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ORIGINAL ARTICLE

Comparison of early effects of right ventricular apical pacing on left ventricular functions in single and dual chamber pacemakers



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KEYWORDS

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Abstract Objectives: Our study aimed to demonstrate the early negative impact of right ventricular apical pacing induced by single (VVI) and dual chamber (DDD) pacemakers on LV functions in patients with preserved EF. And to assess that single brain natriuretic peptide (BNP) after 2 months of implantation is correlated to ventricular dyssynchrony.

Methods: 40 patients with implanted VVI and DDD pacemakers were examined before implantation and again after 2 and 6 months of implantation for BNP, left ventricular (LV) systolic and diastolic functions by echocardiography and pulsed tissue Doppler. After 6 months, patients with DDD pacemakers were crossed over to VVI mode of pacing for 2 weeks with lower rate programmed to 60 beat per minute then sample for BNP was collected again.

Results: There was no statistically significant difference in LV systolic and diastolic functions except for myocardial performance index (MPI) with (P value of 0.03). Mean BNP level in VVI pacing was higher than DDD pacing after two months with P value = 0.001 while comparison after 6 months showed P value = 0.023. There was a statistically significant difference between both groups in results of aortic preejection delay (APED) (P value of <0.05). BNP was correlated to APED ($r = 0.651$ and P value = 0.001) and pacing percentage ($r = 0.687$ and P value = 0.00).

Conclusion: Loss of atrioventricular synchrony in VVI mode leads to a significant difference in LV dyssynchrony between both groups. BNP level is correlated to LV dyssynchrony and pacing percentage.

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1. Introduction

The cardiac pacing at any point of the ventricle alters the natural heart activation and contraction pattern, as stimulus conduction velocity is slower across ventricular myocardium,

when compared to that resulting from the specialized His-Purkinje system.^{1,2}

Right ventricular apical pacing can induce both interventricular dyssynchrony (between the right ventricle (RV) and the left ventricle (LV)), as well as intraventricular dyssynchrony (within the LV).³ It has been demonstrated that the presence of ventricular dyssynchrony is associated with an increased risk of cardiac morbidity⁴ and mortality⁵ in heart failure patients. In addition, it has been suggested that the presence of mechanical dyssynchrony after long-term RV apical pacing is associated with reduced LV systolic function and deterioration in functional capacity.³

However, there are only a few studies that have demonstrated a direct relation between (pacing-induced) ventricular dyssynchrony and clinical heart failure. This suggests that an abnormal activation pattern (left bundle branch block during RV apical pacing) or ventricular dyssynchrony may be directly related to a deterioration of LV function. Therefore, assessment of ventricular dyssynchrony may provide important information in patients with permanent RV apical pacing.^{6,7}

2. Subjects and methods

The study was carried out during the period between April 2012 and November 2013 and included 40 patients with implantation of single and dual chamber permanent pacemakers at the electrophysiology unit of national heart institute. Patients were enrolled into 2 groups, **Group A:** 20 patients with implanted single chamber pacemaker right ventricular pacing (VVI), **Group B:** 20 patients with implanted Dual chamber pacemaker (DDD).

This study compared the early effects of right ventricular (RV) apical pacing on LV functions in VVI and DDD pacemakers using echocardiographically determined parameters of systolic and diastolic functions. Also we assessed if brain natriuretic peptide (BNP) after 2 months of implantation is correlated to ventricular dyssynchrony in different cardiac pacing mode.

2.1. Inclusion criteria

The inclusion criteria are adult patients with age less than 75 years with indication for permanent pacing, patients with normal structural hearts and normal left ventricular functions, body mass index less than 30 Kg/m², and patients enrolled in the study after 2 months of implantation if they have more than 60% pacing dependence and ventricular lead should be in the right ventricular apex.

