ORIGINAL ARTICLE e-ISSN: 2349-0659 p-ISSN; 2350-0964

Assessment of Biventricular Function by Echocardiography in Patients with Right Ventricular Apical Pacing

Rakesh Tumula, Nrushen Peesapati*

Abstract

Introduction: Artificial cardiac pacing is a consolidated, safe, and effective treatment strategy that has been evolving for 60 years. In a normal heart, the left ventricle (LV) contracts in a fast and synchronized manner. Stimulation anywhere in the right ventricle (RV) alters the natural pattern of activation and as a consequence the ventricular contraction. This may lead to the induction of asynchrony with potential risk for the development of ventricular dysfunction**.** The majority of studies have looked into left ventricular dysfunction, while effects on the RV have not been studied. Furthermore, there are little data on the chronology of events that occur in pacemaker patients. We, therefore, aimed to evaluate diastolic and systolic functions of LV and RV in consecutive patients undergoing Ventricle is paced, sensed, and the pulse generator inhibits pacing output in response to a sensed ventricular event (VVI) permanent pacemaker implantation over 6 months using trans-thoracic echocardiography. **Materials and Methods:** This was a hospital-based observational study from October 2017 to April 2019. **Conclusion:** Diastolic abnormalities are first to appear which are followed by appearance of systolic abnormalities of the respective ventricles. The right ventricular dysfunction (tricuspid annular plane systolic excursion <17 mm) developed in 28% at 1 month and in 62% at the end of 6 months. The left ventricular dysfunction (ejection fraction< 50%) occurs later, LV dysfunction developed in 14%, and worsening of LV function developed in 16% of the study population at the end of study, that is, 6 months.

Keywords: Left ventricle function, Permanent pacemaker, Right ventricle function, VVI mode

Asian Pac. J. Health Sci., (2021); DOI: 10.21276/apjhs.2021.8.1.3

Introduction

Artificial cardiac pacing is a consolidated, safe, and effective treatment strategy that has been evolving for 60 years. In a normal heart, the left ventricle (LV) contracts in a fast and synchronized manner. Stimulation anywhere in the right ventricle (RV) alters the natural pattern of activation and as a consequence the ventricular contraction. This may lead to the induction of asynchrony with potential risk for the development of ventricular dysfunction.[1,2] In India, about 10,000 pacemakers implanted every year. There is a considerable cost variation between modern-day pacemakers and conventional pacemakers. Single chamber right ventricular apical pacing continues to be the dominant pacing mode in the developing countries majorly due to economic limitations, despite the well-known advantages of dual-chamber pacing.[3] Pacemaker-induced cardiac dysfunction has been recognized for a long time, and the effect of the right ventricular apical pacing (VVI) on the left ventricular functions has been studied previously.[4-10] Yet, there remain some uninvestigated areas such as the chronology of events to occur. The majority of studies have looked into left ventricular dysfunction, while effects on the RV have not been studied.[11-13] Furthermore, there are little data on the chronology of events that occur in pacemaker patients. We, therefore, aimed to evaluate diastolic and systolic functions of LV and RV in consecutive patients undergoing VVI permanent pacemaker implantation (PPI) over 6 months using trans-thoracic echocardiography.[3]

Aims and Objectives

This study aims to evaluate diastolic and systolic functions of the LV and RV in patients undergoing VVI PPI over 6 months using trans-thoracic echocardiography.

Department of Cardiology, Andhra Medical College, Visakhapatnam, Andhra Pradesh, India

Corresponding Author: Dr. Nrushen Peesapati, Department of Cardiology, Andhra Medical College, Visakhapatnam - 530 001, Andhra Pradesh, India. E-mail: nrushenpspt@gmail.com

How to cite this article: Tumula R, Peesapati N. Assessment of Biventricular Function by Echocardiography in Patients with Right Ventricular Apical Pacing. Asian Pac. J. Health Sci., 2021; 8(1):1- 4.

Source of support: Nil

Conflicts of interest: None

Received: 01/10/2020 **Revised:** 31/10/2020 **Accepted:** 17/11/2020

Materials and Methods

Type of Study: This was a hospital-based observational study

Site of study: KING GEORGE HOSPITAL

Study period: October 2017–April 2019

Sample size: 50

Inclusion Criteria

1. All consecutive patients undergoing VVI pacemaker implantation according to AHA guidelines
2. Sinus node dysfunction with documented symptomatic bradycardia
3. Complete heart block, acquired, or congenital
4. Second degree atrioventricular (AV) block with symptomatic bradycardia
5. Atrial fibrillation, atrial flutter, or with complete heart block or advanced AV block[14-20]

Exclusion Criteria

1. Patients undergoing other mode PPI implantations will be excluded from the study
2. Prior severe LV dysfunction[21-26]

All consecutive patients undergoing RV apical VVI pacemaker implantation were included in the study. Written informed consent was taken for inclusion in the current study.

Procedure

A thorough clinical assessment and resting 12-lead electrocardiogram (ECG) were done on the day of admission, on day 7 after PPI, and at 1 month and 6 months follow-up.

