Echocardiographic evaluation of cardiac dyssynchrony

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First described a decade ago, cardiac resynchronization therapy (CRT) has recently become a proven therapeutic strategy for refractory heart failure. Large clinical trials have shown a reduction in both morbidity and mortality in patients treated with CRT. Initial patient selection has relied mainly on electrocardiographic criteria, which allows identification of only 70% of responders. Accordingly, echocardiographic criteria were developed to identify mechanical dyssynchrony in an effort to improve patient selection. Multiple echocardiographic criteria have since been proposed, with no consensus as to which parameter better predicts CRT response. Although comparison studies using different criteria are underway, current evaluation of dyssynchrony should probably use an integrated multiparameter approach. The objective of the present article was to review the role of echocardiography in the evaluation of cardiac dyssynchrony in clinical practice.

Key Words: *Cardiac resynchronization; Congestive heart failure; Echocardiography; Pacemakers*

Congestive heart failure (CHF) is a major health issue, with as many as 10% of individuals older than 65 years affected (1). Even though the medical management of CHF has improved substantially in recent years, morbidity and mortality remain high, especially for patients with poor functional class, despite optimal therapy. The deleterious effect of intraventricular conduction abnormalities has been known for several years (2). Initially described more than a decade ago (3), cardiac resynchronization therapy (CRT) has only recently become an approved therapeutic option (4), with the ability to restore a more physiological pattern of contractility to dyssynchronized failing hearts. CRT has proven to be effective in reducing both morbidity and mortality in large scale, prospective clinical studies (5-9). So far, patient selection has focused on electrocardiographic criteria, which have shown poor correlation with echocardiographically documented intraventricular dyssynchrony. Furthermore, the absence of response in up to 30% of patients in most studies highlights the need for improved selection criteria (10).

Relying on a more mechanistic approach, echocardiography will likely play a central role in the evaluation of dyssynchrony in the near future. Several echocardiographic modalities have been proposed for dyssynchrony evaluation, from conventional methods, such as M-mode and conventional Doppler assessment, to newer methods, such as tissue Doppler imaging (TDI) and TDI-derived techniques. The purpose of the present article was to review these different

L'évaluation échocardiographique de la dyssynchronie cardiaque

Décrit pour la première fois il y a dix ans, le traitement de resynchronisation cardiaque (TRC) est récemment devenu une stratégie thérapeutique démontrée pour l'insuffisance cardiaque réfractaire. De grands essais cliniques ont révélé une réduction de la morbidité et de la mortalité chez les patients ayant subi un TRC. La sélection initiale des patients se fondait surtout sur des critères électrocardiographiques, par lesquels on ne repérait que 70 % des répondants. Par conséquent, les critères échocardiographiques étaient élaborés pour repérer la dyssynchronie mécanique afin d'améliorer la sélection des patients. De multiples critères échocardiographiques ont été proposés depuis, sans qu'on parvienne à un consensus sur les paramètres pour prévoir la réponse au TRC. Même si des études comparatives à l'aide d'autres critères sont en cours, une évaluation de la dyssynchronie devrait faire appel à une démarche multiparamétrique intégrée. Le présent article vise à analyser le rôle de l'échocardiographie dans l'évaluation de la dyssynchronie cardiaque en pratique clinique.

echocardiographic parameters of mechanical ventricular dyssynchrony, focusing on their added value in CRT decision-making.

PHYSIOPATHOLOGY

Normal left ventricular (LV) contraction occurs with synchronous onset of both electrical and mechanical activation. In the presence of intraventricular conduction abnormalities, such as left bundle branch block (LBBB), premature activation of certain LV areas occurs, creating regions of both early and late activation. Grines et al (2) illustrated the negative effects of these conduction abnormalities on myocardial function, present in as many as 40% of patients with dilated cardiomyopathy. Early activated regions begin contracting, although ejection has not yet started, and late contraction occurs when tension has already developed, thus at a higher energy cost. Overall, this abnormal activation sequence results in a reduction of stroke volume and ejection fraction (EF), as well as an increase in end-systolic volume and wall stress. Delayed activation of the LV free wall has been shown to be an important factor involved in global LV dysfunction (11) and continued remodelling (12). Asynchronous activation of the different myocardial regions also leads to delayed relaxation, resulting in a shortened diastolic filling interval. This, in turn, creates regional imbalances in myocardial bioenergetics and metabolism (13).

