405 Effects of preload alterations on tissue Doppler diastolic myocardial velocities in normal subjects
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Tissue Doppler echocardiography (TDE) is a diagnostic imaging tool that can assess early diastolic tissue velocities (E') which has been proposed as an index of LV relaxation. Recently, animal studies have suggested that E' is preload dependent- this is contradictory to what has been observed clinically in patients with underlying cardiac disease.

METHODS: To determine the effect of preload alterations on E' and early diastolic filling velocity (E) we performed tilt table maneuvers (+90° and ±45° head-up tilt, -6° and -30° head-down tilt) in 8 normal volunteers. Real-time 3D and Doppler echocardiography were performed to determine end-diastolic volume and E. Left ventricular lateral wall (E'lat) and septal (E'sep) velocities were measured by TDE. For each individual, changes in EDV were compared against E'lat, E'sep, and E velocities by linear regression.

RESULTS: Overall, from +90° to -30° EDV increased by 29.1±12.7 ml (EDV=0.22x+113.3 ml/stt, r=0.95). Changes in EDV linearly correlated with E (average correlation: r=0.73±0.18). E'lat and E'sep also correlated with EDV (average correlation: r=0.67±0.25 and r=0.80±0.14 respectively) (see table). Differences in correlations between E'/EDV, E'lat/EDV, and E'sep/EDV were not significant.

RESULTS EDV: End-diastolic volume; E: Early transmitral filling velocity; E'lat and E'sep: Early diastolic tissue velocities measured at the lateral and septal walls. * By repeated measures analysis of variance

<table>
<thead>
<tr>
<th>Tilt Angle (degrees)</th>
<th>-90</th>
<th>-6</th>
<th>45</th>
<th>90</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV (ml)</td>
<td>124.7±15.4</td>
<td>122.3±25.9</td>
<td>101.7±21.5</td>
<td>97.2±22.7</td>
<td>0.05</td>
</tr>
<tr>
<td>E (cm/sec)</td>
<td>65.6±11.5</td>
<td>76.2±8.8</td>
<td>60.1±9.8</td>
<td>58.7±11.5</td>
<td>0.005</td>
</tr>
<tr>
<td>E'lat (cm/sec)</td>
<td>16.4±2.1</td>
<td>16.4±3.6</td>
<td>15.4±1.5</td>
<td>8.9±2.6</td>
<td>0.001</td>
</tr>
<tr>
<td>E'sep (cm/sec)</td>
<td>13.7±1.6</td>
<td>12.3±2.4</td>
<td>8.6±1.2</td>
<td>8.0±1.3</td>
<td>0.002</td>
</tr>
</tbody>
</table>

CONCLUSIONS: In normal subjects, early diastolic myocardial tissue and transmitral filling velocities are preload dependent and correlate with changes in end-diastolic volume.
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Estimation of the left ventricular end-diastolic pressure by the mitral deceleration time of the A wave and by the difference between pulmonary venous and mitral A wave durations:

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The aim of our study was the evaluation of the correlation between the left ventricular end-diastolic pressure (LVEDP) and the mitral deceleration time of the A wave, and the difference in pulmonary venous A wave (Ap) and mitral A wave (Am) durations.

Material: 61 patients (pts), 50 men (82%), mean age 57.05±11.02 years, with coronary disease: stable angina (30.3%) and unstable angina (20.3%). None had evidence of mitral stenosis, severe mitral regurgitation, atrial fibrillation, conduction disturbances such as second- or third-degree heart block or incomplete pulmonary vein Doppler signal.

Methods: The transmural flow variables and the pulmonary vein flow indices were recorded by pulsed Doppler, using a 3.5 MHz transducer. The measurement of LVEDP was done by left heart catheterization. We analyzed the Doppler tracings and the following variables were measured: peak E and A velocities (cm/s), E wave deceleration time (EDt, ms), A wave deceleration time (Adt, ms), E/A ratio; for the pulmonary vein flow: peak S and D velocities (cm/s), Ap velocity and duration, difference between pulmonary vein and mitral A waves durations (Ap-Am, ms). Statistical analysis was performed by linear regression analysis, and standard formulas were used for calculating specificity and sensibility.

Results: The LVEDP values ranged between 4-43 mmHg. We found a very good negative correlation between Am and LVEDP (r=-0.59) in all patients. Thus, Tda EDTA, a modified form of the LVEDP t by index given with a sensitivity of 92% and a specificity of 91%. We found a strong correlation between LVEDP and the difference Am-Am (r=0.58). An increase of the difference Am-Am seems to represent a marked increase of LVEDP4 in 10%, with a sensitivity of 99% and a specificity of 99%. Between the Ap duration and LVEDP there is a good correlation (r=0.74), which is not present between LVEDP and Am (r=0.23).

