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Annals of Clinical and Experimental Hypertension

Research Article

Possible Clinical Implications of High Left Ventricular Ejection Force and Exaggerated Sympathetic Skin Response in Hypertensive Patients

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Submitted: 25 February 2016

Accepted: 12 March 2016

Published: 15 March 2016

ISSN: 2373-9258

Keywords

- Left ventricular ejection force
- Pathophysiology of hypertension
- Basal sympathetic rhythm
- Cortical - hypothalamic axis

Abstract

Introduction: Incidence of hypertension is escalating rapidly; its pathophysiology still remains obscure. Recently high $LVEF_0$ and exaggerated sympathetic activity is found responsible for hypertension. Therefore it is needed to discuss clinical implications of these factors in pathophysiology andmanagement of hypertension.

Methods: $LVEF_0$ was assessed by Echocardiography and SSR with the help of EMG electrodes, in 100 normotensive (group 1) and equal number of hypertensive subjects (group 2). Stage 1 & stage 2 hypertension were categorized in group 2A & 2B respectively.

Results: $LVEF_0$ was high in stage 1 and stage 2 hypertension. High SSR was found in stage 1 hypertension.

Conclusion: High $LVEF_0$ is associated with hypertension.High SSR in stage 1 hypertension denotes high basal sympathetic rhythm, this in turn reflects alteration in cortical-hypothalamic axis. Management strategy should include reduction in LV contractility by pharmacological means or by correction at the level of cortical hypothalamic axis, and may include relaxation techniques.

ABBREVIATIONS

$LVEF_0$: Left Ventricular Ejection Force; SSR: Sympathetic Skin Response; EMG: Electro Myo Graphy

INTRODUCTION

Essential hypertension has no identifiable cause. It accounts for 95% of all hypertensive patients [1]. Despite awareness of various risk factors, pathophysiology of hypertension remains unexplained [2]. Recently role of high left ventricular ejection force **($LVEF_0$)** and high baseline sympathetic activity has been found to be responsible for hypertension [3]. Therefore the main aim of this communication is to analyze clinical implications of these parameters in understanding of pathophysiology of hypertension, correlation of sympathetic skin response with hypertension, possible management strategies of hypertension and, in brief discussion about the assessment methods and results of $LVEF_0$ and sympathetic skin response in hypertensive and normotensive subjects (based on reference study) [3].

MATERIALS AND METHODS

This was a case control study [3], subjects were divided into 2 groups, normotensive (group 1; control group) and hypertensive (group 2; cases). In group 2 subjects having stage 1 hypertension were categorized in group 2A while stage 2 hypertension was categorized in 2B. 100 cases and equal number of controls were taken.

Following parameters were examined

a) Resting pulse rate and respiratory rate

b) Left ventricular ejection force($LVEF_0$)

c) Sympathetic skin response (SSR)

d) Stroke volume (SV)

$LVEF_0$ was assessed with the help of image directed continuous wave Doppler echocardiography in a five chamber transthoracic view. Aortic peak systolic velocity (PSV),aortic

acceleration time (AT),aortic cross section area(CSA) and time velocity integral(TVIac) during the acceleration phase of cardiac cycle were measured and $LVEF_o$ was calculated by using a formula (1.055xCSAxTVIac)x PSV/AT.

Sympathetic skin response was measured with the help of EMG electrodes with standard protocol. Stimulation in the form of hand grip and cold presser was given in one arm and recording was done in other arm. Latency and amplitude was recorded with a computer.

Stroke volume was measured in a transthoracic four chamber view by subtracting ESV (end systolic volume) from EDV (end diastolic volume).

RESULTS

In group 2, 60 cases had stage 1 hypertension, while 40 cases had stage 2 hypertension. Short AT and high $LVEF_o$ was found in all stages of hypertension. Significantly high sympathetic skin response was present in stage 1 hypertension. Stroke volume was significantly high in stage 2 hypertension. Insignificant differencein stroke volume waspresent in normotensive and stage 1 hypertension (Table 1&2).

