146 Left ventricular stroke volume displacement anteriorly the aortic root through left atrial reservoir expansion.

G. Berra, P. Barbier, M.D. Guazzi. Centro Cardiologico Fondazione Monzino, IRCCS, Milan, Italy

Extent of aortic root (AR) systolic anterior movement has been explained as determined by left ventricular (LV) ejection and correlated to stroke volume. Further, the AR sits on the anterior left atrial (LA) wall and diastolic posterior displacement of AR has been related to LV diastolic filling and LA emptying.

Aim: Because past and recent evidence suggests a reciprocal interaction between LA reservoir function and LV stroke volume, aim of this study was that to demonstrate that the AR is directly displaced by extent of LA expansion during reservoir, as a function of LV stroke volume.

Methods: In 20 normal subjects and 80 consecutive patients undergoing diagnostic echocardiography (age 59±15) we analyzed the ability to predict anterior movement of AR, measured in parasternal view with respect to transducer position, at different LA dimensions non contiguous to the AR in the apical 4-chamber (superior-inferior, medio-laternal diameters and area) and 2-chamber (superior-inferior, antero-posterior diameter and area) views. For all dimensions, LA reservoir indexes were calculated as maximum – minimum dimension.

Results: at multiple regression analysis, reservoir expansion of 2-chamber supero-inferior and 4-chamber medio-laternal diameters, and LV bpline stroke volume predicted (p<.001) AR movement.

Conclusion: our analysis suggests that LV stroke volume influences indirectly the systolic anterior displacement of the AR through the direct influence of LA reservoir expansion.

147 Is the diastolic velocity decay from the left ventricular inflow tract to the left ventricular outflow tract affected by the systolic dysfunction?

C. Tiano1, J. Roisini2, V. Volberg2, R. Brunod2, R. Montecchi2. 1University Hospital Center, Department of Cardiology, Albania, Albania; 2University Hospital Center, Department of Cardiology, Tirana, Albania

Background: Diastolic ventricular electromechanical response to thrombolysis for acute myocardial infarction.

Methods: 59 unselected patients, 27 female, mean age 63 ± 17 years (19-91). The left ventricular ejection fraction (LVEF) (Simpson's) was 51 ± 17% (19-76). In 22 p. the LVEF was < 45%. We also measured the isovolumic relaxation time (IVRT), the E and A wave diastolic velocities at the tip of the mitral valve and at the LVOT (Er and Ar), the E deceleration time (DT), the E propagation velocity with LA reservoir function indexes and heart disease. When LA reservoir function indexes were excluded from analysis, only LV stroke volume predicted (p<.001) AR movement.

Conclusion: our analysis suggests that LV stroke volume influences indirectly the systolic anterior displacement of the AR through the direct influence of LA reservoir expansion.

Results: at multiple regression analysis, reservoir expansion of 2-chamber supero-inferior and 4-chamber medio-laternal diameters, and LV bpline stroke volume predicted (p<.001) AR movement.

Conclusion: our analysis suggests that LV stroke volume influences indirectly the systolic anterior displacement of the AR through the direct influence of LA reservoir expansion.

148 Diastolic ventricular electromechanical response to thrombolysis for acute myocardial infarction.

I.S. Ramzy1, M. Dancy2, D. Gibson2, A. Coats2, M. Henein3. 1Central Middlesex Hospital, Cardiology Dept., London, United Kingdom; 2Royal Brompton Hospital, Cardiology, Echo Dept., London, United Kingdom

Background: The effect of acute myocardial infarction (MI) on left ventricular (LV) function differs according to its location, anterior and inferior.

Aim: To study ventricular electromechanical behaviour after thrombolysis for acute MI in patients with anterior and inferior MI.

Methods: We studied 21 patients with acute MI; 11 anterior (age 52±8 years) and 10 inferior (age 59±16 years) at admission during thrombolysis and 30 days after recovery using ECG and echocardiography. Electromechanical segmental delay was taken from the end of the T wave to the onset of long axis lengthening in early diastole at different sites; anterior, posterior, lateral and septal. ST and T wave durations were compared with corresponding segmental mechanical delay.

Results: In patients with inferior MI the delayed onset of the posterior long axis lengthening, with respect to end ejection, was not different from normal 69±28 vs 65±10 ms (NS), at admission. This delay correlated closely with ST segment (r=-0.8, p<0.001) and T wave duration (r=-0.9, p<0.001). In contrast, in anterior MI infarction the onset of anterior long axis lengthening was delayed by 20ms, 80±24 vs 60±9 ms. p<.001 compared to normal. This delay became only related to ST duration 30 days after MI infarction (r=0.8, p<0.001) but not with T wave.

Conclusion: Patients with inferior MI recover their diastolic electromechanical relationship within days after thrombolysis, however with anterior infarction this relationship becomes apparent 30 days after thrombolysis. These findings suggest a significant ventricular remodelling process after thrombolysis for anterior infarction.

149 Is the slowed left ventricular relaxation or augmented atrial transport function the primary abnormality of filling in mild hypertension?

S. Qirko1, T. Goda2. 1University Hospital Center; Department of cardiology, Albania, Albania; 2Hospital de Clinicas, Hospital, Cardiology Dept., London, United Kingdom

Background: The diastolic dysfunction in the early phases of hypertension has been attributed to a primary slowing of LV relaxation, expressed by reduced Doppler E wave. The augmentation of atrial filling, manifested by an increased Doppler A wave, is considered compensatory and secondary. The aim of this study was to evaluate whether the primary abnormality of the LV filling in mild hypertension is the augmented atrial transport or the reduced LV relaxation.

Methods: 35 normotensive (NT) and 45 untreated subjects (HT) were included in the study. They were matched for all. Of any other type of cardiopathy. LV relaxation was assessed by measuring of doppler E wave and velocity and by evaluation of the mitral propagation velocity (Vp) (a load-insensitive method) measured by color M-mode echo. Atrial transport was assessed by doppler A wave velocity. LV mass index (LVM, g/m2) and LV shortening fraction (LVSHF) were measured and calculated by echo.

Results: E wave velocity, Vs, LVMI and FSH were similar for both groups. Significant difference was observed only in A wave velocity, as shown on the table.

<table>
<thead>
<tr>
<th>Relaxation</th>
<th>Atrial Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVMI (g/m2)</td>
<td>FSH (mm²/m²)</td>
</tr>
<tr>
<td>NT</td>
<td>4.27±0.95</td>
</tr>
<tr>
<td>HT</td>
<td>4.37±1.01</td>
</tr>
</tbody>
</table>

Conclusion: The augmentation of the atrial transport is rather than the impaired relaxation the earliest alteration of the LV filling in arterial hypertension.

150 Losartan improves left ventricle diastolic dysfunction in patients with hypertrophic cardiomyopathy.

A. araujo, E. Arteaga, P. Buck, B. Ianni, C. Mady. Heart Institute - Sao Paulo University, Cardiopatias Gerais, Sao Paulo, Brazil

Objective: to determine the effects of angiotensin II (Ang II) blockade on left ventricular (LV) diastolic function of patients with hypertrophic cardiomyopathy (HCM).

Background: Intestinal fibrosis impairs LV compliance in HCM. Ang II has profibrotic effects on myocardial tissue that can be inhibited by an Ang II receptor blocker, Losartan.

Methods: 35 normotensive (NT) and 45 untreated subjects (HT) were included in the study. They were matched for all. Of any other type of cardiopathy. LV relaxation was assessed by measuring of doppler E wave. The augmentation of atrial transport function the primary abnormality of filling in mild hypertension.