European Archives of Oto-Rhino-Laryngology (2019) 276:2097–2104
https://doi.org/10.1007/s00405-019-05362-3

MISCELLANEOUS

Obstructive sleep apnea in 2–6 year old children referred for adenotonsillectomy

Britt Øverland[1] · Hanne Berdal[1] · Harriet Akre[2,3]

Received: 5 December 2018 / Accepted: 23 February 2019 / Published online: 6 June 2019

Abstract

Purpose Adenotonsillectomy is one of the most common surgical procedures performed in children. The indications for surgery are either frequent recurrent throat infections or hypertrophy of the tonsils/adenoid vegetation, which can cause obstructive sleep apnea (OSA). There is disagreement regarding the need for sleep studies before adenotonsillectomy to confirm a diagnosis of OSA. Several studies have evaluated questionnaires and physical examination as tools to identify OSA, with conflicting results. The aim of this study was to evaluate the prevalence of OSA among children referred for adenotonsillectomy and whether questionnaires or physical examination can help identify OSA.

Methods This is a prospective cohort study of children aged 2–6 years, referred for adenotonsillectomy. Polysomnography and an otorhinological examination were performed. Tonsillar size and the oral cavity were graded using Friedman's classification and Mallampati score, respectively. The Pediatric Sleep Questionnaire (PSQ) and OSA-18 were also completed.

Results 100 children were included. The prevalence of OSA was 87%, with 52% having moderate to severe OSA. The usefulness of the PSQ and OSA-18 for detecting OSA was evaluated using multiple cutoff points, but none yielded acceptable values for both sensitivity and specificity. In logistic regression analyses predicting different levels of OSA severity, age, Friedman tonsillar size and Mallampati score were weakly associated with OSA.

Conclusions The prevalence of OSA is high among children referred for adenotonsillectomy and questionnaires and clinical characteristics are not sensitive enough to detect the presence or severity of OSA.

Keywords Obstructive sleep apnea · Pediatrics · Adenotonsillectomy

Introduction

Adenotonsillectomy is one of the most common surgical procedures performed in children. The indications for surgery are either frequent recurrent throat infections or hypertrophy of the tonsils/adenoid vegetation. Hypertrophy of the tonsils/adenoid vegetation may cause obstruction of the upper airways with snoring and obstructive sleep apnea (OSA), and the first line treatment of OSA in children is adenotonsillectomy [1, 2].

Polysomnography (PSG) is considered the gold standard for the diagnosis of OSA in children. The recordings are most commonly performed in hospital, during continuous surveillance, with both video and audio recordings. PSG is resource intensive, not widely available and not routinely performed prior to adenotonsillectomy in typically developing children. For most children, a clinical diagnosis of OSA is established based on medical history and medical examination alone, despite the fact that several studies have proven that clinical evaluation alone is not reliable enough to distinguish when OSA is truly present [3–5].

Meta-analyses have shown that adenoidectomy and tonsillectomy lead to significant improvement of OSA in most cases [6, 7]. However, in recent years, it has become apparent that the outcome of adenotonsillectomy may not be as favorable as expected, particularly in cases where the child has severe OSA [8–11]. Because of this, it is of great

✉ Britt Øverland
brov@lds.no

[1] Pediatric and Adult Sleep Disorder Clinic, Lovisenberg Diaconal Hospital, Postboks 4970, Nydalen, 0440 Oslo, Norway

[2] Department of Otorhinolaryngology/Head and Neck Surgery, Oslo University Hospital, Oslo, Norway

[3] Institute of Clinical Medicine, University of Oslo, Oslo, Norway

importance to know whether the child actually has OSA, and if so, its level of severity.

Children with OSA have an increased risk for respiratory compromise postoperatively, as a result of upper airway edema, increased secretion, respiratory depression secondary to analgesic and anaesthetic agents, and post-obstructive relief pulmonary edema [12–16]. The risk for such complications is particularly high among children younger than 3 years, children with severe OSA, and children with additional medical conditions such as obesity and craniofacial anomalies.

The issue of whether PSG should be performed on a routine basis prior to adenotonsillectomy has been the subject of vigorous debate. Surveys of otolaryngology practice patterns have reported that less than 10% of children undergo PSG to confirm a diagnosis of OSA prior to surgery [17, 18]. Practice guidelines published by the pulmonary medicine and paediatric medical community recommend routine use of PSG prior to adenotonsillectomy [19, 20], while communities in ear, nose and throat (ENT) medicine suggest that preoperative PSG is optional and should be performed only in selected situations [21, 22].

In Norway, as in many other countries, the availability of PSG for children is limited. In the ENT department at our hospital, 500 adenoidectomy and/or tonsillectomy procedures are performed each year in children aged 2–6 years. Most of these children are referred from ENT-specialists directly to surgery, and the OSA diagnosis is based on clinical judgement.

