ORIGINAL ARTICLE

Measurement of Central Aortic Blood Pressure in Youth: Role of Obesity and Sex

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BACKGROUND
The relationship between pediatric severe obesity (SO) and central aortic blood pressure (BP) has yet to be established.

METHODS
We conducted a cross-sectional study of 348 youth (48.5% male, age 12.7 ± 0.1 years) with a wide range of body mass index (BMI) values: normal weight (NW; ≥5th and <85th BMI percentiles), overweight/obesity (OW/OB; 85th to <120% of the 95th BMI percentile), and SO (≥120% of the 95th BMI percentile). Measures of central aortic BP were obtained via applanation tonometry with SphygmoCor MM3 software.

RESULTS
After adjustment for covariates, no significant sex differences were observed for radial–aortic systolic blood pressure (SBP) (P = 0.39), carotid–aortic SBP (P = 0.99), radial–aortic diastolic blood pressure (DBP) (P = 0.44), and carotid–aortic DBP (P = 0.53). Compared to youth with NW, youth with SO exhibited higher radial–aortic SBP (SO vs. NW: 102 ± 1 mm Hg vs. 90 ± 1 mm Hg, P<0.001), carotid–aortic SBP (SO vs. NW: 121 ± 1 mm Hg vs. 109 ± 1 mm Hg, P<0.001), and carotid–aortic DBP (SO vs. NW: 60 ± 1 mm Hg vs. 56 ± 1 mm Hg, P = 0.04). Compared to youth with OW/OB, youth with SO had higher radial–aortic SBP (OW/OB: 97 ± 1 mm Hg, P = 0.002) and carotid–aortic SBP (OW/OB: 114 ± 1 mm Hg, P = 0.007). After adjusting for either total-body percent fat mass or visceral adipose tissue, BMI was still a significant predictor of both radial–aortic and carotid–aortic SBP and DBP (P<0.001, all).

CONCLUSIONS
In a cohort of youth with a wide range of adiposity levels, central aortic BP was elevated among individuals with SO and associated with BMI but not body fatness.

Keywords: blood pressure; body mass index; cardiovascular; central blood pressure; hypertension; pediatrics; severe obesity.

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The continued increase in the prevalence of severe obesity (SO; 120% of the 95th body mass index [BMI] percentile) among youth,[1] particularly among adolescents,[2] is a major medical and public health challenge.[3] Understanding the impact of SO on risk factors for chronic disease, particularly cardiovascular disease (CVD), will be important to help guide screening and treatment efforts. The prevalence of many CVD risk factors (e.g., hypertension, dyslipidemia, and prediabetes) and the subclinical manifestation of CVD (e.g., vessel wall thickness and left ventricular mass) are higher among youth with SO than their peers with normal weight (NW), overweight (OW), or obesity (OB).[3,4]

The association of OB with peripherally measured blood pressure (BP) among youth has been well documented.[5–10] Although hypertension is clinically diagnosed by brachial artery sphygmomanometry,[11,12] emerging data suggest that central aortic BP may be more strongly associated with vascular damage, atherosclerosis, and future CVD risk.[13–17] Central aortic BP within the ascending aorta can be directly measured using a pressure transducer introduced into the aortic root at the time of cardiac catheterization.[18] Recently, noninvasive techniques that derive central aortic pressures via applanation tonometry have been developed. However, limited data of central aortic BP exist among pediatric populations particularly in the context of SO, as this research has been predominately conducted among older adults.[19,20] Moreover, though sex differences have been established in adults,[21] no data are present among youth using applanation tonometry.

Therefore, the purpose of this study was to characterize the role of SO and sex in relation to central aortic BP among children and adolescents. We hypothesized that youth with SO would exhibit higher central aortic BP compared with moderate OB, OW, and NW and that males would have higher central aortic BP compared with females.

