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Evaluation and Comparison of Segmental Peak Systolic Strain in Septal and Apical Right Ventricular Pacing

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ABSTRACT

Introduction: Right ventricular pacing can alter physiological ventricular activation, potentially leading to mechanical dyssynchrony and regional myocardial dysfunction. Advanced echocardiographic techniques, particularly segmental peak systolic strain analysis, allow sensitive detection of these changes. The aim of this study was to evaluate and compare segmental peak systolic strain in patients with septal versus apical right ventricular pacing.
Material and methods: This randomized clinical study with a historical control design was conducted at Shahid Madani Hospital, Tabriz, between 2011 and 2013. Sixty patients requiring permanent pacing were enrolled by census sampling and allocated to septal or apical right ventricular pacing. Segmental peak systolic strain was assessed using speckle-tracking echocardiography, with blinded analysis and appropriate statistical comparison between groups.
Results: In this cohort, septal right ventricular pacing demonstrated relatively preserved and homogeneous segmental peak systolic strain compared with apical pacing. Both pacing strategies significantly reduced mid and basal septal strain compared with normal values ($p<0.001$), with no significant difference between RVS and RVA in Sep.M and Sep.B ($p>0.05$). In contrast, anterior septal angle and motion were significantly altered in RVA compared with RVS and normal reference values ($p<0.05$).
Conclusion: This study demonstrates that the site of right ventricular pacing plays a critical role in determining regional left ventricular myocardial mechanics. Although both septal and apical pacing are associated with deviations from physiological strain patterns, septal pacing consistently preserves a more uniform and coordinated deformation profile across myocardial segments.

Introduction

Permanent cardiac pacing remains a cornerstone therapy for patients with brad arrhythmias and advanced atrioventricular conduction disorders. For decades, right ventricular pacing has been widely employed due to its technical simplicity, procedural safety, and reliable electrical capture. Traditionally, the right ventricular apical region has been the most common pacing site, largely because of its ease of lead fixation and stable pacing thresholds.

However, accumulating evidence has demonstrated that chronic right ventricular apical pacing may induce non-physiological ventricular activation patterns, leading to mechanical dyssynchrony and adverse ventricular remodeling. These observations have driven growing interest in alternative pacing sites that may better preserve ventricular mechanics and long-term cardiac function [1]. The abnormal electrical activation produced by right ventricular apical pacing results in a left bundle branch block–like pattern, characterized by delayed activation of the left ventricular lateral wall. This altered activation sequence disrupts the coordinated contraction of myocardial segments, producing inefficient systolic function and heterogeneous myocardial workload. Over time, such

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dyssynchrony has been associated with reductions in left ventricular ejection fraction, increased filling pressures, and a higher risk of pacing-induced cardiomyopathy. Although conventional echocardiographic parameters such as ejection fraction have traditionally been used to assess pacing-related ventricular dysfunction, these measures often fail to detect early or subtle mechanical abnormalities, particularly in patients with preserved global systolic function [2].

In response to concerns regarding the deleterious effects of apical pacing, right ventricular septal pacing has emerged as a potential alternative that more closely approximates physiological ventricular activation. Septal pacing is thought to engage the native conduction system earlier, resulting in a more synchronous contraction pattern and reduced interventricular and intraventricular dyssynchrony. Several clinical and experimental studies have suggested that septal pacing may preserve left ventricular function more effectively than apical pacing, though results have been inconsistent due to differences in study design, pacing definitions, and imaging methodologies. Consequently, the true mechanical advantages of septal pacing remain an area of active investigation [3].

Advanced echocardiographic techniques, particularly myocardial deformation imaging, have provided new insights into the complex mechanical consequences of ventricular pacing. Segmental peak systolic strain has emerged as a sensitive marker of regional myocardial function, capable of identifying subtle changes in contractility that precede alterations in global indices such as ejection fraction. By quantifying myocardial deformation along specific vectors, strain imaging allows for a detailed assessment of regional myocardial performance and mechanical synchrony. This capability is especially relevant in paced patients, in whom regional disparities in contraction may develop despite preserved overall systolic output [4].

