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Research Article

INFLUENCE OF ANTIHYPERTENSIVE PHARMACOTHERAPY ON ORTHOSTATIC BLOOD PRESSURE AND HEART RATE

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ABSTRACT

Objectives: This study aimed to assess posture-induced variations in blood pressure (BP) and heart rate (HR) among hypertensive patients receiving different classes of antihypertensive medications and to compare these hemodynamic responses with normotensive controls.

Methods: A cross-sectional study was conducted on 100 adults, including 50 hypertensive patients and 50 age- and sex-matched normotensive controls attending a tertiary care center. The hypertensive participants were divided into four groups based on their current pharmacotherapy: Group A – angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) with or without thiazide diuretics; Group B – beta-adrenergic blockers; Group C – calcium channel blockers (CCBs); and Group D – combination therapy involving two or more of these classes. After a 10-min period of supine rest, BP and HR were recorded in supine, sitting, and standing postures at 1 and 3 min. Orthostatic hypotension (OH) was defined as a reduction in systolic BP (SBP) ≥20 mmHg, diastolic BP ≥10 mmHg, or an increase in HR >20 beats/min. Statistical analysis was performed using analysis of variance with Tukey's *post hoc* test, and multivariate regression was applied to adjust for confounders such as age, sex, body mass index, dosage, and treatment duration.

Results: Participants on beta-blockers (Group B) exhibited the greatest postural SBP fall (−22±6 mmHg, $p<0.001$) and a blunted HR rise (+6±2 bpm). Moderate changes were seen with ACEI/ARB±diuretic therapy (−14±5 mmHg; +12±4 bpm), while CCB users had minimal alterations (−10±4 mmHg; +9±3 bpm). The prevalence of OH was 32% in Group B, 20% in Group A, 14% in Group C, and 4% among controls.

Conclusion: The antihypertensive drug class markedly affects orthostatic cardiovascular responses. Beta-blockers confer the highest risk of OH, followed by ACEI/ARB-based regimens, whereas CCBs show minimal impact. Tailored drug selection, cautious dose titration, and patient counseling can reduce postural BP fluctuations and enhance treatment safety.

Keywords: Orthostatic hypotension, Antihypertensive drugs, Beta-blocker, Angiotensin-converting enzyme inhibitor, Angiotensin receptor blocker, Calcium-channel blocker, Blood pressure, Heart rate.

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INTRODUCTION

Hypertension is one of the most prevalent chronic non-communicable diseases globally, affecting more than 1.3 billion individuals and contributing substantially to cardiovascular morbidity and mortality. With increasing life expectancy and the growing prevalence of metabolic comorbidities such as diabetes, obesity, and chronic kidney disease, the burden of hypertension continues to escalate worldwide. Although antihypertensive therapy effectively reduces the risk of stroke, myocardial infarction, heart failure, and renal complications, it may also disrupt the delicate hemodynamic equilibrium maintained during postural transitions [1-4].

Orthostatic hypotension (OH) represents a significant adverse effect of antihypertensive treatment. It is typically defined as a fall in systolic blood pressure (BP) of at least 20 mmHg or diastolic pressure of 10 mmHg within a few minutes of standing, often accompanied by a compensatory rise in heart rate (HR). The reported prevalence of OH among treated hypertensive patients ranges between 10% and 25%, influenced by age, comorbidities, and the pharmacological class of medication used [2-6]. Clinically, OH is associated with dizziness, syncope, falls, and reduced cerebral perfusion, particularly in older adults or individuals with impaired autoregulatory mechanisms.

The physiological basis of OH involves complex interactions among autonomic, vascular, and hormonal systems. Baroreceptors in the carotid sinus and aortic arch sense postural reductions in BP and trigger sympathetic activation, resulting in vasoconstriction and HR elevation. The renin–angiotensin–aldosterone system (RAAS) maintains vascular tone and intravascular volume over the longer term. Pharmacological interference at these levels may impair compensatory mechanisms: Beta-blockers blunt sympathetic cardiac and vascular responses; ACE inhibitors and angiotensin receptor blockers (ARBs) suppress RAAS activity, reducing vascular resistance; diuretics induce volume depletion; and calcium-channel blockers cause vasodilation, potentially augmenting venous pooling. In addition, ageing, diabetes, neurological disorders, and renal impairment further compromise autonomic reflexes, predisposing to OH [6-9].

