Effect of Adenotonsillectomy on Cardiac Function in Children Age 5-13 Years With Obstructive Sleep Apnea

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Changes in left ventricular structure and function have been previously described in children with obstructive sleep apnea (OSA). We aimed to determine if these structural and functional cardiac changes are reversible after treatment of OSA with adenotonsillectomy. Children aged 5 to 13 years with OSA and matched healthy controls were recruited. Adenotonsillectomy occurred within 1 month after diagnosis. Echocardiography and polysomnography were repeated postoperatively. Linear mixed models were fitted to echocardiography measures at baseline and follow-up to assess the effect of OSA on cardiac structure and function. These adjusted for age, gender, race, body mass index, systolic, and diastolic blood pressure. The study sample included 373 children, 199 with OSA and 174 healthy controls. In the control group, 114 children completed the study and 112 completed the study in the OSA group. Children with OSA had reduced diastolic function, lower systolic function, and greater left ventricular mass index at baseline compared with healthy controls (all $p < 0.05$). Measures of active relaxation, elastic recoil and lengthening of the left ventricle impacted overall diastolic function; each of these worsened with increasing OSA severity. Postoperatively, diastolic function improved in children with OSA compared with controls. There were not significant changes in LV mass index or geometry. In conclusion, children with OSA have impaired left ventricular relaxation during diastole indicating early stage diastolic dysfunction. Adenotonsillectomy for OSA signficantly improved diastolic function. Left ventricular remodeling did not change with improvement of OSA. **(Am J Cardiol 2021;141:120−126)**

Obstructive sleep apnea (OSA) is associated with hypertension, stroke, heart failure, coronary artery disease, and atrial fibrillation in adults.[1−5] Currently, there is conflicting evidence regarding the degree of improvement in cardiovascular outcomes in adults who are treated for OSA.[6,7] Several studies have shown that continuous positive airway pressure therapy in adults partially reverses left ventricular (LV) and right ventricular (RV) dysfunction,[6] lowers blood pressure,[8] decreases pulmonary artery pressure[9] and improves endothelial dysfunction.[10] However, randomized trials have not shown a significant benefit in overall cardiovascular morbidity and survival in adults.[10−12] The lack of reversibility of cardiovascular morbidity in adults with OSA does not, however, preclude a causal relationship between OSA and cardiovascular disease. Preclinical cardiovascular changes have been documented in children with OSA.[13,14] Examining the reversibility of these cardiovascular changes in children after treatment may shed light on the possible causal relation between OSA and cardiovascular disease. Therefore, we examined prospectively the reversibility of early structural and functional cardiac abnormalities in children with OSA after treatment with adenotonsillectomy (T&A).

Methods

Children ranging in age from 5 to 13 years with hypertrophy of palatine tonsils who were scheduled for T&A due to nightly snoring were recruited from the otolaryngology and pulmonary clinics and through community advertisements. The inclusion criteria for the OSA group were: (1) absence of chronic medical conditions or genetic syndromes, and (2) overnight polysomnography (PSG) consistent with the diagnosis of OSA (defined as an obstructive apnea-hypopnea index [AHI] > 1/h of sleep). The control group included age- and gender-matched healthy children. Inclusion criteria for the control group were: (1) absence of habitual snoring, and (2) absence of OSA on PSG. Children receiving chronic medications were excluded if they were

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unable to temporarily discontinue their use. The institutional review board at Cincinnati Children's Hospital Medical Center approved this study and written informed consent/assent was obtained.

Data were combined from 2 prospective longitudinal studies to compare children with OSA to healthy controls. Baseline evaluations at the time of enrollment included a history and physical examination, body mass index (BMI) and blood pressure (BP) measurement. All enrolled children had cardiac evaluation with echocardiography (ECHO) and PSG. Children with OSA underwent T&A within 1 month of the baseline assessment. In 1 protocol children were re-evaluated within 6 to 12 months after surgery. The second re-evaluated children 9 to 18 months after surgery . The follow-up period was extended to up to 24 months to accommodate the large number of follow-up visits. Children in the control group underwent the same assessment at baseline and within 6 to 24 months.

