the roles and efficacy of pharmacologic and non-pharmacologic therapies.

Key Words: children, essential hypertension, ambulatory blood pressure monitoring

P-627
PROLONGED USE OF AMLODIPINE IN CHILDREN WITH HYPERTENSION
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Amlodipine (AM) is a long-acting lipophilic calcium antagonist (CA) with unique pharmacologic properties that have led to its widespread use for treatment of hypertensive children (C). While several reports of pediatric AM use have been published, all have focused on demonstration of short-term AM efficacy and safety. To examine the long-term tolerability and efficacy of AM in C, data on prolonged AM use (≥6 months) in 33 hypertensive C were reviewed. All C received AM as sole therapy for their hypertension (HTN). Causes of HTN were solid organ transplant (19 C), renal diseases (8 C) and primary HTN (6 C). Mean patient age at start of AM treatment was 118±58 months (mean±SD), range 16 - 203 months. 19/33 C (58%) were boys. Duration of AM treatment averaged 20.4±11.5 months (range 6-48 months). No patient required discontinuation of AM because of adverse effects. Analysis of BP and dosing data revealed that blood pressure reduction was sustained required discontinuation of AM because of adverse effects. Analysis of BP and dosing data revealed that blood pressure reduction was sustained throughout the period of AM treatment, while AM dose remained stable:

<table>
<thead>
<tr>
<th>AM dose (mg/kg/d)</th>
<th>Pre-AM treatment</th>
<th>First visit on AM</th>
<th>Most recent visit on AM</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td>126 ± 13</td>
<td>120 ± 14*</td>
<td>118 ± 15**</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>77 ± 13</td>
<td>68 ± 10*</td>
<td>66 ± 13**</td>
</tr>
</tbody>
</table>

*P<0.01 compared to pre-treatment; **P=NS compared to first visit on AM.

Further analysis of the data according to age and cause of HTN produced similar results (results not shown).

From these data we conclude that prolonged AM treatment can provide sustained BP control in hypertensive C. Effective AM dose (corrected for patient weight) appears to remain stable over time. Further studies will be necessary to determine what effects, if any, prolonged CA treatment has on the growth and development of C with HTN.

Consultant: Pfizer, Inc.

Key Words: amlodipine, children, pharmacotherapy

P-628
LEFT VENTRICULAR MASS IN HYPERTENSIVE CHILDREN AND ADOLESCENTS AT INITIAL DIAGNOSIS
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Left ventricular hypertrophy (LVH) may be an important marker of hypertensive target organ damage in children and adolescents. To evaluate the distribution of left ventricular mass in this population at diagnosis, all children < age 18 years with newly diagnosed hypertension who underwent M-mode echocardiography within 2 months of diagnosis since November, 1998 in an outpatient pediatric hypertension clinic were included in this retrospective analysis. Patients with a history of underlying structural cardiac disease were excluded. Measurements of left ventricular (LV) internal dimension in diastole, LV posterior wall thickness, and interventricular septum thickness were used for calculation of LV mass indexed for height (LVMI). Left ventricular hypertrophy was defined as LVMI > the 90th %tile for normal children and adolescents (36.69 g/m² for boys and 34.11 g/m² for girls).

We identified 26 children (18M), age 13.6 ± 2.8 y, with hypertension who had undergone echocardiography within 2 months of diagnosis. Most, 23, had essential hypertension, 3 had renal parenchymal disease. LVH was identified in 10 subjects (38%). Subjects with LVH did not differ from subjects without LVH by age, weight, or height. Distribution of LVMI is shown below:

<table>
<thead>
<tr>
<th>LVMI %tile</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50%</td>
<td>8</td>
<td>30.8</td>
</tr>
<tr>
<td>50-89%</td>
<td>8</td>
<td>30.8</td>
</tr>
<tr>
<td>90-94%</td>
<td>3</td>
<td>11.5</td>
</tr>
<tr>
<td>&gt;95%</td>
<td>4</td>
<td>15.5</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>100.0</td>
</tr>
</tbody>
</table>

In summary, left ventricular hypertrophy may be present in a substantial percentage (38%) of children and adolescents at the time of initial diagnosis of hypertension.

