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Left Ventricular and Left Atrial Mechanics in Resistant Hypertension—The Role of Two-Dimensional Speckle Tracking Echocardiography

Azhar Akhmimi[1,2,3,4] | Gregory Y. H. Lip[1,4,5] | Tori Sprung[1,6] | David Oxborough[1,6] | Alena Shantsila[1,4]

[1]Liverpool Centre for Cardiovascular Science at University of Liverpool, Liverpool John Moores University and Liverpool Heart & Chest Hospital, Liverpool, UK | [2]Department of Cardiovascular Technology-Echocardiography, College of Applied Medical Sciences, King Saud bin Abdulaziz University for Health Sciences, Riyadh, Saudi Arabia | [3]King Abdullah International Medical Research Centre, Riyadh, Saudi Arabia | [4]Department of Cardiovascular and Metabolic Medicine, Institute of Life Course and Medical Sciences, University of Liverpool, Liverpool, UK | [5]Department of Clinical Medicine, Aalborg University, Aalborg East, Denmark | [6]Research Institute for Sport & Exercise Sciences, Liverpool John Moores University, Liverpool, UK

Correspondence: Alena Shantsila (s.shantsila@liverpool.ac.uk)

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ABSTRACT

Resistant hypertension (RH) is associated with a high burden of cardiovascular morbidity and mortality. Early detection of subclinical myocardial dysfunction is essential for risk stratification and timely therapeutic intervention. Speckle tracking echocardiography (STE) is an advanced imaging technique that enables sensitive assessment of myocardial mechanics and can identify subtle functional abnormalities before changes in conventional parameters such as ejection fraction become apparent. This review summarizes the current evidence on the role of STE in evaluating left ventricular (LV) and left atrial (LA) myocardial deformation in patients with uncontrolled hypertension and RH. Available data indicate that STE-derived indices, particularly LV global longitudinal strain and LA phasic strain parameters, are frequently impaired in these populations, reflecting early myocardial dysfunction. However, other components of myocardial deformation, including circumferential strain, radial strain and twist mechanics, remain insufficiently investigated in patients with RH. In addition, the absence of multimodality imaging validation and incomplete exclusion of coexisting coronary artery disease in several studies represent important limitations. Overall, STE appears to be a valuable tool for the early detection of subclinical LV and LA dysfunction in RH, and may enhance clinical risk stratification. Further well-designed prospective studies incorporating comprehensive strain analysis and multimodality imaging are required to better define its prognostic and therapeutic implications.

1 | Introduction

Resistant hypertension (RH) is commonly defined as a failure to reduce office systolic and diastolic blood pressure (BP) readings to <140 mmHg and/or <90 mmHg, respectively, in patients with hypertension despite the use of concurrent optimal doses of three or more antihypertensive medications, including a diuretic [1]. Patients with resistant and poorly controlled hypertension suffer an even higher rate of cardiovascular (CV) complications, including chronic kidney disease, myocardial infarction, heart

Abbreviations: BP, blood pressure; CAD, coronary artery disease; CV, cardiovascular; EF, ejection fraction; GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; LAScd, left atrial conduit strain; LASct, left atrial contraction strain; LASr, left atrial reservoir strain; LA, left atrial; LV, left ventricular; MRI, magnetic resonance imaging; RH, resistant hypertension; STE, speckle tracking echocardiography; SR, strain rate.

failure, stroke and death, compared to patients with controlled hypertension [2].

Cardiac structural and functional abnormalities are well recognized consequences of hypertension and contribute significantly to adverse CV outcomes [3]. Therefore, early evaluation of subtle myocardial abnormalities in patients with RH is clinically important for risk stratification and timely therapeutic intervention.

Speckle tracking echocardiography (STE), enables early detection of subclinical myocardial abnormalities by providing quantitative assessment of myocardial deformation. It is a greyscale-based imaging modality that tracks myocardial acoustic reflections ("speckles") across consecutive frames, allowing objective evaluation of both regional and global left ventricular (LV) and left atrial (LA) function [4]. STE is a useful technique to detect subclinical myocardial dysfunction in patients with hypertension [5–7] and could assist in clinical risk stratification and predicting CV morbidity and mortality, beyond conventional echocardiographic parameters [8, 9].

It is important to recognize that STE has demonstrated sensitivity in detecting subclinical myocardial dysfunction across a wide range of CV conditions beyond hypertension, including both atherosclerotic and non-atherosclerotic coronary artery diseases (CAD) [10–14]. Evidence from these populations supports the utility of strain imaging as an early marker of myocardial impairment, reinforcing its potential applicability in RH and supporting its role in broader CV risk assessment.

Given the clinical significance of RH and the increasing adoption of STE, a focused synthesis of LV and LA myocardial deformation (strain) findings in patients with uncontrolled hypertension and RH is warranted to highlight gaps in the current evidence and to inform future research. Accordingly, this review aims to comprehensively summarize the current findings of LV and LA myocardial deformation in patients with uncontrolled hypertension and RH.

2 | Literature Search Method

A focused literature search of PubMed was conducted to identify studies evaluating LV and LA myocardial strain assessed by two-dimensional STE in patients with uncontrolled hypertension and RH. Key search terms included "hypertension", "resistant hypertension", "uncontrolled hypertension", "speckle-tracking echocardiography", "left ventricular strain", "left atrial strain", and "deformation". Relevant original studies, clinical trials, and review articles published in English were considered. Additional relevant articles were identified through manual screening of reference lists.

