

**Evaluation of Autonomic Nervous System Dysfunction in Hypertension: A Cross-sectional Study of Cardiovascular Responses**

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**Abstract**

**Background:** Hypertension is one of the leading causes of cardiovascular morbidity and mortality worldwide. Autonomic nervous system dysfunction, characterized by increased sympathetic activity and reduced parasympathetic activity, plays an important role in the pathogenesis and progression of hypertension. Cardiovascular autonomic function tests and heart rate variability (HRV) analysis are useful non-invasive tools for evaluating autonomic dysfunction in hypertensive patients.

**Methods:** The present hospital-based cross-sectional study was conducted on 100 participants, including 50 hypertensive patients and 50 normotensive controls. Baseline demographic and clinical parameters including age, sex, body mass index (BMI), blood pressure, and pulse rate were recorded. Cardiovascular autonomic function was assessed using heart rate response to deep breathing, Valsalva maneuver, 30:15 ratio, blood pressure response to standing, and sustained handgrip test. HRV parameters were analyzed using time-domain and frequency-domain measures. Statistical analysis was performed using independent t-test, Chi-square test, and Pearson correlation test.

**Results:** The mean age of hypertensive patients was  $49.8 \pm 8.2$  years and that of controls was  $46.7 \pm 7.9$  years. Hypertensive patients had significantly higher BMI, systolic blood pressure, diastolic blood pressure, and pulse rate than controls ( $p < 0.001$ ). E:I ratio, Valsalva ratio, and 30:15 ratio were significantly lower in hypertensive patients ( $p < 0.001$ ). Hypertensive individuals also showed a greater fall in systolic blood pressure on standing and a lower rise in diastolic blood pressure during sustained handgrip testing ( $p < 0.001$ ). HRV parameters including SDNN, RMSSD, pNN50, and HF power were significantly reduced, whereas LF power and LF/HF ratio were significantly increased in hypertensive patients ( $p < 0.001$ ).

**Conclusion:** Hypertensive patients demonstrated significant autonomic dysfunction characterized by sympathetic overactivity and reduced parasympathetic modulation. Cardiovascular autonomic function tests and HRV analysis may be useful for early identification of hypertensive patients at higher cardiovascular risk.

**Keywords:** Hypertension, Autonomic Nervous System Dysfunction, Heart Rate Variability, Cardiovascular Reflex Tests, Sympathovagal Imbalance.

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**Introduction**

Hypertension is one of the most common non-communicable diseases worldwide and remains a leading cause of cardiovascular morbidity and mortality. It affects nearly 1.13 billion individuals globally and significantly increases the risk of stroke, myocardial infarction, heart failure, peripheral arterial disease, chronic kidney disease, and premature death [1,2]. The burden of hypertension is particularly high in low- and middle-income countries, where delayed diagnosis, poor treatment adherence, and inadequate blood pressure control are common [3,4]. In addition, the prevalence of hypertension increases with advancing age and is frequently associated with

other cardiovascular risk factors such as diabetes mellitus, obesity, dyslipidemia, and chronic kidney disease [1,5,6]. The pathogenesis of hypertension is complex and multifactorial, involving genetic, environmental, behavioral, renal, vascular, and neurohumoral mechanisms. Among these, dysfunction of the autonomic nervous system (ANS) has emerged as an important contributor to the development and progression of hypertension [7,8]. The ANS plays a central role in cardiovascular regulation by maintaining a balance between sympathetic and parasympathetic activity. Under normal physiological conditions, sympathetic stimulation increases heart rate,

myocardial contractility, and peripheral vascular resistance, whereas parasympathetic activity exerts inhibitory effects on cardiac function and promotes cardiovascular stability [8,9]. Proper balance between these two components is essential for maintaining normal blood pressure and cardiovascular homeostasis [9].

