Beyond Ejection Fraction: How High-Burden PVCs Reshape Left and Right Ventricular Mechanics on Advanced Echocardiography

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Abstract

Background: Frequent premature ventricular complexes (PVCs) are commonly encountered in clinical cardiology and have traditionally been regarded as benign in patients without overt structural heart disease. Accumulating clinical and imaging evidence, however, indicates that a sustained high PVC burden may exert significant adverse effects on myocardial mechanics, ultimately leading to ventricular dysfunction and potentially reversible cardiomyopathy. Importantly, these mechanical alterations may develop despite preserved left ventricular ejection fraction (LVEF), underscoring the limitations of relying solely on conventional echocardiographic indices to assess ventricular performance in this population.

Advanced echocardiographic modalities have expanded the ability to detect subtle myocardial dysfunction associated with frequent PVCs. Techniques such as tissue Doppler imaging and speckle-tracking echocardiography provide quantitative assessment of myocardial velocities, deformation, and synchrony, enabling identification of early abnormalities in systolic and diastolic function that precede changes in global pump performance. In addition to left ventricular involvement, growing evidence suggests that high-burden PVCs—particularly those originating from the right ventricular outflow tract—may also affect right ventricular mechanics, highlighting the need for comprehensive biventricular evaluation.

The aim of this review is to examine the impact of high-burden PVCs on left and right ventricular mechanics, with particular emphasis on the incremental value of advanced echocardiographic techniques beyond ejection fraction. We summarize current understanding of the pathophysiological mechanisms linking frequent ventricular ectopy to mechanical dysfunction, describe characteristic echocardiographic findings using conventional and deformation-based imaging, and discuss practical considerations for image acquisition and interpretation in patients with frequent ectopy. Special attention is given to the role of strain and strain-rate imaging in identifying subclinical dysfunction, characterizing ventricular dyssynchrony, and monitoring reverse remodeling following effective PVC suppression.

In conclusion, high-burden PVCs represent more than an isolated rhythm disturbance; they may act as a modifiable contributor to adverse ventricular mechanics and remodeling. Incorporation of advanced echocardiographic parameters into routine evaluation allows earlier recognition of PVC-related myocardial impairment, improves risk stratification, and supports timely therapeutic decision-making aimed at preventing progression to PVC-induced cardiomyopathy and preserving long-term ventricular function.

Keywords: *Ejection Fraction, PVCs Reshape , Advanced Echocardiography*

Introduction

Premature ventricular complexes (PVCs) are early depolarizations arising from ectopic ventricular myocardium that interrupt the normal sinus rhythm and are readily identified on surface electrocardiography by a premature, widened QRS complex without a preceding P wave. PVCs may occur as isolated beats or repetitive patterns such as bigeminy, trigeminy, or short non-sustained runs, and their morphology often reflects the site of origin within the ventricular myocardium. For decades, PVCs were largely considered an incidental or benign finding, particularly in individuals without known structural heart disease, leading to a conservative clinical approach focused primarily on symptom management rather than functional consequences [1].

The prevalence of PVCs varies substantially depending on the population studied and the duration of rhythm monitoring. While brief resting electrocardiograms identify PVCs in a minority of asymptomatic individuals, ambulatory Holter monitoring and longer-term wearable devices reveal ventricular ectopy in a large proportion of the general population. Epidemiological studies have demonstrated that PVC prevalence increases with advancing age and is more frequently observed in patients with cardiovascular risk factors or established heart disease, although frequent PVCs are also encountered in apparently healthy hearts [2,3].

Growing longitudinal data have challenged the traditional perception of PVCs as universally benign. Observational studies have linked frequent ventricular ectopy to adverse clinical outcomes, including increased risk of heart failure development, hospitalization, and all-cause mortality, even after adjustment for baseline cardiovascular disease. These findings suggest that PVCs may not simply reflect underlying pathology but may themselves contribute to progressive myocardial dysfunction in susceptible individuals [4,5].

