchi insignificant amount of low pitched and soft rales are heard. Accumulation of the viscous secret in bronchi via active inflammation, are accompanied by coarse and medium bubbling rales that can be altered by coughing or deep inspiration.

Additional methods of examination

Clinical blood analysis*:* without significant changes, sometimes secondary erythrocytosis; in progression assess leukocytosis, neutrophilia, accelerated ESR, eosinophilia (allergic reaction).

Sputum analysis*:* the character of the sputum depends on the stage of disease: in initial stage the sputum is mucous; in progression or later stage – muco-purulant, tenacious or tenacious thick consistency, glass-like or with yellow traces, odorless and absent of layersness. In microscopic study are revealed a lot of columns ciliated

epithelium, leucocytes, alveolar macrophages, eosinophils, fibrin fibers, Charcot-Leyden crystals and large amount of microorganisms (bacterial flora).

X-ray examination: augment and deformity of lung picture over increased in transparent lung tissue.

Test of ventilatory function (spirometric recording and pneumotachymetry): in patients with no obstructive bronchitis results of spirometric recording is comparable with healthy subjects; in patients with bronchial obstruction assess decreased respiratory reserve (75 % of maximum lung ventilation and lower), and decreased Votchal-Tiffeneau index.

ECG: deviation of electric axis of the heart to the right, P-pulmonale in II, III, AVF leads.

Bronchiectatic disease

Etiology

- genetic incompetence of bronchial tree;
- inflammatory and infection diseases of bronchopulmonary system in childhood, particularly with often recurrence;
- changes of bronchial secret characteristics (α_1-deficiency);
- bronchial obstruction due to the foreign corpuses, intrathoracix lymphatic nodes enlargement, protracted chronic bronchitis;
- longterm inspiration of toxic substances;
- bronchopulmonary infection.

Pathogenesis

I. The factors lead to bronchiectasis development:

- obstructive atelectasis via of bronchial secret expectoration;
- decreased of bronchial wall corresponding to improved bronchial dilation (augmented intrabronchial pressure in coughing, enlarged intrapleural negative pressure);
- development of progressive inflammatory process in bronchi (degeneration of smooth muscle tissue and its replacement by fibroses tissue).

II. The factors lead to bronchiectasis infection:

- impaired sputum discharge, congestion and secret infectivity in dilated bronchi;
- damage function of local bronchopulmonary protection and immunity.

Clinical features

Bronchiectatic disease more commonly occurs in age from 5 to 25 years, strangely later. Manifestation of the disease linked to acute respiratory pathology or pneumonia.

The main complaints in patients with bronchiectatic disease are cough, hemoptysis, dyspnea, pain in the chest, hyperthermia, general weakness, loss of ability to work and appetite, perspiration.

Cough is commonly moist, periodic with purulent greenish-yellow strong smell sputum discharge. The daily amount of sputum vary from 10–15 ml to 2 l, the amount of morning sputum is two thirds of the entire daily expectoration and changes of posture can set off coughing and sputum discharge. The prominent particularity of sputum on bronchiectatic disease is its three-layers on standing (pus, plasma and upward mucus). In periods of remission the sputum amount decreases.

Hemoptysis – appears or becomes more intensive in period of disease progression or physical activity. Sometimes occurs substantial bleeding with clots from affected bronchial arteries. In bronchiectasis, however, hemoptysis not uncommonly mixed with mucopurulent sputum.

Dyspnea – has expiratory or mixed character, occurs frequently during increased physical activity or periods of disease progression and is the signs of chronic bronchitis and lung emphysema.

Pain in the chest – isn't permanent complaint and associated with pleura affection, as usual increased during deep inspiration.

Hyperthermia, general weakness, perspiration, loss of appetite are the signs of intoxication syndrome and observed during progression of disease.

Objective examination. *General patient's condition* may be satisfactory (early stage of the disease, the stage of remission); may be middle grave, moderate grave or grave (progression of bronchiectatic disease, destructive process in the lung). Due to the gradual chronic hypoxia in last stages of disease may be observed the deranged consciousness.

In general examination may observe loss or deflection of weight, muscular dystrophy and weakness, lack of secondary sexual characters, nails in a form of "watch glass" (Hippocratic nails). Digital clubbing in its most gross form is seen as a bulbous swelling of the tip of the finger or toe.

The posture of the patients may be active (initial stage, period of stable remission) or observed the forced posture in form of orthopnea (spasm of bronchi, decreasing the breath surface).

The color of the skin is characterized by central or diffuse cyanosis due to the accumulation of the carbon dioxide and reduced restored hemoglobin.

Inspection of the chest may reveal barrel-like (emphysematous) form of the chest with protruded supra- and subclavicular fosses, horizontal direction of the ribs, smoothed and narrow intercostals spaces, increased anteroposterior diameter. As usual the chest is symmetrical, the type of respiration is mixed or thoracic, accessory respiratory muscles active participate in the breathing act. In patient may be

observed tachypnea with shallow respiration depth and poor movement of the chest on affected side.

Palpation of the chest. The chest is painless, elastic or rigid, vocal fremitus is increased on the affected side.

Percussion of the lungs. In comparative percussion of the lungs may be detecting intermediate pulmonary sound; in cause of emphysema – generalized hyperresonance (bandbox sound). In topographic percussion of the lungs is observed unilateral or bilateral lowering of the lower lungs edges (in cause of compensatory pulmonary emphysema), respiratory mobility of the lower borders of the lungs on the affected side decreased.

Auscultation of the lungs. In auscultation of the lungs over the pathologically increased vesicular breathing identify different moist rales decreased after cough and sputum discharge.

In developing of bronchium obstruction over the bronchovesicular or pathological bronchial breathing, a lot of sibilant and sonorous dry rales are detected.

Cardiovascular system. Accordanly to chronic hypoxia and intoxication develops myocardial dystrophy that manifests by tachycardia, palpitation, arrhythmias and decreased loudness of heart sounds.

Complications. Bronchospastic syndrome, lung emphysema, respiratory failure, "cor pulmonale", lung bleeding, renal amiloidosis, metastatic brain abscesses.

Additional methods of examination

Clinical blood analysis: leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated ESR, in prolonged duration – hypo- or normochromic anemia.

Sputum analysis: large amount of mucopurulant, purulent or mucopurulent bloody strong smell sputum that form lays on standing (pus, plasma and upward mucus). In microscopic study are revealed a lot of columns ciliated epithelium, leucocytes, alveolar macrophages, fibrin fibers and large quantity of microorganisms (bacterial flora).

X-ray examination (Roentgenography in two projections): augmentation and deformity of lung picture with character in lower regions, thick wall cavities sometimes with fluid level, lung's "amputation", increased in transparent regions with healthy lung tissue.

Test of ventilatory function (spirometric recording and pneumotachymetry): impaired ventilatory function: restrictive type – significant decrease of total lung capacity; obstructive type – decrease of respiratory reserve and forced expiratory vital capacity or mixed type – significant decrease of total lung capacity and forced expiratory vital capacity.

ECG: deviation of electric axis of the heart to the right, P-pulmonale in II, III, AVF leads.

Bronchial asthma

Bronchial asthma – chronic inflammatory pathology of respiratory tract that caused by significant amount of inflammatory cells, mediators and leads to bronchi hyperreactivity. Disease is manifested by transitory noise and difficult breathing, dyspnea, bronchial asthma attacks, coughing particularly at night and in the morning hours.

In the base of the disease lays chronic inflammatory process in bronchi due to the bronchi smooth muscles spasm (acute obstruction), mucus edema (subacute obstruction) and bronchi obstruction by tenacious secret (chronic obstruction). On longterm duration of disease via fibrosis in bronchial wall develops sclerotic obstruction (Fig. 1.11).

Etiology

I. The risk factors:

- genetic factors;
- atopia (ability of the organism to the increased production of IgE owing to the allergens);
- bronchi hyperreactivity.

II. The cause factors:

- allergens;
- endogenous factors;
- impaired arachidonic acid metabolism;
- bronchi hyper reactivity to physical load;
- nervous and psychological factors;
- dyshormonal state.

III. The initiate factors:

- respiratory infections;
- airs pollutants;
- smoking.

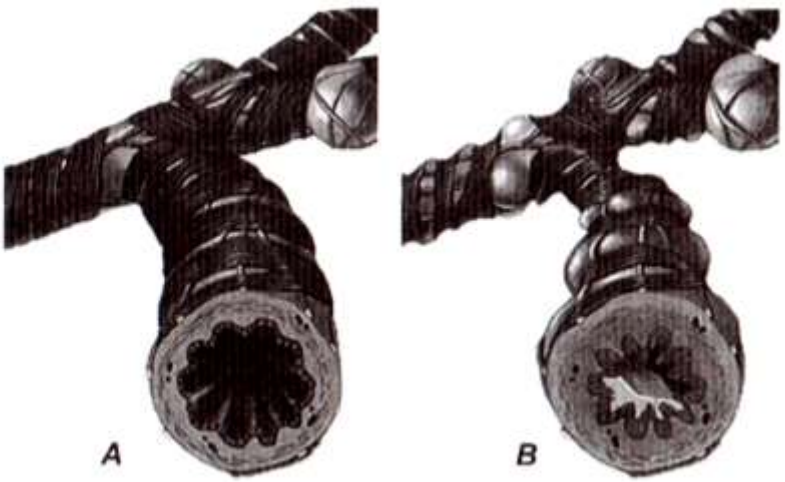
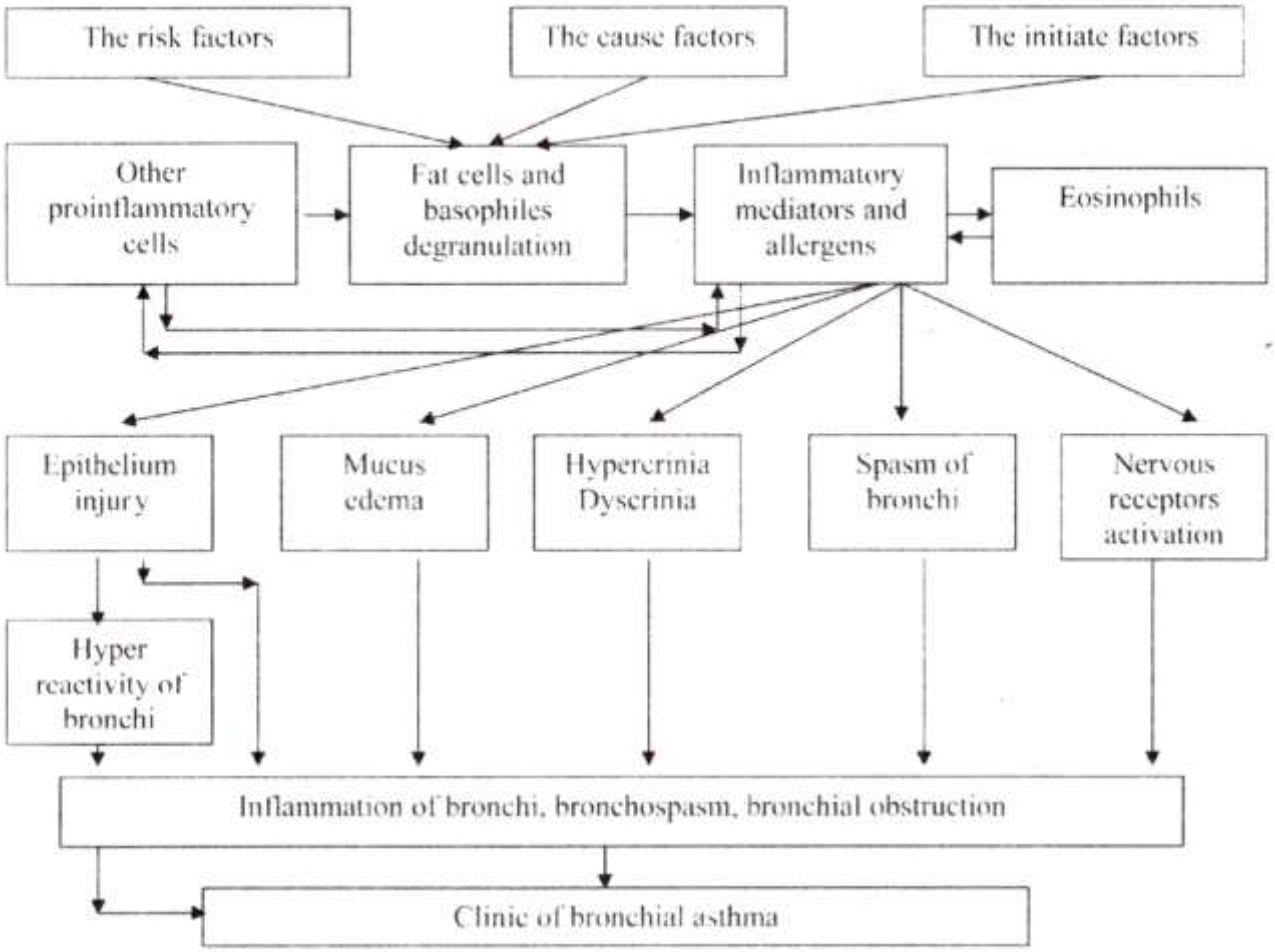


Fig. 1.11. ***A.*** Normal bronchus opening. ***B.*** Bronchus opening during pathology: hypersecretion, dyscrinia, bronchial hyperactivity.

Pathogenesis



Classification

Bronchial asthma is classificated according to the complex of clinical and functional signs of bronchial obstruction.

Degree I. Intermittent bronchial asthma characterized by:

- short-term symptoms less than 1 time per weak;
- short-term periods of aggravation (from several hours to several days);
- night symptoms of asthma appear not frequent than 2 times per month;
- normal function of the lung between aggravations;
- forced expiratory vital capacity during first second (FEV_1) $\geq$ 80 % from normal;
- 24-hour deviation of FEV_1 < 20 %.

Degree II. Easy persistent bronchial asthma characterized by:

- the symptoms frequent than 1 time per weak but less than 1 time per day;
- aggravations of disease can impair activity and sleeping;
- night symptoms of asthma appear more frequent than 2 times per month;
- FEV_1 $\geq$ 80 % from normal;
- 24-hour deviation of FEV_1 – 20–30 %.

Degree III. Moderate gravity persistent bronchial asthma characterized by:

- the symptoms appear every day;
- aggravations of disease impair activity and sleeping;
- every day using of short-term β_2-agonists;
- FEV_1 – 60–80 % from normal;
- 24-hour deviation of FEV_1 > 30 %.

Degree IV. Grave bronchi persistent asthma characterized by:

- the constant symptoms of bronchial asthma;
- frequent aggravations;
- frequent night symptoms of asthma;
- limitation of physical activity via asthma;
- FEV_1 < 60 % from normal;
- 24-hour deviation of FEV_1 > 30 %.

Classification of the bronchial asthma aggravations (according to the anamnesis, intensity of the clinical signs, respiratory and cardiovascular dysfunction):

Degree I – effortless;
Degree II – moderate grave;
Degree III – grave;
Degree IV – risk of breathing stop;

Symptoms	Effortless	Moderate grave	Grave	Risk of breathing stop
Dyspnoea	At walking	At speaking	At rest	–
Conversation	Sentences	Phrases	Words	–
Consciousness	Normal	Exiting	Exiting	Deranged
Breathing rate	Increase	Increase	>30/min	–
Participation of the additional muscles	Absent	Present	Present	Paradox thoracoabdominal breathing
Whistling breathing	At the end of expiration	Loud	Loud	Absent
Pulse/min.	<100	100–120	>120	Bradycardia
FEV_1 after taking broncholytic, % from normal level	> 80 %	60–80 %	< 60 %	Absent
PaO_2	Normal	>60 mm Hg	<60 mm Hg	–
$PaCO_2$	<45 mm Hg	<45 mm Hg	>45 mm Hg	–

Clinical features

The main complaints in patients with bronchial asthma are bronchial asthma attacks: dyspnea, asphyxia, episodic breathlessness and cough. In attacks development there are divide 3 periods: prodromal, manifestation, reverse.

I. The prodromal period: starts at several minutes, hours or sometimes days before asthma attack and characterized by sneezing, itchiness of the skin and eyes, hypersecretion from nose, paroxysmal coughing, breathlessness, headache, weakness and changes of mood.

II. The period of clinical manifestation (bronchial asthma attack): appears feeling of difficult breathing, significant dyspnea (expiratory type) with changes in respiratory rate (tachypnea), depth (shallow respiration) and noisy distant rales. *General patients condition* is from middle grave to extremely grave. Due to the acute hypoxia may be observed depressed or exited deranged consciousness. During asthma attack the patients take the *forced posture* in form of orthopnea – sitting position fixing the shoulder girdle in order to reduce dyspnea. *The color of the skin* is pale with central or diffuse cyanosis. The form of the chest is emphysematous with accessory muscles participate in the breathing act, observed decreased excursion of the chest. The vocal fremitus is badly transmitted and generalized bandbox sound assessed over the lungs during percussion. *Auscultative data* are characterized by sibilant and sonorous dry rales over the pathologically decreased vesicular breathing.

III. The period of asthma attack reverse: the duration of attack is differing and its final may come quickly without any complications through sputum discharge; or may continue for several hours or days accompanied by permanent dyspnea, headache and weakness.

In severe causes bronchial asthma attacks may transform at asthmatic status – lingering bronchial asthma attack that characterized by shallow quick respiration (significant tachypnea), constant dyspnea and formation of "dumb lung". Severity of asthmatic status is characterized by degree of respiratory failure, acidosis, hypercapnia, level of hypoxemic coma and respiratory center paralysis.

In period of stable remission the general patients condition commonly satisfactory or middle grave, however the clinical signs of emphysema are stay be present, particularly in causes of long disease duration and recurrently asthma attacks.

Additional methods of examination

Clinical blood analysis: secondary erythrocytosis; eosinophilia, accelerated ESR.

Sputum analysis: the character of the sputum is mucous, tenacious or tenacious thick consistency, glass-like color and odorless. In microscopic study are revealed columns ciliated epithelium, leucocytes, alveolar macrophages, eosinophils, Charcot-Leyden crystals and Kurshman spirals.

X-ray examination: in initial stages the specific data are absent. During asthma attack and according to the repeatedly periods of progression assess transparent lung tissue, horizontal position of the ribs, dilation of the intercostals spaces, low diaphragm position. In cause of inflammatory and allergic etiology of bronchial asthma observed augment and deformity of lung picture.

Test of ventilatory function (spirometric recording and pneumotachymetry): assess decreased respiratory reserve (75 % of maximum lung ventilation and lower), and decreased Votchal-Tiffeneau index.

Emphysema of the lungs

Emphysema of the lungs – lungs disease characterized by pathologic alveoli dilation localized in terminal bronchi and accompanied by destructive changes of alveolar walls.

Depending on the etiology and mechanism there are the next forms of lungs emphysema:

– primary (idiopathic) emphysema – develops in non-injured lungs where bronchial obstruction is complication;

– secondary emphysema – based on the previous lungs diseases (chronic obstructive bronchitis) and complicated their duration.

Pathogenesis

– progressive decline of the lung surface due to the destruction of interalveolar septum that leads to decrease lungs capacity and respiratory failure;

– changes of the lung tissue mechanical characteristics lead to the secondary bronchial obstruction via small bronchi collapse on inspiration. Afterward large bullae compressed and impaired ventilation of as well functional pulmonary tissue and augment respiratory failure.

Clinical features

The main complaints in patients with lung emphysema are dyspnea and cough.

Dyspnea – in the initial stage appears only during physical activity than takes permanent disposition, has expiratory or mixed character.

Cough – commonly dry, has reflectivity character and appears previously to dyspnea manifistation.

Objective examination. *General patient's condition* may be from middle grave (early stage of the disease) to grave.

The posture of the patients is frequently active or may be forced (orthopnea).

The color of the skin and visible mucosa is characterized by diffuse cyanosis. In patients with secondary emphysema due to significant hypercapnia occurs blue tint of the

skin. Loss of weight depends on large energy expenditure that need for intensive muscles work: hyperfunction of additional respiratory muscles and abdominal muscles.

In inspection of the chest reveal barrel-like (emphysematous) form of the chest with protruded supra- and subclavicular fosses, horizontal direction of the ribs, smoothed and narrow intercostals spaces, increased anteroposterior diameter. As usual the chest is symmetrical, the type of respiration is mixed or thoracic with active participation of accessory muscles in the breathing and restricted chest movement.

In palpation the chest is painless and rigid. Vocal fremitus is badly transmitted.

In comparative percussion of the lungs generalized hyperresonance (bandbox sound) may be heard over the hyper inflated lungs of emphysema.

In topographic percussion of the lungs is observed bilateral lowering of the lower lungs edges, respiratory mobility of the lower borders of the lungs is decreased.

In auscultation of the lungs observed pathologically decreased vesicular breathing, the rales over the pathologically increased vesicular breathing observed only in case of chronic bronchitis.

Additional methods of examination

Clinical blood analysis: secondary erythrocytosis and increased hemoglobin level.

X-ray examination: the signs of increased airiness of the pulmonary tissue, poor bronchial picture in the peripheral zones, low diaphragm's position and its badly excursion and increased retrosternal area.

Test of ventilatory function: the signs of primary emphysema (decrease of respiratory volume and increased of residual air volume); after the bronchial obstruction development (decrease of respiratory reserve and forced expiratory vital capacity and Votchal-Tiffeneau index).

1. Computed tomography. The signs of increased airiness of the pulmonary tissue, poor bronchial picture in the peripheral zones and presence of bullae are revealed. In cause of chronic bronchitis is determined condensation of the bronchial wall and infiltration along bronchi.

ECG: deviation of electric axis of the heart to the right, P-pulmonale in II, III, AVF leads.

Pneumonia

Pneumonia – acute inflammatory lung disease with obligatory alveoli involment and exudative formation in them.

Classification

1. According to the particularities of infection.

- nonhospital pneumonia;

– pneumonia in outpatients;
– pneumonia in inpatients;
– intrahospital pneumonia;
– asperities pneumonia;
– pneumonia in persons with severe immune deficiency.

II. The category of the patients with nonhospital pneumonia:

1 category – pneumonia in patients without associated pathology and other modified factor;

2 category – pneumonia in patients with associated pathology and or other modified factor;

3 category – pneumonia that needs hospitalization (without intensive treatment);

4 category – severe pneumonia that needs intensive treatment (reanimation).

III. The groups with intrahospital pneumonia:

1 group (A) – patients with mild or moderate pneumonia severity (without risk factors) that develops in different period of hospitalization or grave pneumonia with early manifestation (less than 5 days of hospitalization);

2 group (B) – patients with slight or moderate pneumonia severity (with specific risk factors) that develops in different period of hospitalization or grave pneumonia with early manifestation (less than 5 days of hospitalization);

3 group – patients with grave pneumonia in presence of risk factors or pneumonia with late manifestation (more than 5 days of hospitalization).

Nonhospital pneumonia means pneumonia that develops outside from hospital (in conditions of life).

Intrahospital pneumonia means pneumonia that develops in first 48–72 hours after hospitalization in condition of reject infectious in incubation period on the moment of admission to the hospital.

The main risk factors

– smoking;
– taking of alcohol;
– heart failure with congestion in lesser circulation;
– chronic obstructive lung diseases;
– influence of toxic ecologic and professional factors;
– innate defects of bronchopulmonary system;
– chronic infection in nosepharynx;
– the state of immunodeficiency and treatment with immune depressants;
– the status after operation;
– general exhaustion;
– long confinement to bed;
– old age.

The main pathogenic links:

- entrancing of the pathologic agent to the pulmonary tissue;
- impaired local bronchopulmonary resistance;
- development of the local inflammatory process and its overspreading in lung tissue;
- sensebilization advance to infectious agents and input of proinflammatory reactions;
- impaired microcirculation;
- activation of oxidative stress and proteolysis in lung tissue;
- antibody and immune complexes formation.

Clinical features

The appearance of clinical features depends on the pathologic process spreading. There are distinguishing focal and lobar pneumonias. The main complaints in patients with acute pneumonia are hyperthermia, cough, pain in the chest, dyspnea and complaints connecting with general intoxication (general weakness, lost of ability to work, perspiration, loss of appetite, headache, pain in the muscles, joints; in severe cases – deranged consciousness).

Focal pneumonia starts gradually, commonly after respiratory viral infection, the temperature rise to 38,0–38,5 °C during 1–3 days.

Lobar pneumonia (pleuropneumonia) has acute onset, suddenly appears intensive pain in the chest, the temperature rise to 39,0 °C and more for 4–5 days.

Cough – the specific symptom of pneumonia; in the initial stage dry and has reflectivity character, from the second day appears hardly expectorated tenacious sputum. In patients with lobar pneumonia the sputum is sticky and "rusty" initially, later mucous-purulent.

Pain in the chest – occurs in lobar pneumonia in condition of pleura involvement or intercostals nervous irritation. The pain occurs suddenly, intensive and increase during deep inspiration or cough.

Dyspnea – inspiratory or mixed character and depends on pneumonia duration. In lobar pneumonia significant dyspnea accompanied with the feeling of the heaviness in the chest and tachypnea (30–40 breathing acts per minute).

Objective examination. *General patient's condition* may be from middle grave (acute focal pneumonia) to extremely grave (lobar acute pleuropneumonia).

The posture of the patients is frequently active or may be forced (orthopnea).

The color of the skin and visible mucosa is pale; in case of respiratory failure observe diffuse cyanosis. In general examination is specific presence of "face pneumonica" – one-side blush on the same cheek as affected lung, cyanosis, often herpes on the lips and nose (herpes lobar, nasalis).

In inspection occur objective signs of dyspnea – tachypnea, participation of the accessory muscles in the breathing act or even mixed type of respiration. As usual the chest is symmetrical, on dynamic examination may be detect poor chest expansion on the affected side.

In palpation the chest may be painful and rigid, vocal fremitus is increased on the affected side (in focal pneumonia such signs aren't specific).

In comparative percussion of the lungs dull sound is heard over the solid pulmonary tissue. Moreover, lobar pneumonia characterized by stage-depend changes: in initial and recovery stages detect intermediate percussion sound due to the decreased amount of air in alveoli, in consolidation stage – the sound becomes dull due to the alveoli's filled with exudates.

In topographic percussion of the lungs as usual lungs edges have normal characteristics, respiratory mobility of the lower borders of the lungs is decreased on the affected side.

In auscultation of the lungs there are distinguish the pathological changes accordantly to disease's stages. In patients with lobar pneumonia: in initial stage over the region with decreased vesicular breathing detect indux crepitation (quit, remote sound); in consolidation stage – over the region with bronchial breathing detect fine and medium bubbling rales; in recovery stage due to the resolution of exudates over the region with decreased vesicular breathing detect redux crepitation (loud, crackling sound).

In focal pneumonia over the local decreased vesicular breathing detect crepitation.

In case of pleural affection (pleuropneumonia) over the affected region heard pleural friction sound.

Additional methods of examination

Clinical blood analysis: leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated ESR.

Sputum analysis: the character of the sputum depends on the pathologic process spreading: in focal pneumonia the sputum is mucopurulant, tenacious or tenacious thick consistency, glass-like with yellow traces color, odorless. In microscopic study are revealed a lot of columns ciliated epithelium, leucocytes, alveolar macrophages.

In lobar pneumonia the sputum is sticky, rusty initially, later mucopurulant, tenacious or tenacious thick consistency, odorless. In macroscopic study are revealed fibrin clots, changed blood; in microscopic study – columns ciliated epithelium, leucocytes, erythrocytes, hematoidin crystals, alveolar macrophages and microorganisms (bacterial flora).

X-ray examination: in focal pneumonia – the signs of focal pulmonary tissue consolidation (darkening limited by the lung's segment); in lobar pneumonia – the signs of intensive homogenous pulmonary tissue consolidation (darkening limited by the lung's lobe or several lobes).

Pleuritis

Pleuritis – injury of pleura associated with fibrin formation on its surface (dry pleuritis) or fluid accumulation in pleural cavity (exudative pleuritis).

Dry pleurisy

Dry pleurisy (adhesive, fibrinous) is the pathology of the respiratory system that characterized by bands and commissures formation between pleural layers and increase of their thickness due to the inflammation.

Etiology

- infection (tuberculosis, bacterial infection, fungus, viral infection);
- dissemination of the tumor cells to pleura;
- reactive pleuritis (uremia);
- dehydrotation (profuse bleeding, vomiting, diarrhea).

Pathogenesis

- dilation of lymphatic capillaries;
- increased vessels penetration;
- pleural inflammation;
- pleural infiltration;
- fibrin accumulation on visceral and parietal pleura;
- fibrosis development;
- anatomic and functional block of resorbtion apparatus;

Clinical features

Intensity of clinical features depends on the pathologic process spreading. The main complaints in patients with dry pleurisy are: cough, pain in the chest and dyspnea.

Cough – most commonly dry and has reflectivity character.

Pain in the chest – connecting with pleura injury, occurs suddenly on the affected side, intensive and increases during deep inspiration or coughing.

Dyspnea – intensity depends on process spreading.

Objective examination. *General patient's condition* may be from middle grave to grave.

The posture of the patients is forced (lie on the affected side in order to relieve the pain).

The color of the skin and visible mucosa is without changes.

In inspection occur superficial, rapid breathing (via intensive pain); participation of the accessory respiratory muscles in the breathing act or even mixed type of

respiration. In static inspection as usual the chest is symmetrical, on dynamic – detect poor movement of the chest expansion on one side.

In palpation the chest is painful on the damage side, elasticity is saved, vocal fremitus is equal transmitted.

In comparative percussion of the lungs may be observed dull sound over pathological region.

In topographic percussion of the lungs the normal lower borders are revealed, respiratory mobility of the lower border on the affected side is decreased.

In auscultation of the lungs over the region with decreased vesicular breathing detect pleural friction sound.

Additional methods of examination

***Clinical blood analysis**:* leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated ESR.

X-ray examination: – the signs of pleura injury and fibrin deposition.

Exudative pleurisy

Exudative pleurisy – pathology of the respiratory system that characterized by pleural layers inflammation and fluid accumulation in the pleural cavity. As usually pleura injury is secondary to the main pathologic process and may accompanied almost of 80 % of diseases.

Etiology

- infection (tuberculosis, bacterial infection, fungus, viral infection);
- dissemination of the tumor cells to pleura;
- allergic and autoimmune pleurisy;
- pleurisy in diffuse connective tissue pathology;
- posttraumatic pleurisy.

Pathogenesis

- direct pleura injury (trauma, operation, tumor, infection through lymph or blood);
- contact way of process spreading;
- infection and allergic mechanism;
- inflammatory exudation to the pleural cavity;
- impaired lymph and blood circulation;
- oncotic pressure disturbance;
- impaired resorbtion;
- fluid accumulation in pleural cavity.

Clinical features

Intensity of clinical features depends on the pathologic process spreading, etiology, amount and character of exudates. The main complaints in patients with exudative pleurisy are: cough, dyspnea, pain and feeling of heaviness in the chest, supplementary – general weakness, hyperthemia, loss of appetite and perspiration.

Cough – most commonly in initial stage dry and has reflectivity character, along disease progression becomes moist.

Pain in the chest – one of the fist symptoms and connecting with pleura injury, may be different in its intensity (from moderate to acute) and increases during deep inspiration or coughing. In cause of diaphragmatic pleurisy localization the pain can irradiate to the upper abdominal region or via the n. diaphragmatic to the neck. For the period of exudates volume intensity the pain becomes duller but dyspnoea increase.

Dyspnea – has mixed character and its intensity depends on the exudates volume and speed of its accumulation, degree of affected lung ventilation via compression by fluid and mediastenum organs displacement.

Objective examination. *General patient's condition* may be from middle grave to grave.

The posture of the patients is forced (lie on the affected side in order to revile the pain).

The color of the skin and visible mucosa are characterized by diffuse cyanosis. In case of mediastenum fluid localization observed edema of the face and neck, dysphagia and voice changes.

In inspection observe superficial, rapid breathing (via intensive pain); mixed type of dyspnea. In static inspection as usual the chest is asymmetrical, on dynamic – detect poor movement of the chest expansion on the affected side.

In palpation the chest is painful, rigid with badly vocal fremitus transmission on the damaged side.

In comparative percussion of the lungs detect dull sound over the pathological region.

In topographic percussion of the lungs the lower edge on the affected side is elevated, respiratory mobility is increased. In large exudates amount over the lung there are 5 clinical-diagnostic zones (for more detail information seen syndrome of fluid accumulation in pleural cavity).

In auscultation of the lungs in the initial stage on the affected side over the region with decreased vesicular breathing detect pleural friction sound. In large exudates amount according to the five clinical-diagnostic zones there are distinguished: over exudates – the zone with significant decreased vesicular breathing or full absent of breathing sounds; over consolidate pulmonary tissue – the zone with

pathological bronchial breathing; over the free from fluid and healthy side – the zone with increased vesicular breathing.

Additional examination

Clinical blood analysis: leukocytosis, neutrophilia, shift of leukocyte formula to the left, accelerated ESR.

X-ray examination: – the signs of pleura affection, significant darkness with slanting upper border of the fluid and dislocation of mediastenum to the healthy side.

Pleural fluid analysis includes: assessment of macroscopic characteristics (character, transparency, color, consistency, odor, relative density); chemical study (protein, Rivalts's reaction); microscopic study (cellular composition); bacterioscopic study.

Chapter 9

CARDIOVASCULAR SYSTEM

Syndrome of cardiovascular failure

Heart failure is a pathological condition which characterized by decreased contractility of the myocardium, reduction of cardiac output in which the cardiovascular system fails to supply the necessary amount of blood to the organs and tissues for their adequate function.

This condition arises due to the affection of the heart or of the vessels, or it may be combined disorders of the cardiovascular system.

The syndrome of cardiovascular failure is divided into 2 groups:

– heart failure: acute (acute left ventricular heart failure, acute left atrial heart failure, acute right ventricular heart failure) and chronic (chronic left ventricular heart failure, chronic left atrial heart failure, chronic right ventricular heart failure and total chronic heart failure);

– vascular failure: syncope, collapse and shock.

Etiology

Heart failure may develop in case of overloading or overstrain of the myocardium by "pressure" (hypertension, aortic stenosis, stenosis of the pulmonary orifice), by "volume" (mitral or aortic regurgitation), and also owing to diseases affect primarily the myocardium and its metabolism. Affection of the myocardium may be due to infectious, inflammatory and toxic damage of the myocardium (myocarditis, cardiomyopathy, intoxication of the myocardium by alcohol, narcotic drugs, other poisons), insufficient blood supply to the myocardium (disordered coronary circulation, anemia), metabolic disorders, endocrine dysfunction.

Common causes of heart failure:

– ischemic (coronary) heart disease;
– arterial hypertension;
– dilated cardiomyopathy;
– heart valve diseases;
– hypertrophic cardiomyopathy;
– restrictive cardiomyopathy;
– constrictive pericarditis;
– high-output heart failure:

a) chronic anemia;
b) atrioventricular shunts;
c) thyrotoxicosis.

Pathogenesis

The clinical syndrome related to organ hypoperfusion and inadequate tissue oxygen delivery due to a low cardiac output and decreased cardiac reserve, as well as pulmonary and systemic venous congestion. Heart failure is associated with complex neurohormonal changes including activation of the renin-angiotensin-aldosterone and the sympathetic nervous systems. At first these changes may help to compensate cardiac function by altering the afterload or preload and by increasing myocardial contractility. Ultimately they become counterproductive and reduce cardiac output by causing an inappropriate and excessive increase in peripheral vascular resistance. A vicious cycle may be established because a fall in cardiac output will cause further neurohormonal activation and increasing peripheral vascular resistance. The onset of peripheral edema is due to salt and water retention caused by impaired renal perfusion and secondary aldosteronism.

Heart failure may develop as a result of impaired myocardial contraction due to decreasing of number functional activity of cardiomyocites – *systolic dysfunction*, which may observe in patients with inflammation of myocardium, cardiosclerosis, hypertension, non-compensated regurgitation and dilated cardiomyopathy.

Heart failure may arise due to poor ventricular filling caused by disorder of active relaxation and increasing of rigidity of myocardium due to hypertrophy, fibrosis and infiltration – *diastolic dysfunction*, which may observe in patients with constrictive pericardities, hypertrophic cardiomyopathy.

Systolic and diastolic dysfunctions often coexist, particularly in patients with hypertension, ischemic (coronary) heart disease.

Compensatory mechanisms in heart failure:
- tachycardia;
- Frank-Starling's mechanism;
- myocardial hypertrophy;
- tonogenic dilation;
- slow blood flow.

Clinical features

Symptoms and signs of heart failure depend on the prevalence of affected heart chambers. Left ventricular failure corresponds with reduction of the ventricular output and increasing pressure in the left atrium, pulmonary veins and later pulmonary artery. There are clinical picture of congestion in lesser circulation: breathlessness, paroxysmal nocturnal dyspnea, cough, sometimes hemoptysis, orthopnea, cyanosis and crepitation over the lung.

In patients with right ventricular failure due to the reduction of the ventricular output appear the clinical pictures of congestion in greater circulation: pain in the right hypochondrium, swollen jugular veins, edema on lower extremities, enlarged liver. Massive accumulation of fluid may cause ascites, pleural and/or pericardial effusion.

Total heart failure: failure of the left and right heart may develop because the disease process affects both ventricles, or because there is primary affection left heart failure with dilation of left atrium, pulmonary hypertension and as a result subsequently development of right heart failure.

The patient's complaints are fatigue, dyspnea, malaise, edema of legs, the attacks of breathlessness, cough. The general patients condition as usual grave, deranged consciousness, forced posture – orthopnea, cyanosis, anasarca.

Chronic heart failure is sometimes associated with marked weight loss (cardiac cachexia) caused by a combination of anorexia and impaired absorption due to gastrointestinal congestion, poor tissue perfusion due to a low cardiac output and skeletal muscle atrophy due to immobility.

Poor renal perfusion may lead to oliguria and uremia.

Additional methods of examination

Clinical blood analysis is required in order to reveal inflammatory process, anemia.

Clinical urine analysis is required for estimation kidney pathology.

Biochemical blood analysis – creatinin, urine acid, total protein levels, potassium, calcium concentration.

ECG is required for detection of main cardiac process.

X-ray examination in order to estimate lung and heart pathology.

Echo-CG is required for estimation of ejection fraction; structural and functional state of heart.

Echo-stress test with dobutamin.

Measurment of pressure in ventricle:

– end-diastolic pressure in right ventricle using the catheterization of vena cava superior;

– end-diastolic pressure in left ventricle using the Swan-Ganz catheter in pulmonary artery.

Acute heart failure

Acute left ventricular failure

Acute left ventricular failure – is state resulted from suddenly sharp decreased contractility of left ventricle and normal one of right ventricle.

Causes acute left ventricular failure:
- essential hypertension, espessially hypertensive crisis;
- heart valve diseases (aortic regurgitations, aortic stenosis);
- ischemic (coronary) heart disease;
- myocardial infarction;
- myocarditis;
- arrhythmias.

Pathogenesis

Acute heart failure may be provoked by infections, intoxications, physical and nervous strain. The lesser circulation becomes overfilled with blood because during a sharply decreased contractility of the left ventricle the right ventricle continues working normal to pump the blood from the greater circulation to the lesser one: arterial blood pressure increases in pulmonary veins and capillaries, its permeability increases, gas exchange is impaired. If congestion in the lesser circulation progresses, hydrostatic pressure in capillaries is equal or higher than oncotic pressure (25–30 mm Hg). The blood plasma pass from the overfilled pulmonary capillaries to the alveoli and accumulates in the respiratory ducts, pulmonary edema develops. Interstitial edema means the thickening of the alveoli walls due to accumulation of the blood plasma; alveolar edema means the presence of the blood plasma in the alveoli space.

There are two clinical forms of acute left ventricular failure: cardiac asthma and pulmonary edema.

Cardiac asthma

Clinical features

The patient complains on severe dyspnea as attack, with more difficulty inspiration, which often arises during night sleep, so called cardiac asthma. May be dry cough or with expectoration of small amount the tenacious sputum. The patient complains on marked weakness, feeling of fear, excitement.

Objective examination. General patient's condition is from moderate grave to extremely grave. Consciousness is clear but if this state lasts a long time, may be cloudiness.

Posture*:* patient assumes a forced position – sitting with legs hanging down from the bed or he stands up.

The skin becomes pallid and cyanotic, acrocyanosis, cold sweat appears.

Examination of the respiratory system. The accessory muscles take part in the breating, tachypnea – 30–40 per minute. Over the low regions of the both sides of lungs the vocal fremicus is increased. Over the lungs is revealed dull-tympanic sound. Harsh respiration and crepitation in the posterior part of the lungs are heard.

Examination of the cardiovascular system. The apex beat displaced to the left and cardiac dullness and configuration of the heart depends on main pathology. The heart sounds are decreased at the apex, accentuated second heart sound over the pulmonary artery, tachycardia, gallop rhythm. Pulse is frequent, arrhythmia, blood pressure may be normal, or decreased.

In case of appropriate treatment may disappear the clinical picture of cardiac asthma. In some case this state transforms in pulmonary edema.

Pulmonary edema

Patient complains on severe breathlessness, cough with expectoration of sputum, heaviness in the heart.

Objective examination. The general patient's condition is extremely grave, deranged consciousness, disorder of the mental function, forced posture – sitting with trunk slightly bent forward, the skin pall, cyanosis with grey tint, cold sweet.

Respiration becomes rattling and heard even at the distance. Ample foaming sputum with traces of blood (pink or red) is expectorated.

In percussion over the lungs in the posterior inferior parts of the chest – dull sound.

In auscultation harsh respiration, moist rails of various calibers are heard over entire surface of the lungs. The heart sounds are weakened, protodiastolic gallop rhythm, tachycardia.

Pulse – small frequent pulse, alternative, with poor filling.

Blood pressure – decreased.

If patient is not treated urgently, this attack can lead to death.

Additional methods of examination

ECG: hypertrophy of the left ventricle, signs of overloading of the left ventricle.

X-ray examination: congestion changes in lungs.

Echo-CG: hypertrophy of the interventricular septum and the back wall of the left ventricle, decrease of contractility of the myocardium, increase end-systolic end-diastolic dimensions of the left ventricle.

Acute left atrial heart failure

The syndrome of acute left atrial failure develops in patients with mitral stenosis, myxoma of the left atrium (in mechanical obstruction of the intracardiac blood flow) in markedly weakened contractility of the left atrium and normal function of the right ventricle, which continues pumping blood into the lesser circulation. Clini-

cally acute left atrial failure manifested by cardiac asthma and pulmonary edema may resemble acute left ventricular heart failure.

Acute right ventricular heart failure

Acute right ventricular heart failure resulted from the sharply suddenly limitation of the lung's surface from the breathing and consequently development of the congestion in greater circulation.

Etiology

- thromboembolism of the trunk of the pulmonary artery or its branches;
- spontaneous pneumothorax;
- bronchial asthma (status asthmaticus);
- lobar pneumonia;
- lung atelectasis;
- rupture of the aorta aneurism into the pulmonary artery.

Pathogenesis

In thromboembolism of the pulmonary artery or its branches occurs mechanical occlusion of vessel lumen and complete or incomplete blood supply to corresponding part of lung. In case of pulmonary disease develops reduction of blood flow. All these disorders lead to hemodynamic disturbances at first in pulmonary circulation, later intracardial and systemic circulation. As a response to decrease of arterial amount of blood occurs the resistance of pulmonary blood flow. The right ventricular heart failure with dilation of the chamber is developed. Increasing of end diastolic pressure at right ventricle, right atrium and central veins are observed. The features of congestion in greater circulation appear.

Clinical features

Clinical features of acute right ventricular heart failure include symptoms and signs due to common process and presence of complication in a form of acute pulmonary heart.

The main complaints according to leading process: severe acute pain in the chest, cough, hemoptysis in patient with thromboembolism of pulmonary artery. The general condition is extremely grave, deranged consciousness, diffuse cyanosis, pallid skin, cold sweat and dyspnea. Acute pulmonary heart is characterized by complaints on the heaviness in the right hypochondrium and edema at the low extremities.

The objective examination of respiratory, cardiovascular systems reveals the clinical features of main pathology: infarction of the lung, pneumonia, pneumothorax, atelectasis.

The neck veins become swollen, later develops edema of legs. Epigastric pulsation and pulsation left to the sternum are determined as a result of dilation of right ventricle. Right border of the relative heart dullness displaced to the right from the sternum, the heart sound are diminished, splitting of the second sound over the pulmonary artery, tachycardia, gallop rhythm. The pulse is small and frequent. Blood pressure: hypotension.

In palpation of abdomen the enlarged liver is detected.

Additional methods of examination

ECG: in II, III standard leads – P-pulmonale, in $V_{1,2}$ – signs of overloading of the right ventricle.

Chronic heart failure

Chronic left ventricular heart failure

Chronic left ventricular heart failure is characterized by congestion in lesser circulation due to the decreasing contractility of the left ventricle.

Etiology

- hypertension;
- symptomatic hypertension;
- ischemic heart disease;
- aortic valve disease;
- atherosclerotic cardiosclerosis;
- postinfarction cardiosclerosis;
- cardiomyopathy.

Pathogenesis

Etiological factor is development of prolonged venous congestion in the lesser circulation which stimulates growth of connective tissue in the lungs and sclerosis of the vessels. At the increased pulmonary capillary pressure the elastic ability of lungs is decreased, disorders of ventilation occur. The respiratory centre is stimulated by accumulating under-oxidized metabolites in the blood – lactic acid, bicarbonate alkalis, and carbon dioxide. Relevant to increasing content of the reduced hemoglobin in capillary blood appear acrocyanosis. As a compensatory mechanism may be tachycardia.

Clinical features

The patients complain on weakness, decreased work capacity, breathlessness, cough, firstly dry, later with sputum.

Objective examination. Patient's condition and consciousness is defined by stage of pathological process. Posture is forced in patients with pronounced heart failure. As usually the orthopnea is observed. The skin is pale and cold, acrocyanosis, trophic disturbances as a rule on the legs.

Objective examination of the respiratory system. The hemoptysis is observed, as a result of rupture of bronchial overfilled arterioles. Tachypnea is determined. At night time may be the attacks of cardiac asthma, transformed in pulmonary edema. Intermediate sound is detected over the lower lobes of the lung. In auscultation decreased vesicular breathing and moist rales in the posterior-inferior parts on the lung are determined.

Objective examination of the cardiovascular system. Apex beat is displaced to the left. The left border of the relative cardiac dullness displaced to the left. The both heart sounds decreased at the apex. Second sound over the pulmonary artery is accentuated.

Additional methods of examination

ECG – hypertrophy of left ventricle, left bundle branch block.
X-ray examination – congestion in lung.
Echocardiography – hypertrophy of left ventricle.

Chronic left atrial heart failure

Chronic left atrial heart failure is due to overloading of left atrium mostly in case of mitral stenosis and mitral regurgitation in late stage. The clinical features resemble the chronic left ventricular heart failure.

Chronic right ventricular heart failure

Etiology

- venous congestion in lungs (chronic left ventricular failure);
- chronic lung diseases;
- congenital heart defects;
- tricuspid regurgitation;
- stenosis of pulmonary valve orifice;
- kyphoscoliotic chest.

Pathogenesis

As a result of pronounced hemodynamic overload appear the decrease of right ventricle pump function, dilation of right ventricle, relative incompetence of tricuspid valve, that lead to the elevation enddiastolic volume and venous pressure at right

ventricle, right atrium, peripheral veins and capillaries. The central venous pressure is pronouncedly increased. Prolonged venous congestion leads to the enlarged liver, edema, ascites, and transudation of fluid into the pleural cavity and pericardium.

Clinical features

The patients complain on the heaviness in right hypochondrium, thirst, loss of appetite, nausea, vomiting, edema at the low extremities.

Objective examination. In patient there is a clinical sign of main pathology causing the heart failure. The features of congestion in greater circulation: swallowing of the neck veins, pulsation of the vena jugulars, and edema of the legs. Edema initially arises in the evening on feet, has ascending character and overspread on the subcutaneous tissue the whole body – anasarca.

Prolonged edema is accompanied by throphic skin disorders with redness, pigmentation and local ulcers.

The skin color is slightly yellow as a result of liver hypoxia and dysfunction.

Objective examination of the respiratory system may reveal the signs of main process. Due to the hydrothorax dullness and pathologically decreased or even absence of vesicular breathing are determined over one or both lungs.

Objective examination of the cardiovascular system. Cardiac beat and epigastric pulsation which increased in deep inspiration are determined. These signs are explained by hypertrophy and dilation of the right ventricle. The right border of the relative cardiac dullness displaced to the right. The heart sounds are decreased at the apex. Second sound over the pulmonary artery is accentuated.

