Effect of right ventricular lead location on response to cardiac resynchronization therapy in patients with end-stage heart failure

Majid Haghjoo[1]*, Hamid Reza Bonakdar[1], Mohammad Vahid Jorat[1], Amir Farjam Fazelifar[1], Abolfath Alizadeh[1], Zahra Ojaghi-Haghjghi[2], Maryam Esmaielzadeh[2], and Mohammad Ali Sadr-Ameli[1]

[1]Department of Pacemaker and Electrophysiology, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, PO Box: 15745-1341, Mellat Park, Vali-E-Asr Avenue, Tehran 1996911151, Iran; and [2]Department of Echocardiography, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, Iran

Aims	It is currently recommended to implant the left ventricular (LV) pacing lead at the lateral wall. However, the optimal right ventricular (RV) pacing lead location for cardiac resynchronization therapy (CRT) remains controversial. We sought to investigate whether optimizing the site for placement of the RV lead could further improve the long-term response to CRT in patients with advanced heart failure.
Methods and results	Between October 2006 and December 2007, a total of 73 consecutive patients with standard indication for CRT were enrolled. The enrolled patients were divided into two groups based on the RV lead location. There were 50 patients in RV apex (RVA) group and 23 patients in RV high septum (RVHS). The primary study endpoint was a decrease in LV end-systolic volume (LVESV) by $>15\%$ at 6-month follow-up. The secondary endpoints were improvement in New York Heart Association (NYHA) class by ≥ 1 point and decrease in brain-type natriuretic peptide (BNP) levels by $>50\%$ after CRT. At 6-month follow-up, improvement in NYHA class by ≥ 1 point (RVA: 72% vs. RVHS: 74%, $P = 0.76$), decrease in LVESV by $\geq 15\%$ (RVA: 65% vs. RVHS: 64%, $P = 0.76$), and decrease in BNP level by $>50\%$ (RVA: 70% vs. RVHS: 69%, $P = 0.88$) were observed in similar proportion of the two groups. When we separately assessed the significance of RV pacing site in three LV stimulation sites, there were no significant differences in terms of clinical improvement (62 vs. 64%, $P = 0.74$) and decrease in LVESV by $>15\%$ (63 vs. 62%, $P = 0.78$) between RVA and RVHS pacing when the LV stimulation site was lateral cardiac vein. In anterolateral vein pacing site, the RVA stimulation was associated with higher clinical (88 vs. 47%, $P = 0.05$), echocardiographic (75 vs. 32%, $P = 0.02$), and neurohormonal responses (80 vs. 50%, $P = 0.04$) compared with that in RVHS site. When LV was paced from posterolateral vein, RVHS pacing was superior to RVA in terms of the clinical improvement (85 vs. 35%, $P = 0.01$), echocardiographic response (72 vs. 30%, $P = 0.01$), and decrease in BNP levels (75 vs. 50%, $P = 0.04$).
Conclusion	The present study did not show any difference between RVA and RVHS pacing sites in terms of overall improvement in clinical outcome and LV reverse remodelling following CRT. However, effect of RV lead location on CRT response varies depending on LV stimulation site.
Keywords	Cardiac resynchronization therapy • Heart failure • Response • Right ventricular lead

Introduction

Cardiac resynchronization therapy (CRT) has clearly demonstrated its clinical benefits in patients with advanced heart failure and intraventricular conduction abnormalities.[1,2] The hypothesized mechanism of CRT benefit is that pacing of both right ventricle (RV) and left ventricle (LV) results in a synchronized electrical excitation and mechanical contraction of the LV. Left ventricular

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* Corresponding author. Tel: +98 21 2392 2163, Fax: +98 21 2204 8174, Email: majid.haghjoo@gmail.com; haghjoo@rhc.ac.ir

stimulation site appears to be a crucial factor for successful CRT. There are several studies evaluating the impact of LV stimulation site on CRT,[3–8] and it is currently common practice to place the LV lead at the free wall corresponding to the anatomical regions of lateral, anterolateral or posterolateral veins of the coronary sinus (CS). On the other hand, RV lead is conventionally positioned at the apex (RVA), but sometimes an alternative pacing site as far as possible from the LV lead is used. Detrimental haemodynamic effects of RVA pacing have been demonstrated in clinical trials of conventional pacing therapy.[9,10] In addition, a number of acute haemodynamic studies demonstrated advantage of alternative RV pacing sites [RV high septum (RVHS)] over the RVA pacing for univentricular pacing.[11–13] However, little is known as to whether RV lead positioning provides additional long-term benefit to CRT. Therefore, we sought to investigate whether optimizing the site for placement of the RV lead could further improve the long-term response to CRT in patients with advanced heart failure.

