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Prevalence and impact of abnormal blood pressure on left ventricular hypertrophy in adolescents with congenital heart disease☆

Aaron T Walsh [a,b,1,*], Kan N Hor [a,b,#], Mariah Eisner [a,c,d], Chance Alvarado [a,c,d], Mahmoud Kallash [b,e], John David Spencer [b,e], Andrew H Tran [a,b]

[a] The Heart Center, Nationwide Children's Hospital, Columbus, OH, USA
[b] Department of Pediatrics, The Ohio State University College of Medicine, Columbus, OH, USA
[c] Biostatistics Resource at Nationwide Children's Hospital, Columbus, OH, USA
[d] Center for Biostatistics, The Ohio State University College of Medicine, Columbus, OH, USA
[e] Division of Nephrology and Hypertension, Nationwide Children's Hospital, Columbus, OH, USA

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ABSTRACT

Background: Left ventricular hypertrophy (LVH) secondary to hypertension is associated with cardiovascular events in adulthood. Prevalence of abnormal blood pressure and LVH in youths with congenital heart disease (CHD) is understudied despite childhood hypertension predicting adult hypertension. This study aimed to describe the prevalence of hypertension and LVH in adolescents with CHD and factors associated with LVH in this population.

Methods: This was a retrospective analysis of echocardiogram reports from patients with CHD aged 13–17 years with documented systolic blood pressure (SBP), height, weight, and left ventricular mass (LVM) indexed to body size (LVMI-ht$^{2.7}$). Patients were stratified by SBP and CHD type. Hypertension and LVH prevalence were calculated; linear regression models assessed factors associated with LVH.

Results: Of 853 patients (mean age 15.5 ± 1.5 years, 57.1 % male), 25.1 % had elevated SBP, whereas 11.6 % and 5.7 % had stage 1 and stage 2 hypertension, respectively. LVH was more prevalent with higher SBP (37.4 % elevated, 32.3 % stage 1 hypertension, and 40.7 % stage 2 hypertension) versus 19.6 % normotensive. BMI percentile and SBP were significantly associated with LVMI-ht$^{2.7}$; for 10 % BMI percentile and 10 mmHg SBP increases, LVMI-ht$^{2.7}$ increased by 1.2 g/m$^{2.7}$ and 0.93 g/m$^{2.7}$, respectively, after adjustment for age, sex, race, SBP, BMI, and CHD lesion.

Conclusions: Adolescents with CHD have a high prevalence of abnormal SBP and LVH. BMI percentile and SBP were associated with LVMI-ht$^{2.7}$. Findings support screening for BMI and hypertension in youths with CHD as this population has increased baseline cardiovascular risk that may be compounded by obesity and chronic hypertension.

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* Corresponding author at: The Heart Institute, Le Bonheur Children's Hospital, 51 N. Dunlap St., 3rd Floor, Memphis, TN 38105, USA.
E-mail addresses: Awalsh8@uthsc.edu (A.T. Walsh), Kan.Hor@nationwidechildrens.org (K.N. Hor), Mariah.Eisner@osumc.edu (M. Eisner), Chance.Alvarado@nationwidechildrens.org (C. Alvarado), Mahmoud.Kallash@nationwidechildrens.org (M. Kallash), John.Spencer@nationwidechildrens.org (J.D. Spencer), Andrew.Tran@nationwidechildrens.org (A.H. Tran).
[1] Permanent address: 51 N. Dunlap St., 3rd floor, The Heart Institute, Memphis, TN, 38105, USA.
[#] Equal contribution.

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Glossary

LVH: Left ventricular hypertrophy
HTN: Hypertension
CHD: Congenital heart disease
LVM: Left ventricular mass
LVMI-ht$^{2.7}$: Left ventricular mass indexed to patient height to the power of 2.7
NT: Normotensive
E-BP: Elevated blood pressure
HTN-1: Stage 1 hypertension
HTN-2: Stage 2 hypertension
SBP: Systolic blood pressure
LVOT: Left ventricular outflow tract
BMI: Body mass index
LVSF: Left ventricular shortening fraction
TOF: Tetralogy of Fallot

