was used to analyze CTDI images. Six basal segments of the left ventricle were evaluated in different modes: biventricular (BIV), LV, biventricular with offset of 40 ms (BIV-40) and intrinsic pacing (BIVL). velocities were obtained from the averaged LV six basal segments and CI was calculated from the velocity profiles as follows: electro-mechanical delay (EMD), hemo-dynamic delay (HD), isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT). The PSV was significantly lower in DCMP than in ICMP (P < 0.01) and higher in LBBB than in ICMP (P = ns). Measures of CI are shown in figure. The HD, IVCT, and IVRT were significantly maintained even later; however, this pattern was especially observed in R patients.

Conclusion: In the overall population the effect of resynchronization therapy on left ventricular reverse remodeling was more pronounced in the mid-term and was maintained even later; however, this pattern was especially observed in R patients.

460 Simplified semiquantitative assessment of asynchrony and resynchronization with color tissue Doppler


Introduction: Color tissue Doppler (CTDI) allows to analyze myocardial velocity and to assess left ventricular (LV) activation sequence. Objective: To identify CTDI patterns for a simplified evaluation of dysynchrony. Material and methods: 11 patients (p) with good clinical response to cardiac resynchronization were analyzed (mean age 65±11 years, 7 p (64%) with dilated cardiomyopathy, ejection fraction 27±6%). The LV lead was implanted in the postero-lateral region in 8 p and in the antero-lateral one in two. QLab (Philips®) application was used to analyze CTDI images. Six basal segments of the left ventricle were studied in different modes: biventricular (BIV), LV, biventricular with offset of 40 msec (BIV-40) pacing and intrinsic rhythm (INT). Asynchrony was defined by the presence of a triphasic pattern in the velocity registry (Mprolonged isovolumetric phase, short systolic period and postystolic movement) as shown in the figure. The presence of the M sign was evaluated in every segment and averaged in every pacing mode. Results: Shown in the table.

<table>
<thead>
<tr>
<th>M6 segments</th>
<th>BIV</th>
<th>LV</th>
<th>BIV-40</th>
<th>INTRINSIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.06±1.28</td>
<td>2.09±1.03</td>
<td>1.45±1.51</td>
<td>1.45±1.81</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>BIV Biventricular pacing; LV: left ventricular pacing; BIV-40: Biventricular pacing with 40 ms of LV preactivation p&lt; 0.01 between intrinsic and biventricular pacing</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusions: A triphasic color tissue Doppler pattern represents a sign of asynchrony and disappear with cardiac resynchronization.

461 Improvement in exercise tolerance after cardiac resynchronization therapy related to efficiency in timing

A. Jansen 1, C.H. Peels 2, F. Bracke 2, A. Meijer 2, J.M. van Dantzig 2. 11 patients (p) with good clinical response to cardiac resynchronization were analyzed (mean age 65±11 years, 7 p (64%) with dilated cardiomyopathy, ejection fraction 27±6%). The LV lead was implanted in the postero-lateral region in 8 p and in the antero-lateral one in two. QLab (Philips®) application was used to analyze CTDI images. Six basal segments of the left ventricle were studied in different modes: biventricular (BIV), LV, biventricular with offset of 40 ms (BIV-40) pacing and intrinsic rhythm (INT). Asynchrony was defined by the presence of a triphasic pattern in the velocity registry (Mprolonged isovolumetric phase, short systolic period and postystolic movement) as shown in the figure. The presence of the M sign was evaluated in every segment and averaged in every pacing mode. Results: Shown in the table.

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Correlation</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV pre vs. 6 WT pre</td>
<td>r=0.36</td>
<td>0.0085</td>
</tr>
<tr>
<td>LVEF pre vs. 6 WT pre</td>
<td>r=0.53</td>
<td>0.01</td>
</tr>
<tr>
<td>FT pre vs. 6 WT pre</td>
<td>r=0.43</td>
<td>0.0015</td>
</tr>
<tr>
<td>LVEF change vs. 6WT increase</td>
<td>r=0.45</td>
<td>0.0001</td>
</tr>
<tr>
<td>FT change vs. 6WT increase</td>
<td>r=0.44</td>
<td>0.0012</td>
</tr>
<tr>
<td>LV pre vs. 6 WT pre</td>
<td>r=0.34</td>
<td>0.0016</td>
</tr>
</tbody>
</table>

Conclusion: The improvement in exercise tolerance with biventricular pacing is related to efficiency in timing. These results re-emphasize the importance of time interval analysis of the cardiac cycle in CRT.