Methods

Demographic and clinical characteristics of study population

Between October 2006 and December 2007, a total of 73 consecutive patients with symptomatic drug-refractory heart failure, a wide QRS complex ($\geq$120 ms), LV end-diastolic diameter (LVEDD) $\geq$55 mm, and LV ejection fraction (LVEF) $\leq$35% were enrolled. Patients with recent myocardial infarction (<3 months), coronary artery bypass graft surgery within the previous 3 months, primary valvular heart disease, or hypertrophic or restrictive cardiomyopathy were excluded. The study was approved by the Ethics Committee and all patients gave written informed consent. Before inclusion in the study, the patients had been clinically stable for $\geq$3 months and optimally treated with recommendation of at least a diuretic, beta-adrenergic blocker, and angiotensin-converting inhibitor or angiotensin receptor blocker, at the highest tolerated doses. Before CRT implantation, New York Heart Association (NYHA) functional class and distance covered during 6-min hall walk test (6-MHWT) were assessed, and two-dimensional echocardiography was performed to determine LV volumes and LVEF. Left ventricular dyssynchrony was assessed using M-mode echocardiography and color tissue Doppler imaging (TDI). Assessment of LV dyssynchrony was repeated at 1 day after CRT implantation, and clinical status (NYHA class and 6-MHWT distance) was reassessed. Clinical status and changes in LVEF and LV volumes were re-assessed at 6-month follow-up. NYHA class grading and 6-MHWT were done by a physician blinded to the other data.

Implantation technique

CRT devices were implanted via subclavian or axillary venous access. The RV lead was implanted at the apex when the pacing threshold, the detection, and the stability were correct. When these parameters were not acceptable, the RV lead was positioned in the high septum (RV outflow septal region). The procedure for implantation of the RVHS lead was to advance the lead out the pulmonary artery, withdrawing the lead until it dropped below the pulmonic valve, and then advancing the lead into the high septum. Proper RVHS lead was confirmed by fluoroscopy and pacing from this location was expected to produce a left bundle branch block and inferior-axis ECG morphology. Guided by a venogram, the LV lead was placed in a CS tributary in a stable lateral, anterolateral, or posterolateral

position with a <3.0 V pacing threshold. Thereafter, the RA lead was implanted conventionally. Final lead positioning was assessed from three different X-ray views (anteroposterior and right and left anterior oblique views). When an indication for an internal defibrillator existed, a combined device was implanted. The CRT devices and lead implantation were completed without major complications. Cardiac resynchronization therapy devices and leads were used from Medtronic (St Paul, Minnesota, USA; $n = 40$), St Jude Medical (St Paul, Minnesota, USA; $n = 30$), ELA medical (Le Plessis Robinson, Normandie, France; $n = 2$), and Guidant-Boston Scientific (Minneapolis, MN, USA; $n = 1$). Patients in sinus rhythm received an atriobiventricular pulse generator programmed in DDD(R) mode and interfaced with bipolar right atrial and RV leads and unipolar or bipolar LV leads. The atrioventricular delay was optimized individually based on Doppler echocardiographic measurements of transmitral flow 1 day after implantation. The atrioventricular delay was not modified during the 6-month follow-up.

Echocardiographic protocol

A complete M-mode, two-dimensional, Doppler evaluation was performed using ultrasonographic equipment (Vivid 7, General Electric, USA) before CRT implantation, immediately before discharge and 6 months after implantation. Images were obtained via a 3.5 MHz transducer at an appropriate depth in the parasternal and apical views. Left ventricular end-systolic volume (LVES), LV end-diastolic volume (LVED), and LVEF were calculated with the biplane Simpson's technique. Mitral regurgitation was graded according to the jet area method.

Dyssynchrony was assessed at the interventricular and left intraventricular levels as described previously.[14] Interventricular dyssynchrony was defined as a >40 ms interventricular mechanical delay (IVMD) calculated as the difference between LV and RV pre-ejection periods, measured between the onset of the QRS complex and, respectively, onset of aortic and onset of pulmonary ejection flows by pulsed-wave Doppler. The standard deviation of the time to peak myocardial systolic velocity of all 12 segments (Ts-SD) was calculated, and Ts-SD >34 ms was considered as the cut-off point for intraventricular dyssynchrony based on previous observations.[15] Intra- and interobserver variabilities were tested on 10 randomly selected cases. Intraclass correlation coefficients for inter- and intraventricular mechanical dyssynchrony values were between 0.91 and 0.95, respectively. A fixed atrioventricular delay of 110–130 ms was chosen in all cases and was optimized only if the patients did not do well clinically. All the echocardiographic measurements were performed by two experienced physicians who were blinded to the other data of the patients.

