# Essential Hypertension Predicted by Tracking of Elevated Blood Pressure From Childhood to Adulthood: The Bogalusa Heart Study 

Weihang Bao, Sam A. Threefoot, Sathanur R. Srinivasan, and Gerald S. Berenson

It is well known that blood pressure (BP) levels persist over time. The present investigation examines tracking of elevated BP from childhood to adulthood and its progression to essential hypertension.

In a community study of early natural history of arteriosclerosis and essential hypertension, a longitudinal cohort was constructed from two cross-sectional surveys $>15$ years apart: 1505 individuals ( $56 \%$ female subjects, $35 \%$ black), aged 5 to 14 years at initial study.

Persistence of BP was shown by significant correlations between childhood and adulthood levels ( $r=0.36$ to 0.50 for systolic BP and $r=0.20$ to 0.42 for diastolic BP), varying by race, sex, and age. These correlations remained the same after controlling for body mass index (BMI). Twice the expected number of subjects ( $40 \%$ for systolic BP and $37 \%$ for diastolic BP), whose levels were in the highest quintile at childhood, remained there 15 years later. Furthermore, of the childhood characteristics, baseline BP level was most predictive of the follow-up level, followed by change in BMI.

Subsequently, even at ages 20 to 31 years, prevalence of clinically diagnosed hypertension was

[^0]much higher in subjects whose childhood BP was in the top quintile: 3.6 times ( $18 \% \mathrm{v} 5 \%$ ) as high in systolic BP and 2.6 times ( $15 \% v 5.8 \%$ ) as high in diastolic BP, compared to subjects in every other quintile. Of the 116 subjects who developed hypertension, $48 \%$ and $41 \%$ had elevated childhood systolic and diastolic BP, respectively. Hypertension that developed in early adulthood was more prevalent in blacks, in subjects who had higher BP or BMI in childhood, or had gained more BMI from childhood to adulthood. The prediction of hypertension by earlier BP level was enhanced by multiple examinations. Estimated from 419 subjects who participated in four other surveys, individuals showing elevated BP levels at multiple times were more likely to develop future hypertension.

Elevated BP levels persist over time and progress to adult hypertension. Repeated measurements of BP early in life improve the prediction of adult hypertension. Am J Hypertens 1995;8:657-665

KEY WORDS: Longitudinal study, children and young adults, persistence of blood pressure, hypertension, tracking.

Essential hypertension has long been identified as a potent cardiovascular disease risk factor in adults. ${ }^{1}$ It is an underlying basis of congestive heart failure, cerebrovascular accidents, and renal failure and is commonly associated with diabetes mellitus. Early treatment of hypertension has been shown to reduce significantly subsequent cardiovascular morbidity and mortality. ${ }^{2}$ How-
ever, hypertension is poorly understood in its early asymptomatic stages, where development of hypertension involves complex and multiple mechanisms. Among adolescents and young adults, elevated blood pressure is also associated with the presence of early atherosclerotic lesions. ${ }^{3}$ Therefore, understanding the persistence of blood pressure over time and its progression into clinical hypertension would aid in early identification and prevention of hypertension. ${ }^{46}$
Persistence (tracking) of elevated blood pressure over time has been demonstrated in children. ${ }^{7-18}$ However, few large-scale epidemiology studies have shown the long-term persistence of elevated blood pressure from childhood into adulthood, and more important, how the persistent elevation in blood pressure ultimately develops into adult hypertension. Studies of this type not only help understand when and how adult hypertension occurs, but also have implications for early detection and perhaps prevention of the development of hypertension by appropriate dietary and lifestyle modifications.
In a community-based biracial population, the present study examines the tracking of blood pressure from childhood to adulthood, and the predictability of adult hypertension from childhood blood pressure. The enhancement of this predictability by multiple observations was also assessed in a subsample.

## MATERIALS AND METHODS

Population Two cross-sectional surveys were conducted in 1973 to 1974 and 1988 to 1991 as part of the Bogalusa Heart Study, a long-term epidemiologic study of cardiovascular disease risk factors in a biracial ( $64 \%$ white and $36 \%$ black) population of children and young adults. ${ }^{19}$ Chosen for the current study were 1505 subjects who were in both the 1973 to 1974 (baseline) and the 1988 to 1991 (follow-up) surveys. The subjects were 5 to 14 years old at baseline and 20 to 31 years old at follow-up. Age, race, and sex distribution of the study population are given in Table 1. At baseline, participants in the current study did not differ from nonparticipants ( $\mathrm{n}=2360$ ) in weight, height, and blood pressure (data not shown).

