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A Study on Left Ventricular Function in Patient Undergo Right Ventricular Apical Pacing in Medical College and Hospital, Kolkata

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ABSTRACT

Introduction: Right ventricular apical (RVA) pacing is a commonly used pacing modality in patients requiring permanent pacemakers. However, long-term RVA pacing may lead to electrical and mechanical dyssynchrony, which can adversely affect left ventricular (LV) function and potentially result in pacing-induced cardiomyopathy. This study aims to evaluate changes in left ventricular function in patients undergoing right ventricular apical pacing.
Aims: To assess the impact of chronic right ventricular apical pacing on left ventricular systolic and diastolic function.
Materials and Methods: This was an observational study conducted on patients who underwent permanent pacemaker implantation with right ventricular apical lead placement. Baseline clinical evaluation and echocardiographic assessment of left ventricular ejection fraction (LVEF), LV dimensions, and diastolic parameters were recorded prior to implantation and followed up at predefined intervals. Changes in LV function over time were analyzed and correlated with pacing burden and clinical outcomes.
Results: In the present study of 48 patients undergoing right ventricular apical pacing, left ventricular function showed a significant shift from normal to reduced functional categories over the follow-up period. At baseline, 38 patients had normal left ventricular function (LVEF ≥55%), which decreased to 24 patients at 18 months follow-up. Patients with mild left ventricular dysfunction (LVEF 45–54%) increased from 10 at baseline to 18 at follow-up. Moderate left ventricular dysfunction (LVEF 35–44%) was not present at baseline but was observed in 6 patients during follow-up. This deterioration in left ventricular functional status was statistically significant, with a p-value of 0.002.
Conclusion: Right ventricular apical pacing may be associated with deterioration of left ventricular function in a proportion of patients, particularly with high pacing dependency. Regular echocardiographic monitoring is recommended for early detection of pacing-induced LV dysfunction and timely intervention.

***Keywords**:* Right ventricular apical pacing, left ventricular function, pacing-induced cardiomyopathy, echocardiography, ventricular dyssynchrony, permanent pacemaker.

INTRODUCTION

Permanent pacemaker implantation is a well-established and life-saving therapy for patients with symptomatic bradyarrhythmias, including atrioventricular block, sick sinus syndrome, and certain conduction system diseases. Among the various pacing strategies, right ventricular apical (RVA) pacing has historically been the most commonly used due to its technical simplicity, stability of lead placement, and reliable sensing and capture thresholds [1,2]. However, despite its widespread use, accumulating evidence suggests that chronic RVA pacing is not physiologically optimal and may have deleterious effects on cardiac structure and function, particularly on the left ventricle.

The normal activation of the ventricles occurs through the His–Purkinje conduction system, resulting in near-simultaneous depolarization of both ventricles and coordinated contraction. In contrast, RVA pacing initiates electrical activation from the right ventricular apex, leading to an abnormal sequence of ventricular depolarization resembling left bundle branch block (LBBB)-like activation pattern [3]. This abnormal electrical activation results in mechanical dyssynchrony between the interventricular and intraventricular segments, which can impair efficient ventricular contraction, reduce stroke volume, and increase myocardial workload.

Over time, this pacing-induced dyssynchrony may lead to adverse left ventricular remodeling. Several studies have demonstrated that chronic RVA pacing can result in progressive reduction in left ventricular ejection fraction (LVEF), increased left ventricular end-systolic and end-diastolic volumes, and deterioration of both systolic and diastolic function [4,5]. This condition has been described as pacing-induced cardiomyopathy (PICM), a potentially reversible form of cardiomyopathy that develops in a subset of patients exposed to a high burden of right ventricular pacing.

The incidence of PICM varies widely in literature, ranging from 9% to 26% depending on patient population, pacing burden, baseline ventricular function, and duration of follow-up [6]. Patients with pre-existing borderline left ventricular function, wider paced QRS duration, and high percentage of ventricular pacing are at increased risk. Importantly, even patients with initially preserved LVEF may develop significant LV dysfunction over time, highlighting the need for periodic reassessment.

The pathophysiological mechanisms underlying RVA pacing-induced LV dysfunction are multifactorial. Chronic electrical dyssynchrony leads to regional differences in myocardial workload, altered perfusion patterns, and abnormal wall stress distribution. These changes promote maladaptive remodeling, myocardial fibrosis, and neurohormonal activation, ultimately contributing to progressive decline in ventricular performance [7]. In addition, interventricular dyssynchrony may impair diastolic filling and reduce overall cardiac efficiency.

