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Longitudinal left ventricular function is globally depressed within a week of STEMI

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Summary

Sixty percent of stroke volume (SV) is generated by atrioventricular plane displacement (AVPD) in a healthy left ventricle (LV). The aims were to determine the effect of ST-elevation myocardial infarction (STEMI) on AVPD and contribution of AVPD to SV and to study the relationship between AVPD and infarct size (IS) and location. Patients from CHILL-MI and MITOCARE studies with cardiovascular magnetic resonance within a week of STEMI (n = 177, 59 ± 11 years) and healthy controls (n = 20, 62 ± 11 years) were included. Left ventricular volumes were quantified in short-axis images. AVPD was measured in six locations in long-axis images. Longitudinal contribution to SV was calculated as AVPD multiplied by the short-axis epicardial area. Patients (IS 17 ± 10% of LV) had decreased ejection fraction (48 ± 8%) compared to controls (60 ± 5%, P<0.001). Global AVPD was decreased in patients (11 ± 2 mm versus 15 ± 2 mm in controls, P<0.001) and this held true for both infarcted and remote segments. AVPD contribution to SV was lower in patients (58 ± 9%) than in controls (64 ± 8%) (P<0.001). There was a weak negative correlation between IS and AVPD (r²=0.06) but no differences in global AVPD linked to infarct location. Decrease in global and regional AVPD occur even in remote myocardium within 1 week of STEMI. Global AVPD decrease is independent of MI location, and MI size has only minor effect. Longitudinal pumping is slightly lower compared to controls but remains to be the main component to SV even after STEMI. These results highlight the difficulty in determining infarct location and size from longitudinal measures of LV function.

Introduction

Myocardial infarction (MI) is common and linked to high morbidity and mortality (McMurray et al., 2012) (WHO 2017). Patients with ST-elevation myocardial infarction (STEMI) require reperfusion by primary percutaneous coronary intervention (pPCI) or thrombolysis within 2 h of presentation (Ibanez et al., 2018). Despite timely coronary intervention, a significant number of patients develop left ventricular (LV) dysfunction or heart failure after MI. The severity of LV dysfunction after STEMI (Stolfo et al., 2016) and the MI size (van Krantenburg et al., 2014) are important prognostic markers used for risk stratification and therapeutic decision-making.

The most commonly used measure of LV systolic function is ejection fraction (EF). It is a strong predictor of morbidity and mortality in patients with reperfused acute STEMI (Group 1983) and is known to negatively correlate with MI size (Ugander et al., 2008) (Wu et al., 2008). Left ventricular function can also be measured as stroke volume (SV), and it can be further divided into longitudinal, septal and non-septal radial (previously called lateral) components (Stephensen et al., 2014). The longitudinal component is caused by atrioventricular plane displacement (AVPD) towards the apex in systole and back towards the base in diastole. Atrioventricular plane displacement is the main contributor to LV SV, counting for 60% in the normal heart (Stephensen et al., 2014). Reduced AVPD has been associated with ageing (Steding-Ehrenborg...
et al., 2015) and disease (Stephensen et al., 2014), while increased AVPD is seen in male athletes (Steding-Ehrenborg et al., 2013). It is known that MI causes decreased AVPD (Brand et al., 2002) but it is not known how it correlates with MI size and MI location. Mitral annular plane systolic excursion (MAPSE), which corresponds to regional AVPD measured at the mitral annulus, was recently shown to provide strong prognostic information, independent of EF (Rangarajan et al., 2016; Romano et al., 2017). Thus, AVPD may add valuable information when assessing LV function after STEMI. The septal and non-septal radial components of SV account for the remaining 40% of the LV SV. These are generated by the inward epicardial motion from end-diastole (ED) to end-systole (ES) and are a potential new measure of regional LV function (Stephensen et al., 2014). The proportion of longitudinal, septal and non-septal radial pumping in patients with STEMI is unknown.

