lower in the hypertensive group with two or more anxiety symptoms than in the hypertensive group without ($p < 0.05$). In both hypertensive groups with anxiety symptoms tilt left the indexes of sympathetic modulation unchanged. In hypertensive subjects with anxiety symptoms tilt also induced a significant fall in arterial pressure ($p < 0.05$). The mean LVMI was significantly higher in the hypertensive subjects with two or more anxiety symptoms than in those without (144.7 ± 3.0 vs 133.4 ± 2.31, $p < 0.05$).

Conclusion: Normotensive and hypertensive subjects with anxiety symptoms showed increased sympathetic modulation of heart rate at rest. Hypertensive subjects with anxiety symptoms had lower indices of parasympathetic modulation of arterial pressures than subjects without anxiety. Anxiety symptoms seem to be associated with the development of left ventricular hypertrophy.

1156-67 Autonomic Dysfunction and Insulin Resistance

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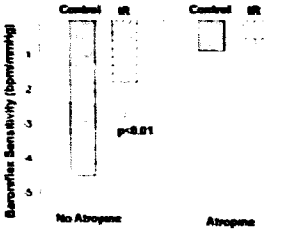
Background: Insulin resistance (IR) increases the risk of sudden death, however, the mechanism(s) remains unknown. The current study assesses baroreflex sensitivity (BS) in a rat model of IR.

Methods: Sprague Dawley rats were randomized to (1) control (C)($n = 9$) or (2) IR ($n = 14$). IR was induced by a fructose rich diet. Blood pressure (BP) was changed in conscious, unrestrained rats, to cause a baroreceptor mediated change in heart rate (HR) using phenylephrine (PE)(0.2–3 μg) or nitroprusside (NTP) (0.5–18 μg). PE induced BP changes were performed in the presence or absence of atropine.

Results: The graph reports the mean ± SEM BS for PE induced elevation in BP with or without atropine. BS without atropine was 2.5 fold lower in IR vs. C rats. This difference was due to differences in HR since BP change induced by PE was similar between groups, but maximum HR change was markedly blunted in IR vs. C (-88 ± 32 bpm vs. -238 ± 36 bpm, $p < 0.01$). Atropine negated this difference between C and IR rats. On the other hand, BS was not different between groups for NTP-induced reduction in BP



Conclusions: These data indicate that IR down regulates parasympathetic function since the difference in BS between groups was eliminated in the presence of atropine. Autonomic dysfunction is known to be arrhythmogenic and could explain the increased risk of sudden death in IR. In addition, this may also play a mechanistic role in the vascular dysfunction in IR.

1156-68 Sympathetic Hyperactivity Plays an Important Role in Blood Pressure Elevation in Obese Normotensives

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Background: It is well-known that obese subjects (O) and hypertensive patients (HT) have higher plasma norepinephrine (NE) and higher insulin (INS) levels. To evaluate the relationship between sympathetic activity, insulin sensitivity, leptin level (Lep) and blood pressure elevation (BP-E) in normotensives (NT) who gain their body weight, we conducted this longitudinal study.

Methods: Body weight (BW), BMI, BP, plasma NE, INS and Lep were measured in 603 NT men at month 0 and month 6. The subjects who had diabetes were excluded.

Results: As body weight increment (WI), no-change (NC) and BW decrement (WD) during 6 months were defined as $\geq +0.5$, $-0.5 \rightarrow +0.5$, ≤ -0.5 kg/m^2 compared to BMI at entry period, WI, NC and WD were noted in 13%, 78% and 9%. BMI and BP levels in WI at entry period was similar to those in WD, however, those in WI at month 6 were significantly greater than those in WD. At entry period, NE, INS and Lep in WI did not differ from those in NC nor WD. On the other hand, NE at month 6 were significantly greater in WI than those in WD, but INS and Lep in WI were similar to those in WD. The changes in NE (ΔNE) from entry period to month 6 in WI was greater than in WD, however, ΔINS and ΔLep in WI at month 6 did not differ from those in WD.

Conclusion: These results demonstrate that sympathetic hyperactivity, not hyperinsulinemia, plays an important role in BP elevation with increasing BMI in NT. Sympathetic hyperactivity is related with obesity-induced hypertension.