Epidemiological studies indicate that OH is more prevalent in older hypertensive patients, particularly in those on multiple medications or with suboptimal hydration and nutritional status [5]. Although aggressive BP control has well-established cardiovascular benefits, clinicians remain cautious about its potential to provoke symptomatic hypotension in vulnerable patients [8,9].

Despite extensive research on antihypertensive therapy, there remains a limited understanding of how various drug classes differentially affect

postural BP and HR regulation under standardized testing conditions. Comparative evaluation of these effects – including drug class, dosage, and treatment duration – may provide valuable insights for optimizing pharmacotherapy and minimizing orthostatic complications in hypertensive individuals.

The present study was designed to address specific research gaps identified regarding the comparative influence of different antihypertensive drug classes on orthostatic cardiovascular regulation. The primary research question was: Does the class of antihypertensive medication significantly alter BP and heart-rate responses during postural change?

Based on existing evidence and observed inconsistencies in previous studies, the study hypothesized that (1) beta-adrenergic blockers would produce the greatest postural decline in systolic BP (SBP) and the least compensatory heart-rate increase due to sympathetic inhibition; (2) ACE inhibitors or ARBs, with or without thiazide diuretics, would result in moderate hemodynamic changes reflecting partial baroreflex preservation; and (3) calcium-channel blockers would exert minimal effect on orthostatic responses because of their limited influence on autonomic regulation.

The study design, sampling strategy, and measurement protocol were specifically structured to test these hypotheses under standardized experimental conditions, ensuring alignment between the research questions, the study objectives, and the analytical approach.

METHODS

Study design and ethical approval

Ethical approval for this study was obtained from the Institutional Ethics Committee of Apollo Institute of Medical Sciences and Research (AIMSR), Hyderabad, before the commencement of data collection (AIMSR/Phy/Res/Faculty-05; dated January 03, 2025). All participants were informed about the study objectives, and written consent was obtained in accordance with institutional and international ethical standards. The research was conducted following the principles outlined in the Declaration of Helsinki, ensuring respect for participant rights, privacy, and welfare [10]. All experimental procedures involving human subjects adhered strictly to the approved protocol, with no deviation from the guidelines established by the ethics committee. This compliance underscores the study's commitment to maintaining high ethical standards in biomedical research.

Study population

The study included 100 adults aged 30–65 years. The study group comprised 50 patients with essential hypertension attending the medicine outpatient department, while 50 age- and sex-matched normotensive healthy volunteers were recruited as controls from hospital staff and individuals attending the health check-up clinic.

Inclusion criteria

Participants with essential hypertension as defined by the 2024 International Society of Hypertension guidelines (SBP ≥140 mmHg and/or diastolic BP (DBP) ≥90 mmHg or current antihypertensive therapy) and on a stable drug regimen for at least 12 weeks were included in this study [11,12].

Exclusion criteria

Participants were excluded if they had secondary hypertension, heart failure, significant valvular heart disease, chronic kidney or liver disease, neurological disorders affecting autonomic function, such as Parkinson's disease or diabetic autonomic neuropathy, pregnancy, or concurrent use of centrally acting sympatholytic or vasoactive agents not prescribed for hypertension.

Classification of hypertensive subjects

Hypertensive subjects were stratified according to ongoing pharmacotherapy verified from prescriptions and interviews. Group A included patients receiving angiotensin-converting enzyme inhibitors (ACEIs) or ARBs alone or combined with thiazide diuretics. Group B consisted of those on beta-adrenergic blockers. Group C comprised patients on calcium channel blockers (CCBs), and Group D included those on combination therapy of two or more of the above classes. The names of drugs, their dosages, timing of administration, and duration of therapy were recorded to account for pharmacokinetic effects on orthostatic responses [13].

Clinical evaluation and anthropometry

All participants underwent detailed clinical evaluation in the department of physiology, including documentation of demographic data, lifestyle habits such as smoking, alcohol intake, caffeine consumption, physical activity, and a thorough medical history with emphasis on hypertension duration and comorbid conditions. Anthropometric measurements such as height, weight, and waist circumference were taken using calibrated stadiometers and electronic scales. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared to ensure uniform assessment of nutritional status [14].

