Apical transverse motion as surrogate parameter to determine regional left ventricular function inhomogeneities: a new, integrative approach to left ventricular asynchrony assessment

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Aims
Left ventricular (LV) asynchrony assessment is mostly based on delays between regional myocardial velocity peaks. Regional function is barely considered. We propose apical transverse motion (ATM) as a new parameter integrating both temporal and functional information, which was tested in different conduction delays.

Methods and results
We examined 67 patients, 11 patients with post-infarct ischaemic left bundle branch blocks (iLBBB) and 25 patients with non-ischaemic left bundle branch block (nLBBB), 12 patients with right bundle branch block (RBBB), and 19 normal healthy volunteers (NORM). Longitudinal colour tissue Doppler data were used to calculate the total transverse apex motion (ATM), the transverse motion in the four-chamber view plane alone (ATM4CV) as well as regional myocardial deformation and conventional LV asynchrony parameters. Median ATM was 1.8 mm in NORM, 1.5 mm in RBBB (P = 0.999), 1.2 mm in iLBBB (P = 0.183), and 4.3 mm in nLBBB (P < 0.001 vs. NORM and RSB). ATM4CV behaved similarly, showed a good correlation with regional deformation data, and distinguished well between NORM and LBBB (AUC = 0.87).

Conclusion
Apical transverse motion is a new and simple parameter integrating information on both regional and temporal function inhomogeneities of the LV. It has a potential role in assessing LV asynchrony in the clinical context.

Keywords
Myocardial function • Dyssynchrony • Conduction delay

Introduction
Regional mechanical left ventricular (LV) dysynchrony plays an important role in the aetiology of LV dysfunction and heart failure. Both regional myocardial dysfunction, as it occurs e.g. in infarcted myocardium, and myocardial activation inhomogeneities, as e.g. in left bundle branch blocks (LBBB), will result in an inhomogeneous contraction of the LV myocardium with unfavourable re-distribution of loading and subsequent inhomogeneous regional remodelling.1 This remodelling leads to further functional deterioration of early or less contracting regions and causes inefficient LV performance.

With the introduction of cardiac resynchronization therapy (CRT) as a therapeutic option to re-synchronize the contraction of LV regions, the correct characterization of regional function inhomogeneities has become a new task of non-invasive imaging. Owing to its wide availability and potential to image and quantify regional function with good spatial and excellent temporal resolution, echocardiography is usually the method of choice. Two

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questions have to be answered in asynchrony analysis: (i) is there a
temporal inhomogeneity in regional myocardial contraction? and
(ii) is there a regional inhomogeneity in residual myocardial func-
tion? The first question is usually easy to answer, whereas answerv-
ing the second is difficult because of the marked interaction of
myocardial regions during the cardiac cycle.

In the past, several proposals have been made to approach LV
asynchrony. Most suggestions were based on the analysis of the
occurrence of myocardial velocity peaks measured by Tissue
Doppler. This approach allows to assess timing inhomogeneities
within the LV. Owing to interaction between regions (tethering), a reliable analysis of the true regional function is difficult.
Accordingly, more recent reports have dampened the enthusiasm
about the clinical value of these parameters. Other authors ana-
lysed regional motion with 3D echo or looked at deformation. Oth-
erwise authors analysed regional motion with 3D echo or looked at deformation. Oth-
erwise authors analysed regional motion with 3D echo or looked at deformation. Oth-
erwise authors analysed regional motion with 3D echo or looked at deformation. Oth-

Herewith, we propose a new, integrative, and thus, simple par-

Methods

Study population

The study population comprised 67 subjects, which is comparable to
previously published studies in the field of echocardiographic asyn-
chrony assessment.

A group of 19 normal healthy volunteers (NORM) without any
history of cardiovascular disease, without abnormal findings during
physical examination, with normal ECG at rest and during bicycle
stress, and normal results of a standard echocardiogram was studied.
Furthermore, 25 patients with non-ischaeamic LBBB (nLBBB) were
included. A coronary angiogram ruled out coronary artery disease in
all of them. Eleven patients with chronic ischaemic LBBB (iLBBB)
were also studied. All had documented myocardial infarction at
least 1 year prior to the study examination. A current relevant
coronary artery stenosis was ruled out by non-invasive stress
testing or coronary angiography. Finally, we included 12 patients
with right bundle branch block (RBBB). In those, ischaemia inducing
coronary artery disease was excluded by non-invasive testing or
coronary angiography.

