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Original Article

The Valence Sleep Score - A Simple Clinical Tool for the Diagnosis of Obstructive Sleep Apnea in Children

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- Obstructive Sleep Apnea; Family history; Nocturnal enuresis; Dental malocclusion; Narrow palate; Friedman score

Abstract

Objectives: Obstructive Sleep Apnea (OSA) is the ultimate stage of sleep disorders breathing. It concerns between 1.2 and 5.2% of the pediatric population, and its prevalence is probably underestimated. Polysomnography is the gold standard for diagnosis but it is an expensive procedure and the difficulty to access makes it necessary to establish clinical score for early diagnosis and treatment. We create a simple clinical tool, compared to home sleep apnea testing (HSAT), including patient history and physical examination, to diagnose OSA in children.

Methods: Prospective, observational multi-center study that included children who underwent clinical examination and HSAT. OSA was identified by respiratory disturbance indices commonly applied in clinical practice. A ROC curve was calculated for each sign and a combined score was calculated with the most important clinical symptoms.

Results: A total of 119 children were included, 108 were classified as having OSA, 11 as primary snorers. The Valence sleep score (VSS) is a combination of signs (scored 1 if the sign is present and 0 if it is not) and the Friedman score: *Family history of OSA + Nocturnal enuresis + Dental Malocclusion + Narrow palate + Friedman Score*. For a VSS ≥ 5, specificity was 81.8%, positive predictive value was 97%, positive likelihood ratio was 3.42, and negative predictive value was 0.46.

Conclusion: VSS can be used to diagnose OSA in children to enable those with a score ≥ 5 to receive early treatment.

ABBREVIATIONS

HSAT: Home Sleep Apnea Testing, OSA: Obstructive Sleep Apnea, PSG: Polysomnography, REI: Respiratory Event Index, SCR: Sleep Clinical Record, SDB: Sleep Disorders Breathing, VSS: Valence Sleep Score

INTRODUCTION

The spectrum of sleep disordered breathing (SDB) in children evolves from primary snoring through upper airway resistance syndrome and finally OSA [1]. The latter is characterized by partial (hypopnea) or total (apnea) interruption of the airway during sleep, resulting in altered gas exchange and a decrease quality of sleep for the child. It concerns between 1.2 and 5.2 % of the pediatric population [2] and it is probably underestimated.

The American Academy of Sleep Medicine (AASM) has proposed practice parameters to establish the indication for polysomnography in children with suspected SDB. The authors established that the assessment of SDB mostly based on clinical evaluation is insufficient for the diagnosis of OSA, which must be confirmed by polysomnography (PSG) [3]. However, even if PSG remains the gold standard for the diagnosis, this procedure is expensive, and its accessibility is limited to a minority of cases due to the low number of pediatric sleep laboratories available. This can delay the diagnosis and the treatment of patients with SDB. The Home sleep apnea testing (HSAT) is frequently used as a diagnostic technique for OSA in adults. The French health authorities (*Haute Autorité de Santé*, HAS) indicated that it can be used as an alternative to PSG when it is performed and interpreted by specialists of SDB in children [4].

Several authors have proposed different clinical scores to create a simple and reproducible instrument to screen patients at high risk of OSA [5-7]. However, the main limitation of these scores is that most consider patient history and physical examination separately. Among those that have considered these aspects together, Villa et al. published in 2012 a study that aimed to develop, in a large sample of children, a clinical history and physical examination -based score to diagnose SDB and compared this to PSG; this Sleep Clinical Record (SCR) had a sensitivity of 96%, a specificity of 67%, a positive likelihood ratio of 2.91, and a negative likelihood ratio of 0.06 [8]. However, the SCR is calculated using a complicated formula with coefficients, which seems difficult to use in everyday practice. The aim of the present study was therefore to create a simple and HSAT-validated tool considering both the subject's clinical history and physical examination to diagnose SDB and to compare it to HSAT and SCR.