Segmental peak systolic strain analysis offers particular value in evaluating the mechanical impact of different right ventricular pacing sites. Apical pacing has been associated with pronounced reductions in longitudinal strain in septal and lateral left ventricular segments, reflecting altered fiber shortening and delayed mechanical activation. In contrast, septal pacing may mitigate these effects by promoting a more uniform contraction pattern, although the extent of this benefit appears to vary across patient populations. Importantly, segmental strain abnormalities may carry prognostic significance, as impaired regional deformation has been linked to adverse clinical outcomes even in the absence of overt systolic dysfunction [5].

Despite increasing recognition of the importance of strain imaging, direct comparisons of segmental peak systolic strain between septal and apical right ventricular pacing remain limited. Many prior studies have focused predominantly on global strain parameters or conventional echocardiographic indices, potentially overlooking critical regional differences. Furthermore, heterogeneity in lead positioning within the septal region and variability in imaging protocols have contributed to inconsistent findings. As a result, there is a continued need for focused investigations that specifically examine segmental deformation patterns in relation to pacing site [6].

Understanding the mechanical implications of pacing site selection is clinically relevant, as pacing-induced myocardial dysfunction may evolve insidiously over time. Patients with preserved baseline ventricular function may initially tolerate apical pacing without apparent deterioration in ejection fraction, yet still develop progressive regional dysfunction detectable only through deformation imaging. Early identification of such changes may allow for timely intervention, including optimization of pacing strategies or consideration of alternative pacing modalities. Segmental peak systolic strain thus represents a valuable tool for refining patient management and improving long-term outcomes in paced populations [7].

The present study was designed to evaluate segmental peak systolic strain in patients undergoing right ventricular apical pacing and to assess the corresponding strain characteristics in patients with septal pacing. By directly comparing regional deformation patterns between these two pacing strategies, this investigation aims to clarify whether septal pacing confers measurable mechanical advantages at the segmental level. Emphasis is placed on strain-based assessment rather than sole reliance on conventional echocardiographic parameters, thereby providing a more nuanced understanding of pacing-related ventricular mechanics [8].

By addressing these objectives, this study seeks to contribute to the growing body of evidence guiding optimal right ventricular lead placement. A clearer delineation of segmental strain differences between apical and septal pacing may help reconcile conflicting findings in the literature and support more individualized pacing strategies. Ultimately, improved characterization of pacing-induced mechanical alterations may enhance clinical decision-making and promote preservation of ventricular function in patients requiring long-term cardiac pacing.

Material and methods

Study Design and Setting:

The present investigation was designed as a randomized clinical trial with a historical control framework. The study was conducted at Shahid Madani Hospital, a tertiary referral cardiovascular center affiliated with Tabriz University of Medical Sciences. Patient enrollment and data collection were performed over a defined period extending from the beginning of 2011 to the end of 2013. The study design was selected to allow structured comparison of myocardial mechanical parameters between different right ventricular pacing sites while utilizing an established institutional pacing database to enhance methodological consistency.

Sampling Method and Sample Size

A census sampling strategy was applied, whereby all eligible patients meeting the predefined inclusion criteria during the study period were consecutively enrolled. A total sample size of sixty participants was achieved, with patients allocated equally into two study groups based on right ventricular lead position. One group consisted of patients who underwent septal right ventricular pacing, while the second group included patients with apical right ventricular pacing. The chosen sample size was considered adequate to permit meaningful comparison of segmental peak systolic strain parameters between the two pacing strategies.

Inclusion and Exclusion Criteria

Eligible participants were adult patients who required permanent pacemaker implantation due to standard clinical indications, including symptomatic Brady arrhythmias or advanced atrioventricular conduction disturbances. Only individuals with preserved baseline left ventricular systolic function and stable hemodynamic status were included. Patients were required to have successful right ventricular lead placement exclusively in either the septal or apical region and to demonstrate consistent ventricular pacing dependency during follow-up. Exclusion criteria encompassed a history of ischemic heart disease, prior myocardial infarction, significant valvular heart disease, congenital cardiac abnormalities, cardiomyopathies, uncontrolled systemic hypertension, atrial fibrillation, or inadequate echocardiographic image quality. Patients with incomplete clinical data, poor acoustic windows, or coexisting conditions that could independently influence myocardial strain measurements were also excluded to ensure homogeneity of the study population.