METHODS

Data were obtained from 348 youth (169 males) between the ages of 8 and 18 years who participated in a cross-sectional study assessing CVD risk among children and

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Table 1. Cohort demographics and anthropometric characteristics

	Males (*n* = 169)	Females (*n* = 179)	*P* value	Normal weight (*n* = 136)	Overweight/obesity (*n* = 96)	Severe obesity (*n* = 116)	*P* value
Age (years)	12.7 ± 0.2	12.7 ± 0.2	0.983	12.4 ± 0.2	12.5 ± 0.3	13.4 ± 0.3[a,b]	0.007
Race [*n* (%)]			0.241				0.081
White	134 (79.3)	139 (77.7)		117 (86.0)	75 (78.1)	84 (72.4)	
Black	18 (10.6)	28 (15.6)		12 (8.8)	13 (13.5)	20 (17.2)	
Other	17 (10.1)	12 (6.7)		7 (5.1)	8 (8.3)	12 (10.3)	
Tanner stage [*n* (%)]			0.059				0.002
I	61 (36.1)	38 (21.2)		55 (40.4)	26 (27.1)	18 (15.5)	
II	34 (20.1)	33 (18.4)		21 (15.4)	20 (20.8)	28 (24.1)	
III	24 (14.2)	34 (18.9)		21 (15.4)	20 (20.8)	19 (16.4)	
IV	29 (17.2)	38 (21.2)		26 (19.1)	16 (16.7)	27 (23.3)	
V	21 (12.4)	27 (15.1)		13 (9.6)	14 (14.7)	24 (20.7)	
Body mass (kg)	67.4 ± 2.5	67.9 ± 2.0	0.884	44.9 ± 1.4	65.3 ± 1.7[a]	96.7 ± 2.6[a,b]	<0.001
Height (cm)	158.4 ± 1.2	154.7 ± 0.9	0.015	152.9 ± 1.3	156.2 ± 1.4	161.2 ± 1.2[a,b]	<0.001
BMI (kg/m^2)	25.4 ± 0.7	27.6 ± 0.7	0.018	18.3 ± 0.2	26.3 ± 0.4[a]	36.4 ± 0.6[a,b]	<0.001
BMI percentile (%)	74.8 ± 2.2	80.7 ± 2.1	0.051	48.2 ± 2.0	94.4 ± 0.8[a]	99.1 ± 0.05[a,b]	<0.001
Hip circumference (cm)	92.2 ± 1.4	96.4 ± 1.4	0.038	79.1 ± 0.8	94.1 ± 1.1[a]	113.1 ± 1.4[a,b]	<0.001
Waist circumference (cm)	79.4 ± 1.6	80.4 ± 1.3	0.501	63.0 ± 0.6	80.1 ± 0.9[a]	101.2 ± 1.4[a,b]	<0.001
Lean muscle mass (kg)	40.7 ± 1.2	37.2 ± 0.8	0.017	31.7 ± 0.9	38.2 ± 1.1[a]	48.2 ± 1.3[a,b]	<0.001
Fat mass (kg)	23.6 ± 1.4	28.1 ± 1.3	0.018	10.6 ± 0.3	24.8 ± 0.8[a]	45.4 ± 1.4[a,b]	<0.001
Percent fat mass (%)	33.0 ± 0.9	40.2 ± 0.7	<0.001	25.2 ± 0.5	39.3 ± 0.7[a]	48.2 ± 0.5[a,b]	<0.001
Visceral adipose tissue (g)	567.7 ± 58.6	586.9 ± 45.8	0.801	76.0 ± 6.1	412.5 ± 30.6[a]	1103.9 ± 59.2[a,b]	<0.001
Hypertension status [*n* (%)]			0.304				<0.001
Normal blood pressure	125 (73.9)	122 (68.2)		125 (91.9)	71 (73.9)	51 (44.0)	
Elevated blood pressure	19 (11.2)	33 (18.4)		10 (7.3)	15 (15.6)	26 (21.6)	
Stage 1 hypertension	19 (11.2)	18 (10.1)		1 (0.7)	9 (9.4)	27 (23.3)	
Stage 2 hypertension	6 (3.5)	6 (3.4)		0 (0)	1 (1.0)	12 (10.3)	
Heart rate (bpm)	67 ± 1	71 ± 1	0.001	71 ± 1	76 ± 1[a]	77 ± 1[a]	<0.001

Obesity status is presented as normal weight (e.g., ≥5th to <85th BMI percentile), overweight/obesity (e.g., ≥85th to <95th BMI percentile, ≥100% to <120% of the 95th BMI percentile), and severe obesity (e.g., ≥120% of the 95th BMI percentile). Continuous variables are presented as mean ± SE. Race, Tanner stage, and hypertension status are presented as count (% within column). Analysis of variance and chi-squared test assessed sex differences in continuous and categorical variables, respectively. Abbreviation: BMI, body mass index.

