STATE ESTABLISHMENT “DNIPROPETROVSK MEDICAL ACADEMY OF HEALTH MINISTRY OF UKRAINE”

Essential Practical Skills in Internal Medicine

For students of higher medical educational institutions

Edited by the Corresponding member of National Academy of Medical Sciences of Ukraine, M.D., Ph.D., Professor Tetyana Pertseva

Dnipro

2017

УДК 616.1/.4-07-08(076.5)
ББК 54.1+53.4+Я7
Г6

Recommended by the Scientific Council of the State Establishment "Dnepropetrovsk Medical Academy of Health Ministry of Ukraine" as a teaching aid for students of higher educational institutions
Protocol of the meeting of the Scientific Council of the the State Establishment "Dnepropetrovsk Medical Academy of Health Ministry of Ukraine" No. 11 of June 20, 2017

Editor:

Tetyana Pertseva, MD – Corresponding member of National Academy of Medical Sciences of Ukraine, M.D., Ph.D., Professor, the Department of Internal Medicine 1, Rector of State Establishment "Dnipropetrovsk medical academy of Health Ministry of Ukraine".

Reviewers: Fushtei Ivan M., M.D., Professor, Vice-Rector Research, Chairman of Therapy, Clinical Pharmacology and Endocrinology Department of Zaporizhzhya Medical Academy of the Postgraduate Education of the Ministry of Health of Ukraine.

Konstantinovich Tetyana V., M.D., Ph.D., Professor of the department of propaedeutics of internal medicine of the Vinnitsa National Medical University. E. Pirogov.

Contributors:

Kateryna Gashynova, MD, Ph.D., Olena Myronenko, MD, Oleksandr Fesenko, MD, Ph.D., Ph.D.,Valeriia Dmytrychenko, MD, Ph.D., Larysa Botvinikova, MD, Ph.D., Kseniia Bielosludtseva, MD, Ph.D., Nataliya Kravchenko, MD, Ph.D.

The handbook presents basic algorithms of practical skills, studied during the course: "Internal Medicine: Module 1". The guidance is intended for use in the learning process of undergraduate medical students in higher education institutions.

The main purpose of practical training is acquiring by a physician basic working knowledge in the in-patient clinic and achieving basic competence in the leading spheres of clinical practice – ability to solve typical professional tasks (organizational, diagnostic and treatment, including emergency care).

Tasks of practical training in Internal medicine

1. The students should master:

- examination of patients with the most common therapeutic diseases;
- evaluation of data of the medical interview and examination of the patient;
- formulation of initial diagnosis;
- planning of additional investigations;
- monitoring the dynamics of the patient's condition and reflecting daily observations in diaries;
- correction of management plan according to the dynamics of the patient's condition under the physician's supervision;
- providing immediate and long-term prognosis;
- making recommendations for the case management at the out-patient unit;
- appropriate keeping of medical records;
- delivery of healthcare in emergency according to modern protocols.

2. The student should be familiar with:

- order of prescription, storage, drug inventory and administration of medications (especially of potent ones and drugs);
- work of physiotherapy department;
- work of functional diagnostics department and laboratory;
- work of department of morbid anatomy.

Structure of practical training

On-the-job training of a medical student "physician's in the hospital" is conducted in the 4th year of study during training course in internal medicine. Overall practice time is 45 academic hours. A student works as a doctor's assistant (subordinator) at a therapeutic department of a hospital. Under the supervision of the staff physician the student follows-up patients. The student is under the command of the physician-supervisor, head of the therapeutic department and practice supervisor (assistant professor of the Internal Medicine Department).

The beginning of practice is synchronous with the beginning of training course "Internal Medicine: Module 1". On the first day of practical training students have to come to the department of Internal Medicine to be distributed in the therapeutic departments of the hospital, where practical training will take place and undergo initial safety training on the organization of the hospital's work. Working day at the therapeutic department starts at 8:30 am.

Beginning with the first day of practical training the student keeps records in a diary (appendix 1), which reflects all kinds of activities performed during the day. Among these obligations are:

- participation in morning organization briefings;
- participation in pathologic and clinical conferences;
- work with patients in wards and filling in observation diaries;
- initial examination of patients with the physician-supervisor and formulation of initial diagnosis;
- planning additional investigations of the patient;
- defining case management tactics and making management plan by choosing adequate pharmacological and physical therapy;
- maintenance of patient's medical records;
- direct participation in diagnostic and therapeutic manipulations. The level of mastering of these manipulations is recorded in student's individual plan of practical training.

Responsibilities of the direct supervisor
(Head of Therapeutic Department)

1. Admits student at the training base, notes the date of arrival and departure, takes attendance of the student in the record.
2. Enables the student to master practical skills according to the individual plan and approved list.
3. On completion of practical training certifies documents (see the list of required documents) by the signature and personal stamp.

Responsibilities of the supervisor of practical training

1. Submits direction for practical training of students to healthcare facility (HCF).
2. Provides students' arriving to HCF, distributes them to departments and provides initial safety training.
3. Provides collaboration with direct supervisor on organization of practical training and gives the student all necessary educational learning materials.
4. Organizes providing students with all educational materials (textbook, example of diary maintenance, a list of necessary skills and requirements for evaluation of practical training by criteria "reviewed", "acquired", "mastered").
5. Controls workplace discipline during the course of practical training.
6. Consults the students on all issues of practical training.
7. At the beginning of academic classes controls the adequacy of the diary maintenance and the level of practical skills mastering.
8. Participates in the final control. Records all information on attendance of practical training, current, final and total scores, received by the student during practical training in the log.
9. Submits the report and proposals as for practical training to the person responsible for practical training.

Responsibilities of the person, liable for practical training

1. Provides registration of contractual relationships with the head of HCF.
2. Fills in passports of practical training bases. The passports include personal data of supervisors, department capacity, technical possibilities for mastering practical skills according to the program and maximum number of students which clinical base can admit simultaneously.
3. Executes direction of students to HCF, conducting practical training.
4. Controls the work of supervisors from the Internal Medicine Department.
5. Controls methodical support of supervisors and students.
6. Controls filling in summary information of students' evaluation in the log.
7. Submits the report on traineeship to the department in charge of practical training of every faculty (attendance, workplace discipline, evaluation).
8. Submits propositions on optimization of traineeship to the department in charge of practical training.

Instructions for filling in individual student's plan of practical skills

At the end of the practical training the evaluation of the level of practical skills mastering is performed. Results of evaluation are recorded into the "Individual student's plan of practical skills". The list of necessary practical skills and three degrees of its mastering ("reviewed", "acquired" and "mastered") are arranged in the relevant sections of the tables.

The level "reviewed" – the student has theoretical understanding of the methodology of manipulations performance and/or was present during its performance, but can't carry out the procedure(s) without assistance, or performs it with gross mistakes.

The level "acquired" – the student has theoretical understanding of the methodology of manipulations performance and/or was present during its performance and directly took part in carrying out of the latter, but makes mistakes in methodology or interpretation of results, if it is a diagnostic manipulation.

The level “mastered” – the student has theoretical understanding of the methodology of manipulations performance and/or attended the manipulation; mastered the method of performing and interpretation of the results and is able to perform the skill on his/her own.

A mark is to be written in the appropriate column according to the level of practical skills handling, date of the exam, and signature of the immediate supervisor or supervisor of the practical training.

The main documents to be completed during the practical training

1. The direction card to traineeship, which indicates the start and end date of practical training. The immediate supervisor signs the card and certifies it with a personal stamp.
2. The practice diary with signatures and stamps of the direct supervisor.
3. “Individual student’s plan of practical skills” with the signature of the supervisor of practical training.

METHODOLOGICAL INSTRUCTIONS ON MAINTENANCE OF MEDICAL RECORDS IN THE HOSPITAL

During the course of practical training, the student should get the basic skills on keeping of medical records. The main documents for assimilation are:

1. Medical record of the in-patient department.
2. Case record of the out-patient (of the in-patient) department.

MEDICAL RECORD OF THE IN-PATIENT DEPARTMENT

The most important document is the Medical Record of the in-patient department. It includes medical case history, medication administration record (MAR), temperature sheet, statistical card and discharge summary. The following algorithm of actions with case history is provided to acquire practical skills in correct quick examination of the patient, formulation of preliminary diagnosis and drawing up of examination and treatment plan.

Stage I: personal data and the data necessary for record keeping on patient's transfers within a hospital and statistical processing of data (to be filled in on the front page of the patient's case history).

It is necessary to clear up the following information:

- patient's first name and last name;
- date of birth;
- gender;
- height and weight;
- place of work with full name, occupation; it is needed to fill in sick-leave;
- address of the permanent residence, phone number;
- that who referred the patient to the hospital, date of initial reference to a doctor;
- diagnosis, made in the institution that referred the patient to the hospital.

Also, on the front page the following information should be presented:

- diagnosis on hospitalization;
- clinical diagnosis made not later than on the third day of hospital stay;
- final clinical diagnosis, that is cleared up on discharge of the patient from the hospital;
- date and time of admission and discharge from the hospital or death;
- presence of drug allergy;
- number of the sick-leave;
- total number of days of hospital staying.

Stage II: Initial examination of the patient

Initial examination of the patient is performed by the generally accepted algorithm:

MEDICAL INTERVIEW

- taking patient's complaints with obligatory detailing;
- history taking;
- patient's life history with a focus on the facts that can impact on the disease directly or indirectly;
- allergic history. It is very necessary to pay attention to the presence of drug allergy.

EXAMINATION

- state of consciousness;
- the patient's position;
- constitutional type;
- condition of the skin and visible mucous membranes (color, moisture, presence of damage, skin tightness, edemas, state of the superficial veins, dermographism);
- condition of the subcutaneous fat;

- face (symmetry, changes of the eyes, presence of damages, developmental defects);
- mouth (changes of the tongue, teeth, peripharyngeal ring);
- neck (deformations, thyroid gland changes);
- condition of the joints (presence of deformations, the range of motions);
- respiratory rate, distance wheezing, involving of additional musculature in respiration, synchronic work of the chest during respiration;
- form of the chest (normosthenic, asthenic, hypersthenic, pathological changes);
- spinal column (physiological bends, abnormal curvature);
- anterior abdominal wall (shape, size, presence of hernias, visible pulsations and/or peristalsis, postoperative scars);
- groin area (presence of hernias, changes from the side of genitals);
- presence of abnormal motion activity;
- determining skin reflexes and skin sensitivity;

PALPATION

- skin (moisture, skin tightness, edemas, temperature, dermographism);
- lymphatic glands;
- thyroid gland;
- thorax (pain on palpation, resistance, apical beat, voice tremor);
- pulse (rate, power, resistance, tension, uniformity on both arms);
- anterior abdominal wall (surface palpation to determine tension and / or pain, deep palpation with defining specific symptoms);
- joints (pain, temperature, mobility, fluctuation, etc.).

PERCUSSION

- topographic percussion of the chest (defining lung contours, mobility of the lower pulmonary edge, height of apexes standing, comparative percussion of the lungs);
- determining absolute and relative borders of the heart;
- defining boundaries and sizes of the liver and spleen;
- defining the lower edge of the stomach;
- defining free fluid in the peritoneal cavity;
- presence of tenderness on percussion of the lumbar area.

AUSCULTATION

- determining physiological and pathological respiratory sounds;
- heart sounds (volume, rhythm, rate, regularity, abnormal rhythms, presence of auscultatory phenomena);
- determining of peristalsis presence.

ADDITIONAL INFORMATION

- appetite;
- sleep;
- general neuropsychological state, mood;
- menses, other vaginal discharge;
- stool;
- urination.

Filling in case histories, it is necessary to describe in more details the system in which the disease is diagnosed/suspected, which caused hospitalization. Thus, for rheumatologic patient, the state of the musculoskeletal system is described in details, for hematologic – system of hemopoiesis, for endocrinologic – detailed description of those glands, that are accessible for physical inspection.

During the initial examination it is also necessary to carry out some instrumental and/or laboratory tests:

- blood pressure measurement;
- thermometry;
- pulseoxymetry;
- if needed: electrocardiography (ECG), complete blood count and urine tests, biochemical blood analyses (if necessary to clarify level of findings, which are specific already on the early stage of patient's management), chest and/or abdomen X-ray, and ultrasound examination.

In the protocol of the patient's examination, all information obtained on physical examination is recorded. Based on the presence of pathology, the initial diagnosis is established. The initial diagnosis is established according to a leading syndrome and symptom complex of the patient.

Stage III: formulation of the preliminary diagnosis

The diagnosis consists of two basic components:

1. Clinical-anatomical diagnosis (pneumonia, hypertension, etc. The exception is only ischemic heart disease and its forms, in which there is no need to mention atherosclerosis).
2. Clinical-functional part, which includes complications if there are any.

Comorbidities are essential for the future patient's management. If it is possible, at the stage of the initial examination of the patient it is necessary to show in fullest clinical-anatomical and clinical-functional diagnosis of concomitant diseases.

Stage IV: drawing up examination and treatment plan

Examination and treatment plan is drafted individually for every patient, considering peculiarities of the disease course, based on the approved local protocols of diagnosis and treatment.

The plan of examination includes laboratory, instrumental methods of examination and counseling by profile specialists.

The treatment plan consists of defining patient's regimen, diet, water intake regimen, drug treatment, physiotherapeutic procedures and exercise therapy. The plan approved during the initial examination is not final. It may be supplemented and changed according to the available data and current patient's condition.

Stage V: dynamic follow-up of the patient.

Every day the patient is followed-up, information is recorded in the diary. The diary includes records on general dynamics of the patient's condition, changes in the initial complaints, leading physical data and vital signs: blood pressure, heart rate, pulse rate, respiratory rate, data on pulseoxymetry, temperature and other ones, which are important to determine the state of the particular patient. Information about sleep, appetite, physical activity, frequency of urination and stool is recorded. At the end of the diary, it is necessary to specify and briefly justify supplements and changes in the treatment and/or examination of the patient. It is essential to indicate which doctor's administrations were not carried out and for what reason, if such facts had been established.

On the third day of patient's hospital stay initial diagnosis should be reviewed and justified on the basis of the obtained additional investigations` results. Justification of the clinical diagnosis is based on:

1. Clinical and paraclinical data, distinguishing leading symptom complex.
2. The analysis of clinical presentation development and causes of the disease in dynamics (history of particular exacerbation, history of past exacerbations).

Every 10 days of patient's hospital stay, interim epicrisis is recorded in the case history. It includes the following information: dynamics of the main symptoms of the disease, data of additional methods of examination, administered treatment and its results, further tactics of the patient's management.

MEDICATION ADMINISTRATION RECORD (MAR)

The document is intended for coordinated work of doctors, nurses and paramedical personnel in the hospital. In MAR the regimen, diet, medications with exact dosage, frequency and route of administration, physiotherapeutic procedures are recorded orderly. The nursing staff registers the procedures, which have been carried out. The physician makes changes and supplements, monitors carrying out of his/her administrations.

TEMPERATURE SHEET

Obligatory temperature sheet should contain information on the dynamics of body temperature. It may also contain the following information: pulse, blood pressure, weight, respiratory rate, the amount of expectorated sputum, diuresis, and stool frequency. In patients with diabetes mellitus - level of blood and urine glucose, the dose of insulin should be recorded.

EPICRISIS

(Discharge summary)

Epicrisis is written on patient's discharge from the hospital according to the following example.

The patient (last name and first name), age, stayed in the department from (date is indicated) till (date is indicated). He/she was hospitalized by the direction of (specify the establishment that referred the patient), taken urgently or according to schedule. The purpose of hospitalization (treatment, additional tests, was directed by the military enlistment office, etc.). In the hospital, the following diagnosis was established: the complete clinical diagnosis with a short substantiation. Data, which made it possible to justify diagnosis without describing in detail all the clinical data and conducted examination techniques should be indicated. Results of specialist's consultations should be noted. All prescribed medications with indication of active substances and trade names, dosage and term

for use are listed (for antibiotics, hormones and cytostatics it is mandatory!). The total assessment of effectiveness of therapy: recovery, improvement (without changes or worsened) and prognosis of recovery and life is given. At the end of the summary, plan of the patient's management after discharge from the hospital with detailed recommendations is provided. Expertise of a stable or temporary disability is performed (get back to work or discharged with the sick leave with indication of date visiting the doctor at the next stage, justification for referral for the working capacity examination). Epicrisis is necessarily certified by the signature and personal seal of the physician, head of the department and stamp of the hospital.

RECOMMENDATIONS ON REGISTRATION AND ANALYSIS OF ECG

Electrocardiography (ECG) is a method of graphic registration of changes of cardiac potential differences, which arise during the process of myocardial excitation.

ECG REGISTRATION TECHNIQUE

Facilities for electrocardiography

Electrocardiographs should be placed in a dry location at temperatures not below than 10°C and not higher than 30°C. During the work the ECG device must be earth grounded.

ECG is performed in a special room at an appropriate distance from potential sources of electrical disturbances: electric motors, physiotherapeutic and X-ray rooms, switchboards. The couch must be placed at a distance not less than 1,5–2 m from the cables. It is reasonable to shield the couch and put a blanket with the sewn wire cloth, which is earth grounded, under the patient.

Investigation is conducted after 10–15 minutes' rest and not earlier than 2 hours after meal. The patient should be stripped to the waist. Legs also should be free from clothes. ECG recording is usually conducted in the patient's supine position, this allows to achieve maximal muscle relaxation.

Applying of electrodes

In clinical practice 12 leads of ECG are used the most widely. Recording of them is mandatory in every ECG examination of the patient: 3 standard leads, 3 enhanced unipolar leads from the limbs and 6 thoracic leads.

On the inner surface of the legs and forearms in their lower thirds, 4 plate electrodes are placed by means of rubber belts. On the patient's chest one or some (in multi-channel recording) thoracic electrodes are applied, using rubber suction cup. To improve ECG quality and to reduce the number of induced currents, it is necessary to ensure good contact of electrodes with the skin. Do this requires:

1) previously to degrease the skin with alcohol in places of electrodes applying;

2) to moisten the skin with soapy solution in places of electrodes applying in case of hirsuties (excessive hair growth);
3) to use the electrode paste or moisten the skin profusely with 5–10% solution of sodium chloride in places of electrodes applying.

Attachment of wires to the electrodes.

The wire which comes from the electrocardiograph and marked with a different color is attached to every electrode placed on the extremities or on the surface of the chest. Generally accepted marking of incoming wires is: right hand – red color; left hand – yellow; left leg – green, right leg (earth-grounding of the patient) – black; chest electrode – white (fig.1).

Enhanced leads from the limbs register potential difference between one of the limbs on which an active positive electrode of this lead is applied (right arm, left arm or leg) and the average potential of the other two limbs. So aVR – enhanced lead from the right hand; aVL – enhanced lead from the left hand; aVF – enhanced lead from the left foot.

To record the ECG, 6 generally accepted positions of active electrode on the front and lateral surface of the chest are used, they are connected with a combined Wilson's electrode and form 6 chest leads:

- lead V1 – in the fourth intercostal space along the right edge of the breastbone;
- lead V2 – in the fourth intercostal space along the left edge of the breastbone;
- lead V3 – between the positions of V2 and V4, approximately on the level of the fourth rib along the left parasternal line;
- lead V4 – in the fifth intercostal space along the left medioclavicular line;
- lead V5 – on the same level in horizontal plane as in V4, along the left anterior axillary line;
- lead V6 – along the left medial axillary line on the same level in horizontal plane, as electrodes of V4 and V5 leads.

So, to the electrode V1 the wire with tag of red color is connected; to the electrode V2 – yellow, V3 – green, V4 – brown, V5 – black and V6 – blue or purple (fig. 2).

Leads V7–V9. Active electrode is fixed along the posterior axillary (V7), scapular (V8) and paravertebral (V9) lines on the horizontal level, on which electrodes V4–V6 are placed. These leads are usually used for the more exact diagnostics of focal changes in the myocardium in the posterior-basal areas of the left ventricle.

Leads V3R–V6R. Thoracic (active) electrode is fixed on the right side of the chest in positions symmetric with usual sites of electrodes applying. These leads are used to diagnose hypertrophy of right parts of the heart.

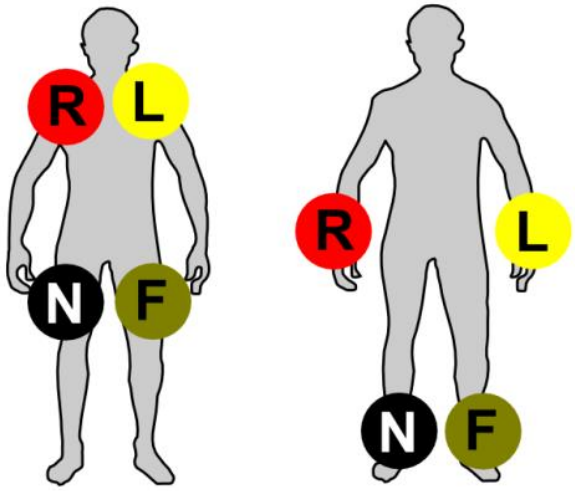


Fig. 1. Scheme of applying standard and enhanced electrodes

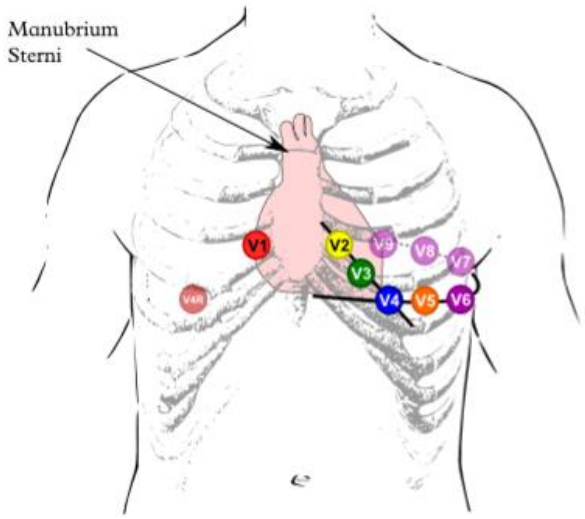


Fig. 2. Scheme of thoracic electrodes fixing

Choice of electrocardiograph amplification

Average amplification of each channel is selected in such a way that voltage of 1 mV causes deviation of galvanometer and recording systems equal to 10 mm. If needed, it is possible to change the amplification: to lower, in too large amplitude of ECG waves (1 mV = 5 mm), or to increase in small amplitude (1 mV = 15 or 20 mm).

ECG recording. ECG recording is performed at quiet breathing and at the height of the inspiration (in lead III). In every lead not less than 4 cardiac cycles of PQRST is recorded. At the beginning of every curve, calibrated signal is shown. As a rule, ECG is recorded at a speed of paper movement of 50 mm/s. A lower rate (25 mm/s) is used if longer period of ECG recording is needed, for example – for arrhythmias diagnosing. To analyze the duration of the waves and ECG intervals, the following calculations may be done**:**

In recording speed of 50 mm/sec

- 3000 mm in 60 seconds = 3000 small cells for 1 min = 600 large cells in 1 min
- 1 small cell = 1 mm = 0,02 sec, 1 large cell = 5 mm = 0,1 sec
- 1 sec = 50 mm (5 cm) = 10 large/50 small cells
- 3 sec = 150 mm (15 cm) = 30 large/150 small cells.

In recording speed of 25 mm/sec

- 1500mm in 60 seconds = 1500 small cells in 1 min = 300 large cells in 1 minute
- 1 small cell = 1 mm = 0,04 sec, 5 mm = 0,2 sec
- 1 sec = 25 mm (2,5 cm) large = 5 large cells/25 small cells
- 3 sec = 75 mm (7,5 cm) = 15 large /75 small cells.

Immediately after the investigation, on the paper tape the name and surname of the patient, date of birth, date and time of the investigation is written.

Waves, intervals and segments of ECG (fig. 3):

- P – depolarization of atria (right and left);
- P-Q interval– the distance between the beginning of P wave and beginning of Q wave, time of impulse conducting through the atrio-ventricular node from the atria to the ventricles;
- P-Q segment– the distance between the end of P wave and beginning of Q wave;
- QRS – the distance between the beginning of Q wave and the end of S wave, ventricular depolarization;
- Q – the first negative wave of the complex after P;
- R – positive wave;
- R' – the second positive wave;
- S – negative wave after R;
- Segment S-T (RS-T) – the distance between the end of QRS complex and beginning of T wave, the period of cardiac cycle when both ventricles are embrassed by excitation; point J ("joint") – end place of QRS complex and the beginning of ST segment (fig. 4);
- T – repolarization of the ventricles;
- Interval Q-T – the distance from the beginning of QRS complex to the end of T wave, the electrical systole of the ventricles (depolarization and repolarization) (fig. 5);
- U – post-depolarization of the ventricles;

Small letters (q, r, s) are used for waves of the small amplitude (less than 5 mm).