The aims of this study were to: (1) determine the prevalence of OSA in typically developing children referred to an ENT-department for adenoidectomy and/or tonsillectomy, and (2) evaluate whether clinical characteristics or questionnaires can predict the presence and severity of OSA.

Method

Patients

This prospective cohort included children aged between 2 and 6 years who were referred to an ENT-department for adenoidectomy and/or tonsillectomy. The main referral sources were specialists in ear, nose and throat diseases, and the children were from both urban and rural areas and were generally representative of the Norwegian population. They were referred for various symptoms, such as mouth breathing, hypertrophy of the tonsils/adenoid vegetation, recurrent throat infections and restless sleep, and most of them presented a history of some degree of snoring. The patients included in the study were randomly selected from the waiting list, using block randomization in which 3 out of every 9 patients were invited to participate. Exclusion criteria were children with craniofacial anomalies, neurological diseases or neuropsychiatric diseases.

Study protocol

The Regional Committee for Medical and Health Research Ethics approved the study protocol (number 2012/117). The parents of the included children provided informed consent.

Clinical data

Clinical measures included a standardized physical examination and questionnaires. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. We transformed an individual child's BMI value to a z score based on the World Health Organization's gender-specific and age-specific reference values. Overweight was defined as a BMI z score of > 1 and obesity as a BMI z score > 2 [23].

Tonsil size was evaluated using the Friedman classification: size 1 tonsils are hidden within the pillars, size 2 tonsils extend to the pillars, size 3 tonsils extend beyond the pillars but not to the midline, and size 4 tonsils extend to the midline [24].

The oral cavity was classified according to Mallampati score [25]. In class 1 the faucial/tonsillar pillars, uvula and soft palate are all visible, in class 2 there is partial visibility of the faucial/tonsillar pillars, uvula and soft palate, in class 3 the base of the uvula, soft and hard palate is visible, and in class 4 only hard palate is visible.

We used two questionnaires: the Pediatric Sleep Questionnaire (PSQ) [26] and the OSA-18 [27]. Both questionnaires were translated into Norwegian by a forward and backward translation procedure. The PSQ can assess a child's risk for OSA and includes three subscales: sleepiness, snoring, and attention/hyperactivity. The questionnaire consists of 22 items with responses of "yes" (= 1), "no" (= 0), or "do not know" (= missing). The total score is calculated as the mean score of non-missing items, with total scores > 0.33 indicating greater risk of OSA. The OSA-18 is a disease-specific quality of life questionnaire for children with OSA. The form contains 18 items grouped into five domains: sleep disturbance, physical suffering, emotional distress, daytime problems and caregiver concerns. Items are scored on an ordinal 7-point scale, where a higher score indicates worse quality of life. A total score below 60 indicates that OSA has a minor impact on quality of life, a score between 60 and 80 indicates a moderate impact and a score above 80 indicates a major impact on quality of life.

Additional variables collected included parent reports of breastfeeding during infancy, prematurity (born before week 37), OSA in a close family member, and whether the child is exposed to tobacco smoke at home.

Polysomnography (PSG)

The PSG (Embla, Resmed, Norway) was performed at the hospital with simultaneous video and audio recordings using the standard recommended set up. Sleep and respiration were scored according to the guidelines for children from American Academy of Sleep Medicine [28].

Apnea–hypopnea index (AHI) was defined as the number of obstructive apneas, mixed apneas and hypopneas per hour of sleep, while oxygen desaturation index (ODI) was defined as the number of oxygen desaturations ≥ 3% per hour of sleep. OSA was defined as an AHI ≥ 1 [29], and was divided into the following categories: mild OSA (AHI 1–4.9), moderate OSA (AHI 5–9.9), and severe OSA (AHI ≥ 10).

Statistics

Statistical analyses were performed using SPSS version 24 (IBM Corp., Armonk, NY). Differences in clinical characteristics between children with various levels of OSA severity were assessed using one-way ANOVA for variables with a normal distribution, the Kruskal–Wallis test for non-normal continuous variables, and either Chi-square test or Fisher's exact test for categorical variables. To evaluate the accuracy of the OSA-18 and PSQ in detecting OSA, sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were calculated. We performed logistic regression analyses with OSA (AHI ≥ 1, AHI ≥ 5, AHI ≥ 10) as the dependent variable, and age, gender, BMI *z* score, Friedman tonsil size and Mallampati score as independent variables.

Results

A total of 100 children who were referred for adenotonsillectomy due to symptoms of infection or upper airway obstruction were included in the study. The overall prevalence of OSA based on PSG was 87%, with 35% having mild OSA, 23% moderate OSA, and 29% having severe OSA. The mean AHI for the sample was 7.7 (range 0–31.7).

