



CODEN [USA]: IAJPBB

ISSN : 2349-7750

**INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES**

SJIF Impact Factor: 7.187

<http://doi.org/10.5281/zenodo.4394918>Available online at: <http://www.iajps.com>

Research Article

**HYPERTENSION AWARENESS AND ITS INFLUENCE
HORMONAL MECHANISM LEADING TO CHRONIC
DISEASE.**¹Dr Hira Saif,²Dr Maria Warraich,³Dr Rumaisa Zahid
^{1,2,3}MBBS, Nawaz Sharif Medical College, Gujrat.**Article Received:** October 2020**Accepted:** November 2020**Published:** December 2020**Abstract:**

Hypertension remains the leading cause of morbidity and mortality worldwide and significantly impacts the risk of all major cardiovascular events which includes sudden cardiac death, stroke, coronary heart disease, peripheral vascular disease, heart failure, and abdominal aortic aneurysm. Important advances in our understanding of its pathophysiology contributed to clarifying the complex origins of the disease, involving dysregulation of multiple homeostatic systems influencing not only blood pressure but also the progression of end-organ damage related to hypertension. Growing evidence suggests that the pathophysiology of hypertension results from complex interactions between environmental and genetic factors, resulting in different risks and age of onset of the disease within the general population. Also, it provides a comprehensive overview of the mechanisms involved in the aetiopathogenesis of hypertension, highlighting their relative importance in different forms of hypertension.

Corresponding author:**Dr. Hira Saif,**

MBBS, Nawaz Sharif Medical College, Gujrat.

QR code



Please cite this article in press Hira Saif et al, *Hypertension Awareness And Its Influence Hormonal Mechanism Leading To Chronic Disease.*, Indo Am. J. P. Sci, 2020; 07(12).

INTRODUCTION:

Hypertension is the leading risk factor for death worldwide. [1] Its proper definition differ, as the latest American guidelines have reduced the thresholds for the proper diagnosis of arterial hypertension to values of systolic blood pressure (SBP) by 130 mmHg and diastolic blood pressure (DBP) by 80 mmHg, [2] while the 2018 European guidelines showed the old thresholds by 140/90 mmHg. Deaths related to hypertension occur most commonly as a result of hemorrhagic stroke ischemic heart disease, and ischemic stroke, which are estimated to account for 4.9, 2.0, and 1.5 million deaths, respectively. [3] Raised systolic blood pressure highly impact on mortality and disability worldwide accounting for 218 million (95% uncertainty interval 198–237 million) global disability-adjusted life years (DALYs) for both male and female. [4] An increased burden of hypertension related cardiovascular disease (CVD) was also showed in data. Between 2007 and 2017 the number of DALYs due to hypertensive heart disease increased by 31%. [4] Reliable estimations of hypertension prevalence and its trends are essential to calculate the burden of future events and costs of intervention policies. The prevalence of hypertension is difficult to find as limited comparable data are available and the population being examined by different methods used to apply these criteria in practice. [5] For example, several surveys used to assess the prevalence of hypertension across different countries have identified the hypertension burden based on measurements obtained from a single evaluation, and it is now well established that the use of this approach carries a substantial risk of a false-positive diagnosis.

Due to the difficulty of obtaining comparable results among countries, the World Health Organization Global Health Observatory represents an important source of data on the prevalence of hypertension and the mean SBP values among 51 European national populations. The first striking feature of these data is the similarity of the pattern of the association between blood pressure and age as well as between blood pressure both male and female in different countries. Systolic blood pressure progressively increases throughout life, with a difference of 20–30 mmHg between early and late adulthood. [6,7] Dystolic blood pressure increases to a lower extent until the fifth decade, and then average Dystolic blood pressure tends to remain constant or, more frequently, to decline slightly. [6,8] For both systolic blood pressure and dystolic blood pressure, the mean levels are higher in men than in women in early adulthood, although this difference narrows progressively and is either

non-existent or reversed by the sixth or seventh decade. [9,10]

DISEASE RELATED RISK:**Cardiovascular disease:**

A striking feature of the association between blood pressure and the risk of cardiovascular disease is that it spans all major cardiovascular events, including stroke and other forms of cerebrovascular disease, sudden cardiac death, coronary heart disease, heart failure, abdominal aortic aneurysm, and peripheral vascular disease. The risk associated with higher blood pressure is similar for different cardiovascular disease conditions. The relationship between blood pressure values and cardiovascular events is independent of other cardiovascular disease risk factors. However, due to the well-documented risk factor clustering that is seen in hypertensive individuals, guidelines on the management of raised blood pressure recommend assessment of total globally cardiovascular disease risk in this population, as only a small fraction of the hypertensive population has an elevation of BP alone. [11] In the Framingham Heart Study, for example, more than 80% of hypertensive individuals had one or more coexisting risk factors, and 55% had two or more risk factors. [12] When hypertension coexists with other cardiovascular risk factors, the total cardiovascular risk increases exponentially rather than resulting from the sum of its components. This is true also for relatively low values of blood pressure. For example, prehypertension leads to a 10% increase in the 10-year absolute risk of cardiovascular disease for middle-age adults without diabetes, which rises to 40% in the presence of diabetes and established cardiovascular disease. [13] Similar results are available for patients with established hypertension. Interheart study showed the presence of a single risk factor (smoking, hypertension, dyslipidaemia, or diabetes) was associated with a two- to threefold increase in the risk of acute myocardial infraction, while when hypertension coexists with the other three risk factors was related to a 20-fold increase in the same risk. [13] A combination of antihypertensive drugs with other therapies is often necessary to obtain adequate control of blood pressure values in individuals with multiple cardiovascular risk factors, to maximize cost-effectiveness of the management of hypertension.

