Diagnosis and management of OSAS: discussion of the ERS 2016 guidelines

Stijn Verhulst, MD, PhD

Pediatric Pulmonology and Pediatric Sleep Medicine

Antwerp University Hospital - Belgium

Universiteit Antwerpen

Introduction

- Spectrum of abnormal respiratory patterns during sleep characterized by snoring and increased respiratory effort

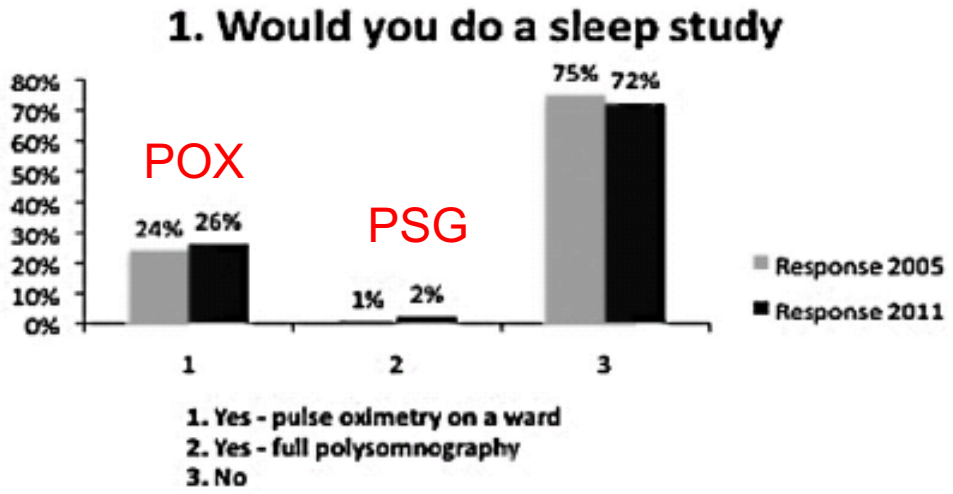
  - Primary snoring
  - Upper airway resistance syndrome
  - Obstructive hypoventilation
  - Obstructive sleep apnea syndrome (OSAS)

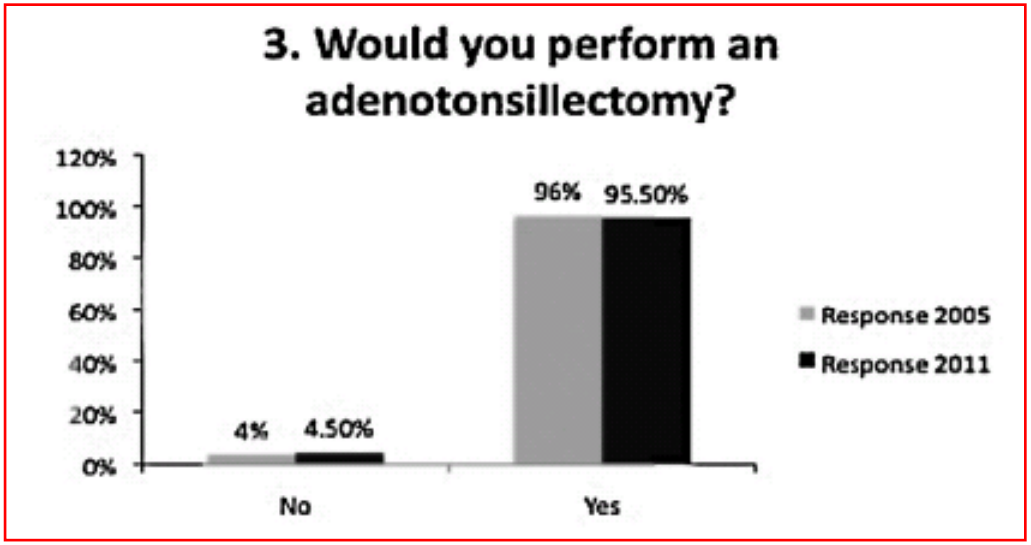
National UK survey on the assessment and surgical management of suspected paediatric obstructive sleep apnoea syndrome

Michael B. Pringle [a,*], Basavaiah G. Natesh [a], Emma. M. Buchanan [b]

International Journal of Pediatric Otorhinolaryngology 77 (2013) 1689–1696

- 5 y.o. child, with loud snoring, mouth breathing, hyponasal speech, recurrent tonsillitis (last 2 years)
- Normal height, weight, facial features
- Decreased nasal airflow, tonsillar hypertrophy





American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

CLINICAL PRACTICE GUIDELINE

Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome

Pediatrics 2012;130;576

"…focuses on uncomplicated childhood OSAS-that is, the OSAS associated with adenotonsillar hypertrophy and/or obesity in an otherwise healthy child…"

Working Party on Sleep Physiology and Respiratory Control Disorders in Childhood

Standards for Services for Children with Disorders of Sleep Physiology

REPORT

September 2009

Royal College of Paediatrics and Child Health

Obstructive sleep disordered breathing in 2- to 18-year-old children: diagnosis and management

Athanasios G. Kaditis[1], Maria Luz Alonso Alvarez[2], An Boudewyns[3], Emmanouel I. Alexopoulos[4], Refika Ersu[5], Koen Joosten[6], Helena Larramona[7], Silvia Miano[8], Indra Narang[9], Ha Trang[10], Marina Tsaoussoglou[1], Nele Vandenbussche[11], Maria Pia Villa[12], Dick Van Waardenburg[13], Silke Weber[14] and Stijn Verhulst[15]

Unique Characteristics of the ERS Statement on the Diagnosis and Management of Obstructive SDB

- refers to the entire severity spectrum of obstructive SDB (i.e. primary snoring to OSAS)
- discusses conditions other than adenotonsillar hypertrophy and obesity predisposing to SDB (e.g. craniofacial abnormalities and neuromuscular disorders)
- takes into account the available diagnostic facilities and accepted treatment policies in different European countries
- describes alternative diagnostic modalities for settings where polysomnography is not available
- considers presence of morbidity as a treatment indication
- suggests a step-by-step diagnostic and treatment approach

Critical Review of Published Evidence

- January 1970-December 2014
- Key words: “adenoidectomy”; “adenoidal hypertrophy”; “adenotonsillar hypertrophy”; “polysomnography”; “sleep apnoea”; “sleep-disordered breathing”; “sleep-related breathing disorders”; “snoring”; “tonsillar hypertrophy”; “tonsillectomy”; “continuous positive airway pressure”; “non-invasive positive pressure ventilation”.
- Methodological quality of the articles was graded as class I-IV according to the American Academy of Neurology Clinical Practice Guideline Process Manual
- The initial search provided 15,149 titles
- 362 references were used to prepare the ERS document

Step 1: Recognize the child at risk for obstructive SDB	**Step 2:** Identify SDB-related morbidity or conditions co-existing with SDB (probably common pathogenesis)	
Step 3: Recognize factors predicting persistence of SDB	**Step 4:** Assess severity of SDB objectively	**Step 5:** Determine indications for treatment
Step 6: Stepwise treatment approach for SDB	**Step 7:** Follow-up, diagnosis and management of persistent SDB	

