Federal budgetary educational establishment of higher education

Ulyanovsk State University

The Institute of medicine, ecology and physical culture

Smirnova A.Yu., Gnoevykh V.V.

Basis of functional and laboratory diagnostics

Textbook of Medicine for medicine faculty students

Ulyanovsk, 2017
Main goal of this textbook is the practical assistance for students in the development of the fundamentals of clinical diagnosis of diseases of internal organs. It contains a description of the main methods of laboratory and instrumental diagnostic tests of diseases of internal organs. The publication is illustrated with charts, drawings and tables. The textbook is intended for students of medical universities.

Smirnova A.Yu., Gnoevykh V.V., 2017
Ulyanovsk State University, 201
# THE CONTENTS OF A TEXT BOOK

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Electrocardiography examination</td>
<td>5</td>
</tr>
<tr>
<td>Algorithm of interpreting ECG. Electrophysiological bases of ECG</td>
<td>5</td>
</tr>
<tr>
<td>(electrocardiography)</td>
<td></td>
</tr>
<tr>
<td>ECG in hypertrophy of atriums</td>
<td>14</td>
</tr>
<tr>
<td>ECG Hypertrophy of ventricles</td>
<td>16</td>
</tr>
<tr>
<td>ECG in ischemic heart disease (IHD)</td>
<td>17</td>
</tr>
<tr>
<td>ECG diagnosis of sinus node automatism disorders, disordered myocardial</td>
<td>25</td>
</tr>
<tr>
<td>conduction and excitability. Cardiac arrhythmia.</td>
<td></td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
<td>26</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>26</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>27</td>
</tr>
<tr>
<td>Sinus (respiratory) arrhythmia</td>
<td>28</td>
</tr>
<tr>
<td>Ectopic arrhythmias</td>
<td>29</td>
</tr>
<tr>
<td>Extrasystolic arrhythmia (Ectopic beats, Extrasystoles)</td>
<td>29</td>
</tr>
<tr>
<td>Paroxysmal tachycardia (PT)</td>
<td>35</td>
</tr>
<tr>
<td>Ventricular flutter and fibrillation</td>
<td>37</td>
</tr>
<tr>
<td>Ciliary arrhythmia</td>
<td>38</td>
</tr>
<tr>
<td>Conduction disorders (heart blocks)</td>
<td>42</td>
</tr>
<tr>
<td>II. The study of gas composition and blood acid-base status (Astrup</td>
<td>51</td>
</tr>
<tr>
<td>microtechnique).</td>
<td></td>
</tr>
<tr>
<td>III. Spirometry</td>
<td>55</td>
</tr>
<tr>
<td>Airflow obstruction syndrome</td>
<td>64</td>
</tr>
<tr>
<td>Respiratory deficiency syndrome</td>
<td>66</td>
</tr>
<tr>
<td>IV. Laboratory tests</td>
<td>69</td>
</tr>
<tr>
<td>Test</td>
<td>Page</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>Sputum test</td>
<td>69</td>
</tr>
<tr>
<td>Complete blood count</td>
<td>73</td>
</tr>
<tr>
<td>Coagulation disorders</td>
<td>86</td>
</tr>
<tr>
<td>Routine biochemistry</td>
<td>89</td>
</tr>
<tr>
<td>Jaundice</td>
<td>89</td>
</tr>
<tr>
<td>Cytolytic syndrome</td>
<td>93</td>
</tr>
<tr>
<td>Laboratory Diagnosis Of Acute Myocardial Infarction</td>
<td>93</td>
</tr>
<tr>
<td>Diagnosis of diabetes mellitus</td>
<td>96</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>97</td>
</tr>
<tr>
<td>Kidneys functional tests</td>
<td>102</td>
</tr>
<tr>
<td>Urine test by Zimnicky</td>
<td>102</td>
</tr>
<tr>
<td>Methods of revealing glomerular filtrating rate</td>
<td>103</td>
</tr>
<tr>
<td>Tests to detect <em>H. pylori</em></td>
<td>104</td>
</tr>
<tr>
<td>Measurement of Pancreatic Enzymes</td>
<td>105</td>
</tr>
<tr>
<td>V. Tests</td>
<td>107</td>
</tr>
<tr>
<td>VI. Examples of interpretation of blood tests</td>
<td>131</td>
</tr>
<tr>
<td>VII. Tasks to consolidate the material</td>
<td>133</td>
</tr>
</tbody>
</table>
I. Electrocardiography examination

Algorithm of interpreting ECG. Electrophysiological bases of ECG (electrocardiography)

Electrocardiography is a method of graphic recording of electric currents generated in the working heart. Contractions of the heart are preceded by its excitation during which physicochemical properties of cell membranes change along with changes in the ionic composition of the intercellular and intracellular fluid, which is accompanied by generation of electric current.

Electrophysiological functions of heart:
- Automaticity – function of pacemaker cells to produce spontaneously the action potential (transient depolarization);
- Conduction - capability to impulse propagation through cardiac tissues;
- Excitability – capability to become excited under the influence of impulses;
- Refractoriness is a property of cardiac cells that defines the period of recovery that cells require before they can be reexcited by a stimulus;
- Contractility – capability of myocardium to contract in response to excitement.

Cardiac conduction system.

The depolarization stimulus for the normal heartbeat originates in the sinoatrial (SA) node or sinus node, a collection of pacemaker cells. These cells fire spontaneously; that is, they exhibit automaticity. Pacemaker cells exhibit automaticity in all departments of conduction system: I- sinus node (SA), II - AV junction (and atrial fibres) and AV node and His-bundle), III- His-bundle branches, Purkinje fibers.

The first phase of cardiac electrical activation is the spread of the depolarization wave through the right and left atria, followed by atrial contraction. Next, the impulse stimulates pacemaker and specialized conduction tissues in the atroventricular (AV) nodal and His-bundle areas; together, these two regions constitute the AV junction. The bundle of His bifurcates into two main branches, the right and left bundles, which rapidly transmit depolarization wavefronts to the
right and left ventricular myocardium by way of Purkinje fibers. The main left bundle bifurcates into two primary subdivisions, a left anterior fascicle and a left posterior fascicle. The depolarization wave fronts then spread through the ventricular wall, from endocardium to epicardium, triggering ventricular contraction. Ventricular depolarization can be divided into two major phases, each represented by a vector. The first phase denotes depolarization of the ventricular septum, beginning on the left side and spreading to the right. Simultaneous depolarization of the left and right ventricles (LV and RV) constitutes the second phase.

Table 1

<table>
<thead>
<tr>
<th>Leads</th>
<th>Position of an electrode</th>
<th>Projection of heart chambers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>Right arm - left arm</td>
<td>Anterior wall of left ventricle</td>
</tr>
<tr>
<td>II</td>
<td>Right arm - left foot</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Left foot - left arm</td>
<td>Posterior wall of left ventricle and right ventricle</td>
</tr>
<tr>
<td>Augmented leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>aVL</td>
<td>Left arm</td>
<td>Anterior wall of left ventricle</td>
</tr>
<tr>
<td>aVR</td>
<td>Right arm</td>
<td></td>
</tr>
<tr>
<td>aVF</td>
<td>Left foot</td>
<td>Posterior wall of left ventricle and right ventricle</td>
</tr>
<tr>
<td>Chest leads</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V1</td>
<td>right sternal border- the 4th intercostal space</td>
<td>Anterior wall of right and left ventricle</td>
</tr>
<tr>
<td>V2</td>
<td>left sternal border- the 4th interspace</td>
<td>Anterior part of interventricular septum</td>
</tr>
<tr>
<td>V3</td>
<td>between V2 and V4</td>
<td>Anterior wall of left ventricle to apex</td>
</tr>
<tr>
<td>V4</td>
<td>left midclavicular line – the 5th interspace</td>
<td>Apex of left ventricle</td>
</tr>
<tr>
<td>V5</td>
<td>left anterior axillary line – the 5th interspace</td>
<td>Side wall of left ventricle</td>
</tr>
<tr>
<td>V6</td>
<td>left midaxillary line – the 5th interspace</td>
<td>Side wall of left ventricle</td>
</tr>
</tbody>
</table>

Recording ECG Twelve-lead ECG recording has gained wide use: three standard leads (classical), six chest, and three augmented unipolar limb leads (Table 1). Special leads are also used in some cases. The six frontal plane and six horizontal plane leads provide a threedimensional representation of cardiac
electrical activity. The frontal plane leads - standard and augmented leads. The horizontal plane leads – chest leads.

**Normal ECG Basic ECG waves and intervals**

During diastole the heart does not generate current and an electrocardiograph records a straight line which is called isoelectric. Action current is represented by a specific curve.

An ECG of a healthy subject has the following elements:

1) positive waves P, R, and T, negative waves Q and S; the positive wave U is accidental;

2) P-Q, S-T, T-P, and R-R intervals;

3) QRS and QRST complexes (Table 2, Fig. 1-3).

Each of these elements characterizes the time and sequence of excitation of various parts of the myocardium. Generation of ECG waves and intervals: P – depolarization of atriums, QRS - depolarization of ventricles, ST,T,U - repolarization of ventricles. The QRS-T cycle corresponds to different phases of the ventricular action potential.

Fig. 1. The six frontal plane and six horizontal plane leads provide a three-dimensional representation of cardiac electrical activity. The frontal plane leads – standard (I, II, III) and augmented leads (aVR, aVF, aVL). The horizontal plane leads – chest leads (V₁-₆).
Fig. 2. Normal ECG of the healthy subject. Sinus rhythm is present with a heart rate of 75 per minute. PR interval is 0.16 s; QRS interval (duration) is 0.08 s; QT interval is 0.36 s; the mean QRS axis is about +70°, transition zone V3.

Fig. 3. Basic waves and intervals of normal ECG.

Table 2

Waves and intervals of normal ECG
In normal conditions, the cardiac cycle begins with excitation of the atria (P wave on an ECG). The ascending portion of the P wave is mainly due to excitation of the right atrium, while the descending one of the left atrium. The wave is small, and its normal amplitude does not exceed 1-2 mm; the length is 0.08-0.1 s. The P wave is followed by a straight line lasting to Q wave; if this wave is small, the line extends to the R wave. This is the P-Q interval. It extends from the beginning of the P wave to the beginning of the Q (or R) wave and corresponds to the time from the beginning of atrial excitation to the beginning of ventricular excitation, i.e. includes the time of pulse propagation in the atria and its physiological delay in the atrioventricular node. The normal length of the P-Q interval is 0.12-0.18 s (to 0.20 s). Excitation of the ventricles corresponds to the QRS complex. Its waves vary in size and are different in various leads. The length of the QRS complex (measured from the beginning of the Q wave to the end of the 5 wave) is 0.06—0.1 s. This is the time of intraventricular conduction. The first wave of this complex is the negative Q wave. It corresponds to excitation of the interventricular septum. Its amplitude is small and does not normally exceed 1A amplitude of the R wave; the length of the Q wave does not exceed 0.03 s. The Q wave may be absent on an ECG. The R wave corresponds to almost complete excitation of both ventricles. It is the highest wave of the ventricular complex; its amplitude varies from 5 to 15
mm. The negative 5 wave is recorded in full excitation of the ventricles; usually it is not high, actually not exceeding 6 mm (2.5 mm on the average). Sometimes the 5 wave is very small. At the moment of complete depolarization of the myocardium, the potential difference is absent and the ECG is therefore a straight line (the S-T interval.) The length of this interval varies greatly depending on the cardiac rhythm; the S-T interval may be displaced from the isoelectric line to not more than 1 mm. The T wave corresponds to the repolarization of the ventricular myocardium. The T wave is normally asymmetrical: the gradual ascent converts into a rounded summit, which is followed by an abrupt descent. Its amplitude varies from 2.5 to 6 mm, the length from 0.12 to 0.16 s. A small positive U wave sometimes follows the T wave in 0.02—0.04 s. Its amplitude exceeds 1 mm in rare cases: the length is 0.09—0.16 s. The origin of the U wave is disputed. The Q-T interval (QRST complex) shows the time of excitation and recovery of the ventricular myocardium i.e. it corresponds to their electrical system. It extends from the beginning of the Q wave (or the R wave, if the Q wave is absent) to the end of the T wave. Its length depends on the rate of cardiac contractions: in accelerated heart rhythm the Q-T interval shortens. The Q-T interval in women is longer than in men (at the same heart rate). For example, at the rate of 60-80 beats per minute, the length of the Q-T interval in men is 0.32-0.37 s and in women—0.35-0.40 s. The T-P interval (from the end of the T to the beginning of the P wave) corresponds to the electrical diastole of the heart. It is located on the isoelectric line because all action currents are absent at this moment. Its length depends on the cardiac rhythm: the faster the heart rate the shorter the T-P interval. The R-R interval is a distance between the summits of two neighbouring R waves. It corresponds to the time of one cardiac cycle, whose length depends on the cardiac rhythm as well.

Interpretation of ECG
In the beginning of interpretation of ECG technical conditions of tape recording must be defined (voltage of the ECG and speed of a tape). The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis (Fig. 4). It is important for correct estimation of heart rate, amplitude and duration of ECG waves and intervals (Fig. 5).

Fig. 4. The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis.

![ECG Graph Paper](image)

Fig. 5. HR determination - multiply in 20 times (.20) the number of R-R intervals during 3 seconds.

The sequence of ECG analysis:

1. Voltage of the ECG is estimated in compliance with standard size of 1 mv =10 mm. To that end, the amplitude of R waves is measured in standard leads. Normal amplitude is 5-15 mm. If the amplitude of the highest R wave does not
exceed 5 mm in standard leads, or the sum of amplitudes of these waves in all three leads is less than 15 mm, the ECG voltage is considered decreased.

2. Speed of tape. If speed of tape is 50 mm/min – 0.02 s in 1 mm of tape (width of QRS=3-4 mm). If speed of tape 25 mm/min – 0.04 s in 1 mm of tape (width of QRS=1-2 mm).

3. Regularity and pacemaker of the cardiac rhythm. Since the sinoatrial node is the pacemaker of a normal heart, and the excitation of the ventricles is preceded by excitation of the atria, the P wave should come before the ventricular complex. The R-R intervals should be equal. Its fluctuations normally do not exceed 0.1 s. Greater variations in the length of the R-R intervals indicate disordered cardiac rhythm. Sinus rhythm signs - P-wave positive in II standard lead and corresponds (previous) to complexes QRS.

4. Heart rate (HR) 60 (seconds in 1 minutes)/ [R-R] (in seconds, i.e. to - in divisions x 0.02)

To that end, duration of one cardiac cycle (the R-R interval) and the number of such cycles in one minute length should be determined. For example, if one cycle lasts 0.8 s, there will be 75 such cycles in a minute (60 : 0.8 = 75). If the cardiac rhythm is irregular, the length of five or ten R-R intervals is determined, the mean R-R interval found, and the cardiac rate is finally determined as for regular cardiac rhythm. Lengths of the maximum and minimum R-R intervals are given in parentheses. Other variant of HR determination - multiply in 20 times (×20) the number of R-R intervals during 3 seconds (Fig. 5).

5. Analysis of myocardial conduction depending duration of: - P wave (≤0.10-0.12 s) – intraatrial conduction; - PQ interval (0.12-0.20 s) – conduction in atrioventricular node; - QRS complex (0.06-0.10 s) - intraventricular conduction.

6. The position of the electrical axis of heart changes with changes of the position of the heart in the chest. The relation between the electrical axis and the magnitude of the QRS complexes in standard leads is described by the so-called Einthoven triangle. The electrical axis of the heart is determined by the shape of ventricular complexes in standard leads: - normal electrical axis - RII> RI> RIII;
- horizontal electrical axis (levogram) - RI> RII> RIII;  - vertical electrical axis (dextrogram) - RIII> RII> RI.

7. Analysis of waves and intervals. The length and size of ECG elements (P wave, R-Q interval and QRST complexes) are then determined in those leads where the waves are the largest (usually in lead II). Moreover, the direction of the P and T waves is determined (they can be positive and negative). Smaller and split waves can be present as well. Additional waves can appear. The shape of the ventricular complex in all leads is thoroughly examined, character of the S-T interval is noted (see Fig.6). The length of the QRST complex (Q-T interval) depends on rate: the higher the rate, the shorter the interval. The ECG of healthy persons depend on their age and constitution, on the posture at the moment of taking an ECG (sitting, lying), on the preceding exercise, etc. ECG may change during deep breathing (the position of the heart in the chest is changed during deep inspiration and expiration), in increased tone of the sympathetic and parasympathetic nervous systems and in some other conditions. It is difficult to overestimate the clinical importance of electrocardiography. It is used to reveal disorders of heart activity, enlargement of heart chambers and to diagnose coronary circulatory disorders.

Fig.6. The shape of the ventricular complex  

13
**ECG in hypertrophy of atriums**

Hypertrophy of auricles is determined by changes of P-wave.

Left atrium hypertrophy is detected by “P mitrale”: wide (>0.1 s), splitted P-wave in I, II, AVL, left chest leads (V5-6); flat or negative P in III, biphasic or negative (>1 mm) P V1 (Fig. 7). Left atrium hypertrophy is typical in mitral valves diseases (mitral stenosis and mitral incompetence).

![ECG in hypertrophy of atriums](image)

**Fig. 7. ECG in hypertrophy of atriums.**

Left atrium hypertrophy is detected by “P mitrale”: wide (>0.1 s), splitted P-wave in I, II, AVL, V5-6; flat or negative P in III, biphasic or negative P (>1 mm) V1.
Right atrium hypertrophy

Right atrium hypertrophy is typical in chronic pulmonary diseases (pulmonary heart) and tricuspid valves incompetence.

Right atrium hypertrophy is detected by “P pulmonale”: high (>2.5 mm) acute P in II, III, AVF and right chest leads (V1-2) (Fig. 7,9).
**ECG Hypertrophy of ventricles**

Hypertrophy of ventricles is determined mainly by changes of ventricular complex QRS (Fig. 10). Ventricular hypertrophy causes the following changes in ECG:

1) the position of the electrical axis is changed: in left-ventricular hypertrophy - levogram, in right-ventricular hypertrophy dextogram;

2) the amplitude of the ventricular complex and its length increase,

3) changed terminal part of the ventricular complex of the ECG because of repolarization abnormalities: ST-segment depression and T-wave inversion in leads with a prominent R wave;

4) in left-ventricular hypertrophy - amplitude of the S wave increases in V1-2; amplitude of the R wave increases >20-25 mm in V4-6 sum of amplitude SV1 + RV5 (or RV6) ≥35 mm (Fig.11);

5) In right-ventricular hypertrophy the changes in the S and R waves are the reverse: high R wave ≥ 7 mm appears in V1-2, deep S wave in V4-6 (Fig.12).

![Fig. 10. ECG in hypertrophy of ventricles.](image)

Left-ventricular hypertrophy (LVH) - amplitude of the S increases in V1-2; amplitude of the R increases >20-25 mm in V4-6. Right-ventricular hypertrophy (RVH) the changes in the S and R waves are the reverse - high R wave ≥ 7 mm in V1-2, deep S wave in V4-6.
ECG in ischemic heart disease (IHD)

Acute ischemia of myocardium causes a current of injury of myocardium:

- **Subendocardial ischemia** - the resultant ST vector directs toward the inner layer of the affected ventricle. Overlying leads therefore will record ST depression.
- **Transmural or epicardial ischemia** - the ST vector is usually shifted in the direction of the outer (epicardial) layers, producing ST elevations and sometimes,
in the earliest stages of ischemia, tall, positive so-called hyperacute T waves over the ischemic zone.

**ECG in myocardial infarction**

Electrocardiographic examination is especially important. It establishes the presence of myocardial infarction and also some important details of the process such as localization, depth of the process, and the size of the affected area.

Three zones of myocardial damage in acute myocardial infarction can be detected by ECG: necrotic zone, ischemic myocardium injury zone, and zone of ischemia.

Myocardial necrosis is detected by pathological Q-wave:

Pathological Q-wave is characterized by width $\geq 0.04$ s (in V4-6 $>0.025$ s), depth $>2$ mm or $>1/4$ R-wave (in V4-6 $>15\%$R) (Fig.13).

Ischemic myocardium injury is detected by ST-interval:

- Transmural or epicardial injury
  - convexing elevation ST with transmission in T-wave (Fig.13);
  - Subendocardial injury – horizontal or concaving depression ST.

Ischemia of myocardium is detected by T-wave:

- Subendocardial ischemia – symmetrical acute high T-wave in overlying leads ($>6$ mm in standard and augmented leads, $>8$-10 mm in chest leads) (Fig.13);

- Transmural or epicardial ischemia - symmetrical acute deep T-wave. The S-T segment and T wave change during the first hours of the disease (Table 3). The descending limb of the R wave transforms into the S-T segment without reaching the isoelectric line. The S-T segment rises above the isoelectric line to form a convexing arch and to coincide with the T wave. A monophase curve is thus formed. These changes usually persist for 3—5 days. Then the S-T segment gradually lowers to the isoelectric line while the T wave becomes negative and deep.
Fig. 13 Cardiac infarction zones

<table>
<thead>
<tr>
<th>STAGE</th>
<th>DURATION</th>
<th>SIGN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage of ISCHEMIA</td>
<td>to 30 min</td>
<td>high pointed (coronary) T wave</td>
</tr>
<tr>
<td>PREACUTE phase</td>
<td>up to 3 days</td>
<td>ST above contour</td>
</tr>
<tr>
<td>ACUTE stage</td>
<td>to 3 weeks</td>
<td>ST above contour with the transition in (-) T; the formation of pathological Q;</td>
</tr>
<tr>
<td>SUBACUTE stage</td>
<td>to 3 months</td>
<td>ST on the contour, T (-), pathological Q;</td>
</tr>
<tr>
<td>SCAR stage</td>
<td>the rest of life</td>
<td>ST on the contour, T (-) or (+), pathological Q</td>
</tr>
<tr>
<td>(final consolidation of)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A deep Q wave appears, the R wave becomes low or disappears at all. The QS wave is then formed, whose appearance is characteristic of transmural infarction. Depending on localization of infarction, changes in the ventricular complex are observed in the corresponding leads (Table 4, Fig. Suppl. 14-21). The initial shape of ECG can be restored during cicastrization, or the changes may remain for the rest of life.