Advances in pacing technology have led to the development of alternative pacing sites and physiological pacing strategies, such as His bundle pacing and left bundle branch area pacing, which aim to preserve normal ventricular activation. These approaches have shown promising results in reducing the risk of pacing-induced ventricular dysfunction compared to traditional RVA pacing [8]. However, RVA pacing continues to be widely used in many centers, especially in resource-limited settings, due to its ease of implantation and lower procedural complexity.

Echocardiography remains the primary non-invasive tool for monitoring left ventricular function in patients with permanent pacemakers. Parameters such as LVEF, global longitudinal strain (GLS), LV dimensions, and diastolic function indices are useful in detecting early ventricular dysfunction before symptomatic heart failure develops [9]. Regular follow-up imaging is therefore essential for early identification of patients at risk of pacing-induced cardiomyopathy.

Given the increasing prevalence of pacemaker implantation worldwide and the potential long-term adverse effects of RVA pacing on cardiac function, it is important to systematically evaluate its impact on left ventricular performance. [10]Understanding the relationship between RVA pacing and LV dysfunction will help in early diagnosis, timely intervention, and consideration of alternative pacing strategies in high-risk patients. This study is therefore undertaken to assess the effect of right ventricular apical pacing on left ventricular function in patients with permanent pacemakers.

MATERIALS AND METHODS

Study design: This study is designed as a hospital-based prospective observational study

Study place: Department of Cardiology Medical College and Hospital, Kolkata**.**

Study duration: The study will be carried out over a period of 18 months, including patient recruitment, baseline assessment, and follow-up echocardiographic evaluation.

Study population: The study population will include adult patients who have undergone permanent pacemaker implantation with right ventricular apical lead placement and who are attending regular follow-up in the cardiology outpatient department.

Sample size: 48 patients.

Inclusion criteria:

- Patients aged ≥18 years undergoing permanent pacemaker implantation
- Patients with right ventricular apical pacing lead position confirmed by chest X-ray/ECG criteria
- Patients with baseline echocardiographic assessment available prior to implantation
- Patients with preserved or mildly reduced left ventricular ejection fraction at baseline
- Patients willing to give informed consent and available for follow-up

Exclusion criteria:

- Patients with pre-existing moderate to severe left ventricular systolic dysfunction (LVEF <40%)
- Patients with prior history of myocardial infarction or established cardiomyopathy
- Patients with significant valvular heart disease (moderate to severe)
- Patients with congenital heart disease
- Patients with chronic atrial fibrillation with uncontrolled ventricular rate
- Patients with previous cardiac resynchronization therapy (CRT) or His bundle pacing
- Patients lost to follow-up or with incomplete echocardiographic data
- Patients unwilling to participate in the study

Statistical analysis :

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests for a difference in mean involved independent samples or unpaired samples. Paired t-tests were a form of blocking and had greater power than unpaired tests. A chi-squared test ($\chi 2$ test) was any statistical hypothesis test wherein the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.

Explicit expressions that can be used to carry out various t-tests are given below. In each case, the formula for a test statistic that either exactly follows or closely approximates a t-distribution under the null hypothesis is given. Also, the appropriate degrees of freedom are given in each case. Each of these statistics can be used to carry out either a one-tailed test or a two-tailed test.

Once a t value is determined, a p-value can be found using a table of values from Student's t-distribution .If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis.
P-value ≤ 0.05 was considered for statistically significant.

RESULT

Table 1: Baseline Demographic Profile of Patients (n = 48)

Variable	Category	Number of Patients	Percentage (%)
Age (years)	<40	8	16.7
	40–60	22	45.8
	>60	18	37.5
Gender	Male	28	58.3
	Female	20	41.7

Table 2: Indication for Pacemaker Implantation (n = 48)

Indication	Number of Patients	Percentage (%)
Complete AV block	26	54.2
Sick sinus syndrome	14	29.2
High-grade AV block	8	16.6

Table 3: Left Ventricular Ejection Fraction (LVEF) – Baseline vs Follow-up

Time Point	Mean LVEF (%)	SD	Median	P value
Baseline	56.8	5.4	57	<0.0001
Follow-up (18 months)	50.2	6.1	50	

Table 4: Change in LV Function Categories (n = 48)

V Function Status	Baseline (n)	Follow-up (n)	P value
Normal (≥55%)	38	24	0.002
Mild dysfunction (45–54%)	10	18	
Moderate dysfunction (35–44%)	0	6	

Table 5: Correlation Between Right Ventricular Pacing Burden and Left Ventricular

Right Ventricular Pacing Burden (%)	Number of Patients (n)	Mean LVEF (%)	P value
<40%	12	54.6 ± 3.2	<0.0001
40–80%	18	51.8 ± 4.1	
>80%	18	47.5 ± 5.0	

