Left Ventricular Function After Permanent Pacemaker in Pacemaker Clinic Follow-Up

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Background: *Chronic right ventricular pacing has deleterious effects to left ventricular function which may be asymptomatic. Prevalence of LV dysfunction (LVEF < 50%) in RV pacing in Thai patients is not known.*
Material and Method: *Patients in the Central Chest Institute of Thailand (CCIT) pacemaker clinic were retrospectively reviewed for their demographic and pacemaker data together with echocardiographic data for LV function. Analysis was done for those who were implanted for more than one year period.*
Results: *Among the studied 118 patients, male = 51 and female = 67, mean follow-up time 6.43 ± 5.66 years, LV systolic dysfunction was identified in 21(17.80%). Compared to those with no LV dysfunction, echocardiographic parameters showed larger LV size (EDD 49.86 ± 8.95 vs. 43.81 ± 5.56 mm) and less thickness of the LV wall (11.05 ± 1.60 vs. 12.49 ± 2.79 mm). Studied clinical and pace parameters for correlation were hypertension (p = 0.048), coronary artery disease (p = 0.008), percent of ventricular pacing (p = 0.06), duration after implantation (p = 0.23), mode of pacing (p = 0.275), indication of implantation (p = 0.32, site of pacing lead (p = 0.279), moderate to severe MR (p = 0.003) and moderate to severe TR (p = 0.04).*
Conclusion: *LV dysfunction after pacemaker implantation was not infrequent. Parameters correlated to it were previous LV dysfunction, hypertension, coronary artery disease and increased amount of ventricular pacing. Mode of pacing and site of pacing lead were not correlated.*

Keywords: *Permanent pacemaker, Left ventricular function*

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Permanent cardiac pacing is essential treatment for patients with irreversible symptomatic bradycardia to improve symptoms and possibly life saving. This is usually life-long treatment. Implantation of a permanent pacemaker in adults is usually done by transcutaneous method with the tip of pacing lead at the right ventricle apex, or recently preferably at right ventricular septum or outflow tract.

Cardiac pacing from the right ventricle has been reported to cause abnormal activation sequences which can lead to valve regurgitation and either acute or chronic left ventricular dysfunction. Owing to literatures, the prevalence of LV dysfunction is quite common. However, in the author's clinical practice from pacemaker clinic follow up, implanted patients need only a few admissions due to heart failure. Since the prevalence and impact of LV dysfunction from pacemaker implantation in Thai patients is actually unknown, the present study aimed to find its prevalence and correlation to other clinical data.

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Materials and Method

Study population

During 15th June to 30th October 2011, all patients who were implanted with permanent pacemaker for more than 1 year for any reasons except for the CRT and AICD pacemaker and routinely followed-up at pacemaker clinic in Central Chest Institute of Thailand, were retrospectively reviewed for the "current echocardiograhic study" (defined as echocardiogram which may be done at time of clinic follow-up or during the previous 6 months) and also for previous echocardiograms. Patients' clinical data, pacemaker implantation data and the echocardiogram data of left ventricular function were together collected. All patients were non-formally informed and all agreed to have an echocardiographic examination.

Left ventricular function was measured by standard echocardiographic methods. The M-mode echocardiogram was done in both parasternal long axis view (PLAX), parasternal short axis view (SAX) and

echo parameters were recorded for end-diastolic diameter (EDD) end systolic diameter (ESD) end-diastolic volume (EDV), end-systolic volume (ESV) and left ventricular ejection fraction (LVEF). Doppler echo study was done for the mitral valve E wave and A wave velocity as well as tissue Doppler of the E' and A' velocity at medial mitral annulus. LV systolic function was defined as normal with LVEF at least 55% in both parasternal long axis and short axis view. LV systolic dysfunction was defined as finding of LVEF less than 50% in either from parasternal long axis view or short axis view study.

