Mechanism behind pre- and postejection velocity spikes in normal left ventricular myocardium

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Background: The velocity trace of normal left ventricular (LV) wall motion has distinct waves corresponding to the different phases of the cardiac cycle. However, there are two unexplained velocity spikes in the trace - one just before and one just after the ejection wave. We hypothesize that the pre- and post-ejection velocity spikes are due to early-systolic shortening and late-systolic lengthening that is interrupted by mitral (MVI) and aortic valve closure (AVC), respectively.

Methods and results: LV long- and short-axis diameters were measured by somonocmetry in 11 anesthesized dogs. Myocardial shortening started prior to MVC (21±10 ms). There was excellent agreement between MVC and interruption of the initial shortening during early systole (4±7 ms) where the interval was defined as peak deceleration of shortening. Onset of lengthening preceded MVC by (31±15 ms). Interruption of the late systolic lengthening, defined as peak deceleration of lengthening, corresponded (0±3 ms) to AVC. We further investigated if abolishing aortic and mitral valve closure by stenting the valves would eliminate the pre- and post-ejection velocity spikes in separate experiments. Stenting of the mitral valve essentially abolished the prejection velocity spike, and stenting the aortic valve essentially abolished the postejection velocity spike. In a group of 10 healthy individuals LV longitudinal and radial shortening were measured by speckle tracking echocardiography. Peak deceleration of the early systolic shortening coincided (2.1±4 ms) with MVC, while peak deceleration of late systolic lengthening coincided (5±12 ms) with AVC.

Conclusion: This study support the hypothesis that the normal LV pre- and post-ejection velocity spikes are attributed to mitral and aortic valve closure that temporarily interrupt early systolic shortening and late systolic lengthening, respectively.

LV FUNCTION – OTHER

737 The effect of preload reduction by hemodialysis on conventional and novel parameters of left ventricular systolic and diastolic function

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Background: Prior studies of the effect of hemodialysis (HD) on left ventricular (LV) function brought ambiguous results, in particular regarding the assessment of LV diastolic function.

Methods: Thirty-two hypertensive patients with concentric LV hypertrophy (LVH) and in aortic banded mice were significantly higher than controls. To test the diagnostic performance of MPI to detect concentric LVH, we performed ROC curve analysis. Cutoff value of LV MPI >0.44 in human had sensitivity 65% and specificity 95%, and cutoff value >0.49 in mouse had sensitivity 100% and specificity 85%.

Conclusion: The MPI was a simple, noninvasive, and feasible Echo parameter to evaluate the left ventricular function both in patients with concentric LVH and in aortic banded mice.

Table 1. Concentric LVH and LV MPI

<table>
<thead>
<tr>
<th></th>
<th>Human control</th>
<th>Concentric LVH</th>
<th>Muirne control mice</th>
<th>Aortic banded mice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitrval inflow E/A ratio</td>
<td>1.3±0.2</td>
<td>0.8±0.2*</td>
<td>1.3±0.3</td>
<td>0.4±0.02*</td>
</tr>
<tr>
<td>Fractional shortening (%)</td>
<td>37.7±3.8</td>
<td>40.4±4.3</td>
<td>34.2±0.9</td>
<td>36.8±6.5</td>
</tr>
<tr>
<td>LV MPI</td>
<td>0.38±0.06</td>
<td>0.47±0.09*</td>
<td>0.42±0.05</td>
<td>0.59±0.09*</td>
</tr>
</tbody>
</table>

*p<0.001 vs each control

MYOCARDIAL VELOCITY IMAGING (DMI) – LV FUNCTION

739 Non-ischemic fibrosis can be reliably detected by a typical „double peak sign“ extracted from regional myocardial deformation curves

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Regional myocardial fibrosis can be non-invasively assessed by magnetic resonance imaging (MRI) using the late enhancement technique. This study investigated if regional non-ischemic fibrosis in hypertrophic myocardium is associated with a typical myocardial deformation pattern assessed by ultrasound strain rate imaging.

Methods: In 10 patients with hypertrophic cardiomyopathy, 10 patients with severe aortic stenosis and 10 patients with Fabry cardiomyopathy MRI with late enhancement imaging was done and the left ventricular segments with fibrosis were defined. In addition, strain rate imaging in all patients and also in 10 healthy controls was done and the strain rate curves were extracted for longitudinal and radial function.

Results: In all segments displaying late enhancement (LE) (n=41) a typical systolic strain rate pattern was detected: this pattern consisted of an early systolic peak followed by a rapid fall of strain rate near to the zero line and then again a second strain rate peak located during the diastolic relaxation period (Figure). This „double peak sign“ was never seen in the segments of the healthy control group. In addition, the „double peak sign“ was also detected in 10 of 79 segments displaying no LE. Interestingly, all these segments belonged to Fabry patients who are known to develop further progression of fibrosis. For 6 of these 10 segments follow-up MRI data after 2.5±1 years were available. All of them displayed LE suggesting that the „double peak sign“ might detect fibrosis earlier than MRI.

Conclusions: The „double peak sign“ assessed by strain rate imaging seems to detect regional fibrosis.