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## ORIGINAL RESEARCH

# Evaluation of Cardiac Autonomic Functions in Prehypertensive individuals with and without a Family History of Hypertension: A Cross-Sectional Study

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## Abstract

**Background:** A blood pressure reading of 140/90 mmHg is considered hypertension, according to the Joint National Committee's seventh report (JNC 7), which was presented in 2003 at the annual scientific meeting of the American Society of Hypertension.

**Materials and Methods:** The present cross-sectional study included 50 prehypertensive individuals without a family history of hypertension and 50 prehypertensive individuals with a family history of hypertension of both genders, aged between 18 and 60 years.

**Results:**The mean age of the participants in group I was 35.50±5.07 years and in group II was 34.34±5.80 years. Group I consisted of 34 men and 16 women, while Group II consisted of 30 men and 20 women. There was no significant difference in age and BMI between the two study groups

**Conclusion:** The cardiac autonomic function tests in the present study show that the sympathovagal balance has been impaired in prehypertensive subjects with a family history of hypertension. The deep breathing test and VR show parasympathetic withdrawal, while the time domain and frequency domain HRV parameters show sympathetic overactivity.

**Keywords:** Heart rate variability, Sympathovagal balance, Prehypertensive

## Introduction

By 2020, one billion or more adults globally—more than 31.5% of the total adult population—will suffer from high blood pressure.[1] A blood pressure reading of 140/90 mmHg is considered hypertension, according to the Joint National Committee's seventh report (JNC 7), which was presented in 2003 at the annual scientific meeting of the American Society of Hypertension. "Prehypertension" is characterised as having a systolic pressure of 120–139 mmHg or a diastolic pressure of 80–89 mmHg.[2] People are predisposed to hypertension by a number of risk factors, the most significant of which is their family history, which is not changeable.[3] About thirty percent of the variance in blood pressure can be attributed to genetic factors; twin studies show a range of 25–65%.[4,5] Individuals who have a family history of hypertension might also be more susceptible due to common environments and other potential risk factors. [6] When hereditary variables are combined with other risk factors, including smoking, obesity, and dyslipidemia, the chance of prehypertension might increase. [7]However, the Autonomic Nervous System (ANS) has long been known to regulate cardiovascular processes and have an influence on blood pressure, both at rest and in response to stimuli from the outside, according to physiological research on the cardiovascular system.[8,9]

## Aim and objectives

The aim of the present study was to evaluate cardiac autonomic functions in prehypertensive individuals with and without a family history of hypertension.

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**Materials and Methods**

The present cross-sectional study conducted at the Department of Physiology, ShriAtalBihari Vajpayee Medical College and Research Institute, Bengaluru, Karnataka, India in collaboration with Department of General Medicine, after obtaining ethical clearance from the Institutional Ethical Clearance Committee. The period of study was from September 1st, 2019 to April 1st 2020.

The present study included 50 prehypertensive individuals without a family history of hypertension and 50 prehypertensive individuals with a family history of hypertension of both genders, aged between 18 and 60 years. All were informed regarding the study, and their written consent was obtained. Data such as name, age, gender, etc. was recorded.

**Inclusion Criteria**

- Patients who give written informed consent.
- Patient's age between 18 and 60 years.
- All prehypertensive indivudual with a SBP of 120 to 139 mmHg and a DBP of 80 to 89 mmHg, regardless of gender
- Available for follow-up.

**Exclusion Criteria**

- Patients do not give written informed consent.
- Individuals with a history of systemic disorders such as diabetes mellitus, cardiovascular diseases, respiratory, hepatic, renal, or neurological diseases, hypothyroidism.
- Pregnant women
- smokers, alcoholics individuals
- Patients with immunocompromised status and patients on chemotherapy or steroid treatment.
- Those unable to attend follow-up.

**Sampling Size Determination**

The following simple formula would be used for calculating the adequate sample size in a prevalence study:

$n = Z^2 P (1-P)/d^2$

n = sample size, Z = level of confidence, P = prevalence, and d = absolute error or precision.