Step 1: Child at risk for obstructive SDB (if one or more)

Assess by:
history +
physical exam +
objective methods

Symptoms of upper airway obstruction

- Snoring
- Reported apneas during sleep
- Difficulty breathing during sleep
- Restless sleep

Findings on physical exam

- Tonsillar hypertrophy
- Obesity
- Mandibular/midface hypoplasia
- Neuromuscular disorders
- Complex abnormalities (achondroplasia, Down syndrome, Prader-Willi syndrome)

Objective findings related to SDB

- Lateral neck X-ray
- Flexible nasopharyngoscopy
- Cephalometry
- Upper airway MRI or CT

History increasing the risk for SDB

- Premature birth
- Family history of SDB

Prader Willi Syndrome and Obstructive Sleep Apnea: Co-occurrence in the Pediatric Population

Karim Sedky, M.D., M.Sc.[1]; David S. Bennett, Ph.D.[2]; Andres Pumariega, M.D.[1]

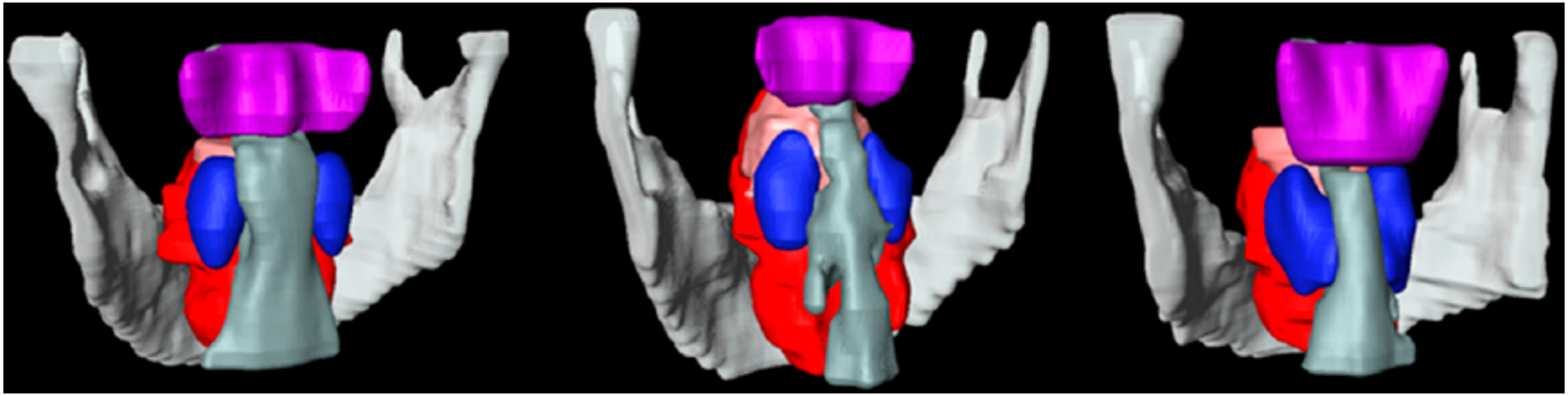
J Clin Sleep Med 2014;10(4):403-409.

- Overall OSAS prevalence: 80% (most patients without symptoms)
- Mild OSAS (apnea-hypopnea index-AHI >1 to <5 episodes/h): 53.1%
- Moderate OSAS (AHI 5-10 episodes/h): 22.3%
- Severe OSAS (>10 episodes/h): 24.6%

Understanding the Anatomic Basis for Obstructive Sleep Apnea Syndrome in Adolescents

Richard J. Schwab[1], Christopher Kim[1], Sheila Bagchi[1], Brendan T. Keenan[1], François-Louis Comyn[1], Stephen Wang[1], Ignacio E. Tapia[1,2], Shirley Huang[3], Joel Traylor[1,2], Drew A. Torigian[4], Ruth M. Bradford[1,2], and Carole L. Marcus[1,2]

Am J Respir Crit Care Med Vol 191, Iss 11, pp 1295–1309, Jun 1, 2015



Step 2: Recognition of morbidity and conditions co-existing with SDB	Morbidity ▪ Cardiovascular system: elevated BP; pulmonary hypertension ▪ Central nervous system: excessive diurnal sleepiness; inattention/hyperactivity; cognitive deficits; academic difficulties; behavioral problems ▪ Enuresis ▪ Inadequate somatic growth rate ▪ Decreased quality of life Conditions co-existing with SDB ▪ Recurrent otitis media, tympanostomy tube placement ▪ Recurrent wheezing or asthma ▪ Metabolic syndrome ▪ Oral-motor dysfunction

RESEARCH ARTICLE

AMERICAN JOURNAL OF medical genetics

155:1546–1551

Sleep Abnormalities in Untreated Patients With Mucopolysaccharidosis Type VI

Ângela John,[1]* Simone Fagondes,[1] Ida Schwartz,[2,3] Ana Cecília Azevedo,[2] Patrícia Barrios,[4] Paulo Dalcin,[1,5] Sérgio Menna-Barreto,[1,5] and Roberto Giugliani[2,3]

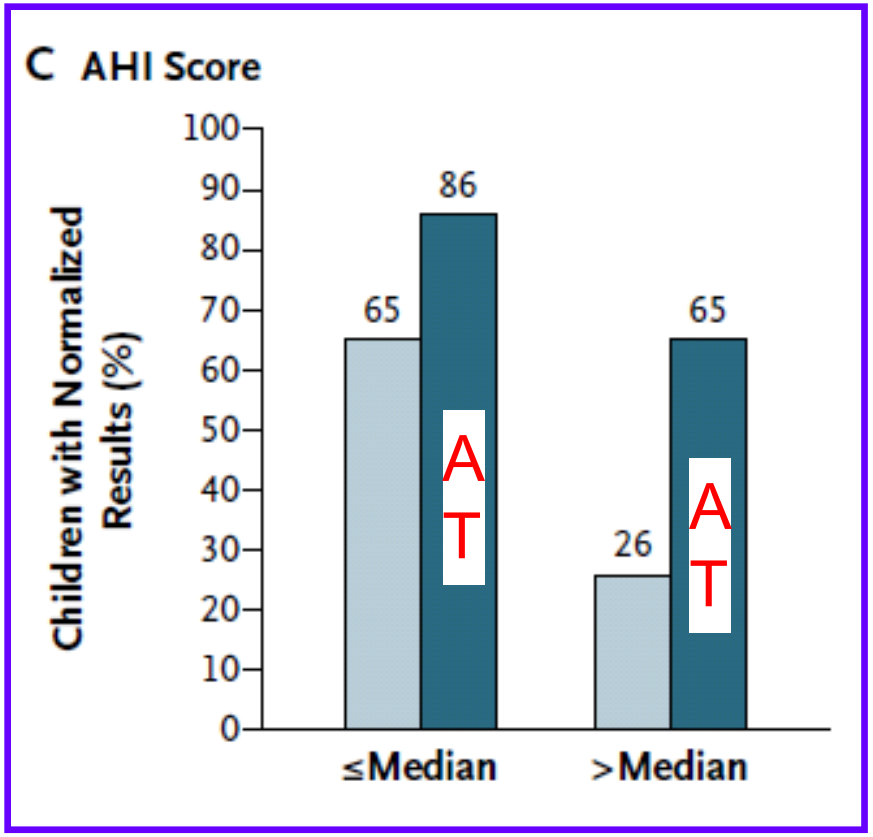
- 28 children (4 - 15.5 y)

