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Original Research Article

Drug Interactions of Antihypertensives with Cardiac Anatomy, Hemodynamics, and Electrophysiological Properties: A Comprehensive Review

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Abstract:

Aim: This review aims to systematically evaluate clinically relevant drug interactions of major antihypertensive drug classes—angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), beta-blockers, calcium channel blockers (CCBs), and diuretics—with other cardiovascular and non-cardiovascular drugs, focusing on their combined effects on cardiac anatomy, hemodynamic parameters, and cardiac electrophysiological properties.

Materials and Methods: A structured literature review was performed using PubMed, NCBI Bookshelf (StatPearls), and major cardiology/pharmacology textbooks to identify original studies, reviews, and practice guidelines on antihypertensive drug interactions. Inclusion criteria were: English-language publications (2000–2026), studies reporting hemodynamic, echocardiographic, or electrocardiographic outcomes, and drug-interaction studies involving ≥1 antihypertensive agent. Data were extracted on drug class, interacting agents, mechanism (pharmacokinetic vs pharmacodynamic), site of interaction (vascular bed, myocardium, conduction system), and clinical outcomes (BP, heart rate, QT interval, AV-node conduction, myocardial mass, chamber size). For illustrative purposes, a hypothetical cohort dataset was constructed to simulate real-world polypharmacy scenarios in hypertensive patients, allowing description of statistical testing methods.

Results: Pharmacokinetic interactions most commonly involve CYP3A4-dependent metabolism, particularly with verapamil and diltiazem, which inhibit CYP3A4 and increase plasma levels of co-administered substrates (e.g., statins, antiarrhythmics, immunosuppressants). Pharmacodynamic interactions include additive hypotension with vasodilator combinations (e.g., dihydropyridine CCB + alpha-1 blocker), enhanced AV-nodal blockade with beta-blocker + verapamil/diltiazem, and QT-prolongation when some antihypertensives (e.g., certain CCBs) are combined with QT-prolonging antiarrhythmics or psychotropics. Nonsteroidal anti-inflammatory drugs (NSAIDs) antagonize the antihypertensive effect of ACEIs, ARBs, and diuretics by inducing sodium retention and vasoconstriction, while also increasing the risk of renal dysfunction when combined with renin–angiotensin–aldosterone system (RAAS) inhibitors. Hemodynamically, beta-blockers and RAAS inhibitors favorably reduce myocardial wall stress and left ventricular hypertrophy, whereas unbalanced vasodilators may transiently increase shear-stress patterns in atherosclerotic segments.

Conclusion: Antihypertensive drug interactions profoundly influence cardiac structure, hemodynamics, and electrophysiology. Awareness of key pharmacokinetic (CYP3A4-mediated) and pharmacodynamic (additive vasodilation, AV-nodal blockade, volume-related) interactions is essential for prescribing safely in hypertensive patients with polypharmacy. Tailoring combinations to underlying cardiac anatomy (e.g., LVH, chamber dilatation) and electrophysiological substrate (e.g., sinus node disease, QT-interval drugs) reduces the risk of hypotension, bradyarrhythmias, and sudden cardiac events.

Keywords: Antihypertensive Drugs; Drug Interactions; Cardiac Anatomy; Hemodynamics; Electrophysiology.

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Introduction

Arterial hypertension remains a leading modifiable risk factor for stroke, myocardial infarction, heart failure, and chronic kidney disease worldwide. First-line therapies include ACE inhibitors, ARBs, beta-blockers, calcium channel blockers, and thiazide-like diuretics, often used in fixed-dose combinations to achieve blood pressure targets. As the prevalence of comorbidities (diabetes, coronary

artery disease, chronic kidney disease, atrial fibrillation) rises, patients frequently receive multiple interacting drugs, increasing the risk of significant pharmacokinetic and pharmacodynamic interactions that alter cardiac function beyond simple blood-pressure reduction.

Cardiac anatomy and hemodynamics are closely linked: long-standing hypertension induces left ventricular hypertrophy, concentric remodeling, and diastolic dysfunction, which antihypertensives can partially reverse. At the same time, changes in preload, afterload, and myocardial contractility affect intracardiac pressures, stroke volume, and coronary perfusion. Electrophysiological properties—depolarization, repolarization, and conduction velocity—are modulated by ion-channel-active antihypertensives (especially beta-blockers and non-dihydropyridine CCBs), which can increase PR interval, slow sinus rate, or prolong QT interval when combined with other ion-channel-modulating drugs.

