Epidemiology and Screening Considerations of Paediatric Obstructive Sleep Apnea

“I do not have any financial interest in any of the products or diagnosis and treatment management suggestions to be discussed in this presentation.

The discussion to be facilitated today is intended to be general in nature and should not be used to substitute a proper detailed clinical assessment of any individual patient. ”

Dr. Carlos Flores-Mir

Paediatric Sleep Disorder Breathing

Why does this matter?

Anti-Snoring Jaw Strap

Anti-Snoring Jaw Strap

- Comfortably wraps behind and around the head
- Aims to reduce snoring and increase REM sleep
- Helps prevent airway restriction
- Helps hold jaw up
- Dimensions: 10"x4"x0.1"
- Weight: 2.4 oz.

For questions pertaining to this deal, click the Ask a Question button below. For post-purchase inquiries, please contact Groupon customer support.

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C$14.99

BUY!

VALUE	DISCOUNT	YOU SAVE
C$36.99	59%	C$22

LIMITED TIME ONLY!
8 days 21:20:51

LIMITED QUANTITY AVAILABLE
Over 520 bought

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In a Nutshell

Soft material gently holds jaw in upward position during sleep, helping snorers get better rest

The Fine Print

- Free returns
- Most orders are delivered within 7 business days from the purchase date. Shipping questions?
- Ships within Canada only
- Price includes all duty and excise taxes. Applicable GST/HST tax added at checkout

2-Pack of Stop Snoring Mouth Guards

Well-Groomed

Stop Snoring Mouth Guards

- Designed to prevent loud wheezing and sawing by positioning the jaw slightly forward
- Works to widen respiratory tract and allow unobstructed airflow
- Helps sleepers breathe easier through entire night
- Made to help relieve both regular snoring and sleep apnea
- Dimensions: 3.35" x 1.1" x 3.35"

C$17.99

BUY!

VALUE	DISCOUNT	YOU SAVE
C$39.99	55%	C$22

LIMITED TIME ONLY!
14 days 17:00:30

LIMITED QUANTITY AVAILABLE
Over 5,000 bought

SHARE THIS DEAL
Like 41

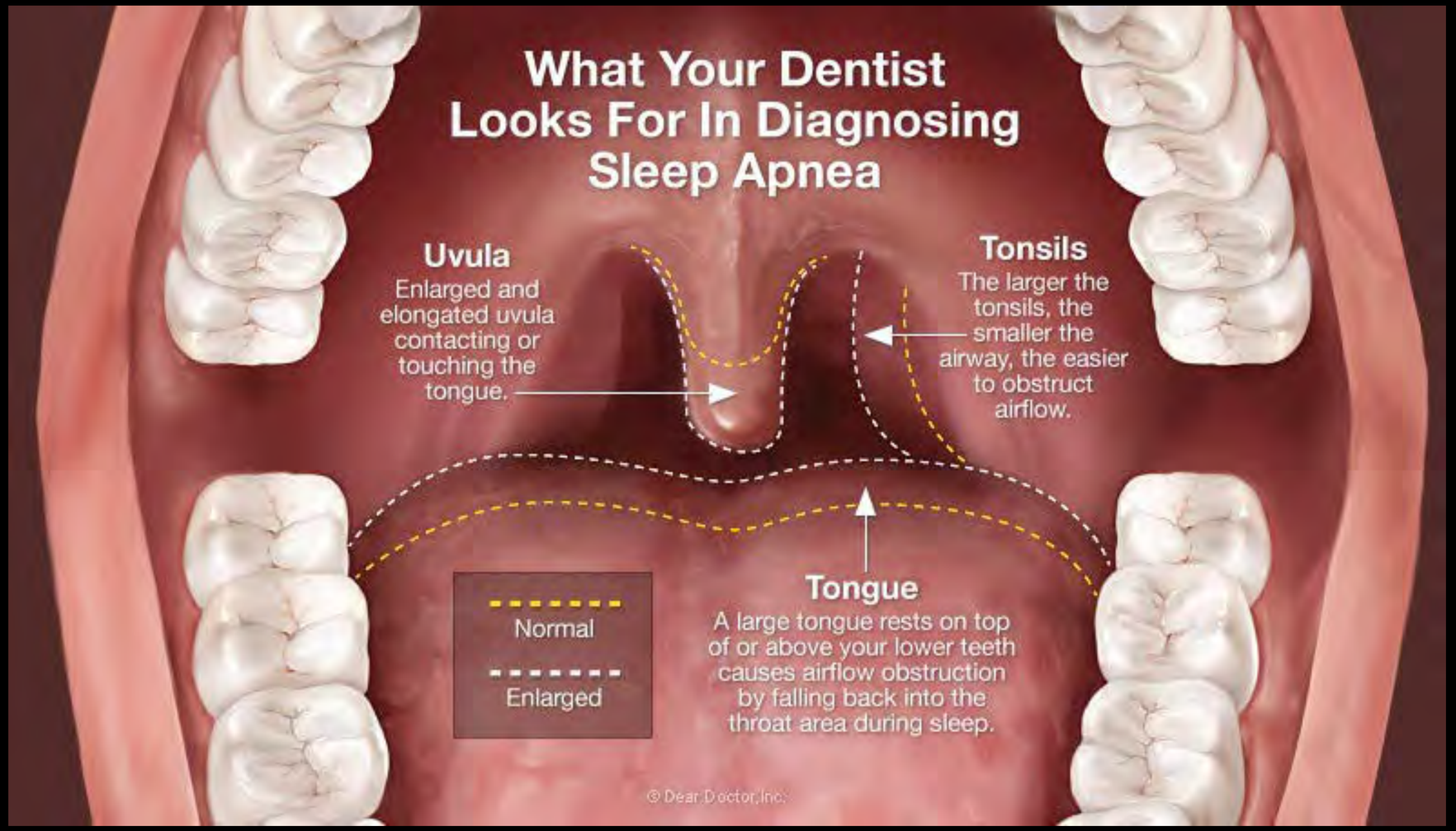
In a Nutshell

Mouth guards designed to relieve snoring and sleep apnea help move the jaw slightly forward to allow unobstructed breathing

The Fine Print

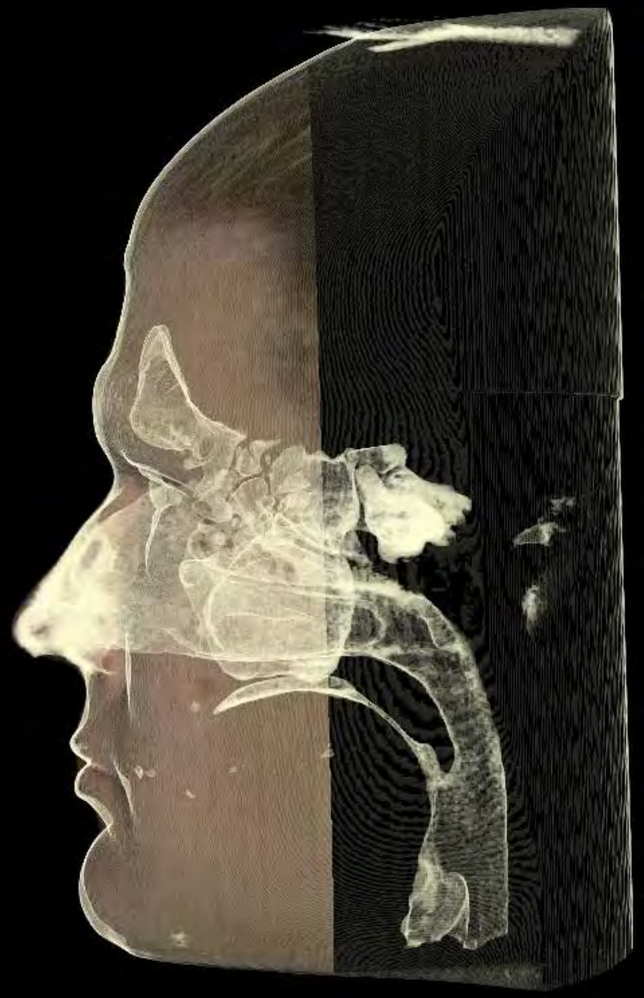
- FINAL SALE; no returns unless defective

PSDB Assessment - Dentists

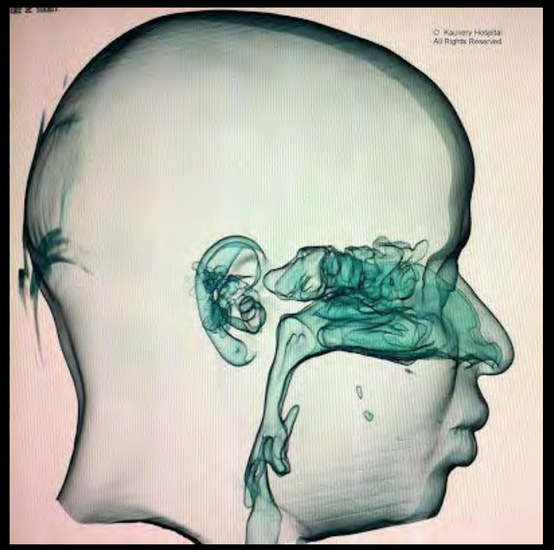


http://martindaledental.com/oral-surgery-procedures/sleep-apnea/

Complexity of the upper airway



https://ct-dent.co.uk/justification-for-x-ray/



http://
kauveryhospital.blogspot.ca/2012/05/3d-imaging-of-upper-respiratory-tract.html

Sleep Disorder Breathing

Group of physiopathologic conditions characterized by abnormal respiratory pattern during sleep.

Snoring-Upper Airway Resistance Syndrome (UARS)-Obstructive Sleep Apneas (OSA)

Chan J et al. Am Fam Physician. 2004; 69: 1147-54.

Sleep Disorder Breathing

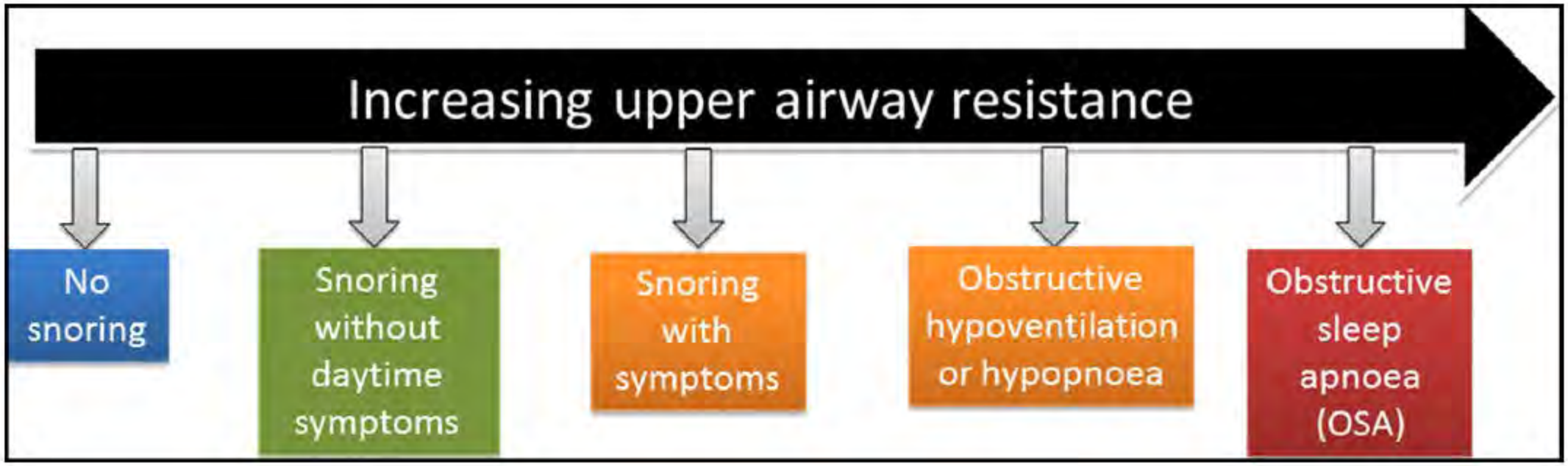


Fig 1. Spectrum of symptoms of pediatric SDB (adapted from Carroll[1]).

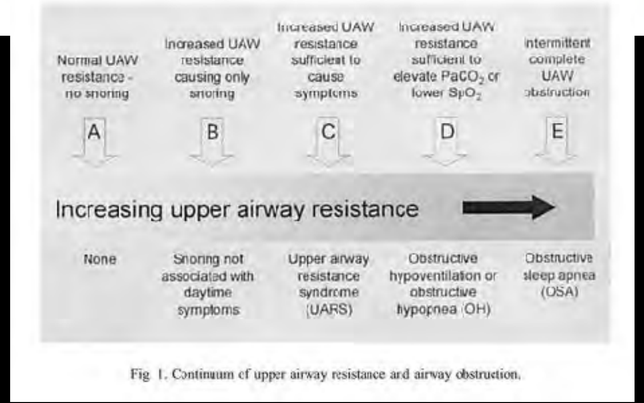
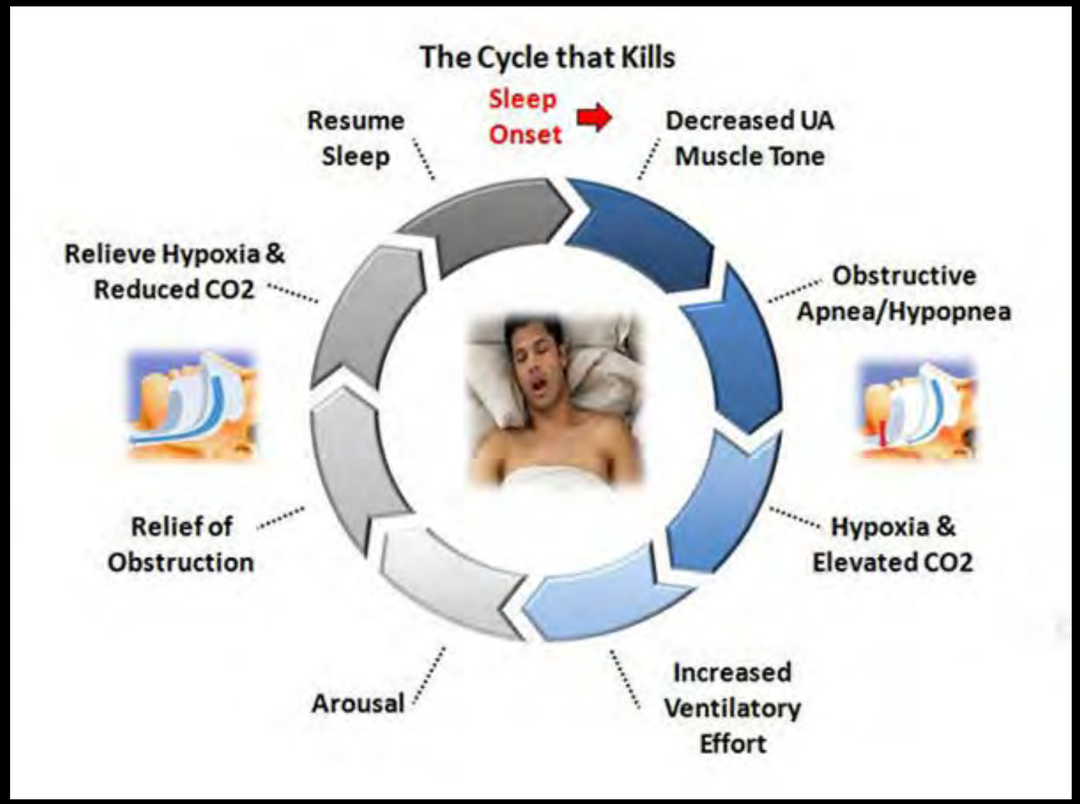


Fig 1. Continuum of upper airway resistance and airway obstruction.

Carroll JL. Clin Chest Med. 2003: 24: 261-82.

POSA Patophysiology

- Breathing interruption because of obstruction
- Hypoxemia
- Oxygen desaturation awakes patient - open airway
- Breathing resumes with noise (snoring)
- No deep sleep cycles



https://proacthealthsolutions.com/providers/obstructive-sleep-apnea-overview/

Related definitions

Apnea: Complete airflow cessation for 10 sec or >.

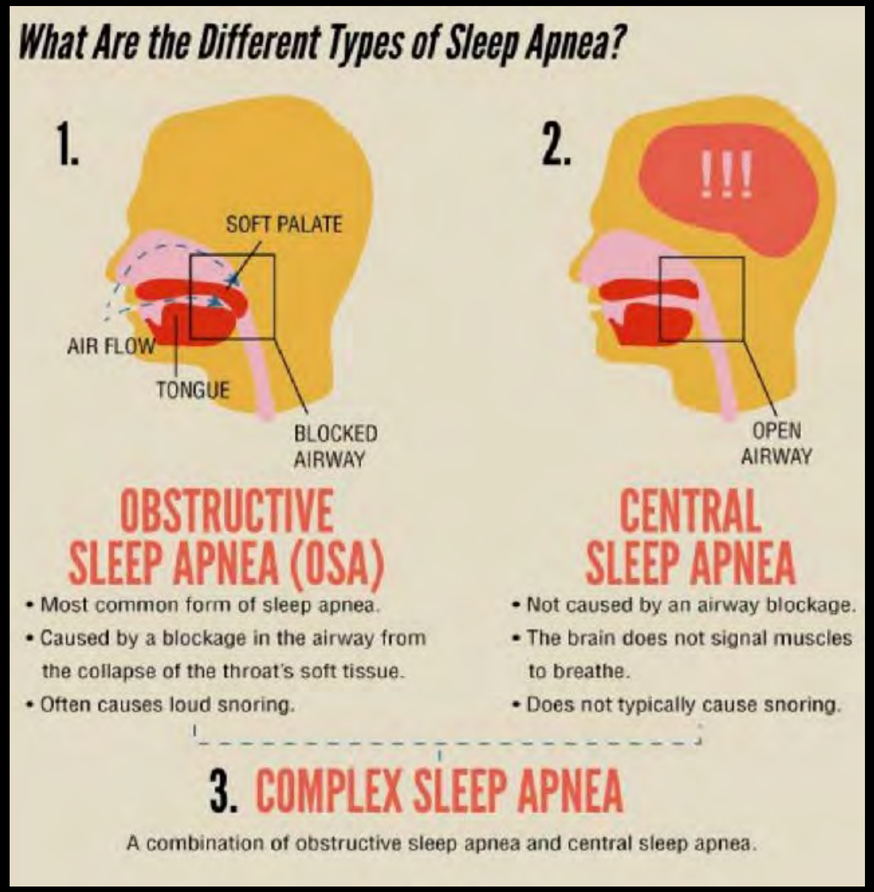
Hypopnea: 50% reduction of airflow for 10 sec or >.

AHI(Apnea/Hypoxia Index): Number of apnea and hypopnea events per hour of sleep.

Arousal: Change in sleep state.

Sleep Disorder Breathing

Sleep Apnoea can be central, obstructive or mixed.



https://www.google.ca/url?sa=i&rct=j&q=&esrc=s&source=images&cd=&ved=0ahUKEwjKoL7FtrLZAhVSxmMKHfNDCyUQjRwIBw&url=https%3A%2F%2Ftwitter.com%2Fnjsleepapnea%2Fstatus%2F738752391856558080&psig=AOvVaw0OfVzNoUhRJPZAJIVIkn7x&ust=1519145067658777

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Central: Cessation of nasal and oral airflow in the absence of any apparent respiratory effort - CNS control.

CSAHS: Central sleep-apnea-hypopnea syndrome.

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Central:

0.4% of the cases.

Brain signals that trigger breathing malfunctioning.

Airway remains open.

Snoring does not necessarily happen.

No inhalation efforts.

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Obstructive: Strong respiratory effort that is ineffective due to lack of airway patency - Upper airway.

OSAHS: Obstructive sleep-apnea-hypopnea syndrome.

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Obstructive:

84% of the cases.

Effort made to breathe.

Airway is temporarily or permanently blocked.

Snoring happens - worst supine - gravity.

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Mixed: Both situations occur simultaneously.

SHVS: Sleep hypoventilation syndrome

Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Sleep Disorder Breathing

Mixed:

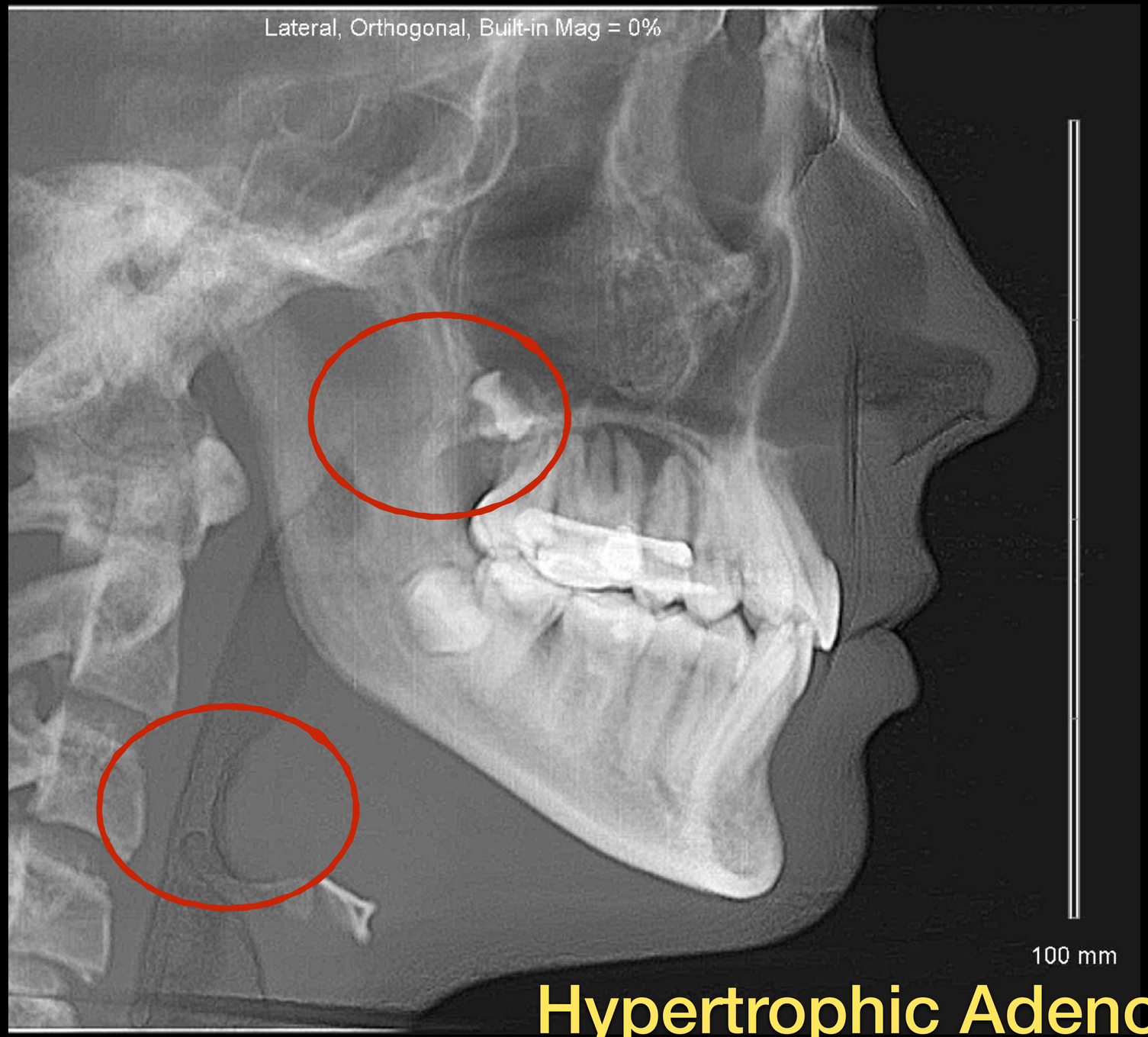
15% of the cases.

Combined signs and symptoms

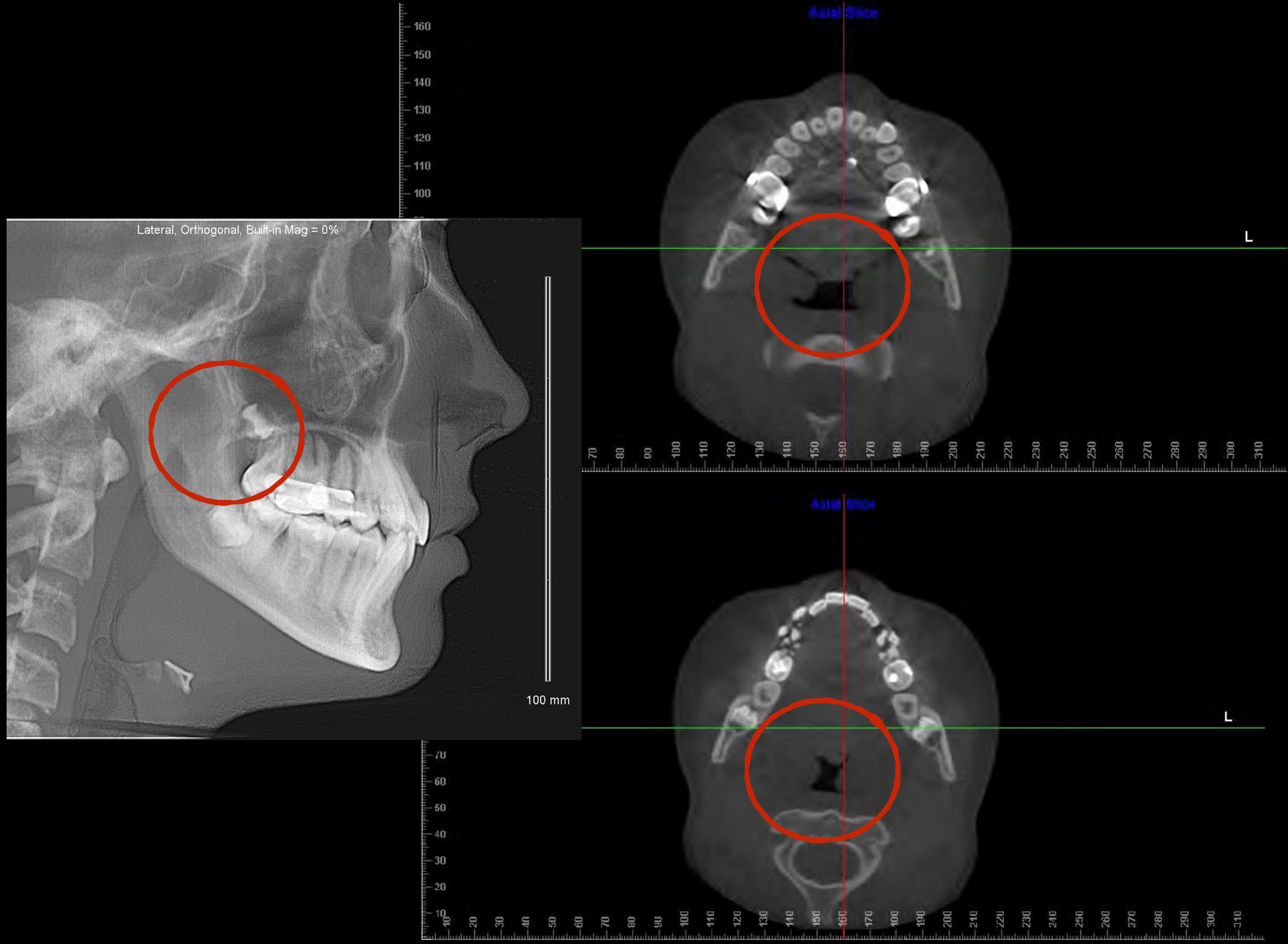
Fleetham J et al. Can Resp J. 2006: 13: Suppl A: 5-47.

Paediatric Sleep Disorder Breathing

Case #1



Hypertrophic Adenoids


Lateral, Orthogonal, Built-in Mag = 0%
100 mm
Axial Slice
L
Axial Slice
L

Epidemiology in adults

- Onset after 40 years, but can occur ay any age.
- More common in male than females.
- Trend to increase with age and BMI.
- Large number of adults go undiagnosed

Young T. Am J Resp Crit Care Med. 2002; 165: 1217-39.

Epidemiology in children

- Up to 15% of children SDB; highest prevalence 3-5 yo.
- 2-6% of children and adolescents POSA. More typical 2-7 yo.
- Equal sex distribution until adolescence. After puberty more in males.

Biggs SN et al. PLoS One. 2015; 10: e0139142.
Marcus C. Am J Respir Crit Care Med. 1999; 159: 1527-32.
Lewis KL. Curr Opin Pulm Med. 2002; 8: 493-7.

Epidemiology in children

- Habitual snoring (> 3 times a week) is relatively common 3-12%.
- Epidemiological studies reported variable prevalence rates 1-24%.
- Most likely prevalence 1-5% of all children.
- The most common aetiology is adenoid and tonsil hypertrophy.

Marcus C et al. Pediatrics. 2012; 130: 714-55.
Loghmanee DA. Pediatr Ann. 2010; 39: 784-9.

Epidemiology in children

- Undiagnosis is quite common due to limited access to diagnosis tools (PSG +) and potentially lack of proper understanding of signs and symptoms.
- Because of robust neurological development throughout childhood, young children are particularly susceptible to SDB impact in that specific area.

AAPD. Pediatr Dent. 2017; 38: 87-89.

Epidemiology in children

- Individual with sleep disorder is rarely aware of it - had to be observed by others.
- Symptoms got unnoticed for long time - it may be perceived as normal.

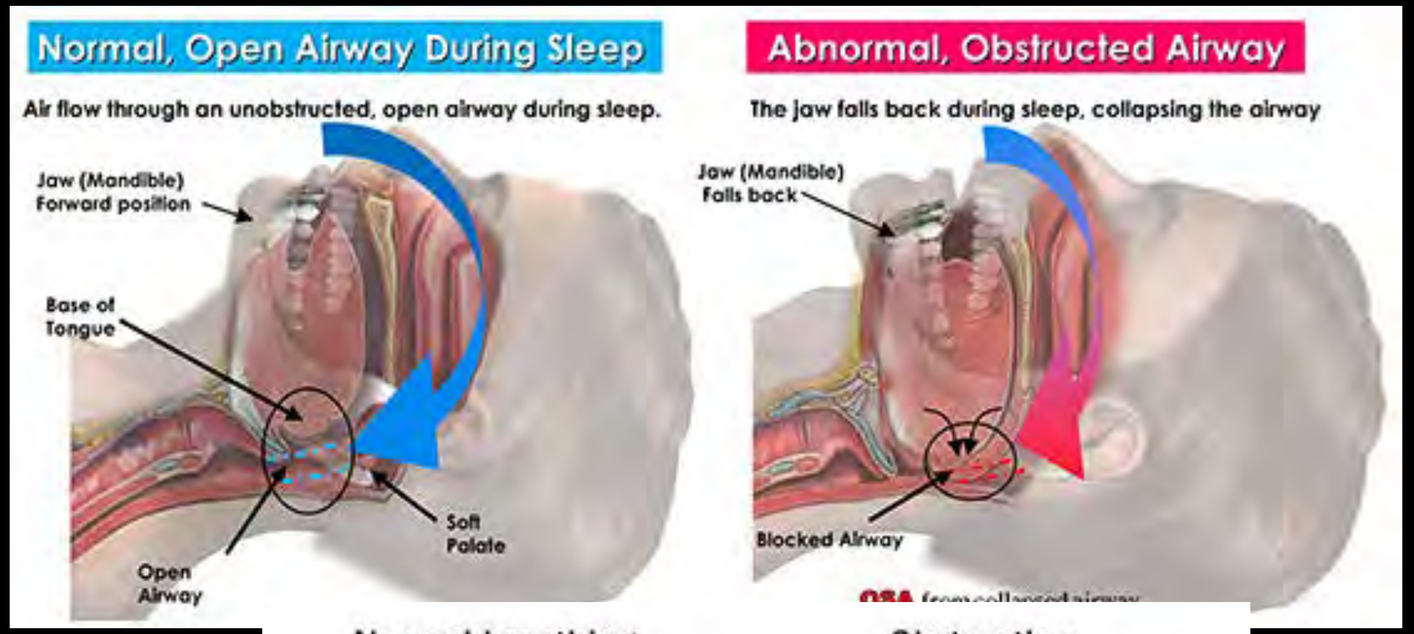
AAPD. Pediatr Dent. 2017; 38: 87-89.

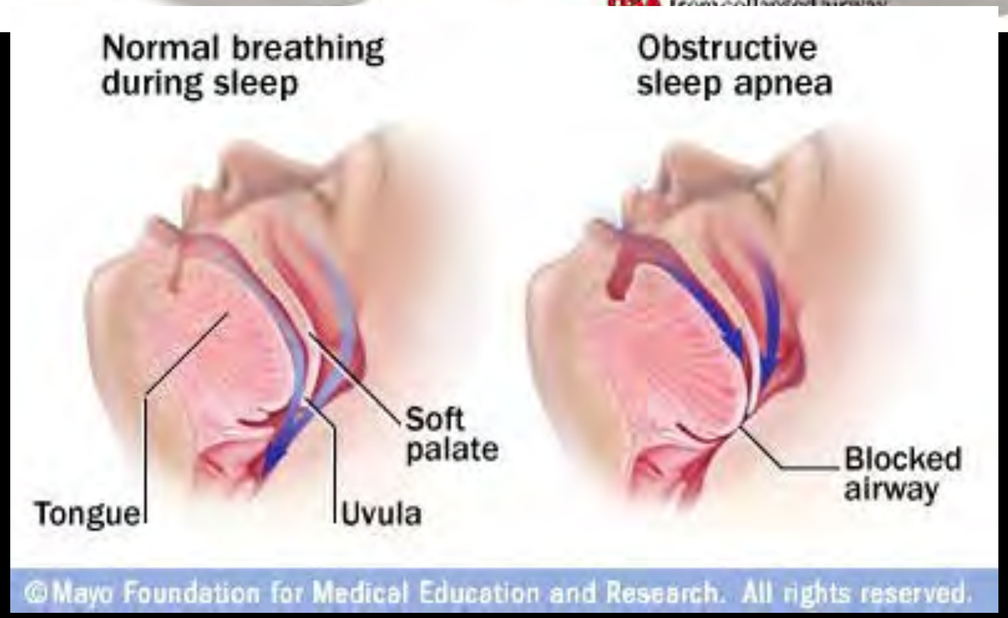
Diagnosis

- Symptoms and PSG characteristics are interpreted substantially different in children compared to adults.

Brunetti. Chest. 2001; 120: 1930-5.

Obstructive Sleep Apnea





OSA Diagnosis

- Standard DX: Overnight polisomnography (PSG)
- Non-validated support from consensus group

OSA Diagnosis

- At home or on the go non-validated alternatives

nPSG Problems

- Onerous - labour intensive
- Inconvenient (specially children)
- Relatively inaccessible - 3 to 6 months wait

nPSG Problems

- It is difficult, however, to define OSA exclusively using PSG-based criteria.
- There is no definitive set of PSG criteria that will reliably discriminate those patients who have OSA and require treatment.

nPSG Problems

- First, the clinically valid cut-off for normal AHI is unclear in children.
- Second, no consensus has been achieved as to whether children with AHI values between the normal cut-off (<1/to 5/hr) should undergo adenotonsillectomy.

nPSG Problems

- In addition, PSG measures are poor predictors of OSA-associated morbidities.
- Patients with similar OSA severity may present with greatly different clinical phenotypes.

nPSG Problems

- Children who are very symptomatic may present a “normal PSG” in the presence of habitual snoring.
- Conversely, asymptomatic snoring children may have concurrent and severe respiratory disturbance in their PSG.

