Clinical Implications of the Revised AAP Pediatric Hypertension Guidelines

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abstract

BACKGROUND AND OBJECTIVES: New pediatric hypertension definitions were recently published in a clinical practice guideline (CPG). We evaluated the impact of the CPG, compared with the previous guideline ("Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents"), on the prevalence of hypertension and associations with target organ damage (TOD) in high-risk youth.

METHODS: Participants (10–18 years old) undergoing an evaluation of the cardiovascular effects of obesity and type 2 diabetes mellitus in youth were studied. Blood pressure was categorized according to the 2 guidelines as normal, elevated, and hypertension (stages 1 and 2). Measures of TOD (carotid artery intima-media thickness, pulse wave velocity, left ventricular mass, and diastolic function) were obtained. Associations between blood pressure categories and TOD and the sensitivity of hypertension classification in identifying TOD were evaluated.

RESULTS: Data were available for 364 participants (65% female sex; 15.1 ± 2.1 years of age). Hypertension was identified in 8% and 13% as defined in the Fourth Report and CPG, respectively ($P = .007$). The 2 guidelines revealed similar associations with TOD; however, the CPG demonstrated improved sensitivity of TOD detection in hypertensive participants. For example, the proportion of participants with an abnormal left ventricular mass categorized as hypertensive increased from 20% to 31% as defined in the Fourth Report and CPG, respectively ($P < .001$).

CONCLUSIONS: Incorporation of the CPG increased the prevalence of pediatric hypertension in a population of high-risk youth and improved the sensitivity of TOD identification in hypertensive participants.

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Drs M. Khoury and Urbina were involved in the conception and design of the study, analysis and interpretation of data, and drafting of the manuscript; Dr P. Khoury was involved in the acquisition, analysis, and interpretation of data and revised the manuscript critically for important intellectual content; Drs Dolan and Kimball were involved in the conception and design of the study and revised the manuscript critically for important intellectual content; and all authors have approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

DOI: https://doi.org/10.1542/peds.2018-0245

Accepted for publication May 15, 2018

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

WHAT'S KNOWN ON THIS SUBJECT: Starting in youth, hypertension is associated with atherosclerosis and target organ damage, including left ventricular hypertrophy. Recently, new pediatric hypertension guidelines were published that included a number of key changes in the definition of pediatric hypertension.

WHAT THIS STUDY ADDS: The new pediatric hypertension guidelines include a significant reclassification of blood pressure categories, yielding an increased prevalence of hypertension and an improved sensitivity of target organ damage detection among those classified as hypertensive.

To cite: Khoury M, Khoury PR, Dolan LM, et al. Clinical Implications of the Revised AAP Pediatric Hypertension Guidelines. *Pediatrics*. 2018;142(2):e20180245

TABLE 1 BP Definitions in the Fourth Report and the 2017 CPG

	Fourth Report[2]	CPG[1]	
		Children Aged 1–13 y[a]	Children Aged ≥13 y
Normal BP	<90th percentile[b]	<90th percentile	<120/<80 mm Hg
Elevated BP	≥90–<95th percentile ≥120/80 in adolescents	≥90th–<95th percentile	120/<80–129/<80 mm Hg
Stage 1 hypertension	≥95th–<99th percentile + 5 mm Hg	≥95th–<95th percentile + 12 mm Hg	130/80–139/89 mm Hg
Stage 2 hypertension	≥99th percentile + 5 mm Hg	≥95th percentile + 12 mm Hg	≥140/90 mm Hg

[a] If BP values exceed criteria used for children aged ≥13 y, then those corresponding cutoffs are used.
[b] All percentiles are age, sex, and height matched. Of note, new reference tables derived from only lean subjects were included in the CPG.

Recently, a clinical practice guideline (CPG) on the screening and management of elevated blood pressure (BP) in children and adolescents was published,[1] serving as an update to the 2004 "Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents" (Fourth Report).[2] Among other changes, the CPG included new reference tables of normative BP values and new definitions of elevated BP and hypertension, including absolute BP cutoff values for adolescents ≥13 years old. These cut points were introduced to emulate the recently updated adult hypertension guidelines[3] and to simplify the process of identifying and classifying hypertension in adolescents. However, the impact of the new CPG (compared with the Fourth Report) on the prevalence of elevated BP and hypertension and associations with measures of target organ damage (TOD) has not been studied. We sought to evaluate this in a population of high-risk youth.

