

Ministry of Health of Ukraine
Kharkiv National Medical University

**AUSCULTATION OF THE HEART. NORMAL HEART SOUNDS,
REDUPLICATION OF THE SOUNDS, ADDITIONAL SOUNDS
(TRIPLE RHYTHM, GALLOP RHYTHM), ORGANIC AND
FUNCTIONAL HEART MURMURS**

Methodical instructions for students

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Authors: T.V. Ashcheulova
O.M. Kovalyova
O.V. Honchar

AUSCULTATION OF THE HEART

To understand the underlying mechanisms contributing to the cardiac tones formation, it is necessary to remember the sequence of myocardial and valvular action during the cardiac cycle.

During ventricular systole:

1. Asynchronous contraction, when separate areas of myocardial wall start to contract and intraventricular pressure rises.
2. Isometric contraction, when the main part of the ventricular myocardium contracts, atrioventricular valves close, and intraventricular pressure significantly increases.
3. The ejection phase, when the intraventricular pressure reaches the pressure in the main vessels, and the semilunar valves open.

During diastole (ventricular relaxation):

1. Closure of semilunar valves.
2. Isometric relaxation – initial relaxation of ventricular myocardium, with atrioventricular and semilunar valves closed, until the pressure in the ventricles becomes lower than in the atria.
3. Phases of fast and slow ventricular filling - atrioventricular valves open and blood flows from the atria to the ventricles.
4. Atrial systole, after which cardiac cycle repeats again.

The noise produced by a working heart is called heart sounds. In auscultation two sounds can be well heard in healthy subjects: the first sound (S_1), which is produced during systole, and the second sound (S_2), which occurs during diastole.

S_1 consists of four pair components: **atrial component**: 1 – tension and contraction of the right atrium, 2 – tension and contraction of the left atrium; **valvular component**: 3 – closure and vibration of mitral valve cusps, 4 – closure and vibration of tricuspid valve cusps; **muscular component**: 5 – isometric tension and contraction of the right ventricle, 6 – isometric tension and contraction of the left ventricle; **vascular component**: 7 - vibration of the initial portion of the aorta, 8 – vibration of the initial portion of the pulmonary trunk.

S_2 consists of two pair components: **valvular component**: 1 – closure and vibration of the aortic valve cusps, 2 – closure and vibration of the pulmonary valve cusps; **vascular component**: 3 – vibration of the aortic walls, 4 – vibration of pulmonary trunk walls.

A weak, low-pitched, dull *third sound* (S_3) is sometimes heard and is thought to be caused by vibration of the walls of the ventricles when they are

suddenly distended by blood from atria (passive rapid filling), occurs 0.12-0.15 s after the onset of S₂. The third sound is heard most clearly at the apex of the heart with the bell of a stethoscope; it may be normal in children, adolescents, or very thin adults, or in patients with high cardiac output.

The *fourth heart sound* (S₄) is a low-pitched, presystolic sound produced in the ventricle during ventricular filling; it is associated with an effective atrial contraction and is best heard with the bell piece of the stethoscope.

Since the sites of the valves projection on the chest are very close to one another, it is difficult to assess which valve is affected if listen them in the points of their actual projection. Therefore the heart sounds are auscultated in the certain listening points where sounds of each valve can be better heard.

Auscultation should be performed in the order of decreasing frequency of valves affection: 1 – mitral valve, 2 – aortic valve, pulmonary valve, 4 - tricuspid valve. The fifth listening point to the left of the sternum at the 3rd and 4th costosternal articulation– so-called Botkin-Erb’s point, was proposed to assess aortic valve sound (see Tab.1).