1. Introduction

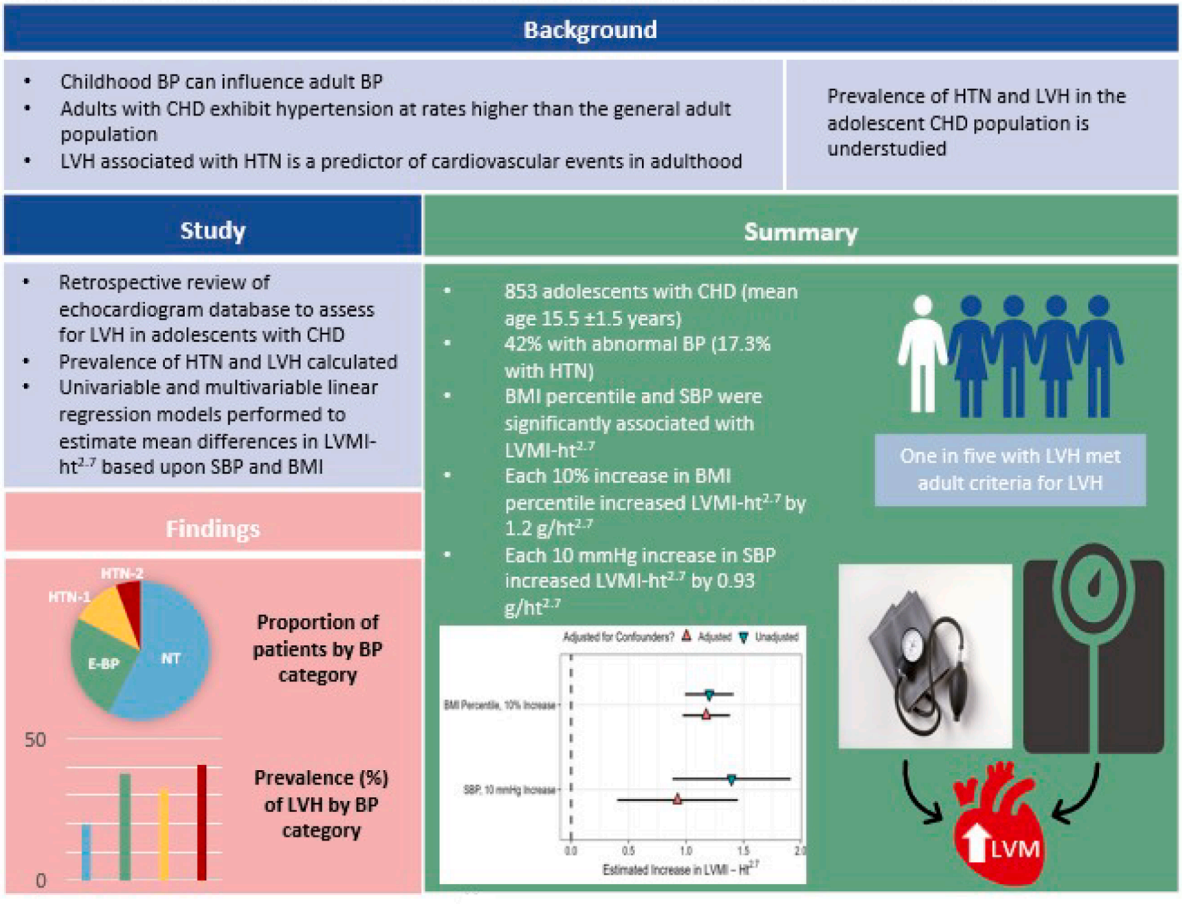
Congenital heart disease (CHD) is the most common birth defect accounting for one-third of all congenital anomalies [1]. With improvements in perinatal care, pharmacology, interventional techniques, and surgical outcomes, more patients born with CHD are living past the first decade of life [2]. Adults with CHD now comprise two-thirds of the total CHD population and manifest several cardiovascular (CV) risk factors at rates higher than the general adult population [3]. Large international population-based studies of adults with CHD found hypertensive disease to be more common in younger adults (<40 years) with CHD compared to their respective general populations [4]. Among a large US adult population, those with CHD were more likely to have hypertension (HTN) than peers without CHD [5]. These studies used a lower age limit of 18 years, thereby excluding the adolescent population who may provide insight into the timing of development of risk factors.

Left ventricular hypertrophy (LVH) as detected by echocardiography is associated with HTN and confers an increased risk of adverse CV events in adulthood. Increased left ventricular mass (LVM) in adults is independently associated with CV disease, death from CV disease, and all-cause mortality even after adjustment for traditional risk factors [6]. The prognostic ability of LVH for CV events has held true across diverse ethnic populations and in those with essential HTN [7,8]. In adolescents with essential HTN, LVM correlated with HTN [9] with some adolescents demonstrating LVM values that when found in adults are associated with a fourfold increase in CV disease [10]. Childhood blood pressure has been shown to correlate with adult blood pressure level [11–13]. However, the prevalence of HTN and LVH in adolescents with CHD is less well-known. Therefore, the aims of our study were to describe the prevalence of HTN and LVH in adolescents with CHD and to assess risk factors for the development of LVH in this ever-growing at-risk population.

