On the possibilities of modeling methods for the diagnosis and treatment of arterial hypertension using the APK "IMEDIS-EXPERT"

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For clarity of the material below, we will conduct a short excursion into the pathophysiological mechanisms of the development of this syndrome.

All arterial hypertension are divided into:

1) Hypertension (HD) or essential hypertension (80%).
At the same time, an increase in blood pressure is the main, sometimes even the only symptom of the disease.

2) Secondary or symptomatic hypertension (20%).

Hypertension occurs most often in highly developed countries and in people with increased psycho-emotional stress, which is direct evidence of the leading role of the central nervous system in the development of hypertension.

1. Prolonged psycho-emotional stress and negative emotions - leading predisposing factor of hypertension.

2. The factor of heredity is of great importance: the frequency the incidence of hypertension in hereditarily predisposed persons is 5–6 times higher. Recently, it has been proven that a violation of the deposition of catecholamines, in particular, norepinephrine, is responsible for heredity in hypertension, which, in turn, is associated with a violation of the corresponding enzymatic system.

3. Nutritional factor also plays an important role: increased table salt content, including in drinking water.

4. Prolonged nicotine intoxication.

5. Sedentary lifestyle, obesity.

6. Chronic alcohol poisoning also plays a role in etiology of hypertension.

With age, there is an increase in the incidence of HD, the peak occurs in the climacteric period. In this case, there are frequent sclerotic changes in the vessels with ischemia of the centers of the hypothalamus and dystrophic changes in them, which disrupts the normal regulation of blood circulation. Also, GB often occurs in persons with a history of brain injury, in this case, hypothalamic dysfunction is also evident. GB is more common in those who have had kidney disease. In acute kidney disease, damage and death of the renal interstitium is observed, the production of kinins and prostaglandins, the body's natural depressive systems, decreases.

The main hemodynamic factors are minute volume and total peripheral vascular resistance, which depends on arterioles.

The circulatory system includes the heart, blood vessels, and the central neuroregulatory apparatus of the circulatory system.

The minute volume depends on the strength and heart rate, the total peripheral vascular resistance depends on the tone of the arterioles. With an increase in tone, the venous return of blood to the heart sharply increases, which also affects its minute volume. With an increase in the work of the heart (running, excitement), the minute volume increases several times, but at the same time, the peripheral resistance decreases significantly, and the average

hemodynamic pressure remains unchanged. Currently, hemodynamic shifts in blood pressure are well known in hypertension:

1) In the initial stages, the minute volume or cardiac ejection, and the total peripheral resistance remains the same; hence the increase in blood pressure. This type of hemodynamic change is called hyperkinetic.

2) Subsequently, increasing the overall peripheral resistance, and cardiac output remains normal - eukinetic type.

3) Later, in a far advanced stage, there is a sharp increase in peripheral resistance against a background of decreased cardiac output. This type is called hypokinetic.

Thus, from the hemodynamic side, GB is heterogeneous and can be represented by three types.

According to Lang's theory, dysfunction of the cerebral cortex and centers of the hypothalamus are of primary importance. This theory, although based on clinical evidence, was largely hypothetical. In subsequent years, during the experiment, when the dorsal nucleus of the hypothalamus was irritated, systolic hypertension was caused, and when the central nucleus was irritated, diastolic hypertension. Irritation of the "emotional centers" of the cortex also led to a hypertensive reaction. Lang believed that hypertension is based on a kind of vascular neurosis - a violation of the reciprocal relationship of the cortex and subcortex, which, over time, necessarily leads to the activation of the sympathetic nervous system. Patients with hypertension are irritable, hyperreflex. With the advent of biochemical methods for the study of catecholamines, it was found that the exchange and excretion of catecholamines in the blood in patients with hypertension remain normal or slightly increased, and only later was the violation of their deposition proven. Sympathetic nerve endings are thickened with a norepinephrine depot. If the fiber is excited, the released norepinephrine stimulates alpha receptors, increasing the sympathetic activity of the corresponding system. Arterioles and venules are especially richly supplied with alpha receptors. The mechanism of inactivation normally consists of:

a) 10% is destroyed by the enzyme oxymethyltransferase; b) reverse transport across the membrane.

In pathology, the release of the mediator remains normal; if its deposition is disturbed, catecholamines act at the receptor level for a longer time and cause longer hypertensive reactions. The activity of the sympathetic nervous system increases, a longer exposure to catecholamines at the level of venules leads to an increase in venous return to the heart (venule spasm), the work of the heart increases, therefore, its minute volume also increases. Norepinephrine acts simultaneously on the alpha receptors of arterioles, thereby increasing the total peripheral resistance. The renal vessels are also richly supplied with alpha receptors, as a result of their spasm followed by ischemia of the kidney, the receptors of the juxtaglomerular apparatus are excited, the cells of which produce renin. The consequence of this is an increase in the level of renin in the blood. Renin itself is not hormonally active, but through the angiotensin systems it leads to:

1) An increase in the tone of arterioles (stronger and longer than norepinephrine).

