ity, in 50 patients followed up for 8 ± 5 months after CRT. An interesting subset of patients who did not have dyssynchrony by longitudinal TD velocities had a favorable response to CRT predicted by speckle-tracking radial dyssynchrony. These data suggest the additive value of assessing dyssynchrony from short-axis planes in addition to long-axis planes. A recent study of 176 patients combined longitudinal DTI velocity data with radial speckle-tracking strain data and found patients who had both positive longitudinal and radial dyssynchrony patterns had a high incidence of improvement in EF after CRT,[59] whereas patients with neither longitudinal nor radial dyssynchrony had a low incidence of EF improvement. Patients with heterogeneous patterns of dyssynchrony had intermediate responses. These data suggest that combining dyssynchrony data may be of additive value.[59]

THREE-DIMENSIONAL ECHOCARDIOGRAPHY

LV dyssynchrony in reality is a 3-dimensional phenomenon. Three-dimensional echocardiography provides a unique and powerful tool for the evaluation of LV dyssynchrony.[60] The advantage of real-time 3-dimensional echocardiography is that it allows for a comparison of synchrony between of the segments of the LV together in the same cardiac cycle (Figure 9). Regional wall-motion patterns can be visualized and quantified after segmentation of the LV chamber with semiautomatic contour tracing algorithms. Preliminary reports suggest that this approach enables a comprehensive analysis of LV wall motion before and during CRT with a direct comparison of endocardial wall motion between all LV segments. Kapetanakis et al calculated a systolic dyssynchrony index from the dispersion of time to minimum regional volume for all 16 LV segments and found this to be predictive of reverse remodeling after CRT in 26 patients.[61] This approach has the potential for a more comprehensive analysis of LV dyssynchrony.[62] However, disadvantages include lower spatial and temporal resolution, with frame rates for 3-dimensional wide-sector image acquisition at approximately 20 to 30 frames/s.

INTERVENTRICULAR DYSSYNCHRONY

Interventricular dyssynchrony, principally assessed as the interventricular mechanical delay (IVMD), is defined as the time difference between RV to LV ejection. This is determined as the time from the onset of the QRS to the onset of LV ejection versus RV ejection, usually measured as the onset of pulsed Doppler flow velocities in the LV and RV outflow tracts, respectively (Figure 10).[63-65] IVMD has been identified as a predictor of worsening symptom status and cardiac mortality in patients with heart failure, and has been shown to be of prognostic value in patients with CRT (usually >40-50 milliseconds).[65] Although IVMD is simple, reproducible, and possible with routine equipment,[15] it appears to be a nonspecific predictor of response to CRT. Bax et al demonstrated that IVMD was similar in 59 responders and 21 nonresponders to CRT: 47 ± 34 vs 49 ± 29 milliseconds, respectively (P = not significant).[16] Achilli et al reported results of the SCART study of 133 patients, where a positive response to CRT was predicted by IVMD longer than 44 milliseconds with a sensitivity of 66% and a specificity of 55%.[63] Richardson et al also showed that an IVMD longer than 50 milliseconds added prognostic information to patients undergoing CRT as part of the CARE-HF trial analysis.[65] The PROSPECT trial recently demonstrated that IVMD and other simple pulsed Doppler measures of dyssynchrony, such as the pre-ejection delay and the LV filling time to cardiac cycle length ratio, had the advantage of a high yield and high reproducibility in a multicenter setting.[18] However, most evidence suggests that interventricular dyssynchrony is not as useful in the prediction of response to CRT as LV intraventricular dyssynchrony, when a technically adequate study is possible. Comparisons of current principal measures of dyssynchrony appear in Table 2.

OTHER APPROACHES TO ASSESS DYSSYNCHRONY

Breithardt et al reported phase analysis using a semiautomatic method for endocardial border delineation.[66] The degree of LV dyssynchrony was quantified in 2D echocardiographic sequences from the apical 4-chamber view, focusing on the septal-lateral relationships. Computer-generated regional wall movement curves were compared by a mathematic phase analysis, based on Fourier transformation. The resulting septal-lateral phase angle difference is a quantitative measure for intraventricular dyssynchrony. Another method to determine dyssynchrony using conventional 2D echocardiography is velocity vector imaging. This method uses a series of unique B-mode pixel tracking algorithms to calculate regional myocardial velocities toward an operator-selected point of reference (Figure 11). A pilot study by Cannesson et al examined 23 patients with heart failure undergoing CRT using digital cineloops from standard apical views, with the user tracing the mid-LV wall from a single frame.[67] Dyssynchrony, defined as the greatest opposing wall peak longitudinal systolic velocity delay among the 3 views greater than or equal to 75 milliseconds, predicted EF response with 85% sensitivity and 80% specificity when patients were followed 8 ± 5 months after CRT.

EFFECTS ON LV REVERSE REMODELING AND MR

LV remodeling is a dynamic process characterized by progressive chamber dilatation, distortion of cavity shape, disruption of the mitral valve geometry with MR, and deterioration in contractile function that culminates in heart failure.[68,69] LV remodeling may be triggered by pressure or volume overload or loss of contracting myocytes from ischemic injury, or may be genetically programmed.[70] Although precise mechanisms and intracellular signaling pathways for LV remodeling are unknown, neurohormones and local trophic factors modulate the dynamic balance between distending forces that favor dilatation and the restraining forces imposed by the extracellular collagen matrix that may affect gene expression of myocyte function.[23] CRT often results in reverse remodeling where LV size and function progressively improve over time. This is a CRT-dependent, dynamic process where subsequent cessation of CRT results in progressive deterioration in LV function toward baseline values.[14] The extent of LV reverse remodeling varied according to cause of heart failure in the MIRACLE and other trials. Reduction in volume and severity of MR and the increase in EF were consistently 2- to 3-fold greater in nonischemic patients than in patients with ischemic heart failure in spite of significantly larger baseline volumes and lower EFs.[68] In an important study of 141 patients who received CRT, those who decreased LV end-systolic volume by at least 10% at 3 to 6 months had a more favorable long-term clinical outcome, including lower all-cause mortality (7% vs 31%), cardiovascular mortality (2% vs 24%), and heart failure events (12% vs 33%; all $P < .005$).[68,71]

