High Blood Pressure Trends in Children and Adolescents in National Surveys, 1963 to 2002

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Background—Secular trend data on hypertension in children and adolescents are scarce and inconsistent. In the face of growing obesity, we sought to assess high blood pressure (HBP) secular trends in children and adolescents enrolled in national surveys and to determine whether the HBP trend reversed its course with the rise in obesity.

Methods and Results—National survey data obtained from multistage probability sampling of the US noninstitutionalized population from 1963 to 2002 were examined; 8- to 17-year-old non-Hispanic blacks and whites and Mexican Americans were included. HBP ascertainment was based on age-, gender-, and height percentile–specific systolic and diastolic BPs. Weighted analyses were performed to account for the complex design. The BP, pre-HBP, and HBP trends were downward from 1963 to 1988 and upward thereafter. Pre-HBP and HBP increased 2.3% (P=0.0003) and 1% (P=0.17), respectively, between 1988 and 1999. Obesity increase, more so abdominal than general obesity, partially explained the rise in HBP and pre-HBP from 1988 to 1999. BP and HBP reversed their downward trends 10 years after the increase in the prevalence of obesity. Additionally, an ethnic and gender gap appeared in 1988 for pre-HBP and in 1999 for HBP; non-Hispanic blacks and Mexican Americans had a greater prevalence of HBP and pre-HBP than non-Hispanic whites, and males had a greater prevalence than females.

Conclusions—HBP and pre-HBP in children and adolescents are on the rise. These new findings have implications for the cardiovascular disease public health burden, particularly the risk of a new cardiovascular disease transition. They reinforce the urgent call for early prevention of obesity and HBP and illustrate racial/ethnic disparities in this age group. (Circulation. 2007;116:1488-1496.)

Key Words: epidemiology □ hypertension □ obesity □ pediatrics □ trends

Hypertension in adults remains a major public health problem. Several studies support the theory that the roots of essential hypertension may extend back to childhood.1 With obesity reaching epidemic proportions2 and obesity in children and adolescents being one of the strongest predictors of young adulthood hypertension,3 along with childhood blood pressure (BP) level and family history of hypertension, it is important to determine whether the prevalence of hypertension is increasing or decreasing in children and adolescents. This question is even more pertinent when we consider that the incidence of type 2 diabetes mellitus in youth is increasing, a direct result of the increase in childhood obesity.4 A recent study in a national sample has documented the BP increase in children and adolescents since the late 1980s.5 However, data on secular trends of BP in children and adolescents are scarce and inconsistent.6,7 Moreover, they are based on samples that are not nationwide. The limited national data on hypertension prevalence in children and adolescents enrolled in the earliest National Health and Nutrition Examination Survey (NHANES) showed a decreasing trend, although the investigators used the adult definition of hypertension.8,9 Trends in adult hypertension in NHANES exhibited an upward slope from 1988 to 1994 after a long period of downward slope.10,11

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We used the US National Health Surveys data (1963 to 2002) (1) to describe the trends of age-adjusted prevalence of high BP (HBP) and mean BP for boys and girls 8 to 17 years of age, (2) to describe these trends by racial/ethnic-gender and age groups, and (3) to examine the impact of increasing obesity on these trends, hypothesizing that the HBP trend would reverse its course in relation to the obesity trend.

Methods

Study Population

The study population consisted of boys and girls aged 8 to 17 years who participated in the National Health Examination (NHES), the Hispanic Health and Nutrition Examination Survey (HHANES), and the NHANES. These surveys were conducted by the National Center for Health Statistics (NCHS) on a nationwide probability sample of the civilian noninstitutionalized US population and based on a...
TABLE 1. Measures of Observer Bias: Weighted Prevalence of Matching of Arm Circumference With Cuff Size

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<thead>
<tr>
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<tbody>
<tr>
<td>Cuff Size</td>
<td>1963–1964 n (%)</td>
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</tr>
<tr>
<td>Adult</td>
<td>N/A†</td>
</tr>
<tr>
<td>Large Adult</td>
<td>N/A†</td>
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<tr>
<td>Adult thigh</td>
<td>N/A†</td>
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</tbody>
</table>

AC/CW indicates ratio of arm circumference to cuff width.

