otherwise healthy. They were studied before and during an infusion of Intralipid and heparin to increase their plasma FA levels (nmol/mL). MFAU, MFAO, MFAE (all in nmol/g/min) and % MFAO were measured with PET and 1-^{11}C-palmitate using well-validated mathematical modeling of the time-activity curves. Paired t-tests were used in the analysis below.
Results: MFAU, MFAO, and MFAE were all higher post-Intralipid. Percent MFAO trended towards being lower post-Intralipid (Table).
Conclusions: Obese women can further increase myocardial FA metabolism in response to increased plasma FA levels, although the capacity to oxidize extracted FA (% MFAO) may be exceeded. These results parallel those of animal models of obesity and support the theory that increased FA delivery increases myocardial FA metabolism. Future studies are needed to determine whether this increase in myocardial FA metabolism in humans contributes to cardiac dysfunction.

	FA	MFAU	MFAO	MFAE	% MFAO
Pre-intralipid	591 ± 179	203 ± 74	182 ± 61	34 ± 38	91 ± 9
Post-intralipid	2118 ± 727	663 ± 216	478 ± 189	247 ± 151	67 ± 17
p value	0.003	0.005	0.02	0.02	0.05

POSTER SESSION

1092 Doppler Imaging of Myocardium

Monday, March 08, 2004, Noon-2:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1092-161 Ischemic Mitral Regurgitation Is Independent of Papillary Muscle Dysfunction: Insights From Tissue Doppler Strain Imaging in Patients With Inferior Myocardial Infarction

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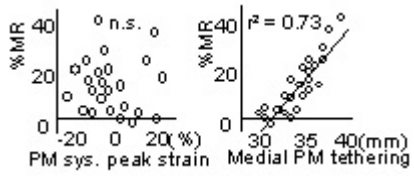
Background: Although the relation between papillary muscle (PM) dysfunction and ischemic MR is controversial, PM dysfunction could be associated with less mitral regurgitation (MR) because of PM elongation, attenuating leaflet tethering. However, associated and variable degree of left ventricular (LV) remodeling to exaggerate leaflet tethering can potentially modify effects of PM dysfunction on the leaflet function.
Method: In 24 patients with prior inferior infarction, LV volume, ejection fraction, mitral annular area, PM tethering distance (PM to annulus), longitudinal PM peak systolic strain and MR fraction were quantified by 2-dimensional and Doppler echocardiography.
Results: 1) MR was not significantly correlated with longitudinal PM peak systolic strain. 2) MR was correlated with LV and mitral annular dilatation, and increased tethering, with the strongest independent contribution by multivariate analysis from increased medial PM tethering.
Conclusions: Ischemic MR is independent of PM dysfunction but is related to geometric changes with medial PM tethering from LV remodeling in patients with inferior MI.



1092-162 Myocardial Strain and Displacement Following Cardiac Resynchronization Therapy

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Background: Cardiac Resynchronization Therapy (CRT) improves left ventricular (LV) function in heart failure patients. Tissue Doppler imaging (TDI) measurements of strain and displacement, may help clarify the mechanisms of LV improvement and remodeling.
Methods: We enrolled forty-five patients with various etiologies of heart failure, a QRS duration ≥ 120ms and EF ≤ 35%. Baseline and 3 months testing included 2-D echocardiogram (GE, Vivid 7) with TDI to assess strain and displacement. From the apical 4, 3, and 2 chamber views, strain and displacement were recorded at the base, mid and apical segments of 6 LV regions at the closure of the aortic valve. Using standard techniques, we calculated the ejection fraction (EF), mitral regurgitation area (MR), end systolic (ESD) and diastolic dimensions (EDD), and end systolic (ESV) and end diastolic volumes (EDV).
Results: CRT improved EF (23.6% ± 9.1 vs. 34.1% ± 12.8, p<0.01), decreased MR (7.4 cm^2 ± 5.6 vs. 4.2 cm^2 ± 3.3, p<0.01), ESD (5.8 cm ± 1.3 vs. 5.3 cm ± 1.6, p<0.01), EDD (6.7 cm ± 1.2 vs. 6.4 cm ± 1.5, p=0.05) and ESV (121.5 ml ± 56.7 vs. 102.1 ml ± 61.0, p<0.01). EDV did not change (158.1 ml ± 59.0 vs. 147.0 ml ± 66.8, p=0.11) but showed a trend towards improvement. Strain and displacement did not consistently improve after CRT. Table.
Conclusions: Despite improvements LV function and remodeling by 2-D echo measures, strain and displacement do not change after 3 months of CRT and do not give further information about LV mechanics.