2) An increase in the work of the heart (the amount of angiotensin II decreases with cardiogenic collapse).

3) Stimulating sympathetic activity.

4) Angiotensin II is one of the most powerful stimulants
release of aldosterone.

Further, the renin-aldosterone mechanism turns on, as it goes on, an even greater restructuring occurs: aldosterone enhances the reabsorption of sodium and water in the renal tubules, a passive intracellular increase in sodium and water content occurs. An intracellular increase in the content of sodium and water also occurs in the walls of blood vessels, as a result of which the vascular wall swells (swells), its lumen narrows and reactivity to vasoactive substances, in particular, to norepinephrine, increases, as a result of which vasospasm joins, which in the complex leads to a sharp increase in peripheral resistance. The activity increases, and the antidiuretic hormone is vigorously released, under the influence of which the reabsorption of sodium and water increases even more, the volume of circulating blood (BCC) increases, and the cardiac output increases.

Natural hypotensive (depressor) defense systems:

a) The system of baroreceptors (reacts to stretching with an increase in blood pressure) in the carotid sinus and in the aortic arch. With hypertension, the baroreceptors are rearranged to a new, higher critical level of blood pressure, at which they work, that is, their sensitivity to an increase in blood pressure decreases. An increase in the activity of antidiuretic hormone is also possibly associated with this.

b) The system of kinins and prostaglandins (especially prostaglandins "A" and "E", which are produced in the interstitial tissue of the kidneys). Normally, with an increase in blood pressure above the critical level, the production of kinins and prostaglandins increases and the baroreceptors of the aortic arch and carotid sinus are triggered, as a result of which the pressure quickly normalizes. With GB this defense mechanism is violated. The action of kinins and prostaglandins: increased renal blood flow, increased diuresis, increased sodium level. Hence, they are ideal saluretics. As the disease progresses, these defense systems are depleted, sodium is retained in the body, which ultimately leads to an increase in blood pressure.

So, in a brief form, the pathogenesis of GB is as follows: under the influence of prolonged psychoemotional stress in persons with burdened heredity, with increased activity of the hypothalamic centers, the tone of the sympathetic system increases, which is largely associated with a violation of the deposition of catecholamines, a violation
hemodynamics predominantly in the hyperkinetic type of blood circulation, labile arterial hypertension occurs due to the increased minute volume, then the disturbance of the water-salt balance becomes more and more important, the sodium content in the vascular wall increases, disturbances in the hypokinetic type of blood circulation appear. It is mainly peripheral resistance that suffers.

In addition to the generally accepted, there are two more theories of the etiopathogenesis of hypertension:

one) Mosaic theory Paige, according to which one etiopathogenetic factor cannot cause factors. GB, only the aggregate matters

2) The theory of membrane pathology: at the heart of GB is a violation permeability of cell membranes for sodium. There is an assumption that this type of membrane pathology is inherited.

Clinic

In the initial stages of the disease, the clinic is not pronounced, the patient may not know about an increase in blood pressure for a long time. However, already during this period, there are nonspecific complaints expressed to one degree or another, such as: rapid fatigue, irritability, decreased performance, weakness, insomnia, dizziness, etc. And it is with these complaints that the patient most often goes to the doctor for the first time.

a) Headaches: most often occipital and temporal localization; "heavy head" in the morning or at the end of the working day. Pain is usually worse when lying down and is better after walking. Usually such pains are associated with changes in the tone of arterioles and veins. The pain is often accompanied by dizziness and tinnitus.

b) Pain in the heart area: since an increase in blood pressure is associated with an increase in the work of the heart (to overcome the increased resistance), myocardial hypertrophy occurs compensatory. As a result of hypertrophy, there is a dissociation between the needs and capabilities of the myocardium, which is clinically manifested by IHD as angina pectoris. This is often observed with GB in old age. In addition to angina pectoris, pain in the heart can be of the type of cardialgia - prolonged dull pain in the apex of the heart.

c) Flashing flies in front of the eyes, shroud, flashing lightning and other photomies. Their origin is associated with a spasm of retinal arterioles. With malignant GB, retinal hemorrhages may occur, which leads to complete loss of vision.

d) GB is a kind of vascular neurosis. There are symptoms of a violation of the central nervous system, which can, for example, manifest as a pseudoneurotic syndrome - rapid fatigue, decreased performance, weakened memory, symptoms of irritability, weakness, affective lability, the predominance of anxiety moods and hypochondriacal fears are noted, sometimes they can acquire, especially after crises, a phobic character ... Often the above phenomena manifest themselves when the blood pressure level changes, but they are far from all patients - many do not experience any unpleasant sensations at all and arterial hypertension is detected by chance.