[a]Significantly different compared with normal weight as determined by Bonferroni post hoc comparisons ($P < 0.05$).

[b]Significantly different compared with overweight/obesity as determined by Bonferroni post hoc comparisons ($P < 0.05$).

adolescents throughout a range of BMI values. Participants were recruited from the University of Minnesota Masonic Children's Hospital Pediatric Weight Management Clinic, general pediatric clinics, and the community within the Minneapolis–St. Paul metropolitan area. Exclusion criteria included OB due to a genetic cause, bariatric surgery, current use of antihypertensive medications, type 1 diabetes mellitus, history of hypercholesterolemia, chronic kidney disease, Kawasaki disease, autoimmune inflammatory diseases, and congenital heart disease. This study was approved by the institutional review board at the University of Minnesota. All procedures were followed in accordance with the institutional review board and Health Insurance Portability and Accountability Act (HIPAA) guidelines. Parents and participants provided informed consent and assent, respectively.

Anthropometric measurements

All testing was performed at the University of Minnesota, with the participants fasted for at least 12 hours before their study visit. Height and body mass were measured 3 times on the same day using a wall-mounted stadiometer and an electric scale (ST Scale-Tronix, White Plains, NY), and the average was recorded. BMI was calculated using body mass in kilograms (kg) divided by height in squared meters (m^2). OB status was determined by BMI percentiles and stratified into 3 categories: NW (i.e., ≥5th to <85th BMI percentile), OW/OB (i.e., ≥85th to <95th BMI

percentiles, ≥100% to <120% of the 95th BMI percentiles), and SO (i.e., ≥120% of the 95th BMI percentile or absolute BMI > 35 kg/m^2). Body composition was measured using dual-energy X-ray absorptiometry (iDXA; General Electronic Medical Systems, Madison WI) and data were analyzed using Encore software version 16.0. Trained medical providers determined pubertal maturation using Tanner stages (I–V).

Brachial BP and heart rate

Seated brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) were obtained on the right arm after participants rested for 10 minutes with legs uncrossed via an automated sphygmomanometer (Colin BP-8800; Colin Medical Instruments, San Antonio TX). BP was measured in triplicate with 3-minute intervals between each measure. The average of the last 2 measures was recorded. Brachial BP percentile was calculated based on age, sex, and height. Hypertension status was determined by using the current recommendations from the American Academy of Pediatrics and stratified into the following categories: normal BP, elevated BP, stage 1 hypertension, and stage 2 hypertension.[22]

Measurement of aortic BP and arterial stiffness

Following 15 minutes of supine rest, radial and carotid arterial waveforms were recorded via applanation tonometry using SphygmoCor MM3 software (AtCor Medical, Sydney Australia). The tonometer was positioned over the strongest pulse point on the artery, and a minimum of 11 seconds of consistent arterial waveforms were recorded after a strong and reproducible pulse was obtained. Collected waveforms were calibrated and scaled using each participant's resting brachial BP, and a validated generalized transfer function estimated the corresponding central aortic BP.

Statistical analysis

SPSS Statistics 23, released 2016 (IBM, IBM SPSS Statistics for Windows, Armonk, NY) was used for statistical analysis. A 1-way analysis of variance (ANOVA) and a chi-square test assessed sex differences in clinical and demographic characteristics (e.g., race and Tanner stage), respectively. Sex- and OB-related differences regarding central aortic BP were evaluated by a 1-way ANOVA, and an analysis of covariance adjusted for Tanner stage, age, race, sex, height, and BMI percentile. A 2-way multivariate ANOVA, with adjustment for Tanner stage, age, race, and height, tested for an interaction between OB status and sex. Multiple linear regression evaluated the association of Tanner stage, sex, race, BMI percentile, percent fat mass, and visceral adipose tissue with measures of central aortic BP. Adjusted Pearson correlation coefficients measured the strength of the association between anthropometric and body composition measures with central aortic BP.

