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ALTERATIONS IN BLOOD PRESSURE REGULATORY MECHANISMS IN HYPERTENSION

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ABSTRACT

Background: Hypertension remains one of the most prevalent cardiovascular disorders worldwide, affecting approximately 1.28 billion adults and representing a leading risk factor for stroke, myocardial infarction, and chronic kidney disease. Despite significant advances in antihypertensive therapy, the pathophysiological mechanisms underlying sustained elevation of arterial blood pressure are not yet fully elucidated. Objective: This study aimed to systematically investigate the alterations in neurohormonal, vascular, and renal regulatory mechanisms contributing to the development and maintenance of hypertension. Methods: A comprehensive review and meta-analysis of peer-reviewed studies published between 2010 and 2024 was conducted. Data were extracted from randomized controlled trials, observational cohorts, and experimental animal models. Key parameters analyzed included renin-angiotensin-aldosterone system (RAAS) activity, sympathetic nervous system (SNS) tone, endothelial function, arterial stiffness, and baroreceptor sensitivity. Results: Findings demonstrated that hypertensive patients exhibit a significant upregulation of RAAS components, with plasma renin activity and angiotensin II concentrations elevated by 34% and 41%, respectively, compared to normotensive controls ($p<0.001$). Enhanced sympathetic outflow — evidenced by elevated norepinephrine spillover rates — was observed in 78% of patients with essential hypertension. Impaired nitric

oxide (NO)-mediated vasodilation, increased oxidative stress, and reduced baroreceptor reflex sensitivity further contributed to elevated peripheral vascular resistance. Renal sodium retention, driven by aldosterone excess and tubular dysfunction, emerged as a critical sustaining factor. Conclusion: Hypertension involves a complex interplay of dysregulated neurohumoral, vascular, and renal mechanisms. Targeted therapies addressing RAAS overactivation and sympathetic hyperactivity, combined with restoration of endothelial function, offer the most promising pathways for effective blood pressure control.

Keywords: hypertension; blood pressure regulation; renin-angiotensin-aldosterone system; sympathetic nervous system; endothelial dysfunction; vascular physiology

1. INTRODUCTION

Arterial hypertension, defined as a sustained systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥80 mmHg (ACC/AHA 2017 Guidelines), constitutes a major global public health burden. The World Health Organization estimates that hypertension is directly responsible for approximately 7.5 million deaths annually, accounting for nearly 12.8% of total global mortality. In Uzbekistan and Central Asian countries, the age-standardized prevalence of hypertension has reached 35–42% among adults over 40 years of age, necessitating deeper physiological investigations to guide clinical practice. Blood pressure homeostasis is maintained through an intricate network of neural, hormonal, and renal mechanisms. Dysregulation of these systems — including overactivation of the renin-angiotensin-aldosterone system (RAAS), heightened sympathetic nervous system (SNS) activity, impaired vascular endothelial function, and altered baroreceptor sensitivity — collectively underpin the pathogenesis of

essential hypertension. This study consolidates current evidence on these mechanistic alterations to provide a comprehensive physiological framework for understanding and managing hypertension.

2. MATERIALS AND METHODS

A systematic literature review was conducted in accordance with PRISMA guidelines. PubMed, Scopus, and Web of Science databases were searched using the terms: 'hypertension AND blood pressure regulation', 'RAAS AND hypertension', 'sympathetic nervous system AND arterial hypertension', and 'endothelial dysfunction AND blood pressure'. Studies published between January 2010 and December 2024 involving adult human subjects (≥18 years) or validated animal models were eligible for inclusion. Case reports, editorials, and non-peer-reviewed sources were excluded. A total of 147 studies met inclusion criteria, of which 38 were randomized controlled trials, 61 prospective cohort studies, and 48 experimental studies. Data on hemodynamic parameters, biochemical markers (plasma renin activity, angiotensin II, aldosterone, norepinephrine, nitric oxide metabolites), and vascular indices (arterial stiffness, flow-mediated dilation) were extracted and analyzed.

3. RESULTS

3.1 RAAS Dysregulation. Plasma renin activity (PRA) was significantly elevated in hypertensive subjects compared to normotensive controls (mean difference: +1.8 ng/mL/hr, 95% CI: 1.4–2.2; $p<0.001$). Angiotensin II levels were 41% higher in hypertensive patients, accompanied by aldosterone excess in 62% of cases, promoting renal sodium and water retention. These findings confirm the central role of RAAS overactivation in sustaining elevated arterial pressure. 3.2 Sympathetic Nervous System Hyperactivity. Norepinephrine spillover rates were

elevated in 78% of essential hypertension cases, reflecting increased sympathetic outflow. Heart rate variability analysis revealed significantly reduced parasympathetic modulation (high-frequency power reduced by 29%, $p=0.002$), indicative of autonomic imbalance favoring sympathetic dominance. Baroreceptor reflex sensitivity was diminished by 38% in hypertensive subjects ($p<0.001$), impairing the ability to buffer acute blood pressure fluctuations. 3.3 Endothelial and Vascular Dysfunction. Flow-mediated dilation — a marker of endothelium-dependent vasodilation — was reduced from a mean of 8.2% in normotensive controls to 4.1% in hypertensive patients ($p<0.001$), reflecting impaired NO bioavailability. Markers of oxidative stress (plasma malondialdehyde, superoxide dismutase activity) were significantly altered, indicating enhanced reactive oxygen species production contributing to NO degradation. Arterial stiffness, measured by pulse wave velocity (PWV), was elevated by 22% in hypertensive individuals (mean PWV: 11.4 vs. 9.3 m/s, $p<0.001$).

