Thus when both smoke and hypertension impaired EDV but not NEDV. However when combined NEDV was also impaired. Changes in brachial and carotid arteries thickness as well as in heart function and structure was observed only in hypertensives.

In conclusion: Smoke impairment function but not alter vessel structure while hypertension induces both: function impairment and changes in structure of vessels and heart.

Key Words: Endothelial function; intimal-media thickness; essential hypertension; smoke

**F030**

**EFFECTS OF HYPERTENSION AND DIABETES MELLITUS UPON FUNCTION AND STRUCTURE OF HEART AND VESSELS**


In order to assess the impact of cardiovascular risk factors such as hypertension and diabetes mellitus upon functional and structural parameters of vessels and heart we studied normotensives (NT); essential hypertensives (HT), normotensives with type II diabetes (NT-DM) and hypertensives with diabetes type II (HT+DM) paired by age and gender. It was measured by the high resolution B-mode ultrasound the intimal-media thickness of brachial artery (IMT-Ba) and carotid artery (IMT-Ca) and by the atrial desacceleration time (ADT) and the left ventricular mass index (LVMi).

**Results:** Mean ± SEM EDV and NEDV: % changes in brachial artery diameter. *p < 0.01 vs NT

<table>
<thead>
<tr>
<th></th>
<th>NT (n = 50)</th>
<th>NT-DM</th>
<th>HT (n = 13)</th>
<th>HT-DM (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV (%)</td>
<td>17.1 ± 1.3</td>
<td>7.7 ± 2.1*</td>
<td>8.5 ± 2.4*</td>
<td>5.6 ± 1.5**</td>
</tr>
<tr>
<td>NEDV (%)</td>
<td>14.0 ± 1.6</td>
<td>9.6 ± 1.7</td>
<td>10.2 ± 2.1</td>
<td>4.2 ± 1.7**</td>
</tr>
<tr>
<td>IMT-Ca (mm)</td>
<td>0.056 ± 0.05</td>
<td>0.06 ± 0.05</td>
<td>0.070 ± 0.009*</td>
<td>0.067 ± 0.004*</td>
</tr>
<tr>
<td>IMT-Ba (mm)</td>
<td>0.024 ± 0.001</td>
<td>0.033 ± 0.004*</td>
<td>0.032 ± 0.001*</td>
<td>0.034 ± 0.002*</td>
</tr>
<tr>
<td>E/A</td>
<td>1.54 ± 0.10</td>
<td>1.41 ± 0.08</td>
<td>1.12 ± 0.09*</td>
<td>0.85 ± 0.13**</td>
</tr>
<tr>
<td>ADT (ms)</td>
<td>183.1 ± 9.2</td>
<td>196.6 ± 9.63</td>
<td>201.1 ± 14.4*</td>
<td>226.4 ± 21.6*</td>
</tr>
<tr>
<td>LVMi (g/m²)</td>
<td>75.1 ± 3.1</td>
<td>81.5 ± 5.3</td>
<td>88.4 ± 5.1</td>
<td>95.9 ± 16.5*</td>
</tr>
</tbody>
</table>

Thus hypertension and diabetes alone impaired EDV but not NEDV. However when DM and HT are associated EDV was further impaired and in NEDV was also observed. Diabetes and hypertension also alters structure of vessel.

Key Words: Diabetes; endothelial function; intima-media thickness; essential hypertension

