MINISTRY OF HEALTH
THE REPUBLIC OF UZBEKISTAN

TASHKENT MEDICAL ACADEMY

Department of faculty and hospital surgery

ARTERIAL HYPERTENSION.
DIAGNOSIS AND SURGICAL TREATMENT

(teaching aid)
for Master-students and medical residents

Tashkent - 2015

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CHAPTER 1.
Definition, epidemiology and etiology of 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.

The definition of hypertension on blood pressure is temporary. It's now known that the blood pressure level of 140/90 mm Hg. is accompanied by a greater risk of cardiovascular complications than blood pressure at 135/85 mm Hg.

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

Threatening complication of hypertensive states cerebral stroke, myocardial infarction, renal failure develop, usually at a late stage in the evolution of hypertension when no therapeutic or preventive measures do not bring the proper effect already.

The high prevalence of hypertension, the problem of its diagnosis and surgical treatment and assessment of the effectiveness of the treatment of hypertension is one of the urgent problems of modern clinical medicine. This is due, above all, a significant frequency of pathology, clinical forms of diversity and complexity of their differential diagnosis. Questions early diagnosis of pathological changes in the kidney, the evaluation of functional changes in the course of the treatment, as well as an objective monitoring of the effectiveness of treatment and the correct prognosis remains unsolved. This is confirmed by the high frequency of diagnostic errors in the early stages of the disease, caused by a latent course of the disease, as well as small informative diagnostic methods. The importance of timely and accurate diagnosis of the disease by the following figures: in patients with diastolic blood pressure of 105 mm Hg the risk of stroke in the brain 10 times, and the risk of 5 times higher than in patients with a diastolic pressure of 76 mm Hg Isolated increase in systolic blood pressure over 160 mmHg also increases the probability of formation of coronary heart disease 4.2 times.

Determine the cause of hypertension is possible to only a small proportion of patients 5-10% of adults with hypertension. Screening patients with secondary forms of hypertension is based on the study of medical history, physical examination and laboratory instrumental methods of investigation.

Problems AG have not only medical but also socio-economic importance, so the researchers face the task of developing practical guidelines on the prevention, diagnosis and treatment of this disease. In this direction, working clinical institutions in many countries, but the results are still far from the goal [1,2].

According to the Russian State Research Center for Preventive Medicine (State Research Center), 25 - 30% of adults have high blood pressure, are aware of this 57%, 17% of them receive treatment. The prevalence of hypertension increases with age [3,4].

According to WHO estimates, in premature mortality in the Russian Federation AG ranked first. About 20 years ago it was demonstrated that when patients treated provide a regular and try to monitor blood pressure, it can reduce mortality from stroke by 48%. Hypertension risk factor remains after cerebral stroke and transient ischemic attacks. Several studies have shown that if patients after stroke, conduct active treatment to reduce blood pressure, can reduce the risk of recurrent stroke by 28% [5,6].

According to the prospective study on the prevention of multifactorial (MRFIT), the risk of developing cardiovascular complications steadily increases with both SBP and DBP. So, if the risk of coronary heart disease with normal blood pressure taken for 1, then an isolated increase in DBP greater than 100 mm RTST similar risk was 3.32; in isolated systolic increase of more than 160 mm RTST - 4.19; and the combined increase in SBP and DBP - 4.57.According to the same study an increase in SBP of 10 mm RTST above baseline increases the risk of renal complications in 1.65 times. Prospective surveillance, held in the US city of Framingham for 34 years, found that those with high blood pressure the risk of chronic heart failure is 2-4 times higher than that of individuals with low blood pressure [7,8,9].

Data from prospective studies suggest that patients with diastolic blood pressure of 105 mm RTST risk of stroke brain is 10 times higher and the risk of CHD is 5 times higher than in patients with diastolic blood pressure of 76 mm Hg. Prolonged reduction DBP 5 - 10 mm RTST reduces the incidence of cerebral strokes 34, 46 and 56% and the IHD 21, 29 and 37%, respectively [10,11].

In the world reports of deaths from diseases of the cardiovascular system hypertension is often viewed as a leading risk factor for coronary heart disease [12,13]. According to the developed world, at autopsy sclerosis of the coronary arteries occurs in 55 - 85% with hypertension [14,15].

Unfortunately, not always the patients with elevated BP unaware of their disease and regularly treated. Thus, according to the study NHANES III, conducted in the US, 35% of patients with SBP above 140 mmHg or diastolic blood pressure above 90 mm Hg did not know about high blood pressure, only 49% of people with high blood pressure received medical treatment, and only 21% of those treated, managed to reduce SBP below 140 mmHg and diastolic blood pressure below 90 mm Hg [16,17]. Similar results were obtained in other countries. For example, in Russia, according to V.I. Makolkin (2002) of a known AH 37.1% of men and 58.9% women and treated -21.6 and 46.7%, respectively.

AG has the highest contribution to mortality from cardiovascular disease (CVD). Most often, patients die from its complications. The relationship between blood pressure levels and the risk of CVD is continuous, constant and independent of other risk factors. In other words, the higher the blood pressure, the greater the risk of developing cardiovascular complications. For example, data from prospective studies conducted in different years in the State Research Center of Preventive Medicine, showed that if the risk of death in men with systolic blood pressure (SBP) less than 115 mm Hg taken as a unit, then the level of this index over 160 mmHg the risk of death from coronary heart disease (CHD) is increased by 4 times, and stroke - almost 9 times (Fig. 1).

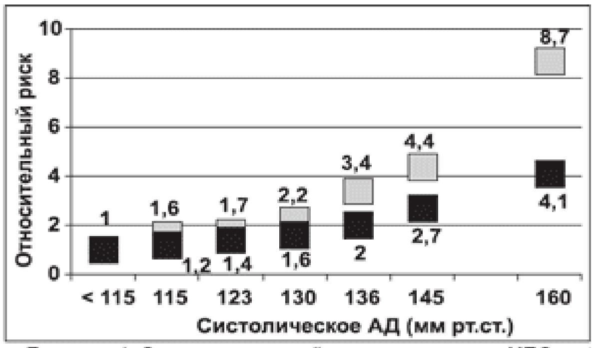


Рисунок 1. Относительный риск смерти от ИБС и инсульта в зависимости от САД (мужчины 40–59 лет)

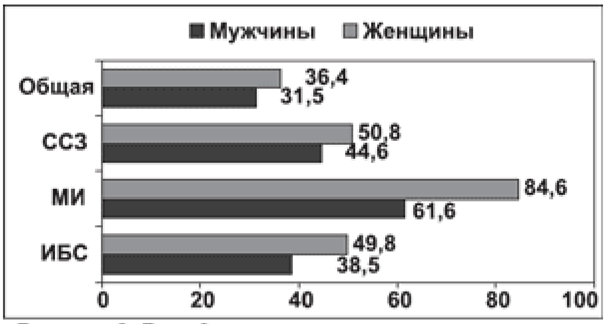


Рисунок 2. Вклад систолического артериального давления в смертность от ССЗ (Р.Г. Оганов и соавт., 2001)

Attributable mortality risk is a function of the relative risk and prevalence, and therefore states with a higher prevalence will have higher values of attributable risk of death. Indeed, analysis of the attributable risk of death in men, depending on the blood pressure showed that mortality from stroke (MI) by more than 60% can be attributed to SBP (Fig. 2). Women, like men, SBP and more significantly the mortality of MI (84.6%). The overall mortality rate is determined by the level of SBP by 31.5% in men and 36.4% women.

Thus, for the effective treatment of hypertension could theoretically save about one-third of men and women. Survival analysis depending on the level of blood pressure demonstrates the dramatic loss of life expectancy of men and women with high blood pressure. According to the State Research Center for Preventive Medicine, men and women with SBP of 180 mm Hg and more live 10 years less than those who have less than SBP of 120 mm Hg [18].

However, as shown by the results of monitoring the epidemiology of hypertension, conducted within the framework of the federal target program "Prevention, diagnosis and treatment of hypertension in Russia", in the last 10-15 years the epidemiological situation associated with hypertension, has not changed. Thus, in 2004 and 2006. the prevalence of hypertension is still accounted for 39% of men and 41% women, which indicates almost complete absence of primary prevention (Fig. 3).

Рисунок 3. Динамика распространенности АГ в Российской Федерации по данным мониторинга 2004 и 2006 гг.

Recommendations for a long time, hypertension were directed only to determine the levels of blood pressure, as well as the need for medical treatment, and the choice of drug treatment. However, in 2003, the recommendations of the European Society of Hypertension and the European Society of Cardiology emphasized that the diagnosis and treatment of hypertension should be determined as total risk [19]. The recommendations of the GFCF this position was supported [20]. Numerous epidemiological studies have shown that only a small proportion of patients with hypertension have only increased blood pressure, but the majority demonstrate the presence of additional risk factors for CVD [21]. Hypertension is associated with metabolic dyslipidemia, impaired glucose tolerance, abdominal obesity, hyperinsulinemia and hyperuricemia. Approximately 63% of cases of ischemic heart disease in male hypertensive recorded with a combination of two or more additional risk factors [22]. Influence of additional risk factors is especially important in the 1st stage of hypertension, when the average risk of high blood pressure is still very small, but many patients must be treated to prevent the development of cardiovascular disease. There is evidence that high-risk patients, target BP level with antihypertensive therapy, and prevention approaches should be different from those in patients with lower risk of cardiovascular disease. These data were taken into account in the recommendations for the prevention of CVP in clinical practice, and the intensity of the intervention in the formation of a prophylactic and therapeutic approach should be conditioned total cardiovascular risk [23]. Of course, it should be borne in mind that the definition of risk factors and methods of measurement can be very different in different studies. However, the likelihood of increased cardiovascular risk in the presence of hypertension associated with other risk factors should be considered established. Unfortunately, the prevalence of these conditions is increasing worldwide. Thus, the analysis of patient data Health Search Database in Italy in 2003 revealed that about 40% of hypertensive patients were three additional factor, whereas in 2000 this figure was only 29% [24].

Problem of AH relevant for Uzbekistan. Recently, increasing the frequency of complications of hypertension, especially of stroke, myocardial infarction and renal failure [25]. Thus, in Tashkent among men aged 40-59 years the prevalence of hypertension was 26.6%; among the unorganized rural population of Tashkent region hypertension was detected in 14.4%. With age, it tends to grow, reaching 50-59 years to 24.2% [26,27]. Epidemiological studies have found a significant and independent association between elevated blood pressure and coronary heart disease, stroke, congestive heart failure and kidney disease [4].

In prophylactic examination in Tashkent in 48% of cases of newly diagnosed hypertension. Of these, 24% with hypertension were aware of the presence of high blood pressure at home, 13% were treated at the time of the survey, and only 5% were receiving effective treatment [5].

Symptomatic hypertension spread much wider than is commonly believed. At secondary hypertension accounts for between 12% to 23% of all cases of elevated

BP. Epidemiological studies in recent years suggest that 23% -the most close to the truth indicator.

The prevalence, hypertension, a high percentage of its complications, loss of people of working age necessitated the search for new methods of diagnosis, treatment and prevention.

CHAPTER 2.
PATHOPHYSIOLOGY OF BLOOD PRESSURE.

Blood pressure (BP) - quite labile value. BP monitoring results indicate that in healthy individuals, it varies considerably throughout the day. The maximum values of BP recorded during the day, especially during emotional or physical stress. At night, the lowest values observed in AD. BP values largely determines the adequacy of the blood supply to organs and tissues. Its values, in turn, depend on the amount of blood flowing from the heart into the aorta per minute (cardiac output) and the resistance exerted by the blood flow in the arterial vessels, preferably in small arteries and arterioles (total peripheral vascular resistance). A significant effect on blood pressure has plenty of circulating blood, taking part in the formation of values of cardiac output (Fig. 4).Changes in any of these parameters in the absence of the other two adaptive changes naturally lead to changes in blood pressure. Under physiological conditions, these three parameters are closely related, which determines the relative constancy of blood pressure. The relative constancy of blood pressure at rest and regular changes at different loadings indicate the presence of a rather complex regulation of blood pressure.

Figure 4. Determinants of blood pressure.
CO - cardiac output;
TPR - total peripheral resistance;
CBV is the 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.

The nervous system and the regulation of blood pressure

Neurogenic control of blood flow is an extremely complex process, which is carried out both directly by the nervous system, and indirectly - through a change in hormonal activity and humoral mechanisms of regulation of blood pressure. It is implemented mainly through changes impulses to the heart, vascular arterial and venous circulation departments and involves the integration and transformation of multiple afferent influences from these regions in the efferent impulses. Significant effect on the severity of the efferent impulses have central structures of the autonomic nervous system, in which the processing and integration of afferent information.

Neurogenic regulation of the circulation loop can be represented in the form of three basic parts: the afferent, central and efferent (Fig. 5).

The most important afferent input in the regulation of blood flow information is to "own" the reflex zones of the cardiovascular system. The most significant in relation to the regulation of blood pressure are receptors located in the aortic arch, sinocarotid areas in the heart and blood vessels of the pulmonary circulation. Perceiving pulse fluctuations vascular wall (arterial baroreceptors and mechanoreceptors) or change the central venous pressure and blood filling of the right heart (low pressure receptors), these receptors respond to deformation (or extension) of the vascular wall or chambers of the heart. Information from mechanoreceptors on afferent nerve enters the medulla and in the upstream parts of the central nervous system related to the regulation of blood pressure.

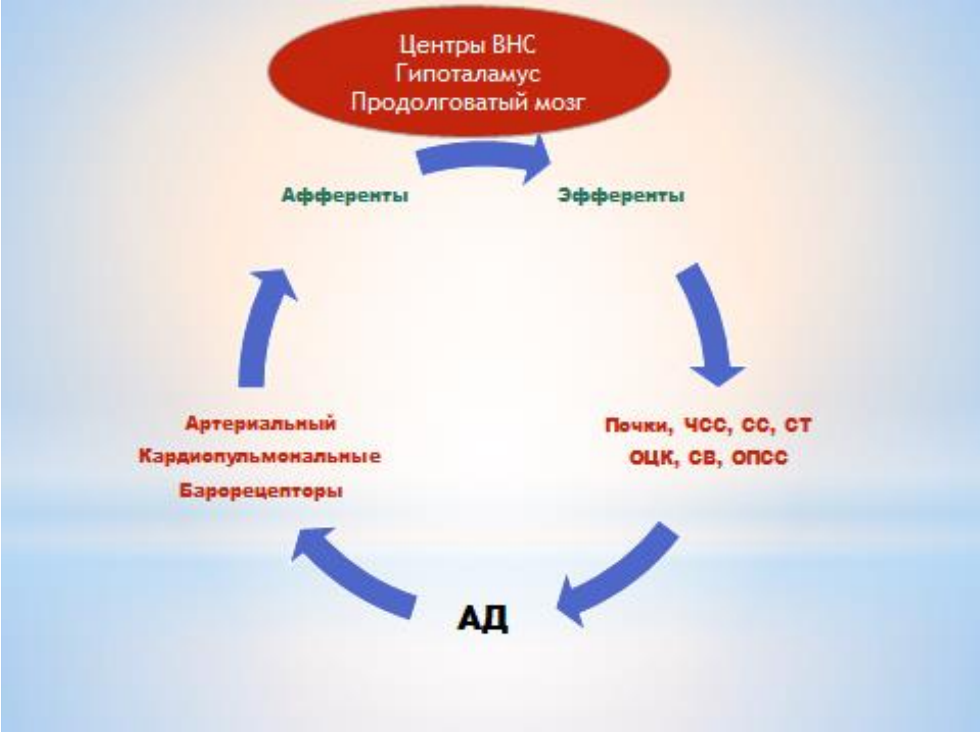


Figure 5. Neurogenic regulation of blood pressure.