2. Methods

2.1. Study population

We performed a retrospective analysis of echocardiogram reports from adolescents with CHD at Nationwide Children's Hospital in Columbus, OH from the time of adoption of the current electronic medical record (2012) up to the time our division transitioned to a new echocardiographic reading software (2019). Reports from all patients with



Central Illustration: Prevalence and Impact of Abnormal Blood Pressure on Left Ventricular Hypertrophy in Adolescents with Congenital Heart Disease.

CHD aged 13–17 years, excluding those with single right ventricular lesions, were reviewed and those with complete data on LVM were included. We chose this age range because blood pressure thresholds used to define hypertension in this age group are congruent with those used in the adult population [14] in which hypertension-associated adverse CV outcomes have been described.

Patients were stratified by CHD lesion into those with left-to-right shunts (atrial septal defect, ventricular septal defect, atrioventricular canal defect, patent ductus arteriosus), left ventricular outflow tract (LVOT) obstruction (subvalvar aortic stenosis, valvar aortic stenosis, bicuspid aortic valve, supravalvar aortic stenosis, and coarctation of the aorta), isolated pulmonary valve stenosis (PS), tetralogy of Fallot (TOF), and conotruncal anomalies (transposition of the great arteries and truncus arteriosus). Patients with uncommon lesions in our cohort (e.g., isolated atrioventricular valve defects, Ebstein's anomaly, coronary artery anomalies, single ventricle physiology) were grouped into an "Other" category. Those with single ventricle physiology included only patients with a systemic left ventricle. Lesions with any obstruction to blood flow out of the left ventricle, regardless of primary defect, were included in the LVOT obstruction group to maintain validity of the data. Peak Doppler velocity through the LVOT or aorta documented on the most recent echocardiogram was utilized to stratify patients with left-sided obstructive lesions by severity of obstruction (none or mild, $\leq$2.9 m/*sec*; moderate or severe, $\geq$3 m/*sec*). Only patients with documented blood pressure, height, weight, body mass index (BMI), and echocardiographic data for LVM calculation at the time of their most recent echocardiogram were included.

2.2. Echocardiogram parameters and LVM

Echocardiographic measurements were taken from a parasternal short axis M-Mode acquisition at the level of the papillary muscles, a standard for LV quantification built into the echocardiogram protocol for all patients in our lab during the study period. Interventricular septum (IVSd) and left ventricular posterior wall (LVPWd) dimensions were measured at end-diastole. Left ventricular dimensions were taken at end-diastole (LVEDD) and end-systole (LVESD). LVM was determined via the formula proposed by Devereux and others: [15,16]

$$\text{LV mass} = 0.8 \times \left(1.04 \times \left[(\text{IVSd} + \text{LVEDD} + \text{LVPWd})^3 - \text{LVEDD}^3\right]\right) + 0.6\,\text{grams}$$

LVM was indexed to patient height to the power of 2.7 (LVMI-ht$^{2.7}$=LVM/height$^{2.7}$) to adjust for differences in body size [16,17]. LV systolic function was assessed by left ventricular shortening fraction (LVSF).

2.3. Blood pressure categorization

Blood pressures were obtained at the time of the echocardiogram. Patients undergoing echocardiogram evaluation in our outpatient clinic are brought to a quiet room where anthropometric data are collected by trained pediatric cardiology clinic staff. Height is measured using a stadiometer and weight is recorded via standard scale. Standard of care is to obtain a manual blood pressure in the right upper extremity. Oscillometric blood pressure measurements were used in a minority of cases where manual blood pressure measurements were unavailable. Patients were stratified by systolic blood pressure (SBP) into normotensive (NT, <120 mmHg), elevated blood pressure (E-BP, 120–129 mmHg), stage 1 HTN (HTN-1, 130–139 mmHg), and stage 2 HTN (HTN-2, $\geq$140 mmHg) per the 2017 Clinical Practice Guideline (CPG) for Screening and Management of Blood Pressure in Children and Adolescents [14].

2.4. LVH

Prevalence of LVH was reported in each group using the pediatric threshold for LVH of LVMI-ht$^{2.7} \geq 38.6$ g/m$^{2.7}$, which corresponds to the 95th percentile for children and adolescents and increases the sensitivity for detecting LVH before it reaches a level associated with CV events in adulthood [9]. LVH prevalence was also calculated using LVMI-ht$^{2.7} \geq$ 51 g/m$^{2.7}$, the adult threshold and current cutoff recommended for LVH in the pediatric 2017 Clinical Practice Guidelines [14].