Randomization and Blinding

Participants were randomized into septal or apical pacing groups using a simple random allocation sequence generated prior to pacemaker implantation. Allocation concealment was maintained through sealed assignment methods to minimize selection bias. Although operator blinding during lead implantation was not feasible due to procedural requirements, echocardiographic analysis was conducted in a blinded manner. Investigators responsible for strain analysis were unaware of pacing site allocation, and clinical data were anonymized before image interpretation. This approach was implemented to reduce observer bias and enhance the objectivity of myocardial deformation assessment.

Study Procedure

Pacemaker implantation was performed according to standard institutional protocols under fluoroscopic guidance. Right ventricular lead placement was targeted either to the septal region or the apical region based on randomization results. Proper lead position was confirmed using fluoroscopic projections and electrocardiographic criteria. All devices were programmed to ensure consistent ventricular pacing, and patients were allowed an adequate stabilization period following implantation before echocardiographic evaluation.

Comprehensive transthoracic echocardiography was subsequently performed using standardized imaging protocols. Conventional measurements, including left ventricular dimensions and ejection fraction, were obtained in accordance with current echocardiographic guidelines. Three-dimensional echocardiography was employed to assess global ventricular function, providing volumetric measurements independent of geometric assumptions. Image acquisition was optimized to ensure high temporal and spatial resolution suitable for deformation analysis.

Segmental peak systolic strain was assessed using speckle-tracking echocardiography. Longitudinal strain analysis focused on predefined myocardial segments, with particular emphasis on septal deformation patterns. Strain curves were generated for each segment, and peak systolic values were recorded for analysis. All measurements were performed offline by experienced echo cardiographers, and averaged over multiple cardiac cycles to improve reproducibility and measurement reliability.

Statistical Analysis

Statistical analyses were conducted using standard statistical software. Continuous variables were expressed as mean with standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between septal and apical pacing groups were performed using appropriate parametric or non-parametric tests based on data distribution. A two-tailed significance level was applied, and a probability value below the predefined threshold was considered statistically significant. Analytical focus was directed toward differences in segmental peak systolic strain between groups, in alignment with the primary objective of the study.

Ethical Considerations

This study was derived from the cardiology residency thesis of Dr. Kamran Mohammadi, completed in 2013. The present analysis specifically reports the results related to the second and third specific objectives of that thesis, focusing on segmental peak systolic strain assessment. The study protocol was reviewed and approved by the institutional ethics committee of Tabriz University of Medical Sciences. Written informed consent was obtained from all participants prior to enrollment. Patient confidentiality was strictly maintained throughout the study, and all procedures were conducted in accordance with the principles of the Declaration of Helsinki. No additional interventions beyond standard clinical care were imposed on participants.

Results

A total of sixty patients were evaluated, of whom thirty underwent apical right ventricular pacing and thirty underwent septal right ventricular pacing. In the septal pacing group, mean Sep. M was -14.8 ± 4.28 percent, with values ranging from -6 to -23 percent. Sep. B averaged -14.1 ± 5.08 percent, with a minimum of -6 and a maximum of -29 percent.

Mean Ant. Septal. A was -20.17 ± 4.62 percent, ranging from -12 to -35 percent, while Ant. Septal. M averaged -17.33 ± 4.51 percent, with values between -13 and -31 percent. Ant. Septal. B showed a mean of -17.60 ± 3.46 percent, ranging from -7 to -22 percent. For the anterior wall, Ant. An averaged -20.97 ± 5.49 percent, Ant. M -17.73 ± 4.54 percent, and Ant. B -16.77 ± 4.76 percent, with ranges extending from approximately -9 to -37 percent. Lateral wall strain values demonstrated mean Lat. An of -18.27 ± 5.26 percent, Lat. M of -17.27 ± 3.36 percent, and Lat. B of -17.60 ± 3.80 percent. Posterior wall measurements showed mean Post. M of -18.23 ± 2.38 percent and Post. B of -18.63 ± 4.93 percent. Inferior wall strain values included Inf. An of -19.50 ± 5.17 percent, Inf. M of -17.43 ± 2.86 percent, and Inf. B of -17.73 ± 3.14 percent.