CRT can reduce MR by improved temporal coordination of mechanical activation of the papillary muscles acutely and later

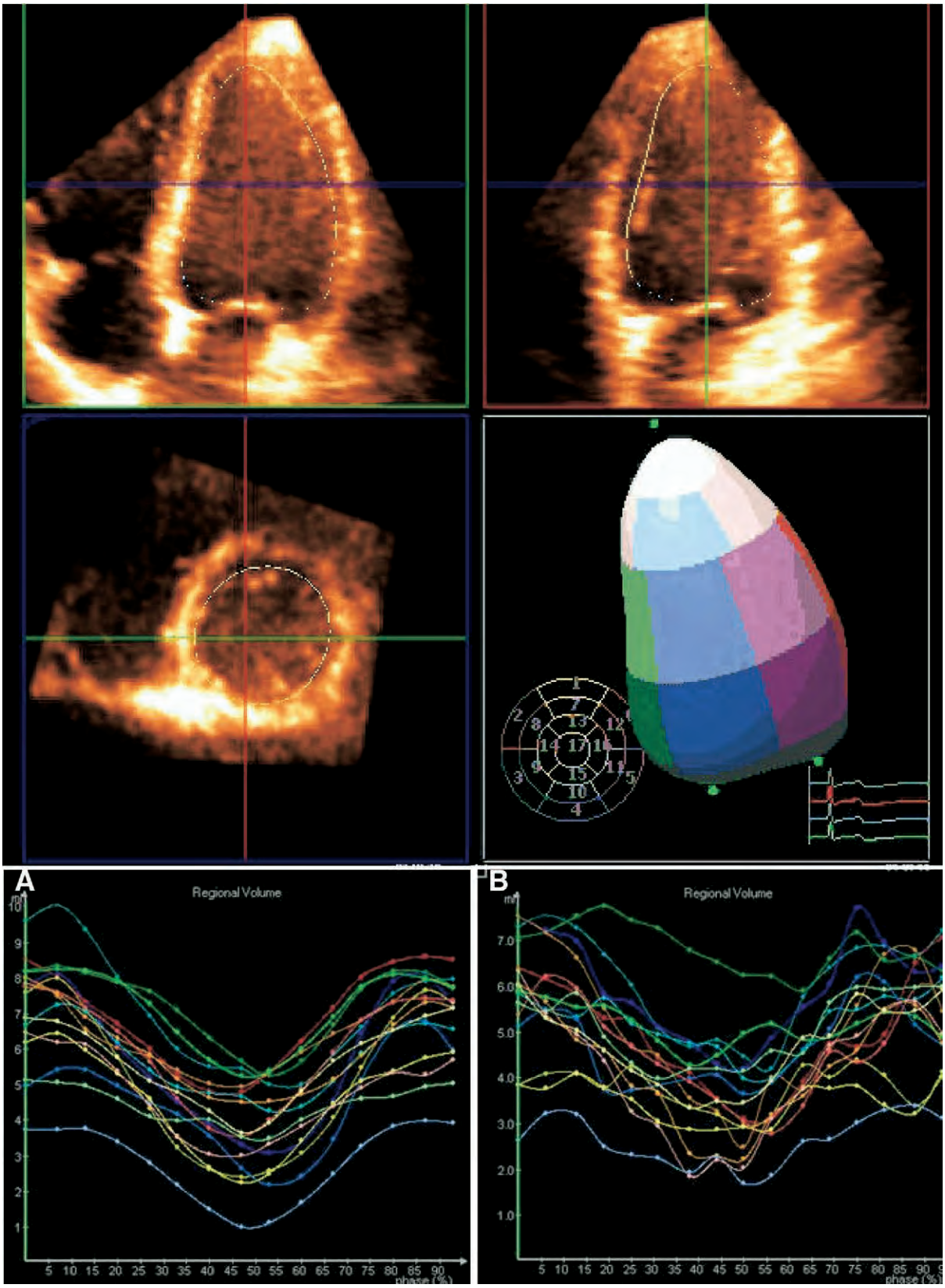

Figure 9 Three-dimensional echocardiographic assessment of segmental volume displacement in patient with normal synchrony (**A**) and with significant dyssynchrony (**B**).

