Electrocardiographic patterns in biventricular pacing delivered by second-generation cardiac resynchronization devices

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ABSTRACT

Background: With increasing use of cardiac resynchronization therapy (CRT), treating physicians should be familiar with different electrocardiographic (ECG) patterns of left ventricular (LV) lead and biventricular (BiV) pacing. However, there are a few publications on ECG patterns during BiV pacing.
Purpose: This study was sought to determine different ECG patterns in patients with BiV pacing.
Methods: Twelve-lead ECGs during BiV pacing (right ventricular leads at apex and LV leads in one of the lateral coronary veins) were analyzed in 181 consecutive patients (121 male; mean age, 62.0 ± 13.5 years) with advanced heart failure and baseline left bundle branch block pattern after at least 6-month of uncomplicated CRT.
Results: During BiV pacing, 65% of the patients showed a dominant R wave in V1. There was a right axis deviation in 57% in frontal plane. However, a left superior axis emerged in 34% and normal frontal plane axis in 9%. Sequential BiV pacing (73% vs. 58%, P = 0.04) and pacing from posterolateral coronary vein (80% vs. 60%, p = 0.045) were more likely to present with a dominant R wave in V1. In sequential pacing, AV interval was significantly longer in patients with negative complex in V1 than in those with positive complex (124 ± 21 vs. 116 ± 8.0, p = 0.005). A Q/q wave was detected in 85% of patients in lead I and 78% in lead aVL.
Conclusions: BiV pacing from lateral coronary venous branches and right ventricular apex characteristically presented with dominant R wave in V1, Q/q wave in leads I and aVL, and right or left superior axis. However, a negative complex in V1, QRS axis in other quadrants, and lack of Q/q wave in leads I and aVL did not necessarily indicate a problem.
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1. Introduction

With increasing use of cardiac resynchronization therapy (CRT), treating physicians should be familiar with different electrocardiographic (ECG) patterns of left ventricular (LV) lead pacing to recognize appropriate capture and lead malfunction. This requires a thorough understanding of the typical ECG signatures of various LV and right ventricular (RV) sites [1—3].

Univentricular pacing from RV or LV is relatively straightforward to recognize on an ECG. However, ECG in biventricular (BiV) pacing with both RV and LV stimulation sites, represents a summated vector of the individual site of activation. The interpretation of the 12-lead ECG in patients with BiV devices requires detailed knowledge of device specifications and familiarity with the multiplicity of clinical situations. Data on ECG patterns during BiV pacing are still inadequate [4,5]. This study was sought to determine different ECG patterns in patients with BiV pacing.

2. Methods

Patient population: The study population consisted of 181 consecutive patients (121 male, 54% ischemic etiology, 62.0 ± 13.5 years) with New York Heart Association (NYHA) class III/IV drug-refractory heart failure, ejection fraction $\leq 35\%$, and left bundle branch block (LBBB) pattern (≥ 120 ms). All patients had BiV pacing delivered by CRT devices at least for 6-month. Patients with baseline right bundle branch block, RV and/or LV lead malfunctions, bifocal RV pacing, and atrial fibrillation were excluded. The study

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was approved by the institutional review board committee and written informed consent was obtained from all patients.

Twelve-lead ECG: Standard supine 12-lead ECGs (25 mm/s, 10 mm/mV) were recorded at least 6-month after CRT. ECGs were analyzed blinded to CRT settings (M.H and A.F). All measurements were made with the use of digital calipers at 200% magnification calibrated for paper speed 25 mm/s.

Normal frontal plane axis was defined as QRS axis between -30 and $+90°$. QRS axis between -30 and $-90°$ referred to as left superior axis (LSA). QRS axis between $+90$ and $-90°$ represented the right axis deviation (RAD); right inferior axis (RIA) $+90$ and $180°$ and right superior axis (RSA) was between 180 and $-90°$ [5]. Ventricular activation in each lead was characterized by 9 possible QRS morphology: (1) R; (2) RS; (3) Rs; (4) rS; (5) QS; (6) qR; (7) QR; (8) Qr; and (9) QRS [5].

Device information: Second-generation CRT pacemaker (CRT-P) and CRT defibrillator (CRT-D) devices with ability for separate programming of RV and LV pacing were included. All patients had RV lead implanted in the apex. LV lead was implanted in one of lateral branches of the coronary venous system: lateral cardiac vein (LCV), anterolateral cardiac vein (ALCV), and posterolateral cardiac vein (PLCV). The choice of vein depend on acceptable pacing threshold, lack of phrenic nerve stimulation, and vein diameter. Anterior and middle cardiac veins were not used for LV lead implantation because of preferential septal stimulation. As a standard practice, all electrodes were placed in basal to mid portion of the LV free wall.