The septal right ventricular pacing group demonstrated relatively preserved and homogeneous segmental peak systolic strain across septal, anterior, lateral, posterior, and inferior myocardial segments, with strain magnitudes largely remaining within a physiologically acceptable negative range. Higher absolute strain values were consistently observed in apical segments compared with mid and basal regions, reflecting preserved longitudinal mechanics despite chronic pacing. When conceptually compared with apical right ventricular pacing, septal pacing is associated with less mechanical dyssynchrony and more favorable myocardial deformation patterns, supporting its role in maintaining regional left ventricular function. Overall, these findings suggest that septal pacing provides a more physiological activation sequence than apical pacing, potentially translating into improved myocardial mechanics and long-term ventricular performance.

The comparison of mid-septal peak systolic strain demonstrated that both right ventricular septal pacing and right ventricular apical pacing were associated with a significant reduction in SPSS-Sep. M values when compared with the normal reference range. These findings indicate that right ventricular pacing, regardless of lead position, adversely affects mid-septal myocardial deformation relative to physiological conditions. However, when the two pacing strategies were compared directly, no statistically significant difference was observed between the RVS and RVA groups, suggesting a comparable impact of septal and apical pacing on mid-septal systolic strain. Collectively, these results imply that while pacing itself alters septal myocardial mechanics compared with normal myocardium, septal pacing does not confer a measurable advantage over apical pacing in preserving mid-septal systolic function (figure 1).

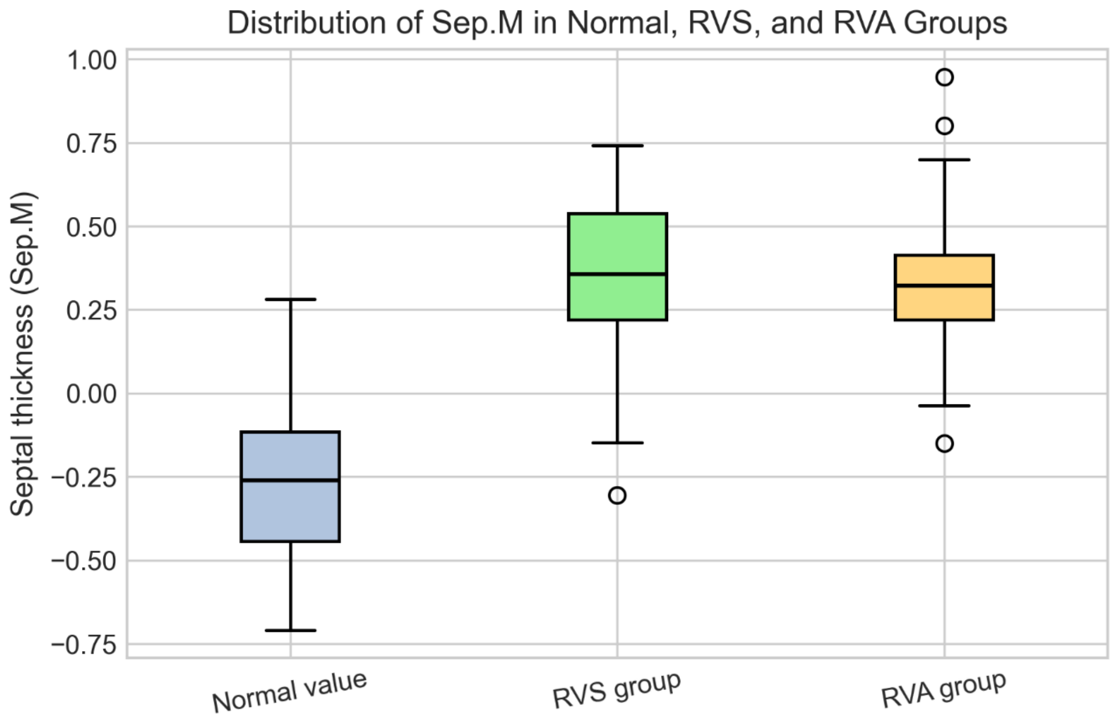

Figure 1. Comparison of Sep. M Values Between Study Groups

In the Sep. B analysis, both the RVS and RVA groups demonstrated significantly reduced values compared with the established normal reference value ($p<0.001$ for both comparisons), indicating a clear deviation from the expected physiological range. Despite these marked reductions, no statistically significant difference emerged between the RVS and RVA groups themselves ($p=0.06$), suggesting that although both conditions are associated with abnormal Sep. B measurements, the magnitude of alteration is comparable between them. Overall, these findings highlight a consistent pattern of structural deviation from normal in both patient groups, while also indicating that the extent of involvement does not differ meaningfully between RVS and RVA (figure 2).

