response of sympathetic and hemodynamic parameters during exercise (all p<0.05) but response after PE-CA was blunted.

Conclusion: In patients with chronic heart failure, hyperoxia did not alter mecanoreflex MSNA activation. In contrast, hyperoxia attenuates metaboreflex activation. This effect could lead to a diminished activation of the sympathetic nervous tone in heart failure and have beneficial effects.

115
Diagnostic and management of patients with heart failure with preserved systolic function in hospital settings: a French observational cohort survey

Olivier Hanon (1), Francois Dievart (2), Pascal De Groote (3), Annelore Le Maux (4), George Psica-Donose (4)
(1) Hôpital Broca, Paris, France – (2) Clinique Villette, Dunkerque, France – (3) Hôpital cardiological CHRU, Lille, France – (4) BMS, Dépt. Medical Cardiovasculaire, Rueil Malmaison, France

Background: Few data are available about real life and management of patients heart failure with preserved systolic function (HF-PSF).

Objective: To describe patients, disease characteristics and treatment strategies implemented in HF-PSF patients hospitalized for confirmed acute HF with LVEF ≥45%.

Methods: An observational study was conducted in 68 hospital in France with 2 steps: (1) a registry part in patients hospitalized for confirmed acute HF for estimation of proportion of HF-PSF patients in the overall HF patients, (2) a detailed part with data collection of patients recently diagnosed for HF-PSF (LVEF ≥ 45% evaluated within 8 days following hospitalization date).

Results: 707 HF patients were enrolled in the registry with a proportion of 51% with HF-PSF. In detailed part 364 HF-PSF patients were selected, with a mean age of 79 years, 59% female, and a mean BMI 27.7 kg/m2.

Main cardiovascular characteristics of HF-PSF patients were: atrial fibrillation (51%), coronary heart disease (32%), peripheral arterial disease (15%) and stroke (13%) as CV antecedents and hypertension (78%), obesity (45.5%) and diabetes (29%) as risk factors.

Mean values were: blood pressure 133/86 mmHg, HR 92 beats/min, creatinine 13.8 mg/L, haemoglobin 12.0 g/dl. BNP was performed in 243 patients with a median value of 684 pg/ml and NT-pro-BNP in 77 patients with a median value of 3999 pg/ml. Mean ejection fraction was 58±8%.

Non CV co-morbidities were: renal failure (39%), COPD (20%), walking disorders (22%), depression (12%), cognitive disorders (8%) and cancers (5%).

Treatment at discharge included: diuretics (85%), ACE inhibitor (57%), BB (56%), Candesartan (27%), ARB (20%), aldosterone blockers (13%), digoxin (16%), anticoagulants (32%), statins (48%), aspirin (32%) and amiodarone (28.5%).

Conclusion: Much is not known about the epidemiology and management of HF-FSP in France but our study try to improve the knowledge on this disease with real life data even if until today there is no specific treatment.

116
Cardiac remodeling and failure late after myocardial infarction is exacerbated by tissue hypothyroidism

Iordanis Mourouzis (1), Nikolaos Tsagoulis (1), Georgios Galanopoulos (1), Maria Gavra (1), Philippou Perimenis (1), Danai Spanou (1), Constantinos Pantos (1), Dennis Cokkinos (2)
(1) University of Athens, Medical School, Pharmacology, Athens, Greece – (2) Onassis Cardiac Surgery Center and Biomedical Research Foundation-Academy, Athens, Greece

Previous studies have shown that thyroid hormone (TH) signaling is altered after acute myocardial infarction or myocardial hypertrophy with potential physiological consequences. We investigated whether such changes are related to the severity of cardiac dysfunction.

Myocardial infarction was induced in rats by coronary artery ligation (AMI), while SHAM operated animals served as controls (SHAM, n=8). Both AMI and SHAM hearts were studied after 34 weeks. AMI were divided to heart failure (AMI-HF, n=6) and non-heart failure group (AMI-NHF, n=7) as assessed by the ratio of lung weight to body weight (LWG/BW) and right ventricle weight to body weight (RV/BW). Contractile function and left ventricular (LV) remodeling was assessed by echocardiography. TRβ1 was increased in both border and remote regions of viable LV of AMI-NHF (2.0 fold and 1.8 fold), while it decreased in AMI-HF (2.0 fold and 1.5 fold) vs SHAM, p<0.05. TRβ1 was reduced in border and remote regions in AMI-HF (2.8 fold and 2.0 fold) and AMI-NHF (3.6 fold and 2.0 fold) vs SHAM, p<0.05. T3 and T4 were not different between groups. Accordingly, this response corresponded to changes in cardiac function (table) and in MHC isoform expression. A marked increase in β-MHC was observed in both border and remote regions of LV of AMI-HF (76% and 75%), p<0.05 vs both SHAM and AMI-NHF while a small but significant increase in β-MHC was found in both border and remote regions of LV in AMI-NHF (62% and 60%) as compared to SHAM (48% and 50%).

<table>
<thead>
<tr>
<th></th>
<th>LGW/BW (mm)</th>
<th>RV/BW (mm)</th>
<th>LVEDD (mm)</th>
<th>LVEDS (mm)</th>
<th>EF%</th>
</tr>
</thead>
<tbody>
<tr>
<td>SHAM</td>
<td>3.77 (0.15)</td>
<td>0.35 (0.02)</td>
<td>6.9 (0.15)</td>
<td>4.0 (0.2)</td>
<td>75.6 (2.3)</td>
</tr>
<tr>
<td>AMI-NHF</td>
<td>3.95 (0.4)</td>
<td>0.43 (0.02)</td>
<td>8.8 (0.22)*</td>
<td>7.1 (0.3)*</td>
<td>41.2 (2.6)</td>
</tr>
<tr>
<td>AMI-HF</td>
<td>6.90 (0.8)**</td>
<td>0.82 (0.1)**</td>
<td>10.4 (0.50)**</td>
<td>9.2 (0.5)**</td>
<td>27 (2.0)</td>
</tr>
</tbody>
</table>

*p<0.05 vs SHAM, **p<0.05 vs SHAM and AMI-HF
Post-infarct remodeling results in tissue hypothyroidism. The occurrence of heart failure is accompanied by distinct changes in TR and MHC isoform expression corresponding to a marked regression to the fetal pattern.