#  PATHOPHYSIOLOGY OF CARDIOVASCULAR SYSTEM

Under the conditions of the organism’s increased needs for nutrients and oxygen the heart activity may be increased 5 times, and total volume of vascular lumen may be increased or decreased 1.5 times. But in spite of this adaptability, under the conditions of severe damage the compensatory possibilities of the cardiovascular system are exhausted, and circulatory (cardiovascular) insufficiency occurs. Circulatory insufficiency is the state when the cardiovascular system cannot provide organism with the oxygen and power materials in amounts corresponding to its metabolic needs.

According to the developmental mechanism and clinical manifestations the following types of the circulatory insufficiency are distinguished:

1) cardiac insufficiency (heart failure) – is caused by weakened activity of the heart as a pump;

2) vascular insufficiency – is caused by weakened vascular tension;

3) mixed cardiovascular insufficiency – in some cases at first the cardiac failure is observed, and then the vascular insufficiency joins up; in other cases – vice versa.

**According to the character and rate of development acute and chronic forms of the circulatory insufficiency are distinguished.**

The acute heart failure includes cardiac asthma and pulmonary edema. The acute vascular insufficiency is observed in shock, collapse and syncope.

The chronic circulatory insufficiency occurs as a result of developing diseases of the blood circulation system (atherosclerosis, coronary insufficiency, hypertensive disease, valvular disease, etc.). 3 stages of the chronic circulatory insufficiency are distinguished:

I - initial (compensated), latent circulatory insufficiency is not revealed in resting state, but the cardiovascular system cannot provide blood supply of organs during physical work and hypoxia occurs; working capacity is limited;

II - marked circulatory insufficiency is characterized by disturbance of hemodynamics in the resting state; organism’s vital activity is maintained by the way of the maximal use of its rserve compensatory mechanisms; work capacity is sharply limited;

III - the decompensated form of the circulatory insufficiency is characterized by the breakdown of the compensatory mechanisms, stable changes in metabolism and distrophy in organs, loss of work capacity.

**Heart failure is the state in which the heart cannot pump into the peripheral blood vessels the blood in amounts required by the organism. Etiologic factors causing cardiac insufficiency are divided into 4 groups:**

1. lesions in the myocardium – infectious and toxic myocardites, disorders of coronary circulation, hypoxia, intoxications, myocardial dystrophies caused by metabolic disorders and avitaminoses, etc;
2. increased cardiac activity and overstrain of myocardium – valvular diseases, increased pressure in the greater and lesser circulation, paroxysmal tachycardia, etc;
3. disturbed pericardiac function;
4. mixed type (for instance, rheumatic myocarditis with thyrotoxicosis, anemia, valvular disease).

Acute heart failure (sudden and rapid development of heart failure) occurs as a result of acute weakness of the cardiac muscle’s contractile function in myocardial infarction, pulmonary embolism, cardiac tamponade, paroxysmal tachycardia, ventricular fibrillation, hypertensive crises, occlusion of the left atrioventricular opening by the ball – valve thrombus, Morgagni – Adams – Stokes syndrome, etc. Frequently the state of the patient in acute heart failure resembles the shock and is called “cardiogenic shock”.

Chronic heart failure is observed in myocardial ischemia resulted from atherosclerotic coronary artery diseases, multivalvular heart disease, arterial hypertension , chronic lung diseases resulting in hypoxia and pulmonary arterial hypertension, progression of acute into chronic failure.

In chronic heart failure compensatory mechanisms (tachycardia, cardiac dilatation and hypertrophy) try to make adjustments so as to maintain adequate cardiac output which often results in well-maintained arterial pressure.

Frequently in cardiac insufficiency activity of one of the ventricles is disturbed more than that of another one. But both left-sided (left ventricular) and right-sided (right ventricular) heart failures may develop into total heart failure. Rarely (for instance, in adhesive pericarditis) simultaneously the total heart failure may develop.

Left ventricular failure is initiated by stress to the left heart and sharply increased resistance in the greater circulation. The major causes are ischemic heart disease, aortic stenosis and mitral insufficiency, left ventricular aneurism, myocardial diseases (cardiomyopathies, myocarditis), arterial hypertension, restrictive pericarditis, etc.

The clinical manifestations of left-sided heart failure result from accumulation of fluid upstream in the lungs and decreased left ventricular output. Accordingly, the major pathological changes are:

1. pulmonary congestion and edema causing dyspnea and orthopnea;
2. decreased left ventricular output causing hypoperfusion and diminished oxygenation of tissues leading to ishemic acute tubular necrosis in kidneys, hypoxic encephalopathy in brain, muscular weakness and fatigue in skeletal muscles, etc.

So, left ventricular failure results in congestion in the lesser circulation. The acute left-sided heart failure manifests itself as cardiac asthma and pulmonary edema. In chronic left ventricle failure the continuous dyspnea and cough are observed.

Right ventricular heart failure occurs more frequently as a consequence of left ventricule heart failure. But some conditions affect the right ventricle primarily producing right-sided heart failure:

1. cor pulmonale (pulmonary heart disease) in which right heart failure occurs due to intrinsic lung diseases (emphysema, pneumosclerosis, etc.);
2. pulmonary stenosis;
3. pulmonary hypertension secondary to pulmonary tromboembolism;
4. myocardial diseases (cardiomyopathies, myocarditis);
5. constrictive pericarditis.

In myocardial diseases usually both ventricles are damaged, but since the right ventricle myocardium is weaker, its function is disturbed earlier. The clinical manifestations of right ventricle failure are upstream of the right heart such as greater circulation and portal venous congestion and reduced cardiac output. Accordingly, the pathological changes are:

1. systemic venous congestion in different tissues and organs (subcutaneous edema on dependent parts, passive congestion of the liver, spleen and kidneys, ascites, hydrothorax, congestion of leg veins and neck veins);
2. reduced cardiac output resulting in circulatory stagnation causing anoxia, cyanosis and coldness of extremities.

## The acute right-sided heart failure results from pulmonary embolism, rupture of interventricular septum, right ventricle infarction.

Thus, in early stage the left heart failure manifests with features of pulmonary congestion and decreased left ventricular output, while the right heart failure presents with systemic venous congestion and involvement of the liver and spleen. But congestive heart failure combines the features of both left and right failures.

Mechanism of clinical manifestations resulting from heart failure can be explained on the basis of mutually inter-dependent backward and forward failure:

1. backward heart failure-either of the ventricles fails to eject blood normally, resulting in rise of end diastolic volume in the ventricle and increase in volume and pressure in the atrium which is transmitted backward producing elevated pressure in the veins;
2. forward heart failure – clinical manifestations result directly from failure of the heart to pump blood, causing diminished flow of blood to the tissues (especially diminished renal profusion and activation of renin – angiotensin aldosterone system).

