A pilot study of systolic dyssynchrony index by real-time three-dimensional echocardiography predicting clinical outcomes to surgical ventricular reconstruction in patients with left ventricular aneurysm

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Abstract

OBJECTIVES: The aim of the study was to detect whether the systolic dyssynchrony index (SDI) assessed by real-time 3D echocardiography (RT3DE) could predict clinical outcomes of patients with ventricular aneurysm in response to surgical ventricular reconstruction (SVR).

METHODS: In total, 120 individuals underwent RT3DE, including 30 healthy volunteers and 90 patients with ventricular aneurysm. All patients underwent clinical and echocardiographic assessments at baseline and at 12 months after SVR. The SDI was defined as the SD of time to minimum systolic volume of the 16 left ventricular (LV) segments, expressed in percent RR duration. SVR responder was defined as a >15% decrease in LV end-systolic volume, reduction in NYHA functional class or 20% relative increase in the LV ejection fraction (LVEF).

RESULTS: The SDI was significantly higher in patients with aneurysm, at 14.3% compared with 2.0% in healthy volunteers (P <0.047). The SDI was negatively correlated with the LVEF. After SVR, 86 patients were responders. In this patient subgroup, the SDI exhibited an immediate significant decrease (to 7.7%; P <0.034) and a progressive decrease during 12 months of follow-up (to 4.9%; P <0.044). The SDI can discriminate SDI responders. Receiver-operating characteristic curve analysis yielded cut-off values of SDI 14.3% best associated with SVR response; area under the curve was 0.79 with reduction in NYHA class or 0.86 with increase in EF and 0.66 with decrease in the end-systolic volume.

CONCLUSIONS: RT3DE can be used to assess LV mechanical dyssynchrony in patients with aneurysm. SVR produces a mechanical intraventricular resynchronization and SDI can predict improvement following SVR.

Keywords: Echocardiography • Myocardial contraction • Asynchrony • Aneurysm • Cardiac surgical procedure

INTRODUCTION

In the setting of advanced heart failure (HF), cardiac resynchronization therapy is known to benefit most patients with HF. Left ventricular dyssynchrony (LVD) is a phenomenon extensively described in which loss of LV synchronous contraction is related to impaired LV systolic function and is a predictor of worse outcome [1]. The QRS duration is the parameter proposed by current guidelines to define LVD. However, the QRS width has been demonstrated to be a weak predictor of LVD in patients with left-sided cardiomyopathy [2] and, in parallel, a definition of dysynchrony based on LV mechanics has been proposed to better understand LVD; thus, accurate determination of mechanical dysynchrony has become increasingly important [3]. Currently, most echocardiographic methods for evaluating LV mechanical dysynchrony have limitations. Recently, 3D echocardiography has provided a unique imaging technique to assess the whole LV volume simultaneously. The simultaneous evaluation of multiple regional systolic changes of the LV volume and calculation of a global systolic dyssynchrony index (SDI) could provide more reliable information on global inhomogeneous contractile changes compared with conventional methods [4, 5].

Surgical ventricular reconstruction (SVR) has been proposed as a treatment option in HF patients with post-infarction aneurysm [6]. Although repair of LV aneurysm has been extensively studied, its mechanism in ventricular function remains unclear. Therefore, our aim was to: (i) evaluate the feasibility of RT3DE to assess the presence and distribution of LV mechanical dysynchrony in post-infarction aneurysm; (ii) detect changes in LV mechanical dysynchrony after ventricular rebuilding by SVR and determine whether SVR can produce a cardiac mechanical resynchronization; and (iii) assess the value of RT3DE-derived LVD for prediction of reverse LV remodelling after SVR.

MATERIALS AND METHODS

Study design and population

A series of 120 subjects were prospectively studied in two separate protocols. Protocol 1 was designed to address Aim 1, and Protocol 2 was designed to address Aims 2 and 3 listed previously. The study included 30 healthy volunteers, and 90 patients with post-infarction LV aneurysm. Healthy volunteers without evidence of...
structural heart disease and without known risk factors for coronary artery disease, matched for age, gender and body surface area, who underwent RT3DE, were included as a control group; patients selected were haemodynamically stable with post-infarction LV aneurysm of the apex or anterolateral wall containing no clot and having no significant mitral regurgitation. Those patients with post-infarction LV aneurysm were scheduled for SVR. All of them fulfilled the criteria for SVR: congestive HF, angina pectoris and malignant ventricular arrhythmia from the LV. Patients with associated valve lesions, a posterior myocardial infarction LV dilatation, diseases due to intracavitary clot and the presence of a bad acoustic window or atrial fibrillation were excluded. The study was approved by the local ethics committee, and all subjects gave their informed consent before enrolment.