*Cuff size used was not recorded for NHES-NHANES.

AC/CW indicates ratio of arm circumference to cuff width.

For these reasons, efforts were undertaken in the present analyses to minimize the effect of measurement variations. Only sitting BP measurements taken in the mobile examination center (n=3) were used. For the BP trend analyses, we tested the first BP measurement (performed by physicians) and the average of all available BP measurements. (In NHANES III, 94% had 3 BP measurements; in NHANES 1999 to 2002, 65% had 3 BP measurements.) The trends were consistent. Therefore, for accuracy, we used the latter definition in all analyses.

We used the method outlined in the Task Force Report Appendix B to classify subjects’ BPs into five categories.12–17 After each participant’s height was converted to a height percentile26 after each participant’s height was converted to a height percentile, the appropriate cuff size was used. For the BP trend analyses, we tested the first BP measurement (performed by physicians) and the average of all available BP measurements. (In NHANES III, 94% had 3 BP measurements; in NHANES 1999 to 2002, 65% had 3 BP measurements.) The trends were consistent. Therefore, for accuracy, we used the latter definition in all analyses.
Exposure and Covariates

Time, the exposure variable for the trend study, was categorized into 6 levels corresponding to the survey’s period of examination (NHES II and III were collapsed).

The sampling plan for NHANES is based on age at interview. Age was defined as a continuous variable for assessment of confounding and categorized into 8 to 11, 12 to 14, and 15 to 17 years for stratified analyses to proxy the effect of puberty. This categorization is clinical and not data based; it was used because the Tanner index of sexual maturation was not measured at all periods. The race/ethnicity categories included non-Hispanic blacks (blacks), non-Hispanic whites (whites), and Mexican Americans.

Obesity was assessed by body mass index (BMI; in kg/m²) for age, an index of overall obesity, and by waist circumference (in cm; not measured in NHANES I and II), an index of visceral adiposity. Body weight was measured to the nearest 0.05 kg and height to the nearest 0.1 cm.

Statistical Analyses

Analyses were performed with SAS and SUDAAN to obtain correct variance estimates. NCHS-computed sampling weights were used in the analyses to account for differential probability of selection resulting from the cluster design, planned oversampling of selected subgroups (children, blacks, and Mexican Americans), unit nonresponse, and noncoverage. Estimates derived from a sample size smaller than the recommended sample size for the design effect or the estimated proportion were considered unreliable and reported with a footnote.

For the trends analyses, prevalence of HBP and pre-HBP and distribution of mean BP were standardized to the 2000 census 5-year age distribution by the direct standardization method. These analyses were conducted on the total population and then stratified by age group and gender-race/ethnicity.

Two methods were used to assess the impact of obesity rise on HBP/pre-HBP increase between NHANES III and IV: multivariate logistic regression to establish the likelihood of HBP by obesity status and direct standardization to quantify the magnitude of HBP/pre-HBP prevalence increase accounted for by the increase in obesity during this same period. In the first procedure, NHANES 1988 to 1994 and 1999 to 2002 datasets were pooled, and the original NCHS-computed weight for each survey was used. For HBP, there was a marginally significant 3-way interaction between obesity and race/ethnicity across time (P = 0.06), resulting mainly from a significant interaction of BMI with race/ethnicity in 1988 to 1994 (P < 0.001) and 1999 to 2002 (P = 0.05). For pre-HBP, there was a marginally significant 3-way interaction between obesity and race/ethnicity across time (P = 0.06), resulting mainly from a significant interaction of BMI with race/ethnicity in 1988 to 1994 (P = 0.03) but not in 1999 to 2002 (P = 0.6). In contrast, the effect of waist circumference was constant across time and racial/ethnic groups. Therefore, subsequent analyses were performed separately for each period to account for the interaction by time. To account for the race/ethnicity-by-BMI interaction, race/ethnicity-specific models were fit that included age, gender, and BMI-for-age z scores. Waist circumference was strongly collinear with BMI; hence, it was analyzed in a separate model. Second, the impact of obesity on the HBP increase was further quantified by direct standardization using the NHANES 1988 to 1994 distribution of BMI or waist circumference to compute the NHANES 1999 to 2002 estimates. The resulting change in HBP and pre-HBP prevalence was evaluated.