No patient had more than mild valve disease or more than mild pul-
monary hypertension. All patients were in sinus rhythm. None had
ventricular arrhythmias exceeding occasional premature beats. Patients
were included irrespective of their LV ejection fraction (EF). No
patient was paced. Characteristics of the groups are summarized in
Table 1. All study participants gave informed consent prior to inclusion.

Selection of study subjects

Volunteers were recruited from a group of hobby speleologists compris-
ing healthy adults in good physical shape in a wide age range. Patients
were recruited via the ECG service of the Medical Clinic II in Erlangen.
Patients with an QRS width of >130 ms were transferred to the echo
lab for a screening echo to evaluate image quality and to exclude major
valve disease. We initially aimed at 60 participants. When this number
was reached, we focused on selective inclusion of additional nLBBB and
RBBB patients who were underrepresented. Inclusion was stopped,
when all groups comprised >10 subjects.

Echocardiography

All patients were scanned after 5 min of rest in a left lateral decubitus
position. We used a Vivid 7 ultrasound scanner (GE Vingmed, Horton,
Norway) to perform a standard echocardiogram and to acquire grey
scale and colour Doppler myocardial imaging (DMI) data from an
apical two-, three-, and four-chamber view. For DMI data acquisition,
care was taken to maximize frame rate by reducing depth and
sector angle (typical frame rate: 100 fps). Blood pool Doppler traces
of all valves were used to determine valve openings and closures. All
acquired image loops contained at least three cardiac cycles. Data
were digitally stored for later off-line analysis.

Post-processing and measurements

As a first step, we used an EchoPac workstation (GE Vingmed) to
review the data and to measure cardiac diameters from a parasternal
long-axis view. Ejection fraction was determined according to the
bi-plane Simpson’s method from apical images. Regional wall motion
was scored as normo-, hypo-, a-, and dyskinetic using a modified 18
segment model.

Secondly, we extracted myocardial velocity and strain rate traces
from the lower half of the apical, medial, and basal segments as well
as the mitral ring of each myocardial wall (septal, anteroseptal,
anterior, lateral, posterior, inferior). Thus, the same 18-segment
model was used for Doppler data analysis. The position of the
regions of interest (ROI) was manually adjusted during the cardiac

<table>
<thead>
<tr>
<th>n</th>
<th>age (years)</th>
<th>EF (%)</th>
<th>Signa (P-value)</th>
<th>QRS width (ms)</th>
<th>Signa (P-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORM</td>
<td>19</td>
<td>35 ± 12</td>
<td>62 ± 6</td>
<td>98 ± 9</td>
<td></td>
</tr>
<tr>
<td>RBBB</td>
<td>12</td>
<td>63 ± 13</td>
<td>63 ± 6</td>
<td>0.989</td>
<td>150 ± 13</td>
</tr>
<tr>
<td>nLBBB</td>
<td>25</td>
<td>63 ± 13</td>
<td>23 ± 11</td>
<td>&lt; 0.001</td>
<td>167 ± 19</td>
</tr>
<tr>
<td>iLBBB</td>
<td>11</td>
<td>69 ± 8</td>
<td>23 ± 17</td>
<td>&lt; 0.001</td>
<td>155 ± 19</td>
</tr>
</tbody>
</table>

*vs. NORM. Ejection fraction (EF) was not significantly different between the two LBBB groups. QRS width was not significantly different between the three patients groups.
In a third step, traces were imported in a dedicated, Matlab (the Math Works Inc., Natick, MA, USA) based analysis software (TVA version 14.3, JU Voigt, University Leuven, Belgium), which allowed to calculate and baseline-correct motion and strain traces and to calculate ATM. From segmental velocity curves, we extracted amplitude and timing of the systolic ($v_{sys}$, $t_{sys}$) peak. From strain curves, we determined the timing of the onset ($t_{onset}$) and end of systolic shortening ($t_{end}$) as well as the amplitude of shortening during ejection time ($e_{et}$) and deformation is calculated as $e_{tot} = e_5 - e_2$ and $e_{et} = e_4 - e_3$. 

