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In clinic blood pressure prediction of normal ambulatory blood pressure monitoring in pediatric hypertension referrals

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Abstract

OBJECTIVE—Since younger patients have low pretest probability of hypertension and are susceptible to reactive and masked hypertension, ambulatory blood pressure monitoring can be useful. To better target use in referred patients, we sought to define in-clinic systolic blood pressure measures that predicted normal ambulatory blood pressure monitoring and target end organ damage.

DESIGN,SETTING,PATIENTS,OUTCOME MEASURES—Data were collected on consecutive patients referred for high blood pressure undergoing an ambulatory blood pressure monitor from 2010–13 (n=248, 33.9% female, mean age 15.5 ± 3.6 years). Candidate in-clinic predictors were systolic maximum, minimum or average blood pressures obtained by auscultative, oscillometric or both. Multivariable logistic regression models were used to determine the prediction of normal ambulatory blood pressure monitoring by in-clinic blood pressure predictors. Separate models considered predicting left ventricular hypertrophy by in-clinic systolic blood pressure versus ambulatory blood pressure monitoring-defined hypertension. Identified predictor utility was tested with receiver operator characteristic curves.

RESULTS—Maximum (OR 0.97 [95%CI 0.94–0.99]; $p=0.047$), minimum (0.96 [0.94–0.99]; $p=0.002$) and average (0.97 [0.95–1.00]; $p=0.04$) in-clinic auscultative systolic blood pressure predicted normal ambulatory blood pressure monitoring. Each had a cstatistic of 0.58. Left ventricular hypertrophy was associated with in-clinic auscultative minimum systolic blood pressure treated continuously (1.05, [1.01 – 1.10], $p=0.01$) or dichotomized at the 90th percentile

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AUTHOR CONTRIBUTIONS

PKJ drafted the article; acquired, analyzed and interpreted, data; and approved the submitted manuscript.

MAF contributed to the design; acquired data; critically revised the manuscript for important intellectual content, and approved of the submitted manuscript.

JPZ conceived the project and design; acquired, analyzed and interpreted data; critically revised the manuscript for important intellectual content; and approved of the submitted manuscript.

(8.23, [1.48 – 45.80], $p=0.02$), as well as ambulatory blood pressure monitoring-defined hypertension (3.31, [1.23 – 8.91], $p = 0.02$). Both predictors had poor sensitivity and specificity.

CONCLUSION—In youth, normal auscultative in-clinic systolic blood pressure indices weakly predicted normal ambulatory blood pressure and target end organ damage.

Keywords

Ambulatory blood pressure monitoring; Blood pressure; Hypertension; Left ventricular hypertrophy; Target end organ damage; white coat hypertension

INTRODUCTION

Measuring blood pressure (BP) is a recommended element of every healthcare encounter in children older than 3 years of age. A significant proportion of these measurements result in high (1.6%) or borderline high (9.4%) BP, requiring further clinical action (1). Increased risk for long-term adverse effects such as mortality and cardiovascular disease events due to childhood and/or adolescent hypertension are well-documented (2–4). Chronic hypertension in youth may also result in left ventricular hypertrophy (LVH), which has been found to predict future cardiovascular events (5). According to a population-based metacohort, resolving elevated childhood BP by adulthood seems to ameliorate subclinical arteriosclerosis (6). Prompt BP screening and accurate identification of hypertension early on is essential in reducing the associated negative health effects, especially considering its ability to “track” throughout a patient’s lifetime (7). In fact, the 2010 Affordable Care Act now mandates coverage for screening BP in children, underscoring universal screening as a possible standard of practice (8).

In youth, while true hypertension is rare, reactive or white-coat hypertension occurs at a very high rate: upwards of 32% in pediatric referral clinics (9, 10). Masked hypertension, defined as normal in-clinic BP but elevated ABPM, appears in only approximately 8% of referred children, but much like true hypertension, associates with cardiovascular risk in adults (10–12). Prehypertension, which arises in patients who have an elevated in-clinic BP as well as a systolic blood pressure (SBP) load of 25–50%, is known to occur in roughly 20% of boys and 13% of adolescent girls (13). While the prehypertensive state itself is not detrimental, it is predicted that 10% of adults and 7% of adolescents who are prehypertensive will develop true hypertension each year (13, 14). Additionally, pediatric patients with prehypertension have demonstrated signs of target-organ damage, including higher carotid intermedial thickness, reduced glomerular filtration rate, elevated proteinuria, and left ventricular mass values comparable to children with sustained hypertension (15).

