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The Evolving Role of Electrocardiography in Cardiac Resynchronization Therapy

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Abbreviations *CRT* Cardiac resynchronization therapy · *ECG* Electrocardiography · *LBBB* Left bundle branch block · *LV* Left ventricle · *RBBB* Right bundle branch block · *RV* Right ventricle

Abstract

Purpose of review This study aims to summarize the literature on the role of electrocardiography (ECG) in (i) patient selection for cardiac resynchronization therapy (CRT), (ii) predicting clinical response after CRT system is implanted, and (iii) optimizing CRT programming.

Recent findings Progress has been made in interpreting ECG beyond QRS duration and left bundle branch (LBBB) morphology to select patients for CRT. We now understand a higher chance of response to CRT in patients with atypical right bundle branch block and lower response rates in subgroups with atypical LBBB. QRS area has emerged as a novel marker to quantify baseline electrical dyssynchrony to improve patient selection. After CRT, the resultant QRS narrowing remains the most validated predictor of long-term favorable outcome. There is increasing awareness of prolonged left ventricular pacing latency hindering the desired response to CRT. There is active interest in using ECG beyond minimizing QRS duration to optimize CRT programming for maximal resynchronization. Novel strategies include fusion of paced and/or conducted wavefronts and minimization of paced QRS area.

Summary ECG remains the ubiquitous method for ventricular electrical mapping in context of CRT. The role of ECG in elucidating baseline electrical dyssynchrony to aptly select patients for this treatment continues to evolve, and ECG is increasingly being evaluated as a reliable endpoint for optimal CRT programming.

Introduction

Cardiac resynchronization therapy (CRT) can have an enormous beneficial impact in heart failure patients with delayed electrical activation of the left ventricular (LV) free wall, also called electrical dyssynchrony, and often recognizable as left bundle branch block (LBBB) on 12-lead surface electrocardiography (ECG) or due to chronic pacing from the right ventricle (RV). A two-site electrical pacing device is used to activate the septal aspect of LV from an electrode in the RV and synchronously activate the LV free wall with electrode(s) often implanted in a LV venous branch of coronary sinus (biventricular pacing) [1]. CRT results in improved hemodynamics, beneficial reverse cardiac remodeling (i.e., decrease in LV volume and increase in ejection fraction), and reduction in heart failure symptoms and hospitalizations. Most importantly, mortality is cut back by one third [2–6]. Specifically, CRT is indicated in patients with chronic symptomatic heart failure with persistently reduced LV ejection fraction ≤ 35% despite guideline-directed medical therapy, and, LBBB or a QRS duration ≥ 150 ms on ECG [7].

LV electrical dyssynchrony can cause acute mechanical dysfunction with unfavorable hemodynamic effect, but more importantly results in chronic adverse structural remodeling that can cause or exacerbate systolic heart failure [8]. It is important to emphasize that CRT is an electrical therapy that can correct the underlying electrical dyssynchrony, and thereby result in salutary reverse structural remodeling [3–6]. However, CRT cannot correct myocardial fibrosis-related mechanical dyssynchrony in the absence of electrical dyssynchrony, as noted in the EchoCRT trial among patients with QRS duration < 130 ms [9]. Although invasive electrical mapping and the novel non-invasive electrocardiographic imaging are insightful tools [10, 11], ECG remains the ubiquitous practical method to study the electrical function of the heart.

More than 200,000 CRT device implants are performed in heart failure patients every year [12]. However, up to 30% of CRT recipients fail to show a clinical response to CRT with large implications on quality and quantity of life and economic healthcare burden [13]. Failure of heart failure to respond to CRT can be attributed to factors like irreversible myocardial fibrosis, absence of sufficient baseline electrical dyssynchrony that is remediable by CRT, suboptimal site for LV pacing, non-adherence to heart failure medications, competing cardiac arrhythmias, e.g., atrial fibrillation or premature ventricular complexes that limit delivery of sufficient CRT, and inadequate non-individualized programming of CRT device [9, 14, 15].

Efforts are warranted to enhance patient selection criteria for CRT, so as to offer this effective treatment to all patients who stand to benefit from it, and not prescribe it to those without plausibility for a favorable response. Further, better understanding of individualized post-implant programming of the CRT device to optimally exploit the deliverable resynchrony and maximize CRT benefit is needed. Similarly, intra- or post-procedure early identification of patients with little possibility of improvement would expedite interventions like repositioning LV lead; CRT reprogramming; surgical epicardial LV lead placement; alternative CRT techniques like selective His-bundle [16], left bundle branch [17, 18], endocardial LV [19], or multisite pacing [20]; or advanced mechanical therapies like LV assist device or cardiac transplantation.

