thickness were not increased in dogs with PRVP, morphologic and functional data indicated pathologic hypertrophy at the cellular level as shown below:

	PRVP (n = 6)	Normal (n = 6)	% Δ
Cell Volume (μm^3)	39,053 ± 2,549*	32,411 ± 1,510	+20
Cell Length (μm)	184 ± 5*	142 ± 2	+29
Cell Width (μm)	23.5 ± 0.8	22.0 ± 1.1	
Length/Width	8.3 ± 0.4*	7.0 ± 0.4	+19
Cell Shortening (%)	4.2 ± 0.8*	7.2 ± 1.1	-42
Cell Relaxation (sec)	1.01 ± 0.1*	0.71 ± 0.07	-42

(* denotes p < 0.05 vs. control)

Conclusions: 1) Cellular hypertrophy contributes to ventricular remodeling in PRVP-induced DCM in dogs, 2) Disproportional changes in myocyte geometry after RVP may contribute to increases in LV wall stress, 3) Increased LV chamber dimensions and depressed ventricular function are well reflected at the cellular level.

1092-27 Early Histopathologic Changes in Familial Dilated Cardiomyopathy

C.J. McKenna, H.M. Kwon, G. Sangiorgi, P.J. Carlson, A. Maree[1], H.A. McCann[1], W.D. Edwards, D.R. Holmes, Jr., R.S. Schwartz, D.D. Sugrue[1]. *Division of Cardiovascular Diseases, Mayo Clinic, Rochester, MN, USA; [1]Department of Clinical Cardiology, Mater Misericordiae Hospital, Dublin, Ireland*

Background: Echocardiographic screening of asymptomatic relatives of dilated cardiomyopathy (DCM) patients identifies a subset with left ventricular enlargement (LVE) who are assumed to have early familial DCM. We report the results of myocardial biopsy specimens obtained during diagnostic workup in five such relatives.

Methods and Results: Each of the 5 cases underwent cardiac catheterization to confirm LVE with normal coronary angiography. Histologic evaluation of myocardial biopsy specimens obtained included H + E and TUNEL staining, and confocal microscopy. The apoptotic index was calculated as the number of positive-staining cells divided by total number of nucleated cells (%) in ten separate high power fields per case. In all cases, mild to moderate myocyte hypertrophy was observed with only mild or no interstitial fibrosis. There was no evidence of inflammation. The apoptotic index ranged from 14–30%, with a mean for the 5 cases of 25%. TUNEL positive cells were more prominent in the interstitium than the myocardium.

Conclusion: Asymptomatic relatives with LVE have ongoing DCM disease activity. This activity does not appear to be inflammatory in nature but is associated with a high apparent apoptotic index. Myocardial biopsy at this early stage may help to identify patients suitable for potentially curative therapy.

1092-28 Improvement of Active Relaxation After Weight Loss in Young Obese Individuals

Gian F. Mureddu, Giovanni de Simone, Anna Cella, Rosanna Greco, Carmela De Caprio, Franco Contaldo. *Federico II University Hospital, Naples, Italy*

Background: Prolonged left ventricular (LV) isovolumic relaxation time (IVRT) has been described in uncomplicated obesity, but the effect of weight loss is unknown.

Methods: Twenty normotensive young obese subjects free from cardiovascular disease and diabetes [age = 26 ± 9 years; body mass index (BMI) = 33.7 ± 3.4 Kg/m^2; 12 women], have been studied by Doppler echocardiography before and after reduction of at least 10% of initial body weight after one to six months of a balanced 1200 Kcal diet.

Results: After weight loss, diastolic blood pressure was significantly decreased (75 ± 10 versus 83 ± 9 mmHg at baseline after echocardiogram; p < 0.01), whereas LV mass index (32 ± 11 versus 35 ± 14 g/m$^{2.7}$), midwall shortening (19 ± 3 versus 18 ± 2%) and ejection fraction (67 ± 5 versus 66 ± 5%) did not show statistical difference. LV end-diastolic diameter as a rough index of preload (4.78 ± 0.6 versus 4.76 ± 0.5 cm) and circumferential end-systolic stress as a measure of myocardial afterload (129 ± 18 versus 123 ± 29 kdynes/cm^2) did not significantly change after diet. IVRT was significantly shortened after weight loss (81 ± 16 versus 94 ± 12 msec ; p < 0.003). Deceleration time of early velocity (147 ± 20 msec versus 154 ± 22 msec) and transmitral peak early filling to peak late filling velocity ratio (1.52 ± 0.45 versus 1.53 ± 0.41) were not influenced by weight loss. Percent reduction of initial IVRT value was positively related to the decrement of BMI (r = 0.69; p < 0.02), but not to change in LV mass, in diastolic blood pressure, in LV chamber dimension and in end-systolic stress (p > 0.2).