In hypertensive individuals, this autonomic balance is often disturbed, leading to increased sympathetic activity and reduced parasympathetic tone [7,8]. Persistent sympathetic overactivity contributes to vasoconstriction, elevated cardiac output, sodium and water retention, activation of the renin-angiotensin-aldosterone system, and vascular remodeling, all of which contribute to the maintenance of high blood pressure [3,4,7]. At the same time, reduced parasympathetic activity and impaired baroreceptor sensitivity decrease the ability of the cardiovascular system to adapt to fluctuations in blood pressure. These autonomic abnormalities may further predispose hypertensive patients to arrhythmias, arterial stiffness, left ventricular hypertrophy, and adverse cardiovascular outcomes [8,9]. Heart rate variability (HRV), which refers to the variation in the time interval between consecutive heartbeats, is considered one of the most reliable non-invasive indicators of autonomic nervous system activity [9]. HRV analysis provides important information regarding the balance between sympathetic and parasympathetic influences on cardiac function. Reduced HRV is commonly observed in hypertensive patients and reflects impaired autonomic regulation [8,9]. Studies have shown that low HRV is associated with an increased risk of myocardial infarction, stroke, arrhythmias, and sudden cardiac death. Therefore, assessment of HRV may be useful in identifying early autonomic dysfunction and predicting cardiovascular risk in patients with hypertension [8,9]. Apart from HRV, several cardiovascular autonomic function tests are available for evaluating autonomic dysfunction in hypertensive patients.

These include heart rate response to deep breathing, Valsalva maneuver, blood pressure response to standing, orthostatic hypotension assessment, sustained handgrip test, and postural blood pressure changes. These tests help assess both sympathetic and parasympathetic functions and may detect early autonomic imbalance before the development of overt cardiovascular complications [8,9]. Recognition of the role of autonomic dysfunction in hypertension has led to the development of therapeutic approaches aimed at reducing sympathetic overactivity and improving parasympathetic function. Lifestyle modifications such as regular exercise, weight reduction, stress management, smoking cessation, and dietary improvement have been shown to improve

autonomic balance and HRV. Similarly, certain pharmacological agents including beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and centrally acting sympatholytic drugs may influence autonomic function and improve cardiovascular outcomes [10,11]. Despite growing evidence regarding the relationship between autonomic dysfunction and hypertension, further studies are still needed to better understand the extent of autonomic imbalance and its cardiovascular implications in hypertensive individuals. Therefore, the present study was conducted to evaluate autonomic nervous system dysfunction in patients with hypertension by assessing various cardiovascular responses and heart rate variability parameters and comparing them with normotensive controls.

## Materials and Methods

**Study Design and Setting:** The present study was conducted as a hospital-based cross-sectional observational study in the Department of Physiology at a tertiary care teaching hospital. The study was carried out over a period of one year

**Study Population:** The study included adult participants aged between 30 and 60 years. A total of 100 participants were enrolled in the study and were divided into two groups:

- Group I: 50 diagnosed hypertensive patients
- Group II: 50 normotensive healthy controls

Hypertensive patients were recruited from the outpatient and inpatient departments of General Medicine. Healthy normotensive controls were selected from hospital staff, attendants, and healthy volunteers.

## Inclusion Criteria

### Hypertensive Group

- Patients aged between 30 and 60 years
- Patients diagnosed with essential hypertension according to standard guidelines
- Patients with systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg or those already receiving antihypertensive treatment
- Patients willing to participate in the study

### Control Group

- Age- and sex-matched normotensive individuals
- Systolic blood pressure  $< 120$  mmHg and diastolic blood pressure  $< 80$  mmHg
- Individuals without any known cardiovascular, metabolic, neurological, or systemic illness
- Individuals willing to participate in the study

## Exclusion Criteria

- Patients with secondary hypertension

- Patients with diabetes mellitus
- Patients with ischemic heart disease, heart failure, arrhythmias, or valvular heart disease
- Patients with chronic kidney disease or chronic liver disease
- Patients with neurological disorders affecting autonomic function
- Patients receiving drugs known to influence autonomic nervous system activity such as beta-blockers, antidepressants, sedatives, or anticholinergic agents
- Smokers and alcohol-dependent individuals
- Pregnant women
- Patients unwilling to participate in the study

**Data Collection Procedure:** After obtaining written informed consent, detailed demographic and clinical information was collected from all participants. Data regarding age, sex, duration of hypertension, body mass index, blood pressure, pulse rate, and medication history were recorded. Blood pressure was measured using a standard mercury sphygmomanometer after the participant had rested for at least 10 minutes in the sitting position. Three readings were taken at an interval of 5 minutes, and the average value was considered for analysis.

