heart failure (ADHF) from other syndromes. Our aim was to get insights into the pathophysiological conditions leading to altered expression of QSOX1 through the use of HF model rats.

**Methods:** Using thoracic aortic constriction (TAC), a model that rapidly progresses from cardiac hypertrophy to AHF as shown by echocardiographic and histological analysis, we analyzed the level of QSOX1 transcript. mRNA expression was measured by RT-qPCR and that of proteins by Western-Blot.

**Results:** Qsox1 mRNAs were up-regulated (x4; p<0.05) in the LV of TAC animals at 12 days post surgery, together with depressed LV shortening fraction. BNP mRNAs showed an earlier rise (x2.1; p<0.05), at 4.5 days post surgery. QSOX1 mRNA level was also up-regulated in the left atria (x2.1; p<0.05). 56 days after surgery, the time when LA hypertrophy is present and AHF is truly developed. Of note all other tissues, so far tested (liver, kidney, skeletal muscle, lung), expressed similar QSOX1 level whatever groups and time after surgery.

Furthermore unaltered QSOX1 expression was noticed in chronic HF cardiac tissues (with ejection fraction <30%) whereas BNP was elevated (x2.8; p=0.03). Thus data in human plasma that links QSOX1 to the AHF pathogenesis are corroborated in the animal models showing QSOX1 induction in both the LV and the LA in AHF rats associated with severe pulmonary congestion.

**Conclusion:** Studies in rat models show that QSOX1 expression is induced as a result of pressure overload leading to heart failure. We propose QSOX1 as a new marker for aid in diagnosis of ADHF patients.

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The effect of bisoprolol on right ventricular function in a cohort of 60 patients with chronic heart failure

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Beta-blocker use improves left ventricular ejection fraction (LVEF) in patients with heart failure. A similar effect of beta-blockers on right ventricular function has not been proposed, although the effect of Bisoprolol on right ventricular function has not been assessed.

This study investigated the short-term effect of Bisoprolol on right ventricular function in chronic heart failure patients. A cohort of 60 heart failure patients who were not taking beta-blockers at baseline was studied prospectively. Right ventricular ejection fraction (RVEF) and LVEF were measured at both baseline and 6 months by echocardiography. Various parameters of the right ventricular function were measured: Simpson RVEF, surface shortening fraction, right ventricular outflow tract (RVOT %), TAPSE (mm), S' wave with tissue doppler (S' dti cm/s), and the Tei index. The threshold of significance was fixed at 5%. Bisoprolol was up-titrated during six months by a preestablished protocol to a target dose of 10 mg/day. Mean age was 65.7±16.3 years. Baseline RVEF was 25.6±2.5% and baseline LVEF was 20.6±4.4%. Mean Bisoprolol dose reached was 25±12.5 mg daily. At 12 months, RVEF was significantly increased by 7.5% (95% confidence interval, 3.9-10.2; p=0.0001) and LVEF also increased significantly by 7.5% (95% confidence interval, 4.0-11.9%; p=0.0003). All the parameters of the right ventricular function were significantly improved.

TAPSE (15.5 vs 12.7; p=0.078), Doppler S’ dti cm/s (10.7 vs 8.2; p=0.002), Tei index (54.10 vs 81.45; p=0.0008, RVOT% (27.1 vs 19.3; p=0.036), dp/dt max (20.7±10.2 vs 34.3±15.8 cm/s, p=0.0017), and the Tei index (54.10 vs 81.45; p=0.0008, RVOT% (27.1 vs 19.3; p=0.036), dp/dt max (20.7±10.2 vs 34.3±15.8 cm/s, p=0.0017), and the Tei index (27.1±19.3; p=0.002).

The efficacy and good tolerance of bisoprolol is demonstrated in this study on chronic heart failure with right ventricular dysfunction when administered in a precise pattern.