OSA Biomarkers

- de Luca Canto G, Pacheco-Pereira C, Aydinoz S, Major PW, Flores-Mir C, Gozal D. Biomarkers Associated with Obstructive Sleep Apnea: A Scoping Review. Sleep Med Rev 2015; 16: 347-57

Sleep Medicine 16 (2015) 347–357

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Biomarkers associated with obstructive sleep apnea and morbidities: a scoping review

Graziela De Luca Canto [a,b], Camila Pachêco-Pereira [b], Secil Aydinoz [c,d], Paul W. Major [b], Carlos Flores-Mir [b], David Gozal [e,*]

[a] Department of Dentistry, Federal University of Santa Catarina, Florianópolis, SC, Brazil
[b] School of Dentistry, Faculty of Medicine and Dentistry, Edmonton Clinic Health Academy, University of Alberta, 11405-87 Avenue, Edmonton, Alberta T6G 1C9, Canada
[c] GATA Haydarpasa Teaching Hospital, Istanbul, Turkey
[d] Section of Pediatric Sleep Medicine, Department of Pediatrics, The University of Chicago, USA
[e] Department of Pediatrics, Pritzker School of Medicine, Biological Sciences Division, The University of Chicago, 5721 S. Maryland Avenue, MC 8000, Suite K-160, Chicago, IL 60637, USA

ARTICLE INFO

Article history:
Received 10 October 2014
Received in revised form 16 December 2014
Accepted 27 December 2014
Available online 15 January 2015

Keywords:
Review
Sleep apnea
Biomarkers
Morbidity

ABSTRACT

Objective: To map potential biomarkers of obstructive sleep apnea (OSA)-associated morbidities in both adults and children, to identify gaps in current evidence, and to determine the value of conducting a full systematic review.

Methods: A scoping review was undertaken of studies in patients with OSA that evaluated the potential value of biological markers in identifying OSA-associated morbidities. Retained articles were only those studies whose main objective was to identify morbidity biomarkers in subjects with OSA, the latter being confirmed with a full overnight polysomnography (PSG) in a laboratory or at-home settings. The methodology of the selected studies was classified using an adaptation of the evidence-quality criteria recommended by the American Academy of Pediatrics. Additionally the biomarkers were categorized according to their potential clinical applicability.

Results: 572 citations were identified of which 48 met inclusion criteria. Thirty-four studies were conducted in adults and 14 involved children. Most of the studies evaluated blood biomarkers, and presented 31 potential diagnostic biomarkers.

Conclusion: The majority of studies that performed explored blood-based biomarkers, with most not identifying definitive morbidity biomarkers. Of the potentially promising morbidity biomarkers, plasma IL-6 and high sensitivity C-reactive protein appear to exhibit a favorable profile, and may discriminate OSA patients with and without morbidities in both adults and children. MMP 8/14 was retained in children as well as cardiovascular morbidity-associated biomarker. Urinary neurotransmitters may also provide a good tool for screening OSA cognitive morbidity in children.

1. Introduction

Obstructive sleep apnea (OSA) has now been recognized as a major public health issue with potential society-wide consequences involving car or work-related accidents, cognitive and behavioral deficits impairing work performance, and potentially leading to cardiovascular and metabolic dysfunction [1]. Indeed, OSA has been associated with serious morbidities such as endothelial dysfunction [2,3], hypertension [4], cardiovascular disease (CVD) [5–7], cognitive and behavioral dysfunction [8], metabolic disorders such as insulin resistance [9], diabetes [10], and dyslipidemias [11], erectile dysfunction in men [12], nocturnal enuresis in children [13], and excessive daytime sleepiness (EDS) [14,15]. Consequently, healthcare costs are substantially increased in patients with OSA, accounting either directly or via its associated morbidities for a substantial proportion of all medical-related costs [16–18].

In adults, the prevalence of OSA varies widely, from 14.7% to 36.5%, depending on age, gender, and ethnicity [19]. It is generally higher in males [19], and although the prevalence in Hispanics is similar to white Caucasians, it is significantly higher in African

Abbreviations: OSA, obstructive sleep apnea; CVD, cardiovascular disease; DTA, diagnostic test accuracy; EDS, excessive daytime sleepiness; PSG, polysomnography; RBC, exhaled breath condensate; RR, risk ratio; OR, odds ratio; AHI, apnea hypopnea index; OAI, obstructive apnea index; RDI, respiratory disturbance index; hrTST, hour of total sleep time; ROC, receiver operating characteristic; AASM, American Academy of Sleep Medicine; hsCRP, high-sensitivity C-reactive protein; IGF, insulin-like growth factor; MMP, matrix-related protein; IL-6, interleukin-6.

* Corresponding author. Department of Pediatrics, The University of Chicago, 5721 S. Maryland Avenue, MC 8000, Suite K-160, Chicago, IL 60637, USA. Tel.: +1 773 702 3360; fax: +1 773 926 0756.
E-mail address: dgozal@uchicago.edu (D. Gozal).

http://dx.doi.org/10.1016/j.smrv.2014.11.007
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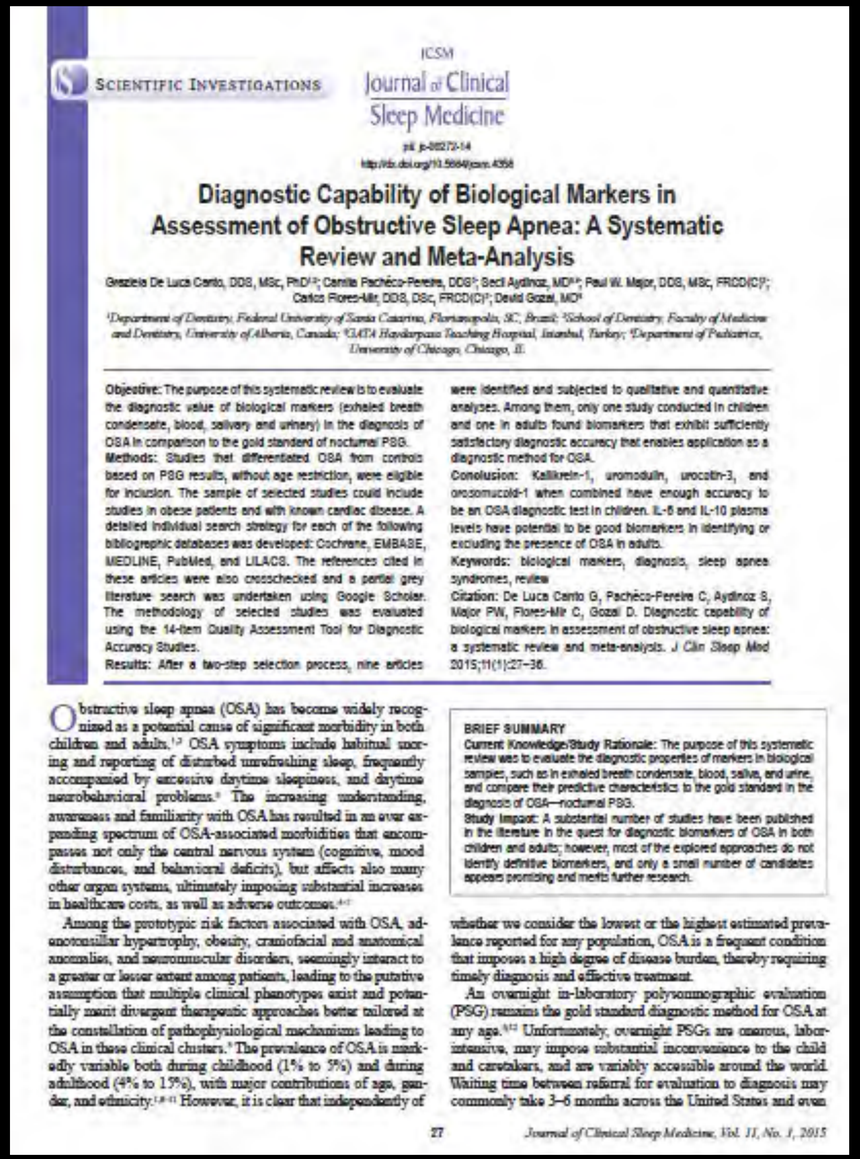
OSA Biomarkers

de Luca Canto G, Pacheco-Pereira C, Aydinoz S, Major PW, Flores-Mir C, Gozal D. Diagnostic Capability of Biological Markers in assessment of Obstructive Sleep Apnea: A Systematic Review and Meta-Analysis. Journal of Clinical Sleep Medicine 2015; 11: 27-36.



SCIENTIFIC INVESTIGATIONS

ICSM Journal of Clinical Sleep Medicine

pii: jc-00272-14

http://dx.doi.org/10.5664/jcsm.4358

Diagnostic Capability of Biological Markers in Assessment of Obstructive Sleep Apnea: A Systematic Review and Meta-Analysis

Graziela De Luca Canto, DDS, MSc, PhD[1,2]; Camila Pacheco-Pereira, DDS[3]; Secil Aydinoz, MD[4,5]; Paul W. Major, DDS, MSc, FRCD(C)[3]; Carlos Flores-Mir, DDS, DSc, FRCD(C)[3]; David Gozal, MD[5]

[1]Department of Dentistry, Federal University of Santa Catarina, Florianopolis, SC, Brazil; [2]School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Canada; [3]GATA Haydarpasa Teaching Hospital, Istanbul, Turkey; [4]Department of Pediatrics, University of Chicago, Chicago, IL.

Objective: The purpose of this systematic review is to evaluate the diagnostic value of biological markers (exhaled breath condensate, blood, salivary and urinary) in the diagnosis of OSA in comparison to the gold standard of nocturnal PSG.

Methods: Studies that differentiated OSA from controls based on PSG results, without age restriction, were eligible for inclusion. The sample of selected studies could include studies in obese patients and with known cardiac disease. A detailed individual search strategy for each of the following bibliographic databases was developed: Cochrane, EMBASE, MEDLINE, PubMed, and LILACS. The references cited in these articles were also crosschecked and a partial grey literature search was undertaken using Google Scholar. The methodology of selected studies was evaluated using the 14-item Quality Assessment Tool for Diagnostic Accuracy Studies.

Results: After a two-step selection process, nine articles were identified and subjected to qualitative and quantitative analyses. Among them, only one study conducted in children and one in adults found biomarkers that exhibit sufficiently satisfactory diagnostic accuracy that enables application as a diagnostic method for OSA.

Conclusion: Kallikrein-1, uromodulin, urocortin-3, and orosomucoid-1 when combined have enough accuracy to be an OSA diagnostic test in children. IL-6 and IL-10 plasma levels have potential to be good biomarkers in identifying or excluding the presence of OSA in adults.

Keywords: biological markers, diagnosis, sleep apnea syndromes, review

Citation: De Luca Canto G, Pacheco-Pereira C, Aydinoz S, Major PW, Flores-Mir C, Gozal D. Diagnostic capability of biological markers in assessment of obstructive sleep apnea: a systematic review and meta-analysis. *J Clin Sleep Med* 2015;11(1):27–36.

Obstructive sleep apnea (OSA) has become widely recognized as a potential cause of significant morbidity in both children and adults.[1,2] OSA symptoms include habitual snoring and reporting of disturbed unrefreshing sleep, frequently accompanied by excessive daytime sleepiness, and daytime neurobehavioral problems.[3] The increasing understanding, awareness and familiarity with OSA has resulted in an ever expanding spectrum of OSA-associated morbidities that encompasses not only the central nervous system (cognitive, mood disturbances, and behavioral deficits), but affects also many other organ systems, ultimately imposing substantial increases in healthcare costs, as well as adverse outcomes.[4-7]

Among the prototypic risk factors associated with OSA, adenotonsillar hypertrophy, obesity, craniofacial and anatomical anomalies, and neuromuscular disorders, seemingly interact to a greater or lesser extent among patients, leading to the putative assumption that multiple clinical phenotypes exist and potentially merit divergent therapeutic approaches better tailored at the constellation of pathophysiological mechanisms leading to OSA in these clinical clusters.[8] The prevalence of OSA is markedly variable both during childhood (1% to 5%) and during adulthood (4% to 15%), with major contributions of age, gender, and ethnicity.[1,9-11] However, it is clear that independently of whether we consider the lowest or the highest estimated prevalence reported for any population, OSA is a frequent condition that imposes a high degree of disease burden, thereby requiring timely diagnosis and effective treatment.

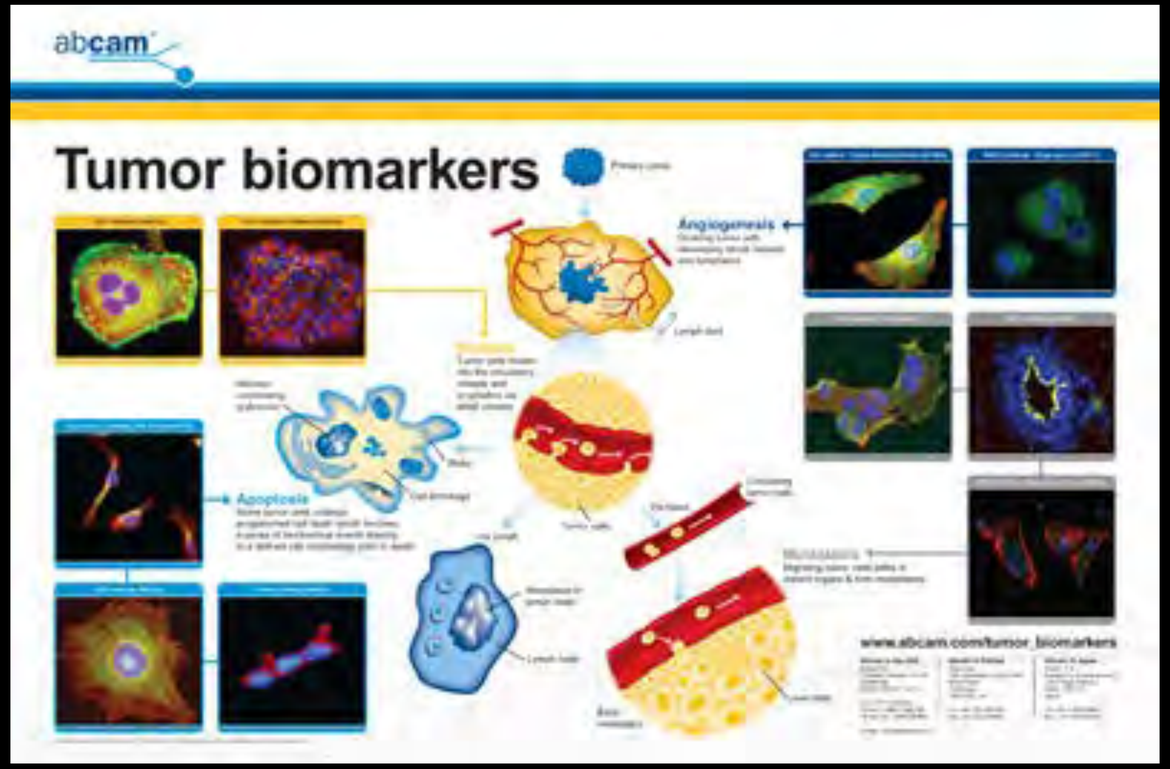
An overnight in-laboratory polysomnographic evaluation (PSG) remains the gold standard diagnostic method for OSA at any age.[9,12] Unfortunately, overnight PSGs are onerous, labor-intensive, may impose substantial inconvenience to the child and caretakers, and are variably accessible around the world. Waiting time between referral for evaluation to diagnosis may commonly take 3–6 months across the United States and even

BRIEF SUMMARY

Current Knowledge/Study Rationale: The purpose of this systematic review was to evaluate the diagnostic properties of markers in biological samples, such as in exhaled breath condensate, blood, saliva, and urine, and compare their predictive characteristics to the gold standard in the diagnosis of OSA—nocturnal PSG.

Study Impact: A substantial number of studies have been published in the literature in the quest for diagnostic biomarkers of OSA in both children and adults; however, most of the explored approaches do not identify definitive biomarkers, and only a small number of candidates appears promising and merits further research.

OSA Biomarkers



- A biomarker is a “biological molecule found in blood, other body fluids, or tissues that is a sign of a normal or abnormal processes, or of a condition or disease”.

OSA Biomarkers

Blood

Exhaled breath condensate

Saliva

Urine

Table 4 Diagnostic test accuracy. Measurements for combined biomarkers (children).

Author	Age (range in years)	Sample Size N.	OSA/NON-OSA	Biomarker	Sensitivity/Specificity (%)	PPV/ NPV (%) #	LR+/ LR- #	DOR #	Younden's Index Value #
Gozal et al[24]	2-9	120	60/60	Kallikrein-1 Uromodulin Urocortin-3 Orosomucoid-1	100/97	97/100	28.60/zero	∞	0.97
Kheirandish-Gozal et al[31]	6.3	70	50/20	Urinary Neurotransmitters	82/90	95/67	8.20/0.20	41.00	0.72

Data not available in the original article. The authors calculated data from information available in the article. OSA=obstructive sleep apnea PPV=positive predictive value. NPV=negative predictive value. LR+=positive likelihood ratio. LR-=negative likelihood ratio. DOR= diagnostic odds ratio.

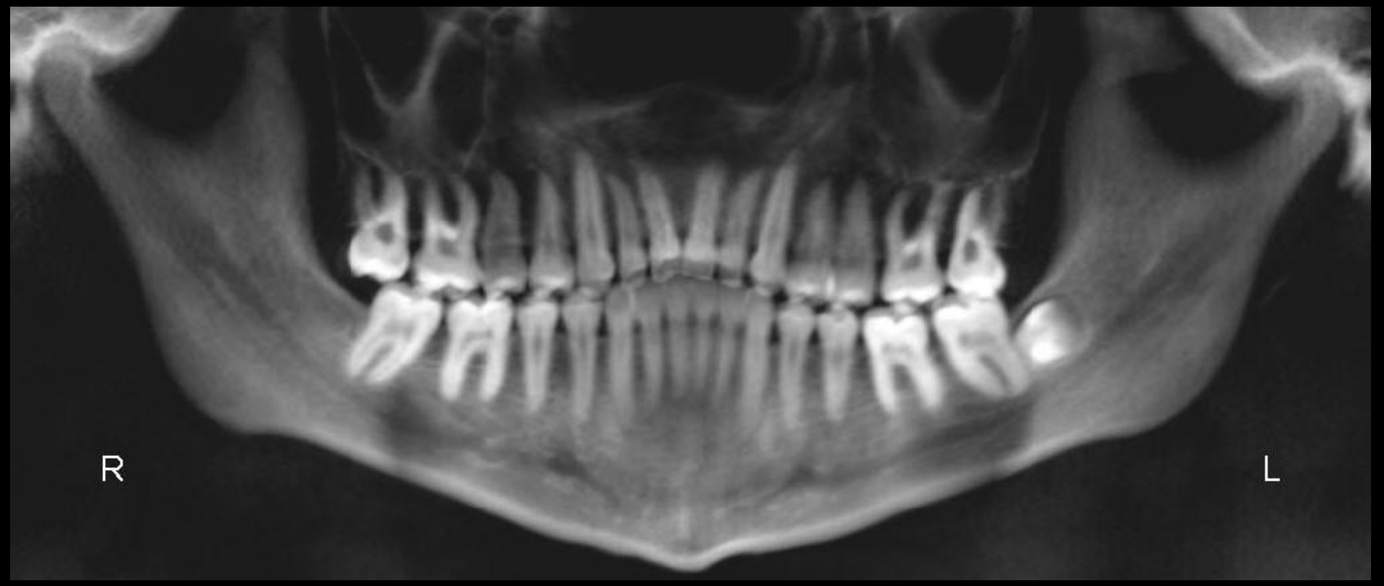
Table 5 Diagnostic test accuracy (adults).

Author	Age (mean in years)	Sample Size N.	OSA/ NON-OSA	Biomarker	Sensitivity (%)/Specificity (%)	PPV (%) #/ NPV (%) #	LR+ #/ LR- #	DOR #	Younden's Index Value #
Guo et al[28]	47.8#	63	54/9	TRX	91/78	96/58	4.08	34.00	0.69
Hirotsu et al[25]	44.6#	456 (male)	192/264	Uric acid	62/57	51/67	1.44	0.02	0.26
Hirotsu et al[25]	44.6#	565 (female)	147/418	Uric acid	63/64	38/64	1.75	3.01	0.27
Lentini et al[29]	54.9	201	182/19	CK	43/95	99/14	8.14	13.57	0.38
Li et al[26]	44.0#	100	68/32	IL-6	100/100	100/100	∞	∞	1.00
Li et al[26]	44.0#	100	68/32	IL-10	100/97	99/100	32.00	∞	0.97
Ursavas et al[27]	50.5#	73	39/34	ICAM-1	69/82	81/70	3.92	10.59	0.51
Ursavas et al[27]	50.5#	73	39/34	VCAM-1	74/65	71/69	2.11	5.41	0.39

Data not available in the original article. The authors calculated data from information available in the article. OSA=obstructive sleep apnea. PPV=positive predictive value. NPV=negative predictive value. LR+=positive likelihood ratio. LR-=negative likelihood ratio. DOR= diagnostic odds ratio. CK=creatine phosphokinase. TRX=thioredoxin, IL-6=interleukin-6, IL-10=interleukin-10, ICAM-1=intercellular adhesion molecule 1. VCAM-1=vascular cell adhesion molecule-1.

Paediatric Sleep Disorder Breathing

Case #2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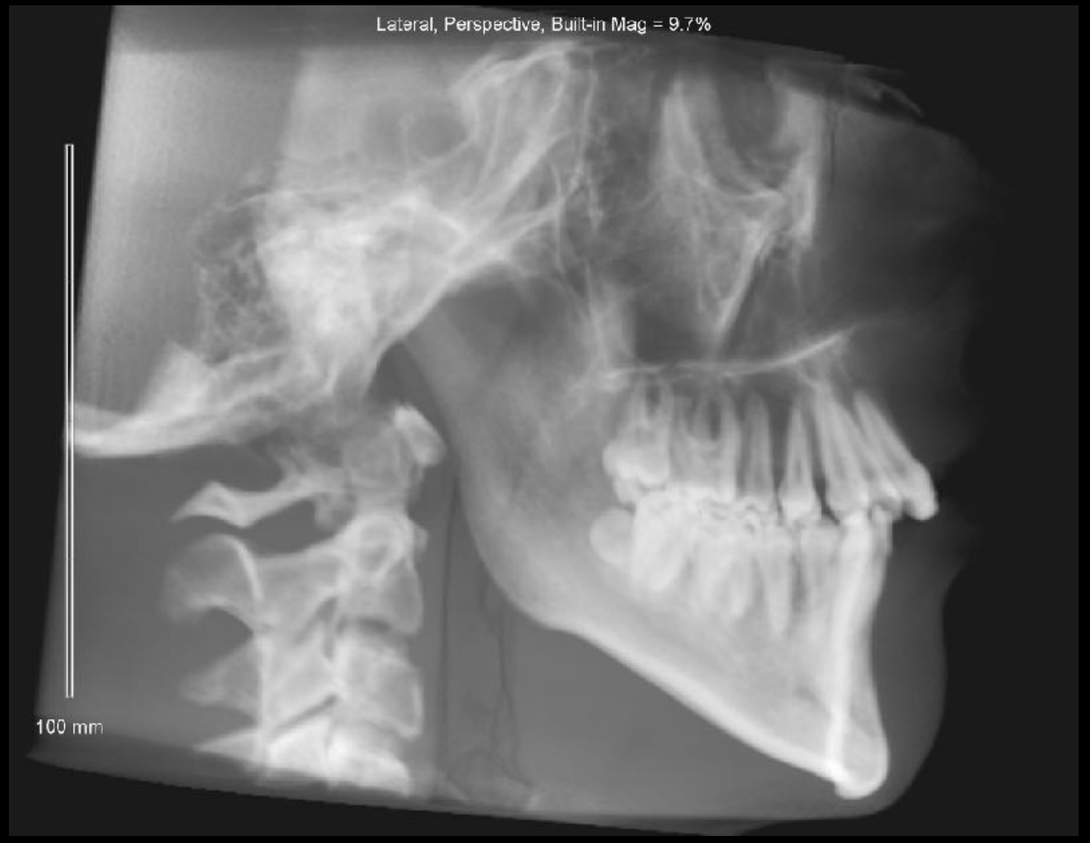
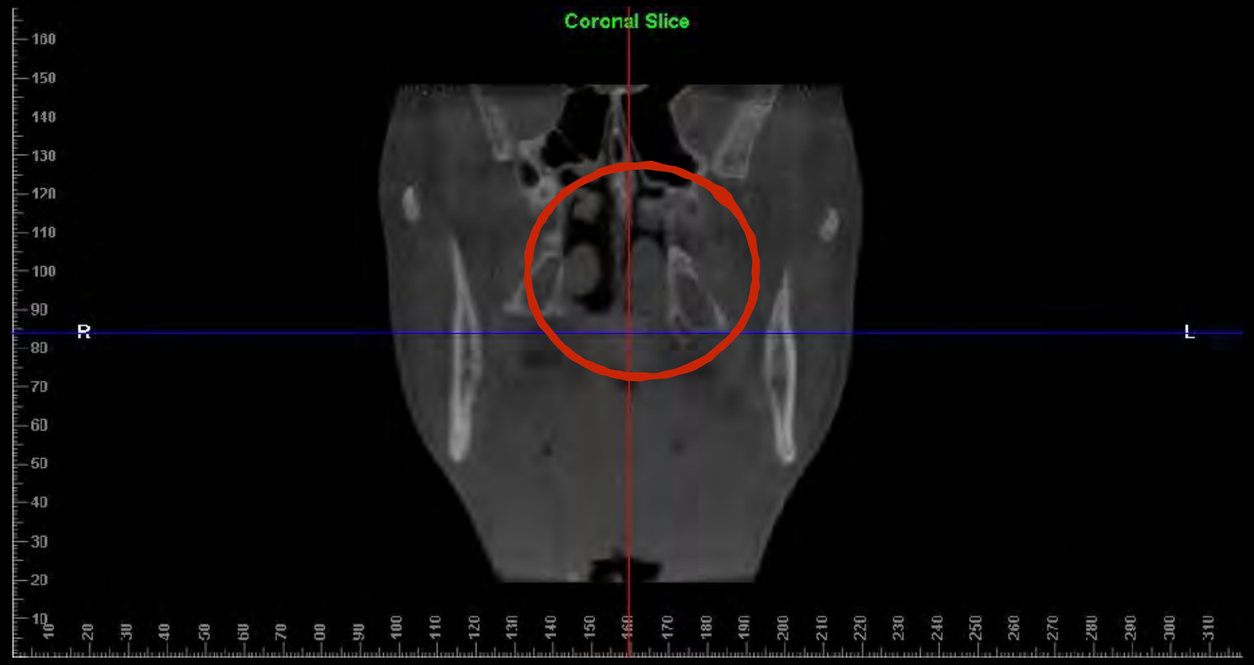
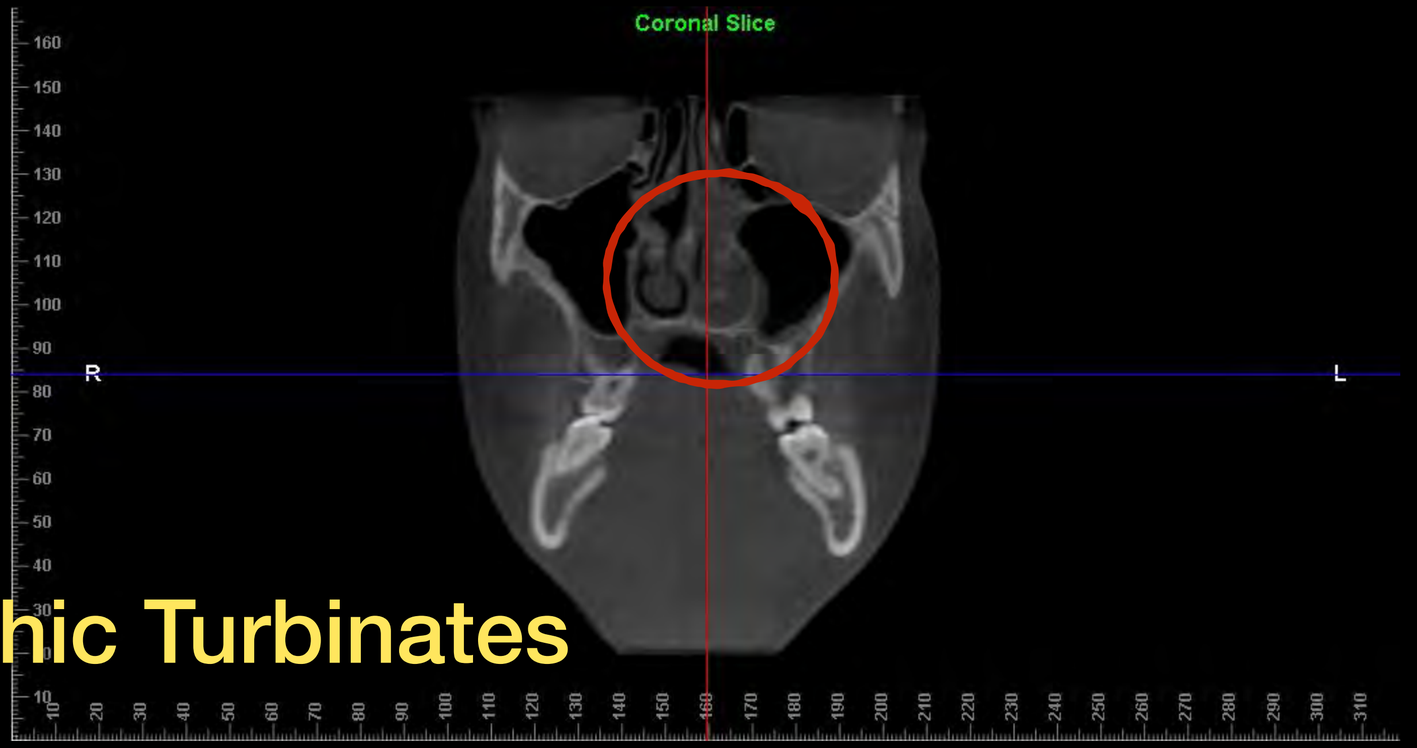

Lateral, Perspective, Built-in Mag = 9.7%
100 mm





Hypertrophic Turbinates

Sleep Disorder Breathing - Symptoms

Snoring >3 times a week

Observed apneas

Abrupt awakening with breath shortness

Dry mouth/Sore throat

Difficulty to stay asleep

Restlessness

Sweating

Frequent morning headaches

AAPD. Pediatr Dent. 2017; 38: 87-89.

Sleep Disorder Breathing Index

AHI in Adults (Apnea/Hypoxia Index):

Number of apnea and hypnosis:

Mild 5-15 events/hour

Moderate 15-30 events/hour

Severe >30 events/hour

Sleep Disorder Breathing Index

AHI in Children (Apnea/Hypoxia Index):

Number of apnea and hypnosis:

Mild 1-5 events/hour

Moderate 5-10 events/hour

Severe >10 events/hour

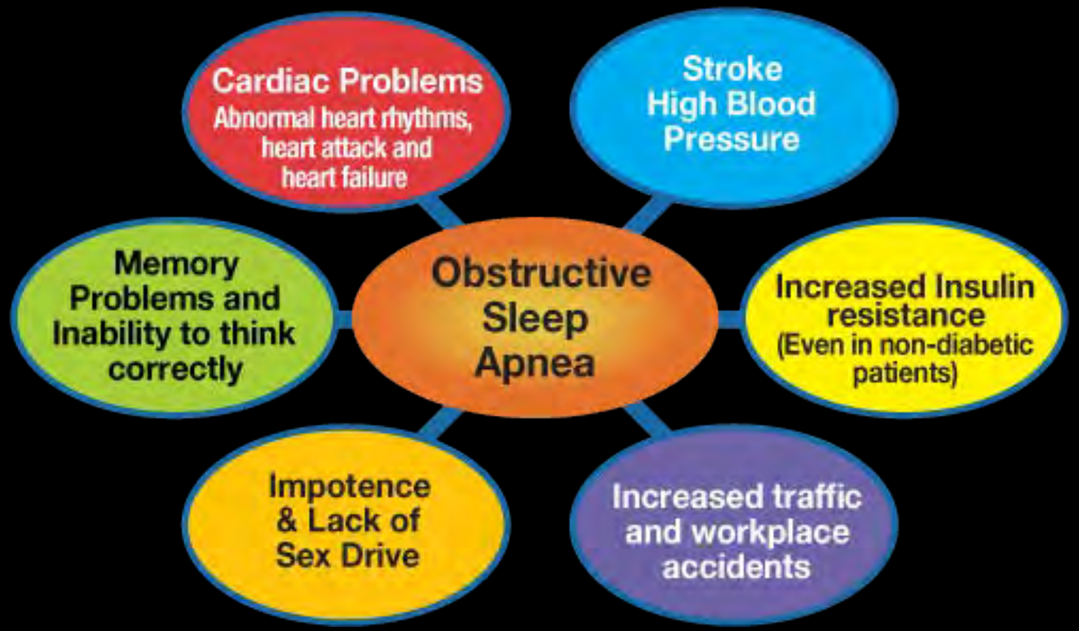
Marcus CL et al. Pediatrics. 2012; 130: 714-25.

Sleep Disorder Breathing

Can act alone or combined with other general health diseases (cardiovascular, endocrine, etc).