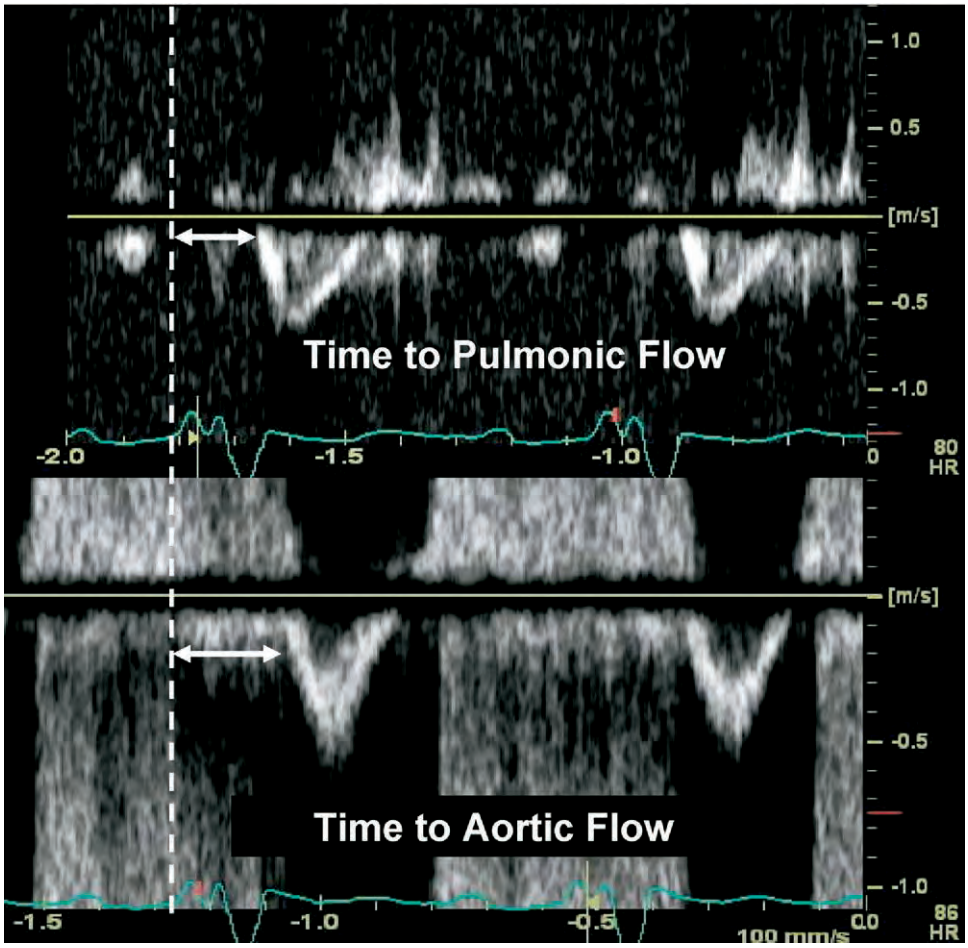

Figure 10 Pulsed Doppler from right ventricular outflow tract and left ventricular (LV) outflow tract demonstrating significant delay in LV ejection (>40 milliseconds).

improvements in LV size and geometry from reverse remodeling[28] (Figure 12). Breithardt et al used the proximal isovelocity surface area method during both pacing-off and CRT in the first week after CRT to report a significantly reduced regurgitant volume from 32 ± 19 to 19 ± 9 mL, and effective regurgitant orifice area from 25 ± 19 to 13 ± 8 mm^2, with CRT.[8] An important factor for acute reduction of MR after CRT appears to be improvement in the coordination of papillary muscle forces on the mitral chordae that increase the area of mitral leaflet coaptation. Kanzaki et al associated reductions in MR after CRT with acute improvements in the timing of mechanical activation of the papillary muscle sites, using mechanical strain activation mapping.[28]

PACING LEAD PLACEMENT

Several studies have suggested a potential role for echocardiographic Doppler methods to direct LV lead placement through identification of the anatomic site of latest mechanical activation. Ansalone et al were among the first to show that LV lead placement concordant with the site of latest velocity activation by TD was associated with a more favorable response to CRT.[30] They observed that the inferior or posterolateral wall was the location of latest mechanical activation in 75% of cases. Murphy et al demonstrated that the color-coded time-to-peak velocities approach described above could identify the site of latest systolic velocity and that lead placement at this site was associated with the greatest clinical and hemodynamic benefit of CRT.[72] There was a graduated response, such that LV pacing at a site one segment away from maximal delay was associated with a modest but more limited benefit, and patients paced at greater than one segment remote from the area of maximal delay had no significant reverse remodeling after a mean follow-up of more than 6 months. Suffoletto et al[54] utilized 2D speckle tracking to analyze LV radial strain to identify the site of latest mechanical activation before CRT and also observed that the patients with concordant LV lead placement had more favorable reverse remodeling. They found that 22 patients who had LV lead placement concordant with the site of latest activation had slightly greater improvements in LV EF (10 ± 5%), as compared with 24 patients who had discordant lead position (6 ± 5%; $P < .05$). Although these studies are encouraging, prospective studies will be needed to determine definitively the role of echocardiography in guiding LV lead placement.

RATIONALE FOR AV DELAY OPTIMIZATION

Because the ventricles are paced in CRT, the AV delay needs to be programmed. The optimal programmed AV delay for an electronic pacemaker has been defined as the AV delay that allows completion of the atrial contribution to diastolic filling resulting in most favorable

Table 2 Principal dyssynchrony indices associated with response to cardiac resynchronization therapy

Index	Method	Normal	Cutoff	Advantages	Disadvantages
Intraventricular longitudinal dyssynchrony					
Opposing wall delay, two sites[12,15,38]	Color tissue Doppler peak velocity (apical 4-chamber or long-axis views)	<50 ms	≥65 ms	Rapidly applied; offline analysis is possible	Requires color TD equipment; affected by passive motion tethering
Maximum wall delay, 12 sites[43,47]	Color tissue Doppler peak velocity (apical 4-chamber, 2-chamber, and long-axis views)	<90 ms	≥100 ms	More complete detection of longitudinal dyssynchrony; offline analysis is possible	Requires color TD equipment; affected by passive motion tethering
Yu index[14,31,43]	Color tissue Doppler, 12-segment SD (apical 4-chamber, 2-chamber, and long-axis views)	<30 ms	≥33 ms	More complete detection of longitudinal dyssynchrony; offline analysis is possible	Requires color TD equipment; more time-consuming; affected by passive motion tethering
Delay in onset of systolic velocity[51]	Pulsed tissue Doppler (apical 4-chamber, 2-chamber, and long-axis views; LV and RV)	<80 ms	≥100 ms	More widely available	Acquisition technically difficult; offline analysis is not possible; affected by passive motion tethering
Delayed longitudinal contraction[41,42]	Color tissue Doppler-strain-strain rate (apical views)	None	N/A	Less affected by passive motion or tethering; offline analysis is possible	Requires specialized color TD equipment; technically demanding
Intraventricular radial dyssynchrony					
Septal to posterior wall delay[34,35]	M-mode (parasternal mid-LV view)	<50 ms	≥130 ms	Widely available; rapidly applied; no advanced technical requirements	Largely affected by passive motion or tethering; difficulties with segmental akinesis
Septal to posterior wall delay[54,57]	Radial strain (parasternal mid-LV view)	<40 ms	≥130 ms	Less affected by passive motion or tethering; speckle tracking may be applied to routine images	Requires specialized instrumentation for analysis; assesses only radial dynamics
Interventricular dyssynchrony					
Interventricular mechanical delay[62-64]	Routine pulsed Doppler (RVOT and LVOT views)	<20 ms	≥40 ms	Widely available; no advanced technical requirements; highly reproducible	Nonspecific; affected by LV and RV function