Our hypothesis was that longitudinal LV function is decreased within a week of STEMI, mostly affecting the infarcted walls of the LV. Our aims were to determine the effects of STEMI on global and regional AVPD, the contribution of AVPD, septal and non-septal radial motion to SV and to study the relationship between AVPD and infarct size (IS) and location.

Methods

Study population

Patients from two recent international multicenter cardioprotection studies, CHILL-MI (Erlinge et al., 2014) (NCT01379261) and MITOCARE (Atar et al., 2015) (EudraCT number 2010-024616-33) were considered for inclusion this study. The patients had a first-time STEMI, were >18 years old, presented with acute chest pain lasting for less than six hours and underwent PCI. All patients included in this study had successful reperfusion of the occluded vessel and underwent cardiovascular magnetic resonance (CMR) within the first week of their STEMI. The 97 patients from CHILL-MI and 93 from MITOCARE who underwent CMR study within a week of STEMI and were considered for this study. Eight patients were excluded due to incomplete data and five due to poor image quality. Thus, a total of 177 patients were ultimately included in the study. In addition, healthy controls (n = 20) were recruited through advertising for comparison and details for this population have previously been described (Asgeirsson et al., 2017). The study was approved by the regional ethics committee and written informed consent was obtained from all patients at inclusion in the original studies.

Cardiovascular magnetic resonance image acquisition

The study protocol included a CMR scan at 2–7 days after pPCI for the STEMI. The imaging protocols have been previously published (Erlinge et al., 2014; Atar et al., 2015). Imaging was performed at multiple centres throughout Europe, and multiple vendors were used (Philips Healthcare, Best, The Netherlands/Siemens AG, Erlangen, Germany/GE healthcare, Waukesha Wi, USA). Patients were examined in supine position, and images were acquired at end-expiratory breath hold with ECG-gating. Steady-state free precession (SSFP) cine short-axis images covering the entire LV were acquired after administration of 0.2 mmol kg\(^{-1}\) gadolinium and were used for analysis of myocardial function. Long-axis cine SSFP images in the two-, three- and four-chamber views were also acquired and used for AVPD measurements. Temporal resolution depended on heart rate but 20–30 time frames were obtained per cardiac cycle. Late gadolinium-enhanced (LGE) images were acquired 15–20 min after injection of a gadolinium contrast agent to determine infarct size. Spatial resolution was typically 1.5 × 1.5 × 8 mm with no slice gap.

Cardiovascular magnetic resonance analysis

Images were analysed using Segment, version 2.0 (http://segment.heiberg.se) (Heiberg et al., 2010) by a CMR core laboratory (Imacor AB, Lund, Sweden). Manual delineations of the endo- and epicardium of the SSFP short-axis cine images were performed in end-diastole (ED) and end-systole (ES). End-systolic and end-diastolic volumes, SV and EF were calculated from endocardial contours.

Short-axis SSFP images were utilized when determining septal and non-septal radial contribution to SV. The most basal slice used for analysis depicted a circumferential LV in both ED and ES and the most apical slice depicted the apex in both ED and ES (Fig. 1). The two right ventricular (RV) insertion points were manually marked on the epicardial border of the LV in both ED and ES. Septal contribution to SV was defined as % of SV generated on the septal side of the RV insertion points and non-septal radial as the volume generated by the movement on the non-septal side of the RV insertion points in all short-axis cine images with circumferential myocardium in systole (Fig.1).

The AVPD was determined by manually marking the AV-plane in six locations (anterior, anteroseptal, inferoseptal, inferior, inferolateral and anterolateral) in ED and ES in three long-axis images. AVPD was determined for each location by subtracting the perpendicular AV-plane position in ES from the position in ED in reference to the apex. Left ventricular AVPD was defined as the mean of the six displacements (Carlsson et al., 2007) (Fig. 2). The SV generated by the AVPD was calculated as LV AVPD (cm) multiplied by the mean of the largest LV short-axis epicardial area (cm\(^2\)) within the AVPD range as previously described (Carlsson et al., 2007).

