VIA MEDICA

REVIEW ARTICLE

Usefulness of the 12-lead electrocardiogram in the follow-up of patients with cardiac resynchronization devices. Part I

S. Serge Barold, Bengt Herweg

Florida Heart Rhythm Institute, Tampa, Florida, USA

Abstract

Cardiac resynchronization therapy (CRT) has added a new dimension to the electrocardiographic evaluation of pacemaker function. During left ventricular (LV) pacing from the posterior or posterolateral coronary vein, a correctly positioned lead V1 registers a tall R wave and there is right axis deviation in the frontal plane with few exceptions. During simultaneous biventricular stimulation from the right ventricular (RV) apex and LV site in the coronary venous system, the QRS complex is often positive (dominant) in lead V1 and the frontal plane QRS axis usually points to the right superior quadrant and occasionally the left superior quadrant. The reported incidence of a dominant R wave in lead V1 during simultaneous biventricular pacing (RV apex) varies from 50% to almost 100% for reasons that are not clear. During simultaneous biventricular pacing from the posterior or posterolateral coronary vein with the RV lead in the outflow tract, the paced QRS in lead V1 is often negative and the frontal plane paced QRS axis is often directed to the right inferior quadrant (right axis deviation).

A negative paced QRS complex in lead V1 during simultaneous biventricular pacing with the RV lead at the apex can be caused by incorrect placement of the lead V1 electrode (too high on the chest), lack of LV capture, LV lead displacement, pronounced latency (true exit block), conduction delay around the LV stimulation site, ventricular fusion with the intrinsic QRS complex, coronary venous LV pacing via the middle or anterior cardiac vein, unintended placement of two leads in the RV and severe conduction abnormalities within the LV myocardium. Most of these situations can cause a QS complex in lead V1 which should be interpreted (excluding fusion) as reflecting RV preponderance in the depolarization process. Barring the above causes, a negative complex in lead V1 is unusual and it probably reflects a different activation of a heterogeneous biventricular substrate (ischemia, scar, His-Purkinje participation). The latter is basically a diagnosis of exclusion. With a non-dominant R wave in lead V1, programming the V-V interval with LV preceding RV may bring out a diagnostic dominant R wave in lead V1 representing the contribution of LV stimulation to the overall depolarization process. In this situation the emergence of a dominant R wave confirms the diagnosis of prolonged LV latency (exit delay) or an LV intramyocardial conduction abnormality near the LV pacing site but it rules out the various causes of LV lead malfunction or misplacement. (Cardiol J 2011; 18, 5: 476–486)

Key words: left ventricular pacing, cardiac resynchronization, biventricular pacing, ventricular fusion, electrocardiography, heart failure, anodal capture, first-degree atrioventricular block

Address for correspondence: S. Serge Barold, MD, Florida Heart Rhythm Institute, Tampa, Florida, USA, tel: 813 891 1922, e-mail: ssbarold@aol.com

Introduction

The "low-tech" paced 12-lead surface ECG has fallen into disuse for routine pacemaker evaluation but it has become an indispensible part of device evaluation and troubleshooting in patients with implanted devices for cardiac resynchronization. A 12-lead ECG should always be available during device programming. A single-channel rhythm strip from a pacemaker programmer is inappropriate and may be misleading. During evaluation of cardiac resynchronization devices the 12-lead ECG yields important information about the balance between right ventricular (RV) and left ventricular (LV) activation (especially by the presence or absence of a dominant R wave of paced beats in lead V1), the presence or absence of fusion with the conducted intrinsic QRS complex, and the presence of RV anodal capture in patients with a certain type of LV leads [1]. Complete assessment requires comparison of the QRS morphology during native conduction, single chamber RV, single chamber LV, biventricular (BIV) pacing and RV anodal stimulation in special cases. Separate programming RV and LV leads may provide additional diagnostic data and is sometimes the surest way to confirm capture by each channel in a BIV system. Thus, the various patterns of RV only pacing and LV only pacing must be known in greater detail than in the past [1].

Conventional right ventricular pacing

Negative QRS complex in lead I

During RV apical pacing the frontal plane axis points superiorly mostly to the left but occasionally to the right (superior quadrant). In the latter case lead I shows a negative QRS deflection. This negativity in lead I (which is normal) has been erroneously interpreted as representing left-sided pacing [2].

Right ventricular pacing from the septum or outflow tract

In our evaluation of over 100 cases, we found that RV outflow or septal pacing invariably generates a paced left bundle branch block (LBBB) pattern in the precordial leads so that there is no dominant R wave in lead V1 in contrast to RV apical pacing where about 10–20% of cases exhibit a dominant R wave in lead V1 [3, 4]. Consequently we believe that the following two statements in a book on resynchronization are erroneous: "Right ventricular leads placed in the right ventricular outflow tract, particularly in more leftward locations, produce a right bundle branch pattern because the right ventricular outflow tract is located on the left side of the body…" Also: "…the relatively leftward location of a pacing site in the right ventricular outflow tract produces a positive deflection or right bundle branch block." [2].

Right ventricular septal or outflow tract pacing may generate qR complexes in leads I and aVL but the precordial leads retain an LBBB configuration without q complexes in the absence of old large anteroseptal myocardial infarction. The frontal axis is usually in the right inferior quadrant. At present there are no reliable ECG patterns capable of localizing the site of pacing along the septum or outflow tract [5–7].

Significance of a small r wave in lead V1

A small early (r) wave (sometimes wide) may occasionally occur in lead V1 during uncomplicated RV apical or outflow tract pacing. There is no evidence that this r wave represents a conduction abnormality at the RV pacing site. Furthermore, an initial r wave during BIV pacing does not predict initial LV activation [2].