ECG is the basic method to evaluate baseline electrical dyssynchrony to select candidates who stand to benefit from CRT. Historically, QRS duration has been used as the main factor in patient selection. A narrow QRS (< 120 ms) essentially excludes delayed LV free wall activation and any role for CRT [9]. Additionally, various QRS morphological features are important determinants for patient selection beyond increased QRS duration by itself [21]. Aside from patient selection, acute CRT response as assessed by ECG is predictive of long-term clinical response. The next frontier in the evolving role of ECG in the context of CRT is its use for optimizing programming of CRT, specifically individualized tuning of the temporal RV-LV pacing offset. If proven, ECG may be an accessible, low-cost, reliable and practical method to maximize the potential benefit derivable from every CRT system. In this review, we therefore discuss the role for ECG in:

i. Patient selection for CRT,
ii. Predicting clinical response after CRT system is implanted, and
iii. Optimizing CRT programming.

Table 1 lists the key definitions relevant to discussion of ECG for CRT.

Patient selection for CRT

QRS duration and LBBB morphology are well-accepted surrogates for LV electrical dyssynchrony, have been used as inclusion criteria in the large randomized controlled trials on CRT, and adopted by practice guidelines [3–7].

QRS duration

QRS duration is a summary parameter for the time it takes for global ventricular excitation. A prolonged QRS duration is a prerequisite for electrical dyssynchrony that is amenable for correction with CRT. In the randomized

Table 1. Definitions

QRS duration	Duration from first onset of QRS in any lead to last offset of QRS in any lead on 12-lead ECG
LV electrical dyssynchrony	Substantial delay in electrical activation of LV free wall relative to the septum
LBBB morphology	QRS with negative V1 (QS or rS)
RBBB morphology	QRS with positive V1 (rsR′, rSR′, Rs, R, or qR)
Typical LBBB [22]	• QRS duration ≥ 140 ms (men) or ≥ 130 ms (women), • V1 and V2: negative (QS or rS), and • Mid-QRS notching/slurring: ≥ 2 out of V1, V2, V5, V6, I, aVL
Typical RBBB [23••]	• QRS duration ≥ 120 ms, • V1 or V2: rsR′ or rSR′ (terminal R′ wider than initial r), and • Leads I and V6: S wave duration > R wave duration or > 40 ms
Intraventricular conduction delay [24••]	• QRS duration ≥ 120 ms • Not meeting criteria for typical LBBB or typical RBBB • V1: categorizable as atypical LBBB or atypical RBBB morphology
Atypical RBBB [23••]	• QRS duration ≥ 120 ms, • V1: RBBB morphology, and • Leads I and aVL: absence of characteristic S wave
Q-LV [25]	Duration from onset of QRS to local electrogram at the LV electrode
Time to intrinsicoid deflection	Duration from onset of QRS to peak of R wave in any ECG lead
3D QRS area [26••]	Root-mean-square of QRS voltage-time integrals in vectorcardiographic x, y, z coordinates
Pacing latency	Duration from pacing stimulus to earliest major QRS deflection in any ECG lead

COMPANION trial on symptomatic systolic heart failure patients with baseline QRS duration ≥ 120 ms, benefit of CRT in reducing mortality and hospitalizations was incrementally greater in subgroups with QRS duration 120–147, 148–168, and > 168 ms, respectively [3]. As a corollary, patients with evidence of mechanical dyssynchrony but with baseline QRS duration < 130 ms in the EchoCRT trial not only failed to derive any benefit but instead had increased mortality with CRT [9].

Left bundle branch block

LBBB is considered to be pathognomic for delayed activation of LV free wall or electrical dyssynchrony. In the MADIT-CRT trial, the benefit of CRT was limited to patients with baseline LBBB morphology and not seen in patients with non-LBBB configurations, i.e., right bundle branch block (RBBB) or nonspecific intraventricular conduction delay [27]. As a consequence, there has been a shift towards avoiding non-LBBB morphologies for CRT patient selection to reduce CRT non-response rates [7].

