Racial Differences Among Children With Primary Hypertension

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KEY WORDS
primary hypertension, race, ethnicity, cardiovascular, obesity, blood pressure, children, adolescents

ABBREVIATIONS
BP—blood pressure
CBP—casual blood pressure
ABPM—ambulatory blood pressure monitoring
AA—African American
LV—left ventricular
LVM—left ventricular mass
LVMI—left ventricular mass index
SBP—systolic blood pressure

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WHAT'S KNOWN ON THIS SUBJECT: Race is a known risk factor for hypertension and overall cardiovascular risk in adults. In children, the normal blood pressure increases seen with age and height are also influenced by race and ethnicity.

WHAT THIS STUDY ADDS: This study of children with primary hypertension reveals differences in cardiovascular risk factors among children of different racial groups not previously been described in the pediatric population.

abstract

OBJECTIVE: Race is a known risk factor for hypertension and cardiovascular disease in adults and influences blood pressure (BP) in children. We sought to determine if there are differences in clinical, laboratory, or echocardiographic characteristics among children with primary hypertension from different racial groups.

PATIENTS AND METHODS: Study participants were 184 children aged 3 to 20 years with a diagnosis of primary hypertension who were examined at 1 of 3 participating centers at the time of initial evaluation of elevated BP. Black children were categorized as African American (AA) and nonblack children as non-AA. Comparisons were made for the entire group and after stratification according to age (<13 or ≥ 13 years).

RESULTS: Overall, children categorized as AA had a higher prevalence of overweight/obesity and left ventricular hypertrophy and had higher plasma renin activity than children who were categorized as non-AA. After age stratification, these differences remained only in the children younger than 13 years old; there were no differences in these findings among children aged 13 years or older. AA children who were aged 13 years or older, however, had higher BPs for both casual and ambulatory measurements. Specifically, they had higher casual diastolic BP, higher 24-hour diastolic BP, higher daytime systolic and diastolic BP, and higher BP loads at night and over a 24-hour period compared with non-AA children who were aged 13 years or older.

CONCLUSIONS: These data indicate that black children with primary hypertension may be at increased cardiovascular risk compared with nonblack children with primary hypertension. However, the high prevalence of overweight/obesity and left ventricular hypertrophy in all youth with primary hypertension demonstrates the need for greater preventive and therapeutic efforts aimed at reducing cardiovascular risk in this vulnerable population. *Pediatrics* 2010;126:931–937

As the prevalence of hypertension in adults and children has increased, more efforts have been aimed at elucidating the risk factors for elevated blood pressure (BP), hypertension, and cardiovascular morbidity and mortality so that this information can be used to tailor preventive efforts. Of the many risk factors that have been examined, race has consistently been shown to influence BP, hypertension, and overall cardiovascular risk in adults. It is well known that black adults are not only at increased risk for hypertension and more likely to be hypertensive compared with their white counterparts,[1,2] they also experience more severe hypertensive disease and suffer more cardiovascular events than do white adults.[3–6]

Despite these differences in adults, the prevalence of hypertension in children has not been shown to differ according to racial or ethnic groups.[7,8] Normal BP, however, increases with age, height, and BMI in children, and the rate of this increase is influenced by race and ethnicity. Of all of the determinants of BP in children, adiposity as reflected by increased BMI has been shown to be most consistently linked to elevated BP, and this effect is most pronounced in white children.[9–11] Specifically, black children have a higher prevalence of elevated BP (≥95th percentile) at lower levels of BMI, but among children with the highest levels of BMI, more white children have elevated BP than black children.[10] In addition, age, perhaps as a proxy for pubertal status, has been shown to have a differential impact on the normal BP trends among children and adolescents and among children of different racial groups.[10]

Given these differences, we sought to find out what additional clinical, laboratory, and echocardiographic differences, if any, existed between children with primary hypertension from different racial groups.

METHODS

Study Design

We conducted a cross-sectional study of all children referred to the pediatric nephrology and hypertension clinics for initial evaluation of elevated BP at the University of Michigan from 1995 to 2000, Johns Hopkins University from 1997 to 2005, and the Children's Hospital at Montefiore from 2001 to 2004. Data from patients' initial visit and workup were included in the study for patients who were 3 to 20 years old at presentation and whose elevated BP was ultimately diagnosed as primary hypertension after a standardized evaluation, as detailed below. Data were excluded for children whose elevated BP was diagnosed as secondary hypertension or who had a previous diagnosis of hypertension or were younger than 3 years. The institutional review boards at all 3 institutions approved the review of patient charts for data collection.