Pulse is frequent. The change of blood pressure depends on main process.

In palpation of abdomen enlarged liver is revealed. In initial stage of chronic right ventricular heart failure liver is soft, with sharp edge, its surface is smooth, and with progression of heart failure liver becomes greater size, with high density. Prolonged venous congestion in greater circulation leads to development of ascites. Congestive kidneys are characterized by oliguria.

Additional methods of examination

Clinical blood analysis is without specific changes.

Clinical urine analysis: oliguria in edematous phase, proteinuria, cylindruria.

ECG – hypertrophy of right ventricle, right bundle branch block.

X-ray examination reveals the features of main process.

Echocardiography – hypertrophy of right ventricle.

Classification of heart failure

1. Clinical stages according to N.D. Strazhesko and V.H. Vasilenko

2. Variant of heart failure:

– with systolic dysfunction of left ventricle (ejection fraction ≤40 %);

– with normal systolic function of left ventricle (ejection fraction >40 %).

3. Functional class according to New York Heart Association.

Classification of heart failure according to

N.D. Strazhesko and V.H. Vasilenko

The three clinical stages of heart failure are distinguished:

I stage – initial, latent (fig. 2.1) there are symptoms during physical exercises: dyspnea, palpitation. These symptoms subside at rest.

II stage is characterized by symptoms and signs of heart failure not only during physical exercises, but at rest. II stage of heart failure subdivided into two stages – II stage A and II stage B.

In stage A there are features of congestion or lesser or greater circulation (fig. 2.2).

The characteristic of II stage B heart failure are the features of congestion in lesser and greater circulation (fig. 2.3). Patients are fully disabled. At rest pronounced cyanosis, swollen jugular veins, edema and ascites are revealed.

III stage heart failure is defined as final, dystrophic with marked congestion in the lesser and greater circulation, hemodynamic disorders, irreversible morphological changes of all organs, functional and metabolic disorders (fig. 2.4).

The patient would have extreme asthenia, loss of weight, cardiac cachexia. Skin is dry, dark, trophic skin ulcers, marked edema, hydrothorax, hydropericardium, ascites, anasarca, fibrosis of liver, lungs and kidney.

Classification of heart failure according to New York Heart Association

Class	New York Heart Association Functional Classification (NYHA)	Canadian Cardiovascular Society Functional Classification
I	Patients with cardiac disease but without resulting limitations of physical activity; ordinary physical activity does not cause dyspnea (or fatigue, palpitation, or anginal pain)	Ordinary physical activity, such as walking and climbing stairs does not cause angina. Angina with rapid or prolonged exertion at work or recreation
II	Patients with cardiac disease resulting in slight limitation of physical activity; they are comfortable at rest; ordinary physical activity results in dyspnea (or fatigue, palpitation, or anginal pain)	Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind, or when under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs and in normal conditions

III	Patients with cardiac disease resulting in marked limitation of physical activity; they are comfortable at rest; less than ordinary physical activity causes dyspnea (or fatigue, palpitation, or anginal pain)	Marked limitation of ordinary physical activity. Walking one to two blocks on the level and climbing more than one flight in normal conditions
IV	Patient with cardiac disease resulting in inability to carry on any physical activity without discomfort; symptoms of dyspnea (or of angina) may be present even at rest; if any physical activity is undertaken, discomfort is increased	Inability to carry on any physical activity without discomfort – anginal syndrome may be present at rest

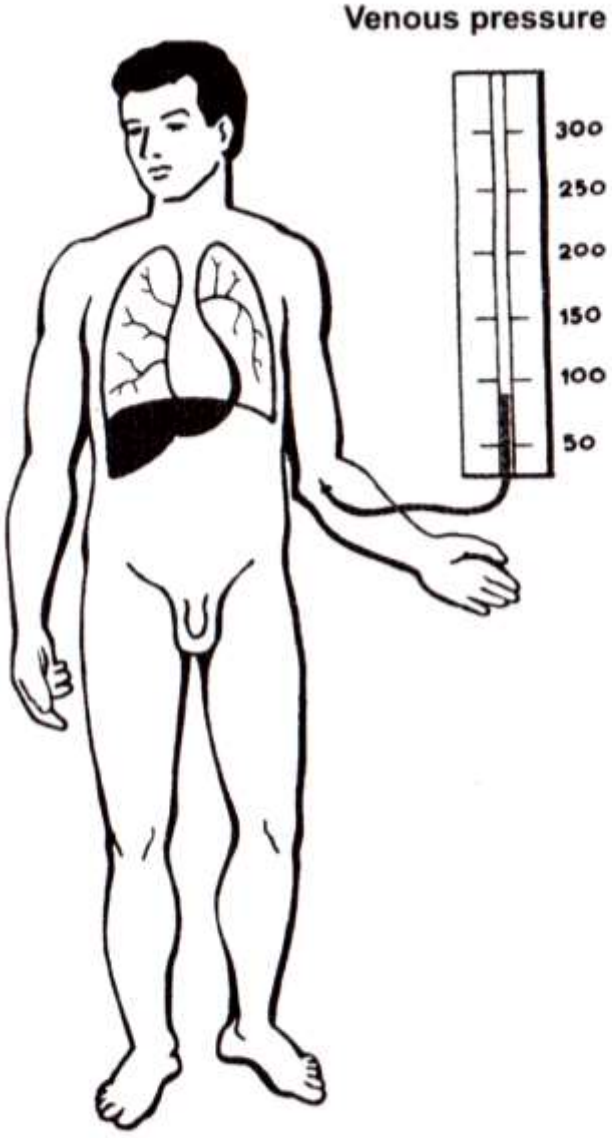


Fig. 2.1. Heart failure, stage I.

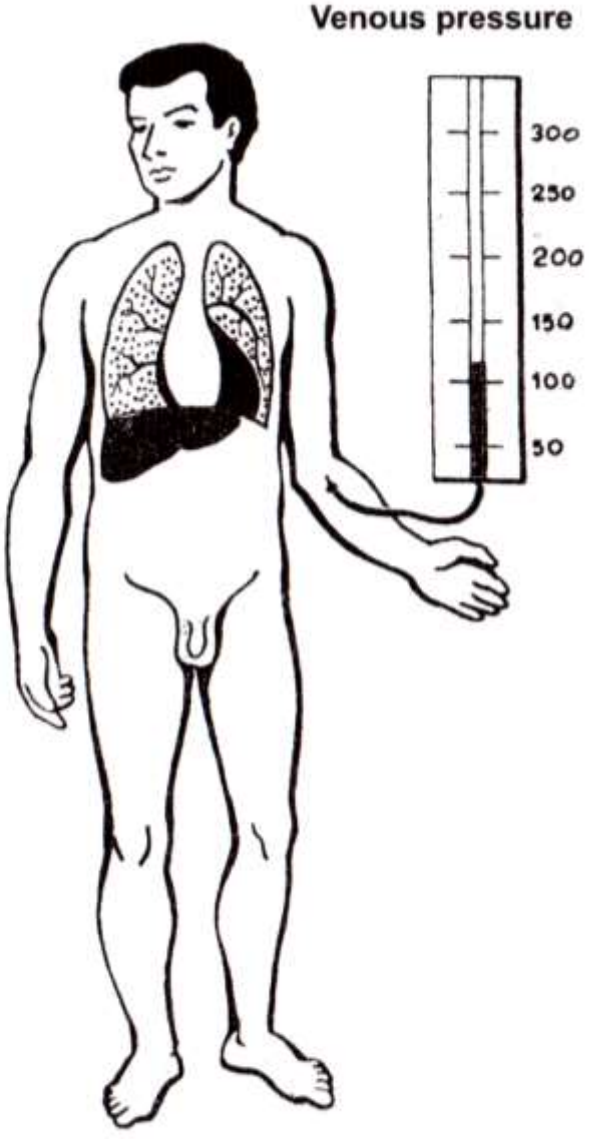


Fig. 2.2. Heart failure, stage II A.

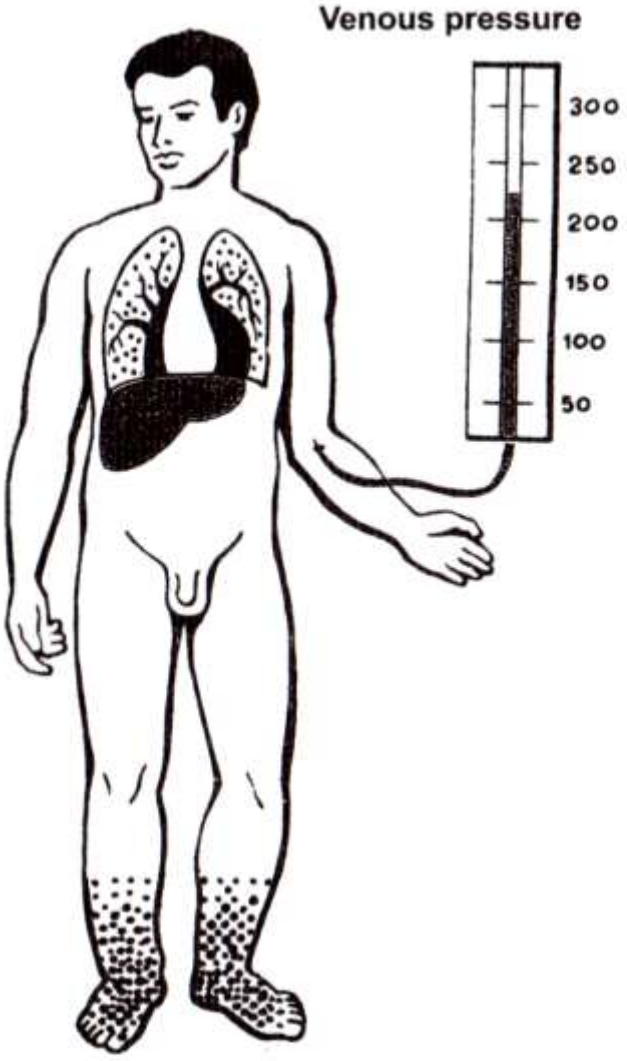


Fig. 2.3. Heart failure, stage II B.

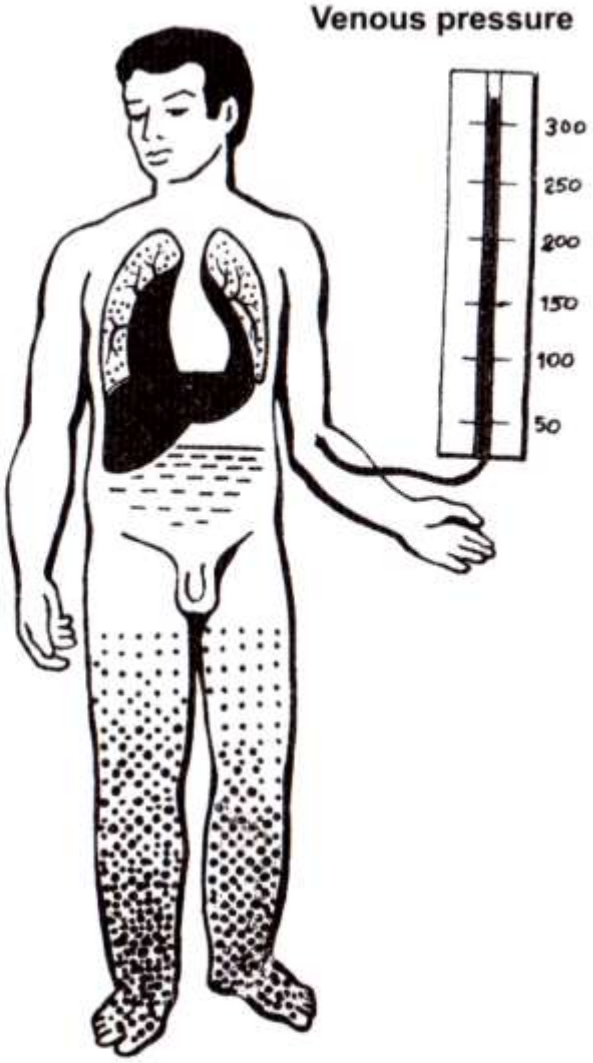


Fig. 2.4. Heart failure, stage III.

Syndrome of vascular failure

Vascular failure is the pathological condition caused by decreasing of the vascular tone and diminishing of the volume circulating blood.

Vascular failure includes: syncope, collapse, shock.

Syndrome of a syncope

Syncope – is a sudden transient loss of consciousness with rapid recovery not requiring electrical or chemical cardioversion.

Classification

Classification of syncope due to the etiology:

– neurally mediated syncopal syndromes vasovagal (carotid sinus, situational);

– orthostatic;
– cardiac arrhythmias as primary cause (bradycardia, tachycardia);
– structural cardiac or cardiopulmonary disease (acute myocardial infarction/ischemia, aortic dissection, pulmonary embolus).

The underlying mechanism of pathogenesis is a relatively abrupt cerebral hypoperfusion.

Clinical features

Presyncope is characterised by weakness, nausea, darkening in the eyes, noise in the ears. Syncope is characterized by loss of consciousness.

Objective examination. The person has a pallid skin, cold sweat, cold limbs, pupils are narrow, its reaction to light is present, pulse is thread, arterial pressure is decreased.

Initial evaluation may lead to a certain diagnosis based on symptoms, signs or ECG finding. This applies to the following cases:

– *vasovagal syncope* is diagnosed if precipitating events such as fear, severe pain, emotional distress, instrumentation or prolonged standing are associated with typical prodromal symptoms;

– *situational syncope* is diagnosed if syncope occurs during or immediately after urination, defecation, cough or swallowing;

– *orthostatic syncope* is diagnosed when there is a documentation of orthostatic hypotension (decrease of SBP ≥20 mmHg or to <90 mmHg) associated with syncope or pre-syncope;

– *syncope due to cardiac ischemia* is diagnosed when symptoms are present with ECG evidence of acute ischemia with or without myocardial infarction;

– *syncope due to cardiac arrhythmia* is diagnosed by ECG when there is:

- sinus bradycardia <40 beats/min or sinoatrial blocks or sinus pauses >3 s;
- atrioventricular block (2nd degree Mobitz's II or 3rd degree atrioventricular block);
- alternating left and right bundle branch block;
- rapid paroxysmal supraventricular tachycardia or ventricular tachycardia;
- pacemaker malfunction with cardiac pauses.

Syndrome of collapse

The collapse is acute vascular failure due to the affection of the vascular tone primary or secondary origin.

Etiology

– toxicosis, acute infections;
– profuse blood loss;
– dehydratation;

– myocardial infarction;
– embolism of the pulmonary artery;
– disordered vasomotor innervation of central origin;
– after taking some drugs;
– metabolic disorders.

Pathogenesis

Collapse develops due to upset central nervous regulation of the vascular tone. Diminished vascular tone disturbs normal distribution of blood in the body: the amount of deposited blood increases, especially in the vessels of the abdominal organs, whereas the volume of circulating blood decreases. The stroke volume of blood decreases and arterial and venous pressure diminish as well.

Clinical features

A collapse characterizes by giddiness, darkening in the eyes, noise in the ears, weakness, then often by loss of consciousness.

Objective examination. Pallid skin with a marble shade, cold sweat, cold limbs, decreasing of body temperature, accelerated and superficial respiration. The heart sounds are decreased, tachycardia, small, accelerated and thread pulse, decreased arterial and venous blood pressure.

Syndrome of shock

Shock is the clinical syndrome that develops when there is critical impairment of tissue perfusion to some organs.

Classification according to pathophisiological picture

Hypovolemic shock secondary to any condition provoking a major reduction in blood volume:
– internal/external hemorrhage;
– severe burns;
– acute pancreatitis;
– dehydration.

Normovolemic shock secondary to capillary damage, arteriovenous shunting and inappropriate vasodilatation:
– septic shock;
– anaphylactic shock.

Cardiogenic shock, caused by any form of severe heart failure:
– myocardial infarction;
– acute massive pulmonary embolism;

– heart tamponade due to pericardial enfusion.

Classification according to etiology

– infections,
– toxic,
– anaphylactic,
– hemorrhagic,
– cardiogenic,
– burn.

Pathogenesis

The syndrome of shock is characterized by widespread failure of the capillary system caused by a complex interaction of hemodynamic and toxic factors. Loss of capillary integrity reduces oxygen delivery to the tissues, disturbs local metabolism, and allows fluid to extravasate into the interstitial space. Generalized cell death and further capillary damage occur due to the combined effects of ischemia, acidosis, and the release of toxic metabolites including catecholamines, angiotensin II, and cytokines such as the interleukins and tumor necrosis factor.

Clinical features

– weakness, cyanosis, pallid skin, cold extremities;
– cold clammy skin;
– drowsiness, confusion, irritability;
– rapid shallow respiration;
– tachycardia (>100 beats per minute);
– thready pulse;
– hypotension (systolic BP <100mmHg)
– oliguria (urine output < 30 ml/hour);
– multi-organ failure.

Complications of shock:

– muscle damage;
– peripheral gangrene;
– visual and cerebral impairment;
– respiratory distress syndrome
– myocardial dysfunction;
– jaundice, impaired liver function;
– acute renal failure;
– disseminated intravascular coagulation/consumption coagulopathy.

Additional methods of examination

Although clinical evaluation is essential, progress can also be assessed by monitoring certain parameters at the bedside.

Addition methods of examination include:

- skin temperature – this reflects cutaneous blood flow and is therefore a useful indirect measurement of peripheral perfusion;
- urinary flow – this is a sensitive measure of renal perfusion and can easily be monitored if a urinary catheter is in place;
- oxygen saturation – this can be monitored easily using a finger or ear lobe probe. In general, oxygenation is satisfactory if the saturation is greater than 90 %;
- ECG – a standard monitor will record rate and rhythm, ST-segment shift, which may be useful in patients with ischemic heart disease;
- central venous pressure – using a catheter to measure the right atrial pressure is a useful means of assessing the circulating blood volume and therefore the appropriate rate of intravenous fluid replacement.
- pulmonary artery wedge pressure – in most situations the central venous pressure is an adequate guide to the filling pressures of both sides of the heart.

Heart valve diseases

Mitral regurgitation

Mitral regurgitation develops due to incomplete closure of the left atrioventricular valve during systole. As a result, the blood is regurgitated from the left ventricle to the left atrium.

Etiology

- rheumatic fever;
- bacterial endocarditis;
- atherosclerosis;
- congenital heart defects;
- mitral valve prolaps;
- perforation, rupture of the mitral valve, trauma of mitral cusps.

Disorders of hemodynamics

Disorders of hemodynamic occur because the bicuspid valve fail to close adequately and during systole of the left ventricle part of blood is regurgitated into the left atrium where added to the normal blood amount delivered from the pulmonary veins. This mitral regurgitation produces volume overload of the left atrium. Pressure in the left atrium increases (in normal 5–7 mm Hg), the atrium becomes hypertrophied. During diastole the amount of blood that is delivered into the left ventricle

from the overfilled left atrium exceeds normal. The left ventricle has to perform excess work and become hypertrophied (fig. 2.5). In late stages of the disease, when the contractile power of the myocardium of the left ventricle weakens, diastolic pressure in it increases and this in turn increases significantly pressure in the left atrium, consequently overfills pulmonary veins, and develops hypertension in the lesser circulation and reflective contraction of the arterioles in the lesser circulation. The raised atrial and pulmonary venous pressure leads to pulmonary edema. Together with the spasm in the arterioles pressure in the pulmonary artery increases significantly. The right ventricle can therefore be hypertrophied (fig. 2.6). In case of progression of disease, the right ventricle is dilated and tricuspid regurgitation occurs (fig. 2.7).

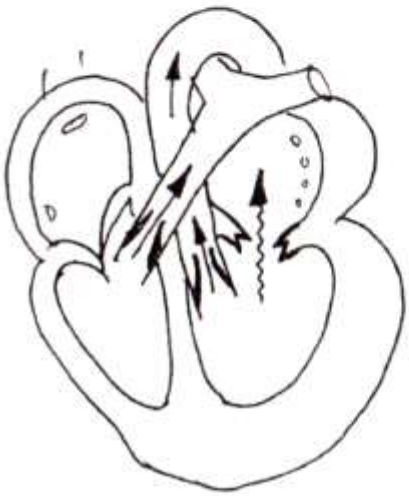


Fig. 2.5. Hypertrophy of left atrium and left ventricle.

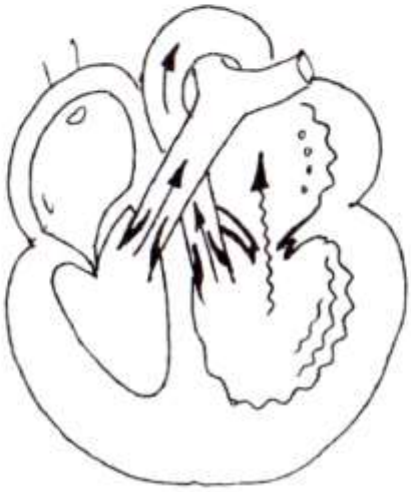


Fig. 2.6. Hypertrophy and dilation of left atrium, left ventricle and hypertrophy of right ventricle.

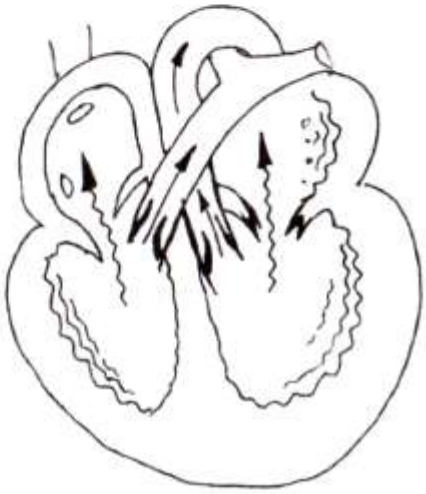


Fig. 2.7. Hypertrophy, dilation of right ventricle, functional tricuspid regurgitation

Clinical features

Clinical features appear when near 10 ml of blood returns from the left ventricle into the left atrium. Most patients have no complaints for a long time and feel like healthy people. Than congestion in the lesser circulation develops, patient feels fatigue, exhaustion, palpitations of the heart, cough, exertional and nocturnal dyspnea.

Objective examination. *The patient's condition* is from satisfactory to grave. Consciousness is clear. Posture is active. In case of development of heart failure with congestion in lesser circulation the posture is active with restriction due to the dyspnea. In late stage may be forced posture – orthopnea. Acrocyanosis is determined.

Examination of respiratory system. Intermediate percussion sound, decreased vesicular breathing and fine bubbling rales, even crepitation are revealed over the low lobes of lungs due to the congestion as a signs of raised pulmonary capillary pressure and chronic pulmonary congestion.

Examination of cardiovascular system. In inspection of heart region the diffuse apex beat is determined, displaced to the left in V, sometimes VI intercostals space.

During palpation: the apex beat is diffuse, intensified and resistant due to hypertrophy of the left ventricle.

In percussion reveals displacement of the upper border of the relative cardiac dullness upward, left border to the left. In the late stage of disease the right border of relative cardiac dullness is displaced to the right due to hypertrophy of the right ventricle. The configuration of the heart becomes mitral with indistinct waist of the heart.

In auscultation reveals decreased first heart sound at the apex because the mitral valves never closed completely. In the late stage, when the blood pressure rises in the lesser circulation, the second heart sound is accentuated and may be splitted over the pulmonary trunk. Blood pressure does not change in compensated mitral incompetence. The characteristic sign of the mitral regurgitation: blowing, decreasing murmur, which is heard at the heart apex it synchronous with the first heart sound. Murmur arises during systole when the stream of blood passes a narrow slit leading from the left ventricle to the left atrium. Murmur can be transmitted in the left axillary region and along the left edge of the sternum, it becomes more intensive after physical exertion, in lying position on the left side during deep expiration.

Complications of mitral regurgitation: acute pulmonary edema, atrial fibrillation/flutter very rare, chronic left ventricular and atrial failure, chronic right ventricular heart failure and chronic total ventricular heart failure.

Additional methods of examination

X-ray examination: smoothed of the left border due to protrusion of the left atrium auricle, moderate enlarging of the pulmonary trunk, protrusion of the left low arch, narrowing of the retrocardial space in the second oblique position, declining of the esophagus on the radius, signs of pulmonary venous hypertension.

ECG: signs of hypertrophy of the left atrium and left ventricle.

Echo-CG: dilatation of the left parts of heart, excursion of the atrioventricular partition and back wall of the left ventricle, different direction of the diastolic motion of mitral valve, unclosing them during systole. Four degrees of mitral regurgitation are distinguished (from 2 up to 5 cm and above).

Mitral stenosis

Mitral stenosis develops due to narrowing of left atrioventricular orifice.

Etiology

1. Rheumatic heart disease;
2. Bacterial (infectious) endocarditis;

Disorders of hemodynamics

Disorders of hemodynamics: due to pathological process, the adhesion of the mitral cusps, it consolidation, thickening and shortening narrow the left atrioventricular orifice. In patient with mitral stenosis orifice becomes 1,5 cm^2 and less instead of normal 4–6 cm^2. Narrowing of an orifice is a mechanical obstacle for a flow of blood from the left atrium to the left ventricle during diastole. The part of blood remains in the left atrium. Besides blood from pulmonary veins comes into the left atrium. In the left atrium the volume of blood is increased (in norm 50–60 ml, at narrowing 100–2000 ml), pressure raises (in norm – 5–7 mm Hg, at narrowing – 20–25 mm Hg). So the left atrium hypertrophies (fig. 2.8). However the muscle of a hypertrophied left atrium weak, therefore its contractile function reduces soon. It leads to dilation of the left atrium and increasing of venous pressure in pulmonary veins and capillaries (fig. 2.9). Increased pressure elevates in the pulmonary veins leads to irritation of baroreceptors, and causes reflex contraction of the arterioles in the lesser circulation (Kitaev's reflex), so pressure in the pulmonary trunk considerably rises, so called pulmonary hypertention. Pulmonary hypertension leads to a hypertrophy of the right ventricle, and subsequently and to its dilation (fig. 2.10, fig. 2.11). The left ventricle receives less blood in diastole, its size a little decreases and diastolic dysfunction develops.

Fig. 2.8. Hypertrophy of left atrium.

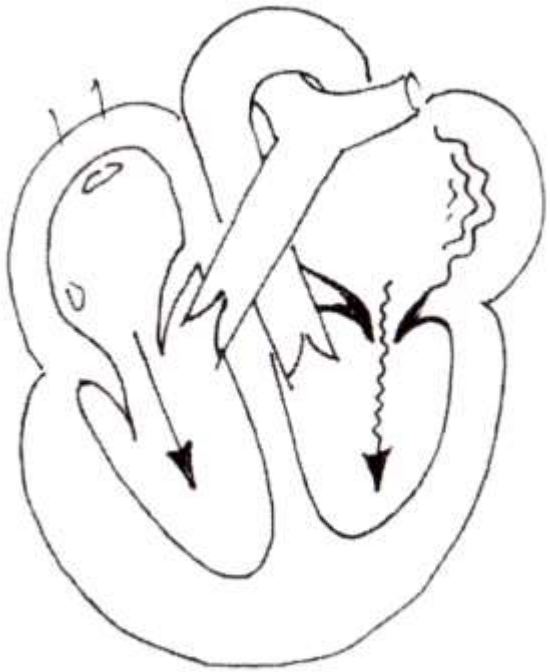


Fig. 2.9. Hypertrophy and dilation of left atrium.

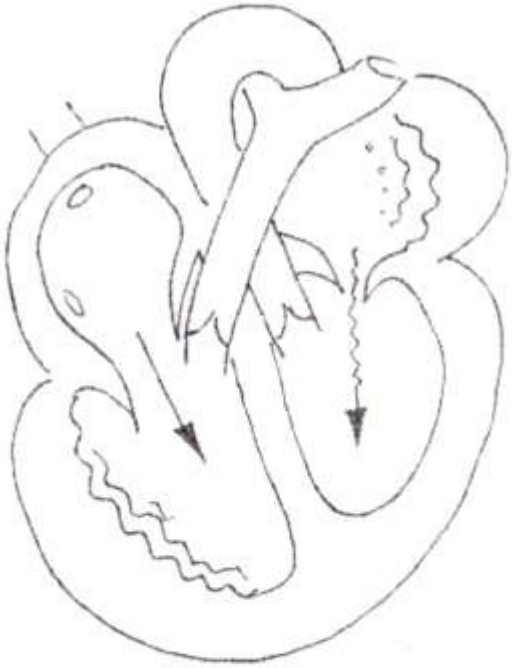


Fig. 2.10. Hypertrophy and dilation of left atrium, and hypertrophy, dilation of right ventricle (diastole).

Fig. 2.11. Hypertrophy and dilation of left atrium, and hypertrophy, dilation of right ventricle (systole).

Clinical features

The specific complaints of the patients with mitral stenosis: exertional and nocturnal dyspnea, cough, palpitation, pain in the heart. Symptoms secondary to arterial/venous emboli are hemoptysis, chest pain. Symptoms of diminished cardiac output are fatigue, tiredness.

Objective examination. In general inspection patient looks younger his age, the mitral face is observed. The characteristic of face: the cyanotic blush on the cheecks.

Examination of the respiratory system reveals the congestion in lesser circulation – moist rales in low lobes of lungs.

Examination of the cardiovascular system. In inspection of heart region the spread pulsation in the III-IV intercostals space along left edge of sternum with synchronous pulsation in the epigastric region are detected. During palpation apex beat is of normal location, area, height and strength. Cat's purr symptom is characteristic for mitral stenosis. Diastolic thrill is palpated at the apex.

In percussion relative cardiac dullness is displaced to the right and upward, protrusion of the upper part of the left contour, indistinct waist of the heart, increasing of absolute cardiac dullness area.

In auscultation the first heart sound at the apex becomes loud and snapping, because the left ventricle receives little amount of blood and fast closing of fibrous

cusps of the mitral valve. An additional sound due to the opening of the mitral valve, which would be explained by sclerosed and connected among themselves cusps. The loud first heart sound, second sound with the sound of opening of the mitral valve give a specific melody of mitral stenosis so called triple rhythm at the apex. The second sound becomes accentuated and splitted over pulmonary artery. At some patients with mitral stenosis cardiac rhythm is irregular, because mitral stenosis is often complicated with atrial fibrillation.

Diastolic murmur at the apex is sign of the mitral stenosis because the orifice from the left atrium to the ventricle during diastole is narrowed. This murmur can be heard to follow the mitral valve opening sound in early diastole (protodiastolic murmur – noise of filling) because the velocity of the flow in early diastole is higher due to the decreased pressure difference in the atrium and the ventricle. The murmur can be heard at the end of diastole, immediately before systole (presistolic). It arises during acceleration of the blood flow at the end of ventricular diastole.

Pulse on the radial arteries may be asymmetrical (p. differens) because the left subclavia artery is compressed by considerable hypertrophy of the left atrium. Blood pressure usually remains normal.

Complications of mitral stenosis: atrial fibrillation, flutter, arterial or venous emboli with massive pulmonary, cerebral, peripheral thromboembolism, acute pulmonary edema, chronic left atrial heart failure, right ventricle heart failure.

Additional methods of examination

***X-ray examination*:** disappearing of the heart waist, enlarged of the left atrium auricle, enlarged of the right ventricle, protrusion of the pulmonary trunk. Narrowing of the retrocardial space in the first oblique position, declining of the esophagus on the small radius and congestion changes are revealed.

***ECG*:** hypertrophy of the left atrium and right ventricle.

***Echo-CG*:**

- the unidirectional movement both cusps of the mitral valve in diastole;
- change of flow character through the mitral orifice in diastole (a turbulent stream);
- narrowing of the orifice of the left atrioventricular valve. At the healthy middle-aged person diameter and square of mitral orifice is from 4 up to 6 cm2 and from 2 up to 3 cm accordingly;
- thickened immobile cusps of mitral valve;
- reduced rate of diastolic filling;
- reduced valve area.

Aortic regurgitation

Aortic regurgitation is defined as incomplete closing of aortic valve during diastole that lead to retrograde blood flow from the aorta into the left ventricle.

Etiology

- bacterial endocardidis;
- rheumatic endocarditis;
- syphilis of the aorta;
- atherosclerosis of the aorta;
- Marfan's syndrome.

Disorders of hemodynamics

During diastole the blood is delivered into the left ventricle not only from the left atrium but also from the aorta due to regurgitation, thus the left ventricle during diastole overfills. The amount of returning blood can reach from 5 up to 50 % of volume of the left ventricle. There is a significant overload of the left ventricle with volume (systolic volume may reach 200 ml and more). During systole the left ventricle has to contract with a greater force in order to expel the large volume of blood into the aorta. Insufficiency of the aortic valve for a long time may be compensated by the strengthened work of hypertrophied powerful left ventricle (fig. 2.12). In case of intense regurgitation of the blood that moves aside the mitral valve is formed functional mitral stenosis (fig. 2.13). During progression of disease increased systolic volume at left ventricle causes its dilation (fig. 2.14). At longstanding course the disease may be accompanied by functional incompetence of mitral valve with regurgitation of blood in left atrium, which is hypertrophied as response to overloading (fig. 2.15).

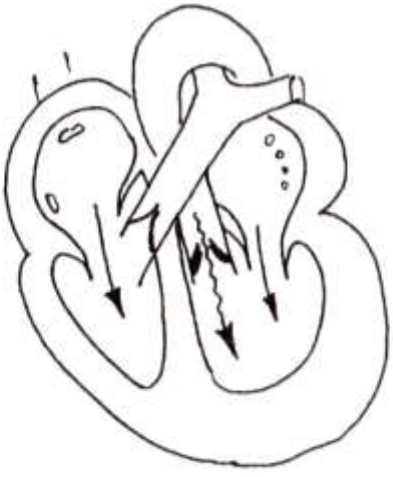


Fig. 2.12. Hypertrophy of left ventricle.

Fig. 2.13. Hypertrophy of left ventricle and functional mitral stenosis.

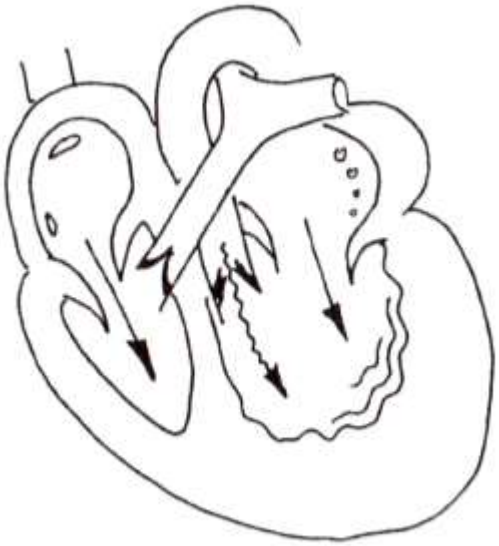


Fig. 2.14. Hypertrophy and dilation of left ventricle.

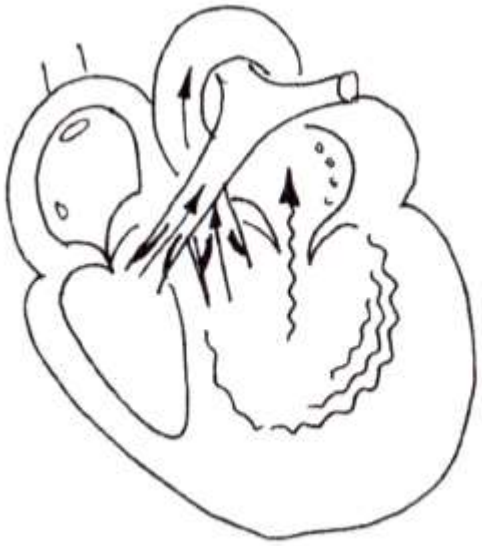


Fig. 2.15. Hypertrophy, dilation of left ventricle, functional mitral regurgitation, hypertrophy of the left atrium.

Clinical features

Patient complains on pain in the heart is due to the relative coronary insufficiency because of pronounced hypertrophy of the myocardium and inadequate filling of the coronary artery under low diastolic pressure in the aorta. Dizziness, headaches, syncope are the results of deranged blood supply to the brain.

In condition of decreased contractile ability of left ventricle the attacks of cardiac asthma occur. The signs of "mitralization" of cardiac regurgitation are dyspnea, cough.

Objective examination. In general inspection may observe such signs:

- the skin of the patient, especially on face, is pallid due to the insufficient filling of the arterial system during diastole, pulsation of the peripheral arteries (carotid, subclavian, brachial, temporal) as a result of marked variation blood pressure in the arterial system during systole and diastole;
- rhythmical movement of the head synchronous with the pulse (de Musset's sign).
- rhythmical change in the color of the nail bed under a slight pressure on the nail end, so-called capillary pulse – Quincke's pulse;
- rhythmical reddening of the skin after rubbing.

Objective examination of the cardiovascular system:

- the apex beat is almost always enlarged and shifted to the left and inferiorly;
- the apex beat is palpable in the sixth and sometimes seventh intercostals space, outside the left midclavicular line, even till the axillary line;
- the apex beat is diffuse, intense, rising like a dome due to the significant enlargement of the left ventricle.

In percussion – the border of the cardiac dullness can be found shifted to the left. The heart becomes "aortic" with pronounced waist of the heart.

In auscultation reveals decreased first heart sound at the apex because the period when the valves are closed is absent. The second sound on the aorta is also weak due to the damage of the valve. Protodiastolic murmur is heard over the aorta and at the Botkin-Erb's listening point. Diastolic murmur (presystolic or mid-diastolic Austin Flint's murmur) can sometimes be heard. It arises due to an intense regurgitation of the blood that moves aside the mitral valve casps and forms the functional mitral stenosis. At auscultation of femoral artery double Traube's tone is heard due to rapid systolic tension and dilation of artery. In case of pressure femoral artery by stethoscope double Vinogradov-Duroziez's murmur can be heard – femoral bruit ("pistol shot").

Blood pressure: systolic pressure rises, diastolic decreased, pulse pressure is therefore high.

Pulse – fast, high large volume (pulses celer et altus).

Complications: cardiac asthma, pulmonary edema, chronic heart failure due to the "mitralization" of aortic regurgitation.

Additional methods of examination

ECG: hypertrophy of the left ventricle: the electrical axis is deviated to the left, the S-wave in the right chest leads are deep and the amplitude of the R-wave is higher in the left chest leads, relative coronary insufficiency: depressed of the ST-interval, inverted T-wave.

***X-ray*:** protrusion of the left ventricle arch, heart apex rounding, marked heart waist, (heart configuration is duck-like), narrowing of the retrocardial space in the second position.

***Echo-CG*:** dilated left ventricle, retrograde blood flow through the aortic valve into the left ventricle during diastole.

Aortic stenosis

Aortic stenosis develops due to the narrowing of the aortic orifice resulted from different origin.

Etiology:

- bacterial endocarditis;
- atherosclerosis;
- congenital aortic stenosis (subvalvular and supravalvular);
- muscular obstructive hypertrophic cardiomyopathy.

Disorders of hemodynamics: At the expressed narrowing of the aorta orifice, when its area decreases up to 1,0–0,75 cm2 (in norm 3 cm2) during systole left ventricle does not empty completely. The gradient of systolic pressure between the left ventricle chamber and an aorta is increased. It exceeds 20 mm Hg, sometimes 100 and more.

At narrowing of aortic orifice the minute volume of blood is reduced. In diastole to this remained blood in the ventricle the normal amount of blood from the left atrium is added that lead to overfilling of left ventricle with blood and to increase of pressure in it. Systolic pressure in the left ventricle raises proportionally degrees of narrowing of the aorta orifice (in norm 120 mm Hg, at narrowing raises in 1,5–2 times in comparison with a normal amount and may reach 250–300 mm Hg). This disorders of heart hemodynamics is compensated by the strengthened work of the left ventricle and causes its hypertrophy (fig. 2.16). Coronary blood flow may become inadequate.

Due to good compensatory abilities of the left ventricle asymptomatic course of this disease may last 10–15 years. At reduction contractile abilities of myocardium develops dilatation of the left ventricle (fig.2.17), later relative insufficiency of the mitral valve adds (fig.2.18), with regurgitation of blood in the left atrium. First there is an intimate insufficiency in the lesser, and then in the lager circulation.

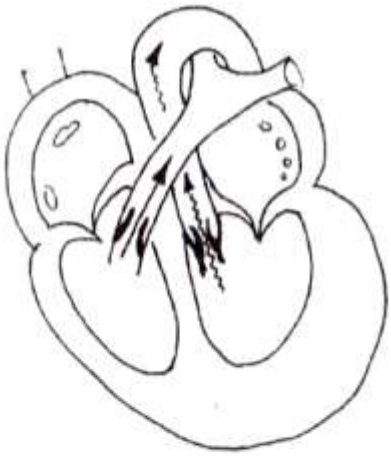


Fig. 2.16. Hypertrophy of left ventricle.

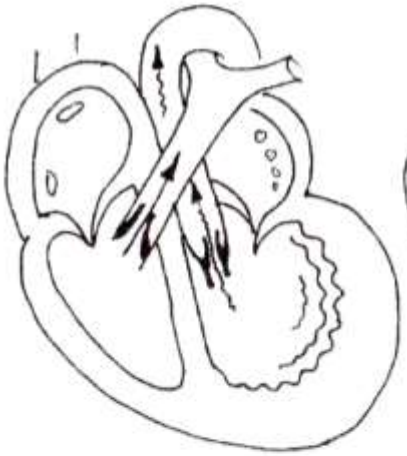


Fig. 2.17. Hypertrophy and dilation of left ventricle.

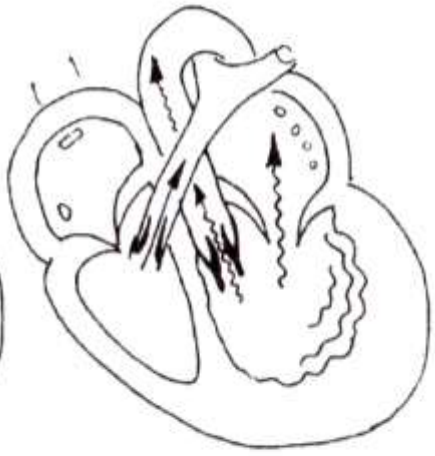


Fig. 2.18. Hypertrophy and dilation of left ventricle, relative mitral regurgitation

Clinical features

Patient complains on pain in the heart (angina type pain) due to the considerable insufficient blood ejection into the arterial system which upset normal blood supply to the hypertrophied myocardium of left ventricle. Disordered blood supply to the brain is manifested by giddiness, headache, tendency to fainting. During sudden decrease of contractile ability of left ventricle may occur acute left ventricles heart failure with clinical signs of cardiac asthma and even pulmonary edema.

Objective examination. In general inspection the patient is pallid.

Examination of the cardiovascular system. The apex beat is displaced to the left, less frequently inferiorly, it is diffuse, high and resistant. Systolic thrill (cat's purr) can be palpated over the aorta. Percussion reveals displacement of the left border to the left, the heart configuration is "aortic" due to considerable hypertrophy of the left ventricle.

Auscultation of the heart at apex reveals diminished first heart sound due to the overfilling of the left ventricle and prolongation of systole.

The second sound is diminished over the aorta because the aortic cusps adhere and are immobile, the second sound can be inaudible.

Rough systolic murmur over the aorta is characteristic. This murmur is generated by the blood flow through the narrowed orifice. It is conducted by the blood on the a. carotis and can sometimes be heard in the interscapular space.

The pulse is small, slow and rare, since the blood slowly passed into the aorta and its volume is decreased.

Systolic blood pressure is usually diminished, while diastole remains normal or increased.

Complications: sudden cardiac death, cardiac asthma, pulmonary edema, heart failure due to "mitralization" of aortic stenosis.

Additional methods of examination

X-ray examination shows hypertrophied left ventricle, "aortic" configuration of the heart and post-stenotic dilatation of the ascending aorta, the cusps of the aortic valve are often calcified on lateral view.

EchoCG: Great number of echo-signals into the aorta space that is related to deformation and quite often calcinosis of aorta valve cusps. Dilation of the left ventricle, enlarging of its back wall and interventricular septum are determined. Doppler-cardiography reveals high-speed flow in stenotic orifice (flow speed may reach 200–500 cm/sec), and also allows to measure a gradient of pressure through aortic orifice.

Syndrome of the arterial hypertension

Arterial hypertension is defined as elevation systolic blood pressure (SBP) to 140 mmHg and higher and diastolic blood pressure (DBP) to 90 mmHg and higher in case of stable elevation confirming on repeating measurement blood pressure (2–3 times in different days during 4 weeks).

Classification on etiology:

1. Secondary (symptomatic) hypertension.
2. Essential arterial hypertension.

Symptomatic arterial hypertension

Symptomatic arterial hypertension causally related to the diseases with damages of some organs, participating in regulation of arterial pressure.

Causes of secondary hypertension

1. Renal diseases:

- *parenchymal and interstitial* diseases of kidneys (glomerulonephritis, chronic pyelonephritis, diabetic nephropathy, amyloidosis, hydronephrosis, postradiation nephrosclerosis);
- *renovascular* pathology (atherosclerosis of kidney artery, fibromuscular dysplasia, aortoarteritis, vasculitis, endarteritis, thrombosis, embolism, aneurysm of kidney artery, stenosis and thrombosis of veins, trauma of kidney vessels);
- *anomalies* of kidney and urinary tract (polycystosis, hypoplasia, anomalies of urinary system);
- *secondary damage* of kidneys at tuberculosis, bacterial metastases and diffuse diseases of connective tissue (lupus, system sclerodermia).

2. Endocrine hypertension:

- phaeochromocytoma;
- primary hyperaldosteronism (Conn's syndrome);
- idiopathic hyperplasia adrenal cortex (pseudoprimary hyperaldosteronism);
- Cushing's disease (syndrome);
- hyperparathyroidism;
- acromegaly;
- climacteric hypertension.

3. Hemodynamic hypertension:

- atherosclerosis of aorta;
- stenosis of carotid and vertebrobasilar arteries;
- coarctation of aorta;
- aortic regurgitation;
- reological hypertension (polycythemia vera).

4. Neurogenic hypertension:

- vascular diseases and tumors of brain;
- inflammatory diseases (encephalitis, meningitis, polyomyelitis);
- trauma of brain (postcontusional syndrome);
- polyneuritis.

5. Special forms of second hypertension (after taking some medicines: anabolic steroids and mineralocorticoids, oral contraceptives, containing progesteron and estrogen, sympathomimetic agents, indometacin and other).

Clinical features

Among all hypertensive states secondary arterial hypertension make approximately 20 %.

Chronic glomerulonephritis meets more frequent in young and middle ages. In anamnesis is acute glomerulonephritis. Clinical features of the glomerulonephritis – proteinuria more than 1 g/day, hematuria, impairment of renal function (early onset with hypo- and isostenuria) and hypertension (mostly increasing of diastolic pressure). Angina pectoris, myocardial infarction and stroke are rare. Rethinopathy develops comparatively lately, arteries are only slightly narrowed, veins are normal. But anemia, which atypical for essential hypertension, is often marked. Final establishment of diagnosis based at the result of isotopic renovasography and biopsy of kidneys, which finds out fibrioblastic, proliferative, membranouse and sclerotic changes in glomerules, tubes and vessels of kidneys.

Chronic pyelonephritis – is a chronic interstitial nephritis resulting from urinary tract infection associated with vesico-uretric reflux. In anamnesis are nephrolitiasis, pyelitis, anomalies of development at kidneys and other diseases of urinary tract. The most important among the morphological features is presence of coarse scars, which is associated with contraction of the related papilla and dilatation of the corresponding calyx. In the half of cases pyelonephritis is accompanied by arterial hypertension. Difficulty of diagnostics of chronic pyelonephritis consists of that in 1/3 cases the signs of inflammatory process in urinary ways are not observed. Diagnosis based at the following signs: hypostenuria, polyuria, pyuria, proteinuria, information of isotopic reno-, urography, ultrasound examination (diminishing of sizes of kidneys, deformation of the tubular system), biopsy of kidneys and angiography. From the general features of course of disease: young age, primary increase of diastolic pressure, absence of coronary and cerebral complications.

Diagnostic criteria of the renoparenhimal hypertension:

- pointing in anamnesis on the previous pyelonephritis, glomerulonephritis, nephropathy at pregnant, nephrolytiasis and other diseases of kidneys;
- characteristic changes of laboratory, instrumental and morphological examination of kidneys and also positive hypotensive effect from specific therapy of kidney disease.

Diagnostic criteria of the renovascular hypertension:

- high systolic hypertension, refractory to treatment;
- in auscultation – systolic murmur over the abdominal aorta and especially in the area of projection of kidney arteries;
- small sizes of one kidney (ultrasound and urography);
- disorders of contrast distribution at kidney (at intravenous urography);
- high level of renin in plasma of blood;
- narrowing of the (one or both) kidney arteries (angiography).