Z = standard normal variate (at 5% type 1 error (P< 0.05) it is 1.96 and at 1% type 1 error (P<0.01) it is 2.58). As in the majority of studies, P values are considered significant below 0.05, hence 1.96 is used in the formula. p = expected proportion in population based on previous studies or pilot studies.

The sample size was calculated using a single population proportion formula by considering a 95% confidence level, a 5% margin of error, and a 6% estimated proportion of overall prevalence.

Sample size = 1.962 × 0.06 (1-0.06)/0.052

=86

Considering a 10% non-response rate, the total minimum sample size for the study was 95 patients. We included 100 patients with acute abdomens in the present study.

All 100 prehypertensive subjects were divided into two groups based on the presence or absence of a family history:

**Group I**: 50prehypertensive individuals without a family history of hypertension.

**Group II:** 50 prehypertensive individuals with a family history of hypertension were enrolled. Family history of hypertension refers to a parental history of hypertension, either with one parent or both parents.[9]

**Methodology**

Anthropometric parameters were measured, including height, weight, and body mass index (BMI). A digital blood pressure monitor was used to measure the SBP, DBP, and basal heart rate in a supine position following a five-minute rest. On both arms, BP and HR were measured twice, with a five-minute break in between. Next, the four recordings' mean was calculated.Before the start of each test, a baseline recording of breathing, an ECG, and the simultaneo us R-R interval were made for 30 seconds. A 15-minute Rest in the supine position was given before each test.

**Statistical Analysis**

The statistical analysis was conducted using SPSS version 25.0. For categorical variables, ratios and proportions were calculated. The chi-square test, if appropriate, was used to evaluate differences in proportions among qualitative variables. The Student's t-test was used for statistical analysis as the

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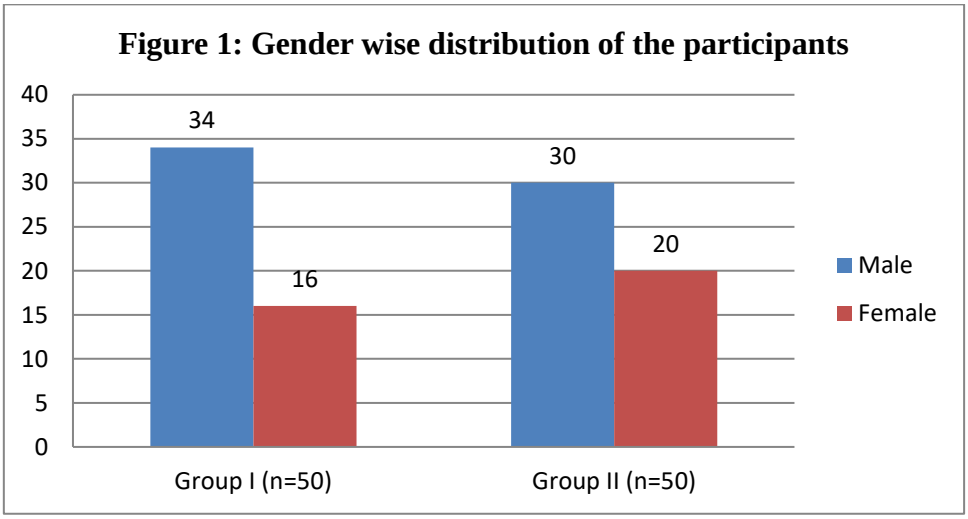
test of significance at a 95% confidence level. A p-value below 0.05 was deemed to have statistical significance.

**Results**

**Table 1: Gender wise distribution of participants**

| Gender | Group I (n=50) | Group II (n=50) |
|---|---|---|
| Male | 34 | 30 |
| Female | 16 | 20 |