Renal Mechanism:

Kidney is known to be an important regulator of blood pressure an impaired renal function is irrespective its main cause and is almost invariably associated with the progression of hypertension. The most important mechanisms by which the kidney controls blood

pressure is homeostasis and are by regulating the pressure–natriuresis with the relationship and the activity of the renin–angiotensin (RAS) system. Also the perturbation of the renal physiology induced by an impaired renal blood flow and an elevation of renal inflammation and oxidative stress burdens stimulate adaptive changes that could be leaded, if sustained, to increased blood pressure. When dietary intake of potassium is low and sodium intake high, a large proportion of the aldosterone-regulated potassium and sodium transport would occur in the distal convoluted tubule and in the connecting tubule, collectively identified as the ‘aldosterone-sensitive distal nephron’. [14] Impaired renal blood flow is the impaired microvascular function leading to a decrease in glomerular blood flow might result in the release of substances (such as renin) that raise blood pressure. As a result of which renal perfusion pressure increases, reducing the risk of parenchymal ischaemia.

Vascular mechanisms:

Structural and functional vascular abnormalities either in the micro- or macro circulation are involved in the pathophysiology of hypertension, leading to increased total peripheral resistance and arterial stiffening. Though the specific contribution of each mechanism involved in the pathogenesis of hypertension can be hardly quantified, accumulating evidence shows that vascular abnormalities play a role which is equally important as renal vascular volume control. Main mechanisms involved in the pathophysiology of essential hypertension diastolic blood pressure; mean blood pressure, pulse pressure, renin–angiotensin system, systolic blood pressure. Microcirculation in which microcirculatory alterations are considered both a cause and consequence of hypertension, particularly in the diastolic and systo-diastolic forms, which are caused by either volume overload and total peripheral resistance increase. Peripheral vascular resistance is controlled mainly at the level of small arteries and arterioles. Their vascular tone is regulated by many factors, including the sympathetic nervous system, humoral factors (mostly endothelium derived), and local autoregulation. Arteriolar and capillary rarefaction, as well as small artery remodelling, are early hallmarks of hypertension and account for most of the end-organ damage related to the disease. Microvascular alterations are part of a vicious cycle that initiates, maintains, and amplifies high blood pressure. [15,16]

Hypertension, and particularly elevated pulse pressure, might accelerate the age-related process of vascular stiffening, amplifying the damage to elastin lamellae and substitutions with collagen fibers.

Building on these pathophysiological concepts, increased large artery stiffness assessed by pulse wave velocity has long been considered a consequence of hypertension. However, recent studies showed that increased pulse wave velocity is not invariably associated with pulse pressure widening, suggesting that it might precede and contribute to the pathogenesis of the disease, rather than being its consequence. [17] Independently from its temporal relationship with hypertension, increased stiffness of the large arteries leads to transmission of excessive pulsatile energy to small resistance arteries and capillaries, causing an arterial wall barotrauma and irreversible damage. The accumulation of microvascular damage accounts for the progression of the end-organ damage in hypertension, particularly in low-resistance organs such as the brain and the kidney. [18] Since most of the antihypertensive drugs target peripheral vascular resistance or cardiac output, with minimal influence on large artery stiffness, isolated systolic hypertension remains the most common cause of treatment-resistant hypertension. [19]

Neural mechanisms:

The sympathetic nervous system plays a central role in cerebrovascular homeostasis. Sympathetic activity plays a key role in both short- and long-term BP control, conferring to the SNS an important role in the pathophysiology of hypertension. [20,21,22,23] Sympathetic activation induces sustained blood pressure increases by several mechanisms, including peripheral vasoconstriction, potentiation of cardiac contraction, reduction of venous capacitance, and modulation of the renal sodium and water excretion. [20,21,22] An increased sympathetic tone is associated with obesity, hyperinsulinaemia altered glucose metabolism, and obstructive sleep apnoea, all conditions associated with hypertension development and with resistant hypertension. [24]

CONCLUSION:

Hypertension is actually the root cause for many fatal diseases. HTN effects the arteries, veins badly leads to vasoconstriction, which progress to the life threatening problems. A comprehensive overview of the mechanisms involved in the aetiopathogenesis of hypertension, highlighting their relative importance in different forms of hypertension e.g. Cardiovascular mechanism, renal function, neural mechanism and vascular mechanism.