The general pathogenesis of the heart failure is connected with diminished amount of the blood pumped by the heart into arteries, and venous congestion. Part of the blood cannot pass from ventricles into aorta and pulmonary trunk in systole (as a result of increased resistance to the blood flow) or once again comes back into ventricles in diastole (in valvular defects). Therefore, to the end of the diastole the pressure in the ventricles is above normal, causing there overstrain and more powerful contractions which lead to hypertrophy of the cardiac muscle. Gradually dystrophical and sclerotic changes occur in myocardium, its contractility is decreased; amount of the blood pumped by the heart into periphery in systole is decreased, congestion in lungs and veins of the greater circulation develops. Thanks to increased heart rate during certain time the cardiac output remains on normal level. In the conditions of the decreased blood flow tissues assimilate the greater part of the oxygen from the arterial blood, and arteriovenous difference in oxygen content is increased. As a result of pulmonary congestion, the vital capacity and respiratory surface of the lungs are decreased. Less oxygen enters the blood and carbon dioxide content is increased. In tissues hypoxia develops, and acid metabolic products are accumulated, respiratory center is stimulated and dyspnea develops.

**In the etiology of the vascular insufficiency mainly the following factors play a part:**

1. Decreased circulating blood volume (resulted from loss of blood, vomiting, diarrhea, adrenal gland dysfunction).
2. Increased total volume of vascular bed. This may be caused by:
3. disturbed vasomotor innervation (resulted from stimulation of the vasomotor center in myocardial infarction, pulmonary embolism,psychogenic reactions, traumata, hyperventilation);
4. toxic damages to blood vessels ( poisoning, acute infections);
5. combinated influence of nervous and humoral factors (anaphylaxis, burn shock, traumatic shock ).

So, the main factor in the pathogenesis of the vascular insufficiency is disturbed correlation between the total volumes of the vascular bed and circulated blood volume. This leads to decreased venous pressure and that of blood flow to the heart which pumps less blood to peripheral vessels; oxygen supply to tissues is weakened.

Acute vascular insufficiency is caused in shock, collapse, syncope.

In chronic vascular insufficiency arterial and venous pressure are decreased, pulse becomes weaker. Dizziness (especially when taking the vertical position) and dyspnea (especially during physical work) are observed.

Circulatory insufficiency is accompained by changes in hemodynamic indices. The stroke volume is sharply diminished. Therefore, even though the heart rate increases, but the cardiac output and cardiac index are decreased. At the first stage of the chronic circulatory insufficiency the cardiac index may be normal. But during tachycardia diastole is shortened, the cavities of the heart are not filled with blood sufficiently, and the systole is weakened. At the decompensated period of the circulatory insufficiency the cardiac output and cardiac index are decreased. In acute vascular (traumatic shock) and cardiovascular (cardiogenic shock) insufficiency these indices may be decreased twise as normal.

In heart failure the rate of blood flow is decreased, and the time of complete circuit of the blood is increased up to 60 sec (against the normal 20-22 sec).

Under conditions of slowed- up blood flow tissues assimilate more oxygen from the blood in capillaries and utilization coefficient of oxygen is increased.

In cardiac insufficiency the blood is mobilized from depots, and erythropoiesis is accelerated. So, frequently circulating blood volume is increased. But sometimes it is decreased in heart failure connected with mitral stenosis. Vascular insufficiency is also accompained by decreased circulating blood volume.

Increased circulating blood volume in circulatory insufficiency is called the positive decompensation. The left ventricle insufficiency accompanied by the positive decompensation is very dangerous. Because congestion in lesser circulation promotes development of the pulmonary edema.

In vascular insufficiency arterial pressure decreases. For cardiac insufficiency decreased systolic pressure is particularly characteristic.

One of the first signs of the heart failure is increased venous pressure. Usually in the left ventricle insufficiency the venous pressure is increased in the lesser circulation, and in the right ventricle insufficiency- in the greater circulation.

The main clinic symptoms of the circulatory insufficiency are the following:

1. Tachycardia- is the compensatory reaction realized by the reflex way in response to the venous congestion, hypoxemia, acidosis. Tachycardia, though promotes increase of cardiac output, but when protracted, it exhausts the cardiac muscle and the symptoms of the heart failure become more severe.
2. Dyspnea- develops in response to hypoxia and acidosis as a result of stimulation of the respiratory center by the humoral and reflex way. Particularly severe dyspnea is observed during congestion in the lesser circulation.

In decompensated heart diseases congestion in the lungs becomes more severe in a lying position of the patient. In the vertical position part of the blood passes into lower extremities and abdominal cavity organs; myocardium's activity becomes facilitated. Sometimes the patient is forced even to sleep in sitting position / forced position /. This is particularly characteristic of left ventricle insufficiency. Sometimes in acute left ventricle insufficiency paroxysmal dyspnea is observed. This is called cardiac asthma. Hypertensive crisis, aortic valve failure, left ventricular myocardial infarction are accompanied by attacks of cardiac asthma.

3. Cyanosis of skin and mucous membranes- develops as a result of accumulation of reduced hemoglobin in the blood. It is more marked in the fingers, lips, tip of the nose where the circulation rate is low. In severe cases cyanasis is observed all over the body.

4. Congestion- develops as a result of insufficiency of the left ventricle (in the lesser circulation) or right ventricle (in the greater circulation ). Protracted right ventricle insufficiency results in changes in the internal organs especially in the liver (atrophy and development of the connective tissue). Venous congestion due to cardiac failure may lead to development of edema.

5. Edema- increased intracapillary pressure, delay of sodium and water in the organism, decreased blood content of albumins and oncotic pressure (as a result of disturbed hepatic function) play a part in the pathogenesis of the cardiac edema. At first edema appears in lower extremities and then spreads all over the subcutaneous space and skin, especially to legs, hands, back, the anterior abdominal wall. As distinct from the renal edema the cardiac edema rarely spreads to the tissues of the face, neck,chest.

6. Polycythemia- is resulted from mobilization of the blood from depots and stimulation of erythropoiesis. Although the organism's compensatory reaction in heart failure, but polycythemia results in increased viscosity of the blood and makes difficult the heart activity.

