

**MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN
REPUBLICAN CENTER FOR MEDICAL EDUCATION DEVELOPMENT
TASHKENT MEDICAL ACADEMY**

**ARTERIAL HYPERTENSION.
DIAGNOSIS AND SURGICAL TREATMENT**

Tashkent - 2018

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«APPROVED»

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Ministry of Health of the Republic of Uzbekistan

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«AGREED»

Director of the Republican Center for
the Development of Medical
Education

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« _____ » _____ 2018 year

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**ARTERIAL HYPERTENSION.
DIAGNOSIS AND SURGICAL TREATMENT**

Teaching-methodical manual for students of 4-5 courses of medical universities

Tashkent – 2018

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The present educational-methodical manual is intended for students of higher medical educational institutions who can get acquainted with the algorithm of diagnostics by arterial hypertension. The knowledge obtained during the lesson will be used for the passage of surgery, neurology, therapy, X-ray radiology and other disciplines.

RATIONALE FOR THEME

Arterial hypertension (AH) - a heterogeneous metabolic syndrome characterized by elevated blood pressure at 140/90 mm Hg and above. The prevalence of hypertension depends on what numbers elevated blood pressure is considered to be at the moment. With the above criteria about 20-30% of the adult population of the globe is hypertensive.

Hypertension is a risk factor for many cardiovascular diseases, especially coronary heart disease, stroke and chronic brain insufficiency, as many authors consider, is badly treated. Hypertensive patients who did not receive treatment die from HD or cardiac failure in 50% of cases, from stroke - in 33%, kidney failure - in 15%.

Problems of AH have not only medical, but also socio-economic importance, so the researchers are faced with the task of developing practical recommendations for the prevention, diagnosis and treatment of this disease. Clinical institutes of many countries work in this direction, but the results are still far from the goal (Alekyan B.G. 2006).

According to the prospective study on multifactor prophylaxis (MRFIT), the risk of cardiovascular complications increases steadily with both SBP and DBP. So, if the risk of developing an ischemic heart disease at a normal blood pressure level is taken as 1, then with an isolated increase in DBP greater than 100 mmHg, a similar risk is 3.32; with an isolated increase in SBP more than 160 mmHg - 4.19; and with a combined increase in SBP and DBP - 4.57. According to the same study, an increase in SBP by 10 mmHg above the baseline level increases the risk of renal complications by 1.65 times. A prospective observation, conducted in the American city of Framingham for 34 years, showed that in people with a high level of AD, the risk of developing chronic heart failure is 2-4 times higher than in those with low blood pressure (Pokrovsky A.V.2004).

Recently, the incidence of AH complications has increased, especially cerebral strokes, myocardial infarction and renal insufficiency. Thus, in Tashkent, among men aged 40-59 years, the prevalence of hypertension was 26.6%; Among the unorganized rural population of the Tashkent region, AH was detected in 14.4%. With age, it tends to grow, reaching 24.5% by the age of 50-59 (Kurbanov R.D. 2016).

Widespread prevalence, hypertension, a high percentage of its complications, and injury to people of working age necessitate the search for new ways of diagnosis, treatment and prevention.

THE PURPOSE OF PRACTICAL SESSION

To strengthen and expand the knowledge of students on the diagnosis of patients with hypertension. To draw the attention of students to the importance of early diagnosis of the disease, the use of special laboratory and instrumental methods of research.

PRIVATE DIDACTIC OBJECTIVES

1. Learn to recognize the clinical symptoms of hypertension.
2. To master methods of diagnosis and differential diagnosis of patients with AH.
3. To acquire students certain practical skills in the examination of patients with AH.
4. To teach students special methods of examining patients with this pathology.

METHOD OF CONDUCTING THE SESSION

The teacher introduces students to the purpose of the lesson, then monitors the initial level of knowledge of the stands with the help of one interactive teaching methods on etiology, pathogenesis, clinic, diagnosis, differential diagnosis, treatment of patients with AH. In the wards, students get acquainted and disassemble patients with hypertension, then report on them with a review of the clinical course, diagnosis, methods of examination, treatment. At the conclusion of the lesson, the students control the final mastery of knowledge.

GENERAL EDUCATIONAL-METHODICAL INSTRUCTION

The task of general practitioners (GPs), therapists, cardiologists and angio-surgeons timely diagnose AH, in order to prevent the development of serious complications such as heart diseases, heart failure, chronic renal insufficiency and strokes.

Arterial pressure (AD) is a rather labile quantity. The results of BP monitoring indicate that in healthy individuals, it changes significantly during the day. Maximal values of blood pressure are recorded during the day, especially during emotional or physical stress. At night, the lowest values of blood pressure are noted. The magnitude of blood pressure largely determines the adequacy of blood supply to organs and tissues. Its value, in turn, depend on the amount of blood incoming from the heart into the aorta per minute (cardiac output), and resistance exerted by the blood flow in the arterial vessels, especially in arterioles and small arteries (total peripheral vascular resistance). A significant influence on the level of blood pressure is the mass of circulating blood, taking part in the formation of cardiac output values (Figure 1). Changes in any of these parameters in the absence of adaptive changes in the other two naturally lead to changes in blood pressure. Under physiological conditions, all three parameters are closely interrelated, which determines the relative constancy of blood pressure. Relative stability of blood pressure in rest and its regular changes under various loads testify to the presence of quite complex regulation of blood pressure.



Figure 1. Determinants of blood pressure.

CO - cardiac output;

TPR - total peripheral resistance;

VCB - volume of circulating blood.

Physiology of blood pressure regulation.

All factors relevant to the regulation of blood pressure, can affect the level of resistance to blood flow through changes in cardiac output or blood volume.

Often, changes in the activity of one or another of the regulatory factors may have an impact on all three parameters that affect the blood pressure. In turn, the activity of all the factors regulating blood circulation is closely linked to the principles of both direct and feedback. In a complex system of regulation of blood flow and, therefore, blood pressure, there are several basic parts, the most significant of which are: the nervous system, hormones and biologically active substances, as well as the kidneys.

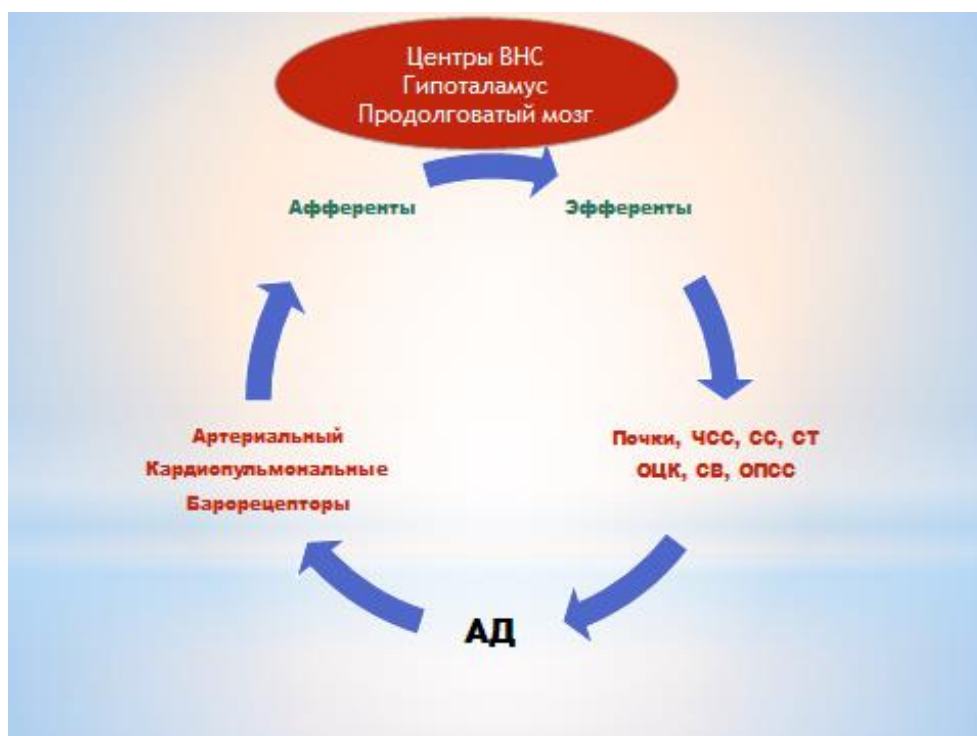


Figure 2. Neurogenic regulation of blood pressure.