1156-69 Increase of Plasma Brain Natriuretic Peptide Precedes the Cardiac Remodeling in Primary Elderly Hypertensives

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Background: Plasma brain natriuretic peptide (BNP) has emerged as a marker for the detection of left ventricular (LV) dysfunction and remodeling. The purpose of this study was to elucidate whether BNP has a predictable value of LV remodeling in primary elderly hypertensives.

Methods: On the basis of BNP (23 ± 6 pg/ml) in 15 normotensives (NT)(67 ± 9 yr), 35 untreated elderly hypertensives (66 ± 10 yr) were divided into two groups with normal BNP (29 ± 14 pg/ml) (HT-I; $n = 24$) and increased BNP (66 ± 22 pg/ml, $p < 0.05$ vs NT) (HT-II; $n = 11$) at baseline. At a mean follow up of 7.9 ± 1.6 months, LV function and geometry were echocardiographically compared with the baseline data.

Results: There were no significant differences in mean blood pressure (BP) and LV mass index (LVMi) at baseline between HT-I and HT-II (BP, 106 ± 12 vs 104 ± 12 mmHg; LVMi, 100 ± 18 vs 104 ± 16 g/m^2). Neither LV fractional shortening (%FS) nor the early to late velocity ratio of transmitral Doppler flow (E/A) were different between HT-I and HT-II (%FS, 41 ± 5 vs 43 ± 5%; E/A, 0.86 ± 0.33 vs 0.82 ± 0.16). At the follow-up period, BP, LV function and BNP were relatively unchanged in both groups. However, in contrast to HT-I (LVMi, 101 ± 17 g/m^2), HT-II showed the significant increase in LVMi (120 ± 15 g/m^2, $p < 0.05$).

Conclusion: Increase of BNP has a predictable value of the cardiac remodeling in primary elderly hypertensives.

1156-70 Brain Natriuretic Peptide as a Diagnostic Marker of Ventricular Hypertrophy – Comparison With the Electrocardiogram

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The presence of LV hypertrophy (LVH) has recognized prognostic implications in patients with cardiovascular disease. The ECG is specific but very insensitive for the detection of LVH. Echocardiography is sensitive but expensive. An inexpensive diagnostic marker of LVH is needed. Enhanced expression of atrial (ANP) and brain (BNP) natriuretic peptide occurs early in the hypertrophic response and the natriuretic peptides may be diagnostic markers of LVH. We measured BNP and C-terminal (C-ANP) and N-terminal (N-ANP) ANP in 264 pts undergoing echocardiography in who measurement of LV mass index (LVMI) by M-mode echocardiography (ASE convention) was possible. A history of hypertension was present in 50% of pts. A history of coronary artery disease was present in 19% of pts. By echo, LVH (LVMI >131 g/m2 in males and LVMI >100 g/m2 in females) was present in 56 pts. Receiver operating characteristic (ROC) analysis was performed. The area under the ROC curve for the detection of LVH was 0.72 for BNP which was larger than that for N ANP = 0.65 or C-ANP = 0.64 ($p < 0.05$). The correlation between LVMI and in BNP was significant ($r = 0.37$, $p < 0.000$) and stronger than that for N-ANP or C-ANP. The sensitivity and specificity of a BNP >37 pg/ml (mean + 2SD in normals) for the detection of LVH were 64% and 72% as compared to 11% and 96% for the ECG. BNP is much more sensitive than the ECG and represents an attractive alternative, inexpensive method to screen for LVH.

1157 Electrophysiology in the Elderly

Tuesday, March 31, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 4:00 p.m.–5:00 p.m.

1157-71 Outcome of Cardiopulmonary Resuscitation After In-hospital Cardiac Arrest in Octogenarians

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Purpose: We sought to investigate the outcome of cardiopulmonary resuscitation (CPR) after in-hospital cardiac arrest in octogenarians.

Methods: A retrospective review was conducted of 91,372 hospital discharges from 1/1/93 until 6/30/96. Cardiac arrest was reported in 956 patients. Patients <80 years old (average 64 years, $n = 482$) were compared to those