- Overall OSAS prevalence: 85.1% (most patients with symptoms)
- Severe OSAS (>10 episodes/h): 51.9%

- Pulmonary hypertension (ECHO): 50%
- Children with pulmonary hypertension had significantly lower SpO_2 mean and nadir values

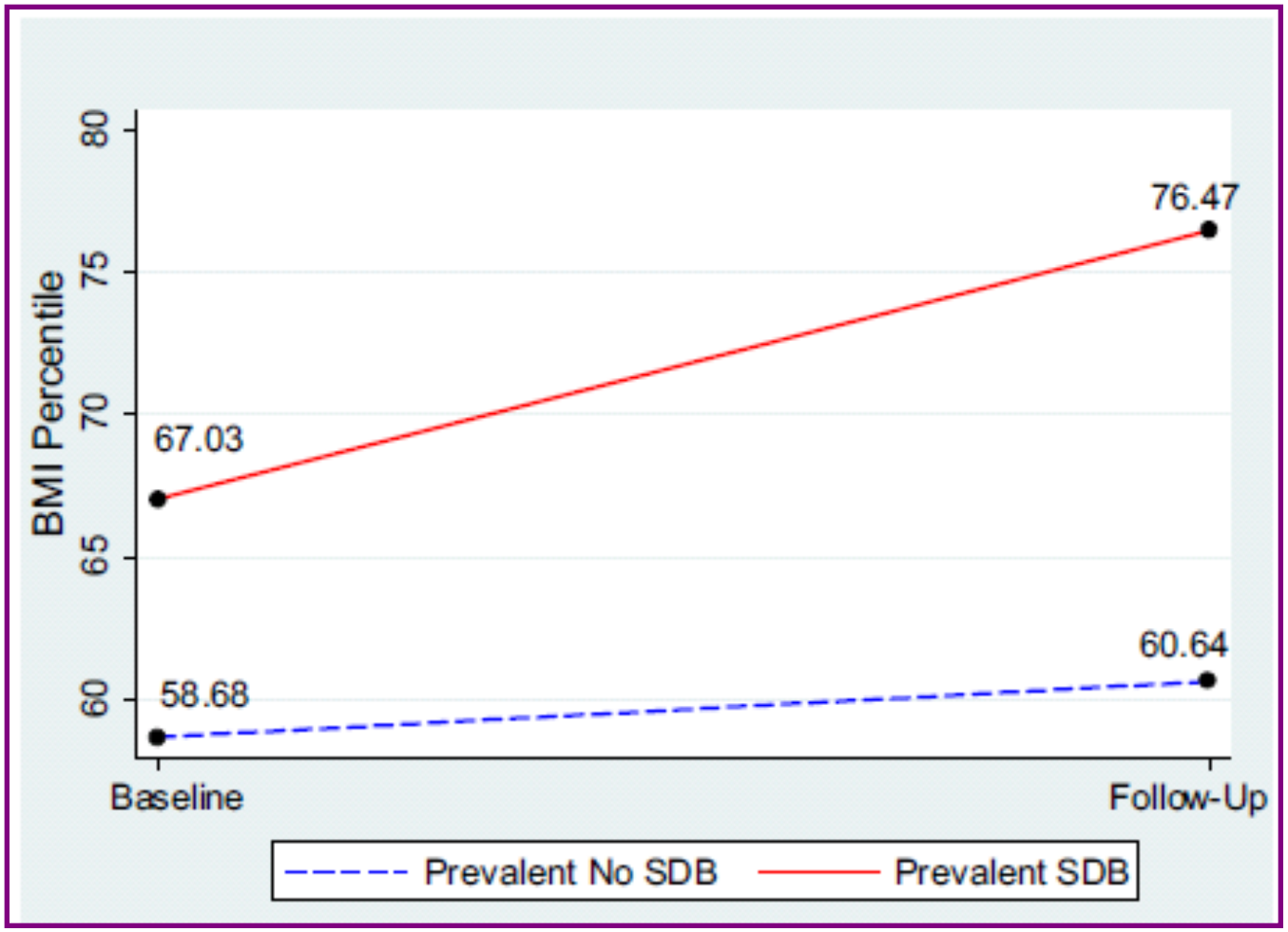
Step 3: Recognize factors predicting persistence of SDB if left untreated	▪ Obstructive AHI >5 episodes/h ▪ Obesity and increasing body mass index percentile ▪ Persistent tonsillar hypertrophy and narrow mandible ▪ Male gender ▪ Black race

Marcus et al. A Randomized Trial of Adenotonsillectomy for Childhood Sleep Apnea. NEJM 2013;157:57-61



Median 4.7 episodes/h

Goodwin et al. Incidence and Remission of SDB and Related Symptoms in 6- to 17-y.o children.
J Pediatr 2010;157:57-61