Phaeochromocytoma – is a rare tumor of the chromaffin tissue which secretes catecholamines and is responsible for less than 0,1 % of causes of hypertension. The tumors are usually benign (10 % malignant) and may arise from any part of the sympathetic chain. In over 90 % of causes the tumor is found in the adrenal medulla.

Clinical features: hypertension usually paroxysmal.

Diagnostic criteria of the phaeochromocytoma:

- transitional arterial hypertension with the signs of activation of the sympathetic nervous system (excitation, trembling, increasing of body temperature), leucocytosis, hyperglicemia. Stable character of arterial hypertension does not exclude phaeochromocytoma;
- negative effect from therapy with beta-blockers;
- positive provocative tests (histamine, injected intravenously in a dose 0,05 mg in 0,5 ml of isotonic solution, causes an increase blood pressure on 60/40 mmHg during the first 4 min; palpation of kidney region provokes hypertensive crisis) and test with alpha-adrenoblockers;
- enlargement of adrenal glands from data of ultrasonic research, computer tomography;
- finding out the high level of adrenalin, noradrenalin. Tumor of adrenal gland found out by the instrumental methods.

Primary hyperaldosteronism (Conn's syndrome) is characterized by overproduction of aldosterone, the main salt-retaining hormone, may be due to a primary abnormality in the zone glomerulosa or secondary to stimulation of aldosterone secretion by angiotensin II following activation of the renin-angiotensin system.

Diagnostic criteria of the primary hyperaldosteronism (Conn's syndrome):

- high blood pressure;
- musclular weakness and neuro-muscular disorders (paraestesia, occasionally tetany because of the metabolic alkalosis with low ionized calcium, transient para- and tetraplegia).
- polyuria, nicturia, thirst;
- hypokaliemia, hypernatriemia, increase of potassium level in blood after the test with veroshpiron;
- alkaline reaction of urine;
- the low level of plasma renin;
- diminishing of tolerance to glucose, rarer is obvious diabetes mellitus;
- finding out the tumor at the adrenal gland by ultrasonic investigation, computer tomography, radioisotope scanning of adrenal glands;
- level of the aldosteron in blood and urine (their increase to 100ng/ml and to 150 mcg/day, respectively).

Cushing's syndrome – is defined as the symptoms and signs associated with prolonged inappropriate elevation of free corticosteroide level. Patients with Cush-

ing's syndrome can be classified into two groups on the basis of whether the condition is adrenocorticotropic hormone (ACTH)-depended or independed:

1. ACTH-depended:

- iatrogenic (ACTH-therapy);
- pituitary-depended bilateral adrenal hyperplasia (Cushng's disease);
- ectopic ACTH syndrome (benign or malignant non-endocrine tumour).

2. non-ACTH-depended:

- iatrogenic (prednisolone therapy);
- adrenal adenoma;
- adrenal carcinoma.

Diagnostic criteria of the Cushing's syndrome:

- general inspection: persons with overweight, obesity, thinning hair, hirsutism, acne, plethora, moon face, presence of purple-violet striates on the skin of abdomen, thighs, in area of armpits;
- examination of organs and systems: arterial hypertension, psychosis, cataracts, peptic ulcer, osteoporosis, exuberant callus with fractures, wasting and weakness of the proximal thigh muscles, menstrual disorders;
- disorder of tolerance to glucose, hyperglycemia;
- changing in the normal day's rhythm of secretion of ACTH and cortisol (in a norm in the morning higher, than in the evening), increasing the level of cortisol and 17-OKS is in blood.

Hemodynamic arterial hypertension is associated with demanding of the heart and large vessels and subdivides into:

a) systolic hypertension at atherosclerosis, aortic regurgitation;

b) regional hypertension at aorta caorctation;

c) hyperkinetic circulatory syndrome at arteriovenouse fistulas.

Diagnostic criteria of the hemodynamic arterial hypertension

Arterial hypertension as a result of atherosclerosis of aorta is diagnosed on the basis of the followings signs: elderly patients, accented of second heart sound and its metallic tint over the aorta, systolic murmur over the aorta, increased systolic arterial pressure, presence the signs of atherosclerosis of peripheral arteries; expansion of aorta detected by ultrasound and X-ray examination.

Arterial hypertension under aortic regurgitation characterized by increased of systolic blood pressure and the decreased diastolic with high pulse pressure.

Arterial hypertension as a result of aorta coarctation is characterized by increasing of blood pressure at the upper extremities and it's decreasing at the lower extremities. In palpation – over the intercostal arteries intensification of pulsation is marked, loosening the pulsation at the peripheral arteries of lower extremities is observed; in auscultation – rough systolic murmur is heard at heart base, over the pectoral aorta (at the anterior chest wall and in interscaspular region), irradiated along the large vessels (carotid, subclavia).

Essential hypertension

Essential hypertension (hypertension) is a disease of the cardiovascular system, which develops due to primary dysfunction of the vascular regulatory centers and subsequent involment of neurohumoral and kidney mechanisms, characterized by arterial hypertension, functional, and at the expressed stages – by the organic changes of kidneys, heart and central nervous system. The essential hypertension can be diagnosed after exception of symptomatic (secondary) hypertension.

Predisposing factors: genetic factors; disorders of the nervous and endocrine systems, obesity; alcohol; smoking; hypodynamia; elderly age; professional factors: noise and vibration; hormonal factors: increased renin, reduced nitric oxide release.

Etiology

Acute and chronic psychoemotional stress, permanent mental overstrain, hypoxia of brain of any origin, age related neuroendocrine rebuilding (climacterium), salt abuse.

Pathogenesis

Elevation of blood pressure arise due to the imbalance between pressor and depressor factors which lead to development of changes in arterioles and precapillares, changing structure and function of cellular membranes, including smooth muscular cells of arterioles, disorders of activity of sodium-calcium pumps, increasing concentration of the ionized calcium in cytoplasm and finally excessive vascular resistance.

Classification

Classification of hypertension according to blood pressure level and organ damage depicted on table 2.1 and table 2.2.

Table 2.1. ***Classification of hypertension by blood pressure level***

Category	SBP (mm Hg)		DBP (mm Hg)
Optimal	<120	and	<80
Normal BP	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade I hypertension	140–159	and/or	90–99
Grade II hypertension	160–179	and/or	100–109
Grade III hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

Table 2.2. ***Classification of hypertension by extent of organ damage***

Stage I	No objective signs of organic changes
Stage II	At least one of the following signs of organ involvement without symptoms or dysfunction: – left ventricular hypertrophy (electrocardiogram, ultrasound); – generalized and focal narrowing of the retinal arteries; – proteinuria and/or slight elevation of plasma creatinine concentration (1,2 – 2,0 mg/dl or to 177 mmol/l); – ultrasound or radiological evidence of atherosclerotic plaque (carotid arteries, aorta, iliac and femoral arteries)
Stage III	Both symptoms and signs have appeared as result of organ damage. These include: – heart (myocardial infarction, heart failure); – brain (stroke, transient ischemic attack, encephalopathy, vascular dimension); – optic fundi (retinal hemorrhages and exudates with or without papilloedema); – kidney(plasma creatinine concentration more than 2,0 mg/dl or 177 mmol/l); – vessels (dissecting aneurysm, symptomatic arterial occlusive diseases)

Clinical features

Complaints: pain at the heart, palpitation, headache, dizziness, disorder of vision. At the expressed left ventricular failure – attacks of dyspnea.

Objective examination. *General patient condition* is usually satisfactory**.** On progression of disease and appearance of complication general patient's condition may be from middle grave to grave (hypertension crisis, acute and chronic heart failure and cerebral attacks).

The color of the skin may be hyperemic. As usually the patients are overweight. At development of heart failure acrocyanosis and peripheral edema are observed.

Objective examination of the cardiovascular system. Apex beat is displaced to the left and downwards, diffuse, high. Displacement of the left border of the relative cardiac dullness to the left is observed. Increased loudness of the first heart sound at the heart apex and accentuated second heart sound over aorta are heard. At the presence of heart failure the gallop rhythm is heard. Blood pressure $\geq$ 140/90 mm Hg. Pulse is firm tension (p. durus).

Protocol of diagnostic procedures for patients with hypertension I-II stages

Obligatory examination:

- inquiry;
- physical examination: measurement of blood pressure on both hands, measurement of blood pressure on lower extremities at persons younger 45 years;
- measurement of body weight of and waist circumference;
- laboratory routine examination hemoglobin and hematocrit, clinical urine analysis, Nechiporenko's test, Zemnicky's test, biochemical blood analysis: serum creatinine, serum potassium, serum total cholesterol, serum low density lipoprotein (LDL) cholesterol, serum high density lipoprotein (HDL) cholesterol, fasting serum triglycerides;
- ECG in 12 standard leads;
- echocardiography;
- fundoscopic examination.

Special examination:

- determination of microalbuminuria;
- daily proteinuria;
- ambulatory blood pressure measurement using monitor;
- ultrasound examination of kidneys.

Protocol of diagnostic procedures for patients with hypertension III stages

Obligatory examination:

- inquiry;
- physical examination: – measurement of blood pressure on both hands, measurement of blood pressure on lower extremities at persons younger 40 years;
- measurement of body weight and waist circumference;
- laboratory routine examination hemoglobin and hematocrit, clinical urine analysis, Nechiporenko's test, Zemnicky's test, biochemical blood analysis: serum creatinine, serum potassium, serum total cholesterol, serum low density lipoprotein (LDL) cholesterol, serum high density lipoprotein (HDL) cholesterol, fasting serum triglycerides;
- ECG in 12 standard leads;
- echocardiography;
- examination of fundu of the eyes;
- X-ray examinationof the chest;
- ultrasound examination of kidneys.

Special examination:

- ambulatory blood pressure measurement using monitor;
- doppler-ultrasound scanner of extracranial vessels;

– computer tomography and magnitoresonance tomography of head;
– in case of coronary heart diseases – cardioventriculography.

Additional methods of examination

Clinical blood analysis: at the prolonged course of hypertension occur hypertensive polycytemia – increased hemoglobin and hematocrit are possible.

Biochemical blood analysis: at development of kidney failure there is increasing level of creatinine.

Clinical urine analyses: at development of nephroangiosclerosis and renal failure – proteinuria, microhematuria, hypo-, isostenuria in Zimnitsky's test.

ECG: the left ventricle hypertrophy, depressed ST-segment, inverted or two-phase T-wave in the 1st and 2nd standard, V_5–V_6 chest leads.

X-ray examination of heart. In the initial period of hypertrophy, rounding of apex of the left ventricle are find out. All chambers of heart are dilated in the late stages.

***Echocardiography*:** hypertrophy of the interventricular septum and the back wall of the left ventricle, decrease of contractility of the myocardium, increase end systolic and diastolic dimensions of the left ventricle.

Ophthalmoscopy is revealed angioretinopathy.

Ischemic (coronary) heart disease

Ischemic heart disease – define as acute and chronic heart damage, caused due to diminishing or stopping blood delivery to myocardium. Disease of the coronary arteries is almost always due to atheroma and its complications, particularly thrombosis.

Classification of ischemic heart disease (IHD)

1. Sudden cardiac death.

2. Angina pectoris:
– stable angina pectoris;
– vasospastic angina (Princmetala's);
– unstable angina.

3. Myocardial infarction (MI):
– acute Q-wave MI;
– acute non-Q-wave MI;
– subendocardial MI;
– acute MI (undetected);
– recurrent MI (3–28 days);
– repeated MI (after 28 days).

4. Postinfarction cardiosclerosis faction.

5. Cardiac arrhythmia.
6. Painless form of the IHD.

Etiology and pathogenesis

Atherosclerosis of coronary arteries; the degree of its expression is different – from small wall affection to complete occlusion of vessel.

Spasm of coronary arteries develops, as a rule, on a background of atherosclerosis of coronary arteries. The physical overloading, mental stress provokes the development of clinical features of IHD.

The main pathophysiological mechanism of IHD is imbalance between the demand myocardium in oxygen and possibilities of coronary arteries satisfied the myocardium by adequate amount of blood.

The followings mechanisms are involved in pathological process:

- mechanical occlusion of coronary arteries due to an atherosclerotic process;
- dynamic occlusion of coronary arteries due to coronarospasm;
- activation of thrombocytes aggregation with development of microagregates in microcirculation;
- promotion of production the pro-coagulating factors, insufficiently level of prostacyclin and endothelin- derived relaxing factor;
- increasing of demand myocardium in oxygen under influencing of the intensive physical loading, mental stress, resulting in the high level catecholamines in blood caused cardiotoxic action;
- insufficiency of collateral circulation of blood;
- activation of the lipid peroxidation;
- activation of immune mechanisms.

Thus the pathological substrate of IHD is almost atheroma narrowing of the coronary arteries. Atheroma or atherosclerosis is a focal disease of the arterial intima. There are some stage of evolution of atherosclerotic process. Initial stage is fatty streaks which develop as circulating monocytes migrate into the intima take up oxidized low density lipoprotein from the plasma and become lipid foam cells. As these foams cells die extracellular lipid pools appear. Smooth muscle cells then migrate into and proliferate within the plaque. A mature fibrinolipid plaque has a core extracellular lipid, separated from the lumen by a thick cap of a collagen-rich fibrous tissue. Such plaque may narrow the lumen of the vessel and often precipitate local vasospasm and thrombosis. The luminal diameter of a coronary artery must be decreased by at least 50 % to 70 % before blood flows becomes inadequate to meet the metabolic demands of the heart during exercise or stress.

The evolution of the atheromatous plague corresponds with clinical forms of IHD. The principal cause of *stable angina* is atherosclerosis involving at least one large epicardial artery that limited coronary flow under some condition. Stable an-

gina is related to a fixed obstruction and it is usually precipitated by an increase in myocardial oxyden demand (demanded ischemia).

Unstable angina is defined as an obstruction of at least one major epicardial artery that occupies at least 70 % of the artery's cross-sectional diameter or an obstruction of the left main coronary artery that occupies at last 50 % of its diameter. Episodes of myocardial ischemia are due to abrupt reduction in coronary blood flow results from plaque rupture, rapid growth of the lesion or incomplete occlusion of the vessel. Unstable angina is a transitory condition. A platelet rich thrombus forms rapidly around the site of the rupture, reducing, but not usually occluding the blood flow in the vessel.

Myocardial infarction is almost always due to the formation of occlusive thrombus at the site of rupture of an atheromatous plaque in a coronary artery. The affected artery is more commonly completely occluded, usually by a fibrin-rich "red" thrombus.

Sudden death in most cases is attributable to IHD and is usually due to arrhitmia or asystole (ventricular fibrillation, sinoatrial block, complete AV-block) related to acute coronary syndrome, heart failure or scarring from a previous myocardial infarction.

Stable angina

The 2002 American College of Cardiology/ American Heart Association (ACC/AHA) guideline update defined chronic stable angina as a clinical syndrome characterized by discomfort in the chest or adjacent areas caused by myocardial ischemia typically aggravated by exertion or emotional stress and relieved by rest or by nitroglycerin. Patients often describe their symptom as discomfort rather than pain.

Clinical features

The main parameters of pain in patients with stable angina are: location, character, intensity, duration, frequency, radiation, associated symptoms and cause of onset, aggravating and relieving factors. The typical location of angina is mid or lower part of sternum. Less typically, discomfort may occur in the epigastric area. The discomfort is usually described as pressure, tightness, heaviness, strangling, constricting, burning, squeezing, suffocating and crushing.

The severity of the discomfort varies greatly. The pain may radiate in arm to the wrist and fingers, lower jaw or teeth, throat, between the shoulder blades. The duration of the discomfort is brief, not more than 10 min in the majority of cases and more commonly even less. Angina equivalents are common and include dyspnea, faintness, and syncope. Chest discomfort may be accompanied by less specific symptoms such as nausea, burping, restlessness, or a sense impending doom. Frequency of the pain may be different.

An important characteristic is the relation to exercise, specific activities, or emotional stress. Symptoms classically triggered by increased levels of exertion, such as walking up an incline or against a breeze, and rapidly disappear within a few minutes, when these causal factors abate. Exacerbations of symptoms after a heavy meal or work are classical features of angina. Buccal or sublingual nitrates rapidly relieve angina.

For patient with stable angina it is useful to classify the symptoms using a grading system which was devised by the Canadian Cardiovascular Society, based on the severity of the angina stressor (tab. 2.3).

Table 2.3. ***Canadian Cardiovascular Society classification of stable angina***

Class	Severity of exertional stress including angina	Limitation ordinary activity
I	Strenuous rapid or prolonged exertion at work or recreation	None
II	Walking or climbing stairs rapidly, walking uphill, walking or stair climbing	Slight
III	Walking one to two blocks on the level and climbing one flight of stairs in normal condition and at a normal pace	Marked
IV	Symptoms may be present at rest	Discomfort in all activity performed

Objective examination. During the attack of stable angina the patient's condition is moderate, clear consciousness, standing up right position (if the patient walking), or sitting position with hand placed over sternum. Patient's face is pale with cyanotic tint. Arcus senilis, xanthelasma are revealed. Extremities are cold.

In auscultation of lung may be detected bilateral basal rales. Apex beat displaced outside. The left border of relative cardiac dullness displaced. Both heart sounds are decreased, paradoxically split S_2 and sometimes may be arrhythmia, premature beat, atrial fibrillation. The clinical features of stable angina are abnormal carotid pulse, decreased peripheral pulse, jugular venous distension. In some patients observe hepatomegaly, pedal edema.

Additional methods of examinations include laboratory investigation and non-invasive and invasive instrumental investigation. These tests are inquired in order to provide differentiative diagnosis.

Laboratory examination includes routine blood analysis. The full blood count incorporates total white cell count as well as hemoglobin. Serum creatinine is a simple method for evaluation of renal function. Fasting plasma glucose and fasting

lipid profile including total cholesterol, high density lipoprotein and low density lipoprotein cholesterol and triglycerides should be evaluated in all patients with suspected ischemic heart disease, including stable angina. If there is a clinical suspicion of instability biochemical markers of myocardial damage such as creatine kinase, creatine kinase MB, serum troponine T, or of the cardio-specific isoform of troponine-1 should be employed to exclude myocardial injury. Thyroid hormones should be examined if there is a clinical suspicion of a thyroid disorder. The standard diagnostic instrumental procedures in chronic stable angina include: X-ray examination, resting ECG, treadmill exercise ECG, echocardiography, stress echocardiography, photon emission computed tomography, coronary angiography, left heart catheterization with left vevtriculogram, tissue Doppler imaging.

Additional methods of examination

Clinical blood analysis is without change.

Biochemical analysis in patients with stable angina may show elevated level of cholesterol, triglycerides, decreased high density lipoprotein cholesterol and increased low density lipoprotein cholesterol. Biochemical markers of myocardial damage in stable angina are in a normal range.

X-ray examination in stable angina does not provide specific information for diagnosis.

- **Resting ECG** may show evidence of previous myocardial infarction, left ventricular hyperthrophy, bundle branch block, pre-excitation, arrhythmias, or conduction defects, but is normal in most patients. Since 12-lead ECG is normal in 50 % of patients with chronic stable angina it cannot exclude IHD. During chest pain the ECG becomes abnormal in half of the angina patients with a normal resting ECG. ST-segment and T-wave depression or inversion on the resting ECG and their pseudo normalization during pain are observed. Sinus tachycardia is common, bradyarrhythmia less go. These findings indicate that resting ECG should be performed during episode of chest pain.
- **Exercise ECG** is more sensitive and specific than the resting ECG for detecting myocardial ischemia. Exercise tolerance test is usually performed using a standard treadmill or bicycle ergometer protocol to ensure a progressive and reproducible increase in work load while monitoring the patient's ECG, blood pressure and general condition. Planar and down sloping ST-segment depression of 1 mm or more is indicative of ischemia; up sloping ST-depression is less specific and often occurs in normal individuals. An exercise test should be carried out only after careful clinical evaluation of symptoms and a physical examination including resting ECG. Exercise ECG testing is not of diagnostic value in the presence of left bundle branch block, paced rhythm, and Wolff-Par-

kinson-White syndrome in which cases the ECG changes cannot be evaluated. Additionally, false positive results are more frequent in patients with abnormal resting ECG in the presence of left ventricular hypertrophy, electrolyte imbalance, intraventricular conduction abnormalities, and use of digitalis. Exercise ECG testing is also less sensitive and specific in women.

- **Resting two-dimensional and Doppler echocardiography** is useful to detect or rule out the possibility of other disorders such as heart valve disease or hypertrophic cardiomyopathy as a cause of symptoms and to evaluate ventricular function. For diagnostic purposes, Echo-CG is useful in patients with clinically detected murmurs, history and ECG changes compatible with hypertrophic cardiomyopathy or previous myocardial infarction and symptoms or signs of heart failure. Tissue Doppler imaging allows regional quantification of myocardial motion and strain rate, imaging allows determination of regional deformation thus improve to detect ischemia earlier in the ischemic cascade.
- **Stress testing in combination with imaging** are used in the diagnosis of stable angina. The most well-established stress imaging techniques are echocardiography and perfusion scintigraphy. Both may be used in combination with either exercise stress. Exercise stress echocardiography has been developed as an alternative to "classica" exercise testing with ECG and as an additional investigation to establish the presence or location and extent of myocardial ischaemia during stress. A resting echocardiogram is acquired before a symptom-limited exercise test is performed, most frequently using a bicycle ergometer, with further images acquired where possible during each stage of exercise and at peak exercise.
- **Exercise testing with myocardial perfusion scintigraphy** is required. Thallium-201 and technetium-99m radiopharmaceuticals are the most commonly used tracers, employed with single-photon emission computed tomography in association with a symptom-limited exercise test on either a bicycle ergometer or a treadmill. With this technique myocardial hypoperfusion in patients with stable angina is characterized by reduced tracer uptake during stress in comparison with uptake at rest.
- **Pharmacological stress testing with imaging techniques.** Pharmacological stress testing with either perfusion scintigraphy or echocardiography is indicated in patients who are unable to exercise adequately or may be used as an alternative to exercise stress. Two approaches may be used to achieve this: infusion of short-acting sympathomimetic drugs such as dobutamine in an incremental dose protocol which increases myocardial oxygen consumption and mimics the effect of physical exercise or infusion of coronary vasodilators (adenosine and dipyridamole).

- **Cardiac magnetic resonance stress testing in conjunction with a dobutamine infusion** can be used to detect wall motion abnormalities induced by ischemia or perfusion abnormalities.

Acute coronary syndrome

Acute coronary syndrome (unstable coronary artery disease) includes both ***unstable angina*** and ***non-Q-wave myocardial infarction.***

Clinical features

Clinical features of acute coronary syndrome:

- increased severity or frequency of the patient's pre-existing angina within the last month;
- rapidly worsening chronic stable angina (crescendo angina);
- new onset of angina pectoris;
- angina at rest;
- post-infarction angina (more than 24 hours after myocardial infarction);
- non-Q-wave myocardial infarction.

Objective examination. During attack of chest pain the patient's condition is grave, forced sitting position, the face is pale with acrocyanosis. The border of relative cardiac dullness displaced outside.

In auscultation both heart sounds are decreased, S_3 or S_4 gallop may be detected during an episode of pain. Mitral regurgitation murmur appears. Arrhythmia is often observed. Blood pressure tends to have less level, than in period free of pain. The signs of congestion failure present: enlarged liver, pedal edema.

Additional methods of examination

Clinical blood analysis is without change, seldom may be slight leukocytosis.

Biochemical blood analysis: commonly there are the signs of disorders of lipid profile: increased level total cholesterol, triglycerides, low density lipoprotein cholesterol.

Small rises in the serum levels of biochemical markers of cardiac injury (creatine kinase, creatine kinase MB)), troponin-T or troponin-1 reflect the development of small foci of myocardial necrosis, minor creatine kinase, creatine kinase MB, which are usually accompanied by elevated troponin-T levels, indicate an increased risk of future events, despite stabilization of their clinical condition. Cardiac troponin-1 is not detectable in the absence of cardiac injury. Because of the lag period before a rise becomes detectable, at least two samples, taken at an interval of 12–24 hours, should always be tested.

Elevated fibrinogen levels at the time of admission are associated with an increased risk of death, myocardial infarction or spontaneous ischemia in patients with unstable angina.

The acute-phase proteins C-reactive protein is sensitive, but non-specific, markers of inflammation. There is much evidence to suggest a role for inflammation in the etiology of unstable angina and myocardial infarction and level of this protein have been observed to be elevated in some patients with acute coronary syndrome. C-reactive protein levels >3 mg/l, as detected by means of sensitive radioimmunoassay, indicate an increased risk of subsequent cardiac events in patients with acute coronary syndrome.

Instrumental examination. ECG monitoring is regarded as an essential part of routine management. All patients with suspected acute coronary syndrome should be admitted to the coronary unit for 12–24 hours of ECG monitoring (Holter monitoring). Admission ECG finding in acute coronary syndrome: ST-segment depression, ST-segment elevation (transient), T-wave inversion, normal ECG.

A normal ECG recorded when the patient is pain free not exclude the diagnosis of acute coronary syndrome, although a normal ECG recorded during an episode of pain makes the diagnosis unlikely, and is associated with an excellent prognosis. Following abnormalities of ECG support a diagnosis of acute coronary syndrome: ST-segment depression >0,5 mm, ST-segment elevation >1 mm, T-wave inversion. Transient elevation of the ST-segment which settles, either spontaneously or in response to nitrate treatment, is fully consistent with the diagnosis acute coronary syndrome. Isolated T-wave inversion on the initial ECG is a relative by benign sign, and is associated with a low risk of future myocardial infarction or death. A total of more than 60 minutes of ischemia during Holter monitoring is associated with a poor prognosis. However, T-wave inversion and change of ST-segment must be considered in the context of the whole clinical picture taking into account the patient's age, presence of other risk factors, levels of biochemical markers of cardiac injury.

Exercise testing undertaken either before or shortly after hospital discharge, is a minimum requirement for patients. Once the patient has been pain-free for 24–48 hours and the ECG stable the risks associated with performing an exercise test are very low. Severe ischemia and low exercise tolerance in a patient who has had either unstable angina or non-Q-wave myocardial infarction is associated with a poor short-term prognosis.

Echocardiography should be performed in all patients in order to evaluate the left ventricular function.

Stress echocardiography can be performed either during or immediately after dynamic exercise or under pharmacological stress administration of dipyridamole or dobutamine. Patients who are unable to perform an exercise test can be usefully assessed by pharmacological induced stress echocardiography.

Myocardial perfusion scintigraphy (tallium or technetium scan) may be particularly valuable in patients who are unable to exercise. Such techniques can outline perfusion defects.

Classification of unstable angina was proposed by E. Braunwald (tab. 2.4).

Table 2.4. ***Braunwald classification system for unstable angina (UA)***

Patients are assessed according to each of the following sets of criteria:

Severity of angina

Class I – New onset of severe angina or increased frequency of attacks
No rest pain

Class II – Angina at rest, sub-acute
Angina at rest within the past month, but not within the preceding 48 hours

Class III – Angina at rest, acute
Angina at rest during the preceding 48 hours

Clinical circumstances

Class A – Secondary UA
Symptoms secondary to an identified condition reducing myocardial oxyden suplly or increasing demand

Class B – Primary UA

Class C – Post-infarction UA

Intensity of treatment

Class I – Minimal or no therapy

Class II – Therapy for chronic stable angina

Class III – Maximum anti-anginal therapy, including intravenous nitrates

ECG changes

Presence or absence of transient ST, T abnormalities

Myocardial infarction

Myocardial infarction is formation of a necrotic foci in the heart muscle due to imbalance between upset coronary circulation and myocardial oxygen supply.

There are three pathophysiological events in development of myocardial infarction: rupture of an atheromatous plaque in a coronary artery, thrombus at a site of ruptured or intact plaque, local or generalized vasoconstriction. In myocardial infarction thrombus as a rule is occlusive. Under condition of complete occlusion of coronary artery the myocardial change occur after 20 minute and necrosis is formed for 6 hours. The time of formed necrosis is individual process which depends on presenting collateral circulation.

Diagnostics of myocardial infarction based on clinical features, ECG data and markers of tissue damage.

Clinical features

There are some clinical variants of myocardial infarction: angina (status anginous), abdominal variant, asthmatic variant, arrhythmic variant, cerebral variant, peripheral variant, painless or "silent" variant, combined variant.

Pain is the cardinal symptom of myocardial infarction. The pain resembles angina pectoris in patients with status anginous, but it usually more severe and often described as tightness, squeezing, pressing heaviness or constriction in the chest. The pain is characterized by inconstant character, lasts longer than angina, more than 20–30 minutes, some hours and even days. The pain irradiates in the left arm, throat, teeth, ear, under the left shoulder blade, sometimes in epigastrium. The chest pain is not relieved at rest or taking nitroclycerin. The pain is accompanied by feeling of fear, impending death, excitation, weakness, sweating and palpitation.

Objective examination. The patient's condition is severe, may be forced sitting position, consciousness is clear, pallor, excessive perspiration, cold peripheries, acrocyanosis. At second-third days of pain the temperature elevation till subfebril or febril level is observed. In percussion of the lung the intermediate sound is revealed in posterior part. Lung crepitation is heard. The borders of cardiac dullness correspond with preceding disease. Tachycardia is appeared as sign sympathetic activation. Decreased first heart sound or decreased both sounds are heard. At mostly patients presystolic and protodiastolic gallop rhythms occur. At 90–95 % of patients the extrasystoles are appeared. In the first listening point is heard loud pansystolic murmur which is explained by sudden onset of severe mitral incompetence with regurgitation due to the myocardial dysfunction or rupture of papillary muscle. A new loud pansystolic murmur may have another origin and caused by rupture of the interventricular septum with left-right shunting through a ventricular septal defect. Temporary, pericardial friction sound may appear at acute period of myocardial infarction as a rule in case of damage of anterior wall of the left ventricle.

Blood pressure can elevate in the period of pain attack. Sign of impaired myocardial function are hypotension, small pulse (pulses porous), oliguria. Sudden death, presumably from ventricular fibrillation or asystole, may occur immediately, within the first hour of chest pain.

According to clinical features and results Additional methods of examination five periods of myocardial infarction are distinguished: very acute, acute, subacute, recovery, stabilization. Acute period lasts approximately two days and characterized by diminished or disappeared chest pain. Nevertheless at this period may be the complications such as acute heart failure, disorders of cardiac rhythm and conduction, cardiogenic shock.

At the peak of first day at patient develops the syndrome related to the resurbtion of necrotic tissue. This syndrome includes elevated temperature, leukocytosis and accelerated ESR.

In case of bening course of disease at the subacute period the patient's condition becomes better, chest pain as usually absent, the heart sound louder, blood pressure remove to normal level. The signs of resorbtion syndrome disappeared. Pro-

longed leukocytosis and accelerated ECR indicate on accompanied complication, such as postinfarction syndrome or presence of inflammatory process as pneumonia, thromboflibites. At the period of recovery and stabilization the myocardial scar is formatted. The patient's condition is satisfactory, temperature is normal, tolerance to exercise load and physical activity are increased. The loudness of cardiac sound is slight decreased or normal. Heart rate is normal. Arrhythmia may preserve, but a number life-threatening arrhythmia is diminished. Hypertrophy of left ventricle reflects the cardiac remodeling in post infarction period.

Laboratory findings are normalized.

Atypical variants of myocardial infarction are particularly common in elderly and diabetic patients.

Abdominal type variant is observed more frequently at posterior diaphragmal myocardial infarction. This variant is characterized by intenstive pain in the epigastrium or in the right hypochondrium, which associated with dyspeptic disorders such as nausea, vomiting, regurgitation by air. Altered intestinal motility leads to diarrhea or constipation, paresis of intestine. On examination there is tenderness of the abdominal wall. Dangerous complication is acute gastrointestinal lesion and ulcer which are responsible for acute hemorrhage. The bleeding is often recurrent and caused shock.

Asthmatic variant is characterized by severe difficulty in breathing, cough with a foamy pink sputum (cardiac asthma, pulmonary edema) and small intensity of chest pain. There gallop rhythm, arrhythmias, decreasing of blood pressure is present. As a rule, this variant is more frequently observed at repeated myocardial infarction, and also at myocardial infarction on background of severe cardiosclerosis and practically always at the myocardial infarction of papillary muscle resulted the relative mitral incompetence.

Arrhythmic variant of myocardial infarction is predominated with disorders of rhythm and cardiac conduction, with slightly pain syndrome. This variant is related mostly with supraventricular or ventricular paroxysmal tachycardia, less frequent – paroxysmal atrial fibrillation or complete atrioventricular block. Arrhytmic variant may be complicated by cardiogenic shock with fall of blood pressure and sharply diminished myocardial perfusion.

Cerebral variant is observed in elderly patients with cerebral atherosclerosis and diminished brain circulation. Simultaneously with myocardial infarction may be spasm or thrombous of cerebral arteries. According to decreased cardiac output relevant with myocardial infarction such symptoms and signs of cerebral ischemia appear: giddiness, nausea and vomiting central origin, syncope, bradycardia, cramps and even, coma. Affection of central nervous system may be in a form of psychomotor anxiety resembles the clinical features of meningitis, epilepsy, polyneuropathy.

Painless, or "silent" variant of myocardial infarction pass unrecognized and may reveal afterwards during ECG recording or Echo-CG examination.

Course and outcomes of myocardial infarction depends on accompanied *complications*. In acute period may be such complications: disorders of rhythm and conduction, acute left ventricular failure (cardiac asthma, pulmonary edema), cardiogenic shock, acute aneurysm of left ventricle, rupture of the ventricle with cardiac tamponade and is usually fatal, pericarditis, thromboembolism, acute lesions and ulcers of gastrointestinal tract. In subacute period may observe: disorders of rhythm and conduction, chronic heart failure, chronic aneurysm of left ventricle, post-infarction angina, thromboembolism, post-infarction remodeling, post-infarction syndrome (Dressler's syndrome).

Nearly all patients with different variants of myocardial infarction have arrhythmias, which may be mild with favorable outcomes, but sometimes cause life threatening events. Ventricular fibrillation occurs in about 5–10 % of patients with myocardial infarction and is the major cause of sudden death. Atrial fibrillation is frequently transient state. Heart block complicating infarction is usually temporary and removes after specific treatment. Heart block complicating arterior infarction has unfavorable prognosis, because asystole may suddenly appear.

Cardiogenic shock – the most severe complication of myocardial infarction. Diagnostic signs of cardigenic shock: deranged consciousness, fall systolic blood pressure less 90 mm Hg, peripheral vasoconstriction and decreased volume of urine less 20 ml/hour. According to the leading mechanism there are three kind of shock: reflectory, arrhythmic, and true cardiogenic shock. Reflectory shock develops at patients with status anginous as a hemodynamic reaction on pain. Arrhythmical shock is resulting from paroxysmal tachycardia or cardiac blockade. True cardiogenic shock is explained by damage of cardiomyocytes, disorders of microcirculation and pronounced decreasing of contractile ability of left ventricle.

Heart failure complicating acute myocardial infarction indicates a bad prognosis. Cardiac asthma and pulmonary edema develop due to the acute left ventricular failure at approximately in up 10–15 % of patients and often lead to death. Classification of the acute heart failure at patients with myocardial infarction was proposed in 1967 by Killip. Four classes of acute heart failure are distinguished: 1 class – absence of pulmonary rales and gallop cardiac rhythm, this class develops at 40–50 % of patients and mortality is till 10 %. 2 class – presence of rales in less 50 % of lung areas or gallop rhythm, this class develops at 30–40 % of patients, mortality is till 20 %. 3 class – presence of rales in more, 50 % of lung areas associated with gallop rhythm, this class develop at 10–15 % of patients, mortality is till 40 %. 4 class – presence of cardiac shock, develops at 5–20 % of patients, mortality is till 50–90 %.

In approximately 10 % of patients full thickness myocardial infarction causes thinning of the infracted segment and develops the bulge at the left ventricle so called ***aneurysm***, revealed during inspection of the heart region as weak restricted pulsation in the III-IV intercostals spaces somewhat laterally from the left sternal edge.

Post-infarction angina occurs in up to 50 % of patients.

Thromboembolism is determined in different vessel sites with clinical features of stroke, pulmonary infarction and ischemic limb. Primary thrombus forms on the endocardial surface of freshly infarcted myocardium and transformed to systemic embolism.

The post-infarction syndrome (Dressler's syndrome) is an autoimmune reaction to necrotic process in myocardium and is characterized by persistent fever, pericarditis and pleurisy. The Dressler's syndrome occurs a few weeks or even month after the myocardial infarction.

Additional methods of examination

Clinical blood analysis – leukocytosis with mild nuclear shift to the left occurs in a few hours after onset of chest pain, reached the peak at 2–4 days and normalized in a week. The degree of leukocytosis depends on amount of damaged myocardial tissue. Accelerated ESR is observed at 2–3 days from onset of chest pain, reached maximal level till 2 week and normalized at 3–4 weeks.

Markers of myocardial infarction are plasma enzymes which are normally concentrated within cardiac cells. During the necrosis of cardiomyocytes their membranes destroyed and the enzymes released at first at microcirculation and later at systemic circulation. Thus myocardial infarction causes a detectable rise in the plasma enzymes which serve as laboratory markers of necrosis: creatine kinase, lactate dehydrogenase, aspartate aminotransferase, tropinin T and I, myoglobin. Optimal time for estimation of myocardial markers of necrosis depicted at table 2.5.

Table 2.5. ***Optimal time for estimation of myocardial markers of necrosis***

Markers	Optimal time for estimation of myocardial markers of necrosis
Myoglobin	In 1–2 hours after chest pain
Creatine kinase	Every 12 hours 3 time
Creatine kinase MB	In 60–90 minutes after chest pain, every 12 hours 3 time
Lactate dehydrogenase	In 24 hours after chest pain, one time
Troponin T	In 12 hours after chest pain, one time
Troponin I	In 12 hours after chest pain, one time

Baseline and peak elevation of markers of myocardial damage is different. Dynamic of laboratory markers of myocardial infarction is depicted at table 2.6.

Table 2.6. ***Dynamic of laboratory markers of myocardial infarction***

Markers	Norma	Time from onset of myocardial infarction		
		Baseline elevation hours	Peak elevation hours	Normalization days
Creatine kinase MB	0–4 ME/L	3–6	12–24	1,5–3
Lactate dehydrogenase	15–30 %	12–24	24–72	7–14
Aspartate aminotransferase	28–125 mmol/l	8–12	24–48	3–5
Troponin T, I	Less 0,1 mkg/l	3–12	12–48	3–16
Myoglobin	20–66 mkg/l	1–4	6–7	1

ECG: one of the most significant uses of a 12 lead ECG is to aid in determining whether a myocardial infarction has occurred.

The usual first finding in an infarction is elevation of the ST-segment, which occurs some hours after infarction. Hours to days later the T-wave inverts, diminution of the size of the R-wave and the Q-wave becomes deep and wide. The height of the R-wave is directly proportional to the amount of living tissue that escapes death. In case of full thickness myocardial infarction the R-wave is disappeared. Days to weeks later the ST-segment returns to near normal isoilectic line position. Weeks to moths later the T-wave becomes upright again, but Q-wave may remain abnormal. As the infarction heals the Q-wave may remain as the only sign of an old coronary occlusion. Since a deep and wide Q-wave is often indicate of an old infarction. The Q-wave may considered abnormal if it is over 0,03 second wide and if it is greater in depth than one fourth the height of the R-wave.

Echo-CG: two-dimensional echocardiography may assess the cardiac structures, pericardium and ascending aorta, allows identification of regional wall motion abnormalities, valvular abnormalities, global left and right ventricular function and detecting important complications such as cardiac rupture, ventricular septal defect, mitral regurgitation and pericardial effusion.

Radioisotope scintigraphy by technetium-99m-pyrophosphate. Scintigraphy is generally used for the diagnosis of myocardial infarction in patients hospitalized

late after the onset of symptoms in which cardiac enzymes are no longer elevated or are unreliable. Imaging is optimal 2–7 days after myocardial infarction. Focal increases in technetium pyrophosphate uptake are generally diagnostic of infarction. This technique is highly sensitive (>90 %) in detecting large transmural infarctien but is less reliable in the detection of small non-Q-wave myocardial infarction.

Radionuclide ventriculography allows to reveal right and left ventricular ejection fraction and assessment of regional wall motion abnormalities. Because radionuclide ventriculography provides less information regarding the cardiac structures, echocardiography is generally preferred in the initial evaluation of patients with myocardial infarction.

Sudden cardiac death

Sudden cardiac death (SCD) is defined as follows: "Natural death due to cardiac causes, heralded by abrupt loss of consciousness within one hour of the onset of acute symptoms; preexisting heart disease may have been known to the present, but the time and mode of death are unexpected". The key concepts that are central in the definition of sudden death are the non-traumatic nature of the event and the fact that sudden death is unexpected and instantaneous.

The single most important cause of death in the adult population of the industrialized world is SCD due to *ischemic (coronary) heart disease*. In patients with sudden cardiovascular collapse, the most often recorded rhythm shows that ventricular fibrillation is present in 75–80 % of cases, whereas bradyarrhythmias are thought to contribute to a minority of SCD. In about 5 % to 10 % of cases, SCD occurs in the absence of coronary artery disease or congestive heart failure.

Hypertrophic cardiomyopathy is a relatively common cardiac disorder (adult prevalence about 1:500) in which sudden unexpected death is the most devastating outcome, occurring throughout life, but particularly in young, often asymotomatic patients.

Dilated cardiomyopathy is the most common cause of death in dilated cardiomyopaty especially in less advanced functional classes. Ejection fraction has been repeatedly identified as best predictor of outcome both with regards to SCD and death from heart failure; occurrence of syncopal events is another accurate indicator of risk of SCD.

Long QT syndrome is associated with high risk of SCD. Risk stratification is mainly based on history of syncopal events, Torsades de Pointe or cardiac arrest.

Brugada syndrome. Diagnosis of Brugada syndrome is established in the presence of spontaneous or induced ST-segment elevation in leads V_1-V_3 with/without right bunde branch block.

Aortic stenosis. SCD occurs in about 20 % of patients whose deaths are attributed to aortic stenosis. In the absence of cardiac symptoms, survival is excellent

without valve replacement. The prognostic value of different hemodynamic and electrophysiologic testing is limited.

Wolff-Parkinson-White syndrome. In patients with Wolff-Parkinson-White syndrome natural history studies have reported SCD rate of 0,15 % /year due to ventricular fibrillation. SCD survivors tend to be symptomatic.

Bradyarrhithmias. It is estimated that 15–20 % of SCD may be attributed to bradyarrhithmias. Advanced AV block and intraventricular conduction disturbances represent a risk factor for bradyarrhithmic deaths.

Clinical features

Complains: giddiness, darkening in the eyes, sudden appearance of dyspnea.

Objective examination: grave condition, passive position, loss of consciousness expansion of pupils, appearance of pale-grey tint of skin, apnea, absence of heart sounds, absence of pulse on large arteries.

Program examination for the prevention of sudden coronary death:

1. Clinical examination of patients with IHD, detection of risk factors, reanimated in the acute period of MI with the heart failure; with angina pectoris at rest after the MI; with the complete blockade of bundle-brancle block.
2. Clinical analysis of blood, urinalysis.
3. Biochemical analysis of blood: total protein, transaminases, creatinphosphokinase, lactatdehydregenase, cholesterol, triglycerids, coagulogram.
4. ECG-Holter-monitoring.

Syndrome of myocarditis

Myocarditis – inflammatory diseases of myocardium of different etiology.

Classification of myocarditises according to etiology:

viral, bacterial, protozoan, mycotic, spirochetotic, rickettsial, vermin, caused by physical, chemical, toxic factors, allergic, transplantations, idiopathic.

According to course of disease:

- acute (duration to 3 months);
- subacute (duration from 3 to 6 months);
- chronic (duration more than 6 months).

According to severity of course:

- mild;
- moderate;
- severe.

Clinical features of mild course

Complaints: general weakness, moderately expressed, permanent heart pains, pricking or aching character, intermissions in heart, palpitations breathe less ness at the physical exertion.

Objective examination: the common state is satisfactory, there are no edema, cyanosis, shortness of breath. The heart borders are not changed, the decreased first heart sound, low systolic murmur on the apex of heart, pulse is normal or slightly frequented, sometimes arrhythmic, blood pressure normal.

Additional methods of examination

Clinical blood analysis: leukocytosis, accelerated ESR.

Biochemical analysis of blood: Moderate increase α_2- and γ-globulins, seromucoid, gaptoglobin levels in blood.

Immune analysis. The titers of antibodies increase to the viruses. Fourfold rise of titles of antibodies during the first 3–4 weeks is revealed.

ECG: inverted T-wave is determined, prolonged duration of PQ interval.

X-ray and Echo-CG without change.

Clinical features of moderate course

Complaints of patients: expressed weakness, heart pain squeezing character, quite often pricking, breathlessness, palpitations and intermissions in heart area, subfebril temperature of body.

Objective examination. Acrocyanosis, edema and orthopnoe are not present. The left border of heart is displaced to the left, first heart sound is decreased, functional systolic murmur is listened, sometimes pericardial friction, pulse is frequent, tachycardia, quite often arrhythmic, blood pressure normal.

Additional methods of examination

Clinical blood analysis: leucocytosis, shift in the leucocytic formula to the left, accelerated ESR, at viral myocarditis – leucopenia is possible.

Biochemical analysis of blood: increased levels of sialic acids, seromucoid, gaptoglobin α2- and γ-globulins.

Immune analysis. Positive reaction in presence the myocardium antigen, diminishing T-lymphocytes and T-suppressors amount, increased level of IgA and IgG.

ECG: premature contraction, atrial fibrillation or flatter, decreasing of ECG voltage, depression of ST-segment, appearance of negative, asymmetrical T-wave is possible; elevation of ST-segment due to pericarditis or subepicardial damages of myocardium; different degrees of AV-blockade.

X-ray of heart and Echo-CG indicates enlargement of heart and its chambers.

Clinical features of severe course

Complaints: dyspnea at rest, palpitations, intermissions and heart pains, pain in right subcostal area, leg edema, cough.

Objective examination. The common state is severe. Patients assume a forced position, orthopnoea. Expressed acrocyanosis, cold sweats, neck veins are swollen, edema of the low extremities is determined. At auscultation of lungs in lower parts – crepitation is heard. Heart borders are displaced to the right and/or to the left (due to concomitant pericarditis). Heart sounds are diminished, tachycardia, gallop rhythm, extrasystolia, may be paroxysmal tachycardia, atrial fibrillation, systolic murmur is determined over the apex, pericardial friction (at concomitant pericarditis). Pulse is frequent, weak filling, quite often threadlike, arrhythmical, blood pressure is reduced.

Additional methods of examination

ECG: premature contraction, paroxysmal tachycardia, atrial fibrillation or flatter are often registered, ST-segment is depressed, the negative T-wave is possible, the AV-blockade and bundle branch block.

X-ray examination: cardiomegaly.

Echo-CG reveals cardiomegaly, dilatation of different chambers of heart, decreasing of the cardiac output, signs of total hypokinesia of myocardium.

Syndrome of pericarditis

Pericarditis – inflammation of visceral or parietal lairs of pericardium.

According to etiology classification

I. Infections:

- rheumatic fever;
- tuberculosis;
- bacterial infection;
- protozoa;
- mycotic;
- viral;
- rickettsial.

II. Aseptic pericarditis:

- allergic;
- diffuse pathology of connective tissue;
- the diseases of blood;
- malignant tumors;
- traumatic;

– autoimmune;
– metabolic disorders – uremia, gout;
– treatment of steroids;
– hypovitaminosis C.

III. Idiopathic pericarditises (unknown etiology).

Clinical classification of pericarditis

A. Acute pericarditis:
– dry (fibrinous);
– exudative: a) with heart tamponade, b) without heart tamponade.
B. Chronic pericarditis:
– exudative;
– adhesive.

Pathogenesis of pericarditises depends on their etiology.

Infectious pericarditises are related to penetration of microorganisms in the cavity of pericardium by hematogenic, lymphogenic way from at the neighbouring organs. Development of infectious-allergic pericarditis is connected with the allergic reaction (general or local) in response to acute or chronic infection. Pericarditis at myocardial infarction arises up as reactive inflammation of pericardium due to necrosis or because of autoimmune reactions (Dressler's syndrome). At the diseases of the connective tissue in pathogenesis of pericarditis main role belongs to autoimmune mechanisms. Combination of infectious, infectiously-allergic, autoimmune and toxic mechanisms is possible in number of cases.

Pericarditis may produce a pericardial effusion which, depending on etiology, may be fibrinous, serous, hemorrhagic or purulent character.

