parameters included in the study. After the follow-up the BP evolution (IP: −18.6/−9.7, C: −17.6/−10.1 mmHg), the percentage of EH controlled (IP: 44.7%; C: 40.6%), the weight loss (IP: −1.28 Kg; C: −1.20 Kg), the reduction in alcohol consumption (IP: −3.5; C: −2.6 g/day) and the increase of physical exercise (IP: 28; C: 25%) were similar in both groups. However EH that either reduced their alcohol consumption or increased their physical activity level exhibit higher reductions of BP.

Our results indicate the failure of the IP to modify the dietary habits and lifestyle of the patients. However, the efficacy of amlodipine was similar in EH following or not the IP, and it was greater in EH that either reduced the alcohol consumption or increased their physical activity.

Key Words: Non-pharmacological intervention; antihypertensive drugs

A093
THE EFFECT OF ANTIHYPERTENSIVE THERAPY ON 24-HOUR BLOOD PRESSURE PROFILE
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A growing body of evidence indicates that hypertensive subjects, whose blood pressure (BP) decreases at night (dippers), have a lower risk of cardiovascular events than nondippers with a similar level of BP. However, it is not known how antihypertensive therapy affects 24-hour BP profile. The aim of our study was to assess the effect of antihypertensive therapy on the dipping status.

We examined the frequency of dipping in 396 (217 untreated and 179 treated) subjects referred for 24-hour ambulatory BP monitoring to our institution. Dipping was defined as nocturnal (10PM–7AM) decrease of 10 mmHg in systolic BP relative to daytime (7AM–10PM) values.

Higher daytime BP was associated with a trend towards higher frequency of dipping in untreated subjects. There was no relationship between the level of BP and dipping in treated subjects. There were significantly fewer dippers in the treated group than in untreated one (table). The association between antihypertensive therapy and non-dipping remained significant after adjustment for age (odds ratio 1.5, P = 0.047).

<table>
<thead>
<tr>
<th></th>
<th>Treated</th>
<th>Untreated</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dippers</td>
<td>52%</td>
<td>65%</td>
<td>0.012</td>
</tr>
<tr>
<td>Age, yr</td>
<td>61.4 ± 13.1</td>
<td>51.9 ± 15.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>77/102</td>
<td>100/117</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.2 ± 4.4</td>
<td>25.7 ± 4.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

In 109 subjects receiving monotherapy, use of a diuretic was associated with a trend towards more dippers than use of β-blockers, calcium channel blockers or angiotensin converting enzyme inhibitors (71%, 53%, 52% and 53%, respectively). This trend did not reach statistical significance.

In conclusion, antihypertensive therapy is associated with decreased nocturnal blood pressure dipping. Diuretics may be associated with less decrease in dipping than other agents. This may potentially result in a better outcome in hypertensive individuals treated with diuretics.

Key Words: Antihypertensive therapy; dipping; ambulatory BP monitoring

A094
LONGTERM FOSINOPRIL EFFECT ON ADHESION MOLECULES AND PROINFLAMMATORY CYTOKINES IN HYPERTENSIVE PATIENTS
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In our previous study hypertensive patients without treatment had statistically higher ICAM-1 and GM-CSF levels compared to controls. These findings might imply that endothelial cells from patients with essential hypertension are activated at the proinflammatory level. To investigate if adequate treatment of hypertension induces reversibility of this effect, we studied the influence of 20 mg fosinopril daily for three months in 15 (49 ± 8 years of age) newly diagnosed, uncomplicated patients with essential hypertension. All patients were free of overt atherosclerosis, diabetes and dyslipidemia. Levels of ICAM-1 and GM-CSF were measured in the supernatants of phytohemagglutinin-cultured peripheral blood monocytes before the institution and three months after treatment with fosinopril. Fosinopril decreased significantly systolic blood pressure (p < 0.0001, 176 ± 14.9 vs 149 ± 10.5) and diastolic blood pressure (p < 0.002, 93.3 ± 9.3 vs 85.8 ± 5.6). There was a posttreatment statistically significant decrease of ICAM-1 levels (p < 0.03, 427.7 ± 120.7 vs 364.2 ± 78.2) and a decrease in GM-CSF, not reaching though significant value (p < 0.2, 7.66 ± 5.79 vs 6.82 ± 4.78). This ACEi treatment probably affects adhesion molecule production but is insufficient to inhibit hypertension-induced proinflammatory process.

