ORIGINAL ARTICLE

A study of Cardiorespiratory Parameters and Sleep Architecture in Patients with Pediatric Obstructive Sleep Apnea undergoing Adenotonsillectomy

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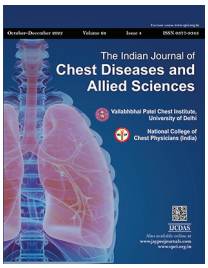

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Abstract

Background: Obstructive sleep apnea (OSA) in pediatric population is associated with cardiac, respiratory, metabolic, neurocognitive, and behavioral dysfunctions. Adenotonsillectomy (AT) is the treatment of choice in children who have hypertrophied adenoid and/or palatine tonsils. However, there is paucity of literature on the impact of AT on cardiorespiratory and sleep parameters in these cases.

Methods: We did a retrospective study on children who had undergone AT from July 2016 to December 2018 at a tertiary hospital in north India. Only those children, whose polysomnography (PSG) was available both before and after AT were enrolled in this study.

- **Cardiac parameters:** Mean heart rate (MHR) and highest heart rate (HHR), number and duration of arrhythmias, and pulse transit time (PTT) drops.
- **Respiratory parameters:** Apnea-hypopnea index (AHI), respiratory disturbance index (RDI), oxygen desaturation index (ODI), mean oxygen saturation (MOS).
- **Sleep parameters:** Time spent in different stages, sleep efficiency (SE), and arousal index (AI) on PSG were compared before and after AT.

Results: A total of 56 children had undergone AT for OSA. Also, PSG, both before and after AT, was available in 37 children. After excluding children having undergone other surgeries for OSA and those with comorbidities, 32 children were enrolled. AT led to significant positive change in AHI (from 7.86 ± 7.91 to 2.03 ± 3.10, $p = 0.01$), RDI (from 16.319 ± 15.64 to 7.38 ± 3.72, $p < 0.01$), AI (from 22.10 ± 14.93 to 15.90 ± 8.48, $p = 0.012$), SE (from 91.47 ± 6.31 to 95.866 ± 3.03, $p < 0.01$), ODI (from 6.7959 ± 5.03 to 1.865 ± 2.09, $p < 0.01$), MOS (from 95.59 ± 2.19 to 97.28 ± 1.27), HHR (from 141.68 ± 17.93 to 120.93 ± 16.98, $p < 0.01$), MHR (86.68 ± 12.95 to 80.29 ± 8.81, $p = 0.01$), and PTT AI (from 36.67 ± 27.72 to 26.93 ± 24.86, $p < 0.01$). There was no non-sinus wide or narrow complex tachyarrhythmia in any child before or after AT. There was no statistically significant change in rapid eye movement (REM) sleep duration or number and duration of bradycardia episodes in these children ($p > 0.05$).

Conclusion: Adenotonsillectomy improved SE and oxygenation, and decreased the number of obstructive events, arousals, heart rate, and PTT AI during sleep in children with OSA. Some children had residual disease after surgery. Heart rate and PTT can be excellent non-invasive parameters for detecting obstructive events during sleep in children and monitoring the impact of various therapeutic modalities.

Keywords: Adenotonsillectomy, Cardiorespiratory parameters, Pediatric obstructive sleep apnea, Sleep architecture.

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Abbreviations used in this Article

OSA = Obstructive sleep apnea; AT = Adenotonsillectomy; PSG = Polysomnography; MHR = Mean heart rate; HHR = Highest heart rate; PTT = Pulse transit time; AHI = Apnea-hypopnea index; RDI = Respiratory disturbance index; ODI = Oxygen desaturation index; MOS = Mean oxygen saturation; SE = Sleep efficiency; AI = Arousal index; REM = Rapid eye movement.

