CLINICAL RESEARCH

Right ventricular pump function after cardiac resynchronization therapy: A strain imaging study

Amélioration de la fonction pompe ventriculaire droite par resynchronisation : étude par imagerie de déformation myocardique

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Summary

Background. — Cardiac resynchronization therapy (CRT) produces an early improvement in left ventricular (LV) function in patients with congestive heart failure (CHF), but little is known about its effects on right ventricular (RV) function.

Aim. — To assess the early effects of CRT on RV function using myocardial strain analysis.

Methods. — Fifty CHF patients (New York Heart Association class III/IV, left ventricular ejection fraction (LVEF) less than 35%, QRS greater than 120 ms) were studied before and three months after CRT. RV chamber dimension was quantified using tricuspid annulus diameter and RV

KEYWORDS
Cardiac function; Cardiac resynchronization therapy;

Abbreviations: CHF, congestive heart failure; CRT, cardiac resynchronization therapy; LV, left ventricular; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVE SV, left ventricular end-systolic volume; NYHA, New York Heart Association; PVC, pulmonary valve closing; PVO, pulmonary valve opening; RV, right ventricular; RV lax, major axis of the right ventricle; RV sax, maximum dimension of the middle third of the right ventricle parallel to the tricuspid annulus; SD, standard deviation; TAPSE, tricuspid annulus plane systolic excursion; TDI, tissue doppler imaging; TV diam, tricuspid valve diameter; Vs, peak systolic velocity of the tricuspid annulus.

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Introduction

Recent clinical trials have demonstrated the beneficial effects of CRT on survival in large groups of patients with CHF related to LV systolic dysfunction [1—5]. The same clinical trials have also shown a consistent increase in LVEF and cardiac output [6—8] and a decrease in LV volumes and mitral regurgitation [9]. This LV reverse remodelling appears to be a marker for decreased risk of death and reduced morbidity [5,10].

In addition to LV function, RV size and function, combined with pulmonary artery pressure assessment, have been shown to play a key role in risk stratification [11—13] and response to medical therapy in patients with CHF [14]. A recent study in a subgroup of patients with severe RV dilatation indicated that CRT may induce a slight RV reverse remodelling six months after implantation [15]. However, reports on the early effects of CRT on RV function are scarce.

Accurate non-invasive assessment of RV function is difficult due to the structural and anatomical complexity [16]. New indices of RV systolic function have been proposed recently, based on pulsed tissue doppler analysis of tricuspid annulus velocities and strain imaging of the RV lateral wall [16—18]. Therefore, the aim of our study was to analyse the short-term effects of CRT on RV function as assessed by TDI and strain imaging.

Methods

Patients and study protocol

Fifty patients with severe systolic heart failure, selected for implantation of biventricular pacing, were included in this study prospectively at two university hospitals (Louis-Pradel Hospital, Lyon and Pontchaillou Hospital, Rennes).
The protocol was approved by the University of Lyon Institutional Review Board. All patients were selected according to the recent recommendations for CRT: NYHA class III or IV, sinus rhythm, optimal pharmacological therapy (including angiotensin-converting enzyme inhibitors and/or angiotensin II receptor antagonists, β-blockers and spironolactone if tolerated), QRS width greater than 120 ms, LVEF less than 35% [19,20].

Patients with heart valve prostheses, recent myocardial infarction and/or coronary revascularization (less than 3 months) were excluded. Clinical, electrocardiographic and echocardiographic parameters were recorded prospectively before implantation and at three-month follow-up.

The study population was divided into two groups according to the presence of LV mechanical delay: mechanical LV asynchrony was defined as a delay between two opposite walls greater than 65 ms, measured as the time difference from the onset of the QRS complex to the $V_s$ wave (TDI mode) within the basal and mid segments (septal versus lateral, inferior versus anterior, anteroseptal versus posterior) [9,21].

