severe AS (valve area < 1.0 cm2, mean pressure gradient ≥ 40 mmHg) and normal EF. Medical records were reviewed for HTN, antihypertensive treatment, and syncope. All measurements and calculations were made according to the American Society of Echocardiography guidelines. LV systolic wall stress was calculated as: S = P x (1+3V/M), where P = LV systolic pressure, V = systolic volume, and M = wall mass. LV outflow impedance (which reflects resistance to ejection imposed by the combined effects of the stenotic valve and arterial pressure) was calculated as: Z = P / SVI, where P=LV systolic pressure, and SVI= stroke volume index. **Results:** A history of HTN was documented in 63 of the 89 patients with severe AS. Over a mean follow up period of 44 months, the prevalence of syncope was not different in the 62 patients with treated HTN (mean 2.2 drugs) compared to those without HTN (8 vs. 11%, p = NS). Of the 62 patients with treated HTN, those with syncope were older than those without syncope (88+/- 6 vs. 78 +/- 9 years, p = 0.02). When those with treated HTN and syncope were compared to an age and gender matched cohort without syncope there were no significant differences in stenosis severity (AVA 0.5 +/- 0.1 vs. 0.6 +/- 0.1 cm2), LVEF (60 +/- 5 vs. 66 +/- 6%), LV mass (124 +/- 11 vs. 121 +/- 20 g/m2), systemic arterial systolic pressure (141 +/- 20 vs. 141 +/- 18 mmHg), or peak LV systolic pressure (192 +/- 15 vs. 182 +/- 16 mmHg); all p=NS. There was no difference in LV peak systolic wall stress (afterload) between the two groups (563 +/- 58 vs. 533 +/- 58 grams/cm2, p=0.36). Afterload-shortening relations were similar in those with versus without syncope. By contrast, the treated hypertensive patients who experienced syncope had a lower stroke volume index (32 +/- 4 vs 40 +/- 6 mL/m2, p=0.02) and a higher valvuloarterial impedance (6.1 +/- 0.8 vs. 4.7 +/- 0.9 mmHg/ml per m2 , p<0.01) than patients without syncope. Results were similar in the 26 patients without HTN. **Conclusion:** The risk of syncope in patients with severe AS and treated HTN is low and comparable to that seen in patients with severe AS without HTN. When controlled for age and gender, syncope is not related to LV mass, afterload, or systolic function. Syncope is related to a high LV outflow impedance which is largely driven by a lower stroke volume.

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Non-Invasive Assessment of Central Venous Pressure Using Near Infrared Spectroscopy

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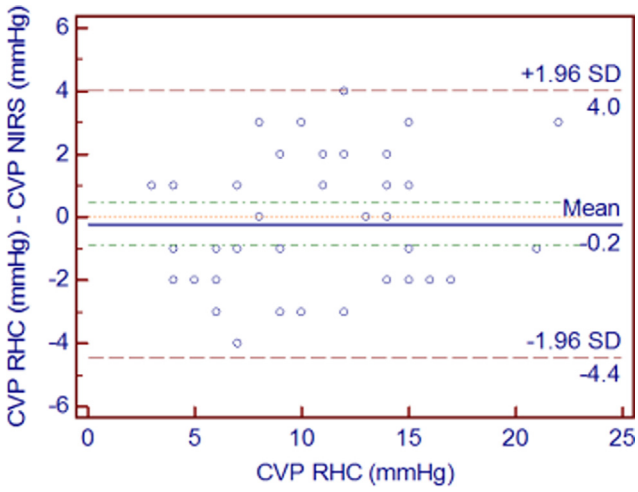
Introduction: Accurate assessment of central venous pressure (CVP) is critical when making therapeutic decisions in heart failure patients. Estimation of CVP by physical exam can be challenging and considerable inter-observer disagreement may exist. Invasive assessment of CVP by right heart catheterization (RHC) has inherent risks and can be time consuming. The objective of this study is to establish whether a novel device incorporating near infrared spectroscopy (NIRS) can provide an accurate, non-invasive assessment of CVP. **Hypothesis:** Near infrared spectroscopy will provide an accurate assessment of central venous pressure. **Methods:** The study population was composed of participants with congestive heart failure or pulmonary hypertension receiving RHC as part of their usual care at a tertiary medical center. Assessment of CVP by NIRS using the Mespere Venus 1000 CVP System (NIRS device) was performed immediately prior to RHC. Correlation of CVP obtained by the NIRS device with RHC was assessed using the Pearson's correlation coefficient (r value) and Bland-Altman technique. **Results:** Of 78 patients screened, 50 patients met study inclusion criteria. 66% of patients were male and 76% were Caucasian. The mean body mass index was 30 (± 7.1). CVP by RHC ranged from 3 to 22 mmHg. The mean left ventricular ejection fraction was 40.4% (±20.1), and 16% had at least moderate to severe tricuspid regurgitation. CVP measurement obtained non-invasively by the NIRS device correlated to CVP measured by RHC (r=0.72, p<0.001). Linear regression equation revealed an intercept of 3.42 (95% CI 1.13 - 5.71, p 0.004), and slope of 0.68 (0.49 - 0.87, p<0.0001). The median difference between the NIRS device and RHC was 0 mmHg (IQR -2 to +2). Two iterations of the standard statistical Box Plot method were applied to identify potential outliers. This identified 8 patient outliers, and after exclusion an additional analysis was performed using the resultant 42 subject dataset. The comparison of the NIRS device with RHC in this dataset demonstrated improved correlation (r=0.89. p<0.001) and linear regression revealed an intercept of 1.14 (-0.58 - 2.88, p 0.19) and slope of 0.91 (0.77 - 1.06, p<0.0001). Bland-Altman analysis, shown in the graph, demonstrated a mean difference of -0.21 mmHg. Accuracy and precision were 2.17 and 2.16 mmHg, respectively. **Conclusions:** Noninvasive assessment of the central venous pressure using near infrared spectroscopy yields readings consistently close to those measured during RHC. These data suggest that the NIRS device would be useful in patient populations where treatment decisions are dependent upon reliable assessment of central venous pressure.