Introduction

The prevalence rate of OSA in children using various diagnostic criteria in different studies across the world has been observed to be between 1 and 4% of the total pediatric population,[1] which makes it one of the commonest chronic disorders in this age group. Obstructive sleep apnea in children is associated with cardiovascular, respiratory, metabolic, neurocognitive, and behavioral dysfunctions.[2–4] American Association of Pediatrics recommends AT as the treatment of choice in children with sleep-disordered breathing who have hypertrophied adenoid and/or palatine tonsils.[5] The impact of AT in these children is generally

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favorable, with a variably positive impact on cardiorespiratory, sleep, metabolic, and neurocognitive parameters.[6] However, a significant proportion of these children continue to have residual abnormalities even after surgery.[7,8]

Some studies have tried to assess the effect of AT on sleep architecture and cardiorespiratory status of children with OSA.[8,9] The results in terms of effectiveness of AT in reversing these abnormalities have been variable. However, most of these studies have restricted the study to a few selected parameters. Modern, attended PSG gives enormous information about the following respiratory parameters: Apnea hypopnea index, RDI, ODI, lowest and MOS; cardiac parameters: heart rate, PTT indices and cardiac arrhythmias in addition to sleep parameters: time spent in different stages, efficiency and AI, etc. This retrospective study was prompted by the paucity of literature on the impact of AT on these cardiorespiratory and sleep parameters in children with OSA who had undergone PSG both before and after surgery.

Materials and Methods

The study was conducted at the Department of Pulmonary, Sleep and Critical Care Medicine, Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi, India. The department receives multiple adult and pediatric patient referrals for evaluation and management of sleep-disordered breathing.

This was a retrospective study involving children less than 18 years of age who had undergone AT after being diagnosed with OSA from July 2016 to December 2018. All children who had undergone a PSG, before as well as after AT, were enrolled in the study. Only those PSGs which were done within 1 month prior to AT, and within 3 months after AT were considered valid. Children were excluded if they had a structural heart disease, neurological, psychiatric disorders, or developmental delays or on any medication which was expected to interfere with PSG interpretation. We also excluded children who had undergone surgical procedures other than AT, such as rapid maxillary expansion or mandibular advancement surgery for sleep disordered breathing. Patients who were already on positive pressure therapy for sleep disordered breathing prior to or after AT were also excluded.

Polysomnography

All the PSGs done during this period were attended level 1 studies. All studies were done on the Alice 6 LDx Diagnostic sleep system of the Philips Respironics (Murrysville, Pennsylvania, US). The studies were analyzed for sleep parameters including sleep stages, namely, SE and AI; respiratory events, namely, apnea hypopnea index, RDI, ODI, lowest oxygen desaturation, MOS; and cardiac parameters, namely, HHR during sleep, MHR during sleep, PTT AI, and the number and duration of cardiac arrhythmias during sleep. All PSGs were reanalyzed by experienced physicians who were neither aware of the clinical details of the children nor about the pre/postsurgery status of the study.

Statistical Analysis

The statistical analysis was done using statistical package for the social sciences (SPSS) software, version 22.0 (IBM Corporation, New York, USA). Parametric data were represented as mean and standard deviations (SDs), and categorical data were presented as percentage. The data in normal distribution was compared using the paired *t*-test and the data not in normal distribution was compared using the Wilcoxon signed-rank test. Proportions if any were compared using the Chi-squared test. A two-tailed *p*-value of less than 0.05 was considered significant.

Results

A total of 56 children had undergone AT for OSA. Polysomnography was done both before and after AT in 37 children out of whom 5 were excluded because of additional comorbidities and other surgical procedures done during the study period (Flowchart 1). That left us with 32 children (25 males and 7 females) who were enrolled for assessment. All the children were under-weighing with a BMI of 17.23 ± 2.87 kg/m^2 (Table 1). Retrognathia was seen in 1 and high arched palate in three children. Although SE improved significantly there was no impact on duration spent in various stages of sleep in these children (Table 2). There was immense improvement in obstructive parameters such as AHI and RDI; consequently, improvement was mirrored in oxygenation parameters: MOS, minimum saturation, and maximum oxygen desaturation in almost all children (Table 3). However, significant residual disease in terms of AHI and RDI was seen after