Blood pressure measurement technique: Use the Korotkoff method.

Objectively:

1) Increase in blood pressure.

2) Signs of left ventricular hypertrophy: increased apical push, accent II tone on the aorta.

3) Intense pulse, in patients with hyperkinetic type - tachycardia, in elderly patients more often bradycardia.

It is necessary to determine the pulse and pressure on the four limbs. Normally, the pressure on the legs is higher than on the hands, but the difference is no more than 15-2O mm Hg. The same pattern is observed with hypertension, since

the caliber of the vessels on the legs is higher.

Additional research methods

1) Signs of left ventricular hypertrophy: a)
according to ECG data;
b) X-ray: a rounded apex of the heart, an increase in the left ventricular arch.

2) Ophthalmological examination: the state of arterioles and venules fundus - this is the only opportunity to see blood vessels, the "calling card" of hypertensive patients. There are 3 (with us) or 4 stages of fundus changes:

1) Hypertensive angiopathy: the tone of the arterioles is sharply increased, the lumen narrowed (symptom of "wire"), venule tone is reduced, lumen is increased. According to Case, an additional 2 more substages are distinguished:

a) the changes are not pronounced;

b) the changes are the same, but sharply expressed.

2) Hypertensive angioretinopathy: degenerative changes in retina + retinal hemorrhage.

3) Hypertensive neuroretinopathy: in the pathological process the papilla of the optic nerve is involved (edema + degenerative changes).

It is customary to distinguish two forms of GB flow:

1. Slow course, gradual development of pathological processes, the disease is relatively benign, the symptoms increase gradually, over 20-30 years. Most often you have to deal with just such patients.

2. In some cases, there is a malignant course of GB. By According to various sources, the malignant course is 0.25-0.5%. At the same time, they find a high activity of the renin-angiotensin system + a high content of aldosterone in the blood serum. The high activity of aldosterone leads to a rapid accumulation of sodium and water in the vascular wall, and hyalinosis occurs quickly. Hence, the criteria for the malignancy of this form of hypertension follow: blood pressure, manifesting itself as high (more than 160 mm Hg), remains at a high level, without a tendency to decrease; ineffectiveness of antihypertensive therapy; neuroretinopathy; severe vascular complications: early strokes, myocardial infarction, renal failure; rapid progressive course, death from renal failure or stroke in 1.5–2 years.

Classification of GB according to E.I. Tareev

1) cerebral,
2) cardiac,
3) renal.

Classification by stages and phases A.L. Myasnikov, adopted by the All-Union Conference of Physicians in 1951:

Stage I. BP is labile and rises in certain situations.

"A" - blood pressure rises only in extreme stressful situations. These are hyperreactors, they are practically healthy, but the threat of disease is increased.

"B" - blood pressure also rises in normal situations: by the end of the working day, with normal physical activity; but during rest it normalizes on its own.

Stage II. Arterial hypertension takes on a permanent character, rest for the normalization of blood pressure is no longer enough.

"A" - blood pressure is almost always increased, but nevertheless there may be a spontaneous normalization of blood pressure during prolonged rest. At this stage, crises are possible, subjective sensations appear, organic changes appear: left ventricular hypertrophy, angioretinopathy.

"B" - persistent increase in blood pressure, stabilization occurred. Spontaneous normalization of blood pressure is impossible; antihypertensive therapy is required to lower blood pressure. There is significant left ventricular hypertrophy and hypertensive angioretinopathy, changes in internal organs, often of the type of dystrophy, but without impairing their functions.

Stage III. In addition to an increase in blood pressure, there are symptoms of circulatory disorders of internal organs, myocardial infarction, cerebrovascular accident, severe visual impairment, nephrosclerosis.

"A" - despite organic changes within severe functional new organs, no disorders, the patient maybe save ability to work.

"B" - the function of the patient suffering from organ, happens disability is sharply impaired.

In 1972, a parallelism was found between the clinical manifestations of hypertension and the level of renin in plasma; based on this, it was suggested to divide GB by:

1) normorenin,

2) hyporenin,

3) hyperrenic.

But in practice it turned out that there is not always a parallelism between plasma renin activity and blood pressure level.

According to the characteristics of hemodynamics, GB is divided into the following forms:

1) hyperkinetic,

2) eukinetic,

3) hypokinetic.

Classification by blood pressure level is also common:

Stage I. Borderline hypertension. 140-160 / 90-95 mm Hg

Stage II. Labile hypertension, blood pressure fluctuates within different limits, periodically normalizes on its own.

Stage III. Stable arterial hypertension, blood pressure is constantly kept at a high level.