DISCUSSION

Incidence of hypertension is on rise [4]. Despite awareness, prevention and management plan, control of this epidemic remains a challenge. Therefore it becomes necessary to identify other risk factors/pathways in pathophysiology of hypertension. Recently high $LVEF_o$ and high SSR has been found to be responsible for hypertension [3].

We divide our discussion into three headings-

1) Pathophysiology of hypertension
2) Correlation of sympathetic skin response with hypertension
3) Possible management plan of hypertension

Table 1: Cardiac Findings.

Subjects	AT (10^{-3} s) mean	$LVEF_o$ (10^{-3} N) mean	SV (10^{-3} L) mean
Normotensive	92	0.351	42
Stage 1 hypertension	53	0.723	43
Stage 2 hypertension	44	1.448	60

Table 2: SSR Findings (cold presser test).

Subjects	Latency(s) mean	Amplitude (10^{-3})V mean
Normotensive	2.81	1.11
Stage 1 hypertension	0.80	6.63
Stage 2 hypertension	2.86	1.24

Abbreviations: N: Newton; L: Litters; S: Seconds; Lv: Left Ventricle; Sv: Stroke Volume; $Lvef_o$: Left Ventricular Ejection Force; At: Aortic Acceleration Time

Pathophysiology of hypertension

a) In stage 1 hypertension high $LVEF_o$ was associated with insignificant difference in stroke volume as compared to normotensive state.In other words, two persons hold equal amount of liquid. However one person throws it gently (LV of normotensive) while another throws it with great force (LV of stage 1 hypertension) against a wall(arterial) thus producing high pressure/high blood pressure in the second condition (Figure 2).

b) SSR was high in stage 1 hypertension and normal in stage 2 hypertension. High SSR represents high basal sympathetic rhythm and not overt sympathetic activity (resting pulse rate normal in all groups) [3].

c) Stage 1 hypertension/high SSR is intermediate stage between normotensive state and stage- 2 hypertension. Therefore sympathetic activity plays role in shifting of blood pressure from normotensive stage to stage 1 (adaptation of baroreceptors) and later on resetting of renal mechanism to reach to stage 2 hypertension (even if it comes back to normal at this stage).

d) Right now we have classified essential hypertension into two types, stage 1 and stage 2 as per JNC VII classification [5]. Referencestudy [3] guides us about the role of high baseline sympathetic activity in shifting of blood pressure. There might be multiple intermediate stages where sympathetic activity increases and fixes blood pressure to a newer level e.g. 160mm Hg to 180mm Hg then again after a period of time again resetting at a higher level e. g. 180 mm Hg to 200 mm Hg.

Therefore, for better understanding of pathophysiology of shifting and a reset to a newer level, short sub staging can be done for every 20-30 mmHg from normotensive stage/stage 1 hypertension to late stage 2 hypertension. (Further work is required to document this)

Correlation of sympathetic skin response with hypertension

High SSR is present in stage 1 hypertension.

Association of high SSR in hypertension suggests that hypertension is not merely a disease of circulatory system. In the absence of overt increase in sympathetic activity SSR primarily assess baseline activity of sympathetic rhythm/Autonomic nervous system(ANS).

Normally there is flow of impulses in sympathetic nervous system at a basal rate i.e. basal sympathetic rhythm/tone. (This tone is sufficient to maintain blood pressure at a normal level) Highest center of ANS is hypothalamus(homeostasis) which in turn is indirectly affected by cortical activity. (Neo cortex, cingulate gyrus) SSR is not simply a spinal cord reflex [3,6-9] (Figure 1).