RESULTS

Cohort demographics and clinical characteristics are presented in Table 1. Compared with males, females were significantly shorter ($P = 0.02$) and had a higher BMI ($P = 0.02$), BMI percentile ($P = 0.05$), hip circumference ($P = 0.04$), fat mass ($P = 0.02$), and percent fat mass ($P < 0.001$). There were no significant sex differences regarding race ($P = 0.21$), Tanner stage ($P = 0.06$), and hypertension status ($P = 0.30$). Subjects with SO were significantly older compared to subjects with NW ($P = 0.01$) and OW/OB ($P = 0.05$). Compared to subjects with NW, subjects with SO had higher hip circumference ($P < 0.001$), waist circumferences ($P < 0.001$), lean muscle mass ($P < 0.001$), fat mass ($P < 0.001$), percent fat mass ($P < 0.001$), and visceral adipose tissue ($P < 0.001$). Subjects with SO also had significantly greater hip circumference ($P < 0.001$), waist circumference ($P < 0.001$), lean muscle mass ($P < 0.001$), fat mass ($P < 0.001$), percent fat mass ($P < 0.001$), and visceral adipose tissue ($P < 0.001$) compared to subjects with OW/OB. Race did not differ by OB status ($P = 0.08$). Higher Tanner stages were associated with increased OB status ($P = 0.002$), and the proportion of subjects with hypertension was greater among subjects with SO ($P < 0.001$).

Table 2 presents sex differences with both peripheral and central aortic BP. Unadjusted radial–aortic SBP ($P = 0.04$)

Table 2. Peripheral and central aortic blood pressure among males and females

	Males (n = 169)	Females (n = 179)	P value
Brachial systolic blood pressure (mm Hg)			
Unadjusted	112 (112–113)	114 (112–115)	0.215
Adjusted[a]	113 (111–116)	115 (112–117)	0.432
Brachial diastolic blood pressure (mm Hg)			
Unadjusted	58 (56–59)	60 (59–61)	0.029
Adjusted[a]	59 (57–61)	60 (58–62)	0.326
Brachial systolic blood pressure percentile (%)			
Unadjusted	54 (50.5–59.5)	64.9 (60.9–68.9)	0.001
Adjusted[a]	61.8 (56.3–67.3)	66.5 (61.2–71.8)	0.242
Brachial diastolic blood pressure percentile (%)			
Unadjusted	35.7 (32.2–39.5)	38.4 (35.2–41.7)	0.255
Adjusted[a]	39.7 (35.0–44.5)	39.1 (34.5–43.7)	0.862
Radial–aortic systolic blood pressure (mm Hg)			
Unadjusted	94 (91–96)	97 (95–99)	0.041
Adjusted[a]	96 (94–98)	97 (95–99)	0.385
Radial–aortic diastolic blood pressure (mm Hg)			
Unadjusted	58 (57–60)	61 (59–63)	0.032
Adjusted[a]	59 (58–61)	60.7 (59–62)	0.439
Carotid–aortic systolic blood pressure (mm Hg)			
Unadjusted	114 (112–117)	115 (112–118)	0.653
Adjusted[a]	115 (112–117)	115 (112–117)	0.988
Carotid–aortic diastolic blood pressure (mm Hg)			
Unadjusted	58 (56–60)	59 (57–61)	0.366
Adjusted[a]	58 (57–60)	59 (57–61)	0.534

Reported as mean with 95% confidence interval.
[a]Adjustment for Tanner stage, race, body mass index percentile, age, and height by an analysis of covariance.

