Received: 10 December 2018 | Revised: 18 March 2019 | Accepted: 28 March 2019

DOI: 10.1111/jch.13540

ORIGINAL PAPER

WILEY

Left ventricular cardiac geometry and ambulatory blood pressure in children

Steffi Shilly BA | Kumail Merchant MD | Pamela Singer MD | Rachel Frank RN | Shari Gurusinghe BA | Lulette Infante RN | Christine B. Sethna MD, EdM

Department of Pediatrics, Division of Pediatric Nephrology, Cohen Children's Medical Center, New Hyde Park, New York

Correspondence
Christine B. Sethna MD EdM, Division of Pediatric Nephrology, 269-01 76th Avenue, New Hyde Park, NY 11040.
Email: csethna@northwell.edu

Abstract

Limited information is available regarding the relationship between ambulatory blood pressure monitoring (ABPM) and cardiac geometry in hypertensive children. ABPM and 2D-echocardiography were retrospectively reviewed in children and adolescents <21 years old with primary hypertension. A total of 119 participants (median age 15.0 [IQR 12, 16] years) with hypertension were included. Left ventricular hypertrophy was diagnosed in 39.5% of participants. Normal geometry was found in 47.1%, concentric remodeling (CR) in 13.4%, concentric hypertrophy (CH) in 15.1%, and eccentric hypertrophy (EH) in 24.4% of children. After adjustment for age, sex, and body mass index z-score, awake systolic blood pressure (BP) index (BPi) (OR 1.07, 95% CI: 1.001-1.14, $P = 0.045$), awake diastolic BPi (OR 1.04, 95% CI: 1.00-1.09, $P = 0.048$), awake systolic BP load (OR 1.02, 95% CI: 1.000-1.04, $P = 0.047$), and sleep systolic BP load (OR 1.02, 95% CI: 1.001-1.04, $P = 0.03$) were directly associated with CH. No ABPM parameters were significant predictors of EH. In conclusion, ABPM parameters were found to be independent predictors of cardiac geometry, specifically CH.

1 | INTRODUCTION

Left ventricular hypertrophy (LVH) is the most commonly encountered end organ effect of hypertension among children and adolescents.[1,2] Hemodynamic overload in hypertension leads to remodeling of the left ventricle (LV) and altered cardiac geometry. Based upon LV mass (LVM) and relative wall thickness (RWT), which is the ratio of LV wall thickness to internal cavity diameter, there are four geometric patterns of LV remodeling. Cardiac geometry is categorized as normal, concentric remodeling (CR), concentric hypertrophy (CH), and eccentric hypertrophy (EH).[3] CH is characterized by increased LVM and LV wall thickness resulting from increased hemodynamic LV filling pressures while increased preload volume leads to dilation of the ventricle and an EH pattern. CH and EH have been shown to be independent predictors of cardiovascular disease and mortality in adults[3-5] and are present in 11%-30% of children with hypertension.[1,6-8]

Ambulatory blood pressure monitoring (ABPM) has been demonstrated to be associated with LVM and RWT in adults.[9-12] Studies have found that ABPM values are more accurate and strongly correlated with LVH when compared to clinic blood pressure.[11] Evidence suggests that blood pressure parameters measured from 24-hour ABPM may serve as predictors of specific cardiac geometries.[13-15] One pediatric study demonstrated differences in ABPM parameters by cardiac geometry[16]; however, limited epidemiological evidence is available regarding the determinants of LV geometry in the pediatric population. The main objective of the current study was to examine the association of ABPM metrics to specific patterns of LV geometry in children and adolescents with primary hypertension.

2 | MATERIALS AND METHODS

2.1 | Study population

Children and adolescents <21 years who were evaluated for hypertension from 2013-2018 in the pediatric nephrology clinic at a single tertiary center were examined in this retrospective chart review. Data were retrieved from ABPM and 2D-echocardiograms that were performed as part of the patients' standard clinical evaluation for

elevated blood pressure. Participants with a confirmed diagnosis of primary hypertension whose diagnostic ABPM and echocardiogram were completed within a 3-month period were included in this study. Participants whose echocardiograms were completed outside of the institution were not included since LV measurements were not available. Participants with secondary hypertension or a known history of hypertension treated with anti-hypertensive medication were excluded from the study. Among the 634 children with ABPM that were screened, 119 participants met the inclusion criteria and were included in the study. This study protocol was approved by the Institutional Review Board of Northwell Health.