METHODS

Data were obtained from a study of 364 youth aged 10 to <18 years who had been enrolled in an evaluation of the cardiac and vascular effects of obesity and type 2 diabetes mellitus (T2DM) in youth. Participants were recruited from clinics at Cincinnati Children's Hospital Medical Center, community clinics, health fairs, and college campuses.[4] Three groups were recruited: participants with obesity and T2DM, participants with obesity without T2DM, and lean participants (serving as controls). Pregnant participants and those with congenital heart disease were excluded. The detailed methodology of this study has been previously published.[5] The diagnosis of T2DM was made by each participant's primary care provider. All participants with obesity underwent a 2-hour oral glucose tolerance test to rule out subclinical T2DM per previous American Diabetes Association guidelines.[6] Written informed consent was obtained from parents or guardians, and written assent was obtained from the participants. This study was approved by the Institutional Review Board at Cincinnati Children's Hospital Medical Center.

Data Collection

After an overnight fast (minimum of 10 hours), participants underwent anthropometric, BP, laboratory, echocardiography, and carotid assessments. Height, weight, and waist circumference were measured in a standardized manner, as previously published.[5] BMI was calculated. Obesity was defined as a BMI ≥95th percentile for age- and sex-specific percentiles from the Centers for Disease Control and Prevention.[7] Lean participants were defined as having a BMI <85th percentile.

BP was measured according to standards described in the Fourth Report.[2] The average of 3 BP measurements was taken by using a mercury manometer. Through the use of age-, sex-, and height-specific percentiles in the Fourth Report and the CPG, BPs were classified as normal, elevated (previously referred to as prehypertension in the Fourth Report), stage 1 hypertension, and stage 2 hypertension. BP categories, as defined in the Fourth Report and the CPG, are summarized in Table 1. Therefore, each participant's mean BP was categorized twice: once as defined in the Fourth Report and once as defined in the CPG. Fasting blood work was obtained, the details of which have been previously published.[5]

Carotid Ultrasonography

As previously reported,[5] a General Electric Vivid 7 system was employed to obtain high-resolution, B-mode ultrasonography of the carotid arteries by using a high-resolution linear array vascular ultrasound variable-frequency transducer that was centered at 7.5 MHz. Each carotid wall and segment was independently examined from continuous angles to identify the thickest carotid intima-media thickness (cIMT) by using a leading-edge–to–leading-edge technique. Three segments were imaged, and the right and left sides were averaged for the common carotid artery, the bifurcation (carotid bulb), and the internal carotid artery. The cIMT from the 3 sites were averaged to form a composite cIMT. For the purposes of this study, a composite cIMT ≥90th percentile of that measured in enrolled lean

control participants was considered abnormal.

Echocardiography

Echocardiography was performed by using a General Electric Vivid 5 or 7 (General Electric, Milwaukee, WI) or a Philips Sonos 5500 (Philips, Andover, MA) ultrasound system as previously described.[4] For each participant, a two-dimensional pulsed Doppler, tissue Doppler, and color Doppler were performed. Measurements were performed off-line by a single technician using a Cardiology Analysis System (Digisonics, Houston, TX). Left ventricular mass (LVM) was calculated by using the left ventricle end-diastolic dimension, end-diastolic posterior wall thickness, and end-diastolic septal thickness.[8] The left ventricular mass index (LVMi) was obtained by dividing the LVM by height in meters raised to 2.7 to reduce the effects of age and height.[9–11] The pediatric cutoff of LVMi ≥38.6 g/m$^{2.7}$ was used to define elevated LVM because this has been shown to correspond with the 95th percentile for LVM in children and adolescents.[9] This cutoff has been used in a number of pediatric hypertension studies.[12–14]

Diastolic function was evaluated as previously described.[4] Transmitral flow velocities were used to measure early left ventricular filling (E-wave) and late left ventricular filling. Tissue Doppler analysis was used to evaluate peak early myocardial velocity (Ea) and late myocardial velocity at both the septal and lateral annuli of the mitral valve. For the purposes of this study, tissue Doppler velocities <10th percentile for enrolled lean participants were considered abnormal. An averaged E-wave/Ea ratio ≥90th percentile for lean participants was considered abnormal.