MATERIALS AND METHODS

A prospective, observational, and multi-center study was conducted between December 2015 and June 2017 in all consecutive patients aged 2 to 16 years (the upper limit of pediatrics in France) who were referred to specialists for sleep evaluation in the ENT and cervico-facial surgery unit of the hospital of Valence (France) and a clinicians' office specialized in children sleep in Lyon (France).

Score development

For the development of the Valence Sleep Score (VSS) both patient history and physical examination were considered, and this score was constructed on the basis of physician experience and literature describing the clinical signs of OSA [8,9].

The first part of the study consisted of collecting information on the patient medical background: familial history of SDB, personal neurological or cardiopulmonary disease, personal allergy, and treatment; nocturnal symptoms: presence of witnessed apneic episodes, abnormally long sleep latency, regular snoring, agitated sleep, nocturnal awakenings, nocturnal sweating, enuresis, cervical hyperextension sleeping position, hypersalivation with persistence of bed-wetting; diurnal symptoms: difficulty to wake up in the morning, daytime somnolence (in class, in transport), increasing need for daytime naps, morning headache, and attention deficit/hyperactivity disorder.

The second part consisted of collection of data from the physical examination of the oropharynx, the dental occlusion, the nose and the facial aspect. Nose: collapse of the nasal valves during inspiration, deviated septum, enlargement of the inferior nasal turbinate; signs of chronic otitis; orthodontic examination: class II or III abnormal dental occlusion, open-bite, deep-bite or overjet; oral and oropharyngeal examination: presence of a narrow palate, primary deglutition, macroglossia, a long face syndrome, circles, labial incompetence. Tonsillar hypertrophy was classified according to the Friedman standardized scale [10,11], class 3 and 4 were considered as positive. Position of the palate was classified according the Mallampati scale [12,13]; class 3 and 4 were considered as positive. Body mass index and growth curve were also recorded.

Home sleep apnea testing

A one-night home sleep apnea testing was performed using a Nox 3 Portable Sleep Monitor™ (Reykjavik, Iceland), a T3 device [14], and analyzed by a pediatric sleep specialist: one in Valence, two in Lyon. The following signals were recorded: thoraco-abdominal efforts by strain gauges, arterial oxygen saturation and pulse by pulse oximeter, snoring by a snore sensor, oronasal airflow by thermocouple and nasal pressure by nasal cannula.

Apnea was defined as a respiratory flow decrease of more than 90% for at least 10 seconds. Hypopnea was defined as a decrease of respiratory flow of more than 30% associated with a desaturation of at least 3%. Respiratory Event Index (REI) was defined as the mean number of apnea and hypopnea episodes per hour of sleep. The criteria established by the AASM to define central, obstructive and mixed apnea were used [15]: OSA was defined as an obstructive REI > 1.5/h total sleep time (TST). Mild OSA was defined as REI > 1.5 and < 5/h of TST, moderate OSA as REI > 5 and <10/h TST, and severe OSA as REI > 10/h TST. The scorer (F.C.) was blinded to the VSS results. If there was a strong clinical suspicion of OSA, and the completion of HSAT could delay treatment, the child was treated immediately.

Statistical analysis

To create the VSS, several combinations were given for each item. The sensitivity and specificity for each sign were compared to the HSAT results to test different combinations and choose the one that offered the best combination of sensitivity, specificity, positive predictive value and negative predictive value.

Differences between patients with and without OSA diagnosis were assessed using t-tests for continuous variables and chi-squared tests for categorical variables. The sensitivity, specificity, and positive and negative predictive values of all the items of case history and clinical examination were assessed using receiver operating characteristic (ROC) curves [16], constructed using SPSS software (version 20.0; SPSS Inc., Chicago, IL, USA), based on REI from the HSAT. The area under the curve was then calculated. The most statistically significant items among those that were clinically interesting were chosen to create the combined VSS. To construct the combined score, R stat was used (17). We chose the combined score with the AUC the greatest. Sensitivity, specificity, positive and negative predictive values for VSS ≥ 3, ≥ 4, ≥ 5 and ≥ 6 were calculated. The VSS was compared with the SCR with DeLong's test for two correlated ROC curves. A p-value < 0.05 was considered significant.