Pacing from the right ventricular apex and a dominant paced R wave in lead V1

A dominant R wave in V1 (R/S > 1) during RV pacing is often called a "right bundle branch block" pattern, but this terminology is potentially misleading because this configuration reflects activation moving from the posterior to the anterior part of the heart and it does not seem related to RV activation delay. A dominant R wave in lead V1 can be seen in about 10–20% of cases of uncomplicated RV apical pacing [3, 4, 8, 9]. It may also be due to placing lead V1 too high in the third or second intercostal space. A tall R wave (Rs or R complex) extending to V3 and V4 signifies that a pacemaker lead is in all likelihood not in the RV. Other causes include pacing in the myocardial relative refractory period, ventricular fusion, epicardial or endocardial LV pacing, LV pacing from the coronary venous system, lead perforation of the RV with migration of the lead in the pericardial space towards the LV. Thus, an ECG of uncomplicated RV pacing with a dominant R wave in lead V1 and a right superior frontal plane axis can closely resemble that from a BIV pacemaker. In uncomplicated RV apical pacing, a dominant R wave in leads V1 and V2 in the 4th intercostal space disappears when the leads are recorded in the right and left 5th intercostal space [3, 4].
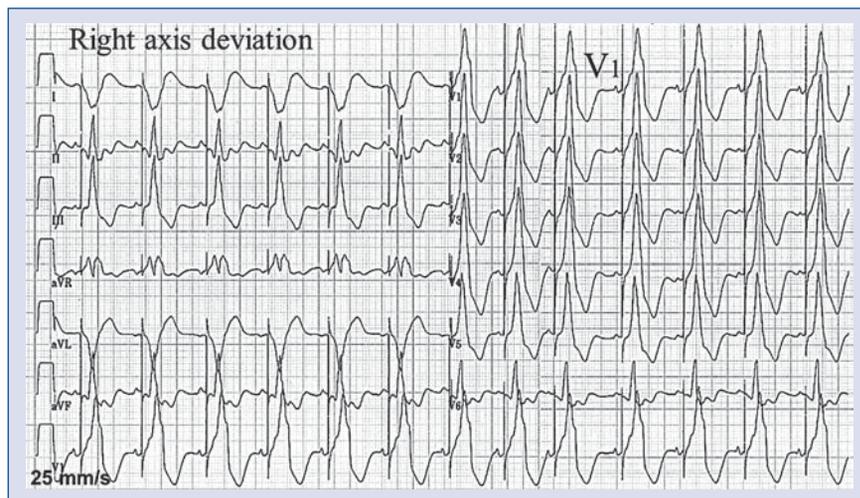
Figure 1. 12-lead ECG showing monochamber left ventricular (LV) pacing from the coronary venous system. There is a typical right bundle branch pattern and right axis deviation. Note the dominant R wave from V1 to V6 consistent with basal (LV) pacing. The AV delay was programmed to a short duration to prevent fusion with the spontaneous QRS complex. LV pacing shown in all the subsequent figures was performed from the coronary venous system. (Reproduced with permission from: [1]).

ECG patterns recorded during left ventricular pacing from the coronary venous system

Left ventricular pacing

An examination of ECG during pacing from the posterior or posterolateral vein in 100 patients revealed that virtually all registered a tall R wave in a correctly positioned lead V1 (Fig. 1) [10]. LV pacing from the coronary venous system that generates a positive complex in lead V1 may not necessarily be accompanied by a positive QRS complex in leads V2 and V3. With apical sites, leads V4–V6 are typically negative. With basal locations leads V4–V6 are usually positive as with the concordant positive R waves during overt pre-excitation in left-sided accessory pathway conduction in the Wolff-Parkinson-White syndrome. During pacing from the posterior or posterolateral coronary vein, the frontal plane axis during LV pacing often points to the right inferior quadrant (right axis deviation) and less commonly to the right superior quadrant. In an occasional patient with uncomplicated LV pacing with a typical dominant R wave in lead V1, the axis may point to the left inferior or left superior quadrant. The reasons for these unusual axis locations are unclear. Pacing from the mid-portion of the middle cardiac vein or the great (anterior) vein produces an LBBB configuration [10]. In a recent series of 40 patients with LV leads in the posterior or posterolateral coronary vein, we found 9 patients that did

not exhibit a dominant R wave in lead V1 during LV pacing: 6 patients had a balanced pattern (R = S) and 3 showed an LBBB pattern but with an initial non-dominant r wave [9]. The 3 patients with a negative QRS complex during LV pacing in V1 all had a non-dominant R wave in lead V1 and QS complexes in leads I and aVL indicating early activation of the LV lateral wall. Hence, no patient had a QS complex in V1 during LV pacing. A QS pattern in lead V1 during LV pacing suggests that the LV lead is not in the posterior or posterolateral coronary vein. An LBBB pattern during LV pacing may also occur from pacing sites in the posterior or posterolateral coronary vein in the setting of severe myocardial disease causing conduction block around the LV electrode and/or at some distance from the LV lead. This abnormality has not has not yet been characterized.

Special circumstances

A dominant R wave may not be seen if leads V1 and V2 are recorded too high (Fig. 2), or with unsuspected RV and LV lead reversal when programming only the LV output. Grimley et al. [11] reported the ECGs of a patient with ischemic cardiomyopathy and cardiac resynchronization therapy (CRT) where LV pacing from the posterolateral wall produced 2 different morphologies at rates of 75 and 85 ppm respectively unaffected by changing the output (Fig. 3). Both LV paced patterns exhibited lack of a dominant R wave in lead V1 and were
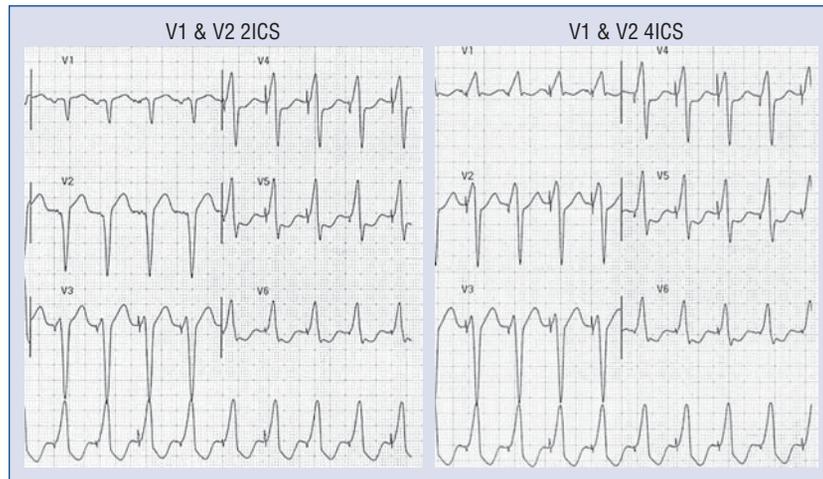
Figure 2. Effect of incorrect position of ECG leads on the QRS pattern in lead V1. Left panel: ECG recorded during left ventricular (LV) pacing with leads V1 and V2 recorded at the level of the second intercostal space in a patient with a thin patient an elongated chest. There is no dominant R wave in lead V1. The ECG during biventricular pacing also failed to show a dominant R wave in V1 at the level of the third intercostal space. Right panel: During LV pacing the dominant R wave in V1 becomes evident only when lead V1 is recorded in the 4th intercostal space. The R wave in V1 recorded in the 4th intercostal space during biventricular pacing also became dominant; ICS — intercostal space. (Reproduced with permission from: [1]).