**Table 10**

Localization of myocardial infarction

<table>
<thead>
<tr>
<th>Pathological changes of ventricular complex (Q-wave)</th>
<th>Localization of myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1-4, I, aVL</td>
<td>Anterior wall of LV</td>
</tr>
<tr>
<td>V1-2</td>
<td>Anterior part of interventricular septum</td>
</tr>
<tr>
<td>I, aVL, V5-6</td>
<td>Lateral wall of LV</td>
</tr>
<tr>
<td>II, III, aVF</td>
<td>Posterior (inferior diaphragmatic) wall of LV</td>
</tr>
<tr>
<td>High R V1-2</td>
<td>Posterior (superior basal) wall of LV</td>
</tr>
</tbody>
</table>
Fig. 15 Anterior infarction

Fig. 16 Anterior infarction
ST elevation in leads II, III and aVF, and often ST depression in I, aVL, and precordial leads are signs of an inferior (lower) infarction. Inferior infarctions may occur due to occlusion of the right circumflex coronary arteries resulting in infarction of the inferior surface of the left ventricle, although damage can be made to the right ventricle and interventricular septum. This type of infarction often results in bradycardia due to damage to the atrioventricular node.
Fig. 19. Lateral infarction

Occlusion of the left circumflex artery may cause lateral infarctions. Lateral infarctions are diagnosed by ST elevation in leads I and aVL.

Fig. 20. Infarction of the inferior and lateral wall
Fig. 21. Inferior infarction

Variability of ECG patterns with acute myocardial ischemia:
- Non–infarction subendocardial ischemia – transient ST depressions;
- Non–infarction transmural ischemia - transient ST elevation or paradoxical T-wave normalization, sometimes followed by T-waves inversions;
- Non–Q-wave (non-ST elevation) infarction - ST depressions or T-inversions without Q-wave;
- Q-wave – infarction - Q-wave with hyperacute T-waves/ST-elevations followed by T-waves inversions.

Exercise stress ECG testing: Because the diagnosis of angina pectoris is usually primarily based on the patient's history, exercise testing in a patient with typical symptoms is generally used to determine functional and ECG response to graded stress (for exercise stress testing using radionuclide imaging; for exercise testing in asymptomatic persons to determine fitness for exercise programs, see below). The patient exercises to a predetermined goal (e.g., 80 to 90% of maximal heart rate, which can be approximated as 220 less the age in years), unless distressing cardiovascular symptoms (dyspnea, reduced endurance, fatigue, hypotension, or chest pain) supervene. The ischemic ECG response during or after exercise is characterized by a flat or downward-sloping ST segment depression >0.1 millivolts (1 mm on the ECG when properly calibrated) lasting >0.08 sec.
ECG diagnosis of sinus node automatism disorders, disordered myocardial conduction and excitability. Cardiac arrhythmia.

Arrhythmias are deviations from the normal rhythm of the heart. Cardiac arrhythmias are common in many organic and functional disorders of circulatory system.

Causes of cardiac arrhythmias include:

1) Affected automaticity of the sinus node;
2) Foci of increased activity in the myocardium can generate impulses to initiate heart contractions apart sinus node (ectopic arrhythmia);
3) Disorders of cardiac conduction system, local conduction disorder (re-entry mechanism);
4) Combined changes in several functions of the heart such as automaticity, excitability, conduction or contractility.

Re-entry mechanism according to up-to-date view of point is a most common cause of cardiac arrhythmia. Re-entry mechanism means a pathological circuit of impulse because of:

1) electrophysiological inhomogeneity (i.e., differences in conduction and/or refractoriness) in two or more regions of the heart connected with each other to form a potentially closed loop;
2) unidirectional block in one pathway;
3) slow conduction over an alternative pathway, allowing time for the initially blocked pathway to recover excitability;
4) reexcitation of the initially blocked pathway to complete a loop of activation.

Classification of cardiac rhythm disorders (arrhythmias):

1. Arrhythmias associated with altered automaticity of the sino-atrial node (sinus arrhythmia).
2. Ectopic (heterotopic) arrhythmias.
3. Arrhythmias due to disordered myocardial conduction (heart blocks).
4. Combined cardiac rhythm disorders.
**Sinus arrhythmia**

Sinus arrhythmia - arrhythmias associated with altered automaticity of the sinus (sinoatrial) node. When automaticity of the sino-atrial node is upset, the rate of impulse generation may either accelerate (sinus tachycardia) or slow down (sinus bradycardia), or the sequence of impulses may be changed with their generation at irregular intervals (sinus arrhythmia). Normal sinus rhythm characteristics (Fig. Suppl. 13): - HR (heart rate) equals 60-80 in min, regular rhythm (differences between minimal and maximal R-R intervals is not more than 15%); - P wave - positive in I, II, aVF, P wave – negative in aVR, PQ ≥0.12 s.

**Sinus tachycardia**

Sinus tachycardia is directly connected with effects of biologically active substances which increase excitability of the sinoatrial node. This phenomenon may also depend on the change in the tone of the vegetative nervous system. It develops with intensified effect of the sympathetic nervous system. The rate of cardiac contractions in sinus tachycardia usually varies from 90 to 120 and sometimes to 150-160 per min. Sinus tachycardia develops during meals, physical exertion and emotional stress. At elevated body temperature, the heart rate increases by 8-10 per min per each degree over 37 °C. Sinus tachycardia is a frequent symptom of myocarditis, heart defects, and other diseases. It develops by reflex mechanism in heart failure and in response to the increased pressure in the orifices of venae cavae. Tachycardia often develops in neurosis, anemia, hypotension, and in many infectious diseases and toxicosis; it can be provoked by some pharmacological preparations (adrenaline, caffeine, atropine sulphate, etc.), and in thyrotoxicosis.

The clinical signs of sinus tachycardia are heart palpitation and accelerated pulse. ECG signs of sinus tachycardia (Fig. 22):

- acceleration of heart rate from 90-100 up to 160-180 in one minute;
- P-wave of the normal form precedes complex QRS; - regular accelerated rhythm (all intervals R-R are identical).
Sinus bradycardia

Sinus bradycardia is connected with slowed excitation of the sino-atrial node, which in turn depends mostly on the increased influence of the parasympathetic nervous system on the heart (or decreased influence of the sympathetic nervous system). Automaticity of the sino-atrial node decreases in sclerotic affections of the myocardium and in the cold. The cardiac rate in sinus bradycardia decreases to 50-40 (in rare cases to 30) beats per min, Bradycardia may occur in well-trained athletes. It is not permanent and the heart rhythm is accelerated during exercise as distinct from pathological bradycardia in atrioventricular block when bradycardia persists during and after exercise. If automaticity of the sino-atrial node sharply decreases (sicksinus syndrome), the second- or third-order centres may function as the pacemaker, i.e. ectopic arrhythmias develop (see below). Sinus bradycardia occurs in increased intracranial pressure (tumour and edema of the brain, meningitis, cerebral hemorrhage), in myxedema, typhoid fever, jaundice, starvation, lead and nicotine poisoning, and due to effect of quinine and digitalis preparations. It may develop by reflex during stimulation of baroreceptors of the carotid sinus and the aortic arch in essential hypertension, and can be provoked by pressure on the eye-ball (Dagnini-Aschner reflex), or by irritation of receptors of the peritoneum and the internal organs. Mild bradycardia is not attended by any subjective disorders, nor does it produce any effect on the circulation. Marked bradycardia (under 40 beats per min) may cause nausea and loss of consciousness due to cerebral anemia. Objective examination reveals slow pulse. ECG signs of sinus bradycardia (Fig. 23):
- decrease of a heart rate less than 60 in one minute;
- P-wave has a normal form;
- regular infrequent rhythm.
**Sinus (respiratory) arrhythmia**

Sinus arrhythmia characterized by irregular generation of impulses is due to variations in the tone of the vagus. It would commonly be associated with respiratory phases (respiratory arrhythmia): the cardiac rhythm accelerates during inspiration and slows down during expiration. Sinus arrhythmia is observed in children and adolescents (juvenile arrhythmia), in patients convalescing from infectious diseases, and in certain diseases of the central nervous system. It can be a sign of pathology in rare cases when arrhythmia is not connected with respiration or when it develops in the aged during normal respiration. Clinically sinus arrhythmia is not attended by any subjective disorders.

ECG signs of sinus arrhythmia (Fig.24):

- different intervals R-R;
- P always precedes complex QRS;
- cardiac rhythm accelerates during inspiration and slows down during expiration.
Additional (heterotopic or ectopic) foci of excitation can arise at any site of the conduction system (in the atria, ventricles, atrioventricular region). They can cause premature contraction of the heart before termination of the normal diastolic pause. This premature contraction is called extrasystole, and the disorder of the cardiac rhythm is called extrasystolic arrhythmia. If the activity of the ectopic focus is very high, it can become a temporary pacemaker, and all impulses governing the heart will during this time be emitted from this focus. The cardiac rhythm is then markedly accelerated. The condition is known as paroxysmal tachycardia. Ectopic arrhythmias are often due to increased excitability of the myocardium. The phenomenon known as re-entry can be another mechanism of ectopic arrhythmia. If an impulse meets an obstacle in the pathway of its conduction (local conduction disorder), the excitation wave can return from this obstacle to excite the myocardium.

**Extrasystolic arrhythmia (Ectopic beats, Extrasystoles)**

Extrasystoles are premature cardiac beats resulting from an abnormal electrical focus or re-entry mechanism in the atria, AV (atrio-ventricular) junction and ventricles. Extrasystole usually develops during normal contractions of the
heart governed by the sino-atrial node (nomotopic contractions). Ectopic foci of excitation can arise at any site of the conduction system. Usually excitations arise in the ventricles, less frequently in the atria, the atrioventricular node, and in the sino-atrial node (sinus extrasystole). A nomotopic contraction of the heart that follows extrasystole occurs in a longer (than normal) interval of time. This can be explained as follows. During the atrial extrasystole, excitation from the ectopic focus is transmitted to the sino-atrial node to "discharge" it, as it were. The next impulse arises in the sino-atrial node only in a lapse of time that is required to "discharge" the node and to form a new impulse.

In ventricular extrasystole, the time between the extrasystolic contraction and subsequent nomotopic contraction is even longer. The impulse from the heterotopic focus, located in the ventricles, propagates only over the ventricular myocardium; it would not be usually propagated to the atria via Aschoff-Tawara (AV) node. The impulse occurs in normal time in the sino-atrial node but it is not transmitted to the ventricles because they are refractory after the extrasystolic excitation. The next impulse from the sinoatrial node will only excite and contract the atria and the ventricles. A long "compensatory" pause therefore follows the ventricular extrasystole which lasts till the next nomotopic contraction. Extrasystolic arrhythmia is quite common. Functional extrasystole may occur in practically healthy individuals as a result of overexcitation of certain sites of the conduction system due to the action of the extracardiac nervous system in heavy smokers and in persons abusing strong tea or coffee; it can occur by reflex in diseases of the abdominal organs, or it may be due to hormonal disorders (thyrotoxicosis, menopause), various intoxications, disorders of electrolyte metabolism, etc. Organic extrasystole often attends various cardiovascular pathological conditions due to inflammatory or dystrophic affections of the myocardium or its deficient blood supply. Patients with extrasystole can feel their heart missing a beat (escape beat) and a subsequent strong stroke. Auscultation of the heart reveals its premature contraction with a specific loud first sound (due to a small diastolic filling of the ventricles). Extrasystole can be easily revealed by
feeling the pulse: a premature weaker pulse wave and a subsequent long pause are characteristic. If extrasystole follows immediately a regular contraction, the left ventricle may be filled with blood very poorly and the pressure inside it may be so small that the aortic valve would not open during the extrasystolic contraction and the blood will not be ejected into the aorta. The pulse wave on the radial artery will not be then detectable (missing pulse).

ECG characteristics of all extrasystoles:
1) premature appearance of the cardiac complex;
2) elongated pause between the extrasystolic and subsequent normal contraction;
3) Compensatory pause - the sum of pre-extrasystolic and postextrasystolic intervals. Complete compensatory pause equals to 2 normal R-R intervals. Incomplete compensatory pause is lesser than 2 normal R-R intervals. If excitability of the myocardium is high, several (rather than one) ectopic foci may exist. Extrasystoles generated in various heart chambers and having different configuration then appear on the ECG (polytopic extrasystole).

Wherever an ectopic focus may arise, its impulses may alternate in a certain order with the normal impulses of the sino-atrial node. This phenomenon is known as allorhythmia. Extrasystole may alternate with each sinus impulse (bigeminy), or it may follow two normal impulses (trigeminy), or three normal impulses (quadrigeminy). If the heterotopic focus is even more active, a normal contraction may be followed by several extrasystoles at a run (group extrasystole), which sometimes precedes an attack of paroxysmal tachycardia.

Classification of extrasystolic arrhythmia
1. According to the origin: functional and organic extrasystoles.
2. According to the site of origin: - atrial, atrioventricular (nodal) (common name of supraventricular); - ventricular (left- and rightventricular).
3. According to quantity of ectopic beats: - single, group, paired, frequent extrasystoles; - allorhythmia - alternaton of extrasystoles with sinus beats - bigeminy (1:2), trigeminy (1:3), quadrigeminy (1:4).
4. According to quantity of ectopic foci: - monotopic and polytopic (polymorphic) extrasystoles.

**Atrial extrasystole**

Excitation of the atria only changes in atrial extrasystole because the impulse is generated not in the sino-atrial node, and the ventricles are excited by the usual way.

ECG signs of atrial extrasystole (Fig. 25):

1) premature appearance of the cardiac complex;
2) P wave - normal shape or slightly disfigured and superimposed on the preceding T wave;
3) normal shape of QRS;
4) slight elongation of the diastolic pause (T-P interval) following the extrasystolic contraction (incomplete compensatory pause.

![Fig. 25. Atrial extrasystole](image)

**Atrioventricular (nodal) extrasystole** (premature atrioventricular junctional complex)

In atrioventricular (nodal) extrasystole the excitation of the atria differs from normal more substantially than in atrial extrasystole. The AV node impulse is
transmitted to the atria retrogradely, from bottom to top. The ventricles are excited in nodal extrasystole in the usual way.

ECG signs of atrioventricular extrasystole (Fig. 26):
1) premature appearance of the cardiac complex;
2) negative P wave because of the retrograde atrial excitation (may be absence of P wave);
3) the position of the P wave with respect to the ventricular complex depends on the rate of propagation of the excitation wave onto the atria and the ventricles;
4) shape of QRS is normal or slightly deformed;
5) incomplete compensatory pause.

Fig. 26. Atrioventricular (nodal) extrasystole

**Ventricular extrasystole (ventricular ectopic beat)**

Heart excitation order changes sharply in ventricular extrasystole. The ventricular impulse is not usually transmitted retrogradely through the AV node and the atria are not therefore excited. Second, the ventricles are not excited synchronously (as in normal cases), but one after another, i.e. that ventricle is excited first where the ectopic focus is located. The time of excitation of the ventricles is therefore longer and the QRS complex wider. The ventricular extrasystole is followed by a long (full) compensatory pause (except in interpolated extrasystoles): the atria are only excited by the sinus impulse that follows the extrasystole because the ventricles are refractory at this moment. The P wave corresponding to the atrial excitation is "lost" in the disfigured extrasystolic ventricular complex. Only next (second to the extrasystole) sinus impulse excites both the atria and the ventricles, while the EGG shows a normal cardiac complex.

ECG signs of ventricular extrasystole (Fig.27):
1) premature appearance of the ventricular complex;
2) absence of the P wave;
3) deformation of QRS complex due to increased voltage and length;
4) the shape and the height of the T wave changes, its direction is opposite to the maximum wave of the QRS complex (T wave is negative if the R wave is high, and positive if the S wave is deep;
5) complete compensatory pause. Sometimes it is possible to determine in which particular ventricle the ectopic focus is located.

![ECG Image](image)

Fig.27. Left-ventricular extrasystole

This can be done from the configuration of the ventricular complex in various ECG leads.

Left-ventricular extrasystole is characterized by a high R wave in III standard lead and the deep S wave in I lead (Fig.27). Left-ventricular extrasystoles are characterized by the appearance of the extrasystolic complex with a high R wave in the right chest leads and a broad or deep S wave in the left chest leads.
In right-ventricular extrasystole, the extrasystolic complex is characterized by a high R wave in I lead, and a deep S wave in the III. Chest leads are very important for the topic diagnosis of ventricular extrasystole.

In right-ventricular extrasystole, on the contrary, the deep S wave is recorded in the right chest leads, and a high S wave in the left chest leads (Fig.28).

**Paroxysmal tachycardia (PT)**

Paroxysmal tachycardia is a sudden acceleration of the regular cardiac rhythm with more than three complexes (to 140-250 beats per min) resulting from an abnormal ectopic impulses or re-entry mechanism in the atria, AV junction and ventricles. At attack of paroxysmal tachycardia may last from several seconds to a few days and terminate just as unexpectedly as it begins. During an attack, all impulses arise from a heterotopic focus because its high activity inhibits the activity of the sino-atrial node. Paroxysmal tachycardia (like extrasystole) may occur in subjects with increased nervous excitability, in the absence of pronounced affections of the heart muscle, but it arises more likely in the presence of a severe heart disease (e.g. myocardial infarction, heart defects or cardiosclerosis).

During an attack of paroxysmal tachycardia, the patient feels strong palpitation, discomfort in the chest, and weakness. The skin turns pale, and if
attack persists, cyanosis develops. Paroxysmal tachycardia is characterized by swelling and pulsation of the neck veins, because during accelerated pulse (to 180-200 per min) the atria begin contracting before the ventricular systole ends. The blood is ejected back to the veins from the atria to cause pulsation of the jugular veins. Auscultation of the heart during an attack of paroxysmal tachycardia reveals decreased diastolic pause, whose length nears that of the systolic one, and the heart rhythm becomes fetal (pendulum). The first sound increases due to insufficient ventricular diastolic filling. The pulse is rhythmic, very fast, and small. Arterial pressure may fall. If an attack persists (especially if it develops in the presence of a heart disease) symptoms of cardiac insufficiency develop. Like in extrasystole, the heterotopic focus in paroxysmal tachycardia may be located in the atria, the atrioventricular node, and the ventricles. It is possible to locate the focus only by electrocardiography: a series of extrasystoles follow on an ECG at regular intervals and at a very fast rate

Classification according to the site of origin:
- atrial, atrioventricular (nodal) (common name of supraventricular) PT;
- ventricular PT.

Supraventricular paroxysmal tachycardia characteristics (Fig. 29):
- Sudden acceleration of the regular cardiac rhythm to 140-250 per min and sudden deceleration of HR;
  - QRS shape is not changed as rule (or slightly deformed);
  - P wave – disfigured (or biphase, negative) prior to QRS in atrial PT or follows QRS in AV nodal PT, or is not differed.

Fig. 29. Supraventricular paroxysmal tachycardia
Ventricular paroxysmal tachycardia is characterized (Fig. 30):

- Sudden acceleration of the regular cardiac rhythm to 140-220 per min and sudden deceleration of HR;

- QRS shape is changed and broadened (>0.12 s); ventricular complexes are similar to those in ventricular extrasystoles;

- P waves not correspond to ventricular complexes QRS (atrioventricular dissociation) or are not differed on ECG.

Ventricular flutter and fibrillation

Ventricular flutter usually appears as a sinusoidal wave with a rate between 150 and 300 per minute. Ventricular fibrillation is a rapid irregular ventricular rhythm due to multiple re-entrant activities associated with essentially zero cardiac output. It is a variant of cardiac arrest (Fig. 31). The absence of adequate ventricular systole and contraction of separate ventricular muscles cause pronounced disorders in the hemodynamic and rapidly lead to death. Ventricular fibrillation occurs in grave affections of the myocardium (diffuse myocardial infarction, etc.). The patient loses consciousness, becomes pallid, the pulse and
arterial pressure become indeterminable. The ECG shows abnormal complexes on which separate waves are distinguished with difficulty.

![ECG of ventricular fibrillation](image)

**Fig. 31. Ventricular fibrillation**

**Ciliary arrhythmia**

Ciliary arrhythmia includes two variants: atrial fibrillation and atrial flutter. According to course of ciliary arrhythmia is divided on paroxysmal and persistent forms of ciliary arrhythmia. Atrial fibrillation Atrial fibrillation is rapid irregular atrial rhythm due to multiple reentrant wavelets. Atrial fibrillation otherwise known as complete or absolute arrhythmia. It arises in cases with suddenly increased excitation of the myocardium and simultaneous conduction disorders. The sino-atrial node fails to function as the pacemaker and many ectopic excitation foci (to 600–800 per min) arise in the atrial myocardium, which becomes only possible with a marked shortening of the refractory period. Since conduction of these impulses is difficult, each of them only excites and causes contraction of separate muscular fibres rather than the entire atrium. As a result, minor contractions develop in the atrium (atrial fibrillation) instead of adequate atrial systole. Only part of the impulses is transmitted to the ventricles through the AV node. Since conduction of atrial impulses is irregular, the ventricles contract at irregular intervals to cause complete arrhythmia of the pulse.

Depending on the conductability of the AV node and according to rate of ventricular contractions three forms of atrial fibrillation are distinguished:

- normasystolic form - 60-100 per min (Fig.32);
- tachysystolic form ->100 per min (Fig. 33, 34);
- bradysystolic form - <60 per min.
Fibrillation is characteristic of mitral heart diseases (especially of mitral stenosis), coronary atherosclerosis, thyrotoxicosis, etc. Fibrillation may occur as a permanent symptom or in attacks of tachyarrhythmia. Clinically fibrillation (bradyarrhythmia) may cause no subjective symptoms. Tachyarrhythmia is usually characterized by palpitation. Examination of the heart reveals complete irregularity of the heart contractions. Variations in the length of diastole account for variations in ventricular filling and hence in the intensity of the heart sounds. The pulse is also arrhythmic, pulse waves vary in height (irregular pulse), and pulse deficit often develops in frequent heart contractions.