Clinically important interactions occur at multiple sites: hepatic metabolism (e.g., CYP3A4 inhibition by verapamil/diltiazem), renal excretion (competition with diuretics, ACEIs, and other renally cleared agents), and vascular/cardiac tissues where additive vasodilation, sympathetic blockade, or ion-channel blockade cumulate. For instance, combining a beta-blocker with verapamil can yield profound AV-nodal blockade and bradycardia, whereas pairing dihydropyridine CCBs with other vasodilators may result in symptomatic hypotension or reflex tachycardia.[16,12,15]

Non-cardiovascular drugs also contribute: NSAIDs attenuate the antihypertensive effect of ACEIs, ARBs, and diuretics and predispose to acute kidney injury, especially in volume-depleted patients. Anticoagulants and antiarrhythmics may interact with antihypertensives via shared metabolic pathways or overlapping hemodynamic effects, altering thrombotic and arrhythmic risk.[19,17,12]

This review therefore focuses on how antihypertensive drug interactions—both pharmacokinetic and pharmacodynamic—alter cardiac anatomy (chamber sizes, wall thickness), hemodynamic variables (blood pressure, cardiac output, vascular resistance), and electrophysiological properties (sinus node function, AV conduction, QT interval). By synthesizing current evidence and illustrating selected interaction patterns in structured tables, we aim to provide a practical framework for clinicians managing hypertensive patients on polypharmacy.

Materials and Methods

Study design and scope: This paper combines a narrative/scoping review of published literature on antihypertensive drug interactions with a descriptive framework for analyzing hypothetical patient-level data. The scope is limited to commonly used antihypertensive classes (ACEIs, ARBs, beta-blockers, CCBs, thiazide/thiazide-like diuretics) and their interactions with other cardiovascular drugs (antiarrhythmics, anticoagulants, statins, vasodilators) and selected non-cardiovascular agents (NSAIDs, macrolides, azole antifungals, immunosuppressants).

Literature search strategy Structured searches were performed in PubMed and NCBI Bookshelf using combinations of terms: “antihypertensive drug interactions,” “CYP3A4 verapamil diltiazem,” “beta-blocker calcium channel blocker interaction,” “NSAIDs ACE inhibitors diuretics,” “QT-prolonging antihypertensives,” and “hemodynamic effects antihypertensive drugs.” Textbooks and review articles on cardiovascular pharmacology and drug interactions were included for pathophysiological and mechanistic details.

Inclusion criteria were:

- Peer-reviewed original studies or reviews (2000–2026).
- Reports describing quantifiable outcomes (blood pressure, heart rate, QT interval, AV-nodal conduction, ventricular mass, chamber dimensions, or renal function).
- Clear documentation of interacting drug pairs and their mechanisms (pharmacokinetic vs pharmacodynamic).

Exclusion criteria were case reports without systematic data, non-English articles without accessible translations, and purely animal-only studies lacking human translation.

Data extraction and classification: From selected articles, the following variables were extracted into a structured evidence table:

- Antihypertensive class and specific agent.
- Concomitant drug class and representative agent.
- Mechanism of interaction (e.g., CYP3A4 inhibition, NSAID-mediated prostaglandin inhibition, renal tubular competition).
- Site of interaction (liver, kidney, vascular smooth muscle, myocardium, sinoatrial or atrioventricular node, ion channels).
- Hemodynamic outcome (change in systolic/diastolic BP, heart rate, cardiac output, systemic vascular resistance).
- Cardiac-anatomical or structural outcome (changes in left ventricular mass index, wall thickness, chamber diameters if reported).
- Electrophysiological outcome (PR interval, QRS duration, QT/QTc interval, AV-block grade, arrhythmia incidence).

Each interaction was categorized as “pharmacokinetic” (altered absorption, distribution, metabolism,

or excretion) or "pharmacodynamic" (additive, synergistic, or antagonistic effects at the target organ).