Flowchart 1: The flowchart of participants in the study

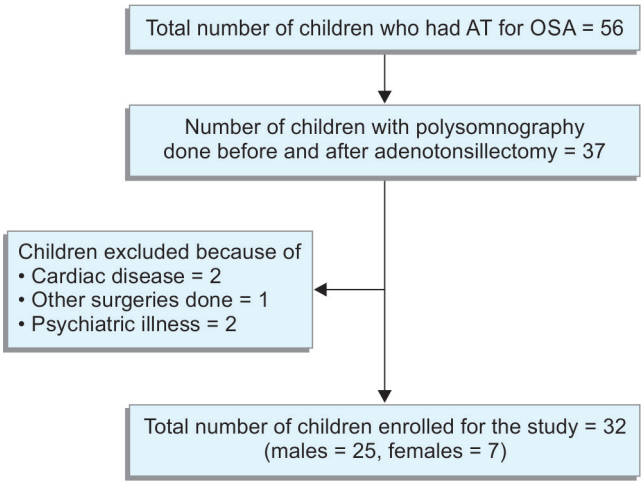

Table 1: General characteristics of children before AT

N = 32	*Range*	*Mean ± SD*
Age (years)	6–15	9.094 ± 2.966
Males *n* (% of total children)	25 (78.1%)	
Females *n* (% of total children)	7 (21.9%)	
Height (in m)	1.024–1.638	1.282 ± 0.207
Weight (kg)	15–60	29.85 ± 13.09
BMI (kg/m^2)	10.36–22.35	17.23 ± 2.87
Neck circumference (in inches)	9–13	10.47 ± 1.09
Tonsils grade	1–3	2.875 ± 0.421
Modified Mallampati grade	1–4	2.563 ± 0.8776
Retrognathia (*n*) (% of total children)	1/32 (3.1%)	
High arched palate (*n*) (% of total children)	3/32 (9.4%)	

Table 2: Sleep parameters in children with OSA before and after AT (*N* = 32)

	Presurgery		*Postsurgery*		
	Range	*Mean ± SD*	*Range*	*Mean ± SD*	*p-value*
N1 (% of total sleep time)	8.9–44.7	22.56 ± 8.29	10.9–31.6	20.41 ± 6.32	0.122
N2 (% of total sleep time)	24.3–44.7	48.13 ± 12.49	22.2–77.6	51.10 ± 14.56	0.331
N3 (% of total sleep time)	1.5–46.8	24.82 ± 12.60	7.3–46.1	22.75 ± 10.50	0.441
REM (% of total sleep time)	0.0–21.7	4.47 ± 5.24	0.00–25.7	5.73 ± 6.37	0.359
SE (%)	71.4–99.5	91.47 ± 6.31	89.1–100	95.866 ± 3.03	**<0.01**

Significant *p*-values are in bold

Table 3: Respiratory parameters in children with OSA before and after AT (*N* = 32)

	Presurgery		*Postsurgery*		
	Range	*Mean ± SD*	*Range*	*Mean ± SD*	*p-value*
ODI	1.10–20.20	6.79 ± 5.03	0–10.28	1.86 ± 2.09	**<0.001**
MOS	88–99	95.59 ± 2.19	95–99	97.28 ± 1.27	**<0.001**
Minimum oxygen saturation	65–95	83.66 ± 8.29	68–99	89.93 ± 6.83	**0.001**
Maximum oxygen desaturation	3–30	10.22 ± 6.57	02–17	5.43 ± 3.86	**<0.001**
AI	3.1–95.7	22.10 ± 14.93	1.2–41.2	15.90 ± 8.48	**0.034**
Apnea hypopnea index	0.2–41.9	7.90 ± 7.88	0.0–16.5	2.03 ± 3.10	**<0.001**
RDI	5.3–89.1	16.31 ± 15.64	1.4–21.5	7.38 ± 3.72	**0.002**