### Echocardiography

Patients were studied immediately before implantation and three months after CRT. All echocardiographic assessments were made using a Vivid 7 system (GE-Vingmed, Milwaukee, Wisconsin, USA). Gray-scale two-dimensional and TDI cineloops were obtained at end-expiratory apnoea from three consecutive cardiac cycles triggered from the QRS complex. Patients were imaged in left lateral decubitus position. Images were obtained using a 3.5 MHz transducer, at a depth of 12 to 20 cm in parasternal and apical views. Stroke volume was calculated with pulsed doppler from the longitudinal long-axis view and LVEF was assessed by biplane Simpson’s rule [22]. Severities of mitral and tricuspid regurgitation were graded semiquantitatively from colour-flow doppler images. Apical four-chamber views were used to quantify mitral and tricuspid regurgitation, which were classified as:

- mild = 1+ (jet area/atrial area less than 10%);
- moderate = 2+ (jet area/atrial area 10–20%);
- moderately severe = 3+ (jet area/atrial area 20–45%);
- severe = 4+ (jet area/atrial area greater than 45%) [23,24].

Pulmonary artery systolic pressure was estimated using continuous wave doppler imaging of the transtricuspid maximal regurgitant flow velocity. All echocardiographic data were analysed offline using commercial software (Echopac, General Electrics) by two independent observers blinded to all other patient data.

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![Figure 1](image-url). RV function assessment. Panel A shows measurement of the TAPSE. Panel B shows the strain curves recorded within the basal (yellow), mid (blue) and apical (red) segments of the RV lateral wall. Panel C shows the velocity curve of the tricuspid annulus.
Assessment of RV dimensions

RV dimensions were assessed as described previously from the apical four-chamber view [15,25] using the diameter of the tricuspid valve annulus (defined as the point of attachment of the septal and posterior leaflets to the atrioventricular junction; TV diam), the maximum dimension of the middle third of the RV parallel to the tricuspid annulus (RV sax) and the major axis of the right ventricle (defined as the distance between the RV apex to the mid-point of the tricuspid annulus; RV lax).

RV function assessment

Longitudinal RV function was assessed using motion and deformation parameters (Fig. 1). With regard to motion, the RV base-to-apex shortening was measured by M-mode as the systolic displacement of the lateral portion of the tricuspid annular plane systolic excursion (TAPSE, mm) [16] and peak systolic velocity ($V_s$, cm s$^{-1}$) was measured using tissue doppler velocity analysis of the tricuspid annulus [17]. Deformation was assessed by strain curves obtained within the basal, mid and apical segments of RV lateral wall strain. Peak systolic strain ($s$, %) within the basal, mid and apical segments of RV lateral wall was measured using strain values, was compared with both LV and RV mechanical delay . A significant.

Reproducibility

To test the reproducibility of echocardiographic parameters, 10 measurements were reanalysed at random. Interobserver and intraobserver variability were calculated as the difference between the two observations divided by the mean of the observations and were expressed as absolute numbers and percentages [28]. The reproducibility is reported in Table 1.

Cardiac resynchronization therapy

CRT was initiated with implantation of an atriobiventricular pacing system (InSync ICD II Marquis 7289; Medtronic, Minneapolis, MN) by an electrophysiologist who had no knowledge of the echocardiographic or tissue doppler data. CRT device and lead implantation were successful in all patients without any complications. For each patient, atrioventricular delay optimization was performed using the iterative method based upon mitral flow duration and lack of A-wave truncation. V-V optimization was performed using the aortic flow velocity time integral. Successful LV resynchronization was defined as LV mechanical delay less than 65 ms (21).

Statistical analysis

Data are presented as mean ± standard deviation (S.D.). A two-tailed Student’s t-test was used to compare paired and unpaired data, as appropriate. Proportional differences were evaluated using Fisher’s exact test. Improvement in RV function, as assessed by the ratio of the difference between post-CRT and baseline strain values divided by baseline strain values, was compared with both LV and RV mechanical delay. A P-value less than 0.05 was considered statistically significant.

