ing glucose (5.98±1.02 vs 6.02±1.43 mmol/l, p=0.8353) which resulted in increase of HOMA index (2.45±2.08 vs 1.87±1.27, p=0.0300). These results suggested that the hypertensive patient who showed a morning rise in blood pressure had an advanced left ventricular hypertrophy and insulin resistance.

Key Words: Home Blood Pressure Monitoring, Left Ventricular Hypertrophy, Insulin Resistance

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INFLUENCE OF LONG-TERM OF CARVEDIOL AND METOPROLOL THERAPY ON R-R VARIABILITY IN YOUNGER AND OLDER ADULTS WITH CHRONIC HEART FAILURE

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Recent evidence shows that beta-blockers help in preventing sudden death during chronic heart failure (CHF). Our aim in this study was to examine the effect of metoprolol and carvedilol on heart rate variability in younger and older subjects with CHF. We subdivided subjects with CHF for age (younger than 65 years and 65 years or older) and submitted them to a short placebo run-in followed by they were then assigned to a 12-month course of therapy with metoprolol or carvedilol. All subjects underwent short time resting recording of RR and then power spectral analysis and calculation of the power law (PL), a measure of heart rate variability. A total 53 subjects completed the study, 32 in the younger group (15 assigned to metoprolol and 17 to carvedilol) and 22 in the older group (10 assigned to metoprolol and 11 to carvedilol). Both treatments significantly increased the PL. In older subjects both treatments induced a similar increase (metoprolol group from -1.61±0.1 to -0.99±0.1, p<0.05; carvedilol group from -1.49±0.1 to -0.94±0.1, p<0.001); in younger subjects carvedilol (from -1.34±0.07 to -0.99±0.04, p<0.05) induced a significantly higher increase (p<0.001) than metoprolol (from -1.37±0.05 to -1.27±0.05, p<0.05). Notably, both beta-blockers significantly increased heart rate variability, the PL increasing by almost 40%. Even though no specific data exist for mortality during treatment with beta-blockers in elderly patients with CHF, our data indicate that these subjects increment heart rate variability, which along with the improvement in other hemodynamic indices induced us to retain that the treatment with both beta-blockers is advantageous. Conversely, subjects younger than 65 years would benefit more from carvedilol than from metoprolol.

Key Words: Beta-Blocker, Chronic Heart Failure, Sudden Death

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INCREASED AORTIC PULSE PRESSURE ASSOCIATED WITH CONCENTRIC LEFT VENTRICULAR HYPTERTROPHY IN ARTERIAL HYPERTENSION

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In hypertensive patients, LV mass appears to be more strongly related to peripheral pulse pressure and to measurements of hemodynamic pulsatile load than to mean arterial pressure and measurements of steady load. This study evaluated the relationship between aortic pulse pressure (PP) and left ventricular mass in hypertensives with concentric left ventricular hypertrophy (LVH). We studied 14 control subjects and 28 hypertensives with normal LV mass or LVH (each n = 14). The 3 groups were matched for age, sex and body height. LVH was echocardiographically defined after correction of LV mass for the power of its allometric relation to body height. High-fidelity aortic pressure, total peripheral resistance, arterial compliance, effective arterial elastance, LV peak isovolumic and end-systolic wall stress and LV end-diastolic pressure were documented. As compared to hypertensives with normal LV mass, hypertensives with LVH had a higher systolic aortic pressure and a lower diastolic pressure, resulting in a higher PP (107±18 vs 68±17 mmHg, P<0.001), while mean aortic pressure was similar. PP was the pressure-derived variable most strongly related to LV mass (n=42; r=0.80; P<0.001) and was the only variable that could be used to discriminate between hypertensives with LVH (PP>85 mmHg) and both controls and hypertensives with normal LV mass (PP<=85mmHg), without overlap. Within each study group, there was no relationship between LV mass and PP, thus confirming that factors other than mechanical ones also play a major role in determining LV mass. Our study confirmed the strong link between LV mass and pulsatile hemodynamic load in hypertensives, and suggested that an aortic PP > 85 mmHg may help identify those hypertensives with concentric LV hypertrophy.

Key Words: Hypertrophy, Pulse Pressure, Hemodynamics

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BLOOD LYMPHOCYTE POPULATIONS IN PRIMARY PULMONARY HYPERTENSION

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Primary pulmonary hypertension (PPH) is a rare fatal disease with unknown etiology, and most of the patients die in young due to heart failure progression. There is some evidence of the immunological genesis of PPH, but the data are scant.

The aim of this study was to estimate blood lymphocyte populations in PPH. 7 PPH patients (mean age 35), NYHA functional classes II-III, were included. Blood samples were obtained, and the analysis of lymphocyte subsets was performed using “Simultest IMK-Lymphocyte” kit and FaccCalibur flow cytometer equipment (Becton Dickinson Immunocytometry Systems, CA, USA). The control group consisted of 7 patients (mean age 40) with secondary pulmonary hypertension (SPH) due to the interatrial septum defect, NYHA functional classes II-III.

We found a significant (p<0.05) two-fold decrease in natural killer (CD56+CD16+) content in PPH patients. The total number of T cells (CD3+) was greater in PPH patients mainly due to the expansion of T suppresor/cytotoxic population (CD8+). The quantities of T helper (CD4+) and B cells (CD19+) did not differ in PPH and SPH groups (Table, data are presented as mean+/SD).

We propose, that primary pulmonary hypertension may be associated with the alterations in cell immunity.

|                | CD3+ | CD4+ | CD8+ | CD19+ | CD56+16 |
|----------------|------|------|------|-------|========|
| PPH            | 2.1±0.5 | 78±4 | 41±7 | 35±9  | 9±3    | 10±2   |
| SPH            | 2.0±0.4 | 66±9 | 38±11| 28±12 | 9±2    | 22±7   |

Key Words: Primary Pulmonary Hypertension, Blood Lymphocyte Populations, NK Cells