Infarct size was quantified in LGE images after manual delineation of the endocardial and epicardial borders using a validated automatic algorithm with manual corrections as previously described (Engblom et al., 2016) (Erlinge et al., 2014; Atar et al., 2015). Phase-sensitive inversion recovery LGE was used for infarct quantification in 28% of the patients.
Longitudinal strain was determined for the anterior, anteroseptal, inferoseptal, inferior, inferolateral and anterolateral walls of the LV by measuring the distance from the AV-plane to the apex at ED and ES and dividing the difference by the ED distance as previously described and validated (Riffel et al., 2015):

\[
\text{Longitudinal strain} = \frac{(\text{Distance in ES} - \text{distance in ED})}{\text{distance in ED}}
\]

**Statistical analysis**

Statistical analysis was performed using Microsoft Excel 2010. Continuous variables were presented as mean ± SD. Student t-test was used to compare results between patients and controls. Linear regression analysis was performed to determine the correlation between infarct parameters (size, transmurality, myocardium at risk) and functional parameters (EF, AVPD and AVPD contribution to stroke volume). One-way ANOVA with Tukey post hoc test was used to compare results between patients with different infarct locations. A result with a P-value <0.05 was considered statistically significant. Interobserver and intraobserver variability was calculated in 20 subjects and was reported as bias ± SD and intraclass correlation (ICC). Internal validation of the contributions SV was obtained by adding AVPD, septal and non-septal radial contributions to SV for each subject where the result ideally would add up to 100% of SV.
Results

Subject characteristics

Table 1 summarizes baseline characteristics. Patients and controls were of similar age while a higher proportion of the patients were men (87% versus 60%). Infarct size ranged 2-46% of LV mass (LVM). Stroke volume and global AVPD (mm) was lower in patients than in controls (P<0.01 and P<0.001, respectively). Patients with right coronary artery (RCA) infarcts had higher EF (51 ± 6%) than patients with left anterior descending (LAD) (45 ± 9%, P<0.001) and left circumflex (LCx) (47 ± 9%, P<0.05) infarcts (Fig 3). All infarct groups had lower EF compared to controls. Infarct size was largest in patients with LAD infarction (23 ± 10%) followed by LCx (15 ± 8%) and RCA (13 ± 7%).

Atrioventricular plane displacement in patients was decreased in all walls of the LV compared to controls (P<0.001) (Table 2, Fig. 4). The decrease was not only significant in the LV walls directly affected by the MI (for example anterior and anteroseptal walls for LAD infarction) but also in walls remote to the MI (inferolateral and inferior walls for LAD infarction) (Table 3). There was a negative correlation between EF and IS (R²=0.33, P<0.001) (Fig. 5) and a weak negative correlation between AVPD and IS (R²=0.06, P<0.001). There was no differences between global AVPD in the different MI location (LAD, RCA or LCx), P = 0.18.

Atrioventricular plane displacement contribution to SV was lower in patients (58 ± 9%) than in controls (64 ± 8%) (P<0.001) (Table 4) but did not differ between the LAD, RCA and LCx groups (P = 0.46). The non-septal radial contribution was no different in patients and controls except for patients with LCx infarcts who had lower lateral contribution to SV compared to patients with LAD and RCA infarcts (P<0.001). Consequently, septal contribution was increased in LCx infarcts when compared to controls and patients with RCA and LAD infarcts (P<0.05, P<0.01 and P<0.001, respectively). The epicardial short-axis areas used for calculation of AVPD contribution to SV was 40 ± 6 cm² for controls, 43 ± 7 cm² for the entire patient group (44 ± 7 cm² for LAD, 42 ± 7 cm² for RCA and 43 ± 7 cm² for patients with LCx infarcts). Only patients with LAD infarction had significantly larger epicardial short-axis area compared controls (P<0.05).