2.2. Exclusion criteria

The exclusion criteria are patients with poor echo window, patients with symptoms of overt heart failure, previous cardiac surgery or structural heart diseases (eg. Dilated cardiomyopathy, valvular heart diseases, congenital cardiac anomalies and prosthetic valves). Also we excluded patients with documented chronic heart dysrhythmias, patients with previous coronary artery disease detected by evidence of LV regional wall motion abnormalities at the echocardiogram or pathological Q waves in electrocardiogram, or any form of acute coronary syndrome within the past 4 weeks, patients with history of chronic

obstructive lung disease, pulmonary hypertension or recent pulmonary embolism, renal impairment, pregnancy, and patients with terminal co-morbidities such as end stage malignancy, end stage renal or liver diseases.

After written informed consent and full history taking with history of the medications, complete general and local examinations were done for all patients. Patients were also subjected to measurements of QRS duration, Chest X-ray to verify the position of the ventricular lead-electrode, Urea and creatinine level and Pacemaker analysis.

BNP samples were obtained after 2 months and 6 months by direct venipuncture of an antecubital vein after the patient had been placed in supine position for at least 15 min. Venous blood sample was collected in tubes containing potassium EDTA. After 6 months, patients in group B were crossed over to VVI mode of pacing by programing for a period of 2 weeks with lower rate programed to 60 beat per minute then a venous blood sample was collected again for BNP to test the effect of right apical pacing in VVI mode on the heart. These patients were programed again to DDD mode after taking the blood sample.

Echocardiographic studies were done using a commercially available system (Samsung Medison EKO 7, Samsung Medison Building, 1003 Daechi-dong Gangnam-gu, Seoul 135-280 Korea) with a 2.5–3.5 MHz transducer. Patients were examined before implantation and again after 2 months and 6 months of implantation for left ventricular dimension, left ventricular systolic and diastolic functions. Myocardial performance index (MPI). Pulsed tissue Doppler imaging (TDI) was used to obtain septal and lateral velocities for both E and S waves.

Mechanical dyssynchrony was assessed after 2 months and 6 months by the following defined conventional parameters:

- (i) Aortic pre-ejection delay (APED) by pulsed wave (PW)-Doppler is measured between the onset of QRS complex and the beginning of the aortic flow by pulsed wave Doppler. Intraventricular dyssynchrony is defined by an APED of 140 ms or more.^{8,9}
- (ii) Interventricular mechanical delay (IVMD) by PW-Doppler. To calculate the IVMD, time from onset of the QRS to onset of pulmonary flow was measured at the parasternal short-axis view, using pulsed-wave Doppler and the difference between it and APED resulted in the IVMD. Interventricular dyssynchrony is defined by an IVMD of 40 ms.^{8,9}
- (iii) Septal-posterior wall motion delay (SPWMD) by identifying the time delay from peak inward septal motion to peak inward posterior wall. Intraventricular dyssynchrony is defined by an SPWMD of 130 ms.^{8,9}

3. Statistical analysis

The collected data were tabulated and statistically analyzed using SPSS version 20.0 for Windows (SPSS Inc, Chicago, IL, USA). Comparisons between the groups were performed using the unpaired Student's *t* test. Comparisons within the group were performed using the paired Student's *t* test. A probability value of 0.05 was considered statistically significant. DDD pacing as compared with VVI pacing, was assessed using one-way analysis of variance (ANOVA) for repeated measures. Partial correlations were used to measure the linear

association between variables while controlling the effects of one or more additional variables and multiple linear regression analysis were performed to provide regression analysis and analysis of variance for one dependent variable as BNP levels and other variables.

4. Results

No significant difference between both groups regarding baseline characteristics of the study is outlined in Table 1.

In our study there was no statistically significant difference in left ventricular EF (P value >0.19), end systolic (P value >0.069) and end diastolic (P value >0.078) internal dimensions between both groups over time as shown in Table 2. Also there was no statistically significant difference for septal and lateral E and S waves by pulsed tissue Doppler in both groups

over time (P value >0.05). Using paired sample t test, there was a significant difference between Septal S wave velocity when compared to lateral S wave velocity within each group after 6 months with P value of 0.005 for group A and P value of 0.001 for group B.

