Cardiac Magnetic Resonance to evaluate the left ventricular diastolic function after revascularization with and without left ventricular restoration Surgery

Gopal Ghimire, Jyotshana Shrestha, Kamal Joshi, Peter Hill, Steven Boyce, Anthon Fiusz, Gaby Weissman

Introduction

An acute myocardial infarction (AMI) produce alterations in the topography of the infarcted regions of the left ventricle (LV) and significant LV dilation occurred in 20-34% of patients despite successful reperfusion therapy.1-4 The viability assessment of myocardium remote from the infarcted region also undergoes remodelling leading to compensatory hypertrophy and expansion.5-7 Whilst, this tends to maintain the stroke volume by operation of the Frank-Starling mechanism through the operation of Laplace’s law, this dilation would augment diastolic and systolic wall stress and thereby stimulate further ventricular enlargement.8 Evidence suggests that LV volume to be a more predictive outcomes than ejection fraction (EF) after AMI.9 Hence, various surgical ventricular reconstruction (SVR) operations have been described that are based on the concept that reducing the LV cavity volume attenuates the wall tension, restores the elasticity.8 A recent RCT showed that SVR reduced the end-systolic volume index by 19%, as compared with a reduction of 6% with CABG, however this improvement in LV volume did not translate into a measurable benefit for the patients.9 The negative outcome of this study may be explained by the negative effect of this procedure on LV diastolic function.

Cardiac magnetic resonance (CMR) has been used as a reference standard for LV volumes and EF based on segmentation of isolated end-diastolic and end-systolic images.10 Cine-CMR acquires dynamic images throughout the cardiac cycle and thereby contains intrinsic data concerning volumetric changes during diastole and the volume curve can give an estimate of diastolic function.11 Recent evidence has established its usefulness in evaluating the diastolic LV filling profile when compared with echocardiography.12

Original Hypothesis

Left ventricular restoration surgery is associated with impairment of the left ventricular diastolic function

Methods

20 patients underwent either CABG + SVR or CABG alone with CMR performed pre and post procedure. The exams were performed on a 1.5T Philips Intera platform, using a BTFE sequence. LV volumetric quantification was performed using a View Forum workstation with manual planimetry of the endocardial contours on all phases of the contiguous short-axis slices from the level of the mitral annulus through the LV apex (Figure 1). The temporal phase volumes were used to obtain the volume time curve, and the first derivative which provide dV/dt to obtain various diastolic parameters:

1. Peak filling rate (PFR): Maximal LV filling rate defined by maximal change in LV volume between sequential temporal phases (A volume/ A phase). This index was also added to EDV to generate normalized peak filling rate (NPFPR).

2. Time to peak filling rate (TPFR): Time interval between end-systole and peak filling rate.


4. Filling fraction (FF): that portion of the total stroke volume achieved between end-systole and the TFP.

The volumetric filling curve was also transformed to the first derivative to obtain a biphasic curve, and the first derivative which provide dV/dt to obtain various diastolic parameters:

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The volumetric filling curve was also transformed to the first derivative to obtain Figure 2. Representative LV filling curves (x axis, temporal phase; y axis, volumetric change). PFR, defined as maximal slope of (toumoluem)/ (temporal phase), and TPFR, volume time interval between end-systole and peak filling rate. DVR calculated as proportion of diastole necessary to recover a threshold of 80% LV stroke volume.

Figure 2. Representative LV filling curves (x axis, temporal phase; y axis, volumetric change). PFR, defined as maximal slope of (toumoluem)/ (temporal phase), and TPFR, volume time interval between end-systole and peak filling rate. DVR calculated as proportion of diastole necessary to recover a threshold of 80% LV stroke volume.

Figure 3. Mean E/A ratio of patient pre and post procedure: A: SVR and CABG . B: CABG alone

Total of 40 CMR images were obtained from 20 patients with coronary artery disease undergoing CABG with (n=10) and without (n=10) SVR. The images were obtained preoperatively and postoperatively after a mean duration of 4.3 months for CABG, and 32.5 months for CABG-SVR. In the SVR+CABG cohort the sequential EF, LVEDV and LVESEV were 41.29%, 219.72 ml and 131.09 ml followed by 32.86 % (P<0.009), 177.4 ml (p<0.031) and 91.41% (p=0.028). In CABG alone cohort, the changes were EF: 49.4% vs. 56.4 % (p=0.046), LVEDV: 163.2 ml vs. 147.4 ml (p=0.03) and LVESEV: 112 ml vs. 70ml (p=0.162). The LV diastolic parameters obtained are depicted in the table.

Table 1: Diastolic Function Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CABG pre</th>
<th>CABG post</th>
<th>SVR pre</th>
<th>SVR post</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF (%)</td>
<td>49.4</td>
<td>56.4</td>
<td>49.4</td>
<td>56.4</td>
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<tr>
<td>LVEDV/ml</td>
<td>163.2</td>
<td>147.4</td>
<td>163.2</td>
<td>147.4</td>
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<tr>
<td>LVESEV/ml</td>
<td>112</td>
<td>70</td>
<td>112</td>
<td>70</td>
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<tr>
<td>DVRp (%)</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>FF (%)</td>
<td>0.76</td>
<td>0.76</td>
<td>0.76</td>
<td>0.76</td>
</tr>
<tr>
<td>PFR (ml/sec)</td>
<td>32.86</td>
<td>177.4</td>
<td>32.86</td>
<td>177.4</td>
</tr>
<tr>
<td>TPFR (sec)</td>
<td>1.53</td>
<td>1.32</td>
<td>1.53</td>
<td>1.32</td>
</tr>
<tr>
<td>PFR (%)</td>
<td>4.3</td>
<td>2.11</td>
<td>4.3</td>
<td>2.11</td>
</tr>
<tr>
<td>DVRp (%)(SVR)</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
</tbody>
</table>

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Results

As anticipated revascularization with coronary artery by pass surgery, with or without surgical ventricular reconstruction augmented the left ventricular ejection fraction. Similarly our study replicated the fact that surgical ventricular reconstruction is effective in reducing the end systolic volumes. Interestingly and contrast to the published data on the past surgical ventricular reconstruction is not associated with worsening of diastolic function although a part of the left ventricle is surgically excluded.

Conclusion

This study is limited by small sample size. We only included the patient who survived and did not get any implantable devise, thus sicker patient population might be excluded. The diastolic filling parameters might be used for the LV filling parameters and direct comparison with echocardiography was not done.

References