

Original Article Cardiovascular

Utility of HRV, BRS, and BPV to Evaluate the Physiological Response to Antihypertensive Therapy in Women with Hypertension

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ABSTRACT

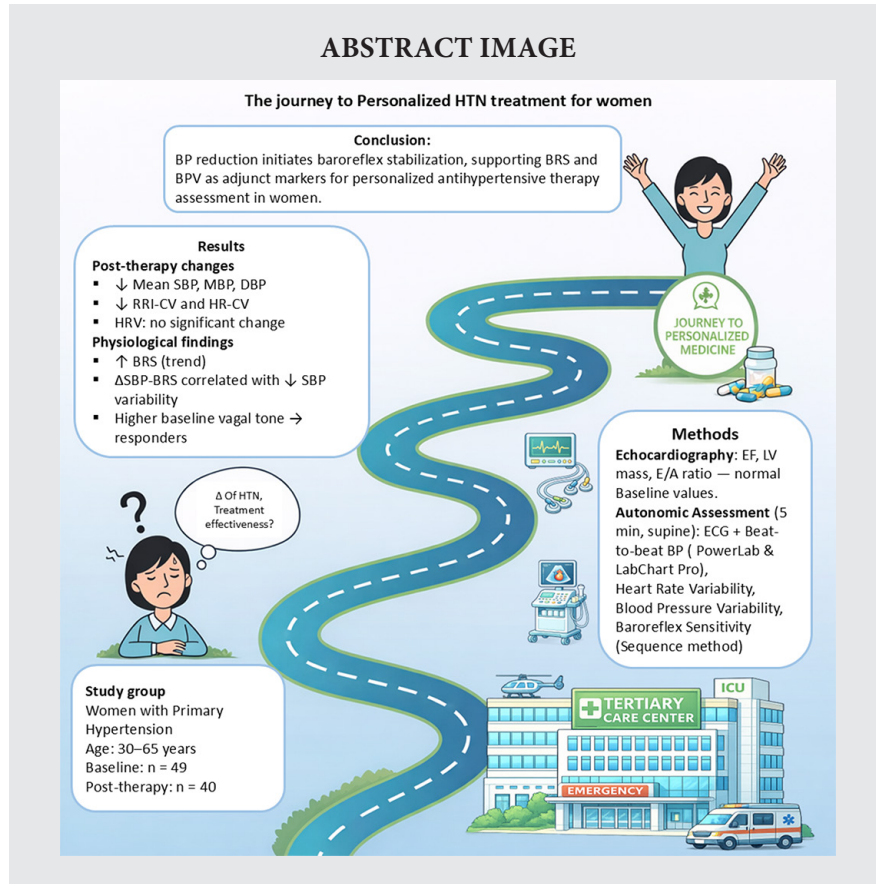
Objectives: To evaluate the non-invasive autonomic function parameters heart rate variability, baroreceptor sensitivity, blood pressure variability (HRV, BRS, and BPV) in assessing the physiological suitability of antihypertensive therapy in women with primary hypertension.

Materials and Methods: Forty-nine individuals were assessed at baseline for HRV, BRS, and BPV using validated non-invasive methods. Post-medication follow-up was completed in forty participants. Time- and frequency-domain HRV measures, BRS indices, and BPV characteristics were analyzed. Echocardiographic parameters including ejection fraction (EF), left ventricular (LV) mass, and early-to-atrial filling velocity ratio (EA ratio) were evaluated. Correlation analysis was performed to assess relationships between autonomic indices and cardiovascular measures.

Results: Antihypertensive therapy resulted in significant reductions in systolic, mean, and diastolic BP ($P < 0.001$) and a decrease in RRI and HRV indices, indicating improved autonomic regulation. Although HRV measures did not show statistically significant changes, BRS indices demonstrated an upward trend following treatment. Correlation analyses revealed a significant association between changes in BRS and changes in systolic BPV ($P = 0.008$), while age showed a significant negative association with baroreflex improvement ($P = 0.009$).

Conclusion: Antihypertensive therapy reduces pressure load and improves autonomic stability. While HRV indices do not change significantly over the short term, BRS and BPV demonstrate favorable physiological adjustments. Autonomic markers, particularly BRS, may provide valuable insights into treatment response beyond conventional BP monitoring.