3 | Speckle Tracking Echocardiography: An Overview

STE is a non-invasive and reliable echocardiographic technique commonly used to provide a comprehensive assessment of myocardial deformation during the cardiac cycle [4]. STE overcomes several limitations associated with conventional echocardiography. A key advantage of STE over earlier techniques used to quantify myocardial deformation, such as Tissue-Doppler imaging, is its relative independence from the angle of insonation. As a result, it enables more detailed evaluation of the various components of myocardial deformation. In addition, STE-derived strain parameters are less influenced by loading conditions compared with conventional echocardiographic measures, such as LV ejection fraction (EF). Therefore, STE is an effective technique and sensitive enough to detect subclinical myocardial dysfunction and may support therapeutic decision-making and improve prognostic assessment in many clinical conditions [15, 16]. This technique has been shown to be reliable and has been validated by comparison to sonomicrometry and tagged cardiac magnetic resonance imaging (MRI) measurements, which are the gold standard tools [17].

STE measures the strain, which is a dimensionless measurement of myocardial deformation throughout the cardiac cycle, by tracking bright myocardial speckles in greyscale echocardiographic images. These bright speckles are small dots created because of the interaction between the ultrasound beam and the myocardium. With the use of STE software, these speckles are identified and tracked from frame to frame using tracking algorithms, which can be either a sum of absolute differences or Fourier analysis, as shown in Figure 1. From these data, the magnitudes of different myocardial deformation components are automatically analyzed by the software, and as a result, strain and strain rate (SR) curves are generated [4, 15].

Strain is a measure of myocardial deformation and is defined as the fractional change in the length of a myocardial segment over a given time period, typically expressed as a percentage (%). The rate at which this deformation occurs during the cardiac cycle is referred to as SR (i.e., the deformation speed) and is often expressed in s^{-1}. Strain and SR values are negative when myocardial fibres shorten and positive when they are lengthen or thicken. In STE, strain and SR are commonly derived using the Lagrangian approach, which calculates deformation as the difference between the length after deformation and the original reference length, typically measured at end-diastole [4, 15].

3.1 | Left Ventricular Mechanics—Myofibril Architecture and Myocardial Deformation

The LV myocardium is composed of multiple myofibrils arranged in a complex helical architecture across three layers: (1) subepicardial, (2) mid-myocardial and (3) subendocardial. The subepicardial layer consists of oblique helical fibres arranged in a clockwise direction (left-handed helix), whereas the subendocardial layer comprises oblique helical fibres oriented in a counterclockwise direction (right-handed helix). Together, these layers contribute to the structure of the interventricular septum, LV free wall and cardiac apex. The mid-myocardial layer is primarily composed of horizontally oriented fibres, occupying approximately 55% of the myocardial wall thickness, and does not extend to the cardiac apex or the interventricular septum [18, 19]. The distinct orientation of myocardial fibres plays a critical role in optimizing cardiac contractile function and maintaining adequate stroke volume. Computational modelling studies have demonstrated

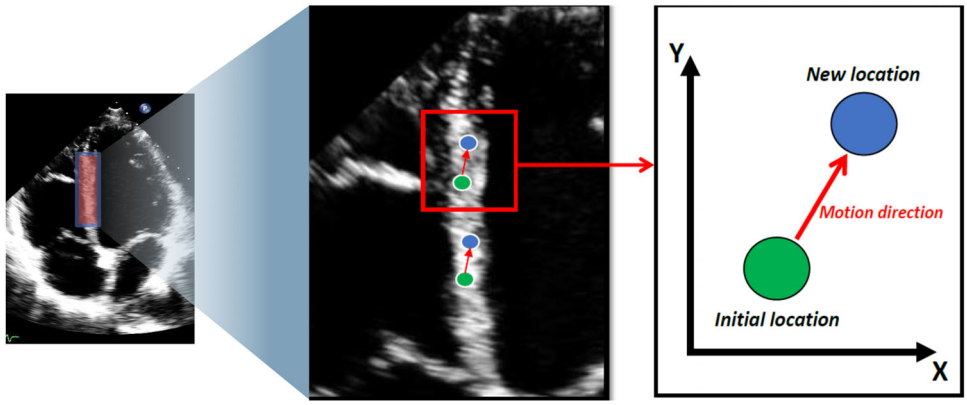

FIGURE 1 | The basic principle of speckle tracking echocardiography. The speckles are identified and tracked frame by frame in a greyscale echocardiographic image. Created in BioRender. Akhmimi, A. (2026) https://BioRender.com/qgvf7da.

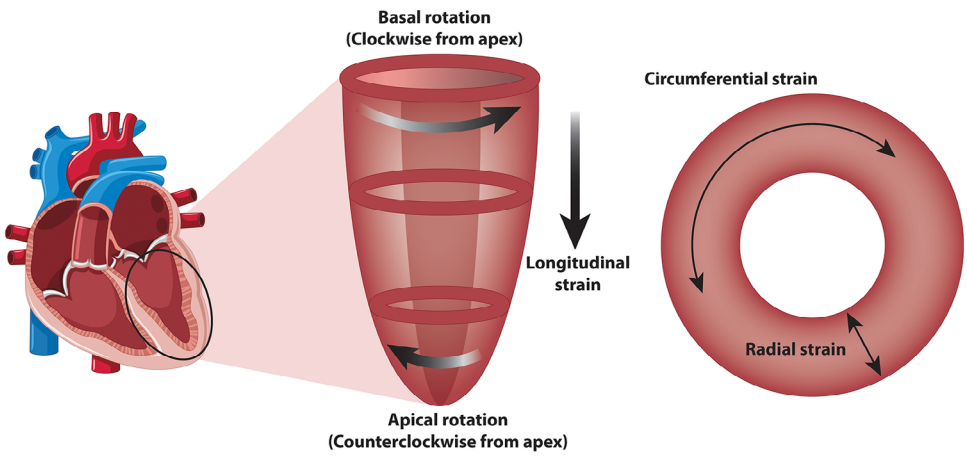

FIGURE 2 | Different components of left ventricular myocardial deformation. The image on the left shows the longitudinal and rotational deformation of the left ventricular myocardium, whilst the image on the right shows the circumferential and radial deformation of the left ventricular myocardium.

that fibre orientation significantly influences EF. For instance, when myocardial fibres are arranged solely in longitudinal or circumferential directions, EF is markedly reduced (approximately 15% and 28%, respectively), whereas the presence of obliquely oriented fibres is associated with a substantially higher EF (> 60%) [20].