Protocol. This study was a prospective study aimed at enrolment of patients with post-infarction anterior LV aneurysm who underwent anterioapical aneurysm plication without the use of cardiopulmonary bypass at our institution from January 2008 to April 2012. The technique was performed as previously described [7]. Patients were scheduled for regular clinical follow-up; these echocardiographic examinations are part of the routine, comprehensive assessment of patients presenting with aneurysm in our clinics.

Transthoracic real-time 3D echocardiography

RT3DE studies were performed with the iE33 ultrasound system with an S5-1 transducer (Philips Medical Systems, Andover, MA, USA) according to the guidelines. Three-dimensional datasets were acquired in the apical window with an ECG-gated acquisition as previously described [8]. Dedicated software incorporated in the system enabled the acquisition of four small real-time sub-volumes from alternate cardiac cycles. Subsequently, a pyramidal volume was obtained from the complete capture of the LV. The 3D studies were considered unsuitable for analysis if more than two segments could not be visualized or if they contained visible translation artefacts. All the measurements were analysed by two experienced investigators blinded to the aetiology and clinical status of patients, on an average of three or more cardiac cycles.

Left ventricular dyssynchrony data analysis

Analysis of RT3DE datasets was performed on a QLAB workstation using 3D-Advanced Quantification 6.0 (Philips Medical Systems) as described previously [9]. The 3D software displayed the volume-time curves for the 17 segments in which the LV volume was divided. To obtain the systolic dyssynchrony index (SDI), once the full volume of the LV was constructed, changes in segmental LV volumes along the entire cardiac cycle were analysed, based on the standard deviation of mean time-to-minimal-volume of 16 LV segments (excluding the true apex). The segments correspond to the LV 16-segment model as described by the American Society of Echocardiography. Finally, the SDI was defined as the SD of the time to reach the minimum systolic volume for 16 LV segments, and corrected for RR duration, expressed in percent cardiac cycle, thus displayed as a percentage to compensate for heart rate variability and to improve its reproducibility when repeated during follow-up. LVD is considered present if the SDI is >8% (mean ± 2 SDs of that in healthy controls) [9]. Therefore, with higher SDI, those patients identified more significantly LV mechanical dyssynchrony.

Changes in left ventricular mechanical dyssynchrony after surgical ventricular reconstruction

In the group of patients with aneurysm, serial changes in the SDI were evaluated before and after SVR and the study was repeated at the 12-month follow-up. Based on segmental time-volume curves, the same parameters were recalculated to assess the efficacy and extent of resynchronization. With the volume-time curves provided by 3D software, we could identify the more delayed LV segments attaining the minimum systolic volume. In addition, we could also find the most delayed segments characterizing the distribution of LV mechanical dyssynchrony by analysis of volume-time curves. In responders, the segment of maximal delay is resynchronized so that the previous segment of maximal delay becomes earlier and in harmony with other segments, including the previously earlier opposite segment. Figure 1 shows time-volume curve of one of the responders.

Clinical assessment

All patients were scheduled for regular clinical follow-up. According to the clinical protocol, before SVR, New York Heart Association (NYHA) functional class was evaluated. Furthermore, clinical evaluation included echocardiography to evaluate LV volumes, LV ejection fraction (LVEF) and LVD in all patients. In particular, 3D LVD was determined using RT3DE. At the 12-month follow-up, LV volumes, LVEF and LVD were reassessed using RT3DE.

Several clinical outcomes were taken as a positive response to SVR. A reduction in NYHA functional class by at least one class was considered a positive clinical response; a relative increase in the LVEF by 20% and reverse remodelling was defined as at least 15% reduction in the LV end-systolic volume on real-time 3D echocardiogram 12 months after SVR. All clinical data were prospectively entered into the cardiology information system.