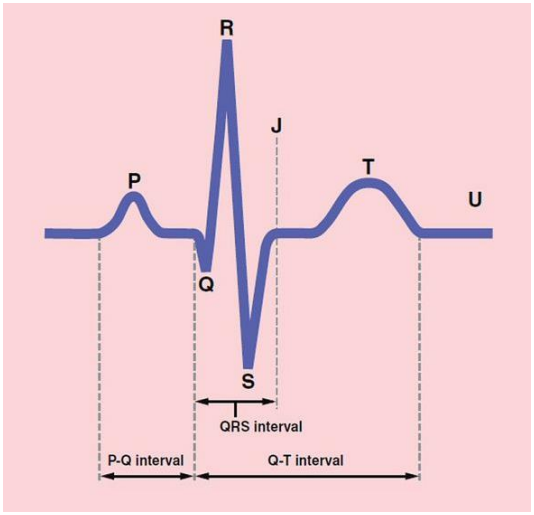


Fig. 3. Waves, intervals and segments of ECG

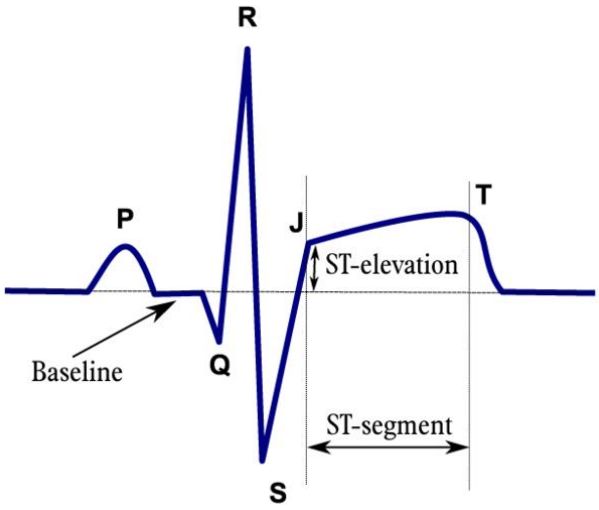


Fig. 4. Defining of ST segment and “j” point on the ECG

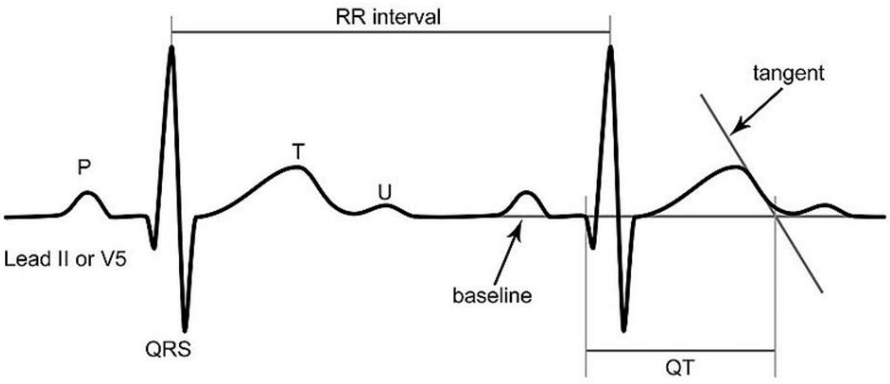


Fig. 5. Defining of Q-T interval

Version of normal ECG (25 mm/s) is shown on fig. 6.

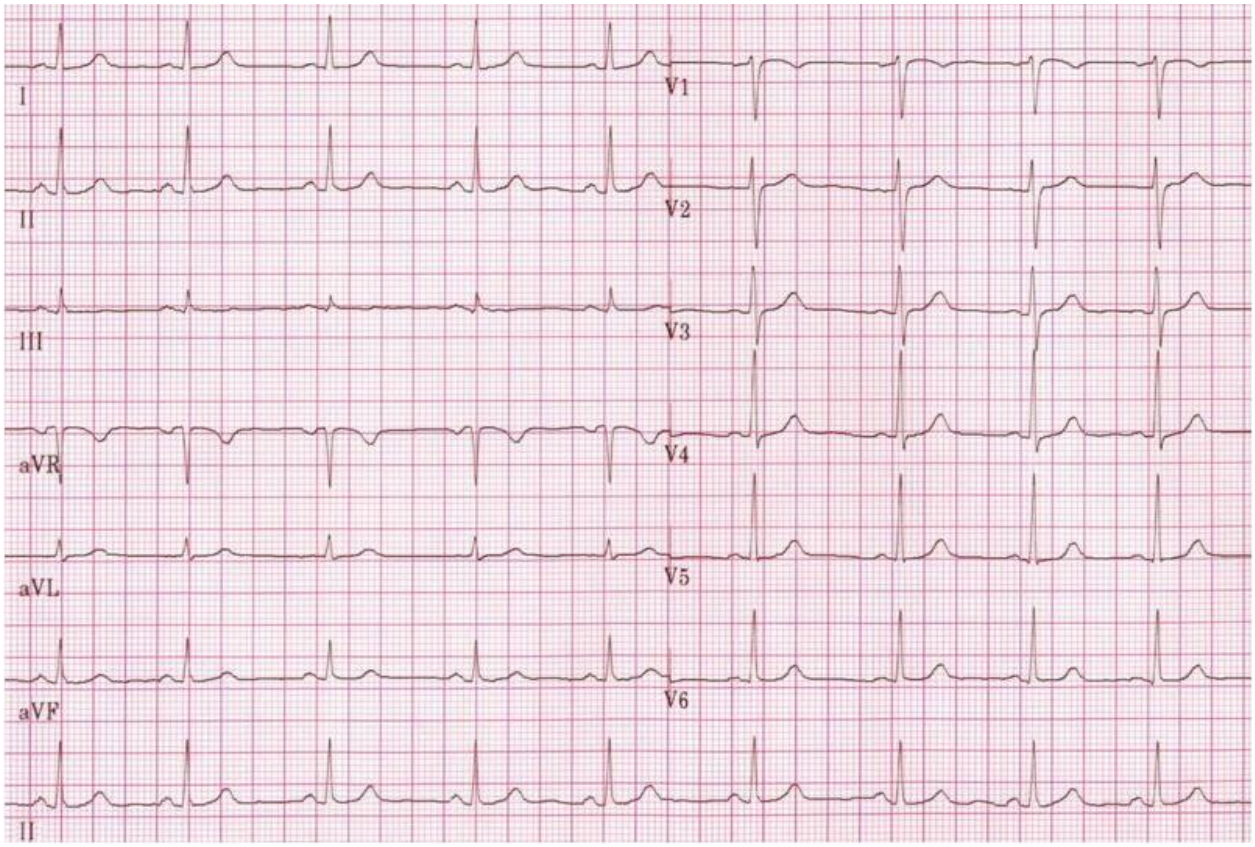


Fig. 6. Normal 12-leads ECG

GENERAL PLAN OF ECG DECODING

I) Analysis of the heart rhythm and conductivity:

1) defining of excitement source;

2) assessing of regularity of heart rate;

3) calculation of HBR;

4) evaluation of conductivity function.

II) Defining of position and rotation of electrical heart axis in the frontal plane;

III) Analysis of atrial P wave.

IV) Analysis of ventricular complex QRST:

1) analysis of QRS complex;

2) analysis of S-T segment;

3) analysis of T wave;

4) analysis of Q-T interval.

V) Electrocardiographic conclusion.

For more detailed ECG analysis, algorithm of record decoding, regarding the most common changes is given.

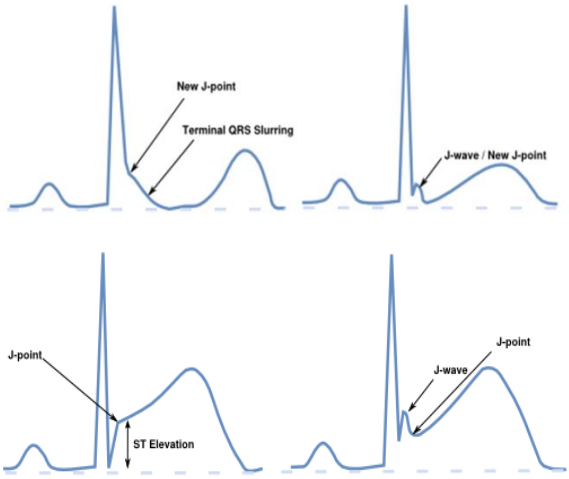


Fig. 7. Elevation of ST segment due to early ventricular repolarisation syndrome

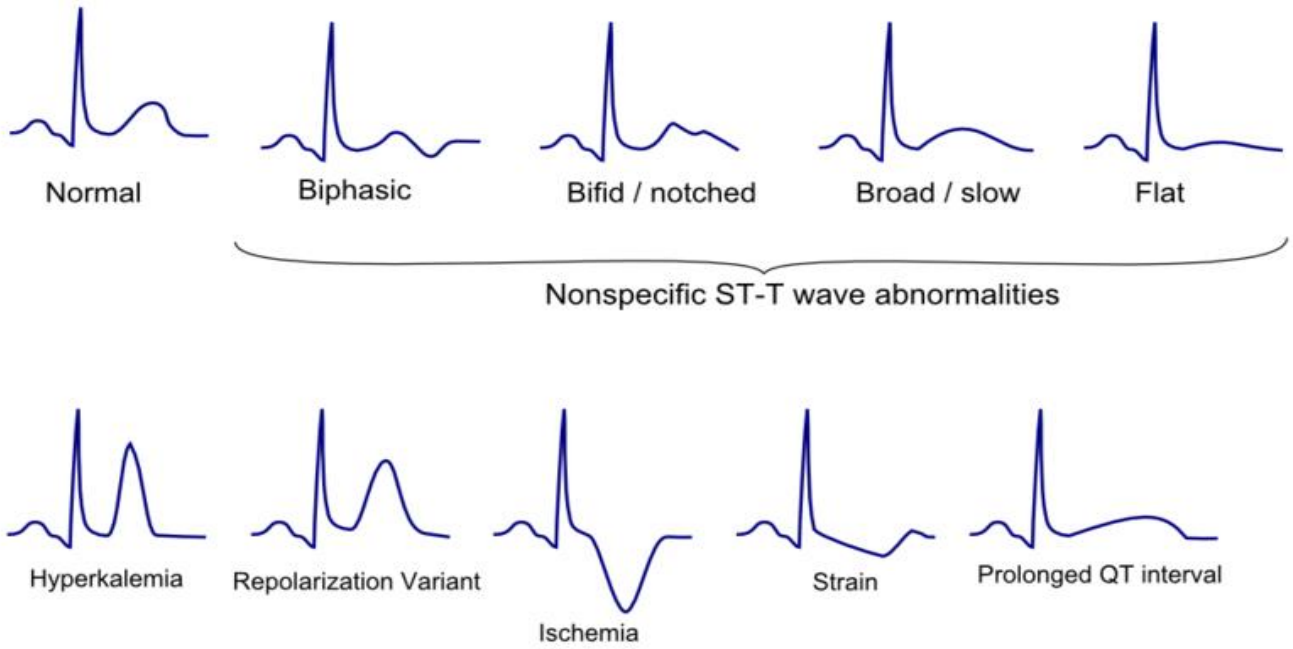
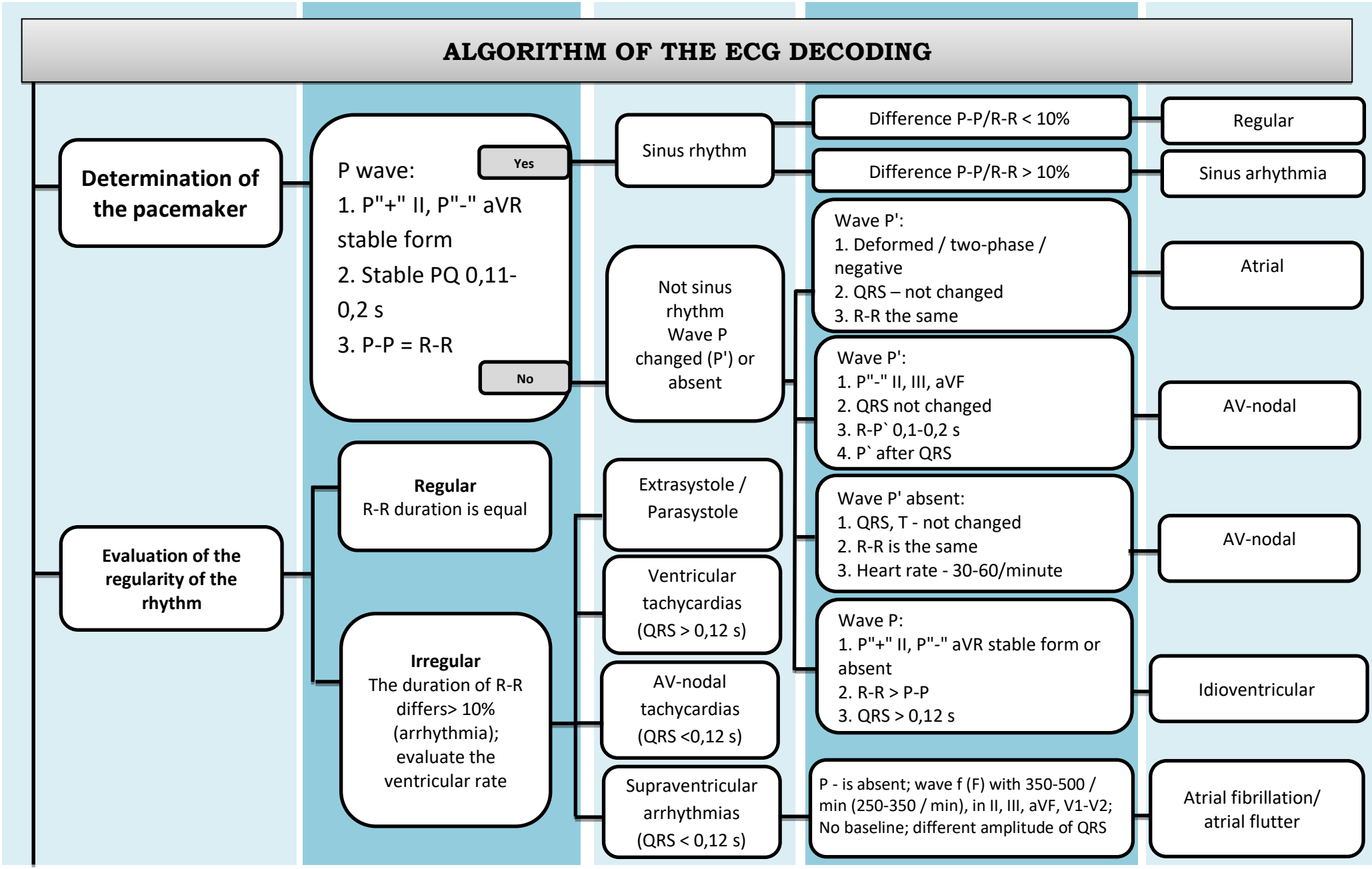


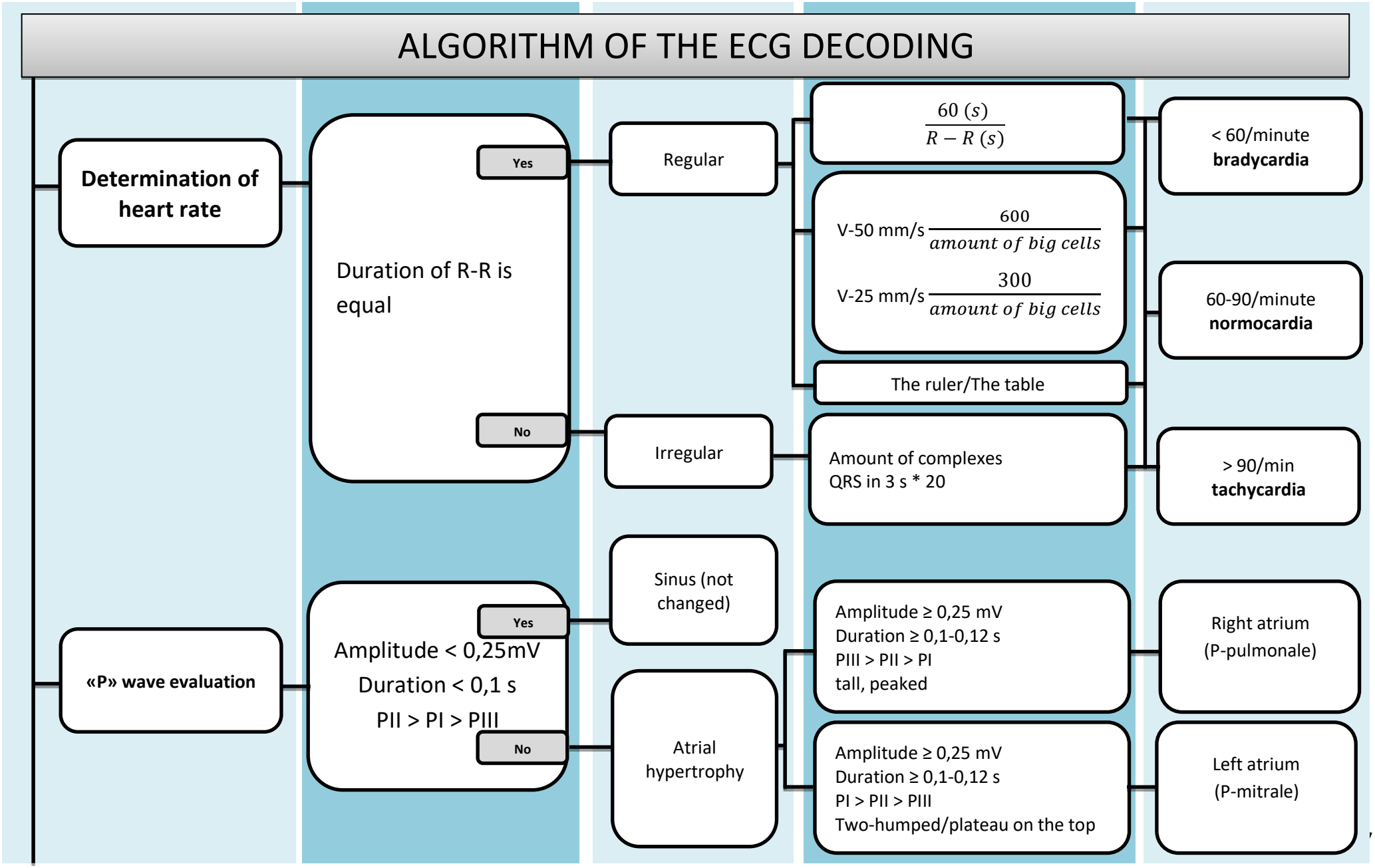
Fig. 8. Changes of T wave on ECG. Voltage is saved QRS > 5 mm standard leads, QRS > 8mm thoracic leads

Examples of conclusion formulation.

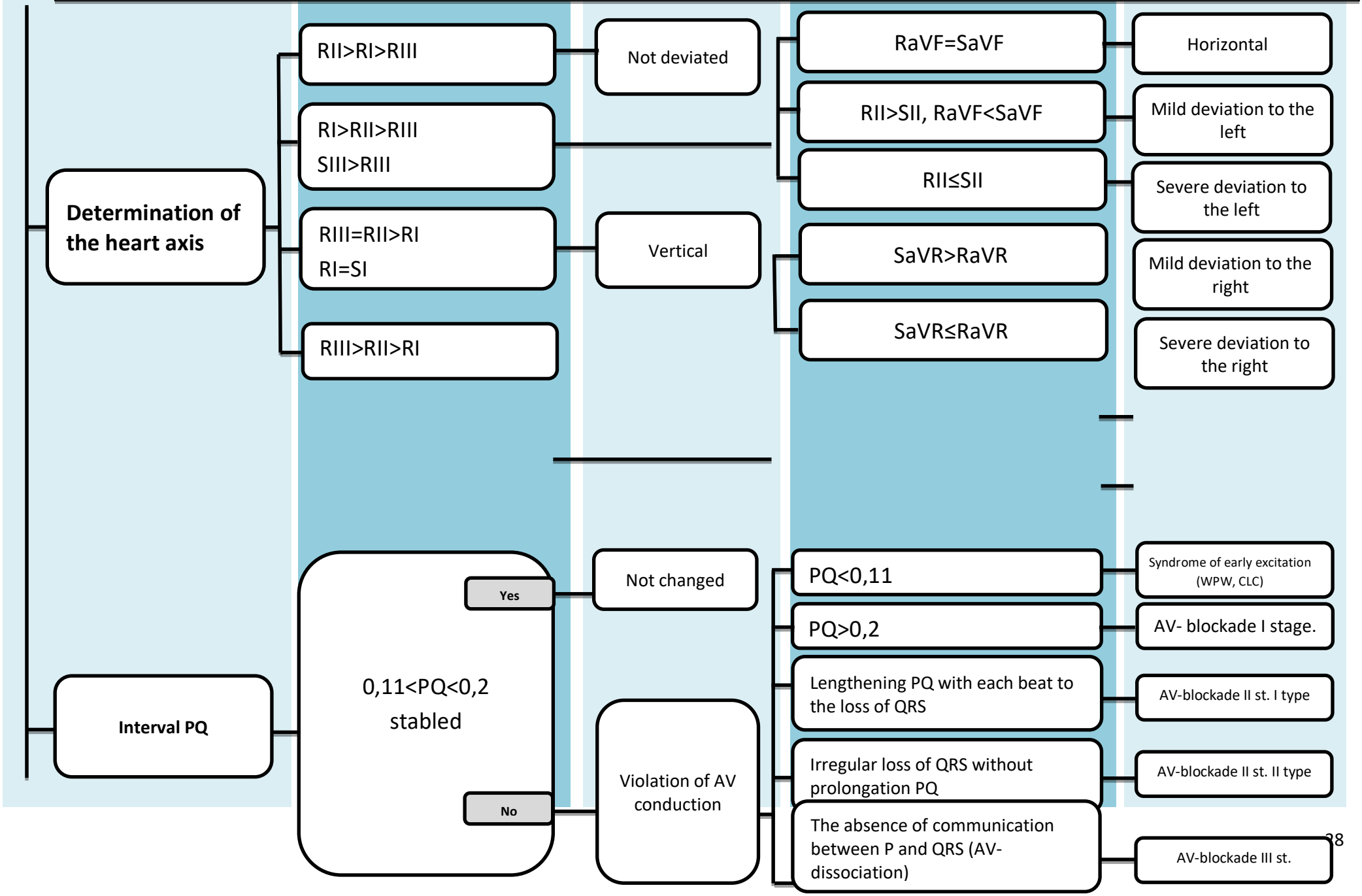
Example 1. Conclusion: Sinus rhythm, regular. Heart rate is 68 bpm. Normal heart axis. Voltage is sufficient.

Example 2. Conclusion: Non-sinus rhythm, irregular. Atrial fibrillation with the ventricular rate of 110/min. Horizontal heart axis. Signs of LVH. Voltage is sufficient.

