978-119 Abnormal Left Ventricular Volume Response to Upright Exercise in Patients with Pulmonary Hypertension: Resolution Following Lung Transplantation

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Upright exercise in normals is accompanied by an increase in indexed left ventricular end-diastolic volume (LVEDVI) which is felt to enhance cardiac output via the Frank-Starling mechanism. To assess whether an inability to increase LVEDVI during exercise may exist in pts with pulmonary hypertension (PHTN), we examined data from right heart catheterization and rest and exercise first-pass radionuclide ventriculography (RVG) in 55 pts referred for lung transplantation (LTX) evaluation. Grp 1 (n = 16; 46 ± 3 yrs, mean ± SEM) had pulmonary vascular resistance (PVR) ≥250 dynes-sec/cm^5 (mean 452 ± 44); Grp 2 (n = 39; 48 ± 2 yrs) had PVR <250 dynes-sec/cm^5 (mean 179 ± 6). Pulmonary capillary wedge pressure did not differ between groups (8.2 ± 1.0 vs. 7.2 ± 0.6 mmHg). Right ventricular ejection fraction (RVEF) at rest (0.35 ± 0.02 vs. 0.39 ± 0.01; $p < 0.05$) and exercise (0.36 ± 0.02 vs. 0.42 ± 0.01; $p < 0.005$) were lower in Grp 1. All pts reached a pulmonary limit to exercise; exercise capacity, as determined by maximal oxygen consumption or watts achieved, did not differ between groups. Rest and exercise heart rate, indexed LV stroke volume, and LVEF did not differ between groups. Rest (re) and exercise (ex) LVEDVI, and the change in LVEDVI with exercise, were as follows:

	Grp 1	Grp 2	
reLVEDVI (ml/m^2)	69.9 ± 4.4	69.6 ± 2.2 NS	
exLVEDVI (ml/m^2)	70.5 ± 3.4	82.2 ± 2.6	*
ΔLVEDVI (ml/m^2)	0.5 ± 2.5	12.6 ± 2.1	*

Grp 1 vs. Grp 2, *$p < 0.01$

19 pts (11 Grp 1, 8 Grp 2) underwent LTX with follow-up rest and exercise RVG 15 ± 2 mos later. Following LTX, there was no difference between the groups in exercise capacity or LVEF, and no longer a difference in RVEF or in the ability to increase LVEDVI with exercise. *Conclusion:* Pts with PHTN have an abnormal LVEDVI response to upright exercise which may contribute to their exercise intolerance. This abnormality resolves following LTX.

978-120 Exercise Performance in Moderate Mitral Stenosis is Limited by Cardiac Output Reserve Rather than Maximal Pulmonary Capillary Wedge Pressure

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To assess the hemodynamic limitations of exercise in patients with mitral stenosis (MS), 29 patients with MS (mean age 49, mean valve area 1.4 cm^2) and 29 age matched controls underwent symptom limited upright bicycle exercise with simultaneous expired gas analysis, right heart catheterization and radionuclide angiography. Supine rest hemodynamics and left ventricular volumes were abnormal in MS patients compared to control. Despite maximal (MAX) exercise effort, MS patients achieved a lower MAX heart rate (HR) and oxygen consumption (VO_2/kg) compared to control. With exercise, other intergroup differences were accentuated, especially for MAX cardiac index (CI), pulmonary capillary wedge pressure (PCWP), and end-diastolic volume index (EDVI).

Rest and Exercise Hemodynamics

		VO_2kg	HR	PCWP	CI	EDVI
REST	MS	2.6	75.8	19.1	2.4	51.5
	Control	3.4	68.4	8.2	3.4	76.6
MAX	MS	10.4	131.0	29.1	4.0	43.1
	Control	23.5	162.3	8.4	7.8	65.2

Mean values shown ($p \leq 0.05$ MS vs Control for all values)

For the MS group, MAX VO_2/kg was predicted by MAX CI ($r = 0.6$) but not by MAX PCWP. Exercise was limited by fatigue rather than shortness of breath in 18/29 patients in the MS group, suggesting a low output state as a reason for exercise termination. No difference was seen in MAX PCWP between patients stopping due to fatigue and those stopping due to shortness of breath (27.3 vs 32.0 mmHg, $p = 0.4$).