Heart rate - the number of heartbeats, CC - cardiac contractility, VT - vascular tone, BV - blood volume, CO - cardiac output, PVR - total peripheral vascular resistance.

The most important in the integration of different influences and modulation of afferent reflex cardiovascular response neurons of the nucleus of the solitary tract, in turn, these neurons are widely interconnected with various parts of the central nervous system. Afferent information they receive on the adrenergic (including dopaminergic) nerves.

It is also possible effects on neurons of these nuclei hormonal factors and cerebrospinal fluid due to their close ties with *areapostrema*. Axons of catecholamine cell nucleus of the solitary tract commonly associated with different parts of the brain: the sympathetic and vagal preganglionic neurons, modulating autonomous control of circulation; other neurons of the brain stem; the higher centers of the brain, including vasomotor neurons of the hypothalamus, has a pronounced effect on the effector cardiovascular responses.

Efferent link neurogenic regulation of the circulation loop is represented vasomotor sympathetic neurons localized in the thoracolumbar spinal cord. The axons of these neurons are typical of cholinergic nerves. They leave the spinal cord as part of the anterior roots and enter the paravertebral sympathetic ganglia. From the sympathetic ganglia postganglionic axons directed to the heart and blood vessels. Increased activity of the sympathetic nervous system is accompanied by increased release of norepinephrine, which through adrenergic receptors alters the number of contractions of the heart and myocardial contractility (changes in cardiac output), as well as tone of vascular cells (change in resistance to blood flow and, to a lesser extent, venous return of blood to the heart) . Changes in the activity of the vagus mainly affects the number of heartbeats.

Stimulation of the receptors of the carotid sinus and aortic arch, occurs in response to an increase in pressure leads to a slowing of the heart rate and, to a lesser extent, a decrease in the tone of resistance vessels. The opposite influence blood pressure reduction. Cardiopulmonary receptors zone does not have a significant impact on the number of heartbeats. Change their activity mainly affect changes in blood flow in arteries and veins. The decrease in tensile mechanical receptors (decrease venous return of blood to the heart) causes an increase in the tone of blood vessels, increasing the tension (increase in venous return) - vasodilation. In addition, cardiopulmonary mechanoreceptors carry sympathetic control of renin release, regulate the reabsorption of sodium and water, have an impact on the value of the effective renal blood flow. Through these mechanisms cardiopulmonary baroreceptors involved in the regulation of circulating blood volume. On the volume of circulating blood may affect the reflex changes in pre- and post-capillary vascular tone, leading to pronounced changes in hydrostatic pressure in the capillaries and moving into the vascular or interstitial space of significant volumes of liquid.

There is no doubt that the central nervous system affects blood pressure, not only through baroreceptor mechanisms. Changes in the functional state of the central nervous system can affect blood pressure as a means of altering the activity of the autonomic nervous system, and by changing the rate of synthesis and release of many hormones and bioactive substances that are relevant to the regulation of blood pressure.

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: • changes in heart rate • changes in contractile activity Vessels: • Changing the tone of the smooth muscle cells of arteries and veins
Indirect	Change in the rate of synthesis and release of renin *, * Prostaglandins, * Kinins, * Vasopressin * ACTH * Hormones and other biologically active substances.

	Changing the speed of the water and sodium reabsorption in the kidney.

Kidney and blood pressure regulation

Kidneys occupy a central place in the regulation of blood pressure. Numerous studies A.Guyton indicate a close relationship between the level of blood pressure and renal function. The kidneys are the main regulatory body for the constancy of sodium and water in the body. Even minor changes in the body content of sodium and extracellular fluid affect the level of blood pressure. Changes in blood pressure, in turn, lead to a change in glomerular filtration rate and the reabsorption of sodium and water with the restoration of water and electrolyte homeostasis. Adapting to the needs of renal function of the body is provided by a number of interconnected systems and biologically active substances. Some of these substances are synthesized in the kidney, where implemented, and their effect (renal system kinins, prostaglandins, renal dopamine).

Regulating blood pressure effects of other systems (renin-angiotensin system, autonomic nervous system) and hormones (vasopressin) due to both the intra- and extrarenal action. Finally, some of the hormones secreted by extrarenal, but have an impact on blood pressure primarily by the kidney (aldosterone, Na-uretic hormones).

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.

In recent years, studies have been performed in which established the possibility of the formation of angiotensin-II from angiotensin-I converting enzyme without (Fig. 6).

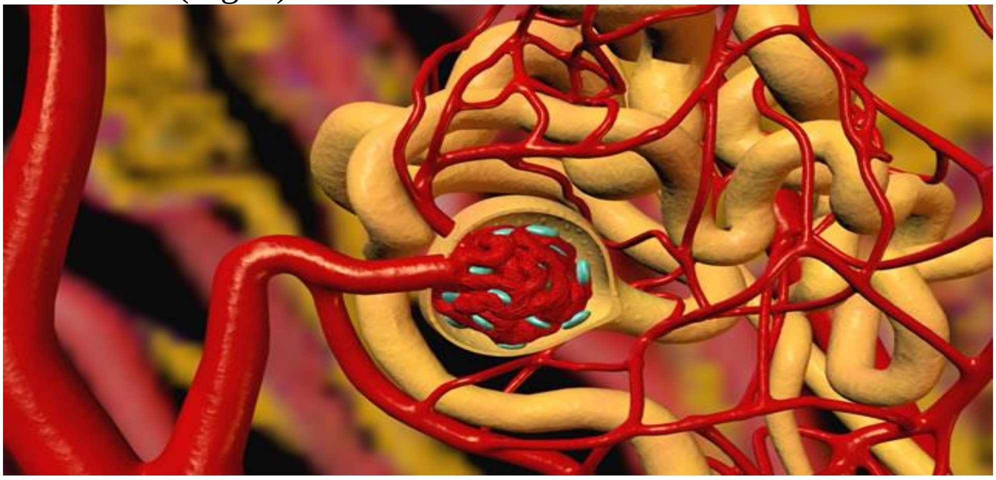


Figure 6. Glomerular unit

Thus the formation of angiotensin-II from angiotensin-I occurs with the participation of serine proteases, called chymase (Fig.7). Renin synthesis occurs in the membrane-bound cytoplasmic granules of specialized cells of the distal part of the afferent arterioles preglomerular. These cells, together with a portion of afferent arterioles, in which they are located, as well as with a dense patch *(macula densa)* and located between these two structures *lads* cells merged into one functional unit - juxtaglomerular apparatus.

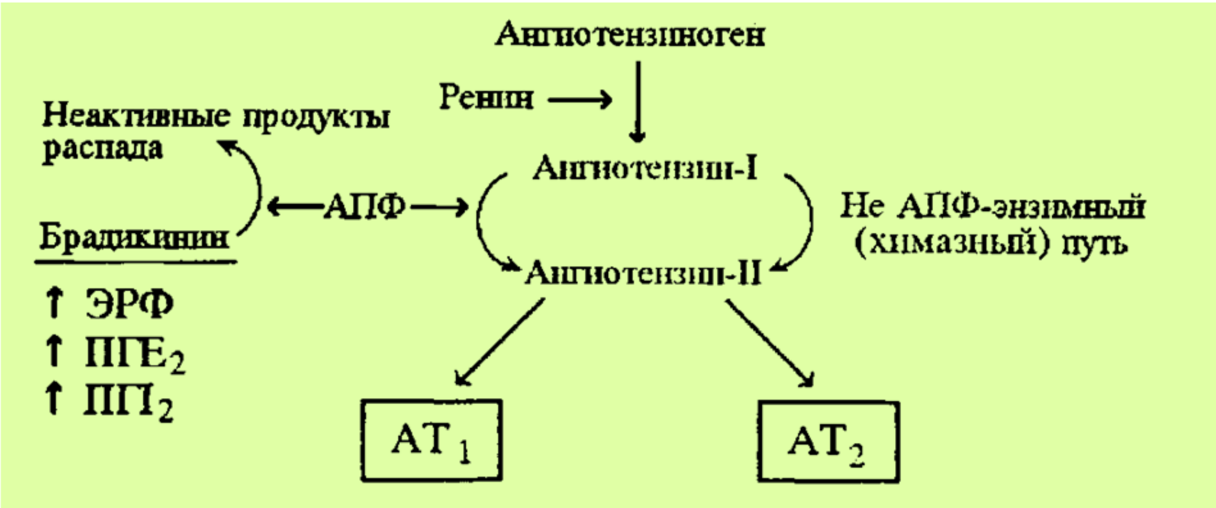


Figure 7. The scheme of the renin-angiotensin system.

Cellular elements *macula densa* are part of the epithelial cells of the distal tubule is in close contact with a ball, leading outlet and arterioles.

The rates of synthesis and release of renin governed by several factors acting either directly on the secretory cells, and indirectly - by changing tone and arterioles leading elements *macula densa*. Specifically, secretory cells themselves have adrenergic and apparently cholinergic innervation. Thus the stimulatory effect of sympathetic nerves as circulating catecholamines realized through the membrane disposed in the surface of the secretory cells of alpha-adrenergic receptors. Stimulate the secretion of renin lead tonicity enhancing glomerular arterioles, estrogens, prostacyclin and prostaglandin E, while angiotensin-II, aldosterone, vasopressin and renin release suppressed. Increase in extracellular fluid volume inhibits renin secretion, decrease - stimulates.

Directly related to the regulation of renin secretion and has *macula densa*, cell receptors which perceive changes in the concentration of sodium, potassium and chlorine possible. Reducing the concentration of sodium and chlorine at *maculaderisa* stimulates the release of renin, an increase - inhibited. Has the opposite effect change in the concentration of potassium.

Angiotensin-II has a broad spectrum of action. He, together with vasopressin plays a key role in the regulation of the constancy of extracellular fluid volume (through the regulation of the synthesis of aldosterone and tubuloglomerulyarnyh relationships within a single nephron). All these effects are realized through specific receptors located on the outer cell membrane. Apparently, there are several types of angiotensin-II - ATI and ATII receptor subtypes. Ultimately effect of angiotensin-II on the cardiovascular system and other organs are not only determined by its concentration in the blood, but also the sensitivity of receptors.

The main mechanisms of action of angiotensin-II realized through ATI 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 (tubuloglomerulyarnyh relations);

• Increased sodium reabsorption in the tubules.

Main components of the renin-angiotensin detected not only in blood, but also in many tissues of the body where they are synthesized locally (kidneys, vascular wall, the myocardium, brain) and exhibit a local effect.

The second system, relevant to the regulation of blood pressure at the level of the kidneys, a kallikrein-kinin system. The final compounds of this system - bradykinin and its analogs - have expressed vasodilatator effect. The main components of this system are presented in the blood and all organs. Kinin system in the kidney is largely autonomous and not dependent on blood kinin system. Bradykinin from kininogen formed under the influence of the enzyme kallikrein. In the kidney, bradykinin causes vasodilation, the maximum in the inner cortex. Under the influence of increasing levels of kinins prostaglandins endoteliyrelaksiruyuschego factor (EGF), increased diuresis and natriuresis. Change sodium reabsorption may be due to vasodilation, changes in osmotic gradient of sodium in the medulla of the kidney or the direct influence of kinins on the reabsorption of sodium in the distal tubule.

Mainly at the level of the kidneys realized and regulating blood pressure effects of prostaglandins from arachidonic acid synthesized by the endothelium (prostacyclin) and fibroblast-like interstitial cells of the papilla and medulla of the kidney. Since prostaglandins are destroyed very quickly, their effect is manifested locally. At the same time, the kidneys increase regional blood flow combined with its redistribution (increasing it in the inner cortex) without significant change in glomerular filtration rate. The regulatory role of prostaglandin blood flow, apparently under minimum normal kidney function, but it increases rapidly with any change in renal function (changes in blood volume and the extracellular fluid perfusion pressure).

The logical result of renal prostaglandin is increased diuresis and natriuresis. Increased excretion of sodium and water may be due to redistribution and increased renal blood flow. Not ruled out an immediate ability to prostaglandin (PG E) to inhibit the reabsorption of sodium in the tubules.

Finally, the compounds synthesized in the kidney directly expressed natriuretic effect of dopamine is inherent. The main stimulators of synthesis and release of dopamine in the kidney are sodium and sympathetic nervous system. Selective increase in dopamine excretion in the urine is observed in natriuresis, caused by sodium load and activation cardiopulmonary mechanoreceptors (increased blood volume, the centralization of circulation).

The mechanism of action of dopamine natriuretic not been fully elucidated. Established its ability to cause vasodilation, to stimulate renal blood flow and inhibit the release of renin. Not exclude the presence of renal epithelium pas specific dopaminergic receptors, stimulation of which inhibits the reabsorption of sodium. Thus, its main effects on the kidney is an antagonist of dopamine norepinephrine and angiotensin-II. Although the position of the role of dopamine in the regulation of natriuresis and diuresis in physiological conditions is recognized by most researchers, but its mechanism of action, as well as relationships with other hormones and vasoactive substances is far from clear.

Hormones and the regulation of blood pressure

Adrenal hormones

Among the hormones of the adrenal significant impact on the regulation of the cardiovascular system have aldosterone and catecholamines (Fig. 8).

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 (Fig. 9).

Figure 8. The right and left adrenal gland

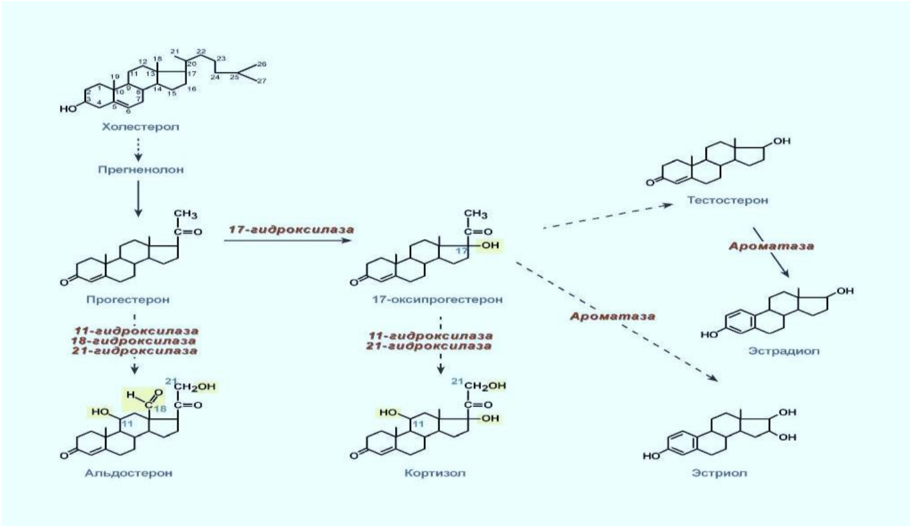


Figure 9. The formation of aldosterone from cholesterol

The main regulator is its synthesis of angiotensin-II, acting directly on aldosterone - producing cells.

