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ORIGINAL RESEARCH

Heart rate variability in hypertensive and normal individuals

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ABSTRACT

Background: An ongoing autonomic nervous system imbalance is a common and significant risk factor for fatal cardiovascular events as well as other severe cardiovascular events. The present study was conducted to assess heart rate variability in hypertensive and normotensive individuals. **Materials & Methods:** 72 hypertensive patients of both genderswere put in group I and normotensive subjects in group II. HRV such as total power (TP), normalized low frequency power (LFnu), normalized high frequency power (HFnu), ratio of low frequency power to high frequency power (LF-HF ratio), standard deviation of normal-to-normal RR intervals (SDNN), root mean square successive difference (rMSSD) and the proportion of NN50 to the total number of NN intervals (pNN50) were recorded. **Results:** Group I had 35 males and 37 females and group II had 36 males and 36 females.SDNN (ms) was 152.2 in group I and 140.6 in group II, pNN50 was 13.5 in group I and 10.4 in group II, rMSSD (ms) was 45.6 in group I and 40.2 in group II. LFnu was 76.0 in group I and 85.4 in group II, HFnu was 53.2 in group I and 37.5 in group II and LF/HF ration was 2.57 in group I and 3.96 in group II. The difference was significant (P< 0.05). **Conclusion:** When compared to participants with normotension, hypertension patients showed reduced parasympathetic modulation and heart rate variability.

Key words: autonomic nervous system, arterial hypertension, heart rate variability

INTRODUCTION

An ongoing autonomic nervous system imbalance is a common and significant risk factor for fatal cardiovascular events as well as other severe cardiovascular events.[1] It is reasonable to assume that any element that causes the sympathetic nervous system to activate inappropriately will negatively impact these measurements. Anything that increases vagal tone often leads to better results.[2] The autonomic nervous system (ANS) is thought to be a key pathophysiologic component in the development of arterial hypertension since it is essential to the regulation of heart rate and arterial blood pressure. Heart rate variability research can currently be used to determine the state of the heart's autonomic functioning.[3] Because the ANS constantly promotes adjustments to maintain the cardiovascular system, heart rate fluctuates per beat.These changes can be measured by comparing the R-R interval variations, which together make up the heart rate variability. Heart rate variability is determined by the integration of the parasympathetic and sympathetic modulations.[4]
The top causes of death globally are cancer, diabetes mellitus, hypertension, hyperlipidemia, coronary heart disease, and obesity. Diabetes mellitus and cardiovascular disease are highly prevalent in emerging nations.[5] Physical inactivity is one of the main risk factors for cardiovascular illnesses and higher cardiovascular mortality among the other risk factors. It is estimated that globally, physical inactivity is the primary cause of roughly 21-25% of colon and breast cancers, 27% of diabetes cases, and roughly 30% of ischemic heart disease cases.[6]The present study was conducted to assess heart rate variability in hypertensive and normotensive individuals.

MATERIALS & METHODS

The present study comprised of72 hypertensive patients of both genders. All subjects were informed and their written consent was obtained.
Data such as name, age, gender etc. were recorded. A thorough clinical examination was done. Analysis of blood pressurewas done. Hypertensive patients were put in group I and normotensive subjects in group II. HRV such as total power (TP), normalized low frequency power (LFnu), normalized high frequency power (HFnu), ratio of low frequency power to high

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frequency power (LF-HF ratio), standard deviation of normal-to-normal RR intervals (SDNN), root mean square successive difference (rMSSD) and the proportion of NN50 to the total number of NN intervals (pNN50) were recorded. Results of the study was recorded and subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of subjects