Results

Patient disposition and characteristics

We studied 50 consecutive patients with CHF referred for CRT; three (6%) of these patients were excluded because of poor image quality that was unsuitable for quantitative analysis. In the remaining 47 patients, strain analysis was feasible in all RV segments.

Baseline patient characteristics are shown in Table 2. All patients were classified as NYHA class III (68%) or IV (32%). LV function at baseline is summarized in Table 3. All patients presented with a dilated left ventricle. Mean QRS duration was 163 ± 28 ms (all QRS durations were greater than 120 ms). The RV lead position was apical in 12 (26%) patients and septal in 35 (74%) patients. The LV lead position was lateral in 31 (66%) patients and posterolateral in 16 (34%) patients.

Mean intraventricular mechanical delay was 118 ± 81 ms. Before CRT implantation, LV longitudinal dyssynchrony was

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Reproducibility of echocardiographic parameters (n=15).</th>
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<tbody>
<tr>
<td></td>
<td>$V_s$ (cm s$^{-1}$)</td>
</tr>
<tr>
<td>Intraobserver variability (%)</td>
<td>5</td>
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<tr>
<td>Interobserver variability (%)</td>
<td>7</td>
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<table>
<thead>
<tr>
<th>Table 2</th>
<th>Baseline patient characteristics (n=47).</th>
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<tr>
<td></td>
<td>Mean age ± S.D. (years)</td>
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<td></td>
<td>Men (%)</td>
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<td></td>
<td>Heart failure aetiology</td>
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<td></td>
<td>Ischaemic, n (%)</td>
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<td></td>
<td>Non-ischaemic, n (%)</td>
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<td></td>
<td>Mean QRS duration ± S.D. (ms)</td>
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Right ventricular pump function after cardiac resynchronization therapy

Table 3  LV and RV dimensions and function at baseline and three months after CRT.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Three-month follow-up</th>
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<tbody>
<tr>
<td><strong>LV dimensions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>232 ± 73</td>
<td>219 ± 78*</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>187 ± 77</td>
<td>162 ± 67*</td>
</tr>
<tr>
<td><strong>Left atrial area (mm²)</strong></td>
<td>30 ± 10</td>
<td>28 ± 10*</td>
</tr>
<tr>
<td><strong>Mitral regurgitation (grade)</strong></td>
<td>1.8 ± 1.0</td>
<td>1.6 ± 0.9*</td>
</tr>
<tr>
<td><strong>LV function</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>22 ± 6</td>
<td>27 ± 9*</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>3.1 ± 1.1</td>
<td>3.6 ± 1.1*</td>
</tr>
<tr>
<td><strong>RV dimensions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TV diam (mm)</td>
<td>31 ± 6</td>
<td>31 ± 6</td>
</tr>
<tr>
<td>RV sax (mm)</td>
<td>29 ± 9</td>
<td>27 ± 8</td>
</tr>
<tr>
<td>RV lax (mm)</td>
<td>83 ± 10</td>
<td>81 ± 10</td>
</tr>
<tr>
<td><strong>RV function</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TAPSE (mm)</td>
<td>15 ± 5</td>
<td>16.8 ± 3.7</td>
</tr>
<tr>
<td>Vₛ (cm s⁻¹)</td>
<td>5.3 ± 2.4</td>
<td>6.4 ± 1.8*</td>
</tr>
<tr>
<td>Basal strain (%)</td>
<td>23 ± 9</td>
<td>28 ± 9*</td>
</tr>
<tr>
<td>Mid strain (%)</td>
<td>20 ± 7</td>
<td>25 ± 8*</td>
</tr>
<tr>
<td>Apical strain (%)</td>
<td>20 ± 10</td>
<td>22 ± 12</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mmHg)</td>
<td>34 ± 14</td>
<td>30 ± 9</td>
</tr>
<tr>
<td>Tricuspid regurgitation (grade)</td>
<td>2.5 ± 2.5</td>
<td>1.8 ± 1.0*</td>
</tr>
<tr>
<td>RV dyssynchrony (ms)</td>
<td>71 ± 48</td>
<td>43 ± 30*</td>
</tr>
</tbody>
</table>

Data are presented as mean ± S.D.