Tab. 1. Projection of the heart valves on the chest wall and standard listening points of the heart

Valve	1 Mitral	2 Aortic	3 Pulmonary	4 Tricuspid	5 Botkin- Erb’s point
Site of projection	To the left of the sternum at the level of the 3 rd costosternal junction	Middle of the sternum at the level of the 3 rd costosternal junction	In the 2 nd intercostal space 1-1.5 cm to the left of the sternum	On the sternum midway between 3 rd left and 5 th right costosternal articulation	Additional information on aortic valve
Listening point	Heart apex	2 nd intercostal space to the right of the sternum	2 nd intercostal space to the left of the sternum	Base of the xiphoid process	Site of the 4 th left sterno-costal junction

The *first heart sound*, a dull, prolonged ‘lub’ marks the onset of the ventricular systole. The *second heart sound*, a short, sharp ‘dup’ occurs at the beginning of ventricular diastole. Diastole is subdivided in protodiastole, mesodiastole, and presystole. Both heart sounds can be heard over precordium,

but their intensity changes depend on nearness of valves that take part in formation of S_1 or S_2 .

In rhythmic heart activity S_1 and S_2 can be differentiated according to the following signs (Tab. 2).

Tab. 2. Differential signs of S_1 and S_2

Main sign	First sound	Second sound
Listening point	Heart apex	Heart base
Relation to cardiac pause	Follows the long pause	Follows the short pause
Duration	0.09-0.12 s	0.05-0.07 s
Relation to apex beat	Synchronous	Follows the apex beat
Relation to carotid pulse	Synchronous	Asynchronous

Factors determining the loudness of S_1 : the state of the atrioventricular valves structures, their position during systole; contractile ventricular function, state of ventricular filling; ventricular contraction speed.

Factors determining the loudness of S_2 : the state of the aortic and pulmonary semilunar valves structures; pressure levels in large and small circles of blood circulation; elastic properties of the aorta and pulmonary trunk.

A weak, low-pitched, dull *third sound* (S_3) is sometimes heard and is thought to be caused by vibration of the walls of the ventricles when they are suddenly distended by blood from atria (passive rapid filling), occurs 0.12-0.15 s after the onset of S_2 . The third sound is heard most clearly at the apex of the heart with the bell of a stethoscope; it may be normal in children, adolescents, or very thin adults, or in patients with high cardiac output.

The *fourth heart sound* (S_4) is a low-pitched, presystolic sound produced in the ventricle during ventricular filling; it is associated with an effective atrial contraction and is best heard with the bell piece of the stethoscope.

Cardiac rhythm. In healthy subjects S_1 and S_2 , S_2 and S_1 follow one another at regular intervals: the heart activity is said to be rhythmic or regular. When the cardiac activity is arrhythmic, the heart sounds follow at irregular intervals.

Heart rate (HR) in normal conditions is 60-80 beats per minute. Acceleration of the heart rate to more than 90 beats per minute is called *tachycardia*. A heart rate less than 60 beat per minute is called *bradycardia*.

In heart sounds analysis their loudness and timbre should be assessed.

Loudness of the heart sounds depends on the point of auscultation.

In the first and fourth listening points first heart sound is louder than second one $S_1 > S_2$, in the second and third – second heart sound is louder than the first $S_1 < S_2$, the second sound over aorta and pulmonary artery is of the same loudness $A_2 = P_2$.

The loudness of the heart sounds can be changed in several physiological and pathological conditions. Loudness of one or both heart sounds may increase or decrease. Changes in both S_1 and S_2 loudness may occur due to the causes listed in tables 3 and 4.

Tab. 3. Both heart sounds decreasing
(in all listening points)

CAUSES			
Extracardiac		Cardiac	
Physiological	Pathological	Primary	Secondary
Excessive muscles development	Obesity Swelling of the chest wall Pulmonary emphysema Effusive pericarditis Effusive left-sided pleurisy	Myocarditis Myocardiosclerosis Myocardial infarction Myocardiopathy	Collapse Shock

Tab. 4. Both heart sounds increasing
(in all listening points)

CAUSES	
Physiological	Pathological
Thin chest wall Nervous excitement Hard physical exertion	Thyrotoxicosis Anemia Wrinkled pulmonary edges Inflammatory consolidation of pulmonary edges Fever

Changes in only one heart sound are very important diagnostically. Different variants of this phenomenon and their diagnostical significance are listed in tables 5-12.