Patient Evaluation

Patients underwent a standardized evaluation to exclude secondary causes of hypertension. Details of this evaluation, which was performed according to recommendations by consensus organizations,[12] have previously been published.[13,14] The overlapping working relationships between the coauthors at the 3 institutions (Michigan, Drs Flynn and Parekh; Montefiore, Drs Flynn and Brady; Johns Hopkins, Drs Parekh and Brady) ensured that the same evaluation protocol was applied to all patients included in this report.

BP Measurement

Measurements of casual BP (CBP) were obtained by use of an automated oscillometric device (Dinamap [Critikon Inc, Tampa, FL]) from the right arm while the patient was seated. In most cases, elevated BP measurements were confirmed by a physician who used an aneroid sphygmomanometer, and multiple CBP measurements were available at the time of initial evaluation.

Twenty-four–hour ambulatory BP monitoring (ABPM) was performed according to standard institutional policy and procedure as previously described[15] by using Spacelabs oscillometric ambulatory monitors (model 90217 [Spacelabs Medical, Issaquah, WA]). Daytime and nighttime periods were determined according to standard cutoffs (daytime between 8 AM and 8 PM, nighttime from 10 PM to 6 AM, with transitional times [6–8 AM, 8–10 PM] included in the 24-hour calculations but excluded from the day and nighttime calculations), or according to diary entries.

Data Collection and Variables

Data collected from chart review included age at first visit, gender, race (African American [AA], white, white Hispanic, Caribbean Hispanic, Asian, or African, as designated in the patient record), anthropometrics (weight in kilograms, height in meters), family history of hypertension (yes/no), CBP in mm Hg, and laboratory results (serum sodium, potassium, calcium, total cholesterol, and triglyceride concentrations all in mmol/L and plasma renin activity in ng/mL per hour). Results of ABPM measurements and echocardiographic evaluation (left ventricular [LV] end-diastolic dimension, posterior wall thickness, intraventricular septal thickness, and diagnosis by a cardiologist) were also collected. The data were obtained by manual chart review by 2 investigators who separately entered the variables into a database; the data entry was later reconciled to ensure data validity.

BMI was calculated for each child. Growth charts from the Centers for Disease Control and Prevention were used to determine percentiles for each child's height, weight, and BMI.[16] We calculated each child's casual 95th-percentile BP index by dividing their CBP by the 95th-percentile BP for age, gender, and height according to normative values from the 2004 National High BP Education Program working-group report.[12] A BP index of ≥1 was considered elevated. Similar calculations were conducted to determine the ABPM index. For ABPM studies, BP loads were calculated as the percentage of readings above the 95th percentile. Because of the lack of normative pediatric ABPM data that included minority children, the ABPM studies were analyzed by using CBP threshold cutoffs as defined by the National High BP Education Program.

LV mass (LVM) was calculated by using the Devereaux equation: LVM (in grams) = 0.81 [1.04(intraventricular septal thickness + posterior wall thickness + LV end diastolic internal dimension)^3 − (LV end diastolic internal dimension)^3] + 0.06. LVM index (LVMI) was calculated as LVM/(height in meters)$^{2.7}$. This indexing method standardized LVM measurements were according to body size without discounting the effects of overweight and obesity, and allowed for comparison of children with recently published norms.[17] LVM measurements were considered to indicate LV hypertrophy (LVH) in our study participants if the cardiologist who interpreted the echocardiogram diagnosed LVH or if the child's calculated LVMI was in the 95th percentile or higher.[17]

Children were categorized as AA or non-AA. The AA group consisted of children who were AA and those who were African. All other children were included in the non-AA group. We also analyzed data stratified according to age (<13 or ≥13 years), because previous data have shown that age and pubertal status may have an impact on normal BP differences in children of different races.[10]

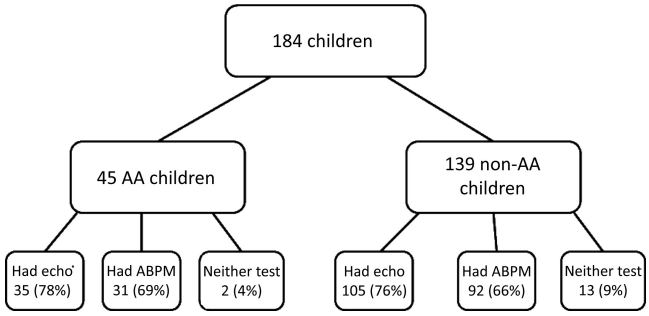

FIGURE 1
Number of children who underwent each diagnostic procedure, according to race. *P* value was not significant for all comparisons between groups (had echocardiogram [echo], had ABPM, and did not have either test).