Key Words: Left Ventricular Mass, Pediatric Hypertension, Target Organ Damage

P-629
TWENTY FIVE - YEAR FOLLOW-UP STUDY OF CHILDREN WITH PRIMARY HYPERTENSION: TARGET-ORGAN DAMAGE

The aim of the study is to establish cardiac and fundus oculi vascular damages occurring from childhood to adulthood in follow-up children with primary hypertension (PH).

In a prospective study (1972-1998) from 251 children (6-15 yrs) with PH from Sofia 152 (60.55%) were followed up into adulthood (31-40 yrs.). Funduscopia, 2D Echocardiography and Doppler - sonography were carried out in 90 of them randomly selected. Fisher’s exact test or $\chi^2$ and Independent Sample T-test were used for statistic analysis.

Gradual development of Angiopathia retinæ hypertonica (ARH) was established from 8.75% at onset in childhood, 32.48% at the 5th year (in adolescence), 26.78% at the 10th year (young adults), 37.77% at the 25th year (adulthood). Throughout the 25 year period the damages were predominantly of functional character - grade I-II according to Keith-Wagener Barker. At the 25th year of follow-up LVH (LVMI>150 g/m²) was established in 21.83% of the patients.

In the group of patients with ARH and LVH at the end of the study higher prevalence of hypertensive crises (p=0.0001), hypertergliceridemia ($>$2.30 mmol/l) (p=0.008), obesity (BMI>30) (p=0.014) and persisting PH into adulthood (p=0.008) were found.

This study demonstrates that a considerable part of the children, with PH go into adulthood not only with elevated BP but also with target organ damages. The data suggest the necessity of early prevention and treatment of Juvenile hypertension.

Key Words: children, follow-up, Hypertension

P-630
AMBULATORY BLOOD PRESSURE AND GENES IN WHITE COAT HYPERTENSIVE ADOLESCENTS
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45% of hypertensive children referred to our clinic are white coat hypertensives (WCH) and they are offspring of hypertensive parents. The
aim of this study was to evaluate blood pressure and heart rate at the office and by ambulatory monitoring (ABPM) and gene variants associated with hypertension in 44 adolescents with WCH (13.5 ± 2 years old, 19 m, BMI 26.7 ± 6.3 kg/m²) and compare them to 50 normotensive offspring of hypertensive parents (N) matched for age, height and sex (13.5 ± 2 y.o., 21 m, BMI 25.5 ± 5 kg/m²) and 54 essential hypertensives (EH) (12.7 ± 3.1 y.o., 5 m, BMI 23 ± 5 kg/m²). In a subgroup of 36 N, 15 WCH and 19 EH polymorphic variants M235T and T174M of the angiotensinogen gene (AGT), and I/D polymorphism of ACE gene were evaluated by PCR, RFLP or ASO.

At the office WCH had significantly higher heart rate than EH (91 ± 13 beats/min and 83 ± 11, p = 0.002). By ABPM, blood pressure and heart rate were similar in WCH and N. Homozygotes TT for the M235T variant of AGT were more prevalent in WCH than in N (Fisher p < 0.05, OR 5.25). No associations were found for the other gene variants studied.

In conclusion, 1) HR at the office could be a marker of WCH. 2) 235T polymorphism of AGT is associated to WCH, similar to the findings in hypertensives.

