

Impact of Right Ventricular Pacing Burden on Left Ventricular Deformation and Rotational Mechanics: A Cohort Study

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Abstract

Background

Permanent right ventricular pacing may impair left ventricular function through abnormal electrical activation and mechanical dyssynchrony. Early effects on myocardial mechanics in structurally normal hearts remain unclear, as ejection fraction may not detect subtle dysfunction. Speckle-tracking echocardiography allows sensitive assessment of myocardial deformation. This study evaluated the short-term impact of right ventricular pacing burden on left ventricular function and mechanics

Methods

This prospective study included 117 patients undergoing permanent pacemaker implantation for conduction abnormalities (single-chamber or dual-chamber). A pacing burden threshold of 40% was selected a priori, based on prior evidence of this level as a clinically meaningful inflection point for pacing-induced left ventricular dysfunction. Patients were stratified into $\leq 40\%$ ($n = 50$) and $> 40\%$ ($n = 67$) pacing-burden groups. Clinical and echocardiographic evaluation, including conventional and speckle-tracking imaging, was performed at baseline and 6 months.

Results

Among 117 patients (mean age 61.8 ± 13.3 years; 51.3% female) stratified by pacing burden into $\leq 40\%$ ($n = 50$) and $> 40\%$ ($n = 67$) groups, global longitudinal strain (GLS) declined significantly in the $> 40\%$ group ($18.7 \pm 4.8\%$ vs $16.3 \pm 3.7\%$, $p < 0.001$; group $\times$ time interaction $p = 0.05$) but was unchanged in the $\leq 40\%$ group. LVEF and circumferential strain declined numerically more in the $> 40\%$ group, without a significant group $\times$ time interaction. Radial strain and rotational mechanics (twist, torsion) did not change significantly in either group. Pacing burden $> 40\%$ (OR 2.32, 95% CI 1.01–5.37) and RV apical pacing site (OR 2.62, 95% CI 1.07–6.39) were independently associated with reduced GLS at 6 months (both $p < 0.05$).

Conclusion

Increased right ventricular pacing burden is associated with early impairment of left ventricular GLS, with a less certain effect on circumferential strain and no significant effect detected on radial or rotational mechanics at 6 months. Speckle-tracking echocardiography, particularly GLS, may enable earlier detection of subclinical dysfunction than ejection fraction.

Introduction

Cardiac pacemakers are an established therapy for the management of symptomatic bradyarrhythmia, including atrioventricular conduction disorders and sick sinus syndrome(1). Conventional right ventricular (RV) pacing alters the physiological sequence of ventricular activation, leading to electrical and mechanical dyssynchrony. This abnormal activation pattern may impair coordinated myocardial contraction, adversely affect ventricular hemodynamic, and alter myocardial deformation(2)

The long-term consequences of chronic RV pacing, particularly pacing-induced cardiomyopathy, are well recognized. Emerging evidence suggests that the degree of ventricular pacing burden may influence the development of ventricular dysfunction(3). However, the threshold at which RV pacing begins to adversely affect left ventricular (LV) systolic performance remains uncertain. Most available studies have primarily evaluated long-term outcomes or have included patients with underlying structural heart disease, thereby limiting the understanding of the isolated and early effects of RV pacing burden on myocardial mechanics.

Left ventricular ejection fraction (LVEF), although widely used in clinical practice, may not adequately reflect subtle changes in myocardial function during the early phase following pacemaker implantation. (4) Speckle-tracking echocardiography enables a sensitive and reproducible assessment of myocardial deformation, allowing detection of subclinical alterations in longitudinal, circumferential, and radial mechanics before overt systolic dysfunction becomes apparent.

Therefore, the present study aims to evaluate the short-term impact of varying degrees of RV pacing burden on LV systolic function and myocardial deformation parameters in patients without underlying structural heart disease.

Methodology

2.1 Patient selection

This prospective cohort study was conducted between February 2018 and June 2023 at a South Indian tertiary care center. Patients diagnosed with sick sinus syndrome or higher degrees of atrioventricular conduction block and planned for permanent pacemaker implantation were screened for eligibility and enrolled in the study (Fig. 1). Exclusion criteria included significant ischemic heart disease, dilated cardiomyopathy, valvular heart disease of more than moderate severity, congenital heart disease, genetic cardiac disorders, and prior open cardiac surgery. These criteria excluded overt structural heart disease; however, comorbidities such as hypertension, which was present in 57.3% of the cohort, were not exclusionary and may independently influence myocardial deformation (see Limitations).

A pacing burden threshold of 40% was selected a priori to stratify patients, based on prior evidence identifying this level as a clinically meaningful inflection point for subclinical left ventricular dysfunction on strain imaging (5)

A total of 117 subjects were included, of whom 24 received single-chamber ventricular (VVI) pacemakers and 93 received dual-chamber (DDD) pacemakers. Baseline clinical and echocardiographic assessments were performed at the time of admission prior to pacemaker implantation. All patients were subsequently followed up with repeat clinical evaluation and echocardiography at **6 months** after implantation to assess changes in left ventricular function and myocardial mechanics.

The study was approved by the Institutional Ethics Committee, Kasturba Medical College, Manipal (IEC No. 875/2017), and registration was obtained from the Clinical Trials Registry of India. Written informed consent was obtained from all participants prior to inclusion in the study

2.2 Echocardiographic evaluation

Echocardiographic evaluation was done at baseline and 6 months after pacemaker implantation. It was performed using 2.5–3.5 MHz transducer of the Vivid Platform Echocardiography machine from GE healthcare system.

Each subject underwent a standard baseline 2-dimensional, M-mode, conventional Doppler, Tissue Doppler Imaging, strain and strain rate by Speckle Tracking Echocardiography to assess cardiac structure and function (6)

Interventricular septal (IVS) and posterior wall (PW) thickness and left ventricular internal dimensions at end-diastole and end-systole were obtained using parasternal long-axis views with the cursor positioned perpendicular to the mitral valve tip

LV end-diastolic and end-systolic volumes and LVEF were calculated using the biplane Simpson's method of disks from apical four- and two-chamber views, in accordance with current chamber quantification guidelines. Three-dimensional echocardiography was not performed, as it was not routinely available for this cohort; biplane Simpson's method remains the guideline-recommended standard technique for LV volumetric assessment when 3D imaging is not feasible. LV function was also assessed by measuring Mitral Annular Plane Systolic Excursion (MAPSE) from M-mode obtained at lateral mitral annulus. Mitral and tricuspid inflow velocities were measured using conventional pulsed wave doppler (PWD) during early E and late A diastole with the sample volumes placed at apical four chamber view. Tissue doppler imaging (TDI) was used to obtain tissue annular velocities across cardiac cycles which included early diastolic tissue annular velocity (E'), late diastolic tissue annular velocity (A') and systolic tissue annular velocity (S'). The transmittal E wave to annular E' wave ratio was calculated to determine left ventricular (LV) diastolic function. Tei index (myocardial performance index, MPI) measures combined LV systolic and diastolic function, it was derived by acquiring tissue annular velocities across mitral annulus. The Tei index was calculated using the formula $(IVRT + IVCT) / ET$, where IVRT is isovolumic relaxation time, IVCT is isovolumic contraction time, and ET is ejection time

RV function was evaluated through the measurement Tricuspid Annular Plane Systolic Excursion (TAPSE) from M-mode obtained at lateral tricuspid annulus.