462 Cardiac incoordination and left ventricular function in patients with cardiomyopathy and left bundle branch block

M. Quintana 1, S. Govind 1, S. Saha 1, S. Rounima 2, F. Del Furia 1, L.A. Brodin 2. 111 patients (p) with good clinical response to cardiac resynchronization were analyzed (mean age 65-11 years, 7 p (64%) dilated cardiomyopathy, ejection fraction 27±6%). The LV lead was implanted in the postero-lateral region in 8 p and in the antero-lateral one in two. QLab (Philips®) application was used to analyze CTDI images. Six basal segments of the left ventricle were studied in different modes: biventricular (BIV), LV, biventricular with offset of 40 ms (BIV-40) pacing and intrinsic rhythm (INT). Asynchrony was defined by the presence of a triphasic pattern in the velocity registry (Mprolonged isovolumetric phase, short systolic period and postystolic movement) as shown in the figure. The presence of the M sign was evaluated in every segment and averaged in every pacing mode. Results: Shown in the table.

Conclusions: A triphasic color tissue Doppler pattern represents a sign of asynchrony and disappear with cardiac resynchronization.

464 Dynamic changes of dysynchrony induced by dobutamine are related to both resynchronization and left ventricular functional changes postbiventricular pacing

K. Papadopoulos 1, G. Anthanassopoulos 2, Y. Zhu 1, S. Karagiannis 1, T. Maounis 1, G. Karatsakas 1, E. Leontiadis 1, D.V. Kokkinos 1. 111 patients (p) with good clinical response to cardiac resynchronization were analyzed (mean age 65-11 years, 7 p (64%) dilated cardiomyopathy, ejection fraction 27±6%). The LV lead was implanted in the postero-lateral region in 8 p and in the antero-lateral one in two. QLab (Philips®) application was used to analyze CTDI images. Six basal segments of the left ventricle were studied in different modes: biventricular (BIV), LV, biventricular with offset of 40 ms (BIV-40) pacing and intrinsic rhythm (INT). Asynchrony was defined by the presence of a triphasic pattern in the velocity registry (Mprolonged isovolumetric phase, short systolic period and postystolic movement) as shown in the figure. The presence of the M sign was evaluated in every segment and averaged in every pacing mode. Results: Shown in the table.

Conclusions: CI was worse in DCM than in ICMP and was associated with further impairment of LV function. TVE is a valuable method to quantify CI, to assess LV function and to characterize patients with LBBB and DCM or ICMP.
We assessed the dynamic changes in DYS post DOB and related them to resting regional LV velocities, as well as to LV functional and volume changes post biventricular pacing (BIV).

Methods: 20 consecutive patients (pts) who underwent BIV were studied (age 58±9, NYHA II-IV), ejection fraction: 50±7.

Using Doppler tissue imaging (DTI) time delays (dt: sec) from QRS to peak of systolic velocities were measured at basal (1), mid (2) and apical (3) regions of septal (S) and lateral (L) wall (4 chamber apical view), at baseline (NoPace) and during BIV. The respective delay was also measured at rest (R) and DOB. Time differences (D-dt) between D-dt and R for each region at NoPace and BIV, as well as between BIV and NoPace were calculated.

Results: 1. At NoPace, DOB decreased D-dt compared to R in S1 (p=0.05) and in L3 (p=0.036).

D-dt between DOB/R at NoPace was increased from base to apex of lateral wall (p=0.029), while it was unaltered from base/apical to spical regions of S.

The D-dt at NoPace/BIV at R was related with D-dt at NoPace between DOB/R: L2 (r=0.98, p=0.014), L3 (r=0.68, p=0.004). S2 (r=0.44, p=0.006), S3 (r=0.61, p=0.005).

2. During BIV, the DOB further decreased D-dt homogeneously compared to R: S2 (p=0.02), S3 (p=0.01), L1 (p=0.03, L2:0.04).

