

## Physiological Bases of Hypertension

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Many different theories of the pathogenesis of hypertension have been created, but the true mechanism of its course remains unclear to this day. The article discusses the concept of hypertension as a neurosis of the higher autonomic centers, which is transformed into a metabolic syndrome.

The processes occurring during hypertension in the body are regulated by the autonomic nervous system. Inhibition of the noradrenergic system and a decreased level of noradrenaline in the blood are associated with an increase in blood pressure and lead to the activation of the cholinergic system. This activation causes impaired blood flow and damage to the vascular endothelium, which contributes to arterial hypertension.

A decrease in the amount of intracellular fluid in patients with essential hypertension entails the destruction of DNA, and as a consequence, a violation of ATP synthesis and transport of substances by protein carriers.

A nonspecific mobilization response to irritation is realized not only by the nervous, but also by the endocrine pathway. Under conditions of constant stress, adrenal hormones are released, the level of which is significantly increased in hypertension. With the failure of the inhibitory system, hypothalamic liberins are released, which act similar to the cholinergic system. Thus, the hierarchically organized hormonal system aggravates the effect of neurogenic influences and closes the pathological circle of the disease.

An increase in blood pressure in humans attracted the attention of medical scientists after the invention of an apparatus for measuring pressure according to the Korotkov method N.S. in 1905. This method of non-invasive measurement of blood pressure has steadily entered the medical and diagnostic practice and has not lost its significance to this day.

In 1922, the Soviet therapist G.F. Lang proposed a hypothesis of the etiopathogenesis of hypertension as a neurosis of the higher autonomic centers of the brain [1,2]. However, foreign doctors were more inclined to consider the cause of hypertension as kidney damage, accompanied by a violation of the regulation of hormones of the pituitary and adrenal glands. Another theory was developed by Yu. S. Postnov in 1987-2008, she explains the pathogenesis of hypertension by energy disturbances in cells, in particular, ATP deficiency in the cell [3,4].

Currently, there is a popular opinion that GB is a hereditary disease. That is, there is a violation of the order of the alleles of the gene and the destruction of the DNA molecule. Nevertheless, there is still no clarity in the pathogenesis of hypertension.

The processes occurring during hypertension in the body are regulated by the autonomic nervous system. The noradrenergic part of the autonomic nervous system is inhibitory. The mechanism is based on the binding of the postsynaptic membrane adrenergic receptor to norepinephrine, which is released from the presynaptic

membrane. Chemical transmission of a nerve impulse at a synapse allows information to be transmitted to the periphery according to the "more or less" principle, i.e. gradual [5]. Then the information is converted from chemical to digital. When the frequency of impulses on the postsynaptic membrane decreases, inhibition occurs and the transmission of nerve impulses to the periphery stops [6].

The normal distribution law is characterized by the formula for the probability density (Gauss formula), the normal distribution curve [5,7].

In 1935, in experiments on animals, the British electrophysiologist E. D. Adrian, acting on the sciatic nerve with an electric current, found inhibition of the response to exposure to electricity [8,9]. B.A. Medvedev in 1977 conducted experiments in which clonidine caused an increase in the release of norepinephrine from the presynaptic membrane, and as a result, the systemic arterial pressure decreased [10]. HER. Gogin in 1978 found that in hypertension of the 2<sup>nd</sup> stage, the level of adrenaline was significantly lower than normal [11]. M.S. Kushakovskiy in 1993 also found a relationship between the level of blood pressure and the level of noradrenaline in the blood plasma [12]. Thus, it can be argued that with hypertension, a deficiency of norepinephrine is found in the blood plasma of patients with hypertension.

On January 20, 2004, I sent documents for a vaccine for the treatment of hypertension at FIBS [13].

In conditions of a decrease in the function of the adrenergic system, another system of the ANS-cholinergic system is activated. The main component of the cholinergic system is the cholinergic receptor. Acetylcholine is released from the presynaptic membrane and enters the postsynaptic membrane, but at the same time, a calcium ion is released after acetylcholine, which actively joins the M-cholinergic receptors of the smooth muscles of the arterioles. There is a contraction of smooth muscle cells of arterioles up to tetanus [14]. The flow of blood in the vascular bed instead of laminar becomes vortex, turbulent [15]. In this case, the delicate endothelium of the vessels is injured and platelets, which are close to the endothelium in the bloodstream, come to the site of the defect, and macrophages also join there. This formation may be the prototype of a plaque. These processes contribute to an increase in blood pressure, and with age, collagen is lost, which is replaced by connective tissue [16].