Pacing parameters were obtained during the follow-up about the indication of implantation *i.e.* time since first implantation, mode of pacing (VVI, DDD, VDD, AAI), site of the implanted lead (RV apex, RV septum or RVOT), and percent of right ventricular pacing at last pacemaker follow-up.

Data were analysis by statistical program, SPSS version 18. Continuous data was analysis using mean and standard deviation and compared between group by t-test. Categorical data was analysis by Chi-square. Significant value defined by p-value less than 0.05.

The present study protocol was approved by ethical committees of Chest Disease Institute of Thailand.

Results

Total number of 505 patients attended pacemaker clinic during the present study period with number of AICD and CRT for 97 patients. The remaining 408 patients, when excluded those who were implanted for less than 1 year and those had no available current echocardiographic results study, 118 patients (represented about 28.9% of all the follow-up pacemaker patients) entered the present study. Demographic data (Table 1) showed the number of male patients = 51, female = 67, age range = 21 to 91 years (median age 71, mean 67.26 $\pm$ 15.77 years). The underlying diseases were hypertension in 84 patients (62.7%), diabetes 18 (15.3%), dyslipidemia 38 (32.2%), coronary artery disease 9 (7.6%), atrial fibrillation 7 (5.9%) and previous report of LV systolic dysfunction 9 patients (7.6%). Indications for implantation were symptomatic heart block in 73 patients (61.9%), sick sinus syndrome 45 patients (38.1%). Implantation modes of pacing were VVI in 78 patients (66.10%), DDD 39 patients (33.1%). The position of pacing lead tip were at RV apical position in 72 patients(61.0%), RV septum or RVOT 43 patients (36.4%). Most clinical parameters (HT, DM, CAD, dyslipidemia) and mean of various echocardiographic parameters (such as LA volume, LVEF, E and E' velocity) were all comparable between male and female groups, except for the slight larger of mean left ventricle volume (EDD 46.82 $\pm$ 7.17 vs. 43.42 $\pm$ 5.90 mm) and left ventricular wall thickness (IVS 13.63 $\pm$ 2.70 vs. 12.57 $\pm$ 2.52 mm) in the male group.

Duration since first implantation of device ranged from 1 to 31 year, with median duration of 4.50 years, mean 6.43 $\pm$ 5.66 years. Percent of the ventricular pacing ranged from 1% to 100%, with mean 67.85 $\pm$ 39.29. Number of pace dependent (need almost all times pacing or more than 80% of ventricular beat paced) was 67 patients (56.8%).

Current Echocardiographic study (Table 1 and 2) showed mean LA diameter 38.12 $\pm$ 7.18, inter-ventricular septum (IVS) 13 $\pm$ 2.64 and posterior wall (PW) 12.2 $\pm$ 2.67 mm. Number of those with normal LVEF patients was 94 (79.66%), overall mean LVEF = 62.36 $\pm$ 11.34%, median LVEF = 63.5%. The diastolic function was difficult to assess since the values varied very much, with mean mitral E wave velocity = 77.76 $\pm$ 23.69, medial mitral annulus E' velocity = 7.67 $\pm$ 5.49. Trivial to mild mitral regurgitation was present in 95 patients (80.51%), trivial to mild TR in 94 (79.66%), with moderate to severe MR in 23 (19.49%) and moderate to severe TR in 21 (17.80%). No severe AR was detected in the present study.

LV dysfunction was found in 21 patients (17.80%). When compared to those without LV dysfunction (Table 2), this group had more dilatation of LV chamber size (EDD 49.86 $\pm$ 8.95 vs. 43.81 $\pm$ 5.56 mm) with less thickness of the LV wall (11.05 $\pm$ 1.60 vs. 12.49 $\pm$ 2.79 mm), hence indicate eccentric LV dilatation.

Clinical and pacing parameters were grouped and analysis for overall data (Table 3), the studied parameters that had correlation with presence of LV dysfunction were hypertension ($p = 0.048$), coronary artery disease ($p = 0.008$), percent of ventricular pacing ($p = 0.06$). There was no correlation with the duration after implantation ($p = 0.23$), mode of pacing ($p = 0.275$), indication of implantation ($p = 0.32$), site of pacing lead ($p = 0.279$).