OSA Health Impact

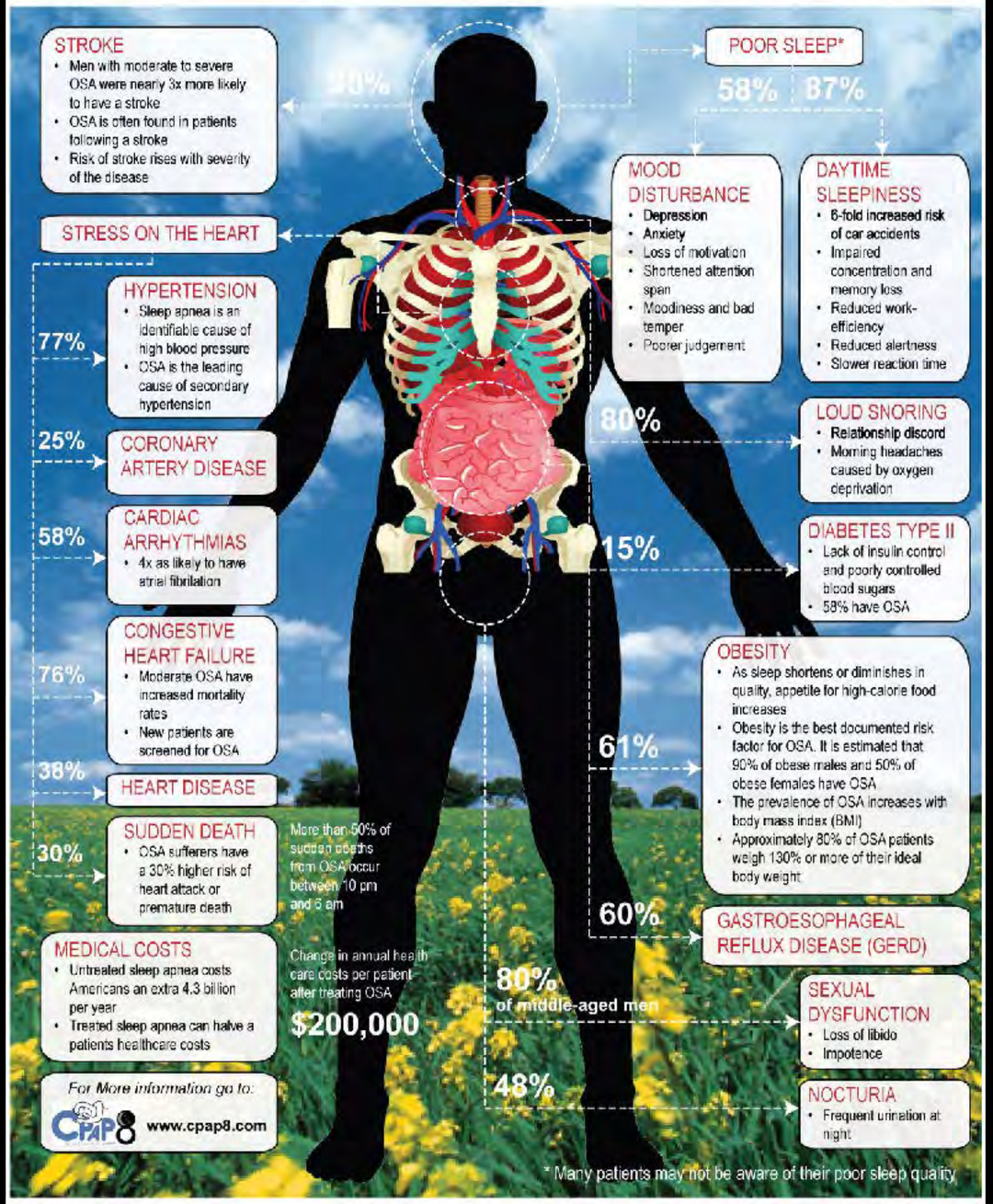
- Major public concern
- Associated to:
  - behavioural and cognitive deficits
  - increased cardiovascular morbidity
  - increased risk of metabolic dysfunction



http://helptostopsnoring.com/what-exactly-is-sleep-apnea/

THE CONSEQUENCES OF SLEEP APNEA

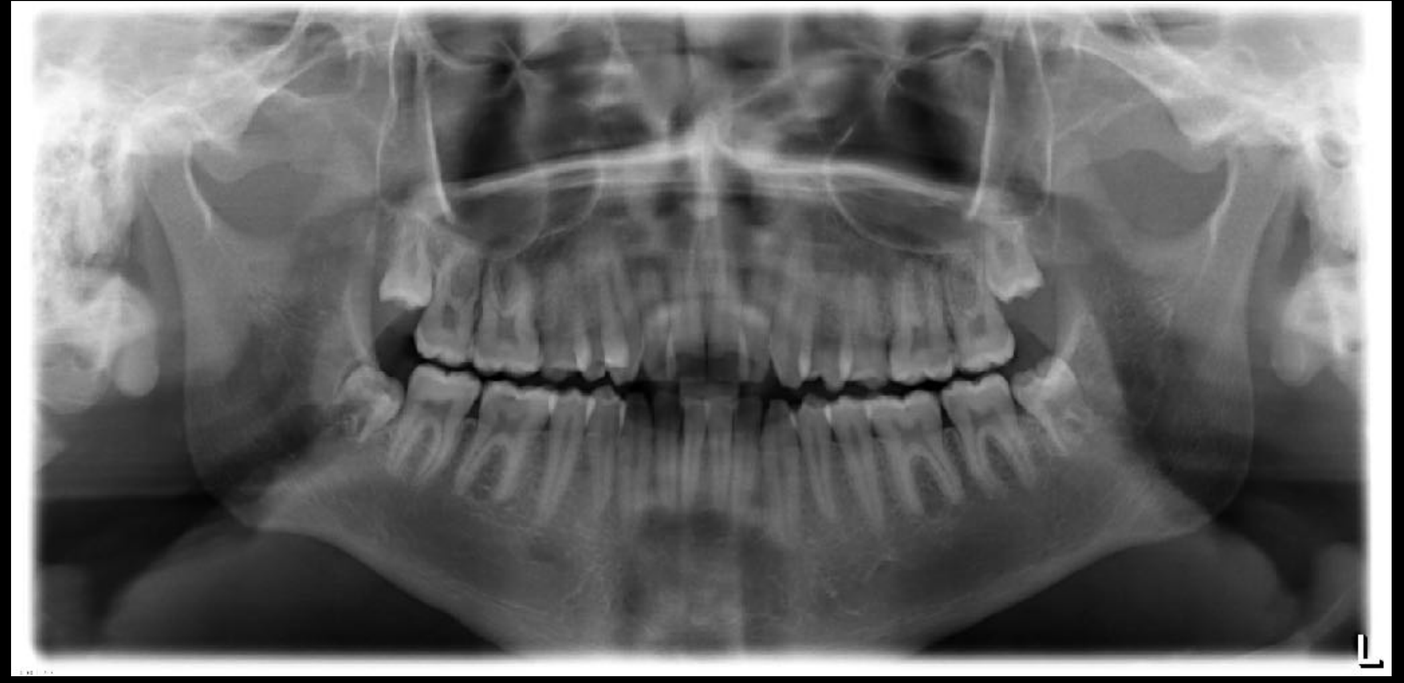
Sleep Apnea afflicts 1 in every 5 Americans. What other problems arise for OSA patients?



www.snorecentre.com/blog/2013/03/27/infographic-the-consequences-of-obstructive-sleep-apnoea/trackback/

Paediatric Sleep Disorder Breathing

Case #3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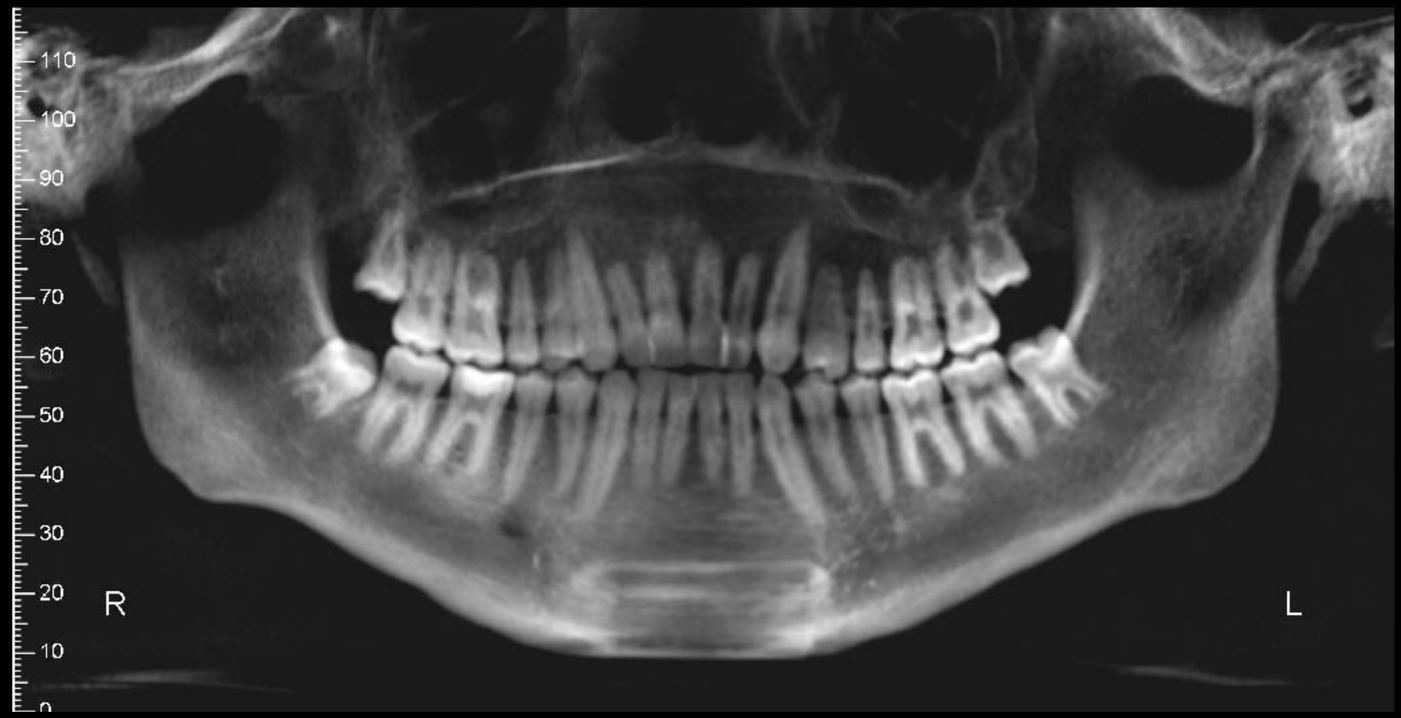
110
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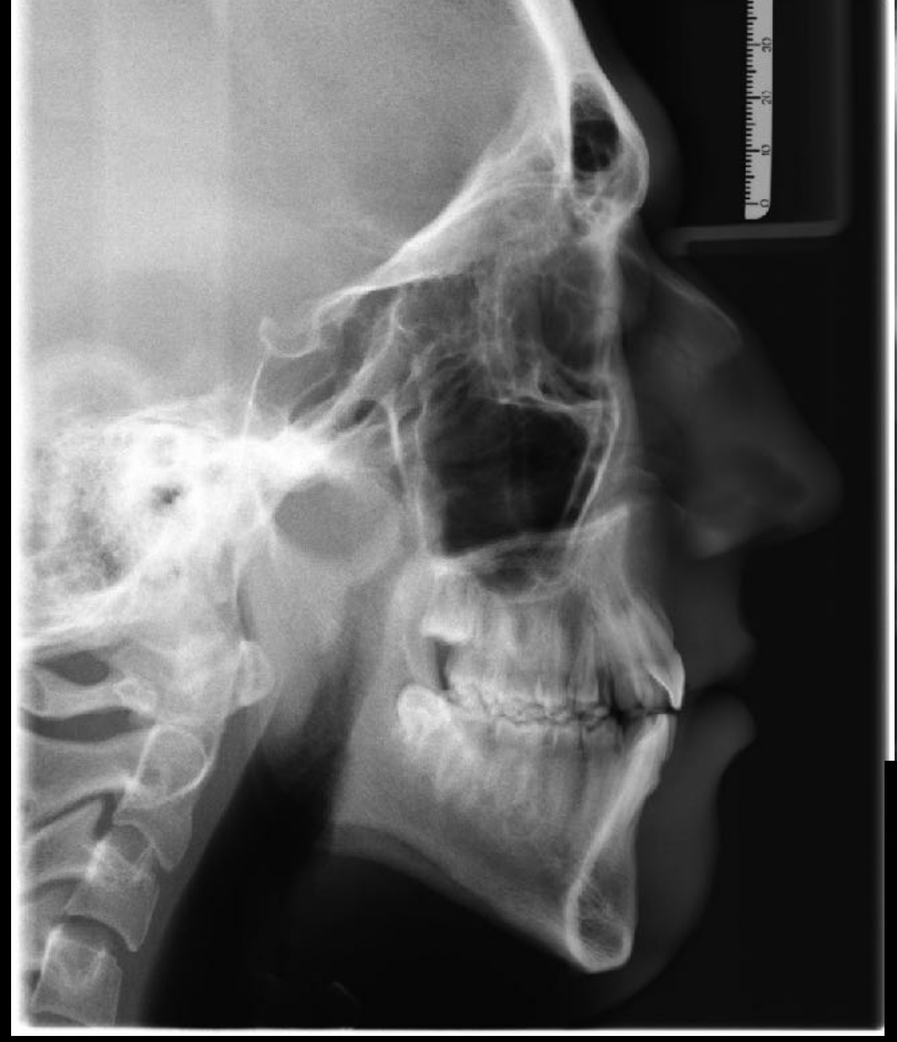
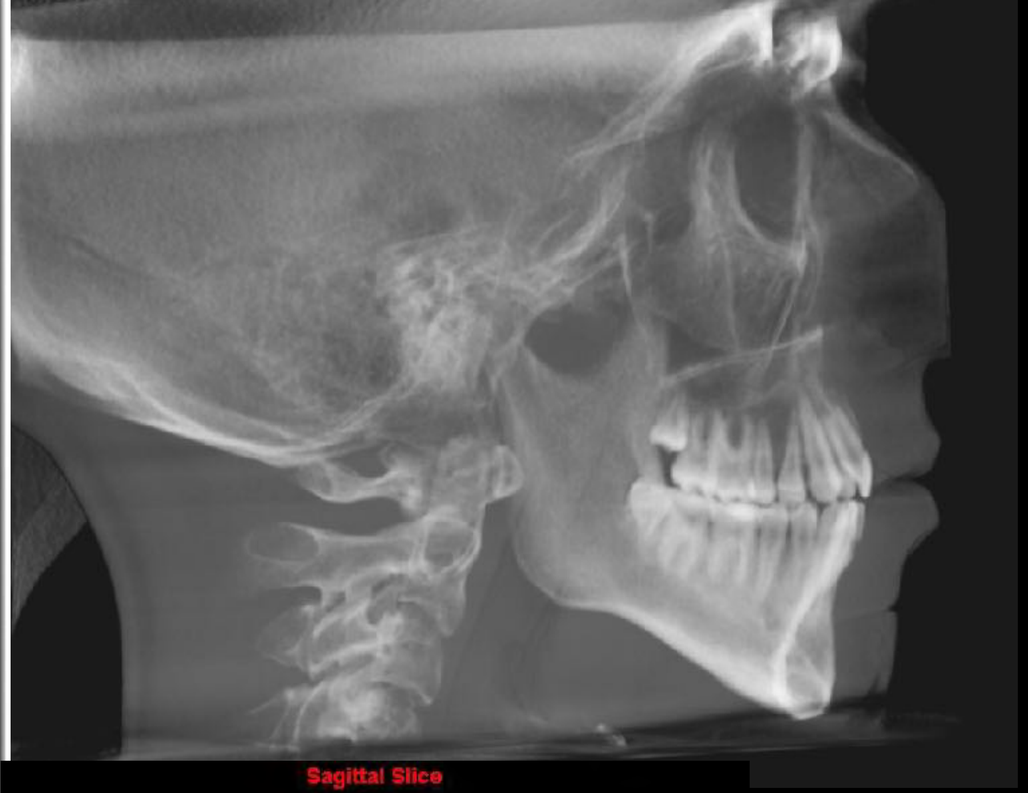
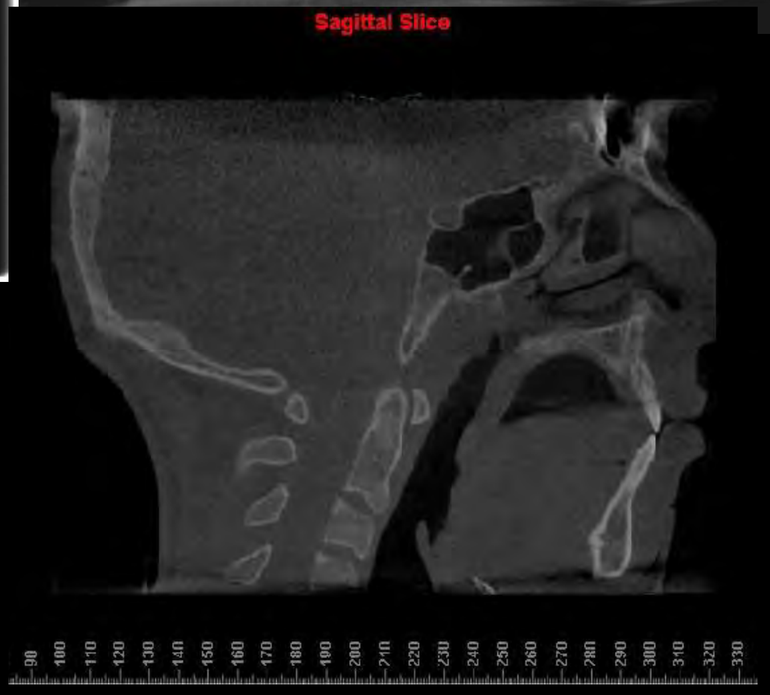
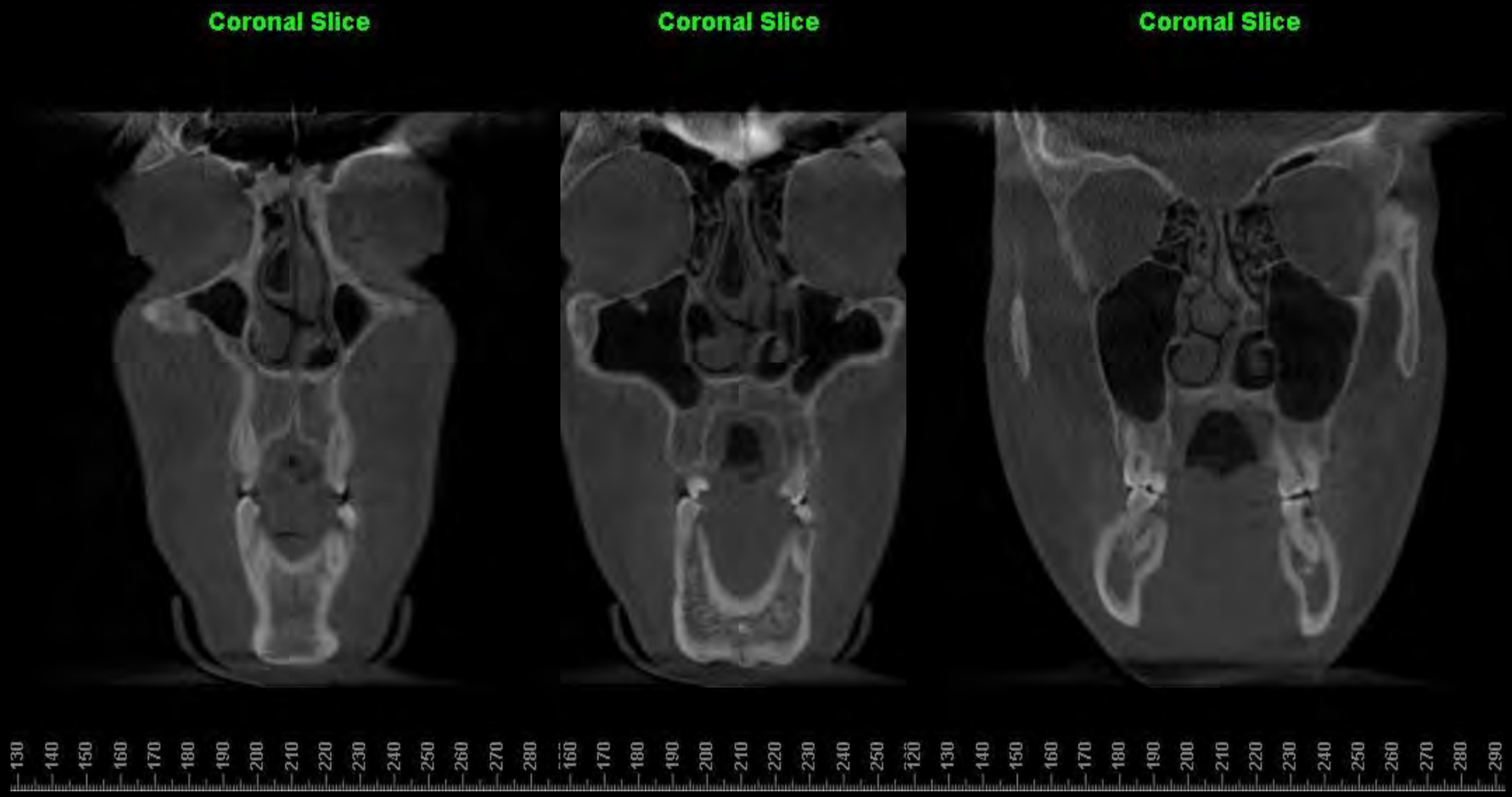




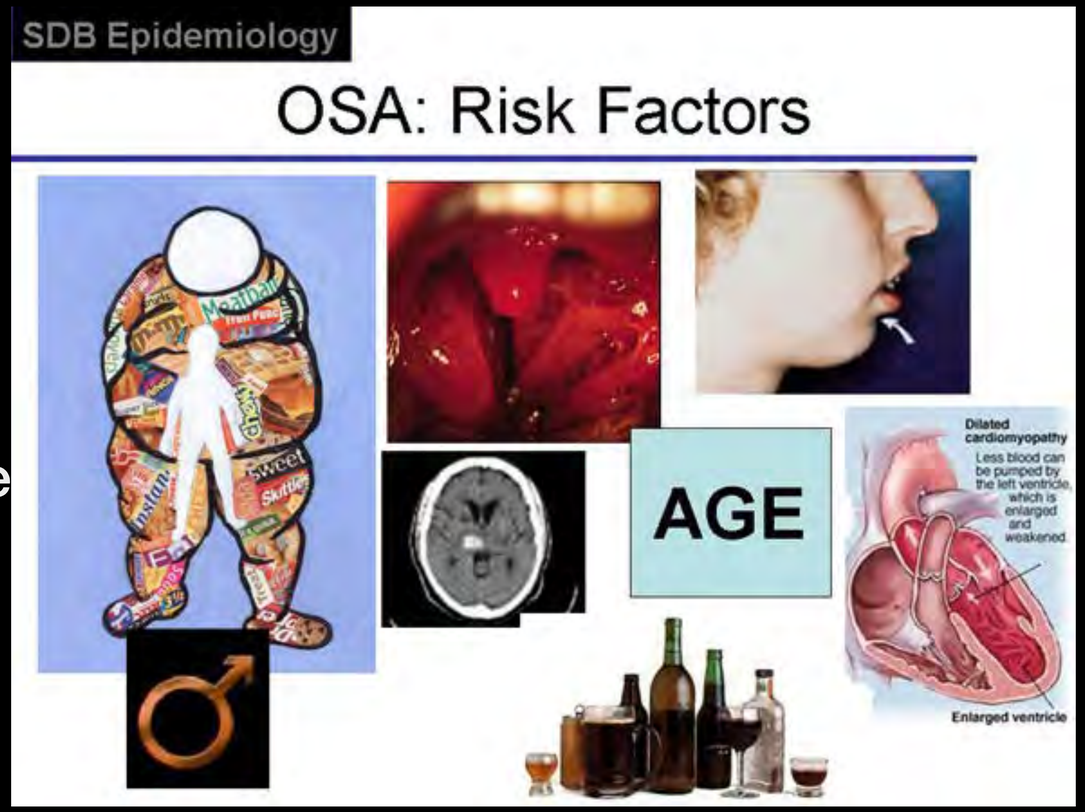

Sagittal Slice



Deviated Nasal Septum -

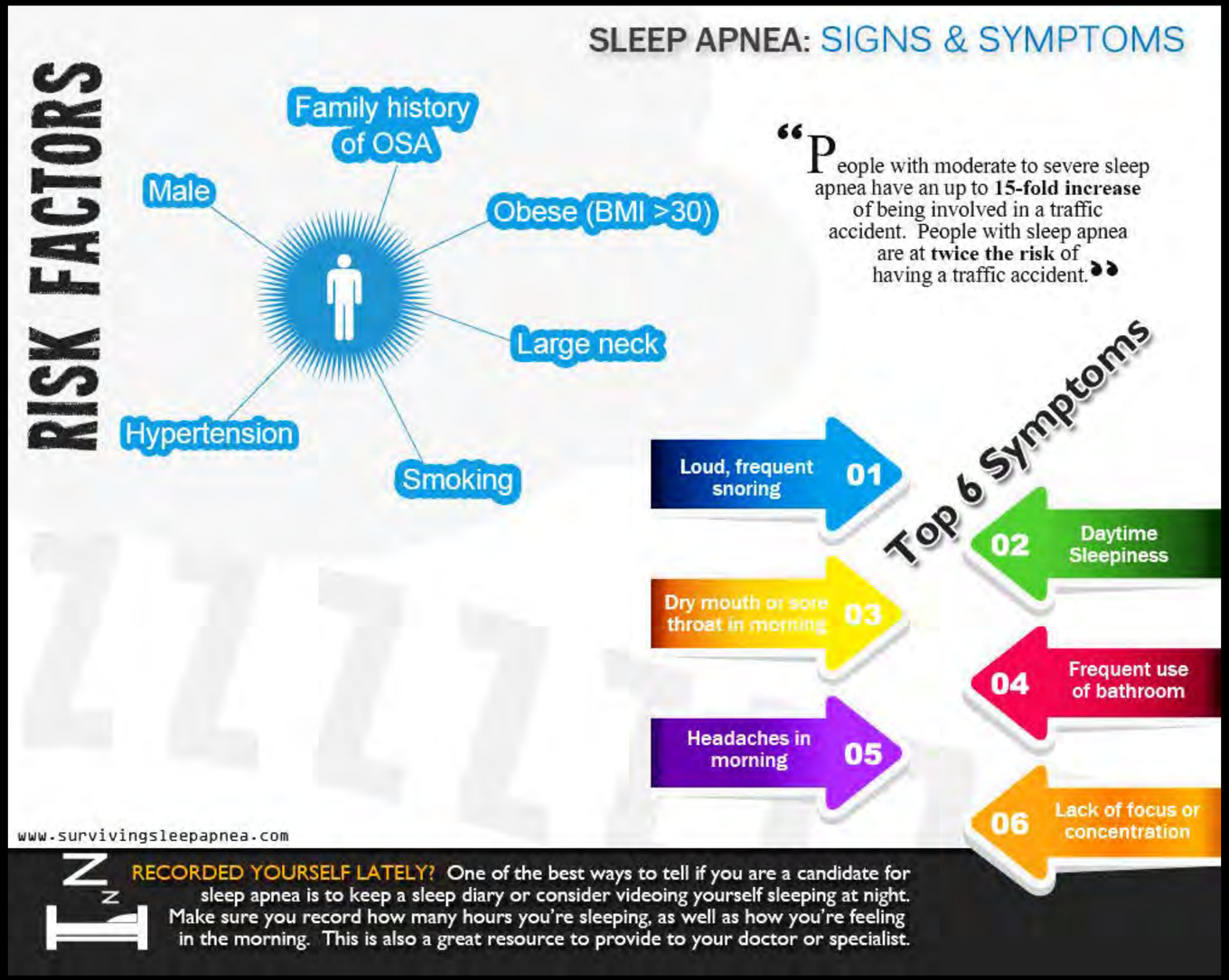
OSA Risk Factors Adults

- Prevalence varies - 15% to 37%
- > males than females (34% to 15%)
- Increased risk for Afro Americans and Asians
- Obesity - BMI - Short neck w/ large circamference
- Upper airway anatomy - obstruction
- Craniofacial anomalies - genetics
- Smoking
- Medication (alcohol, benzodiazepines)



http://www.dom.pitt.edu/PACCM/images/faculty-figures/givelberfigure1.jpg

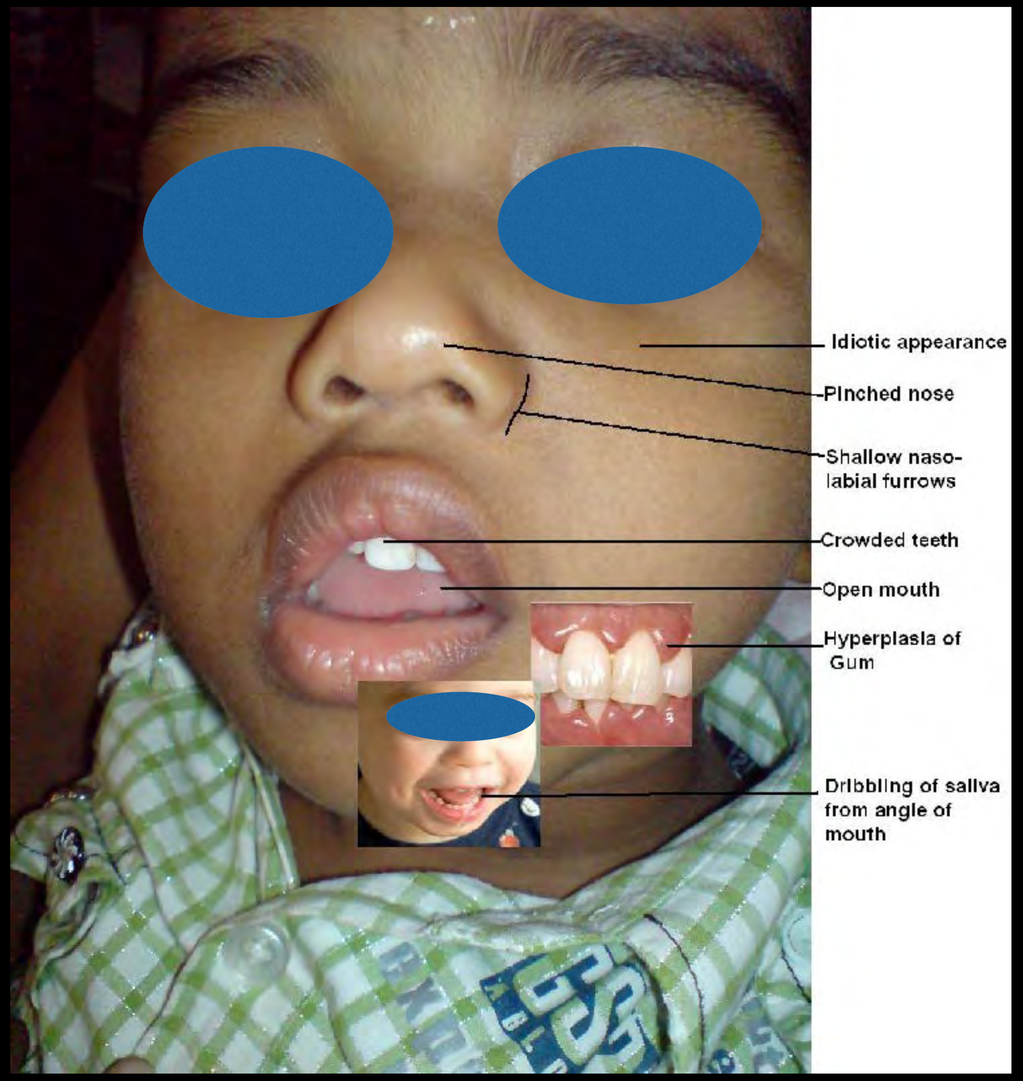
OSA Risk Factors Adults



https://survivingsleepapnea.com/wp-content/uploads/2013/07/Infographic3.jpg

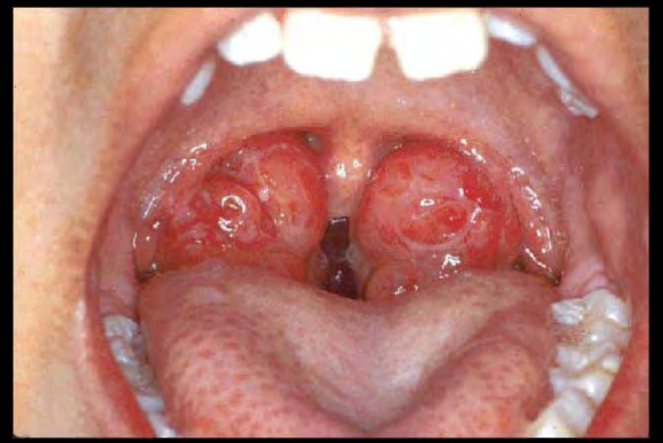
OSA Risk Factors Children

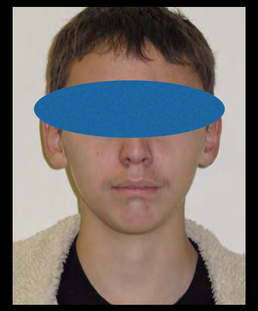
- Adenotonsillar hypertrophy
- Obesity
- Craniofacial anomalies (upper/lower jaw)
- Neuromuscular disorders (cerebral palsy)
- Syndromes affecting upper airway anatomy (Treacher Collins, Pierre-Robin, Marfans, Apert, Trisomy 21)

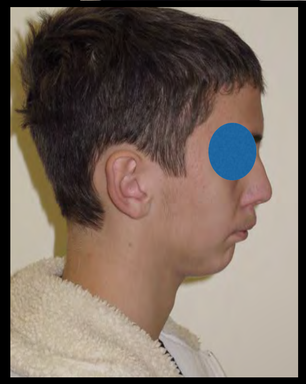


POSA Etiology

- Tonsil and/or Adenoids hypertrophy most common cause. Chronic rhinitis and septum deviation also mentioned.
- Obesity
- Craniofacial anomalies
- Neuromuscular disorders
- Nasal abnormalities
- Preterm birth







POSA Etiology

- Waist circumference
- Metabolic factors
- Neck circumference
- Ethnicity
- Asthma
- Local environmental irritants

Clinical Approach - Screening

- **Medical history** - Allergic rhinitis, frequent colds or sinusitis, snoring, attention disorders, sleepiness during the day
- **Clinical examination** - mouth posture, craniofacial characteristics, tonsilar size

PSDB & Craniofacial Development

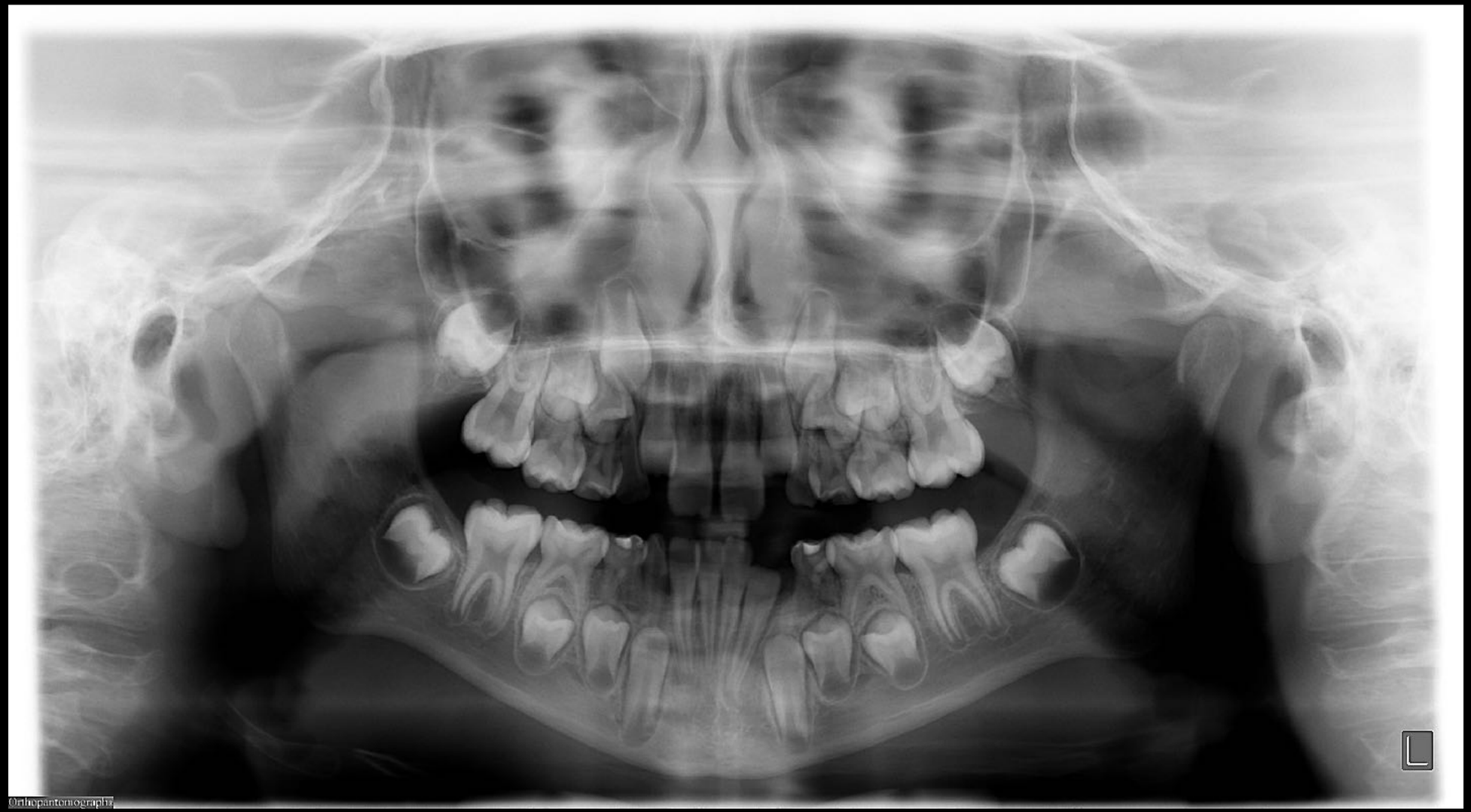
- Cause or effect? Children with POSA frequently have abnormalities in craniofacial morphology.
- Common observations: steep mandibular plane, retrusive chin, inferior hyoid bone, transversely constricted maxilla are.