During ventricular systole, the fibres of all three LV myocardial layers are shortened, leading to reductions in LV length and circumference, accompanied by wall thickening due to myocardial incompressibility. Furthermore, the LV base rotates in a clockwise direction, and the LV apex simultaneously rotates in a counterclockwise direction. During ventricular diastole, the opposite movements take place [4]. The coordinated interaction between myocardial layers results in complex patterns of myocardial deformation.

The principal components of LV myocardial deformation assessed by echocardiography include longitudinal, circumferential and radial strain (Figure 2). Longitudinal strain represents the longitudinal motion of the LV myocardial fibres from the base toward the apex. Circumferential strain denotes the shortening and lengthening of the LV circumference fibres, while radial strain depicts the thickening and thinning of the LV myocardial wall in the short axis. In addition to these components, there are complex rotational cardiac motions commonly known as LV twist and untwist. The LV twist occurs due to the opposite LV base's clockwise rotation and the LV apex's counterclockwise rotation during systole, which contributes to LV systolic function. The LV untwist occurs subsequently during diastole, with the LV base rotates in a counterclockwise direction, while the LV apex rotates in a clockwise direction, promoting LV filling in early diastole; thus, it is a representative parameter of LV diastolic function. Torsion is defined as the LV twist normalized to LV end-diastolic length [15].

Longitudinal strain is predominantly dependent on the subepicardial and the subendocardial layers. Circumferential strain

is mainly based on the mid-myocardial layer, and the radial strain is due to the thickening of the fibres in all three layers. The LV twist motion occurs due to the interaction between the longitudinal and circumferential fibres contracting at distinct timings. Mechanistically, overall cardiac contraction is primarily governed by LV deformation, which also has a substantial impact on the other cardiac chambers [16].

3.2 | Left Ventricular Myocardial Deformation in Uncontrolled and Resistant Hypertension

STE has considerably improved the understanding of cardiac mechanics and has been demonstrated to be an effective and valuable tool to detect subclinical myocardial dysfunction caused by hypertension [16]. The association between hypertension and subclinical LV myocardial dysfunction is well established in many previous studies [5–7, 21–25]. To date, a limited number of studies have examined LV myocardial deformation in patients with uncontrolled hypertension [26–32] and RH [33–35]. Data from these recent cross-sectional studies demonstrate impaired LV myocardial function, as assessed by STE, in patients with uncontrolled hypertension and RH [26–35], as summarized in Table 1.

Celic et al. [27] studied 52 patients with uncontrolled hypertension and reported significantly reduced LV global longitudinal strain (GLS), global circumferential strain (GCS), and untwisting rate, alongside preserved global radial strain (GRS) and increased LV twist compared with well-controlled hypertension and control groups. Similarly, Chen et al. [29] demonstrated a further significant reduction in LV GLS, GCS, GRS, and torsion in the uncontrolled BP group compared with the controlled BP group, suggesting more pronounced subclinical myocardial dysfunction in patients with uncontrolled hypertension. Evidence of reduced LV GLS was reported in recent studies conducted in patients with uncontrolled hypertension by Bendiab et al. [30, 32] and Ahmed et al. [31]. In the latter study, however, the reported LV GLS value did not reach statistical significance level ($p = 0.3$), which may be attributable to the small sample size or the heterogeneity of the study population [31]. It is noteworthy that patients in these studies were classified as having "uncontrolled hypertension", which may represent a distinct cohort from RH per se, as not all patients with uncontrolled hypertension meet the criteria for true RH [36].

With regard to studies specifically investigating patients with RH, Dobrowolski et al. [33] evaluated LV systolic function using STE in 155 patients with RH. They demonstrated that patients with RH, obstructive sleep apnoea, and metabolic syndrome had significantly reduced GLS. However, this finding may be influenced by the presence of obstructive sleep apnoea and metabolic syndrome, both of which are known to contribute to the development of subclinical LV dysfunction [37].

More recent studies published in 2021 [34] and 2023 [35] reported deteriorated LV GLS in patients with RH. For instance, Tadic et al. observed a significant reduction in GLS in patients with RH (−17.7±2.1%) compared to patients with well-controlled hypertension (−19.2±2.4%; $p < 0.01$) and normotensives (−20.7±2.3%; $p < 0.01$) [34]. Similarly, Souza et al. reported a significantly lower LV GLS in patients with RH (−17±3%) when compared to patients with controlled hypertension (−20±3%; $p < 0.05$) and normotensive controls (−20±2%; $p < 0.05$) [35]. However, in the latter study, the prevalence of CV risk factors, including older age, higher body mass index, diabetes and dyspnea, was significantly higher within the RH group compared to the other groups [35]. The presence of these CV risk factors might contribute to the further deterioration of LV GLS observed in the RH group.