Another consequence of delayed and asynchronous activation involves the mitral valve, with potential deterioration of

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Figure 1) Measurement of aortic and pulmonary pre-ejection delays using conventional pulsed-wave Doppler. The systolic signals are obtained in a standard fashion: the pulsed-wave Doppler gate is placed in the left ventricular outflow tract just under the aortic valve from the apical five-chamber view for the aortic ejection signal, and in the right ventricular outflow tract just under the pulmonary valve from the parasternal short axis view for the pulmonary ejection signal. Measurements are performed by placing the first calliper at the QRS onset and the second calliper at the onset of the pulsed-wave Doppler ejection signal. A difference of more than 40 ms between right and left pre-ejection intervals confirms the presence of interventricular dyssynchrony

pre-existing mitral regurgitation (MR) and even the development of diastolic MR associated with long PR intervals. LV dilation and increased spherical deformation cause an increase in the tethering forces acting on the mitral leaflets, while the closing forces are reduced by the delayed rate of pressure rise associated with LV dysfunction (14). These geometric changes underlie the functional MR often associated with dilated hearts.

DYSSYNCHRONY EVALUATION

Basic evaluation

Dyssynchrony evaluation begins with a standard, comprehensive echocardiographic examination, including evaluation of LV and right ventricular (RV) systolic functions, LV diastolic function and filling pressures. Particular attention should be paid to the presence, mechanism and severity of MR. Although not an indication for CRT in itself, the presence of significant MR lends additional support to an indication for biventricular pacing. Indeed, a decrease in MR grade has consistently been shown in patients undergoing CRT (6,15). Most of this reduction is immediate, and related to improved ventricular synchrony and increased LV closing force (as reflected by an increased delayed rate of pressure rise) (14), as well as improved coordination of papillary muscle activation (16). A further improvement attributed to LV reverse remodelling has also been reported.

According to current guidelines (4), patients eligible for CRT must have symptomatic CHF (New York Heart Association [NYHA] functional class III or IV) with a prolonged QRS duration (more than 120 ms) and an LVEF of less than 35% despite optimal medical treatment. After establishing patient eligibility, a thorough assessment of dyssynchrony

includes evaluation of both interventricular (between LV and RV) and intraventricular dyssynchrony.

INTERVENTRICULAR DYSSYNCHRONY

The presence of interventricular dyssynchrony is easily assessed by measuring the difference between left and right pre-ejection intervals. These intervals are measured from the onset of the QRS complex to the onset of the corresponding Doppler ejection signal, as shown in Figure 1. Whereas values of 20 ± 10 ms have been measured in healthy control subjects, a delay of more than 40 ms is considered indicative of interventricular dyssynchrony (17,18).

Measurement of the delay between peak systolic velocity of the RV free wall and the LV by TDI has also been used to assess interventricular dyssynchrony (19,20). The presence of interventricular dyssynchrony, however, appears to be weakly correlated with morbidity, mortality (17,18) or response to CRT (19,20), thus limiting its clinical usefulness. It is interesting to note that interventricular delay is well correlated with QRS duration, an unsatisfactory criterion for CRT patient selection (21). Although not an indication for biventricular pacing per se, interventricular dyssynchrony may support the indication for CRT when associated with intraventricular dyssynchrony.

INTRAVENTRICULAR DYSSYNCHRONY

Qualitative evaluation

The assessment of intraventricular dyssynchrony should first rely on qualitative visual evaluation. This essential first step constitutes a screening evaluation and is also useful to support information obtained by purely quantitative methods. In cases of dilated cardiomyopathy, an undulating systolic movement can be observed beginning at the level of the interventricular septum and extending to the other LV segments, typically with late activation of the LV lateral wall.

Quantitative evaluation

An evaluation based on quantitative criteria is fundamental in establishing the diagnosis and assessing the severity of LV dyssynchrony. The evaluation usually begins with standard echocardiographic criteria and proceeds with more advanced measurements to establish the presence of dyssynchrony.