2.2 | Ambulatory blood pressure monitoring

Ambulatory blood pressure monitoring was conducted over a 24-hour time period using the Spacelabs Model 90201-IQ (Spacelabs Medical). The device automatically obtained blood pressure measurements in the non-dominant arm at 20-minute intervals while awake and at 30-minute intervals while sleeping. Average values were calculated for the 24-hour time period, awake period, and sleep period. Systolic (SBP) and diastolic (DBP) mean blood pressure, blood pressure load, and ambulatory blood pressure index (BPi) were computed for all time periods. Ambulatory BPi was calculated for each participant by dividing the blood pressure by the appropriate sex and height-specific 95th percentile. Nocturnal non-dipping was defined as <10% percentage decline in SBP and DBP levels between awake and sleep time periods. Blood pressure loads were determined by calculating the percentage of readings that exceeded the sex and height-specific 95th percentile. ABPM readings were interpreted by the primary investigator, and participants were diagnosed as hypertensive from their ABPM data using the classification criteria from the American Heart Association.[13,17]

2.3 | Cardiac geometry

In accordance with the American Society of Echocardiography pediatric guidelines,[18] two-dimensional guided M-mode echocardiograms were obtained from all hypertensive children. A pediatric cardiology ultrasound technician performed the echocardiograms and one of our 11 pediatric cardiologists analyzed the study. Intraclass correlation coefficients for readings in our echo lab range from 0.90 to 0.96.[19]

Participants were assigned a diagnosis of LVH using the LVMI >95th percentile presented in the reference data from Khoury et al[20] LVMI was calculated for each echocardiogram using the M-mode measurements of interventricular septum (IVS), LV internal diameter (LVID), and LV posterior wall (LVPW). The Devereux formula for LV mass was used (LV mass = 0.8(1.04[(IVS + LVID + LVPQ)3-LVID3] + 0.6))[21] and indexed to height$^{2.7}$.[22] RWT was calculated to assess LV geometry using the formula: (IVS + LVPW)/LVID. RWT was considered abnormal if it was >0.42.[16] LV geometry was defined as follows: normal when RWT <0.42 and no LVH present, CR when RWT >0.42 and no LVH present, CH when RWT >0.42 and LVH present, and EH when RWT <0.42 and LVH present. Participants were categorized into these four groups based upon LVMI and RWT.[16,23]

2.4 | Demographic and clinical variables

Medical records were abstracted for demographic information, height, weight, and medical history. BMI z-scores and percentiles were calculated from CDC reference values.[24] BMI ≥95th percentile was categorized as obese. Clinic blood pressure was measured using an aneroid sphygmomanometer on the same day as the ABPM study. Systolic and diastolic blood pressures were transformed to BP z-scores.[23]

2.5 | Statistical analyses

Descriptive statistics were used to characterize outcome measurements in participants. Differences in demographic and ABPM measures between children with LVH and no LVH were compared using Student's *t* test and chi-square test. Comparisons among cardiac geometry groups were made using one-way ANOVA and chi-square with post hoc analysis using Tukey tests and Bonferroni tests. Multiple logistic regression models were used to test the associations of ABPM variables with cardiac geometry (CH and EH) after adjusting for sex, age, and BMI z-score. Using a two-tailed tests of hypotheses, a *P*-value < 0.05 was the criterion for statistical significance. Statistical analyses were performed using the SPSS 25.0 (IBM Inc) statistical package.

3 | RESULTS

Among the 119 children included in the study, the mean age was 14.0 ± 3.3 years (median 15.0 [IQR 12, 16] years), 84 (70.6%) were male, 35 (29.4%) were Black, and 33 (27.7%) were obese. The mean LVMI for the patient population was 38.9 ± 11.5 g/m$^{2.7}$ (range 11-77 g/m$^{2.7}$). LVH was present in 39.5% (N = 47) of children. Comparisons of children with LVH to those without LVH are presented in Table 1. Age and BMI z-score were greater in children with LVH compared to those without LVH ($P < 0.05$). All other demographic, clinical, and ABPM parameters were not significantly different between those with and without LVH (all $P > 0.05$).