Tab. 5. Increased loudness of the first heart sound at the heart apex

Causes	Mechanism
Mitral stenosis Tachycardia Left ventricular extrasystole Complete atrioventricular block in synchronous contraction of atria and ventricles – ‘pistol-shot’ sound according Strazhesko	Not adequate filling of the left ventricular cavity during diastole, quick and intense contraction of the myocardium

Tab. 6. Decreased loudness of the first heart sound at the heart apex

Causes	Mechanism
Mitral regurgitation Aortic regurgitation Aortic stenosis	Anatomic abnormalities of the valve Absence of closed valve period Overfilling of the left ventricular cavity

Tab. 7. Different loudness of the first heart sound at the heart apex

Causes	Mechanism
Complete heart block Atrial fibrillation Extrasystolic arrhythmia Ventricular flutter	Different ventricular filling in each cardiac cycle

Tab. 8. Accentuated second heart sound over aorta

Causes		Mechanism
Physiological	Pathological	
Emotional exertion Physical exertion	Essential hypertension Symptomatic hypertension Aortic atherosclerosis Syphilitic mesoaortitis	Pressure elevation in the greater circulation, decreased elasticity of the aorta

Tab. 9. Decreased second sound over aorta

Causes	Mechanism
Aortic regurgitation (A)	Anatomic changes of valve (A)

Aortic stenosis (B)	Low pressure in the aorta at the beginning of the diastole (B)
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Tab. 10. Accentuated second heart sound over pulmonary artery

Causes		Mechanism
Physiological	Pathological	
In children Thin chest wall	Mitral valvular diseases Diseases of the broncho-pulmonary system Adhesion of the pleural layers Kyphoskoliotic chest	Pressure elevation in the pulmonary circulation

Tab. 11. Decreased second sound over pulmonary artery

Causes	Mechanism
Pulmonary artery stenosis Pulmonary regurgitation	Anatomical valve changes Low pressure in the pulmonary artery before diastole onset

Tab. 12. Decreased loudness of the first heart sound at the base of the sternum

Causes	Mechanism
Tricuspid regurgitation	Anatomic changes of the valve Absence of closed valves period Overfilling of the right ventricular cavity

Reduplication and splitting of the heart sounds may be revealed in auscultation, which are caused by asynchronous work of right and left chambers of the heart.

Reduplication – two short sounds follow one another are heard instead S_1 or S_2 .

Splitting – two short sounds follow one another at a short interval, and therefore they are not perceived as two separate sounds

Splitting of the two high-pitched components of S_1 by 10-30 ms is a normal phenomenon, recorded by phonocardiography. The third component of S_1 is attributed to mitral valve closure, and the fourth to tricuspid valve closure. Widening of the interval between these two components is heard as S_1 splitting or reduplication at the heart apex or at the base of the xiphoid process.

Physiological splitting of S_1 is heard in the upright position of the patient during very deep expiration, when the blood delivers to the left atrium with a greater force to prevent the closure of the mitral valve. The valvular component of the left ventricle is therefore splits and is perceived as a separate sound.

Pathological splitting of S_1 is due to:

- Sclerosis of the initial part of the aorta;
- Decreased left ventricular contractility in hypertension, nephritis leads to asynchronous contraction of the ventricles;
- Aortic regurgitation (“interrupted contraction of the left ventricle - Obratsovs’ bisystolia”);
- Complete right bundle branch block and resulting delay in onset of the right ventricular systole.

Splitting of S_2 occurs more frequently than S_1 .