Complications of GB:

1. Hypertensive crisis occurs with a sudden sharp increase in blood pressure with the obligatory presence of severe subjective disorders. There are two types of crises:

a) Adrenaline - associated with the release of adrenaline into the bloodstream, characterized by a sharp increase in blood pressure, lasting several hours, sometimes minutes. More typical for the early stages of GB. Clinically it is usually manifested by tremors, palpitations, headache. The increase in blood pressure is usually small.

b) Norepinephrine - occurs mainly in the late stages of hypertension, lasts from several hours to several days; Blood pressure increases more slowly, but reaches high values. Characterized by a bright clinic: autonomic disorders, visual impairment, severe headache.

This type of crisis is sometimes called hypertensive encephalopathy.

Hypertensive crises are often provoked by:

1) Changes in meteorological conditions.

2) Changes in the function of the endocrine glands.

3) However, most often the crisis is associated with psycho-emotional trauma. Characterized by severe headache, dizziness, nausea, vomiting, sometimes loss of consciousness, visual impairment, up to short-term transient blindness: mental disorders, weakness. Manifestations from the central nervous system are caused by cerebral edema, the pathogenesis of which is as follows: spasm of cerebral vessels impaired permeability leakage of blood plasma into the medulla swelling of the brain. There may be focal disorders of cerebral hemopoiesis, leading to hemiparesis. In the initial stage of the disease, crises, as a rule, are short-lived, occur more easily. During a crisis, cerebral circulation disorders of a dynamic nature with transient focal symptoms, hemorrhage in the retina and its detachment, cerebral stroke, acute pulmonary edema, cardiac asthma and acute left ventricular failure, angina pectoris, myocardial infarction, coronary artery disease with all clinical manifestations can develop. GB is one of the main risk factors for the development of coronary artery disease.

Visual impairment is associated with the development of angio- and retinopathy, with retinal hemorrhage, its detachment with central artery thrombosis.

In violation of cerebral circulation, the mechanisms are different, most often the formation of a microaneurysm with subsequent rupture, that is, according to the type of hemorrhagic stroke, when cerebral vascular thrombosis or ischemic stroke; the outcome is paralysis and paresis. development of renal Nephrosclerosis with failure is a relatively rare complication GB, more often - at malignant form currents. Delaminating aneurysm aorta. Subarachnoid hemorrhage.

Differential diagnosis

GB diagnosis should be placed only when excluding secondary symptomatic hypertension, but this is often very difficult. Persons with secondary hypertension make up about 10%, and in the age group under 35 years old - 25%. Secondary hypertension is divided into:

1) Hypertension of renal origin; are the most common.

2) Arterial hypertension of endocrine origin.

3) Hemodynamic arterial hypertension.

4) Hypertension with brain damage (the so-called centrogenic hypertension).

5) Others: medication, with polyneuritis, etc.

I. Hypertension of renal origin

a) With chronic diffuse glomerulonephritis; the history often indicates renal pathology, from the very beginning there are at least minimal changes on the part of urine - slight hematuria, proteinuria, cylindruria. With GB, such changes occur only in advanced stages. BP is stable, may not be particularly high, crises are rare. Kidney biopsy helps.

b) In chronic pyelonephritis: a bacterial disease, there are signs of infection. Dysuric disorders. A history of acute inflammation with chills, fever, back pain, and sometimes renal colic. With pyelonephritis, the concentration function of the kidneys suffers (but only with a 2-sided lesion), early thirst and polyuria occur. Often, the beating syndrome on the lower back is positive. In the analysis of urine leukocyturia, mild to moderate proteinuria. Nechiporenko's test - the number of leukocytes in 1 ml of urine; normally - up to 4000. Urine culture is of certain importance - a large number of colonies are revealed. Bacteriuria may occur. It is necessary to sow urine several times, because outside exacerbation, the number of colonies may be small, but they are constant (a sign of the constancy of colonies). When setting Zimnitsky's sample: hypo and isostenuria. Sometimes, when bacteriuria is detected, they resort to provocative tests: pyrogenaclovy or a test with iv prednisolone, then the Nechiporenko test is performed. With pyelonephritis, there is a latent leukocyturia. Pyelonephritis, even 2-sided, is always asymmetrical, which is detected by radioisotope renography (separate kidney function is determined). The main diagnostic method is excretory urography, while the deformation of the calyx-pelvic apparatus is determined, and not just a dysfunction.

c) Polycystic kidney can also cause an increase in blood pressure. This is a congenital disease, therefore, it is often an indication of the family nature of the pathology. Polycystic disease often proceeds with an increase in the size of the kidneys, which are clearly palpable, the concentration function of the kidneys, early thirst and polyuria are early impaired. When diagnosing, the method of excretory urography helps.