Strauss et al. described that approximately one-third of patients with LBBB morphology on ECG may not have typical LBBB but rather a combination of left ventricular hypertrophy and left anterior fascicular block. They proposed a stricter criterion for LBBB: QRS duration ≥ 140 ms for men and ≥ 130 ms for women (instead of > 120 ms for all comers), negative QRS in leads V1 and V2 (QS or rS) and mid-QRS notching/slurring in ≥ 2 of leads V1, V2, V5, V6, I, and aVL [22]. Garcia-Seara et al. demonstrated that patients with typical LBBB had a substantially greater benefit with CRT as opposed to those with atypical LBBB [28••].

Beyond QRS duration and LBBB

The benefit of CRT becomes less clear in the absence of typical LBBB, especially when the QRS duration is < 150 ms. However, among selected patients without typical LBBB, CRT can still be useful [7]. There is increasing literature utilizing different QRS morphological features beyond QRS duration and LBBB morphology to select patients for CRT.

Q-LV, a direct measure of delayed activation of LV free wall, is the duration from onset of native QRS on 12-lead ECG to local activation time at the LV pacing lead. A long Q-LV proves LV electrical dyssynchrony, but more importantly, pacing from a site with long Q-LV results in a favorable long-term clinical response to CRT [25]. Q-LV of ≥ 95 ms at the site of LV pacing is a strong predictor for beneficial CRT response.

Sex differences

There are sex differences in response to CRT that are worth consideration. In the MADIT-CRT trial, women were more likely to have a non-ischemic etiology for their cardiomyopathy, were more likely to have LBBB, and less likely to have renal dysfunction [29]. Furthermore, women had better outcomes with CRT when compared with men. Similarly, among non-ischemic cardiomyopathy patients with LBBB, Varma et al. showed greater rate of echocardiographic reverse remodeling with CRT among women as compared with men [30]. Importantly, response rate among women was high even when baseline QRS duration was < 150 ms (86 versus 36% in men). Women with a typical LBBB have narrower QRS (142 ± 22 versus 156 ± 24 ms in men) and therefore benefit from CRT despite QRS duration < 150 ms [31•].

Atypical LBBB

Pastore et al. have shown that among patients with atypical LBBB morphology, there are predictors of a larger Q-LV that can be utilized to improve patient selection [24••]. These include (i) QRS duration > 150 ms, (ii) notching/slurring in ≥ 1 lateral lead, (iii) absence of R wave (≥ 1 mm) in lead V1 and/or Q wave (≥ 1 mm) in lead aVL, and (iv) time to intrinsicoid deflection in lead V6 > 60 ms.

Not all RBBB is the same

Pastore et al. have demonstrated the clinical benefit of CRT among patients with non-LBBB morphologies [23••, 24••]. They highlight the significance of differentiating typical and atypical RBBB. Atypical RBBB is defined by the absence of the characteristic S wave in leads I and aVL ($S_{\text{duration}} < R_{\text{duration}}$ and < 40 ms). This absence of the S wave in lateral ECG leads with RBBB pattern is suggestive of a concomitant delayed activation of LV free wall and commonly presents with left-axis deviation. Patients with atypical RBBB as compared with typical RBBB have a longer Q-LV (> 110 ms) and accordingly favorably respond to CRT (71 versus 19%).

ECG measures of dyssynchrony

Vereckei et al. have formulated ECG criteria to predict intra-LV and interventricular dyssynchrony [32•]. On ECG, intra-LV dyssynchrony is the difference between the times to intrinsicoid deflection in leads aVL (lateral wall) and aVF (inferior wall) divided by the total QRS duration, i.e., $(\text{aVL}_{\text{ID}} - \text{aVF}_{\text{ID}}) / \text{QRS}_{\text{duration}}$. Interventicular dyssynchrony is the difference in times to intrinsicoid deflection in leads V5 (LV) and V1 (RV) divided by QRS duration, i.e., $(\text{V5}_{\text{ID}} - \text{V1}_{\text{ID}}) / \text{QRS}_{\text{duration}}$. Either of these ECG dyssynchrony parameters > 25% predict benefit with CRT.

Plesinger et al. measured ventricular electrical activation delay among patients with LBBB morphology using an optimized computerized approach [33•]. ECGs sampled at 1 kHz were filtered at high-frequency bands, and the maximal amplitude for the lateral (V5 and V6) and septal (V1 and V2) leads was obtained from the median high-frequency QRS shape. Ventricular activation delay, computed as the time between maxima in the septal and the lateral leads, when ≥ 31.2 ms (first quartile) predicted greater echocardiographic and clinical response to CRT.

