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Review

A Review of the Application Progress of Non-Invasive Hemodynamic Monitoring in the Precision Treatment of Hypertension

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ABSTRACT: Hypertension affects many patients worldwide, and its precise treatment is the focus of clinical research. Currently, conventional clinical methods for monitoring blood pressure can only intermittently measure systolic and diastolic blood pressure and cannot monitor important hemodynamic parameters such as cardiac output (CO), systemic vascular resistance (SVR), and arterial elasticity, thereby affecting the formulation of individualized treatment plans. In recent decades, the emergence of non-invasive hemodynamic monitoring methods has addressed these clinical challenges. These methods use non-invasive methods to monitor parameters such as cardiac pumping function, vascular resistance, and volume status, helping clinicians better understand the pathophysiology of hypertension and facilitating a shift from "empirical blood pressure reduction" to "precision treatment based on hemodynamics". This article aims to introduce the technical principles, main parameters, and clinical applications of non-invasive hemodynamic monitoring, with a focus on discussing its clinical value in hypertension classification, formulation of individualized treatment plans, assessment of treatment effects, and management of special populations. Based on this, future application and development directions are proposed, aiming to provide references and evidence for the clinical practice of precise hypertension treatment.

Keywords: Non-invasive hemodynamics; Hypertension; Precision treatment; Monitoring technology; Individualized treatment

1. Introduction

Hypertension is a multifactorial genetic disorder caused by the combined effects of genetic, environmental, and neuroendocrine factors. The fundamental pathological changes involve remodeling of small arteries throughout the body, accompanied by disturbances in hemodynamic balance. According to 2023 statistics from the World Health Organization, approximately 1.28 billion adults worldwide are affected by hypertension [1]. However, the control rate remains below 20%. One of the main reasons for

this is the limitations of the current management model. Clinically, the diagnosis and monitoring of hypertension still largely rely on intermittent measurements using clinic or home blood pressure monitors. These "point-like" data can only reflect blood pressure at a given moment and cannot fully capture the dynamic, continuous characteristics of hemodynamics, such as cardiac output (CO), systemic vascular resistance (SVR), arterial elasticity, and the coupling relationship between the heart and blood vessels [2].

Studies have confirmed that the hemodynamic phenotypes of hypertension exhibit significant heterogeneity: children and adolescent patients with primary hypertension often present with increased heart rate (HR), increased CO, and increased stiffness of the proximal aorta, rather than merely increased peripheral resistance [2,3]; in elderly hypertension, the main manifestations are increased SVR due to arteriosclerosis and abnormal left ventricular work index (LCWI), along with impaired diastolic function. A more crucial point is that, from a hemodynamic perspective, hypertension does not follow a single pattern. According to the classic formula (blood pressure = CO × SVR), its essence can be regarded as a continuous spectrum shifting from "dominated by increased CO" to "dominated by increased SVR" [4]. The former is more common in patients with excessive activation of the sympathetic nervous system (SNS) in the early stage or in younger individuals, while the latter is more prevalent in elderly patients with long-term poorly controlled blood pressure and vascular remodeling.

Despite the potential of hemodynamic phenotyping to improve treatment precision, current major guidelines, such as those from the American College of Cardiology/American Heart Association (ACC/AHA), do not yet recommend routine phenotyping in clinical practice. This is primarily due to the lack of large-scale prospective trials validating the clinical utility of phenotype-guided therapy, the heterogeneity of non-invasive measurement techniques, and the absence of standardized clinical pathways for integrating hemodynamic parameters into routine hypertension management. However, accumulating evidence suggests that phenotype-guided approaches may improve blood pressure control rates and reduce adverse drug reactions, supporting the need for further research and clinical integration [5].

Traditional blood pressure measurement methods cannot distinguish between these two types, often leading to treatment stuck in a "one-size-fits-all" approach. For example, overusing vasodilators in patients with a high-output state may cause reflexive tachycardia, while using beta-blockers too early in patients with a high-resistance state may further suppress CO.

The emergence of non-invasive hemodynamic monitoring techniques has provided a breakthrough solution to this contradiction. Through non-invasive methods such as bioimpedance, ultrasound Doppler, and arterial tonometry, continuous and dynamic acquisition of parameters such as CO, stroke volume (SV), SVR, and arterial elasticity can be achieved. This enables precise identification of patients' hemodynamic phenotypes and the subsequent formulation of individualized treatment strategies [6]. For example, beta-blockers are preferred for patients with high CO to reduce heart rate and myocardial contractility, while angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) are selected for patients with high SVR to improve vascular endothelial function. This "targeted intervention" model has been shown to achieve blood pressure control more quickly and reduce adverse drug reactions.