In a cell, the volume of intracellular fluid in patients with hypertension is 15 - 20% lower than in healthy people [17]. With an excessive intake of calcium into the cell, the concentration of calcium in the cell rises to 10<sup>-4</sup> M instead of 10<sup>-7</sup> M in the norm [15]. At the same time, the amount of Na<sup>+</sup> ions in the cell decreases, lowering the pH level to 5.4 and "acidification" occurs in the cell [18]. Antiport Na<sup>+</sup>/H<sup>+</sup> is violated.

Based on the foregoing, the amount of Na<sup>+</sup> decreases inside the cell, as well as the amount of water. Water, being a crystal, is bound to the DNA of the cell [19]. Lack of water leads to the destruction of the DNA molecule, and, consequently, disruption of the transcription of the protein, which is an integral part of membranes and mitochondria and makes up 70 - 80% [17]. As a result of these processes, there is a violation of ATP synthesis, as well as a violation of the entry of sugar and insulin into the cell, because these substances enter the cell only when accompanied by protein.

G. Selye designated the non-specific mobilization response of the body to any irritation as stress [20]. This is an extreme reaction of the body, which is based on the syndrome of adaptation. It is realized through the body's neuroendocrine response. The subtle reaction of the body to external influences is carried out not only due to the ANS, but also due to hormones. The adaptation mechanism includes CNS hormones - neurotransmitters, norepinephrine, GHB. With short-term stress, the process is quickly restored and the level of hormones returns to normal, however, if stress persists for a long time, then, in accordance with Gauss's law, hormones are depleted. In this case, the release of the underlying hormones of the hypothalamus, pituitary, adrenal hormones is activated [21]. Hormones in the body represent a hierarchical system, vertically located and functionally working according to the principle of feedback.

Adrenal hormones are called survival hormones. Epinephrine, vasopressin, aldosterone, cortisol increase in blood plasma by 100 - 150% during stress. In hypertension, in comparison with the norm, their level is significantly higher. So, vasopressin is increased by 30% [22,23]. Particularly noteworthy is the participation of peptide hormones of the hypothalamus in hypertension. In conditions of failure of the inhibitory system in hypertension, the action of liberins prevails, which function similarly to the cholinergic system of the ANS [24]. Hormones complete the pathological circle.

## Conclusion

To summarize, it can be assumed that HD is based on the neurosis of the higher autonomic centers, associated with inhibition of the noradrenergic and activation of the cholinergic systems. The neurosis is modified into a metabolic syndrome, which manifests itself in the form of a violation of ATP synthesis and transport of substances. At the same time, the hierarchically organized hormonal system aggravates the effect of neurogenic influences and closes the pathological circle of the disease.