The primary outcomes of interest were echocardiographic measures of cardiac structure and function at baseline and after T&A. Multiple parameters were documented for both PSG and ECHO as outlined here.

All PSG studies were performed according to American Thoracic Society standards.[15] Specifically, overnight, in-laboratory, 16 channel PSGs were completed in a pediatric sleep laboratory using a computerized system (Grass Telefactor, Astro-Med Inc., Westwarwick, Rhode Island). Standard criteria were used to define and score sleep staging, arousals and obstructive apneas/hypopneas.[15]

Two-dimensional and 2-dimensionally directed M-mode echocardiographic images were used to assess cardiac structure and function. Cardiac function was also assessed using Tissue Velocity Imaging (TVI)[16] according to the recommendations of the American Society of Echocardiography.[17]

LV structure LV mass and LV mass index were calculated using the formula previously described by Devereux[18] and de Simone[19] respectively. LV geometry was classified as normal, concentric remodeling, eccentric hypertrophy, or concentric hypertrophy on the basis of LV mass and relative wall thickness as described by Ganau.[20]

RV structure RV end-diastolic dimensions were assessed using M-mode echo images and indexed to body surface area.[17]

Systolic function Systolic function (SF) was determined from M-Mode echo.[17] The SF (s') of the LV and RV were estimated using TVI across the mitral valve (MV) and tricuspid valve (TV), respectively (cm/sec).

Diastolic function was estimated from *M-Mode* ECHO by measuring (1) *E wave*, the peak velocity in early diastole (cm/sec) (2) *A wave*, the peak velocity in late diastole (cm/sec) (3) the E/A ratio, which correlates with LV filling patterns, and (4) the isovolumic relaxation time (*IVRT*), the time from aortic valve closure to MV opening.[21] Diastolic function was also assessed using TVI by measuring (1) late diastolic *a' velocity* (a measure of global atrial function determined using the mean of septal and lateral annulus in the 4 apical chambers across the MV and TV) (cm/sec), (2) *e' velocity* (a measure of ventricular relaxation in early diastole measured by the mean of septal and lateral annulus across the MV and TV (cm/sec),[22] and (3) E/e' ratio, which correlates well with LV end diastolic pressure or pulmonary capillary wedge pressure.[16]

Pulmonary artery systolic pressure was estimated from the maximal tricuspid regurgitation velocity.

BP was obtained after resting period of 15 minutes. Subjects were at seated position, feet flat on the floor with both back and arm supported. The cuff size was selected to allow the bladder to encircle at least 80% of the arm circumference. The reported BP is the average of 3 consecutive measurements. Hypertension was defined as systolic or diastolic BP $\geq 95^{th}$ percentile for children 1 to 13 years based age, gender, and height, and $\geq$130/80 for adolescents 13 years and older.[23]

Data were reported as mean with standard deviation for continuous variables and as frequency with percentage for discrete variables. Student's *t* tests were used for continuous data, and chi-square tests for discrete data. Bivariate comparisons for baseline demographics, clinical characteristics, and PSG findings were completed within each study group to compare patients that did and did not complete the study. Linear regression models were fitted to each baseline and after T&A ECHO measure to assess the effect of OSA by group (or, of continuous OAHI) and to measure the association between change in OAHI and change in each ECHO measures adjusting for age, gender ("male" as a reference), race ("white" as a reference), and BMIz (or weight percentile, when height was already included in the formula of the echo meausure). To determine whether the echo measures deviated from published norms, a Z score was compared between groups. Linear mixed effect models were fitted to examine the change from baseline to follow-up between 2 groups (OSA vs control, using "control" as a reference) for each ECHO measure. The following covariates were included: time varying age, gender, race, time varying BMI (or time varying weight), BMI-group interaction (or weight-group interaction), visit, and group-visit interaction. All analyses were conducted using SAS v9.4 (Cary, North Carolina) and all reported p-values were 2-sided with $p \leq 0.05$ considered statistically significant.