According to the transmural flow velocities, as expressed in the E/A ratio, the study pts were assigned to the following 3 groups: group 1 with E/A<1, with improved relaxation pattern (20 pts, 40%), group 2 with 1<EA<2 which may signify a normal or "pseudonormal" filling pattern (20 pts, 33%); group 3 with E/A>2 representing "restrictive" filling pattern (11 pts, 16%). In all 3 groups we found a very good correlation between LVEDP and Etd, and the Ap-Am difference respectively.

Conclusions: A shortened A wave deceleration time (Etdms) and an elevated Ap-Am difference (10ms) represent markers for an elevated LVEDP (20-30mmHg). We found a very good correlation between these parameters and LVEDP in all 3 types of mitral diastolic flow patterns.

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Left ventricular systolic function as the main determinant of early transmural flow propagation velocity

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The slope of the color Doppler M-mode early transmural flow propagation velocity (FPV, cm/s) has been experimentally related to left ventricular (LV) relaxation. However, the determinants of FPV and the relation of FPV to LV function have not been studied in the clinical setting. We measured FPV (indexed by BSA) in 131 consecutive patients subdivided as: normals (Norm, 28), coronary artery disease without (CADn, 11) and with (CADa, 14) LV hypokinesia, hypertensive LV hypertrophy (LVH, 18), idiopathic dilated cardiomyopathy (DCM, 20), mitral or aortic regurgitation (Reg, 10), mitral stenosis, severe mitral regurgitation, atrial fibrillation, conduction disturbances such as second- or third-degree heart block or incomplete pulmonary vein Doppler signal.

Methods: The transmural flow variables and the pulmonary vein flow indices were recorded by pulsed Doppler, using a 3.5 MHz transducer. The measurement of LVEDP was done by left heart catheterization. We analysed the Doppler tracings and the following variables were measured: peak E and A velocities (cm/s), E wave deceleration time (EDt, ms), A wave deceleration time (Adt, ms), E/A ratio; for the pulmonary vein flow: peak S and D velocities (cm/s), Ap velocity and duration, difference between pulmonary vein and mitral A waves durations (Ap-Am, ms). Statistical analysis was performed by linear regression analysis, and standard formulas were used for calculating specificity and sensibility.

Results: The LVEDP values ranged between 4-43 mmHg. We found a very good negative correlation between Am and LVEDP (r=-0.59) in all patients. Thus, Tda EDTA, a modified form of the LVEDP t by index given with a sensitivity of 92% and a specificity of 91%. We found a strong correlation between LVEDP and the difference Am-Am (r=0.58). An increase of the difference Am-Am seems to represent a marked increase of LVEDP4 in 10%, with a sensitivity of 99% and a specificity of 99%. Between the Ap duration and LVEDP there is a good correlation (r=0.74), which is not present between LVEDP and Am (r=0.23).

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Conclusions: A shortened A wave deceleration time (Etdms) and an elevated Ap-Am difference (10ms) represent markers for an elevated LVEDP (20-30mmHg). We found a very good correlation between these parameters and LVEDP in all 3 types of mitral diastolic flow patterns.

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Response of the restrictive left ventricle diastolic filling pattern to Valsalva manoeuvre is predictor of events in patients with advanced left ventricle systolic dysfunction

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The aim of this study was to evaluate the predictive value of the response of restrictive transmural inflow flow pattern to standardized Valsalva manoeuvre, in patients with advanced left ventricle systolic dysfunction. Methods: 59 patients (pts) with ischemic and dilated cardiomyopathy and advanced systolic left ventricular dysfunction and EF<30% (mean 23±3), who showed restrictive left ventricle diastolic filling pattern (E/A>2, DT <150 ms) were investigated. Mitral inflow velocities were recorded with sample volume positioned at the leaflet tips, first at the end of normal expiration and then at the end of the strain phase of standardized Valsalva manoeuvre. Results In 44 (75%) patients Valsalva manoeuvre induced decrease of both E and A velocity during straining phase, but the restrictive left ventricle diastolic filling pattern remained unchanged (Restrictive pattern). In the remaining 15 (25%) patients E velocity was decreased and A velocity was either increased (12 patients) or slightly decreased (3 patients) and diastolic filling pattern was changed (E/A<2) and pseudonormalized (improved filling pattern). Patients were followed up for 12±4 months. In patients who showed no reversion of the restrictive filling pattern during Valsalva manoeuvre there were 6 deaths (13.6%) and 13 hospitalization (30%) on account of heart failure worsening. In patients who showed improvement of the diastolic filling pattern during manoeuvre there were 1 death (0.7%,NS) and only 2 Hospitalization (13.3% P<0.05). Conclusion Restrictive filling pattern represents advanced left ventricular diastolic dysfunction. Unimprovement of the restrictive filling pattern in response to preload reduction by Valsalva manoeuvre can detect patients with more adverse outcome.

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