cent bands corresponding to BNP and ANP were quantified in densitometric units.
Results: mRNA expression for both BNP and ANP increased significantly in untreated HF dogs compared to NL. Treatment with the CSD reduced mRNA expression of both BNP and ANP to near NL levels. Data are shown in the table.
Conclusions: In dogs with HF, long-term therapy with the Acorn CSD is associated with reduced mRNA expression of both BNP and ANP. These results support earlier findings that benefits of the CSD in HF are largely mediated through prevention of myocardial stretch.

	Normal	HF Untreated	HF + CSD
BNP	2.7 ± 0.4	5.5 ± 0.2*	3.1 ± 0.6
ANP	1.5 ± 0.1	2.4 ± 0.1*	2.0 ± 0.1

*=P<0.05 vs. Normal. Based on ANOVA followed by Student-Neuman-Keuls test

1061-159 Presentation of Acute Decompensated Congestive Heart Failure in African-Americans and Their Response to Treatment With Nesiritide

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Background: There are often racially based differences in response to cardiovascular medications and in disease presentation. Nesiritide (B-type natriuretic peptide) is a newly available agent that in clinical trials was shown to: reduce preload and afterload, improve symptoms, promote natriuresis, and blunt sympathetic and renin-angiotensin-aldosterone axis activation in acutely decompensated congestive heart failure (CHF) patients.
Methods: To determine the baseline characteristics and hemodynamic and clinical effects of nesiritide in African-American (AA) patients, data from the AA patients in a large randomized controlled trial (VMAC) of patients with decompensated CHF was analyzed. All patients could receive standard therapy, including IV diuretics, dobutamine, and dopamine at their physician's discretion.
Results: In VMAC, 119/489 (24%) of treated patients were AA. Compared to non-AA, the primary etiology of chronic cardiomyopathy (CM) in AA patients was more often nonischemic (59% vs. 31%, respectively, $p < 0.001$). More AA had hypertensive CM vs. non-AA and had a history of hypertension ($p < 0.001$). Compared to non-AA patients, AA presented less frequently with rales (62% [AA] vs. 77% [non-AA], $p = 0.002$), but more commonly had an S4 (29% [AA] vs. 19% [non-AA], $p = 0.04$) and hepatomegaly (48% [AA] vs. 35% [non-AA], $p = 0.018$). Baseline NYHA class and ejection fraction were similar in AA and non-AA. Like non-AA patients, AA patients who received standard care + nesiritide ($n = 30$) had greater reductions in PCWP than those who received standard care + placebo ($n = 14$) at every time point from 15 minutes to 3 hours ($p \leq 0.05$ for all points). At 3 hours, the primary endpoint, PCWP decreased by -5.9 mmHg in the nesiritide (+ standard care) vs. -0.6 mmHg in the placebo (+ standard care) groups ($p = 0.001$). The percentage of patients with improved dyspnea was 88% (AA) vs. 65% (non-AA) ($p = 0.028$).
Conclusions: AA patients have a greater preponderance of hypertensive cardiomyopathy and evidence of left ventricular noncompliance. The AA population within the VMAC study had similar improvements over standard care in hemodyamics and symptoms when treated with nesiritide as compared to the non-AA VMAC patients.

1061-160 Effects of the Creation of Arteriovenous Shunt for Hemodialysis on Cardiac Function and Natriuretic Peptide Levels in Chronic Renal Failure

Yoshio Iwashima, Takeshi Horio, Yoichi Takami, Takashi Inenaga, Toshio Ogihara, Yuhei Kawano, *Geriatric Medicine,Osaka University Medical School, Suita, Japan, National Cardiovascular Center, Suita, Japan.*

Background: Heart failure is caused occasionally by the creation of vascular access for hemodialysis. However, the effect of arteriovenous (AV) shunt on cardiac function has not been fully elucidated. This study investigated the serial changes in cardiac function and hormonal levels by the AV shunt operation. Methods: Sixteen patients with end stage renal disease underwent echocardiographic studies before and 3, 7, and 14 days after the AV shunt operation. Plasma atrial natriuretic peptide (ANP) and brain natriuetic peptide (BNP) concentrations were measured before and 1, 3, 6, 10, and 14 days after the operation. Results: The creation of AV shunt produced significant elevations of left ventricular end-diastolic diameter (+4% at 7 days, P=0.005), left ventricular end-diastolic volume (+10% at 7 days, P=0.006), fractional shortening (+8% at 7 days, P=0.001), and cardiac output (+15% at 7 days, P=0.01). In transmitral flow by Doppler echocardiography, the deceleration time of the early diastolic filling wave shortened (-12% at 14 days, P=0.001) and the ratio of the peak velocity of early diastolic to atrial filling (E/A) increased (+18% at 14 days, P=0.004). That is, the creation of AV shunt induced the restrictive impairment in LV diastolic function. Blood pressure showed a tendency to decrease and heart rate did not change significantly through the study. Both ANP and BNP increased after the operation, and the maximal increases were observed after 10 days (ANP: +48%, BNP: +68%). In the relationship between cardiac function and hormonal response, the increase in cardiac output was associated with the elevation of ANP (r=0.61, P=0.01), but not BNP. On the other hand, the increase in E/A was correlated only with BNP elevation (r=0.60, P=0.01). Therefore, the stimulation of ANP and BNP secretions was differently regulated by other hemodynamic factors. Conclusion: This is the first report that shows the serial changes in cardiac function and hormonal levels after the AV shunt operation. Our observations indicate that the creation of AV shunt has significant effects on cardiac performance and that ANP release is induced by volume loading, but BNP release is stimulated by left ventricular diastolic dysfunction.