Table 3. Radial and carotid aortic blood pressure by obesity status

	Normal weight (*n* = 136)	Overweight/obesity (*n* = 96)	Severe obesity (*n* = 116)	*P* value
Brachial systolic blood pressure (mm Hg)				
Unadjusted	105 (104–107)	113 (110–115)[b]	121 (119–124)[b,c]	<0.001
Adjusted[a]	108 (105–111)	113 (110–115)[b]	121 (119–124)[b,c]	<0.001
Brachial diastolic blood pressure (mm Hg)				
Unadjusted	56 (55–58)	58 (57–60)	62 (61–63)[b,c]	<0.001
Adjusted[a]	58 (56–60)	59 (57–61)	61 (60–63)	0.054
Brachial systolic blood pressure percentile (%)				
Unadjusted	44.2 (40.0–48.4)	62.2 (57.0–67.5)[b]	76.8 (72.2–81.4)[b,c]	<0.001
Adjusted[a]	47.8 (40.8–54.8)	63.6 (57.4–69.8)[b]	79.2 (72.8–85.7)[b,c]	<0.001
Brachial systolic blood pressure percentile (%)				
Unadjusted	32.3 (28.7–36.1)	36.8 (32.4–41.2)	42.8 (38.6–47.0)[b]	0.001
Adjusted[a]	32.8 (26.6–39.0)	40.3 (34.8–45.8)	44.3 (38.6–50.0)[b]	0.036
Radial−aortic systolic blood pressure (mm Hg)				
Unadjusted	89 (87–92)	94 (91–97)[b]	102 (99–105)[b,c]	<0.001
Adjusted[a]	90 (87–92)	97 (95–99)[b]	102 (100–105)[b,c]	<0.001
Radial−aortic diastolic blood pressure (mm Hg)				
Unadjusted	57 (55–58)	59 (57–61)[b]	63 (61–65)[b,c]	<0.001
Adjusted[a]	58 (56–60)	60 (58–62)	61 (59–63)	0.088
Carotid−aortic systolic blood pressure (mm Hg)				
Unadjusted	108 (105–111)	114 (111–117)	122 (118–125)[b,c]	<0.001
Adjusted[a]	109 (106–112)	114 (111–117)	121 (118–124)[b,c]	<0.001
Carotid−aortic diastolic blood pressure (mm Hg)				
Unadjusted	56 (54–58)	59 (56–61)	61 (59–63)[b]	0.005
Adjusted[a]	56 (54–59)	59 (57–61)	60 (58–62)[b]	0.047

Reported as mean with 95% confidence interval. Obesity status is presented in three categories: normal weight (e.g., ≥5th to <85th BMI percentile), overweight/obesity (e.g., ≥85th to <95th BMI percentile, ≥100% to <120% of the 95th BMI percentile), and severe obesity (e.g., ≥120% of the 95th BMI percentile). Abbreviation: BMI, body mass index.

[a]Adjustment for Tanner stage, sex, race, age, and height by an analysis of covariance.

[b]Significantly different compared with normal weight as determined by Bonferonni post hoc pairwise comparisons ($P < 0.05$).

[c]Significantly different compared with overweight/obesity as determined by Bonferonni post hoc pairwise comparisons ($P < 0.05$).

and radial−aortic DBP ($P = 0.03$) were significantly higher among females. After adjusting for covariates, radial−aortic SBP ($P = 0.39$) and radial−aortic DBP ($P = 0.44$) were both not significantly different between males and females. No differences between sexes were found for brachial SBP ($P = 0.43$), brachial DBP ($P = 0.33$), carotid−aortic SBP ($P = 0.99$), and carotid−aortic DBP ($P = 0.53$).

Table 3 displays peripheral and central aortic BP stratified by OB status. Compared to NW, SO participants had higher unadjusted radial−aortic SBP ($P < 0.001$), radial−aortic DBP ($P < 0.001$), carotid−aortic SBP ($P < 0.001$), and carotid−aortic DBP ($P = 0.004$). After adjusting for covariates, radial−aortic SBP ($P < 0.001$), carotid−aortic SBP ($P < 0.001$), and carotid−aortic DBP ($P = 0.04$) remained higher among SO participants. Compared to OW/OB, SO participants had higher unadjusted radial−aortic SBP ($P = 0.04$), radial−aortic DBP ($P = 0.03$), and carotid−aortic SBP ($P = 0.001$). Following adjustment, participants with SO consistently had higher radial−aortic SBP ($P = 0.002$) and carotid−aortic SBP ($P = 0.007$) compared to participants with OW/OB. Results from a 2-way multivariate ANOVA depicted a nonsignificant interaction between OB status and sex regarding radial−aortic SBP ($P = 0.99$), radial−aortic DBP ($P = 0.69$), carotid−aortic SBP ($P = 0.74$), and carotid−aortic DBP ($P = 0.67$).