LV, Left ventricular; *N/A*, not applicable; *OT*, outflow tract; *RV*, right ventricular; *TD*, tissue Doppler.

preload before ventricular contraction.[73] An AV delay programmed too short will result in absence or interruption of the atrial component (mitral A wave) by the premature ventricular contraction and closure of the mitral valve. An AV delay programmed too long can result in suboptimal LV preload or diastolic MR, or may even allow native LV conduction, which defeats the purpose of CRT.

Although the importance of AV synchrony is unquestioned, the need for routine echocardiographic Doppler AV timing optimization in all patients with CRT is controversial because an ideal approach has not yet been defined and there are often logistic challenges coordinating the echocardiography laboratory with electrophysiology technical staff for device programming. Auricchio et al concluded that although AV delay often positively impacts hemodynamics, LV resynchronization of intraventricular dyssynchrony is more important.[74] Many centers currently use empiric out-of-the-box AV delay device settings of approximately 100 to 130 milliseconds for CRT. Other centers rely on AV delay optimization algorithms based on ECG data to approximate the optimal AV delay optimal as [PR (ms) × 0.50], if QRS > 150 ms or [PR (ms) × 0.70], if QRS < 150 ms.[75] Sawhney et al recently conducted a prospective randomized trial of 40 patients comparing aortic Doppler optimized AV intervals to a fixed AV interval of 120 milliseconds after CRT.[76] AV optimized patients exhibited improved NYHA class and quality of life, but no significant improvement in 6-minute walk distance or EF at 3 months postimplant. A larger report of 215 patients undergoing Doppler-guided AV optimization found small differences between the baseline and post-AV optimization average AV delay (120 vs 135 milliseconds, respectively).[77] Furthermore, AV optimization enhanced LV hemodynamics in only a minority of patients with CRT, suggesting that a significant percentage of patients do not need to undergo formal AV optimization. Patients with intra-atrial conduction delay at baseline appeared to benefit greatest by prolonging the AV delays (150-250 milliseconds) during AV optimization (Figure 13).[77] These patients were identified by complete loss of the mitral inflow A wave