The sample had a mean age of 3.6 years, and 42% were girls. The mean BMI *z* score was 0.45, and 71.7% of the children were in the normal weight range, while 8.7% were classified as obese. Most of the children (84%) were of Caucasian ethnicity. None of the children were exposed to tobacco smoke at home. Most of the sample (91.4%) had been breastfed as infants, 8.0% had a premature birth, and 27.8% had a family history of OSA.

The clinical characteristics of children with different severity levels of OSA are summarized in Table 1. We found a significant difference in tonsil size score between the groups. There were no significant differences between the OSA severity groups for any of the other variables.

We calculated sensitivity and specificity for PSQ and OSA-18 using multiple cutoff points for both OSA-18

Table 1 Clinical characteristics at different severity levels of OSA

	n	No OSA AH1 < 1 (*n* = 13)	Mild OSA AHI 1–4.9 (*n* = 35)	Moderate OSA AHI 5–9.9 (*n* = 23)	Severe OSA AHI ≥ 10 (*n* = 29)	*p* value
Age; mean (SD)	100	3.94 (1.33)	3.90 (1.05)	3.27 (0.72)	3.30 (0.89)	0.06
Gender, female; *n* (%)	100	2 (15.4)	18 (51.4)	12 (52.2)	10 (34.5)	0.08
BMI z score; mean (SD)	92	0.05 (1.17)	0.58 (1.01)	0.53 (0.83)	0.43 (1.60)	0.62
BMI z score > 2; *n* (%)	92	0	4 (13.3)	1 (4.5)	3 (10.7)	0.54
Caucasian; *n* (%)	100	11 (84.6)	28 (80.0)	21 (91.3)	24 (82.8)	0.72
Prematurity; *n* (%)	87	0	2 (6.9)	1 (4.8)	4 (14.8)	0.60
Breastfeeding; *n* (%)	92	9 (81.8)	31 (93.9)	21 (95.5)	23 (88.5)	0.47
OSA in family; *n* (%)	90	1 (10.0)	9 (26.5)	6 (28.6)	9 (36.0)	0.52
Mallampati score; *n* (%)	90					
Score 1		4 (36.4)	5 (16.1)	1 (4.8)	1 (3.7)	0.10
Score 2		4 (36.4)	14 (45.2)	8 (38.1)	11 (40.7)	
Score 3		2 (18.2)	10 (32.3)	6 (28.6)	13 (48.1)	
Score 4		1 (9.1)	2 (6.5)	6 (28.6)	2 (7.4)	
Friedman tonsil size	91					
Size 1		6 (54.5)	5 (15.6)	–	2 (7.4)	≤ 0.001
Size 2		2 (18.2)	6 (18.8)	6 (28.6)	3 (11.1)	
Size 3		1 (9.1)	20 (62.5)	10 (47.6)	14 (51.9)	
Size 4		2 (18.2)	1 (3.1)	5 (23.8)	8 (29.6)	

BMI body mass index, *AHI* apnea–hypopnea index, *SD* standard deviation

Table 2 Calculations of the sensitivity, specificity, positive predictive value and negative predictive value for the PSQ score and different levels of OSA-18 score and the apnea–hypopnea index (AHI)

N = 97	PSQ AHI ≥ 1	PSQ AHI ≥ 5	PSQ AHI ≥ 10	OSA-18 ≥ 60 AHI ≥ 1	OSA-18 ≥ 60 AHI ≥ 5	OSA-18 ≥ 60 AHI ≥ 10	OSA-18 ≥ 80 AHI ≥ 1	OSA-18 ≥ 80 AHI ≥ 5	OSA-18 ≥ 80 AHI ≥ 10
Sensitivity	75.6	82.4	85.7	54.7	58.8	67.9	19.8	23.5	28.6
Specificity	45.5	37.0	31.9	45.5	50.0	50.7	90.9	87.0	85.5
PPV	91.6	59.2	33.8	88.7	56.6	35.8	94.4	66.7	44.4
NPV	19.2	65.4	84.6	11.4	52.3	79.5	12.7	50.6	74.7

AHI apnea–hypopnea index, *PSQ* Pediatric Sleep Questionnaire, *PPV* positive predictive value, *NPV* negative predictive value

Table 3 The mean apnea–hypopnea index (AHI) for different Friedman tonsil sizes and Mallampati scores

		AHI Mean (SD)
Friedman tonsil size ($n = 91$)		
1	14.3%	3.69 (6.95)
2	18.7%	6.72 (6.59)
3	49.5%	8.00 (6.93)
4	17.6%	11.38 (8.38)
Mallampati score ($n = 90$)		
1	12.2%	4.17 (6.92)
2	41.1%	7.52 (7.01)
3	34.4%	9.30 (8.09)
4	12.2%	8.06 (6.56)

AHI apnea–hypopnea index, *SD* standard deviation

and the AHI (Table 2). Generally, the specificity was too low when the sensitivity was acceptable, and vice versa. The NPV was highest for AHI ≥ 10 for both PSQ and OSA-18, while the highest PPV was for AHI ≥ 1 for both questionnaires.