Keywords: Antihypertensive therapy, Autonomic indices, Baroreflex sensitivity, Blood pressure variability, Heart rate variability



INTRODUCTION

Blood pressure (BP) oscillations, occurring over seconds to years, arise due to hemodynamic changes, neuronal reflex responses, and dietary influences.^[1] Physiologically, BP variability (BPV) contributes to cardiovascular homeostasis and reflects autonomic regulation. Reduced oscillatory variability in BP may suggest potential autonomic cardiovascular dysfunction.^[2,3] Heart rate variability (HRV) is recognized as a surrogate of autonomic balance and as a prognostic indicator in hypertension.^[2] Elevated BPV has been proposed as a therapeutic target for lifestyle and pharmacological intervention.^[4]

BPV is commonly described using frequency-based measures and simple dispersion indices such as standard deviation (SD) and coefficient of variation. It can be assessed over different time scales, ranging from beat-to-beat fluctuations to daily, 24-h, and long-term changes over years.^[5] Spectral techniques are relevant for beat-to-beat recordings, while weighted standard deviation (SD) and average real variability (ARV) have been proposed to overcome confounding influences of nocturnal dipping in 24-h indices. Notably, the prognostic value of BPV indices varies, outcome-based evidence supports SD for

clinic and home BPV, and ARV or weighted SD for 24-h BPV.^[6]

The noninvasive assessment of baroreflex–heart rate (HR) coupling by quantifying spontaneous beat-to-beat fluctuations in BP and RR intervals (RRI), performed under supine resting conditions for <10 min, allows detailed evaluation of autonomic feedback mechanisms.^[7] Development of noninvasive tonometric devices (human NIBP nanosystem) improved accessibility of BRS assessment and has shown strong correlation with invasive BP recordings and equivalent prognostic ability.^[1,8,9] The spontaneous sequence method identifies three or more consecutive cardiac cycles in which progressive rises or falls in systolic BP (SBP) are accompanied by parallel RRI lengthening or shortening.^[10]

Spectral BRS estimation quantifies beat-to-beat gain based on oscillations in low frequency (LF) (≈ 0.1 Hz) and high frequency (HF) (respiratory), provided coherence exceeds 0.5. Variations of spectral algorithms include autoregressive and transfer-function approaches.^[8] Reduced reliability of spectral BRS may occur in conditions with diminished BPV or frequent ectopy.^[11]

Physiologically, the arterial baroreceptor reflex provides rapid buffering of arterial pressure fluctuations through

reciprocal vagal-sympathetic modulation.^[12,13] Respiration interacts continuously with baroreflex-mediated vagal modulation (respiratory gating).^[12,14] Carotid baroreceptors appear to dominate baroreflex control, and carotid sinus denervation results in pronounced increases in BPV.^[15] Hypertension involves reduced BRS, persistent sympathetic activation, and disrupted cardiovascular balance.^[16] Blunted BRS is associated with elevated cardiovascular risk and worsened prognosis.^[17] BRS reflects RRI per unit change in BP, typically expressed in ms/mmHg. Average values around 15 ms/mmHg have been reported in healthy subjects.^[18] Age and BP strongly influence BRS, likely due to reduced arterial compliance. BRS also correlates with several cardiometabolic risk parameters, including SBP, diastolic BP (DBP), pulse pressure, cholesterol, LDL, triglycerides, and blood glucose.^[16,19]

Thus, BRS may function as an integrated cardiovascular risk indicator with potential predictive utility. HRV, BRS, and BPV are valuable autonomic indicators for evaluating antihypertensive treatment efficacy in hypertensive women. By monitoring changes over time and identifying poor responders, the study sought to determine whether these non-invasive autonomic function markers can aid in assessing the efficacy and tolerability of antihypertensive treatment in women with primary hypertension.

MATERIALS AND METHODS

Study design

This was a prospective observational study conducted in the Autonomic Function Testing Lab, Department of Physiology.

Participants

We studied 49 women aged 30–65 with newly diagnosed primary hypertension to capture early autonomic changes. Diagnosis used standard clinical sphygmomanometer readings based on American Heart Association (AHA) guidelines.^[20] Baseline evaluation involved Autonomic Indices and 2D-echocardiography before initiation of antihypertensive therapy. Participants, on stable antihypertensive therapy for at least 3 months, were reassessed after 3 months using artifact-free recordings to evaluate treatment effects. To minimize confounding, autonomic tests were standardized with fasting, rest, and hormonal phase control. Inclusion and exclusion criteria are shown in Figure 1. The study followed the Declaration of Helsinki and was approved by the Institutional Ethics Committee (IEC Ref. No: AIIMS/BBN/IEC/Apr/2025/667-R). Patient confidentiality was maintained throughout the study, and written informed consent was obtained from all participants.

