OR-68
GROWTH OF LEFT VENTRICULAR MASS AND ITS MODERATORS FROM CHILDHOOD TO EARLY ADULTHOOD IN AFRICAN AMERICAN AND EUROPEAN AMERICAN YOUTH
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Objective: Increased left ventricular mass has been established as a strong risk factor for cardiovascular morbidity and mortality. The aim of this study was to evaluate growth of left ventricular mass (LVM) from childhood into early adulthood and its possible sociodemographic, anthropometric, and hemodynamic moderators.

OR-67
LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSIVE CHILDREN: A REPORT FROM THE INTERNATIONAL PEDIATRIC HYPERTENSION ASSOCIATION
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Objectives: To determine the prevalence of left ventricular hypertrophy (LVH) by both pediatric and adult criteria in children and adolescents with hypertension.

Methods: Pooled data from 1998 to 2001 from 3 sites belonging to the International Pediatric Hypertension Association were reviewed. Both treated (i.e., antihypertensive meds) and untreated pts undergoing echocardiography to detect LVH as part of the evaluation for hypertension were included for analysis. Left ventricular mass was calculated from 2-D guided m-mode echocardiographic measurements of the LV using the equation reported by Devereux. Measurements of the LV internal dimension, interventricular septal thickness, and posterior wall thickness were made during diastole according to methods established by the American Society of Echocardiography. LVMI was calculated as LVM/height^2.7. LVH by adult criteria was defined as LVMI > 51 gm/m^2 and by pediatric criteria as LVMI > 38.6 gm/m^2 (based on normative pediatric LVMI data).

Results: 133 pts were included for analysis. Clinical characteristics were: age 13.6 ± 3.6 yrs, male 67%, height 160 ± 18 cm, weight 72 ± 27 kg, and BMI 27.5 ± 7.4 kg/m^2. Mean LVMI was 37.5 ± 13.1 gm/m^2. Compared to females, males had greater LVMI (39.2 vs. 34.0 gm/m^2, p < 0.05) and were taller (164 vs. 152 cm, p < 0.001). Males and females did not differ in age, weight or BMI. LVMI correlated significantly with BMI (r = 0.41, p < 0.0001) and weight (r = 0.29, p < 0.001), but did not correlate with age. The overall prevalence of LVH by adult criteria was 15.8% and by pediatric criteria was 38.3%. Using either criteria, pts with LVH were heavier (p < 0.01) and had greater BMI (p < 0.001) than those without LVH. Males were more likely than females to have LVH by pediatric criteria (45% vs. 25%, p < 0.01), but not by adult criteria. Among untreated pts (n = 77), the prevalence of LVH was 14.3% by adult criteria and 39.0% by pediatric criteria, which did not differ significantly from the overall LVH prevalences.

Conclusions: LVH, even by adult criteria, occurs commonly in children and adolescents with hypertension and is positively associated with increased BMI. Echocardiography may therefore be helpful in the determination of the need for antihypertensive therapy. Further studies are needed to determine whether treatment of the hypertension results in regression of LVMI to the normal range.

Key Words: Left Ventricular Hypertrophy, Child, Hypertension
no significant difference in response to amloidipine according to cause of HTN. Linear regression analysis revealed a significant relationship between the dose of amloidipine in mg/kg and changes in both SBP (P=0.03) and DBP (P=0.02), with predicted mean reductions of 9.2/4.9 mmHg for an amloidipine dose of >0.06 mg/kg. Six children (2.2%) were discontinued from amloidipine treatment for drug-related adverse events, and the dose was reduced in one additional child. The most commonly reported adverse effect of amloidipine was headache. We conclude that amloidipine is an effective antihypertensive agent in children. The results of this study should provide valuable guidance to physicians who prescribe amloidipine for children with HTN.

Key Words: Amlodipine, Children, Clinical Trials

OR-70
EFFECTS OF THE ACE INHIBITOR, LISINOPRIL (L), IN CHILDREN AGE 6-16 YEARS WITH HYPERTENSION
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This multicenter study assessed the antihypertensive safety and efficacy of L (PRINIVIL®) in children with hypertension age 6 to 16 yrs. The study was comprised of 115 pts; 41 from the U.S., 74 ex-U.S., 54 (47%) of L. PRINIVIL/H23041 was tolerated. Evaluation of the dose response suggests that a starting dose of 0.07 mg/kg (up to 5 mg) once daily is appropriate, whereas a dose of 0.625/1.25 mg (0.02 mg/kg) was not effective in this population.

Key Words: Prinivil®/lisinopril, Pediatric Hypertension, Clinical Trials

OR-71
PERINATAL PROGRAMMING AND THE RENIN-ANGIOTENSIN SYSTEM
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Adult human cardiovascular disease is inversely related to birthweight, though the pathogenesis is not fully known. We and others have shown in rats that maternal dietary protein restriction results in renal dysfunction and hypertension in offspring in adult life. We previously found perinatal suppression of renin mRNA and protein, and Ang II, in male pups of protein-restricted mothers; nephron number in adult male littersmates was decreased. In contrast, female offspring were less markedly affected, requiring a greater maternal insult to develop such abnormalities. In the present studies Sprague-Dawley rats were fed either a normal protein (19% protein = 21% casein, NP), normal sodium (0.20%) diet or a modestly protein-restricted (8.5%, LP). Normal sodium diet ad lib throughout pregnancy. At delivery, all dams were placed on normal diet, and pups weaned to normal diet at 22 d. Some newborn animals were used for renal tissue measurements; littersmates were allowed to grow to adulthood for physiological studies. Real-Time PCR was used to compare male and female newborn pups at day 1 for intrarenal RAS components -angiotensinogen [ang-n], renin, ACE, AT1, and AT2 mRNA [*, p<0.05]:

<table>
<thead>
<tr>
<th></th>
<th>M LP/NP ratio</th>
<th>F LP/NP ratio</th>
<th>M LP/NP sig</th>
<th>F LP/NP sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE mRNA</td>
<td>0.38</td>
<td>0.62</td>
<td>* NS</td>
<td>NS</td>
</tr>
<tr>
<td>AT1 mRNA</td>
<td>1.24</td>
<td>0.98</td>
<td>* NS</td>
<td>NS</td>
</tr>
<tr>
<td>AT2 mRNA</td>
<td>0.39</td>
<td>1.24</td>
<td>* NS</td>
<td>NS</td>
</tr>
<tr>
<td>Ang-n</td>
<td>1.55</td>
<td>1.41</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Renin</td>
<td>0.21</td>
<td>1.24</td>
<td>* NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Angiotensin II levels, as well as tissue renin activity levels are more suppressed in male offspring than in female. Mean arterial pressure [MAP] was the same in adult female offspring of LP [121+/−2mmHg] and NP mothers [120+/−3 mmHg], as was GFR, ERPF, and filtration fraction. In contrast adult male offspring of LP mothers had sig. higher MAP than NP offspring [136+/−2 mmHg vs. 125+/−2 mmHg, p =0.004]; they also showed differences in GFR, ERPF and FF. Thus, maternal protein restriction clearly and markedly decrease the expression of the intrarenal RAS in newborn male offspring, leading to permanent changes that influence the subsequent course of cardio renal health, whereas female rats are relatively resistant to programming for adult hypertension by perinatal protein restriction. This resistance may be due to the fact that modest maternal protein restriction does not reduce the number of glomeruli with which females are endowed as it does in males. The intrarenal RAS during development may play a key role in this protective effect of female gender.

Key Words: Perinatal Programming, Renin-Angiotensin System, Nephrogenesis