In SSR stimulation is given from one arm and recording is done in other arm.Its efferent connections start from hypothalamus. Cerebral cortex has extensive connections with hypothalamus; hypothalamus receives afferent from limbic system (cingulate gyrus) neocortex and other areas (cortical-hypothalamic connections/axis). The hypothalamus (dorso medial nuclei and lateral hypothalamic area) gives efferent to pre

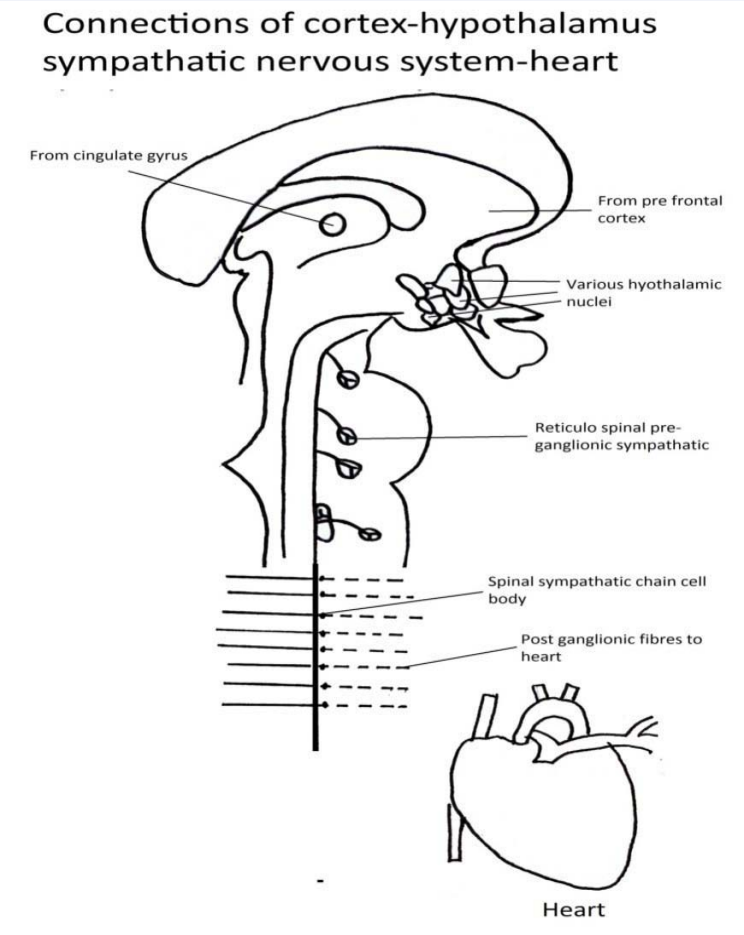

Figure 1 Connections of cortex-hypothalamus sympathetic nervous system-heart.