Linear regression analysis showed no relationship between the AVPD contribution to SV and infarct size (P = 0.31, R²=0.006), infarct transmurality (P = 0.97, R²<0.001) or myocardium at risk (P = 0.30, R² = 0.006).

Longitudinal strain values are shown in Table 5. Similar to AVPD longitudinal strain was globally decreased compared to controls irrespective of infarct location.

Gender differences

Table 6 presents findings by gender. Only 13% of the patient population were women, and they were significantly older than the male patients. Infarct size was smaller in women with a resulting higher EF. The lower SV in women compared to men is explained by a smaller LV size. However, these differences in EF did not translate into gender differences in longitudinal, septal or non-septal radial contributions to SV.

Intra- and interobserver variability

Intraobserver variability of the measurements of AVPD in 20 randomly selected patients with >12 months apart, showed a bias of 0-2 mm ± 0-7 mm, ICC 0.95 and r = 0.96. Interobserver variability (n = 20) showed a bias of 0-6 ± 0-7 mm, ICC 0.92, r = 0.95. Internal validation of the measurements by adding the longitudinal, septal and non-septal radial contributions to stroke volume showed 99 ± 9% for patients and 103 ± 9% for controls.

Discussion

This study has shown that AVPD and longitudinal strain is decreased in both infarcted and remote areas of the LV within a week of STEMI, indicating an effect of longitudinal LV function on a global level. Patients have slightly lower longitudinal...
contribution to SV (58 ± 9%) than controls (64 ± 8%), while septal and non-septal radial contributions were largely unchanged. We found only a weak negative correlation between global AVPD and IS and no relationship between AVPD and MI location.

The decreased AVPD and longitudinal strain in both infarcted and remote LV walls is most likely due to the direct (infarction) and indirect (stunning) result of ischaemia. Furthermore, the global inflammatory response after STEMI with inflammatory cells and changes in contractile and mitochondrial proteins also in remote myocardium seen experimentally may explain the globally decreased function seen in our study (Binek et al., 2017). Another possible explanation for the global decrease could be the use of medications, such as...
Table 3  Atrioventricular plane displacement (AVPD) (mm) in infarcted and remote left ventricular walls. P<0.001 for all.

<table>
<thead>
<tr>
<th>Vessel and LV wall</th>
<th>AVPD patients (mm)</th>
<th>AVPD controls (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD infarct Anteroseptal</td>
<td>9 ± 2</td>
<td>13 ± 2</td>
</tr>
<tr>
<td>LAD remote Inferoseptal</td>
<td>13 ± 2</td>
<td>18 ± 2</td>
</tr>
<tr>
<td>RCA infarct Anteroseptal</td>
<td>12 ± 3</td>
<td>17 ± 13</td>
</tr>
<tr>
<td>RCA remote Inferoseptal</td>
<td>11 ± 3</td>
<td>15 ± 3</td>
</tr>
<tr>
<td>LCx infarct Inferoseptal</td>
<td>13 ± 3</td>
<td>18 ± 2</td>
</tr>
<tr>
<td>LCx remote Anteroseptal</td>
<td>10 ± 2</td>
<td>13 ± 2</td>
</tr>
</tbody>
</table>

Table 4  Absolute and relative atrioventricular plane displacement, septal and non-septal-radial contributions to stroke volume.