Mechanistically, frequent PVCs can impair ventricular performance through several interrelated pathways. Repetitive ectopic activation outside the His–Purkinje system induces ventricular dyssynchrony, leading to inefficient myocardial contraction, abnormal regional wall stress, and adverse remodeling over time. Additional contributors include altered calcium handling, post-extrasystolic potentiation with mechanical instability, and neurohormonal activation, all of which may cumulatively depress myocardial function despite preserved resting hemodynamics [6].

The concept of premature ventricular complex–induced cardiomyopathy has emerged from clinical observations demonstrating improvement or normalization of ventricular function following effective PVC suppression, particularly with catheter ablation. Although a higher PVC burden is the most consistently identified risk factor, the threshold at which cardiomyopathy develops varies widely, indicating that burden alone does not fully explain susceptibility. Factors such as PVC origin, QRS duration, coupling interval, and baseline myocardial reserve appear to modulate the mechanical impact of ventricular ectopy [7,8].

Echocardiography remains the first-line imaging modality for evaluating patients with frequent PVCs, primarily through assessment of left ventricular ejection fraction and chamber dimensions. However, reliance on LVEF alone is increasingly recognized as insufficient for early detection of PVC-related dysfunction, as subtle abnormalities in myocardial mechanics may precede measurable reductions in global systolic performance. This limitation has driven interest in advanced echocardiographic techniques capable of capturing deformation, timing, and regional mechanics with greater sensitivity [9].

Speckle-tracking echocardiography, tissue Doppler imaging, and strain-derived indices have revealed that patients with frequent PVCs may exhibit impaired longitudinal systolic function, altered diastolic relaxation, and mechanical dyssynchrony despite normal conventional parameters. Importantly, these

abnormalities have been shown to improve after PVC suppression, supporting a causal relationship rather than incidental association. Such findings underscore the potential role of advanced echocardiography not only in diagnosis but also in longitudinal monitoring and therapeutic decision-making [10,11].

Despite these advances, important gaps remain in the standardized evaluation and risk stratification of patients with high-burden PVCs. There is no universally accepted echocardiographic framework that integrates conventional and advanced parameters to identify early dysfunction, assess biventricular involvement, and guide timing of intervention. The aim of this review is therefore to synthesize current evidence on how high-burden PVCs reshape left and right ventricular mechanics and to highlight the incremental value of advanced echocardiographic modalities beyond ejection fraction in detecting subclinical disease, refining prognosis, and monitoring functional recovery [12].

Epidemiology, PVC Burden, and Determinants of Mechanical Impact

Premature ventricular complexes are highly prevalent in both the general population and in patients undergoing cardiac evaluation, with detection rates strongly influenced by the intensity and duration of rhythm monitoring. Large population-based cohorts using ambulatory electrocardiography have demonstrated that PVCs are detected in a substantial proportion of adults, even in the absence of overt cardiovascular disease. The prevalence increases progressively with age and is higher in individuals with hypertension, coronary artery disease, or cardiomyopathic processes, although frequent PVCs are also observed in structurally normal hearts, particularly during extended monitoring [13,14].

Quantification of PVC burden has emerged as a central element in understanding their mechanical and clinical significance. PVC burden is typically expressed as the percentage of ectopic beats relative to total heartbeats over a 24-hour or longer monitoring period. Multiple studies have demonstrated a graded relationship between increasing PVC burden and declining ventricular function, with higher burdens associated with greater likelihood of left ventricular dilation, reduced systolic performance, and adverse remodeling. However, burden is not static and may vary considerably day-to-day, emphasizing the limitations of short-duration monitoring for accurate risk assessment [15,16].

Although a PVC burden threshold of approximately 10% to 15% has frequently been cited as clinically significant, this cutoff should be interpreted with caution. Several investigations have shown that some patients develop ventricular dysfunction at lower burdens, while others tolerate higher ectopic loads without measurable impairment. This interindividual variability highlights that PVC burden alone is an incomplete predictor of mechanical impact and suggests that myocardial susceptibility and PVC characteristics play important modifying roles [17,18].