Speckle Tracking Echocardiography

Speckle-tracking analysis was applied to estimate GLS, systolic strain rate (SSR), and early and late diastolic strain rate (EDSR and LDSR) longitudinal, circumferential, and radial, measured from apical and short-axis views, respectively (Fig. 2).

Standard apical views (4C, 2C, 3C) and parasternal short-axis views at the level of the mitral valve and apex were recorded for each participant. For offline analysis, five consecutive beats in each view were digitally stored. Sector width was optimized, frame rate was set between 50 and 100 frames per second, and gain settings were optimized.

Offline analysis was performed using a commercially available speckle-tracking system on an Echo PAC workstation. LV rotational mechanics were reported as twist (degrees), torsion rate ($^{\circ}/s$), and time to peak twist; normalized torsion (twist indexed to LV end-diastolic length, $^{\circ}/cm$) was not calculated.

Reduced LV GLS was defined as an absolute GLS value $< 18\%$, based on established categorization in which GLS $< 16\%$ is considered abnormal, $> 18\%$ normal, and $16-18\%$ borderline(7)

Reliability of Echocardiographic Measurements

All echocardiographic images were acquired and analyzed by a single, dedicated, experienced echocardiographer, who was blinded to pacing group assignment ($\leq 40\%$ vs $> 40\%$) and clinical outcome at the time of analysis. To assess measurement reproducibility, a randomly selected subset of 10 patients was re-analyzed by the same observer, blinded to the prior measurements. Intra-observer agreement for LV GLS, quantified using the intraclass correlation coefficient (ICC) based on a two-way mixed-effects model with absolute agreement, was excellent (ICC = 0.900, 95% CI: 0.734–0.965)

2.3 Statistical analysis

Statistical analysis was performed using SPSS16 version. All categorical variables were represented using frequencies and percentages. Continuous variables were reported as mean and standard deviation. Within-group comparisons of means between the two time-points (baseline vs 6 months) were performed using the paired t-test, and the group ($\leq 40\%$ vs $> 40\%$) $\times$ time interaction was assessed using a two-way repeated-measures ANOVA model. A P value of < 0.05 was considered statistically significant. Binary logistic regression analysis was performed to identify predictors associated with reduced GLS. Correlation between right ventricular pacing percentage and left ventricular strain parameters (GLS, GCS, GRS) at 6-month follow-up was assessed using Spearman's rank correlation coefficient, given the non-normal distribution of pacing percentage

Results

3.1 Baseline demographics

A total of 117 patients with permanent pacemakers were included. The mean age was 61.8 ± 13.3 years, and 51.3% were female. Hypertension was present in 57.3% of patients, while diabetes mellitus was noted in 13.7%. DDD pacing was the predominant mode (79.4%), and the RV apex was the most common pacing site (80.3%). Baseline characteristics are summarized in Table 1.

Table 1
Baseline demographic, clinical, and device-related characteristics of the study population

PARAMETER	N = 117
Age	61.82 ± 13.31
Gender	57(48.7)
Male	60(51.3)
Female	
Height	159.63 ± 15.12
Weight	57+/-10.44
BMI	22.51+/-3.61
BSA	1.59+/-0.17
SBP	121.36+/-23.44
DBP	75.81+/-10.68
HR	52.44+/-17.02
QRS duration	110.9 ± 27.1
HTN	67(57.3)
DM	16(13.7)
Dyslipidaemia	4(3.4)
Type of PPI	24(20.5%)
VVI	93(79.4%)
DDD	
RV pacing site	94(80.3)
Apex	15(12.8)
Apical septum	8(6.8)
Mid septum	
<i>Data are expressed as mean ± SD or n (%). BMI: body mass index; BSA: body surface area; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; HTN: hypertension; DM: diabetes mellitus; PPI: permanent pacemaker implantation; RV: right ventricle.</i>	

The patients were also categorized based on pacing dependency. Out of 117 patients 67 were considered to be pacemakers dependent (Vp > 40%).

The $\leq 40\%$ (n = 50) and $> 40\%$ (n = 68) pacing burden groups were similar in age, sex, and comorbidities (all $p > 0.05$). However, the $> 40\%$ group had significantly longer QRS duration (117.8 ± 31.2 ms vs. 101.8 ± 17.0 ms, $p = 0.001$) and a higher proportion of VVI pacing (29.4% vs. 10.0%, $p = 0.011$). RV pacing site distribution did not differ significantly ($p = 0.097$). Baseline LVEF and strain parameters (GLS, GRS, GCS) were comparable between groups (all $p > 0.05$), indicating no pre-existing difference in myocardial deformation before pacemaker implantation (Table 2)

Table 2
Baseline characteristics of the $\leq 40\%$ and $> 40\%$ pacing burden groups.

Variable	Pacing burden $\leq 40\%$ (n = 50)	Pacing burden $> 40\%$ (n = 68)	p value
Age, years	60.3 ± 13.3	63.1 ± 13.3	0.259
Male sex, n (%)	24 (48.0)	33 (48.5)	0.995
Hypertension, n (%)	24 (48.0)	44 (64.7)	0.071
Diabetes mellitus, n (%)	7 (14.0)	10 (14.7)	0.915
Dyslipidemia, n (%)	1 (2.0)	3 (4.4)	0.479
QRS duration, ms	101.8 ± 17.0	117.8 ± 31.2	0.001
Pacing mode, n (%)			0.011
VVI (single-chamber)	5 (10.0)	20 (29.4)	
DDD (dual-chamber)	45 (90.0)	48 (70.6)	
RV pacing site, n (%)			0.097
Apex	43 (86.0)	52 (76.5)	
Apical septum	6 (12.0)	9 (13.2)	
Mid septum	1 (2.0)	7 (10.3)	
LVEF, %	63.6 ± 5.5	63.5 ± 5.2	0.935
Baseline GLS, %	19.9 ± 4.0	18.4 ± 4.8	0.170
Baseline GRS, %	39.1 ± 12.3	37.1 ± 14.6	0.448
Baseline GCS, %	19.5 ± 4.4	18.4 ± 4.2	0.110
Values are presented as mean $\pm$ SD or n (%). Between-group comparisons for continuous and binary variables used independent-samples t-tests. GLS, global longitudinal strain; GRS, global radial strain; GCS, global circumferential strain; LVEF, left ventricular ejection fraction; RV, right ventricular.			

