

occurred between day 10 ± 1 and 3 month. 4 patients died after hospital discharge (no death directly related to thromboembolic disease).

Thus no higher risk of PE can be seen in patients with free floating prox-DVT and anticoagulant therapy should be efficient to prevent recurrent PE in such patients.

980-89

Lack of Evidence for Atrial Stunning After DC Shock in Patients in Normal Sinus Rhythm Undergoing ICD Insertion: A Study Using TEE

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Transient left atrial (LA) dysfunction ("stunning") and the appearance of spontaneous echo contrast ("smoke") have been reported in patients undergoing DC cardioversion of atrial fibrillation. It has been suggested that cardioversion-induced LA dysfunction may promote new thrombus formation leading to thromboembolic complications in AF patients even in the absence of demonstrable LA thrombus prior to cardioversion. In order to confirm previous observations of LA stunning and to investigate the determinants of this response, we investigated LA function in a series of patients undergoing implantable defibrillator insertion and testing. Eight patients (67 ± 6 yrs, 5 M/3 F) with ischemic heart disease (n = 6) or cardiomyopathy (n = 2) and VT/VF were studied. All patients were in NSR, had significant impairment of systolic LV function, and demonstrated inducible VT or VF in the EP lab. Intraoperative transesophageal echocardiography with a biplane or multiplane probe was used to image the LA, mitral valve and LA appendage (LAA). Blood flow velocities were measured at the tips of the MV leaflets and LAA orifice by pulsed-wave Doppler before and 30 to 120 seconds after DC shocks of 15–20 joules with transvenous intracardiac electrodes (in SVC and RV apex, n = 7) or epicardial patches (n = 1) while in sinus rhythm. Measurements from three different cardiac cycles were averaged for each patient. Baseline LAA orifice flow velocity was 44 ± 16 cm/s and remained unchanged after DC shock, 46 ± 20 cm/s (p = ns). Similarly, MV peak A-wave velocity was 70 ± 34 cm/s at baseline and was not significantly different after DC shock, 69 ± 42 cm/s (p = ns). No evidence of LA "smoke" was seen in any patient before or after the shock. **Conclusion:** As opposed to previous reports after cardioversion of atrial fibrillation, DC shocks alone do not produce LA dysfunction in patients in NSR even in the presence of left ventricular dysfunction. LA stunning after DC shocks may require prior atrial fibrillation or specific atrial substrate.

980-90

Superior Vena Cava Versus Right Atrium Central Venous Catheter Placement Avoids Thrombosis: The Role of Transesophageal Echocardiography in Diagnosis and Evaluation — A Prospective Study

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Intravenous central line (Hickman type) catheters (CL) are routinely used in the management of chronically ill patients. These CLs are placed in the superior vena cava (SVC) or right atrium (RA) and are often associated with complications, mainly thrombosis or infection. The introduction of transesophageal echo (TEE) has significantly improved the imaging of intracardiac structures, especially left atrial thrombi and right atrial masses. We explored the use and importance of TEE (and compared to transthoracic echo (TTE)) for early evaluation of CL placement and detection of related masses. **Methods:** We prospectively studied fifty-five (55) bone marrow transplantation (BMT) patients by TTE and TEE at an asymptomatic stage within a week post-Hickman catheter (CL) implantation and on a follow-up study after 6–8 weeks. We looked for the exact CL tip placement and searched for possible presence of any related abnormalities. **Results:** Of the fifty-five patients in the first study, the CL tips could be demonstrated in 48 (87%) of them by TEE compared to only 4 (8%) by TTE. 13 were placed in the right atrium (RA), 8 at the superior vena cava-right atrium junction (SVC-RA), and 27 in the superior vena-cava (SVC). *An abnormal mass was found in six patients (12.5%).* All of these presumed thrombi were seen in patients in whom the CL tip was placed in the RA (Table)

No. of Patients	CL location	Thrombi
27	SVC	0
8	SVC-RA	0
13	RA	6 (p < 0.001)

Conclusions: TEE studies performed in an asymptomatic setting of BMT patients within a week post-routine CL implantation demonstrated *unexpected, asymptomatic catheter-tip related masses, consistent with thrombosis, in the RA of 12.5% of patients.* These findings suggest that: (1) CL should be placed in the SVC or SVC-RA junction, in contrast to the RA. (2) TEE is

a useful tool for guiding CL's placement in severely immunocompromised, chronically ill patients, to avoid formation of thrombi.