PSDB & Craniofacial Development

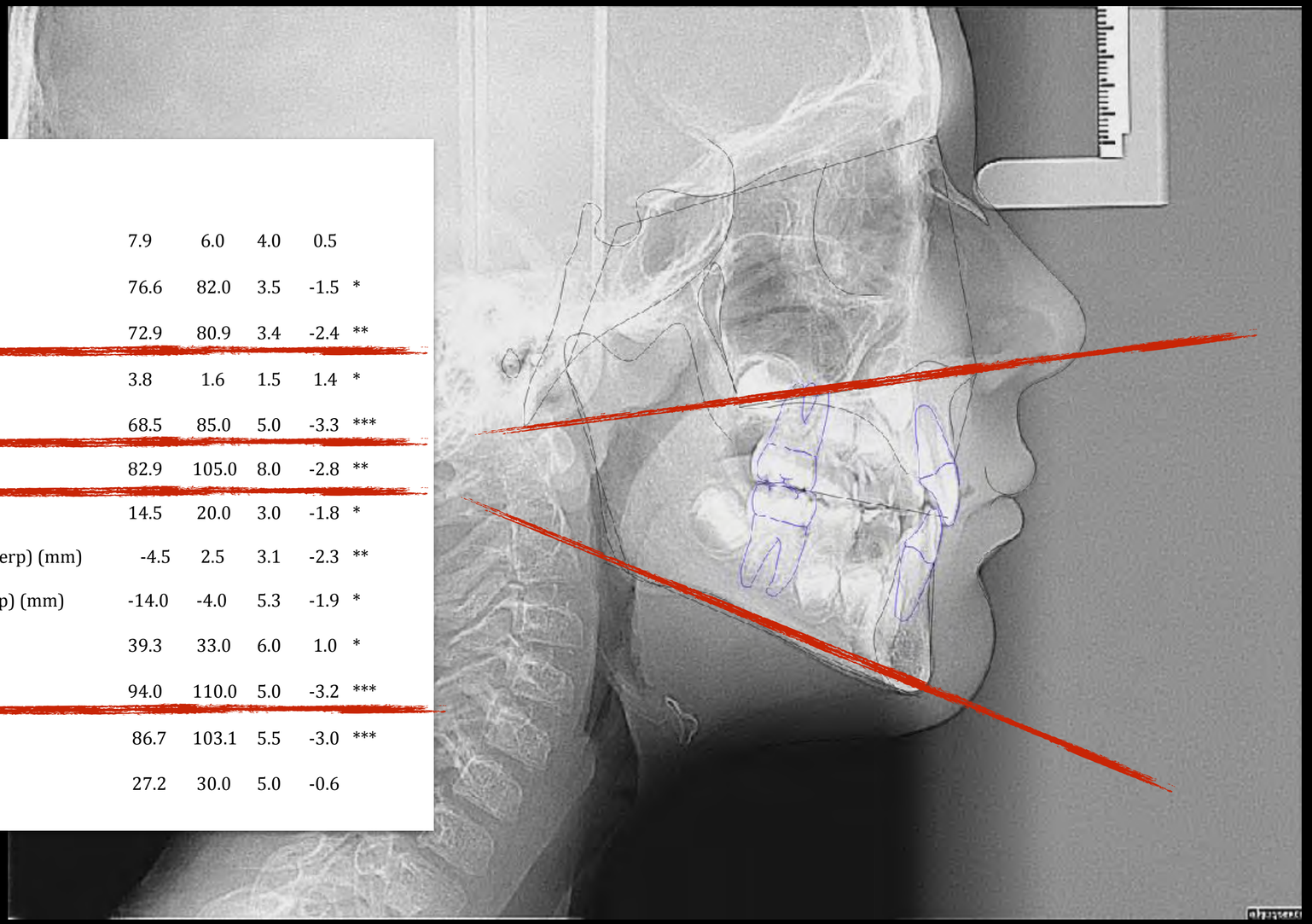
- These craniofacial variations targets for orthodontic interceptive therapy (functional appliances and ME) in mild to moderate POSA children.
- These approaches are similar in patients with these craniofacial characteristics regardless of their POSA status.

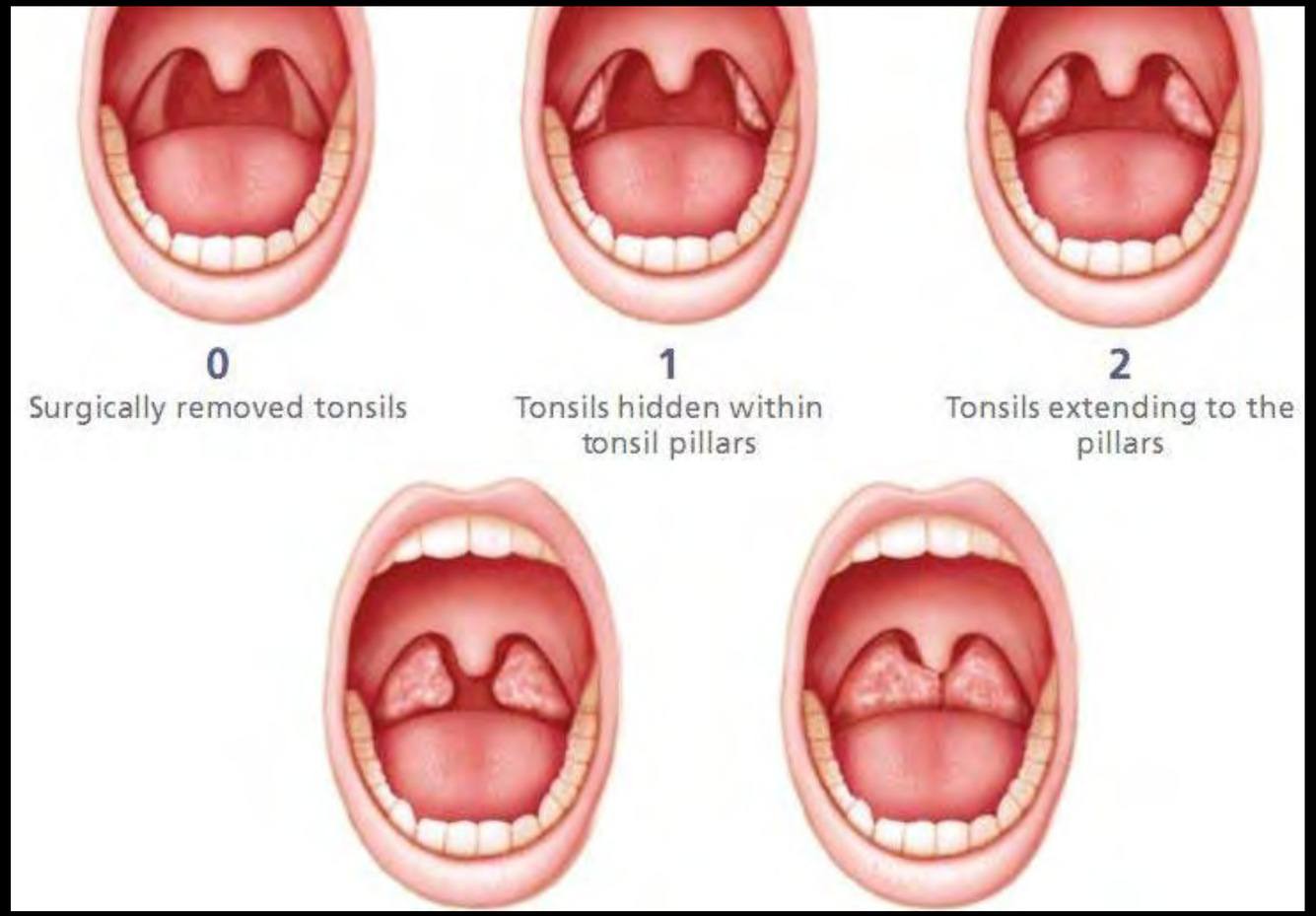
Paediatric Sleep Disorder Breathing

Case #4

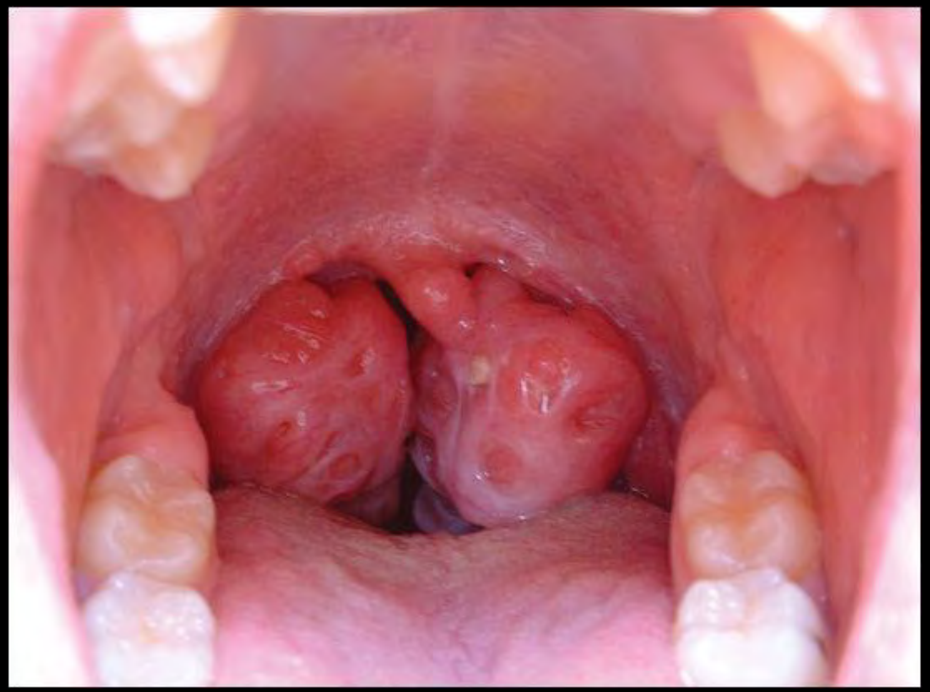

Orthopantomograph

FH - SN (∫)	7.9	6.0	4.0	0.5	
SNA (∫)	76.6	82.0	3.5	-1.5	*
SNB (∫)	72.9	80.9	3.4	-2.4	**
ANB (∫)	3.8	1.6	1.5	1.4	*
Mx Unit Length (Co-ANS)	68.5	85.0	5.0	-3.3	***
Md Unit Length (Co-Pog)	82.9	105.0	8.0	-2.8	**
Harvold (CoPog)-(CoANS)	14.5	20.0	3.0	-1.8	*
Maxillary Skeletal (A-Na Perp) (mm)	-4.5	2.5	3.1	-2.3	**
Mand. Skeletal (Pg-Na Perp) (mm)	-14.0	-4.0	5.3	-1.9	*
MP - SN (∫)	39.3	33.0	6.0	1.0	*
U1 - Palatal Plane (∫)	94.0	110.0	5.0	-3.2	***
U1 - SN (∫)	86.7	103.1	5.5	-3.0	***
U1 - ANS (mm)	27.2	30.0	5.0	-0.6	





http://tonsilcure.com/wp-content/uploads/2014/07/tonsil_grading-730x510.jpg



Pediatric Sleep Questionnaire

(Screening)

Name of the child:

Person completing

Date that you are

Instructions: Please answer the questions about how your child **IN THE PAST MONTH.** Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

Please answer the following questions as they pertain to your child in the past month.

		YES	NO	Don't Know
1.	**While sleeping, does your child:**			
	Snore more than half the time?	(Y)	N	DK
	Always snore?	(Y)	N	DK
	Snore loudly?	(Y)	N	DK
	Have "heavy" or loud breathing?	(Y)	(N)	DK
	Have trouble breathing, or struggle to breath?	Y	(N)	DK
2.	**Have you ever seen your child stop breathing during the night?**	Y	(N)	DK
3.	**Does your child:**			
	Tend to breathe through the mouth during the day?	Y	(N)	DK
	Have a dry mouth on waking up in the morning?	(Y)	N	DK
	Occasionally wet the bed?	Y	(N)	DK
4.	**Does your child:**			
	Wake up feeling unrefreshed in the morning?	Y	(N)	DK
	Have a problem with sleepiness during the day?	Y	(N)	DK
5.	**Has a teacher or other supervisor commented that your child appears sleepy during the day?**	Y	(N)	DK
6.	**Is it hard to wake your child up in the morning?**	Y	(N)	DK
7.	**Does your child wake up with headaches in the morning?**	Y	(N)	DK
8.	**Did your child stop growing at a normal rate at any time since birth?**	Y	(N)	DK
9.	**Is your child overweight?**	Y	(N)	DK
10.	**This child often:**			
	Does not seem to listen when spoken to directly	Y	(N)	DK
	Has difficulty organizing tasks and activities	Y	(N)	DK
	Is easily distracted by extraneous stimuli	Y	(N)	DK
	Fidgets with hands or feet, or squirms in seat	Y	(N)	DK
	Is "on the go" or often acts as if "driven by a motor"	Y	(N)	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK

Paediatric Sleep Disorder Breathing

ENT referral

Adenotonsillectomy completed July 14

Significant decrease of PSDB signs/symptoms

What is next?

Pediatric Sleep Questionnaire

(Screening)

Name of the child: [redacted]

Person completing [redacted]

Date that you are completing the questionnaire: April 29th 2014

Instructions: Please answer the questions about how your child **IN THE PAST MONTH.** Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

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	Is "on the go" or often acts as if "driven by a motor"	Y	(N)	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK

Pediatric Sleep Questionnaire

(Screening)

Name of the child: [redacted]

Person completing [redacted]

Date that you are completing the questionnaire: Feb 10th 2015

Instructions: Please answer the questions about how your child **IN THE PAST MONTH**. Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

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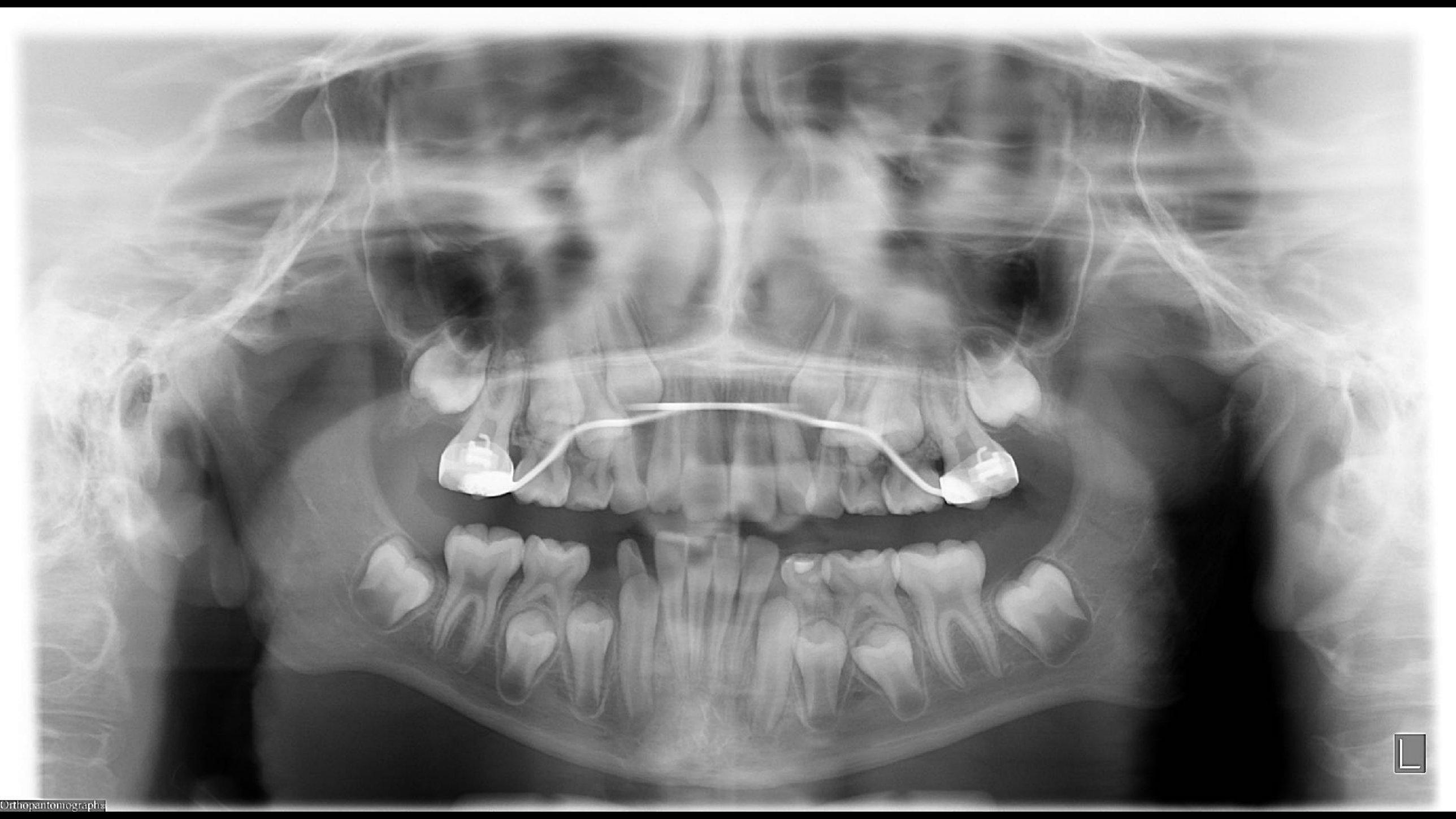
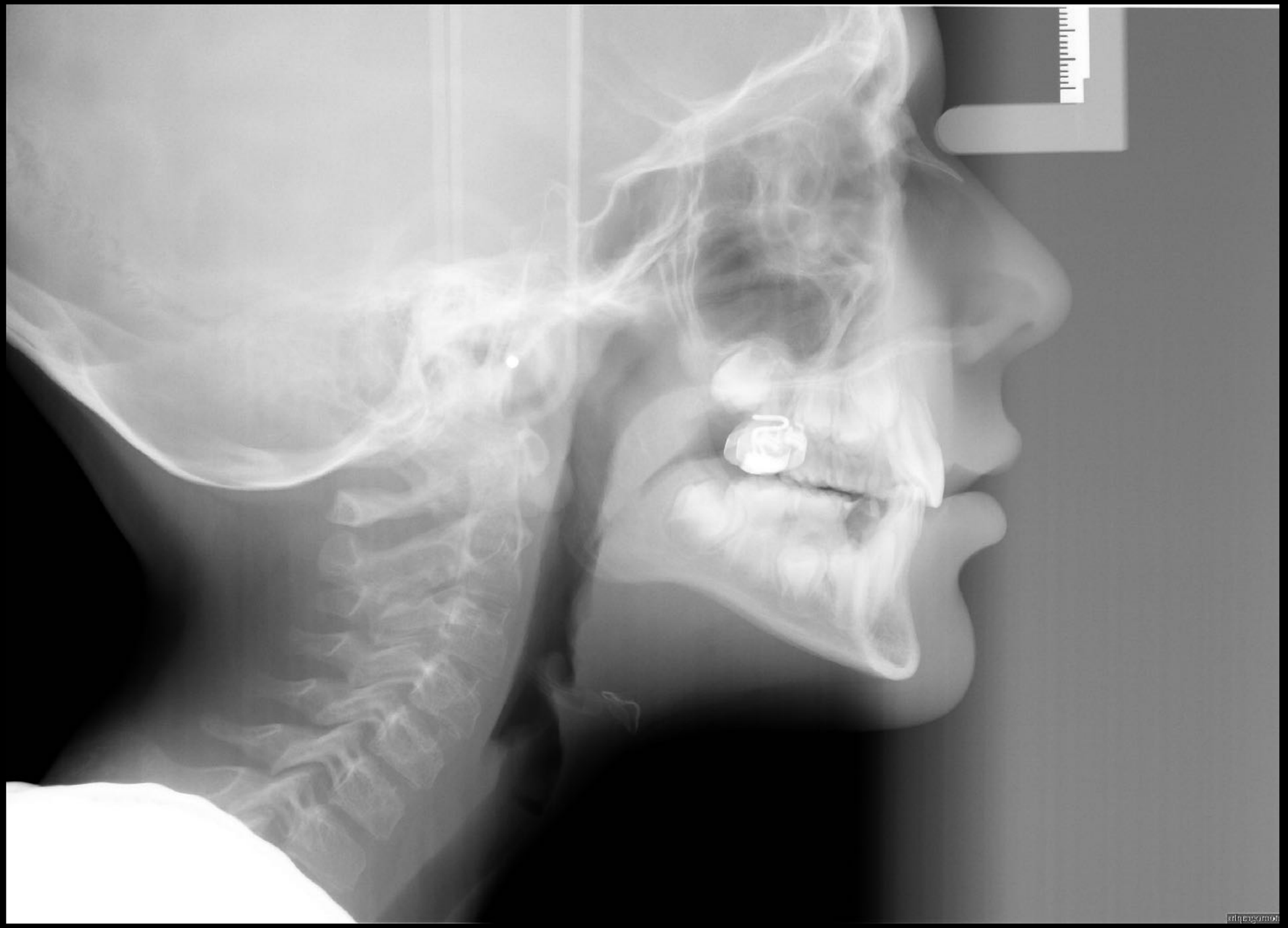
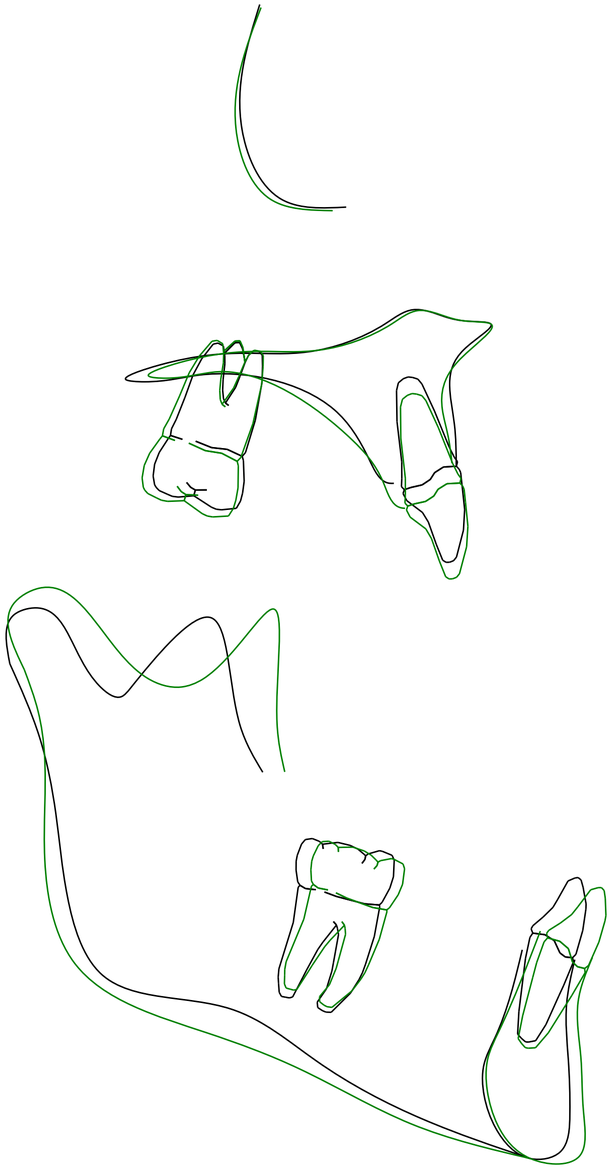
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK


L
Orthopantomograph





Pediatric Sleep Questionnaire

(Screening)

Name of the child: [redacted]

Person completing

Date that you are completing the questionnaire: April 29th 2014

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	Is "on the go" or often acts as if "driven by a motor"	Y	(N)	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK

Pediatric Sleep Questionnaire

(Screening)

Name of the child: [redacted]

Person completing

Date that you are completing the questionnaire: Feb 10th 2015

Instructions: Please answer the questions about how your child **IN THE PAST MONTH**. Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

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	Is "on the go" or often acts as if "driven by a motor"	Y	(N)	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK

Pediatric Sleep Questionnaire

(Screening)

Name of the child: [redacted]

Person completing

Date that you are completing the questionnaire: 12/7/17

Instructions: Please answer the questions about how your child **IN THE PAST MONTH**. Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

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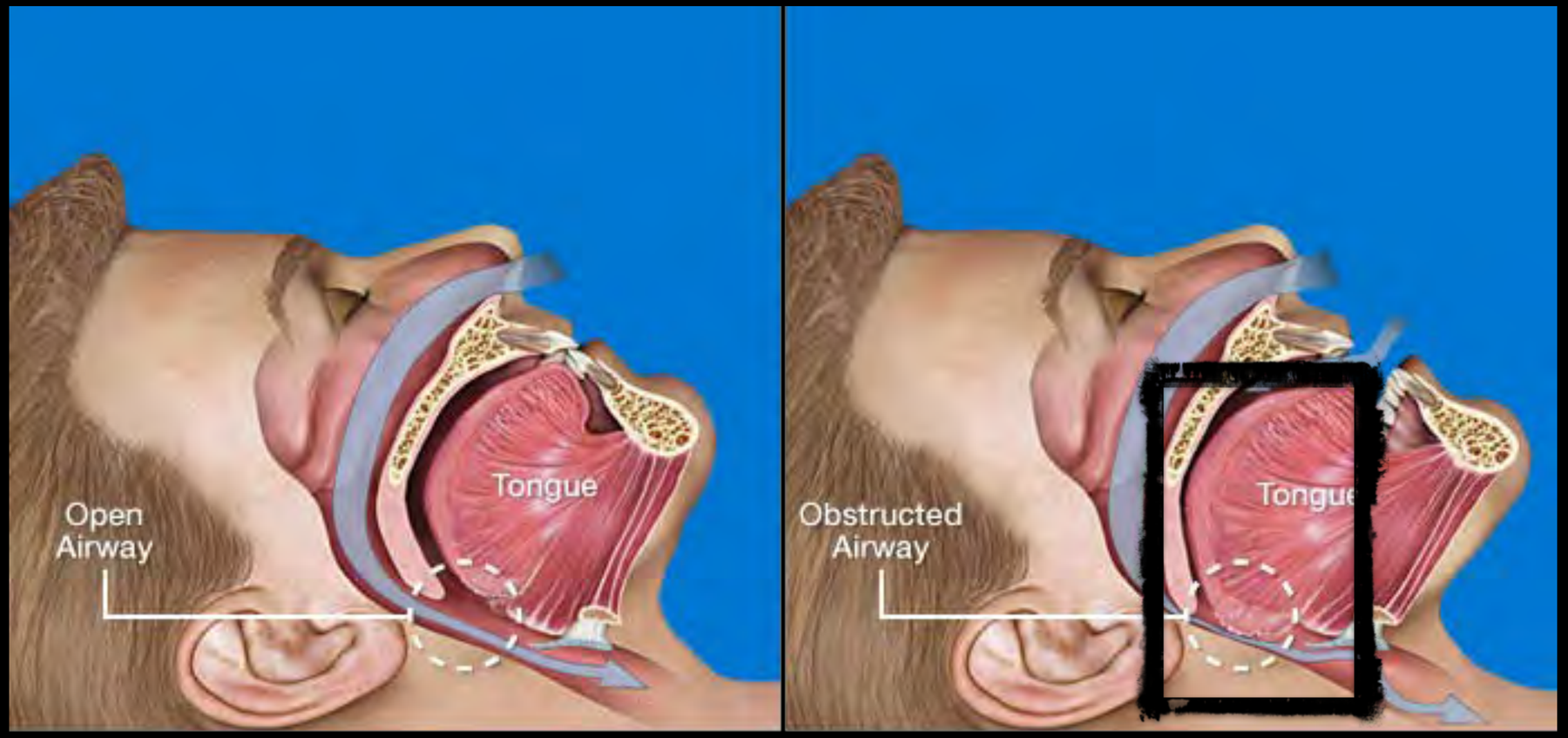
1.	**While sleeping, does your child:**			
	Snore more than half the time?	(Y)	N	DK
	Always snore?	Y	(N)	DK
	Snore loudly?	(Y)	N	DK
	Have "heavy" or loud breathing?	(Y)	N	DK
	Have trouble breathing, or struggle to breath?	Y	(N)	DK
2.	**Have you ever seen your child stop breathing during the night?**	Y	(N)	DK
3.	**Does your child:**			
	Tend to breathe through the mouth during the day?	(Y)	N	DK
	Have a dry mouth on waking up in the morning?	(Y)	N	DK
	Occasionally wet the bed?	Y	(N)	DK
4.	**Does your child:**			
	Wake up feeling unrefreshed in the morning?	Y	(N)	DK
	Have a problem with sleepiness during the day?	Y	(N)	DK
5.	**Has a teacher or other supervisor commented that your child appears sleepy during the day?**	Y	(N)	DK
6.	**Is it hard to wake your child up in the morning?**	Y	(N)	DK
7.	**Does your child wake up with headaches in the morning?**	(Y)	N	DK
8.	**Did your child stop growing at a normal rate at any time since birth?**	Y	(N)	DK
9.	**Is your child overweight?**	Y	(N)	DK
10.	**This child often:**			
	Does not seem to listen when spoken to directly	Y	(N)	DK
	Has difficulty organizing tasks and activities	Y	(N)	DK
	Is easily distracted by extraneous stimuli	Y	(N)	DK
	Fidgets with hands or feet, or squirms in seat	Y	(N)	DK
	Is "on the go" or often acts as if "driven by a motor"	Y	(N)	(DK)
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	(N)	DK

Obstructive Sleep Apnea
ORIGIN



Non-Obstructed Airway

Obstructed Airway

Sleep Disorder Breathing

Obstruction location:

Nasal area (Nasopharynx)

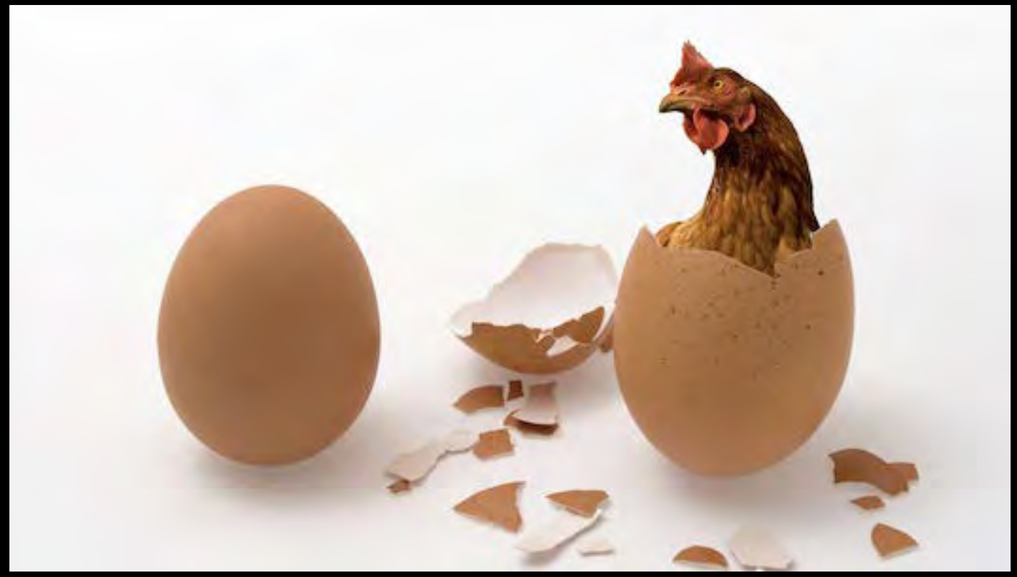
Hard/Soft palate/Uvula (Velopharynx)*

Base/back Tongue (Oropharynx)*

Lymphoid tissues sidewalls (*)

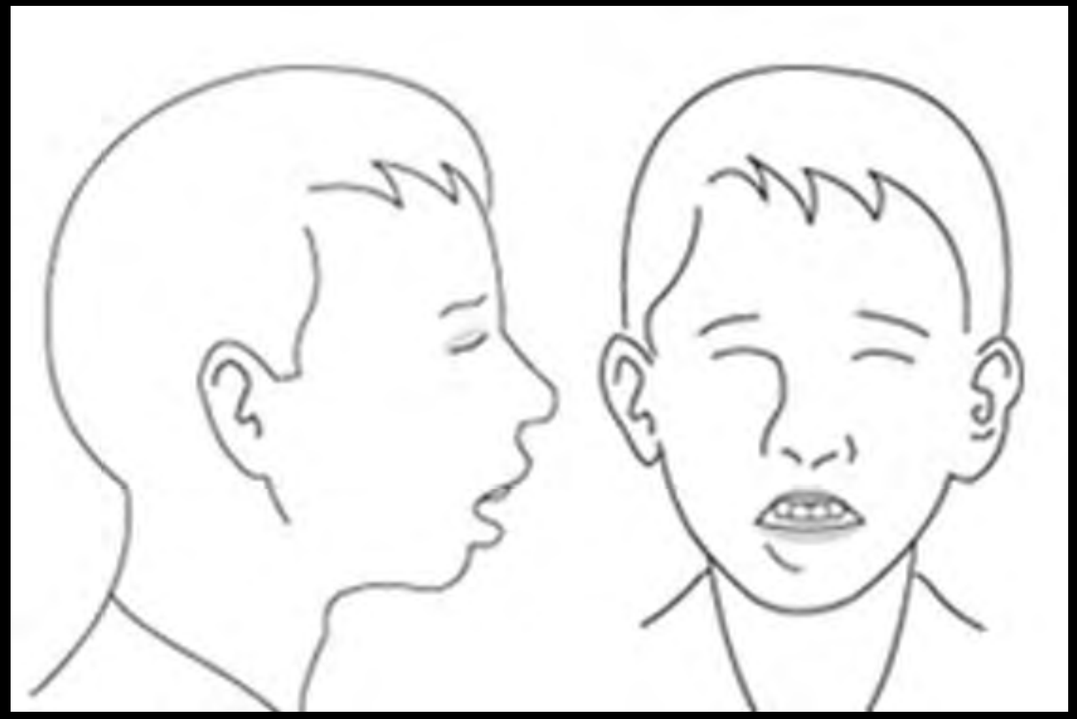
PSDB & Craniofacial Development

Cause or effect? Children with POSA frequently have abnormalities in craniofacial morphology.