3.2 Effect of RV pacing burden on cardiac dimensions

Patients were stratified based on ventricular pacing percentage into $\leq 40\%$ pacing (n = 50) and $> 40\%$ pacing groups (n = 67). (Table 3)

Table 3

Changes in cardiac dimensions over 6 months according to right ventricular pacing percentage

$\leq 40\%$ (n = 50)				$> 40\%$ (n = 67)			
Parameter	Baseline	6 months	P value	Baseline	6 months	P value	Repeated measure ANOVA p value
Cardiac Dimensions							
EDD	47.32 $\pm$ 5.62	45.94 $\pm$ 4.18	0.06	47.80 $\pm$ 5.25	47.16 $\pm$ 4.81	0.259	0.71
ESD	30.00 $\pm$ 4.07	28.78 $\pm$ 2.95	0.023	30.65 $\pm$ 3.99	30.71 $\pm$ 4.04	0.883	0.51
ALEF	63.56 $\pm$ 5.54	62.40 $\pm$ 4.32	0.079	63.47 $\pm$ 5.21	61.53 $\pm$ 4.43	0.003	0.39
FS	36.00 $\pm$ 3.07	35.52 $\pm$ 2.81	0.285	35.94 $\pm$ 5.74	34.71 $\pm$ 5.15	0.011	0.27
EDV	65.66 $\pm$ 16.98	65.38 $\pm$ 16.05	0.882	71.38 $\pm$ 20.07	68.37 $\pm$ 17.73	0.081	0.29
ESV	24.00 $\pm$ 7.97	24.64 $\pm$ 7.80	0.496	26.21 $\pm$ 9.43	27.40 $\pm$ 11.30	0.319	0.73
LA (AP)	31.84 $\pm$ 4.70	31.50 $\pm$ 4.07	0.464	31.06 $\pm$ 5.38	31.46 $\pm$ 3.64	0.443	0.31
LA IS	50.40 $\pm$ 5.84	49.16 $\pm$ 4.96	0.035	49.14 $\pm$ 7.45	49.16 $\pm$ 6.99	0.979	0.13
RA IS	50.02 $\pm$ 5.42	48.82 $\pm$ 4.85	0.021	49.46 $\pm$ 6.83	49.43 $\pm$ 6.31	0.951	0.10
Data are presented as mean $\pm$ standard deviation. P value within each pacing group represents comparison between baseline and 6-month follow-up. Repeated measures ANOVA P value represents interaction between time and pacing group. EDD – left ventricular end-diastolic diameter; ESD – left ventricular end-systolic diameter; LVEF – left ventricular ejection fraction; FS – fractional shortening; EDV – end-diastolic volume; ESV – end-systolic volume; LA (AP) – left atrial anteroposterior diameter; LA IS – left atrial inferior–superior diameter; RA IS – right atrial inferior–superior diameter.							

In the $\leq 40\%$ pacing group, there was a modest but statistically significant reduction in left ventricular end-systolic diameter (ESD) over 6 months (30.00 $\pm$ 4.07 mm vs 28.78 $\pm$ 2.95 mm, p = 0.023). Left atrial inferior–superior (LA IS) and right atrial inferior–superior (RA IS) dimensions also showed small but significant reductions (p = 0.035 and p = 0.021 respectively). Other parameters including LV end-diastolic diameter (EDD), LV ejection fraction (LVEF), fractional shortening (FS), end-diastolic volume (EDV), end-systolic volume (ESV), and LA anteroposterior diameter did not show significant changes over time.

In the > 40% pacing group, LVEF and fractional shortening showed a significant decline at 6 months compared to baseline (LVEF: $63.47 \pm 5.21\%$ vs $61.53 \pm 4.43\%$, $p = 0.003$; FS: $35.94 \pm 5.74\%$ vs $34.71 \pm 5.15\%$, $p = 0.011$). However, no significant changes were observed in LV dimensions, volumes, or atrial dimensions. The corresponding group×time interaction terms for LVEF (repeated-measures ANOVA $p = 0.39$) and fractional shortening ($p = 0.27$) were not statistically significant (Table 3), so these within-group declines should not be interpreted as a significantly greater temporal decline in the > 40% group compared with the $\leq 40\%$ group.

3.3 Changes in diastolic function parameters according to pacing burden

In the $\leq 40\%$ pacing group, mitral E velocity showed a significant decline at 6 months compared with baseline (0.71 ± 0.23 m/s vs 0.63 ± 0.18 m/s, $p = 0.028$). Aortic peak velocity also decreased significantly over time ($p = 0.019$). Tissue Doppler parameters demonstrated a reduction in lateral wall E' velocity ($p = 0.006$), while A' at the interventricular septum and lateral wall showed small but significant increases ($p = 0.026$ and $p = 0.030$ respectively). Other parameters, including MV A velocity, tricuspid velocity, pulmonary velocity, S' velocities, and E/E' ratio, did not change significantly.(Table 4)

Table 4

Comparison of diastolic function parameters at baseline and 6-month follow-up according to right ventricular pacing burden

