MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHIA STATE MEDICAL AND PHARMACEUTICAL UNIVERSITY
DEPARTMENT OF GENERAL PRACTICE – FAMILY MEDICINE
AND INTERNAL DISEASES

INTRODUCTION IN THE CLINIC OF INTERNAL DISEASES

MANUAL

for 2nd years students
speciality 221 «Dentistry»

Zaporizhzhia
2023

UDC 616.1/.4(075.8)
I-69

Recommended for publication by Central methodical Council
of Zaporizhzhia State Medical and Pharmaceutical University as a study guide
(Protocol № 2 of 25.05.2023)

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I-69 **Introduction in the clinic of internal diseases**: manual for the practical classes and individual work for 2nd year students of international faculty (speciality 221 «Dentistry») Discipline ««Propedeutics of internal medicine » / N. S. Mykhailovska, A. V. Grytsay, O.V. Shershnyova [et al.]. – Zaporizhzhia : ZSMPHU, 2023. – 132 p.

Manual compiled in accordance with the program of «Propedeutics of internal medicine». Guidelines are intended to help students prepare for practical classes and learn the material. Can be used for training of 2nd years students of international faculty, discipline «Propedeutics of internal medicine».

Введення в клініку внутрішніх хвороб: навчальний посібник до практичних занять та самостійної роботи студентів II курсу міжнародного факультету (спеціальність «Стоматологія») з дисципліни «Пропедевтика внутрішньої медицини» / Н. С. Михайловська, А. В. Грицай, І.О. Стецюк [та ін.]. – Запоріжжя : ЗДМФУ, 2023. – 132 с.

Навчальний посібник складений відповідно до програми «Пропедевтика внутрішньої медицини». Видання має на меті сприяти кращому засвоєнню теоретичних знань студентами під час підготовки до практичних занять та підсумкового контролю. Посібник рекомендований для використання студентами II курсу міжнародного факультету з дисципліни «Пропедевтика внутрішньої медицини».

UDC 616.1/.4(075.8)

CONTENT

PREFACE

The educational and profesional programme of students of the dentistry faculty of Ukrainian medical academies and universities should help forming abilities to distiguish the leading symptom or syndrom of either oral diseases or others life-treating conditions, to differentiate these conditions, to make the clinical diagnosis and to provide emergency. At the same time, preparing for the further professional activity the medical student have to acquire the skill set to fully assess each patient and knowledge of instrumental and physical methods for the human body examination.

This student's manual contents the specific educational material for the new thematic plan for the course of propeudeutics of internal diseases (the medical case history, main rules of the interview and examination of patients, the general inspection of the patient, physical and instrumental methods of examination of the broncho-pulmonary, cardiovascular system, laboratory and instrumental tests etc.). Several units are out of this programme for the purose to highlight the current level of the medical science, especially therapy. Such approach will help to use this manual for the individual mastering and for the further practical activity. Furthermore, will prepare students for the face-to-face contacts with emergency.

This manual is published for the first time.

The cover image was downloaded from website https://vitalehastanesi.com.

This student's manual provides theoretical material which would improve theoretical knowledge of students of the 2nd international fculty, speciality "Dentistry".

ABBREVIATIONS

ACS – acute coronary syndrome
AF – atrial fi brillation
AH – arterial hypertension
AHA – American Heart Association
ALS – advanced life support
ALT – alanine transaminase or alanine aminotransferase
AST – aspartate transaminase or aspartate aminotransferase
AV – atrioventricular
BMI – body mass index
BLS – basic life support
BP – blood pressure
CBC – complete blood count
CK – creatine phosphokinase
CK–MB – creatine phosphokinase MB isoenzyme
COPD – chronic obstructive pulmonary disease
CPR – cardiopulmonary resuscitation
CT – computed tomography
CVS – cardiovascular system
CXR – chest X-ray
ECG – electrocardiography
ESR – erythrocyte sedimentation rate
ESRD – end-stage renal disease
GFR – glomerular fi ltration rate
GI – gastrointestinal
Hb – hemoglobin
HDL – high density lipoprotein
HF – heart failure
HR – heart rate
IHD – ischemic heart disease
IM – intramuscular
ISH – International Society of Hypertension
IV – intravenous
JVP – jugular venous pressure
LDL – low density lipoprotein
LDH – lactate dehydrogenase
LVH – left ventricular hypertrophy
MI – myocardial infarction
MRI – magnetic resonance imaging
NMU – National Medical University
OSCE – Objective Structured Clinical Exam
PAC – premature atrial contraction
PE – physical examination
PVC – premature ventricular contraction
RBC – red blood cells or erythrocytes
SA – sinoatrial
SC – subcutaneous
SVT – supraventricular tachycardia
UTI – urinary tract infection
VF – ventricular fi brillation
WBC – white blood cells or leucocytes
WHO – World Health Organization

INTRODUCTION IN THE CLINIC OF INTERNAL DISEASES

INTRODUCTION IN THE CLINIC OF INTERNAL DISEASES. MAIN RULES OF THE INTERVIEW AND EXAMINATION OF PATIENTS. THE BASIC METHODS OF THE EXAMINATION OF PATIENTS. THE SCHEME OF THE EXAMINATION OF PATIENTS. THE GENERAL INSPECTION OF THE PATIENT. THE INSPECTION OF SEPARATE PARTS OF A BODY

The medical case history includes several parts, such as:

I Passport part
II Complaints
III Interrogation of systems
IV Anamnesis of disease
V Anamnesis of life
VI Objective examination
VII Suppositional diagnosis
VIII Facts of additional investigation
IX Terminal clinical diagnosis
X Treatment
XI The dairy observation
XII Epicrisis

I. General biographical particulars (passport part)

1. Surname, name, middle name
2. Age, date of birth
3. Sex
4. Residence address
5. Place of employment
6. Occupation
7. The date of admission to the hospital (day, month, year, hours with minutes)
8. The date of discharge from hospital
9. Number of days spent in the hospital

II. The Present Complaints

The examination begins with an interview. The patient tells his complaints which often are of no less importance than a thorough objective examination of the patient. Some diseases are diagnosed almost exclusively by the patient's complaints. Angina pectoris for example, is frequently diagnosed almost entirely from the character of pain in the region of the heart. Cholelithiasis is diagnosed by attacks of pain in the right upper abdominal quadrant.

The reliability of complaints related by patients varies. Some patients forget to mention the most important symptoms while others tell on unimportant and irrelevant details.

The main complaints of the patient should first be determined. If the patient complains of retrosternal pain, the character and exact location of this pain, its focus and intensity should be determined; the time of the onset, and possible causes that provoked the pain (strain, cough, taking food, etc.) should be established. The patient should be asked which remedies remove this pain. Other complaints should also be analysed. In pneumonia, for example, the patient would normally complain of weakness, high temperature, side pain (pleurodynia), and cough; he would note that the onset of the disease was marked a few days ago by a sudden chill and pricking in the side when coughing and breathing deeply.

The study of the main complaints can often lead the examiner to a conclusion concerning the general character of the disease, e.g. high body temperature would normally indicate an infectious process, cough and expectorated sputum indicate possible disease of the lungs. Knowledge of the exact time of the onset of the disease is informative of the character of the disease (acute or chronic).

The inquiry should not be limited to these main points. So as not to omit any symptoms and determine the functional condition of all the organs (status functionalis) the patient should be questioned according to a specially outlined scheme. Changes in the patient's general state should be established (loss of weight, fever, weakness, edema, headache). The condition of the respiratory system (cough, expectoration of sputum and blood, pain in the throat) should also be established. Next is the car-diovascular system (tachycardia, dyspnoea, heart pain, swelling of

feet). Then follows the gastro-intestinal system (appetite, swallowing, vomiting, epigastric pain, etc.).

The condition of the nervous system is established by asking the patient about his subjective condition, his sleep, irritability or indifferent attitude to the surroundings, weakness, excitement, headache, state of consciousness and the main senses. The patient should be asked about his conduct, responses to external stimuli, his attitude to work and his associates. This is necessary to establish the special properties of his higher nervous activity at the present time and in the past, and the type of his nervous system according to Pavlov's classification. The inquiry at this stage gives the physician information concerning the condition of various organs and systems of the patient (respiration, blood circulation, digestion, urinary function, motor function, nervous system, etc.).

III. Interrogation of systems

a) General state of the patient: weakness, temperature rise, chills, edemas (localization, persistency, causes of appearance). General and local itch, hemorrhages, rashes, ulcers; changes in organs of locomotor system: joint pains, restriction of movement, joint deformation.

b) Respiratory Organs

Breathing through the nose (free, difficult). Rhinorrhea.

Nasal bleeding

State of olfactory function (distinguish smells)

Nasopharynx: pain on swallowing

Larynx: change of voice, aphonia

Cough: dry, productive, intensity, time appearance; periodical or persistent

Sputum: quantity, character, color and odor

Hemoptysis: frequency,

Breathlessness (dysphnea): inspiratory, expiratory, mixed; patient’s position during dyspnea (on the side, orthopnea)

Asphyxia – time of appearance, duration, reversal

Chest pain: localization, character (acute, dull etc.), and connection with respiration, with cough

c) Cardiovascular system

Palpitation: while walking, at rest, when exited, at paroxysmal event; accompanied with vertigo, dropped heart beating, pain.

Pain near the heart: time of appearance, intensity, character (piercing, dull pressing etc.), duration, irradiation. Suggested reasons of pain (psycho-emotional loads, exercise stress, food intake, reversal), relieving factors.

d) Digestive organs

Dry mouth. Sialorrhea. Abnormal taste (metal, bitter). Appetite. Swallowing: free, difficult.

Dyspeptic phenomenon: regurgitation (gaseous, sour, musty smelling). Heartburn. Nausea: on an empty stomach or after meals. Vomit: a) poor with mucus, b) abundant with pieces of food, c) coffee-grounds, d) artificially induced in order to relieve pain.

Heaviness and pain in epigastric area, intensity, character of pain, periodicity (night pain, season pain), and connection with the quantity and quality of food, food intake time and position of the body. Irradiation of pain into spinal column, scapulae, shoulders etc.

Enlarged abdomen, feeling of heaviness, abdominal murmur, pain (persistent, spasmodic). Stools – constipation, diarrhea, pain on defecation and tenesmus. Color of the fecal matter (usual, too light, dark, tarry), admixture of mucus and blood in fecal masses. Discharge of parasites. Anal pruritus. Piles, anus bleeding.

e) Liver

Pain on the right hypochondrium, their character: paroxysmal, their intensity, irradiation, accompanied with jaundice, temperature rise, chills, nausea, vomit. Influence of food on the beginning and intensity of pains. Pains on the left hypochondrium.

f) Urogenital system

Low back pain (persistent or paroxysmal). Frequency and duration, radiation into femur and genitals. Low back pain accompanied with rise in temperature, chills, nausea, and vomit. Daily diuresis, polyuria, oliguria, anuria, nocturia, pollakiuria, hematuria, pyuria.

Sexual potency (men). Menstrual function (women). Urinary bladder – feeling of heaviness and pain over the pubis, painful and difficult urination. Pain and burning in urethra. Discharge from urethra.

g) Nervous system

Irritability, apathy. Capacity for mental work, memory, sleep. Headache: localization, duration. Epiphenomena: nausea, vomiting, dizziness, flickering in front of eyes, tinnitus.

h) Organs of sense

Vision, hearing.

So, the **main complaints** (list them) **and complementary complaints** (list in the order of revelation during the questioning on systems).

IV. Anamnesis of the Disease

Exact answers should be obtained from the patient concerning the following aspects of his present disease (anamnesis morbi): (1) the time of the onset of the disease; (2) the character of the first symptoms; (3) the course of the disease; (4) examinations and their results, if any; (5) treatment, if any, and its efficacy. The answers to these questions may give the physician the necessary information on the present disease.

The history of the disease should include information concerning the onset of the disease and its development until the present. The patient's general condition before the disease should first be determined and the causes that might have provoked the disease established wherever possible. The patient should be questioned in detail about the first signs of the disease and the chronology of their development (dynamics), about relapses or exacerbations, remissions and their duration. If the patient was examined during an exacerbation of the disease by some other physician, the results should be studied. Excess verbosity of the patient should be prevented, because the results of the examinations and treatment only are important (therapy with cardiac glycosides, vasodilators, diuretics, antibiotics, hormones, etc.). Motives for hospitalization should also be determined (exacerbation of the disease, verification of the diagnosis, etc.).

V. Anamnesis of Life

The past history is often very important for establishing the character, the cause, and conditions for the onset of the disease. Anamnesis vitae is a history or a medical biography of the patient in every period of his life (infancy, childhood, adolescence, and maturity).

Collecting the anamnesis begins with the general biographical information. Birth place is important, because some diseases (e.g. endemic goiter) usually predominate in one locality and are not met in others. The age of he parents is also important. The patient should be asked if he was born at term, if there were other children in the family, if he was breast fed or artificially; the age at which the patient began walking and talking is important, and the patient should inform the physician if he had marred signs of rickets during his childhood. This information is important to evaluate the patient's health at birth and during childhood. Conditions of life in childhood and adolescence and health during these periods of life are im-portant information. It is necessary to find out if the patient's physical and mental development was retarded and what was his progress at school. The time of sex maturity should be determined. Women should report the number of pregnancies and parturitions, and the course of labor.

Social conditions are important, for the health of people. The patient should inform the physician on the conditions of his housing (separate apartment, hostel, country house, illumination, the presence of dampness, if any, hygienic conditions, etc.). The composition of the family is important: large or small family, their health, well-being, income etc. Malnutrition is an important factor for the onset of some diseases. The patient should be asked if his diet is sufficiently rich in vegetables. fruits, etc. The way in which the patient spends his leisure time is also important. The patient should report on the time he sleeps, rests, walks in the fresh air, and what sports and exercises he goes in for.

Unfavorable labor conditions and industrial hazards (some harmful dusts) are important, for they may cause bronchial asthma and chronic diseases of the bronchi and lungs. Strong noise, vibration, high ambient temperature, drafts, and cold (work in the open) can cause pathology. Industrial poisoning by mercury, lead, carbon monoxide and other harmful agents, and also exposure to radiation (improper safety measures) may also cause disease. The working schedule is also important.

Establishing whether there are unfavorable industrial factors helps the physician give recommendations for organization of the patient's work.

Past illnesses are also important. Some infectious diseases, such as measles or scarlet fever, do not recur because of acquired immunity, while other diseases, such as rheumatism or erysipelas, tend to recur. Rheumatism or diphtheria often provoke heart diseases. Nephropathy often develops after scarlet fever, and incompetence of heart valves often results from the previous endocarditis.

It should be remembered that the patient may not know about his past diseases. Therefore in dubious cases the physician should ask the patient whether he had certain symptoms by which a suspected past disease might manifest itself (e.g. prolonged fevers, swelling of and pain in the joints are characteristic of rheumatism, general edema indicates kidney disease, attacks of right hypochondriac pain may be the cause of the gall bladder disease, etc.). Contacts with infectious patients are important, especially in the presence of epidemics (e.g. influenza).

Family history. Health of the parents, sisters or brothers is often informative. If some of the family have had tuberculosis, the other members of the family may also develop tuberculosis. Syphilis may be transmitted by an intrauterine route. By comparing the pathology of the patient with diseases of his relatives, the physician can make a conclusion on the role of hereditary factors in the development or origin of the disease.

Life of man is tightly connected with the environment, and pathology always depends on external effects. Harmful environmental factors may affect the patient's offspring: his children may be predisposed to some diseases. But this predisposition does not obligatory provoke the disease. Special conditions are usually required for the disease to develop, and if these special factors are absent the person will not develop the disease. Moreover, if conditions favour, the person may strengthen his health and eradicate the hereditary predisposition to an illness.

Hereditary or familial (genotypical) and non-hereditary (paratypical) diseases are distinguished. But this classification is only conventional. As genetics progresses it becomes more obvious that some diseases that would be considered to be resistant to the hereditary factors, are actually genotypical diseases. Internists mainly deal with diseases that are not usually transmitted to the offspring but merely predispose them

to these diseases (e.g. essential hypertension, atherosclerosis, cholelithiasis, etc.). Under certain environmental conditions this predisposition may enable the person to develop the disease. It should be remembered that the inherited character may have varying expressivity, or hereditary disease may develop n one member of the family only, or it may be inherited by an offspring after several generations, or it may develop only in family members of one sex (e.g. only males develop hemophilia which is transmitted from a grandfather to a grandson through a healthy daughter). The onset of certain hereditary diseases is sometimes erroneously attributed to an external factor, which was actually only the stimulus that provoked the disease.

In order to establish the hereditary character of a disease, the familial factors should be first given a thorough clinical-genealogical analysis. For the sake of convenience, genealogical schemes should be made out, using he special conventional symbols.

According to the adopted terminology, the patient is called a proband. His brothers and sisters are given in the order of their birth, from left to right. The Roman numerals are used to designate (at the left) successive generations. Each member of the generation is designated by an Arabic numeral. Symbols designating the proband's relatives, who were affected by the same disease, are shaded. The diagram must include data concerning he disease occurring in both parental lines of the proband.

Three main types of inheritance have been established. The first, autosomal-dominant type, is the most prevalent. It is characterized by full penetrance of the mutant gene. In this type of inheritance the disease is directly transmitted from both parents to their offspring with 50 per cent of both sexes being affected. Those who do not inherit the mutant gene have normal offspring. If penetrance of the mutant gene is incomplete, the direct order of inheritance is more difficult to detect. Suspicion that an inherited disease is dominantly transmitted can be verified by analysis of the offspring from repeated marriages (affected children from each marriage). The direct order of inheritance occurs, for example, in subjects with anatomical abnormalities (of the internal organs included).

Heterozygote carriage of the recessive gene in autosomal-recessive inheritance does not cause the disease, which only develops in homozygote carriers. A blood

relationship between parents is often revealed in recessive inheritance (otherwise difficult to reveal). Many enzymopathies, certain diseases of the nervous system, etc. are often inherited by the autosomal-recessive type.

VI. Objective examination of a patient (Status praesens)

General state of the patient: satisfactory, moderate grave, grave, extremely grave.

Consciousness: clear, dull, lost.

Patient's posture: active, passive, forced.

Expression of the face: impartial, restless, frightened, dull, exhausted, face of Hyppocratica, distressed, sardonic smile.

Eyes: eye-slit state, exophtalmos, enophtalmos, eyelid ptosis, pupillary constriction (miosis) or pupillary dilation (mydriasis), pupillary irregularity disproportion (anisocoria). Horner's syndrome.

Height, weight, body constitution: strong or weak. Constitutional type: normosthenic, asthenic, hypersthenic. BMI. Nutritional state: moderate, reduced and excessive. Cachexia. Obesity (I – IV degree).

Skin and visible mucosal membranes: dry, moist (sweating); color – normal, pale, red, bile-tinged (icteric, subicteric), cyanotic (acrocyanosis, diffusive cyanosis). Pigmentation disturbance (depigmentation, hyperpigmentation). Rashes, hemorrhages, scars. Bedsores. Skin elasticity (turgor), hair, nails.

Subcutaneous fat: the degree of its development.

Edemas: spreading, localization, level of intensity, subcutaneous edema (anasarca).

Lymphatic system: submandibular, cervical, occipital, supra- and subclavian, axillary, inguinal, femoral lymph nodes. Size, consistency, cohesion with subjacent tissues, painfulness, skin colour over lymphatic nodes.

Muscular system: muscular system state (good, moderate, weak), muscular tonus (increased, normal and lowered), induration, hypertrophies, atrophies, muscular pain (independent, painful movements, pain on palpation).

Bone system: thickening of periosteum and bones, changes in value and form of bones, pain in bones.

Backbone: lordosis, kyphosis, skoliosis, kyphoskoliosis.

Joints: size, form (fusiform, nodular), mobility (active, passive). Joints immobility (ankylosis). Painful palpation and painful movements. Crunching. Compositional fluctuation (shaking). Color of skin and temperature round joints. “Drum-stick” fingers.

Respiratory system

Shape of the chest: cylindrical, flat, paralytic, emphysematous (barrel) etc.; symmetry of half-chests; state of supra- and subclavicular fossae; state of intercostal spaces (intercostal retraction, transdural herniation, intercostal spaces width); scapulas position. Chest circumference (cm) on normal breathing, on deep inhalation and exhalation. Respiratory movements symmetry, respiration rate, rhythm and type of respiration (thoracic, abdominal, mixed, Cheyne-Stokes, Biot’s, Kussmaul’s, Grocco’s).

Chest palpation: painfulness ribs and intercostal spaces, vocal fremitus (normal, intensified, weakened, precise localization of its changes). Elasticity of the chest.

Percussion of lungs. Comparative percussion: determination of percussion sound quality (clear pulmonary, dull, tympanic). Localization of percussion sound changes.

Topographic percussion of lungs: determination of apexes height from the front and behind, Krenig’s areas width from the right and from the left, lower borders of lungs along all vertical lines. Determination of respiratory mobility of lungs’ lower edge (excursion of lungs).

Auscultation of lungs: types of respiration (vesicular - normal, puerile, diminished, strengthened, harsh respiration, interrupted; bronchial - normal or pathological: amphoric, metallic and stenotic; mixed or undefined). Rales: dry (sibilant (weezes) or sonorous,), moist (fine, medium and coarse bubbling rales: consonating and unconsonating), crepitation. Pleural friction rub, splashing sound by Hippocrates, falling drop sound. Point out the localization of revealed changes. Bronchophony.

Cardiovascular system

Examination of heart region and big vessels: protrusion of the heart region (cardiac hump). Systolic chest retraction. Epigastric pulsation. Pulsation in the second

intercostal space, pulsation in the jugular fossa, pulsation of carotids and other arteries (temporal, brachial). Capillary pulse. Swelling of jugular veins and their pulsation, positive venous pulse.

Palpation: apex beat (location, area, height, strength or resistance), cardiac beat. “Cat’s purr” (systolic, diastolic, localization).

Percussion: determination of relative and absolute heart dullness borders. Determination of vascular fascicle width.

Auscultation: heart rate and rhythm (regular, irregular), respiratory arrhythmia, ectopic beats, atrial fibrillation).

Heart sounds: loudness of the both heart sounds, characteristic of heart sounds in places of typical listening: *heart apex* - S1 (normal, loud or soft), flapping of the I heart sound, cannon sound of N. D. Strazhesko, *heart base* - accentuated S2 over aorta or pulmonary artery; splitting and reduplication of the heart sounds (gallop rhythm - presystolic, systolic or protodiastolic).

Cardiac murmurs (systolic, diastolic and their variants - presystolic, protodiastolic, mesodiastolic). Places of the best listening of cardiac murmurs, murmur conducting. Friction rub. Pleuropericardial murmur. Cardiopulmonary murmur.

Auscultation of carotids, femoral arteries, phenomenon of Traube – Vinogradov – Durozye. Auscultation of cervical veins (whipping - top sound).

Pulse: rate, rhythm, filling, tension, value, form. Pulse asymmetry; absence of pulse (Takayasus disease), deficiency of pulse.

Blood pressure measurement in the both brachial arteries.

Digestive system

Examination of oral cavity: bad smell from the mouth, colour of lips and visible mucous membranes, fissures in the mouth angles, inflammatory erythema, cyanosis, pigmentation, gingivae edge (when poisoning with bismuth, lead etc.).

State of teeth: carious teeth, absence of teeth, artificial teeth.

Tongue: humid, dry, coated, pale, brightly red, cyanotic. Lingual papilla atrophy (Addison – Biermer disease). Tongue aphthae.

Fauces and throat: paleness, hyperemia, dryness, pathologic spots. Tonsils: enlarged, presence of scars, purulent plugs in crypts, spots on the tonsils, their colour, easiness of tearing away.

Examination of abdomen: shape, symmetry, participation in breathing, abdominal swelling in hypogastric area and retraction in epigastrial area at the same time. Venous dilation of cutaneous veins. Visible stomach and intestinal peristalsis. State of umbilicus: inverted, thrown out, smoothed. Postoperative scars on the abdominal wall. Hernias (omphalocele, incisional, inguinal, femoral).

Palpation of abdomen: superficial palpation: determination of abdominal wall's rigidity, tenderness (defuse, local), Shchetkin – Blumberg's peritoneal syndrome. Determination of ascite by the fluctuation method.

Deep, sliding, methodical, systematic, topographic palpation according to the V. P. Obraztov-N. D. Strazhesko begins with sigmoid colon palpation (localization, mobility, tenderness, consistency, diameter, the condition of the surface, the absence or presence of rumbling sounds), ascending colon, then caecum is palpated in order to determine of its state, descending colon, afterwards the greater curvature of stomach and transverse colon are palpated.

Liver: determination of the liver low edge character (it can be soft, firm, rough, sharp, rounded, tender, etc.). Determination of sizes by percussion according to Kurlov. Special examination of gallbladder, its tenderness, enlargement. Courvoisier's symptom, phrenicus – symptom.

Spleen: location, consistency and tenderness.

Kidneys and urinary bladder: bimanual palpation of kidneys (nephroptosis, painfulness, tuberosity). Pasternatsky's symptom. Painfulness along ureter. Examination of suprapubic area (painfulness, swelling).

VII. Tentative (provisional) diagnosis (Scheme of substantiation of a diagnosis)

The patient complains of … (detailed description of patient's complaints).

Anamnesis of the present disease. The patient considers himself to be ill since….. when…(briefly state the main stages of the disease, which confirm the diagnosis: beginning, symptoms, the course of the disease, the cause of the disease etc.).

Anamnesis of life (anamnesis vitae). Describe only those diseases and unfavourable factors, which might contribute to the development of the present disease. For example, old anginas provoke the development of kidney diseases, rheumatism; alcohol and tobacco addiction provoke ulcerous disease.

Objective data: the results of examination, palpation, percussion, auscultation, which confirm the diagnosis.

On the basis of the above-stated it may be considered that the patient has … (formulate the full diagnosis)

VIII. Additional investigations data

Blood, urine, and stool analyses. Biochemical blood investigation data (acute phase reactants: fibrinogen, C–reactive protein, albuminous blood fractions, antistreptolysin-O, antistreptohyaluronidase, antistreptokinase etc., liver functional tests: thymol, sublimate, blood bilirubin etc., blood ferments).

Instrumental methods of the investigation: ECG, phonocardiogram, ultrasonic investigation of the heart, organs of the abdominal cavity, X-ray investigation and etc. Professional advice.

IX. Final clinical diagnosis **(The example of substantiation)**

On the basis of patient's complaints of pain near the heart, heartbeating, short of breath while walking, shin edema in the evening, subfebrile temperature in the evenings, asthenia, undue fatiguability.

On the basis of anamnesis: the patient fell ill 6 years ago after he had had the angina. The patient had subfebrile temperature during a month, heartbeating especially at night, pain in the area of heart, dyspnea on exertion, hyperhidrosis, undue fatiguability. Almost annual relapses of the disease in autumn and spring. These exacerbations are nasopharyngeal infections-dependant. The last impairment appeared after acute pharyngitis. Frequent anginas in anamnesis.

On the basis of physical examination data: paleness of skin, acrocyanosis, leg edemata, sour smell of the patient's sweat, swelling of cervical veins, the apex beat displacement to the left and downwards, minor dilation of absolute heart dullness borders to all sides, weakening of the first apical heart sound, accent of the second sound over the pulmonary artery, systolic apical cardiac murmur and it

radiates to the left axilla, tachycardia up to 100 beats per minute, enlarged liver which is 3 cm protruded from hypochondrium.

On the basis of additional investigations data: in blood bulk analysis – leukocytosis, increase of ESR up to 24 mm/hour, positive rheumatism activity immune-biochemical test (antistreptococcal antibodies and acute stage indices (DPA, sialic acids, seromucoid, C-reactive protein, hyperfibrinogenemia, dysproteinemia with protein coefficient decrease up to 1,1); chest X-ray – mitral configurated heart; ECG – intra-auricular block, signs of the left ventricle and left atrium overload and hyperthrophia, non-defined signs of the right ventricle hyperthrophia; PCG – decrease of the first apical heart sound amplitude, increase of amplitude of the second sound over the pulmonary artery, the systolic noise of different frequencies and different amplitudes on the apex and in "O" point beginning with the I sound, the third sound is registered with some increase of amplitude.

Final clinical diagnosis can be formulated: chronic rheumatic disease, in active phase, second degree of activity, carditis, mitral valve incompetence, heart failure of III functional class (NYHA).

X. Treatment

The prescribed treatment: the regime, the diet, drugs and other applied methods of treatment.

XI. Observation diary

The diary reflects the patient's state in dynamics taking into consideration the effectiveness of the applied treatment.

XII. Epicrisis

Epicrisis includes short summary of anamnesis, objective examination data, clinical diagnosis of the disease with its substantiation and the most important additional methods of the investigation, which confirm the diagnosis. The applied treatment and its effectiveness (the improvement, impairment, without any changes) should be indicated.

