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The effect of adenotonsillectomy on pulmonary hypertension in pediatric obstructive sleep apnea

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Abstract

Background: In a majority of OSA children with adenotonsillar hypertrophy, very mild symptoms or no symptoms at all are related to the cardiopulmonary system, but symptomless chronic changes may slowly occur in these children. Therefore, it is wise to monitor these patients by an easy, noninvasive cost-effective method; this can easily be done by monitoring mean pulmonary artery pressure (mPAP) of these children with Doppler echocardiography. Doppler echocardiography has been demonstrated to have a perfect correlation with cardiac catheterization. This study aims to determine the pulmonary arterial systolic pressure (PASP) in OSA children with hypertrophied tonsils and adenoid and to clarify whether adenotonsillectomy has any effect on pulmonary arterial pressure of these children.

Methods: Study was conducted on 50 children of both sexes aged from 4 to 15 years. Children complain of loud snoring and obstructive sleep apnea due to hypertrophied tonsils and adenoids. Children fit for general anesthesia and adenotonsillectomy. In all subjects, Doppler echocardiography was done before and after adenotonsillectomy.

Results: Comparison between preoperative PASP and after 2 months showed that normal PASP were found in 25 (50.0%) and 50 (100.0%) for preoperative and after 2 months respectively. There was statistically significant difference between preoperative PASP and after 2 months. However, there was no correlation between preoperative PASP and age, sex, or duration of symptoms.

Conclusion: From this study, we conclude that adenotonsillar hypertrophy causes higher PASP in OSA children, PASP is worse in neglected OSA cases with long duration of symptoms, and adenotonsillectomy is an effective therapeutic measure in such patients.

Keywords: Pediatric OSA, Adenotonsillectomy, Pulmonary hypertension, Pulmonary artery pressure, Doppler echocardiography

Background

Pediatric OSA is multifactorial in its association with narrowing of the upper airway during sleep. Unlike that in adults, however, adenotonsillar hypertrophy is most often responsible to be a major factor. The peak incidence is between 3 and 6 years of age, when tonsils and adenoids are largest in relation to airway size. Most children with OSA are of normal weight or may even experience poor weight gain [1].

Most notably, a child with significant OSA will frequently not exhibit loud irregular snoring but, rather, only a louder breathing noise [2]. Restless sleep, increased sweating, and parasomnias have been described. Daytime symptoms may include mouth breathing due to aggravated nasal respiration and frequent respiratory tract infections and occasional daytime sleepiness, and failure to thrive is reported in more than 50% of cases [3].

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The cardiovascular complications of OSA are of immediate importance because their earlier recognition could theoretically allow for formulation of interventional strategies, aiming to reverse this process in childhood and, thus, prevent its consequences later in adult life [4, 5].

Recurrent hypoxic and hypercapnic episodes of OSA elevate pulmonary vascular resistance leading to pulmonary hypertension. Similarly to adults, evidence of right ventricular dysfunction has been shown in children with OSA [6]. Such elevations in pulmonary artery pressures may potentially lead to cor pulmonale. Treatment of OSA in adult patients reduced pulmonary vascular resistance [7]. In a recent report, increased plasma levels of B-type natriuretic peptide, a marker of ventricular strain, have been found in children with OSA with a decrease following adenotonsillectomy. Improvements in echocardiographic parameters of increased pulmonary pressure were also reported following adenotonsillectomy (T&A) [8].

Pulmonary hypertension is defined as pressure within the pulmonary arterial system elevated above the normal range. It may be defined as a pulmonary artery systolic pressure greater than 30 mm Hg or a pulmonary artery mean pressure greater than 20 mmHg. That subsequently result in the normal adaptive response of hypertrophy and dilatation. Right ventricular (RV) failure may develop in either setting when the ventricle is unable to further respond to the hemodynamic burden, what is referred as cor pulmonale. RV dilatation causes the interventricular septum to shift to the left, thereby decreasing LV volume and compliance [9].

Accurate noninvasive measurement of pulmonary arterial blood pressure can be obtained with the Doppler echocardiogram, but the physician must rely on clinical information obtained from the history and physical examination to select individuals for Doppler echocardiographic examination [10].