Statistical Analyses

Continuous variables in this study were calculated as means and SDs and categorical variables as frequencies and percentages. Student's *t* tests and χ^2 analyses were used to compare demographic, anthropometric, and clinical data between children in the different groups. *P* values of <.05 were considered significant. All analyses were conducted by using Stata Statistical Software 10.1 (Stata Corp LP, College Station, TX).

RESULTS

There were 184 children who met study criteria at the 3 institutions. Among these children, 45 were AA and 139 were non-AA. Of the 184 children, 140 underwent echocardiography, 123 underwent 24-hour ABPM, and 94 underwent both ABPM and echocardiography. Of the 140 echocardiogram reports, 89 had the required measurements to calculate LVMI. There were no age, gender, or racial differences between those children who underwent either ABPM or echocardiography and those who did not, or between those who underwent both procedures and those who did not (Fig 1 and data not shown).

The clinical, laboratory, and CBP values for all 184 children according to racial group and age are listed in Table 1. When the data were not stratified according to age, analysis results indicated that AA children had a higher prevalence of overweight/obesity (BMI percentile ≥ 85%; 87% vs 71%; *P* = .03), a higher prevalence of LVH (49% vs 30%; *P* = .05), and a higher plasma renin activity (7.5 vs 3.9; *P* = .04) compared with non-AA children. Among the ABPM measurements, AA children had a higher 24-hour mean systolic BP (SBP) index (1.01 ± 0.1 vs 0.97 ± 0.1; *P* = .04), higher daytime SBP index (1.05 ± 0.1 vs 1.0 ± 0.1 ng/mL per hour; *P* = .02), higher nocturnal SBP index (0.96 ± 0.1 vs 0.91 ± 0.1; *P* = .03), and greater nocturnal diastolic load (31% ± 30% vs 19% ± 22%; *P* = .02) compared with non-AA children.

After the data were stratified according to age, analysis results indicating the increased prevalence of overweight/obesity and LVH among AA children compared with non-AA children persisted only among those children aged 13 years or younger, and these results were no longer significant among those aged 13 years or older. Similar results were found for plasma renin activity. Casual diastolic BP, both measured as mm Hg and indexed to age, gender, and height norms, was higher in AA adolescents compared

TABLE 1 Characteristics According to Race and Age of 184 Children With Primary Hypertension at the Time of Referral for Hypertension

Characteristics	AA Children ($N = 45$)		Non-AA Children ($N = 139$)		P for Age < 13 y[a]	P for Age ≥ 13 y[a]
	Age < 13 y ($n = 24$)	Age ≥ 13 y ($n = 21$)	Age < 13 y ($n = 56$)	Age ≥ 13 y ($n = 83$)		
Age, mean ± SD, mo	119 ± 29	185 ± 20	114 ± 32	188 ± 18	.5	.6
Male, *n* (%)	14 (58)	18 (86)	27 (48)	60 (72)	.4	.2
Height percentile, mean ± SD	84 ± 19	62 ± 27	69 ± 30 ($n = 54$)	66 ± 28 ($n = 82$)	.03	.5
Weight, mean ± SD, kg	60 ± 25	95 ± 37	52 ± 24 ($n = 55$)	87 ± 26	.2	.3
BMI, mean ± SD	26 ± 6	32 ± 11	25 ± 7 ($n = 54$)	29 ± 9	.4	.2
BMI percentile, mean ± SD	95 ± 8	85 ± 24	87 ± 20 ($n = 54$)	85 ± 20 ($n = 82$)	.06	.9
BMI percentile ≥85%, *n* (%)	23 (96)	16 (76)	40 (74) ($n = 54$)	56 (69) ($n = 82$)	.02	.5
Family history of hypertension, *n* (%)	21 (95) ($n = 22$)	20 (95)	44 (81) ($n = 54$)	70 (90) ($n = 78$)	.2	.7
Average SBP, mean ± SD, mm Hg	133 ± 9	144 ± 10	130 ± 15	141 ± 11	.4	.2
Average DBP, mean ± SD, mm Hg	74 ± 9	82 ± 12	74 ± 10	76 ± 11	.9	.03
Casual SBP 95% index, mean ± SD[b]	1.1 ± 0.06	1.1 ± 0.07	1.1 ± 0.1 ($n = 54$)	1.07 ± 0.08 ($n = 82$)	.7	.2
Casual DBP 95% index, mean ± SD	0.93 ± 0.11	0.98 ± 0.15	0.95 ± 0.12 ($n = 54$)	0.90 ± 0.13 ($n = 82$)	.6	.02
Plasma renin activity, mean ± SD, ng/mL per h	8.7 ± 15 ($n = 16$)	6.0 ± 12 ($n = 13$)	3.6 ± 2.7 ($n = 35$)	4.1 ± 6.4 ($n = 51$)	.05	.4
LVH, %[c]	56 ($n = 18$)	41 ($n = 17$)	26 ($n = 43$)	34 ($n = 62$)	.04	.6