Ambulatory blood pressure monitoring (ABPM) has demonstrated many advantages over clinical BP measurement alone, including accurate identification of reactive, masked, and pre-hypertension, and information on nocturnal BP patterns (16). Despite this utility, ABPM is not emphasized in routine elevated BP work-up in the 2004 National High Blood Pressure Educational Program Fourth Report or 2011 Expert Panel on Cardiovascular Health and Risk Reduction in Children and Adolescents (5, 17). ABPM therefore plays an unclear role in BP diagnosis. While the per unit cost of ABPM would seem to be prohibitive, formal

analysis reveals that the use of ABPM may be cost-effective, if not cost-saving (9, 18, 19). Additional targeting of ABPM use would likely further enhance its cost-effectiveness and avoid unnecessary intrusive testing in healthy children during clinical management.

Within a pediatric referral hypertension clinic patient population, we sought to identify simple in-clinic SBP predictors of normal ABPM, including normotension and reactive hypertension, and ABPM's deviating from the norm (masked, pre-hypertension, ambulatory and severe ambulatory hypertensions). ABPM hypertension and in-clinic SBP's ability to predict target organ damage in the form of LVH was also examined.

METHODS

Consecutive patients ages 8–25 referred between 2010–2013 to Renal or Preventive Cardiology clinics for elevated BP at a pediatric, academic, tertiary care institution were eligible at the time of initial oscillometric ABPM (Spacelabs 90,217 or 90,207-Ultralite; Snoqualmie, WA). ABPM were ordered for clinical indications and were completed over a 24-hour period with readings occurring in 20-minute intervals from 6am to 10pm and 60-minute intervals from 10pm to 6am. Standard 10pm to 6am sleep/wake diurnal patterns were adjusted by patient diary entries. We excluded patients with repeat ABPM, if less than 100% of daytime readings were captured or more than 2 hours of nighttime readings were missed, persons already on blood pressure therapy, and young children (less than 8 years old) in order to focus on the patients in our system with the most success in completing ABPM and least selection bias.

Clinic variables including anthropometrics, demographics, medical history, medications, echocardiographic findings and clinic BP were extracted. Absolute body mass index (BMI) was converted to z-score using Centers for Disease Control age-sex derived BMI z-score for 2–20 year olds. Clinic BPs were performed per The Fourth Report On The Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents: right arm, patient in the seated position, legs squarely supported by the floor or step, and adequate cuff selection whereby 40% of mid-arm circumference was covered (5). Variable numbers of clinic BPs were obtained at the preference of the treating physician. This study was approved by the Institutional Committee on Clinical Investigation with a patient consent waiver.

Categorizing Outcomes and Predictors

The outcome of interest was normal ABPM. Normal ABPM was defined as the combination of a) 24 hour average SBP below the 95th percentile for age and sex and b) the proportion of all SBP readings for the 24 hour period above the 95th percentile threshold amounting to less than 25%. ABPMs outside either of these two limits were deemed abnormal, i.e. load over 25% and/or 24-hour average greater than the 95th percentile. This definition of abnormal is consistent with the ABPM classification recommended schema for children and adults and may include prehypertension, ambulatory hypertension, severe ambulatory hypertension and masked hypertension (16, 20). The secondary outcome was LVH by a 2D echocardiogram obtained at the date nearest the completed ABPM. For present purposes, LVH was defined as LVM to volume ratio age-sex-height referenced z-score greater than 2.00 (21). Predictors

included maximum, minimum and average clinic SBP, either auscultative or automated. Covariates included age, sex, and height at in-clinic visit.

Statistical Analysis

Clinical characteristics between normal ABPM versus not normal ABPM groups were compared by chi-squared analysis for categorical variables or Student *t* test for continuous variables. Logistic regression models adjusted for age, sex, and height were used to determine the relation between normal ABPM, as the binary outcome, and clinic SBP indices (auscultative, automated; highest, lowest, and average), which were sequentially replaced as the predictor of interest. Clinic SBP indices were treated as continuous variables and then as a binary high-low variable dichotomized at the age-sex-height referenced 90th percentile. Receiver operator characteristic (ROC) curves at 95% confidence were then used to test the respective utility of significant in-clinic BP indices in the prediction of normal ABPM.