The statistical method to obtain variance estimates was Taylor linearization. We used the Student t test for 2 independent samples (2-sided) to test whether the difference in HBP/pre-HBP prevalence between NHANES III and NHANES 1999 to 2002 and the impact of BMI rise on this difference were statistically significant. The nominal cutoff points for statistical significance were 0.05 for main effect and 0.10 for interactive effects. The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Study Population Characteristics

Table 2 displays the study population characteristics by race/ethnicity. The age and gender distribution remained almost stable over time. As expected, general and abdominal obesity increased over time in all racial/ethnic groups.

Trends of HBP and BP

High Blood Pressure

Table 3 displays the distribution of HBP. Males tended to have slightly greater HBP prevalence than females. Age-adjusted HBP prevalence tended to decrease between 1963 and 1988 to 1994 and to increase thereafter. In 1999 to 2002, the age-adjusted prevalences of HBP were 4.2% (SE, 0.7%), 3.3% (SE, 0.6%), and 4.6% (SE, 0.6%) for blacks, whites, and Mexican Americans, respectively. The distribution of HBP exhibited many unreliable estimates (Table 3). A gender and ethnic disparity appeared in 1999, whereas it appeared in 1988 for pre-HBP (data not shown).

Table 4 details the increase in prevalence that occurred between 1988 and 2002. Pre-HBP and HBP increased by 2.3 (P = 0.0003) and 1 (P = 0.17) percentage points, respectively. The pre-HBP increase was significant for blacks and Mexican Americans, whereas the HBP increase was significant for Mexican American women and white females. However, we must place a note of caution for the HBP data because in many cases the sample size is too small to yield reliable estimates, even though there were significant differences.

Blood Pressure

Figure 1 displays the BP trends for lean, at risk for overweight, and overweight, along with the prevalence trends for the latter 2 groups. As expected, the trend of SBP and DBP mirrored that of HBP. The mean increase in age-adjusted BP between 1988 to 1994 and 1999 to 2002 was greater for DBP (8.4 mm Hg; SE, 1.1 mm Hg; P < 0.001) than for SBP (1.3 mm Hg; SE, 0.5 mm Hg; P = 0.01). The age-adjusted mean increase in SBP between 1988 to 1994 and 1999 to 2002 was comparable for lean (1.0; SE, 0.4), at-risk-for-overweight (0.9; SE, 1.1), and overweight (0.6; SE, 1.0) children and adolescents, whereas the DBP increase during that same period was greater and significant for lean (9.2; SE, 1.2) than for at-risk-for-overweight (7.4; SE, 1.8) and overweight (4.7; SE, 2.3) children.

Upward Trends of HBP and Obesity

Obesity increased in a monotonic fashion beginning with the earliest survey (1963 to 1970) for blacks and whites (Table 2). However, the trends differed in their patterns: stepwise for at risk for overweight and curvilinear for overweight (Figure 1). The increase in BP/HBP/pre-HBP lagged 10 years behind the increase in obesity (Figure 1 and Table 3; data not shown for pre-HBP).

As expected, both BMI and waist circumference significantly increased the likelihood of HBP. In 1999 to 2002, the odds ratios (ORs) for HBP were 2.1 (95% confidence interval [CI], 1.5 to 3.0), 1.8 (95% CI, 1.2 to 2.6), and 3.2 (95% CI, 2.4 to 4.4) for blacks, whites, and Mexican Americans, respectively, for 1 BMI z-score unit increment. For pre-HBP,
they were 1.6 (95% CI, 1.4 to 1.7), 1.7 (95% CI, 1.4 to 2.1), and 1.7 (95% CI, 1.4 to 2.0). Relative to a child at the median BMI for age and sex, a child 1z score higher would have a BMI that was between 2.1 and 3.5 kg/m² higher, depending on age and sex. Conversely, the OR for waist circumference was constant for both outcomes. The corresponding ORs were 1.28 (95% CI, 1.26 to 1.31), 1.28 (95% CI, 1.24 to 1.33), and 1.35 (95% CI, 1.32 to 1.38) for HBP and 1.22 (95% CI, 1.21 to 1.23), 1.28 (95% CI, 1.26 to 1.31), and 1.28 (95% CI, 1.24 to 1.33) for pre-HBP among the 3 respective ethnic groups for a 5-cm increment in waist circumference. Obesity rise had the greatest impact in blacks and Mexican Americans for HBP and in Mexican Americans only for pre-HBP (Figures 2 and 3).