Key Words: ICAM-1; GM-CSF; Fosinopril

A095
CHRONIC TREATMENT WITH ACE-INHIBITORS INDUCES LEFT VENTRICULAR CHANGES INDEPENDENTLY OF BLOOD PRESSURE DECREASE
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Using 24-h ambulatory blood pressure monitoring (ABPM) and digitized M-mode echocardiography, we evaluated the left ventricular (LV) morpho-functional changes induced by chronic treatment with ACE-inhibitors (AEI) ineffective in reducing blood pressure (BP).

After at least 18 months from the evaluation before treatment, we checked the essential hypertensives (H) (24-hour BP > 140 and/or 90 mmHg) lost at follow-up. Among them we found 21 H who have regularly taken the prescribed ACEi, but at the 2nd evaluation had BP not controlled and similar to pretreatment values. We evaluated: 24-h, daytime, nighttime systolic, diastolic BP and heart rate, % nocturnal fall of BP, LV end-diastolic diameter (DD), LV mass.
index (LVMi), peak shortening (−dD/dt) and peak lengthening rate (+dD/dt) of LV diameter, peak thinning rate of LV posterior wall (dD/dt).

Mean interval length between 1st and 2nd evaluation was 28 ± 7 months. BMI and heart rate (24-h, day, night) were unchanged, as were all BP parameters. LVDD and peak shortening rate of LV diameter, index of systolic function, were normal in all and did not change. At the basal evaluation 9 pts. had LV hypertrophy (LVMi > 130 g/m² men, >110 g/m² women) and 12 pts. had impaired diastolic function (+dD/dt < 3.6 sec⁻¹ and/or dD/dt < 8.5 cm/sec); at the 2nd evaluation 4 pts had LV hypertrophy and 5 diastolic dysfunction.

<table>
<thead>
<tr>
<th>SBP24</th>
<th>DBP24</th>
<th>SBPd</th>
<th>DBPd</th>
<th>SBPn</th>
<th>DBPn</th>
<th>LVMi</th>
<th>+dD/dt</th>
<th>dD/dt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>148 ± 13</td>
<td>92 ± 9</td>
<td>156 ± 12</td>
<td>98 ± 9</td>
<td>135 ± 16</td>
<td>84 ± 9</td>
<td>124 ± 29</td>
<td>3.8 ± 1.4</td>
</tr>
<tr>
<td>2nd</td>
<td>147 ± 12</td>
<td>93 ± 8</td>
<td>154 ± 11</td>
<td>97 ± 8</td>
<td>134 ± 16</td>
<td>86 ± 8</td>
<td>100 ± 30</td>
<td>4.9 ± 1.4</td>
</tr>
</tbody>
</table>

*p < 0.001.

The difference between 1st and 2nd evaluation as regards LV diastolic parameters lost the statistical significance after correction for LVMi values.

In conclusion, despite the lack of BP decrease, chronic treatment with ACEi influences the LV, with reduction of LV mass and improvement of LV diastolic function. These effects of ACEi, not due to BP reduction, have therefore to be ascribed to a direct action of the drugs on the myocardium.