Arterial Stiffness Measurements

Pulse wave velocity (PWV) was measured by using the SphygmoCor SCOR-PVx System (Atcor Medical, Sydney, Australia), as previously decribed.[15] Electrocardiogram-gated arterial pulse measurements at proximal (carotid) and distal (femoral) sites by using an arterial tonometer was performed.[16] Increased PWV reveals increased vascular stiffness and is associated with other cardiovascular risk factors, LVM, atherosclerotic burden, and cardiovascular mortality.[15–23] The average of 3 recordings for carotid–femoral PWV were used for analysis. The researchers in our laboratory have demonstrated excellent reproducibility with repeat measures, with coefficients of variability <7% (E.M.U., unpublished data). A PWV ≥90th percentile for lean participants was considered abnormal.

Statistical Analysis

Data were reported as means with SDs and frequencies as appropriate. Agreement in BP categorization as defined in the Fourth Report and the CPG were evaluated by using Bowker's test of symmetry. This test is used to identify significant classification differences between the 2 guidelines. Bowker's test statistic has an asymptotic χ^2 distribution under the null hypothesis of symmetry. The associations between BP categories as defined in the Fourth Report and the CPG with TOD were evaluated as continuous variables by using an analysis of covariance model, with which we produced a β coefficient and which represented the difference in the intercept from the reference level (normal BP) for a given variable at higher BP categories. The odds of abnormal measures of TOD with increasing BP categories as defined in the Fourth Report and the CPG were evaluated through the use of logistic regression analysis. Bowker's test of symmetry was used to compare the proportion of participants with TOD who were classified as hypertensive by using the Fourth Report and the CPG. This allowed for a sensitivity analysis of hypertensive categorization in the identification of TOD for each guideline. All statistical analyses were performed by using SAS version 9.4 (SAS Institute, Inc, Cary, NC).

RESULTS

Data were available for 364 participants (65% female sex; 15.1 ± 2.1 years old). Obesity was present in 213 participants (59%), and T2DM was present in 111 (30%). Demographic and clinical characteristics of the study population, stratified by BP category as defined in the Fourth Report and the CPG, are presented in Table 2. Increasing BP category, defined according to either the Fourth Report or the CPG, was significantly associated with worsening measures of cardiovascular risk and TOD (Table 2).

BP classification as defined in the CPG resulted in an increased prevalence of hypertension (13% [10% stage 1 and 3% stage 2 hypertension]) compared with classification as defined in the Fourth Report (8% [6% stage 1 and 2% stage 2] hypertension; P = .007; Table 2). Of the 75 participants with elevated BP as defined in the Fourth Report, 19 (25%) were reclassified to stage 1 hypertension as defined in the CPG. Of these 19, all were >13 years old. Six were reclassified because of their systolic BP, 8 because of their diastolic BP, and 5 because of both their systolic and diastolic BPs. Participants who were reclassified to stage 1 hypertension were significantly older (16.5 ± 0.9 vs 15.5 ± 1.7 years; P = .02) and had greater BMIs (38.8 ± 8.2 vs 33.6 ± 7.4; P = .01) and diastolic BPs (76.5 ± 8.7 vs 62.1 ± 12.2 mm Hg; P < .001)

TABLE 2 Demographic and Clinical Characteristics of the Study Population by BP Category