The local ethics committee approved the study protocol.

RESULTS

Two hundred twenty-two children consulted for SDB between December 2015 to June 2017, and among these 119 (53.6%) had a HSAT performed. Those for whom HSAT was not performed received adenotonsillectomy (n=53), orthodontic treatment (n=7), or medical treatment (n=3); others were not suspected of having OSA (n=6), or did not go to HSAT appointment (n=34). Among those who had HSAT, the mean ±SD age was 8.03 ± 3.1 years, and there were 66 (55.5%) boys. Fourteen (13%) children were recruited in Lyon, and 94 (87%) in the Valence center. One hundred and eight were classified as having OSA after HSAT, and eleven were classified as primary snorers. The prevalence of OSA was 90.8%. The mean ± SD REI was 3.77 events/h ± 3.02 (range: 0 - 17.7 events/h).

A ROC curve for each symptom was established. The highest AUCs were found for dental malocclusion, headache, Friedman score, narrow palate, supra-occlusion, agitated sleep, family history of OSA, difficulty to wake up in the morning, nocturnal enuresis, and primary deglutition Patients with OSA suffered significantly more often of headache, and had more often dental malocclusion and a narrow palate than patients with primary snoring ($p < 0.05$; Table 1).

Five combined scores were calculated (Figure 1) and the score with the greatest AUC (Score 5) was retained (hereafter, VSS; Table 2). The VSS is a combination of 5 signs which were noted 1 or 0 if the sign is present or not: *Family history of OSA*

Table 1: Clinical and anthropometric parameters.

	OSA (n = 108)	Primary snoring (n = 11)	p value	AUC	p AUC
Demographic data					
Age, years	8.1 ± 3.0	7.7 ± 3.7	0.43*	0.554	0.56
Male/Female gender ratio	1.3:1	0.8:1	0.48	0.445	0.54
BMI, kg/m^2	16.9 ± 3.5	16.9 ± 2.7	0.67*	0.468	0.72
History					
Family history of OSA	53 (49)	3 (27.3)	0.17	0.609	0.14
Allergy, Atopy	24 (22.2)	3 (27.2)	0.70	0.475	0.78
Apneic events	15 (13.9)	3 (27.3)	0.24	0.433	0.46
Hard to wake up	62 (57.4)	4 (37)	0.18	0.605	0.25
Abnormally long sleep latency	41 (37.9)	7 (63.6)	0.10	0.372	0.16
Regular snoring	54 (50)	6 (54.5)	0.77	0.477	0.80
Agitated sleep	83 (76.8)	7 (63.6)	0.33	0.614	0.21
Nocturnal awakenings	26 (24.1)	3 (27.3)	0.81	0.452	0.60
Nocturnal sweating	55 (50.9)	5 (45.4)	0.73	0.527	0.76
Nocturnal enuresis	41 (37.9)	2 (18.2)	0.19	0.599	0.13
Hyperextension of the head	30 (27.8)	6 (54.5)	0.07	0.366	0.14
Nocturnal drooling	71 (65.7)	9 (81.8)	0.28	0.420	0.38
Daytime sleepiness	58 (53.7)	6 (54.5)	0.96	0.496	0.96
Intrusive naps	25 (23)	4 (37)	0.33	0.434	0.47
Headache	30 (27.8)	0 (0)	**0.04**	0.639	0.13
Learning difficulties, hyperactivity	58 (53.7)	4 (37)	0.27	0.587	0.34
Physical examination					
Growth problem	10 (9.2)	0 (0)	0.29	0.546	0.61
Dark circles	89 (82.4)	8 (72.7)	0.43	0.548	0.59
Oral breathing	94 (87)	11 (100)	0.20	0.435	0.48
Malocclusion	90 (83.3)	6 (54.5)	**0.02**	0.644	0.07
Adenoid phenotype	34 (31.4)	3 (27.3)	0.77	0.521	0.82
Narrow palate	86 (79.6)	6 (54.5)	**0.05**	0.625	0.12
Open-bite	26 (24)	3 (27.3)	0.91	0.485	0.87
Supra-occlusion	45 (41.7)	2 (18.2)	0.13	0.617	0.20
Over-jet	41 (37.9)	3 (27.3)	0.48	0.553	0.56
Primary deglutition	96 (88.9)	8 (72.7)	0.12	0.581	0.38
Lips incompetence	7 (6.5)	1 (9.1)	0.74	0.487	0.89
Macroglossia	6 (5.5)	0 (0)	0.42	0.528	0.76
Friedman score	57 (52.8)	3 (27.3)	0.10	0.637	0.09
Mallampati score	5 (4.6)	1 (9.1)	0.52	0.390	0.23
Turbinal hypertrophy	5 (4.6)	0 (0)	0.47	0.523	0.80
Septum nose deviation	5 (4.6)	0 (0)	0.47	0.523	0.80
Recurrent otitis media, otitis with effusion	12 (11.1)	0 (0)	0.24	0.556	0.54