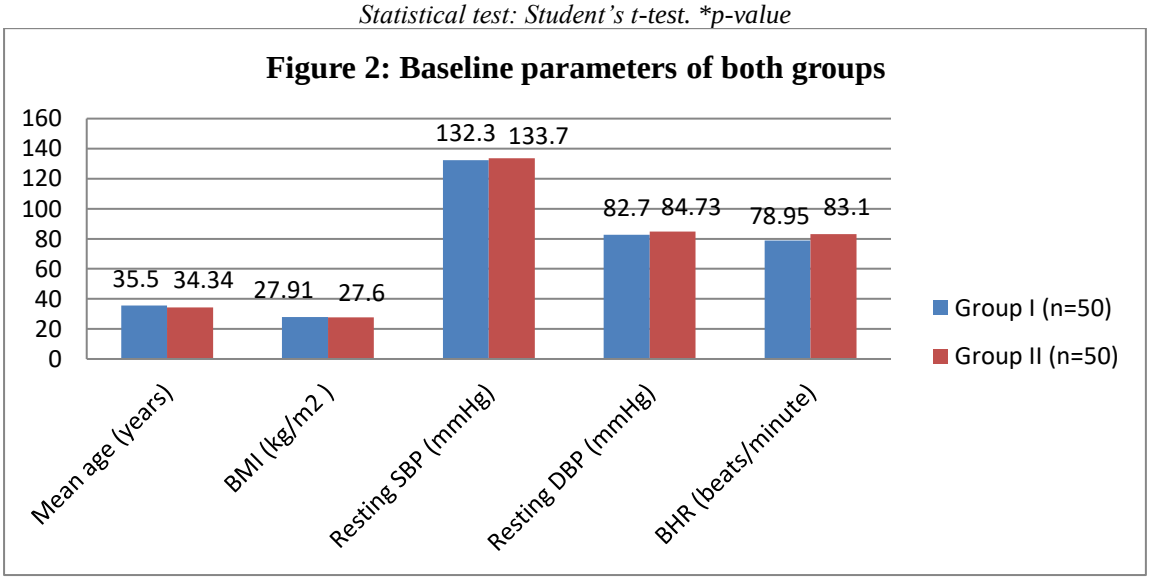



A total of 50 prehypertensives without a family history of hypertension and 50 prehypertensives with a family history of hypertension, aged 18 to 60 years, were included. Group I consisted of 34 men and 16 women, while Group II consisted of 30 men and 20 women. The proportions of gender in the two groups showed that they were comparable. In the present study, there was no significant difference in BMI between the two study groups [Table-1].

**Table -2: Comparison of baseline characteristics among both the study groups**

| Parameters | Group I (n=50) | Group II (n=50) | P value |
|---|---|---|---|
| Mean age (years) | 35.50±5.07 | 34.34±5.80 | 0.71 |
| BMI (kg/m2 ) | 27.91±2.65 | 27.60±3.59 | 0.85 |
| Resting SBP (mmHg) | 132.30±4.10 | 133.70±3.92 | 0.01 |
| Resting DBP (mmHg) | 82.70±4.65 | 84.73±4.91 | 0.03 |
| BHR (beats/minute) | 78.95±4.80 | 83.10±4.50 | 0.001 |

*BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BHR: Basal heart rate; Statistical test: Student's t-test. *p-value*



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In the present study, the mean age of the participants in group I was 35.50±5.07 years and in group II was 34.34±5.80 years. There was no significant difference in age and BMI between the two study groups. The BHR (beats/minute)of the participants in group I was 78.95±4.80and in group II was83.10±4.50beats/minute respectively. The difference was significant (p=0.001) [Table 2, Figure 2].

**Table -3: Comparison of time domain and frequency domain characteristics of Heart Rate Variability (HRV) among the study groups**

| Parameters | Group I (n=50) | Group II (n=50) | P value |
|---|---|---|---|
| Mean RR (ms) | 761.34±32.86 | 715.30±43.01 | 0.001 |
| RMSSD | 29.91±2.72 | 25.38±2.52 | 0.001 |
| pRR50 | 7.85±0.91 | 7.50±0.60 | 0.01 |
| TP ($ms^2$) | 861.05±120.30 | 815.20±102.75 | 0.02 |
| LF (nu) | 54.30±5.39 | 57.26±4.18 | 0.002 |
| HF (nu) | 29.51±2.61 | 29.34±2.10 | 0.61 |
| LF:HF ratio | 1.9±0.18 | 1.8±0.15 | 0.01 |

*RR: RR interval; RMSSD: root mean square of successive differences between normal heartbeats; pRR50: percentage of adjacent RR intervals that differ from each other by more than 50 ms; TP: total power; LF (nu): normalised low frequency power; HF (nu): normalised high frequency power; statistical test: student's t-test.*

When comparing Group II to Group I, the time domain variables mean RR, RMSSD, and pRR50 showed a significant reduction. Group II had a significant reduction in total power, a frequency domain variable, in comparison with Group I. In Group II, there was a significant rise in the LF nu. Despite being elevated in Group II, the HF nu was not statistically significant (Table 3).