Mean BNP level in VVI pacing (group A) was higher than DDD pacing (group B), after two months follow up group A showed a mean BNP of 196.5 ± 123 pg/dl compared to 79.35 ± 65.36 pg/dl in group B with a significant difference between both groups (P value = 0.001). Comparison of both groups after 6 months showed a mean BNP of 200.85 ± 106.6 pg/dl in group A and a mean of 121.5 ± 105.15 pg/dl in group B with a statistically significant difference and P value = 0.023. There was a statically significant difference within repeated measurements of BNP level in group B after converting pacing from dual chamber (DDD) to single chamber (VVI) for 2 weeks with a mean of 172 ± 90 and P value of 0.001.

Table 1 Baseline characteristics of studied groups.

Variables	Group A ($n = 20$)	Group B ($n = 20$)	P value
Age in years (m + SD)	(60.75 \pm 7.41)	56.15 \pm 7.49	0.58
Body mass index in kg/m ² (m + SD)	(26.35 \pm 3.16)	25.94 \pm 4.11	0.72
Male n (%)	6(30)	11(55)	0.11
Female n (%)	14(70)	9(45)	
Smoking n (%)	6(30)	11(55)	0.32
Diabetes n (%)	4(20)	3(15)	0.67
Hypertension n (%)	10(50)	10(50)	1

m = mean, SD = standard deviation, % is the percentage within the group; n means number of patients within the group.

Table 2 Comparison between studied groups regarding echocardiographic data.

Variables		At baseline		At 2 months		At 6 months		P value For repeated measure over time
		Mean \pm SD	P value	Mean \pm SD	P value	Mean \pm SD	P value	
LVEDD (mm)	Group A	51 \pm 5.96	> 0.05	52 \pm 4.87	> 0.05	53 \pm 6.98	> 0.05	> 0.05
	Group B	51 \pm 5.2		53 \pm 4.1		49 \pm 4.3		
LVESD (mm)	Group A	30 \pm 6.7	> 0.05	32 \pm 5.7	> 0.05	33 \pm 3.9	> 0.05	> 0.05
	Group B	29 \pm 4.3		28 \pm 6.1		31 \pm 4.8		
EF	Group A	61.5% \pm 4.8	> 0.05	59.4% \pm 4.7	> 0.05	56.4% \pm 5.6	> 0.05	> 0.05
	Group B	63.8% \pm 5.5		62.4% \pm 4.9		60.9% \pm 6.6		
DT (ms)	Group A	193.4 \pm 19.7	> 0.05	219.5 \pm 25.1	> 0.05	244.6 \pm 22.8	0.01*	0.01*
	Group B	185.9 \pm 30.8		204.2 \pm 25.9		214.4 \pm 29.1		
MPI	Group A	0.36 \pm 0.06	> 0.05	0.52 \pm 0.13	> 0.05	0.60 \pm 0.11	0.03*	0.03*
	Group B	0.38 \pm 0.05		0.47 \pm 0.10		0.53 \pm 0.06		
sE (cm/s)	Group A	10.5 \pm 2.2	> 0.05	9.4 \pm 1.7	> 0.05	9.2 \pm 2.1	> 0.05	> 0.05
	Group B	11.9 \pm 2.7		10.7 \pm 3.1		10.4 \pm 3		
IE (cm/s)	Group A	12.6 \pm 1.3	> 0.05	11 \pm 1.8	> 0.05	10.3 \pm 2.2	> 0.05	> 0.05
	Group B	13.7 \pm 2.6		12.2 \pm 3.1		11.5 \pm 3.3		
sS (cm/s)	Group A	10.8 \pm 1.7	> 0.05	9.3 \pm 1.8	> 0.05	8.2 \pm 1.9	> 0.05	> 0.05
	Group B	11.6 \pm 1.9		10.3 \pm 1.7		9.2 \pm 2.4		
IS (cm/s)	Group A	11.3 \pm 1.6	> 0.05	9.7 \pm 2.1	> 0.05	8.9 \pm 2.1	> 0.05	> 0.05
	Group B	12.4 \pm 2		11.1 \pm 2.6		10.3 \pm 2.6		

* Means significant P value. LVEDD = left ventricular internal end diastolic dimension, LVESD = left ventricular internal end systolic dimension. EF = ejection fraction, DT = deceleration time and MPI = Myocardial performance index, sE = septal E wave velocity, IE = lateral E wave velocity. sS = septal S wave velocity, IS = lateral S wave velocity. (mm) = millimeter, (ms) = milliseconds and SD = standard deviation.