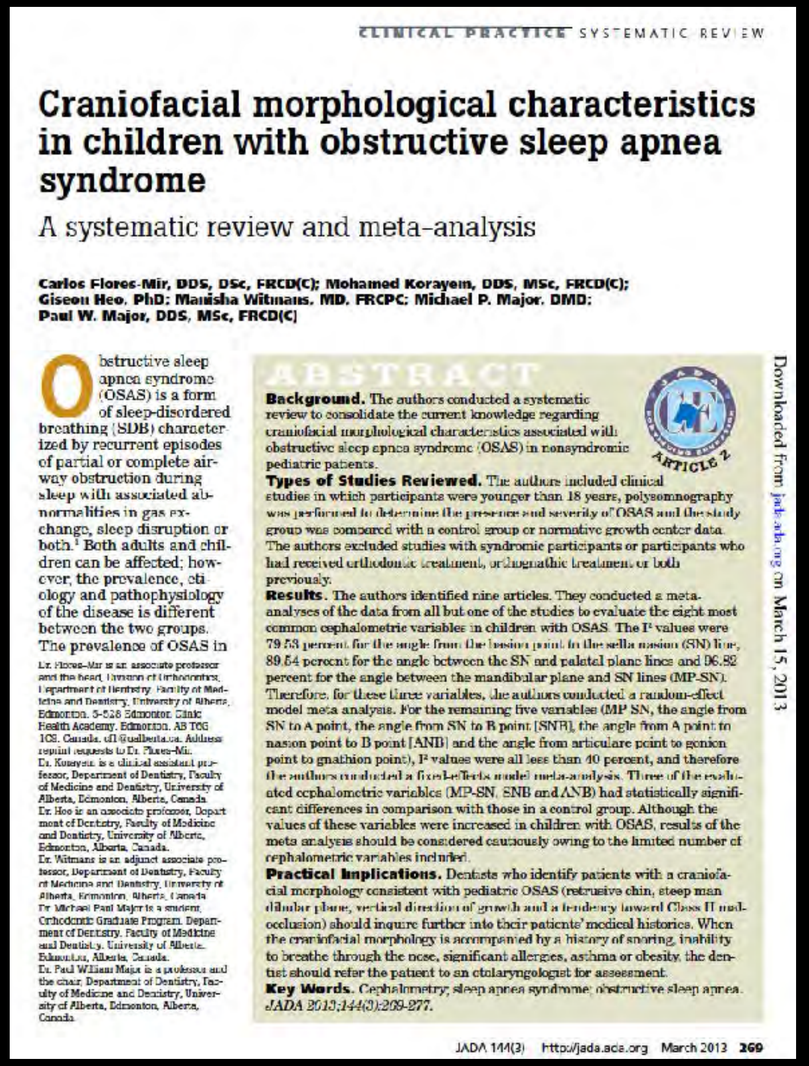
PSDB & Craniofacial Development

Common observations: steep mandibular plane, retrusive chin, inferior hyoid bone, transversely constricted maxilla.



PSDB Screening

Flores-Mir C, Korayem M, Heo G, Witmans M, Major MP, Major PW. Craniofacial Morphological Characteristics in Children with Obstructive Sleep Apnea Syndrome: A Systematic Review and Meta-Analysis. J Am Dent Assoc 2013; 144(3): 269-77



CLINICAL PRACTICE SYSTEMATIC REVIEW

Craniofacial morphological characteristics in children with obstructive sleep apnea syndrome

A systematic review and meta-analysis

Carlos Flores-Mir, DDS, DSc, FRCD(C); Mohamed Korayem, DDS, MSc, FRCD(C); Giseon Heo, PhD; Manisha Witmans, MD, FRCPC; Michael P. Major, DMD; Paul W. Major, DDS, MSc, FRCD(C)

Obstructive sleep apnea syndrome (OSAS) is a form of sleep-disordered breathing (SDB) characterized by recurrent episodes of partial or complete airway obstruction during sleep with associated abnormalities in gas exchange, sleep disruption or both.[1] Both adults and children can be affected; however, the prevalence, etiology and pathophysiology of the disease is different between the two groups. The prevalence of OSAS in

Dr. Flores-Mir is an associate professor and the head, Division of Orthodontics, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton. 5-528 Edmonton Clinic Health Academy, Edmonton, AB T6G 1C9, Canada. cfl@ualberta.ca. Address reprint requests to Dr. Flores-Mir.
Dr. Korayem is a clinical assistant professor, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.
Dr. Heo is an associate professor, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.
Dr. Witmans is an adjunct associate professor, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.
Dr. Michael Paul Major is a student, Orthodontic Graduate Program, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.
Dr. Paul William Major is a professor and the chair, Department of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.

ABSTRACT

Background. The authors conducted a systematic review to consolidate the current knowledge regarding craniofacial morphological characteristics associated with obstructive sleep apnea syndrome (OSAS) in nonsyndromic pediatric patients.

Types of Studies Reviewed. The authors included clinical studies in which participants were younger than 18 years, polysomnography was performed to determine the presence and severity of OSAS and the study group was compared with a control group or normative growth center data. The authors excluded studies with syndromic participants or participants who had received orthodontic treatment, orthognathic treatment or both previously.

Results. The authors identified nine articles. They conducted a meta-analyses of the data from all but one of the studies to evaluate the eight most common cephalometric variables in children with OSAS. The I^2 values were 79.53 percent for the angle from the lesion point to the sella-nasion (SN) line, 89.54 percent for the angle between the SN and palatal plane lines and 96.82 percent for the angle between the mandibular plane and SN lines (MP-SN). Therefore, for these three variables, the authors conducted a random-effect model meta-analysis. For the remaining five variables (MP-SN, the angle from SN to A point, the angle from SN to B point [SNB], the angle from A point to nasion point to B point [ANB] and the angle from articulare point to gonion point to gnathion point), I^2 values were all less than 40 percent, and therefore the authors conducted a fixed-effects model meta-analysis. Three of the evaluated cephalometric variables (MP-SN, SNB and ANB) had statistically significant differences in comparison with those in a control group. Although the values of these variables were increased in children with OSAS, results of the meta-analysis should be considered cautiously owing to the limited number of cephalometric variables included.

Practical Implications. Dentists who identify patients with a craniofacial morphology consistent with pediatric OSAS (retrusive chin, steep mandibular plane, vertical direction of growth and a tendency toward Class II malocclusion) should inquire further into their patients' medical histories. When the craniofacial morphology is accompanied by a history of snoring, inability to breathe through the nose, significant allergies, asthma or obesity, the dentist should refer the patient to an otolaryngologist for assessment.

Key Words. Cephalometry; sleep apnea syndrome; obstructive sleep apnea.
JADA 2013;144(3):269-277.

TABLE 3

Summary of meta-analysis for the eight most frequently reported variables in the selected articles.*

VARIABLE†	NO. OF STUDIES	MEAN DIFFERENCE, DEGREES (STANDARD ERROR)	*P* VALUE	95% CONFIDENCE INTERVAL
MP-SN	5	4.20 (0.45)	< .001	3.32 to 5.07
SNA	4	−0.32 (0.40)	.79	−1.10 to 0.46
SNB	4	−1.79 (0.42)	< .001	−2.61 to −0.97
ANB	4	1.38 (0.28)	< .001	0.83 to 1.92
BaSN	3	−1.12 (1.50)	.23	−4.06 to 1.82
ArGoGn	2	0.53 (0.89)	.28	−1.21 to 2.27
SN-PP	3	−0.77 (1.14)	.25	−3.01 to 1.47
MP-PP	2	7.12 (4.45)	.06	−1.60 to 15.84

* All of the measurements are angular.
† All of the variables are defined in Table 2.

Common reported findings across studies.

- Narrow maxillary dental arch with high palatal vault and posterior crossbites
- Longer lower anterior face height
- Steeper (more obtuse) gonial angle (vertical growth pattern)
- Posterior-inferior (clockwise) rotation of the mandible (mandibular plane angle)
- Retrusive chin
- Tendency toward anterior open bite and lip incompetence
- Smaller nasopharyngeal airway spaces

PSDB Screening

Korayem M, Witmans M, MacLean J, Heo G, El-Hakim H, Flores-Mir C, Major PW. Craniofacial Morphology in Pediatric Patients with Persistent Obstructive Sleep Apnea with or without Positive Airway Pressure Therapy: A Cross-Sectional Cephalometric Comparison with Controls. Am J Orthod Dentofac Orthoped 2013; 144(1): 78-85.

ORIGINAL ARTICLE AJO-DO

Craniofacial morphology in pediatric patients with persistent obstructive sleep apnea with or without positive airway pressure therapy: A cross-sectional cephalometric comparison with controls

Mohammed M. Korayem,[a] Manisha Witmans,[b] Joanna MacLean,[c] Giseon Heo,[d] Hamdy El-Hakim,[e] Carlos Flores-Mir,[f] and Paul W. Major[g]
Edmonton, Alberta, Canada

Introduction: Compression on the midface with nasal mask-delivered positive airway pressure (PAP) therapy in growing patients might contribute to midface retrusion. The objective of this study was to investigate the association between long-term PAP use and craniofacial morphologic pattern in children with persistent obstructive sleep apnea. **Methods:** Images generated with cone-beam volumetric imaging were used to complete lateral cephalometric analyses of anteroposterior projection of the midface region. The study group included 12 subjects (10 boys, 2 girls; mean age, 9.0 years) who used PAP therapy for at least 6 months and at least 6 hours per night. Measurements from this group were compared with those of a control group of 11 subjects (5 boys, 6 girls; mean age, 9.6 years) with obstructive sleep apnea who did not have PAP. Measurements were taken at 1 time point. **Results:** No significant differences were identified between the groups for any cephalometric variable. Multivariate linear regression analysis also did not identify a significant association between the number of months of PAP therapy and the cephalometric variables. Cephalometric data for both groups were pooled for comparison with appropriate published normative values for age and sex. Anterior cranial base length, overall anteroposterior length of the maxillary base, and mandibular body length were significantly shorter than normal in the subjects compared with published normative values. **Conclusions:** No association was demonstrated between midface projection and PAP use in growing patients. When compared with normative data for anterior cranial base, children with obstructive sleep apnea had shorter maxillary and mandibular lengths. (Am J Orthod Dentofacial Orthop 2013;144:78-85)

Obstructive sleep apnea (OSA) is a form of sleep-disordered breathing characterized by recurrent episodes of partial or complete airway obstruction during sleep. The reported prevalence of OSA in children varies by age range, method of assessment, and definition of OSA. Recent studies using polysomnography in elementary school children have reported prevalence rates of 1.0% to 2.2%.[1-4] The pathophysiology of OSA in any patient depends on the specific primary etiologic factors, but the overarching

From the University of Alberta, Edmonton, Alberta, Canada.
[a]Clinical instructor, Division of Orthodontics, School of Dentistry, Faculty of Medicine and Dentistry.
[b]Associate professor, Department of Pediatrics, Faculty of Medicine and Dentistry; Northern Alberta Pediatric Sleep Laboratory, Stollery Children's Hospital, Edmonton, Alberta, Canada.
[c]Assistant professor, Division of Respiratory Medicine, Department of Pediatrics, Faculty of Medicine and Dentistry; Northern Alberta Pediatric Sleep Laboratory, Stollery Children's Hospital, Edmonton, Alberta, Canada.
[d]Associate professor, Division of Orthodontics, School of Dentistry, Faculty of Medicine and Dentistry, and Department of Mathematical and Statistical Sciences, Faculty of Science.
[e]Associate professor, Divisions of Pediatric Surgery and Otolaryngology Head and Neck Surgery, Department of Surgery, Stollery Children's Hospital; University of Alberta Hospitals.
[f]Associate professor and head, Division of Orthodontics, School of Dentistry, Faculty of Medicine and Dentistry.
[g]Lead, School of Dentistry; professor and chair, Department of Dentistry, Faculty of Medicine and Dentistry.
All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.
Reprint requests to: Paul W. Major, Department of Dentistry, Faculty of Medicine and Dentistry, 5-478, Edmonton Clinic Health Academy (ECHA), University of Alberta, 11405-87 Ave, Edmonton, Alberta, Canada T6G 1C9; e-mail, major@ualberta.ca.
Submitted, September 2012; revised and accepted, February 2013.
0889-5406/$36.00

http://dx.doi.org/10.1016/j.ajodo.2013.02.027

Table V. Comparisons to normative data

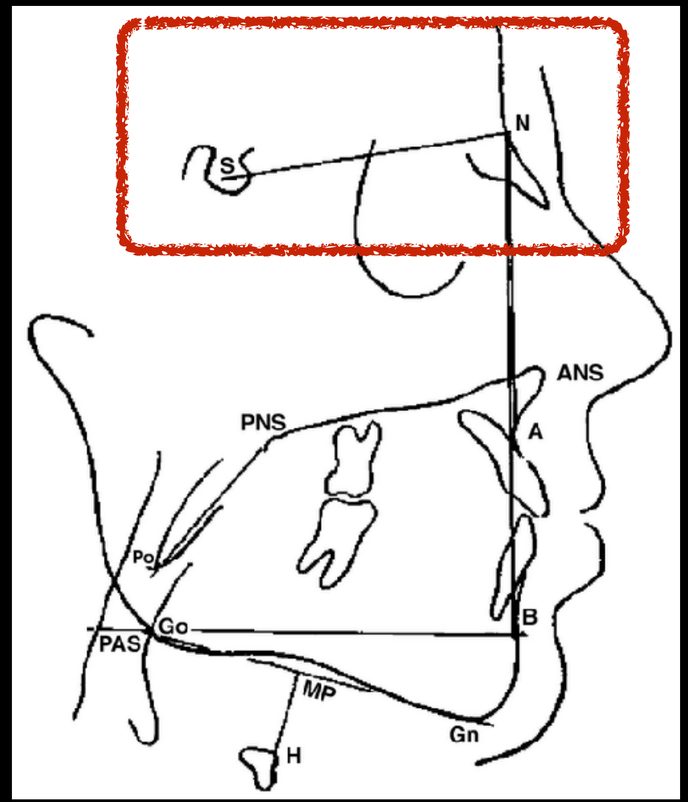
	PAP		*Control*		*Norm*			
	Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*	P *value PAP vs norm*	P *value control vs norm*
SN (mm)	61.5	3.4	64.0	3.7	70.8*	2.9	<0.001	<0.001
BaSN (°)	131.5	8.4	131.3	4.0	134.9†	5.4	0.084	0.077
SNA (°)	81.5	3.0	80.5	3.8	80.1†	3.7	0.239	0.735
PP-SN (mm)	7.2	4.1	6.2	3.7	6.7*	2.8	0.637	0.648
CoANS (mm)	78.4	5.8	79.7	5.0	88.5*	4.0	<0.001	<0.001
ANS-PNS (mm)	44.9	4.5	45.6	3.0	48.1*	2.1	0.001	0.011
U1-PP (°)	105.1	9.3	106.4	9.1	109.0†	5.4	0.093	0.275
ANperp (mm)	2.7	2.1	0.6	4.8	−3.3†	3.1	<0.001	0.001
OLp-A (mm)	66.8	5.1	68.6	3.9	79.0‡	3.7	<0.001	<0.001
SNB (°)	79.3	3.7	77.2	4.7	77.5†	3.1	<0.001	0.784
ArGoMe (°)	123.8	8.9	129.5	8.4	123.4†	5.3	0.843	0.009
GoMe (mm)	63.3	8.0	61.2	7.7	73.0*	3.8	<0.001	<0.001
ANB (°)	1.8	2.2	2.6	3.2	2.6†	2.1	0.335	0.950
Wits (mm)	−2.2	3.9	−1.5	2.7	−1.0†	2.1	0.159	0.551

*Thilander et al[51] (2005), sample of 42 white Swedish children; average age, 9.5-11.2 years; †Obloj et al[52] (2008), sample of 34 white Polish children; average age, 10.4 years; ‡Wu et al[50] (2010), sample of 86 white British children; average age, 12.4 years.

PAP and Control samples from patients that were unresponsive to A&T

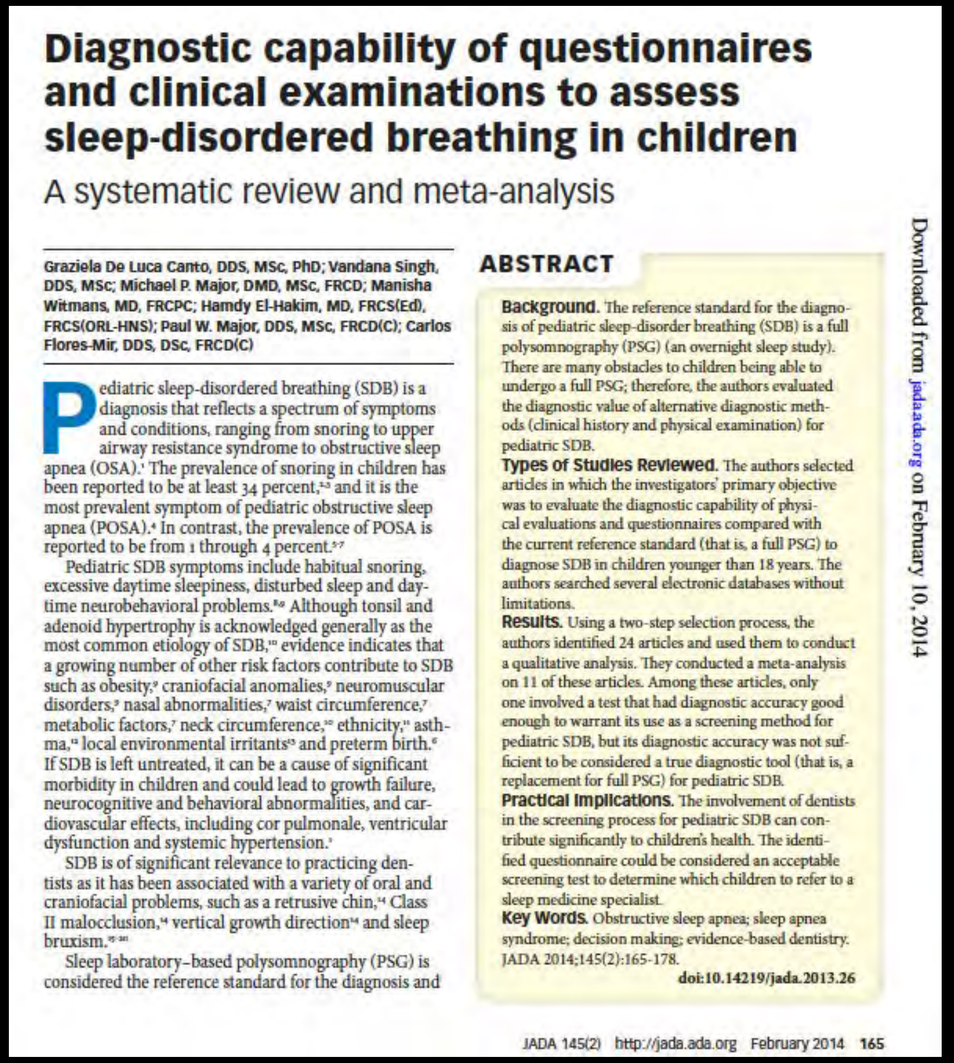
PSDB Screening

Short SN: Role of craniofacial growth in the cracial base - genetic influence on OSA?



PSDB Screening

de Luca Canto G, Singh V, Major MP, Witmans M, El-Hakim H, Major PW, Flores-Mir C. Diagnostic Capability of Questionnaires and Clinical Examinations to Assess Sleep-disordered breathing in Children: A Systematic Review and Meta-Analysis. J Am Dent Assoc 2014; 145(2): 165-78.



Diagnostic capability of questionnaires and clinical examinations to assess sleep-disordered breathing in children

A systematic review and meta-analysis

Graziela De Luca Canto, DDS, MSc, PhD; Vandana Singh, DDS, MSc; Michael P. Major, DMD, MSc, FRCD; Manisha Witmans, MD, FRCPC; Hamdy El-Hakim, MD, FRCS(Ed), FRCS(ORL-HNS); Paul W. Major, DDS, MSc, FRCD(C); Carlos Flores-Mir, DDS, DSc, FRCD(C)

Pediatric sleep-disordered breathing (SDB) is a diagnosis that reflects a spectrum of symptoms and conditions, ranging from snoring to upper airway resistance syndrome to obstructive sleep apnea (OSA).[1] The prevalence of snoring in children has been reported to be at least 34 percent,[2,3] and it is the most prevalent symptom of pediatric obstructive sleep apnea (POSA).[4] In contrast, the prevalence of POSA is reported to be from 1 through 4 percent.[5-7]

Pediatric SDB symptoms include habitual snoring, excessive daytime sleepiness, disturbed sleep and daytime neurobehavioral problems.[8,9] Although tonsil and adenoid hypertrophy is acknowledged generally as the most common etiology of SDB,[10] evidence indicates that a growing number of other risk factors contribute to SDB such as obesity,[9] craniofacial anomalies,[9] neuromuscular disorders,[9] nasal abnormalities,[7] waist circumference,[7] metabolic factors,[7] neck circumference,[10] ethnicity,[11] asthma,[12] local environmental irritants[13] and preterm birth.[6] If SDB is left untreated, it can be a cause of significant morbidity in children and could lead to growth failure, neurocognitive and behavioral abnormalities, and cardiovascular effects, including cor pulmonale, ventricular dysfunction and systemic hypertension.[1]

SDB is of significant relevance to practicing dentists as it has been associated with a variety of oral and craniofacial problems, such as a retrusive chin,[14] Class II malocclusion,[14] vertical growth direction[14] and sleep bruxism.[15-20]

Sleep laboratory–based polysomnography (PSG) is considered the reference standard for the diagnosis and

ABSTRACT

Background. The reference standard for the diagnosis of pediatric sleep-disorder breathing (SDB) is a full polysomnography (PSG) (an overnight sleep study). There are many obstacles to children being able to undergo a full PSG; therefore, the authors evaluated the diagnostic value of alternative diagnostic methods (clinical history and physical examination) for pediatric SDB.

Types of Studies Reviewed. The authors selected articles in which the investigators' primary objective was to evaluate the diagnostic capability of physical evaluations and questionnaires compared with the current reference standard (that is, a full PSG) to diagnose SDB in children younger than 18 years. The authors searched several electronic databases without limitations.

Results. Using a two-step selection process, the authors identified 24 articles and used them to conduct a qualitative analysis. They conducted a meta-analysis on 11 of these articles. Among these articles, only one involved a test that had diagnostic accuracy good enough to warrant its use as a screening method for pediatric SDB, but its diagnostic accuracy was not sufficient to be considered a true diagnostic tool (that is, a replacement for full PSG) for pediatric SDB.

Practical Implications. The involvement of dentists in the screening process for pediatric SDB can contribute significantly to children's health. The identified questionnaire could be considered an acceptable screening test to determine which children to refer to a sleep medicine specialist.

Key Words. Obstructive sleep apnea; sleep apnea syndrome; decision making; evidence-based dentistry.
JADA 2014;145(2):165-178.
doi:10.14219/jada.2013.26

POSA Screening

A. Questionnaire

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Yang and Colleagues,[38] 2010	19	16	9	19	0.68 (0.48-0.84)	0.54 (0.37-0.71)
Chan and Colleagues,[40] 2012	14	33	14	41	0.50 (0.31-0.69)	0.55 (0.43-0.67)
Chervin and Colleagues,[21] 2000	56	2	13	10	0.81 (0.70-0.90)	0.83 (0.52-0.98)

B. Questionnaire and Physical Examination

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Goldstein and Colleagues,[1] 2012	51	11	15	17	0.77 (0.65-0.87)	0.61 (0.41-0.78)
Li and Colleagues,[48] 2006	50	32	16	131	0.76 (0.64-0.85)	0.80 (0.73-0.86)
Sproson and Colleagues,[52] 2009	26	26	12	14	0.68 (0.51-0.82)	0.61 (0.39-0.80)

C. Questionnaire and Physical Examination and Other Tests

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Chervin and Colleagues,[51] 2007	32	18	9	46	0.78 (0.62-0.89)	0.72 (0.59-0.82)
Goldstein and Colleagues,[50] 1994	12	12	1	5	0.92 (0.64-1.00)	0.29 (0.10-0.56)
Lamm and Colleagues,[24] 1999	5	2	6	10	0.45 (0.17-0.77)	0.83 (0.52-0.98)
Yang and Colleagues,[43] 2012	229	34	135	129	0.63 (0.58-0.68)	0.79 (0.72-0.85)

D. Physical Examination and Other Tests

Study	TP	FP	FN	TN	Sensitivity (95% CI)	Specificity (95% CI)
Supriyatno and Colleagues,[10] 2010	26	0	16	68	0.62 (0.46-0.76)	1.00 (0.95-1.00)

Figure 2. Forest plot with the diagnostic accuracy (sensitivity, specificity and 95 percent confidence interval) of each study. TP: True positive. FP: False positive. FN: False negative. TN: True negative. CI: Confidence interval.

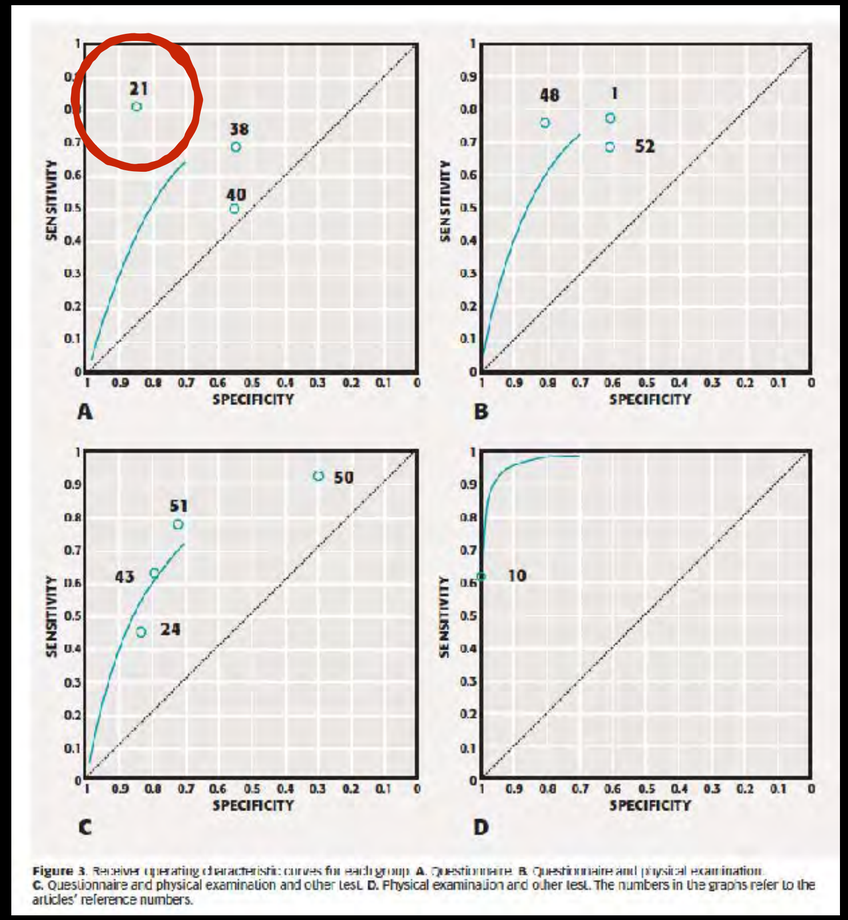


Figure 3. Receiver operating characteristic curves for each group. **A.** Questionnaire. **B.** Questionnaire and physical examination. **C.** Questionnaire and physical examination and other test. **D.** Physical examination and other test. The numbers in the graphs refer to the articles' reference numbers.

PSDB Screening

Pediatric Sleep Questionnaire

(Screening)

Name of the child:_____ Date of birth:_____

Person completing this form: _____

Date that you are completing the questionnaire: _____

Instructions: Please answer the questions about how your child **IN THE PAST MONTH**. Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

Please answer the following questions as they pertain to your child in the past month.

		YES	NO	Don't Know
1.	**While sleeping, does your child:**			
	Snore more than half the time?	Y	N	DK
	Always snore?	Y	N	DK
	Snore loudly?	Y	N	DK
	Have "heavy" or loud breathing?	Y	N	DK
	Have trouble breathing, or struggle to breath?	Y	N	DK
2.	**Have you ever seen your child stop breathing during the night?**	Y	N	DK
3.	**Does your child:**			
	Tend to breathe through the mouth during the day?	Y	N	DK
	Have a dry mouth on waking up in the morning?	Y	N	DK
	Occasionally wet the bed?	Y	N	DK
4.	**Does your child:**			
	Wake up feeling unrefreshed in the morning?	Y	N	DK
	Have a problem with sleepiness during the day?	Y	N	DK
5.	**Has a teacher or other supervisor commented that your child appears sleepy during the day?**	Y	N	DK
6.	**Is it hard to wake your child up in the morning?**	Y	N	DK
7.	**Does your child wake up with headaches in the morning?**	Y	N	DK
8.	**Did your child stop growing at a normal rate at any time since birth?**	Y	N	DK
9.	**Is your child overweight?**	Y	N	DK
10.	**This child often:**			
	Does not seem to listen when spoken to directly.....	Y	N	DK
	Has difficulty organizing tasks and activities.....	Y	N	DK
	Is easily distracted by extraneous stimuli	Y	N	DK
	Fidgets with hands or feet, or squirms in seat	Y	N	DK
	Is "on the go" or often acts as if "driven by a motor"	Y	N	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	N	DK

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PSDB Screening

While sleeping, does your child:

Snore more than half the time? .....

Always snore? .....

Snore loudly? .....

Have “heavy” or loud breathing? .....

Have trouble breathing, or struggle to breath? .....

PSDB Screening

Have you ever seen your child stop breathing during the night? .....

Does your child:

Tend to breathe through the mouth during the day?

.....

Have a dry mouth on waking up in the morning? .....

Occasionally wet the bed? .....

Does your child:

Wake up feeling unrefreshed in the morning? .....

Have a problem with sleepiness during the day? .....

PSDB Screening

Has a teacher or other supervisor commented that your child appears sleepy during the day? .....

Is it hard to wake your child up in the morning? .....

Does your child wake up with headaches in the morning? .....

Did your child stop growing at a normal rate at any time since birth? ....

Is your child overweight? .....

PSDB Screening

This child often:

Does not seem to listen when spoken to directly.....

Has difficulty organizing tasks and activities.....

Is easily distracted by extraneous stimuli .....

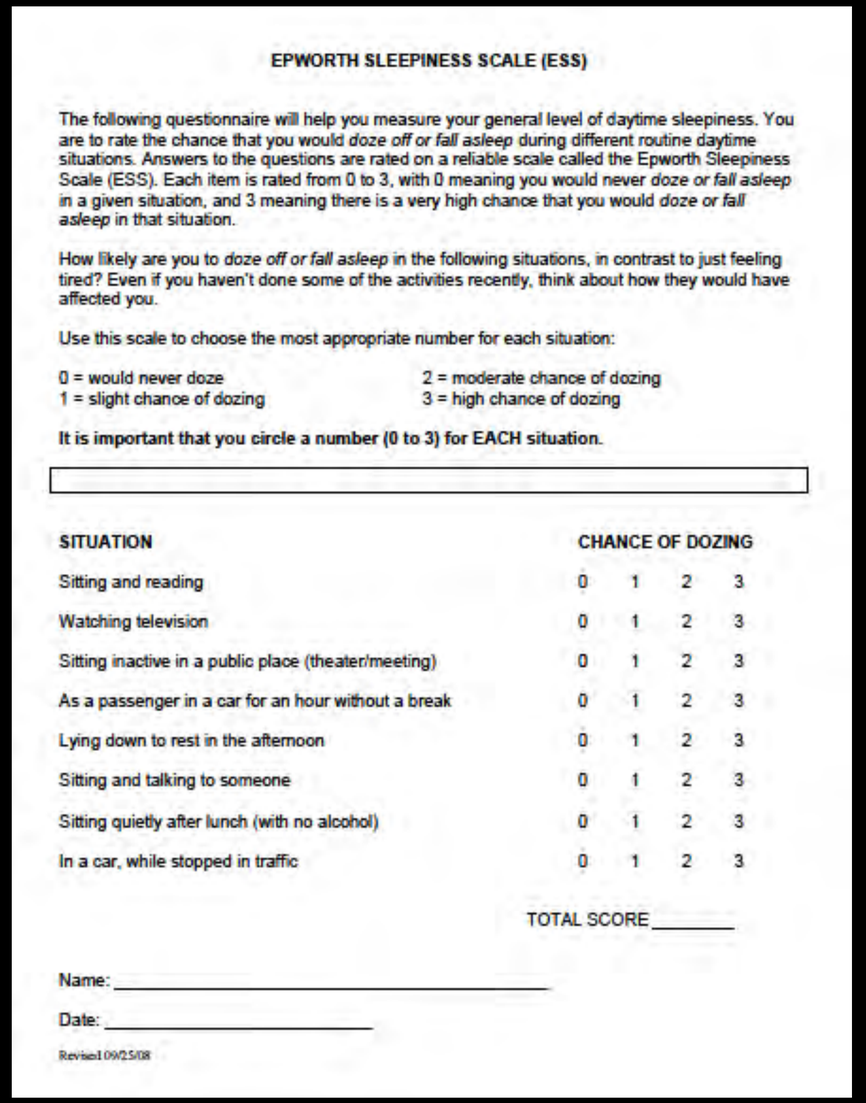
Fidgets with hands or feet, or squirms in seat .....

Is “on the go” or often acts as if “driven by a motor” .....

Interrupts or intrudes on others (eg butts into conversations or games) ..

Questionnaries

- Epworth Sleepiness Scale (ESS) - subjective assessment of pre-tx sleepiness.
- http://epworthsleepinesscale.com/about-epworth-sleepiness/



EPWORTH SLEEPINESS SCALE (ESS)

The following questionnaire will help you measure your general level of daytime sleepiness. You are to rate the chance that you would *doze off or fall asleep* during different routine daytime situations. Answers to the questions are rated on a reliable scale called the Epworth Sleepiness Scale (ESS). Each item is rated from 0 to 3, with 0 meaning you would never *doze or fall asleep* in a given situation, and 3 meaning there is a very high chance that you would *doze or fall asleep* in that situation.

How likely are you to *doze off or fall asleep* in the following situations, in contrast to just feeling tired? Even if you haven't done some of the activities recently, think about how they would have affected you.

Use this scale to choose the most appropriate number for each situation:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

It is important that you circle a number (0 to 3) for EACH situation.