$\leq 40\%$ (n = 50)				$> 40\%$ (n = 67)			
Parameter	Baseline	6 months	P value	Baseline	6 months	P value	Repeated measure ANOVA p value
Diastolic function parameters							
MV E(m/s)	0.71 $\pm$ 0.23	0.63 $\pm$ 0.18	0.028	0.83 $\pm$ 0.33	0.68 $\pm$ 0.25	< 0.001	0.16
MV A(m/s)	0.58 $\pm$ 0.31	0.62 $\pm$ 0.27	0.429	0.62 $\pm$ 0.38	0.67 $\pm$ 0.29	0.258	0.68
Aortic velocity(m/s)	1.35 $\pm$ 0.27	1.25 $\pm$ 0.25	0.019	1.44 $\pm$ 0.39	1.34 $\pm$ 0.29	0.002	0.80
Tricuspid velocity(m/s)	0.49 $\pm$ 0.11	0.48 $\pm$ 0.08	0.472	0.53 $\pm$ 0.15	0.50 $\pm$ 0.11	0.172	0.63
Pulmonary velocity(m/s)	0.83 $\pm$ 0.16	0.78 $\pm$ 0.16	0.064	0.84 $\pm$ 0.22	0.77 $\pm$ 0.13	0.019	0.69
E' IVS(m/s)	0.07 $\pm$ 0.02	0.06 $\pm$ 0.02	0.070	0.07 $\pm$ 0.03	0.06 $\pm$ 0.02	0.017	0.37
A' IVS(m/s)	0.07 $\pm$ 0.03	0.07 $\pm$ 0.03	0.026	0.06 $\pm$ 0.04	0.07 $\pm$ 0.03	0.226	0.70
S' IVS(m/s)	0.08 $\pm$ 0.09	0.06 $\pm$ 0.01	0.211	0.06 $\pm$ 0.02	0.06 $\pm$ 0.01	0.083	0.35
E' LW(m/s)	0.09 $\pm$ 0.03	0.08 $\pm$ 0.02	0.006	0.09 $\pm$ 0.04	0.07 $\pm$ 0.03	0.003	0.37
A' LW(m/s)	0.07 $\pm$ 0.03	0.08 $\pm$ 0.03	0.030	0.06 $\pm$ 0.04	0.07 $\pm$ 0.03	0.071	0.87
S' LW(m/s)	0.08 $\pm$ 0.02	0.08 $\pm$ 0.02	0.072	0.07 $\pm$ 0.02	0.07 $\pm$ 0.02	0.040	0.66
LV MPI	0.39 $\pm$ 0.05	0.33 $\pm$ 0.04	0.087	0.59 $\pm$ 0.10	0.69 $\pm$ 0.09	0.042	0.65

Data are expressed as mean $\pm$ standard deviation. P value within each pacing group represents comparison between baseline and 6-month follow-up. Repeated measures ANOVA P value represents the interaction between time and pacing burden groups ($\leq 40\%$ vs $> 40\%$). MV E – mitral early diastolic inflow velocity; MV A – mitral late diastolic inflow velocity; IVS – interventricular septum; LW – left ventricular lateral wall; E' – early diastolic tissue Doppler velocity; A' – late diastolic tissue Doppler velocity; S' – systolic tissue Doppler velocity; E/E' – ratio of mitral inflow E velocity to tissue Doppler E' velocity. MPI – Myocardial performance index

≤ 40% (n = 50)				> 40% (n = 67)			
E/E'	11.11 ± 4.02	11.09 ± 4.97	0.973	11.83 ± 5.44	11.97 ± 5.63	0.838	0.87
<i>Data are expressed as mean ± standard deviation. P value within each pacing group represents comparison between baseline and 6-month follow-up. Repeated measures ANOVA P value represents the interaction between time and pacing burden groups (≤ 40% vs > 40%). MV E – mitral early diastolic inflow velocity; MV A – mitral late diastolic inflow velocity; IVS – interventricular septum; LW – left ventricular lateral wall; E' – early diastolic tissue Doppler velocity; A' – late diastolic tissue Doppler velocity; S' – systolic tissue Doppler velocity; E/E' – ratio of mitral inflow E velocity to tissue Doppler E' velocity. MPI – Myocardial performance index</i>							

In the > 40% pacing group, mitral E velocity and aortic peak velocity showed significant reductions at follow-up ($p < 0.001$ and $p = 0.002$ respectively). Pulmonary velocity also showed a modest decline ($p = 0.019$). Tissue Doppler parameters demonstrated reductions in septal E' and lateral wall E' velocities ($p = 0.017$ and $p = 0.003$ respectively), while lateral wall S' showed a small but significant change ($p = 0.040$). Other parameters remained unchanged.

However, repeated measures ANOVA did not demonstrate a significant interaction between pacing burden and temporal changes in diastolic parameters, suggesting that the pattern of change over time was not significantly different between the two pacing groups.

3.4 Effect of RV pacing burden on Speckle tracking parameters

In the ≤ 40% pacing group, LV global longitudinal strain and longitudinal systolic strain rate remained unchanged over 6 months. However, longitudinal early diastolic strain rate decreased, while longitudinal and circumferential late diastolic strain rates increased significantly. Radial deformation parameters showed no significant changes except for an increase in radial late diastolic strain rate.(Table 5)

Table 5

Comparison of left ventricular myocardial deformation parameters at baseline and 6-month follow-up according to right ventricular pacing burden

≤ 40% Pacing (n = 50)				> 40% Pacing (n = 67)			
Parameter	Baseline	6 months	P value	Baseline	6 months	P value	RM-ANOVA P value
LV longitudinal deformation							
LV GLS (%)	19.91 ± 4.01	19.09 ± 3.07	0.184	18.74 ± 4.81	16.27 ± 3.74	< 0.001	0.05
LV SSR	1.27 ± 0.24	1.21 ± 0.21	0.075	1.22 ± 0.23	1.09 ± 0.23	< 0.001	0.17
LV EDSR	1.48 ± 0.44	1.36 ± 0.39	0.024	1.56 ± 0.50	1.31 ± 0.34	< 0.001	0.13
LV LDSR	0.99 ± 0.43	1.20 ± 0.34	0.002	0.97 ± 0.47	1.07 ± 0.39	0.155	< 0.001
LV radial deformation							
Radial strain (%)	39.12 ± 12.28	36.91 ± 11.92	0.372	37.14 ± 14.61	33.75 ± 12.84	0.172	0.74
Radial SSR	2.69 ± 0.88	2.76 ± 0.76	0.601	2.56 ± 0.93	2.57 ± 0.78	0.951	0.70
Radial EDSR	2.54 ± 0.85	2.61 ± 0.89	0.658	2.47 ± 1.06	2.42 ± 0.80	0.665	0.54
Radial LDSR	1.51 ± 0.73	2.04 ± 0.69	< 0.001	1.71 ± 1.02	1.92 ± 0.81	0.080	0.71
LV circumferential deformation							
Circumferential strain (%)	19.53 ± 4.36	18.73 ± 3.36	0.239	18.40 ± 4.14	16.13 ± 3.52	0.033	0.59
Circumferential SSR	1.78 ± 0.42	1.69 ± 0.34	0.218	1.63 ± 0.36	1.65 ± 0.31	0.802	0.26
Circumferential EDSR	1.82 ± 0.40	1.72 ± 0.37	0.170	1.70 ± 0.45	1.72 ± 0.38	0.779	0.25
Circumferential LDSR	1.01 ± 0.45	1.30 ± 0.36	< 0.001	0.97 ± 0.45	1.15 ± 0.42	0.008	0.27
Data are presented as mean ± standard deviation. P value within each pacing group represents comparison between baseline and 6-month follow-up. RM-ANOVA P value represents the interaction between time and pacing burden groups (≤ 40% vs > 40%). GLS – global longitudinal strain; SSR – systolic strain rate; EDSR – early diastolic strain rate; LDSR – late diastolic strain rate; LV – left ventricle.							