Renovascular hypertension. It is associated with damage to the renal arteries, narrowing of their lumen. Causes: in men it is often as an age-related atherosclerotic process, in women it is more often of the type of fibromuscular dysplasia - a kind of isolated lesion of the renal arteries of unclear etiology. Often occurs in young women after pregnancy. Sometimes the cause is thrombosis or thromboembolism of the renal arteries (after surgery, with atherosclerosis).

Pathogenesis: as a result of narrowing of the lumen, microcirculation decreases, the renin-angiotensin system is activated, and the aldosterone mechanism is activated again.

Signs: rapidly progressing high stable hypertension, often with a malignant course (high renin activity): vascular murmur over the projection of the renal artery: on the anterior abdominal wall just above the navel, in the lumbar region. Noise is heard better on an empty stomach.

Additional research methods

The function of the ischemic kidney suffers, the other kidney compensatory increases in size. Therefore, an informative method for a separate study of the kidneys is radioisotope renography, in which the vascular part of the segment is reduced, the curve is stretched + asymmetry.

Excretory urography - the contrast agent enters the ischemic kidney more slowly (slowing down in the first minutes of the study) and is excreted more slowly (in the last minutes of the contrast agent delay). Described as late arrival and hyperconcentration in late

terms, that is, asynchronism of contrasting takes place - a sign of asymmetry.

When scanning a diseased kidney due to wrinkling, it is reduced in size and poorly outlined, a healthy kidney is compensatory enlarged.

Aortography is the most informative method, but, unfortunately, it is not safe, therefore it is used by the latter.

The plastic of the vessel leads to a complete cure. But early surgery is important, before the onset of irreversible changes in the kidney. It must also be remembered that there is functional stenosis.

Nephroptosis occurs due to the pathological mobility of the kidney. The pathogenesis of hypertension consists of 3 points: tension and narrowing of the renal artery ischemia of the kidney vasospasm hypertension; violation of the outflow of urine along a bent, sometimes twisted, curved ureter, accession of infection pyelonephritis, irritation sympathetic nerve in the vascular pedicle spasm.

Signs: more often at a young age, hypertension with crises, severe headaches, severe autonomic disorders, but generally labile hypertension; in the supine position, blood pressure decreases. For diagnosis, aortography and excretory urography are mainly used. Surgical treatment: fixation of the kidney. Other hypertension of renal genesis: with amyloidosis, hypernephron, diabetic glomerulosclerosis.

II Arterial hypertension of endocrine genesis

a) Itsenko-Cushing's syndrome is associated with lesions of the adrenal cortex, the production of glucocorticoids increases sharply. The typical appearance of patients is characteristic: a moon-shaped face, redistribution of adipose tissue.

b) Pheochromocytoma: this is a tumor of mature cells of the chromophinic tissue of the medulla, less often a tumor of the paraganglia of the aorta, sympathetic nerve nodes and plexuses. Chromophinic tissue produces adrenaline and norepinephrine. Usually, with pheochromocytoma, catecholamines are periodically released into the bloodstream, which is associated with the occurrence of catecholamine crises. Clinically, pheochromocytoma can occur in two ways:

1. Crisis arterial hypertension.

2. Persistent arterial hypertension. Blood pressure rises suddenly, in within a few minutes over 300 mm Hg. It is accompanied by pronounced vegetative manifestations of the "storm": palpitations, tremors, sweating, fear, anxiety, skin manifestations. Catecholamines actively interfere with carbohydrate metabolism - blood sugar rises, therefore, during a crisis, thirst is observed, and after polyuria. There is also a tendency to orthostatic fall in blood pressure, which is manifested by loss of consciousness when trying to change the horizontal position to vertical (hypotension in orthostasis). With pheochromocytoma, a decrease in body weight is also observed, which is associated with an increase in basal metabolism.

Diagnostics

1) hyperglycemia and leukocytosis during a crisis; develop early hypertrophy and dilation of the left ventricle may be tachycardia, fundus changes; the main diagnostic method: determination of catecholamines and their metabolic products; vanilmandelic acid, with pheochromocytoma it

the content exceeds 3.5 mg / day, the content of adrenaline and norepinephrine exceeds 100 mg / day. in urine;

2) test with alpha-blockers: phentolamine (regitin) 0.5% - 1 ml IV or i / m or tropafen 1% - 1 ml i / v or i / m. These drugs have antiadrenergic effects, block the transmission of adrenergic vasoconstrictor impulses. Reduction of systolic pressure by more than 80 mm Hg, and diastolic pressure by 60 mm Hg. after 1-1.5 minutes. after administration of the drug indicates the sympathetic-adrenal nature of hypertension, and the test for pheochromocytoma is considered positive. The same drugs (phentolamine and tropafen) are used to relieve catecholamine crises.