Standard echocardiography

One of the first proposed quantitative criteria relied on M-mode recordings in association with transmitral pulsed-wave Doppler imaging (22). When the interval between QRS onset and peak posterior wall motion is greater than the interval between QRS onset and E wave onset, active systolic contraction is occurring during diastole, and dyssynchrony is considered to be present (Figure 2). Although used as one of the dyssynchrony criteria in the CArdiac REsynchronization in Heart Failure (CARE-HF) study (22) (in patients with QRS duration between 120 ms and 150 ms), this method is limited by the difficulty in precisely identifying systolic peaks and obtaining perpendicular M-mode tracings in some patients.

Another simple echocardiographic criterion described by Pitzalis et al (23) consists of measuring the delay between peak systolic contraction of the septal and posterior walls on M-mode recordings from the parasternal short-axis view (Figure 3). This delay was measured in 20 patients with LBBB and refractory CHF (LVEF of less than 35% and NYHA class III), before and one month after CRT. A septal-to-posterior wall motion

Figure 2) Comparison of the timings of posterior wall contraction with rapid filling phase. The M-mode recording is obtained from the parasternal long axis view, with the cursor at the tip of the mitral leaflets, and the delay (D1) from QRS onset to maximal posterior wall excursion is measured on the M-mode display. The transmitral pulsed-wave signal is obtained in a standard fashion, with the gate placed at the tip of the mitral leaflets in the apical four-chamber view and the delay (D2) from QRS onset to the mitral flow onset on the Doppler signal is measured. When contraction extends beyond the onset of rapid filling (D1 greater than D2), thus occurring during early diastole, intraventricular dyssynchrony is considered to be present

delay (SPWMD) longer than 130 ms was found to be a good predictor of reverse remodelling after CRT initiation (specificity of 63% and positive predictive value of 80%), with responders defined as patients having a 15% or more decrease in LV end-systolic volume. This criterion was later prospectively validated by the same group in 60 patients with similar inclusion criteria and was found to be associated with fewer clinical events during long-term follow-up (24). Although easily applied, this method is again limited by the difficulty in precisely identifying systolic peaks, particularly for the posterior wall, and in the presence of an akinetic interventricular septum. Of note, the largest and most recent study to evaluate SPWMD retrospectively analyzed the baseline echocardiograms of 79 patients from the CONTAK CD database (25). The authors reported an accurate identification of systolic peaks in only 45% of patients, in addition to low interobserver reproducibility. Most importantly, SPWMD did not predict clinical or echocardiographic response to CRT.

Finally, the aortic pre-ejection interval, measured from the onset of the QRS to the onset of aortic flow, has been proposed as a marker of LV dyssynchrony. Values ranging from 140 ms (6) to 160 ms have been used as indicators of dyssynchrony. In a study of 66 CHF patients with advanced NYHA functional class, an aortic pre-ejection delay of more than 150 ms predicted response to CRT (26). Although easily obtained, the main limitation of this method is that it evaluates global LV systolic dyssynchrony, rather than comparing the timing of activation of the different LV walls.

TDI

TDI is a relatively recent imaging modality that allows regional myocardial velocity measurements. Precise determination of the amplitude, timing of onset and peak systolic and diastolic velocities can be obtained in relation to the electrocardiogram signal. Several new techniques have been derived from TDI that yield a quantitative and detailed evaluation of LV dyssynchrony.

Pulsed-wave TDI

A standard TDI recording, with the sample volume placed in the centre of a myocardial segment (rather than on the mitral annulus as for diastolic function evaluation), normally shows two systolic and two diastolic deflections (Figure 4).

Figure 3) Measurement of septal-to-posterior wall motion delay between peak contraction of the interventricular septum and posterior wall on the M-mode display obtained from the parasternal long axis view, with the cursor at the tip of the mitral leaflets. Intraventricular dyssynchrony is present if the delay is more than 130 ms

Figure 4) Normal pulsed-wave tissue Doppler imaging recording obtained with the region of interest placed on the basal segment of the anterolateral wall from the apical four-chamber view. The Doppler tracing displays two systolic deflections, S wave and isovolumic contraction (IVC) signal, and two diastolic deflections, Ea and Aa waves, corresponding to early rapid filling and atrial contraction, respectively

Immediately after QRS onset, a first positive low amplitude systolic signal, corresponding to the isovolumic contraction period, is observed. The second positive systolic deflection, the S wave, is usually of higher amplitude than the first (amplitude between 10 cm/s and 20 cm/s and duration of 100 ms to 300 ms) (27). In diastole, two negative deflections are recorded, the Ea and Aa waves, corresponding to early rapid filling and atrial contraction, respectively.