**Assessment of Autonomic Function:** Cardiovascular autonomic function was assessed using standard non-invasive autonomic function tests.

**Heart Rate Response to Deep Breathing:** Participants were instructed to breathe deeply at a rate of six breaths per minute. The maximum and minimum heart rates during inspiration and expiration were recorded using electrocardiography, and the E:I ratio was calculated.

**Valsalva Maneuver:** Participants were asked to blow into a mouthpiece attached to a manometer and maintain a pressure of 40 mmHg for 15 seconds. Heart rate changes during and after the maneuver were recorded, and the Valsalva ratio was calculated.

**Heart Rate Response to Standing:** The 30:15 ratio was measured after the participant moved from a supine to standing position. The ratio of the longest R-R interval around the 30th beat to the shortest R-R interval around the 15th beat was calculated.

**Blood Pressure Response to Standing:** Blood pressure was measured in the supine position and again immediately after standing and after 2 minutes. The fall in systolic blood pressure on standing was recorded.

**Sustained Handgrip Test:** Participants were asked to maintain handgrip at 30% of their maximum voluntary contraction using a handgrip dynamometer for 3 minutes. The rise in diastolic blood pressure during the test was recorded.

**Heart Rate Variability Analysis:** Heart rate variability was assessed using a standard electrocardiographic recording in resting supine position. A 5-minute ECG recording was obtained in a quiet room under controlled conditions.

#### Time-Domain Parameters

The following time-domain parameters were recorded:

- Mean heart rate
- Mean R-R interval
- SDNN (standard deviation of normal-to-normal intervals)
- RMSSD (root mean square of successive differences)
- pNN50 (percentage of adjacent R-R intervals differing by more than 50 ms)

#### Frequency-Domain Parameters

The following frequency-domain parameters were analyzed:

- Low-frequency power (LF)
- High-frequency power (HF)
- LF/HF ratio

**Non-Linear Parameters:** Non-linear HRV parameters such as SD1 and SD2 were also evaluated.

**Statistical Analysis:** The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25.0. Continuous variables were expressed as mean  $\pm$  standard deviation, whereas categorical variables were expressed as frequency and percentage.

Comparison between hypertensive patients and normotensive controls was performed using the independent t-test for continuous variables and Chi-square test for categorical variables. Correlation between blood pressure parameters and autonomic function variables was assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

#### Results

A total of 100 participants were included in the study, comprising 50 hypertensive patients and 50 normotensive controls.

**Table 1: Age Distribution of Study Participants**

Age Group (Years)	Hypertensive Group (n=50)	Control Group (n=50)	Total (n=100)	p-value
30–39	10 (20.0%)	14 (28.0%)	24 (24.0%)	
40–49	18 (36.0%)	20 (40.0%)	38 (38.0%)	
50–60	22 (44.0%)	16 (32.0%)	38 (38.0%)	0.36

The majority of participants in the hypertensive group belonged to the age group of 50–60 years (44.0%), whereas most controls were in the age group of 40–49 years (40.0%). However, the difference in age group distribution between the two groups was not statistically significant (Chi-square test,  $p = 0.36$ ). The mean age in the

hypertensive group was  $49.8 \pm 8.2$  years, compared to  $46.7 \pm 7.9$  years in the control group. The median age was 50 years (IQR: 43–56) in the hypertensive group and 46 years (IQR: 40–53) in the control group. The difference in mean age between the two groups was not statistically significant (Independent t-test,  $p = 0.07$ ).

**Table 2: Sex Distribution of Study Participants**

Sex	Hypertensive Group (n=50)	Control Group (n=50)	Total (n=100)	p-value
Male	31 (62.0%)	29 (58.0%)	60 (60.0%)	
Female	19 (38.0%)	21 (42.0%)	40 (40.0%)	0.68

Male participants constituted the majority in both groups, accounting for 62.0% of hypertensive patients and 58.0% of controls. There was no statistically significant difference in sex distribution between the groups (Chi-square test,  $p = 0.68$ ).