Results

The study sample included a total of 373 children, 199 with OSA and 174 healthy controls. Two hundred twenty six children completed both baseline and follow up assessments, including 114 in the control group and 112 in the OSA group (Figure 1). There were no statistically significant differences in demographic factors or PSG findings between children who completed the follow up visit, and those that did not, in either of the study groups (OSA and control). In the control group, those who did not complete the study had significantly lower baseline diastolic BP compared with children that completed the study (58 ± 7 mm Hg vs 60.3 ± 7.2 mm Hg, $p = 0.041$). Children with OSA who did not complete the study had significantly higher systolic BP percentile compared with children that completed the study ($73.7 \pm 21\%$ vs $64 \pm 28\%$, $p = 0.01$). Demographics and clinical characteristics by OSA level for the total 373 children are presented in Table 1. PSG characteristics by OSA level for the total 373 and 262 children who completed the second visit are presented in Table 2.

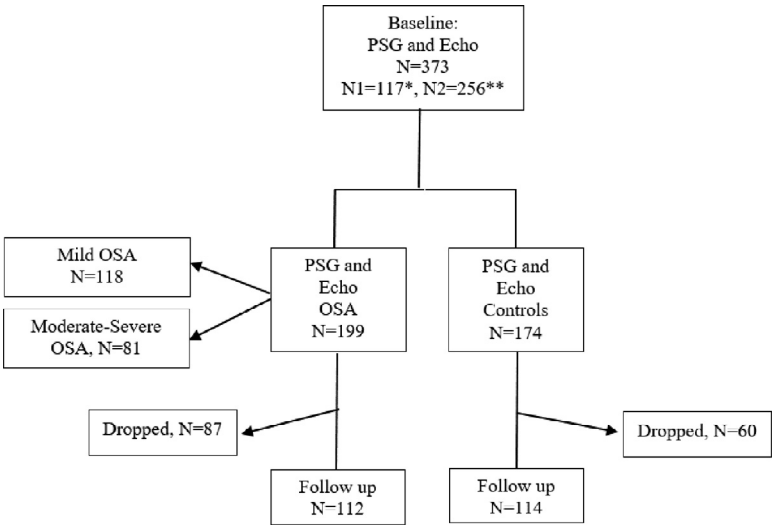

Figure 1. Flow diagram of subjects included in the study. *N1=study 1, **N2=study 2

A. Group Comparison: Control versus OSA

In a model including age, gender, race, and BMIZ score, children with OSA, compared with healthy controls, had reduced diastolic and SF as well as altered LV structure (Table 3). Models including systolic or diastolic BP yielded similar results. Additionally, 17% of children with OSA had E/A Z score of -2 - -1 compared with 7% of healthy controls (p = 0.015).Overall, there were no structural or functional abnormalities of the right ventricle. (Table 3).

Table 1
Demographics and clinical characteristics are reported as mean with SD for continuous data and as frequency with percentage for categorical data

Variable	Control	OSA	p
Number of subjects	174	199	
Age (years)	9.9 ± 2.5	9.4 ± 2.5	0.05
Boys	81(49%)	81(46%)	0.5
White	120(69%)	102(51%)	0.0005
Baseline			
BMI-z score	0.7 ± 0.9	1.1 ± 1.1	0.0006
Obesity	33(19%)	84(42%)	<.0001
Systolic BP (mmHg)	104.9 ± 11.0	106.4 ± 11.1	0.19
Systolic BP (percentile)	63.4 ± 27.0	68.2 ± 25.7	0.09
Systolic hypertension	7(4%)	18(9%)	0.05
Diastolic BP (mmHg)	59.5 ± 7.2	60.6 ± 7.2	0.15
Diastolic BP (percentile)	48.1 ± 22.5	51.8 ± 21.3	0.12
Diastolic hypertension	2(1%)	7(4%)	0.14
Follow up			
Number of subjects	114	112	
Baseline to follow up (years)	1.4 ± 0.5	1.5 ± 0.5	0.32
BMI-z score	0.7 ± 0.9	1.2 ± 1.1	0.0004
Rate of obesity	20(18%)	50(45%)	<.0001
Systolic BP (mmHg),	105.5 ± 11.1	105.4 ± 11.7	0.93
Systolic BP (percentile)	65.7 ± 25.6	63.9 ± 28.1	0.63
Systolic hypertension	5(4%)	11(10%)	0.97
Diastolic BP (mmHg)	60.3 ± 7.2	60.0 ± 6.2	0.69
Diastolic BP (percentile)	51.1 ± 22.0	50.0 ± 18.9	0.70
Diastolic hypertension	1(1%)	2(2%)	0.99