NHB - the number of heart beats, CC - cardiac contractility, VT - vascular tone, BV - blood volume, CO - cardiac output, TPVR - total peripheral vascular resistance.

Multilateral nervous system influence on blood circulation determines its part in ensuring not only the rapidly advancing adaptive hemodynamic changes, but in the long-term control of blood pressure (Table 1).

Table 1. Path nervous system influence on the cardiovascular system

The nature of the impact	Effects
Direct	Heart: <ul style="list-style-type: none"> • changes in heart rate • changes in contractile activity Vessels: <ul style="list-style-type: none"> • Changing the tone of the smooth muscle cells of arteries and veins
Indirect	Change in the rate of synthesis and release of renin *, <ul style="list-style-type: none"> * Prostaglandins, * Kinins, * Vasopressin * ACTH * Hormones and other biologically active substances. Changing the speed of the water and sodium reabsorption in the kidney.

Kidneys occupy a central place in the regulation of blood pressure.

To date, the most thoroughly studied the role of renin-angiotensin system in the regulation of blood pressure. The main active compound of this system is angiotensin-II, which is formed of a renin substrate (angiotensinogen) from exposure to a number of enzymes. Renin-substrate is synthesized primarily in the liver, where it enters the blood. In the blood under the influence of renin forms renin-I, which is emitted under the influence of the endothelium converting enzyme converted into angiotensin-II - one of the most potent pressor compounds.

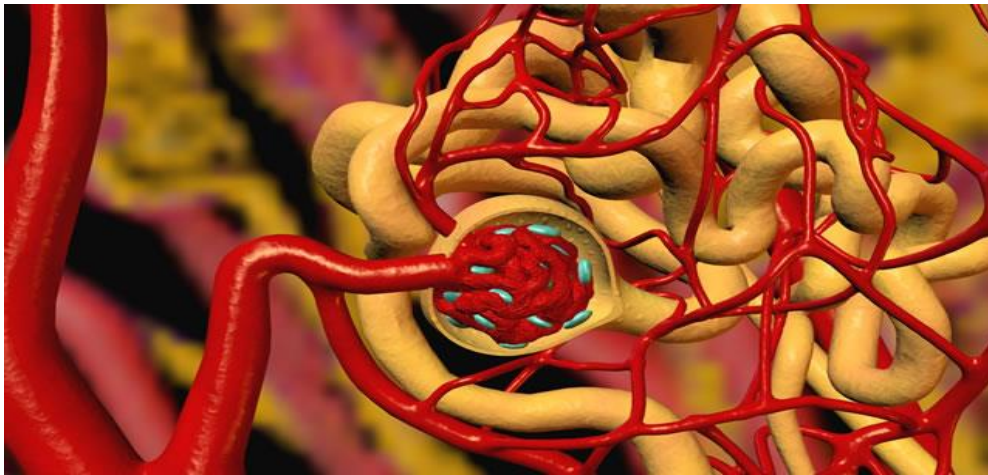


Figure 3. Glomerular unit

Thus the formation of angiotensin-II from angiotensin-I occurs with the participation of serine proteases, called chymase (Fig.4).

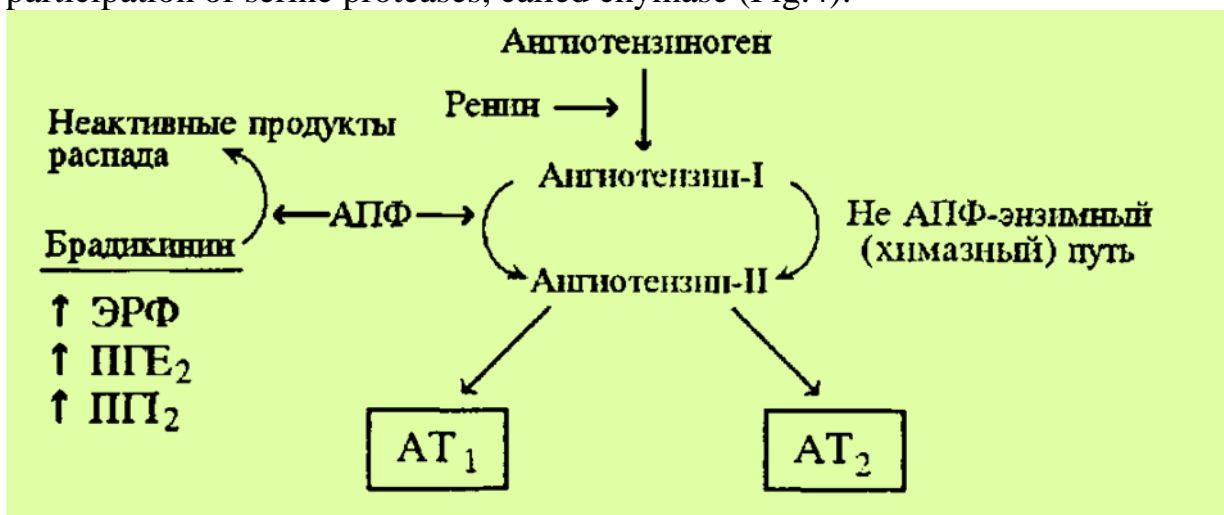


Figure 4. The scheme of the renin-angiotensin system.

The main mechanisms of action of angiotensin-II realized through AT_I receptors:

- increase in the tone of smooth muscle cells of the vessel;
- stimulation of synthesis and release of aldosterone;
- facilitate the transfer of impulses in sympathetic ganglia;
- stimulation of the release of norepinephrine from nerve endings;
- stimulation of vasopressin release;
- inhibition of norepinephrine reuptake nerve endings;
- stimulation of the synthesis of prostaglandin E;
- inhibition of renin release;
- central hypertensive effect;
- positive inotropic effect of;
- intrarenal regulation of the functional activity of nephrons (tubuloglomerular relations);
- Increased sodium reabsorption in the tubules.

Among the hormones of the adrenal significant impact on the regulation of the cardiovascular system have aldosterone and catecholamines.

The participation of the other hormones in the regulation of blood pressure in physiological conditions can hardly be considered significant. Aldosterone plays a key role of regulation of water-salt balance in the body. The synthesis is carried out in its outer zona glomerulosa of the adrenal cortex from cholesterol.



Figure 5. The right and left adrenal gland

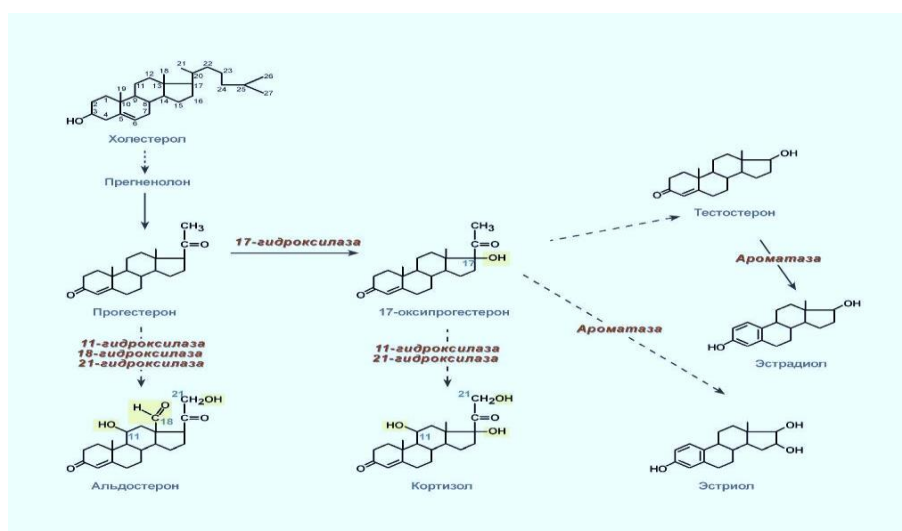


Figure 6. The formation of aldosterone from cholesterol

Among the identified to date hormones synthesized in the hypothalamus, the most significant role in the regulation of blood circulation belongs to vasopressin, ACTH and hypothalamic natriuretic factor (hormone).

The influence of many factors on blood pressure in whole or in part due to changes of sodium reabsorption in the kidney.