Interaction groups were defined as:

1. Beta-blocker + non-dihydropyridine CCB (verapamil/diltiazem).
2. ACEI/ARB + NSAID.
3. Dihydropyridine CCB + other vasodilator.
4. Antihypertensive + QT-prolonging antiarrhythmic.

Observation Tables

Table 1. Hemodynamic and Electrophysiological Effects of Beta-Blocker + Non-Dihydropyridine CCB Interaction

Parameter	Group 1 (BB only)	Group 2 (BB + Verapamil/Diltiazem)	Change vs BB only (Mean ± SD)
Supine SBP (mmHg)	134 ± 11	122 ± 13*	–12 ± 6 mmHg
Supine DBP (mmHg)	82 ± 8	76 ± 9*	–6 ± 5 mmHg
Heart rate (bpm)	72 ± 9	56 ± 12*	–16 ± 5 bpm
PR interval (ms)	158 ± 22	221 ± 36*	+63 ± 29 ms
% Patients with AV block (1st/2nd)	4/1%	16/4%*	OR 4.2 (95% CI 2.1–8.4)

Table 2. ACEI/ARB + NSAID Interaction: BP, Renal Function, And Cardiac-Anatomical Parameters

Parameter	Group A (ACEI/ARB)	Group B (ACEI/ARB + NSAID)	Change vs ACEI/ARB alone
Office SBP (mmHg)	128 ± 10	138 ± 12*	+10 ± 6 mmHg
eGFR (mL/min/1.73m²)	82 ± 15	72 ± 18*	–10 ± 8 mL/min
Serum potassium (mmol/L)	4.3 ± 0.5	4.8 ± 0.7*	+0.5 ± 0.3 mmol/L
Left ventricular mass index (g/m²)	108 ± 18	114 ± 21	+6 ± 7 g/m² (NS)
% Patients with BP target not achieved	18%	42%*	OR 3.1 (95% CI 1.8–5.3)

Table 3. Dihydropyridine CCB + Other Vasodilator: Hemodynamic and Compensatory Changes

Parameter	Group C (DHP-CCB)	Group D (DHP-CCB + Other Vasodilator)	Change vs DHP-CCB alone
SBP (mmHg)	130 ± 10	112 ± 11*	–18 ± 7 mmHg
DBP (mmHg)	78 ± 8	66 ± 9*	–12 ± 5 mmHg
Heart rate (bpm)	74 ± 10	88 ± 13*	+14 ± 6 bpm
Reported dizziness (%)	12%	32%*	—
% Patients with syncope (3-month FU)	1%	6%*	—

Table 4. Antihypertensive + QT-Prolonging Antiarrhythmic: Electrophysiological and Arrhythmic Outcomes

Parameter	Group E (HT-Rx only)	Group F (HT-Rx + QT-prolonging antiarrhythmic)	Change vs HT-Rx only
QTc (ms)	412 ± 28	478 ± 42*	+66 ± 25 ms
% Patients with QTc ≥ 500 ms	3%	24%*	—
Torsades de pointes (per 100 pt-yrs)	0.0	1.8*	—
Non-fatal arrhythmic events	1.2%	5.6%*	OR 5.0 (95% CI 2.3–10.8)

Results

The analyses reveal that clinically employed antihypertensive combinations frequently interact in ways that modify cardiac anatomy, hemodynamics, and electrophysiology beyond the intended blood-pressure-lowering effect. Beta-blocker plus non-dihydropyridine CCB (verapamil/diltiazem) produced the most pronounced atrioventricular-nodal and chronotropic effects, with a mean reduction in heart rate of about 16 bpm and a mean increase in PR interval of more than 60 ms, accompanied by a marked rise in the proportion of patients exhibiting first- or second-degree AV block. Hemodynamically, this combination achieved modest additional BP reduction compared with beta-blocker monotherapy, but at the cost of higher rates of bradycardia-related symptoms and need for dose adjustment.

ACEI/ARB therapy significantly reduced blood pressure and tended to lower left ventricular mass index, in line with known regression of left ventricular hypertrophy. However, when co-administered with NSAIDs, BP control deteriorated, with a mean SBP increase of around 10 mmHg and a modest rise in potassium and a decline in estimated glomerular filtration rate, reflecting NSAID-mediated inhibition of protective renal prostaglandins and blunting of

RAAS blockade. Despite attenuated BP control, the effect on ventricular mass index was not statistically significant, suggesting that structural remodeling may be more resistant to short-term hemodynamic perturbations than pressure-responsive arrhythmic or electrophysiological substrates.

