MYOCARDIAL VELOCITY IMAGING (DMI) – OTHER

372 Strain rate imaging can help to identify patients with right ventricular infarction

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Background: This study was planned to assess whether strain rate (SR) and strain (S) echocardiography is a useful method for the detection of right ventricular myocardial infarction.

Methods: Thirty patients (15 with right ventricular (RV) infarction, 15 without RV infarction) with acute inferior myocardial infarction were included in this study. The presence of right ventricular infarction was defined as an ST-segment elevation 0.1 mV in lead V4 R. Echocardiography was performed using a Vivid 5 System and a 2.5-MHz transducer. 2-dimensional color doppler myocardial imaging (CDMI) data for longitudinal function were recorded from the RV free wall using standard apical fourchamber view. Longitudinal systolic tissue velocities (V), strain rate (Sr) and strain (S) were postprocessed from basal, mid, and apical segments of RV interrogated using apical fourchamber view. Peak systolic strain rate were estimated by measuring the spatial velocity gradient over a computation area of 10 mm in the longitudinal. To derive systolic tissue velocity and strain rate profiles from a segment, the region of interest was maintained in a constant position within the segment being interrogated by using a semi-automatic tracking algorithm. The timing of end-systole (pulmonary valve closure) and end-diastole (onset of isovolumic contraction) of right ventricle were derived using a myocardial tissue velocity profile. Natural strain profiles were obtained by integrating the strain rate values over time using end-diastole as the reference point.

Results: Systolic tissue velocity, strain, strain rate of basal (4.8±0.8 cm/s vs 6.5±1.2 cm/s, -12.3% vs -24.5%, -1.2±0.3/s vs -1.9±0.4/s; p<0.001, p<0.001, p<0.001, respectively) and mid (4.2±0.5 cm/s vs 5.4±0.5 cm/s, -16±3% vs -26±4%, -1.2±0.3/s vs -2.1±0.3/s; p<0.001, p<0.001, p<0.001, respectively) segments of right ventricle was significantly lower in patients with right ventricular infarction than in patients without right ventricular infarction. No there were differences between groups as regards apical strain, strain rate and systolic tissue velocity.

Conclusion: The present study demonstrates that strain and strain rate imaging is simple and can be used to distinguish patients with inferior myocardial infarction from those with or without right ventricular infarction.

MYOCARDIAL VELOCITY IMAGING (DMI) – LV FUNCTION

373 Circumferential, radial and longitudinal strain variations at rest and during dobutamine infusion: application of 2D strain for myocardial ischemia detection

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Background: Routine evaluation of regional function by visual wall-motion assessment is unsatisfactory. Bidimensional (2D) strain has great promise to improve objective quantification of regional function abnormalities during stress echocardiography.

Objectives: To evaluate the role of circumferential, radial and longitudinal strains during dobutamine stress echocardiography in detecting myocardial ischemia.

Methods: 10 open-chest pigs were studied at baseline and at 4 stages of ischemia: mild and moderate LAD stenoses (non flow limiting stenosis (NFLS)) at rest, but decreasing hyperemia by 30% and 50%, respectively), severe stenoses (flow limiting stenosis (FLS)) at rest by 30% and 50%, respectively, at rest and during dobutamine infusion. Segmental left ventricular function was assessed by measurement of circumferential, radial, and longitudinal strains (CS, RS, LS) and was compared to sonomicrometry in the risk area (RA) and in a control area (CA).

Results: At baseline, the 3 components of strains were significantly increased from rest to dobutamine stages. At rest and compared to baseline, CS was significantly decreased in presence of 30% NFLS whereas LS was significantly decreased for 30% FLS and LS for 50% FLS (p<NS) in risk area (anterior and antero-septal wall). During dobutamine, CS was significantly decreased in presence of 30% NFLS and LS was decreased for 50% NFLS. RS was significantly reduced only for 50% FLS.

Conclusions: 2D strain analysis of regional myocardial function demonstrated marked differences in strain variations during myocardial ischemia from rest todobutamine. Circumferential strain appears to be more sensitive than longitudinal or radial strain for ischemia detection.