Possible mechanisms of action of sodium on blood pressure:

- an increase in cardiac output due to increased volume of circulating plasma;
- increasing the concentration of free calcium in the cells smooth intramuscularly;
- activation of ganglionic transmission of impulses;
- stimulation of the release of noradrenaline in the nerve endings;
- inhibition of the reuptake of noradrenaline nerve endings;

- increased activity of the sympathetic nervous system due to the stimulation of the central neurogenic mechanisms regulation of circulation;
- increasing the water content in the vascular wall with decrease of the inner radius of the arterioles and increase of the resistance to blood flow;
- increased sensitivity of vessels to noradrenaline and other vasopressor effects;
- stimulating the secretion of hypothalamic natriuretic factor;
- stimulation of secretion of vasopressin;
- partial depolarization of the membrane of smooth muscle cells.

Thus, the regulation of blood pressure - is a complex multifactorial process. Under physiological conditions, the most important factors of its regulation are the nervous system, the renin-angiotensin system, kidneys, as well as the mechanisms of autoregulation of blood flow. However, under pathological conditions, significantly increases the role of other factors, while secondary hypertension is often a dominant role in raising blood pressure can belong to one of the mechanisms involved in the regulation of blood pressure.

Symptomatic hypertension.

Symptomatic (secondary) arterial hypertension occur in many diseases (50). The presence of hypertension is a disease syndromes is not necessary, and raising blood pressure in the pathogenesis of these diseases are generally known (Tab. 2).

Table 2. The main groups of symptomatic arterial hypertension

Group	Disease
<p>Nephrogenic hypertension • parenchymal kidney disease</p> <ul style="list-style-type: none"> • renovascular <p>(Violation of blood flow in the main renal arteries)</p> <ul style="list-style-type: none"> • violation of the outflow of urine 	<ul style="list-style-type: none"> • glomerulonephritis • pyelonephritis • polycystic • diabetic glomerulosclerosis • tumors • Tuberculosis • atherosclerosis • fibromuscular dysplasia • thromboembolism • aneurysm, fistula • hypoplasia • nephroptosis, hydronephrosis, ureterohydronephrosis, compression of the urinary tract • reflux nephropathy

<p>Endocrine hypertension</p>	<ul style="list-style-type: none"> • primary hyperaldosteronism (Conn's syndrome) • disease and Cushing's syndrome • pheochromocytoma • acromegaly • thyrotoxicosis • syndrome of excessive production of deoxycorticosterone
<p>Hemodynamic hypertension</p>	<ul style="list-style-type: none"> • atherosclerosis of the aorta and carotid arteries • coarctation of the aorta • nonspecific aortoarteriitis • atrioventricular block
<p>Hypertension with organic lesions of the nervous system</p>	<ul style="list-style-type: none"> • diencephalic syndrome • brain tumor • encephalitis • meningitis • polyneuritis
<p>Hypertension caused by intake of drugs (iatrogenic)</p>	<ul style="list-style-type: none"> • Glucocorticoids • oral contraceptives • Erythropoietin • cyclosporine

Renovascular hypertension (RVH) - a form of symptomatic arterial hypertension, which develops as a result of violations of the main renal blood flow without primary lesions of the renal parenchyma and urinary tract.

The renovascular hypertension is 2-5% among all forms of arterial hypertension.

The basis of renovascular hypertension is always a one- or bilateral renal artery constriction of any one or more of its major branches.



Figure 7. The main reasons of development of narrowing of the renal arteries

Etiology. Atherosclerosis is the leading cause of renovascular hypertension in persons older than 40 years and is 60-85% of cases. Atherosclerotic plaques are predominantly localized in the mouth or in the proximal third of the renal artery. In most cases, there is a unilateral lesion of the renal artery, while its bilateral disease occurs in about 1/3 of cases and leads to a more severe course of renovascular hypertension. The disease frequently (2-3 times) in males.

Fibromuscular dysplasia as the cause of renovascular hypertension is second only to atherosclerosis. Fibromuscular dysplasia occurs predominantly in young and even children's age (12 to 44 years); the average age is 28-29. In women, it is found in 4-5 times more often than men. Fibromuscular dysplasia morphologically manifested in the form of dystrophic and sclerosing changes, exciting predominantly middle and inner membrane of the renal arteries and their branches. When this muscle hyperplasia wall elements can be combined to form microaneurysms. As a result, there is an alternation of contraction and expansion areas (aneurysms), which gives a peculiar form of the arteries - a thread of pearls or beads. Pathological process, though, and is common, but in 2/3 of the cases is one-sided.

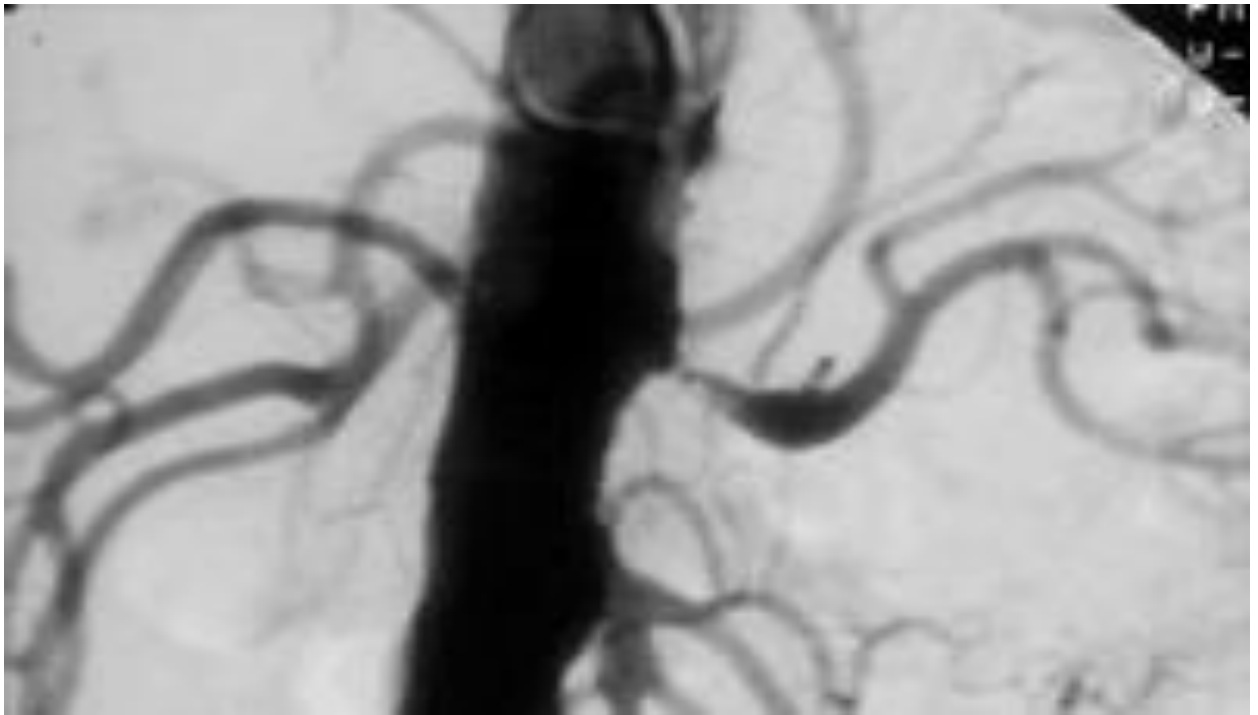


Figure 8. Stenosis of the left renal artery

Renovascular hypertension may occur due to extravasal compression of renal artery, resulting in thrombosis or embolism of the renal artery, aneurysm formation, the main renal artery hypoplasia, nephroptosis, tumors, cysts, and kidney malformations al.