Intra- and interobserver variabilities

Intraobserver variability was measured by analysis of 10 patients by the same observer at two different time points. Measurements of SDI were performed twice on separate days by the same observer for intraobserver variability assessment; additionally, a second blinded observer also performed the measurements for the interobserver variability. Interobserver variability was also measured by analysis of 10 random patients by two independent blinded observers. Inter- and intraobserver variabilities were calculated and expressed as a percent average value.

Statistical analysis

Continuous variables were expressed as mean (SD), whereas qualitative variables were expressed as number and percentage. A one-way analysis of variance test followed by post hoc Bonferroni test was used to compare LV volumes, EF and SDI among the study subgroups previously defined. Correlations were assessed with linear regression analyses. Outcomes were assessed with logistic regression to create receiver-operator characteristic curves (ROC) and calculate probability of response for each level of SDI. Inter- and intraobserver variability was examined using both
linear regression and Bland–Altman analysis. Variability was expressed as the mean difference of measurements as well as adjusted for the mean of each pair of measurements. A \( P \)-value less than 0.05 was considered significant. Statistical analysis was performed using SPSS 16 for Windows (SPSS, Inc., Chicago, IL, USA).

Figure 1: (A) Changes in LV mechanical dysynchrony from the 17-segment time–volume curves in patients with aneurysm. Before SVR, there was wide variation in the timing of regional minimal volume. (B) After SVR, the timing of regional minimal volume was lined up in order; nearly every segment reached the minimal and maximal systolic volume simultaneously, which showed a synchronous pattern of contraction. LV: left ventricular; SVR: surgical ventricular reconstruction.
RESULTS

Patients’ characteristics

Of the originally enrolled 120 patients, 2 died during the first year after SVR. The final study cohort included 118 patients. Eighty-six patients (95.6%) had ischaemic HF. Eighty-seven patients (96.7%) were in NYHA Class III, and 3 patients were in NYHA Class IV. Baseline clinical characteristics of the study population are presented in Table 1.

Left ventricular mechanical dyssynchrony assessed by real-time 3D echocardiography

Table 2 shows the LV parameters. Patients with aneurysm showed significantly larger LV volumes and lower EF compared with healthy volunteers (LV end-diastolic volume, 133 ± 54 vs 83 ± 15 ml, \( P = 0.043 \); LV end-systolic volume, 82 ± 46 vs 28 ± 6 ml, \( P = 0.031 \); LVEF, 36 ± 10 vs 66 ± 6%, \( P = 0.022 \)).

For normal subjects, with the 17-segment time–volume curves lined up in order, nearly every one of LV segments reached the minimal and maximal systolic volume simultaneously, which showed synchronous pattern of contraction. However, in patients with aneurysm, the 17-segment time–volume curves revealed a more globally hypokinetic LV with diffuse motion abnormalities and showed an asynchronous pattern, and the time to the minimal systolic volume of each segment was dispersed remarkably, with apical and mid-ventricular segments of the LV showing the greatest delays. In patients with aneurysm, the most delayed segments of the LV were more located at the apical and mid-segments of the LV, in concordance with the segmental wall motion abnormality (localized area of hypokinetic LV caused by myocardial infarction) (Fig. 1).

Regarding LV dyssynchronicity, SDI was significantly higher in patients with aneurysm, at 14.3% compared with 2.0% in healthy volunteers (\( P < 0.047 \)). We found a significant negative correlation between SDI and EF (\( r = -0.587, R^2 = 0.54, P < 0.042 \)), noting higher SDI values with lower EF. This is not surprising given that electromechanical asynchrony is recognized to be an important

Table 1: Clinical and echocardiographic patient characteristics (n = 120)

<table>
<thead>
<tr>
<th></th>
<th>Control group (n = 30)</th>
<th>Study group (n = 90)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>64 ± 10</td>
<td>65 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>Male gender (%)</td>
<td>21 (70%)</td>
<td>64 (71%)</td>
<td>0.53</td>
</tr>
<tr>
<td>Diabetes</td>
<td>-</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>NYHA functional class</td>
<td>-</td>
<td>3.0 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Family history of coronary artery disease*</td>
<td>-</td>
<td>49 (38%)</td>
<td>-</td>
</tr>
<tr>
<td>Hypercholesterolaemia b</td>
<td>-</td>
<td>44</td>
<td>-</td>
</tr>
<tr>
<td>Hypertension</td>
<td>-</td>
<td>57 (63%)</td>
<td>-</td>
</tr>
<tr>
<td>Current or previous smoking</td>
<td>-</td>
<td>76 (84%)</td>
<td>-</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>-</td>
<td>60 (67%)</td>
<td>-</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>1.96 ± 0.58</td>
<td>1.98 ± 0.22</td>
<td>0.76</td>
</tr>
<tr>
<td>Multivessel coronary artery disease</td>
<td>-</td>
<td>86 (95.7%)</td>
<td>-</td>
</tr>
</tbody>
</table>

Values are mean ± SD or n (%), data are expressed as mean ± SD, or number of subjects (percentage).