Epicrisis is concluded with recommendations for the patient (regime, diet, treatment, including a sanitary – resort one and etc.).

GENERAL INSPECTION OF THE PATIENT. INSPECTION OF SEPARATE PARTS OF A BODY

Inspection technique. The body should be inspected by successively uncovering the patient and examining him in direct and side light. The trunk and the chest are better examined when the patient is in a vertical posture. When the abdomen is examined, the patient may be either in the erect (upright) or supine (dorsal) position. The examination should be carried out according to a special plan, since the physician can miss important signs that otherwise could give a clue for the diagnosis (e.g. liver palm or spider angiomata which are characteristic of cirrhosis of the liver).

The appearance of the patient is described: general condition, consciousness, carriage, gait, position in bed, expression of the face, constitution, stature, skin, visible mucosa, and conjunctiva, subcutaneous fat, edemas, muscles, bones, joints, lymph nodes.

The general condition of the patient is characterized by the following signs: consciousness and the psyche, posture and body-built.

Consciousness. It can be clear or deranged. Depending on the degree of disorder, the following psychic states are differentiated.

1.**Stupor.** The patient cannot orient himself to the surroundings, he gives delayed answers. The state is characteristic of contusion and in some cases poisoning.

2.**Sopor**. This is an unusually deep sleep from which the patient recovers only for short periods of time when called loudly, or roused by an external stimulus. The reflexes are preserved. The state can be observed some infectious diseases and at the initial stage of acute uremia.

3.**Coma.** The comatose state is the full loss of consciousness with complete absence of response to external stimuli, with the absence of reflexes, and deranged vital functions. The causes of coma are quite varied but the loss of consciousness in a coma of any etiology is connected with the cerebral cortex dysfunction caused by some factors, among which the most important are disordered cerebral circulation and anoxia. Edema of the brain and its membranes, increased intracranial pressure, effect of toxic substances on the

brain tissue, metabolic and hormone disorders, and also upset acid-base equilibrium are also very important for the onset of coma. Coma may occur suddenly or develop gradually, through various stages of consciousness disorders. The period that precedes the onset of a complete coma is called the precomatose state. The following forms of coma are most common.

Alcoholic coma. The face is cyanotic, the pupils are dilated, the respiration shallow, the pulse low and accelerated, the arterial pressure is low; the patient has alcohol on his breath.

Apoplexic coma (due to cerebral haemorrhage). The face is red, breathing is slow, deep, noisy, the pulse is full and rare.

Hypoglycaemic coma can develop during insulin therapy for diabetes.

Diabetic (hyperglycemic) coma occurs in non-treated diabetes mellitus.

Hepatic coma develops in acute and subacute dystrophy and necrosis of the liver parenchyma, and at the final stage of liver cirrhosis.

Uremic coma develops in acute toxic and terminal stages of various chronic diseases of the kidneys.

Epileptic coma. The face is cyanotic, there are clonic and tonic convulsions, the tongue is bitten. Uncontrolled urination and defecation. The pulse is frequent, the eye-balls are moved aside, the pupils are dilated, breathing is hoarse.

4. **Irritative disorders of consciousness** may also develop. These are characterized by excitation of the central nervous system in the form of hallucinations, delirium (delirium furibundum due to alcoholism; in pneumonia, especially in alcoholics; quiet delirium in typhus, etc.).

General inspection can also give information on other psychic disorders that may occur in the patient (depression, apathy).

Attention should be paid to *gait* and *carriage*. In healthy person the carriage is straight, the gait is steady. An unsteady gate is observed in blood loss, nervous breakdown, high fever, cerebellar tumors, etc. A specific gait can be seen in some diseases. For example, in patients with ascites the carriage is proud, the upper part of the body is reclined, the abdomen is jutted out. In hemiplegia (paralysis of one half of the body) or paralysis of one lower extremity, the patient draws the leg making circular movements to the front and inside. In tabes dorsalis the gait is

ataxic. The extremity is thrown out forward, having put it down the patient continues to feel for a stable support. In coccitis (inflammation of the coxofemoral joint) the leg is thrown out forward with the movement of the whole pelvis without bending in the hip joint. Similar gait is sometimes observed in lumbosacral radiculitis.

The posture or attitude of the patient is often indicative of his general tone, the degree of muscle development, and sometimes of his occupation and habits. Most patients with grave diseases or with psychic depression are often stooped. Erect posture, easy gait, and free and unconstrained movements indicate the normal condition of the body.

Position of the patient. It can be active, passive, or forced.

1) The patient is *active* if the disease is relatively mild or at the initial stage of a grave disease. The patient readily changes his posture depending on circumstances. But it should be remembered that excessively sensitive or alert patients would often lie in bed without prescription of the physician.

2) *Passive posture* is observed with unconscious patients or in rare cases, with extreme asthenia. The patient is motionless, his head and the limbs hang down by gravity, the body slips down from the pillows to the foot end of the bed.

3) *Forced posture* is often assumed by the patient to relieve or remove pain, cough, dyspnoea. For example, the sitting position relieves orthopnoea: dyspnoea becomes less aggravating in cases with circulatory insufficiency. The relief that the patient feels is associated with the decreased volume of circulating blood hi the sitting position (some blood remains in the lower limbs and the cerebral circulation is thus improved). Patients with dry pleurisy, lung abscess, or bronchiectasis prefer to lie on the affected side. Pain relief in dry pleurisy can be explained by the limited movement of the pleural layers when the patient lies on the affected side. If a patient with lung abscess or bronchiectasis lies on the healthy side, coughing intensifies because the intracavitary contents penetrate the bronchial tree. And quite the reverse, the patient cannot lie on the affected side if the ribs are fractured because pain intensifies if the affected side is pressed against the bed. The patient with cerebrospinal meningitis would usually lie on his side with his head thrown back and the thighs and legs flexed on the abdomen. Patients with angina pectoris and intermittent claudication prefer to stand upright. The

patient is also erect (standing or sitting) during attacks of bronchial asthma. He would lean against the edge of the table or the chair back, with the upper part of the body slightly inclined forward. Auxiliary respiratory muscles are more active in this posture. The supine posture is characteristic of strong pain in the abdomen (acute appendicitis, perforated ulcer of the stomach or duodenum). The prone position (lying with the face down) is characteristic of patients with tumors of the pancreas and gastric ulcer (if the posterior wall of the stomach is affected). Pressure of the pancreas on the solar plexus is lessened in this posture.

The concept of habitus includes the body-build, i.e. constitution, height, and body weight.

Constitution (L *constituero* to set up) is the combination of functional and morphological bodily features that are based on the inherited and acquired properties, and that account for the body response to endo- and exogenic factors. The classification differentiates between the following three main constitutional types: asthenic, hypersthenic, and normosthenic.

The *asthenic* constitution is characterized by a considerable predominance of the longitudinal over the transverse dimensions of the body by the dominance of the limbs over the trunk, of the chest over the abdomen. The heart and the parenchymatous organs are relatively small, the lungs are elongated, the intestine is short, the mesenterium long, and the diaphragm is low. Arterial pressure is lower than in hypersthenics; the vital capacity of the lungs is greater, the secretion and peristalsis of the stomach, and also the absorptive power of the stomach and intestine are decreased; the hemoglobin and red blood cells counts, the level of cholesterol, calcium, uric acid, and sugar in the blood are also decreased. Adrenal and sexual functions are often decreased along with thyroid and pituitary hyperfunction.

The *hypersthenic constitution* is characterized by the relative predominance of the transverse over the longitudinal dimensions of the body (compared with the normosthenic constitution). The trunk is relatively long, the limbs are short, the abdomen is large, the diaphragm stands high. All internal organs except the lungs are larger than those in asthenics. The intestine is longer, the walls are thicker, and the capacity of the intestine is larger. The arterial pressure is higher; haemoglobin and red blood cell count and the content of cholesterol are also higher; hypermobility and hypersecretion of the stomach are more normal. The secretory and the absorptive function of the intestine are high. Thyroid hypofunction is common, while the function of the sex and adrenal glands is slightly increased.

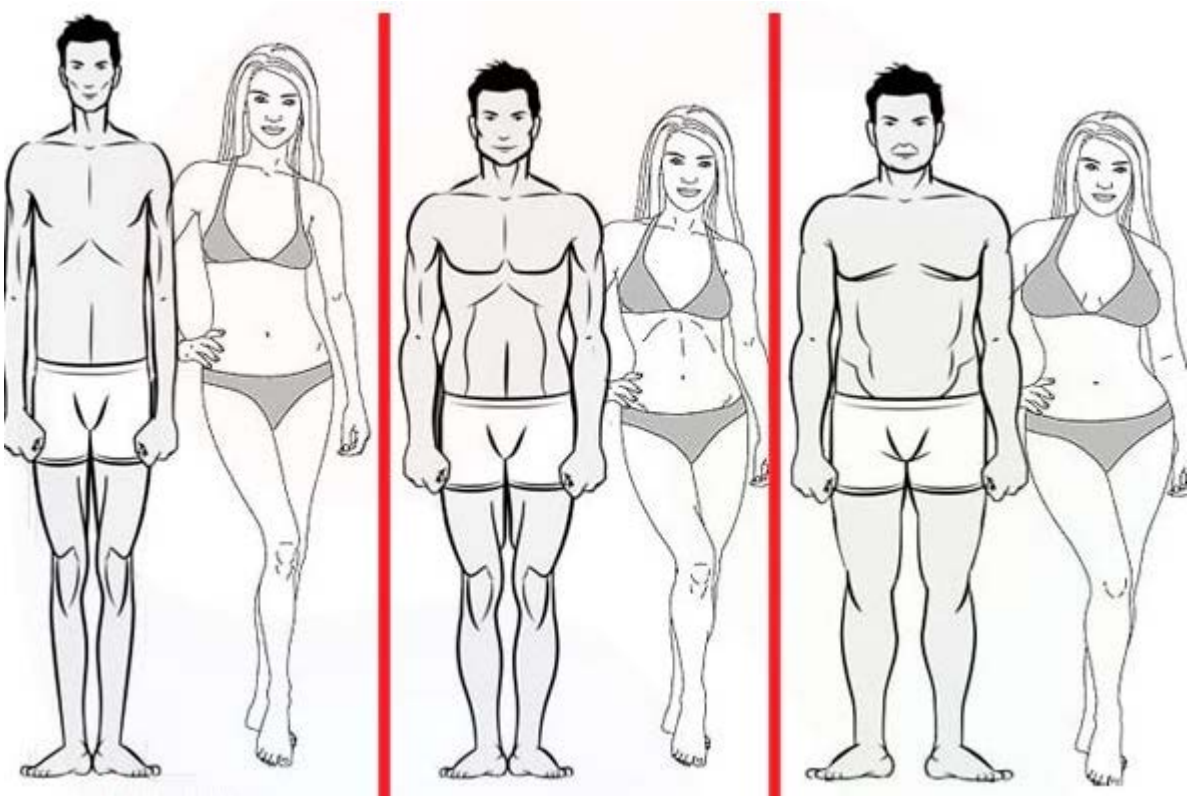

Image 1. Constitution of the body
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Normosthenic constitution is characterized by a well proportioned make-up of the body and is intermediate between the asthenic and hypersthenic constitutions.

The colour, elasticity, and moisture of the skin, eruptions and scars are important.

The *colour of the skin* depends on the blood filling of cutaneous vessels, the amount and quality of pigment, and on the thickness and translucency of the skin. Pallid skin is connected with insufficiency of blood circulation in the skin vessels due to their spasms of various etiology or acute bleeding, accumulation of blood in dilated vessels of the abdominal cavity in collapse, and in anemia. In certain forms of anemia, the skin is specifically pallid: with a characteristic yellowish tint in Addison-Biermer anemia, with a greenish tint in chlorosis, earth-like in malignant anemia, brown or ash-coloured in malaria, cafe au lair in subacute septic endocarditis. Pallid skin can

also be due to its low translucency and considerable thickness; this is only apparent anemia, and can be observed in healthy subjects.

Red colour of the skin can be transient in fever or excess exposure to heat; persistent redness of the skin can occur in subjects who are permanently exposed to high temperatures, and also in erythraemia. Cyanotic skin can be due to hypoxia in circulatory insufficiency, in chronic pulmonary diseases, etc. Yellowish colour of the skin and mucosa can be due to upset secretion of bilirubin by the liver or due to increased haemolysis. Dark red or brown skin is characteristic of adrenal insufficiency. Hyperpigmentation of the breast nipples and the areola, in women, pigmented patches on the face and the white line on the abdomen are signs of pregnancy. When silver preparations are taken for a long time, the skirl becomes grey on the open parts of the body (argyria). Foci of depigmentation of the skin (vitiligo) also occur.

The skin can be wrinkled due to the loss of elasticity in old age, in prolonged debilitating diseases and in excessive loss of water.

Elasticity and turgor of the skin can be determined by pressing a fold of skin (usually on the abdomen or the extensor surface of the arm) between the thumb and the forefinger. The fold disappears quickly on normal skin when the pressure is released while in cases with decreased turgor, the fold persists for a long period of time.

Moist skin and excess perspiration are observed in drop of temperature in patients recovering from fever and also in some diseases such as tuberculosis, diffuse toxic goitre, malaria, suppuration, etc. Dry skin can be due to a great loss of water, e.g. in diarrhea or persistent vomiting (toxicosis of pregnancy, organic pylorostenosis).

Eruptions on the skin vary in shape, size, colour, persistence, and spread. The diagnostic value of eruptions is great in some infections such as measles, German measles, chicken- and smallpox, typhus, etc. *Roseola* is a rash-like eruption of 2-3 mm patches, which disappears when pressed. This is due to local dilatation of the vessels. Roseola is a characteristic symptom of typhoid fever, paratyphus, louse-borne typhus, and syphilis.

Erythema is a slightly elevated hyperaemic portion of the skin with distinctly outlined margins. Erythema develops in some persons hypersensitive to strawberries, eggs, and canned crabs. Erythema can develop after taking quinine, nicotinic acid,

after exposure to a quartz lamp, and also in some infectious diseases, such as erysipelas and septic diseases.

Weals (urticaria, nettle rash) appear on the skin as round or oval itching lesions resembling those, which appear on the skin bitten by stinging nettle. These eruptions develop as an allergic reaction.

Herpetic lesions are small vesicles 0.5 to 1 cm in size. They are filled with transparent fluid, which later becomes cloudy. Drying crusts appear in several days at the point of the collapsed vesicles. Herpes would normally affect the lips (herpes labialis, or cold sore) and the ala nasi (herpes nasalis). Less frequently herpetic lesions appear on the chin, forehead, cheeks, and ears. Herpetic lesions occur in acute lobar pneumonia, malaria, and influenza.

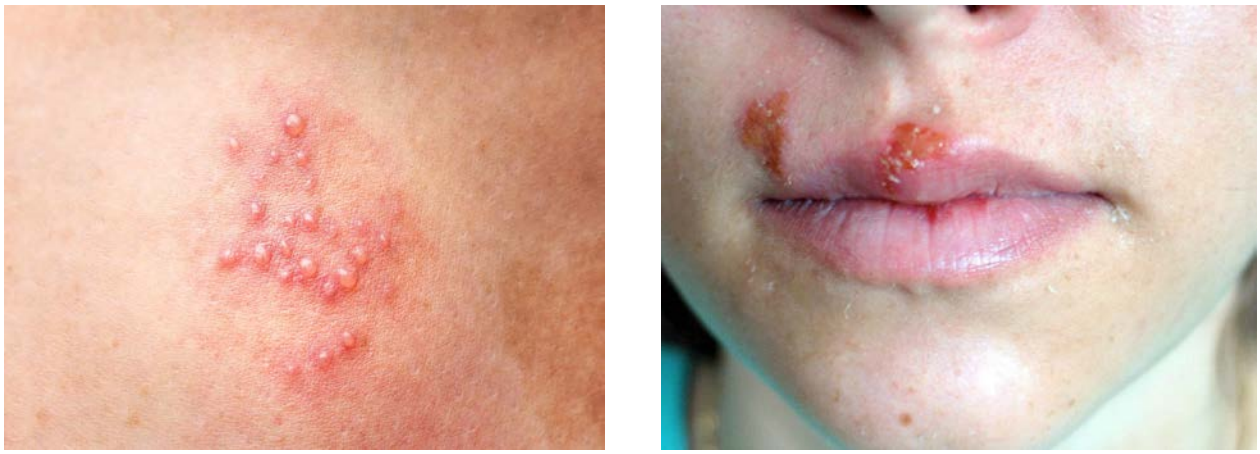

Image 2. Herpetic lesions
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Purpura is a haemorrhage into the skin occurring in Werlhoff's disease, haemophilia, scurvy, capillarotoxicosis, and longstanding mechanical jaundice. The lesions vary in size from small pointed haemorrhages (petechiae) to large black and blue spots (ecchymoses). Lesions of the skin are quite varied in character when they appear as allergic manifestations.

Desquamation of the skin is of great diagnostic value. It occurs in debilitating diseases and many skin diseases. Scars on the skin, e.g. on the abdomen and the hips, remain after pregnancy (striae gravidarum), in Itsenko-Cushing disease, and in extensive oedema. Indented stellar scars, tightly connected with underlying tissues, are

characteristic of syphilitic affections. Postoperative scars indicate surgical operations in past history.

Cirrhosis of the liver is often manifested by development of specific vascular stellae (telangiectasia). This is a positive sign of this disease.

Abnormal growth of hair is usually due to endocrine diseases. Abnormally excessive growth of hair (hirsutism, hypertrichosis) can be congenital, but more frequently it occurs in adrenal tumours (Itsenko-Cushing sydrome) and tumors of the sex glands. Deficient hair growth is characteristic of myxoedema, liver cirrhosis, eunuchoidism, and infantilism. Hair is also affected in some skin diseases.

Nails become excessively brittle in myxoedema, anemia and hypovitaminosis, and can also be found in some fungal diseases of the skin. Flattened and thickened nails are a symptom of acromegaly. Nails become rounded and look like watch glass in bronchiectasis, congenital heart diseases and some other affections.

Subcutaneous fat can be normal or to various degrees excessive or deficient. The fat can be distributed uniformly or deposited in only certain parts of the body. Its thickness is assessed by palpation. Excessive accumulation of subcutaneous fat (adiposis) can be due to either exogenic (overfeeding, hypodynamia, alcoholism, etc.) or endogenic factors (dysfunction of sex glands, the thyroid, or pituitary gland). Insufficient accumulation of subcutaneous fat may result from constitutional factors (asthenic type), malnutrition, or alimentary dysfunction. Excessive wasting is referred to as cachexia, and may occur in prolonged intoxication, chronic infections (tuberculosis), malignant newgrowths, diseases of the pituitary, thyroid and pancreas, and in some psychological disorders as well. Weighing the patient gives additional information about his diet and is an objective means in following up on the patient's weight changes during the treatment of obesity or cachexia.

Edema can be caused by penetration of fluid through the capillary walls and its accumulation in tissues. Accumulated fluid may be congestive (transudation) or inflammatory (exudation). Local edema is a result of some local disorders in the blood or lymph circulation; it is usually associated with thrombosis of the veins, that is, compression of the veins by tumors or enlarged lymph nodes. General edema associated with diseases of the heart, kidneys or other organs is characterized by

general distribution of edema throughout the entire body (anasarca) or by symmetrical localization in limited regions of the body. These phenomena can be due to the patient lying on one side. If edema is generalized and considerable, transudate may accumulate in the body's cavities: in the abdomen (ascites), pleural cavity (hydrothorax) and in the pericardium (hydropericardium). Examination reveals 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 in edema, and is especially apparent on loose subcutaneous tissues (the eyelids, the scrotum, etc.). In addition to observation, edema can also be revealed by palpation. When 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.

During examination of the *muscular system* the physician should assess its development, which depends on the patient's occupation, his sporting habits, etc. Local atrophy of muscles, especially muscles of the extremities, is diagnostically important. Atrophy can be determined by measuring the girth of the symmetrical muscles of both extremities. Determination of muscular strength and detection of functional muscular disturbances (cramps) are also important for diagnosis. Muscular dysfunction may occur in renal insufficiency (eclampsia), disorders of the liver (hepatic insufficiency), affections of the central nervous system (meningitis), tetanus, cholera, etc.

Defects (deformities or bulging) of the *bones* of the skull, chest, spine, and the extremities, may be revealed by external inspection. But in many cases palpation is necessary. Peripheral bones of the extremities (of the fingers, toes), cheek bones or the mandible grow abnormally in acromegaly. Rachitic changes occur in the form of the so-called pigeon breast, rachitic rosary (beading at the junction of the ribs with the cartilages), deformities of the lower extremities, etc. Tuberculotic lesions (the so-called haematogenic osteomyelitis) are localized mainly in the epiphyses of the bones, with formation of fistulae through which pus is regularly discharged. Multiple affections of the flat bones of the skeleton (the skull included) that can be seen radiographically as round light spots (bone tissue defects) are typical of myeloma. Diseases of the spine cause deformation of the spinal column and the chest. Considerable deformities of the spine (kyphosis, scoliosis) can cause dysfunction of the thoracic organs.

When examining *the joints* attention should be paid to their shapes, articulation, tenderness in active or passive movements, edema, and hyperemia of the adjacent tissues. Multiple affections of large joints are characteristic of exacerbated rheumatism. Rheumatoid arthritis affects primarily small joints of the hands with their subsequent deformation. Metabolic polyarthritides, e.g. in gout, are characterized by thickening of the terminal phalanges of the fingers and toes (so-called Heberden's nodes). Monarthritis (affection of one joint) would be usually observed in tuberculosis and gonorrhoea.

Normal *lymph nodes* cannot be detected visually or by palpation. Depending on the character of the process, their size varies from that of a pea to that of an apple. In addition to simple inspection, the physician should resort to palpation in order to make a conclusion on the condition of the lymphatic system. Attention should be paid to the size of the lymph nodes, their tenderness, mobility, consistency and adherence to the skin. Submandibular, axillary, cervical, supraclavicular, and inguinal lymph nodes are commonly enlarged. Submandibular nodes swell in the presence of inflammation in the mouth. Chronic enlargement of the cervical lymph nodes is associated with development of tuberculosis in them, which is characterized by purulent foci with subsequent formation of fistulae and immobile cicatrices.

Cancer of the stomach and, less frequently, cancer of the intestine can metastasize into the lymph nodes of the neck (on the left). The axillary lymph nodes are sometimes enlarged in mammary cancer. In the presence of metastases the lymph nodes are firm, their surface is rough, palpation is painless. Tenderness of a lymph node in palpation and reddening of the overlying skin indicates inflammation in the node. Systemic enlargement of the lymph nodes is observed in lympholeukaemia, lymphogranulomatosis, and lymphosarcomatosis. In lymphatic leukemia and lymphogranuloma the nodes fuse together but do not suppurate. Puncture or biopsy of the lymph nodes is required to diagnose complicated cases.

Changes in the size and shape of the *head* can give diagnostic clues. Excessive growth of the skull occurs in hydrocephalus. An abnormally small head is typical of microcephalus, which is also marked by mental under development. A square head, flattened on top, with prominent frontal tubers, can indicate congenital syphilis or rickets in past history. The position of the head is also important in diagnosing

cervical myositis or spondylarthritis. Involuntary movements of the head (tremor) are characteristic of parkinsonism. Rhythmical movements of the head in synchronism with the cardiac pulse are characteristic of aortic incompetence (Musset's sign). The presence of scars on the head may suggest the cause of persistent headache. It is necessary to find out whether the patient has vertigo, which is typical particularly for Meniere's syndrome, or epileptiform attacks.

Countenance. The facial expression can indicate the mental composure and various psychic and somatic conditions. It also depends on age and sex and can therefore give diagnostic clues when diagnosing some endocrine disorders (woman-like expression in men and masculine features in women). The following changes in the face are diagnostically essential:

1. A puffy face is observed in general edema characteristic of renal diseases; local venous congestion in frequent fits of suffocation and cough; compression of lymph ducts in extensive effusion into the pleural and pericardial cavity, in tumor of mediastinum, enlarged mediastinal lymph nodes, adhesive mediastinopericarditis, compressed superior vena cava (Stokes' collar).

2. Corvisart's fades is characteristics of cardiac insufficiency. The face is edematous, pale yellowish, with a cyanotic hue. The mouth is always half open, the lips are cyanotic, the eyes are dull and the eyelids sticky.

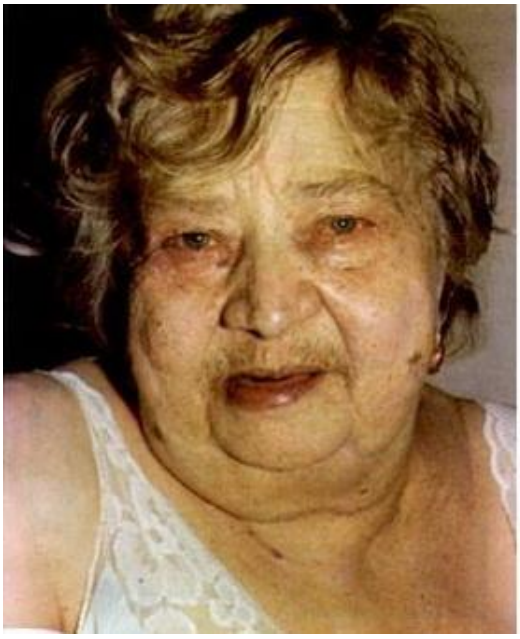

Image 3. Corvisart's face

The image was downloaded from website https://slideplayer.com

3. Facies febrilis is characterized by hyperaemic skin, sparkling eves and excited expression. There are special features of facies febrilis characteristic of some infectious diseases: feverish redness in acute lobar pneumonia (more pronounced on the side of the affected lung); general hyperemia of the puffy face is characteristic of louse-borne typhus, the sclera is injected (“rabbit eye”); slightly icteric yellow colour is characteristic of typhoid fever. Tuberculosis patients with fever have “burning” eyes on an exhausted and pale face with blush localized on the cheeks. An immobile face is characteristic of septic fever; the face pale, sometimes slightly yellowish.

4. Face and its expression are altered in various endocrine disorders: face with enlarged prominent parts (such as nose, chin, and cheek bones) and enlarged hands are characteristic of acromegalia (hands become enlarged in some pregnancies); myxedematous face indicates thyroid hypofunction: the face may be uniformly puffy with edematous mucosa, narrowed eye slits, the face features smoothed down, the hair is absent on the outward portions of the eyebrow, the presence of a blush on a pale face resembles the appearance of a doll; facies basedovica this is the face of a patient with thyroid hyperfunction: the face is lively with widened eye slits and abnormally sparkling eyes, the eyes are protruded and face looks as if frightened; an intense red, moon-like glittering face with a beard and mustaches in women is characteristic of the Itsenko-Cushing disease.

5. Facies leonjina with nodular thickening of the skin under the eyes and over the brown, with flattened nose is observed in leprosy.

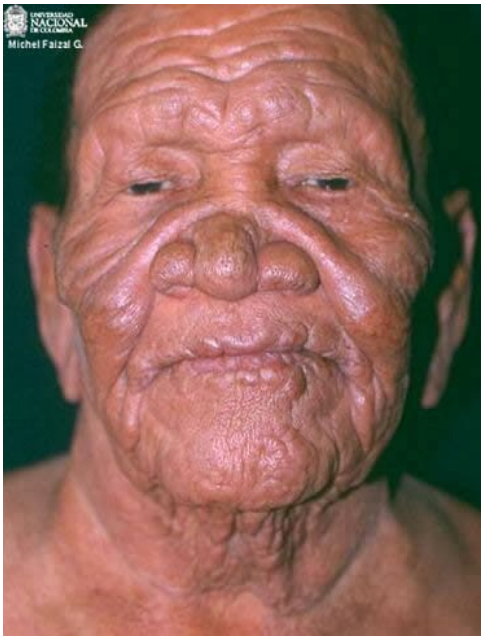

Image 4. Facies leonjina

The image was downloaded from website https://pt.quora.com

6. Parkinson’s mask (or facies) is an amimic face characteristic of encephalitis patients.

7. A slightly puffy wax-doll, very pale face with a yellowish tint, and seemingly translucent skin, is characteristic of Addison-Biermer anemia.

8. 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.

9. Facies Hippocratica (first described by Hippocrates) is associated with collapse in grave diseases of the abdominal organs (diffuse peritonitis, perforated ulcer of the stomach or duodenum, rupture of the gall bladder). The face is characterized by sunken eyes, pinched nose, deadly livid and cyanotic skin, which is sometimes covered with large drops of cold sweat.

10. Asymmetric movements of facial muscles indicate a history of cerebral hemorrhage or facial neuritis.

Inspection of the *eyes* and eyelids can reveal some essential diagnostic signs. Edema of the eyelids, especially of the lower eyelids, is the first indication of acute nephritis; it is also observed in anemia, frequent attacks of cough, and deranged sleep; edema of the eyelids can also occur in the morning in healthy persons as well.