A fibrinous exudates may eventually lead to varying degrees of adhesion whereas serous pericarditis often produces a large effusion of turbid, straw-colored fluid with a high protein content.

A hemorrhagic effusion is often due to malignant disease, particularly carcinoma of the breast, bronchus and lymphoma.

A purulent pericarditis is rare and may occur as a complication of septicaemia, by direct spread form an intrathoracic infection, or form a penetrating injury.

Dry pericarditis

Clinical features:

– pain in heart of different intensity (from the easy pricking to very intensive) and duration, not relieves after taking nitroglycerine, increase at cough, sneeze, breathing, a change of position;

- vomiting is possible as a result of irritation of the diaphragmal nerve;
- sweats, subfebril temperature of body;
- in auscultation pericardial friction of scraping character (resembles the crunch of snow), is listened during systole and diastole, it better heard in the area of absolute dullness of heart, increased at pressing by stetoscope.

Additional methods of examination is determined shift of leucocytes formula to the left.

ECG: concordant depressed ST-segment in the standard and chest leads with appearance high positive T-wave. This displacement of ST-segment persists two days, than ST-segment goes down to isolinea, gradually positive T-wave becomes flat or negative.

Exudative pericarditis

Clinical features

The clinical features depend on main pathology, amount of liquid in pericardial cavity and duration of the pathological process. The complaints are:

- expressed permanent dyspnea;
- signs of compression of the adjacent organs and nerves (dysphagia, dry cough, changing of voice):
- weakness, decreased work capacity.

***Objective examination*:** forced position of patient – sitting with inclination forward. Due to the compression of v. cava superior with exudates occurs swelling of veins of neck, increased on inspiration. Edema and cyanosis of face, neck appears. The liver enlarges and becomes painful, ascites appears, edema of extremities appears.

Examination of the respiratory system. At percussion of lungs over the left lower lobe the decreased vocal fremitus and dulled sound are determined. At auscultation in left subscapular region the vesicular breathing is diminished, and may heard the bronchial tint of breathing due to with swelling of pulmonary tissue by pericardial exudate.

Examination of the cardiovascular system: weakened or disappearance of apex beat. In the heart region the intercostal spaces are smoothed. Expansion of area of cardiac dullness in both sides and borders of vascular bundle in second intercostal space are detected by percussion. Configuration of heart resembles a trapezium. In percussion of the heart region the area of absolute dullness of heart becomes equal to the relative dullness.

In auscultation the heart sound diminished, in grate amount of liquid in pericardial cavity may be inaudible.

Pulse is small, frequent, quite often arrhythmical, pulsus paradoxus (an excessive fall in blood pressure during inspiration).

Blood pressure is reduced.

Signs of tamponade of heart: sharply expressed dyspnea; expressed edema of face and neck, cyanosis, cold sweats; swelling of neck veins, peripheral edema, pulse is small, rapid, arrhythmical; blood pressure falls (to a collapse); quickly increasing ascites, hepatomegaly.

Additional methods of examination

Clinical blood analysis: increased amounts of leucocytes and shift of leukocytic formula to the left.

Biochemical analysis of blood: increasing the level of α_2-globulins, fibrinogen, gaptoglobin.

Examination of pericardial liquid (pericardial puncture). The gravity of pericardial liquid is 1,018–1,020 g/l level albumen exceeds 30 g/l, the Rivalt's reaction is positive, neutrophils (if pericarditis develops after the pneumonia or after other bacterial infection) or lymphocytes (at the tubercular etiology) are present in pericardial liquid. The atypical cells are revealed in malignant pericarditis, at lymphogranolumatosis – Beresovskiy-Shteinberg cells are detected. At lupus etiology of pericarditis the lupus cells are revealed in exudate.

ECG: declining at voltage of all waves, T-wave becomes negative, ST-segment may be depressed or elevated.

X-ray examination informative in presence of 200–400 ml of liquid in pericardial cavity: enlarged size of heart; transition of acute hepatic-cardiac angle in dull; disappearance of waist of heart.

Ultrasound is a useful and very sensitive method for the detecting a pericardial effusion – presence of liquid is in the cavity of pericardium.

Chapter10

DYGESTIVE SYSTEM

Syndrome of jaundice

The syndrome of jaundice is one of the most widespread syndromes of the digestive system pathology that based on the significant hyperbilirubinemia and bilirubin accumulation in the tissue and skin.

Etiology

Depending on the etiological factor there are the next forms of jaundice:

A. Exogenic (false) jaundice or xanthosis.

B. Endogenic (true) jaundice:

I. Suprahepatic (hemolytic):

1. Hereditary hemolytic anemia (talassemia, Minkovskogo-Shofara).
2. Acquired hemolytic anemia (autoimmune, posthemotransfusion).
3. Increased erythrocytes hemolysis on different diseases:

– infections;
– burns;
– tumors;
– hemorrhages (hematoms, infarctions);
– on diseases with deranged erythropoesis (B_{12}-deficiency anemia, primary erythrocytosis, sideroplastic anemia).

II. Hepatic (parenchimatous):

1. Liver disease:

– different types of hepatitis;
– liver cirrhosis;
– tumor of the liver;
– Gilber's syndrome;
– Kriglera-Nayara syndrome;
– Dabina-Dgonsona syndrome;
– Rotor syndrome.

2. Toxico-allergic and immune liver affection.
3. Metastatic affection of the liver.
4. Pathology of the bile ducts:

– primary biliary cirrhosis;
– cholangitis;
– intrahepatic cholestasis.

III. Subhepatic (mechanical):

1. Mechanical jaundice with tumor genesis:
- cancer of the pancreas;
- cancer of the major duodenal papilla;
- cancer of the bile bladder;
- cancer of the extra hepatic bile ducts
2. Mechanical jaundice with non-tumor genesis:
- calculus cholecystitis.

Pathogenesis

Depending on the causes there are the next mechanisms of jaundice:

A. Exogenic (false) jaundice or xanthosis:

Xanthosis related with prolonged using of carotin (carrots), oranges, tangerines and administration of ethacridine lactate (rivanol), picric acid.

B. Endogenic (true) jandice:

I. ***Suprahepatic jaundice*** (*icterus colore citricoluteo s. icterus suprahepatica*) occurs due to the excessive hemolysis of erythrocytes in the cells of the reticulohistocytic system (spleen, liver, bone marrow). Hemoglobin brakes down to the globin and hem. Bilirubin is formed from the released hem and accumulates in blood. Observe in malaria, sepsis, poisoning by hemolytic substances, inherited or acquired hemolytic anemia;

II. ***Hepatic jaundice*** (*icterus colore rubiginoso s. icterus hepatica*) occurs due to the damage of hepatocytes and disorders of their function (inversion of unbound bilirubin to bound), observe in acute and chronic hepatitis, poisoning;

III. ***Subhepatic jaundice*** (*icterus colore luteoviridi s. icterus infrahepatica*) occurs due to the accumulation of bilirubin (the product of gradual oxidation of bilirubin) resulted from partial or complete obstruction of the common bile duct in patients with stones in the gall bladder, cancer of the head of the pancreas, cancer of the major duodenal papilla.

Clinical features

The main complaints in patients with jaundice are yellow color of the skin and visible mucosa, bitterness in a mouth, nausea, skin itch and eruptions.

Thus, initial and moderate yellow skin is named subicterus, pronounced yellow color defines as jaundice. According to their etiology pathological jaundice associated with next particularities:

On suprahepatic jaundice – it is characterized by lemon-yellow tint, moderate intensity without itching of the skin and hematological signs of anemia and hyperbilirubinemia.

On parenchymatous jaundice (hepatic jaundice) – it is characterized by orange-yellow tint, presence of anamnesis data specifying for viral hepatitis or other liver diseases; the references on medicines taken, influence of nutritious or toxic factors and associated with the signs of hepatobiliary system affection (asteno-neurotic and dyspeptic syndromes), hyperthermia, hepatomegalia or hepatosplenomegalia. In laboratory methods of examination observed hyperbilirubinemia, hyperfermentemia, urobilinogenuria, bilirubinuria, decrease of stercobilin level in the stool.

On obstructive jaundice (subhepatic jaundice) – it is characterized by greenish-yellow tint, with early appearance of skin itching (may be before jaundice manifestation). Moreover, in mechanical jaundice with tumor genesis the patients have complaints on the symptoms of general tumor-induced intoxication (anorexia, loss of weight, est.), dull pain in epigastria and hypochondria regions, stable acholia and choliuria, observed hepatomegalia or hepatosplenomegalia and the signs of cytolysis (moderate hyperfermentemia). In mechanical jaundice with non-tumor genesis are specific the references on chronic calculus cholicistitis or biliar colic in anamnesis, bad tolerance of fat food, appearance of jaundice after painful attack with fever, vomiting, significant pain in right hypochondrium region and positive Ortner's symptom, devoid of splenomegalia, more seldom of hepatomegalia. In some cases the skin becomes yellow pallid due to hemorrhage from varicose esophageal or hemorrhoid vein in portal cirrhosis of the liver.

Objective examination. *General patients condition* may be from satisfactory (early stage of the disease) to extremely grave. Due to the acute or gradual intoxication may be observed the deranged consciousness.

The posture of the patients is frequently active. May be observed the passive posture in situation of hepatic coma develops.

The color of the skin and visible mucosa is characterized by jaundice with different tint and intensity, observed scratching of the skin.

The results of inspection, palpation, percussion and auscultation depend on the primary disease.

Additional methods of examination

Clinical blood analysis: anemia, leukocytosis, neutrophilia and accelerated ESR.

Clinical urine analysis: the color is greenish-yellow or greenish-brown (beer-like), odorless, bilirubinuria and urobilinogenuria.

Biochemical blood analysis. *In suprahepatic jaundice*: bilirubinemia – increased of total bilirubin mainly due to the unbound bilirubin; dysproteinemia, positive thymol test; elevated aldolase; alanine aminotransferases;

In hepatic jaundice: bilirubinemia – increased of total bilirubin due to the unbound and bound fractions; dysproteinemia, positive thymol test; elevated pro-

thrombin index (in significant degree of hepatic-cellular failure – decreased), decrease of total cholesterol, increased concentration of aldolase, alanine aminotransferases, aspartate aminotransferases, alkaline phosphatase, lactate dehydrogenase, cholinesterase, sorbitol dehydrogenase and ceruloplasmin.

In subhepatic jaundice – increased of total bilirubin mainly due to the bound bilirubin; dysproteinemia, negative thymol test; elevation of prothrombin index, significant increase of total cholesterol and β-lipoproteins, moderate (non-obligatory) increase of *alanine aminotransferases*, aspartate aminotransferases, alkaline phosphatase and ceruloplasmin.

Combination of changes of enzyme activity gives important diagnostic information:

- increased activity of alanine aminotransferases, aspartate aminotransferases, alkaline phosphatase, lactate dehydrogenase, cholinesterase, sorbitol dehydrogenase and hyperbilirubunemia with predominance of bound fraction indicates mainly necrotic damage of the liver (hepatocytolisis, liver necrosis) – *cytolisis syndrome*;
- increased activity of alkaline phosphotase in combination with hyperbilirubunemia, hypercholesterolemia, and β-lipoproteinemia is typical to cholestatic hepatitis, long-standing and grave obstructive jaundice – *cholestatic syndrome;*
- decreased albumins, cholesterol, protrombin contents in combination with hyperbilirubinemia is characteristic of *hepatic-cellular failure syndrome*;
- increased γ-globulins level, hyperproteinemia, elevated thymol test and immunoglobulins G and M contents are typical to immunoreactive damage of the liver – *immunoinflammatory syndrome*.

Determination of total protein and its fraction in the blood serum:

- may be detect: increasing of total protein level (chronic diseases of the liver) or decreasing of the total protein level (grave affection of the liver with hepatic-cellular failure);
- on damage of the liver parenchyma (chronic hepatitis, liver cirrhosis, liver cancer) observed albumin levels increasing in the blood serum and its intensity depends on the injury degree.

Globulins levels increasing reflect activity of chronic hepatitis and indicate immunoinflammation:

- α_1-globulins (reaction of the acute phase) – acute, subacute and chronic inflammatory process, malignant tumor;
- α_2-globulins – subacute and chronic inflammatory processes, hemolysis, malignant tumor;
- β-globulins – primary and secondary hyperlipoproteinemia;
- γ-globulins – chronic diseases of the liver, chronic infections, autoimmune hepatitis, liver cirrhosis, chronic active hepatitis.

Coprological study (macroscopic, chemical, microscopic and bacteriological studies). In macroscopic study: the color of the faeces is grayish-white, clayish, or sandy ("acholic faeces") – in the disease of the liver and bile ducts with upset bile excretory function. In achylia, gastroentestomy connective tissue in a form of whitish or grayish fibrous structures can be found. Also after biliary colic, sometimes in some days after it, and in rare cases without preliminary colic can be detect concrements, which are of bilious, pancreatic or intestinal origin.

In chemical study: reaction of the faeces becomes alkaline in achylia, pancreatitis due to significant creatorrhea and putrid flora is activation. Absence of stercobilin in the stools (acholic stool) in obstructive jaundice is observed in complete obstruction of common bile duct (obturation by stone, tumor). Sharp decreasing or absence of stercobilin in jaundice suggests grave parenchymatous damage of the liver. Increase stercobilin content in the stool are found in intensification of erythrocytes hemolysis (anemia).

Microscopic study: can be without particularities.

In order to establish the etiology is required the examination of viral hepatitis markers in the blood.

Laboratory non-obligatory methods (under indication):

- immunoglobulins and immune complexes detection;
- coprological study with protozoa and helminthes detection: should be better detected in fresh stool. Such helmints as ascarides (female length is 15–45 cm, male – 15–25 cm), acanthocephala, and platyhelmints can be seen by an unaided eye in stools. Ova of following helminthes can be revealed: Taenia solium, Taenia saginata, Taenia nana, Botriocephalus latus, Distomum hepaticum, Ascaris lumbricoides, Enterobius seu oxyuris vermicularis, Trichocephalus dispar, etc. Telemann's method is more effective to evaluate the presence of helminthes ova in the stools. Cysts of following protozoa: Amoeba dysenteria seu histolitica, Lamblia, Balantidia can be identified by staining with Lugol's solution;
- evaluation of B_{12} and folic acid level in the blood;
- detection of C-reactive protein, TNF-α and other proinflammatory cytokines activity in plasma or serum;
- evaluation of antinuclear and antymitochondrial antybodies titer; antybodies titer to smooth muscles or microsomal /renal/hepatic/ atybodies 1-type;
- detection of oncomarkers (α-phet protein).

Instrumental obligatory methods. X-ray examination and radiography. Plain abdominal radiographs may provide help to obtain the required information in the majority of cases. Barium sulphate suspension continues to be widely used for routine examination of the gastrointestinal tract.

Barium-meal examination of the small intestine is normally made following examination of esophagus, stomach and duodenum. It is indicated when disorders causing morphological changes in the small intestine, such as Crohn's disease, tuberculosis, neoplasms, radiation damage, ischemia or diverticulum, are suspected. The functional properties can be also assessed by x-ray study. Cancers of the small intestine have no specific radiological patterns.

Enlargement of the liver may be identified on plain radiographs, but adds little to clinical examination. Calcification is occasionally seen in the liver and the most common causes are old granulomatous disease and hydatid cyst.

Plain radiographs are normally the initial diagnostic procedure in patients with acute symptoms of disease of the biliary tract. The plain film may show pathological calcification of the gallbladder, opaque calculi, gas in the biliary tree, or radioopaque bile in the gallbladder.

The plain radiograph of the upper abdomen is an important investigation in patients presenting with symptoms of acute or chronic disease of the pancreas. In acute pancreatitis it may show the colon 'cut-off' sign, a sentinel loop, evidence of displacement of the stomach, or of a pancreatic abscess. Pancreatic calcification may be seen in patients with chronic pancreatitis.

The plain radiograph will usually show an enlarged spleen as is found in blood disorders of the reticuloendothelial system, infection, hepatic cirrhosis, and trauma.

Ultrasound examination of the digestive organs. Ultrasound examination of the liver is safe, cheap, and accurate in experienced hands. Abscesses appear as black transonic areas surrounded by high-intensity echoes, whilst cysts have black transonic areas surrounded by a thin echogenic rim.

Neoplasia produces areas of discontinuity in the homogeneous pattern of the liver. Most commonly, the echo amplitude is less than that of the surrounding liver, but some metastases, particularly from the colon, produce high-intensity echoes.

Direct ultrasonography of the liver exposed at surgery may show lesions not visible by the normal transcutaneous technique. Cirrhosis produces higher amplitude of echoes than does the normal liver and a large portal vein may be demonstrated. Colour-flow Doppler can help to differentiate hemangiomas from other neoplasms and is invaluable in the assessment of the portal and hepatic veins. Diagnostic biopsy of liver tumors is greatly facilitated by ultrasound control.

High-definition sector scanners provide an excellent real-time image of the gallbladder, and the intrahepatic and extrahepatic bile ducts. The small probe can be used between the ribs and allows scanning with the patient erect. The accuracy in detecting gallstones is similar to that of oral cholecystography with the added advantage that the bile ducts may be examined at the same time. Ultrasonography has replaced oral cholecystography as the method of choice for detecting biliary-tract

calculi and as the initial investigation for suspected gallbladder disease. A thickened gallbladder wall is sometimes seen in acute cholecystitis. Dilated hepatic and common bile ducts may be identified and, if the bowel is relatively free from gas, intraductal calculi or an enlarged head of pancreas may be identified.

Ultrasonography allows the measurement of size and the visualization of parenchyma of the pancreas. Acute pancreatitis, neoplasms, and pseudocysts may be identified and, if a neoplasm is diagnosed, it may be biopsied using a Chiba needle guided by ultrasound. Ultrasound is often used to investigate epigastric masses with the advantage that other organs in the region of the pancreas including the aorta, para-aortic lymph nodes, and adrenal glands may be seen. Peroperative ultrasonography may identify insulinomas not identified by other techniques. Ultrasound may be used to measure spleen size and in identifying spleen cysts.

Instrumental non-obligatory methods (under indication). Angiography – continues to have a role in the investigation of gastrointestinal bleeding and in the preoperative evaluation of the liver and pancreas. Coeliac-axis angiography is useful in identifying hemangiomas, as they have vessels of normal size but with a slow flow of contrast through the lesion. Angiography will help to differentiate hepatic tumors and may be done during computed tomography to maximize the detection of metastases, but is more commonly used to identify the exact site and blood supply of neoplasms before partial hepatectomy. Coeliac angiography will usually show insulinomas of the pancreas, but transhepatic portal catheterization will allow samples of blood to be taken from the splenic vein to localize insulinomas if computed tomography or angiography is unsuccessful.

Percutaneous transhepatic cholangiography is widely used for demonstrating the bile ducts in obstructive jaundice.

Endoscopic retrograde cholangiopancreatography is frequently used to demonstrate the ducts in patients with obstructive jaundice. Endoscopic sphincterotomy can be done during the procedure, thus allowing bile-duct calculi to pass freely into the duodenum, and frequently relieving bile-duct obstruction due to calculi.

Radionuclide studies can also be used to locate the site of obscured bleeding from the gastrointestinal tract. The general anatomical location of bleeding can be identified in many patients and further investigations such as angiography or barium studies can then be made to define the site and cause of bleeding more precisely.

Ultrasonography and computerized tomography have largely replaced radionuclide studies of the liver.

The spleen can be examined by radioisotopes and this may be particularly useful in trauma or where a small splenunculus is suspected.

Computed tomography gives the possibility to detect metastatic disease and primary neoplasia of the liver. Cysts and abscesses also show well on computed

tomography, but cirrhosis may be difficult to identify with certainty. The ability of high-resolution computed tomography to detect small intrapancreatic pseudocysts, pseudocysts containing gas or solid contents, pancreatic calcification, and peripancreatic fascial thickening make it the most accurate method for evaluating pancreatitis. Accurate assessment of carcinoma of the pancreas is also possible with computerized tomography.

Magnetic resonance imaging. This technique is excellent for differentiating malignant neoplasms from benign cysts, and for providing useful information in patients with cirrhosis and with metastatic deposits. The improved sensitivity produced by the use of contrast agents, the elimination of artifacts, and the characterization of tissue both by magnetic resonance imaging and magnetic resonance spectroscopy provide a useful, radiation-free adjunct to ultrasonography and computerized tomography.

Cholecyctography. About 80 to 85 per cent of gallstones are not radio-opaque and oral cholecystography remains the method of choice for examining the gallbladder with contrast medium to detect calculi when ultrasound is not available or is inconclusive. Abnormalities causing a change in the outline of the gallbladder such as adenomyomatosis are well demonstrated at oral cholecystography.

Syndrome of bile ducts dyskinesia (dysfunctional bile tract disorders)

The syndrome of bile ducts dyskinesia is the complex of clinical symptoms which developing connects with moto-tonic dysfunction of gallbladder, bile ducts and sphincters.

Dysfunctional bile tract disorders include not coordinated, untimely, insufficient or excessive reduction of a gallbladder and sphincters (Oddy, Lutkensa and Mirricy). More frequent this pathology observed in young age women with labile mentality, unsatisfactory nutrition and asthetic constitution Dyskinesia of Oddy's sphincter is specific for 1,5 % of patients suffering from cholecystitis and for 14 % of patients with postcholecystectomic syndrome. In 33 % of patients suffering from Oddy's sphincter dysfunction, this disorder accompanied with distal part of common bile duct obstruction.

Etiology

I. Primary dysfunctional bile tract disorders (genetically grounded decrease of muscular mass, decrease of receptors apparatus sensitivity to neurohumoral stimulation);

II. Secondary dysfunctional bile tract disorders:

- digestive system pathology;
- endocrine system pathology;

– nervous system pathology;
– pregnancy;
– non-rational nourishment.

Pathogenesis

The primary role in the syndrome of bile ducts dyskinesia formation is belongs to impaired synchronization of gall bladder and sphincters work via sympathetic and parasympathetic nervous system dysregulation.

Nervous system dysregulation may be provoked by psychogenes factors: psychoemotional overload, stress, neurosis. Vegetative nervous system dystonia, increasing or decreasing of vagus/sympathetic impulses, hypothalamic disorders leads to the not coordinated reduction of a gallbladder and sphincters (Oddy, Lutkensa and Mirricy) relaxation and bile passage dilation.

The major role in dysfunctional bile tract disorders plays endocrine pathology that accompanied with insufficiency of thyroid gland and sex gland hormons, oxythocin and glucocorticoids. Also to the syndrome manifestation involves the particularities of the diet, character and regime of nutrition.

Classification

Depending on the etiological factor, localization and functional state there are the next forms of dysfunctional bile tract disorders:

I. According to the etiology:

1. Primary dysfunctional bile tract disorders (hereditary decrease of muscular tone, decrease of receptors apparatus sensitivity to neurohumoral stimulation);
2. Secondary dysfunctional bile tract disorders.

II. According to the localization:

1. Dysfunction of the gall bladder;
2. Dysfunction of the Oddy's sphincter.

III. According to the functional state:

1. Hyperfunction;
2. Hypofunction

Clinical features

In hyperkinetic form of the gall bladder or/and bile ducts dysfunction the main complaints are: pain in the abdomen and clinical signs of neurotic syndrome.

Pain in the abdomen – is periodic, recurrent, colic like, localized in the right hypochondrial region with radiation to the back, right scapulae and right shoulder, aggravated more frequent at night-time after improper feeding, alcohol, augment physical or psychical activity.

The clinical signs of neurotic syndrome include – irritability, fatigue, perspiration, tachycardia, and headache.

In hypokinetic form of the gall bladder or/and bile ducts dysfunction the main complaints are: pain in the abdomen, clinical signs of neurotic and dyspeptic syndromes.

Pain in the abdomen is periodic, recurrent, has dull holding apart character with localization in the right hypochondrial region and radiation to the back, right scapulae and right shoulder, aggravated during bending of body and at night-time after improper feeding, alcohol, augment physical or psychical activity.

The clinical signs of neurotic syndrome include – irritability, fatigue, perspiration, tachycardia, and headache.

The clinical signs of dyspeptic syndrome include – bitterness in a mouth, nausea, vomiting and difficult defecation.

Moreover, secondary dysfunctional bile tract disorders are accompanied by the symptoms of the basic disease.

Objective examination. *General patient's condition* as usual satisfactory, consciousness is clear, posture is frequently active or active with restriction in cause of intensive biliar colic.

The color of the skin and visible mucosa has corporeal color (*cutis coloris somatici*), without eruption, moderate moisture and elasticity, preserved turgor, may observe transient subicteria of the skin.

The results of inspection, palpation, percussion and auscultation of respiratory and cardiovascular systems are without particularities.

In superficial tentative oriental palpation of the abdomen detect moderate pain in right hypochondria. Muscular resistance, diastases recti, and fluctuation symptoms are negative.

In penetrative palpation of the abdomen identify tenderness in gall bladder point (Ker point).

Additional methods of examination

Clinical blood analysis: without pathological changes.

Clinical urine analysis: without pathological changes;

Biochemistry blood analysis: increased activity of alkaline phosphotase and aspartate aminotransferases (more than in twice during two-multiple analisis), in combination with pancreatic ferments elevation (amylasa, lipasa), hyperbilirubunemia with predominance of bound fraction.

Medicament test (morphincholeretic test Debrea or morphinneostigmin test Nardy) – provocation of typical bile colic.

Ultrasound examination of the digestive organs. With obligatory evaluation of functional gall bladder state (use of bile discharge stimulated breakfast – 29g. sorbitol in 100ml water) – specific constriction of gall bladder less than 40 %, increase of choledoch diameter more than after fat food.

Endoscopy. Endoscopic visualization of the biliary tree is now the best diagnostic procedure for stones, tumors, and strictures of the bile duct and is the only reliable means of diagnosing primary sclerosing cholangitis. Furthermore, it offers the therapeutic procedures of sphincterotomy, stone withdrawal, and the insertion of stents across strictures.

In patients with dysfunctional bile tract disorders the endoscopic sign are: edema and stricture of duodenal papilla.

Study of duodenal secretion. Reduction of gall bladder reflex (amount of bladder bile increase to 100–150ml in norm 30–70ml; the bile excreted by little portions; dilation of bile discharge more than 45min).

Syndrome of gastrointestinal bleeding

The syndrome of gastrointestinal bleeding includes the states of gastrointestinal system damage that accompanied with blood allocation and clinically manifested in *hematomesis, melena and hematochezia.*

Hematomesis is defined as the vomiting of blood, melena as the passage of black and tarry stools due to the presence of blood and *hematochezia* – the passage of red blood per rectum.

Classification

Depending on the localization, duration and functional state there are the next variants of gastrointestinal bleeding:

I. According to the localization:

- upper gastrointestinal hemorrhage;
- lower gastrointestinal hemorrhage.

II. According to the duration:

- acute;
- chronic.

III. According to the functional state:

- compensated;
- decompensated.

IV. According to the intensity:

- profuse bleeding;
- latent (occult) bleeding.

Etiology

The most common causes of upper gastrointestinal hemorrhage are:

1. Erosive or hemorrhagic gastropathy
2. Duodenal ulcer

3. Gastric ulcer
4. Mallory-Weiss tear
5. Varices or portal hypertensive gastropathy
6. Arteriovenous malformations

The most common causes of lower gastrointestinal bleeding

Under age 55	Over age 55
Anorectal disease (hemorrhoids, fissures)	Anorectal disease (hemorrhoids, fissures)
Colitis (inflammatory bowel disease)	Diverticulosis
Diverticulosis	Angiodysplasia
Polyps, cancer (hyperplastic, hamartomas)	Polyps, cancer
Angiodysplasia	Enterocolitic (ischemic, infectious, inflammatory bowel disease, radiation)

Pathogenesis

Hematemesis usually indicates bleeding proximal to the ligament of Treitz.

While bleeding sufficient to produce hematemesis usually results in melena, less than half of patients with melena have hematemesis.

Melena usually denotes bleeding from the esophagus, stomach, or duodenum, but lesions in the jejunum, ileum, and even ascending colon may occasionally because melena provided the gastrointestinal transit time is sufficiently prolonged. Approximately 60 mL of blood is required to produce a single black stool; acute blood loss greater than this may produce melena for as long as 7 days. After the stool color returns to normal, tests for occult blood may remain positive for over a week. The black color of melena results from contact of the blood with hydrochloric acid to produce hematin. Such stools are tarrying ("sticky") and have a characteristic odor. This tarry consistency is in contrast to black or dark gray stools occurring after the ingestion of iron, bismuth, or licorice.

Hematochezia, the passage of red blood per rectum, generally signifies bleeding from a source distal to the ligament of Treitz. However, brisk proximal bleeding can cause hematochezia due to rapid transit.

Gastrointestinal bleeding, even if detected only by positive tests for occult blood, indicates potentially serious disease and must be further investigated.

Clinical features

The clinical manifestations of gastrointestinal bleeding depend on the extent and rate of hemorrhage and the presence of coincidental diseases.

The color of vomited blood depends on the concentration of hydrochloric acid in the stomach and the duration of its contact with the blood.

Thus, if vomiting occurs shortly after the onset of bleeding, the vomits appear red, and later the appearance will be dark red, brown, or black. Precipitated blood clots and acid-degraded blood in the vomits will produce a characteristic "coffee grounds" appearance when vomited.

Blood loss of less than 500 ml is rarely associated with systemic signs; exceptions include bleeding in the elderly or in the anemic patient in whom smaller amounts of blood loss may produce hemodynamic alterations.

Rapid hemorrhage of greater volume results in decreased venous return to the heart, decreased cardiac output, and increased peripheral resistance due to reflex vasoconstriction. Orthostatic hypotension greater than of 10 mm Hg usually indicates a 20 per cent or greater reduction in blood volume. Concomitant symptoms may include lightheadedness, syncope, nausea, sweating and thirst. When blood loss is 25 to 40 per cent of blood volume, shock frequently ensues with pronounced tachycardia and hypotension. Pallor is prominent, and the skin is cool. However, in the presence of beta-adrenergic and calcium channel blockers, these clinical signs may be blunted.

Additional methods of examination

Clinical blood analysis: common laboratory findings include anemia, mild leukocytosis and thrombocytosis, which develop within 6 h after the onset of bleeding.

Clinical urine analysis: without pathological changers or in cause of profuse bleeding may observed oliguria/anuria due to the acute decrease of total circulated blood volume and homodynamic disorders.

Coprological study: *macroscopically* insignificant bleeding from the upper part of the gastrointestinal tract is not determined; more significant bleeding changes the color of the stools (the stools are *black*, tarry (melena) due to formation of sulfur compound of iron). In bleeding from the lower part of the gastrointestinal tract, unaltered of red color blood is seen on the stool surface. Blood is easily found, when it mixed with mucus, like in dysentery and ulcerative colitis. In all doubtful cases the blood in the stool can be detected by chemical study. *Microscopically* may be detected: *columnar (intestinal) epithelium* cells presence in large quantity (indicates inflammation of mucus membrane of the large intestine), increase amount of leucocytes (in ulcerative processes in the large intestine – dysentery, tuberculosis, ulcerative colitis, cancer) and *macrophages* (inflammatory processes, especially in bacterial dysentery). In bleeding caused by amoebic dysentery, ankylostomidosis, non-specific ulcer colitis, and in some helminthiases large quantity of eosinophils is observed in the stools. *Erythrocytes* unaltered are seen in the faeces in bleeding from the large intestine (ulcerative affection, fissures of the anus, hemorrhoids). If

the blood originates from the upper part of gastrointestinal tract, erythrocytes are decomposed and can be only detected by chemical study.

Biochemical blood analysis: the blood urea nitrogen may be elevated out of proportion to the creatinine, particularly in upper gastrointestinal bleeding, due to breakdown of blood proteins to urea by intestinal bacteria as well as mild reduction in the glomerular filtration rate.

Hematocrit detection: in the setting of rapid hemorrhage, the initial hematocrit may not accurately reflect the magnitude of blood loss, since equilibration with extravascular fluid and hemodilution often require over 8 h.

Tests for occult bleeding detection. *Benzidine test* for latent blood is commonly used. Benzidine test is very sensitive, it is possible to determine insignificant amount of blood (0,2 %) in stool. It should be remembered however that positive reaction with benzidine could be observed in ingestion of foods containing blood (meat, fish). Therefore, such foods should be excluded from the diet 2–3 days before test.

Detection by card test for hemoglobin peroxidase, is an important means of finding colorectal neoplasia at earlier, potentially curable stages. Testing is advocated for patients over age 50 as a part of the yearly checkup. Multiple stools should be tested (usually two samples from three stools), and if any sample is positive, additional studies should be performed. A positive result can be due to physiologic blood loss, dietary peroxidases, undercooked meat, or any cause of upper or lower gastrointestinal bleeding. The daily ingestion of over 500 mg of vitamin C may result in a false-negative test. To limit the confounding variables, patients should be tested on a high-fiber and low-meat diet with no ingestion of nonsteroidal anti-inflammatory agents or vitamin C, although the daily low dose of aspirin (80 to 325 mg) taken to prevent cardiovascular disease generally does not lead to false-positive results.

Endoscopy: provides the ability to visualize the esophagus, stomach, duodenum, and colon directly, detect the cause of bleeding and receive biopsy specimens and cytological samples. So, obtained data depends on major disease. Endoscopy study includes; fibrogastroscopy, colonoscopy and rectoromanoscopy.

Fibrogastroscopy is the investigation of choice for the causes of upper gastrointestinal bleeding. Gastritis (acute, chronic, superficial, atrophic), the presence of erosions and ulcer is readily recognized and biopsy specimens can be obtained for histological diagnosis. This has become particularly important since all gastric ulcers, even if they appear benign, require biopsy and cytological brushing to exclude malignancy. Endoscopy is also essential for symptomatic patients who have had previous gastric surgery, as the postoperative stomach is notoriously difficult to examine radiologically and determination of Mallory-Weiss tear, varices or portal hypertensive gastropathy, arteriovenous malformations.

Colonoscopy and ***rectoromanoscopy*** are being increasingly used for the diagnosis of colonic disease in preference to a double-contrast barium enema.

Plain abdominal radiographs may provide help to obtain the required information the shape, size, position and mobility of the stomach; carcinomas, ulcers and ulcer scars that have a converging fold pattern are easily detected. Small lesions and slight irregularity of the mucosa can be identified.

Barium-meal examination of the small intestine is normally made following examination of esophagus, stomach and duodenum. It is indicated when disorders causing morphological changes in the small intestine, such as Crohn's disease, tuberculosis, neoplasms, radiation damage, ischemia or diverticulum, are suspected. The functional properties can be also assessed by x-ray study. Cancers of the small intestine have no specific radiological patterns.

The barium examination of the large intestine is important diagnostic procedure that gives information about its motor function, length, position, shape, and tone.

Instrumental non-obligatory methods (under indication):

- *angiography;*
- radionuclide studies;
- computed tomography.

Syndrome of portal hypertension

The syndrome of portal hypertension is the syndrome of pressure increase in portal vein system with impaired blood circulation that develops due to the primary liver diseases or hepatic veins pathology.

Classification

Depending on the etiology and mechanism of developing there are the next forms of the portal hypertension syndrome:

I. Suprahepatic block:

- hepatic veins thrombosis;
- hepatic veins compression;
- vena cava inferior compression and/or thrombosis.

II. Intrahepatic block:

- chronic hepatitis
- liver cirrhosis
- tumor of the liver
- metastatic liver damage.

III. Subhepatic block:

- congenital anomaly of vena porta;
- compression of a portal collector by a tumor;
- spasms.

Clinical features

In patients with the syndrome of portal hypertension the main complaints are connected with: dyspeptic and asthenic syndrome manifestation, presence of general intoxication, hepato-cellular insufficiency and pressure increase in portal vein system with impaired blood circulation.

The clinical signs of asthenic syndrome and general intoxication include – general weakness, irritability and loss of appetite, fatigue, perspiration, tachycardia, and headache.

The clinical signs of dyspeptic syndrome include – nausea and vomiting.

The clinical signs of hepato-cellular insufficiency and pressure increase in portal vein system – dull pain and feeling of pressure in the abdomen, enlargement of the abdomen in size.

The clinical signs of impaired blood circulation and heart failure are tachycardia, blood pressure fluctuation, hydrothorax, hydropericardium and peripheral edema.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. May observed deranged consciousness with hepatic coma develops at final stage of diseases. The posture may be active, active with restriction or forced in form of orthopnea, in cause of hepatic come observed passive posture.

The color of the skin and visible mucosa in initial stage has corporeal color, without eruptions. With disease progression pale color of the skin or even pathological jaundice occurs. Skin become dry, lost elasticity and turgor, develops eruptions, hemorrhage lesions and teleangioectasia – dark-red spots on the skin and mucosa, the visible dilation of small subcutaneous blood vessels, as a rule on the upper part of the trunk, disappeared after pressing due to the excessive production of estrogens in patients with portal liver cirrhosis, and edema of the lower extremities, buttocks, waist, down to anasarca.

In inspection, palpation, percussion and auscultation of *respiratory system* may be detecting the signs of congestion in lesser circle circulation and hydrothorax syndrome.

In inspection, palpation, percussion and auscultation of *cardiovascular system* may be detecting decrease loudness of heart sounds and triplet rhythm, tachycardia, decrease of blood pressure, the signs of hydropericardium and congestion in large circulation.

In inspection of the digestive system may be detect: enlarged abdomen, symmetrical, anterior abdominal wall take part in the breathing act, umbilicus and venous network are pronounced, scars, eruption, telangioectasia, scratches are present (tab. 3.1).

Table 3.1. ***Differential signs of meteorism and ascitis***

Sign	Meteorism	Ascitis
Skin	Doesn't change	"Rice-paper" (thin, shining)
Umbilicus	Retracted	Smoothed, or protruded
Fluctuation symptom	Absent	Present
Percussion	Tympanic sound	Dull sound in sloping sites

In superficial tentative oriental palpation and percussion of the abdomen may be distinguish moderate pain in right hypochondria; muscular resistance, diastesis recti are negative; fluctuation symptoms are positive.

Ascitic fluid seeks the lowest point in the abdomen, producing bulging flanks that are dull to percussion (fig. 3.1). The umbilicus may protrude. Turn the patient onto one side to detect the shift in position of the fluid level (shifting dullness) (fig. 3.2).

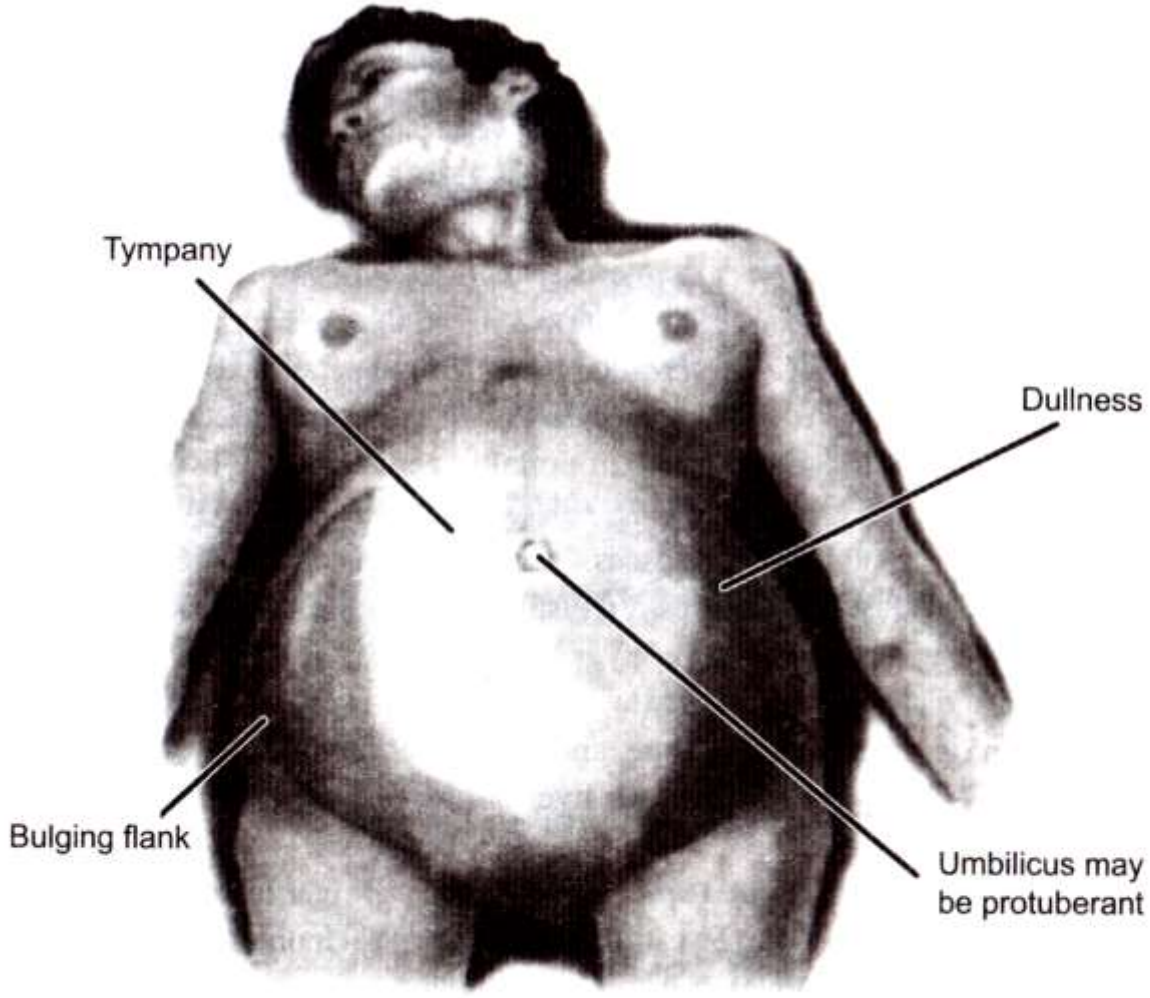


Fig. 3.1. Percussion sounds over abdomen.

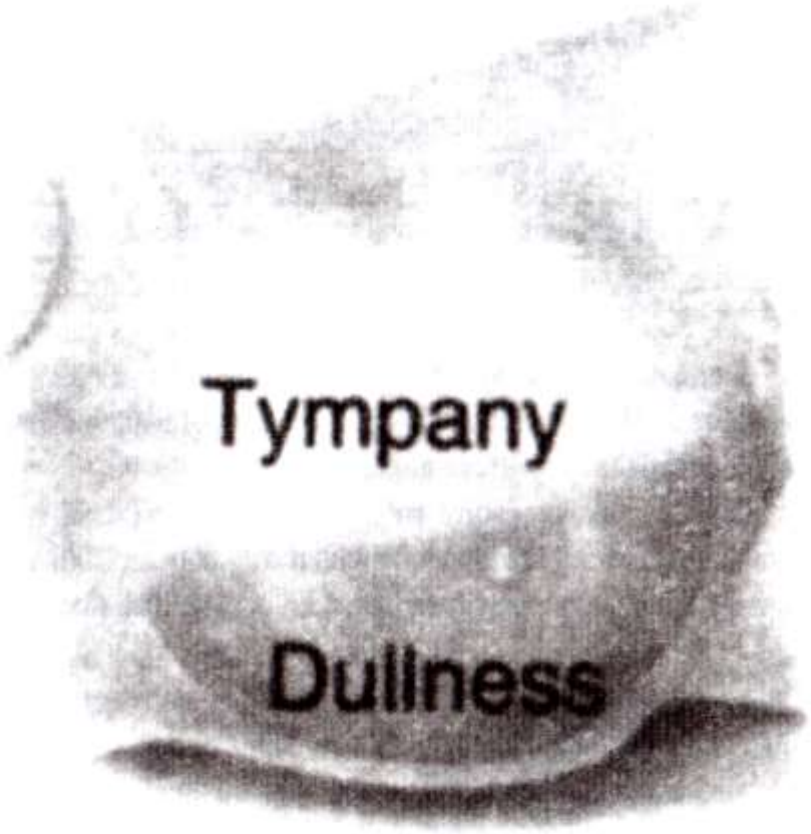


Fig. 3.2. Percussion sounds over abdomen

Penetrative palpation and deep sliding palpation of the abdomen are usual non-informative via significant fluid accumulation in the abdominal cavity.

In percussion of the liver according M.G. Kurlov due to the primary liver pathology may be detecting enlargement or decline of the liver sizes. Also via hepatic damage in patients with the syndrome of portal hypertension the liver borders can vary.

In considerable tumor, liver abscess, when the liver is enlarged the span of liver dullness is increased (that is upward displacement of the upper liver border and downward displacement of the lower liver border).

Downward displacement of the lower liver border is caused by hepatitis, congestive liver in right ventricular failure, and acute liver failure.

Upward displacement of the lower liver border observes in atrophic stage of the liver cirrhosis.

In palpation of the liver and spleen the changes of liver lower edge, surface, consistency, and tenderness can be estimated, observed splenomegalia. However, in patients with significant amount of the fluid in abdominal cavity the enlarged lower liver border and spleen are not accessible for palpation.

Additional methods of examination

Clinical blood analysis: leukopenia, thrombocytopenia, anemia.

Clinical urine analysis: without pathological changes.

Coprology study: may detect melena due to the portal hypertensive gastropathy or red color blood on the stool surface in cause of dilated hemorrhoid veins.

Biochemical blood analysis: the signs of: cytolysis syndrome – increased activity of alanine aminotransferases, aspartate aminotransferases, alkaline phosphatase, lactate dehydrogenase, cholinesterase, sorbitol dehydrogenase and hyperbilirubunemia with predominance of bound fraction indicates mainly necrotic damage of the liver (hepatocytolisis, liver necrosis); hepatic-cellular failure syndrome – decreased albumins, cholesterol, protrombin contents in combination with hyperbilirubinemia.

Tests for occult bleeding detection – may be positive.

Endoscopy: provides the ability to visualize the esophagus, stomach, duodenum, and colon directly, detect the cause of bleeding and receive biopsy specimens and cytological samples. Obtained data depends on major disease and in patients with *the syndrome of portal hypertension* can include varicose dilation of esophagus and hemorrhoid veins, atrophy and thickness of mucosa, propensity to bleedings.

Plain abdominal radiographs may provide to obtain the required information about the shape, size, position and mobility of the stomach; carcinomas, ulcers and ulcer scars that have a converging fold pattern are easily detected. Small lesions and slight irregularity of the mucosa can be identified.

Ultrasound examination (ultrasonography): is useful for evaluation of the liver and spleen sizes, study of their parenchyma state and measurements of portal vein diameter.

Ultrasound angiography of hepatic vessels: reveals impaired blood circulation in veins portal system.

Instrumental non-obligatory methods (under indication):

- selective angiography;
- radionuclide studies;
- computed tomography.

Syndrome of functional dyspepsia

Syndrome of functional dyspepsia – the complex of the symptoms that includes the pain and feeling of the discomfort in epigastria, heaviness and feeling of overflow after meal, early saturation, swelling of a stomach, nausea, vomiting, eructation, heartburn and other signs at which it is not possible to reveal organic pathology (without biochemical or morphological cause).

Prevalence in the developed countries of the world makes 30–40 %. The basic mechanism of functional dyspepsia formation belongs to motor stomach disturbances when physiological antro-duodenal coordination impaired (desynchronization of gastric fundus peristaltic and gastroesophageal sphincter activity with pyloric sphincter opening and duodenal motor function).

Etiology

- alimentary faults;
- bad habits;
- reception of medicines;
- psychological stresses;
- infectious of a stomach mucous by Helicobacter pilory.

Pathogenesis

In a basis of the functional dyspepsia syndrome lays various types of disorders of gastro-duodenal mobility:

- reduction of a gastric fundus motoricy (gastroparesis);
- gastric dysrrhytmias – frustration of a gastric peristaltic rhythm (tachygastria, antral fibrillation, bradygastria);
- disorders of antrocardal and antroduodenal coordination;
- duodeno-gastral reflux;
- impaired mobility of a stomach (ability of a proximal part to relaxation);
- increased sensitivity of a stomach receptor apparatus to a stretching (visceral hypersensitivity);
- infection by Helicobacter pilory at persons without visible morphological attributes of a gastritis.

Clinical features

In patients with functional dyspepsia the clinical picture includes the general neurologic displays – sleeplessness, migraines, irritability, bad mood and special (gastric) that depend on a type of dyspepsia.

Ulcer-like type – is characterized by periodic pain in epigastria, the moderate intensity, as a rule without irradiation, arising on an empty stomach (hungry pains) or at night (night pains), relieved after reception of food and/or antacids.

Dysmotonic type – is characterized by the feeling of early saturation, weight, overflow, a swelling in the epigastria; sensation of discomfort after meal; nausea, sometimes vomiting; decrease in appetite.