It is important to note that most studies in RH have not incorporated advanced imaging modalities such as cardiac MRI to confirm subclinical myocardial dysfunction. The absence of multimodality imaging limits the ability to fully characterize myocardial tissue alterations, such as fibrosis, and highlights the need for future studies integrating STE with cardiac MRI-based techniques. In addition, it is worth mentioning that most of the published studies in patients with RH have primarily focused on LV GLS, while other LV strain parameters have not been sufficiently investigated. While GLS has been the most extensively studied parameter in this population, other components of myocardial deformation, including GCS, GRS, global area strain, and twist mechanics, have received limited attention. This represents a significant gap in the literature, as these multidirectional strain parameters may provide complementary insights into myocardial mechanics and the progression of hypertensive heart disease. Future studies incorporating comprehensive strain assessment are therefore warranted to better characterize the full spectrum of myocardial dysfunction in RH. The concomitant assessment of these components alongside GLS could potentially provide comprehensive information about the extent of progressive myocardial function deterioration in the course of hypertensive disease.

A previously published investigation reported, for the first time, the impact of BP optimization on LV systolic function in patients admitted with hypertension crisis, defined as a severe elevation in BP readings (≥180/120 mmHg) [26]. Using 2D-STE, the authors demonstrated reduced LV GLS, GCS, and global longitudinal systolic SR, which significantly improved following the medical treatment, although no significant changes were observed in LV-EF.

Additionally, impaired LV GLS was also observed in 30% of patients with uncontrolled hypertension and preserved LV-EF in a clinical trial conducted by Cheng et al. [28] with significant improvement after 6 months of antihypertensive therapy. However, in this study [28], the diagnosis of uncontrolled BP was not confirmed using 24-h ambulatory BP monitoring or home BP monitoring, both of which are recommended to establish persistently elevated BP levels [1]. These findings highlight the importance of early BP control in mitigating myocardial dysfunction and reducing the risk of major CV outcomes. Notably, there is a lack of data on the impact of BP optimization on LV myocardial deformation assessed by 2D-STE in patients with RH.

Collectively, these findings suggest that subclinical myocardial dysfunction becomes more pronounced as hypertension progresses and becomes more difficult to control, and may partially reverse with effective BP management. An important consideration when interpreting strain abnormalities in hypertensive populations is the potential confounding effect of co-existing

TABLE 1 | Left ventricular myocardial deformation assessment using two-dimensional speckle tracking echocardiography in uncontrolled hypertension and resistant hypertension population.

Author (year)	Study design	Patient population (sample size)	Age (mean±SD, years)	HTN duration (mean±SD, years)	Method (vendor)	Main findings
Souza et al. (2023) [35]	Cross-sectional	RH ($n = 32$) Controlled HTN ($n = 32$) Normotensive ($n = 32$)	62±8 61±12 51±12	not reported	• 2D-STE (EchoPAC, GE)	• Reduced GLS in RH compared to controlled HTN and normotensives (−17±3% vs. −20±3% and −20±2%, respectively, $p < 0.05$).
Bendiab et al. (2022) [32]	Cross-sectional	Controlled BP ($n = 228$) Uncontrolled BP ($n = 172$)	62±10 61±10	not reported	• 2D-STE (EchoPAC, GE)	• Reduced GLS in uncontrolled BP compared to controlled BP (−15.0±2.6% vs. -18.4±2.9%, $p <$ 0.0001).
Tadic et al. (2021) [34]	Cross-sectional	RH ($n = 31$) Uncontrolled HTN ($n = 58$) Well-controlled HTN ($n = 70$) Controls ($n =$ 45)	58±10 57±9 55±9 54±8	not reported	• 2D-STE (EchoPAC, GE)	• Reduced GLS in both RH and uncontrolled HTN compared to well-controlled HTN and controls (−17.7±2.1% and −18.0±2.2% vs. −-19.2±2.4% and −20.7±2.3%, respectively, $p < 0.01$), with a significant difference noted between well-controlled HTN and controls ($p < 0.01$).
Ahmed et al. (2020) [31]	Cross-sectional	Controlled HTN ($n = 23$) Uncontrolled HTN ($n = 28$)	44±14 49±10	5±5 6±5	• 2D-STE (QLAB, Philips)	• Similar GLS (−16±3% vs. −17±3%, $p = 0.3$) and lower GCS (−15±4% vs. -19±4%, $p =$ 0.003) in uncontrolled HTN compared to controlled HTN.
Bendiab et al. (2017) [30]	Cross-sectional	Controlled BP ($n = 91$) Uncontrolled BP ($n = 109$)	62±10 62±10	not reported	• 2D-STE (EchoPAC, GE)	• Reduced GLS in patients with uncontrolled BP compared to those with controlled BP (-15.9±3.1% vs. −18.2±3.0%, $p <$ 0.0001).
Chen et al. (2016) [29]	Cross-sectional	HTN1: Controlled BP ($n = 145$) HTN2: Uncontrolled BP ($n = 131$) Healthy controls ($n =$ 85)	60±12 59±12 57±8	8±1 7±1 NA	• 2D-STE (QLAB, Philips)	• Reduced GLS (−16.7±2.0% and -18.0±2.0% vs. −19.7±1.7%), GCS (-18.1±2.4% and −19.8±2.3% vs. −22.3±2.3%), GRS (-19.0±3.0% and −21.1±3.2% vs. −22.6±3.6%) and torsion (10.6±2.2° and 11.4±2.6° vs. 12.5±2.7°) in HTN2 and HTN1 groups compared to controls ($p <$ 0.001) with further reduction in all strain and torsion parameters in the HTN2 compared to HTN1 ($p < 0.001$ and $p = 0.002$, respectively).

(Continues)

CAD, which is known to impair both LV and LA mechanics even in its subclinical stages. Notably, several studies included in this review excluded patients with known CAD based on clinical history but did not systematically report the use of coronary angiography or other imaging modalities to exclude sub-clinical disease. As a result, the contribution of occult ischemic heart disease to the observed strain abnormalities cannot be fully excluded. Future investigations should incorporate appropriate screening for CAD to better isolate the independent effects of hypertension on myocardial deformation.