**Figure 1** Longitudinal velocity and strain curve examples from a lateral wall region of interest with typical position of measurement points: (0) onset of QRS, (1) peak systolic velocity, (2) onset of shortening, curve maximum, (3 and 4) strain at the beginning and end of ejection, (5) maximal shortening, curve minimum. AVO, AVC, MVO, MVC—opening and closure of aortic and mitral valve as derived from spectral Doppler. Time-to-peak systole is calculated as $t_{sys} = t_1 - t_0$; time to onset of shortening as $t_{onset} = t_2 - t_0$; time to end of shortening as $t_{end} = t_5 - t_0$. Deformation is calculated as $e_{tot} = e_5 - e_2$ and $e_{et} = e_4 - e_3$.

**Figure 2** Data acquisition and ATM calculation, exemplified for the two-chamber view. Data from apical long axis and two-chamber views are processed likewise. (A) Assuming that the transverse apex motion in the four-chamber view is the average of the longitudinal apical septal and the inverted apical lateral motion, longitudinal velocity traces are obtained in the two apical segments by tissue Doppler. (B) For ATM calculation, velocity traces are integrated to displacement traces. After inversion of the lateral, curves can be averaged to obtain the ATM in the plane of the four-chamber view. The numbers in the ATM curve indicate the measurement positions to measure the total amplitude of ATM (Point 4 – Point 1) and ATM during ejection time (ET) (Point 3 – Point 2). The example shows a typical LBBB pattern with early short septal motion of the apex during isovolumic contraction time and lateral motion during the entire ejection time.
during the entire cardiac cycle ($e_{\text{mm}}$) (Figure 1). Flow Doppler traces from the mitral and aortic valve were used to determine the time intervals of the cardiac cycle. Velocities are expressed in cm/s, motion in mm, strain rate in 1/s, and strain in %. The ECG served as common time base for all timing measurements with the beginning of QRS as reference. Timing data are expressed in ms or in per cent of the cardiac cycle.

**Calculation of apical transverse motion**

A direct measurement of ATM is not possible with DMI, because the motion occurs perpendicular to the ultrasound beam. We therefore assumed the apex to behave as a 'cap' with homogeneous material properties where the different walls are pulling on. We used the longitudinal velocity traces of the apical segments to estimate the velocities of the 'cap rim' (Figure 2A). By integrating the velocity traces, motion was obtained. Curves from two opposite walls were averaged after inverting the anterior, lateral, or anteroseptal curve, respectively, in order to obtain transverse motion within each of the three apical scan planes (Figure 2B). Apical transverse motion perpendicular to the LV long axis was then reconstructed for the entire cardiac cycle assuming the scan planes of the three apical views to intersect at a 60° angle (Figure 3). Apical transverse motion was quantified by determining direction and amplitude of motion during the cardiac cycle and during systole. Data acquisition (J.U.V.) and analysis (T.M.S., M.S., E.G.) were done by different persons. Patient data were not available during analysis.

**Conventional parameters of ventricular asynchrony**

Conventional parameters of inter- and intraventricular asynchrony were calculated according to the original publications. We obtained the delay in onset of aortic and pulmonary outflow (interventricular delay (IVD)\(^{10,11}\)), the standard deviation of time-to-peak systolic velocity in the 12 mid- and basal segments (TsSD\(_{12}^{2}\)), the maximal dispersion in time-to-peak systolic velocity in basal segments of the two- or four-chamber view (SysDisp\(_{3}^{3}\)), and in septal and lateral basal segments only (SLDisp\(_{5}^{5}\)).