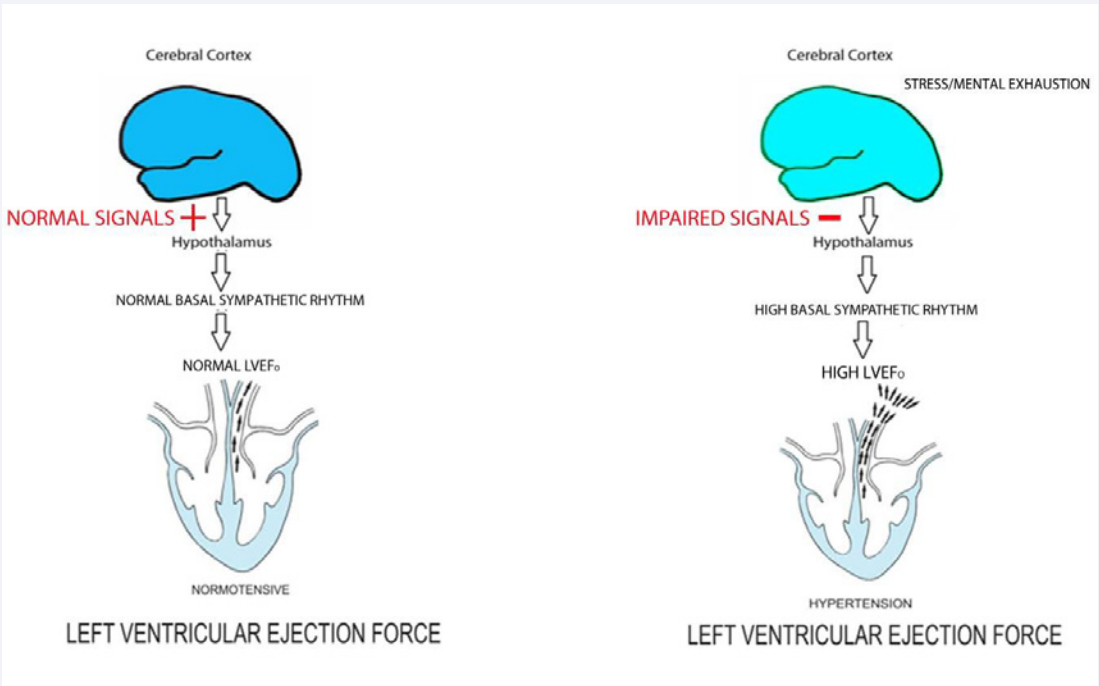

Figure 2 Left Ventricular Ejection Force.

ganglionic sympathetic neurons via reticulo-spinal tract; reaches to inter-medio lateral column of spinal cord (ends on cell body). From here post ganglionic sympathetic fibres arise from thoraco lumber (T1-L2) area of spinal cord. These fibres reaches to heart. Thus cortex has some indirect control (modulating effect over hypothalamus) over sympathetic nervous system (Figure 1) [8,9].

Possibly various conditions like chronic stress, mental exhaustion (more mental work and less mental rest), and fast mental speed [7,8] may result in impaired cortical-hypothalamic signals.Impaired hypothalamic signals lead to increased preganglionic and therefore increased post ganglionic basal sympathetic rhythm. Therefore this increased sympathetic activity leads to high $LVEF_o$ and stage 1 hypertension (Figure 1,2).

Possible management plan of hypertension

1) Assessment of $LVEF_o$, AT, SV and SSR in nonresponsive patients to antihypertensive therapy.

2) Even short AT may (40 to 50 m sec) indirectly suggest high $LVEF_o$.

3) In all stages of hypertension single/ combination of drugs e.g. calcium channel blocker, beta blocker which reduces left ventricular force of contraction should be given.

4) In addition to reduction in salt intake, alcohol consumption and weight reduction, long term adequate control of blood pressure may require normalization of basal sympathetic rhythm by correcting altered cortical-hypothalamic axis. Various relaxationtechniques/ taking sound sleep/ balancing mental/physical workcan be used for this purpose [10-13].

In addition to SSR, Cortical-hyothalamicaxis [8,9] /Cortical activity can be judged by EEG (Electro-encephalography). Fast de-synchronised rhythm (Low voltage, fast beta activity) in eye closure state suggests stress/ fast mental speed/ mental exhaustion [8,14,15]

Meditation (active concentration) techniques- These include, Omkara meditation, Zen meditation/Buddhist meditation(concentration on respiration, some mantra, or the picture of God/Godess) are useful in dealing with stress and fast mental speed.

Relaxation (No active concentration) techniques, such as Shavasana, Makrasana (relaxation postures where body and mind is allowed to relax), sound sleep andreduction in excess mental work (i.e. excess use of mobile, computers, office work) help in prevention/recovery from mental exhaustion.

CONCLUSION

Hypertension is not merely a disease of circulatory system alone .Its pathogenesis involves alteration in ANS (autonomic Nervous System) and likelyin cortical -hypothalamic axis. Assessment of ANS and Cortical-hypothalamic axis may be required for complete hypertension workup.

ACKNOWLEDGEMENT

AlokRaizada (clinical assistant).

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Cite this article

Saxena T, Saxena M (2016) Possible Clinical Implications of High Left Ventricular Ejection Force and Exaggerated Sympathetic Skin Response in Hypertensive Patients. Ann Clin Exp Hypertension 4(1): 1035.