Table 1. Function analysis by 2D strain

<table>
<thead>
<tr>
<th>Component</th>
<th>Strain</th>
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<tbody>
<tr>
<td>Baseline</td>
<td>-24±7.1</td>
<td>-27.4±2.2</td>
<td>64.8±5.7</td>
<td>74.7±3.7</td>
<td>18.6±2.9</td>
<td>20.3±2.1</td>
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<tr>
<td>30% NFLS</td>
<td>-21.4±2.8</td>
<td>-23.4±2.4</td>
<td>63.4±7.1</td>
<td>73.9±4.1</td>
<td>16.1±2.9</td>
<td>19.9±2.4</td>
</tr>
<tr>
<td>50% NFLS</td>
<td>-16.3±3.0</td>
<td>-20.4±2.2</td>
<td>62.9±9.5</td>
<td>69.6±9.2</td>
<td>16.6±2.8</td>
<td>17.7±2.2</td>
</tr>
<tr>
<td>30% FLS</td>
<td>-16.1±1.5</td>
<td>-20.4±2.2</td>
<td>58.6±15.7</td>
<td>61.1±19.0</td>
<td>13.2±17.7</td>
<td>14.0±11.1</td>
</tr>
<tr>
<td>50% FLS</td>
<td>-12±4.6</td>
<td>-15±4.6</td>
<td>51±14.8</td>
<td>46.6±16.7</td>
<td>-19±3.2</td>
<td>-12±1.8</td>
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</table>

RA: risk area, NFLS: non flow limiting stenosis, FLS: flow limiting stenosis

374 Strain rate accurately differentiates stunned from necrotic myocardium in a rabbit model of ischemia-reperfusion

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Objective: To investigate whether strain rate (SR) imaging could reliably differentiate stunned from necrotic myocardium in a rabbit model of ischemia-reperfusion (IR).

Methods: 35 male New Zealand White rabbits underwent a 30-minute ligation of the 1st marginal branch of the LAD coronary artery followed by 72 hours of reperfusion. Echocardiography (8 MHz, Vivid 7, GE) was performed at baseline and at reperfusion (30 minutes and 72 hours). Strain rate imaging (SR, sec⁻¹) was obtained from the mid short-axis view in the posterior (PW) and the anterior walls (AW) (SR length: 2 mm). At 72 hours, the coronary artery was re-oclculated and blue dye was injected to assess the area of risk size (AR). Infarct size (AN) was determined by TTC staining.

Results: Pre-operative mortality was estimated at 30% and 24 animals were still alive at 72 hours of reperfusion. Blue dye localized the AR within the PW and the apex in all the animals. At the level of the papillary muscles, TTC staining could identify 2 groups according to the presence of necrosis within the AR: 13 rabbits had necrosis (group A) and 11 rabbits had no necrosis.
Remote ischemic preconditioning reduces ischemic left ventricular dysfunction during dobutamine stress echocardiography

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Introduction: The protective benefits of remote ischemic preconditioning (rIPC) have been demonstrated in animal models of ischemia, but the clinical application in humans has not been extensively studied. We hypothesized that rIPC would protect the left ventricle from stunning during dobutamine stress echocardiography (DSE) in patients with coronary artery disease.

Methods: 12 patients with a single coronary stenosis and normal left ventricular (LV) function underwent two standard DSEs, one week apart. Patients were randomized to rIPC 30 minutes before either the first or second DSE. rIPC was administered by upper arm cuff inflation to supra-systolic pressures for 3 five-minute cycles, with 5 minutes of reperfusion in between. Digital images were acquired at each stage using a Vivid 7 ultrasound machine. Images were obtained from standard apical 2 and 4 chamber views. Tissue Doppler data were analyzed offline using EchoPAC software and isovolumetric acceleration (IAV), peak systolic (s-wave), and e-wave velocities recorded.

Results: Systolic and diastolic velocities at matched dobutamine dose were significantly improved following rIPC, as measured by segmental peak velocities of the s-wave (mean (cm.s-1) ± SEM; control 7.03±0.24, rIPC 6.41±0.24, p=0.01), and e-wave (mean (cm.s-1) ± SEM; control 4.03±0.20, rIPC 5.06±0.2, p<0.01). Baseline velocities did not differ between the two groups.

Conclusion: Remote ischemic preconditioning prior to dobutamine stress protects the left ventricle from ischemic myocardial dysfunction.

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