The colour of the eyelids is important. The eyelids are dark in diffuse toxic goitre and Addison disease. Xanthomas indicate deranged cholesterol metabolism. A dilated eye slit with the eyelids that do not close is characteristic of paralysis of the facial nerve; persistent drooping of the upper eyelid (ptosis) is an important sign of some affections of the nervous system. Narrowing of the eye slit occurs in myxoedema and general edema of the face. Exophthalmos (protrusion of the eyeball) is observed in thyrotoxicosis, retrobulbar tumors, and also in strong myopia. Recession of the eyeball in the orbit (enophthalmos) is typical of myxoedema and is an important sign of "peritoneal face". Unilateral recession of the eye into the orbit attended by narrowing of the eye slit, drooping of the upper eyelid and narrowing of the pupil, is the Horner's (Bernard-Homer) syndrome caused by the affection of the pupil sympathetic enervation of the same side (due to various causes).

The shape of the pupils, their symmetry, response to light, accommodation and convergence, and also their "pulsation" are of great diagnostic significance in

certain diseases. Abnormally contracted pupil (miosis) is observed in uraemia, tumors and intracranial haemorrhages, and in morphine poisoning. Enlargement of the pupil (mydriasis) occurs in comatose states (except uraemic coma) and cerebral haemorrhages, and also in atropine poisoning. Anisocoria (unequal size of the pupils) occurs in some affections of the nervous system. Squinting results from paralysis of the ocular muscles due to lead poisoning, botulism, diphtheria, affections of the brain and its membranes (syphilis, tuberculosis, meningitis, cerebral haemorrhage).

The size of the *nose* may attract attention providing some diagnostic signs, e.g. it has an abnormal size in acromegaly, or its shape deviates from the normal in rhinoscleroma. The nose may be sunken as a result of syphilis in the past history (saddle nose). Soft tissues of the nose are disfigured in lupus.

When inspecting the *mouth* attention should be paid to its shape (symmetry of the angles, permanently open mouth), the colour of the lips, eruption on the lips (cold sores, herpes labialis), and the presence of fissures. The oral mucosa should also be inspected (for the presence of aphthae, pigmentation, Filatov-Koplik spots, thrush, contagious aphthae of the foot and mouth disease, hemorrhage). Marked changes in the gums can be observed in some diseases (such as pyorrhoea, acute leukaemia, diabetes mellitus, and scurvy) and poisoning (with lead or mercury). The teeth should be examined for the absence of defective shape, size, or position. The absence of many teeth is very important in the etiology of some alimentary diseases. Caries is the source of infection and can affect some other organs.

Disordered movement of the tongue may indicate nervous affections, grave infections and poisoning. Marked enlargement of the tongue is characteristic of myxoedema and acromegaly; less frequently it occurs in glossitis. Some diseases are characterized by the following abnormalities of the tongue: the tongue is clear, red, and moist in ulcer; crimson-red in scarlet fever; dry, with a brown coat and grooves in grave poisoning and infections; coated in the centre and at the root, but clear at the tip and margins in typhoid fever; smooth tongue without papillae (as if polished) is characteristic of Addison-Biermer disease. The glassy tongue is characteristic of gastric cancer, pellagra, sprue, and ariboflavinosis; local thickening of the epithelium is characteristic of smokers (leucoplakia). Local pathological processes, such as ulcers

of various etiology, scars, traces left from tongue biting during epileptic fits, etc., are also suggestive of certain diseases.

During inspection of the *neck* attention should be paid to pulsation of the carotid artery (aortic incompetence, thyrotoxicosis), swelling and pulsation of the external jugular veins (tricuspid valve insufficiency), enlarged lymph nodes (tuberculosis, lympholeukaemia, lymphogranulomatosis, cancer metastases), diffuse or local enlargement of the thyroid gland (thyrotoxicosis, simple goitre, malignant tumor).

Examination of the *extremities* can reveal varicosity of the veins, edema, changes in the skin, muscles, tremor of the extremities, deformities, swelling and hyperaemia of the joints, ulcers, and scars. Diseases of the central nervous system (tumors, cerebral hemorrhage) and also of the peripheral nervous system can cause atrophy and paralysis of the muscles.

Hippocrates fingers or clubbing of the terminal phalanges of the fingers and toes are important diagnostic ally. The changed shape of the nails resembles hour glass. This symptom is characteristic of prolonged diseases of the lung (chronic purulent processes), heart (subacute septic endocarditis, congenital heart defects) and liver (cirrhosis). Periodically occurring vascular spasms in the extremities cause the development of the symptom known as the dead finger, transient pallor of the fingers and toes, which is characteristic of Raynaud's disease. Prolonged spasms of blood vessels can cause gangrene of the fingers.

When examining the legs, attention should be paid to possible flat foot. Saber shins occur in rickets and sometimes in syphilis. Uneven thickening of the leg bones indicates periostitis, which can sometimes be of syphilitic etiology.

PHYSICAL AND INSTRUMENTAL METHODS OF EXAMINATION OF THE BRONCHO-PULMONARY SYSTEM. THE INTERVIEW AND THE GENERAL EXAMINATION OF PATIENTS WITH THE RESPIRATORY SYSTEM PATHOLOGY. THE BRONCHOOBSTRUCTIVE SYNDROME. THE ANALYSIS OF THE RESPIRATORY FUNCTION. CHANGES IN THE ORAL CAVITY IN PATIENTS WITH BRONCHO-OBSTRUCTIVE SYNDROME

The most characteristic symptoms of diseases of the respiratory system are: cough, breathlessness, haemoptysis and chest pain.

Cough it is a protective reflex of an organism arising from irritation of respective parts of respiratory tract and pleura, which can be manifested as:

Dry (unproductive) - usually irritating, persistent, arises from tracheitis, acute bronchitis, pleuritis, during inhalation of irritating gases and in bronchogenic carcinoma.

Some diseases are attended only by dry cough, e.g. laryngitis, dry pleurisy or compression of the main bronchi by the bifurcation lymph nodes (tuberculosis, lymphogranulomatosis, cancer metastases, etc.). Bronchitis, pulmonary tuberculosis, pneumosclerosis, abscess, or bronchogenic cancer of the lungs can be first attended by dry cough, which will then turn into moist one with expectoration of the sputum.

Wet (productive) with mucous expectoration:

Serous sputum - thin mixed with blood, in lung edema (pinkish);

Mucous sputum - mostly viscous, at the beginning of acute bronchitis, in asthmatic attack;

Pus-mucous sputum - yellowish, yellow-greenish, occurs in chronic bronchitis, bronchiectasia, and tuberculosis.

If a patient complains of cough with sputum, the physician should try to determine the amount of sputum expectorated during one fit and during the entire day; it is also important to know the time of the day during which the sputum is expectorated and the position of the body at which cough is provoked; the colour, odour, and other properties of sputum are also important. Morning cough is characteristic of patients with chronic bronchitis, bronchiectasis, lung abscess, and cavernous tuberculosis of the lungs. The sputum accumulates during the night sleep

in the lungs and the bronchi, but as the patient gets up, the sputum moves to the neighboring parts of the bronchi to stimulate the reflexogenic zones of the bronchial mucosa. This causes cough and expectoration of the sputum. The amount of the sputum expectorated during the morning may amount to two thirds of the entire daily expectoration. Depending on the gravity of the inflammatory process in patients with mentioned diseases, the daily amount of the expectorated sputum may vary from 10-15 ml to as much as 2 liters. In unilateral bronchiectasis, sputum may be better expectorated in a definite posture, for example, on the right side with bronchiectasis in the left lung, and vice versa. If bronchiectasis is found in the anterior region of the lungs, expectoration is easier in the supine position, and if in the posterior parts, in the prone position.

Dyspnea (Breathlessness) is a symptom, not a sign, and is one of several sensations a patient may describe. A healthy person notes the increased ventilation required during exercise but does not interpret it as being particularly unpleasant unless extreme. Unpleasant or worrisome awareness that a small amount of exercise leads to a disproportionately large increase in ventilation is a common type of dyspnea, usually described as breathlessness or shortness of breath on exertion. At high altitude, a healthy person notes a similar disproportionately large increase in ventilation resulting from exertion and finds it limiting but usually not otherwise unpleasant.

Other sensations include awareness of increased muscular effort required to expand the chest during inspiration or to expel air from the lungs, sensations of fatigue in the respiratory muscles, awareness of a delay in air leaving the lungs during expiration, the uncomfortable sensation that an inspiration is urgently needed before expiration is completed, and various sensations most often described as tightness in the chest. The last can probably include awareness of collapse or hyperinflation of lung units, obstruction of airways, and distortion or displacement of the lungs, mediastinum, diaphragm, or chest wall.

Afferent impulses to the brain that generate the sensation of dyspnea come from many different sites, such as the lungs, articulations of the rib cage, and the respiratory muscles, including the diaphragm. Peripheral and central chemoreceptors provide part of the sensory input that appears to be involved in dyspnea, either

directly or indirectly; other visceral, neural, and emotional stimuli may also participate.

The two major causes of pulmonary dyspnea are a restrictive defect with low compliance of the lungs or chest wall and an obstructive defect with increased resistance to airflow. Patients with restrictive dyspnea (due to pulmonary fibrosis or chest deformities) are usually comfortable at rest but intensely dyspneic when exertion causes pulmonary ventilation to approach their greatly limited breathing capacity. In obstructive dyspnea (in chronic obstructive pulmonary disease or asthma), increased ventilatory effort induces dyspnea even at rest, and breathing is labored and retarded, especially during expiration; this type of dyspnea always worsens during effort and exercise.

According to clinical manifestation the following types of breathlessness can be recognised:

Inspiratory breathlessness - with more difficult inspiration (aspiration of foreign body, stenosis of larynx, compression of trachea and bronchi);

Expiratory breathlessness - with markedly prolonged expiration (bronchial asthma);

Mixed breathlessness - with difficult inspiration and expiration (heart failure).

Abnormal breathing with stridor (severe inspiratory or expiratory breathlessness) accompanied by loud wheezy sounds can be caused by spasm (stenosis) of great airways, edema, foreign body, or outside compression (carcinomas, enlarged thyroid gland).

Physical findings may help determine the cause (pleural effusion, pneumothorax, and sometimes interstitial lung disease). The signs of emphysema, bronchitis, and asthma are frequently helpful in defining the nature and severity of the underlying obstructive lung disease. Pulmonary function testing can provide numeric values for any restriction or airflow obstruction present.

Orthopnea is respiratory discomfort that occurs while the patient is supine, impelling him to sit up. It is precipitated by an increase in venous return of blood to a failing left ventricle that cannot handle the increased preload. Of less importance is the increased effort of breathing in the supine position.

In evaluating chest pain, the first task-not always easy is to differentiate respiratory pain from pain related to other systems. Chest pain related to bronchopulmonary diseases occurs relatively rarely (sensitive innervations of lungs and visceral pleura is missing). Difficulties are caused by affection of parietal pleura. Most noncardiac chest pain arises from the pleura or the chest wall. Pleuritic pain is typically made worse by deep breathing or coughing and may be controlled by immobilization of the chest wall; eg, the patient may hold his side, avoid deep breathing, or suppress his cough. The patient can usually identify the site of pleuritic pain. Over time it may move from one site to another. If a pleural effusion develops, the pain may disappear as the inflamed pleural surfaces are separated. A friction rub is often associated with pleuritic pain, but either may occur alone.

Pain arising from the chest wall may be exacerbated by deep breathing or coughing, but it can usually be distinguished by localized tenderness. Although some tenderness may be present with pleuritic pain (eg, in pneumococcal pneumonia), it is usually slight, poorly localized, and elicited only by deep pressure. Chest wall trauma or a broken rib is often obvious from the history, but torn muscle fibers or even a rib fracture can result from severe coughing. A tumor infiltrating the chest wall may cause local pain or, if it involves intercostal nerves, referred pain. Herpes zoster, before the eruption appears, may present as puzzling chest pain. Physical examination and chest x-rays can usually determine the cause.

Pleural pain - is manifested by a strong sharp pain connected to breathing and cough (dry pleuritis, lung infarction, bronchopneumonia with pleural reaction).

Tracheal pain - is characterised by intensive stinging, retrosternal pain in acute phase of illness (diagnostically essential to exclude myocardial infarction).

Tumorous pain - caused by the tumour is growing into the brachial plexus (Pancoast tumour is a peripheral form of bronchogenic carcinoma). It is manifested by intensive shoulder pain with irradiation into the arm.

Hemoptysis means expectorating of blood when minor or major vessels of respiratory tract are damaged. Massive bleeding is life-threatening state condition. The most common reasons are:

Bronchopulmonary - bronchogenic carcinoma, tuberculosis, bronchiectasia, chronic bronchitis;

Cardiac - mitral stenosis, congenital heart defects, vascular malformation, lung infarction;

Hematological - hemorrhagic diathesis, unadjusted anticoagulation therapy.

Anamnesis. When questioning the patient the physician should determine the time the disease began. Acute onset is characteristic of acute pneumonia, especially acute lobar pneumonia. Pleurisy begins more gradually. A non-manifest onset and a prolonged course are characteristic of pulmonary tuberculosis and cancer. The onset of many diseases may be provoked by chills (bronchitis, pleurisy, pneumonia).

Determining epidemiological conditions is very important for establishing the cause of the disease. Thus influenzal pneumonia often occurs during epidemic outbreaks of influenza. Establishing contacts with tuberculosis patients is also very important. Specific features of the course of the disease and the therapy given (and its efficacy) should then be established.

When collecting the life anamnesis, the physician should pay attention to conditions under which the patient lives and works. Damp premises with inadequate ventilation or work in the open (builders, truck drivers, agricultural workers, etc.) can become the cause of acute inflammation of the lungs with more frequent conversion into chronic diseases. Some dusts are harmful and cause bronchial asthma. Coal dust causes a chronic disease of the lungs called anthracosis. Regular exposure to silica dust (cements, pottery, etc.) causes silicosis, the occupational fibrosis of the lungs.

The patient should give a detailed report of his past diseases of the lungs or pleura, which help the physician, establish connections between the present disease and diseases of the past history.

Examination of the chest should be done according to a definite plan. The general configuration of the chest should the next step is to define the type, rhythm and frequency of breathing, respiratory movements of the left and right shoulder blades, and of the shoulder girdle, and involvement of the accessory respiratory muscles in the breathing act. The patient should be better examined in the upright (standing or sitting) position with the chest being naked. Illumination of the body should be uniform.

The shape of the chest may be normal or pathological. A normal chest is characteristic of healthy persons with regular body built. Its right and left sides are

symmetrical, the clavicles and the shoulder blades should be at one level and the supraclavicular fossae equally pronounced on both sides. Since all people with normal constitution are conventionally divided into three types, the chest has different shape in accordance with its constitutional type. Pathological shape of the chest may be the result of congenital bone defects and of various chronic diseases (emphysema of the lungs, rickets, tuberculosis).

Normal shape of the chest

1. *Normosthenic (conical) chest* in subjects with normosthenic constitution resembles a truncated cone whose bottom is formed by well-developed muscles of the shoulder girdle and is directed upward. The anteroposterior (sternovertebral diameter of the chest is smaller than the lateral (transverse) one, and the supraclavicular fossae are slightly pronounced. There is a distinct angle between the sternum and the manubrium (angulus Ludowici); the epigastric angle nears 90°. The ribs are derately inclined as viewed from the side; the shoulder blades closely fit to the chest and are at the same level; the chest is about the same height as the abdominal part of the trunk.

2. *Hypersthenic chest* in persons with hypersthenic constitution has the shape a cylinder. The anteroposterior diameter is about the same as the transverse one; the supraclavicular fossae are absent (level with the chest). the manubriosternal angle is indistinct; the epigastric angle exceeds 90°; the ribs in the lateral parts of the chest are nearly horizontal, the intercostal space is narrow, the shoulder blades closely fit to the chest, the thoracic part of the trunk is smaller than the abdominal one.

3. *Asthenic chest* in persons with asthenic constitution is elongated, narrow both the anteroposterior and transverse diameter are smaller than normal); the chest is flat. The supra- and subclavicular fossae are distinctly pronounced. There is no angle between the sternum and the manubrium: the sternal bone and the manubrium make a straight "plate". The epigastric angle is less than 90°. The ribs are more vertical at the sides, the tenth ribs are not attached to the costal arch (costa decima fluctuens); the intercostal spaces are wide, the shoulder blades are winged (separated from the chest), the muscles of the shoulder girdle are underdeveloped, the shoulders are sloping, the chest is longer than the abdominal part of the trunk.

Pathological chest

1. *Emphysematous (barrell-like) chest* resembles a hypersthenic chest in its shape, but differs from it by a barrel-like configuration, prominence of the chest wall, especially in the posterolateral regions, the intercostal spaces are enlarged. This type of chest is found in chronic emphysema of the, lungs, during which, elasticity of the lungs decreases while the volume of the lungs increases, the lungs seem to be as if at the inspiration phase. Natural expiration is therefore difficult not only during movements but also at rest (expiratory dyspnoea is found). Active participation of accessory respiratory muscles in the respiratory act (especially m. sternocleidomastoideus and m. trapezius), depression of the intercostal space, elevation of the entire chest during inspiration and relaxation of the respiratory muscles and lowering of the chest to the initial position during expiration become evident during examination of emphysema patients.

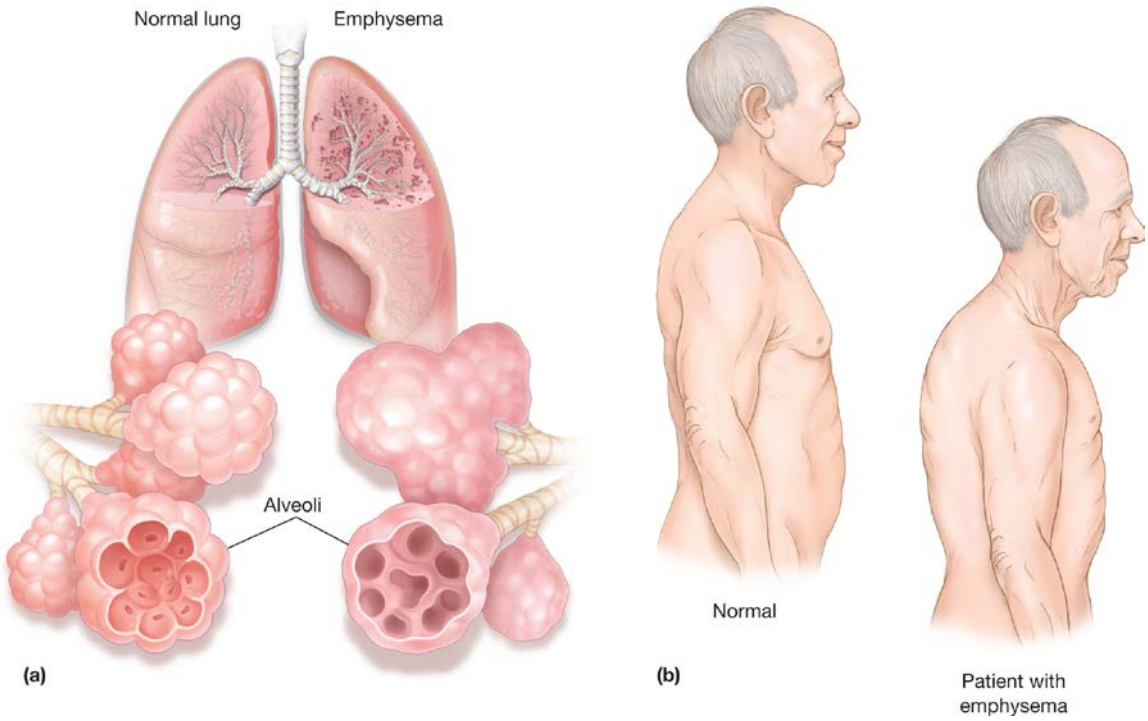

Image 5. Chronic emphysema

The image was downloaded from website https://biology-forums.com

2. *Paralytic chest* resembles the asthenic chest. It is found in emaciated patients, in general asthenia it often occurs in grave chronic diseases, more commonly in pulmonary tuberculosis and pneumosclerosis, in which fibrous tissue contract the lungs and diminishes their weight due to the progressive chronic

inflammation. During examination of patients with paralytic chest, marked atrophy of the chest muscles and asymmetry of the clavicles and dissimilar depression of the supraclavicular fossae can be observed along with typical signs of asthenic chest. The shoulder blades are not at one level either, and their movements during breathing are asynchronous.

3. *Rachitic chest (keeled or pigeon chest).* It is characterized by a markedly greater anteroposterioir diameter (compared with the transverse diameter) due to the prominence of the sternum (which resembles the keel of a boat.) The anterolateral surfaces of the chest are as if pressed on both sides and therefore the ribs meet at an acute angle at the sternal bone, while the costal cartilages thicken like beads at points of their transition to bones (rachitic beads). As a rule, these beads can be palpated after rickets only in children and youths.

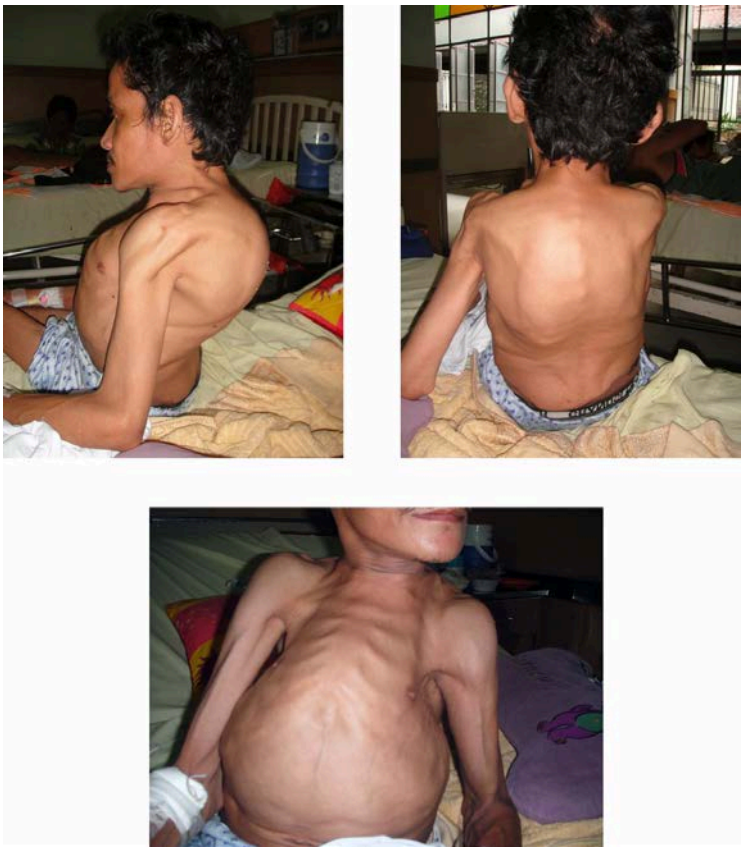

Image 6. Rachitic chest in a young adult male

The image was downloaded from website https://casereports.bmj.com

4. *Funnel chest* can occur in normosthenic, hypersthenic or asthenic subjects; it has a funnel-shaped depression in the Tower part of the sternum. This deformity can be regarded as a result of abnormal development of the sternum or

prolonged compressing effect. In older times this chest would be found in shoemaker adolescents. The mechanism of formation of the funnel chest was explained by the permanent pressure of the chest against the shoe; the funnel chest was therefore formerly called cobbler chest.

5. *Foveated chest* is almost the same as the funnel chest except that the depression is found mostly in the upper and the middle parts of the anterior surface of the chest. This abnormality occurs in syringomyelia, a rare disease of the spinal cord. The chest may be abnormal in subjects with various deformities of the spine, which arise as a result of injuries, tuberculosis of the spine, rheumatoid arthritis (Bekhterev's disease), etc.

6. Four types of spine deformities are distinguished: *scoliotic chest* is observed in lateral curvature of the spine; *kyphotic chest* is excessive forward and backward curvature of the spine; *kyphoscoliotic chest* is combination of the lateral and forward curvature of the spine; backward curvature (lordosis) of the spine may result in *lordotic chest*. Scoliosis is the most frequently occurring deformity of the spine. It mostly develops in school children due to bad habitual posture. Kyphoscoliosis occurs less frequently. Lordosis only occurs in rare cases. Curvature of the spine, especially kyphosis, lordosis, and kyphoscoliosis cause marked deformation of the chest to change the physiological position of the lungs and the heart and thus interfere with their normal functioning.

The shape of the chest can readily change due to enlargement or diminution of one half of the chest (asymmetry of the chest). These changes can be transient or permanent.

The enlargement of the volume of one half of the chest can be due to escape of considerable amounts of fluid as the result of inflammation (exudate) or non-inflammatory fluid (transudate) into the pleural cavity, or due to penetration of air inside the chest in injuries (pneumothorax). Leveling or protrusion of the intercostal spaces, asymmetry of the clavicles and the shoulder blades and also unilateral thoracic lagging can be observed during examination of the enlarged part of the chest. The chest assumes normal shape after the air or fluid is removed from the pleural cavity.

One part of the chest may diminish due to pleural adhesion or complete closure of the pleural slit after resorption of effusion (after prolonged presence of the fluid in the pleural cavity); contraction of a considerable portion of the lung due to growth of connective tissue (pneumosclerosis) after acute or chronic inflammatory processes, such as acute lobar pneumonia (with subsequent carnification of the lung), lung infarction, pulmonary abscess, tuberculosis, etc.; resection of a part or the entire lung; atelectasis (collapse of the lung or its portion) that may occur due to closure of the lumen in a large bronchus by a foreign body or a tumor growing into the lumen of the bronchus and causing its obturation. The closure of the air passage into the lung with subsequent resorption of air from the alveoli and a decrease in the volume of the lung diminish the corresponding half of the chest. The chest thus becomes asymmetrical, the shoulder of the affected side lowers, the clavicle and the scapula lower as well, and their movements during deep respiration become slower and limited; the supra- and subclavicular fossae become more depressed, the intercostal spaces decrease in size or become invisible. The marked depression of the supraclavicular fossae on one side often depends on the diminution of the apex of a fibrosis-affected lung.

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

The type, frequency, depth and rhythm of respiration can be determined by carefully observing the chest and abdomen. Respiration can be costal (thoracic), abdominal, or mixed type.

Thoracic (costal) respiration. Respiratory movements are carried out mainly by the contraction of the intercostal muscles. The chest markedly broadens and slightly rises during inspiration, while during expiration it narrows and slightly lowers. This type of breathing is known as costal and is mostly characteristic of women.

Abdominal respiration. Breathing is mainly accomplished by the diaphragmatic muscles; during the inspiration phase the diaphragm contracts and lowers to increase rarefaction in the chest and to suck in air into the lungs. The intraabdominal pressure increases accordingly to displace anteriorly the abdominal wall. During expiration the muscles are relaxed, the diaphragm rises, and the abdominal wall returns to the initial position. The type of respiration is also called diaphragmatic and characteristic of men.

Mixed respiration. The respiratory movements are carried out simultaneously by the diaphragm and the intercostal muscles. In physiological conditions this respiration sometimes occurs in aged persons and in some pathological conditions of the respiratory apparatus and the abdominal viscera. For example, in women with dry pleurisy, pleural adhesion, myositis and thoracic radiculitis, the contractile activity of the intercostal muscles decreases and the respiratory movements are carried out by the accessory movements of the diaphragm. In extensive pleural adhesion, lung emphysema, and in strong pain in the chest due to acute inflammation of the intercostal muscles or nerves, respiration is temporarily carried out by the diaphragmatic muscles exclusively. Mixed respiration occurs in men with underdeveloped diaphragmatic muscles, in diaphragmatitis, acute cholecystitis, perforating ulcer of the stomach or the duodenum.

Respiration rate. Respiration rate can be determined by counting the movements of the chest or the abdominal wall, with the patient being unaware of the procedure. The pulse rate should first be taken and then the respiration rate. The number of respiratory movements in a healthy, adult at rest should be 16 to 20 per minute. The respiration rate decreases during sleep to 12-14 per minute, while under physical load, emotional excitement, or after heavy meals the respiration rate increases.

The respiration rate alters markedly in some pathological conditions. The causes of accelerated respiration may be narrowing of the lumen of small bronchi due to spasms or diffuse inflammation of their mucosa (bronchiolitis occurring mostly in children), which interfere with normal passage of air into the alveoli; decreased respiratory surface of the lungs due to their inflammation and tuberculosis, in collapse or atelectasis of the lung due to its compression (pleurisy

with effusion, hydrothorax, pneumothorax, tumor of mediastinum), in obturation or compression of the main bronchus by a tumor, in thrombosis or embolism of the pulmonary artery, in pronounced emphysema, when the lung is overfilled with blood or in a case of lung edema in certain cardiovascular diseases; insufficient depth of breathing (superficial respiration) which can be due to difficult contractions of the intercostal muscles or the diaphragm in acute pain (dry pleurisy, acute myositis, intercostal neuralgia, rib fracture, or tumor metastasis into the ribs), in a sharp increase in the intraabdominal pressure and high diaphragm (ascites, meteorism, late pregnancy), and finally in hysteria.