Data are presented as n (%) or mean +/- SD, unless otherwise stated. *p-value calculated by t-test; others calculated by Chi-squared test. AUC: area under the curve. In bold are significative parameters.

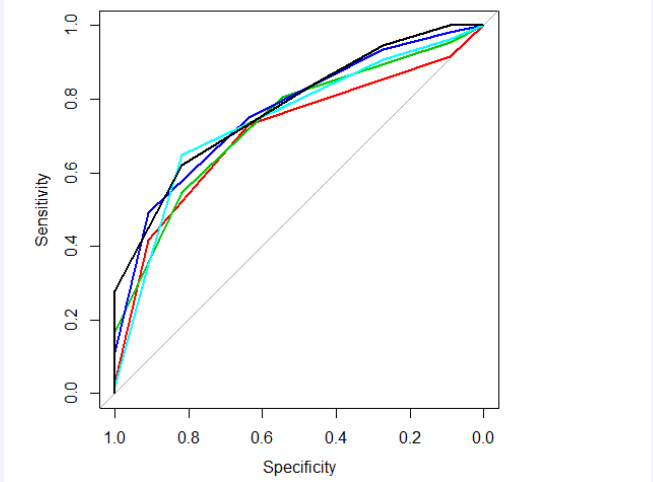

Figure 1 Receiving operating curve analysis.

Table 2: AUC of combined scores.

Combined score	AUC	p
Score 1	0.713	$< 10^{-3}$
Score 2	0.738	$< 10^{-3}$
Score 3	0.767	$< 10^{-5}$
Score 4	0.746	$< 10^{-3}$
Score 5 (VSS)	0.778	$< 10^{-5}$
SCR	0.680	0.04

p-value calculated by DeLong's test for two correlated ROC curves
Score 1 (Malocclusion + Friedman score)
Score 2 (Malocclusion + Friedman score + Nocturnal enuresis)
Score 3 (Malocclusion + Friedman score + Nocturnal enuresis + Narrow palate)
Score 4 (Malocclusion + Friedman score + Narrow palate)
VSS (Malocclusion + Friedman score + Narrow palate + Nocturnal enuresis + Familial history of OSA)

(0 if not, 1 if present) + Nocturnal enuresis (0 if not, 1 if present) + Dental Malocclusion (0 if class 1, 1 if class 2 or 3) + Narrow palate (0 if not, 1 if present) + Friedman Score (1 if stage 1, 2 if stage 2, 3 if stage 3, 4 if stage 4). The minimum score is 0, maximum 8. The AUC of the VSS was estimated to be 0.778 ($p < 10^{-5}$) and that of the SCR to be 0.680 (p = 0.04; Table 2). The AUCs of VSS and SCR were not significantly different (p = 0.217; Figure 2).

A VSS ≥ 5 represented the best combination of sensitivity, specificity, positive and negative predictive values. A VSS ≥ 5 had sensitivity of 62.2% and a specificity of 81.8% (Table 3). A positive VSS (≥ 5) had a positive likelihood ratio of 3.42 which increased the probability of having OSA to 97%. A negative or inconclusive VSS (<5) had a negative likelihood ratio of 0.46 which lowered the probability of having OSA of 82%.