Stimulate the synthesis of aldosterone and ACTH and potassium ions. The inhibitory effect of aldosterone synthesis of sodium ions is relatively small. It is mainly due to changes in the sensitivity of cells to synthesize aldosterone and ACTH inhibition of renin secretion.

Effects on blood pressure aldosterone due to its ability to increase sodium reabsorption in the distal tubules while increasing and decreasing the reabsorption of water - potassium. This leads to an increase in extracellular fluid volume and perfusion pressure. The result of these changes is the inhibition of renin secretion with a decrease in the generation of angiotensin-II, followed by a decrease in aldosterone production, increase natriuresis and the return of water and electrolyte balance to normal levels (Fig. 10).

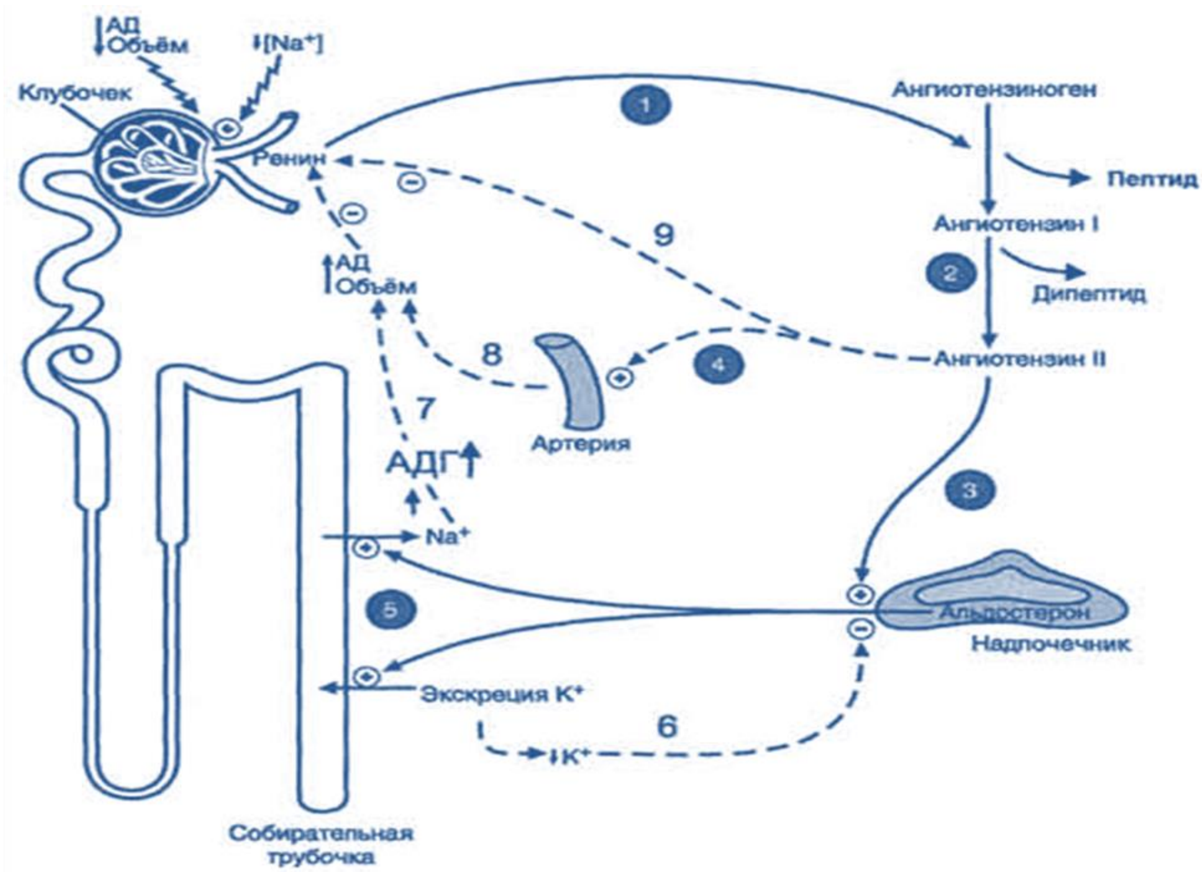


Figure 10. The interaction of aldosterone with other vasoactive substances in the regulation of blood pressure.

Other mineralocorticoid synthesized in the adrenal glands (deoxycorticosterone, 18-hydroxydesoxycorticosterone and corticosterone) mechanism of action is not substantially different from aldosterone, although their effect is expressed to a much lesser extent. As cortisol production is the main regulator of ACTH is, its effects on hemodynamics in physiological conditions and also due to low inherent weak mineralocorticoid activity, moderate, positive inotropic effect, and finally the ability to increase the sensitivity of the vascular wall to a variety of vasopressors.

Natriuretic action of progesterone, obviously, due to its ability to cause the action of aldosterone blockade at the level of the renal epithelium.

Pituitary hormones, hypothalamic area

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).

Effect of vasopressin on blood pressure due both to its direct vasopressor action, and with the ability to enhance fluid reabsorption in the distal tubule. It also potentiates the effect on the blood vessels of angiotensin-II and norepinephrine by enhancing calcium entry into smooth muscle cells. The rate of synthesis and release of vasopressin is regulated by plasma osmolarity, extracellular fluid volume and acute onset changes in blood pressure. These same mechanisms regulate the release and hypothalamic natriuretic factor. Caused by this factor natriuresis due to inhibition of sodium reabsorption in the ascending portion of the loop of Henle and distal tubule due to inhibition of Na, K-ATPase. With this mechanism, data related factor and the resulting increase in vascular resistance and of the reactivity of the smooth muscle cells in larger concentrations of calcium and sodium (Fig. 11).

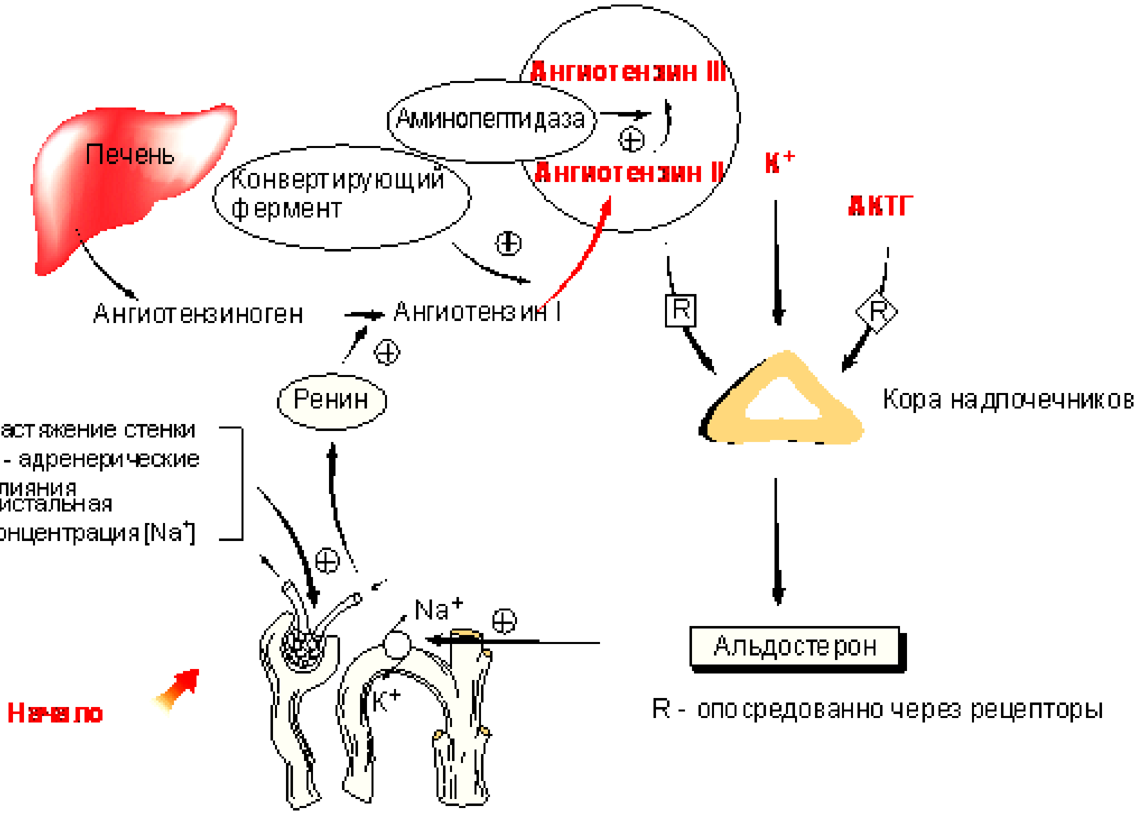


Figure 11. The renin-angiotensin-aldosterone system

The action of adrenocorticotropic hormone in the circulation of the blood due to its regulatory effect on the secretion of cortisol and mineralocorticoids, mainly aldosterone. Regulation of ACTH secretion is largely associated with downstream effects upstream of the central nervous system. Unconditional value belongs to and influence on the production of ACTH feedback is carried out mainly changes in the concentration of cortisol in the blood. Under physiological conditions, ACTH is essential in regulating the response of the cardiovascular system to emotional stress.

Hormones and other biologically active substances

Atrial natriuretic hormone (factor). Among the different factors regulating natriuresis, one of the most significant places takes atrial hormone. It is synthesized in the form of atrial cardiomyocytes prohormone. Activation hormone occurs in cardiomyocytes directly influenced by specific proteases. The main stimulant hormone receptors are atrial tension. Atrial tension (increase in the size of the cavity) stimulates the release of hormones. Its ability to stimulate the release of vasopressin and epinephrine is inherent.

The mechanism of action of hormone related mainly to its effect on the kidneys. He is the most powerful of all known natriuretics. Most researchers believe that it inhibits the reabsorption of sodium predominantly in the distal parts of the nephron and collecting ducts. This does not prevent the ability of the hormone to increase renal blood flow and glomerular filtration rate. Renal action of the hormone is partly due to the stimulation of renal kinin system. Furthermore, Atrial kidney hormone level is an antagonist of angiotensin-II. Atrial hormone has a direct vasodilating effect, reduces the sensitivity of vessels to vasoconstrictor influences and inhibits the production of aldosterone. Vasodilatation explains the hypotensive effect of the hormone.

A number of *biologically active substances* may have a local effect on vascular tone. These compounds play an important role in the regulation of local blood flow. However, in recent years, data on their possible involvement in the regulation of blood pressure through changes in vascular resistance. All of these compounds are very rapidly destroyed in the blood, which defines "local" their effects on vascular tone. These compounds include factors secreted primarily endothelium. So, secreted by the endothelium relaxation factor (nitric oxide) causes a marked relaxation of smooth muscle cells and vasodilation. Expressed local action is inherent and prostacyclin. Local influence and connections can have a clear common action. In particular, the main components of the renin-angiotensin system and kinins identified in many organs, where they can modify the flow velocity and rate of metabolism of various cells. Most clearly the role of local factors in the regulation of blood pressure is presented in the kidneys, where they can both directly and indirectly - through changes in renal blood flow and its redistribution - affect the rate of sodium reabsorption.

Ions and the regulation of blood pressure

The influence of many factors on blood pressure in whole or in part due to changes of sodium reabsorption in the kidney. The increase in sodium reabsorption and its content in the body accompanied by an increase in blood pressure. Hypertensive mechanism of action of sodium is not fully understood, although there is no doubt of its complex effects on blood pressure. First of all, an increase in plasma sodium content volume expansion accompanied with the increase of the plasma SR n elevated blood pressure. Under these conditions, increases diuresis and natriuresis, which leads to the normalization or apoplasmia normalization sodium content thereof and urinary sodium excretion. Thus, the newly established balance between intake and excretion of sodium. No less important in terms of the regulation of blood pressure is the change of intracellular sodium, largely determines the rate of sodium transport across the membranes of smooth muscle cells. This process is also controlled by a number of hormones and biologically active substances. Changes in the concentration of sodium in the cytoplasm of smooth muscle cells alter calcium concentrations and, consequently, the degree of interaction of actin and myosin. Furthermore, the degree of change sodium sympathetic nervous system activity, affects the rate of synthesis of many hormones and biologically active substances.

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 gladkomy 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.

The role of other ions in the regulation of blood pressure studied in less detail. Changes in the concentration of the absolute majority of them within the physiological values in plasma do not affect the level of blood pressure. Furthermore it is essential to update the content of certain ions in smooth muscle cells. In particular, the decrease in the concentration of free calcium in the cytoplasm of smooth muscle cells reduces the actin-myosin interaction and leads to vasodilation of resistance with a decrease in blood pressure.

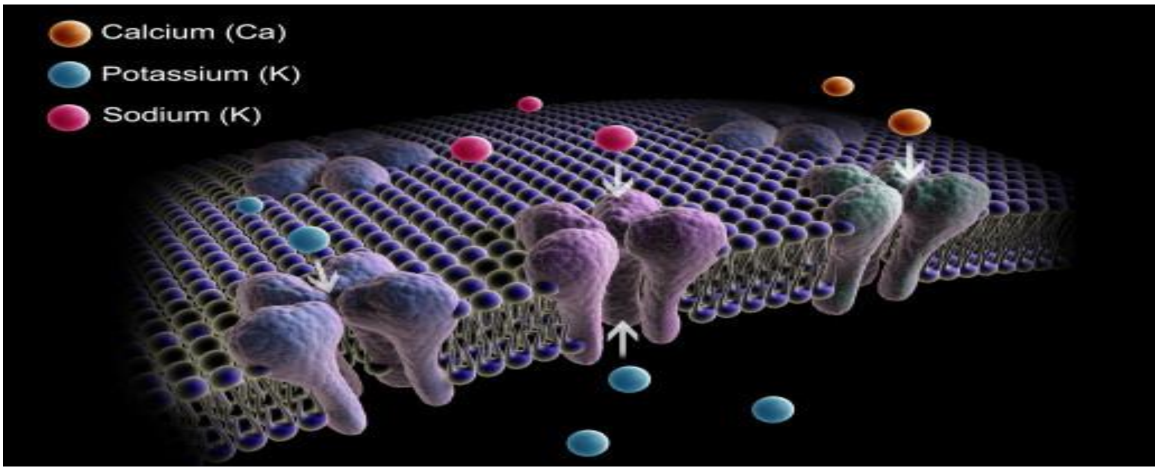


Figure 12. The increase in the potassium content in the cell

A similar effect causes an increase in potassium, which is due to membrane hyperpolarization of smooth muscle cells and decrease their sensitivity to various types of vasoconstrictor effects (Fig. 12).

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.

CHAPTER 3.
Summary of essential hypertension.

Hypertension - the main clinical syndrome of hypertensive disease, is largely determining the outcome of the disease and the prognosis of the patient. At the basis of increased blood pressure in hypertensive disease there are complex disorders of blood circulation, details of which are still far from being understood. To be sure, the pathogenetic mechanisms underlying the emergence of a pathological condition, and factors supporting BP at a consistently high level in a patient with long-existing hypertension, is not the same.