**Statistical analysis**

Segmental timing, motion, and deformation data were grouped and averaged per patient prior to any statistical analyses to account for the clustered nature of these data. Data were presented as mean ± SD or median and inter-quartile range, as appropriate. Comparisons between groups were done by means of a t-test or Mann–Whitney U-test. For multiple groups, comparisons were made using analysis of variance or a Kruskal–Wallis test, with post hoc pairwise comparisons between the groups whereby adjustments to the significance level were made using the Tukey–Kramer method or Dunn’s multiple comparison test, respectively. For the comparison of paired samples, a paired-test or Wilcoxon signed-rank test was used. Correlations between asynchrony parameters and regional function data were described by Pearson correlation coefficients. Receiver-operating-characteristics (ROC) curves and their area under the curve were calculated to assess the discriminative power of parameters.
AUC values were compared using the method offered by MedCalc. To determine the reproducibility of velocity, strain, and ATM estimations, 21 additional patients with different heart diseases were examined by two experienced observers at the beginning or the end of a regular echo examination. The agreement of measurements from both acquisitions was assessed according to the methods described by Bland and Altman. All statistical tests were two-sided and assessed at the 5% significance level. Owing to the exploratory nature of the study, no further adjustments were made to the significance level. We used the programs InStat 3 and Prism 4 (GraphPad Software Inc., La Jolla, USA) and MedCalc (MedCalc Software, Mariakerke, Belgium) for statistical calculations.

Results

Study population

Characteristics of the study population are given in Table 1. All patient groups had significantly wider QRS than NORM. Both LBBB groups had significantly reduced global LV function. Ischaemic LBBB patients had on average 3.5 infarcted segments. Thirty-eight per cent of the infarcted segments were localized in septal, anteroseptal, and anterior walls, 62% in lateral, posterior, and inferior regions. Thirty-six per cent of the infarcted segments were located in the apex, 33% in the mid, and 31% in the basal LV. Infarct scars were hypo- or akinetic, no dyskinesia was found. Average wall motion score was 1.35.

Feasibility and reproducibility

From 1608 possible segmental and annular velocity traces, 1568 were recorded. Of those, 38 traces could not be analysed (97% feasibility). From 1176 possible segmental deformation traces, 111 could not be analysed (90% feasibility). Apical transverse motion could be calculated in 63 patients (94% feasibility).

Reproducibility of data acquisitions and readings of two experienced echocardiographers was tested. Segmental peak systolic velocity and time-to-peak velocity measurements correlated excellently ($r = 0.94$ and $r = 0.99$, respectively). Bland–Altman analysis revealed only minor differences (mean ± SD) 0.0 ± 0.6 cm/s and 1.4 ± 20.4 ms, respectively. Peak syst strain and time-to-peak strain correlated well ($r = 0.77$ and $r = 0.82$, respectively). Difference between readings (mean ± SD) was small (0.6 ± 3.3% and 0.0 ± 70 ms). ATM$_{4CV}$ estimates for the entire cardiac cycle ($r = 0.97$) as well as during ejection time ($r = 0.98$) correlated very well and differed only minimally between readings (mean ± SD) by 0.1 ± 0.6 and 0.2 ± 0.6 mm, respectively (Figure 4).

Figure 4 Interobserver variability of ATM$_{4CV}$ estimates according to the proposed Doppler-based method in (mm). (A) ATM$_{4CV}$ during the entire cardiac cycle. (B) ATM$_{4CV}$ during ejection time. Solid black lines indicate the linear regression between readings, the grey lines the lines of identity. (C and D) Bland–Altman plot of the same data. Solid black lines indicate the mean difference between readings (C: −0.10; D: +0.20), the dotted black lines the mean difference ± 1.96 ± SD (C: −1.27 to 1.08; D: −0.93 to 1.33). The confidence interval of the latter was ± 0.05 in both plots.
Temporal and regional distribution of myocardial function

In order to characterize the temporal and regional distribution of myocardial function, we measured the segmental onset of shortening ($t_{\text{onset}}$) as well as the amplitude of segmental shortening during ejection time ($\varepsilon_{\text{et}}$) and during the total cardiac cycle ($\varepsilon_{\text{tot}}$). In NORM, LV segmental shortening began shortly after QRS in the septal wall and spread over the LV within 25 ms. Right ventricular (RV) shortening occurred during the same time. Average shortening amplitude of LV segments was $\varepsilon_{\text{tot}} = 219.0 \pm 2.1\%$ and $\varepsilon_{\text{et}} = 214.8 \pm 2.3\%$. Inferior, septal, and anteroseptal walls showed on average significantly higher deformation values compared with the other walls ($\varepsilon_{\text{et(inf,sep,ans)}} = -16.8 \pm 2.4\%$ vs. $\varepsilon_{\text{et(ant,lat,pos)}} = -12.6 \pm 2.6\%$, $P < 0.001$) (Figure 5A).