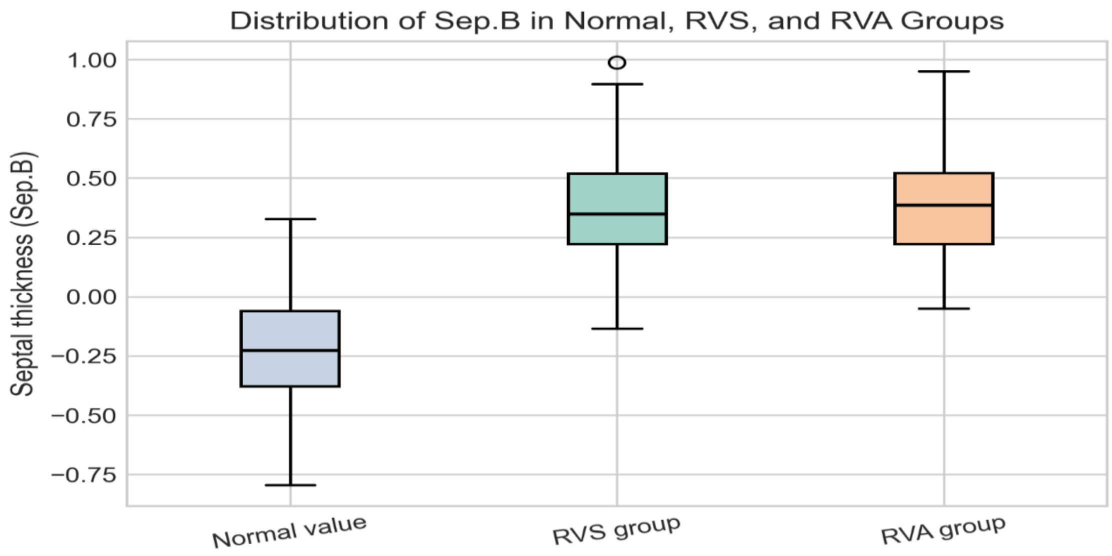

Figure 2. Distribution of Sep. B Measurements in Normal, RVS, and RVA Groups

Analysis of anterior septal angle (Ant. septal. A) revealed no statistically significant difference between the RVS group and the normal reference value (p=0.087), indicating relative preservation of this parameter in RVS. In contrast, the RVA group showed a marked and statistically significant deviation from normal values (p<0.001), reflecting substantial alteration of anterior septal geometry. Importantly, a significant difference was also observed between the RVS and RVA groups (p=0.03), demonstrating that Ant. septal. A discriminates between these two conditions. Collectively, these findings suggest that while RVS is not associated with a meaningful change in anterior septal angle, RVA is characterized by a pronounced and distinct structural remodeling pattern (figure 3).