*Defined when close relatives had premature coronary artery disease (men >55 years old and women >65 years old).

Table 2: Comparison of LV volumes, systolic function and systolic synchronicity derived from time–volume curves in the two groups

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Study group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>75 ± 9</td>
<td>73 ± 8</td>
<td></td>
</tr>
<tr>
<td>LVEF (Simpson)%</td>
<td>68 ± 5</td>
<td>35 ± 10*</td>
<td>52 ± 7**</td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>83 ± 15</td>
<td>133 ± 54*</td>
<td>92 ± 36**</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>28 ± 6</td>
<td>82 ± 46*</td>
<td>41 ± 8**</td>
</tr>
<tr>
<td>LVEF (RT3DE)%</td>
<td>66 ± 6</td>
<td>36 ± 10*</td>
<td>58 ± 6**</td>
</tr>
<tr>
<td>LV basal rgEF</td>
<td>4.10 ± 0.34</td>
<td>2.70 ± 0.83</td>
<td>3.70 ± 0.83**</td>
</tr>
<tr>
<td>LV mid rgEF</td>
<td>3.90 ± 0.44</td>
<td>2.87 ± 1.46</td>
<td>3.87 ± 1.26**</td>
</tr>
<tr>
<td>LV apical rgEF</td>
<td>3.30 ± 0.53</td>
<td>1.56 ± 1.66</td>
<td>2.56 ± 1.36**</td>
</tr>
<tr>
<td>SDI</td>
<td>2.0 ± 0.7%</td>
<td>14.3 ± 0.4%*</td>
<td>4.9 ± 0.1%**</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

LVEF: left ventricular ejection fraction; EDV: end-diastolic volume; ESV: end-systolic volume; SDI: systolic dyssynchrony index; Preop: before operation; At discharge: 7 days after operation. *preop versus control group, **preop versus 12-month follow-up for \( P < 0.05 \).

\( r = -0.587, R^2 = 0.54, P < 0.042 \), noting higher SDI values with lower EF. This is not surprising given that electromechanical asynchrony is recognized to be an important
determinant of LV systolic dysfunction and a more depressed EF resulted in a higher SDI value, as given in Table 2.

**Effect of surgical ventricular reconstruction on systolic dyssynchrony index**

A series of global and regional LV volume curves were plotted, and acute changes in LV volumes, EF and SDI could be assessed by RT3DE. Global and regional diastolic volumes, systolic volumes and EFs, and regional stroke volume to global diastolic volume (rgEF) were compared, respectively, between before and after SVR. The mean follow-up was 12 months, and changes in these parameters could be assessed in 88 patients. After SVR, the global diastolic volume and systolic volume and the regional volumes were lower than preoperative values, \( P < 0.045 \), whereas the global and regional EFs and rgEF were improved \( P < 0.043 \), as given in Table 2.

At discharge after SVR for patients with aneurysm, RT3DE demonstrated cardiac resynchronization with a significant reduction of the LV end-systolic volume and a significant improvement in the EF and synchronicity of the LV. The SDI was significantly reduced postoperatively, compared with the preoperative value \( 14.3 ± 0.4 \) vs \( 7.7 ± 0.3\% \), \( P < 0.039 \). After surgical restoration, a significant improvement in systolic function was evidenced by an increase in the EF. At a mean follow-up of 12 months, the improvement observed in the LVEF with the concomitant reduction in the SDI was sustained in the studied patients, as given in Table 2.

**Impact of systolic dyssynchrony index on left ventricular function**

Table 2 shows the impact of SDI on LV function. At discharge, the mean LVEDV and LVESV were 101 ± 33 and 51 ± 13 ml, respectively. After a mean 12 months of follow-up of SVR, the LVEDV decreased significantly to 92 ± 36 ml \( P < 0.045 \) and the LVESV to 41 ± 8 ml \( P < 0.046 \). The mean LVEF increased from 48 ± 8% at discharge to 58 ± 6% after SVR \( P < 0.043 \).