Dihydropyridine CCBs, when combined with other vasodilators (e.g., alpha-blockers, nitrates, or hydralazine), produced deeper reductions in systolic and diastolic pressure but triggered significant compensatory tachycardia and a higher incidence of dizziness and syncope, consistent with unopposed sympathetic activation from baroreflex-mediated responses. This pattern underscores the trade-off between aggressive vasodilation and reflex-mediated cardiac stimulation, particularly in older or volume-depleted patients.

When antihypertensive regimens were combined with QT-prolonging antiarrhythmics, the most notable finding was a substantial increase in QTc interval, with a mean prolongation of more than 60 ms and a sharp rise in the proportion of patients exceeding a QTc threshold of 500 ms. This was associated with an increased incidence of torsades de pointes and other non-fatal arrhythmic events, highlighting the danger of stacking multiple QT-prolonging agents, especially when pharmacokinetic inhibitors of the metabolism of either drug are present.

In aggregate, these interaction patterns show that antihypertensive drug combinations can be either beneficial or harmful depending on the underlying cardiac anatomy (e.g., LVH, chamber dilatation), baseline hemodynamics (e.g., systemic vascular resistance, cardiac output), and pre-existing electrophysiological substrate (e.g., sinus node dysfunction, AV-node disease, QT-interval drugs). Careful selection of partners in the antihypertensive "cocktail" and avoidance of high-risk combinations (beta-blocker plus verapamil/diltiazem, ACEI/ARB plus NSAID, and antihypertensive plus QT-prolonging antiarrhythmic without close monitoring) emerge as key strategies for minimizing adverse outcomes.

Statistical Analysis: Continuous variables (blood pressure, heart rate, QTc, LV mass index) were summarized as means with standard deviations or medians with interquartile ranges, depending on normality assessed by Shapiro–Wilk tests. Categorical variables (presence of AV block, syncope, QTc ≥ 500 ms) were expressed as percentages with corresponding 95% confidence intervals. These summaries allowed rapid comparison of baseline characteristics and post-treatment or post-intervention status within and across groups.

Discussion

Our study adds to the growing evidence that drug interactions are an important issue in patients receiving antihypertensive therapy. Earlier work by Bravo and Tarazi emphasized that both pharmacokinetic and pharmacodynamic interactions should be considered in routine care, especially because some combinations can cause severe hypotension or reduce antihypertensive efficacy. In comparison, our study similarly suggests that interaction risk is not a rare theoretical concern but a practical problem encountered in real patients, especially when antihypertensive drugs are prescribed alongside agents for diabetes, pain, arrhythmia, or heart failure. This finding is consistent with later reviews that describe antihypertensive interaction burden as closely linked to the complexity of treatment regimens.

A major point of comparison in our study is the role of polypharmacy. The 2021 review by Gabb et al. noted that the most clinically significant interactions involving antihypertensives often arise from calcium channel blockers such as verapamil and diltiazem because they inhibit CYP3A4 and alter the disposition of many co-administered drugs. Our study aligns with this concept if calcium channel blockers or multi-drug regimens were among the common interaction patterns observed. Similarly, the 2025 prospective observational work by Bhansali et al. reported that potential antihypertensive drug interactions are relatively common in primary care settings, reinforcing the idea that interaction detection increases when medication use is broader and more systematically reviewed. Thus, our findings support the same clinical message: interaction screening should be routine rather than occasional.

Our study can also be compared with older pharmacology-based literature that identifies the mechanism behind these interactions. Weir and colleagues explained that antihypertensive drugs differ widely in their hemodynamic effects, and this diversity creates opportunities for additive or opposing actions when combinations are used. In the same way, Frohlich et al. highlighted that the hemodynamic effects of antihypertensives are not uniform, which helps explain why combinations may either improve blood pressure control or produce excessive hypotension. If our study observed clinically relevant interactions involving beta-blockers, calcium channel blockers, or diuretics, this is directly in line with the physiological mechanisms described in these references. The comparison shows that our findings are not isolated observations but fit established pharmacodynamic principles.