The aim of this study is to determine the pulmonary arterial systolic pressure (PASP) in OSA children with hypertrophied tonsils and adenoid and to clarify whether tonsillectomy and adenoidectomy have any effect on pulmonary arterial pressure of these children.

Methods

Patient selection

The study was prospectively conducted on 50 consecutive children with significant OSA symptoms who underwent adenotonsillectomy at the ENT Department of Alexandria Main University hospitals between January and December 2018.

Patients' inclusion criteria included children of both sexes aged from 4 to 15 years, children complaining of loud snoring and OSA due to hypertrophied tonsils and adenoids, and children fit for general anesthesia and adenotonsillectomy, whereas exclusion criteria included the presence of other chronic disease, other causes of obstructive sleep apnea such as morbid obesity, endocrine diseases, septal deviation, or craniofacial deformities, and children who had small adenoids and tonsils (less than grade 2).

Patient evaluation

All children were subjected to the following:

1. Complete history taking from parents using OSA-18 questionnaire [11]
    a. Number of recurrent attacks of adenotonsillitis in the last year
    b. The presence of loud snoring
    c. Night sleep disturbance and whether the child exerts an effort to breathe or not
    d. Daytime sleepiness, headache, nocturnal enuresis, decreased concentration, and dyspnea on effort
    e. History of chest and heart problems
2. ENT examination: With stress on the nose (to exclude causes of obstruction other than adenoid such as deviated septum, allergy, or polypi) and the oropharynx (for detection of tonsillar size with grading according to Brodsky scale) [12], as in Fig. 1
3. Diagnosis of sleep apnea was based mainly on history taking from the parents using OSA-18 questionnaire [13].
4. Chest and heart examination: By the cardiologist for detection of cardiomegaly or pulmonary edema to exclude lower airway pathology which can be a cause of cor pulmonale
5. Radiological examination: Lateral plain x-ray of the nasopharynx as in Fig. 2
6. Echocardiography (as in Figs. 3 and 4): The following modalities are used:
    a. M-mode echocardiography (detects cardiac anatomy and dimensions)
    b. Two-dimensional echocardiography (detects spatial relation of the cardiac structures)
    c. Doppler and colored Doppler echocardiography (measures tricuspid valve flow through the cardiac wall and measures pressure gradient of the tricuspid regurge which helps to measure PASP)

Operation

Endoscopic coblation-assisted adenotonsillectomy is carried out for all cases under general anesthesia after full

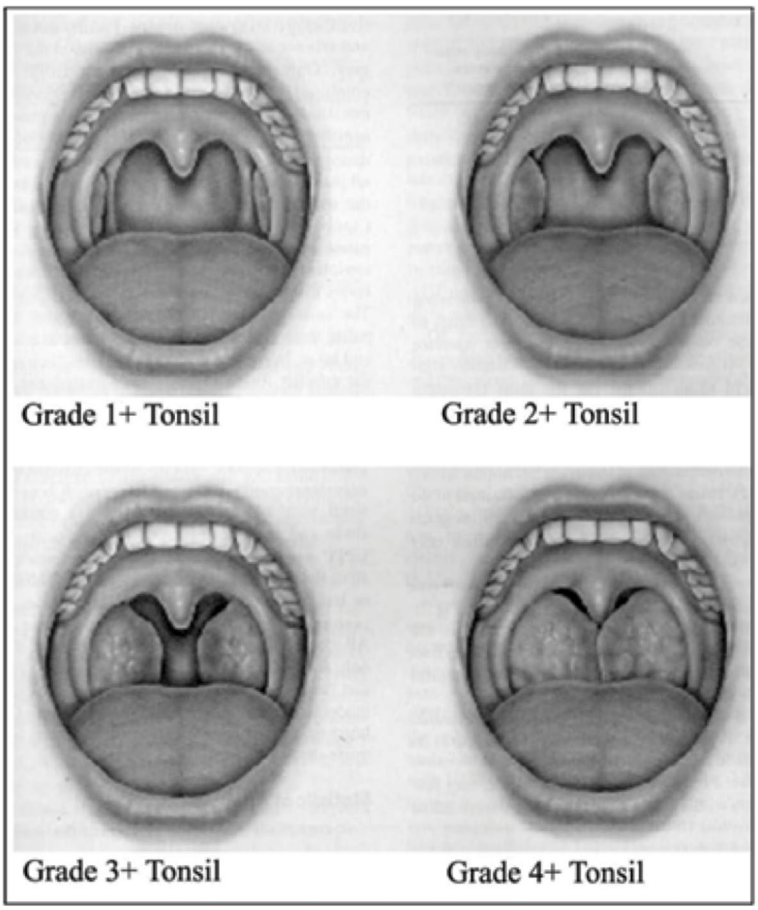

Fig. 1 Grading of palatine tonsils hypertrophy proposed by L. Brodsky [12]