Pathological deceleration of respiration occurs in functional inhibition of the respiratory centre and its decreased excitability. It can be due to increased intracranial pressure in patients with cerebral tumor, meningitis, cerebral haemorrhage, or edema of the brain, and also due to the toxic effect on the respiratory centre when toxic substances are accumulated in the blood, e.g. in uraemia, hepatic or diabetic coma, and in certain acute infectious diseases.

Respiration depth. The depth of breathing is determined by the volume of the inhaled and exhaled air at rest. This volume varies in an adult from 300 to (500 ml on the average). Depending on depth, breathing can be either deep or superficial. Superficial (shallow) breathing often occurs in pathologically accelerated respiration when the length of the inspiration and the expiration phases becomes short. Deep and slow breatting is, on the con trary, associated in most cases with pathological deceleration of the respiration rate. Deep and slow respiration, with marked respiratory movements, is sometimes attended by noisy sounds. This is *Kussmaul's respiration* occurring in deep coma. In some pathological conditions, however, rare respiration can be shallow, while accelerated breathing deep. Rare superficial respiration can occur in sharp inhibition of the respiratory centre, pronounced lung emphysema, and sharp narrowing of the vocal slit or the trachea. Respiration becomes accelerated and deep in high fever and marked anemia.

Respiration rhythm. Respiration of a healthy person is rhythmic, of uniform depth and equal length of the inspiration and expiration phases. Rhythm of the respiratory centre can be inhibited in some types of edema. Derangement of the respiratory function can cause edema in which a series of respiratory movements

alternates with a pronounced (readily detectable) elongation of the respiratory pause (lasting from a few seconds to a minute) or a temporary arrest of respiration (apnoea). This respiration is known as periodic.

Biot's respiration is characterized by rhythmic but deep respiratory movements, which alternate (at approximately regular intervals) with long respiratory pauses (from few seconds to half a minute). Biot's respiration occurs in meningitis patients and in agony with disorders of cerebral circulation.

Cheyne-Stokes' respiration is characterized by periods (from few seconds to a minute) of cessation of respiration, followed by noiseless shallow respiration, which quickly deepens, becomes noisy to attain its maximum at the 5-7th inhalation, and then gradually slows down to end with a new short respiratory pause. During such pauses, the patient often loses his sense of orientation in the surroundings or even faints, to recover from the unconscious condition after respiratory movements are restored. This respiratory disorder occurs in diseases causing acute or chronic insufficiency of cerebral circulation and brain hypoxia, and also in heavy poisoning. More frequently this condition develops during sleep and is more characteristic of aged persons with marked atherosclerosis of the cerebral arteries.

Undulant (wave-like) Grocco's respiration somewhat resembles Cheyne-Stokes' respiration except that a weak shallow respiration occurs instead of the respiratory cause with subsequent deepening of the respiratory movement, follow by slowing down. This type of arrhythmic dyspnoea can probably be regarded as the early stages of the same pathological processes, which are responsible for Cheyne-Stokes respiration.

Palpation is used as an additional means of examination to verify findings of observation (the shape of the chest, its dimensions, respiratory movements), for determining local or profuse tenderness of the chest, its elasticity (resilience), vocal resonance, pleural friction and sounds of fluid in the pleural cavity.

Palpation should be done by placing the palms on the symmetrical (left and right) parts of the chest. This examination helps follow the respiratory excursions and deviation of the chest movements from their normal course. The epigastric angle is determined by palpation as well. The thumbs should be pressed tightly against the costal arch, their tips resting against the xiphoid process (ensiform cartilage).

Resilience or elasticity of the chest is determined by exerting pressure of the examining hands from the front to the sides of the chest or on the back and the sternum, and also by palpation of the intercostal spaces. The chest of a healthy person is elastic, plaint and yields under the pressure. In the presence of pleurisy with effusion, or pleural tumor, the intercostal space over the affected site becomes rigid. Rigidity of the chest increases in general in the aged due to ossification of the costal cartilages, development of the lung emphysema, and also with filling of both pleural cavities with fluid. Increased resistance of the chest can then be felt during examining the chest by compression in both the anteroposterior and lateral directions.

Palpation is used for determining the strength of voice conduction to the chest surface – *voice resonance* (fremitus pectoralis). The palms of the hands are placed on the symmetrical parts of the chest and the patient is asked to utter loudly a few words (with the letter 'r' in them to intensify vibration). The voice should be as low as possible since voice vibrations are better transmitted by the air column in the trachea and the bronchi to the chest wall in this case. Fremitus vocalis can also be determined by one hand as well: the palm of the examining hand should be placed alternately on the symmetrical parts of the chest.

Vocal resonance of about the same intensity in the symmetrical parts of the chest of a healthy person. Vocal vibrations are louder in the upper parts of the chest and softer in its lower parts. Vocal resonance can be stronger or weaker (or in some cases it can even be impalpable) in pathological conditions of the respiratory organs. In focal affections, vocal resonance becomes unequal over symmetrical parts of the chest.

Vocal resonance is **intensified**, when a part of the lung or its whole lobe becomes airless and more uniform (dense) because of a pathological process. According to the laws of physics, dense and uniform bodies conduct sound better than loose and non-uniform. Induration (consolidation) can be due to various causes, such as acute lobar pneumonia, pulmonary infarction, tuberculosis, etc. Vocal resonance is also intensified in the presence in the pulmonary tissue of an air cavity communicated with the bronchus.

Vocal resonance becomes **weaker**, when liquid or gas are accumulated in the pleural cavity; they separate the lung from the chest wall to absorb voice vibrations propagating from the vocal slit along the bronchial tree; in complete obstruction of the bronchial lumen by a tumor which in terferes with normal conduction of sound waves to the chest wall; in asthenic emaciated patients (with weak voice); insignificant thickening of the chest wall in obesit. Low-frequency vibrations due to pleural friction (friction fremitus) in dry pleurisy, crepitation sounds characteristic of subcutaneous emphysema of the lungs, vibration of the chest in dry, low (low-pitch buzzing) rales can also be determined by palpation.

Percussion. Tapping various parts of the human body produces sounds by which one can learn about the condition of the underlying organs.. The difference in the sounds of percussed lungs, liver, spleen, heart, stomach and other organs depends on (a) the different amount of gas or air inside or round the percussed organ; (b) tension of the tissue; and (c) different strength of the percussion stroke transmitted to this gas or air.

Main rules of percussion.

1. 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 position. When the patient is percussed from his back, he should be sitting on a chair, his face turned to the chair back. The head should be slightly bent forward, his arms should rest against his lap. In this position muscle relaxation is the greatest and percussion thus becomes more easy.

2. The room should be warm and protected from external noise.

3. The physician should be in a comfortable position as well.

4. A pleximeter or the middle finger of the left hand, which is normally used in the finger-to-finger percussion, should be pressed tightly to the examined surface. The neighbouring fingers should be somewhat set apart and tightly pressed to the patient's body. This is necessary to delimit propagation of vibrations arising during percussion. The physician's hands should be warm.

5. The percussion sound should be produced by the tapping movement of the hand alone. The sound should be short and distinct. Tapping should be uniform, the force of percussion strokes depending on the object being examined.

6. In topographic percussion, the finger or the pleximeter should be placed parallel to the anticipated border of the organ. Organs giving resonant note should be examined first: the ear will better detect changes in sound intensity. The border is marked by the edge of the pleximeter directed toward the zone of the more resonant sounds.

Percussion is done by tapping with a plexor (hammer) on a pleximeter placed on the body, or by a finger on another finger. This is *mediate* percussion. In *immediate* percussion the examined part of the body is struck directly by the soft tip of the index finger. To make tapping stronger, the index finger may be first held by the side of the middle finger and then released. This method was proposed by Obraztsov. Its advantage is that the striking finger feels the resistance of the examined part of the body.

Percussion is done with a slightly flexed middle finger on the dorsal side of the second phalanx of the middle finger of the opposite hand, which is pressed tightly against the examined part of the body. Percussion should be done by the movement of the wrist alone without involving the forearm into the movement. Striking intensity should be uniform, blows must be quick and short, directed perpendicularly to the intervening finger. Tapping should not be strong.

Tapping strength can vary depending on the purpose of the examination. Loud percussion (with a normal force of tapping), light, and lightest (threshold) percussion are differentiated. The heavier the percussion stroke, the greater is the area and depth to which the tissues are set vibrating, and hence the more resonant is the sound. In heavy or deep percussion, tissues lying at a distance of 4-7 cm from the pleximeter are involved. In light or surface percussion the examined zone has the radius of 2- 4 cm. Heavy percussion should therefore be used to examine deeply located organs, and light percussion for examining superficial organs.

Light percussion is used to determine the size and borders of various organs (liver, lungs and heart). The lightest percussion is used to determine absolute cardiac dullness. The force of the percussion stroke should be the slightest (at the threshold of sound perception). The Goldscheider method is often used for this purpose, the middle finger (flexor) of the right hand is used to tap the middle finger of the left

hand flexed at the second phalanx and placed at a right angle touching the surface only with the soft tip of the terminal phalanx (pleximeter).

Topographic percussion is used to determine the borders, size and shape of organs. Comparison of sounds on symmetrical points of the chest is called *comparative percussion.*

Sounds obtained by percussion differ in strength (clearness), pitch, and tone. Sounds may be strong and clear (resonant) or soft and dull; they may be high or low, and either tympanic or non-tympanic (and with metallic tinkling).

Clear pulmonary (Resonant) sounds are low pitched, hollow sounds heard over normal lung tissue.

Dull sounds are normally heard over dense areas such as the heart or liver.

Tympanic sounds are hollow, high, drumlike sounds. Tympany can be heard during percussion of the stomach and the intestine of healthy people.

Comparative percussion should be carried out on exactly symmetrical parts of the body. A certain sequence is followed in comparative percussion. Percussion sounds over the lung apices (in the front) on the symmetrical points of the chest are first compared; the pleximeter finger is placed parallel to the clavicle. The plexor finger is then used to strike the clavicle, which is used as a pleximeter in this case. During percussion of the lungs below the clavicle, the pleximeter finger is placed in the interspace at the strictly symmetrical points of the left and right sides of the chest. The percussion sounds are compared only to the level of the 4th rib along the medioclavicular line (and medially). The heart lying below this level changes the percussion sound. For comparative percussion of the axillary region, the patient should raise his arms and clamp the hands at the back of the head. Comparative percussion of the lungs on the back begins with suprascapular areas. The pleximeter finger is placed horizontally while during percussion of the regions between the scapulae, the pleximeter should be vertical. The patient should cross his arms on the chest to displace the scapulae anteriorly (away from the backbone). During percussion of the points lying below the scapulae, the pleximeter should again be horizontal; in the interspace it should be placed parallel to the ribs.

Percussion sounds of the lungs of a healthy person cannot be of equal strength, length or pitch even if the percussion blows are uniform at symmetrical

points. This depends on the mass and thickness of the pulmonary layer and also on the influence of the adjacent organs on the percussion sound. It is softer and shorter:

- over the right upper lobe because it is located somewhat below the left (due to the shorter right upper bronchus) and also because of the better-development of the muscles of the apt side of the shoulder girdle;
- in the second and third interspace on the left, because of the closer location of the heart;
- over the upper lobes of the lung (compared with the lower lobes) because of the varying thickness of pneumatic pulmonary tissue;
- in the right axillary region (compared with the left one) because of the closer location of the liver.

The cause of occurrence of a dull percussion sound

Pathological processes in the pulmonary tissue:

- Indurations in the pulmonary tissue:

- acute lobar pneumonia at the consolidation stage,
- tumour of the lung;
- tuberculosis;
- pulmonary infarct.

- Formation in the lung of a large cavity, which is filled with the inflammatory-fluid (sputum, pus, echinococcous acid);
- Obstructive atelectasis;
- Accumulation of fluid in the pleural cavity (transudate, exudate, blood);
- Pneumosclerosis and fibrous.

Pathological processes outside the pulmonary tissue:

- Over development of a subcutaneously - fatty tissue, muscles of a shoulder girdle;
- Substantial growth of heart;
- Appreciable aortectasia;
- Augmentation of lymphonoduses of a mediastinum.

The reasons of occurrence of tympanic percussion sound

- Presence of air in the pleural cavity – pheumothorax;

- Presence of a lumen filled with air and communicated with the bronchus (abscess, tuberculotic cavern) - the dimensions of a lumen should be not less than 6 cm;
- Decreased elasticity of the tense pulmonary tissues.

- lung emphysema;

- compression atelectasis;

- when the alveoli of the affected lobe, in addition to air, contain also a small amount of fluid (acute lobar pneumonia at its first and third stage, in an initial and final stage of an edema of the lung).

Variants tympanic percussion sound

- Bandbox sound - lung emphysema is of the increased airiness of the pulmonary tissue and decreased elasticity of the tense pulmonary tissues;
- Dull with a tympanic tone - the not expressed compression atelectasis and acute lobar pneumonia at its first and third stage, in an initial and final stage of an edema of the lungs;
- *Metallic sound* - very large smooth-wall cavity in the lung;
- *Cracked-pot sound* - cavity is located superficially and is communicated with the bronchus through a narrow slit.

Topographic Percussion of the Lungs.

To designate the location of the revealed normal or pathological findings it is convenient to use vertical (ordinates) and horizontal (abscissas) lines. The ribs can play the role of abscissas, the vertical lines drown through the definite points on the chest can serve as ordinates.

The lines are as follows:

1. Anterior median line (*l. mediana*) going vertically through the middle of the chest.
2. Right and left sternal lines (l. sternalis dextra et sinistra) going along the both edges of the breastbone.
3. Right and left parasternal lines (l. parasternalis dextra et sinistra) going vertically between the two above mentioned.
4. Right and left medioclavicular lines (l. medioclaviculare dextra et sinistra) going through the middle of the both collarbones.

5. Right and left anterior axillary lines (l. axillare anterior dextra et sinistra) going through the anterior edges of the armpits.

6. Right and left middle axillary lines (l. axillare medius dextra et sinistra) going through the middle edges of the armpits.

7. Right and left posterior axillary lines (l. axillare posterior dextra et sinistra) going vertically through the posterior edges of the armpits.

8. Right and left scapular lines (l. scapulare dextra et sinistra) going vertically through the angles of the shoulder blades.

9. Right and left paravertebral lines (l. paravertebrale dextra et sinistra) going vertically between the scapular line and the line going through the processes of the vertebrae.

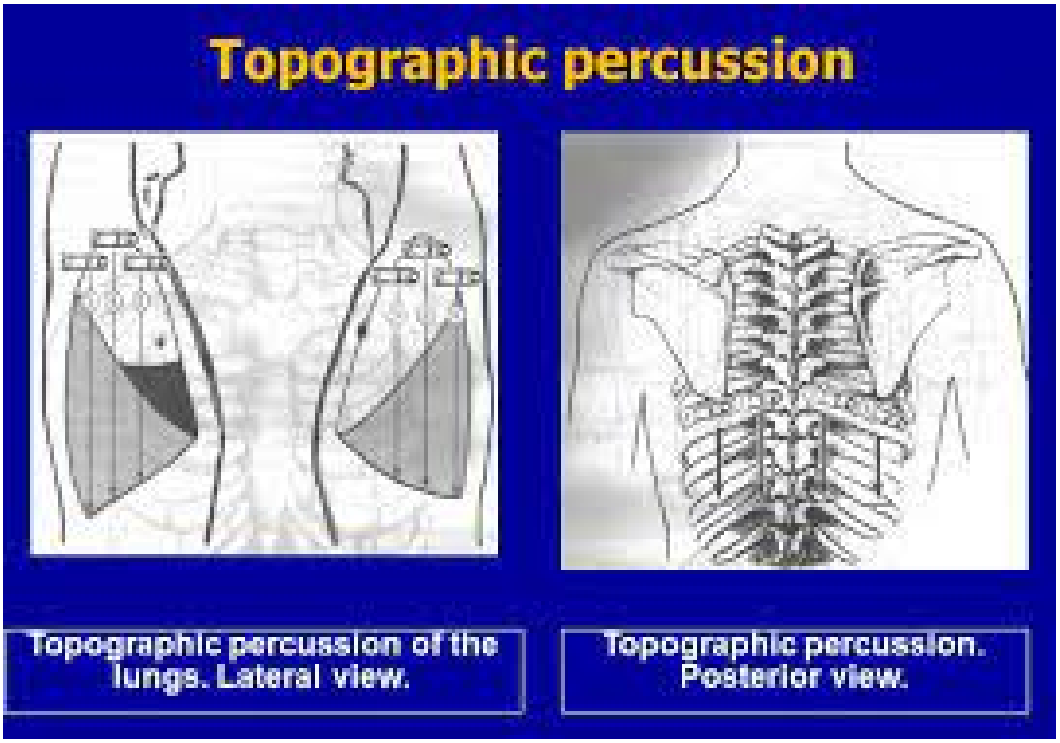

Image 7. Topographic percussion of the lungs

The image was downloaded from website https://medicalrojak.wordpress.com

Topographic percussion is used for determining: the upper borders of the lungs or the upper level of their apices and their width (Kroenigs area); the lower borders of the lungs, and variation mobility of the lower border of the lung.

The position of the *upper borders* (apices) of the lungs is determined both anteriorly and posteriorly. In order to locate the apex of the lung, the pleximeter

finger is placed parallel to the clavicle and percussion is effected from the middle upwards and slightly medially, to dullness. The upper level of the apices in healthy persons is 3 - 4 cm. The upper posterior border of the lungs is always determined by their position with respect to the spinous process of the 7th cervical vertebra. The plexímeter finger is placed over the supraspinous fosse parallel to the scapular spine and stroked from the middle. The plexímeter finger is moved gradually upward to the point located 3 - 4 cm laterally to the spinous process of the 7th cervical vertebra, at its level, and percussion is then continued until dullness. Normal height of the lung, apices (posterior) is about at the level of the spinous process of the 7th cervical vertebra.

The so-called *Kroenig's area* is a band of clear resonance over the lung apices. The width of these areas is determined by the low anterior border of the trapezius muscle and is (on an average) 5-6 cm wide, but its width can vary from 3 to 8 cm. The anterior border of the trapezius muscle divide the Kroenig area into its anterior field, which extends to the clavicle, and the posterior one that widens toward the supraspinous fossae. Light or subliminal percussion is used to determine the width of the lung apex. The plexímeter finger is held over the middle portion of m. trapezius, perpendicular to its anterior margin, and percussion is first carried out medially, and then laterally, to dullness. The distance between the points of transition of the clear pulmonary resonance to dullness is measured in centimeters.

The upper border of the lungs and the width of the Kroenig area can vary depending on the amount of air in the apices. If the amount of air is high (which may be due to emphysema) the apices increase in size and move upwards. The Kroenig area widens accordingly. The presence of connective tissue in the lung apex (which usually develops *during infiammation as* in tuberculosis or pneumonia or inflammatory infiltration) decreases the airiness of the pulmonary tissue. The upper border of the lung thus lowers and the width of the Kroenig area decreases.

To outline the *lower borders* of the lungs their percussion is carried out in the downward direction along conventional vertical topographical lines. The lower border of the right lung is first determined anteriorly along the parasternal and the medioclavicular lines, then laterally along the anterior, medial and posterior axillary lines, and posteriorly along the scapular and paraspinal lines. The lower border of the

left lung is determined only laterally, by the three axillary lines, and posteriorly by the scapular and paraspinal lines. The lower border of the left lung is not determined anteriorly because of the presence of the heart. The pleximeter finger is placed in the interspaces, parallel to the ribs, and the plexor finger produces slight and uniform strokes over it. Percussion of the chest is usually begun anteriorly, from the second and third costal interspace (with the patient in the lying or upright position). The examination of the lateral surface of the chest is performed from the axillary fossae (arm pit). The patient either sits or stands with the hands behind the back of the head. The examination ends with the posterior percussion from the seventh costal interspace, or from the scapular angle, which ends at the seventh rib.

The lower border of the right lung is as a rule at the point of transition of the clear pulmonary resonance to dullness (lung-liver border). In exceptional cases, when air is present in the abdominal cavity (e.g. in perforation of gastric or duodenal ulcer), liver dullness may disappear. The clear pulmonary resonance will then convert to tympany. The lower border of the left lung by the anterior and midaxillary lines is determined by the transition of clear pulmonary resonance to dull tympany. This is explained by the contact between the lower surface of the lung (through the diaphragm) and a small airless organ, such as the spleen and the fundus of the stomach, which give tympany (Traube's space).

Table 1

Normal Lower Border of the Lungs

Percussion point	*Right lung*	*Left lung*
Parasternal line	Upper edge of 6th rib	4th rib
Midclavicular line	Low edge of 6th rib	4th rib
Anterior axillary	Low edge of 7th rib	Low edge of 7th rib
Midaxillary line	Upper edge of 8th rib	Upper edge of 8th rib
Posterior axillary	Low edge of 8th rib	Low edge of 8th rib
Scapular line	9th rib	9th rib
Paraspinal line	Spinous process of the 11th thoracic vertebra	

The position of the border varies depending on the constitutional properties of the body. The lower border of the lungs in asthenic persons is slightly lower than in normosthenics and is found at the interspace (rather than on the rib as in

normosthenics) whereas this border is slightly higher in hypersthenic persons. The lower border of the lungs rises temporary during late pregnancy.

The position of the lower border of the lungs can vary in various pathological conditions that develop in the lungs, the pleura, the diaphragm, and the abdominal viscera. The border can both rise and lower from the normal level. This displacement can be uni- or bilateral.

Bilateral lowering of the lower border of the lungs can occur in acute and chronic dilation of the lungs (attack of bronchial asthma and emphysema of the lungs, respectively) and also in sudden weakening of the tone, of the abdominal muscles and lowering of the abdominal viscera (splanchnoptosis). Unilateral lowering of the lower border of the lungs can be due to vicarious (compensatory) emphysema of one lung with inactiva-hemiparesis of the diaphragm).

The elevation of the lower border of the lungs is usually unilateral and occurs in shriveling of the lung due to development of connective tissue (pneumosclerosis); complete obstruction of the lower lobe bronchus by a tumor which causes gradual collapse of the lung, atelectasis: accumulation of fluid or air in the pleural cavity which displace the lung upwards and medially toward the root: marked enlargement of the liver (cancer, echinococcosis), or of the spleen (chronic myeloleukaemia). Bilateral elevation of the lower borders of the lungs occurs in the presence of large amounts of fluid (ascites) or air in the abdomen due to an acute perforation of gastric or duodenal ulcer, and also in acute meteorism.

After determining the lower border of the lungs at rest, *respiratory mobility* of pulmonary borders should be determined by percussion during forced inspiration and expiration. This mobility is called active, and is usually measured by the difference in the position of the lower border of the lungs between the two extremes. Measurements are done by line axillary. The normal variation of the lower border of the lungs is 6-8cm.

The respiratory mobility of the lungs is determined as follows. The lower border of the lungs in normal respiration is first determined and marked by a dermograph. The patient is then asked to make a forced inspiration and to keep breath at the height. The pleximeter finger should at this moment be held at the lower border of the lung (determined earlier). Percussion is now continued by

moving the pleximeter downwards to complete dullness, where the second mark should be made by a dermograph at the upper edge of the pleximeter finger. The patient is then asked to exhale maximum air from the lungs and to keep breath again. The percussion is now continued in the upward direction until the clear vesicular resonance appears. The third dermographic mark should be made at the point where relative dullness is heard. The distance between the extreme marks is measured. It corresponds to the maximum respiratory mobility. The patient in a grave condition is unable to keep breath and another method is recommended to determine the respiratory mobility of the lungs.

After marking the lower border of the lung in quiet breathing, the patient is asked to make deep inhalation and exhalation without keeping breath. Percussion should be continuous during deep breathing and the pleximeter finger should gradually move downwards. First the percussion sound is loud and low during inhalation and soft and high during exhalation. Soon a point is attained where the sounds become of the same pitch and strength during both inhalation and exhalation. This point is the lower border of the lung at forced inspiratortion. The lower border at forced exhalation is determined in the same way.

Respiratory mobility of the lower border of the lungs is diminished in inflammatory infiltration or congestive plethora of the lungs, decreased elasticity of the pulmonory tissue (emphysema), profuse pleural effusion, and in pleural adhesion or obliteration.

Auscultation (L *auscultare* to listen) means listening to sounds inside to body. Auscultation is *immediate (direct)* when the examiner presses his in to the patient's body, or *mediate (indirect,* or instrumental).

Like percussion, auscultation of the lungs should be carried out according to a plan. Stethoscope or phonendoscope should be placed in strictly symmetrical points of the right and left sides of the chest. Auscultation begins with the anterior wall of the chest, from its upper part, in the supra- and subclavicular regions, and then the stethoscope should be moved downward and laterally. The lungs are then auscultated in the same order from the posterior wall of the chest and in the axillary regions. In order to increase the area of auscultation between the scapulae, the patient should be asked to cross his arms on the chest and in this way to displace his shoulder-blades

laterally from the spine, while for convenience of auscultation of the axillary regions he should place his hands on the back of the head.

The posture does not matter, but the patient should better sit up on a stool with his hands on the laps. The patient may stand, but the physician should remember that deep breathing (hyperventilation of the lungs) may cause vertigo and the patient may faint. Bearing this in mind, and also to ensure a tight contact between the stethoscope and the skin (especially if a one-piece stethoscope is used) the physician should always use his free hand to support the patient on the side opposite to the point of application of the stethoscope bell gradually increasing. This sound can be simulated by pronouncing the sound during inspiration, or by drawing tea from a saucer alveolar walls still. A shorter second phase of the vesicular breathing, which is heard only during the first third of the expiration phase, because vibrations of elastic alveolar walls are quickly dampened by the decreasing tension of the alveolar walls.

Vesicular breathing appearance is caused by vibrations of extending elastic alveolar walls, heard the whole inspiration and only during the first third of the expiration phase. Normal vesicular breathing is better heard over the anterior-surface of the chest, in the axillary regions and below the scapulae. The largest masses of the pulmonary tissue are located. While carrying out comparative auscultation, it should be remembered that the expiration sounds are louder and longer in the right lung due to a better conduction of the laryngeal sounds by the right main bronchus, which is shorter and wider. The respiratory sound sometimes becomes bronchovesicular over the right apex; or it may be mixed due to more superficial and horizontal position of the right apical bronchus.

Bronchial breathing. Respiratory sounds known as bronchial or tubular breathing arise in the larynx and trachea as air passes through the vocal slit. As air is inhaled, it passes through the vocal slit to enter wider trachea where it is set in vortex-type motion. Sound waves thus generated propagate along the air column throughout the entire bronchial tree. Sounds generated by the vibration of these waves are harsh. During expiration, air also passes through the vocal slit to enter a wider space of tie larynx where it is set in a vortex motion. But since the vocal slit is narrower during expiration, the respiratory sound becomes louder, harsher and longer. This type of

breathing is called laryngotracheal. Bronchial breathing is well heard in physiological cases over the larynx and trachea, at points of projection of the tracheal bifurcation anteriorly, over the manubrium sterni, at the point of its junction with the sternum, and posteriorly in the interscapular space at the level of the 3rd and 4th thoracic vertebrae). Bronchial breathing is not heard over the other arts of the chest because of large masses of the pulmonary tissue found between the bronchi and the chest wall.

Bronchial breathing can be heard instead of vesicular over the chest in pulmonary pathology. This breathing is called *pathological bronchial respiration*. It is conducted to the surface of the chest wall only under certain conditions, the main one being indurations of the pulmonary tissue where the alveoli are filled with effusion (acute lobar pneumonia, tuberculosis), with blood (lung infarction), and due to compression of the alveoli by the air and fluid accumulated in pleural cavity, and compression of the lung against its root (compression atelectasis). In such cases the alveolar walls do not vibrate, while consolidated airless pulmonary tissue becomes a good conductor of sound waves in laryngotracheal respiration to the surface of the chest wall. Lungs may be consolidated due to replacement of the inflammatory pulmonary tissue by connective tissue (pneumosclerosis, carnification of the lung lobe, which sometimes occurs in acute lobar pneumonia due to growth of connective tissue into the inflamed lobe of the lung).

Amforic respiration arises in the presence of a smooth-wall cavity (not less than 5-6cm in diameter) communicated with a large bronchus. Because of a strong resonance additional high overtones appear along with the main low-pitch laryngotracheal breathing. These overtones alter the man tone of the bronchial respiratory sound. Sounds of this kind can be produced by blowing over the mouth of an empty glass or clay air. This altered bronchial breathing is therefore called amphoric.