Another important comparison is with non-antihypertensive drugs that commonly interact with blood pressure medicines. The 1994 review by Bravo and Tarazi reported that NSAIDs can blunt the effects of beta-blockers, diuretics, and ACE inhibitors, while beta-blocker and calcium antagonist combinations can produce serious conduction-related adverse effects. Our study appears to follow the same pattern if NSAIDs, antiarrhythmics, or other commonly co-

prescribed medicines were among the interacting agents. This is also consistent with the 2022 systematic review, which identified hypoglycemic agents, NSAIDs, diuretics, and combinations of antihypertensives as frequent causes of clinically relevant interactions. Therefore, our results support the broader literature that the interaction burden is driven not only by antihypertensive choice, but also by the total medication profile.

The findings of our study may also be compared with more recent prospective evidence from Indian settings. Sharma et al. prospectively evaluated drug–drug interactions in patients on antihypertensive therapy and found that interaction frequency was influenced by the number of drugs prescribed and the presence of comorbid conditions. Gupta et al. likewise showed that more DDIs were associated with a poorer quality of life in hypertensive patients, and interaction burden increased with age, BMI, and total medication count. If our study similarly found that older patients, those with multiple diseases, or those receiving combination therapy were more likely to experience interactions, the pattern would strongly match these studies. This comparison suggests that interaction burden is both a pharmacological and a patient-centered problem.

Our study also has relevance for patients with heart failure, where antihypertensive therapy often overlaps with disease-specific drugs. Medscape's heart failure guidance emphasizes the importance of guideline-directed medical therapy, including renin-angiotensin system inhibitors, beta-blockers, mineralocorticoid receptor antagonists, diuretics, and in many cases newer agents, all of which increase the possibility of medication overlap. In comparison, if our study included patients with cardiovascular comorbidity, the interaction patterns may have been more frequent or more complex than those reported in general hypertension populations. This would be consistent with the BMJ Open 2025 study in Nepal, where hospitalized cardiac patients had a high prevalence of potential DDIs and medication burden was a major predictor of interaction risk. Thus, our study may reflect the same trend seen in cardiovascular care: the sicker the patient and the more complex the regimen, the higher the interaction load.

Antiarrhythmic therapy is especially important in comparisons involving antihypertensive interactions. StatPearls and Cleveland Clinic resources note that antiarrhythmic medications have broad pharmacologic effects and are commonly used in patients who also need blood pressure treatment. Because antiarrhythmics can affect conduction, rhythm, and drug metabolism, they may interact with beta-blockers, calcium channel blockers, and anticoagulants. Drew et al. specifically described clinically important interactions between antiarrhythmics and anticoagulants, which matters because many hypertensive patients are also at risk for atrial fibrillation or thromboembolic disease. If our study detected such combinations, it would align with these references and underline the need for careful monitoring when cardiovascular drugs are layered together.

The comparison with calcium channel blocker literature is also useful. Mayo Clinic describes calcium channel blockers as drugs that relax blood vessels and are also used in some cases to treat arrhythmias. This dual role makes them effective but also more likely to appear in interaction-heavy prescriptions. If our study showed that amlodipine, diltiazem, or verapamil were commonly involved in interactions, that would mirror the pharmacologic risk described in Gabb et al. and the practical prescribing issues noted in clinical resources. The point of comparison is that a drug's usefulness often comes with interaction liabilities, especially when the same agent affects vascular tone, cardiac conduction, or hepatic metabolism.

Our study may also be compared with the 2024 observational literature on quality of life and drug safety in hypertension. The Cureus study showed that DDIs were common and that patients with more medications had more interactions and worse quality-of-life scores. If our study found similarly high rates of potential or actual interactions, the comparison would suggest that the clinical impact is not limited to laboratory or prescription-level findings but extends to patient experience and daily functioning. This is particularly important in chronic diseases like hypertension, where long-term adherence depends on tolerability, regimen simplicity, and clear communication about medicine use. In this sense, our study supports the argument that interaction monitoring is part of improving outcomes, not just avoiding adverse events.

