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Application of tissue tracking and dobutamine stress echocardiography in the diagnosis of coronary artery disease.

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Objective: The combination of tissue tracking technique and Dobutamine stress echocardiography were studied to evaluate the ischemic regions in patients with coronary artery disease (CAD).

Method: A total of 25 patients with suspected CAD underwent dobutamine stress echocardiography, and the systolic mitral annular displacement (MAD) was deter-

mined at rest and during stress by tissue tracking technique. Apical four cham-
ber, three chamber and two chamber views were used to determine the MAD at 6 sites (post interventional septum PIVS, anterior interventional septum AIVS, anterior ANT, lateral LAT, posterior POST and inferior INF). Coronary arteriography was per-
formed within 1 week after echocardiographic examination. All patients were divided into two groups according to the result of coronary arteriography. Group A included 23 patients with more than 70% stenosis in left anterior descending coronary artery (LAD). Group B consisted of 27 patients with no significant stenosis of LAD.

Result: (1) The systolic MAD at rest 10ug/kg/min, 20ug/kg/min, 30ug/kg/min were not significantly different between Group A and group B (P>0.05), but at 40ug/kg/min, the systolic MAD in ANT in group A was lower than that in group B (P<0.01). The systolic MAD at other sites was not significantly different between the group A and group B (P>0.05). (2) The systolic MAD of group A in ANT and AIVS during stress were not significantly different from that at rest (P>0.05). However it was higher in POST and LAT at 30ug/kg/min, 40ug/kg/min, and in INF and PIVS at 20ug/kg/min, 30ug/kg/min, 40ug/kg/min than that at rest in group A (P<0.005-0.01). The systolic MAD at the 6 sites during stress was higher than that at rest in Group B (P<0.01).

Conclusions: Tissue tracking imaging combined with dobutamine stress echocardiography can early and accurately detect abnormal mitral annular displacement in patients with coronary artery disease.

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Application of a novel non tissue doppler based method for real-time quantitative assessment of myocardial function in normal subjects during exercise echocardiography.

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Objectives: To assess the feasibility of a novel software for real-time quantitative assessment of myocardial function in normal subjects during exercise echocardiography.

Background: Reliable methods for quantitative assessment of myocardial function for stress echocardiography are limited.

Methods: 12 patients underwent standard exercise echocardiography. Apical views at baseline and peak exercise were stored in a cineloop format for off-line analysis. The novel software is based on the estimation that a discrete set of tissue velocities per each of many small elements on ultrasound image show only mild shift on subsequent frames. Tracking can be controlled in real-time by the operator. Tis-

sue velocities, strain and strain rate at baseline and at peak exercise were obtained and displayed in real time by the software. We also introduced a new parameter: Strain acceleration index - the ratio of systolic strain and time to peak systolic strain corrected for heart rate.

Results: 216 myocardial segments were assessed. Adequate tracking of the myo-

cardium by the new software was possible in 93% of the segments at rest and in 80% at peak exercise. Velocities were maximal in basal segments. Strain was homogenous over the myo-

cardium. Velocities, strain and strain rate were significantly higher at peak exercise. Corrected time to the peak systolic strain was shorter at peak exercise than at rest; strain acceleration index was higher at peak exercise than at rest (Table).

Quantitative parameters during exercise

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before exercise</th>
<th>Post exercise</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal velocities (cm/sec)</td>
<td>4.2 ± 1.3</td>
<td>5.28 ± 1.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Apical velocities (cm/sec)</td>
<td>1.87 ± 0.93</td>
<td>2.34 ± 1.43</td>
<td>0.03</td>
</tr>
<tr>
<td>Strain rate (sec⁻¹)</td>
<td>1.02 ± 0.39</td>
<td>1.43 ± 0.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Strain (%)</td>
<td>16.71 ± 5.06</td>
<td>18.09 ± 6.23</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Time to peak strain (CU)</td>
<td>11.4 ± 5.23</td>
<td>14.94 ± 9.69</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SA (%) (CU)</td>
<td>1.42 ± 0.63</td>
<td>1.88 ± 0.97</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

CU - corrected units (corrected for heart rate), SA - strain acceleration index=strain/time to peak strain (%CU).

Conclusion: This novel non-Doppler based software may provide real-time quan-
titative assessment of global and regional myocardial function at rest and during exercise echocardiography.