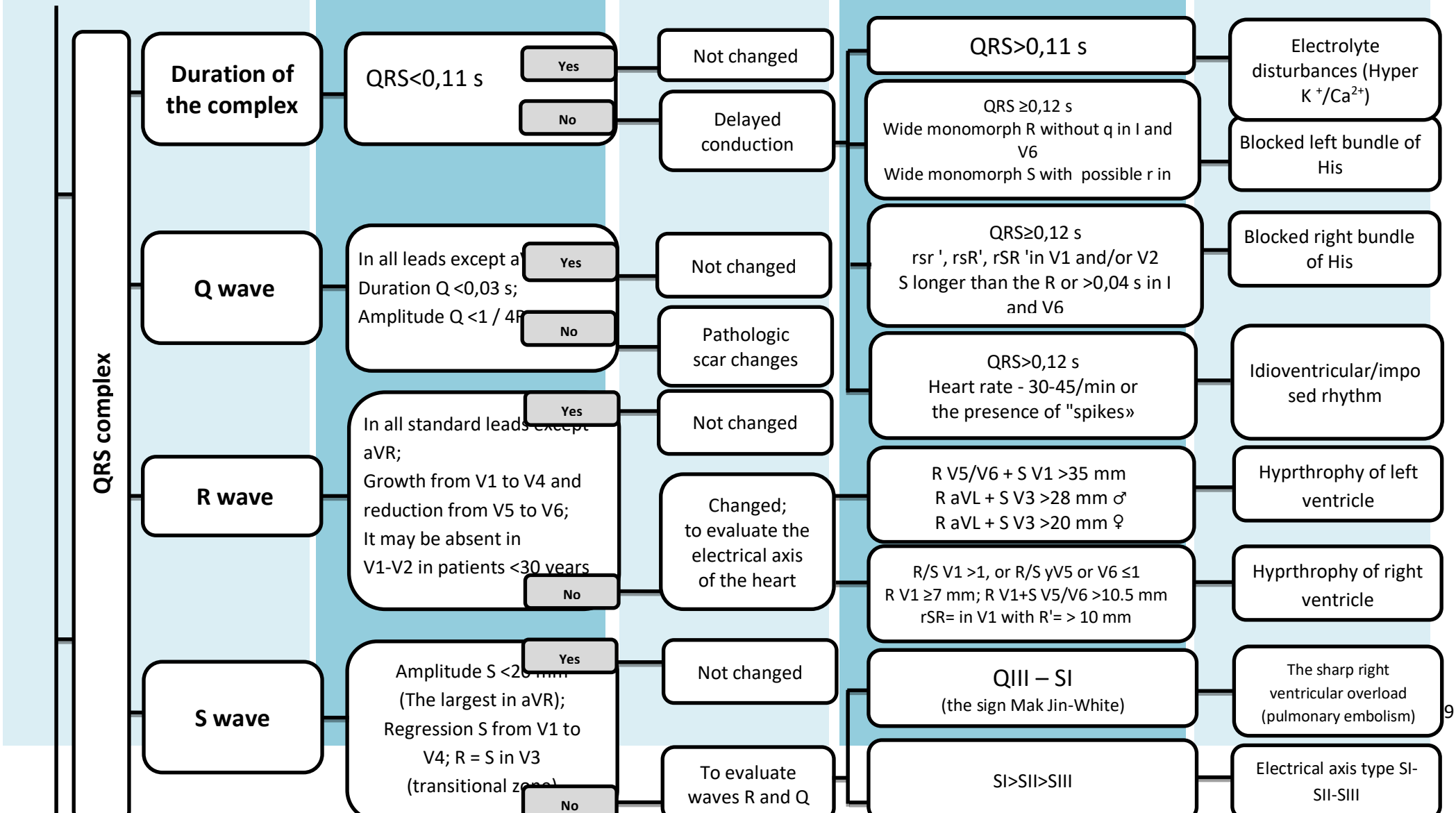

ALGORITHM OF THE ECG DECODING
Determination of the pacemaker
P wave:
1. P"+" II, P"-" aVR stable form
2. Stable PQ 0,11-0,2 s
3. P-P = R-R
Yes
No
Sinus rhythm
Difference P-P/R-R < 10%
Regular
Difference P-P/R-R > 10%
Sinus arhythmia
Not sinus rhythm
Wave P changed (P') or absent
Wave P':
1. Deformed / two-phase / negative
2. QRS – not changed
3. R-R the same
Atrial
Wave P':
1. P"-" II, III, aVF
2. QRS not changed
3. R-P` 0,1-0,2 s
4. P` after QRS
AV-nodal
Wave P' absent:
1. QRS, T - not changed
2. R-R is the same
3. Heart rate - 30-60/minute
AV-nodal
Wave P:
1. P"+" II, P"-" aVR stable form or absent
2. R-R > P-P
3. QRS > 0,12 s
Idioventricular
Evaluation of the regularity of the rhythm
Regular
R-R duration is equal
Irregular
The duration of R-R differs> 10% (arrhythmia); evaluate the ventricular rate
Extrasystole / Parasystole
Ventricular tachycardias (QRS > 0,12 s)
AV-nodal tachycardias (QRS <0,12 s)
Supraventricular arrhythmias (QRS < 0,12 s)
P - is absent; wave f (F) with 350-500 / min (250-350 / min), in II, III, aVF, V1-V2; No baseline; different amplitude of QRS
Atrial fibrillation/ atrial flutter


ALGORITHM OF THE ECG DECODING
Determination of heart rate
Duration of R-R is equal
Yes
No
Regular
Irregular
$\frac{60\ (s)}{R-R\ (s)}$
V-50 mm/s $\frac{600}{amount\ of\ big\ cells}$
V-25 mm/s $\frac{300}{amount\ of\ big\ cells}$
The ruler/The table
Amount of complexes QRS in 3 s * 20
< 60/minute bradycardia
60-90/minute normocardia
> 90/min tachycardia
«P» wave evaluation
Amplitude < 0,25mV
Duration < 0,1 s
PII > PI > PIII
Yes
No
Sinus (not changed)
Atrial hypertrophy
Amplitude ≥ 0,25 mV
Duration ≥ 0,1-0,12 s
PIII > PII > PI
tall, peaked
Amplitude ≥ 0,25 mV
Duration ≥ 0,1-0,12 s
PI > PII > PIII
Two-humped/plateau on the top
Right atrium (P-pulmonale)
Left atrium (P-mitrale)

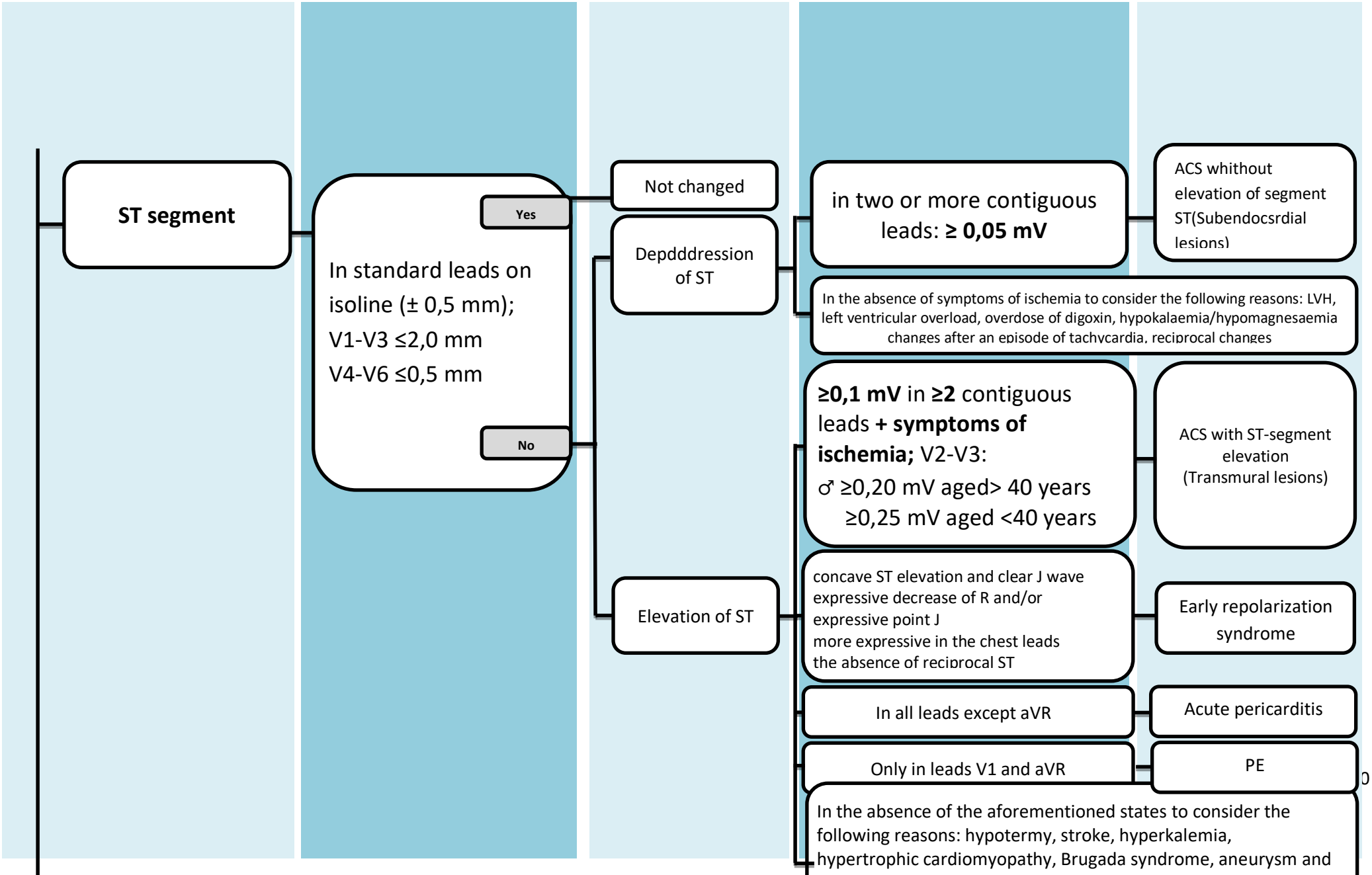
ALGORITHM OF ECG DECODING



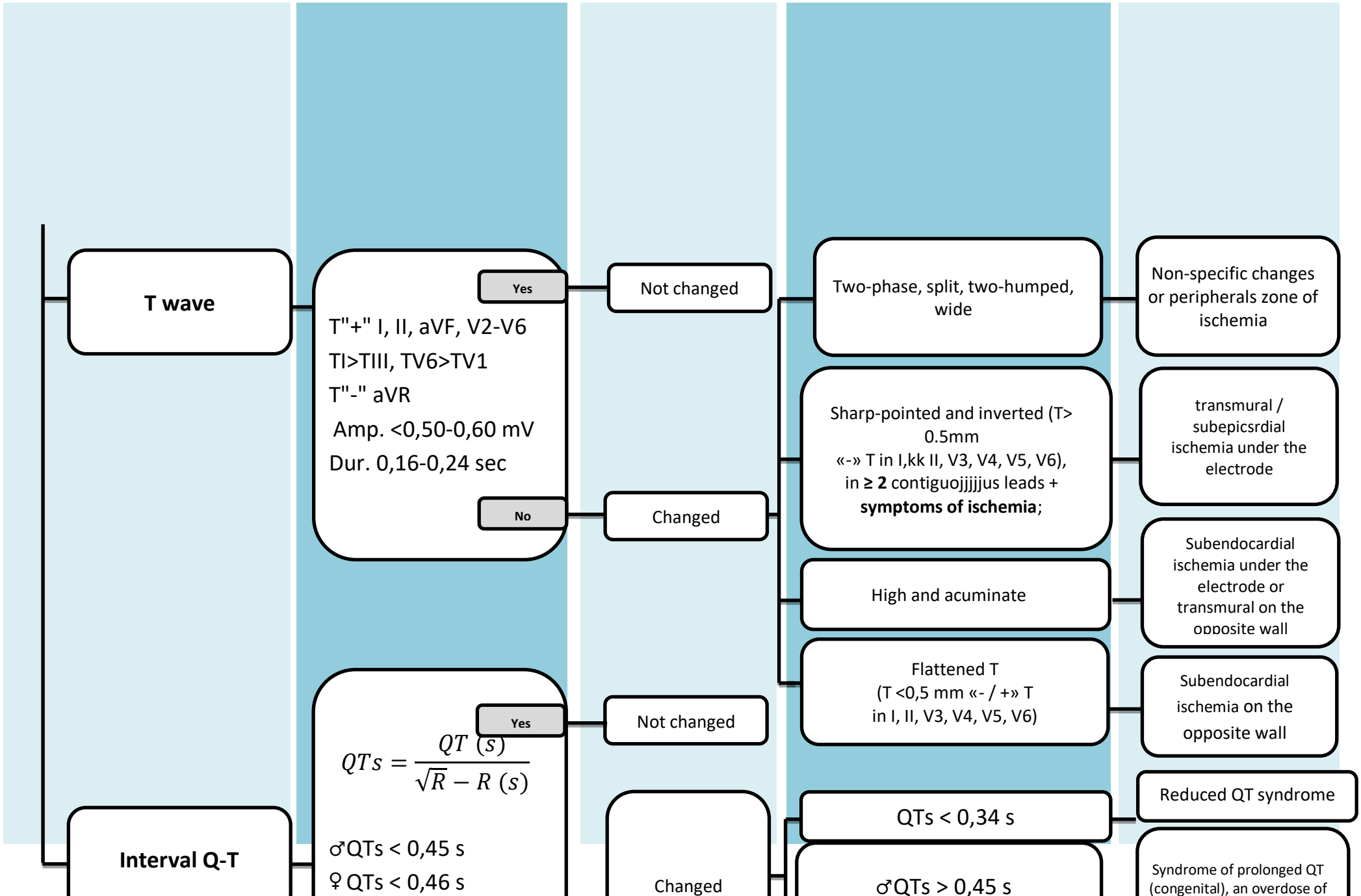
ALGORITHM OF THE ECG DECODING



ALGORITHM OF THE ECG DECODING



ALGORITHM OF ECG DECODING



Notes. ACS – acute coronary syndrome; LVH – left ventricular hyperthrophy; LBBB – left bundle branch block; RBBB – right bundle branch block; PE – pulmonary embolism; LAH – left anterior hemiblock; LPH – left posterior hemiblock.

ALGORITHM OF BLOOD PRESSURE (BP) MEASUREMENT

Blood pressure measurement on the upper extremities

The position of the patient

- Lying and standing, sitting in a comfortable position. The shoulder is located at the heart level.

Conditions

- The state of rest at least 5 minutes, do not smoke or drink coffee during 30 minutes before the examination. If the examination was preceded by a significant physical loading, the rest period should be prolonged to 15 minutes.

Equipment

- Mercury tonometer is recommended. Other devices require regular calibration. Standard cuff size (12-13 cm wide and 35 cm long) should cover at least 80% of the arm circumference and 2/3 of its length. Using too narrow and short cuff leads to higher blood pressure indicators, too wide - to their underestimation. In patients with muscular and thick arms the cuff length of 42 cm should be used; in children under 5 years - 12cm.

Frequency rate of measurement

- To perform at least twice with intervals of 1 minute. The measurement is carried out repeatedly after a few minutes when the difference of results is more than 5 mm Hg. The final result is considered to be the average value of the last two measurements.

Measurement technology

- The cuff is placed in the middle of the shoulder and at the level of the heart. Its lower edge is 2-2,5 cm above the cubital fossa. Between the cuff and the shoulder surface a finger must pass. On the first measurement of blood pressure it is recommended firstly to determine SBP by palpatory method. For this, it is necessary to determine the pulse on the a.radialis and then quickly pump up the cuff to 70 mm Hg. Further it is necessary to pump by 10 mmHg to the value, when the pulsation on a.radialis disappears. Then start to release air from the cuff. The rate at which pulsation on a.radialis appears during air discharging corresponds to the SBP. Such palpation method of determining blood pressure helps to avoid errors associated with "auscultatory flop" - the disappearance of Korotkoff sounds immediately after their first appearance. Measurement of blood pressure by auscultatory method: the air is pumped by 20-30 mmHg above the SBP values which were determined by palpation. The air is deflated slowly - 2 mm per second and the phase I of Korotkoff sounds (appearance) and phase V (disappearance) is determined, which correspond to the SBP and DBP. On auscultation of Korotkoff tones at very low values or up to 0, DSB level is considered to be the level of BP, which is fixed at the onset of phase V.
- **NB! Blood pressure is measured with an accuracy of 2 mmHg.**

Specific recommendations

- Measurement of blood pressure on the first and fifth minutes after transition in orthostasis is mandatory in elderly patients, patients with diabetes mellitus and in all cases of orthostatic hypotension or when it is suspected.

Blood pressure measurement on the lower extremities

(peculiarities and differences)

The position of the patient	• Procumbent position (lying face down).
Conditions	• The state of rest during 5 minutes; it is conducted after BP measurement on the upper extremities.
Equipment	• Large size of the cuff (42 cm in length)
Frequency rate of measurement	• Twice, with an interval of 1 minute
Measurement technology	• The cuff is placed 2-3 cm above the popliteal fossa according to the rules of investigation on the upper extremities. The membrane of the phonendoscope is placed in the popliteal fossa.
Specific recommendations	• BP measurement on the ankles to determine malleolar-brachial index of systolic blood pressure is carried out by doplerometry or pulsometry.

METHODICAL RECOMMENDATIONS ON EVALUATION OF TOTAL RISK OF CARDIOVASCULAR DISEASES

The risk of cardiovascular diseases (CVD) is the probability of cardiovascular events development in a person due to atherosclerosis within a specified period of time.

At the current stage it is proposed to use the pattern of total risk determining based on the SCORE system (Systematic Coronary Risk Evaluation – systematic evaluation of coronary risk). The system estimates a 10-years' risk of development of the first fatal event due to arteriosclerosis (myocardial infarction, stroke, aortic aneurysm, etc.). In the SCORE system the following risk factors are used: gender, age, smoking, SBP, total cholesterol or cholesterol and HDL ratio.

The total cardiovascular risk can be easily calculated using the diagram (fig. 9).

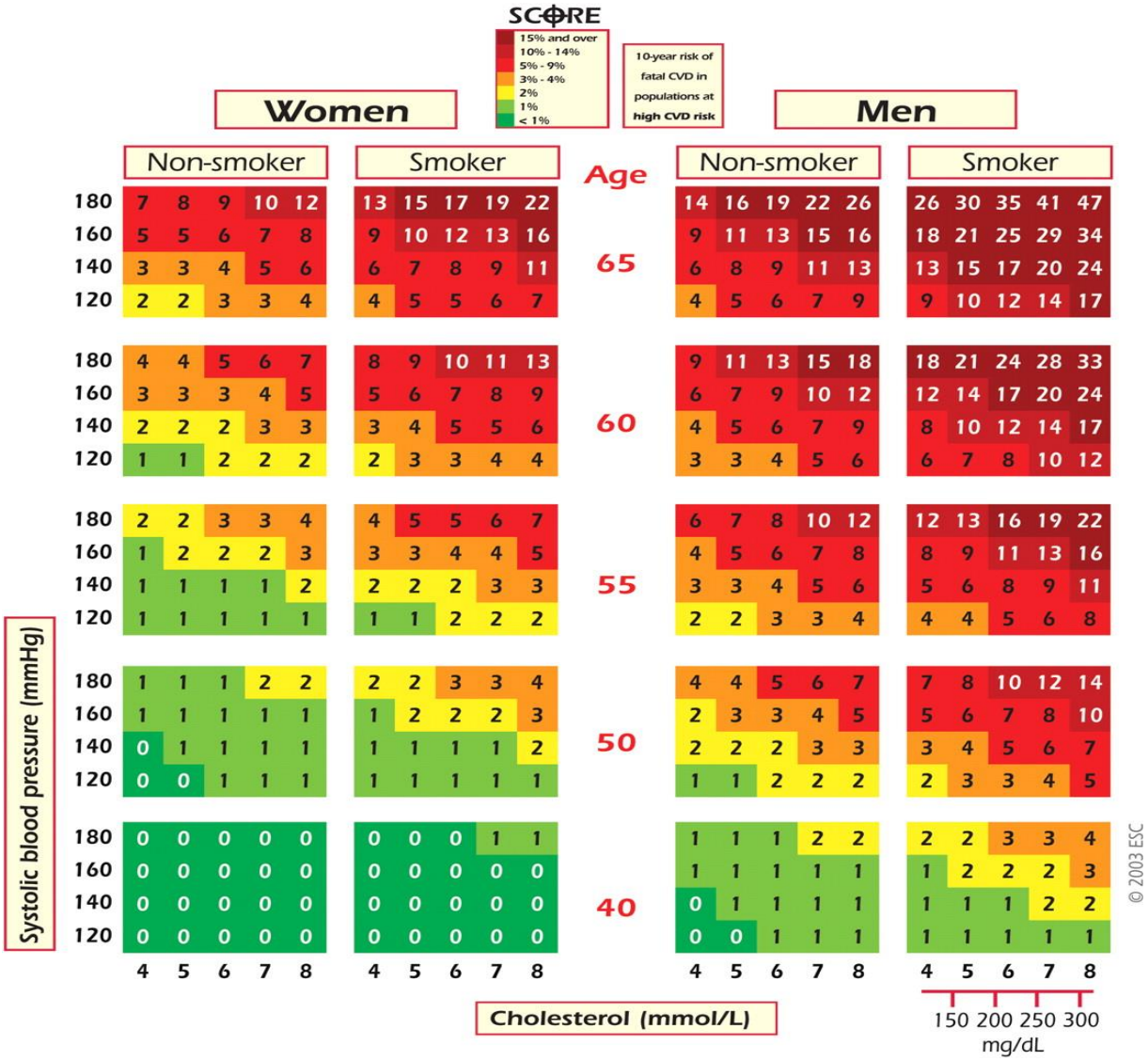


Fig. 9. 10-years' risk of fatal CVD in high-risk regions of Europe, taking into account gender, age, systolic blood pressure (SBP), total cholesterol and smoking.

Instructions for using the diagram. To assess personal 10-years' risk of cardiovascular death it is necessary to find an appropriate place in the table considering sex, age, smoking status. Find a place in the table with the closest value of SBP (mmHg) and total cholesterol (mmol/L or mg/dL).

ALGORITHM OF EVALUATION OF TOTAL RISK OF CARDIOVASCULAR DISEASES ESTIMATION

An adult person that has:
- desire to assess the overall risk, or
- one or more risk factors such as smoking, excessive body mass and hyperlipidemia, or
- positive family history of premature cardiovascular disease development or major risk factors (hyperlipidemia), or
- symptoms that indicate the presence of CVD

Asking about complaints and anamnesis taking, measurement of blood pressure, smoking status precising, determination of total cholesterol level, low density lipoprotein (LDL), high density lipoproteins (HDL) and triglycerides (TG) in serum

Evaluation of total cardiovascular risk by the **SCORE** scale **(Fig. 9)**

< 1%	**≥ 1% and < 5%**	**≥ 5% and < 10%** or Significantly elevated single risk factor, such as familial dyslipidemia and severe hypertension. Patients with diabetes mellitus type 1 and 2 without risk factors or damage of target organs. Patients with moderately severe chronic kidney disease (GFR 30-59 ml/min/1,73m2)	**≥ 10%** or The established diagnosis of CVD, MI in anamnesis, ACS, revascularization of the coronary arteries was performed, ischemic stroke, peripheral artery atherosclerosis. Patients with diabetes mellitus type 2 and type 1 with damage of target organs (microalbuminuria, etc.). Patients with severe chronic kidney disease (GFR <30 ml/min/1,73m2)
Low risk	**Moderate risk**	**High risk**	**Very high risk**
Recommendations on lifestyle modifications, dynamic monitoring	Recommendations on lifestyle modifications, re-evaluation by the SCORE scale and determination of serum lipids after 3 months	Recommendations on lifestyle modifications, drug therapy to achieve target levels of LDL	Recommendations on lifestyle modifications, drug therapy to achieve target levels of LDL
	LDL <3,0 mmol/L	**LDL <2,5 mmol/l**	**LDL <1,8 mmol/L, and / or reduction of ≥50% if it is impossible to achieve the target level**

DEFINING AND EVALUATION OF BODY MASS INDEX

Body mass index (*body mass index (BMI)* – value which allows to evaluate the degree of conformity of body mass and height of a human, and thereby to evaluate if body mass is deficient, normal or excessive. This index is important for determining indications of necessity of treatment.
Defining body mass index by Kettler was done by the formula:

$$BMI\ (kg/m^2) = body\ mass\ (kg)/height^2\ (m)$$

Normal BMI is considered to be within the limits of 18,5–24,9 kg/m^2, deficit – < 18,5 kg/m^2, excessive body mass – 25–29,9 kg/m^2, obesity I degree – 30–34,9 kg/m^2, obesity II degree – 35–39,9 kg/m^2, obesity III degree – > 40 kg/m^2 [166].
BMT is necessary to evaluate in patients with cardiovascular diseases, diseases of endocrine glands, as well as in patients with community-acquired pneumonia. In community-acquired pneumonia both overweight/obesity and deficit body mass is an unfavorable factor of the disease course.

GUIDELINES ON INTERPRETATION OF EXTERNAL RESPIRATION STUDY

The study of external respiration function (ERF) is performed by spirometry with graphic representation of "flow-volume" loop. Spirometry is a method of determining ERF by measuring the amount of air that the patient can expire from the lungs with maximal effort after maximal deep inspiration.

Indications for spirometry are:

1. Determining presence and type of ventilation disorders.
2. Determining reversibility of violations in the existing bronchial obstruction.
3. Determining the impact of therapy
4. Monitoring of ventilation disorders in dynamics.

There are no absolute contraindications for spirometry!

Relative contraindications for spirometry are:

1. Acute coronary syndrome at the time of the study or suffered less than 6 months ago.
2. Violation of the heart rate.
3. Mental disorders.
4. Severe cardiac and respiratory failure (presence of dyspnea at rest).
5. Hyperthermia.
6. Expiratory apnea.
7. Pulmonary tuberculosis.
8. Pneumothorax

Rules of patient's preparation for spirometry

1. Spirometry is performed in clinically stable condition of a patient without concomitant infection of the respiratory tract.
2. Short-acting bronchodilators are not used for at least 4 hours, prolonged – 12 hours, methylxanthines of prolonged action – 24 hours prior to the study.
3. The patient should avoid smoking for at least 1 hour prior to the study.
4. The study should not be conducted strictly on an empty stomach, but patients should refrain from eating large quantities of food and/or beverages.