3) provocative test: intravenously injected histamine dihydrochloride 0.1%, 25-0.5 ml (histamine is produced in 0.1% - 1 ml). Pheochromocytoma is characterized by an increase in blood pressure by 40/25 mm Hg. and more after 1–5 minutes. after injection. The test is indicated only if the blood pressure without seizures does not exceed 170/110 mm Hg. At higher pressures, test with phentolamine or tropafen only. In about 10% of cases, a histamine test may be positive even in the absence of pheochromocytoma. The mechanism of action of histamine is based on reflex stimulation of the adrenal medulla.

4) resacral oxysuprarenography (into the perirenal space oxygen is injected and a series of tomograms is done).

5) pressure on palpation in the kidney area can lead to release of catecholamines from the tumor into the blood and be accompanied by an increase in blood pressure.

6) also helps the study of the vessels of the fundus and ECG.

c) Cohn's syndrome or primary hyperaldosteronism. This disease is associated with the presence of adenoma or benign tumor, less often - carcinoma, as well as with 2-sided hyperplasia of the glomerular adrenal cortex, where aldosterone is produced. The disease is associated with an increased intake of aldosterone into the body, which enhances the tubular reabsorption of sodium, as a result of which intracellular potassium is replaced by sodium, with the distribution of potassium and sodium leads to the accumulation of sodium, and then water intracellularly, including in the vascular wall, which narrows the lumen of blood vessels and leads to an increase in blood pressure. An increase in sodium and water content in the vascular wall leads to an increase in sensitivity to humoral pressor substances, which results in diastolic arterial hypertension.

The second group of symptoms is associated with excessive excretion of potassium from the body, therefore, in the clinical picture there will be signs of severe hypokalemia, manifested primarily by muscle disorders: muscle weakness, weakness, parasthesia, there may be paresis and even functional muscle paralysis, as well as changes in the cardiovascular system : tachycardia, extrasystole and other rhythm disturbances. On the ECG of lengthening electrical systole, an increase in the ST interval, sometimes a pathological U wave appears. Cohn's syndrome is also called "dry hyperaldosteronism", because with it there is no visible edema.

Diagnostics

1) blood test for potassium and sodium content: potassium concentration falls below 3.5 mmol / l, sodium concentration increases over 130 mmol / l, potassium in urine is increased, and sodium is low; -increased urine catecholamines (see above); urine reaction is usually neutral or alkaline; a test with hypothiazide is of certain importance: first, the potassium content in the blood serum is determined, then the patient receives hypothiazide at 100 mg / day. within 3-5 days. Further, blood potassium is re-examined, - in patients with Cohn's syndrome, there is a sharp drop in the concentration of potassium, in contrast to healthy ones;

2) test with veroshpiron - aldosterone antagonist, which appoint 400 mg / day. This leads to a decrease in blood pressure after a week, and the potassium in the blood rises;

3) determination of aldosterone in urine (the technique is not clearly established);

4) determination of renin, with primary hyperaldosteronism activity the juxtaglomerular apparatus of the kidneys is sharply depressed, little renin is produced;

5) X-ray: tomography of the adrenal glands, but only a tumor weighing more than 2 g; if the tumor is small: diagnostic laparotomy with revision of the adrenal glands.

If the disease is on time not diagnosed, joins kidney disease - nephrosclerosis, pyelonephritis. Thirst and polyuria appear.

d) Acromegaly. Blood pressure rises due to the activation of the function of the adrenal cortex.

e) Kimmelsteel-Wilson syndrome: diabetic glomerulosclerosis in diabetes mellitus.

f) Thyrotoxicosis: there is an increased excretion of calcium through the kidneys, which contributes to the formation of stones and ultimately leads to increase in blood pressure.

g) Hyperreninoma - swelling of the juxtaglomerular apparatus - but this, rather, casuistry.

h) Contraceptive arterial hypertension, at application hormonal contraceptive drugs.

III. Hemodynamic arterial hypertensionassociated with primary defeat of large great vessels.

a) Coarctation of the aorta is a congenital disease associated with thickening of the muscle layer in the area of the aortic isthmus. There is a redistribution of blood, - the vessels are sharply overfilled with blood before or above the constriction, i.e. vessels of the upper half of the body; the vessels of the lower extremities, on the contrary, receive little and slow blood supply. The main symptoms of the disease appear by puberty, usually by the age of 18. Subjectively, headaches, a feeling of heat or flushing to the head, nosebleeds are noted.

Objectively: disproportion; powerful upper half of the body and poorly developed lower; hyperemic face; the pulse on the radial artery is full, tense; cold feet, weakened pulse on the legs; to the left of the sternum, a rough systolic murmur; the apical impulse is sharply increased; BP on the brachial artery is high, on the legs - low; rib patterns on the radiograph; the main diagnostic method is aortography.