Data 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 chi-square and Fisher's exact tests for cell count less than 5. Patient's characteristics were compared using Student's t-test in the case of the continuous variables with normal distribution. Otherwise, a non-parametric test of Mann-Whitney U test was used. A two-tailed P-value less than 0.05 was considered statistically significant. The software SPSS version 21.0 (SPSS Inc., Chicago, IL, USA) was employed for data storage and analysis.

3. Results

The study population consisted of 181 patients with CRT device. One patient with epicardial LV lead was excluded. The mean age was 62 ± 13.5 years. Overall, 67% were male, and 54% had an ischemic cardiomyopathy (ICM). In this cohort, all patients had RV lead implanted in the apex. LV leads were implanted in LCV (Fig. 1) in 50%, ALCV (Fig. 2) in 20% and PLCV (Fig. 3) in 30%. In this series, we had no case of LV lead implantation in vein collaterals. CRT delivered either as simultaneous (n = 78) or sequential (LV first, n = 103) BiV pacing. In total, BiV stimulation resulted in dominant R wave in V1 in 65% of the patients; a Q/q wave was detected in 85% of patients in lead I and 78% in lead aVL. There was a right axis deviation in 57% in frontal plane (RSA: 34% and RIA: 23%). However, a LSA was emerged in 34% and normal frontal plane axis in 9%.