The distribution of cardiac geometry was as follows: normal cardiac geometry was diagnosed in 47.1%, CR in 13.4%, CH in 15.1%, and EH in 24.4% of participants. Demographics and ABPM measures by cardiac geometry are summarized in Table 2. LVMI was significantly different across the four cardiac geometry categories ($P < 0.0001$). In post hoc analysis, those with CH had significantly greater LVMI compared with EH, CR and normal geometry groups ($P < 0.05$). The EH group had significantly greater LVMI than the CR and normal groups ($P < 0.05$). BMI z-score and obesity were also significantly different among groups; the EH group had greater BMI z-scores than the other groups, and CH had a greater proportion

TABLE 1 Comparison of children with and without left ventricular hypertrophy

Mean ± SD or N (%)	Overall	No LVH	LVH	*P*-value
N = 119	56 (47.1)	18 (15)	16 (13.4)	
Age (y)	14 ± 3.3	13.4 ± 3.4	15 ± 2.9	0.01
Male	84 (70.6)	48 (66.7)	36 (76.6)	0.25
Black race	35 (29.4)	20 (27.8)	15 (31.9)	0.63
BMI z-score	1.24 ± 1.0	0.97 ± 1.1	1.64 ± 0.91	0.001
Obese	33 (28.2)	13 (18.6)	20 (42.6)	0.02
LVMI ($g/m^{2.7}$)	38.9 ± 11.5	31.9 ± 6.6	49.6 ± 8.9	<0.0001
Clinic SBPi	1.05 ± 0.27	1.08 ± 0.27	1.03 ± 0.27	0.3
Clinic DBPi	0.86 ± 0.25	0.85 ± 0.25	0.87 ± 0.25	0.72
Awake SBPi	0.97 ± 0.09	0.96 ± 0.09	0.98 ± 0.09	0.25
Awake DBPi	0.93 ± 0.14	0.92 ± 0.15	0.95 ± 0.13	0.24
Sleep SBPi	1.02 ± 0.14	1.03 ± 0.16	1.03 ± 0.11	0.93
Sleep DBPi	1.01. ± 0.17	1.0 ± 0.17	1.03 ± 0.19	0.32
Awake SBP load (%)	47.3 ± 32.4	37.7 ± 34.5	48 ± 28.1	0.09
Awake DBP load (%)	31.4 ± 29.9	28.1 ± 30.2	36.5 ± 29	0.13
Sleep SBP load (%)	48.9 ± 33.4	44.1 ± 33	56.3 ± 32.9	0.05
Sleep DBP load (%)	39.9 ± 34	36 ± 32.6	45.7 ± 35.5	0.13
Systolic non-dipper	50 (42.4)	28 (38.9)	22 (47.8)	0.34
Diastolic non-dipper	58 (49.2)	32 (44.4)	26 (56.5)	0.2

Abbreviations: BMI, body mass index; CH, concentric hypertrophy; CR, concentric remodeling; DBP, diastolic blood pressure; DBPi, diastolic blood pressure index; EH, eccentric hypertrophy; LVMI, left ventricular mass index; RAAS, renin aldosterone angiotensin system; SBP, systolic blood pressure; SBPi, systolic blood pressure index.

of obesity compared to the other groups ($P < 0.05$). There were no significant differences in ABPM parameters by cardiac geometry in bivariate analysis.

The results of multiple logistic regression analysis adjusted for age, sex, and BMI z-score are reported in Table 3. Clinic SBPi and DBPi were not associated with CH or EH. Awake systolic blood pressure (BP) index (BPi) (OR 1.07, 95% CI: 1.001-1.14, $P = 0.045$), awake diastolic BPi (OR 1.04, 95% CI: 1.00-1.09, $P = 0.048$), awake systolic BP load (OR 1.02, 95% CI: 1.000-1.04, $P = 0.047$), and sleep systolic BP load (OR 1.02, 95% CI: 1.001-1.04, $P = 0.03$) were directly associated with CH. No ABPM parameters were significant predictors of EH.