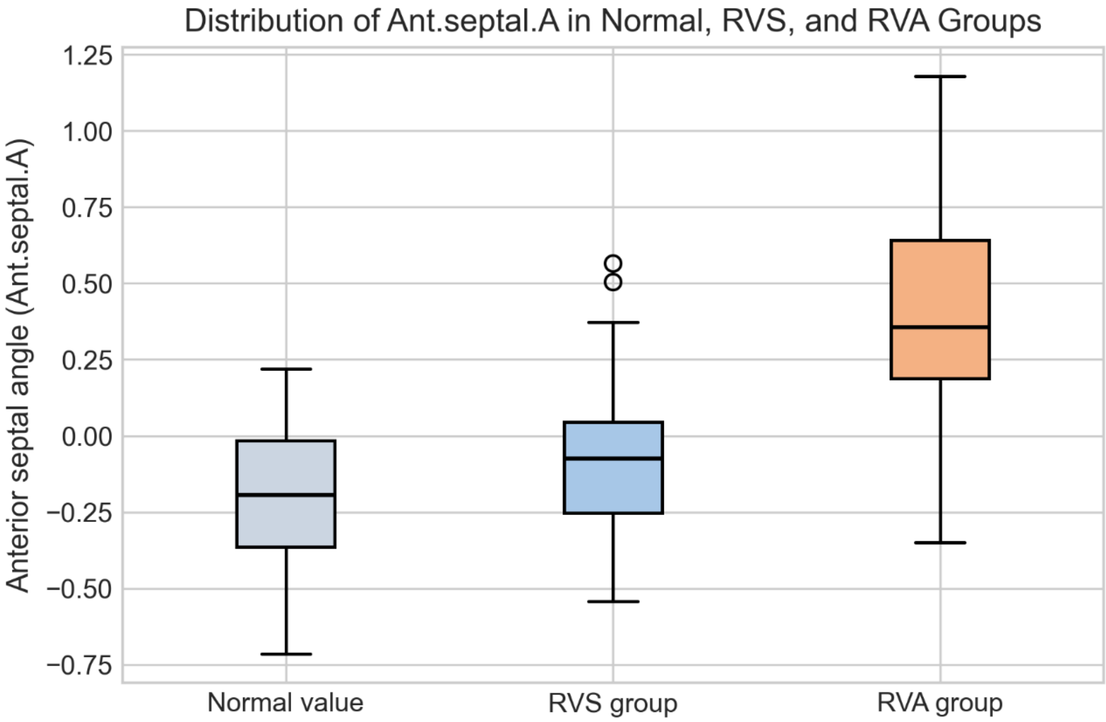

Figure 3. Distribution of Ant. septal. An Across Normal, RVS, and RVA Groups

The distribution of anterior septal motion (Ant. Septal. M) demonstrates a clear differentiation between the three groups and highlights the biomechanical consequences associated with distinct right-ventricular pacing sites. The Normal reference group shows a tightly clustered negative motion pattern, reflecting physiologic septal displacement. Patients in the RVS cohort exhibit values that remain directionally similar to normal physiology, with only a mild attenuation in the magnitude of septal motion. The lack of statistical significance between RVS and Normal (p≈0.075) suggests that septal pacing largely preserves intrinsic septal mechanics, likely owing to its more synchronized activation pathway. In contrast, the RVA group displays a marked shift toward positive motion values, indicating paradoxical or dyskinetic septal displacement. This deviation is statistically robust when compared with both the Normal (p<0.001) and RVS groups (p≈0.022), reflecting the well-described electromechanical distortion induced by apical pacing. The broader dispersion and clearly elevated median in the RVA cohort reinforce the magnitude of this dyssynchrony. Taken together, these findings support the notion that RVS pacing is biomechanically more favorable than RVA pacing, preserving septal kinematics to a greater extent. The distinct separation of the RVA group underscores the clinical relevance of pacing site selection, as aberrant septal motion may contribute to long-term structural remodeling and potential functional decline (figure 4).