Patients were discharged with a programmed pacing rate of 70/min. At follow-up visits, patients were evaluated for any evidence of dyspnea, in the absence of any recognizable recent myocardial infarction, respiratory illness, anemia, and new LV ejection fraction of less than 50% or worsening of LV ejection function is noted. The left ventricular function was assessed by 2D echocardiography by a single operator.[27-31]

Echocardiography Protocol

Baseline echocardiographic evaluation of patients was done within 24 h of hospital admission, with the temporary pacemaker rate being kept at 70/min. 2D-echocardiography was repeated at day 7 of hospital admission, 1 month, and 6 months after implantation during follow-up. Each measurement was taken in three consecutive paced beats not preceded by a sinus P wave, and the average value was considered as the representative value.

The left ventricular internal diameter in systole and diastole were measured in the parasternal long-axis view using the M-mode. RV dimension is best estimated at end diastole from a RV-focused apical 4-chamber view. The right ventricular internal diameter in systole (RVIDs) and diastole (RVIDd) were taken in the apical 4-chamber view and their mean was chosen as the RVID. The left atrial size was measured in the end systole on M-mode parasternal long-axis view perpendicular to the long axis of the LA posterior wall, leading edge to leading edge measured at the level of the aortic sinuses.[32-38]

LV ejection time (LVET) was calculated using Doppler aortic flow. The LVET was measured as the duration of flow using standard pulsed wave Doppler with sample volume in the left ventricular outflow tract just below the aortic valve leaflets. The RV ejection time (RVET) was calculated using Doppler pulmonary flow.

The LV diastolic parameters were calculated in apical 5-chamber view keeping the Doppler cursor at a point where both mitral inflow and aortic outflow could be obtained. The LV isovolumic relaxation time (LV-IVRT) was taken as a period from the end of Doppler aortic flow to the beginning of mitral inflow.

The deceleration time of mitral inflow (MV-DT) was measured from the peak of the mitral flow to return to baseline with the Doppler cursor placed just beyond the mitral annulus in apical four-chamber view. The tricuspid deceleration time (TV-DT) was measured in a manner similar to mitral DT. Color Doppler was used to conforming and quantifying tricuspid valvular regurgitation, using the area of the regurgitant jet.[39-41]

A quantitative approach to assess RV function is the measurement of the tricuspid annular plane systolic excursion (TAPSE). The TAPSE estimates RV systolic function by measuring the level of systolic excursion of the lateral tricuspid valve annulus toward the apex in the RV focused four-chamber view. The approach appears reproducible and proved to be a strong predictor of prognosis in heart failure. It is abnormal <17 mm.[42]

Statistical Methods

Descriptive and inferential statistical analysis has been carried out in the current study. Results on continuous measurements are presented on Mean ± SD (Min-Max), and results on categorical measurements are shown in number (%). Significance is assessed at 5% level of significance. The following assumptions on data are made,[43-46]

Paired *t*-test has been used to find the significance of study parameters on the continuous scale within each group.

The statistical software, namely, SPSS 22.0 and R environment ver. 3.2.2 were used for the analysis of the data, and Microsoft Word and Excel have been used to generate graphs, tables, etc.

Results

The study population consisted of 50 patients with symptomatic bradycardia requiring pacemaker and underwent VVI mode pacing. Over the 6 months follow-up, no patient died. Data could, therefore, be obtained for all 50 patients at baseline, day 7, 1 month, and at 6 months. All the patients were from the Andhra Pradesh during the study period October 2017–April 2019.

The majority of the patients are in the age group of 50–60 years [42.0%] followed by 61–70 years and 41–45 years [36%]. Twenty-seven patients were men, and 23 were women.

Hypertension found in 60% of the total study population. Hypertension diagnosed in four patients, diabetes mellitus observed in 26 patients, that is, 52% of the study population. Hypothyroidism found in 13 patients out of which 5 patients are newly detected. Underlying coronary artery disease found in 10% of study population of varying duration.

Twenty-five patients (50%) presented with syncope and 15 patients (30%) are presented with dyspnea while attending the emergency (NYHA II in 13 patients, NYHA III in 7 patients, and NYHA IV in 5 patients). Both syncope and dyspnea were observed in 10 patients (20%) [Figure 1].

Out of 50 patients, 33 patients were not on any beta-blockers antecedent to the admission and 17 patients (34%) were on beta-blockers for hypertension, and drugs were stopped 5 days before pacemaker implantation.

Most common indication was degenerative complete AV nodal block occurred on 26 patients (52% of study population), followed by sinus node dysfunction in 15 patients, high-grade AV block in 5 patients (10%), and Mobitz type 2 block, in 4 patients [Figure 2].

The mean LV ejection fraction (LVEF) of the group was 59.66 ± 7.55% at the day 1 of study population as assessed by modified Simpson method. Eight patients had LVEF of <50% with four having dilated cardiomyopathy with normal coronaries and other four having mild coronary artery disease on coronary angiogram.