To compare the ability of identified predictors of normal ABPM versus ABPM itself in the prediction of target organ damage, secondary analyses used logistic regression to determine associations between LVH and in-clinic BP predictors identified in the primary analysis or separately ABPM hypertension (e.g. severe hypertension, ambulatory hypertension or masked hypertension) (16). Then, ROC curves were constructed, whereby positive predictive values (PPV), negative predictive values (NPV), sensitivity and specificity values, and likelihood ratios were determined. Analyses performed used SPSS (SPSS, Inc. version 21.0, Aramark, NY).

RESULTS

Study Population

Our cohort included 248 patients with mean age 15.5 ± 3.6 and 33.9% female, following the exclusion of 58 patients for repeat study, age, or inadequate capture. Amongst normal ABPM (N = 127) and not normal ABPM (N = 121) groups, no significant differences in clinical characteristics, including anthropometrics, heart rate, pertinent medical histories (diabetes, genetic syndrome, prematurity, cardiac or renal disease affecting cardiac outflow tracts), or medications (attention deficit hyperactive disorder, oral contraceptive, or anti-psychotic medications) were found [Table 1]. By definition, ABPM parameters between the two groups were markedly different. The normal ABPM group was populated by 58.0% with normotension and 42.0% with reactive hypertension, while the not normal ABPM group was comprised of 33.9% with prehypertension, 17.3% with masked hypertension, 11.0% with ambulatory hypertension, and 37.8% with severe ambulatory hypertension.

Clinical Indices Associated with Normal ABPM and LVH

Multivariable adjusted logistic regression demonstrated that maximum, minimum and average clinic auscultative SBP indices were associated with normal ABPM, while automated indices were not [Table 2]. ROC curves with normal ABPM as the outcome variable and clinic auscultative SBP maximum, minimum, and average as the predictors each had c-statistics of 0.58 (CI: 0.50 – 0.66). Maximum, minimum, and average in-clinic

auscultative SBP at or above the 90th percentile had poor sensitivity, specificity, PPV and NPV for normal ABPM. For normal ABPM, maximum auscultative clinic SBP >90th had sensitivity of 43.3% and specificity of 33.3%, minimum auscultative clinic SBP >90th had a sensitivity of 36.1% and specificity of 51.0%, while average auscultative clinic SBP >90th had sensitivity of 38.1% and a specificity of 59.4%. For LVH, ABPM hypertension had sensitivity of 61.1% and specificity of 67.4%, while minimum auscultative clinic SBP >90th had a sensitivity of 63.6% and a specificity of 58.8%.

With respect to target organ damage, clinic auscultative maximum SBP was significantly associated with LVH treated as a binary variable dichotomized at the 90th percentile (N=191, OR: 8.23, [CI: 1.48 – 45.8], p=0.02), while clinic auscultative minimum SBP approached significance (N=191, OR: 3.92, [CI: 0.98 – 15.66], p=0.05) and average clinic auscultative SBP was not significant (N=191, OR: 2.96, [0.75 – 11.77], p=.12).

Alternatively, ABPM hypertension was also significantly associated with LVH (OR: 3.31, [CI: 1.23 – 8.91], p = 0.02). For LVH, ABPM hypertension and auscultative minimum SBP at or above the 90th percentile were found to have similar test characteristics, especially in terms of sensitivity (61.1% and 63.6%, respectively) and NPV (95.7% and 96.4% respectively). Likelihood ratios for the prediction of LVH and ABPM hypertension was calculated to be 1.89, while for dichotomized minimum SBP it was 1.54.

DISCUSSION

This study aimed to identify in-clinic SBP predictors of normal ABPM, thereby obviating the need for ABPM. We also examined the predictive role of in-clinic BP or ABPM to identify youth with target end organ damage, specifically LVH (5). Maximum, minimum and average in-clinic auscultative SBP indices were indeed associated with normal ABPM, but no reasonable in-clinic BP threshold could be found to dichotomously predict normal ABPM. Furthermore, although ABPM-defined hypertension and in-clinic auscultative minimum SBP were significant predictors of LVH, each had similarly poor sensitivity and specificity.