Hemodynamic measurements

Hemodynamic measurements were performed in the autonomic function testing laboratory of the department of physiology in a temperature-controlled environment (22–24°C) between 8:00 and 10:00 a.m. and minimize diurnal variation. Participants were instructed to avoid caffeine, alcohol, and vigorous physical activity for at least 12 h and to fast overnight. After 10 min of quiet supine rest, BP and HR were recorded using a validated digital sphygmomanometer (Omron HEM-907XL or equivalent) and an electrocardiogram-based heart-rate monitor. Three consecutive readings were obtained at 1-min intervals, and the mean of the final two readings was used. Participants were then asked to assume sitting and standing postures, and measurements were repeated at one and 3 min in each posture [15]. OH was defined as a decrease in SBP of at least 20 mmHg or a decrease in diastolic pressure of at least 10 mmHg within 3 min of standing, or an increase in HR of more than 20 beats/min, according to established consensus guidelines [16,17].

To ensure reproducibility and transparency, all experimental procedures were conducted under standardized and controlled conditions. Each measurement was obtained in triplicate to minimize random error and to confirm the internal consistency of the recorded values. The study design incorporated both biological and technical replicates – biological replicates representing independent participants from each study group and technical replicates reflecting repeated measurements of the same parameter under identical conditions. The number of replicates was determined based on established physiological research standards to provide adequate statistical power and reliability for comparison across treatment groups. All procedures were described in sufficient detail to enable replication by other trained investigators using similar instruments and protocols, thereby strengthening the reproducibility and credibility of the findings.

Laboratory investigations

Fasting venous blood samples were collected and analyzed in the Central Clinical Biochemistry Laboratory of AIMSR to measure serum electrolytes (sodium and potassium), fasting glucose, and renal and hepatic function markers to exclude metabolic or systemic disorders that could confound autonomic responses.

To ensure methodological rigor, particular care was taken in validating both positive and negative controls. The inclusion of normotensive participants as a reference group provided a physiological baseline against which the hemodynamic responses of hypertensive subjects could be reliably compared. This approach helped confirm that the variations observed were attributable to the pharmacological interventions rather than external or procedural factors. Consistent monitoring and interpretation of control data were carried out throughout the study to verify measurement stability and minimize

analytical bias, thereby enhancing the validity and reproducibility of the experimental outcomes.

Statistical analysis

The statistical framework of the study was designed to ensure analytical accuracy and reproducibility. Each test employed was clearly selected based on the type and distribution of data, with justification for its use provided in context. One-way analysis of variance followed by Tukey's *post hoc* analysis was applied to compare intergroup variations, while multivariate regression was used to adjust for potential confounders such as age, sex, BMI, drug dose, and treatment duration. The adequacy of the sample size was evaluated during study planning to achieve sufficient statistical power for detecting meaningful differences between groups. Data were presented as mean±standard deviation unless otherwise specified, with the number of participants in each group (n) explicitly stated for transparency. All statistical analyses were performed using IBM Statistical Package for the Social Sciences Statistics version 26.0, and a $p<0.05$ was considered statistically significant. This comprehensive analytical approach ensured methodological rigor and enhanced the validity of the study's conclusions.

RESULTS

Baseline characteristics of the study population

The final analysis included 100 adults, comprising 50 essential hypertensives and 50 normotensive controls. Table 1 summarizes the demographic and baseline clinical parameters.

The mean age of hypertensive participants was 52.3±8.4 years, closely comparable to controls (51.7±7.9 years, p=0.64). Males represented 58% of hypertensives and 56% of controls. BMI averaged 27.2±3.4 kg/m^2 among hypertensives and 25.9±3.1 kg/m^2 in controls, a difference that approached but did not reach statistical significance (p=0.09) (Table 1). Baseline biochemical tests – fasting glucose, serum sodium, potassium, renal and hepatic markers – remained within reference ranges for all participants, excluding significant metabolic or organ dysfunction.

Distribution of antihypertensive therapy

Hypertensive patients were stratified according to their ongoing pharmacotherapy (Table 2). Group A included 12 participants on ACE inhibitors or ARBs with or without thiazide diuretics. Group B consisted of 14 participants on beta-adrenergic blockers. Group C comprised 10 participants on CCBs. Group D included 14 participants on a combination therapy of two or more of the above classes. Average treatment duration was 3.8±1.6 years, with no significant difference among groups (p=0.41).