The distribution of Friedman tonsil size and Mallampati score is shown in Table 3. Half of the children were classified as tonsil size 3 (49.5%), and most of the children had a Mallampati score of 2 (41.1%) or 3 (34.4%). The distribution of AHI among the distinct groups of Friedman and Mallampati scores is illustrated in Fig. 1. The mean AHI was higher for increasing tonsil size, and the difference was significant. For the Mallampati score, there was a tendency for lower AHI among children with a Mallampati score of 1, but there were no significant differences between the groups.

In univariate logistic analyses, Friedman scores 3 and Mallampati score 3 were significantly associated with OSA (AHI ≥ 1), but only Friedman score 3 remained significant in the multivariate analyses. We also performed logistic analyses predicting moderate/severe (AHI ≥ 5) and severe (AHI ≥ 10) OSA. Age was associated with moderate/severe OSA, and remained significant in the multivariate analyses ($p = 0.01$). Friedman scores 3 and 4 and Mallampati scores 3 and 4 were associated with moderate/severe OSA. In the multivariate analyses, only Friedman score 4 and Mallampati

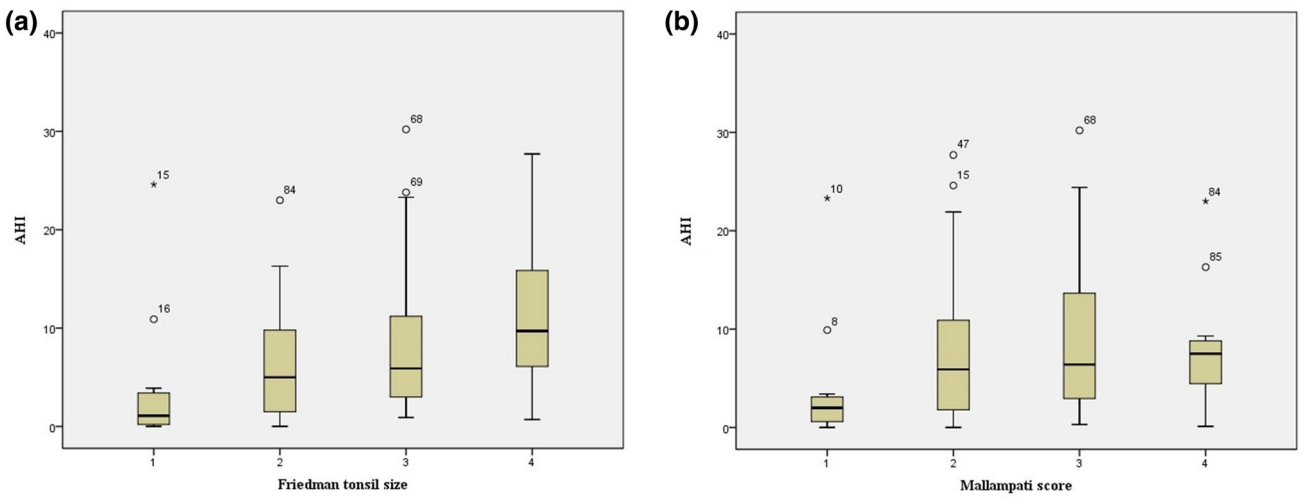

Fig. 1 Distribution of AHI in the different groups of Friedman tonsil size (**a**) and Mallampati score (**b**). *AHI* apnea–hypopnea index

score 4 remained significant ($p < 0.05$) (Table 4). None of the variables were associated with severe OSA.

Discussion

In this sample of children aged 2–6 years referred for adenotonsillectomy, we found a high prevalence of OSA, and as many as 52% had moderate to severe OSA. Lower age, larger tonsil size and higher Mallampati score were associated with OSA to some degree.

Many different risk factors for OSA in children have been described, such as obesity, ethnicity and prematurity [30–32]. There is great variability in the different populations studied, whether they are clinical or population based, and according to age range and ethnicity. Several studies have reached the conclusion that it is difficult to identify a single or combination of clinical parameters that can distinguish primary snoring from OSA, nor a set of clinical parameters that can distinguish different levels of OSA [3, 33, 34].

In our sample of children, we found younger age to be an independent risk factor for moderate/severe OSA, although most other studies have not found age to be a risk factor [34–36]. There are few studies focusing on the young age group we have studied, and according to our findings, it seems that age is an important risk factor amongst the youngest children.

We also found that tonsil size was associated with OSA and moderate/severe OSA to some degree. Our sample of children was in the age span during which the relative size of the tonsils, adenoids and upper airway is most critical [37], and could be an explanation for why tonsil size had a stronger association with OSA in this study than others have found. Prior studies have found conflicting evidence for the benefit of using tonsil size to assess OSA risk [34, 38–41]. One explanation for this has been that the majority of children have tonsils size 2 or 3 [38]. In our study, a large portion (68%) of the children had tonsils size 3 and 4, which may be related to their young age.