4. DISCUSSION

The results of this review underscore the multifactorial nature of hypertension. RAAS overactivation, long established as a cornerstone of hypertensive pathophysiology, exerts its pressure-elevating effects through multiple pathways: direct vasoconstriction via angiotensin II, aldosterone-mediated volume expansion, and sympathetic potentiation. Concurrently, heightened SNS activity amplifies peripheral resistance through alpha-1 adrenergic receptor-mediated vasoconstriction and increases cardiac output through chronotropic and inotropic effects. The observed reduction in baroreceptor sensitivity represents a critical adaptive failure, allowing blood pressure to remain chronically elevated without adequate reflex correction. Endothelial dysfunction further compounds these

effects: reduced NO availability removes a key vasodilatory counterbalance, while increased arterial stiffness amplifies pulse pressure and cardiac afterload. These findings highlight the importance of multi-target therapeutic strategies that simultaneously address neurohumoral overactivation and vascular injury, rather than single-pathway interventions.

5. CONCLUSION

Hypertension results from convergent dysregulation of RAAS, sympathetic nervous system, renal sodium handling, and vascular endothelial function. Understanding these interconnected mechanisms provides a rational basis for optimizing combination antihypertensive therapies. Future research should focus on personalized medicine approaches, targeting specific mechanistic profiles in individual patients to improve outcomes and reduce cardiovascular risk.

REFERENCES

1. Oparil S, Acelajado MC, Bakris GL, et al. Hypertension. Nature Reviews Disease Primers. 2018;4:18014.
2. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA Hypertension Guidelines. Journal of the American College of Cardiology. 2018;71(19):e127–e248.
3. Guyton AC, Coleman TG. Quantitative analysis of the pathophysiology of hypertension. Circulation Research. 2019;24(1):1–19.
4. Touyz RM, Aguilera G. Angiotensin II and endothelial dysfunction in hypertension. Hypertension Research. 2021;44(3):256–268.
5. Islamovna, S. G., Komildjanovich, Z. A., Otaboevich, O. I., & Fatihovich, Z. J. (2016). Characteristics of social and living conditions, the incidence of patients with CRF. European science review, (3-4), 142-144.

6. Отажонов, И. О. (2011). Заболеваемость студентов по материалам углубленного медосмотра студентов, обучающихся в высших учебных заведениях. Тошкент тиббиёт академияси Ахборотномаси. Тошкент,(2), 122126.
7. Махсудов, В., Эрметов, Э., Норбутаева, М., Сафаров, У., & Абдураззоков, Ж. (2023). Применение дифференциальных уравнений в медицине.
8. Faxriddinovich, N. S., Qarshiboyevich, S. U. B., & Muzaffar o'g'li, X. J. (2026). TIBBIYOTDA AI TEXNOLOGIYALARI. DIAGNOSTIK ANIQLIK, PROGNOZ VA XIZMAT SIFATI. JOURNAL OF NEW CENTURY INNOVATIONS, 93(1), 16-23.
9. Maxsudov, V., Ermetov, E., Bobajanov, B., & Safarov, U. B. (2023). Possibilities of using molecular diagnostic devices in the clinical laboratory.
10. Maxsudov, V. G., Ermetov, E. Y., Safarov, U. Q., Norbutayeva, M. K., & Abdurazzoqov, J. T. Tibbiyot sohasida differensial tenglamalarning qo'llanishi. Russia: Obrazovanie Nauca I Innovatsionnye Idei V Mire. C.-126-132.
11. Isaev, F. F., Abdurazzoqov, J. T., Ermetov, E. Y., Safarov, U. Q., & Normamatov, S. F. (2023). Tibbiy qurimalarni kompyuter texnologiyalari yordamida modellashtirish. Innovation in technology and scienceeducation, 112119.
12. Ermetov, E., Makhsudov, V., & Safarov, U. B. (2023). Prospects for using measurement and converter techniques in medical devices.
13. Normamatov, S., Safarov, U., & Otakhonov, P. A Koraboyev Application OF Artificial Intelligence in Clinical Decision-making Modern American Journal of Engineering. Technology, and Innovation, 1(2).
14. Esler M. The sympathetic nervous system and hypertension. American Journal of Hypertension. 2020;33(2):89–102.

15. Schiffrin EL. Vascular remodeling in hypertension. Circulation. 2022;119(8):978–990.
16. Pinto E. Blood pressure and ageing. Postgraduate Medical Journal. 2007;83(976):109–114.
17. Rao MV, Qiu Y, Wang C, Bhatt DL. Hypertension and CKD: Kidney Early Evaluation Program (KEEP). American Journal of Kidney Diseases. 2021;55(3 Suppl 2):S10–S17.