DBP indicates diastolic BP.

[a] Student's *t* test or χ^2 analysis.

[b] BP index is measured SBP or DBP/95th-percentile SBP or DBP.

[c] Based on LVMI ≥ 95th percentile or cardiologist diagnosis.

with non-AA adolescents, a finding not present in the children younger than 13 years. There was a statistically significant difference in serum sodium concentrations between the AA and non-AA adolescents that was small and not clinically significant (1.2 mmol/L). When the 2 groups were stratified according to age, there were no differences between the 2 groups in concentrations of serum potassium, calcium, total cholesterol, or triglycerides.

The ABPM results revealed that AA adolescents had higher diastolic BP during the day and during 24-hour time periods, as well as higher SBP during the day, compared with non-AA adolescents. In addition, SBP in AA adolescents was elevated for a longer period during the night and the overall 24-hour period (as shown by their SBP load) than SBP in non-AA adolescents (Table 2). There was no difference in the nocturnal dipping pattern (ie, no difference in the percentage of children who experienced a 10% or greater decline in their BP at night) between the groups.

DISCUSSION

BP normally increases throughout childhood, and earlier studies have revealed that race has an impact on this expected increase among normotensive children, with white children for the most part having higher BPs than black children.[10] In our study we also observed a differential impact of age and race on the degree of BP elevation among children with primary hypertension. Although there was no difference in BP among hypertensive children younger than 13 years, among children 13 years of age and older, black adolescents had more pronounced BP elevations than nonblack adolescents, as evidenced by the higher indexed BP observed for both casual and ambulatory measurements

TABLE 2 ABPM of 123 Children With Primary Hypertension at the Time of Referral for Hypertension

Characteristics	AA Children ($N = 31$)		Non-AA Children ($N = 92$)		P for Age < 13 y[a]	P for Age ≥ 13 y[a]
	Age < 13 y ($n = 13$)	Age ≥ 13 y ($n = 18$)	Age < 13 y ($n = 34$)	Age ≥ 13 y ($n = 58$)		
24-h mean SBP index[b]	1.02 ± 0.1 ($n = 11$)	1.0 ± 0.1 ($n = 9$)	0.99 ± 0.1 ($n = 27$)	0.95 ± 0.1 ($n = 34$)	.3	.08
24-h mean DBP index	0.9 ± 0.1 ($n = 11$)	0.90 ± 0.1 ($n = 9$)	0.9 ± 0.1 ($n = 27$)	0.80 ± 0.1 ($n = 34$)	.9	.05
Daytime mean SBP index	1.06 ± 0.1 ($n = 11$)	1.04 ± 0.1 ($n = 9$)	1.02 ± 0.1 ($n = 26$)	0.98 ± 0.1 ($n = 34$)	.3	.04
Daytime mean DBP index	0.94 ± 0.1 ($n = 11$)	0.94 ± 0.1 ($n = 9$)	0.95 ± 0.1 ($n = 26$)	0.85 ± 0.1 ($n = 34$)	.7	.02
Nocturnal mean SBP index	0.98 ± 0.1 ($n = 11$)	0.94 ± 0.1 ($n = 9$)	0.92 ± 0.1 ($n = 27$)	0.90 ± 0.1 ($n = 32$)	.07	.3
Nocturnal mean DBP index	0.82 ± 0.1 ($n = 11$)	0.81 ± 0.2 ($n = 9$)	0.79 ± 0.1 ($n = 27$)	0.75 ± 0.1 ($n = 24$)	.5	.2
24-hour SBP load, %[c]	59 ± 34	57 ± 26	58 ± 27	41 ± 28	.9	.04
24-hour DBP load, %	33 ± 29	28 ± 21	30 ± 22	19 ± 18	.6	.06
Daytime SBP load, %	60 ± 35	56 ± 32	59 ± 27 ($n = 32$)	40 ± 30 ($n = 57$)	.9	.06
Daytime DBP load, %	32 ± 28	29 ± 23	33 ± 21 ($n = 32$)	20 ± 19 ($n = 57$)	.9	.1
Nocturnal SBP load, %	65 ± 35	63 ± 28	62 ± 35 ($n = 32$)	46 ± 32 ($n = 55$)	.8	.05
Nocturnal DBP load, %	31 ± 35	30 ± 28	19 ±19 ($n = 32$)	18 ± 23 ($n = 55$)	.1	.09