DISCUSSION

This prospective study reports the VSS that is based on a combination of five clinical items, and that it had a high specificity and a high positive likelihood ratio demonstrating its capacity for the diagnosis of OSA in children. A cut-off ≥ 5 provided a high specificity and a high predictive positive value.

Several studies have been conducted to define a clinical score to identify patients with OSA in order to prioritize polysomnography and allow prompt treatment [5–8,18–21]. This objective is motivated by the complications secondary to repetitive apnea and hypopneas during sleep, leading to intermittent hypoxia, oxidative stress, inflammation [22–24], increased sympathetic activity [25–29], increased serum cortisol [30], increased insulin-resistance [31,32]. Moreover, Li et al. reported an elevation of blood pressure even in primary snoring children [33]. That is why around half of the included patients were immediately treated after consultation, even before realization of HSAT. This attitude was taken if the HSAT delayed the treatment of a child on which there was no doubt about the positive diagnosis of OSA.

Brouillette et al., was the first to propose a clinical score based on three items of case history and calculated as follows: 1.42D + 1.41A + 0.71S – 3.83 where D is dyspnea during sleep, A is observed apnea, and S is snoring;a score < -1 being indicative of the absence of OSA and a score > 3.5 indicative of OSA [5]. Even if it was a widely used score that had high sensitivity and high specificity, it could be adversely affected by the subjective nature of the questions, and its application in specific patient populations can be questioned [35]. Another well-accepted tool is the questionnaire developed by Chervin et al., the Pediatric Sleep Questionnaire. A positive response for more than 7 of the 22 items composing the questionnaire indicates possible OSA, and has 85% sensitivity and 81% specificity for the diagnosis of OSA. However, it remains a subjective scale completed by parents, and

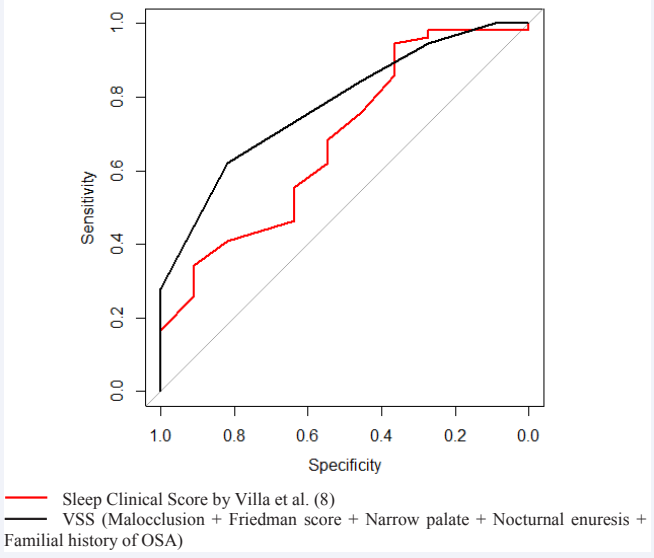

Figure 2 Receiving Operating Characteristics Curve Analysis.

Table 3: Sensitivity, specificity, positive predictive value and negative predictive value for each VSS score.

VSS	Sensitivity	Specificity	Positive predictive value	Negative predictive value
≥ 3	94.3%	27.3%	92.8%	29.7%
≥ 4	83.9%	45.4%	93.9%	21.8%
≥ 5	62.2%	81.8%	97%	19%
≥ 6	28.3%	100%	100%	12%

the authors indicate that it can be useful for research but not for most individual patients [6].

To the best of our knowledge, the first to have reported the development of a composite score including both case history and physical examination is Xu et al., who proposed six predictors of OAH: observable apnea, nocturnal enuresis, intrusive naps, mouth breathing observed, tonsillar hypertrophy, and upper airway narrowing on radiography; this score had a sensitivity of 93.5% for the detection of OSA. Nevertheless, the small sample size, and the use of an REI > 5 as a cut-off can deserved this study [7].

