Impact of Right Ventricular Pacing on Right Ventricular Function

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Background: *The benefits of right ventricular pacing in patients with symptomatic bradycardia are well recognized. Currently, left ventricular (LV) function after cardiac pacing has already been extensively investigated. However, existing data on right ventricular (RV) function in these patients is extremely limited.*
Material and Method: *To test this, records of RV and tricuspid valve function of patients with a pacemaker measured at least a year after implantation were reviewed for a prevalence of RV dysfunction. The patients were also divided into those with and without RV dysfunction. Factors affecting the two groups were evaluated.*
Results: *RV dysfunction and moderate to severe tricuspid valve regurgitation were found in approximately 4% and 21% respectively in cardiac pacing patients with mean implantation duration of 6.4 years. Compared to normal RV function, factors presumed to affect on RV dysfunction including site of pacing, pacing mode and percentage of ventricular pacing were not significantly different (p = 0.54, 0.37 and 0.12 respectively).*
Conclusion: *Based on these data, the prevalence of right ventricular dysfunction appears to be infrequent and factors that were assumed as contributors to LV dysfunction failed to show significant contributions to RV dysfunction.*

Keywords: *Right ventricular pacing, Right ventricular dysfunction, Tricuspid valve regurgitation*

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Right ventricular (RV) pacing is the treatment of choice for patients with sick sinus syndrome and atrioventricular disturbance, as it is very effective, safe and easy to conduct[1]. However, there are some uncertain detrimental effects related to cardiac pacing. Left ventricular dysfunction is a major concern that can occur from nonphysiologic stimuli[2]. Direct electrical activation of the right ventricle can induce dyssynchronous ventricular contraction resulting in a decreased stroke volume and abnormal left ventricular relaxation[3]. Furthermore, the pacemaker lead can cause tricuspid valve regurgitation. Mechanisms of pacemaker related tricuspid regurgitation (TR) include leaflet laceration or perforation, interference of leaflet mobility[4] and adhesions from scar tissue causing entrapment of the leaflets[5]. Apart from these controversial issues, right ventricular function which is also crucial for predicting exercise tolerance has not been extensively investigated, in particular after ventricular pacing.

The main purpose of the present study was to assess the prevalence of RV dysfunction and tricuspid regurgitation in patients with RV pacing. Another purpose was to assess factors influencing the RV function in these particular patients.

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Material and Method

Participants

The present study was approved by the Central Chest Institute of Thailand Ethics Committee. This was a retrospective study. All patients attending the Pace Clinic at the Central Chest Institute of Thailand (CCIT) from July 1st to September 30th 2011 were recruited. The inclusion criterion of the samples is the patients who had permanent pacemakers implanted for at least a year with a history of either sick sinus syndrome or atrioventricular disturbance.

The patients with cardiac resynchronization therapy (CRT) or automatic implantable cardioverter defibrillator (AICD) implanted and no right ventricular or tricuspid valve function recorded were excluded. The patients' medical records, particularly the records of transthoracic echocardiography at least a year after implantation were reviewed. All baseline demographic data, clinical information, echocardiographic results and pacing parameters including duration of implantation, mode and percentage of pacing and implanted lead site were recorded.

Echocardiographic measurement

Transthoracic echocardiography was evaluated using a Philips iE33 x MATRIX ultrasonography machine and a 3.5 MHz transducer. Standard echocardiogram technique was used. This involved obtaining two-dimensional (2-D) mode, M-mode and Doppler technique in the parasternal long axis, parasternal short axis, 4-chamber and 5-chanber views to evaluate for standard parameters. Tissue Doppler imaging (TDI) measuring the velocity of myocardial motion was used to identify left ventricular diastolic function and right ventricular systolic function. Tricuspid valve regurgitation was evaluated with Color Doppler mode and graded in severity as trace, mild, moderate or severe.

RV systolic function was evaluated using two parameters; Tricuspid Annular Plane Systolic Excursion (TAPSE) or Tricuspid Annular Motion (TAM) and tissue Doppler-derived tricuspid lateral annular systolic velocity (S'). RV dysfunction was indicated when TAPSE was less than 16 mm and S' velocity was less than 10 cm/s.