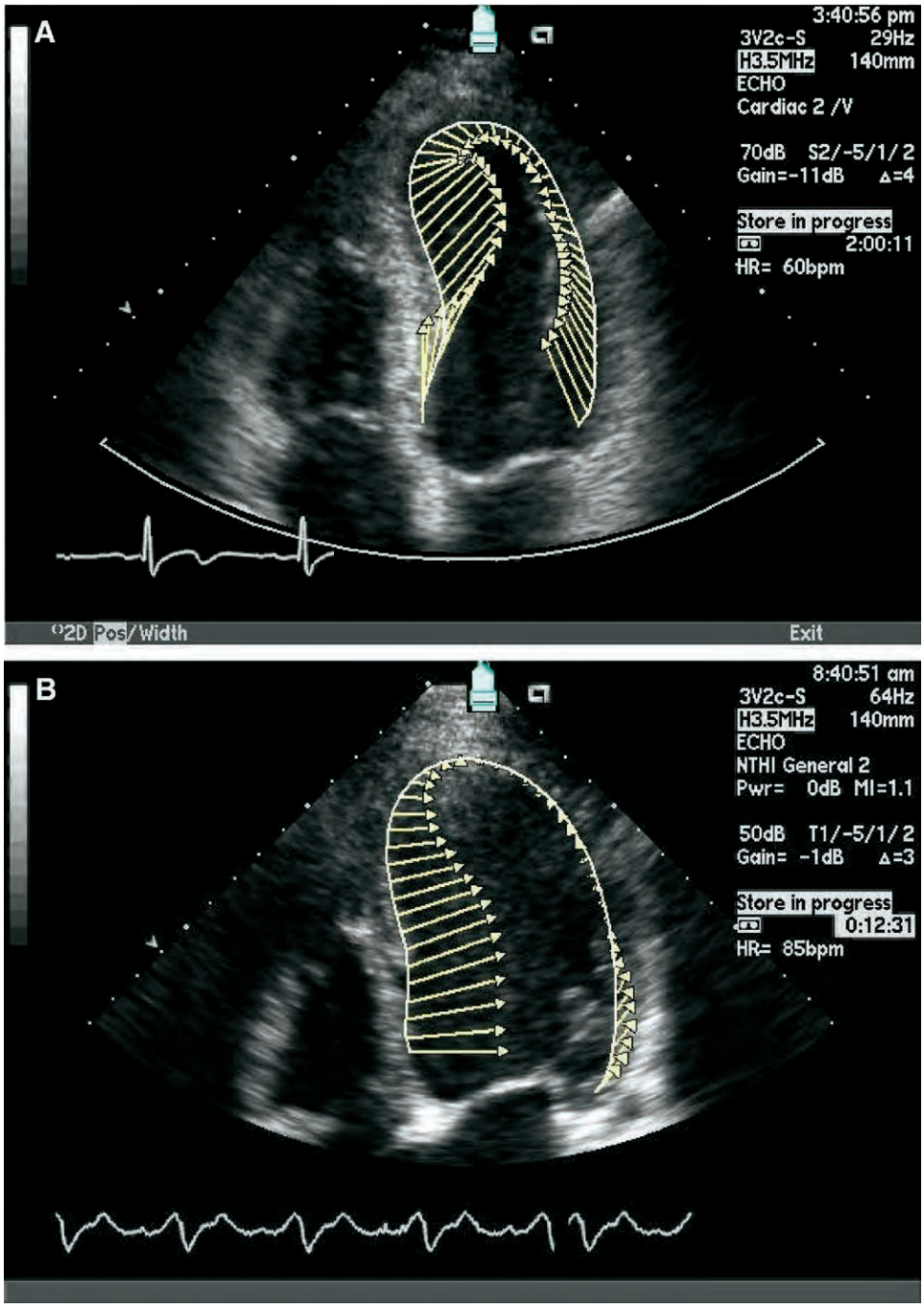

Figure 11 Velocity vector images demonstrating synchrony of velocity convergence toward center of left ventricle in healthy individual (**A**) and severe septal-lateral wall dyssynchrony in patient with heart failure and left bundle branch block (LBBB) referred for resynchronization therapy (**B**).

with an empiric setting that was too short. Although a recommendation for routine care has not been established, the following section provides guidelines for AV optimization after CRT.

RITTER AND ITERATIVE METHODS FOR AV DELAY OPTIMIZATION

Pulsed Doppler interrogation of mitral inflow to assess LV filling, and either pulsed Doppler or continuous wave Doppler sampling of the LV outflow tract to assess LV ejection, are utilized for the Ritter and iterative AV optimization protocols.[78,79] The method of Ritter et al attempted to optimally synchronize the termination of atrial contraction with the onset of ventricular systole.[79] This method requires programming the AV delay to a short (50 milliseconds) and then a long (200 or 250 milliseconds) interval while testing their impact on end-diastolic filling. The AV delay is then determined by correcting the long AV delay by the time shift from short and long Doppler tracings. The iterative method is simpler and begins by programming

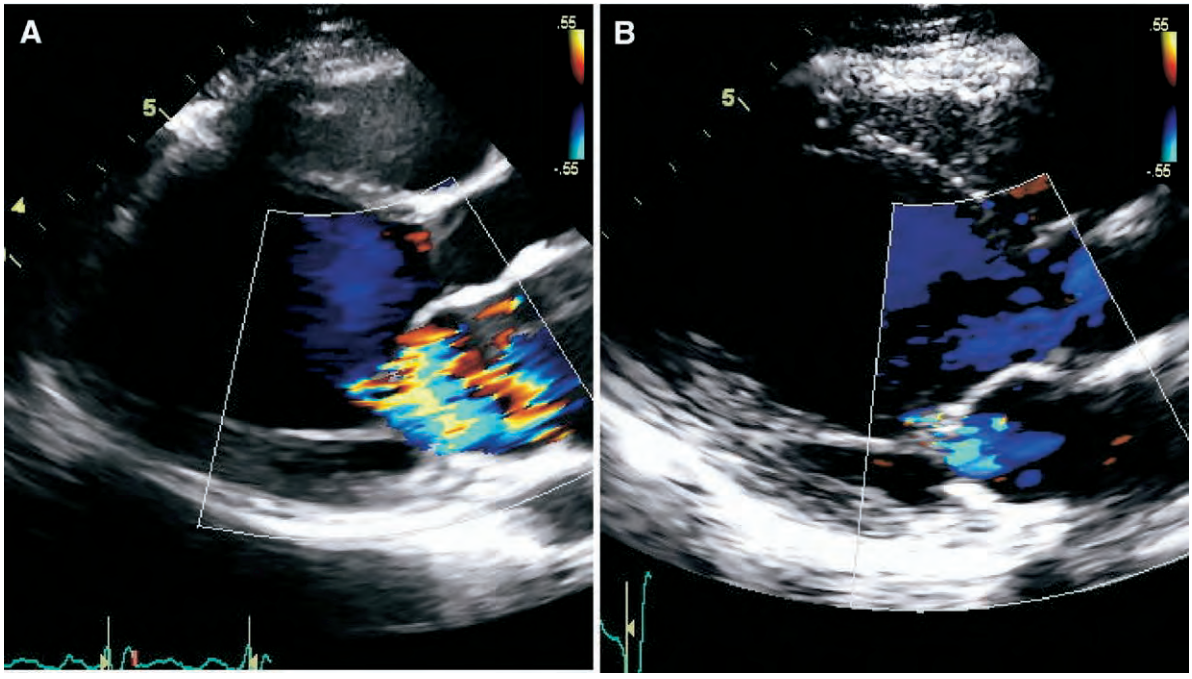

Figure 12 Parasternal long-axis view demonstrating reduction in mitral regurgitation in patient from before (**A**) to day after (**B**) resynchronization therapy.

the CRT device in atrial synchronous V pacing mode testing a series of AV intervals sequentially. This usually begins with an AV delay of 200 milliseconds, then decreases in increments of 20-millisecond intervals to a minimum AV delay as short as 60 milliseconds. The minimal AV delay that allows for adequate E and A wave separation and termination of the A wave at approximately 40 to 60 milliseconds before the onset of the QRS would be considered the optimal AV delay, and usually corresponds with a stage I diastolic filling pattern.[80] Technical features include positioning the pulsed wave sample volume deeper toward the left atrium (as opposed to the standard position at the mitral leaflet tips) to optimize detection of the mitral valve closure click, preparing settings of high sweep speeds and low filters, and inputting the ECG signal from the device directly to the ultrasound system, if possible. A variation on the iterative method for AV optimization uses transaortic Doppler velocities as a surrogate for stroke volume. The optimal sensed and paced AV delay is determined by the maximum aortic time-velocity integral value at 6 selected paced and sensed AV delays. A typical protocol will include measurements at AV delays of 60, 80, 100, 120, 140, and 160 milliseconds, with each paced and sensed AV delay setting separated by a rest period of at least 10 to 15 beats.