Villa et al., aimed to develop a composite tool to screen children with SDB; this SCR, based on a sample of 279 children, was defined as 2*oral breathing + 2*nasal obstruction + 2* septum nose deviation + 2*tonsillar hypertrophy + 2*Friedman palate position (III-IV) + 2*dental/skeletal malocclusion + 2*narrow palate + 2*phenotype + 0.5*Brouillette score + 0.5*other neurological symptoms + 1*hyperactivity/attention rating scale. A total score >6 is positive. It had a sensitivity of 96% and a negative likelihood ratio of 0.06 and can be useful to exclude the diagnosis of SDB in suspected children. However, this instrument is not useful to identify patients with OSA owing to a the lack of specificity (67%) and positive predictive value (88%), and authors conclude that the SCR might be helpful to screen patients candidate for PSG for suspected OSA [36]. In addition, its applicability could be difficult in daily practice as it included 11 items with different coefficient and questionnaire.

The strength of the VSS is its ease of use as it is composed of only five items, and the relative simplicity of the questionnaire (both the parents and the operator providing yes/no replies according to whether the symptom is present or not. The physician notes the Friedman score. Its accuracy was higher and not significantly different from the score developed by Villa et al., we applied to the study population, while being far simpler. Moreover, several parents noticed that the siblings of the patient also presented some of the characteristics included in the questionnaire and have asked to consult for these children. This could be considered as a first step for OSA detection in the general population.

There are, however, some limitations in this study that should be mentioned. The primary limitation of this study is the heterogeneity of sample between the two groups of patients who performed the HSAT. The difference is explained because of the patients' recruitment method, who were referred to our centre by general physicians or orthodontists for high suspicious of OSA. Some children were assessed in our consultation after the HSAT results, during orthodontic treatment or for surgical treatment. Then, all patients sent to our hospital were enrolled, including children from two to 16 years. However, it is known that OSA presents itself in different ways depending on the patient. A 3-year-old toddler will have more often eating disorders with poor eating resulting in a failure to thrive and repetitive upper respiratory tract infection, while a teenager will complain about headaches and difficulties to wake up in the morning [9]. That is why authors do not agree with the definition of children OSA and some says that the disease is completely different between children and teenagers. Although, PSG is the gold standard for the diagnosis of SDB and using HSAT can provide bias for the diagnostic of OSA in children. First, some technical problems could occur, in particular with the registration by cannulas monitored by parents at home [20]. Then, the registration time by HSAT is different than the sleep time of PSG and it is easy to understand that errors could appear by overestimation or underestimation the REI. Third, the HSAT miss some hypopneas, causing arousals without desaturation, leading to underestimate REI. Tan et al. explained that basing the therapeutic management decision on HSAT instead of PSG results changed the clinical management in 23% of all patients, particularly in children with mild and moderate obstructive sleep apnea (1 < REI < 10/h total sleep time) [37].

Our clinical centers are not equipped with PSG and the HSAT as standard to diagnosis OSA or primary snoring in children was chosen. This choice is supported by the HAS recommendation of May 2012 which clearly insists that despite the fact that HSAT is not formally validated by literature in children, it is possible to use it if it is conducted and interpreted by a children respiratory sleep disorders specialists [4].

Furthermore, a child who sleeps at home will spend a better night in natural and comfortable environment than a child who performs a PSG in a specialized laboratory.

In addition, Alonso-Alvarez et al. showed that HSAT provides a reasonably valid alternative to in-laboratory PSG for the diagnosis of OSA in children with a high index of clinical suspicion for the presence of OSA. They defined a HSAT cut-off Obstructive Respiratory Disturbance Index (ORDI) as 5.6 per hour which can be associated with a PSG ORDI at 3 per hour, this approach exhibited excellent sensitivity at 90.9% and specificity at 94.1% [27]. The French criteria based on the AASM Rules for scoring respiratory events in sleep [15] was chosen, with a positive IAH defined > 1.5.