Follow-up

Patients were followed up in our device clinic at 1 month post-implant and then 3 monthly. Assessment of LV dyssynchrony was repeated at 1 day after CRT implantation, and clinical status (NYHA class and 6-MHWT distance) was re-assessed. Clinical status and changes in LVEF, LV volumes, and dyssynchrony were re-assessed at 6-month follow-up. The primary study endpoint was a decrease in LVESV by >15%. The secondary endpoints were improvement in NYHA class by $\geq$1 point and decrease in brain-type natriuretic peptide (BNP) level by >50% after CRT. These endpoints were determined by a physician blinded to the RV lead locations.

Statistical analysis

The variables are expressed as mean $\pm$ SD for the continuous variables and as absolute or relative frequencies for the categorical variables. The categorical characteristics were compared using the χ^2

and Fisher's exact tests for cell count <5. Patient's characteristics were compared using Student's *t*-test in the case of the continuous variables. Otherwise, a non-parametric test of Mann–Whitney *U* test was used. A two-tailed *P* < 0.05 was considered statistically significant. The software SPSS version 13.0 (SPSS Inc., Chicago, IL, USA) was employed for data storage and analysis.

Results

The study population consisted of 73 patients (54 men, mean age 55 ± 15 years). Patients had severe LV dysfunction (mean LVEF 17.5 ± 6%, range 10–35%) with extensive dilation (mean LVED volume 209 ± 81 mL). The QRS duration was prolonged ranging from 120 to 200 ms. The origin of cardiomyopathy was ischaemic in 59% of the patients and idiopathic in the remaining 41%. Before implantation, 66 patients were in NYHA functional class III and 7 in class IV. All the patients were in sinus rhythm at the time of study. A biventricular implantable cardioverter-defibrillator was implanted in 48 patients and a biventricular pacemaker in 25 patients. In all patients, it was technically possible to implant an LV lead into a cardiac vein running along with the LV free wall and to achieve acceptable capture threshold. A lateral branch was used in 37 patients (50.7%), a posterolateral branch in 23 patients (31.5%), and an anterolateral branch in 13 patients (17.8%). Right ventricular lead was positioned at the RVA in 50 patients (68%) and the RVHS in 23 patients (32%). The right atrial lead was implanted in the appendage in all patients. Adequate pacing and sensing properties of all leads were tested. Based on the Doppler echocardiographic measurements of transmitral flow 1 day after implantation, AV optimization was required in three patients (one in RVA group and two in RVHS group).

There were no device and lead-related complications in any patients.

Comparisons of baseline characteristics of the patients according to right ventricular lead location

A total of 73 patients were studied, including 50 patients in RVA pacing group and 23 patients in RVHS pacing group. Age, gender, the QRS duration and morphology, aetiology of the heart failure, and history of myocardial infarction were similar between groups (*Table 1*). In addition, no significant differences in the NYHA class and 6-MHWT distance were observed between two groups before cardiac resynchronization. Patients with RVA pacing had a longer PR interval than those with RVHS pacing. Before enrolment, LV dimensions, LV function, severity of mitral regurgitation, aortic velocity–time integral (Ao-VTI), and incidence of inter- and intraventricular dyssynchrony were also similar between groups (*Table 1*).

Relation of right ventricular lead location to clinical outcome after cardiac resynchronization

As an alternative for RVA during biventricular pacing, RVHS stimulation resulted in similar long-term improvement in NYHA class and 6-MHWT distance. They also had similar degree of QRS shortening after biventricular stimulation (*Table 2*). In order to determine the optimal combination of RV and LV pacing sites, we evaluated the role of RV pacing site in the patients with LV lead at lateral (LCV), anterolateral (ALCV), or posterolateral (PLCV) branches of cardiac venous system. Concerning the improvement in clinical status and QRS shortening, RVHS pacing was equivalent

Table 1 Comparison of the demographic, clinical, electrocardiographic, and echocardiographic characteristics of the heart failure patients with biventricular pacing according to right ventricular lead pacing site