Mollo et al. described another measurement in patients with LBBB morphology that predicts CRT response: time from peak of the R wave to the nadir of the S wave in lead V1 (V1_{RS}) [34]. A much higher response rate to CRT was observed in patients with baseline $\text{V1}_{\text{RS}} \geq 45$ ms compared with < 45 ms (86 versus 33%).

Poposka et al. showed in both LBBB and non-LBBB morphologies predictors of CRT response simply include larger R amplitude, smaller S amplitude, and a larger $R_{\text{amplitude}}/S_{\text{amplitude}}$ ratio in lead V6 [35•].

3D QRS area

Ventricular depolarization results in generation of the electrical potential recorded as the QRS on ECG. Synchronized LV activation results in cancelation of opposing wavefronts and smaller overall QRS amplitude. Conversely, dyssynchronous or unopposed sequential depolarization of the LV results in additive overall large QRS amplitude. Three-dimensional (3D) QRS area or area under the QRS is the voltage-time integral of the QRS from vectorcardiography. The vectorcardiogram can be reconstructed from standard 12-lead ECG using a conversion matrix. The area under the QRS is then traced in the x, y, and z coordinates, and the root-mean-square of the three spatial QRS areas gives the 3D QRS area [26••, 36••]. The QRS area combines the effects of time taken for ventricular activation with the magnitude of the electrical potential and has emerged as a novel marker for electrical dyssynchrony.

van Stipdonk et al. reported that CRT recipients with a baseline 3D QRS area greater than median 108 μVs had a markedly lower rate of adverse heart failure clinical endpoints including death and much higher echocardiographic response [26••]. The discrimination of CRT response was superior by baseline 3D QRS area than QRS duration or LBBB morphology. Emerek et al. have confirmed these findings and shown ability of 3D QRS area to predict clinical response to CRT among subgroups by QRS duration ≥ 150 or < 150 ms, and LBBB or non-LBBB morphology [36••].

Fractionation in QRS

A small study by Celikyurt et al. studied non-ischemic cardiomyopathy patients with LBBB who had fractionation in their baseline QRS [37•]. They observed that a short QRS onset to onset-of-QRS-fractionation time (< 32.5 ms) and a longer fractionation duration were strong correlates of echocardiographic CRT non-response. The authors hypothesize that LV electrical dyssynchrony produces short-duration fractionation in mid-QRS, and a pattern of early and prolonged fractionation are atypical features suggestive of fibrotic scar or delayed activation in regions other than LV free wall.

Predicting response after CRT

After implantation of a CRT device, ECG allows evaluation of the delivered electrical result. Table 2 summarizes multiple studies that have evaluated a variety of markers on post-CRT ECG as a predictor of clinical response. Most publications have evaluated narrowing of QRS with CRT as a favorable prognostic finding. In a recent study, Jastrzebski et al. showed a strong correlation between acute QRS shortening with CRT and subsequent survival free of clinical heart failure endpoints including death [38•].

Among all the varied published QRS features correlating with a good response to CRT, the common element is the degree of contribution from early recruitment of the LV free wall. Early activation of the LV free wall, in combination with activation of the LV septum, reduces the QRS duration. With activation spreading in a posterior to anterior direction from the free wall, the anteriorly placed leads V1 and V2 develop a positive R wave. Similarly, activation directed away from the lateral leads recording the LV free wall results in a negative S wave in the lateral leads I and aVL, and a right axis deviation.

Table 2. Post-CRT ECG correlates of favorable clinical response

QRS duration	• Narrower QRS (absolute or indexed to baseline QRS duration); QRS narrowing ≥ 25 ms versus baseline [38•, 39–46] • QRS normalization [40]
Lead V1 (or V2)	• Presence of R wave [40, 43, 47, 48] • R wave amplitude (V1 or V2) [49] • $R_{amplitude}/S_{amplitude} \geq 1$[50] • RS interval narrowing ≥10 ms versus baseline [34]
Lead I (or aVL)	• Presence of S wave [47] • $R_{amplitude}/S_{amplitude} \leq 1$ [50] • Reversal from positive to negative [51]
QRS in frontal plane	• Shift from left to right axis deviation [49] • QRS vector amplitude is halfway between that in LBBB and LV pacing [52]

Why some CRT recipients do not respond?