*P < 0.05 compared with baseline values.

present in 35 (74%) patients using the opposing wall delay cut-off value greater than 65 ms. Patients with LV mechanical dyssynchrony presented with a higher LVEDV than those without LV mechanical dyssynchrony. However, LVEF was similar in both groups before CRT (Table 4).

**RV baseline characteristics**

RV dimensions at baseline showed overall dilated morphology. Mean baseline values for tricuspid valve diameter, RV short axis and long axis and RV function parameters are listed in Table 3. In comparison with control values reported in the literature, RV function was depressed as shown by the low values for TAPSE, systolic annulus velocity and strain. Table 4 shows RV function parameters in patients with and without LV mechanical dyssynchrony; no differences were observed between the two groups at baseline. The RV long axis was significantly larger in patients with LV mechanical dysynchrony than in those without LV mechanical dysynchrony. No significant relationship was found between RV function parameters and RV dimensions at baseline (Fig. 2).

**Effects of CRT at three-month follow-up**

At three-month follow-up, the NYHA class was 2.8 ± 0.8 (versus 3.3 ± 0.5 at baseline; P < 0.01), QRS duration was 139 ± 23 ms (versus 163 ± 28 ms at baseline; P < 0.01) and LV mechanical delay was 32 ± 33 ms (versus 118 ± 81 ms at baseline; P < 0.01). LV resynchronization was considered to be successful in 41 patients (87%) based on an intraventricular mechanical delay value less than 65 ms.

**LV function**

CRT induced a significant increase in LVEF (P < 0.01) and a significant decrease in LVEDV (P < 0.05) between baseline and three-month follow-up. In parallel, there were significant decreases in mitral regurgitation and left atrial dimensions (Table 3). Moreover, CRT induced a significant improvement in LV diastolic parameters between baseline and three-month follow-up:

- a reduction in E-wave velocity (79 ± 36 cm s⁻¹ versus 54 ± 29 cm s⁻¹, respectively; P < 0.01);
- a reduction in E/A velocity ratio (1.4 ± 1.0 versus 0.9 ± 0.4, respectively; P = 0.01);
- an increase in E-wave deceleration time (201 ± 63 ms versus 229 ± 68 ms, respectively; P = 0.03);
- an increase in E wave duration (170 ± 42 ms versus 213 ± 54 ms, respectively; P = 0.04).

LVEF increased significantly in patients with mechanical LV dyssynchrony at baseline but not in patients without LV mechanical delay (Table 4). Of note, CRT did not induce any change in systolic pulmonary artery pressure between baseline and three-month follow-up (P = 0.36).

**RV function**

RV dimensions were similar at baseline and three-month follow-up (Table 3). In contrast, RV function improved between baseline and three-month follow-up, as shown by the increase in both motion and deformation parameters (Fig. 3). Vₛ, RV lateral wall basal strain and RV lateral wall mid strain increased significantly (P = 0.001, P = 0.009 and
RV apical lead (0.37% versus 0.04% = 0.79). This improvement in RV function was not related to changes in pulmonary artery pressure (from 34 ± 14 mmHg at baseline to 30 ± 9 mmHg at three-month follow-up; P = 0.36). Tricuspid regurgitation also decreased significantly between baseline and three-month follow-up (P = 0.05; Table 3).