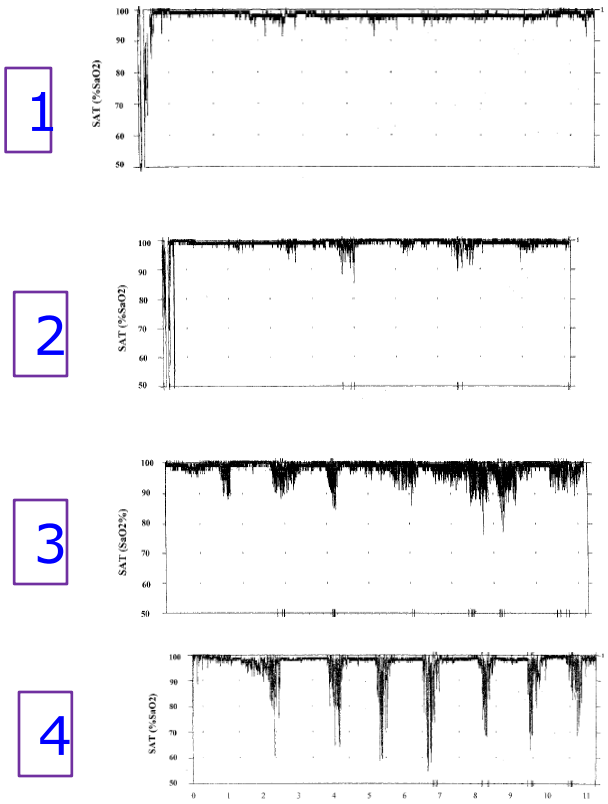


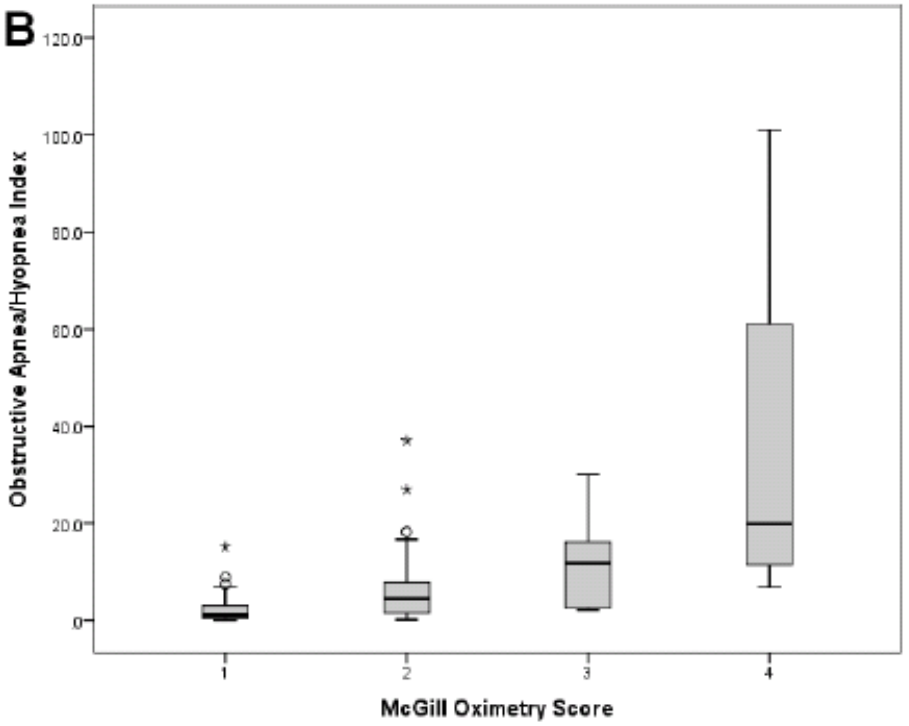
Step 4: Assess severity of SDB objectively (if equipment available)	Indications for nocturnal polysomnography or polygraphy in the child at risk of SDB ▪ prior to AT if: obesity; craniofacial deformities; neuromuscular disorders; complex abnormalities (e.g. Down syndrome, Prader-Willi syndrome); unclear need for treatment ▪ post-AT if: persistent symptoms of OSAS; moderate-to-severe OSAS pre-op; any of the above conditions ▪ prior to and after: rapid maxillary expansion; orthodontic appliances; CPAP; NPPV If PSG not available (especially for healthy children with adenotonsillar hypertrophy): ▪ Ambulatory PSG or polygraphy ▪ Nocturnal oximetry ▪ OSA-18, PSQ, SCR

Overnight Pulse Oximetry for Evaluation of Sleep Apnea among Children with Trisomy 21

Andrea M. Coverstone, M.D.[1]; Merielle Bird, M.S.N., R.N., F.N.P.[2]; Melissa Sicard, M.S.N., R.N., P.N.P.[1]; Yu Tao, M.B., M.S.[3]; Dorothy K. Grange, M.D.[1]; Claudia Cleveland, RPSGT[2]; David Molter, M.D.[4]; James S. Kemp, M.D.[1]

J Clin Sleep Med 2014; 10:1309-15





114 children (1.8 m.o.-21.4 y.o.) with Down syndrome underwent PSG;
50% with OSAS (AHI ≥2.5 episodes/h)

McGill score >2
Positive predictive value 94%
Specificity 98%

Title: The role of nocturnal pulse oximetry in the screening for obstructive sleep apnea in obese children and adolescents.

Author: Van Eyck Annelies, Lambrechts Chinouk, Vanheeswijck Liesbeth, Van Hoorenbeeck Kim, Haentjens Dominique, Boudewyns An, De Winter Benedicte Y., Van Gaal Luc, De Backer Wilfried, Verhulst Stijn L.

Results. A total of 130 obese patients (38% boys, mean age 12 years) were included. Forty four patients (34%) were diagnosed with OSA according to polysomnography. Oximetry, classified 16 patients as positive, 43 as negative and 71 as inconclusive. Further analysis of the positive and negative oximetry results showed a sensitivity and specificity of 58% and 88% respectively, and a negative predictive value and positive predictive value of 81% and 69%. A second analysis, using the oxygen desaturation index showed inferior results compared to scoring by Brouillette (sensitivity 57%, specificity 73%).

Marcus et al. A Randomized Trial of Adenotonsillectomy for Childhood Sleep Apnea. NEJM 2013;157:57-61

OSAS-Definition 1: obstructive AHI ≥2 episodes/h or an obstructive apnoea index ≥1 episode/h in the context of SDB symptoms

OSAS-Definition 2:
SDB symptoms and an AHI ≥1 episode/h

Sedky et al. Attention deficit hyperactivity disorder and sleep disordered breathing in pediatric populations: A meta-analysis. Sleep Med Rev 2014;18:349-56

Scholle et al. The Normative values of polysomnographic parameters in childhood and adolescence: cardiorespiratory parameters. Sleep Med 2011;12:988-96

In children without SDB symptoms or morbidity or abnormalities predisposing to SDB, the 90th percentile for the AHI (AASM 2007 scoring rules) is:

- 3.2 episodes/h for the 2nd year of life
- up to 2.5 episodes/h for ages >2 and ≤6 years
- up to 2.1 episodes/h for ages >6 and <18 years

Step 1: Recognize the child at risk for obstructive SDB	**Step 2:** Identify SDB-related morbidity or conditions co-existing with SDB (probably common pathogenesis)	
Step 3: Recognize factors predicting persistence of SDB	**Step 4:** Assess severity of SDB objectively	**Step 5:** Determine indications for treatment
Step 6: Stepwise treatment approach for SDB	**Step 7:** Follow-up, diagnosis and management of persistent SDB	

INDICATIONS FOR TREATMENT

Indications for treatment

- AHI > 5:
  - Risk of morbidity
  - Better response to adenotonsillectomy (versus AHI < 5 in the CHAT trial)

