to verify the relationship between the LVC and CFR we tested a new index derived from the ratio of both parameters and labelled it as contractility/ perfusion index (CPI). Results: Syndrome X pts showed similar values in terms of LVC 6.5±1.4 vs 6.4±1.3 and lower values of CFR 2.3±0.5 vs 3.2±0.4. The Contractility/perfusion index had a lower mean value in the normal group (2.1±0.5) vs Syndrome X pts (2.0±0.8). By matching the CPI in all the study population, we were able to accurately predict the presence of a microvascular dysfunction with a higher statistical significance (t=4.944; p=0.000004), and therefore avoid coronary angiography in these cases.

Conclusions: Patients Syndrome X showed normal LV contractility, quite similar to normal subjects, but lower values in terms of coronary flow reserve and therefore by matching these two parameters we can obtain a new index-CPI capable of predicting the presence of microvascular dysfunction.

184 The extent of subendocardial dysfunction in hypertrophic cardiomyopathy compared with basal septal hypertrophy of similar resting gradient.

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Background: Hypertrophic cardiomyopathy (HCM) and basal septal hyper- trophic X pts (HCM-sep) cause similar impairment of left ventricular (LVMi) left ventricular mass. Besides, have been evaluated mean pulmonary artery pressure pressure (SPAP) by tricuspidal regurgitation velocity (TVR) in 187

Table 1

<table>
<thead>
<tr>
<th></th>
<th>BASELINE</th>
<th>6 MONTHS</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>EDV (mL/m²)</td>
<td>53</td>
<td>53</td>
<td>0.888</td>
</tr>
<tr>
<td>ESV (mL/m²)</td>
<td>36</td>
<td>34</td>
<td>0.017</td>
</tr>
<tr>
<td>EF (%)</td>
<td>45</td>
<td>53</td>
<td>0.000</td>
</tr>
<tr>
<td>LAD CFR</td>
<td>1.82</td>
<td>2.30</td>
<td>0.000</td>
</tr>
<tr>
<td>PDCR CFR</td>
<td>1.80</td>
<td>2.25</td>
<td>0.000</td>
</tr>
</tbody>
</table>

186 Metabolic management of heart failure with trimetazidine and mildronate: sensitivity and specificity of stress-echocardiography in patients with severe systolic dysfunction.

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Background: Trimetazidine (TMZ) and mildronate represent antianginal drugs shifting myocardial metabolism from fatty acid to glucose oxidation.

Methods: 65 patients with heart failure (>6-months after myocardial infarction) and LVEF<40% were randomly assigned to receive for a 6-month period TMZ modified release 35 mg bid (28 patients - T-group) or mildronate 500 mg bid (25 patients - M-group) additionally to standard therapy or usual drug therapy. Patients - control group (22 patients - W-group). Patients were evaluated at baseline and 6-m with clinical examination, quality of life (MLHF), low dose dobutamine stress-echocardiography (DSE).

Conclusions: Our study confirmed that in the acute phase of ABS, the underlying mechanism, whatever this may be, leads to significant CFR impairment and consequently to a reduction of left ventricle EF. After 6 months, despite normalization in terms of LVEF, CFR on LAD and PDCA remains slightly impaired as an expression of intrinsic microvascular dysfunction. This is an aspect which has never been highlighted before.

185 Apical ballooning syndrome: Can microvascular dysfunction have a pivotal role?

F. Rigo1; C. Piergentili2; G. Ossena3; V. Cutola4; A. Raviolle1
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Background: The real pathophysiological mechanism at the basis of apical ballooning syndrome (ABS) is still open to debate. The absence of critical stenosis at the angiogram opens up a new hypothesis regarding the role of vasospasm but BSH is known for its benign outcome with preserved LV systolic function.

Methods: We compared the extent of subendocardial dysfunction using dobutamine stress echocardiography by segments of 37 HCM and 28 BSH pa- tients of similar age (54.5±15.5 years 24 male; 67.1±7.7 years, 9 male), and 17 controls (58±12 years, 5 male). Systolic anterior motion (SAM) of the mitral valve was present in 40% of all patients.