Physiological splitting of S_2 into audibly distinct aortic (A_2) and pulmonic (P_2) components is due to a normal physiological cause: respiration. Normally, the aortic valve closes just before the pulmonary valve, but they are so close together that the sound is a uniform and instantaneous S_2 . When a person takes in a deep breath, the decrease in intrathoracic pressure causes an increase in venous return. This causes the right atrium and ventricle to fill slightly more than normal, and it takes the ventricle slightly longer during systole to eject this extra blood. This delay in ejection forces the pulmonary valve to stay open a bit longer than usual, and the normally small difference between aortic and pulmonary valve closure becomes noticeable as a split S_2 at the heart base.

Pathological splitting of S_2 may be due to many causes: delayed activation of the right ventricle in right bundle branch block, left ventricular ectopic beats, a left ventricular pacemaker; or delayed pulmonic valve closure because of right ventricular volume overload associated with right ventricular failure.

Pathological splitting of S_2 occurs in:

- Mitral stenosis – delayed pulmonic valve closure because of right ventricular volume overload, and prolongation of the right ventricular ejection;
- Pulmonary stenosis or pulmonary embolism is characterized by prolongation of the right ventricular systolic ejection period and thus delay closure of the pulmonic valve;
- Shortening of the left ventricular systole and early aortic valve closure occurring with mitral regurgitation because blood passes in two direction – into aorta and in the left atrium, also may produce splitting of S_2 ;

- In the patients with a ventricular septal defect blood ejected into aorta and throughout the defect to the right ventricle, left ventricular systole is thus shortened, and occurs splitting of the S_2 a result early aortic component of S_2 .
- An atrial septal defect leads to increased diastolic filling of the right ventricle and early aortic valve closure.

A delay in aortic valve closure causing P_2 to precede A_2 results in so-called reversed (paradoxical) splitting of S_2 . The most common causes of reversed splitting of S_2 are left bundle branch block and delayed excitation of the left ventricle from a right ventricle ectopic beat. Mechanical prolongation of the left ventricular systole, resulting in reversed splitting of S_2 , also may be caused by severe aortic outflow obstruction, a large aorta-to-pulmonary artery shunt, systolic hypertension, and coronary heart disease or cardiomyopathy with left ventricular failure.

Three-sound rhythms, caused by appearance of additional sounds

Triple rhythm is three-sound rhythm, which is heard at the heart apex in the patient with mitral stenosis

Triple rhythm consists of loud (snapping) S_1 , normal S_2 and additional sound, which is heard 0.07-0.13 s following S_2 , and termed OS (opening snap). The cusps of the normal mitral valve open noiseless; they are freely forced back by the blood flow ejected from the atria to the ventricle. In mitral stenosis blood thrusts against the sclerosed valve, cusps of which cannot freely move, to produce OS. The opening snap is a brief, high-pitched, early diastolic sound. This phenomenon is of considerable diagnostic value because it is heard only in the mitral stenosis.

Gallop rhythm.

Three-sound rhythm of a peculiar acoustic character, termed gallop rhythm (*bruit de galop* or *rythme de galop* according to Laubry and Pezzi), is also of considerable diagnostic value. The sounds of gallop rhythm are usually soft and low, resemble the galloping of a horse, and are best heard in direct auscultation. Gallop rhythm is heard as three separate audibly distinct sounds in approximately equal intervals.

Gallop rhythm is classified as presystolic (at the end of diastole), protodiastolic (at the beginning of diastole), and mesodiastolic (at the middle of the diastole) depend on the time of appearance of the extra sound in diastole.

Three-sound rhythm at the heart apex, in which S_1 is decreased, S_2 is normal, and the first sound is weakest – is presystolic gallop rhythm.

Presystolic gallop rhythm is heard in the patients with:

- Rheumocarditis;

- Cardiosclerosis;
- Essential hypertension;
- Chronic nephritis with arterial hypertension syndrome;
- Toxic and infectious affection of the myocardium.