The circulatory insufficiency connected with lesions in myocardium may be caused by disturbances in coronary circulation, intoxications, hypovitaminoses, anemias, infectious, inflammatory and autoallergic processes all of which are usually accompanied by disorders in myocardial metabolism. In some of these cases sufficient energy is not formed in the cardiac muscle, in others- synthesis of proteins participating in it's contraction, is disturbed.

Disorders in the coronary circulation, acute and chronic respiratory insufficiency and anemia play a special part in lesions of myocardium connected with hypoxia (weakened aerobic metabolism).

The causes of the metabolic disorders in myocardium are the following:

1. deficiency of the oxidation substrate;
2. decreased activity of the oxidation enzymes (oxidases);
3. disturbed relation between oxidation and phosphorylation

2/3 of the mortality in cardiovascular diseases is resulted from ischemic heart disease (IHD) or coronary heart disease (CHD). This is acute or chronic form of cardiac disability arising from imbalance between the myocardial supply and demand for oxygenated blood. Narrowing or obstruction of the coronary arterial system is the most common cause of myocardial anoxia. Men develop ischemic heart disease earlier than women and death rates are also slightly higher for men than for women until the menopause.

**Coronary heart disease includes stenocardia (angina pectoris), myocardial infarction and cardiosclerosis.**

In resting state through the coronary arteries 200-250 ml of blood passes per minute. Decrease of this level is called the absolute deficiency of the coronary circulation. In physical overstrain the coronary circulation may increase up to 3-4 l/min. Sometimes even the increased blood supply of the myocardium cannot satisfy the increased needs (as a result of functional overstrain) of the cardiac muscle. This is called the relative insufficiency of the coronary circulation.

In 90 % of the cases of stenocardia and myocardial infarction atherosclerosis of the coronary arteries is revealed (protracted functional overstrain of the cardiac muscle atherosclerotic changes). Spasm of the coronary arteries may cause myocardial ischemia. Clinically coronary spasm manifests itself as stenocardia. But when it continues 20-30 min., necrotic foci (infarct) develop in myocardium. Etiologic factors of the ischemic heart disease include also thrombosis, thromboembolism of the coronary arteries and rarely-inflammatory processes in them (panarteritis, periarteritis nodosa, rheumatic vasculitis).

The factors promoting development of the ischemic heart disease, that is, “risk factors”, are the following: hypercholesterolemia, arterial hypertension, obesity, hypokinesia, nicotinism, alcoholism, some endocrine diseases (diabetes mellitus, hypothyrosis, etc.).

**Stenocardia (angina pectoris) is a clinical syndrome of ishemic heart disease resulting from transient myocardial ischemia.** This disease is characterized by sudden attacks of intense pain in the substernal or precordial region of the chest which is aggravated by an increase in the demand of the heart and relieved by a decrease in its work. Often the pain radiates to the left shoulder, arm, neck, jaw or right arm. It is also marked by increased sensitivity zones in the skin (the Head zones). The pain is due to accumulation of lactic acid, pyruvic acid, ketone bodies, potassium ions, kinins in the ischemic parts of the heart. The stimuli transmitted from the heart through sympathetic fibers and the stellate ganglion or sympathetic trunk to the spinal cord irradiate to the sensory cells of the given segment. The reflex contractions of the intercostal muscles give rise to a sense of constriction in the chest (stenocardia). Various vegetative reflexes arise, including tachycardia, elevated blood pressure, perspiration, salivation and sometimes altered sensitivity and a drop in the temperature of the skin in the cardiac region. The patient develops a sense of apprehension of impending death. The attacks may end lethally, as a result of paralysis of the heart. Angina pectories most commonly occurs in elderly people.

There are 3 overlapping clinical patterns of angina pectoris with some differences in their pathogenesis:

1. Stable or typical angina- is the most common pattern. It is characterized by attacks of pain following physical exertion or emotional excitement and is relieved by rest. Its pathogenesis is connected with stenosing atherosclerosis of coronary arteries which cannot perfuse myocardium adequately when the workload on the heart increases. During the attack there is depression of ST segment in the ECG due to poor perfusion of the subendocardial region of the left ventricle but there is no elevation of enzymes in the blood as there is no irreversible myocardial injury.
2. Prinzmetal’s variant angina- is characterized by pain at rest and has no relationship with physical activity. It may occur as a result of sudden vasospasm of coronary trunk induced by coronary atherosclerosis, or may be due to release of humoral vasocostrictors by most cells in the coronary adventitia. ECG shows ST segment elevation due to transmural ischemia. These patients respond well to vasodilatory like nitroglycerin.
3. Unstable or crescendo angina (also referred to as **“** pre- infarction angina **”** or **“** acute coronary insufficiency **”)** - is the most serious pattern model of angina which is characterized by more frequent onset of pain of prolonged duration and occuring often at rest. It is thus indicative of an impending myocardial infarction. Multiple factors are involved in its pathogenesis which include stenosing coronary atherosclerosis, complicated coronary plaques (superimposed thrombosis, hemorrhage, rupture, ulceration), platelet thrombi over atherosclerotic plaques and vasospasm of coronary arteries. More often the lesions lie in a branch of the major coronary trunk so that colloterals prevent infarction.

**Acute myocardial infarction is the most important consequence of coronary artery disease. Myocardial infarction is the necrosis in a part of cardiac muscle which develops as a result of cessation or weakening of the blood circulation.**

In the most of cases the myocardial infarction develops in the area which is supplied by the coronary artery with severe atherosclerotic changes. Because atherosclerotic plaques narrow lumen of the coronary artery, and myocardium’s blood supply is disturbed. Besides, there are more suitable conditions for thrombogenesis in the vessels with atherosclerotic changes; atherosclerosis increases sencibility of the coronary vessels to the factors causing spasm and decreases that to the spasmolytic factors. The atherosclerotic plaque may reject and, as embolus, obstruct the narrow part of the coronary artery.

The clinical manifestations of the myocardial infarction depend on the organism’s reactivity, localization and size of the necrotic area. One of the main signs of this disease is pain. The developmental mechanism and localization of the pain in myocardial infarction are similar to those in stenocardia. But in myocardial infarction the pain is more violent, continues long and does not pass of under the influence of drugs eliminating coronary spasm (nitroglycerin).

In 18-20 % of patients, especially in persons with protracted myocardial ischemia (atherosclerotic cardiosclerosis), myocardial infarction proceeds without pain. In these cases the disease begins with symptoms of the heart failure.

Pain syndrome is the main motive factor of the neurohumoral changes caused by myocardial infarction in the organism. Under the influence of pain a large amount of catecholamines and glucocorticoids is secreted into the blood which cause changes in the metabolism and hemodynamic indices. Excess of catecholamines exercises histotoxic action: necrotic processes in cardiac muscle become deeper, zone of infarction is enlarged and pain sensation becomes more violent. So, the vicious circle comes into being.