2.5. BMI stratification

BMI percentiles were obtained from the Centers for Disease Control and Prevention growth charts [18]. BMI z-scores were calculated by BMIz = (BMI/M)L-1/(L*S), where L, M, and S are reference values from the growth chart for the corresponding child age and sex. BMI percentiles were calculated from z-scores based on the normal distribution. Subjects were categorized based on BMI percentile as normal weight (< 85th percentile), overweight (85th percentile to < 95th percentile), or obese ($\geq$ 95th percentile).

2.6. Statistical analysis

Data were summarized using frequency (percentage) for categorical variables, mean (standard deviation, SD) for continuous symmetric variables, and median (interquartile range, IQR) for continuous skewed variables. Data were quality checked and distributions were visualized using bar and violin plots. For model stability, race groups were aggregated into White, Black, or other (includes American Indian, Asian, Hispanic, Native American, Pacific Islander, biracial, other races, and unknown race). Prevalence of LVH among each blood pressure group was calculated. Among adolescents that met pediatric LVH criteria, the percentage reaching the adult LVH criteria was also calculated. Pre-planned comparisons were performed for the echocardiogram parameters between the normotensive group and each of E-BP, HTN-1, and HTN-2 groups using the Wilcoxon rank sum test. A baseline significance level of $\alpha = 0.05$ was adjusted for 27 pre-planned comparisons using a Bonferroni correction.

Univariable and multivariable linear regression models were implemented to derive estimated mean differences in LVMI-ht$^{2.7}$ based on specific exposures. Both the univariable and multivariable regression analyses were subset to patients with complete information on BMI, SBP, CHD lesion type, age, sex, and race. Primary exposures of interest were scaled for ease of interpretation – a one unit change represents a 10 % increase in BMI percentile or a 10 mm Hg increase in SBP. Univariable regression models are reported for primary exposures of interest (BMI and SBP). An individual multivariable regression model was run for each primary exposure (BMI and SBP). Multivariable regression models included CHD lesion type (categorical, 7 levels), age (continuous), sex (binary), and race (categorical, three levels) as covariates to account for potential confounding. Potential violations to model assumptions were evaluated visually using residuals plots. Metrics of model fit including residual standard error (RSE) and adjusted R^2 were calculated and reported for all developed models. To avoid the "table 2 Fallacy," we only reported effect estimates for our primary exposures. Ninety-five percent confidence intervals not including 0 were considered significant. All statistical analyses were performed in R version 4.0 (R Core Team, Vienna, Austria).

3. Results

3.1. Patient characteristics

A total of 853 adolescents with CHD (mean age 15.5 $\pm$ 1.5 years; range 13.0 to 17.9), 57.1 % male, and 78.5 % White met inclusion criteria (Table 1).

Table 1
Patient characteristics.

Characteristics	Total study population $n = 853$	Normotensive $n = 491$	Elevated $n = 214$	Stage 1 HTN $n = 99$	Stage 2 HTN $n = 49$
Age, y, mean (SD)	15.5 (1.5)	15.3 (1.5)	15.6 (1.5)	15.9 (1.2)	15.6 (1.4)
Female, n (%)	366 (42.9)	244 (49.7)	78 (36.4)	28 (28.3)	16 (32.7)
Race, n (%)					
White	670 (78.5)	390 (79.4)	162 (75.7)	80 (80.8)	38 (77.6)
Black	116 (13.6)	69 (14.1)	31 (14.5)	11 (11.1)	5 (10.2)
Other	67 (7.9)	32 (6.5)	21 (9.8)	8 (8.1)	6 (12.2)
Height, cm, mean (SD)	165 (13)	163 (13)	168 (13)	170 (12)	169 (11)
Body surface area, m^2, mean (SD)	1.71 (0.30)	1.62 (0.27)	1.78 (0.28)	1.87 (0.28)	1.96 (0.40)
Weight, kg*	61 [52, 73]	56 (48, 66)	66 (56, 78)	71 (61, 85)	82 (63, 100)
Body mass index, kg/m^2 *	22.0 [19.2, 25.9]	20.7 (18.5, 23.9)	23.0 (19.9, 26.8)	24.4 (21.2, 29.3)	27.3 (21.9, 32.6)
Body mass index, percentile*	68 [37, 92]	60 (29, 84)	78 (49, 94)	86 (59, 96)	95 (78, 98)
BMI category, n (%)					
Normal	571 (66.9)	377 (76.8)	129 (60.3)	48 (48.5)	17 (34.7)
Overweight	127 (14.9)	59 (12.0)	37 (17.3)	22 (22.2)	9 (18.4)
Obese	155 (18.2)	55 (11.2)	48 (22.4)	29 (29.3)	23 (46.9)
Systolic blood pressure, mm Hg*	117 [108, 126]	110 (104, 114)	124 (122, 126)	133 (131, 136)	146 (142, 152)
Diastolic blood pressure, mm Hg*	64 [59, 70]	62 (58, 68)	66 (60, 72)	67 (60, 76)	72 (64, 80)

* Presented as median (interquartile range).

