Results:

<table>
<thead>
<tr>
<th></th>
<th>S</th>
<th>POH-NF</th>
<th>POH-F</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Wt (g)</td>
<td>1.21 ± 0.1</td>
<td>1.69 ± 0.03</td>
<td>1.65 ± 0.04²</td>
</tr>
<tr>
<td>Lung Wt (g)</td>
<td>1.03 ± 0.00</td>
<td>2.01 ± 0.05</td>
<td>4.21 ± 0.04³</td>
</tr>
<tr>
<td>LV Ees (mm Hg/100 ml)</td>
<td>620 ± 21</td>
<td>644 ± 14</td>
<td>338 ± 16³</td>
</tr>
<tr>
<td>Max Ees (g/cm²)</td>
<td>13.4 ± 3.9</td>
<td>15.1 ± 1</td>
<td>14.1 ± 0.6</td>
</tr>
<tr>
<td>V0 (ml)</td>
<td>0.125 ± 0.005</td>
<td>0.138 ± 0.002</td>
<td>0.180 ± 0.008²</td>
</tr>
<tr>
<td>ERBS (mm)</td>
<td>0.63 ± 0.35</td>
<td>0.06 ± 0.22</td>
<td>1.61 ± 0.03</td>
</tr>
</tbody>
</table>

*p < 0.01 vs S, P < 0.05 vs POH-NF and S

Conclusions: The onset of LV failure in POH appears to be more closely related to the detrimental consequences of chamber enlargement and remodeling than myocardial dysfunction.

1143-24 Does Left Ventricular Shape Change as a Function of Remodeling in Elite Athletes Involved in Predominantly Endurance Training (rowing)?

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Background: Athletic training is associated with increased LV cavity dimension, wall thickness and mass. It is still not known, however, if LV shape is altered as a consequence of this LV remodeling.

Methods: To address this, we used Fourier analysis to study LV global geometry in 22 male elite Italian rowers (25 ± 5 yrs) and 12 age matched sedentary controls (28 ± 3 yrs). By digitizing LV endocardial borders in the apical 3 chamber 2D echo view at end-diastole (ed) and end-systole (es), we derived the respective Fourier power indexes (Pl, uncleased) as a measure of the diastolic and systolic global LV shape. Higher PI represents a more elliptical ventricle, while lower value represents a more spherical ventricle.

Results (M ± SD): Elite rowers, when compared to sedentary controls, showed substantially increased LV end-diastolic volume (168 ± 23 vs. 129 ± 22 ml P < 0.001) and mass (302 ± 34 vs. 152 ± 33 g, p < 0.001), but standard deviation (sd) x 10 for rowers and sedentary group: P(67 + 6% and 54 ± 8%, P = ns). The Fourier PI did not differ in rowers and sedentary group: P(67 ± 6% and 54 ± 8%, P = ns), P(21 ± 10 vs. 15 ± 6 P = ns).

Conclusion: Elite rower athletes show a substantially enlarged LV volume and mass, but no significant changes of global diastolic and systolic LV shape. Retention of normal LV shape is consistent with the physiologic nature of the LV remodeling observed in athletes.

1143-25 Right Ventricular Volume to Mass Ratio: A 3D Echocardiographic Measure of Right Ventricular Remodeling

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Background: While conventional echocardiographic methods have characterized the left ventricular response to hypertension, analysis of right ventricular (RV) remodeling is hampered by its complicated geometry. Three-dimensional echocardiography (3D Echo) overcomes these limitations and has been recently applied to studies of RV volume and mass. We postulated that a ratio of RV volume to mass (RV VM), an inverse correlate of relative wall thickness, may relate to hemodynamic measurements in pulmonary hypertension.

Methods and Results: 3D Echo (FreeScan K3 Systems, Acuson Corp.) was performed in 9 patients (7F, 2M; mean age 36 ± 9 years) with pulmonary hypertension and correlated with pulmonary vascular resistance indexed to BSA (PVRI) and transpulmonary gradient (TPG) obtained at catheterization. Viable cells were spatially registered cross-sectional images were obtained from the pulmonic valve to the RV apex. RV volumes were calculated by a polyhedral surface reconstruction of the manually traced images. RV mass (RVM) was calculated from the difference between the end-diastolic and end-systolic volumes. Mean pulmonary pressure was 54 ± 8. Mean RVMI was 71 ± 33 g/m². There was a significant inverse correlation between RVMI and both PVRi (r = -0.75, P < 0.05) and TPG (r = -0.9, P < 0.01).