Key parameters of spirometry:

Vital capacity (VC) – the amount of air at the maximal inspiration and expiration (measured in liters).

Forced vital capacity (FVC) – vital capacity, which is determined during a forced breathing maneuver (measured in liters).

FEV1 – forced expiratory volume in the first second with the maximal expiratory flow.

FEV1/FVC – FEV1 and FVC ratio, which is designated as a fraction (previously was designated in percentages).

Regulatory parameters are determined for every patient individually, taking into account gender, height, age and race.

Evaluation of the spirometry data is done by relative parameters that reflect the ratio of patient's absolute values, obtained in the course of investigation to appropriate (predicted).

RF values

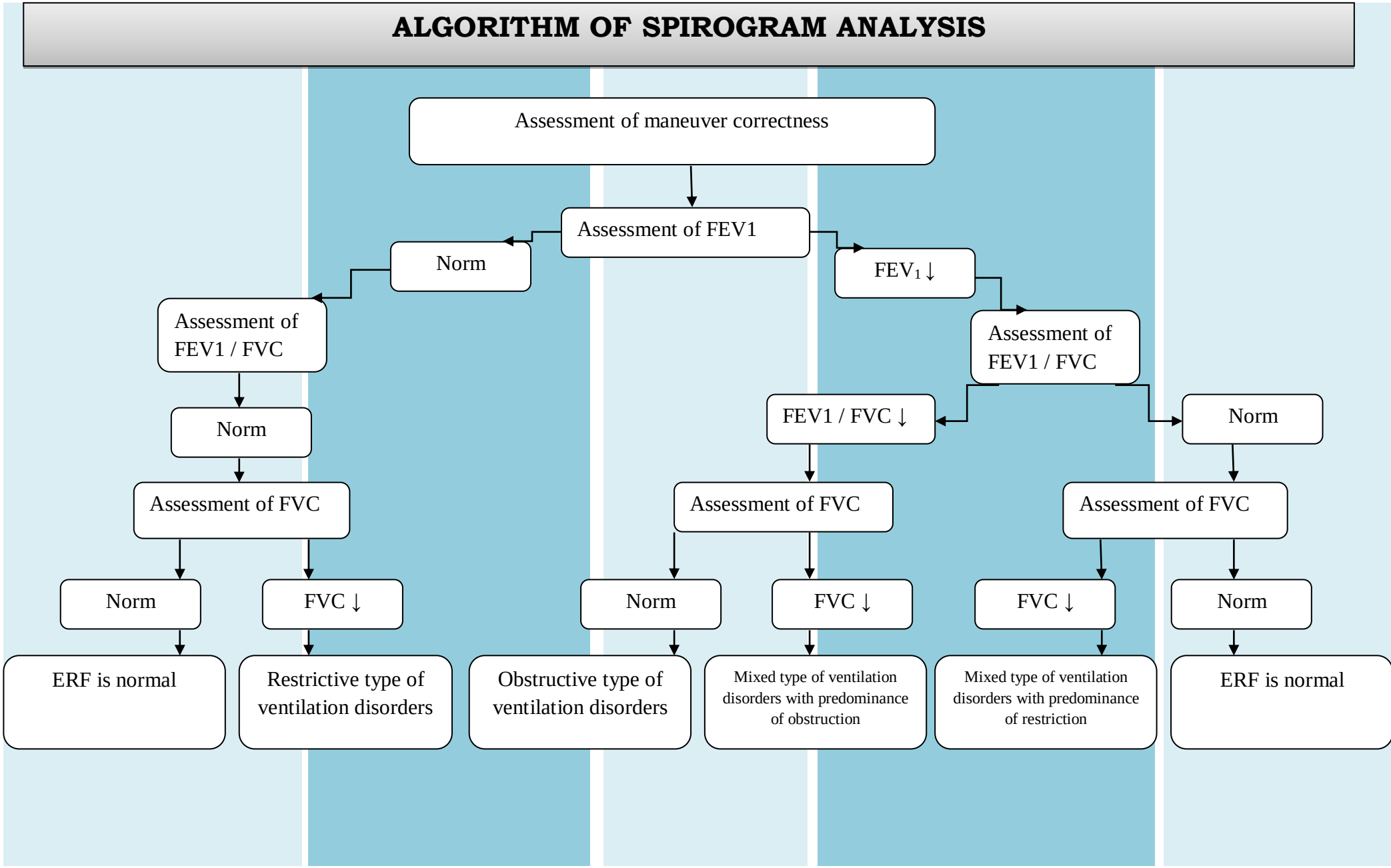
Parameters,	Norm	Ventilation violations		
		moderate	significant	severe
VC, % from predicted	≥ 80	79-60	59-51	<50
$FEV_{1,}$ % from predicted	≥ 80	79-60	59-41	<40
FEV_1/FVC	0,7-0,8	not taken into account in determining severity of ventilation violations *		

*Note: *- important in determining type of ventilation disorders.*

Examples of conclusions of ERF study:

1. Respiratory function is normal: all parameters are normal
2. Ventilation disorders (moderate, significant or severe) according to obstructive type: in conditions of FEV1 decrease and/or FEV1/FVC less than 0.7).
3. Ventilation disorders (moderate, significant or severe) according to restrictive type: in conditions of VC or FVC decrease (if VC parameter is not available).
4. Ventilation disorders (moderate, significant or severe) according to mixed type. *The degree of severity depends on the parameter (VC or FEV1), which is decreased to a large extent.*
5. Ventilation disorders (moderate, significant or severe) according to mixed type with predominance of obstruction: in conditions when FEV1/FVC is less than 0,7.
6. Ventilation disorders (moderate, significant or severe) according to mixed type with predominance of restriction: in conditions when FEV1/FVC is more than 0,9.

ALGORITHM OF SPIROGRAM ANALYSIS



Examples of study protocols

Example 1

Parameter	Predicted, l	Actual, l	Relative index, %
FVC	5,81	6,87	118,3
FEV_1	4,65	5,70	122,4
FEV_1/ FVC		82,8 (0,8)	

Conclusion: ERF is normal

Example 2

Parameter	Predicted, l	Actual, l	Relative index, %
FVC	2,84	1,73	60,8
FEV_1	2,42	1,56	64,4
FEV_1/ FVC		90,4 (0,9)	

Conclusion: moderate ventilation disorders by mixed type.

Example 3

Parameter	Predicted, l	Actual, l	Relative index, %
FVC	3,76	3,30	87,7
FEV_1	2,79	1,03	36,8
FEV_1/ FVC		31,2 (0,3)	

Conclusion: severe obstructive ventilation disorders by obstructive type.

Example 4

Parameter	Predicted, l	Actual, l	The relative index, %
FVC	3,76	2,80	74,46
FEV_1	2,79	1,03	36,8
FEV_1/ FVC		31,2 (0,3)	

Conclusion: severe ventilation disorders by mixed type with predominance of obstruction.

Bronchodilator reversibility test

In case of obstructive ventilation insufficiency by obstructive type, bronchodilator reversibility test is conducted using bronchodilator drugs (e.g. salbutamol 400 mcg).

Bronchodilator reversibility test is performed in 15 minutes after drug using.

Evaluation of the results: increase of FEV1 more than by 200 ml or 12% as compared to its basic value points to reversibility of bronchial obstruction (positive test), if it is less than 200 ml or 12% - negative one.

Percentage of rise of FEV1 parameter is calculated by the formula:

EV1(after test) – FEV1(before test)/ FEV1(before test) x 100%

Examples of study protocols (continuation)

Example 5

Parameter	Predicted, l	Actual pre test, l	Relative index, %	Actual post test, l
FVC	3,03	2,44	80,4	2,67
FEV_1	2,62	1,62	61,7	1,66
FEV_1/ FVC		66,3 (0,6)		61,95 (0,6)

((FEV_1 post) – FEV_1 pre)/ FEV_1 pre))×100 % = ((2,67–2,44)/2,44)×100 %=9,42%. FEV1 reversibility less than 200 ml.

Conclusion: moderate obstructive ventilation disorders. Test on reversibility of airflow obstruction is negative.

Example 6

Parameter	Predicted, l	Actual pre test, l	Relative index, %	Actual post test, l
FVC	2,38	2,39	100,3	2,76
FEV_1	2,40	1,01	50,6	1,23
FEV_1/ FVC		42,26 (0,4)		44,46 (0,4)

((FEV_1 post) – FEV_1 pre)/ FEV_1 pre))×100 % = (1,23–1,01)/1,01×100 %=21,78 %. FEV_1 reversibility more than 200 ml.

Conclusion: moderate obstructive ventilation disorders. Test on reversibility of airflow obstruction is positive.

.

Algorithm of performance and analysis of peak flowmetry results

Peak flowmetry is a method of functional diagnostics to determine the volumetric rate of peak expiratory forced rate (PEFR). PEFR is the main index which correlates with bronchial obstruction – forced expiratory volume in the first second (FEV1), so it can be recommended as its analogue for the use in the outpatient conditions and for individual use by the patient primarily.

Aim of the study are:

• revealing of bronchial obstruction as a screening method (used only when spirometry is inaccessible!);

• monitoring of bronchial obstruction with possible prediction of exacerbations;

• monitoring of treatment effectiveness in patients with bronchial-obstructive pathology.

An indicator, calculated for the population considering sex, age and height of the patient can be considered as the conventional norm. An indicator, which is calculated for the general population does not always correspond to normal one for a particular patient. In this case it is optimally to consider average PEFR, measured in remission period when the patient is in the best state of health as the normal one.

As a standard, it is necessary to accept measuring amplitude of fluctuations of PEFR (difference between morning value before bronchodilator usage, if a patient uses it, and evening value), which is expressed as percentage and calculated by the formula:

$$\text{Daily fluctuation} = \frac{\text{evening PEFR} - \text{morning PEFR}}{\frac{1}{2}(\text{evening PEFR} - \text{morning PEFR})} \times 100$$

Value of amplitude of fluctuations in daily measuring of PEFR is a reliable index of stability and/or disease severity. Value of amplitude of fluctuations more than 20% is considered to be significant:

PEFR less than 20% - stable state

PEFR more than 20% - exacerbation, increase of hyperactivity

For patients with bronchial asthma the system of signal zones under the principle of traffic lights is used:

Green Zone - the best value of the patient is multiplied by 0,8. PEFR value more than the obtained result is within the green zone – zone of a stable state, when value of amplitude of daily fluctuations is less than 20%.

Yellow Zone – values between the green zone and the value, obtained in multiplying the best patient's PEFR value by 0,6. This zone corresponds to non-severe exacerbation, when amplitude of daily fluctuations makes up 20-30%.

Red Zone – patient's PEFR value is below the yellow zone, daily fluctuations exceed 30%, this means exacerbation.

В методичке этой таблицы нет. Проверить не могу.***Table 1***

Peak flow readings according to the American Lung Association

Zone	Reading	Description
Green Zone	80 to 100 percent of the usual or normal peak flow readings are clear.	A peak flow reading in the green zone indicates that the asthma is under good control.
Yellow Zone	50 to 79 percent of the usual or normal peak flow readings	Indicates caution. It may mean that respiratory airways are narrowing and additional medication may be required.
Red Zone	Less than 50 percent of the usual or normal peak flow readings	Indicates a medical emergency. Severe airway narrowing may be occurring and immediate action needs to be taken. This would usually

		involve contacting a doctor or hospital.

Algorithm of performance and analysis of peak flowmetry results

Patient's position

- study may be conducted both in standing and sitting position of a patient, but the position chosen should be constant during peak flowmetry performance;

Circumstance

- in the morning after sleep, or when the patient wakes up because of asthma attack, in the evening before going to bed;

Equipment

- mechanical peak flowmeter (a plastic tube or box with a graduated scale) or electronic one; when using mechanic one, diagram should be drawn, based on two-dimensional scale, where the X axis represents time of the day and the day, and the axis Y - PEFR values in L/min. Electronic peak flowmeter stores data in its own memory devices and needs transferring them to a computer for further analysis;

Multiplicity of measurement

- PEFR is studied twice a day, in the morning and in the evening, the maneuver is repeated three times with small intervals, maximal value is recorded and written down in the diagram;

Measuring technique

- peak flowmeter must be placed strictly horizontally, it is neded to avoid contact with a graduated scale to prevent mechanical dislocation of indicating hand (when using a mechanical peakflow meter);
- in the beginning of measurement the indicating hand is set to zero (in electronic device - display is set to "zero"), a patient performs a maximal quiet breath, then tightly clasps mouthpiece with lips and performs a forced exhalation;

Specific recommendations

- standards for healthy persons of different age groups, patients of different height and gender are evaluated according to calculation tables (see below), but the maximal value for patients is measured in remission period and/or control of the disease.

Table 2

MARGINAL PEFR VALUES IN MEN

AGE (YEARS) HEIGHT (SM)	5	8	11	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85
100	39	39	39															
105	65	65	65															
110	92	92	92															
115	118	118	118															
120	145	145	145															
125	171	171	171															
130	197	197	197															
135	224	224	224															
140	250	250	250	414	456	481	494	499	497	491	480	467	452	436	418	400	381	362
145	276	276	276	423	466	491	504	509	508	501	491	477	462	445	427	408	389	370
150	303	303	303	432	475	501	514	519	518	511	500	487	471	454	436	417	397	378
155	329	329	329	440	484	510	524	529	527	520	510	496	480	463	444	425	405	385
160	356	356	356	448	492	519	533	538	536	530	519	505	489	471	452	432	412	392
165	382	382	382	456	500	527	542	547	545	538	527	513	497	479	460	440	419	399
170	408	408	408	463	508	535	550	555	554	546	535	521	504	486	467	447	426	405
175	435	435	435	469	515	543	558	563	561	554	543	528	512	493	474	453	432	411
180	PEFR in children before 11 years of age depends on height only			476	522	551	566	571	569	562	550	536	519	500	480	459	438	417
185				482	529	558	573	578	576	569	557	543	525	506	486	465	444	422
190				488	536	564	580	585	583	576	564	549	532	513	492	471	450	428

Table 3

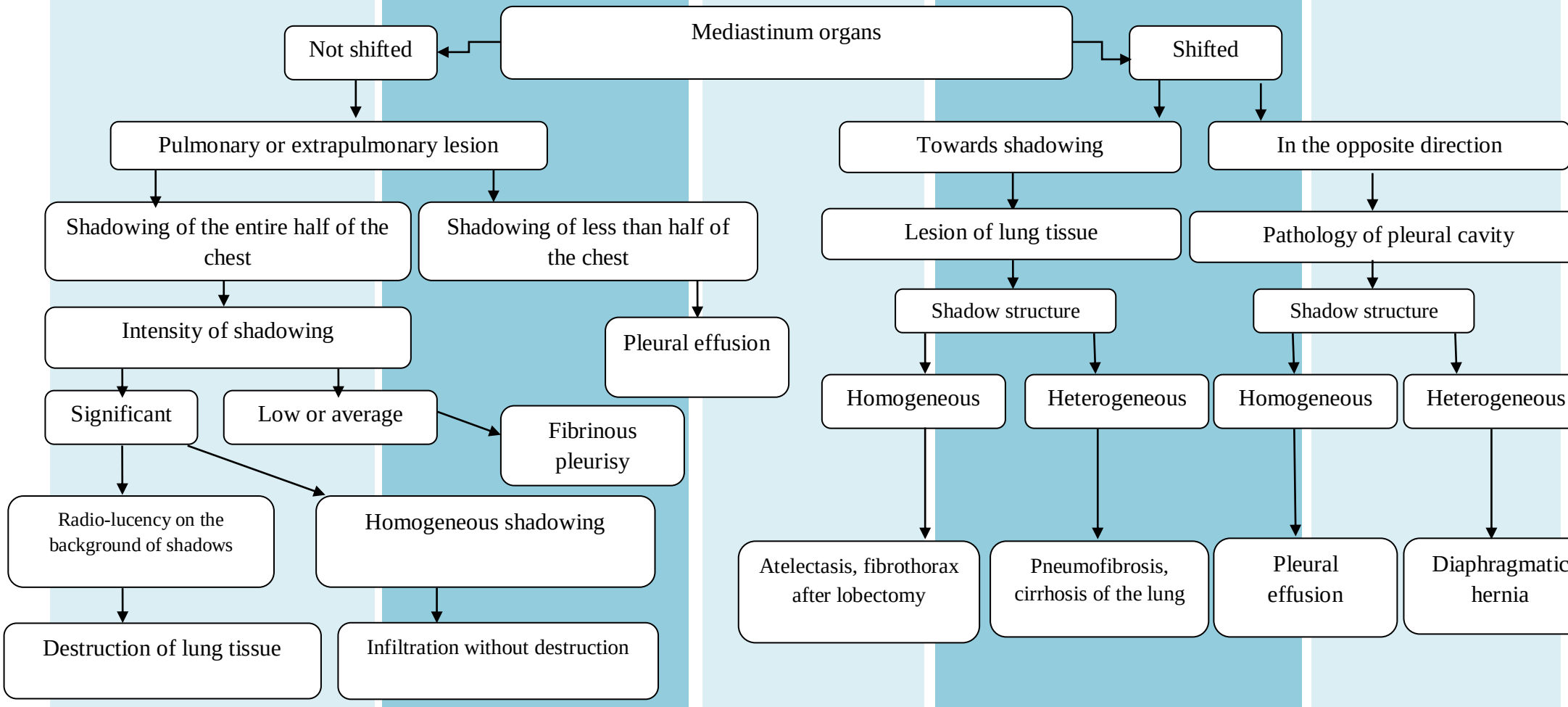
MARGINAL PEFR VALUES IN WOMEN

AGE (YEARS) / HEIGHT (SM)	5	8	11	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85
100	24	24	24															
105	51	51	51															
110	77	77	77															
115	104	104	104															
120	130	130	130															
125	156	156	156															
130	183	183	183															
135	209	209	209															
140	236	236	236	348	369	380	384	383	379	371	362	352	340	328	316	302	289	276
145	262	262	262	355	376	387	391	390	385	378	369	358	347	334	321	308	294	281
150	289	289	289	360	382	393	397	396	391	384	375	364	352	340	327	313	300	286
155	315	315	315	366	388	399	403	402	397	390	381	370	358	345	332	318	304	290
160	342	342	342	371	393	405	409	408	403	396	386	375	363	350	337	323	309	295
165	368	368	368	376	398	410	414	413	408	401	391	380	368	355	341	327	313	299
170	394	394	394	381	403	415	419	418	413	406	396	385	372	359	346	331	317	303
175	421	421	421	385	408	420	424	423	418	411	401	389	377	364	350	335	321	307
180	PEFR in children before 11 years of age depends on height only			390	413	390	413	425	429	428	423	415	405	394	381	368	354	339
185				394	417	394	417	429	433	432	427	419	409	398	385	372	358	343
190				398	421	398	421	433	438	436	432	424	414	402	389	375	361	347

GUIDELINES ON CHEST X-RAY INTERPRETATION

Image projection	• frontal or back frontal, right or left lateral, frontal or back oblique: the first or the second.
Special conditions	• in sitting or lying position due to severity of patient's state; with respiratory dynamic blurrness of image due to patient's unconsciousness, etc.
Image quality	• physical and technical characteristics: optical density, contrast, acutance; lack of artifacts and veil
Soft tissues of the chest	• volume, structure, presence of foreign bodies or free gas after injuries, etc.
Chest skeleton and shoulder girdle	• position, shape, size and structure of bones, ribs, sternum, visible cervical and thoracic vertebrae, clavicles, shoulder blades, heads of shoulder bones; state of ossification center and areas of growth in children and young people
Lung fields	• area, shape, transparency; • in detecting symptoms of pathology (extensive or limited shadowing or radio-lucency of the focus - round or ring-like shadow) - detailed description of their position, shape, sizes, shadow density, structure, contours; • lung pattern, distribution of elements, architectonics, caliber, character of contours.
Lung roots	• position, shape, size, structure, contours of elements, presence of additional structures.
Mediastinum	• position, shape and width as a whole and characteristics of separate organs.
Conclusion	• X-ray (clinical and radiological) conclusion; • recommendations.

ALGORITHM OF CHEST RADIOGRAPHS ANALYSIS



ALGORITHM OF DETERMINING SEVERITY OF STATE OF PATIENTS WITH CAP

According to modern principles of management of adult patients with CAP, a great number of them should be safely treated in out-patient units. Outpatients with CAP treated with oral antibiotics return to work or usual activity level on average 6-9 days earlier than those admitted to the hospital with equivalent severity-of-illness. Hospital admission increases CAP treatment costs nearly by 20 times, as compared to outpatient therapy.

Recently, two separate risk stratification instruments, CURB-65 and Pneumonia Severity Index (PSI) have been developed.

CURB-65 is a simple scoring system easily used in the outpatient office or emergency room, it assigns 1 point for each of 5 clinical finding (table 4).

In 2006 Australian-American team of experts proposed scale SMART-COP (table 8) to determine patients' requirements in respiratory support and vasopressors. Also it is possible to use its variant, SMRT-CO (without specifying levels of albumin and pH of arterial blood).

Table 4

CURB-65/CRB-65

Clinical Finding	Points
C – Confusion	1
U – Blood urea nitrogen ≥ 20 mg/dL	1
R – Respiratory rate ≥ 30 breaths/min	1
B – Systolic BP < 90 mm Hg or Diastolic BP ≤ 60 mm Hg	1
Age ≥ 65 years	1

CURB-65 ALGORITHM

0 or 1 – home treatment

2 points – short-term hospitalization or day-patient treatment

3 points – hospitalization

4-5 points – hospitalization and follow-up in ICU

It is important to emphasize that clinical judgment should be the primary factor while deciding on hospital admission, with the CURB-65 score providing assistance to this decision making (table 4).

Table 5

CURB-65 ALGORITHM

Total Score	**Mortality %**	**Risk Level**	**Suggested Site-of-Care**
0	0.6	Low	Outpatient
1	2.7	Low	Outpatient
2	6.8	Moderate	Short inpatient / supervised outpatient
3	14.0	Moderate to High	Inpatient
4 or 5	27.8	High	Inpatient / ICU

Pneumonia Severity Index (table 6, 7), or PSI, is a validated risk stratification instrument which can help in identifying CAP patients who can safely be treated with antibiotics in outpatient conditions. Point values are given for a variety of clinical and laboratory parameters. The PSI involves calculating a score, which places a given patient into one of 5 risk classes. Classes I, II, and III are at low risk for death, and may be considered for outpatient treatment. Risk classes IV and V patients should usually be hospitalized.