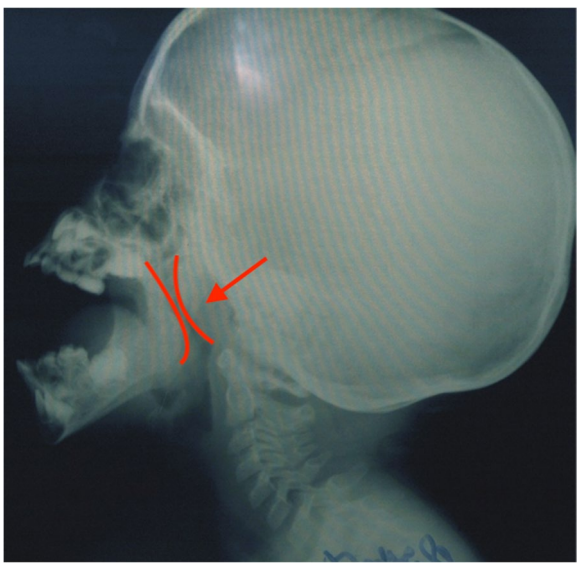

Fig. 2 Nasopharyngeal soft tissue x-ray (lateral view) shows nearly complete attenuation of nasopharyngeal airway (large adenoid)

preoperative evaluation and investigations. The coblation wand used was the EVac 70 Xtra HP® at the power of 7 ablation/5 coagulation using cold saline irrigation, and transoral 70° endoscope was used to ensure complete bloodless adenoidectomy.

Postoperative assessment

All cases were admitted overnight at the hospital for observation; systemic steroids were given. Follow up by using echocardiography 3 months post-operatively to judge improvement of preoperative cardiac problems.

Statistical analysis

All analyses were performed with STATA 12.1 software (Stata Corp., College Station, TX, USA). Paired *t*-test and two-sample *t*-test were used to compare means within group. Correlations between two quantitative variables were assessed using Pearson coefficient. Probability values lower than 0.05 were considered statistically significant.

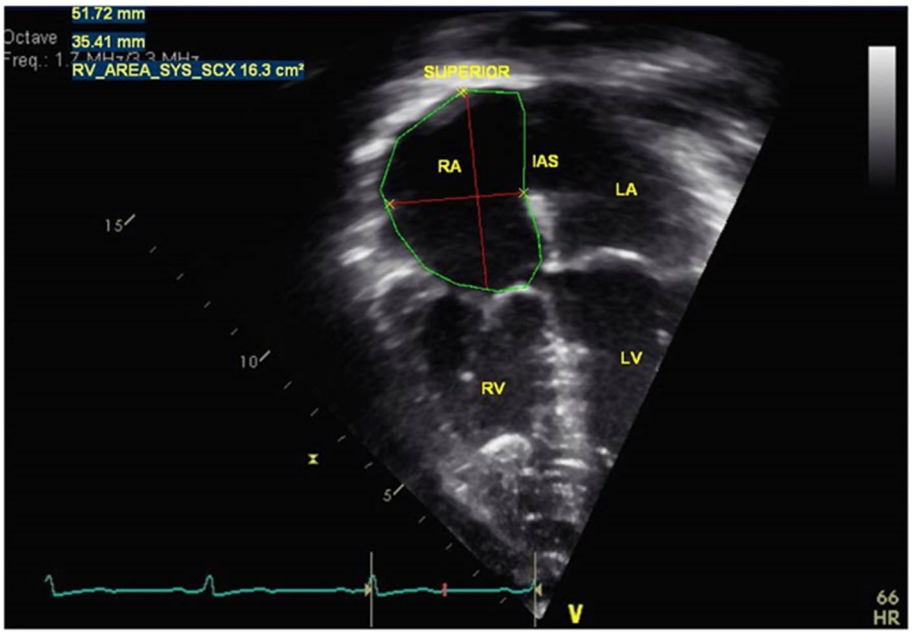

Fig. 3 Measurement of right atria dimensions (major and minor axis) and right atrial area in end systole in a pulmonary hypertension patient