The results of clinical and experimental studies suggest that the occurrence of hypertensive disease due to a combination of genetic disorders and changes in the regulation of blood flow that occur during a person's life. Today the pathogenesis of hypertensive disease is considered as a multifactorial process, the role of various factors in raising blood pressure in different patients varies considerably. These differences occur even in the characteristics of the change of systemic hemodynamics in patients with early signs of hypertensive disease (the so-called *"soft"* or *"mild"* arterial hypertension). In general, this group of patients with a disposition to an increase in cardiac output due to an increase in the number of heartbeats in the absence of significant changes in blood volume. Peripheral resistance to blood flow tends to decrease, but this decrease can not be regarded as adequate in relation to the high values of cardiac output (Fig. 13). One of the features of hemodynamics at this stage is to increase the central (cardio-pulmonary) blood volume due to the increase in the tone of veins of the systemic circulation with a decrease in their capacity.

However, even within this stage of the disease in people with the same values of BP there is a significant range of variation of core indicators of systemic hemodynamics. Most of the patients at the heart of increasing blood pressure is a preferential increase in cardiac output (hyperkinetic type) in less - as a moderate increase in cardiac output and vascular resistance to blood flow (eukinetic type) or an isolated increase in vascular resistance to blood flow (hypokinetic). Characteristic of patients, even with the initial stages of the disease is to increase vascular resistance in the kidney with a decrease in effective renal blood flow in the absence of significant changes in the glomerular filtration rate.

In patients with initial manifestations of hypertensive disease increase in cardiac output associated with an increase in sympathetic nervous system effects on the cardiovascular system. This is manifested in the increase in the number of heartbeats and contractile activity of the myocardium. Already in this period, a decrease of vagal influences on the heart. The third factor that determines high values of cardiac index, is the increase in venous return to the heart with an increase in the volume of the central circulation.

Among the various genetic factors that determine the occurrence of hypertensive disease, the most widely discussed the role of disorders of cell membranes (Yu.V. Postnov, S.N. Orlov). In hypertensive patients revealed different membrane defects, such as increases in passive entry sodium cage anti transport speed increase "sodium-sodium" and, finally, reducing the ability of the sodium-potassium pump sodium out of the cell output.

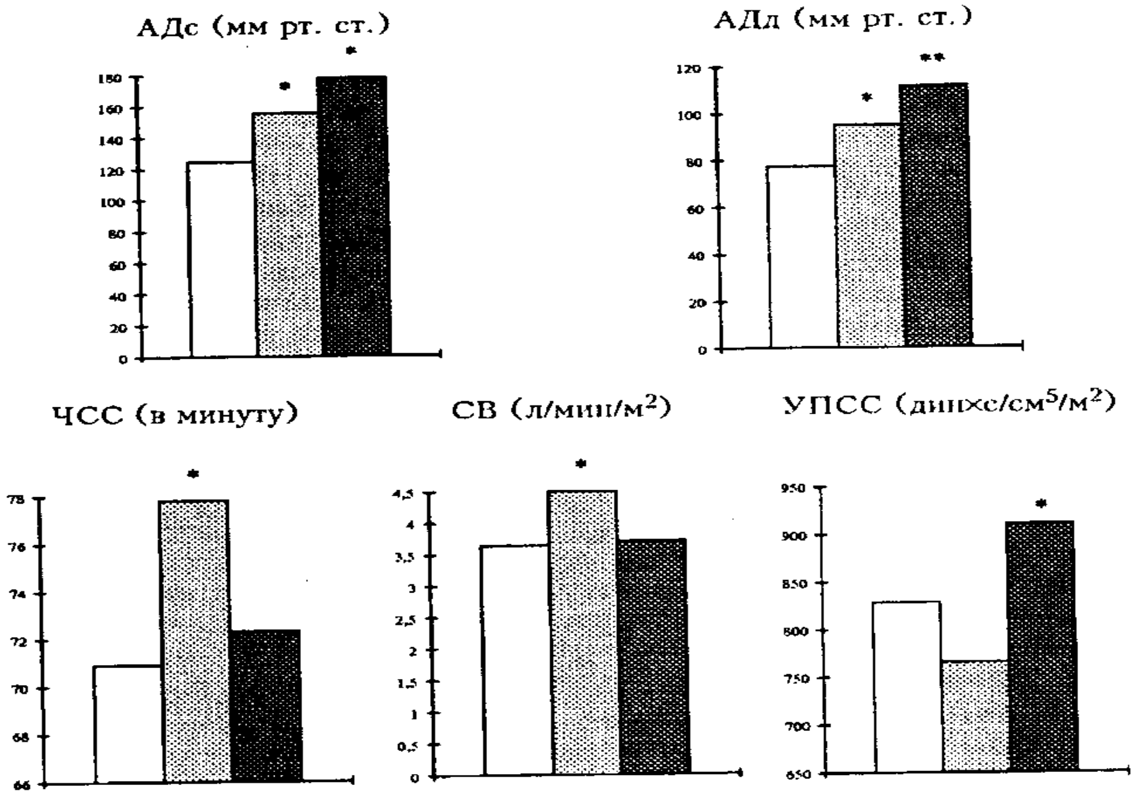


Figure 13. Indicators of systemic hemodynamics in patients with hypertensive disease. Light bars - healthy person, gray bars - GB 1 tbsp. (Mild hypertension), dark bars - GB 2 tbsp. (Moderate hypertension). Significant differences from the values in healthy subjects at $p < 0.05$.

Changes in cell membrane can also affect the state of the sympathetic nervous system, and effector organs (kidney, smooth muscle cells). In recent years, data were obtained on the importance and other genetic disorders that predispose to the development of hypertension (changes in activity of the sympathetic nervous system, the renin-angiotensin system, etc.). Genetic disorders can be seen as the background against which these or other changes in the regulation of blood circulation will lead to an increase in blood pressure.

Among the disorders of blood circulation, responsible for increased blood pressure at the stage of hypertension, the most detailed study of the role of the nervous system and kidneys.

Understanding of the role of neurogenic disorders of blood circulation in the pathogenesis of hypertension was made by clinicians (G.F. Lang, A.L.

Myasnikov). In recent years, these ideas have found and experimentally confirmed. Typical for the majority of patients with early signs of the disease is the increased activity of the sympathetic nervous system. For patients with initial stages of hypertension is characterized by a decrease in the inhibitory activity of the sympathetic nervous system influences from the baroreceptors, which may be due as amended (the restructuring) receptors themselves, and disorders of the central simpatho-ingibitors influences. At the same time decreases and parasympathetic control chronotropic cardiac function.

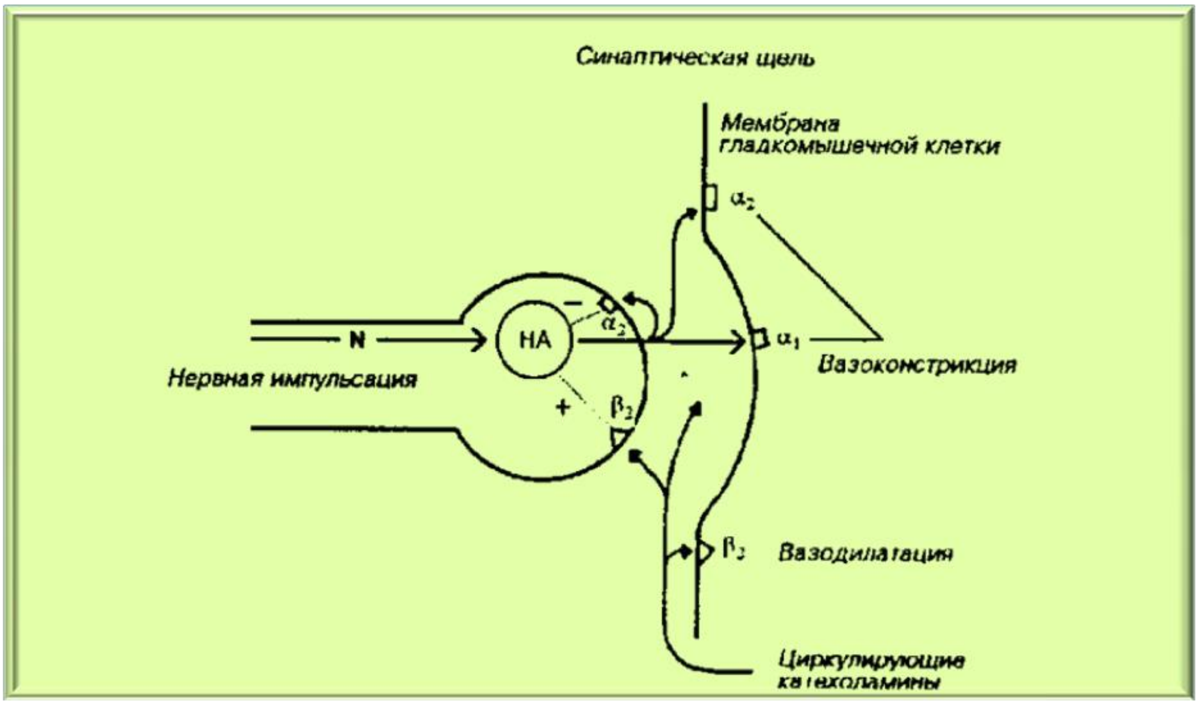


Figure 14. Scheme of adrenergic synapse.

Greater influence of the sympathetic nervous system on the cardiovascular system may be due not only to changes in impulse activity of the sympathetic nerves. The same result can cause changes in the metabolism of noradrenaline at the level of "nerve ending - synaptic cleft" (Fig. 14). These changes include a possible increased excretion of norepinephrine in the synaptic cleft during sympathetic stimulation and a violation of the mechanism of its reuptake *(Re* - Take).

At the heart of these disorders may lie as a reduction in the number of presynaptic (alpha-adrenergic receptors, which are on a feedback mechanism regulate the release of norepinephrine, as well as a decrease in the sensitivity of muscarinic receptors, inhibiting the release of norepinephrine from nerve endings.

The regulation of norepinephrine release may affect the condition of the presynaptic beta-adrenoceptor activity and the sodium-potassium pump, since it helps to alleviate the inhibition of noradrenaline release from nerve terminals.

A significant impact on the severity of the hemodynamic response during stimulation of the sympathetic nervous system has a functional state of adrenergic receptors located on vascular smooth muscle cell membranes and heart. These receptors interact with noradrenaline released from the nerve endings, so the increase in their number or sensitivity can be one of the leading factors of increasing the reactivity of vascular smooth muscle and increases the resistance to blood flow.

As for the concentration of catecholamines in the blood, most researchers found no significant changes in her, but for patients with early signs of hypertension is characterized by greater than normal rise in the concentration of noradrenaline and adrenaline in response to emotional and physical stress.

The end result of the increase of the sympathetic nervous system influence on the cardiovascular system is an increase in cardiac output and total peripheral resistance. At the heart of improving cardiac output may lie increase in the number of heartbeats, increased myocardial contractile activity and an increase in venous return blood to the heart due to the increased venous tone.

Increase in resistance to blood flow in the early stages of the disease due to increased tone of smooth muscle cells in the absence of organic changes in the vascular wall. Sympathicotonic changes noted in the cardiovascular system may be caused by indirect effects, among which the most important are the effects on the kidneys.

Changes in renal function at the stage of hypertension, primarily characterized by a decrease in Na-uretic renal function in approximately 1/3 of patients (so called salt-sensitive person). Although basal conditions in all patients sodium content in plasma and red blood cells, as well as his daily urinary excretion, no different from the norm, in salt-sensitive individuals have delayed excretion of sodium salt loads.

Plasma renin activity tends to increase, though its range of values varies within wide limits. At the stage of the disease in patients with an absolute majority of plasma renin activity is normal (normo-renin forms of hypertension), a minority of - increased (hyper-renin) or low (hyper-renin form).Characteristic of hypertensive patients is a big boost renin activity on various "provocative" load (bicycle ergometry, diuretics). The aldosterone concentration changes less regularly.

The majority of patients with the initial stage of the disease is marked activation of humoral depressor systems (increased activity of kinin system kidneys, increased synthesis of vasodilatory prostaglandins), which can be regarded as a compensatory reaction in response to an increase in blood pressure.

Changes in other hormones and bioactive substances that are relevant to the regulation of blood pressure, less natural.

Mechanisms underlying the stabilization of blood pressure and keeping it in high numbers

Patients with persistently high BP levels have lower variability of the indices as hemodynamics, and factors related to the regulation of blood pressure, compared to patients with labile hypertension. The most characteristic hemodynamic changes is a significant increase in resistance to blood flow caused not so much functional as organic changes of small arteries and arterioles of large diameter (hypertrophy of the muscle layer, increase in connective tissue, increasing the stiffness of the arteries), leading to a reduction in the inner diameter. Cardiac index and blood volume tend to decrease. Typical for patients with this group is a sharp increase in regional blood flow resistance in the kidney with a decrease in effective renal blood flow and plasma flow, renal fraction of cardiac output. As the progression of hypertensive disease reduced glomerular filtration rate and an increasing number of patients, delaying sodium salt loads.

Organic changes in the blood vessels and are the main reason that determines the stabilization of blood pressure in high numbers. As the progression of morphological changes in the kidneys and decrease their ability Na-diuretic increases and the role of the kidney in the maintenance of arterial hypertension (the so-called renal hypertension).

Some contribution to the maintenance of blood pressure values at high numbers can make and regulatory violations. The most significant of these are the reduction of baroreceptor sensitivity, decreased activity depressor humoral systems (kinins, vasodilatory prostaglandins, endothelial derived relaxing factor) in the relative prevalence of pressor such as aldosterone.

Pathogenetic heterogeneity of hypertension

As has been pointed out, for hypertensive patients in the stage of the disease characterized by large individual variations major hemodynamic variables and factors that regulate blood circulation. These data were the basis for the understanding of the pathogenesis of the disease heterogeneity (Paige), and attempts to highlight its different options.

There are currently quite clearly outlined one version (form) - hyperrenin hypertension. Characteristic for this group of patients is hemodynamic option hemodynamic changes, the tendency to increase in weight of the extracellular fluid (volume depending hypertension) and sodium content, low plasma renin activity blood at normal or slightly elevated values of aldosterone. Histological examination often reveals small nodular adrenal hyperplasia. Patients in this group features high sensitivity to salt. Most often, this option course of hypertensive disease observed in women during menopause and postmenopausal. In addition, often in the progression of hypertension takes on the attributes of a typical hyperrenin. Pathogenesis of hyperrenin form of hypertensive disease has not yet been clarified. Some researchers have linked it to the primary defect of the kidneys (the inability to adequate amounts of sodium excretion from the body), the other - with increased mineralocorticoid adrenal function or insulin resistance syndrome.

The solution to all these issues, as well as the appropriateness of assigning different pathogenetic types of hypertension, it will be possible only with the expansion of knowledge about the mechanisms of regulation of blood circulation.

CHAPTER 4.
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). So, hypertension in endocrine diseases usually associated with increased production of a hormone. When Conn's syndrome (primary hyperaldosteronism) - aldosterone, with Cushing's syndrome - cortisol in pheochromocytoma - catecholamines, etc.