RV mechanical dyssynchrony decreased significantly after CRT (P = 0.002; Table 3). The increase in RV basal strain after CRT was related significantly to RV dyssynchrony at baseline (r = 0.74; P < 0.05) suggesting that the greater the RV dyssynchrony was before CRT, the greater the improvement in RV function was after CRT (Fig. 4). Conversely, LV mechanical dyssynchrony at baseline was not predictive of the improvement in RV function after CRT as assessed by the increase in RV basal strain (r = 0.04, P = 0.79).

The improvement in RV function was of particular relevance in patients with RV septal lead as they presented with a significantly higher increase in RV strain than patients with RV apical lead (0.39 ± 0.49% versus 0.04 ± 0.37%, respectively; P = 0.03).

**Discussion**

This study is the first to show that CRT induces a significant and early (after three months) improvement in RV function, independent of any RV reverse remodelling or decrease in pulmonary artery pressure.

CRT has been shown to improve morbidity and mortality in NYHA class III or IV patients with CHF with abnormal electrical activation and LVEF less than 35% [1–4]. The potential mechanisms explaining these results are related to the early and on-going LV reverse remodelling induced by CRT, as demonstrated by an improvement in LV systolic function [6–8] and a decrease in mitral regurgitation [9] and LV volumes [5,29]. Our results are consistent with these observations.

The early LV remodelling occurred only in patients with LV mechanical dyssynchrony at baseline, emphasizing the important role for mechanical markers of LV dyssynchrony in identifying patients with a high likelihood of response to CRT [9,10,13,21,30–33]. Quantifying mechanical LV dyssynchrony is complex despite the variety of parameters provided by conventional echocardiography [31], TDI [9,21,30,33] and strain and strain rate imaging [32,34,35]. We assessed LV mechanical dyssynchrony by tissue doppler analysis of longitudinal myocardial velocities and defined it as a delay between two opposite walls greater than 65 ms [9,21,33].

RV function plays an important role in the prognosis of CHF patients [11–13]. Moreover, it seems that severe RV dysfunction might impair CRT efficiency [13]. Recently, CRT has been shown to induce both LV and RV reverse remodelling at six-month follow-up [15]; the decrease in RV dimensions occurred mainly in patients with severe RV dilatation at baseline and was associated with a decrease in tricuspid regurgitation and pulmonary artery pressure. Hence the positive effects of CRT on RV dimensions were related to the improvement in the loading conditions of the right ventricle. In addition, the RV reverse remodelling was shown to be related to the presence of LV dyssynchrony at baseline, but no mention was made of the effects of CRT on RV function.

Our data demonstrate that CRT induces early improvement in RV function, as shown by the significant increase in V̇e and RV myocardial strain at three-month follow-up. This improvement occurred without any change in RV dimensions and pulmonary artery pressure, indicating that the increase in systolic RV function was not related to changes in RV workload. We also demonstrated that the increase in systolic RV function was related to the extent of RV mechanical

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**Table 4** LV and RV dimension and function parameters at baseline and three months after CRT in patients with and without LV mechanical dyssynchrony at baseline.

<table>
<thead>
<tr>
<th></th>
<th>No LV mechanical dyssynchrony at baseline (n = 12)</th>
<th>LV mechanical dyssynchrony at baseline (n = 35)</th>
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<tbody>
<tr>
<td></td>
<td>Before CRT</td>
<td>After CRT</td>
</tr>
<tr>
<td><strong>Left ventricle</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV mechanical delay (ms)</td>
<td>37 ± 19</td>
<td>31 ± 32</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>208 ± 68</td>
<td>204 ± 92</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>178 ± 110</td>
<td>163 ± 77</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>24 ± 4</td>
<td>25 ± 4</td>
</tr>
<tr>
<td><strong>Right ventricle</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TAPSE (mm)</td>
<td>16 ± 5</td>
<td>16 ± 4</td>
</tr>
<tr>
<td>V̇e (cm s⁻¹)</td>
<td>4.7 ± 2.4</td>
<td>6.5 ± 1.5</td>
</tr>
<tr>
<td>Basal strain (%)</td>
<td>19 ± 7</td>
<td>27 ± 14</td>
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<td>Mid strain (%)</td>
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<tr>
<td>RV sax (mm)</td>
<td>27 ± 7</td>
<td>29 ± 10</td>
</tr>
<tr>
<td>RV lax (mm)</td>
<td>75 ± 12</td>
<td>81 ± 10</td>
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</tbody>
</table>

Data are presented as mean ± S.D.