Data are presented as mean ± SD. DBP indicates diastolic BP.

[a] Student's *t* test or χ^2 analysis.

[b] BP index is measured SBP or DBP/95th-percentile SBP or DBP.

[c] BP load is the percentage of readings above the 95th percentile SBP or DBP.

in black adolescents. With hypertension a well-established cardiovascular risk factor,[18] this age-dependent difference in the degree of BP elevation suggests that age, and possibly pubertal status, may have a more significant impact on cardiovascular risk in black children than nonblack children. In other words, adolescent black children may have more severe hypertension than their nonblack counterparts, a finding that mimics the risk profiles of black adults compared with nonblack adults. This similarity also contributes to the argument that adult hypertension and its sequelae have their origins in childhood.[19]

Possible etiologies for these racial differences include issues related to access to care, dietary habits, and differences in vascular reactivity. Differences in heritability and socioeconomic status as a cause are also possibilities, but neither genetic characteristics nor lower socioeconomic status have been shown to account for the racial differences in BP seen in children.[20–22] It has also been postulated that low birth weight, more common in the black community than the white community, may be the biggest predictor of BP levels and development of hypertension.[23] Investigators have shown that at all ages, lower birth weight is associated with higher SBP, and this effect increases with increasing age.[24,25] Low birth weight can lead to reduced nephron mass, hyperfiltration, increased glomerular pressure, and ultimately hypertension. Although we unfortunately did not have birthweight information in this study population, the increased BP burden seen in hypertensive black adolescents is clearly a cause for concern and raises many issues that warrant additional investigation not only to determine the cause of this difference but also to measure the effect it might have on long-term cardiovascular risk and overall health status.

Another significant finding in this study was the increased prevalence of obesity/overweight and LVH among the black children younger than 13 years. It is interesting that the black adolescents did not have increased rates of these cardiovascular risk factors even though they had an increased BP burden as measured by CBP and ABPM. Given the conventional thinking that LVH is caused by increased LV afterload from sustained elevations in BP, this result seems counterintuitive. Our findings suggest that obesity, not race or degree of BP elevation, may play a bigger role in the development of LVH among hypertensive children. Results of earlier studies have corroborated this finding[26] and demonstrated that younger children who are obese are more likely to have clustering of cardiovascular risk factors than are older children. Specifically, investigators for the Bogalusa Heart Study reported that the presence of increasing numbers of cardiovascular risk factors was most strongly associated with obesity among children younger than 10 years, and that this association varied according to race.[11] When obesity/overweight prevalence was not different among the groups, and when the degree of BP elevation was increased, as was the case among those aged 13 years and older, the prevalence of LVH was not significantly different.

Findings of racial differences in obesity prevalence are not new. The National Health and Nutrition Examination Survey[27] revealed that younger non-Hispanic black children had more overweight/obesity (BMI $>$ 85th percentile) compared with non-Hispanic white children across all age strata. A large epidemiologic study also revealed that after they were 8 years old, likely coincident with puberty, both black and white girls had a significant increase in BMI. We were unable to further stratify our groups of children according to gender because of sample-size considerations, but given this evidence it is likely that the change in prevalence in overweight/obesity between groups of children younger than 13 years and 13 years and older was attributable to pubertal maturation.