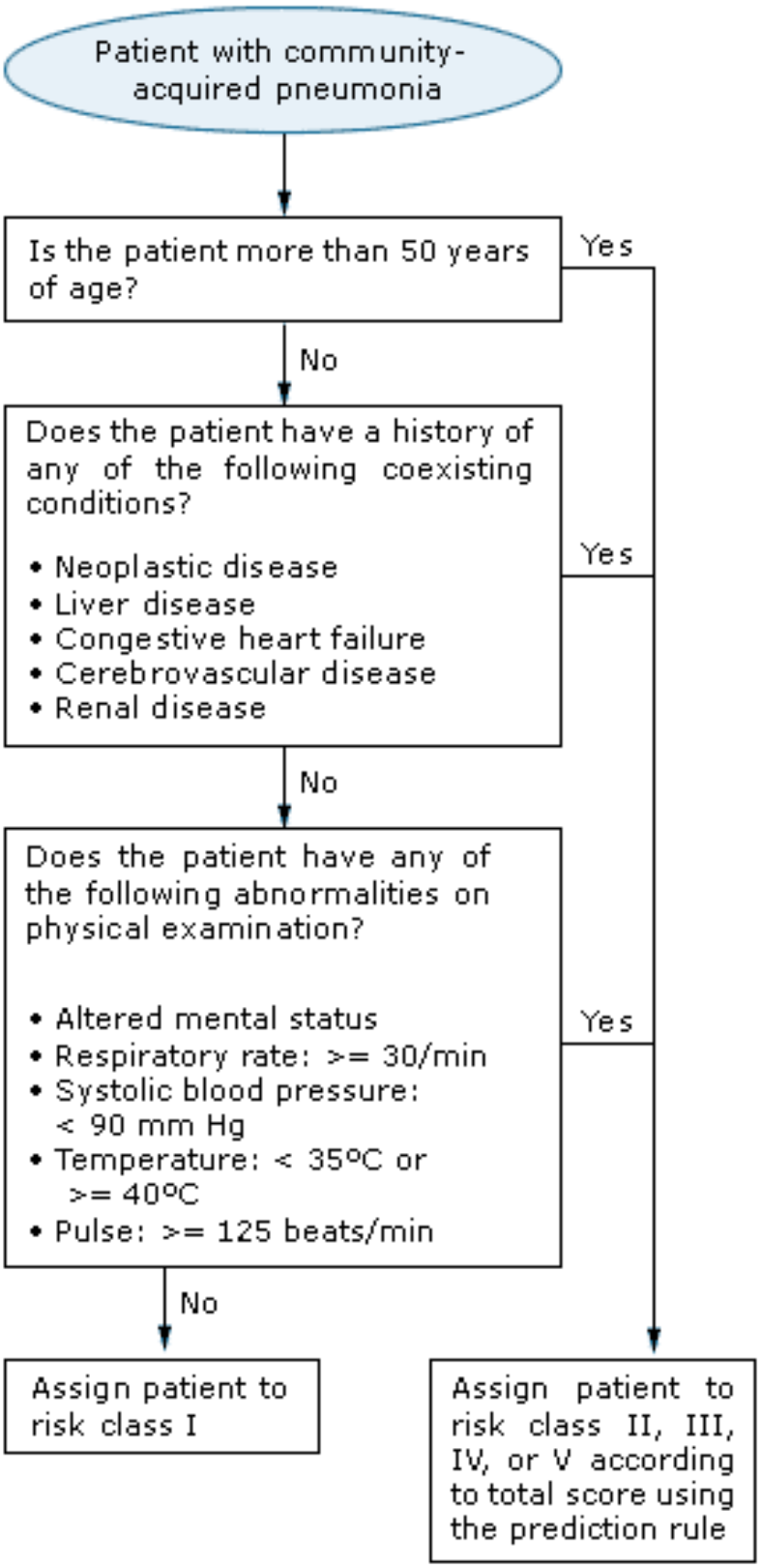

Patient with community-acquired pneumonia
Is the patient more than 50 years of age?
Yes
No
Does the patient have a history of any of the following coexisting conditions?
• Neoplastic disease
• Liver disease
• Congestive heart failure
• Cerebrovascular disease
• Renal disease
Yes
No
Does the patient have any of the following abnormalities on physical examination?
• Altered mental status
• Respiratory rate: >= 30/min
• Systolic blood pressure: < 90 mm Hg
• Temperature: < 35°C or >= 40°C
• Pulse: >= 125 beats/min
Yes
No
Assign patient to risk class I
Assign patient to risk class II, III, IV, or V according to total score using the prediction rule

Table 6

Scale PORT and PSI index

Demographic factors	
Age	
Men	Age (in years)
Women	Age (in years) – 10
Nursing home resident	+10
Concomitant diseases	
Neoplastic diseases in anamnesis	+30
Liver diseases	+20
Congestive heart failure	+10
Cerebrovascular diseases	+10
Renal diseases	+10
Findings on physical examination	
Altered mental status	+20
Respiratory rate ≥ 30/min	+20
Systolic blood pressure <90 mm Hg	+20
Temperature < 35ºC or ≥ 40ºC	+15
Pulse ≥ 125 beats/min	+10
Laboratory and radiographic findings	
Arterial pH <7.35	+30
Blood urea nitrogen >= 30/mg/dl (11 mmol/lr)	+20
Sodium < 130 mmol/l	+20
Glucose >= 250 mg/dl (14 mmol/l)	+10
Hematocrit <30%	+10
Partial pressure of arterial oxygen < 60 mm Hg or oxygen saturation < 90%	+10
Pleural effusion	+10
PORT (index PSI)	Total

Table 7

Stratification of Risk Score

Risk	Class PSI	Points	Mortality	Treatment
Low	I	0-50	0.1%	Outpatient treatment
Low	II	51-70	0.6%	Outpatient treatment
Low	III	71-90	0.9%	Short-term hospitalization
Moderate	IV	91-130	9.3%	Hospital admission
High	V	>131	27.0%	Hospital admission, ICU

Identifying patients at high-risk for CAP mortality (who will benefit from intensive care unit admission):

CURB-65 and PSI have relatively poor predictive value for identifying patients who will deteriorate after admission, and subsequently require transfer to an intensive care unit (ICU).

Infectious Diseases Society of America (IDSA) and American Thoracic Society (ATS) have recently developed criteria to assist with the decision concerning ICU admission. It is important to emphasize that these criteria are meant for guidance only, and still require prospective validation before they can be recommended for routine use.

Major Criteria (1 or more = ICU admit)

- Endotracheal intubation and mechanical ventilation
- Shock, requiring vasopressors

Minor Criteria (3 or more = ICU admit)

- Respiratory rate > or = 30 min-1
- PaO2-to-FiO2 ratio < or = 250
- Multilobar infiltrates
- Confusion or delirium
- Blood urea nitrogen (BUN) > or = 20 mg/dL
- Leukopenia (WBC count < 4000 cells/mm3)
- Thrombocytopenia (platelet count < 100,000 cells/mm3)
- Hypothermia (core temperature < 36oC)
- Hypotension requiring aggressive fluid resuscitation

Note: These criteria await prospective validation. Physician's judgment should continue to be the primary determinant for patient admission to an intensive care unit.

Table 8

SMART-COP

Parameters	Points
S – systolic BP less than 90 mm Hg	2
M – multilobar chest X-ray involvement	1
A – albumin less than 35 g/L	1
R – respiratory rate 25 r/min or more	1
T – tachycardia 125 bpm or more	1
C – confusion (acute)	1
O – oxygen low PaO2 less than 70 mm Hg, or O2 saturation 93% or less, or PaO2 /FiO2 less than 333	2
P – pH less than 7.35	2
Total points score (maximum 11)	

Interpretation of SMART-COP score

Points	Risk of need for intensive respiratory or vasopressor support (IRVS)
	low risk of need for intensive respiratory or vasopressor support (IRVS)
3–4	moderate risk (1 in 8) of need for IRVS
5–6	high risk (1 in 3) of need for IRVS
> 7	very high risk (2 in 3) of need for IRVS

Severe CAP = a SMART-COP score of 5 or more points.

Interpretation of SMRT-CO score

Points	Risk of need for intensive respiratory or vasopressor support (IRVS)
0	Very low risk, do not need hospitalization
1	Low risk (1 з 20), do not need hospitalization
2	Moderate risk (1 з 10), in-patient hospitalization
3	High risk (1 з 6), hospitalization in intensive care
>4	High risk (1 з 3), hospitalization in intensive care

Applying of these prognostic scales for evaluation of patients with CAP is definitely useful, as it allows to reduce the frequency of inappropriate hospitalization of patients with a low risk of unfavorable prognosis and select the category of patients who need intensive care. However, their use has some difficulties: first, they help to assess the severity of the patient and/or prognosis for a specific period of time, but they do not take into account the variability of the clinical picture of CAP and quick progression of the disease. Prognostic scales do not include such factors as decompensation of concomitant chronic diseases that very often are a major cause of hospitalization of patients, as well as non-medical indications for hospitalization. Therefore, any of prognostic scales may serve as a landmark only in choosing the place of treatment and in every case doctor should decide patient-specific.

COMPLETE BLOOD COUNT (CBC) INTERPRETATION ALGORITHM

Hemoglobin (m – 130–160 g/l, f – 120–140 g/l)	Increased hemoglobin level appears in: primary and secondary erythremias, dehydration, burns, diarrhea, use of diuretics.
	Reduction in hemoglobin is observed in: anemia, acute bleeding, hydration, pregnancy.
Red blood cells (m – 4,0–5,5 T/l, f – 3,9–4,7 g/l)	Increased level of red blood cells occurs in: primary erythremia, congenital heart defects, lung disease, stay in the highlands, renal polycystic, edema of renal pelvis, neoplasms (hemangioblastoma, hepatoma, pheochromocytoma), impact of corticosteroids, Cushing's disease and syndrome, treatment with steroids .
	Reduction in: blood loss, anemia, pregnancy, rapid destruction of red blood cells, hydration, reduction in intensity of red blood cells formation in the bone marrow.
The morphology of red blood cells	Re-sizing – **anisocytosis**; reshaping– **poikilocytosis**; color change – anisochromia (normochromia, hyperchromia, normochromia).
Hematocrit (m – 40–52 %, f – 36–42%)	Increased level in: erythrocytosis, polycythemia, congenital heart defects, lung diseases, stay in the highlands, kidney polycystic, tumors, which are accompanied by increased production of erythropoietin.
	Reduction of level in: hyperproteinemia, anemia, pregnancy, rapid destruction of red blood cells, hydration, reduction in intensity of red blood cells formation in the bone marrow.
Color index (0,86–1,05)	Rise of level more than 1.1 is observed in anemia (lack of vitamin B12, folic acid, cancer, intestinal polyps).
	Reduction of level less than 0.5-0.7 is observed in: iron deficiency anemia, anemia due to lead toxicity, anemia in

Reticulocytes (0,2–1,2 %)	Increase is registered in: blood loss, hemolytic anemia, on the background of treatment of Addison-Byrmer's anemia with vitamin B12, effective treatment of anemia.
	Reduction is observed in: hypoplastic anemia, recurrence of Addison-Byrmer's anemia, radiation sickness, use of cytostatic drugs.
Erythrocyte sedimentation rate (m – 1–10 mm/h, f – 2–15 mm/h)	Acceleration of ESR is revealed in: infectious and inflammatory diseases (acute and chronic infections, pneumonia, rheumatism, myocardial infarction, syphilis, tuberculosis, sepsis), collagenic diseases (rheumatism, rheumatoid arthritis), liver damage, kidney disease, diabetes, thyrotoxicosis, anemia, Hodgkin's disease, multiple myeloma, pregnancy, postpartum period, menstruation, inflammatory conditions, trauma, fractures, surgery, anemia, poisoning (arsenic, lead), hypercholesterolemia, hyperfibrinogenemia, influence of drugs (morphine, dextran, methyldopa, vitamin D) .
	Reduction of ESR is revealed in: polycythemia, hyperbilirubinemia, increased levels of bile acids, erythremia and reactive polycythemia, hypofibrinogenemia, chronic heart failure.
Platelets (180–320 G/l)	Increased platelet count (**thrombocytosis**) is determined in: polycythemia vera, chronic myeloleukosis, blood loss, erythremia, metastases of malignant tumors in the bone marrow, after splenectomy, chronic inflammation (rheumatoid arthritis, tuberculosis, sarcoidosis, granulomatosis, colitis, enteritis), acute infections, hemolysis, microcytic hypochromic anemia.
	Reduction of platelet count (**thrombocytopenia**) is revealed in: leukemia, aplastic anemia, paroxysmal nocturnal hemoglobinuria, alcoholism, megaloblastic anemia, liver cirrhosis with splenomegaly, Gaucher's disease, idiopathic thrombocytopenic purpura, thrombocytopenia after blood transfusions, lymphoma, systemic lupus erythematosus, labor, sepsis, craniocerebral trauma, metastatic tumors, massive transfusion of blood and blood products.

Neutrophils

White blood cells (4–9 G/l)

Physiological increase in white blood cells count is observed after exercise, after meal, in premenstrual period, pregnancy (especially in the last months), after childbirth and breastfeeding, stress, after taking cold and hot baths.

Increase of white blood cells count more than 9 g/l (leukocytosis) is observed in: acute inflammatory processes, purulent processes, infectious diseases (bacterial, fungal,, except for typhoid fever and epidemic typhus, measles and influenza), malignant neoplasmas, tissue trauma, leukemia, uremia, myocardial infarction, hemorrhagic stroke, bleeding, attacks of paroxysmal tachycardia, sepsis, major burns, infectious mononucleosis, diabetic coma after splenectomy.

Physiological decrease is observed in the elderly. Decrease in white blood cells count less than 3 g/l (**leukopenia**) is in: aplasia and hypoplasia of bone marrow, damage to bone marrow with chemicals, radiation sickness, effect of ionizing radiation, hyperfunction of spleen, aleukemic leukemia, plasmacytoma, sepsis, typhoid fever, viral disease, Addison-Byrmer's disease, collagenic diseases, use of drugs (sulfonamides and certain antibiotics, NSAIDs, thyrostatics, antiepileptic drugs).

Stab neutrophiles (1–6 %)

Increased level of neutrophils (**neutrophilia**) is determined in: erythremia, malignant tumors, infections (bacterial: sepsis, septic infection, viral: herpes zoster; fungal and parasitic), traumatic tissue damage, myocardial infarction, pulmonary infarction, necrotic processes of other tissues, condition after blood loss, uremia, diabetic ketoacidosis, gout, eclampsia of pregnancy, scarlet fever.

Segmented neutrophiles (47–72 %)

Reduction of neutrophils level (**neutropenia**) is determined in: aplastic anemia, agranulocytosis, viral infections (hepatitis, measles, rubella, influenza), fungal infections, histoplasmosis, toxoplasmosis, malaria, rickettsial infections, postinfection states, chronic bacterial infections (staph- and streptococcal, tuberculosis, brucellosis).

Eosinophils (0,5–5 % or 0,02–0,3 G/l)

Increase of eosinophils count more than 0.4 g/l (**eosinophilia**, **eosinophilosis**) is registered in: bronchial asthma, allergic skin lesions, nodular periarteriitis, eosinophilic vasculitis, hay fever, helminthic invasions, eczema, after introduction of antibiotics, in patients with decreased thyroid function, rheumatism, acute leukemia, parasitic infestation.

Reduction of eosinophils level below 0.05 g/l (**eosinopenia)** is observed in: typhoid fever, dysentery, acute appendicitis, sepsis, traumas, burns, surgery, the first day of myocardial infarction, physical overload, stress

Monocytes

(3-11% or 0,09-0,6G/l

Increased number of basophils more than 0.3 g/l (**basophilic leukocytosis**) is observed in: allergic conditions, polycythemia, acute leukemia, chronic myeloproliferative syndromes, chronic myeloid leukemia and myeloma, Hodgkin's disease, hemophilia, acute inflammation of the liver, diabetes, hypothyroidism, ulcerative intestinal inflammation, treatment with estrogens, prolonged exposure to low doses of ionizing radiation.

Reduction of basophils less than 0.01 g/l (**basonepia**) is observed in: prolonged radiotherapy, acute infections, pneumonia, hyperthyroidism, stress-induced situations, in some cases of acute leukemia.

Increase of monocytes level more than 1 g/l (**monocytosis**) is typical for: tuberculosis, syphilis, protozoa infections, monocytic and myelomonocytic leukemia, infectious mononucleosis, brucellosis, endocarditis, malignant tumors, rheumatoid arthritis, systemic lupus erythematosus, period of recovery after acute conditions, surgical interventions.

Reduction of monocytes level less than 0.03 g/l is observed in: glucocorticoid treatment, infections with neutropenia.

Increase of lymphocytes level above 4 g/L (**absolute lymphocytosis**), **relative lymphocytosis** – increasing in percentage of lymphocytes. Lymphocytosis is revealed in: severe physical loadings, during menstruation period, chicken pox, measles, rubella, whooping cough, influenza, adenovirus infection, acute infectious lymphocytosis, infectious mononucleosis, cytomegalovirus infection, tuberculosis, syphilis, malaria, toxoplasmosis, diphtheria, brucellosis.

Reduction of lymphocytes level less than 1 g/l is observed in: pancytopenia, secondary immunodeficiency, Hodgkin's disease, certain liver diseases, renal failure, circulatory failure, malignant tumors, taking corticosteroids, severe viral infections.

ALGORITHM OF COMMON URINALYSIS EVALUATION

Total amount of daily urine,

1200-1500 ml

Increase amount of urine – **polyuria**: diabetes mellitus and insipidus, in

Reduce of daily urine – **oliguria**, dehydration, diarrhea, vomiting, increased edema, accumulation of fluids in the cavities.

Arrest of urine flow into the bladder – **anuria:** acute blood loss, persistent vomiting, acute nephritis, severe kidney diseases, obstruction of the ureter with calculus, compression of the ureter with tumors (cancer of the uterus, of uterine appendages, of the bladder).

Retention of urine in the bladder due to inability to natural urination – **ischuria:** adenoma and prostate cancer (in men), prostatitis, urethral stricture, obstruction due to calculus or tumor in the bladder outlet, violation of the neuromuscular apparatus of the bladder in severe infections, intoxication after surgery and childbirth, neurological diseases.

Color (light-yellow)

Increase of intensity of urine color - loss of fluid in edema, diarrhea, vomiting.

Reddish color (meat slops) - hematuria, hemoglobinuria.

Dark- yellow with greenish hue - presence of bile pigments in case of jaundice; **greenish-yellow** in obstructive jaundicee; **greenish-brown** (color of beer) in parenchymatous jaundice.

Greenish-yellow – large amount of pus in urine (pyuria).

Soot- brown - pyuria in alkaline pH.

Dark, almost black – hemoglobinuria in hemolytic anemia.

Whitish - plenty of phosphates (phosphaturia), lipids (lipuria).

Red - use of antipyrine, amiopyrin, santonin.

Pink - use of aspirin, carrots, beets.

Brown - use of phenol, cresol, lysol, uva ursi, activated carbon (carbolen).

Dark brown – use of salol, naphthol.

Transparency	Turbidity due to urates presence, disapears while heating or adding alkali. Turbidity due to phosphates presence, increases while hearting and disappears when adding acetic acid. Turbidity due to calcium oxalates presence, disappears when adding hydrochloric acid. Turbidity caused by presence of pus, does not disappear while heating or adding acids or alkali.
Gravity (1010 - 1025)	**Increase of gravity (hyperstenuria)**: insufficient fluid intake, oliguria, loss of large amounts of fluid, diabetes.
	Reduction of gravity (hypostenuria): polyuria, prolonged fasting and protein-free diet, renal failure (chronic glomerulonephritis and nephritis), diabetes insipidus.
pH (5–7)	**Increase of acidity** occurs in: starvation, states, accompanied by fever, diabetes mellitus, after heavy physical loadings, tuberculosis.
	Uric acid concrement is formed at pH below 5.5; oxalate calculus - pH 5.5-6; phosphate calculus - pH 7,0-7,8.
	In predominance of meat food –pH of urine is more acid, in vegetable food – urine is alkaline.
Protein (0,002 g/l or 30-50 mg/day)	**Proteinuria** is divided by the degree of severity: mild, moderate and severe. **Mild** (156-506 mg / day): in acute streptococcal glomerulonephritis, chronic glomerulonephritis, hereditary nephritis, tubulopathy, interstitial nephritis, obstructive uropathy. **Moderate** (500-2000 mg / day): in acute streptococcal glomerulonephritis, hereditary nephritis, chronic glomerulonephritis. **Severe** (more than 2000 mg / day): in amyloidosis, nephrotic syndrome.
	By localization, proteinuria is distinguished as following: **Prerenal** - in increased disintegration of proteins in tissues and hemolysis; **Renal** - glomerular (more pronounced), tubular (less pronounced); **Postrenal** – is associated with pathology of urinary tract (ureter, bladder, urethra, genitals).
	Physiological proteinuria - temporary appearance of protein in the urine and occurs in muscular tension, sports, after taking cold bath or shower, after stresses or emotions.
	Functional proteinuria - orthostatic proteinuria.
	Bence-Jones protein – is excreted with urine in multiple myeloma, Waldenstrom's macroglobulinemia.

Bile pigments (urobilinogen is contained in small amount and during tests is not detected)

Urobilinemia is observed in: parenchymatous jaundice, hemolytic anemia, lead poisoning. **Urobilinogen** does not get in the urine in obstructive jaundice.

Bilirubinuria is observed in: obstructive jaundice, parenchymatous jaundice (direct (conjugated) bilirubin).

In violation of heme synthesis, intermediate products of porphyrin ring synthesis and products of breakdown of hemoglobin appear in urine:
δ-aminolevulinic acid (2-3 mg/day);
Porphobilinogen (up to 2 mg/day);
Uroporphyrin (about 6 mg/day);
Coproporphyrinogens (about 70 mg/day);
Protoporphyrin (about 12 mg/day).
Increasing number of these products is observed in porphyrias, which appear in: lead poisoning, aplastic anemia, liver cirrhosis, alcoholic intoxication, use of drugs (barbiturates, organic arsenic compounds).

Glucose
(absent or up to 0,8 mmol/l)

Physiological glucosuria - nutritional, during and after stress, in pregnant women;
Pathological is observed in: diabetes mellitus, acute pancreatitis, hyperthyroidism, pheochromocytoma, acromegaly, kidney diseases with violation of processes of reabsorption of glucose (renal diabetes), steroid diabetes, brain tumors, head trauma, meningitis, encephalitis, hemorrhagic stroke, poisoning with morphine, strychnine, phosphorus, chloroform.

In addition to glucose, other sugars in the urine can be detected:
pentosuria - use of a large number of fruits,
lactosuria - in nursing mothers,
galactosuria - in galactosemia.

Ketone bodies
(absent or 20–50 mg/day)

Ketonuria develops in increased formation and deprivation of oxidation process and is observed in: decompensated diabetes, starvation, food, poor in carbohydrates, severe infection, comatose states, hepatorenal glycogenosis, increased corticosteroids level (Cushing's disease, corticosteroid therapy), thyrotoxicosis, cachexia, acromegaly, eclampsia.

Erythrocytes (females – 0–3, males – 0–2 in the field of vision)

Hematuria is observed in**:** hemorrhagic cystitis, urolithiasis (kidney stone, ureter stone, stone in the bladder), glomerulonephritis, acute pyelonephritis, tumor of kidney, bladder tumor, tumor of ureter, prostate cancer, renal tuberculosis, tuberculosis of bladder, trauma of genitourinary system, including kidney trauma, systemic lupus erythematosus, hypertension, circulatory failure, poisoning with: anticoagulants, toxins (poisonous mushrooms, snake bites), aniline, benzene, malfunction of blood clotting system (thrombocytopenia, hemophilia, anticoagulant overdose), benign familial hematuria, varicose veins of bladder neck.

Hematuria is divided into microhematuria (urine does not change color) and macrohematuria (visually, urine changes its color to red).
Unchanged erythrocytes (fresh) - contain hemoglobin and are often revealed in diseases of urinary tract, which led to direct vascular damage, cystitis, urolithiasis, urethritis
Modified erythrocytes (lysed) – do not contain hemoglobin and are

Erythrocytes (females – 0–3, males – 0–2 in the field of vision)

Leukocyteuria is revealed both in inflammatory processes of urogenital system (pyelonephritis, cystitis, urethritis, prostatitis, vesiculitis, renal tuberculosis) and in non-infectious ones (glomerulonephritis, interstitial nephritis).

If on the background of the increased number of leukocytes bacteriuria is absent, it is sterile luekocyturia. This picture may be associated with non-infectious diseases of urinary tract, or there is a bacterial process in which the agent is not detected by clinical analysis of urine or by standard bacteriological study (genitourinary tuberculosis, chlamydiosis, mycoplasmosis, ureaplasmosis).

Leukocytes (females – 0–6, males – 0–3 in the field of vision)

Depending on the degree of increase in the number of leukocytes, one distinguishes: **insignificant leukocyturia** – 8–40 in the field of vision, **moderate leukocyturia** – 50–100 in the field of vision and **expressed leukocyturia** - white blood cells cover the entire field of vision – **pyuria**

In some cases urocytogramma is performed, it shows exactly what white blood cells are present in urine, determines the nature of process, which caused leucocyturia:

neutrophils – pyelonephritis, cystitis, urethritis, tuberculosis, prostatitis
mononuclear cells - glomerulonephritis, interstitial nephritis
lymphocytes – systemic lupus erythematosus, rheumatoid arthritis
eosinophils – allergic cystitis, allergic nephritis.

Squamous epithelium (0–3), transitional (single ones in the field of vision), renal (not detected)

Squamous epithelium - lines the urethra – its number increases in inflammation of the urethra (urethritis).

Transitional epithelium - lines bladder, ureters, pelvis - increases in urine in inflammatory processes and tumors of the relevant localization (cystitis, bladder tumor, ureteral tumor, pelvic tumor, renal calculi).

Renal epithelium - lines kidney tubules - appears in urine in lesions of renal parenchyma (pyelonephritis, glomerulonephritis, tubular necrosis, taking of salicylates, cortisol, heavy metals poisoning).

Cylinders (not detected)

Cylindruria indicates various kidney damages.

Hyaline cylinders have protein structure. Hyaline cylinders appear on the background of diseases which are accompanied by renal proteinuria (glomerulonephritis, interstitial nephritis, pyelonephritis). Also, this type of cylinders may appear on physical exertion, orthostatic proteinuria, fever.

Granular cylinders – cast of kidney tubule, consisting of protein, rolled in acidic urine, on the surface of which burned-out cells of epithelial tubules have stuck. As a result, they get granular appearance, and their color is darker as compared to hyaline cylinders. Granular cylinders. appear on the background of diseases which are accompanied by renal tubular lesions and proteinuria (protein in urine), chronic glomerulonephritis, renal amyloidosis, diabetic nephropathy, pyelonephritis, viral diseases, accompanied by fever.