980-91

Predictors of Thrombus Formation After Anterior Myocardial Infarction: Evidence of Protective Effect of Mitral Regurgitation

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It has been shown that mitral regurgitation (MR) prevents thrombus (T) formation in pts with dilated cardiomyopathy. Also, the relationship between low velocity ventricular flow and T formation after MI was recently demonstrated. However, no clinical study has shown that MR has independent effect on T formation in acute myocardial infarction (MI). In order to determine predictors of T after anterior MI, we have analyzed *clinical* (age, sex, Killip class, thrombolysis, peak CK values), *echocardiographic* (left ventricular end-diastolic volume index-EDVi, end-systolic volume index-ESVi, ejection fraction-EF, wall motion score index-WMSi, apical wall motion abnormalities, presence of MR) and *angiographic* (extent of coronary artery disease-CAD, patency of infarct related artery-IRA) variables in 54 consecutive pts with anterior MI. Two-dimensional and Doppler echocardiographic examinations were performed in the following sequence: day 1, day 2, day 3, day 7, after 3 and 6 weeks, 3 and 6 months and 1 year following MI. Pts with and without T were similar regarding age, sex and antithrombotic therapy. According to Cox's regression model p < 0.1 was considered significant.

Results: T was detected in 31/54 pts (30/31 in the first week after MI). Univariate analysis showed that T was associated with Killip class >1 (beta = 0.6, p = 0.01), larger initial EDVi (beta = 1.7, p = 0.04) and ESVi (beta = 2.3, p = 0.002) and higher WMSi (beta = 0.7, p = 0.02). According to Cox's proportional regression model, *significant independent predictors* of T after MI were: high peak CK values (beta = 4.1, p = 0.06), initial EF ≤40% (beta = -0.8, p = 0.07), absence of MR (beta = -0.6, p = 0.06), and multivessel CAD (beta = 0.5, p = 0.06).

Conclusion: Our data demonstrate that T after anterior MI is associated with large infarcts, poor left ventricular function and multivessel CAD. Since the absence of MR is also associated with T, it appears that MR may have protective effect on T formation after anterior MI.

981

Exercise Physiology in Heart Failure

Tuesday, March 21, 1995, 3:00 p.m.–5:00 p.m.

Ernest N. Morial Convention Center, Hall E

Presentation Hour: 3:00 p.m.–4:00 p.m.

981-46

Impact of a Comprehensive Management Program on the Hospitalization Rate for Patients with Advanced Heart Failure

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Patients with advanced heart failure have a course that is often characterized by frequent hospitalizations and progressive deterioration. These patients are commonly referred to specialized centers for consideration of heart transplantation (Tx). To assess the impact of the changes in therapy made in conjunction with heart transplantation evaluation on patient outcomes, we assessed the hospitalization rate and patient's functional status in the 6 months prior to referral compared to the 6 months after referral. Since 1/91, 214 patients were evaluated, accepted for Tx, and discharged having undergone adjustments in medical therapy and a comprehensive patient education program. At time of referral patients had mean LVEF 0.21, NYHA class 3.3, VO₂ max 11.0 ml/kg, and had undergone a total of 429 hospitalizations in the previous 6 months. During evaluation patients had their ACE inhibitor dose increased by a mean 91.5 mg/day of captopril or the equivalent, were diuresed a mean 4.2 liters, were placed on a flexible regimen of loop diuretics, and were counseled on dietary management and home based progressive aerobic exercise. After 6 months of follow-up there were only 63 hospitalizations required (mean hospitalization rate per patient over the 6 months pre-evaluation 2.00 ± 1.45 vs post-evaluation 0.29 ± 0.53 p < 0.00001). Patient's NYHA class improved to 2.4 (p < 0.0001) and VO₂ max increased to 15.2 (p < 0.001). Excluding the 12 elective status Tx, 14 urgent status Tx, and 9 deaths within 6 months yielded similar results (344 pre vs 34 post-evaluation hospitalizations). 64 patients (30%) improved their functional status to the point that transplantation was deferred in favor of sustained medical therapy.

Referral to a heart failure specialty program is associated with a dramatic

reduction in the need for repeat hospitalizations and improved functional capacity for patients with advanced heart failure.

981-47 Increased Chemoreceptor Sensitivity — A Contributory Cause of Dyspnea in Chronic Heart Failure?