BMI, BMI percentile, and body surface area (BSA) trended higher in the groups with abnormal blood pressure. Over half (57.6 %) of patients were normotensive, with the proportion of patients with E-BP, HTN-1, and HTN-2 25.1 %, 11.6 %, and 5.7 %, respectively. Breakdown of our cohort by CHD lesion is provided in Table 2.

Simple left-to-right shunts (29.8 %) and LVOT obstruction (29.1 %) were the most common lesions. HTN was more common in those with LVOT obstruction, with HTN being twice as prevalent in patients with ≥ moderate LVOT obstruction compared to those with less than moderate residual LVOT obstruction (45.2 % vs. 22.1 %). At least 12.6 % of patients in each CHD lesion group had HTN based on SBP.

Table 2
Percentage of population by CHD lesion.

CHD Lesion Type	$n = 853$	Hypertension	LVH
Left to Right Shunts	254 (29.8)	32/254 (12.6)	67/254 (26.4)
LVOT	248 (29.0)	62/248 (25.0)	82/248 (33.1)
≤2.9 m/sec^	217 (25.4)	48/217 (22.1)	67/217 (30.9)
≥3.0 m/sec^	31 (3.6)	14/31 (45.2)	15/31 (48.4)
PS	59 (6.9)	10/59 (16.9)	9/59 (15.3)
TOF	59 (6.9)	10/59 (16.9)	14/59 (23.7)
Conotruncal Anomalies	25 (2.9)	7/25 (28.0)	10/25 (40.0)
Other*	208 (24.4)	27/208 (13.0)	46/208 (22.1)

Abbreviations: CHD, congenital heart disease; LVH, left ventricular hypertrophy; LVOT, left ventricular outflow tract; PS. pulmonary stenosis; TOF, tetralogy of Fallot;.
Data are n (%).
* Other = Isolated atrioventricular valve disease, Ebstein's anomaly, coronary anomalies, single left ventricle disease.
^ Peak velocity through LVOT.

3.2. Echocardiogram parameters

LVEDD was higher in the E-BP and HTN-1 groups compared to the NT group (Table 3). LVPWd, IVSd, LVM, and LVMI-$ht^{2.7}$ were all significantly higher in all abnormal blood pressure groups compared to the NT group. Left ventricular systolic function was preserved across BP categories.

3.3. LVH

LVH was more common in patients with abnormal BP than normotensive patients (36.5 % vs. 19.6 %, Table 4.). Nearly one-third of patients (32.3 %) with HTN-1 and 40.8 % of patients with HTN-2 had LVH. Further, of those patients with LVH based on the pediatric LVMI-$ht^{2.7}$ threshold, one-fifth (21.5 %) also met the adult LVH threshold associated with increased CV risk (LVMI-$ht^{2.7} \geq 51\ g/m^{2.7}$). Although most patients diagnosed with LVOT obstruction had no residual or only mild obstruction (87.5 %), patients with at least moderate LVOT obstruction had the highest prevalence of LVH (48.4 %) amongst all CHD groups (Table 2) followed by those with conotruncal anomalies (40 %), no or mild LVOT obstruction (30.9 %), and tetralogy of Fallot (23.7 %).