Protodiastolic gallop rhythm is caused by appearance of pathological additional sound 0.12 - 0.02 s after S_2 as a result of considerably decreased tone of the ventricular myocardium (Fig. 4.31). Ventricles distended quickly during their filling with blood at the beginning of the diastole and the vibrations of myocardium thus generated are audible as an extra sound. **Presystolic gallop rhythm** occurs due to delayed atrioventricular conduction, when atrial systole is separated from the ventricular systole by a longer than normal period, and is heard as separate sound (Fig. 4.30).

Three-sound rhythm at the heart apex, in which S_1 is decreased, and the third sound is weakest – is protodiastolic gallop rhythm. This auscultation phenomenon is observed in the patients with:

- Acute and chronic myocarditis;
- Myocardiosclerosis;
- Heart failure;
- Toxicosis;
- Thyrotoxicosis;
- Anemias.

Mesodiastolic (summation) gallop rhythm arises in severe dystrophic affection of the myocardium in the patients with myocardial infarction, essential hypertension, heart valvular diseases, myocarditis and chronic nephritis. Mesodiastolic gallop rhythm is characterized by appearance of the additional sound in the middle of diastole caused by increase intensity of the S_3 and S_4 , which are heard as one gallop sound.

Systolic clicks – auscultation phenomenon, which denote prolapse of one or both cusps of the mitral valve. They also may be caused by tricuspid valve prolapse. Auscultation symptomatic may be very different: systolic clicks may be single or multiple, they may occur at any time in systole with or without a late systolic murmur. Typical peculiarity – changes of the auscultation data depend on position of the patient and exercise test. If the patient squat click and murmur slightly delayed; in the upright posture click and murmur are closer to S_1 (Fig. 4.32).

Pericardial knock – high-pitched sound occurs 0.01 – 0.06s after S_2 in the patients with constrictive pericarditis due to vibration of the adherent pericardium in abrupt dilation of the ventricle at the beginning of diastole.

Pericardial knock is better heard at the heart apex or medially toward to xiphoid.

Embryocardial or pendulum rhythm occurs in severe heart failure, attacks of paroxysmal tachycardia, high fever, etc. Tachycardia makes diastolic pause almost as short as the systolic one. A peculiar auscultative picture, in which heart sounds are similar in intensity, resembles foetal rhythm is termed embryocardia.

Cardiac murmurs

In addition to the normal heart sounds, abnormal sounds known as murmurs may be heard in auscultation. Cardiac murmurs may both endocardiac and exocardiac. *Endocardiac* murmurs occur in dysfunction of the intact valves – *functional murmurs* or in anatomical changes in the structure of the heart valves – *organic murmurs*.

Organic cardiac murmurs.

When a valve is stenotic or damaged, the abnormal turbulent flow of blood produces a murmur, which can be heard during the normally quiet times of systole or diastole.

The *mechanisms* of cardiac murmurs can be explained by the physics laws concerning the flow of liquids in tubes

Such condition as liquid flowing through a partially narrowed portion of the tube (Fig. 4.33.a) can cause turbulent flow. The intensity of noise depends on the extent of narrowing: the narrower lumen of the tube, the more intense noise. In significant narrowing of the tube, noise may weaken or even disappears. Liquid flowing from a smaller portion of the tube to a larger one can also cause vortex movement (Fig. 4.33.b). Murmur can be caused by blood flow in the vascular lumen partially obstructed by atherosclerotic plaque or thrombus (Fig. 4.33.c).

Following characteristics used to describe cardiac murmurs are timing, intensity, pitch, quality, configuration, duration, location and radiation.

Murmurs are defined in terms of their *timing* within the cardiac cycle. ***Systolic murmur*** terminates between S_1 and S_2 or begins instead of significantly decreased S_1 . ***Diastolic murmur*** begins with or after S_2 and terminates at or before the subsequent S_1 .