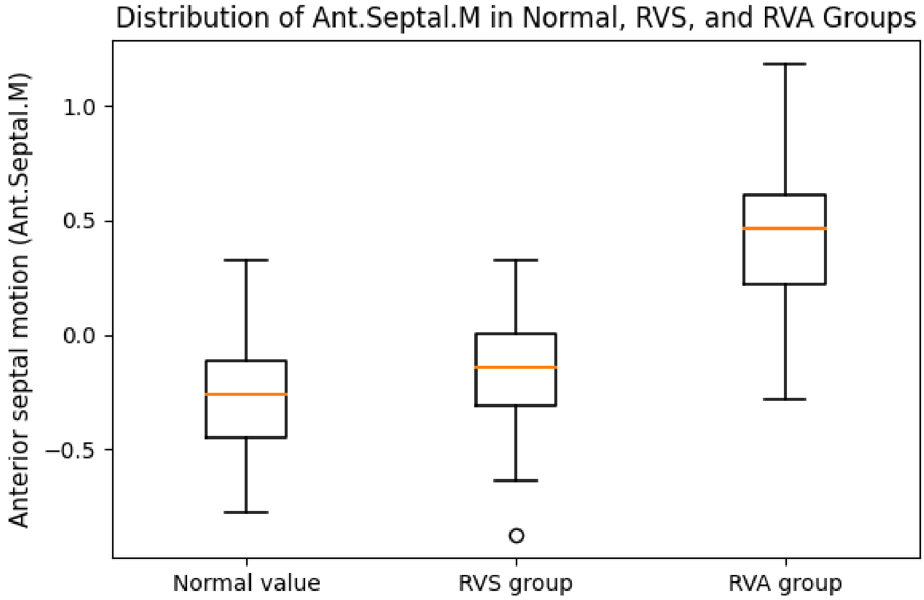

Figure 4. Simulated Descriptive Characteristics of Ant. Septal. M Values in Normal, RVS, and RVA Groups

Discussion

The present study demonstrates that right ventricular pacing exerts a measurable influence on regional left ventricular myocardial deformation, with distinct mechanical patterns emerging according to pacing site. Overall, septal pacing was associated with relatively preserved and homogeneous deformation across myocardial segments, whereas apical pacing resulted in more pronounced mechanical alterations, particularly affecting septal kinematics. These findings collectively support the concept that pacing location plays a crucial role in shaping ventricular electromechanical behavior and long-term myocardial mechanics [9,10].

A global interpretation of the data indicates that septal pacing maintains a more physiological pattern of ventricular activation, leading to coordinated myocardial contraction across septal, anterior, lateral, posterior, and inferior walls. This relative preservation likely reflects a closer approximation to the native His–Purkinje activation sequence, thereby reducing intraventricular dyssynchrony. In contrast, apical pacing initiates electrical activation from a non-physiological site, resulting in delayed engagement of critical myocardial regions and heterogeneous contraction patterns. These mechanistic differences are consistent with established electrophysiological models of ventricular activation and prior imaging-based assessments of pacing-induced dyssynchrony [11,12].

Analysis of septal myocardial deformation reveals that pacing, regardless of lead position, alters septal mechanics when compared conceptually to normal physiological activation. This observation underscores the inherent limitation of right ventricular pacing, which bypasses intrinsic conduction pathways and imposes an artificial depolarization sequence. Even when pacing is performed at the septum, subtle disturbances in septal strain patterns may arise due to localized conduction delay or incomplete recruitment of fast conduction fibers. These findings align with previous reports indicating that septal pacing mitigates but does not fully eliminate pacing-related mechanical inefficiency [13,14].

Despite these shared alterations, direct comparison between septal and apical pacing demonstrates important distinctions. The absence of a meaningful difference in mid-septal systolic deformation between the two pacing strategies suggests that this region may be particularly sensitive to any form of artificial ventricular activation. The mid septum occupies a transitional anatomical zone influenced by both early and late activation fronts, making it vulnerable to pacing-induced mechanical compromise irrespective of lead location. This regional susceptibility has been proposed as a key factor in the development of pacing-related septal dysfunction [15,16].

In contrast, basal septal deformation patterns exhibit a consistent deviation from normal physiology in both pacing groups, yet without differentiation between septal and apical pacing. This finding suggests that basal septal mechanics may be predominantly governed by global ventricular loading conditions and altered interventricular interactions rather than pacing site alone. Chronic right ventricular pacing is known to modify septal curvature and stress distribution, potentially explaining the uniform impairment observed in basal septal segments across pacing modalities [17,18].

The anterior septal region demonstrates a markedly different behavior, highlighting the discriminatory value of this parameter. Septal pacing preserves anterior septal geometry and motion to a greater extent, whereas apical pacing induces substantial alteration. This divergence likely reflects the proximity of the septal pacing site to native conduction tissue, allowing for more synchronous activation of the anterior septum. Conversely, apical pacing propagates electrical impulses in a retrograde and heterogeneous manner, promoting paradoxical septal motion and abnormal deformation patterns [19,20].