At a nonspecific type there can be various attributes, which difficultly carry to any of described variants.

For functional dyspepsia there are specific three attributes (according to Roman (II) diagnostic criteria):

- constant or recurrent dyspepsia (a pain or the discomfort localized in epigastria), which duration not less than 12 weeks for last 12 months (between aggravations there can be light intervals);
- on the basis of the anamnesis, endoscopic researches of the upper part of a gastrointestinal tract and ultrasound examination of abdominal cavity organs there are absent proofs of organic disease;
- absence of proofs, that dyspepsia is facilitated by defecation or connected with change of frequency of a stool.

Establishment of the diagnosis probably only by exception of disease with a similar clinical picture, especially that connected with the «symptoms of alarm» (a fever, an impurity of blood in stool, an anemia, accelerated ESR, unmotivated behaviors).

Classification

I. According to the type of dyspepsia there are distinguish:

- the ulcer-like type;
- the dysmotor type;
- the nonspecific type.

II. According to the stage of dyspepsia there are distinguish:

- stage of an aggravation;
- stage of unstable remission;
- stage of remission.

Objective examination. *General patient's condition* is satisfactory. The consciousness is clear and the posture is active.

The color of the skin and visible mucosa has corporeal color, without eruptions.

In inspection, palpation, percussion and auscultation of respiratory, cardiovascular and digestive systems there are no essential diagnostic value.

Additional methods of examination

Clinical blood analysis: without pathological changes.

Clinical urine analysis: without pathological changes.

Coprology study: without pathological changes.

Biochemical blood analysis and study of liver enzyme: without pathological changes.

Glucose test: normal.

Tests for occult bleeding detection – negative.

Fibroesohagogastroduodenoscopia is required for exception of reflux-esophagitis, stomach ulcer and tumor of a stomach.

Ultrasound examination (ultrasonography): – for exception of pancreatitis, cholecystitis and stones in gall bladder.

Indication of Helicobacter pilory is required for specification of diseases etiology.

Chrome endoscopia of a stomach – for exception of the esophagus and stomach mucous dysplasia.

Plain abdominal radiographs are required for exclusion of organic affection and revealing of gastrparesis.

Intragastrul PH-metria of esophagus and stomach.

Biopsy is required for specification of the diagnosis.

Chronic gastritis and duodenitis

Chronic gastritis and duodenitis – chronic inflammatory-dystrophic process in the stomach and duodenum mucous with recurrent duration that passing with cells regeneration disturbances, progressive atrophies of secretory epithelium, impairment secretory, motoric and incretory functions of the stomach.

Chronic gastritis is morphologic concept with stereotypic reactions in the stomach mucosa: inflammation, atrophy, impaired cells regulation with metaplasia and dysplasia. At general, chronic gastritis is not represents nosologic connotation: it is heterogeneous group of diseases that have different etiology and are incorporated by similar pathologic mechanisms.

Etiology:

- the leading role in development of the chronic gastritis belongs to Helicobacter pylori;
- genetic predisposition;
- influence of other infectious factors (Gastrospirillium homiunus, Treponema pallidum, Mycobacterium tuberculosis, parasites invasions, virus infectious, fungus damage);
- autoimmune factors;
- particularities of nutrition;
- food allergy;
- influence of harmful factors of an environment;
- radiating irradiation;
- influence of medicamentous therapy;
- somatic, nervous end endocrine diseases.

Pathogenesis

Infection of the human organism by Helicobacter pylori and influence of other etiologic factors leads to the stomach's inflammatory mucous membrane infiltration via

activation of polymorphnuclear leukocytes. Moreover, adhesion of bacterium on gastric epithelium causes cytosceleton reorganization and proinflammatory cytokines production. They stimulate disorders of cellular proliferation, regenerations and apoptosis.

Classification. ***The international classification of chronic gastritises, 1996***

The type of gastritis	Synonyms	Etiologic factors
Non-atrophy	Superficial Diffuse Antral gastritis Chronic antral gastritis Interstitial Hypersecretory Type B	Helicobacter pylori Other factors
Atrophy Autoimmune Multifocal	Type A Diffuse Associated with pernicious anemia	Autoimmune Helicobacter pylori Particularities of nutrition Influence of harmful factors of an environment
The special forms		
Chemical	Reactive reflux-gastritis Type C	
Radiating		Radiating irradiation
Lymphocytary	Variolomorphy Associated with celiakia	Idiopathic mechanisms Autoimmune mechanisms Helicobacter pylori
Non-infective granulomatous	Isolated granulomatous	Crone's disease Sarcoidosis Forging corpuses
Eoshynophily	Food allergy Other allergens	Allergic
Other infections		Bacteria Viruses Fungus Parasites

Clinical features

In patients with chronic gastrodoudenitis the main complaints are pain in epigastrium, connected with food reception, heaviness in abdomen after the meal, earlier saturation, deterioration of appetite, nausea, eructation and vomiting.

Objective examination. *General patient's condition* is usually from satisfactory to moderate grave. The consciousness is clear, the posture active.

The color of the skin and visible mucosa has corporeal color, without eruptions. The cutis is elastic with reserved turgor, free from eruptions, hemorrhage lesions and teleangioectasias. With disease progression and prolonged duration may occurs pale color of the skin and trophic infringements, loss of weight and muscular dystrophy.

The data of inspection, palpation, percussion and auscultation of respiratory and cardiovascular systems are without peculiarities.

In superficial tentative oriental palpation and percussion of the abdomen may be distinguish moderate pain in epigastrium and umbilical regions; muscular resistance, diastases recti and fluctuation syndrome are negative;

In penetrative palpation in cause of duodenum damage assessed moderate pain in duodenum bulb projection point that places 2 cm from the umbilicus on the bisector of right angle formed by anterior median line and line drawing through the umbilicus.

In deep sliding palpation of the abdomen results are usual comparable with healthy subjects.

Palpation of the liver and spleen – as usual the liver is non-palpable or detect about 1–2 cm below the right costal margin in the midclavicular line, the edge is soft, sharp, tenderness and surface is smooth. The spleen is not palpable.

Additional methods of examination

Clinical blood analysis – norm al or moderately expressed anemia.

Clinical urine analysis is without pathology.

Coprology study – attributes of dysbacteriosis.

Research of blood electrolytes (sodium, calcium, magnesium, copper, chlorine) – is possible revealing of electrolytes dysbalance.

Biochemical blood analysis *(hepatic complex)* is without pathology.

Biochemical blood analysis *(renal complex)* is without pathology.

Determination of total protein and its fraction in the blood serum is within the limits of norm.

Fibroesophagogastroduodenoscopia with biopsy and the subsequent morphological research of the biopates is required.

Investigation of Helicobacter pylori is required for purpose of adequate etiotropic therapy.

Stomach chromendoscopia is required for early revealing mucous membrane dysplasia.

Intragastral pH-metria is required for stomach acid secretor function definition.

Ultrasound examination *(ultrasonography)* – revealing of accompanying pathology signs and character.

Instrumental non-obligatory methods (under indications):

– serological tests – research of serum pepsinogen-1 and gastrin-17, antibodies to parietal cells;

– ultrasound examination of a thyroid gland and pelvis organs;

– rentgenoscopy of the thorax and abdominal cavity;

– ECG;

– colonoscopia.

Chronic hepatitis

Chronic hepatitis is chronic polyetiological inflammatory-destructive process in the liver duration over 6 months.

Etiology

– virus (viruses of hepatitis B, C, D, F, a cytomegalovirus, a virus of a simple herpes, etc.);

– influence of alcohol;

– jatrogenic (medicines induce);

– influence of toxic and chemical substances;

– hereditary-dependents pathology and metabolic disorders (Wilson-Konovalov disease, hemochromatosis, α_1 antytripsini deficiency).

Classification

I. According to the etiology and pathogenesis:

– chronic hepatitis B;

– chronic hepatitis C;

– chronic hepatitis D;

– uncertain chronic virus hepatitis (F, G?);

– autoimmune hepatitis (type 1, type 2, type 3);

– toxic hepatitis;

– alcoholic hepatitis;

– criptogenic hepatitis.

II. According to the clinico-biochemical and histological criteria:

1. According to the degree of activity (defined by heaviness of inflammatory-necrotic process):

a) minimal;

b) moderated;

c) expressed.

2. According to the index of histologic activity (IHA) on Knodell in points:

a) periportal hepatocytis necrosis, including the bridge-like – 0–10 points;

b) intrasegmental focal necrosis and hepatocytis dystrophy – 0–4 points;

c) inflammatory infiltrate in portal tracts – 0–4 points;

d) fibrosis – 0–4 points.

IHA from 1 up to 6 points testifies to presence of the "minimal" chronic hepatitis, 7–12 points – the "moderate", 13–18 points – a "grave" chronic hepatitis.

III. According to the stage of a chronic hepatitis (defined by prevalence of fibrosis and development of liver cirrhosis):

0 – fibrosis is absent;

1 – poorly expressed periportal fibrosis;

2 – moderated fibrosis with porto-portal septs;

3 – expressed fibrosis with porto-central septs;

4 – liver cirrhosis.

In direction «Chronic hepatitis» are include also such diseases as Wilson-Konovalov disease, primary biliary cirrhosis, sclerosive cholongitis, in a kind of that they long time proceed with a picture of a chronic hepatitis.

Chronic virus hepatitis

Chronic virus hepatitis (CVH) – the chronic disease of the liver caused by viruses of hepatitis B, C, and D which develops in 6 months after the transferred acute virus hepatitis.

About 75–85 % of all chronic hepatitis – have the virus nature. Now in the world by virus of hepatitis B are infected nearly 2 milliards of people, by virus of a hepatitis C – 350 millions. Furthermore, data of official statistics are incomplete, as up to 80 % of cases of acute hepatitis proceeds without jaundice, with the minimal clinical symptoms, and as a rule, do not get in sight doctors before development of complications. The chronic virus hepatitis is diagnosed later 6 months after the transferred acute virus hepatitis if are kept a jaundice, and/or hepatomegalia, splenomegalia, increase of transaminases activity and persistent of virus hepatitis markers.

Classification of the chronic virus hepatitis

Classification	CVH B	CVH C
The stage of the process	Integration Replication (progression)	Latent Reactivation (progression)
The process activity (according to the index of histologic activity – IHA on Knodell and alanine aminotransferases level)	Minimal Moderate Grave	Alanine aminotransferases level increase up to 3 point Alanine aminotransferases level increase up to 7–10 point Alanine aminotransferases level increase more than 7–10 point

Clinical features

Clinical features are defined by a degree of chronic hepatitis activity and its stage.

More often, the patients do not show specific complaints from digestive system and suffering from weakness, fast fatigue and decrease in working capacity.

In some cases the clinical picture is characterized by presence of jaundice, heavy feeling in right and/or left hypochondrias due to increase of the liver and the spleen sizes, occurrence of dyspeptic syndrome, general intoxication and extra hepatic displays.

Extra hepatic displays include: the complaints caused by affection of joints and skeletal muscles (arthralgias and myalgias), development of myocarditis, pericarditis, pancreatitis, Shegren syndrome, vasculitis, Reino syndrome and affection of kidneys (they are shown at 40–70 % of patients with CVH).

In the anamnesis, considering the basic ways of infection with a virus, the important diagnostic value has the anamnesis of hemotransfusions, use of drugs, operative interventions and transferred sharp hepatitis. Infection also is possible at carrying out of stomatologic and intravenous manipulations, performance of tattourage, manicure and sexual contacts.

Objective examination. *General patient's condition* is from satisfactory to extremely grave. May observed deranged consciousness with hepatic coma develops at final stage of diseases. The posture may be active, active with restriction, forced or passive in case of hepatic come.

The color of the skin and visible mucosa in stage of integration or latent current has corporeal color, without eruptions; with disease progression pale color of the skin or even jaundice occurs. Cutis become dry, lost elasticity, occurs propensity to hematomas and teleangioectasia formation, bleeding from the gums. In patient may distinguish loss of weight, muscular hypotrophy and signs of joints affection.

In inspection, palpation, percussion and auscultation of *respiratory system* the signs are as usual without particularities.

In inspection, palpation, percussion and auscultation of *cardiovascular system* may be detecting the signs of myocarditis and pericarditis.

In inspection of the digestive system may be detect: enlarged abdomen in size, symmetrical, anterior abdominal wall take part in the breathing act, umbilicus and venous network are pronounced, scars, eruption, teleangioectasia, scratches are present.

In superficial tentative oriental palpation and percussion of the abdomen may be distinguish moderate pain in right and left hypochondrias; muscular resistance, diastesis recti and fluctuation symptoms are negative.

Penetrative palpation and deep sliding palpation of the abdomen are usual without specific particularities.

In percussion of the liver according M.G. Kurlov due to the primary liver pathology may be detecting enlargement or decline of the liver sizes.

In palpation of the liver and spleen the changes of liver lower edge, surface, consistency, and tenderness can be estimated.

Additional methods of examination

Clinical blood analysis: without pathological changes; in severe cases observed – leucopoenia, thrombocytopenia, anemia.

Clinical urine analysis: without pathological changes.

Coprology study: without pathological changes.

Biochemical blood analysis and examination of liver enzymes: increased activity of alkaline phosphotase in combination with hyperbilirubunemia for account of both fraction, increased activity of alanine aminotransferases, aspartate aminotransferases.

Glucose test: normal.

Determination of total protein and its fraction in the blood serum: hypoproteinemia, dysproteinemia with α-2 and γ-globulins fractions elevation.

Tests for occult bleeding detection – negative.

Coagulogramma – prothrombin index normal or decreased.

Detection of ferrum and transferring concentration in the plasma and urine – within of normal limits.

Detection of copper concentration in the plasma and urine – within of normal limits.

Detection of cerruloplasmin concentration in the plasma – within of normal limits.

The study of viral hepatitis markers in the blood – (serological markers, detection of virus genome fragments) – for CVH B diagnosis – HBs Ag, Hbe Ag, anti-Hbe, HB anti cor, IgM and IgG, PCR-DNA; for CVH C diagnosis – antiHCV, IgM and IgG, NS_3, NS_4, PCR-RNA; for CVH D diagnosis – antiHDV, PCR-DNA.

Detection of antibodies to AIDS – test is negative.

Detection of α_1-antitripsini in serum – within of normal limits.

Detection of α-phetoprotein, antinuclear, antismooth muscles, antimitochondria antibodies are in diagnostic titers within of normal limits.

Instrumental obligatory methods:

- ultrasound examination (ultrasonography) – enlargement of liver size with increase of its acoustic density;
- biopsy of the liver with cytoserologial and hystomorphological examination of the bioptates.

Instrumental non-obligatory methods (under indications):

– fibrogastroduodenoscopia: for exception of portal hypertension signs;
– computed tomography and magnetic resonance imaging: for cancer exclusion.

Peptic ulcers (ulcer disease)

Ulcer disease or peptic ulcer – pathologic process based on stomach or duodenum mucous membrane inflammation with its local erosive damage due to infectious or non-infectious mechanism as response to impaired endogenous balance between local factors of "aggression" and "protection".

Etiology

– associated with Helicobacter pylori;
– influence of drugs;
– results of pathological hypersecretion;
– mixed etiology.

Classification

According to the localization there are distinguish:

– the ulcer of the stomach;
– the ulcer of duodenum;
– the ulcer of the stomach and duodenum (combinated);
– the gastro-jejunum ulcer.

According to the etiology there are distinguish:

– Helicobacter pylori-positive ulcer;
– Helicobacter pylori-negativee ulcer;
– mixed.

According to the stage of the process there are distinguish:

– active;
– in phase of scarring;
– the phase of scar;
– the phase of extensive unscarring.

According to the accompanied morphological changes there are specified:

– localization and activity of gastritis or duodenitis;
– the presence and stage of mucous atrophy;
– the presence and stage of intestine metaplasia;
– the presence of erosions and polyps;
– the presence of gastro-esophagus or duodeno-gastrul reflux;
– the characteristic of secretor and motor functions.

According to the complications development are distinguish:

- non-complicated;
- complicated:

a) gastrointestinal bleeding
b) perforation;
c) penetration;
d) stenosis;
e) malignization.

Clinical features

In patients with chronic peptic ulcer the main complaints are abdominal pain and displays of dyspeptic syndrome.

In order to describe the pain it is necessary the detail discription:

- location of the pain (epigastric region, right or left hypochondrium, umbilical region);
- character of the pain (periodical or paroxysmal (at certain time of the day); permanent or seasonal (in spring or autumn); intensity (dull, stabbing);
- connection with meals (fasting pain, after meal);
- early – occurring 30–40 min after meals;
- late – 90–120 min after meals;
- nocturnal, hunger pain, which is abated after taking food);
- radiation of the pain;
- relieving factors (vomiting, taking food or soda, spasmolytics, warmly);
- possible connection between pain and physical strain (weight lifting, traffic jolting).

In peptic ulcer the pain is localized in epigastric region, may radiate to the back and is of variable quality: gnawing, burning, boring, aching, pressing, or hunger like. The pain is intermittent. Duodenal ulcer is more likely than gastric ulcer can cause pain that wakes the patient at night, and occurs intermittently over a few weeks, then disappears for month, and then recurs. Food and antacids may bring relief, but not necessarily in any of these disorders and least commonly in gastric ulcer.

Associated symptoms are: nausea, vomiting, belching, bloating; heartburn (more common in duodenal ulcer); weight loss (more common in gastric ulcer). Gastric ulcer is more common in the older (over 50 yr), and duodenal ulcer in those from 30–60 year.

Objective examination. *General patient's condition* is usually from moderate grave to extremely grave. The consciousness is clear, the posture usually active or may be forced in cause of complications development.

The color of the skin and visible mucosa has corporeal color, devoid of eruptions. The cutis is elastic with reserved turgor, free from eruptions, hemorrhage le-

sions and teleangioectasias. With disease progression and prolonged duration may occurs pale color and loss of weight. Assessed strike on tongue.

The data of inspection, palpation, percussion and auscultation of respiratory and cardiovascular systems are without peculiarities.

In superficial tentative oriental palpation and percussion of the abdomen may be distinguish pain in epigastrium and umbilical regions with local muscular resistance; diastesis recti and fluctuation syndrome are negative;

In penetrative palpation in cause of duodenum ulcer assessed pain in duodenum bulb projection point that places 2 cm from the umbilicus on the bisector of right angle formed by anterior median line and line drawing through the umbilicus.

In deep sliding palpation of the abdomen results are usual comparable with healthy subjects.

Palpation of the liver and spleen – as usual the liver is non-palpable or detect about 1–2 cm below the right costal margin in the midclavicular line, the edge is soft, sharp, tenderness and surface is smooth. The spleen is not palpable.

Complications:

- gastrointestinal bleeding (anemia, melena, vomiting by "coffee grounds");
- punching (sudden intensive knee-like pain in epigastrium area, collapse, muscular resistens of the forward abdominal wall, delay of gases, pallor of the skin and mucosa with cyanotic tint, bradycardia, superficial breath, absence of the anterior abdominal wall in breath, positive Shetkin-Blumberg symptom, reduction or is fuller disappearance of hepatic dullness at percussion with progressing development of peritonitis clinic);
- penetration (intensive, constant pain in the abdomen, moderated leikocytosis, accelerated ESR, subfebril temperature);
- piloroduodenal stenosis (sensation of the stomach overflow, heartburn, eructation sour, vomiting by food);
- malignisation (transformation to the cancer of the stomach with corresponding clinical features developing).

Additional methods of examination

Clinical blood analysis – the signs of ferric deficiency anemia at chronic or sharp bleeding, leucocytosis at the complicated current, possible moderated reticulocytosis).

Clinical urine analysis is without pathology.

Biochemical blood analysis (pancreatic complex) increase amylase level at ulcers penetration in pancreas head.

Biochemical blood analysis (hepatic complex) is usual without pathology.

Tests for occult bleeding detection – may be positive in latent or acute gastrointestinal bleeding.

Determination of total protein and its fraction in the blood serum are within the limits of norm.

Determination of the iron in blood – decrease maintenance.

Coagulogramme – decrease in the maintenance of blood coagulating system factors.

Definition of serum gastrin level – expressed hypergastrinemia at Zolonger-Elissone syndrome.

Fibroesophagogastroduodenoscopia with biopsy and the subsequent morphological research of a bioptates – confirms presence of ulcer defect and specifies of its localization, depth, the form, the sizes, condition of the bottom and edges of the ulcer and reveal accompanying changes of the mucous membrane.

Morphological research of the bioptates – defines character.

Investigation Helicobacter pylori is required for purpose of adequate etiotropic therapy.

Stomach chromendoscopia is required for early revealing mucous membrane dysplasia.

Intragastral pH-metria is required for stomach acid secretor function definition.

Ultrasound examination *(ultrasonography)* – revealing of accompanying pathology signs and character.

Instrumental non-obligatory methods (under indications):

- rentgenoscopy of the stomach and duodenum;
- longitudinally pH monitorin;
- serological tests – research of serum pepsinogen-1 and gastrin-17, antibodies to parietal cells;
- ultrasound examination of a thyroid gland and pelvis organs;
- rentgenoscopy of the thorax;.
- ECG;
- colonoscopia.

Calculus cholecistitis (cholelitiasis)

Calculus cholecistitis (the stone of the gall bladder) is chronic disease that caused by impaired cholesterol exchange and/or bilirubin metabolism with stones formation in the gall bladder (chlelitiasis) and/or in bilious tract (choledocholitiasis).

Etiology:

- metabolic dysbalance (impaired cholesteric exchange, adiposity and increased estrogen development);
- hypodinamia;
- an irrational nutrition (high-caloric food, low contents of vegetative fibers in the meal);

- eldery;
- treatment by hyperlipidemic fibrates;
- diseases of the gastrointestinal tract, accompanied with acquire incompetence, biliar tract infections and bile;
- hemolytic anemias.

Pathogenesis:

- stages of bilious stones formation;
- stage of saturation;
- stage of crystallization;
- stage of growth.

Cholesteric concrements in the gall bladder are formed at presence in it the bile overload by cholesterol. Thus in the liver the superfluous quantity of cholesterol and insufficient quantity of bilious acids is synthesized, including lecithin that is also is in the dissolved condition. As a result cholesterol drops out in a deposit. For the further formation of stones the condition contractivity functions of the gall bladder and presence of inflammatory mucous damage is of important sense. Under influence of nucleation factors (bile glicoproteins) from the dropped out crystals of cholesterol the first microlits appear. In condition of decreased evacuator functions they are start to grow.

Classification

The I stage – the stage of the beginning or prior to the stone formation;
The II stage – the stage of stones formation with indication:

- according to localization;
- according to the stones amount;
- according to the stones composition;
- according to the clinic duration.

The III stage – the stage of complications.

Clinical features

Probably may be long asymptomatic current, clinically manifested, not complicated and complicated current.

The most typical complaints are attacks biliary colic. Sudden obstruction of the cystic duct or common bile duct by a gallstone in biliary colic causes epigastric or right upper quadrant steady, aching pain (not colicky) that may radiate to the right scapula and shoulder. This pain is characterized by rapid onset over a few minutes, lasts one to several hours and subsides gradually. Anorexia, nausea, vomiting, restlessness often accompany the pain in biliary colic.

In *acute cholecyctitis* due to inflammation of the gall bladder, usually from obstruction of the cystic duct by a gallstone steady, aching pain arises in right upper quadrant or upper abdominal, which may radiate to the right scapular area. Gradual onset and course longer than in biliary colic is typical. Jarring and deep breathing aggravate pain, and it is usually accompanied by anorexia, nausea, vomiting, and fever.

Also the patients show complaints to the bitterness in a mouth, sub fibril temperature.

Objective examination. *General patient's condition* is from satisfactory to moderate grave. The consciousness is clear and the posture is active or forced. At survey – the raised weight of a body as a rule is defined.

The color of the skin and visible mucosa has corporeal color (*cutis coloris somatici*), without eruption, moderate moisture and elasticity, preserved turgor, may observe transient subicteria of the skin or even yellow color due to obstruction – development of mechanical type of jaundice.

The results of inspection, palpation, percussion and auscultation of respiratory and cardiovascular systems are without particularities.

In superficial tentative oriental palpation of the abdomen detect moderate pain in right hypochondria. Muscular resistance, diastases recti, and fluctuation symptoms are negative.

In penetrative palpation of the abdomen identify tenderness in gall bladder point (Ker point) and positive Kerras', Murphys', Ortners' and Mussis' symptoms.

In deep sliding palpation of the abdomen in absence of accompanied pathology received date usually non-informative and comparable with healthy subjects.

In palpation of the liver and spleen the changes of liver lower edge, surface, consistency, and tenderness usually not estimated. At cholangitis and reactive hepatitis development – observed moderated hepatomegalia.

Additional methods of examination

Clinical blood analysis – leucocytosis with shift of the formula to the left, accelerated ESR.

Clinical urine analysis – presence of bilious pigments.

Coprological study – increase maintenance of fat acids. After biliary colic, sometimes in some days after it, and in rare cases without preliminary colic can be detect concrements, which are of bilious, pancreatic or intestinal origin. Absence of stercobilin in the stools (acholic stool) in obstructive jaundice is observed in complete obstruction of common bile duct (obturation by stone, tumor).

Biochemical blood analysis – increase of the common bilirubin due to direct fraction, increase of alanine aminotransferases and aspartate aminotransferases activity

(at development of hepatitis and in the period biliary colic), increase activity of alkaline phosphotase and moderate increase of amylase, cholesterol and ß-lipoproteins levels.

The analysis of blood glucose – norm.

Tests for occult bleeding detection – negative.

Determination of total protein and its fraction in the blood serum – the general protein level usually without pathological changers, absence of dysproteinemia or insignificant hypergammaglobulinemia is typical.

Definition of pancreatic elastase-1 – for diagnostics of the complications connected with development of a chronic pancreatitis and cholangitis.

Ultrasound examination of the digestive organs – revealing the signs of cholecystitis and stones in the gall bladder.

Cholecyctography. About 80 to 85 per cent of gallstones are not radio-opaque and oral cholecystography remains the method of choice for examining the gall bladder with contrast medium to detect calculi when ultrasound is not available or is inconclusive. Abnormalities causing a change in the outline of the gall bladder such as adenomyomatosis are well demonstrated at oral cholecystography.

Endoscopic study is required for verification of the diagnosis and carrying out of differential diagnostics.

Endoscopic retrograde cholangiopancreatography is frequently used to demonstrate the ducts in patients with obstructive jaundice. Endoscopic sphincterotomy can be done during the procedure, thus allowing bile-duct calculi to pass freely into the duodenum, and frequently relieving bile-duct obstruction due to calculi.

Survey roentgenography examination of the belly cavity – revealing stones in the gall bladder and attributes of complications.

Survey radiological research of the thorax is required for carrying out of differential diagnostics with respiratory and cardiovascular pathology.

ECG is required for carrying out of differential diagnostics with stenocardia and sharp heart attack (myocardial infarction).

Computed tomography – for the diagnosis verification and carrying out of differential diagnostics.

Study of duodenal secretion – may be observed reduction of gall bladder reflex (amount of bladder bile increase to 100–150ml in norm 30–70ml; the bile excreted by little portions; dilation of bile discharge more than 45min).

Liver cirrhosis

Liver cirrhosis – the chronic progressing disease of a liver described by diffuse affection of liver's parenchyma and stroma with quantity reduction of functioning cells, their nodular regeneration and excessive development of connective tissue that leads to cytoarchitectonic reorganization of the liver and its vascular system and

development of hepatic insufficiency with involving in pathological process of other organs and systems. Prevalence makes about 90 cases on 100 thousand populations.

Etiology

Liver cirrhosis is an outcome of:

I. Chronic hepatitis various etiology

- chronic hepatitis B;
- chronic hepatitis C;
- chronic hepatitis D;
- uncertain chronic virus hepatitis (F, G?);
- autoimmune hepatitis (type 1, type 2, type 3);
- toxic hepatitis;
- alcoholic hepatitis;
- criptogenic hepatitis.

II. Bile tract diseases.

III. Genetically caused disorders of a metabolism:

- deficiency of alpha-1-antitripsini;
- shortage of galactoso-1-phosphatituridiltransferase;
- deficiency of amylo-1,6-glicozidase;
- Konovalov-Wilson's disease.

IV. Not specified etiology.

Pathogenesis

In cirrhosis changes of the liver cytoarchitectonic develop as a result of direct damaging influence of the etiological agent. Thus arises parenchyma's necrosis and development of fibrous septs, that alongside with hepatocytis reserved regeneration and leads to "false" segments formation. Cirrhotic reorganization of the liver causes disorders of a blood circulation in the organ. Lack of blood supply to the liver's parenchyma leads to destruction which is accompanied by functional insufficiency of the organ and supports the progressing of cirrhotic process.

Thus it is switched off metabolic functions of the organ owing to their true insufficiency, shunting of blood through anastamosis and change of cellular permeability.

The portal hypertensia at cirrhosis of the liver is caused due to compression of hepatic veins by a fibrous tissue, units of regeneration, sinusoid fibrosis, the increased inflow of blood to portal veins system on arterial-venous anastamosis from a hepatic artery. Portal pressure increase is accompanied by strengthening of collateral blood circulation that at the beginning warns its further increase. At time there are formed anastamosis between portal vein and bottom hollow veins in a forward belly wall, in sub mucous layer of the bottom third of gullet and cardiac department

of a stomach, between spleen and left hepatic veins, in pools of mesenterial and hemorrhoid veins.

Increase of hydrostatic pressure, hypoalbuminemia and reduction of effective volume of plasma with the subsequent renin-angiotensin-aldosteron system activation and increased of antydiuretic hormone secretion are the major factors of ascitis development on liver cirrhosis.

Clinical features

They are defined by a stage of process and presence of complications – from full absence of symptoms up to common clinical picture of hepatic coma.

The sharp painful syndrome is not specific. More often, the patients have complaints on the feeling of weight and dull pains in right hypochondria and epigastria, less often – in left hypochondria, that amplifying after taking food and physical activity.

The patients also suffering from the dyspeptic symptoms that connected with disorders of digestion, general intoxication and accompanying pathologies of a gastro-intestinal tract: the swelling of a stomach, less often – a nausea, vomiting, a heartburn, bitterness in a mouth, infringements of a stool (in the beginning of disease constipation, in process of progressing process – diarrheas).

Also can be present the general complaints – weakness, fatigue, decrease in working capacity, weight reduction, rise in temperature (asteno-vegetative syndrome), yellowness of the skin and visible mucosa, skin itch, hemorrhages, nasal and uteri bleedings (coagulopathy syndrome).

The liver cirrhosis allocates the following clinical syndromes:

– the syndrome of portal hypertension (includes edematous-ascitic syndrome);
– the syndrome of hepato-cellular insufficiency;
– hepatic encephalopathy;
– hepatolienal syndrome.

Objective examination. *General patient's condition* is from satisfactory to extremely grave. May observed deranged consciousness with hepatic coma develops at final stage of diseases. The posture may be active, forced or passive in cause of hepatic come.

In general inspection may detect jaundice, expansion of the veins on the forward abdomen wall, palmary erythema, Dupuitrens' contracture, hynecomastia at men, traces of scratches on all body. There can be an expressed loss of weight of a body down to cachexia, enlargement of abdomen in sizes, edematous ascitic syndrome even anasarca.

The results of inspection, palpation, percussion and auscultation of respiratory and cardiovascular systems depend on complications development.

In superficial tentative oriental palpation of the abdomen may be detect moderate pain in right and left hypochondrias, muscular resistance and positive fluctua-

tion symptoms, the signs of diastesis recti are negative.

Penetrative palpation and deep sliding palpation of the abdomen may be non-informative trough significant fluid accumulation in the abdominal cavity (in case of ascitis formation).

In percussion of the liver according M.G. Kurlov and palpation of the liver and spleen may be detecting enlargement of the liver and spleen sizes with increase of their density and rough surface. However, in patients with significant amount of the fluid in abdominal cavity the enlarged lower liver border and spleen are not accessible for palpation.

Complications: encephalopathy, hepatic insufficiency, portal hypertension, hepatorenal syndrome, bacterial peritonitis, bleeding from varicous expanded veins.

Additional methods of examination

Clinical blood analysis: leukopenia, thrombocytopenia, anemia, increased ESR.

Clinical urine analysis: proteinuria, bacteriouria.

Coprology study: may detect melena due to the portal hypertensive gastropathy or red color blood on the stool surface in cause of dilated hemorrhoid veins.

Tests for occult bleeding detection – may be positive.

The study of viral hepatitis markers in the blood – (serological markers, detection of virus genome fragments) – for CVH B diagnosis – HBs Ag, Hbe Ag, anti-Hbe, HB anti cor, IgM and IgG, PCR-DNA; for CVH C diagnosis – antiHCV, IgM and IgG, NS_3, NS_4, PCR-RNA; for CVH D diagnosis – antiHDV, PCR-DNA.

The biochemical analysis of blood (hepatic complex).

The biochemical analysis of blood (renal complex).

Determination of total protein and its fraction in the blood serum: – revealing of disorders of the liver protein-synthetic and signs of mesenchima – inflammatory syndrome;

Coagulogramma – detect coagulated blood system incompetence.

Detection of antinuclear, antismooth muscles, antimitochondria antibodies is required for diagnostic titers within normal or increased limits.

Research of blood electrolits (sodium, calcium, magnesium, copper, chlorine) – revealing of electrolyte dysbalance.

Detection of α-phetoprotein is required for screening on malignant transformation of cirrhosis.

Research of the ceruloplasmin maintenance – etiologic factor establishment (Konkvalov-Wilson's disease).

Ultrasound examination *(ultrasonography)* – revealing of hepatomegalia, sleenomegalia and infringement of hepatic structure.

Fibroesophagogastroduodenoscopia: – revealing of varicous expanded veins of an esophagus and stomach.

Rectoromanoscopia – detection of varicous dilated veins of rectal textures.

Biopsy of the liver with cytoserologial and hystomorphological examination of the bioptates is required for verification of the diagnosis and an establishment of the etiologic factor.

Instrumental non-obligatory methods (under indications):

- static hepatoscyntigraphia;
- dynamic hepatoscyntigraphia;
- rentgenoscopy of the thorax;
- ECG;
- colonoscopy;
- computed tomography and magnetic resonance imaging.

Chapter 11
KIDNEY AND URINARY SYSTEM

There are such syndromes of urinary system: nephritic syndrome, the urinary syndrome, the syndrome of acute renal failure, the syndrome of chronic renal failure.

Nephritic syndrome

Nephritic syndrome (nephrosis, nephrosis sui genesis, minimal disease) – clinical-laboratory symptomocomplex that include proteinuria, hypoproteinemia (hypoalbuminemia), hyperlipidemia and pronounced edema.

Etiology (A position statement from Kidney Disease: Improving Global Outcomes (KDIGO), 2005):

I. Chronic renal disease

A. Essential glomerular diseases;
B. Secondary glomerular damage due to the:
  a) systemic connective tissue diseases;
  b) systemic vasculitis;
  c) diabetes mellitus I or II type;
  d) viral hepatitis B or C type;
  e) the syndrome of arterial hypertension;
  f) later hystosis;
  g) other reasons.
C. Hereditary nephropathies;
D. Non-infection tubulointerstitial diseases;
E. Pielonephritis:
  a) complicated;
  b) non-complicated.
F. Polycystic disease;
G. Damage (disease) of renal transplantation.

II. Acute renal diseases:

A. Glomerulonephritis;
B. Pielonephritis:
  a) complicated;
  b) non-complicated.
C. Tubulointerstitial nephritis;
D. Renal failure:
  a) suprarenal;
  b) renal;
  c) infrarenal.

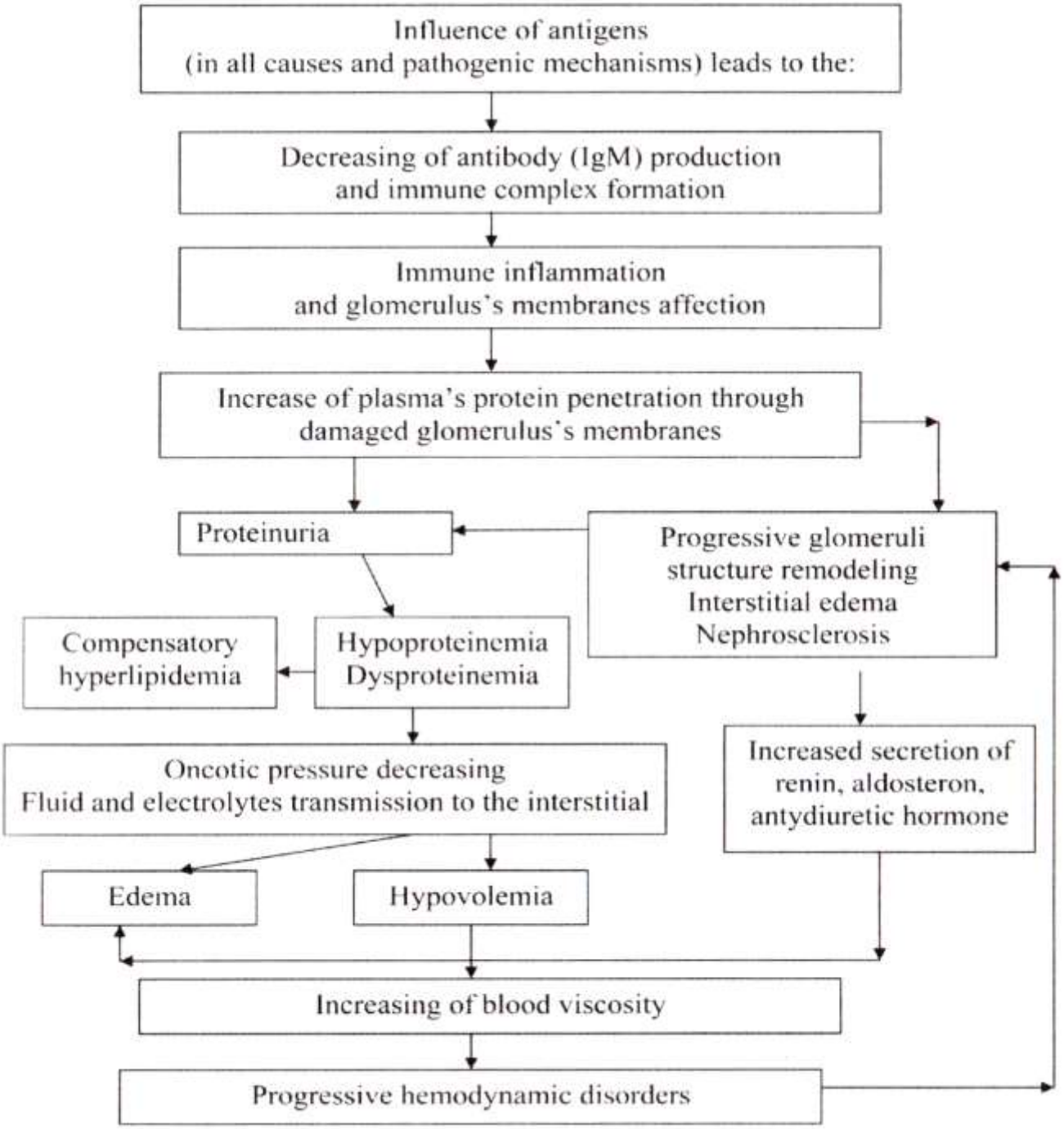
III. Fast advance renal affection:

A. Essential;

B. Secondary.

Pathogenesis

Nephritic syndrome occurs due to the different renal diseases, influence of chemical or toxic exogenous and endogenous factors and based on the immune conflict.



As a spring of the antigens may be:

- exogenous factors: bacterial, viral, parasitic, drugs, foods, junction of heavy metals;
- endogenous factors: DRA, denaturant nucleoproteins, tumor-produced proteins, tyreoglobin.

Depending on the cause and duration there are the next forms of nephritic syndrome

I. According to the cause:

a) Primary nephritic syndrome (due to the essential renal disorders);
b) Secondary nephritic syndrome (due to the complications of extrarenal pathology with autoimmune mechanisms).

II. According to the variants of duration:

1. Episodic – nephritic syndrome occurs only in the initial state of the disease with transmission to the stable remission;
2. Persistent – nephritic syndrome observed during several years without renal failure formation;
3. Progressive – nephritic syndrome for the period of 1–2 years transmitted to the chronic renal failure.

Clinical features

The clinical features more frequently develop gradually accordantly to the proteinuria level and depend on the basic disease.

The main complaint in patients with nephritic syndrome is *edema* that initially arises on the face and in disease progression spreads from the face downward up to hydrothorax, hydropericardium and anasarca.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. In patient may be observed the deranged consciousness in form of stupor, sopor or renal coma.

In general examination detects "*facies nephritica*" – the face is edematous and often pale. Swelling usually appears first around the eyes in the morning and eyes may become slit like when edema is pronounced.

The color of the skin characterized by pathological pale, observed decreased turgor and elasticity of the skin, scars on the abdomen and hips due to the over stretching of the skin.

Edema in patients with nephritic syndrome (edema renalis) characterized by symmetrical localization, in initial stages arises on the face in the morning, has descending character and spreads on extremities, loin region with next fluid accumulation in cavities (hydrothorax, hydropericardium and anasarca). The skin over edema is glossy.

The data of percussion and renal palpation aren't specific.

Addition methods of examination

Clinical blood analysis: anemia, erythropenia, and increase of accelerated ERS.

Clinical urine analysis: olyguria, high specific gravity (1,030–1,040), proteinuria (≈3g/24h). In microscopic study observed large amount of cylinders (hyaline, granular and waxy casts) and tubular (renal) epithelium calls, may be crystals of cholesterin and drops of neutral lipids.

Biochemistry blood analysis: hypoproteinemia (the level of protein less then 60 g/l; albumines 20–30 % with α- and β-globulines increase); hypercholisterinemia (more then 6,6 mmol/l).

Special test. Renal biopsy: use for differential diagnosis and determination of the nephritic syndrome cause.

Urinary syndrome

Urinary syndrome include changes detecting during chemical study of urine – proteinuria and in microscopic study – microhematuria, leukocyturia and cylinduria.

All this parameters may be observed in different combination and degree of changes depends on etiological factors. Furthermore, the urinary syndrome may be accompanied with clinical manifestation of the disease or may be at all only separated diagnostic finding.

Etiology

1. Pathology of the kidney and urinary tract:

I. Chronic renal disease

II. Acute renal diseases:

A. Glomerulonephritis;

B. Pyelonephrities.

III. Fast advance renal affection;

IV. Infection of urinary tract:

A. Infection of urinary tract without certain topic;

a) Complicated;

b) Non-complicated.

B. Acute or chronic cystitis

a) Complicated;

b) Non-complicated.

C. Urethritis, urethrical syndrome

V. Asymptomatic changes of urine:

A. Asymptomatic bacteriuria;

B. Asymptomatic proteinuria or/and erythrocituria.

2. Extrarenal pathology

I. Oncopathology;
II. Heart failure;
III. Influence of exogenous factors;
IV. Enlargement and pathology of prostate gland
V. Tuberculosis.
VI. Impaired metabolism (diabetes mellitus)

Particularities of the urinary syndrome manifestation depend on the disease

Acute glomerulonephritis. In cause of monosyndrome variant characterized by: hematuria (micro- or macrohematuria); moderate proteinuria, cylindruria (erythrocytes unaltered casts, granular casts). In presence of glomerular hematuria, the urine usually contains much protein (protein-erythrocyte dissociation).

Chronic glomerulonephritis. In cause of its latent form, that is the most widespread among other clinical forms of disease, perceive moderate proteinuria, microhematuria and cylindruria (erythrocytes altered casts).

Acute pyelonephritis is characterized by leucocyturia, bacteriuria, moderate proteinuria, insignificant microhematuria, cylindruria (consant hyaline casts) that suggests proteinuria.

Chronic pyelonephritis. Besides the typical clinical features the urinary syndrome in chronic pielonephritis includes leukocyturia, bacteriuria, moderate proteinuria (less than 1g/l), hematuria, cilindruria (unconstant laboratory sign). In development of kidney the level of protein decrease, concentration of urine samples becomes pure, gradually decreased hematuria, leukocyturia and cylindruria.

In Nechiporenko method observed leucocytes/erythrocytes dissociation with prevalence of active leucocytes (the level of neitrophyls 95–100 %).

Acute cystitis. In urine test detect leukocyturia, bacteriuria, proteinuria. The amount of proteins isn't large and depends on the forms elements degradation intensity.

In three glasses test due to the non-glomerular origin of hematuria and inflammation of the urine bladder are determined erythrocytes in last portion.

Chronic cystitis. In period of chronic disease progression in urine test identify leukocyturia, bacteriuria, moderate proteinuria, enlarged mucus level.

Urethritis and urethrical syndrome

Besides the typical clinical features the urinary syndrome in these patients includes leukocyturia, bacteriuria, hematuria and cylindruria (unconstant laboratory sign). In Nechiporenko's method observed leucocytes/erythrocytes dissociation with prevalence of active leucocytes (the level of neutrophyls 95–100 %).

Kidney congestion

Congestion kidney or kidney in cardiovascular diseases is clinical syndrome that develops on heart failure (more frequently on congestion in large circle circulation). The most significant sign is moderate proteinuria. In microscopic study of urine detect non-significant amount of erythrocytes, leucocytes and gyaline cylinders.

In such cause the urinary syndrome correlates with heart failure intensity and cardiac function compensation associated with urine test normalization.

Diabetic nephropathy

In diabetic mellitus renal pathology may debut by microalbuminuria which progression connected with glycemia level. In microscopic study of urine observe gyaline cylinders and erythrocytes. Moreover, metabolic profile particularities lay in the basis of intermittent pelvis renalis and urinary tract inflammation that associated with leucocyturia and bacteriuria.

Podagric nephropathy

Podagric nephropathy develops due to the increased urine acid level in the blood via impaired nucleoprotein metabolism. Parallel to clinic of joint damage develop visceral complications among which nephropathy due to the interstitial injury takes the fist place. This process manifests by non-significant urinary syndrome – non-significant proteinuria, microhematuria and cylindruria. Leukocyturia appears in case of secondary infection. In addition, via impaired urine acid metabolism, in patients frequently observed stone formation that clinically can manifests by renal colic. In such condition detect urinary acid, increase erythrocyturia till macrohematuria.

Syndrome of acute renal failure

Syndrome of acute renal failure is as usual develops suddenly due to the acute renal damage and can be inversed.

Classification depending on the cause, character of duration and period:

According to the cause (if it known) (tab. 4.1):

– suprarenal;

– renal;

– subrenal.

According to the gravity stage:

I – the impairments that compensated by conservative treatment without infusive therapy;

II – the impairments that need infusive therapy;

III – the impairments that need dialysis treatment.

According to the period:

- the period of anuria/oliguria;
- the period of diuresis reparation;
- the period of complete renal function reimbursement.

Table 4.1. ***The forms of acute renal failure (according to the cause)***

Suprarenal	Renal	Subrenal.
Acute decline of blood circulated volume: via acute blood pressure decrease in all types of shock, bleeding, diarrhea and vomiting and overdose of diuretics.	Renal vessels occlusion, inflammatory renal diseases, intoxication by nephrotoxic substances.	Urinary tract obstruction by uratis, oxalates, blood clots, proteins coagulates and adenoma of prostate gland.

Clinical features

Strength of acute renal failure clinical sings, particularly in the initial stage, depends on the etiologic factor. Within disease progression, differences in clinical picture become smoothed and complaints explained by intoxication via abnormal nitrogen metabolism.

Depending on disease particularities the primary position obtains the next clinical symptoms and syndromes:

- respiratory syndrome: dyspnea, the clinic of dry or effusive pleurisy;
- affection of the cardiovascular system: arterial hypotension, pericarditis, arrhythmias and acute left ventricular failure;.
- gastro-intestinal syndrome: loss of appetite, dysphagia, nausea, vomiting, stomatitis, gastric and intestinal ulcer formation, the signs of peritoneum irritation);
- neurological syndrome and central nervous system damage: in initial state – symptoms of asthenia (fatigue, memory impairments, irritation, dreadful sleeping); symptoms of depression (bad mood, decreased mental activity, suicidal ideas) and headache; later – deranged consciousness (stupor, sopor, coma), vascular complications (hemorrhagic or ishematic stroke) and small paresis and paralysis;
- anemic-hemorrhagic syndrome: anemia, sometimes leukocytosis, eruption and dryness of the skin, hemorrhage lesions;
- dismetabolic syndrome: Pain and muscular weakness, cramps, proximal myopathy, aseptic bones necrosis, arthritis, intra and subcutaneous calcinatis,

accumulation of urine crystals in the skin, ammonium smelling from the mouth and hyperlipidemia;

– affection of immune system: intercurrent infections.

Objective examination. *General patients condition* from moderate grave to extremely grave. In patient may be observed the deranged consciousness.