With timely diagnosis, treatment leads to complete recovery. If left untreated, nephrosclerosis develops after about 30 years.

b) Pulseless disease, or Takayashi's syndrome. Synonyms: panaortitis, panarteritis of the aorta and its branches, aortic arch disease. The disease is of an infectious allergic nature, most often occurs in young women. There is a proliferative inflammation of the walls of the aorta, mostly intima, as a result of necrosis plaques are formed, fibrinoid swelling occurs. History of prolonged subfibrillation, reminiscent of
feverish condition, and allergic reactions.

An ischemic syndrome appears in the vessels of the limbs and the brain, which is manifested by fainting, dizziness, loss of vision, short-term loss of consciousness, weakness in the arms. Arterial hypertension is detected as a result of blood redistribution. This disease is also called “reverse coarctation”. On the hands, the pressure is lowered, and asymmetrically, and on the legs, the pressure is greater. Further, vasorenal or ischemic hypertension joins, which is of a malignant nature. A renal shunt appears.

Diagnostics: the use of aortography method is mandatory, ESR is often increased, a high content of gamma globulin, a test with an aortic antigen (UAE) is proposed.

IV. Centrogenic arterial hypertension associated with defeat
brain - encephalitis, tumors, hemorrhages, ischemia, skull injuries, etc. In cerebral ischemia, hypertension is obviously compensatory in nature and is aimed at improving the blood supply to the brain. In the development of hypertension with organic lesions of the brain, damage and functional changes in hypothalamic structures are of undoubted importance, which is accompanied by a violation of the central nervous regulation of blood pressure.

V. Medicinal arterial hypertension:

a) When using adrenergic drugs: ephedrine, adrenaline. b) With long-term treatment with hormonal agents
(glucocorticoids).

c) When using drugs that have a damaging effect on the kidneys (phenacetin).

Hypertension treatment

Diet: limiting table salt, it is useful to reduce weight if you are overweight. Patients are assigned a diet number 10.

Mode: transfer to one-shift work; labor regulations - exclude night shifts, etc .; improvement and rationalization of working conditions; rest mode (good sleep, rest after work); fight against hypodynamia -
move more.

General principles of hypertension treatment:

a) Exactly establish the nature of arterial hypertension.

b) In some cases, hypertension may be asymptomatic. leak

c) All patients with arterial hypertension, regardless of the presence

symptoms, antihypertensive therapy is indicated. With a decrease in blood pressure during treatment, the state of health can sometimes worsen, therefore it is important to choose the correct rate of pressure decrease, taking into account the patient's age, the duration of arterial hypertension, the presence or absence of vascular disorders. In the absence of vascular complications, at a young age, blood pressure is reduced to normal levels quickly. In old age, the reduction is carried out to a subnormal level, that is, to the danger zone.

d) Therapy should be carried out from the point of view of the pathogenesis of the disease. Given the need for pathogenetic treatment, therapy should be complex or combined, since it is necessary to influence the various links of pathogenesis.

For clarity of the proposed research algorithm and the resulting treatment tactics, we offer the following case history.

Patient VS, 59 years old, came to us on 20.02.2005 about obesity, high blood pressure, headaches, most often occipital and temporal localization; in the morning or at the end of the working day "heavy head". Pain is usually worse when lying down and is better after walking. HELL 210/140, which during the day, according to the patient, fluctuated up to 230 and 90 units.

Since the tone of the arteries and arterioles is associated with the sympathetic division of the ANS, we will be interested, first of all, in the reason for the increase in sympathetic tone, which in patients with hypertension should always be of several degrees, which can be absolute or relative.

To do this, we test the organopreparation Aorta in potencies higher than D6, i.e. in potencies corresponding to hyperfunction.

The patient was tested

Aorta D10, D12, D15 **+** anabolism of 1 degree + alkalinity of 1 degree + VNS voltage 1, 2, 3 degrees **+** ANS department (sympathicus) D3, D4, D5 **.**

If we tested the depletion of the ANS in the parasympathetic division, this would mean a relative increase in sympathetic tone and the presence of metabolic disorders in the arteries themselves.

In this chain 2, 3, we turn on the VNS voltage degree in inversion and write it down, in transfer mode, in the 1st container for 2–3 grains.

We transfer the resulting drug to the second glass and turn on the BRT, in the drug testing mode, without connecting the electrodes.

Next, we must find an organ that can provide us with such a condition on the aorta, which we ordered, and find out how, i.e. in what potency and with what metabolic shifts, it should work.

Aorta D10, D12, D15 **+** anabolism of 1 degree + alkalinity of 1 degree + VNS voltage 1, (2, 3 in inversion) degree **+** ANS department (sympathicus) D3, D4, D5 in the second container in the MT + organopreparations mode.