<table>
<thead>
<tr>
<th>Stroke volume (ml)</th>
<th>Patients n = 177</th>
<th>LAD n = 66</th>
<th>RCA n = 87</th>
<th>LCx n = 24</th>
<th>Controls n = 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct size (%)</td>
<td>85 ± 19***</td>
<td>84 ± 20*</td>
<td>87 ± 20</td>
<td>97 ± 20</td>
<td></td>
</tr>
<tr>
<td>AVPD SV (ml)</td>
<td>17 ± 10***</td>
<td>23 ± 10***</td>
<td>13 ± 7***</td>
<td>15 ± 8***</td>
<td></td>
</tr>
<tr>
<td>Septal SV (%)</td>
<td>49 ± 12***</td>
<td>48 ± 12***</td>
<td>49 ± 13***</td>
<td>51 ± 10**</td>
<td></td>
</tr>
<tr>
<td>LV SV (ml)</td>
<td>58 ± 9***</td>
<td>57 ± 8***</td>
<td>58 ± 10**</td>
<td>60 ± 10</td>
<td></td>
</tr>
<tr>
<td>Septal SV (%)</td>
<td>10 ± 6</td>
<td>8 ± 5</td>
<td>10 ± 5</td>
<td>14 ± 6*</td>
<td></td>
</tr>
<tr>
<td>Non-septal radial SV (ml)</td>
<td>26 ± 9*</td>
<td>27 ± 10</td>
<td>27 ± 9</td>
<td>23 ± 9</td>
<td>28 ± 9</td>
</tr>
<tr>
<td>Non-septal radial SV (%)</td>
<td>31 ± 10</td>
<td>31 ± 8</td>
<td>33 ± 10</td>
<td>27 ± 11</td>
<td>29 ± 7</td>
</tr>
</tbody>
</table>

*p<0.05, **p<0.01, ***p<0.001 compared to healthy controls.

Table 5  Longitudinal strain for patients and controls in the walls of the left ventricle (P<0.001 for all except the anterolateral wall in patients with LCx infarct <0.01).

<table>
<thead>
<tr>
<th>Anterior</th>
<th>Anteroseptal</th>
<th>Inferoseptal</th>
<th>Inferior</th>
<th>Inferolateral</th>
<th>Anterolateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD</td>
<td>−0.01 ± 0.03</td>
<td>−0.09 ± 0.03</td>
<td>−0.11 ± 0.03</td>
<td>−0.13 ± 0.03</td>
<td>−0.13 ± 0.03</td>
</tr>
<tr>
<td>RCA</td>
<td>−0.12 ± 0.03</td>
<td>−0.11 ± 0.03</td>
<td>−0.12 ± 0.02</td>
<td>−0.13 ± 0.03</td>
<td>−0.15 ± 0.03</td>
</tr>
<tr>
<td>LCx</td>
<td>−0.11 ± 0.02</td>
<td>−0.11 ± 0.04</td>
<td>−0.12 ± 0.02</td>
<td>−0.14 ± 0.04</td>
<td>−0.13 ± 0.03</td>
</tr>
<tr>
<td>Controls</td>
<td>−0.14 ± 0.02</td>
<td>−0.14 ± 0.02</td>
<td>−0.16 ± 0.02</td>
<td>−0.19 ± 0.03</td>
<td>−0.19 ± 0.03</td>
</tr>
</tbody>
</table>

Table 6  Gender differences

<table>
<thead>
<tr>
<th></th>
<th>Male (n = 154)</th>
<th>Female (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>58 ± 12</td>
<td>66 ± 10***</td>
</tr>
<tr>
<td>EF (%)</td>
<td>48 ± 8</td>
<td>52 ± 8*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71 ± 9</td>
<td>85 ± 12***</td>
</tr>
<tr>
<td>IS (%)</td>
<td>17 ± 9</td>
<td>12 ± 9*</td>
</tr>
<tr>
<td>LV SV (ml)</td>
<td>87 ± 18</td>
<td>70 ± 10***</td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>184 ± 38</td>
<td>140 ± 38</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>97 ± 31</td>
<td>70 ± 26***</td>
</tr>
<tr>
<td>AVPD (mm)</td>
<td>11.5 ± 2-3</td>
<td>10.8 ± 2-2</td>
</tr>
<tr>
<td>AVPD contribution</td>
<td>58 ± 9</td>
<td>56 ± 11</td>
</tr>
<tr>
<td>to SV (%)</td>
<td>10 ± 9</td>
<td>10 ± 5</td>
</tr>
<tr>
<td>Septal contribution to SV (%)</td>
<td>31 ± 9</td>
<td>32 ± 12</td>
</tr>
</tbody>
</table>

