

0.56; 95%CI [0.33-0.95]). Subgroup analysis including only OSA showed a similar result after adjustment ($p=0.017$; HR: 0.40; 95%CI [0.19-0.95]). In multivariate cox analysis including all the polygraphic variables, only CSR and minimal oxyhemoglobin saturation predicted adverse outcomes in all CHF patients untreated for SDB but AHI.

Conclusion: In patients with CHF, ventilatory treatment of SDB is associated with a better outcome independently of confounding factors.

107

Diagnostic performance of midregional proadrenomedullin in heart failure: comparison with brain natriuretic peptide levels and the echocardiographic assessment of left ventricular filling pressures

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Background: Acute decompensated heart failure is a challenging diagnosis when natriuretic peptides are misleading. Midregional-proadrenomedullin (MR-proADM) is a new prognostic peptide in heart failure.

Objectives: This preliminary study sought to assess the diagnostic accuracy of MR-proADM in heart failure in comparison with B-type natriuretic peptide (BNP) according to the echocardiographic estimation of left ventricular filling pressures (LVFP). The second purpose was to establish a cut-off value of MR-proADM between stable and decompensated heart failure.

Methods: We measured BNP and MR-proADM plasma levels in 35 consecutive patients. Concomitantly, we performed a doppler echocardiography, measuring the ratio of mitral velocity to early diastolic velocity of the mitral annulus (E/e'). 25 patients were suspected of congestive heart failure and 10 patients were suffering from stable heart failure.

Results: Ischemic and dilated cardiomyopathies were the two major causes of heart failure in our population (43% vs 31%), with only 17% of heart failure with preserved ejection fraction. Median MR-proADM and BNP levels were 0.94 nmol/L and 552 pg/mL respectively. Both plasma levels were scaling up with the NYHA classification. The correlation between the E/e' ratio and BNP levels was better than between E/e' and MR-proADM levels ($r=0.63$ vs 0.41 respectively). A mild correlation was found between BNP and MR-proADM, probably due to their shared prognostic value. MR-proADM could classify most of the patients with intermediate BNP levels according to the echocardiographic estimation of LVFP.

Conclusions: This preliminary study shows that MR-proADM may be of interest for the diagnosis of decompensated heart failure in patients with intermediate BNP levels. MR-proADM levels >0.74 nmol/L would correlate with the elevation of LVFP as determined by the E/e' ratio, and thus be useful when echocardiography is not available.

108

Metaboreflex attenuation as a potential cause of improvement in the ventilatory response after cardiac resynchronization therapy

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Background: Patients suffering from heart failure (HF) have constant exaggerated hyperventilation (high VE/VCO_2) leading to breathlessness and reduced exercise capacity. Metaboreflex over activation has been proposed to partly explain this high ventilatory response during exercise.

Objective: We aim to investigate the modification of the metaboreflex activation as a determinant in the improvement of the ventilatory response 6 months after CRT.

Methods and results: 10 HF patients (mean left ventricular ejection (LVEF) $27\pm4\%$, peak VO_2 14 ± 4 ml/kg/min, NYHA class 2.6 ± 0.5 ; QRS duration >120 ms) scheduled for CRT implantation were prospectively studied. At baseline and after 6 month follow up two maximal cardiopulmonary exercise tests with and without regional circulatory occlusion (RCO) were performed with continuous VE , respiratory ratio (RR), VCO_2 and VO_2 measurements. RCO was achieved by inflation of bilateral upper thigh tourniquets 30 mmHg above peak systolic blood pressure during 3 minutes after peak exercise as previously

described. Metaboreflex contribution to the ventilatory response was assessed as the difference in ventilatory data at the third minute during recovery between the two tests ($RCO - no\ RCO = \Delta$). At baseline, patients had enhanced VE/VCO_2 slope (40 ± 9) and an evident metaboreflex contribution to the high ventilatory response (ΔVE : 3 ± 4 L/min; $p=0.01$, ΔRR : 4.5 ± 4 /min; $p=0.01$). 6 months after CRT implantation, NYHA class, LVEF, peak VO_2 and VE/VCO_2 were significantly improved (1.5 ± 0.5 ; $p<0.001$, $42\pm7\%$; $p<0.001$, 16 ± 4 ml/kg/min; $p=0.005$; 33 ± 9 ; $p=0.001$). Metaboreflex contribution to ventilation was reduced compared with baseline (ΔVE : -1 ± 5 ; $p=0.08$, ΔRR : -1 ± 3 ; $p=0.01$).

Conclusion: 6 months after CRT metaboreflex contribution to ventilation is reduced and appears as an explanation in the ventilatory response improvement in HF population.

109

Tricuspid regurgitation changes after cardioverter-defibrillator implantation: a prospective study

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Introduction: it remains controversial whether pacemaker lead implantations could interfere with tricuspid valve function. The use of rigid and thicker ventricular leads with implantable cardioverter-defibrillators (ICD) may increase this risk. We realised a prospective study to analyse the tricuspid valve function in this setting.

Method: we included patients needing a first ICD between october 2009 and may 2010 and performed echocardiographic recordings the day before implantation, during the 3 following days and at 6 weeks. These records were analysed offline independently by 2 cardiologists to assess and grade tricuspid valve regurgitation (TR) using PISA and semi-quantitative method, and to measure ventricular dimensions, function and pressures.

Results: we included 32 patients, 29 males, aged 61 ± 14 . Hypokinetic cardiopathy accounted for 26 and 24 were implanted for primary prevention. Ejection fraction was $35\pm14\%$. Non significant TR was found in 23 patients and only 2 patients has a tricuspid regurgitation greater than mild. The only significant changes occurring immediately after defibrillator implantation concerned TR, with a mean volume raising from 3.1 ± 3.2 ml to 6.1 ± 7.0 ml ($p<0.01$) and a ROS from 0.03 ± 0.3 to 0.06 ± 0.07 cm² ($p<0.01$). Six TR changed from not significant to minime (3) or mild (3), 3 from minime to mild. Two patients with a baseline mild TR raised the regurgitant volume from 11 ml to 19 ml and 30 ml. Mean regurgitant fraction increased from 5.5% to 10.5% and was higher in patients with a low ejection fraction and mild TR at baseline. At 6 weeks, the results were similar. From a clinical standpoint, no death occurred, and no worsening of heart failure was noticed.

Conclusion: we found a significant but minor increase in TR after defibrillator implantation, with no foreseeable consequences. However, our data hint that this increase may be relevant in patients with the greater baseline regurgitation and the lower cardiac output

110

Heart failure patterns in Djibouti (Horn of Africa): an epidemiologic transition perspective

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Purpose: Cardiovascular disease patterns are changing in Africa. We aimed to document the current heart failure (HF) patterns in Djibouti.

Methods: We prospectively included Djiboutian adults hospitalized for HF in the French Military Hospital (Djibouti) between August 2008 and December 2010. Clinical and prognosis data were recorded.

Results: Among 1688 adults hospitalized in the medicine department, 45 (2.7%) had symptomatic HF: 38 (84%) males, mean age 55.8 years (range 27-75). Twenty five (56%) patients were initially hospitalized for acute pulmonary oedema. New York Heart Association (NYHA) class was 2 (40%), 3 (44%) and