The site of PVC origin is a critical determinant of mechanical consequences. PVCs originating from the right ventricular outflow tract are among the most common idiopathic forms and are often associated with preserved global systolic function; however, even these ectopic beats can induce subtle left ventricular dysfunction when frequent. In contrast, PVCs arising from the left ventricle, epicardial sites, or papillary muscles often produce broader QRS complexes and greater electrical and mechanical dyssynchrony, translating into a higher risk of ventricular dysfunction and remodeling [19,20].

QRS duration and morphology of PVCs further modulate their hemodynamic impact. Wider QRS complexes reflect slower myocardial activation and are associated with more pronounced intraventricular dyssynchrony, increased regional wall stress, and reduced mechanical efficiency. Studies have demonstrated that PVC QRS duration is an independent predictor of PVC-induced cardiomyopathy, even after adjustment for ectopic burden, underscoring the importance of qualitative ECG assessment alongside quantitative burden measurement [21,22].

Coupling interval characteristics also influence ventricular mechanics. Short-coupled PVCs may compromise diastolic filling and reduce stroke volume, while highly variable coupling intervals may reflect complex underlying mechanisms and increased arrhythmic instability. Additionally, repetitive post-extrasystolic potentiation can generate oscillations in myocardial contractility that, over time, contribute to mechanical inefficiency and adverse remodeling. These beat-to-beat mechanical effects are not captured by static measures such as ejection fraction but are increasingly recognized as

contributors to dysfunction [23,24].

Autonomic modulation represents another determinant of PVC impact on ventricular performance. Sympathetic activation can increase PVC frequency and exacerbate myocardial oxygen demand, while parasympathetic influences may alter coupling intervals and ectopic patterns. Experimental and clinical data suggest that chronic autonomic imbalance associated with frequent PVCs may promote adverse ventricular remodeling through neurohormonal pathways, further linking rhythm disturbance with mechanical disease [25,26].

Importantly, the interaction between PVC burden, electrophysiological characteristics, and myocardial substrate ultimately determines whether frequent ectopy remains benign or progresses to clinically meaningful dysfunction. Recognition of these determinants provides the foundation for imaging-based phenotyping using advanced echocardiography, which can detect early mechanical consequences of high-burden PVCs before irreversible cardiomyopathy develops. This epidemiological and mechanistic context sets the stage for understanding why conventional echocardiographic assessment may be insufficient and why advanced deformation imaging has become central to modern evaluation strategies [27,28].

Pathophysiological Links Between Frequent PVCs and Ventricular Mechanical Dysfunction

The development of ventricular mechanical dysfunction in the setting of frequent premature ventricular complexes is increasingly understood as a consequence of repetitive electrical and mechanical inefficiency rather than fixed myocardial injury alone. Unlike ischemic or inflammatory cardiomyopathies, PVC-related dysfunction often evolves gradually and may remain subclinical for prolonged periods, particularly when assessed using conventional measures such as ejection fraction. The hallmark pathophysiological feature is abnormal ventricular activation that disrupts the coordinated sequence of myocardial contraction, leading to inefficient stroke generation and altered regional loading conditions [29].

Electrical dyssynchrony represents a central mechanism linking frequent PVCs to impaired ventricular mechanics. PVCs bypass the His–Purkinje system and propagate slowly through ventricular myocardium, resulting in asynchronous contraction of opposing ventricular segments. Repetitive dyssynchrony increases regional wall stress and reduces mechanical efficiency, promoting ventricular dilation and systolic dysfunction over time. Experimental models and clinical imaging studies have demonstrated that dyssynchrony severity correlates with the degree of ventricular dysfunction and improves after effective PVC suppression, supporting a causal relationship [30,31].

Abnormal calcium handling further contributes to mechanical impairment in frequent PVCs. Each ectopic beat perturbs intracellular calcium cycling, and sustained ectopy may lead to cumulative alterations in excitation–contraction coupling. Post-extrasystolic potentiation, a transient increase in contractility following a PVC, reflects increased calcium availability but also introduces beat-to-beat variability in myocardial force generation. Chronic repetition of this pattern may exhaust myocardial reserve, impair relaxation, and promote mechanical instability that is not reflected by resting ejection fraction measurements [32,33].