MODERATED POSTER SESSION

1062MP Moderated Poster Session...Ventricular Remodeling

Sunday, March 17, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G

3:00 p.m.

1062MP-121 Reverse Remodeling Following Long-Term Carvedilol Therapy Is Associated With Improvement in Survival: The Stanford Carvedilol Echocardiographic Registry

W.H. Wilson Tang, Mary S. Larson, Randall H. Vagelos, Lisa Prikazsky, Bob S. Hu, Michael B. Fowler, *Stanford University, Stanford, California.*

Background: Beta-blocker therapy reverses the process of ventricular remodeling and reduces mortality in patients with heart failure. However, direct association between reverse remodeling and improvement in survival has not been demonstrated. Few clinical parameters have been shown to provide prognostic information during ongoing heart failure treatment.
Methods: We prospectively followed 257 patients with chronic heart failure treated with carvedilol for over 6 months between 1993-9. Serial echocardiographic assessments of left ventricular ejection fraction (LVEF, n=257) and left ventricular end-diastolic dimensions (LVIDd, n=242) were measured at baseline and at 6-12 months ("serial") following carvedilol therapy. "Reverse remodeling" was defined as improvement in LVEF ≥10% or reduction in LVIDd ≥10 mm in serial measurements. Clinical end-points were all-cause mortality (by clinical records and/or by Social Security Death Index) and transplantation (by clinical records) up to May 2001. Comparisons between groups based on echocardiographic criteria (baseline and serial LVEF/LVIDd) were performed using Kaplan-Meier survival curves.
Results: Of the 257 patients studied (mean age 52±13 years, mean LVEF 24.8±8%, mean LVIDd 70±10mm), 127 (49%) had reverse remodeling by LVEF criteria, and 65 out of the 242 patients (27%) had reverse remodeling by LVIDd criteria. During a mean follow-up of 4 years, 53 (20.6%) died or underwent transplantation. Kaplan-Meier survival curves of patients were not significantly different between groups according to baseline LVEF (<25% versus ≥25%) or LVIDd (<70mm versus ≥70mm) measurements; but were significantly different at the log-rank test between groups with or without reverse remodeling according to serial LVEF or LVIDd measurements (log-rank χ^2, $p < 0.001$).
Conclusion: Baseline LVEF and LVIDd did not predict survival following carvedilol therapy. However, echocardiographic parameters of reverse remodeling were associated with improvement in survival following carvedilol therapy. Serial measurements in such parameters during ongoing treatment may therefore provide additional prognostic information for patients with heart failure.

3:12 p.m.

1062MP-122 Effects of Valsartan on Left Ventricular Ejection Fraction and Diastolic Diameter in Patients With Heart Failure: Val-HeFT Echocardiographic Study

Maylene Wong, Lidia Staszewsky, Roberto Latini, Yann-Tong Chiang, Alberto Volpi, Christer Hoglund, Jay N. Cohn, for the Val-HeFT Investigators, *West LA VA Medical Center, Los Angeles, California, Istituto Mario Negri, Milano, Italy.*

Objective: To evaluate the effect of the angiotensin receptor blocker valsartan on left ventricular (LV) structure and function when added to standard heart failure (HF) therapy.
Methods: At 302 multi-national sites, 5010 pts in NYHA class II to IV HF on stable therapy which could include ACE inhibitor and betablocker were randomized to valsartan (V) and placebo (P) groups and followed for a mean duration of 22.4 mos. Serial echocardiographic (echo) measurements of ejection fraction (EF) and LV internal diastolic diameter (LVID) were recorded. Reproducibility calculated to 90% power at 5% significance revealed detectable differences of 0.86% for EF and 0.09 cm for LVID. Mean group changes from baseline over time were compared.
Results: Baseline EF and LVID for V and P groups were similar: 26.60 vs 26.87 (%); 3.64 vs 3.66 (cm/m2). Consistent, significant increase in EF and decrease in LVID began at 4 mos, reached a plateau by 1 year, and persisted to 2 years in V compared to P pts. Similar trends were seen in males and females, and pts <65 and >65 years. Racial differences in response to V were suggested, but the number of black pts was small.
Conclusions: Val-HeFT provided a large HF pts recruitment and multiple sequential echocardiograms for studying effects of V on echo measurements. The data demonstrated that V treated pts had a decrease in LV size and increase in systolic function that began to develop by 4 mos and continued for at least 2 years.* p<0.001; § p=0.032; # p=0.034. ANCOVA controlling for center and baseline.

Interval		4 mos n=4,556	12 mos n=4,014	18 mos n=3,018	24 mos n=1,991
EF	V	2.73*	4.12*	4.49*	4.50#
	P	2.02	2.82	3.21	3.66
LVIDd/BSA	V	-0.07*	-0.09*	-0.12*	-0.10§
	P	-0.03	-0.05	-0.05	-0.06