From these recordings, two different time intervals may be measured: the electromechanical delay, from QRS onset to S wave onset (17,28), and the electrosystolic delay, from QRS onset to peak systolic contraction (29) (Figure 5). A delay of more than 40 ms between opposite LV walls for each of these intervals is indicative of dyssynchrony, whereas normal subjects display values of approximately 20 ms (17). Limitations of pulsed-wave TDI include the restriction of

Figure 5 Measurement of the electromechanical delay (EMD) and electrosystolic delay (ESD) using pulsed-wave tissue Doppler imaging with sampling in the basal segments of opposite left ventricular walls (the interventricular septum and the anterolateral wall) in the apical four-chamber view. The EMD is measured from the QRS onset on the electrocardiogram tracing to the onset of the S wave, and the ESD is measured from the QRS onset to the peak of the S wave. A difference of more than 40 ms between opposite walls for either EMD or ESD indicates intraventricular dyssynchrony

measurements to single points during one cardiac cycle and the sometimes difficult identification of low-amplitude systolic waves, especially in the presence of interfering motion artifacts. Thus, to compare different myocardial segments, multiple, nonsimultaneous acquisitions are required with the inherent limitation of variations due to changes in heart rate, loading conditions or respiration.

Reconstructed TDI analysis

To overcome some of the limitations of pulsed-wave TDI, colour TDI loops may be recorded and analyzed off-line as reconstructed signals. Integrated software allow to position different region of interest, with instantaneous visualization of time-velocity curves (Figure 6). These curves correspond to the spectral TDI pulsed-Doppler displays with the same systolic and diastolic deflections. Numeric signals offer the advantage of direct and simultaneous comparison between opposite LV segments, with immediate visualization of LV dyssynchrony. Reconstructed TDI is limited by poor temporal resolution, with differentiation of the two systolic waves (isovolumic contraction and S wave) sometimes being difficult.

Colour TDI has been studied in different dyssynchrony models, ranging from two to 12 segments. Bax et al (30) used a two-segment model (basal septal and lateral segments) in 22 patients with LBBB and refractory CHF (NYHA class III to IV and LVEF of less than 35%) (30). Colour TDI acquisitions were performed before and immediately after CRT implantation, and LV dyssynchrony was assessed by comparing septal with lateral electrosystolic delays. An immediate increase in LVEF (from $21 \pm 7\%$ to $31 \pm 9\%$, $P < 0.05$) was observed with CRT, with a corresponding improvement in functional status at three months. The septal-to-lateral delay decreased from 110 ± 33 ms before CRT to 20 ± 22 ms after initiation of therapy. It is interesting that this delay returned to baseline values when biventricular pacing was withheld. Subsequently, the same authors used a four-segment model to study dyssynchrony, evaluating basal segments in the septal, lateral, anterior and inferior walls. A delay between any two segments of more than 65 ms was shown to predict response to CRT with both sensitivity and specificity of 92% (19). Moreover, dyssynchrony severity was also found to correlate with prognosis (cardiac death and hospitalization for heart failure) in the study.

Figure 6 Numerical tissue Doppler imaging tracings reconstructed from colour tissue Doppler imaging acquisitions from the apical four-chamber view. This method allows direct, simultaneous comparison of systolic velocities and timing between opposite left ventricular walls, in this case the basal septum (yellow) and basal lateral (green) wall

Other authors (31) have recommended a more systematic approach with analysis of 12 LV segments from the three standard apical views, studying six basal and six mid-LV segments. This analysis was performed before and after CRT in 30 patients with CHF, LVEF less than 40%, NYHA class III to IV and prolonged QRS (31). An 'asynchrony index' was calculated from the average of the standard deviation of all 12 electrosystolic delays and responders were defined as patients having a 15% or greater reduction of indexed LV systolic volume. A cut-off value of more than 32.6 ms for the 'asynchrony index' predicting response to CRT was derived. In multivariate analysis, this index was the only parameter found to be correlated with reverse remodelling, and it could differentiate all responders from nonresponders. An assessment of multiple segments has the advantage of being more comprehensive, but it is, however, limited by time-consuming off-line analysis.