## Bibliography

- Shlyakhto EV and Konradi AO. "Classification of arterial hypertension: from Bright's disease to the cardiovascular continuum". *Arterial Hypertension* 10.2 (2004): 98-103. [Shlyakhto E. V., Konradi A. O. Classification of Arterial Hypertension: from Bright Disease to Cardiovascular Continuum. *Arterial'naya Gipertenziya*. 2004; Vol. 10 (2): 98-103. (In Russian)].
- Lang GF. "Hypertonic disease. L.: Medgiz, Leningrad. Separation (1950). [Lang GF. "Hypertonic Disease". Leningrad: Medgiz (1950). (In Russian)].
- Postnov YuV and Orlov SN. "Primary hypertension as a pathology of cell membranes". M.: Medicine (1987). [Postnov YuV and Orlov SN. "Primary hypertension as a pathology of cell membranes". Moscow: Meditsina (1950). (In Russian)].
- Postnov YuV., *et al.* "Disruption of energy conversion in cell mitochondria with a decrease in ATP synthesis as a cause of a stationary increase in the level of systemic arterial pressure". *Cardiology* 48.8 (2008): 49-59. [Postnov YuV., *et al.* "Violation of energy conversion in mitochondria of cells with a decrease in ATP synthesis as a cause of a stationary increase in systemic blood pressure". *Kardiologiya* 48.8 (2008): 49-59. (In Russian)].
- Leshchenko VG and Ilyich GK. "Medical and biological physics: textbook". M.: INFRA-M; Minsk: New knowledge (2017). [Leshchenko VG and Il'ich GK. "Medical and Biological Physics: Textbook". Moscow: INFRA-M; Minsk: Novoe Znanie (2017). (In Russian)].
- Halperin SI. "Human anatomy and physiology (age characteristics with the basics of school hygiene): textbook". 2<sup>nd</sup> edition. M.: Higher school (1974). [Gal'perin SI. "Human anatomy and physiology (age-related features with the basics of school hygiene): Textbook". 2<sup>nd</sup> edition. Moscow: Vysshaya Shkola (1974). (In Russian)].
- Leshchenko VG and Ilyich GK. "Medical and biological physics: textbook". Minsk: New knowledge: INFRA-M (2012). [Leshchenko VG and Il'ich GK. "Medical and Biological Physics: Textbook". Minsk: Novoe Znanie: INFRA-M (2012). (In Russian)].
- Adrian ED and Yamagiwa K. "The origin of the Berger Rhythm". *Brain* 56.3 (1935): 323-351.
- Adrian ED. "Basics of sensations". M.: State. honey. publishing house (1931). [Adrian ED. "The basis of sensation". Moscow: Gosudarstvennoe meditsinskoe izdatel'stvo (1931). (In Russian)].
- Medvedev BA. "Pharmacological study of clonidine". *Pharmacology and Toxicology* 3 (1977): 288-296. [Medvedev BA. "Pharmacological study of clonidine". *Farmakologiya i Toksikologiya* 3 (1977): 288-296. (In Russian)].
- Gogin EE., *et al.* "Arterial hypertension". L.: Medicine, Leningrad. Department (1978). [Gogin EE., *et al.* "Arterial hypertension". Leningrad: Meditsna (1950). (In Russian)].
- Kushakovskiy MS. "Hypertension and secondary arterial hypertension". Edition. 3, erased. L.: Medicine; (1983). [Kushakovskiy MS. "Hypertension and secondary arterial hypertension". Edition. 3, erased. Leningrad: Meditsna (1983). (In Russian)].
- Lebedeva VD. "Pathogenetic method of treatment of patients with essential hypertension, taking into account its metabolic mechanisms". *Clinical Pathophysiology* 23.3 (2017): 23-28. [Lebedeva VD. "Pathogenetic method of treating patients with hypertension taking into account its metabolic mechanisms". *Klinicheskaya Patofiziologiya* 23.3 (2017): 23-28. (In Russian)].
- Golikov SN., *et al.* "Cholinergic regulation of biological systems of the cell". M.: Medicine (1985). [Golikov SN., *et al.* "Cholinergic regulation of cell biological systems". Moscow: Meditsna (1985). (In Russian)].
- Guyton AK and Hall JE. "Medical Physiology: Textbook". M.: Logosphere (2008). [Guyton Arthur C and Hall John E. "Medical Physiology: Textbook". Moscow: Logosfera (2008). (In Russian)].

16. Kanungo MS. "Biochemistry of aging". M.: Mir (1982). [Kanungo MS. "Biochemistry of Aging". Moscow: Mir (1982). (In Russian)].
17. Ivanova NV. "Effect of propranolol on the volume of extracellular fluid, renin activity, plasma aldosterone concentration and central hemodynamics in patients with stage II hypertension". Dissertation. L (1983). [Ivanova NV. "The effect of propranolol on extracellular fluid volume, renin activity, plasma aldosterone concentration and central hemodynamics in patients with stage II hypertension". The dissertation. Leningrad (1983). (In Russian)].
18. Skulachev VP, *et al.* "Membrane bioenergy: textbook". Moscow: Moscow University Publishing House; (2010). [Skulachyov VP, *et al.* "Membrane Bioenergy: A Training Manual". Moscow: Moscow University Press (2010). (In Russian)].
19. Костенко ВГ. "Живая клетка глазами химика-органика. СПб.: ВВМ (2009). [Kostenko VG. "Living cell through the eyes of an organic chemist". Saint Petersburg: ВВМ (2009). (In Russian)].
20. Selye HA. "Syndrome produced by Diverse Nocuous Agents". *Nature* 138.3479 (1936): 32.
21. Rob AI. "The relationship of endocrine complexes under stress". Chisinau: Shtiintsa (1982). [Robu AI. "The relationship of endocrine complexes under stress". Chisinau: Stiincea (1982). (In Russian)].
22. Raitsek DE., *et al.* "Secrets of arterial hypertension". M.: BINOM (2005). [Hricik Donald E., *et al.* "Hypertension secrets". Moscow: BINOM (2005). (In Russian)].
23. Meerson FZ. "Pathogenesis and prevention of stress and ischemic heart damage". M.: Medicine (1984). [Meerson FZ. "Pathogenesis and prevention of stress and ischemic heart damage". Moscow: Meditsna (1984). (In Russian)].
24. Zaichik ASh and Churilov LP. "General pathophysiology (with the basics of immunopathology): A textbook for students of medical universities. 4<sup>th</sup> edition. SPb: ELBI 1 (2008). [Zaychik A and Sh Churilov LP. "General pathophysiology (with the basics of immunopathology): A textbook for students of medical universities". 4<sup>th</sup> edition. T.1. Saint Petersburg: ELBI (2008). (In Russian)].

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