Metallic respiration differs from both bronchial and amphoric. It is loud and high, and resembles the sound produced when apiece of metal is struck. Metallic respiration is heard in open pneumotorax when the air of the pleural cavity communications with the external air.

Stenotic respiration is exaggerated laryngotracheal breathing, which is heard in cases with narrowed trachea or a large bronchus (due to a tumor); it is heard mainly at points where physiological bronchial breathing is normally heard.

Bronchovesicular or mixed respiration heard in lobular pneumonia or infiltrative tuberculosis, and also in pneumosclerosis, with foci of consolidated tissue being seated deeply in the pulmonary tissue and far from one another. Mixed breathing, when the inspiration phase is characteristic of vesicular breathing and the expiration phase of bronchial breathing, is often heard in such cases instead of weak bronchial breathing.

Bronchophony. This is the voice conduction by the larynx to the chest, is determined by auscultation. But as distinct from vocal fremitus, the words containing sounds 'r' or 'ch' are whispered during auscultation. In physiological conditions, voice conducted to the outer surface of the chest is hardly audible on either side of the chest in symmetrical points. Exaggerated bronchophony suggests consolidation of the pulmonary tissue and also cavities in the lungs which act as resonators to intensify the sounds. Bronchophony is more useful than vocal fremitus in revealing consolidation foci in the lungs of a patient with soft and high voice.

Adventitious sounds are rales, crepitation and pleural friction. Rales arise in pathology of he trachea, bronchi, or if cavern is formed in the affected lung. Rales are classified as dry (rhonchi) and moist rales.

Dry rales are rhonchi, may be due to various causes. The main one is constriction of the lumen in the bronchi. Constriction may be total (in bronchial asthma) non-uniform (in bronchitis), or local (in tuberculosis tumor of the bronchus). Dry rales can be due to:

1) spasms of smooth muscles of the bronchi during fits bronchial asthma;
2) swelling of the bronchial mucosa during its inflammation;
3) accumulation of viscous sputum in the bronchi which adheres to the wall of the bronchus its narrows its lumen;
4) formation if fibrouse tissue in the walls of separate bronchi and in the pulmonary tissue with subsequent alteration of their architectonics (bronchiectasis, pneumosclerosis);
5) vibration of viscous sputum in the lumen of large and medium size bronchi during inspiration and expiration: being viscous, the sputum can be drawn (by the aire stream) into threads, which adhere to the opposite walls of the bronchi and vibrate like strings.

Dry rales are heard during inspiration and expiration and vary greatly in their loudness, tone and pitch. According to the quality and pitch of the sounds produced, dry rales are divided into sibilant (high-pitched and whistling sounds) and sonorous rales (low-pitched and sonoring sounds). High-pitched rales are produced when the lumen of the small bronchi is narrowed, while low-pitched sonorous rales are generated in stenosis of medium calibre and large calibre bronchi or when viscous sputum is accumulated in their lumen.

Moist rales are generated because of accumulation of liquid secretion (sputum, edematous fluid, blood) in the bronchi through which air passes. Air bubbles pass through the liquid secretion of the bronchial lumen and collapse to produce the specific cracking sound. This sound can be simulated by bubbling air through water using a fine tube. Moist rales are heard during both the inspiration and expiration but since the air velocity is higher during inspiration moist rales will be better heard at this respiratory phase.

Depending on the calibre of bronchi where rales are generated, they are classified as fine, medium and coarse bubbling rales. Fine bubbles rales generated in fine bronchi and are percepted by the ear as short multiple sounds. Kales originating in the finest bronchi and bronchioles are similar to crepitation from which they should be differentiated. Medium bubbles rales are produced in bronchi of a medium size and coarse bubbles rales in large caliber bronchi, in large bronchiectases, and in pulmonary cavities (abscess, cavern) containing liquid secretions and communicating with the large bronchus. Large bubbling rales are characterized by a lower and louder sound.

Moist rales originating in superficially located large cavities (5-6 cm and over in diameter) may acquire a metallic character. If segmentary bronchiectases or cavities are formed in the lung, rales can usually be heard over a limited area of the chest. Chronic bronchitis or marked congestion in the lungs associated with failure of the left chambers of the heart is as a rule attended by bilateral moist rales of various calibre, which occur at the symmetrical points of the lungs.

Crepitation. As distinct from rales, crepitation originates in the alveoli. Some authors erroneously classify these, sound as crepitant and subcrepitant rales. Crepitation is a slight crackling sound that can be imitated by dubbing a lock of hair. The main condition for generation of crepitation is accumulation of a small

amount of liquid secretion of the alveoli. During expiration the alveoli stick together, while during inspiration the alveolar walls separated with difficulty and only at the end of the inspiratory movement. Crepitation is therefore only heard during the height of inspiration. In other words, crepitation is the sound produced by many alveoli during their simultaneous reinflation.

Crepitation is mainly heard in inflammation of the pulmonary tissue, at the first (initial) and third (final) stages of acute lobar pneumonia, then the alveoli contain small amounts of inflammatory exudate, in pulmonary tuberculosis, lungs infarction, and finally in congesons that develop due to insufficient contractile function of the left ventricular myocardium or in marked stenosis of the left venous orifice of it heart and in compressive atelectasis. By its acoustic properties, crepitation can often resemble moist fine lies that are produced m fine bronchi or bronchioles filled with liquid secretion. Differential diagnostic or these rales and crepitation are as follows: moist fine rales are heard during both inspiration and expiration; they can be intensified had disappear after coughing, while crepitation can only be heard at the height of inspiration or not does it change after coughing.

Pleural friction sound. In physiological conditions visceral and parietal layers of the pleura are constantly "lubricated" by a capillary layer of noiseless. Various pathological conditions alter the physical properties of the pleural surfaces and their friction against one another becomes more intense to generate a peculiar adventitious noise, known as the pleural friction sound. Fibrin is deposited in inflamed pleura to make its surface through; moreover, cicatrices, commissures, and bands are formed between pleural layers at the focus of inflammation. Tuberculosis or cancer are also responsible for the friction sounds.

Pleural friction sounds are heard during both inspiration and expiration. Intensity, or loudness, length, and over which they are heard differentiate the sounds. During early dry pleurisy the sounds are soft can be imitated by rubbing silk or fingers in the close vicinity of the ear. The character of pleural friction sound is altered during the active course of dry pleurisy. It can resemble crepitation or fine bubbling rales sometimes crackling of snow. In pleurisy with effusion, during the period of rapid resorption of exudate, the friction sound becomes coarser due to passive deposits on the pleural surfaces. The time during which pleural friction sound

can be heard varies with diseases. For example, in rheumatic pleurisy pleural friction is only heard during a few hours; after a period of quiescence it reappears. Pleural friction persists for a week and over in dry pleurisy of tuberculous etiology and pleurisy with effusion at the stage of resorption. Pleural friction sounds can be heard in some patients for years after pleurisy because of large cicatrices and roughness of the pleural surfaces.

Pleural friction sounds can be differentiated from fine bubbling rales and crepitation by the following signs:

1) the character of rales is altered or rales can disappear for a short time after coughing, while pleural friction sound does not change in these conditions;

2) when a stethoscope is prested tighter against the chest, the pleural friction sound is intensified, while rales do not change;

3) crepitation is only heard at the height of inspiration;

4) if a patient moves his diaphragm in and out while his mouth and nose are closed, the sound produced by the friction of the pleura due to the movement of the diaphragm can be heard, while rales and crepitation cannot because there is no air movement in the bronchi.

Instrumental 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 lung 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 bronchial 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.

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.

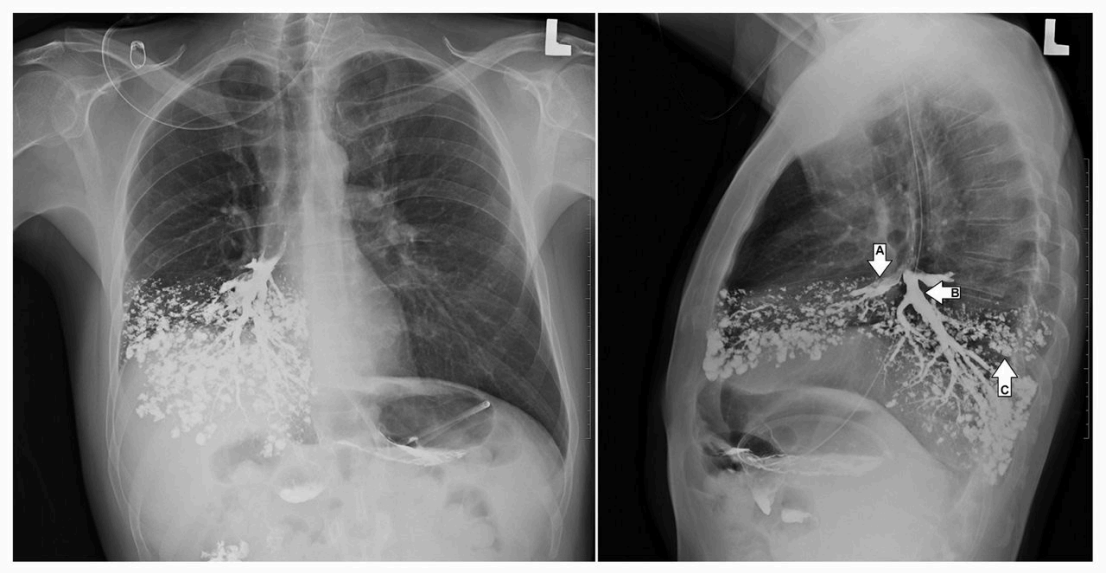

Image 8. Accidental barium Bronchography

Radiographs of the posteroanterior (left image) and lateral (right image) chest showing barium bronchograms of the right middle (arrow A) and right lower (arrow B) lobe bronchi. Extensive tree-in-bud opacifications in the right middle and lower lobes are indicative of bronchiolar filling (arrow C).

The image was downloaded from website https://www.cmaj.ca/

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.

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): $MV = f \cdot RV$. 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 CO2 absorption system. Obtained spirogram compared then with spirogram that records with apparatus filled with O2.

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. O2 content in units of volume;

2. oxygen capacity of the blood (the amount of O2 that can bound by a blood unit);

3. percentage of O2 saturation of the blood (95% in norm);

4. partial pressure of O2 in the blood (90-100 mm Hg in norm);

5. CO2 content in arterial blood (about 48% v/v);

6. partial pressure of CO2 (about 40 mm Hg in norm).

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 s*putum 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

Microscopic study

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

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 arefound 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.

THE BRONCHOOBSTRUCTIVE SYNDROME

Bronchoobstructive syndrome (BOS) is a collective term that includes a number of symptoms of clinical manifestations of bronchial obstruction with underlying narrowing or occlusion of the airway. Clinically severe bronchoobstructive syndrome is most common in children, especially young children, but it is not a rare disease among the adult population. Its emergence and development is influenced by various factors, primarily respiratory viral infection. Early diagnosis and treatment of BOS in a therapeutic practice can significantly reduce the number of complications of the disease, improve survival and quality of life of patients.

The term "BOS" can not be used as an independent diagnosis. BOS is a symptom complex of any disease, the etiology of which is necessary to determine in all cases of the development of bronchial obstruction.

There are primary and secondary bronchial obstruction:

1. Primary bronchial obstruction (mandatory) is specific for COPD and BA.

2. Secondary (optional) bronchial obstruction can be observed in acute inflammatory pathology (ARVI, acute bronchitis, pneumonia) and in different forms of chronic internal pathology (pulmonary sarcoidosis, fibrosing alveolitis, pneumoconiosis, pneumomycosis, parasitic lung disease, bronchiectasis, cystic hypoplasia, mucoviscidosis, lung tumors, systemic connective tissue diseases, etc.).

The main pathogenic variants of the development of bronchial obstruction:

Spastic is a frequent option of BOS (> 70% of all cases) caused by bronchospasm (bronhiolospasm) due to dysfunction in the systems of bronchial tone control.

Inflammatory is the mechanism caused by edema of alveolar-capillary membrane, infiltration of the airways and hyperemia of the bronchial mucosa (the obstruction formed by mucous plugs).

Dyscrinic is due to the disturbances of mucociliary transport (stimulation of goblet cells` enzymes and the bronchial glands), deterioration of sputum properties (increase in the ratio of gel over sol).

Allergic is due to hyperreactivity of the bronchial tree, edema and inflammation of the bronchial mucosa with the obstruction formed by mucous plugs.

Dyskinetic is when bronchial passability is disrupted due to congenital hypoplasia of the membranous part of trachea and bronchi, promoting the closing of their lumen during inspiration.

Emphysematous is accompanied by the collapse of the small bronchi due to a decrease and loss of lung elasticity.

Hemodynamic is developed secondarily in violation of hemodynamics of the pulmonary circulation: hypertension of the pre- and postcapillaries, stagnation in the bronchial veins, as well as in hypertensive crisis.

Hyperosmolar is observed because of decreasing the humidity of the mucous membranes of the bronchi (breathing cold air).

The luminal narrowing of the bronchial tree in BOS is manifested by airflow limitation and diagnosed based on complaints, anamnesis, physical examination (palpation, percussion, auscultation), laboratory studies (general blood and sputum tests), X-ray data and methods of study of external respiratory function (peak flow measurement, spirometry, body plethysmography).

BOS classification is based on the etiologic basis, the duration of the course and spirometry data.

Based on the etiological basis:

- infectious (AR, bronchitis, COPD, pneumonia, bronchiectasis)
- allergic (BA, aspergillosis)
- hemodynamic (cardiovascular diseases);
- obstructive (airway foreign bodies).

Based on the duration:

- acute (up to 10 days)
- lingering (more than 10 days);
- recurrent.

According to the spirometry data:

- mild;
- moderate;
- heavy;
- hidden obstruction

BOS diagnosis is based on key clinical symptoms:

- cough (paroxysmal dry or productive) is the earliest sign;
- expiratory dyspnea (whistling expiration with lengthening > 5 sec. in time with the participation of accessory muscles of respiration;
- sudden attacks of breathlessness or shortness of breath, feeling a lack of air (respiratory discomfort), agitation, sweating;
- remote rales (dry, moist) (especially when you exhale);
- palpitations;
- forced half-sitting position.

Anamnesis:

- assosiation with frequent respiratory viral infections, especially transferred in childhood (bronchiolitis, ARVI, influenza, sinusitis);
- smoking (active or passive);
- professional factors (fumes, dustiness, etc);
- physical activity;
- strong psycho-emotional stress;
- positive allergic history;
- hereditary predisposition;
- uncontrolled intake of medications (NSAIDs, β-blockers, ACE inhibitors, amiodarone, sulfonamides).

Physical examination data

Medical examination: diffuse cyanosis, increased RR, especially with physical activity; large barrel-shaped (emphysematous) chest (the extension of intercostal spaces and their retraction during breathing), participation of accessory respiratory muscles in the act of respiration (intercostal and abdominal).

Palpation: the weakening of voice tremor in symmetrical areas of the chest; chest stiffness.

Percussion: bandbox percussion sound above lungs, an increase of the tops height and expansion of Kroenig`s fields, shift down lower lung fields, reduced active and passive mobility of the lungs, decrease or absence of the absolute cardiac dullness area.

Auscultation: diminished breath sounds, impaired vesicular breathing with extended exhalation (> 5 sec.); dry whistling or low-pitched moist rales; bronchophony is sharply weakened or not heard.

Methods of BOS medical imaging:

1. Radiography: increased lung transparency (symptom of emphysema), flattened and low standed diaphragm, expanded intercostal spaces, narrow cardiac silhouette, increased retrosternal area; bullous emphysema and signs of pulmonary heart may be determined.

Image 9. Spirometry

The image was downloaded from website https://www.bfwh.nhs.uk

2. Spirometry with the analysis of the "flow - volume" curve is the study of indicators of the external respiration function (ERF). Integral and the most sensitive indicators at the BOS are the amount of forced expiratory volume in the first second (FEV1) and the modified Tiffeneau index (ratio of FEV1 to the forced vital lung capacity (FVC). The bronchial obstruction is stated if FEV1 is less than 80% of the proper value, and Tiffeneau index is less than 0.7.

PHYSICAL AND INSTRUMENTAL METHODS OF THE EXAMINATION OF THE CARDIOVASCULAR SYSTEM. THE INTERVIEW AND THE GENERAL EXAMINATION OF THE PATIENT WITH THE DISORDER OF THE CARDIOVASCULAR SYSTEM. PHYSICAL METHODS OF THE EXAMINATION. LABORATORY AND INSTRUMENTAL TESTS IN CARDIOLOGY AND RHEUMATOLOGY

Main complaints of the patients with heart diseases are pains in the heart area, palpitation, dyspnea, edema. It is important to know every detail.

Pain is an important clinical sign. The patients are not usually able to describe the peculiarities of the attack of pain. The physician should strictly follow the algorithm of the complaint study.

The following can be assessed:

1) Character
2) Localisation
3) Irradiation
4) Provocation
5) Relieving movements

The most significant and frequent cause of pain in the precordial area associated with a heart disease is myocardium hypoxia resulting from discrepancies between the requirements of the cardiac muscle in oxygen and capabilities of the coronary circulation to meet them.

This pain develops in angina of effort, rest angina, myocardial infarction and may be the only sign of the disease. The pain is typical and the correct questioning allows making a diagnosis.

It is necessary to establish the place of pain. The patient should be asked to show with his finger the place or the region of pain. Typical anginal pain is localized behind the breastbone, commonly behind its body, sometimes behind its lower third, behind the xiphoid process, in the epigastric area and the manubrium. The pain rarely begins on the left of the sternum in the area of the heart apex. Typical anginal pains in the area of the left scapula, in the interscapular region, under one or both collarbones, in the left radiocarpal joint, jaw have also been reported.

Radiation of the pain is noted in 50 % of the patients with coronary artery disease. The pain usually radiates to the left arm at a various distance distally along the inner surface of the arm and forearm, to the elbow, wrist, 5th and 4th fingers, frequently the pain radiates to the both extremities and very rarely the pain is felt in the right upper extremity, the left or right side of the neck, lower jaw, chin, pharynx, ear, abdomen, lumbar area. Unusual radiation of the pain may suggest myocardial infarction.

The character of the pain is diagnostically significant: in coronary artery disease the pain is squeezing, pressing, the patient complains of feeling of weigh or pressure preventing free breathing behind the breastbone. The phenomenon of "breathing deceleration", when the patient stops on walking, is equivalent to angina. The pain is sometimes accompanied by feeling of fear, horror (angor praecordialis).

The conditions in which the pain develops are frequently decisive for making diagnosis: the pain is caused by the factors, which increase the requirement of the heart muscle in oxygen. The attack usually occurs with physical exertion and is quickly relieved by rest. The pain may appear on any physical exercise, but most frequently on fast walking for a long time, on walking upstairs, with cold, wind, after meals, emotions. The pain in light cases develops on walking upstairs, running, quick walking. In medium-severity cases the pain develops during the first 300 meters of walk. In severe cases the pain may develop on slow walking. The pain decreases and even disappears after stopping. In some cases the pain disappears suddenly, in the others gradually, sometimes not simultaneously behind the breastbone and the places of radiation. The attack of angina frequently develops after a quiet sleep. But sometimes the attack of pain develops in a lying position, especially at night (decubital angina). This attacks are longer (30 min and more), intensive, the pain decreases when the patients tries to sit up. Frequently these attacks appear in hypertensive patients, in cor pulmonale, valve defect, heart failure, they are associated with increase of minute heart volume in a lying position. An important sign of angina if the pain is relieved with nitroglycerin.

Anginal pain - stenocardia, occurs in ischemic heart disease. Usually caused by physical activity (walk, walk up the hill) or by excitement. It is worsened by cold, e.g. transition from warmth to cold. After stopping of effort the pain eases

off. It may happen also during inactivity. It is compressive, seizing or stinging flat pain localised behind the sternum, sometimes in the whole precordium. It irradiates into the neck, lower jaw, left shoulder, ulnar side of the left arm, but also into the back and epigastrium. It lasts a few minutes (5- 10, angina pectoris), but if it lasts more than 20 minutes and it happens during inactivity, it is necessary to consider myocardial infarction. (The intensity of pain is then higher, the pain is usually accompanied by vegetative reactions, such as nausea, anxiety, and sweating). The effect of nitrates given sublingually in angina pectoris should appear in 5 minutes, if the pain lasts longer; myocardial infarction should be suspected.

Pericardial pain is sharp, localised rather precordially; it has long-lasting duration. It worsens after changing position and is dependent on breathing. It is usually not related to physical activity, easing of pain occurs when sitting or bending forward. It is usually caused by viral infection, myocardial infarction and following cardiosurgical operation.

Pain in dissection of aortal aneurysm is very sharp, intensive, myocardial-infarction-like-pain, irradiating to the back or abdomen, happens suddenly, as "snap of a whip", often after effort (example: lifting the weight).

Functional precordial pain is stinging, pricking pain, localised to the area of the heart apex, occurring in inactivity or in psychical activity in young people; it is often accompanied by the feeling of impossibility to breathe enough air. Within the frame of differential diagnostics of chest pain, it is necessary to exclude extracardiac causes, mainly vertebrogenic ones, intercostal neuralgia, and pain in gastrooesophageal reflux disease.

Palpitations are unpleasant, intensively perceived manifestations of heart activity characterised mainly by:

- short-term irregular pulse;
- "skipped" heart beats;
- feeling of "short-term stopped beats";
- fast regular heart pounding (paroxysmal tachycardia);
- fast irregular pounding heartbeat (atrial fibrillation).

Tachycardia and palpitation are different phenomena. Increased heart rate is not felt by healthy persons. Sensation of heartbeat usually accompanies accelerated

and increased heart contractions, this is frequently observed in persons with increased nervous sensitivity on pronounced physical load, quick walking upstairs, drinking large amounts of black coffee, alcohol, smoking. These are the cases of physiological palpitation. It is observed in asthenic constitution, in flatulence, aerophobia, phrenic hernia, vegetovascular dystonia.

Palpitation is a frequent sign of aortic valve incompetence, mitral stenosis, hyperthyrosis, Itsenko-Cushing disease, hypertension, anemia, fever.

Palpitation accompanies different disorders of the rhythm. Solitary beats at various intervals, sometimes one, two, three, and more beats, accompanied by feeling of sinking heart, arrest, lack of air are characteristic for preliminary heart contractions, i.e. extrasystolic arrhythmia, which is often is not a consequence of anorganic heart disease but occurs in regulation disorders. It is said that the more unpleasant is the feeling of extrasystole, the more probable is their functional origin.

Short or long attacks of palpitation can result from paroxysmal tachycardia or atrial flatter. Paroxysmal supraventricular or ventricular tachycardia occur with frequency >150 beats per minute in an attack-like manner with a distinct onset and end, regular heart activity; they last form several minutes to hours and even days. The attacks appear at various intervals.

The rhythm of paroxysms of atrial flatter is irregular, the diagnosis is easily made by objective study during the attack. During the attack-free period the patient can be asked to illustrate the heart activity tapping on a hard surface with a finger. It should be emphasized that diagnostic significance of the complaint on palpitation is decreased by the frequency of this sensation in healthy persons and in those without cardiac diseases as well as the fact how often the patient does not have unpleasant sensations even in marked disorders of the rhythm (constant ciliary arrhythmia, frequent extrasystole caused by organic heart diseases) as well as powerful beats of the enlarged heart causing thrill of the whole anterior chest wall.

Dyspnea (dyspnoe, breathlessness) difficult breathing accompanied by sensation of lack of air, increased respiratory effort, which are evident to the patient.

Normal breathing is not accompanied by difficulties and unpleasant sensations even when it is accelerated due to exercise or emotions (tachypnoe, hyperpnoe).

It can be only a subjective symptom, is discovered by history taking, and thus depends on the ability of the patient to describe his/her sensations. The sensation of dyspnea is associated with participation of the auxiliary muscles in the act of respiration, which appears after minute respiratory volume exceeds a definite critical value, dyspnea threshold. A healthy person feels dyspnea when minute respiratory volume increases 4 times, >30 % of the vital capacity of the lungs participate in respiration (in normal conditions this is 10-20 %). Anaerobic metabolism prevails in the muscles and excess of lactic acid results in metabolic acidosis, the cause of centrogenic dyspnea.

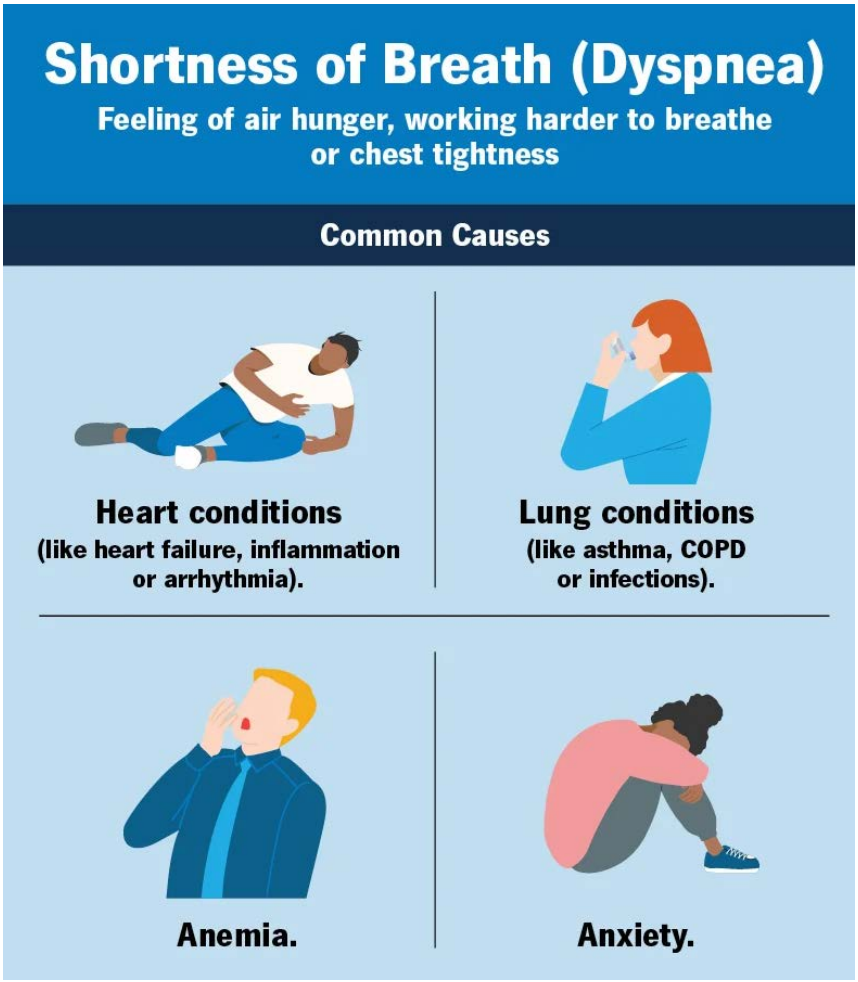

Image 10. Dyspnea, or shortness of breath

The image was downloaded from website https://my.clevelandclinic.org

The amount of physical load at which the patient lacks air depends on the age, sex, body weight, physical development, the state of the nervous system, etc. Dyspnea of effort may disappear in healthy persons with gradual increase of physical load, reduction of the body weight.

Breathlessness is a serious clinical sign of left heart insufficiency in ischemic heart disease, hypertension and valve defects. It manifests during exertion, or inactivity, and can be paroxysmal. The patients with heart diseases reach dyspnea threshold on significantly lower physical exercise then healthy ones. Cardiac dyspnea appears at blood congestion in the pulmonary veins resulting in reduction of the pulmonary tissue elasticity. Various factors may lead to increase in the blood volume in the pulmonary system: disturbances in blood outflow from the pulmonary veins to the left atrium and left ventricle at narrowed left atrioventricular orifice, reduction of diastolic relaxation of the left ventricle in constrictive pericarditis as well as in decreased blood ejection from the left ventricle at decreased contractile function of the left ventricle. It is classified according to NYHA criteria (New York Heart Association):

- NYHA class I - ordinary physical activity does not cause breathlessness or anginal pain; it is caused only by enormous load.
- NYHA class II - ordinary physical activity causes breathlessness or anginal pain (but tolerance of ordinary daily activity is good).
- NYHA class III - small physical activity (slow walk on the level, dressing up, toilette) causes breathlessness, but without difficulties during inactivity.
- NYHA class IV - manifestation of breathlessness in inactivity.

Thus, the following types of dyspnea associated with heart disease can be distinguished:

1) exercise dyspnea;
2) orthopnea, decubital dyspnea (in decubitus);
3) constant dyspnea;
4) fit-like dyspnea:

a) short attacks of nocturnal dyspnea;
b) cardiac asthma;
c) acute pulmonary edema.