BMI= body mass index; BP= blood pressure.

B. Baseline echocardiographic findings and OSA severity

In a model including age, gender, race and BMIZ score, there was a significant positive association between OAHI and 2 ECHO parameters: IVRT and abnormal LV geometry (LV remodeling and hypertrophy combined). The odds of abnormal LV geometry, a measure that assesses remodeling and hypertrophy, increased with increasing OAHI (OR: 1.044, 95% CI: 1.012 to 1.076, p = 0.0063) after adjusting for all covariates. Models including systolic or diastolic BP produced similar estimates. In the same model, there was a negative association between OAHI and E wave, E/A ratio, MV e' and TV e' (Table 4). Overall, children with OSA had significantly reduced RV diastolic function compared with healthy controls; diastolic dysfunction increased with increasing severity of OSA.

C. Pulmonary artery systolic pressure

Measurable tricuspid regurgitation was identified in 59% of the study population (57%−−controls and 61%−−OSA group). Tricuspid regurgitation velocity did not differ significantly between children with OSA and healthy controls (p = 0.7).

D. Change in echocardiographic measures after T&A

Multiple ECHO measures demonstrated a significant improvement in diastolic function. Specifically, there was a significant decrease in A wave, and a significant increase in the E/A ratio, in the OSA group compared with controls. There was a decrease noted in IVRT that did not reach statistical significance (p = 0.09) (Table 5). Models including BP yielded similar estimates. E/A Z scores improved significantly in the OSA group compared with controls (coefficient = 0.3368, SE = 0.12, p = 0.017).

Discussion

The findings from this prospective study indicate that at baseline, children with OSA have subclinical impairment in LV diastolic function and increased LV mass. Diastolic dysfunction, and abnormal LV geometry, worsened as OSA

Table 2
Polysomnographic characteristics at baseline and at follow up (reported as mean with SD for continuous data and frequency with percentage for categorical data)

	Baseline			Follow up		
Variable	Control (n = 174)	OSA (n = 199)	p	Control (n = 114)	OSA (n = 112)	p
AHI events/hour	1.0 ± 1.3	8.5 ± 11.5	<.0001	1.2 ± 1.5	2.0 ± 2.1	0.0005
oAHI events/hour	0.3 ± 0.3	7.7 ± 10.8	<.0001	0.7 ± 1.5	1.4 ± 1.8	0.002
Sleep efficiency	80.8 ± 10.5	79.6 ± 10.1	0.26	79.8 ± 10.7	81.2 ± 10.3	0.30
Arousal index events/hour	9.0 ± 3.0	12.9 ± 8.3	<.0001	10.3 ± 5.3	9.8 ± 3.1	0.37
Mean saturation (%)	96.6 ± 3.2	96.2 ± 6.1	0.41	97.2 ± 1.0	96.9 ± 1.6	0.13
Max $EtCO_2$ mmHg	50.4 ± 3.2	51.8 ± 6.1	0.008	51.2 ± 5.6	51.3 ± 4.3	0.9
Average EtCO2 mmHg	44.2 ± 2.8	42.7 ± 5.8	0.001	43.4 ± 3.5	43.8 ± 3.8	0.46
%Hypoventilation	5(2.9%)	20(10.0%)	0.0057	4(3.5%)	9(8%)	0.14

AHI = apnea-hypopnea index, EtC02 = End tidal CO2), OAHI = obstructive apnea-hypopnea index.