The RVID showed an earlier increase from baseline ($P < 0.05$) at day 7 and progressively increased to mean of 3.28 ± 0.66 cm in the 6 months follow-up period. RV dimensions are statistically significant at day 7, 1 month, and 6 months [Figure 3].

The left ventricular dimension showed a progressive increase from baseline ($P < 0.05$) at 1 month and at 6 months for the LV internal diameter during diastole and ($P < 0.05$) at 6 months for the LV internal diameter during systole.

The left atrial size increased from day 1 to 1 month and statistically significant increase in the left atrial diastolic diameter was observed at 6 months ($P < 0.05$) of the study period.

Tricuspid regurgitation considered mild if regurgitate jet area is <5 cm^2, moderate if jet area is 5–10 cm^2, and severe if jet area is >10 cm^2. Mild regurgitation is present in all patients at the end of 6 months, moderate tricuspid regurgitation noted in 10 patients a month, that is, (20% of study population) and 36% at the end of 6 months and severe tricuspid regurgitation noted in eight patients at the end of 6 months.

TAPSE is a parameter of global RV function which describes apex-to-base shortening. TAPSE correlates closely with the RVEF and has been found to be both highly specific and easy to measure. As the right ventricular systolic function concerned, mean value of TAPSE reached statistically significant difference at the end of 1 month with a mean of 18.48 ± 2.13 and 6 months with a mean of 17.18 ± 2.61 and RV dysfunction noted in 31 patients (62% study population), that is, TAPSE <17 mm over 6 months.

The right ventricular ejection period as calculated by Doppler pulmonary flow decreased significantly from the end of 1 month and maintained statistical significance till the end of study period.

The left ventricular ejection fraction at baseline was a mean of 59.66 ± 7.55% and at the end of 1 month was 56.48 ± 6.99% and at the end of 6 months was 51.16 ± 7.23% which is statistically significant. New-onset LV dysfunction, that is, <50% developed in 7 patients (14%) and worsening of LV function was noted in 8 patients (16%) at the end of study.

LV IVRT at baseline was 84.40 ± 8.26 ms, at the end of 1 week was 87.20 ± 9.95 ms, at the end of 1 month was 110.70 ± 11.66 ms, and at the end of 6 months was 126.00 ± 10.45 ms. Statistically significant at the end of 1 month and 6 months [Table 1].

MV-DT was measured from the peak to return to baseline of the mitral inflow with the Doppler cursor placed just beyond the mitral annulus in apical four-chamber view has a normal of 178.2 + 43 ms, the present study MV-DT at baseline was 148.60 ± 16.76 ms and at the end of 6 months was 194.90 ± 19.97 ms which is statistically significant.

The LVET as assessed by duration of pulse wave Doppler of aortic flow just below the aortic leaflets has a normal value of 302 ± 22 ms, the present study LVET at baseline was 304.60 ± 12.42 ms gradually progressed to 287 ± 11.92 ms at the end of 1 month and 276 ± 13.63 ms at the end of 6 months and is statistically significant [Table 2].

Discussion

The majority of the patients were in between 50 and 60 years of age group, that is, 42% and followed by 61–70 years age group (36%) with a mean age was 61.8 ± 10.9, and male patients were 54% more common than female patients (46%).

A study by Dwivedi *et al*.[3] did in 48 consecutive VVI pacemaker patients with a mean age of 65.6 ± 11.8 years, with 66.712% were male.

Kanse *et al*.[47] did in 2015 with 37 patients, 21 (57%) were male, and the mean age of the study group was 63.24 years.

de sá *et al*.[2] did in 20 patients with a mean age of 58 ± 11 with 60% male subjects with a follow-up period of 2 years.

Silva *et al*.[48] study with 75 patients, mean age 70.9 ± 14 years, of whom 22.6% were male, and evaluations were done over 6 months.

Compared to the previous studies, the mean age was younger and male gender is predominantly affected.

In the present study, 60% of the study population had hypertension. About 52% of patients had diabetes hypothyroidism noted in 13 patients, and a history of previous coronary artery disease was observed in 10% of the study population.

A study by Dwivedi *et al*.[3] observed diabetes in 28.9% hypertension in 26.7% and coronary artery disease in 13.3% of the study population. Kanse *et al*.[47] observed diabetes in 21.7% hypertension in 59.6% coronary artery disease in 13.55% and thyroid disease in 16.6% of the study population.

In the present study, syncope was the predominant presenting symptom 50% followed by dyspnea 30% and both syncope and dyspnea in 20%.

A study by Dwivedi *et al*.[3] observed recurrent syncope 80%, dyspnea 8.9%, and both syncope and dyspnea in 8.9% and acute myocardial infarction in 2.2% study population. According to Kanse *et al*.,[47] the most common presenting symptoms were syncope, palpitation, and dyspnea observed in 59.9%, 56.7%, and 56.7% of patients, respectively.

The present study showed a higher prevalence of diabetes in the study population compared to previous studies and dyspnea as a presenting complaint is also more noticed.

Seventeen patients were on beta-blockers antecedent to admission out of 50 patients, and drugs stopped 5 days before pacemaker implantation.