For the oral cavity classified on the Mallampati scale, we found an association between OSA and moderate/severe OSA, but only score 4 remained significant for moderate/severe OSA after adjusting for the other risk factors. A couple of other studies have the same findings [42, 43], but there are few studies in children examining the clinical use of the Mallampati score to predict OSA severity. To get a clear visual assessment of the oral cavity and the tonsils, you need to have good cooperation from the child, and that is not always possible. The score is also subjective and depends of the experience of the physician assigning the score, which might explain some of the differences found in the different studies.

Several studies have found an association between obesity and OSA [30, 34, 44], even though the association has been weak in several cases. It has been hypothesized that obesity may play a larger role in white patient samples [45]. This was not the case in our sample, which mainly consisted of white Caucasian children, and we found no association between being overweight or obese and OSA severity.

Environmental tobacco smoke has been described as risk factor for OSA [32, 45]. In our cohort, none of the children

Table 4 Logistic regression analysis predicting OSA as defined by AHI ≥ 1 and AHI ≥ 5

	AHI ≥ 1						AHI ≥ 5					
	Unadjusted			Adjusted			Unadjusted			Adjusted		
	OR	95% CI	*p* value	OR	95% CI	*p* value	OR	95% CI	*p* value	OR	95% CI	*p* value
Age	0.69	0.39–1.19	0.18	0.55	0.23–1.31	0.18	0.52	0.34–0.80	< 0.01	0.42	0.24–0.76	0.01
Gender (reference = female)	0.21	0.05–1.02	0.05	0.16	0.02–1.31	0.09	0.97	0.44–2.16	0.95	1.12	0.40–3.18	0.83
BMI *z* score	1.42	0.82–2.46	0.21	2.12	0.87–5.14	0.10	1.03	0.73–1.46	0.85	1.13	0.75–1.69	0.57
Friedman tonsil size												
Size 1		Reference			Reference			Reference			Reference	
Size 2	6.43	1.03–40.26	0.05	10.98	0.93– 130.35	0.06	6.19	1.04–36.78	0.05	4.59	0.65–32.23	0.13
Size 3	37.71	3.93–362.23	0.01	49.51	2.90– 844.58	0.01	6.29	1.25–31.65	0.03	5.74	0.94–35.0	0.06
Size 4	6.00	0.95–37.76	0.06	3.13	0.28– 34.98	0.35	23.83	3.35–169.39	< 0.01	11.77	1.38–100.11	0.02
Mallampati scoe												
Score 1		Reference			Reference			Reference			Reference	
Score 2	4.71	0.94–23.54	0.06	6.37	0.59–68.58	0.13	4.75	0.90–25.04	0.07	3.61	0.50–26.02	0.20
Score 3	8.29	1.26–54.71	0.03	8.92	0.51–157.48	0.14	7.13	1.31–38.77	0.02	3.83	0.50–29.18	0.20
Score 4	5.71	0.52–62.66	0.15	21.33	0.79–575.82	0.07	12.00	1.58–91.08	0.02	15.67	1.26–195. 18	0.03

CI confidence interval, *AHI* apnea–hypopnea index, *OR* odds ratio

were exposed to tobacco smoke. There has been a great change in Norwegians' smoking habits since a new law was introduced in 2004, and as a result, the number of regular smokers has decreased and most parents do not smoke near their children.

We found no association between OSA in close family members and OSA in this sample of children, despite the proportion having a family history of OSA (28%) being considerably higher than estimated in the Norwegian adult population (16%) [46].

We also wanted to examine whether breastfeeding was associated with the probability of having OSA, since breastfeeding would reduce the risk of infections due to circulating antibodies from the mother. However, we found no such association.

Our calculations of sensitivity, specificity, PPV and NPV indicate that neither the PSQ nor the OSA-18 is a useful screening tool for OSA. None of the cut off scores evaluated yielded acceptable values for both sensitivity and specificity or for both PPV and NPV. These results are consistent with other studies [47, 48] and suggests that the PSQ and OSA-18 are not sufficiently sensitive to detect either the presence or absence of OSA. Most studies that have evaluated the OSA-18 as a tool to diagnose OSA have concluded that the OSA-18 does not correlate with AHI in pediatric patients [47–49], and seems to be more of a tool for evaluating the effect of treatment (adenotonsillectomy). Studies using the PSQ as a tool to detect OSA also have divergent results. It seems like the questionnaire is most useful in combination with other screening tools, and that some of the questions are more predictive than others [26, 50–52].

One limitation of the study is that, even though it used standardized questionnaires that had been rigorously translated into Norwegian for use in this study, the validity and reliability of the Norwegian versions have not been fully evaluated.