General Examinations All examinations followed the same protocols. ${ }^{19}$ Subjects were instructed to fast for 12 h before the screening, and compliance was determined by an interview on the morning of the examination. Blood was drawn by antecubital venipuncture to obtain serum and plasma.
Height was measured to within 0.1 cm , weight to within 0.1 kg , and subscapular and triceps skinfolds to within 1.0 mm . As a measure of obesity, the body mass index (BMI $=$ weight [in kilograms]/height [in

TABLE 1. AGE, RACE AND SEX DISTRIBUTION OF THE BOGALUSA HEART STUDY COHORTS

| Race/Sex | Age <br> 5 to 9 <br> Years | Age 10 to 14 Years | Total |
| :---: | :---: | :---: | :---: |
| Whites |  |  |  |
| Male | 176 | 261 | 437 |
| Female | 232 | 309 | 541 |
| Blacks |  |  |  |
| Male | 101 | 121 | 222 |
| Female | 142 | 163 | 305 |
| Total | 651 | 854 | 1505 |

Values are numbers of subjects for each subgroup.
meters $]^{2}$ ) was calculated. Blood pressure levels were measured on the right arm with subjects in a relaxed, sitting position. The cuff size used for blood pressure determinations was based on measurements of right arm length and circumference. Systolic blood pressure was recorded at the first Korotkoff phase, and diastolic at fourth and fifth phases. The fifth phase was used to determine diastolic hypertension in young adults, and the fourth phase was used in the rest of the analyses. The blood pressure levels reported were the mean of six replicate readings taken by two randomly assigned trained nurses. During an interview, the subjects were also asked if they had ever been treated for hypertension. In the follow-up examination parental history of disease, including hypertension, was obtained through a self-reported questionnaire.

Statistical Analyses The Statistical Analysis System was used. ${ }^{20}$ Changes in blood pressure over time were evaluated by analysis of variance. Stratified by age, Pearson correlations were used to examine the associations between baseline and follow-up levels, with and without adjustment for BMI. The persistence of high and low blood pressure over time was then illustrated by constructing a baseline race-, sex-, and age-specific quintiles of the blood pressure.

The predictability of follow-up blood pressure was examined in a stepwise regression analysis. Independent variables included baseline blood pressure, age, race, sex, weight, height, and BMI, and changes in weight, height, and BMI from baseline to follow-up.

At follow-up, subjects were determined to be clinically hypertensive if they had a systolic blood pressure $>140 \mathrm{~mm} \mathrm{Hg}$, or fifth phase diastolic blood pressure $>90 \mathrm{~mm} \mathrm{Hg}$, or had been treated for hypertension. Prevalence of hypertension was analyzed with respect to previous blood pressure levels. To study independent predictors of hypertension, a stepwise logistic regression analysis was also conducted, using age, race, sex, parental history of hypertension, baseline systolic and diastolic blood pressure, baseline

BMI, and change of BMI from baseline to follow-up as independent variables. In a subgroup of the population who were examined in four other cross-sectional surveys between baseline and follow-up, following the same protocol, the number of prior blood pressure elevations ( $>80$ th percentile) was counted and examined with respect to hypertension.

## RESULTS

Longitudinal Changes of Blood Pressure Baseline and follow-up systolic and diastolic blood pressures are given by race, sex, and age in Figures 1 and 2. Irrespective of race, sex, and age of the cohort, significant increases were observed in blood pressure as expected. Increase of both systolic and diastolic blood pressures were more apparent in male than in female subjects, more apparent in the younger subjects than in the older ones.

Persistence of Blood Pressure Subjects with relatively high blood pressure levels at childhood were more likely to remain at high levels in adulthood. As shown in Table 2, among different race, sex, and age groups, year 1 versus year 15 systolic blood pressure correlations ranged from 0.36 to 0.50 ( $P=.0001$ ). They were not significantly different from each other. Similar persistence was seen in diastolic blood pressure, especially in the older age group ( 10 to 14 years old at baseline), in whom the correlations ranged from 0.29 to 0.42 ( $P \leq .001$ ). These correlations remained virtually the same after controlling for BMI.

As an illustration of tracking, the percentage of individuals who ranked high or low in blood pressure levels at baseline and maintained these respective ranks at follow-up were examined. In Figure 3, baseline and follow-up blood pressures are stratified by


FIGURE 1. Longitudinal changes in systolic blood pressure from childhood into young adulthood by age, race, and sex over a 15 -year period in the Bogalusa Heart Study ( $n=1505$ ). Systolic blood pressure increased significantly, more apparent in male subjects and younger age groups. $W M=$ white male; $W F=$ white female; $B M=$ black male; $B F=$ black female.