TABLE 1 | (Continued)

Author (year)	Study design	Patient population (sample size)	Age (mean±SD, years)	HTN duration (mean±SD, years)	Method (vendor)	Main findings
Celic et al. (2014) [27]	Cross-sectional	Well-controlled treated HTN (n = 49) Uncontrolled treated HTN (n = 52) Untreated HTN (n = 51) Controls (n = 50)	48±10 50±9 47±10 46±9	6±3 7±3 NA NA	• 2D-STE (EchoPAC, GE) • 3D-STE	• Reduced GLS in uncontrolled HTN, untreated HTN and well-controlled HTN compared to controls (−18.6±1.7%, −18.9±2.1% and −19.9±0.2% vs. −21.1±2.4%, respectively, $p < 0.05$) with further reduction in the uncontrolled HTN compared to well-controlled HTN ($p < 0.05$). • Reduced GCS in uncontrolled HTN and untreated HTN compared to controls (−21.1±2.9% and −21.6±3.0% vs. −23.4±3.1%, $p < 0.05$). • Similar GRS in all groups (p = 0.33). • Increased LV twist (13.5±3.0° vs. 11.9±2.8° and 10.5±2.6°, $p < 0.05$) and reduced LV untwisting rate (−108±38°/s vs. −129±37°/s and −136±39°/s, $p < 0.05$) in uncontrolled HTN compared to well-controlled HTN and controls, respectively.
Dobrowolski et al. (2014) [33]	Cross-sectional	Group 1: RH without OSA, MS (n = 42) Group 2: RH & OSA without MS (n = 14) Group 3: RH & MS without OSA (n = 46) Group 4: RH & OSA & MS (n = 53)	44±11 51±13 47±11 50±9	10±9 12±9 10±8 11±8	• 2D-STE (EchoPAC, GE)	• Reduced GLS in group 4 compared to groups 1 and 3 (−12.6±3.3% vs. −15.6±4.0% and −14.8±3.3%, $p < 0.05$).
Cheng et al. (2014) [28]	Clinical trial	Uncontrolled HTN (n = 182) Before and after 24 weeks of antihypertensive therapy	60±10	not reported	• 2D-STE (TomTec)	• Reduced GLS in (32%) of the patients at baseline. • Improved GLS after 24 weeks of antihypertensive therapy in the total sample (−16.8±3.8% to −18.7±3.4%, $p < 0.0001$) and in patients with reduced GLS at baseline (−13.1±2.2% to −17.0±2.9%, $p < 0.0001$).

(Continues)

TABLE 1 | (Continued)

Author (year)	Study design	Patient population (sample size)	Age (mean±SD, years)	HTN duration (mean±SD, years)	Method (vendor)	Main findings
Alam et al. (2013) [26]	Cross-sectional	Patients with HTN crisis (n = 30) Before (baseline) and after treatment (follow-up) for HTN crisis	54±13	not reported	• 2D-STE (EchoPAC, GE)	• Improved GLS (−10±4% to −12±4%, p = 0.01), global longitudinal systolic SR (−1.0±0.4s^{-1} to −1.4±0.6s^{-1}, p = 0.01) and GCS (-6.0±3.2% to -11.3±5.4%, p = 0.03) with medical treatment from baseline to follow-up.

Note: Across studies, LV GLS is consistently reduced in patients with uncontrolled and resistant hypertension, with progressive impairment observed across increasing disease severity. Evidence for other strain parameters (GCS, GRS, global area strain, twist and untwist) remains limited in patients with RH.
Abbreviations: 2D, two-dimensional; 3D, three-dimensional; BP, blood pressure; GCS, global circumferential strain; GLS, global longitudinal strain; GRS, global radial strain; HTN, hypertension; LV, left ventricular; MS, metabolic syndrome; NA, not applicable; OSA, obstructive sleep apnoea; RH, resistant hypertension; SR, strain rate; STE, speckle tracking echocardiography.

Multiple interrelated pathophysiological mechanisms contribute to impaired myocardial function in hypertension, including LV pressure overload leading to hypertrophy and remodeling, excessive myocardial and vascular collagen deposition, increased myocardial stiffness, coronary microcirculatory dysfunction, arterial stiffness, endothelial dysfunction and neurohumoral activation [23, 25, 30, 38, 39]. The interplay between increased arterial stiffness, coronary microvascular dysfunction, elevated BP, and LV hypertrophy, is closely associated with development of subendocardial ischemia and fibrosis, which in turn impairs LV longitudinal strain [25]. As subendocardial function deteriorates, the contribution of the subepicardial layer becomes relatively dominant during systole, resulting in increased LV twist. In contrast, LV untwisting during diastole is delayed due to increased myocardial stiffness and impaired relaxation, leading to abnormalities in early LV diastolic filling and elevated LV end-diastolic filling pressure [25]. Neurohumoral activation may further contribute to impaired LV untwisting in patients with hypertension [25]. In addition, elevated levels of fibrosis and collagen turnover biomarkers have been associated with impaired LV systolic longitudinal strain, altered LV twist/untwist mechanics, and both vascular and coronary microvascular dysfunction [23, 25]. These changes likely reflect the effects of excessive collagen deposition and fibrosis within the myocardium and vascular walls, which may impair myocardial contractility and the release of its stored energy during systole [25, 40], limit coronary vasodilation [41], and increase arterial stiffness [42].