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The precise mechanism of dyspnea in chronic heart failure (CHF) is not known. We studied the chemosensitivity of 14 CHF patients (11 men; age 62 ± 1 [mean ± SEM] years; radionuclide left ventricular ejection fraction 29 ± 4) and 9 controls (6 men; age 56 ± 3 years) by assessing the ventilatory response to hypoxia using transient pure nitrogen inhalation and to hypercapnia using single breath inhalation of 13% carbon dioxide in air. The ventilatory response to hypoxia was significantly increased in CHF compared with normal subjects (0.67 ± 0.14 vs 0.24 ± 0.05 l/min/% SaO₂, P = 0.013) but not to hypercapnia (0.31 ± 0.03 vs 0.26 ± 0.05 l/min/mmHg CO₂, P = NS). Mean maximal oxygen consumption on cardiopulmonary exercise testing was 16.4 ± 0.8 vs 26.7 ± 1.8 ml/min/kg (P < 0.01) and the slope relating minute ventilation (V_E) and carbon dioxide production (VCO₂) was 34.1 ± 2.0 vs 25.8 ± 0.8 (P < 0.01). There was a correlation between the hypoxic ventilatory response and V_E/VCO₂ slope (r = 0.43, P = 0.043) and an inverse trend between this response and maximal oxygen consumption (r = -0.36, P = 0.099). There was no correlation between the hypoxic ventilatory response and ejection fraction (r = -0.25, P = NS).

We conclude that there is an increased chemosensitivity to hypoxia in CHF and this may contribute to the mechanism of dyspnea in this condition.

981-48 Washout Rate of Cardiac ¹²³I-Metaiodobenzylguanidine Reflects Cardiac Chronotropic Reserve and Exercise Capacity in Patients with Chronic Heart Failure

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The reduced reserve of cardiac sympathetic nerve activity is one of the important factors determining the exercise capacity in patients with chronic heart failure (CHF). Recently, ¹²³I-metaiodobenzylguanidine (MIBG), a norepinephrine analogue, has been used to evaluate the integrity of sympathetic efferent innervation. However, little is known whether MIBG imaging reflects exercise capacity and cardiac sympathetic nerve reserve in patients with CHF. The purpose of this study was to examine the correlation between quantitative markers of MIBG cardiac imaging and exercise performance in patients with CHF. Twenty-one patients with CHF (NYHA I-III) underwent MIBG cardiac scintigraphy and a symptom-limited exercise using a multistage sitting ergometer. Planar images were obtained in anterior view of the chest 15 minutes and 4 hours after injection of MIBG. The heart to mediastinum uptake ratio (H/M) on the image 4 hours after MIBG injection and the washout rate of cardiac MIBG from 15 minutes to 4 hours (WR) were assessed as markers of sympathetic nerve activity. H/M significantly correlated with % fractional shortening (r = 0.71, p < 0.001) obtained by echocardiography and the plasma level of noradrenaline (r = 0.59, p < 0.01) at rest, but not with the maximal work load, peak heart rates and the increase in heart rates during exercise. On the other hand, WR significantly correlated with the maximal work load (r = 0.70, p < 0.001), peak heart rates (r = 0.51, p < 0.05) and the increase in heart rates during exercise (r = 0.57, p < 0.01). Thus, H/M reflects cardiac dysfunction and sympathetic activity at resting state, whereas WR reflects cardiac chronotropic reserve and exercise capacity. It is, therefore, indicated that we need to take into consideration not only the myocardial uptake but also the washout rate of MIBG for monitoring sympathetic nerve activity in patients with CHF.

981-49 Post Exercise Expired Gas Analysis in Patients with Dilated Cardiomyopathy

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In normal subjects, after a maximal exercise, oxygen consumption (VO₂) declines exponentially towards baseline value. Few studies have examined the kinetics of VO₂ after exercise in patients with dilated cardiomyopathy (DCM). We studied the kinetics of post exercise VO₂ in 167 cardiopulmonary exercise tests performed in 153 ambulatory patients with DCM and in 45 controls. Patients and controls performed the same exercise (bicycle: 10 watts/min, Medical Graphic analyzer). Mean left ventricular ejection fraction was 29.8 ± 12% (mean ± SD). The VO₂-time relationship was fitted to a monoexponential curve for the determination of the time constant of recovery (tRec). We calculated the ratio between total VO₂ during exercise and during recovery (RVO₂, area under VO₂ curves), the half recovery time of peak VO₂

(1/2pVO₂), the post exercise anaerobic time (PEAT: time during recovery necessary for the respiratory exchange ratio (VCO₂/VO₂) to become <1) and the ratio between exercise duration and PEAT (RPEAT). Patients were divided in 3 subgroups according to their peak VO₂ (>15, >10 and ≤10 ml/min/kg).