FIGURE 2. Longitudinal changes in diastolic blood pressure from childhood into young adulthood by age, race, and sex over a 15 -year period in the Bogalusa Heart Study ( $n=1505$ ). Diastolic blood pressure increased significantly, more apparent in male subjects and younger age groups. $W M=$ white male; $W F=$ white female; $B M=$ black male; $B F=$ black female.
quintiles. Of those individuals who had systolic blood pressure levels above the 80th percentile at baseline, $40 \%$ had levels above this percentile 15 years later. Another 23\% had levels between 60th and 80th percentile. Only $7 \%$ had levels below the 20th percentile. A similar persistence for low ranks was seen at the lower quintiles.

As for the diastolic blood pressure, weaker but similar persistence was observed. Among individuals whose diastolic blood pressure levels were above the 80th percentile at baseline, $37 \%$ remained in the top quintile 15 years later. Nine percent had levels below the 20th percentile.
Correlates of Follow-up Blood Pressure A stepwise multiple regression was used to determine the relative predictability of selected variables for the blood pressure after 15 years. Significant predictors are given in Tables 3 and 4. The regression coefficients indicate the difference in follow-up level corresponding to a unit change of the predictors. For example, among the white male subjects, a 10 mm Hg higher baseline systolic blood pressure level predicted a 5 mm Hg higher follow-up level; a $10 \mathrm{~kg} / \mathrm{m}^{2}$ higher BMI predicted a 7 mm Hg higher follow-up systolic blood pressure level. The relative importance of the predictors is indicated by the standardized regression coefficients. As might be expected, irrespective of race and sex, the best predictor of follow-up systolic blood pressure level was the baseline level. Change of weight, especially relative to height (BMI), was also predictive. Among the race-sex groups, these variables explained $22 \%$ to $32 \%$ of the variability of the follow-up systolic blood pressure. When all the subjects were studied together, systolic blood pressure averaged 2 mm Hg higher in blacks than in whites,

TABLE 2. LONGITUDINAL PEARSON CORRELATION BETWEEN CHILDHOOD AND ADULT BLOOD PRESSURE OVER A 15-YEAR PERIOD: THE BOGALUSA HEART STUDY

| Age, Race and Sex | Systolic BP |  | Diastolic BPt |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Unadjusted | BMI Adjusted* | Unadjusted | BMI Adjusted |
| Age 5-9 at year 1 |  |  |  |  |
| White male | .43\# | .41\# | . 261 | .28 ${ }^{11}$ |
| White female | .49\# | .52\# | .29\# | 28\# |
| Black male | .39\# | . 38 \# | .19 $\ddagger$ | $22 \ddagger$ |
| Black female | .38\# | .42\# | .21§ | 21§ |
| Age 10-14 at year 1 |  |  |  |  |
| White male | .47\# | . 42 \# | 42\# | .38\# |
| White female | . 36 \# | .42\# | .29\# | .33\# |
| Black male | . 39 \# | .41\# | .38\# | 40\#\# |
| Black female | .50\# | .47\# | .41\# | . 35 \# |

$\ddagger \mathrm{P} \leq .05 ; \S \mathrm{P} \leq .01 ; \mathrm{P} \leq .001 ; \# \mathrm{P} \leq .0001$.
tIn white male and black female subjects, correlation between year 1 and year 16 diastolic blood pressure was significantly higher in the older age group than in the younger age group.
*Adjusted for both childhood (year 1) and adulthood (year 16) BMI.
and 5 mm Hg higher in male than in female subjects, independent of weight gain or increase in BMI.

To a smaller extent, but similarly, baseline diastolic blood pressure level was the best predictor of the follow-up level: every 10 mm Hg higher level at the baseline predicted 3 mm Hg higher level at the fol-low-up in the total population. The change of BMI from baseline to follow-up was also the next best predictor. It was noted in black male subjects that aging contributed independently to the follow-up blood pressure. These predictors explained $13 \%$ to $25 \%$ of the variability in the follow-up diastolic blood pressure levels, varying by race and sex. Examination on the total population did not show a race difference in the prediction equation, but showed a difference of 4 mm Hg higher levels in male subjects.

Hypertension in Young Adults At the follow-up examination, 116 (7.7\%) subjects had developed hyper-
tension, 99 of whom had received medical treatment. The prevalence of hypertension is given by age-, race-, and sex-specific quintiles of baseline blood pressure level (Figure 4). Subjects who were in the highest quintile were much more likely to develop hypertension: 3.6 times ( $18 \% v 5 \%$ ) in systolic blood pressure and 2.6 times ( $15 \% \mathrm{v} 5.8 \%$ ) in diastolic blood pressure. All others had similar low prevalence around $5 \%$, indicating that the hypertensive subjects were primarily from the top quintile. It was observed that at baseline, $48 \%$ of the hypertensive subjects showed systolic elevation and $41 \%$ showed diastolic elevation.