ECG characteristics of atrial fibrillation (Fig. 32-34):

1) P wave disappears,
2) multiple small irregular f waves,
3) QRS ventricular complexes follow are irregular, their shape does not change.

Fig. 32. Atrial fibrillation (normasystolic form).
Atrial flutter

Atrial flutter is a rapid regular atrial rhythm due to a constant well-defined macro-reentrant circuit in the right atrium. Atrial flutter is the upset cardiac rhythm, which nears in its pathogenesis to fibrillation. As distinct from fibrillation, the number of impulses arising in fluttering atria does not usually exceed 250-300 per min, and their conduction through the AV node is usually rhythm. As a rule,
not all atrial impulses are conducted to the ventricles. Each other, third or fourth impulse, is only conducted to the ventricles since partial (incomplete) atrioventricular block develops simultaneously.

Conduction of the AV node sometimes constantly changes: each other impulse is now conducted; then the rhythm changes to conduction of each third impulse, and the ventricles contract arrhythmically. Like fibrillation, atrial flutter occurs in mitral defects, coronary atherosclerosis, and thyrotoxicosis; flutter sometimes develops in poisoning with quinine or digitalis. Patients with accelerated heart rate (high conduction of the AV node) complain of palpitation. Examination reveals tachycardia that does not depend on the posture of the patient, exercise or psychic strain, since the sino-atrial node does not function as the pacemaker in atrial flutter (being governed by extracardial nerves). Heart contractions are arrhythmic in patients with varying conduction of the AV node.

ECG characteristics of atrial flutter (Fig. 35, 36):

1) high F waves instead of the normal atrial P waves;
2) The number of F waves preceding each ventricular complex depends on the AV conduction;
3) QRS complexes follow at regular intervals.

Fig.35 Atrial flutter
Conduction disorders (heart blocks)

Heart blocks are delayed conduction or complete absence of conduction in some department of cardiac conduction system. Transmission of the impulse may be blocked at any part of the heart's conduction system. Block may develop in inflammatory, dystrophic, and sclerotic affections of the myocardium (e.g. rheumatic and diphtheritic myocarditis or cardiосclerosis). The conduction system may be affected by granulomas, cicatrices, toxins, etc. Conduction is often impaired in disordered coronary circulation, especially in myocardial infarction (the interventricular septum is involved). Block may be persistent and intermittent. Persistent block is usually connected with anatomic changes in the conduction system, whereas intermittent block depends largely on the functional condition of the atrioventricular node and the His bundle and is often connected with increased influence of the parasympathetic nervous system; atropine sulphate is an effective means that restores conduction.

Clinical and ECG signs of block depend on its location. The following types of heart blocks are distinguished:

- sino-atrial block - impairment of conduction between sinus node and atria;
- intra-atrial block - impairment of conduction through the atrial myocardium;
- atrioventricular block - impairment of conduction between atria to the ventricles;
- intraventricular block (His bundle branch block)
- impairment of conduction through the His bundle and its branches.

Sino-atrial block
Sino-atrial block is characterized by periodic missing of the heart beat and pulse beat.

ECG signs of sino-atrial block (Fig.37):
- periodic missing of the heart complex (PQRST) in the presence of a regular sinus rhythm;
- the length of diastole doubles. Intra-atrial block Intra-atrial block can only be detected electrocardiographically because clinical signs are absent.

![Fig.37. Sino-atrial block: (1) periodic missing of the heart complex (PQRST) in the presence of a regular sinus rhythm; (2) the length of diastole doubles.](image)

ECG signs of intra-atrial block (Fig.38):
- P waves are broadened ≥0.11 s and splitted;
- P wave in the V1 lead has two phases.
Atrioventricular block

Atrioventricular block is most important clinically. It is classified into three degrees by gravity.

I degree of AV block This block cannot be detected clinically, except that splitting of the first sound may sometimes be detected by auscultation (splitting of the atrial component) (Fig. 39).

I degree of AV block can only be revealed electrocardiographically:
- increased P-Q interval (> 0.21 s to 0.3-0.4 s and more) without missing QRS;
- regular heart rhythm.

II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type)

Conduction of the AV node and His bundle is impaired: each impulse transmitted from the atria to the ventricles increases and the P-Q interval on the ECG becomes longer with each successive beat.

A moment arrives at which one impulse does not reach the ventricles and they do not contract, hence the missing QRS complex on an ECG. During a long
diastole, which now follows, the conduction power of the atrioventricular system is restored, and next impulses will again be transmitted, but their gradual slowing down will be noted again; the length of the P-Q interval will again increase in each successive complex. The length of diastole which follows the P wave is called the Samoilov-Wenckebach period. This type of block is characterized clinically by periodically missing ventricular contractions, and hence missing pulse beats, which correspond to the Samoilov-Wenckebach period.

ECG signs of II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type) (Fig.40):
- gradual elongation PQ (Samoilov-Wenckebach periods);
- periodically missing ventricular contractions.

![Fig.40. II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type)](image)

II degree of AV block (Mobitz-2 type)

The second-degree atrioventricular block can be characterized by a worse conduction. The P-Q interval remains constant, but only each second, third, or (less frequently) fourth impulse is transmitted to the ventricles. The number of P waves on the ECG is therefore larger than of ventricular complexes. This is known as incomplete heart block with a 2:1, 3:1, etc. ratio. Considerable deceleration of the ventricular rhythm and slow pulse are characteristic, especially in 2:1 block. If each third or fourth beat is missing, the pulse is irregular and resembles trigeminy or quadrigeminy with early extrasystoles and pulse deficit. If the heart rhythm
slows down significantly, the patient may complain of giddiness, everything going black before his eyes, and transient loss of consciousness due to hypoxia of the brain.

ECG signs of II degree of AV block (Mobitz-2 type) (Fig.41):
- periodic missing QRS without gradual elongation PQ (2:1, 3:1);
- PQ can be normal or a little bit prolonged.

III degree of AV block (complete heart block). There is no electrical communication between the atria and the ventricles, and sinus node becomes the only pacemaker for the atria. The ventricles contract by their own automaticity in the centres of the second or third order (about 30-40 per min).

ECG signs of complete heart block (Fig.42-44):
1) atrial P waves and ventricular complexes QRS are recorded independently of each other;
2) the number of QRS is usually much smaller than the number of atrial P waves;
3) the shape of the ventricular complex does not change if the pacemaker arises from the AV node or His bundle;
4) with lower location of the pacemaker in the conduction system, the QRST complexes are altered.
Fig. 42. Complete heart block

Fig. 43. Complete heart block

Fig. 44. Complete heart block
The heart rate in persistent complete heart block may be sufficiently high (40-50 beats/min) but the patient may be unaware of the disease for a long time. Examination of such patients reveals slow, rhythmic, and full pulse. The heart sounds are dulled but a loud first sound ("pistol-shot" sound according to Strazhesko) may be heard periodically. It occurs due to coincidence of the atrial and ventricular contractions. If the ventricular rhythm slows down significantly (to 20 beats/min and less), or the heart misses a beat when incomplete heart block converts into a complete one, i.e. when the impulses from the atria are not conducted to the ventricles, while their automaticity has not yet developed, attacks (the Morgagni-Adams-Stokes syndrome) may occur due to disordered blood supply, mainly to the central nervous system. During an attack the patient loses consciousness, falls, general epileptiform convulsions develop, the respiration becomes deep, the skin pallid, the pulse very slow or even impalpable. When the ventricular automaticity restores, the patient regains his consciousness and all other signs of the syndrome disappear. If automaticity is not restored for a time, fatal outcome is possible.

His bundle branch blocks

Intraventricular block usually develops as the right or left bundlebranch block. The left limb of the His bundle ramifies almost immediately to give left anterior and left posterior branches. Only one branch can therefore be blocked. Block of the right limb may be combined with block of the branches of the left limb. In complete block of either of the limbs, the impulse from the sino-atrial node is normally conducted through the AV node and the main part of the His bundle to meet an obstacle to its conduction in that ventricle whose branch is affected. The ventricle with the intact branch is therefore first excited and excitation is transmitted to the ventricle with the affected branch. The ventricles are thus excited slowly and in an unusual way.

ECG signs of His bundle branch blocks:
1) P wave does not change;
2) ventricles contract rhythmically by the impulse from the sinus node;
3) QRS complexes are markedly altered and widened ≥ 0.12-0.18 s and resemble complexes in ventricular extrasystole;

4) The shape of the ventricular complexes depends on the particular bundle branch which is blocked.

ECG in left bundle branch block is characterized (Fig. 45):
- wide and deformed QRS has the form of qR in I, II, V5-6; rS in III, aVF, V1-2 (the shape of the ventricular complexes resembles that of right ventricular extrasystoles);
- disconcordance of ST, T and the main wave of QRS;
- levogram.

![Fig. 45. Left bundle branch block](image)

ECG in right bundle branch block is characterized (Fig. 46):
- wide QRS in III, V1-2 has the form of rsR, rSR, RsR´(similar to “M”) (the shape of the ventricular complexes resembles that of left ventricular extrasystoles);
- wide S in I, aVL, V5-6; - negative ST and T in V1-2;
- dextrogram.
Fig. 46. Right bundle branch block
II. The study of gas composition and blood acid-base status (Astrup microtechnique).

Normally, in a healthy person the constancy of pH (7.4; 7.35-7.45) is maintained by buffer systems of blood and lung and kidney (Shmidt R., Tevs G., 1996). Equilibrium in acid-base status is determined by ratio of hydrogen (H+) and hydroxyl (OH-) ions. With increasing concentration of H+ ions oxidizes the blood, while increasing OH- ions oxalacetate. Bicarbonate buffer system regulates the buffering capacity of the blood due to the change PaCO₂ - voltage of carbon dioxide in the blood (level PaCO₂ depends on lung ventilation and at the same time affects it). Adjusting the tension of CO₂ in blood, respiratory system facilitates effective buffer system in General.

Important buffer parameters:
1. BB - the sum of all the buffer bases of the blood ≈ 48 mmol/L. This value does not change when the shifts PaCO₂;
2. BE - the excess of bases (shows how the concentration of buffer bases is deviated from the normal value (≈ 48 mmol/l). The normal value of VE varies from -2.5 to +2.5 mmol/L. the Level BE > -5 mmol/l – metabolic acidosis.<5 mmol/l is characteristic of metabolic alkalosis.
3. SB - standard bicarbonate. SB ≈ 24 mmol/l. SB corresponds to the content of bicarbonate in the plasma of fully oxygenated and equilibrated with a gas mixture (PCO2 = 40 mm Hg) at 37 degrees Celsius.

For the study of gas composition of blood and acid-base status is used microtechnique Astrup. Analyzed blood obtained by puncture of the scarifier terminal phalanx of one finger, pre-warmed for 10 minutes at a temperature of about 40 °C and treated with alcohol. Portion blood type in special capillaries that are on both sides immediately after the blood sample are isolated from the air "plugs". Oxygen tension is measured polarographically (Shmidt R., Tevs G., 1996).
Define:
1. indicators in acid-base status: pH, BE, BB, SB, etc.;
2. indicators of gas composition of blood arterialization: partial tension of oxygen \( \text{PaO}_2 \) (normal > 80 mm Hg) partial tension of carbon dioxide \( \text{PaCO}_2 \) (normal 35-45 mm Hg), the saturation of hemoglobin by oxygen \( \text{SaO}_2 \) (normal > 95%).

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

1. Arterial hypoxemia (\( \text{PaO}_2 < 80 \) mm Hg). At persons of young age \( \text{PaO}_2 \) is about 95 mm Hg to 40 years it is reduced to 80 mm Hg and 70 years approximately 70 mm Hg. In the clinic chronic respiratory diseases are the most frequent cause of arterial hypoxemia is the development of chronic respiratory insufficiency (IR) in COPD patients

2. Arterial hypocapnia (\( \text{PaCO}_2 < 35 \) mm Hg). Characteristic of the early stages IR when arterial hypoxemia is compensated by the increased pulmonary ventilation, resulting in lower \( \text{PaCO}_2 \) and development of respiratory alkalosis.

3. Arterial hypercapnia (\( \text{PaCO}_2 > 45 \) mm Hg). A characteristic feature of the later stages IR when the removal of carbon dioxide through the alveolo-capillary membrane is disturbed. It is often noted respiratory acidosis is an important sign of severity IR.

4. Respiratory alkalosis (\( \text{PaCO}_2 < 35 \) mm Hg, \( \text{BE} = \pm 2.5 \) mmol/l).

5. Respiratory acidosis (\( \text{PaCO}_2 > 45 \) mm Hg, \( \text{BE} = \pm 2.5 \) mmol/l)

6. Metabolic alkalosis (\( \text{PaCO}_2 = 35-45 \) mm Hg \( \text{BE} > +5 \) mmol/l) is atypical for a primary lesion of the lung.

7. Metabolic acidosis (\( \text{PaCO}_2 = 35-45 \) mm Hg \( \text{BE} < -5 \) mmol/l) - metabolic acidosis atypical for a primary lesion of the lung.

The study of gas composition and blood acid-base balance is a powerful technique for objectifying the severity of chronic respiratory failure. To determine blood gas composition in COPD patients is recommended when the level of
oxygen saturation of hemoglobin (SpO₂) by pulse oximetry data < 92% and FEV₁ < 50%.

The response to drugs and the therapeutic response to oxygen can then be monitored easily. Haemoglobin saturation reflects oxygen carriage by the blood and thus the adequacy of tissue oxygenation (if perfusion is satisfactory) and the requirement for oxygen therapy. This can be measured noninvasively by pulse oximetry. Normally haemoglobin saturation is 95-99%. There is a type of pulse oximetry providing 12-24 hours monitoring of oxygen saturation (SaO₂) during night sleep (Fig.47). It is important for diagnosis of night hypoxemia in COPD patients and obstructive apnea.

Fig.47. Night sleep monitoring of oxygen saturation (SaO₂) in COPD patient (our own observation).
Decrease of oxygen saturation of hemoglobin min SpO2 decreased to 77%, SpO2max - 99%, SpO2 medium – 95.18%, total number of desaturation during night sleep – 57.

There is a type of pulse oximetry providing monitoring of oxygen saturation (SaO₂) during physical exercises, for example 6 minutes walking test (6MWT) (Fig.48).

Fig.48. Monitoring of oxygen saturation (SaO₂) during 6 minutes walking test (our own observation).

When conducting a test with 6 minute walk of marked reduction in distance travelled (63% of theoretical), decrease of oxygen saturation of hemoglobin and hemodynamics: min SpO2 decreased to 66%, SpO2max - 84%, SpO2 medium - 74.2%, range SpO2 shifted in ranges of less than 84% (SpO2 in the range of 95-100% 94-90% is not registered, SpO2 in the range of 80-84% was 14% of the recording time, SpO2 in the range of 75-79% - 35%, SpO2 in the range of 70-74% - 37% and SpO2 in the range < 70 – 14% of the time entries); index T5(Δ≥5%) has doubled, reaching 21.3 per cent. Cardio-vascular system also highlights the negative trends of heart rate: the heart rate medium 106 beats/min, min heart rate is 71 BPM, max heart rate - 144 beats/min, recorded one episode of tachycardia duration of 10.8% of the recording time.
III. Spirometry

*Static lung volumes* (see Fig. 49) reflect the elastic properties of the lungs and chest wall.

Fig. 49. Static lung volumes  ATS/ERS Task Force: Standardisation of lung function testing, 2005
Vital capacity (VC or "slow VC") is the maximum volume of air that can be expired slowly after a full inspiratory effort. Simple to perform, it is one of the most valuable measurements of pulmonary function. Because VC decreases as a restrictive lung disorder (eg, pulmonary edema, interstitial fibrosis) worsens, it can be used along with the diffusing capacity to follow the course of such a disorder and its response to therapy. The VC also reflects the strength of the respiratory muscles and is often used to monitor the course of neuromuscular disorders.

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

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

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

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

The FRC has two components:

- **Residual volume** (RV), the volume of air remaining in the lungs at the end of a maximal expiration, and
- **Expiratory reserve volume** (ERV); ERV = FRC - RV.

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

**Dynamic lung volumes** reflect the caliber and integrity of the airways. Spirometry (see Fig. 51) records lung volume against time during an FVC maneuver.

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

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

![Fig.51. Dynamic lung volumes.](image)
Prolongation of expiratory flow rates is increased by bronchospasm (in asthma), impacted secretions (in bronchitis), and loss of lung elastic recoil (in emphysema). In fixed obstruction of the upper airway, flow is limited by the caliber of the narrowed segment rather than by dynamic compression, resulting in equal reduction of inspiratory and expiratory flow rates.

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

Retesting pulmonary function after the patient inhales a bronchodilator aerosol (eg, albuterol, ipratropium) provides information about the reversibility of an obstructive process (ie, the asthmatic component). Improvement in FVC or FEV$_1$(L) of > 15 to 20% is usually considered a significant response (Fig.52). In patients with airway obstruction, absence of a response to a single exposure to a bronchodilator, however, does not preclude a beneficial response to maintenance therapy. In bronchoprovocation testing, a significant decrease in flow rates after inhaling methacholine (a cholinergic drug) may indicate asthma.

![Fig.52. FEV$_1$ before and after bronchodilator aerosol inhale.](image-url)
**Maximal voluntary ventilation** (MVV) is determined by encouraging the patient to breathe at maximal tidal volume and respiratory rate for 12 sec; the volume of air expired is expressed in L/min. The MVV generally parallels the FEV1 and can be used to test internal consistency and estimate patient cooperation. The MVV can be estimated from the spirogram by multiplying the FEV1(L) x 40.

When the MVV is disproportionately low in a patient who seems to be cooperating, neuromuscular weakness should be suspected. Except in advanced neuromuscular disease, most patients can generate fairly good single-breath efforts (eg, FVC). Because the MVV is much more demanding, it can reveal the diminished reserves of weak respiratory muscles. The MVV decreases progressively with increasing weakness of the respiratory muscles and, along with maximum inspiratory and expiratory pressures (see below), may be the only demonstrable pulmonary function abnormality in patients with moderately severe neuromuscular disease.

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

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

The shape of the loop reflects the status of the lung volumes and airways throughout the respiratory cycle. Characteristic changes occur in restrictive and in obstructive disorders. The loop is especially helpful in detecting laryngeal and tracheal lesions. It can distinguish between fixed obstruction (eg, tracheal stenosis) and variable obstruction (eg, tracheomalacia, vocal cord paralysis) of the upper airway. Fig. 53. illustrates some characteristic flow-volume loop abnormalities.
(A) Normal. Inspiratory limb of loop is symmetric and convex. Expiratory limb is linear. Flow rates at midpoint of VC are often measured. MIF 50%FVC is > MEF 50%FVC because of dynamic compression of the airways. Peak expiratory flow is sometimes used to estimate degree of airway obstruction but is very dependent on patient effort. Expiratory flow rates over lower 50% of FVC (ie, approaching RV) are sensitive indicators of small airways status. (B) Restrictive disease (eg, sarcoidosis, kyphoscoliosis). Configuration of loop is narrowed because of diminished lung volumes, but shape is basically as in (A). Flow rates are normal (actually greater than normal at comparable lung volumes because increased elastic recoil of lungs and/or chest wall holds airways open). (C) COPD, asthma. Though all flow rates are diminished, expiratory prolongation predominates, and MEF is < MIF. (D) Fixed obstruction of upper airway (eg, tracheal stenosis, bilateral vocal cord paralysis, goiter). Top and bottom of loop are flattened so that the configuration approaches that of a rectangle. The fixed obstruction limits flow equally during inspiration and expiration, and MEF = MIF. (E) Variable extrathoracic obstruction (eg, vocal cord paralysis). When a single vocal cord is paralyzed, it moves passively in accordance with pressure gradients across the glottis. During a forced inspiration, it is drawn inward, resulting in a plateau of decreased inspiratory flow. During a forced expiration, it is passively blown aside and expiratory flow is unimpaired, ie, MIF 50%FVC is < MEF 50%FVC. (F) Variable intrathoracic obstruction (eg, tracheomalacia). During a forced inspiration, negative pleural pressure holds the “floppy” trachea open. With forced expiration, the loss of structural support results in narrowing of the trachea and a plateau of diminished flow (a brief period of maintained flow is seen before airway compression occurs). (From Oslopov B.N, Sadykova A.R., Karamysheva I.V. Introduction to internal diseases. Manual. Part V.2nd edition., 2008).
**Ordering Pulmonary Function Tests.** As a general preoperative screen, determination of the FVC, FEV1, FEV1 %FVC, and MVV usually suffices. Testing should be performed before chest or abdominal surgery in smokers > 40 yr old and in patients with respiratory symptoms. In patients with suspected laryngeal or tracheal disorders, a flow-volume loop should be requested. If weakness of the respiratory muscles is suspected, the MVV, MIP, MEP, and VC are the appropriate tests.

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

Table 4 is intended as general guidelines for interpreting pulmonary function tests.

<table>
<thead>
<tr>
<th><strong>Restrictive lung disease</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Impairment</td>
</tr>
<tr>
<td>VC (%predicted)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
</tr>
<tr>
<td>MVV</td>
</tr>
<tr>
<td>RV(%predicted)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Obstructive lung diseases</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Impairment</td>
</tr>
<tr>
<td>VC (%predicted)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
</tr>
<tr>
<td>MVV</td>
</tr>
<tr>
<td>RV(%predicted)</td>
</tr>
</tbody>
</table>
In the home for monitoring of lung function is recommended to determine the peak expiratory flow (PEF) using the peak flow. This method belongs to an early screening-diagnosis of bronchial obstruction – one of the most important biomarkers of asthma and COPD. The below-mentioned patient after necessary training the health worker is able to apply independently for objective control of their condition and timely treatment to the doctor in case of its functional state. Note, however, that the below-mentioned will never be able to replace spirometry, because peak flow can be used to identify only the peak expiratory flow (PEF).