3.4. Univariable and multivariable linear regression models

Regression models showed low explained variation (adjusted R^2) and high residual standard error (RSE). This held true for both univariable models (BMI: $R^2 = 0.13$, RSE = 9.84, SBP: $R^2 = 0.03$, RSE = 10.36) and multivariable models (BMI: $R^2 = 0.19$, RSE = 9.47, SBP: $R^2 = 0.08$, RSE = 10.08). Despite this, effect estimates showed that, on average, for increases in BMI or SBP, a significant increase in LVM was seen in each model. A 10 % increase in BMI percentile was associated with an average 1.2 $g/m^{2.7}$ increase in LVMI-$ht^{2.7}$ after adjusting for CHD lesion type, age, sex, and race (Fig. 1, Supplementary Table 1.). A 10 mmHg SBP increase was associated with an average 1.4 $g/m^{2.7}$ increase in LVMI-$ht^{2.7}$, which remained significant after adjustment (0.93 $g/m^{2.7}$). While the effect estimate percent changes were larger for SBP versus BMI as the primary exposure, results remained statistically significant (Supplementary Table 2.). In our data, there was a difference in SBP and LVMI-$ht^{2.7}$ amongst CHD lesion types with those with moderate residual LVOT obstruction having the highest median LVMI-$ht^{2.7}$ and SBP (Supplementary Table 3.). However, when assessing interaction terms between CHD lesion type and SBP, the magnitude of association between SBP and LVMI-$ht^{2.7}$ did not significantly vary by CHD type after controlling for other factors. Absolute LVMI-$ht^{2.7}$ and SBP values were higher in males than females ($p < 0.001$, Supplementary Table 4). However, there was no significant difference in the magnitude of association between LVMI-$ht^{2.7}$ and SBP by sex when assessing a SBP/sex interaction term and controlling for other factors (age, CHD diagnosis, race). In addition, precision of estimates between models with and without subjects with at least moderate LVOT obstruction were similar (% change in width of 95 % confidence intervals ≤2.4 %).

4. Discussion

4.1. HTN

Our analysis of 853 adolescents with CHD raises concerns about risk of future cardiovascular morbidity in this population. The proportion of our cohort having abnormal blood pressure is higher than that previously reported for youths without CHD [19]. Patients with CHD often have several risk factors for HTN including chronic renal insufficiency and changes in arterial wall architecture [20,21]. Indeed, Mivelaz et al.

Table 3
Comparison of echocardiogram parameters by blood pressure category adjusted using Bonferroni correction.

Characteristic	Total Study Population $n = 853$	Normotensive $n = 491$	Elevated $n = 214$	Stage 1 HTN $n = 99$	Stage 2 HTN $n = 49$	p-value
LVEDD, cm	4.80 (4.40, 5.20)	4.70 (4.30, 5.10)	4.88 (4.40, 5.30)	5.00 (4.70, 5.38)	4.80 (4.50, 5.20)	‡, §
LVESD, cm	3.00 (2.70, 3.30)	2.96 (2.60, 3.28)	3.09 (2.79, 3.38)	3.00 (2.78, 3.30)	2.97 (2.70, 3.30)	#
LVPWd, cm	0.80 (0.70, 0.90)	0.78 (0.70, 0.85)	0.81 (0.74, 0.90)	0.85 (0.75, 0.94)	0.90 (0.80, 1.00)	‡, §, ||
IVSd, cm	0.80 (0.70, 0.90)	0.80 (0.70, 0.90)	0.90 (0.72, 0.96)	0.90 (0.80, 0.95)	1.00 (0.80, 1.06)	‡, §, ||
LVM	128 (99, 162)	115 (94, 145)	140 (111, 175)	147 (122, 183)	156 (132, 181)	‡, §, ||
LVM indexed to BSA	75 (63, 90)	71 (61, 86)	80 (65, 94)	78 (67, 91)	78 (66, 92)	‡
LVMI $ht^{2.7}$, $g/m^{2.7}$	33 (27, 39)	31 (26, 37)	35 (28, 43)	35 (29, 41)	36 (32, 44)	‡, §, ||
LVSF, %	36.7 (33.3, 41.2)	36.4 (33.0, 40.7)	36.2 (33.3, 41.2)	39.3 (35.7, 42.9)	39.0 (35.1, 43.3)	§
RWT	0.34 (0.30, 0.38)	0.33 (0.29, 0.38)	0.34 (0.31, 0.39)	0.34 (0.30, 0.38)	0.37 (0.35, 0.41)	||

Data are median (IQR).
Abbreviations: HTN, hypertension; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVPWd, left ventricular posterior wall in end diastole; IVSd, interventricular septum in end diastole; LVM, left ventricular mass; BSA, body surface area; LVMI $ht^{2.7}$, left ventricular mass index to height to the power of 2.7; LVSF, left ventricular shortening fraction; RWT, relative wall thickness.
‡ = $p <$ Bonferroni-adjusted α when NT compared to E-BP.
§ = $p <$ Bonferroni-adjusted α when NT compared to HTN-1.
|| = $p <$ Bonferroni-adjusted α when NT compared to HTN-2.
= $p \geq$ Bonferroni-adjusted α when NT compared to BP categories.

Table 4
Prevalence of LVH by blood pressure category.

Characteristic	Normotensive $n = 491$	Elevated $n = 214$	Stage 1 HTN $n = 99$	Stage 2 HTN $n = 49$
LVH, n (%)	96 (19.6)	80 (37.4)	32 (32.3)	20 (40.8)

Abbreviations: HTN, hypertension; LVH, left ventricular hypertrophy.