Significant *p*-values are in bold

Table 4: Cardiovascular parameters in children with OSA before and after AT (*N* = 32)

	Presurgery		*Postsurgery*		
	Range	*Mean ± SD*	*Range*	*Mean ± SD*	*p-value*
Mean heart rate	53.50–112.00	86.68 ± 12.95	58.7–94.7	80.29 ± 8.81	**0.005**
Highest heart rate	97–170	141.68 ± 17.93	92–162	120.93 ± 16.98	**<0.01**
HHR (95 percentile of time)	84.0–137	103.19 ± 12.31	78–117	94.47 ± 9.39	**<0.01**
Number of sinus bradycardia episodes	0–19	1.31 ± 3.71	0–9	0.531 ± 1.88	0.040
Lowest heart rate in sinus bradycardia	42–49	44.33 ± 2.73	44–48	46.0 ± 1.82	0.066
Total duration of sinus bradycardia	13–112	40.66 ± 36.86	6–69	26.75 ± 29.12	0.068
Number of sinus tachycardia episodes	11–138	63.34 ± 36.34	9–109	38.96 ± 25.22	**<0.01**
HHR during a sinus tachycardia	97–170	141.34 ± 17.81	92–162	120.56 ± 16.66	**<0.01**
Total duration of sinus tachycardia	129–997	503.15 ± 257.03	69–801	304.7 ± 163.56	**<0.01**
Number of wide complex tachycardia	0	NA	0	NA	NA
Number of narrow complex tachycardia	0	NA	0	NA	NA
Number of asystoles	0	NA	0	NA	NA
Number of atrial fibrillation	0	NA	0	NA	NA
Number of other arrhythmias	0	NA	0	NA	NA
Total number of PTT drops during sleep	31–1340	331.63 ± 282.75	10–1180	297.50 ± 326.91	**0.374**
PTT AI	5.1–122.5	36.67 ± 27.72	1.9–98.9	26.93 ± 24.86	**0.001**

PTT, pulse transit time; Significant *p*-values are in bold

AT (post-AT AHI = 2.03 ± 3.10, post-AT RDI = 7.38 ± 3.72); 15 children had a post-AT AHI of >1/hour.

There was a significant drop in both mean and HHR as well as the number of episodes of tachycardia, though there was no statistically significant change in number of episodes and time spent in sinus bradycardia (Table 4). We did not see any episode of atrial fibrillation or any other wide or narrow complex tachyarrhythmia in these children before or after AT. There was significant decrease in the number of total PTT drops in sleep as well as PTT AI in these children after AT (Table 4). We also compared the various parameters between those who were cured and those who had residual OSA (Tables 5 and 6); none of the cardiac and sleep architecture parameters were different in both the groups suggesting that even when the surgery does

Table 5: Sleep and respiratory parameters in children with OSA after AT

	Cured (n = 17)	*Not cured (n = 15)*	
	Mean ± SD	*Mean ± SD*	*p-value*
N1 (% of total sleep time)	20.08 ± 5.89	20.78 ± 6.97	0.761
N2 (% of total sleep time)	53.43 ± 14.44	48.46 ± 14.74	0.344
N3 (% of total sleep time)	20.80 ± 9.84	24.96 ± 11.13	0.271
REM (% of total sleep time)	5.68 ± 6.61	5.79 ± 6.34	0.962
SE (%)	95.04 ± 3.17	96.80 ± 2.68	0.103
ODI	0.79 ± 0.86	3.08 ± 2.43	**0.001**
MOS	97.18 ± 1.42	97.40 ± 1.12	0.629
Minimum oxygen saturation	91.65 ± 3.32	88.00 ± 9.13	0.134
Maximum oxygen desaturation	4.12 ± 2.03	6.93 ± 4.89	**0.038**
AI	13.51 ± 5.19	18.62 ± 10.65	0.089
Apnea hypopnea index	0.39 ± 0.30	3.90 ± 3.76	**0.001**
RDI	6.65 ± 2.85	8.22 ± 4.48	0.241