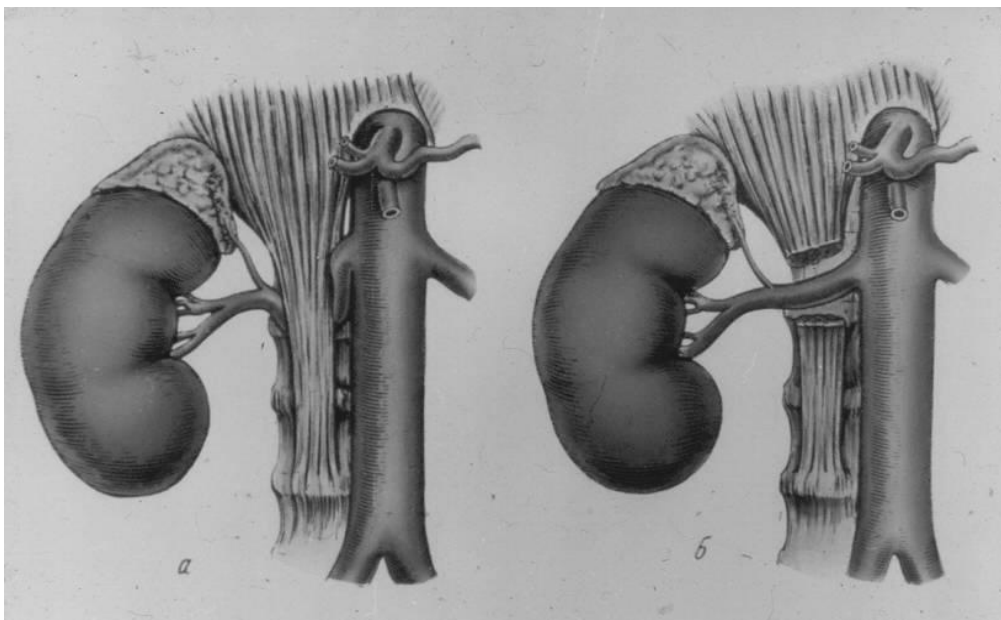


Figure 9. Compression of the renal artery leg aperture and decompression after crossing the last

Clinic. There are no specific symptoms renovascular hypertension characteristic of some forms of hypertension (Conn's syndrome, Cushing's syndrome, pheochromocytoma).

Complaints of the patients can be divided as follows:

1. Complaints specific to cerebral hypertension, - headaches, a feeling of heaviness in the head, tinnitus, pain in the eyeballs, memory loss, poor sleep.
2. Complaints related with overload of the left departments hearts and coronary insufficiency - pain in area and heart palpitations, feeling of gravity behind breastbone.
3. A feeling of heaviness in the lumbar region, not intensive pain, hematuria in the case of renal infarction.
4. Complaints specific to ischemia of other organs, major arteries which struck simultaneously with the renal arteries.
5. Complaints that are typical of the syndrome of inflammation in general (non-specific aortoarteriitis).
6. Complaints that are typical of secondary hyperaldosteronism: muscle weakness, paresthesias, seizures, tetany, izohypostenuria, polyuria, polydipsia, nocturia.

However, it should be noted that approximately 25% of patients with renovascular hypertension asymptomatic.

Diagnosics. For the diagnosis of AH important is medical history:

1. Stable development of hypertension in children and adolescents.
2. Stabilization and refractory to treatment of hypertension in persons older than 40 years who previously a benign disease, and antihypertensive therapy was effective, identifying these patients intermittent claudication or\and symptoms of chronic cerebrovascular insufficiency.
3. Feedback develop hypertension of pregnancy and childbirth (without nephropathy)
4. Communication of hypertension start with instrumental examination and manipulation in the kidneys, with operations in the kidneys and abdominal aorta.
5. Development of hypertension after an attack of pain in the lumbar region and haematuria in patients with heart disease, arrhythmias, or in patients with myocardial infarction, and episodes of arterial embolism in other basins.

On examination, measure the pressure on the upper and lower limbs that would eliminate coarctation syndrome and identify arterial lesions of the upper and lower extremities, as well as in the horizontal and vertical position. If orthostatic blood pressure above position, you can think about nephroptosis.

Need auscultation abdominal aorta and renal arteries - about 40% of patients auscultated systolic murmur in the projection of the renal arteries or abdominal aorta. Diagnosis can help listening systolic murmur over the superficial arteries: carotid, subclavian and femoral - as a sign of systemic lesions in atherosclerosis and aortitis.

Based on the survey and a series of studies can reveal the following features that can be suspected renovascular hypertension:

- hypertension, resistant to two or more antihypertensive drugs and diuretics;

- occurrence of hypertension before age 20 years in women, or after 55 years;
- rapidly progressive or malignant hypertension;
- the existence of different manifestations of multifocal atherosclerosis;
- azotemia, especially developing during treatment with ACE inhibitors or angiotensin receptor blockers II;
- systolic murmur over the abdominal aorta and the renal arteries;
- differences in the size of the kidneys in excess of 1.5 cm (based on the US);

The most authentic and reliable method for diagnosing renovascular hypertension is renal angiography, which can be performed in specialized vascular centers. Angiography to determine the cause of the stenotic process, assess the degree of stenosis and its location, which is crucial to decide on surgical treatment.

However, there are a number of minimally invasive, screening methods, which can detect loss of the renal arteries and to determine the indications for angiography and avoid applying it to those patients who may have a different genesis of hypertension.

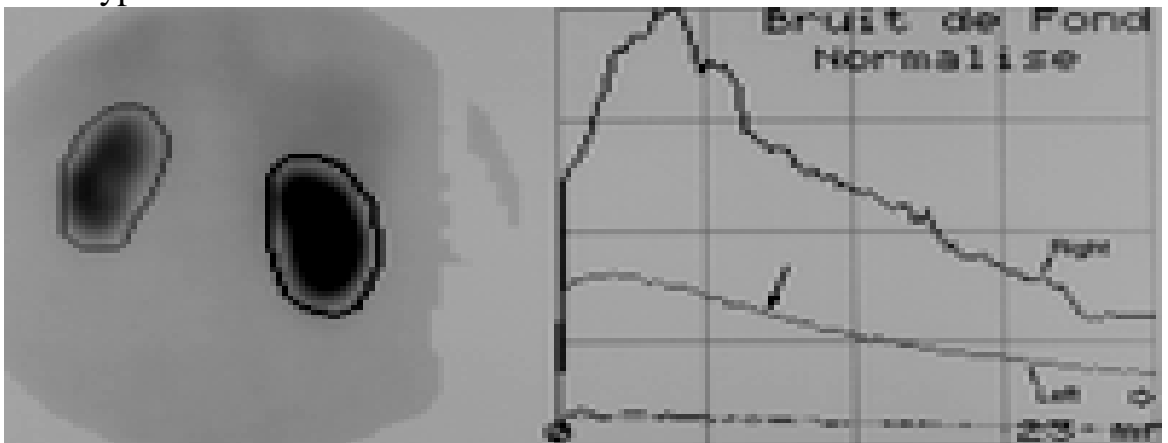


Figure 10. renoscintigraphy kidney

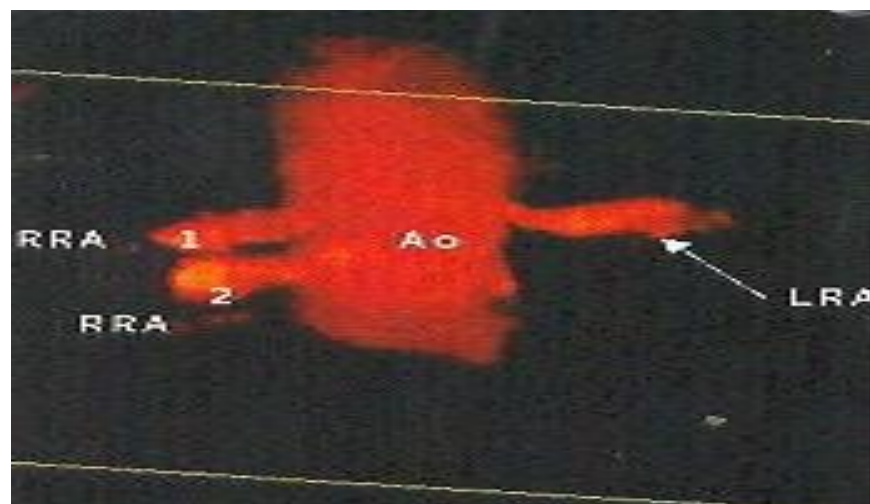


Figure 11. Three-dimensional duplex scanning (shown marked stenosis of the left renal artery and right renal artery extra).

Renoscintigraphy with inhibitors angiotensin-converting enzyme (ACE). The use of ACE inhibitors in functionally significant renal artery stenosis leads to a decrease in glomerular filtration rate, as a result of eliminating or significantly reducing constriction of efferent arterioles. This results in characteristic changes in renography.