In addition to having higher rates of overweight/obesity, black children younger than 13 years also had higher levels of plasma renin activity. Obesity has been thought to contribute to the development of hypertension by increasing sympathetic nervous system activity and by increasing salt and water retention by several mechanisms related to increased activity of the renin-angiotensin-aldosterone system. Results of studies in adults have shown that elevated aldosterone is more strongly associated with the development of hypertension in black and obese individuals[28,29] and that chronic exposure to aldosterone may lead to significant damage to the heart independent of BP level.[30] Although we did not measure aldosterone in our study participants, the finding of increased plasma renin activity among black children younger than 13 years with confirmed primary hypertension raises the possibility that these children may also have elevated aldosterone concentrations and that it is not race but exposure to aldosterone that is contributing to this difference in LVH prevalence. Of the other clinical variables we measured in our study participants at initial presentation, we also found that serum sodium concentrations differed among children aged 13 years and older, although this finding was not clinically significant and has little clinical relevance. A more suitable measurement would have been 24-hour urinary sodium quantification, which is something that should be assessed in future studies.

Although the cross-sectional nature of this study enables us only to describe differences and hypothesize causality, our data suggest that black children with primary hypertension may be at increased cardiovascular risk compared with nonblack hypertensive children. Our findings of higher rates of overweight/obesity and LVH in patients younger than 13 years and the increased BP burden as these children progress into adolescence support this conclusion. Although it is possible that the higher prevalence of LVH found in black children younger than 13 years may be influenced by the larger number of children in this group with overweight/obesity, this finding does not eliminate the fact that this group of children is at increased cardiovascular risk because they have hypertension, obesity, and LVH.

There are several limitations to these data. As stated above, the cross-sectional study design did not allow for an inference of causality. Some children did not undergo all laboratory tests, echocardiography, and/or ABPM, and for those children who did undergo ABPM, some ABPM variables of interest were unavailable in a small percentage of patients. In addition, we were not able to collect data regarding additional variables of interest, specifically birth weight, gestational age, pubertal status, aldosterone concentrations, 24-hour urinary sodium excretion, and presence of microalbuminuria. Finally, the somewhat small sample size in each race/age stratum limited our analyses and may have contributed to our inability to demonstrate significant differences. Despite these limitations, we observed significant differences between the study groups that were consistent with data reported in the literature in regard to adult as well as pediatric hypertension.

A unique feature of this study was that despite the fact that patients were seen for an extended time period and at 3 institutions, all patients were evaluated by use of a common protocol. This consistency was possible only because of the overlapping working relationships of the investigators. In addition, the diverse referral areas of the 3 centers helped us to generate a multiethnic study cohort not possible at a single pediatric center.

The results of this study have several important implications. Results of studies in adults have shown that BP burden, measured by length of time with elevated BP, may contribute to the more rapidly advancing renal disease seen in black adults. The finding that the black children aged 13 years and older in our study demonstrated a higher BP load (ie, a longer period of time with elevated BP as measured by 24-hour ABPM) may have implications for young black adults and their risk for future renal disease. In addition, although we found no racial differences in the rate of overweight/obesity or presence of LVH in the adolescent ($\geq$13 years) age group, more than half of all children with primary hypertension were overweight or obese and 35% overall had LVH. Even with the use of the most recent normative data, which provided the 95th percentile LVMI for children according to age and gender,[17] we found the prevalence of LVH to be consistent with earlier reports.[12,31,32] Because hypertension in childhood has been shown to persist into adulthood more readily with increasing BMI, increased efforts at lifestyle modification and control of hypertension are critical for children with primary hypertension, because they already manifest several cardiovascular risk factors.

CONCLUSIONS

We report significant racial differences among a diverse group of children with confirmed primary hypertension. These findings are similar to those reported in the literature for adult study participants and suggest that black children with hypertension may be at greater cardiovascular risk because of their increased prevalence of obesity, LVH, and more pronounced BP elevations. More studies are needed to enable us to better elucidate the etiology of the differences seen between black and nonblack children with hypertension so that we may be better equipped to develop focused preventive efforts. Methods to enhance the early diagnosis and treatment of pediatric hypertension will not only positively influence the health of these children but may have a positive impact on their cardiovascular morbidity and mortality later in life.

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