Why some patients do not benefit with CRT while others do? In some cases this may be due to absence of delayed LV free wall activation to begin with, that would be responsive to CRT, i.e. Q-LV is not prolonged despite placing the LV lead on the free wall [25]. This can be overcome by improved patient selection for CRT, the crucial factor being evidence of delayed electrical activation of the LV free wall. As a corollary, the key feature of a favorable post-CRT ECG result is presence of traits confirming early recruitment of the LV free wall. Many patients, despite presence of baseline electrical dyssynchrony, may not develop a favorable ECG result due to inability of CRT to adequately resynchronize the LV. These patients understandably do not derive the anticipated clinical response.

Ineffective resynchronization

The inability of CRT to adequately correct the electrical dyssynchrony in an individual patient could be on account of a host of different reasons. These include placement of the LV lead outside the region of electrical delay, due to absence of a feasible coronary vein in the target region or poor target selection, with an ensuing smaller Q-LV. Regions remote from the target basal-to-mid lateral LV wall, i.e. sites that are too superior (anterior interventricular vein), too inferior (middle cardiac vein), or too apical, are not effective in resynchronizing the LV free wall [15]. Some patients may, however, not develop a favorable ECG result despite lead placement at a putatively suitable site. Sometimes this may be due to irrecoverable advanced cardiomyopathy. But on other occasions, pacing from a seemingly satisfactory site may still fail to recruit the LV free wall in a timely fashion [53].

LV pacing latency

Latency in recruiting enough myocardium to initiate a QRS complex is not uncommon when pacing cardiomyopathic hearts from the LV epicardium [54, 55]. This can be due to regions of conduction blocks or slow conduction that the paced wavefront has to circumnavigate before breaking out to the global ventricular myocardium. Yagishita et al. reported that approximately 40% of their CRT recipients did not respond despite Q-LV ≥ 95 ms [56••]. Non-response was dependent on LV stimulation to QRS onset (S-QRS) ≥ 37 ms during pure LV pacing. This latency prolonged the pure LV paced QRS duration (stimulation to QRS offset), and even though RV-LV pacing offset was optimized to minimize the biventricular paced QRS duration, over two-thirds of patients did not respond to CRT. The solution, when feasible, may be either to find a pacing site with Q-LV ≥95 ms without substantial pacing latency, else to program RV-LV offset with adequate early stimulation of LV relative to RV to overcome the effect of LV pacing latency. Other authors have also reported a longer LV paced QRS duration as a predictor of CRT non-response, and it is unclear how much is due to irreversible advanced cardiomyopathy and how much is circumventable by optimizing RV-LV pacing offset [43, 57•].

Optimization of CRT programming

Among individual CRT recipients without ECG markers predictive of a good CRT response in reported studies, it is unclear if the cause was remediable RV-LV offset programming, or non-programmable factors like electrode location and myocardial fibrosis. It therefore remains unknown how many patients will convert to a favorable ECG with RV-LV offset optimization [53].

Non-electrical imaging-based techniques have been evaluated to individually program RV-LV offset to acutely optimize cardiac mechanics and hemodynamic performance with variable results. These methods included transthoracic echocardiography [58–60], gated SPECT perfusion imaging [61], invasive hemodynamics [62, 63], digital photoplethysmography [64], and pacemaker lead sensors measuring peak endocardial acceleration [65, 66]. However, these techniques have failed on account of being cumbersome, non-standardized, lacking reproducibility, and failure to show improvement in clinical outcomes. Echocardiography to prospectively guide RV-LV offset optimization has not been demonstrated to be superior to simultaneous biventricular pacing without RV-LV offset [67, 68].

The aforementioned techniques focus on acute mechanical effects, rather than evaluation of electrical resynchrony. Their disappointing results have reinforced the maxim that an electrical problem requires an electrical solution [69], and the long-term effects of CRT may be causally dependent on its electrical rather than acute mechanical result. Yet, ECG has only been evaluated prospectively in limited-scope small studies for RV-LV offset optimization. There are no well-accepted and prospectively validated strategies for CRT programming demonstrating long-term clinical benefit. However, hope remains that advances in electrical optimization may improve the magnitude of response to CRT and increase the proportion of clinical responders.