Mechanical bradycardia is another underrecognized contributor to PVC-related dysfunction. Although heart rate measured by electrocardiography may appear normal, frequent non-conducted or hemodynamically ineffective PVCs reduce effective forward flow, lowering net cardiac output. This phenomenon is particularly relevant in patients with interpolated PVCs or short coupling intervals, where diastolic filling and stroke volume are compromised despite preserved average heart rate. Over time, reduced effective cardiac output may stimulate neurohormonal activation and adverse remodeling [34,35].

Neurohormonal and autonomic pathways further amplify the mechanical consequences of frequent PVCs. Chronic ectopy has been associated with heightened sympathetic activity and altered vagal tone, which can increase myocardial oxygen consumption, promote fibrosis, and exacerbate ventricular remodeling. Experimental studies suggest that autonomic afferent signaling triggered by frequent PVCs may directly contribute to cardiomyopathy development, providing a mechanistic link between

arrhythmia burden and structural remodeling [36,37].

Importantly, these pathophysiological mechanisms often operate synergistically rather than in isolation. Dyssynchrony-induced wall stress, calcium cycling abnormalities, mechanical bradycardia, and neurohormonal activation interact to create a self-perpetuating cycle of mechanical inefficiency and remodeling. The relative contribution of each mechanism likely varies among individuals, depending on PVC characteristics, baseline myocardial reserve, and comorbid conditions. This heterogeneity helps explain why some patients develop cardiomyopathy at relatively modest PVC burdens while others remain unaffected despite frequent ectopy [38,39].

The recognition that frequent PVCs can induce ventricular dysfunction through primarily functional and potentially reversible mechanisms has important implications for imaging. Because early disease is characterized by subtle abnormalities in timing, deformation, and regional mechanics rather than global pump failure, advanced echocardiographic techniques are particularly well suited to detect these changes. Understanding the underlying pathophysiology therefore provides a conceptual framework for interpreting strain, strain-rate, and dyssynchrony indices in patients with high-burden PVCs and highlights why these parameters often precede reductions in ejection fraction [40,41].

Conventional Echocardiographic Findings in Patients with High-Burden PVCs

Conventional transthoracic echocardiography remains the initial imaging modality for evaluating patients with frequent premature ventricular complexes, primarily to exclude overt structural heart disease and to establish baseline ventricular size and systolic function. Standard two-dimensional imaging provides assessment of chamber dimensions, wall thickness, and global systolic performance, with left ventricular ejection fraction (LVEF) serving as the most widely used marker of function. In many patients with high-burden PVCs, LVEF is preserved at presentation, which has historically led to reassurance and conservative follow-up despite ongoing exposure to frequent ectopy [42].

Evaluation of left ventricular volumes and geometry offers additional insight into early remodeling associated with frequent PVCs. Several studies have reported subtle increases in left ventricular end-diastolic and end-systolic volumes in patients with high PVC burden, even when LVEF remains within the normal range. These volumetric changes are thought to reflect cumulative mechanical inefficiency from repetitive dyssynchronous activation, although they may remain within guideline-defined normal limits, limiting their sensitivity for early disease detection [43,44].

Conventional Doppler echocardiography allows assessment of diastolic function through transmitral inflow velocities, deceleration time, and isovolumic relaxation time. In patients with frequent PVCs, diastolic abnormalities such as impaired relaxation or pseudonormal filling patterns have been described, particularly in those with longer duration of ectopy or evolving systolic dysfunction. However, Doppler indices are highly influenced by loading conditions and beat-to-beat variability, which are exaggerated in the presence of frequent ectopy and complicate interpretation [45].

Tissue Doppler–derived annular velocities are often included as part of standard echocardiographic protocols and provide indirect information about longitudinal function and filling pressures. Reduced mitral annular systolic velocity (S′) or early diastolic velocity (E′) may be observed in patients with frequent PVCs, suggesting early longitudinal dysfunction or impaired relaxation. Nevertheless, these measurements are angle-dependent and susceptible to translational motion, limiting their reliability as standalone markers in patients with irregular rhythms [46].