Waxy cylinders – casts of distal renal tubules, consisting of dead epithelial cells. In appearance they are shorter and wider than hyaline and granular, and in their structureless mass they resemble wax. Formation of waxy cylinders occurs in severe acute kidney diseases (malignant glomerulonephritis, which makes rapid progress), or in some lesions of renal tissue. Thus, appearance of waxy cylinders in urine is an unfavorable sign.

Leukocyte cylinders – are made up of protein and leukocytes. Their appearance is typical for pyelonephritis.

Erythrocyte cylinders – are made up of protein and red blood cells and are characteristic for diseases, accompanied with hematuria; acute glomerulonephritis, renal tumor, renal infarction, renal veins thrombosis, high blood pressure ...

Pigment cylinders – contain pigment (hemoglobin, myoglobin) and appear in diseases accompanied by hemoglobinuria. Pigment cylinders have brown color.

Epithelial cylinders - consist of desquamated epithelium, are determined in glomerulonephritis, nephrotic syndrome.

Bacteria (not detected)

In a healthy person urine, which is formed in kidneys and collected in bladder is sterile. However even in the norm, during laboratory diagnostics a small number of bacteria in urine may be revealed, they are admixed when urine passes through the urethra. So, in clinical urinalysis a small number of bacteria can normally be detected, and during bacteriological study – up to 100 000/ml.

Increased number of bacteria in urine - **bacteriuria** - indicates to a possible presence of inflammation in the genitourinary system: pyelonephritis, cystitis, urethritis, prostatitis, vesiculitis. There are several ways of pathogen penetration into urinary tract:

Descending route – in infectious lesions of the kidney

Ascending route – infectious agent enters urinary tract through the urethra. This variant of infection is more typical for women due to anatomical peculiarities (short and wide urethra). In addition, this mechanism of penetration of bacteria into the urine is very likely in such instrumental manipulations as bladder catheterization, urethroscopy, cystoscopy, bouginage of urethra, transurethral surgeries).

Bacteria (not detected)

Hematogenous route – pathogen enters in the urinary tract with blood from distant foci of infection.
Lymphogenous route of transmission is through lymphatic ways from infectious foci, close to genitourinary system.
One distinguishes **true** bacteriuria (bacteria live and multiply in the urine) and **false** bacteriuria (bacteria penetrate in urine with blood from distant foci of infection, do not multiply).
It should be remembered that bacteria in the urine may change its pH (acid-base properties of urine) both in one way and another. In addition, severe bacteriuria may lead to reduced transparency of urine – turbid urine.

Mucus (not detected)

Inflammatory processes (pyelonephritis, cystitis, urethritis, prostatitis) and tumors of the urinary tract may be accompanied by increased mucus secretion. In this case mucous appears in clinical urinalysis. Another possible cause of mucus in the urine is the lack of hygiene of genitals before collecting material for analysis. The source of mucus in this case may be vaginal discharge or content from the foreskin of the penis (especially in case of balanopostitis).

Yeast fungi (not detected)

However, in some states in clinical urinalysis yeast fungi, mold fungi or Actinomyces may be detected. Certain conditions are required for their appearance in urine: primary and secondary immunodeficiency (including patients receiving immunosuppressors and AIDS patients), inappropriate antibiotic therapy, various pathological processes in the urinary tract (pyelonephritis, cystitis, urethritis).
More often fungi of *Candida* genus are detected in urine. Mold fungi and Actinomyces are detected rarely.

Salt crystals (small amount)

Colloid system of urine in a healthy person is able to prevent salts precipitation, even in significant increase of their usual concentration.
Common causes for which salt crystals may be formed in urine:
- Violation of colloid properties of urine: genetic predisposition, kidney diseases, involving violation of their function, poor blood circulation in the kidney;
- Increase of salt concentration in urine: intake of large amounts of food rich in protein, dehydration (causes concentration of urine), metabolic disorders (urine acid diathesis, gout, oxalosis, Wilson's disease, xanthinuria).
Precipitation of salt crystals on the one hand may indicate to possible presence of kidney disease or metabolic disorders, on the other - is one of lithogenesis stage.

Salt crystals (small amount)

Salts of uric acid – urates, concentrated urine, acidic urine, urine acid diathesis, gout, necrotic processes in the body.
Amorphous phosphates: alkaline urine, Fanconi's syndrome, hyperfunction of parathyroid gland.
Oxalates – salt of oxalic acid: intake of food, containing large amounts of oxalic acid (cabbage, potato, tomatoes, asparagus, sorrel, spinach, oranges, apples), oxalosis.
Phosphate of lime: arthropathia of rheumatoid origin (arthritis, arthrosis), iron deficiency
Cystine: cystinosis, liver diseases (viral hepatitis, cirrhosis), Wilson's disease
Xanthine: xanthinuria
Salts of hippuric acid: intake of food containing large amount of benzoic acid – blueberries, cranberries, putrid processes in the intestine.

RULES OF URINE SAMPLING FOR DIFFERENT ANALYSES

Universal terms for urine sampling

Before sampling of urine, toilet of external genital organs should be performed. It is forbidden to collect urine during menstruation period, because the discharge from the genitals may get into urine.

Collection of urine is carried out at unforced urination, the urine is collected into to dry, clean, colorless containers (container does not touch the body), closed tightly. You cannot take urine from a bedpan or pot.

After cystoscopy it is possible to collect urine not earlier than in 5-7 days. Collected urine should be immediately transported to the laboratory.

It is acceptable to storage urine in the refrigerator (temperature from +2 to +4 ° C) but not longer than 1.5 hour.

Common Urinalysis

The first portion of morning urine is taken for the analysis (last urination should be not later than at 2 a.m.).

It is necessary to shake the sample and pour it into the container for urine with sealed lid in a volume up to 100 ml.

Analysis of urine by Nechyporenko

Collection of morning urine is carried out by the method of "three glass test" – the patient begins urination in the first glass, continues in the second, finishes in the third. By the volume, the second portion should be the largest. Second portion of urine (15-20 ml) is analyzed.

Analysis of urine by Zimnitskiy

At 6 a.m. a patient empties the bladder (this portion is poured out).

Then every 3 hours (from 9 a.m. to 6 a.m. of the next morning) 8 urine portions should be collected in clean, dry containers with volume of 250-500 ml. If the patient cannot hold urine for 3 hours, he/she collects urine during these three hours in one container. On every glass time of collecting urine is indicated.

All portions are stored at room temperature and all portions are delivered to the laboratory.

The test is carried out at the usual drinking regimen (up to 2 liters).

Analysis of daily urine

The patient collects urine during 24 hours, preserving the usual drinking regimen (1.5-2 liters).

At about 6-8 a.m. the patient empties the bladder, all urine is poured out. Then all urine is collected in a clean dark glass container, which has a wide neck (with a capacity not less than 2 liters). The last portion of urine is taken at the same time, when urine collection was started the day before (start time and end time is indicated).

Container with urine is stored in a cool place.

Volume of urine is measured and recorded, after shaking, about 100 ml of urine is poured out and this portion is delivered to the laboratory.

ALGORITHM OF EVALUATION OF URINANALYSIS BY NECHYPORENKO

Unlike common urinalysis, in this case quantitative indicators are calculated not in the field of vision but in 1 ml.

Norm:

- **leukocytes – in men** not more than 2,000 in 1 ml, **in women** not more than 4,000 ml in 1 ml;
- **erythrocytes** - not more than 1,000 in 1 ml;
- **cylinders** - not more than 20 in 1 ml.

Analysis by Nechyporenko is made to reveal **occult** leukocyturia and erythrocyturia (which were not detected in common urinalysis) and to follow-up the effectiveness of the treatment. In addition, this analysis allows to make initial differential diagnosis between infectious diseases and other pathology caused by another factor. So if there is an overwhelming increase in the number of white blood cells, it is necessary to think about infectious lesions (cystitis, pyelonephritis), if there is a marked increase in the number of red blood cells, it is necessary to exclude urolithiasis (renal calculi, ureteral calculus, bladder stones), glomerulonephritis, neoplastic processes (tumor of the kidney, ureteral tumor, bladder tumor).

ALGORITHM OF EVALUATION OF URINANALYSIS BY ZIMNITSKIY

Evaluation of analysis results:

- urine volume, excreted per day (first 4 portions: from 9:00 to 21:00) must exceed urine excreted per night (4 portions: from 21:00 to 9:00) by 2 times;
- specific gravity should be at least 1,012-1,016 and at least one portion of it should be 1,017 or more;
- low specific gravity of urine in different portions as well as reduction of daily fluctuations of this index indicates to decrease of concentration ability of the kidneys. Often, this is one of the first manifestations of kidney failure and is formed on the background of various kidney diseases: glomerulonephritis, chronic pyelonephritis, hydronephrosis, polycystic;
- combination of low density of urine with its very small daily fluctuations (not more than 1003-1004) may indicates to a so-called "diabetes insipidus" - hormonal disease in which production of antidiuretic hormone (vasopressin) is reduced.

PROCEDURE OF BLOOD SAMPLING AND INTERPRETATION OF DATA OF BIOCHEMICAL BLOOD TESTS

RULES OF BLOOD SAMPLING FOR BIOCHEMICAL ANALYSIS

To eliminate the factors that may affect the results of the study, it is necessary to comply with the following rules:

- an important condition of blood sampling for laboratory tests is fasting blood test performed in 8-12 hours after overnight fasting. On the day of the study it is acceptable to drink a small amount of water;
- alcohol intake; smoking; fried and fatty food should be excluded preferably 3 days before; to limit physical activity;
- to discontinue medication, but if it is impossible - to inform laboratory technician;
- if the patient cannot visit the lab in the morning, blood can be taken in the afternoon, after 4-6 hours' fasting period, fatty food should be excluded from the breakfast.

RULES OF GLUCOSE TOLERANCE TEST

Glucose tolerance test is performed to detect diabetes and hidden carbohydrates metabolic disorders. The principle of the test is to measure glucose 2 times: before load and in 2 hours after it.

Indications for the test are: controversial results of glucose measurement, incidentally revealed hyperglycemia or glucosuria and clinical signs of diabetes with normal glucose content. It should be noted that if the diagnosis "diabetes mellitus" is beyond a doubt, this test can lead to glycaemic shock development.

Overnight fast during 8 hours (maximum 14 hours) precedes the test, patient may drink water. Last evening meal should contain 30-50 grams of carbohydrates. The patient is recommended to refrain from smoking, drinking coffee and alcohol, as well as from heavy physical exercise for 8 hours before the study and during it.

Glucose load test: 75 gr of anhydrous glucose is dissolved in 25–300 ml of water, patient must drink it during 3–5 minutes on an empty stomach.

The doctor in charge and doctor-laboratory assistant should know about the drugs taken by the patient, which can affect the outcome of the study. If necessary, these drugs are discontinued (3 days before: diuretics, oral contraceptives, glucocorticoids). It is worth to inform the patient about symptoms of hypoglycemia (weakness, restlessness, irritability, hunger, sweating) if they appear, the patient must immediately inform the doctor.

ALGORITHM OF EVALUATION OF GLUCOSE TOLERANCE TEST

In case of oral glucose tolerance test the following values are base-line (in capillary and venous blood):

- normal glucose tolerance is characterized by glycaemia level <6,7 mmol/l in 2 hours after glucose load.
- increased concentrations of plasma glucose in 2 hours after a glucose load to values >7,8 mmol/ (>140 mg/dl), but <11,1 mmol/l (<200 mg/dl) indicates to impaired glucose tolerance.
- glucose in plasma >11,1 mmol/l (> 200 mg/dl) in venous blood in 2 hours after glucose load can be the basis for the preliminary diagnosis of diabetes melitus.

ALGORITHM OF EVALUATION OF PROCALCITONIN (PCT) LEVEL

To determine **possibilities of sepsis development** the following parameters of PCT are used:

- PCT <0,5 ng/ml – low risk of severe sepsis and/or septic shock, it is recommended to repeat the measurement in 6–24 hours;
- PCT from 0,5 to 2 ng/ml – moderate syndrome of systemic inflammatory response (SSIR) - "gray zone." For certain diagnosis of sepsis is impossible to make, it is recommended to repeat the measurement in 6–24 hours, followed by daily measurement;

- PCT> 2 and <10 ng/ml – severe SSIR, high risk of severe sepsis and/or septic shock, it is recommended to repeat the measurement in 6–24 hours, followed by daily measurement;
- PCT> 10 ng/ml – pronounced SSIR - almost always is due to severe bacterial sepsis or septic shock. These levels of PCT are often associated with syndrome of multiple organ failure and indicate to a high risk of lethal outcome, daily measurements are recommended.

To determine **possibilities of acute inflammations of the lower respiratory tract** (asthma, COPD, community acquired pneumonia) the following PCT parameters are used:

- PCT <0.1 ng/ml - a very low risk of bacterial infection, administration of antibacterial agents is strongly not recommended, it is recommended to repeat the measurement in 6-24 hours;
- PCT from 0.1 to 0.25 ng/ml - low risk, administration of antibacterial agents is not recommended, it is recommended to repeat the measurement in 6-24 hours;
- PCT from 0.25 to 0.5 ng/ml – probably a bacterial infection, antibiotics are recommended, the dynamics of PCT changes should be assessed;
- PCT> 0.5 ng/ml - a high risk of bacterial infection, antibacterial agents are strongly recommended, the dynamics of PCT changes should be assessed.

ALGORITHM OF EVALUATION OF LIPID PROFILE

Atherogenic dyslipidemia (DL) – is the primary polygenic DL, the development of which is caused by the interaction of risk factors related to lifestyle (smoking, poor nutrition, low physical activity) and polygenic predisposition; less often it is monogenic hereditary DL, leading to early family atherosclerosis (AS). The degree of atherogenicity of lipoproteins depends on their size and concentration. The most atherogenic lipoproteins are low-density lipoproteins (LDL) and very low density (VLDL) ones. Antiatherogenic effect is produced by

high-density lipoproteins (HDL), which implement the reverse transport of cholesterol to the liver.

Today, lipid risk factors of AS are:

- Total cholesterol (TC)> 5,0 mmol/l;
- LDL cholesterol > 3,0 mmol/l;
- HDL cholesterol <1,0 mg/dl in men and <1,2 mmol/l in women;
- Plasma triglycerides (TG)> 1,2 mmol/l;
- The ratio of LDL/HDL cholesterol> 5 (atherogenic index).

At the present stage to characterize violations of lipid profile, the following terms are used: dyslipidemia, hyperlipidemia and hyperlipoproteinemia.

The term **dyslipidemia** is the broadest because it includes both increased levels of lipids and lipoproteins higher than the optimal value, and/or possible decreases of the lipid spectrum part, namely HDL or alpha-lipoproteins.

The term **hyperlipoproteinemia** (HLP) means any increase in the levels of lipids and lipoproteins in plasma above the optimum value.

The term **hyperlipidemia** is the simplest, because for its use it is enough to determine only increased levels of blood lipids (cholesterol and triglycerides) above the optimum value.

In 1967 D.Fredrikson proposed classification of primary DL with five main types of DL (table 9). It is the basis of modern classification, approved by the WHO.

Table 9

Classification of hyperlipoproteidemia

Phenotype	Cholesterol	LDL	TG	Changes of LP	Atherogenic degree
I	Increased	Lowered or normal	Increased or normal	↑ Ch	Non-atherogenic phenotype
IIa	Increased	Increased	Normal	↑ LDL	High
IIb	Increased	Increased	Increased	↑ LDL and VLDL	High
III	Increased	Lowered or normal	Increased	↑ HDL	High
IV	Most normal	Normal	Increased	↑ LDL	Moderate*
V	Increased	Normal	Increased	↑ Ch and LDL	Low

Note: ↑ – increase of concentration; * – IV phenotype becomes atherogenic if it is accompanied by decrease in HDL cholesterol, as well as other metabolic disorders (hyperglycemia, insulin resistance, impaired glucose tolerance), LDL - low density lipoprotein; Ch - chylomicrons.

It was found that the greatest risk of CHD and AS development occurs in **II**, **III** and **IV** types of hyperlipoproteidemia.

WHO classification does not take into account the phenotype, which is characterized by the selective reduction of HDL cholesterol (hypoalphalipoproteidemia). This phenotype is more common in men and is accompanied by lesion of coronary and cerebral vessels. Importantly, that this classification precludes possiblity to diagnose the disease which caused dyslipidemia, but allows the physician to establish the degree of its atherogenicity.

In the medical literature to assess the levels of lipoproteids, the classification of components of lipid profile, proposed in the Third report on treatment of

dyslipidemia in adults (Adult Treatment Panel – ATP-III) of the National Cholesterol Education Program of USA is commonly used (table 10).

Table 10

Classification ATP-III (2001)

LDL cholesterol, mg / dL (mmol / L)	
< 100 (< 2,6)	optimal
100–129 (2,6–3,3)	above optimal
130–159 (3,4–4,0)	extremely high
160–189 (4,1–4,8)	high
≥ 190 (≥ 4,9)	very high
Total cholesterol, mg / dL (mmol / L)	
< 200 (< 5,2)	preferred (normal)
200–239 (5,2–6,1)	extremely high
≥240 (≥ 6,2)	high
HDL cholesterol, mg / dL (mmol / L)	
< 40 (< 1,0)	low
≥ 60 (≥ 1,6)	high
Triglycerides, mg / dL (mmol / L)	
<150 (< 1,7)	normal
150–199 (1,7–2,2)	extremely high
200–499 (2,3–4,4)	high
≥ 500 (≥ 4,5)	very high

The diagnosis of dyslipidemia, hyperlipidemia and HLP is not independent, it must be included in the primary clinical diagnosis of cardiovascular disease. For a wide use in clinical diagnosis it is proposed to use a simplified version of dyslipidemia classification.

ALGORITHM OF BLOOD CHEMISTRY EVALUATION

NB! When conducting data interpretation of biochemical studies, the difference in the reference values of the indicators provided by different laboratories should be taken into account and, if possible, to carry out the subsequent dynamic sudy in the same laboratory.

General protein (65–85 g/l)

Increased protein level (**albuminosis**) is revealed in: multiple myeloma, Hodgkin's disease, autoimmune diseases, sarcoidosis, cirrhosis without severe hepatocellular insufficiency (absolute); extensive burns, prolonged vomiting, diarrhea, diabetes insipidus, chronic nephritis, increased sweating, intestinal obstruction (relative).

Reduced protein level (**hypoproteinemia)** is observed during pregnancy and lactation (physiological); starvation, inflammatory disorders of gastrointestinal tract, esophageal stricture, poisoning, hepatitis and cirrhosis of the liver, albuminemia, Wilson’s disease, malignant tumors, major burns, hyperthyroidism, after prolonged fevers, injuries, hydration, physical loads, increased excretion of protein with urine (diabetes, glomerulonephritis, nephrotic syndrome, long-term (chronic) diarrhea), protein displacement in "other areas" (pleural exudate and transsudate, ascitis), bleedings (absolute); water loading, arrest of urination, reduced urine output, massive intravenous infusion of glucose solution, impaired renal excretory function, increased secretion of the antidiuretic hormone

Albumin (35–55 g/l)

Increased albumin occurs very rare, more often it is caused by dehydration and in hemoconcentration (introduction of "concentrated" solutions of albumin)

Reduced level (**hypoalbuminemia**) occurs in: insufficient entry of protein to the body (starvation, esophageal stricture and others); malabsorption of protein breakdown products through the mucous membrane of the gastrointestinal tract (enteritis, resection of the stomach because of stomach ulcer, cancer); reduced albumin synthesis (toxic damages liver, cirrhosis); increased loss of protein (exudate, bleeding) in the lumen of the intestine (in volvulus, ulcerative colitis, peritonitis), on the burn surface (in extensive burns), with urine in patients suffering from nephrotic syndrome, acute and chronic glomerulonephritis; in bleedings, exiting into the "other areas" (formation of exudate and transsudate); in the postoperative period.

C-reactive protein (5 mg/l)	Increased C-RP level is revealed in: acute inflammation, myocardial infarction, systemic lupus erythematosus, rheumatoid arthritis, infectious nonspecific arthritis, nephritis, Hodgkin's disease, chronic infections, tuberculosis, obesity, transplant rejection, smoking, metabolic syndrome, usage of estrogens and oral contraceptives. In bacterial infections it can rise to the level of 100 mg/l and higher, whereas in viral infections – only up to 20 mg/l.
Fibrinogen (1,5–4,0 g/l or 5,8–11,6 mcmol/l)	Increased fibrinogen level occurs in: various inflammatory processes in the vital organs: lungs (pneumonia), kidneys (nephrotic syndrome, acute and chronic pyelonephritis, glomerulonephritis, hemolytic uremic syndrome); liver; peritoneum (peritonitis); reactions of acute phase (fever, inflammation and necrotic processes, infectious diseases, acute myocardial infarction, trauma, burns, major surgery); collagenic diseases, radiation sickness, malignant tumors, especially of lungs; physiological increase in pregnancy and during menstruation period.
	Reduced fibrinogen level is observed in: hereditary deficiency of fibrinogen (non- and hypofibrinogenemia), syndrome of disseminated intravascular coagulation, condition after bleeding, pregnancy, accompanied by placental abruption, amniotic fluid embolism, rapid labor, thrombolytic therapy, asparaginase therapy, leukemia, meningococcal meningitis, acute and chronic liver disease, prostate cancer with metastases, liver cirrhosis, poisoning with hepatotropic agents, metastatic lesions of the bone marrow, usage of certain medications (phenobarbital, streptokinase, urokinase).
D-dimer (< 0,25 mg/l or < 0,5 mg/l FEU)	Increased level is observed in: disseminated intravascular coagulation, pulmonary embolism, deep vein thrombosis, surgeries, sepsis and septicemia, pregnancy, eclampsia, liver disease, malignant neoplasms, systemic autoimmune disease, myocardial infarction, stroke, renal infarction, Badda-Chiari's syndrome, congestive heart failure, snake bite.

Procalcitonin (< 0,05 ng/ml)	Increased procalcitonin (PCT) occurs in states, associated with infections: sepsis with confirmed or unconfirmed bacterial infection; conditions, associated with sepsis, such as acute pancreatitis; distinct systemic infection that can occur in pneumonia or acute pyelonephritis; clear systemic viremia, fungal infections, severe malaria; pulmonary diseases: aspiration and ventilator-associated pneumonia; adult respiratory disease syndrome (ARDS); pulmonary neuroendocrine hyperplasia that occurs in chronic obstructive pulmonary disease (COPD) or chronic bronchitis associated with smoking; malignant tumors, medullary thyroid cancer (cancer of the thyroid C-cell); small-cell lung cancer; non-small-cell lung cancer; carcinoid tumor; other neuroendocrine tumors (pheochromocytoma, insulinoma); breast cancer. Increase is not associated with infections: burns; injuries; sunstroke (heat stroke), first days of therapy with immunosupressors and other drugs that cause the release of inflammatory cytokines; the first 2 days of life after birth; prolonged or severe cardiogenic shock; heavy and prolonged violation of microcirculation.
Serum glucose (3,3–6,1 mmol/l)	Increased glucose level is revealed in: diabetes mellitus, traumatic lesion of the central nervous system, brain tumor, severe liver damage, acute and chronic pancreatitis, pancreatic cancer, thyrotoxicosis, acromegaly, Cushing's disease and syndrome, pheochromocytoma, burns, stress situations, excessive use of carbohydrates by patients on dialysis, after consuming caffeine, adrenaline, strychnine, narcotic agents and hypnotics (ether, opium, morphine, veronal, chloroform, etc.).
	Reduced glucose level is observed in: overdose of insulin and other oral antidiabetic drugs, starvation, hyperinsulinism, insulinoma, hormonal deficiency of thyroid gland (hypothyroidism), adrenal glands, pituitary gland; congenital metabolic conditions (galactosemia, fructose intolerance, glycogen storage diseases), toxic liver damage (poisoning with chloroform, salicylic acid), some kidney lesions, lesions of small intestine, large resection of the stomach.