SITUATION	CHANCE OF DOZING
Sitting and reading	0 1 2 3
Watching television	0 1 2 3
Sitting inactive in a public place (theater/meeting)	0 1 2 3
As a passenger in a car for an hour without a break	0 1 2 3
Lying down to rest in the afternoon	0 1 2 3
Sitting and talking to someone	0 1 2 3
Sitting quietly after lunch (with no alcohol)	0 1 2 3
In a car, while stopped in traffic	0 1 2 3

TOTAL SCORE_____

Name: _____

Date: _____

Revised 09/25/08

PSDB Screening

8 PSQ positive answers
should trigger a referral to an ENT

Dx consideration real world

- Pre-test probability
- POSA 3%

PPV = 42.9 %

For <u>every 100</u> who **test positive**

43 … *will have POSA*

57 … **False Positive !!**

$$\mathrm{PPV} = \frac{\text{sensitivity} \times \text{prevalence}}{\text{sensitivity} \times \text{prevalence} + (1 - \text{specificity}) \times (1 - \text{prevalence})}$$

NPV = 99.9 %

For every 100 who **test negative**

Almost all are POSA free

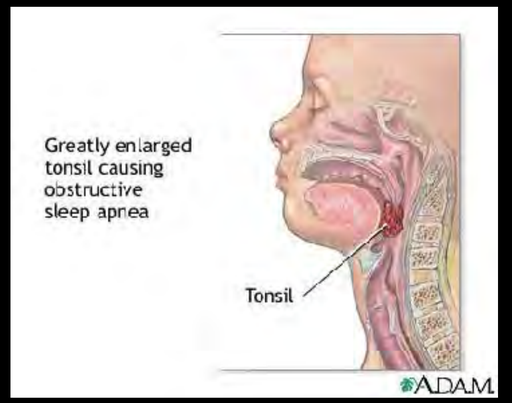
Negligible … False Negative !!

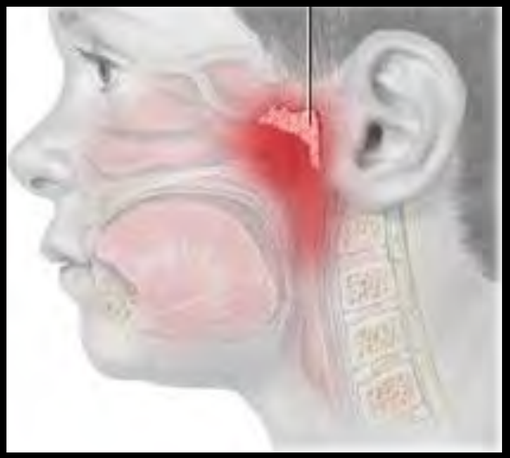
$$\mathrm{NPV} = \frac{\mathrm{specificity} \times (1 - \mathrm{prevalence})}{(1 - \mathrm{sensitivity}) \times \mathrm{prevalence} + \mathrm{specificity} \times (1 - \mathrm{prevalence})}$$

The strength of the PSQ screen test is instead in its negative predictive value – which, if negative for an individual, gives us a high confidence that its negative result is true.

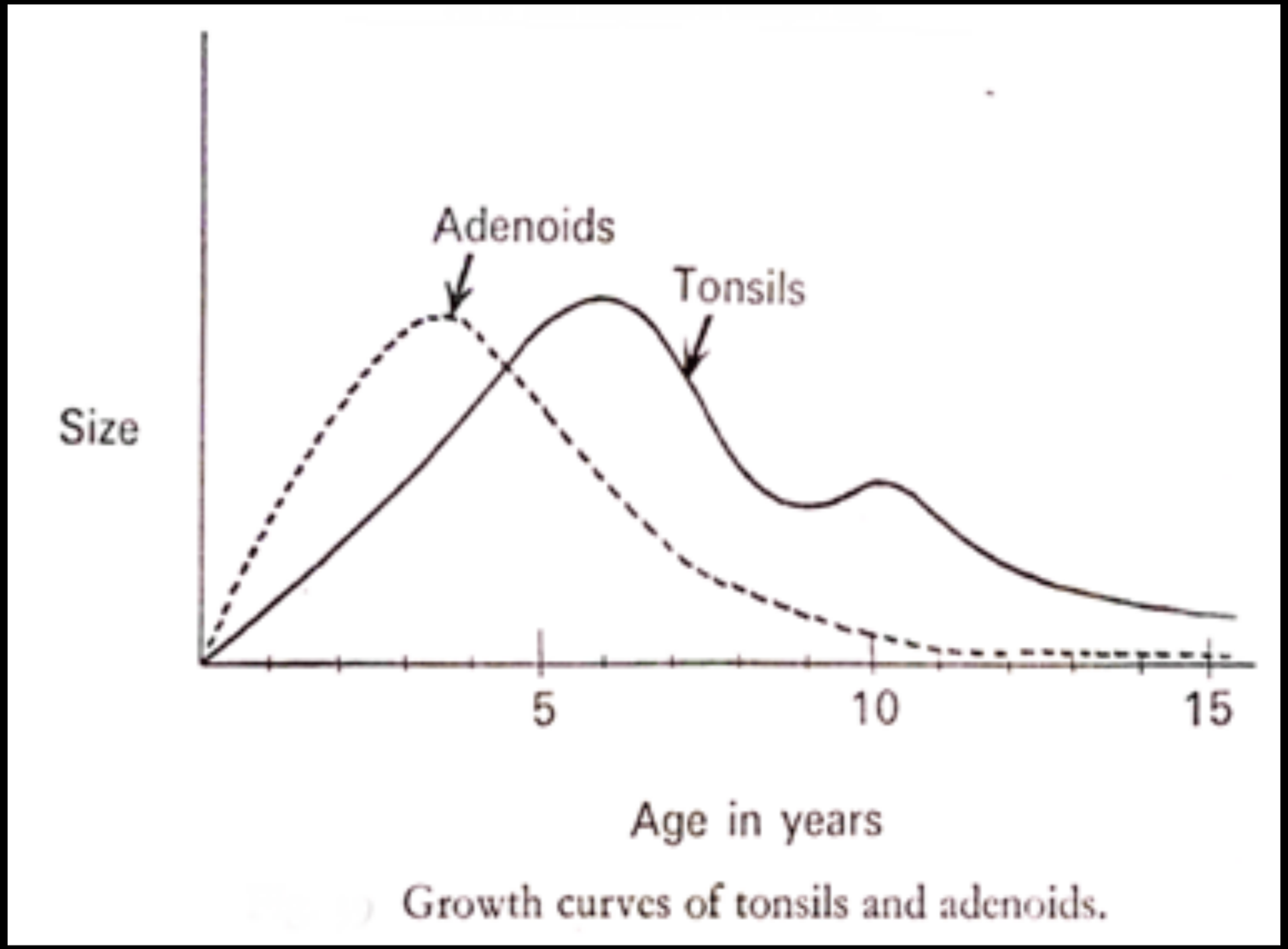
Adenoid Hypertrophy Risk Factor

- Estimated frequency is 19 to 58% from 6 months to 15 years.
- Impaired nasal airflow could result in SDB - mouth breathing
- Nasoendoscopy is the gold standard





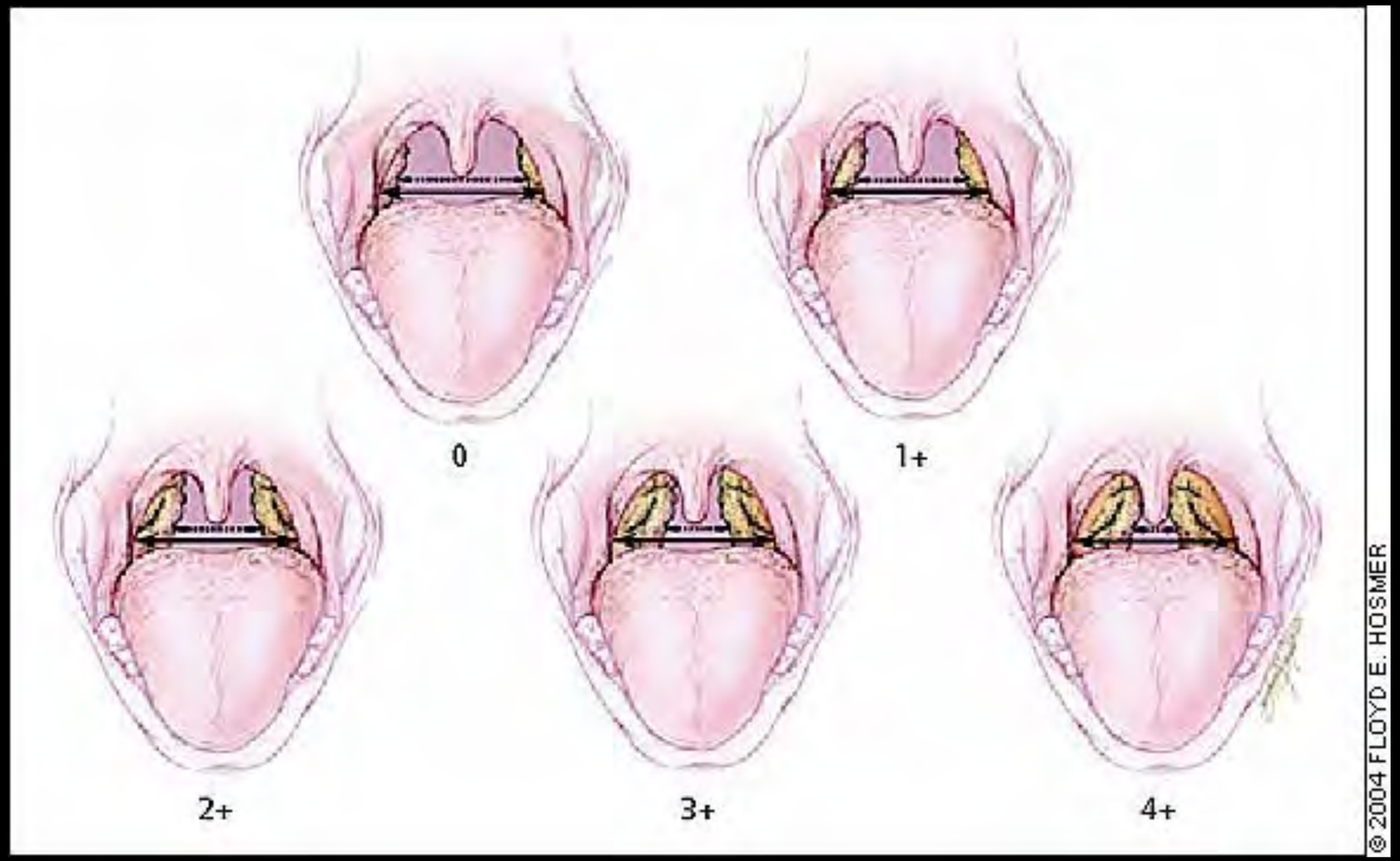
Adenoid Hypertrophy Dx



Growth curves of tonsils and adenoids.

Linder-Aronson 1970

Tonsil Hypertrophy Dx



Malparti Grading Scale

Adenoid Hypertrophy Dx

Pereira L, Monyror J, Almeida FT, Almeida FR, Guerra E, Flores-Mir C, Pacheco-Pereira C. Prevalence of adenoid hyperthrophy: A Systematic Review and meta-analysis. Sleep Med Rev 2018; 38: 101-12.

Sleep Medicine Reviews 38 (2018) 101–112

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

CLINICAL REVIEW

Prevalence of adenoid hypertrophy: A systematic review and meta-analysis

Lara Pereira [a], John Monyror [b], Fabiana T. Almeida [c, e], Fernanda R. Almeida [d], Eliete Guerra [c], Carlos Flores-Mir [e], Camila Pachêco-Pereira [c, e, *]

[a] Faculty Saint Jean, University of Alberta, Edmonton, Canada
[b] Faculty of Sciences, University of Alberta, Edmonton, Canada
[c] Health Sciences Faculty, University of Brasília, Brasília, Brazil
[d] Division of Orthodontics of Faculty of Dentistry, University of British Columbia, Vancouver, BC, Canada
[e] School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Canada

ARTICLE INFO

Article history:
Received 12 November 2016
Received in revised form
16 March 2017
Accepted 6 June 2017
Available online 14 June 2017

Keywords:
Adenoids
Adolescent
Child
Hypertrophy
Prevalence

SUMMARY

The purpose of our review was to synthesize the existing literature about the prevalence of adenoid hypertrophy (AH) in children and adolescents confirmed by the reference standard – the nasoendoscopy (NE). Six electronic databases and partial grey literature were searched. Studies were included if they reported the prevalence of AH confirmed via NE. Studies involving participants with associated comorbidities and/or fully diagnosed sleep apnea in their sample were excluded. The MASTARI tool assessed the potential risk of bias (RoB) among the studies, while the GRADE approach determined the level of evidence. A total of 5248 patients were included. Seventeen studies were included in the meta-analysis showing an AH prevalence of 49.70% (confidence interval (CI): 39.92 to 59.50). The studies were then divided into 3 groups based on the RoB assessment and patient selection method. The AH prevalence for group 1 (studies having low RoB) was 42.18% (CI: 34.93 to 49.60; n = 2794), for group 2 (studies having moderate RoB) was 70.02% (CI: 40.102 to 92.690; n = 538), and finally for group 3 (studies with randomly collected samples) was 34.46% (CI: 10.507 to 63.742; n = 1446). High heterogeneity between the studies was found. The GRADE approach classified the quality of evidence as moderate. In summary, in a randomized representative sample the prevalence of AH was 34.46%; however, in convenience samples the prevalence ranged from 42 to 70%.

Introduction

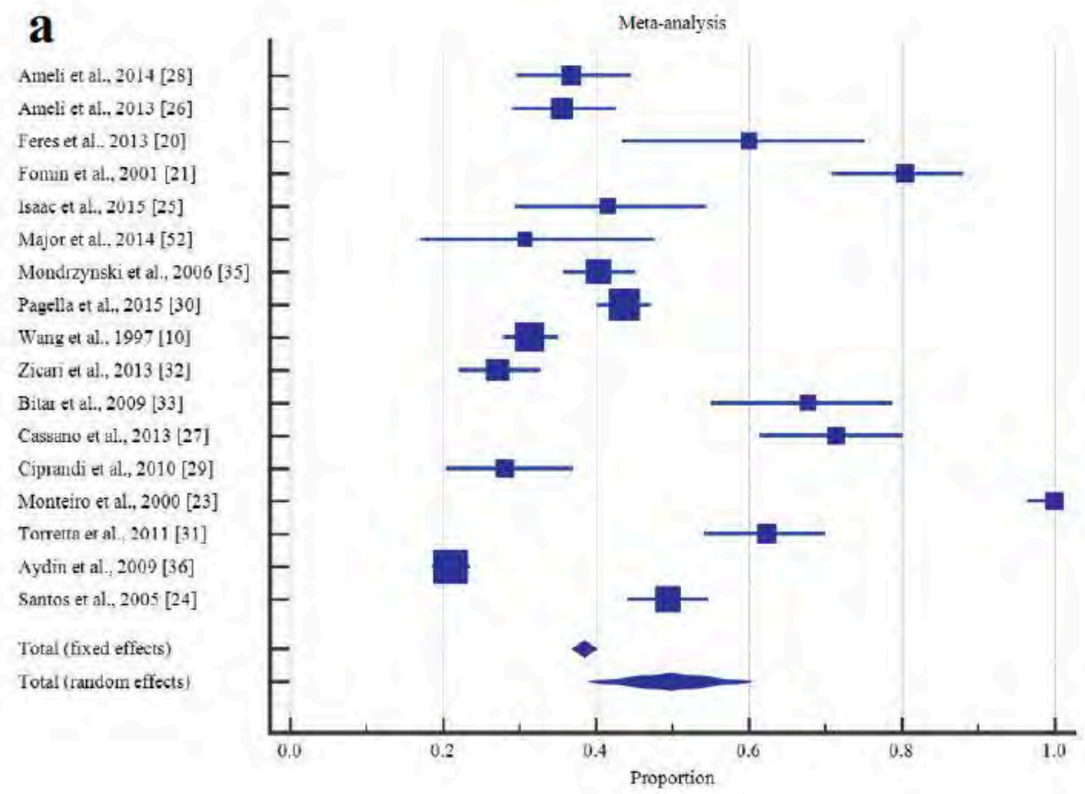
The most common cause of upper airway obstruction in children and adolescents is adenoid hypertrophy (AH). AH is a natural response to increased immunologic activity in early life [1]. Mouth breathing, nasal diseases, asthma, speech problems and obstructive sleep apnea are some of the health issues that can be facilitated, at least partly, by upper airway inflammation and/or obstruction [2,3]. The outcomes of upper airway dysfunction should not be taken lightly as significant upper airway disorders may lead to even more severe conditions such as sleep apnea, altered craniofacial growth and cognitive impairment [4,5].

The current reference standard to diagnose AH is the nasoendoscopy (NE), more specifically nasopharyngoscopy, supported by a comprehensive clinical examination [2]. Ear, nose and throat (ENT) specialists grade adenoid relative airway obstruction using various scales such as proposed by Parikh et al. [6] to measure the severity of the adenoid enlargement and proportionate airway space blockage.

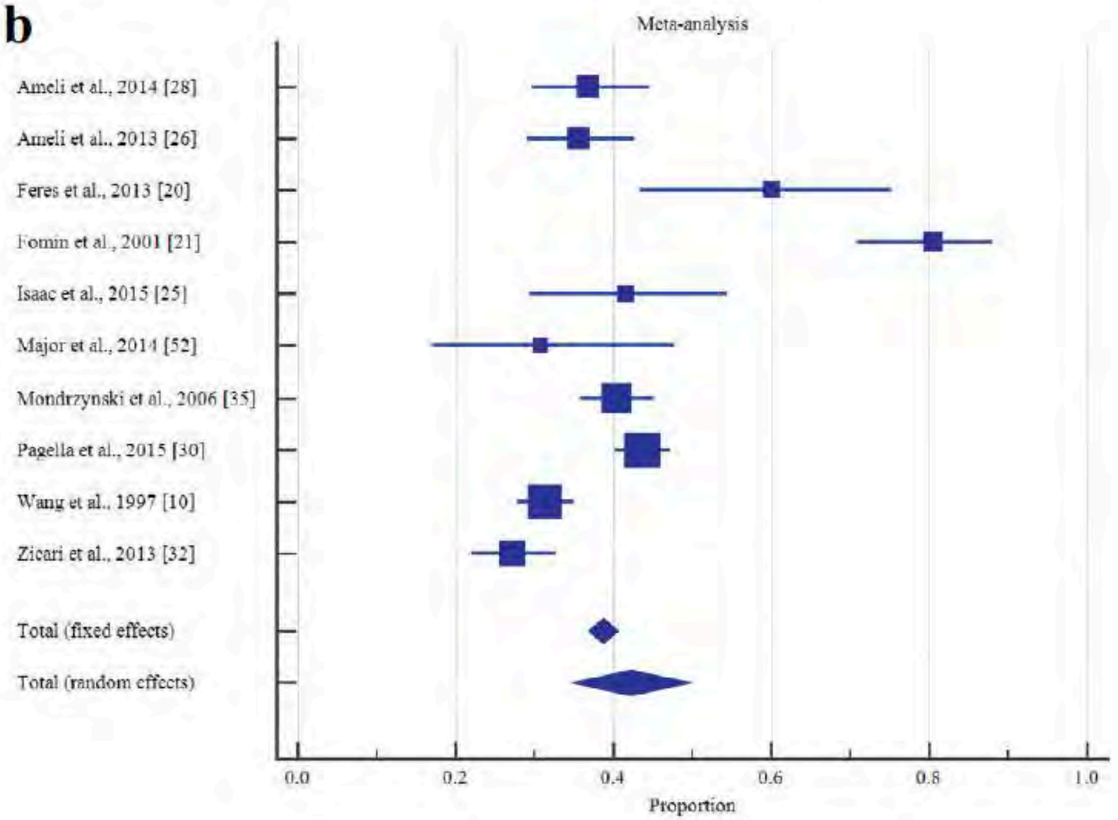
A relatively common treatment approach for obstructive AH is an adenoidectomy [2]. Considering the relatively high demand, associated costs and complications of this surgery, precise and timely recognition of AH is imperative [7]. In this regard, a crucial statistic for clinical decision-making and public health management is the prevalence of different AH grades. It is important for

* Corresponding author. School of Dentistry Faculty of Medicine and Dentistry, 5-533 Edmonton Clinic Health Academy, University of Alberta, 11405-87 Av NW, Edmonton, AB, T6G1C9, Canada.
E-mail address: cppereir@ualberta.ca (C. Pachêco-Pereira).

https://doi.org/10.1016/j.smrv.2017.06.001
1087-0792/



Meta-analysis	proportion	confidence interval
Fixed effects	38.441	37.061 to 39.835
Random effects	49.703	39.917 to 59.501

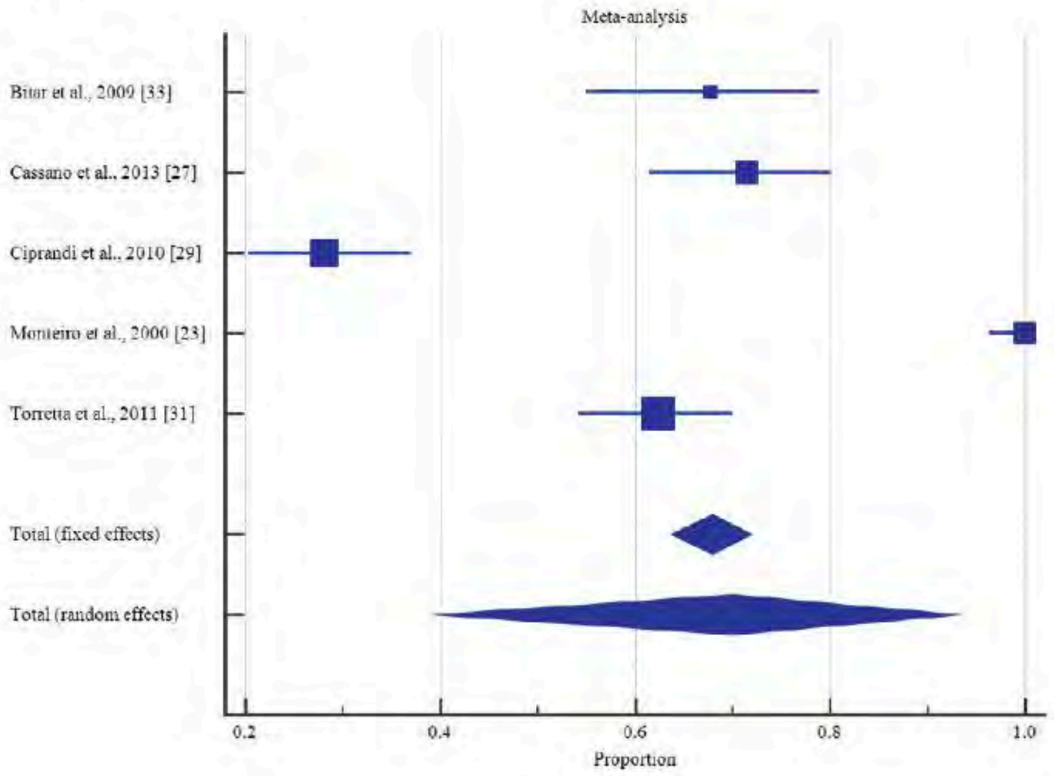


Meta-analysis	proportion	confidence interval
Fixed effects	38.694	36.886 to 40.525
Random effects	42.180	34.935 to 49.599

All studies

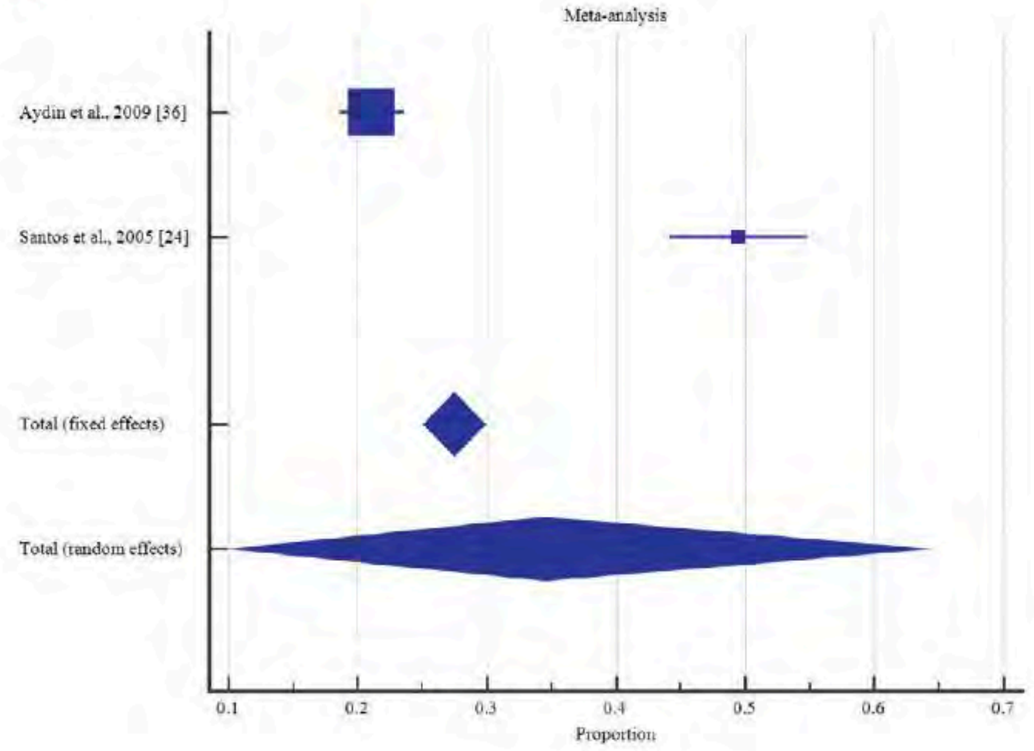
Low RoB studies

c



Meta-Analysis	proportion	confidence interval
Fixed effects	67.918	63.811 to 71.830
Random effects	70.022	40.102 to 92.690

d



Meta-Analysis	proportion	confidence interval
Fixed effects	27.432	25.147 to 29.809
Random effects	34.458	10.507 to 63.742

Moderate RoB studies

Random samples studies

Conclusion

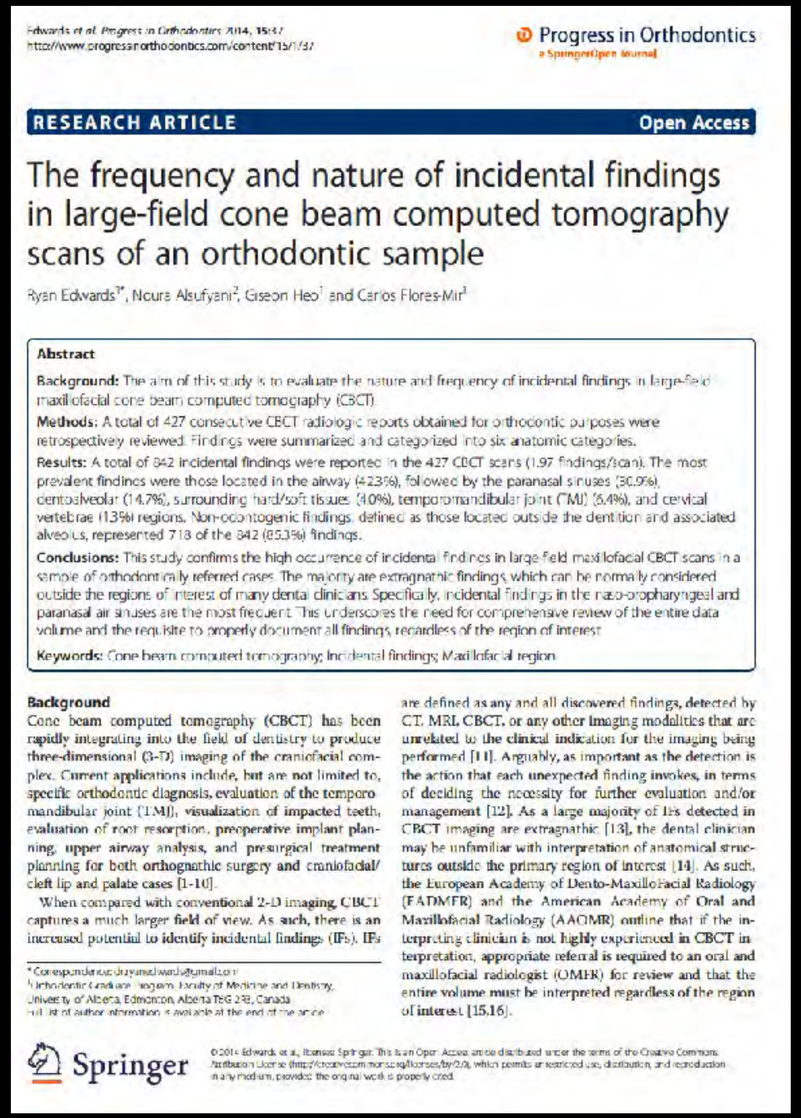
This study suggests that in a randomized representative population, the prevalence of AH was 34%. Otherwise, in convenience samples, including patients referred to ENT clinics, participants without comorbidities and/or fully diagnosed sleep apnea, the prevalence ranged from 42% to 70%.

Practice points

- This meta-analysis showed that in random population samples (meta-analysis for group 3), on patients ranging from 5-14 y old, around 1 in every 3 individuals had confirmed AH.
- In patients referred to ENT specialists because of AH suspicion the average prevalence of confirmed AH was 1 in every 2 patients.
- The prevalence of AH has never been investigated by a meta-analysis. The prevalence numbers presented by our review provide a useful reference on AH prevalence as supported by nasoendoscopy (current gold standard).

Adenoid Hypertrophy Dx

Edwards R, Alsufyani N, Heo G, Flores-Mir C. The frequency and nature of incidental findings in large-field cone beam computed tomography scans of an orthodontic sample. Prog Orthod 2014; 15: 37.



Edwards et al. Progress in Orthodontics 2014, 15:37
http://www.progressinorthodontics.com/content/15/1/37

Progress in Orthodontics
a SpringerOpen Journal

RESEARCH ARTICLE Open Access

The frequency and nature of incidental findings in large-field cone beam computed tomography scans of an orthodontic sample

Ryan Edwards[1*], Noura Alsufyani[2], Giseon Heo[1] and Carlos Flores-Mir[1]

Abstract

Background: The aim of this study is to evaluate the nature and frequency of incidental findings in large-field maxillofacial cone beam computed tomography (CBCT).

Methods: A total of 427 consecutive CBCT radiologic reports obtained for orthodontic purposes were retrospectively reviewed. Findings were summarized and categorized into six anatomic categories.

Results: A total of 842 incidental findings were reported in the 427 CBCT scans (1.97 findings/scan). The most prevalent findings were those located in the airway (42.3%), followed by the paranasal sinuses (30.9%), dentoalveolar (14.7%), surrounding hard/soft tissues (4.0%), temporomandibular joint (TMJ) (5.4%), and cervical vertebrae (1.3%) regions. Non-odontogenic findings, defined as those located outside the dentition and associated alveolus, represented 718 of the 842 (85.3%) findings.

Conclusions: This study confirms the high occurrence of incidental findings in large-field maxillofacial CBCT scans in a sample of orthodontically referred cases. The majority are extragnathic findings which can be normally considered outside the regions of interest of many dental clinicians. Specifically, incidental findings in the naso-oropharyngeal and paranasal air sinuses are the most frequent. This underscores the need for comprehensive review of the entire data volume and the requisite to properly document all findings, regardless of the region of interest.

Keywords: Cone beam computed tomography; Incidental findings; Maxillofacial region

Background

Cone beam computed tomography (CBCT) has been rapidly integrating into the field of dentistry to produce three-dimensional (3-D) imaging of the craniofacial complex. Current applications include, but are not limited to, specific orthodontic diagnosis, evaluation of the temporomandibular joint (TMJ), visualization of impacted teeth, evaluation of root resorption, preoperative implant planning, upper airway analysis, and presurgical treatment planning for both orthognathic surgery and craniofacial/cleft lip and palate cases [1-10].

When compared with conventional 2-D imaging, CBCT captures a much larger field of view. As such, there is an increased potential to identify incidental findings (IFs). IFs are defined as any and all discovered findings, detected by CT, MRI, CBCT, or any other imaging modalities that are unrelated to the clinical indication for the imaging being performed [11]. Arguably, as important as the detection is the action that each unexpected finding invokes, in terms of deciding the necessity for further evaluation and/or management [12]. As a large majority of IFs detected in CBCT imaging are extragnathic [13], the dental clinician may be unfamiliar with interpretation of anatomical structures outside the primary region of interest [14]. As such, the European Academy of Dento-Maxillofacial Radiology (EADMFR) and the American Academy of Oral and Maxillofacial Radiology (AAOMR) outline that if the interpreting clinician is not highly experienced in CBCT interpretation, appropriate referral is required to an oral and maxillofacial radiologist (OMFR) for review and that the entire volume must be interpreted regardless of the region of interest [15,16].

* Correspondence: drryanedwards@gmail.com
[1]Orthodontic Graduate Program, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta T6G 2R3, Canada
Full list of author information is available at the end of the article

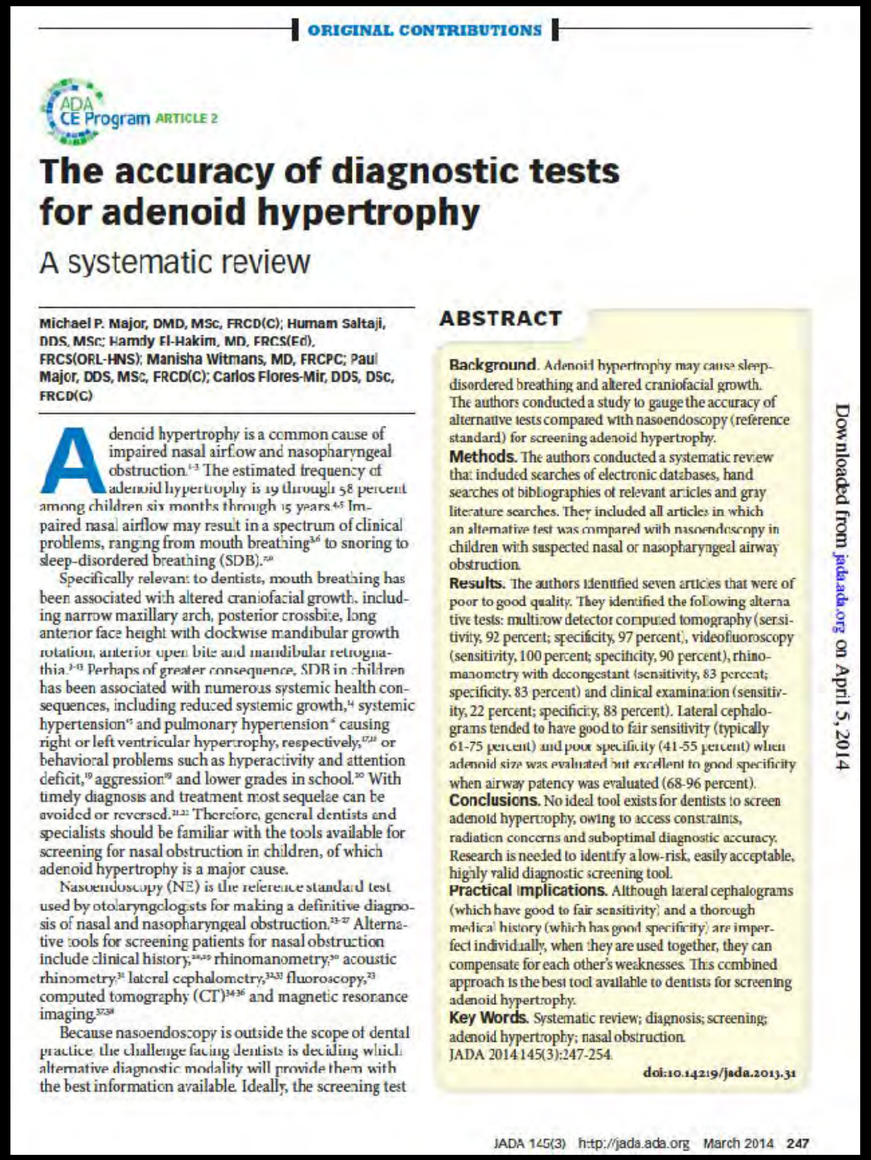
Adenoid Hypertrophy Risk Factor

1.97 IFs/CBCT scan.

The most prevalent findings were those located in the upper airway (42.3%).