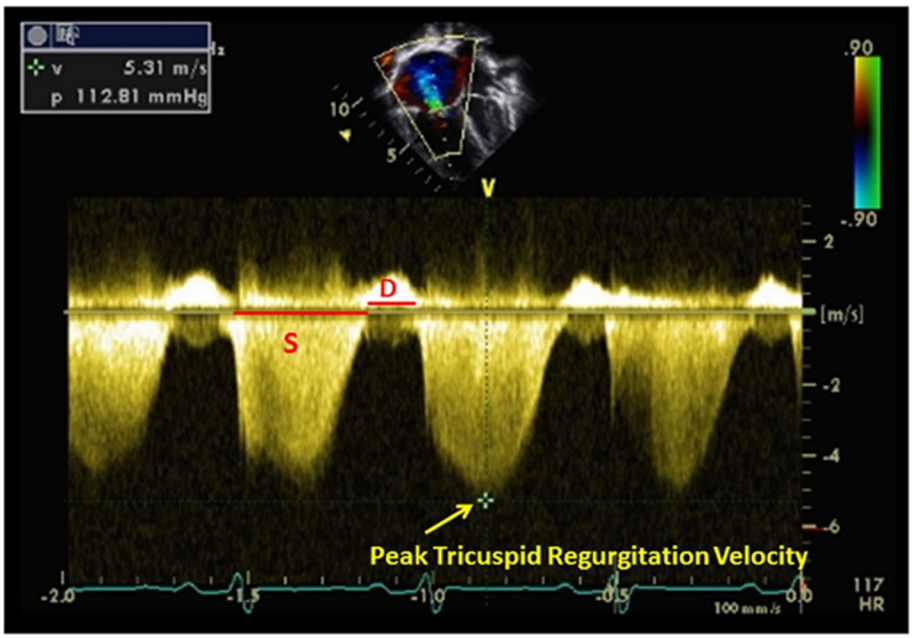

Fig. 4 Tricuspid regurgitation estimating right ventricular pressure in a patient with pulmonary hypertension

Results

The distribution of the studied 50 patients was as follows: age ranged between 4.0 and 16.0 years with the mean of 8.34 ± 3.57 years: 24 (48.0%) males and 26 (52.0%) females. Duration of symptoms was less than or equal to 12 months in 12 cases (24.0%), between 12 and 24 months in 28 cases (56.0%), and more than 24 months in 10 cases (20.0%). Duration ranged between 12.0 and 36.0 months with the mean of 22.56 ± 8.27 months.

Preoperative PASP

Statistical analysis of the studied cases according to preoperative PASP was presented in Table 1; it showed that normal preoperative PASP were found in 25 (50.0%), and abnormal also were found in 25 (50.0%). Preoperative PASP ranged between 33.0 and 52.0 with the mean of 41.40 ± 6.34 mmHg (Fig. 5).

Preoperative PASP and after 2 months of adenotonsillectomy

Comparison between preoperative PASP and after 2 months was presented in Fig. 5; it showed that normal PASP were found in 25 (50.0%) and 50 (100.0%) for preoperative and after 2 months respectively. There was statistically significant difference between preoperative PASP and after 2 months ($P = 0.001$).

Correlations

Table 2 shows correlation between preoperative PASP with duration of symptoms; it demonstrated that there was no statistically significant correlation between PASP with duration of symptoms ($r = 0.294$, $P = 0.154$).

Table 3 shows correlation between preoperative PASP with age; it illustrated that there was no statistically significant correlation between PASP with age ($r = 0.360$, $P = 0.098$).