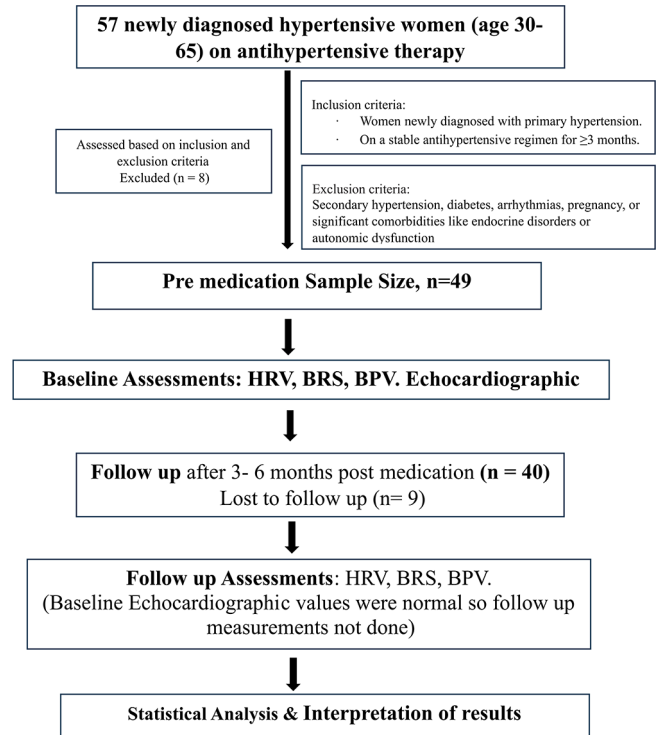


Figure 1: Methodology flowchart. (HRV: Heart rate variability, BRS: Baroreflex sensitivity, BPV: Blood pressure variability).

Recordings

Autonomic indices

HRV, baroreflex sensitivity (BRS), and BPV were recorded simultaneously using ADInstruments PowerLab with LabChart Version 8 Pro (Sydney, Australia) under controlled lab conditions. The equipment has been validated by multiple previous studies.^[9] Recordings occurred between 9:00 a.m. and 1:00 p.m. in a quiet, dimly lit, temperature-controlled room. Participants rested supine for 20–30 min; breathing was paced at 12–15 breaths/min with the help of mobile based digital metronome. Electrocardiogram (ECG) electrodes were placed per standard protocol, and NIBP was measured through a finger cuff at 1000 Hz. The first 1–2 min was excluded, and a stable 5-min segment was used for analysis. LabChart handled filtering, peak detection, and artifact removal.

2D-Echo

Prospective transthoracic echocardiography was performed using a Philips Affinity 70 system (Washington, United States) with standard parasternal and apical views obtained in the left lateral decubitus position according to American Society of Echocardiography (ASE) guidelines.

Parameters

HRV analysis

HRV was analyzed from a clean, artifact-free, resting 5-min RRI, per Task Force guidelines.^[21] R-wave detection was visually checked, with ectopic/noisy beats removed. Time- and frequency-domain indices were calculated and exported to Excel for analysis.

BRS analysis

A synchronized, clean, artifact-free (ectopic/noisy beats), resting 5-min Electrocardiography (ECG) and beat-to-beat BP segment was analyzed. Data free from respiratory/postural effects and signal artifacts (e.g., lead detachment) were used. LabChart data were exported as “.txt” and opened in Nevrokard.

Sequence method criteria: RRI change >0.5 ms; SBP change >2 mmHg; Sequence length >3 beats; • Correlation >0.6; SBP–RRI delay: 2 beats.