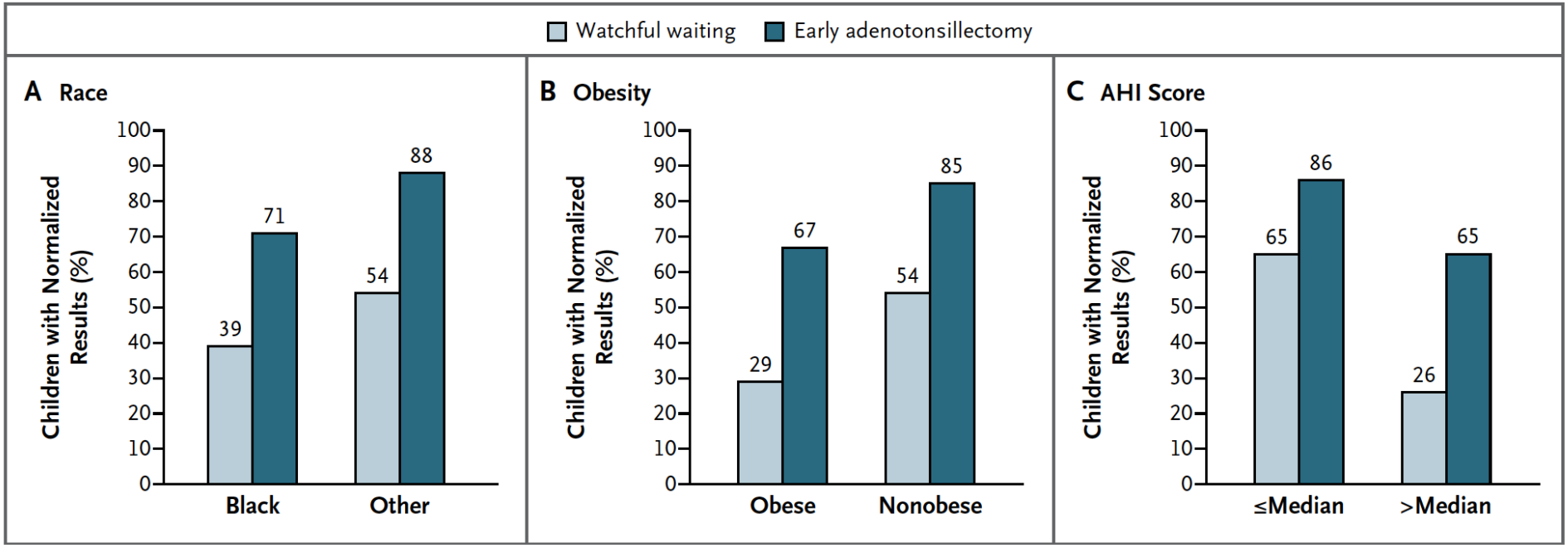
Indications for treatment

- Mild OSAS: Positive effects of treatment especially in the presence of

  - Morbidity of the cardiovascular system
  - Nocturnal enuresis
  - Somatic growth delay
  - Decreased quality of life
  - Risk factors for persistence of OSAS

Indications for treatment

- Health benefits of reducing AHI in children with mild OSAS are unclear:
  - Low to moderate quality studies
  - Subclinical parameters



Nearly half the children in the watchful-waiting group showed normalization of the AHI score. This improvement may have been due to growth of the airway or regression of lymphoid tissue, routine medical care, or regression to the mean.

Indications for treatment

- Health benefits of reducing AHI in children with mild OSAS are unclear:

  - Low to moderate quality studies
  - Subclinical parameters
  - Inconsistent effect on neurocognitive and behavioural deficits – no correlation with pre-operative OSA severity

Table 2. Outcome Measures.*

Outcome	Normative Mean	Watchful Waiting		Early Adenotonsillectomy		Effect Size†	P Value
		Baseline	Change from Baseline to 7 Mo	Baseline	Change from Baseline to 7 Mo		
Primary outcome							
NEPSY attention and executive-function score‡	100±15	101.1±14.6	5.1±13.4	101.5±15.9	7.1±13.9	0.15	0.16
Secondary outcomes							
Conners' Rating Scale score§	50±10						
Caregiver rating		52.6±11.7	−0.2±9.4	52.5±11.6	−2.9±9.9	0.28	0.01
Teacher rating		55.1±12.8	−1.5±10.7	56.4±14.4	−4.9±12.9	0.29	0.04
BRIEF score¶	50±10						
Caregiver rating		50.1±11.5	0.4±8.8	50.1±11.2	−3.3±8.5	0.28	<0.001
Teacher rating		56.4±11.7	−1.0±11.2	57.2±14.1	−3.1±12.6	0.18	0.22
PSQ-SRBD score‖	0.2±0.1	0.5±0.2	−0.0±0.2	0.5±0.2	−0.3±0.2	1.50	<0.001
PedsQL score**	78±16	76.5±15.7	0.9±13.3	77.3±15.3	5.9±13.6	0.37	<0.001
Apnea–hypopnea index — no. of events/hr††	NA						
Median		4.5	−1.6	4.8	−3.5	0.57	<0.001‡‡
Interquartile range		2.5 to 8.9	−3.7 to 0.5	2.7 to 8.8	−7.1 to −1.8		

Indications for treatment

- Mild sleep apnea tend to resolve in some children

  - Follow-up time
  - Seasonality effect?
  - Risk factors for disease progression
  - Ethnicity effect
  - Anti-inflammatory medication?
  - Association with morbidity?

Montelukast for Children With Obstructive Sleep Apnea: A Double-blind, Placebo-Controlled Study

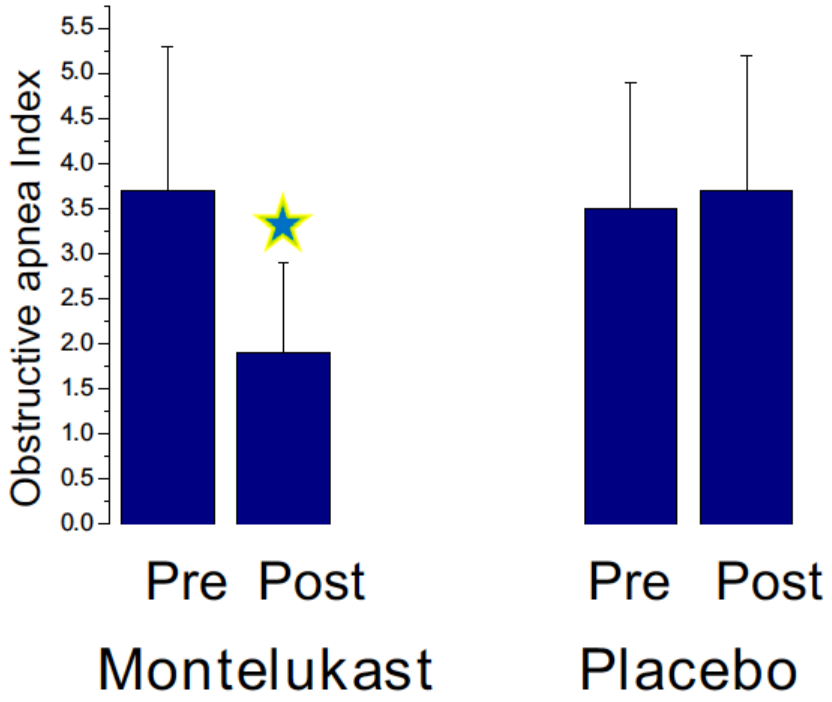


FIGURE 1

Montelukast treatment resulted in a significant improvement in the OAI. The pretreatment average of 3.7 ± 1.6 before (pre) dropped to 1.9 ± 1.0 after (post) treatment; $P < .05$. In contrast, 12 weeks of placebo treatment did not significantly change the OAI; means: 3.5 ± 1.6 (pre) vs 3.7 ± 1.0 (post) treatment; $P = .75$. Star indicates a significant difference between pre and post values.