Variable	RVA pacing (*n* = 50)	RVHS pacing (*n* = 23)	*P*-value
Age (mean ± SD, years)	57 ± 15	50 ± 15	0.06
Sex ratio (male/female, *n*)	36/14	18/5	0.63
Ischaemic aetiology (%)	62.5	52	0.38
History of myocardial infarction (%)	35	48	0.57
QRS duration (ms)	153 ± 25	149 ± 29	0.46
QRS morphology (LBBB/RBBB, *n*)	39/11	18/5	0.75
PR interval (ms)	206 ± 45	180 ± 43	0.027
NYHA functional class (III/IV, *n*)	46/4	20/3	0.22
6-Min walk distance (mean ± SD, m)	192 ± 82	184 ± 84	0.75
LVES volume (mean ± SD, mL)	169 ± 73	199 ± 80	0.12
LVED volume (mean ± SD, mL)	208 ± 75	210 ± 85	0.52
LVEF (mean ± SD, %)	17 ± 6	17 ± 5	0.70
Mitral regurgitation (moderate to severe, %)	60	52	0.77
IVMD (mean ± SD, ms)	49 ± 22	39 ± 19	0.08
Ts-SD (mean ± SD, ms)	34 ± 12	34 ± 10	0.86
Aortic velocity–time integral (mean ± SD, cm)	12.5 ± 4	12 ± 4	0.48

RVA, right ventricular apical area; RVHS, right ventricular high septal area; LBBB, left bundle branch block; RBBB, right bundle branch block; NYHA, New York Heart Association; LVES, left ventricular end-systolic; LVED, left ventricular end-diastolic; LVEF, left ventricular ejection fraction; IVMD, interventricular mechanical delay; Ts-SD, standard deviation of time to peak velocity among the 12 left ventricular segments.

Table 2 Comparison of the clinical and demographic characteristics of the heart failure patients with biventricular pacing divided based on the right ventricular and left ventricular pacing combinations

Variable	LCV stimulation site		P-value	ALCV stimulation site		P-value	PLCV stimulation site		P-value	Total		P-value
	RVA pacing	RVHS pacing		RVA pacing	RVHS pacing		RVA pacing	RVHS pacing		RVA pacing	RVHS pacing	
Increase in 6-MHWT distance >50 m (%)	61	64	0.65	77	35	0.04	40	86	0.01	70	70	0.96
Decrease in NYHA class by ≥1 point (%)	62	64	0.74	88	47	0.05	35	85	0.01	72	74	0.76
Decrease in LVESV by >15% (%)	63	62	0.78	75	32	0.02	30	72	0.01	65	64	0.76
Increase in stroke volume by >15% (%)	43	41	0.44	60	20	0.03	34	68	0.04	55	54	0.81
Increase in LVEF by ≥25% (%)	58	56	0.65	78	33	0.02	35	71	0.05	70	68	0.64
PostCRT severe mitral regurgitation (%)	6	5	0.57	0	10	0.05	14	1	0.01	7	6.5	0.66
IVMD after CRT (mean ± SD, ms)	31 ± 21	29 ± 26	0.82	29 ± 18	23 ± 15	0.60	24 ± 18	23 ± 15	0.80	28 ± 20	24 ± 21	0.46
Ts-SD after CRT(mean ± SD, ms)	29 ± 11	28 ± 15	0.87	23 ± 10	24 ± 9	0.85	24 ± 9	24 ± 8	0.92	28 ± 12	28 ± 13	0.95
Post-CRT Ao-VTI (mean ± SD, cm)	17.6 ± 4.5	19.5 ± 7	0.38	21 ± 5	12 ± 4	0.03	11 ± 4	20 ± 5	0.03	16.7 ± 7.2	16.7 ± 6.8	0.97
Post-CRT QRS shortening (mean ± SD, ms)	15.5 ± 7	17 ± 7	0.63	40 ± 20	14 ± 26	0.02	8 ± 2	14 ± 3	0.24	17 ± 7	15 ± 7	0.77
Post-CRT reduction in BNP >50% (%)	55	68	0.05	80	50	0.04	50	75	0.04	70	69	0.88

LCV, lateral cardiac vein; ALCV, anterolateral cardiac vein; PLCV, posterolateral cardiac vein; RVA, right ventricular apical area; RVHS, right ventricular high septal area; 6-MHWT, 6-min hall walk test; NYHA, New York Heart Association; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; CRT, cardiac resynchronization therapy; IVMD, interventricular mechanical delay; Ts-SD, standard deviation of time to peak velocity among the 12 left ventricular segments; Ao-VTI, aortic velocity−time integral; BNP, brain-type natriuretic peptide.

to RVA stimulation in combination with LCV pacing site. Compared with pacing from RVHS area, RVA stimulation was associated with better clinical outcome and more QRS shortening when it is combined with ALCV pacing. Furthermore, RVHS pacing combined with PLCV stimulation induced better clinical response than RVA–PLCV pacing combination (*Table 2*).