associated with different mechanical activation. Biventricular pacing (RV lead at the apex) showed an LBBB pattern identical to monochamber RV pacing (Fig. 3). There was no evidence of abnormal LV latency. The observations were attributed to delay of the emerging LV impulse in a region of scarring near the LV stimulation site in the absence of prolonged latency.

Biventricular pacing with the right ventricular lead located at the apex

The frontal plane QRS axis usually moves superiorly from the left (RV apical pacing) to the right superior quadrant (BIV pacing) in an anticlockwise fashion if the ventricular mass is predominantly depolarized by the LV pacing lead (Fig. 4). The frontal plane axis may occasionally reside in the left superior rather than the right superior quadrant during BIV pacing from the posterior or posterolateral coronary vein and the RV apex. A mean frontal plane axis in the other 2 quadrants is distinctly unusual but does not necessarily indicate a problem. The QRS complex is often positive or dominant in lead V1 during BIV pacing by this arrangement (Fig. 5) [1]. The combination of a right superior axis and a dominant R wave in lead V1 is not diagnostic of BIV pacing because monochamber RV apical pacing may sometimes produce the same combination. Hence,

the importance of sequentially programming monochamber RV and LV pacing for the proper evaluation of BIV pacing.

Negative QRS complex in lead V1 during CRT + RV apical pacing

A negative paced QRS complex (LBBB pattern) in lead V1 may occur in the following circumstances: incorrect placement of lead V1 (too high on the chest), lack of LV capture, LV lead displacement, marked LV latency (exit block) [12] marked delay in the local propagation of LV activation from the stimulation site (with or without abnormal latency) [11], ventricular fusion with the conducted QRS complex, coronary venous pacing via the middle cardiac vein (also the anterior cardiac vein) [10], or even unintended placement of 2 leads in the RV (Figs. 6, 7). A QS pattern in lead V1 suggests that the LV lead either lies in the middle or anterior cardiac vein or there is exessive RV representation overshadowing LV activity. Barring other causes (Fig. 8) a negative QRS complex in lead V1 during simultaneous BIV pacing (with LV pacing from the posterior or posterolateral coronary vein) probably reflects different activation of an heterogeneous BIV substrate (ischemia, scar, His-Purkinje participation in view of the varying patterns of LV activation in spontaneous LBBB etc.) and does not necessarily indicate a poor (electrical or mechanical)
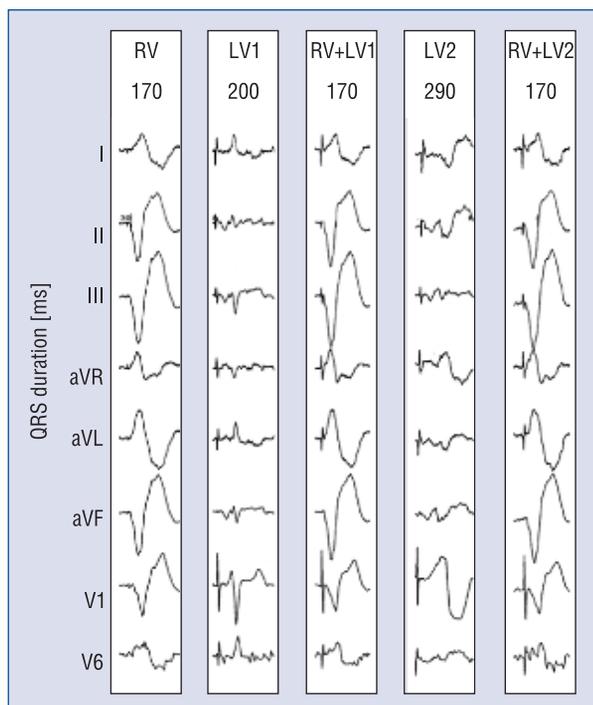
Figure 3. Unusual QRS morphology in lead V1 during left ventricular (LV) pacing related to emergence of the LV pacing impulse from a region of scaring and slow conduction. ECGs were recorded during pacing from the right ventricle (RV) alone, LV alone and simultaneous biventricular pacing; LV1 = morphology at a pacing rate of 75 ppm; LV2 = morphology at a pacing rate of 85 ppm. Simultaneous biventricular pacing was performed with LV1 morphology (RV + LV1 at a rate of 75 ppm) and LV2 morphology (RV + LV2 at 85 ppm). The R/S ratio is < I in lead V1 during monochamber LV and biventricular pacing. See text for details. (Reproduced with permission from: [11]).
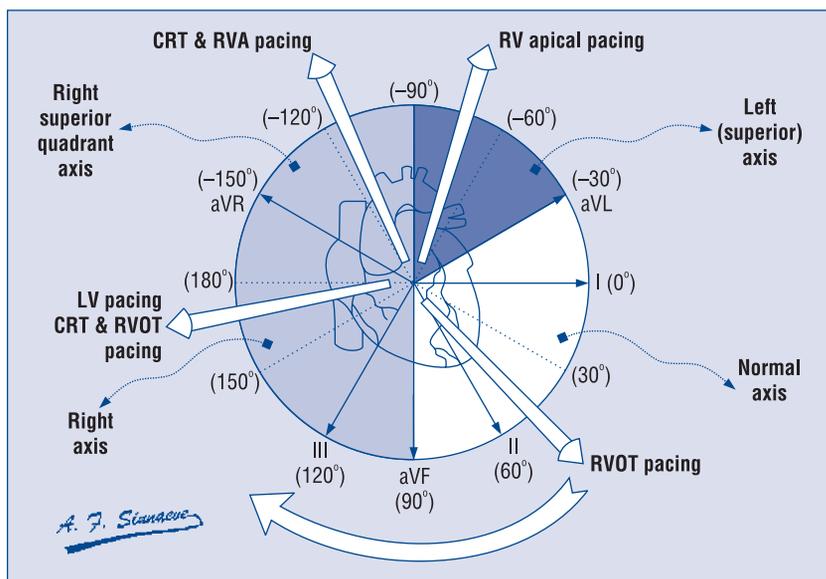