Assessment of right ventricular structure and function using conventional parameters is frequently overlooked in patients with high-burden PVCs. Measures such as tricuspid annular plane systolic excursion, right ventricular fractional area change, and tricuspid annular systolic velocity are routinely used but may remain normal despite early right ventricular involvement. This limitation is particularly relevant for PVCs originating from the right ventricular outflow tract, where subtle RV dysfunction may precede overt structural changes [47].

Intermittent post-extrasystolic potentiation represents a unique challenge for conventional echocardiographic interpretation in patients with frequent PVCs. Enhanced contractility following a PVC can transiently augment stroke volume and systolic indices, potentially masking underlying

dysfunction when measurements are averaged or obtained from post-ectopic beats. Careful beat selection and exclusion of post-extrasystolic cycles are therefore essential to avoid overestimation of ventricular performance [48].

Overall, while conventional echocardiography is indispensable for excluding established cardiomyopathy and monitoring global ventricular function, its ability to detect early PVC-related mechanical impairment is limited. The reliance on LVEF and traditional Doppler parameters may delay recognition of subclinical dysfunction and underestimate biventricular involvement. These shortcomings provide the rationale for incorporating advanced echocardiographic techniques that directly quantify myocardial deformation, synchrony, and regional mechanics, which are discussed in subsequent sections [49,50].

Tissue Doppler Imaging: Early Functional Signals in High-Burden PVCs

Tissue Doppler imaging (TDI) was one of the first echocardiographic techniques to move beyond purely anatomical assessment by enabling direct measurement of myocardial velocities. By quantifying longitudinal systolic (S′) and diastolic (E′ and A′) velocities at the mitral and tricuspid annulus, TDI provides insight into ventricular performance that may not be apparent on conventional two-dimensional imaging. In patients with high-burden premature ventricular complexes, TDI has proven particularly useful for identifying early abnormalities in longitudinal function despite preserved global systolic indices [51].

Several studies have demonstrated that patients with frequent PVCs and normal LVEF may exhibit reduced mitral annular S′ velocity, reflecting early impairment of longitudinal systolic mechanics. Because longitudinal fibers are especially sensitive to mechanical inefficiency and increased wall stress, reductions in S′ may precede declines in circumferential shortening and ejection fraction. These findings support the concept that longitudinal dysfunction represents an early marker of PVC-related myocardial impairment [52,53].

Diastolic parameters derived from TDI also provide valuable information in the setting of frequent ectopy. Reduced early diastolic annular velocity (E′) and elevated E/E′ ratios have been reported in patients with high PVC burden, suggesting impaired relaxation and increased filling pressures. Importantly, these abnormalities may be present even when transmitral Doppler indices remain within normal ranges, highlighting the incremental value of TDI in detecting subclinical diastolic dysfunction [54].

Interpretation of TDI in patients with frequent PVCs requires careful attention to beat selection. Myocardial velocities are strongly influenced by post-extrasystolic potentiation, and inclusion of post-PVC beats may artificially elevate S′ and E′ values. To minimize this effect, measurements should be obtained from sinus beats that are not immediately preceded by ectopy, and multiple cardiac cycles should be averaged when feasible. Failure to account for these factors may lead to underestimation of mechanical impairment [55].

Despite its clinical utility, TDI has important limitations that restrict its role as a comprehensive assessment tool. Measurements are angle-dependent and susceptible to translational motion, which can be exaggerated in patients with irregular rhythms and frequent ectopy. In addition, annular velocities reflect regional motion and may not accurately represent global myocardial deformation, particularly in the presence of dyssynchrony induced by PVCs [56,57].

Nevertheless, TDI occupies an important transitional role between conventional echocardiography and more advanced deformation imaging. Its widespread availability and relative ease of acquisition make it a practical screening tool for early functional abnormalities in patients with high-burden PVCs. When interpreted in conjunction with conventional parameters and clinical context, abnormal TDI findings may prompt closer surveillance or further evaluation with speckle-tracking echocardiography, which offers a more comprehensive assessment of myocardial mechanics [58,59].