Scintigram using angiotensin-converting enzyme (ACE) inhibitors should be interpreted consistently with low, medium and high probability of renovascular hypertension. The most specific diagnostic criterion for renovascular hypertension scintigraphy is an ACE inhibitor-induced change.

These criteria are:

1. Normal scintigram using ACE inhibitors indicates a low probability of RVH, less than 10%.
2. Reduced poorly functioning kidney (capture less than 30% of maximum activity over time [T-max] 2 minutes, which shows no change in scintigraphy with ACE inhibitors and bilateral symmetric disorders, such as cortical tubular delay agent indicates the average probability of RVH.
3. Criteria related to high probability of RVH include deterioration of scintigraphic curve, decrease in the slope, and the extension of renal parenchymal transit time, increase by 20 minutes / peak ratio capture rate, and the extension of T-max.

Doppler - ultrasonography. This study has advantages in view of its non-invasive and inexpensive. Two methods are used to detect the RVH using Doppler - ultrasound: direct visualization of the renal arteries and analysis of Doppler waveforms.

Direct visualization of the renal arteries. The first method involves the direct viewing of the main renal artery with color and energy Doppler - ultrasound velocity analysis with renal artery using spectral Doppler - ultrasound. Signal enhancement can be achieved by taking a contrast medium which facilitates the visual image of the renal arteries.

Three-dimensional ultrasound angiography provides a detailed visualization of the renal arteries and image accuracy comparable to the three-dimensional magnetic resonance angiography.

Magnetic resonance tomography is now available as a high-resolution system with high image quality, which is capable of forming a three-dimensional image. Blood provides bright, while still remain dark tissue.



Figure 12. Magnetic resonance tomography angiography of the renal arteries and aorta.

Multislice computed tomography angiography (MSCTA). MSCTA is a non-invasive method, however, requires the introduction of up to 150 ml of contrast medium. It can be adapted to measure renal blood flow in patients renovascular hypertension, and also receive the three-dimensional image of blood vessels.

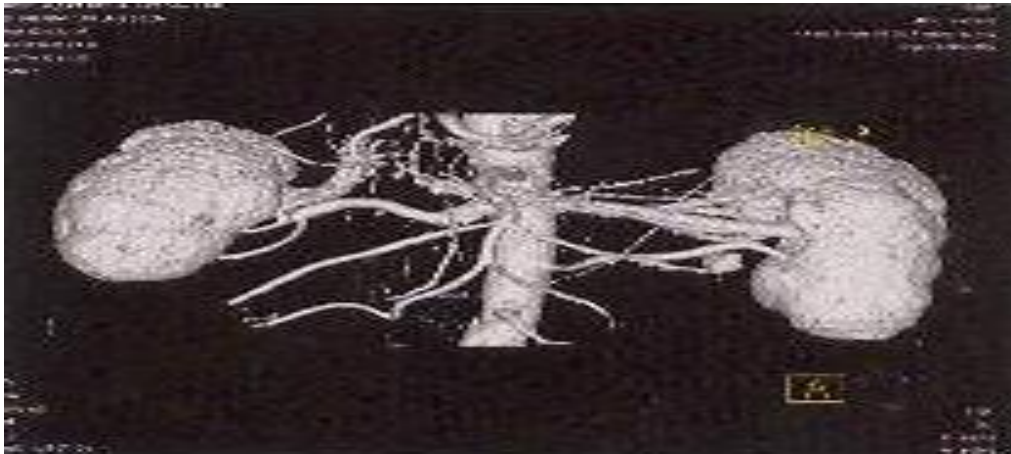


Figure 13. MSCTA of the abdominal aorta and its branches

Treatment. There are the following types of treatment:

1. Conservative - with contraindications to surgery.
2. Surgical methods:
 - Reconstructive surgery: transaortic endarterectomy, reimplantation of the renal artery, renal artery resection, prosthetic renal artery.
 - Traditional operations - nephrectomy.
3. Roentgen-endovascular methods: transluminal angioplasty of the renal artery (or roentgen-endovascular balloon dilatation -RED) with or without stenting; simultaneous REI on the adrenal glands to correct secondary hyperaldosteronism.

The most effective treatment for renovascular hypertension - surgery aimed at removing the causes of renal artery stenosis and the restoration of normal renal blood flow. Until 1952 the only method of surgical treatment was nephrectomy, which was used in a unilateral lesion and obviously in an advanced stage of the disease. Nephrectomy is applied at the moment, if the restriction is dominated by intrarenal vessels or in severe hypoplasia of the affected kidney and substantial violation of its functions. Indication for nephrectomy is to reduce the size of the kidneys to 8 cm or less. In other instances, well-used organ operations aimed at restoring renal blood flow. Results of surgical treatment more effective, the earlier the diagnosis of renovascular hypertension, and the reason for its occurrence.

At the same time in patients with renovascular hypertension, even with malignant course is sometimes possible to achieve a good effect with individually selected antihypertensives. However, with proven renal artery stenosis is not recommended drug therapy, as a decrease in blood pressure leading to further deterioration of renal blood flow and development in a short time secondary renal scarring and loss of its function.

Depending on the etiology of the disease in 80% of cases can be successful balloon dilatation or stenting (Figure 14).

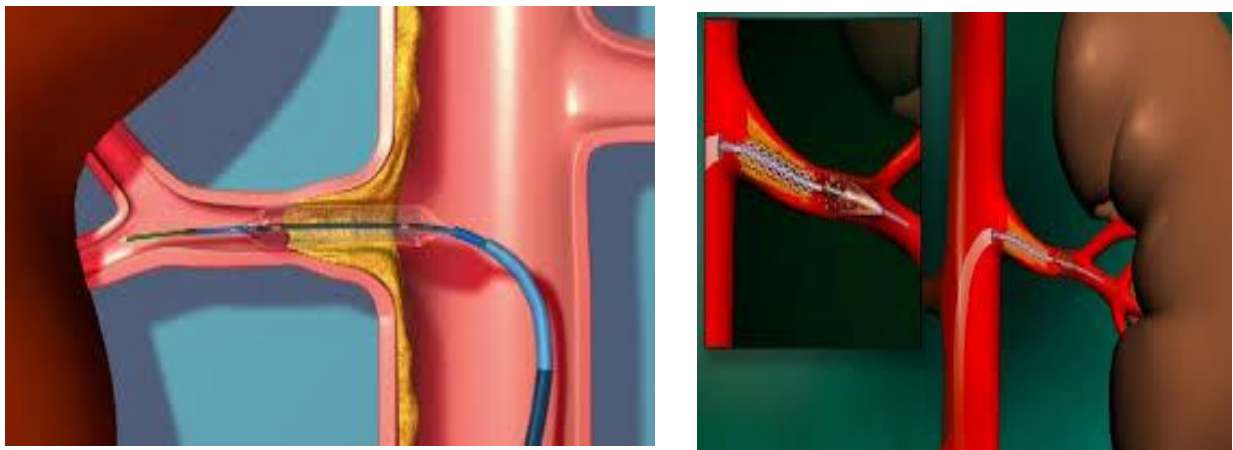


Figure 14. Dilation and stenting of the renal arteries

However, these procedures are invasive and can lead to rupture or dissection of an artery, an atheromatous emboli or renal lower limbs, due to acute renal failure, nephropathy induced by contrast, bleeding at the puncture site and side (rarely) the death of the patient.

Surgical revascularization remains the reserve method for those patients who have failed balloon dilatation and stenting, as well as for patients with concomitant abdominal aortic lesion requiring surgical intervention. Patients with high and poorly controlled hypertension, if this reduced the size of the kidneys and significantly reduced its function, shows a nephrectomy.

ARTERIAL HYPERTENSION OF ADRENAL GENESIS.

Adrenal hypertension caused most of his tumors. The most common are: aldosteronoma, pheochromocytoma, mixed tumor of the adrenal cortex,

corticosteroma and androsteroma. All these types tumors may be either benign or malignant.

Aldosteronoma (primary hyperaldosteronism, Conn's syndrome) develops from the glomerular zone of the adrenal cortex. In the majority of patients tumor is benign and only 5% of detected malignant growth pattern. Tumor tissue develops in excess aldosterone.