To study the independent contribution from obesity, blood pressure tracking, or parental history of hypertension to adult hypertension, a stepwise logistic regression analysis was conducted. The presence of hypertension in adulthood was used as the dependent variable. Independent variables included age,


FIGURE 3. Tracking of blood pressure ( $B P$ ) over 15 years by age-, race-, and sex-specific quintiles from childhood to young adulthood in the Bogalusa Heart Study ( $n=1505$ ). Individuals who had elevated blood pressure ( $>80$ th percentile) in childhood tended to have elevated blood pressure 15 years later, $40 \%$ ranked by systolic blood pressure and $37 \%$ ranked by diastolic blood pressure. Similar persistency was observed in subjects who ranked low in blood pressure.

## TABLE 3. PREDICTORS OF FOLLOW-UP SYSTOLIC BLOOD PRESSURE BY RACE AND SEX IN THE BOGALUSA HEART STUDY COHORT

| Independent Variablet | White Males $(n=432)$ | White Females ( $\mathrm{n}=538$ ) | Black <br> Males $(n=218)$ | Black Females ( $\mathrm{n}=299$ ) | Total $(\mathrm{n}=1487)$ |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Baseline systolic BP (mm Hg) | 0.50 (0.49) | 0.44 (0.47) | 0.47 (0.40) | 0.45 (0.45) | 0.46 (0.42) |
| $\Delta$ BMI ( $\mathrm{kg} / \mathrm{m}^{2}$ ) | 0.71 (0.27) |  | 1.00 (0.31) | $0.23(0.18)^{*}$ | 0.33 (0.14)* |
| $\Delta \mathrm{Wt}$ (kg) |  | 0.20 (0.35) |  |  | 0.08 (0.14) |
| Black $v$ white | - | - | - | - | 2.17 (0.10) |
| Male $v$ female | - | - | - | - | 4.73 (0.22) |
| $\mathrm{r}^{2}$ | 0.29 | 0.26 | 0.24 | 0.21 | 0.32 |

Values are regression coefficients (standardized regression coefficients).
${ }^{*} \mathrm{P} \leq .001$; others: $\mathrm{P} \leq .0001$.
tIndependent variables included age, race, and sex, baseline systolic blood pressure (BP), weight ( $\mathrm{W} t$ ), height and body mass index (BMI), and changes from baseline to follow-up in weight, height, and BMI. Variables entered the model at $\mathbf{P} \leq .05$. Baseline $=5$ to 17 years of age; follow-up $=20$ to 31 years of age.
race, sex, parental history of hypertension, baseline systolic and diastolic blood pressure, baseline BMI, and change of BMI from baseline to follow-up. As shown in Table 5, adult onset hypertension was more prevalent in blacks, and more prevalent in subjects who had higher blood pressure or BMI in childhood, or had gained more weight (with respect to height) from childhood to adulthood. Interestingly, compared to whites, blacks were shown to acquire hypertension more rapidly with aging in early adulthood.

## Multiple Examinations in the Prediction of Hyper-

 tension Within the study population, 419 ( $28 \%$ ) of the 1505 subjects had participated in four other crosssectional surveys approximately 3 years apart between baseline and follow-up. Twenty-four ( $5.7 \%$ ) of these subjects had developed hypertension. Using the five previous examinations, the prediction of hypertension was greatly improved, with the likelihoodof detection increased with the risk of hypertension. By ranking within the respective race, sex, and age groups in each survey, a blood pressure previously elevated was defined as those above the 80th percentile. In Figure 5, the hypertension status at follow-up examination was related to the number of previous blood pressure elevations. As expected, the prevalence of hypertension in subjects with four or five prior elevations was much higher than in those with only one elevation, indicating the higher the subjects were at risk, the more likely they would be identified. It should be noted that the actual prevalence of hypertension within subjects who had multiple blood pressure elevations should be much higher than that estimated in this subpopulation of 419 individuals. This was not only because hypertension was more prevalent in the total study population $(7.7 \%)$ than in this subpopulation ( $5.7 \%$ ), but also because the adults were only 20 to 31 years of age.