Paroxysmal breathlessness (fit-like dyspnea)

The term "cardiac asthma" is frequently used to define this type of dyspnea as this is associated with cardiac diseases and manifests by respiration disturbances and physical findings characteristic for bronchial asthma (pronounced expiration dyspnea, wheezing, distance rales, mucous sputum discharge, sometimes slightly stained with blood, the patient takes a characteristic forced position (sitting, resting the hands on the chair, knees, bed). Cardiac asthma originates in acute left heart insufficiency, which leads to the gathering of blood (haemostasis) in lungs. The patient wakes up by a feeling of a lack of air approximately 2-3 hours after falling asleep; moves to orthopnia position, breathlessness can ease off (decreasing of vein return) or can progress further more.

Pulmonary edema (caused by a leakage of fluid into the interstitium and alveoli). Increase of pressure in the pulmonary capillaries is the most important factor of pulmonary edema development: elevation of the pressure in the pulmonary capillaries above the level of oncotic pressure of the blood proteins (25 mm Hg) causes fluid transudation from the pulmonary capillaries to the alveoli. But the correlation between the pressure value in the pulmonary capillaries and pulmonary edema does not always exist. A role is played by other factors, which increase vascular wall permeability, sensitivity of the respiratory center. It is marked by extreme breathlessness, anxiety, raucous breathing, often well audible from distance (resembles gurgling), presence of pinkish fluid in oral cavity. It occurs at night; in mitral stenosis at the time of the maximum exertion.

Blood congestion in the pulmonary system causes cough. It frequently occurs in decubitus and awakes the patient. But it may develop at daytime, on physical load or after meals, occurs in attacks lasting from several minutes to more than an hour, frequently appears with dyspnea, accompanying the attack of cardiac asthma or pulmonary edema. The cough is dry at first then it is accompanied by some amount of mucous viscous discharge sometimes with traces of blood.

The cough may be a sign of trachea or bronchi compression with aortic arch aneurysm, in this case it becomes barking, loud, coarse. Similar cough appears at compression of the left recurrent nerve and bronchi with dilated pulmonary artery, enlarged left atrium in mitral stenosis. Hemoptysis can be a sign of lung infarction

resulting from embolism of the branches of the pulmonary artery or their thrombosis. The amount of blood coughed up can be large (haemoptoe) and blood can be mixed to the sputum (haemoptysis). Sometimes the cause of hemoptysis is rupture of varicose bronchial veins and collaterals between the bronchial and pulmonary veins in the submucous membrane of the bronchi.

Discharge of the foamy pink sometimes bloody sputum is typical for pulmonary edema.

Hemoptysis can be associated with atherosclerosis of the intrapulmonary branches of the pulmonary artery or bronchial arteries, with primary atherosclerosis of the pulmonary artery, pulmonary hypertension in congenital heart defects.

Hemoptysis:

- Caused by stasis - manifests in mitral stenosis due to rupture of endobronchial collaterals.
- Caused by lung infarction - is recognised expectoration of dark red blood, simultaneous breathlessness, and pleural pain.
- Caused by lung edema - expectoration of pinkish sputum in acute left heart insufficiency.

Syncope is short-term unconsciousness lasting several minutes, caused by insufficient perfusion of the brain.

Cardiac syncope:

- Caused by arrhythmia - extreme tachyarrythmia and bradyarrhythmia cause sudden decrease of minute volume (Adam-Stockes syndrome is the name of syncope occurring in temporary asystolia or ventricular tachycardia)
- Caused by aortal stenosis - manifests in during or following exertion. Syncope is caused by a limited blood flow through stenosis.
- Caused by obstruction of the mitral orifice - due to - myxoma or a big thrombus in the left vestibule; atrium; occurrence of syncope depends on position or exertion.

Circulatory syncope

Orthostatic syncope - may occur when standing, due to accumulation of blood in lower limbs, it is associated with a defect of baroreceptors. Dehydration,

varicose veins in lower limbs, or medication (diuretics, hypotensives and nitrates) are contributing factors.

Vasovagal syncope - manifests usually in healthy people is related to pain, fear, hunger, or stuffiness. Syncope happens suddenly; after a fall or being horizontally positioned, the consciousness recovers quickly.

The syndrome of carotic sinus is manifested by irritation of carotic sinus especially in sensitive people; it leads to bradycardia, hypotension, and unconsciousness. It is caused by head turning or moving back, or by a pressure of a tight collar.

Extracardiac syncope is usually caused by arteriosclerosis, coughing, or hyperventilation.

Cyanosis is classified to central and peripheral - for further details see section "Complete examination". Mixed cyanosis, combination of central and peripheral, manifests in left heart insufficiency.

Headache in the occipital area appears frequently in the morning in hypertensive patients and those with atherosclerosis of the brain vessels. Headache is a frequent sign of aortic heart defects. This sign suggests disturbances of cerebral circulation. Sleep disorders are frequently caused by blood congestion in the pulmonary system and may appear earlier than the patients faints or feels dyspnea.

Dizziness (vertigo) may occur in atherosclerosis of the vessels supplying the brain and vestibular apparatus, blood congestion in the brain vessels as well as be the consequence of vagotonic vascular reactions.

Nosebleed (epistaxis) may develop in arterial hypertension, rheumatism, bacterial endocarditis.

Punctate hemorrhages (petechid) and large subcutaneous hemorrhages (ecchymoses) also appear in bacterial endocarditis, chronic heart failure, atherosclerosis.

Thirst is a frequent sign of fluid retention in patients with developing cardiac failure.

Thirst may be accompanied by **edema.** Cardiac edemas appear on the feet and legs, are bilateral, at first they are felt as tight footwear, then the patients notice imprints of laces, socks, footwear on the legs, later they notice edema of the lower

legs, dorsal surfaces of the feet and lower legs. The edema appears in the evening at first and subsides by the morning, later it becomes constant and rises cranially, usually up to the umbilicus, but may involve higher regions in advanced stages of cardiac failure.

Development of cardiac edemas is associated with decreased secretion of atrial natriuretic factor, which is accompanied by retention of sodium and water as well as increased hydrostatic capillary pressure. Secondary hyperaldosteronism due to blood congestion in the liver and cardiac cirrhosis and increased production of antidiuretic hypophyseal hormone, ACTH, and glycocorticoids also play a role.

Edema is a frequent sign of other diseases (those of kidneys, vessels, and thyroid gland, tumors) that is why this complaint advocates for thorough history taking.

The Pulse and Arterial Pressure Study

The most frequent place to **study the pulse** is the radial artery as it is located superficially under the skin between the styloid process and the tendon of the inner radial muscle. The topography of the radial artery allows to press the vessel to the bone, which facilitates the study of the pulse. The hand of the patient is held with the physician's right hand in the area of the radioulnar joint, the thumb of the physician should be on the elbow side, the fingers on the radial side. After the artery is felt it is pressed with the point and middle fingers. When the wave passes the artery, the physician feels dilation of the artery, that is the pulse.

Then it is necessary to determine whether the pulses are equal on the both hands. Normally they are equal. If the pulses are unequal, this is called *pulsus differens.* Pulsus differens in observed in anomalies of the radial artery (it goes to the back side of the hand and the usual place is occupied by its branch), in pathological changes: aortic arch aneurysm, mediastinal tumors, narrowing of the left atrioventricular orifice when enlarged left atrium presses the subclavicular artery and the pulse of the left hand, especially in the left decubitus, decreases (Popov-Saveliev sign), when a tumor or enlarged lymph nodes compress the artery, when the lumina of the large vessels are compressed with scars.

After comparison of the pulse on the both hands, it is necessary to study the properties the pulse on one hand. If the pulse is different on the both hands, it is studied on the hand where it is more intensive.

The following properties are to be determined. **Pulse rate**, the number of pulse beats per minute. In healthy individuals pulse rate is 60-80 beats per minute. Rapid pulse (pulsus frequens, may be due to physiological conditions. In women the pulse rate is 7-8 beats more than in man, the pulse accelerates with physical work, excitement, during digestion, on breathing in, in persons over 60, in some diseases (it increases by 8-10 beats per each degree of the body temperature, in thyrotoxicosis, anemia, acute and chronic diseases of the heart, endocarditis, myocarditis, pericarditis, cardiac failure, after taking some drugs and poisons, such as alcohol, atropine, caffeine, adrenaline). In typhoid fever at fever of 40 degrees the pulse may be 76-80 per minute (relative bradycardia), in tuberculous meningitis due to excitation of the vagus nerve under the influence of increased intracranial pressure bradycardia may be observed.

Pulse rate disorders: a) rapid; b) rare. In healthy subjects a rare pulse (pulsus rarus, <60) is not frequent, chiefly observed in sleep. Pulse deceleration is observed in the following pathological conditions: complete atrioventricular blockage, stenosis of the aorta orifice, cachexia, hanger, jaundice, cerebral hemorrhage, brain tumors, fracture of the skull, myxedema.

Rhythm of the pulse, the beats follow with equal intervals and are equal, i.e. regular pulse (pulsus regularis). In disturbances of the heart function, this regularity changes, it becomes arrhythmical, irregular, an irregular pulse (pulsus irregularis). Three types of arrhythmias are observed: extrasystole (extraordinary heart contractions), the interval between this and the following contraction can be unusually long (compensatory pause), ciliary arrhythmia (disordered pulse waves are palpated), paroxysmal tachycardia (very frequent pulsation which is difficult to count, appearing and disappearing suddenly).

If the pulse is arrhythmical, it is necessary to determine if the number of the pulse waves corresponds to the number of the heart contractions. The difference between the number of the heart contractions and pulse waves per one minute is termed *pulse deficiency*, the pulse is called *a deficiency pulse* (pulsus deficiens). The

more is the deficiency, the more unfavorable is its effect on the blood supply of the organs and tissues. Pulse rhythm disorders: a) extrasystole; b) bigeminal pulse; c) ciliary arrhythmia.

Pulse tension is the pressure of the blood exercised on the wall of the artery. It is determined by the force, which should be exercised to compress the artery completely in order to arrest the blood flow in it. This property of the pulse gives the information about the state of the vascular system and the arterial pressure. In healthy persons the pulse tension is satisfactory. In a tense pulse, the force of compression to arrest the pulse wave should be great *(pulsus durus)*, this is a sign of hypertension of various origin or arterial sclerosis. Reduction of tension, *soft pulse (pulsus mollis)* suggests decreased arterial pressure (reduction of the heart contractile function, shock, collapse, blood loss).

Pulse filling is the amount of blood in the vessel. This property is most difficult to determine, namely according to the maximum and minimum volume of the vessel (how the diameter of the vessel changes in the period of dilation and collapse). To do this, proximal fingers on the radial artery should press the vessel gradually, the distal finger determines its maximum filling. In healthy persons the pulse is satisfactory. In reduction of the volume of circulating blood (blood loss, shock, collapse), disturbances of contractile function of the heart, the pulse filling decreases, *pulsus vacuus,* in increased volume of the circulating blood, blood filling increases, *full (strong) pulse* (pulsus plenus). Pulse filling and tension give similar information.

Pulse value is a collective concept, uniting such properties as filling and tension. It depends of the degree of the artery widening during systole and its collapse during diastole. In healthy persons the pulse is sufficient. With the increase of the stroke blood volume, great fluctuations of the arterial pressure as well as decreased tone of the arterial wall, the value increases, *pulsus magnus;* in insufficiency of the aortic valve, thyrotoxicosis, fever, the tone of the aorta wall decreases. Reduction of the stroke volume, increased tone of the arterial wall reduces the number of pulse waves, *small pulse (pulsus parvus).* This is observed in stenosis of the aorta opening, mitral stenosis, tachycardia, heart failure; in shock, massive blood loss the pulse is poorly felt, *thready pulse (pulsus filiformis).*

The shape or rate of the pulse is the rate of dilation and the following contraction of the artery. This property depends of the rate of the pressure changes in the arterial system during systole and diastole. In aortic valve incompetence, *an abrupt pulse (pulsus celer)* or *a bouncing pulse (pulsus silens)* as well as *pulsus altus:* the stroke blood volume and systolic blood pressure are increased, during diastole the pressure drops quickly as the blood returns from the aorta to the left ventricle can be present.

Slow pulse (pulsus tardus) is opposite to an *abrupt pulse.* This is associated with slow increase of the blood pressure in the arterial system and its small fluctuations during a cardiac cycle. This is observed in stenosis of the aorta opening. Due to reduction of the pulse waves it is not only slow but also small (pulsus parvus). Pulse shape disorders: a) bouncing pulsus magnus; c) slow small pulse.

Dicrotic pulse (pulsus dicroticus) is a second additional wave after reduction of a normal pulse wave. In healthy subjects it is not palpated but registered on sphygmogram. A dicrotic pulse is present in reduced tone of the peripheral arteries (fever, infections, severe pneumonia).

An alternating pulse (pulsus alterans) is alterations of large and small pulse waves when the pulse is rhythmical (severe affection of the myocardium, i.e. myocarditis, cardiomyopathy.

A paradoxical pulse (pulsus paradoxus) is reduction of the pulse waves during breathing in (in adhesion of the pericardium layers due to compression of the large veins and reduction of the heart filling during expiration.

In addition to arterial, capillary and venous pulses are also distinguished. *Capillary pulse* is observed in insufficiency of the aortic valve, sometimes in thyrotoxic goiter. It is determined in the following way: it necessary to press the tip of the nail until a white spit appears in the center. It will widen and narrow with each pulse beat. Similarly hyperemic spot produced by rubbing the skin (e.g. on the forehead) may widen and narrow. The pulse is termed capillary, which is not accurate, it depends on the pulse fluctuations or arteriole blood filling.

In physiological conditions, a slight *pulsation of the jugular veins* can be observed. A normal vein pulse is called negative. In pathological conditions examination of the jugular veins demonstrates a wave of synchronous with the

carotid artery pulsation. This is a so-called positive venous pulse. This pulse is present in insufficiency of the tricuspid valve. Besides tricuspid valve insufficiency, positive venous pulse is observed in ciliary arrhythmia, atnoventricular (nodular) rhythm, when the atria and ventricles contract simultaneously.

Arterial pressure is the stress exerted by the blood on the walls of the vessels (lateral pressure) and the column of the blood from the site of the pressure to periphery (end pressure).

Constant arterial pressure is due to two factors: blood in-flow to the arterial system (pumping function of the heart) and the tone of the arterioles. The values of the arterial pressure drop from the center to the periphery, especially at the level of arterioles.

Investigation technique. Arterial pressure can be measured with a direct and indirect methods. Direct measurement is performed with artery puncture. This is mainly used in cardiosurgery. Three methods are used for indirect measurement: auscultation, palpation, oscillographic. The most practical is an auscultation method proposed by N. S. Korotkoff in 1905. It allows measuring both systolic and diastolic pressure. The measurement is done using a sphygmomanometer (mercury, Riva-Rocci apparatus, spring, electronic). The pressure is usually measured on the brachial artery. The cuff is wrapped and fastened around the bare upper arm of the patient. The cuff should be tightened to allow only one finger between it and the patient's skin. The edge of the cuff with the rubber tube should face downward. The zero level of the apparatus, the artery and the patient's heart should be at the same level. The patient's arm should rest comfortably with the palm upright and the muscles relaxed. Than the valve of the apparatus is turned off and the cuff is inflated with air until the pressure in it exceeds the 30 mm the level when pulsation of the brachial and radial artery is not felt. After that the valve is turned on and the air is allowed to escape slowly from the cuff. At the moment the pressure in the cuff becomes a little lower than systolic pressure, the first slight pulsations of the radial artery will appear (palpation method of measurement systolic blood pressure). Diastolic pressure cannot be determined using this method.

The most frequently used is Korotkoffs method, which allows determining both minimal and maximal pressure. With this method, when the pressure in the cuff

is a little lower than systolic pressure, sounds simultaneous with the heartbeat are heard with a phonendoscope over the brachial artery. When the sound appears, the values noticed correspond to systolic pressure. N. S. Korotkoff described four phases of sound phenomena, which are heard during blood pressure measurement over the vessel. Phase 1 is appearance of the sounds over the artery, that is first portions of the blood entering the vessel under the place of narrowing causing vibrations in the relaxed wall of the empty vessel. While the pressure in the cuff is dropping, more blood can pass the narrow area thus turbulent blood movement appears above the narrowing, the sound resembles murmurs (phase 2). Gradually, more blood enters the vessel increasing the vibration of its wall and the sound increases (phase 3, low sounds). When the pressure in the cuff equals diastolic pressure, the obstacle to the blood flow disappears, the vibrations decrease sharply. This moment is characterized by evident weakening and disappearing sounds (phase 4) and corresponds to diastolic pressure.

Oscillography allows to register both systolic, mean, diastolic pressure as a curve, oscillogram. The pressure fluctuations are registered on a paper band with an arterial oscillograph. Systolic pressure is pictured as low-amplitude waves. The highest oscillations correspond to the dynamic or mean pressure. The last wave corresponds to the level of diastolic pressure. Normal oscillogram: Mx - maximal pressure, Mn - minimal pressure, My - mean pressure.

Arterial pressure in healthy people is subjected to physiological changes depending on physical load, emotions, body position, meals, and other factors. It is lower in the morning, on an empty stomach, at rest, in conditions of basal metabolism. This pressure is called basal.

Arterial pressure is measured in millimeters mercury. Normal systolic pressure ranges within 100-140 mm Hg (13-18 kPa), diastolic pressure 60-90 mm Hg (8-11 kPa).

Inspection and palpation of the precardial area

The precordial area is the area of the anterior surface of the chest to which the heart and large vessels are projected. The borders of the precordial area are as follows: to the right - right medioclavicular line, top - clavicles, to the left - anterior axillary line, bottom - right and left costal arches.

During the examination the studied area should be well lightened.

At norm, the vessels of the skin on the chest are not seen or are slightly noticeable. Sometimes widened small veins of the skin forming a band of several centimeters (so-called Stokes's band) can be seen. These changes do not have any diagnostic significance and can be observed in healthy individuals. Branching curling veins in the area of the manubrium of sternum can be a sign of aortic aneurysm, mediastinal tumor, insufficiency of the right heart, adhesive pericarditis. Dilated pulsating arteries are characteristic for aorta coarctation.

Stable diffuse outpouching of the precordial area, or cardiac hump (*gibbus cardiacus*), suggests heart enlargement, which develops in childhood or early youth in rheumatics or congenital heart defect. Temporal diffuse outpouching of the precordial area, dilation and filling of the intercostal spaces occur in exudative pericarditis in persons with an elastic chest. Outpouching in the epigastric area can develop in marked accumulation of fluid in the pericardial sac (Auenbrugger's sign).

Limited pulsating outpouching of the sternal region and neighboring intercostal spaces can be seen in aneurysms of ascending aorta (on the right), its root (on the left), arch (manubrium of sternum). Palpation of this outpouching reveals systolic pulsation seen on visual examination.

Pulsation to the left of the sternum in the 2nd and 3rd intercostal spaces can develop due to hypertrophy of the right ventricle, dilation of the trunk of the pulmonary artery.

Pulsation in the cardiac area associated with left ventricle systole is called apical thrust, or apical beat (ictus apicalis). This is a conventional term as apical thrust does not correspond to the physical position of the heart apex. The pulsation involves the areas of the left ventricle located cranially and medially the anatomical heart apex. Apical thrust does not coincide with the left border of the heart and is located more internally. The mechanism of apical thrust is rather complicated.

The possibility to see and feel the apical thrust depends on many factors: age, position of the body, the position of the diaphragm, the shape of the chest, the state of the lung tissue, the type of cardiac activity. In adults the apical thrust is not seen and not palpated (thick chest, covered with a rib). The apical thrust can be felt when the

patients stands, lies on the left side, after holding the breath at the height of forced expiration, after physical load.

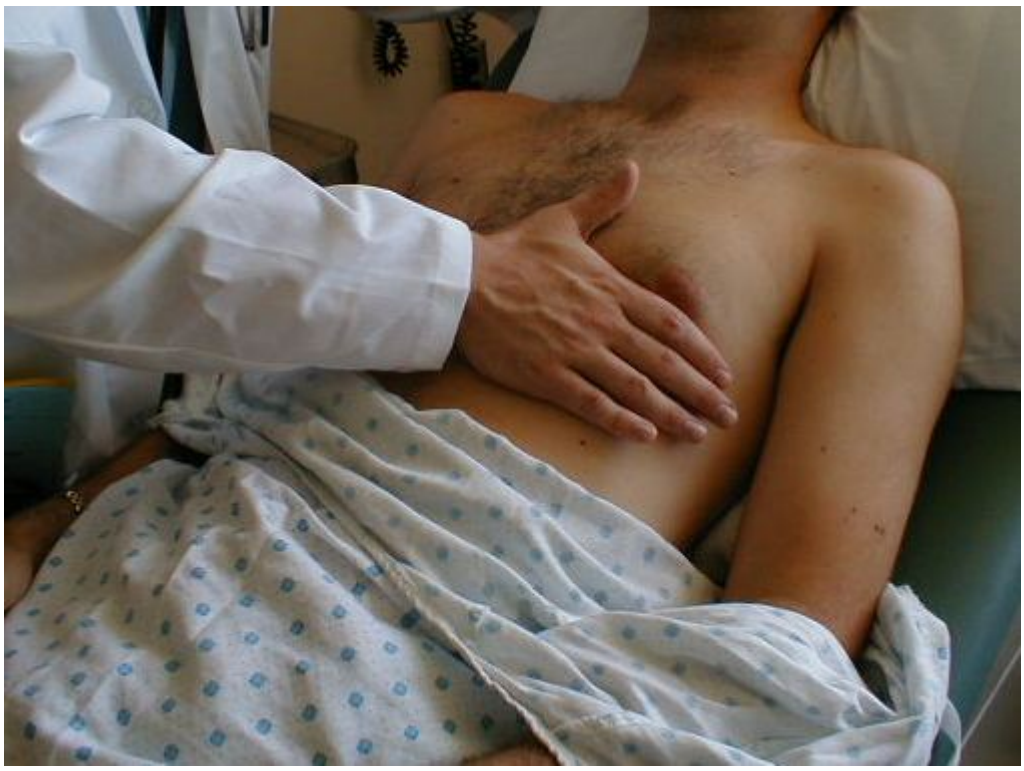

Image 11. Precordial examination: Palpation of the Precordium
Image courtesy of Charlie Goldberg, M.D., UCSD School of Medicine and VA Medical Center, San Diego, California

Technique of apical thrust (apex beat) investigation. The palmar surface of the right hand is placed on the precordial area between the presternal and anterior axillary lines and 3rd - 4th ribs.

First, location of the thrust is determined with the whole palm, then it is placed perpendicular to the ribs; the end phalanges of the fingers are used to determine the characteristics of the apical thrust: location, area, height, resistance. The lowest left point of the outpouching, which is felt by a non-lateral surface of the finger, is considered the location of the apical thrust. The place where the apical thrust is felt surely is decisive for determining the size of the thrust. This place is located a little cranially and medially the anatomical apex of the heart inner the left heart border.

In adult males and in the majority of women apical thrust can be palpated in a supine position in the **5th intercostal space 1-1.5 cm inner left medioclavicular line**. In left lateral decubitus the apical thrust moves 2 cm to the left. If, on turning to

the left side, the location the thrust remains unchanged, this may indicate adhesions between the layers of the pericardium.

In deep breathing in the apical thrust moves downwards and inwards and can disappear as it is covered by extended lungs. On deep breathing out the apical thrust moves upward to the left.

In a vertical position of the heart (long chest, asthenic constitution) the apical thrust is low: behind the 4th rib 2 cm inward the medioclavicular line.

In hypersthenics and in children the apical thrust moves to the 4th intercostal space and can be palpated near the medioclavicular line. In elderly individuals apical thrust is frequently palpated in the 6th intercostal space in the result of a low position of the diaphragm.

Apical thrust can displace to the left and upward in flatulence, pregnancy, ascites, enlarged liver and spleen. Apical thrust moves to the left behind the medioclavicular line in right hydrothorax, pneumothorax, cirrhosis, atelectasis of the left lung, pleuropericardial adhesions on the left side.

Apical thrust displaces to the right when the volume of the right lung decreases or the pressure in the left pleural cavity increases. The location of the apical thrust depends on the size of the heart.

At widening of the left ventricle, apical thrust displaces downwards to the left to the anterior axillary line and the 6th intercostal space. In hypertrophy of the left ventricle without dilation, location of the apical thrust does not change.

When the right ventricle dilates, apical thrust can move to the left. The left ventricle is displaced to the right and the heart apex is formed by the right ventricle.

The area of the apical thrust is the area of the outpouching. Normally this is about 2 cm^2. An enlarged area is called generalized thrust, a decreased one limited.

Generalized apical thrust is observed on breathing out, in tumors of the posterior mediastinum, in shrinkage of the lungs, a high position of the diaphragm, when the anterior borders of the lungs depart from the anterior surface of the heart resulting in increase of the area. Generalized apical thrust up to diffuse systolic thrill of the cardiac area to the left of the sternum (cardiac thrust) can be seen in children, asthenics, at excitation, on physical overstrain, in fever, hyperthyroidism.

When the above causes are absent, enlargement of the thrust area is the consequence of the heart enlargement (not only the left ventricle) as enlargement of any portion of the heart increases the intrathoracic pressure, the borders of the lungs collapse opening a large surface of the heart.

Limited apical thrust is observed when the heart is displaced backward from the chest in emphysema, a low position of the diaphragm, exudative pericarditis, hydro- and pneumothorax, in hypersthenics, thick chest. It is possible that apical thrust will not be determined.

Height of the apical thrust is the amplitude of movement of the outpouching, that is the distance to which this area displaces forward. If this distance increases, apical thrust is termed high, if decreases - low. High apical thrust is observed in the same conditions as the generalized thrust. The causes are the same. High apical thrust is also called "elevating".

Resistance (force) of apical thrust is resistance, which is produced by the cardiac muscle to the pressure of the palpating fingers. Resistance is determined by density, thickness of the cardiac muscle, and force with which the heart hits the chest. Resistant apical thrust is a sign of hypertrophy of the left ventricle muscle. If the left ventricle is dilated, hypertrophic apical thrust becomes resistant and high. The palpating hand feels a dense, elastic thick dome, the so-called dome-shaped apical thrust is frequently present in mitral valve incompetence. Thrust of the left ventricle can be that forceful that causes shaking of the whole cardiac region.

Hypertrophy and dilation of the right ventricle can cause systolic pulsation to the left of the sternum between the 3rd - 5th ribs and may develop in the epigastric area (***cardiac thrust***). In this case the apical thrust is not palpable, sometimes it is produced by the hypertrophic right ventricle. In marked enlargement of the both ventricles, shaking of the whole cardiac region can occur.

Epigastric pulsation can also be produced by the abdominal aorta or liver.

Pulsation of the right ventricle is seen directly under the xiphoid process, increases on breathing in, drawing in is more prominent that sticking out (***cardiac thrust***).

Pulsation of the abdominal aorta is noted in slim individuals with thin weak abdominal muscles, in enteroptosis, dilation of the aorta, aortic aneurysm. Pulsation

of the abdominal aorta is located in the middle of the line connecting the xiphoid process with the umbilicus, is directed forward. The aorta is frequently palpable, its pulsation decreases in breathing in. The signs of aneurysm are limited pulsating formation in the abdominal cavity above the umbilicus or in the left hypochondrium.

Pulsation of the liver may be true and "transmitted". True pulsation consists of increase and reduction of the liver volume in case of tricuspid valve incompetence due to systolic regurgitation of blood from the right ventricle to the right atrium, which causes increased pressure in the vena cava and hepatic veins during systole and enlargement of the liver in all directions. "Transmitted" pulsation is caused by the beat of the right ventricle or abdominal aorta, the liver moves forward.

Other pulsations of the cardiac region. Aneurysm of the heart can cause pulsation in a limited area cranially the heart apex in the 4th intercostal space.

In marked mitral insufficiency with dilation of the left atrium pulsation along the medioclavicular line between the 3rd and 4th ribs may be observed (the left atrium displaces to the right half of the chest).

In tricuspid valve insufficiency along the whole lower portion of the chest on the right to the 4th rib there is forceful systolic pulsation transmitted from the liver pulsating synchronously with the systole of the right ventricle.

Aortic pulsation can occur in the 2nd intercostal space to the right at the border of the sternum in shrinkage of the border of the right lung, in aneurysm of the ascending aorta or its arch, in insufficiency of the aortic valve. In aneurysm pulsation is expansive, is felt on the bottom of the fossa, is directed upward (the patient lifts the shoulders and bends the head forward).

Pulsation of the pulmonary artery is felt in the 2nd intercostal space to the left of the sternum in shrinkage of the border of the left lung and dilation of the pulmonary artery (mitral stenosis). The beat is felt during diastole.

Atrial pulsation is sometimes seen and felt in the area of the heart base. This pulsation is associated with diastolic filling of the atria coinciding with the systole of ventricles and is observed at shrinkage of the lungs, in considerable dilation of the atria in insufficiency of the mitral valve or tricuspid valve (pulsation is caused by blood regurgitation).