**Table 3: Body Mass Index of Study Participants**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Mean BMI (kg/m <sup>2</sup> )	$28.4 \pm 3.7$	$24.6 \pm 3.1$	<0.001
Median BMI (kg/m <sup>2</sup> )	28.1 (IQR: 25.8–30.6)	24.4 (IQR: 22.5–26.7)	

The mean BMI was significantly higher in the hypertensive group compared to the control group ( $28.4 \pm 3.7$  kg/m<sup>2</sup> vs  $24.6 \pm 3.1$  kg/m<sup>2</sup>). The median BMI was also higher in hypertensive patients. This difference was statistically significant (Independent t-test,  $p < 0.001$ ).

**Table 4: Baseline Blood Pressure and Pulse Rate**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Systolic Blood Pressure (mmHg)	$148.6 \pm 12.8$	$116.2 \pm 8.7$	<0.001
Median Systolic BP	150 (IQR: 140–158)	116 (IQR: 110–122)	
Diastolic Blood Pressure (mmHg)	$94.8 \pm 8.6$	$74.3 \pm 6.4$	<0.001
Median Diastolic BP	95 (IQR: 88–100)	74 (IQR: 70–79)	
Pulse Rate (beats/min)	$84.5 \pm 9.1$	$76.8 \pm 7.4$	<0.001
Median Pulse Rate	84 (IQR: 78–91)	76 (IQR: 71–82)	

Hypertensive patients had significantly higher systolic blood pressure, diastolic blood pressure, and pulse rate compared to normotensive controls. All differences were statistically significant (Independent t-test,  $p < 0.001$ ).

**Table 5: Heart Rate Response to Deep Breathing**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
E:I Ratio	$1.11 \pm 0.08$	$1.23 \pm 0.10$	<0.001
Median E:I Ratio	1.10 (IQR: 1.06–1.16)	1.22 (IQR: 1.17–1.29)	

The mean E:I ratio was significantly lower in hypertensive patients than in normotensive controls, indicating reduced parasympathetic activity in the hypertensive group (Independent t-test,  $p < 0.001$ ).

**Table 6: Valsalva Ratio in Study Participants**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Valsalva Ratio	$1.29 \pm 0.14$	$1.52 \pm 0.18$	<0.001
Median Valsalva Ratio	1.28 (IQR: 1.18–1.39)	1.50 (IQR: 1.39–1.64)	

The mean Valsalva ratio was significantly reduced in hypertensive patients compared to controls, suggesting impaired autonomic responsiveness among hypertensive individuals (Independent t-test,  $p < 0.001$ ).

**Table 7: Heart Rate Response to Standing (30:15 Ratio)**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
30:15 Ratio	$1.02 \pm 0.07$	$1.18 \pm 0.09$	<0.001
Median 30:15 Ratio	1.01 (IQR: 0.97–1.07)	1.17 (IQR: 1.12–1.24)	

The 30:15 ratio was significantly lower in the hypertensive group than in controls, indicating impaired parasympathetic function in hypertensive patients (Independent t-test,  $p < 0.001$ ).

**Table 8: Blood Pressure Response to Standing**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Fall in Systolic BP on Standing (mmHg)	12.4 ± 4.8	6.1 ± 3.2	<0.001
Median Fall in Systolic BP	12 (IQR: 9–15)	6 (IQR: 4–8)	

Hypertensive patients showed a significantly greater fall in systolic blood pressure on standing compared to controls, reflecting impaired sympathetic compensation (Independent t-test,  $p < 0.001$ ).

**Table 9: Sustained Handgrip Test**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Rise in Diastolic BP (mmHg)	10.8 ± 4.1	16.5 ± 4.8	<0.001
Median Rise in Diastolic BP	10 (IQR: 8–13)	16 (IQR: 13–20)	

The increase in diastolic blood pressure during the sustained handgrip test was significantly lower in hypertensive patients than in controls, indicating reduced sympathetic function (Independent t-test,  $p < 0.001$ ).

**Table 10: Time-Domain Heart Rate Variability Parameters**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
Mean RR Interval (ms)	712.4 ± 85.3	785.8 ± 76.9	<0.001
SDNN (ms)	31.2 ± 10.4	48.7 ± 12.8	<0.001
RMSSD (ms)	21.6 ± 8.7	39.8 ± 11.5	<0.001
pNN50 (%)	6.8 ± 3.9	14.7 ± 5.8	<0.001

All time-domain HRV parameters were significantly lower in hypertensive patients compared to controls, indicating impaired autonomic regulation and reduced parasympathetic activity (Independent t-test,  $p < 0.001$ ).