In the > 40% pacing group, LV global longitudinal strain, longitudinal systolic strain rate, and longitudinal early diastolic strain rate showed significant reductions at 6 months. Circumferential strain also decreased modestly, while circumferential late diastolic strain rate increased. Radial deformation parameters did not demonstrate significant changes. Of the parameters above, only the group×time interaction terms for GLS ($p = 0.05$) and longitudinal late diastolic strain rate ($p < 0.001$) reached or approached significance; the interaction terms for circumferential strain ($p = 0.59$), longitudinal systolic strain rate ($p = 0.17$), and longitudinal early diastolic strain rate ($p = 0.13$) were not significant (Table 5), so these should be interpreted as within-group trends rather than confirmed burden-dependent effects.

Repeated measures ANOVA demonstrated a significant interaction between pacing burden and time only for LV longitudinal late diastolic strain rate

3.5 Effect of pacing percentage on left ventricular twist mechanics

LV twist mechanics were compared between patients with low pacing percentage ($\leq 40\%$) and high pacing percentage ($> 40\%$) at baseline and 6 months (Table 6). In the $\leq 40\%$ pacing group, twist ($14.10 \pm 7.21^\circ$ vs. $13.60 \pm 6.87^\circ$), torsion rate (2.24 ± 1.17 vs. $2.22 \pm 1.06^\circ/\text{s}$), and peak twist (350 ± 73.6 vs. 374 ± 86.4 ms) did not change significantly over 6 months ($p = 0.780$, $p = 0.976$, and $p = 0.124$, respectively). Likewise, in the $> 40\%$ pacing group, no significant changes were observed in twist ($11.90 \pm 8.91^\circ$ vs. $12.30 \pm 8.46^\circ$), torsion rate (1.88 ± 1.41 vs. $1.96 \pm 1.37^\circ/\text{s}$), or peak twist (371 ± 89.5 vs. 378 ± 100.0 ms) at 6 months ($p = 0.790$, $p = 0.687$, and $p = 0.484$, respectively). RM-ANOVA showed no significant between-group interaction over time for twist, torsion rate, or peak twist ($p = 0.704$, $p = 0.797$, and $p = 0.539$, respectively), suggesting that pacing percentage did not differentially affect LV twist mechanics over the follow-up period.

Table 6
Comparison of left ventricular rotational mechanics at baseline and 6-month follow-up according to right ventricular pacing burden

$\leq 40\%$				$> 40\%$			
Parameter	Baseline	6 months	P	Baseline	6 months	P	RM-ANOVA
Twist	14.10 ± 7.21	13.60 ± 6.87	0.780	11.90 ± 8.91	12.30 ± 8.46	0.790	0.704
Torsion rate	2.24 ± 1.17	2.22 ± 1.06	0.976	1.88 ± 1.41	1.96 ± 1.37	0.687	0.797
Peak twist	350 ± 73.6	374 ± 86.4	0.124	371 ± 89.5	378 ± 100.0	0.484	0.539
Data are presented as mean $\pm$ standard deviation. P values represent within-group comparisons between baseline and 6-month follow-up (paired t-test). RM-ANOVA P value represents the interaction between time and pacing burden groups ($\leq 40\%$ vs $> 40\%$).							

3.6 Correlation between ventricular pacing percentage and left ventricular strain

Spearman correlation analysis demonstrated a significant negative correlation between ventricular pacing percentage and both GLS ($\rho = -0.421$, $p < 0.001$) and GCS ($\rho = -0.404$, $p < 0.001$) at 6-month follow-up, indicating worsening myocardial deformation with increasing pacing burden. However, no significant correlation was observed between pacing percentage and GRS ($\rho = -0.128$, $p = 0.166$). (Fig. 3)

3.7 Predictors of reduced GLS (< 18%)

Binary logistic regression analysis demonstrated that higher pacing burden (> 40%) was significantly associated with reduced GLS (OR 2.32, 95% CI 1.01–5.37, $p = 0.048$). In addition, right ventricular pacing site was an independent predictor of GLS reduction (OR 2.62, 95% CI 1.07–6.39, $p = 0.035$). Other clinical variables including hypertension, diabetes mellitus, dyslipidaemia, and QRS duration were not significantly associated with reduced GLS (Table 6) At 6 months, 74 of 117 patients (63.2%) had left ventricular GLS reduced below the 18% threshold. Of these, 10 patients (13.5% of those meeting the threshold) already had GLS < 18% at baseline; the remaining 64 patients (54.7% of the total cohort) demonstrated a new reduction in GLS below 18% during follow-up and were classified as incident events for the logistic regression analysis. With 6 covariates included in the model, this corresponds to an events-per-variable (EPV) ratio of 10.7, meeting the conventionally recommended minimum threshold for model stability (Peduzzi et al., 1996).(Table 7)

Table 7
Predictors of reduced GLS at 6 months

Predictor	Odds Ratio (OR)	95% CI	p value
Pacing group > 40%	2.32	1.01–5.37	0.048
Hypertension	1.21	0.52–2.79	0.662
Diabetes mellitus	1.63	0.50–5.29	0.418
Dyslipidemia	0.59	0.05–6.38	0.665
QRS duration	1.01	0.996–1.03	0.117
RV pacing site	2.62	1.07–6.39	0.035
Binary logistic regression analysis identifying predictors of reduction in the study outcome. Results are expressed as odds ratios with 95% confidence intervals. OR > 1 indicates increased odds of reduction, whereas OR < 1 indicates decreased odds			

Discussion

The impact of right ventricular pacing on left ventricular function has been widely investigated in recent years. The present study aimed to evaluate the short-term effects of ventricular pacing on left ventricular

function and myocardial mechanics using conventional echocardiography and advanced deformation imaging techniques.