**Discussion**

**Summary of Results**

The present study explored the trends of HBP in children and adolescents in a nationally representative sample of 8- to 17-year-old subjects over the past 40 years and the impact of obesity increase on these trends in the past 10 years. We found that the prevalence of elevated BP has been on the rise among US children and adolescents since the late 1980s, after a long period of decreasing trend. The BP rise lags behind the increase in obesity. The increase in obesity, more so abdominal obesity than general obesity, accounts for part of the upward trend of HBP. The ethnic divergence in BP distribution paralleled the ethnic changes in obesity. Finally, the racial/ethnic disparity in HBP is a recent phenomenon in this age group. To the best of our knowledge, this is the first report of such findings.

**Comparison With Other Studies**

The few studies of children that have examined secular BP trends as a function of secular obesity yielded inconsistent results.6,7 In the Minneapolis Children’s BP Study of 10- to 14-year-old children,7 SBP percentiles were significantly higher and DBP percentiles were significant lower in 1996 than in 1986, whereas in the Bogalusa study,6 both SBP and DBP decreased at the end of the study periods (1975 to 1981 and 1984 to 1992) but obesity increased in both cohorts. The effect of obesity was evaluated in the former study and explained the SBP secular increase only. That DBP increased more rapidly than SBP in our study may be a reflection of early subclinical vascular disease because impairment of arterial compliance and endothelial function have been linked to weight (ie, low birth weight37 and excess current weight38) in youth.

Despite the BP measurement variation, the downward BP trends seem real and universal; they have been described in a variety of settings. In the 1960s, such a downward trend was reported in children and adolescents by the NCHS investigators using the adult definition of hypertension, that is, BP ≥140/90 mm Hg.8,9 The prevalence of hypertension decreased from 0.6% to 0.4% among the 6- to 11-year-old children and from 6.4% to 3.6% among the 12- to 17-year-old adolescents between 1971 to 1975 and 1976 to 1980. The same downward pattern has been described in adults enrolled in NHANES10 and among young adults in the United King-
dom and Australia. Goff et al analyzed the BP decline in adults 18 to 74 years of age enrolled in NHES/NHANES 1960 to 1994 by 10-year birth cohorts from 1887 to 1975. They also found that more recent cohorts had lower BPs than older cohorts.

The upward pattern also mirrors the adult hypertension trends in NHANES and the BP trends in children and adolescents. In these studies, obesity rise (as assessed by BMI) accounted for a variable proportion of the BP/hypertension increase: 29% of SBP and 12% of DBP increase in youth and 44% of hypertension increase in adults between 1988 to 1994 and 1999 to 2000. In the present study, the increase in HBP was attenuated by approximately 44% when standardized by BMI or waist circumference distribution in NHANES III, and the increase in pre-HBP also was attenuated by 27% and 68%, respectively, suggesting that the recent increase in the prevalence of pediatric HBP is explained, at least in part, by the increase in BMI and waist circumference. That the 1984 to 1999 DBP increase is greatest among lean individuals reinforces the fact that obesity is not the sole factor (this observation is not an artifact because the median for lean remained constant over time). The greater impact of abdominal obesity on elevated BP rise is consistent with findings in adult hypertension.

Another intriguing finding is that the BP/HBP increase lagged 10 years behind the obesity increase. The question then is why the downward BP trends persist in the face of increasing obesity trends. Although this question is beyond the scope of our study, we might speculate on a few reasons. In Figure 1, we notice that in 1963 to 1980, the prevalence of overweight was stable, whereas that of overweight remained constant over time. The greater impact of abdominal obesity on elevated BP rise is consistent with findings in adult hypertension.