TABLE 4. PREDICTORS OF FOLLOW-UP DIASTOLIC BLOOD PRESSURE BY RACE AND SEX IN THE BOGALUSA HEART STUDY COHORT

| Independent Variablet | White Males ( $\mathrm{n}=432$ ) | White <br> Females $(n=538)$ | $\begin{gathered} \text { Black } \\ \text { Males } \\ (\mathrm{n}=218) \end{gathered}$ | Black Females ( $\mathrm{n}=299$ ) | Total $(\mathrm{n}=1487)$ |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Baseline diastolic BP ( mm Hg ) | 0.32 (0.32) | 0.27 (0.31) | 0.39 (0.31) | 0.38 (0.35) | 0.33 (0.31) |
| $\triangle \mathrm{BMI}\left(\mathrm{kg} / \mathrm{m}^{2}\right)$ | 0.63 (0.29) | 0.34 (0.21) | 0.95 (0.36) |  | 0.39 (0.21) |
| Baseline Wt (kg) | 0.11 (0.21) |  |  |  |  |
| Baseline Ht (cm) |  | $0.05(0.12)^{*}$ |  |  | 0.06 (0.13) |
| Age (yr) |  |  | 0.73 (0.21) |  |  |
| Male $v$ female | - | - | - | - | 3.66 (0.21) |
| $\mathrm{r}^{2}$ | 0.24 | 0.15 | 0.26 | 0.13 | 0.21 |

[^1]

FIGURE 4. Association between childhood blood pressure and prevalence of adult hypertension over 15 years in the Bogalusa Heart Study ( $n=1505$ ). It was the subjects who ranked in the highest age-, race-, and sex-specific quintile of baseline blood pressure developed hypertension even when they were 20 to 31 years old.

## DISCUSSION

The current study is based on the community population of Bogalusa, Louisiana, in which blood pressure was monitored repeatedly as part of cardiovascular risk factor surveys. Several previous reports from this community have shown that blood pressure tracked for periods of 3 and 8 years in children and adolescents. ${ }^{8-10}$ The current study noted that the tracking phenomenon was not much weaker over a remarkably longer period of 15 years from childhood to adulthood and independent of growth in weight/ height ${ }^{2}$. In addition, this study demonstrated that earlier blood pressure elevations could ultimately become recognizable hypertension. These findings agree with or can be anticipated from studies within other populations. ${ }^{7,11,13,14,21}$ The degree of tracking
of blood pressure may vary among studies, partly attributable to the method of recording. ${ }^{22-24}$ Also the current study shows that multiple observations early in life greatly helped in predicting future hypertension. This finding is in agreement with our previous study, which observed that individuals classified as high in blood pressure by multiple measurements were more likely to remain high after 8 years. ${ }^{25}$ Exactly how many measurements during the growth period are adequate to establish a risk of developing hypertension remains to be explored. In the current study it was also shown that other than baseline blood pressure, change of weight or BMI had a strong influence on future blood pressure. This agrees with many studies showing a positive association between body size and blood pressure in both pediatric and adult populations. ${ }^{7,26-29}$

TABLE 5. PREDICTORS OF ESSENTIAL HYPERTENSION IN ADULTS 20 TO 31 YEARS OF AGE BY RACE AND SEX IN THE BOGALUSA HEART STUDY COHORT

| Independent Variablest | White Males ( $\mathrm{n}=432$ ) | White Females ( $\mathrm{n}=538$ ) | $\begin{gathered} \text { Black } \\ \text { Males } \\ (\mathrm{n}=218) \end{gathered}$ | Black Females ( $\mathrm{n}=299$ ) | Total $(\mathrm{n}=1487)$ |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Age (5-year difference) |  |  | 1.3* | 2.2 * |  |
| Baseline systolic BP (107v 93 mm Hg ) | 2.6 * | 1.7 | $2.6 \ddagger$ | $2.3 \ddagger$ | $2 \S$ |
| Baseline diastolic $\mathrm{BP}^{*}$ ( $68 v 57 \mathrm{~mm} \mathrm{Hg}$ ) | 2.1 |  |  |  | 1.5* |
| Baseline BMI ( $19.1 v 15.3 \mathrm{~kg} / \mathrm{m}^{2}$ ) | 1.5 |  |  | $1.6 \ddagger$ | $1.3 *$ |
| $\begin{aligned} & \Delta \mathrm{BMI} \\ & \quad\left(9.8 v 7.5 \mathrm{~kg} / \mathrm{m}^{2}\right) \end{aligned}$ | $1.7^{*}$ | $1.9 \ddagger$ | $2.4 \ddagger$ |  | $1.6{ }^{\text {II }}$ |
| Black $v$ white | - | - | - | - | 2.41 |

[^2]

Frequency of Previous BP > 80th Percentile

FIGURE 5. Prevalence of adult hypertension related to the frequency of blood pressure (BP) elevations (within the top $20 \%$ rankings by age, race, and sex) in five previous surveys of Bogalusa Heart Study. Individuals detected at multiple times were more likely to develop hypertension even at age 20 to 31 years.