When data analysis was limited for those who were implanted for less than 5 years, the mentioned factors had lost their correlation with LV dysfunction ($p = 0.247, 0.815, 0.407$ for HT, CAD and percent pacing successively). Same findings also extinct when data was limited for those with less than 50% of ventricular pacing ($p = 0.59, 0.69$ for HT and CAD) (Table 4).

Table 1. Demographic data of the studied patients

Parameters	Number total n = 118 (%)
Male: Female	51: 67
Mean age ± SD (range)	67.26 ± 15.77 (21-91)
Diabetes	18 (15.3)
Hypertension	84 (71.2)
Coronary disease	9 (7.6)
Dyslipidemia	38 (32.2)
Previous LV dysfunction	9 (7.6)
Heart block	73 (66.1)
Sick sinus syndrome	45 (38.1)
Pacing mode (VVI)	78 (66.1)
Pacing mode (DDD)	39 (33.0)
Pacing site (RV apex)	72 (61.0)
Pacing site (RV septum or RVOT)	43 (36.4)
Pace dependent (patients need more than 80% of pacing)	67 (56.7)
Atrial fibrillation	7 (5.9)
Trivial to mild mitral regurgitation	95 (80.5)
Moderate to severe mitral regurgitation	23 (19.4)
Trivial to mild tricuspid regurgitation	94 (79.6)
Moderate to severe tricuspid regurgitation	21 (17.8)

Table 2. Echocardiographic parameters

Parameters (mean ± SD)	Total n = 118	Patients with LV dysfunction n = 21	Patients with No LV dysfunction n = 97	p-value
Age	67.26 ± 15.77	64.05 ± 15.83	67.96 ± 15.76	0.305
Previous LVEF**	62.58 ± 11.90	54.27 ± 15.97	64.89 ± 9.45	0.002
Post implantation time**	6.43 ± 5.66	8.71 ± 7.39	5.94 ± 5.12	0.041
Percent A (atrium) pace	51.47 ± 33.20	54 ± 43.14	51.24 ± 32.99	0.893
Percent V (ventricle) pace	67.85 ± 39.29	76.13 ± 39.10	66.06 ± 39.31	0.289
LA diameter	38.12 ± 7.18	40.15 ± 6.09	37.66 ± 7.36	0.163
LV EDD**	44.89 ± 6.67	49.86 ± 8.95	43.81 ± 5.56	< 0.001
LV EDS**	29.56 ± 7.0	38.34 ± 8.63	27.65 ± 4.85	< 0.001
IVS	13.03 ± 2.64	12.71 ± 3.32	13.01 ± 2.48	0.554
PW**	12.24 ± 2.67	11.05 ± 1.60	12.49 ± 2.79	0.024
LVEF (PLAX)**	62.36 ± 11.34	45.29 ± 9.9	66.06 ± 7.65	< 0.001
LVEF (SAX)**	60.65 ± 11.03	44.20 ± 7.93	63.35 ± 7.77	< 0.001
TR peak gradient	22.15 ± 9.58	24.58 ± 9.18	21.76 ± 9.64	0.346
E velocity	77.76 ± 23.69	84.51 ± 30.78	76.37 ± 21.96	0.228
E' velocity	7.67 ± 5.49	9.72 ± 11.89	7.24 ± 2.69	0.112
A velocity	77.26 ± 22.29	60.63 ± 30.78	78.39 ± 22.44	0.186
E/E' ratio	11.99 ± 4.49	13.05 ± 4.4	11.78 ± 4.5	0.320

Note: PLAX = parasternal examination view, SAX = short axis examination view, (** = significant different between group)