Degenerative complete AV nodal block occurred on 26 patients (52% of the study population), is the most common cause of pacemaker implantation in the current study followed by sinus node dysfunction in 15 (30%) patients, high-grade AV block in 5 patients (10%), and Mobitz type 2 block in 4 patients.

A study by Dwivedi *et al*.[3] did VVI pacemaker in 45 patients out of which 34 patients (75.6%) were paced because of CHB and 11 patients (24.4%) were paced for sick sinus syndrome. Kanse *et al*.[47] did a VVI pacemaker in 37 patients, and the study population consisted of 24 (64.9%) patients of AV block, 10 (27.1%) patients of SSS, and 3 (8.0%) patients of trifascicular block. de sá *et al*.[2] observed complete AV block or type 2 second-degree AV block accounted for 70% of the sample.

The right ventricular internal diameter at baseline was 2.62 ± 0.58 cm, at the end of 1 week, 1 month, and at 6 months was 2.77 ± 0.57 cm, 2.95 ± 0.57 cm, and 3.28 ± 0.66 cm, respectively. $P \leq 0.05$ achieved at 1 week, 1 month, and 6 months. A study by Dwivedi *et al*.[3] showed, RVID was 1.26 ± 0.41 cm, 144 ± 0.44 cm, and 1.50 ± and 1.77 ± 0.45 cm at baseline and at the end of 1 week, 1 month and 6 months with $P \leq 0.05$ at all levels.

In post-pacemaker patients, changes in the RV dimensions started early after 1 week and continue to increase till the 6 months, and it is one of the earliest changes in post-pacemaker patients.

The left ventricular internal diameter in systole and diastole were analyzed, mean LV diameter during systole (LVDs) at baseline was 3.57 ± 0.40 cm and at the end of 6 months was 3.91 ± 0.45 cm with $P \leq 0.05$, mean LV during diastole (LVDd) at baseline was 5.04 ± 0.30 cm and at the end of 1 month and 6 months was 5.29 ± 0.29 cm and 5.29 ± 0.29 cm, respectively, with $P \leq 0.05$ [Table 3].

The left atrial size at baseline was 3.11 ± 0.22 cm and at the end of 6 months was 3.61 ± 0.26 cm with $P \leq 0.05$. About the left-sided cardiac chambers is concerned LVDd started increasing at the end of 1 month and LVDs and left atrial size showed statistical significance at the end of 6 months.

A Study done by de sa *et al*.[2] in 20 right ventricular pacing patients (14 septal and 6 apical) who are analyzed for 2 years,

baseline LVDd was 50.25 ± 3.44 mm and at the end of 2 years was 50.55 ± 7.18 mm with P = 0.7559 which is non-significant may be attributable to higher septal pacing than apical patients. A study by Dwivedi *et al*.[3] no significant change in LVDd during the 6 months and changes in LVDs and LA dimensions significant at 6 months.

The right ventricular diastolic parameters concerned, TV-DT was 131.50 ± 11.27 ms at baseline and at the end of 1 week was 135.90 ± 13.72 ($P \leq 0.001$) and maintained at the end of 6 months.

A study by Dwivedi *et al*.[3] showed that the RV-DT showed a significant increase by 1 month ($P < 0.02$) with a mean of 153.09 ms and at 6 months was 172.90 ms.

TAPSE is a measure of systolic function of RV which was 20.50 ± 1.82 mm at baseline decreases to 18.48 ± 2.13 mm at 1 month and 17.18 ± 2.61 mm at 6 months and 28% developed RV dysfunction at 1 month, and 62% developed RV dysfunction at 6 months [Table 4].

TAPSE values are independent of hypertension and diabetic status and are no statistical significance though TAPSE at 1 month showed a trend toward significance ($P = 0.053$).

The RVET at baseline was 309.80 ± 23.85 ms and at the end of 1-month RVET decreased to 290.00 ± 16.12 ms ($P \leq 0.05$) and maintained till the end of 6 months [Table 5].

A study by Dwivedi *et al*.[3] showed the RVET decreased significantly by 6 months ($P < 0.02$) with a mean value of 280.37 ± 37.71 ms.

New tricuspid valvular regurgitation (TR) developed in 20% of patients of moderate severity at 1 month and 36% of patients developed moderate TR and 16% developed severe TR at the end of 6 months.

As the left ventricular systolic parameters is concerned mean ejection fraction at baseline was 59.66 ± 7.55%, at 1 month was 56.48 ± 6.99% (P = 0.06), and at 6 months was 51.16 ± 7.23% ($P \leq 0.001$).

New-onset LV dysfunction developed in 14% of patients and worsening of LV function in 16% of patients. Ejection fraction changes were independent and largely uniform regarding the gender hypertension and diabetic status of the study population throughout the study period.

de sá *et al*.[2] the average ejection fraction at baseline was 64.50% and at the end was 60.65%, but there was no statistical difference ($P = 0.1602$).

