observed a clinical response to infusions of physiologic doses of arginine vasopressin (AVP). This led us to hypothesize that patients manifesting vasodilatory shock after CPB might have an endogenous vasopressin deficiency similar to that described in states of autonomic dysfunction. We therefore examined the incidence and significance of AVP deficiency in 109 patients undergoing elective CPB. *Methods:* The study group consisted of 109 consecutive patients undergoing CPB for elective coronary artery bypass and/or valve replacement surgery. Serum AVP levels were measured upon weaning from CPB, and perioperative hemodynamic parameters and exogenous pressor requirements were recorded. Post-bypass hypotension was defined as a mean arterial pressure (MAP) <70 mmHg upon weaning from bypass coupled with an exogenous norepinephrine (NE) requirement persisting for ≥2 hours postoperatively, and was characterized as cardiogenic (cardiac index <2.5 L/min/m^2) or vasodilatory (cardiac index >2.5 L/min/m^2). All values are expressed as mean ± SEM, and comparisons made by ANOVA. *Results:* 14 of 109 patients studied (13%) met criteria for post-bypass hypotension, with a mean MAP of 64 ± 2 mmHg and a mean norepinephrine requirement of 12.1 ± 4.2 μg/min. As expected, patients without hypotension had low AVP levels (15.7 ± 0.9 pg/ml). However, although AVP levels in the 4 patients with cardiogenic hypotension were appropriately elevated (35.0 ± 5.6 pg/ml), they were inappropriately low in the 10 patients with vasodilatory hypotension (12.9 ± 3.1 pg/ml, p = 0.005 vs. cardiogenic hypotension). Furthermore, the degree of vasodilation correlated inversely with AVP levels (r^2 = 0.68). *Conclusions:* Pressor-dependent vasodilatory hypotension after cardiopulmonary bypass is associated with an endogenous vasopressin deficiency, suggesting a potential therapeutic role for exogenous AVP in these patients. Whether this deficiency is due to impaired central release or to increased peripheral uptake of this vasoactive hormone is under current study.

736 Importance of Measures Other Than Left Ventricular Systolic Function in Determining Exercise Capacity

Tuesday, March 18, 1997, 8:30 a.m.–10:00 a.m.
Anaheim Convention Center, Room A19

8:30

736-1 Exercise Lactate Responses in Elderly Patients with Heart Failure Due to Diastolic vs. Systolic Left Ventricular Dysfunction

D.W. Kitzman, W.E. Pollock, P.H. Brubaker, B.K. Rayburn, D. Wesley. *Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, NC, USA*

Exercise intolerance is a prominent symptom in chronic heart failure (HF), and data from patients with HF due to LV systolic dysfunction (SD) suggest that it is associated with altered lactate kinetics. However, limited data are available regarding exercise lactate kinetics in elderly HF patients, particularly those with HF due to primary LV diastolic dysfunction (DD). Therefore, we examined 3 age-matched groups (mean 68 yrs): HF due to primary DD (n = 25, mean EF 68%, range 55–78%); HF due to primary SD (n = 25, mean EF 29%, range 15–45%) and 9 healthy normal (NL). Subjects underwent symptom-limited maximal upright staged cycle ergometry. Mean respiratory exchange ratio was ≥1.12 in all subject groups. Variables assessed included: submaximal (SM, defined as 50% of max exercise time), peak, and 2 minute recovery systemic venous blood lactate (LA, mMol/L); peak oxygen uptake (peak VO_2, ml/kg/min); exercise time (ET, seconds). Mean ± S.E.M.; *p ≤ 0.05 vs NL.

	LA (Rest)	LA (SM)	LA (Peak)	LA (Rec)	Peak VO_2	ET
DD	1.2 ± 0.1	2.0 ± 0.2	3.1 ± 0.3	4.7 ± 0.4*	14.0 ± 0.5*	471 20*
SD	1.5 ± 0.1	1.6 ± 0.1	2.5 ± 0.2*	4.0 ± 0.3*	14.7 ± 0.6*	474 ± 32*
NL	1.2 ± 0.1	1.7 ± 0.2	3.6 ± 0.5	6.1 ± 0.7	18.4 ± 1.1	599 ± 69

Conclusion: Peak and recovery exercise lactate responses and aerobic capacity were similar in elderly patients with heart failure due to diastolic dysfunction vs. systolic dysfunction and were reduced in both compared to age-matched normals. These data suggest that patients with HF due to primary DD may have peripheral mechanisms of exercise intolerance that are similar to patients with primary SD, despite markedly different LV EF.