This article systematically reviews the application progress of non-invasive hemodynamic techniques in the precise treatment of hypertension, combined with the latest research evidence, clarifying how they promote "treatment based on the specific condition" by revealing hemodynamic mechanisms, and discussing the key directions for future technology optimization and clinical integration.

2. Literature Search Strategy

This review was conducted following a systematic approach. A comprehensive literature search was performed in PubMed, Embase, Web of Science, and Cochrane Library for articles published from January 2010 to December 2025. The search terms included combinations of: ("non-invasive hemodynamic monitoring" OR "impedance cardiography" OR "bioimpedance" OR "arterial tonometry" OR

"photoplethysmography") AND ("hypertension" OR "blood pressure") AND ("precision treatment" OR "phenotype" OR "personalized therapy"). The search was restricted to English-language publications. Inclusion criteria were: (1) original research or review articles on non-invasive hemodynamic monitoring in hypertensive patients; (2) studies reporting at least one hemodynamic parameter. Exclusion criteria were: (1) case reports with fewer than 5 patients; (2) invasive hemodynamic monitoring studies; (3) conference abstracts without full text.

3. Non-Invasive Hemodynamic Monitoring Technology: Principles, Methods, and Core Parameters

3.1. Main Technical Principles and Clinical Applicability

The core of non-invasive hemodynamic monitoring technology lies in obtaining functional parameters of the cardiovascular system through different physical principles.

The bioimpedance method measures CO by monitoring changes in thoracic impedance, based on the change in electrical resistance caused by the volume change of blood in the thoracic cavity. This method offers simplicity and low cost, making it suitable for long-term monitoring and outpatient follow-up. However, its accuracy can be affected by respiratory movements, poor electrode contact, and body fluid status [7]. Ultrasonic Doppler is based on the Doppler effect and directly measures blood flow velocity, providing real-time, dynamic information. It has become a common method for clinical assessment of cardiac function. Its main limitation is operator dependence; measurement errors in aortic diameter can lead to CO deviations exceeding 10% [8]. Photoplethysmography (PPG) indirectly calculates hemodynamic parameters by analyzing the waveform characteristics of peripheral pulses and is often integrated into wearable devices due to its portability. PPG signals are sensitive to skin pigmentation, ambient light, and motion artifacts [9]. Arterial tonometry relies on continuous monitoring of arterial pressure waveforms and uses mathematical models to calculate CO and vascular resistance. It has demonstrated good accuracy in perioperative settings but requires precise sensor placement and calibration [10]. Each of these techniques has its own advantages. When choosing a method in actual clinical practice, factors such as the monitoring target, patient's specific conditions, and medical conditions need to be comprehensively considered.

Meanwhile, emerging technologies represented by wearable cloud stethoscopes and AI hemodynamic monitoring systems are systematically expanding the boundaries of non-invasive monitoring through three driving forces: hardware miniaturization and multimodal integration, data cloudification and remote processing, as well as intelligent and deep analysis [11,12]. They not only enable continuous and non-invasive acquisition of heart sounds, electrocardiograms, and even derived hemodynamic parameters, but also transform data into deep clinical insights through AI, thereby extending the monitoring scenario from core hospital departments to outpatient clinics, communities, and even households [13,14].

3.2. Monitoring Parameters and Their Clinical Significance

The core parameters of non-invasive hemodynamic monitoring mainly assess cardiac function, vascular status, and overall circulatory load.

3.2.1. Parameters of Cardiac Pumping Function

Cardiac output (CO, L/min) refers to the total volume of blood ejected by the left ventricle into the systemic circulation per minute. It is a fundamental index for evaluating overall circulatory function [15]. In the early stage of hypertension, CO often increases as a compensatory response (defined as a high-output hemodynamic state); however, long-term poorly controlled hypertension can induce left ventricular myocardial hypertrophy, which may lead to a gradual decrease in CO in the advanced stage [16]. Stroke volume (SV, mL/beat) represents the volume of blood pumped by the left ventricle per systolic contraction, directly reflecting myocardial systolic function. A persistent decrease in SV is a typical indicator of

impaired cardiac pumping function. Cardiac index (CI, L/min/m^2) is the CO normalized by body surface area, eliminating the influence of individual body size differences and enabling a more objective comparison of cardiac function across different populations [17].