In summary, tissue Doppler imaging can reveal early systolic and diastolic abnormalities associated with frequent PVCs that are not captured by ejection fraction alone. However, its technical limitations and susceptibility to rhythm-related artifacts underscore the need for more robust, angle-independent

techniques to fully characterize PVC-related mechanical dysfunction, particularly when guiding long-term management and therapeutic decision-making [60].

Speckle-Tracking Echocardiography and Left Ventricular Strain in High-Burden PVCs

Speckle-tracking echocardiography (STE) has emerged as a cornerstone modality for evaluating myocardial mechanics in patients with frequent premature ventricular complexes because it directly quantifies myocardial deformation and is relatively angle independent. By tracking natural acoustic markers within the myocardium throughout the cardiac cycle, STE provides measures of strain and strain rate that reflect intrinsic myocardial fiber shortening and lengthening. In the context of high-burden PVCs, STE is particularly valuable because it can detect subtle abnormalities in myocardial performance that precede changes in ejection fraction [61].

Left ventricular global longitudinal strain (LV-GLS) is the most extensively studied STE parameter in patients with frequent PVCs. Multiple investigations have demonstrated that LV-GLS is significantly reduced in patients with high PVC burden despite preserved LVEF, indicating early systolic dysfunction that is not apparent on conventional imaging. Longitudinal fibers are highly sensitive to increased wall stress and dyssynchronous activation, making LV-GLS a robust marker of early PVC-related mechanical impairment [62,63].

Regional strain analysis further enhances understanding of the mechanical impact of frequent PVCs. Segmental reductions in longitudinal strain often correspond to regions activated late during ectopic beats, reflecting localized dyssynchrony and inefficient contraction. This regional heterogeneity may be averaged out when assessing global parameters such as LVEF, but it is readily apparent on strain bull's-eye plots. Such patterns provide mechanistic insight into how repetitive ectopic activation translates into progressive remodeling [64].

Beyond systolic mechanics, strain-derived indices offer valuable information about diastolic function. Early diastolic strain rate has been shown to identify impaired relaxation in patients with frequent PVCs even when conventional Doppler criteria for diastolic dysfunction are not fulfilled. Because diastolic strain rate reflects active myocardial relaxation rather than passive filling alone, it may be particularly sensitive to early functional changes induced by repetitive ectopy [65,66].

Three-dimensional speckle-tracking echocardiography has further expanded the ability to characterize PVC-related dysfunction by enabling simultaneous assessment of longitudinal, circumferential, and radial deformation. Studies using three-dimensional strain have demonstrated reductions in global strain indices and increased mechanical dispersion in patients with frequent PVCs, supporting the concept that ectopy affects multiple components of ventricular mechanics. Although currently less widely available, three-dimensional strain offers a more comprehensive assessment of ventricular performance [67].

Importantly, LV strain abnormalities associated with frequent PVCs have demonstrated reversibility after effective ectopy suppression. Both medical therapy and catheter ablation have been associated with improvement in LV-GLS, often preceding recovery of LVEF. This temporal pattern reinforces the role of strain imaging as an early marker of myocardial recovery and a sensitive tool for monitoring treatment response in PVC-related cardiomyopathy [68,69].

Despite its advantages, STE requires meticulous acquisition and analysis in patients with frequent ectopy. Beat selection is critical, as post-extrasystolic potentiation can artificially enhance strain values if not excluded. In addition, vendor-related variability and differences in software algorithms can affect absolute strain measurements, emphasizing the importance of consistent methodology and longitudinal comparison within the same platform [70].

In summary, speckle-tracking echocardiography—particularly assessment of LV global and regional longitudinal strain—has fundamentally reshaped the evaluation of patients with high-burden PVCs. By uncovering subclinical systolic and diastolic dysfunction beyond ejection fraction, STE provides critical insights into disease severity, prognosis, and reversibility, and it has become an essential component of advanced echocardiographic assessment in this population [71,72].