8:45

736-2 Correlation of Invasive Hemodynamic Parameters with Exercise Performance in Patients with Hypertrophic Cardiomyopathy

R.R. Brandt, R.A. Nishimura, T.G. Allison, A.J. Tajik, D.R. Holmes, Jr.. *Mayo Clinic and Foundation, Rochester, MN, USA*

Hypertrophic cardiomyopathy (HCM) can cause severe symptoms of angina, dyspnea and syncope due to multiple pathophysiologic mechanisms such as outflow obstruction, diastolic dysfunction and mitral regurgitation. Limited information is available on the mechanism of exercise limitation in these patients. The purpose of this study was to correlate catheter hemodynamic measurements from high-fidelity micromanometer catheters placed in the left atrium and left ventricle with peak oxygen consumption (VO_2 max) during symptom-limited treadmill exercise testing. Thirty-four patients (17 males and 17 females, mean age 55 ± 16 yrs) exercised for 6.5 ± 2.7 min achieving a VO_2 max of 20.0 ± 7.9 mL/kg/min. Mean left atrial pressure (LAP) was 17.7 ± 7.5 mmHg, cardiac index 2.7 ± 0.6 L/min/m^2 and left ventricular outflow pressure gradient 60 ± 41 mmHg. Multivariate analysis correcting for gender, age and effort disclosed a significant correlation between VO_2 max and peak negative dp/dt (r = 0.8, p < 0.05) but not with resting mean LAP or left ventricular outflow pressure gradient.

Conclusion: The study shows that in patients with HCM there is no correlation between maximal oxygen consumption and resting mean left atrial pressure or left ventricular outflow pressure gradient. Patients with a lower negative dp/dt (abnormally slow relaxation) have a greater limitation of exercise tolerance.

9:00

736-3 Exercise Performance in Stable Patients With Systolic Dysfunction is Related to Left Ventricular Diastolic Function

P. de Groote, M. Lantreibecq, A. Millaire, L. Goullard, J.-M. Lablanche, G. Ducloux. *University of Lille, France*

The mechanism of exercise intolerance in patients (pts) with systolic dysfunction is not well understood. The aim of the study was to find a relationship between echo-Doppler parameters and exercise performance. We performed a symptom limited exercise with the determination of peak VO_2 and an echo-Doppler study at rest and during exercise in 25 ambulatory pts in sinus rhythm, aged 47.8 ± 11.6 years (mean ± SD), with stable systolic dysfunction (NYHA II/III, left ventricular ejection fraction: 30.4 ± 9.6%, determined by radionuclide angiography). Systolic and diastolic echo-Doppler parameters were determined at rest and during exercise at a heart rate of 120/min. Each parameter was the average of at least 6 measurements. Peak VO_2 was 17.3 ± 7.1 ml/min/kg (58.5 ± 20.8% of maximal predicted value). At rest, only the isovolumic relaxation time (IRT: 104 ± 25.4 ms) was correlated with the % of peak VO_2 (r = 0.42, p < 0.05). During exercise, the mitral time velocity integral (TVI: 11.3 ± 2.57 cm) and the mitral diastolic flow propagation (DFP, determined by color M-mode: 65 ± 15 cm/sec) were correlated with % of peak VO_2 (r = 0.55, p < 0.01, r = 0.59, p < 0.01, respectively). The % of change from rest to exercise of the IRT and the DFP were correlated with the % of peak VO_2 (r = 0.45, p < 0.05, r = 0.63, p < 0.01, respectively). There was no correlation between systolic parameters and peak VO_2. *In conclusion,* in pts with stable systolic dysfunction, exercise performance is related to left ventricular diastolic function and not to systolic function. Diastolic function plays a role in determining exercise capacity in pts with congestive heart failure.

9:15

736-4 Does Pulmonary Hypertension Precardiac Transplantation Impact on Post-Transplant Exercise Capacity?

Y.-M. Cha, N. Osada, A. Stelken, T. Donohue, T. Wolford, M.B. Cishek, L.W. Miller, B.R. Chaitman. *St. Louis University School of Medicine, St. Louis, MO, USA*

The role of moderate pulmonary hypertension (PHT) in compensated heart failure pts considered for cardiac transplantation was evaluated in 44 pts who underwent cardiopulmonary exercise testing and right heart catheterization before and 1–3 years after transplantation. The PHT group consisted of 29 pts with mean PA pressure (PAM) >30 mm Hg. The results were compared to 15 pts whose PAM was <30 mm Hg (non-PHT group).