*P < 0.05 compared with before CRT; **P < 0.05 compared with no LV mechanical dyssynchrony group.
dyssynchrony at baseline, suggesting that CRT has a positive effect on the coordination of the contractility of the RV lateral wall and the interventricular septum, in addition to its correction of LV mechanical dyssynchrony. This improvement in the coordination of RV contractility may explain the decrease in tricuspid regurgitation despite the lack of significant RV remodelling. We can postulate, therefore, that during LV pacing, RV function is improved through mechanical interaction with the left ventricle and resynchronization of the right ventricle. Recent preclinical and clinical studies have supported this hypothesis [36,37]. In a dog model of left bundle branch block, resynchronization was found to improve both RV and LV function, but RV function assessed by maximum rate of LV pressure change was improved by LV and biventricular pacing if RV activation remained synchronous [37]. In addition, biventricular pacing in patients
with heart failure was found to be associated with an acute improvement in \( V \) \[36\]. This observation was unique to the biventricular mode and was not observed in RV or LV pacing modes \[36\].

Finally, we found that patients with RV septal lead presented with a significantly higher increase in RV strain than patients with RV apical lead. This observation indicates the importance of the optimal RV lead positioning to promote clinical and haemodynamic improvement and LV reverse remodelling \[38\]. Midseptal positioning of the RV lead has been shown to be associated with a significant decrease in LVEDV and an increase in maximum oxygen uptake capacity. Unlike RV, apical pacing, RV septal pacing is associated with faster ventricular activation, reduced myocardial perfusion defects and wall motion abnormalities \[38\].

We did not find any statistically significant RV reverse remodelling as described by Bleeker et al. \[15\]; however, we studied patients three months rather than six months after CRT. As improvement in LV function precedes LV reverse remodelling \[1,5,8,9,21,29,32,33\], our data suggest that RV function improvement might precede RV reverse remodelling after CRT.

**Study limitations**

This cohort of 47 patients was followed for a short period of three months. A longer follow-up period would have facilitated assessment of the long-term effects of CRT on RV size and function. However, our principal aim was to demonstrate an early improvement in both LV and RV function. Our findings are in agreement with those of other studies that have documented the early effectiveness of CRT in inducing LV reverse remodelling and improvement in systolic function in patients with advanced heart failure \[29\]. In this study, clinical improvement was assessed by NYHA-classification, which is a subjective parameter. Unfortunately, peak oxygen consumption was not available at three-month follow-up.

**Figure 3.** RV strain and velocity at baseline and three months after CRT: both RV strain and velocity were improved three months after CRT in this illustrative patient.

**Figure 4.** Relationship between RV dyssynchrony at baseline and percentage increase in RV basal strain three months after CRT; the percentage increase in RV basal strain three months after CRT was related significantly to RV dyssynchrony at baseline.
Conclusions

We are reporting for the first time an early beneficial effect of CRT on RV function, in addition to an improvement in LV function. The cardiac resynchronization in Heart Failure Study has shown that CRT reduces mortality significantly due to a decline in deaths attributed to worsening heart failure [3]. The ability of CRT to reduce mortality is dependent on improvements in cardiac function and beneficial LV remodelling. The effect of CRT on RV function might also explain part of the positive effect observed, but this remains to be confirmed.

References

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diographic images to quantify dyssynchrony and predict response to cardiac resynchronization therapy. Circulation 2006;113(7):960–8.