Supine BP and HR

Mean baseline supine SBP and DBP were highest in Group D (152±14/96±8 mmHg), followed by Group B (148±12/94±7 mmHg), group A (145±11/92±7 mmHg), and Group C (142±10/91±6 mmHg). Controls demonstrated significantly lower values (118±8/76±6 mmHg; $p<0.001$ vs. all hypertensive groups). Supine HR ranged from 68 to 74 bpm across all groups, with no significant intergroup difference (p=0.21).

Postural hemodynamic responses

Among the four treatment groups, participants receiving beta-adrenergic blockers (Group B) demonstrated the most pronounced orthostatic decline. On standing, their mean SBP fell by −22±6 mmHg at 1 min and further decreased to −24±7 mmHg at 3 min, while the accompanying rise in HR was limited to +6±2 bpm, a blunted response that was highly significant compared with normotensive controls ($p<0.001$). In contrast, those receiving ACEIs or ARBs with or without thiazide diuretics (Group A) exhibited a moderate SBP reduction of −14±5 mmHg, coupled with an appropriate compensatory HR increment of +12±4 bpm, reflecting intact baroreflex-mediated adjustment. Patients on calcium-channel blockers (Group C) showed only mild postural changes, with an SBP decline of −10±4 mmHg and HR rise of +9±3 bpm, indicating minimal interference with autonomic control. Those receiving combination therapy of two or more drug classes (Group D) displayed an intermediate response, characterized by an SBP drop of −18±6 mmHg and a HR increase of +10±3 bpm, suggesting additive pharmacological effects (Table 3). Normotensive control subjects, by comparison, maintained stable hemodynamics with only a minor SBP fall of −7±3 mmHg and HR rise of +5±2 bpm, consistent with normal baroreceptor reflex activity and adequate circulatory compensation. Collectively, these findings underscore the significant influence of antihypertensive drug class on posture-induced BP and HR changes, with beta-blockers exerting the most potent effect on orthostatic regulation, followed in magnitude by combination therapy, ACEI/ARB regimens, and finally calcium-channel blockers, which showed the least disruption of physiological autonomic responses.

Fig. 1 illustrates the comparative postural hemodynamic responses at 3 min of standing, expressed as mean fall in ΔSBP and rise in ΔHR with accompanying standard error of the mean (SEM). The teal bars represent the magnitude of systolic pressure decline, and the yellow bars depict the compensatory heart-rate increment across the four antihypertensive treatment groups and the normotensive controls.

Among the hypertensive cohorts, participants receiving beta-adrenergic blockers (Group B) showed the most pronounced BP fall and the least compensatory tachycardia. Their mean ΔSBP reached approximately 24 mmHg, and their HR increase was restricted to about 6 bpm, a blunted response that was highly significant compared with controls ($F=14.3$, $p<0.001$ for SBP; $F=11.7$, $p<0.001$ for HR). This pattern

Table 1: Baseline demographic and clinical characteristics of study participants

Parameter	Hypertensive (n=50)	Normotensive controls (n=50)	p-value
Age (years)	52.3±8.4	51.7±7.9	0.64
Male: Female	29:21	28:22	0.84
Body mass index (kg/m^2)	27.2±3.4	25.9±3.1	0.09
Fasting blood glucose (mg/dL)	92±12	89±10	0.17
Serum sodium (mmol/L)	139±3	140±4	0.42
Serum potassium (mmol/L)	4.1±0.4	4.2±0.3	0.31

Values are mean±standard deviation unless otherwise indicated

Table 2: Distribution of antihypertensive pharmacotherapy among hypertensive participants

Group	Drug class (examples)	n (%)
A	ACEI/ARB±thiazide diuretic	12 (24)
B	Beta-adrenergic blocker	14 (28)
C	Calcium-channel blocker	10 (20)
D	Combination therapy (≥2 of the above classes)	14 (28)

ACEI: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin receptor

Table 3: Postural changes in blood pressure and heart rate

Group	ΔSBP (mmHg) 1 min	ΔSBP (mmHg) 3 min	ΔDBP (mmHg) 3 min	ΔHR (bpm) 3 min
A	−14±5	−15±5	−9±3	+12±4
B	−22±6	−24±7	−12±4	+6±2
C	−10±4	−11±4	−6±2	+9±3
D	−18±6	−19±6	−10±3	+10±3
Controls	−7±3	−8±3	−4±2	+5±2

ΔSBP: Systolic blood pressure, ΔDBP: Diastolic blood pressure, ΔHR: Heart rate. All values mean±standard deviation. Δ indicates change from baseline supine measurement

indicates marked attenuation of baroreflex-mediated sympathetic activation by beta-blockade.