Significant *p* values are in bold

Table 6: Cardiovascular parameters in children with OSA after AT

	Cured (n = 17) Mean ± STD	*Not cured (n = 15) Mean ± STD*	*p-value*
Mean heart rate	79.34 ± 8.18	81.37 ± 9.66	0.525
Highest heart rate	116.12 ± 12.34	126.40 ± 20.12	0.088
HHR (95 percentile of time)	92.24 ± 8.89	97.00 ± 9.61	0.156
Number of sinus bradycardia episodes	0.41 ± 1.46	0.67 ± 2.32	0.717
Lowest heart rate in sinus bradycardia	44.50 ± 0.70	47.50 ± 0.70	0.051
Total duration of sinus bradycardia	14.50 ± 12.02	39.00 ± 42.43	0.561
Number of sinus tachycardia episodes	33.76 ± 20.59	45.33 ± 29.47	0.255
HHR during a sinus tachycardia	116.12 ± 12.34	125.6 ± 19.73	0.122
Total duration of sinus tachycardia	272.59 ± 130.58	339.13 ± 191.66	0.268
Total number of PTT drops during sleep	271.88 ± 375.48	326.53 ± 271.70	0.645
PTT AI	25.45 ± 30.68	28.62 ± 16.95	0.725

PTT, pulse transit time; Significant *p*-values are in bold

not completely cure the OSA, it led to comparable improvements in each group.

Discussion

Studies have demonstrated the positive impact of AT on obstructive parameters such as AHI; RDI; oxygenation status, namely, ODI, MOS and lowest oxygen saturation; sleep parameters, namely, AI and SE.[8,10] There was an immense improvement in almost all these parameters in our subjects. Like other studies, residual OSA was also seen in our study group.

Previous studies have had an apparent disagreement in which sleep parameters significantly changed after surgery. Bhattacharjee et al.[11] observed a decrease in sleep stage 1 without an increase in slow-wave sleep while Marcus et al.[7] observed that treatment of OSA decreases the percentage of sleep time in stage N1 and no changes were observed in stage N3 or REM sleep in children. A recent meta-analysis by Lee et al. reported that after surgery, sleep architecture was altered by a significant decrease in sleep stage 1, an increase in slow-wave sleep and the REM stage, and enhanced SE.[10] In contrast, though we observed an improvement in the SE; there was no change in time spent in various sleep stages in terms of percentage of the sleep period. This difference may be due to the varying period (up to 3 months) after the surgery when the PSG was done; and the relatively small sample size.

Heart rate and rhythm have been described as a surrogate of obstruction during sleep;[12] the rate was significantly decreased after AT in our study. The number and duration of tachycardia episodes also decreased significantly. There is a paucity of literature on cardiac arrhythmia in children with sleep-disordered breathing. We did not see any episode of atrial fibrillation or any other narrow or wide complex non-sinus tachycardia in any child before or after surgery. Sinus bradycardia (number of episodes, total duration of episodes, and lowest heart rate) did not show significant change after AT.

Pulse transit time refers to the time it takes a pulse wave to travel between two arterial sites. The speed at which this arterial

pressure wave travels is directly proportional to blood pressure. PTT is a reflector of arterial wall compliance and has been suggested as a non-noninvasive surrogate of blood pressure.[13] Transient rise in blood pressure following a respiratory obstructive event and arousal during sleep leads to stiffening of the arterial wall and a drop in PTT.[14] The PTT arousal has been suggested as a surrogate marker of arousal that measures the autonomic consequences of arousal.[13] This "PTT arousal" has been shown to be as sensitive as EEG arousals in pediatric sleep studies in detection of respiratory events.[15] PTT has also been found to be inversely related to severity of OSA in children and greater in REM sleep as compared to other stages in these children reflecting the changes in blood pressure in these circumstances.[16] We did not find any study on the impact of AT on PTT in children. There was a significant decrease in PTT AI after AT in our subjects which reflects decrease in the episodes of nocturnal rise in blood pressure in these children. This shows a positive impact of AT on upper airway obstruction in these children which is the cause of arousals and rise in blood pressure.