Statistical analysis

Descriptive analyses were employed. Baseline clinical and echocardiographic data were compared with the recent data. Mann-Whitney U-test statistics for continuous variables and the χ^2 test and Fishers' exact test statistics for categorical variables were used. A 2-tailed p-value < 0.05 was considered statistically significant. Analyses were performed using SPSS (SPSS, Inc, Chicago, Illinois).

Results

Patient demographic parameters

There were 418 patients recruited and 96 patients met entry criteria. Forty-five percent of the patients were male with a mean age of 68 years. Of these patients, 69%, 19% and 37% had hypertension, diabetes and dyslipidemia respectively. Coronary artery disease and atrial fibrillation were also found in approximately 6%.

Complete heart block was the major cause of pacemaker implantation (64%), followed by sick sinus syndrome (36%). Mean duration of pacing implantation was approximately 6.4 years (ranging from 1 to 31 years). In this group, modes of pacing were VVI (68%), and DDD (31%). Sixty percent of the patients had RV apical pacing and the average percentage of pacing was 68% (67.7 ± 39 %) (Table 1).

Echocardiographic assessment

Overall mean left ventricular ejection fraction (LVEF) was approximately 65% and moderate to severe TR was found in 21 patients (21.8%) (Table 1). Standard parameters for left and right sided heart function are shown in Table 2.

In the present study group, four patients (4.1%) had RV dysfunction. A comparison between the normal RV function and the RV dysfunction group, age, gender and other underlying diseases showed no statistical difference. There were no significant differences in LVEF (61.5% vs. 64.8%, $p = 0.57$), the degree of tricuspid regurgitation ($p = 0.16$) and mean time of implantation (6.4 years vs. 6.3 years, $p = 0.89$). Mode of pacing and percentage of pacing were also not significantly different between the two groups ($p = 0.37$ and 0.176 respectively) (Table 3).

Discussion

This retrospective study demonstrated a 4% prevalence of right ventricular dysfunction and 21% of moderate to severe tricuspid regurgitation in patients who had a permanent pacemaker implanted over an average of 6.4 years. Site of pacing, pacing mode and percentage of ventricular pacing did not influence right ventricular function.

The benefits of cardiac pacing in patients with symptomatic bradycardia are already well recognized[1]. However, there are concerns of some harmful effects, in particular deterioration of LV function after cardiac pacing.

LV dyssynchrony from stimulation through the myocardium rather than the His-Purkinje conduction system can cause a decrease in stroke volume and abnormal LV relaxation[2]. There are some studies demonstrating that long term pacing causes LV remodeling with asymmetric hypertrophy and dilatation, mitral regurgitation, decrease in myocardial perfusion and decrease in ejection fraction[6,7]. All of

Table 1. Clinical and echocardiographic findings of overall patients

	Overall (n = 96)
Age, years	67.65 ± 15.06
Male (%)	43 (44.8%)
Medical history-No. (%)	
HT	66 (68.8)
DM	18 (18.8)
CAD	6 (6.3)
Dyslipidemia	35 (36.5)
Atrial fibrillation	6 (6.3)
LVEF (%)	64.67 ± 11.27
Tricuspid regurgitation	
Trace-Mild	75 (78.1)
Moderate-Severe	21 (21.8)
TRPG (mmHg)	17.22 ± 10.61
TAPSE (cm)	2.12 ± 0.35
RV lateral annulus velocity (cm/s)	12.48 ± 3.08
Indication for pacing	
Complete heart block	61 (63.5)
Sick sinus syndrome	35 (36.5)
Post implant time (year)	6.44 ± 5.92
Pacing mode	
VVI	65 (67.7)
DDD	30 (31.3)
VDD	1 (1)
Site of pacing lead	
Apex	58 (60.4)
RV septum	38 (39.6)
Atrial pacing (%)	45.54 ± 32.32
Ventricular pacing (%)	67.70 ± 39.09
Ventricular pacing group	
< 40%	27 (28.1)
40-80%	15 (15.6)
> 80%	54 (56.3)
Ventricular pacing group	
≤ 50%	30 (31.3)
> 50%	66 (68.8)