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Dobutamine versus levosimendan stress echocardiography for the prediction of recovery of left ventricular dysysnergies after revascularization.

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Objectives: To compare the accuracy of levosimendan (L) and dobutamine stress echocardiography (DSE) for the prediction of recovery of left ventricular dysysnergies after revascularization.

Methods: Twenty eight patients with left ventricular dysfunction due to previous myocardial infarction scheduled for revascularization (18 PTCA and 10 CABG) un-

derwent low-dose DSE (5-10ug/kg/min) and LSE. Levosimendan was infused at least 1h after dobutamine infusion, at 2 doses of 12 and 24ug/kg, over a 5 min period each. Left ventricular wall motion score was assessed using a 16-segment model. Myocardial viability was detected if improvement of > =1 grade of regional wall motion score in at least two contiguous segments was noted, during either dobutamine or levosimendan infusion. All patients also underwent resting echocardi-

ography within 6 months after successful revascularization.

Results: Of the 448 segments studied, 212 (47%) was dysysnergic at rest. Dobo-

tamime infusion resulted in augmented contraction in 98/212 (46%) abnormal seg-
ments while 88/90(90%) of these showed functional improvement after revasculariza-

tion. During LSE 110/220(52%) dysysnergic segments improved and 100/91(91%) of these recovered function after revascularization. Analysis of results showed a sig-
ificantly lower sensitivity of LSE compared with DSE (73% vs 94%, respectively, p<0.01) but a similar specificity (89% vs 80%, respectively, p=ns) for the prediction of postrevascularization recovery of left ventricular dysysnergies.

Conclusions: LSE can predict postrevascularization recovery of left ventricular dysysnergies with higher accuracy than DSE.

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Evidence for interplay between cytokines, macrophage colony stimulating factor and brain natriuretic peptide plasma levels changes during dobutamine stress echo irrespectively from test positivity.

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Introduction: Cytokins (interleukins - IL, tumour necrosis factor - TNF) and macro-

phage colony stimulating factor (MCSF) plasma levels are mediators in pathophys-

iology of acute coronary syndromes. Brain natriuretic peptide (BNP) is produced due to increased cardiac wall stress and implies left ventricular (LV) dysfunction. Dobo-

tamine stress echo (DSE) may induce acute ischemia and ventricular dysfunction.

Aim of the study was to assess dynamics of these parameters during DSE and their potential interrelationships.

Methods: We studied 75 consecutive pts by DSE (age 60±11, 10 women, with ejection fraction EF 40±11%, a previous myocardial infarction documented in 26). The IL1, tumour necrosis factor (TNF), IL6, MCSF were measured at rest (R), peak stress and during recovery, 15min post DSE (Rec). BNP was measured at R and Rec. Interleukin 1 (IL1) and tumour necrosis factor (TNF) were measured in a subgroup of 11 consecutive pts. A 16 segments model was used for DSE analysis.

Results: BNP at Rec had strong relationship with BNP at R (r=0.90, p<0.0001), but both absolute and the % changes were independent from its R values.

The BNP changes had no relationship with heart rate-blood pressure product changes.

The % changes of BNP was related to peak IL6 (r=0.25, p=0.02), recovery MCSF (r=0.29, p=0.036) and at R (r=0.29, p=0.023). TNF at Rec had close relationship with both absolute and % BNP (r=0.53, p<0.05, r=-0.74, p=0.016 respectively).

A similar trend was found between IL1 and %BNP (R: r=-0.50, p=0.06, Rec: r=-0.52, p=0.06).

When pts with ischemic DSE were analyzed separately, then BNP Rec was corre-

lated exclusively with MCSF at R (r=0.33, p=0.02) and at R (r=0.44, p=0.02).

In contrast when pts without an ischemic DSE response were analysed, then both absolute and % BNP changes were related with IL6 at R and P (IL6 R/P: r=0.54, p=0.038 for both and r=-0.47, p=0.07, r=-0.56, p=0.034 respectively).

Conclusions: DSE is related with changes in both BNP and inflammatory indices irrespectively from the detectable wall motion abnormalities.

Even in the absence of an ischemic DSE response, an increase of IL6, an inflam-

matory marker, is related to an increase of BNP thus implying subtle LV function.

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