3.2.2. Vascular Resistance and Elasticity Parameters

Systemic vascular resistance (SVR, dyn·s·cm^{-5}) reflects the resistance of small arteries to blood flow. An increase in SVR is a core feature of high-resistance hypertension and is closely related to vascular endothelial dysfunction and arteriosclerosis [18]. Systemic vascular resistance index (SVRI, dyn·s·cm^{-5}·m^2) is SVR corrected for body surface area and more accurately reflects overall vascular load. Pulse wave velocity (PWV, m/s) is the gold standard for assessing arterial stiffness [19]. A faster PWV (such as >10 m/s) indicates vascular sclerosis and increases the risk of cardiovascular events.

3.2.3. Volume and Coupling Parameters

Left ventricular work index (LCWI, g·m/m^2) comprehensively assesses the efficiency of cardiac work. Abnormal LCWI indicates an imbalance in the coupling between the ventricles and the vasculature. Elderly hypertensive patients often have elevated LCWI accompanied by impaired diastolic function. By monitoring changes in CO and peripheral resistance during the transition from supine to standing, autonomic regulation function can be assessed (e.g., a ≥ 15% decrease in CO during standing in elderly patients suggests risk of orthostatic hypotension). The comprehensive analysis of these parameters can precisely differentiate hypertension subtypes and provide direct evidence for individualized treatment [20].

4. Application of Non-Invasive Hemodynamic Monitoring in Hypertension Classification: From "Phenotype" to "Precision Phenotype"

4.1. Classification Criteria for Hypertension Based on Hemodynamic Factors

The traditional classification of hypertension has relied mainly on blood pressure levels (such as grade 1/2/3) or causes (primary/secondary), but has overlooked the heterogeneity of hemodynamic mechanisms. Advances in non-invasive monitoring have promoted the development of "hemodynamic classification" [21]. Currently, the "CO-SVR two-dimensional classification" is widely adopted internationally, dividing hypertension into four phenotypes (Table 1). Non-invasive technologies such as bioimpedance cardiography and echocardiography can accurately distinguish these types, providing an objective basis for precise classification [22].

Table 1. The Four Major Phenotypes of Blood Pressure.

Classification	CO	SVR	Pathophysiological Characteristics	Common Population
High-output type	Significantly increased (>average value of the same age group)	Normal or slightly increased	Activation of the SNS, increased HR, enhanced cardiac contraction	Young/obese patients
High peripheral resistance type	Normal or slightly decreased	Significantly increased (>1500 dyn·s·cm^{-5})	Vascular remodeling, endothelial dysfunction, arteriosclerosis	Elderly/long-term uncontrolled patients
Mixed type	Increased with elevated SVR	Increased	Abnormal coordination of CO and SVR during disease progression	Moderate to severe hypertension patients
Low activity type	Decreased (below the average of the same age group)	Compensatory increase	Heart failure, myocardial ischemia, excessive use of beta-blockers	Older patients/patients with comorbidities

Note: The normal range of SVR is typically 800–1200 dyn·s·cm^{-5} (after correcting for body surface area, SVRI is 1500–2000 dyn·s·cm^{-5}·m^2).

4.2. Clinical Value of Phenotyping

4.2.1. Guiding Individualized Treatment Strategies

There are significant differences in response to antihypertensive drugs among different subtypes. Based on pathophysiological reasoning and preliminary evidence, it has been hypothesized that patients with high-output type might benefit from beta-blockers or non-dihydropyridine calcium antagonists to reduce heart rate and myocardial contractility [23], whereas those with high-resistance type could potentially respond to ACEIs, ARBs, or calcium channel blockers. For mixed-type patients, combination therapy (e.g., beta-blocker plus ACEI) has been suggested to balance CO and SVR. However, these phenotype-guided therapeutic strategies remain largely hypothesis-generating, as they are derived from small observational studies and have not yet been incorporated into major hypertension guidelines. High-quality randomized controlled trials are needed before any definitive treatment algorithm can be recommended.

4.2.2. Prediction of Drug Reactivity and Efficacy

Studies have shown that patients with high resistance type have a significantly greater reduction in blood pressure when treated with calcium antagonists compared to those with high-output type, while beta-blockers have a significantly better effect on controlling resting heart rate in patients with high-output type compared to other types. This “type-matched-drug” model can reduce ineffective treatments and improve treatment compliance [22].