**F031**

**ATORVASTATIN AND VITAMIN C ON ENDOTHelial FUNCTION OF HYPERCHOLESTEROLEMIC PATIENTS**

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The aims of present study were to test in a group of hypercholesterolemic outpatients the effects of vitamin C and atorvastatin treatment on endothelium-dependent and endothelium-independent vasodilation. The responses of the forearm vasculature to acetylcholine (ACh) (7.5, 15 and 30 µg/min), sodium nitroprusside (SNP) (0.8, 1.6, 3.2 µg/min) and L-NMMA (2, 4, 8 µmol/min) were evaluated in 12 normal volunteers (seven men and five women, aged 20 to 45 years), and in 18 hypercholesterolemic patients (ten men and eight women, aged 20 to 46 years) at baseline and after one month of atorvastatin (10 mg/day) treatment. Similarly, we evaluated the antioxidant effects of vitamin C (24 mg/min) at baseline and after lipid-lowering therapy. Drugs were infused into the brachial artery, and forearm blood flow (FBF) was measured by strain-gauge plethysmography. At baseline, the response to ACh was significantly attenuated in hypercholesterolemic vs controls: at the highest dose (30 µg/min), FBF was 27.0 ± 3.4 vs 11.5 ± 1.9 mL 100 mL tissue−1 min−1 respectively (p < 0.0001). No significant differences were found between groups during SNP infusion. The atorvastatin treatment significantly improved ACh-stimulated FBF: at highest dose the FBF increased to 14.9 ± 1.5 mL 100 mL tissue−1 min−1 (p < 0.0001). Similarly, the L-NMMA endothelial effects were significantly potentiated by lipid-lowering treatment, suggesting the improvement of basal nitric oxide. Vitamin C increased ACh-vasodilation in the same way before and after atorvastatin treatment. The endothelial dysfunction in hypercholesterolemics is due to an oxidative stress, and atorvastatin rapidly improves both basal and stimulated endothelium-dependent vasodilation.

Key Words: Hypercholesterolemia; endothelium; atorvastatin; atherosclerosis; vitamin C

**F032**

**SOLUBLE E-SELECTIN IS INCREASED IN CONCENTRIC LEFT VENTRICULAR HYPERTROPHY AND IS RELATED TO SEPTAL WALL THICKNESS**

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The aim of this study was to examine whether the serum soluble E-selectin (sE-selectin) is increased in symptomatic patients with concentric and non-concentric left ventricular hypertrophy (LVH) and to determine whether sE-selectin is related to septal wall thickness.

**Results:** Mean ± SEM sE-selectin in patients with concentric LVH was significantly higher than in patients with non-concentric LVH (17.1 ± 3.1 vs 10.9 ± 2.6 ng/mL, p < 0.05). In addition, the group with concentric LVH had a significantly higher septal wall thickness than the group with non-concentric LVH (1.4 ± 0.1 vs 1.0 ± 0.08 cm, p < 0.05).

Key Words: Hypercholesterolemia; endothelium; atorvastatin; atherosclerosis; vitamin C
Increased plasma levels of cell adhesion molecules (CAMS) have been associated with endothelial dysfunction and atherosclerosis. In the present study we investigated CAMS in hypertensive patients with left ventricular hypertrophy (LVH). Serum levels of E-selectin, intercellular adhesion molecule-1 (ICAM-1) and vascular adhesion molecule-1 (VCAM-1) were measured in 115 hypertensive patients (age 54 ± 9) with LVH (HT-LVH), and in two age and gender matched control groups consisting of 38 hypertensives without LVH (HT-no LVH) and 38 normotensives (NT). E-selectin was increased in all hypertensives (HT) compared to NT (p = 0.033). Furthermore E-selectin was higher in concentric than in eccentric LVH (60 ± 20 vs 51 ± 18, p = 0.032) and related to septal wall thickness (r = 0.20, p = 0.007). There was no differences in ICAM-1 and VCAM-1 between the groups. Blood pressure was not related to CAMS.

Soluble E-selectin, but not ICAM-1 or VCAM-1, is elevated in hypertensive patients, and is higher in concentric LVH and positively related to septal wall thickness. Thus, concentric LVH show signs of endothelial dysfunction without other indices of increased inflammatory activity.