The software detected natural baroreflex sequences where SBP and RRIs rose or fell together. BRS was estimated from the slope of their relationship, using only well-correlated sequences ($r \geq 0.6$). The method aligns with principles validated in intra-arterial recordings and is physiologically supported in previous studies.^[20]

BPV analysis

Asynchronized clean, artifact-free (ectopic/noisy beats), resting 5-min ECG and beat-to-beat BP segment was analyzed from LabChart to assess short-term variability in time and frequency domains. Data unaffected by respiratory/postural changes or artifacts were exported as “.txt” and opened in Nevrokard. The software analyzed BP waveform dispersion and oscillations. Time-domain metrics and frequency-domain values were derived. These measures reflect autonomic vascular modulation and have recognized methodological and physiological relevance, as highlighted in prior publications on BPV.^[22] BPV and BRS values, obtained from the same BP–RR pairs, ensured physiological coherence and were exported with HRV data for statistical analysis.

Drug therapy

The patient whoever included in the study were started on oral monotherapy with telmisartan 40 mg (once daily) after the first visit and baseline autonomic and echocardiographic assessment. Telmisartan provides continuous 24-h control and is especially beneficial during high-risk early morning hours.^[23]

Statistical analysis

Data were first checked for normal distribution using the Kolmogorov–Smirnov test and are presented as mean \pm SD. Comparisons were made using appropriate parametric or non-parametric tests, with statistical significance set at $p < 0.05$, [Tables 1 and 2]. Relationships between variables were examined using correlation analysis [Figure 2]. All analyses were performed using Jamovi statistical software version 2.5.6.

For analytical purposes, participants were *post hoc* classified into responder and non-responder subgroups based on (i) achieved post-treatment systolic BP and (ii) magnitude of improvement in BRS. The thresholds were derived from the observed data distribution [Table 3] supported by physiological relevance and prior literature.^[24]

RESULTS

The 49 participants had a mean age of 47.46 ± 9 years. Clinical SBP and DBP were 134.4 ± 13.72 mmHg and 86.4 ± 9.49 mmHg, respectively. Baseline echocardiography showed a mean ejection fraction (EF) of $63.71 \pm 2.22\%$, left ventricular (LV) mass of 142.58 ± 18.18 g, and early to late mitral inflow (E/A) ratio of 1.09 ± 0.30 . As baseline cardiac structure and function were normal, post-treatment echocardiographic assessment was not repeated.

No statistically significant changes were found in time-domain HRV indices (standard deviation of R-R intervals, standard deviation of successive differences, root mean square of successive differences [RMSSD], percentage of pairs of successive RR intervals >50 msec [pRR50]) or frequency-domain metrics (total power, Low Frequency [LF], High Frequency [HF], Low Frequency/ High Frequency [LF/HF]) pre- and post-medication. However, LF/HF showed a declining trend (2.24 ± 4.53 – 1.53 ± 1.43 , $P = 0.06$) [Table 1].

BPV and BRS showed physiologically significant changes following medication. Significant reduction in mean systolic (SBP), mean arterial (MBP), and mean DBP were found in post-medication analyses (all $P < 0.001$). Following medication, both RRI-CV and HR-CV were significantly lower [Table 2].

All BRS mean values of BRS (SBP-BRS, MBP-BRS, DBP-BRS) showed post-treatment improvement, but none of these changes were statistically significant ($P > 0.05$) [Table 2].

According to correlation analysis, there was a significant positive correlation between the pre-post change of the same individual (Δ) in systolic BPV (Δ CV-SBP) and the change in SBP-BRS (Δ SBP-BRS) ($r = 0.375$, $P = 0.008$). Δ SBP-BRS showed a positive correlation with age ($r = 0.380$, $P = 0.009$). In addition, Δ SBP-BRS did not significantly correlate with baseline EF, LV mass, or the LF/HF ratio [Figure 2].

Table 1: Baseline (Pre- medication) and post medication heart rate variability indices.

HRV Parameters in study group			
Characteristic	Pre- medication <i>n</i> =49 (Mean±SD)	Post medication <i>n</i> =40 (Mean±SD)	<i>P</i> -value (Kruskal-Wallis Test)
Time domain			
Average RR (ms)	730.10±103.08	719.65±142.14	0.806
SDRR (ms)	29.92±17.35	29.08±15.55	0.605
SDSD (ms)	24.27±19.81	20.51±13.24	0.142
RMSSD (ms)	24.24±19.79	20.94±13.79	0.646
pRR50%	5.27±10.57	3.87±7.75	0.551
Frequency domain			
Total power	952.72±1366.04	702.82±707.52	0.9
LF	241.63±423.11	193.96±195.54	0.42
HF	302.66±600.31	191.4±219.34	0.99
LF/HF	2.24±4.53	1.53±1.43	0.06

Average RR: Average R-R interval, SDRR: Standard deviation of R-R intervals, SDSD: Standard deviation of successive differences, RMSSD: Root mean square of successive differences, pRR50 (%): Percentage of successive R-R intervals that differ by more than 50 ms, LF: Low frequency power, HF: High frequency power, LF/HF: Ratio of low frequency to high frequency power. Bold font indicates/highlights statistically significant values.