Previous studies have shown that prolonged right ventricular pacing can cause histopathological changes in the myocardium, which may lead to left ventricular dyssynchrony and functional impairment(4, 8). However, most of these studies have focused on patients with underlying cardiac disease. The effects of right ventricular pacing in patients with structurally normal hearts have not been extensively investigated(9). Therefore, this study was conducted to assess the early changes in left ventricular function and mechanics associated with pacing burden.

The findings of this study provide a nuanced understanding of the temporal and mechanical progression of pacing-induced cardiomyopathy (PICM). Our data suggest a pattern in which high-burden pacing is most clearly associated with longitudinal deformation (GLS), with a weaker, not independently significant, association for circumferential shortening, while radial deformation and rotational mechanics showed no significant change at the 6-month mark. This pattern of preserved twist and torsion contrasts with earlier reports describing an acute, global drop in rotational mechanics immediately after RV pacing initiation, suggesting a distinct, later-stage compensatory phase not previously characterized. In addition, our identification of RV pacing site, independent of pacing burden, as a predictor of reduced GLS corroborates and extends the findings of Iqbal et al., who reported that a pacing burden > 40% significantly affected GLS specifically when the lead was in the RV apex; the present study adds a multivariable-adjusted analysis of this association in an independent cohort

In the present study, left ventricular ejection fraction showed a mild decline during follow-up in both pacing groups. However, the reduction reached statistical significance only in patients with higher pacing burden (> 40%). In contrast, patients with lower pacing percentage ($\leq$ 40%) demonstrated a small but non-significant reduction in EF over the study period. These findings suggest that increased ventricular pacing burden may contribute to early deterioration in left ventricular systolic function. This aligns with established evidence that RV apical pacing creates an abnormal activation sequence and asynchronous contraction, leading to an immediate and progressive decrease in stroke volume and LVEF(10–14). Conversely, the < 40% group showed a significant reduction in left ventricular end-systolic diameter (ESD), which may be associated with a return toward more normal dimensions once bradycardia-induced compensatory mechanisms resolve after pacemaker implantation(15)

Mitral E velocity and aortic peak velocity declined significantly in both groups at follow-up, which may reflect early diastolic changes, although the E/E' ratio a more load-independent index of diastolic function did not change significantly in either group, so this interpretation should be regarded as preliminary. These findings are supported by research suggesting that pacing at higher heart rates shortens diastolic filling time, thereby decreasing the end-diastolic volume (EDV) and myocardial stretching forces(16)

Furthermore, high-burden pacing specifically resulted in reduced septal and lateral wall E' velocities, suggesting that pacing-associated dyssynchrony and the resulting left bundle branch block (LBBB)

pattern may adversely affect both systolic and diastolic long-axis function(17)

Our data reinforces the consensus that GLS is a more sensitive indicator of subclinical LV dysfunction than standard 2D ejection fraction(18–20)While GLS remained stable in the low-pacing group, the > 40% group experienced a significant reduction in GLS and longitudinal systolic strain rate at 6 months .This study identified a pacing burden > 40% as a strong predictor of reduced GLS (OR 2.32), consistent with literature showing that subclinical changes in strain as early as one month can predict the future development of pacing-induced cardiomyopathy.(21)

A notable observation in this study is the absence of a statistically significant change in twist and torsion mechanics despite the significant decline in GLS. LV twist, torsion rate, and peak twist remained stable in both groups, with no significant interaction between pacing burden and time. This preservation of rotational mechanics presents a significant contrast to the acute phase documented in the literature. Studies by Delgado et al. and Yaseen et al. have (10, 20)shown that RV apical pacing induces an immediate mechanical shock, resulting in a sharp drop in both LV twist and rotation within minutes to 24 hours of initiating pacing. We propose that following this initial acute insult, the heart enters a stabilization phase where the left ventricle utilizes its complex fibre architecture to maintain global performance. The anatomical basis for this compensation lies in the differential sensitivity of myocardial fibre layers. GLS reflects the contraction of the subendocardial longitudinal fibres, which are widely recognized as the most sensitive early indicators of subclinical dysfunction and are typically the first to fail under pacing-induced dyssynchrony. In contrast, LV twist is generated by the oppositely directed contraction of obliquely oriented helical myofibres. The stability of twist and torsion in our cohort suggests that these helical fibres, particularly in the sub epicardium, may serve as a mechanical buffer, temporarily preserving the "wringing motion" of the heart to maintain stroke volume while longitudinal efficiency is compromised.(22) This fibre-level explanation is speculative, as this study did not directly assess myocardial fibre architecture or function, and should be tested in future work.

This theory is further supported by our finding that Global Radial Strain (GRS) showed no significant correlation with cumulative pacing percentage, indicating that radial thickening also remains a preserved compensatory mechanism at the 6-month mark. Existing research supports this, noting that even when regional contractility is reduced, the heart can employ compensatory mechanisms such as ventricular geometry changes to ensure stroke volume remains normal at rest(23)

The correlation analysis revealed a significant negative relationship between cumulative pacing percentage and both GLS and GCS, while finding no correlation with GRS. This suggests that myocardial deformation changes associated with higher pacing burden are primarily manifest in the longitudinal and circumferential planes during the first 6 months post-implantation. The lack of radial impairment and preserved twist mechanics indicate that compensatory mechanisms may temporarily maintain global LV output before structural remodeling, such as expanded end-systolic volume, becomes more evident in the long term

The literature suggests that the transition from subclinical longitudinal decline to global functional failure is characterized by a "delayed deterioration" that typically manifests between 12 and 18 months post-implantation(22). This timeline is derived from a pediatric/young-adult cohort with long-term pacing and may not directly generalize to the present adult population. Continued clinical vigilance and regular echocardiographic monitoring beyond the 6-month window are therefore paramount to detect the exhaustion of compensatory mechanisms before they transition into irreversible pacing-induced cardiomyopathy.