The importance of early blood pressure elevation and its persistence into adulthood is underscored by the fact that complications related to essential hypertension, such as the presence of anatomic cardiovas-cular-renal changes, already exist in children. ${ }^{3,30,31}$ Understanding this concept is important, as anatomic evidence of hypertension and target organ changes occur in childhood at much lower levels than those considered clinically abnormal in adulthood. Autopsy studies have shown that young individuals with higher blood pressure levels have increased vascular endothelial thickness, a response of intimal fibroplasia, medial wall thickness, and a vascular cellular response. ${ }^{32,33}$ Echocardiographic studies in pediatric age groups have shown that elevated blood pressure, at the 90 th percentile, was associated with increases of left ventricular measurements ${ }^{34-37}$ and alterations in hemodynamic functions. ${ }^{38,39}$ Ultrasonic studies for carotid artery stiffness have indicated increased wall stiffness, aorta stiffness, and loss of elasticity in children with obesity and high blood pressure. ${ }^{40,41}$ In a recent study, young individuals whose blood pressures were in the upper quartiles showed a greater amount of microalbuminuria, especially in black male subjects. ${ }^{42}$

In the present study, the race-, sex-, and agespecific 80th percentiles were used to define blood pressure elevation in children. Other cutoff points may also be used to define blood pressure elevation. Studies have indicated that increasing the stringency of the prior measurement cutoff point will result in increased specificity and positive predictive value, but decreased sensitivity and negative predictive value. ${ }^{25}$ In general, abnormal blood pressure levels in children are much lower than the adult criteria for clinical diagnosis of hypertension, as shown by the above anatomic correlates of high blood pressure in young individuals.

The current study shows that childhood blood
pressure elevation serves as a good predictor of elevated blood pressure and hypertension in adulthood, especially when multiple observations are available. The required clinical practice is very simple, taking a blood pressure at each medical examination. This prediction will be improved by the inclusion of genetic and environmental correlates of blood pressure. The present study showed that both childhood weight (obesity) and weight gain during the growth toward adulthood independently predicted future hypertension. Particularly, the weight gain from childhood to adulthood, which was independently associated with future elevation of blood pressure, reflected strong influences from behavior and environment. Many studies have shown that parental history of hypertension relates to elevation of blood pressure in offspring at various age. ${ }^{10,43-47}$ In the current study, significance of parental history of hypertension for hypertension prediction in adult offspring was not observed. Even in the blood pressure prediction (Table 3), including parental history of hypertension, explained only $1 \%$ additional variability in white female subjects (data not shown). This implies that the genetic predisposition to hypertension represented by parental history of hypertension may be greatly accounted for by childhood blood pressure levels. These childhood levels already had a strong association with parental hypertension, irrespective of the young age of the offspring. ${ }^{47}$ Also, with the inaccuracies associated with self-reported parental history, the risk associated with parental history may not be fully apparent. ${ }^{47}$ Technical improvement in blood pressure measurement, such as ambulatory blood pressure monitoring, may also help predict future hypertension because of its greater reproducibility than casual, resting blood pressure. ${ }^{48}$

As a nonintervention study, the Bogalusa Heart Study pointed out that elevated blood pressure in children progresses long before the clinically evident
hypertension and hypertensive disease, at least as set by adult criteria. The impact from childhood elevation and from the change of obesity in this regard point to the need for preventive measures aimed at developing healthy lifestyles starting in childhood.

## ACKNOWLEDGMENTS

The Bogalusa Heart Study is a joint effort of many investigators and staff members whose cooperation is gratefully acknowledged. We especially thank the Bogalusa schools, teachers, parents, and most importantly the children and young adults from Bogalusa, Louisiana. We thank the Bogalusa Heart Study field staff and the Core Laboratory staff.