Groups	Group I	Group II
M:F	35:37	36:36

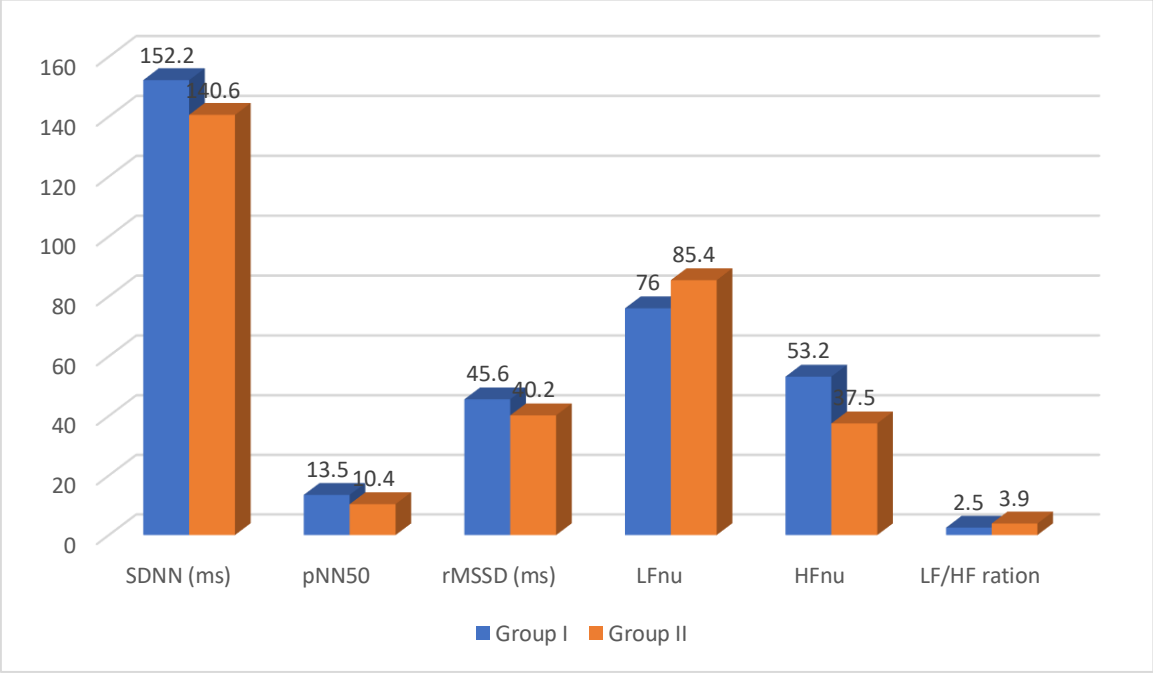
Table I shows that group I had 35 males and 37 females and group II had 36 males and 36 females.

Table II Assessment of domain

Parameters	Group I	Group II	P value
SDNN (ms)	152.2	140.6	0.05
pNN50	13.5	10.4	0.01
rMSSD (ms)	45.6	40.2	0.05
LFnu	76.0	85.4	0.02
HFnu	53.2	37.5	0.01
LF/HF ration	2.5	3.9	0.05

Table II, graph I shows that SDNN (ms) was 152.2 in group I and 140.6 in group II, pNN50 was 13.5 in group I and 10.4 in group II, rMSSD (ms) was 45.6 in group I and 40.2 in group II. LFnu was 76.0 in group I and 85.4 in group II, HFnu was 53.2 in group I and 37.5 in group II and LF/HF ration was 2.57 in group I and 3.96 in group II. The difference was significant (P< 0.05).

Graph I Assessment of domain



DISCUSSION

Heart rate (HR) and cardiac muscle contraction (inotropic activity) are two of the ways that the parasympathetic and sympathetic nervous systems (PNS and SNS) regulate cardiac innervation.[7] Peripheral vascular resistance is caused by the SNS, which also controls the peripheral vasculature.[8] Blood pressure (BP) is also mediated by the baroreceptor reflex (BR), which is mediated by the SNS. Cardiovascular autonomous dysfunction is a major driving shift of the autonomous function in primary hypertension.[9] It is characterized by an imbalance between sympathetic and parasympathetic activity with diminished vagal (parasympathetic) tone and increased peripheral sympathetic activity.[10] As a result, surrogate measures of autonomic control research, like HRV, could prove helpful in monitoring the development of hypertensive disorders.[11,12]The present study was conducted to assess heart rate variability in hypertensive and normotensive individuals.

