Wall Thickening Assessment With Tissue Harmonic Echocardiography Results in Improved Risk Stratification for Patients With Non-ST-Segment Elevation Acute Chest Pain

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Background: The objective of this study was to demonstrate whether the improved imaging quality gained by using tissue harmonic echocardiography (THE) results in the improved risk stratification of patients presenting with non-ST-elevation acute chest pain. THE allows acquisition of higher frame rates (up to 150Hz) and higher temporal and velocity resolution, which is important in assessing heart function and tissue. Furthermore, THE provides better signal-to-noise ratio, which is useful in assessing myocardial mass. THE also reduces artifacts, which are often present in fundamental echocardiography (FE).

Methods: Eighty consenting patients with over 30 minutes of non-ST-elevation chest pain that had lasted less than six hours were recruited. All patients were assessed and underwent resting THE and FE scans. Diagnosis of index acute myocardial infarction (AMI) was made on a 24 hour creative kinase-MB sample. Echocardiographic images were reviewed by two experienced blinded observers using a 14 segment model. Patients were divided into 2 groups according to wall thickening abnormality demonstrated with FE. The optimal cutoff value for DDTLAD was 730ms for DDTLAD (sensitivity 94%, specificity 60%) and 665ms for DDTmyo (sensitivity 66%, specificity 80%).

Results: DDTLAD was significantly shorter in patients with AMI compared to healthy controls (p<0.001). DDTLAD of healthy controls was 852 ± 6 (normoxia) vs 768 ± 309 ms (p<0.001). DDTLAD of healthy controls was 852 ± 6 (normoxia) vs 768 ± 309 ms (p<0.001).

Conclusions: Tissue harmonic echocardiography is superior to fundamental echocardiography for accurate assessment of systolic wall thickening and hence risk stratification for patients presenting with acute chest pain and non-diagnosable ECG changes.

Tissue harmonic echocardiography results in improved risk stratification for patients with non-ST-segment elevation acute chest pain.

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Tuesday, April 01, 2003, 2:00 p.m.-3:30 p.m.
McCormick Place, Room S104

Effects of ACAT Inhibition by Piolecan Emission Tomography Measured Myocardial Blood Flow: A Double-Blind, Randomized, Multicenter Trial

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Purpose: Acyclic adenosine-5'-cyclic monophosphate (ACAT) inhibitor has demonstrated anti-atherosclerotic properties in animals. To determine whether acyclic adenosine 5'-cyclic monophosphate (ACAT) inhibitor affects the myocardial regional flow reserve (rMFR) in patients with documented or at risk for coronary artery disease (CAD). METHODS: 327 patients (age 17 to 75 years; 101 female) with a rMFR < 2.5 were randomized to placebo plus atorvastatin 10mg (atova) or avasimibe 50 mg QD plus atorvastatin 10mg QD (ava50, combination). PET measured myocardial blood flow rates at rest and during adenosine stress at baseline and at 54 weeks to determine rMFR pre and post treatment. Results: At baseline, rMFR was similar in the three groups (1.62±0.05 SE; 1.58±0.06 SE; 1.57±0.06 SE; p=NS). At 54 weeks, rMFR had improved by 0.27±0.11 (rMFR) for atova, by 0.40±0.15 for avasimibe and by 0.49±0.11 for avasimibe combination therapy. The incremental, treatment-related rMFR differences (over control) of 0.13 and 0.22 between avasimibe and avasimibe-combination and the atova control group, while favoring avasimibe, were statistically not significant (p=0.06 and p=0.13). Defining a ≥ 20% rMFR increase as a positive response, more patients responded to avasimibe and to avasimibe-combination than to atova (36% and 44%; respectively vs. 27%; p=0.01 avasimibe vs. atova). Conclusions: The observed trend of a greater rMFR increase in the combination arm in a subset of patients, though statistically not significant, together with a significantly higher incidence of positive rMFR responses in the avasimibe treatment group suggest a possible "beneficial" anti-atherosclerotic effect of avasimibe in patients with documented or at risk for CAD.

Transatheloreal Doppler Echocardiographic Assessment of Intramyocardial Artery Flow and Coronary Flow Velocity Pattern Predict Wall Motion Recovery Following Acute Myocardial Infarction

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Background: Previous study using a Doppler guide wire has reported that coronary flow velocity pattern (CFV) with a rapid diastolic deceleration time (DDT) immediately after AMI nor for an adverse cardiac event during follow up. CFV of intramyocardial artery (DDTmyo) provides some information on intramyocardial artery in the infarcted area and wall motion recovery following AMI. Methods: CFV of intramyocardial artery in the infarcted area and wall motion recovery following AMI. Results: Cross-correlation of DDTLAD and maximal diastolic flow velocity (MDV) significantly correlated with FE (r=0.631, p=0.0001), respectively. The optimal cutoff value was 730ms for DDTLAD (sensitivity 94%, specificity 60%) and 665ms for DDTmyo (sensitivity 66%, specificity 80%). DDTLAD was significantly shorter in patients with AMI compared to healthy controls (p<0.001). DDTLAD of healthy controls was 852 ± 6 (normoxia) vs 768 ± 309 ms (p<0.001).

Conclusions: Tissue harmonic echocardiography is superior to fundamental echocardiography for accurate assessment of systolic wall thickening and hence risk stratification for patients presenting with acute chest pain and non-diagnosable ECG changes.

Caffeine Decreases Coronary Flow Reserve at Exercise in Healthy Volunteers

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Background: Caffeine antagonizes adenosine-induced hyperemic myocardial blood flow (MBF). Its effect on exercise-induced coronary flow reserve (CFR) remains unknown. Aim: To determine the acute effect of caffeine on CFR during bicycle exercise at baseline and during exposure to simulated altitude. Methods: 150 labeled H2O and Positron Emission Tomography (PET) was used to measure CFR in 16 healthy volunteers (mean age 28 ± 1y) before and after 50 minutes after oral ingestion of caffeine (250mg). Supine bicycle exercise (mean workload 154 Watts, 100% of predicted) was used as stress. Measurements were performed at normoxia (n=6) or during simulation of an altitude of 4500m by inhalation of 12.5% oxygen in N2 (n=6). Results: Caffeine levels were zero at baseline in all subjects and increased to 14.7 ± 2 mmol/minute after caffeine intake. Rate pressure product (rest and exercise) was not affected by caffeine. Caffeine decreased resting MBF significantly by 21±18% (normoxia, p=0.0005) and 35±15% (hypoxia), whereas hyperemic MBF decreased significantly by 17±18% (normoxia, p=0.0005) and 25±11% (hypoxia, p=0.0005), resulting in a decrease in CFR of 24% (normoxia) and 47% (hypoxia). Conclusions: Caffeine decreases CFR in healthy volunteers under both hypoxic conditions.