Figure 4. Diagram showing the usual direction of the mean frontal plane axis during apical right ventricular (RV) pacing, RV septal/outflow tract pacing, monochamber left ventricular (LV) pacing from a posterior or posterolateral coronary vein, biventricular (BIV) pacing with LV from a posterior or posterolateral coronary vein + RV from the apex or BIV pacing with LV from a posterior or posterolateral coronary vein + RV from the septal/outflow tract. (1) Monochamber RV pacing. During septal or RV outflow tract (RVOT) pacing the axis may be in the "normal" site in the left inferior quadrant and it moves to the right inferior quadrant (right axis deviation) as the site of stimulation moves more superiorly towards the pulmonary valve. (2) Monochamber LV pacing from the posterior or posterolateral coronary vein. The axis often points to the right inferior quadrant (right axis deviation) and less commonly in the right superior quadrant. (3) Biventricular pacing (LV lead in the posterior or posterolateral coronary vein) with RV apical stimulation. The axis moves superiorly from the left (starting with monochamber RV apical pacing in the left superior quadrant) to the right superior quadrant in an anticlockwise fashion during BIV pacing. This is the commonest axis direction but the axis may less commonly reside in the left superior quadrant and rarely in the other quadrants. (4) Biventricular pacing (from the posterior or posterolateral coronary vein) with RV septal/outflow tract stimulation. The axis is often directed to the right inferior quadrant (right axis deviation). The curved arrow indicates that the axis during septal/RVOT pacing can also reside in the right inferior quadrant; CRT — cardiac resynchronization therapy; RVA — RV apex. (Adapted with permission from: Barold SS, Stroobandt RX, Sinnaeve AF. Cardiac pacemakers and resynchronization step by step. An illustrated guide. Wiley-Blackwell, Hobocken NJ 2010: 324).
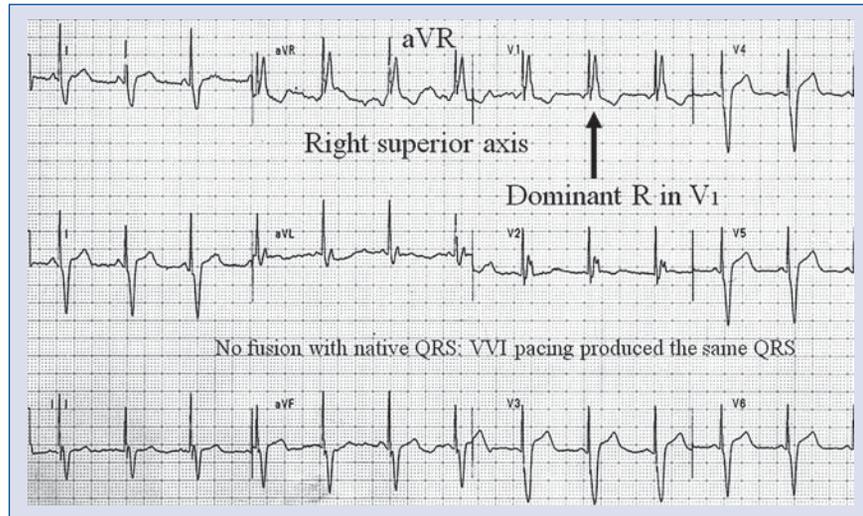
Figure 5. ECG during biventricular pacing with the right ventricular lead at the apex. There is a dominant R wave is V1 and a right superior axis in the frontal plane. The QRS complex was relatively more narrow (170 ms) than during single chamber right ventricular or left ventricular pacing. (Reproduced with permission from: Barold SS, Giudici MC, Herweg B. Importance of the surface electrocardiogram for the assessment of cardiac resynchronization. In: Yu CM, Hayes D, Auricchio A eds. Cardiac resynchronization therapy. 2nd Ed. Wiley-Blackwell. Malden, MA 2008: 309–323).
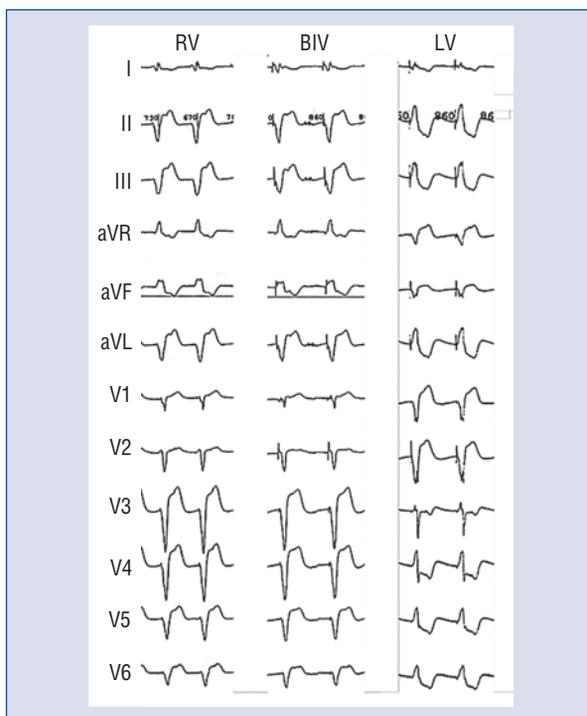