Adenoid Hypertrophy Dx

Major MP, Saltaji H, El-Hakim H, Witmans M, Major PW, Flores-Mir C. The Accuracy of Diagnostic Tests for Adenoid Hypertrophy: A Systematic Review. J Am Dent Assoc 2014; 145(3): 247-54.



ORIGINAL CONTRIBUTIONS

ADA CE Program ARTICLE 2

The accuracy of diagnostic tests for adenoid hypertrophy

A systematic review

Michael P. Major, DMD, MSc, FRCD(C); Humam Saltaji, DDS, MSc; Hamdy El-Hakim, MD, FRCS(Ed), FRCS(ORL-HNS); Manisha Witmans, MD, FRCPC; Paul Major, DDS, MSc, FRCD(C); Carlos Flores-Mir, DDS, DSc, FRCD(C)

ABSTRACT

Background. Adenoid hypertrophy may cause sleep-disordered breathing and altered craniofacial growth. The authors conducted a study to gauge the accuracy of alternative tests compared with nasoendoscopy (reference standard) for screening adenoid hypertrophy.

Methods. The authors conducted a systematic review that included searches of electronic databases, hand searches of bibliographies of relevant articles and gray literature searches. They included all articles in which an alternative test was compared with nasoendoscopy in children with suspected nasal or nasopharyngeal airway obstruction.

Results. The authors identified seven articles that were of poor to good quality. They identified the following alternative tests: multirow detector computed tomography (sensitivity, 92 percent; specificity, 97 percent), videofluoroscopy (sensitivity, 100 percent; specificity, 90 percent), rhinomanometry with decongestant (sensitivity, 83 percent; specificity, 83 percent) and clinical examination (sensitivity, 22 percent; specificity, 88 percent). Lateral cephalograms tended to have good to fair sensitivity (typically 61-75 percent) and poor specificity (41-55 percent) when adenoid size was evaluated but excellent to good specificity when airway patency was evaluated (68-96 percent).

Conclusions. No ideal tool exists for dentists to screen adenoid hypertrophy, owing to access constraints, radiation concerns and suboptimal diagnostic accuracy. Research is needed to identify a low-risk, easily acceptable, highly valid diagnostic screening tool.

Practical Implications. Although lateral cephalograms (which have good to fair sensitivity) and a thorough medical history (which has good specificity) are imperfect individually, when they are used together, they can compensate for each other's weaknesses. This combined approach is the best tool available to dentists for screening adenoid hypertrophy.

Key Words. Systematic review; diagnosis; screening; adenoid hypertrophy; nasal obstruction.

JADA 2014;145(3):247-254.

doi:10.14219/jada.2013.31

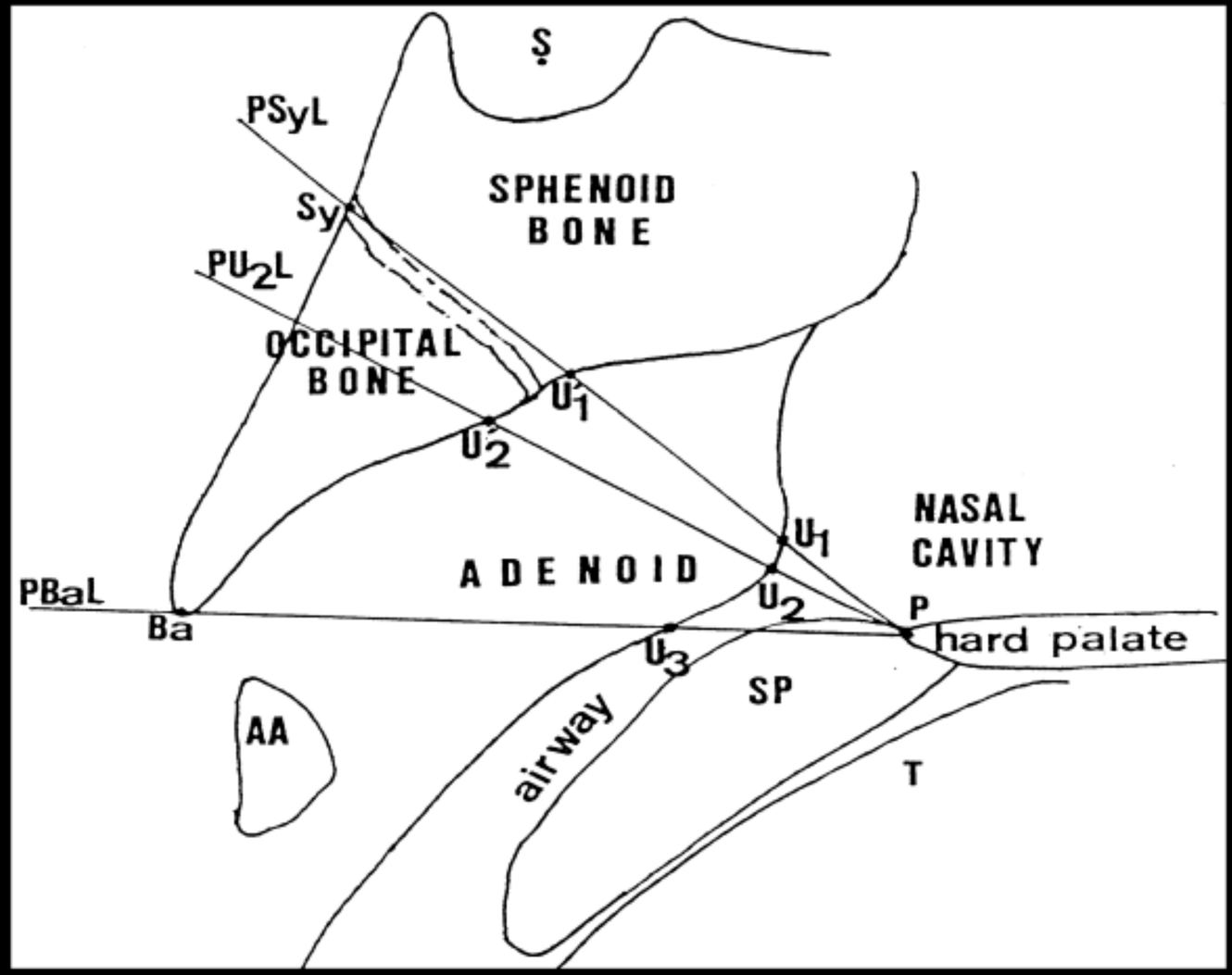
Adenoid hypertrophy is a common cause of impaired nasal airflow and nasopharyngeal obstruction.[1-3] The estimated frequency of adenoid hypertrophy is 19 through 58 percent among children six months through 15 years.[4,5] Impaired nasal airflow may result in a spectrum of clinical problems, ranging from mouth breathing[3,6] to snoring to sleep-disordered breathing (SDB).[7,8]

Specifically relevant to dentists, mouth breathing has been associated with altered craniofacial growth, including narrow maxillary arch, posterior crossbite, long anterior face height with clockwise mandibular growth rotation, anterior open bite and mandibular retrognathia.[3-13] Perhaps of greater consequence, SDB in children has been associated with numerous systemic health consequences, including reduced systemic growth,[14] systemic hypertension[15] and pulmonary hypertension[16] causing right or left ventricular hypertrophy, respectively,[17,18] or behavioral problems such as hyperactivity and attention deficit,[19] aggression[19] and lower grades in school.[20] With timely diagnosis and treatment most sequelae can be avoided or reversed.[21,22] Therefore, general dentists and specialists should be familiar with the tools available for screening for nasal obstruction in children, of which adenoid hypertrophy is a major cause.

Nasoendoscopy (NE) is the reference standard test used by otolaryngologists for making a definitive diagnosis of nasal and nasopharyngeal obstruction.[23-27] Alternative tools for screening patients for nasal obstruction include clinical history,[28,29] rhinomanometry,[30] acoustic rhinometry,[31] lateral cephalometry,[32,33] fluoroscopy,[23] computed tomography (CT)[34-36] and magnetic resonance imaging.[37,38]

Because nasoendoscopy is outside the scope of dental practice, the challenge facing dentists is deciding which alternative diagnostic modality will provide them with the best information available. Ideally, the screening test

Adenoid Hypertrophy Dx



Adenoid Hypertrophy Dx

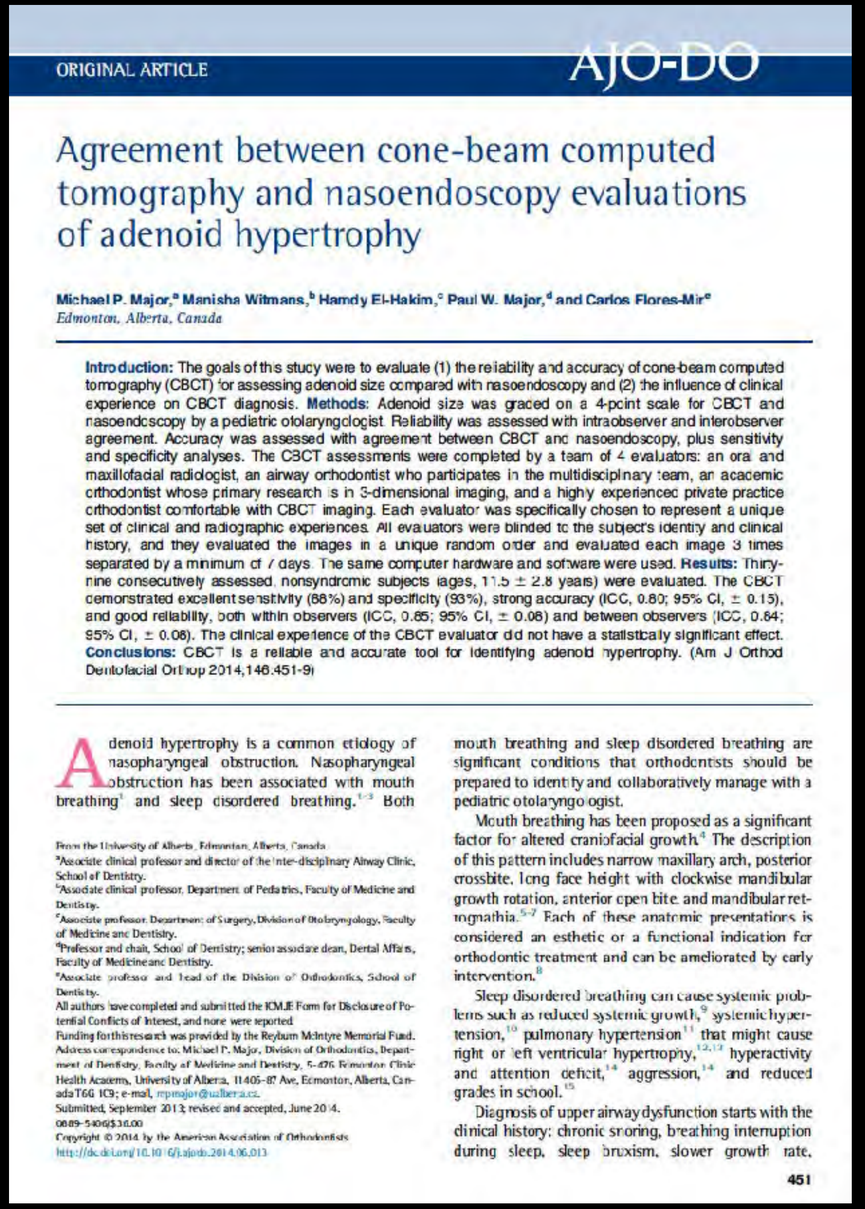
Diagnostic Test	Sensitivity	Specificity	N
Clinical Exam			
NOI[46]	22%	88%	154
Rhinomanometry			
without decongestant[50]	81%	34%	71
with decongestant[50]	83%	83%	52
Lateral Cephalogram			
adenoid size[45]	86%	41%	48
A/N ratio[45]	41%	95%	48
adenoid – turbinate airway[45]	61%	68%	48
airway-palate ratio[45]	66%	96%	48
airway size (McNamara line)[47]	75%	86%	30
subjective assess[27]	70%	55%	40
subjective assess[48]	70%	52%	70
Video Fluoroscopy			
Ysunza 2008[27]	100%	90%	40
Ysunza 2011[48]	100%	93%	70
Multi-row detector CT			
Hoppe et al 2002[49]	92%	97%	29

Adenoid Hypertrophy Dx

- No ideal tool exists for dentists to screen for adenoid hypertrophy
- Current concerns access constrains, radiation and suboptimal dx capability
- Combination of lateral cephalogram (good to fair SE) and medical history (good SP) best tool available

Adenoid Hypertrophy Dx

Major MP, El-Hakim H, Witmans M, Major PW, Flores-Mir C. Agreement between CBCT and Nasoendoscopy Evaluations of Adenoidal Hypertrophy. Am J Orthod Dentofac Orthop 2014; 146: 451-9.



ORIGINAL ARTICLE AJO-DO

Agreement between cone-beam computed tomography and nasoendoscopy evaluations of adenoid hypertrophy

Michael P. Major,[a] Manisha Witmans,[b] Hamdy El-Hakim,[c] Paul W. Major,[d] and Carlos Flores-Mir[e]
Edmonton, Alberta, Canada

Introduction: The goals of this study were to evaluate (1) the reliability and accuracy of cone-beam computed tomography (CBCT) for assessing adenoid size compared with nasoendoscopy and (2) the influence of clinical experience on CBCT diagnosis. **Methods:** Adenoid size was graded on a 4-point scale for CBCT and nasoendoscopy by a pediatric otolaryngologist. Reliability was assessed with intraobserver and interobserver agreement. Accuracy was assessed with agreement between CBCT and nasoendoscopy, plus sensitivity and specificity analyses. The CBCT assessments were completed by a team of 4 evaluators: an oral and maxillofacial radiologist, an airway orthodontist who participates in the multidisciplinary team, an academic orthodontist whose primary research is in 3-dimensional imaging, and a highly experienced private practice orthodontist comfortable with CBCT imaging. Each evaluator was specifically chosen to represent a unique set of clinical and radiographic experiences. All evaluators were blinded to the subject's identity and clinical history, and they evaluated the images in a unique random order and evaluated each image 3 times separated by a minimum of 7 days. The same computer hardware and software were used. **Results:** Thirty-nine consecutively assessed, nonsyndromic subjects (ages, 11.5 ± 2.8 years) were evaluated. The CBCT demonstrated excellent sensitivity (88%) and specificity (93%), strong accuracy (ICC, 0.80; 95% CI, ± 0.15), and good reliability, both within observers (ICC, 0.85; 95% CI, ± 0.08) and between observers (ICC, 0.84; 95% CI, ± 0.08). The clinical experience of the CBCT evaluator did not have a statistically significant effect. **Conclusions:** CBCT is a reliable and accurate tool for identifying adenoid hypertrophy. (Am J Orthod Dentofacial Orthop 2014;146:451-9)

Adenoid hypertrophy is a common etiology of nasopharyngeal obstruction. Nasopharyngeal obstruction has been associated with mouth breathing[1] and sleep disordered breathing.[1-3] Both mouth breathing and sleep disordered breathing are significant conditions that orthodontists should be prepared to identify and collaboratively manage with a pediatric otolaryngologist.

Mouth breathing has been proposed as a significant factor for altered craniofacial growth.[4] The description of this pattern includes narrow maxillary arch, posterior crossbite, long face height with clockwise mandibular growth rotation, anterior open bite, and mandibular retrognathia.[5-7] Each of these anatomic presentations is considered an esthetic or a functional indication for orthodontic treatment and can be ameliorated by early intervention.[8]

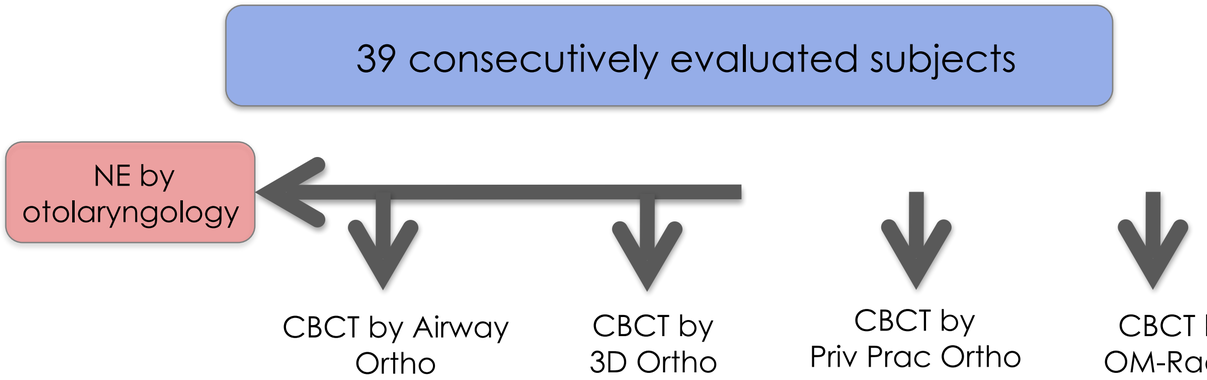
Sleep disordered breathing can cause systemic problems such as reduced systemic growth,[9] systemic hypertension,[10] pulmonary hypertension[11] that might cause right or left ventricular hypertrophy,[12,13] hyperactivity and attention deficit,[14] aggression,[14] and reduced grades in school.[15]

Diagnosis of upper airway dysfunction starts with the clinical history: chronic snoring, breathing interruption during sleep, sleep bruxism, slower growth rate,

From the University of Alberta, Edmonton, Alberta, Canada.
[a]Associate clinical professor and director of the Inter-disciplinary Airway Clinic, School of Dentistry.
[b]Associate clinical professor, Department of Pediatrics, Faculty of Medicine and Dentistry.
[c]Associate professor, Department of Surgery, Division of Otolaryngology, Faculty of Medicine and Dentistry.
[d]Professor and chair, School of Dentistry; senior associate dean, Dental Affairs, Faculty of Medicine and Dentistry.
[e]Associate professor and head of the Division of Orthodontics, School of Dentistry.
All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and none were reported.
Funding for this research was provided by the Reyburn McIntyre Memorial Fund.
Address correspondence to: Michael P. Major, Division of Orthodontics, Department of Dentistry, Faculty of Medicine and Dentistry, 5-476 Edmonton Clinic Health Academy, University of Alberta, 11405-87 Ave, Edmonton, Alberta, Canada T6G 1C9; e-mail, mpmajor@ualberta.ca.
Submitted, September 2013; revised and accepted, June 2014.
0889-5406/$36.00

http://dx.doi.org/10.1016/j.ajodo.2014.06.013

Adenoid Hypertrophy Dx



- CBCT data: collected prospectively through blinded & randomized evaluations
- NE data: collected retrospectively through chart review

Adenoid Hypertrophy Dx

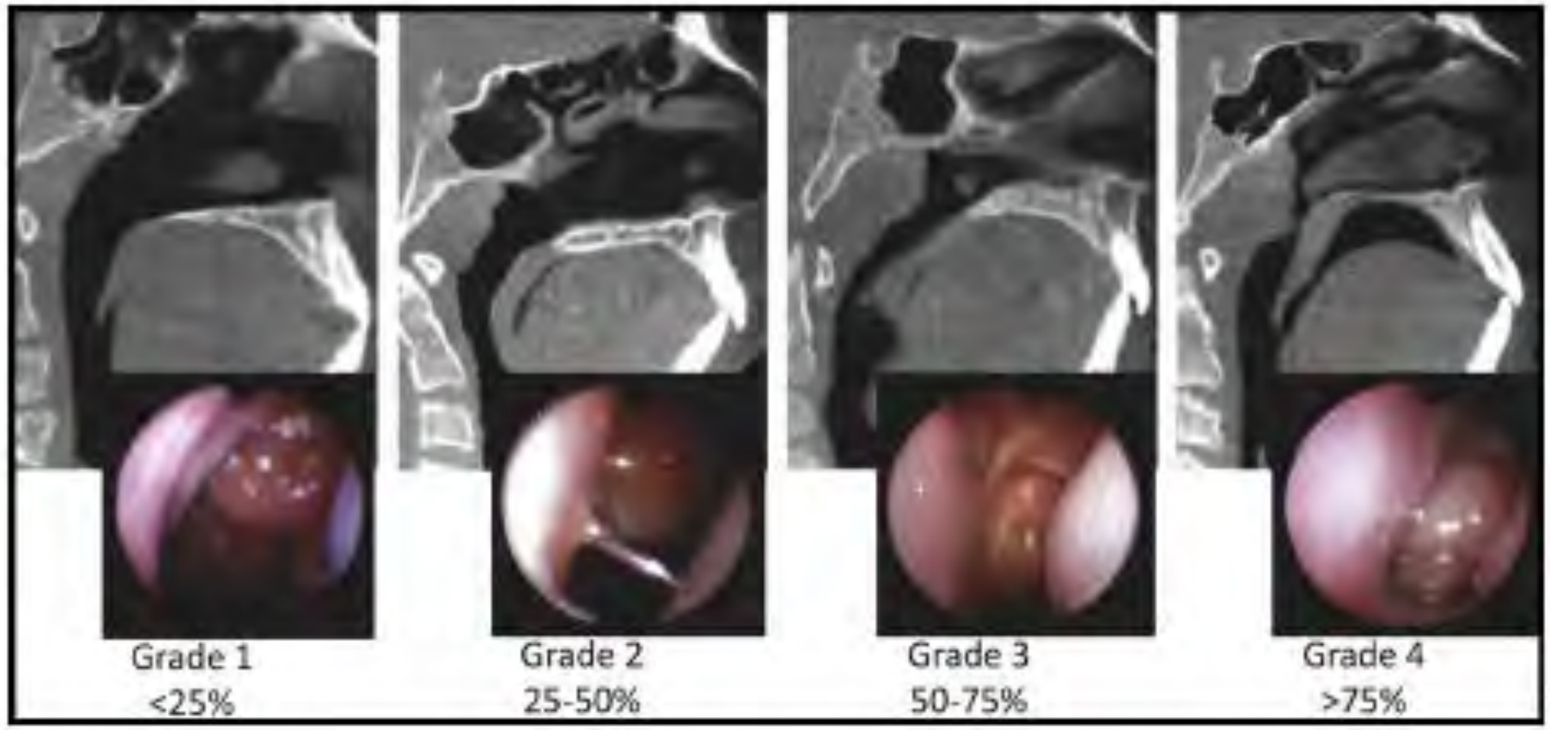


Fig 1. Adenoid size in CBCT midsagittal slice and corresponding adenoid viewed with NE.

Adenoid Hypertrophy Dx

- Reliability (ICC) of CBCT adenoid assessment across evaluators

	Intra-observer agreement between repeated CBCT (ICC ± 95% CI)	Inter-observer agreement between NE & CBCT (ICC ± 95% CI)
Maxillofacial Radiologist	*0.89 ± 0.07*	*0.81 ± 0.14*
Airway Ortho	*0.89 ± 0.07*	*0.81 ± 0.14*
Adademic 3D Ortho	*0.90 ± 0.07*	*0.82 ± 0.14*
Private Practice Ortho	*0.74 ± 0.13*	*0.74 ± 0.18*
Average	*0.85 ± 0.08*	*0.80 ± 0.15*

Adenoid Hypertrophy Dx

- Accuracy (sensitivity / specificity) of CBCT adenoid assessment across evaluators

	Sensitivity	Specificity
Maxillofacial Radiologist	*83.3%*	*92.6%*
Airway Ortho	*83.3%*	*96.2%*
3D-Research Ortho	*91.7%*	*92.6%*
Private Practice Ortho	*91.7%*	*88.9%*
Average	*88%*	*93%*

Adenoid Hypertrophy Dx

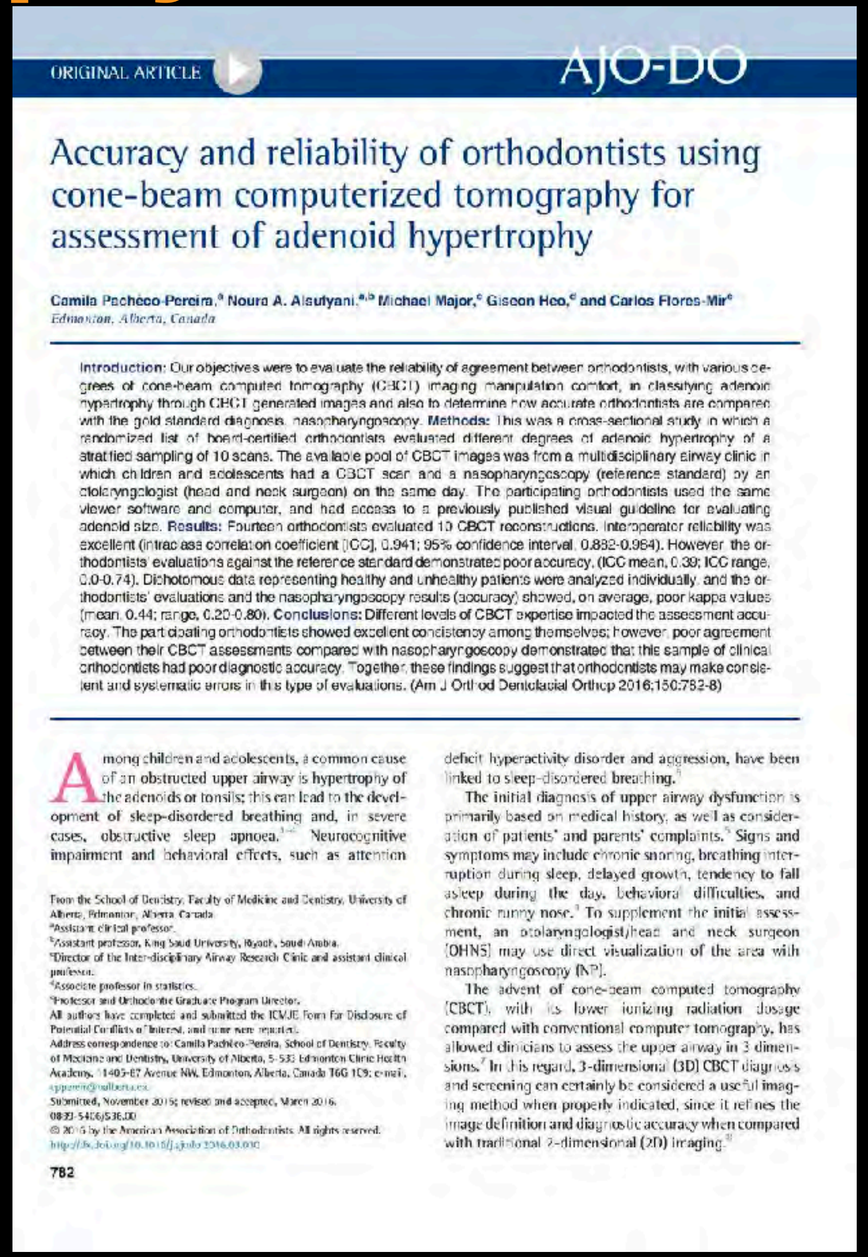
Alternative Test	Sens	Spec
Clinical Exam (NOI)	***22%***	***88%***
Rhinomanometry	81-83%	34-83%
Lateral Cephalogram	***41%-86%***	***41%-96%***
Video Fluoroscopy	100%	90-93%
Multi-row CT	***92%***	***97%***
CBCT	***88%***	***93%***

Adenoid Hypertrophy Dx - Clinical Trial

- Private practice orthodontists may be less accurate with grading adenoid size,
BUT
appear equally effective at identifying clinically relevant adenoid hypertrophy

Adenoid Hypertrophy Dx

Pacheco-Pereira C, Alsufyani N, Major MP, Heo G, Flores-Mir C. Accuracy and reliability of orthodontists using CBCT for assessment of adenoid hypertrophy. Am J Orthod Dentofac Orthop 2016; 150: 782-8.



ORIGINAL ARTICLE

AJO-DO

Accuracy and reliability of orthodontists using cone-beam computerized tomography for assessment of adenoid hypertrophy

Camila Pacheco-Pereira,[a] Noura A. Alsufyani,[a,b] Michael Major,[c] Giseon Heo,[d] and Carlos Flores-Mir[e]
Edmonton, Alberta, Canada

Introduction: Our objectives were to evaluate the reliability of agreement between orthodontists, with various degrees of cone-beam computed tomography (CBCT) imaging manipulation comfort, in classifying adenoid hypertrophy through CBCT generated images and also to determine how accurate orthodontists are compared with the gold standard diagnosis, nasopharyngoscopy. **Methods:** This was a cross-sectional study in which a randomized list of board-certified orthodontists evaluated different degrees of adenoid hypertrophy of a stratified sampling of 10 scans. The available pool of CBCT images was from a multidisciplinary airway clinic in which children and adolescents had a CBCT scan and a nasopharyngoscopy (reference standard) by an otolaryngologist (head and neck surgeon) on the same day. The participating orthodontists used the same viewer software and computer, and had access to a previously published visual guideline for evaluating adenoid size. **Results:** Fourteen orthodontists evaluated 10 CBCT reconstructions. Interoperator reliability was excellent (intraclass correlation coefficient [ICC], 0.941; 95% confidence interval, 0.882-0.964). However, the orthodontists' evaluations against the reference standard demonstrated poor accuracy. (ICC mean, 0.39; ICC range, 0.0-0.74). Dichotomous data representing healthy and unhealthy patients were analyzed individually, and the orthodontists' evaluations and the nasopharyngoscopy results (accuracy) showed, on average, poor kappa values (mean, 0.44; range, 0.20-0.80). **Conclusions:** Different levels of CBCT expertise impacted the assessment accuracy. The participating orthodontists showed excellent consistency among themselves; however, poor agreement between their CBCT assessments compared with nasopharyngoscopy demonstrated that this sample of clinical orthodontists had poor diagnostic accuracy. Together, these findings suggest that orthodontists may make consistent and systematic errors in this type of evaluations. (Am J Orthod Dentofacial Orthop 2016;150:782-8)

Among children and adolescents, a common cause of an obstructed upper airway is hypertrophy of the adenoids or tonsils; this can lead to the development of sleep-disordered breathing and, in severe cases, obstructive sleep apnoea.[1-3] Neurocognitive impairment and behavioral effects, such as attention deficit hyperactivity disorder and aggression, have been linked to sleep-disordered breathing.[4]

The initial diagnosis of upper airway dysfunction is primarily based on medical history, as well as consideration of patients' and parents' complaints.[5] Signs and symptoms may include chronic snoring, breathing interruption during sleep, delayed growth, tendency to fall asleep during the day, behavioral difficulties, and chronic runny nose.[1] To supplement the initial assessment, an otolaryngologist/head and neck surgeon (OHNS) may use direct visualization of the area with nasopharyngoscopy (NP).

The advent of cone-beam computed tomography (CBCT), with its lower ionizing radiation dosage compared with conventional computer tomography, has allowed clinicians to assess the upper airway in 3 dimensions.[7] In this regard, 3-dimensional (3D) CBCT diagnosis and screening can certainly be considered a useful imaging method when properly indicated, since it refines the image definition and diagnostic accuracy when compared with traditional 2-dimensional (2D) imaging.[8]

From the School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada.
[a]Assistant clinical professor.
[b]Assistant professor, King Saud University, Riyadh, Saudi Arabia.
[c]Director of the Inter-disciplinary Airway Research Clinic and assistant clinical professor.
[d]Associate professor in statistics.
[e]Professor and Orthodontic Graduate Program Director.
All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and none were reported.
Address correspondence to: Camila Pacheco-Pereira, School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, 5-532 Edmonton Clinic Health Academy, 11405-87 Avenue NW, Edmonton, Alberta, Canada T6G 1C9; e-mail, cpereira@ualberta.ca.
Submitted, November 2015; revised and accepted, March 2016.
0889-5406/$36.00

http://dx.doi.org/10.1016/j.ajodo.2016.03.030

Adenoid Hypertrophy Dx

- Different levels of CBCT expertise impacted assessment accuracy.
- Excellent consistency among themselves, but poor agreement/accuracy.
- Orthodontists may make consistent and systematic errors in this type of evaluations.

Adenoid Hypertrophy Dx

Pacheco-Pereira C, Alsufyani N, Major MP, Flores-Mir C. Accuracy and reliability of oral maxillofacial radiologists when evaluating CBCT imaging for adenoid hypertrophy screening: a comparison with nasopharyngealngoscopy. Oral Surg Oral Med Oral Pathol Oral Radiol 2016; 121: e168-74.

Vol. 121 No. 6 June 2016

Accuracy and reliability of oral maxillofacial radiologists when evaluating cone-beam computed tomography imaging for adenoid hypertrophy screening: a comparison with nasopharyngoscopy

Camila Pacheco-Pereira, DDS, MBA, MSc,[a] Noura A. Alsufyani, BDS, MSc, PhD, FRCD(c), Diplm. ABOMR,[b] Michael P. Major, DMD, MSc, FRCD(c),[c] and Carlos Flores-Mir, DDS, PhD, FRCD(c)[d]

Objective. To determine how accurate and reliable oral maxillofacial radiologists (OMFRs) are in screening for adenoid hypertrophy when using cone-beam computed tomography (CBCT) imaging compared with nasopharyngoscopy (NP).
Study Design. CBCT scans of 10 patients with distinct levels of adenoid hypertrophy were randomly selected. Fourteen board-certified OMFRs classified the levels of hypertrophy. The intraclass correlation coefficient (ICC) was used to assess accuracy by comparing their diagnosis against an NP diagnosis, which is the reference standard. OMFRs' interreliability was assessed. Kappa statistics were used to analyze dichotomous data from healthy and unhealthy patients.
Results. Overall, the reliability among OMFRs was good (ICC = 0.79 with confidence interval [CI] 0.63–0.93). The "statistical mode" was very good (ICC = 0.87; CI 0.43–0.94). The accuracy of OMFRs against NP was good (ICC_{mean} = 0.69; CI 0.43–0.94). On average, the Kappa statistics (K_{mean} = 0.77, CI 0.62–0.92) demonstrated a good agreement between OMFRs and NP diagnoses. The individualized results from each evaluator were presented and investigated according to their performance.
Conclusions. Compared with the reference standard, the accuracy of OMFRs to classify adenoid hypertrophy on a four-level scale was moderate to strong and improved when adenoid hypertrophy was classified as healthy or unhealthy. The reliability of the OMFRs was greater than 80%, assuring their consistency and reliability on screening adenoids hypertrophy via CBCT.
(Oral Surg Oral Med Oral Pathol Oral Radiol 2016;121:e168-e174)

Among the first structures in our immune system that protect us from foreign bodies are adenoids and tonsils. These lymphoid tissues are constantly exposed to antigens coming from allergic diseases, passive smoking, or food.[1,2] The body's reactions to these constant irritants could cause recurrent episodes of localized upper respiratory inflammation processes.[3]

The diagnosis of upper airway obstruction is primarily made on the basis of a medical history, associated with a chief complaint, either from patients and/or parents. The adenoids are strategically located superiorly and posteriorly in the nasopharynx. If the determination of an upper airway obstruction remains inconclusive after the clinical assessment, direct visualization of the adenoid becomes mandatory. Nasopharyngoscopy is the "reference standard" tool for adenoidal direct examination, being a noninvasive and radiation-free technique that facilitates the assessment of adults and children without sedation.