Table 3 Comparison between studied groups regarding BNP and parameters of dyssynchrony.

Variables		At 2 months		At 6 months		<i>P</i> value for repeated values over time
		Mean \pm SD	<i>P</i> value	Mean \pm SD	<i>P</i> value	
BNP	Group A	196.5 \pm 123	0.001	200.85 \pm 106.6	0.23	0.048
	Group B	79.35 \pm 65.36		121.5 \pm 105.15		
SP (ms)	Group A	85.6 \pm 33.2	> 0.05	106 \pm 37.2	> 0.05	> 0.05
	Group B	108 \pm 43.6		114.4 \pm 46.2		
APED (ms)	Group A	145.3 \pm 9.7	0.001*	158.3 \pm 25.7	0.026*	0.03*
	Group B	113 \pm 27.8		140.7 \pm 22.3		
IVMD (ms)	Group A	35 \pm 8.3	> 0.05	41.6 \pm 9.8	> 0.05	> 0.05
	Group B	36.6 \pm 12.8		39.6 \pm 11.5		

* Means significant *P* value, SD = standard deviation, ms = milliseconds, SP = septal to posterior wall motion delay, APED = aortic preejection delay, IVMD = interventricular mechanical delay and RD = radial time to peak strain difference.

Regarding ventricular dyssynchrony, our results showed that SPWMD values at 2 months were 85.6 ± 33.2 ms for group A and 108 ± 43.6 ms for group B while values at 6 months were 106 ± 37.2 ms for group A and 114.4 ± 46.2 ms for group B. There were no statistically significant differences between both groups (*P* value of 0.22 at 2 months and 0.53 at 6 months). IVMD values at 2 months were 35 ± 8.3 for group A and 36.6 ± 12.8 for group B while values at 6 months were 41.6 ± 9.8 for group A and 39.6 ± 11.5 for group B. There were no statistically significant differences between both groups (*P* value of 0.54 at 2 months and 0.64 at 6 months). In contrast a statistically significant difference between both groups appeared in results of APED (*P* value of 0.001 at 2 months and 0.026 at 6 months intervals) as shown in Table 3.

Our results showed a statistically significant positive correlation between the BNP level and pacing percentage ($r = 0.687$ and *P* value = 0.00) as shown in Fig. 1, QRS duration ($r = 0.42$ and *P* value = 0.01), and APED ($r = 0.651$ and *P* value = 0.001) as shown in Fig. 2. At multiple linear regression analysis, the pacing percentage and APED remained only significant and independent predictor of BNP levels, even after adjustment for age and LVEF with *P* value of 0.007 for APED and *P* value of 0.0001 for pacing percentage. So we can infer

that the higher the percentage of pacing and the longer the APED, the higher the BNP level.

5. Discussion

Long-term effects of right ventricular apical pacing have been studied, and not much information is available on the acute and early effects of right ventricular pacing on left ventricle function and left ventricle dyssynchrony.¹⁰ Our study aimed to demonstrate the early negative impact of right ventricular apical pacing induced by single and dual chamber pacemakers on LV systolic and diastolic functions in patients with preserved EF.