A study by Dwivedi *et al*.[3] showed the LV ejection fraction decreased progressively from study onset (61.82 ± 10.35%) and was statistically significant at 6 months (52.52 ± 12.1%, $P < 0.05$). A study by Silva *et al*.[48] (75 patients with RV pacemaker with LVEF> 55% and LVEDd< 53 mm) LVEF decreased from 72.6% to 69.8% ($P = 0.0025$) and LVEDD increased from 46.4 mm to 48.6 mm ($P = 0.0001$), and both changes were statistically significant with a minimum follow-up of 6 months.

Ahmed *et al*.[49] showed that a significant (>5%) decline in EF was observed on the late echocardiogram in 28 (31%) patients, in whom end-systolic volume increased.

The LVET at baseline was 304.60 ± 12.42 ms, decreased progressively to 287.20 ± 11.92 ms at the end of 1 month and 276.00 ± 13.63 ms at the end of 6 months ($P \leq 0.05$ at 1 month and 6 months).

A study by Dwivedi *et al*. showed LVET decreased from baseline (303.33 ± 37.90 ms) to 280.71 ± 37.71 ms at the end of 6 months ($P < 0.05$).

The left ventricular diastolic parameters LV-IVRT increased significantly at 1 month and 6 months ($P \leq 0.001$) while MV-DT increased significantly at 6 months ($P \leq 0.05$).

A study by Dwivedi *et al*. showed LV-IVRT increased significantly at 1 month ($P < 0.001$) and continued to increase progressively till 6 months. The mitral valve DT also increased similarly becoming statistically significant by 1 month ($P < 0.01$).

Table 1: LV-IVRT

LV IVRT	*Min–Max*	*Mean±SD*	*Difference*	*t-value*	*P-value*
Day 1	76.00–100.00	84.40 ±8.26	-	-	-
Day 7	87.00–120.00	87.20 ±9.95	−2.800	−2.645	0.09
1 month	90.00–130.00	110.70 ±11.66	−26.300	−30.180	<0.001**
6 months	110.00–140.00	126.00 ±10.45	−41.600	−44.490	<0.001**

LV-IVRT: Left ventricle isovolumic relaxation time. Significant < 0.05

Table 2: LVET

LVET	*Min–Max*	*Mean±SD*	*Difference*	*t-value*	*P-value*
Day 1	280.00–324.00	304.60 ±12.42	-	-	-
Day 7	275.00–319.00	297.20 ±12.70	7.400	19.251	0.0821
1 month	265.00–310.00	287.20 ±11.92	17.400	35.102	<0.001**
6 months	256.00–298.00	276.00 ±13.63	28.600	15.708	<0.001**

LVET: Left ventricular ejection time. Significant < 0.05

Table 3: Left ventricle dimensions

Variables	*Min–Max*	*Mean±SD*	*Difference*	*t-value*	*P-value*
LVIDd					
Day 1	4.60–5.60	5.04±0.30	-	-	-
Day 7	4.70–5.60	5.09±0.30	0.052	0.252	0.502
1 month	4.90–5.90	5.29±0.29	−0.250	−14.533	<0.001**
6 months	5.10–6.00	5.44±0.39	−0.400	−20.870	<0.001**
LVIDs	*Min–Max*	*Mean±SD*	*Difference*	*t-value*	*P-value*
Day 1	3.10–4.20	3.57±0.40	-	-	-
Day 7	3.20–3.90	3.56±0.23	0.010	0.348	0.729
1 month	3.30–4.40	3.67±0.38	0.110	0.157	0.343
6 months	3.40–4.70	3.91±0.45	−0.340	−23.338	<0.001**

LVIDs: Left ventricular internal diameter in systole, LVIDd: Left ventricular internal diameter in diastole. Significant < 0.05

Table 4: TAPSE assessment at different study periods

TAPSE	*Day 1 (%)*	*Day 7 (%)*	*1 month (%)*	*6 months (%)*
<17	0 (0)	0 (0)	14 (28)	31 (62)
>17	50 (100)	50 (100)	36 (72)	19 (38)
Total	50 (100)	50 (100)	50 (100)	50 (100)

Table 5: RVET

RVET	*Min–Max*	*Mean±SD*	*Difference*	*t-value*	*P-value*
Day 1	267.00–345.00	309.80±23.85	-	-	-
Day 7	278.00–332.00	301.80±18.07	8.000	5.739	0.059*
1 month	267.00–317.00	290.00±16.12	19.800	14.119	<0.001**
6 months	265.00–294.00	279.80±10.81	30.000	10.264	<0.001**