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Effects of Exposure to a High Altitude on Left Ventricular Function, NT-proBNP, and Functional Capacity in Patients with Chronic Heart Failure

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Background: Exposure to a high altitude generates specific changes on hemodynamics, metabolism and functional capacity. Patients with chronic heart failure (CHF), given their especial clinical and hemodynamic features, could have a greater impact. **Objectives:** To assess the response to a high altitude exposure at a simulated 3,000 m altitude (hypobaric chamber), compared to sea level, in patients with CHF, in terms of left ventricular function parameters (LVEDV, LVESV, systolic volume, EF), NT-proBNP, vital signs (heart rate, mean blood pressure, and O_2 saturation) and 6-minute walk test. **Materials and Methods:** 20 clinically stable CHF patients were assessed, and underwent the above mentioned tests at sea level (FiO_2 21%) and at a 3,000 m altitude (FiO_2 13.1%). Left ventricular function parameters were measured by means of a Tc-99m sestamibi SPECT. NT-proBNP was also measured (Roche systems analyzer). Median values were determined and compared using the Wilcoxon test. **Results:** 20 CHF patients: 90% male; mean age 65±11 years; median EF 25%; 90% treated with beta blockers, 100% with ACEI/ARB, 40% with spironolactone; NYHA II 75%, NYHA III 25%. Parameters obtained at 3,000 m and at sea level were: mean arterial pressure [MAP] (100 mmHg vs 100 mmHg, p=NS); heart rate (70 bpm vs 72 bpm, p=0.04); 02 Sat (98% vs 90%, p=0.0001); 6-minute walk test (255 m vs 250 m, p=0.06); NT-proBNP (1235 pg/dL vs 2132 pg/dL, p=0.0006); LVEDV (233 mL vs 207 mL, p=0.06); LVESV (116 mL vs 228 mL, p=0.001); systolic volume (53 mL vs 49 mL, p=0.009); EF (25% vs 18%, p=0.005). **Conclusions:** The exposure of CHF patient to a high altitude causes: reduction of LV performance as measured by an increase of LVESV and reduction of systolic volume and EF, and significant increase of NT-proBNP expression, with no impact on functional capacity. Although the population size was small in our study, these findings suggest the need for a more careful evaluation of CHF patients before the exposure to an altitude of 3,000 m or higher.

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Do Patients with Depressed and Preserved Left Ventricular Systolic Function Exhibit Differences in Left Ventricular Function Response to High Altitude Exposure?

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Background: Chronic heart failure (CHF) patients exhibit hemodynamic and left ventricular function abnormalities that make them prone to a higher risk when exposed to a high altitude. **Objectives:** To compare a group of clinically stable CHF patients with depressed left ventricular systolic function (dLVSF) with a group of patients with preserved left ventricular systolic function (pLVSF), in terms of left ventricular function parameters measured at sea level and at a simulated altitude of 3,000 m (hypobaric chamber). **Materials and Methods:** Two groups of patients were assessed: Group A (dLVSF) and Group B (pLVSF). The cut-off point for the left ventricular ejection fraction (EF) in patients with pLVSF was 40%. The following parameters were measured both at sea level and at a 3,000 m altitude (hypobaric chamber): LVEF, LVEDV, LVESV. These parameters were measured by means of a Tc-99m sestamibi SPECT. Median values of each parameter were compared at each step in both groups (Wilcoxon test), and then between groups (Mann-Whitney test). **Results:** Group A: 20 clinically stable patients; 90% male; mean age 65±11 years; median EF 25%; 90% treated with beta blockers, 100% with ACEI/ARB, 40% with spironolactone; NYHA II 75%, NYHA III 25%. Group B: 20 patients, mean age 50.7±7 years; 70% male; median EF 54%; treated with beta blockers 30%, ACEI/ARB 40%. The following parameters were measured; Group A: sea level vs 3,000 m altitude: LVEDV (233 vs 207 mL, p=0.06), LVESV (116 vs 228 mL, p=0.001), EF (25 vs 18%, p=0.005); Group B: sea level vs 3,000 m altitude: LVEDV (100.5 vs 103.5 mL, p=0.005), LVESV (45 vs 48.5 mL, p=0.01), EF (54 vs 53%, p=NS). Comparison between Group A and Group B: LVEDV, p=0.07; LVESV, p=0.02; EF, p=0.06. **Conclusions:** Upon exposure to a simulated high altitude, compared to patients with pLVSF, patients with dLVSF exhibit a more significant impairment of left ventricular systolic performance, shown by a reduction in EF (trend) and an increase of LVESV, with no statistically significant differences in LVEDV.