Method of measurement of PEF:

Measurement of PEF is once in the morning before bronchodilator. The procedure is repeated three times and the highest peak expiratory flow (PEF) is recorded. This can be compared with a nomogram that shows the patient’s sex, age, and weight, and plotted on a chart to show the progress or response to treatment. Measurements should be made 2-3 weeks (minimum 1 week). If the measurement result is classified as proper or best for the period of measurement the indicator PEF (the so-called "index PEF-variability or PSV-lability respiratory tract") <80%, you should change basic therapy (Fig. 54). (GINA, 2002)
Airflow obstruction syndrome

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

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

A. Initial level of PEF is low (55%), index PEF-variability is very high (60%, Normally -20%).

B. After change of basic therapy PEF is 110%, index PEF-variability is 7%.

Fig.54. Monitoring of PEF.
Symptoms and signs. The main symptoms are cough, expiratory dyspnea. In lingering course of airflow obstruction syndrome it is necessary to mark the particular clinical meaning of cough not only as the symptom and sign of bronchi disorder but as factor, which worsened pulmonary parenchyma lesion itself.

There are few outward manifestations of abnormality in patients with mild airflow obstruction. However, as the process becomes more severe, the patient's distress becomes evident from
- the labored breathing,
- the use of the accessory muscles on respiration,
- inspiratory retraction of the supraclavicular fossae and lower interspaces, and
- the positioning of the chest near total lung capacity.

When the patient is asked to empty the lungs forcibly and completely, it is evident that expiration is prolonged and difficult, with pulmonary emptying incomplete. The degree of expiratory slowing may be estimated by measuring the forced expiratory time (which normally measures 4 seconds or less), with a watch and a stethoscope. Auscultation over the larynx permits accurate determination of the end of expiration. Sounds are audible at this site at the low airflows occurring near residual volume, when breath sounds are no longer audible over the lungs.

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

In prominent acute bronchial obstruction the picture of —silent lung appears, when the bronchial patency is altered so much that breath sounds aren't listened at all.
Functional tests: FEV\textsubscript{1} <80 % predicted, FEV\textsubscript{1} / FVC<70% predicted and FEF25-75% decrease.

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

**Respiratory deficiency syndrome.**

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

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

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

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

Two RI groups are distinguished:

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

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

1. disturbance of central regulation of respiration (traumatic, metabolic, circulatory, toxic, infectious and other brain lesions);
2. respiratory muscles lesion (trauma, intoxication, myalgia, myodystrophy, etc.) or peripheral nerves lesion (poliomyelitis, polyradiculoneuritis, tetanus);

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

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

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

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

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

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

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

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

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

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

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

Attention should be paid to the fact that the majority of pulmonary pathologic processes are accompanied by disturbance of several gas exchange mechanisms. For example, in pneumonia restriction lesions are mainly observed, obstructive lesions are somewhat less frequent, gases diffusion through alveolocapillary membrane decreases, number of functioning alveoli lessens, etc.
In chronic obstructive bronchitis alongside with pronounced obstructive lesions disturbances of ventilation-perfusion proportions are observed due to significant unevenness of pulmonary ventilation, etc.

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

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

Almost all existing definitions of RI clearly indicate a gas composition of arterial blood as a possible indicator of the degree days. The division of chronic respiratory insufficiency in severity depends on the levels of the two most important indicators of gas composition of arterial blood – PaO2 and SaO2 (saturation of hemoglobin with oxygen). This approach to the classification of the severity RI presented in table 5. (Avdeev S. N., 2004).

<table>
<thead>
<tr>
<th>Degree of RI</th>
<th>PaO2, мм Hg</th>
<th>SaO2, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norm</td>
<td>&gt;80</td>
<td>&gt;95</td>
</tr>
<tr>
<td>I</td>
<td>60–79</td>
<td>90–94</td>
</tr>
<tr>
<td>II</td>
<td>40–59</td>
<td>75–89</td>
</tr>
</tbody>
</table>

Table 5.

Classification of severity of respiratory insufficiency (S. N. Avdeev, 2004)
Main manifestations of respiratory insufficiency. Among a large number of respiratory insufficiency signs the following are most significant in clinical practice:

1. dyspnea;
2. central (diffuse) cyanosis;
3. enhanced work of respiratory muscles;
4. intensification of circulation (tachycardia, minute volume increase);
5. change of respiratory volumes and capacities.

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

IV. Laboratory tests

Sputum test

Information should be obtained about its quantity, colour (white, grey, black, pink, yellow or green), viscosity (serous or tacky), taste and odour (Table 7).

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

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

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

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

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

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

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

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

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

**Viral culture**

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

**Special procedures**

**Tuberculosis** is caused by a slow-growing bacteria called *Mycobacterium tuberculosis*. Because it does not easily grow using routine culture methods, special procedures are used to grow and identify this bacteria. When a sputum sample for tuberculosis first comes into the laboratory, a small portion of the sputum is smeared on a microscope slide and stained with a special stain, called an acid-fast stain. The stained sputum is examined under a microscope for tuberculosis organisms, which pick up the stain, making them visible. This smear is a rapid screen for the organism, and allows the physician to receive a preliminary report within 24 hours.

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

Other microorganisms that cause various types of lower respiratory tract infections also require special culture procedures to grow and identify. *Mycoplasma pneumoniae*
Pneumonia causes a mild to moderate form of pneumonia, commonly called walking pneumonia; *Bordetella pertussis* causes whooping cough; *Legionella pneumophila*, Legionnaire's disease; *Chlamydia pneumoniae*, an atypical pneumonia; and *Chlamydia psittaci*, parrot fever.

*Pneumocystis carinii* causes pneumonia in people with weakened immune systems, such as people with AIDS. This organism does not grow in culture. Special stains are done on sputum when pneumonia caused by this organism is suspected. The diagnosis is based on the results of these stains, the patient's symptoms, and medical history.

Sputum culture is also called sputum culture and sensitivity.

It is possible that sputum cultures will eventually be replaced in the diagnosis of tuberculosis by newer molecular techniques. These advanced methods speed the diagnostic process as well as improve its accuracy. As of late 2002, four molecular techniques are increasingly used in laboratories around the world to diagnose TB. They include polymerase chain reaction to detect mycobacterial DNA in patient specimens; nucleic acid probes to identify mycobacteria in culture; restriction fragment length polymorphism analysis to compare different strains of TB for epidemiologic studies; and genetic-based susceptibility testing to identify drug-resistant strains of mycobacteria.

**Complete blood count**

- Red blood cell data
  - Total red blood cell count (RBC)
  - Hemoglobin (Hgb)
  - Hematocrit (Hct)
  - Mean corpuscular volume (MCV)
– Red blood cell distribution width (RDW)

• White blood cell data
  – Total white blood cell (leukocyte) count (WBC)
  – A white blood cell count differential may also be ordered

• Platelet Count (PLT)

**Red blood cells**

• Number - Generally done by automated counters, using impedance measures

• Size - Large, normal size, or small; all same size *versus* variable sizes (*anisocytosis*). Mean volume by automated counter

• Shape - Normal biconcave disc, *versus* spherocytes, *versus* oddly shaped cells (*poikilocytosis*)

• Color - Generally an artifact of size of cell

Table 6.

**Normal values**

<table>
<thead>
<tr>
<th>RBC Parameters</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>4.5-5.2*10^9 per microliter</td>
</tr>
<tr>
<td>Males</td>
<td>4.7 t- 6.1*10^9 per microliter</td>
</tr>
<tr>
<td>Hematocrit</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>35-47%</td>
</tr>
<tr>
<td>Males</td>
<td>40-52%</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>120-160mg/l</td>
</tr>
<tr>
<td>Males</td>
<td>135-175 mg/l</td>
</tr>
</tbody>
</table>
Hemoglobin

The hemoglobin concentration is a measure of the amount of Hgb in the peripheral blood, which reflects the number of red blood cells in the blood.

- Hgb constitutes over 90% of the red blood cells
- Decrease in Hgb concentration = anemia
- Increase in Hgb concentration = polycythemia

Hematocrit

Hematocrit is a measure of the percentage of the total blood volume that is made up by the red blood cells.

- The hematocrit can be determined directly by centrifugation (“spun hematocrit”)
- The height of the red blood cell column is measured and compared to the column of the whole blood

- More commonly the Hct is calculated directly from the RBC and MCV
  - Hematocrit % = RBC (cells/liter) x MCV (liter/cell)
  - Because the Hct is a derived value, errors in the RBC or MCV determination will lead to spurious results

MCV

The MCV is a measure of the average volume, or size, of an RBC.

- It is determined by the distribution of the red blood cell histogram
  - The mean of the red blood cell distribution histogram is the MCV
- The MCV is important in classifying anemias
  - Normal MCV = normocytic anemia
  - Decreased MCV = microcytic anemia
– Increased MCV = macrocytic anemia

Red Blood Cell Distribution Width

• RDW is an indication of the variation in the RBC size (referred to anisocytosis)
  • It is derived from the red blood cell histogram and represents the coefficient of variation of the curve
  • In general, an elevated RDW (indicating more variation in the size of RBCs) has been associated with anemias with various deficiencies, such as iron, B12, or folate
  • Thalassemia is a microcytic anemia that characteristically has a normal RDW

Table 7

<table>
<thead>
<tr>
<th>Test</th>
<th>Full name</th>
<th>Examples of causes of low result</th>
<th>Examples of causes of high result</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>Red Blood Cell Count (Seereference range)</td>
<td>Known as anemia</td>
<td>Known as polycythemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Acute or chronic bleeding</td>
<td>• Dehydration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• RBC destruction (e.g., hemolytic anemia, etc.)</td>
<td>• Lung (pulmonary) disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Nutritional deficiency (e.g., iron deficiency, vitamin B12 or folate deficiency)</td>
<td>• Kidney or other tumor that produces excess erythropoietin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Bone marrow disorders or damage</td>
<td>• Smoking</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Chronic inflammatory disease</td>
<td>• Living at high altitude</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Chronic kidney</td>
<td>• Genetic causes (altered oxygen sensing, abnormality in hemoglobin oxygen release)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Polycythemia vera— a rare disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>Hb</strong></td>
<td>Hemoglobin (See reference range)</td>
<td>Usually mirrors RBC results, provides added information</td>
<td></td>
</tr>
<tr>
<td><strong>Hct</strong></td>
<td>Hematocrit (See reference range)</td>
<td>Usually mirrors RBC results</td>
<td></td>
</tr>
<tr>
<td><strong>RBC indices</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MCV</strong></td>
<td>Mean Corpuscular Volume (See reference range)</td>
<td>Indicates RBCs are smaller than normal (microcytic); caused by iron deficiency anemia or thalassemias, for example.</td>
<td></td>
</tr>
<tr>
<td><strong>MCH</strong></td>
<td>Mean Corpuscular Hemoglobin (See reference range)</td>
<td>Mirrors MCV results; small red cells would have a lower value.</td>
<td></td>
</tr>
<tr>
<td><strong>MCHC</strong></td>
<td>Mean Corpuscular Hemoglobin Concentration (See reference range)</td>
<td>May be low when MCV is low; decreased MCHC values (hypochromia) are seen in conditions such as iron deficiency anemia and thalassemia.</td>
<td></td>
</tr>
<tr>
<td><strong>RDW (Not always reported)</strong></td>
<td>RBC Distribution Width</td>
<td>Low value indicates uniformity in size of RBCs.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indicates mixed population of small and large RBCs; young RBCs tend to be larger. For example, in iron deficiency anemia or pernicious anemia, there is high variation (anisocytosis) in RBCs.</td>
<td></td>
</tr>
</tbody>
</table>
Reticulocyte Count (Not always done)

| Reticulocytes (absolute count or %) | In the setting of anemia, a low reticulocyte count indicates a condition is affecting the production of red blood cells, such as bone marrow disorder or damage, or a nutritional deficiency (iron, B12 or folate). | In the setting of anemia, a high reticulocyte count generally indicates peripheral cause, such as bleeding or hemolysis, or response to treatment (e.g., iron supplementation for iron deficiency anemia). |

**White Blood Cell Count**

White blood cells (WBCs), also called leukocytes, are cells that circulate in the blood and the lymphatic system that help protect the body against infections. They are an important part of the body's immune system and also have a role in inflammation, allergic responses, and protection against cancer. A WBC differential totals the number of each of the different types of WBCs in a person's sample of blood.

There are five types of white blood cells, each with different functions. The differential reveals if the cells are present in normal proportion to one another, if the number of one cell type is increased or decreased, or if abnormal and/or immature cells are present. This information is helpful in diagnosing specific types of illnesses that affect the immune system and the bone marrow.

A differential may be performed in conjunction with a complete blood count (CBC), a test often used as a general health check, or it may be performed in follow-up to abnormal results on a CBC. Most often, a differential is performed on an automated blood analyzer but also may be performed manually by a trained laboratorian who examines a blood smear under a microscope. The values are
typically reported as absolute numbers of cells but may be expressed as the relative percentages of the total number of WBCs.

White blood cells develop from precursor cells produced in the bone marrow. The five different types of WBCs include:

- **Granulocytes**—these white blood cells have granules in their cytoplasm. The granules contain chemicals and other substances that are released as part of an immune response. The three types of granulocytes include:
  - **Neutrophils** (neu) normally make up the largest number of circulating WBCs. They move into an area of damaged or infected tissue, where they engulf and destroy **bacteria** or sometimes **fungi**.
  - **Eosinophils** (eos) respond to infections caused by **parasites**, play a role in allergic reactions (hypersensitivities), and control the extent of immune responses and inflammation.
  - **Basophils** (baso) usually make up the fewest number of circulating WBCs and are thought to be involved in allergic reactions.

- **Lymphocytes** (lymphs) exist in both the blood and the lymphatic system. They are divided into three types, but the differential does not distinguish among them. All lymphocytes differentiate from common lymphoid progenitor cells in the bone marrow. The differential counts and reports all lymphocytes together. Separate specialized testing (like **immunophenotyping**) must be done to differentiate the three types:
  - B lymphocytes (B cells) are antibody-producing cells that are essential for acquired, antigen-specific immune responses. Plasma cells are fully differentiated B-cells that produce antibodies, immune proteins that target and destroy bacteria, **viruses** and other "non-self" foreign antigens.
  - T lymphocytes (T cells) finish maturing in the **thymus** and consist of a few different types. Some T cells help the body distinguish between "self" and "non-self" antigens. Others initiate and control the extent of an immune response, boosting it as needed and then slowing it as
the condition resolves. Other types of T cells directly attack and neutralize virus-infected or cancerous cells.

- Natural killer cells (NK cells) directly attack and kill abnormal cells such as cancer cells or those infected with a virus.

- **Monocytes** (mono), similar to neutrophils, move to an area of infection and engulf and destroy bacteria. They are associated more often with chronic rather than acute infections. They are also involved in tissue repair and other functions involving the immune system.

When there is an infection or an inflammatory process somewhere in the body, the bone marrow produces more WBCs, releasing them into the blood. Depending on the cause of infection or inflammation, one particular type of WBC may be increased as opposed to other types. As the condition resolves, the production of that type of WBC subsides and the number drops to normal levels again.

In addition to infections and inflammation, there are a variety of conditions that can affect the production of WBCs by the bone marrow or their survival in the blood, resulting in either increased or decreased numbers. The differential, along with the other components of the CBC, alerts the healthcare provider to possible health issues. Results are often interpreted in conjunction with additional tests such as a blood smear review, which can reveal the presence of abnormal and/or immature populations of WBCs.

In a few serious diseases, some immature forms of the cells are released from the bone marrow into the circulation and may be detected by the WBC differential. This may occur with bacterial infection, leukemia, bone marrow involvement by solid tumor, myelodysplastic syndrome, or myeloproliferative neoplasms, for example. Some immature cells that may be detected include metamyelocytes, myelocytes, promyelocytes, and/or blasts.
If results indicate a problem, a wide variety of other tests may be performed in order to help determine the cause. A healthcare provider will typically consider an individual's signs and symptoms, medical history, and results of a physical examination to decide what other tests may be necessary. For example, as needed, a bone marrow biopsy will be performed to evaluate the bone marrow status.

WBC differentials are either performed manually or by an automated instrument.

“Manual” WBC differentials are performed by trained medical technologists who count and categorize typically 100 white blood cells via microscopic examination of a Romanowsky-stained peripheral blood smear.

In addition to the differential count, evaluation of the smear provides the opportunity to morphologically evaluate all components of the peripheral blood, including red blood cells, white blood cells and platelets.

The manual differential allows for the detection of disorders that might otherwise be lost in a totally automated system.

This applies to < 20% of specimens.

The instrument is programmed with criteria to flag an operator when a manual differential should be performed.

The clinical laboratory may perform an “automated differential” via instruments with the capability of performing differential leukocyte counts.

Usually based on the determination of different leukocyte cellular characteristics that permit separation into subtypes by using flow-cytometric techniques.

Reference ranges for differential white blood cell count in normal adults is as follows:

- Neutrophils - 2.0–7.0×10^9/l (40–80%)
- Lymphocytes - 1.0–3.0×10^9/l (20–40%)
- Monocytes - 0.2–1.0×10^9/l (2–10%)
- Eosinophils - 0.02–0.5×10^9/l (1–6%)
- Basophils - $0.02–0.1 \times 10^9/l$ ($< 1–2\%$)

The reference ranges may vary depending on population studies, the individual laboratory, instruments, and methods.

Table 8

Possible Causes of high and low WBC differential results

<table>
<thead>
<tr>
<th>Type of WBC</th>
<th>Abbreviations</th>
<th>Examples of causes of a high count</th>
<th>Examples of causes of a low count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophils (Absolute neutrophil count, percent neutrophils)</td>
<td>Neu, Polys, PMNs, ANC, % Neu</td>
<td>Known as neutrophilia - Acute <em>bacterial</em> infections and also some infections caused by <em>viruses</em> and <em>fungi</em> - Inflammation (e.g., <em>inflammatory bowel disease</em>, <em>rheumatoid arthritis</em>) - Tissue death (necrosis) caused by trauma, major surgery, <em>heart attack</em>, burns - Physiological (stress, rigorous exercise) - Smoking - <em>Pregnancy</em>—last trimester or during labor - <em>Chronic leukemia</em> (e.g., <em>myelogenous leukemia</em>)</td>
<td>Known as neutropenia - <em>Myelodysplastic syndrome</em> - Severe, overwhelming infection (e.g., <em>sepsis</em>—neutrophils are used up) - Reaction to drugs (e.g., penicillin, ibuprofen, phenytoin, etc.) - <em>Autoimmune disorder</em> - Chemotherapy - Cancer that spreads to the bone marrow - <em>Aplastic anemia</em></td>
</tr>
<tr>
<td>Lymphocytes (Absolute lymphocyte count, percent)</td>
<td>Lymphs, lym, ly,</td>
<td>Known as lymphocytosis - Acute viral infections (e.g., <em>hepatitis</em>, <em>chickenpox</em>)</td>
<td>Known as lymphopenia or lymphocytopenia</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>ALC, % lymphs</td>
<td>Certain bacterial infections (e.g., pertussis (whooping cough), tuberculosis (TB))</td>
<td>Autoimmune disorders (e.g., lupus, rheumatoid arthritis)</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>------------------------------------------------------</td>
</tr>
<tr>
<td></td>
<td>n pox, cytomegalovirus (CMV), Epstein-Barr virus (EBV), herpes, rubella</td>
<td>Lymphocytic leukemia</td>
<td>Infections (e.g., HIV, TB, hepatitis, influenza)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lymphoma</td>
<td>Bone marrow damage (e.g., chemotherapy, radiation therapy)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Immune deficiency</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Monocytes (Absolute monocyte count, percent monocytes)</th>
<th>Monos, AMC, % monos</th>
<th>Known as monocytosis</th>
<th>Known as monocytopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Monocytosis</td>
<td>Chronic infections</td>
<td>Usually, one low count is not medically significant.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(e.g., tuberculosis, fungal infection)</td>
<td>Repeated low counts can indicate:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infection within the heart (bacterial endocarditis)</td>
<td>Bone marrow damage or failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Collagen vascular diseases (e.g., lupus, scleroderma, rheumatoid arthritis, vasculitis)</td>
<td>Hairy-cell leukemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inflammatory bowel disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monocytic leukemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic myelomonocytic leukemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Juvenile myelomonocytic leukemia</td>
<td></td>
</tr>
<tr>
<td>Eosinophils (Absolute eosinophil count, percent eosinophils)</td>
<td>Eos, AEC, % eos</td>
<td>Known as eosinophilia</td>
<td>Known as eosinopenia</td>
</tr>
<tr>
<td>----------------------------------------------------------</td>
<td>----------------</td>
<td>----------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Asthma, allergies such as hay fever</td>
<td>This is often difficult to determine because numbers are normally low in the blood. One or an occasional low number is usually not medically significant.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Drug reactions</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inflammation of the skin (e.g., eczema, dermatitis)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Parasitic infections</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inflammatory disorders (e.g., celiac disease, inflammatory bowel disease)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Certain malignancies/cancers</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Hyperesinophilic myeloid neoplasms</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Basophils (Absolute basophil count, percent basophils)</th>
<th>Baso, ABC, % baso</th>
<th>Known as basophilia</th>
<th>Known as basopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>- Rare allergic reactions (e.g., hives, food allergy)</td>
<td>As with eosinophils, numbers are normally low in the blood; usually not medically significant.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inflammation (rheumatoid arthritis, ulcerative colitis)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Some leukemias (e.g., chronic myeloid leukemia)</td>
<td></td>
</tr>
</tbody>
</table>

**Platelet Count (PLT)**

A count of the number of platelets (thrombocytes) per cubic milliliter of blood

A decreased number of platelets = Thrombocytopenia

An increased number of platelets = Thrombocytosis

**Table 9**

<table>
<thead>
<tr>
<th>Test</th>
<th>Full name</th>
<th>Examples of causes of low result</th>
<th>Examples of causes of high result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plt</td>
<td>Platelet</td>
<td>Known as</td>
<td>Know as thrombocytosis:</td>
</tr>
<tr>
<td>Count (Seerference range)</td>
<td>thrombocytopenia:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Viral infection (mononucleosis, measles, hepatitis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Rocky mountain spotted fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Platelet autoantibody</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Drugs (acetaminophen, quinidine, sulfa drugs)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Cirrhosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Autoimmune disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Sepsis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Leukemia, lymphoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Myelodysplasia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Chemo or radiation therapy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| MPV (Not always reported) | Mean Platelet Volume | Indicates average size of platelets is small; older platelets are generally smaller than younger ones and a low MPV may mean that a condition is affecting the production of platelets by the bone marrow. | Indicates a high number of larger, younger platelets in the blood; this may be due to the bone marrow producing and releasing platelets rapidly into circulation. |

| PDW (Not always reported) | Platelet Distribution Width | Indicates uniformity in size of platelets | Indicates increased variation in the size of the platelets, which may mean that a condition is present that is affecting platelets |
Coagulation disorders

Abnormal bleeding can result from disorders of the coagulation system, of platelets, or of blood vessels. Disorders of coagulation can be acquired or hereditary.