Systolic drawing in of the cardiac region can be revealed in the area of the heart apex as well as in the whole cardiac region. Frequently this drawing in is observed in asthenics in the area of the apical thrust, if the thrust is covered by the rib and reduction of the heart volume during systole may be associated with sucking effect near the areas where the heart adjoins the cardiac region. In these cases it is necessary to investigate the apical thrust in left and right decubitus. If the drawing in remains in place, this is the sign of pathology and can be attributed to marked enlargement of the right ventricle, sudden reduction of the heart volume (tricuspid valve insufficiency), adhesive pericarditis.

The heart immured in dense cicatrix adhesions cannot turn forward during systole and contracting it will involve the chest (negative apical thrust).

Palpation of the precordial area can elicit a special phenomenon, shaking, vibration of the chest resembling the feeling which can be felt when you put your hand on the chest of a cat ("cat's purring"). This phenomenon occurs when the blood passes a narrowed opening forming turbulent currents causing vibration of the heart muscle transmitted to the cardiac region.

"Cat's purring" is better felt with a palm put to the cardiac region.

In mitral stenosis, "cat's purring" is reveled in the area of the heart apex, sometimes in left decubitus, during diastole before beginning of systole (presystolic) or during the whole diastole. Sometimes such presystolic shaking occurs in aortic valve incompetence (the flow of blood regurgitation lifts the anterior cusp of the mitral valve narrowing the mitral opening and causes oscillations). "Cat's purring" can develop in considerable enlargement of the left ventricle (relative narrowing of the left venous orifice). In the right 2nd intercostal space and in the area of the manubrium of sternum systolic thrill is formed in aortic stenosis, it is revealed during forced expiration and after physical load. In defects of the interventricular septum, systolic "cat's purring" is felt in the middle of the sternum near its left edge. In the 2nd and 3rd intercostal spaces to the left of the sternum, systolic thrill is a sign of pulmonary artery stenosis, continuous systolodiastolic thrill in this area can be felt in patent arterial duct.

The heart percussion allows determining its size, shape as well as the size of the vascular band. The heart is spherical and only a small area of its anterior surface adjoins the chest.

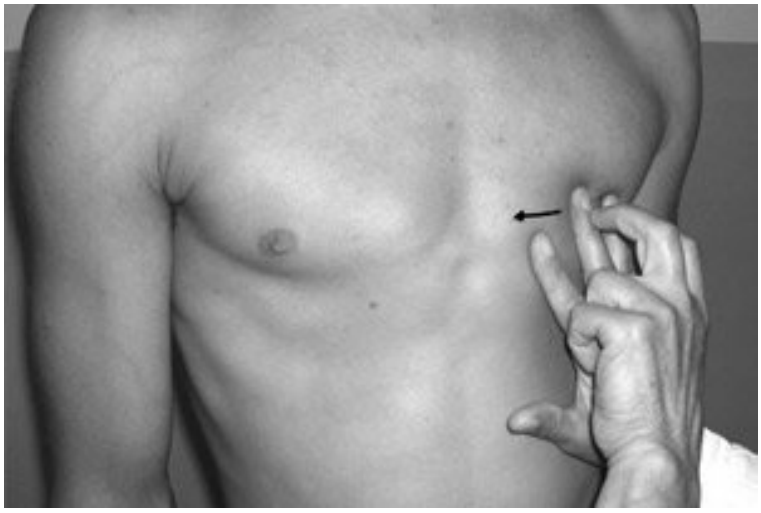

Image 12. Heart percussion

The image was downloaded from website https://m.iliveok.com/

The normal outlines of the heart are presented by the superior vena cava in the 2nd and 3rd intercostal spaces, the right atrium in the 4th intercostal space. Its left border is the left potion of the aortic arch and further the trunk of the pulmonary artery in the 2nd intercostal space. At the level of the 3rd rib is limited by the auricle of the left atrium, its lower border is presented by a narrow band of the left ventricle at 4-5th ribs.

The heart is a dense air-free organ. Percussion over the heart produces a dull sound. A smaller portion of the anterior surface of the heart adjoins the chest, its larger portion is covered with the borders of the lungs. The area of the heart not covered by the lungs, which produces the dull sound on percussion, is termed absolute heart dullness. It corresponds to the position of the right ventricle, which forms the anterior border of the heart. The portion of the heart covered with the lungs produces a deadened sound termed relative dullness. This is the projection of the anterior heart surface on the chest and corresponds to the true heart borders. To diagnose cardiac diseases topographic percussion is used, its purpose to determine the borders of relative and absolute dullness of the heart. Percussion of the heart can be done both in a sitting and upright position of the patient. It should be remembered

that the size of the heart dullness in an upright position is smaller than in a lying position. This is associated with the heart mobility and shifting of the diaphragm with the change in the position.

Before percussion it is necessary to determine the level of the diaphragm as the changes in the position of the diaphragm influence the location of the heart in the chest and the borders of the heart dullness. With this purpose, the location of the lower border of the right lung (normally in the 6th intercostal space) is determined along the right medioclavicular line. Percussion is started from the 1st intercostal space along the intercostal spaces from a clear to dull sound.

The position of the diaphragm corresponds to the lower border of the lungs, that in the 6th intercostal space. After it has been determined, the right, upper and, at last, left borders of relative cardiac dullness should be determined. The lower border of the heart cannot be determined as it borders with the liver producing similar dull sound. To determine **the borders of relative dullness**, percussion with a medium force is used. After determining the position of the diaphragm the plessimeter finger is shifted one intercostal space upper (or two ribs, in the 4th intercostal space) placing it parallel to the right border of the heart. Making taps using medium force, the plessimeter finger is moved along the intercostal space to the heart until deadened sound appears. The right border of the heart is noted along the external border of the piessimeter pointing to the clear sound.

The right border is formed by the right atrium and is normally in the 4th intercostal space 0.5 cm outer (to the right) the right sternal line or along the right edge of the sternum. To determine the upper border of the heart the plessimeter finger is placed in the first intercostal space 1 cm outer the left sternal line (not on the left medioclavicular line) and is shifted downwards making taps using medium force until a deadened sound appears. The upper border of the heart is noted along the upper (external) border of the plessimeter finger (closer to clear percussion sound). *The upper border* of relative heart dullness is formed by the auricle of the left atrium and the trunk of the pulmonary artery and is normally located in the 3rd rib. The left border of the relative dullness is determined in the same intercostal space, where apical thrust is located, usually in the 5th intercostal space.

Percussion is performed in the 5th intercostal space from the left anterior axillary line towards the sternum. The plessimeter finger is placed parallel the supposed border, the percussion is accomplished from a clear until a deadened sound. *The left border* is marked along the outer border of the plessimeter finger facing the clear sound. Normally, it is formed by the left ventricle and is in the 5th intercostal space 1-2 cm inner the left medioclavicular line and coincides with the apical thrust.

Table 2

Borders of relative dullness in healthy people

The right border	0.5 cm outer (to the right) the right sternal line or along the right edge of the sternum
The upper border	3rd rib
The left border	5th intercostal space 1-1.5 cm inner the left medioclavicular line

The 4th and 5th intercostal spaces, in which the borders of relative heart dullness are determined, are the uttermost points of the outer outline of the heart. Changes in the heart borders in these spaces are also accompanied by the changes in other spaces. That is why percussion is usually performed in the 4th intercostal space for the right border, in the 5th intercostal space for the left border.

Then **the borders of absolute heart dullness** (the area of the heart which is not covered with the lungs and adjoins the breastbone) are determined. The whole area of absolute dullness is formed by the anterior surface of the right ventricle.

Light percussion is used for this purpose. The percussion of this area produces a dull sound. There are two methods, which allow determining the borders of absolute heart dullness. The first one uses light percussion done from the relative dullness until a dull sound appears. The border of absolute dullness is marked along the outer edge of the plessimeter finger facing the deadened sound. To determine the right border of absolute heart dullness after determining the border of relative dullness in the 4th intercostal space, the plessimeter finger is placed parallel the sternum and then is moved inward until dull sound appears. The border of the absolute dullness is marked along the outer edge of the finger facing the border of relative dullness. Normally *the right border* of absolute heart dullness is noted along

the left edge of the breastbone. Actually the right border of absolute heart dullness is located in the middle of the sternum along the inner border of the right lung. Accurate determining of this border is hindered by the breastbone, which also vibrates on percussion. To determine the upper border of absolute heart dullness, the plessimeter finger is placed in the 3rd intercostal space parallel the ribs, the percussion is performed downward the intercostal spaces until a dull sound appears. *The upper border* is marked along the outer edge of the plessimeter finger pointing upward. This is normally located in the upper part of 4^{th} rib. To determine the left border of absolute heart dullness percussion is done along the 5th intercostal space from the border of relative heart dullness until a dull sound appears. *The left borde*r is marked along the outer edge of the plessimeter finger facing the left border of relative heart dullness. This is normally 1-2 cm inner the left border of relative dullness.

Table 3

Borders of absolute dullness in healthy people

The right border	left edge of the sternum
The upper border	upper part of 4^{th} rib
The left border	5th intercostal space 1-1.5 cm inner the left medioclavicular line or 1-2 cm inner

Auscultation of the heart

Auscultation of the heart is an objective method of examination, which consists in listening to and evaluation of acoustic phenomena developing when the heart works.

Rules of heart auscultation. The chest should be bare, the areas with thick hair on the chest should be moistened with water to reduce the friction of the hair. The room should be warm and silent. The funnel of the stethoscope is densely pressed to the chest, but is should not compress the tissues. The patient is asked not to breathe deeply. Auscultation is performed at quiet respiration, when the patients does not breathe, at the height of the expiration and inspiration, in a lying and upright position, on the left side, and after dosed physical load.

Auscultation of the heart is performed in definite regions of the precordial area (**main auscultation points**), their choice is determined by better conduction of the acoustic phenomena related to the functioning of various heart structures: muscular strain, stroke of the blood at the cusps of the valves, stretching of the walls of the large vessels, as well as conversion of laminate blood flow to turbulent.

Point 1 - the area of apical thrust, in case it is absent, left border of relative cardiac dullness should be listened to. The acoustic phenomena which occur at the beginning of systole at tension of the muscular structures of the left ventricle, the stroke of the blood in the phase of isometric strain with closed cusps of the mitral valve and their vibration (first sound) are better heard in this point.

Point 2 - lower edge of the sternum in the place of xiphoid process origin. The acoustic phenomena evoked at the beginning of systole at strained muscles of the right ventricle, stroke of the blood moving in the direction of the lowest pressure at the closed cusps of the tricuspid valve and their vibrations.

Point 3 - 2nd intercostal space to the right near the edge of the breastbone. The acoustic phenomena which occur at the beginning of diastole as a result of the stroke of the blood moving in the direction of the lowest pressure with closed cusps of the aortic valve are heard here (second sound).

Point 4 - 2nd intercostal space on the left edge of the breastbone. The phenomena which are evoked by the stroke of the blood at the closed cusps of the pulmonary artery and their vibrations are hard here (second sound).

Point 5 (Botkin-Erb) is listened to in two positions of the stethoscope: in the middle of the breastbone at the level of the 3rd costal cartilages and in the 3rd intercostal space at the left edge of the breastbone. These positions arc associated with the projection of the aortic valve on the precordial area. It is the place where acoustic phenomena developing at the beginning of diastole, especially when the cusps of the aortic valves are not closed (diastolic sound of blood regurgitation), are well heard.

In healthy adults the heart melody consists of two sounds (first and second), this is divided by two pauses (systolic and diastolic).

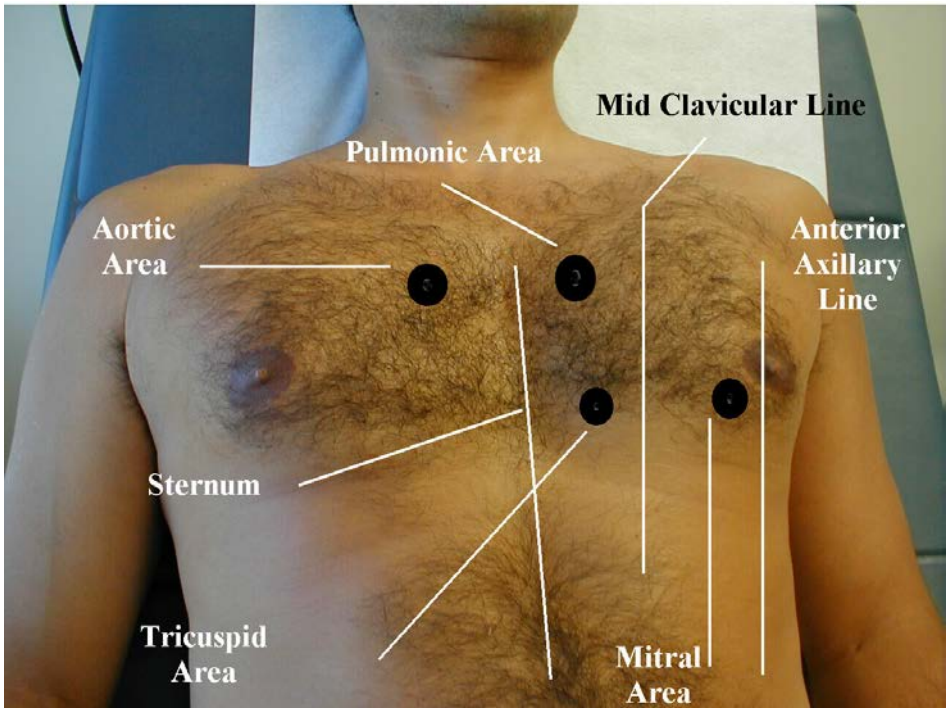

Image 13. Precordial examination: Auscultation of the Heart

The image was downloaded from website https://www.wikidoc.org/index.php/Precordial_examination

Heart sounds are short acoustic phenomena resulting from tissue vibration, which occur at vibrations of strained muscles of the ventricles and valve cusps. The first sound (systolic) is heard at the beginning of systole, the second heart sound (diastolic) is heard at the beginning of diastole.

In children and teen-agers accessory sounds (third and fourth) are sometimes heard.

Mechanism of sound formation. **The first sound** consists of three components: muscular, valvular, vascular. The *muscular component* is formed during asynchronous contraction (contraction of papillary muscles and the neighboring muscles of the ventricles) and at the beginning of isometric strain as a result of vibration of these muscular structures. Some authors believe that vibrations of atrial muscles at their contraction also take part in formation of this component and distinguish the atrial component of the first sound. But distinguishing the atrial component of the first sound seems groundless, as atrial systole is related to the end of ventricular diastole (so-called pre-systole) and ends by the beginning of asynchronous contraction: the first sound is a systolic phenomenon. The muscular

component of the first sound is presented by low-frequency and law-amplitude vibrations.

Valvular component of the first sound is formed at stroke of the blood at closed cusps of the atrioventricular valves in the phase of isometric strain. The blood moves in the direction of the lowest pressure of the ventricular walls to the atria and strikes the cusps of the atrioventricular valves, making them to do vibration movements.

The vibrations, which compose the ventricular component of the first sound, have the highest frequency and amplitude when compared with the muscular component.

The vascular component of the first sound is associated with vibrations of the walls of the aorta and pulmonary artery at the beginning of blood ejection from the ventricles. This component consists of low-frequency and low-amplitude vibrations.

The second sound also consists of two components. The first component, *cusp*, occurs when the blood strikes the closed cusps of the aorta and pulmonary artery while moving in the direction of the lowest pressure (to the ventricles). This component consists of high-frequency and high-amplitude vibrations. The second component, *vascular*, is formed when the walls of the vessels vibrate during the period of reverse movement from the vascular valves. The vascular component is presented by low-frequency and low-amplitude vibrations.

Changes of cardiac melody. The changes of cardiac melody can be associated with the changes in the sound loudness, timber, and number.

The loudness of the first sound is mainly determined by the cusp component and depends on the following: the position of the cusps of the atrioventricular valves immediately before the onset of ventricular systole, their anatomical structure, kinetic energy of blood movement during isometric tension.

Weakening of the first sound is observed when isometric strain is absent when the cusps of the atrioventricular valves do not close due to shrinkage, deformity, widening of the atrioventricular opening in dilation of the ventricles), the blood at the beginning of systole passes through the cleft between the cusps to the atria, which decreases the force of beat, determining the cusp component of the first sound. The degree of weakening of the first heart sound reflects the degree of the defect.

In mitral valve incompetence, both absolute (heart defect) and relative (dilation of the left ventricle in prolonged arterial hypertension, dilation cardiomyopathy), the first sound weakens over the apex.

In organic tricuspid valve incompetence (heart defect), relative tricuspid valve incompetence at dilation of the right ventricle (mitral stenosis, cor pulmonale, congenital defects), the first heart sound is weak in point 4.

Increase of the first heart sound has the highest diagnostical importance in narrowing of the left and right atrioventricular orifice and is explained by insufficient filling with blood of the left ventricle during diastole.

In mitral and tricuspid stenosis, the fused cusps of the atrioventricular valves form a kind of membrane, which sags deeply during diastole to the ventricular cavity due to high pressure in the atria and low in the ventricles. In the period of isomeric tension, the blood moves at a high rate in the direction of the atria as its amount in the ventricular cavities is lower than in healthy subjects, kinetic energy of the stroke increases and the first sound becomes more forceful. The closed valves move along longer distance due to insufficiently filled ventricles, sag in a dome-like manner in the cavity, and vibrate with high frequency. The first sound acquires a special timber, resembling a flopping sail, and is called Hopping.

A flopping first sound suggests that the valve has preserved some elasticity. In calcification of the valve, insufficient mobility, rigidity of the cusps, arrhythmia this change of the timber may disappear.

Increase of the first sound is heard in other conditions, accompanied by decreased filling of the ventricles with blood (extrasystole, hyperthyrosis, fever).

Loudness of the second sound depends mainly on the valve component and is determined by the kinetic energy of the blood moving in the direction of the valves at the beginning of diastole under the influence of difference in pressure in the large vessels and the respective valves. Besides, elasticity of the vascular wall, the valves of the aorta, and the pulmonary artery play a role: the less elastic are the vessels receiving the blood, the lower is the degree of extension, and the higher is the pressure in the respective vessel, the louder is the second sound. The second heart sound is heard in points 2, 3, 5.

Accent of the second sound over the pulmonary artery is the sign of increased blood pressure in the pulmonary system resulting from mitral heart defects, especially mitral stenosis, insufficiency of contractile function of the left ventricle, chronic diseases of the lungs (emphysema, chronic obstructive bronchitis), in patent arterial duct, idiopathic sclerosis of the pulmonary artery (Ayerza's disease).

If the second sound is increased in points 2 and 3, it is necessary to consider over which point the second sound is accentuated. It is recommended to move the stethoscope outwards to the left and to the right at equal distance. If the second sound is still heard on one side and disappeared on the other, the accentuated is the sound over the valve where it is heard.

Weakening of the second sound is usually caused by stenosis of the aorta orifice (point 2), stenosis of the pulmonary artery orifice (point 3), insufficiency of the respective semilunar valves. In narrowing of the vascular openings less blood is pumped to the vessels during systole, which is accompanied by reduction of the force of stroke on the closed cusps of the valves at the beginning of diastole and results in weakening of the second sound.

Intensification in the both sounds is frequently associated with extracardiac factors (thin flat chest, shrunken lungs, infiltrations of the lung borders, high position of the diaphragm) as well as shortening of diastole and decreased filling of the ventricles with blood at increased heart rhythm (tachycardia at physical exercise, emotions, fever, anemia, hyperthyroidism.

Weakening of the both sounds can be associated with extracardiac causes: thick outpouching chest, pulmonary emphysema, left hydrothorax, low position of the diaphragm. As a rule, the both sounds weaken and are poorly heard (surditas cordis — deafness of the heart) in affection of the cardiac muscle (myocarditis, myocardial infarction, cardiosclerosis, cardiomyopathy), in acute and chronic heart failure, sharp drop of the blood pressure (collapse), hydropericarditis, fibrinous pericarditis.

Changes in the number of heart sounds. This phenomenon can occur at either splitting of the heart sounds or at appearing accessory sounds (extrasounds).

Split and dual heart sounds are produced by nonsimultaneous contraction and relaxation of the ventricles and can be ether physiological or pathological.

At splitting, two consequent sounds are heard without a pause; dual sounds are divided by a pause.

Splitting of the first sound can be heard in all healthy individuals medially the heart apex in the 5th intercostal space to the left of the sternum, near the xiphoid process, in the 4th intercostal space near the parasternal line. This phenomenon is frequently heard by the end of breathing in and at the beginning of breathing out, in standing position, at physical exercise. Physiological splitting of the first sound is associated with the fact that the mitral valve closes earlier than the tricuspid valve.

Dual first sound is heard over a large area with its maximum between the heart apex and the breastbone. It does not depend on external factors and is associated with hypertrophy of the left ventricle, blockade of the pedicles of the bundle of His, chiefly the right one, ventricular extrasystole, antesystole (WPW syndrome), in tachysystolic form of atrial flatter.

Split and dual second sound is observed in healthy individuals and is heard in the second and third intercostal spaces to the left of the sternum in a supine position at deep breathing in. This phenomenon disappears with holding the breath on expiration. Delay in the closure of the valves of the pulmonary artery at the height of the inspiration, which is the cause of split and dual second sound, is associated with greater in-flow of the blood to the right ventricle during inspiration due to increase of negative intrathoracic pressure in the dilating chest. The blood is held in the dilated vessels of the lungs, the amount of the blood entering the left atrium and later the left ventricle decreases, the systole ends earlier than during breathing out.

Quail's rhythm. This is a three-part rhythm resembling dual second sound, is a pathognomonic sign of mitral stenosis.

Quail's rhythm results from appearance in the early diastole an accessory sound associated with anatomical changes of the mitral valve. This short abrupt, snapping or knocking sound follows the second sound like a loud echo, frequently more intensive that the second sound and is called opening snap. In healthy individuals the mitral valve opens silently, its cusps are freely pushed aside to the walls of the ventricle with the blood, they do not strain or vibrate. Fused dense cusps of the mitral valve in heart defects cannot open freely, before opening the valve sharply protrudes into the cavity of the left ventricle with a specific snapping sound. This accessory sound is

better heard in the 4th and 5th intercostal spaces between the left edge of the sternum and the heart apex, sometimes at the left edge of the sternum in these spaces, in the axillar and even under the angle of the left scapula and above the projection of the mitral valve, and increases on breathing out. The degree of mitral stenosis is in reverse correlation with the interval between the snap and the second sound.

Gallop rhythm is a three-part rhythm, in which the sounds are separated by approximately equal pauses and repeat regularly resembling the sound of a galloping horse. This rhythm is heard with the heart rate of 100 beats per minute, when the rate is <70 or >120 beats per minute this rhythm disappears.

Gallop rhythm results from formation of accessory diastolic sounds: at the end of diastole - presystolic fourth sound, in the beginning - protodiastolic third sound. It is frequently difficult to differentiate presystolic and protodiastolic gallop rhythm, especially in tachycardia or elongation of the interval between the contraction of the ventricles and atria, when the contraction of the atria coincides with the protodiastolic period of quick filling in of the ventricles. This rhythm is called summary.

Protodiastolic gallop is heard over the heart apex or in the region between the apex and the sternum, better in a lying than in a sitting position, more distinctly on the left side and during breathing out. It disappears on pressing the precordial area with the stethoscope. The accessory third sound can mimic dual second sound.

It is frequently difficult to distinguish gallop rhythm. It should be remembered that gallop rhythm occurs in persons with myocardium affection and signs of cardiac failure: dyspnea, edema, attacks of cardiac asthma, heart enlargement, tachycardia, arterial hypertension.

Physiological third and fourth sounds develop mainly in children and young persons with normal heart borders, with unchanged first and second sounds without the signs of cardiac failure. Physiological split and dual sounds are not constant phenomena, they depend on the position of the patient and phases of respiration, better heard in the upright position and are not accompanied by tactile sensations.

Dual second sound in mitral stenosis is heard over the pulmonary artery, the second sound is increased.

The sound of the mitral valve opening is characterized by a clear timber, sonority, abruptness, it is closer to the second sound than a pathological sound. The first sound is increased, squelching.

But it should be noted that the most reliable way of distinguishing gallop rhythm is constant training of auscultation skills, attentive thorough listening to the patient.

Neither description of this phenomenon can characterize the whole complex of auditory, tactile, visual sensations occurring in gallop rhythm.

Any gallop rhythm suggests cardiac failure.

Left-ventricle gallop rhythm is observed in coronary artery disease, frequently in myocardial infarction, stable arterial hypertension, aortic valve incompetence, cardiomyopathy, acute myocarditis, and severe anemia.

Right-ventricle gallop rhythm develops in chronic and acute cor pulmonale, congenital heart defects. Other signs of right-ventricle insufficiency are also present: enlarged liver, edema, relative incompetence of the tricuspid valve.

Heart murmurs are prolonged acoustic phenomena produced by the working heart. They are divided into two groups: extracardiac and intracardiac.

Extracardiac murmurs are pericardial friction rub and cardiopulmonary murmurs. *Intracardiac murmurs* develop inside the heart and large vessels and can be explained by the following mechanisms:

1. Laminar blood flow turns into turbulent.
2. Formation of turbulent movements in the blood.
3. Forceful stream of the blood produced by pressure.

Functional murmurs are observed in 20-70 % of children and young individuals (under 20). Functional murmurs are not constant, they are changeable, depend on the position of the body and the phase of respiration. They may develop on physical overstrain and disappear at rest, they increase on deep breathing, most distinctly by the end expiration. This is almost always a systolic murmur over the pulmonary artery and the heart apex, as a rule it is low, short, blowing high-pitched.

An organic murmur develops in anatomical disturbances of the valve structure. If, due to anatomical disorders (fusion of the cusps), the valve cannot open completely, venous or arterial opening of the heart narrows. If the cusps of the valve do not close due to their fibrous thickening, shortening, destruction, or

perforation, the blood goes back to the heart chamber from which it has been ejected, this defect is called incompetence (insufficientia valvuarum), the backward blood flow is called regurgitation (regurgitatio).

If incomplete closure of the unchanged valves occurs due to considerable enlargement of the heart or affection of the papillary muscles, this causes relative valvular incompetence. One of the openings can be narrower than usually when compared with the enlarged cavity (relative narrowing of the opening). The murmurs developing in this case are called muscular or organ-functional.

Valvular murmurs heard in the period of the orifice opening are the sign of the orifice narrowing. The murmurs, developing when the orifice must be closed, suggest incompetence of the respective valve.

The murmur occurring during systole (systolic) is the sign of atrioventricular valve incompetence or narrowing of the arterial orifice. The murmur developing during diastole (diastolic) is the sign of the venous orifice narrowing or incompetence of the semilunar valves.

The following properties of the murmur have diagnostic significance:

- association of the murmur with the phase of the cardiac cycle (systolic, diastolic);

- the area where it is heard best (heart apex, 2nd intercostal space to the right and left of the sternum, basis of the xiphoid process);

- direction of the murmur radiation (axillary area, cervical vessels, subscapular area);

- loudness, duration of the murmur;

- the timber of the murmur (blowing, sawing, scratching, musical);

- relation of the murmur and the sound after which it is heard (merges with the sound, separated from it);

- if the murmur increases or decreases during the pause; - influence of the position, physical exercise, phases of respiration.

The area, where the murmur is best heard, allows to decide on the location of the lesion: the murmur over the apex is the signs of mitral valve affection, in the 2nd intercostal space On the right that of aortic valves, in the 2nd interspace on the left that of valves of the pulmonary artery, in the base of the xiphoid process - tricuspid

valve. The murmur radiates along the blood flow causing it, the range of propagation is related to the loudness of the murmur. A systolic murmur in narrow aortic opening has the largest area of radiation: carotid arteries, heart apex, epigastric, interscapular regions. A diastolic murmur in the aortic valve incompetence radiates to the heart apex and the left edge of the sternum. A systolic murmur over the heart apex in mitral valve incompetence radiates to the left axillar and under the angle of the left scapular.

As to the pathogenesis, the murmurs are divided into ejection, regurgitation, filling murmurs.

Ejection murmurs develop as a result of the blood flow acceleration when passing the narrowed orifice at contraction of the heart muscle.

A systolic ejection murmur is characteristic for stenosis of the aorta, pulmonary artery, in increased stroke volume when the valves are intact. This murmur begins after a pause separating the murmur from the first sound, its duration is directly proportional to the degree of stenosis. The murmur gains its maximum in mesosystole, then decreases and dies before the appearance of the second sound separated from it by a pause. It is often accompanied by a "cat's purring". A systolic ejection murmur associated with increased circulation begins immediately after the first sound, it is short and reaches its maximum during protosystole.

A presystolic ejection murmur in mitral and tricuspid stenosis appears during telediastole resulting form atrium contraction. This special rumbling murmur increases by the moment the first sound merges with it and is accompanied by a cat's purring.