Owing to lesion of biological membranes large amount of intracellar enzymes are released into blood from zone of infarction. Increased activity of these enzymes in the blood is one of the important differential- diagnostic signs of the myocardial infarction.

Weakened contractility of the cardiac muscle (as a result of ischemia and necrosis) results in decreased amount of the blood that is pumped into aorta in systole, and coronary blood flow becomes weaker. Zone of infarction becomes wider, and contractility of the myocardium is still more decreased. So, another vicious circle comes into being.

3 zones are distinguished in the damaged area in the acute period of the myocardial infarction: 1) necrotic zone (in the center); 2) zone of lesion (around the necrotic zone); 3) ischemic zone (between the zone of lesion and intact cardiac muscle).

These changes may be of subendocardial, subepicardial, intramural or transmural (complete transverse lesion of the cardiac wall) character.

The electrocardiographic symptoms of ischemia, lesion and necrosis depend on their size, depth, localization, stage, of the disease, etc. Therefore, the electrocardiographic changes in myocardial infarction are diverse. Ischemia manifests itself mainly in changing of T wave, lesion - in shift of S-T segment. In the area of necrosis QRS inversion is observed. Charachteristic ECG changes include ST segment elevation, T wave inversion and appearance of wide, deep Q waves. In transmural infarction QS complex is observed, that is, R wave completely disappears. Changes in surface potentials of myocytes and formation of ectopic foci of exitation in the acute period of the myocardial infarction frequently result in different disturbances in cardiac rhythm (paroxymal tachycardia, extrasystolic arrhythmia, etc). In severe cases arrhythmias result in ventricular fibrillation which is one of the dangerous complications of the myocardial infarction often causing death.

One of the main complications on the acute period of the myocardial infarction is cardiogenic shock. The cardiogenic shock is observed in 15% of all persons with myocardial infarction and about 80% of these cases result in death.

The main cause of the cardiogenic shock is pain. Its clinical manifestations are: the sudden weakness, pallor and cyanosis of skin, sharp decrease of the arterial pressure, weak pulse, general malaise, cold sweat. Sometimes the short loss of consciousness is observed.

Low contractility of the cardiac muscle leads to decreased stroke volume and cardiac output. This results in reflex hypersecretion of catecholamines. Vascular tension and peripheral resistance are increased, microcirculation is disturbed. Under these conditions aggregation of blood cells is accelerated, microaggregates and microthrombi in capillary system still more increase the peripheral resistance, the heart activity becomes difficult, the blood flow into the heart is decreased, blood supply of tissues is aggravated, the state of hypoxia becomes deeper. So, in the course of the myocardial infarction accompanied by cardiogenic shock the vicious circle comes into being: metabolic disorders in tissues result in secretion of large amounts of vasoactive substances into the blood which aggravate the circulatory disorders. These, in their turn, deepen metabolic disorders.

As a result of metabolic disorders and acidosis the intracellular enzyme systems are activated, and numerous small necrotic foci are formed in cardiac muscle, liver, kidneys.

Myocardial infarction ends with cicatrization of the necrotic area (organization), after which the cicatricial tissue remains. This is called postmyocardial infarction cardiosclerosis. If autolysis predominates in the necrotic zone, it is rapidly dissolved (myomalacia). This may result in perforation of the cardiac wall and hemorrhage into the pericardiac cavity which causes cardiac tamponade. Dilatation of the cadiac cavities in the area of vast postmyocardial infarction scar causes aneurysm.

The widespread method to reproduce the experimental model of the myocardial infarction is ligation of one of the large branches of the coronary artery (frequently- the anterior interventricular artery). Sometimes myocardial infarction is reproduced by the way of injection into the cardiac muscle of chemical substances causing necrosis (acids, alkalies, calcium chloride solution, etc ).

Some types of myocardial necrosis that are not connected with disorders of coronary circulation, are caused by electrolytes, hormones, metabolic products of toxic action, autoimmune processes, etc:

1. Elictrolyte- steroid necrosis of myocardium was first reproduced by Selye who administered steroid hormones (cortisone, desoxycorticosterone) into white rats which were given a food rich of sodium chloride. The mechanism of this type of necrosis is the following. Corticosteroids increase the amount of sodium entering the myocytes, osmotic pressure in the cells is raised, more water enters them, and the cell membrane is injured. Therefore, salt-free diet must be prescribed to the patients in the period of treatment with corticosteroids.
2. Neurohumoral necrosis- develops under the conditions of chronic irritation of vagus nerve, hyperfunction of the sympathetis nervous system, damage to different areas of the brain (hypothalamus, brain stem, etc.). Similar changes may be caused by administration of a large amount of epinephrine and norepinephrine into the blood. In stress situations a large amount of catecholamines are accumulated in the cardiac muscle. Their small doses stimulate power metabolism and improve functional state of the cardiac muscle, whereas large doses cause hypoxia and exercise histotoxic action; cell enzymes are activated and biological membranes are damaged.
3. Toxic and inflammatory necroses- are caused by inflammatory processes of different origin (rheumatism, diphtheria) and a number of intoxications.
4. Necrosis of autoimmune origin- is observed in persons who got over myocardial infarction. Because the proteins which pass into the blood from necrotic area of the myocardium, possess autoimmune property and cause synthesis of antibodies.

One of the diseases causing disturbances in cardiac muscle contractility is cardiosclerosis. Its two types are distinguished:

1. Atherosclerotic cardiosclerosis- results from atherosclerosis of coronary arteries. It is connected with chronic ischemia of myocardium. Usually diffuse development of connective tissue elements cause diffuse cardiosclerosis. Postmyocardial infarction cardiosclerosis is distinguished by the vast cicatricial tissue (macrofocal cardiosclerosis).
2. Myocarditic cardiosclerosis- develops after myocardites of different etiology.

The main clinical symptoms of cardiosclerosis are connected with weakness of the cardiac muscle. Frequently this disease is accompanied by arrhythmias. Cicatrical tissue may injure the conductive system of the heart and cause heart block.

**Pathological changes in heart’s properties of automatism, excitability, conductivity and contractibility lead to disturbances in cardiac rhythm, that is, arrhythmias.** Protracted changes in frequency and rhythm of heart activity, as well as in sequence of contractions of its different parts may cause severe circulatory insufficiency.