The RV pacing site is a critical determinant of subclinical myocardial injury, appearing in our results as the most significant independent predictor of GLS reduction at the 6-month follow-up. Notably, the site of lead placement was a stronger predictor of longitudinal injury than the > 40% cumulative pacing burden, suggesting that where the heart is activated may be more critical to mechanical preservation than the total volume of pacing in the mid-term. The detrimental impact of apical pacing observed in our data is supported by established literature characterizing the RV apex as hemodynamically inferior. RV apical pacing (RVAP) induces an abnormal electrical activation sequence that bypasses the specialized His-Purkinje system, forcing depolarization to spread through the slower-conducting myocardium. This non-physiologic sequence results in acute interventricular and intraventricular dyssynchrony(8)Our findings suggest that the clinical consequences of a high pacing burden are often associated with lead location. This mirrors the findings of Iqbal et al.,(17) who demonstrated that a pacing burden > 40% only significantly affected GLS when the lead was in the RV apex; conversely, if the lead was placed in the RV outflow tract (RVOT), a high pacing burden did not cause a significant decline in GLS

Conclusion

RV pacing burden was associated with early changes in left ventricular myocardial mechanics within six months of pacemaker implantation. A higher pacing burden (> 40%) was associated with a significant decline in longitudinal strain (GLS), with a weaker, not independently significant, association for circumferential strain; radial strain and rotational mechanics showed no significant change, though these comparisons were not formally powered. Global longitudinal strain proved to be a more sensitive marker of early ventricular dysfunction than conventional ejection fraction. Additionally, right ventricular lead location emerged as a determinant of myocardial injury, corroborating and extending prior reports, with apical pacing showing greater association with strain reduction. Early strain-based assessment may therefore help identify patients at risk for pacing-induced cardiomyopathy and support closer echocardiographic surveillance.

Limitations

This study has several limitations. First, it was a single-center observational study with a relatively modest sample size, which may limit the generalizability of the findings. Second, the majority of patients had right ventricular apical pacing, which limited the ability to compare the effects of different pacing sites. Third, other factors that may influence myocardial mechanics, such as medication use and

underlying comorbidities, were not extensively evaluated; in particular, although patients with overt structural heart disease were excluded, 57.3% of the cohort had hypertension, which can independently affect myocardial strain. Hypertension was not, however, a significant independent predictor of reduced GLS in our multivariable model (Table 6), arguing against it being the principal driver of the observed strain changes. Also this study did not include a dedicated intraventricular dyssynchrony index, such as time-to-peak strain standard deviation or septal-to-lateral wall delay, to directly quantify mechanical dyssynchrony between the two pacing groups. While GLS reduction and altered strain patterns provide indirect evidence of pacing-related dyssynchrony, future studies incorporating dedicated dyssynchrony indices would help clarify the mechanistic link between pacing burden, electromechanical dyssynchrony, and the observed decline in longitudinal strain.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Ethics Committee, Kasturba Medical College, Manipal (IEC No. 875/2017) and was registered with the Clinical Trials Registry of India. All procedures involving human participants were performed in accordance with the relevant guidelines and regulations and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment in the study.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests

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Author Contribution

RKS conceived and designed the study, collected and analysed the data, and drafted the manuscript. JS contributed to the study design and echocardiographic image acquisition and interpretation. UP contributed to patient recruitment and clinical data collection. PR contributed to the interpretation of pacing data and critically revised the manuscript. MP contributed to statistical analysis and interpretation of results. TD contributed to patient management and clinical oversight. RN contributed to

data collection and manuscript preparation. SS contributed to literature review and manuscript drafting. KN, as the senior/corresponding author, supervised the study, contributed to conception and design, and critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript

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Nil

Data Availability

The datasets generated and/or analysed during the current study are available from the corresponding author upon reasonable request

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Figures

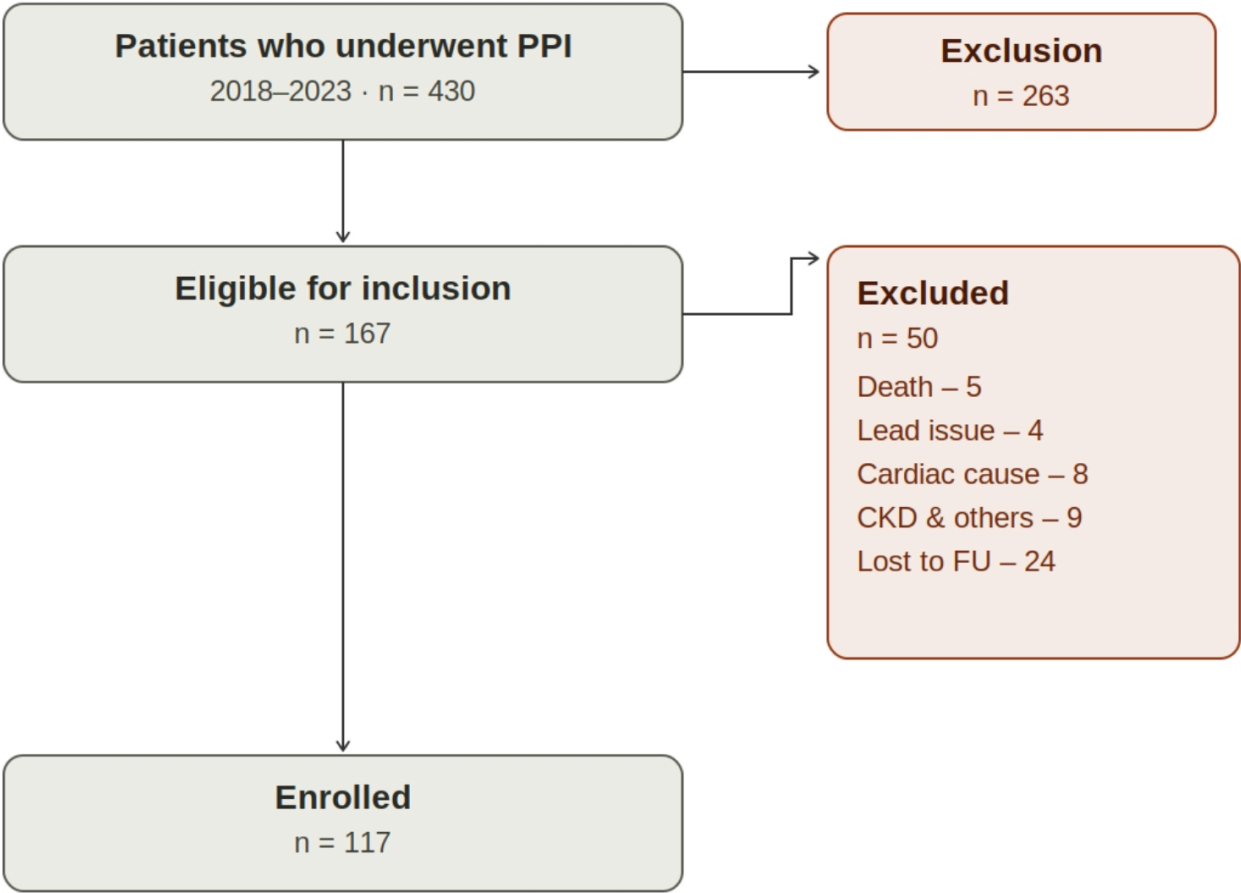


Figure 1

Flow diagram of patient screening, exclusion, and enrollment (2018–2023)