**Table 11: Frequency-Domain Heart Rate Variability Parameters**

Parameter	Hypertensive Group (n=50)	Control Group (n=50)	p-value
LF Power (ms <sup>2</sup> )	512.8 ± 148.6	438.7 ± 126.4	0.01
HF Power (ms <sup>2</sup> )	218.5 ± 82.4	376.2 ± 105.7	<0.001
LF/HF Ratio	2.41 ± 0.86	1.24 ± 0.51	<0.001

Hypertensive patients had significantly higher LF power and LF/HF ratio, whereas HF power was significantly lower compared to controls. These findings suggested increased sympathetic activity and reduced parasympathetic modulation in hypertensive individuals (Independent t-test).

**Table 12: Correlation between Systolic Blood Pressure and HRV Parameters in Hypertensive Patients**

Parameter	Correlation Coefficient (r)	p-value
SDNN	-0.48	<0.001
RMSSD	-0.52	<0.001
LF/HF Ratio	0.46	0.001

Systolic blood pressure showed a significant negative correlation with SDNN and RMSSD and a significant positive correlation with LF/HF ratio.

These findings indicated that higher blood pressure levels were associated with worsening autonomic dysfunction in hypertensive patients (Pearson correlation test).

## Discussion

In the present study, the mean age of hypertensive patients was 49.8 ± 8.2 years compared to 46.7 ± 7.9 years among normotensive controls, although the difference was not statistically significant ( $p = 0.07$ ). Male predominance was observed in both groups, with males accounting for 62.0% of hypertensive patients and 58.0% of controls. The mean BMI was significantly higher in hypertensive patients than controls (28.4 ± 3.7 kg/m<sup>2</sup> vs 24.6 ±

3.1 kg/m<sup>2</sup>,  $p < 0.001$ ), indicating a greater burden of obesity among hypertensive individuals. These findings were consistent with studies showing that hypertension is more prevalent in middle-aged and older adults and is strongly associated with obesity and metabolic risk factors. DeMarco et al. reported that obesity contributed substantially to hypertension through increased sympathetic activation, insulin resistance, and vascular dysfunction. Similarly, Coats and Cruickshank found that hypertensive patients with metabolic disturbances had greater sympathetic nervous system activation and worse cardiovascular risk profiles [12]. In the present study, hypertensive patients had significantly lower E:I ratio (1.11 ± 0.08 vs 1.23 ± 0.10,  $p < 0.001$ ), Valsalva ratio (1.29 ± 0.14 vs 1.52 ± 0.18,  $p < 0.001$ ), and 30:15 ratio (1.02 ± 0.07 vs 1.18 ± 0.09,  $p < 0.001$ ) compared to