Metaanalyses of the adult literature state the sensitivity of clinic BP for ABPM hypertension is 85.6% while specificity is 45.9% (22). Turning to pediatrics, our results extend previous results demonstrating the weak predictive utility of in-clinic SBP for predicting ABPM outcomes (19). An often-cited prior investigation found that higher BP made white-coat hypertension, i.e. normal ABPM results despite abnormal clinic BP, less likely (23). In an adult study, SBP measurements in a single clinic visit were found to predict patients unlikely to have a large white-coat or masked effect, potentially allowing for better targeting of home monitoring in clinical practice (24). The present results confirm that higher clinic BP makes normal ABPM less likely, but also clarifies that using clinic SBP may be insufficiently sensitive to rule out the occurrence of actionable ABPM findings. Additionally, we observed associations in only auscultative BPs, but not automated.

ABPM hypertension's association with LVH was comparable to previous work, which demonstrated in children an increased likelihood to accrue LVH with more severe hypertension (25). LVH may be related to duration of exposure to hypertension (26–28).

Metanalyses show higher LVM values in hypertensive versus normotensive patients (29). Emerging data is now filling in the evidence gap to disentangle the relative contributions of hypertension versus obesity on the development of cardiovascular consequences in young adulthood. Data from the Coronary Artery Risk Development in Youth study demonstrated in young adults that progressive loss of ideal cardiovascular disease health markers, such as the occurrence of obesity or hypertension, predicts the progressive increase in LVM (13). Extending this observation to childhood, investigators from the Bogalusa Heart Study found that children with elevated BMI and elevated BP developed higher LVM in adulthood, but obesity was a stronger predictor (27). Similarly, investigators from the Fels longitudinal study found childhood BMI, but not fat mass or other adiposity indices, predicted future LVH (28). In each of these studies, BMI was a stronger predictor of future LVH than was BP. Pinpointing LVH early on and being able to accurately assess the risk of having LVH is significantly beneficial to hypertensive patients as LVH leads to higher rates of morbidity, congestive heart failure, and cardiac events such as myocardial infarction (18, 22, 30). BMI may also affect LVH directly through increased volume, or indirectly through neurohormonal changes in atrial natriuretic peptide, insulin, aldosterone levels (31, 32). Since BMI is a more stable phenotype throughout a patient's lifetime than BP, observing BMI tracking trends may be more useful (33). Our data suggests that ABPM and in-clinic BP are suboptimal predictors of LVH, perhaps suggesting other evidence-based criteria ought to be used to identify lifestyle-associated LVH.

There are several limitations within this study. Since the subject population is retrospective in nature, direct prospective extrapolations cannot be made. The number of auscultative, automated or combined number of in-clinic BPs were determined by treating clinicians and are therefore variable, reflecting real world practice. Only patients seen in referral clinics were included in the study, creating a selection bias towards the clinical indications for undergoing an ABPM. Consequently, results of this study should not be applied to the general population. Variation in BP measurement technique between clinics is another factor potentially influencing our results; however, the data gathered ultimately reflects dissimilarities in the true clinical setting. Highly severe hypertensive patients may have been admitted to inpatient wards, possibly lowering the number of severe ambulatory hypertensive patients in our study. Clinic BP data in the primary care setting was not obtained, so no correlations of BP variability before, during and after the child's referral were made.

In conclusion, our data suggest that in-clinic auscultative SBP (maximum, minimum and average) was a significant but weak predictor of normal ABPM, and ABPM hypertension along with auscultative minimum SBP associated with LVH in pediatric patients referred for abnormal BP. Future prospective research is needed to look into the various clinic predictors of ABPM diagnoses in both referral and primary care settings.