5.1. Effect on LV systolic functions

Left ventricular EF is a surrogate parameter that describes myocardial pump function. Even if contractility is reduced, compensatory mechanisms (ie, ventricular dilatation, geometry changes) can still assure that stroke volume remains normal at least at rest.¹¹ In our study there was no statistically significant difference within repeated measurements of left ventricular EF, systolic and diastolic internal dimensions in both group

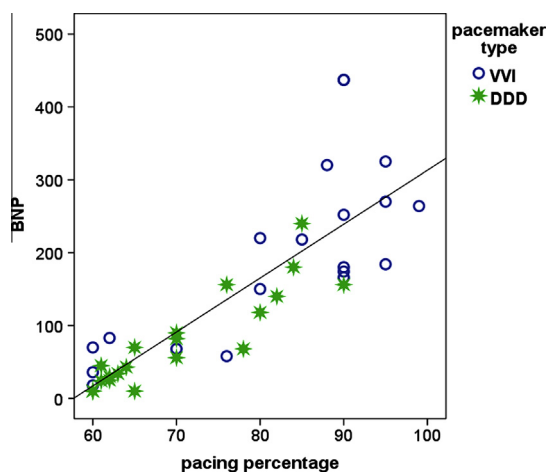


Figure 1 Scatter plot graph showed the relationship between BNP and pacing percentage ($r = 0.687$, $P < 0.0001$).

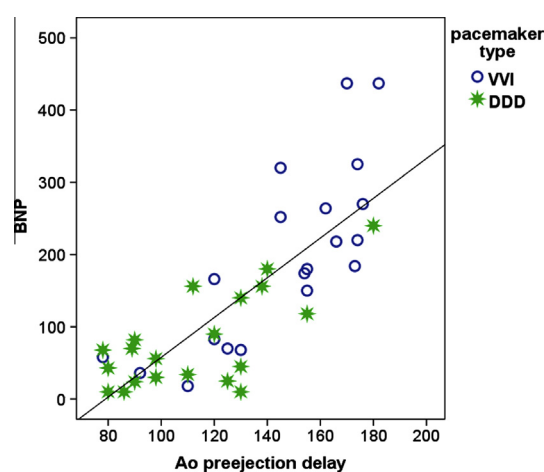


Figure 2 Scatter plot graph showed the relationship between BNP and aortic preejection delay ($r = 0.651$, $P < 0.0001$).

over time (P value >0.05) and this is due to short term of follow up. Our results were consistent with that of previous studies.^{12–14} On the other hand, likewise, Fehrsson et al. found greater resting LV volumes and lesser EF in patients paced on VVI rather than with physiological pacing mode.¹⁵ Dwivedi et al. studied 48 patients with VVI pacing and found that left ventricular EF decreased progressively from baseline ($61.82 \pm 10.36\%$) and was statistically significant at 6 months ($52.52 \pm 12.11\%$, $P < 0.05$). The cardiac dimensions, left ventricular end diastolic dimension and left ventricular end systolic dimension increased significantly by 6 months over their corresponding baseline values ($P < 0.05$).¹⁶ However the last aforementioned study used a higher percentage of pacing $>90\%$ in all patients and this may be the cause of early effects on left ventricular dimension and systolic functions.

S wave by pulsed tissue Doppler is another predictor of systolic dysfunction.¹⁷ In our study there was no statistically significant difference for septal and lateral S waves by pulsed tissue Doppler in both groups over time (P value >0.05). Our results were the same as Kojuri and colleagues in the above mentioned study. They found no significant difference in comparing EF and S wave by pulsed tissue Doppler in DDD pacing mode, VDD and VVI pacing modes with P value of 0.45 in spite that they had selected patients with more than 90% pacing percentage.¹⁷

Using paired sample t test, there was a significant difference between septal S wave velocity when compared to lateral S wave velocity within each group after 6 months with P value of 0.005 for group A and P value of 0.001 for group B. Ventricular pacing reduces mechanical work in the septum during RV apical pacing by 50% and increases it by 50% in the LV free wall. Under RV apical pacing, the LV septal wall was activated early and forced the LV lateral wall to be pre-stretched at that time. Then when all regions have been activated, the fiber of the LV lateral wall was longer than fibers in the septal wall. Therefore, RV apical pacing caused the difference of local preload between LV septal and lateral walls. By virtue of a local “Frank–Starling” relation, the later activated regions are stronger and shorten more during the ejection phase. Therefore, the regional differences in contraction pattern during ventricular pacing can be regarded as differences in effective local preload.¹⁸