Diastolic Mechanics and Strain-Rate Analysis in Frequent PVCs

Diastolic dysfunction represents an important yet often underrecognized component of ventricular

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impairment in patients with frequent premature ventricular complexes. While systolic abnormalities have traditionally received greater attention, repetitive ectopic activation can significantly disrupt ventricular relaxation and filling dynamics. These diastolic alterations may occur early in the disease process and contribute to symptoms such as exercise intolerance or fatigue, even when systolic function and chamber dimensions appear preserved on conventional echocardiography [73].

Traditional Doppler-based indices of diastolic function, including transmitral E and A velocities, E/A ratio, and deceleration time, are limited in patients with frequent PVCs because they are highly sensitive to heart rate variability and beat-to-beat changes in loading conditions. Frequent ectopy introduces substantial variability in diastolic filling, making it difficult to reliably classify diastolic function using standard guideline algorithms. As a result, early diastolic abnormalities related to PVC burden may be overlooked when relying solely on conventional Doppler parameters [74,75].

Strain-rate imaging derived from speckle-tracking echocardiography provides a more direct assessment of myocardial relaxation. Early diastolic strain rate (SRe) reflects the rate of myocardial lengthening during early diastole and is less dependent on loading conditions than transmitral Doppler indices. In patients with frequent PVCs, reduced SRe has been reported even when traditional Doppler measures do not meet criteria for diastolic dysfunction, highlighting its sensitivity for detecting early impairment of active relaxation [76,77].

Mechanistically, impaired diastolic strain rate in frequent PVCs is thought to result from altered calcium reuptake, residual contractile activation following ectopic beats, and cumulative effects of post-extrasystolic potentiation. These factors prolong myocardial relaxation and increase diastolic stiffness, leading to inefficient filling and elevated filling pressures during exertion. Because strain-rate imaging captures intrinsic myocardial behavior, it aligns closely with these pathophysiological processes [78].

Diastolic strain analysis also provides prognostic information in patients with high-burden PVCs. Studies have suggested that impaired diastolic strain rate may precede overt systolic dysfunction and predict progression toward PVC-induced cardiomyopathy. Furthermore, improvement in diastolic strain parameters following successful PVC suppression supports a causal link and suggests that diastolic mechanics may recover earlier than global systolic indices such as ejection fraction [79,80].

From a practical standpoint, acquisition of diastolic strain-rate measurements in patients with frequent ectopy requires careful attention to rhythm and beat selection. As with systolic strain, post-extrasystolic beats should be excluded to avoid artificial augmentation of diastolic parameters. Averaging multiple sinus beats that are not immediately preceded by PVCs improves reproducibility and enhances the clinical utility of diastolic strain assessment [81].

In summary, diastolic mechanics assessed by strain-rate imaging provide critical insight into the early functional consequences of frequent PVCs that are not adequately captured by conventional Doppler echocardiography. Incorporation of diastolic strain-rate analysis into advanced echocardiographic evaluation enhances detection of subclinical dysfunction, improves understanding of symptom mechanisms, and supports earlier identification of patients at risk for progression to PVC-induced cardiomyopathy [82].

Right Ventricular Mechanics and Strain Imaging in High-Burden PVCs

Right ventricular (RV) involvement in patients with frequent premature ventricular complexes has historically received less attention than left ventricular dysfunction, largely because conventional echocardiographic parameters often remain within normal ranges. However, growing evidence indicates that high-burden PVCs can adversely affect RV mechanics, particularly when ectopic activity originates from the right ventricular outflow tract or other RV structures. Because the RV is highly sensitive to changes in activation sequence and loading conditions, repetitive ectopic stimulation may lead to early mechanical inefficiency that is not captured by routine measures [83].

Conventional indices of RV systolic function, such as tricuspid annular plane systolic excursion, RV fractional area change, and tricuspid annular systolic velocity, are widely used because of their simplicity and reproducibility. In patients with frequent PVCs, these parameters often remain preserved despite substantial ectopic burden, leading to the assumption that RV function is unaffected. However,

these indices primarily reflect longitudinal annular motion and may fail to detect subtle regional or global deformation abnormalities associated with electrical dyssynchrony [84,85].