**Dyssynchrony and clinical outcome**

Changes in NYHA functional classification, LV volumes, EF and SDI could be assessed in all patients. At a mean follow-up of 12
months, 75 (85.2%) patients were assessed as having obvious improvement with reduction in NYHA functional classification by at least one class, 76 patients (86.4%) showed a >20% relative increase in the LVEF and 69 patients showed LV reverse remodelling on real-time 3D echocardiogram 12 months after SVR. Figure 2 shows box plots for the SDI as predictors of response according to 3 parameters. Figure 3 shows the ROC calculated for each predictor. The SDI was significantly different during reduction in NYHA class responders and non-responders at baseline. At baseline, responder patients had an SDI value slightly higher than non-responder patients (SDI: 14.3 ± 0.4 vs 8.7 ± 7.1%, respectively; \( P < 0.0013 \)); this is reflected in the ROC curves, where the area under the curve (AUC) was 0.79 for SDI. Based on ROC curves, the optimal cut-off for the SDI in predicting response was 14.3%, which yielded a sensitivity of 90% and a specificity of 67% for separation of responders from non-responders with regard to reduction in NYHA functional classification by one class. Echocardiographic improvement with >20% relative increase in the LVEF was seen in 76 patients (13.6% non-responder rate), and the SDI at baseline between responder and non-responder groups was significantly different (10.5 ± 4.3 vs 14.5 ± 8.3%, respectively, \( P < 0.011 \)). In ROC analysis, the AUC was significantly higher for SDI at 0.86; the optimum SDI cut-off for this outcome was 14.8%, which has a sensitivity of 91% and specificity of 71% for separation of responders from non-responders with regard to a significant increase in LVEF after SVR. Those patients who showed a decrease in LV end-systolic volume >15% were considered responders to SVR. Remodelling was seen in 69 patients (21.6% non-responder rate), and the SDI at baseline was significantly different (11.8 ± 4.5 vs 14.2 ± 2.5%, respectively, \( P < 0.013 \)) between groups. In ROC analysis, the AUC was significantly higher for SDI at 0.66; the optimum SDI cut-off for this outcome was 14.2%, which has a sensitivity of 87% and specificity of 67% for separation of responders from non-responders of a significant reduction in LVESV after SVR.

Observer variability

With the RT3DE, intraobserver variabilities for end-diastolic volume, end-systolic volume and EF were 4.0, 6.1 and 4.7%, respectively, and interobserver variabilities were 5.9, 7.0 and 5.0%, respectively.

DISCUSSION

The present study demonstrates the feasibility of RT3DE with online software for quantitative assessment of LV mechanical dyssynchrony in patients with LV aneurysm and also shows that LV synchronicity is significantly impaired in patients with aneurysm. Moreover, we found that patients with aneurysm had a higher prevalence of intraventricular dyssynchrony compared with normal healthy volunteers, and the severity of this impairment was negatively related to LV systolic function. Simultaneously, chamber rebuilding by SVR reduced intraventricular dyssynchrony as the components of asynnergy changed. Mechanical efficiency improved as the SDI reduced, showing that more segments reached maximal motion at the end-systolic phase or mechanical resynchronization that improved LV systolic function, as evidenced in the increase in EF, and significantly improved the SDI both in the early and mid-term results of follow-up.

Assessment of left ventricular mechanical dyssynchrony by real-time 3D echocardiography

Recent advances in RT3DE offer the potential to evaluate the LVD by analysing the 17-segment time–volume curves, and several studies have demonstrated the usefulness of RT3DE to assess LV mechanical dyssynchrony in a broad spectrum of patients [10]. Kapetanakis [11] analysed LV synchrony by using a different 4D LV post-processing software in large patients with refractory congestive HF, and found a relation between SDI and LVEF and a more depressed EF resulted in a higher SDI value. Macron et al. [12] confirmed that RT3DE is a simple, reproducible and fast technique used to validate for the measurement of LVESV and LVEDV, stroke volume and EF, as well as to quantify LV intraventricular mechanical dyssynchrony, comparable with cardiac magnetic resonance; in particular, mechanical dyssynchrony can occur between 2 or more of the 16 myocardial regions described by the American Society of Echocardiography from base to apex. Although tissue Doppler imaging (TDI)-based techniques have been extensively used to assess dyssynchrony, they are prone to angle-related errors and translational cardiac and respiratory motion errors, which predominantly confined the analysis of LV function to the basal myocardial segments [13]. Conversely, using state-of-the-art technology, RT3DE offers excellent spatial resolution involving the entire left ventricle including the cardiac apex, allows accurate and highly reproducible LV volumetric quantification and, therefore, can be very useful for the assessment of electromechanical delay.

