surgery, but systolic function is preserved. Strain rate imaging combined with tissue Doppler imaging may be a feasible method to evaluate regional systolic left ventricular function with apparently abnormal wall motion.

1140-50 **Atorvastatin Improves Myocardial Contractility in Moderately Hypercholesterolemic Patients as Assessed by Tissue Doppler Imaging During Dobutamine Stress Echocardiography**

Manolis Bountioukos, Don Poldermans, Jeroen J. Bax, Vittoria Rizzello, Eleni C. Vourvouri, Arend F. Schinkel, Miklos D. Kertai, Jos R. Roelandt, Thoraxcenter, Erasmus MC, Rotterdam, The Netherlands

Background: Statins have shown to exert a beneficial effect on cardiovascular system, via a number of "pleiotropic actions". Reversal of endothelial dysfunction is evidently one of them. Our aim was to determine the effect of atorvastatin on myocardial function, using pulsed-wave tissue Doppler imaging (pw TDI).

Methods: Twenty patients (age 57±11 years) with moderate hypercholesterolemia and peripheral arterial disease, without known coronary artery disease, were enrolled on atorvastatin. All patients underwent pw TDI before the initiation of atorvastatin therapy (10mg daily) and 6 months later. TDI was performed using a six-segment model. Myocardial systolic ejection velocity (V_s), and early (V_e) and late (V_a) diastolic velocities were measured at rest and during low dose dobutamine infusion (LDDI). No changes in medical therapy and no major cardiac events occurred during the follow up.

Results: Wall motion score index had no significant changes, while left ventricular ejection fraction at LDDI increased significantly at 6 months. The changes in myocardial TDI-velocities at rest and during LDDI are shown in Table.

Table

	Before atorvastatin	After 6 months	p value
V_s (rest)	7.36	8.40	<0.001
V_s (low dose)	10.36	11.59	<0.001
V_e (rest)	9.00	9.30	0.492
V_e (low dose)	10.26	10.38	0.802
V_a (rest)	9.38	10.35	0.009
V_a (low dose)	10.96	12.11	0.007
V_e/V_a (rest)	0.98	0.90	0.109
V_e/V_a (low dose)	0.96	0.87	0.107

V_s, V_e, and V_a are expressed in cm/s

P values < 0.05 were considered significant

Conclusion : Myocardial V_s at rest and at low dose dobutamine infusion increased significantly in patients with moderate hypercholesterolemia and peripheral arterial disease, after treatment with atorvastatin. This might be the result of statin-induced normalization of dysfunctional coronary endothelium.

POSTER SESSION

1141 Myocardial Remodeling and Myopathy by Magnetic Resonance Imaging

Monday, March 31, 2003, 3:00 p.m.-5:00 p.m.
McCormick Place, Hall A
Presentation Hour: 4:00 p.m.-5:00 p.m.

1141-31 **Angiotensin Converting Enzyme Inhibitors Stabilize Left Ventricular Remodeling in Chronic Aortic Regurgitation Despite Abnormal Resting Mechanics**

Victor A. Ferrari, Susan Matulevicius, Leon Axel, Craig H. Scott, Ewa Ksiezycka, Kevin Duffy, Martin G. St. John Sutton, University of Pennsylvania, Philadelphia, PA

Background: Prior studies have demonstrated impaired intramural function despite preserved global LV function in eccentric hypertrophy. We studied whether abnormalities in resting intramural mechanics in 14 patients (mean age 44) with eccentric LVH due to isolated asymptomatic moderate AR would return toward normal with acute afterload reduction (ALR) after nitroprusside (NP), and be sustained with chronic ALR using ACE inhibitors (ACEIs). Methods: We used tagged MRI to study global and regional LV systolic function before and after acute ALR, using NP to reduce systolic blood pressure by 10-20 mmHg without changing heart rate (mean dose 0.8 mg/kg/min), and after ACEIs for 3.5 years. We measured LV volumes (LVEDV and LVESV), mass, and EF. A 2-D homogeneous strain analysis measured the principal orthogonal strains E1 (greatest systolic elongation) and E2 (greatest systolic shortening). Symptoms were assessed at Baseline (B) and at 3.5 years. Results: Resting E1 was normal (Table), increased after NP, and remained normal at 3.5 years. E2 was reduced at B, and did not increase with acute or chronic ALR. LVEDV, LVESV, mass, and LVEF were unchanged, as were symptoms. Conclusions: In eccentric hypertrophy due to chronic AR, systolic lengthening (E1) improved with acute ALR, and was maintained with chronic ALR, however, systolic shortening (E2) remained abnormal. Failure to augment E2 with acute and chronic ALR did not predict progressive adverse ventricular remodeling or development of symptoms long-term.