More complicated is the pathogenesis of hypertension in diabetes, where it is based on the development of organic changes in the kidneys (diabetic glomerulosclerosis). Some importance in raising blood pressure and hyperinsulinemia may be the most common in the so-called non-insulin-dependent diabetes mellitus. The hypertensive insulin action may be due to its ability to stimulate the sympathetic nervous system, and also directly increase sodium reabsorption in kidney.

When hemodynamic arterial hypertension increase in blood pressure due to changes in hemodynamics - the redistribution of blood flow to the sharp increase in its upper half of the body (coarctation of the aorta).

Iatrogenic arterial hypertension associated with the reception of certain drugs (steroids, oral contraceptives et al.).

Not fully elucidated the pathogenesis of hypertension in organic lesions of the nervous system. Apparently there is a critical mechanism activation of the sympathetic nervous system.

More complicated is the pathogenesis of hypertension with renal disease. Stenosis of the renal artery in the early stages of the disease a major factor increasing blood pressure is the activation of the renin-angiotensin system, due to a sharp increase in renin production of ishemized kidney. Later, with the development of morphological changes in the kidney, including the contralateral, begins to form renal mechanism (weight reduction of functioning nephrons) and depressor sharply reduced kidney function (impaired synthesis of kinins, renal vasodilatory prostaglandins and dopamine). All this leads to disruption of excretion of sodium and increase its content in the body.

Table 2. The main groups of symptomatic arterial hypertension

Group	Disease
Nephrogenic hypertension • parenchymal kidney disease • renovascular (Violation of blood flow in the main renal arteries) • violation of the outflow of urine	• glomerulonephritis • pyelonephritis • polycystic • diabetic glomerulosclerosis • tumors • Tuberculosis • atherosclerosis • fibromuscular dysplasia • thromboembolism • aneurysm, fistula • hypoplasia • nephroptosis, hydronephrosis,

	ureterohydronephrosis, compression of the urinary tract • reflux nephropathy
Endocrine hypertension	• primary hyperaldosteronism (Conn's syndrome) • disease and Cushing's syndrome • pheochromocytoma • acromegaly • thyrotoxicosis • syndrome of excessive production of deoxycorticosterone
Hemodynamic hypertension	• atherosclerosis of the aorta and carotid arteries • coarctation of the aorta • nonspecific aortoarteriitis • atrioventricular block
Hypertension with organic lesions of the nervous system	• diencephalic syndrome • brain tumor • encephalitis • meningitis • polyneuritis
Hypertension caused by intake of drugs (iatrogenic)	• Glucocorticoids •oral contraceptives • Erythropoietin • cyclosporine

The pathogenesis of arterial hypertension in parenchymal renal disease is not fully studied. Increased blood pressure is associated with stimulation of the intrarenal renin-angiotensin system and the decrease in renal function depressant. As the progression of renal scarring increasingly important in maintaining blood pressure gets high figures renal mechanism.

CHAPTER 5.
Renovascular hypertension, diagnosis and surgical treatment.

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 hypertensions.

The basis of renovascular hypertension is always a one- or bilateral renal artery constriction of any one or more of its major branches (Fig. 15, 16). As a result, through the artery to diseased narrowed opening into the kidney is supplied per unit time is less than blood. This leads to the development of renal tissue ischemia, the severity of which depends on the degree of stenosis of the affected artery.

Figure 15. Atherosclerotic plaque in the lumen of the artery

Figure 16. 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 (Fig. 17).

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.

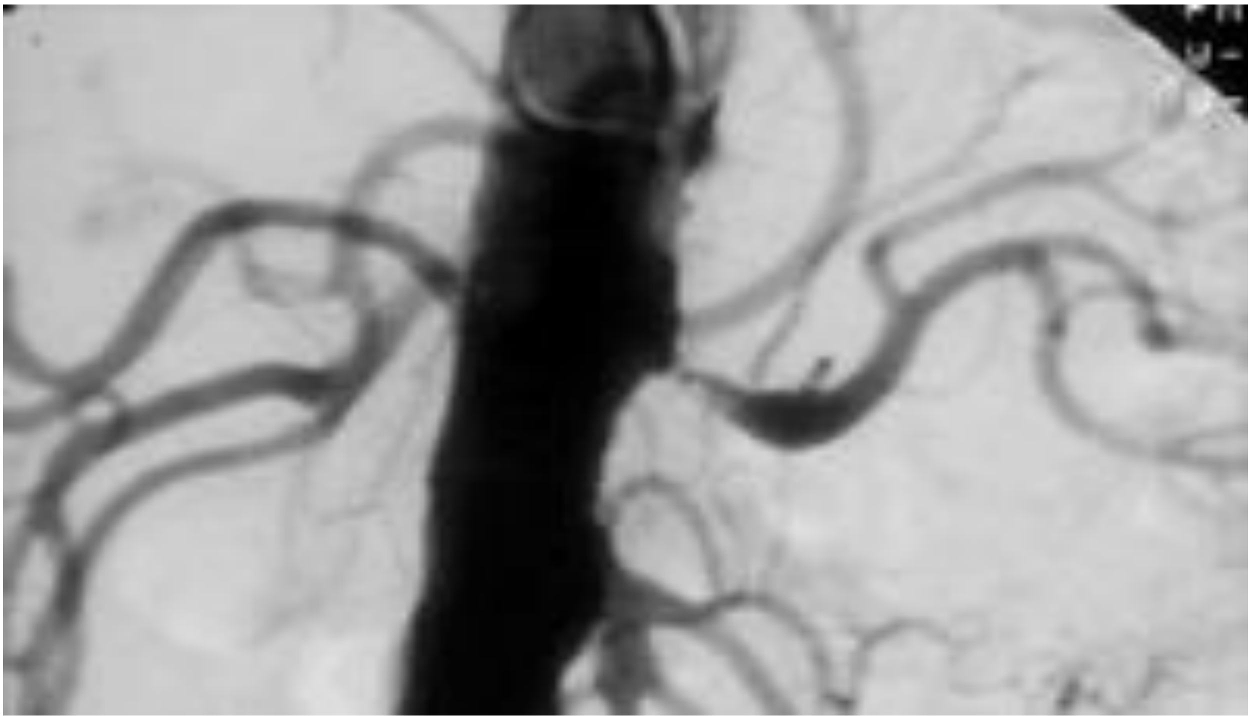


Figure 17. Stenosis of the left renal artery

Renovascular hypertension may occur due to compression of extravasal renal artery (Fig. 18), resulting in thrombosis or embolism of the renal artery, aneurysm formation, the main renal artery hypoplasia, nefroptosis, tumors, cysts, and kidney malformations al.

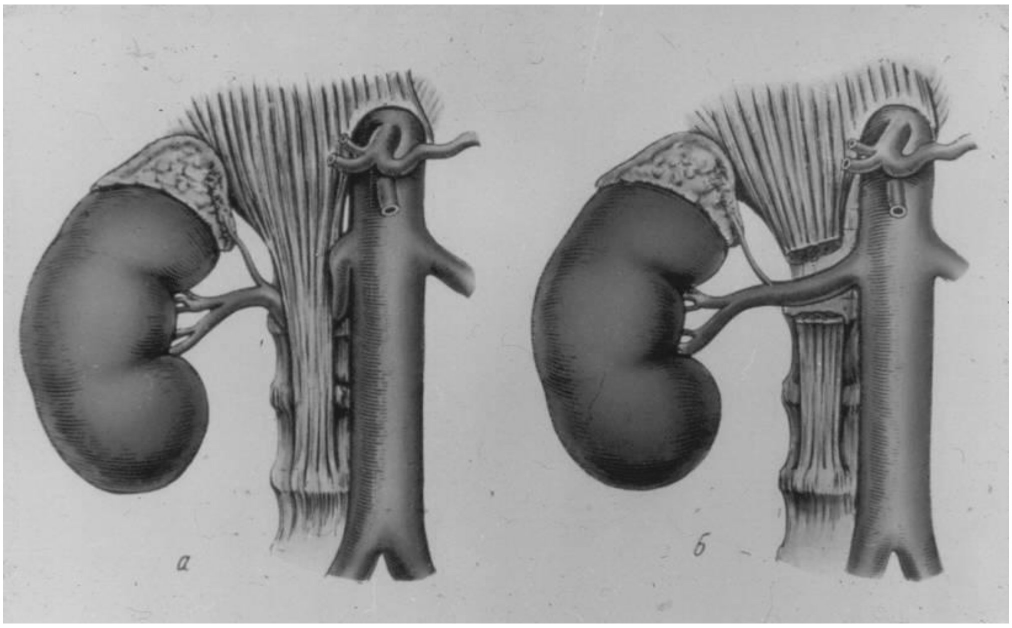


Figure 18. Compression of the renal artery leg aperture and decompression after crossing the last

Narrowing or occlusion of the renal artery leads to a reduction in renal blood flow and perfusion pressure reduction. Development of renal ischemia leads to hyperplasia of juxtaglomerular cells apparatus (JGCA), resulting hypersecretion of renin. Renin (this - enzyme) coming from the liver converts angiotensinogen angiotenzin I in which, under the influence of the angiotensin converting enzyme in transformed angiotenzin II. Angiotenzin II - one of the most powerful vasoconstrictor, which is directly affecting the systemic arterioles, causing them to spasm and dramatically increases peripheral resistance. In addition, angiotensin-aldosterone stimulates the adrenal cortex, resulting in the development of secondary hyperaldosteronism, with sodium and water retention. Peripheral vasoconstriction, hypernatriemia and hypervolemia exacerbate hypertension.

To the natural flow of atherosclerotic RVH characterized by progressive decline in renal blood flow, which ultimately leads to a complete loss of kidney function ("ischemic nephropathy"). This disease is manifested in the middle or old age. On the contrary, fibromuscular dysplasia usually manifests at a young age, is more common in women who did not have progressive course and rarely leads to ischemic nephropathy.

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.

Diagnostics. For the diagnosis of these important 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 the aorta

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 above features allow only suspect assume renovascular hypertension, often quite reasonable, but they are not allowed to fully confirm this diagnosis. To confirm or exclude the diagnosis of renovascular hypertension more research is needed. 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. In particular, high sensitivity have scintigraphy with ATE inhibitors (Figure. 19), duplex scanning of the abdominal aorta and the renal arteries (Fig. 20), magnetic resonance angiography and computed tomography, and they can be used separately or in combination to achieve adequate screening patients to conventional angiography or revascularization.

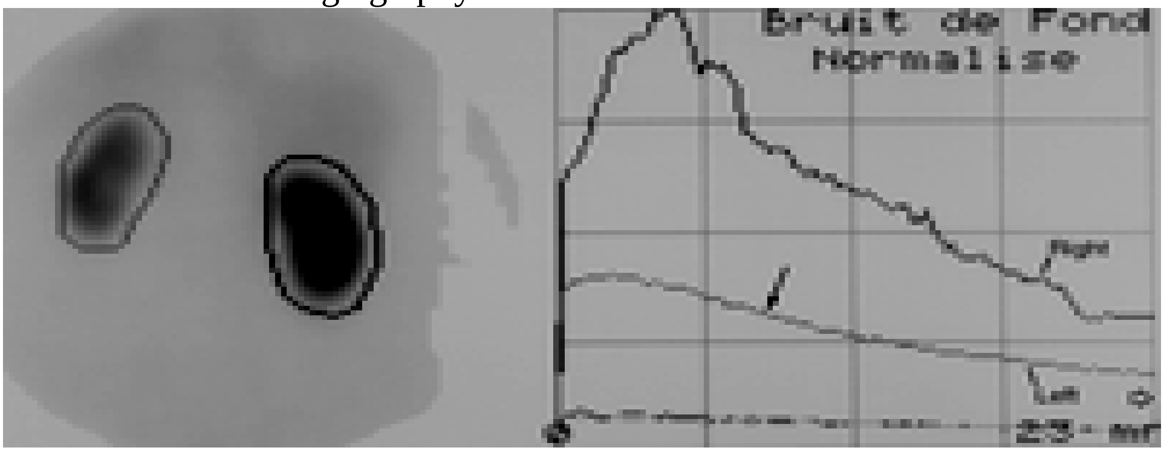


Figure 19. renoscintigraphy kidney

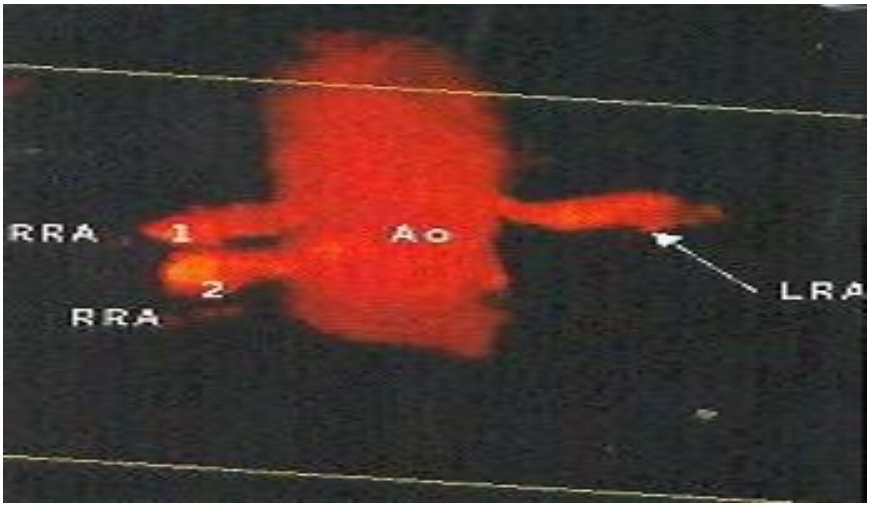


Figure 20. Three-dimensional duplex scanning (shown marked stenosis of the left renal artery and right renal artery extra).

Renoscintigraphy with inhibitors angiotensin-converting enzyme (ACE). The use of ATE 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 a characteristic changes renogrammy.

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 changes.

These criteria are:

1. Normal stsintigrama using ACE inhibitors indicates a low probability of VRH, 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 scintigraphic curve decreases in the grip, 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.

Four criteria are used for the diagnosis of significant proximal stenosis or occlusion of the renal artery:

1. Increase in the maximum systolic velocity in the renal artery (in the literature, the threshold for significant renal artery stenosis - 100-200 cm / sec);
2. renal aortic ratio of maximum systolic velocity greater than 3.5;
3. Turbulent flow in poststenotic region;
4. Visual observation of the renal artery without detectable Doppler signal, which indicates that occlusion.

In addition, with the help of ultrasound can detect indirect signs of RVH, in particular - the reduction in the size of the kidneys due to ischemic atrophy of it. Kidneys of less than 7-8 cm is usually heavily damaged by ischemia. In these circumstances, revascularization usually does not lead to recovery of function or hypertension, and patients shows nephrectomy.

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 (Fig. 21)

Figure 21. Magnetic resonance tomography angiography of the renal arteries and asthma.

Multislice computed tomography angiography (MSCTA). multislice computed tomography angiography (CTA) 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 (Fig. 22)

The first step in diagnosing renovascular hypertension is a clinical diagnosis and selection of patients with moderate to high probability of this disease on clinical criteria. Non-invasive screening tests provide the impact the selection of patients with a high probability of renal artery stenosis, thereby reducing the frequency of the potential side effects of X-ray angiography with its wide use. In patients with a high probability of the disease should be taken X-rays to determine the intended renal artery stenosis. Spiral CT can provide excellent visualization of the renal vessels, but requires a lot of contrast. Currently MRA gives good image of the renal vessels without risk to the patient.