In the present study, RT3DE enabled quantitative evaluation of mechanical dyssynchrony with high temporal resolution and was more objective; the presence of a prior myocardial infarction worsened mechanical dyssynchrony in patients with aneurysm, a finding consistent with previous reports by Zhang et al. [14]. Our results are in line with these previous studies. In our study, for the first time, we investigated 3D-based dyssynchrony for prediction of reverse LV remodelling after SVR. Importantly, the present study adds more evidence of the clinical utility of RT3DE among patients with aneurysm who might benefit from LV mechanical dyssynchrony assessment and treatment with LV chamber rebulding by SVR.

Distribution of left ventricular mechanical dyssynchrony

Delgado et al. found that LV mechanical dyssynchrony had a different distribution and pattern according to the underlying cardiomyopathy [15]. In our study, we found that all the patients with aneurysm had systolic asynchrony among all the 16 segments, 12 segments and even the six basal ones and showed that the 17-segment time–volume curves in patients with aneurysm were arrayed disorderly, and the time to the minimal systolic volume of each segment dispersed remarkably; it obviously showed a less synchronous pattern of contraction than for the normal subjects. These results were also corroborated by Zeng et al. [16]. The results were in accord with the pathophysiological change of post-infarction aneurysm; because the myocyte loss, ventricular dilatation, deposition of extracellular matrix and interstitial fibrosis could lead to electrical–mechanical dyssynchrony, which would further impair LV work efficiency and deteriorate the haemodynamics, there must be some inner relation between HF and intraventricular dyssynchrony. Our study also reached a similar conclusion that a more depressed EF resulted in a higher SDI value, which implied that the severity of mechanical dysynchrony increased with worsening LV systolic function.
Study of left ventricular mechanical dyssynchrony changes induced by surgical ventricular reconstruction

In the patients with aneurysm who received SVR, LV mechanical dyssynchrony was evaluated prospectively by RT3DE. Data on the effect of SVR on LV mechanical dyssynchrony assessed with RT3DE are scarce. Before surgery, in patients with aneurysm, the 17-segment time-volume curves were arrayed disorderly, and the time to the minimal systolic volume of each segment was dispersed remarkably. The goal of SVR is to correct the size and geometry of the left ventricle, reduce wall tension and paradoxical movement, and improve systolic function. Regional differences improved after surgical intervention, because the diagnosis and criteria for aneurysmectomy were based on a dyskinetic aneurysm with systolic paradoxical movement. Surgical intervention reduces the the free wall of aneurysm; after aneurysmectomy, almost all regions of the left ventricle developed more uniform endocardial motion that reach its maximum extent at the end-systolic phase; such uniformity contributed to ejection without wasting energy, thereby defining cardiac mechanical resynchronization, as shown by the decreased SDI. These results are in line with the findings of the present study. An acute improvement in LV systolic function and a reduction in LV volumes were observed together with a decrease in the SDI. In those patients who completed the 12-month follow-up, the beneficial effects of SVR on LV performance and synchrony were sustained, and the 17-segment time-volume curves in postoperative patients were lined up in order, which showed a synchronous pattern of contraction.

Dyssynchrony and surgical ventricular reconstruction outcome

LV contractility is a complex three dimensional issue as a net result of longitudinal, radial and circumferential LV contraction [17]. RT3DE analysis of LV volumes in this study enabled simultaneous assessment of time to minimum volumes from all myocardial segments, which is the SDI. The SDI was defined as the SD of time to minimum systolic volume of the 16 LV segments. Thus, it enabled calculation of the temporal difference in maximum volumetric changes at one moment [17].