Strain and strain rate – tissue tracking

Noninvasive evaluation of regional function may be performed by measuring myocardial strain, or deformation. Strain is a dimensionless index, expressed as a percentage of change in length of myocardial fibres from their original length. By convention, lengthening, or expansion, has a positive value, while shortening, or compression, is assigned a negative value. Strain rate, the time derivative of strain, represents the rate of myocardial deformation. Derived from TDI velocity data, these parameters have the advantage of distinguishing active contraction from passive motion caused by tethering of adjacent myocardial regions. Data may be expressed either as bidimensional colour maps or tracings that depict strain/strain rate variations according to time. Parametric imaging of LV walls displacement, or tissue tracking (GE Medical Systems, Norway), is also derived from colour TDI data. Tracking represents regional longitudinal shortening (measured in millimetres) and is calculated as the velocity-time integral of the systolic TDI velocity curve. When displayed as a colour map, tissue tracking offers a simple visual evaluation of regional myocardial function and timing, thus allowing rapid assessment of LV dyssynchrony. Alternatively, data may be expressed as tracings of LV walls displacement as a function of time.

Figure 7) Myocardial deformation, or strain tracings, showing simultaneous longitudinal deformation of basal lateral (green) and basal septal (yellow) left ventricular walls from the apical four-chamber view. Intraventricular dyssynchrony is present, as evidenced by one of the segments contracting after aortic valve closure. DLC Delayed longitudinal contraction

Because colour maps and tracings identify myocardial segments with inappropriate contraction timing (Figure 7), these techniques have been proposed as markers of intraventricular dyssynchrony. In a study of 25 patients (32) with advanced, symptomatic CHF (NYHA class III to IV) and predominant LBBB, the percentage of segments with delayed longitudinal contraction (DLC) at the LV base was determined using tissue tracking and strain rate analysis. The authors suggested that DLC or postsystolic shortening, when defined as more than 30% of 12 LV segments contracting after aortic valve closure, could predict CRT response. In responders, the improvement in LVEF at follow-up was correlated with the reduction in segments displaying DLC at baseline. Despite theoretical advantages, clinical application of strain imaging remains limited, mainly because of variable reproducibility and significant operator dependency. Moreover, the correlation between DLC and LV dyssynchrony could not be duplicated in a study that compared strain imaging and TDI-derived asynchrony index to predict response to CRT (20). Eventually, the study of the other components of normal strain, namely, radial and circumferential strain, may prove to be more useful in predicting response to resynchronization (33).

Tissue synchronization imaging

Tissue synchronization imaging (TSI) (GE Medical Systems, Norway) is one of the newest methods to assess dyssynchrony. Derived from TDI, TSI consists of an automated analysis of electro-systolic delay data, with colour coding of this delay with reference to the QRS. An automated colour-coding algorithm assigns a different colour according to the timing of peak velocity, with green for earliest activation, followed by yellow-orange for intermediate activation and red for latest activation. Reconstructed as a colour map, TSI offers direct and rapid visualization of segments with asynchrony (Figure 8). In a recent study of 56 CHF patients (34) before and three months after initiation of CRT, TSI combined with TDI-derived asynchrony index (12 segments)

Figure 8) Apical four-chamber view using the tissue synchronization imaging mode. This parametric imaging modality allows direct visualization of segments with early displacement (in green) and late activation (in yellow-orange)

Figure 9) Colour map of intraventricular dyssynchrony derived from real-time three-dimensional acquisitions before and after cardiac resynchronization therapy. Early activated regions are represented in blue, while late activated regions are coloured red. **Left panel:** spontaneous rhythm; **Right panel:** Biventricular pacing 'on'

allowed accurate identification of CRT responders (sensitivity of 82% and specificity of 87%).

Real-time three-dimensional echocardiography/multislice imaging

Three-dimensional echocardiography, first as reconstructed images and more recently as real-time acquisition, provides reproducible assessment of LV volumes, dimensions and systolic function. Using dedicated reconstruction software, semi-automated border detection allows continuous volume calculation of all 16 myocardial segments simultaneously. Represented as regional volumetric curves during the cardiac cycle, the timing of minimal volume for each segment can be analyzed. With normal homogeneous activation, the different LV segments reach their minimal volume at the same time, whereas in the presence of dyssynchrony, minimal volumes are attained at different time points. Displayed as a polar colour map, real-time three-dimensional imaging offers simultaneous visualization of all segments, with easy identification of dyssynchrony (Figure 9). Two recent studies have used three-dimensional echocardiography to evaluate LV dyssynchrony. A preliminary study of 13 patients demonstrated the feasibility of three-dimensional echocardiography to determine the timing of regional volumetric changes (35).