	Normal BP		Elevated BP		Hypertension[a]	
	Fourth Report	CPG	Fourth Report	CPG	Fourth Report	CPG
	n = 259 (71%)	*n* = 258 (71%)	*n* = 75 (21%)	*n* = 59 (16%)	*n* = 30 (8%)	*n* = 47 (13%)
Age, y	14.9 (2.2)	14.9 (2.2)	15.7 (1.6)	15.5 (1.8)	15.9 (1.9)	16.1 (1.6)
Female sex, (%)	181 (70)	181 (70)	39 (52)	35 (59)	17 (57)	21 (45)
Multiracial or people of color, (%)	156 (60)	155 (60)	41 (55)	35 (59)	20 (67)	27 (58)
Height, cm	162.4 (10.0)	162.4 (10.0)	171.2 (9.8)	169.4 (10.5)	169.0 (10.6)	171.5 (9.8)
Systolic BP, mm Hg	106.9 (8.4)	106.9 (8.4)	123.6 (5.2)	123.8 (3.0)	137.4 (10.1)	132.1 (12.2)
Diastolic BP, mm Hg	58.6 (12.0)	58.7 (12.0)	65.7 (13.0)	61.9 (12.4)	71.4 (14.9)	73.6 (13.2)
BMI	28.0 (9.5)	28.0 (9.5)	35.0 (7.9)	33.7 (7.3)	41.8 (7.9)	40.7 (8.2)
Obesity, (%)	122 (47)	121 (47)	62 (83)	47 (80)	29 (97)	45 (96)
T2DM, (%)	49 (19)	48 (19)	41 (55)	30 (51)	21 (70)	33 (70)
TOD						
LVMi, $g/m^{2.7}$	31.1 (8.4)	31.1 (8.4)	37.8 (8.4)	36.1 (8.4)	46.1 (12.1)	43.6 (11.3)
LVMi ≥38.6 g/m^2, (%)	46 (18)	46 (18)	30 (41)	20 (35)	20 (69)	30 (67)
cIMT, mm[b]	0.48 (0.082)	0.48 (0.082)	0.52 (0.085)	0.52 (0.081)	0.51 (0.10)	0.52 (0.10)
cIMT ≥90th percentile	20 (8)	20 (8)	20 (27)	13 (22)	8 (28)	15 (33)
PWV, m/s	5.4 (0.8)	5.4 (0.8)	5.9 (0.8)	5.9 (0.7)	6.8 (0.9)	6.6 (0.9)
PWV ≥90th percentile	39 (16)	39 (16)	22 (32)	14 (25)	18 (72)	26 (68)
Average E-wave/Ea ratio	1.6 (0.3)	1.6 (0.3)	1.7 (0.4)	1.7 (0.4)	2.0 (0.3)	1.9 (0.4)
Average E-wave/Ea ratio ≥90th percentile	30 (18)	30 (18)	17 (31)	13 (31)	14 (64)	18 (53)
Septal Ea, cm/s	13.5 (2.2)	13.6 (2.2)	12.8 (2.6)	12.9 (2.6)	12.5 (2.6)	12.5 (2.7)
Septal Ea <10th percentile	37 (14)	36 (14)	22 (29)	15 (25)	8 (27)	16 (34)
Lateral Ea, cm/s	17.0 (3.0)	17.0 (2.9)	16.5 (2.8)	16.6 (2.8)	15.1 (3.4)	15.4 (3.2)
Lateral Ea <10th percentile	29 (11)	28 (11)	10 (13)	7 (12)	8 (27)	12 (25)

N = 364; however, some data were missing for nondemographic variables. Data are displayed as means (SD) or *n* (%) as appropriate. All variables except race revealed significant differences ($P < .01$) across BP categories for both the Fourth Report and the CPG.

[a] Stage 1 and stage 2 hypertension categories combined.

[b] cIMT recorded as a composite score (see text).