Glycosylated hemoglobin (HbAl) (< 6 %)	Increased HbAl level occurs in diabetes (5,5–8% – well compensated diabetes, 8–10% – quite well compensated diabetes, 10–12% – partly compensated diabetes, more than 12% – uncompensated diabetes).
	Reduced HbAl level occurs in: active synthesis of hemoglobin, hemolysis, regeneration of erythropoiesis after blood loss.
Alanine aminotransferase (AlT) (m – 22 U/l, f – 17 U/l)	Increased ALT is observed in: viral hepatitis, toxic liver damage (with chloroform, pesticides, salts of heavy metals and organic-chlorine compounds), infectious mononucleosis, cholestasis, liver cirrhosis, complicated myocardial infarction, treatment with large doses of salicylates, prolonged use of fibrates, sulfonylurea drugs of the first generation .
Aspartate transaminase (AST) (m – 18 U/l, f –15 U/l)	Increased AST level is revealed in: muscular dystrophy, myocardial infarction, strokes and severe tachyarrhythmias, acute rheumatism, cardiac surgery, angiocoronarography, pulmonary embolism, toxic liver damage (with chloroform, pesticides, salts of heavy metals, organic-chlorine compounds), infectious mononucleosis, cholangitis, compensated cirrhosis, acute alcohol poisoning, hemolytic syndrome, acute pancreatitis, amebiasis.
γ- hlutamyl transferase (GHT) (m – 18 U/l, f –15 U/l)	Increased GHT level is revealed in: mechanical and congestive jaundice, cholelithiasis, cholecystitis, acute viral chronic hepatitis, toxic liver damage and radiation, posthepatic cirrhosis (compensated), alcoholic cirrhosis, bacony liver, primary liver tumors, malignant tumors with metastases in the liver, acute and chronic pancreatitis, pancreatic cancer, chronic glomerulonephritis, renal amyloidosis, myocardial infarction, alcoholism, treatment with anti-epileptic agents, rifampicin, oral contraceptives, anabolic steroids, thiazide diuretics.
	Reduced GHT level is revealed in decompensated cirrhosis.

α- amylase (up to 120 U/l)	Increased amylase level is observed in: acute pancreatitis, exacerbation of chronic pancreatitis, mumps, inflammation of the pancreas on the background of perforated peptic ulcer, renal failure, diabetic acidosis, after using alcohol, introduction of epinephrine, corticosteroids, oral contraceptives, narcotic agents (opiates, morphine heroin, codeine), tetracyclines, salicylates, furosemide, methanol
	Reduced amylase level is revealed in: necrosis of the pancreas, thyrotoxicosis, myocardial infarction.
Creatine phosphokinase (CPK) (m – < 80 U/l, f – < 70 U/l)	Increased CPC is observed in: myocardial infarction, muscular tissue lesions (trauma, dermatomyositis, Duchenne's myodystrophy, polymyositis, rheumatic heart disease), severe physical exertion and running, hypothyroidism, stroke, acute alcohol intoxication, schizophrenia, epilepsy, head trauma, acute radiation sickness.
Lactate dehydrogenase (LDG) (120–240 U/l)	Increased LDH is revealed in: myocardial infarction, insufficient function of the cardiovascular and pulmonary systems, inflammatory diseases of the liver and kidneys, pneumonia, pulmonary infarction, viral hepatitis, leukemia, erythremia, malignant tumors, muscle damage, muscle atrophy.
Alkaline phosphatase (AP) (20–130 U/l)	Increased amylase is registered in: pregnancy, liver disease with cholestasis, obstructive jaundice, rickets in children, bone disease, osteomalacia, Paget's disease, leukemia, inflammation of the bile duct, hepatic jaundice, liver cirrhosis, infectious mononucleosis, multiple myeloma, Hodgkin's disease with bone lesions, breast adenoma, malignant tumors of the ovaries, cancer of the cervix and endometrium, hyperparathyroidism, diffuse toxic goiter, limited scleroderma, sarcoidosis, exposure to drugs (sulfonamides, phenylbutazone, erythromycin, tetracycline, lincomycin, novocaine).
	Reducing of AP is revealed in: myxedema, hypothyroidism, senile osteoporosis, accumulation of radioactive substances in bones, severe anemia, slow growth in children, C hypovitaminosis, D hypervitaminosis.

Total bilirubin (3,4–20,5 mcmol/l) **Indirect bilirubin (free) (1,7–17,1 mcmol/l)** **Direct bilirubin (conjugated) (0,86–5,3 mcmol/l)**	Increased levels of total and indirect (free) bilirubin (hyperbilirubinemia) are observed in: increased degradation of red blood cells (acute and chronic hemolytic anemia), hemolytic disease of newborns, B12-deficient anemia, thalassemia, large hematomas. Increased direct and total bilirubin may be acquired and innate. Acquired lesions include: infectious and viral hepatitis, cirrhosis, fatty liver, cholelithiasis, jaundice, pancreatic cancer, drug therapy. Level of direct (conjugated) bilirubin increases under the influence of drugs which provoke cholestasis (penicillin, erythromycin, sulfonamides, oral contraceptives, estrogens, androgens, nicotinic acid).
Rest nitrogen (14–28 mmol/l)	Increased rest nitrogen level is observed in: conditions associated with increased protein breakdown (malignant neoplasms, pulmonary tuberculosis, typhus, diphtheria, scarlet fever, severe pneumonia, diabetes, severe liver cirrhosis, acute yellow liver, peritonitis); retention of nitrogenous toxins in the body (acute and chronic renal failure and other renal diseases, violation of blood circulation due to weakening of cardiovascular activity); relative hyperazotemia due to loss of water (profuse diarrhea, increased sweating).
Blood urea (3,5–8,3 mmol/l)	Increased urea level is revealed in: enhanced formation due to protein-rich diet, excessive catabolism of protein, leukosis, jaundice, severe infections, burns, dysentery, shock, intestinal obstruction; reduced excretion with urine (acute and chronic renal failure, tumors of excretory tract, of prostate, urolithiasis, heart failure); bleeding from upper gastrointestinal tract; the use of drugs (sulfonamides, chloramphenicol, tetracycline, gentamicin, furosemide, dopegit, nevigramon, lasix), dehydration.
Urea nitrogen (2,9–8,9 mmol/l)	Reduced urea level is revealed in: severe liver damage (poisoning with phosphorus, arsenic and other hepatotropic agents), decompensated cirrhosis, starvation, reduced protein catabolism, after administration of glucose, after dialysis, cachexia.

Creatinin (m – 44–115, f – 44–97 mcmol/l)	Increased creatinine level is observed in: acute expressed violations of liver function, cardiovascular insufficiency, inflammatory lung diseases, fever, intestinal obstruction, renal failure, urinary tract obturation, acromegaly, gigantism, diabetes mellitus, starvation, enhanced muscular work, acute breakdown of muscular tissue, impact of nephrotoxic drugs.
	Decreased creatinine level is observed in: fasting, pregnancy, prolonged use of corticosteroids.
Sodium (130–150 mmol/l)	Increased sodium level is revealed in: Cushing's syndrome and disease, aldosteron-producing tumors, diarrhea, vomiting, increased diuresis, increased sweating, diabetes insipidus, reduced water intake, increased salt intake, nephritis and nephrotic syndrome, hyperventilation, primary hyperaldosteronism, stenosed renal artery, uncompensated diabetes mellitus.
	Reduced sodium level is observed in: insufficient sodium input into the body (salt-free diet), diarrhea, vomiting, bleeding, burns, treatment with diuretics, Addison's disease, removal of ascetic fluid, hyperglycemia.
Potassium (3,6–5,4 mmol/l)	Increased level of potassium is detected in: generalized necrosis, intravascular hemolysis of red blood cells, tumors, severe traumas, starvation, tissue hypoxia, metabolic or respiratory acidosis, acute and chronic renal failure, oliguria and anuria, massive introduction of potassium, primary and secondary failure of adrenal cortex, polyuria, medications (indomethacin, captopril, diuretic and antihypertensive drug therapy).
	Reduced level of potassium is observed in: insufficient sodium input into the body (loss of appetite, prolonged starvation, lack of food intake), Kon's syndrome, Fanconi's syndrome, unrestrained vomiting, prolonged profuse diarrhea, acute and chronic diarrhea, fistulas (stomach, intestine) after administration of glucose, insulin, epinephrine, ACTH, mineral corticoids, anorexia
Chlorides (95–110 mmol/l)	Increased level of chlorides is revealed in: dehydration, hyperventilation, acute renal failure, nephropathy, inflammatory kidney disease, diabetes insipidus, severe cardiovascular insufficiency.

Chlorides (95–110 mmol/l)	Reduced level of chlorides is determined in: edemas, excessive sweating, diarrhea, prolonged vomiting, loss of contents of the small intestine, pneumonia, gastric hypersecretion, severe infectious diseases, lactatacidosis, increased intake of water.
Iron (m – 14,3–25,1 f – 10,7–21,5 mcmol/l)	Increased iron level is determined in: hemolytic anemia, hypoplastic anemia, thalassemia, pernicious anemia, B_{12} and folic deficiency anemia, use of oral contraceptives, lead intoxication, viral hepatitis, liver damages (acute hepatitis, acute hepatic necrosis, chronic cholecystitis).
	Reduced iron level is observed in: iron deficiency anemia due to its insufficient entry with food, gastritis with decreased secretion, gastric and colon tumors, gastric resection, inflammation, purulent septic infections, osteomyelitis, rheumatoid arthritis, myocardial infarction, hemosiderosis of internal organs, chronic renal failure, nephrotic syndrome, pregnancy, liver cancer, obstructive jaundice, uterine fibroids, bleeding, deficiency of vitamin C.
Total transferrin (26,85–41,17 mcmol/l)	Increased total transferrin level (total iron binding capacity of blood) is observed in: iron deficiency anemia, use of oral contraceptives, acute hepatitis and cirrhosis, long-term iron therapy, frequent blood transfusions, late term of pregnancy, excessive intake of iron into the body.
	Reduced total transferring level (total iron binding capacity of blood) is observed in: reduce of total protein in starvation, nephrotic syndrome, cancer and other oncologic diseases, chronic infections, hemosiderosis, states, accompanied by lack of iron in the body.
Transferrin (m – 23–43, f – 21–46 mcmol/l)	Increased transferrin level is observed in: iron deficiency, pregnancy, blood loss.
	Reduced transferring level is observed in: inflammation, malignant tumors, nephrotic syndrome, hepatopathy, hemochromatosis, hyperchromic anemia, thalassemia.

HEMOSTATIC SYSTEM STUDY

Hemostatic system is a biological system (complex of cellular and humoral factors) that ensures preservation of sparse state of circulating blood and adequate blood supply to organs, as well as prevention and quick control of bleeding by maintaining the integrity of blood vessel walls and their rapid thrombosing in case of damage.

Hemostasis is realized by the interaction of three structural and functional components:

1. blood vessel wall (endothelium and subendothelial structures)
2. blood cells (platelets)
3. plasmatic enzyme systems (coagulation, fibrinolytic/plasmatic, kallikrein-kinin and complement system).

Thus, evaluation of hemostatic system is based on the analysis of indicators:

• vascular-platelet hemostasis,

• plasmatic-coagulation hemostasis

• state of fibrinolytic/anticoagulant system.

Indicators of vascular-platelet hemostasis:

- bleeding time (duration) – Duke's test – duration of capillary bleeding after prick of fingertip with disposable lancet to a depth of 4 mm, the stop of which is mainly due to the presence and functional activity of platelets. Normally it lasts for 2–5 minutes. Extension of time: thrombocytopenia, thrombocytopathy, von Willebrand's disease, severe forms of disseminated intravascular coagulation (DIC) syndrome, severe heparinemia.
- Index of blood clot retraction – the process of reduction, densification and discharge of serum with blood clot after fibrin formation under the influence of contractile protein which is found in platelets. Norm is 48–64%. Reducing of index is observed in severe thrombocytopenia and thrombasthenia.

- Study of platelet aggregation function – aggregatographia with the usage of stimulants of aggregation (epinephrine, ADP, collagen, rystocytinum).

ALGORITHM OF PLEURAL EFFUSION STUDY

Pleural cavity is a closed space located between the surface of the lungs and chest with the presence of a small amount of pleural fluid (approximately 0,1–0,2 ml/kg on each side of the chest) and protein (less than 1 g/l) to grease leaves, due to its presence the movements of the lungs are facilitated during inhalation and exhalation. Under normal conditions a stable equilibrium between the liquid coming into the pleural cavity and absorbed from it is present.

Accumulation of fluid in the pleural cavity depends on changes of countereffort of hydrostatic and oncotic pressure (in transsudate) and changes of pleural membrane permeability (in exudate). The difference of gradients facilitates transsudation of fluid into the pleural cavity, but existing lymphatic drainage prevents excessive accumulation of fluid.

The immediate causes of excessive formation/accumulation of pleural fluid:

- increase in hydrostatic pressure in the capillaries of the parietal and visceral pleura;
- violation of lymphatic drainage at different levels, which leads to increased osmotic pressure of the pleural fluid;
- decrease of oncotic pressure of blood plasma;
- increase of capillary permeability;
- increase of the negative pressure in the pleural space;
- violation of the integrity of pleura and/or directly adjacent large vessels, especially of lymphatic thoracic duct;
- increase of protein content in the pleural cavity;
- immunological inflammation.

Puncture of the pleural cavity may be of therapeutic and diagnostic purpose.

Indications for pleural puncture with diagnostic purpose: to clarify the nature of effusion (transsudate or exudate) whether the fluid contains blood, pus or lymph.

Indications for pleural puncture with medical purpose: inflammatory exudates, congestive exudates, spontaneous or traumatic pneumothorax, hemothorax, chylothorax, empyema.

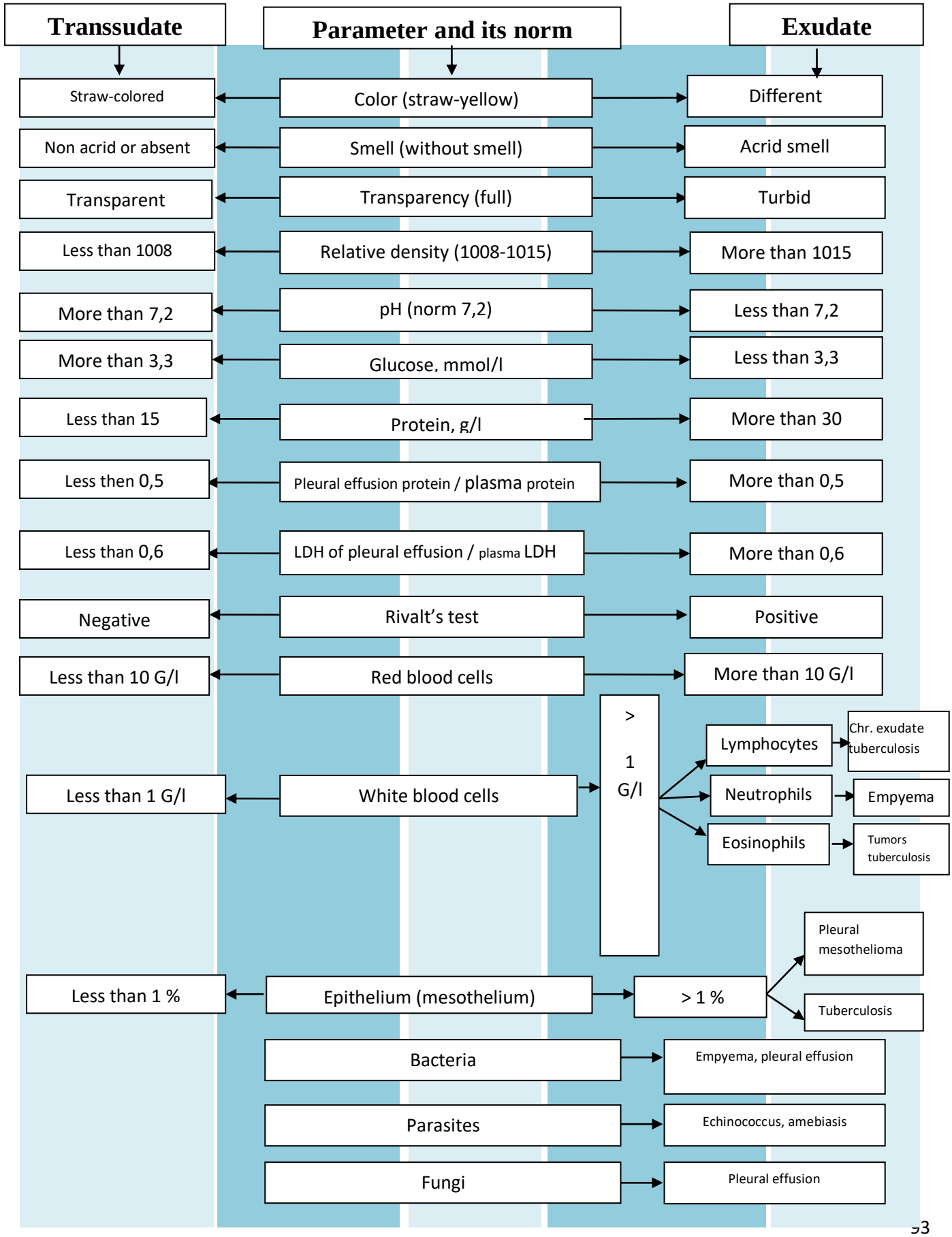
PROCEDURE OF PLEUROCENTESIS PERFORMANCE

Puncture of the pleural cavity is performed under the local anesthesia. Puncture site depends on the nature of the pathological process. In case of pneumothorax puncture is performed in frontal position in the second intercostal space along the mid clavicular line. In case of fluid accumulation – in the lower parts of the chest.

Choice of patient's position depends on his/her state. In severe state when the patient is not able to sit, it is possible to perform puncture in the recumbent position with the head end summed on the side. In all other cases the patient is sitting with the arms crossed on the chest. After defining puncture site (by using X-ray, ultrasound or via percussion), the skin is treated with antiseptic solution. Puncture site should be located 2–3 cm below the level of the fluid (usually below the angle of the scapula), but not lower than 8th intercostal space (due to the risk of liver damage), at the level of the upper edge of the rib which is located below. Puncture should be performed by the needle with the cut being atop the rib, because along the lower edge of the rib the groove, containing intercostal artery and nerves passes. Puncture site is infiltrated with novocaine and then along the passage of a needle a local anesthetic is being administered. In penetration of the needle into the pleural cavity, feeling of gap of the needle may appear. In correct position of the needle, pleural fluid or air appears in the syringe, depending on the disease. After removal of the pathological content the needle with the syringe are removed and aseptic bandage is applied. After the manipulation, a radiographic

study of the lungs is performed to control quality of puncture and the fluid obtained is sent for analysis.

ALGORITHM OF PLEURAL PUNCTATE ANALYSIS



EVALUATION ALGORITHM OF FRACTIONAL DUODENAL INTUBATION

I фаза, порція А

- contents of the duodenum before stimulus introduction, during 20–40 minutes 15–20 ml of golden-yellow bile is discharged, it is transparent, pH - neutral or alkaline, density - 1003-1016, bile acids - 17,4–52,9 mmol /l, cholesterol - 1,3–2,9 mmol /l, bilirubin - 0,17–0,34
- reduced amount – hyposecretion (after cholecystectomy), in excluded gallbladder, hemolytic jaundice);
- intermittent discharge – hypertension of Oddi's sphincter (duodenitis, angiocholitis, calculi, malignancies);
- change of color: dark yellow - in reflux of bile of B portion and hemolytic jaundice; light yellow - in lesions of the liver parenchyma, viral hepatitis, cirrhosis of the liver; blood staining - in duodenal ulcer, major duodenal papilla tumor, hemorrhagic diathesis.

II фаза (закритого сфінктера Одді)

- time before appearance of bile portion from the common bile duct (so-called portion A1) – 3–6 minutes;
- shortening of time – hypotonia of Oddi's sphincter, increased pressure in the common bile duct;
- lengthening – hypertrophy of Oddi's sphincter, stenosis of duodenal papilla.

III фаза

- content of the common bile duct, 3–4 minutes, 3–5 ml of light yellow bile;
- lengthening – atony of the gallbladder, its blockade of spastic or organic origin (gallstones)

IV фаза, порція В (міхурова)

- during 20–30 minutes 20–50 ml of bile of olive color is discharged, alkaline pH, density 1016–1032, content of bile acids 57,2–184,6 mmol/l, bilirubin – 6,8 mmol/l;
- accelerated secretion – hyperkinetic dyskinesia;
- lengthening – hypotonic dyskinesia of the gallbladder;
- reduced amount – decrease of bladder volume in sclerotic changes, cholelithiasis;
- absent in: obstruction of duct with stone, tumor, violation of bladder motility. rarely in cholecystitis; lack of bladder reflex;
- change of color: white bile – chronic inflammation with atrophy, very dark bile - formation of

V фаза ("печінкової" жовчі)

- light yellow, alkaline pH, density 1007–1011, bile acids 13–57,2 mg/dl, bilirubin 0,17–0,34 mmol/l;
- pale color – hepatitis, cirrhosis; dark (pleyochromic) – hemolytic jaundice; red – tumors;
- flows permanently while probe is in;
- absent - in compression of common bile duct with stone or tumor or in arrest of bile secreting function of the liver in severe parenchymatous lesions.

Мікроскопія

- Norm: no cellular elements!
- mucus, catarrhal inflammation of biliary tract, duodenitis;
- red blood cells: trauma during intubation;
- white blood cells: may be present only in portion A, single ones in field of vision; localization of inflammatory process is revealed on the basis of the largest concentration in certain portion;
- cholesterol crystals: cholelithiasis;
- !!! in the norm bile is sterile!

SPUTUM

Sputum is a pathological secretion of the lungs and airways (bronchi, trachea, larynx), which is discharged in expectoration. Normally, glands of the large bronchi and trachea constantly produce up to 100 ml of secretion a day, which is generally swallowed and in healthy people it is not discharged.

Indications for sputum analysis:

- cough with sputum discharge;
- pneumonia;
- bronchitis;
- asthma;
- COPD;
- lung abscess;
- TB;
- suspicion for cancer;
- helminthes and fungi;
- bronchiectasis.

Procedure of sputum collection

The patient collects sputum in the morning on an empty stomach. Beforehand teeth should be brushed and mouth should be rinsed with boiled water. If the patient wears dentures, they should be removed. Sputum is collected into sterile container during attack of cough or deep forced clearing of throat. To facilitate the discharge of mucus it is recommended to previously perform inhalation of pharynx with 0,9 % solution of sodium chloride (using nebulizer) or to take expectorants.

Collection of sputum for general analysis is performed irrespective of antibiotic treatment. Containers for sputum collection should be made of durable material with tightly closed lid, to prevent possible spread of infection. Containers should be transparent in order to assess the quantity and quality of the collected

sample without opening the lid. If the sputum was collected at home or if laboratory is located in another medical facility, transporting time should not exceed 2 hours from the time of collection, if it is impossible – limits of sample storage in the refrigerator is 8 hours at a temperature of + 4–8° C.

If it is planned to make a bacteriological investigation of sputum, sputum collection should be done before antibiotics taking orally or in three weeks after discontinuation of antimicrobial treatment. In case of failure of administered antimicrobial therapy, collecting is performed as occasion requires, following the previously described technique.

Some quantitative and macroscopic features of sputum

Amount of sputum, depending on a pathological process can range from a few milliliters to several liters per day. A small amount of sputum is discharged in acute bronchitis, pneumonia, heart failure or at onset of asthma attack. A large amount of sputum is discharged in lung edema, suppurative processes in the lungs (in abscess, bronchiectasis, lung gangrene, in tuberculosis process, is accompanied by the disintegration of tissue). Changes of sputum amount can help to access the course of inflammatory process in the lungs.