The normal cardiac rhythm depends on activity of the sinus node which is connected by the extrcardiac nerves (sympathetic and vagus nerves). According to mechanisms of disturbanses in sinus rhythm they may be divided into 3 groups:

1. changes in the activity of the extracardiac nerves (disturbances in the activity of the cerebral cortex, basal ganglia, tonus of the vegetative nerves, reflex stimuli from the peripheral receptors, etc.);
2. changes in the reaction of the sinus node to the extracardiac influences (functional and organic disturbanses in the sinus node);
3. humoral changes (changes in the blood content of hormones, carbon dioxide and oxygen, drugs which influence the functional state of the sinus node).

The normal rate of the sinus rhythm is 60-90 beats per minute. The following changes in sinus rhythm are distinguished :

1. sinus tachycardia;
2. sinus bradycardia;
3. sinus arrhythmia.

Sinus tachycardia, that is, acceleration of the heart rate (more than 90 beats per minute) results from increased tonicity of the sympathetic nervous system or decreased tonicity of the parasympathetic nervous system. It is caused by a number of external and internal factors psychoemotional exertion, physical activity, fever, some endocrine disorders, heart failure).

The maximal frequency of impulses generated in sinus node is 160 per minute. The pulse rate surpassing this frequency is connected with focus of excitation in the cardiac muscle out of sinus node (heterotropic focus) which causes heterotropic rhythm.

The main cause of sinus bradycardia, that is, slowing of the heart rate (less than 60 beats per minute, sometimes –40 beats per minute), is increased vagus nerve tonicity. This may be resulted from increased intracranial pressure (tumors of the brain, menigitis), irritation of plexus of the vagus (inflammation and tumors of the mediastiniun organs), increase of the vagus tonicity by reflex way (diseases of middle ear, liver, kidneys). Increased tonicity of vagus nerve under the influence of humoral and pharmacological agents (poisoning by preparations of digitalis or reserpine, thyroid gland hypofunction) also may lead to sinus bradycardia.

Mechanisms of bradycardia connected with lesion in sinus node are the following:

1. disturbances in blood supply of the sinus node as a result of coronarosclerosis;
2. sinus node weakness after severe infectious diseases (abdominal typhoid);
3. decreased excitability of the sinus node caused by protracted starvation.

The neurogen bradycardia disappears after the injection of atropine, whereas in other types of bradycardia atropine’s action is weak.

Rarely sinus bradycardia may be congenital. Sinus bradycardia in healthy persons (sportsmen) is accompanied by increased stroke volume.

To cause reflex bradycardia vagus nerve receptors in the carotid artery wall are pressed. Compression of the carotid artery causes sharp decrease of the pulse rate (Chermak’s reflex). This reflex as well as Aschner-Dagnini (oculocardiac) reflex are used to eliminate fits of poroxysmal tachycardia

Sinus arrhythmia is characterized by periodic acceleration and slowing of heart rate. In most cases it is connected with the act of breathing (respiratory arrhythmia) and results from changes in the excitation of the vagal center. There are also cases of sinus arrhythmia which are connected with breathing. These are due to rheumatic myocarditis or other disturbances in the function of the sinus node (mainly of an infectious origin).

The widespread type of arrhythmias is extrasystole, that is, extraordinary contraction of the heart or its part. Extrasystole may occur also in healthy people as a result of emotional strain (raised blood content of catecholamines, which increase cardiac muscle excitability). Strong tea, coffee, nicotine, alcohol also may cause extrasystole. It may be observed in pregnant women or in the period of menopause. In neuroses that are accompanied by vagotonia often in resting state extrasystole occurs, and in the period of physical strain the cardiac rhythm is recovered. In a number of diseases of internal organs (cholecystisis, gastric and duodenal peptic ulcer, etc.) extrasystole may occur as a result of viscerocardial reflex.

 Among causes of extrasystole heart diseases play a significant part. In people over 50 one of the main causes of extrasystole is coronarosclerosis. Stenocardia is observed in the period of stenocardic attack and in the acute period of the myocrdial infarction. Frequent extrasystole in myocardial infarction may be complicated by ventricular fibrillation. Extrasystolic arrhythmia may be resulted from disturbances in electrolytic metabolism (vomiting, diarrhea, excessive intake of diuretics). Depending on the site of origination of the extraordinary excitation (heterotropic focus) the following types of extrasystole are distinguished:

1. Atrial extrasystole –extraordinary bioelectrical impulse arises in the atrial wall and its ECG sign is premature low-tension P wave which may be negative.
2. Atrioventricular extrasystole –additional impulse arises in Aschoff-Tawara node, and negative P wave is observed which may be recorded before or after QRS complex (depending on the site of heterotopic, that is, ectopic focus).
3. Ventricular extrasystole-is most common. The stimulus may arise at any point of the ventricles from the bundle of His to its peripheral terminations. As distinct from atrial extrasystole, the ventricular extrasystole is followed by compensatory pause. Because after the extrasystole the next contraction of the heart does not take place in view of the refractory state. The compensatory pause together with the brief pause before the extrasystole is equal to 2 normal pauses. The ventricular extrasystole is also characterized by absence of the P wave because the atria do not participate in extrasystolic contraction and by deformation of the ventricular complex which most occurs because the ventricles do not contract simultaneously.

High-frequency ectopic rhythm causes paroxysmal tachycardia when the heart rate may be 200 and more per minute (in children up to 300). In cases of cardiac insufficiency extrasystolic arrhythmias may lead to still greater cardiac weakness due to the fatigue of the heart working under excessive strain and the uneven filling of the vascular bed.

Disturbances in cardiac conduction are called heart block. According to the site of block the following types of disturbances in cardiac conduction are distinguished:

1. sinoauricular block;
2. intraatrial block;
3. atrioventricular block;
4. intraventricular block.

 In sinoauricular block the impulses originated in sinus node reach the auricles comparatively late (partial sinoauricular block) or does not reach at all (complete sinoauricular block). This may be resulted from increased tonicity of vagus nerve, disturbed electrolytic balance, dystrophic changes in the cardiac muscle. Experimentally sinoauricular block may be produced in frog by applying Stannius ligature (between the sinus venosus and the atrium).

In intraatrial block conduction of impulses in intraatrial conductive fibers is disturbed and in ECG duration of P wave is increased (in norm- 0.08-0.12 seconds).

Atrioventricular block arises as a result of impaired conduction in the atrioventricular node. 4 degrees of this block are distinguished:

I-prolonged conduction may not affect the rhythm of ventricular contractions but ECG shows longer PQ interval (up o.4 sec. instead of the normal 0.12-0.20 sec.).