4 | DISCUSSION

Left ventricular hypertrophy is highly prevalent in children diagnosed with hypertension.[1] Cardiac remodeling associated with LVH occurs as a result of adaptation to the hemodynamic overload from hypertension.[25] The superiority of ABPM in predicting LV changes has been proven in earlier findings.[9-12] Previous epidemiologic studies have demonstrated the relationship between ABPM and cardiac geometry in adults[1,13-15]; however, our study differs by exploring whether ABPM determinants are associated with specific cardiac geometries in children.

Our results indicated that awake SBPi and DBPi were significantly associated with CH. Additionally, awake and sleep SBP load were found to be significant predictors of CH. No ABPM parameters demonstrated to be predictive of EH.

Similar to our findings, past adult studies have established a relationship between ABPM parameters and CH. A study of 165 hypertensive adults demonstrated that subjects with CH had higher systolic blood pressures.[25] Another adult study conducted by Devereux et al[26] observed both high daytime systolic and diastolic ambulatory pressures in patients with CH. To our knowledge, the finding that awake SBPi and DBPi are predictors of CH has not been previously reported in the pediatric population. Earlier adult studies have also established a relationship between SBP load and CH. Cunha and colleagues reported that SBP load was greater in CH compared to patients with CR or normal geometry.[14] Tsioufis et al[15] had also observed higher ambulatory SBP load related to CH in a study which investigated 335 hypertensive adults.

Ambulatory blood pressure monitoring parameters were not found to be independent predictors of EH in this present study. Our results are corroborated by work from Matteucci and colleagues, in which the presence of a significant relationship between ABPM

TABLE 2 Demographic and ambulatory blood pressure measures in children with hypertension by cardiac geometry

Mean ± SD or N (%)	Normal	CR	CH	EH	*P*-value
N = 119	56 (47.1)	16 (13.4)	18 (15.1)	29 (24.4)	
Age (y)	13.2 ± 3.7	14.4 ± 2.6	15.7 ± 1.8	14.5 ± 3.3	0.03
Male	35 (62.5)	13 (81.3)	13 (72.2)	23 (79.3)	0.29
Black race	13 (23.2)	7 (43.8)	8 (44.4)	7 (24.1)	0.17
BMI z-score	1.03 ± 1.1	0.79 ± 0.99	1.4 ± 1.1	1.77 ± 0.76	0.004
Obese	10 (18.5)	3 (18.8)	8 (44.4)	12 (41.4)	0.04
LVMI ($g/m^{2.7}$)	30.6 ± 6.1	36.4 ± 6.6	55.9 ± 9.7	45.7 ± 5.	<0.0001
Clinic SBPi	1.0 2 ± 0.27	1.04 ± 0.29	1.07 ± 0.3	1.08 ± 0.25	0.76
Clinic DBPi	0.85 ± 0.27	0.85 ± 0.27	0.87 ± 0.23	0.87 ± 0.23	0.99
Awake SBPi	0.97 ± 0.08	0.96 ± 0.12	1.0 ± 0.11	0.97 ± 0.06	0.28
Awake DBPi	0.92 ± 0.14	0.93 ± 0.17	0.99 ± 0.14	0.92 ± 0.11	0.25
Sleep SBPi	1.03 ± 0.17	1.01 ± 0.13	1.06 ± 0.11	1.0 ± 0.11	0.66
Sleep DBPi	1.0 ± 0.16	1.0 ± 0.2	1.06 ± 0.23	1.01 ± 0.16	0.65
Awake SBP load (%)	36.5 ± 33.5	41.8 ± 38.4	58.3 ± 30.1	41.5 ± 25.3	0.1
Awake DBP load (%)	27.3 ± 29.3	30.8 ± 34.1	43.9 ± 32.1	32 ± 26.9	0.24
Sleep SBP load (%)	44.3 ± 32.6	43.4 ± 35.4	65.4 ± 29.3	50.6 ± 34.2	0.11
Sleep DBP load (%)	36. ± 32.4	35.8 ± 34.4	48.6 ± 38.3	44 ± 34.2	0.47
Systolic non-dipper	21 (37.5)	7 (43.8)	9 (52.9)	13 (44.8)	0.7
Diastolic non-dipper	26 (46.4)	6 (37.5)	9 (52.9)	17 (58.6)	0.54