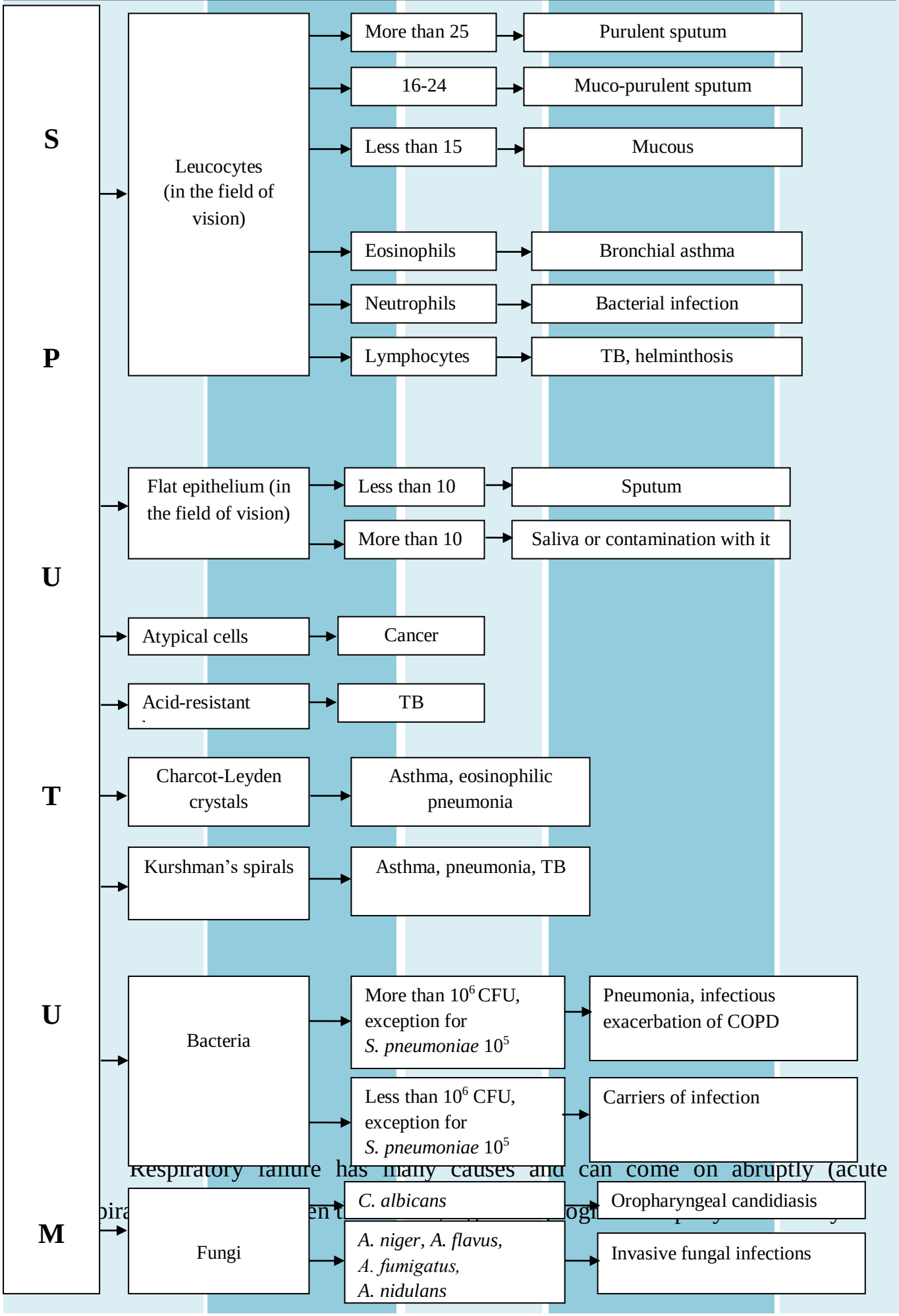
Color of sputum. Sputum is often colorless. Green tint may indicate joining of purulent inflammation, but in cases of acute bronchitis one should consider this issue individually. Different tints of red color indicate admixture of fresh blood while rusty – the traces of destruction of erythrocytes (crupous pneumonia and others). Grayish or blackish sputum contains coal dust and can be found in patients with pneumoconiosis or in smokers. Some drugs can change the color of the sputum (e.g. rifampicin).

Odor. Sputum is usually odorless. Putrid smell appears in cases of putrefactive infection (abscess, lung gangrene, putrid bronchitis, bronchiectasis, lung cancer complicated with necrosis). A peculiar "fruity" odor is specific for the drained cyst.

Nature of sputum. Mucoid sputum is observed in catarrhal inflammation of the respiratory tract, for example, in patients with acute and chronic bronchitis or tracheitis. Mucopurulent sputum is observed in bronchitis, pneumonia, bronchiectasis, tuberculosis. Purulent sputum is typical for purulent bronchitis, abscess, lung actinomycosis, gangrene. Bloody sputum is discharged in lung infarction, tumors, lung trauma, actinomycosis and other factors of bleeding in the respiratory organs.

Sputum consistency depends on the amount of phlegm and cellular elements, it can be liquid, thick or viscous.

ALGORITHM OF SPUTUM ANALYSIS



Respiratory failure has many causes and can come on abruptly (acute

ACUTE RESPIRATORY FAILURE
algorithm of emergency care

Acute respiratory failure (ARF) is a syndrome with signs of maximal tension of compensatory mechanisms, with failure of sufficient oxygen saturation of organs and systems and removal of CO_2.

At pre-hospital stage patient's state is assessed according to CABDE algorithm.

CABDE

C – Circulation

- evaluate color of the skin: pale, pink, blue or marble;
- evaluate temperature of the extremities: cold or worm;
- evaluate capillary filling (norm - up to 2 sec.);
- evaluate vein filling: moderately filled or suncken due to hypovolemia;
- evaluate heart rate and blood pressure;
- listen to heart sounds.

A – Airway

- identify symptoms of airway obstruction, violation of airway patency often contributes to paradoxical breathing and activation of additional respiratory muscles; central cyanosis is a late symptom of airway obstruction; in patients who are critically ill, impaired consciousness often causes airway obstruction (falling back of the tongue or soft palate);
- treatment of choice in these patients is therapy with high concentrations of oxygen (> 10 l / min), delivered trought mask with a reservoir.

B – Breathing

- identify symptoms that may indicate respiratory failure: excessive sweating, central cyanosis, work of additional muscles or abdominal breathing;
- determine the frequency of breathing movements (normal 12- 20 per minute);
- evaluate the manner of breathing, depth of breaths and symmetrical movements of the chest;
- conduct percussion and auscultation of the lungs;
- pay attention to excessive filling of neck veins (e.g. in severe asthma or tense pneumothorax), the availability and permeability of pleural drainage, etc.;
- determine position of the trachea.

D– Disability

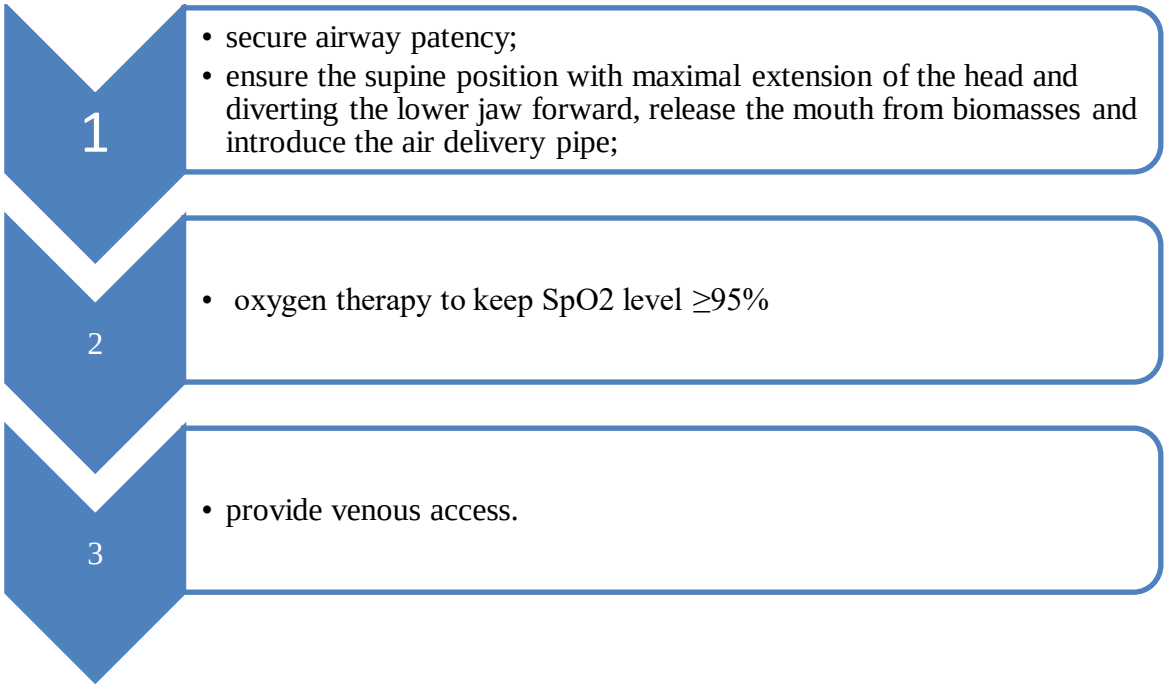
- assess pupils (diameter, symmetry and response to light);
- assess the state of consciousness of the patient by scale AVPU: Alert (oriented), Vocal (responds to voice), Pain (reacts to pain), Unresponsive (does not respond to any stimuli);
- determine the level of glucose to prevent hypoglycemia. If the glucose level is lower than 3 mmol / l, provide introduction of 50.0 ml of 20% glucose solution intravenously.

E – Exposure

- collect medical history from the patient or his/her relatives;
- study patient's medical records, check vital parameters and their changes in dynamics, check the list of prescribed medications and find out which of them he really takes.

ALGORITHM OF THERAPEUTIC MANAGEMENT IN ACUTE RESPIRATORY FAILURE

Pre-hospital stage



Hospital stage

Taking into account etiopathogenetic factors!

1	Anesthesia in cases of severe thoracic trauma or polytrauma (i.v. or i.m. administration of NSAIDs or narcotic analgesics); • ensure the supine position with maximal extension of the head and diverting the lower jaw forward, release the mouth from biomasses and introduce the air delivery pipe;
2	Oxygen therapy by means of nasopharyngeal mask, anesthesia-respiratory mask or endotracheal or tracheostomy tube;
3	Keep airways open; Prescribe bronchodilators and mucolytics
4	Stimulate respiratory center
5	Mechanical ventilation, in case of inefficient spontaneous breathing (RR 40 or more breaths per minute)

ALGORITHM OF THERAPEUTIC MANAGEMENT IN ACUTE RESPIRATORY FAILURE

Pre-hospital stage

Gastrointestinal bleeding is an acute or chronic leakage of blood into the lumen of the gastrointestinal tract in presence of pathological processes in the esophagus, stomach, small or large intestine.

First of all, the emergence of symptoms of this condition requires immediate emergency call.

1	• keep patient at complete rest; • transfer the patient to a horizontal position; • apply cold (wrapped ice) on the abdomen or give the patient to drink cold water
2	• ensure venous access to the cubital vein, if possible; • introduce i.v.calcium gluconate or chloride, 10% – 10,0; • introduce chloride solution of dycinone 12.5% – 5,0 and saline solution to restore the volume of circulating blood.

ALGORITHM OF THE FIRST AID FOR ACUTE HEPATIC COLIC

Acute hepatic colic is one of the manifestations of gallstone disease, which is characterized by attacks of pain in the right hypochondrium.

First of all, appearance of these symptoms requires immediate emergency call.

1	**Calm the patient;** **Ensure position on the right side, previously having put a warm heating pad;**
2	Introduce antispasmodic (no-spa, atropine) or anesthetic agents in parenteral form form: 0.1% atropine - 0.5-1.0 subcutaneously or nospanum 2% – 2,0–4,0 i./m. or analginum 50% – 2,0 i./m., baralgin – 5,0 i.m. or i.v.;
3	Hospitalization (to decide a question on treatment tactics)

Table 10

Differentiated approach to the treatment of patients with complicated hypertonic crises

Target organs damage	**First line of treatment**	**Aim of the therapy**	**Medications of choice**	**Medications strongly not recommended**
Acute hypertensive encephalopathy	Initial BP level > 140/90	Decrease of BP by 25% during 8 hours	labetalol, nicardipinum, esmololum	Nitroprussid, hydralazin
Acute ischemic stroke	In performing thrombolytic therapy SBP > 185 or DBP > 110 mm Hg.	Decrease and maintenance of SBP < 180 and DBP <105 during 24 hours	labetalol, nicardipinum, urapidilum, nitropaste	Nitroprussid
	Without thrombolytic therapy SBP >220 or DBP >120 mm Hg.	Decrease of medium BP by 10–15% in 2–3 hours, and by 15-25% during 24 hours	labetalol, nicardipinum, urapidilum, nitropaste	Nitroprussid
Hemorrhagic stroke	SBP >180 or medium BP >130 mm Hg.	If the intracranial pressure is not elevated (<25) – SBP <160 and medium BP<110 during 24 hours If intracranial pressure is elevated (>25) – SBP <180 medium BP <130 and perfusion pressure of the brain >60–80. Decrease of SBP to 140 mm Hg is considered safe	labetalol, nicardipinum, urapidilum, esmololum	Nitroprussid, hydralazin
Subarachnoid hemorrhage	SBP > 160 mm Hg.	Before surgery – decrease and	**labetalol, nicardipinum,**	Nitroprussid, hydralazin

Target organs damage	First line of treatment	Aim of the therapy	Medications of choice	Medications strongly not recommended
		keep SBP <140 mm Hg. After surgery – keep level of SBP <200	**urapidilum** **Nimodipine to all patients**	
Acute coronary syndrome	SBP > 160 or DBP > 100 mm Hg.	Decrease medium BP by 20-30 %	Beta-blockers, nitroglycerin	Nitroprussid, enalaprilat
Acute left ventricular failure	Initial BP level > 140/90	Decrease of medium BP by 20-30 %	Main - nitroglycerin/sodium nitroprussid + loop diuretics Alternative – enalaprilat, urapidil	Esmolol, metoprolol, labetalol
Aortic dissection	SBP >120 mm Hg.	SBP from 100 to 120 mm Hg, medium BP <80 mm Hg. (it is desirable to reduce HBR<60 per minute)	Esmolol/labetalol/ metoprolol (first line) or dilthiasem/ verapamil (if ß-bockers are contraindicated) + sodium nitroprussid, nikardypin, enalaprilat, urapidil (second line – if ß-bockers are ineffective)	Prescribe vasodilators before ß-blockers use
Intra- and postoperative hypertension	SBP or medium BP >20% form BP level before operation	Decrease of DBP by 10-15% or to 110 mm Hg in 30-60 min. In general decrease of medium BP not more than by 25%. Decrease of BP is on the background of moderate infusion therapy.	**Urapidil, labetalol, esmolol**	-

Target organs damage	**First line of treatment**	**Aim of the therapy**	**Medications of choice**	**Medications strongly not recommended**
	cardiosurgery - BP>140/90 mm Hg or medium BP>105 mm Hg.	Maintain SBP <140 or DBP <90 mm Hg.	urapidil, nitroglycerin, labetalol, esmolol, nitroprussid	ß-blockers are not recommended in concomitant cardiovascular insufficiency
Eclampsia	Seizures when BP ≥ 140/90 mm Hg in pregnant or parturient	Stop the seizures, provide open airways	Magnesium sulfate	ACE inhibitors
Hyper tonus of sympatic system (pheochromocytoma/ intoxication with cocaine, amphetamines, etc./ withdrawal of clonidine)	Initial BP level > 140/90 mm Hg.	Decrease of medium BP by 20-30%	Alpha-adrenoblocker (urapidil) Alternative: nitroglycerin/ nitroprusside sodium, verapamil	Beta-adrenoblockers without previous prescription of alpha blockers

Table 2

Medications for management of uncomplicated crises

Medication	**Dose and route of administration**	**Onset of action (min)**	**Side effects**
Nifedipine	10–20 mg orally or sublingually	15–30	Headache, tachycardia, reddening, angina pectoris
Captopril	12,5–50 mg per orally or sublingually	15–45	Hypotension in patients with renin-dependent hypertension
Prazosin	0,5–2 mg orally	30	Orthostatic hypotension
Propranolol	20–80 mg orally	30–60	Bradycardia, bronchial constriction
Dibazol	1% 3,0–5,0 i.v. or 4,0–8,0 i.m.	10–30	More effective in combination with other antihypertensive agents
Piroksan	1% 2,0–3,0 i.m.	15–30	Orthostatic hypotension
Diazepam	0,5% 1,0–2,0 i.m.	15–30	Dizziness, drowsiness
Furosemide	40–120 mg orally or i.m.	5–30	Orthostatic hypotension, weakness
Torasemide	10–100 mg orally or i.m.	5–30.	Orthostatic hypotension, weakness
Metoprolol	50–100 mg orally or 5–10 mg i.v. slowly	20–30 3–5	Bradycardia, bronchial constriction
Clonidine	0,01 % 0,5-2,0 i.m. 0,075-0,3 mg orally	30–60	Dry mouth, drowsiness. Contraindicated to patients with A-V blockade, bradycardia

Algorithm of emergency care in hypoglycemic coma

Clinical picture

- Feeling of hunger, trembling, sweating, diplopia after excessive insulin administration or oral hypoglycemic agents or excessive physical work, hunger.
- Excitement that passes into coma, pulse rate – normal, rapid or slow, blood pressure – normal or elevated, skin – moist, tonus of eyeballs is normal or elevated, urine output – normal.

Laboratory diagnosis

Hypoglycemia: plasma glucose <2.8 mmol/L.
Hypoglycemic coma – as a rule, <2.2 mmol/L.

Intensive care

- Put the patient in lateral position, release the mouth from residual food.
  In case of loss of consciousness it is not permitted to pour sweet solutions into the mouth (risk of asphyxia!).
- **Administer i.v. bolus of 40–100 ml of 40% glucose till full recovery of consciousness**. As an alternative – 1 ml of glucagon subcutaneously or intramuscularly.
- If after i.v. bolus of glucose the patient is still unconscious – start i.v. drop infusion of 5 or 10% of glucose and as soon as possible transfer the patient to ICU.
- If the cause of hypoglycemia is the overdose of long-acting hypoglycemic drugs, continue i.v. drop infusion of 5% or 10 % glucose till normalization of glycemia and full elimination of the drug from the body.

Algorithm of emergency care in ketoacidotic coma

Clinical features	Development of collapse, signs of cardiovascular insufficiency, skin cyanosis, tachycardia, atrial fibrillation, drop of blood pressure (cardiovascular or collaptoid version). • nausea, vomiting, abdominal pain and tension of the abdominal muscles (abdominal or pseudo-peritonitis version). • oligoanuria with severe urinary syndrome - proteinuria, hematuria, cylinderuria, hypostenuria (renal variant). • clinical picture of acute stroke due to intoxication, focal symptoms, asymmetry, loss of reflexes, hemiparesis, signs of cerebral edema (encephalopathic version).
Laboratory diagnostic	**Complete blood count**: leukocytosis <15,000 – stress, > 15,000 – infection; **Biochemical blood analysis**: hyperglycemia, hiperketonemia, ↑ creatinine (intermittent; due to transitory "pre- renal" failure caused by hypovolemia), transitory ↑of transaminases and CPK, Na^+ more often is normal, sometimes ↓ or ↑, K^+ more often is normal, sometimes ↓, • **Acid-alkaline ratio (AAR**): **decompensated metabolic acidosis.**

Algorithm of emergency care in hyperosmolar coma

Intensive care	**Insulin therapy** Mode of small doses of short-acting insulin (SAI) i.v. bolus, then i.v. by drops, regarding the level of blood glucose. If glycemia is 17–39 or ↑ - 0,1 U/kg/hr; If glycemia is from 11 to 17 – 0,05 U/kg/hr; If glycemia is lower than 11 – start with 4–6 U subcutaneously every 3-4 hours and add 5% glucose solution. The rate of blood glucose decrease is not more than 4 mmol/l /hr; on the first day of therapy do not decrease plasma glucose level less than 13–15 mg/dL.	**Rehydration**: 0,9% of sodium chloride; if plasma glucose is ≤ 13 mg/dL, 5% or 10% of glucose (+ 3.4 U of SAI for every 20 g of glucose); **Rehydration rate:** 1 l in the 1st hour, 0.5 liters - during 2nd and 3rd hours and by 0.25 liters during following hours. The total volume of infusion during the first 12 hours of treatment - not more than 10% of body weight. Rehydration rate is adjusted depending on CVP. If CVP <4 mm of water column- 1 liter/hr., 5–12 - 0.5 l/hr, >12 – 250–300 ml/hr.	**Restoration of electrolyte imbalance** If concentration of K^+ is known and in absence of renal dysfunction, start i.v. infusion of K^+ simultaneously with insulin therapy. If concentration of K^+ is unknown, start potassium infusion not earlier than in an hour after the start of insulin therapy, under the control of ECG and urine output.	**Correction of metabolic acidosis** Indications for administration of sodium bicarbonate: - blood **pH <7,0** or standard level of standard sodium bicarbonate <5 mmol/l. **-pH 6.9 – 7,0** - administer 4 g of sodium bicarbonate (200 ml of 2% solution i.v. slowly during 1 hour.) **-pH is< 6,9 -** administer 8 g of sodium bicarbonate (400 ml of 2% solution during 2 hours). **Without determining pH/AAR administration of sodium bicarbonate is contraindicated!**	Broad-spectrum antibiotics (high likelihood of infections as the cause of DKA). **Nutrition** After full recovery of consciousness, ability to swallow, in the absence of nausea and vomiting – small portions with additional subcutaneous injection of 1–2 units of SAI per 1 of BU. In 1–2 days from the start of food intake, without signs of gastrointestinal pathology – patient can proceed to a normal diet.

<table>
<tr><td>Clinical features</td><td colspan="4">Polymorphic neurological symptoms (seizures, dysarthria, bilateral spontaneous nystagmus, hyper- or hypotonia of muscles, paresis and paralysis, hemianopsia, vestibular disorders, etc.). Those symptoms which fail any distinct syndrome; they are variable and disappear when the osmolality is normalized.</td></tr>
<tr><td>Laboratory diagnostic</td><td colspan="4">•Complete blood count: leukocytosis <15,000 – stress, > 15,000 – infection;
•Urinalysis: presence of high levels of glucose and protein (intermittent); absence of ketones;
• Clinical blood analysis: extremely high hyperglycemia, absence of ketones, osmolarity of plasma> 320 mosml/L ,↑ creatinine (intermittent); the level of Na^+ ↑, the level of K^+ is normal, sometimes ↓, in CRF maybe ↑.
• AAR: no acidosis: pH> 7,3, sodium bicarbonate> 15 mmol/l, anion gap <12 mg/dL.</td></tr>
<tr><td>Intensive care</td><td>Rehydration
In the first hour – 1liter of 0, 9% NaCl, then – depending on the level of Na^+:
-in adjusted Na^+>165 mmol/L: saline solutions are contraindicated.
-in decrease of adjusted Na^+ to <145 mmol/l switch to 0.9% NaCl;
- in case of hypovolemic shock (blood pressure <80/50 mm Hg.) start with very quick infusion of 1 liter of 0.9% NaCl or colloidal solutions.
Rehydration rate: 1st hour – 1–1,5 liter, 2nd and 3rd hour – by 0,5–1 l, followed by 0,25–0,5 l (on the background of controlled CVP).</td><td>Insulin therapy
In early stages of infusion therapy insulin is not administered or administered in very small doses – 0,5-2 U/hr. i.v., maximum 4 U/h i.v.
If hyperglycemia remains for 4–5 hours, switch to insulin dosing regimen recommended for the treatment of DKA.
Plasma glucose level should not be decreased faster than 4 mmol/l/hr. and serum osmolarity – not more than by 3 mOsm/l/hr.</td><td>Correction of electrolyte imbalance
If concentration of K^+ is known and in absence of renal dysfunction, start i.v. infusion of K^+ simultaneously with insulin therapy.
If concentration of K^+ is unknown, start potassium infusion not earlier than in an hour after the start of insulin therapy, under the control of ECG and urine output.</td><td>Broad-spectrum antibiotics
(high likelihood of infections).
Nutrition
After full recovery of consciousness, ability to swallow, in the absence of nausea and vomiting – food intake by small portions with additional subcutaneous injection of 1-2 units of SAI per 1 of BU. After 1-2 days from the start of food intake, without signs of gastrointestinal pathology – patient can proceed to a normal diet.</td></tr>
</table>

Algorithm of first aid in lactacidemia coma				
Clinical features	Nausea, vomiting, muscle and chest pain, drowsiness, lethargy, Kussmaul's breathing without smell of acetone, sharp decrease in blood pressure, tachycardia, oliguria or anuria, history of treatment with biguanides combined with diseases that are accompanied by hypoxia.			
Laboratory diagnostic	• **Biochemical blood test**: **lactate> 4,0 mmol/L, sometimes - 2,2 –4 mmol/l;** any glycemia: more often - hyperglycemia; often– ↑ creatinine, hyperkalemia. • **AAR: decompensated metabolic acidosis: pH <7,3, level of serum sodium bicarbonate ≤ 18 mg/dL, anion gap of more than 10 mg/dL.**			
Intensive care	**To reduce production of lactate:** short-acting insulin **by 2–5 U/hr. i.v.** (regimen of infusion is the same as in DKA), 5% glucose solution by 100–125 ml/hr.	**To remove excess of lactate and biguanides (if used):** hemodialysis with lactate-free buffer; in acute overdose of metformin - activated carbon or another sorbent internally.	**To restore AAR :** mechanical ventilation in hyperventilation mode to eliminate the excess of CO_2 (target: pCO_2 25–30 mm Hg.); administration of sodium bicarbonate - only in pH <7.0, not more than 100 ml of 4% solution once, i.v. slowly, followed by increasing ventilation, to remove excess of CO_2 (produced in administration of sodium bicarbonate).	**Shock control and hypovolemia** According to the general principles of intensive therapy. Infusion of 0,9% of NaCl solution i.v. at a rate of 1 l/hr.