A systolic regurgitation murmur in mitral or tricuspid valve insufficiency is long, frequently pansystolic, merges with the first sound and decreases by the second sound appearance.

A diastolic regurgitation murmur in incompetence of the semilunar valves of the aorta and pulmonary artery is joined with the second sound, its loudness increases before telediastole, it is, as a rule, blowing and low.

A diastolic filling murmur in atrioventicular stenosis is separated with a pause from the second sound, it follows the snap produced by opening mitral and tricuspid valves, it is long, decreasing.

A systolic murmur at the heart apex is frequently observed causing the problems for diagnosis.

Organic incompetence of the mitral valve is a rare cause of this murmur. In typical cases it merges with a weakened first sound, is of moderate loudness, is long (not less than 2/3 of systole), can be blowing, noisy, whistling, sometimes rough and musical. It is better heard directly at the heart apex or a little cranially, radiates to the left axillar and to the angle of the left scapula, is frequently heard in the 3rd-4th interspaces at the left edge of the sternum, it increases in left decubitus and in a lying position. A mesosystolic decreasing murmur frequently appearing after systolic click is characteristic for mitral prolapse.

A presystolic murmur suggests sufficient contractile function of the atria, it disappears on dilation and development of ciliary arrhythmia.

A diastolic murmur in mitral stenosis is heard over a limited area, frequently in the area of the heart apex, sometimes medially or laterally, to the axillar and midaxillary line. It can frequently be heard at the lower angle of the left scapula. In a supine position and in left decubitus after physical load this murmur increases and sometimes disappears after several cardiac cycles following the physical exercise.

A diastolic filling murmur is characterized by a pause between the second sound and the murmur, which occurs as a result of delay in opening of the changed mitral valve.

Laboratory and instrumental tests in cardiology and rheumatology

The ECG leads

ECG leads are formed by placing electrodes at specific places on the body and amplifying and recording the electrical activity that occurs along this pathway.

ECG leads are formed by placing electrodes at specific places on the body and amplifying and recording the electrical activity that occurs along this pathway. A vector is a force of a known magnitude and direction. The recordings may be said to display certain vectors or electrical forces traveling in the direction between the leads.

Ten electrodes are used for a 12-lead ECG. They are labeled and placed on the patient's body as follows:

Table 4

The ECG leads. Electrode label and Electrode placement

Electrode label	Electrode placement
Red	On the **right arm** (RA), avoiding bony prominences
Yellow	In the same location that RA was placed, but on the **left arm** this time
Green	On the **left leg** (LL), avoiding bony prominences
Black	In the same location that LL was placed, but on the **right leg** this time

The **precordial leads** provide points of reference across the chest wall as illustrated. They differentiate right-sided and left-heart events.

The electrode is placed successively at 6 positions: V1 - right sternal border, the 4th intercostal space; V2 - left sternal border, the 4th interspace; V3 - left parasternal line, between the 4th and 5th interspace; V4 - left midclavicular line, the 5th interspace; V5 - left anterior axillary line, the 5th interspace; V6 - left midaxillary line, the 5th interspace. Unipolar chest leads proposed by Wilson are more popular now. The chest electrode, which is attached to the positive pole of the electrocardiograph is only active; the electrodes leading from the limbs are united and connected to the negative terminal of the apparatus. With this connection, the total potential difference recorded from the limbs is practically zero.

To record a routine ECG, 12 leads are used: on the limbs: I, II, III, AVR, AVL, AVF and on the chest: V1, V2, V3, V4, V5, V6.

The significance of each wave and interval of the ECG

The **P wave** is the first upward deflection and represents the atrial depolarization. Enlargement of the P wave might occur in such conditions as mitral stenosis or chronic obstructive pulmonary disease, which would cause atrial hypertrophy. The P wave is usually considered enlarged if it is more than 3 mm high and 0.10 second wide.

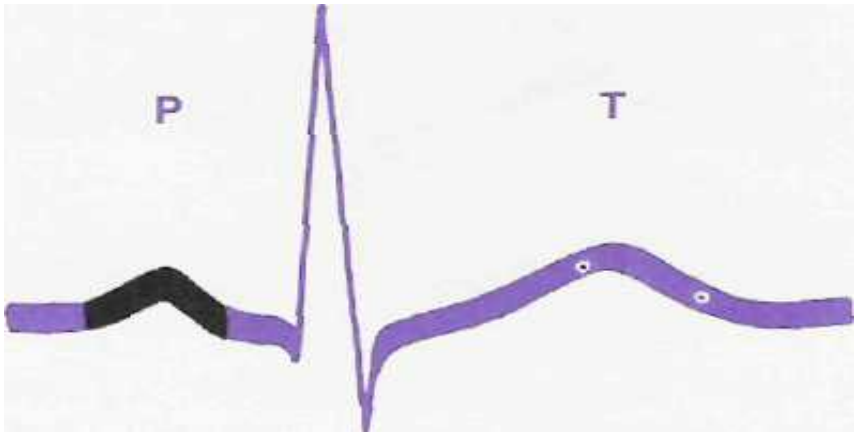

Image 14. The P wave
The image was downloaded from website https://litfl.com/pr-segment-ecg-library

The **PR (PQ) interval** extends from the beginning of the P wave to the onset of the QRS. It represents conduction of the impulse through the atria and the AV node. The PR interval is abnormally lengthened when the impulse is forced to travel at a slower rate, which can occur in arteriosclerosis, inflammation, insufficient oxygen supply, or scarring from rheumatic heart disease. It can also occur as an effect of depressant drugs or digitalis. The normal PR interval is 0.12-0.20 second wide.

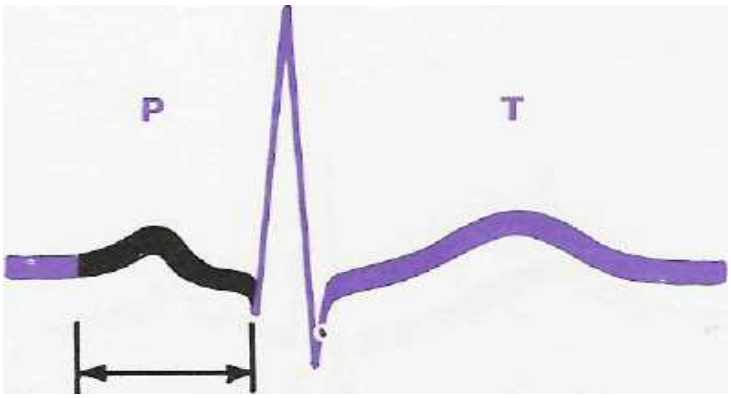

Image 15. The PR (PQ) interval
The image was downloaded from website https://litfl.com/pr-segment-ecg-library

PR interval

The **QRS complex** consists of three deflections: Q wave, the downstroke before the R; R wave, the first upward deflection; and S wave, the downstroke following the R wave. Not every QRS complex shows a discrete

Q, R, and S wave, but the configuration is still referred to as the QRS complex to denote a ventricular impulse. An enlarged Q wave (Its amplitude is small and does not normally exceed one-fourth amplitude of the R wave; the length of the Q wave does not exceed 0.03 second. The Q wave may be absent on an ECG.) may indicate a myocardial infarction. A vertically enlarged R wave usually indicates enlarged ventricles. The normal duration of the QRS is 0.06 – 0.10 second wide.

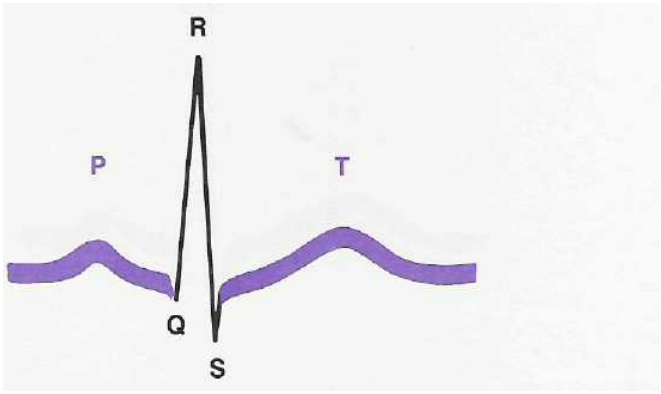

Image 16. The QRS complex

The image was downloaded from website https://litfl.com/pr-segment-ecg-library

The **ST segment** begins at the end of the S wave (the point where the line turns right) and ends at the beginning of the T wave. It is elevated in an acute myocardial infarction or muscle injury. It is depressed when the heart muscle isn't getting a sufficient supply of oxygen - for example, during an episode of angina or coronary insufficiency. It may sag as an effect of digitalis. ST changes are usually transient.

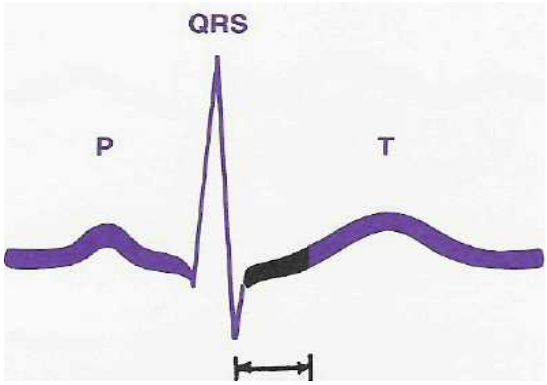

Image 17. The ST segment

The image was downloaded from website https://litfl.com/pr-segment-ecg-library

The **T wave** represents electrical recovery of the ventricular contraction. (The electrons are in the process of moving back into the normal resting position.) The T wave is flat or inverted in response to ischemia, position change, food intake, or certain drugs. It may be elevated when the serum potassium is elevated. The normal T wave is no more than 10 mm high in the precordial (chest) leads and 5 mm high in the remaining leads.

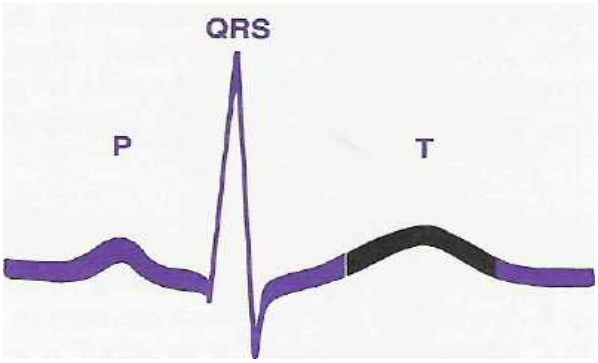

Image 18. The T wave

The image was downloaded from website https://litfl.com/pr-segment-ecg-library

The U wave is a small upward deflection following the T wave. It is seldom present, but may occur when the serum potassium level is low.

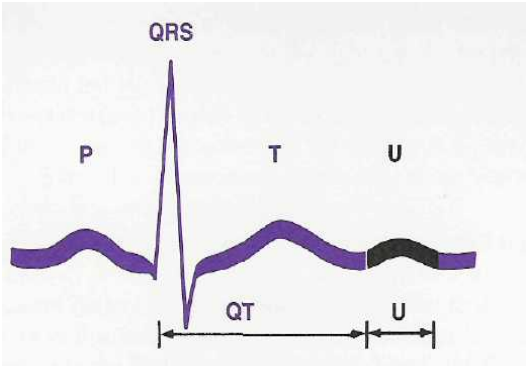

Image 19. The QT interval and the U wave

The image was downloaded from website https://litfl.com/pr-segment-ecg-library

The **QT interval** represents the time from the beginning of the Q wave (downward deflection following the P wave) through the QRS and the T wave. It includes the time until the T wave is completed (goes back to the baseline). The time of this interval should be less than one-half of the R-R interval (from the peak of one R

wave to the peak of the next R wave). If the QT time is prolonged, it presents an extended opportunity for stray irritable impulses to excite the heart tissue and trigger dangerous ventricular rhythms. After the T wave is completed the tissue is repolarized and at rest, ready to respond normally. Impulses that arrive during the T wave find the ventricular tissue incompletely recovered and vulnerable to an erratic response. The Q-T interval in women is longer than in men (at the same leart rate). For example, at the rate of 60-80 beats per minute, the length if the Q-T interval in men is 0.32-0.37 second and in women: 0.35-0.40 second.

Two-dimensional (2D) echocardiography

• Cardiovascular imaging plays an essential role in the practice of cardiology

• Two-dimensional (2D) echocardiography is able to visualize the heart directly in real time using ultrasound, providing instantaneous assessment of the myocardium, cardiac chambers, valves, pericardium, and great vesselsmanagement of cardiac arrhythmias.

Doppler echocardiography

• Doppler echocardiography measures the velocity of moving red blood cells and has become a noninvasive alternative to cardiac catheterization for assessment of hemodynamics.

Transesophageal echocardiography (TEE)

• Transesophageal echocardiography (TEE) provides a unique window for high-resolutionimaging of posterior structures of the heart, particularly the left atrium, mitral valve, and aorta.

Stress echocardiography

• Exercise stress testing is usually done with exercise protocols using either upright treadmill or bicycle exercise. In patients who are not able to exercise, pharmacologic testing can be performed by infusion of dobutamine to increase myocardial oxygen demand.

• Dobutamine echocardiography has also been used to assess myocardial viability in patients with poor systolic function and concomitant CAD; when used for this purpose, dobutamine is administered at lower doses than standard pharmacologic stress doses.

• Doppler echocardiography can be used at rest and during exercise in patients with valvular heart disease to determine the hemodynamic response of valve gradients and pulmonary pressures.

• In patients with low-output, low-gradient aortic stenosis, the response of the gradient to dobutamine stimulation is of diagnostic and therapeutic valu.

Rentgenoscopy is a very important instrumental method for the study of the cardiovascular system. Routine X-ray studies include roentgenoscopy and roentgenography. In direct projection, the patient faces the screen with the X-ray tube being behind the patient's back. In oblique projections, the patient is positioned at an angle of 45° to the screen: first with the right and then with the left shoulder forward.

When examining the silhouette of the heart and the great vessels in direct projection, it is necessary to pay attention to the magnitude of the angle formed by the bundle of the great vessels and the heart silhouette on the left. The angle becomes more significant when the left ventricle is enlarged. Since it is more pronounced in aortic incompetence, this configuration of the heart is known as "aortic". The left atrium is enlarged and the pressure in the pulmonary artery increases in mitral incompetence. In this connection the second and third arches of the left contour formed by the pulmonary trunk, the left pulmonary artery, and the auricle of the left atrium, become protruded. This configuration of the heart is known as "mitral".

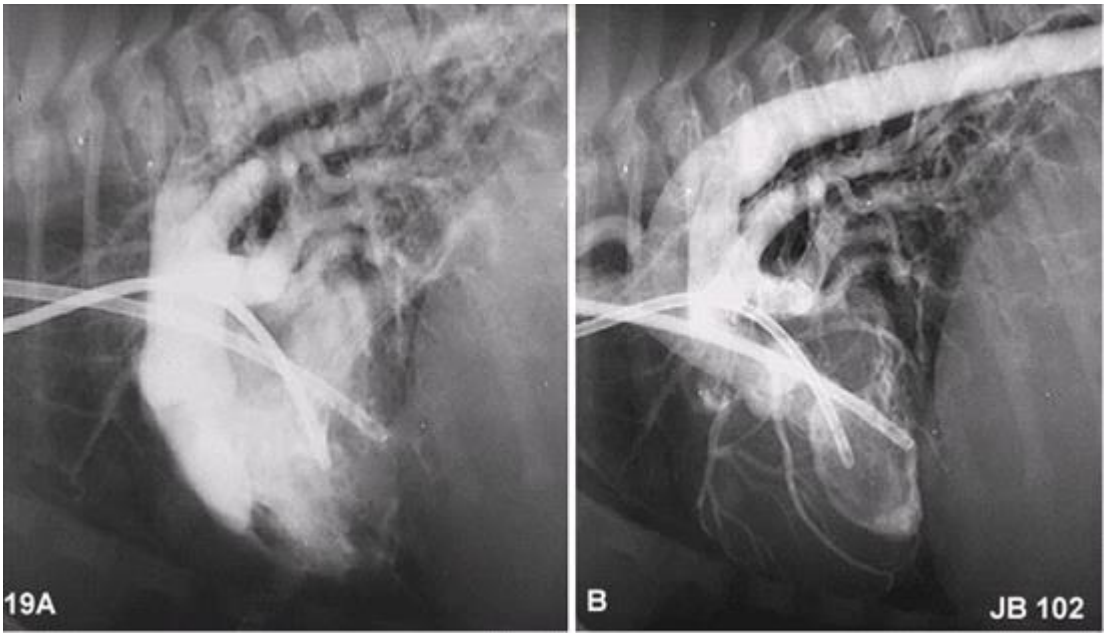

Image 20. Angiocardiography

The image was downloaded from website https://www.vin.com

Angiocardiography is the method of X-ray examination by which pictures of various heart chambers or the great vessels can be taken after administration of special contrast substances into them. Venous angiocardiography and selective angiocardiography are distinguished. In the former case a contrast substance (cardiotrast, diotrast, etc.) is injected into a peripheral vein and X-ray pictures are taken to record the entry of the substance into the right chambers of the heart and the vessels of the lesser circulation. The left chambers are poorly contrasted because the contrast substance is highly diluted in the blood flowing in the left chambers and the vessels of the lesser circulation. In selective angiography contrast substance is administered through a catheter directly into the right or left chambers of the heart.

Image 21. Computed tomography of the heart
The image was downloaded from website https://www.inovanewsroom.org

Angiocardiography is very useful in diagnosing congenital heart defects. It reveals pathological communications between the heart chambers and the great vessels, determines the direction and amount of blood ejected from one chamber of the heart to another, locates stenosed portions of the vessels, and determines the degree of stenosis. Moreover, angiocardiography helps diagnose complicated acquired heart defects and evaluate indications for surgical treatment in cases where clinical findings are insufficiently informative. Selective angiography of the aorta and

its branches (aortography) is used to study the condition of the vessels. This method is widely used to determine the condition of the coronary arteries (coronography).

Computed tomography of the heart creates an image of the heart, using the technology of CT, with or without injection of intravenous contrast to visualize the anatomy of the heart and vessels (including the aorta, the pulmonary veins and arteries), coronary circulation.

With magnetic resonance imaging of the heart we can identify:

1. Structural features of the chambers of the heart and blood vessels;
2. Intracardial hemodynamic;
3. Blood flow in large vessels;
4. Congenital heart disease;
5. Aortic aneurysm, coarctation;
6. Tumors of the heart;
7. Cardiomyopathy, especially hypertrophic cardiomyopathy;
8. Pulmonary hypertension of unknown origin, the pathology of the right heart

Laboratory research methods are very widely used in the clinic. The excretions and secretions of an organism, feces, blood, exudates and transudates are studied.

Laboratory tests are conducted in the following ways:

1) the study of the general properties of the material, including the physical (number, color, appearance, odor, contamination, relative density, etc.);

2) microscopic examination;

3) determination of various substances in the studied material (normal metabolic products, trace elements, hormones and their conversion products, etc.);

4) definition of not unpeculiar to the organism substances that appear only during the disease or intoxication;

5) bacteriological and virological studies;

6) serological diagnostics, and etc.

LIST OF FINAL CONTROL QUESTIONS

Basic methods of examination of patients in the clinic of internal medicine

1. Basic methods of diagnosis of internal diseases.
2. The scheme of questioning the subject. The main structural parts of the anamnesis.
3. The sequence of the general examination of the patient.
4. Types of physique and their main criteria.
5. The sequence of palpation of lymph nodes and the characteristics of the data.
6. Rules of examination of the head and neck.
7. The sequence of examination of the torso and limbs.
8. Static examination of the chest, the diagnostic value of the main symptoms.
9. Dynamic examination of the chest, diagnostic significance of the main symptoms.
10. Examination of the atrial area, the diagnostic value of the main symptoms.
11. The sequence of examination of the abdomen, the definition of the main symptoms.
12. The main properties of the pulse, rules and sequence of their definition. 13. Rules for measuring blood pressure. Determination of systolic and diastolic pressure by the method of Korotkov, calculation of pulse, mean dynamic pressure.
14. Palpation of the chest: the sequence, the clinical significance of the main symptoms.
15. Palpation of the atrial area, determination of the clinical significance of the symptoms found.
16. Superficial palpation of the abdomen: an algorithm for conducting and analyzing the data.
17. Theoretical principles and principles of deep methodical sliding palpation of the abdomen by the method of Obraztsov-Strazhesk.
18. Palpation of the sigmoid, cecum, terminal ileum, their properties are normal.
19. Rules of palpation of the ascending and descending colon, their properties are normal.

20. Methods for determining the lower limit of the stomach.

21. Palpation of the transverse colon, the main properties.

22. Rules of palpation of the liver, the diagnostic value of the main symptoms.

23. Palpation of the spleen.

24. The sequence of comparative percussion of the lungs. Basic percussion tones and the mechanism of their formation.

25. Algorithm of topographic percussion of the lungs. Topographic parameters of the lungs in normal and in pathology.

26. Percussion examination of the heart - relatively dull cardiac dullness: normal limits and their displacement with changes in the chambers of the heart.

27. Percussion examination of the heart - absolute cardiac dullness: normal limits and their displacement due to cardiac and extracardiac causes.

28. Percussion determination of the vascular bundle, its diagnostic value.

29. Percussion of the liver by the method of Exemplary: the sequence, the parameters are normal and in pathology.

30. Percussion of the liver by the method of Kurlov: the sequence, normal parameters and pathology.

31. Percussion determination of the boundaries of the spleen. Rules of carrying out, the reasons of increase in a spleen.

32. Auscultation of the lungs - determination of the main respiratory noises, their qualitative and quantitative changes.

33. Auscultation of the lungs - determination of additional respiratory noises, their classification, algorithm for characterizing the auscultatory picture of the lungs.

34. Mechanisms of formation and types of rales, their diagnostic value.

35. The main causes of crepitation and noise of pleural friction. Their diagnostic value and methods of differentiation.

36. Human silt and consistency study bronhofonii, its diagnostic value.

37. Auscultation of the heart - heart tones, the mechanism of their formation and changes in strength and timbre.

38. Splitting and bifurcation of heart tones, the concept of accentuation of the second tone.

30. Additional heart tones - quail rhythm and gallop rhythm.

40. Auscultation of heart murmurs: classification and conditions.

41. Auscultation of heart murmurs: sequence of characteristics, differences between organic and functional noises.

42. Diastolic functional noises (Flint, Coombs, Graham-Steele): conditions of occurrence and diagnostic value.

43. Rules of ECG analysis. Criteria for sinus rhythm, heart rate calculation and determination of the position of the electrical axis of the heart.

TASKS FOR FINAL CONTROL

Main rules of the interview and examination of patients. The basic methods of the examination of patients

1. To which section of the case history is the complaint of dyspnea entered:
   A. Present complaints
   B. Details of the complaints
   C. Questioning about the organs and systems
   D. Anamnesis morbi
   E. Anamnesis vitae

2. Inheritance linked to the gender is characteristic:
   A. Ulcer disease
   B. Hypertension disease
   C. Diabetes mellitus
   D. Hemophilia
   E. Bronchial asthma

3. Previous diseases are described in the following section:
   A. Anamnesis vitae
   B. Passport part
   C. Questioning about organs and the systems
   D. Present complaints
   E. Anamnesis morbi

4. Which section of the case history is called medical biography:
   A. Present complaints
   B. Passport part
   C. Anamnesis vitae
   D. Anamnesis morbi
   E. Asking about the organs and systems

5. A 28-year-old patient complains of a pronounced productive cough, weakness, perspiration, fatigue, loss of appetite. His main complaint is:
   A. Weakness
   B. Fatigue
   C. Loss of appetite
   D. Perspiration
   E. Productive cough

6. To which section is the complaint of weight loss entered?
   A. Asking about general condition
   B. Present complaints
   C. Anamnesis vitae
   D. Anamnesis morbi
   E. Asking about organs and systems

7. A detailed description of the complaints is entered to the following section:
   A. Asking about organs and systems

B. Present complaints
C. Anamnesis morbi
D. Anamnesis vitae
E. Passport part

8. Asking about the systems is started from:
A. The system the patient complains on
B. Nervous system
C. Cardiovascular system
D. Genitourinary system
E. Respiratory system

9. In which section of the case history are unhealthy habits described?
A. Present complaints
B. Anamnesis morbi
C. Anamnesis vitae
D. Asking about organs and systems
E. Asking about general condition

10. History taking is:
A. Subjective method
B. Objective method
C. Additional method
D. Laboratory method
E. Instrumental method

Answers

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

Physical and instrumental methods of examination of the broncho-pulmonary system. The interview and the general examination of patients with the respiratory system pathology. The bronchoobstructive syndrome

1. Paralitic chest shape is observed in:
A. Acute bronchitis
B. Pneumonia
C. Bronchopneumonia
D. Lungs tumor
E. Exudation pleurisy

2. A Boat-shaped chest is observed in:
A. rachitis
B. scoliosis
C. syringomyelia
D. tuberculosis
E. bronchitis

3. Barrel-shaped chest is typical for:
A. pulmonary tuberculosis
B. emphysema of the lungs
C. exudation pleurisy

D. pneumothorax
E. acute bronchitis

4. Enlargement of one part of the chest is observed in:
A. hydrothorax
B. pneumosclerosis
C. obstructive atelectasis of the lung
D. bronchopneumonia
E. bronchitis

5. Diminished one part of the chest is observed in:
A. Exudation pleurisy
B. pneumothorax
C. bronchopneumonia
D. pneumosclerosis
E. pulmonary emphysema

6. Kussmaul respiration is observed in:
A. diabetic coma
B. stroke
C. heart failure
D. lung failure
E. pulmonary tuberculosis

7. Lateral curvature of the spine is observed in:
A. lordosis
B. scoliosis
C. kyphosis
D. rachitis
E. kyphoscoliosis

8. Cheyne-Stocks respiration is typical for:
A. acute insufficiency of the brain circulation
B. pulmonary emphysema
C. pneumothorax
D. bronchial asthma
E. hydrothorax

9. Increased voice resonance is observed in:
A. hydrothorax
B. compression atelectasis
C. pulmonary emphysema
D. pneumothorax
E. pneumothorax

10. Decreased voice resonance is observed in
A. hydrothorax
B. exudation pleurisy
C. pulmonary emphysema
D. pneumothorax
E. Acute pneumothorax

Answers

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

Physical and instrumental methods of the examination of the cardiovascular system. The interview and the general examination of the patient with the disorder of the cardiovascular system. Physical methods of the examination

1. Which color of the skin is typical in the patients with aortic regurgitation?
   A. Pale
   B. Peripheral cyanosis
   C. Jaundice
   D. Diffuse cyanosis
   E. Purple
2. Edema of the feet, more pronounced in the evening, acrocyanosis, cold skin over edema are typical in:
   A. Pericarditis
   B. Glomerulonephritis
   C. Heart failure
   D. Liver cirrhosis
   E. Thyrotoxicosis
3. Cardiac hump is observed in:
   A. Mitral heart valvular disease that arises in 30 years old patient
   B. Aortic aneurism
   C. Congenital heart disease
   D. Pericarditis with effusion
   E. Hydrothorax
4. Which heart chamber takes part in the cardiac beat formation?
   A. Left ventricle
   B. Right ventricle
   C. Left atrium
   D. Right atrium
   E. Left atrium and left ventricle
5. Which color of the skin is typical in the patients with mitral stenosis?
   A. Pale
   B. Peripheral cyanosis
   C. Jaundice
   D. Diffuse cyanosis
   E. Purple
6. In which pathology protrusion of the heart region, leveling of the intercostals spaces are observed in inspection?
   A. Mitral stenosis
   B. Pericarditis with effusion
   C. Aortic aneurism
   D. Pulmonary artery stenosis
   E. Tricuspid regurgitation
7. In which pathology apex beat is impalpable?
   A. Right-sided pleurisy with effusion
   B. Right-sided lobar pneumonia
   C. Left-sided lobar pneumonia
   D. Left-sided pleurisy with effusion
   E. Right-sided spontaneous pneumothrorax
8. Which color of the skin is typical in the patients with aortic stenosis?
   A. Pale
   B. Peripheral cyanosis
   C. Jaundice
   D. Diffuse cyanosis
   E. Purple
9. Edema of the lower limbs, more pronounced in the evening is typical in:

A. Pericarditis
B. Glomerulonephritis
C. Heart failure
D. Liver cirrhosis
E. Thyrotoxicosis

A. Mitral regurgitation
B. Mitral stenosis
C. Pericarditis with effusion
D. Myocarditis
E. Aortic arch aneurism

10. In which pathology pulsation in the jugular fossae is observed?

Answers

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

RECOMMENDED LITERATURE

Basic

1. Kovalyova O. M. Propedeutics of Internal Medicine: textbook for English learning Students of higher medical schools. Part 1: Diagnostics /O. M. Kovalyova, T. V. Ashcheulova. – 5th ed. – Vinnytsya: Nova Knyha, 2020. – 424 p.

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