Figure 6. 12-lead ECGs during right ventricular (RV) apical pacing, left ventricular (LV), and biventricular (BIV) showing a left bundle branch block (LBBB) configuration in all 3 tracings. There is a tiny initial r wave in lead V1 during BIV pacing. The LBBB pattern during monochamber LV pacing should be considered as coming from lead misplacement until proven otherwise because this pattern is very unusual when pacing from the posterior or posterolateral veins. The LV lead was in the anterior coronary vein (see next figure for lead location).
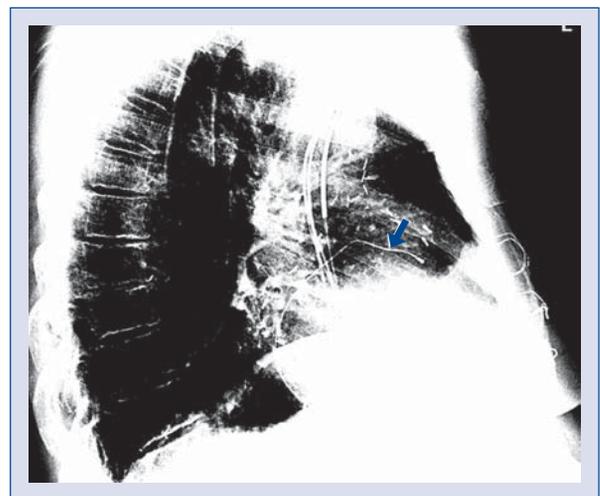

Figure 7. Same patient as the previous figure. Lateral chest X-ray showing anterior location of the left ventricular lead in the anterior coronary vein (arrow).

contribution from LV stimulation. LV intramyocardial conduction delay causing a negative complex in lead V1 is unusual and is basically a diagnosis of exclusion (Fig. 8).

Q, or q configuration in lead I and lateral leads during biventricular pacing using the right ventricular apex

Georger et al. [13] observed a q wave in lead I in 17 of 18 patients during biventricular pacing us-
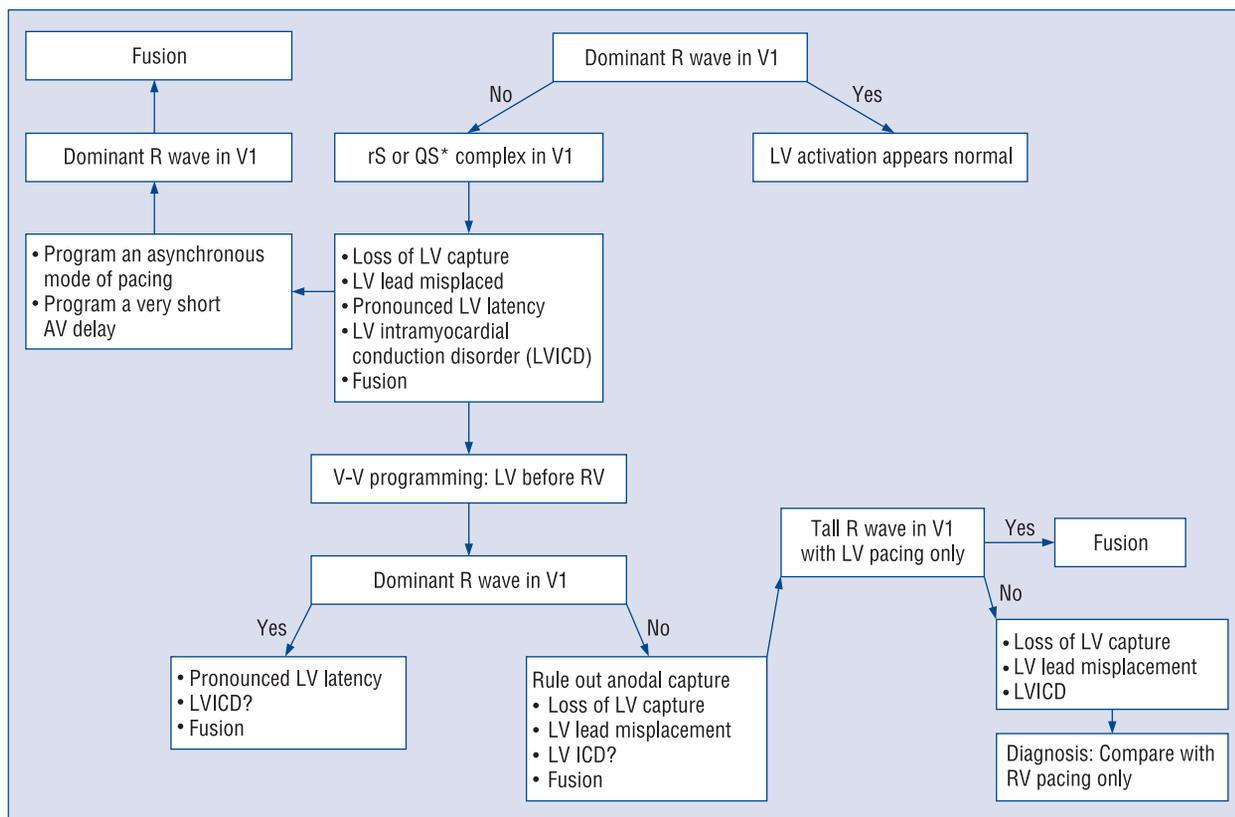
Figure 8. Algorithm to evaluate the configuration of the paced ECG in lead V1 during simultaneous biventricular pacing. Ventricular fusion with the intrinsic rhythm is the great ECG imitator and appears at several levels. A misplaced left ventricular (LV) lead means location in the anterior or the middle cardiac vein; LVICD — LV intramyocardial conduction delay. Little is known about this entity and precisely where it fits in the algorithm. However, it should always be a diagnosis of exclusion. A QS complex (barring fusion with the intrinsic rhythm) is not diagnostic of any problem but it should be cause for concern (*) as it represents an unfavorable situation with right ventricular (RV) preponderance when LV activation is delayed (or absent) and being overshadowed by RV activation. The diagnosis of fusion with the intrinsic rhythm can be made as a first step in the evaluation of a negative lead V1 (top left) simply by programming to the asynchronous mode or with a short AV delay. Then, in practice there is no real need to consider fusion in the other parts of the protocol. We elected to show in this figure the importance of fusion at several levels to emphasize its ubiquity in the ECG evaluation of cardiac resynchronization therapy. For more precise LV lead location the algorithm of Ploux et al. [17] can be consulted.

ing the RV apex (Fig. 9). A Q/q wave (followed by positivity) is rare in lead I during uncomplicated monochamber RV apical pacing and these workers observed this configuration in only 1 patient. Georger et al. [13] found that loss of the q wave in lead I during BIV pacing (with apical RV pacing) was 100% predictive of loss of LV capture. Q/q waves (followed by a positive deflection) are not uncommon in the lateral leads (I, aVL, V5 and V6) during uncomplicated BIV pacing using the RV apex. These patterns do not indicate the presence of an extensive anteroseptal myocardial infarction as they do in monochamber RV pacing [14]. A Q/q wave may also occur during RV pacing in leads I and aVL but not the precordial leads during uncomplicated septal or outflow tract RV pacing in the absence of myocardial infarction.

