Cardiol Cardiovasc Med 2021; 5 (1): 57-60 *DOI: 10.26502/fccm.92920181*

Review of Literature

Essential Hypertension: Pathophysiology& Management (Current View)

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Received: 26 December 2020; **Accepted:** 04 January 2021; **Published:** 11 January 2021

Citation: Tarun Saxena, Azeema Ozefa Ali, Manjari Saxena. Essential Hypertension: Pathophysiology& Management (Current view). Cardiology and Cardiovascular Medicine 5 (2021): 57-60.

Abstract

The rising burden of essential hypertension necessitates the need for further understanding of pathophysiology. Recent studies have focused upon sympathetic over-activity. Sympathetic overactivity is associated with increased left ventricular contractility and various mechanisms of hypertension. Disturbed cortical impulses (stress) to the hypothalamus shifts the vasomotor center to a higher level resulting in increased basal sympathetic discharge and hypertension.

Keywords: Essential hypertension; Hypothalamus; Sympathetic system over-activity

Introduction

World- wide essential hypertension is an important cause of rising mortality and morbidity. Despite the availability of various drugs for hypertension, the epidemic is poorly controlled especially in developing countries. This indicates a further understanding of pathophysiology and treatment plan of hypertension. Multiple mechanisms have been proposed for pathophysiology including genetics, insulin resistance, Obstructive sleep apnea (OSA), Renin- angiotensin–aldosterone system (RAAS), sympathetic system overactivity, endothelial dysfunction, etc. Out of these mechanisms, the

sympathetic system over-activity has gained attention in newer studies. The simple reason is that the sympathetic system overactivity is linked to different mechanisms of hypertension including RAAS, OSA, insulin resistance, etc. This review focuses upon sympathetic overactivity (its origin, basal/overt discharge), its relation with pathophysiology, and a possible connection between its normalization and control of blood pressure. This mini-review also reemphasizes the central role of hypertension [1-10].

Review of literature

Pathophysiology of hypertension starts from chronically disturbed cortical impulses (neocortex, cingulate gyrus) (as evidenced in EEG, low voltage fast beta activity in eye closure state suggestive of desynchronized rhythm present in stress, fast mental speed, mental exhaustion), to the hypothalamus; leading to a shift of VMC (vasomotor center) at a higher level.

The shift of VMC is followed by high basal sympathetic discharge (according to VMC level) from the hypothalamus, leading to increased cardiac contractility (associated with high LV ejection force, short acceleration time in aortic flow in Echocardiography) and stage 1 hypertension. There is shifting of baroreceptors and renal mechanism to a new higher level (increase in stroke volume associated with high LV ejection force, short acceleration time in aortic flow in Echocardiography) and stage 2 hypertension i.e. a new higher value of B.P. is achieved.

Repetition of the same process shifts BP even higher. The findings are suggested by a study that found, high left ventricular ejection force (LVEFo), short acceleration time (AT) in aortic flow in all stages of hypertension.

There was significantly elevated sympathetic skin response (SSR) and normal stroke volume (SV) in stage1 hypertension and high stroke volume and normal sympathetic skin response in stage 2 hypertension respectively. The findings suggested that sympathetic discharge came back to normal after setting the blood pressure to a newer level. Resting pulse rate was within normal limits (no overt increase) in all the study subjects.

The sympathetic activity was supposed to play a role in the shift of blood pressure from normotensive to stage 1 and then to stage 2 and subsequently increased levels in stage 2. Sympathetic skin response is not a cord reflex its efferent pathway starts from the hypothalamus. In absence of an overt increase in sympathetic activity, SSR indicates baseline activity of the sympathetic system. This explains the central origin of elevated sympathetic rhythm and its role in the shift of blood pressure in different stages [11-13].

Other important mechanisms include the RAAS system i.e. high renin levels which increase angiotensin 1 and angiotensin 2 (a potent vasoconstrictor) and increase the activity of aldosterone which reabsorbs salt and water and increases B.P. is present in only 10-20 % of cases. Insulin resistance is associated with hypertension, is linked to physical inactivity, chronic stress, and increased basal sympathetic discharge (desynchronized EEG), and metabolic syndrome.

Obesity-related hypertension and sleep apnea are associated with sympathetic over-activity. Endothelial dysfunction is associated with hypertension. Sympathetic over-activity increases ATP demand in endothelial cells produces stiffness; also associated with coronary events [14].

Conclusion

Maximum association of essential hypertension is found with sympathetic overactivity which is directly or indirectly present in different mechanisms of hypertension including RAAS, OSA, obesity, etc. The disturbed basal sympathetic tone arises from the hypothalamus possibly has cortical influences. Various conditions like chronic stress, mental exhaustion may result in impaired cortical hypothalamic signals, the shift of vasomotor center to a higher level, increased basal sympathetic discharge, and hypertension.

Suggestions

1. Assessment of LVEFo, AT, SV, and SSR in unresponsive patients to antihypertensive therapy.
2. Use of drugs single or in combination which reduces cardiac contractility (LVEFo) e.g. beta- blocker, calcium channel blocker in all stages of hypertension.
3. The main focus should be at the central level to reduce elevated basal sympathetic discharge by correcting the altered cortical-hypothalamic signals/ axis. This can be achieved by assessment and correction of lifestyle factors i.e. chronic stress, mental exhaustion, fast mental speed, sleep pattern/duration.
4. There should be a balance between mental and physical work.
5. Use of centrally acting drugs, and various stress relaxation techniques like Vipassana, meditation which help in reducing centrally elevated sympathetic discharge [15-17].

Conflict of Interest: None

Funding Source: Mittal Hospital, Ajmer

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