In this sample of children referred directly to surgery, there seems to be a good selection process in advance of the referral and, therefore, it may not be necessary to require PSG in advance of surgery. Because of the high prevalence of moderate to severe OSA, and the increased risk of complications, it is important to underline that one should provide special attention to these children during and after surgery and to their postoperative care and observations. There is also an increased risk for having rest OSA after surgery, so the threshold for doing a new PSG after surgery should be low.

Compliance with ethical standards

Conflict of interest Author B. Ø.: declares that se has no conflict of interest. Author H.B.: declares that se has no conflict of interest. Author H. A.: declares that se has no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the national research committee and with the 1964 Helsinki declaration and is later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all participants included in the study.

References

1. Baugh RF, Archer SM, Mitchell RB, Rosenfeld RM, Amin R, Burns JJ, Darrow DH, Giordano T, Litman RS, Li KK, Mannix ME, Schwartz RH, Setzen G, Wald ER, Wall E, Sandberg G, Patel MM (2011) Clinical practice guideline: tonsillectomy in children. Otolaryngol Head Neck Surg 144(1 Suppl):S1–S30. https://doi.org/10.1177/0194599810389949
2. Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, Schechter MS, Sheldon SH, Spruyt K, Ward SD, Lehmann C, Shiffman RN (2012) Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 130(3):576–584. https://doi.org/10.1542/peds.2012-1671
3. Brietzke SE, Katz ES, Roberson DW (2004) Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. Otolaryngol Head Neck Surg 131(6):827–832. https://doi.org/10.1016/j.otohns.2004.07.002
4. Rosen CL (1999) Clinical features of obstructive sleep apnea hypoventilation syndrome in otherwise healthy children. Pediatr Pulmonol 27(6):403–409
5. Suen JS, Arnold JE, Brooks LJ (1995) Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg 121(5):525–530
6. Brietzke SE, Gallagher D (2006) The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: a meta-analysis. Otolaryngol Head Neck Surg 134(6):979–984. https://doi.org/10.1016/j.otohns.2006.02.033
7. Friedman M, Wilson M, Lin HC, Chang HW (2009) Updated systematic review of tonsillectomy and adenoidectomy for treatment of pediatric obstructive sleep apnea/hypopnea syndrome. Otolaryngol Head Neck Surg 140(6):800–808. https://doi.org/10.1016/j.otohns.2009.01.043
8. Amin R, Anthony L, Somers V, Fenchel M, McConnell K, Jefferies J, Willging P, Kalra M, Daniels S (2008) Growth velocity predicts recurrence of sleep-disordered breathing 1 year after adenotonsillectomy. Am J Respir Crit Care Med 177(6):654–659. https://doi.org/10.1164/rccm.200710-1610OC
9. Bhattacharjee R, Kheirandish-Gozal L, Spruyt K, Mitchell RB, Promchiarak J, Simakajornboon N, Kaditis AG, Splaingard D, Splaingard M, Brooks LJ, Marcus CL, Sin S, Arens R, Verhulst SL, Gozal D (2010) Adenotonsillectomy outcomes in treatment of obstructive sleep apnea in children: a multicenter retrospective study. Am J Respir Crit Care Med 182(5):676–683. https://doi.org/10.1164/rccm.200912-1930OC
10. Mitchell RB, Kelly J (2004) Outcome of adenotonsillectomy for severe obstructive sleep apnea in children. Int J Pediatr Otorhinolaryngol 68(11):1375–1379. https://doi.org/10.1016/j.ijporl.2004.04.026
11. Mitchell RB, Kelly J (2007) Outcome of adenotonsillectomy for obstructive sleep apnea in obese and normal-weight children. Otolaryngol Head Neck Surg 137(1):43–48. https://doi.org/10.1016/j.otohns.2007.03.028
12. Brown KA, Morin I, Hickey C, Manoukian JJ, Nixon GM, Brouillette RT (2003) Urgent adenotonsillectomy: an analysis of risk