Excess aldosterone production causes various biochemical and morphological changes in the organism. The disease most often affects women. Symptoms of aldosteronoma can be divided into 3 groups:

- 1) neuromuscular
- 2) renal
- 3) associated with high blood pressure

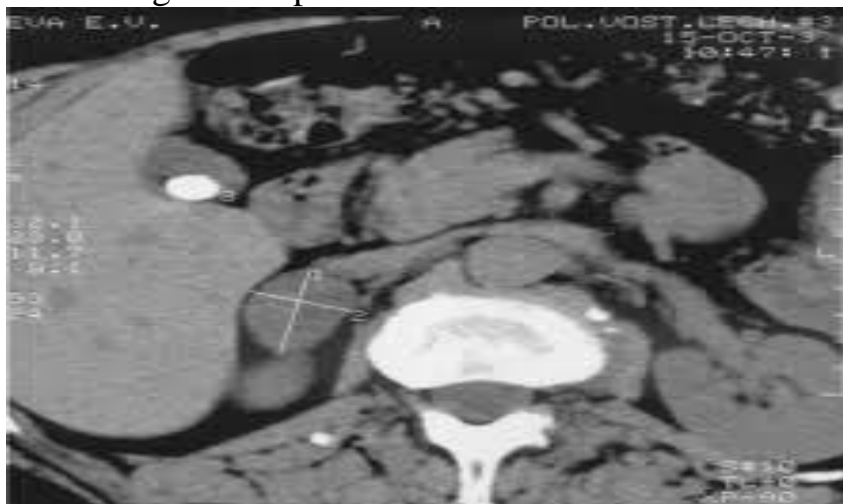


Figure 15. aldosteronoma of left adrenal gland

Pheochromocytoma - a tumor of neuroectodermal origin of the chromaffin tissue, producing catecholamines (epinephrine, norepinephrine, dopamine). The most commonly develops from the adrenal medulla (90%). In 10% of detected pheochromocytoma (paraganglia) not supernal gland localization (often in the para-aortic sympathetic ganglia, bladder, posterior mediastinum). In the pathogenesis of disorders developing in patients with pheochromocytoma, primary importance is the hypersecretion of catecholamines and periodic volley throw them into the systemic circulation.

The clinical picture. Main symptom of pheochromocytoma is hypertension, which can be of three types - a stable, paroxysmal and mixed, in connection with which emit corresponding types of clinical currency of disease.

Diagnostics. The leading role in establishing the diagnosis pheochromocytoma, along with the clinical picture belongs to study the concentration of catecholamines in the urine (daily or collected after the crisis). Given the large size of the tumor, they can easily be identified by ultrasound and CT.

Pheochromocytoma is treated only surgically - removing of the tumor.

Among other diseases of the adrenal glands is necessary to select a symptom of endogenous giperkorticism that combines various pathogenesis, but similar

clinical manifestations of the disease. A similar clinical picture is caused due to the overproduction of glucocorticoid hormones, primarily cortisol. Distinguish Kushing's syndrome and Kushing's disease (nontumor form).

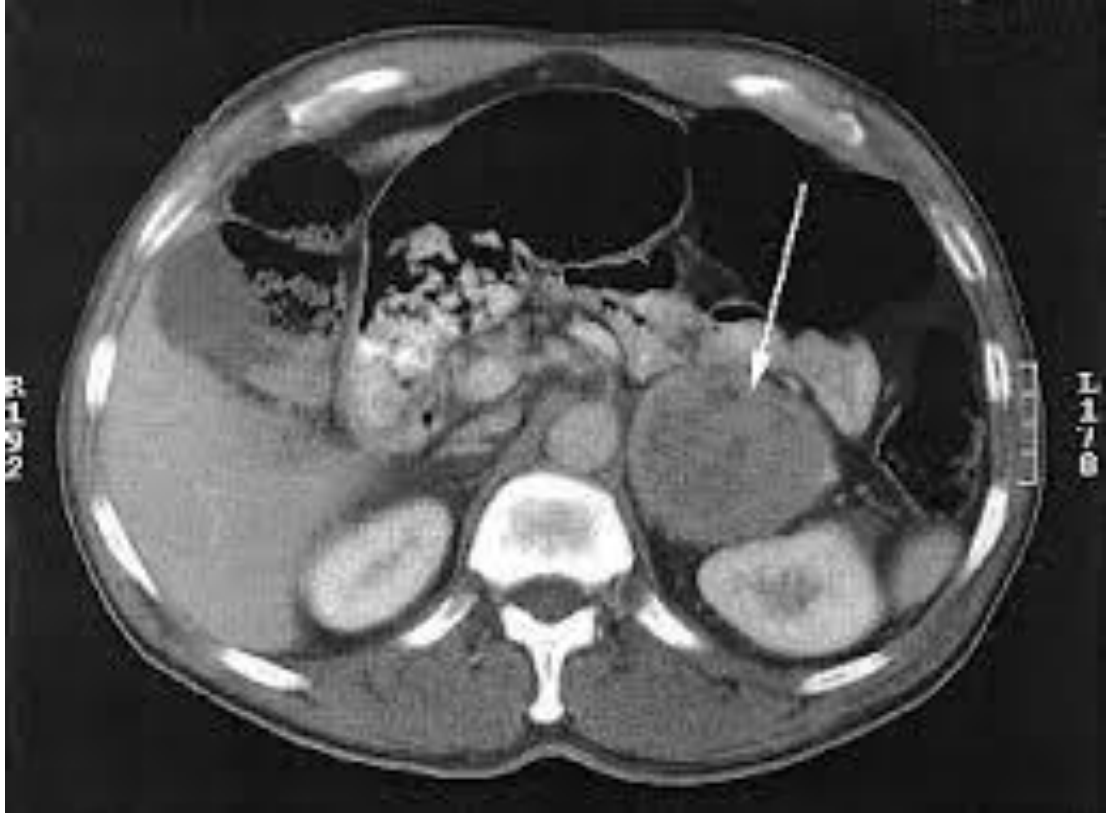


Figure 16. CT pheochromocytoma on the right.

Cushing's syndrome is caused by a tumor that develops from the beam cortex of suprarenal gland (benign tumor - corticosteroma, malignant - cortikoblastoma). Tumor tissue in an excess of cortisol produces. Come sick more women (almost 80%) aged 20-40 years. Clinical picture of syndrome and Cushing's disease is quite typical. The most constant symptom is obesity and hypertension. Appear early fatigue and muscle weakness, decreased performance, sexual dysfunction. In a later date joins osteoporosis. Obesity is associated with excessive production of cortisol and ACTH, retarding fat-mobilizing effect of growth hormone. Arterial hypertension in Cushing's syndrome has a stable flow, without crises, there is a proportional increase in systolic and diastolic blood pressure, resistant to antihypertensive therapy. Characterized by the appearance of patients - moon face, purple-bluish color of the face and upper chest, the presence of "red stretch marks" - purple-bluish stripes on the skin of the abdomen, waist, breasts, thighs. The skin becomes dry, the limbs become bluish-colored marble.



Figure 17. View of the patient, the presence of "red stretch marks" - purple-bluish stripes on the skin of the abdomen.

Diagnosis: the decisive role belongs to the study concentration level of 17 corticosteroids (17-KS) in blood and urine. When corticosteroma this figure significantly increased, especially in malignant nature of the tumor.

Diagnostics - ultrasound, CT.

Treatment: surgical - adrenalectomy - removing of the tumor (corticosteroma) with the adrenal gland.

Androsteroma develops from the zona reticularis of the adrenal cortex. The clinical picture is caused by overproduction of androgens. The disease occurs in young and middle age. More common in women. In childhood, girls appear hypertrichosis, accelerated growth, excessively developed muscles, voice becomes low, rough. In boys, puberty begins early, characterized by strengthening muscles development, short stature, short legs. In women, the disease manifests itself with the appearance of symptoms of masculinization of male sexual characteristics - reduction sub skin layer of fat, gain muscle development, atrophy of the breasts, menstrual dysfunction; often appears hirsutism.

In the study of the hormonal profile of the patient's attention is drawn to the contents of a huge 17-KS in urine. Revealing for tumor localization used ultrasound and CT.

Treatment: surgical - adrenalectomy.

The choice of a method of treatment for patients with hyperaldosteronism and arterial hypertension is one of the most difficult tasks. Conservative antihypertensive therapy in these patients, as a rule, is not effective enough, and traditional surgical methods of treatment are accompanied by a number of serious complications and do not always provide the desired effect (Rybakov S.I, Komissarenko I.V. 2004).