4.2.3. Assessing the Risk of Disease Progression

Research data support that patients with hypertension exhibiting high resistance or refractory characteristics have a significantly elevated risk of cardiovascular events [24]. An Italian study followed up 340 patients with hypertension (including 130 with refractory hypertension) for approximately 5 years. The results showed that the risk of cardiovascular events in patients with truly refractory hypertension was 2.94 times higher than that of hypertensive patients in the control group [25]. Regular monitoring of SVR and PWV can help identify high-risk individuals early and strengthen intervention.

4.2.4. Mechanistic Insights and Targeted Therapy

Hemodynamic classification provides a new perspective on the pathophysiological study of hypertension [26]. For instance, patients with high-output type often have elevated levels of markers of sympathetic activity (such as norepinephrine), suggesting that targeted sympathetic modulation (such as renal artery ablation) may be a potential treatment strategy [27]. For patients with high resistance type, impaired vascular endothelial function (such as reduced nitric oxide bioavailability) suggests the potential value of endothelial protectants (such as statins) [28].

5. The Core Role of Non-Invasive Hemodynamics in Hypertension Treatment: From “Empirical” to “Hypothesis-Driven Adjustment”

5.1. A Hypothesis-Generating Framework for Initial Drug Selection Based on Hemodynamic Phenotypes

Traditional antihypertensive therapy follows an empirical stepwise approach, which may delay blood pressure control and cause adverse drug reactions. It has been hypothesized that non-invasive hemodynamic monitoring could potentially inform phenotype-specific initial drug selection by identifying a patient’s predominant hemodynamic pattern, but this remains an area of active investigation.

Based on pathophysiological reasoning and preliminary evidence from small-scale studies, several potential associations have been suggested: high-output type might benefit from beta-blockers or non-dihydropyridine calcium channel blockers to reduce heart rate and contractility [29]; high-resistance type

may respond to ACEIs, ARBs, or dihydropyridine CCBs; mixed type may necessitate dual therapy; low-activity type would theoretically require cautious afterload reduction and avoidance of excessive beta-blockade [30]. These proposed strategies, however, remain largely hypothesis-generating. While small studies suggest that phenotype-guided initial therapy may achieve faster blood pressure control with fewer drug substitutions than empirical treatment [5], high-quality randomized controlled trials are urgently needed to validate these preliminary observations before any clinical algorithm can be recommended.

5.2. Efficacy Evaluation and Dynamic Adjustment of the Plan

5.2.1. Short-Term Efficacy Evaluation

During the initial treatment phase (1–4 weeks), monitoring changes in CO and SVR could provide insights into drug selection, though this approach requires further validation. For example, from a physiological perspective, if SVR does not significantly decrease (still >1500 dyn·s·cm^{-5}) after starting ACEI therapy, one might consider the possibility that a dose increase or a more potent ARB could be investigated; if CO becomes excessively low (<4.0 L/min) while blood pressure is controlled but the patient experiences fatigue, it might be reasonable to evaluate whether reducing the beta-blocker dose is appropriate. However, it must be emphasized that specific thresholds for intervention remain to be established and require rigorous validation in future studies [5].

5.2.2. Long-Term Prognosis Prediction

The relationship between longitudinal hemodynamic changes and long-term outcomes in patients with hypertension remains a current research hotspot. Some observational studies have suggested that persistently elevated systemic vascular resistance may be associated with an increased risk of subsequent cardiovascular events, while simultaneous improvements in cardiac output and vascular resistance may be beneficial for target organ recovery [31]. However, evidence supporting specific quantitative estimates is currently limited and often derived from small samples or post-hoc analyses, and has not been consistently validated in large-scale prospective cohort studies. Therefore, high-quality, long-term prospective studies are needed in the future to establish robust risk prediction models based on continuous non-invasive hemodynamic parameters.

5.2.3. Identification of Treatment Resistance

Approximately 15–20% of hypertensive patients have “refractory hypertension” (blood pressure uncontrolled despite three or more medications) [32]. Non-invasive monitoring can reveal the underlying mechanisms—such as hidden volume overload due to elevated LCWI, excessive sympathetic overactivation resulting in abnormally elevated SVRI, or compensatory increase in CO-induced renal artery stenosis—which are secondary hypertension. Adjusting strategies based on these mechanisms can significantly improve control rates [33].