## REFERENCES

1. 1988 Joint National Committee: The 1988 Report of the Joint National Committee on detection, evaluation, and treatment of high blood pressure. Arch Intern Med 1988;148:1023-1038.
2. Hypertension Detection and Follow Up Program Cooperative Group: Five-year findings of the hypertension detection and follow up program: I. Reduction in mortality of persons with high blood pressure, including mild hypertension. JAMA 1979;242:2562-2571.
3. Newman WP, Freedman DJ, Voors AW, et al: Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Heart Study. N Engl J Med 1986;314:138-144.
4. Berenson GS, Cresanta JL, Webber LS: High blood pressure in the young. Annu Rev Med 1984;35:535560.
5. Lauer RM, Shekelle RB: Childhood Prevention of Atherosclerosis and Hypertension. New York, Raven Press, 1980.
6. Hoffman A: Blood pressure in childhood: an epidemiological approach to the aetiology of hypertension. J Hypertens 1984;2:323-328.
7. Clarke WR, Schrott HG, Leaverton PE, et al: Tracking of blood lipids and blood pressure in school age children: the Muscatine Study. Circulation 1978;58:626634.
8. Voors AW, Webber LS, Berenson GS: Time course studies of blood pressure in children-the Bogalusa Heart Study. Am J Epidemiol 1979;109:320-334.
9. Webber LS, Cresanta JL, Voors AW, et al: Tracking of cardiovascular disease risk factor variables in school age children. J Chronic Dis 1983;36:647-660.
10. Shear CL, Burke GL, Freedman DS, et al: Value of childhood blood pressure measurements and family history in predicting future blood pressure status: results from 8 years of follow-up in the bogalusa Heart Study. Pediatrics 1986;77:862-869.
11. Rosner B, Hennekens CH, Kass EH, et al: Age-specific correlation analysis of longitudinal blood pressure data. Am J Epidemiol 1977;106:306-313.
12. Zinner SH, Margolius HS, Rosner B, et al: Stability of blood pressure rank and urinary kallikrein concentration in childhood: an eight-year follow-up. Circulation 1978;58:908-915.
13. Kuller LH, Crook M, Aimes MJ: Dormont high school blood pressure study. Hypertension 1980;4:109-116.
14. Hoffman A, Valkenburg HH, Maas J, et al: The natural history of blood pressure in childhood. Int J Epidemiol 1985;14:91-96.
15. McCue CM, Miller WW, Mauck HP, Jr., et al: Adolescent blood pressure in Richmond, Virginia schools. VA Med 1979;106:210-220.
16. Levine RS, Hennekens CH, Klein B: A longitudinal evaluation of blood pressure in childhood. Am J Public Health 1979;69:175-177.
17. Hait HI, Lemshow S, Rosenman KD: A longitudinal study of blood pressure in a natural survey of children. Am J Public Health 1982;72:1285-1287.
18. Michels VV: Tracking and prediction of blood pressure in children. Mayo Clin Proc 1987;62;875-881.
19. Berenson GS, McMahan CA, Voors AW, et al: Cardiovascular risk factors in children, in Andrews C, Hester HE: The Early Natural History of Atherosclerosis and Essential Hypertension. New York, Oxford University Press, 1980, pp 1-450.
20. SAS Institute Inc.: SAS/STAT User's Guide, Version 6, 4th ed. Cary, NC, SAS Institute Inc., 1989.
21. Leitschuj ML, Cupples LA, Gagnon D, Chobanian A: High normal blood pressure progression to hypertension in the Framingham Study. Hypertension 1991;17: 22-27.
22. National Heart, Lung and Blood Institute's Task Force on Blood Pressure Control in Children: Report of the Task Force on Blood Pressure Control in Children. Pediatrics 1977;59:797-807.
23. Berenson GS, Voors AW, Webber LS, Frerichs RR: Blood pressure in children and its interpretation. Task Force Report (letter). Pediatrics 1978;61:333.
24. National Heart, Lung, and Blood Institute's Task Force on Blood Pressure Control in Children: Report of the Second Task Force on Blood Pressure Control in Children. Pediatrics 1987;79:1-25.
25. Shear CL, Burke GS, Freedman DS, et al: Designation of children with high blood pressure-considerations on percentile cutpoints and subsequent high blood pressure: the Bogalusa Heart Study. Am J Epidemiol 1987;125:73-84.
26. Lauer RM, Clarke WR, Beaglehole R: Level, trend and variability of blood pressure during childhood: the Muscatine Study. Circulation 1984;69:242-249.
27. Webber LS, Cresanta JL, Voors AW, Berenson GS: Tracking of cardiovascular disease risk factor variables in school-age children. J Chronic Dis 1983;36:647-660.
28. Harlan WE, Cornoni-Huntley J, Leaverton PE: Blood pressure in childhood: the National Health Examination Survey. Hypertension 1979;1:559-565.
29. Yong LC, Kuller LH, Rutan G, Bunker C: Longitudinal study of blood pressure: changes and determinants from adolescence to middle age. The Dormont high school follow-up study, 1957-1963 to 1989-1990. Am J Epidemiol 1993;138:973-983.
30. Berenson GS (ed): Causation of Cardiovascular Risk Factors in Children: Perspectives on Cardiovascular Risk in Early Life. New York, Raven Press, 1986, p 408.
31. Folkow B: The Fourth Volhard Lecture. Cardiovascular structured adaptation: its role in the initiation and maintenance of primary hypertension. Clin Sci Mol Med 1978;8(suppl):3S-22S.
32. Tracy RE, Berenson GS, Cueto-Garcia L, et al: Nephrosclerosis and aortic atherosclerosis from age 6 to 70 years in the United States and Mexico. Virchows Arch [A] Pathol Anat 1992;420:479-488.
33. Tracy RE, Newman WP, III, Wattigney WA, et al: Histologic features of atherosclerosis and hypertension from autopsies of young individuals in a defined geographic population. The Bogalusa Heart Study. Atherosclerosis (in press).
34. Schieken RM, Clarke WR, Lauer RM: Left ventricular hypertrophy in children with blood pressure in the upper quintile of the distribution: the Muscatine Study. Hypertension 1981;3:669-675.
35. Culpepper RA, III, Sodt PC, Messerli FH, et al: Cardiac status in juvenile borderline hypertension. Ann Intern Med 1983;98:1-7.
36. Schieken RM: Measurement of left ventricular wall mass in pediatric populations. Hypertension 1987;9: 47-52.
37. Burke GL, Arcilla RA, Culpepper WS, et al: Blood pressure and echocardiographic measures in children: the Bogalusa Heart Study. Circulation 1987;75:106114.
38. Soto LF, Kikuchi DA, Arcilla RA, et al: Echocardiographic functions and blood pressure levels in children and young adults from a biracial population: the Bogalusa Heart Study. Am J Med Sci 1989;296:271279.
39. Johnson GL, Kotchen JM, McKean HE, et al: Blood pressure related echocardiographic changes in adolescents and young adults. Am Heart J 1983;105:113-118.
40. Riley WA, Freedman DS, Higgs NA, et al: Decreased arterial elasticity associated with cardiovascular dis-
ease risk factors in the young. Arteriosclerosis 1986;6: 378-386.
41. Schieken RM, Moskowitz WB, Bodurtha J, et al: Aortic stiffness: a new Doppler echocardiographic measure predictive of systolic blood pressure in children. J Am Coll Cardiol 1988;11:1297-1300.
42. Jiang $X$, Srinivasan SR, Radhakrishnamurthy B, et al: Microalbuminuria in young black adults related to blood pressure in a biracial (black-white) population: the Bogalusa Heart Study. Am J Hypertens 1994;7: 794-800.
43. Shear CL, Webber LS, Freedman DS, et al: The relationship between parental history of vascular disease and cardiovascular disease risk factors in children: the Bogalusa Heart Study. Am J Epidemiol 1985;122:762771.
44. Blonde CV, Webber LS, Foster TA, Berenson GS: Parental history and cardiovascular disease risk factor variables in children. Prev Med 1981;10:25-37.
45. Clarke WR, Schrott HG, Burns TL, et al: Aggregation of blood pressure in the families of children with labile high systolic blood pressure: the Muscatine Study. Am J Epidemiol 1986;123:67-80.
46. Munger RG, Prineas RJ, Gomez-Marin O: Persistent elevation of blood pressure among children with a family history of hypertension: the Minneapolis Children's Blood Pressure Study. J Hypertens 1988;6:647653.
47. Bao W, Srinivasan SR, Wattigney WA, Berenson GS: The relation of parental cardiovascular disease to risk factors in children and young adults: the Bogalusa Heart Study. Circulation 1995;91:365-371.
48. Palatini P, Mormino P, Canali C, et al: Factors affecting ambulatory blood pressure reproducibility. Results of the HARVEST Trial. Hypertension 1994;23: 211-216.