The posture of the patients is frequently active with restrictions or passive.

The skin and visible mucosa are pale and dry.

In objective examination frequently observed the data of purulent parotitis, rhinitis, bronchitis, pleurisy and pericarditis.

Palpation of the kidney is painful due to their edema and enlargement in size.

Additional methods of examination

Clinical blood analysis: anemia, erythropenia, leukocytosis.

Biochemistry of the blood: increase of creatinin, ammonium and urine acid levels.

Blood electrolytes detection: – the level of potassium, magnum, sulphatis and phosphates is increased; sodium, calcium, chloral and hydrocarbonatis concentration is decreased. Specific – metabolic acidosis.

Glomerulus's filtration speed – is decreased.

Clinical urine analysis: at the initial stage and period of anuria/oliguria observed decreased amount of excreted urine, it is dark yellow or red-yellow ("meat slops") color with high specific gravity. In period of diuresis reparation (from several days to 3–4 weeks after disease beginning) is observed polyuria, isuria, hyposthenuria and low specific gravity (less than 1,018). In some patients can be distinguish moderate proteinuria, hematuria and leukocyturia. In grave causes occurs anuria.

Syndrome of chronic renal failure

Syndrome of chronic renal failure – clinical-laboratory symptomocomplex that occurs due to the significant decrease of nephrones quantity and quality that leads to the impaired secretory and excretory renal function, homeostasis disbalance, disturbances of all substances exchange, acid-alkaline disorder and abnormal all organs and systems work.

Etiology chronic renal failure is a final of different kidney disease

The most frequent causes:

– glomerulonephritis (≈30 %);
– pyelonephritis (≈20 %);
– polycystic disease (≈10 %);
– systemic disease with renal injury (≈8 %);
– hereditary nephropathies (≈10 %);

– tumor of the kidney (≈5 %);
– other pathology (≈7 %);
– unknown etiology (≈10 %).

Pathogenesis

In chronic renal failure define not only decrease of nephrones quantity but also significant remodeling of the last one (hypertrophy and dilation). The process develops step by step – from latent functional incompetence to significant uremia. Accordanly to renal failure progression increase impossibility of kidney for metabolic products excretion that leads to their accumulation in organism.

Metabolic disorders complicated by uremia intoxication that leads to: nitrousemia, anemia, osteodystrophy, acid-alkaline disbalance, arterial hypertension, hemorrhagic syndrome and immune deficiency.

The clinic of chronic renal failure augments gradually with slowly changes of homeostasis: increase concentration of creatinin and uric acid in plasma, levels of guanidine acid, sulfates, phosphates and other metabolites. Metabolic acidosis develops. With oliguria advance patient's condition becomes worth: hurriedly increase nitrousemia and acidosis, decrease sodium, calcium and chloral level with hyper concentration in plasma of magnum and potassium. Combination of those impairments lays in the basis of renal failure symptoms.

Clinical features

Intensity of chronic renal failure clinical sings, particularly in the initial stage, depends on the etiologic factor. Within disease progression, differences in clinical picture become smoothed and complaints explained by intoxication via abnormal nitrogen metabolism.

Depending on disease particularities the primary position obtains the next clinical symptoms and syndromes:

– affection of the cardiovascular system: arterial hypertension, pericarditis, uremic cardiomyopathy, arrhythmias and acute left ventricular failure;
– gastro-intestinal syndrome: mucosa injury – cheilitis, glossitis, stomatitis, esophagitis, gastritis, enteritis, colitis, gastric and intestinal ulcer; organic glands damage (parotisis, pancreatitis);
– neurological syndrome and central nervous system damage: uremic encephalopathy: symptoms of asthenia (fatigue, memory impairments, irritation, dreadful sleeping); symptoms of depression (bad mood, decreased mental activity, suicidal ideas); phobias, changes of character and conduct (emotional weakness, indifference, eccentric conduction); deranged consciousness (stupor, sopor, coma), vascular complications (hemorrhagic or ishematic stroke); uremic polyneuropathy: small paresis and paralysis, other changes of felling and moving function;

– endocrine syndrome: endocrine pathology (hyperparathyreoidism, loss of libido, impotencies, impairment of spermatogenesis, hynecomastia, olygo- and menorrhea, sterility); pain and muscular weakness, cramps, proximal myopathy, aseptic bones necrosis, arthritis, intra and subcutaneous calcinatis, accumulation of urine crystals in the skin, ammonium smelling from the mouth and hyperlipidemia);
– anemic-hemorrhagic syndrome: anemia (normochromic, sometimes erythropoetin deficient or iron deficient), lymphopenia, non-significant thrombocytopenia; clinical symptoms (pale color of the skin and visible mucosa with yellowish tint, eruption and dryness of the skin, hemorrhage lesions;
– affection of immune system: intercurrent infections, decrease of immunity.

There are such signs of chronic renal failure according to the periods:

I. The early signs of chronic renal failure:

1. Clinical: polyuria, nocturia, arterial hypertension, hypochromic anemia;
2. Laboratory: decrease of concentrated and filtrated function of kidneys.

II. The late signs of chronic renal failure:

1. Laboratory: nitrousemia (increased creatinin level, ammonium and urine acid concentration in plasma);
2. Instrumental: decrease of both renal size and cortex according to ultrasound examination and uroroentgenogram.

Additional methods of examination

Clinical blood analysis: anemia, erythropenia.

Biochemical blood analysis: increase of creatinin and ammonium levels, hyperuricemia (non-constant sign). Blood electrolytes detection: – decrease of sodium and calcium concentration, the level of chloral normal or decreased, the level of potassium normal or increased, concentration of magnum and phosphorus increased. Specific is development of metabolic acidosis.

Determination of the glomerulus's filtration rate:

– radiological method – with use of inulini iotalamatis, EDTA;

– classic method – according to plasma's creatinin level, its 24h urine excretion and diuresis per minute;

– method of Galt-Cockroft – calculation by formula, where:

$$\text{Creatinin clearance} = \frac{140 - \text{age (years)} \times \text{weight (kg)}}{\text{Creatinin in plasma (mmol/l)} \times 810}$$

Decrease of glomerulus's filtration (GF) – is the earliest sign of renal failure.

Clinical urine analysis: at the initial stage observed polyuria, nocturia, isuria and hypostenuria, low specific gravity (less than 1.018), in late stages – oliguria till anuria, hypostenuria and isuria stay be present.

***Additional instrumental methods of examination*:** plain radiography of the urinary tract, excretion urography (synonyms: intravenous pyelography; IVP; IVU), retrograde pyelography/ureterography, renal arteriography, renal venography, computed tomography (CT scanning), magnetic resonance imaging (MRI).

Nuclear renal imaging. The value of radiolabelled traces in the investigation of renal disease lies in the ability to obtain important information about organ function as opposed to the predominantly structural information obtained from the previously described imaging procedures. In particular, nuclear imaging of the kidneys provides the only non-invasive quantitative assessment of individual kidney function. Radionuclides (such as ^{123}I, $^{99}Tc^{m}$) are linked to compounds that depend on either glomerular filtration alone, tubular excretion, or a combination of both for excretion from the body. These compounds can therefore provide quantitative information on these functions of the kidney, in addition to dynamic images.

Renal biopsy: use for differential diagnosis and determination of the chronic renal failure cause.

Classification of chronic renal diseases (NKF, USA)

Stage	Characteristic	Glomerular filtration rate (GFR, ml/min/1.73 m^2)	Recommendation
	Risk factors presence	More than 90	Observation, risk factors correction
I	Renal damage with normal or decreased GRF	More than 90	Lowering of risk progression of the main desease
II	Renal damage with insignificant decreased GRF	60–89	Lowering of risk progression of the main desease and cardiovascular complications
III	Moderate degree of GRF decreasing	30–59	Complications treatment
IV	Significant degree of GRF decreasing	15–29	Preparing to replacement therapy
V	Chronic renal failure	Less than 15 or dialysis	Replacement therapy

Glomerulonephritis

Glomerulonephritis is immuno-inflammatory renal disease with obligatory glomerulus's injury and including to the pathological process of all renal structures.

In more cases glomerulonephritis is an independent nozological form but may be a result of systemic pathology or pathological states.

Classification of glomerulonephritis

I. Acute glomerulonephritis

1. According to the variant:
   - with urinary syndrome;
   - with nephritic syndrome.
   - with hypertension syndrome;
   - mixed.
2. According to the duration:
   - recidivated;
   - lingering.

II. Subacure glomerulonephritis

III. Fast advance glomerulonephritis.

IV. Chronic glomerulonephritis

1. According to the variant:
   - with urinary syndrome;
   - with nephritic syndrome.
   - with hypertension syndrome;
   - mixed:
   - latent.
2. According to the stage:
   - anhypertensive;
   - hypertensive;
   - renal failure:

- the period of anuria/oliguria;
- the period of diuresis reparation;
- the period of complete renal function reimbursement.

3. According to the duration:
   - stable;
   - progressive.
4. According to the phase:
   - remission;
   - aggravation.

Etiology

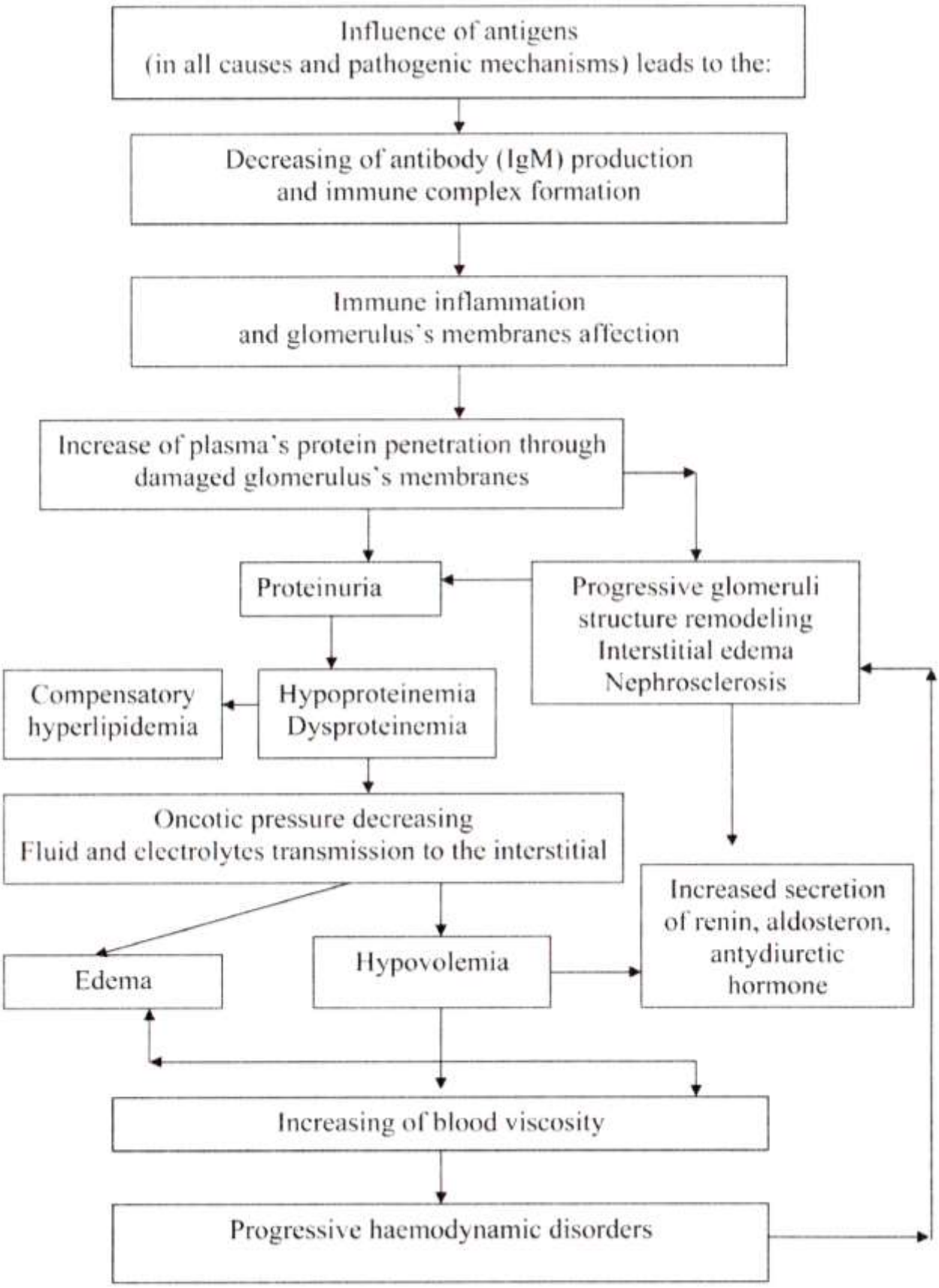
I. Influence of infection agent (primary streptococcus);

II. Endogenous antigens:
- systemic connective tissue diseases;
- systemic vasculitis;
- diabetes mellitus I or II type;
- viral hepatitis B or C type;
- the syndrome of arterial hypertension;
- later hystosis;
- other reasons.

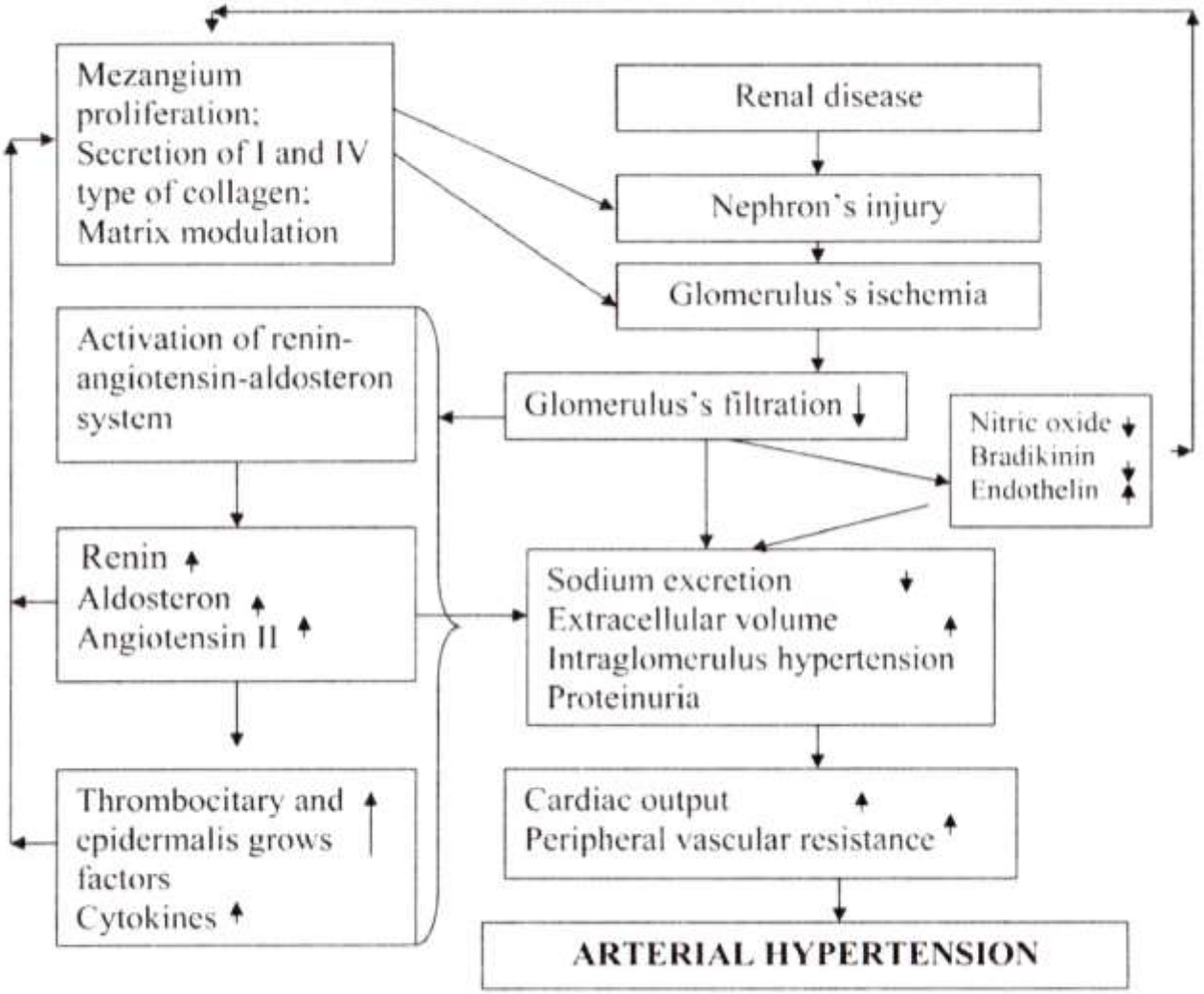
III. Exogenous antigens:
- alcohol;
- toxic substances;
- poisons;
- bite of animals and insect;
- drugs.

IV. Hereditary origin.

Pathogenesis (in most causes is similar to nephritic syndrome formation)



Pathogenesis of arterial hypertension on glomerulonephritis



Acute glomerulonephritis

Acute glomerulonephritis is acute immuno-inflammatory renal disease with obligatory glomerulus's injury and afterward including to the pathological process of all renal structures.

Clinical features

The main complaint in patients with acute glomerulonephritis are weakness, thirst, pain in the back, dyspnea, palpitation, headache, nausea, vomiting, edema and lost of vision.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. In general inspection detect "*facies nephritica*" – the face is edematous and often pale. Swelling usually appears first around the eyes in the morning and eyes may become slit like when edema is pronounced.

The color of the skin characterized by pathological pale, observed decreased turgor and elasticity of the skin, scars on the abdomen and hips due to the over stretching of the skin.

Edema in patients with nephritic syndrome (edema renalis) characterized by symmetrical localization, in initial stages arises on the face in the morning, has descending character and spreads on extremities, loin region with next fluid accumulation in cavities (hydrothorax, hydropericardium and anasarca). The skin over edema is glossy.

In heart percussion observed displacement of the left border of the heart to the left.

In heart auscultation detects tachycardia and gallops rhythm. Arterial pressure is increased.

Complications: acute renal failure, acute heart failure, encephalopathy, stroke, eclampsia, transitory vision impair.

Outcomes of disease: complete recovery, transformation to the chronic form.

Additional methods of examination

Clinical blood analysis: leukocytosis and increase of accelerated ESR.

Clinical urine analysis: in macroscopic study – urine is "meat wastes" color, cloudiness, without odor, olyguria, low specific gravity and moderate or significant proteinuria: in microscopic study observed large amount of altered erythrocytes (hematuria), cylinders (hyaline, erythrocytes and waxy casts) and leucocytes (non-constant).

Zimnitsky's test: izostenuria.

Nechiporenko's method: prevalence of erythrocytes under leucocytes: casts more 250 in ml.

Biochemistry of the blood: hypoproteinemia (hypoalbuminemia) and dysproteinemia

Renal biopsy: use for differential diagnosis and determination of the glomerulonephritis cause.

Fast advance glomerulonephritis

Fast advance glomerulonephritis – the variant of glomerulus's injury that characterized by fast development of renal failure and uremia. The death comes at 6–18 months from disease onset.

Clinical features

The main complaints in patients with fast advance glomerulonephritis are pain in the back, edema and constant excretion of red urine.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. In general examination detects "*facies nephritica*" and edema renalis with next fluid accumulation in cavities.

The color of the skin characterized by pathological pale, observed decreased turgor and elasticity of the skin, scars on the abdomen and hips due to the over stretching of the skin. The skin over edema is glossy.

In objective examination detect displacement of the left border of the heart to the left, tachycardia and gallops rhythm, stable secondary arterial hypertension, retina break off and fast clinical progression of uremia.

Complications: renal failure, heart failure, encephalopathy, stroke, eclampsia.

Outcomes of disease: fast progression with poor prognosis, renal failure and death.

Additional methods of examination

Clinical blood analysis: leukocytosis and increase of accelerated ESR, anemia.

Clinical urine analysis: in macroscopic study – urine is "meat wastes" color, cloudiness, without odor, olyguria, low specific gravity and significant proteinuria: in microscopic study observed large amount of altered erythrocytes (hematuria), cylinders (hyaline, erythrocytes and waxy casts) and leucocytes (non-constant).

Zimnitsky's test: olyguria, nocturia, izostenuria.

Nechiporenko's method: prevalence of erythrocytes under leucocytes: casts more 250 in ml.

Biochemical blood analysis: increase of creatinin, ammonium and urine acid levels, hypoproteinemia (hypoalbuminemia) and dysproteinemia, increased potassium, magnum, sulphatis and phosphates level with sodium, calcium, chloral and hydrocarbonatis concentration is decreased. Decrease of Glomerulus's filtration rate.

Renal biopsy: use for differential diagnosis and determination of the glomerulonephritis origin.

Chronic glomerulonephritis (nephritic form)

Chronic glomerulonephritis (nephritic form) is the variant of glomerulus's injury that characterized by the prevalence in clinic of nephritic syndrome signs.

Clinical features

The clinical features more frequently develop gradually accordantly to the proteinuria level.

The main complaint in patients with nephritic syndrome is *edema* that initially arises on the face and in disease progression spreads from the face downward up to hydrothorax, hydropericardium and anasarca.

Objective examination. General patient's condition is from moderate grave to extremely grave.

In general examination is detected "*facies nephritica*" – the face is edematous and often pale. Swelling usually appears first around the eyes in the morning and eyes may become slit like when edema is pronounced.

The color of the skin characterized by pathological pale, observed decreased turgor and elasticity of the skin, scars on the abdomen and hips due to the over stretching of the skin.

Edema in patients with nephritic syndrome (edema renalis) characterized by symmetrical localization, in initial stages arises on the face in the morning, has descending character and spreads on extremities, loin region with next fluid accumulation in cavities (hydrothorax, hydropericardium and anasarca). The skin over edema is glossy.

In heart auscultation detects decreased loudness of the heart sounds. The data of percussion and renal palpation aren't specific.

Complications: edema of the brain, edema of the retina, shock, phlebothrombosis, infections, chronic renal failure, nephritic crisis.

Outcomes of disease: the duration is persistence or continuously progressive.

Addition methods of examination

Clinical blood analysis: anemia, erythropenia, and increase of accelerated ESR.

Clinical urine analysis: olyguria, high specific gravity (1,030–1,040), proteinuria (≈3g/24h); in microscopic study observed large amount of cylinders (hyaline, granular and waxy casts) and tubular (renal) epithelium calls, may be crystals of cholisterin and drops of neutral lipids, microhematuria – non-constant sign.

Zimnitsky's test: olyguria, nocturia, izostenuria.

Biochemical blood analysis: hypoproteinemia (the level of protein less then 60 g/l; albumines 20–30 % with α- and β-globulines increase); hypercholisterinemia (more then 6.6 mmol/l).

Renal biopsy: use for differential diagnosis and determination of the chronic gromerulonephritis origin.

Chronic glomerulonephritis (hypertensive form)

Chronic glomerulonephritis (hypertensive form) is the variant of glomerulus's injury that characterized by the stable blood pressure increase.

Clinical features

The clinical features more frequently develop gradually accordanly to the proteinuria level.

The main complaint in patients are headache, lost of vision, dizziness, lost of sleeping and *edema* that initially arises on the face and in disease progression spreads from the face downward up to hydrothorax, hydropericardium and anasarca.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. In general examination detects "*facies nephritica*", pale color of the skin and visible mucus, edema renalis.

In heart percussion detect displacement of the left heart border outward and downward. *In heart palpation* – apex beat displaced outward and downward, diffuse, high and strong strength. *In heart auscultation* detects decreased loudness of the heart sounds, accentuated II heart sound over aortic valve. Stable blood pressure increases (with high diastolic blood pressure level).

Complications: edema of the brain, edema of the retina, myocardial infarction, cardiac asthma, encephalopathy and chronic renal failure.

Outcomes of disease: the duration is persistence or continuously progressive.

Additional methoda of examination

Clinical blood analysis: without significant changes.

Clinical urine analysis: decrease of specific gravity, proteinuria; in microscopic study cylinderuria, microhematuria.

Zimnitsky's test: olyguria, nocturia, izostenuria.

Biochemcal blood analysis: increase of creatinin, ammonium and urine acid levels, decrease of Glomerulus's filtration speed, non-constant – hypoproteinemia and dysproteinemia.

***ECG*:** the signs of left ventricle hypertrophy and systolic overload.

***In ophthalmoscope examination*:** the signs of renal retinopathy – edema of the retina, spasm of the arteries, dilation of the veins, hemorrhages.

Renal biopsy: use for differential diagnosis and determination of the chronic gromerulonephritis origin.

Chronic glomerulonephritis (mixed form).

Chronic glomerulonephritis (mixed form) is the variant of glomerulus's injury that characterized by the presence of hypertensive and nephritic syndromes.

Clinical features

The clinical features more frequently develop gradually accordantly to the proteinuria level.

The main complaint in patients is *edema* that initially arises on the face and in disease progression spreads from the face downward up to hydrothorax, hydropericardium and anasarca.

Also may be present general weakness, thirst, loss of appetite, headache, vision changes and dizziness.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. In general examination detects "*facies nephritica*" and edema renalis with next fluid accumulation in cavities.

The color of the skin characterized by pathological pale, observed decreased turgor and elasticity of the skin, scars on the abdomen and hips due to the over stretching of the skin. The skin over edema is glossy.

In objective examination detect displacement of the left border of the heart to the left, tachycardia, decrease loudness of the heart sounds and stable secondary arterial hypertension.

Complications: cardiac asthma, pulmonary edema, stroke, shock and chronic renal failure.

Outcomes of disease: the duration is persistence or continuously progressive.

Additional methods of examination

Clinical blood analysis: moderate leukocytosis and increase of accelerated ESR (in stage of aggravation), anemia.

Clinical urine analysis: in initial stage – poliuria, in late – olyguria with low specific gravity and significant proteinuria: in microscopic study observed non-constant hematuria, cylinders (hyaline, erythrocytes and waxy casts) and leucocytes (non-constant).

Zimnitsky's test: olyguria, nocturia, hypo- or izostenuria.

Biochemical blood analysis: increase of creatinin, ammonium and urine acid levels, decrease of creatinin clireance, non-constant – hypoproteinemia and dysproteinemia.

ECG: the signs of left ventricle hypertrophy and impaired repolarization.

Renal biopsy: use for differential diagnosis and determination of the glomerulonephritis origin.

Chronic glomerulonephritis (latent form)

Chronic glomerulonephritis (latent form) is the variant of glomerulus's injury that characterized by the non-symptomatic duration or clinical manifestation with isolated urinary syndrome.

Clinical features

As usual the patients don't have any complaints.

Objective examination. *General patient's condition* is from satisfactory to moderate grave. During general examinations in the early stagers of disease aren't

detect any particularities. In later stagers may appears clinic of nephritic and hypertensive syndromes.

Complications: development of chronic renal failure.

Outcomes of disease: the duration is slowly progressive.

Additional methods of examination

Clinical blood analysis: anemia.

Clinical urine analysis: in initial stage – normal specific gravity and non-significant proteinuria: in microscopic study observed non-constant hematuria, cylinders (hyaline, erythrocytes and waxy casts) and leucocytes (non-constant).

Nechiporenko's method: prevalence of erythrocytes under leucocytes: casts more 250 per ml.

Biochemical blood analysis*:* increase of creatinin, ammonium and urine acid levels and decrease of creatinin clireance detect in 25 % of patients, non-constant – hypoproteinemia and dysproteinemia.

ECG: may be the signs of impaired repolarization.

Renal biopsy: use for differential diagnosis and determination of the glomerulonephritis origin.

Pyelonephritis

Pyelonephritis – inflammatory renal disease with obligatory renal parenchyma and pelvis injury.

Classification of pyelonephritis

I. According to the duration:

- acute;
- chronic.

II. According to the complication development:

- complicated;
- non-complicated

Etiology

I. Primary infection of renal structures (more frequently bacterial, protozoa, fungus);

II. Secondary to the:

1. Renal system pathology:
   - urine tract infection and injury (cystitis, urethritis, strangulation of stones or foreign bodies in the urethra; phimosis);
   - prostate gland diseases (prostatitis, prostate adenoma, prostate tumor);
   - uterus and uteri cervical diseases.

2. Extra renal pathology:
   – sepsis;
   – diabetes;
   – inflammatory process with different localization;
   – immune deficiency states;
   – post operative period.

Pathogenesis

Infectious agents may be transmitted by contact, hematogenous or lymphatic ways in obligatory presence of urodynamic abnormalities:

1. Primary vesicourethral reflux due to the:
   – urine bladder obstruction,
   – inflammation or mucus edema in zone of ureters confluence;
   – obstruction of renal pelvis;
   – ureters stricture;
   – other reasons.
2. Pyelovenous or pyelolymphatic reflux with effortlessly transmission of microbus agent to the renal vessels and structures through increased pelvises pressure.

Subsequently, over impaired urodynamic, infections and inflammation may spread in either ascendant or descendent ways.

Acute pyelonephritis

Acute pyelonephritis – acute non-specific inflammatory process that characterized by primary affection of renal parenchyma, renal pelvis and tubules with futher involvement to the pathological process of glomerulus and vessels.

Clinical features

The main complaints in patients with acute pyelonephritis are fever, dull, constant and increasing in intensity pain in the back, perspiration, headache, nausea, vomiting and pain in the muscles, arthralgia and disorders of urination.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. The temperature is constant increase to 38–39,5° C or has hectic type. May observed the clinic of bacterial shock. Pasternatsky's symptom is positive on one or bilateral, may detect tenderness of the muscles in loin region.

Complications: renal abscess, urosepsis, paranephritis.

Outcomes of disease: complete recovery, transformation to the chronic form.

Additional methods of examination

Clinical blood analysis: leukocytosis and accelerated ERS.

Clinical urine analysis: in macroscopic study – urine is yellow color, cloudiness, without odor, high specific gravity and non-significant proteinuria: in microscopic study observed large amount of leucocytes, pyuria, non-concstant microhematuria, cylindruria, in bacteriological study – bacteriuria.

Nechiporenko's method: prevalence of leucocytes under erythrocytes: casts more 250 in ml.

Biochemical blood analysis: without changes.

Additional instrumental methods: excretory urographia, ultrasound examination.

Chronic pyelonephritis

Chronic pyelonephritis – chronic non-specific inflammatory process that characterized by primary affection of renal parenchyma, renal pelvis and tubules with father involvement to the pathological process of glomerulus and vessels.

Clinical features

The main complaints in patients with chronic pyelonephritis are sub febrile fever, dull, constant loin pain, perspiration, headache, nausea, arthralgia and disorders of urination.

Objective examination. *General patient's condition* is from moderate grave to extremely grave. The temperature is periods of progression increase to 38–39,5° C or has hectic type. May observed lost of weight. Pasternatsky's symptom is positive on one side or bilateral, may detect tenderness of the muscles in loin region. In part of patients detect the syndrome of arterial hypertension.

Complications: chronic renal failure.

Outcomes of disease: duration with period of remission and progression, prognosis – satisfactory.

Addition methods of examination

Clinical blood analysis: leukocytosis and accelerated ERS, anemia, erythropenia.

Clinical urine analysis: in macroscopic study – urine is yellow color, cloudiness, without odor, high specific gravity and non-significant proteinuria: in microscopic study observed large amount of leucocytes, non-constant microhematuria, cylindruria, in bacteriological study – bacteriuria.

Zimnitsky's test: in normal limited.

Nechiporenko's method: prevalence of leucocytes under erythrocytes: casts more 250 per ml.

Biochemical blood analysis: may observed increase of creatinin level.

Additional instrumental methods: excretory urography, ultrasound examination, renography, computed tomography, magnetic resonance imaging

Chapter 12

BLOOD SYSTEM

There are such syndromes of the pathology of the blood system: the syndrome of anemia, myeloproliferative syndrome, syndrome of bleeding disorders.

Syndrome of anemia

Anemia is a clinical syndrome associated with reduction in the number of total red blood cells and/or hemoglobin concentration in a blood unit volume.

There are two main classifications of anemia:

1. The etiological classifications based on the cause of the anemia.
2. The morphological classification based on the characteristics of the red cell as determined by blood examination.

Classification of anemia

I. Blood loss:

- acute post-hemorrhagic anemia;
- chronic post-hemorrhagic anemia.

II. Impaired red cell formation:

Disturbance of bone marrow function due to deficiency of substances essential for erythropoiesis:

- iron deficiency anemia;
- megalobastic macrocytic anemias due to deficiency of vitamin B_{12} or folic acid;
- aplastic anemia.

III. Increased red cell destruction (hemolytic anemias):

- hemolytic anemias due to corpuscular defect (intracorpuscular or intrinsic abnormality). The basic defect may in any of three main components of the cell: the membrane, the hemoglobin molecule and the enzymes related to cell metabolism;
- hemolytic anemias due to an abnormal hemolytic mechanism (extracorpuscular or extrinsic abnormality). These are acquired and result from either an immune or non-immune mechanism.

Etiology

Etiology of anemia different and include some factors: genetic, environmental, influence of chronically diseases, infections, jatrogenic factors, hemorrhage.

Pathogenesis

Pathogenesis depends on the forms of anemia.

Clinical features

Clinical features of anemia reflect the diminished oxygen-carrying capacity of the blood, reduced supplying oxygen to the organs and tissue such as heart, brain, muscles.

The general symptoms and signs appear which are common to all anemias: fatigue, malaise, lassitude, weakness, loss of strength, reduced capacity for exercise, shortness of breath, especially during exercise – dyspnea, difficult breathing when lying down, palpitation, pain in the heart, anorexia, nausea, flatulence, constipation and diarrhea. Neurological disorders occur: digginess, headache, faintness, inability to concentrate. In women with childbearing age may be observed menstrual irregularity and even amenorrhea, defects of urination.

During objective examination the pallor skin, mucous membranes of mouth and nail beds may be detected. The evaluation of slight cyanosis may be made. Cyanosis in patients with anemia appears due to the total amount of reduced hemoglobin; methemoglobin or sulfhemoglobin present. Jaundice may be observed in the conjunctivae, mucous membranes and skin in anemic patients with hemolytic components.

Objective examination of the respiratory system reveals increased depth and rate of respiration in patients with severe anemia. Cardiovascular disorders due to the anemia are tachycardia, sometimes cardiac enlargement, functional cardiac murmurs, diminished heart sounds, low volume of pulse, hypotension.

Gastrointestinal dysfunction of anemic patients are characterized by smooth and red mouth, enlarged spleen or/and liver in some cases.

Except these symptoms and signs there are a lot of clinical specific features relevant to every kind of anemias.

Additional methods of examination

Additional methods of examination include: erythrocyte count, determination of hemoglobin, red cell absolute values, erythrocyte morphology, bone marrow examination, bilirubin measurement, estimation of iron metabolism, evaluation of hemoglobin structure and biosintesis, immunogematological tests for detection of hemolytic anemia.

Clinical blood analysis

Normal values of erythrocytes count:

men $5,5 \pm 1,0 \times 10^{12}$/l;

women $4,8 \pm 1,0 \times 10^{12}$/l.

The International Committaee for Standardization in Hematology has recommended that the following units be used (SI units): hemoglobin, "g/dl" (deciliters).

Normal values of hemoglobin:

men	15,5±2,5 g/dl;
women	14,0±2,5 g/dl.

Red cell absolute values

These can be calculated from:

a) hematocrit packed cell volume (PCV);
b) hemoglobin estimation;
c) red cell count.

The International Committee for Standardization in Hematology has recommended that the following units be used (SI units): main cell volume as "fl" (femtoliters), mean cell hemoglobin as "pg" (picograms) and mean cell hemoglobin concentration as "g/dl".

Red cell absolute values are analyzed using the formulas:

the mean cell volume (MCV):

$$MCV = \frac{\text{Packed cell volume} \times 10^{15}}{\text{Red cell count per litre}} \text{ fl}$$

the mean cell hemoglobin (MCH):

$$MCH = \frac{\text{Hemoglobin in gm\%} \times 10^{13}}{\text{Red cell count per litre}} \text{ pg}$$

the mean cell hemoglobin concentration (MCHC):

$$MCHC = \frac{\text{Hemoglobin in g\% gm \%}}{\text{Packed cell volume}}$$

Normal values of packed cell volume (PCV: hematocrit):

men	0,47±0,07 l/l	40–54 %;
women	0,42±0,05 l/l	37–47 %;
infants, full-term, cord blood	0,54±0,10 l/l	44–64 %;
children, 3 months	0,38±0,06 l/l	32–44 %;
children, 10–12 years	0,41±0,04 l/l	37–45 %.
Mean cell volume (MCV):		
adults	86±10 fl	76–96 fl;
infants, full-term, cord blood	106 fl (mean);	
children, 1 year	78±8 fl	70–86 fl;
children, 10–12 years	84±7 fl	77–91 fl.
Mean cell hemoglobin (MCH):		
Adults	29,5±2,5 pg	27–32 Pg %.
Mean cell hemoglobin concentration (MCHC):		
adults and children	33±2 g/dl	31–35 g %.

Colour index. Once the quantity of erythrocytes and hemoglobin in a given blood specimen is known, it is possible to calculate the hemoglobin content of each

erythrocyte. There are many methods by which hemoglobin saturation can be determined. One of them is the calculation of the *colour index*. This is a conventional value derived from the ratio of hemoglobin to the number of erythrocytes. This value is found by dividing a triplet quantity of hemoglobin in grams by the first three figures expressing the quantity of erythrocytes. Normally this value approaches 0,85–1,1. If it is less than 0,8, the erythrocyte saturation of hemoglobin is insufficient; it the value exceeds 1,1 the volume of erythrocytes is higher than normal.

Red blood cell morphology can be determined from a thin blood film stained with Romanowsky dyes. The three basic features of a red blood cell are its size, its shape and its inclusions.

Size of erythrocytes. Normal erythrocyte are nearly uniform in size with diameter of 7,2 to 7,9 nm. An increasing and decreasing in the size of a red blood cell is known as anisocytosis.

Shape of normal erythrocytes is a biconcave disc, which is thickest at its edges. The presence of many abnormal shapes on a blood smear is known as poikilocytosis. Qualitative changes of erythrocytes are depicted in table 5.1.

Inclusions in erythrocytes. The normal red blood cell filled mainly with hemoglobin. In pathological states blood films will show red blood cells with colored spots or rings inside their cytoplasm.

Howell-Jolly bodies. These are small, well-defined, round, densely staining basophilic inclusion bodies about 1 μm in diameter, which usually occur singly but sometimes in multiples. They appear after splenectomy and are also seen in cases of severe anemia from a variety of causes. They contain DNA and may be chromosomal remnants or nuclear fragments.

Cabot rings. These are blue-staining, threadlike inclusions in the red cells in severe anemia. They may appear as rings, or twisted and convoluted in a variety of shapes. They may occupy the entire periphery of the cell but frequently are much smaller. They are not often seen. It has been postulated that they are remnants of the mitotic spindle, but others have found that they contain histone and iron.

Heinz bodies can be seen with special supravital stains such as methylviolet Heinz bodies are granules of precipitated hemoglobin.

Bone marrow examination emploed to aspirate some cells of bone marrow using special technique – aspirate needle with stylet. A needle is inserted in to the soft center of a bone and a small quantity of bone marrow tissue is aspirated. The contents are smeared, then fixed, stained and examined. A bone marrow smear give information about proportion of the different cell lines, about size and shape of developing cells, about architecture of the bone marrow. In adults aspiration is usually done from the sternum and the iliac crest.

Table 5.1. ***Description and significance of various forms of red blood cells***

Type of cell	Description	Physiologic significance	Clinical disorders
Macrocyte	Larger than normal (>7,9 nm in diameter). Well filled with hemoglobin	1. Young red blood cells (RBC) 2. DNA Synthesis-impaired, megaloblastic maturation	1. Accelerated erythropoiesis 2. B_{12} or folate deficiency.
Thin macrocyte	Diameter increased but MCV normal; often hypochromic (see target cells)	Membrane cholesterol and lecithin increased	Liver disease, postsplenectomy
Microcyte	Smaller than normal (< 7,2 nm)	Differs according to whether or not: a) well filled with hemoglobin b) normal in shape	See below
Hypochromic cell	Exaggeration of central pallor (> central 1/3rd); usually also microcytic	Failure of hemoglobin due to: a) lack of iron b) defective globin synthesis c) defective porphyrin synthesis	Iron deficiency anemia, anemia of chronic disease Thalassaemia Sideroblastic anemias
Target cell	Hypochromic, with central pigment; thin cell; surface/ volume ratio increased	1. Spleneclomy decreases rate and extent of loss of lipids from reticulocytes 2. Accumulation of both cholesterol and phospholipid on RBC 3. Congenital	As for hypochromic cells. Also 1. Postsplenectomy 2. In liver disease, especially obstructive jaundice
Spherocyte	Spherical, not hypochromic; usually also microcytic; surface/volume ratio decreased, no central pallor	1. RBC membranes abnormality 2. RBC's lose fragment after impact with fibrin strands, walls of diseased vessels and artificial surfaces in circulation	1. Hereditary spherocytosis 2. Acquied irnminohemolytic anemia
Elliptocyte	Elliptical in shape, not hypochromic	1. Hereditary abnormality 2. Acquired alteration	1. Hereditary elliptocytosis 2. In various anemias especially megaloblastic
Sickle cell	Sickle shaped, form assumed under hypoxia (deprivation of oxygen)	Molecular aggregation of HbS	HbS trail of disease. Also seen in some other hemoglobinopathies
Schistocyte	Helmet or triangular shaped, fragmented or greatly distorted RBC; smaller than normal	RBC's lose fragments after impact with fibrin stands, walls of diseased vessels and artificial surfaces in circulation	1. Microangiopathic hemolytic anemia 2. Hemolytic anemia due to physical agents 3. Also in uremia, malignant hypertension
Stomatocyte	Uniconcave, as contrasted with normal biconcave RBC; slit-like instead of circular area of central pallor in RBC	1. Hereditary,primary defects in membrane structure or function resulting in abnormalities of cation permeability, content and flux 2. Acquired alteration in cation content and flux	1. Hereditary stomatocytosis. 2. Smaller number seen in alcoholic cirrhosis, acute alcoholism, obstructive liver disease, malignancies

In the biopsy method a piece of bone marrow is removed intact without disturbing the architecture of the bone. Biopsies are done with a fairy thick, hollow biopsy needle from the iliac crest bone. Tissue from bone marrow fixed. Stained and examined under the microscope.

Biochemical blood analysis

Bilirubin measurement aimed to reveal hemolysis or increased ineffective hematopoesis.

Examination of iron metabolism. Iron is indispensable for hem synthesis. Its daily requirement is 20–25 mg. About 95 per cent of this quantity is received by the organism from hemoglobin of disintegrating erythrocytes and only 5 per cent (some 1 mg) is supplied with food. Iron released during erythrocyte breakdown is utilized for hemoglobin formation in the marrow and deposited in the form of ferritin (in the liver and gastric mucosa) and hemosiderin (in the marrow, liver and spleen). Depot contains 1–1,5 g of iron used up in rapidly changing erythropoiesis rate. The protein transferrin transports iron from the depot and intestine where iron supplied with food is absorbed.

For estimation iron metabolism the next parameters are used: serum iron, unbound iron-binding capacity, serum ferritin, erythrocyte ferritin.

Special test

Evaluation of hemoglobin structure and biosynthesis. 28 % of the red blood cells mass is composed by hemoglobin. The hemoglobin molecule is roughly spherical with a maximum molecular diameter of about 6,4 μm. It is a tetramer, consisting of two pairs of polypeptide chains. To each of the four chains is attached a highly coloured prosthetic group – *hem,* a complex of iron and protoporphyrin The protein portion of the molecule is called globin.

An adult mature red cell has:

– hemoglobin A_1 (α_2 & β_2 chains) – 97 %;

– hemoglobin A_2 (α_2 & δ_2 chains) – 2,5 %;

– hemoglobin F (α_2 & γ_2 chains) – < 1 %.

Hemoglobin is synthesized in erythroblasts and normoblasts of the bone marrow. In degradation of erythrocytes hemoglobin is converted into the bile pigment bilirubin after hem is cleaved. Bilirubin is transported to the intestine with bile where it is transformed into stercobilin and urobilin eliminated in the faeces and urine. About 8 g of hemoglobin, i.e. some 1 per cent of its blood content, is broken down per 24 hours and converted into the bile pigments.

Under pathological condition the abnormal hemoglobin are formed causing the anemias.

In order to detect hemoglobinopathy the next methods are performed: peripheral blood smear, hemoglobin electrophoresis, HbF examination, Heinz bodies detection, X-ray examination of hemoglobin molecule.

Immunological tests for detection hemolytic anemia: direct Coomb's test and Ham's test. Direct Coomb's test is classified as an antiglobulin agglutination test in which the red blood cells clump, or agglutinate after Coomb's serum is added if the red blood cells are covered with IgG. Coomb's serum is anti-IgG (antiserum or antiglobulin) produced in rabbits. Ham's test is based on the ability of a small amount of activated complement to lyse sensitive red blood cells. Lysis of red blood cells determined and compared with normal controls. Normal red blood cells will note lyse under these condition, but complement-sensitive cells will rupture readily.

Myeloproliferative syndrome

Myeloproliferative syndrome refers to a group of diseases of blood system characterized by proliferationof one or more bone marrow blood cell lines. Myeloproliferative syndrome is characteristic of hemoblastosis. The development of a proliferative abnormality can proceed in one direction (for example: myeloid metaplasia – acute myeloblastic leukemia or chronic myeloblastic leukemia) or in several direction simultaneously (for example: polycythemia vera).

Clinical feature include intoxication, necrotic complication, secondary infections, lympfadenopathy, splenohepatomegaly, bleeding. For detection suchabnormality it is necessary to perform clinical blood analysis, the punction of the bone marrow with examination of the different cell lines, and special immunological tests for identification of blood cells.

Syndrome of bleeding disorders

The bleeding disorders are a heterogeneous group of syndromes characterized by easy bruising and spontaneous bleeding from the blood vessels.

Classification:

I. Disorders of coagulation (coagulopathy) – hemophilia.
II. Disorders of platelets (thrombocytopenia) – Werlhoff's disease.
III. Vascular disorders (vasopathy) Henoch-Schoenlein purpura.

The three major components of the hemostatic mechanism are: the platelets, blood vessels, and the plasma protein factors involved in coagulation and fibrinolysis.

The function of platelets in hemostatic process:

- platelet are the instrumental in maintaining the integrity of the endothelial lining of the blood vessels;
- platelet play a major role in repairing any injury in the vascular system, especially at the microcirculatory level;
- platelets take part in regulation of local inflammatory reaction and immune damage initiation;

– platelets are responsible for the specific reaction related to the formation of hemostatic plug.

There are three stages of platelets activation:

– signal transduction from platelet membrane to the structure responsible for the specific reaction;
– platelet adhesion, release of chemical substances of platelet, aggregation and finally formation a plug or clot in vessel damage.

The chemical substances of platelet include a number of enzyme, epinephrine, norepinephrine, ATP and ATP-ase. Many specific compounds participating in clotting of the blood have been revealed in platelets. There are called thrombocytic or platelet factors and are designated by Arabic numerals.

The liquid state of the blood and the closed uninterrupted system of blood vessels in which blood circulates are the principal conditions for body functioning. They are provided by the system of blood coagulation (hemocoagulation system). It keeps blood in a liquid state and restores the wholeness of the pathways of its circulation by formation of blood thrombi (plugs, clots) in the damaged vessels.

The coagulation blood system comprises blood and tissues which produce, utilize and secrete substances from the body that are indispensable for the process of coagulation. The neurohumoral apparatus also belongs to this system.

The coagulation of blood is the process of clotting of whole blood, which results in the formation of a fibrin clot. Three processes are involved in blood clotting such as formation of prothrombinase, thrombin and fibrin. In addition, the phase preceding and the phase following blood coagulation are distinguished. The primary phase is accompanied by vascular thrombocytic hemostasis (i.e. processes involved in stoppage of bleeding) in which bleeding from the microcirculatory vessels with low blood pressure is arrested. This process is also known as microcirculatory hemostasis. In the second phase two processes simultaneously occur, i.e. retraction and fibrinolysis of the blood clot. Thus, the process of hemostasis involves three components: vessel walls, formed blood elements, and enzymatic plasma system of blood clotting.

Clinical features

The patients complain on the appearance on the skin spontaneous bruising. There may also oral, nasal, gastrointestinal or genitourinary bleeding.

Objective examination. There are different types of skin bleeding disorders: petechial, purpura, ecchymosis, hematoma.