### TABLE 3. Age-Adjusted and Age-Specific Prevalence (%) of HBP by Gender and Race/Ethnicity Over Time

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<td>17.8 (2.2)</td>
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<td>3.7 (0.5)</td>
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<td>…</td>
<td>2.5 (0.6)</td>
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<tr>
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<td>4.8 (1.2)</td>
<td>2.5 (0.7)</td>
<td>4.6 (0.6)</td>
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<td><strong>Males, y</strong></td>
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<td>9.3 (2.3)</td>
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<td>4.1 (0.8)</td>
<td>5.5 (0.9)</td>
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<td>8–11</td>
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<td>8.2 (2.3)</td>
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<td>4.8 (1.5)</td>
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*HBP (≥95th percentile) frequency is <7.
†HBP (≥95th percentile) frequency is 0.
whereas the prevalence of at risk for overweight remained stable, although at a higher level. During 1988 to 2002, we observed the reversed, upward HBP trend. It is therefore conceivable that it may take obesity that amount of time to induce cardiovascular modifications that will lead to HBP years later or that there is a threshold effect at the population level. Only cohort studies can adequately address such a question.

**TABLE 4. Increase in Age-Adjusted Prevalence (SE) of Pre-HBP and HBP During 1988 to 2002 by Gender and Race/Ethnicity**

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<td>Total, n</td>
<td>Cases, n</td>
<td>% (SE)</td>
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<td>3.7 (0.8)</td>
</tr>
<tr>
<td>HBP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>118</td>
<td>2.7 (0.5)</td>
<td>3.7 (0.4)</td>
</tr>
<tr>
<td>Non-Hispanic blacks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>29</td>
<td>4.1 (0.8)</td>
<td>5.5 (0.9)</td>
</tr>
<tr>
<td>Females</td>
<td>24</td>
<td>3.2 (0.7)</td>
<td>3.0 (0.7)</td>
</tr>
<tr>
<td>Non-Hispanic whites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>17</td>
<td>3.6 (1.0)</td>
<td>3.5 (0.8)</td>
</tr>
<tr>
<td>Females</td>
<td>7</td>
<td>1.3 (0.5)</td>
<td>3.2 (0.9)</td>
</tr>
<tr>
<td>Mexican Americans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>22</td>
<td>3.1 (1.1)</td>
<td>5.3 (0.6)</td>
</tr>
<tr>
<td>Females</td>
<td>19</td>
<td>1.9 (0.6)</td>
<td>3.9 (0.8)</td>
</tr>
</tbody>
</table>

*P value, pooled t test for H0: \( P_{(1999-02)} = P_{(1988-94)} \); df = 93 (49 for NHANES III + 44 for NHANES 1999 to 2002).
A latent factor that may explain the downward BP trend is lifestyle changes. We cannot rule out the effect of increased awareness of cardiovascular disease risk factors and consequential lifestyle changes, as suggested by the study by Goff et al40 in which the BP downward shift in adults had bearing on the whole BP distribution. Although the interventions were directed mostly at adults, the whole family may have reaped the benefits of healthy behavioral changes. Sodium intake and physical inactivity, which are strong correlates of HBP, were not consistently measured in NHANES. Hence, their potential beneficial effect cannot be evaluated. Current data suggest an increased sedentary lifestyle in children and adolescents, as defined by television viewing time,42,43 and increased sodium intake.44 Thus, these 2 habits would not explain the downward trend either. In their review of secular trends in BP in early life, McCarron et al39 allude to other factors that may reduce adulthood hypertension prevalence, such as birth weight, childhood growth, and early diet, including salt restriction in infancy, breastfeeding, and increased intake of fruits and vegetables. Further work is required to understand the mechanisms by which SBP and HBP continued to decrease while obesity increased. These protective factors may be promoted in this phase of a reversed, upward BP/HBP trend.