TABLE 3 Odds Ratios (95% Confidence Intervals) for Abnormal Measures of TOD in Participants With Elevated BP and Hypertension as Defined in the Fourth Report and the CPG

	Elevated BP		Hypertension (Stage 1 and 2)	
	Fourth Report	CPG	Fourth Report	CPG
LVMi ≥38.6 $g/m^{2.7}$	3.2 (1.8–5.6)	2.4 (1.3–4.5)	10.1 (4.3–23.7)	9.1 (4.5–18.2)
PWV ≥90th percentile	2.5 (1.3–4.6)	1.7 (0.9–3.4)	13.2 (5.2–33.9)	11.1 (5.2–23.9)
cIMT ≥90th percentile	4.4 (2.2–8.8)	3.3 (1.6–7.2)	4.5 (1.8–11.5)	5.9 (2.7–12.8)
Average E-wave/Ea ratio ≥90th percentile	2.0 (1.0–4.1)	2.0 (0.9–4.3)	7.8 (3.0–20.2)	5.0 (2.3–10.9)
Septal Ea <10th percentile	2.5 (1.4–4.6)	2.1 (1.1–4.2)	2.2 (0.9–5.3)	3.2 (1.6–6.4)
Lateral Ea <10th percentile	1.2 (0.6–2.6)	1.1 (0.5–2.7)	2.9 (1.2–7.1)	2.8 (1.3–6.0)

compared with those who remained in the elevated BP category. Although the finding was not significant, 68% of those who were reclassified were of male sex compared with 41% who were not reclassified ($P = .06$). Of the 23 participants with stage 1 hypertension as defined in the Fourth Report, 3 (13%) were reclassified as having stage 2 hypertension according to the CPG. No significant differences were identified between those who remained in stage 1 and those who were reclassified as having stage 2 hypertension as defined in the CPG.

Increasing BP category was associated with increased odds of an abnormal TOD measure (Table 3). The Fourth Report and the CPG revealed similar odds (with overlapping 95% confidence intervals), suggesting that the 2 guidelines produce similar associations with TOD. Moreover, β coefficients across BP categories were similar between the Fourth Report and the CPG for TOD measures (Supplemental Table 6).

Participants with abnormal measures of TOD were among those who were reclassified from elevated BP to hypertension as defined in the CPG. For example, of the 30 participants with elevated LVM categorized as elevated BP in the Fourth Report, 11 (37%) were reclassified as having hypertension according to the CPG. Only 1 participant with elevated LVM was reclassified to a lower BP category (from hypertension to elevated BP). As a result, hypertension categorization as defined in the CPG accounted for 31% of participants with increased LVM compared with 20% as defined in the Fourth Report ($P < .001$),

TABLE 4 Proportion of Participants With Abnormal TOD Measures Who Were Categorized as Hypertensive as Defined in the Fourth Report and the CPG

	Fourth Report (%)	CPG (%)	*P*[a]
LVMi ≥38.6 g/m^2 (*N* = 96)	20 (21)	30 (31)	<.001
PWV ≥90th percentile (*N* = 79)	18 (23)	26 (33)	<.001
cIMT ≥90th percentile (*N* = 48)	8 (17)	15 (31)	<.001
Average E/Ea ≥90th percentile (*N* = 61)	14 (23)	18 (30)	<.001
Septal Ea <10th percentile (*N* = 67)	8 (12)	16 (24)	<.001
Lateral Ea <10th percentile (*N* = 47)	8 (17)	12 (26)	<.001

[a] *P* values derived from χ^2 analysis.

TABLE 5 Stage 1 Hypertension BP Threshold Values as Defined in the Fourth Report and CPG

Age and Height Percentile	Male Sex		Female Sex	
	Fourth Report	CPG	Fourth Report	CPG
10 y				
5th percentile	115/77	112/76	116/77	112/75
50th percentile	119/80	116/77	119/78	116/76
95th percentile	123/82	121/78	122/80	120/76
13 y				
5th percentile	121/79	130/80	121/80	130/80
50th percentile	126/81	130/80	124/81	130/80
95th percentile	130/83	130/80	128/83	130/80
15 y				
5th percentile	126/81	130/80	124/82	130/80
50th percentile	131/83	130/80	127/83	130/80
95th percentile	135/85	130/80	131/85	130/80
17 y				
5th percentile	131/84	130/80	125/82	130/80
50th percentile	136/87	130/80	129/84	130/80
95th percentile	140/89	130/80	132/86	130/80

thus improving the sensitivity of hypertension categorization in detecting LVM. Similar increases in sensitivity were noted with the other measures of TOD (Table 4).