Table 4 shows correlation between preoperative PASP with sex; it demonstrated that preoperative PASP ranged between 12.0–32.0 and 15.0–36.0 with the mean of 22.1 ± 7.36 and 27.6 ± 8.25 for males and females, respectively; there was no statistically significant correlation between PASP with sex ($P = 0.365$)

Table 1 Statistical analysis of the studied cases according to preoperative PASP ($n = 50$)

	No.	%
Preoperative PASP		
Normal	25	50.0
Abnormal	25	50.0
Min.-max.	33.0–52.0	
Mean ± SD	41.40 ± 6.34	
Median	40.0	

Discussion

It has been previously reported that increased upper airway resistance resulting from hypertrophied tonsils and adenoid can cause intermittent airway obstruction and chronic alveolar hypoventilation and even lead to severe cardiopulmonary complications like cor pulmonale [14].

The main mechanism of pulmonary hypertension is hypoxia-related pulmonary vasoconstriction; other contributing factors are hypercapnia-induced pulmonary vasoconstriction and exaggerated negative intrathoracic pressure during obstructive episodes [15].

Since 1966, Luke et al. [16] stated that hypoxia has 2 effects on cardiopulmonary system. Firstly, it leads to pulmonary vasoconstriction, an increase in m PAP and right ventricular afterload, and finally causing right ventricular hypertrophy and right ventricular heart failure; secondly, it causes pulmonary edema by increasing capillary permeability.

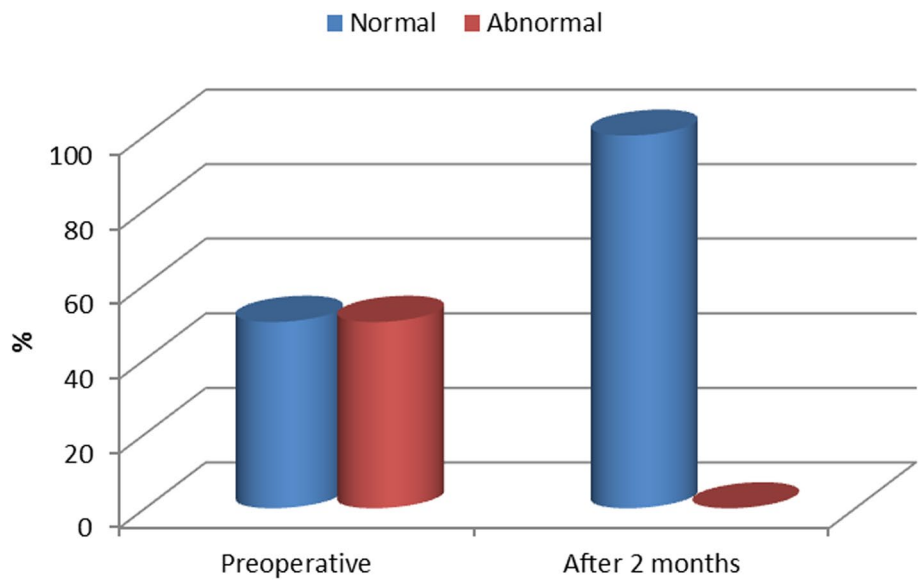

Fig. 5 Comparison between preoperative PASP and after 2 months

Table 2 Correlation between preoperative PASP with duration of symptoms

	Preoperative PASP	
	r	*p*
Duration of symptoms (months)	0.294	0.154

r, Pearson coefficient

Table 3 Correlation between preoperative PASP with age

	Preoperative PASP	
	r	*p*
Age (years)	0.360	0.098

Table 4 Correlation between preoperative PASP and sex

Preoperative PASP	Male	Female
Range	12.0–32.0	15.0–36.0
Mean	22.1	27.6
SD	7.36	8.25
T	0.985	
p	0.365	

Otolaryngologists and pediatricians must know that obvious anamnesis of upper airway obstruction is sufficient to indicate surgical intervention. Adenotonsillar hypertrophy and upper airway obstruction take the first order among the adenotonsillectomy indications [17].

Luke et al. proved the efficacy of adenoidectomy and tonsillectomy in treatment of cardiorespiratory complications caused by adenotonsillar enlargement. They studied four cases with upper airway obstruction by adenoidal or tonsillar hyperplasia with complications ranging from moderate cardiomegaly to severe heart failure and pulmonary edema. Improvement, following surgery, of the four cases was documented by radiological and electrocardiography examination [16].