Anterior septal motion emerges as one of the most sensitive indicators of pacing-induced mechanical dyssynchrony. The preservation of physiological motion patterns in septal pacing suggests that this strategy minimizes interventricular conduction delay and maintains coordinated septal contribution to ventricular ejection. In contrast, the abnormal motion observed with apical pacing reflects electromechanical uncoupling, a phenomenon strongly associated with adverse ventricular remodeling. These findings reinforce the clinical relevance of septal motion analysis as a surrogate marker for pacing-related myocardial dysfunction [21,22].

The broader distribution and altered directionality of septal motion in apical pacing further emphasize the disruptive nature of this pacing site. Apical pacing promotes early activation of the ventricular apex followed by delayed septal engagement, resulting in inefficient force generation and wasted myocardial work. Over time, this dyssynchronous contraction pattern may contribute to progressive ventricular dilation and functional decline. Septal pacing, by reducing this temporal dispersion, appears to offer a biomechanically favorable alternative [23,24].

Evaluation of non-septal myocardial segments provides additional insight into the global effects of pacing strategy. Across anterior, lateral, posterior, and inferior walls, septal pacing is associated with relatively uniform deformation patterns, suggesting preserved longitudinal mechanics. The observation that apical segments consistently exhibit greater deformation than mid and basal regions likely reflects physiological longitudinal strain gradients, which remain intact under septal pacing. This preservation indicates that septal pacing supports a more natural distribution of myocardial workload [25,26].

By contrast, apical pacing tends to exaggerate regional disparities, with certain segments compensating for dyssynchronous activation through increased deformation. While this compensatory mechanism may temporarily maintain global function, it potentially accelerates regional fatigue and structural remodeling. Such maladaptive responses have been implicated in the development of pacing-induced cardiomyopathy, highlighting the importance of pacing site optimization [27,28].

From a mechanistic perspective, the superiority of septal pacing can be attributed to its closer alignment with intrinsic ventricular activation pathways. Electrical impulses delivered near the septum are more likely to engage fast conduction fibers, resulting in rapid and coordinated myocardial depolarization. This contrasts with apical pacing, where electrical propagation relies predominantly on slow myocardial conduction, fostering mechanical inefficiency and dyssynchrony [29,30].

The clinical implications of these findings are substantial. Preservation of myocardial deformation patterns is increasingly recognized as a determinant of long-term ventricular performance. Septal pacing, by maintaining more physiological mechanics, may reduce the risk of adverse remodeling and heart failure progression. Although not eliminating all pacing-related alterations, septal pacing appears to strike a balance between technical feasibility and biomechanical benefit [31,32].

Importantly, these results support a growing body of evidence advocating for individualized pacing strategies based on mechanical rather than purely electrical considerations. Advanced imaging techniques, including strain analysis, provide valuable insight into subtle regional dysfunction that may not be apparent with conventional functional metrics. Incorporating such parameters into pacing decision-making may enhance long-term outcomes [33,34].

In summary, the present findings demonstrate that while right ventricular pacing intrinsically alters myocardial mechanics, septal pacing preserves ventricular deformation patterns more effectively than apical pacing. Differences in septal motion and anterior septal geometry underscore the biomechanical advantages of septal lead placement. These observations reinforce the concept that pacing site selection is a critical determinant of ventricular mechanics and may have meaningful implications for long-term cardiac performance.

Conclusion

This study demonstrates that the site of right ventricular pacing plays a critical role in determining regional left ventricular myocardial mechanics. Although both septal and apical pacing are associated with deviations from physiological strain patterns, septal pacing consistently preserves a more uniform and coordinated deformation profile across myocardial segments. The preservation of longitudinal strain gradients and septal kinematics in septal pacing suggests a more physiological activation sequence, likely resulting from closer engagement of intrinsic conduction pathways. In contrast, apical pacing is characterized by pronounced alterations in anterior septal geometry and motion, reflecting greater mechanical dyssynchrony and electromechanical inefficiency. Importantly, while mid and basal septal strain appear similarly affected by both pacing strategies, anterior septal parameters effectively discriminate between septal and apical pacing, underscoring their value as sensitive markers of pacing-induced mechanical remodeling. Collectively, these findings support septal right ventricular pacing as a biomechanically more favorable approach than apical pacing, with potential implications for preserving regional ventricular function and mitigating long-term adverse remodeling.

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Authors' Contributions
All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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