Table 3
Regression coefficient estimates comparing OSA and control groups at baseline with regard to echocardiographic measures adjusted for age, gender, race, and BMIz or weight percentile

	Analytic sample (n = 373)	
Echocardiographic measure	Regression coefficient of OSA vs Control (SE)	p
LV structure		
LV mass index (gm/m2.7)*,†,¶	1.94 (0.78)	0.0134
LV systolic function		
SF (%)	-1.19 (0.56)	0.0338
MV s' †	0.24 (0.23)	0.2896
LV diastolic function		
E wave (cm/s)*	-1.0 (1.46)	0.4961
A wave (cm/s)*,†	3.40 (1.40)	**0.015**
E/A ratio†	-0.13 (0.06)	**0.0178**
E/e'*,‡,§	0.03 (0.15)	0.8525
IVRT (sec)*	4.88 (1.60)	**0.0024**
MV a' (cm/s)‡	0.20 (0.14)	0.1575
MV e' (cm/s)*,‡,§	-0.11 (0.27)	0.6847
RV structure		
RV/ BSA	0.02 (0.04)	0.623
RV systolic function		
TV s'	-0.06 (0.3)	0.8432
RV diastolic function		
TV e'‡	-0.25 (0.34)	0.46
TV a' (cm/s)*	0.34 (0.27)	0.2209
Tricuspid max pressure gradient *,‡,§	0.24 (0.73)	0.7434

* age is statistically significant (p<0.05).
† gender is statistically significant (p<0.05).
‡ race is statistically significant (p<0.05).
§ BMIZ is statistically significant (p<0.05).
¶ weight% is statistically significant (p<0.05).

Table 4
Regression coefficient estimates comparing OSA severity (OAHI at baseline) with echocardiographic measures adjusted for age, gender, race, BMIz or weight percentile

	Analytic sample (n = 373)	
Echocardiographic measure	Regression coefficient of OAHI (SE)	p
LV structure		–
LV mass index (gm/m2.7)*,†,¶	0.03 (0.04)	0.5565
LV systolic function		
SF (%)‡	-0.0022 (0.03)	0.9457
MV s'*,‡,§	0.0002 (0.012)	0.9889
LV diastolic Function		–
E wave (cm/s)*	-0.21 (0.08)	0.0092
A wave (cm/s)*,†	0.14 (0.08)	0.0619
E/A ratio†	-0.009 (0.003)	0.0036
E/e'*,‡,§	0.02 (0.01)	0.06
IVRT (sec)*	0.22 (0.09)	0.0139
MV a' (cm/s)‡	-0.013 (0.008)	0.0927
MV e' (cm/s)*,‡,§	-0.06 (0.01)	<.0001
RV structure		–
RV/ BSA	0.002 (0.002)	0.2852
RV systolic function		–
TV s'	-0.02 (0.02)	0.2941
RV diastolic Function		
TV e' (cm/s)‡	-0.05 (0.02)	0.0107
TV a' (cm/s)*	-0.02 (0.01)	0.1578
Tricuspid max pressure gradient*,‡,§	0.04 (0.04)	0.3002

OAHI = obstructive apnea hypopnea index.
* age is statistically significant (p<0.05).
† gender is statistically significant (p<0.05).
‡ race is statistically significant (p<0.05).
§ BMIZ is statistically significant (p<0.05).
¶ weight% is statistically significant (p<0.05).

severity increased, quantified here by OAHI. These changes are not mediated by age, gender, race, obesity, systolic or diastolic BP. Following T&A, there was an improvement in diastolic dysfunction after adjusting for covariates.