Aside from selection of the target LV vein for delivering the LV pacing lead, a few factors can have an operational impact on the resultant ECG. These include selecting the pacing electrode from the available choices in context of multi-electrode (often quadripolar) leads, pacing from multiple LV electrodes (multipoint pacing) [70], selecting short atrioventricular delay to promote CRT pacing over dyssynchronous intrinsic conduction, allowing LV pacing to fuse with intrinsic atrioventricular conduction (adaptive LV pacing) [71], and lastly, appropriately selecting RV-LV pacing offsets to maximize electrical resynchrony with biventricular pacing.

Minimizing paced QRS duration

Bertini et al. showed over a decade ago that using minimum biventricular paced QRS duration to select RV-LV offset correlated with programming to optimize acute LV hemodynamics on echocardiography [45]. In a recent Turkish trial, Sipal et al. showed that evaluation of biventricular paced QRS from the multiple target LV veins for guiding lead placement to minimize paced QRS duration results in substantial QRS narrowing and much greater likelihood of reverse remodeling compared with unguided lead placement [72•].

Fusion optimized programming

Trucco et al. have recently shown that compared with nominal settings, programming atrioventricular delays and RV-LV offset to achieve the narrowest QRS by fusion of LV pacing, intrinsic conduction, and RV pacing correlated with greater long-term LV reverse remodeling [73•].

Is QRS duration the best measure of resynchrony?

Programming to minimize the QRS duration may still not be the most optimal method for RV-LV offset selection [74]. Ventricular activation from pacing via epicardial coronary veins, distant from the endocardial His-Purkinje conduction system, is inherently different from intrinsic conduction or endocardial pacing. Epicardial pacing results in an initial low-amplitude slurred pseudo-delta wave on ECG as the local wavefront slowly propagates transmurally before global breakout generates the larger QRS deflection. In other words, it takes longer for the LV epicardial stimulation to engage the entirety of the LV free wall. This latency can be further amplified by conduction slowing and blocks from epicardial and mid-myocardial fibrosis commonly seen in various cardiomyopathies. Synchronous activation of the LV free wall may then require substantial advancement of LV pacing compared with RV pacing. This may be able to maximize resynchrony, yet result in a wider QRS on account of the initial latency and pseudo-delta wave [53].

Using RV and LV pacing latency

In a small study by Vidal et al., RV-LV offset chosen to minimize QRS duration did not predict acute mechanical resynchrony on tissue Doppler echocardiography [74]. Rather, adjustment of RV-LV offset to account for the respective RV and LV pacing latencies resulted in an improved acute response.

Wavefront fusion

ECG can be used to select RV-LV offset that results in a fusion of the LV-paced wavefront with RV pacing or intrinsic conduction [52]. Gage et al. have shown such optimization of CRT programming, as compared with nominal non-optimized programming, to result in favorable reverse remodeling in patients with presence of myocardial scar on MRI [75•].

Adaptive LV pacing

This refers to LV only pacing timed to fuse with intrinsic conduction with LBBB, resulting in a normalized or narrow QRS complex, and has been shown to be superior to echocardiographically optimized biventricular pacing [71].

Electrocardiographic imaging and 3D QRS area

Hitherto unpublished, we have studied the spectrum of RV-LV pacing offsets in individual patients using panoramic non-invasive electrocardiographic imaging [10]. We found that the RV-LV offset that minimized ventricular electrical uncoupling (difference in mean activation time over RV versus LV epicardium) [11] correlated with minimal 3D QRS area, and not with shortest QRS duration. As a summed global voltage-time integral, 3D QRS area is a conceptually appealing marker of resynchrony but is yet to be evaluated prospectively for programming CRT [26••, 36••].

Summary: ECG for CRT optimization

Currently, there is scant literature on prospective strategies for electrical optimization of CRT. The de facto target of ECG optimization is narrowing the paced QRS. Though QRS narrowing is definitively useful, in many situations, a wider paced QRS with advancement of LV relative to RV pacing results in improved fusion between LV and RV wavefronts and may be a more synchronized result. Electrocardiographic imaging or 3D QRS area rather than the QRS duration alone may be better gauges of optimal RV-LV offset programming but await prospective evaluation.

Conclusion

ECG remains the ubiquitous practical method for global ventricular electrical mapping in context of CRT. ECG has an evolving role especially in defining baseline electrical dyssynchrony to further improve patient selection for CRT and is increasingly being evaluated as a simple and reproducible endpoint for optimal CRT programming.

Compliance with Ethical Standards

Conflict of Interest

The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

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