Segment	N	Strain at AVC (%)			Myocardial displacement at AVC (mm)		
		Baseline	3M	p-value	Baseline	3M	p-value
IS-Base	43	-5.7 ± 8.0	-10.6 ± 10.2	0.01	2.3 ± 2.5	3.0 ± 2.6	0.08
IS-Mid	42	-6.1 ± 8.0	-3.7 ± 8.1	NS	1.5 ± 2.3	1.5 ± 2.6	NS
IS-Apex	38	-2.3 ± 4.3	-4.3 ± 8.1	NS	0.8 ± 2.0	0.3 ± 1.3	NS
Lat-Base	44	-6.6 ± 10.1	-7.4 ± 12.9	NS	3.0 ± 2.3	3.8 ± 2.6	0.05
Lat-Mid	38	-6.3 ± 7.9	-2.3 ± 7.2	0.02	1.3 ± 1.9	1.8 ± 2.2	NS
Lat-Apex	29	-3.8 ± 7.9	-4.8 ± 7.5	NS	0.1 ± 1.5	1.4 ± 2.1	0.001
Inf-Base	43	-6.4 ± 9.5	-7.7 ± 13.2	NS	3.2 ± 2.9	4.9 ± 2.9	<0.001
Inf-Mid	41	-3.1 ± 6.4	-4.8 ± 7.7	NS	1.9 ± 2.7	2.2 ± 2.5	NS
Inf-Apex	32	-5.6 ± 6.9	-6.2 ± 7.6	NS	1.2 ± 1.9	1.0 ± 1.7	NS
Ant-Base	40	-8.5 ± 9.4	-10.1 ± 12	NS	2.4 ± 1.6	3.4 ± 2.1	0.002
Ant-Mid	29	-2.1 ± 7.0	-5.3 ± 9.7	0.08	1.0 ± 1.2	1.6 ± 1.4	NS
Ant-Apex	19	-2.1 ± 2.6	-6.3 ± 8.9	0.05	0.2 ± 0.6	0.8 ± 1.1	0.04
Pos-Base	29	-6.7 ± 12.6	-9.3 ± 16.3	NS	3.6 ± 2.5	4.0 ± 2.5	NS
Pos-Mid	28	-5.5 ± 5.7	-6.3 ± 7.4	NS	1.7 ± 1.9	2.1 ± 2.0	NS
Pos-Apex	23	-4.9 ± 7.9	-4.0 ± 8.5	NS	0.5 ± 1.6	1.0 ± 1.5	NS
AS-Base	22	-3.8 ± 7.4	-4.9 ± 8.7	NS	1.6 ± 1.5	2.3 ± 1.7	0.08
AS-Mid	16	-2.2 ± 4.7	-4.9 + 5.5	0.05	1.0 ± 1.8	0.8 ± 0.9	NS
AS-Apex	5	-4.5 ± 5.9	3.6 ± 3.9	0.06	0.3 ± 0.8	-0.3 ± 0.8	0.06

AVC = Aortic valve closure, 3M = 3 month, IS = Inferior Septum, Lat = Lateral, Inf = Inferior, Ant = Anterior, Pos = Posterior, AS = Anteroseptal.

1092-163 Tissue Doppler Analysis of Nonuniform Transmural Recovery of Myocardial Contractility in Cardiac Raynaud's Phenomenon in Sclerodermic Cardiomyopathy

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Background: Cardiac Raynaud's phenomenon (C-Raynaud) is reversible myocardial ischemic change introduced by cold provocation and is often detected in progressive systemic sclerosis (PSS). This study investigated transmural differences of myocardial contractile recovery in cardiac Raynaud's phenomenon using tissue Doppler analysis. Methods: We studied 21 PSS patients with C-Raynaud and induced C-Raynaud by cold provocation. Using tissue velocity imaging, peak systolic myocardial velocities (PVs) were measured in inner, mid, and outer layers of left ventricular wall at base line, during and after cold provocation. Results: Under baseline, PVs was significantly greater in inner layer than mid and outer layers. During cold provocation all patients had transmural hypokinesis exhibiting reduced PVs in all layers. At 10 minutes after cold provocation, increase of PVs was significantly depressed in inner layer compared with mid and outer lyers. At 20 minutes after cold provocarion, PVs in mid and outer layers recoverd to base line, whereas PVs in inner layer was still depressed. At 40 minutes after cold provocation PVs in inner layer recovered to base line. Conclusion: This study demonstrates nonuniform transmural recovery of myocardial contractility in C-Raynaud using tissue Doppler analysis and indicates that recovery of myocardial function after transient ischemia is slower in inner layer than outer layer.

1092-164 Echocardiographic Tissue Synchronization Imaging Predicts Acute Response to Biventricular Pacing Therapy

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Background: Cardiac resynchronization therapy (CRT) can be beneficial to patients with heart failure (HF) and abnormal electrical activation. However, prospective identification of patient responders to CRT remains unclear. Our objective was to test the hypothesis that the novel color-coding system with tissue Doppler, tissue synchronization imaging (TSI), may assist prediction of patients with acute response to CRT.
Methods: Twenty-nine HF patients (age 65 ± 10 yrs, ejection fraction 26 ± 6%, QRS duration 175 ± 34 ms) were studied before and < 48 hrs after CRT with Vivid 5 or 7 (GE Vingmed). TSI consisted of real time color-coding of time-to-peak velocities detected automatically: green (20-100 ms), yellow (100-300 ms) through red (300-500 ms) in default. Tissue velocity plots of basal and mid LV segments from 3 apical views were assessed after averaging for 3 cardiac cycles.
Results: Fifteen patients (52%) had an acute response to CRT defined as a ≥15% increase in LV stroke volume. Defferences in baseline TSI time-to-peak velocity between opposing LV walls were greater in responders than non-responders: 120 ± 148 vs. 35 ± 153 ms (p<0.05). A ≥ 65 ms delay from anteroseptum to posterior wall from the apical long-axis view had 87% sensitivity and 100% specificity for predicting acute response.
Conclusion: TSI quantified LV dyssynchrony which was associated with acute response