In RBBB patients, onset of LV segmental shortening was earliest in the lateral wall, reaching the anteroseptum and the RV within 43 ms (no significant difference vs. NORM). The average shortening amplitude of LV segments was also similar ($\varepsilon_{\text{tot}} = -18.2 \pm 2.4\%$, $\varepsilon_{\text{et}} = -14.1 \pm 2.3\%$, both not significant vs. NORM). Distribution of strain between the walls was similar to NORM ($\varepsilon_{\text{et(inf,sep,ans)}} = -15.6 \pm 2.8\%$ vs. $\varepsilon_{\text{et(ant,lat,pos)}} = -12.7 \pm 2.4\%$, $P = 0.002$) (Figure 5B).

In nLBBB patients, shortening was earliest seen in the RV and spread over the heart reaching the LV posterior wall 144 ms later ($P < 0.001$ vs. NORM). The average LV segmental shortening was significantly reduced ($\varepsilon_{\text{tot}} = -14.4 \pm 3.4\%$, $\varepsilon_{\text{et}} = -7.4 \pm 3.4\%$, both $P < 0.001$ vs. NORM). The total strain amplitude did not differ between walls ($P = 0.655$), whereas the ejection time strain was inhomogeneous, with significantly lower average shortening of the inferior, septal, and anteroseptal walls compared with others ($\varepsilon_{\text{et(inf,sep,ans)}} = -5.9 \pm 4.3\%$ vs. $\varepsilon_{\text{et(ant,lat,pos)}} = -8.2 \pm 4.0\%$, $P = 0.002$) (Figure 5C).

**Figure 5** Bull’s eye displays of average regional strain distribution (first two rows) in (A) controls, (B) patients with RBBB, (C) non-ischaemic LBBB, and (D) ischaemic LBBB. Note the rather homogeneous distribution of the total myocardial deformation during the cardiac cycle ($\varepsilon_{\text{tot}}$), while strain during ejection time ($\varepsilon_{\text{et}}$) distribution differs between groups. In NORM and RBBB, $\varepsilon_{\text{et}}$ is almost as high as $\varepsilon_{\text{tot}}$ and more homogeneous. In nLBBB, $\varepsilon_{\text{et}}$ is lower and occurs predominantly in the left-sided regions of the LV. In iLBBB, the distribution is less homogeneous. The last row shows the temporal distribution of onset of shortening (see text for details).

### Table 2 Conventional parameters of asynchrony

<table>
<thead>
<tr>
<th></th>
<th>IVD</th>
<th>TsSD12</th>
<th>SysDisp</th>
<th>SLDisp</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORM</td>
<td>0 (-7 to 7)</td>
<td>34 (12–49)</td>
<td>49 (34–105)</td>
<td>21 (9–60)</td>
</tr>
<tr>
<td>RBBB</td>
<td>43 (30–48)</td>
<td>33 (23–47)</td>
<td>34 (13–61)</td>
<td>17 (9–47)</td>
</tr>
<tr>
<td>nLBBB</td>
<td>-56 (-75 to -30)*</td>
<td>55 (44–71)*</td>
<td>92 (69–112)</td>
<td>71 (50–95)*</td>
</tr>
<tr>
<td>iLBBB</td>
<td>-19 (-35 to -3)</td>
<td>47 (31–57)</td>
<td>49 (36–86)</td>
<td>49 (27–86)</td>
</tr>
<tr>
<td>Cut-off*</td>
<td>40</td>
<td>34.4</td>
<td>65</td>
<td>65</td>
</tr>
</tbody>
</table>

All values are given as median (and interquartile range in brackets) in (ms).