Methodological Issues
The most important methodological issue is the variation of BP measurement across study periods. If the multiplicity of BP measures within a visit attenuates the effect of the child’s anxiety toward this procedure, the lack of repeat BP measurements over time overestimates the individual’s values by not accounting for the effect of regression toward the mean.45,46 However, the impact on trends should be minimal, if any, because this emotional component is constant over time.

The improvement in quality-control monitoring measures in national surveys over time24,47,48 is demonstrated by the reduction of prevalence of 0-end-digit preference. Australian investigators found that digit-preference bias tended to overestimate hypertension prevalence.49 Digit-preference bias

Figure 2. Impact of obesity rise as assessed by BMI and waist circumference (WC) on the rise of HBP in 1988 to 1999 by race/ethnicity and gender. *Standardized (std) by the distribution of BMI or WC in NHANES III.

Figure 3. Impact of obesity rise as assessed by BMI or waist circumference (WC) on the rise of pre-HBP in 1988 to 1999 by race/ethnicity and gender. *Standardized (std) by the distribution of BMI or WC in NHANES III.
would not play a great role in our study because the NHES survey had among the lowest prevalence of digit preference (24%) yet the greatest HBP prevalence.

Another important potential source of error is the use of inappropriate cuff size in relation to arm circumference. It has been demonstrated that undercuffing overestimates and over-cuffing underestimates BP. As a result of the data in Table 1, the 1988 to 1999 HBP prevalence gradient may be smaller than observed.

Finally, we cannot exclude the latent confounding effect of inconsistently measured variables such as physical activity or sexual maturation, which play an important role in hemodynamics and physiological maturation. These limitations do not minimize the strengths of this study. This study uses national representative data that included multiethnic groups to document for the first time the HBP and pre-HBP trends and racial/ethnic disparities in children and adolescents. It also documents the new fact that the cardiovascular risk for the young Mexican-American male is about to surpass that for young non-Hispanic blacks.

Conclusions

HBP and pre-HBP are on the rise in children and adolescents after a long period of decline. This observation has great public health importance because elevated BP is a major risk factor for cardiovascular disease. This rise can be explained, at least in part, by obesity, particularly abdominal obesity. These results point to the need to adopt multiple strategies aimed at preventing the clustering of risk factors, called metabolic syndrome, in children and adolescents, particularly those belonging to racial/ethnic minority groups. Prevention is particularly important in view of the possibility that the current generation of children may be the first to have shorter life expectancies than their parents.50

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Disclosures

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**CLINICAL PERSPECTIVE**

Using the 1963 to 2002 national survey data, we found that, after a long period of decline, the prevalence of elevated blood pressure has reversed its trends since 1988. However, the increase over the last 2 survey periods (1988 to 1994 and 1999 to 2002) is greater and significant for pre–high blood pressure (2.3%; P<0.0003) than for high blood pressure (1%; P=0.17). Because this is the beginning of the upward trend, strong action taken now may prevent the progress of such a trend. Therefore, it is advisable to measure blood pressure at every visit with the appropriate technique to rank the child’s measured blood pressure from the Centers for Disease Control and Prevention growth charts and the gender-, age-, and height-specific blood pressure table (published by the National Task Force) and to follow the recommendations proposed by this body for elevated blood pressure measurement. The second observation was that obesity, more so abdominal than general obesity, partially accounts for the elevated blood pressure trend. For example, had the distribution of waist circumference remained at the 1988 to 1994 level, the prevalence of pre–high blood pressure in 1999 to 2002 would have been lower by almost two-thirds. We draw 3 conclusions: (1) Measurement of waist circumference should become a routine clinical act, along with measurement of height and weight; (2) components of the metabolic syndrome should be monitored; and (3) pre–high blood pressure should be managed as vigorously as high blood pressure, especially because the Bogalusa studies have shown that the former leads to early target-organ damage in young adulthood. Special attention must be paid to minorities, particularly young Mexican American boys. Finally, the Centers for Disease Control and Prevention should consider building growth charts for waist circumference similar to the other anthropometric variables charts.
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Rebecca Din-Dzietham, Yong Liu, Marie-Vero Bielo and Falah Shamsa

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