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### Essential Hypertension

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al hypertension(EH) is known the most common modifiable factor for cardiovascular related in the United States, er fifty percent of patients sed with hypertension (HTN) uncontrolled (Fontil, Bibbins-go, Nguyen, Guzman, & on, 2017). Incidences of olled HTN are greater among with low socioeconomic There is an opportunity our country to assist ble communities in ating to control HTN, leaving se impassioned by the ge of assisting communities ng health.

- Hypertension can have many deleterious effects if left unrecognized or untreated including stroke, renal failure, and myocardial infarction (Wise & Charchar, 2016).
- Increased work of the heart from HTN leads to increased risk for coronary artery disease (CAD), left ventricular hypertrophy, and heart failure.
- Chronic elevation in pressure creates mechanical damage to vascular system, heart, eyes, brain, and kidney's which results in disease to these organs.
- Weakening of vessel within the aorta and brain from the sustained elevation of pressure within the vessels can lead to aneurysms.

- Hypertension typically presents as asymptomatic creating a necessity for all patients to be screened for the disease.
- Symptoms may be present in the event of sustained hypertension with end organ disease.

- United States Preventative Services Task Force (USPSTF) recommends screening all patients over 18 for hypertension and states there are relatively few major harms and substantial benefits (USPSTF, 2016).
- Intra office blood pressure monitoring should be done while the patient is seated and with a minimum of 2 measurements at least 5 minutes between readings.
- Ambulatory or home monitoring should be complete as 15- 30% of the population is estimated to have lower BP outside of the office setting (USPSTF, 2016).
- Dietary sodium restrictions should be taught to all patients with hypertension.
- Excess adipose tissue stimulates the SNS increasing the risk of hypertension. Overweight and obese patients should be taught to reduce their weight to normal limits.
- Exercise can also be used to assist in BP control; and should include at least 30 minutes of aerobic activity a day for most days.
- Additional lifestyle modifications should include smoking cessation, alcohol limited at 1 oz of ethanol per day for men and 0.5 oz for women

EH is defined as sustained blood pressure (BP) greater than 140/90 and can be broken down into three subtypes including diastolic HTN in middle age, isolated systolic hypertension in older adults, and isolated systolic hypertension in young adults. All subtypes are attributed to an increase in cardiac output and (or) total peripheral vascular resistance (PVR) and can be related complex interactions involving the following mechanisms: neural, renal, hormonal, and vascular. These can all be influenced by environmental factors.

- Sympathetic over activity has been associated with HTN.
- Baroreceptors are reset to defend higher levels of BP (Bonow, Mann, Zipes, & Libby, 2012).
- Sympathetic nervous system (SNS) is thought to be activated in obesity to burn fat, this activation also targets vascular smooth muscle and kidneys thus creating a hypertensive state.
- SNS effects short term and long term BP regulation. In the long-term, beta 1 receptors in the juxtaglomerular apparatus are stimulated and create the release of renin; stimulation of alpha adrenergic 1 receptors produces renal sodium reabsorption.

- Renin angiotensin aldosterone system (RAAS) is a great contributor to vascular remodeling and endothelial dysfunction. In RAAS, angiotensin (renin substrate produced within the liver) is broken down by renin (a protease produced by the juxta glomeruli). This produces angiotensin I (AI) which is then converted, by angiotensin converting enzyme (ACE) to angiotensin II (AII) which creates vasoconstriction, reactive oxygen species (ROS) production, vascular and cardiac remodeling, production of aldosterone, and vascular inflammation. This mechanism is protective against hypovolemic hypotension, however, its activation also produces disease in the form of hypertension. Aldosterone interacts with renal epithelial cytosol sodium receptors and recruits them to the endothelial surface allowing them to increase sodium reabsorption creating an increase in plasma and, thus, an increase in BP.



- Fourteen genes have been identified as causing mendelian forms of hypertension and can account for as many as 70% of familial aggregation of hypertension (Bonow et al., 2012).
- DNA histone modification creates the underlying modifications in DNA expression and is the basis of epigenetics. These modifications related to many environmental factors can create hypertensive disease (Wise & Charchar, 2016).

- Endothelial cell dysfunction can be contributed in large part to the production of reactive oxygen species which reduces the availability of endothelial- derived relaxing factors including nitric oxide and endothelium- derived hyperpolarizing factor. This creates an increase in PVR. There is increased release of endothelial proinflammatory, constricting, growth and prothrombic factors.
- Vascular remodeling- hypertrophic gene expression leads to an increase in medial thickness in addition to accumulation of extracellular collagen from activation of tumor growth factor- beta (TGF-B) thus creating stiffness in the large arteries.



- The fundamental abnormality is the kidneys ability to excrete excessive sodium load. This can be acquired or inherited (Bonow et al., 2012).
- Sodium retention in the renal system increases plasma volume and also triggers an increase in systemic vascular resistance.
- Sodium excretion in hypertensive population is the same as that of normotensive individuals, however the excretion is completed at a higher BP.

- Hypertension effects 30% pf the population with incidences higher in vulnerable communities.
- Hypertension is a major modifiable risk factor for disease that cause morbidity and mortality including CAD and stroke.
- Nursing plays a major role in assisting patients in controlling their HTN.
- The pathophysiologic processes behind the disease are multifactorial and well-studied.
- Factors creating HTN include neuronal, hormonal, renal, vascular, genetic, and epigenetic.
- RASS plays a great role in the development of hypertension.
- Health care workers are in a position to assist patients in alteration of lifestyle to control hypertension.

Bonow, R., Mann, D., Zipes, D., Libby, P. (2012). *Braunwald's heart disease: A textbook of cardiovascular medicine*. Philadelphia: Saunders Elsevier.

United States Preventative Services Task Force. (2016). *Final recommendation statement: High blood pressure in adults: Screening*. Retrieved from <https://www.uspreventiveservicestaskforce.org/Pag/Document/Recommendationstatementfinal/high-blood-pressure-in-adults-screenin>

## Additional Resources

Kovell, L., Ahmed, H., Misra, S., Blumenthal, R., McEvoy, J. (2017). US hypertension management guidelines: A review of the recent past and recommendations for the future. *Journal of the American Heart Association*, 6(6), 1-11.  
<http://dx.doi.org/10.1161/JAHA.115.002315>

Mennuni, S., Rubattu, S., Pierelli, G., Tocci, G., Fofi, C., & Volpe, M. (2014). Hypertension and kidneys: Unraveling complex molecular mechanisms underlying hypertensive renal damage. *Journal of Human Hypertension*, 28(2), 74-79. <http://dx.doi.org/10.1038/jhh.2013.55>

Muñoz-Durango, N., Fuentes, C., Castillo, A., González-Gómez, L., Vecchiola, A., Fardella, C., & Kalergis, A. (2016). Role of the renin-angiotensin-aldosterone system beyond blood pressure regulation: Molecular and cellular mechanisms involved in end-organ damage during arterial hypertension. *International Journal of Molecular Sciences*, 17(7), 1-17. <http://dx.doi.org/10.3390/ijms17070797>



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