*p<0.05, **p<0.01 ***p<0.001 compared to men.

as beta-blockers after MI. The global decrease may decrease the possibility to determine MI location or culprit vessel from long-axis measurements or longitudinal strain. This is supported by Rosendahl (Rosendahl et al., 2010) who found only a 64% sensitivity of longitudinal strain by echocardiography to detect infarction with a 80% specificity. Furthermore, an echocardiography study in 19 MI patients also have shown the inability to localize the infarction from AVPD measurements (Stoylen & Skjærpe, 2003). The recently shown prognostic significance of using only the lateral AVPD (Rangarajan et al., 2016; Romano et al., 2017) can further be understood by this global effect on AVPD of a STEMI.

The proportion of longitudinal, septal and non-septal radial contributions to LV SV was 64%, 29% and 10%, respectively for controls. The longitudinal contribution is slightly larger than the 61% previously found in younger healthy controls (Stephensen et al., 2014). The controls in this study were older than those in Stephenson’s study (66 years versus 32 years old) and this could be a possible reason for this difference. Healthy children have been found to have an even lower longitudinal contribution to SV (50%). Thus, there may be a gradual increase in longitudinal contribution to SV with age even though the absolute AVPD decreases with age (Ochs et al., 2017) and this may possibly be explained by decrease
in absolute SV and increase in short-axis LV diameter. In our study, women had smaller infarcts and less decrease in EF but the contributions to SV was similar to men.

Stokke et al., (2017) recently showed that circumferential strain contributes more than twice as much as longitudinal strain to LV EF. Their findings may appear conflicting to the results in the present study (that AVPD is responsible for about 60% of SV). However, both findings are valid and not contradictory. The geometrical model used by Stokke et al. give short-axis circumferential function to be dominant due to (i) more fibres are in the circumferential direction and (ii) longitudinal shortening contributes to wall thickening due to a small epicardial displacement and conservation of myocardial tissue volume. Also, the myocardial fibres are arranged in a syncytium, and the intricate helical relationship between them and resulting twist/torsion explain how a 15% sarcomere shortening give rise to a much higher ejection fractions than longitudinal shortening (Sallin, 1969; Taber et al., 1996). This helical arrangement means that circumferential shortening (strain) contribute to longitudinal shortening. The approach in this article uses an atrioventricular reciprocating volume approach where the LV is a piston pump generating the SV, where the movement of the piston is the AVPD, and the epicardial area is the piston area. In addition to being the main contributor to LV SV, AVPD generates systolic atrial filling from the great veins and thus plays an important role in LV diastolic filling (Smiseth et al., 1999; Steding-Ehrenborg et al., 2013). AVPD thus accounts for the majority of blood ejected from the LV and plays an important role in filling the LV in both healthy person and after a STEMI. The decrease in longitudinal contribution to SV in MI patients thus results in a decreased filling of the heart into the left atrium during systole. This will be seen as a decrease in the Doppler S-wave velocity of the pulmonary veins and a reduced systolic fraction with VTI of the pulmonary venous flow echocardiography and may therefore influence the use of these parameters when estimating mean atrial pressure (Nagueh et al., 2016). In summary, the results from Stokke et al. studying myocardial strain and our results are complementary.