Results:	Controls	>15	>10	≤10	p
n	45	88	53	26	
Duration exercise s	1299 ± 375	844 ± 171	619 ± 114	445 ± 111	<0.0001
Peak VO ₂ ml/min	2641 ± 893	1515 ± 362	995 ± 166	637 ± 176	<0.0001
Peak VCO ₂ /VO ₂	1.24 ± 0.11	1.26 ± 0.12	1.26 ± 0.12	1.28 ± 0.13	NS
RVO ₂	5.61 ± 2.48	2.9 ± 0.9	1.97 ± 0.63	1.41 ± 0.65	<0.0001
1/2pVO ₂ s	70.4 ± 29.4	105 ± 43.4	136 ± 57.7	173 ± 71.8	<0.0001
PEAT s	428 ± 147	516 ± 166	535 ± 165	544 ± 208	<0.01
RPEAT	3.5 ± 1.89	1.78 ± 0.61	1.26 ± 0.45	0.97 ± 0.54	<0.0001
tRec s	80.3 ± 26.3	128 ± 120	142 ± 73.3	204 ± 158	<0.0001

In multiple regression analysis, the parameter most closely correlated with peak VO₂ was the RPEAT (F = 127, P < 0.0001). No relation was found between age or ejection fraction and post exercise parameters. In conclusion, recovery of VO₂ is delayed in patients with DCM and is related to the degree of exercise impairment. Post exercise parameters are a novel marker for exercise intolerance.

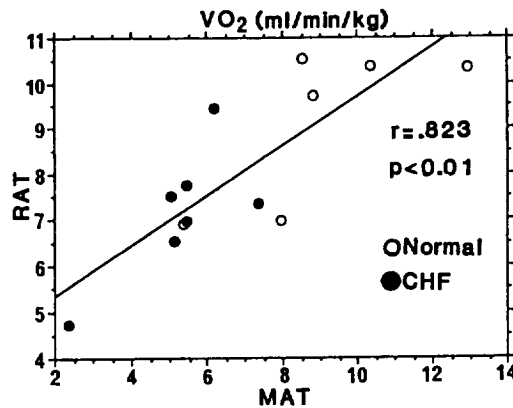
981-50 Contribution of Skeletal Muscle Fatigue versus Dyspnea to Exercise Intolerance in Chronic Heart Failure

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Background and Purpose: Decreased exercise capacity is a major clinical problem in patients with chronic heart failure (CHF) and is associated with skeletal muscle fatigue and/or dyspnea. To study the factors contributing to the exercise limitation of CHF, we observed the oxygenation of both working skeletal and respiratory muscles during exercise. **Method:** Thirteen subjects, 6 normal subjects and 7 patients with CHF, were studied. Using near-infrared (NIR) spectroscopy, relative changes in oxygenated Hb and Mb (oxy Hb/Mb) and deoxygenated Hb and Mb (deoxy Hb/Mb) contents in both vastus lateralis and serratus anterior muscles were monitored simultaneously during incremental maximal supine bicycle exercise (ramp protocol, 15 w/min, 50 cycle/min). Anaerobic threshold (AT) by respiratory gas analysis (VAT) was obtained using the V-slope method, and AT in the vastus lateralis (MAT) and the serratus anterior (RAT) muscles were determined by points obtained from deoxy Hb/Mb ≥ oxy Hb/Mb in each muscle.

Results: (1) In total subjects, VO₂ at VAT was 10.1 ± 2.41 ml/min/kg (mean ± SD), VO₂ at RAT 8.1 ± 1.80 and VO₂ at MAT 7.0 ± 2.74 (VAT vs RAT p < 0.01, VAT vs MAT p < 0.01, RAT vs MAT p < 0.05).

(2) In all of the subjects, there was significant correlation between VO₂ at VAT and that at MAT (r = 0.608, p < 0.05), while there was no significant correlation between VO₂ at VAT and that at RAT. The relations between VO₂ at MAT and that at RAT are shown on the graph.



(3) Comparisons between patients with CHF and normal subjects.

	VO ₂ /kg (ml/min/kg)		
	VAT	RAT	MAT
Normal (n = 6)	11.74 **	9.14 *	9.02 **
CHF (n = 7)	8.62	7.18	5.29

**p < 0.01, *p < 0.05.

Conclusions: During exercise, the oxygenation profile of exercising skeletal

TUESDAY PM