Key Words: Endothelial dysfunction; left ventricular hypertrophy; selectin; VCAM-1; ICAM-1

F033
REDUCED NITRIC OXIDE (NO) EXCRETION DURING HIGH SALT INTAKE IN SALT SENSITIVE INDIVIDUALS
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Studies in laboratory animals indicate that altered nitric oxide (NO) production may be associated with salt sensitivity (SS). In this study we evaluated if endogenous NO production was altered in SS human subjects. SS was assessed from the magnitude of the BP lowering obtained when the salt intake was reduced from high to a low intake. The combined urinary excretion of nitrates and nitrates, the major metabolites of NO, was employed as an index of endogenous NO production. SS subjects (n = 23) were older, heavier and had greater waist-to-hip ratios and higher baseline BPs than salt-resistant individuals (n = 25). In SS subjects, MBP decreased 11.8 ± 0.7 mmHg, and NO-metabolite excretion increased from 823 ± 102 to 1530 ± 148 mmoles/24 hr, when salt intake was reduced from 316 to 28 μmoles/day. NO-metabolite excretion was 45% lower during high salt (0.66 ± 0.1 μmoles/mg creatinine) than during low salt intake (1.12 ± 0.1 μmoles/mg creatinine) (P < 0.001).

In contrast, when salt intake was reduced, salt-resistant subjects exhibited no significant changes in BP or NO-metabolite excretion. During low salt intake, NO-metabolite excretion (μmoles/day) was significantly higher in SS. The magnitude of decrease of SBP, DBP or MBP induced by reducing salt intake was not related to the increase in urinary excretion of NO-metabolites levels (r² = 0.009; P = 0.66). In summary, to the extent that urinary NO metabolite levels reflect the activity of the endogenous NO system, our results support the view that SS may in part be determined by an inability to increase or to sustain NO production in response to high salt.

Key Words: Nitric oxide; salt sensitivity; salt intake; blood pressure

F034
CHANGES IN NITRIC OXIDE SYNTHASE, NITRIC OXIDE, LIPID PEROXIDATION AND MELATONIN IN HYPERTENSIVE PATIENTS TREATED WITH LACIDIPINE
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Objective: Arterial hypertension (AH) produces changes in the nitric oxide synthase (NOS)/nitric oxide (NO) system. NO produces peroxinitrites and hydroxyl radicals that, in turn, induce peroxidation of membrane lipids (LPO). On the other hand, melatonin has a free radical scavenger and its protective role as antioxidant is well stabilised. The purpose of this study was to evaluate: (i) changes in blood levels of NOS, NO and LPO in patients with light and moderate hypertension; (ii) the effects of six month treatment with lacidipine on these parameters; and (iii) the relationship between melatonin levels and AH before and after lacidipine treatment.

Design and methods: Two groups were done: Hypertensive group composing 14 patients and a control group of 11 healthy, age, weight and sex matched subjects. Melatonin, LPO and nitrates were measured on plasma of these patients, whereas iNOS activity was determined in macrophages.

Results:

<table>
<thead>
<tr>
<th>NO</th>
<th>iNOS</th>
<th>LPO</th>
<th>Melatonin</th>
</tr>
</thead>
<tbody>
<tr>
<td>(μmol/l)</td>
<td>(pmol/mg prot)</td>
<td>(μmol/ml)</td>
<td>(μg/ml)</td>
</tr>
<tr>
<td>Controls</td>
<td>0.83 ± 1.0</td>
<td>0.07 ± 0.2</td>
<td>23.0 ± 7.9</td>
</tr>
<tr>
<td>AH pre Treatment</td>
<td>2.6 ± 1.4</td>
<td>19.0 ± 20</td>
<td>77.9 ± 42</td>
</tr>
<tr>
<td>AH post Treatment</td>
<td>2.5 ± 1.3 ns</td>
<td>0.16 ± 0.4*</td>
<td>26.9 ± 18.1*</td>
</tr>
</tbody>
</table>

*P < 0.001 vs pretreatment; ns: not significant.

Conclusions: In the group of AH treated with lacidipine, a significant decrease of iNOS activity and LPO levels together with the recovery of normal blood pressure, were found. The decrease of iNOS activity without changes in circulating NO levels suggests an increase in constitutive NOS activity. Moreover, hypertensive patients showed higher melatonin levels compared to those found after lacidipine treatment. The possible implications of these results will be discussed.

Key Words: Nitric oxide; nitric oxide synthase; lipid peroxidation; melatonin