Recent publications on blood pressure (BP) in children and adolescents report evidence that the prevalence of childhood hypertension is increasing,1,2 and the increasing rates of hypertension are largely attributable to the childhood obesity epidemic. The current estimates on the prevalence of childhood hypertension, based on repeated BP measurements ≥95th percentile (for sex, age, and height), are 3.2% to 3.6%.3,4 The prevalence of prehypertension, using the same method of repeated BP measurements, is 3.4% throughout childhood,3 with even higher rates among adolescents.4 These reports and others consistently document a significant effect of obesity on BP level throughout childhood. BP levels that are in the prehypertension range as well as in the hypertension range are considered high-risk BP levels. The combined child high BP risk of hypertension plus prehypertension now approaches 7%, a prevalence that ranks high BP as a leading childhood health issue.

The report in this issue by Lurbe et al5 provides a novel view of the childhood obesity–BP relationship. Because of BP variability in the young, BP status is best characterized by multiple BP measurements. These investigators used ambulatory BP monitoring to achieve a more rigorous ascertainment of the BP phenotype in a total of 422 children 10 to 18 years of age. Children with clinical hypertension were excluded, and all participants were considered healthy. The children were then stratified to normal weight and obese groups and then subgrouped by normal birth weight and low birth weight. Developing ambulatory BP monitoring data on 422 healthy children is a substantial achievement. With this body of data, the investigators were able to examine the effect of 2 leading BP determinants of childhood BP: obesity and low birth weight. Their results demonstrate the advantage of ambulatory BP monitoring over office BP in defining the BP phenotype in late childhood and adolescence. The ambulatory BP monitoring data clearly identify a significant effect of low birth weight on BP in both nonobese and obese children. This is an important finding because most of the reports on the relationship of birth weight with later BP have been confounded by childhood or adult obesity. Systolic BP from both office and ambulatory BP monitoring measurements were highest in the obese low birth weight group. Although they did not identify an interaction of low birth weight with obesity on BP, their data do demonstrate that the effects of low birth weight and childhood obesity have a modifying effect on BP.

The other component of this investigation is even more interesting. Recent reports from clinical investigations in the young provide evidence that high BP in the young is not just a risk factor for future hypertension. Detectable markers of vascular injury are not uncommon in children with high BP. Studies in children with high BP report left ventricular hypertrophy, increased carotid intimal thickness, and even some clues on alterations in brain function.6 In their sample of children, Lurbe et al investigated functional vascular change by measuring aortic-derived parameters from a peripheral radial arterial waveform with the SphygmoCor device (AtCor Medical).

In a previous study, using the same methodology, on younger children, these investigators reported a higher augmentation index (AI) not only in low birth weight children (birth weight <2500 g) but also in the next birth weight stratum (2500 to 3000 g) compared with children with birth weight >3000 g.7 The incidence of low birth weight for gestational age is ~5%, when excluding premature infants who have a birth weight that is appropriate for gestational age. The concept that low birth weight has a substantial impact on the population prevalence of hypertension is dampened by the fact that low birth weight occurs in <5% of newborn infants. However, the data in the previous publication by Lurbe et al suggested that the changes in AI relative to birth weight could be continuous. In this report, the investigators replicated their previous finding and demonstrated a significantly higher AI in the nonobese low birth weight children but not in the obese low birth weight children. Although these findings are of great interest, exactly what these aortic-derived measures indicate about vascular structure and function in young children is not entirely clear, especially in the context of childhood growth and development.

Several studies of wave reflection, particularly AI, in adults demonstrate that AI is a strong predictor of cardiovascular (CV) and all-cause mortality.8 As arteries stiffen, the transit time of reflected waves to reach central circulation is impacted. AI is a measure of the magnitude, or intensity, of wave reflection in central circulation. It depends on characteristics of the arterial tree, such as age, height, and pulse wave velocity, among others. Increased wave reflection augments central BP in late systole, thereby increasing cardiac workload. Many studies of wave reflection in adults have been conducted in populations with a high burden of CV disease, such as end-stage renal disease and hypertension. AI also correlates with target organ damage, such as left ventricular hypertrophy, as seen in a study of untreated hypertensive adults.9 Prevalent CV disease, particularly coronary artery disease, is associated with increased AI, especially among younger adults (<60 years of age).10

The opinions expressed in this editorial commentary are not necessarily those of the editors or of the American Heart Association.

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Other investigators have similarly found age-related changes in AI to be more prominent among younger adults (<50 years of age) compared with other measures of arterial stiffness. The findings in this investigation from Lurbe et al of increased AI in low birth weight adolescents appear to follow the trends seen in adults. Therefore, measurement of AI may provide information about early vascular changes and subsequent CV risk that could be linked with low birth weight.

Brachial (peripheral) pulse pressure is often used as a surrogate for arterial stiffness. An elevation in brachial pulse pressure is associated with increased CV morbidity and mortality in adults. Arterial stiffness, indirectly reflected by an increased pulse pressure, is thought to be largely responsible for the progressive rise in systolic BP seen in adults with advancing age. Because age is also closely tied to increased pulse pressure, it is not surprising that this was not a prominent finding in this study of adolescents and children. In a cohort of children studied by Lurbe et al, elevated office and aortic BPs were associated with obesity and low birth weight, but pulse pressure was not. However, brachial pulse pressure has some limitations and therefore is not an exact measure of arterial stiffness. In addition, brachial pulse pressure does not always reflect pressure seen in the aorta attributable to pulse pressure amplification. Pulse pressure amplification describes the phenomenon whereby there is a differential between the higher pressures seen in the periphery and lower pressures seen centrally. Age, arterial stiffness, and wave reflection, as well as other factors, are the forces driving this phenomenon.

The fact that age is a primary force driving pulse pressure amplification probably explains why peripheral pulse pressure measurement is a closer estimate of arterial stiffness and CV risk in older adults compared with younger individuals. In this current study of children and adolescents, the ratio of peripheral/aortic pulse pressure was not significantly associated with either obesity or low birth weight.

Aortic pulse pressure may be a better predictor of CV risk compared with brachial pulse pressure, particularly among groups with a high burden of CV risk. Aortic pulse pressure is influenced by age, arterial stiffness and wave reflection, among other factors, and cannot always be predicted from peripheral BP measurement. Aortic pulse pressure, but not brachial pulse pressure, was predictive of all-cause mortality in a cohort of patients with end stage renal disease. The Conduit Artery Function Evaluation (CAFE) study demonstrated that CV end points were predicted by aortic rather than brachial systolic pressure. The emerging clinical data suggest that among individuals with heightened CV risk, aortic, or central, systolic, and pulse pressure may be better predictors of CV events compared with standard brachial BP. Most of the available data on aortic pulse pressure are derived from investigations on adults. However, the data reported by Lurbe et al indicate that these alterations may be detectable at a younger age. Moreover, these observations also raise the possibility that these vascular changes are not limited to the later phases of CV disease but may extend to an earlier phase and age as well.

Current evidence from large clinical trials suggests that central aortic BP measurement provides information that adds to our knowledge of CV risk assessment. These measurements are not currently a part of routine clinical practice for use in diagnostics or clinical practice in adults, and its role in these settings continues to be a matter of debate. The report by Lurbe et al adds to our knowledge of central and peripheral BP trends in children and adolescents. Certainly, more studies in younger individuals, especially longitudinal investigations, are needed to better assess the role for central BP measurement and AI in children and adolescents.

Disclosures

None.

References

Refining the Blood Pressure Phenotype in Children: When Does Target Organ Damage Begin?
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