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REFERENCES

1. Kit BK, Kuklina E, Carroll MD, Ostchega Y, Freedman DS, Ogden CL. Prevalence of and trends in dyslipidemia and blood pressure among US children and adolescents, 1999–2012. *JAMA pediatrics*. 2015 Mar; 169(3):272–279. PubMed PMID: 25599372. [PubMed: 25599372]
2. Franks PW, Hanson RL, Knowler WC, Sievers ML, Bennett PH, Looker HC. Childhood obesity, other cardiovascular risk factors, and premature death. *N Engl J Med*. 2010 Feb 11; 362(6):485–493. PubMed PMID: 20147714. Pubmed Central PMCID: 2958822. Epub 2010/02/12. eng. [PubMed: 20147714]
3. Sundstrom J, Neovius M, Tynelius P, Rasmussen F. Association of blood pressure in late adolescence with subsequent mortality: cohort study of Swedish male conscripts. *BMJ*. 2011; 342:d643. PubMed PMID: 21343202. Pubmed Central PMCID: 3042737. Epub 2011/02/24. eng. [PubMed: 21343202]
4. Gray L, Lee IM, Sesso HD, Batty GD. Blood pressure in early adulthood, hypertension in middle age, future cardiovascular disease mortality: HAHS (Harvard Alumni Health Study). *J Am Coll Cardiol*. 2011 Nov 29; 58(23):2396–2403. PubMed PMID: 22115646. Pubmed Central PMCID: 3253414. Epub 2011/11/26. eng. [PubMed: 22115646]
5. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004 Aug; 114(2 Suppl 4th Report):555–576. PubMed PMID: 15286277. Epub 2004/08/03. eng. [PubMed: 15286277]
6. Juhola J, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, et al. Combined effects of child and adult elevated blood pressure on subclinical atherosclerosis: the International Childhood Cardiovascular Cohort Consortium. *Circulation*. 2013 Jul 16; 128(3):217–224. PubMed PMID: 23780579. Pubmed Central PMCID: 3875837. Epub 2013/06/20. eng. [PubMed: 23780579]
7. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008 Jun 24; 117(25):3171–3180. PubMed PMID: 18559702. Pubmed Central PMCID: 3568631. [PubMed: 18559702]
8. [cited 2013 November] Patient Protection and Affordable Care Act 2010. Available from: <http://www.gpo.gov/fdsys/pkg/BILLS-111hr3590enr/pdf/BILLS-111hr3590enr.pdf>
9. Swartz SJ, Srivaths PR, Croix B, Feig DI. Cost-effectiveness of ambulatory blood pressure monitoring in the initial evaluation of hypertension in children. *Pediatrics*. 2008 Dec; 122(6):1177–1181. PubMed PMID: 19047231. Epub 2008/12/03. eng. [PubMed: 19047231]
10. Florianczyk T, Werner B. Usefulness of ambulatory blood pressure monitoring in diagnosis of arterial hypertension in children and adolescents. *Kardiol Pol*. 2008 Jan; 66(1):12–17. discussion 8. PubMed PMID: 18266183. Epub 2008/02/13. eng. [PubMed: 18266183]
11. Bjorklund K, Lind L, Zethelius B, Andren B, Lithell H. Isolated ambulatory hypertension predicts cardiovascular morbidity in elderly men. *Circulation*. 2003 Mar 11; 107(9):1297–1302. PubMed PMID: 12628951. Epub 2003/03/12. eng. [PubMed: 12628951]
12. Lurbe E, Torro I, Alvarez V, Nawrot T, Paya R, Redon J, et al. Prevalence, persistence, and clinical significance of masked hypertension in youth. *Hypertension*. 2005 Apr; 45(4):493–498. PubMed PMID: 15767467. Epub 2005/03/16. eng. [PubMed: 15767467]
13. Falkner B, Gidding SS, Portman R, Rosner B. Blood pressure variability and classification of prehypertension and hypertension in adolescence. *Pediatrics*. 2008 Aug; 122(2):238–242. PubMed PMID: 18676538. [PubMed: 18676538]
14. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *Jama*. 2003 May 21; 289(19):2560–2572. PubMed PMID: 12748199. [PubMed: 12748199]
15. Flynn JT, Daniels SR, Hayman LL, Maahs DM, McCrindle BW, Mitsnefes M, et al. Update: ambulatory blood pressure monitoring in children and adolescents: a scientific statement from the American Heart Association. *Hypertension*. 2014 May; 63(5):1116–1135. PubMed PMID: 24591341. Pubmed Central PMCID: 4146525. [PubMed: 24591341]
16. Urbina E, Alpert B, Flynn J, Hayman L, Harshfield GA, Jacobson M, et al. Ambulatory blood pressure monitoring in children and adolescents: recommendations for standard assessment: a

scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee of the council on cardiovascular disease in the young and the council for high blood pressure research. *Hypertension*. 2008 Sep; 52(3):433–451. PubMed PMID: 18678786. Epub 2008/08/06. eng. [PubMed: 18678786]

17. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011 Dec; 128(Suppl 5):S213–S256. PubMed PMID: 22084329. Epub 2011/11/16. eng. [PubMed: 22084329]
18. Lovibond K, Jowett S, Barton P, Caulfield M, Heneghan C, Hobbs FD, et al. Cost-effectiveness of options for the diagnosis of high blood pressure in primary care: a modelling study. *Lancet*. 2011 Oct 1; 378(9798):1219–1230. PubMed PMID: 21868086. Epub 2011/08/27. eng. [PubMed: 21868086]
19. Davis ML, Ferguson MA, Zachariah JP. Clinical predictors and impact of ambulatory blood pressure monitoring in pediatric hypertension referrals. *Journal of the American Society of Hypertension : JASH*. 2014 Sep; 8(9):660–667. PubMed PMID: 25065681. Pubmed Central PMCID: 4167561. [PubMed: 25065681]
20. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, et al. Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation*. 2005 Feb 8; 111(5):697–716. PubMed PMID: 15699287. Epub 2005/02/09. eng. [PubMed: 15699287]
21. Guerra VC, Leister EC, Williams PL, Starc TJ, Lipshultz SE, Wilkinson J, et al. Long-Term Effects of In Utero Antiretroviral Exposure: Systolic and Diastolic Function in HIV-Exposed Uninfected Youth. *AIDS research and human retroviruses*. 2016 Jan 21. PubMed PMID: 26794032.
22. Hodgkinson J, Mant J, Martin U, Guo B, Hobbs FD, Deeks JJ, et al. Relative effectiveness of clinic and home blood pressure monitoring compared with ambulatory blood pressure monitoring in diagnosis of hypertension: systematic review. *BMJ*. 2011; 342:d3621. PubMed PMID: 21705406. Pubmed Central PMCID: 3122300. [PubMed: 21705406]
23. Sorof JM, Poffenbarger T, Franco K, Portman R. Evaluation of white coat hypertension in children: importance of the definitions of normal ambulatory blood pressure and the severity of casual hypertension. *Am J Hypertens*. 2001 Sep; 14(9 Pt 1):855–860. PubMed PMID: 11587149. Epub 2001/10/06. eng. [PubMed: 11587149]
24. Sheppard JP, Holder R, Nichols L, Bray E, Hobbs FD, Mant J, et al. Predicting out-of-office blood pressure level using repeated measurements in the clinic: an observational cohort study. *J Hypertens*. 2014 Nov; 32(11):2171–2178. discussion 8. PubMed PMID: 25144295. Pubmed Central PMCID: 4222615. [PubMed: 25144295]
25. McNiece KL, Gupta-Malhotra M, Samuels J, Bell C, Garcia K, Poffenbarger T, et al. Left ventricular hypertrophy in hypertensive adolescents: analysis of risk by 2004 National High Blood Pressure Education Program Working Group staging criteria. *Hypertension*. 2007 Aug; 50(2):392–395. PubMed PMID: 17592068. Pubmed Central PMCID: 3582191. [PubMed: 17592068]
26. Gidding SS, Liu K, Colangelo LA, Cook NL, Goff DC, Glasser SP, et al. Longitudinal determinants of left ventricular mass and geometry: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Circulation Cardiovascular imaging*. 2013 Sep; 6(5):769–775. PubMed PMID: 23922005. Pubmed Central PMCID: 3873157. [PubMed: 23922005]
27. Lai CC, Sun D, Cen R, Wang J, Li S, Fernandez-Alonso C, et al. Impact of long-term burden of excessive adiposity and elevated blood pressure from childhood on adulthood left ventricular remodeling patterns: the Bogalusa Heart Study. *J Am Coll Cardiol*. 2014 Oct 14; 64(15):1580–1587. PubMed PMID: 25301461. Pubmed Central PMCID: 4194028. [PubMed: 25301461]
28. Sabo RT, Yen MS, Daniels S, Sun SS. Associations between childhood body size, composition, blood pressure and adult cardiac structure: the Fels Longitudinal Study. *PLoS One*. 2014; 9(9):e106333. PubMed PMID: 25191997. Pubmed Central PMCID: 4156369. [PubMed: 25191997]
29. Kollias A, Dafni M, Poulidakis E, Ntineri A, Stergiou GS. Out-of-office blood pressure and target organ damage in children and adolescents: a systematic review and meta-analysis. *J Hypertens*. 2014 Dec; 32(12):2315–2331. discussion 31. PubMed PMID: 25304469. [PubMed: 25304469]

30. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med*. 1990 May 31; 322(22):1561–1566. PubMed PMID: 2139921. [PubMed: 2139921]
31. Volpe M, Rubattu S, Burnett J Jr. Natriuretic peptides in cardiovascular diseases: current use and perspectives. *European heart journal*. 2014 Feb; 35(7):419–425. PubMed PMID: 24227810. Pubmed Central PMCID: 4023301. [PubMed: 24227810]
32. Huynh K, Bernardo BC, McMullen JR, Ritchie RH. Diabetic cardiomyopathy: mechanisms and new treatment strategies targeting antioxidant signaling pathways. *Pharmacology & therapeutics*. 2014 Jun; 142(3):375–415. PubMed PMID: 24462787. [PubMed: 24462787]
33. Deshmukh-Taskar P, Nicklas TA, Morales M, Yang SJ, Zakeri I, Berenson GS. Tracking of overweight status from childhood to young adulthood: the Bogalusa Heart Study. *European journal of clinical nutrition*. 2006 Jan; 60(1):48–57. PubMed PMID: 16132057. [PubMed: 16132057]

Table 1

Clinical Characteristics.

<i>Patient Variable</i>	<i>ABPM Category</i>		<i>p-value</i>
	Normal (N = 121)	Not Normal (N = 127)	
Age (years)	15.8 ± 3.2	15.3 ± 4.0	.28
% Female	32.2%	35.4%	.69
Height (cm)	167.1 ± 14.4	164.9 ± 16.4	.26
Height z score	0.30 ± 1.07	0.44 ± 1.04	.32
Weight (kg)	77.3 ± 25.3	74.6 ± 26.3	.42
Weight z score	1.27 ± 1.17	1.27 ± 1.14	.97
BMI (kg/m ²)	27.0 ± 8.1	26.6 ± 7.0	.63
BMI z score	1.46 ± 3.29	1.19 ± 0.99	.37
Clinic heart rate	84 ± 19	85 ± 17	.74
ABPM average SBP	121 ± 8	131 ± 8	p <.001
ABPM average DBP	66 ± 6	73 ± 8	p <.001
ABPM SBP Load	16.5%	56.0%	p <.001
ABPM SBP Dip	7.0%	9.2%	.02
BP Diagnosis Category (% of ABPM category)			
Normotensive	70 (57.9%)	-	-
Reactive hypertension	51 (42.1%)	-	-
Pre-hypertension	-	43 (33.9%)	-
Masked hypertension	-	22 (17.3%)	-
Ambulatory hypertension	-	14 (11.0%)	-
Severe ambulatory hypertension	-	48 (37.8%)	-

Normal indicates ABPM normal and white coat (reactive) hypertension. Not normal indicates prehypertension, masked, ambulatory and severe hypertension.

BMI: body mass index; LVM: left ventricular mass; ABPM: ambulatory blood pressure monitor; BP: blood pressure.

Data presented as mean ± standard deviation or proportion as listed. Chi-squared analysis for categorical variables or Student *t*-test for continuous variables Student's *t*-test was used to identify differences among normal and not normal groupings, where a p-value is shown.

Table 2

Clinic SBP predicting normal ABPM: logistic regression results

<i>Clinic SBP Indices</i>		<i>Normal ABPM (per mmHg clinic SBP)</i>	
	n	Odds Ratio- (95% CI)	p-value
Automated			
Maximum	230	0.99 (0.97 – 1.01)	0.29
Minimum	230	0.98 (0.96 – 1.01)	0.14
Average	230	0.98 (0.96 – 1.01)	0.14
Auscultative			
Maximum	191	0.97 (0.94 – 0.99)	0.047
Minimum	191	0.96 (0.94 – 0.99)	0.002
Average	191	0.97 (0.95 – 1.00)	0.04

SBP: systolic blood pressure; ABPM: ambulatory blood pressure monitor; CI: confidence interval.

Odds ratio adjusted for age, sex, and height. Normal ABPM is the binary outcome while clinic SBP indices are sequentially replaced as the predictor.