Our findings suggest that children with OSA develop a narrowing of the LA-LV pressure gradient during diastole. The prolongation of IVRT independent from BP suggests that delayed relaxation time is likely underlying the smaller LA-LV pressure gradient. The small transmitral gradient associated with delayed relaxation limits early diastolic filling. Consequently, residual blood in the atrium is then ejected into the left ventricle during atrial systole. This increases peak A-wave velocity and decreases the E/A ratio. The parameters of transmitral flow doppler worsen with increasing severity of OSA; decreases in the peak E-wave velocity and E/A ratio occur as OSA severity increases, along with prolongation of the IVRT.

The observations from the mitral tissue doppler in our study also suggest functional impairment of LV diastolic function. Increasing OSA severity was associated with a decline in e', a measure of the velocity of mitral annular ascent during the rapid filling period. The velocity of mitral

Table 5
Change from baseline to follow-up between two groups (and with decreasing OSA severity) for echocardiographic measures using linear mixed models (n=226)

	Baseline (LSM with SE)		Follow up (LSM with SE)			
Echocardiographic measure	Control	OSA	Control	OSA	Estimate difference in OSA comparing to control (SE)	p
LV structure			–	–		–
LV mass index (gm/m2.7)	30.36 (0.73)	33.16 (0.70)	29.26 (0.71)	32.39 (0.73)	0.32 (1.04)	NS
LV systolic function						
SF (%)	37.76 (0.54)	35.54 (0.54)	37.93 (0.55)	36.18 (0.55)	0.48 (0.89)	NS
MV s'	10.29 (0.23)	10.21 (0.23)	10.13 (0.24)	10.03 (0.23)	−0.02 (0.39)	NS
LV diastolic Function						
E wave (cm/s)	95.39 (1.35)	92.99 (1.35)	96.79 (1.39)	97.37 (1.36)	2.98 (1.83)	NS
A wave (cm/s)	47.99 (1.13)	51.64 (1.12)	48.31 (1.15)	47.47 (1.13)	−4.50 (2.04)	**0.0287**
E/A ratio (ms)	2.07 (0.05)	1.93 (0.05)	2.07 (0.05)	2.16 (0.05)	0.23 (0.09)	**0.01**
E/e'	5.38 (0.12)	5.43 (0.13)	5.44 (0.13)	5.65 (0.12)	0.16 (0.18)	NS
IVRT (sec)	53.87 (1.56)	60.52 (1.57)	55.29 (1.60)	57.73 (1.57)	−4.20 (2.54)	0.0989
MV a' (cm/s)	6.22 (0.14)	6.45 (0.14)	6.31 (0.14)	6.18 (0.13)	−0.36 (0.23)	NS
MV e' (cm/s)	16.09 (0.24)	15.92 (0.24)	15.94 (0.25)	15.95 (0.24)	0.18 (0.37)	NS
RV structure			–	–		–
RV/ BSA	1.30 (0.03)	1.30 (0.03)	1.22 (0.03)	1.27 (0.03)	0.06 (0.05)	NS
RV systolic function			–	–		–
TV s'	12.88 (0.28)	12.27 (0.28)	12.82 (0.28)	12.15 (0.28)	-0.06 (0.41)	NS
RV diastolic function			–	–		–
TV e' (cm/s)	15.85 (0.32)	15.58 (0.32)	15.83 (0.33)	15.58 (0.32)	0.02 (0.51)	NS
TV a' (cm/s)	8.59 (0.25)	8.54 (0.25)	8.31 (0.26)	8.33 (0.25)	0.08 (0.40)	NS
Tricuspid max pressure gradient	14.88 (0.61)	15.03 (0.58)	14.54 (0.58)	15.03 (0.58)	0.52 (0.96)	NS
Change in Echocardiogrpahic measures with decreasing OSA severity*						
MV e' (cm/s)					−0.221(0.1)	0.0295
E/e'					0.141(0.05)	0.0051

Models adjusted for time varying age, gender, race, time varying BMI, group, BMI-group interaction, visit, and group-visit interaction. Data presented as LSM with SE for echocardiographic measure at baseline and follow for control and OSA groups, group-visit interaction estimates with SE, and p-value for the group-visit interaction.