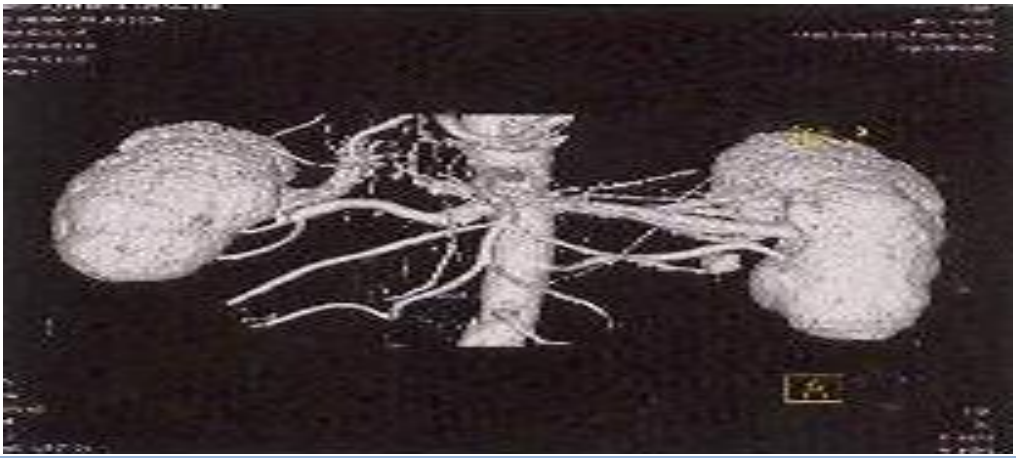


Figure 22. multislice computed tomography angiography of the abdominal aorta and its branches

But, with its higher cost and lower availability, it should be reserved for patients with indeterminate results functional image, but a high clinical suspicion of VRG, and patients who have contraindications to standard angiography: renal failure or allergy to iodine preparations

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 CHTPA or stenting (Figure 23).

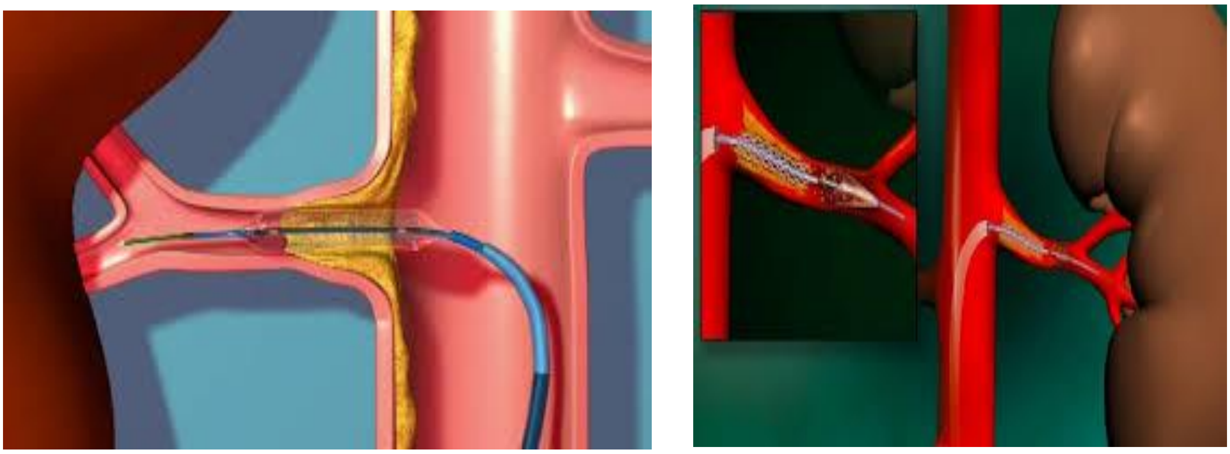


Figure 23. Dilation and stenting of the renal arteries

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 CHTPA 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.

CHAPTER 6.
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, androsteroma, kortikoesteroma. 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. First of all, for this disease is characterized by marked electrolyte disturbances. Aldosterone affecting tubules leads to a decrease in potassium and water reabsorption, and conversely, to increase reabsorption of sodium. Increased urinary excretion of potassium leads to the development of hypokalemia (less than 3.0 mmol/l.) of potassium ions in the cell are replaced by sodium ions and hydrogen. Reduced sodiumuresis increases the content of sodium ions in the extracellular space and inside the cell sodium ion keeps being hydrophilic and attracts water. As a result of edema of tissues, especially vascular wall, decreasing its inner lumen at the level of arterioles, increased vascular tone and peripheral vascular resistance, and hypertension develops.

The disease most often affects women. Symptoms of aldosteroma can be divided into 3 groups:

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

Neuromuscular symptoms are caused by hypokalemia and associated with disorders of neuromuscular conduction. Patients complain of severe muscle weakness, the degree of which varies - from fatigue to flaccid paralysis, covering most of the leg muscles. It is often observed paresthesia and cramps.

Among renal symptoms most frequently observed polyuria, nictury, gipostenuriy. In connection with the loss of large amounts of fluid in the urine develops a thirst.

Hypertension - the main, sometimes the only symptom aldosteroma. During hypertension usually stable. The level of increase in blood pressure ranges from mild (160/100 mm Hg) to severe (220-250/120-140 mm Hg). Most patients complain of severe headaches, which are caused by high blood pressure. Hypertension leads to severe left ventricular hypertrophy on electrocardiogram showing signs of hypokalemia. Very often, a vascular lesion of the fundus with an impaired vision.

Diagnosis is based on analysis of clinical manifestations and laboratory data. Radioimmunoassay reveals an increase in plasma aldosterone concentration in basal conditions and its paradoxical decrease after the test with a 4-hour walk, a decrease in plasma renin activity. Biochemical studies reveal hypokalemia, hypernatremia. Certain diagnostic value may have alkaline reaction of urine. Among the instrumental methods are important ultrasound and CT. Due to the fact that aldosteroma have small dimensions (1.5 cm -2) by means of ultrasound can reveal approximately 60% of patients. The most accurate method of diagnosis is computed tomography. CT revealed the formation of low density (12-14 units. Hn) (Fig. 24).

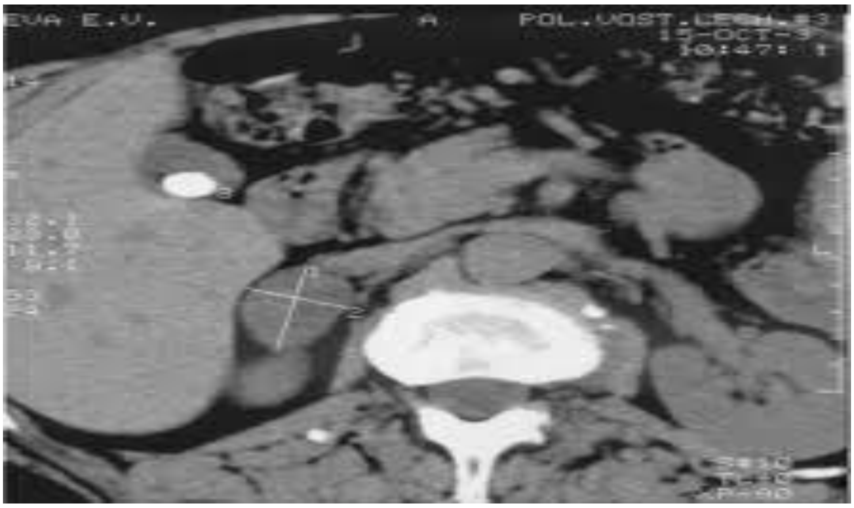


Figure 24. aldosteroma of left adrenal gland

Treatment: surgical - adrenalectomy

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). The tumor may be single or multiple, benign and malignant. The disease most often occurs in middle age men about equally often. There are reports of familial pheochromocytoma.

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. Catecholamine levels during the crisis, particularly norepinephrine, is ten times higher than normal, and their excess causes stimulation of alpha- and beta-adrenergic receptors, which leads to a marked spasm at the level of arterioles and a sharp increase in total peripheral resistance, thereby increasing both systolic and diastolic blood pressure.

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. In paroxysmal hypertensive crises are marked with an increase in blood pressure up to 250 - 300 mm Hg or higher. The sudden increase in blood pressure accompanied by sharp headaches, palpitations, fear of death, chills, fever, sweating. Often marked shortness of breath, pain in the lumbar region, in the abdomen, behind the breastbone. There may be nausea and vomiting. Stroke duration from a few minutes to several hours. For catecholamine crisis characterized hyperglycemia, and glucosuria. BP crisis is normal and diseased no complaints.

When a stable form of hypertension observed a persistent increase in blood pressure without crises. When mixed form catecholamine crises observed against the background of high blood pressure (160/100-180/120 mm Hg). Undocked catecholamine crisis can lead to death, that may be caused acute heart failure, pulmonary edema, bleeding in the brain.

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). Hyperproduction of norepinephrine and increased urinary excretion of the hormone at normal concentrations of adrenaline are usual for no suprarenal gland tumor localization. The simultaneous increase in the concentration of both hormones in the urine is more characteristic of adrenal tumor localization in practice often used to determine the concentration acid in urine. This acid is a metabolite of both hormones, and its concentration in urine is several tens of times higher than the concentration of epinephrine and norepinephrine. For typical pheochromocytoma significant increase in concentration of vanillylmindal acid in urine. Given the large size of the tumor, they can easily be identified by ultrasound and CT (Fig. 25).

Feohrotsitoma is treated only surgically - removing of the tumor (feohromotsitoma).

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).

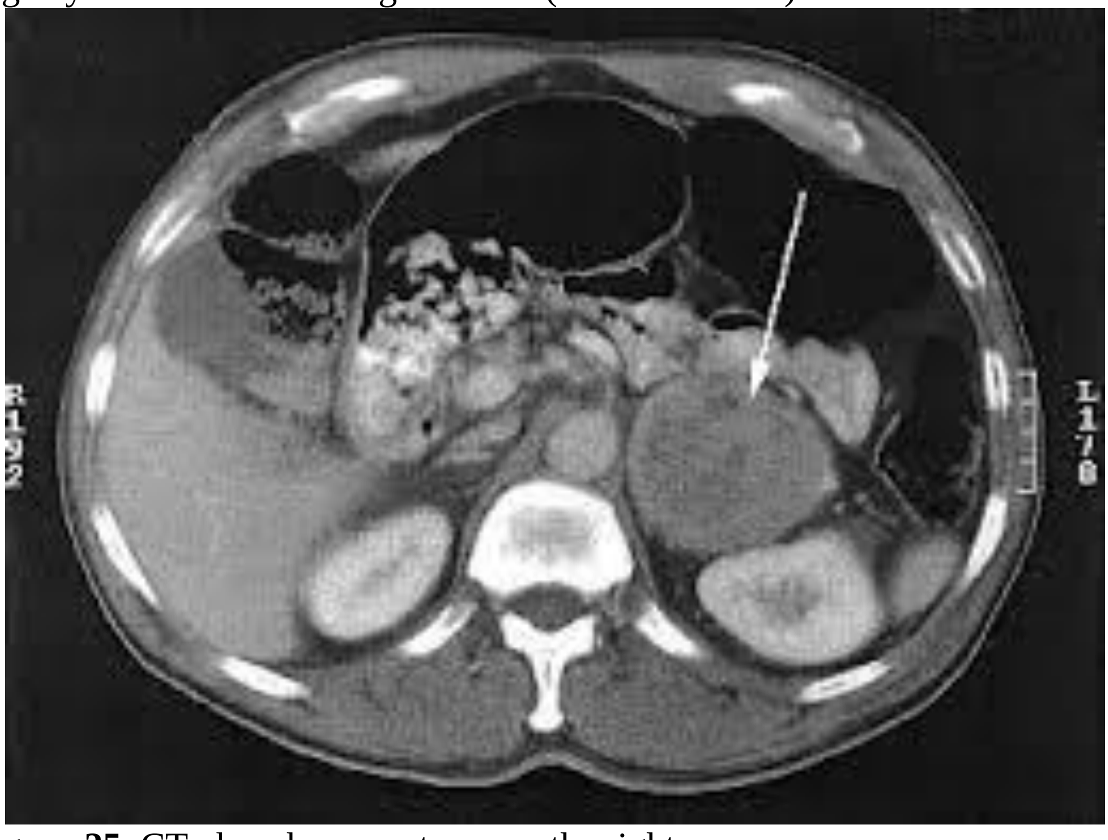


Figure 25. CT pheochromocytoma on the right.

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 (Fig. 26).

Figure 26. 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) along 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 hypertrihos, 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.

CHAPTER 7.
METHODS OF INTERVENTIONAL RADIOLOGY IN THE TREATMENT OF HYPERTENSION.

Method of endovascular occlusion of the venous network of the adrenal gland

In determining the increase in adrenal up to 3 cm, it is advisable to use a mini invasive surgical treatment, namely coagulation adrenal central vein (Figure 27).

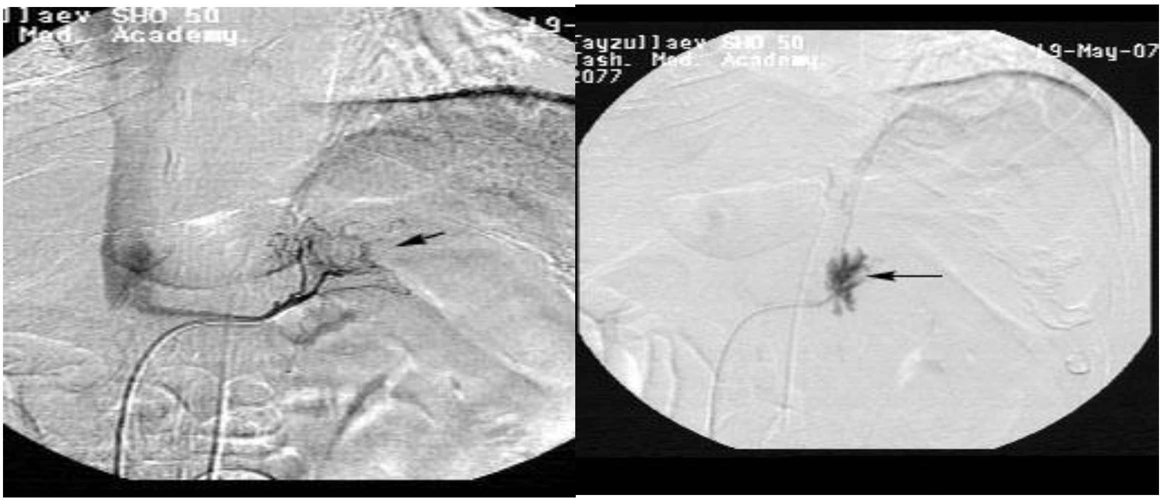


Figure 27. The left adrenal phlebography with electrocoagulation of the central vein of the left adrenal gland - a method of diagnosis and treatment of tumors of the left adrenal gland up to 1 cm and its hyperplasia.

Endovascular occlusion of the central vein of the adrenal gland and its tributaries is transcatheter occlusion of the venous system of adrenal glands.