ECG patterns during simultaneous BiV pacing from RV apex and ALCV: lead I showed a Q/q in 87% of the patients. Q/q wave was followed by positive deflection in 14% of the patients. Similarly, a Q/q wave in lead aVL was observed in 93% of patients; there was a positive deflection in 25% of these cases. Lead V1 showed a dominant R wave in 55% of the patients.
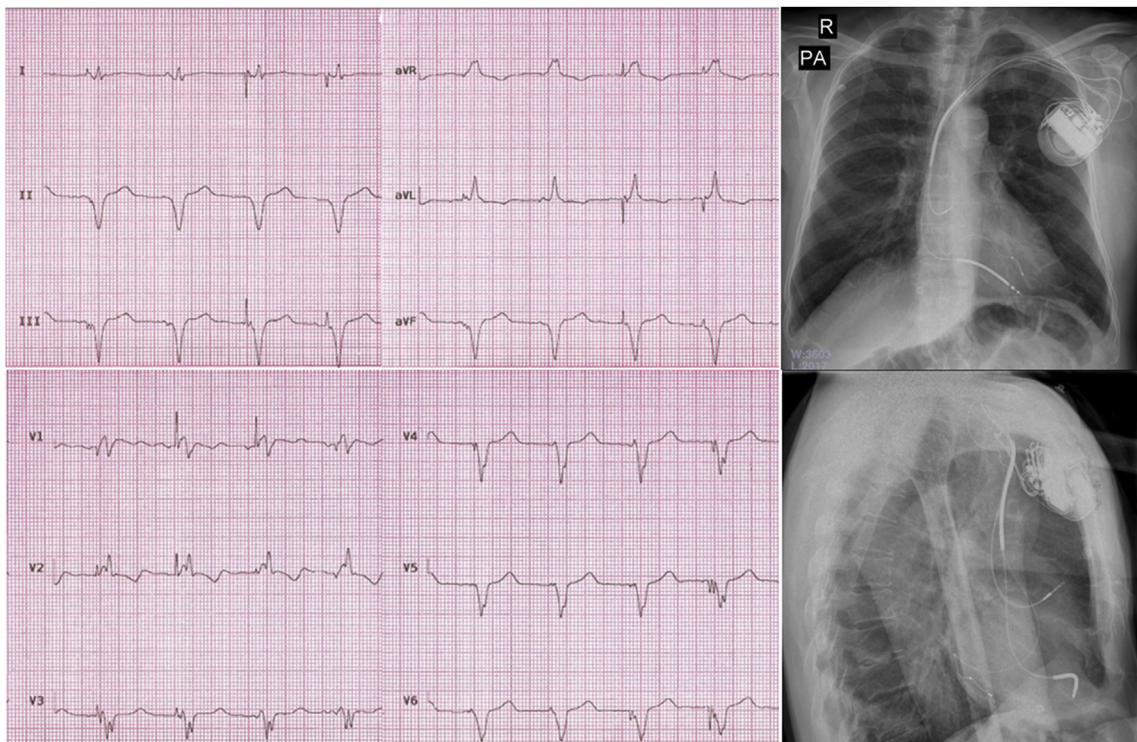
ECG patterns during simultaneous BiV pacing from RV apex and LCV: a Q/q in lead I was observed in 80% of patients. In 16% of the cases, Q/q wave was followed by a positive deflection. Similarly, a Q/q wave was detected in 77% of patients in lead aVL; 16% of the cases showed positive deflection after Q/q wave. Lead V1 showed a
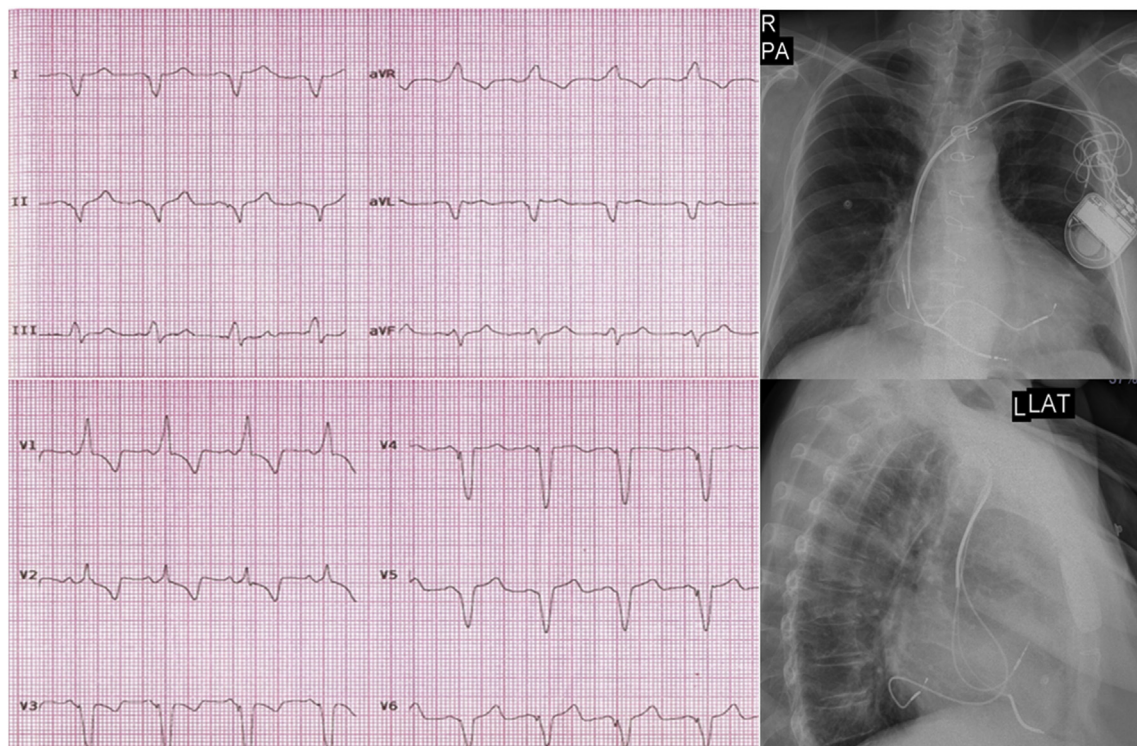
Fig. 1. Twelve-lead ECG and chest x-ray (PA and lateral views) of a patient with CRT-D with LV lead in LCV and RV lead in RVA. ECG is characterized by dominant R wave in V1, QS in leads I and aVL, and right superior axis.
(Abbreviations: PA: posteroanterior; CRT-D: defibrillator cardiac resynchronization device; LV: left ventricle; LCV: lateral coronary vein; RV: right ventricle; RVA: right ventricular apex).
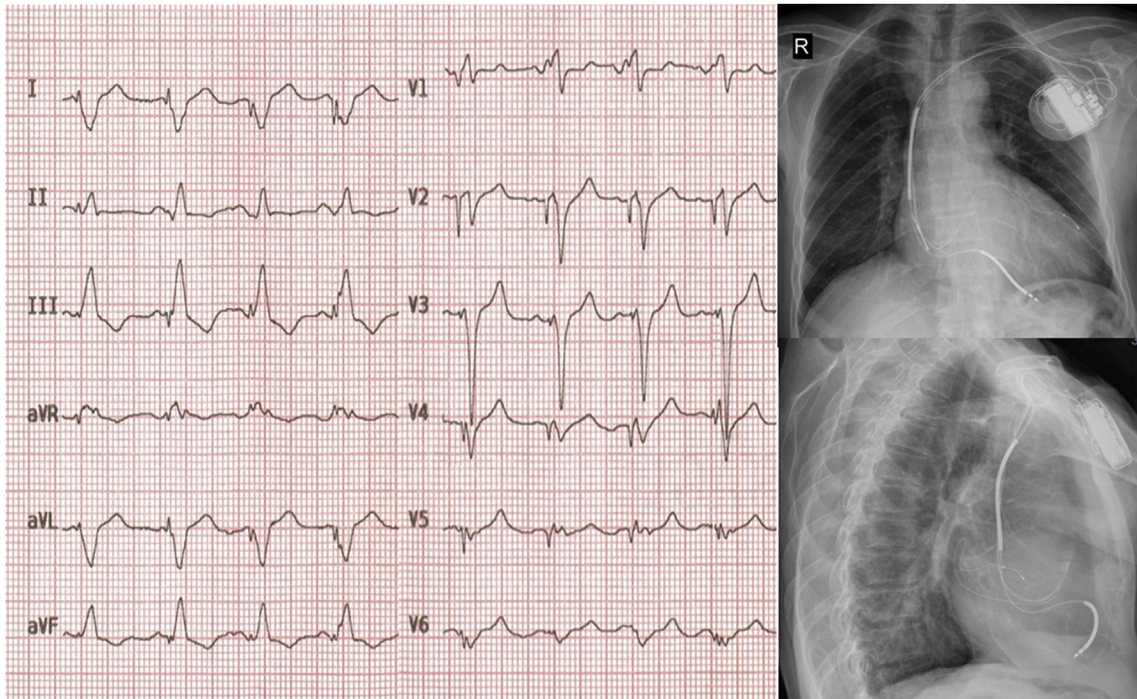
Fig. 2. Twelve-lead ECG and chest x-ray (PA and lateral views) of a patient with CRT-D with LV lead in ALCV and RV lead in RVA. ECG is characterized by dominant R wave in V1, QS in leads I and aVL, and right inferior axis.
(Abbreviations: PA: posteroanterior; CRT-D: defibrillator cardiac resynchronization device; LV: left ventricle; ALCV: anterolateral coronary vein; RV: right ventricle; RVA: right ventricular apex).