LSM, Least Square Means; OSA, obstructive sleep apnea; SE, standard error.

* Only statistically significant echo measures are presented as regression coefficient of OAHI.

annular motion is determined by the active relaxation, the elastic recoil and the lengthening of the LV during diastole. The inverse relationship between e' and OAHI suggests that OSA negatively impacts these three physiological processes; each of these processes supports rapid filling of the left ventricle during diastole.

In the present study, LV diastolic function improved after T&A which suggests a causal relationship; no similar echocardiographic changes occurred in the controls. As OAHI improved following T&A, the E/ e' ratio decreased and e' increased. E, is a surrogate for the LA-LV pressure gradient, while e', reflects the extent to which this gradient is generated by ventricular suction. A high e' velocity indicates the presence of vigorous suction created by robust active relaxation and elastic recoil.[24] Therefore, the E/ e' ratio may be considered an estimate of mean LAP.[24]The changes in these parameters indicate that treating OSA leads to improved LV filling; LV filling is improved as LV suction increases and LA pressure decreases.

The parameters we identified that demonstrated early changes in myocardial function are relevant to clinial care and highlight the distinct negative impact of OSA on myocardial function. Identifying parameters to follow is essential as cardiovascular morbidity from OSA may extend from childhood into adulthood. A recent longitudinal study, including a 15-year follow up period, showed that the incidence rate of major adverse cardiovascular events was significantly higher in those diagnosed with OSA during childhood, compared with those who were not. Interestingly, no major cardiovascular events occurred in those who received continuous positive airway pressure treatment or pharyngeal surgery.[25] Given the high prevalence of OSA, affecting up to 25% of children,[26] and the chronic nature of cardiovascular risks, the reversibility of early cardiovascular changes in children is important.

Previous literature pertaining to cardiovascular changes in children due to OSA is limited. Chan et al explored the structure and function of the RV and LV in 66 children with OSA and 35 controls, demonstrating reversibility in subclinical RV and LV dysfunction. However, both T&A and nasal steroids were suggested as treatment options in this study and the attrition rate was high.[27] Attia et al[28] found echo measures assessed by TVI improved in 42 children with OSA and 45 controls. However, analyses did not account for age, gender and BMI. Cincin et al[29] reported higher pulmonary artery pressure and impaired RV function in 30 children with OSA-specific symptoms compared with 30 controls. RV function improved after surgery in 1 additional study, though OSA was not confirmed by PSG and analyses did not adjust for potential confounders.[30]

Although we anticipated that LV mass would decrease with successful treatment of OSA, since significant group differences were observed after adjustment for BMI, the results of the study did not support our hypothesis. It is

plausible that persistent obesity precludes any potential improvement in LV remodeling that may result from OSA treatment. These findings also suggest that OSA impairs diastolic function through a mechanism other than LV hypertrophy.

Pulmonary hypertension has been considered the primary manifestation of cardiac dysfunction in children due to OSA. Existing data pertaining to this association are limited and conflicting, however. Our estimates of pulmonary artery systolic pressure from tricuspid regurgitation velocity did not indicate elevations. The fairly large percentage of children without measurable tricuspid regurge was unexpected, and our results should be considered in that context.

This study has limitations. The attrition rate was 39%, and thus selection bias may have influenced our results. However, there were no significant differences in demographics or PSG findings between children who did and did not follow up, in either of the study groups. There was variability in timing of follow up studies, ranging from 6 to 24 months. However, adjustment for age in the linear mixed model likely mitigated this variability.

In conclusion, this is the first large cohort longitudinal study to include children newly diagnosed with OSA by PSG, with full cardiac assessments and PSG at baseline and follow up. Early diastolic dysfunction correlated with the severity of OSA and was reversible following T&A. Consequently, surgical treatment for OSA during childhood may reduce cardiovascular risk over time.

Author Contributions

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Declaration of interests

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

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