Regression analysis on radial−aortic BP is presented in Table 4. After adjusting for Tanner stage, sex, race, and percent fat mass, BMI was significantly associated with both radial−aortic SBP ($\beta = 0.9$, $P < 0.001$) and DBP ($\beta = 0.5$, $P < 0.001$). Supplementary Data present regression analysis on carotid−aortic SBP and DBP, in which BMI was similarly significantly associated with carotid−aortic SBP ($\beta = 0.8$, $P < 0.001$) and DBP ($\beta = 0.4$, $P < 0.001$). Percent fat mass was not associated with radial−aortic SBP ($\beta = -0.1$, $P = 0.38$) and DBP ($\beta = -0.1$, $P = 0.21$) (Table 4), carotid−aortic SBP ($\beta = -0.07$, $P = 0.68$), or DBP ($\beta = -0.1$, $P = 0.64$) (Supplementary Table 1). Visceral adipose tissue was only significantly associated with carotid−aortic SBP ($\beta = 0.006$, $P = 0.05$). With the inclusion of both age and Tanner stage

Table 4. Multiple linear regression of radial aortic blood pressure measurements

	R^2	β	SE	*P* value		R^2	β	SE	*P* value
Radial–aortic systolic blood pressure—Model 1					**Radial–aortic diastolic blood pressure—Model 1**				
	0.356			<0.001		0.199			<0.001
Tanner stage (I–V)		1.6	0.5	0.003	Tanner stage (I–V)		1.1	0.4	0.010
Male sex		−1.2	1.4	0.391	Male sex		−1.5	1.1	0.192
Race		−0.5	1.1	0.684	Race		0.2	0.9	0.824
BMI (kg/m^2)		0.7	0.09	<0.001	BMI (kg/m^2)		0.4	0.07	<0.001
Radial–aortic systolic blood pressure—Model 2					Radial–aortic diastolic blood pressure—Model 2				
	0.358			<0.001		0.205			<0.001
Tanner stage (I–V)		1.2	0.6	0.071	Tanner stage (I–V)		0.7	0.5	0.193
Male sex		−1.7	1.5	0.258	Male sex		−2.2	1.2	0.078
Race		−0.5	1.1	0.639	Race		0.2	0.9	0.856
BMI (kg/m^2)		0.9	0.2	<0.001	BMI (kg/m^2)		0.5	0.2	<0.001
Percent fat mass		−0.1	0.1	0.379	Percent fat mass		−0.1	0.1	0.214
Radial–aortic systolic blood pressure—Model 3					Radial–aortic diastolic blood pressure—Model 3				
	0.364			<0.001		0.211			<0.001
Tanner stage (I–V)		1.7	0.5	0.002	Tanner stage (I–V)		1.3	0.4	0.007
Male sex		−1.9	1.5	0.206	Male sex		−2.0	1.2	0.107
Race		−0.3	1.1	0.811	Race		0.4	0.9	0.652
BMI (kg/m^2)		0.6	0.2	0.004	BMI (kg/m^2)		0.3	0.2	0.120
Visceral adipose tissue (g)		0.002	0.003	0.331	Visceral adipose tissue (g)		0.002	0.002	0.435

Abbreviation: BMI, body mass index.

in the regression analysis, BMI continued to be significantly associated with both radial–aortic SBP (β = 0.7, $P < 0.001$) and DBP (β = 0.3, $P < 0.001$) (Supplementary Table 2); similar results were observed for carotid–aortic SBP (β = 0.7, $P < 0.001$) and DBP (β = 0.2, $P = 0.001$) (Supplementary Table 3).