Key Words: ACE inhibitors; diastolic function; myocardial hypertrophy

A096

ANGIOTENSIN II RECEPTOR BLOCKADE: ROLE OF BRADYKININ

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Angiotensin II (AII) is a potent vasopressor peptide and has been implicated as a pathogenetic factor in hypertension. Inhibition of AII has therefore been considered a therapeutic approach to treat hypertension. AII has two major receptor isoforms, AT1 and AT2. The AT1 is responsible for the majority of the known effects of AII, much less is known about the AT2 receptor stimulation.

The aim of this study was to evaluate whether Telmisartan and Irbesartan two selective antagonist of AT1 receptors could play a protective role for the heart and kidneys.

In one experimental model of hypertension (AII-induced hypertension with osmotic pumps) the AT1 receptor antagonist was not only an effective antihypertensive agent (162 ± 5 mmHg in HT vs. 110 ± 4 in HT-treated, p < 0.01) but also reduced proteinuria (78 ± 9 mg/d in HT vs. 26 ± 5 in HT-treated, p < 0.01). In angiotensin-induced hypertension as well as in one experimental model of chronic heart failure due to aorto-caval shunt, both AT1 antagonist significantly protect against cardiac hypertrophy (1.82 ± 12 g in shunt vs. 1.28 ± 7 g in shunt-treated, p < 0.01). This cardioprotective effect was abolished by the kinin B2 receptor antagonist, icatibant. This would favour the contention that the blockade of the AT1-mediated effects of angiotensin explains the antihypertensive action, leaving the AT2 receptor unopposed. We conclude that the organ-protective actions of the AT1 receptor antagonist may involve AT2 and bradykinin B2-receptors mediated effects, perhaps acting via release of NO or hyperpolarization caused by activation of potassium channels.

Key Words: Angiotensin; receptors; cardiovascular protection; hypertension

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A097

MODERN POSSIBILITIES OF THE ARTERIAL HYPERTENSION MANAGEMENT IN HYPERLIPIDEMIC PATIENTS

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It has been widely assumed that hypertension may accelerate the development of atherosclerosis. Thus it is important to use antihypertensives drugs without negative effects on lipid metabolism. The aim of our study was to estimate the antihypertensive efficacy angiotensin II receptor blocker irbesartan and its influence on lipid metabolism in hyperlipidemic hypertensives. Twenty patients (8 m, 12 f, mean age 55.8 ± 7.8 years) with mild to moderate arterial hypertension and hyperlipidemia (IIA, IIB by Fredrickson) were included into the study. Irbesartan administered in doses 150–300 mg once daily during 12 weeks. Ambulatory blood pressure monitoring (ABPM) and lipid profile were estimated at the baseline, after 4, 8 and 12 weeks of the treatment. Daytime/nighttime systolic (SBP) and diastolic blood pressure (DBP) according to 24-h ABPM were 160.1 ± 8.3/148.6 ± 13.4 mm Hg and 98.2 ± 5.3/86.8 ± 7.2 mm Hg at the baseline, respectively. The main values of lipid parameters before the treatment were followed: triglycerides (TG)—2.7 ± 0.6 mmol/l, total cholesterol (TC)—6.9 ± 1.0 mmol/l, high-density lipoprotein cholesterol (HDL-C)—1.0 ± 0.2 mmol/l, low-density lipoprotein cholesterol (LDL-C)—4.6 ± 0.9 mmol/l. After the treatment daytime/nighttime SBP and DBP reduced by 15%/18.2% (p < 0.001) and 14.1%/17.7% (p < 0.001), respectively. Besides that, reduction of high measurements of BP variability with remaining of its normal values under the treatment were demonstrated. There were no noted negative changes in lipid parameters under irbesartan treatment. Furthermore the tendency to decrease of TG and TC levels was revealed. Irbesartan was well tolerated. Adverse events (headache, fatigue) were noted in 15% of patients and discontinued without cancel of drug. So we conclude that irbesartan is effective antihypertensive agent with a positive influence on BP variability and can be recommended for using in hyperlipidemic hypertensives.

Key Words: Angiotensin II receptor blocker; ABPM; lipid profile