Palazzuoli et al. studied 133 patients 6–12 months after MI and found a linear relationship between scar extension and regional wall motion abnormalities especially in patients with transmural infarcts (Palazzuoli et al., 2015). They did not, however, study if the wall motion abnormality occurred in the infarcted or remote walls. They found greater systolic dysfunction in patients with transmural scar. We found a negative correlation between myocardial IS and EF \( (r^2 = -0.33 \text{ which equals } r = -0.57) \) that is similar to that found by Eitel et al. (Eitel et al., 2015) \( (r = -0.5) \) and Ugander et al. (Ugander et al., 2008) but less strong than the correlation found by Wu et al. (Wu et al., 2008) \( (r = -0.75) \) (Palazzuoli et al., 2015). The mean IS in the study by Wu et al. was larger than in our population (22% and 17% respectively) and the mean EF was lower (41% and 48%) but it is unclear if that is the reason for the difference in correlation between EF and IS. The proportion of AVPD, septal or non-septal radial contribution to SV did not correlate with IS. This suggests that the proportions are desirable to maintain optimal LV function.

Longitudinal function has prognostic effects in patients with heart disease. Brand et al. (Brand et al., 2002) showed that all-cause mortality was strongly related to LV AVPD measured with echocardiography in patients after MI. They also found a higher rate of hospitalization for heart failure in patients with low AVPD further suggesting that AVPD is an important predictor of post-MI morbidity. Rangarajan et al. recently showed that MAPSE, which corresponds to the anterolateral AVPD, is an independent predictor of major adverse cardiovascular events (Rangarajan et al., 2016) using CMR. The hazard ratio for major cardiovascular events was 1.34 for 2 mm decrease in MAPSE and patients with MAPSE <11 · 1 mm (median) had 12% events compared to 24% for patients with MAPSE <11 · 1 mm. In our study population, the mean anterolateral AVPD was ~13 mm and thus higher compared to the study of Rangarajan et al., even though STEMI patients were included one week after the acute event. Also, Romano et al. showed strong independent prognostic significance on all-cause mortality of lateral MAPSE in patients with EF <50% (Romano et al., 2017). The global decrease in AVPD even in a localized MI may help to explain the strong prognostic impact of measuring AVPD at the lateral position (MAPSE).

Although AVPD is not routinely measured and reported from clinical CMR studies it could easily be added. The images needed for analysis are routinely obtained, and measuring AVPD is relatively fast and easy. We have demonstrated low interobserver and intraobserver variability and a very strong ICC. Adding AVPD to routine CMR evaluations could provide incremental information when determining patients’ LV function.

Our patient group consisted of predominantly male patients, and female patients were older, had smaller infarcts and higher EF. These findings are similar to other STEMI trials (Kelbaek et al., 2016) (Reinstadler et al., 2016). However, these differences between female and male patients did not result in gender differences in the proportional contribution to SV.

Variability of measurements between core laboratories have been shown to be low for both volumetric measurements (Suiniesiaputra et al., 2015) and IS (Klem et al., 2017). In the MITOCARE and CHILL-MI studies, all images were assessed by one observer and the analysis checked for quality by another observer, both having EACVI level 3 certification. The reproducibility of manual interactions in the patient population of CHILL-MI and MITOCARE have been shown to be very high (bias 1 ± 1%, \( r = 0.99 \)) and the bias of infarct quantification versus the reference standard of ex. vivo TTC is very low (−1 ± 1%, \( r = 0.98 \)) (Engblom et al., 2016).

Limitations

The group of healthy controls was comparatively small. The study population includes patients from two clinical trials with
treatment given to half the patient population. However, none of the primary studies showed any significant differences in IS, LV volumes or EF between the treatment and control arms (Erlinge et al., 2014; Atar et al., 2015). Patients were selected to participate in the clinical trials, but we do not have the information on what percentage of patients the selection represents at the participating hospital and thus there may be a selection bias.

Conclusion

Patients have significantly decreased global and regional AVPD within 1 week of STEMI, even in myocardium remote to the infarct. The global AVPD decrease is independent of MI location, and MI size has only a minor effect on the degree of AVPD decrease. Longitudinal contribution is slightly lower compared to controls but remains to be the main component to SV even after MI. These results highlight the difficulty in determining infarct location and size from longitudinal measures of LV function.

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