The major causes of acquired coagulation disorders are

- Vitamin K deficiency
- Liver disease
- Disseminated intravascular coagulation
- Development of circulating anticoagulants

Severe liver disease (e.g., cirrhosis, fulminant hepatitis, acute fatty liver of pregnancy) may disturb hemostasis by impairing clotting factor synthesis. Because all coagulation factors are made in the liver, both the PT and PTT are elevated in severe liver disorders. (PT results are typically reported as INR.) Occasionally, decompensated liver disease also causes excessive fibrinolysis and bleeding due to decreased hepatic synthesis of alpha 2-antiplasmin.

The most common hereditary disorder of hemostasis is

- von Willebrand disease (VWD)

The most common hereditary coagulation disorders are

- The hemophilias

Symptoms of coagulation disorders with difficulty clotting include:

- Blood in the urine or stool
- Bruising easily and excessively
- Extreme fatigue
- An injury that will not stop bleeding
- Joint pain caused by internal bleeding
- Nosebleeds that seem to have no cause
- A painful headache that will not go away
- Prolonged bleeding from ordinary cuts or from surgery or dental work
- Sudden pain, swelling and warmth in joints or muscles
• Vision problems, such as double vision
• Vomiting repeatedly

Symptoms of coagulation disorders with too much clotting include:
• A blood clot in one of the deep veins of the body (also called deep vein thrombosis or DVT).
  
  Symptoms of DVT include:
  o Pain in a particular area of the body
  o Swelling of an arm or leg
  o Redness or color change
  o Warmth of the skin

• A blood clot that has traveled to the lung (called a pulmonary embolism or PE).
  
  Symptoms of a PE include:
  o Chest pain
  o Shortness of breath
  o Fast heartbeat

• A heart attack or stroke at a young age
• Recurrent pregnancy loss or stillbirth

**Testing**

Patients in whom a coagulation disorder is suspected require laboratory evaluation beginning with prothrombin time (PT) and partial thromboplastin time (PTT). CBC with platelet count and a peripheral blood smear are also done. Results of these tests narrow the diagnostic possibilities and guide further testing.

**Normal results** on initial tests exclude many bleeding disorders. The main exceptions are VWD and hereditary hemorrhagic telangiectasia. VWD is a common entity in which the associated deficiency of factor VIII is frequently insufficient to prolong the PTT. Patients who have normal initial test results, along with symptoms or signs of bleeding and a positive family history, should be tested for VWD by measuring plasma von Willebrand factor (VWF) antigen, ristocetin
cofactor activity (an indirect test for large VWF multimers), VWF multimer pattern, and factor VIII levels.

If **thrombocytopenia** is present, the peripheral blood smear often suggests the cause. If the smear is normal, patients should be tested for HIV infection. If the result of the HIV test is negative and the patient is not pregnant and has not taken a drug known to cause platelet destruction, then idiopathic thrombocytopenic purpura is likely. If there are signs of hemolysis (fragmented RBCs on smear, decreasing Hb level), thrombotic thrombocytopenic purpura (TTP) or hemolytic-uremic syndrome (HUS) is suspected, although sometimes other hemolytic disorders can cause these findings. HUS occurs in patients with hemorrhagic colitis. An "atypical" form of HUS occurs uncommonly in individuals with congenital abnormalities of the alternative complement pathway. The Coombs test is negative in TTP and HUS. If the CBC and peripheral blood smear demonstrate other cytopenias or abnormal WBCs, a hematologic abnormality affecting multiple cell types is suspected, and a bone marrow aspiration and biopsy are necessary for diagnosis.

**Prolonged PTT with normal platelets and PT** suggests hemophilia A or B. Factor VIII and IX assays are indicated. Inhibitors that specifically prolong the PTT include an autoantibody against factor VIII and antibodies against protein-phospholipid complexes (lupus anticoagulant). Clinicians suspect one of these inhibitors when a prolonged PTT does not correct after 1:1 mixing with normal plasma.

**Prolonged PT with normal platelets and PTT** suggests factor VII deficiency. Congenital factor VII deficiency is rare; however, the short half-life of factor VII in plasma causes factor VII to decrease to low levels more rapidly than other vitamin K–dependent coagulation factors in patients beginning warfarin anticoagulation or in patients with incipient liver disease.

**Prolonged PT and PTT with thrombocytopenia** suggest DIC, especially in association with obstetric complications, sepsis, cancer, or shock. Confirmation is
by finding elevated levels of D-dimers (or fibrin degradation products) and decreasing plasma fibrinogen levels on serial testing.

**Prolonged PT or PTT with normal platelet count** occurs with liver disease or vitamin K deficiency or during anticoagulation with warfarin, unfractionated heparin, or the newer oral inhibitors of thrombin or factor Xa. Liver disease is suspected based on history and is confirmed by finding elevations of serum aminotransferases and bilirubin; hepatitis testing is recommended.

**Routine biochemistry**

**Jaundice**

Jaundice is a yellow pigmentation of the skin and mucous membranes caused by the presence in the blood of an excess of bile pigments. It is best seen in daylight. Jaundice may be due to increased production of bile pigments, defective transport or conjugation of bilirubin within the liver cell or obstruction to the outflow of bile from the liver to the duodenum.

Some knowledge of the biochemistry of bile pigments is essential for the proper understanding of jaundice.

In healthy subjects unconjugated bilirubin (haemo-bilirubin) is water-insoluble and derived from the breakdown of red cells by the reticuloendothelial system. It passes, attached to plasma albumin, to the liver where it is conjugated with glucuronide and possibly other substances.

Conjugated bilirubin glucuronide (hepatobilirubin) is water-soluble and is the major constituent of bile, which passes into the intestine. There it is changed by bacterial action into urobilinogen; the major part is excreted in the faeces but some is reabsorbed to enter the liver and a small part absorbed into the general circulation to appear in the urine.

**Prehepatic (haemolytic) jaundice**

This form of jaundice is due to the presence in the blood of an excess of unconjugated bilirubin. Although haemolysis is the most important cause of
prehepatic jaundice, it is now recognized that about 1% of the population have a mild unconjugated hyperbilirubinaemia of an entirely benign nature - Gilbert's syndrome. The jaundice is often not clinically detectable, but may deepen during fasting or intercurrent illness, resulting in a mistaken diagnosis of hepatitis.

Haemolytic jaundice may result from an inherited abnormality in the red cells or from acquired causes. Since these forms of haemolysis are usually accompanied by anaemia, they are dealt with in a chapter on anaemias. Sometimes a breakdown of red cells, as in gross pulmonary infarction or incompatible blood transfusion, causes prehepatic jaundice without anaemia.

When the red cells themselves are abnormal, as in hereditary spherocytosis, thalassaemia and to a lesser extent in pernicious anaemia, the cells may become osmotically and mechanically more fragile and are thus destroyed by the reticuloendothelial system. There may be a history of previous attacks of jaundice or a family history of jaundice. Auto-antibodies, neoplasia and certain virus infections may similarly cause acquired haemolytic jaundice.

In most forms of prehepatic jaundice, the skin and mucosae are delicately jaundiced (a lemon-yellow tint), but the urine and faeces remain normal in colour, though the urine may darken on standing due to oxidation of the excess urobilinogen.

**Hepatocellular jaundice**

This results from damage to the liver parenchyma interfering with the transport or conjugation of bi-lirubin and sometimes with its excretion through the canaliculi.

The commonest cause of hepatocellular jaundice is a virus hepatitis, so that a history of transfusion, contact with another case or, in hospital workers, contact with the blood of a carrier may be obtained. The possibility of exposure to a medicinal liver toxin, such as chlorpromazine, testosterone, halothane or
rifampicin, or an industrial one such as carbon tetrachloride, should always be considered. Hepatocellular jaundice also occurs in congestive cardiac failure and in the later stages of cirrhosis. When hepatic damage is accompanied by obstruction to the bile canaliculi (cholestatic jaundice), the characteristics of the jaundice itself are similar to those described under post-hepatic obstruction. The history of events preceding the jaundice, notably the prodromal period of anorexia and nausea in virus hepatitis, helps to differentiate the hepatocellular and posthepatic varieties. Liver function tests may also be helpful.

**Posthepatic (obstructive) jaundice**

This form of jaundice results from obstruction to the bile ducts outside the liver. The common causes include gallstones, primary carcinoma of the head of pancreas or bile ducts, and secondary carcinomatous masses in the porta hepatis. When the obstruction is due to gallstones the jaundice is usually preceded by biliary colic and may be intermittent. Jaundice due to carcinoma tends to be insidious in onset and progressive in its course, and the gallbladder is sometimes palpable.

Obstructive jaundice varies in intensity from a slight yellowish tinge in the skin and mucous membranes to a pronounced canary yellow, or, in longstanding cases, a dark greenish-yellow discoloration. It affects the skin of the whole body, but is most marked on the trunk and proximal parts of the limbs.

Even before the skin is affected, the yellowing is seen in the mucous membranes and should be sought in the conjunctivae and soft palate. Intolerable itching is common and is probably due to bile salts, as it may precede the actual pigmentation of the skin and mucosae.

The excess of bile pigments (conjugated bilirubin) in the blood leads to their appearance in the urine, which may be visibly bile-stained or in which bile may be detected by special tests. The lack of the normal flow of bile into the duodenum deprives the faeces of one of their colouring constituents and further interferes with the digestion and absorption of fats because of the lack of bile salts. As a result, the
faeces have a lighter colour than normal and are often clay-coloured. In complete obstruction, urobilinogen is absent from the urine.

It must be stressed that more than one of the three types of jaundice can exist in the same patient. Intra-hepatic obstruction is common in hepatocellular jaundice, and obstruction due to pigment stones may also occur in haemolytic jaundice. Moreover, liver-cell dysfunction can result from the damming back of bile and ascending infection in obstructive jaundice.

Laboratory investigations are therefore needed for the precise diagnosis of jaundice and for the differentiation of the three types.

Table 8.

The differential diagnosis of jaundice

<table>
<thead>
<tr>
<th></th>
<th><strong>Prehepatic</strong> (haemolytic)</th>
<th><strong>Hepatocellular</strong></th>
<th><strong>Posthepatic</strong> (obstructive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanism</td>
<td>Increased bilirubin formation</td>
<td>Hepatocellular failure</td>
<td>Bile duct obstruction</td>
</tr>
<tr>
<td>Common cause</td>
<td>Haemolysis, Gilbert's syndrome</td>
<td>Virus hepatitis. Drugs, e.g. chlorpromazine. Chronic liver disease. Cirrhosis</td>
<td>Gallstones. Carcinoma of pancreas</td>
</tr>
<tr>
<td>Past history</td>
<td>May be previous attacks or a family history</td>
<td>Contact with similar case History of injections or of taking hepatotoxic drugs</td>
<td>May be previous attacks (stone)</td>
</tr>
<tr>
<td>Mode of development</td>
<td>Rapid, with anaemia and sometimes fever and rigors. Periodic attacks</td>
<td>After a period of anorexia and nausea; gradual onset and recovery</td>
<td>After an attack of pain Rapid and sometimes intermittent (stone). Insidious and progressive (carcinoma)</td>
</tr>
<tr>
<td>Pruritus (bile salt retention)</td>
<td>Absent</td>
<td>Occasional (if cholestasis). Primary biliary cirrhosis</td>
<td>Present</td>
</tr>
<tr>
<td>Skin colour</td>
<td>Faint lemon-</td>
<td>Yellow</td>
<td>Brilliant or dark</td>
</tr>
<tr>
<td></td>
<td>yellow</td>
<td></td>
<td>yellow</td>
</tr>
<tr>
<td>----------------------</td>
<td>--------</td>
<td>----------------------</td>
<td>--------</td>
</tr>
<tr>
<td>Faeces</td>
<td>Normal</td>
<td>Pale (if cholestasis)</td>
<td>Pale</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>Nil</td>
<td>Nil</td>
<td>May be palpable in carcinoma; not with stone</td>
</tr>
<tr>
<td>Enlarged spleen</td>
<td>Usually</td>
<td>Sometimes</td>
<td>Nil</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Unconjugated</td>
<td>Mixed</td>
<td>Conjugated</td>
</tr>
<tr>
<td>Serum alkaline phosphatase</td>
<td>Normal</td>
<td>Raised (if cholestasis)</td>
<td>Markedly raised</td>
</tr>
<tr>
<td>Tests for hepatocellular function</td>
<td>Normal</td>
<td>Grossly abnormal</td>
<td>Slightly abnormal</td>
</tr>
<tr>
<td>Tests for haemolysis</td>
<td>Positive</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

**Cytolytic syndrome**

Cytolytic syndrome is characterized by increase of serum transaminase, which reflects level of hepatocytes necrosis in acute and chronic hepatic diseases of different etiology. The peak increase is detected in acute viral hepatitis.

Transaminases are divided into 2 types: aspartate aminotransferase (AST) and alanin aminotransferase (ALT).

AST is an enzyme that is present in hepatocytes and myocytes (both skeletal muscle and cardiac).

Elevations in AST are most commonly a reflection of hepatocellular injury.

But they may also be elevated in myocardial or skeletal muscle injury.

**Laboratory Diagnosis Of Acute Myocardial Infarction**

Creatine kinase (CK) emerged as the primary indicator of MI. Total CK starts to rise within 3 to 8 hours after MI, peaks at 10 – 24 hours and returns to normal by 3 – 4 days. It can be markedly elevated with skeletal muscle trauma or brain injury. Other skeletal muscle diseases including dystrophy, myopathy and myositis show
increase. Electrical cardioversion shows an increase as does cardiac catheterisation without myocardial damage.

Total CK is increased in hypothyroidism, stroke, surgery and in patients with convulsions who have skeletal muscle damage. Therefore total CK is not specific for MI. Three isoenzymes in blood comprise the total fraction of these: CK-MM (CK-1) (Skeletal Muscle) > 95% of total, CK- MB (CK-2) (Myocardial) 1 and is rarely greater than 1.3. LDH-1 to LDH-2 rise above base line at around 10 hours following myocardial infarction, peak at about 24 to 48 hours and stay elevated in blood for up to 14 days post MI.

The Aspartate Aminotransferase (AST) is principally found in liver, myocardium, skeletal muscle and kidney. AST rises and falls after AMI in a pattern similar to that of CK - slightly later and slightly less when activities are expressed as multiples of the upper reference limit. It can be elevated in patients with skeletal muscle disease, pulmonary emboli, hepatic disease and also by intramuscular injections. However, in the patient with a minor infarction or a non-Qwave infarction there may be no change in the AST values. According to the National Academy of Clinical Biochemistry (NACB), do to low specificity and the availability of more specific alternative biomarkers of necrosis, total CK, Lactate Dehydrogenase (LDH) and Aspartate Aminotransferase (AST) should no longer be used for the diagnosis of MI. So, the imperfect sensitivity and specificity of the traditional enzymatic markers for the detection of myocardial injury are well known. The ECG shows in approximately 60% of patients ST segment change within seconds of the ischemic evidence. However, the ECG can be inconclusive in the remaining 40% of cases, therefore, showing a globally low sensitivity. Chest pain is an unreliable indicator: up to 33% of patients with AMI may have no chest pain and are clinically silent. On the other side, many people experience chest pain, resulting from plaque rupture and subsequent deposit of platelets but do not have an AMI. This occlusion may not be large enough to cause an acute infarct but it may cause minor myocardial damage with a result – leak of Cardiac Troponins from the damaged myocytes. They can be detected and measured long before the
development of traditional AMI. It was thus necessary to establish a new diagnostic group with troponin-positive patients who did not meet the WHO criteria for AMI. This new category became known as ‘acute coronary syndrome’ or ACS. It includes AMI as the most serious form of ACS. The new ACS model required new diagnostic criteria to classify AMI and the other ACS stages. In 1999 the U.S. National Academy of Clinical Biochemistry published its guidelines for cardiac marker testing. This guideline specified that the cardiac troponins are the most specific and sensitive available biochemical cardiac markers. While redefining myocardial infarction, recommendations by the Joint European Society of Cardiology and American College of Cardiology (ESC/ACC) Committee in 2000 stated that ECG was no longer sufficient to diagnose AMI. Final diagnose of ACS, according to this committee, should depend on cardiac biomarkers, especially cardiac troponins.

Troponin is a complex of three proteins on the thin filaments of skeletal and cardiac muscle fibres. During muscle contraction troponin complex regulates the interaction between the thick and thin filaments. This complex consists of troponin T (TnT; Tropomyosin binding), troponin I (TnI, Inhibitory component) and troponin C (TnC, Calcium binding component). Troponin C is identical in skeletal and cardiac muscle but the amino acid sequences of troponin T and troponin I found in cardiac muscle are different from that of the troponins in skeletal muscle. These isoforms of cardiac troponins, cTnT and cTnI, are very specific to cardiac muscle and their presence in blood indicates cardiac tissue necrosis. Also, cardiac troponins have been established as sensitive and specific markers of minor myocardial lesions in patients with acute coronary syndrome. Because of this specificity, cardiac troponin T or I is now the preferred cardiac marker. Both troponins are considered to be acceptable. Troponin T is a cardio-specific polypeptide mostly bound to contractile elements of myocardial cells, but with small amounts also present free in the cytoplasm. Cytosolic cardiac troponin T is released within the first few hours after infarction. Release of myofibrillar cTnT occurs more slowly, over a period of days. This biphasic release results in an early
rise in serum levels (3-4 hours after the infarct) which is sustained for 10 days or more. This makes it a very useful marker. Minor elevations occur in unstable angina. Three distinct tissue-specific isoforms of Troponin I have been identified: two in skeletal muscle and one in cardiac muscle.

Cardiac troponins T and I begin to rise 4-8 hours after myocardial damage, peak at approximately 12 - 24 hours, and remain elevated for up to 10 days. It should be remembered that cardiac troponins reflect myocardial damage but do not indicate its mechanism. Thus, an elevated value in the absence of clinical evidence of ischemic heart disease should prompt a search for situations in which various degrees of myocardial injury may be present. Elevation of cardiac troponins without ischemic heart disease can be observed in:

- Acute rheumatic fever;
- Amyloidosis;
- Cardiac trauma (including contusion, cardioversion, cardiac surgery);
- Cardiotoxicity from cancer therapy;
- Chronic renal failure;
- Congestive heart failure;
- Hypertension;
- Myocarditis;
- Postoperative noncardiac surgery;
- Pulmonary embolism;
- Sepsis

**Diagnosis of diabetes mellitus**

- A fasting venous plasma glucose level greater than 7.8 mmol/litre (140 mg/dl) on more than one occasion; or
- A 2-hour (plus one other) venous plasma glucose level in excess of 11.1 mmol/litre (200 mg/dl) in a formal 75 g oral glucose tolerance test (GTT).

Impaired glucose tolerance
Impaired glucose tolerance (IGT) is often classified as «chemical», «borderline» or «latent» diabetes. It is defined as the finding of a fasting venous plasma glucose level below 7.8 mmol/litre, and a 2-hour sample, after an oral GTT, with levels between 7.8 and 11.1 mmol/litre.

Urine testing for glucose is still widely used, but glucose will be found in the urine only when it rises above the renal threshold (usually about 10 mmol/litre); urine tests are simple and cheap; enzyme strip tests are specific for glucose.

- Urine testing for ketone bodies is also simple; the presence of ketones suggests loss of control.
- Glycosylated haemoglobin and other proteins: measurement of these proteins reflects the degree of diabetic control in the previous 4-6 weeks and is of value in long-term management and control.

**Urinalysis**

- collection of urine specimens – first voided morning (most common) – random (for emergency) – clean-catch, midstream (for urine culture)
- Attention: need to be examined within 1 hour
- urine specimens examination
  - physical (appearance, volume, specific gravity (SG)) – chemical
  - microscopic examination – urine for culture and sensitivity

**Urine specimens examination physical appearance**

- Color – normal, pale to dark yellow (urochrome)
  - abnormal
  - some drugs cause color changes
  - red urine (hematuria, hemoglobinuria, myoglobinuria, pseudohematuria)
  - yellow-brown or green-brown urine (bilirubin: obstructive jaundice)
- Clarity – normal, clear
  - abnormal, cloudy
  - crystals or nonpathologic salts
  - phosphate, carbonate in alkaline urine
  - uric acid in acid urine
• various cellular elements (leukocytes, RBCs, epithelial cells)

• Appearance - causes of discoloration of urine include

• cholestatic jaundice, haemoglobinuria, drugs such as rifampicin, use of fluorescein or methylthioninium chloride (methylene blue), and ingestion of beetroot.

  o microscopic hematuria (urinary tract source (urethra or bladder, prostate, ureter or kidney), non-urinary tract source (vagina, anus or rectum)

  o pseudohematuria (myoglobinuria, hemoglobinuria, phenolphthalein laxatives, phenothiazines, porphyria, rifampin, pyridium, bilirubinuria, phenytoin, pyridium, red diaper syndrome, foods (beets, blackberries, rhubarb)

  o causes of asymptomatic gross hematuria (acute cystitis, bladder cancer, benign prostatic hyperplasia, nephrolithiasis, benign essential hematuria, prostatitis, renal cancer, pyelonephritis, prostate cancer, urethral stricture)

• Volume - CKD or diabetes insipidus, impairment of concentrating ability requires increased volumes of urine to be passed, given the same daily solute output. normal adult average – (400 – 2000) ml/24h

  • increase average (polyuria) – > 2000 ml/24h

    – physiological (water intake, some drugs, intravenous solutions)

    – pathologic (CKD, diabetes mellitus, diabetes insipidus)

  • decrease average (oliguria - < 400 ml/24h, anuria - < 100ml/24h)

    – prerenal (hemorrhage, dehydration, congestive heart failure)

    – postrenal (obstruction of the urinary tract, may be stones, carcinoma)
– renal parenchymal disease (acute tubular necrosis, chronic renal failure)

- Specific gravity -density of the urine (compares the density of urine to the density of water)

• normal average in adults: 1.001 - 1.040

• increased (dehydration, fever, vomiting, diarrhea, diabetes mellitus, other glycosuria, congestive heart failure, syndrome of inappropriate ADH secretion (SIADH), adrenal insufficiency)

• decreased (urine volume↓ and SG↑) in diabetes insipidus (urine volume↑ and SG↓)

- urine PH: normal 5 - 9 (depends on diet), increased (alkaline urine: drugs (sodium bicarbonate), classic renal tubular acidosis, alkalosis (metabolic or respiratory), decreased (acid urine: drugs (ammonium chloride), acidosis (metabolic or respiratory)

- Proteinuria

-Most reagent strips can detect protein if albuminuria exceeds 300 mg/d. They react primarily with albumin and are relatively insensitive to globulin and Bence Jones proteins. > 3.5 g/ day: nephrotic syndrome.