Ainger et al., in 1968, reported six children with hypoxia, hypercarbia, and cor pulmonale due to large tonsils and adenoid and stated that relief of the upper airway obstruction by tonsillectomy and adenoidectomy was followed by a prompt return of arterial blood gases to normal, heart size often decreasing rapidly. P wave is the first to be normal, and other electrocardiographic abnormalities resolved more slowly [18].

Potsic and Marsh, in 1989, stated that children with craniofacial anomalies and obstructive sleep breathing also respond to adenotonsillectomy, even when there is little lymphoid hyperplasia. On some occasions, improvement is considerable but does not relieve all the obstruction. This is particularly true in anomalies such as Crouzon's syndrome, where the cranial base severely restricts the A-P dimension of the nasopharynx [19].

Kudoh and Sanai, in 1996, reported that adenotonsillectomy is very effective in treating sleep-associated breathing disorders of severely obese children with large tonsils or adenoid [20].

Rosenfeld and Green sated in 1990 that tonsillectomy may relieve OSA in children, even in the absence of clinical tonsillar hypertrophy. The palatoglossal fold may obscure a significant portion of the tonsil, and in the supine position, in which the tongue and soft tissues of the oropharynx fall backward, even small tonsils can cause obstruction [21].

In this study, comparison between preoperative PASP and after 2 months showed that normal PASP were found in 25 (50.0%) and 50 (100.0%) for preoperative and after 2 months respectively. There was statistically significant difference between preoperative PASP and after 2 months.

Diagnosis of sleep apnea in children can be achieved by clinical history, as reported by Attal et al. (1992) [22], who stated that diagnosis of sleep apnea can be achieved by clinical history. Moreover, Brouilette et al. (1984) [23] stated that difficulty in breathing during sleep observed by parents strongly suggests that the child has OSA. In this study, it was found that 17 (56.7%) out of thirty snorer children had sleep apnea. These results agreed with those of Yilmaz et al. (2005) [14] who found that sleep apnea in 31 (59%) out of 52 children and also agreed with Richardson et al. (1980) [24] who found sleep apnea in 60% of their cases with adenotonsillar hypertrophy. However, our results (56.7%) were higher than those of Ischizuka and Kakuta (1996) [25] who reported it in 40–45% of children with adenotonsillar hypertrophy as the cause of snoring, and their results were supported by the use of polysomnography. The difference between our results and results of Ishizuka and Kakuta [25] may be due to the subjective assessment of apnea by parents as they may wrongly note the presence or absence of apneic episodes when the child is asleep; also, there is one limitation of this study as we could not manage to perform a polysomnography (the gold standard for diagnosis of OSA). However, Carroll et al. [26] reported, in 1995, that snoring cannot be reliably distinguished from childhood obstructive sleep apnea by clinical history alone.

Conclusion

From this study, we conclude that adenotonsillar hypertrophy may cause higher PASP in OSA children, PASP was not correlated with age, sex, or duration of

symptoms, and adenotonsillectomy is an effective therapeutic measure in such patients. However, we acknowledge the limitations of this study, in that patient numbers were not large and diagnosis of OSA is based on history not formal sleep testing.

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Authors' contributions
AB performed all surgeries. ME performed echocardiography for all children. AA did statistical analysis. YB was a major contributor in writing the manuscript. The authors read and approved the final manuscript.

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Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the ethics committee of Alexandria University. Informed written consent was obtained from all parents whose children is included in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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References