II-is characterized by an appreciable change in the interrelation between the atrial and ventricular contractions; from time to time, after every 7-10 beats, one ventricular contraction is lost. The periodic intervals between ventricular complexes are called Wenkebach-Samoylov periods.

III-after every 2-3 beats one ventricular contraction is lost.

So, an incomplete atrioventricular heart block (the II and III degrees) may give rise to such a rhythm when one ventricular contraction corresponds to two or three atrial contractions.

IV-completely interrupted conduction between the atria and ventricles results in complete atrioventricular heart block (complete atrioventricular dissociation): the atria contract in response to the impulses arising in the sinoatrial node, which are not transmitted to the ventricles and the latter contract independently of the atria in responses to impulses arising automatically in the atrioventricular node or in the bundle of His. The atria contract in one rhythm (60-80 beats per minute), and the ventricles in another rhythm (20-40 beats per minute), that is, the number of P waves is more than that of QRS complexes.

Atrioventricular blocks of the III-IV degrees may result in dangerous complication called Morgagni-Adams-Stokes syndrome, the signs of which are connected with temporary cessation of the heart’s effective activity: pallor of the skin, deep breathing, loss of consciousness, epileptiform convulsive fits. Fits continue from several seconds to several minutes and may result in death.

Atrioventricular block is frequently caused by inflammatory diseases of myocardium (rheumatism, diphtheria), cardiosclerosis, lesions of the conductive system connected with necrosis (myocardial infarction), and rarely - by disturbances in innervation (increased vagus tonicity), hemorrhage into the conductive system cells, tumors and traumata of the heart, etc.

Intraventricular block is resulted from disturbed conductivity in the bundle of His lower the site of ramification. Impulses from the sinus node reach one of the ventricles, whereas the other ventricle does not get them or receive weakened impulses through interventricular septum. Therefore, the ventricles cannot contract by equal power. Duration of QRS complex is increased (in norm 0.08-0.1 sec).

Under pathological conditions the frequency of the bioelectrical impulses generated in sinus node may be increased almost inlimitedly. When their frequency is 240-360 per minute, auricular flutter is observed. In auricular fibrillation the frequency of impulses may be 360-600 per min. and more. Different groups of auricular muscle fibers contract asynchronously and incoherently, that is, their contractions are disconnected. This is called ciliary arrhythmia.

The mechanism of auricular flutter and fibrillation may be connected with weakness of sinus node. The impulses generated in this node can’t be accepted by auricular muscles, and outside the node focus of excitation is formed which generates high-frequency impulses. The auricular flutter is connected with influence of impulses from one focus, whereas in ciliary arrhythmia there are many heterotopic foci, impulses from which are spread in different directions and cause chaotic contractions of muscles. All of auricular muscles cannot contract simultaneously, and the systoles become weaker.

Maximal frequency of impulses that can pass through atrioventricular node is 200-220 per min. Therefore, in auricular flutter and fibrillation part of impulses generated in the sinus node are blocked up in the atrioventricular node. In auricular flutter the ratio of the number of contractions of auricles to that of ventricles may be 2:1; 3:1 and so forth; rhythm of ventricular systoles is regular. In ciliary arrhythmias the frequency of impulses originated in auricles is higher, their rhythm is irregular, and more impulses are blocked.

In auricular flutter and ciliary arrhythmia the frequency of ventricular systole may be different according to which normosystolic (60-90 per minute), tachysystolic (more than 90 per minute) and bradysystolic (less than 60 per minute) arrhythmias are distinguished.

In auricular flutter instead of P wave toothed large f waves are recorded the number of which is more than that of QRS complexes, in ciliary arrhythmia uneven small f waves are recorded. In both cases QRS complexes are not changed.

Auricular flutter and fibrillation result from severe changes in cardiac muscle such as dystrophy, inflammation, necrosis, fibrosis (in myocardial infarction, cardiosclerosis, mitral stenosis, thyrotoxicosis, etc.).

Since contractions of auricles during flutter and fibrillation are not full-blooded, sufficient quantity of blood is not accumulated in the ventricles hemodynamics is disturbed. Systoles of ventricles become vain, sometimes pulse wave doesn’t come into being, and pulse rate becomes less than heart rate (pulse deficiency). Functional adaptability of heart becomes weaker.

The mechanisms of ventricular flutter and fibrillation are similar to those in auricles, but pathological foci of exitation are localized in ventricles. The main sign of the ventricular flutter and fibrillation is weak and vain contractions of cardiac muscle 150-300 times per min. In flutter the ventricles contract comparatively rhythmically, whereas in fibrillation the intervals between contractions are uneven.

Ventricular flutter and fibrillation may be caused by acute myocardial infraction, severe operations, infections and intoxications, disturbances in electrolytic balance (especially potassium metabolism); increased sensibility of the organism to some drugs, etc.

During flutter and fibrillation contractions of ventricular muscle fibers are incoherent, and the heart cannot pump the blood into periphery.

In ventricular flutter QRS complex is deformed; in fibrillation this deformation is more marked, and intervals between QRS complexes are uneven. R wave amplitudes are decreased; sometimes they disappear and asystolia occurs.

Both ventricular flutter and fibrillation may lead to death. The resuscitation measures include defibrillation of the heart by the help of electric impulses, closed chest massage, artificial respiration, injection of calcium chloride, epinephrine and norepinephrine into the heart.

A group of circulatory insufficiencies is connected with fatigue of the heart as a result of long strenous activity caused by increased amount of the blood or increased pressure in heart cavities.

Increased blood flow into heart cavities during physical work is accompanied by proportional changes in the amount of the blood pumped into aorta and pulmonary trunk.

Pathological increase in quantity of the blood accumulated in heart cavities occurs in acquired valvular insufficiencies and congenital septal defects. In valvular insufficiency deformed cusps cannot completely close orifice of corresponding cavity of the heart, part of blood flows against the blood stream back into that cavity during each systole, and the amount of the blood in cavity increases.

The main etiologic factors of the circulatory insufficiencies connected with increased pressure in heart cavities (increased pressure against movement of the blood), are the following:

1. increased arterial pressure in the greater or lesser circulations (arterial hypertension, pheochromocytoma, atherosclerosis, pulmonary emphysema);
2. stenoses of orifices of heart cavities.

In cases of stenosis more powerful contractions of cardiac muscle are required to pump the blood through the constriction, pressure in corresponding cavity increases. Nevertheless, part of the blood fails to pass through the constriction.