	Baseline		3.5 yr follow-up
	Pre-NP	Post-NP	
E1	0.23+/-0.02	0.30+/-0.01*	0.26+/-0.02
E2	-0.16+/-0.02	-0.17+/-0.03	-0.17+/-0.01

1141-32 **Gadolinium Enhanced Cardiovascular Magnetic Resonance Differentiates Anderson-Fabry Disease From Hypertrophic Cardiomyopathy**

James C. Moon, Bhavesh Sachdev, Andrew G. Elkington, William J. McKenna, Dudley J. Pennell, Perry M. Elliott, Royal Brompton Hospital, London, United Kingdom, St. George's Hospital, London, United Kingdom

Background: Patients with Hypertrophic Cardiomyopathy (HCM) have areas of myocardial hyperenhancement on gadolinium enhanced Cardiovascular Magnetic Resonance (CMR). The cardiac manifestations of Anderson-Fabry disease (AFD) may mimic HCM. AFD is potentially treatable with enzyme replacement therapy and thus diagnosis is important. We hypothesised that gadolinium-DTPA CMR would differentiate HCM from AFD.

Methods: Fifteen male patients (mean 44 ± 12 years) and 7 female heterozygotes (mean 46 ± 12 years) with AFD were compared to 40 patients with familial HCM (28 male, mean 49 ± 17 years). Cine and late gadolinium CMR was performed.

Results: Eight male AFD patients (53%) had myocardial hyperenhancement (mean 7.7 ± 6.1%, range 3.6-20.6%) of total myocardium. The extent of hyperenhancement related to LV mass index (r= 0.79, p=0.0004) but not ejection fraction or ventricular volumes. Hyperenhancement was found in 3 (43%)females. In 10 (91%) patients, hyperenhancement occurred in the mid-myocardial layer of the basal lateral wall (figure 1); 2 male patients with severe LVH and systolic impairment had additional hyperenhancement in other myocardial segments. Hyperenhancement in the basal-lateral wall was not seen in the 40 patients with familial HCM.

Conclusions: Individuals with AFD have a unique pattern of myocardial hyperenhancement that can differentiate them from familial HCM. The reason for this distribution is unknown.

Figure 1: AFD (left) vs a typical pattern in HCM (right).

1141-33 **Myocardial Blood Flow in Patients With Dilated Cardiomyopathy: Quantitative Assessment With Velocity-Encoded Cine Magnetic Resonance Imaging of the Coronary Sinus**

Norbert Watzinger, Gunnar K. Lund, Maythem Saeed, Gautham P. Reddy, Philip A. Araoz, Ming Yang, Alan B. Schwartz, Martin Bedigian, Charles B. Higgins, University of California, San Francisco, San Francisco, CA, Novartis Pharmaceuticals, East Hannover, NJ

Background: Recent studies reported that microcirculatory dysfunction may play a role in the pathophysiology and may be of prognostic value in patients with idiopathic dilated cardiomyopathy (IDC). This study sought to determine the feasibility of magnetic resonance imaging (MRI) to evaluate coronary flow reserve in IDC patients and healthy subjects.

Methods: A total of 19 subjects (12 healthy volunteers and 7 IDC patients) were studied using cine MRI to measure left ventricular mass and a velocity-encoded cine MRI technique to measure coronary sinus flow at rest and after dipyridamole induced hyperemia. Absolute values of total myocardial blood flow (MBF) were calculated from coronary sinus flow and left ventricular mass.

Results: At baseline, MBF was not significantly different in patients with IDC (0.48 ± 0.07 ml/min/g) and healthy subjects (0.55 ± 0.19 ml/min/g, p = 0.41). After dipyridamole administration, MBF in IDC patients increased to a level significantly less than that in normal volunteers (1.05 ± 0.35 ml/min/g versus 1.99 ± 1.05 ml/min/g, p < 0.05). Consequently, MBF reserve was impaired in patients with IDC (2.19 ± 0.77) compared to that in healthy subjects (3.51 ± 1.29, p < 0.05). A moderate correlation was found between MBF reserve and left ventricular ejection fraction (r = 0.48, p < 0.05).

Conclusion: MBF reserve is reduced in patients with IDC indicating that coronary microcirculatory flow is impaired. This new MRI approach allows noninvasive assessment of microcirculatory function in humans and may have the potential to study the effects of pharmacological interventions on coronary microcirculation.