Natural history and predictors for progression of mild childhood obstructive sleep apnoea

A M Li,[1] C T Au,[1] S K Ng,[2] V J Abdullah,[2] C Ho,[3] T F Fok,[1] P C Ng,[1] Y K Wing[3]

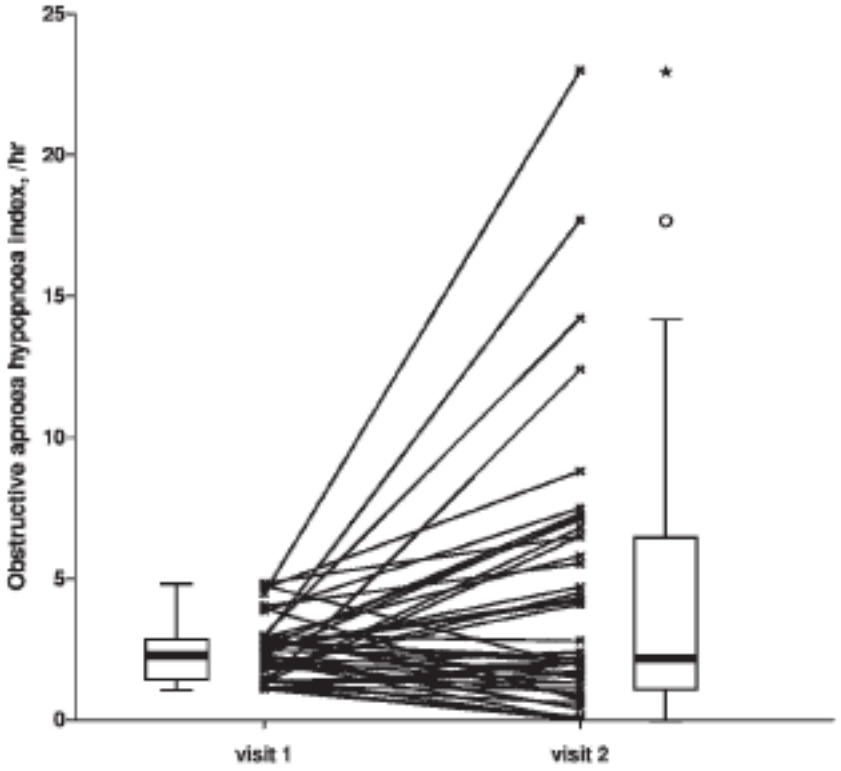


Figure 1 Change in obstructive apnoea-hypopnoea index of each subject over 2-year follow-up period.

Table 3 Linear regression analyses assessing the association between the change in OAHI and different independent variables (n = 45)

	Univariate		Multivariate			
			Model 1*		Model 2†	
	β (SE)	p Value	β (SE)	p Value	β (SE)	p Value
Male gender	2.10 (1.44)	0.153	4.69 (1.29)	<0.001	2.60 (1.18)	0.033
Baseline characteristics						
Age	−0.64 (0.39)	0.106	−0.92 (0.34)	0.009	−0.01 (0.36)	0.987
BMI	0.26 (0.18)	0.174				
BMI z-score	1.33 (0.66)	0.048	0.97 (0.55)	0.084		
Waist	0.03 (0.06)	0.643				
Waist z-score	0.81 (0.63)	0.206				
OAHI	0.76 (0.65)	0.248				
Large tonsils	3.75 (1.33)	0.007	4.36 (1.24)	0.001		
Habitual snoring	2.85 (1.38)	0.045	0.92 (1.21)	0.453		
Changes over 2 years						
BMI	0.51 (0.45)	0.261				
BMI z-score	0.44 (1.50)	0.769				
Waist	0.41 (0.09)	<0.001			0.30 (0.09)	0.002
Waist z-score	2.48 (1.09)	0.028				
Persistent large tonsils	6.26 (1.51)	<0.001			5.69 (1.36)	<0.001

*Adjusted for baseline characteristics including age, gender, body mass index z-score, presence of large tonsils and habitual snoring.
†Adjusted for age at baseline, gender, change in waist circumference and presence of persistent large tonsils.
BMI, body mass index; OAHI, obstructive apnoea hypopnoea index; waist, waist circumference.

Prognosis for Spontaneous Resolution of Obstructive Sleep Apnea in Children

Ronald D. Chervin, M.D, M.S.,[1] Susan S. Ellenberg, Ph.D.,[2] Xiaoling Hou, M.S.,[2] Carole L. Marcus, M.B B.Ch.,[3] Susan L. Garetz, M.D.,[4] Eliot S. Katz, M.D.,[5] Elise K. Hodges, Ph.D.,[6] Ron B. Mitchell, M.D.,[7] Dwight T. Jones, M.D.,[8] Raanan Arens, M.D.,[9] Raouf Amin, M.D.,[10] Susan Redline, M.D.,[11] Carol L. Rosen, M.D.[12 *]

Results: After 194 children aged 5-9 years underwent 7 months of watchful waiting, 82 (42%) no longer met polysomnographic criteria for OSAS. Baseline predictors of resolution included lower AHI, better oxygen saturation, smaller waist circumference or percentile, higher-positioned soft palate, smaller neck circumference, and non-African-American race (each $p<.05$). Among these, the independent predictors were lower AHI and waist circumference percentile <90%. Among 167 children with baseline PSQ scores ≥0.33, only 25 (15%) experienced symptomatic resolution. Baseline predictors were low PSQ and PSQ snoring subscale scores; absence of habitual snoring, loud snoring, observed apneas, or a household smoker; higher quality of life; fewer Attention-Deficit/Hyperactivity Disorder symptoms; and female gender. Only lower PSQ and snoring scores were independent predictors.

Conclusions: Many candidates for AT no longer have OSAS on polysomnography after 7 months of watchful waiting, whereas meaningful improvement in symptoms is not common. In practice, a baseline low AHI and normal waist circumference, or low PSQ and snoring score, may help identify an opportunity to avoid adenotonsillectomy.

Indications for treatment

- Need for clear and practical tools that can identify children with mild OSA at risk for morbidity

  - Questionnaires – definition of cut-off values
  - Biomarkers

Urinary Neurotransmitters Are Selectively Altered in Children With Obstructive Sleep Apnea and Predict Cognitive Morbidity

Leila Kheirandish-Gozal, MD; Corena J. T. McManus, MS; Gottfried H. Kellermann, PhD; Arash Samiei, MD; and David Gozal, MD, FCCP

Table 3—*Demographic, Polysomnographic, and Overnight Changes in Urinary Neurotransmitter Levels in 20 Children With OSA and Preserved GCA Scores and 16 Children With OSA and Low GCA Scores*

Characteristics	OSAab (n = 16)	OSAn (n = 20)	*P* Value
Age, y	6.1 ± 1.8	6.2 ± 1.7	NS
Sex, male, %	50%	50%	NS
White ethnicity, %	68.7	65	NS
BMI Z score	1.30 ± 0.91	1.25 ± 0.87	NS
Apnea-hypopnea index (events/h)	12.24 ± 3.2	8.93 ± 4.87	NS
SaO_2 nadir, %	83.5 ± 5.1	85.6 ± 4.7	NS
TAI/hrTST	18.2 ± 8.2	17.1 ± 7.9	NS
GCA score	87.3 ± 13.9	101.2 ± 14.5	<.01
Overnight percent change in			
Norepinephrine	152.3 ± 69.6	90.3 ± 15.7	0.06
Epinephrine	120.3 ± 33.8	143.7 ± 30.8	NS
Taurine	−170.3 ± 29.1	−45.8 ± 5.7	<.0001
GABA	60.2 ± 5.7	38.6 ± 8.0	<.0001
PEA	−66.9 ± 5.3	−32.4 ± 5.1	<.0001

Data for changes in urine neurotransmitter level changes are shown as mean ± SEM; all other values are shown as mean ± SD. See Table 1 legend for expansion of abbreviations.