Discussion

Patients may need implantation of a permanent pacemaker for many reasons, most of the indications are for symptomatic complete or advanced heart block and symptomatic sick sinus syndrome. The usual popular way of pacemaker implantation in adults is via transcutaneous method. In this conventional method, the pacemaker impulse generator is put under subcutaneous tissue at the patient's right or left shoulder with the implanted lead inserted through subclavian vein

Table 3. Clinical data groups and correlation to the presence of current LV dysfunction

Parameter groups and LV dysfunction correlation	p-value
Presence of previous LV dysfunction	0.002
Presence of hypertension	0.048
Presence of coronary artery disease	0.008
Percent ventricular pace	0.060
Duration of implantation	0.230
Presence of moderate to severe MR/TR	0.003/0.04

Table 4. Clinical data groups and correlation to the presence of current LV dysfunction, with data analysis limited to specific patients

Parameter groups and LV dysfunction correlation	p-value for the group of patients implanted for less than 5 years	p-value for the group of patients with less than 50% ventricular pacing
Presence of hypertension	0.247	0.59
Presence of coronary artery disease	0.815	0.69
Percent ventricular pace	0.407	-

down to right ventricular endocardium surface with pacing tip at RV apex. With active fixation method, pacing tip could be placed at other positions such as RV septum, outflow tract and even at the His-bundle. In young children or in patients who underwent cardiac surgery, the pacing lead may be surgically placed at the epicardial surface of the heart. In a dual chamber pacemaker implantation (DDD), another lead is placed in the right atrium and pacemaker generator is then programmed to the desired mode such as AAI, VVI, DDD or VDD. In the present studied patients, the indications of pacing were quite the same as in other reports *i.e.* symptomatic heart block 61.86% and sick sinus syndrome 38.14% and the lead positions were at RV apex in 61.02%, at RVOT or RV septum 38.14%, major mode of pacing was VVI 66.1% and DDD 33.05%.

Although pacemaker implantation usually improves both prognosis and symptoms from bradycardia, there are some concerns about the long term harmful effects, not only directly from pacemaker generator or lead related complications such as the infection at the implanted site, lead tip perforation, lead fracture, dislodged and migration, but also indirectly from the effect of right ventricular pacing. Right ventricular apex pacing, compared to normal heart activation by which electrical sequences begin from the base of the heart and rapidly going down via conduction system towards apex, is considered markedly abnormal since it causes reverse activation wavefront from the apex towards base with slow activation time (like the activation pattern in left bundle branch block). Clinical reviews showed that this may result in delay of the contraction between the right and left ventricle (inter-ventricular-dyssynchrony) and also delay time to contraction of the muscle mass between left ventricular septal wall and lateral wall (LV intra-ventricular-dyssynchrony)[1]. Goo-Yeong Cho showed that dyssynchrony was detected in more than 50% of the patients after RV pacing, both RV apex and RV septum, in tissue Doppler and strain echo study[2]. Abnormalities in these contractions can lead to presystolic mitral regurgitation, change in LV wall thickness, degenerative fibrosis of LV, LV dysfunction and LV remodeling[1,3]. Apical RV pacing can cause changes in RV and LV diastolic function[4], changes in cardiac metabolism and changes in coronary perfusion even in the absence of coronary artery disease[5,6]. The pacing also affects left atrium, it causes increase of the LA size even in absence of LV dysfunction[7] and causes abnormal atrium function evaluated by strain echocardiography[8]. Simon DR. Thackray showed that RV apical pacing frequently lead to symptoms of heart failure in both LV systolic dysfunction group and those with rather preserved LV function[9].

