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Original Article

Assessment of left ventricular ejection force and sympathetic skin response in normotensive and hypertensive subjects: A double-blind observational comparative case-control study

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ABSTRACT

Background: Pathophysiology of essential hypertension remains obscure. Correlation among ventricular ejection force, sympathetic activity, and hypertension is less clearly narrated in hypertensive subjects.

Aims and objectives: To assess correlation among ventricular ejection force, sympathetic activity, and hypertension in hypertensive subjects, and to be compared with normotensive subjects.

Methods: This is a case-control study to assess left ventricular ejection force (LVEF) and sympathetic skin response, in normotensive (group 1; control), and hypertensive subjects (group 2; cases). 100 cases were selected. Subjects having stages 1 and 2 hypertension were categorized in groups 2A and 2B, respectively. LVEF was calculated by using echocardiography observing aortic acceleration time (AT) and peak systolic velocity. Comparison among groups was done by using one-way ANOVA.

Results: Both groups were comparable. In group 2, 60 cases had stage 1 hypertension and 40 had stage 2 hypertension. Significantly short AT and significantly high LVEF were found in hypertension (groups 2A and 2B) ($p < 0.0001$). Sympathetic activity was high in group 2A ($p < 0.0001$). Stroke volume (SV) was high in group 2B ($p < 0.0001$).

Conclusion: Stage 1 hypertension is a stage of increased sympathetic activity, leading to increased LVEF and hypertension (resetting of baroreceptors); stage 2 hypertension is a stage of normal sympathetic activity, increased LVEF, increased SV, and hypertension (possibly a stage of shift of renal equilibrium curve/renal output curve and blood pressure to a newer level).

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1. Introduction

Essential hypertension¹ affecting 95% of hypertensive patients has no identifiable cause.²⁻⁴ According to Joint National Committee report (JNC 7), blood pressure $\geq 140/90$ mmHg is hypertension.⁵ Various risk factors associated with hypertension are obesity,⁶ salt sensitivity,⁷ genetics,⁸ obstructive sleep apnea,⁹ insulin resistance,¹⁰ sympathetic over activity,^{11,12} etc. Despite awareness of multiple risk factors, pathophysiology of hypertension remains ambiguous. In normotensive subjects, sympathetic stimulation results in increase in heart rate, cardiac contractility (ejection force), and peripheral resistance,¹³ but in hypertensive subjects, correlation among sympathetic activity, cardiac contractility and hypertension is less clearly illustrated. Therefore, the main aim of the study was to assess any correlation among these factors in hypertensive subjects and to be compared with normotensive subjects, in a case-control manner.

2. Methods

This is a double-blind comparative observational exploratory case-control study to assess left ventricular ejection force (LVEF), sympathetic nervous system activity, and stroke volume (SV) in normotensive (group 1; control group) and hypertensive subjects (group 2; cases). All the cases were newly diagnosed, i.e. no previous history of treatment of hypertension. Arbitrarily, 100 controls and an equal number of hypertensive cases were opted for the study. Informed written consent and approval of institutional ethical committee was taken. In group 2, subjects having stage 1 hypertension were categorized in group 2A while stage 2 hypertension was categorized in group 2B. Cases and controls were randomly selected from medical Out Patient Department (OPD) of Mittal Hospital and Research Centre, Pushkar Road, Ajmer, Rajasthan, India. Recruitment was done from January 2015 to March 2015. In both the groups, the age group was 35-40 years; all participants were male. Cases having coronary artery disease, thyroid disease, diabetes, left ventricular hypertrophy, aortic valvular disease, and history suggestive of neuropathy, which could affect echocardiographic findings/sympathetic skin response (SSR), were excluded. Assessment was done only once, i.e. at the time of first examination (0 month). The following parameters were examined

- (1) Complete examination including body mass index (BMI), resting pulse rate, and respiratory rate
- (2) Left ventricular ejection force (LVEF)
- (3) Sympathetic skin response (SSR)
- (4) Stroke volume (SV)

Left ventricular ejection force – This was assessed with the help of echocardiography by applying Newton's second law of motion.¹⁴ This law states that force is equal to the product of mass and acceleration. Ventricular ejection force does not require estimation of ventricular volume and is independent of ventricular configuration. Five-chamber

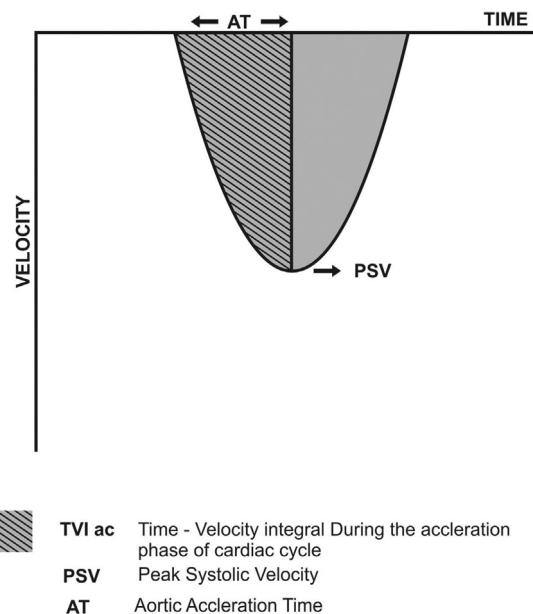


Fig. 1 – Aortic flow.

transthoracic view was used with the help of image-directed continuous wave Doppler echocardiography^{15,16} (GE Vivid S6 with probe frequency 1.7 MHz). Aortic peak systolic velocity (PSV), aortic acceleration time (AT)/time to peak velocity interval (TTP), time-velocity integral during the acceleration phase of the cardiac cycle (TVI_{ac}), and heart rate were measured. TVI_{ac} represents the area under the Doppler envelope from the beginning of systole to PSV (Fig. 1). Three consecutive cardiac cycles were examined and their mean was used for analysis. The diameter of aortic valve was measured from frozen real time images during systole by using leading edge to leading edge method. The mass of blood accelerated across aortic valve over a time period was calculated by multiplying the density of blood, which is 1.055, by the cross-sectional area (CSA) and TVI_{ac}. The acceleration component was calculated by dividing the PSV by TTP. LVEF was calculated by using a formula $(1.055 \times \text{CSA} \times \text{TVI}_{ac}) \times (\text{PSV}/\text{TTP})$.^{17,18}

Sympathetic skin response was done with standard protocol in supine, relaxed, semi-darkened room with ambient temperature control at 22-24 °C in the upper limbs. In this process, standard surface electromyography electrodes (Recorder and Medicare system model Aleron 401) were applied with conducting jelly to the palm and dorsum of the hand, with a reference electrode on the forearm. Hand grip and cold pressor were used as provocative methods. Skin potential changes during and between the tests were analyzed by a computer. Latency was measured from the onset of stimulus artifact to the beginning of response. Amplitude was recorded peak to peak (Fig. 2).¹⁹⁻²¹

All readings of echocardiography and SSR were recorded by a senior resident and a senior technician.

Stroke volume was measured in transthoracic 4-chamber view by subtracting ESV (end systolic volume) from EDV (end diastolic volume).

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