At the present time, the concept of minimally invasive interventions is dominant in the treatment of many surgical diseases.

At the moment, there are the following methods of treatment of AH adrenal genesis:

1. Endovascular methods of treatment - embolization or electrocoagulation of the central adrenal vein.

2. Open adrenalectomy.

3. Video endoscopic adrenalectomy. Here there are various operational accesses:

Transabdominal (laparoscopic)

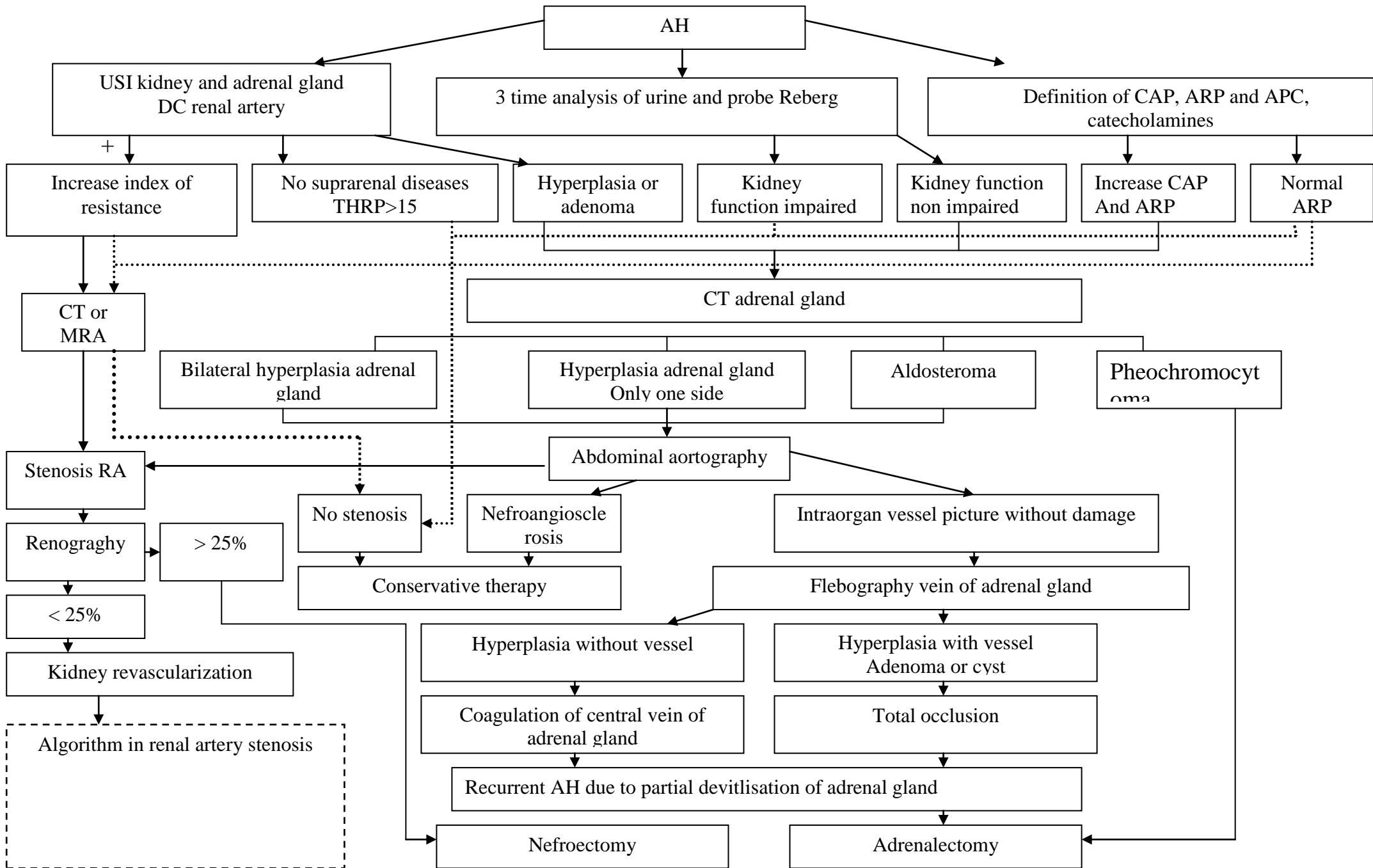
1. Direct

2. Side

Translumbial (retroperitoneal)

1. The lateral

2. back access



Therapeutic diagnostic algorithm.

Conclusion

The methodical recommendation is devoted to solving a very urgent problem. The algorithm of examination and treatment of patients with AH is an integral part of the daily work of primary care physicians, neuropathologists, cardiologists, therapists, ophthalmologists and otorhinolaryngologists. Since their role in the timely diagnosis and prevention of such formidable complications as myocardial infarction, stroke and heart failure is quite large.

In the methodical recommendation, the possibilities of modern methods of studying patients with AH are reflected and studied. The diagnostic value of each method of investigation in the early prevention of AH complications is estimated. The urgency and significance of timely diagnostics of arterial hypertension in modern medicine is briefly discussed.

The developed algorithm for diagnostics of hypertension should become a desktop tool for general practitioners, neuropathologists, cardiologists, therapists, ophthalmologists and otorhinolaryngologists, and also actively introduce the curriculum of senior students of medical universities.

USED IN THIS SESSION, NEW PEDAGOGICAL TECHNOLOGIES:

Method of round table

Students are located around the study table. The teacher on a sheet of paper writes down the task - list the methods of treatment of hypertension - and lets in a circle.

In our opinion, the method of the round table can be conducted in two versions.

1. The student writes down his own version of answers, signs, transposes it in such a way that the answer is closed and passes to the next student. The next student in the same way writes down his own answer, transposes the sheet and transfers it to the next student and so on until the end of the circle.

2. Each student writes on the sheet of paper only one answer and passes it to the neighbor. At the same time, the list of tasks can pass 2-3 circles, it is important that the previous answer is not repeated.

At the end, when all students answer a question, all options are discussed, correct ones are identified, wrong ones are crossed out. The teacher evaluates the knowledge of each student: 100-86% - excellent, 85-71% - good, 70-56% - satisfactory, 55% and lower - unsatisfactory.

USING THE WEB METHOD

Steps:

1. In advance, students are given time to prepare questions on the class they have attended.

2. Participants sit in a circle.

3. One of the participants is given a skein of threads, and he asks his prepared question (to which he himself must know the full answer), holding the end of the thread and rolling the hank to any student.

4. The student who received the skein answers the question (the participant who asked him comments on the answer) and passes the baton of the question further. Participants continue to ask questions and answer them until everyone is in the web.

5. As soon as all the students finish asking questions, the student holding the skein returns it to the participant, from whom he received the question, while asking his question, etc., until the ball is completely unwound.

Note: Warn the students that they should be attentive to each answer, because they do not know who will throw the skein.

USING THE "BLACK BOX" METHOD

The method provides for joint activities and active participation in the classes of each student, the teacher works with the entire group.

Each student takes out a question from the black box. (Questions are enclosed.) Students must detail their answer in detail.

The student is given 3 minutes to think over each answer. Then the answers are discussed, an addition is given for etiopathogenesis, clinical course. At the end of the method, the teacher comments on the correctness of the answer, its validity, the degree of student activity.

This technique contributes to the development of the student's speech, the formation of the foundations of critical thinking, tk. in this case, the student learns to defend his opinion, analyze the answers of fellow participants of this competition.