Generally it is the valves of the left heart that are most commonly affected, the valves of the right heart are affected much less frequently. Valvular defects of the right heart occur predominantly during the embryonal period (in intrauterine life the greater part of work is done by the right heart and after birth by the left heart). Each valvular heart disease has its own peculiarities.

Mitral insufficiency occurs most frequently. During ventricular systole part of the blood from the left ventricle returns into the left auricle. The blood pressure in the left auricle noticeably rises, the following atrial systole increases; the auricle dilates and becomes hypertrophied. During each diastole the left ventricle receives a larger volume of blood, its dilatation, increased muscular tension and development of eccentric hypertrophy take place. If the defect in the mitral valve is more marked, congestion and elevated pressure will develop in the pulmonary circulation.

Mitral stenosis (constriction of the left atrioventricular orifice) involves a rise in blood pressure in the left auricle which has difficulty in driving the blood into the left ventricle. Extreme dilatation of the left auricle occurs which sometimes turns into a flabby thin-walled chamber. Congestion develops in the pulmonary circulation and the right ventricle becomes hypertrophied.

In aortic insufficiency owing to incomplete closure of the semilunar valves the left ventricle receives blood during each diastole not only from the left auricle, but also from the aorta. Hypertrophy of the left ventricle develops. Augmented difference between the maximum and minimum blood pressure characterizes aortic insufficiency. Because the rise in blood pressure during systole is rapidly followed by a drop in pressure during diastole (part of the aortic contents rushes back into the ventricle). Hypertrophy and dilatation of the left ventricle cause dilatation of the left atrioventricular orifice, and relative (secondary) mitral insufficiency develops.

Aortic stenosis presents difficulties for discharge of the blood from the left ventricle. The blood pressure in the left ventricle rises, its hypertrophy develops.

Valvular defects of the right heart are usually congenital, and in adults develop as a result of lesions in the left heart.

In triscupid insufficiency during ventricular systole part of the blood from the right ventricle is returned to the right auricle, and it is hypertrophied. Congestion in vena cava superior and vena cava inferior, in liver, in venous system of the greater circulation develops.

Pure forms of tripcupid stenosis present difficulties for discharge of the blood from right auricle which is hypertrophied. In the period of decompensation congestion develops in vena cava superior and vena cava inferior, liver, in venous and capillary systems of the greater circulation.

A group of circulatory insufficiencies is due to diminished filling of the heart cavities which may be resulted from diminished inflow of blood to the heart by veins or disability of heart cavities to be expanded sufficiently in diastole.

Increased pressure in pericardiac cavity may limit expansion of heart cavities in diastole. Affections of the pericardium are most commonly of an inflammatory character (pericarditis). Circulatory disturbances are observed also in case of hydropericardium and hemorrhages into the pericardial cavity. Owing to accumulation of fluid and elevation of pressure in the pericardial cavity the diastolic filling of the auricles and ventricles diminishes, and functional myocardial insufficiency develops. In cases of relatively low elasticity of the pericardium the accumulated fluid exerts greater pressure on the muscular wall, cardiac tamponade develops and leads to cardiac arrest. Inflammatory exudates in the pericardium, as well as penetration of air into the pericardial cavity in injuries to the pericardium or in hemorrhages may also produce cardiac tamponade with all the ensuing consequences.

Cardiac insufficiency results in compensatory reactions in the organism some of which are connected with changes outside the heart and others result from functional and morphological changes that occur immediately in the heart. The compensatory processes connected with heart are the following:

1) tachycardia;

2) tonogenic dilatation;

3) hypertrophy of myocardium.

Tachycardia promotes increase of cardiac output. But this compensatory mechanism is not profitable from the power standpoint. Because myocardium consumes more oxygen. Besides diastole is shortened, and cardiac muscle cannot rest enough and restore its functional state.

Tonogenic dilatation of the heart is connected with accumulation of excess of the blood in the heart cavities during diastole. According to Frank- Starling law of heart this causes more powerful contractions of the heart muscle in systole, increase of heart’s mechanical work by this heterometric mechanism of compensation. Excessive dilatation of the heart’s cavities causes disorders in coronary circulation and nutrition of myocardium.

When movement of the blood in heart cavities becomes difficult homeometric mechanisms of compensation predominate, that is, the length of the cardiac muscle fibers change slightly, whereas their contractions cause high tension and the pressure in heart cavities is increased.

In certain limits of the resistance against movement of the blood the power of cardiac muscle contraction increases proportionally to this resistance. Exhaustion of the heart’s compensatory possibilities promotes circulatory insufficiency.

Contraction of myocardium by homeometric mechanism requires more power and oxygen consumption than that by heterometric mechanism. Therefore, the processes which are connected with heterometric compensatory mchanism (valvular insufficiency) go on easier than those compensated by homeometric mechanisms (stenoses).

In physiological hypertrophy (in sportsmen) the mass of heart increases proportionally to the mass of the skeletal muscles and all the heart cavities increase evenly. The heart's mechanical capacity for work increases. Since the nervous regulation of the heart also changes correspondingly, it is able to increase its activity according to the organism's needs.

But in pathological hypertrophy increase of cardiac muscle mass does not depend on skeletal muscles mass. Cardiac muscle's possibilities to increase its capacity for work are limited. Myocardium's potential reserves and adaptability to the external environment decrease. Because blood supply of the hypertrophied cardiac muscle fibers is aggravated, development of the intracardial nerve fibers lags behind that of muscles.

3 stages are distinguished in the cardiac muscle hypertrophy:

I - emergency stage - heart activity is accelerated, the functional and morphological possibilities of the cardiac muscle are mobilized and its mass is rapidly increased;

II - the stage of completed hypertrophy and relatively stable hyperfunction - the heart's mass is increased, hemodynamic indices approach the normal level, the state of complete compensation is established;

III - the stage of progressing cardiosclerosis and exhaustion of the myocardium - is characterized by profound metabolic changes and morphological disorders; part of muscle fibers perish and are replaced by connective tissue elements. These changes are accompanied by the signs of the heart failure.

The dystrophic processes in myocardium still more increase its contractility and weaken the cardiac muscle. Myogenic dilatation occurs. As distinct from the tonogenic dilatation that is observed in the period of compensation, in myogenic dilatation the amount of the blood which remains in the heart cavities after systole, is decreased. This causes increase of pressure in right ventricle and caval veins, reflex acceleration of heart rhythm (Bainbridge reflex). So, on the one hand, the hypertrophy of the myocardium as an adaptation mechanism promotes increase of the heart's mechanical work (up to 5 times), but on the other hand, it forms the basis of the functional and morphological changes in the cardiac muscle.