DISCUSSION

In the current study, the incorporation of the new pediatric CPG for hypertension resulted in an increased prevalence of participants being categorized as hypertensive (from 8% to 13%) compared with the previous guideline, the Fourth Report. The CPG and Fourth Report revealed similar strengths of association between higher BP categories and measures of TOD. Despite these similar associations, the increased prevalence of hypertension when defined according to the CPG resulted in more participants with TOD (such as increased LVM) being classified as having hypertension. Therefore, this increased the sensitivity of TOD detection among those categorized as having hypertension according to the CPG compared with the Fourth Report. Given that more involved diagnostic evaluations and more aggressive treatment approaches are recommended in those diagnosed with hypertension, the CPG may have important clinical implications for youth with increased BP.

The CPG included a number of important modifications to the BP categorization of youth.[1] For example, given the strong associations between overweight and obesity and BP,[24,25] new reference tables excluding data from youth with overweight and obesity were formulated, resulting in 1 to 4 mm Hg drops in the elevated (≥90th percentile) and hypertension (≥95th percentile; Table 5) BP thresholds for children.[26] The implications of this subtle change are that fewer children <13 years old with elevated BPs will be missed. In our study, of the 259 children with normal BP measures according to the Fourth Report, only 1 was reclassified as having elevated BP as defined in the CPG, revealing that the definition changes between the Fourth Report and the CPG likely yield only subtle changes with respect to the classification of those with more normal BPs.

The most prominent change in the CPG BP categorizations was the introduction of absolute BP thresholds for defining elevated BP, stage 1 hypertension, and stage 2 hypertension for children ≥13 years old (Table 1). The rationale for this change was to simplify the detection of hypertension in adolescents (because it is often underrecognized[27]) and incorporate the same cut points used in the new adult guidelines.[3] The adult cut points are based on hard cardiovascular outcomes,[28] in contrast to pediatric guidelines that previously included

definitions of hypertension that were based on percentiles from normative data that were not linked to events in adulthood.[1,2] These changes seem logical given that many BPs ≥95th percentile according to the Fourth Report for older and taller adolescents are at BPs well above what would be considered hypertensive for an adult (≥130/80; Table 5). In the current study, 19 (25%) participants who had elevated BP according to the Fourth Report were reclassified as having stage 1 hypertension as defined in the CPG. The diastolic BPs were significantly higher in those who were reclassified from elevated BP to stage 1 compared with those who did not. As a result of the reclassifications, the CPG revealed an increase in hypertension prevalence (stages 1 and 2) from 8% to 13%.

Increased BP is associated with TOD, including increased cIMT,[5,29] arterial stiffness,[15,30] and LVM, both in childhood[12,31,32] and adulthood.[33] Given its association with cardiovascular disease events in adults with hypertension,[34] LVM has been identified as a key measure of TOD in pediatric patients.[35] Consistent with these studies, we demonstrated strong associations between higher BP categories and TOD measures. The CPG and Fourth Report revealed similar odds of abnormal measures of TOD with increasing BP categories and similar β coefficients when the measures were evaluated as continuous variables.

Although the associations between BP categories and TOD as defined in the 2 guidelines were similar, more participants with TOD were hypertensive as defined in the CPG. For example, 37% of those with abnormal LVM with elevated BP according to the Fourth Report were reclassified as being hypertensive as defined in the CPG, resulting in 30 participants with elevated LVM in the hypertensive category compared with 20 as defined in the Fourth Report. As a result, the sensitivity of hypertension categorization in the detection of abnormal LVM increased from 20% as defined in the Fourth Report to 31% as defined in the CPG. The relatively low sensitivity observed with both guidelines is likely secondary to the high prevalence of obesity in our study population (59%) and the strong associations known to exist between obesity and LVM independent of BP.[9,12,36] For example, in a study by Falkner et al,[36] 24% of adolescents with obesity and normal BPs (<120/80) had increased LVM compared with 19% of lean adolescents with raised BP. Moreover, previous studies involving our study population revealed strong associations between LVM and BMI *z* scores.[4] In the current study, 46 of 96 participants with elevated LVM had normal BP; 44 of these participants were obese. Despite this, the finding in our study that the CPG yielded an increased prevalence of hypertension and increased the proportion of those with elevated LVM having hypertension reveals that incorporation of the CPG may improve the clinical detection of increased LVM in youth with raised BP. This finding has important potential clinical implications given that the CPG includes recommendations for echocardiography to assess for cardiac TOD at the time of consideration of pharmacologic therapy[1] because raised LVM may aid in the risk stratification of patients with hypertension. Further studies, however, are needed to truly audit the clinical impact of incorporating the CPG with respect to rates of subspecialty referral, investigations for TOD, and the use of antihypertensive medications in youth.