Peak Velocities (Vel), strain (S) and strain rate (SR) were measured in these 6 LV walls were measured. Peak velocities at the basal (1), mid (2) and apical (3) regions of septal (S) and lateral (L) wall, using 4 chamber apical view, at baseline (NoPace) and during BIV.

Methods: 20 consecutive patients who underwent BIV were studied (age 58±9, NYHA II-IV), ejection fraction: 50±7.

In Vel: S1: 0.95±0.09, p=0.023, S2: 0.54±0.9, p=0.003, S3: 0.52±0.7, p=0.006.

D-dt between DOB/R at NoPace was increasing from base to apex of lateral wall (L1: -0.089±0.048, L2: -0.019±0.65, L3: -0.018±0.055, p=0.022), where it was unaltered from base/apical to spical regions of S.

During BIV the following increases (%d:) were documented in LV regions.

Results: At NoPace, DOB decreased D-dt compared to R in S1 (0.015±0.034, p=0.05) and in L3 (0.02±0.055, p=0.036).

D-dt between DOB/R at NoPace was increasing from base to apex of lateral wall (L1: -0.089±0.048, L2: -0.019±0.65, L3: -0.018±0.055, p=0.022), where it was unaltered from base/apical to spical regions of S.

During BIV the following increases (%d:) were documented in LV regions.

Conclusion: 1. Delay of the peak of LV longitudinal systolic Vel was decreased during DOB in basal septal and distal lateral wall.

2. Improvement in LV function and volume post BIV is related with dynamic DOB improvement before BIV in both septal and lateral regions.

464 Left ventricular longitudinal systolic function improvement post biventricular pacing: relationship with changes of dysynchrony induced by low dose dobutamine before pacing

K. Papadopoulou 1, G. Athanassopoulos 2, Y. Zhu 3, S. Karagiannis 4, T. Maounis 1, G. Karalasakis 1, E. Leontiades 1, D.V. Cokkinos 1, A. Martiniello 1, E. Case 2, C. Cioppa 2, A. D’Andrea 5, L. Sanlucare 5, A.R. Martiniello 1, S. Case 2, C. Cioppa 2, A. D’Andrea 5, L. Sanlucare 5

Introduction: Left ventricular (LV) dyssynchrony (DYS) is an evolving heart failure (HF) treatment. Evaluation of LV dyssynchrony is based upon resting time delays of peak systolic velocities and there are no data about dyssynchrony post dobutamine (DOB). We assessed the dynamic changes of regional time delays post DOB and related them to LV longitudinal function by Doppler tissue imaging (DTI) post BIV.

Methods: 20 consecutive patients who underwent BIV were studied (age 58±9, NYHA II-IV), ejection fraction: 50±7.

Using DTI, time delays (dt: sec) from QRS to the peak of systolic velocities were measured at the basal (1), mid (2) and apical (3) regions of septal (S) and lateral (L) wall, using 4 chamber apical view, at baseline (NoPace) and during BIV.

The respective dt (sec) was measured at rest (R) and post low dose (5min stages of 5 and 10tg/kg/min) DOB.

Time differences (D-dt) between DOB/R and R for each region at NoPace and BIV, as well as between BIV and NoPace were calculated.

Peak Velocities (Vel), strain (S) and strain rate (SR) were measured in these 6 LV regions.

Results: At NoPace, DOB decreased D-dt compared to R in S1 (0.015±0.034, p=0.05) and in L3 (0.02±0.055, p=0.036).

D-dt between DOB/R at NoPace was increasing from base to apex of lateral wall (L1: -0.089±0.048, L2: -0.019±0.65, L3: -0.018±0.055, p=0.022), where it was unaltered from base/apical to spical regions of S.

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Conclusion: 1. At NoPace, DOB decreased D-dt compared to R in S1 (p=0.05) and in L3 (p=0.036).

D-dt between DOB/R at NoPace was increased from base to apex of lateral wall (l1: -0.089±0.048, L2: -0.019±0.65, L3: -0.018±0.055, p=0.022), where it was unaltered from base/apical to spical regions of S.

During BIV the following increases (%d:) were documented in LV regions.