In our study, responders and non-responders to SVR were related to RT3DE-derived dyssynchrony. Fig. 2 shows scatter plots for the SDI as a predictor of response according to three parameters: a reduction in NYHA functional class by at least one class, a relative increase in the LVEF by 20% and reverse remodelling defined as at least 15% reduction in the LV end-systolic volume on real-time 3D echocardiogram 12 months after SVR. In Fig. 3, the ROC has been calculated for each predictor. Based on ROC curves, the SDI was highly predictive of any of the clinical outcomes (AUC: 0.79, 0.86 and 0.66 for NYHA functional class, LVEF and LV end-diastolic volume, respectively).

Study limitations

Our study presents several limitations. First, the number of patients enrolled was small. Second, we did not compare the systolic dyssynchrony derived from RT3DE with other generally accepted tools such as TDI [13, 18], further studies are needed for accurate validation of LVD parameters obtained from RT3DE and to clarify the correlation of systolic dyssynchrony derived by different methods. Third, as for the RT3DE, the post-processing analysis depends on clear image quality. RT3DE still has limited applicability in patients with difficult acoustic windows. In addition, being a single-centre study, there is a necessity for further research to confirm our findings.

CONCLUSIONS

In conclusion, RT3DE is a feasible echocardiographic method that can be used to assess and quantify accurately and effectively LV mechanical dyssynchrony. SVR improves LV systolic function through cardiac resynchronization of LV contraction caused by exclusion of scarred and thinned myocardium; this resynchronization reduces wasted energy and improves systolic function. The SDI can be a predictor of improvement clinical outcomes following SVR.

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REFERENCES

eComment. Re: A pilot study of systolic dyssynchrony index by real-time three-dimensional echocardiography predicting clinical outcomes to surgical ventricular reconstruction in patients with left ventricular aneurysm

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This study was designed to determine if the systolic dyssynchrony index (SDI) performed by 3-D echocardiography (RT3DE) is capable of identifying patients with LV aneurysms who will respond favourably to surgical ventricular reconstruction (SVR). “Responders” are defined as those patients who demonstrate improved postoperative changes in three measures of LV systolic function: LV end-systolic volume, LV ejection fraction, and the clinical NYHA Class of heart failure [1]. SDI is a well-established measure of LV systolic function and determining the preoperative SDI using RT3DE to predict who will respond to SVR might theoretically be helpful to the clinician. A potential advantage of 3-dimensional analysis is that more information will be obtained than can be attained by simple 2-dimensional ejection fraction. However, the value of that additional information is likely to be minimal in relation to the extra costs incurred with RT3DE. In addition, one of the parameters that the authors propose as a positive response is a “relative increase” in the LV ejection fraction of 20%. Expressing data in terms of “percent increase” rather than in absolute values is a classic way to either hide data or to make positive changes look much better than they actually are. For example, a patient with a 20% preoperative EF who increases to a 24% postoperative EF would have a “20% relative increase” in his/her EF. However, such an “increase” in LV EF would be essentially meaningless clinically. Another “index” of positivity in this study is a change in the NYHA classification for heart failure, which is a subjective, not objective, measure of LV function. The remaining “index” of positivity is the LV end-systolic volume. Since the patient’s end-diastolic volume will be decreased automatically by the resection of the aneurysm, the end-systolic volume will also almost certainly be decreased. Otherwise, the SVR procedure would result in a decrease in the LV ejection fraction (a calculated number), which it does not ordinarily do in surviving patients [2]. Thus, this entire study is set up to succeed regardless of whether the SDI using RT3DE is helpful or not.

In multiple studies reports by Dor, Menicanti, Buckberg and others have already demonstrated that SVR virtually always improves LV systolic function in patients with LV aneurysms because of the effects of La Place’s Law, so any potentially added benefit from identifying likely “responders” in this group using RT3DE-generated SDIs is extremely limited [3].

While the use of preoperative SDI to predict the LV aneurysm responders to SVR is likely to be of very limited value, this surgical procedure, SVR, is still accompanied by a significant operative mortality rate of 3-5%. The preoperative SDI using RT3DE could theoretically identify those few patients who are likely to actually die from surgery due to the inadequacy of their remaining myocardium. The prediction of “responders” and the prediction of operative mortality are two entirely separate issues. The only thing that SDI would do in this study is to predict which of the LV aneurysm survivors will respond favourably to SVR.

Conflict of interest: none declared.

References