The *intensity* of the murmurs is graded according to the Levine scale:

- **Grade I** – Lowest intensity, difficult to hear even by expert listeners
- **Grade II** – Low intensity, but usually audible by all listeners
- **Grade III** – Medium intensity, easy to hear even by inexperienced listeners, but without a palpable thrill
- **Grade IV** – Medium intensity with a palpable thrill

- **Grade V** – Loud intensity with a palpable thrill. Audible even with the stethoscope placed on the chest with the edge of the diaphragm
- **Grade VI** – Loudest intensity with a palpable thrill. Audible even with stethoscope raised above the chest.

A cardiac murmur's *pitch* varies from high to low.

Common descriptive terms of a murmur's *quality* include rumbling, blowing, machinery, scratchy, harsh, rough, squeaky, or musical.

Location. Cardiac murmurs may not be not audible over all areas of the chest, and it is important to note where it is heard best and where it radiate to.

The location on the chest wall where the murmur is best heard and the areas to which it radiates can be helpful in identifying the cardiac structure from which the murmur originates (see Tab. 13).

Tab. 13. Best auscultatory areas of a cardiac murmurs.

Topographic classification of murmurs

Auscultatory areas	Murmur	Heart valvular disease
Heart apex	Systolic	Mitral regurgitation
	Diastolic	Mitral stenosis
Second intercostal space at the right sternal edge	Systolic	Aortic stenosis
	Diastolic	Aortic regurgitation
Second intercostal space at the left sternal edge	Systolic	Pulmonary stenosis
	Diastolic	Pulmonary regurgitation
Base of the xiphoid	Systolic	Tricuspid regurgitation
	Diastolic	Tricuspid stenosis

Radiation. Some cardiac murmurs may be heard not only in standard auscultatory areas but also transmitted in the direction of blood flow. This phenomenon is known as radiation (Tab. 14).

Murmurs radiate in either a forward (*ejection murmurs*) or backward direction (*regurgitation murmurs*).

Tab. 14. Auscultatory areas and radiation of murmurs in heart valvular diseases

Heart valvular disease	Murmur	Auscultatory areas	Radiation areas
Mitral regurgitation	Systolic	Heart apex	Axillary region
Mitral stenosis	Diastolic	Heart apex	No radiation

Aortic regurgitation	Diastolic	Second intercostal space at the right sternal edge	Botkin-Erb's point, sometimes heart apex
Aortic stenosis	Systolic	Second intercostal space at the right sternal edge	Subclavian, carotid arteries, interscapular region

Systolic murmurs

Aortic stenosis. One of the most frequent pathologic systolic murmurs is due to aortic stenosis. The murmur of aortic stenosis heard best over “aortic area”, second intercostal space along right sternal border, with radiation into the neck, along carotid arteries, into the interscapular region (ejection murmur). The intensity of murmur varies directly with the cardiac output. It has a harsh quality, are usually crescendo-decrescendo in configuration (as the velocity of ejection increases, the murmur gets stronger, and as ejection declines, its diminished), is typically midsystolic murmur (starts shortly after S₁, when the left ventricular pressure becomes enough to open aortic valve; ends before left ventricular pressure falls enough to permit closure of the aortic leaflets).

Pulmonary stenosis. The murmur of pulmonary stenosis is heard best in the “pulmonic area”, second intercostal space along the left sternal border. The murmur can be heard radiating into the neck or the back (ejection murmur), has a harsh quality, a crescendo-decrescendo shape, and midsystolic duration.

Mitral regurgitation. Systolic murmur in mitral regurgitation is best heard at the heart apex, with radiation into the left axilla (regurgitant murmur). The quality of murmur is usually described as blowing, frequency – as high-pitched, the configuration of murmur may vary considerably, and its duration is holosystolic.

Tricuspid regurgitation. The holosystolic murmur of tricuspid regurgitation is best heard at the base of the sternum, generally softer than that of mitral regurgitation, and frequently increases during inspiration.