**Discussion**

The age differences between the two groups were insignificant. Although there was no significant difference in BMI between the two groups, Group I's elevated BMI of 27.91±2.65 kg/m2, which falls within the overweight category of the BMI classification, may have been attributable to the rise in resting blood pressure. Group II's elevated resting blood pressure could be explained by the presence of a family history of hypertension. This explains the necessity of looking at these people's lipid profiles, catecholamine levels, and other variables. The elevated resting SBP and DBP are similar to the results of Arun Kumar B and Nirmala N.[10]

Group II's higher basal heart rate than Group I may have resulted from a genetic predisposition in prehypertensive subjects with a family history of hypertension, which alters vagal regulation. This was comparable to Pal GK's findings.[11]

One measure of the cardiovascular system's and the autonomic nervous system's ability to adapt to environmental changes is the high variability of the RR interval, one of the time-domain variables of resting heart rate. As a result, the lower mean RR in Group II than in Group I may be associated with ANS integrity being impaired by Group II's genetic predisposition. Research by Pitzalis MV found a comparable drop in mean RR in normotensives with a family history.[12]

In Present Study, when comparing Group II to Group I, the time domain variables mean RR, RMSSD, and pRR50 showed a significant reduction. Group II had a significant reduction in total power, a frequency domain variable, in comparison with Group I. In Group II, there was a significant rise in the LF nu. Despite being elevated in Group II, the HF nu was not statistically significant. RMSSD is an important short-term measure of parasympathetic drive since it reflects the vagal regulation of heart rate. Poor vagal regulation in Group II is reflected in the considerable fall in RMSSD observed in that group. The percentage of adjacent RR intervals that differ by more than 50 ms, or pRR50, is closely correlated with parasympathetic activity. According to the results, Group II's vagal activity was lower than Group I's. Short-term resting HRV monitoring was used in the present study to calculate the time domain variables; however, it was not as accurate as 24-hour Holter monitoring. However, compared to Group I, Group II shows significant parasympathetic withdrawal, as shown by a decrease in RMSSD and pRR50. Subjects with a family history showed changes in time domain variables, according to a study by Jha A et al.[13]

A highly significant variation in frequency domain parameters is revealed by the present study. The LF and HF values are influenced by total power, which is the sum of the four spectral bands and

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shows how much variation is overall. Normalised HF and LF values were used with the objective to minimise this effect. Group II had more sympathetic activity than Group I, as indicated by the significantly higher LF (nu) in Group II. The HF (nu) value, which measures parasympathetic activity, did not significantly differ between the two groups. Group I's and Group II's LF:HF ratios, which are reliable for predictingsympathovagal balance, were 1.90±0.18 and 1.8±0.15, respectively. The LF:HF ratio's p-value of 0.01 showed a significant overall sympathovagal imbalance in both groups, with Group II showing significantly higher parasympathetic withdrawal and sympathetic overactivity. This was consistent with the findings of Pal GK[14] and Wadoo OK et al.[15]

**Limitation(s) of the study**

The small sample size and short study duration are the study's limitations. Limitations on time limited the collection of 24-hour ambulatory HRV measurement data on physical activity, salt intake, and serum lipid profile.

**Conclusion(s)**

The cardiac autonomic function tests in the present study show that the sympathovagal balance has been impaired in prehypertensive subjects with a family history of hypertension. The deep breathing test and VR show parasympathetic withdrawal, while the time domain and frequency domain HRV parameters show sympathetic overactivity. More investigation regarding other risk factors influencing blood pressure is also needed. A prospective study that included follow-up following lifestyle modifications might shed further light on the ANS's integrity. A catecholamine assay, which has been shown to be a direct measure of the sympathetic nervous system, may have also been performed.

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