LVEF = left ventricular ejection fraction, RV = right ventricular, TRPG = tricuspid regurgitation peak gradient, TAPSE = Tricuspid Annular Plane Systolic Excursion, HT = hypertension, DM = diabetes mellitus, CAD = coronary artery disease

Table 2. Echocardiographic parameters for left and right sided heart function

Variables	Parameter
Left side	
Parasternal long axis view (PLAX)	
LA diameter (mm)	37.88 ± 6.93
LVEDD (mm)	45.06 ± 6.95
LVESD (mm)	29.88 ± 7.06
IVSd (mm)	12.99 ± 2.77
EF (%)	62.41 ± 11.66
Parasternal short axis view (PSAX)	
LA diameter	38.30 ± 7.10
LVEDD (mm)	45.49 ± 6.86
LVESD (mm)	30.53 ± 6.80
IVSd (mm)	13.28 ± 2.85
EF (%)	60.73 ± 11.16
E/A ratio	0.93 ± 0.32
E/E'	11.63 ± 4.04
Right side	
Chamber dimensions	
Parasternal view	
RVOT PLAX proximal diameter (mm)	28.65 ± 5.20
RVOT PSAX proximal diameter (mm)	27.71 ± 5.02
RVOT PSAX distal diameter (mm)	24.11 ± 4.28
PA diameter (mm)	20.20 ± 4.01
Apical 4-chamber view	
RV basal (mm)	31.30 ± 5.92
RV minor dimension (mm)	25.40 ± 5.74
RV longitudinal dimension (mm)	65.30 ± 8.64
Subcostal view	
RV basal (mm)	30.14 ± 5.76
RV minor dimension (mm)	22.94 ± 6.27
RV longitudinal dimension (mm)	59.51 ± 10.81
Systolic function	
TAPSE (cm)	2.12 ± 0.35
Pulsed Doppler peak velocity at the annulus (S') (cm/s)	12.48 ± 3.08
IVC maximum size (mm)	13.09 ± 4.21
IVC minimum size (mm)	8.18 ± 4.17
TRPG (mmHg)	17.22 ± 10.61

LA = Left atrium, LVEDD = Left ventricular end diastolic diameter, LVESD = Left ventricular end systolic diameter, IVSd = Interventricular septum in diastole, EF = Ejection fraction, RVOT = Right ventricular outflow tract, PA = Pulmonary artery, RV = right ventricle, TAPSE = Tricuspid Annular Plane Systolic Excursion, IVC = Inferior vena cava, TRPG = Tricuspid regurgitation peak gradient

these can result in a higher risk of morbidity and mortality.

RV function has traditionally been considered less clinically important than LV function. Nevertheless, the right ventricle plays a major role in the morbidity and mortality of patients with cardiopulmonary disease[8]. Moreover, assessment of RV function and geometry is limited due to its complex shape. The right ventricle is anatomically, structurally and functionally divided into two parts, the inflow and outflow tract[9]. The RV wall motion is complicated[10,11]. During the systolic phase, there is a longitudinal shortening from base to apex. There is also a radial motion towards the

Table 3. Baseline characteristics of patients divided into group 1 (RV dysfunction) and group 2 (RV normal function). Data are presented as mean ± SD or number (%)