5.2. Effect on LV diastolic functions

We did not find any statistically significant difference between both groups regarding septal and lateral E' waves by pulsed tissue Doppler. This was the same as previous studies; they also found no significant changes in diastolic function. Naegeli et al. in single-blind, randomized crossover study by evaluating the impact of DDD(R) versus VVI(R) mode on objective and functional parameters. They found no significant changes in transmitral flow propagation rate and the E to E' ratio.¹⁹ Kojuri and colleagues also found no significant changes in E' and A' by pulsed tissue Doppler between DDD and VVI.¹⁷

In our study mitral deceleration time showed a statistically significant difference between both groups only at 6 months (P value of 0.01). Doppler patterns of mitral inflow reflect the pressure gradient between the left atrium and LV, transmitral velocities are directly related to left atrial pressure (preload)

and independently and inversely related to ventricular relaxation. Because mitral inflow patterns are highly sensitive to preload and can change dramatically as diastolic dysfunction progresses, the use of mitral valve inflow patterns to assess diastolic function remains limited. Tissue Doppler assessment of diastolic function is less load dependent than that provided by standard Doppler techniques. Unlike conventional mitral inflow patterns, E' is resistant to changes in filling pressure.²⁰

5.3. Effect on global LV systolic and diastolic functions

Our findings showed increase in myocardial performance index (MPI) in both groups with statistically significant difference between both groups at 6 months (P value of 0.03). MPI incorporates both systolic and diastolic aspects of function and have also been shown to correlate well with known invasive indexes of LV systolic and diastolic functions.²¹ Our data were consistent with previous observations and suggested that LV dyssynchrony was implicated in deteriorating ventricular function in single chamber pacing.^{17,22} This also can be attributed to the shorter LV ejection interval.²³ Byung et al. evaluated 40 patients with sick sinus syndrome before and after (12 months) single-chamber ventricular pacemaker implantation (VVI). MPI significantly increased after 12 months of implantation.²⁴ Burn et al. found that RV paced patients had significantly longer isovolumic contraction times, which is likely a consequence of slower pressure development during a dyssynchronous LV contraction. Longer isovolumic contraction limits the time available for adequate systolic ejection and diastolic filling. Heart rates were also slightly higher in paced patients, further shortening ejection and filling times. This may be an important mechanism in reducing LV function in RV paced patients.²⁵

5.4. Effect on BNP level and LV dyssynchrony

This study showed that mean BNP level in VVI pacing (group A) was higher than DDD pacing (group B), after two months follow up with highly significant difference between both groups (P value = 0.001). Comparison of both groups for BNP level after 6 month showed a statistically significant difference and P value = 0.023.

Our results were concordant with previous studies, Nikoo MH et al., who compared the effects of right ventricular septal versus apical pacing on plasma natriuretic peptide levels in VVI and DDD pacemakers showed that despite the increase in BNP levels in patients with the VVI mode, compared with those with the DDD(R)/VDD mode ($P = 0.02$), the pacing sites had no effect on BNP levels, irrespective of the pacing mode. They concluded that hemodynamic improvement could be substantially influenced by pacing mode, more than by pacing site.²⁶ Kojuri and colleagues found that level of pro-BNP is lower in double chamber pacing in comparison with single chamber pacing. Therefore, it seems that dual chamber pacing causes less LV dysfunction.¹⁷

In this study DDD pacemakers were crossed over to VVI mode of pacing by programing for a period of 2 weeks with lower rate programed to 60 beat per minute. We found a statistically significant difference within repeated measurements of BNP level in group B after converting pacing from DDD to VVI for 2 weeks with a mean of 172 ± 90 and P value of