Table 2: Baseline (Pre- medication) and post medication Baroreflex sensitivity and blood pressure variability indices.

BRS and BPV parameters in study group			
Characteristic	Pre- medication <i>n</i> =49 (Mean±SD)	Post medication <i>n</i> =40 (Mean±SD)	<i>P</i> -value (Kruskal-Wallis Test)
BPV parameters			
HR Mean NN	84.86±13.82	83.26±13.32	0.005
SBP Mean NN	118.85±26.32	111.11±25.11	<0.001
MBP Mean NN	88.14±20.63	80.52±22.32	<0.001
DBP Mean NN	72.79±19.36	66.11±19.53	<0.001
RRI CV	6.63±8.15	6.01±4.83	0.031
HR CV	21.41±86.02	9.77±23.01	0.014
SBP CV	6.12±3.23	5.93±3.09	0.286
MBP CV	6±3.7	5.65±3.12	0.169
DBP CV	6.6±4.41	6.67±4.49	0.252
BRS parameters			
All SBP BRS	9.55±14.2	12.24±15.91	0.505
All MBP BRS	11.98±22.55	15.38±25.08	0.844
All DBP BRS	8.15±13.16	11.41±14.19	0.677
Alpha_LF	7.64±6.83	10.62±15.95	0.375
Alpha_HF	11.48±11.39	18.96±35.13	0.305

HR Mean NN: Heart rate mean of normal-to-normal intervals, SBP: Systolic blood pressure, MBP: Mean blood pressure, DBP: Diastolic blood pressure, Coefficient of variation (CV), RRI CV - R-R Intervals, baroreflex sensitivity (BRS), Alpha_LF: Baroreflex gain (Alpha index) in the Low-frequency range, Alpha_HF: Baroreflex gain (Alpha Index) in the High-frequency range. Bold font indicates/highlights statistically significant values.

A subset analysis was performed based on physiological improvement patterns. The participants were further classified as responders or non-responders using two parallel approaches: The change in BPV indices (Δ BPV) [Table 3] and the magnitude of change in BRS (Δ BRS) [Table 4].^[24]

Using a post-treatment SBP cut-off of 111.11 mmHg, responders and non-responders had similar baseline autonomic and cardiac measures, though responders showed numerically higher SDNN, RMSSD, and LF/HF ratios [Table 1].

Significant autonomic differences emerged when participants were reclassified by BRS-SBP (cut-off ≥ 10 ms/mmHg) [Table

Table 3: Responder and non-responder classification based on post-medication mean SBP cut off 111.11mmHg.

Characteristic (baseline)	Responders (n=23) (Mean±SD)	Non-responders (n=15) (Mean±SD)	P-value (Independent t test)
Time domain HRV			
Age (years)	47.83±10.718	43.73±5.612	0.183
Average RR (ms)	723.78±115.538	753.5±82.32	0.372
SDRR (ms)	34.57±20.774	26.2±8.567	0.127
SDSD (ms)	25.99±21.134	20.29±10.16	0.312
RMSSD (ms)	25.96±21.11	20.27±10.14	0.312
pRR50%	6.59±13.18	4.34±8.19	0.599
Frequency Domain HRV			
Total power	1363.71±1864.02	646.34±378.359	0.551
LF	339.08±579.201	182.77±179	0.386
HF	420.04±834.182	183.93±171.004	0.766
LF/HF	2.04±2.56	2.95±7.007	0.481
ECHO Parameters			
EF %	64.17±2.188	63.82±2.099	0.613
LV Mass in gms	140.01±20.336	143.84±16.292	0.527
EA Ratio	1.16±0.312	1.08±0.297	0.418
BRS Parameters			
All SBP BRS	8.84±8.518	13.32±21.658	0.743
All MBP BRS	14.62±27.534	13.11±20.473	0.795
All DBP BRS	7.33±6.593	12.76±20.16	0.233
Alpha_LF	7.76±5.692	8.7±9.297	0.699
Alpha_HF	11.13±7.007	15.08±16.725	0.812
Average RR: Average R-R interval, SDRR: Standard deviation of R-R intervals, SDSD: Standard deviation of successive differences, RMSSD: Root Mean Square of successive differences, pRR50 (%): Percentage of successive R-R intervals that differ by more than 50 ms, LF: Low frequency power, HF: High frequency power, LF/HF: Ratio of low frequency to high frequency power, EF: Ejection fraction, LV Mass: Left ventricular mass, EA Ratio : Early diastolic filling velocity/Atrial contraction filling velocity ratio, SBP: Systolic blood pressure, MBP: Mean blood pressure, DBP: Diastolic blood pressure, Baroreflex sensitivity (BRS), Alpha_LF: Baroreflex gain (Alpha Index) in the low-frequency range, Alpha_HF: Baroreflex gain (alpha index) in the high-frequency range.			