Effect of right ventricular lead pacing site on left ventricular reverse remodelling after cardiac resynchronization

At 6-month follow-up, biventricular stimulation resulted in similar reduction of LVES (40 ± 37 vs. 44 ± 34 ml, P = 0.66) and LVED (41 ± 40 vs. 43 ± 38 ml, P = 0.84) volumes in patients with RVA pacing and RVHS pacing. The proportion of the patients with significant reverse remodelling, defined as reduction in LVES volume by >15%, was also similar in two RV pacing groups. Increases in LVEF, stroke volume, and Ao-VTI were also comparable regardless of RV pacing location. Furthermore, there were no significant differences in the degree of the improvement in inter- and intra-ventricular dyssynchrony (*Table 2*).

When we separately assessed the effect of RV pacing site on echocardiographic parameters in three LV stimulation sites (LCV, ALCV, and PLCV), incidence of significant LV reverse remodelling and increase of LVEF by ≥25% was similar in two RV pacing groups when LV stimulation site was LCV. When LV stimulation site was ALCV, significant LV reverse remodelling and increase in LVEF by ≥25% was observed in a higher proportion of the patients with RVA-LV pacing configuration. When LV stimulation site was PLCV, RVHS pacing promoted LV reverse remodelling and LVEF improvement of ≥25% in higher percentage of the patients than the RVA pacing.

Effect of right ventricular lead pacing site on brain-type natriuretic peptide level after cardiac resynchronization

