11:15

840-4 Antioxidant Effect of Carvedilol and Metoprolol in Congestive Heart Failure

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Although the antioxidant properties of Carvedilol (C) have been measured *in vitro* in cell preparations, they have not been shown *in vivo* in heart failure patients. It has been previously demonstrated that thiobarbituric acid reactive substances (TBARS), which are markers of lipid peroxidation and oxidative stress, are elevated in CHF patients compared to controls. TBARS also increase with worsening NYHA class. To date, there have been no direct comparisons of C and Metoprolol (M) in a CHF population.

We randomly assigned 56 patients (open label) to M or C. Baseline evaluation included ejection fraction (EF), symptom score (SS), QOL index, 6 min walk, mVO_2, and TBARS as measured by spectrophotometry in the plasma. β-blocker was gradually titrated to a target dose of 25 mg bid (50 mg bid if wt >85 kg) for both C and M. Background therapy consisted of A2 or ACEI (95%), digoxin and diuretics. Of the 56 pts (mean age 58) 43 completed 6 months of long-term therapy. There were 41 men and 15 women; etiology: 21 ischemic, 30 idiopathic, 5 valvular with NYHA class II/III/IV: 11/39/6.

The entire group (C & M) showed significant, parallel improvements ($p < 0.01$) in EF (18 to 22%), SS (10.3 to 7.5), QOL (50 to 37), walk (1152 to 1247 feet), and mVO_2 (10.8 to 12.0 ml/kg/min) (by ANOVA) with no between group difference.

TBARS concentration (ng/ml) at weeks -2, 0 (baseline averaged) and Months 4 & 6 with C declined from 5.2 to 4.4 to 4.3 and with M from 4.8 to 4.6 to 3.9. P = 0.0002 for overall change over time in both groups with no between group difference.

Thus, β-blockers demonstrate significant clinical, hemodynamic exercise, and antioxidant benefits over 6 months in this CHF population with no discernible difference between C and M in any of the measured parameters.

11:30

840-5 Effects of Carvedilol on LV Function and Remodeling in Patients With Dilated Cardiomyopathy With Persistent LV Dysfunction Despite Optimal Conventional Therapy

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Background: In order to analyze the additive effect of carvedilol (C) in dilated cardiomyopathy (IDC) with long history (61 ± 51 months) of symptomatic LV dysfunction despite chronic tailored metoprolol (M) and ACE inhibitor treatment, we enrolled 30 pts (peak VO2 < 25 ml/kg/min and LVEF < 0.40) to randomly continue open M (n = 16) (142 ± 44 mg/day) or to cross to C (n = 14), uptitrated to the maximum tolerated dose (74 ± 23 mg/day), for 12 months.

Results:

	Baseline			Δ 12 months-baseline		
	M	C	p	M	C	p
NYHA (1–4)	1.9 ± 1	1.6 ± 1	*	-0.1 ± 0.2	-0.2 ± 0.6	*
LVEF (%)	31 ± 7	30 ± 6	§	1 ± 8	+7 ± 12	†
LVEDV (ml/m2)	126 ± 47	115 ± 33	§	+7 ± 22	-8 ± 28	§
LVESV (ml/m2)	91 ± 10	80 ± 7	§	-6 ± 4	7 ± 5	*
Peak VO2 (ml/kg/m)	17 ± 4.1	18.1 ± 3	§	-1 ± 2	1 ± 2	†
Rest HR (bpm)	60 ± 8	60 ± 7	§	0 ± 1	+1 ± 1	§

Legend. EDV: end-diastolic volume; ESV: end-systolic volume; HR heart rate. (*): p = NS. (§) p < 0.10; (†): p < 0.05.

Conclusions: in pts with IDC and long history of symptomatic LV dysfunction despite chronic optimal treatment, C, as compared to M, showed a significant additive effect on LV function and remodeling, although significantly decreased maximal exercise tolerance.