More recently, Kapenatakis et al (36) derived a systolic dyssynchrony index by studying the temporal dispersion of

Figure 10) Multislice imaging with simultaneous acquisition of three standard apical views (two-, three- and four-chamber). This technique allows direct comparison of numeric tissue Doppler imaging tissue tracking tracings for all 12 basal and mid-left ventricular segments from the apical window (shown on right, simultaneous tissue tracking tracings of the six basal left ventricular segments). AVC Aortic valve closure

regional LV volumes, a higher value being associated with more important dyssynchrony. In a small subgroup of patients undergoing CRT, reverse remodelling occurred in responders and was associated with a reduction in dyssynchrony index. Limitations of current softwares include suboptimal imaging in patients with poor acoustic windows and time-consuming off-line analysis. Furthermore, the role of three-dimensional echocardiography for dyssynchrony assessment has not yet been thoroughly validated.

Figure 11) Diagram comparing the different phases of systole assessed by each dyssynchrony parameter. Electromechanical delay (EMD) and electro-systolic delay (ESD) evaluate early- and mid-systole, respectively, whereas delayed longitudinal contraction (DLC) assesses end-systole. ECG Electrocardiogram

Recently, another new imaging modality using a three-dimensional/four-dimensional matrix phased-array transducer was introduced, permitting simultaneous acquisitions of three standard apical views. Multislice imaging (triplane) enables direct comparison of all 12 basal and mid-LV segments simultaneously from the apical window (Figure 10). From these triplane acquisitions, multiple numeric TDI or tissue tracking tracings may be compared. This technology allows the creation of polar maps from TSI acquisitions, again offering a simple, rapid visual evaluation of dyssynchrony.

Parameter comparison

As detailed above, several individual parameters have been developed and validated. Limitations of these studies include the small number of patients, as well as different methodologies and end points used, rendering any comparison difficult. Thus, there is no consensus as to which parameter or parameters of dyssynchrony should be used. One study (20) compared a TDI-derived asynchrony index with strain rate imaging for prediction of LV reverse remodelling after initiation of biventricular pacing in 56 patients with conventional CRT indications. The standard deviation of all 12 electro-systolic delays was the best predictor ($r=-0.74$), whereas strain-derived parameters were found to be poor predictors of reverse remodelling. Furthermore, multiple segment models were better predictors than models evaluating a smaller number of segments. These results are in agreement with our experience in clinical practice, where significant variation between individual dyssynchrony parameters is often observed. In the absence of large comparative trials, dyssynchrony evaluation should probably

be an integrated multiparameter approach, not relying on a single parameter, which is somewhat similar to diastolic function assessment.

Of interest is the fact that these different parameters provide complementary information by studying different phases of systole: TDI-derived electromechanical and electrosystolic delays evaluate early- and mid-systole, tissue tracking characterizes mid- and end-systole, and strain analysis-derived DLC assesses end-systole (Figure 11). Hence, when comparing these parameters, it is important to understand that they assess dyssynchrony at different times of the cardiac cycle. Thus, an individual patient may have evidence of LV dyssynchrony in mid-systole (with a prolonged electrosystolic delay) and normal synchronicity in late systole (absence of DLC). As part of the remaining questions with dyssynchrony, it is not yet known which phase of systole is the most important, ie, if dyssynchrony has the same consequences in any of the three phases of systole.

CONCLUSION

CRT is now considered a standard therapeutic strategy for refractory heart failure. Current patient selection criteria based on ECG criteria are insufficient to properly identify CRT responders, and echocardiographic assessment of dyssynchrony should be included in evaluation algorithms. From the various echocardiographic techniques developed, TDI has been the most extensively studied and validated to predict CRT responders. New techniques still under active evaluation, such as real-time three-dimensional echocardiography, could prove to be useful. The ongoing Predictors of Response to cardiac Resynchronization Therapy (PROSPECT) study (37), a multicentre study designed to compare the different echocardiographic dyssynchrony parameters in their abilities to predict response to CRT, will likely add important information. Until the results of further studies shed light on these issues, optimal echocardiographic evaluation of cardiac dyssynchrony should probably use an integrated multiparameter approach.

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