0.001. The increased BNP level in our crossover design suggests the loss of atrioventricular synchrony while on VVI stimulation is directly responsible for the increased levels of natriuretic peptides, most likely as a result of increased atrial and ventricular wall stretch and pressure.²⁷

This cross over was concordant with results from Naegeli et al., who showed single-blind randomized crossover study by evaluating the impact of DDD(R)/VDD versus VVI(R) mode on objective and functional parameters. They found that patients experience a highly significant; two- to threefold increase in BNP and NT-pro BNP levels during VVI(R) pacing compared with synchronized atrioventricular pacing with DDD(R)/VDD pacing with $P < 0.001$.¹⁹ Dual chamber pacing preserves atrial-ventricular synchrony and decreases ventricular end-diastolic pressure and increases cardiac output.^{4,22}

Regarding ventricular dyssynchrony, our results showed no statistically significant difference between both groups regarding SPWD and IVMD in both groups over time, (P value of > 0.05). In contrast a statistically significant difference between both groups appeared in results of APED (P value of < 0.05). We believe that the difference was caused by loss of atrioventricular synchrony and a larger part by ventricular pacing percentage.

Sá et al. observed the prolonging of APED in a small group of patients, who were Chagasic with normal left ventricular EF, throughout an eight-month period. The authors observed that although the APED measurements did not reach the cutoff required for the diagnosis of ventricular dyssynchrony (≥ 140 ms), this was the only assessed ventricular dyssynchrony measure that showed increase throughout follow-up.²⁸

Our results showed a significant correlation between the BNP level and pacing percentage, QRS duration and APED. At multiple linear regression analysis, the pacing percentage and APED remained only significant and independent predictor of BNP levels, even after adjustment for age and LVEF with P value of 0.007 for APED and P value of 0.0001 for pacing percentage. So we can infer that the higher the percentage of pacing and the longer the APED, the higher the BNP level. Results of the DAVID trial revealed that RV paced patients with LV dysfunction, requiring a defibrillator, who were actively paced in DDDR-70 mode had a 60% greater risk for hospitalization or death than patients who received minimal back-up pacing in VVI-40 mode.²⁹ From this we can infer the strong relationship between pacing percentage and BNP increment.

The correlation between QRS duration and BNP in our study is explained by a recent study of Chen et al. who found that prolonged paced QRS could be a useful predictor to identify patients who are at risk of heart failure events during right ventricular apical pacing.³⁰ Abreu CD et al., demonstrated significant correlation between the APED and BNP ($r = 0.38$, $P < 0.0001$), regardless of LVEF and age.³¹ The studies MOST,³² DAVID²⁹ and MADIT II³³, in turn, indicated that ventricular dyssynchrony can create an anatomofunctional substrate capable of impairing heart function in the long term, by observing an increase in the risk of atrial fibrillation, mitral regurgitation and hospital admissions due to heart failure in patients with a high percentage of right ventricular paced beats, particularly those with ventricular dysfunction prior to the implant.³⁴

6. Limitations

The present analysis focuses on the short-term impact of right ventricular apical pacing on LV functions and LV dyssynchrony and does not provide information about the long-term consequences. However, previous studies have demonstrated a close link between ventricular dyssynchrony and long-term clinical outcome.^{33,29,32,12,14} We considered 6 month follow up to be sufficient to detect relevant changes in natriuretic peptide however levels have been found after even shorter time periods when used in other clinical settings.^{17,19,26} We could not fully achieve the blinding of the echocardiographer because the additional lead was visible in the right atrium and the difference between VVI pacing and AV synchrony was also apparent. Finally, the number of patients was relatively small in this study and the data resulting from these analyses should be considered as preliminary observations.

7. Conclusion

We concluded that both VVI and DDD pacing modes have the same effect on LV functions. The only difference is the loss of atrioventricular synchrony in VVI mode which leads to significant difference in LV dyssynchrony between both groups appeared in the results of APED. Also we find that BNP level is correlated to APED and pacing percentage and can predict LV dyssynchrony in RV paced patient which occurs early than LV dysfunction.

Conflict of interest

None declared.

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