Figure 1 : Left Ventricular Ejection Fraction (LVEF) – Baseline vs Follow-up

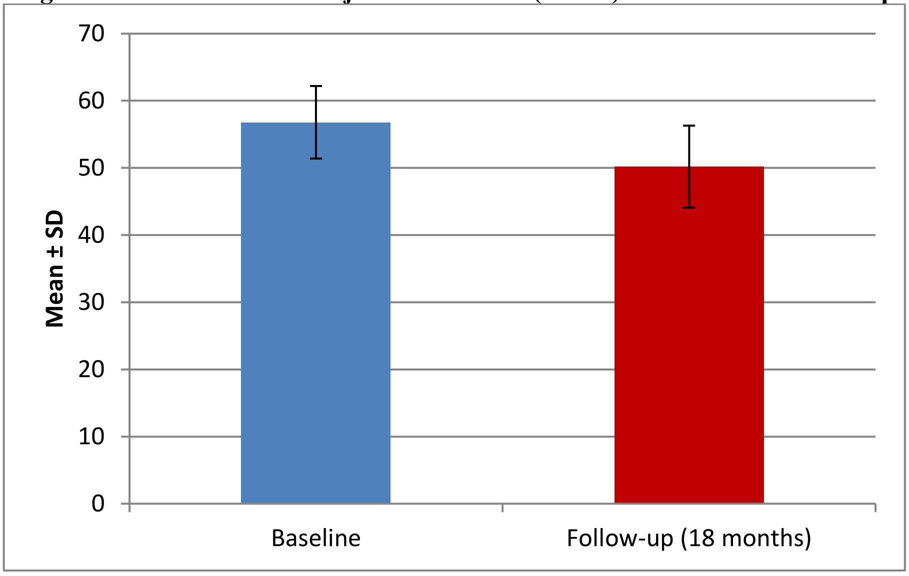
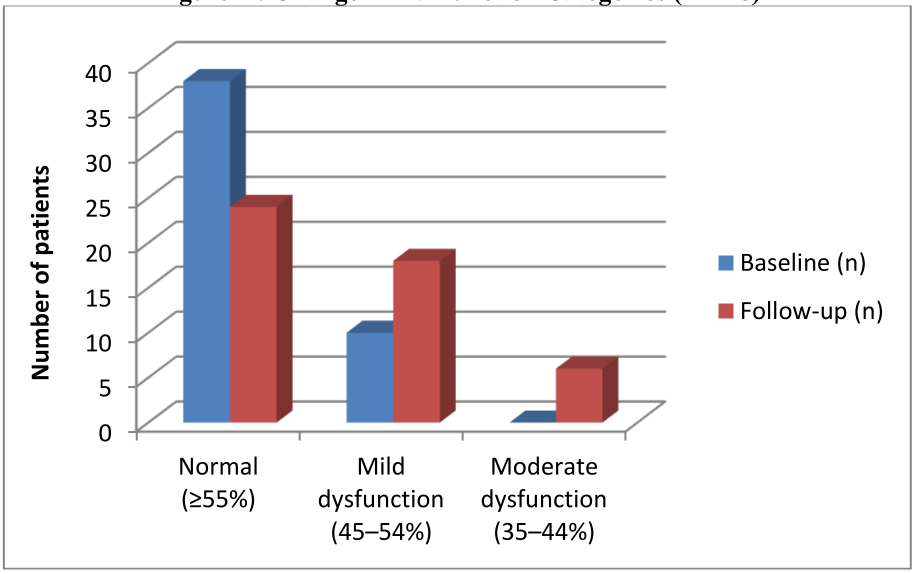


Figure 2 : Change in LV Function Categories (n = 48)



In the present study comprising 48 patients who underwent right ventricular apical pacing, the majority belonged to the 40–60 years age group, with 22 patients (45.8%), followed by 18 patients (37.5%) aged more than 60 years, and 8 patients (16.7%) aged less than 40 years. Regarding gender distribution, 28 patients (58.3%) were male and 20 patients (41.7%) were female.

In the present study of 48 patients undergoing right ventricular apical pacing, the most common indication for permanent pacemaker implantation was complete atrioventricular (AV) block, observed in 26 patients (54.2%). This was followed by sick sinus syndrome in 14 patients (29.2%). High-grade AV block was the indication in 8 patients (16.6%).

In the present study of 48 patients undergoing right ventricular apical pacing, the mean left ventricular ejection fraction (LVEF) at baseline was 56.8 ± 5.4. At follow-up after 18 months, there was a significant reduction in mean LVEF to 50.2 ± 6.1. This decline in left ventricular systolic function was statistically highly significant, with a p-value of <0.0001.