Overall, the baseline BNP levels and incidence of significant decrease in BNP level, defined as a decrease in BNP by >50%, were similar between RVA and RVHS pacing sites. Similar to echocardiographic data, however, the degree of improvement in BNP level was significantly different in RV pacing groups depending on the LV stimulation area. When the LV stimulation site was LCV, improvement in BNP level was marginally greater in RVHS pacing group relative to RVA group. However, there was a significant difference in proportion of the patients with significant decrease in BNP level between the RVA and RVHS pacing group when the LV was stimulated from the ALCV or PLCV. Decrease in BNP level of >50% was more frequent in RVA pacing group in combination with ALCV stimulation site. Furthermore, combination of RVHS–PLCV pacing was more likely to be associated with significant BNP level improvement after cardiac resynchronization than RVA–PLCV combination (*Table 2*).
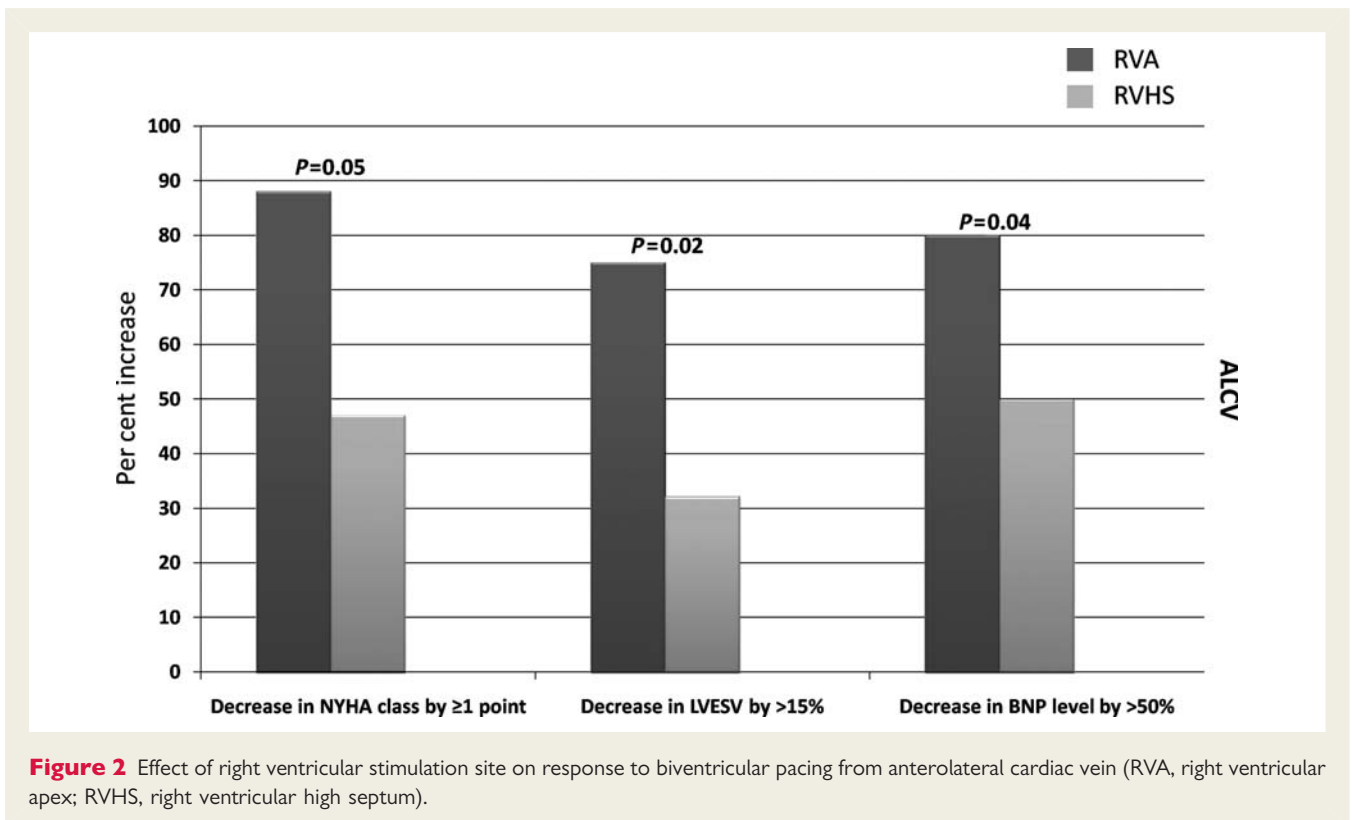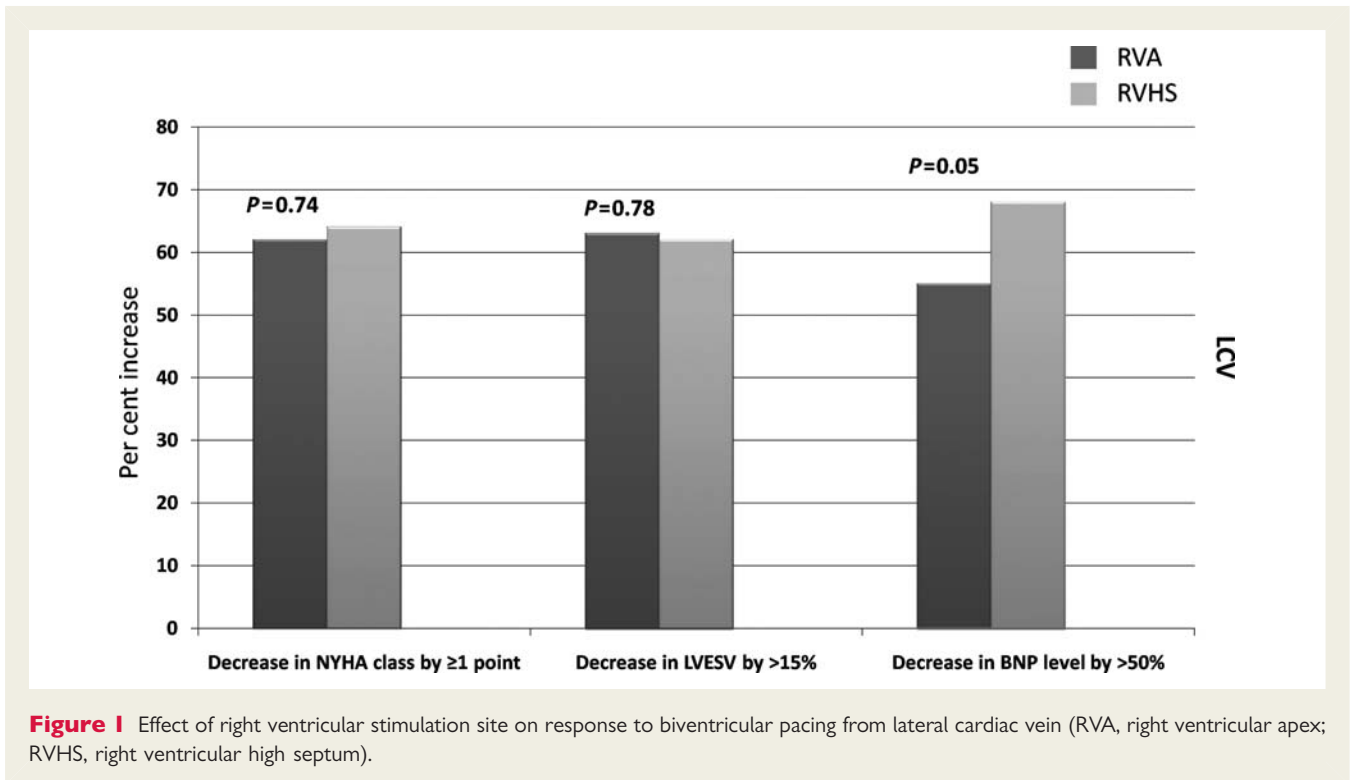
Discussion