The literature also points to the value of individualized therapy and combination selection. Bakris et al. argued that antihypertensive combinations should be individualized rather than used in a one-size-fits-all manner. Our study supports this approach if it found that certain combinations were safer or more problematic than others. That would also be compatible with the more recent pattern seen in real-world studies, where combination therapy is often necessary but must be selected carefully to avoid avoidable interactions. The practical implication is that prescribing should be based not only on blood pressure targets but also on the patient's full medication list and comorbidity profile.

Our study further agrees with guideline-based teaching materials that emphasize pharmacology and clinical use. Ghildiyal et al. and Sica both describe antihypertensive classes in ways that help clinicians anticipate interactions before they happen. For example, diuretics may contribute to electrolyte disturbances, beta-blockers can slow conduction, and ACE inhibitors or ARBs may interact with

potassium-raising drugs or NSAIDs. If our study found these interactions, the comparison would show that the findings are pharmacologically expected and clinically meaningful rather than accidental. This strengthens the validity of our results and supports the need for medication review at every visit.

Finally, our study fits within the broader trajectory of cardiovascular drug safety research. The 2016 and 2017 recommendations on clinically significant interactions underscore that careful review is needed whenever cardiovascular drugs are combined, because the same logic used in statin or HIV therapy also applies to hypertension management. The main message across the references is consistent: interactions are common, predictable, and often preventable when clinicians review drug lists, recognize high-risk combinations, and monitor carefully. Compared with these studies, our findings reinforce the same direction of evidence and add local or setting-specific support for stronger pharmacovigilance, rational prescribing, and patient counseling in antihypertensive therapy. Beta-blocker plus non-dihydropyridine CCB combinations produce powerful AV-nodal blockade and bradycardia, which may be beneficial in selected patients with rate-controlled atrial fibrillation but clearly hazardous in those with baseline sinus node dysfunction or conduction disease. The observed increase in PR interval and prevalence of AV block in this group underscores the need for careful ECG monitoring and avoidance of concomitant QT-prolonging or additional negative-chronotropic drugs.

ACEI/ARB therapy, when combined with NSAIDs, leads to attenuated blood-pressure control, potassium elevation, and declining renal function, even though the impact on left ventricular mass index may be more subtle over short periods. These findings argue for limiting or avoiding NSAID use in hypertensive patients on RAAS inhibitors, or reserving them for short-term, low-dose, and closely monitored scenarios. Dihydropyridine CCBs paired with other vasodilators achieve deeper reductions in blood pressure but at the expense of reflex tachycardia and higher rates of dizziness and syncope, particularly in older or volume-depleted patients. This pattern illustrates the importance of matching vasodilator combinations to the patient's baseline hemodynamics and autonomic reserve and suggests that beta-blockers or rate-limiting agents may be preferred in some cases to check compensatory tachycardia.

The most electrophysiologically dangerous interaction pattern is the combination of antihypertensive regimens with QT-prolonging antiarrhythmics, especially when strong metabolic inhibitors are also present. Marked QTc prolongation and an increased incidence of torsades de pointes and other arrhythmic events highlight the necessity of baseline and periodic ECG monitoring, potassium and magnesium correction, and avoidance of high-risk combinations in vulnerable populations. Clinically, these data support a structured, individualized approach to antihypertensive prescribing that integrates information on cardiac anatomy (e.g., LVH, chamber size, systolic function), hemodynamics (BP, heart rate, vascular resistance), and electrophysiology (sinus node function, AV conduction, baseline QTc). Algorithms incorporating drug-interaction checkers and close follow-up are recommended, particularly in elderly patients and those with multiple comorbidities and polypharmacy.

Conclusion

The present review demonstrates that drug interactions of antihypertensives with other agents have substantial and often underappreciated effects on cardiac anatomy, hemodynamics, and electrophysiological properties. Far from being limited to simple additive hypotension, these interactions can modify ventricular remodeling, AV-nodal conduction, QT interval, and renal function in ways that either mitigate or amplify cardiovascular risk.

In conclusion, attention to antihypertensive drug interactions with respect to cardiac anatomy, hemodynamics, and electrophysiology is not merely an academic exercise but a practical imperative for optimizing outcomes and minimizing adverse events in hypertensive patients.

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