1. Richardson MA, Cotton RT (1984) Anatomic abnormalities of the pediatric airway. Pediatr Clin North Am. 31(4):821–834
2. Courey MS, Airway obstruction. (1995) The problem and its causes. Otolaryngol Clin North Am. 28(4):673–684
3. Washington RL (2015) 50 years ago in the Journal of Pediatrics: reversible pulmonary hypertension in a child with respiratory obstruction and cor pulmonale. J Pediatr. 167(2):316
4. Bhattacharjee R, Kheirandish-Gozal L, Pillar G, Gozal D (2009) Cardiovascular complications of obstructive sleep apnea syndrome: evidence from children. Prog Cardiovasc Dis. 51(5):416–433
5. Kaditis A (2010) From obstructive sleep apnea in childhood to cardiovascular disease in adulthood: what is the evidence? Sleep. 33(10):1279–1280
6. Shiomi T, Guilleminault C, Stoohs R, Schnittger I (1993) Obstructed breathing in children during sleep monitored by echocardiography. Acta Paediatr. 82(10):863–871
7. Arias MA, Garcia-Rio F, Alonso-Fernandez A, Martinez I, Villamor J (2006) Pulmonary hypertension in obstructive sleep apnoea: effects of continuous positive airway pressure: a randomized, controlled cross-over study. Eur Heart J. 27(9):1106–1113
8. Goldbart AD, Levitas A, Greenberg-Dotan S, Ben Shimol S, Broides A, Puterman M et al (2010) B-type natriuretic peptide and cardiovascular function in young children with obstructive sleep apnea. Chest. 138(3):528–535
9. Badesch DB, Raskob GE, Elliott CG, Krichman AM, Farber HW, Frost AE et al (2010) Pulmonary arterial hypertension: baseline characteristics from the REVEAL Registry. Chest. 137(2):376–387
10. James AL, Runciman M, Burton MJ, Freeland AP (2003) Investigation of cardiac function in children with suspected obstructive sleep apnea. J Otolaryngol. 32(3):151–154
11. Constantin E, Tewfik TL, Brouillette RT (2010) Can the OSA-18 quality-of-life questionnaire detect obstructive sleep apnea in children? Pediatrics. 125(1):e162–e168
12. Kumar DS, Valenzuela D, Kozak FK, Ludemann JP, Moxham JP, Lea J et al (2014) The reliability of clinical tonsil size grading in children. JAMA Otolaryngol Head Neck Surg. 140(11):1034–1037
13. Arezzo E, Festa P, D'Anto V, Michelotti A, De Vincentiis GC, Sitzia E et al (2020) Linguistic adaptation and validation of Italian version of OSA-18, a quality of life questionnaire for evaluation of children with obstructive sleep apnea-hypopnea syndrome (OSAS). Int J Pediatr Otorhinolaryngol. 129:109727
14. Yilmaz MD, Onrat E, Altuntas A, Kaya D, Kahveci OK, Ozel O et al (2005) The effects of tonsillectomy and adenoidectomy on pulmonary arterial pressure in children. Am J Otolaryngol. 26(1):18–21
15. Bady E, Achkar A, Pascal S, Orvoen-Frija E, Laaban JP (2000) Pulmonary arterial hypertension in patients with sleep apnoea syndrome. Thorax. 55(11):934–939
16. Luke MJ, Mehrizi A, Folger GM Jr, Rowe RD (1966) Chronic nasopharyngeal obstruction as a cause of cardiomegaly, cor pulmonale, and pulmonary edema. Pediatrics. 37(5):762–768
17. Miman MC, Kirazli T, Ozyurek R (2000) Doppler echocardiography in adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol. 54(1):21–26
18. Ainger LE (1968) Large tonsils and adenoids in small children with cor pulmonale. Br Heart J. 30(3):356–362
19. Potsic WP (1989) Obstructive sleep apnea. Pediatr Clin North Am. 36(6):1435–1442
20. Kudoh F, Sanai A (1996) Effect of tonsillectomy and adenoidectomy on obese children with sleep-associated breathing disorders. Acta Otolaryngol Suppl. 523:216–218
21. Rosenfeld RM, Green RP (1990) Tonsillectomy and adenoidectomy: changing trends. Ann Otol Rhinol Laryngol. 99(3 Pt 1):187–191
22. Attal P, Bobin S, Lepajolec C, Harboun-Cohen E (1992) Syndrome of obstructive sleep apnea in children. Ann Pediatr (Paris). 39(8):513–517
23. Brouilette R, Hanson D, David R, Klemka L, Szatkowski A, Fernbach S et al (1984) A diagnostic approach to suspected obstructive sleep apnea in children. J Pediatr. 105(1):10–14
24. Richardson MA, Seid AB, Cotton RT, Benton C, Kramer M (1980) Evaluation of tonsils and adenoids in sleep apnea syndrome. Laryngoscope. 90(7 Pt 1):1106–1110
25. Ishizuka Y, Kakuta H (1996) Treatment of sleep apnea and snoring in children. Acta Otolaryngol Suppl. 523:234–235
26. Carroll JL, McColley SA, Marcus CL, Curtis S, Loughlin GM (1995) Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. Chest. 108(3):610–618

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