The limitation of the current study is the retrospective nature of the study and the relatively small sample size. Also, the post-AT PSG was done within 3 months of the surgery, and it does not assess the long-term impact of the surgery on the PSG parameters.

Conclusion

Adenotonsillectomy has a definite positive impact on cardiorespiratory and sleep status of children although the benefit is variable and may not be complete. Larger trials are needed to look for determining factors for a favorable outcome. Monitoring heart rate and PTT during sleep can be used in conjunction with AHI for detecting respiratory events in these children as well as monitoring the outcome in these children undergoing AT.

References

1. Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc 2008;5(2):242–252. DOI: 10.1513/pats.200708-135MG.
2. Nixon GM, Brouillette RT. Sleep. 8: Paediatric obstructive sleep apnoea. Thorax. 2005;60(6):511–516. DOI: 10.1136/thx.2003.007203.
3. Sinha D, Guilleminault C. Sleep disordered breathing in children. Indian J Med Res 2010;131:311–320. PMID: 20308756.
4. Suri JC, Sen MK, Sharma R, et al. Metabolic changes in normal- and underweight children with obstructive sleep-disordered breathing. Sleep Med 2015;16(11):1366–1371. DOI: 10.1016/j.sleep.2015.07.026.
5. Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2012;130(3):576–584. DOI: 10.1542/peds.2012-1671.
6. Dehlink E, Tan HL. Update on paediatric obstructive sleep apnoea. J Thorac Dis 2016;8(2):224–235. DOI: 10.3978/j.issn.2072-1439.2015.12.04.
7. Marcus CL, Moore RH, Rosen CL, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. N Engl J Med 2013;368(25):2366–2376. DOI: 10.1056/NEJMoa1215881.
8. Suri JC, Sen MK, Venkatachalam VP, et al. Outcome of adenotonsillectomy for children with sleep apnea. Sleep Med 2015;16(10):1181–1186. DOI: 10.1016/j.sleep.2015.02.539.
9. Baumert M, Pamula Y, Martin J, et al. The effect of adenotonsillectomy for childhood sleep apnoea on cardiorespiratory control. ERJ Open Res 2016;2(2):00003–2016. DOI: 10.1183/23120541.00003-2016.
10. Lee CH, Hsu WC, Chang WH, et al. Polysomnographic findings after adenotonsillectomy for obstructive sleep apnoea in obese and non-obese children: A systematic review and meta-analysis. Clin Otolaryngol 2016;41(5):498–510. DOI: 10.1111/coa.12549.
11. Bhattacharjee R, Kheirandish–Gozal L, Spruyt K, et al. Adenotonsillectomy outcomes in treatment of obstructive sleep apnea in children: A multicenter retrospective study. Am J Respir Crit Care Med 2010;182(5):676–683.
12. Rossi VA, Stradling JR, Kohler M. Effects of obstructive sleep apnoea on heart rhythm. Eur Resp J 2013;41(6):1439–1451. DOI: 10.1183/09031936.00128412.
13. Smith RP, Argod J, Pépin JL, et al. Pulse transit time: An appraisal of potential clinical applications. Thorax 1999;54(5):452–457. DOI: 10.1183/09031936.00128412.
14. Galland BC, Tan E, Taylor BJ. Pulse transit time and blood pressure changes following auditory-evoked subcortical arousal and waking of infants. Sleep 2007;30(7):891–897. DOI: 10.1093/sleep/30.7.891.
15. Katz ES, Lutz J, Black C, Marcus CL. Pulse transit time as a measure of arousal and respiratory effort in children with sleep-disordered breathing. Pediatr Res 2003;53(4):580–588.
16. Nisbet LC, Yiallourou SR, Biggs SN, et al. Preschool children with obstructive sleep apnea: The beginnings of elevated blood pressure? Sleep 2013;36(8):1219–1226. DOI: 10.5665/sleep.2890.