[22] demonstrated elevated aortic stiffness in a similar cohort of adolescents with biventricular CHD, which is a vascular marker linked to future CV morbidity and mortality [23]. In our cohort, SBP was associated with cardiac hypertensive end-organ changes such that for each 10 mmHg increase in SBP, a significant increase in LVMI-$ht^{2.7}$ was observed, even after correction for potential confounders such as CHD lesion type. As hypertensive youths are prone to becoming adults with HTN [11–13], identification of abnormal blood pressure in adolescents with CHD may play an important prognostic role in the long-term care of adults with congenital heart disease.

4.2. LVH

LVM was higher in subjects with higher blood pressure which is consistent with previous findings in children and adolescents without CHD [24]. However, one-fifth of our patients with LVH met the adult threshold associated with increased CV risk which is two and a half times higher than levels previously reported for hypertensive youths without CHD, [10] suggesting a compounding effect of underlying CHD. Patients with CHD may have peripheral vascular changes secondary to intrinsic vascular alterations or postoperative effects which may influence ventricular remodeling to favor hypertrophy as compared to adolescents without CHD [20–22]. Residual LVOT lesions comprised the largest proportion of patients with LVH; however, we found that even those with mild residual LVOT gradients and those with history of a conotruncal anomaly had LVH at rates higher than hypertensive youths without CHD [10]. Although our study included patients with left heart outflow obstructions, we adjusted for CHD lesion type in our regression models. Additionally, the majority of documented LVOT gradients on the echocardiograms used for this study demonstrated no or mild residual pressure gradient, indicating that a residual obstruction is less likely to be responsible for any observed increase in LVM. We performed a sensitivity analysis for our univariable and multivariable regression models after excluding patients with moderate or worse LVOT gradient then regenerated model coefficients for the primary exposures of interest. Although the effect estimates changed for both BMI and SBP, results remained statistically significant (Supplementary Table 1.)

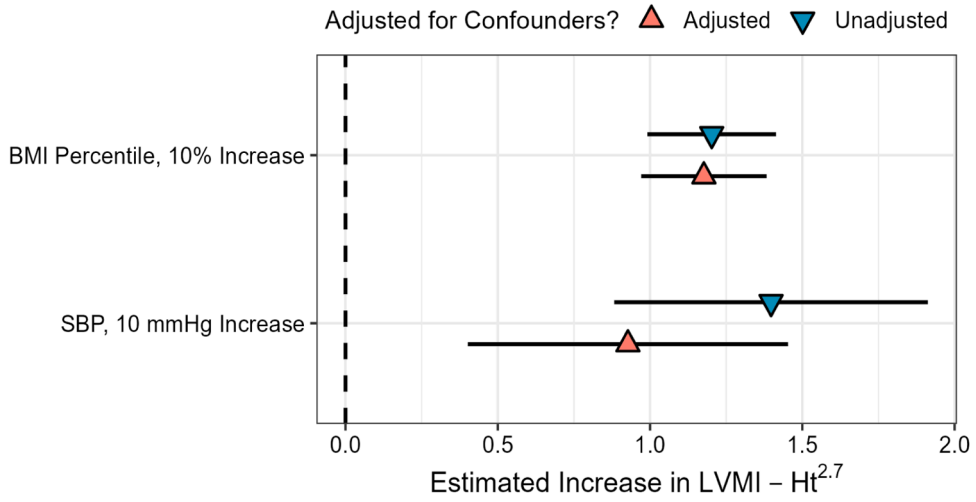


Fig. 1. Effects of BMI and SBP on LVMI-$ht^{2.7}$. Confounders adjusted for include CHD lesion type, age, sex, and race. Vertical line indicates no effect. Horizontal lines are 95 % confidence intervals.

4.3. BMI

Weight continues to be proven a crucial factor in the development of LVH. The current study of adolescents with CHD is consistent with prior findings in hypertensive children without CHD showing that those overweight or obese had increased LVM compared to those with healthy weight [25]. In our multivariable analysis, BMI percentile remained significantly associated with LVM. In our data, higher BMI and SBP were associated with a significant increase in LVMI-ht$^{2.7}$; however, a greater increase in LVMI-ht$^{2.7}$ was seen with increasing BMI versus SBP. Increased body mass and hypertension are associated with cardiomyocyte adaptations. High blood pressure increases afterload and leads to compensatory concentric hypertrophy in order to reduce wall tension [26]. Through interactions between the autonomic nervous system, inflammatory cascade, and metabolic systems, obesity drives left ventricular hypertrophy through an increase in stroke volume [26]. Perhaps those with CHD are more susceptible to these hemodynamic alterations, particularly a volume-loaded state, leading to a more pronounced LVH response to body mass versus afterload. CHD may be associated with increased risk of cardiovascular disease throughout the lifespan [27]. Our study emphasizes the importance of screening adolescents with CHD for additional factors associated with increased LVM to enable clinicians to address these risk factors to mitigate the development of LVH. Body size may be a modifiable risk factor for subclinical CV disease.