Hypertrophy of the heart involving its dilatation is called eccentric, and hypertrophy without dilatation - concentric;

Pathological processes may affect all types of blood vessels, but there are pathological processes that are more characteristic of certain type of vessels. For instance, in atherosclerosis infiltrative - proliferative changes in compensatory vessels (aorta and elastic type arteries) predominate, damage to metabolic vessels (capillaries and venules) cause mainly changes in their permeability, pathology in resistive vessels (arterioles and venules) is resulted from changes in their tension (hypertension and hypotension).

Disturbances in the cerebral cortex functions (neuroses), as well as damage to certain hypothalamic nuclei and medulla oblongata, disorders in the transmission of impulses from baroceptors of aortal and sinocarotid zones result in changes in blood pressure (particularly hypertension). Certain forms of encephalitis, tumors of the IV ventricle, traumata and hemorrhages in the diencephalon (particularly in hypothalamus) cause the state of long excitation in vasomotor (pressor) centers and hypertensive reactions.

Arterial hypertension is one of the frequent signs of certain diseases which are accompanied by disturbances in renal blood circulation. To explain the mechanism of influence of kidneys on the arterial pressure renopressive and renoprival theories were propounded.

According to the renopressive theory influence of kidneys on arterial pressure is connected with renin which is synthesized in juxtaglomerular cells. Ischemia in renal parenchyma results in accelerated secretion of renin which acts on angiotensinogen, causing formation of angiotensin I, then angiotensin II. Being the most powerful vasoconstrictive substance, angiotensin II increases tonicity of vascular wall muscular layer. At the same time it accelerates synthesis and secretion of norepinephrine in nerve endings and secretion of aldosterone in adrenal cortex. Aldosterone accelerates reabsorption of sodium in tubules of kidneys and causes delay of water in the organism. So, arterial pressure is still more increased.

Reciprocal relationship exists in the organism between the sodium balance and secretion of renin: decreased amount of sodium causes accelerated secretion of renin, and increased amount of sodium causes delay of its synthesis.

The renoprival theory explains the mechanism of arterial hypertension in renal diseases by decrease of depressive factors. A and E prostaglandins synthesized in the renal medulla pass into cortical substance, dilate vessels, improve blood circulation and decrease synthesis of renin. Decreased secretion of prostaglandins promotes increase of arterial pressure. Arterial hypertension was observed in a dog after removal of kidneys (its life was maintained by the help of artificial kidney).

In reality renopressive and renoprival theories are the complement of one another. So, kidneys may be regarded as universal regulators of the arterial pressure.

The systolic pressure above 160 mm Hg and diastolic pressure above 100 mm Hg are considered arterial hypertension. Since the systolic pressure between 140-160 mm Hg and diastolic pressure between 90-100 mm Hg are pathological for some (especially young) and normal for other (especially elderly) persons, they are regarded as conditionally pathological. Frequently the systolic and diastolic pressures are increased equally, but sometimes one of them may be increased to the greater degree. From this point of view two types of hypertension are distinguished:

1. systolic hypertension - results from increased contractility of the myocardium and cardiac output (in thyrotoxicosis, aortic valve failure);
2. diastolic hypertension - is connected with increased tension of arterioles and peripheral arterial resistance; it causes hypertrophy of the left ventricular myocardium.

Hypertension is generally divided into 2 types:

1. primary or essential hypertension (hypertensive disease) - constitutes about 85% patients of hypertension;
2. secondary or symptomatic hypertesion, in which the increase in blood pressure is caused by diseases of kidneys (diffuse glomerulonephritis, pyelonephritis), endocrines (pheochromocytoma, thyrotoxicosis) or some other organs - comprises about 15% cases of hypertension.

Hypertensions of hemodynamic type developing as a result of cardiovascular diseases are also classified as symptomatic hypertensions. For instance, in coarctation of aorta arterial pressure is increased only in the upper part of the body. Sometimes organic changes in the central nervous system (tumors, traumata, hemorrhages of diencephalon and medulla oblongata) are accompanied by symptomatic hypertension.

In the hypertensive disease increased arterial pressure is the first and the main symptom. In its etiology psychical trauma and emotional stress play a great part. The theory about the nervous origin of hypertensive disease is based on the teaching about the higher nervous activity and corticovisceral theory. Neurosis of the vasomotor centers localized in the cerebral cortex and subcortical area form the basis of this disease, that is, stimulations from the external environment cause long (stagnant) foci of excitation in vasopressor centers. Sympathetic nervous system is activated by conditioned and unconditioned reflex way, secretion of catecholamines is accelerated, which causes increase in peripheral vascular tension and rise in arterial pressure. Reactions of the nervous system to the external environment (hyperreactivity, age, heredity, nutrition (caloric value of the food, content of sodium chloride) are factors causing predisposition to the hypertension disease.

The main factor in the pathogenesis of the hypertension is increased tension of arterioles.

In experiment reflexogenic hypertension is produced by denervation of depressor nerve of aorta and carotid sinus in dog. To produce renal hypertension in rat its kidneys are squeezed by cellophane capsules which causes ischemia in them.

Hypotension is a condition in which the arterial pressure in adults is persistently diminished to 100/60 mm Hg and lower. The general diminution in vascular tone cannot be compensated by constriction of arteries in any part of the organism. This condition is marked by chronic circulatory insufficiency and is a result of diminished tone of small arteries, arterioles and capillaries.

Arterial hypotension may be acute (shock, collapse) or chronic. Chronic arterial hypotension is divided into 2 groups:

1. primary arterial hypotension or neurocirculatory dystonia of hypotensive type;
2. secondary or symptomatic hypotension.

In primary or neurocirculatory hypotension decreased arterial pressure is one of the first and main symptoms of the disease. It is connected with disturbances in the central regulatory mechanisms of the vascular tension. As distinct from the hypertensive disease with predominance of excitation in the activity of the vasomotor centers in neurocirculatory hypotension the inhibitory process predominates in the cerebral cortex and spreads into subcortical vasomotor centers. The tension of arterioles and peripheral resistance against the movement of the blood are decreased.

The symptomatic arterial hypotension is observed in the following cases:

1. diseases of heart (valvular defects, myocarditis);
2. diseases of liver (hepatitis, mechanical jaundice);
3. diseases of endocrine glands (myxedema, Addison's disease);
4. anemias;
5. exogenous and endogenous intoxications.

Pathogenesis of the symptomatic hypotension may be connected with the following factors:

1. weakening of the cardiac muscle contractility;
2. decrease of the circulating blood volume;
3. decrease of tension of the resistive vessels.