Fig. 3. Twelve-lead ECG and chest x-ray (PA and lateral views) of a patient with CRT-D with LV lead in PLCV and RV lead in RVA. ECG is characterized by dominant R wave in V1, QS in leads I and aVL, and left superior axis.
(Abbreviations: PA: posteroanterior; CRT-D: defibrillator cardiac resynchronization device; LV: left ventricle; PLCV: posterolateral coronary vein; RV: right ventricle; RVA: right ventricular apex).

dominant R wave in 60% of the patients.

ECG patterns during simultaneous BiV pacing from RV apex and PLCV: a Q/q in lead I was observed in 76% of patients. In this group, Q/q wave was followed by positive deflection in only 9% of the cases. On the other hand, a Q/q wave was detected in 56% of patients in lead aVL; 10% of the cases showed positive deflection after Q/q wave. Sixty-six percent (66%) of the patients showed a dominant R wave was in lead V1.

Frontal plane QRS axis during simultaneous BiV pacing: frontal plane QRS axis usually points to the right superior quadrant (31%) and the left superior quadrant (37%). Right inferior axis deviation and normal axis were seen in 23% and 9%, respectively.

ECG patterns during sequential BiV pacing from RV apex and ALCV: a Q/q in lead I was observed in 91% of patients. There was positive deflection after Q/q wave in 24% of the ECGs in this group. Prevalence of Q/q wave was higher in aVL (95%). Q/q wave was followed by a positive deflection in 40% of the patients. Lead V1 showed a dominant R wave in 60% of the patients.

ECG patterns during sequential BiV pacing from RV apex and LCV: a Q/q wave was observed in 81% of patients. Q/q wave was followed by a positive deflection in 24% of the patients. However, Q/q wave was seen in 78% of the patients in aVL. There was a positive deflection after Q/q wave in 32% of the patients. Lead V1 showed a dominant R wave in 73% of the patients.

ECG patterns during sequential BiV pacing from RV apex and PLCV: Lead I showed a Q/q wave in 77% of the patients. There was a positive deflection after Q/q wave in 36% of the patients. Lead aVL showed a Q/q wave in 66% of the patients; a positive deflection after Q/q wave was present in 26% of the patients. Lead V1 showed a dominant R wave in 80% of the patients.

Frontal plane QRS axis during sequential BiV pacing: frontal plane QRS axis usually points to the right superior quadrant (39%) or occasionally to the left superior quadrant (29%). Right inferior axis and normal axis were seen in 22% and 10%, respectively.

Effect of clinical characteristics and device features on QRS morphology in V1: Among the clinical characteristics age, gender, and underlying heart disease were studied. Age was dichotomized to <60 years and ≥60 years. Fifty-nine percent of younger patients showed a dominant R wave in lead V1 whereas 68% of older patients demonstrated a dominant R wave in the same lead. However, the difference did not reach statistical significance (P = 0.39). Morphology of QRS complex was similar in men and women (positive QRS complex: 58% vs. 68%, P = 0.25). Similar to age and gender, underlying heart disease had no significant effect on morphology of V1 (DCM group: 58% vs. ICM group: 70%, P = 0.12).