Abbreviations: BMI, body mass index; CH, concentric hypertrophy; CR, concentric remodeling; DBP, diastolic blood pressure; DBPi, diastolic blood pressure index; EH, eccentric hypertrophy; LVMI, left ventricular mass index; RAAS, renin aldosterone angiotensin system; SBP, systolic blood pressure; SBPi, systolic blood pressure index.

parameters and EH in adults with hypertension was not detected.[27] They reasoned that the presence of subjects with either obesity or low normal blood pressure may have contributed to the masking of any possible relationships.[27] Moreover, hypertension may not be solely responsible for the development of LVH.[28] Rather, LVH may be a complex condition that is caused by a variety of risk factors, including genetics[29-31] and metabolic syndrome.[32,33] In contrast to our results, a study of adults with hypertension by Ganau et al[25] reported that DBP was associated with EH. The participants with EH in Ganau's study displayed higher stroke volumes and larger spherical left ventricular cavities.[25] Mechanistically, this supports the theory that increased intravascular volume causes strain in diastolic function, forming eccentric hypertrophy.[1]

Interestingly, although we found relationships between ABPM and cardiac geometry on multivariate analysis, we did not find differences in individual ABPM parameters among the cardiac geometry groups on bivariate analysis. This may have been due to the high proportion of obese children in the cohort. Contrary to our results, a previous pediatric study, using older criteria for LVH,[16] found significant differences in various 24-hour ABPM parameters among EH and CH groups. Mean 24-hour SBP was greater in EH compared to the normal geometry group, and mean 24 hours DBP and diastolic BP load were greater in EH compared to CH and normal geometry groups. This study did not examine daytime or night-time ABPM parameters, nor did it examine ABPM in multivariate analysis.

The main limitation of this analysis is the single-center, retrospective nature of the study. The small sample size may introduce selection bias therefore, an increase in the sample size would allow for better generalization and accuracy of results. Additionally, the only normative values that are available for interpretation of ABPM are based on a non-obese, Caucasian European cohort, which is not representative of the United States population of children and adolescents and not representative of the population in this study.[17] Furthermore, LV geometry is categorized based on ratios of individual measurements that are keen to measurement error. Thus, miscategorization can happen if an error occurs. Despite the inherent limitations present in this study, we believe that the observations of the study are significant and can be generalizable to other populations.

TABLE 3 Multiple regression models of blood pressure measures to predict cardiac geometry

	Cardiac geometry			
	CH		EH	
	OR (95% CI)[a]	*P*-value	OR (95% CI)[a]	*P*-value
Clinic SBPi	1.37 (0.16-11.6)	0.77	1.69 (0.23-12.5)	0.61
Clinic DBPi	1.02 (0.12-9.1)	0.99	1.41 (0.17-11.6)	0.75
Awake SBPi	1.07 (1.001-1.14)	0.045	1.01 (0.96-1.07)	0.61
Awake DBPi	1.04 (1.00-1.09)	0.048	1.01 (0.98-1.05)	0.59
Sleep SBPi	1.02 (0.99-1.06)	0.18	0.98 (0.95-1.02)	0.36
Sleep DBPi	1.02 (0.98-1.05)	0.35	1.008 (0.98-1.04)	0.57
Awake SBP load	1.02 (1.000-1.04)	0.047	1.002 (0.99-1.02)	0.84
Awake DBP load	1.01 (0.99-1.03)	0.11	1.004 (0.99-1.02)	0.65
Sleep SBP load	1.02 (1.001-1.04)	0.03	1.006 (0.99-1.02)	0.44
Sleep DBP load	1.007 (0.99-1.02)	0.37	1.01 (0.98-1.03)	0.11
Systolic non-dipper	1.78 (0.6- 5.2)	0.3	1.4 (0.56-3.6)	0.47
Diastolic non-dipper	1.3 (0.44-3.8)	0.64	1.9 (0.71-4.9)	0.21

Abbreviations: ABPM, ambulatory blood pressure monitor; CH, concentric hypertrophy; DBP, diastolic blood pressure; DBPi, diastolic blood pressure index; EH, eccentric hypertrophy; OR, odds ratio; SBP, systolic blood pressure; SBPi, systolic blood pressure index.
[a]Models adjusted for age, sex, and body mass index z-score. BP indices multiplied by 100.