Conclusion: Weight loss in uncomplicated obesity is associated to improvement of LV active relaxation that was not related to change in left ventricular mass, filling and load.

1093 Exercise and Diagnostic Studies in *Hypertrophic Cardiomyopathy*

Tuesday, March 31, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 9:00 a.m.–10:00 a.m.

1093-41 Exercise Capacity Is Related to Echocardiographic Indexes of Diastolic Function in Patients With Hypertrophic Cardiomyopathy

C. Briguori, S. Betocchi, M. Romano, F. Manganelli, M. Angela Losi, R. Gottilla, Q. Ciampi, M. Condorelli, M. Chiariello. *Federico II University, Naples, Italy*

Background: A limited exercise capacity is common in patients with hypertrophic cardiomyopathy (HC). Diastolic dysfunction is an important determinant of exercise capacity in HC. Echocardiography is a noninvasive tool to investigate diastolic function. In this study we sought the correlation between exercise capacity and echocardiographic indexes of diastolic function in patients with HC.

Methods: We studied 42 patients with HC while off drugs. The New York Heart Association (NYHA) functional class was I in 25, II in 12, and III in 5 patients. The *global* and *active* left atrial (LA) fractional shortening (FS), the slope of early posterior aortic wall displacement during LA emptying (Slope) were calculated by M-mode; pulsed Doppler was employed to record transmittal and pulmonary venous flow velocities. Peak oxygen consumption and anaerobic threshold (ml/kg/min) were measured by gas analysis during a symptom-limited exercise by cicloergometer.

Results: NYHA functional class correlated with LA *global* FS and mitral regurgitation (r = 0.65; p < 0.001; standardized coefficients = −0.38 and 0.37; F = 11.572); peak oxygen consumption correlated with LA *global* FS and Slope (r = 0.68 p < 0.001; standardized coefficients = 0.40 and −0.39; F = 13.611); anaerobic threshold correlated with peak of pulmonary venous diastolic flow velocity and LA *active* FS (r = 0.61; p = 0.002; standardized coefficients = 0.42 and 0.37; F = 8.935).

Conclusions: Exercise capacity in patients with HC is related to diastolic dysfunction assessed by the combined evaluation of LA function and pulmonary venous flow velocity profiles. Transmitral flow velocity profiles do not provide further information.

1093-42 Abnormal Systolic Blood Pressure Response During Exercise Is Related to Subendocardial Hypoperfusion Detected by Exercise ^{201}Tl Myocardial Scintigraphy in Patient With Hypertrophic Cardiomyopathy

N. Yoshida, H. Ikeda, T. Wada, S. Maki, A. Matsumoto, E. Sumida, T. Imaizumi. *Kurume University School of Medicine, Kurume, Japan*

Background: Abnormal systolic blood pressure (SBP) responses during exercise are frequently documented in patients with hypertrophic cardiomyopathy (HCM) and these findings have been proposed as a possible risk factor of sudden cardiac death in HCM. However, these clinical significance and mechanism are not fully known. In this study, we hypothesized that exercise-induced abnormal SBP response might be due to myocardial ischemia during exercise.

Methods and Results: 104 patients with HCM (mean age 50 yrs) underwent exercise ^{201}Tl myocardial scintigraphy and cardiac catheterization. When patients were divided into the two groups of normal (n = 87) and abnormal (n = 17) SBP responses during exercise (ΔSBP ≥25 and <25 mmHg respectively from baseline to peak exercise), patients with abnormal SBP responses had higher prevalence rate of left ventricular cavity dilatation assessed by exercise thallium scintigraphy, which may indicate subendocardial hypoperfusion, than patients with normal SBP responses (47.0 vs. 9.4%, p < 0.0001). Regarding to reversible and fixed perfusion defect by exercise thallium scintigraphy, no differences were found between the two groups. Furthermore, patients with abnormal SBP responses had significantly elevated left ventricular end-diastolic pressure than patients with normal SBP responses (17.6 ± 8.4 vs. 14.3 ± 5.3 mmHg, p < 0.05).

Conclusions: Our data suggest that exercise-induced abnormal SBP responses in HCM may be due to the subendocardial hypoperfusion associated with elevation in diastolic pressures during exercise.

TUESDAY POSTER