*Significant difference vs. NORM. See text for further details.

*Cut-off to best distinguish responders and non-responders to CRT as suggested in the original publications.
ATM in this plane only (ATM$_{4CV}$). ATM$_{4CV}$ values differed only slightly from ATM. Also in ATM$_{4CV}$, nLBBB patients showed significantly more apical motion than NORM and RBBB (NORM 1.6 (1.0–2.0) mm; RBBB 1.1 (0.8–1.5) mm, \( P = 0.999 \) vs. NORM; nLBBB 4.0 (2.6–4.9) mm, \( P < 0.001 \) vs. NORM and RBBB), whereas iLBBB showed significantly more apical motion compared with RBBB patients [2.1 (1.8–3.4) mm, \( P = 0.114 \) vs. NORM, \( P = 0.022 \) vs. RBBB] (Figure 7B). On average, ATM$_{4CV}$ during ejection time (ATM$_{4CV}$$_{et}$) showed comparable relations, but lower motion amplitudes.

ATM$_{4CV}$ did not correlate with QRS width (all \( r < 0.15 \)) or EF (all \( r < 0.25 \)) within the groups of NORM, RBBB, and LBBB.

To further analyse the relation of ATM towards regional contraction inhomogeneity within the LV, we calculated the difference between the averaged strains of the septal and lateral wall segments during ejection time (\( \varepsilon_{\text{diff}} = \varepsilon_{\text{et}}(\text{sep}) - \varepsilon_{\text{et}}(\text{lat}) \)). We found a significant correlation of ATM$_{4CV}$ with this marker of regional function inhomogeneity (\( r = 0.74, \text{CI} \ 0.57–0.85, P < 0.001 \)). Only IVD correlated (weaker) as well, whereas none of the conventional asynchrony parameters showed a relevant correlation to this marker of regional function inhomogeneity (Figure 8).

The ROC analysis for the distinction between NORM and LBBB groups revealed for ATM$_{4CV}$ an area under the curve of 0.87 (CI 0.78–0.97, \( P < 0.001 \)). See Figure 9 for comparison to conventional tissue velocity-based parameters.

**Discussion**

**Main findings of the study**

In this study, we could demonstrate inhomogeneities in temporal and regional distribution of myocardial shortening, particularly during ejection time, which differed among healthy volunteers and patients with different conduction delays.

We introduced the new parameter of ATM. We could show that ATM estimation by means of tissue Doppler echocardiography is technically feasible and reproducible and that it allows to quantify and distinguish typical motion patterns of the LV. Apical transverse motion was strongly related to LV temporal and regional function inhomogeneities and summarized them in a single parameter. Apical transverse motion could distinguish well between NORM and LBBB study groups.

**Temporal and regional function inhomogeneities**

Our findings confirm earlier works in animals\(^{1,12}\) and humans\(^{13,14}\) which reported a delayed onset of systolic shortening in the lateral and posterior LV region of patients with LBBB. They also confirm that in LBBB, the lateral and posterior walls contract mainly during ejection,\(^{12}\) whereas septal regions of the LV shorten very early and, thus, can hardly contribute to the stroke work. We found that the average ejection time strain per wall mirrors these conditions well: in NORM and RBBB, the \( \varepsilon_{\text{et}} \)-differences between walls were low with slightly higher values in the septal region, whereas LBBB patients revealed a significant imbalance in \( \varepsilon_{\text{et}} \) favouring the lateral wall. We found a significantly time delay in onset of regional shortening in the lateral region of nLBBB patients. This relation was less pronounced in LBBB patients with ischaemic background.
which may be attributed to the different location and extent of infarct scar in this group.

Conventional asynchrony parameters

To estimate this temporal and functional LV inhomogeneities in our patients, we choose Doppler- and tissue-Doppler-based parameters which were previously suggested for use in the context of CRT.\textsuperscript{2,3,5,10,11} Although our study population was not investigated with the intention to predict CRT response, it may be assumed that those parameters are able to reflect LV asynchrony.