Among the device features effect of LV lead position, VV interval, and AV interval were investigated. While moving LV pacing site from the ALCV to the PLCV and getting more closer to RV pacing site, we found a higher rate of dominant R wave in V1 (Table 1).

However, effect of LV lead pacing site on QRS morphology in V1 achieved statistical significance only in sequential BiV stimulation group (ALCV: 80% vs. PLCV: 60%, P = 0.04). Compared with simultaneous BiV stimulation, sequential stimulation increased the prevalence of dominant R wave in V1 (73% vs. 58%, P = 0.04). Closer look in the sequential BiV pacing showed a significantly longer AV interval in patients with negative complex in V1 than in those with positive complex (124 ± 21 vs. 116 ± 8.0, p = 0.005). However, AV interval was similar in patients with simultaneous BiV stimulation with negative and positive QRS complexes in V1 (117 ± 8.0 vs. 117 ± 10, p = 0.86).

Effect of LV lead location on mean frontal plane axis: Effect of LV lead location on mean frontal plane axis were summarized in the Table 2.

4. Discussion

The major finding of the present study can be summarized as follows: (1) during simultaneous BiV stimulation from the RVA and the lateral branches of the coronary venous system, the QRS complex is often dominantly positive in lead V1 and the frontal plane QRS axis usually points to the left and right superior quadrants; (2) during sequential BiV stimulation from the RVA and the lateral coronary venous branches, QRS complex of the lead V1 is dominantly positive in majority of the patients and the frontal plane QRS axis is usually in the right superior quadrant and occasionally in the left superior quadrant; (3) a negative complex in V1 is mainly caused by marked latency or slow conduction during simultaneous BiV stimulation and ventricular fusion with intrinsic rhythm during sequential BiV pacing.

BiV stimulation with RV lead in the apex: By this arrangement, reported incidence of a dominant R wave in lead V1 varies from 50% to nearly 100% [1]. The frontal plane QRS axis usually resides in the right superior quadrant and may occasionally points to the left superior quadrant. A mean frontal plane axis in the other 2 quadrants is distinctly unusual but does not necessarily indicate a problem.

Table 2
QRS frontal plane axis according to the LV lead location.

LV lead Location	Mean Frontal Plane Axis			
	RSA	RIA	LSA	NLA
ALCV	31%	44%	17%	8%
LCV	29%	22%	36%	13%
PLCV	44%	9%	43%	4%

Abbreviations: RSA: right superior axis; RIA: right inferior axis; LSA: left superior axis; NLA: normal axis; LV: left ventricle; ALCV: anterolateral coronary vein; LCV: lateral coronary vein; PLCV: posterolateral coronary vein.

Table 1
Effect of left ventricular lead stimulation site on QRS morphology in V1.

VV group	LV lead location	QRS morphology in V1		P-value[a]
		Dominant R wave	Dominant negative complex	
Sequential	ALCV	60%	40%	0.04
	LCV	73%	27%	
	PLCV	80%	20%	
Simultaneous	ALCV	55%	45%	0.38
	LCV	60%	40%	
	PLCV	66%	34%	
Total	ALCV	58%	42%	0.17
	LCV	62%	38%	
	PLCV	72%	28%	

Abbreviations: VV: interventricular pacing interval; LV: left ventricle; ALCV: anterolateral coronary vein; LCV: lateral coronary vein; PLCV: posterolateral coronary vein.
[a] Comparison between ALCV and PLCV group.

Sweeney et al. [5] evaluated the ECGs of 202 consecutive patients who received simultaneous BiV pacing at least for 6 months. A dominant R wave in V1 and right axis deviation was observed in 53% and 67% (RSA: 58%, RIA: 9%) of the patients, respectively. In contrast, 29% had LSA and 3% had normal axis. This study was limited by the fact that locations of RV and LV leads were not stated and only simultaneous BiV pacing was used. Refaat et al. [4] analyzed the ECGs of 54 patients with CRT device (RV apex). In this study, 93% (n = 50) showed a dominant R wave in lead V1 and the position of the LV lead was verified to be in the LCV (n = 30) or PLCV (n = 20). Mean frontal plane axis was in right superior quadrant (64%) or left superior quadrant (34%). Herweg et al. [6] evaluated 40 CRT-responsive patients. Only patients with LV leads in the LCV or PLCV and RV lead in the apex were included. A total of 31 of 40 patients (78%) showed a dominant R-wave in lead V1 during simultaneous BiV pacing. After AV/VV optimization, 35 of 40 patients (87.5%) showed a dominant R-wave in V1.