- Timed 24-hour urinary excretion rates provide the most precise measure of microalbuminuria. - 30-300 mg/ day. - can be early indicator of DM.

- Glucose

Renal glycosuria is uncommon, so that a positive test for glucose always requires exclusion of diabetes mellitus.
• Bacteriuria -based on the detection of nitrite produced from the reduction of urinary nitrate by bacteria and also for the detection of leucocyte esterase, an enzyme specific for neutrophils.

• Microscopy

- White blood cells. The presence of 10 or more WBCs per cubic millimetre in fresh mid-stream urine samples is abnormal and indicates an inflammatory reaction within the urinary tract such as urinary tract infection (UTI), stones, tubulointerstitial nephritis, papillary necrosis, tuberculosis and interstitial cystitis.

- Red cells. The presence of one or more red cells per cubic millimetre in unspun urine samples results is abnormal.

  Erythrocytes in the urinary sediment may be:

  - Isomorphic (unmodified) – yellowish-greenish color due to hemoglobin having a disk shape or a biconcave lens. The reaction of such urine is usually slightly acidic (pH 6.5), neutral (pH 7.0) or slightly alkaline (pH of 7.5).

  - Dysmorphic (changed) – this is usually red blood cells lack hemoglobin, have no color, they form single-circuit or two-circuit, substantially less than normal erythrocyte (Fig.28; Fig.19, see color insert). These erythrocytes occur in the urine with low specific gravity, an acid reaction (pH 5-6) or prolonged their stay in the urine.

  The appearance of unmodified erythrocytes in the urine is characteristic in lesions of the urinary tract (cystitis, urethritis, urolithiasis).

  Modified or dysmorphic erythrocytes are formed when the filtration through the renal filter, which increases its permeability. Detecting in the sample a large number dysmorphic erythrocytes suggests a renal cause of hematuria.
Fig. 28 Different types of dysmorphic erythrocytes

(A- normal erythrocyte)

- Casts (cylindrical bodies, moulded in the shape of the distal tubular lumen) may be hyaline, granular or cellular.

- Coarse granular casts occur with pathological proteinuria in glomerular and tubular disease.

- Red-cell casts – even if only single – always indicate renal disease (Fig.20, see color insert).

- White cell casts may be seen in acute pyelonephritis (Fig.21, see color insert). They may be confused with the tubular cell casts that occur in patients with acute tubular necrosis.
The test of urine by Nechiporenko — quantitative determination of urine leukocytes, erythrocytes and cylinders for the differential diagnosis of glomerulonephritis and pyelonephritis.

Diagnostic value.

Normal values: leukocytes — to 2000 /ml; red blood cells — to 1000 /ml; cylinders — up to 20 /ml. In pathology: pyuria increase in the number of leukocytes (pyelonephritis, cystitis, urethritis); erythrocyturia (hematuria) - the appearance of red blood cells in the urine (glomerulonefritis, urolithiasis); cylindruria - appearance in the urine protein or cellular formations of tubular origin (casts), having a cylindrical shape and a varying amount (excessive exercise, condition after an epileptic seizure, hypertension, valvular heart disease, cardiac decompensation, toxemia of pregnancy, viral hepatitis, gout, etc.).

Kidneys functional tests

Urine test by Zimnicky is carried out to assess the concentration of kidney function.

The main indications for use: the clinical signs of renal failure, chronic glomerulonephritis, chronic pyelonephritis, diagnosis of diabetes insipidus, hypertension.

For the test prepare 8-10 containers with labels. Each of them put a sequence number (from 1 to 8, two banks — spare) and indicate the surname, initials of the patient, the room number and the interval of time over which urine must be collected in each container. Urine is collected during the day (24 h): for every 3 hours, including at night, the patient urinates into a separate container.

Rules for collection of urine.

1) To 6 a.m. the patient empties the bladder (urine not collected);

2) Next, the patient is consistently collects urine in 8 containers; depending on the frequency of urination per container he urinates once or several times, but only for 3 hours. If during this period of time the patient has no urge to urinate, nurse
reminds him of the need to empty the bladder (if urine is missing the container remains empty); if the container is filled with urine before the expiration of the 3-hour period, the patient takes a container without it and urinates into it (he must inform the nurse);

3) the next morning all containers must be sent to the lab, completing required documentation.

Diagnostic value.

Normal values: 50-250 ml per serving, specific gravity - 1.005 - 1.028, the largest share - at least the portions of urine, 3:1 is the ratio of daytime and nighttime diuresis.

In pathology reveal hyposthenuria (decrease in specific gravity), urine equal portions - hypoizostenuriya, nocturia (increased amount of urine secreted during the night).

**Methods of revealing glomerular filtrating rate**

- **GFR** (glomerular filtrating rate) is a test of how much the kidneys are filtering
  - Norm = about 100 mL/min (This means that the kidneys are removing all the creatinine found in 100mls of blood every minute)
  - Measured GFR - Injecting a tiny amount of a radioactive substance and measuring how quickly it disappears from the blood, or appears in the urine, is used to calculate GFR
  - eGFR - Using blood tests, age, sex, and sometimes other information to estimate the GFR from the MDRD equation (eGFR). This isn't as good as measuring it, but is much simpler as it requires just one blood test.
  - Creatinine clearance (blood creatinine measurements by collecting urine for 24 hours and measuring how much creatinine is in the urine at the same time as finding out how much is in the blood. *If any urine produced during the 24 hours is not collected the result will not be accurate*).
– Abbreviated MDRD (Modification of Diet in Renal Disease) equation for eGFR

  • eGFR (ml/min/1.73 m²) = 186 x (S.cr)-1.154 x (age)- 0.203 x(0.742 if female) x (1.210 if Black). Normal GFR is about 100ml/minute/1.73m².

– Cockroft-Gault equation (in fact gives the creatinine clearance (CCr))

  • CCr (ml/min) = (140-age) x lean body weight (kg) x 0.85 (if female) / 72xS.cr (mg/dl). Normal creatinine clearance is about 100ml/minute.

Table 5. Staging of CKD based on eGFR

<table>
<thead>
<tr>
<th>Stage</th>
<th>eGFR (ml/min)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;90</td>
<td>Damage with normal or increased GFR</td>
</tr>
<tr>
<td>2</td>
<td>60-89</td>
<td>Mild decrease in GFR</td>
</tr>
<tr>
<td>3A</td>
<td>45-59</td>
<td>Moderate decrease in GFR</td>
</tr>
<tr>
<td>3B</td>
<td>30-44</td>
<td>Moderate decrease in GFR</td>
</tr>
<tr>
<td>4</td>
<td>15-29</td>
<td>Severe decrease in GFR</td>
</tr>
<tr>
<td>5</td>
<td>&lt;15</td>
<td>Kidney failure</td>
</tr>
<tr>
<td>5D</td>
<td>&lt;10</td>
<td>Dialysis</td>
</tr>
</tbody>
</table>

Tests to detect *H. pylori*

For patients in whom diagnosis will alter treatment, diagnostic tests to detect *H. pylori* consist of noninvasive and invasive techniques.

Noninvasive testing is less expensive and does not require endoscopy. Laboratory and office-based serologic assays most frequently use technology to detect IgA and IgG antibodies to *H. pylori*. Sensitivity and specificity are > 85% for detecting initial *H. pylori* infection.

Urea breath tests use 13C- or 14C-labeled urea po. In an infected patient, the organism metabolizes the urea and liberates labeled CO2, which is exhaled and can be quantified in breath samples taken 20 to 30 min after ingestion (The sensitivity and specificity are > 90%). Urea breath tests are well suited for confirming eradication of the organism after therapy.
Invasive testing requires gastroscopy and mucosal biopsy and should be reserved for patients with an a priori indication for endoscopy. Histologic staining of gastric mucosal biopsies has a sensitivity and specificity > 90%. Because it is accurate, easy to perform, and relatively inexpensive, RUT should be considered the invasive diagnostic method of choice.

**Measurement of Pancreatic Enzymes**

A hallmark of pancreatic disease is an increased level of pancreatic enzymes in the blood.

Although a number of extrapancreatic sources may supply pancreatic or salivary-type amylase to the serum, for practical purposes in the patient with abdominal pain, clinicians should consider pancreatic disease first whenever the serum amylase is elevated.

Ordinarily, the serum amylase level rises within a few hours after the onset of acute pancreatitis to levels 10 to 12 times normal or more, rapidly dropping to normal within 2 or 3 days. In acute pancreatitis, the serum amylase level tends to increase in parallel with the lipase, but decrease more rapidly than the lipase.

The *urinary amylase* tends to remain elevated for a longer period than serum amylase and may be elevated for 5 to 7 days after the serum amylase level has returned to normal. Their clinical utility is largely supplanted by serum lipase levels and by imaging techniques.

*Chronic pancreatic disease* is reflected in deterioration of pancreatic endocrine as well as exocrine function, with disordered glucose tolerance and evidence of malabsorption. It is often necessary to carry out a full malabsorption workup to pinpoint the origin of steatorrhea in the pancreas.

*Stool Trypsin and Chymotrypsin* The quantitative measurement of stool trypsin and chymotrypsin appears to be popular in the diagnosis of chronic pancreatic insufficiency, but studies of stool trypsin and chymotrypsin are of little diagnostic value in the patient with mild pancreatic insufficiency.
Pancreatic Secretion. The direct study of pancreatic secretion can be accomplished in two ways, neither of which is currently very popular.

(1) The secretin test is the more standard and more sensitive, though detecting alterations in pancreatic function sometimes so slight as to lack clinical reflection.

(2) The Lundh test meal evaluates by direct aspiration of a test meal from the duodenum the status of the digestive process. The test is reliable only when there is a moderate diminution of pancreatic secretion.

Because the secretin test is less physiologic and its stimulus of greater potency, it will usually display lesser degrees of pancreatic dysfunction than the Lundh test meal.
V. Tests

Tests on the theme “Algorithm of interpreting ECG. Electrophysiological bases of ECG (electrocardiography). ECG in hypertrophy”

1. The division of the conducting system in normal non pacemaker:
   a) the Sinus node
   b) Atrioventricular node
   c) Right leg of bundle branch block
   d) Left leg of bundle branch block

2. Enhanced single-pole leads, it -
   a) I, II, III
   b) aVF, aVL, aVF,
   c) V1-V6

3. Specify which interval of the ECG is called electrical systole of the ventricles:
   a) PQ
   b) QRS
   c) QT
   d) ST

4. One of the standard speeds ECG record is:
   a) 30 mm/s
   b) 25 mm/min.
   c) 50 mm/min.
   d) 50 mm/s

5. Chest, or precordial, leads of call diversion:
   a) I, II, III
   b) aVR, aVL, aVF
   c) V1-V6
   d) A, D, I

6. Specify one of the signs of sinus rhythm:
   a) positive prong R in lead aVR
   b) the amplitude of P wave > 2.5 mm
   c) positive prong R in leads I, II, aVF, V2-V6
   d) the duration of the PQ interval less than 0.12 with

7. What part of the ECG reflects the depolarization process of the Atria:
   a) prong R
   b) PR interval
   c) the QRS complex
d) ST segment

8. Specify the normal duration of the QRS complex:
   a) not more than 0.06 with
   b) from 0.06 to 0.12 with
   c) from 0.06 to 0.10 with
   d) from 0.08 to 0.10 with

9. The normal position of the electrical axis of the heart corresponds to the angle alpha:
   a) from 0 to +30°
   b) +30 to +70°
   c) from +70 to +90°
   d) from 0 to -90°

10. The horizontal position of the electrical axis of the heart corresponds to the angle alpha:
    a) from 0 to +30°
    b) from +30 to +70°
    c) from +70 to +90°
    d) from 0 to -90°

11. ECG signs of sinus bradycardia:
    a) maintaining correct sinus rhythm with a heart rate less than 60 / min
    b) a wide QRS complex
    c) lack of teeth R on the ECG
    d) all of the above

12. Signs of hypertrophy of the right atrium is:
    a) two widened prong R in lead II
    b) high pointed prong R in lead II
    c) negative prong R in II and III leads
    d) negative prong R in aVR

13. Hypertrophy of the right ventricle on the ECG are observed:
    a) prong deep S in V1,V2, tall R in V5,V6
    b) high R-wave in V1,V2 and deep S in V5,V6
    c) deep tine S in V1,V2 and a negative prong T in V5,V6
    d) high R-wave in aVL and deep S in prong III, and aVF leads

14. Signs of hypertrophy of the left atrium are the:
    a) two widened prong R in lead II
    b) high pointed prong R in lead II
    c) negative prong R in II and III leads
d) negative prong R in aVR

15. Hypertrophy of the right ventricle on the ECG are observed:
   a) prong deep S in V1, V2, tall R in V5, V6
   b) high R-wave in V1, V2 and deep S in V5, V6
   C) deep tine S in V1, V2 and a negative prong T in V5, V6
   d) high R-wave in aVL and deep S in prong III, and aVF leads

16. On ECG, the RR interval is 12 mm, recording speed 25 mm/s. What is heart rate?
   a) 64 beats/min, normogastria
   b) 64 beats/min, bradville
   c) 125 BPM, tachysystole
   d) 125 BPM, tachysystole

17. On ECG there are positive z. R in I, II lead, the PQ interval 0.15, consecutive RR intervals: 12 mm, 32 mm, 18 mm, About 11 mm. what pathology can be discussed?
   a) this is a normal ECG
   b) sinus arrhythmia
   C) sinus bradycardia
   d) sinus tachycardia,

18. The patient is 56 years old. ECG electrical axis of the heart rejected to the left, z. R in lead V5, mm 27, h. S in lead V1 13 mm in leads I, aVL, V5, V6 there are signs of reduction of ST segment below the isoline and negative T. So what kind of pathology can we talk?
   a) right ventricular hypertrophy
   b) right ventricular hypertrophy with signs of systolic overload of the
   C) left ventricular hypertrophy
   d) left ventricular hypertrophy with signs of systolic overload of the

19. Patient diagnosed with right ventricular hypertrophy. For electrical axis of the heart in this case is typical:
   a) normal position
   b) horizontal position
   C) deviation to the left
   d) deviation to the right

20. Which statement is true for the vertical position electric axis?
   a) RII > RI > RIII
   b) RI > RII > RIII
   c) RIII > RII > RI
   d) none of the above
Tests on the theme “ECG in ischemic heart disease (IHD)”

21. What part of the ECG reflects the process of ventricular repolarization:
a) wave R  
b) the QRS complex  
c) wave T  
d) QT interval

22. Enter the correct sequence of development stages of myocardial infarction:
a) sharp, acute, subacute, scar, stage of ischemia,  
b) ischemic, acute, acute, subacute, scar,  
c) ischemic, sharp, acute, scar, subacute,  
d) acute, scar, acute, subacute, ischemic,

23. Signs subepicardial ischemia is:
a) depression of ST segment,  
b) negative z. T  
c) ST segment elevation,  
d) right a) and C)

24. Signs of the upper subendocardial ischemia is:
a) ST segment elevation,  
b) negative z. T  
c) depressed ST segment,  
d) none of the above

25. Duration of acute stage of myocardial infarction is:
a) up to 30 minutes  
b) up to 3 days  
c) to 3 weeks  
d) up to 3 months

26. Which of the following biochemical parameters is NOT a marker of myocardial necrosis?
a) KFK-MV  
b) troponin I, troponin T  
c) myoglobin  
d) bilirubin

27. Diagnostically significant increase in the level of CPK-MB is:
a) greater than 0.10 and 0.13 u/l  
b) more than 1.0 to 1.3 u/l
c) more than 10-13 u/l
d) more than 100-130 u/l

28. The most specific and sensitive marker of myocardial necrosis is:
a) myoglobin
b) troponin I, T
C) KFK
d) AST

29. signs of a pathological Q wave
a) the increase in the length of more than 0.03 to
b) increase in the amplitude of more than ¼ z. R
c) double amplitude
d) right a) and b)

30. One of the absolute contraindications to the stress tests is:
a) chest pain
b) physical fatigue
c) myocardial infarction in the first 48 hours
d) diabetes mellitus

31. Diagnostically significant ST segment elevation is considered to be:
a) any ST segment elevation in two or more ECG leads
b) elevation of ST segment elevation of 1 mm or more in at least one ECG lead
c) ST elevation 2 mm or more in at least two adjacent ECG leads
d) elevation of ST segment elevation of 1 mm or more in at least two adjacent ECG leads

32. Diagnostically significant depression of the ST segment is considered to be:
a) any depression of the ST segment in two or more ECG leads
b) ST depression of 1 mm or more in at least one ECG lead
C) ST depression 2 mm or more in at least two adjacent ECG leads
d) ST depression of 1 mm or more in at least two adjacent ECG leads

33. On the ECG in leads V5, V6 was depression of the ST segment with a depth of 2 mm. This is a sign:
a) the upper subendocardial ischemia of the lateral wall of the left ventricle,
b) subepicardial ischemia of the lateral wall of the left ventricle,
C) the upper subendocardial ischemia of the anterior wall of the left ventricle,
d) subepicardial ischemia of the anterior wall of the left ventricle,

34. On the ECG in leads I, aVL, V1-V4 was depression of the ST segment with a depth of 2 mm. This is a sign:
a) ischemia of the anterior wall of the left ventricle
b) infarction of the anterior wall of the left ventricle
C) ischemia of the lateral wall of the left ventricle
d) infarction of the lateral wall of the left ventricle

35. On the ECG in leads II, III, aVF was signs of heart attack. What is the localization of necrosis:
a) the tip of the left ventricle
b) the side wall
c) Anterior part of intraventricular septum
d) inferior wall

36. ECG has ST-segment elevation, the ST segment merges with T, high R-wave, Q-wave shallow. What stage of myocardial infarction corresponds to that description?
a) ischemic
b) an acute
C) acute
d) subacute

37. On the ECG in leads III, aVF was discordant changes in the form of depression of the ST segment. What is the localization of necrosis?
a) the front wall of the left ventricle
b) inferior wall of the left ventricle
C) lateral wall of the left ventricle
d) none of the above

38. For myocardial infarction of the lateral wall of the left ventricle corresponding to the characteristic ECG changes in leads:
a) II, III, aVF
b) I, aVL,
C) V1-V4
d) V5-V6

39. For scar stage of myocardial infarction characteristic
a) the disappearance of the pathologic Q wave
b) elevated ST segment above contours
c) positive or flattened T wave
d) the narrowing of the Q wave to 0.02

40. ECG chest leads, with high pointed h. T in the form of triangle. This is a sign
a) early (ischemic) stage of myocardial infarction,
b) the upper subendocardial ischemia
c) subepicardial ischemia
d) right a) and b)
Tests on the theme “Cardiac arrhythmia”

1. What part of the ECG reflects the time required for the passage of the impulse from the atrial to the ventricles:
   a) wave R
   b) PQ interval
   C) the QRS complex
   d) ST segment

2. What part of the ECG reflects the depolarization process of the Atria:
   a) wave R
   b) PR interval
   C) the QRS complex
   d) ST segment

3. Purkinje Fibers:
   a) carry out the excitation from the apex of the heart to the AV node
   b) carry out the wave propagation of excitation in the ventricular myocardium
   C) carry out the spread of the excitation wave in myocardium of the Atria
   d) connect the sinus node and AV-node

4. Select the most typical changes of the ECG for atrial fibrillation:
   a) Missing tooth R
   b) Negative prong P before QRS complex
   c) the Negative prong R behind QRS complex
   d) Notes the same duration of intervals R-R

5. ECG – there are no teeth R, instead of f waves of different amplitude and duration, are best visible in leads II, III, aVF, V1-V2. What is the rhythm disturbance most likely in the patient?
   a) Atrial fibrillation
   b) atrial premature beats
   C) Atrioventricular block I degree
   d) Atrioventricular block II degree

6. For any extrasystoles characterized by a complete compensatory pause:
   a) atrial
   b) atrioventricular connection
   c) ventricular
   d) right a) and b)
7. For most of ventricular extrasystole is characterized by:
   a) absence of P wave, regular QRS complex, incomplete compensatory pause
   b) change in shape of P wave, regular QRS complex, incomplete compensatory pause
   C) the absence of P wave, widened QRS, incomplete compensatory pause
   d) absence of P wave, widened QRS, full compensatory pause

8. For atrial arrhythmia the most typical:
   a) absence of P wave, regular QRS complex, incomplete compensatory pause
   b) change in shape of P wave, regular QRS complex, incomplete compensatory pause
   C) the absence of P wave, widened QRS, incomplete compensatory pause
   d) absence of P wave, widened QRS, full compensatory pause