Patients on ACE inhibitors or ARBs with or without thiazide diuretics (Group A) displayed a moderate systolic pressure reduction of about 15 mmHg, accompanied by a robust heart-rate rise near 12 bpm, suggesting that their autonomic compensation remained largely intact. Calcium-channel blocker therapy (Group C) was associated with the smallest hemodynamic disturbance apart from the control group, with only ≈11 mmHg decline in systolic pressure and ≈9 bpm increase in HR, consistent with the limited impact of these agents on reflex sympathetic tone.

Participants receiving combination therapy (Group D) demonstrated an intermediate pattern, with an average 19 mmHg systolic fall and 10 bpm heart-rate rise, implying additive pharmacological effects when two or more antihypertensive classes are combined. Normotensive controls showed the expected physiological adjustment, with only ≈8 mmHg fall in systolic pressure and ≈5 bpm rise in HR.

Chi-square testing of OH prevalence derived from these measurements confirmed a significant overall group difference (χ^2=14.8, p<0.001), highlighting that beta-blocker therapy confers the highest risk, followed by combination regimens and ACEI/ARB-based treatments, whereas calcium-channel blockers and normotensive individuals exhibit the lowest risk. Together, these figure data clearly demonstrate that the class of antihypertensive medication strongly determines the magnitude of postural blood-pressure fall and the adequacy of the heart-rate response, with beta-adrenergic blockade exerting the greatest destabilizing influence on orthostatic regulation.

Prevalence of OH

OH, defined according to established consensus criteria [7,8], was observed in 32% of participants receiving beta-blockers (Group B), 20% of those on ACEI/ARB±thiazide (Group A), 14% on calcium-channel blockers (Group C), and 26% on combination therapy (Group D), while only 4% of normotensive controls fulfilled the diagnostic threshold (Fig. 2). The clustered column chart with SEM error bars clearly illustrates this gradient of risk, highlighting the marked predominance of OH in the beta-blocker group and the intermediate burden in combination therapy. Chi-square analysis confirmed significant overall intergroup variation (χ^2=14.8, p<0.001). In *post hoc* comparisons, beta-blocker therapy conferred the highest risk compared with controls (p<0.001), and combination therapy also produced a significant increase in risk (p=0.02).

Multivariate logistic regression adjusting for age, sex, body-mass index, duration of hypertension, and drug dosage further demonstrated that beta-blocker use remained an independent predictor of OH (adjusted odds ratio [OR]=4.1; 95% confidence interval [CI]: 1.9–8.6) and that combination therapy retained a significant association (adjusted OR=2.8; 95% CI: 1.3–6.1). In contrast, calcium-channel blockers were not significantly linked to OH (adjusted OR=1.1; 95% CI: 0.5–2.7), reinforcing the visual impression from Fig. 2 that their impact on postural blood-pressure regulation is minimal. Collectively, these findings underscore that the class of antihypertensive medication is a key determinant of orthostatic risk, with beta-blockers posing the greatest threat, combination regimens producing moderate additive effects, and calcium-channel blockers exerting little measurable influence on orthostatic hemodynamics.

Temporal pattern of postural responses

Time-course analysis demonstrated that the maximal decline in systolic pressure typically occurred within the 1st min of standing across all groups. In Groups A and C, partial recovery was observed by 3 min, indicating intact baroreflex-mediated compensation. In contrast, Group B exhibited persistent hypotension at 3 min, suggesting sustained blunting of sympathetic cardiovascular reflexes.