SIMPLIFIED DOPPLER SCREENING FOR AV OPTIMIZATION

A simplified Doppler screening protocol after CRT implantation is proposed using pulsed Doppler mitral inflow, because no consensus currently exists for the routine performance of AV optimization after CRT[77,81] (Figure 14).

Step 1: Optimize the ECG signal, including inverting the QRS complex if necessary.

Step 2: Optimize pulsed Doppler mitral inflow velocity using high sweep speeds, low filters, and the sample volume set at mitral annular level to determine closure clicks.

Step 3: Examine mitral inflow pattern. No AV optimization protocol is required if:

a. E and A waves are clearly identified and separated.
   and
b. Termination of the A wave occurs at least 40 milliseconds before QRS onset or mitral valve closure click.

Note that the mitral valve closure click should be aligned with the QRS complex as a surrogate for the beginning of LV systole. A pattern consistent with stage I diastolic filling (E wave lower than A wave) has not been shown to be improved on by AV alterations after CRT, and it is suggested by Kedia et al that formal AV optimization is not required in these patients.[77] AV optimization is recommended if any of the following are observed: A wave is not identified, E and A waves are merged, or A wave is truncated by mitral closure. AV optimization should be considered if stage II (pseudonormal) or stage III (restrictive) diastolic dysfunction is noted.[77,80] An absent A wave may be associated with intra-atrial conduction delay and often requires a longer AV pacing delay. On the other hand, E and A waves merge when the AV pacing delay is set too long. A truncated A wave requires lengthening of the AV delay. For these scenarios, either the iterative or Ritter methods described in detail above may be performed depending on the preference. Patients in atrial fibrillation or with frequent ventricular ectopy or tachycardia would not be appropriate candidates for AV optimization. Patients with mitral prosthetic valves may also be problematic.

BIVENTRICULAR (V-V) OPTIMIZATION

The recent generation of CRT devices allows for optimization of interventricular delays (V-V delays).[82-86] The first evidence of benefit from V-V optimization was reported by Sogaard et al.[87] The CRT settings were further optimized by V-V timing in 20 patients, resulting in an additional increase in LV EF (from 22 ± 6% at baseline to 30 ± 5% after CRT to 34 ± 6% after V-V optimization, $P < .01$). In

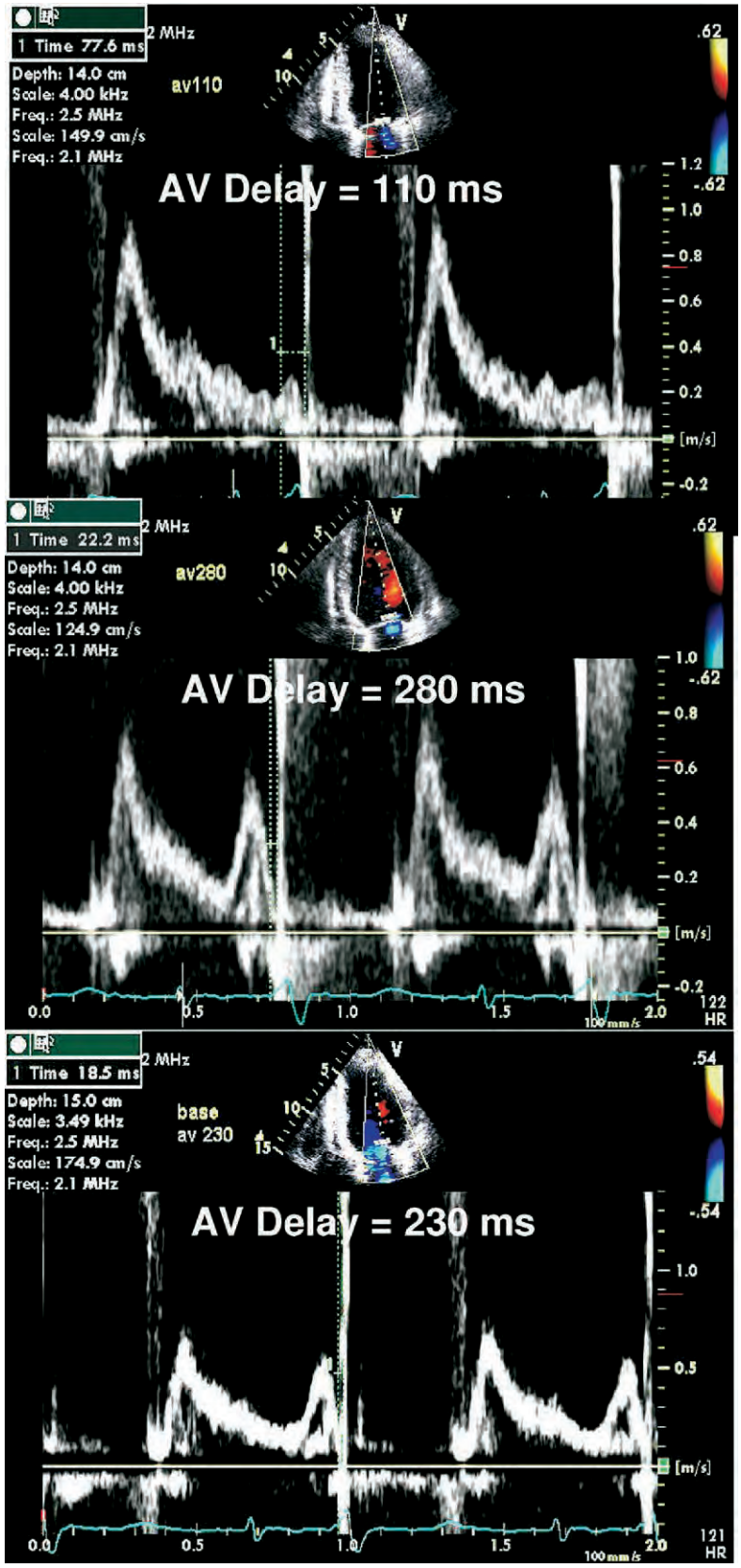

Figure 13 Atrioventricular optimization using mitral inflow velocities in patient with intra-atrial conduction delay. Default setting of 110 milliseconds resulted in loss of mitral inflow A wave (*top*). Delays of 280 milliseconds (*middle*) and 230 milliseconds (*bottom*) improved filling with contribution of atrial component. Alignment of mitral closure click with end of A wave was believed to be optimal with 230-millisecond delay.