RVET: Right ventricular ejection time. Significant < 0.05

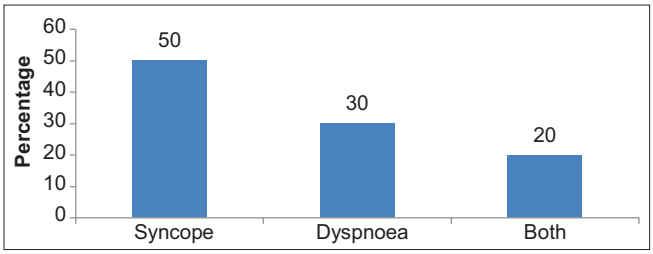

Figure 1: Symptoms of patients studied

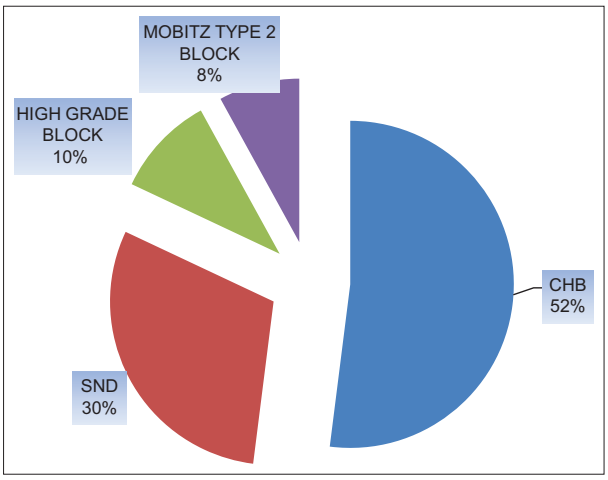

Figure 2: Electrocardiogram of the study population

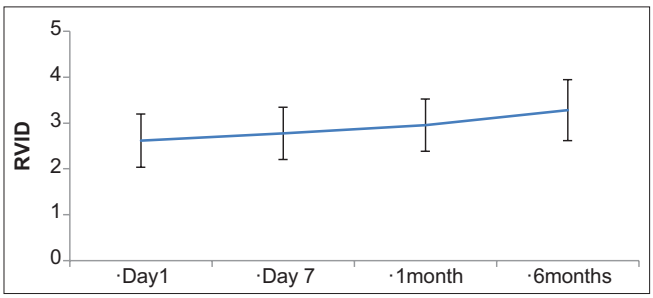

Figure 3: Right ventricle dimensions (mean) at different study periods

Intuitively, one expects a clinical benefit from the closer approximation of sinus rhythm provided by dual-chamber pacing. Maintaining the normal sequence of atrial and ventricular activation will tend to optimize ventricular filling and cardiac output. This, in turn, should improve exercise capacity and quality of life. The pacemaker syndrome, a constellation of symptoms associated with asynchronous or retrograde atrial activation from ventricular pacing, may also be prevented by dual-chamber pacing. Observational studies suggest that dual-chamber pacing reduces the risk of atrial fibrillation, stroke, and death compared with ventricular pacing.[8,50-53] If important reductions in mortality, stroke, and atrial fibrillation were reliably demonstrated, it would amply justify the increased cost of dual-chamber pacing.

Conclusion

The present study highlighting the sequence of changes in the LV and RV diastolic and systolic function in VVI paced patients**.**

It appears that changes in cardiac dimensions start appearing as early as 1 week after VVI pacing, which are limited to the RV followed by LV diastolic dimensions at 1 month and LV systolic dimensions at 6 months.

Diastolic abnormalities are first to appear which are followed by appearance of systolic abnormalities of the respective ventricles.

The right ventricular dysfunction (TAPSE <17 mm) developed in 28% at 1 month and in 62% at the end of 6 months.

The left ventricular dysfunction (EF< 50%) occurs later, LV dysfunction developed in 14%, and worsening of LV function developed in 16% of study population at the end of study, that is, 6 months.

Acknowledgments

We thank the study participants and my colleagues for their personal time and commitment and help in this study.

References

1. Ferrari AD, Borges AP, Albuquerque LC, Sussenbach CP, da Rosa PR, Piantá RM, *et al*. Cardiomyopathy induced by artificial cardiac pacing: Myth or reality sustained by evidence? Rev Bras Cir Cardiovasc 2014;29:402-13.
2. de Sá LA, Rassi S, Batista MA. Is conventional cardiac pacing harmful in patients with normal ventricular function? Arq Bras Cardiol 2013;101:545-53.
3. Dwivedi S, Bansal S, Puri A, Makharia M, Narain V, Saran R, *et al*. Diastolic and systolic right ventricular dysfunction precedes left ventricular dysfunction in patients paced from right ventricular apex. Indian Pacing Electrophysiol J 2006;6:142-52.
4. Samet P, Bernstein WH, Nathan DA, López A. Atrial contribution to cardiac output in complete heart block. Am J Cardiol 1965;16:1-10.
5. Kroetz FW, Leonard JJ, Shaver JA, Leon DF, Lancaster JF, Beamer VL. The effect of atrial contraction on left ventricular performance in valvular aortic stenosis. Circulation 1967;35:852-67.
6. Geka RK. Physiological cardiac pacing and its methods. Rinsho Kyobu Geka 1982;2:291-4.
7. Nathan AW, Davies DW. Is VVI pacing outmoded? Br Heart J 1992;67:285-8.
8. Andersen HR, Thuesen L, Bagger JP, Vesterlund T, Thomsen PE. Prospective randomised trial of atrial versus ventricular pacing in sick-sinus syndrome. Lancet 1994;344:1523-8.
9. Pehrsson SK, Åström H, Bone D. Left ventricular volumes with ventricular inhibited and atrial triggered ventricular pacing. Acta Med Scand 1983;214:305-10.
10. Sethi KK, Bajaj V, Mohan JC, Arora R, Khalilullah M. Comparison of atrial and VVI pacing modes in symptomatic sinus node dysfunction without associated tachyarrhythmias. Indian Heart J 1990;42:143-7.
11. Dalia T, Amr BS. Pacemaker indications. In: Stat Pearls. Treasure Island, FL: Stat Pearls Publishing; 2020.
12. The NASPE/BPEG Generic Pacemaker Code for Anti Bradyarrhythmia and Adaptive-Rate Pacing and Anti Tachyarrhythmia Devices Request PDF; 2020.
13. Gregoratos G, Cheitlin MD, Conill A, Epstein AE, Fellows C, Ferguson TB, *et al*. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: Executive summary-a report of the American college of cardiology/American heart association task force on practice guidelines (committee on pacemaker implantation). Circulation 1998;97:1325-35.
14. van Hemel NM, van der Wall EE. 8 October 1958, D day for the implantable pacemaker. Neth Heart J 2008;16 Suppl 1:S3-4.
15. Lamas GA, Ellenbogen KA. Evidence base for pacemaker mode selection: From physiology to randomized trials. Circulation 2004;109:443-51.
16. de Cock CC, Giudici MC, Twisk JW. Comparison of the haemodynamic