In the present study of 48 patients undergoing right ventricular apical pacing, left ventricular function showed a significant shift from normal to reduced functional categories over the follow-up period. At baseline, 38 patients had normal left ventricular function (LVEF ≥55%), which decreased to 24 patients at 18 months follow-up. Patients with mild left ventricular dysfunction (LVEF 45–54%) increased from 10 at baseline to 18 at follow-up. Moderate left ventricular dysfunction (LVEF 35–44%) was not present at baseline but was observed in 6 patients during follow-up. This deterioration in left ventricular functional status was statistically significant, with a p-value of 0.002 .

In the present study of 48 patients undergoing right ventricular apical pacing, a significant correlation was observed between the percentage of right ventricular pacing burden and left ventricular ejection fraction (LVEF). Patients with a pacing burden of <40% included 12 patients and had a mean LVEF of 54.6 ± 3.2. In the 40–80% pacing burden group, comprising 18 patients, the mean LVEF decreased to 51.8 ± 4.1. The lowest mean LVEF of 47.5 ± 5.0 was observed in patients with a pacing burden of >80%, which included 18 patients. This progressive decline in LVEF with increasing pacing burden was statistically highly significant, with a p-value of <0.0001.

DISCUSSION

In the present study comprising 48 patients undergoing right ventricular apical (RVA) pacing, there was a significant decline in left ventricular systolic function over 18 months. The mean LVEF decreased from 56.8 ± 5.4 at baseline to 50.2 ± 6.1 at follow-up ($p < 0.0001$), indicating that chronic RVA pacing adversely affects left ventricular performance. Similar observations were reported by Sweeney MO et al., who in the MOST trial demonstrated that higher ventricular pacing burden is associated with increased risk of heart failure hospitalization and adverse ventricular remodeling [11]. In addition, Wilkoff BL et al. also showed that patients with frequent RV pacing have worse clinical outcomes compared to those with minimal ventricular pacing exposure [12].

In our study, a significant shift in LV functional status was noted, where normal LV function decreased from 38 to 24 patients, while mild and moderate dysfunction increased over time ($p = 0.002$). This finding is consistent with the work of Tops LF et al., who demonstrated that RVA pacing induces electrical and mechanical dyssynchrony even in patients with initially normal ventricular function [13]. Similarly, Thambo JB et al. reported that chronic RV pacing in young patients leads to progressive LV dilation and systolic dysfunction due to abnormal activation patterns [14].

A strong relationship between pacing burden and LV dysfunction was also observed in the present study. Patients with >80% pacing burden had significantly lower LVEF (47.5 ± 5.0) compared to those with <40% pacing burden (54.6 ± 3.2), with $p < 0.0001$. This finding is supported by Khurshid S et al., who identified high RV pacing percentage as an independent predictor of pacing-induced cardiomyopathy [15]. Likewise, Santos JM et al. demonstrated that increased RV pacing burden correlates directly with worsening LV function and heart failure symptoms in long-term follow-up patients [16].

The underlying mechanism of LV dysfunction in RVA pacing has been explained by Prinzen FW and Peschar M, who showed that abnormal electrical activation from the RV apex causes regional mechanical inefficiency and ventricular dyssynchrony [17]. Furthermore, Miyazaki S et al. highlighted that this dyssynchrony leads to adverse remodeling, increased wall stress, and progressive reduction in systolic function [18].

Recent studies have compared RVA pacing with physiological pacing strategies. Abdelrahman M et al. demonstrated that His bundle pacing is associated with lower risk of heart failure hospitalization and better preservation of LV function compared to RVA pacing [19]. Similarly, Zanon F et al. reported that left bundle branch area pacing provides more physiological ventricular activation and reduces pacing-induced cardiomyopathy risk [20]. These findings suggest that alternative pacing strategies may be more beneficial in patients requiring long-term pacing support.

CONCLUSION

The present study demonstrated that right ventricular apical pacing is associated with a significant decline in left ventricular systolic function over time. A statistically significant reduction in mean LVEF was observed during follow-up, along with a progressive shift from normal to impaired LV functional status. Furthermore, a strong association was identified between higher right ventricular pacing burden and worsening left ventricular function. These findings suggest that chronic RVA pacing may contribute to pacing-induced ventricular dyssynchrony and subsequent left ventricular dysfunction. Therefore, regular echocardiographic monitoring is essential in patients with permanent pacemakers, and alternative physiological pacing strategies should be considered in patients requiring high ventricular pacing dependency to prevent long-term adverse cardiac remodeling.

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