4]. Responders ($n = 11$) had lower LF/HF ratios ($P = 0.003$), higher parasympathetic indices, and significantly higher HF power ($P = 0.002$).

DISCUSSION

In 49 newly diagnosed hypertensive women, autonomic and cardiovascular indices were assessed pre- and post-treatment. Participants were middle-aged with preserved systolic function, normal LV mass, and EA ratio, indicating intact cardiac structure without abnormalities that could confound autonomic evaluations.^[4,24] Lower baseline BP during testing likely reflects controlled laboratory recording conditions compared to the outpatient department. Antihypertensive treatment reduced LF and HF, but there was no significant improvement in HRV. Longer treatment or greater hemodynamic change may be needed for measurable HRV gains.^[2] The LF/HF ratio trended toward reduction, suggesting

a shift toward vagal predominance.^[25] The trend suggests possible normalization of sympathovagal balance, especially in those with high baseline sympathetic activity (baseline mean LF/HF in nonresponders is much higher compared to overall baseline mean LF/HF and overall post mean LF/HF).

In contrast to HRV, significant reductions were observed in BPV parameters: SBP Mean NN, MBP Mean NN, and DBP Mean NN following therapy, indicating effective afterload control. The reduction in HR Mean NN reflects a modest decrease in average HR, the direction of which may accompany decreased sympathetic tone or the pharmacological profile of administered drug, as also seen with ARBs such as telmisartan and beta-blockers.^[23,26] RRI-CV and HR-CV decreased significantly, suggesting improved stability of autonomic modulation over the cardiac cycle,^[4,14] whereas SBP-CV, MBP-CV, and DBP-CV did not change significantly. The BPV indices showed selective modulation in response to therapy, reduced absolute pressure values –

Table 4: Responder and non-responder classification based on post-medication All SeqBRS-SBP cut off 10ms/mmHg.

Characteristic (Baseline)	Responders (n=11) (Mean±SD)	Non-responders (n=27) (Mean±SD)	P-value (Independent t test)
Time Domain HRV			
Age (years)	46±11.15	46.29±8.494	0.930
Average RR (ms)	777.95±130.132	720.659±87.673	0.116
SDRR (ms)	40.113±24.740	27.56±11.92	0.036
SDSD (ms)	34.11±25.17	19.57±11.6	0.016
RMSSD (ms)	34.07±25.15	19.55±11.58	0.016
pRR50%	11.73±17.71	3.324±6.671	0.126
Frequency Domain HRV			
Total Power	1858.064±2399.627	755.672±767.363	0.148
LF	469.432±815.093	198±179.346	0.455
HF	730.857±1116.705	163.732±214.344	0.002
LF/HF	0.623±0.355	3.11±5.629	0.003
ECHO Parameters			
EF %	63.545±2.207	64.207±2.111	0.387
LV Mass in gms	132.818±14.77	144.986±19.017	0.064
EA Ratio	1.1±0.2	1.131±0.338	0.777
BPV Parameters			
SBP Mean NN	114.06±21.649	119.206±25.639	0.559
MBP Mean NN	87.915±20.943	88.576±19.094	0.925
DBP Mean NN	74.845±21.342	73.261±16.867	0.807
SBP CV	7.525±4.872	5.807±2.8	0.170
MBP CV	7.805±5.537	5.443±3.092	0.094
DBP CV	8.720±6.623	5.982±3.575	0.099
Average RR: Average R-R Interval, SDRR: Standard deviation of R-R intervals, SDSD: Standard deviation of successive differences, RMSSD: Root mean square of successive differences, pRR50 (%): Percentage of successive R-R intervals that differ by more than 50 ms, LF: Low frequency power, HF: High frequency power, LF/HF: Ratio of low frequency to high frequency power, EF: Ejection fraction, LV Mass: left ventricular mass, EA ratio: Early diastolic filling velocity/atrial contraction filling velocity ratio, SBP: Systolic blood pressure, MBP: Mean blood pressure, DBP: Diastolic blood pressure, CV: Coefficient of variation			