factors associated with postoperative respiratory morbidity. Anesthesiology 99(3):586–595
13. Gerber ME, O'Connor DM, Adler E, Myer CM 3rd (1996) Selected risk factors in pediatric adenotonsillectomy. Arch Otolaryngol Head Neck Surg 122(8):811–814
14. Lalakea ML, Marquez-Biggs I, Messner AH (1999) Safety of pediatric short-stay tonsillectomy. Arch Otolaryngol Head Neck Surg 125(7):749–752
15. McColley SA, April MM, Carroll JL, Naclerio RM, Loughlin GM (1992) Respiratory compromise after adenotonsillectomy in children with obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 118(9):940–943
16. Rosen GM, Muckle RP, Mahowald MW, Goding GS, Ullevig C (1994) Postoperative respiratory compromise in children with obstructive sleep apnea syndrome: can it be anticipated? Pediatrics 93(5):784–788
17. Mitchell RB, Pereira KD, Friedman NR (2006) Sleep-disordered breathing in children: survey of current practice. Laryngoscope 116(6):956–958. https://doi.org/10.1097/01.MLG.0000216413.22408.FD
18. Weatherly RA, Mai EF, Ruzicka DL, Chervin RD (2003) Identification and evaluation of obstructive sleep apnea prior to adenotonsillectomy in children: a survey of practice patterns. Sleep Med 4(4):297–307
19. American Thoracic Society (1996) Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 153(2):866–878. https://doi.org/10.1164/ajrccm.153.2.8564147
20. Clinical Practice Guideline (2002) Diagnosis and management of childhood obstructive sleep apnea syndrome Pediatrics 109(4):704–712
21. Nath Zallek S, Chervin RD (2000) Improvement in cluster headache after treatment for obstructive sleep apnea. Sleep Med 1(2):135–138
22. Paradise JL (1996) Tonsillectomy and adenoidectomy. Pediatric otolaryngology, 4th edn. Saunders, Philadelphia
23. World Health Organization WHO Global Database on Child Growth and Malnutrition. http://www.who.int/nutgrowthdb. Accessed 27 May 2018
24. Friedman M, Ibrahim H, Joseph NJ (2004) Staging of obstructive sleep apnea/hypopnea syndrome: a guide to appropriate treatment. Laryngoscope 114(3):454–459. https://doi.org/10.1097/00005537-200403000-00013
25. Mallampati SR, Gatt SP, Gugino LD, Desai SP, Waraksa B, Freiberger D, Liu PL (1985) A clinical sign to predict difficult tracheal intubation: a prospective study. Can Anaesth Soc J 32(4):429–434
26. Chervin RD, Hedger K, Dillon JE, Pituch KJ (2000) Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. Sleep Med 1(1):21–32
27. Franco RA Jr, Rosenfeld RM, Rao M (2000) First place–resident clinical science award 1999. Quality of life for children with obstructive sleep apnea. Otolaryngol Head Neck Surg 123(1 Pt 1):9–16. https://doi.org/10.1067/mhn.2000.105254
28. Berry RBBR, Gamaldo CE, Harding SM, Marcus CL, Vaughn BV (eds) (2012) The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications, version 2.0. American Academy of Sleep Medicine, Darien
29. American Academy of Sleep Medicine (2014) International classification of sleep disorders, 3rd edn. American Acedemy of Sleep Medicine, Darien
30. Redline S, Amin R, Beebe D, Chervin RD, Garetz SL, Giordani B, Marcus CL, Moore RH, Rosen CL, Arens R, Gozal D, Katz ES, Mitchell RB, Muzumdar H, Taylor HG, Thomas N, Ellenberg S (2011) The childhood adenotonsillectomy trial (CHAT): rationale, design, and challenges of a randomized controlled trial evaluating a standard surgical procedure in a pediatric population. Sleep 34(11):1509–1517. https://doi.org/10.5665/sleep.1388
31. Rosen CL, Larkin EK, Kirchner HL, Emancipator JL, Bivins SF, Surovec SA, Martin RJ, Redline S (2003) Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr 142(4):383–389. https://doi.org/10.1067/mpd.2003.28
32. Tamanyan K, Walter LM, Davey MJ, Nixon GM, Horne RS, Biggs SN (2016) Risk factors for obstructive sleep apnoea in Australian children. J Paediatr Child Health 52(5):512–517. https://doi.org/10.1111/jpc.13120
33. Certal V, Catumbela E, Winck JC, Azevedo I, Teixeira-Pinto A, Costa-Pereira A (2012) Clinical assessment of pediatric obstructive sleep apnea: a systematic review and meta-analysis. Laryngoscope 122(9):2105–2114. https://doi.org/10.1002/lary.23465
34. Mitchell RB, Garetz S, Moore RH, Rosen CL, Marcus CL, Katz ES, Arens R, Chervin RD, Paruthi S, Amin R, Elden L, Ellenberg SS, Redline S (2015) The use of clinical parameters to predict obstructive sleep apnea syndrome severity in children: the Childhood Adenotonsillectomy (CHAT) study randomized clinical trial. JAMA Otolaryngol Head Neck Surg 141(2):130–136. https://doi.org/10.1001/jamaoto.2014.3049
35. Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G (1999) Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 159(5 Pt 1):1527–1532. https://doi.org/10.1164/ajrccm.159.5.9809079
36. Goodwin JL, Kaemingk KL, Fregosi RF, Rosen GM, Morgan WJ, Sherrill DL, Quan SF (2003) Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children—the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep 26(5):587–591
37. Jeans WD, Fernando DC, Maw AR, Leighton BC (1981) A longitudinal study of the growth of the nasopharynx and its contents in normal children. Br J Radiol 54(638):117–121. https://doi.org/10.1259/0007-1285-54-638-117
38. Howard NS, Brietzke SE (2009) Pediatric tonsil size: objective vs subjective measurements correlated to overnight polysomnogram. Otolaryngol Head Neck Surg 140(5):675–681. https://doi.org/10.1016/j.otohns.2009.01.008
39. Nolan J, Brietzke SE (2011) Systematic review of pediatric tonsil size and polysomnogram-measured obstructive sleep apnea severity. Otolaryngol Head Neck Surg 144(6):844–850. https://doi.org/10.1177/0194599811400683
40. Tang A, Benke JR, Cohen AP, Ishman SL (2015) Influence of tonsillar size on OSA improvement in children undergoing adenotonsillectomy. Otolaryngol Head Neck Surg 153(2):281–285. https://doi.org/10.1177/0194599815583459
41. Bixler EO, Vgontzas AN, Lin HM, Liao D, Calhoun S, Vela-Bueno A, Fedok F, Vlasic V, Graff G (2009) Sleep disordered breathing in children in a general population sample: prevalence and risk factors. Sleep 32(6):731–736
42. Guilleminault C, Huang YS, Glamann C, Li K, Chan A (2007) Adenotonsillectomy and obstructive sleep apnea in children: a prospective survey. Otolaryngol Head Neck Surg 136(2):169–175. https://doi.org/10.1016/j.otohns.2006.09.021
43. Kumar HV, Schroeder JW, Gang Z, Sheldon SH (2014) Mallampati score and pediatric obstructive sleep apnea. J Clin Sleep Med 10(9):985–990. https://doi.org/10.5664/jcsm.4032
44. Kohler M, Lushington K, Couper R, Martin J, van den Heuvel C, Pamula Y, Kennedy D (2008) Obesity and risk of sleep related upper airway obstruction in Caucasian children. J Clin Sleep Med 4(2):129–136
45. Weinstock TG, Rosen CL, Marcus CL, Garetz S, Mitchell RB, Amin R, Paruthi S, Katz E, Arens R, Weng J, Ross K, Chervin