In patients with hypertension, LV GLS is typically the earliest parameter to deteriorate amongst strain parameters [5, 43]. This is largely due to the vulnerability of the subendocardial fibre layer to ischemia and fibrosis, resulting in impaired longitudinal contractile function [21, 23]. In contrast, GCS and GRS are often preserved or even enhanced during early stages of hypertensive remodeling, likely reflecting compensatory mechanisms that help maintain normal LV-EF [16, 44]. Preservation of circumferential and radial function of the LV may also be related to increased LV wall thickness and cross-fibre shortening [39]. Furthermore, the mid-wall myocardial fibres are relatively less affected by early pressure overload, as their small radius of curvature results in reduced wall stress compared with longitudinal fibres, thereby preserving the LV short-axis function [44, 45]. However, a progressive deterioration of GCS and GRS may occur in the more advanced stage of hypertension as the LV function worsens, which may contribute to the progression from hypertension to heart failure [16, 44, 45]. The deterioration of GCS and GRS is related to progressive LV hypertrophy and fibrosis extending from the subendocardium toward the subepicardium [22, 44]. This progressive impairment of short-axis function may contribute to the transition from hypertensive heart disease to overt heart failure and may explain the more pronounced abnormalities observed in patients with uncontrolled hypertension.

4 | Left Atrial Mechanics

The LA has a pivotal role in maintaining LV filling and cardiac output through its mechanical function contribution. The LA function conventionally consists of three distinct phases: reservoir as LA receives oxygenated blood from the pulmonary veins during LV systole, conduit by allowing passive LV filling during early diastole, and active booster pump by augmenting LV filling in late diastole contributing to LV stroke volume approximately by 15 to 30% [46].

STE comprehensively evaluates LA phasic function by tracing the LA endocardial wall through the cardiac cycle, generating segmental and mean LA triphasic strain and SR curves (Figure 3). From these curves, the LA longitudinal myocardial strain and SR are then calculated for each of the LA phases as the difference between two measurement points on the strain curve. The derived LA longitudinal strain measures are LA reservoir strain (LASr), LA conduit strain (LAScd), and LA contraction strain (LASct) [47, 48]. LASr is measured as the difference between the strain value at peak and the end-diastolic value. It has a positive value, given that the LA wall lengthens during ventricular systole when it fills with blood from the pulmonary veins. LAScd is measured as the difference between the strain value at the onset of atrial contraction and the peak value (LASct—LASr). It has a negative value because the LA wall shortens when it passively

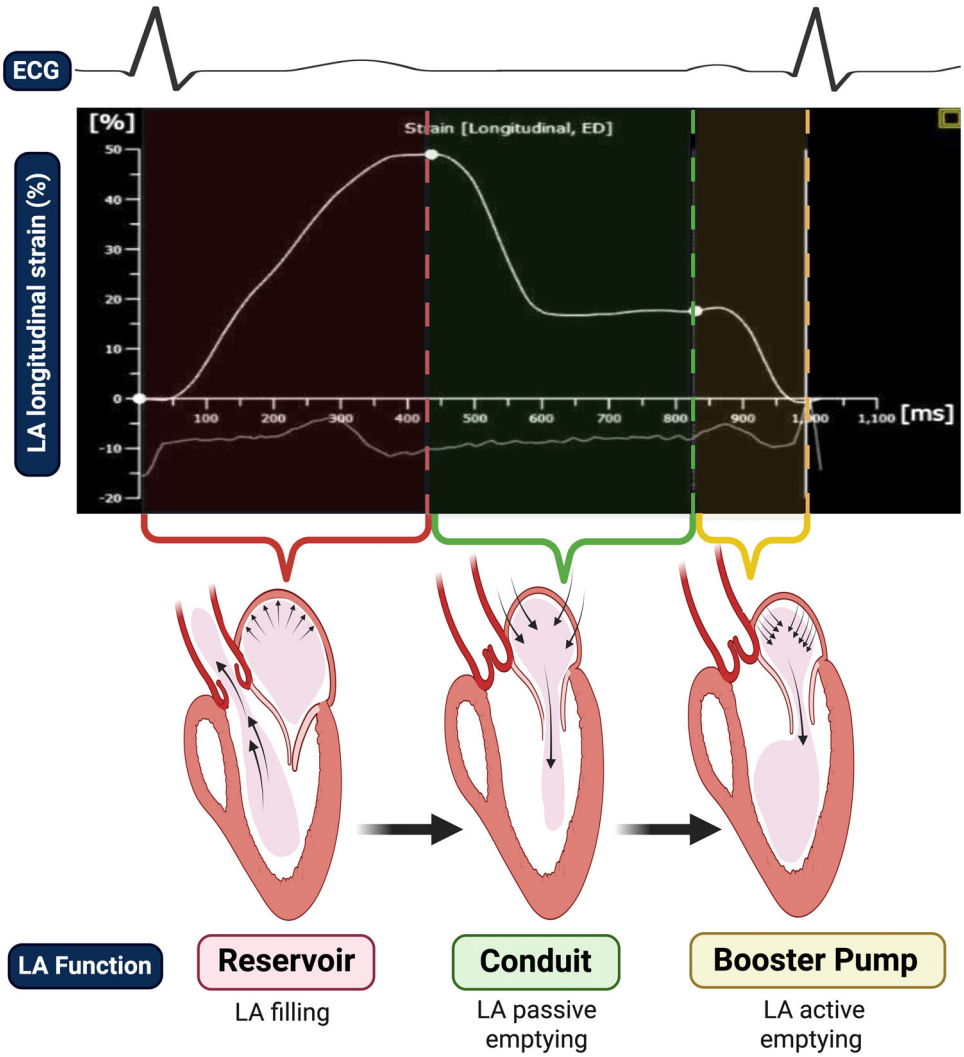

FIGURE 3 | Left atrial myocardial deformation. Left atrial phasic function in relation to the cardiac cycle (top) with the corresponding left atrial longitudinal strain curve during these phases (bottom). Vertical colors designate reservoir (red), conduit (green), and booster pump/contraction (yellow) functions, respectively. ECG, electrocardiogram; LA, left atrial. Created in BioRender. Akhmimi, A. (2026) https://BioRender.com/qgvf7da.

transfers the blood from the pulmonary veins into the LV in early diastole. LASct is measured only in patients with sinus rhythm, as the difference between the strain value at end-diastole and the value at the onset of atrial contraction. It has a negative value as the LA wall shortens when it actively contracts in late diastole to empty the blood into the LV.