Our study was novel in having a large cohort of patients inclusive of a broad spectrum of CHD and in applying the most recent clinical practice guidelines for the assessment of blood pressure in adolescents. With the knowledge of early abnormal blood pressure and evidence of increased LVM in patients with CHD, it becomes vital to screen and consider intervention in patients who meet indication. Targeted diet and lifestyle recommendations such as the Dietary Approaches to Stop Hypertension (DASH) [14] and the Cardiovascular Health Integrated Lifestyle Diet (CHILD) recommended by the American Academy of Pediatrics [28] should be stressed particularly in this patient population. Physical activity recommendations should also be emphasized [29] depending on appropriate activity clearance for the heart lesion. Indeed, fear or apprehension may oftentimes lead to restriction in physical activity or sports participation in patients with CHD. However, literature supports that activity promotion is in fact safe for most patients and can lead to improvements in several markers of cardiometabolic health, including BMI [30,31]. Ambulatory blood pressure monitoring is already recommended for patients with history of coarctation of the aorta [14] but its use may warrant consideration in the broader adolescent CHD population to screen for underlying blood pressure abnormalities. This is especially important considering evidence that regression in LVM leads to a lesser risk of CV disease [8].

4.4. Limitations & conclusions

Our single-center retrospective cross-sectional study is one of the largest to describe BP and LVM in the pediatric CHD population, yet there are several limitations. Due to the study design, we are unable to determine causation for increased LVM. Individuals included in our cohort were predominantly White which is not representative of the global adolescent CHD population. Significant intracardiac shunting may impact ventricular geometry but given the ages studied, we assumed most patients had undergone repair of their CHD lesion and would not continue to exhibit significant sequelae of their original lesion. However, patients with minor shunting lesions or valvular regurgitation not meeting indication for intervention were likely included. Quantification of LVM may be more technically challenging to obtain in those with CHD since standard formulas used to calculate LVM by echocardiogram rely on a left ventricular shape without significant distortion. Our echocardiogram protocol includes M-mode acquisition for all studies regardless of indication and is performed by sonographers certified in pediatric echocardiography which may reduce this risk. In addition, BMI does not comprehensively differentiate muscle mass from fat distribution and is not the best marker for visceral adiposity which has been shown to correlate better with CV risk factors. The addition of other markers of central adiposity (i.e. waist circumference, waist-to-hip ratio) in future studies would prove useful. Whether patients were diagnosed as hypertensive was not investigated and information on active medications at the time of echocardiogram was not included. However, blood pressure management by anti-hypertensive therapy could have predisposed our findings to non-significance if patients were compliant with their prescriptions. Therefore, our findings may indicate inadequate control of blood pressure through medication or an underappreciation of hypertension in this group, each of which would warrant further investigation. Serial blood pressure evaluation and incorporation of ambulatory blood pressure monitoring data would further enhance our understanding of the true prevalence of HTN in this cohort and provide insight into the timing of development of end-organ changes. Despite the possibility of misclassification of blood pressure category due to the single time point measurement, we saw a difference in echocardiographic parameters between blood pressure groups, suggesting end-organ damage was in fact already present. Future studies should be aimed at assessing longitudinal changes in blood pressure and LVH development in youths with CHD and the impact of pharmacological intervention and social determinants on HTN, obesity, and cardiac dysfunction in the pediatric CHD population.

Author agreement

All authors have seen and approved the final version of the manuscript being submitted. This manuscript is the authors' original work, hasn't received prior publication, and isn't under consideration for publication elsewhere

CRediT authorship contribution statement

Aaron T Walsh: Writing – review & editing, Writing – original draft, Methodology, Data curation, Conceptualization. **Kan N Hor:** Writing – review & editing, Supervision, Methodology, Data curation, Conceptualization. **Mariah Eisner:** Writing – review & editing, Methodology, Formal analysis. **Chance Alvarado:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis. **Mahmoud Kallash:** Writing – review & editing. **John David Spencer:** Writing – review & editing. **Andrew H Tran:** Writing – review & editing, Supervision, Methodology, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary materials

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