Diastolic murmurs

Aortic regurgitation. The murmur of aortic regurgitation best heard in the second intercostal space along left sternal edge, it widely radiates along the left sternal border (Botkin-Erb's point) and to be well transmitted to the heart apex (regurgitant murmur). This murmur is usually characterized as blowing, generally high-pitched, decrescendo (since there is progressive decline in the volume of regurgitation during diastole), and early diastolic murmur. In severe regurgitation, it may be holodiastolic. The soft, rumbling, low-pitched, mid- to late diastolic murmur at the heart apex (*Austin Flint murmur*) may be detected in severe aortic regurgitation. It is thought to be due to a functional mitral

stenosis, as the backflow blood from the aorta presses on the mitral valve, slightly occluding the flow from the left atrium.

Pulmonary regurgitation. The murmur of pulmonary regurgitation is best heard in the second intercostal space to the left of the sternum, with radiation along left sternal edge (regurgitant murmur), high-pitched, decrescendo, early diastolic murmur. The diastolic murmur of pulmonary regurgitation without pulmonary hypertension is softer, and low- to medium-pitched.

In mitral stenosis functional early diastolic, high-pitched, with a decrescendo quality murmur is heard over the pulmonic area. This murmur, known as *Graham Steel murmur*, begins with accentuated S₂, and is caused by dilation of the pulmonary artery due to significant pulmonary hypertension.

Mitral stenosis. The murmur of mitral stenosis is best heard at the heart apex with a little radiation. It is usually described as low-pitched, rumbling, characteristically follows OS, and can be heard best with the patient in the left lateral decubitus position. The murmur is nearly holodiastolic with presystolic accentuation, or presystolic crescendo, or early diastolic (protodiastolic) decrescendo.

Tricuspid stenosis. The diastolic murmur associated with tricuspid stenosis is localized to a relatively limited area over the xiphoid, low-pitched, rumbling, and like most right-sided events, may be stronger during inspiration.

TEST CONTROL

1. ***In which pathology decreased first sound at the heart apex is observed?***
 - A. Pulmonary artery regurgitation
 - B. Aortic stenosis
 - C. Mitral stenosis
 - D. Tricuspid regurgitation
 - E. Tricuspid stenosis
2. ***In which listening point the second sound is louder than the first?***
 - A. 5th intercostal space on the left midclavicular line
 - B. 2nd intercostal space to the right of the sternum
 - C. In the Botkin-Erb's point
 - D. On the base of the sternum
 - E. 3rd intercostal space to the right of the sternum
3. ***In which pathology triple rhythm is auscultated?***
 - A. Aortic stenosis
 - B. Mitral regurgitation
 - C. Aortic regurgitation
 - D. Mitral stenosis

- E. Pulmonary artery stenosis
- 4. ***Which factors define the loudness of the second heart sound?***
 - A. Condition of the right atrial myocardium
 - B. Condition of the left atrial myocardium
 - C. Pressure in the pulmonary artery
 - D. Condition of the mitral valve
 - E. Condition of the tricuspid valve
- 5. ***In which pathology increased first sound at the heart apex is observed?***
 - A. Mitral valve regurgitation
 - B. Aortic stenosis
 - C. Mitral stenosis
 - D. Tricuspid stenosis
 - E. Pulmonary artery regurgitation
- 6. ***What is the character of the first sound in the triple rhythm?***
 - A. Increased in the Botkin-Erb's point
 - B. Increased at the heart apex
 - C. Decreased at the heart apex
 - D. Increased over aorta
 - E. Decreased over pulmonary artery
- 7. ***Which factors define loudness of the first heart sound?***
 - A. Condition of the left ventricular myocardium
 - B. Pressure in the pulmonary artery
 - C. Condition of the pulmonary artery valve
 - D. Pressure in the aorta
 - E. Condition of the aortic valves
- 8. ***In which pathology decreased second sound over aorta is observed?***
 - A. Mitral regurgitation
 - B. Mitral stenosis
 - C. Atherosclerosis of the aorta
 - D. Aortic regurgitation
 - E. Essential hypertension
- 9. ***In which heart valvular disease systolic murmur in the second intercostal space to the right of the sternum is heard?***
 - A. Mitral stenosis
 - B. Mitral regurgitation
 - C. Aortic stenosis
 - D. Aortic regurgitation
 - E. Tricuspid regurgitation
- 10. ***Which components take part in the formation of the second heart sound?***
 - A. Atrial component