RD, Ellenberg S, Wang R, Redline S (2014) Predictors of obstructive sleep apnea severity in adenotonsillectomy candidates. Sleep 37(2):261–269. https://doi.org/10.5665/sleep.3394
46. Hrubos-Strom H, Randby A, Namtvedt SK, Kristiansen HA, Einvik G, Benth J, Somers VK, Nordhus IH, Russell MB, Dammen T, Omland T, Kvaerner KJ (2011) A Norwegian population-based study on the risk and prevalence of obstructive sleep apnea. The Akershus Sleep Apnea Project (ASAP). J Sleep Res 20(1 Pt 2):162–170. https://doi.org/10.1111/j.1365-2869.2010.00861.x
47. Baldassari CM, Alam L, Vigilar M, Benke J, Martin C, Ishman S (2014) Correlation between REM AHI and quality-of-life scores in children with sleep-disordered breathing. Otolaryngol Head Neck Surg 151(4):687–691. https://doi.org/10.1177/0194599814547504
48. Ishman SL, Yang CJ, Cohen AP, Benke JR, Meinzen-Derr JK, Anderson RM, Madden ME, Tabangin ME (2015) Is the OSA-18 predictive of obstructive sleep apnea: comparison to polysomnography. Laryngoscope 125(6):1491–1495. https://doi.org/10.1002/lary.25098
49. Kobayashi R, Miyazaki S, Karaki M, Hoshikawa H, Nakata S, Hara H, Kodama S, Kikuchi A, Kitamura T, Mori N (2014) Evaluation of adenotonsillectomy and tonsillectomy for pediatric obstructive sleep apnea by rhinomanometry and the OSA-18 questionnaire. Acta Otolaryngol 134(8):818–823. https://doi.org/10.3109/00016489.2014.905703
50. Bertran K, Mesa T, Rosso K, Krakowiak MJ, Pincheira E, Brockmann PE (2015) Diagnostic accuracy of the Spanish version of the pediatric sleep questionnaire for screening of obstructive sleep apnea in habitually snoring children. Sleep Med 16(5):631–636. https://doi.org/10.1016/j.sleep.2014.10.024
51. Certal V, Silva H, Carvalho C, Costa-Pereira A, Azevedo I, Winck J, Capasso R, Camacho M (2015) Model for prediction of pediatric OSA: proposal for a clinical decision rule. Laryngoscope 125(12):2823–2827. https://doi.org/10.1002/lary.25438
52. Pena-Zarza JA, Osona-Rodriguez de Torres B, Gil-Sanchez JA, Figuerola-Mulet J (2012) Utility of the pediatric sleep questionnaire and pulse oximetry as screening tools in pediatric patients with suspected obstructive sleep apnea syndrome. Sleep Disord 2012:819035. https://doi.org/10.1155/2012/819035