SBP Mean NN, MBP Mean NN, and DBP Mean NN; but relatively preserved proportional variability- SBP-CV, MBP-CV, and DBP-CV,^[27] indicating that antihypertensive therapy predominantly influenced the average load rather than the proportional dispersion of BP. The calcium channel blockers also tend to reduce vascular variability without consistent baroreflex improvement.^[27,28] This pattern suggests an intact autonomic buffering despite an overall reduction in vascular tone.^[28]

BRS indices demonstrated an upward trend, although not statistically significant. All SBP BRS, MBP BRS, and DBP BRS increased following antihypertensive therapy, reflecting improved reflex buffering. Alpha_LF and Alpha_HF also trended upward. This upward shift in BRS aligns with multiple physiological expectations like reduced BP load, which reduces baroreceptor “strain fatigue,”^[29] improved vascular compliance enhances afferent baroreceptor input,^[30] and improvement in sympathovagal balance may indicate a

more stable autonomic feedback mechanism.^[31] While short-term single drug intervention could not produce significant BRS improvement, the directionality suggests appropriate autonomic adaptation.^[32]

Analysis between Δ SBP- BRS and Δ SBP-CV showed a significant positive correlation, suggesting that better regulation of short-term BPV is correlated with improved BRS.^[8] The lack of correlation between Δ LF/HF and Δ SBP-BRS indicates that they may represent distinct neural control mechanisms. Age positively correlates with Δ SBP-BRS, likely due to lower baseline BRS in older adults, making changes appear larger in response to BP challenges.^[18] Baseline EF did not correlate with Δ SBP-BRS, indicating that short-term baroreflex improvement may be independent of systolic contractile performance in subjects with preserved EF. Similarly, baseline LV mass showed no significant association, suggesting that normal structural cardiac parameters did not dictate autonomic adaptation in this cohort.^[33]