Biventricular pacing with the right ventricular lead in the outflow tract

In a series of over 100 cases, we found that during BIV pacing with the RV lead in the right septal area or outflow tract and the LV lead in the posterior or posterolateral coronary vein, the paced QRS in lead V1 is often negative and the frontal plane paced QRS axis is often directed to the right inferior quadrant (right axis deviation) (Fig. 10).
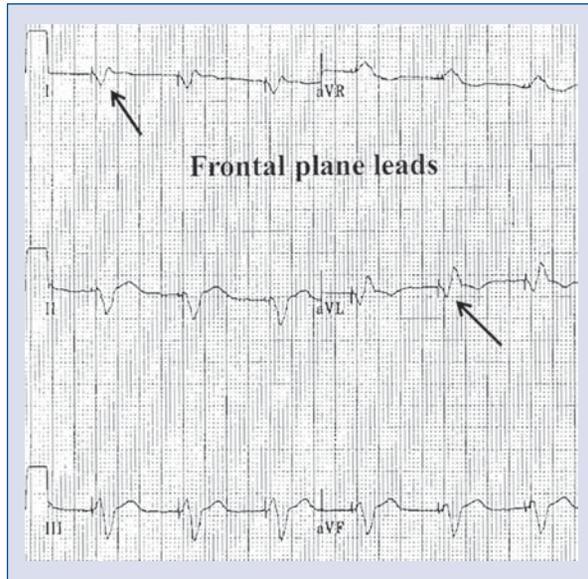
Figure 9. Uncomplicated biventricular pacing (right ventricular lead at the apex) in a patient with nonischemic cardiomyopathy. The interventricular (V-V) interval was programmed at 40 ms with left ventricular activation first. The 6-lead ECG shows a Qr complex in lead I and a QR complex in lead aVL. These changes may occur during uncomplicated biventricular pacing and do not indicate an old myocardial infarction. The frontal plane axis lies in the right superior quadrant as expected with this pacing arrangement. (Reproduced with permission from: 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: 471–490).

This may create a problem in troubleshooting because the ECG may resemble that of simple monochamber RV septal or outflow tract pacing with an LBBB pattern and right inferior frontal axis deviation mimicking loss of LV pacing.

Frontal plane axis of the paced QRS complex

Figure 4 shows the importance of the frontal plane axis of the paced QRS complex in determining the arrangement of pacing with the LV only, the RV only, and LV + RV. The shift in the frontal plane QRS axis during programming the ventricular output is helpful in determining the effect of programming manipulations.

Systematic evaluation of the ECG after CRT

Sweeney et al. [15] analyzed the ECGs of 202 consecutive CRT patients who received devices according to standard indications excluding those with right bundle branch block, and a previous pacemaker. A positive response to CRT was defined as a ≥ 10% reduction of the LV end-systolic volume (LVESV) at 6 months. Only 45.5% of patients met the end point of ≥ 10% reduction of the LVESV. Right axis deviation for this study was defined as −90° ⇔ +90°. A rightward axis as defined emerged in 68% of patients (right superior axis in 59%, and
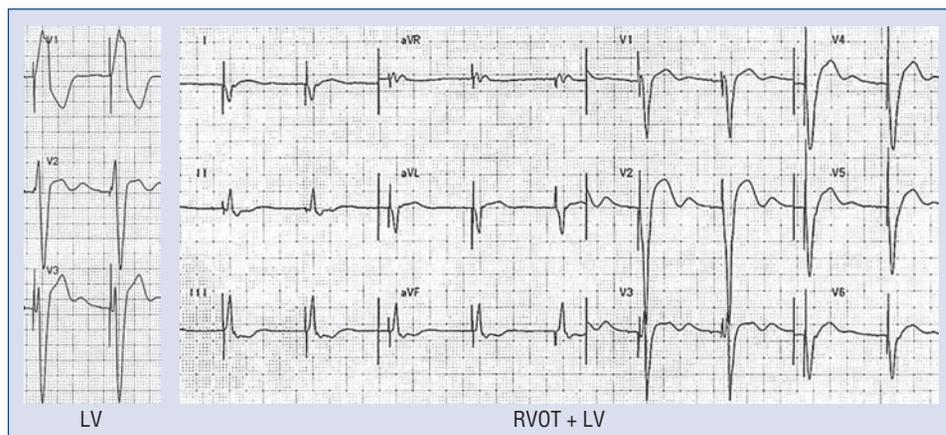

Figure 10. Biventricular (BIV) pacing with the right ventricular (RV) lead in the outflow tract. There was a very prominent R wave in lead V1 during monochamber left ventricular (LV) pacing. Note the typical absence of a dominant R wave in lead V1, and the presence of right axis deviation, an uncommon finding during BIV pacing with the RV lead at the apex. The presence of ventricular fusion with the spontaneous conducted QRS complex was ruled out when the QRS configuration did not change by programming to the BIV VVI mode at a faster rate. Note that the pattern during uncomplicated BIV pacing resembles that of monochamber ventricular pacing from the RV outflow tract (RVOT). This similarity can make troubleshooting difficult. (Reproduced with permission from: 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: 471–490).

right inferior axis in 9%) providing evidence of left to right reversal of activation. An increase in the R wave (dominant) in lead V1 was documented in 50% of patients and in lead V2 in 24% of patients reflecting a reversal of activation (posterior to anterior). A dominant R wave in these leads was associated with an increased probability of reverse remodeling. This effect was not observed until the mean change in the R wave was ≥ 4.5 times the baseline value. A shift from left axis deviation to right axis deviation as defined by Sweeney et al. [15], was also associated with an increased probability of reverse modeling. Indicators of paced ventricular fusion (from LV and RV stimulation) provided a positive predictor of reverse remodeling. It is disconcerting that only two thirds of the patients exhibited ECG evidence of ventricular fusion wavefronts from the RV and LV pacing sites.