CONCLUSION

Our prospective study reports that the VSS score is a simple, and HSAT-validated tool, with five items score which combine information on history case and on the physical examination, on a large size sample. It can be useful to the positive diagnosis of children with OSA due to its high specificity of 81.8% and high

positive predictive value of 97%. In addition, it can be a helpful tool for parental screening of alerts signs in siblings. A PSG-validated study remains necessary to compare this score to the gold standard for the diagnosis of OSA.

REFERENCES

1. Guilleminault C, Pelayo R, Leger D, Clerk A, Bocian RC. Recognition of sleep-disordered breathing in children. Pediatrics. 1996; 98: 871-82.
2. Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012; 130: e714–755.
3. Aurora RN, Zak RS, Karippot A, Lamm CI, Morgenthaler TI, Auerbach SH, et al. Practice parameters for the respiratory indications for polysomnography in children. Sleep. 2011; 34: 379-88.
4. Haute Autorité de Santé - Place et conditions de réalisation de la polysomnographie et de la polygraphie respiratoire dans les troubles du sommeil - Rapport d'évaluation [Internet]. 2016.
5. Brouilette R, Hanson D, David R, Klemka L, Szatkowski A, Fernbach S, et al. A diagnostic approach to suspected obstructive sleep apnea in children. J Pediatr. 1984; 105: 10–14.
6. Chervin RD, Weatherly RA, Garetz SL, Ruzicka DL, Giordani BJ, Hodges EK, et al. Pediatric sleep questionnaire: prediction of sleep apnea and outcomes. Arch Otolaryngol Head Neck Surg. 2007; 133: 216–222.
7. Villa MP, Paolino MC, Castaldo R, Vanacore N, Rizzoli A, Miano S, et al. Sleep clinical record: an aid to rapid and accurate diagnosis of paediatric sleep disordered breathing. The European Respiratory Journal. 2013; 41: 1355–1361.
8. Guilleminault C, Lee JH, Chan A. Pediatric obstructive sleep apnea syndrome. Archives of Pediatrics & Adolescent Medicine. 2005; 159: 775–785.
9. Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc. 2008; 5: 242–252.
10. Friedman M, Tanyeri H, La Rosa M, Landsberg R, Vaidyanathan K, Pieri S, et al. Clinical predictors of obstructive sleep apnea. Laryngoscope. 1999; 109: 1901-7.
11. Mallampati SR, Gatt SP, Gugino LD, Desai SP, Waraksa B, Freiberger D, et al. A clinical sign to predict difficult tracheal intubation: a prospective study. Can Anaesth Soc J. 1985; 32: 429-34.
12. Liistro G, Rombaux P, Belge C, Dury M, Aubert G, Rodenstein DO. High Mallampati score and nasal obstruction are associated risk factors for obstructive sleep apnoea. Eur Respir J. 2003; 21: 248–252.
13. Ferber R, Millman R, Coppola M, Fleetham J, Murray CF, Iber C, et al. Portable recording in the assessment of obstructive sleep apnea. ASDA standards of practice. Sleep. 1994; 17: 378-92.
14. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med. 2012; 8: 597–619.
15. Robin X, Turck N, Hainard A, Tiberti N, Lisacek F, Sanchez J-C, et al. pROC: an open-source package for R and S+ to analyze and compare ROC curves. BMC Bioinformatics. 2011; 12: 77.
16. R Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. 2013.
17. Kadmon G, Shapiro CM, Chung SA, Gozal D. Validation of a pediatric obstructive sleep apnea screening tool. Int J Pediatr Otorhinolaryngol. 2013; 77: 1461–1464.
18. Xu Z, Cheuk DKL, Lee SL. Clinical evaluation in predicting childhood obstructive sleep apnea. Chest. 2006; 130: 1765–1771.
19. Brietzke SE, Katz ES, Roberson DW. Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. Otolaryngol Head Neck Surg. 2004; 131: 827–832.