effects of right ventricular outflow-tract pacing with right ventricular apex pacing: A quantitative review. Europace 2003;5:275-8.
17. de Cock CC, Giudici MC, Twisk JW. Comparison of the haemodynamic effects of right ventricular outflow-tract pacing with right ventricular apex pacing: A quantitative review. Europace 2003;5:275-8.
18. Tantengco MV, Thomas RL, Karpawich PP. Left ventricular dysfunction after long-term right ventricular apical pacing in the young. J Am Coll Cardiol 2001;37:2093-100.
19. Nelson GS, Curry CW, Wyman BT, Kramer A, Declerck J, Talbot M, *et al*. Predictors of systolic augmentation from left ventricular preexcitation in patients with dilated cardiomyopathy and intraventricular conduction delay. Circulation 2000;101:2703-9.
20. Prinzen FW, Peschar M. Relation between the pacing induced sequence of activation and left ventricular pump function in animals. Pacing Clin Electrophysiol 2002;25:484-98.
21. Vassallo JA, Cassidy DM, Miller JM, Buxton AE, Marchlinski FE, Josephson ME. Left ventricular endocardial activation during right ventricular pacing: Effect of underlying heart disease. J Am Coll Cardiol 1986;7:1228-33.
22. Lee MA, Dae MW, Langberg JJ, Griffin JC, Chin MC, Finkbeiner WE, *et al*. Effects of long-term right ventricular apical pacing on left ventricular perfusion, innervation, function and histology. J Am Coll Cardiol 1994;24:225-32.
23. Cosedis NJ, Rud AH, Bloch TP, Leif T, Thomas MP, Thomas V, *et al*. Heart failure and echocardiographic changes during long-term follow-up of patients with sick sinus syndrome randomized to single-chamber atrial or ventricular pacing. Circulation 1998;97:987-95.
24. Nahlawi M, Waligora M, Spies SM, Bonow RO, Kadish AH, Goldberger JJ. Left ventricular function during and after right ventricular pacing. J Am Coll Cardiol 2004;44:1883-8.
25. Ebert M, Jander N, Minners J, Blum T, Doering M, Bollmann A, *et al*. long-term impact of right ventricular pacing on left ventricular systolic function in pacemaker recipients with preserved ejection fraction: Results from a large single-center registry. J Am Heart Assoc 2016;5:e003485.
26. Sisu RC, Cinteza M, Vinereanu D. Interventricular septal or standard apical pacing in pacing dependent patients: Still a dilemma? Maedica (Bucur) 2010;5:193-200.
27. Leclercq C, Gras D, Le Helloco A, Nicol L, Mabo P, Daubert C. Hemodynamic importance of preserving the normal sequence of ventricular activation in permanent cardiac pacing. Am Heart J 1995;129:1133-41.
28. de Sá LA, Rassi S, Batista MA. Is conventional cardiac pacing harmful in patients with normal ventricular function? Arq Bras Cardiol 2013;101:545-53.
29. Faerestrand S, Ohm OJ. A time-related study by Doppler and M-mode echocardiography of hemodynamics, heart size, and AV valvular function during activity-sensing rate-responsive ventricular pacing. Pacing Clin Electrophysiol 1987;10:507-18.
30. Leclercq C, Gras D, Le Helloco A, Nicol L, Mabo P, Daubert C. Hemodynamic importance of preserving the normal sequence of ventricular activation in permanent cardiac pacing. Am Heart J 1995;129:1133-41.
31. Rosenqvist M, Isaaz K, Botvinick EH, Dae MW, Cockrell J, Abbott JA, *et al*. Relative importance of activation sequence compared to atrioventricular synchrony in left ventricular function. Am J Cardiol 1991;67:148-56.
32. Wilkoff BL, Cook JR, Epstein AE, Greene HL, Hallstrom AP, Hsia H, *et al*. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: The dual chamber and VVI implantable defibrillator (DAVID) trial. JAMA 2002;288:3115-23.
33. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, *et al*. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877-83.
34. Lamas GA, Lee KL, Sweeney MO, Silverman R, Leon A, Yee R, *et al*. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. N Engl J Med 2002;346:1854-62.
35. Barold SS, Herweg B, Sweeney MO. Minimizing right ventricular pacing. Am J Cardiol 2005;95:966-9.