Speckle-tracking echocardiography has significantly advanced the assessment of RV mechanics by enabling quantification of RV global longitudinal strain and RV free-wall strain. Several studies have demonstrated that patients with high PVC burden may exhibit reduced RV strain values despite normal conventional RV parameters, indicating early subclinical systolic dysfunction. This finding is particularly relevant in idiopathic outflow tract PVCs, where repeated abnormal activation of the RV may directly impair myocardial deformation [86,87].

Regional RV strain analysis provides additional insight into the mechanical effects of frequent PVCs. Reduced strain is often most pronounced in the RV free wall, which is highly susceptible to altered activation patterns and wall stress. These regional abnormalities may contribute to inefficient RV contraction and impaired interventricular interaction, further influencing left ventricular filling and overall cardiac performance. Such complex biventricular interactions are not adequately reflected by isolated LV-focused assessment [88].

Importantly, RV strain abnormalities associated with frequent PVCs appear to be at least partially reversible. Studies evaluating patients before and after successful catheter ablation of frequent PVCs have reported significant improvement in RV strain parameters, often in parallel with improvements in LV mechanics. This reversibility supports a causal relationship between ectopic burden and RV dysfunction and underscores the value of RV strain imaging for monitoring treatment response [89,90].

Technical considerations are particularly important when assessing RV strain in patients with frequent ectopy. The thin RV free wall, complex geometry, and susceptibility to translational motion increase measurement variability, necessitating high-quality image acquisition and careful beat selection. As with LV strain, exclusion of post-extrasystolic beats and averaging of representative sinus cycles are essential to ensure reliable RV strain assessment [91].

In summary, right ventricular mechanics are an integral component of the functional impact of high-burden PVCs and should not be overlooked. Speckle-tracking–derived RV strain imaging provides a sensitive tool for detecting early RV involvement, complementing LV-focused evaluation and enabling comprehensive biventricular assessment. Incorporation of RV strain into advanced echocardiographic protocols may improve risk stratification and guide therapeutic decision-making in patients with frequent PVCs [92].

Conclusion

High-burden premature ventricular complexes should no longer be regarded as a purely electrical phenomenon or a benign incidental finding in patients with preserved ejection fraction. Accumulating evidence demonstrates that frequent PVCs can exert a sustained adverse effect on ventricular mechanics through dyssynchronous activation, altered myocardial deformation, impaired relaxation, and reduced mechanical efficiency. These changes often develop insidiously and may remain undetected when assessment is limited to conventional echocardiographic parameters, particularly left ventricular ejection fraction.

Advanced echocardiographic techniques have fundamentally reshaped the evaluation of patients with frequent PVCs by enabling direct assessment of myocardial motion and deformation. Tissue Doppler imaging provides early signals of longitudinal dysfunction, while speckle-tracking echocardiography offers robust, angle-independent quantification of systolic and diastolic mechanics. Strain and strain-rate parameters consistently identify subclinical ventricular dysfunction in patients with high PVC burden despite preserved global systolic performance, and they provide mechanistic insight into regional dysfunction and ventricular dyssynchrony. Importantly, these abnormalities are not confined to the left ventricle; right ventricular mechanics may also be affected, particularly in outflow tract PVCs, underscoring the need for comprehensive biventricular assessment.

A key clinical implication of these findings is the recognition that PVC-related myocardial dysfunction is often functional and potentially reversible. Improvement in ventricular mechanics following effective PVC suppression—most notably after catheter ablation—supports a causal relationship and highlights

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the value of advanced echocardiography for longitudinal monitoring. In many cases, strain-based parameters improve earlier than ejection fraction, offering a sensitive marker of recovery and therapeutic response.

Incorporating advanced echocardiographic modalities into routine evaluation of patients with high-burden PVCs enables earlier detection of myocardial involvement, more accurate risk stratification, and better-informed clinical decision-making. Moving beyond ejection fraction toward a mechanics-based imaging strategy provides a more complete understanding of the true functional impact of frequent PVCs and supports timely intervention aimed at preventing progression to PVC-induced cardiomyopathy and preserving long-term ventricular health.

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