The present study did not elicit any difference between RVA pacing and RVHS pacing in terms of overall long-term improvement in clinical outcome (decrease in NYHA class by ≥1 point), LV reverse remodelling (decrease in LVESV by >15%), and neurohormonal status (decrease in BNP level >50%) after CRT. When we separately assessed the significance of RV lead location in individual LV segments, RVA pacing produced clinical, echocardiographic, and neurohormonal responses similar to RVHS pacing in combination with LCV stimulation (*Figure 1*). However, RVA pacing was more efficacious than RVHS pacing when used in combination with ALCV site (*Figure 2*), and RVHS pacing was superior to RVA pacing in combination with PLCV site (*Figure 3*).

There are limited data regarding the optimal RV lead pacing location in patients who underwent CRT.[16–19] The available studies have mainly evaluated acute haemodynamic changes and/or QRS duration in relation to the RV lead position. Hay *et al.*[16] reported the results of a comparison of the acute haemodynamic effects of biventricular pacing using RVA and RVHS sites in combination with LCV stimulation in nine patients with CHF, AF, and AV block. The RVHS lead position had no or less beneficial impact than RVA on CRT. Leclercq *et al.*[17] defined optimal biventricular pacing mode by the degree of QRS narrowing and found that biventricular pacing with the RV lead inserted in the RV outflow tract was superior in 11 patients (61%) and in RVA in the remaining 7 (39%) patients. However, some studies[20,21] have indicated that the degree of QRS narrowing seems to be a controversial predictor of clinical improvement and invasive haemodynamic or echocardiographic assessment appears to be more relevant in this context. Shimano *et al.*[18] also compared the acute haemodynamic response to biventricular pacing at two different RV stimulation sites: RVA and RVHS. There are many similarities between the results of this study and ours. In both studies, RVHS stimulation had no overall advantage over the RVA during biventricular pacing. These studies also demonstrated that RVA was superior to RVHS when used together with ALCV pacing. Contrary to the Shimano's study, we found that RVHS produced better long-term response to CRT than RVA in combination with PLCV pacing and RVA is equivalent to RVHS in conjuncture with LCV pacing. Until now, only one study[19] evaluated the effect of RV lead pacing site on long-term outcome of CRT, in which mid-septal positioning of the RV lead led to a significant reduction in LVED dimension compared with RVA location 12 months after biventricular device implantation. However, no significant differences were observed in terms of the improvement in NYHA class, VO2 max, and LVEF in this study.

To the best of our knowledge, the present study is the first of this kind to evaluate the effect of RV lead position on long-term clinical, echocardiographic, and neurohormonal responses to CRT in individual LV segments. The lack of advantage of the RVHS over the RVA in combination with LCV stimulation may be explained by a similar distance between RVA or RVHS and LCV stimulation sites. When LV was stimulated from the ALCV, the greater beneficial effect of CRT in patients with the RVA relative to the RVHS stimulation sites may be explained more appropriate LV–RV interlead distance in the former than the latter patients. A similar mechanism may be responsible for the superior efficacy of the biventricular pacing from the RVHS vs. RVA in combination with PLCV. The importance of RV–LV interlead distance in predicting the acute haemodynamic response to CRT has also

Figure 1 Effect of right ventricular stimulation site on response to biventricular pacing from lateral cardiac vein (RVA, right ventricular apex; RVHS, right ventricular high septum).

Figure 2 Effect of right ventricular stimulation site on response to biventricular pacing from anterolateral cardiac vein (RVA, right ventricular apex; RVHS, right ventricular high septum).

been evaluated in a radiographic study.[22] The horizontal component of the LV–RV interlead distance on the lateral chest radiograph was greater in acute responders to CRT (14.4 ± 5.4 cm) compared with non-responders (9.2 ± 5.8 cm, $P = 0.002$).