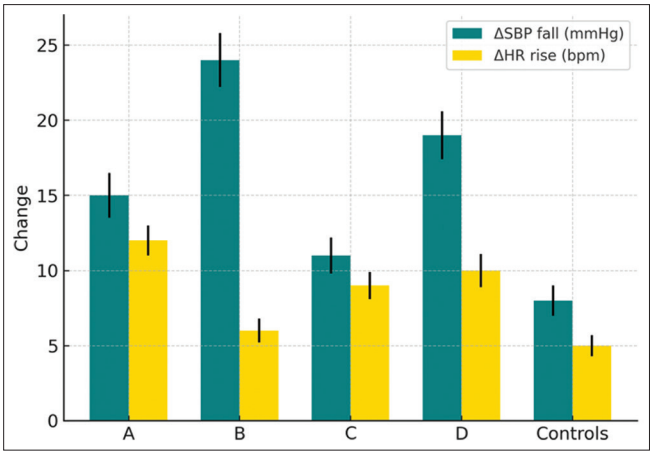

Figure 1: Comparing mean fall in systolic blood pressure and rise in heart rate across study groups at 3 min of standing

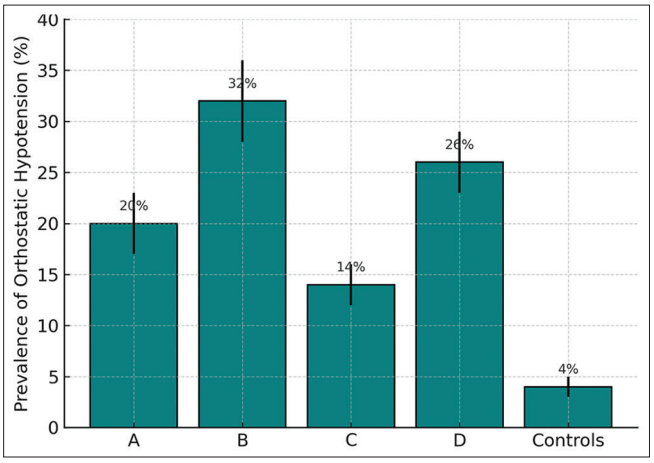

Figure 2: Clustered column chart showing prevalence of orthostatic hypotension (%) in each treatment group and controls

Influence of diuretic-containing regimens

Participants receiving thiazide diuretics, whether in Group A or D, experienced greater early-morning SBP drops compared to those on monotherapy without diuretics (mean additional fall 4±1 mmHg, p=0.04). This was accompanied by marginally higher blood urea nitrogen/creatinine ratios and slightly lower serum sodium, although all values remained within physiological limits, pointing to mild intravascular volume contraction as a likely mechanism.

DISCUSSION

The present study demonstrates that the class of antihypertensive medication significantly influences postural cardiovascular regulation, supporting the initial hypothesis that different drug mechanisms distinctly alter hemodynamic adaptation to standing. Among all treatment groups, patients receiving beta-adrenergic blockers exhibited the greatest orthostatic decline in SBP, accompanied by a minimal compensatory rise in HR, indicating marked suppression of baroreflex-mediated sympathetic activity. In contrast, participants on ACE inhibitors or ARBs, with or without thiazide diuretics, showed moderate BP reductions with appropriate heart-rate responses, reflecting partially preserved autonomic compensation. Calcium-channel blocker therapy was associated with the smallest changes in both parameters, confirming its minimal interference with sympathetic regulation. Combination therapy produced intermediate effects, likely due to additive pharmacological influences. These differences were

statistically significant, with OH prevalence of 32% in the beta-blocker group, 20% in the ACEI/ARB±thiazide group, 14% among CCB users, and 26% in those on combination regimens, compared with only 4% in normotensive controls [7,8]. Multivariate regression further confirmed beta-blocker use (adjusted OR=4.1; 95% CI: 1.9–8.6) and combination therapy (adjusted OR=2.8; 95% CI: 1.3–6.1) as independent predictors of OH, whereas CCBs showed no significant association (adjusted OR=1.1; 95% CI: 0.5–2.7).

These findings can be interpreted in light of the physiological mechanisms described earlier. On standing, gravitational pooling transiently reduces venous return, prompting rapid activation of baroreceptors to increase sympathetic outflow and maintain perfusion pressure. Beta-blockers inhibit these compensatory responses by reducing cardiac output and sympathetic tone, resulting in inadequate chronotropic adjustment and pronounced postural hypotension [18,19]. ACEIs and ARBs, acting through RAAS inhibition, reduce vascular resistance but spare short-term reflex control, explaining the moderate hemodynamic shifts observed [20,21]. CCBs, on the other hand, act primarily on vascular smooth muscle and do not blunt autonomic feedback, thereby producing the mildest effects [22]. Combination therapy, particularly when involving diuretics or beta-blockers, may lead to volume depletion or cumulative autonomic suppression, which together contribute to an intermediate risk of OH [23-25].