The LA has an interactive and dynamic relationship with the LV; thus, LA function is not only influenced by its elastic and contractile properties but also by LV compliance and performance [46]. The LA reservoir function is primarily governed by LA compliance and relaxation, LV longitudinal systolic function via the descent movement of the LV base toward the apex, as well as right ventricular systolic pressure transmitted through the pulmonary venous return. The LA conduit function is mainly driven by LV diastolic properties (i.e., LV relaxation, compliance, and early diastolic pressure), while the LA contractile function is modulated by LA preload and intrinsic contractility, LV compliance, and end-diastolic pressure [49].

STE-derived LA strain has demonstrated considerable feasibility and reproducibility for evaluating LA function in numerous studies [50–52] and has been recently indicated as a very promising tool to implement in clinical practice by the European Association of Cardiovascular Imaging/European Heart Rhythm Association Expert Consensus [53]. Furthermore, LASr has recently emerged as a valuable parameter in the refined diagnostic algorithm recommended by the European Association of Cardiovascular Imaging to evaluate LV filling pressure in patients with heart failure and preserved EF when the traditional echocardiographic criteria give inconclusive results, with a cut-off value <18%, indicating elevated LV filling pressure [54]. Indeed, the prognostic utility of LA strain, particularly the reservoir strain, is supported by an emerging and growing body of evidence in the literature. It has been recognized as a remarkable independent predictor in various contexts, including LV diastolic dysfunction [55], LV filling pressures [56–58], atrial fibrillation development in patients with hypertension [59], and CV morbidity and mortality in the general population [8].

4.1 | Left Atrial Myocardial Deformation in Uncontrolled and Resistant Hypertension

It is well established that hypertension is associated with LA structural and functional abnormalities [3, 60]. In this context, LA strain has emerged as a promising tool, as of the adverse effects of hypertension on LA phasic function have been demonstrated using 2D-STE in patients with hypertension. Previous studies have highlighted that subclinical progressive impairment of LA strain precedes LA dilatation and other structural alterations [61, 62], and may be evident even in the absence of LV remodeling and early signs of diastolic dysfunction [63], as well as independently of LV longitudinal dysfunction [64], in patients with hypertension. Moreover, reductions in all LA phasic strain indices are more pronounced in patients with hypertension and LV hypertrophy [65, 66], as well as in those with non-dipper hypertension compared to those with dipper hypertension [67]. The presence of additional CV risk factors or comorbidities, such as diabetes [61], obesity [68], and paroxysmal atrial fibrillation [69], may further exacerbate impairment of LA strain parameters in patients with hypertension.

In the current literature, there is a paucity of studies investigating the effects of uncontrolled BP and specific hypertension phenotypes, especially RH, on STE-derived LA strain, as summarized in Table 2 [35, 70]. Chen et al. reported that patients with uncontrolled BP had reduced LA strain during reservoir, conduit and contraction phases when compared to normal controls, as well as a more pronounced reduction in the conduit strain in patients with uncontrolled BP than in those with controlled BP [70].

These findings have recently been extended to patients with RH. Souza et al., [35] reported, for the first time, that LA strain parameters during reservoir and conduit phases were significantly reduced in patients with RH, whereas contraction strain remained unchanged when compared with both normotensives and those with controlled hypertension. However, these findings may be influenced by the coexistence of multiple CV risk factors and comorbidities, including older age, diabetes, dyslipidaemia, higher body mass index and dyspnea, which may contribute additively to LA phasic functional impairment in the RH group [61, 71–74].

Overall, these findings suggest that STE may be a useful tool for detecting early abnormalities in LA function in patients with RH. Given that RH represents a high-risk phenotype within the hypertension spectrum, careful evaluation and longitudinal monitoring are warranted. Further studies are needed to better characterize LA phasic function using STE in RH and to determine its clinical relevance.

STE may be useful in assessing the effects of antihypertensive treatments on LA function in patients with hypertension. In patients with mild to moderate hypertension, 12-month treatment with angiotensin receptor blockers and beta blockers improved the LA strain during reservoir, conduit, and contraction phases [75]. These improvements occurred alongside BP reduction and were accompanied by decreases in LA volumes. The observed improvement in LA function appeared to be more closely related to BP control than to the specific class of antihypertensive therapy [75]. However, no prospective studies to date have specifically evaluated the impact of BP control on STE-derived LA strain in patients with RH.

LA dysfunction in hypertension is likely multifactorial and may be attributed to elevated LV filling pressures due to LV diastolic dysfunction, impaired LV longitudinal function, and increased LA myocardial fibrosis and stiffness [3, 64]. Abnormal LV relaxation leads to a rise in LA pressure by which the LA becomes mechanically stressed, and hence its reservoir and conduit functions reduce while its contractile function initially increases in the early stages of hypertension as a compensatory mechanism to maintain adequate LV filling in accordance with the Frank-Starling mechanism [49, 76]. However, with disease progression and persistently elevated LV filling pressures, LA contractile function deteriorates due to a gradual increase in LA stiffness and decreased compliance, ultimately impairing LA contributions to LV filling and potentially promoting the development of atrial fibrillation [77, 78].