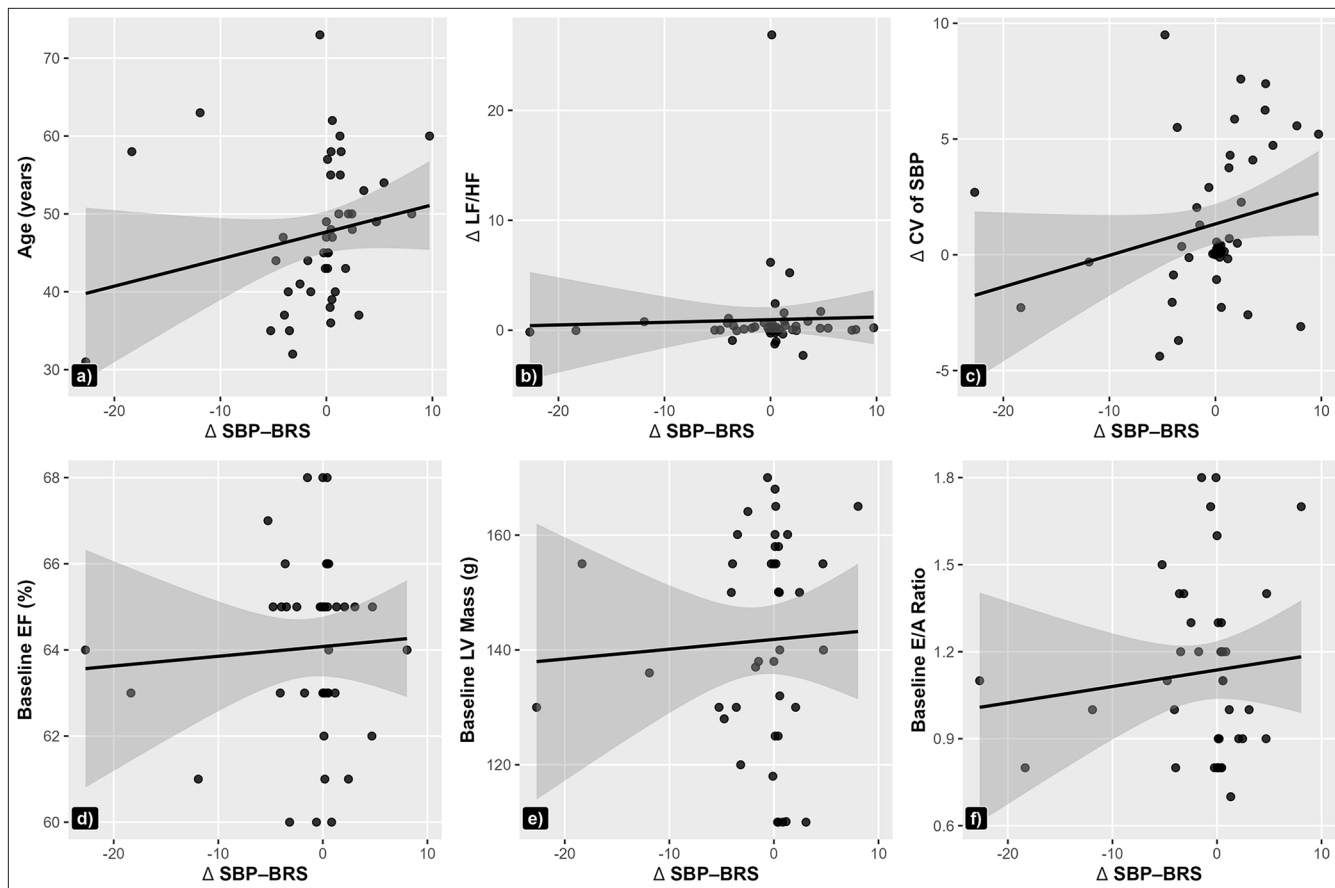


Figure 2: Correlation Analysis (a) Age vs Δ SBP BRS; (b) Δ LF/HF vs Δ SBP BRS; (c) Δ CV SBP vs Δ SBP BRS; (d) Baseline EF % vs Δ SBP BRS; (e) Baseline LV Mass vs Δ SBP BRS). (SBP: Systolic blood pressure, BRS: Baroreflex sensitivity, CV: Coefficient of variation, LV: Left ventricular, HF: High frequency, EF: Ejection fraction).

BRS-based classification ($\text{BRS-SBP} \geq 10$ ms/mmHg) showed that responders had significantly higher parasympathetic tone, greater HRV, and better baroreflex adaptability (post All Seq BRS-SBP was >10 ms/mmHg), indicating improved autonomic balance and stronger vagal modulation compared to non-responders.^[34] Thus, HRV and BRS assessments, alongside cardiac imaging, may improve the prediction of antihypertensive response and autonomic dysfunction in hypertensive patients, despite no BPV or echocardiographic differences between responders and non-responders.^[33] These results show that following antihypertensive therapy, despite effective BP reduction, autonomic responses vary and baroreflex responsiveness depends on baseline autonomic reserve and cardiac structure.^[35] Study limitations include small sample size, attrition bias, and intrinsic heterogeneity, but physiological validity was supported by subgroup differences and significant $\Delta\text{BRS}-\Delta\text{BPV}$ correlations.

CONCLUSION

Short-term telmisartan therapy effectively reduced BP but did not produce statistically significant improvements in

HRV or BRS. Trends toward improved BRS and significant $\Delta\text{BRS}-\Delta\text{BPV}$ correlation suggest early baroreflex-related physiological modulation with inter-individual variability. These findings indicate that autonomic indices may complement clinical BP measurements in evaluating an individual's physiological response to antihypertensive therapy, warranting confirmation in larger, controlled studies.

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Ethical approval: The research/study was approved by the Institutional Review Board at AIIMS Bibinagar, number AIIMS/BBN/IEC/Apr/2025/667-R, dated May 06, 2025.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given consent for their clinical information to

be reported in the journal. The patient understands that the patient's names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that they have used artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript or image creations.

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