36. Barold SS. Adverse effects of ventricular desynchronization induced by long-term right ventricular pacing. J Am Coll Cardiol 2003;42:624-6.
37. Brunner M, Olschewski M, Geibel A, Bode C, Zehender M. Long-term survival after pacemaker implantation. Prognostic importance of gender and baseline patient characteristics. Eur Heart J 2004;25:88-95.
38. Sweeney MO, Hellkamp AS, Ellenbogen KA, Greenspon AJ, Freedman RA, Lee KL, *et al*. Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in a clinical trial of pacemaker therapy for sinus node dysfunction. Circulation 2003;107:2932-7.
39. Simantirakis EN, Vardakis KE, Kochiadakis GE, Manios EG, Igoumenidis NE, Brignole M, *et al*. Left ventricular mechanics during right ventricular apical or left ventricular-based pacing in patients with chronic atrial fibrillation after atrioventricular junction ablation. J Am Coll Cardiol 2004;43:1013-8.
40. Lupi G, Sassone B, Badano L, Peraldo C, Gaddi O, Sitges M, *et al*. effects of right ventricular pacing on intra-left ventricular electromechanical activation in patients with native narrow QRS. Am J Cardiol 2006;98:219-22.
41. Xiao HB, Brecker SJ, Gibson DG. Differing effects of right ventricular pacing and left bundle branch block on left ventricular function. Br Heart J 1993;69:166-73.
42. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, *et al*. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American society of echocardiography and the European association of cardiovascular imaging. J Am Soc Echocardiogr 2015;28:1-39.e14.
43. Lee MA, Dae MW, Langberg JJ, Griffin JC, Chin MC, Finkbeiner WE, *et al*. Effects of long-term right ventricular apical pacing on left ventricular perfusion, innervation, function and histology. J Am Coll Cardiol 1994;24:225-32.
44. Marketou ME, Simantirakis EN, Prassopoulos VK, Chrysostomakis SI, Velidaki AA, Karkavitsas NS, *et al*. Assessment of myocardial adrenergic innervation in patients with sick sinus syndrome: Effect of asynchronous ventricular activation from ventricular apical stimulation. Heart 2002;88:255-9.
45. Nakata T, Miyamoto K, Doi A, Sasao H, Wakabayashi T, Kobayashi H, *et al*. Cardiac death prediction and impaired cardiac sympathetic innervation assessed by MIBG in patients with failing and nonfailing hearts. J Nucl Cardiol 1998;5:579-90.
46. Johnson M, Hunt W, Charlotte H, Capener D, Charlie E, Robin C, *et al*. Tricuspid deceleration time predicts right atrial pressure in patients with suspected pulmonary hypertension: Preliminary experience with cardiac magnetic resonance imaging. Eur Respir J 2015;46 Suppl 59:PA3749. Available from: https://www.erj.ersjournals. [Last accessed on 2020 Apr 14].
47. Kanse VY, Chongtham DS, Salam KS, Nemichandra SC, Upretti S, Singh SD. Clinical profiles and outcomes of patients undergoing pacemaker implantation. J Med Soc 2015;29:40.
48. Silva RT, Filho MM, de Oliveira JC, de Lima CE, Martins DG, Guirao CI, *et al*. Ventricular remodeling in right ventricular apical pacing. Arq Bras Cardiol 2007;88:152-8.
49. Ahmed M, Gorcsan J, Marek J, Ryo K, Haugaa K, Ludwig DR, *et al*. Right ventricular apical pacing-induced left ventricular dyssynchrony is associated with a subsequent decline in ejection fraction. Heart Rhythm 2014;11:602-8.
50. Connolly SJ, Kerr C, Gent M, Yusuf S. Dual-chamber versus ventricular pacing. Circulation 1996;94:578-83.
51. Markewitz A, Schad N, Hemmer W, Bernheim C, Ciavolella M, Weinhold C. What is the most appropriate stimulation mode in patients with sinus node dysfunction? Pacing Clin Electrophysiol 1986;9:1115-20.
52. Morbidity and Mortality of Patients with Sinus Node Disease: Comparative Effects of Atrial and Ventricular Pacing-ZANINI-1990-Pacing and Clinical Electrophysiology-Wiley Online Library; 2020.
53. Zanini R, Facchinetti AI, Gallo G, Cazzamalli L, Bonandi L, Cas LD. Morbidity and mortality of patients with sinus node disease: Comparative effects of atrial and ventricular pacing. Pacing Clin Electrophysiol 1990;13:2076-9.