To alleviate this problem, alternative sites other than RV apex, such as RV septum and RV outflow tract sites and new pacing algorithms to reduce ventricular pacing, had been studied. Irene et al had reported a

long term follow-up results in children with congenital heart block that, with both the RV epicardial pacing and RV endocardial pacing, the LV function was subnormal in 10-14% but not with LV epicardial pacing[10]. Epicardial RV free wall pacing in very young pediatric patients had been reported to have marked affection of LV dysfunction[11]. Pacing from septum and outflow tract seem to have more physiological activation sequences than from apex. Outflow tract pacing had promising data from meta-analysis for favorable hemodynamic findings[12]. However, data from long term follow-up of septal pacing still showed no benefit in both LVEF and exercise capacity compared to RV apex pacing. Algorithm of pacing mode such as minimal ventricular pacing by using AV hysteresis search algorithm was studied in the INTRINSIC RV (Inhibition of Unnecessary RV Pacing with ASVH in ICDs Study) and could significantly reduce the percent in RV pacing[13]. Study of algorithm to reduce percent ventricular paced in the SAVE PACe (Search AV Extension and Managed Ventricular Pacing for Promoting Atrioventricular Conduction) trial had shown benefit to reduce development of persistent atrial fibrillation, however, the present study did not show significant difference in heart failure and mortality[14].

It is also suggested that unfavorable effects of RV pacing may be more pronounced in certain patient groups. Pastore et al had shown that there was higher prevalence and degree of LV dyssynchrony after RV apical pacing in patients with lower LVEF[15]. Barsheshet et al had recently reported long term effects of RV pacing in the poor LV systolic patients with AICD in MADIT-II that, after 4 years, the survival benefit of AICD was attenuated in high RV pacing (more than 50% pacing) while still preserved in the low RV pacing subgroup[16]. In the present study, clinical factors that affect the LV function includes previous LV dysfunction, hypertension, coronary artery disease, and increased amount of ventricular pacing while mode of pacing, site of pacing lead seem not to correlate. It is possible that these underlying diseases may prime the myocardium for the deleterious effects of RV pacing by mechanisms as previously described. However, in the present study, when the data analysis was limited to those who had been implanted less than 5 years and those with ventricular pacing less than 50%, the clinical parameters with previously significant correlation to LV dysfunction had much less significant p-value. This should emphasize the importance of the time effect of long-time pacing and amount of ventricular pacing to produce changes in the LV function.

From the present study, the prevalence of LV dysfunction in Thai patients with pacemaker was about 17.80%. Since there had been a strong relation of current LV dysfunction to the history of previous LV dysfunction, it is suggested that echocardiogram should be done at baseline in every patient who is planned for pacemaker implantation and then should have a repeat of study at least in 5 years thereafter. Primary coronary prevention measurements, especially the aggressive treatment of hypertension, is mandatory for implanted patients. Those who had baseline abnormal LFEF should be followed-up more frequently by routinely echocardiogram to detect early changes in LV function. Lastly, efforts to minimize atrial and ventricular pacing by proper programming, perhaps to less than 50% of ventricular pacing, should be done during the follow-up in the pacemaker clinic.

Conclusion

The prevalence of LV systolic dysfunction in Thai patients with pacemaker was not infrequent. Left ventricular dysfunction was related to the history of hypertension, coronary artery disease, previous LV dysfunction. High percentage of ventricular pacing and long duration time after implantation, but not the pacing site at apex or septum site of the right ventricle, also related to LV dysfunction.

Limitations

Since this was the data in one center pacemaker clinic and it was a retrospective study, the patients might not represent all of the implanted population. The present study also had limitation in the data collection to gather other important factors that might affect the LV function such as routine coronary angiogram, history of angina, smoking, alcohol, psychosocial aspects and all of the medications used, etc. Finally, with resource limitation, there were no serial echocardiograms for every case to follow changes in the LV function, so the true effect of ventricular pacing to the ventricular function could not be properly assessed in the present study.

Potential conflicts of interest

None.