- B. Tension and vibration of the mitral valve
 - C. Vibration of the aortic wall
 - D. Tension and vibration of the tricuspid valve
 - E. Tension of the right ventricular myocardium
11. ***In which pathology accentuated second sound over aorta is observed?***
- A. Mitral regurgitation
 - B. Aortic regurgitation
 - C. Mitral stenosis
 - D. Atherosclerosis of the aorta
 - E. Aortic stenosis
12. ***In which heart valvular disease systolic murmur is conducted by the blood flow onto the carotid arteries?***
- A. Mitral stenosis
 - B. Mitral regurgitation
 - C. Aortic stenosis
 - D. Aortic regurgitation
 - E. Tricuspid regurgitation
13. ***In which pathology decreased first sound over heart apex and increased second over pulmonary artery are observed?***
- A. Mitral stenosis
 - B. Mitral regurgitation
 - C. Tricuspid regurgitation
 - D. Tricuspid stenosis
 - E. Aortic stenosis
14. ***In which pathology decreased first sound on the base of the sternum is observed?***
- A. Mitral regurgitation
 - B. Aortic regurgitation
 - C. Mitral stenosis
 - D. Tricuspid regurgitation
 - E. Aortic stenosis
15. ***What is listening point of murmur in mitral regurgitation?***
- A. Heart apex
 - B. 2nd intercostal space to the right of the sternum
 - C. 2nd intercostal space to the left of the sternum
 - D. Base of the xiphoid process
 - E. Botkin-Erb's point
16. ***In which pathology accentuated second sound over pulmonary artery is observed?***
- A. Mitral stenosis
 - B. Aortic stenosis

- C. Aortic regurgitation
 - D. Pulmonary artery regurgitation
 - E. Tricuspid regurgitation
17. ***What is the listening point of the murmur in the mitral stenosis?***
- A. 2nd intercostal space to the right of the sternum
 - B. 2nd intercostal space to the left of the sternum
 - C. Heart apex
 - D. Base of the xiphoid process
 - E. Botkin-Erb's point
18. ***Which component take part in the formation of the first heart sound?***
- A. Closing and vibration of the aortic valve
 - B. Vibration of the aortic wall
 - C. Closing and vibration of the pulmonary artery valves
 - D. Vibration of the pulmonary artery wall
 - E. Tension and contraction of the left ventricular myocardium
19. ***In which pathology increased first sound at the heart apex and accentuated second sound over pulmonary artery is observed?***
- A. Mitral stenosis
 - B. Pulmonary artery stenosis
 - C. Mitral regurgitation
 - D. Tricuspid regurgitation
 - E. Aortic stenosis
20. ***What is the best listening point of the murmur in the aortic stenosis?***
- A. Heart apex
 - B. 2nd intercostal space to the right of the sternum
 - C. 2nd intercostal space to the left of the sternum
 - D. Base of the xiphoid process
 - E. Botkin-Erb's point

Answers: 1B, 2B, 3D, 4C, 5C, 6B, 7A, 8D, 9C, 10C, 11D, 12C, 13B, 14D, 15A, 16A, 17C, 18E, 19A, 20B.

Methodical instructions

**AUSCULTATION OF THE HEART. NORMAL HEART SOUNDS,
REDUPLICATION OF THE SOUNDS, ADDITIONAL SOUNDS
(TRIPLE RHYTHM, GALLOP RHYTHM), ORGANIC
AND FUNCTIONAL HEART MURMURS**

Methodical instructions for students

Authors: T.V. Ashcheulova
O.M. Kovalyova
O.V. Honchar

Chief Editor Ashcheulova T.V.

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