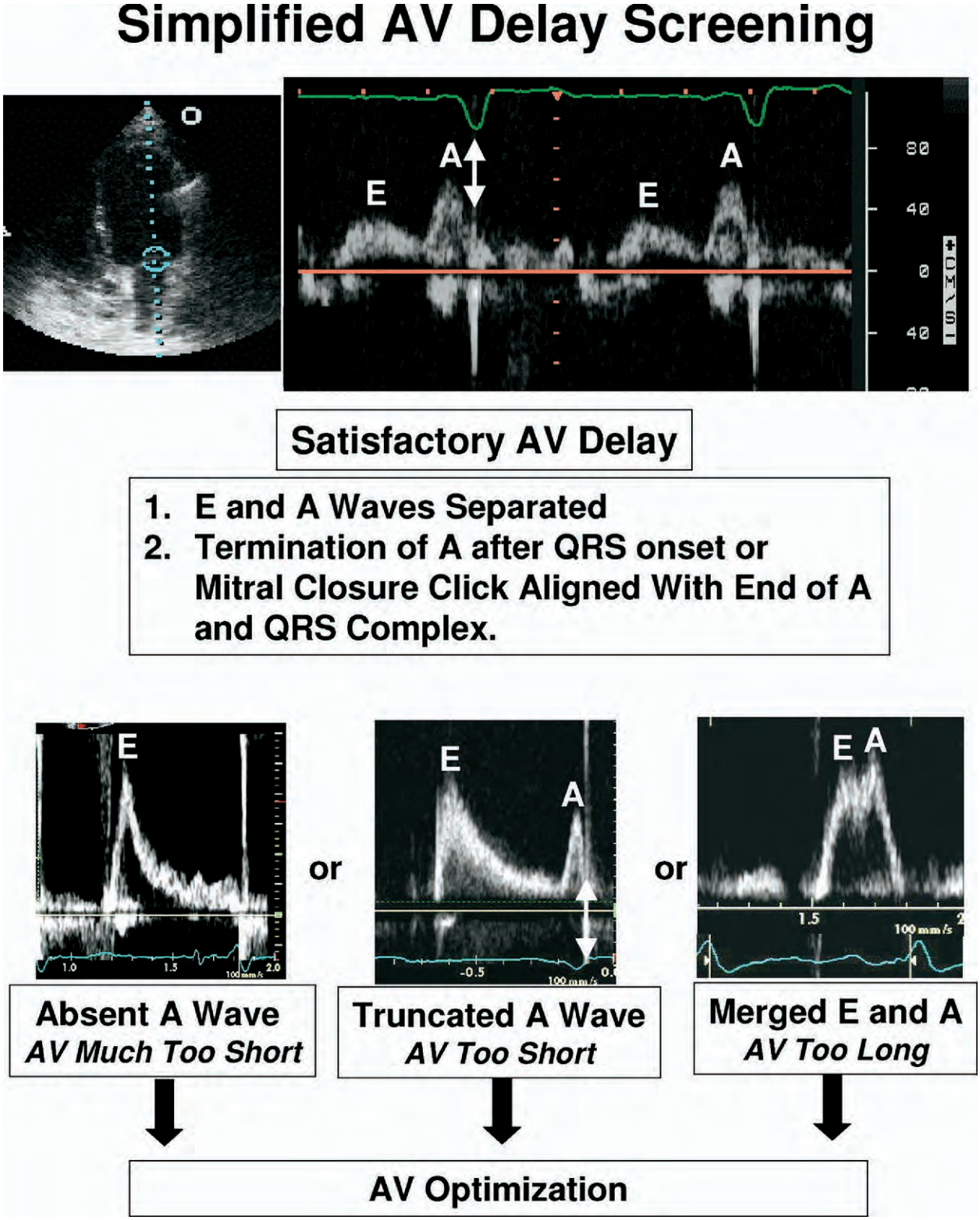

Figure 14 Simplified atrioventricular (AV) delay screening using mitral inflow Doppler velocities. Sample volume is placed within mitral valve to see closure click. AV optimization may not be necessary if E and A waves are separated, and termination of A wave is before QRS onset or mitral closure click aligned with end of A and QRS complex (usually type I diastolic dysfunction with E lower than A) (*top*). AV optimization is indicated if A wave is truncated, E and A waves are merged, or A wave is absent (*bottom*). Optimization may be considered if stage II (pseudonormal) or stage III (restrictive) diastolic filling patterns are present.

addition, Bordachar et al demonstrated that V-V optimization resulted in a significant reduction in MR.[88,89] V-V optimization is generally performed by changing the V-V sequence, starting with the LV being activated before the RV, and then stepwise lengthening or shortening of the V-V interval (eg, with intervals of 20 milliseconds) and measuring the highest aortic time-velocity integral. Current studies have shown that subsets of patients do acutely benefit from V-V optimization, but long-term benefit has yet to be determined.