normotensive controls. These findings indicated impaired parasympathetic function in hypertensive individuals. Hypertensive patients also demonstrated a significantly greater fall in systolic blood pressure on standing ( $12.4 \pm 4.8$  mmHg vs  $6.1 \pm 3.2$  mmHg,  $p < 0.001$ ) and a significantly lower rise in diastolic blood pressure during sustained handgrip testing ( $10.8 \pm 4.1$  mmHg vs  $16.5 \pm 4.8$  mmHg,  $p < 0.001$ ), suggesting impaired sympathetic responsiveness. These findings were in agreement with previous studies showing impaired cardiovascular reflex responses in hypertensive patients. Cardiovascular function tests such as heart rate response to deep breathing, Valsalva ratio, and 30:15 ratio have been widely recognized as measures of parasympathetic function, whereas blood pressure response to standing and sustained handgrip are considered indicators of sympathetic function. Pandian et al. reported that deep breathing test, Valsalva maneuver, and 30:15 ratio were useful indicators of cardiovagal dysfunction, while handgrip and orthostatic blood pressure responses reflected adrenergic dysfunction. Similar findings were also described in autonomic assessment studies that demonstrated reduced E:I ratio, Valsalva ratio, and 30:15 ratio in subjects with autonomic imbalance [13]. In the present study, the mean RR interval was significantly lower in hypertensive patients than controls ( $712.4 \pm 85.3$  ms vs  $785.8 \pm 76.9$  ms,  $p < 0.001$ ). Similarly, SDNN, RMSSD, and pNN50 were significantly reduced in hypertensive patients. The mean SDNN was  $31.2 \pm 10.4$  ms in hypertensive patients compared to  $48.7 \pm 12.8$  ms in controls, while RMSSD was  $21.6 \pm 8.7$  ms versus  $39.8 \pm 11.5$  ms, respectively ( $p < 0.001$  for both). The pNN50 was also lower in hypertensive individuals ( $6.8 \pm 3.9\%$  vs  $14.7 \pm 5.8\%$ ,  $p < 0.001$ ). These findings suggested a reduction in global HRV and diminished parasympathetic modulation in hypertensive patients. Similar results were reported by Julario et al., who found that median SDNN was 109 ms in hypertensive patients compared to 129 ms in non-hypertensive subjects. They also reported significantly lower rMSSD and pNN50 values in hypertensive patients. Shah et al. similarly observed that SDNN and total power were significantly reduced in hypertensive individuals compared to normotensive adults. De Andrade et al. also reported lower SDNN, TINN, and SD1/SD2 ratio in elderly hypertensive patients, indicating reduced overall HRV [14]. In the present study, LF power was significantly higher in hypertensive patients than controls ( $512.8 \pm 148.6$  ms<sup>2</sup> vs  $438.7 \pm 126.4$  ms<sup>2</sup>,  $p = 0.01$ ), whereas HF power was significantly lower ( $218.5 \pm 82.4$  ms<sup>2</sup> vs  $376.2 \pm 105.7$  ms<sup>2</sup>,  $p < 0.001$ ). The LF/HF ratio was markedly elevated in hypertensive patients ( $2.41 \pm 0.86$  vs  $1.24 \pm 0.51$ ,  $p < 0.001$ ). These findings indicated increased sympathetic activity

with reduced parasympathetic modulation among hypertensive individuals. Similar observations were reported in studies of hypertensive and pre-hypertensive individuals, where HF power was reduced and LF/HF ratio was elevated, suggesting sympathovagal imbalance. A recent study among normotensive offspring of hypertensive parents reported HF power values of  $420 \pm 120$  ms<sup>2</sup> in the study group compared to  $620 \pm 150$  ms<sup>2</sup> in controls, while LF/HF ratio increased from  $0.94 \pm 0.28$  in controls to  $1.82 \pm 0.45$  in the study group. Maciorowska et al. also emphasized that hypertension was associated with altered HRV parameters reflecting autonomic dysfunction and adverse hemodynamic changes [15]. In the present study, systolic blood pressure showed a significant negative correlation with SDNN ( $r = -0.48$ ,  $p < 0.001$ ) and RMSSD ( $r = -0.52$ ,  $p < 0.001$ ), whereas LF/HF ratio showed a positive correlation with systolic blood pressure ( $r = 0.46$ ,  $p = 0.001$ ). These findings suggested that increasing blood pressure levels were associated with worsening autonomic dysfunction. Previous studies also demonstrated similar associations between higher blood pressure and lower HRV indices. Maciorowska et al. reported that autonomic dysfunction was closely related to the hemodynamic profile of hypertensive patients and that worsening HRV indices were associated with poorer blood pressure control. Farheen et al. further demonstrated that higher LF/HF ratio and reduced HRV were independently associated with vascular dysfunction and elevated cardiovascular risk [16].

#### Conclusion:

Hypertensive patients showed significant autonomic dysfunction compared to normotensive controls. Parasympathetic activity was reduced, while sympathetic activity was increased in hypertensive individuals. Cardiovascular reflex tests and HRV parameters demonstrated impaired autonomic regulation in hypertension. Higher blood pressure levels were associated with worsening autonomic imbalance. Overall findings: Early assessment of autonomic dysfunction may help identify hypertensive patients at higher cardiovascular risk and improve management strategies.

#### Limitation:

The present study had certain limitations. It was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the findings. As the study had a cross-sectional design, a causal relationship between hypertension and autonomic dysfunction could not be established. Only short-term HRV recordings were used, while 24-hour monitoring was not performed. In addition, factors such as duration of hypertension, lifestyle habits, and the

effect of different antihypertensive medications were not evaluated separately.

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