Hemarthrosis – hemorrhage into a joint cavity observed in patients with severe coagulation disorders, such as hemophilia. Hemarthrosis is characterized by painful, deformed joints. Hematuria is characteristic hemophilia and anticoagulant

therapy. Blood in stool arises usually when is gastric or intestinal bleeding. Excess vaginal bleeding may be in women with thrombocytopenia.

Additional methods of examination

Tests for vascular – platelet factors. Tests for platelet factors include the quantitative platelet count, its morphology, platelet aggregation and adhesiveness tests, bleeding time test, estimation of platelet components in plasma.

Platelet aggregation test. An aggregating agent (activated thrombin, epinephrine, ADP, and collagen) is added to a suspension of platelet rich plasma and the response is measured in a spectrophotometer. Special devices called aggregometers are used to measure platelet aggregation.

Platelet adhesiveness test measures the ability of cells to adhere to glass surface. Adhesiveness can be determined by counting the number of platelet in the anticoagulated blood before they are passed through the column with glass beads, and by counting them again after they have passed through the column.

Bleeding time test measures time required for the cessation of bleeding after a standardized puncture through the skin 3 mm deep. The Duke test involves puncturing the earlode with a lancet, drops of blood are blotted every 30 second and the time at which bleeding stops is noted. Normal times for the Duke test are 1 to 3 minutes. The Ivy test have similar procedure but added a blood pressure cuff, which is placed on the upper arm and inflated to 40 mm Hg the skin is pieced with a lancet in the lower forearm. Blood is blotted every 30 second until the bleeding stops. Normal times for the Ivy test are between 2 and 6 minutes.

Tests for plasma factors involved in coagulation and fibrinolisis

Prothrombin time (PT) measures the extrinsic system (factor VII) as well as factors common in both systems (factor X, V, II and I). Prothrombin time test is performed by adding tissue extract (factor III – tissue factor) and calcium to the plasma. Normal prothrombin time – 10–17 second.

Activated partial thromboplastin time (APTT or PTTK) measures the intrinsic system's factors VIII, IX, XI and XII, in addition to factors common to both systems. Three substances – phospholipid, a surface activator (Kaolin) and calcium are added to the plasma. The normal PTTK is 30–40 seconds.

Fibrinogen determination test is performed by addition 0,2 ml thromboplastin and 0,1–0,5 % solution of calcium chloride to 1 ml platelet-rich plasma. Formed clot is dried and weighed. The normal fibrinogen levels in the blood are 200 to 400 mg per deciliter of plasma.

Thrombin time or fibrinogen deficiency test is performed by added the activated thrombin to blood plasma and measure the time in takes to form a clot. The test reflects fibrinogen-fibrin conversion. Normal thrombin time – 10–12 second.

Iron deficiency anemia

Iron deficiency anemia – clinical and hematological disordes due to the iron deficiency in organism: blood, bone marrow and iron stores resulted to the disturbances of hem and iron containing enzymes production.

Etiology

Blood loss

Uterine (menorrhagia, metrorrhagia).

Chronic gastrointestinal blood loss:

- esophageal varices;
- hiatus hernia;
- peptic ulcer;
- chronic aspirin ingestion;
- carcinoma of stomach, colon, caecum, rectum;
- ulcerative colitis;
- hemorrhoids;
- diverticulosis;
- hookworm infestation (anemia with eosinophilia).

Urine bladder and kidney:

- glomerulonephritis;
- carcinoma of kidney and urine bladder.

Increased requirements:

- prematurity (diminished iron stores);
- growth (infants and young children);
- females in reproductive age group: menstruation, pregnancy, lactation.

Impaired absorption:

- achlorhydria (especially in middle aged females);
- atrophic gastritis;
- gastrectomy;
- gastroenterostomy;
- tropical sprue or coeliac disease.

Inadequate intake:

- improper feeding in infants and young children;
- poverty;
- dietary fads;
- anorexia (nervosa, of pregnancy or malignancies).

Pathogenesis

The amounts of iron in organism are distributed between active iron pools (hemoglobin and tissue enzymes) and iron stores (ferritin and hemociderin). Iron

deficiency develops when iron loss more than iron intake with food or when increased natural iron requirements or impaired iron absorption – appears negative iron balance between iron intake and iron stores that causes decreased iron supply to the bone marrow and iron-deficient erythropoiesis. The several stages of iron deficiency are distinguished: iron-store depletion, iron-deficiency erythropoiesis, iron-deficiency anemia.

The pathogenesis of clinical features may be explained by decreased amount of iron resulted by insufficient tissue supply with oxygen. Reduction in oxygen carrying capacity leads to tissue hypoxia symptoms referable to systems with high oxygen requirements, such as skeletal musculature, cardiovascular system and central nervous system are particularly prominent. Specific action of iron deficiency on the activity of hem content enzymes leads to the trophic disturbance of tissue.

Clinical features

The clinical features include two syndromes: general anemic and specific one due to the iron lack.

Anemic syndrome. The patients complaint on fatigue, tiredness, faintness, easy fatigability, dyspnea, palpitation, heart pain, headache, giddiness, spots before the eyes, lack of concentration, drowsiness, numbness, coldness, tingling of hands and feets. Mild fever 37,2–38,2 °C is observed. Physical examination of the cardiovascular system reveals the displacement of the left relative cardiac border outside, diminished first sound, functional systolic murmurs over sound points with maximal intensity over pulmonary artery, systolic bruits over carotic arteries. ECG changes occur – ST-segment depression and flattening or inversion of T-wave. Amenorrhoea, menorrhagia in females and loss of libido in the males appear.

Cideropenic syndrome was first described by Basenstrom in 1930. The patient complains on the generalized muscular weakness, disorders of muscular sphincters and disorders of urination.

The colour of the skin is pallor with greenish tint. Pallor of the nail beds, mucous membranes of the mouth, conjunctivae, sclerae are revealed. The skin is dry with creak (chirp rattle) on the legs and hands leukoplakia. The nail beds became dry, fragile, with sketch, spoon-shaped named koilonychias. The hair became thin, fragile, and grey.

The gastrointestinal disorders are the specific symptoms and signs of iron deficiency anemia. The patients complain on the difficulties during swallowing solid food – Plummer-Vinson syndrome according to the atrophy of the postcricoid esophageal web. In chronic, severe iron deficiency the patients have specific features – pica chlorotica, which characterized by the eating of unusual items such as coal, earth, chalk, clay, starch, ice (pagophagia) and smell acetone, petroleum.

Nausea, regurgitation, pain and dulling at the epigastric region after meal, diarrhea, anorexia are the specific symptoms of the patients with iron deficiency anemia. The clinical signs of anemia – glossitis with redness and papillae atrophy, angular stomatitis, inflammation of the gum, cheilosis.

During the endoscopic investigations and biopsy the atrophy esophagitis and gastritis are detected. Sometimes may be splenomegaly.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration is decreased;
- red blood cells count decreased normal or slightly decreased;
- mean cell volume (MCV) < 76 fl;
- mean cell hemoglobin (MCH) < 27 pg;
- mean cell hemoglobin concentration (MCHC) < 30 gm%;
- color index < 0,8;
- anisocytosis, microcytic red cells;
- poikilocytosis, pencil shaped cells and target cells;
- hypochromia, ring or pessary cells;
- few polychromatophils;
- reticulocyte count is variable;
- red blood cells osmotic fragility is slightly decreased;
- hematocrit low.

Bone marrow:

- micronormoblaslic erythroid hyperplasia;
- predominantly intermediate normoblasts;
- cytoplasm decreased and shows differential staining;
- bone marrow iron is reduced or absent.

Biochemical blood analysis:

- serum iron level is reduced;
- total iron binding capacity is increased;
- unsaturated iron binding capacity is also raised;
- percentage saturation reduced.

Megaloblastic anemia

Megaloblastic anemia is blood disorder which characterized by abnormalities in the DNA synthesis of the blast cells due to the deficiency of vitamin B_{12} and/or folic acid.

Etiology of vitamin B_{12}-deficiency anemia

1. Reduced intake: nutritional deficiency.
2. Strict veganism.
3. Impaired absorption:
   - gastric cause: total or partial gastrectomy;
   - intestinal cause: chronic tropical sprue, intestinal stagnant loop syndrome (e.g. jejunal diverticulosis, blind loop, strictures), scleroderma, Crohn's disease and ileal resection, congenital selective malabsorption with proteinuria, Zollinger Ellison syndrome, severe pancreatitis, coeliac disease;
   - hemodialysis;
   - transport protein defects: hereditary lack of transcobalamin II, abnormal transcobalamin II, abnormal B_{12} binding protein.
4. Competition for cobalamin:
   - bacterial colonization of the small intenstine;
   - fish tapeworm infection;
   - bacteria "blind loop" syndrome.
5. Impaired metabolism:
   - inhibitors of dihydrofolate reductase;
   - purine antagonists;
   - pyrimidine antagonists;
   - alcohol.

Etiology of folic acid deficiency anemia

Reduced intake:
- poor diet;
- alcohol;
- liver disease;
- malabsorption.

Increased requirements:
- pregnancy;
- childhood and adolescence;
- neoplastic disease.

Drugs:
- cytotoxic drugs;
- contraceptive pill.

Pathogenesis

Methylcolabamin is an essential cofactor in the conversion folic acid to its active form. When this reaction is impaired folate metabolism is deranged and occurs

defects in DNA synthesis with megaloblastic maturation patterns in patients who are deficient to colabamin.

The lack of vitamin B_{12} lead to the biochemical disorder such as conversion of homocysteine to methionine which takes part in production of phospholipids required for myelin formation. This biochemical abnormality may contribute to the neurological complication of cobalamine deficiency.

The jaundice may be explained by the excess breakdown of hemoglobin from immature erythroid bone marrow which easily damaged than normal erythrocytes and hence have a shortened life span.

Clinical features

There are three clinical syndromes: anemic, affection of the digestive system and neurological syndrome.

Anemic syndrome includes such complaints: fatigue, tiredness, palpitation, dyspnea, giddiness. The skin is pallor with lemon yellow tint, slightly icteric skin and sclerae, swallowing face, slight pedal edema. Physical examination of the cardiovascular system reveals tachycardia, systolic murmur at the apex and pulmonary artery, systolic bruits over carotid arteries, ischemic changes on ECG, heart failure. The symptoms and signs of gastrointestinal affection: anorexia, Hunter's glossitis (sore, smooth red tongue, with ulcer over the edge), atrophic gastritis, bladder and bowel dysfunction, diarrhea, enlarged liver and sometimes spleen. Neurological syndrome includes peripheral neuropathy and combined degeneration of the spinal cord where the posterior and lateral columns undergo demyelization. The symptoms and signs are next: numbness, tingling, paresthesia in the extremities, difficulty in walking, ataxia, position and vibration senses are diminished, clumbness. There may be sphincter disturbance. Reflexes may be diminished or increased. The Romberg and Babinski signs may be positive. Affections of the mental state reflect irritability, diminished memory, even severe dementia or psychosis. In young females there may be infertility.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration decreased moderately;
- red blood cell count decreased pronouncly;
- mean cell volume ranging from 100 to 140 fl;
- color index > 1,2;
- moderate leucopenia;
- mild, usually asymptomatic thrombocytopenia;
- anisocytosis – macrocytosis;

– poikilocytosis – ovalocytosis;
– hyperchromia, ring or pessary cells;
– red blood cells may show: Howel-Jolly bodies, Cabot rings;
– hypersegmentes neutrophils;
– macropolycytes (large neutrophils).

Bone marrow:

– hyperplasia of erythroid elements;
– megaloblasts – gigantic cells with large nucleus oval shape and basophilic cytoplasm;
– gigantic metamyolocytes;
– megakaryocytes.

Biochemical blood analysis:

– increased level of unconjugated bilirubin;
– increased level faeces stercobilin;
– increased level of lactatdehydrogenasa.

Special tests for diagnosing viamin. B_{12} deficiency:

– low serum vitamin B_{12} assay;
– increased urinary excretion of methylmalonic acid;
– low radioactive vitamin B_{12} absorption test (Schilling's test);
– reticulocyte response to vitamin B_{12} administration.

Hemolytic anemias

Hemolytic anemias are the geterogenous group of anemias which characterized by shortened life span of erythrocytes in the circulation resulting from their accelerated destruction.

Classification of hemolytic anemias

Hereditary hemolytic anemias

Defects of the cell membrane:
– hereditary spherocytic anemia;
– hereditary elliptocytic anemia.

Defects of erythrocytic metabolism:
– glucose-6-phosphate dehydrogenase (G-6-PD) deficiency anemia.

Abnormal hemoglobins:
– sickle cell anemia;
– thalassemia.

Acquired hemolytic anemia

Immunological destruction of red blood cells:
– transfusion with incompatible blood;

– hemolytic disease of the newborn;
– autoimmune hemolytic anemia (AIHA) (warm-active AIHA and cold-active AIHA).

Physical destruction of red blood cells:
– march hemoglobinuria;
– traumatic cardiac hemolytic anemia.

Hemoyitic anemia induced by chemical agents.

Hemolytic anemia caused by microorganism:
– anemia of malaria;
– anemia of clostridia.

Hemolytic anemia secondary to other disease.

Paroxysmal nocturnal hemoglobinuria.

Etiology

The causes of hemolytic anemias may be hereditary or acquired. The causes of hereditary hemolytic anemia are grouped into three categories: 1) defect of the cell membrane; 2) defects of erythrocyte metabolism; 3) abnormal hemoglobins.

Pathogenesis

Acquired hemolytic anemias have numerous causes hence corresponds with different pathogenesis. Destruction of red blood cells refers to inappropriate activation of the body's immune system and appearance either alloantibodies or autoantibodies. A number ingestion of drugs and chemicals may result to shortened life span of erythrocytes. Inflammation of blood vessels or presence of blood clots may interfere the structure and function of red blood cells and lead to early destruction. Such physical factors as vascular protheses, heart valves protheses cause accelerated hemolysis of red blood cells. Some infectious agents for example malaria parasite (Plasmodium falciparum, Clostridiax) use red blood cells for their propagation and this process destroy them. Hemolytic anemia could develop as a secondary effect of certain clinical condition such vitamin B_{12}-deficiency anemia, splenomegalia, liver disease and renal failure.

Hemolysis may occur intravascularly or extravascularly. Hemoglobin liberated into the plasma is bound mainly by the alpha-2 globin, haptoglobin, to form a complex too large to be lost in the urine. It is taken up by the liver and degraded. Some hemoglobin is partially degraded and bound to albumin to form methemoglobin. If all the haptoglobin has been consumed, free hemoglobin may be lost in the urine. In small amounts this is reabsorbed by the renal tubules where the hemoglobin is degraded and the iron stored as hemosiderin. Sloughing of the renal tubular cells gives rise to hemosiderinuria which, if found, always indicates intravascular hemo-

lysis. Hemoglobinuria occurs when greater amounts of hemoglobin are lost, giving the urine a black appearance (black water).

Extravascular hemolysis occurs in the phagocytic cells of the spleen, liver, bone marrow and other organs and there may be little or no depletion of haptoglobin.

Clinical features

Clinical features include three indications: anemia, jaundice and splenomegalia. The symptoms of anemia are common as most other one: weakness, fatigue, dispnea, palpation, headache, dizziness, inability to concentrate. The most important sign of hemolytic anemia is jaundice, which vary from slightly yellow tint to intense lemon color of mucosa membrane, sclera and skin. Splenomegaly is specific sign, explained by hyperplasia of cells which take part in phagocytosis. Commonly spleen is enlarged moderately.

Latent compensated hemolytic anemia explained by capacity of bone marrow to produce increased number of reticulocytes and in the peripheral circulation red blood cell counts may be fairly normal.

However the bone marrow will no longer be able to compensate and breakdown rate of erythrocytes becomes greater than the production rate of new erythrocytes. In acute cases is developed the hemolytic crisis with abrupt onset, high temperature, severe fatigue, nausea, vomiting pain in the abdomen, pronounced pallor with yellow color of mucosa and skin, hemorrhage lesions. Patient has grave condition, may be occur hemolytic coma. Tachycardia, systolic murmur, hypotension are observed. During palpation of abdomen the hepatosplenomegalia is detected.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration decreased;
- red blood cells count decreased;
- reticulocytes increased;
- macrocytosis;
- polychromasia;
- polymorphonuclear.

Bone marrow:

- compensatory erythroid hyperplasia.

Biochemical blood analysis:

- increased plasma unconjugated bilirubin;
- increased urinary urobilinogen;
- increased faecal urobilinogen;
- increased plasma lactatdehydrogenasa.

Findings of intravascular hemolysis:

- reduced or absence of haptoglobin in the blood;
- presence of free hemoglobin in the blood;
- presence of free hemoglobin in the urine;
- presence of methemalbumemia.

Special test for determining red blood cell life span using the ^{51}Cr.

Hereditary spherocytic anemia

This is an inherited disorder that is transmitted as an autosomal dominant trait.

Pathogenesis

The three main mechanisms causing this disease are discussed. In the maintenance of normal red blood cell shape number of important structural proteins such as actin, spectrin and ankyrin play a major role. People suffering from spherocytic anemia have abnormal mutant gene for the protein spectrin, their lack weakens the structure of the red blood cell membrane. Next mechanism related to abnormally permeability to sodium and sodium pumps seem not to be able to function properly. This will upset the osmotic equilibrium and lead to an influx of water into the cell, which becomes more spherical.

Spherocytes are rigid cells and hence they can not pass through slit like openings of splenic cords and sinuses and may remain there for over 10 hours, hypoxia is created which compromises red cell metabolism consequently leading to loss of red cell membrane, this causes further sphering and rigidity. Further conditioning initiates phagocylosis by reticuloendothelial cells in spleen.

Clinical feature

The disease can present at any age, but most patients present in the first decade of life. Onset may be with anemia, accompanied by general anemic symptoms. Anemia is fairly mild, because the reduced life span of the red blood cells is compensated by an increased erhytrocyte production in bone marrow. Symptoms can vary to severe anemia. Episodic jaundice may be noted. The severity of the disorder tends to hemolytic crisis at times often precipitated by infections or due to no obvious cause. The spleen is often palpably enlarged slight to moderate, firm, non tender. Abdominal pain due to the hemolytic crises or splenic infarction is observed. Gallstones with calcium bilirubinate are common which may result in biliary obstruction and the pain.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration decreased;
- red blood cells count decreased;

– platelets decreased;
– reticulocytes increased sharp (from 5 to 20 %);
– red blood cells and platelets count return to normal within 7–10 days;
– MCV normal or reduced;
– MCH normal;
– MCHC often increased (34–40 %);
– spherocytes;
– microspherocytes (microcytes without central pallor);
– polychromatophils.

Bone marrow:

– normoblastemia.

Biochemical blood analysis:

– increased plasma unconjugated bilirubin.

Special tests:

– the Coomb's test is negative;
– osmotic fragility of erhytrocytes are raised;
– ^{51}Cr red cell life span reduced with excessive counting over spleen.

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency anemia

This hemolytic anemia has hereditary origin due to the enzyme deficiencies involving in red blood cell metabolism.

Etiology

Deficiency of G-6-PD is an inherited disorder with sex linked transmittion with intermediate dominance because gene for G-6-PD is located on the X-chromosome. In males the presence of a G-6-PD deficient gene will result in the absence of any normal G-6-PD and as result in an accelerated hemolysis of the red blood cells. Heterozygous deficiency female with one normal and one abnormal gene has red blood cells that are either normal or lack the active G-6-PD enzyme. Overt signs of anemia are rare in heterozygous deficient females.

There are agents who may lead to hemolytic anemia in G-6-PD deficiency: antimalarials, analgesics, antibacterial and miscellaneous. There is phenomenon of favism. Ingestion of the fava bean or even inhalation of fava pollen may result in a severe hemolytic crisis in a small portion of people carrying the Gd Mediterranean gene. It is found mainly in populations that border the Mediterranean Sea such as Sicilians, Sardinians, Greeks, Turks, Lebanese, Sephardic Jews and Arabs.

Pathogenesis

In normal individuals the level of oxidant activity and hence the level of sulfhemoglobin is carefully controlled by the compound known as reduced glutathione which neutralizes the activity of the oxidant drugs or oxidant products of infections with the aid of NADPH.

The NADPH needed for reaction is provided by the hexose monophosphate shunt via the enzyme G-6-PD. Persons with lack G-6-PD have an abnormal form this enzyme will unable to generate sufficient amount of NADPH. As a consequence there will be a steady increase in the amount of oxidant in the red blood cells, resulting in the formation of sulfhemoglobin, which will precipitate out as Heinz bodes. In situation in which the body is suddenly exposed to large amount of oxidants people with abnormal or deficient G-6-PD be unable to eliminate these toxic compounds, resulting in a rapid outbreak of hemolytic anemia.

Clinical features

The disease is characterized by acute onset associated with acute illness or infection, by taking of an offending drug or chemical, or the ingestion of fava beans. Acute hemolytic episode is observed with pallor, jaundice, abdominal pain and dark urine. Despite continuous drug intake, the hemolytic process ends spontaneously after approximately one week.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration decreased during the acute phase;
- polychromasia;
- reticulocytosis;
- basophilic stippling;
- fragmented red blood cells;
- Heinz bodies.

Screening tests

Tests demonstrate the presence/absence of G-6-PD by testing the ability of cells to generate NADPH from NADP, a reaction direct by dependent upon G-PD availability:

- brilliantcresyl blue reduction test;
- methemoglobin reduction test;
- Heinz body test;
- fluorescence spot test;
- acrobat-cyanid test;
- assays of G-6-PD activity.

Thalassemias

Thalassemias syndrome are a heterogeneous and complex group of inherited diseases resulted of abnormal hemoglobin synthesis.

Etiology

The thalassemia trait occurs with high frequency in certain population. The term thalassemia comes from the Greek words "thalas", meaning sea, and "emia", which stands for blood. Thalassemia is most prevalent in populations that border the Mediterranean sea in the Far East, in certain African population as well as in Afro-Americans black.

Pathogenesis

Normal adult hemoglobin contains two α- and β-chains. In the thalassemias production of one of these globin chains is deficient. Consequently less than the normal amount of adult hemoglobin is produced and occur anemia. Thalassemia may be divided into two major groups – according to the globin chain that is deficient – into α-thalassemia and β-thalassemia.

α-thalassemia

In this group of diseases the deficiency is in the synthesis of α-globin. The β-globins are not affected and are produced at their normal rate. Since people normally have four genes for α-globins, the following four different patterns of α-thalassemia can occur: one-gene deletion – silent carrier; two-gene deletion – α-thalassemia trait; three-gene deletion – HbH disease; four-gene deletion – failure of HbA and HbF synthesis.

Clinical features

Clinical features depend on molecular variant of inherited forms. If one gene is deleted there is no clinical effect. The α-thalassemia trait is also asymptomatic as hemoglobin levels usually reach 10 to 12 g/dl. In patient may be a mild hypochromic anemia. Hemoglobin HbH disease, also known as α-thalassemia proper. People with this disorder will have from 5 to 40 % of HbH in their blood, which tends to precipitate with formation of Heinz bodies. Such red blood cells are then catched by the mononuclear phagocytic cells of the spleen and shortened life span of these red blood cells. Hemoglobin levels are usually between 7 and 11 g/dl ranging from mild to severe anemia. The clinical features include skeletal changes in these patients due to the hyperactivity of bone marrow, leg ulcers, icterus and splenomegalia. Anemia frequently worsens during pregnancy and infectious disease.

In case of four α-globin genes deletion followed complete failure of HbA and HbF synthesis. Total absence α-globin leads to death either in utero or very soon after birth due to hypoxia. This phenomenon is known as hydrops fetalis.

Additional methods of examination

Clinical blood analysis:

- hemoglobin concentration decreased corresponds to type thalassemia. The total red blood cell count may be fairly normal due to a moderate increase in reticulocytes.
- microcytic hypochromic cells;
- target cells.

Special tests:

Electrophoresis of the blood hemoglobin allow to detect the presence of HbH.

β-thalassemia

β-thalassemia are characterized by a deficiency in β-globin chain synthesis.

Etiology and pathogenesis

Etiology and pathogenesis β-thalassemia are caused by mutations affecting the functional capacity of mRNA and normal transcription of β-globin genes.

There are the some forms of β-thalassemia: β-thalassemia major, homozygous (or β_0-thalassemia) with complete suppression of HbA; β-thalassemia inter media, β^+-thalassemia with incomplete suppression HbA (10–20 %); β-thalassemia minor, heterozygous (Hb – 90–95 %).

Clinical feature

β-thalassemia major appears in first year of life with insidious onset and development of severe chronic anemia. In the absence of transfusion therapy, hemoglobin falls to 3 to 5 gm%. From marrow expansion and cortical thinning are observed skeletal changes in hands and feed, the metacarpals, and phalanges, which become rectangular and frankly convex shaped. Stature is shortened, head is large and abdomen protrudes. In the long bones widening of medullary portions predisposing to pathologic fractures. Compression factures of vertebrae may occur. The facial manifestations of β-thalassemia are specific. Thickening of the cranial bone produces frontal bossing. Prominence of the check bones lend to obscure base of the nose and expose the upper teeth. Growth and mental retardation in early childhood occurs due to anemia. Because these children receive regular blood transfusion excess iron is deposited in many vital organs such as heart, liver, the islets of Langerhans, the gonads causing numerous complications. More important is myocardial hemo-

siderosis with arrhythmias and leading effect – heart failure, which cause death in transfused patients with thalassemia. Hepatomegaly is due to myeloid metaplasia but later results from iron deposits suggested microscopically hemochromatosis. One of the major clinical features of β-thalassemia is splenomegaly. In children are observed the endocrine disorders: diabetes mellitus, failure of pituitary or hypothalamic maturation, puberty is delayed.

Additional methods of examination

Clinical blood analysis:

- erythrocytes count decreased;
- platelet count normal but decreased;
- leucocytosis;
- reticulocytosis (10 %);
- hemoglobin level below normal;
- MCV, MCH, MCHC are diminished;
- marked microcytosis with hypochromia;
- numerous Heinz bodies;
- target cells;
- normoblasts;.
- granular inclusions in cytoplasm.

Bone marrow:

- erythroid hyperplasia;
- normoblasts.

Special test:

- osmotic fragility of erythrocytes is decreased;
- hemolysis – serum bilirubin elevated.

β-thalassemia minor

The individual has at least one normal functioning β-globin gene, which produces sufficient quantities of HbA. Thus heterozygous state prevents severe symptoms of anemia. The patient clinically mild with little or no anemia lives a normal life. Seldom may the disease manifestate with icterus, splenomegaly, by ulcers or radiographic changes of long bones. Sometimes pathology is detected occasionally during routine examination.

Additional methods of examination

Clinical blood analysis:

- erythrocytes count decreased;
- hemoglobin level slightly below normal or normal;

– MCV and MCH are reduced;
– MCHC is normal;
– microcytic hypochromic cells;
– target cells;
– stippled erythrocytes.

Bone marrow is normal except for mild erythroid hyperplasia.

Biochemical blood analysis:

– serum bilirubin may be slightly elevated.

Special tests:

– osmotic fragility of erythrocytes is decreased;
– erythrocytes life slightly shortened.

Sickle cell anemia

Sickle cell anemia – chronic hemolytic anemia, which is characterized by sickling phenomenon of red blood cells, causing by inheritance of pathological gene, determined the synthesis of abnormal HbS.

Etiology

Etiology sickle cell anemia is inherited as an autosomal codominant trait. In patient may be genetic defect: one normal and one abnormal gene – heterozygous, both abnormal genes – homozygous. People who are heterozygous for HbS normally produce more than 50 % of their hemoglobin in the form of HbA. Only under condition of extreme stress, such as severe hypoxia or severe infections, does some degree of sickling occur.

Pathogenesis

There are different hypotheses regarding the molecular events of relationships between structure of HbS and formation of sickled cells. As the red blood cells sickle, they lose their flexibility and become rigid and in this form they may obstruct the capillary flow, then leads to local tissue hypoxia.

Clinical features

Clinical features vary according to the inheritance type. In cases of homozygous sickle cell disease the clinical syndrome include specific phenotype: phenomenon of vessels obstruction and hemolytic anemia. The onset of disease usually begin at age less 2 years, because from birth to 6 month child is protected by HbF, which later replaced with HbS. Sickling of red blood cells may produce infection in many vital organ. This would impair normal growth and development of children. They have bossing of the skull, prominent malar bones and protuberant teeth, remain small and

sickly. The specific syndrome of disease is painful crisis due to the vascular obstructions with consequence of disorders of the local microcirculation by intravascular sickling which will produce local tissue injury. Painful crisis occurs periodically after local infections, childbirth surgery, change of altitude, extreme exercise, and similar situation that result in sudden deoxygenation. These tissue infarcts may occur in a vide variety of organs including lung, heart, kidneys, spleen, brain. After about 3 year of age slugging of blood occurs in the larger bones of the extremities the spin, rib cage and periarticular structures, producing bone and joint crises. Pulmonary crises characterized by fever, tachypnoe, chest pain, pulmonary infiltrates, lung embolisms. Myocardial infection, associated with sickling may result in congestive heart failure. Abdominal crises are attributable to small infarcts of mesentry and abdominal viscera and are characterized by severe abdominal pain of peritoneal irritation. Sometimes impairment to the liver may result in the formation of gallstones in the gallbladder. Splenic sequestration leads to the splenomegaly evident by 6^{th} month. Impairments to the kidneys may appear as symptoms of glomerylonephritis wits high levels of hematuria. Damage may also occur to the skin with chronic ulcers. Central nervous crises occur in children and young adults. There is sudden occlusion of cerebral vessels with hemiparesis, aphasia, sensory deficits. Intracerebral and subarachnoid hemorrhage may result from hypoxia necrosis of vessels that lead even to death. Hemolytic crises occur suddenly following by oxidant drugs and infections with common signs conjuctival, skin icterus and pallor. Patient with homozygous sickle cell anemia may also experience occasional hypoplastic crises, associated with or follows a febrile illness. The combination of accelerated hemolysis and temporary arrest of erythropoiesis may result in severe life-threatening anemia.

Additional methods of examination

Clinical blood analysis:

- low concentration of hemoglobin: 6 to 9 g/dl;
- MCV and MCH are normal;
- many sickled cells;
- Howell-Jolly bodies;
- microcytosis.

Biochemical blood analysis:

- increased plasma unconjucted bilirubin;
- in hemolytic crises, haptodlobin level is low.

Hemoglobin electrophoresis:

- presence of large amount of HbS (80 to 95 %);
- considerable amount of HbF (5–15 %);
- total absence of HbA.

X-ray examination of bones:

- osteoporosis;
- periosteal thickening;
- osteosclerosis of long bones;
- aseptic femoral head necrosis.

Hemoblastosis

Hemoblastosis is a disease of the whole blood system characterized by:

1) progressive cell hyperplasia in the hemopoietic organs with pronounced prevalence of proliferation of certain cells;
2) metaplasia of these pathological cells instead of normal cells to hemopoietic organs;
3) development of pathological foci of hemopoiesis in various organs.

Classification of hemoblastosis depicted at table 5.2.

Table 5.2. ***Classification of hemoblastosis***

Primary affection of the bone marrow		Tumor growth outsides the marrow bone
Myeloproliferative disorders	Lymphoproliferative disorders	Hodgkin's disease Malignant lymphoma Reticulosarcoma Limphosarcoma
Acute myeloblastic leukemia Chronic myelocytic leukemia Polycythemia vera Myelofibrosis leukemia	Acute lymphoblastic leukemia Chronic lymphocytic leukemia Multiple myeloma	

Acute myeloblastic leukemia

Acute myeloblastic leukemia is characterized by profuse proliferation of the blast element of blood with their subsequent disturbed differentiation, with development of foci pathological hemopoiesis in various organs.

Acute myeloblastic leukemia occurs in all age groups but commonly in adults and less in children. Acute myeloblastic leukemia is predominantly a disease of adults with two peaks, one at 15 to 20 years of age and another peak after 50 years of age.

Etiology

The reason of the appearance of leukemia is still unknown. Most authors regard hemoblastosis as tumors whose morphological basis are hemopoietic cells of various organs.

Some factors can provoke acute myeloblastic leukemia:

1) chemical concerogenous substances (benzpyrence, benzol);
2) radiaionizing;
3) viruses theory connect the appearance of acute myeloblastic leukemia with DNA or RNA damage, but only animal experimental studies support this point of view;
4) genetic theory: according to this theory, acute leukemia develops due to the congenital or acquired damage to the chromosome structures of low differentiated cells of the hemopoietic organs.

Pathogenesis

A clone theory has been adopted recently, according to which hemoblastosis arises due to primary mutation in one of the hemopoietic cells with its subsequent multiplication and formation of a clone of blast cells.

These cells fail to differentiate properly and proliferate without maturing to the normal nonproliferating stages. The accumulation of this immature, continually dividing cells results to the replacement of the normal hematopoietic precursor cells by these neoplastic cells. This will cause complete bone marrow failure. Expansion and infiltration of tissue and organs with abnormal white blood cells lead to the main clinical features.

Classification

French-American-British Group (FAB)

M1 – undifferentiated
M2 – differentiated
M3 – promyelocytic
M4 – myelomonocytic
M5 – monocytic
M6 – erythroleukemia
M7 – megakaryoblastic

Clinical features

The onset of the disease is in most cases acute or subacute. In some cases the onset disease is gradually with non specific general symptoms: weakness, fatigue, subfebrile temperature, weight loss.

There are some syndromes of acute leukemia: intoxication, ulcerative necrotic, infections, bleeding, anemia, splenomegaly and hepatomegaly, bone pain, neurological syndrome:

– syndrome of intoxication are as follows: high temperature (remittent or hectic), profuse sweating, chills, pronounced weakness, reduced exercise toler-

ance, general loss of strength. Fever chills and sweating are explained by the pyrogenic effects of purenes released in great quantity during the decomposition of immature leucocytes;

- ulcerous – necrotic syndrome is characterized pain in the throat, swallowing becomes painful. Ulcerous of the oral mucosa occurs commonly in acute myeloblastic leukemia. There may be infiltration of the gums with swelling and bleeding. Ulcerous and necrotic tonsillitis, gingivitis and stomatitis are quite characteristic of this disease.

Despite the markedly increased production of white blood cells, their function is inadequate. This fact is explained such complications as secondary infections due to the pathogenetic agents: gram negative bacteria, staphylococci and streptococci, viral – herpes simplex and zoster, fungal – Candida, protozoal – pneumocystitis carinii.

The infections of skin, mouth, throat, respiratory and urinary tract including septicemia are common usually. Pericarditis and pleuritis are possible.

Bleeding syndrome: traces of subcutaneous and intracutaneuos hemorrhages can be seen. The lesions vary in size from small pointed hemorrhages (petechiae) to large black and blue spots (ecchymosed) which appear spontaneously or at points of injections. Spontaneous bruises, purpura, bleeding gums and bleeding from venepuncture sites because of thrombocytopenia are common. Occasionally there may be major internal hemorrhage.

Syndrome of anemia is explained by depressed erythropoiesis; increased bleeding; accelerated destruction of red blood cells. The degree of anemia depends of the speed of the development of acute myeloblastic leukemia as well as on iron and folic acid stores. There are general symptoms of anemia: fatigue, dizziness and dyspnea. The mucous membranes and nail beds are pallor.

Lymphadenopathy is rare sign in patients with acute myeloblastic leukemia. Sometimes enlarged cervical and supraclavicular lymph nodes are detected in the superficial areas by palpation. Tenderness of the sternum may be quite pronounced. Blast infiltration of meningeal membranes lead to neurological disorders with clinical features of meningitis.

Additional methods of examination

Clinical blood analysis:

- leucocytosis 30–300x10^9/l;
- the white blood cells count may be normal or even decreased;
- red blood cells count decreased;
- hemoglobin concentration decreased;
- severe thrombocytopenia;
- blast cells – 95 %.

– blood film examination show variable numbers of blast cells. In patients with acute myeloblastic leukemia the blast cells are the myeloblasts or erythroblasts. The blasts may show Auer rods and other abnormal cells may be present: promyelocytes, myelocytes, agranular neutrophils;
– absence of eoisinophyls and basophils.

Bone marrow:

– bone marrow is hypercellular with a marked proliferation of blast cells which typically amount to over 75 % of the marrow cell total.

Chronic myelocytic leukemia

Chronic myelocytic leukemia – is defined as the myeloproliferative disorders. Chronic myelocytic leukemia is as a neopastic disease of bone marrow stem cell – precursor of myelopoiesis, which is common for granulocytes, erythrocytes and megakariocytes with excessive production of granulocytes in the bone marrow and other hematopoietic organs.

Etiology

The specific factors haven't been established. The causative agents of chronic myelocytic leukemia are suspected such as ionizing radiation, exposure some chemical carcinogens, which damage bone marrow.

Pathogenesis

Excess radiation or mutagenic chemicals cause mutation in the bone marrow stem cell, damage of DNA, and transformation it in neoplastic cell with chromosomal disorders. This cell produces clones of changed bone marrow stem cells with abnormal chromosome, known as Philadelphia chromosome, resulting from the translocation of chromosome 22 to chromosome 9. This chromosomal abnormality leads to the formation new onkogene bcr/abl which processes an increased enzyme activity. Clones of bone marrow stem cells develop an ability to proliferate excessively, especially the granulocytic cell lines, resulting in a tremendous increase in leukocytes in the peripheral blood stream. Some of these cells preserve the ability to differentiate in mature cells. Bone marrow may be further damaged by infiltration abnormal cells with replacement of normal hematopoietic process because interfere with normal production of hemapoietic cell lines. Continuous proliferation of mutative cells promotes their expansion in the organs and tissue in a form of metastasizing tumor cells.

Clinical features

The presence of neoplastic bone marrow element shows the following consequences. Increased nonfunctional white blood cell production and decreased normal

white blood cell production result in infections. Decreased red blood cell production leads to anemia. Decreased platelet production will result in a greater tendency to bleeding. There are three clinical stages of disease: stage I – initial, stage II – accelerated phase, stage III – dystrophy and blast crisis. The disease develops gradually. The initial symptoms and signs are not specific such as general malaise, fatigue, weight loss, low-grade fever. These disorders are detected accidentally as a rule. Some patients are symptomatic at diagnosis. In the pronounced stage weakness becomes considerable, night sweating profuse, elevation of temperature periodically to 37,5–39 °C, pain in the left hypochondrium, abdominal fullness and discomfort. Myeloid infiltration in the lung can be caused some additional symptoms, such as coughing. Many patients will complain on pain of the bones expecially the sternum and ribs, which are the major sites of blood cell production.

Objectivel examination reveals pallid skin with yellowish or grayish tint due to the anemia. The specific sign of disease is skin leukemic infiltration and local lesions. Infiltrations of the bones and the joints may cause localized lesions. Features of anemia may include dyspnea and tachycardia, lethargy. Chronic myelocytic leukemia is associated with increased susceptibility to infections such as pneumonia, plevritis, pyelonephritis.

In patients occurs considerable nonsymmetrical enlargement of abdomen predominantly in the left hypochondrium, due to the marked enlarged spleen. In about 10 % patients the enlargement is massive, extending to over 15 cm below the costal margin. The spleen is usually firm, smooth and painless. The presence of splenomegaly may be explained by excessive work to eliminate senescent and abnormal white blood cells. Enlarged liver is fairly common also. Less common neurological presentations include dizziness, visual disturbances, convulsions, paralysis resulting from the affection of the brain and spinal cord.

In patients is common bleeding syndrome with bruising, epistaxis, menorrhagia or hemorrhage from other sites.

In stage III may observe cachexia, secondary prolonged infections, progressive anemia, great tendency to bleeding. Finally, this chronic condition may transform to blast crises similar to an acute form of leukemia with the production of large number of immature myeloblasts in the bone marrow and the bloodstream. This new blastic phase is fatal.

Additional methods of examination

Clinical blood analysis:

– the number of blood cells is usually between $50x10^9/l$ and $300x10^9/l$;
– the number of red blood cells decreased;
– the number of platelet decreased;

– ESR accelerated;
– complete spectrum of myeloid cells is seen: blast cells, myelocytes, metamyelocytes, band cell, nature polymorphonuclear neutrophils;
– myeloblasts are usually less than 10 %;
– increased count of eosinophils and basophils.

Bone marrow:

– hypercellular with granulopoietic predominance;
– Phyladelphia chromosome in bone marrow cells is detected during cytogenetic analysis.

Polycythemia vera

Polycythemia vera may be defined as a myeloproliferative disorder characterized by a neoplastic hyperproduction of erythrocytic, granulocytic and megakaryocytic cell lines.

Etiology

Etiology is still unknown. Reactive phenomenon of blood cell lines possible related to cancerogenous agents.

Pathogenesis

The development of a proliferative abnormality common pluripotent stem cell of mesenchymal origin is accompanied by sequential formation of pathological cell clone, uncontrolled overproduction of all myeloid elements. Excessive erythropoiesis, megacaryocytosis lead to increased red blood cells and platelet count and as a result in increased total blood volume, hyperviscosity, tendency to thrombosis. Despite the increased platelet count above 1 million per microliter their function is impaired.

Clinical features

Polycythemia vera is more often seen in elderly individuals and affects both sexes equally. Clinical features reflect the pathogenic appearance and are the result of hypervolemia, stagnation of the blood flow.

The patients complain on the headache, insomnia, pain in the heart, bones, dyspnea, blurred vision, toggling of the finger. The specific symptom is pruritus expecially after contact with water. A significant number of patients with polycythemia vera will complain of itching after bathing.

Very important in diagnosis is plethoric appearance – redness cyanosis, of the skin and mucosa conjuctival suffusion, retinal venous enlargement. These symptoms are explained by high concentration of hemoglobin and increased number of

red blood cells, disturbances of arterial oxygen saturation, presence of methemoglobin. According to increased blood volume and peripheral resistance the hypertension and further left ventricular hypertrophy occur.

Splenomegalia presents at patient, as a result of myeloid metaplasia and excessive work for elimination of abnormal red blood cells. During the disease progression appears complication in a form of thrombosis of arterial and venous vessels. Thrombosis may occur at any site. Visual disturbances may be manifestations of polycythemia. Myocardial infarctions, strokes, thrombosis in the hepatic-portal system, phlebitis are the specific signs of this disease due to the increased viscosity of the blood, a large number of platelet and microcirculation disorders. Many platelets have functional defects that lead in some patients with polycythemia vera to hemorrhage. The most common form is bleeding into the gastrointestinal tract.

Additional methods of examination

Clinical blood analysis

– increase in hemoglobin	above 17.5 g/dl in males; above 15.5 g/dl in females;
– increase in red cell count:	above 6×10^{12}/l in males; above 5.5×10^{12}/l in females;
– increase in hematocrit:	above 55 % in males; above 47 % in females.

– leucocytosis;
– thrombocytosis;
– neutrophilic leukocytosis;
– in some cases basophilia.

Bone marrow:

– bone marrow is hypercellular with large numbers of erythrocytic, granulocytic precursor cells later may develop myelofibrosis.

Acute lymphoblastic leukemia

Acute lymphoblastic leukemia characterized by proliferation of poorly differentiated lymphoid blast cells with reduction of normal hematopoietic cells.

Etiology

The exact etiology of acute lymphoblastic leukemia is unknown. Genetic factors may be implicated in disease, because chromosome abnormalities are observed in some patients. Higher incidence of acute lymphoblastic leukemia occurs in persons with Down's syndrome. Environmental factors with mutagenic properties can be causal for onset.

Virus theory: at the present time more than 20 viruses have been isolated that can cause leukemia in animals. Previous studies have suggested the possibility development two kind of hemoblastosis resulted by virus: a) virus Epstein-Barr causes lymphoma Burkett's; b) T-lymphtropic virus causes T-cells leucosis.

Pathogenesis

Acute lymphoblastic leukemia is associated with a somatic mutation in the bone marrow stem cells, which may produce clones with excessively proliferation especially lymphoblast cell lines. Immunological markers indicate that most of these lymphoblasts belong to the non B-cell group and B-cell group of lymphocytes.

The FAB-classification

L1: presence of small blast cells with immunological markers of non B-cell group of lymphocytes. This type of acute lymphoblastic leukemia observed in 85 % children and 5–10 % adults and characterized by slowly progressive course with most favorable prognosis.

L2: morphological features of lymphocytes are large and small cells with immunological markers of non B-lymphocytes, T-cells and natural killer cells. This form is common for adults.

L3: Burkett's type of acute lymphoblastic leukemia. The cells are large and homogenous, belongs to the B-cell group. In children, adolescents acute lymphoblastic leukemia is very rare.

Clinical features

The most important sign of acute lymphoblastic leukemia is infiltration by malignant lymphoblasts almost any body organs and tissue first of all lymph nodes. Lymphadenopathy is a common feature, as a rule of the lymphoblastic form of leukemia. Approximately 75 % of acute lymphoblastic leukemia cases exhibit lymph node enlargement. Infiltration of the skin may result in local lesions.

Infiltration of the bones and the joints may cause localized lesions and pain. Tender bones, painful sternum (sternalgia) are explained by organ infiltration with extramedullary hematopoiesis. The lung is common site of infiltration by cancerous lymphoblasts which may result in pulmonary lesions.

The infiltration of gastrointestinal tract is also common in acute lymphoblastic leukemia cause damage the mucosa and local ulceration with hemorrhage.

Neurological syndrome include headache, nausea, vomiting, blurring of vision and diplopia. Fundal examination may reveal papilloedema and sometimes hemorrhage. Special type of acute lymphoblastic leukemia known as meningeal leukemia due to the infiltration with leukemic lymphoblasts the membranes surrounding the brain may observe.

Testicular infiltration by malignant lymphoblasts is observed in male patients with acute lymphoblastic leukemia.

The mortality in acute lymphoblastic leukemia arises mainly from neutropenia, thrombocytopenia and anemia because of bone marrow failure.

Additional methods of examination

Clinical blood analysis

- white blood cells count may vary from low level (less than $10x10^9/l$) to high level (more than $5x10^{11}/l$);
- red blood cell count decreased;
- thrombocytopenia;
- lymphoblast cells.

Bone marrow

- lymphoblast cells;
- normal erytrhropoietic, granulopoietic, megakaryocytic elements reduced.

Special test

- immunological identification of certain surface markers of the neoplastic lymphocytes.

Chronic lymphocytic leukemia

Chronic lymphocytic leukemia is now regarded as a tumor of the immunocompetent lymphatic tissue.

Etiology

Some environmental factors such ionizing radiation, mutagenic chemicals have been implicated at the onset of disease.

Pathogenesis

The cause of disease is mutation of cell – precursors of lymphopoiesis with formation of lymphoid hyperplasia. This disease is characterized by the presence of increased number of small, normal-looking lymphocytes in the blood, bone marrow and lymphoid tissue. Over 90 % of all cases are caused by B-cells, the remaining are T-cells. Lymphocytes morphologically mature, but functionally inadequate. Immunological failure occurs from reduced humoral and cellular immune processes. Lymphoid infiltration of the organs and tissue (lung, brain, spleen, liver, skin) leads to clinical features. Generalized hyperplasia of lymphoid organs causes the main signs of disease.

Clinical features

Chronic lymphocytic leukemia is a disease of middle-aged and elderly persons. There is peak occurrence between 50 and 60 years of age. This disease has slowly onset with non specific complaints on loss of weight, decreased appetite and fatigue. The first specific clinical finding at the beginning of the disease is symmetrical, discrete, non tender peripheral lymphadenopathy, which observes at about 80 % patients. Palpation is used to assess the enlargement of the lymph nodes and their properties. The lymph nodes are elastic, they do not fuse with the skin or with one another; they are painless in most cases. The lymph nodes never ulcerate or suppurate.

Under condition of benign process the initial stage may last for a long time. As the disease progresses the peripheral lymph nodes become enormous, firm. Lymphadenopathy processes becomes generalized character with involvement of cervical, submandibular, supraclavicular, axillary, inguinal lymph nodes. They fuse together with formation of conglomerates. Dyspnea and attacks of asphyxia are observed related to the enlargement of mediastinal lymph nodes and compression of trachea and bronchi. Diffuse swelling of the neck and face may occur with obstruction of the superior vena cava due to lymphous. Enlarged mesenterial lymph nodes cause compression of vena cava. Abdominal fullness, belching discomfort, abdominal pain may arise from intestinal obstruction by lymph nodes. Pronounced enlarged lymph nodes may obstruct the gallbladder, urinary tract, upper respiratory tract.

The progression of the disease is characterized by high temperature, intoxication, dispnoe, skin itching. In some patients may observe the appereance on skin the specific signs – lymphoma and non-specific signs such as herpetic lesions, weals. Widespread erythroderma occurs in some cases. Skin leukemic lymphoderma on the face made in like 'lion face".

Affection of the gastro-intestinal tract by lymphoid infiltration: dyspepsia, diarrhea. The liver and the spleen are enlarged and consolidated. Infraction of the spleen can occur; its palpation then becomes tender.