Indications for treatment

- Primary snoring:
  - Association with elevated nocturnal blood diastolic blood pressure, cognitive deficits and behavioural abnormalities
  - No studies assessing the effects of treatment

Indications for treatment

- Prioritize treatment:
  - Major craniofacial abnormalities
  - Neuromuscular disorders
  - Achondroplasia
  - Chiari malformation
  - Down syndrome
  - MPS
  - Prader-Willi syndrome

  - Higher risk of pulmonary HT, positive effects on symptoms and QoL, higher rate of residual OSA

TREATMENT

Treatment

- Different treatment modalities
  - Non-invasive to very invasive
  - Combination of treatments
  - Stepwise treatment plan

Adeno-tonsillectomy and rapid maxillary distraction in pre-pubertal children, a pilot study

Christian Guilleminault · Pierre-Jean Monteyrol · Nelly T. Huynh · Paola Pirelli · Stacey Quo · Kasey Li

Table 1 Studied groups and results after each treatment

Variables	Baseline		*p* value	Treatment 1		*p* value	Treatment 2		*p* value
	Group 1	Group 2		Group 1 surgery	Group 2 orthodontics		Group 1 orthodontics	Group 2 surgery	
Sleep variables									
TST, min	431.3±4.1	423.1±2.2	0.09	429.1±5.6	425.1±5.3	0.51a	445.7±8.7	438.7±4.7	0.72a
						0.99b			0.00b
						0.28c			0.51c
REM, %	18.6±0.4	18.9±0.3	0.60	20.5±0.3	20.1±0.2	0.13a	22.0±0.2	21.9±0.3	0.32a
						0.00b			0.00b
						0.81c			0.45c
Sleep apnea variables									
AHI, events/h	12.5±0.8	11.1±0.7	0.20	4.9±0.6	5.4±0.6	0.15a	0.9±0.3	0.9±0.3	0.16a

Weight loss

- Substantial weight loss achieved by inpatient treatment centres or bariatric surgery is effective in morbidly obese adolescents with OSAS

  - No data on modest weight loss regimes
  - No data in children
  - No data on long-term compliance
  - Incorporate weight management program in every obese child with OSAS

Pharmacological treatment

- Nasal steroids
- Montelukast

  - Treatment for 6-12 weeks
  - Less improvement in older and obese children
  - No data on effects on OSA-related morbidity
  - Post-adenotonsillectomy residual OSA

Antiinflammatory Therapy Outcomes for Mild OSA in Children

Leila Kheirandish-Gozal, MD; Rakesh Bhattacharjee, MD; Hari P. R. Bandla, MD, FCCP; and David Gozal, MD, FCCP

TABLE 2] Changes in Polysomnographic Findings Following 12-Wk Treatment With an Intranasal Corticosteroid and Oral Montelukast in 445 Children

Characteristic	Mild OSA Pretreatment (n = 445)	Mild OSA Posttreament (n = 445)	*P* Value
Age, y	6.2 ± 1.9	6.6 ± 1.9	...
Male sex, %	55.1	...	...
White, %	56.5	...	...
Black, %	26.8	...	...
BMI *z*-score	1.17 ± 0.81	...	...
Obese (BMI *z*-score > 1.65), %	33.8	...	...
Elapsed time between beginning treatment[a] and second NPSG, mean, d	...	114.8 ± 39.2	...
Tonsillar size	2.39 ± 0.77	1.87 ± 0.62	< .01
Adenoid size	2.17 ± 0.77	1.34 ± 0.68	< .001
Mallampati score (n)	1.89 ± 0.62 (412)	1.83 ± 0.64 (412)	...
Total sleep duration, min	472.1 ± 51.2	470.9 ± 49.1	...
Stage 1, %	4.7 ± 3.1	4.2 ± 3.4	...
Stage 2, %	37.8 ± 8.3	29.3 ± 9.7	...
Stage 3, %	40.6 ± 16.2	41.2 ± 15.8	...
REM sleep, %	19.3 ± 6.4	27.5 ± 7.8	< .01
Sleep latency, min	24.7 ± 16.1	27.9 ± 17.2	...
REM latency, min	138.1 ± 54.7	135.3 ± 62.9	...
Total arousal index, events/h TST	15.1 ± 9.3	12.2 ± 8.7	< .01
Respiratory arousal index, events/h TST	2.9 ± 1.7	0.8 ± 1.5	< .001
Obstructive AHI, events/h TST	4.5 ± 2.0	1.4 ± 0.0.9	< .01
Spo_2 nadir, %	87.5 ± 3.1	92.3 ± 2.1	< .001
Patients with normal NPSG, No. (%)	...	276 (62.0)	...

Data given as mean ± SD unless otherwise indicated. NPSG = nocturnal polysomnography. See Table 1 legend for expansion of other abbreviations.
[a]Intranasal corticosteroids plus oral montelukast for 12 wk.

Adenotonsillectomy

- First line treatment in children with OSAS and adenotonsillar hypertrophy
  - Largest effects in children with AHI > 5
  - Success rate in healthy children of about 75%
    - Definition of success
    - Tools to increase success rate

Drug-induced sedation endoscopy in pediatric obstructive sleep apnea syndrome ☆

A. Boudewyns [a,*], S. Verhulst [b], M. Maris [a], V. Saldien [c], P. Van de Heyning [a]

[a] Department of Otorhinolaryngology, Head and Neck Surgery, University Hospital Antwerp, Antwerp University, Belgium
[b] Department of Pediatrics, University Hospital Antwerp, Antwerp University, Belgium
[c] Department of Anesthesiology, University Hospital Antwerp, Antwerp University, Belgium