The patient was tested

liver D10, D12, D15 **+** catabolism of 1, 2 degrees + alkalinity of 1 degree + tension in the parasympathetic department in D5,

that is, it becomes clear that the increased tone of the aorta is associated with liver function, and in order to relieve the increased tone of the aorta, the liver must work in hyperfunction and destroy something at the same time.

We leave the entire identified chain turned on and add the address at the end, i.e.
liver D10, D12, D15 + catabolism 1.2 degree + 1 degree alkalinity
+ tension in the parasympathetic division in D5 + Aorta D10, D12, D15

We turn off the MT and under load conditions with this complex, in the BRT mode, we determine the meridians, during testing of which the measurement level will decrease.

We carry out therapy along the identified meridians under load conditions with this complex until the measuring level recovers to the initial high value.

Then we record in the first container for 2-3 minutes. Determine the dose, which was 8 balls.

This will be BR-drug # 1.

Since a broken chain appeared on the liver, i.e. the body sacrifices some function of the liver, which, for sure, was inadequate; in order to solve the problem, we need to find an organ that would ensure the normal functioning of the liver itself.

For this
liver D10, D12, D15 + (catabolism 1, 2 degrees inverted) + 1 degree alkalinity + tension in the parasympathetic division in D5 ,
those. we do not want catabolism in these potencies.

In this chain, we turn on catabolism of 1, 2 degrees in inversion and write it down, in transfer mode, in the 1st container for 2-3 grains.

We transfer the resulting drug to the second container and turn on the BRT, in the drug testing mode, without connecting the electrodes.

Next, we must find an organ that can provide us with such a condition on the liver, which we ordered, and find out how, i.e. in what potency and with what metabolic shifts it should work.

The patient was tested
Small intestinal mucosa D10 + catabolism 1.2 degree + 1 degree alkalinity + voltage VNS 1 + sympathicus D5 ,
those. it becomes clear that inadequate catabolism in the liver is associated with inadequate functioning of the small intestine, and in order to remove inadequate catabolism in the liver, the mucous membrane of the small intestine must work in hyperfunction and destroy something at the same time.

We leave the entire identified chain turned on and add the address at the end, i.e.
Small intestinal mucosa D10 + catabolism 1, 2 degrees +
1 degree alkalinity + voltage VNS 1 + sympathicus D5 + liver D10, D12, D15.

We turn off the MT and under load conditions with this complex, in the BRT mode, we determine the meridians, during testing of which the measurement level will decrease.

We carry out therapy along the identified meridians under load conditions with this complex until the measuring level recovers to the initial high value.

Then we record in the first container for 2-3 minutes. Determine the dose, which was 8 balls.

This will be BR-drug # 2.

Since two levels of catabolism began to be determined in the intestine, it is necessary to correct this.

For this

Small intestinal mucosa D10 **+** catabolism 1, (2 tbsp. in inversion) degree **+** silkiness 1 degree **+** voltage VNS 1 + sympathicus D5

In this chain, we turn on the catabolism of the 2nd degree in inversions and write it down, in the transver mode, in the 1st container for 2-3 grains.

We transfer the resulting drug to the second container and turn on the BRT, in the drug testing mode, without connecting the electrodes.

Next, we must find an organ that can provide us with such a state on the mucous membrane of the small intestine, which we ordered, and find out how, i.e. in what potency and with what metabolic shifts, it should work.

The patient was tested

Pancreas D10 **+** anabolism 1 degree **+** 1 degree alkalinity + VNS voltages 1, 2 degrees + vagus D5 **,**

those. it becomes clear that inadequate catabolism on the mucous membrane of the small intestine is associated with inadequate functioning of the pancreas.

We leave the entire identified chain turned on and add the address at the end, i.e.

Pancreas D10 **+** anabolism 1 degree **+** silkiness 1 degree **+** voltage VNS 1 degree + vagus D5 **+** Small intestinal mucosa D10.

We turn off the MT and under load conditions with this complex, in the BRT mode, we determine the meridians, during testing of which the measurement level will decrease.

We carry out therapy along the identified meridians under load conditions with this complex until the measuring level recovers to the initial high value.

Then we record in the first container for 2-3 minutes. Determine the dose, which was 8 balls.

This will be BP drug # 3.

The drugs were prescribed for admission strictly according to the numbers №1, №2, №3. those. # 3 will work if # 2 is accepted, and # 2 if # 1 is accepted.

Two weeks later, when re-visiting blood pressure 150/90 without hesitation during the day, the patient lost 5 kg, although she complained of increased appetite.

Of the preparations, only No. 3 worked, which was left for further use.

Over the past 2 years, the patient did not seek medical help anywhere and did not take any medications, her condition is stable.