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Emergency extra-corporeal membrane oxygenation in cardiac shock and cardiac arrest in hospital without on-site cardiac surgical facilities

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**Purpose:** Emergency extra-corporeal membrane oxygenation (ECMO) implantation for severe cardiac shock or refractory cardiac arrest (under cardiac massage), conducted in hospitals with on-site cardiac surgical facilities, has been reported as both safe and effective. We report the feasibility, in-hospital complications and outcomes of ECMO implantation, in a local hospital without on-site cardiac surgical facilities.

**Methods:** This single-centre, prospective, consecutive cohort study involved 50 pts who had ECMO implantation (2006-2010). Of these pts, 26 were in severe cardiac shock and 24 in refractory cardiac arrest. Implantations were done by an interventional cardiologist team available 24/7, and in collaboration with the nearest cardiac surgical institution, located 100 km away, to which patients were transferred subsequently.

**Results:** Stable ECMO implantation was achieved in 89% of pts in severe cardiac shock and in 79% in refractory cardiac arrest. In-hospital complications occurred in 20/23 pts in cardiac shock and 13/23 were discharged alive. In-hospital complications occurred in 18/19 pts in refractory cardiac arrest and 1/19 was discharged alive.

**Conclusion:** In a hospital with no cardiac surgical facilities, rates of implantation failure, initial complications, and hospital outcomes in pts who had ECMO implantation for severe cardiac shock or refractory cardiac arrest were concordant with previous reports from hospitals with cardiac surgical facilities. We observed a low rate of survival among implanted pts in refractory cardiac arrest.

**Table – Outcomes for patients in severe cardiac shock or refractory cardiac arrest undergoing ECMO implantation**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Severe cardiac shock</th>
<th>Refractory cardiac arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable ECMO implantation achieved (%)</td>
<td>23 (89)</td>
<td>19 (79)</td>
</tr>
<tr>
<td>Median duration of external cardiac massage, min (IQR)</td>
<td>–</td>
<td>90 (43-57)</td>
</tr>
<tr>
<td>In-hospital complications (%)</td>
<td>20/23 (87)</td>
<td>18/19 (95)</td>
</tr>
<tr>
<td>In-hospital death</td>
<td>10/23 (44)</td>
<td>18/19 (95)</td>
</tr>
</tbody>
</table>

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Peripheral chemoreflex activation contributes to sympathetic baroreflex impairment in chronic heart failure

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Chemoreflex-mediated sympathetic activation contributes to both initiation and progression of chronic heart failure (CHF).

**Aims:** to study the direct role of increased peripheral chemosensitivity in reducing sympathetic baroreflex function in CHF patients.

**Methods and results:** we compared sympathetic baroreflex function assessed by the slope of the relationship between muscle sympathetic nerve activity (MSNA) and diastolic blood pressure in CHF patients with augmented (n=20) peripheral chemosensitivity. Using a double-blind, randomized, vehicle-controlled study we examined the effect of chemoreflex deactivation (by breathing 100% oxygen for 15 min) on sympathetic baroreflex function in CHF patients with elevated and with normal chemosensitivity. Baseline MSNA was elevated (60.6±3.2 versus 48.9±3.7 bursts/min; P<0.05) and sympathetic baroreflex function impaired (3.06±0.55 vs 5.31±0.69 %bursts/mmHg, P<0.05) in CHF patients with increased peripheral chemosensitivity compared with controls. Administration of 100% oxygen led to a significant decrease in MSNA (from 60.6±3.2 to 52.6±3.2 bursts/min; P<0.001) and increase in sympathetic baroreflex function (from 2.9±0.5 to 6.1±0.7 %bursts/mmHg, P<0.05) in CHF patients with enhanced chemoreflex sensitivity. In contrast, neither room air nor 100% oxygen changed MSNA, hemodynamics or sympathetic baroreflex function in CHF patients with normal chemosensitivity.

**Conclusion:** we report for the first time that increased peripheral chemoreflex sensitivity directly decrease sympathetic baroreflex function in CHF patients. This interaction contributes to sympathetic overactivity and blunted sympathetic baroreflex function of CHF patients and may explain how chemoreceptors contribute to the bad prognosis of CHF patients.