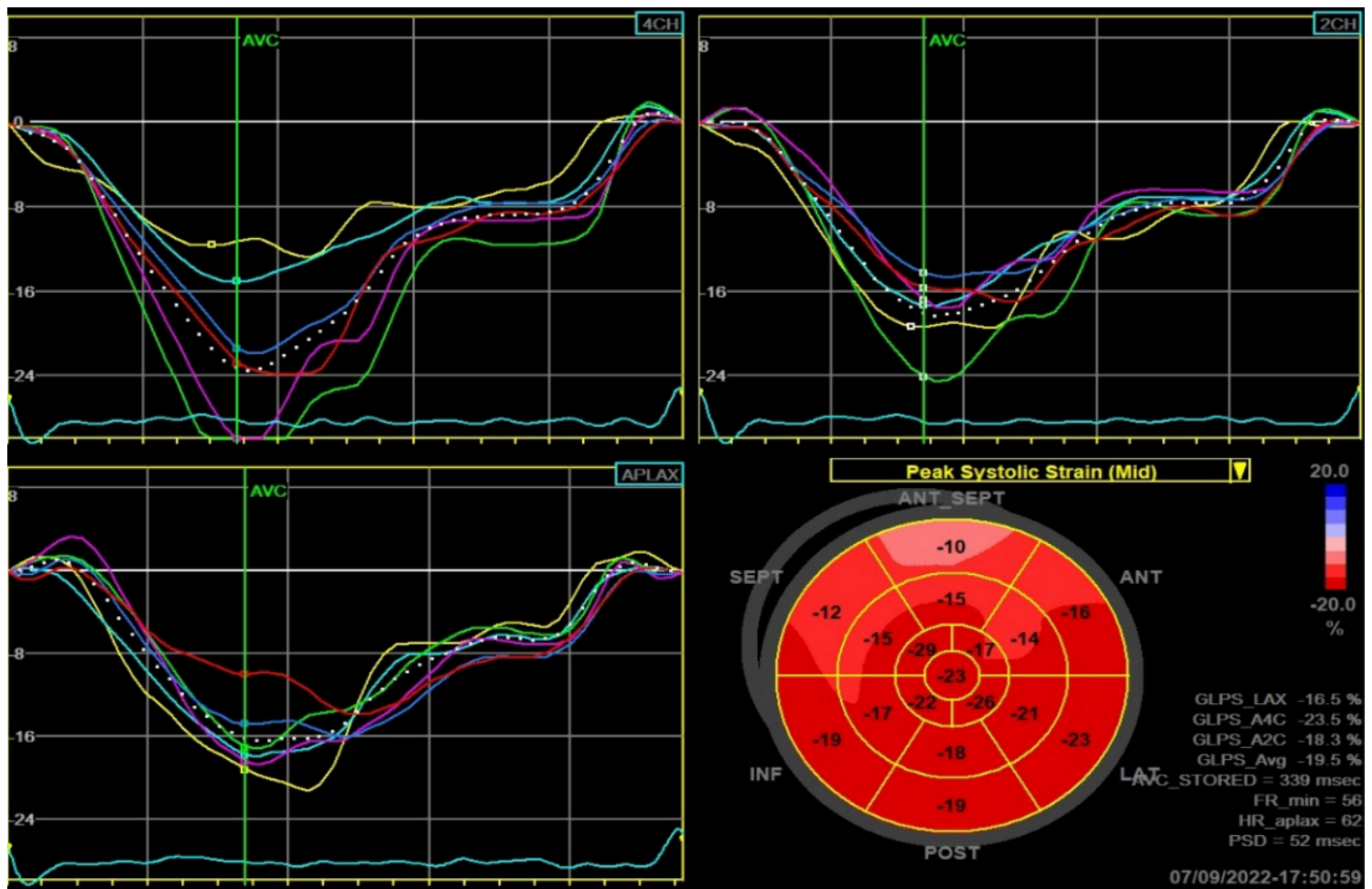


Figure 2

Assessment of left ventricular function using speckle-tracking echocardiography.

Representative speckle-tracking echocardiographic images demonstrating left ventricular strain analysis. Longitudinal strain curves from apical four-chamber, two-chamber, and long-axis views are shown, along with the corresponding bull's-eye plot illustrating segmental and global longitudinal strain.

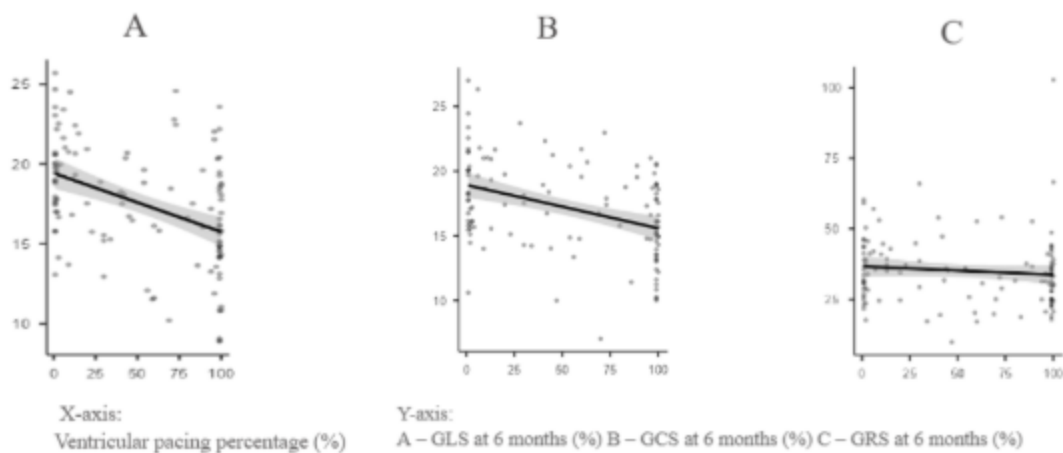


Figure 3

Correlation between ventricular pacing percentage and left ventricular strain parameters at 6 months.

Scatter plots demonstrating the association between ventricular pacing burden and (A) global longitudinal strain (GLS), (B) global circumferential strain (GCS), and (C) global radial strain (GRS). The solid line represents the linear regression with 95% confidence interval.

Supplementary Files

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