IVD showed differences between groups and correlated weakly with the septal–lateral strain difference. TsSD12 was significantly prolonged in nLBBB. No other significant difference was detected by the tissue-Doppler-based parameters (Table 2). None of the tissue-Doppler-based parameters correlated significantly with the septal–lateral strain difference (Figure 8). We conclude, thus, that the conventional tissue-Doppler-based asynchrony parameters may in part reflect temporal asynchrony, but do not directly reflect inhomogeneities of regional LV function.

Apical transverse motion

In this study, we suggest for the first time to use the new parameter of ATM for the assessment of LV asynchrony. We hypothesize that apical motion is a surrogate parameter,
Limitations

The assessment of ATM was tissue-Doppler-based, assuming that the apex behaves as a homogeneous ‘cap’. A more direct assessment using modern speckle tracking approaches was not feasible due to the inherent image artefacts in the apex region. Furthermore, the chosen way of ATM estimation is theoretically more resistant to an overall sideward motion of the LV than tracking based approaches, since tissue-Doppler data are collected more longitudinally and are, thus, not influenced by a possible sideward component of myocardial motion.

Apical transverse motion could not be validated against a gold standard. Such a direct validation proved to be difficult because of the lack of a gold standard method. In particular, MRI tagging was attempted, but failed due to the flawed signals in the thin apical myocardium, the insufficient temporal and spatial resolution of the method, and the influence of the above mentioned overall heart motion. Echocardiographic tracking methods are frequently disturbed by the stationary artefacts in the apical region. Ultrasonic crystals would be an option, but cannot be used in humans, would influence the apical motion due to the open pericardium procedure, and would theoretically also be subject to overall heart motion interference.

Since ATM was shown to be related to functional inhomogeneities in LV walls, a relation between ATM and the extend and location of myocardial scar tissue must be expected. Our study is underpowered to address this specific question which leaves room for further investigation.

The mean age of our control group was lower than the age of the patients. We therefore performed a sub-analysis of our data which compared ATM4CV of volunteers <30 years and >50 years, which revealed no significant differences (1.47 ± 0.65 mm vs. 1.61 ± 0.38 mm, P = 0.640). Furthermore, we noted no correlation between age of the volunteers and ATM4CV (r = 0.12).

Conclusion

Apical transverse motion is a new and simple parameter of LV asynchrony. It integrates information on both regional and temporal function inhomogeneities of the LV and, thus, may be used to assess LV asynchrony in the clinical context. Our data suggest that ATM has the ability to differentiate patients with differing function and conduction abnormalities. Apical transverse motion estimation is feasible and well reproducible. Further studies will determine its potential role in predicting response to CRT.

Funding

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Conflict of interest: none declared.

References


Potential advantages of apical transverse motion

Apical transverse motion appears to better reflect the true sequence of regional myocardial shortening in the LV walls and the different magnitudes of deformation. It may, therefore, be used to characterize different entities of LV pathology better than with traditional, velocity peak timing-based methods. This may be of an advantage in analysing LV dyssynchrony and in selecting CRT candidates. comprizing information on both regional myocardial function inhomogeneities and temporal inhomogeneities of myocardial contraction due to activation delays. Thus, a pronounced apical motion (‘apical rocking’) will occur, if either one of two opposing wall contracts weaker (e.g. scar tissue) or delayed (conduction delay).

This is supported by the facts that ATM correlates well with the septal–lateral difference in myocardial wall strain as a marker of function inhomogeneities and that ATM showed different typical apical motion patterns in different conduction delays. It, furthermore, could distinguish between the normal hearts and LBBB patients with and without scars.

Given the good reproducibility of the ATM measurements, the scatter of ATM within the LBBB groups may be interpreted as a confirmation that mechanical and electrical dyssynchrony are only loosely related.15

Figure 9 Receiver-operating-characteristic analysis for ATM4CV and different conventional Doppler based dyssynchrony parameters for the distinction between NORM and LBBB patient groups. Significance levels are (1) $P < 0.001$, (2) $P = 0.003$ ($P = 0.1532$ vs. ATM4CV), (3) $P = 0.003$ ($P = 0.1322$ vs. ATM4CV), (4) $P = 0.058$ ($P = 0.0221$ vs. ATM4CV).