11:45

840-6 Beta Blockade Therapy in Chronic Heart Failure: Diastolic Function and Mitral Regurgitation Improvement by Carvedilol

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Background: In patients with chronic heart failure the use of carvedilol therapy induces clinical and hemodynamic improvements. However, data about the effects of Carvedilol on the left ventricular diastolic function and mitral regurgitation are not well defined.

Objective: to identify the clinical, functional effects of Carvedilol, focusing the diastolic function and mitral regurgitation variations.

Methods: twenty-four consecutive patients (52 ± 8 yrs) with CHF (22 ± 6%) due to dilated ischemic/nonischemic (9/15) cardiomyopathy - treated with Carvedilol (mean dose 44 ± 30 mg) - were matched to a control group for clinical (functional class NYHA, CHF duration) and haemodynamic (Cardiac Index and pulmonary wedge pressure) characteristics. We obtained two groups: G1: (24 pts: Carvedilol group) and G2: (24 pts: control group). Clinical and echocardiographic variables measured in two groups at baseline and after six months were compared.

Results:

	baseline		6 months	
	G1	G2	G1	G2
FC (bpm)	87 ± 17	83 ± 15	72 ± 9*	84 ± 12
LVEDVI (ml/m2)	72 ± 9	75 ± 12	70 ± 10	73 ± 12
LVESVI (ml/m2)	62 ± 10	64 ± 14	57 ± 11*	68 ± 17
NYHA (I/II/III)	3/18/3	4/16/4	6/18/0	5/17/2
EF%	22 ± 6	23 ± 7	26 ± 7*	22 ± 8
E/A	1.5 ± 0.8	1.6 ± 1	1.7 ± 0.9	1.5 ± 0.9
DT (msec)	132 ± 56	129 ± 43	198 ± 76*	136 ± 51
MR (cm2)	5.9 ± 2	6 ± 2.5	3 ± 3*	6.2 ± 1.8

(*p < 0.05 bas vs 6 months †p < 0.05 G1 vs G2)

Conclusions: After 6 months of treatment, Carvedilol induces a significant reduction of left ventricular end-systolic volume with improvement of systolic function. These changes were associated with restoration of diastolic dysfunction and concomitant decrease of mitral regurgitation.

841 Cardiovascular Surgery for Congenital Heart Disease

Tuesday, March 31, 1998, 10:30 a.m.–Noon
Georgia World Congress Center, Room 365W

10:30

841-1 A New Strategy for the Surgical Treatment of Aortic Coarctation Associated With Ventricular Septal Defect in Infants Using an Absorbable Pulmonary Artery Band

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Background: Decision making for the surgical management of aortic coarctation (CoA) associated with ventricular septal defect (VSD) may be difficult in infants. The alternative is single stage repair versus CoA repair with banding of the pulmonary artery. As a large proportion of the VSD associated with CoA are prone to close spontaneously, we propose a new strategy using CoA repair with absorbable pulmonary banding.

Methods: Nine infants [mean weight 3050 ± 800 g, range 1435–3800 g] underwent a CoA repair with a polydioxanone banding through a posterolateral approach at a mean age of 22 days [8–41 days]. Five had a trabecular VSD and 4 a perimembranous VSD [4–7 mm] with isosystemic pulmonary hypertension. The pulmonary band was tightened until the systolic pulmonary pressure fell below 50% of the aortic pressure.

Results: There was no hospital death. The resorption of the pulmonary band was complete after 4.5 months in all patients [3–5.5 months]. The VSD closed completely in 2 infants and partially in 6 in whom the pulmonary artery pressure was normal without evidence for significant left-to-right shunt. One patient with a large muscular VSD underwent surgical closure of his defect after 4 months. Finally, a subsequent open heat surgery could be avoided in 89% (8/9) patients.

Conclusion: Provided the VSD belongs to types characterized by a high incidence of spontaneous closure, this effective policy may reduce the number of surgical procedures per infant, in-hospital mortality and morbidity rates. It should be proposed as an alternative in the panel of surgical options in this disease.

10:45

841-2 Minimally Invasive Approach for ASD Repair

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Background: A minimally invasive right thoracotomy incision, when feasi-