The study of Sweeney et al. [15] is limited because the site of RV pacing was not stated (but presumed to be apical) and there were no ECGs of monochamber LV pacing to rule out unusual pacing sites and other abnormalities such as pronounced LV latency or myocardial disease with delayed conduction around the LV pacing site. The study failed to use a programmable V-V interval (to bring out a dominant R wave in lead V1) and rule out the presence of ventricular fusion with the spontaneous QRS complex a situation with triple ventricular activation that can mask or diminish the paced contribution from the two pacing sites.

Refaat et al. [8] analyzed the paced ECGs of 54 consecutive patients who received a BIV device (RV apex) over a period of 2 years. (1) Fifty patients (92.6%) demonstrated a dominant R wave (R/s > 1) in lead V1. A right superior axis was found in 64% of the patients and left axis deviation in 36%. In this group, the position of the LV lead was verified to be in the posterior or posterolateral coronary vein by coronary sinus angiography at the time of implantation and fluoroscopy. (2) There were 4 patients (7.4%) with an LBBB pattern in lead V1. The LV lead was found in all to be in the middle cardiac vein. Importantly, these 4 patients exhibited a QS pattern in lead V1 during BIV pacing. No patients received a device with an interventricular offset. The very high incidence of a dominant R wave (100% in the 50 patients without pacing from the middle cardiac vein) is difficult to explain because some of the patients must have had ventricular fusion with the intrinsic rhythm producing substantial changes in lead V1 as a results AV optimization. Furthermore, with a non-responsive CRT rate of at least 30%, the observations suggest that a dominant R wave in lead V1 should not be automatically equated with a favorable mechanical response.

We evaluated 40 consecutive CRT patients who had shown long-term improvement. Patients with LV leads at sites other than the posterolateral or lateral coronary veins were excluded [9]. Patients with RV anodal stimulation were also excluded. During simultaneous RV and LV pacing 5 patients showed prolonged latency (≥ 40 ms defined as the time from the pacemaker stimulus to the onset of the paced QRS complex recorded in a 12-lead ECG), and 35 patients presented with no latency (< 40 ms). Four of the 5 patients with prolonged latency showed an LBBB pattern in lead V1 and one displayed a dominant R wave in lead V1 during simultaneous BIV pacing (Fig. 8). All 4 LBBB patients with marked LV latency (> 40 ms) exhibited a QS pattern in lead V1 during simultaneous BIV pacing. Barring the 5 cases of marked latency, 30 of 35 patients showed a dominant R wave in lead V1, 2 had a balanced QRS complex in lead V1 and the other 3 showed a negative complex in lead V1 (one QS and 2 rS configurations). A total of 7 patients (18%) presented a LBBB (4 latency and 3 others) during simultaneous LV and RV pacing. Therefore 31 of 40 patients (78%) presented with a dominant R wave in lead V1 during simultaneous BIV pacing.

After AV and V-V optimization, 35 patients in the report of Herweg et al. [9] showed a dominant R wave in lead V1, 2 a balanced QRS complex and 3 showed a LBBB pattern. Two of the 3 patients with an LBBB pattern were found to have ventricular fusion with the spontaneous QRS complex. The proof of fusion was obtained by the appearance of a dominant R wave in lead V1 during asynchronous BIV pacing or during regular BIV pacing with a very short AV delay at an appropriate rate from the RV and LV (Fig. 8). We cannot explain the mechanism of the LBBB in the patient without fusion but we postulated there was slow conduction from the LV lead in a scar zone (not latency), a situation where V-V programming may be especially useful in establishing favorable hemodynamics even if the paced ECG continues to show an LBBB pattern of activation. This entity is a diagnosis of exclusion because all the other causes of an LBBB pattern during BIV pacing must be ruled out (Fig. 8). Interestingly, our 3 patients with an LBBB after AV and V-V optimization have all been CRT responders. All in all, in our series a dominant R wave in lead V1 was eventually found in 93% of the patients including those with fusion (with the intrinsic rhythm) or abnormal latency where the dominant R wave in lead V1 was brought out by appropriate device programming [9].

Do we need a new algorithm?

The usefulness of a paced ECG algorithm in the early development of CRT was limited because it did not address malposition of the LV lead and was too simplistic [16]. Ploux et al. [17] evaluated 100 CRT patients to determine the LV lead position during BIV pacing. They then constructed an algorithm to determine the site of LV pacing (posterior, lateral, inferior and anterior). A dominant R wave in lead V1 provided 3 possibilities: posterior, lateral and inferior. The differentiation of the first two possibilities is really unimportant clinically but knowing the presence of an inferior position may be somewhat useful. Ploux et al. [17] claimed that an inferior position is never associated with a positive complex in lead V6. A substantial number of patients showed an anterior LV lead along the interventricular septum. Most of these patients exhibited a QS complex in lead V1 and less commonly an rS complex. According to Ploux et al. [17] a QS complex is specific for an anterior lead position. This interesting algorithm omitted the causes of a negative QRS complex. Nevertheless it has a high sensitivity, specificity and predictive value.

Paced QRS duration

In a review of 23 studies in 1995 we found only 3 studies where there was a correlation between the degree of QRS shortening after CRT and hemodynamic benefit and/or the clinical response [18––21]. Since then, several studies have re-explored the issue with better data [22–25]. In the positive studies, the degree of shortening is so small as to be virtually undetectable on a standard ECG. Furthermore in this type of study, the large overlap of responders and non-responders in terms of QRS duration makes this measurement not clinically useful. The sensitivity of the findings regarding the duration of the paced QRS complex is somewhat satisfactory but the specificity is poor. In some cases the QRS complex after CRT may actually lengthen or remain unchanged despites substantial improvement in mechanical LV dyssynchrony. Increased QRS duration with CRT does not necessarily reflect the presence of ventricular areas with slow conduction resulting in more heterogeneous myocardial activation. Indeed with monochamber LV pacing there is an obvious discrepancy between the long QRS duration (compared with baseline) and hemodynamic and clinical improvement. Some patients with monochamber LV pacing exhibit an equal or superior degree of mechanical resynchronization compared to BIV pacing despite a very wide paced QRS complex [26]. Thus, a wide paced QRS duration cannot be assumed to reflect a more heterogeneous propagation pattern of LV activation and prolonged duration of mechanical activation.