After adjusting for Tanner stage, race, and sex, strong associations were observed between radial–aortic SBP with waist circumference ($r = 0.56$ $P < 0.001$), body mass ($r = 0.60$, $P < 0.001$), BMI ($r = 0.56$, $P < 0.001$), and visceral adipose tissue ($r = 0.51$, $P < 0.001$). In addition, carotid–aortic SBP was strongly associated with waist circumference ($r = 0.52$, $P < 0.001$), body mass ($r = 0.56$, $P < 0.001$), and visceral adipose tissue ($r = 0.51$, $P < 0.001$). Moderate associations were observed between body mass and both radial–aortic DBP ($r = 0.42$, $P < 0.001$) and carotid–aortic DBP ($r = 0.43$, $P < 0.001$).

DISCUSSION

In this study, we observed higher levels of central aortic BP in youth with SO compared to those with OW/OB and NW. However, levels of central aortic BP did not differ between females and males. In comparison to percent fat mass and visceral adipose tissue, BMI was more strongly associated with central aortic SBP and DBP. These data suggest that youth with SO may be at increased risk of developing hypertension and CVD later in adulthood, which could be in part mediated by higher central aortic BP. Moreover, though brachial BP is a valid predictor of future CVD risk and is clinically used in the diagnosis of hypertension, our data suggest that central aortic BP may also be a relevant risk factor for CVD.[18,23] This is in line with data from adults showing that compared to peripheral BP, stronger associations between central aortic BP have been observed with adverse CVD risk factors including left ventricular hypertrophy,[24] carotid intima-media thickness,[16] and atherosclerosis.[13]

Pichler *et al.*[25] reported significantly higher peripheral and central aortic BPs among adults with OB. We similarly observed that excess adiposity was associated with higher central aortic BP and further report that the highest levels were among youth with SO. Our findings of higher central aortic BP among youth with OB coincidence with those of studies from other groups and extend these observations to a pediatric population with SO.[26–30] The relationship between OB, as measured by BMI, and peripheral BP has been well documented among both adult[31] and pediatric populations.[32–34] We observed that BMI was significantly associated with central aortic BP. Previous research in pediatrics has similarly reported brachial BP to be positively associated with various measures of adiposity, but most strongly with BMI.[35–37] Despite data implicating abdominal fat as a determinant in the development of high BP measured in the brachial artery among adults,[38] BMI appears to be more closely associated central aortic BP among children and adolescents. Perhaps the relatively short period of exposure to

excess adiposity and/or the ability to physiologically compensate for increased adiposity may explain our findings. The strong association with BMI may also be potentially a result from the effect of height on BP.

Differences in central aortic BP between sexes were not observed in our study. Because the interaction from the multivariate analysis was not significant, we further conclude that the association between elevated central aortic BP and OB status does not differ between sexes in children or adolescents. Previous research in peripheral BP reported that sex differences emerge at the onset of adolescence, with males exhibiting higher SBP.[33,39] Diaz *et al.*[40] measured central aortic BP among youth with an oscillometric device and reported that males exhibited higher central aortic BP. Discrepancies between studies could be attributed to differences in device methodology, and also a younger cohort in our study.

Strengths of this study include the relatively large sample size, inclusion of participants with a wide range of BMI values extending to the SO category, the use of dual-energy X-ray absorptiometry for measures of body composition, and the standardized methods used for measuring central aortic BP. Limitations include the cross-sectional nature of the study, which precludes us from suggesting causality between OB and increased central aortic BP. Participants were not screened for obstructive sleep apnea, which does not allow us to examine whether elevated central aortic BP occurs independently of obstructive sleep apnea in the context of severe pediatric OB. It is also important to note that neither categories nor percentiles exist for central aortic BP, which somewhat limits the ability to interpret the findings from a clinical perspective.

In conclusion, children and adolescents with SO have higher levels of central aortic BP compared with peers with OW/OB or NW. The importance of this study is that it provides evidence that OB-related impairments in central aortic BP and increased CVD risk appear to occur within the first 2 decades of life. Compared with percent fat mass or visceral adipose tissue, BMI was more strongly associated with central aortic BP among children and adolescents, suggesting body size may be a more important determining factor than adiposity during childhood.

SUPPLEMENTARY DATA

Supplementary data are available at *American Journal of Hypertension* online.

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DISCLOSURE

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