As an embolic agent in the left adrenal vein EO can be used sclerosing agent "trombovar." 10-15 minutes after the test is performed EO venography, which is usually determined by the occlusion or severe reduction of blood flow to the adrenal-phrenic trunk, as well as flush contrast agent into the left renal vein or inferior vena.

Endovascular electrocoagulation technique adrenal central vein.

The procedure is performed by femoral access. Central venous catheterization of the adrenal gland and its endovascular occlusion produced a high frequency current on angiographic guide. We used the standard method proposed by EB Mazo in 1990

The methodology of the acute occlusion of venous adrenal combined with electrocoagulation adrenal central vein

After venography and evaluation angioarchitehtonics of adrenal gland quickly injected 5.0 ml of a 3% solution trombovar and produce electrocoagulation adrenal central vein. Hemostasis is achieved by pressing a femoral vein.

Technique of endovascular dilatation and stenting of renal artery stenosis

Endovascular dilatation (ED) of the renal arteries is performed under local anesthesia in most cases by femoral and less - radial access. Catheterization of the renal artery is used catheter "Cobra" diameter 6F. After catheterization to patients administered 5000 units of heparin, and after that through the catheter into the renal artery of the J-way wire. Then poststenotic part through a wire inserted catheter "Cobra" and the introduction of a small dose of contrast medium is controlled by the position of the tip. Then reintroduced guide catheter is removed, and in the remaining area of the stenosis in the handler introduces the balloon catheter.

By femoral access is used two ways of ED. The first time you perform a selective catheterization of the affected renal artery, and the catheter is introduced in poststenotic part of a metallic conductor. After that, the conductor left in poststenotic part of the artery, producing selective replacement of the standard double-lumen balloon catheter on. In the second method instead of the standard use of selective catheter guiding catheter, which is positioned at the mouth of the affected artery and through the inner lumen of the guiding catheter is introduced into the zone of stenosis balloon dilatation catheter.

After the establishment of the catheter in the area of stenosis administered intraarterially 5000-15000 units of heparin and expands the balloon diluted contrast agent under a pressure of 5-6 atm. until disappearance of indentations in the walls of the container, the hourglass type. To achieve success in the dilation of stenosis may require 3-5 minutes. expanding the balloon. Gradient residual is then measured in the region of stenosis. With a high residual gradient dilation is repeated. Do not always achieve full recovery vessel diameter, and even more so-called cosmetic dilation. Better and safer to focus on the change of the pressure gradient in the area of arterial stenosis. After complete disappearance of the pressure gradient or gradient residual is not more than 20 mm Hg. Art. Control angiography should be performed. It is necessary for documenting the results of dilatation, and to identify possible complications (dissection of the intima, embolization, thrombosis branches of the renal artery, renal infarction) whose symptoms do not always appear immediately except thrombosis and renal infarction. During the procedure, administered heparin and intravenous drip - low molecular weight dextran (reopolyuglukin, reoglyuman) at a dose of 400 ml with 2-4ml Glockenspiel.

Endovascular stenting of renal artery stenosis (Fig. 28).

Figure 28. The left renal artery stenting. The arrows indicate the site of stenosis before and after stenting.

After balloon dilatation with indications for stenting in the conductor at the mouth of dilated renal artery angiographic catheter selectively established, through which the dilation of the area is carried straight or J-shaped conductor with diameter 0.20 Inch. length - 180-200sm. Through a wire replacing diagnostic catheter to guide catheter diameter 8F, which is set at the mouth of the RA. Then, the appropriate size of the stent is delivered to the site of implantation through the guiding catheter. After the establishment in the area of stenosis stent balloon deals with diluted contrast medium under a pressure of 5-6 atm. to the disappearance of impressions on the walls of the cylinder under x-ray control. Then the balloon catheter is removed, and the test is performed angiography.

CHAPTER 8.
LAPAROSCOPIC ADRENALECTOMY.

Adrenalectomy - removing of the adrenal gland. First K.Thornton (1889) reported the removing of a large adrenal education. Later, S.P. Fedorov in 1912 removed the adrenal gland in a patient with renal tumors that growed in the gland. And already in 1920 A. Bryuning purposefully performed adrenalectomy. Thus, the targeted removing of the adrenal glands began at the beginning of the twentieth century. If we trace the dynamics of the implementation adrenalectomy during the last century, the celebrated passive dynamics of the flesh to the 1960s of the twentieth century. Starting from the 1970s, with the advent of ultrasound and computed tomography, perform adrenalectomy quickened, also expanded the indications for this type of surgery.

A significant event was the performance of laparoscopic adrenalectomy in 1992. On the first execution endosugical adrenalectomy by transabdominal surgical access reports Gagner M. In 1996, Russia was performed first laparoscopic adrenalectomy by Emelyanov S.I.

In 2 - clinic ofTMA in 2009 for the first time laparoscopic adrenalectomy was performed.

To date, laparoscopic adrenalectomy indications are:

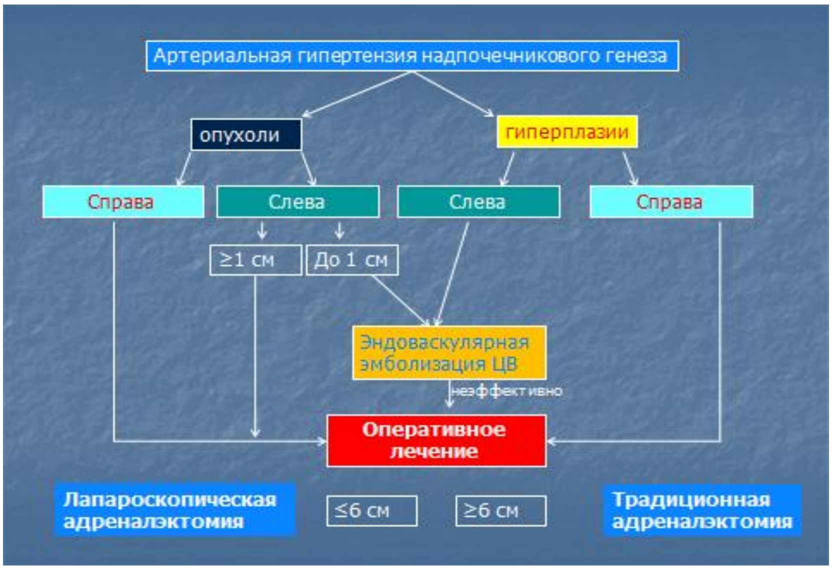
 hormonally active adrenal tumors;

 hormonally inactive adrenal tumors (intsedentalomy).

Contraindication for laparoscopic adrenalectomy is considered to be age, cardiac and pulmonary pathology, obesity, disorders of hemostasis, and most importantly, the large size of the tumor of the adrenal glands and possible malignancy.

In 1996 HenryJ.F. reported in the journal “Annals of Endocrinology” that laparoscopic adrenalectomy is not indicated for tumors greater than 6 cm. The same author in 1999 in the journal “Langenbecks Arch Surgery” publishes an article where there is the position that laparoscopic adrenalectomy can be performed with tumor in sizes up to 12 cm and at reasonable suspicion for malignancy .

On the basis of experience and based clinics TMA-2, as well as carrying out research work at the department faculty and hospital surgery medical faculty of TMA, the algorithm management of patients with hypertension adrenal origin (Fig. 29)



- **Figure 29.** Algorithm for the treatment of patients with hypertension adrenal origin.
-
- To identify pathology, the following research methods, and the binding are:
- - ACTH;
- - Cortisol;
- - Aldosterone;
- - Renin in blood;
- - Ultrasound;
- - MSCT.
- To clarify the diagnosis, suspected secondary adrenal hyperplasia, you must determine the level of these hormones and bioactive substances:

- - Parathyroid hormone, calcitonin;
- - Insulin, C-peptide, gastrin;
- - Chromogranin A;
- - CEA, IL-6 in blood;
- If necessary, diagnostic methods are also used, such as:
- - MRI;
- Selective venography;
- - Lower cavagraphy;
- - MSCTA.

Ultrasound examination reveals an increase in the size of the adrenal glands from 1 cm to continue (Fig. 30).

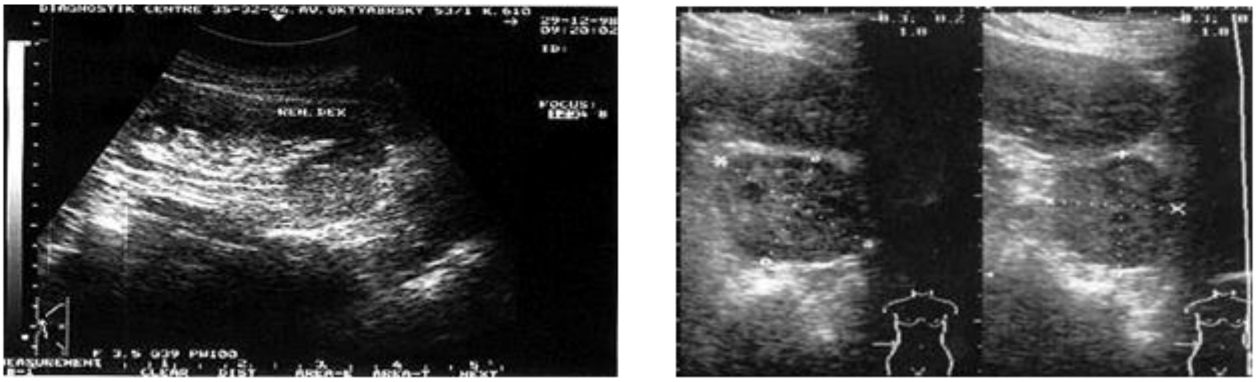


Figure 30. The ultrasound picture enlarged adrenal glands in patients with hypertension.

As already mentioned MSCT is required detection method that allows to identify the size of the enlarged adrenal gland, and allows you to define the structure, and the density (Fig. 31).

Location enlarged adrenal gland, is the cause of hypertension, is the postulate definition of tactics planned treatment.

So in our practice in 55% of cases, the location of the enlarged adrenal gland was the left.

The dimensions of the structures can be varied from 2 to 9 cm.

Histological studies show that, in 34% of cases revealed adenoma in 28% of cases aldosteronoma, 17% of pheochromocytoma, 15% of the cyst of the adrenal gland and in 5-6% of cases of adrenal hyperplasia.

Figure 31. MSCT increase picture right adrenal gland.

When you delete a large adrenal which caused hypertension taken to perform transabdominal access. Despite the wide spread use of this access, the latter has a number of advantages and disadvantages.

The reasons for rejection of other surgical approaches is:

Translumbal access:

□ significant limitation of the width of the surgical field (cavity)

□ angle of inclination of the axis of the operational activities is less than 45 °

□ overlap vascular structures adrenal tissue

Peritoneal access:

- lack of clear anatomical landmarks, which requires additional use of intraoperative ultrasound guidance
- overlap vascular structures adrenal tissue

Particularly important position of the patient on the operating table. The patient is in the lateral position, the opposite side of the tumor location, with a bend at the waist and slightly bent at the knees.

Under the contralateral lumbal region is enclosed roller, and the operating table is bent, thus increasing the distance between the iliac crest and costal arch (Fig. 32)

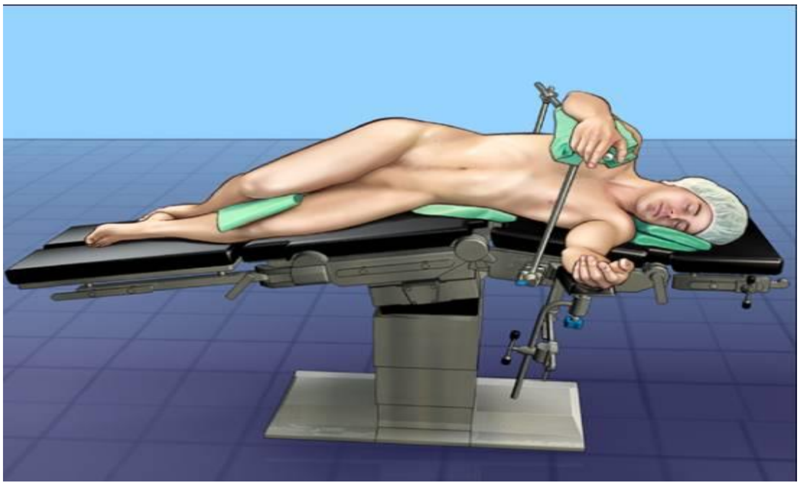


Figure 32. The position of the patient during laparoscopic adrenalectomy right.

Placing the surgical team is considered important for the operation, as well as to achieve adequate surgical field and the interaction of the joint (Fig. 33).

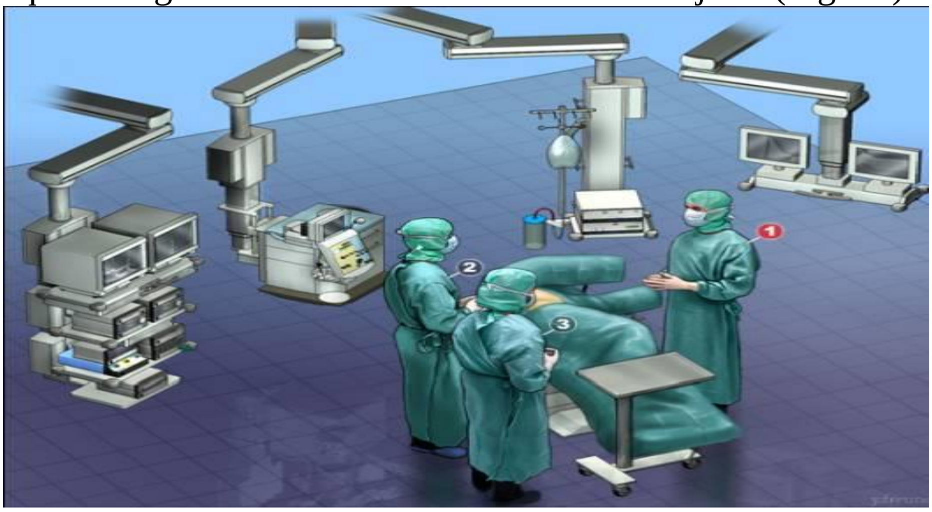


Figure 33. Location of surgeons during laparoscopic adrenalectomy.

Next superimposed pneumoperitoneum. Pneumoperitoneum was applied in the hypochondrium for safe procedure (Fig. 34).

Figure 34. pneumoperitoneum during laparoscopic adrenalectomy.

Further operative technique allows optimal access to the adrenal glands. In this example, a first stage of the operation (Fig. 35).

Figure 35. The first phase of the operation

- The dissection of the peritoneum in the field of obstructive;
- The retraction of the liver in the cephalic direction;
- Branch triangular ligament of the liver can achieve the necessary degree of mobilization.

When sided laparoscopic adrenalectomy is necessary allocation of adrenal central vein and the inferior vena cava, in fact this is the second stage of the operation (Fig. 36).