5 | CONCLUSION

This study retrospectively investigated whether various parameters measured by ABPM can serve as predictors of cardiac geometry. We found that awake SBPi and awake DBPi were significantly associated with CH. Additionally, awake SBP load and sleep SBP load were found to be significant predictors of CH. No ABPM parameters were predictive of EH. Given the increasing prevalence of LVH in the pediatric population,[34] it is important to effectively identify abnormal LV geometry. Prevention and treatment of abnormal cardiac geometry via blood pressure control are potential modifiable factors that may lead to a decrease in the development of cardiovascular disease among patients with hypertension.[35] Future studies should aim to further investigate the relationship between ABPM metrics and cardiac geometry.

CONFLICT OF INTEREST

None.

AUTHOR'S CONTRIBUTIONS

Research idea and study design: CS, SS; data acquisition: SS, KM, SG; data analysis/interpretation: CS, KM, SS, SG, PS, LI, RF; statistical analysis: SS, CS; supervision or mentorship: CS. Each author contributed important intellectual content during manuscript drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

ORCID

Christine B. Sethna https://orcid.org/0000-0002-7666-5792

REFERENCES

1. Richey PA, Disessa TG, Somes GW, Alpert BS, Jones DP. Left ventricular geometry in children and adolescents with primary hypertension. *Am J Hypertens*. 2010;23:24-29.
2. National High Blood Pressure Education Program Working Group on High Blood Pressure in C, Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004;114:555-576.
3. Krumholz HM, Larson M, Levy D. Prognosis of left ventricular geometric patterns in the Framingham Heart Study. *J Am Coll Cardiol*. 1995;25:879-884.
4. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. *Ann Intern Med*. 1991;114:345-352.
5. Verdecchia P, Schillaci G, Borgioni C, et al. Adverse prognostic significance of concentric remodeling of the left ventricle in hypertensive patients with normal left ventricular mass. *J Am Coll Cardiol*. 1995;25:871-878.
6. Pruette CS, Fivush BA, Flynn JT, Brady TM. Effects of obesity and race on left ventricular geometry in hypertensive children. *Pediatr Nephrol*. 2013;28:2015-2022.
7. Pieruzzi F, Antolini L, Salerno FR, et al. The role of blood pressure, body weight and fat distribution on left ventricular mass, diastolic function and cardiac geometry in children. *J Hypertens*. 2015;33:1182-1192.
8. Riehle TJ, Mahle WT, Parks WJ, Sallee D 3rd, Fyfe DA. Real-time three-dimensional echocardiographic acquisition and quantification of left ventricular indices in children and young adults with congenital heart disease: comparison with magnetic resonance imaging. *J Am Soc Echocardiogr*. 2008;21:78-83.