9. In paroxysmal supraventricular tachycardia are:
   a) regular rhythm rate is 140-220 / min, the teeth R are absent, the QRS complex is not changed
   b) the rhythm is wrong, all the intervals R-R have a different value, the prongs of R are missing, the teeth R different amplitude
   C) the rhythm is wrong, instead of teeth, R - large, the same forms of atrial waves with a frequency of 300 per minute, complexes QRS are not changed
   d) the rhythm is right, instead of teeth, R - large, the same forms of atrial waves with a frequency of 300 per minute, complexes QRS are not changed

10. Typical ECG signs of ventricular paroxysmal tachycardia:
    a) heart rate is 130 per minute; QRS normal shape
    b) heart rate - 120 per minute; QRS is 0.10 sec
    c) heart rate - 150-200 per minute; QRS and 0.12 seconds; strain
    d) heart rate - 120 per minute; QRS is widened; R - strain

11. With atrial flutter rate of Atria reaches:
    a) 100-200 per minute
    b) 200-400 per minute
    C) 400-500 per minute
    d) 500-600 per minute

12. What is the difference between fibrillation waves, f waves flutter F:
    a) their frequency
    b) form
    C) regularity
    d) all of the above

13. What form of atrial flutter are called correct.
a) at normal heart rate  
b) the presence of F waves of the same shape  
c) regular conduction of impulses from the Atria to the ventricles (every second, every third, etc.)  
d) all of the above  

14. On ECG there are signs of atrial fibrillation. This:  
a) positive prong P in all leads  
b) the prong R is missing in all leads, instead - krupnopanelnoye, the same waveform F  
c) prong P is absent in all leads, instead - a low-amplitude, different wave forms f  
d) the R-R interval is the same in all leads  

15. Life threatening condition is:  
a) sinus arrhythmia  
b) atrial premature beats  
C) atrial fibrillation  
d) ventricular fibrillation  

16. On ECG in men 55 years, the wave P is not recorded. Have atrial F waves with a frequency of 250 per minute, equal in length, shape and height ("teeth of a saw"), are clearly visible in leads II, III, avF, V1. Intervals R-R same. The QRS complex is not changed. What is the rhythm disturbance in the patient?  
a) atrial Fibrillation  
b) atrial Flutter, the right shape  
C) Sinus arrhythmia  
d) Atrioventricular block I degree  

17. On ECG in men 75 years the wave P is not recorded. Have atrial F waves with a frequency of 270 per minute, equal in length, shape and height, consist of steep upward and shallow downward knee (teeth of saw), are clearly visible in leads II, III, avF, V1. Intervals R-R are different. The QRS complex is not changed. What is the rhythm disturbance in the patient?  
a) atrial Fibrillation  
b) atrial Flutter, irregular shape  
C) Sinus arrhythmia  
d) Atrioventricular block I degree  

18. The woman 34 years old suffering from diffuse toxic goiter, heart rate – 128 in 1 min, PS – 78 in 1 min, arrhythmic. ECG: RR - miscellaneous, f waves in II, III avF , V1 ; wave P is missing. What is the rhythm disturbance most likely in a patient?  
a) atrial fibrillation  
b) atrial flutter
C) atrial premature beats  
g) ventricular premature beats

19. Female 66 years old, during a routine ECG loses consciousness, ECG marked by the appearance of frequent, regular and similar in shape and amplitude of the waves, reminiscent of sinusoidal curve, a frequency of about 200 per minute. What is the rhythm disturbance most likely in a patient?
a) atrial fibrillation  
b) atrial flutter  
C) flickering of ventricles  
d) atrial flutter ventricular

20. The patient the feeling of "tumbling" and bated breath, a heartbeat. On ECG: rhythm is incorrect, the individual ventricular complexes are widened to 0.12 sec, strain, R-R before them shortened, and the pause after them, elongated prong R in front of them. What is the rhythm disturbance in this patient?
a) Ventricular premature beats  
b) Atrioventricular extrasystole  
C) atrial premature beats  
d) atrial fibrillation

Tests on the theme “ECG diagnosis of sinus node automatism disorders, disordered myocardial conduction and excitability”

1. Atrioventricular block is:
a) violation of the impulse through the Atria  
b) violation of the impulse from the Atria to the ventricles  
C) violation of the pulse in the ventricles  
d) violation of the impulse from the ventricles to the Atria

2. Prolongation of the interval P-Q means:
a) heart rate was significantly reduced  
b) the heart rate slowed considerably  
c) impulse conduction from the Atria to the ventricles is slower than normal  
d) in the myocardium, there is an ectopic focus of excitation

3. Prolongation of the interval P-Q is observed at:
a) Atrio-ventricular blockade of I degree  
b) With complete atrio-ventricular blockade  
C) Atrial arrhythmia  
d) Ventricular arrhythmia
4. Under the influence of impulses from a center of reduced automaticity of the Atria with complete AV-blockade:
a) Sinus node
b) the Upper division of the atrioventricular node
c) the Middle division of the atrioventricular node
d) the Lower portion of the atrioventricular node

5. Under the influence of impulses from a center of reduced automaticity in the ventricles during complete AV-blockade:
a) Sinus node
b) Atrioventrikulyarnogo site
c) of the lower part of the bundle of his and Purkinje fibers
d) is true b) and C)

6. When atrioventricular block I degree interval P-Q is equal to:
a) 0,12 seconds
b) 0,14 seconds
c) 0,16 seconds
d) is 0.26 seconds

7. Periods Wenckebach-Samoilova most typical for:
a) Atrioventricular block I degree
b) Atrioventricular block II degree
c) Full atrioventricular blockade
d) Complete blockade of the left bundle branch block

8. Specify one of the ECG signs of blockade of the left bundle branch block:
a) wide, deformed, deep prong S-in leads V1, V2
b) wide, deformed QRS complex of the type RSR in leads V1, V2
c) high and wide R-wave in leads V1, V2
g) the interval P-Q is more than 0.2 s

9. What are the ECG signs of blockade of right bundle branch block:
a) wide, deformed QRS complex, the complex type rSR in leads V1, V2
b) wide, deformed QRS complex, increasing the amplitude of the R wave in leads V5, V6
c) deep and wide prong S-in leads V1, V2
d) narrow QRS, the increase in the amplitude of the R wave in leads V1, V2

10. For complete blockade of the left bundle branch block is NOT typical:
a) deformation and broadening of the R-wave in leads V5, V6 and prongs S in lead V1
b) deformation of the R-wave in leads I and aVL
c) strain broadening of the R-wave in lead V1 and prongs S in lead V6
d) the broadening of the prong S III leads, aVF

11. ECG signs of atrio-ventricular blockade of II degree is:
   a) Increase of PQ interval over 0.20 seconds
   b) arise periodic discontinuation of the separate impulses from the Atria to the ventricles
   C) Deformation and/or changing the polarity of the wave P and
g) isolation of atrial and ventricular rhythms

12. ECG characteristics of atrioventricular block III degree are:
   a) the Number of contractions of the ventricles 70 beats per minute and less
   b) the Number of cuts желудочков 180-220 beats per minute
   C) the Number of contractions of the ventricles 90-130 beats per minute
d) a Complete dissociation of atrial and ventricular rhythms

13. If heart rate 40 per minute, PQ – 0.20, three of P wave have a QRS complex. This ECG pattern is typical for:
   a) atrioventricular block II degree, Mobitz II
   b) atrioventricular block III degree
   c) sinoaurikuliarnaya blockade
d) atrial fibrillation, rediform

14. Loss of QRS without progressive lengthening of PQ interval observed in:
   a) atrioventricular block I degree
   b) atrioventricular block, Mobitz I
   C) atrio-ventricular blockade of II Mobitz
d) complete atrio-ventricular blockade

15. The pacing of the ventricles during complete blockade of the right bundle branch block may be:
   a) the sinus node
   b) atrioventricular node
   C) bundle branch block
d) Purkinje fibers

16. Check ECG sign of complete blockade of the left bundle branch block:
   a) rSR in AVF
   b) rSR V1,V2
   C) QRS is less than 0, 12 seconds
   d) broad serrated tooth R in I, AVL,V5-V6 deep S in II, III, AVF, V1-V2

17. On ECG: rhythm of the Atria right heart rate-80 per minute. The rhythm of the ventricles independent of the Atria with a frequency of 40 per minute. What is the rhythm disturbance?
a) atrial flutter  
b) complete blockade of the left bundle branch block  
c) complete atrio-ventricular blockade  
d) atrial fibrillation  

18. Male 65 years came with complaints of disruption of the heart. On the ECG, the increase of PQ interval of more than 0.24 sec. Which is likely the patient developed?  
a) Full blockade of the left bundle branch block  
b) Atrioventricular block I degree  
c) Incomplete blockage of right branch of atrioventricular bundle  
d) Full blockade of the right bundle branch block  

19. In the clinic a woman came in 70 years. On ECG gradually increasing the elongation of interval P-Q dropout of the ventricular complex, then there is a long pause. These changes most typical for the following pathology:  
a) Atrioventricular block I degree  
b) Atrioventricular block II degree  
c) Atrioventricular block III degree  
d) Atrial fibrillation  

20. A man of 60 years suddenly lost consciousness. The condition was accompanied by convulsions, involuntary urination and defecation. ECG was atrioventricular block II degree, Mobitz I. What changes are detected on the ECG in this pathology?  
a) widening of the QRS complex in leads V5-V6  
b) prolongation of the interval P - Q 0.20 seconds  
c) gradual from complex to the elongation of interval P-Q with subsequent loss of one or more QRS complexes  
d) uniformly lengthened the interval P - Q with the loss of 2 of each of the QRS complex  

Tests on the theme “Spirometry”  

1. Indications for conducting of spirography is:  
a) identifying the causes of respiratory symptoms  
b) monitoring of bronchial obstruction  
c) monitoring of restrictive disorders  
d) all of the above is true  

2. Indications for conducting of spirography is NOT  
a) pronounced attack of bronchial asthma  
b) assessment of the effectiveness of treatment of bronchopulmonary diseases  

119
C) objectification of complaints of the patient with professional pathology of the lungs 
d) assessment of risk of operative intervention

3. Conducting of spirography is contraindicated:
a) patients developed pneumothorax and for 2 weeks after his permission  
b) in the first 2 weeks after myocardial infarction after ophthalmic and abdominal surgery;  
C) severe continuing hemoptysis  
d) all of the above is true

4. The forced vital capacity of lungs is:
a) the maximum breath + maximum exhalation  
b) the maximum amount of air a person can inhale after the deepest possible exhalation.  
c) the maximum amount of air a person can exhale after maximum deep inhalation.  
d) vital capacity + residual capacity of lungs

5. The amount of air removed from the lungs in the first second of exhalation is:  
a) FEV1  
b) MEF 25  
c) PEF

6. Vital capacity of lungs is:  
a) the volume of air that can exhale in the complete exhalation after a maximal inspiration  
b) the volume of air that can exhale during a forced exhalation after a maximal inspiration  
C) the amount of air to exhale on full expiration after a forced inhalation  
d) the volume of air that can exhale after a relaxed exhalation

7. Respiratory maneuver is considered to be reproducible if:  
a) exhalation lasts at least 6 seconds, performed with maximum effort from beginning to end  
b) exhale is not interrupted by coughing, overlap of the epiglottis  
C) on the curve "flow-volume" missing artifacts  
d) all of the above is true.

8. Due values of spirometric parameters depend on :  
a) growth  
b) floor  
C) age  
d) all of the above is true
9. The inspiratory part of the curve "flow-volume" is characterized by:
   a) has the shape of a nearly right triangle with base in the form of FVC
   +b) has the shape of a symmetrical arc
   C) is upward knee records
   d) is downward knee records

10. Expiratory part of the curve "flow-volume" is characterized by:
    a) has the shape of a nearly right triangle with base in the form of FVC
    b) has the shape of a symmetrical arc
    C) is upward knee records
    d) is downward knee records

11. The pathophysiological mechanism of obstructive ventilation disorders is:
    a) shortening of the respiratory tract
    b) increased resistance of the respiratory tract
    C) a decrease in distensibility of the respiratory tract
    d) none of the above

12. Pathophysiological mechanism of restrictive ventilation disorders is:
    a) shortening of the respiratory tract
    b) increase in airway resistance
    c) reduced distensibility of the respiratory tract
    d) none of the above

13. Pathophysiological mechanism of mixed ventilation disorders is:
    a) the extension of the lumen of the respiratory tract with a reduction in lung volumes
    b) the extension of the lumen of the Airways with increased lung volumes
    C) narrowing of the lumen of the Airways with increased lung volumes
    d) narrowing of the lumen of the respiratory tract with a reduction in lung volumes.

14. Obstructive ventilation disorders are characterized by:
    a) a decrease in the ratio of FEV1/FVC with normal FVC
    b) a decrease in the ratio FEV1/FVC reduced FVC
    C) a normal ratio of FEV1/FVC with increased FVC
    d) a normal ratio of FEV1/FVC decreased FVC

15. Restrictive ventilation disorders are characterized by:
    a) in the initial stage may FEV1/FVC
    b) all of the above is true
    c) peak volumetric rate usually remains normal
    d) with the progression of the disease, reduced VC, the curve flow-volume becomes high and narrow
16. Mixed ventilation disorders recorded in the case:
a) reduction in FVC, FEV1 in the background of the increased ratio of FEV1/FVC.
b) increase in FVC, FEV1 in the background of the decrease of the ratio FEV1/FVC.
C) normal values FVC, FEV1 and decreased ratio of FEV1/FVC.
d) simultaneous decrease in FVC, FEV1 and FEV1/FVC.

17. There are the following types of upper airway obstruction:
a) variable extracurricula obstruction
b) the thoracic variable obstruction
c) all of the above options are correct
d) fixed obstruction

18. To assess the severity of obstructive disorders using:
a) FEV1
b) FVC

19. The reversibility of bronchial obstruction is determined by:
a) decrease in FEV1, FVC or both after inhalation bronchodilator
b) increase MEF25-75, after inhalation bronchodilator
c) decrease MEF 25-75 after inhalation bronchodilator
d) increase FEV1, FVC or both after inhalation bronchodilator

20. Indications for bronchodilation test:
a) determining the potential effect of medical therapy;
b) establish the reversibility of bronchial obstruction, including patients with normal baseline spirometry;
C) monitoring dynamics of pulmonary function in patients with chronic respiratory disease with long-term (multi-year) observation.
d) all of the above is true

21. What disease increases FVC, in the absence of changes in other indicators of spirography?
a) emphysema
b) pneumothorax
c) acromegaly
d) funnel-shaped chest deformity

22. Is NOT an indication for the bronchodilation test:
+a) the establishment of the degree of bronchial obstruction
b) establish the reversibility of bronchial obstruction,
C) determining the effect of medical therapy
d) monitoring dynamics of pulmonary function
23. To conduct a bronchodilation test, do NOT use:
   a) salbutamol
   b) fenoterol
   c) tiotropiya bromide
   d) beklazon

24. Bronchodilation test is considered positive if:
   a) after inhalation of bronchodilator FEV1 is over 10%, and the absolute increase was more than 100 ml
   b) after inhalation of bronchodilator FEV1 is more than 12% and an absolute increase of more than 200 ml
   C) after inhalation of bronchodilator FEV1 is more than 20% and an absolute increase of more than 300 ml
   d) after inhalation of bronchodilator FEV1 is more than 22% and an absolute increase of more than 220 ml

25. Click on the pattern curve corresponding to the most significant decrease in FVC
   a) A
   b) B
   c) D
   d) C

26. Click on the picture curves corresponding obstructive disturbance of ventilation
   a) A, B, C
   b) C,D, I
   c) A, B, D
   d) B, C, D

27. Click on the picture curves corresponding restrictive disturbance of ventilation
   a) A, B
   b) B,C
   C) D, I
   d) C, D
28. Select the drawing curve that most closely corresponds to pneumectomy
   a) A
   b) B
   c) D
   d) I

29. Some of the indicators of spirogramma will remain in the normal range with
    the development of restrictive disorders:
   a) Vital capacity
   b) PEF
   C) FVC
   d) FEV1/FVC

30. The most early and sensitive indicator of the development of obstructive
    syndrome is:
   a) PEF 25-75
   b) MEF25
   c) FEV1
   d) FEV1/FV

Tests on the theme “Blood tests. Clotting disorders”

1. In anemia there is a reduction in the number of:
   a) platelets
   b) leukocytes
   C) granulocytes
   d) hemoglobin

2. For a group of diseases characterized by the appearance of a hiatus leukaemicus
   a) acute leukemias
   b) chronic leukemias
   C) leukemoid reaction
   d) inflammatory diseases

3. For any defect of hemostasis characterized by the appearance gematomny type
   of bleeding:
   a) for violations of the platelet level
   b) for vascular hemostasis
   c) for disorders of coagulation hemostasis
   d) all of the above options are correct

4. For what disease is characterized by the appearance of macrocytosis
   a) iron deficiency anemia
b) B12 deficiency anemia  
C) anemia of chronic diseases  
d) aplastic anemia  

5. Transferrin is:  
a) β-globulin responsible for iron deposition  
b) form dwuhvalentnoe iron  
c) form of ferric  
d) β-globulin responsible for iron transport  

6. Eosinophilia is found with the following pathology:  
a) worm infestation  
b) allergic reactions  
C) bronchial asthma  
d) all of the above  

7. In pernicious anemia, characterized by:  
a) the development of iron deficiency, mikrocytos  
b) deficiency of folic acid, mikrocytos  
c) deficiency of cyanocobalamin, macrocitos  
d) combined deficiency of iron and folic acid  

8. On the average concentration of hemoglobin in the erythrocyte is demonstrated by the following figure:  
a) sit  
b) MCV  
c) MCHC  
d) RDW  

9. Jolie's bodies and rings Kebot detected with the following pathology:  
a) iron deficiency anemia  
b) anemia of chronic diseases  
C) aplastic anemia  
d) B12 deficiency anaemia  

10. To the coagulation system include:  
a) antithrombin III  
b) heparin  
c) factor Stuart-Prauera  
d) the proteins S and C  

11. The clinic enters the patient 42 years with complaints of weakness, fatigue, palpitations. According to laboratory tests: Erythrocyte 2,11x1012/l, hemoglobin-92 g/l, mean corpuscular volume 72 FL. What kind of pathology should think?
a) normochromic anemia  
b) normochromic microcytic anemia  
C) hypochromic anemia  
d) hypochromic microcytic anemia

12. On admission, the patient turns 20 years old, with complaints of petechiae and ekhimozy of the type "leopard skins". General analysis of blood has the following abnormalities: hemoglobin 118 g/l, erythrocytes-4,15x10\(^{12}\)/l, platelets 20x10\(^9\)/l, pronounced polymorphism of the platelet. What kind of pathology should think?
   a) hemophilia  
b) thrombocytopenic purpura  
C) disease Weber-Rendu-Osler  
d) von Willebrand's disease

13. While on duty, the doctor gets tests the patient received about 1 hour ago. According to the General analysis of blood: leukocytes 17x10\(^9\)/l, metamyelocytes – 0, “Polys”– 11, “Segs”– 45, erythrocyte sedimentation rate 44 mm/h. When the morphology of the observed toxic granularity of neutrophils. What kind of pathology should think?
   a) leukocytosis with a shift to the right leucoformula  
b) leukocytosis with a left shift leucoformula  
c) acute or chronic leukemia

14. At the local doctor observed the patient 18 years of age. Its the main complaints of prolonged bleeding after tooth extraction, episodic pain in large joints, causing minor injuries. According coagulation: PT 100%, reduced factor VIII blood clotting. What kind of pathology should think?
   a) thrombocytopenic purpura  
b) anemia  
C) thrombophilia  
d) hemophilia

15. The major causes of acquired coagulation disorders are
   a) Vitamin K deficiency  
b) Liver disease  
c) Disseminated intravascular coagulation  
e) Development of circulating anticoagulants  
f) All above

16. What are the reasons of prolonged PT or PTT with normal platelet count ?
   a) liver disease  
b) vitamin K deficiency  
c) during anticoagulation with warfarin  
d) trombocytopenia
17. What are the reasons of Prolonged PT with normal platelets and PTT?
   a) liver disease
   b) vitamin K deficiency
   c) during anticoagulation with warfarin
   d) factor VII deficiency

18. What are the reasons of Prolonged PTT with normal platelets and PT?
   a) hemophilia A or B
   b) vitamin K deficiency
   c) during anticoagulation with warfarin
   d) factor VII deficiency

19. Symptoms of coagulation disorders with difficulty clotting include:
   a) Blood in the urine or stool
   b) Bruising easily and excessively
   c) Extreme fatigue
   d) cough
   e) headache

20. What are the reasons of basophilia?
   a) allergic reactions (e.g., hives, food allergy)
   b) Inflammation (rheumatoid arthritis, ulcerative colitis)
   c) Some leukemias (e.g., chronic myeloid leukemia)
   d) All above

**Tests on the theme “Urinalysis”**

1. Creatinine is
   a) the end product of the conversion of purines
   b) end product of metabolism of creatine phosphate
   C) the end product of protein metabolism
   d) the end product of the metabolism of oligosaccharides

2. Gipostenuriya is
   a) an impaired ability of the kidneys to concentrate the urine in the form of lower relative density during the day
   b) an impaired ability of the kidneys to concentrate the urine in the form of higher relative density during the day
   C) the impaired ability of the kidneys to the urine dilution in the form of slight fluctuations in the specific gravity of urine during the day
   d) the impaired ability of the kidneys to the urine dilution in the form of considerable fluctuations of the specific weight of urine during the day
3. The level of uric acid in serum may be increased with:
   a) chlamydia
   b) pneumonia
   C) fasting
   d) gout

4. That is correct when you run the sample Nechiporenko
   a) in 1 ml of less $4 \times 10^3$ leukocytes, erythrocytes less $2 \times 10^3$
   b) in 1 liter of less $4 \times 10^3$ leukocytes, erythrocytes less $2 \times 10^3$
   c) in 1 ml of $2 \times 10^3$ less leukocytes, erythrocytes less $1 \times 10^3$
   d) in 1 liter of less $2 \times 10^3$ leukocytes, erythrocytes less $1 \times 10^3$