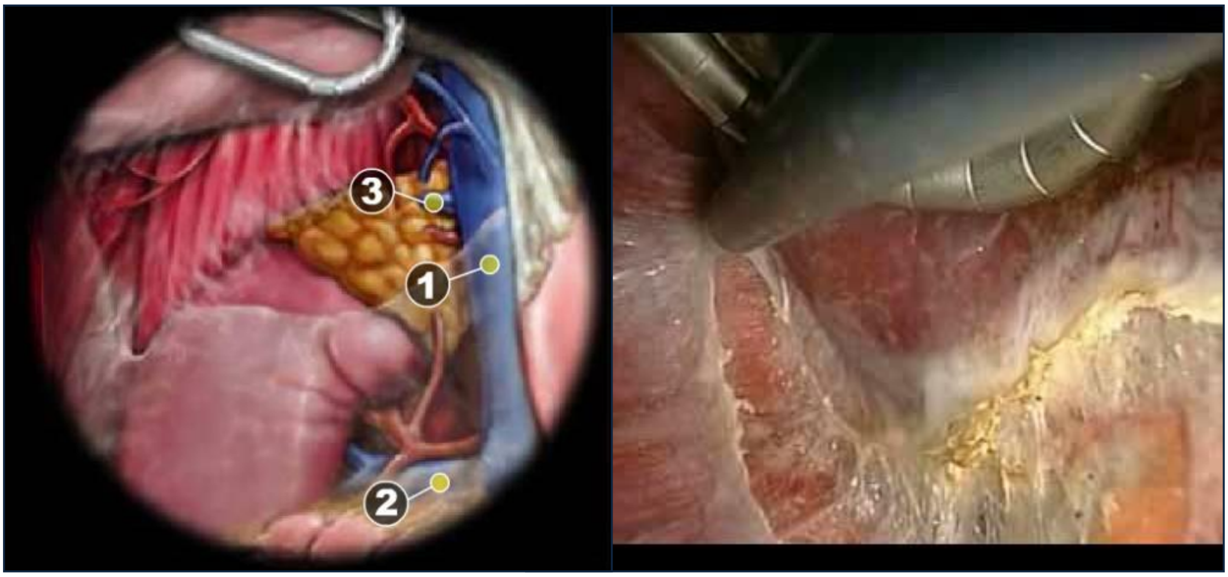


Figure 36. Mobilization of liver allows identification of the inferior vena cava

To identify the adrenal central vein inferior vena first in the caudal, and then in cephalic direction - the first vessel, identify with this dissection - central Vienna adrenal gland.

The situation with the left adrenal gland is quite different, which may cause some technical difficulties in the allocation of the latter. This is due to the high position of the left adrenal gland and spleen to the location immediately. (Fig. 37).

Figure 37. The retraction of the spleen in the medial direction while crossing the phrenic-splenic and renal-splenic ligament. Features access provisions of the patient's body provide passive exposure creation of the surgical field.

As in the case of the right adrenal gland, necessary to select the adrenal central vein (Fig. 38).

Figure 38. To identify the adrenal central vein dissection is performed in the direction of anterolateral medial edge of the left kidney.

After highlighting the lastis clipped (Fig. 39).

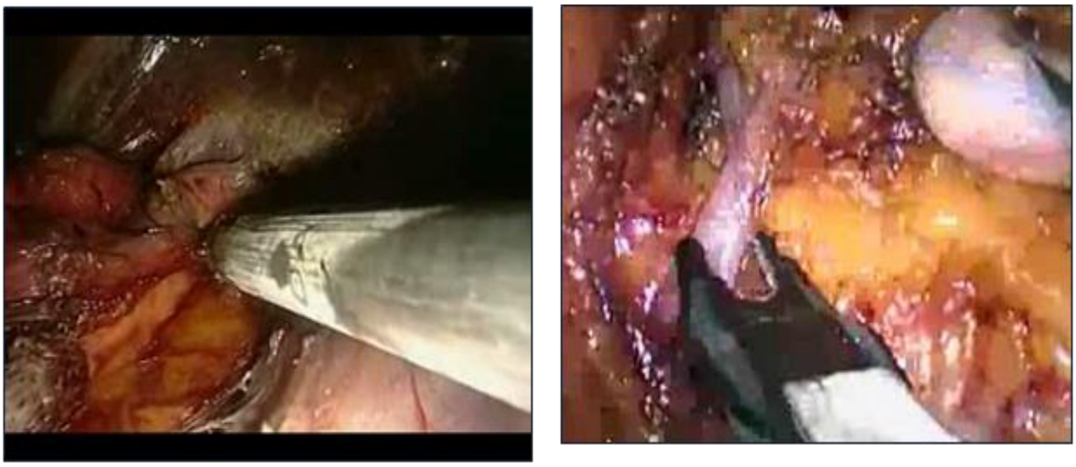


Figure 39. Clipping of adrenal central vein.

To cross the upper, middle and lower adrenal arteries is common enough in the targeted coagulation clipping is not necessary (Fig. 40).

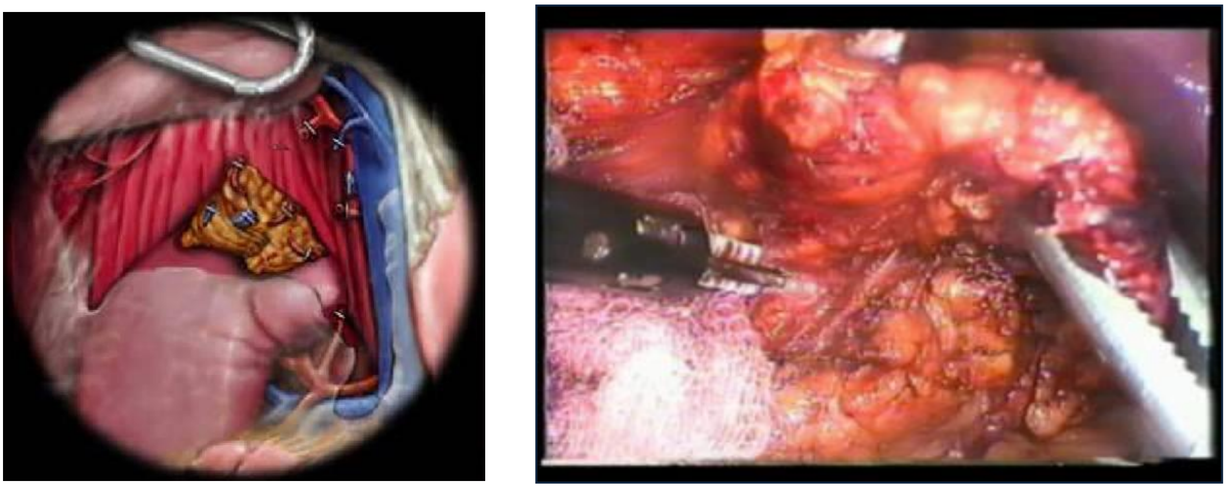


Figure 40. Coagulation of adrenal arteries.

Finally stage IV - dissection of the adrenal gland, which is considered a major milestone and concludes the operation (Fig. 41).

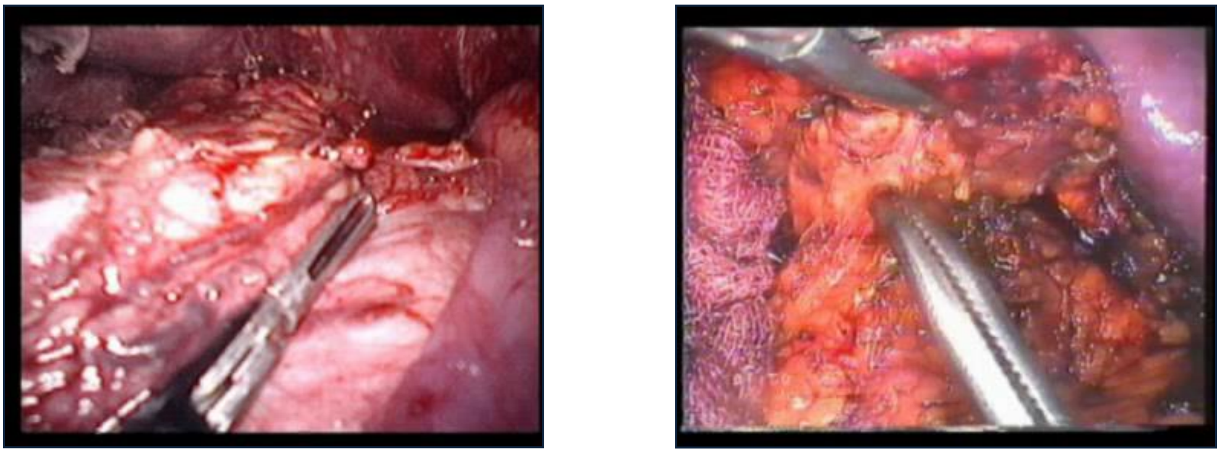


Figure 41. The release of the adrenal gland from the surrounding tissues for later disposition.

Thus, in our opinion, laparoscopic adrenalectomy disadvantages are:

- Educational. Manipulations are unusual, so if you need to learn the method of training, followed by a long period of improvement of skills.
- Technic. The surgeon, who often rely on their skills, experience and knowledge, becomes dependent on the quality of the work of many complex electronic devices on the qualifications of the auxiliary and technical staff.
- Camcorder. Even such a sophisticated and modern video camera, as used in surgery, remains still less perfect than the human eye, two-dimensional image.
- Tactile sensations are scarce - no way to directly fingers palpate the body to determine its consistency, mobility, and so on. Nonsensitive fingers.

However, the prospects of this trend in medicine will:

- Empowerment endovideosurgery with adrenal tumors greater than 6 cm by using a technique «hand-assist».
- Use for dissection of modern equipment - Harmonic, Liga-Sure.
- Use for visual inspection 3D monitors.
- A tech is the possibility of partial adrenalectomy in patients with bilateral adrenal lesions.

CONCLUSION

Thus, analysis of the literature and the results of a survey of patients in our clinic shows that the diagnosis of adrenal genesis of hypertension (hyperplasia, adenoma, pheochromocytoma, and cysts) CT is a highly informative method. CTA and MRA have approximately the same sensitivity in the diagnosis of renal artery stenosis. MRA is indicated for kidney function and iodine intolerance solid matter. Angiography remains the method of choice in the diagnosis of renovascular hypertension, considering the possibility of simultaneous correction of pathology. In the diagnosis of hyperaldosteronism is advisable to investigate the adrenal hormones. And for renal disease and renal arteries - renal scintigraphy with captopril sample PSM renal arteries with the study of blood flow.

Endovascular interventions for adrenal suppression methods are effective in the treatment of hypertensive patients with adrenal origin. And in large sizes (more than 3 cm to 7 cm) in these patients, laparoscopic adrenalectomy is shown. When tumors more than 7 cm, it is necessary to perform open adrenalectomy.

Patients with VRH open surgical procedures are indicated for: renal inefficiency or stenting; aneurysms of the renal arteries; diffuse lesions; aortoarteriites with lesions of the renal arteries and other visceral branches of the aorta.

We have developed a diagnostic and treatment algorithm (Fig. 42), which allows the use of diagnostic medical intervention as adequately conduct landmark and one-stage simultaneous intervention. This algorithm examination and treatment of patients with hypertension allows maximum accuracy to identify the etiology and pathogenesis of hypertension and send endovascular or surgical treatment of the fact pathogenetic link in the chain of hypertension, which is responsible for the most severe and malignant course of the disease.

The use of modern methods of diagnosis and sparing surgical techniques endovascular treatment can significantly improve outcomes in patients with symptomatic hypertension.

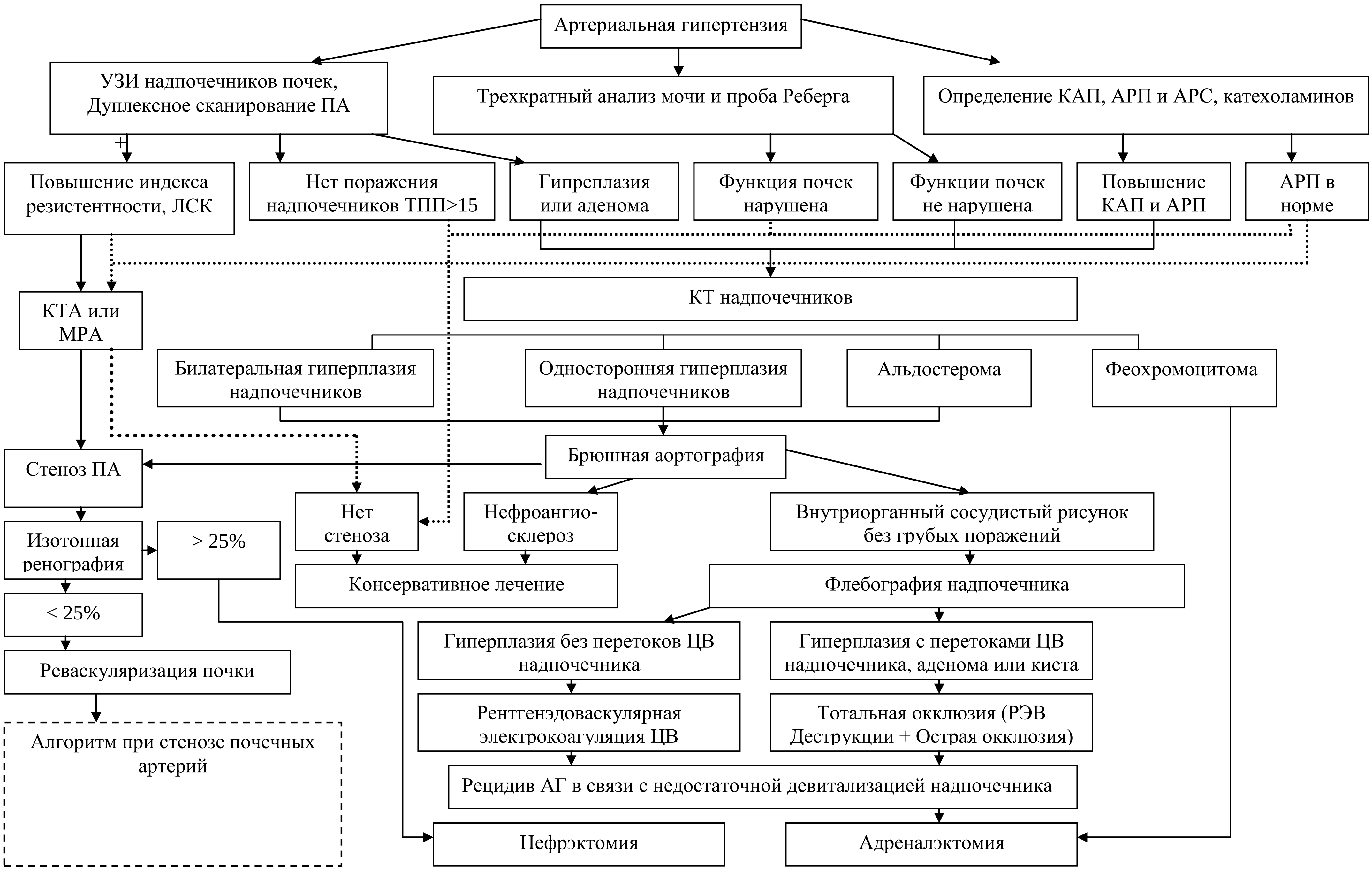


Рис. 42. Лечебно – диагностический алгоритм.

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