Conclusion: Exercise tolerance in patients with moderate mitral stenosis is not limited by MAX PCWP but by failure to augment cardiac output due to abnormal left ventricular diastolic filling and to chronotropic incompetence.

978-121 Ventricular Function During Exercise Testing and Training

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Left ventricular (LV) function deteriorates during incremental exercise (GXT) in pts with ischemia (ISCH). Responses during steady state (SS) exercise, typical of that used in exercise training, are unknown. We compared LV function in pts with documented CHD who either had (+) or did not have (−) ISCH. First pass RNA was performed during upright cycle GXT (Rest, ventilatory threshold (VT), & Max) and SS at VT (20, 30 min). RNA allowed measurement of ejection fraction (EF), end diastolic (EDV), end systolic volume (ESV) and stroke volume (SV). ISCH was mild, angina being relieved by momentary reductions in workload during SS. Results in −/+ ISCH:

	GXT			SS	
	Rest	VT	Max	20′	30′
HR	68/ 73	118/130	160/153	134/135	146/142
EF	52/ 54	59/ 51	57/ 49	61/ 50	61/ 52
EDV	176/147	203/153	210/175	195/153	181/146
ESV	86/ 72	85/ 80	94/ 95	85/ 77	78/ 73
SV	91/ 75	118/ 73	116/ 80	117/ 76	108/ 73

Although +ISCH demonstrated the expected deterioration of LV function during GXT (decreased EF, increased ESV, unchanged SV), there was no evidence for progressive deterioration of LV function during SS despite the presence of mild ISCH. We conclude that mild ischemia may be tolerated during relatively prolonged SS exercise without progressive deterioration of LV function.

978-122 Ultra Exercise Induces Right Ventricular Dysfunction

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There is concern that very extensive "ultra exercise" might lead to left ventricular (LV) dysfunction and cardiac damage. Accordingly, we studied 23 runners participating in a 100 mile race (Hardrock 100, Silverton, Colorado) at high altitude (9318 feet). Subjects had baseline echocardiograms, finger tip oximeter readings and bloods for cardiac troponin I (cTnI) and MB creatine kinase (CK). In the 14 subjects (mean age = 43 ± 8 years) who finished the race, an immediate (20–30 min after finishing) post race echocardiogram, oximeter reading and blood sample for cTnI and MBCK were obtained. Studies were repeated 24 hours later. At baseline, all oximeter readings, echocardiograms, cTnI and MBCK values were normal. Post race and at 24 hours, oximeter readings were unchanged and all cTnI values were normal except for one subject with a minor elevation. MBCK was elevated in all finishers (mean 52 ± 28 ng/ml post race) and was not related to echocardiographic abnormalities. In 5 patients with bronchospasm post race, right ventricular (RV) dilation, depressed function of the RV free wall and paradoxical septal motion was observed (a pattern of "RV pressure and volume overload"). In these subjects, systolic pulmonary artery pressure estimated from the tricuspid regurgitant jet had increased from 28 ± 2 to 55 ± 10 mmHg, $p < 0.005$. The subject with the elevated value of cTnI had severe RV dysfunction and the highest pulmonary artery pressure, 65 mmHg. No new LV wall motion abnormalities were observed. RV dysfunction resolved completely in all subjects by 24 hours post race. Subjects with bronchospasm indicated that it had occurred previously during long races. These data do not support studies that contend that ultra exercise damages the left ventricle. They do suggest that in susceptible subjects, ultra exercise can induce pulmonary hypertension that is severe enough to cause reversible RV dysfunction.

979 New Insights Into Coronary Flow Determinants

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.

979-31 Intravenous Infusion of ATP Increases Coronary Blood Flow and Improves Ischemia via Elevation of Myocardial Adenosine Levels due to Degradation of ATP

Koichi Node, Masafumi Kitakaze, Kazuo Komamura, Tetsuo Minamino, Toshinao Kurihara, Yukihiro Koretsune, Michihiko Tada, Michitoshi Inoue, Masatsugu Hori, Takenobu Kamada. *The First Dept. of Med., Osaka Univ., Osaka, Japan*

Adenosine is known to be cardioprotective for the ischemic heart, however, an intravenous administration of adenosine can not increase coronary blood

TUESDAY PM