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การทำงานของหัวใจห้องล่างซ้ายในผู้ป่วยที่ใส่เครื่องกระตุ้นไฟฟ้าหัวใจชนิดถาวร

จรินทร์ อัศวหาญฤทธิ์, พีรพัฒน์ เกตุค้างพลู, พรวลี ปรปักษ์ขาม

ภูมิหลัง: *การกระตุ้นหัวใจจากเครื่องกระตุ้นไฟฟ้าหัวใจโดยการกระตุ้นจากหัวใจทางด้านขวานั้นไม่เป็นไปตามธรรมชาติของการกระตุ้นไฟฟ้าหัวใจปกติ และอาจมีผลเสียต่อการทำงานของหัวใจห้องล่างซ้ายได้โดยอาจจะยังไม่มีอาการแสดง อย่างไรก็ตามอุบัติการณ์และปัจจัยที่มีผลต่อการตรวจพบดังกล่าวในคนไทยยังไม่ได้มีการศึกษาที่ชัดเจน*

วัสดุและวิธีการ: *ผู้นิพนธ์ได้ทำการตรวจการทำงานของหัวใจห้องล่างซ้ายของผู้ป่วยที่มารับการตรวจในคลินิกเครื่องกระตุ้นไฟฟ้าหัวใจ โดยตรวจจากภาพเสียงสะท้อนรูปหัวใจและรวบรวมปัจจัยต่างๆ ทางคลินิกที่พบโดยใช้ข้อมูลจากผู้ป่วยที่ใส่เครื่องกระตุ้นไฟฟ้าหัวใจนานอย่างน้อยหนึ่งปีเพื่อทำการศึกษา*

ผลการศึกษา: *ผู้ป่วยที่ทำการศึกษาจำนวน 118 ราย เป็นเพศชาย 51 ราย เพศหญิง 67 ราย ในจำนวนนี้พบว่ามีจำนวนที่ตรวจพบการทำงานลดลงของกล้ามเนื้อหัวใจ (LV dysfunction, LVEF < 50%) จำนวน 21 ราย (17.80%) เมื่อเทียบกับกลุ่มที่ไม่มีภาวะดังกล่าว คนไข้กลุ่มนี้มีขนาดของห้องหัวใจล่างซ้ายใหญ่กว่า (EDD 49.86 $\pm$ 8.95 เทียบกับ 43.81 $\pm$ 5.56 มม.) มีความหนาของผนังห้องหัวใจน้อยกว่า (11.05 $\pm$ 1.60 เทียบกับ 12.49 $\pm$ 2.79 มม.) ปัจจัยทางคลินิกที่เกี่ยวกับเครื่องกระตุ้นไฟฟ้าหัวใจที่ได้ศึกษาถึงความสัมพันธ์กับการตรวจพบ (p-value) ดังกล่าวได้แก่ การมีภาวะความดันโลหิตสูง (p = 0.048) โรคหลอดเลือดหัวใจ (p = 0.008) จำนวนเปอร์เซนต์ ของการกระตุ้นหัวใจ (p = 0.06) ระยะเวลาตั้งแต่เริ่มใส่เครื่องถึงปัจจุบัน (p = 0.23) หมวดหรือชนิดของการกระตุ้นหัวใจ (p = 0.275) ข้อบ่งชี้ในการใส่เครื่อง (p = 0.32) ตำแหน่งของการกระตุ้นหัวใจ (p = 0.279) การตรวจพบลิ้นหัวใจ ไมตรัลรั่วที่สำคัญ (p = 0.003) และการตรวจพบลิ้นหัวใจไตรคัสปิดรั่วที่สำคัญ (p = 0.04)*

สรุป: *ภาวะหัวใจห้องล่างซ้ายบีบตัวลดลงในผู้ป่วยที่ใส่เครื่องกระตุ้นไฟฟ้าหัวใจพบได้บ่อย ปัจจัยที่มีความสัมพันธ์กับการตรวจพบดังกล่าวได้แก่ การเคยตรวจพบว่ามีหัวใจบีบตัวลดลงมาก่อนหน้านั้น ภาวะความดันโลหิตสูง โรคหลอดเลือดหัวใจ จำนวนเปอร์เซนต์ของการกระตุ้นหัวใจ ส่วนปัจจัยที่ไม่มีความสัมพันธ์ ได้แก่ หมวดหรือชนิดของเครื่อง และตำแหน่งของการกระตุ้นหัวใจ*