REFERENCES:

1. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based

- measurement studies with 19.1 million participants. *Lancet* 2017;389:37–55.
2. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Himmelfarb CD, DePalma SM, Gidding S, Jamerson KA, Jones DW, MacLaughlin EJ. 2017
 3. Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, Alexander L, Estep K, Abate KH, Akinyemiju TF, Ali R. Global burden of hypertension and systolic blood pressure of at least 110 to 115 mm Hg, 1990-2015. *Jama*. 2017 Jan 10;317(2):165-82.
 4. GBD 2013 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* 2018;392:1923–94.
 5. Pereira M, Lunet N, Azevedo A, Barros H. Differences in prevalence, awareness, treatment and control of hypertension between developing and developed countries. *J Hypertens* 2009;27:963–75.
 6. Baksi AJ, Treibel TA, Davies JE, Hadjiloizou N, Foale RA, Parker KH, Francis DP, Mayet J, Hughes AD. A meta-analysis of the mechanism of blood pressure change with aging. *J Am Coll Cardiol* 2009;54:2087–92.
 7. Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, Hense HW, Joffres M, Kastarinen M, Poulter N, Primatesta P, Rodríguez-Artalejo F, Stegmayr B, Thamm M, Tuomilehto J, Vanuzzo D, Vescio F. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *JAMA* 2003;289:2363–9.
 8. Franklin SS, Gustin Wt, Wong ND, Larson MG, Weber MA, Kannel WB, Levy D. Hemodynamic patterns of age-related changes in blood pressure. The Framingham Heart Study. *Circulation* 1997;96:308–15.
 9. Coylewright M, Reckelhoff JF, Ouyang P. Menopause and hypertension: an age-old debate. *Hypertension* 2008;51:952–9.
 10. Taddei S. Blood pressure through aging and menopause. *Climacteric* 2009;12 Suppl 1:36–40.
 11. Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Böhm M, Christiaens T, Cifkova R, De Backer G, Dominiczak A, Galderisi M. 2013 ESH/ESC Guidelines for the management of arterial hypertension. *Arterial Hypertension*. 2013;17(2):69-168.
 12. Kannel WB. Risk stratification in hypertension: new insights from the Framingham Study. *Am J Hypertens* 2000;13(1 Pt 2):3S–10S.
 13. Thomas F, Bean K, Pannier B, Oppert JM, Guize L, Benetos A. Cardiovascular mortality in overweight subjects: the key role of associated risk factors. *Hypertension* 2005;46:654–9.
 14. Egan BM, Stevens-Fabry S. Prehypertension – prevalence, health risks, and management strategies. *Nat Rev Cardiol* 2015;12:289–300.
 15. Struijker-Boudier HA, Heijnen BF, Liu YP, Staessen JA. Phenotyping the microcirculation. *Hypertension* 2012;60:523–7.
 16. Tsioufis C, Dimitriadis K, Katsiki N, Tousoulis D. Microcirculation in hypertension: an update on clinical significance and therapy. *Curr Vasc Pharmacol* 2015;13:413–7.
 17. Kaess BM, Rong J, Larson MG, Hamburg NM, Vita JA, Levy D, Benjamin EJ, Vasan RS, Mitchell GF. Aortic stiffness, blood pressure progression, and incident hypertension. *JAMA* 2012;308:875–81.
 18. Laurent S, Agabiti-Rosei E. The cross-talk between the macro- and the microcirculation. In: Nilsson P, Olsen MH, Laurent S, eds. *Early Vascular Aging (EVA): New Directions in Cardiovascular Protection*. London: Elsevier; 2015; pp 105–18.
 19. Mitchell GF. Arterial stiffness and hypertension: *Hypertension* 2014;64:210–4.
 20. Fink GD. Arthur C. Corcoran Memorial Lecture. Sympathetic activity, vascular capacitance, and long-term regulation of arterial pressure. *Hypertension* 2009;53:307–12
 21. Joyner MJ, Charkoudian N, Wallin BG. A sympathetic view of the sympathetic nervous system and human blood pressure regulation. *Exp Physiol* 2008;93:715–24.
 22. Grassi G. Assessment of sympathetic cardiovascular drive in human hypertension: achievements and perspectives. *Hypertension* 2009;54:690–7.
 23. Grassi G, Mark A, Esler M. The sympathetic nervous system alterations in human hypertension. *Circ Res* 2015;116:976–90.
 24. Okada Y, Galbreath MM, Shibata S, Jarvis SS, VanGundy TB, Meier RL, Vongpatanasin W, Levine BD, Fu Q. Relationship between sympathetic baroreflex sensitivity and arterial stiffness in elderly men and women. *Hypertension* 2012;59:98–104.