There may be an increased incidence of bacterial, viral or fungal infections due to a lack of functional white blood cells.

In thrombocytopenic patients appear bruising or purpura. Features of anemia are pallor skin and mucosa, tachycardia.

At terminal stages observe bleeding complications, intoxication, hyperthermia, heart and renal failure.

Additional methods of examination

Clinical blood analysis

– leucocytosis 3×10^{10}/l-3×10^{11}/l;

– red blood cell count decreased;

– platelet count decreased;
– between 70 and 90 % of white cells on blood film appear as mature lymphocytes;
– the structure of their nuclea and cytoplasm is sometimes quite peculiar; the cells are very soft and are easily destroyed, when preparing a smear; specific Botkin-Gymprecht shadows are formed.

Bone marrow

– bone marrow aspiration shows lymphocyte replacement of normal marrow elements. Lymphocytes comprise 25–95 % of all the cells in early stage;
– in terminal stage total lymphoid metaplasia;
– enlargement of mediastinal lymph nodes can be detected by standard X-ray examination or by tomography.

Hemophilia

Etiology

– congenital blood coagulation disorder;
– inheritance is sex linked, males are affected while females act as carrier;
– some cases do not have any family history and presumably result from spontaneous genetic mutations.

Pathogenesis

There are the major types of hemophilia: hemophilia A, hemophilia B (Chistmas disease) and hemophilia C.

Hemophilia A occurs as a result of low level or either absence of factor VIII, which primary synthesized by liver, but other organs such as the spleen. Kidney may also contribute to the plasma level. The factor VIII gene is localized on the X chromosome that is way the hemophilia A sex-linked disorder. All daughters of patient with hemophilia are obligate carriers and sisters have a 50 % chance of being a carrier. If a carrier has a son, he has a 50 %, chance of having hemophilia, and daughter has a 50 % chance of being a carrier. 33 % cases do not have family history. Lack of factor IX gene is known as hemophilia B (or Christmas' disease). Hemophilia C occurs in patients with lack of factor XI (Rosenthal syndrome).

Clinical feature

The main patients' complaints: spontaneous bleeding after trauma, dental extraction, surgery manipulation. Sometimes may be nasal, pulmonary hemorrhage and from gastrointestinal, genitourinary systems. The patient complains of the joint enlargement.

Objective examination. *General patient's condition* is usually satisfactory. In case of prolonged and recurrent hemorrhages and loss of large amount of blood general condition may be middle, grave or grave. The posture of the patients is active with restriction due to the pain and walking difficulties in affected joints and muscles caused by spontaneous bleeding.

The color of the skin and visible mucosa as a rule is pallor, with hemorrhages lesions: petechia, ecchymoses, and hematoma. Bleeding into the joints is known as hemarthrosis begin spontaneously without apparent trauma. The joints most commonly affected are knees, elbows, ankles and hips. Bone destruction occurs due to tepeated sub-perioctal hemorrhages. The defects undergo neoossification causing expansion and pathological fractures in the bones. The deformities of joints and bones are specific signs of hemophilic patients. Muscle hemotomas are also characteristic of hemophilia secondary to hematomas appears atrophy of muscles. These occur most commonly in the calf and psoas muscles but they can arise in almost any muscle and cause the pressing on the nerve with consequent parasthesia and weakness in the extremities, progressive muscle and nerve damage resulting neuropathy. Hemophilic pseudotumours may occur in long bones, pelvis, ringers and toes.

The course of disease is characterized by early onset in babies about 6 months old, when superficial bruising or a hemarthrosis may occur. The spontaneous bleeding episodes, joint deformity and crippling are observed entire the patient life. Hematuria is more frequently than gastrointestinal bleeding. Intracranial hemorrhage is rare, but in severe and prompt case it may be fatal outcome. Operative and postoperative hemorrhage is dangerous.

Additional methods of examination

Clinical blood analysis

- activated partial thromboplastin time increased;
- whole blood coagulation time is raised;
- factor VIII dolling assay (VIII C) reduced;
- immunological methods show normal VIII R, AG;
- bleeding time and prothrombin time tests normal;
- carrier females have half the clotting activity (VIII C) expected for the level of VIII R: AG.

X-ray examination

- broadening of femoral epicondyles;
- sclerosis, osteophyte and bony cists;
- atrophy of muscles.

The computer tomography scan

– intracerebral hematoma.

Hemophilia B (Christmas' disease)

Hemophilia B (Christmas disease) occurs as a result of a deficiency of factor IX. Like Hemophilia A it is also X-linked recessive trait. The clinical feature similar to the hemophilia A but bleeding is usually not as severe because factor IX is more stable than factor VIII:C.

Additional methods of examination

1. Activated partial thromboplastin time is raised.
2. Whole blood clotting time (severe cases) is raised.
3. Factor IX clotting assay is reduced.
4. Both bleeding time and prothrombin lime tests are normal.

Hemophilia C

Hemophilia C – may be defined as a bleeding disease caused by a deficiency of factor XI. It is inherited as a recessive trait. The symptoms and signs are similar to other type of hemophilia.

Idiopathic thrombocytopenic purpura (Werlhoff's disease)

Thrombocytopenia is most common form of bleeding disorders due to the quantitative abnormalities of platelets. Because a number of platelets reduce in the blood stream, their function is impaired.

Etiology

Causes of decreased platelet production:

– selective megakaryocytic depression in bone marrow: drug-induced, chemicals.

Infiltration of bone marrow:

– aplastic anemia;
– leukemia;
– myelosclerosis;
– multiple myeloma;
– megaloblastic anemia;
– carcinoma.

Increased destruction of platelets:

- disseminated intravascular coagulation;
- idiopathic thrombocytopenic purpura;
- viral infections – Epstein-Barr virus, HIV;
- bacterial infections – septicemia.

Pathogenesis

Decreased platelet production result from three mechanisms: failure of megakaryocyte maturation, excessive platelet consumption or their sequestration in an enlarged spleen. The pathogenesis of idiopathic thrombocytopenic purpura is associated with activation of the immune system, production of the auto-antibodies, often directed against platelet membrane glycoprotein IIb-IIIa. Platelet destruction results from increased phagocytosis of antigen-antibodies immune complexes adhere to platelet by monocyte-macrophage system of the spleen and liver. The antibody covered platelets premature removal from the circulation. The reason for production of the antibody is unknown, thus this form of disease is defined as idiopathic.

Clinical feature

Idiopathic thrombocytopenic purpura more commonly affects females at an early age. The main complaints are easy bruising in skin and bleeding from mucosa with sudden onset after easy trauma and sometimes spontaneously. Very often symptoms are the bleeding from nose, gastrointestinal tract, lung and kidney hemorrhage, in women – menorrhagia. The course of disease is chronic, with remissions and relapses.

Objective examination. General patient's condition is satisfactory. If bleeding persists for more than some days resulted acute posthemorrhage anemia the patient's condition become grave and required immediately treatment. The main clinical signs are the presence features of skin bruising different size: petechiae, purpura and even hematoma, which located at the anterior part of trunk and extremities. According to the term of bruising appearance may be change of color: with different tint: read, blue, green and yellow.

Skin bruising sometimes accompanied with profuse mucosa bleeding and become insidious character because occur posthemorrhage anemia. Spontaneous bleeding does not usually occur until the platelet count falls below about $30x10^9/l$.

Severe thrombocytopenia results in eye-ground hemorrhage, but intracranial hemorrhage is rare.

Splenomegaly is observered in about 10 % of the cases.

Additional methods of examination

- the platelet count is usually 10–50x10^9/l;
- the blood film shows reduced numbers of platelets;
- the bone marrow usually shows increased number of megakaryocytes;
- sensitive tests can demonstrate antiplatelet IgG either alone or with complement, on the platelet surface or in the serum in most patients.

Henoch-Schoenlein syndrome

The vascular disorders are a heterogeneous group of syndromes characterized by easy bruising and spontaneous bleeding from the blood vessels.

Vascular disorders resulting in abnormal hemostasis are classified into two major groups: inherited and acquired.

Henoch-Schoenlein syndrome belongs to acquired vascular disorders.

Etiology

Etiology is still unknown, but it was observed that this disease often begin after the ingestion of certain drugs or as a result of a group A streptococcal infection.

Pathogenesis

Pathogenesis may considered as hypersentivity response to the external factors with development of allergic reaction associated with acute inflammation of the small blood vessels and disorders of microcirculation, resulting in increased vascular permeability and easy bleeding.

Clinical features

This disease is most common in children, athough it also may occur in adults. The abrupt onset with pain in the joints and elevated temperature are the first features. The specific signs are the appearance on the legs and arms small pinpoint hemorrhages at the skin known as petechia accompanied with itching. The mucosa doesn't affect in adults. During severe course of disease appear the additional points anywhere in the body with swelling and necrosis. The hematuria may be the additional sign of disease. After two weeks the lesions disappeared without skin changing.

Additional methods of examination

Clinical blood analysis:

- normal erythrocytes and platelets count;
- leucocytes count increased;
- regenerative nuclear shift to the left;
- ESR accelerated.

Chapter 13

ENDOCRINE SYSTEM

The pathology of thyroid gland

Predominantly thyroxin (T_4), only small amount of triiodothyronine (T_3) is produced by the thyroid gland. Approximately 85 % of T_3 is produced in liver, muscle and kidney. T_4 may be regarded as prohormone, because it is not metabolically active until converted to T_3. Production of T_3 and T_4 in the thyroid gland is stimulated by thyrotrophin (thyroid-stimulating hormone, TSH) a glycoprotein released from the thyrotroph cells of the anterior pituitary in response to the hypothalamic tripeptide, thyrotrophin-releasing hormone (TRH).

Hormones of thyroid gland play an important biological role in organism:

- metabolic (enchanced tissue oxidation and responsible for energy balance);
- take part in the synthesis of protein;
- regulate energy production by changing of metabolism carbohydrate;
- interfere in metabolism carbohydrate in intestine, stimulate glyconeogenesis, glycogenolysis, promotes lipolysis;
- take part in metabolism of fats, accelerates lipolysis;
- regulate the function of cardiovascular, nervous and reproductive systems;
- regulate the vitamins metabolism.

In pathological conditions may be decreasing and increasing function of the thyroid gland: hypothyroidism and hyperthyroidism.

Hypothyroidism

Hypothyroidism (hypothyroid syndrome) is pathological condition which resulted from insufficient secretion the thyroid hormones by thyroid gland.

Etiology

- inherited disorder of thyroid hormones biosynthesis;
- defects of embryonic development, resulting to hypo- or aplasia of thyroid gland;
- infectious-inflammatory processes, including chronic infections (tuberculosis, syphilis), attended with the degenerative changes of gland parenchyma;
- residence in endemic region with the deficit of iodine in environment;
- Hashimoto's thyroiditis;
- jatrogenic hypothyroidism due to thyroidectomy or radioactive iodine therapy;
- outcomes of treatment the diffuse toxic goiter with antithyroide drugs;

– pathology of the hypotalamo-hypophysal system, relevant with decreasing of thyrotrophin-releasing hormone.

Pathogenesis

Hypothyroidism is a result of organ-specific autoimmune process which leads to destructive lymphoid infiltration of the thyroid gland transformed in fibrosis, atrophy and decreased amount of functional cells that accompanied delayed synthesis of thyroid hormones and impairment of functional activity of gland. The deficiency of the thyroid hormones are responsible for disorders every forms of metabolism. The specific sign of hypothyroidism is accumulation in extravascular tissue hydrophilic mucopolysaccharides, hyaluronic acid and chondroitin sulfate which possessing ability to retain water in organism and development of mucous edema.

The other causes of edema are increased vascular permeability and decreased lymph flow. Hypothyroidism is accompanied by hemodynamic disorders: decreasing of the rate and force of cardiac contractility, cardiac output, increasing peripheral resistance, cerebral, cutaneous and renal blood flow are reduced with corresponding clinical appearance.

The metabolic disorders are accompanied by following features: decreased energy productioin; electrolyte exchange; the expressed dystrophic changes in all organs and tissues; diminished erythropoesis; the considerable changes of central and peripheral nervous system; cardiovascular system with predominance of hypokinetic type of hemodinamic, digestive systems with decreased secretory and motor functions of stomach and intestine. The pathology of hypothalamo-hypophysal region and decreased release of TSH secretion lead to depressed synthesis of thyroid hormone, which results in development of the secondary hypothyroid syndrome.

Clinical features

The complaints are general weakness, tiredness, somnolence, cold intolerance, poor hearing, disorders of mental abilities, dyspnea, constipation, weight gain, impairment of sexual function, headache and dizziness. Subjective muscle dysfunction is the myalgia, muscle cramps and stiffness. The complaints of muscle weakness and fatigability are common. Hypothyroid patients may complain of arthralgia and joint stiffness.

Objective examination

The condition of the patient is satisfactory. In case of prolonged hypothyroidism and accompanying changes of organs the condition becomes grave and even extremely grave if the coma occurs. The patient with hypothyroidism as a rule

is adynamic. Patient has specific features of face, so called myxoedematous face (facies myxoedemica): periorbital puffiness narrowing of the eye slit, poor mimic, purplish lips, the hair is thinned or absent on the outward portions of the eyebrows. The patient looks older his real age.

The skin of patient is pale with icterus tint. Pallor develops due to vasoconstriction and anemia. Yellow color is explained by carotinemia resulted from delayed turnover of carotin to vitamin A. The skin becomes rough, scaly and thickened. Trophic disorders of the skin and its derivates are characterized by such signs: dryness, fragility and loss of hairs, nails are thin, brittle with transvered lines, grow slowly. Infiltration of the skin with specific substances which content mucopolysaccharides leads to edema which does not pit with pressure. The edema is most marked in the skin of the hands, feet. The periorbital edema is observed. As a rule edema is nonpitting. If the edema is pitting, especially in the legs, should suspect the presence of complication as heart failure as a result of myxoedematous heart.

The skin is cold due to the both thickening and decreased blood flow, poor peripheral circulations. The patients are usually overweight.

Signs of muscular affection are dystrophy, muscle stiffness, mainly at the proximal part of extremities, muscular weakness and painful spasms, myotonia. Chronic hypothyroid myopathy occurs due to increased muscle mass (pseudohyperthrophy). Movements may be slow and clumsy. Some patients have synovial thickening and synovial effusions, usually of the knees.

Respiratory system. The signs related to the upper airways and respiratory system include chronic nasal congection, shortness of breath and sleep apnea. Complaints referable to the airways are explained by mucinous edema of the nasal mucosa and larynx. Shortness of breath may inducate on the presence of pleural effusion. The patient has low-pitched hoarse voice due to the edema of vocal cords. Simultaneously with enlarged tongue and lips therefore speech become very slowly with lingering intervals. The course of hypothyroidism is characterized by frequent attacks vasomotor rhinitis, pneumonia.

Disorders of the *cardiovascular system* are characterized so-called, myxoedematous heart due to the interstitial myocardial edema, dystrophy of its fibers. The apex beat is displaced to the left, diffuse and weak. In percussion the borders of relative cardiac dullness are displaced outside. In auscultation decreased loudness of both sounds over in all listening points are revealed. In case of pericardial effusion the loudness of cardiac sounds extremely decreased or even disappears. Constant and stable bradycardia less than 60 beats per minute is typical sign of hypothyroidism. Appearance of premature beat is possible. Blood pressure mainly systolic is decreased, pulse pressure diminished. Hypothyroidism promotes and accelerates the development of atherosclerosis with affection different vessels. These patients as usually have clinical features of ischemic heart disease.

Changes from the *digestive system* occur due to the edema of gastrointestinal tract, which associated with motor and secretory dysfunction. These alterations appear as persistent constipation, flatulence, paresis of intestine. Decreased intestinal motility may cause abdominal distension and constipation, produce paralytic ileus or megacolon with the clinical feature of intestinal obstruction. Sometimes is observed the propensity to parodontosis. The liver usually is unaffected. Ascitis is uncommon sign of hypothyroidism.

Renal blood flow is reduced according to the decrease in cardiac output. The glomerular filtration rate is usually reduced and mild proteinuria is revealed. Due to the hypotension develops oliguria till anuria.

Neurological dysfunction manifested by carpal tunnel syndrome due to the edema of the flexor of the wrist. Patient has slow deep tendon reflexes with prolonged contraction and relaxation phases.

Cerebellar dysfunction includes such signs as ataxia, intention tremor and nystagmus. Disorders thermoregulation may present in a form of cold intolerance.

Hypothyroid patients have the signs of mental dysfunction due to chronic insufficiency of cerebral circulation, hypoxia and brain edema. The main features are somnolence, and even lethargy. The patient may sleep longer at night or may go to asleep frequently during the day.

The patient becomes physically and mentally slow: the speech is slow, memory loss, significant limitation of activity, adynamia, and apathy. In some patients may observe depression, indifference to surrounding or severe anxiety and agitation, maniac state. Lingering hypothyroidism leads to cognitive impairment and overt dementia. Outcomes of hypothyroidism may be disorders of pituitary gland. In men observe decreased sexual potential till impotence, deranged spermatogenesis. In women appears amenorrhea or menorrhagia, infertility. In patients of both gender groups occur diminished libido, no pubic and armpit hair. One of the dangerous complications of disease may be hypothyroid coma. The patient's condition is grave, unconsciousness, passive posture, declining body temperature till 23 °C, deep and slow breating sinus bradycardia till 30 beats per minute, hypotension. Accumulation of fluid in pericardial cavity may cause the compression of the heart with acute heart failure. Constant and severe constipation resemble ileus.

Additional methods of examination

Clinical blood analysis: normocytic, microcytic or macrocytic anemia (pernicious-like). Macrocytic anemia is associated with vitamin B_{12} malabsorption. Leukocyte, lymphocyte and platelet count are normal, accelerated ESR.

Biochemical blood analysis: elevated serum creatine kinase (MM fraction) concentration due to the release from skeletal muscle as a result of increased sarcolemmal permeability.

The serum concentration of total and low density lipoprotein cholesterol and sometimes triglycerides are elevated; hypoalbuminemia with hyperglobulinemia.

Elevated serum lactate dehydrogenase, aminotransferase concentrations iron deficiency in premenopausal women.

Radioimmunologocal method: diminishing of thyroxin level (T4) and triiodothyronin (T3). Thyroidal radioidine uptake is reduced in most patients with hypothyroidism.

ECG: bradycardia, low amplitude P wave and QRS complex, conduction disturbances, depressed ST-segment and inverted T-wave.

Echo-CG: septal and wall thickening, decreased myocardial contractility and relaxation, pericardial effusion.

Ultrasound examination of thyroid gland allows reveal the changes at echogenic density.

Radioisotope scanning by thechnetium-99 gives the information about functional activity of gland, size, form, presence of "cold" and "hot" zones.

Hyperthyroidism

Hyperthyroidism (hyperthyroid syndrome) – complex of symptoms, which results from hyperfunction of thyroid gland with increased secretion of thyroid hormones and expose them on the body tissues.

Etiology

In over 90 % of patients with hyperthyroidism is due to Graves' disease. Other causes of excess production thyroid hormones are thyroiditis, multinodular goiter, iodide-induced after lingering taking some drugs (e.g. amiodarone). Extra thyroidal source of thyroid hormone excess is due struma ovary or excess secretion of TSH which may originate from pituitary in case of tumor – choriocarcinoma or hydatidiform mole.

The factors which provoke the development hyperthyroidism include:

- inherited predisposition;
- acute and chronic psychical traumas;
- acute and chronic infections (flu, quinsy, measles, whooping-cough, tuberculosis, chronic tonsillitis, encephalitis, rheumatism);
- immunological disturbances;
- neuro-endocrine alteration in woman (pubertat period, pregnancy, lactation, climax);
- chronic disease of liver and kidneys, attended with disorders of metabolism of thyroid hormones.

Pathogenesis

The development of hyperthyroidism is explained by immunological theory. The production of IgG antibodies directed against the TSH-reseptor on the thyroid follicular cell stimulates thyroid hormone production.

In blood there are increased concentration of thyroxin and triiodthyronin that lead to disorders of metabolism – proteins, lipids carbohydrates. Increased tone of sympathetic nervous system causes the development of hyperkinetic type of hemodynamic. Heart rate, cardiac contractility, stroke volume and cardiac output are increased and peripheral resistance is decreased. These alterations are explained by direct chronotropic and inotropic effects of thyroid hormones. The appearance of ophthalmopathy is caused by rise in retrobulbar pressure due to elevated interstitial fluid content and chronic inflammation, proliferation of fibroblasts. The eye is displaced forwards – exophthalmos.

Clinical feature

The main symptoms of the hyperthyroidism:

- excitability, anxiety, nervousness, irrability;
- increased sweating, heat intolerancy;
- fatigue, muscular weakness;
- despite normal or increased appetite loss of weight;
- dyspnea on exertion;
- exacerbation of asthma;
- palpitation, pains in heart region;
- escaped beat;
- thirst, anorexia, vomiting, diarrhea;
- loss of libido, impotence.

Objective examination. The condition of patient is satisfactory. In case of hyperthyroid crises may be life-threatening condition. The consciousness is clear, posture is active. The patient is characterized by fast changing of mood, impossibility to be concentrated, motor, emotional and vocal lability. Patient looks younger his age.

The face has specific signs: lively with widened eye slits, exophtalmos, excessive lacrimation, corneal ulceration, hyperemia of conjunctiva.

There are specific eye signs in patients with hyperthyroidism:

- upper lids are symmetrically retracted so that some sclera is visible;
- Kraus' sign (abnormally sparkling eyes);
- Ellinec' sign (pigmentation of eyelids related to adrenal insufficiency);
- Rosenbach' sign (shallow tremor of the closed eyelids);
- Greffe' sign (lid lag during fixing of slowly downward moving object);
- Mebius' sign (weakness of convergence or loss of ability to fix a object at short distance);
- Stelvag' sign (rare blinking, less than 6–8 times in a minute).

Dermopathy is characterized by moist, hyperemic skin with palmar erythema, spider naive, sometimes appearance pigmentation, vitiligo and giblet clubbing. The hair may become thin and fine in texture and alopecia can occur. The nail is soft and separated from the nail bed – onchyolysis. Perspiration is increased. In patient may be pretibial myxoedema in the form of pink coloured or purplish plagues on the anterior part of the leg, accompanied with itching. The muscles are atrophic, their force and tone are reduced.

Thyroid enlargement of some type is a common sign.

Classification of diffuse toxic goitre according to degree of enlargement of thyroid gland: 0 – there are no thyroid gland at palpation; 1 – at palpation enlarged isthmus of gland and slightly lateral lobes; 2 – thyroid gland is noticeable at swallowing, at palpation determined well; 3 – «thin neck» (enlarged thyroid gland is well noticeable at examination); 4 – the expressed goitre, sharply changing configuration of neck; 5 – degree is goitre of largeness.

The size and consistency varies according to the pathology caused hyperthyroidism. In Graves' disease the thyroid is twofold to fourfold enlarged slightly tender and the surface is usually smooth. The thyroid bruit or thrill related to increased thyroid blood flow are heard. Thyroiditis usually characterized by slight diffuse thyroid enlargement.

The affection of respiratory system in patient is uncommon. In severe hyperthyroidism may be pneumonia and abnormalities in respiratory function. Decreased vital capacity decreased pulmonary compliance, respiratory muscle weakness cause dyspnea, aggravation of bronchial asthma.

The leading signs of affection of cardiovascular system, which accompanied the hyperthyroidism at every stage are defined as thyroidtoxic heart. The apex beat displaced to the left, diffuse, high and strong. The main sign of hyperthyroidism is stable arrhythmic tachycardia more than 90 beats per minute. The heart sounds are loud. Functional systolic murmur can appear over all auscultative points. Pulse is high and fast (altus et celer). Functional murmur can be heard over a. carotis and v. jugularis. Systolic blood pressure is increased, diastolic blood pressure is decreased, pulse pressure is elevated. In case of constant longstanding atrial fibrillation in patients with hyperthyroidism may be complication in the form of heart failure with congestion in lesser and grater circulation.

Due to the increased motility of stomach and intestine appear the pain and hyperdefecation. Intestinal hypermotility lead to more rapid small and large intestinal transport, resulting in steatorrhea. Inactivation of thyroid hormones takes place in liver hence in condition of their excess circulation develop hepatic dysfunction with raised concentrations of enzymes. In severe course of disease the enlarged liver and jaundice are observed.

Renal blood flow, glomerular filtration rate and secretory capacities are increased. The common sign are polydipsia and polyuria. Urine concentrating ability may be impaired due to the dehydratation.

Neuromuscular signs of the hyperthyroidism: shallow symmetrical tremor of fingers when the hands are extended, but it may involve the arms, legs, tongue and head. The movements are rapid and low amplitude. Instability in the Romberg' posture, hyperreflexia, muscle weakness, proximal myophaty, increased reflexes of tendons are observed. Myopathy can also involve the respiratory and oropharyngeal musculature, causing difficulties in swallowing or hoarseness. After exercise may be the attacks of periodic paralysis. The signs of central nervous system dysfunction are anxiety, irritability, episodes of paranoia, impairment of cognitive function.

Disorders of endocrine system include gonadal dysfunction. In women menstrual cycles are normal, although some of them have oligomenorrhea or amenorrhea accompanied with infertility.

In men hyperthyroidism results in decreased potency and loss of libido. Gynecomastia may be observed.

Additional methods of examination

Clinical blood analysis: normochromic anemia, reduction of hemoglobin values, leucopenia, relative lymphocytosis, granulocytopenia, easy thrombocytopenia.

Biochemical blood analysis: oral glucose tolerance is impaired, modest reduction in serum total and LDL cholesterol concentrations, serum triglyceride concentration is normal, hepatic lipase activity and plasma free fatty acid concentration are increased. Serum alanine and aspartate aminotransferase, bilirubin concentrations are increased.

Immunological examination: decreased of T-lymphocytes and T-lymphocytes-supressors, increased of level of immunoglobulins, antibodies to thyroglobulin; appearance of thyrostimulating immunoglobulins.

Radioimmunological examination: increased serum total and free T_4 and T_3 concentrations and decreased TSH concentration. Thyroid radioiodine uptake is increased in most patients.

ECG: atrial fibrillation, depresses ST-segment and inverted T-wave.

Diabetes mellitus

Diabetes mellitus is a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, metabolism resulting from defects of insulin secretion, insulin action, or a combination of both.

There are two types of diabetes mellitus: insulin dependent diabetes mellitus (IDDM) called type 1 diabetes and non-insulin dependent diabetes mellitus (IN-DDM) called type 2 diabetes.

Table 6.1. ***Etiological classification of glycemia disorders***

Type 1 (β-cell destruction, usually leading to absolute insulin deficiency)
Autoimmune
Idiopatic
Type 2 (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with or without insulin resistance)
Other specific type
Genetic defects of β-cell function
Genetic defect in insulin action
Diseases of the exocrine pancreas: pancreatitis, trauma/pancreatectomy, neoplasia, cystic fibrosis
Endocrinophaties: Cushing's syndrome, acromegaly, phaeochromocytoma, hyperthyroidism
Drug or chemical-induced: cortisone, anti-depressant drugs, beta-blockers,, thiazide, infections: cytomegalovirus
Uncommon forms of immune-mediated diabetes
Other genetic syndromes sometimes associated with diabetes: Down's syndrome, Friederich's ataxia, Klinefelter's syndrome, Wolfram's syndrome
Gestational diabetes

Classification according to clinical feature

Severity of diabetes:
- mild (1 degree),
- moderate (II degree),
- severe (III degree).

Compensation state:
- compensated,
- subcompensated,
- decompensated.

Complications:
- ketoacidotic coma, hyperosmolar coma, lactacidotic coma, hypoglycemic coma;
- microangiopathy – retinopaty, nephropaty;
- macroangiopathy,
- neuropathy.

The type 1 diabetes, it is due to a lack of endogenous pancreatic insulin production, whereas in type 2 diabetes, the rising blood glucose results from a combination of genetic predisposition, unhealthy diet, physical inactivity, and increasing weight with a central distribution resulting in complex pathophysiological processes.

Etiology and pathogenesis of insulin dependent diabetes mallitus

The precise cause is still unknown in both main types of diabetes, nevertheless the etiology is considered as interaction of environmental factors with a genetic susceptibility. The pattern of these factors differ in IDDM and NIDDM.

Insulin dependent diabetes mellitus has polygenic genetic predisposition. Recently the role of genetic factor has finally proved as principal reason on development diabetes mellitus. Over 50 % of the heritability is contributed by the HLA class II genes which determines immune responsiveness and located in 6 chromosome being the marker of the predisposition to the autoimmune damage pancreatic islet insulin-secreting cells, wich becomes insulin-auto-antibodies. Selective destruction of beta cells occurs. In clinical expression of the disease an important role have environmental factors. Viral infections can induce diabetes by two pathogenic mechanisms: destruction of the pancreatic beta cells by direct cytolysis result from possessing tropism to the Langergans' islet (German measles, Coxsackie B4 virus, virus of hepatitis B, epidemic parotitis, cytomegalovirus); induction of an autoimmune destructive process results from infections.

In modern diabetology the following stage of pathogenesis are offered:

The I stage is genetic predisposition, by presence of certain antigens of HLA-system, and also genes 10 and 11 chromosomes.

The II stage is initiation of autoimmune process in beta-cells under influencing of pancreotroping viruses, cytostatics and other unknown factors.

The III stage is the stage of active immune processes with formation of antibodies to beta-cells, to insulin and by development of autoimmune insulitis.

The IV stage is progressive declining of secretion of insulin, by the stimulated glucose (1 phase of secretion of insulin).

The V stage is clinical overt diabetes. This stage develops, in case of destruction and death of 85–90 % beta-cells.

The VI stage is complete destruction of beta-cells, complete absence of secretion of insulin and C-peptide.

Type 1 diabetes characterized by deficiency of insulin due to destructive lesions of pancreatic beta-cells; usually progresses to the stage of absolute insulin deficiency. Typically, it occurs in young subjects with acute onset with typical symptoms of diabetes together with weight loss and propensity to ketosis, but type 1

diabetes may occur at any age, sometimes with slow progression. People, who have antibodies to pancreatic beta-cells such as glutamic-acid-decarboxylase (GAD), are likely to develop either typical acute-onset or slow-progressive insulin-dependent diabetes. Today antibodies to pancreatic β-cells are considered as a marker of type 1 diabetes, although such antibodies are not detectable in all patients.

Etiology and pathogenesis of insulin nondependent diabetes mellitus

A genetic factor has a large value in development of INDDM. Inherited predisposition is more important of this type of diabetes than in IDDM with concordance for NIDDM approaching 100 %. Risk of development of disease increased from twofold to sixfold at presence of NIDDM at parents or close relatives. However the certain genetic defect is still unknown.

NIDDM is commonly associated with several other disorders such as obesity, hypertension and hyperlipidemia, so-called metabolic syndrome. The patients are often obese or have been obese in the past and have typically been physically inactive. The risk of development of INDDM at obesity 1 degree is increased in 2 times, at II degree – in 5 times, and at III degree – more than in 10 times. Obesity especially abdominal type, acts as a diabetogenic factors through increasing resistance to the action of insulin. Epidemiologic studies of NIDDM provide evidence that food of these patients are diabetigenic features and content the high caloric easy absorbed carbohydrates, sweets, alcohol and deficit of vegetable cellulose. Such lifestyle factor, as over-eating, especially when combined with obesity and under activity lead to development of NIDDM.

Ageing is an important risk factor for NIDDM because the disease mainly develops after middle age, in contrast to IDDM, which commonly affects younger people.

Pathogenesis of NIDDM connected with combination decreased insulin secretion and decreased insulin sensitivity. Early stage of NIDDM is characterized by insulin resistance which means the reduced sensitiveness of peripheral tissue commonly skeletal muscle to insulin. There are three levels of insulin resistance: pre-receptor, receptor and post receptor. Pre-receptor insulin resistance is related to production of inactive insulin molecule or disturbances of proinsulin transformation in insulin with store a excess amount of proinsulin which is biologically inactive. Receptor insulin resistance is due to the synthesis of abnormally inactive receptors to insulin in target organs (liver, fat and muscle) results in appearance of antibodies to the insulin receptors. Post receptor insulin resistance is characterized by reduced metabolic activity of insulin in cells due to decreased tirozin kinase activity. In development of insulin resistance take part circulating in blood antago-

nists to insulin such as antibodies and contrinsulin hormones – cortisol, thyroid hormone, glucagons, catecholamines, prolactin. Thus insulin resistance may be to one of three general cauŝes: an abnormal insulin molecule, an excessive amount of circulating antagonists and target tissue disturbances.

Clinical features

The symptoms and signs of diabetes may be divided into three group regards to main metabolic consequences of lack of insulin (hyperglycemia); specific long-term complications of diabetes (microangiopathy); including retinopathy with potential blindness, nephropathy with a risk of progression to renal failure, nephropathy with a risk for foot ulcers, amputation, and Charcot joints and autonomic dysfunction such as sexual impairment; acceleration of comorbid pathology due to diabetes (atherosclerosis, infections). Thus DM is associated with development of specific long-term organ damage (diabetes complication).

Symptoms of hyperglycemia. When the concentration of glucose in the plasma exceeds the renal threshold glucosuria occurs. In case of consistent glucosuria (generally >180 mg/dl or 10,0 mM throughout the day and night) the classical symptoms of diabetes appears: polydipsia, polyuria and polyphagia which is related directly to the degree of glucosuria. Polydipsia is accompanied by thirst and dryness in the mouth. In the period decompensation patient can drink more than 5 liters of water in day, quite often he drink a lot of water at night.

Characteristic of diabetes is increased appetite (polyphagia). Nevertheless inspite the increased of food intake, such patients lose weight because of the loss of glucose in the urine. Weight loss is specific features for patients with IDDM and not expressed or absent at INDDM which, as a rule, is accompanied by obesity. Polyphagia goes down sharply during severe decompensation, commonly at ketoacidosis. Frequent and abundant urination (polyuria and pollakiuria) both in the day and night time present at diabetic patients. Osmotic diuresis due to lack of insulin may contribute to pathophysiology of polydipsia and polyuria. Increased catabolism lead to augmentation of glycogenolysis, gluconeogenesis, lipolysis result in protein wasting and increased urinary nitrogen loss. These changes in protein and fat metabolism contribute to wasting, loss of weight, growth retardation in children. Protein wasting in patients with uncontrolled diabetes may be responsible for general and muscle weakness, poor wound healing. Sharp changes of glucose concentration and plasma osmolarity may cause visual blurring. Persistent glucosuria is frequently accompanied by skin itching, in women in region of genitals due to vulvovaginitis with odorous vaginal discharge.

The specific symptoms related to diabetic mocroangiopathy correspond with affection of retina, kidneys, nervous system. The main complaints are partial an temporary impairment of vision and even blindness, appearance of edema due to the nephritic syndrome as evidence of diabetic nephrophaty, change of diuresis at initial stage in a form of polyuria, later oliguria. In stage of renal failure – appear anuria. Involvement of the nervous system in diabetes gives such clinical manifestations: numbness, tingling, paresthesias, burning and sharp pain in the distal portions of the lower extremities, less frequently of the upper extremities. Abnormalities of the autonomic nervous system occur in patients with long-standing diabetes with involvement cardiovascular, gastrointestinal, urogenital systems. The patients complain on palpitation escape beat, nausea, vomiting, dysphagia, diarrhea, urinary incontinence, failure to empty the urinary bladder fully. Impotence presents in 40 % of diabetic patients.

At early stage of diabetes are there metabolic abnormalities which serve as pathophysiological basis for increased predisposition to atherosclerosis of different vessels: coronary, peripheral. Coronary artery disease occurs more frequently in diabetic patients compared with general population. Clinical manifestation of comorbid pathology is chest pain different duration and intensity, arrhythmia. Atherosclerotic affection of peripheral vessels commonly low extremities is characterized by intermittet claudication and in more severe vessel occlusion, pain at rest.

Objective examination. *The patient condition* depends on stage of disease and evidence of complications. In early stage of disease the patient' condition is satisfactory. As a result of diabetic nephropathy, renal failure, visual impairment, clinical signs of neuropathy, cardiovascular disorders the condition becomes grave and even extremely grave in case of diabetic ketoacidosis, which characterized by passive position and unconsciousness. There are some features of diabetic dermopathy as a direct result of metabolic abnormalities: dryness of skin, decreased turgor and elasticity, shiny spots, scaly patches, diabetic bullae which located in the pretibial area. In diabetic patients with marked hypertryglyceridemia may observe eruptive xanthomas, which located in the elbow, shin; knees, buttocks and posterior thigh areas. In area of eyelids quite often it is possible to find xantelasm yellow spots with content of lipid. Red-brown papulae are observed on the skin of shins, later transformed in pigmental atrophy spots. In case of dilation of skin capillaries the hyperemia of skin in area of cheek and neck are appeared so called diabetic blush. Infectious complications of skin lead to furuncules which in diabetic patients progress to disseminated process with formation carbuncules. Some factors ischemia, peripheral polyneuropathy and infections cause foot uncler, cel-

lulitis and gangrene. Neuropathic ulcers may appear in the ares of callus formation. Changes of joints related to neuropathic arthrophaty occur at the ankle or in the foot at the tarsometatarsal or metatarsophalangeal joints. So called Charcot's joints are characterized by unilateral painless swelling, erythema in association with joint instability.

Affection of the respiratory system in diabetes may assume progressive and threatening forms due to the infections. Patients with diabetes are predisposed to tuberculosis which progresses very rapidly. Prognosis for diabetic patients is worse than for nondiabetic person in case of tuberculosis. Infections of various types lead to complications such as nasal sinusitis with purulent process and gangrene of the nasal mucous membrane, bronchitis, pneumonia.

Affection of the cardiovascular system may take the form of diabetic cardiomyopathy and coronary heart disease due to the acceleration of atherosclerotic process. Clinically diabetic cardiomyopathy may manifests as chest pain and features of congestive heart failure: dyspnea, cough, edema. Decreased ventricular ejection fraction is revealed by echocardiography and radionuclide ventriculography.

Arterial hypertension as a rule has secondary origin as a sign of diabetic nephropathy, chronic pyelonephritis, atherosclerosis of kidney arteries, cerebral atherosclerosis. Patients with diabetes are at a particularly high risk for cardiovascular, cerebrovascular and peripheral artery disease relevant to atherosclerosis. The risk of myocardial infarction and sudden death are higher twofold in diabetic patients as compare with nondiabetic population. Myocardial infarction occurs at young age, has severe course and poor outcomes, complicated by cardiogenic shock, thromboembolism of pulmonary artery, aneurism of left ventricle. Period of rehabilitation lasts more longer and myocardial remodeling more frequent results in development of heart failure. A greater prevalence of "silent" myocardial infarction among diabetic patients have been revealed. Death rate relevant to myocardial infarction account on the average 38–50 % at patients with diabetes.

The factors responsible for the accelerated atherosclerosis in the diabetic population are hypercholesterolemia, hypertriglyceridemia, increased concentration of low density lipoproteins, reduced concentration of high density lipoproteins, increased adhesiveness and sensitivity to aggregating agents. Cluster of impaired glucose homeostasis, abdominal obesity, hyperlipidemia and hypertension have been described as metabolic syndrome with specific entity "dead quartet". This term considered the meaning of metabolic abnormalities as crucial factors for atherosclerosis and vascular complication more commonly in NIDDM. It seems likely that insulin resistance being the primary defect of metabolic syndrome.

Signs of peripheral atherosclerosis may include diminished or impalpable pulses in the feet, bruits over the carotid or femoral arteries. According to the atherosclerotic oclusion of tibiae and popliteal arteries occurs vascular impairment in the leg and/or foot with development of ulcer and gangrene. Infected necrotic lesions, complicated by cellulites, osteomylitis and generalized septicemia required surgical amputation.

Gastrointestinal disorders at diabetes are explained by decreased motility with distension of stomach and gallbladder which clinically are manifested by poor esophageal contraction, diarrhea, "blind loop" syndrome, malabsorption. Hepatomegaly due to marked fatty infiltration of the liver is observed in patients with decompensated diabetes.

The most dangerous complication of both IDDM and NIDDM is diabetic nephropathy, which responsible for development or renal failure and is a major cause of morbidity and mortality in the diabetic population. The earliest clinical manifestation is microalbuminuria which is defined as an increase in urinary albumin measurable by radioimmune assay. Further progression from early stage to overt diabetic nephropathy is manifested by gross proteinuria in excess of 500 mg/24 h. The proteinuria may reach massive proportions, resulting in the nephritic syndrome with hypoalbuminemia and edema. The course of diabetic nephropathy is complicated by the presence of symptomatic hypertension. The outcomes of nephropathy are renal failure.

Urogenital dysfunction results from incompetence of internal vesicle sphincter, dilation of urine bladder, accompanied by chronic recurrent urinary tract infections: pyelonephritis, cystitis. In women the vulvogavinitis is detected. The women in fertile age are predisposed to reproductive dysfunction and have an increased incidence of stillborn, abnormally large and heavy babies and babies with congenital defects.

Involvement of the nervous system in diabetes has been described as clinical specific syndrome of diabetic neuropathy. Another reason of nervous dysfunction is a consequence of accelerated atherosclerosis leading to infarction of a spinal or cerebral artery. The various forms of diabetic neuropathy may be divided into three major clinical groups: symmetric peripheral polyneuropathy, mononeuropathy and autonomic neuropathy. The main signs of diabetic neuropathy are loss of vibratory sensation distally in the legs, depression or loss of the tendon reflexes at the ankles. Deep and penetrating neuropathic ulcers at the feet may develop. In case of mononeurupathy muscle atrophy may arise.

One of the serious complications of diabetes is retinopathy as the leading cause of blindness. Diabetic patients are prediposided to the development of glau-

coma. Metabolic cataract is most commonly observed in patients with uncontrolled diabetes with high hyperglycemia.

The classical clinical features of the two main types of diabetes as regard to family history, peculiarities of symptoms, age at onset of disease are differ (tab. 6.2).

Table 6.2. ***Comparative clinical features of IDDM and NIDDM***

Clinical features	IDDM	NIDDM
Age of onset	<40 years	>50 years
Genetic prediposition	Moderate, requires environ-mental for expression	Strong – 80–100 %
Family history	No	Yes
Duration of symptoms	Weeks	Months – years
Precipitating factors	Viral, other trigger autoimmune disease	Obesity, age
Autoantibodies	Yes	No
Insulin resistance	No	Yes
Ketonuria	Yes	No
Response to stress withdrawal of insulin	Ketoacidosis	Hyperglycemia without ketoacidosis
Treatment	Diet, insulin	Diet, oral antidiabetic drugs
Rapid death without treatment with insulin	Yes	No

Typically, the early stage of type 2 diabetes is characterized by insulin resistance and decreased ability for insulin secretion causing excessive post-prandial hyperglycemia. This is followed by a gradually deteriorating first-phase insulin response to increased blood glucose concentration. Type 2 diabetes, comprising over 90 % of adults patients are often obese or have been obese in the past and have typically been physically inactive. Ketoacidosis is uncommon, but may occur in the presence of severe infection or severe stress.

Classification of diabetes includes both etiological types (tab. 6.1) and different stages of hyperglycemia. The first classification of diabetes was proposed by the National Diabetes Data Group in 1979 and World Health Organization (WHO) in 1980. A few modifications have been introduced by the WHO and the American Diabetes Association (ADA) (tab. 6.3).

Table 6.3. ***Criteria used for glucmetabolic classification according to the WHO (1999), ADA (1997, 2003)***

Glucmetabolic category	Source	Classification criteria mmol/L (mg/dL)
Normal glucose regulation (NGR)	WHO ADA (1997) ADA (2003)	FPG <6,1 (110) + 2-h PG <7,8 (140) FPG <6,1 (110) FPG <5,6 (100)
Impaired fasting glucose (IFG)	WHO ADA (1997) ADA (2003)	FPG ≥6,1 (110) and <7,0 (126) + 2-h PG <7,8 (140) FPG ≥6,1 (110) and <7,0 (126) FPG ≥5,6 (100) and <7,0 (126)
Impaired glucose tolerance (IGT)	WHO	FPG <7,0 (126) + 2-h PG ≥7,8 and <11,1 (200)
Impaired glucose homeostasis (IGH)	WHO	IFG and IGT
Diabetes mellitus (DM)	WHO ADA (1997) ADA (2003)	FPG ≥7,0 (126) or 2-h PG ≥11,1 (200) FPG ≥7,0 (126) FPG ≥7,0 (126)

Values are expressed at venous plasma glucose.

FPG = fasting plasma glucose;

2-h PG = two-hour post-load plasma glucose (1 mmol/L= 18 mg/dL).

Additional examination

Clinical blood analysis reveals inflammatory changes (leukocytosis, neutrophilia in case of infections complication). Anemia may be detected in patients with renal failure.

Clinical urine analysis may identify the kidney function during examination of physical properties of the urine. Polyuria or oligyria, low specific urine gravity indicate to appearance of renal failure. Chemical study of urine includes assessment of glucose, ketone bodies and protein. Glucosuria is a specific sign of diabetes. Ketone bodies are determined in patients with decompensation and presence of ketoacidosis. Microalbuminuria and proteinuria indicate to development of diabetic nephropathy. Patients with diabetes should be screened for albuminuria. Given the insidious onset. INDDM patients should be screened for albuminuria at the time of the initial diagnosis. Patients with IDDM should be screened within 5 years of diagnosis.

Microalbuminuria is defined as a urinary albumin excretion (UAE) of 30–300 mg in a 24-hour collection period; albuminuria is defined as a UAE >300 mg/24 hours. The specific definition is depicted at table 6.4.

Table 6.4. ***Definition of albumin excretion rates (AERs)***

	Urinary AER (mg/24 hour)	Urinary AER (mg/minute)	Urinary albuminuria to creatinin ratio (mg/g)
Normal	<30	<20	<30
Microalbuminuria	30–300	20–200	30–300
Albuminuria	>300	>200	>300

Several screening methods are available:
- spot collection-albumin to creatinine ratio;
- 24-hour collection;
- 4-hour overnight collection microalbuminuria;
- spot urinary dipsticks (requires confirmation with other screening methods)

Albuminuria is the clinical hallmark of the development of nephropathy.

Biochemical blood analysis. According to modern determination of glucose should be performed in venous plasma for diagnostics of diabetes. The methods for conversion factors between plasma and other vehicles for glucose values depicted at table 6.5.

Table 6.5. ***Conversion factors between plasma and other vehicles for glucose values***

Plasma glucose (mmol/L) = 0,558 + 1,119 × whole blood glucose (mmol/L)
Plasma glucose (mmol/L) = 0,102 + 1,066 × capillary blood glucose (mmol/L)
Plasma glucose (mmol/L) = 0,137 + 1,047 × serum glucose (mmol/L)

Criteria of diabetes: fasting plasma glucose ≥7,0 mmol/L. A standardized oral glucose tolerance test (OGTT) performed in the morning, after an overnight fast (8–14 h); one blood sample should be taken before and one 120 min after intake of 75 g glucose dissolved in 250–300 mL water in a course of 5 min (note: timing of the test is from the beginning of the drink). Impaired glucose tolerance (IGT) can be recognized by the result of OGTT only: 2-h post-load plasma glucose (2hPG) ≥7,8 and <11,1 mmol/L (≥140 and <200mg/dL).

Special test for diagnostic of diabetes is glycated hemoglobin. Glycated hemoglobin (HbA_{1C}), a useful measure of metabolic control and the efficacy of glucose-lowering treatment, is a integrated summary of circadian blood glucose during the preceding 6–8 weeks, equivalent to the lifespan of erythrocytes. It provides a mean value but does not reveal any information on the extent and frequency of blood glucose excursions. HbA_{1C} has never been recommended as a diagnostic test for diabe-

tes. A primary reason is the lack of a standardized analytical method and therefore lack of a uniform, non-diabetic reference level between various laboratories. A high HbA_{1C} may only identify a fraction of asymptomatic people with diabetes. HbA_{1C} is insensitive in the low range and a normal HbA_{1C} cannot exclude the presence of diabetes or IGT.

Serum creatinin concentration increased in patients with diabetic nephropathy.

ECG is revealed the signs of ischemia (inverted T), arrhythmia (extrasystole).

Echo-CG – sign of left ventricular hypertrophy, decreased ejection fraction.

Renal biopsy in patient with end-stage renal disease should be directed at the detection of primary kidney disease or renal complication of diabetes.

Ophtalmoscopic examination of the fundus is required for evaluation of diabetic retinopathy.

Instrumental invasive examination of the coronary, kidney, peripheral arteries is required for detecting the macroangiopathy.

Contents

GENERAL PART

SPECIAL PART

Educational publication

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PROPEDEUTICS OF INTERNAL DISEASES

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