Key Words: white coat hypertension, adolescents, angiotensinogen gene polymorphism

P-631

INFLUENCE OF PARENT BLOOD PRESSURE AND BMI ON DEVELOPMENT OF OFFSPRING BLOOD PRESSURE AND INSULIN RESISTANCE SYNDROME (IRS)

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We have studied the relation of BP and BMI in parents to BP, obesity, and cardiovascular risk in their offspring in childhood and young adulthood. 456 children (236 male, 220 female) had BP and anthropometric measures at age 7.2 ± 0.04 yrs and the same measures plus fasting insulin and lipids at age 23.1 ± 0.04 yrs. Natural parents (268 fathers, 44.6 ± 0.4 yrs; 429 mothers, 41.5 ± 0.3 yrs), had BP and anthropometric measurements during the children’s high school years. There was a significant correlation for SBP, height, weight (WT), BMI, and triceps skinfold (r = 0.42-0.68, all p < 0.0001) between ages 7 and 23. When children were 7, fathers’ SBP was significantly related only to children’s SBP (r = 0.12, p < 0.04), and fathers’ BMI was significantly related to children’s BMI (r = 0.12, p < 0.03), and WT, BMI, and triceps (r = 0.29-0.33, all p < 0.0001). Mothers’ DBP was significantly related to children’s BP (r = 0.12, p = 0.02) and mothers’ BMI was significantly related to children’s BP, WT, BMI, and triceps (r = 0.19-0.34, all p < 0.0001). When the children were 23, fathers’ SBP was significantly related to children’s BP and WT (r = 0.13, all p < 0.04) and mothers’ BMI was significantly related to children’s BP, WT, BMI, triceps and fasting insulin (r = 0.17-0.40, all p < 0.006); mothers’ DBP was significantly related to children’s BP and BMI (r = 0.16, p < 0.04), and mothers’ BMI was significantly related to BP, WT, BMI, triceps, and fasting insulin (r = 0.22-0.42, all p < 0.0001), and triglycerides, HDL-C and LDL-C (r = 0.12-0.16, all p < 0.01). After adjusting for children’s BMI, none of the parent-child relations was significant. These results show: 1) parents’ BP and BMI are significantly related to risk factors comprising the IRS in their children; and 2) BMI appears to play the major role in these relations. We speculate that the parent-child relations operate through BMI and become stronger as the children age, thus emphasizing the importance of preventing overweight during childhood development.

Key Words: Children, Blood pressure, insulin resistance syndrome

P-632

THE SUCCESSFUL IMPLEMENTATION OF A PROTOCOL TO NORMALIZE SODIUM INTAKE IN AFRICAN-AMERICAN YOUTHS

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We and others have hypothesized that stress induced changes in sodium regulation contributes to the development of salt sensitive hypertension. It is important to normalize the sodium intake in studies that examine this hypothesis to insure that the subjects are at similar levels of sodium balance when they are tested. This is a particular problem in adolescents because of their lifestyles. We have developed a 3-day dietary protocol to control the sodium intake in a study of stress induced changes in sodium handling in African-American youths aged 15 to 18 years. The purpose of this report is to document our success with this dietary plan and describe the factors related to differences in this success. The dietary plan is based on a limited choice diet containing the normal sodium intake of teenagers in our region of the country (4000 ± 200 mg/day) and utilizes pre-packaged foods. A research assistant meets with each subject to select the diet for each day. The selected foods are packed in coolers which the subjects pick up each day. The coolers are then returned the next day with all the wrappers and an overnight urine collection to determine dietary compliance. Of the 64 subjects tested thus far, 60 (93.7%) have complied with the diet as determined by urinary sodium values from the overnight collections. The variability in sodium intake across subjects was significantly reduced from the first day to the third day of the diet (153 ± 67 to 121 ± 53 mmol/L (p < 0.03). This resulted in a standard deviation in UNaV of only 6.7 mEq/hr at the beginning of the test period. There were sex differences in the amount of sodium that was consumed based on returned foods and wrappers. Males (n = 29) consumed an average of 88.7% of the total sodium they were provided compared to 77.7% for the females (n = 31). Further analysis showed that within the females these differences were significantly correlated with body weight (r = 0.48; p < .007). There was not a significant relationship in the males. However, these sex differences did not result in sex differences in baseline UNaV during the testing procedure (21 ± 12 vs 19 ± 11 mEq/hr). In conclusion, we have developed a dietary protocol that we believe is effective in normalizing the dietary intake of sodium in adolescents. This protocol can be used in any study in which it is important to bring adolescents into comparable levels of sodium balance.

Key Words: Sodium, Diet, Adolescents