Electrical remodeling

A number of studies but not all suggest that the duration of the intrinsic QRS complexes shortens slightly with the passage of time [27–30]. This may reflect changes in the conduction system or intramyocardial impulse transmission. Electrical remodeling may be associated with a positive response to CRT, and antiarrhythmic effect. The small magnitude of intrinsic QRS shortening renders its precise measurement uncertain.

Disclosures

The authors do not report any conflict of interest regarding this work.

References

1. Barold SS, Herweg B, Giudici M. Electrocardiographic follow-up of biventricular pacemakers. Ann Noninvasive Electrocardiol, 2005; 10: 231–255.
2. Asirvatham SJ. Electrocardiogram interpretation with biventricular pacing devices. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ eds. Resynchronization and defibrillation for heart failure. A practical approach. Blackwell-Futura, Oxford UK 2004: 73–97.
3. Barold SS, Falkoff MD, Ong LS, Heinle RA. Electrocardiographic analysis of normal and abnormal pacemaker function. Cardiovasc Clin, 1983; 14: 97–134.
4. Klein HO, Beker B, Sareli P, DiSegni E, Dean H, Kaplinsky E. Unusual QRS morphology associated with transvenous pacemakers. The pseudo RBBB pattern. Chest, 1985; 87: 517–521.
5. McGavigan AD, Roberts-Thomson KC, Hillock RJ, Stevenson IH, Mond HG. Right ventricular outflow tract pacing: Radiographic and electrocardiographic correlates of lead position. Pacing Clin Electrophysiol, 2006; 29: 1063–1068.
6. Burri H, Park CI, Zimmermann M et al. Utility of the surface electrocardiogram for confirming right ventricular septal pacing: Validation using electroanatomical mapping. Europace, 2011; 13: 82–86.
7. Balt JC, van Hemel NM, Wellens HJ, de Voogt WG. Radiological and electrocardiographic characterization of right ventricular outflow tract pacing. Europace, 2010; 12: 1739–1744.
8. 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: 289–295.
9. Herweg B, Ali R, Ilercil A 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: 1382–1391.

10. Giudici MC, Tigrett DW, Carlson JI, Lorenz TD, Paul DL, Barold SS. Patterns in cardiac resynchronization therapy. Pacing the great cardiac and middle cardiac veins. Pacing Clin Electrophysiol, 2007; 30: 1376–1380.

11. Grimley SR, Suffoletto MS, Gorcsan J 3rd, Schwartzman D. Electrocardiographically concealed variation in left ventricular capture: A case with implications for resynchronization therapy in ischemic cardiomyopathy. Heart Rhythm, 2006; 3: 739–742.

12. Herweg B, Ilercil A, Madramootoo C et al. Latency during left ventricular pacing from the lateral cardiac veins: A cause of ineffectual biventricular pacing. Pacing Clin Electrophysiol, 2006; 29: 574–581.

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

14. Barold SS, Herweg B, Curtis AB. Electrocardiographic diagnosis of myocardial infarction and ischemia during cardiac pacing. Cardiol Clin, 2006; 24: 387–399.

15. 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: 626–634.

16. Ammann P, Sticherling C, Kalusche D et al. An electrocardiogram-based algorithm to detect loss of left ventricular capture during cardiac resynchronization therapy. Ann Intern Med, 2005; 142: 968–973.

17. Ploux S, Bordachar P, Deplagne A et al. Electrocardiogram-based algorithm to predict the left ventricular lead position in recipients of cardiac resynchronization systems. Pacing Clin Electrophysiol, 2009; 32 (suppl. 1): S2–S7.

18. Kashani A, Barold SS. Significance of QRS complex duration in patients with heart failure. J Am Coll Cardiol, 2005; 46: 2183–2192.

19. Lecoq G, Leclercq C, Leray E et al. Clinical and electrocardiographic predictors of a positive response to cardiac resynchronization therapy in advanced heart failure. Eur Heart J, 2005; 26: 1094–1100.

20. Yeim S, Bordachar P, Reuter S et al. Predictors of a positive response to biventricular pacing in patients with severe heart failure and ventricular conduction delay. Pacing Clin Electrophysiol, 2007; 30: 970–975.

21. Molhoek SG, Van Erven L, Bootsma M et al. QRS duration and shortening to predict clinical response to cardiac resynchronization therapy in patients with end-stage heart failure. Pacing Clin Electrophysiol, 2004; 27: 308–313.

22. Kamireddy S, Agarwal SK, Adelstein E, Jain S, Saba S. Correlation of electrical and mechanical reverse remodeling after cardiac resynchronization. Ann Noninvasive Electrocardiol, 2009; 14: 153–157.

23. Boriani G, Biffi M, Martignani C et al. Electrocardiographic remodeling during cardiac resynchronization therapy. Int J Cardiol, 2006; 108: 165–170.

24. Rickard J, Popovic Z, Verhaert D et al. The QRS narrowing index predicts reverse left ventricular remodeling following cardiac resynchronization therapy. Pacing Clin Electrophysiol, 2011 [Epub ahead of print].

25. Iler MA, Hu T, Ayyagari S et al. Prognostic value of electrocardiographic measurements before and after cardiac resynchronization device implantation in patients with heart failure due to ischemic or nonischemic cardiomyopathy. Am J Cardiol, 2008; 101: 359–363.

26. Leclercq C, Faris O, Tunin R et al. Systolic improvement and mechanical resynchronization does not require electrical synchrony in the dilated failing heart with left bundle branch block. Circulation, 2002; 106: 1760–1763.

27. Tereshchenko LG, Henrikson CA, Stempniewicz P, Han L, Berger RD. Antiarrhythmic effect of reverse electrical remodeling associated with cardiac resynchronization therapy. Pacing Clin Electrophysiol, 2011; 34: 357–364.

28. Stockburger M, Nitardy A, Fateh-Moghadam S et al. Electrical remodeling and cardiac dimensions in patients treated by cardiac resynchronization and heart failure controls. Pacing Clin Electrophysiol, 2008; 31: 70–77.

29. Henrikson CA, Spragg DD, Cheng A et al. Evidence for electrical remodeling of the native conduction system with cardiac resynchronization therapy. Pacing Clin Electrophysiol, 2007; 30: 591–595.

30. Vogt J, Krahnefeld O, Lamp B et al.; Pacing Therapies in Congestive Heart Failure Study Group. Electrocardiographic remodeling in patients paced for heart failure. Am J Cardiol, 2000; 86: 152K.