Present study showed a dominant R-wave in lead V1 in 65% of the patients (73% in sequential group vs. 58% in simultaneous group). The mean frontal plane paced QRS axis usually points to right superior (34%) or left superior quadrant (34%). BiV pacing often shifts QRS axis to the right superior quadrant (39%) in sequential group and left superior quadrant (37%) in simultaneous group. While moving LV pacing site from ALCV toward PLCV, there is clear increase in prevalence of dominant R wave in V1. However, this difference reached statistical significance only in sequential BiV stimulation group. This finding may be explained by the fact that most of LV free wall is activated by LV lead impulse while pacing from PLCV, however, pacing from ALCV permits a higher contribution of RV lead impulse in LV free wall activation. Furthermore, LV lead location had a marked influence on frontal plane QRS axis. Pacing from ALCV shifted QRS axis markedly in a counterclockwise fashion to the right inferior quadrant (44%) whereas mean frontal plane axis often directed to the right superior axis (44%) in PLCV pacing. LCV pacing showed more balanced distribution of frontal plane axis in different quadrants.

Negative QRS in lead V1 during BiV pacing with RV lead at the apex: a negative QRS (LBBB pattern) in V1 during BiV pacing from RV apex (Fig. 4) may be due to: LV lead malfunction, significant LV latency or slow conduction from stimulation site, ventricular fusion with intrinsic QRS complex, pacing from middle cardiac vein or anterior interventricular vein, incorrect recording of lead V1 from a higher intercostal space (3rd or 2nd space), and bifocal RV pacing [1].

In the present study, malfunctioning LV leads and bifocal RV pacing were excluded. There was no pacing from middle cardiac vein or anterior interventricular vein. All ECGs were recorded by experienced staffs according to AHA recommendations for
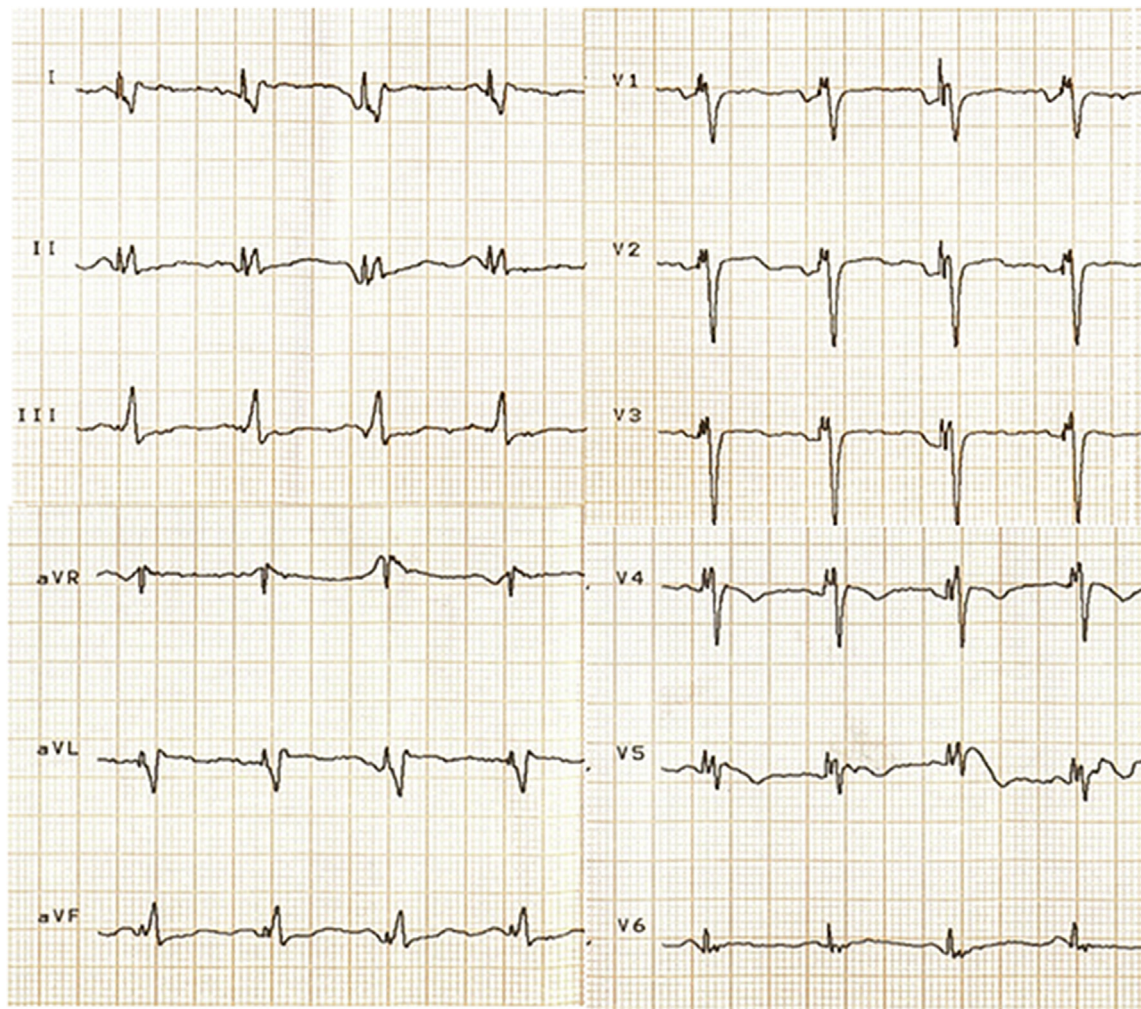
Fig. 4. Twelve-lead ECG of simultaneous biventricular pacing from ALCV and RVA. ECG is characterized by dominant negative complex in V1, QS in leads I and aVL, and right inferior axis.
(Abbreviations: ALCV: anterolateral coronary vein; RVA: right ventricular apex).