5 | Clinical Implications and Future Research

The evidence demonstrates the importance of incorporating STE as a part of the cardiac function assessment tools in patients with RH due to its higher sensitivity in detecting subclinical myocardial dysfunction than the conventional echocardiographic parameters used nowadays. LV strain, especially GLS, is an independent prognostic indicator of CV morbidity and mortality superior to EF in patients with hypertension [79, 80]. Additionally, LA strain, particularly the reservoir strain, appears to be a sensitive prognostic marker in several diseases, including hypertension [81], and is a more robust predictor of CV events than conventional parameters of LA assessment (e.g., LA volume and EF) [82]. Therefore, the identification of subclinical myocardial dysfunction using STE-derived indices in patients with RH may assist in clinical risk stratification and may have important prognostic and therapeutic implications.

However, there are clear areas that require further investigation. There are limited studies investigating LV deformation using 2D-STE specifically in patients with RH [33–35], with a no data related to different components of LV myocardial deformation, including circumferential, radial, global area strain, twist and untwist. Moreover, there is insufficient data on LA deformation, assessed by 2D-STE, in patients with RH [35]. Patients with RH, as is well described in the literature, are at high risk of morbidity and mortality and may have significant impairments in their myocardial deformation. Because of this, these patients specifically may benefit from a more sensitive and specific risk stratification tool. Therefore, performing STE to evaluate LV and LA myocardial deformation in patients with RH may aid in identifying myocardial dysfunction earlier and more effectively.

Further research should also focus on the use of STE as a risk stratification tool for patients with RH and whether these lead to a change in clinically relevant outputs. Furthermore, the association of LV and LA mechanics with cardiac microvascular perfusion, arterial stiffness, endothelial function and autonomic function indices has not yet been fully investigated in the RH

TABLE 2 | Left atrial myocardial deformation assessment using two-dimensional speckle tracking in uncontrolled and resistant hypertension population.

Author (year)	Study design	Patient population (sample size)	Age (mean±SD, years)	Method (vendor) / measurement method	Main findings
Souza et al. (2023) [35]	Cross-sectional	RH ($n = 32$) Controlled HTN ($n = 32$) Normotensive ($n = 32$)	62±8 61±12 51±12	• 2D-STE (EchoPAC, GE) / R-R ECG gating (ventricular end-diastole)	• Reduced LASr in the RH group compared to controlled HTN and normotensives (27±7% vs. 34±6% and 33±6%, $p < 0.05$) with no differences between controlled HTN and normotensives. • Reduced LAScd in both RH and controlled HTN groups compared to normotensives (−12±5% and −16±5% vs. −19±6%, $p < 0.05$) with a further reduction in RH compared to controlled HTN ($p < 0.05$). • No differences in LASct between the RH group compared to controlled HTN and normotensives (−15±4% vs. −17±4% vs. −15±3%, $p > 0.05$). • Increased LASct in controlled HTN group compared to normotensives (−17±4% vs. −15±3%, $p < 0.0075$).
Chen et al. (2017) [70]	Cross-sectional	Optimal BP control ($n = 146$) Suboptimal BP control ($n = 133$) Normal controls ($n = 83$)	60±11 60±11 57±8	2D-STE (Qlab, Philips) / R-R ECG gating (ventricular end-diastole)	• Reduced LASr (35.1±8.3% and 36.6±7.8% vs. 50.0±10.9%), LAScd (−17.2±5.3 and −19.8±8.3% vs. −30.1±7.7%) and LASct (−17.9±4.5% and -17.6±3.9 vs. −19.9±6.4%) in both patients with suboptimal BP and optimal BP compared to normal controls, respectively ($p < 0.001$) with a further reduction in LAScd in patients with suboptimal BP control compared to patients with optimal BP ($p = 0.003$).

Abbreviations: 2D, two-dimensional; BP, blood pressure; ECG, electrocardiogram; HTN, hypertension; LAScd, left atrial conduit strain; LASct, left atrial contraction strain; LASr, left atrial reservoir strain; RH, resistant hypertension; STE, speckle tracking echocardiography.

population. This might be of value to better understand the cardiac and vascular changes, which may explain the increased risk of CV complications in those patients. Future research should also focus on the efficacy of intensified BP management on LV and LA STE-derived indices, evaluating whether they are susceptible to improvement following optimized antihypertensive therapy and whether their improvement is associated with better clinical outcomes.

It should be acknowledged that LV and LA strain parameters are not fully load-independent. In addition, inter-observer and inter-vendor variability as well as differences in image acquisition may limit direct comparison between studies [15, 16]. These considerations are important when interpreting strain abnormalities and in the design of future studies.

In addition, the limited use of multimodality imaging approaches, particularly cardiac MRI, and the inconsistent exclusion of coexisting CAD represent important methodological limitations in the current body of evidence. Addressing these gaps will be essential for improving the accuracy and interpretability of future studies.

6 | Conclusion

In summary, available evidence indicates that both LA and LV myocardial deformation are impaired in patients with uncontrolled hypertension and RH. However, current data remain limited, particularly with respect to multidirectional LV strain parameters and detailed assessment of LA mechanics in patients with RH. The lack of integration with advanced imaging modalities and incomplete exclusion of coexisting CAD represents important limitations in the existing literature. In addition, no prospective studies have yet examined the impact of intensive BP control on LV and LA mechanics in patients with RH. Further well-designed, prospective studies incorporating comprehensive strain analysis and multimodality imaging are needed to better define the pathophysiological mechanisms and clinical implications of myocardial dysfunction in this high-risk population.

Early identification of subclinical myocardial abnormalities using STE in patients with RH may play a valuable role in improving risk stratification and guiding targeted therapeutic strategies.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

No new data were generated or analyzed in support of this research.

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