	RV dysfunction (group 1, n = 4)	RV normal function (group 2, n = 92)	p-value
Age, years	70.5 ± 7.0	67.52 ± 15.33	0.869
Male (%)	2 (50%)	41 (44.6%)	0.60
LVEF (%)	61.50 ± 13.99	64.8 ± 11.21	0.57
Tricuspid regurgitation			
Trace-Mild	2 (50)	73 (79.3)	
Moderate-Severe	2 (50)	19 (20.7)	0.165
TRPG (mmHg)	12.25 ± 4.35	17.43 ± 10.76	0.32
Indication for pacing			
Complete heart block	2 (50)	59 (64.1)	
Sick sinus syndrome	2 (50)	33 (35.9)	0.62
Medical history-No. (%)			
HT	2 (50)	64 (69.6)	0.59
DM	1 (25)	17 (18.5)	0.57
CAD	0	6 (6.5)	1.0
Dyslipidemia	2 (50)	33 (35.9)	0.62
Atrial fibrillation	0	6 (6.5)	1.0
Pacing mode			
VVI	4 (100)	61 (66.3)	
DDD	0	30 (32.6)	
VDD	0	1 (1.1)	0.37
Post implant time (year)	6.25 ± 4.99	6.44 ± 5.98	0.89
Site of pacing lead			
Apex	3 (75)	55 (59.8)	
RV septum	1 (25)	37 (49.2)	1.0
Ventricular pacing (%)	38.25 ± 44.79	68.98 ± 38.59	0.176
Ventricular pacing group			
< 40%	2 (50)	25 (27.2)	
40-80%	1 (25)	14 (15.2)	
> 80%	1 (25)	53 (57.6)	0.43

LVEF = left ventricular ejection fraction, RV = right ventricular, TRPG = tricuspid regurgitation peak gradient, TAPSE = Tricuspid Annular Plane Systolic Excursion, HT = hypertension, DM = diabetes mellitus, CAD = coronary artery disease

common septum, which occurs at the RV inflow tract with circumferential motion. This gives a rotation or a squeeze of the ventricle[12]. RV ejection at the outflow tract occurs later and gives an overall peristaltic ventricular motion[13]. Septal motion is considered to cooperate with LV function and plays a major role in overall RV performance[14,15].

Many parameters were used to evaluate RV function. Some of which have demonstrated more clinical utility than others. They are RV index of myocardial performance (RIMP), tricuspid annular plane systolic excursion (TAPSE), two dimensional RV fractional area change (2D RV FAC) and tissue Doppler-derived tricuspid lateral annular systolic velocity (S')[8]. In the present study, the authors used two parameters which were TAPSE and S'. TAPSE and S' can be measured easily, reliably and are more easily reproducible. Even though they measure RV longitudinal function, it is generally accepted that this has good correlation with other methods such as radionuclide-derived RV EF, 2D RV FAC and 2D RV EF. A TAPSE measurement of less than 16 mm or S' velocity less than 10 cm/s indicate RV systolic dysfunction[8].

To the best of our knowledge, the impact of RV pacing on RV function has not been reported. Apart from non-physiological electrical stimuli directly affecting the myocardium causing dyssynchrony of the ventricles, TR can also deteriorate RV function[16]. Several studies[4,17-19] have demonstrated the influence of permanent cardiac pacing on tricuspid valve function.

However, the results are still inconclusive. There are several potential causes of pacemaker related TR. These may involve anatomic defects such as leaflet perforation or entrapment from fibrous adhesions of scar tissue and functional disorders by interfering with leaflet movement.

In the present study, the authors found that the RV dysfunction detected by TAPSE less than 16 mm was approximately 4% in patients with permanent pacemaker implantation. Also, a moderate to severe degree of TR was found in around 21% of this particular group at mid term follow-up.

There are factors to consider that impact on LV function in cardiac pacing patients. These include site of pacing, pacing mode and the percentage of pacing. RV apical pacing was blamed for deteriorating LV function compared to RV septal pacing, as the latter more closely approximates the normal conduction system. Therefore, it causes less electrical delay and LV dyssynchrony[20]. However, the benefits of septal pacing were shown only in short term[21,22], but not in long term trials[23].