Test questions and tasks:

1. What are the causes of vasorenal hypertension:

- A. Urolithiasis
- B. Pyelonephritis
- C. Glomerulonephritis
- D.FMD
- E. Phlebitis of the renal vein

2. What can be the cause of vasorenal hypertension:

- A. Glomerulonephritis
- B. Pyelonephritis
- C. NAA
- D. Urolithiasis
- E. Phlebitis of the renal vein

3. What reconstructive surgery is performed with stenoses of the renal artery?
- A. Endarterectomy of the renal artery
 - B. Resection and prosthetics of the renal artery
 - C. Transaortal endarterectomy
 - D. Resection of the renal artery and reimplantation on the aorta
 - E. All answers are correct
4. What drugs are used for hypertension:
- A. Antihistamines
 - B. Coronarolytics
 - C. ACE Inhibitors
 - D. Protease Inhibitors
 - E. Holinomimetics
5. What drugs are used for hypertension:
- A. Antihistamines
 - B. Coronarolytics
 - C. Betta Blakator
 - D. Protease Inhibitors
 - E. Holinomimetics
6. How is the secretion of aldosterone in the body regulated?
- A. With the activity of the renin-angiotensin system
 - B. With the concentration of sodium and potassium ions in blood plasma
 - C. With the help of the hormone ACTH
 - D. With the activity of the kinin-kallikrein system
 - E. All answers are correct
7. What determines the secretion of renin?
- A. Decrease in pressure in the juxtaglomerular apparatus of the kidney
 - B. the state of the sympathetic nervous system
 - C. Decrease in the concentration of sodium ions in "macula densa"
 - D. Correct answers A, B, C.
 - E. Correct answers A, B.
8. What is inactivated by catecholamines:
- A. Catechol-O-methyltransferase (COMT)
 - B. monoamine oxidase (MAO)
 - C. Correct answers A and B
 - D. Transaminase
 - E. Neuroaminidase
9. Indicate signs of primary hyperaldosteronism (Conn's syndrome)
- A. Hypertension
 - B. Hypokalemia (potassium below 3.0 mmol / l);

- C. Hyperaldosteronism
- D. Giporeninemia
- E. All answers are correct

10. Indicate the types of clinical course of pheochromocytoma:

- A. Paroxysmal form
- B. Constant form
- C. Mixed form
- D. All answers are correct

Situational challenge

The patient is 64 years old with Leriche syndrome. In the patient of the last 7 months, the blood pressure rises to 210/110 mm Hg. Conservative therapy is not effective.

- Reason AH
- What research needs to be done to confirm the diagnosis
- What diseases lead to hypertension
- Tactics of GPs

REFERENCES

1. A. V. Pokrovsky. Clinical Angiology Volume 1, 2004. p. 745.
2. A. V. Pokrovsky. Clinical Angiology Volume 2, 2004. p. 734.
3. Alekryan B.G., Buzishvili Yu.I., Golubova K.Z. et al. The immediate and long-term results of stenting of the renal arteries in patients with vasorenal hypertension. // Angiology and vascular surgery. - 2006. - №1. - P. 55-62.
4. Batalov I.Kh. Risk assessment and choice of endovideosurgical access in patients with adrenal pathology. // Author's abstract. diss. Cand. honey. sciences. - St. Petersburg. - 2008. - 25 pp.
5. Belov Yu.V., Bogopolskaya O.M. Vasorenal hypertension frequency, etiology, pathogenesis. Drug treatment. // Angiology and vascular surgery. - 2007; 13 (2): p. 135-141.
6. Belov Yu.V., Stepanenko A.B., Kosenkov A.N. Surgery of vasorenal hypertension. - MIA. - M. - 2007. - 263 p.
7. Emelyanov SI, Veredchenko VA, Mitichkin AE The use of three-dimensional computed tomography in the planning of laparoscopic adrenalectomy. // Wedge. and expert. hir. - 2008. - №1. - C. 35-34.
8. Emelyanov SI, Veredchenko V.A. The possibilities of two-dimensional radial imaging in the differential diagnosis of adrenal neoplasms. // Endoscope. hir. - 2008. - №3. - P. 35-40.

9. Sunnatov R.D. Rationale and choice of the method of X-ray endovascular interventions in patients with arterial hypertension of adrenal origin. // Diss. Cand. honey. sciences. - Tashkent. - 2000. - 120 pages.

10. Abela R., Ivanova S., Lidder S., Morris R., Hamilton G. An analysis comparing open surgical and endovascular treatment of atherosclerotic renal artery stenosis. // *Eur. J. Vasc. Endovasc. Surg.* – 2009. Dec.; 38(6): P. 666-675.

11. Alhadad A., Sterner G., Herlitz H. Treatment of atherosclerotic renal artery stenosis. Low dosage ACE inhibitors and angiotensin-receptor blockers are justified in certain cases. // *Lakartidningen.* – 2009. Oct.-Nov.; 106(44): P. 2836-2838, 2840.

12. Balzer K.M., Pfeiffer T., Rossbach S., Voiculescu A., Godehardt E., Sandmann W. Prospective randomized trial of operative vs interventional treatment for renal artery ostial occlusive disease (RAOOD). // *J. Vasc. Surg.* – 2009. Mar.; 49(3): P. 667-675.

13. Beck A.W., Nolan B.W., De Martino R., Yuo T.H., Tanski W.J., Walsh D.B., Powell R.P., Cronenwett J.L. Predicting blood pressure response after renal artery stenting. // *J. Vasc. Surg.* – 2010. Feb.; 51(2): P. 380-385.

14. Chrysochou C., Kalra P.A. Atheromatous renovascular disease: overview and challenges. // *J. Ren. Care.* – 2008. Dec.; 34(4): P. 179-190.

15. Chrysochou C., Kalra P.A. Epidemiology and natural history of atherosclerotic renovascular disease. // *Prog. Cardiovasc. Dis.* – 2009. Nov.-Dec.; 52(3): P. 184-195.

16. Chua S.K., Hung H.F. Renal artery stent fracture with refractory hypertension: a case report and review of the literature. // *Catheter. Cardiovasc. Interv.* – 2009. Jul.; 74(1): P. 37-42.

17. Colapinto R.J., Stroneell R.D., Harrier-Jones E.P. et al. Percutaneous transluminal dilatation of the renal artery: Follow-up studies on renovascular hypertension. // *Amer. J. Surg.* – 1993. – V. 4. – P. 728-732.

18. Connell J.M.C., Fraser R., MacKenzie S et al. Is altered adrenal steroid biosynthesis a key intermediate phenotype in hypertension. // *Hypertens.* – 2003; 41: P. 993-999.

19. Corriere M.A., Pearce J.D., Edwards M.S., Stafford J.M., Hansen K.J. Endovascular management of atherosclerotic renovascular disease: early results following primary intervention. // *J. Vasc. Surg.* – 2008. Sep.; 48(3): P. 580-588.

20. Courtade A., Atat I., Forzy G., Filoche B., Lucidarme D., Corman N., Desrousseaux B. Computed tomography of the adrenal glands in hypertension. // *J. Chir.* – 1997. – №7. – P. 291-295.

21. Covic A., Gusbeth-Tatomir P. The role of the renin-angiotensin-aldosterone system in renal artery stenosis, renovascular hypertension, and ischemic nephropathy: diagnostic implications. // *Prog. Cardiovasc. Dis.* – 2009. Nov.-Dec.; 52(3): P. 204-208.

22. Daou R. Hypertension and low plasma renin activity presumptive evidence for mineralcorticoid excess. // *Ann. Chir.* – 1992. – №7. – P. 625-628.

23. Das C.J., Neyaz Z., Thapa P., Sharma S., Vashist S. Fibromuscular dysplasia of the renal arteries: a radiological review. // *Int. Urol. Nephrol.* – 2007; 39(1): P. 233-238.

24. Davis R.P., Pearce J.D., Craven T.E., Moore P.S., Edwards M.S., Godshall C.J., Hansen K.J. Atherosclerotic renovascular disease among hypertensive adults. // *J. Vasc. Surg.* – 2009. Sep.; 50(3): P. 564-571.

25. De Donato G., Setacci C., Chisci E., Setacci F., Palasciano G. Renovascular hypertension. 8 years experience of a vascular surgery centre. // *J. Cardiovasc. Surg. (Torino).* – 2007. Aug.; 48(4): P. 403-409.

26. Gao J., Li J.C., Xiao M.S., Ng A., Trost D., Goldstein M., Kapur S., Wang J., Serur D., Dai Q., Jiang Y.X., Min R.J. Color duplex sonography in severe transplant renal artery stenosis: a comparison of end-to-end and end-to-side arterial anastomoses. // *Clin. Imaging.* – 2009. Mar.-Apr.; 33(2): P. 116-122.

27. Tsunoda K., Abe K., Yamada M., Kato T., Yaoita H., Taguma Y., Goto Y., Ioridani N. A case of primary aldosteronism associated with renal artery stenosis and preclinical Cushing's syndrome. // *Hypertens. Res.* – 2008. Aug.; 31(8): P. 1669-1675.