The main strength of this study was its novel design and objective; the clinical impact of the new CPG has not been studied to date. The availability of measures of TOD and surrogate markers of atherosclerosis are helpful in pediatric studies. Given the difficulty of evaluating hard cardiovascular end points in the pediatric population, these measures aid in assessing risk associated with pediatric hypertension.

There are a number of limitations to consider when interpreting the results of this study. First, the cross-sectional study design precludes the ability to establish causation with respect to associations between BP categories and TOD. Given the increased prevalence of obesity (59%) and T2DM (30%) in our study population compared with the general population, the findings from this study may not be generalizable to the population at large. To this end, although the prevalence of hypertension in the general population is ~2% to 4%,[27,37,38] the prevalence of hypertension in the current study was significantly higher (13% as defined in the CPG and 8% as defined in the Fourth Report). However, given the strong associations between obesity and essential hypertension, the study population may be similar to what clinicians see in hypertension clinics.[24,25] Because minimal reclassification was expected to occur at more normal BP levels (as described above), studying a higher-risk population allowed us to better evaluate the extent of reclassification that occurs with the new CPG. The study was underpowered to properly evaluate the differences in demographic and clinical characteristics between participants who were reclassified to a different BP category compared with those who were not. Although the CPG defines elevated LVM using the adult criteria (>51g/m$^{2.7}$), this cut point is >99th percentile for children and adolescents[10,39] and would have resulted in only 23 participants having increased LVM,

thus limiting our analysis. Therefore, the pediatric cut point of 38.7 g/m$^{2.7}$ was used, corresponding with the 95th percentile for LVM in children and adolescents,[9] as has been done in previous pediatric hypertension studies.[12–14] Finally, the use of arterial stiffness and diastolic dysfunction measurements for risk stratification in children and adolescents is limited by the lack of normal values across ages, sexes, and races and/or ethnicities. We therefore resorted to using percentiles of our lean population.

CONCLUSIONS

The new CPG revealed a higher prevalence of hypertension as a result of significant BP reclassification to higher BP categories. Although the Fourth Report and the CPG revealed similarly increased associations between higher BP categories and the TOD, a greater proportion of participants with TOD were categorized as hypertensive as defined in the CPG compared with the Fourth Report, thus improving the sensitivity of TOD detection in patients with hypertension. This finding, combined with the increased prevalence of hypertension with the incorporation of the CPG, reveals that the CPG may contribute to an increased detection of abnormal LVM and other measures of TOD. This, in turn, may contribute to risk stratification in clinical decision-making for youth presenting with BP concerns. Further studies, however, are required to confirm the impact of the new guidelines on the prevalence of hypertension in the general population and the clinical and economic implications with respect to the management of hypertension and the detection of TOD in the pediatric population.

ABBREVIATIONS

BP: blood pressure
cIMT: carotid intima-media thickness
CPG: clinical practice guideline
Ea: peak early myocardial velocity
E-wave: early left ventricular filling
LVM: left ventricular mass
LVMi: left ventricular mass index
PWV: pulse wave velocity
T2DM: type 2 diabetes mellitus
TOD: target organ damage

FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: Supported by the National Institutes of Health National Heart, Lung, and Blood Institute grant R01 HL076269 (CV Diseases in Adolescents with Type 2 Diabetes). Funded by the National Institutres of Health (NIH).

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

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