standard 12-lead ECG recording [7]. Therefore, negative QRS (LBBB pattern) in V1 during BiV pacing is mainly explained by marked LV latency/slow conduction and ventricular fusion. Compared to the sequential pacing group, simultaneous pacing group had a lower rate of dominant positive R wave in V1 (58% vs. 73%). This finding indicate that negative QRS in V1 in simultaneous pacing is mainly explained by prolonged LV latency that can be corrected by advancing LV pacing before RV pacing. Unfortunately, we have no data on actual prevalence of prolonged latency in our population. In the study of Herweg et al. [6], a negative QRS complex in lead V1 during simultaneous biventricular pacing predicted LV latency ≥ 40 ms with a sensitivity of 80% and a specificity of 97%. To investigate underlying causes of negative QRS in V1 during sequential BiV pacing, we compared the programmed AV intervals in patients with positive and negative QRS in V1. In sequential BiV pacing, AV interval was significantly longer in patients with negative complex in V1 than in those with positive complex. This finding indicates that ventricular fusion with native conduction may explain to some extent the presence of a negative QRS complex in V1 in patients with sequential BiV pacing and programmed long AV intervals. Vatasescu et al. [8] performed contact electro-anatomical mapping in 15 patients with echocardiographically optimized CRT. Fusion with intrinsic depolarization was found in 8 of 15 (53%) patients. The intrinsic PR interval was shorter in patients with fusion (164 ms vs. 234 ms, p = 0.006).

Except for the previously mentioned causes, a negative QRS in V1 probably reflects LV intramyocardial conduction delay (LVICD) and does not necessarily indicate LV lead malfunction [2]. LVICD is essentially a diagnosis of exclusion.

Q or q wave in leads I or aVL during BiV stimulation: although a Q/q wave may also occur during monochamber RV apical pacing in leads I and aVL, presence of Q/q wave (especially when followed by positivity) characteristically indicates LV capture in BiV pacing. Georger et al. [9] reported presence of a q wave in 17 of 18 patients during BiV pacing with RV lead at apex. However, this finding has not been reproduced in larger studies. Sweeney et al. [5] observed post-CRT Q wave emergence in leads I and aVL in 71% and 29%, respectively. In our study, a Q/q wave was detected in 85% of patients in lead I and 78% in lead aVL. These discrepancies may be related to differences in the underlying cause of cardiomyopathy, LV lead location, and programmed VV intervals.

Biventricular pacing with RV lead in nonapical position: There are limited number of the studies in this setting. In a series of more than 100 patients, Barold et al. [10] found that during BiV pacing with the RV lead in the right septal area or outflow tract and the LV lead in the PLCV, the paced QRS in lead V1 often shows LBBB pattern and the frontal plane paced QRS axis is often directed to the right inferior quadrant. This may create a problem in trouble-shooting because the ECG may resemble that of univentricular RV septal or outflow tract pacing with an LBBB pattern and right inferior frontal axis deviation mimicking LV lead malfunction.

Utility of ECG in AV and VV interval optimization: there are several methods to optimize AV and VV intervals during BiV pacing. ECG method offers a noninvasive, simple, and reproducible method to boost CRT device performance. Vidal method is a simple approach to optimize VV interval [11]. In this technique, the first time interval (T1) was measured from the pacing spike (LV lead pacing) to the onset of the earliest fast deflection of the QRS complex in the precordial leads. The second time interval (T2) was measured between pacing spike (RV lead pacing) and the earliest fast deflection of the QRS complex in the precordial leads. The difference between T1 and T2 was considered the optimal time delay to depolarize the LV simultaneously from the lateral wall and septum.