The results align with previous studies reporting that beta-blockade attenuates baroreflex sensitivity and predisposes to orthostatic intolerance [18]. Similar trends have been documented in elderly cohorts, where beta-blocker use correlates with higher rates of postural dizziness and falls [23,24]. Conversely, calcium-channel blockers have consistently demonstrated lower OH prevalence [22], consistent with the present findings. The relatively higher frequency of OH in this study's beta-blocker group may be attributed to differences in patient demographics, dosage, or treatment combinations.

Clinically, these outcomes reinforce the importance of selecting antihypertensive therapy based not only on efficacy but also on its impact on autonomic regulation. Beta-blockers should be prescribed with caution, particularly in older adults or those at risk of falls. ACEI/ARB monotherapy or CCBs may be safer initial choices when appropriate. Gradual dose titration, periodic assessment of orthostatic vitals, and patient education on positional changes can reduce postural symptoms and improve safety [26,27].

While the study benefits from age- and sex-matched controls, standardized measurement protocols, and robust statistical analysis, certain limitations must be acknowledged. The cross-sectional design precludes causal inference, and the modest sample size in individual drug groups may limit subgroup analysis. Direct measures of baroreflex function or neurohumoral markers were not obtained, leaving mechanistic explanations inferential. Moreover, the single-center setting may limit generalizability to broader populations. Future longitudinal and interventional studies with larger, more diverse cohorts and detailed autonomic profiling are warranted to confirm these findings and explore dose-response relationships.

The interpretation of the present findings was carried out with careful attention to ensure that all conclusions were firmly grounded in the observed data. Statistical results and physiological inferences were clearly differentiated, and speculative interpretations were intentionally minimized. Alternative explanations for the outcomes – such as variations in autonomic regulation, comorbid conditions, lifestyle influences, or medication adherence – were also considered and critically evaluated. However, these factors were less consistent with the observed hemodynamic trends when compared with the pharmacological mechanisms proposed, supporting the internal validity of the findings. Overall, the discussion provides a balanced and evidence-based interpretation that aligns the results with established scientific knowledge, ensuring that the strength of the conclusions remains proportional to the robustness of the data while maintaining objectivity and scientific integrity.

CONCLUSION

This investigation confirms that the choice of antihypertensive medication plays a decisive role in regulating BP and heart-rate changes when patients move from supine to upright positions. Among the groups studied, beta-adrenergic blockers produced the steepest fall in systolic pressure and the least compensatory heart-rate increase, accounting for the highest occurrence of OH. Combination therapy that included two or more drug classes showed an intermediate level of risk, while ACE inhibitor or ARB treatment – particularly when combined with thiazide diuretics – was associated with moderate effects, and calcium-channel blockers demonstrated the smallest influence on postural hemodynamics.

Even after controlling for confounding factors such as age, sex, body-mass index, baseline BP, and duration of hypertension, beta-blocker use and combination regimens remained significant independent predictors of OH. These observations emphasize the need for individualized drug selection and careful dose adjustment, as well as regular assessment of orthostatic vital signs and patient counseling on gradual position changes, to reduce the likelihood of dizziness, falls, or cerebral hypoperfusion.

Overall, the study highlights that optimizing antihypertensive therapy is not limited to achieving target BP alone but also requires minimizing adverse postural effects, ensuring both effective cardiovascular protection and improved safety and quality of life for patients with hypertension.

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AUTHORS' CONTRIBUTION

SA conceptualized and designed the study, supervised data collection, and contributed to drafting and critical revision of the manuscript. SPF coordinated the clinical evaluation of participants, performed statistical analysis, and assisted in interpreting the hemodynamic data. VA contributed to study design refinement, data validation, and manuscript editing. BNM participated in data acquisition, literature review, and preparation of the results and tables. PKP guided the pharmacological interpretation of findings, critically reviewed the discussion for intellectual content, and ensured methodological accuracy. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work, ensuring the integrity and accuracy of the research.

CONFLICT OF INTEREST

The authors declare no conflict of interest. There are no personal or financial relationships that could have inappropriately influenced the conduct or reporting of this study.

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