5. Specify valid values for glucose in the urine
   a) up to 20%
   b) up to 10%
   c) up to 2%
   d) (0-0.02%)

6. Protein Bence-Jones appears in:
   a) multiple myeloma
   b) gout
   C) diabetes
   d) acute myocardial infarction

7. Which of the formulas to calculate glomerular filtration requires consideration of body mass
   a) MDRD
   b) Cockcroft-Gault
   C) CKD-EPI
   d) none of the formulas does not require the body mass index

8. Which sample urine test is used for identifying the source of hematuria?
   a) sample Nechiporenko
   b) sample Kakhovskogo-Addisa
   C) test of General
   d) three glasses sample

9. Which is most characteristic of nephrotic syndrome
   a) a relatively high density of urine
   b) oliguria
   C) the high proteinuria
   d) all above
10. What changes will be in the General analysis of urine during exacerbation of chronic pyelonephritis?
   a) pyuria, bacteriuria
   b) erythrocyturia
   c) oxaluria
   d) glycosuria

11. What changes of urine occur most frequently in glomerulonephritis?
   a) leukocyturia
   b) glycosuria
   c) bacteriuria
   d) hematuria (macro- or microscopic hematuria), proteinuria

12. Zymnickii test is used to determine
   a) degree of pyuria
   b) the degree of erythrocyturia
   c) daily fluctuations in volume and density of urine
   d) daily proteinuria

13. Terminal stage of chronic renal failure corresponds to:
   a) stage 1 CKD
   b) stage 3 CKD
   C) stage 4 CKD
   d) stage 5 CKD (uremic)

14. In the presence of leukocyturia on the results of the urinalysis, which of the extended sample should be appointed:
   a) a sample Nechiporenko,
   b) a sample of Addisa-Chukovskogo,
   C) urinalysis
   d) right a) and b)

15. To determine the stage of chronic kidney disease requires the following laboratory parameters:
   a) weight
   b) glomerular filtration rate, daily microalbuminuria.
   C) age
   d) residual nitrogen

16. According to the result of urinalysis detected: specific gravity 1018, color light yellow, transparent, protein – 0 sugar – 0, the reaction is slightly alkaline, leukocytes 2-3 in p/z, erythrocytes 6-8 p/z, salt "++++" the oxalates. Additionally: it is known that the patient observes cramping pain in the lumbar region. What most probable pathology should be thinking?
17. On admission, the patient turns 19 years old. She was concerned about the increase in body temperature to 37.2°C, according to the General analysis of blood - leukocytosis, accelerated erythrocyte sedimentation rate 33 mm/h, according to the urinalysis – leukocyturia, bacteriuria. What kind of pathology should think?
   a) glomerulonephritis
   b) urolithiasis
   c) pyelonephritis
   d) diabetes mellitus

18. Patient 78 years three glasses test. The greatest number of erythrocytes in the first portion of the urine. It is most likely that the problem is localized at the level of:
   a) kidney
   b) of the urethra
   C) ureters
   d) right a) and C)

19. The patient of 68 years to the doctor complaining of dry mouth, thirst. According to the urine – glycosuria. What kind of pathology should think?
   a) pyelonephritis
   b) glomerulonephritis
   C) Addison's disease
   d) diabetes mellitus

20. The patient has increased creatinin, uremia, hyperammonemia, uremia; proteinuria. What kind of pathology should think?
   a) the initial stages of chronic kidney disease
   b) urolithiasis
   c) terminal renal failure.
   d) glomerulonephritis
VI. Examples of interpretation of blood tests

CBC

WBC  19.5  [4.0-10.0] k/ul
RBC  3.49  [3.60-5.50] m/ul
Hgb  10.4  [12.0-16.0] gm/dl
Hct  31.2  [34.0-51.0] %
MCV  82   [85-95] fl
MCH  28.3 [28.0-32.0] pg
MCHC 33.3 [32.0-36.0] gm/dl
RDW  16.6 [11.0-15.0] %
Plt Count  98 [150-400] k/ul

CBC demonstrates
Leukocytosis
Microcytic anemia with elevated red cell distribution width
Thrombocytopenia

The following CMP is from a patient who presented with systolic congestive heart failure exacerbation

Complete Metabolic Panel
- Glucose 112 H [70 – 100] mg/dl
- Blood Urea Nitrogen 39 H [7 - 22] mg/dl
- Creatinine 1.6 H [0.7 - 1.5] mg/dl
- Calcium 8.9 [8.5 - 10.5] mg/dl
- Sodium 132 L [136 - 146] mmol/L
- Potassium 4.0 [3.5 - 5.3] mmol/L
- Chloride 93 L [98 - 108] mmol/L
- Carbon Dioxide 23 [20 - 32] mmol/L
- Albumin 3.1 L [3.6 - 5.0] gm/dl
- Protein, Total 5.8 L [6.2 - 8.0] gm/dl
- Alkaline Phosphatase 200 [25 - 215] IU/L
- AST 35 [5 - 40] IU/L
- Bilirubin, Total 1.9 H [0.2 - 1.4] mg/dl

- BUN and creatinine are elevated with a BUN:Creat ratio greater than 20:1 consistent with pre-renal azotemia, the result of inadequate renal perfusion and resulting reduced urea clearance.
Hepatic congestion leads to hypoxia and altered function of the liver cells. Bilirubin, especially the indirect fraction, and enzymes, like alkaline phosphatase, may be elevated. Total protein may decline at the expense of the decreased albumin produced in the liver.

The electrolyte changes, especially hyponatremia, reflect a dilutional effect with water retention and decreased glomerular filtration rate (poor perfusion).

Hyperglycemia is present but it is not known whether this was a fasting or random sample.
VII. Tasks to consolidate the material

**Sputum test:**
Blood-stained Mucoid
Epithelial cells - 2-3,
Leukocytes 3-4-5
Red blood cells - much
*Mycobacterium tuberculosis* No. 3 is not detected.

1. Rate of the received data.
2. What kind of disease can you think of?

**Sputum test:**
Muco-purulent, yellow, viscous,
Epithelial cells - 4-5,
Leukocytes - a large number
Red blood cells 0-1-2,
macrophages 5-6 in p/Zr.
*Mycobacterium tuberculosis* was not detected.

1. Rate of the received data.
2. What kind of disease can you think of?

**Sputum test:**
slimy, grey, viscous,
Epithelial cells 5-6,
Leukocytes - 7-10 in p/Zr.,
eosinophils - 3%
Spiral Churchman
The Charcot-Leyden Crystals
*Mycobacterium tuberculosis* was not detected.

1. Rate of the received data.
2. What kind of disease can you think of?

**Sputum test:**
Muco-purulent, liquid,
Epithelial cells 2-3,
Leukocytes 30-40
Red blood cells – 4-5
atypical cells discovered
*Mycobacterium tuberculosis* was not detected.
1. Rate of the received data.
2. What kind of disease can you think of?

**Complete blood count**

Red blood cells 3.5 x 10¹²/l
Hemoglobin 78 g/l
Color. the figure of 0.6 (MCV ≤ 80)
Reticulocytes 10%
Leukocytes (WBC) 6.6 x 10⁹/l
Leucoformula:
eosinophils 2%
“Polys” 4%
“Segs” 64%
lymphocytes 22%
monocytes 8%
Revealed microcytosis, anisocytosis and poikilocytosis.
Erythrocyte sedimentation rate 30 mm/h
Serum iron 7.0 mmol/l

1. Rate of the received data.
2. What kind of disease can you think of?

**Complete blood count**

Red blood cells 2.5 x 10¹²/l
Hemoglobin 68 g/l
Color. the figure of 0.6 (MCV ≤ 80)
Reticulocytes 12%
Leukocytes 6.8 x 10⁹/l
Leucoformula:
eosinophils 1%
“Polys” 5%
“Segs” 63%
lymphocytes 21%
monocytes 8%
Revealed microcytosis, anisocytosis and poikilocytosis.
Erythrocyte sedimentation rate 30 mm/h
Serum iron 6.8 mmol/l

1. Rate of the received data.
2. What kind of disease can you think of?
**Complete blood count**

Erythrocytes - $4.0 \times 10^{12}$/l
Hemoglobin 132 g/l
Color figure 0,9
The platelet count $160 \times 10^9$/l
WBC - $17 \times 10^9$/l

Leucoformula:

- Basophils 1%
- Eosinophils 1%
- "Polys" 12%
- "Segs" 52%
- Lymphocytes 25%
- Monocytes 5%
- Erythrocyte sedimentation rate 45 mm/h

1. Rate of the received data.
2. On what condition should you think of?

**General analysis of blood**

Erythrocytes - $4.0 \times 10^{12}$/l
Hemoglobin 132 g/l
Color figure 0,9
The platelet count $160 \times 10^9$/l
The WBC - $15 \times 10^9$/l

Leucoformula:

- Basophils 1%
- Eosinophils 1%
- "Polys" 10%
- "Segs" 54%
- Lymphocytes 25%
- Monocytes 5%
- Erythrocyte sedimentation rate 35 mm/h
1. Rate of the received data.
2. On what condition should you think of?

**Complete blood count**

Erythrocytes - 3.2 x 10^12 /l  
Hemoglobin 92 g/l  
Color figure 0.86 (MCV = 85)  
The platelet count 160 x 10^9 /l  
The reticulocytes of 15%  
WBC - 7.2 x 10^9 /l  

Leucoformula:

- Basophils 1%  
- Eosinophils 1%  
- “Polys” 4%  
- “Segs” 59%  
- Lymphocytes 23%  
- Monocytes 7%  

ESR 25 mm/h  

Bilirubin blood plasma:

total 58 µmol/l  
direct 9.5 µmol/l  
indirect 48.5 mmol/l  

1. Rate of the received data.  
2. On what condition should you think of?

**Complete blood count**

Erythrocytes - 2.2 x 10^12 /l  
Hemoglobin 72 g/l  
Color figure 0.82  
The platelet count 160 x 10^9 /l  
The reticulocytes of 15%
The WBC - $7.2 \times 10^9$/l

Leucoformula:

- Basophils 1%
- Eosinophils 1%
- Lymphocytes 23%
- “Polys” 4%
- “Segs” 59%
- Monocytes 7%

ESR 25 mm/h

Bilirubin blood plasma

total 78 µmol/l
direct 8.5µmol/l
indirect 69.5 mmol/l

1. Rate of the received data.
2. On what condition should you think of?

**Complete blood count**

The red blood cells of $1.24 \times 10^{12}$/l
Hemoglobin 50 g/l
Color the rate is 1.2% (MCV $\geq 100$)
Reticulocytes 0.2%
The platelet count $133 \times 10^9$/l
WBC – $3.8 \times 10^9$/l

Leukogram:

- “Polys” 1%
- “Segs” 47%
- Lymphocytes 44%
- Monocytes 8%

Erythrocyte sedimentation rate 63 mm/h

The anisocytosis, macrocytosis. Megalocytes. Jolly's bodies and rings Kebot? Red blood cells with a large number of segments of the nucleus (6-7 or more).

1. Rate of the received data.
2. What kind of illness should think?

**Complete blood count**

The red blood cells of $2,05 \times 10^{12}$ /l  
Hemoglobin 57 g/l  
Color: the rate is 1.2%  
Reticulocytes 0.2%  
The platelet count $133 \times 10^9$/l

WBC – $3.5 \times 10^9$/l  
Leukogram:  

“Polys”1%  
“Segs” 47%  
Lymphocytes 44%  
Monocytes 8%  

Erythrocyte sedimentation rate 53 mm/h

The anisocytosis, macrocytosis. Megalocytes. Jolly's bodies and rings Kebot, Red blood cells with a large number of segments of the nucleus (6-7 or more).

1. Rate of the received data.  
2. What kind of disease should think?

**Blood biochemistry:**

total bilirubin is 15.7 µmol/l,  
direct bilirubin – 6.7 mmol/l,  
bilirubin indirect – 9.0 mmol/l,  
AST - 182, ALT – 87

1. Rate of the received data.  
2. What kind of disease should think?

**Blood biochemistry:**

total bilirubin – 55.7 µmol/l,  
direct bilirubin is 46.7 mmol/l,  
bilirubin indirect – 9.0 mmol/l,
AST - 282, ALT - 87,
1. Rate of the received data.
2. What kind of disease should think?

**Blood biochemistry:**

total bilirubin – of 65.7 µmol/l,
bilirubin direct – 36.7 mmol/l,
the unconjugated bilirubin – 29.0 mmol/l,
AST - 182, ALT - 687,
1. Rate of the received data.
2. What kind of disease should think?

**Blood biochemistry:**

total bilirubin is 85.7 µmol/l,
bilirubin direct – 62.7 mmol/l,
bilirubin indirect – 23.0 mmol/l,
GGTP – 456 nmol/l
Alkaline phosphatase - 57 µmol/l
AST - 32, ALT - 38,
1. Rate of the received data.
2. What kind of disease should think?

**Blood biochemistry:**

total bilirubin – 55.7 µmol/l,
direct bilirubin is 46.7 mmol/l,
bilirubin indirect – 9.0 mmol/l,
GGTP - 321 nmol/l
Alkaline phosphatase – 32 mmol/l
AST - 38, ALT - 42,
1. Rate of the received data.
2. What kind of disease should think?
**Blood biochemistry:**
total bilirubin – of 65.7 µmol/l,
bilirubin direct – to 56.7 mmol/l,
bilirubin indirect – 9.0 mmol/l,
GGTP - 276 nmol/l
Alkaline phosphatase - 37 µmol/l

1. Rate of the received data.
2. What kind of disease should think?

**Blood biochemistry:**
creatine 139 umol/l,
urea of 11.6 mol/l,
nitrogen — 37.0 mmol/l,
GFR was 67 ml/min

**Blood biochemistry:**
creatine 139 umol/l,
urea of 11.6 mol/l,
nitrogen — 37.0 mmol/l,
GFR – 23 ml/min.

**Blood biochemistry:**
creatine - 523 µmol/l,
urea is 13.6 mol/l,
nitrogen — 37.0 mmol/l,
GFR – 13 ml/min.

**Urinalysis**

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
</table>

140
<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>100 ml</td>
</tr>
<tr>
<td>Color</td>
<td>yellow</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1015</td>
</tr>
<tr>
<td>Transparency</td>
<td>clear</td>
</tr>
<tr>
<td>Protein</td>
<td>0</td>
</tr>
<tr>
<td>Sugar</td>
<td>0</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>1-2 п/з</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>1-2 п/з</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>0</td>
</tr>
<tr>
<td>Cylinders</td>
<td></td>
</tr>
<tr>
<td>Salt</td>
<td>-</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

### Urinalysis

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>150 мл</td>
</tr>
<tr>
<td>Color</td>
<td>yellow</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1005</td>
</tr>
<tr>
<td>Transparency</td>
<td>cloudy</td>
</tr>
<tr>
<td>Protein</td>
<td>3,5</td>
</tr>
<tr>
<td>Sugar</td>
<td>0</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>2-4 п/з</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>2-4 п/з</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>2-4-6 п/з</td>
</tr>
<tr>
<td>Cylinders</td>
<td>hyaline, waxy</td>
</tr>
<tr>
<td>Salt</td>
<td>-</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

### Urinalysis

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>250</td>
</tr>
<tr>
<td>Color</td>
<td></td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1009</td>
</tr>
<tr>
<td>Transparency</td>
<td>cloudy</td>
</tr>
<tr>
<td>Protein</td>
<td>1,37</td>
</tr>
<tr>
<td>Sugar</td>
<td>-</td>
</tr>
<tr>
<td>------</td>
<td>---</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>2-4</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>4-6</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>8-10 (\pi/3)</td>
</tr>
<tr>
<td>Cylinders</td>
<td></td>
</tr>
<tr>
<td>Salt</td>
<td>-</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

**Urinalysis**

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>100</td>
</tr>
<tr>
<td>Color</td>
<td>yellow</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1019</td>
</tr>
<tr>
<td>Transparency</td>
<td>cloudy</td>
</tr>
<tr>
<td>Protein</td>
<td>0.066</td>
</tr>
<tr>
<td>Sugar</td>
<td>-</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>10-12 (\pi/3)</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>much</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>10-12 (\pi/3)</td>
</tr>
<tr>
<td>Cylinders</td>
<td>-</td>
</tr>
<tr>
<td>Salt</td>
<td>oxalates</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

**Urinalysis**

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>300</td>
</tr>
<tr>
<td>Color</td>
<td>yellow</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1009</td>
</tr>
<tr>
<td>Transparency</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>0.176</td>
</tr>
<tr>
<td>Sugar</td>
<td>-</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td></td>
</tr>
<tr>
<td>Leukocytes</td>
<td>4-5 (\pi/3)</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>8-10 (\pi/3) (dysmorphic)</td>
</tr>
<tr>
<td>Cylinders</td>
<td>waxy and granular</td>
</tr>
<tr>
<td>Salt</td>
<td>-</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?
### Urinalysis

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>180</td>
</tr>
<tr>
<td>Color</td>
<td>red</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1030</td>
</tr>
<tr>
<td>Transparency</td>
<td>cloudy</td>
</tr>
<tr>
<td>Protein</td>
<td>0.266</td>
</tr>
<tr>
<td>Sugar</td>
<td>-</td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>1-2</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>4-3</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>50 (dysmorphic)</td>
</tr>
<tr>
<td>Cylinders</td>
<td>waxy and granular</td>
</tr>
<tr>
<td>Salt</td>
<td>-</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

### Urinalysis

<table>
<thead>
<tr>
<th>Name</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>quantity</td>
<td>200</td>
</tr>
<tr>
<td>Color</td>
<td>yellow</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1012</td>
</tr>
<tr>
<td>Transparency</td>
<td>cloudy</td>
</tr>
<tr>
<td>Protein</td>
<td>0.066</td>
</tr>
<tr>
<td>Sugar</td>
<td></td>
</tr>
<tr>
<td>Ep. Cells</td>
<td>5-6 пр/з</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>10-12 пр/з</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>1-2</td>
</tr>
<tr>
<td>Cylinders</td>
<td></td>
</tr>
<tr>
<td>Salt</td>
<td>++</td>
</tr>
<tr>
<td>Bacteria</td>
<td>+++</td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

### Urine test by Zimnicky

<table>
<thead>
<tr>
<th>time</th>
<th>quantity</th>
<th>Specific gravity</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-9</td>
<td>320</td>
<td>1.005</td>
</tr>
<tr>
<td>Time</td>
<td>Quantity</td>
<td>Specific Gravity</td>
</tr>
<tr>
<td>-------</td>
<td>----------</td>
<td>------------------</td>
</tr>
<tr>
<td>9-12</td>
<td>270</td>
<td>1,005</td>
</tr>
<tr>
<td>12-15</td>
<td>260</td>
<td>1,004</td>
</tr>
<tr>
<td>15-18</td>
<td>250</td>
<td>1,004</td>
</tr>
<tr>
<td>Day output</td>
<td>1100 ml</td>
<td></td>
</tr>
<tr>
<td>18-21</td>
<td>230</td>
<td>1,004</td>
</tr>
<tr>
<td>21-24</td>
<td>170</td>
<td>1,005</td>
</tr>
<tr>
<td>24-03</td>
<td>130</td>
<td>1,004</td>
</tr>
<tr>
<td>03-06</td>
<td>170</td>
<td>1,005</td>
</tr>
<tr>
<td>Night output</td>
<td>700 ml</td>
<td></td>
</tr>
<tr>
<td>Dieresis</td>
<td>1800 mk</td>
<td></td>
</tr>
<tr>
<td>Water intake</td>
<td>1200 ml</td>
<td></td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

### Urine test by Zimnicky

<table>
<thead>
<tr>
<th>Time</th>
<th>Quantity</th>
<th>Specific Gravity</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-9</td>
<td>400</td>
<td>1006</td>
</tr>
<tr>
<td>9-12</td>
<td>200</td>
<td>1005</td>
</tr>
<tr>
<td>12-15</td>
<td>200</td>
<td>1006</td>
</tr>
<tr>
<td>15-18</td>
<td>200</td>
<td>1004</td>
</tr>
<tr>
<td>Day output</td>
<td>1000 ml</td>
<td></td>
</tr>
<tr>
<td>18-21</td>
<td>200</td>
<td>1004</td>
</tr>
<tr>
<td>21-24</td>
<td>200</td>
<td>1005</td>
</tr>
<tr>
<td>24-03</td>
<td>300</td>
<td>1003</td>
</tr>
<tr>
<td>03-06</td>
<td>300</td>
<td>1005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>--------</td>
<td>----------</td>
</tr>
<tr>
<td>Night output</td>
<td>1000 ml</td>
<td></td>
</tr>
<tr>
<td>Dieresis</td>
<td>2200 ml</td>
<td></td>
</tr>
<tr>
<td>Water intake</td>
<td>1000 ml</td>
<td></td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?

**Urine test by Zimnicky**

<table>
<thead>
<tr>
<th>time</th>
<th>quantity</th>
<th>Specific gravity</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-9</td>
<td>350</td>
<td>1006</td>
</tr>
<tr>
<td>9-12</td>
<td>250</td>
<td>1004</td>
</tr>
<tr>
<td>12-15</td>
<td>250</td>
<td>1005</td>
</tr>
<tr>
<td>15-18</td>
<td>270</td>
<td>1005</td>
</tr>
<tr>
<td>Day output</td>
<td>1130 ml</td>
<td></td>
</tr>
<tr>
<td>18-21</td>
<td>250</td>
<td>1004</td>
</tr>
<tr>
<td>21-24</td>
<td>150</td>
<td>1005</td>
</tr>
<tr>
<td>24-03</td>
<td>170</td>
<td>1006</td>
</tr>
<tr>
<td>03-06</td>
<td>150</td>
<td>1008</td>
</tr>
<tr>
<td>Night output</td>
<td>670ml</td>
<td></td>
</tr>
<tr>
<td>Dieresis</td>
<td>1800 ml</td>
<td></td>
</tr>
<tr>
<td>Water intake</td>
<td>1200 ml</td>
<td></td>
</tr>
</tbody>
</table>

1. Rate of the received data.
2. What kind of disease should think?