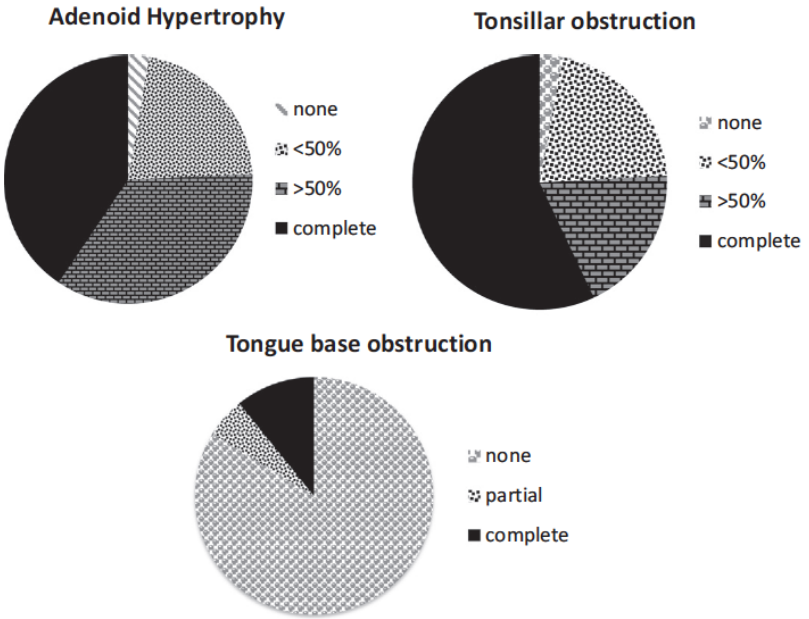


Fig. 2. Drug-induced sedation endoscopy findings for upper airway obstruction at the level of the adenoids, tonsils, and tongue base.

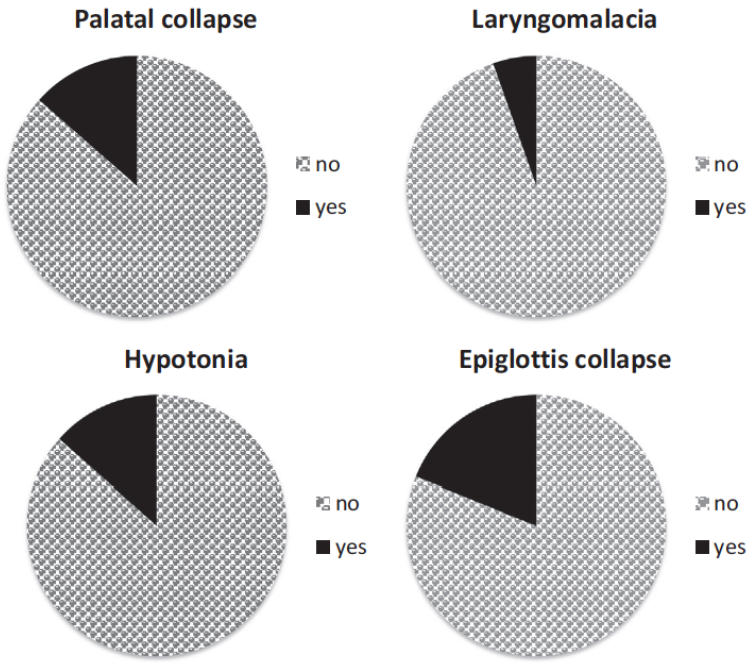


Fig. 3. Drug-induced sedation endoscopy findings for dynamic upper airway collapse at the level of the palate, epiglottis, presence of laryngomalacia and hypotonia.

- Increased success percentage of 91%

Adenotonsillectomy

- Risk factors for persistence of OSA:
  - Severe OSA
  - Obesity
  - Asthma
  - Inferior turbinate hypertrophy
  - Nasal septum deviation
  - Mallampati score 3 or 4
  - Retroposition of the mandible
  - Midface hypoplasia
  - Down syndrome
  - Other craniofacial syndromes

Adenotonsillectomy

- Positive effects on:
  - QoL
  - OSA-related symptoms
  - Growth delay
  - Enuresis
  - Pulmonary hypertension
  - Behavioural deficits

Adenotonsillectomy

- Side effects:
  - Dehydration, nausea and vomiting
  - Local bleeding
  - Respiratory complications
    - Severe OSA (high AHI and/or frequent desaturations)
    - < 3 years
    - Obesity or failure to thrive
    - Neuromuscular, craniofacial or genetic disorders

Rapid maxillary expansion

- Children with OSA and maxillary constriction
  - Decrease in oAHI
  - Increase in Qol

Non-invasive ventilation

- Residual OSAS
- Obesity
- Craniofacial abnormalities
- Genetic syndromes
- Neuromuscular disorders
- Improvements in symptoms, behaviour and QoL.
- Complications

Other treatment options

- Craniofacial surgery
- Tracheostomy

STEP 5. Indications for treatment of SDB:

5.1 a. AHI >5 episodes/h irrespective of the presence of morbidity

b. Treatment may be beneficial if AHI 1-5 episodes/h especially in the presence: of i) morbidity from the cardiovascular system (see 2.1); ii) morbidity from the central nervous system (see 2.1); iii) enuresis; iv) somatic growth delay or growth failure; v) decreased quality of life; vi) risk factors for OSAS persistence (see 3.1)

c. If at risk for OSAS and PSG or polygraphy not available, treatment is considered when positive oximetry or SDB questionnaires (see 4.4) or morbidity present

5.2 No evidence for treating primary snoring (evaluation yearly)

5.3 OSAS treatment is a priority if:

a. Craniofacial abnormalities

b. Neuromuscular disorders

c. Achondroplasia

d. Chiari malformation

e. Down syndrome

f. Mucopolysaccharidoses

g. Prader-Willi syndrome

STEP 6. Stepwise treatment approach to SDB:

6.1 A stepwise treatment approach (from 6.2 to 6.9) is usually implemented until complete resolution of SDB

6.2 Weight loss if the child is overweight or obese

6.3 Nasal corticosteroids and/or montelukast po

6.4 Adenotonsillectomy

6.5 Unclear whether adenoidectomy or tonsillectomy alone are adequate

6.6 Rapid maxillary expansion or orthodontic appliances

6.7 Continuous positive airway pressure (CPAP) or noninvasive positive pressure ventilation (for nocturnal hypoventilation)

6.8 Craniofacial surgery

6.9 Tracheostomy

For details on indications, efficacy, adverse effects or complications of different treatment interventions see text

STEP 7. Recognition and management of persistent SDB:

7.1 a. Outcomes monitored after intervention (6 wks-12 months): symptoms, PSG, quality of life, cardiovascular or central nervous system morbidity, enuresis, growth rate

b. If PSG not available: polygraphy, oximetry/ capnography

c. PSG ≥6 weeks after adenotonsillectomy (persistent SDB symptoms or at risk of persistent OSAS preoperatively); after 12 weeks of montelukast/nasal steroid

d. PSG after 12 months of rapid maxillary expansion (earlier if symptoms persist) and after 6 months with an oral appliance

e. PSG for titration of CPAP, NPPV and then annually; PSG as predictor of successful decannulation with tracheostomy

f. Airway re-evaluation by nasopharyngoscopy, drug-induced sleep endoscopy, MRI

CONCLUSIONS

Conclusions

- A lot more work to do!
  - Markers of morbidity
  - Effects of pharmacological treatment on morbidity
  - Definitions of treatment response
  - OSA in infants