6. Management of Hypertension in Special Populations

6.1. Elderly Hypertensive Patients: Focus on Vascular Stiffness and Orthostatic Hypotension

The pathological features of elderly hypertensive patients (≥65 years) typically include arteriosclerosis, increased SVR, and impaired diastolic function [34]. Non-invasive hemodynamic monitoring can precisely quantify these changes. For example, assessing PWV via arterial tonometry helps set more reasonable blood pressure targets, avoiding excessively low blood pressure that may compromise cerebral perfusion. Monitoring changes in CO from supine to standing can identify orthostatic hypotension risk individuals

[35]. Based on this, treatment can be adjusted, for example, prioritizing long-acting calcium channel blockers (CCB) over alpha-blockers.

6.2. Patients with Refractory Hypertension: Uncovering Mechanisms and Optimizing Intervention

Refractory hypertension refers to a condition where blood pressure remains uncontrolled even after the administration of three or more antihypertensive drugs. The underlying mechanisms include volume overload, excessive activation of the sympathetic nervous system/renin-angiotensin-aldosterone system (RAAS), and vascular hyperreactivity [36]. Traditional empirical treatment struggles to precisely target the core components, whereas hemodynamic classification offers a new approach for individualized treatment. For example, an SVRI > 2500 dyn·s·cm^{-5}·m^2 indicates excessive activation of the renal artery stenosis (RAS), and ACEI/ARB is preferred; an LCWI > 4.2 kg·m/m^2 indicates volume overload, requiring intensified diuretics (such as spironolactone). In addition, for patients considering renal artery ablation, non-invasive monitoring identifies those with SVRI > 2500 dyn·s·cm^{-5}·m^2 and LCWI > 4.2 kg·m/m^2, whose treatment response rate reaches 75%, significantly higher than that of the unscreened population (45%) [37]. A prospective study confirmed that patients with baseline peripheral vascular resistance index >3000 dyn·s·cm^{-5}·m^2 had a significantly reduced response rate to spironolactone treatment (35% *vs*. 68%, $p = 0.003$) [38].

6.3. Patients with Gestational Hypertension: Dynamic Monitoring and Maternal-Fetal Safety

The hemodynamic characteristics of gestational hypertension (including preeclampsia) change dynamically with gestational weeks. In early normal pregnancy, peak CO can reach 150% of baseline, and SVR decreases by approximately 35% [39]. However, patients with preeclampsia exhibit vascular resistance 20–30% higher than in normal pregnancy and a reduced increase in CO. These changes can be detected 4–6 weeks before clinical symptoms appear [4]. Studies have shown that uterine artery PI > 1.2 and umbilical artery S/D ratio > 3.0, assessed by ultrasound Doppler, can predict 90% of severe preeclampsia [40]. Regarding antihypertensive strategy selection, hemodynamic classification-guided treatment shows significant advantages: patients with high-resistance type (SVRI > 1200 dyn·s·cm^{-5}·m^2) respond better to alpha-blockers, while those with high-output type (CI > 4.0 L/min/m^2) are more suitable for calcium channel blockers [39]. Research has confirmed that treatment based on non-invasive classification reduces severe maternal complications by 40% and the incidence of fetal growth restriction by 35% [4].

7. Technical Limitations and Clinical Challenges

7.1. Technical Limitations

7.1.1. Measurement Accuracy Is Affected by Interference

Bioimpedance is susceptible to respiratory movements and poor electrode contact; PPG signals are sensitive to skin pigmentation and ambient light; ultrasound Doppler relies on the operator experience—errors in aortic diameter measurement can lead to CO deviations exceeding 10% [41].

7.1.2. Inconsistent Performance Among Devices

Measurement results from different technical principles (such as bioimpedance *vs*. ultrasound) lack a unified calibration standard. Currently, no non-invasive device can fully match the accuracy of invasive pulmonary artery catheters, and the range of agreement remains wide [42].

7.1.3. Operational Dependency

Calculation of complex parameters such as LCWI and PWV requires specialized software and standardized procedures. Primary care physicians may misinterpret these parameters due to insufficient training [43].

7.2. Clinical Application Challenges

7.2.1. Insufficient Evidence Quality

Most studies were single-center, small-sample or retrospective analyses, lacking large-scale prospective trials to confirm the independent predictive value of non-invasive parameters for long-term prognosis of outcomes such as myocardial infarction and stroke [44].

7.2.2. Health Economic Considerations

Although non-invasive devices avoid complications such as infections and bleeding associated with invasive procedures, it remains unclear whether the costs of equipment purchase, maintenance, and increased examination frequency translate into sufficient health economic benefits in terms of improved patient prognosis and reduced hospital stay [45].