DYSSYNCHRONY IN THE NARROW QRS PATIENT POPULATION

Mechanical dyssynchrony may exist in a subset of patients with heart failure who have narrow QRS duration (<120 milliseconds). If CRT can be shown to be of benefit to these patients, the application of echocardiographic assessment of dyssynchrony is potentially of great importance for patient selection for therapy.[20,21,64] Bleeker et al showed CRT to benefit 33 patients with NYHA class III/VI heart failure and EF less than or equal to 35%, but QRS less than 120 milliseconds, who had mechanical dyssynchrony defined as a septal-to-lateral wall time-to-peak systolic velocity delay of greater than or equal to 65 milliseconds by TD.[90] In a separate study, Yu et al reported results on 51 patients with heart failure with narrow QRS (<120 milliseconds) who had CRT based on TD measures of dyssynchrony. CRT resulted in significant reductions of LV end-systolic volume, and improvement of NYHA class, 6-minute hall-walk distance, and EF, similar to patients with wide QRS who underwent CRT.[91] The first randomized trial of CRT in patients with heart failure with narrow QRS complexes (<130 milliseconds), known as the RethinQ trial, was recently published by Beshai et al.[92] Dyssynchrony was defined as a TD septal-to-lateral wall cutoff of greater than or equal to 65 milliseconds from either apical 4-chamber views or apical long-axis views, or M-mode septal to posterior wall delay greater than or equal to 130 milliseconds. All patients who met inclusion criteria (96% by TD) had CRT devices implanted, and 172 were randomized to either CRT-off as a control or CRT-on. This trial failed to show a therapeutic effect of CRT on the primary end point of peak myocardial oxygen consumption. Although a positive effect of CRT was observed on the secondary end point of improvement in NYHA functional class, other parameters including quality-of-life score, 6-minute walk test, and LV reverse remodeling did not change. Benefit of CRT on 6-minute walk distance, however, was demonstrated in patients with nonischemic disease. A prespecified subgroup analysis of patients with borderline QRS duration between 120 and 130 milliseconds and dyssynchrony showed benefit of CRT by significantly improving their peak myocardial oxygen consumption and NYHA functional class.[92] In summary, the RethinQ randomized trial concluded as mostly negative, however, many unanswered questions remain. It is unclear whether the type or degree of dyssynchrony may be refined in this narrow QRS population to predict response to CRT, or whether other patient selection factors may impact results. Clearly, future larger randomized clinical trials are required to determine the role of CRT in patients with narrow QRS, and the potential pivotal role that echocardiographic Doppler will play in their selection for therapy.

APPLICATION OF DYSSYNCHRONY ANALYSIS IN CLINICAL PRACTICE AND REPORTING

Although a number of echocardiographic dyssynchrony methods discussed have suggested superiority to ECG QRS width for predicting response to CRT, evidence from large-scale clinical trials and current practice guidelines do not include an echocardiographic Doppler dyssynchrony study for patient selection.[13] **Accordingly, this writing group currently does not recommend that patients who meet accepted criteria for CRT should have therapy withheld because of results of an echocardiographic Doppler dyssynchrony study**.[13]

We acknowledge that many centers are currently applying these analyses as an adjunct to assist with clinical decision making for CRT for selected patients who may have borderline inclusion criteria, such as a borderline QRS duration. Although limited data are available from clinical trials, enrollment in the CARE-HF randomized CRT trial required patients with borderline QRS duration between 120 and 149 milliseconds to meet two of 3 additional criteria for dyssynchrony: an aortic pre-ejection delay longer than 140 milliseconds, an IVMD longer than 40 milliseconds, or delayed activation of the posterolateral LV wall.[6] In addition, the subgroup analysis of patients with QRS 120 to 129 milliseconds and evidence of mechanical dyssynchrony in RethinQ demonstrated benefit from CRT.[92] Other possible clinical settings where dyssynchrony analysis may potentially play a role is in patients with borderline EF or ambiguous clinical histories for NYHA functional class. If there is a clinical request for a dyssynchrony echocardiogram for these or other scenarios, it is the consensus of this group that it is reasonable for the following dyssynchrony measures to be performed and reported.

TD Opposing Wall Delay (the Maximum Time from S Wave Peak of One Wall to the S Wave Peak of the Opposing Wall) in Apical 4-chamber or Apical Long-axis Views

A cutoff of greater than or equal to 65 milliseconds is consistent with significant dyssynchrony, or Yu index (12-site SD) using longitudinal TD velocities from 3 standard apical views. A cutoff of greater than or equal to 33 milliseconds is consistent with significant dyssynchrony.

IVMD Using Pulsed Doppler from RV Outflow Tracts and LV Outflow Tracts

A cutoff of greater than or equal to 40 milliseconds is consistent with significant dyssynchrony.

Radial Dynamics, Which May be Additive Value, Include Septal-to-Posterior Wall Delay Using M-Mode in Patients With Non-Ischemic Disease With Technically High Quality Data, Or Using Speckle Tracking Radial Strain

A cutoff of greater than or equal to 130 milliseconds is consistent with significant dyssynchrony.

Other indices that appear in Table 2 may be included, if desired by individual laboratories. A conservative approach to carefully exclude mechanical dyssynchrony is advised, because an optimal approach has not yet been clearly defined. Agreement with more than one of these measures improves the confidence in the dyssynchrony analysis,[59] although a precise scheme to their collective additive value is currently unknown. **We advise that the dyssynchrony reporting should not include a recommendation whether a patient should undergo CRT, as this should be a clinical decision on a case-by-case basis for these borderline or**

challenging cases. Many other methods described in this report are promising, but may currently be too technically challenging or underdeveloped.

Echocardiography plays an exciting and evolving role in the care of the patient with CRT, from quantifying improvements in ventricular function and MR to optimizing the device after implantation. Although a great deal of work has been done to quantify mechanical dyssynchrony in hopes of refining patient selection and guiding lead placement, this is a complex and challenging field with future work needed and several promising studies ongoing. Technologic improvements in echocardiographic data acquisition and analysis as well as advances in our understanding of the pathophysiology of dyssynchrony and CRT have great potential to impact future clinical practice and improve patient outcome.

The Dyssynchrony Writing Group is indebted to Ashley Prather and the American Society of Echocardiography staff for administrative and organizational assistance.

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