Results: At rest and long axis amplitude was reduced in HCM vs BSH vs normals, but DTA Sa was significantly lower in HCM vs BSH (2.0±2.4 vs 12.2±3.5 cm/s, p<0.002). DTA Sa did not increase significantly in HCM (p=0.04 ±1.1 vs 0.0 cm, p<0.001). QRS duration was prolonged in HCM (103±28 vs 90±8 ms, p<0.001). Peak LVOT velocity was significantly increased in HCM vs BSH and controls (2.0±0.9 ms vs 1.4±0.3 ms (p<0.01) and 1.0±0.24 m/s), respectively. At peak stress, long axis amplitude did not increase in HCM and BSH vs normals, but DTA Sa was significantly lower in HCM vs BSH (8.2±2.4 vs 19.2±3.5 cm/s, p<0.002). DTA Sa did not increase significantly in HCM (0.08±0.11 to 0.13±0.15 cm, p=0.06). QRS duration increased significantly only in HCM (103±28 to 110±29 ms, p<0.008), reflecting ischemic changes. Peak LVOT velocities increased in HCM and BSH vs controls (4.3±1.67 m/s; 4.5±1.21 m/s; 1.73±1 m/s, p<0.001). Mean blood pressure (MAP) decreased significantly only in BSH (-9.3 mm Hg, p<0.02). Heart rate increased similarly in the groups (HCM =42, BSH =41, controls =43 bpm; p<0.001).

Conclusion: Left ventricular long axis function representing the suben- cardium is impaired at rest in HCM. In HCM, increase in heart rate and exer- cise intolerance may be due to development of subendocardial ischemic changes, while in BSH exercise intolerance seems to be associated with a fall in mean arterial pressure despite no change in QRS duration. Therefore, subendocardial disturbances may explain differences in the natural history of HCM and BSH.

187 Evaluation of pulmonary pressure during exercise in athletes.

Echocardiography study

A. Moretti1; M.C.R. Vono1; M. Gianassi1; L.: Toncelli1; P. Manetti1; V. Di Tante1; 1Florence, Italy

Background: athletes revealed a fall in mean arterial pressure despite no change in QRS duration. There- fore, subendocardial disturbances may explain differences in the natural history of HCM and BSH.

Methods: 37 HCM and 26 BSH patients with heart failure (>6-months after myocardial infarction) were evaluated at baseline and 6-months in T-group but not in M-group or control group. Special algorithm was implemented to compare viability results of DSE (any contractility im- provement) and radionuclide methods (perfusion/metabolism mismatch).

Original formulas were invented to convert semi-quantative DSE results from 16-segment to 20-segment model based on the squares of the corresponding segments. Low sensitivity (50,4%) and negative predictive value (43%) of DSE was found in patients with severe systolic dysfunction and heart failure in comparison with SPECT/PET criteria. At the same time high specificity (85,7%) and positive predictive value (89%) of DSE in detecting hibernated myocardium was confirmed.

Conclusions: Significant functional improvement in patients with ischemic heart failure was observed both in T-group and M-group. PET with F18-FDG showed anti-ischemic action of both drugs in ischemic segments of left ventricle. But significant improvement of regional and global left ventricular func- tion by results of SPECT and DSE was found only with therapy by TMZ. DSE showed low sensitivity in patients with severe systolic dysfunction and dia- roxide viability testing in such patients is recommended.

188 Evaluation of pulmonary pressure during exercise in athletes.

A. Moretti1; M.C.R. Vono1; M. Gianassi1; L.: Toncelli1; P. Manetti1; V. Di Tante1; 1Florence, Italy

Background: The aim of our study is the evaluation of the pulmonary pressure pressure (SPAP) by tricuspidal regurgitation velocity (TVR) during exercise in a large population of athletes and un- trained subjects.

Methods: We compared the extent of subendocardial dysfunction using dobutamine stress echocardiography by segments of 37 HCM and 28 BSH pa- tients of similar age (54.5±15.5 years 24 male; 67.1±7.7 years, 9 male), and 17 controls (58±12 years, 5 male). Systolic anterior motion (SAM) of the mitral valve was present in 40% of all patients.

Results: At rest and long axis amplitude was reduced in HCM vs BSH vs normals, but DTA Sa was significantly lower in HCM vs BSH (2.0±2.4 vs 19.2±3.5 cm/s, p<0.002). DTA Sa did not increase significantly in HCM (0.08±0.11 to 0.13±0.15 cm, p=0.06). QRS duration increased significantly only in HCM (103±28 to 110±29 ms, p<0.008), reflecting ischemic changes. Peak LVOT velocities increased in HCM and BSH vs controls (4.3±1.67 m/s; 4.5±1.21 m/s; 1.73±1 m/s, p<0.001). Mean blood pressure (MAP) decreased significantly only in BSH (-9.3 mm Hg, p<0.02). Heart rate increased similarly in the groups (HCM =42, BSH =41, controls =43 bpm; p<0.001).

Conclusion: Left ventricular long axis function representing the suben- cardium is impaired at rest in HCM. In HCM, increase in heart rate and exer- cise intolerance may be due to development of subendocardial ischemic changes, while in BSH exercise intolerance seems to be associated with a fall in mean arterial pressure despite no change in QRS duration. Therefore, subendocardial disturbances may explain differences in the natural history of HCM and BSH.

187 Evaluation of pulmonary pressure during exercise in athletes.

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