9. Verdecchia P, Porcellati C, Schillaci G, et al. Ambulatory blood pressure. An independent predictor of prognosis in essential hypertension. *Hypertension*. 1994;24:793-801.
10. White WB. Ambulatory blood pressure and target organ involvement in hypertension. *Clin Invest Med*. 1991;14:224-230.
11. Mancia G, Zanchetti A, Agabiti-Rosei E, et al. Ambulatory blood pressure is superior to clinic blood pressure in predicting treatment-induced regression of left ventricular hypertrophy. SAMPLE Study Group. Study on Ambulatory Monitoring of Blood Pressure and Lisinopril Evaluation. *Circulation*. 1997;95:1464-1470.
12. Verdecchia P, Porcellati C. Defining normal ambulatory blood pressure in relation to target organ damage and prognosis. *Am J Hypertens*. 1993;6:207S–210S.
13. Flynn JT, Daniels SR, Hayman LL, et al. Update: ambulatory blood pressure monitoring in children and adolescents: a scientific statement from the American Heart Association. *Hypertension*. 2014;63:1116-1135.
14. Cunha DM, da Cunha AB, Martins WA, et al. Echocardiographic assessment of the different left ventricular geometric patterns in hypertensive patients. *Arq Bras Cardiol*. 2001;76:15-28.
15. Tsioufis C, Stefanadis C, Goumas G, Pitsavos C, Toutouzas P. Relation of ambulatory blood pressure load with left ventricular geometry in untreated patients with mild-to-moderate hypertension. *J Hum Hypertens*. 1999;13:677-682.
16. Daniels SR, Loggie JM, Khoury P, Kimball TR. Left ventricular geometry and severe left ventricular hypertrophy in children and adolescents with essential hypertension. *Circulation*. 1998;97:1907-1911.
17. Wuhl E, Witte K, Soergel M, Mehls O, Schaefer F, German Working Group on Pediatric H. Distribution of 24-h ambulatory blood pressure in children: normalized reference values and role of body dimensions. *J Hypertens*. 2002;20:1995-2007.
18. Lopez L, Colan SD, Frommelt PC, et al. Recommendations for quantification methods during the performance of a pediatric echocardiogram: a report from the Pediatric Measurements Writing Group of the American Society of Echocardiography Pediatric and Congenital Heart Disease Council. *J Am Soc Echocardiogr*. 2010;23(5):465-495; quiz 576-467.
19. Mirchandani D, Bhatia J, Leisman D, et al. Concordance of measures of left-ventricular hypertrophy in pediatric hypertension. *Pediatr Cardiol*. 2014;35:622-626.
20. Khoury PR, Mitsnefes M, Daniels SR, Kimball TR. Age-specific reference intervals for indexed left ventricular mass in children. *J Am Soc Echocardiogr*. 2009;22:709-714.
21. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. *Anatomic validation of the method*. *Circulation*. 1977;55:613-618.
22. de Simone G, Daniels SR, Devereux RB, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. *J Am Coll Cardiol*. 1992;20:1251-1260.
23. Flynn JT, Falkner BE. New clinical practice guideline for the management of high blood pressure in children and adolescents. *Hypertension*. 2017;70:683-686.
24. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat 11*. 2002:11(246):1-190.
25. Ganau A, Devereux RB, Roman MJ, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. *J Am Coll Cardiol*. 1992;19:1550-1558.
26. Devereux R, Pickering TG. What is normal blood pressure?: Comparison of ambulatory pressure level and variability in patients with normal or abnormal left ventricular geometry. *Am J Hypertens*. 1993;6:211s-215s.
27. Matteucci MC, Wühl E, Picca S, et al. Left ventricular geometry in children with mild to moderate chronic renal insufficiency. *J Am Soc Nephrol*. 2006;17:218-226.
28. Giles TD. Assessment of global risk: a foundation for a new, better definition of hypertension. *J Clin Hypertens (Greenwich)*. 2006;8:5-14; quiz 39.
29. Sharma P, Middelberg RP, Andrew T, Johnson MR, Christley H, Brown MJ. Heritability of left ventricular mass in a large cohort of twins. *J Hypertens*. 2006;24:321-324.
30. Arnett DK, de las Fuentes L, Broeckel U. Genes for left ventricular hypertrophy. *Curr Hypertens Rep*. 2004;6:36-41.
31. Verhaaren HA, Schieken RM, Mosteller M, Hewitt JK, Eaves LJ, Nance WE. Bivariate genetic analysis of left ventricular mass and weight in pubertal twins (the Medical College of Virginia twin study). *Am J Cardiol*. 1991;68:661-668.
32. Foppa M, Duncan BB, Arnett DK, et al. Diabetes, gender, and left ventricular structure in African-Americans: the atherosclerosis risk in communities study. *Cardiovasc Ultrasound*. 2006;4:43.
33. Mule G, Cerasola G. The metabolic syndrome and its relationship to hypertensive target organ damage. *J Clin Hypertens (Greenwich)*. 2006;8:195-201.
34. Foster BJ, Mackie AS, Mitsnefes M, Ali H, Mamber S, Colan SD. A novel method of expressing left ventricular mass relative to body size in children. *Circulation*. 2008;117:2769-2775.
35. Oktay AA, Lavie CJ, Milani RV, et al. Current perspectives on left ventricular geometry in systemic hypertension. *Prog Cardiovasc Dis*. 2016;59:235-246.

How to cite this article: Shilly S, Merchant K, Singer P, et al. Left ventricular cardiac geometry and ambulatory blood pressure in children. *J Clin Hypertens*. 2019;21:566–571. https://doi.org/10.1111/jch.13540