7.2.3. Clinical Integration Challenges

Seamlessly integrating continuous or intermittent non-invasive monitoring data into electronic medical records and forming effective clinical decision support alerts without causing "alert fatigue" remains a key challenge [44]. Clinician acceptance and training needs are another major bottleneck. Integrating new non-invasive parameters into diagnostic thinking requires time, and physicians need systematic training to correctly interpret these parameters and avoid misuse or over-reliance [46]. For unstable patients or those receiving vasoactive drugs, non-invasive blood pressure monitoring may differ significantly from invasive measurements, limiting its complete substitution in critical settings [47]. Therefore, promoting the clinical application of non-invasive hemodynamic technologies requires coordinated efforts in evidence accumulation, health economics assessment, physician education, and system integration.

8. Future Development Direction and Research Prospects

8.1. Technological Innovation: From Precise Measurement to Smart Wearables

First, equipment upgrades: combine the Internet of Things and micro-nano processing technology to develop patch-type sensors that can simultaneously monitor electrocardiogram, blood pressure, and PPG, integrating them into multi-parameter wearable devices for long-term dynamic monitoring. Develop miniaturized conductance chips to evaluate circulatory parameters such as capillary blood flow velocity. Second, artificial intelligence empowerment: use deep learning models to analyze hemodynamic time series data such as pulse waveforms and CO trends, automatically identify phenotypes, and predict treatment responses, thereby improving clinical decision-making efficiency. Finally, integrate non-invasive hemodynamic data with imaging data and omics data to construct a comprehensive "blood pressure—hemodynamics—molecular mechanism" assessment system.

8.2. Distinguishing Established Knowledge, Promising Concepts, and Future Directions

To provide clarity on the current state of evidence, we summarize the key findings of this review according to their level of validation:

Established knowledge: Hemodynamic parameters such as CO and SVR can be reliably measured non-invasively and provide valuable information on cardiovascular function [6,48]. The four hemodynamic

phenotypes (high-output, high-resistance, mixed, low-output) are well recognized in research settings and are associated with distinct pathophysiological mechanisms [49,50].

Promising concepts: Phenotype-guided antihypertensive therapy has shown potential in observational and small-scale interventional studies to improve blood pressure control and reduce adverse effects [51,52]. However, these findings require confirmation in large-scale randomized controlled trials before they can be incorporated into clinical guidelines [53].

Future research directions: Key areas for future investigation include: (1) large-scale prospective cohort studies to validate the predictive value of non-invasive parameters for cardiovascular events; (2) cost-effectiveness analyses to determine optimal use strategies across different healthcare settings; (3) integration of AI, wearable technologies, and multi-omics approaches to enable personalized hemodynamic management; and (4) development of standardized protocols and expert consensus guidelines to facilitate clinical adoption.

8.3. Key Research Focuses

Carry out large-scale prospective cohort studies to validate the independent predictive value of non-invasive parameters such as PWV and SVRI for cardiovascular events; optimize cost-benefit analysis model to clarify the optimal economic use strategy of non-invasive monitoring in different medical settings, such as community hospitals and tertiary hospitals; and formulate standardized operating procedures and expert consensus, such as the "Guidelines for the Application of Non-invasive Hemodynamic Monitoring in Hypertension" to standardize parameter interpretation and clinical pathways.

9. Conclusions

Non-invasive hemodynamic monitoring offers a promising avenue to move hypertension management from empirical blood pressure reduction toward individualized care informed by hemodynamic phenotypes. By assessing cardiac function, vascular resistance, and volume status, this technology enables more precise phenotyping. Preliminary evidence suggests that phenotype-guided treatment may improve blood pressure control and safety, but these findings stem from limited, mostly small studies and require validation in large prospective trials. Current challenges include accuracy limitations, inter-device variability, and insufficient evidence for routine clinical adoption. With advances in AI, wearables, and multi-omics, non-invasive hemodynamic monitoring holds future promise. However, the proposed shift from "blood pressure reduction" to "hemodynamic correction and target organ protection" remains a direction requiring further rigorous research and standardization before clinical integration.

Statement of the Use of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this manuscript, the authors used AI services in order to language polishing. After using this service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Author Contributions

F.B.: methodology; visualization; validation; writing–original draft; writing review and editing. X.W., Y.L., X.Q., F.Z. and X.Z.: methodology; visualization; validation; writing–original draft; writing–review and editing. All the authors read and approved the final submitted paper.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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