[^0]:    Received October 24, 1994. Accepted February 14, 1995.
    From the Tulane National Center for Cardiovascular Health, Tulane School of Public Health and Tropical Medicine, New Orleans, Louisiana.

    This study was supported by grant HL38844 from the National Heart, Lung, and Blood Institute of the U.S. Public Health Service. Address correspondence and reprint requests to Gerald S. Berenson, MD, Tulane Center for Cardiovascular Health, Tulane School of Public Health and Tropical Medicine, 1501 Canal Street, 14th Floor, New Orleans, LA 70112-2824.

[^1]:    Values are regression coefficients (standardized regression coefficients).
    $* \mathrm{P} \leq .01$; others: $\mathrm{P} \leq .0001$.
    tIndependent variables included age, race, and sex, baseline diastolic blood pressure (BP), weight (Wt), height (Ht), and body mass index (BMI), as well as changes from baseline to follow-up in weight, height, and BMI. Variables entered the model at $\mathrm{P} \leq .05$. Baseline $=5$ to 17 years of age; follow-up $=20$ to 31 years of age .

[^2]:    Values are odds ratios, comparing high (75th percentile) v low (25th percentile) values.
    ${ }^{*} \mathrm{P} \leq .05 ; \not \mathrm{P} \leq .01 ; \S \mathrm{P} \leq .001 ;{ }^{\mathrm{H}} \mathrm{P} \leq .0001$, others $\mathrm{P} \leq .07$.
    HIndependent variables included age, race, sex, baseline systolic and diastolic blood pressure (BP), baseline body mass index (BMI), and change of BMI from baseline to follow-up. Variables entered the stepwise logistic regression model at $\mathrm{P} \leq .15$. Baseline $=5$ to 17 years of age; follow-up $=20$ to 31 years of age.