Majority of the current techniques on AV interval optimization

are performed by combination of echo and ECG [12]. ECG patterns during AV interval optimization is not as important as VV interval optimization.

5. Limitations

Results of the present study should be interpreted in the light of certain limitations. First, there were no ECGs of isolated LV pacing to evaluate for prolonged LV latency/slow conduction and no ECGs of shorter AV interval to rule out the ventricular fusion with native conduction. However, we looked in to LV latency by comparing simultaneous and sequential BiV pacing and investigated the ventricular fusion by comparing AV intervals in sequential BiV pacing group with and without dominant R wave in V1. Second, all patients had RV lead implanted at the apex, therefore, results of the present study are not applicable to BiV pacing with RV lead in the other locations. Third, we have no data on correlation between ECG and echo data along with clinical outcome.

6. Conclusions

BiV pacing from lateral coronary venous branches and RV apex characteristically presented with dominant R wave in V1, Q/q wave in leads I and aVL, and right or left superior axis. However, marked LV latency/slow conduction and ventricular fusion may change this typical pattern and simulate LV lead malfunction or pacing from middle cardiac vein/anterior interventricular vein.

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Conflict of interest

None declared.

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References

[1] Barold SS, Herweg B. Usefulness of the 12-lead electrocardiogram in the follow-up of patients with cardiac resynchronization devices. Part I Cardiol J 2011;18(5):476—86.
[2] Barold SS, Herweg B. Usefulness of the 12-lead electrocardiogram in the follow-up of patients with cardiac resynchronization devices. Part II Cardiol J 2011;18(6):610—24.
[3] Barold SS, Herweg B, Giudici M. Electrocardiographic follow-up of biventricular pacemakers. Ann Noninvasive Electrocardiol 2005;10(2):231—55.
[4] Refaat M, Mansour M, Singh JP, Ruskin J, Heist EK. Electrocardiographic characteristics in right ventricular vs biventricular pacing in patients with paced right bundle-branch block QRS pattern. J Electrocardiol 2011;44(2):289—95.
[5] Sweeney MO, van Bommel RJ, Schalij MJ, Borleffs CJ, Hellkamp AS, Bax JJ. Analysis of ventricular activation using surface electrocardiography to predict left ventricular reverse volumetric remodeling during cardiac resynchronization therapy. Circulation 2010;121(5):626—34.
[6] Herweg B, Ali R, Ilercil A, Madramootoo C, Cutro R, Weston MW, et al. Site-specific differences in latency intervals during biventricular pacing: impact on paced QRS morphology and echo-optimized V-V interval. Pacing Clin Electrophysiol 2010;33:1382—91.
[7] Kligfield P, Gettes LS, Bailey JJ, Childers R, Deal BJ, Hancock EW, et al., American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. Recommendations for the standardization and interpretation of the electrocardiogram: part I: the electrocardiogram and its technology a scientific statement from the american heart association electrocardiography and arrhythmias committee, council on clinical cardiology;

the american college of cardiology foundation; and the heart rhythm society endorsed by the international society for computerized electrocardiology. J Am Coll Cardiol 2007;49(10):1109–27.

[8] Vatasescu R, Berruezo A, Mont L, Tamborero D, Sitges M, Silva E, et al. Midterm 'super-response' to cardiac resynchronization therapy by biventricular pacing with fusion: insights from electro-anatomical mapping. Europace 2009;11: 1675–82.

[9] Georger F, Scavee C, Collet B. Specific electrocardiographic patterns may assess left ventricular capture during biventricular pacing. Pacing Clin Electrophysiol 2002;25:56 [abstract].

[10] Barold SS, Giudici MC, Herweg B, Curtis AB. Diagnostic value of the 12-lead electrocardiogram during conventional and biventricular pacing for cardiac resynchronization. Cardiol Clin 2006;24:471–90.

[11] Vidal B, Tamborero D, Mont L, Sitges M, Delgado V, Berruezo A, et al. Electrocardiographic optimization of interventricular delay in cardiac resynchronization therapy: a simple method to optimize the device. J Cardiovasc Electrophysiol 2007;18:1252–7.

[12] Meluzín J, Novák M, Müllerová J, Krejcí J, Hude P, Eisenberger M, et al. A fast and simple echocardiographic method of determination of the optimal atrioventricular delay in patients after biventricular stimulation. Pacing Clin Electrophysiol 2004;27:58–64.