20. Brockmann PE, Schaefer C, Poets A, Poets CF, Urschitz MS. Diagnosis of obstructive sleep apnea in children: a systematic review. Sleep Med Rev. 2013; 17: 331–340.
21. Spruyt K, Gozal D. Pediatric sleep questionnaires as diagnostic or epidemiological tools: a review of currently available instruments. Sleep Med Rev. 2011; 15: 19–32.
22. Larkin EK, Rosen CL, Kirchner HL, Storfer-Isser A, Emancipator JL, Johnson NL, et al. Variation of C-reactive protein levels in adolescents: association with sleep-disordered breathing and sleep duration. Circulation. 2005; 111: 1978–1984.
23. Kaditis A, Gozal D, Snow AB, Kheirandish-Gozal L, Alexopoulos E, Varlami V, et al. Uric acid excretion in North American and Southeast European children with obstructive sleep apnea. Sleep Medicine. 2010; 11: 489–493.
24. Tauman R, O'Brien LM, Gozal D. Hypoxemia and obesity modulate plasma C-reactive protein and interleukin-6 levels in sleep-disordered breathing. Sleep Breath. 2007; 11: 77–84.
25. Baharav A, Kotagal S, Rubin BK, Pratt J, Akselrod S. Autonomic cardiovascular control in children with obstructive sleep apnea. Clin Auton Res. 1999; 9: 345-51.
26. Bhattacharjee R, Kheirandish-Gozal L, Pillar G, Gozal D. Cardiovascular complications of obstructive sleep apnea syndrome: evidence from children. Prog Cardiovasc Dis. 2009; 51: 416-33.
27. Muzumdar HV, Sin S, Nikova M, Gates G, Kim D, Arens R. Changes in heart rate variability after adenotonsillectomy in children with obstructive sleep apnea. Chest. 2011; 139: 1050–1059.
28. Aljadeff G, Gozal D, Schechtman VL, Burrell B, Harper RM, Ward SL. Heart rate variability in children with obstructive sleep apnea. Sleep. 1997; 20: 151-7.
29. Kaditis AG, Alexopoulos EI, Damani E, Hatzi F, Chaidas K, Kostopoulou T, et al. Urine levels of catecholamines in Greek children with obstructive sleep-disordered breathing. Pediatr Pulmonol. 2009; 44: 38–45.
30. Bratel T, Wennlund A, Carlström K. Pituitary reactivity, androgens and catecholamines in obstructive sleep apnoea. Effects of continuous positive airway pressure treatment (CPAP). Respir Med. 1999; 93: 1–7.
31. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. Ann N Y Acad Sci. 2008; 1129: 287-304.
32. Flint J, Kothare SV, Zihlif M, Suarez E, Adams R, Legido A, et al. Association between inadequate sleep and insulin resistance in obese children. J Pediatr. 2007; 150: 364–369.
33. Li AM, Au CT, Ho C, Fok TF, Wing YK. Blood pressure is elevated in children with primary snoring. J Pediatr. 2009; 155: 362-368.
34. Cohen-Lévy J, Potenza J, Couloigner V. [Pediatric obstructive sleep apnea syndrome: Treatment strategy]. Arch Pediatr. 2017; 24: S39-47.
35. Villa Asensi J, De Miguel Díez J, Romero Andújar F, Campelo Moreno O, Sequeiros González A, R Muñoz Codoceo. [Usefulness of the Brouillette index in the diagnosis of obstructive sleep apnea syndrome in children]. An Esp Pediatr. 2000; 53: 547-52.

36. Villa MP, Paolino MC, Castaldo R, Vanacore N, Rizzoli A, Miano S, et al. Sleep clinical record: an aid to rapid and accurate diagnosis of paediatric sleep disordered breathing. Eur Respir J. 2013; 41: 1355-61.

37. Hui-Leng Tan, David Gozal, Helena Molero Ramirez, Hari P R Bandla, Leila Kheirandish-Gozal. Overnight polysomnography versus respiratory polygraphy in the diagnosis of pediatric obstructive sleep apnea. Sleep. 2014; 37: 255–260.

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