We found that group I had 35 males and 37 females and group II had 36 males and 36 females. Singh et al[13]compared measures of HRV between hypertensive

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and normotensive subjects and examined the role of HRV as a predictor of new-onset hypertension. The first 2 hours of ambulatory ECG recordings obtained from 931 men and 1111 women attending a routine examination at the Framingham Heart Study were processed for HRV. Three time-domain and 5 frequency-domain variables were studied: standard deviation of normal RR intervals (SDNN), percentage of differences between adjacent normal RR intervals exceeding 50 milliseconds, square root of the mean of squared differences between adjacent normal RR intervals, total power (0.01 to 0.40 Hz), high frequency power (HF, 0.15 to 0.40 Hz), low frequency power (LF, 0.04 to 0.15 Hz), very low frequency power (0.01 to 0.04 Hz), and LF/HF ratio. On cross-sectional analysis, HRV was significantly lower in hypertensive men and women. Among 633 men and 801 women who were normotensive at baseline (systolic blood pressure <140 mm Hg and diastolic blood pressure <90 mm Hg and not receiving antihypertensive treatment), 119 men and 125 women were newly hypertensive at follow-up 4 years later. After adjustment for factors associated with hypertension, multiple logistic regression analysis revealed that LF was associated with incident hypertension in men (odds ratio per SD decrement [OR], 1.38; 95% confidence interval [CI], 1.04 to 1.83) but not in women (OR, 1.12; 95% CI, 0.86 to 1.46). SDNN, HF, and LF/HF were not associated with hypertension in either sex. HRV is reduced in men and women with systemic hypertension. Among normotensive men, lower HRV was associated with greater risk for developing hypertension.

We found that SDNN (ms) was 152.2 in group I and 140.6 in group II, pNN50 was 13.5 in group I and 10.4 in group II, rMSSD (ms) was 45.6 in group I and 40.2 in group II. LFnu was 76.0 in group I and 85.4 in group II, HFnu was 53.2 in group I and 37.5 in group II and LF/HF ration was 2.57 in group I and 3.96 in group II. Piccirillo G et al[14]showed that adrenergic receptor stimulation can induce left ventricular hypertrophy. Using an autoregressive algorithm in a power spectrum analysis of heart-rate variability in 14 subjects with mild hypertension (mean age 41 ± 9.0 years) and 9 age-matched normotensives They compared autonomic nervous system function at baseline (rest) and during sympathetic stress (passive head-up tilt). The spectrum comprised four spectral frequency-domains: total power (0.0033–0.40 Hz), high-frequency power (0.16–0.40 Hz), low-frequency power (0.04–0.15 Hz) and very-low-frequency power (0.0033–0.04). The high-frequency spectral component predominantly reflects vagal activity, the low-frequency component sympathetic nervous system activity. The ratio between low- and high-frequency power expresses the sympathovagal balance. Results were expressed as natural logarithms of power and normalized units. In addition, we compared spectral densities obtained, with the left ventricular mass index evaluated by M-mode echocardiography. Hypertensive subjects had greater low-frequency and low-high frequency ratio values ($P < 0.001$) than normotensive controls. They also had a low capacity for increase after tilt. Multiple regression analysis showed that the left-ventricular mass index was independently associated with the body mass index ($P < 0.0027$), very-low frequency ($P < 0.043$), and low frequency ($P < 0.0138$) expressed as the natural logarithm, low-high frequency ratio ($P < 0.0172$) and systolic blood pressure ($P < 0.0353$). Our findings confirm enhanced sympathetic activity in hypertensive subjects.

CONCLUSION

Authors found that when compared to participants with normotension, hypertension patients showed reduced parasympathetic modulation and heart rate variability.

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