Conclusion: In this preliminary study, RV VM correlates well with measures of RV afterload and may offer insight into patterns of RV remodeling in pressure and volume overload.

1143-26 Preserved Inotropic and Lusitropic Responses to Exercise-induced Auerorenugon Stimulation in Patients With Compensated Hypertensive Left Ventricular Hypertrophy

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Background: The force- and relaxation-frequency relations by exercise-induced adrenergic stimulation have not been well examined in patients with compensated hypertensive left ventricular (LV) hypertrophy characterized by increased LV mass and normal LV function.

Methods: We measured LV pressure (high-fidelity micromanometer) during supine ergometer exercise and right atrial pacing (peak pacing: 120 bpm in 10 patients with compensated hypertensive LV hypertrophy (LVH) and both LV mass index and systolic blood pressure in patients with hypertensive LVH were greater than those in controls (178 ± 23 g/m² vs. 102 ± 19 g/m², 160 ± 11 mmHg vs. 132 ± 10 mmHg, p < 0.01, respectively). We used LV dp/dtmax and T1/2 at each heart rate to evaluate inotropic and lusitropic responses.

Results: In control group, LV dp/dtmax increased by 31% and T1/2 decreased by 14% at peak pacing, LV dp/dtmax increased by 80% and T1/2 decreased by 30% at peak exercise. In hypertensive LVH group, LV dp/dtmax increased by 19% and T1/2 decreased by 30% at peak pacing, LV dp/dtmax increased by 100% and T1/2 decreased by 44% at peak exercise (Figure). Similar changes in LV dp/dtmax/P40, a relatively load-independent parameter, were observed during pacing and exercise.

Conclusion: Patients with compensated hypertensive LVH might preserve inotropic and lusitropic responses to exercise-induced adrenergic stimulation.

1143-27 GLUT-4 Messenger RNA Expression in Right Ventricular Human Myocardium Is Reduced in Hypertensive Patients

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Background: Transmembrane glucose transport, cellular metabolism and thus contractility of cardiac and smooth muscle as well as H2O distribution in the compartments depend largely on insulin responsive GLUT-4 isoform of the transmembrane glucose transport molecule. At the level of gene expression, GLUT-4 regulation is achieved by modulating transcription rates as well as mRNA stability. Changes in cellular GLUT-4 density was also reduced in hypertensive rats.

Methods: Here we investigate probes (60-150 ng) of right atrial auricular tissue from 46 patients (25 male/21 female) of which were snap frozen in liquid NO and stored at −70°C until homogenization. T RNA was isolated using guanidium thiocyanate, phenol-chloroform extraction and alcohol precipitation (for details see our earlier paper Mol Cell Biol 8: 2394–2400, 1988). Total RNA was hybridized with 32P labelled hortal GLUT-4 cDNA and re-hybridized with a human GAPDH cDNA probe to correct for equal amounts of RNA. Quantification was performed by scanning densitometry.

Results: Our results represent the first measurements of GLUT-4 mRNA expression in human myocardial tissue. Sixteen patients had systemic hypertension (RR > 160/90 or >180/110 if over 65), 22 served as controls. GLUT-4 mRNA expression was 56.2 ± 2.5 (n = 24) in the control group and 42.5 ± 3.7 (n = 18) in the hypertensive group (P = 0.0028 ± SEM). In male patients, the difference between hypertensive and normotensive was especially pronounced: GLUT-4 mRNA was 38.0 ± 11.4 (n = 19), in the controls and 37.9 ± 9.2 (n = 6) in the hypertensive group (P = 0.0068 ± SEM).

Conclusion: Our results show that myocardial mRNA expression of the insulin-responsive GLUT-4 isoform is decreased in right auricle of patients with systemic hypertension. This suggests that altered cellular GLUT-4 expression of the he tricentric actuar may play a decisive role in the development of arterial hypertension.