Concerning pacing mode, it has been suggested that dual-chamber pacing (DDD), may lead to less LV dyssynchrony compared to single-chamber pacing (VVI). Nevertheless, large recent trials showed no clinical benefit of physiologic DDD pacing over VVI[24,25]. One study[26] revealed that percentage of right ventricular pacing more than 40% in patients with DDD pacing and more than 80% in patients with VVI was associated with an increasing incidence of heart failure hospitalization. Therefore, a strategy of minimal ventricular pacing was developed. With respect to the present study, factors impacting on right ventricular dysfunction were analyzed. The authors found that the site of the pacing lead, the pacing mode, as well as the percentage of ventricular pacing did not significantly affect the difference between normal and abnormal right ventricular function.

Conclusion

The prevalence of right ventricular dysfunction is quite low in patients with permanent pacemaker implantation at mid term follow-up. Site of pacing, mode of pacing and percentage of pacing did not influence right ventricular function.

Limitations

There are some limitations in the present study. First, it was a retrospective analysis requiring medical retrieval. Some data were missed due to the variability of the quality of the medical records. Secondly, there was no routine evaluation of right ventricular function in the CCIT echo unit prior to pacemaker implantation. Therefore, data of right ventricular function before implantation was not obtained to compare with data after the procedure.

Potential conflicts of interest

None.

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การศึกษาประสิทธิภาพการทำงานของหัวใจล่างขวา ในผู้ป่วยที่ได้รับการฝังเครื่องกระตุ้นหัวใจห้องขวา

พรวลี ปรปักษ์ขาม, ปราโมทย์ ปรปักษ์ขาม, จรินทร์ อัศวหาญฤทธิ์, บุญชัย กิจสนาโยธิน, คีธ วิงซิง

***ภูมิหลัง**: ประโยชน์ของการฝังเครื่องกระตุ้นหัวใจห้องขวาในผู้ป่วยที่มีอาการจากหัวใจเต้นช้าเป็นที่ยอมรับอย่างกว้างขวาง ในปัจจุบันการศึกษาส่วนใหญ่มุ่งเน้นเกี่ยวกับผลของการกระตุ้นหัวใจห้องล่างขวาในผู้ป่วยที่ได้รับการฝังเครื่องกระตุ้นหัวใจ กับประสิทธิภาพการบีบตัวของหัวใจห้องล่างซ้าย แต่การศึกษาผลกระทบต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวายังมีอยู่น้อยมาก*

***วัสดุและวิธีการ**: เพื่อเป็นการศึกษาถึงผลกระทบต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวาได้มีการศึกษาข้อมูลจากเวชระเบียนของผู้ป่วยที่ได้รับการฝังเครื่องกระตุ้นหัวใจมาแล้วอย่างน้อย 1 ปี เพื่อหาอุบัติการณ์ของการทำงานของหัวใจล่างขวาที่ลดลง และหาปัจจัยที่มีผลกระทบต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวา*

***ผลการศึกษา**: อุบัติการณ์ของประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวาที่ลดลง และลิ้นหัวใจไตรคัสปิดรั่วระดับปานกลางจนถึงมากพบได้ร้อยละ 4 และ ร้อยละ 21 ตามลำดับ โดยระยะเวลาที่ผู้ป่วยทั้งหมดได้รับการฝังเครื่องกระตุ้นหัวใจห้องขวาเป็นระยะเวลาเฉลี่ย 6.4 ปี เมื่อศึกษาปัจจัยที่คาดว่าจะมีผลกระทบต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวาที่ลดลง ซึ่งได้แก่ตำแหน่งการกระตุ้นของหัวใจ ชนิดของเครื่องกระตุ้นหัวใจ และร้อยละของการกระตุ้นหัวใจห้องขวาล่าง มาเปรียบเทียบระหว่าง 2 กลุ่ม พบว่าไม่แตกต่างอย่างมีนัยสำคัญทางสถิติกับกลุ่มที่ประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวาปกติ*

***สรุป**: จากการศึกษานี้พบว่าอุบัติการณ์ของการทำงานของหัวใจล่างขวาที่ลดลงพบได้ไม่บ่อย ปัจจัยที่ได้รับการยอมรับว่ามีผลกระทบต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างซ้าย กลับไม่พบว่ามีผลต่อประสิทธิภาพการบีบตัวของหัวใจห้องล่างขวาที่ลดลง*