The idea that RVHS pacing may offer more beneficial effects than RVA during biventricular pacing is based on the prior studies that explored alternative sites for univentricular RV pacing.[9,13] These data suggested that RVA pacing might be
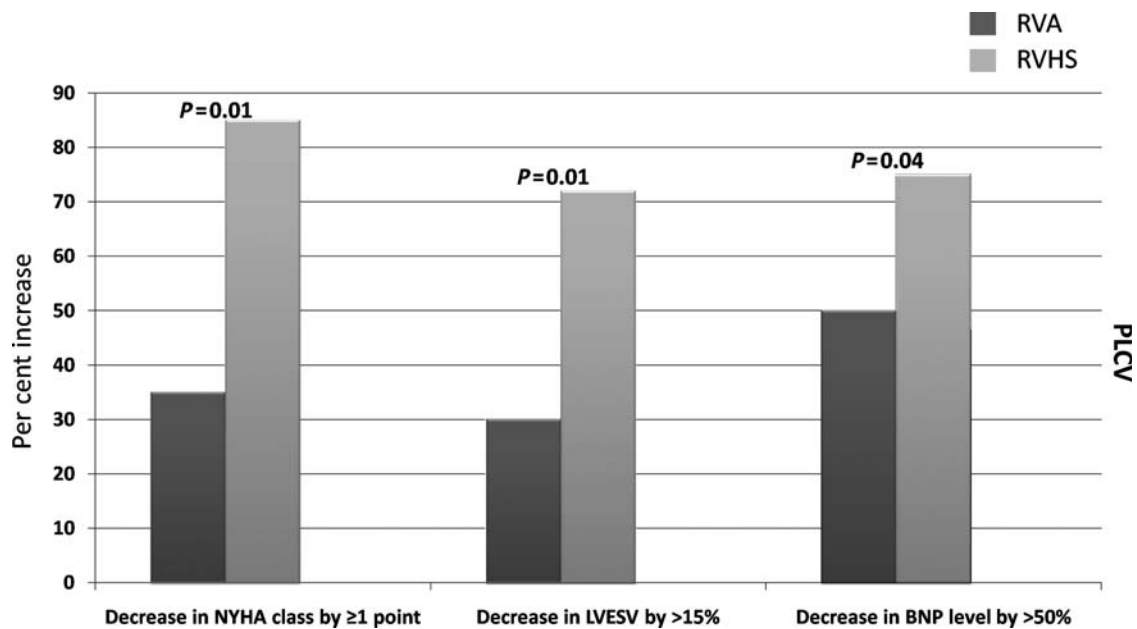
Figure 3 Effect of right ventricular stimulation site on response to biventricular pacing from posterolateral cardiac vein (RVA, right ventricular apex; RVHS, right ventricular high septum).

detrimental to LV function, presumably because bypass of the His-Purkinje system produces dyssynchronous LV contraction. Results of acute haemodynamic studies, although controversial, have shown increased cardiac output as a result of RVHS pacing, relative to RVA pacing.[12,13] However, no symptomatic improvement or haemodynamic benefit was noted after 3 months of RVHS pacing, in comparison with RVA pacing.[23] Taken together, no overall benefit was demonstrated for the RVHS pacing compared with the RVA pacing both in univentricular and biventricular pacing studies.

Limitations

The results of the present study should be interpreted in the light of certain limitations. First, non-random selection of the patients for RV lead location may have influenced results of the study. Another limitation of the study is related to the small number of the patients in some LV pacing site.

Clinical implications

The results of the present study indicate that RV lead location is not an important determinant of CRT response when LV can be stimulated from the LCV. However, optimization of the RV lead location should be an important consideration when it is not possible to place LV lead in the LCV.

Several studies have evaluated the role of LV lead position on the haemodynamic response to the CRT and the LV free wall was shown to be optimal pacing site.[3–8] However, because of anatomic and technical limitations, it may not be possible to place the LV lead at these optimal sites, which can result in diminished response to CRT. In contrast, an electrode implanted in the RV is much easier to re-position. Therefore, our recommendation is to first implant LV lead and then, based on its location, to aim

for a specific area for the RV lead placement. In addition, in the case of CRT non-response, it is likely to improve the CRT system function by relocating the RV lead position to obtain the ideal RV–LV pacing configuration. However, confirmation of these findings in a prospective randomized study is reasonable to determine general guidelines.

Conclusions

Overall, biventricular pacing resulted in the similar degree of clinical improvement and LV reverse remodelling in patients with advanced heart failure regardless of RV lead pacing location. However, the effect of RV pacing site on CRT response varies depending on LV stimulation site. There was no difference in the responder rate between RVA and RVHS in biventricular combination with LCV pacing. When stimulation site is ALCV, CRT response was observed in a higher proportion of patients with RVA pacing. When stimulation site was PLCV, CRT responder was seen in a greater proportion of patients with RVHS pacing.

Conflict of interest: none declared.

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