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Prognostic value of cardiac metaiodobenzylguanidine in patients with advanced heart failure: relationship with peak oxygen consumption and brain natriuretic peptide

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Objectives: We sought to prospectively compare the prognostic value of cardiac iodine-123 (I-123) metaiodobenzylguanidine (MIBG) imaging with peak oxygen consumption (VO2) and plasma BNP level in patients with advanced heart failure (AHF) waiting for heart transplant.

Background: In mild to moderate heart failure, cardiac fixation of MIBG reflecting presynaptic uptake is reduced, exercise capacity is altered and plasma BNP level is increased. In AHF prognostic value of these parameters is still unknown.

Methods: Fifty one patients with advanced heart failure were studied with planar MIBG imaging, cardio-pulmonary exercise tests, hemodynamic and neurohormonal parameters. Early (30 min) and late (4 h) MIBG acquisition, as well as their ratio (washout rate, WOR) were determined. Prognostic value was assessed by survival curves (Kaplan-Meier method) and uni- and multivariate Cox analyses.

Results: Early and late cardiac MIBG uptake were correlated with ejec tion fraction (r=-0.33 and r=0.42). With a median follow up of 494 days, NYHA (p=0.03), plasma BNP (p=0.002), peak VO2 (p=0.03) were predictive of death or heart transplantation, but only plasma BNP emerged by multivariate analysis. WOR=36.63% (1st quartile) was predictive on kaplan-Meier analysis.

Conclusions: In AHF patients, VO2 and BNP plasma level are stronger prognosticicator than MIBG imaging related parameters. MIBG should be reserved to patients with mild to moderate heart failure while BNP remains the most powerful prognostic index whatever the severity of heart failure.

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Levosimendan improves hemodynamics functions without sympathetic activation in severe heart failure patients: direct evidence from sympathetic neural recording

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Background: Levosimendan is a new inodilatory agent with calcium sensitizing activity. A major concern regarding the use of inotropic agent in heart failure is their effect on the sympathetic tone. This effect could explain increase in short term mortality with other inotropes. In this setting, the aim of our study was to assess the effect of levosimendan on sympathetic tone measured directly by microneurography.

Methods: In a group of acute decompensated heart failure patients, we assessed cardiac performance by digital plethysmography (Finomter©) measurement. Sympathetic tone was assessed through recording of muscle sympathetic nerve activity by microneurography. Recording were done blindly, for each patient after dobutamine perfusion was stopped (baseline) and 48 hours after levosimendan infusion. Clinical, biological and morphological data were collected. We compared cardiac parameters and sympathetic nerve activity before and after administration of levosimendan.

Results: 13 patients with refractory chronic heart failure were recruited (aged 48±3.6 years). Systolic blood pressure and rate pressure product (mmHg x Beat/min) decreased significantly after levosimendan infusion (p<0.05). Cardiac output and stroke volume assessed by Finometer were significantly increased after levosimendan infusion (p<0.05). A significant decrease of muscle sympathetic nerve activity is observed after levosimendan infusion (p<0.01). Levosimendan cause a significant reduction of plasma brain natriuretic peptide (BNP) after the initiation of the drug (p<0.01).

Conclusion: Levosimendan induced improvement of cardiac performance is associated with a decreased in MSNA. This study show for the first time that levosimendan has no direct detrimental effect on the sympathetic nervous system.

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Significance of increased plasma BNP level in Patients with Hypertrophic Cardiomyopathy

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Background: Plasma BNP is widely used in the management of patients with systolic heart failure; however it has been less studied in patients with hypertrophic cardiomyopathy. We hypothesized that increased plasma BNP level could be associated with heart failure symptoms and increased left ventricle filling pressure in patients with HCM.

Methods: We studied 62 consecutive patients with HCM admitted for evaluation in our outpatient clinic. In all patients, clinical examination, complete echocardiography and plasma BNP and creatinine measurements were performed.

Results: The mean BNP value was 297 ± 282 pg/mL. In univariate analysis, BNP showed a significant relationship to age (R²=0.1862; p = 5.10-4), magnitude of dyspnea, expressed by New York Heart Association (NYHA) functional class: I, 114 ± 114 pg/mL; II, 224 ± 168 pg/mL; III, 523 ± 280 pg/mL; IV, 816 ± 368 pg/mL (p < 0.10-4), atrial fibrillation (p < 0.009) and creatinine clearance (R² = 0.2328; p < 10-4). BNP levels were also correlated with left atrial annular Ea velocity (R² = 0.2148; p = 10-4), increased LV filling pressure defined as E/A > 2 or E/Ea > 10 (R² = 0.376; p < 10-4), left atrial size (R² = 0.1686; p = 9.10-4), mitral lateral Ea velocity, increased LV filling pressure, and creatinine clearance.

Conclusion: BNP levels in patients with HCM are related to the presence and magnitude of heart failure symptoms and LV filling pressure. Its prognostic value should be evaluated in these patients.

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Metaboreflex is deactivated by hyperoxia in chronic heart failure

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Introduction: In healthy subject hyperoxia enhances metaboreflex sensitivity during static exercise. However the influence of chronic heart failure on metaboreflex sensitivity remains a matter of controversies. Moreover the effect of hyperoxia on this reflex in CHF patients is unknown. This is of importance since these patients regularly receive chronic administration of nasal oxygen during hospital admission.

Methods: The effects of breathing 21% (normoxia) and 100% oxygen (hyperoxia) at rest and during isometric handgrip at 30% of maximal voluntary contraction on MSNA, heart rate (HR), blood pressure (systolic, diastolic, mean and pulse pressure) and O2 saturation (SaO2) were determined in 14 patients with heart failure. The isometric handgrips were followed by 3 min of post-exercise circulation arrest (PE-CA) to allow metaboreflex activation in the absence of other reflex mechanisms.

Results: In normoxia, hangrip and PE-CA induced an expected increase in MSNA and hemodynamic parameters (BP, HR ; all p<0.05). Hyperoxia enhanced resting diastolic, mean blood pressure and SaO2 (all p<0.05), but without effects on MSNA responses. Hyperoxia did not modify expected