In the medical[4] and dental[5] fields, two-dimensional skull radiograph was traditionally elected as screening tool for determining the adenoid size. However, superimposition of structures as a result of a static view (evaluation on a single-plane view) and the limitation to two space planes do not allow for an accurate reflection of the craniofacial lymphoid tissues.[5,6] Specifically in dentistry, cone-beam computed tomography (CBCT) has provided the opportunity to assess the cross-sectional area and volumetric portrayal of the upper airway simultaneously, with refined image definition

[a]Oral Radiology Specialty & Assistant Clinical Professor, School of Dentistry, Faculty of Medicine and Dentistry, School of Dentistry, University of Alberta, Edmonton, Canada.
[b]Oral & Maxillofacial Radiologist - Professor, School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Canada; Oral Maxillofacial Radiologist, Assistant Professor, College of Dentistry, King Saud University, Riyadh, Saudi Arabia.
[c]Director of the Inter-disciplinary Airway Research Clinic & Assistant Clinical Professor in Orthodontics, School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Canada.
[d]Professor & Orthodontic Graduate Program Director, School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Canada.
Received for publication Nov 25, 2015; returned for revision Feb 23, 2016; accepted for publication Mar 9, 2016.

2212-4403/$ - see front matter
http://dx.doi.org/10.1016/j.oooo.2016.03.010

Statement of Clinical Relevance

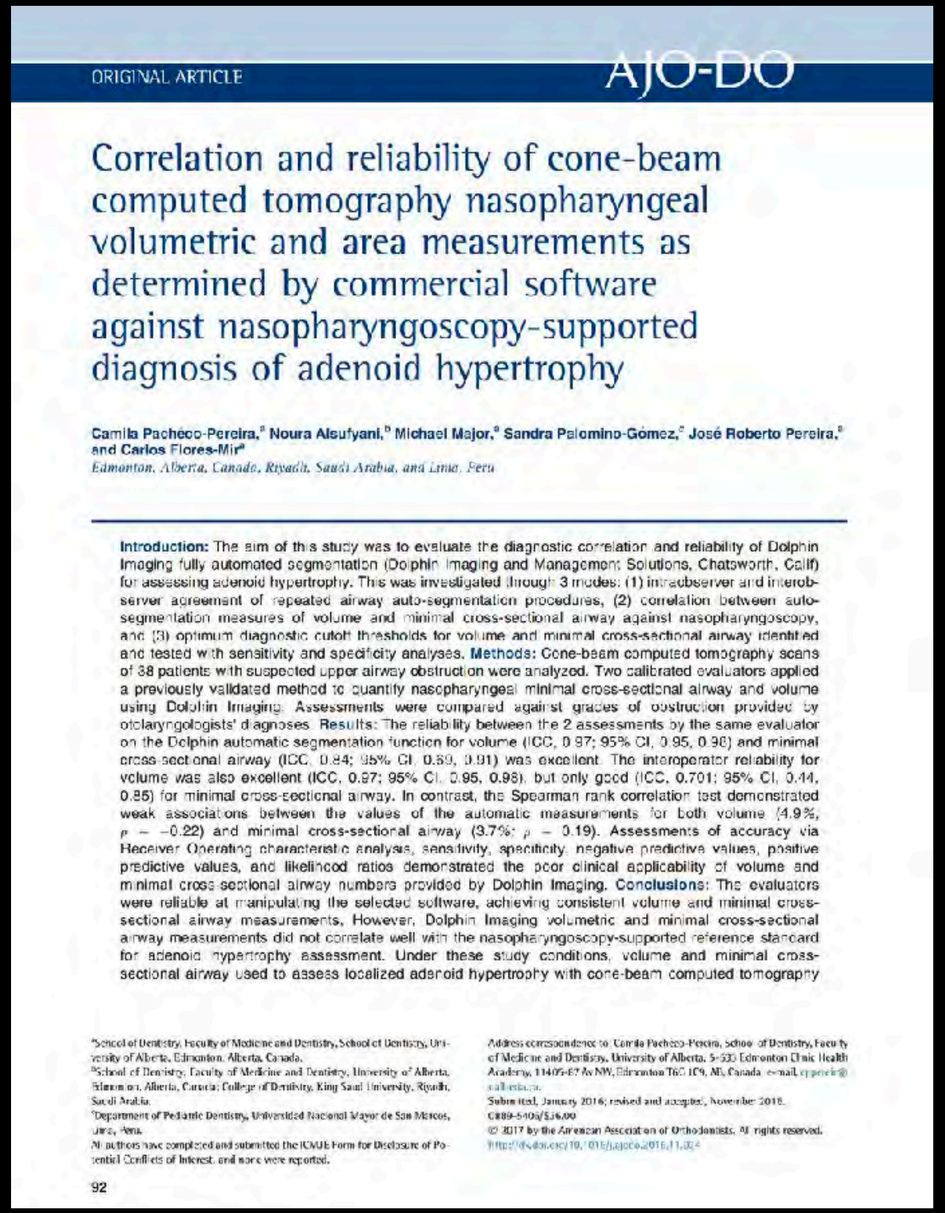
Upper airway dysfunction and adenoid hypertrophy affect children and their families. Oral maxillofacial radiologists have the opportunity of analyze incidental findings on the upper airway area while interpreting three-dimensional imaging. Early screening of sleep disordered breathing could prevent mouth-breathing tendencies.

Adenoid Hypertrophy Dx

- OMFRs showed moderate to strong accuracy.
- When healthy/unhealthy classification was used it improved.
- Reliability was greater than 80%.

Adenoid Hypertrophy Dx

Pacheco-Pereira C, Alsufyani N, Major MP, Palomino-Gomez S, Pereira JR, Flores-Mir C. Correlation and reliability of CBCT nasopharyngeal volumetric and area measurements as determined by commercial software against nasopharyngosscopy-supported diagnosis of adenoid hypertrophy. Am J Orthod Dentofac Orthop 2017; 152: 92-103.



ORIGINAL ARTICLE

AJO-DO

Correlation and reliability of cone-beam computed tomography nasopharyngeal volumetric and area measurements as determined by commercial software against nasopharyngoscopy-supported diagnosis of adenoid hypertrophy

Camila Pacheco-Pereira,[a] Noura Alsufyani,[b] Michael Major,[a] Sandra Palomino-Gómez,[c] José Roberto Pereira,[a] and Carlos Flores-Mir[b]
Edmonton, Alberta, Canada; Riyadh, Saudi Arabia; and Lima, Peru

Introduction: The aim of this study was to evaluate the diagnostic correlation and reliability of Dolphin Imaging fully automated segmentation (Dolphin Imaging and Management Solutions, Chatsworth, Calif) for assessing adenoid hypertrophy. This was investigated through 3 modes: (1) intraobserver and interobserver agreement of repeated airway auto-segmentation procedures, (2) correlation between auto-segmentation measures of volume and minimal cross-sectional airway against nasopharyngoscopy, and (3) optimum diagnostic cutoff thresholds for volume and minimal cross-sectional airway identified and tested with sensitivity and specificity analyses. **Methods:** Cone-beam computed tomography scans of 38 patients with suspected upper airway obstruction were analyzed. Two calibrated evaluators applied a previously validated method to quantify nasopharyngeal minimal cross-sectional airway and volume using Dolphin Imaging. Assessments were compared against grades of obstruction provided by otolaryngologists' diagnoses. **Results:** The reliability between the 2 assessments by the same evaluator on the Dolphin automatic segmentation function for volume (ICC, 0.97; 95% CI, 0.95, 0.98) and minimal cross-sectional airway (ICC, 0.84; 95% CI, 0.69, 0.91) was excellent. The interoperator reliability for volume was also excellent (ICC, 0.97; 95% CI, 0.95, 0.98), but only good (ICC, 0.701; 95% CI, 0.44, 0.85) for minimal cross-sectional airway. In contrast, the Spearman rank correlation test demonstrated weak associations between the values of the automatic measurements for both volume (4.9%, $p = -0.22$) and minimal cross-sectional airway (3.7%; $p = 0.19$). Assessments of accuracy via Receiver Operating characteristic analysis, sensitivity, specificity, negative predictive values, positive predictive values, and likelihood ratios demonstrated the poor clinical applicability of volume and minimal cross-sectional airway numbers provided by Dolphin Imaging. **Conclusions:** The evaluators were reliable at manipulating the selected software, achieving consistent volume and minimal cross-sectional airway measurements. However, Dolphin Imaging volumetric and minimal cross-sectional airway measurements did not correlate well with the nasopharyngoscopy-supported reference standard for adenoid hypertrophy assessment. Under these study conditions, volume and minimal cross-sectional airway used to assess localized adenoid hypertrophy with cone-beam computed tomography

[a]School of Dentistry, Faculty of Medicine and Dentistry, School of Dentistry, University of Alberta, Edmonton, Alberta, Canada.
[b]School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada; College of Dentistry, King Saud University, Riyadh, Saudi Arabia.
[c]Department of Pediatric Dentistry, Universidad Nacional Mayor de San Marcos, Lima, Peru.
All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and none were reported.

Address correspondence to: Camila Pacheco-Pereira, School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, 5-533 Edmonton Clinic Health Academy, 11405-87 Ave NW, Edmonton T6G 1C9, AB, Canada. e-mail, cpereira@ualberta.ca.
Submitted, January 2016; revised and accepted, November 2016.
0889-5406/$36.00

http://dx.doi.org/10.1016/j.ajodo.2016.11.024

Adenoid Hypertrophy Dx

- Although imaging software can be reliable, volumetric and cross-sectional area measurements were not strongly correlated to the ENT nasopharyngospic-based diagnosis.
- Poor sensitivity and specificity (43-66%) for assessing upper airway adenoid hypertrophy obstruction assessment.

Adenoid Hypertrophy Dx

IF CBCT HAS BEEN INDICATED FOR OTHER REASONS THEN IT COULD BE USED FOR ADENOID HYPERTROPHY SCREENING

Adenoid Hypertrophy Dx

Major MP, El-Hakim H, Witmans M, Major PW, Flores-Mir C. Adenoid Hypertrophy in Pediatric Sleep Disordered Breathing and craniofacial Growth: The Emerging Role of Dentistry. J Dent Sleep Medicine 2014; 1(2): 83-7

JDSM

ORIGINAL ARTICLES

http://dx.doi.org/10.15331/jdsm.3894

Adenoid Hypertrophy in Pediatric Sleep Disordered Breathing and Craniofacial Growth: The Emerging Role of Dentistry

Michael P. Major, DMD, MSc, FRCD(c)[1,2]; Hamdy El-Hakim, FRCS(Ed), FRCS(ORL)[3]; Manisha Witmans, MD, FRCPC, FAASM[1]; Paul W. Major, DDS, MSc, FRCD(c)[1]; Carlos Flores-Mir, DDS, DSc, FRCD(c)[4]

[1]School of Dentistry, [2]Inter-disciplinary Airway Research Clinic (IARC), [3]Pediatric Otolaryngology, and [4]Orthodontics, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, Alberta, Canada

Study Objectives: Summarize and synthesize the most recent evidence about adenoid hypertrophy, impact on craniofacial growth, role in sleep disordered breathing, and effects of treatment.

Methods: Literature review of relevant manuscripts from dentistry, orthodontics, otolaryngology, and sleep medicine.

Results: Adenoid hypertrophy is the most common cause of nasopharyngeal obstruction in children; the most common cause of pediatric sleep disordered breathing (SDB); and can be an etiologic cause of altered craniofacial growth characterized by long face, retrusive chin, and narrow maxilla. Early detection and treatment may mitigate or resolve negative effects of adenoid hypertrophy. Adenoidectomy remains a front line treatment for the majority of cases, although alternative treatments must be considered when different SDB etiologies and co-morbidities are present. Best available evidence suggests that rapid maxillary expansion and adenoidectomy work synergistically to resolve SDB symptoms, and often both treatments are necessary for full treatment effect.

Conclusions: Primary care dentists, pediatric dentists, and orthodontists have an important role in early detection of adenoid hypertrophy. Emerging evidence continues to demonstrate dental treatments as playing an increasingly important role in multidisciplinary management of pediatric SDB.

Keywords: adenoids, adenoidectomy, craniofacial growth, diagnosis, palatal expansion, obstructive sleep apnea, orthodontics, sleep disordered breathing

Citation: Major MP, El Hakim H, Witmans M, Major PW, Flores Mir C. Adenoid hypertrophy in pediatric sleep disordered breathing and craniofacial growth: the emerging role of dentistry. *Journal of Dental Sleep Medicine* 2014;1(2):83–87.

The adenoids are a collection of lymphatic tissue located in the most superior-posterior aspect of the nasopharynx. They are situated at the inflection point between the horizontally oriented nasal passage and the vertically oriented oropharynx. Being a lymphoid tissue, the adenoids play a role in immunity housing large numbers of immunocompetent cells such as B cells, T cells, lymphocytes, and macrophages.[1] As a result, the adenoids are highly prone to inflammation when an immune response is elicited against foreign antigens.[1]

Even in healthy children, a physiologic amount of adenoid enlargement is a part of normal craniofacial growth and development. The adenoid lymphoid tissue naturally increases to its largest size sometime between age 5-10 years, then continually decreases in size until adulthood.[2,3] Since children of this age range naturally have some element of relative lymph enlargement, additional inflammation—actual inflammatory hypertrophy beyond physiologic adenoid enlargement—can introduce partial or complete nasopharyngeal obstruction.[4]

Epidemiologic studies have reported a high prevalence of adenoid hypertrophy in children. One large study of 1,132 subjects observed a frequency of 27% for children between 5 and 7 years, and 19% to 20% for children between the age of 8 and 14 years.[5] Other smaller studies have observed frequencies of 37.9% among 370 children between 3 and 9 years[6] and 57.7% among 213 children between 6 months and 15 years.[7]

When adenoid hypertrophy occurs in a chronic state, there can be long periods of partial or complete impairment of nasal function,[1] which may lead to mouth breathing to overcome the limited passage of air through the nasopharynx.[6] Chronic nasopharyngeal obstruction is believed to increase the risk for altered craniofacial growth and increase the risk of pediatric sleep disordered breathing.

THE EFFECT OF ADENOID HYPERTROPHY

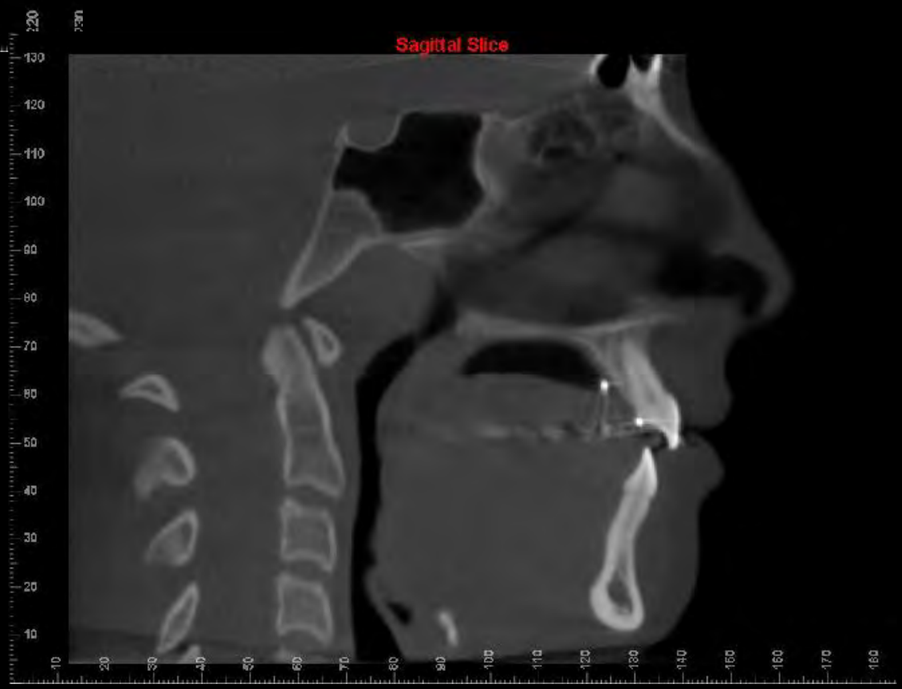
Adenoid Hypertrophy and Altered Craniofacial Growth

Although previous research studied the link between nasal function and facial pattern, it was Linder-Aronson's seminal work that helped solidify the association between adenoid hypertrophy and altered human craniofacial growth. He noted that adenoid obstruction occurred in all facial types, but children with adenoid hypertrophy presented more frequently with a recurrent craniofacial phenotype. This phenotype was characterized by a narrow maxillary dental arch, posterior dental crossbite, steep mandibular plane, and long anterior face height.[8] Such a craniofacial phenotype was often termed "adenoid facies."

Linder-Aronson acknowledged that, in theory, a genetically driven facial pattern could also cause the nasopharyngeal obstruction. However, he favored a hypothesis that nasopharyngeal obstruction—whether by adenoid hypertrophy or other etiology—increased resistance to nasal airflow such that children were obligated to mouth breathe. The resulting open mouth posture became the driving force behind altered craniofacial growth. He theorized that during mouth breathing, the tongue assumed a lower posture to facilitate oral airflow and therefore no longer rested in the palate. Without the tongue providing internal muscular force, transverse maxillary

Paediatric Sleep Disorder Breathing

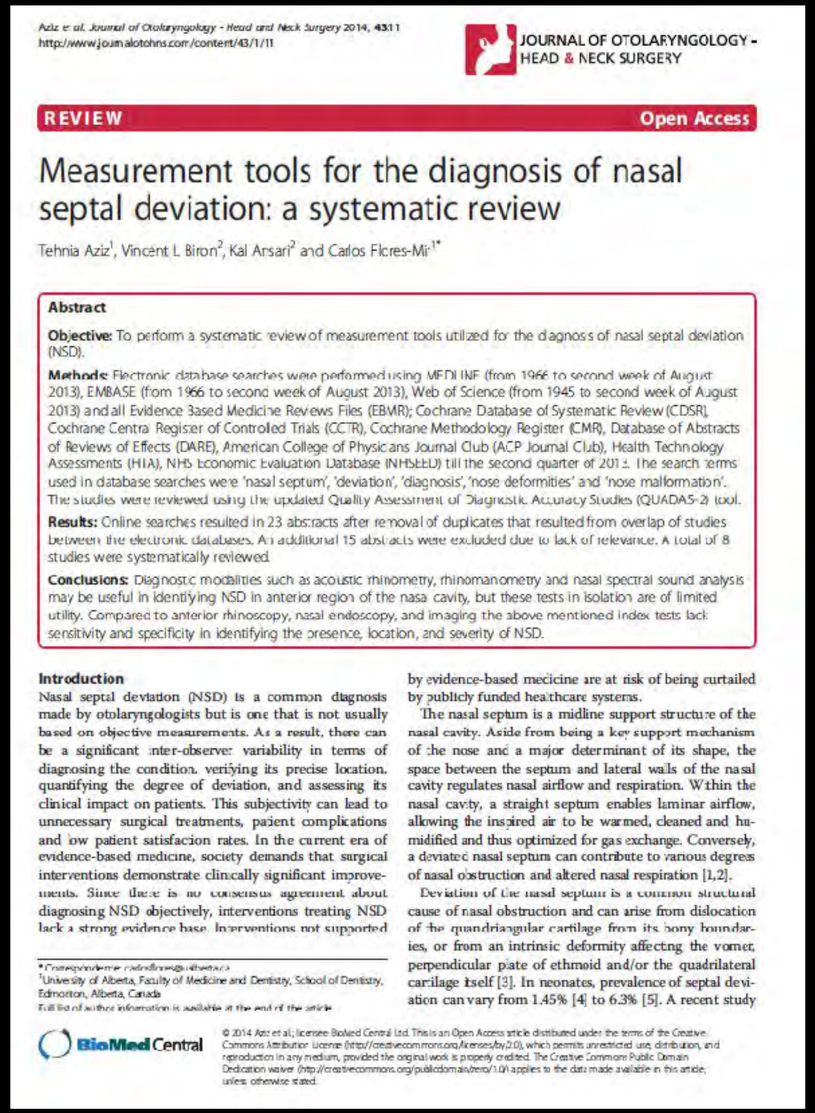
Case #5



Deviated Nasal Septum

Deviated Nasal Septum

Aziz T, Biron VL, Ansari KL, Flores-Mir C. Measurement tools for the diagnosis of nasal septal deviation: a systematic review. J Otolaryngol Head Neck Surg 2014; 43: 11



Aziz et al. Journal of Otolaryngology - Head and Neck Surgery 2014, 43:11
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JOURNAL OF OTOLARYNGOLOGY - HEAD & NECK SURGERY

REVIEW Open Access

Measurement tools for the diagnosis of nasal septal deviation: a systematic review

Tehnia Aziz[1], Vincent L Biron[2], Kal Ansari[2] and Carlos Flores-Mir[1*]

Abstract

Objective: To perform a systematic review of measurement tools utilized for the diagnosis of nasal septal deviation (NSD).

Methods: Electronic database searches were performed using MEDLINE (from 1966 to second week of August 2013), EMBASE (from 1966 to second week of August 2013), Web of Science (from 1945 to second week of August 2013) and all Evidence Based Medicine Reviews Files (EBMR); Cochrane Database of Systematic Review (CDSR), Cochrane Central Register of Controlled Trials (CCTR), Cochrane Methodology Register (CMR), Database of Abstracts of Reviews of Effects (DARE), American College of Physicians Journal Club (ACP Journal Club), Health Technology Assessments (HTA), NHS Economic Evaluation Database (NHSEED) till the second quarter of 2013. The search terms used in database searches were 'nasal septum', 'deviation', 'diagnosis', 'nose deformities' and 'nose malformation'. The studies were reviewed using the updated Quality Assessment of Diagnostic Accuracy Studies (QUADAS-2) tool.

Results: Online searches resulted in 23 abstracts after removal of duplicates that resulted from overlap of studies between the electronic databases. An additional 15 abstracts were excluded due to lack of relevance. A total of 8 studies were systematically reviewed.

Conclusions: Diagnostic modalities such as acoustic rhinometry, rhinomanometry and nasal spectral sound analysis may be useful in identifying NSD in anterior region of the nasal cavity, but these tests in isolation are of limited utility. Compared to anterior rhinoscopy, nasal endoscopy, and imaging the above mentioned index tests lack sensitivity and specificity in identifying the presence, location, and severity of NSD.

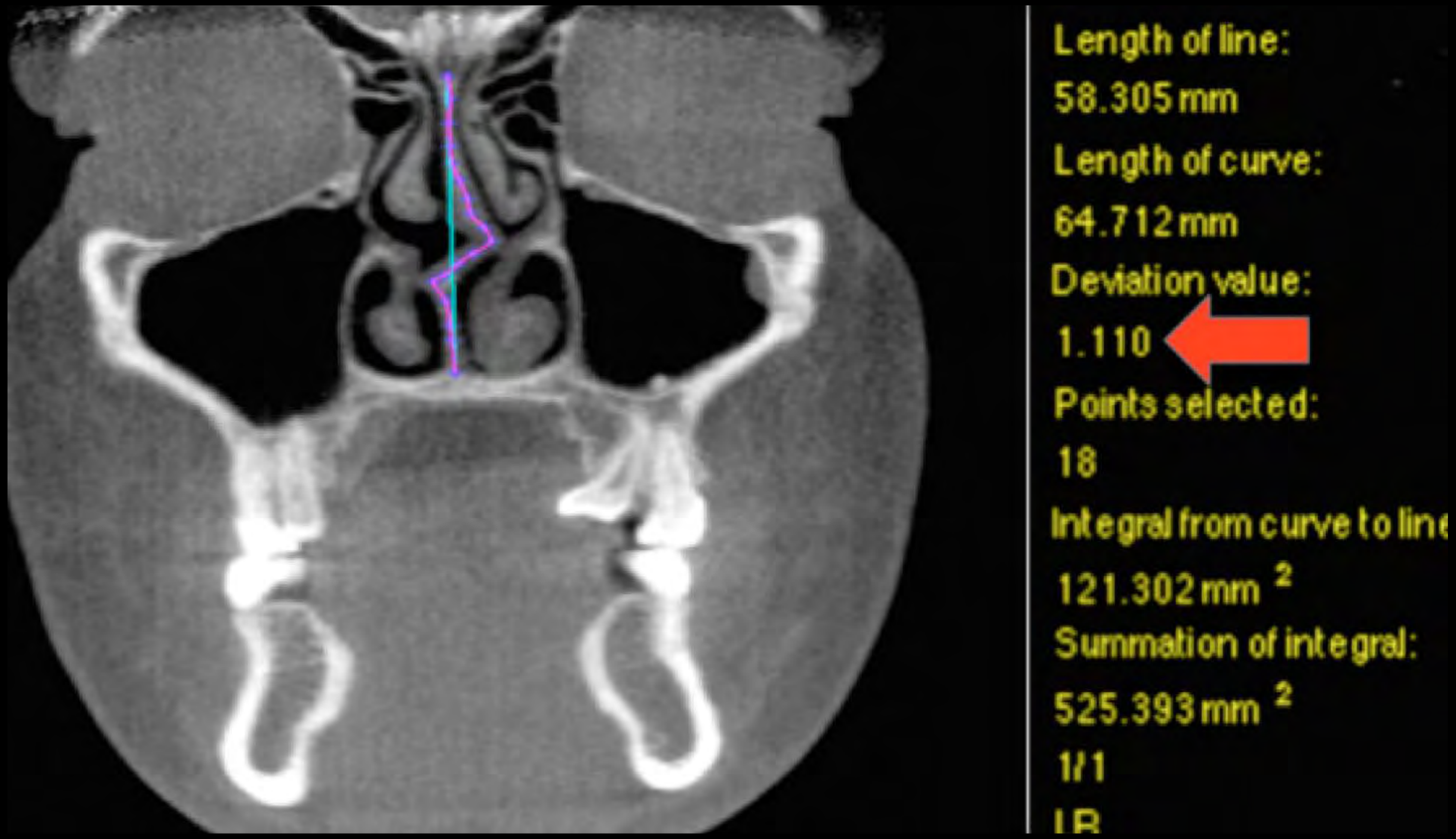
Introduction

Nasal septal deviation (NSD) is a common diagnosis made by otolaryngologists but is one that is not usually based on objective measurements. As a result, there can be a significant inter-observer variability in terms of diagnosing the condition, verifying its precise location, quantifying the degree of deviation, and assessing its clinical impact on patients. This subjectivity can lead to unnecessary surgical treatments, patient complications and low patient satisfaction rates. In the current era of evidence-based medicine, society demands that surgical interventions demonstrate clinically significant improvements. Since there is no consensus agreement about diagnosing NSD objectively, interventions treating NSD lack a strong evidence base. Interventions not supported by evidence-based medicine are at risk of being curtailed by publicly funded healthcare systems.

The nasal septum is a midline support structure of the nasal cavity. Aside from being a key support mechanism of the nose and a major determinant of its shape, the space between the septum and lateral walls of the nasal cavity regulates nasal airflow and respiration. Within the nasal cavity, a straight septum enables laminar airflow, allowing the inspired air to be warmed, cleaned and humidified and thus optimized for gas exchange. Conversely, a deviated nasal septum can contribute to various degrees of nasal obstruction and altered nasal respiration [1,2].

Deviation of the nasal septum is a common structural cause of nasal obstruction and can arise from dislocation of the quandriangular cartilage from its bony boundaries, or from an intrinsic deformity affecting the vomer, perpendicular plate of ethmoid and/or the quadrilateral cartilage itself [3]. In neonates, prevalence of septal deviation can vary from 1.45% [4] to 6.3% [5]. A recent study

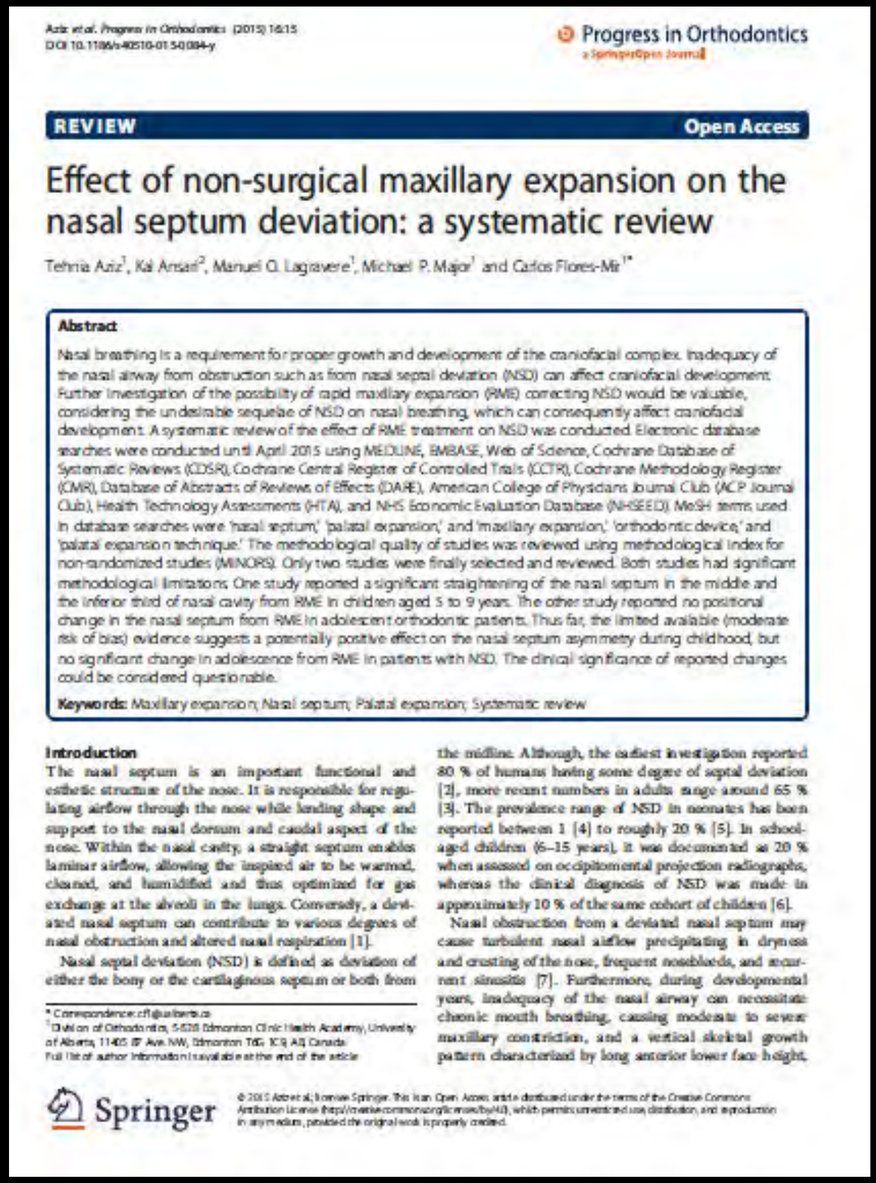
*Correspondence: carlosflores@ualberta.ca
[1]University of Alberta, Faculty of Medicine and Dentistry, School of Dentistry, Edmonton, Alberta, Canada
Full list of author information is available at the end of the article



Deviated Nasal Septum Measurements

Deviated Nasal Septum

Aziz T, Ansari KL, Lagravere MO, Major MP, Flores-Mir C. Effects of Rapid Maxillary Expansion on the Nasal Septum Deviation. PiO 2015; 16: 15.



Aziz et al. Progress in Orthodontics (2015) 16:15
DOI 10.1186/s40510-015-0084-y

Progress in Orthodontics
a SpringerOpen Journal

REVIEW Open Access

Effect of non-surgical maxillary expansion on the nasal septum deviation: a systematic review

Tehnia Aziz[1], Kal Ansari[2], Manuel O. Lagravere[1], Michael P. Major[1] and Carlos Flores-Mir[1*]

Abstract

Nasal breathing is a requirement for proper growth and development of the craniofacial complex. Inadequacy of the nasal airway from obstruction such as from nasal septal deviation (NSD) can affect craniofacial development. Further investigation of the possibility of rapid maxillary expansion (RME) correcting NSD would be valuable, considering the undesirable sequelae of NSD on nasal breathing, which can consequently affect craniofacial development. A systematic review of the effect of RME treatment on NSD was conducted. Electronic database searches were conducted until April 2015 using MEDLINE, EMBASE, Web of Science, Cochrane Database of Systematic Reviews (CDSR), Cochrane Central Register of Controlled Trials (CCTR), Cochrane Methodology Register (CMR), Database of Abstracts of Reviews of Effects (DARE), American College of Physicians Journal Club (ACP Journal Club), Health Technology Assessments (HTA), and NHS Economic Evaluation Database (NHSEED). MeSH terms used in database searches were 'nasal septum,' 'palatal expansion,' and 'maxillary expansion,' 'orthodontic device,' and 'palatal expansion technique.' The methodological quality of studies was reviewed using methodological index for non-randomized studies (MINORS). Only two studies were finally selected and reviewed. Both studies had significant methodological limitations. One study reported a significant straightening of the nasal septum in the middle and the inferior third of nasal cavity from RME in children aged 5 to 9 years. The other study reported no positional change in the nasal septum from RME in adolescent orthodontic patients. Thus far, the limited available (moderate risk of bias) evidence suggests a potentially positive effect on the nasal septum asymmetry during childhood, but no significant change in adolescence from RME in patients with NSD. The clinical significance of reported changes could be considered questionable.

Keywords: Maxillary expansion; Nasal septum; Palatal expansion; Systematic review

Introduction

The nasal septum is an important functional and esthetic structure of the nose. It is responsible for regulating airflow through the nose while lending shape and support to the nasal dorsum and caudal aspect of the nose. Within the nasal cavity, a straight septum enables laminar airflow, allowing the inspired air to be warmed, cleaned, and humidified and thus optimized for gas exchange at the alveoli in the lungs. Conversely, a deviated nasal septum can contribute to various degrees of nasal obstruction and altered nasal respiration [1].

Nasal septal deviation (NSD) is defined as deviation of either the bony or the cartilaginous septum or both from the midline. Although, the earliest investigation reported 80 % of humans having some degree of septal deviation [2], more recent numbers in adults range around 65 % [3]. The prevalence range of NSD in neonates has been reported between 1 [4] to roughly 20 % [5]. In school-aged children (6–15 years), it was documented as 20 % when assessed on occipitomental projection radiographs, whereas the clinical diagnosis of NSD was made in approximately 10 % of the same cohort of children [6].

Nasal obstruction from a deviated nasal septum may cause turbulent nasal airflow precipitating in dryness and crusting of the nose, frequent nosebleeds, and recurrent sinusitis [7]. Furthermore, during developmental years, inadequacy of the nasal airway can necessitate chronic mouth breathing, causing moderate to severe maxillary constriction, and a vertical skeletal growth pattern characterized by long anterior lower face height,

* Correspondence: cf@ualberta.ca
[1]Division of Orthodontics, 5-020 Edmonton Clinic Health Academy, University of Alberta, 11405 87 Ave NW, Edmonton T6G 1C9, AB, Canada
Full list of author information is available at the end of the article

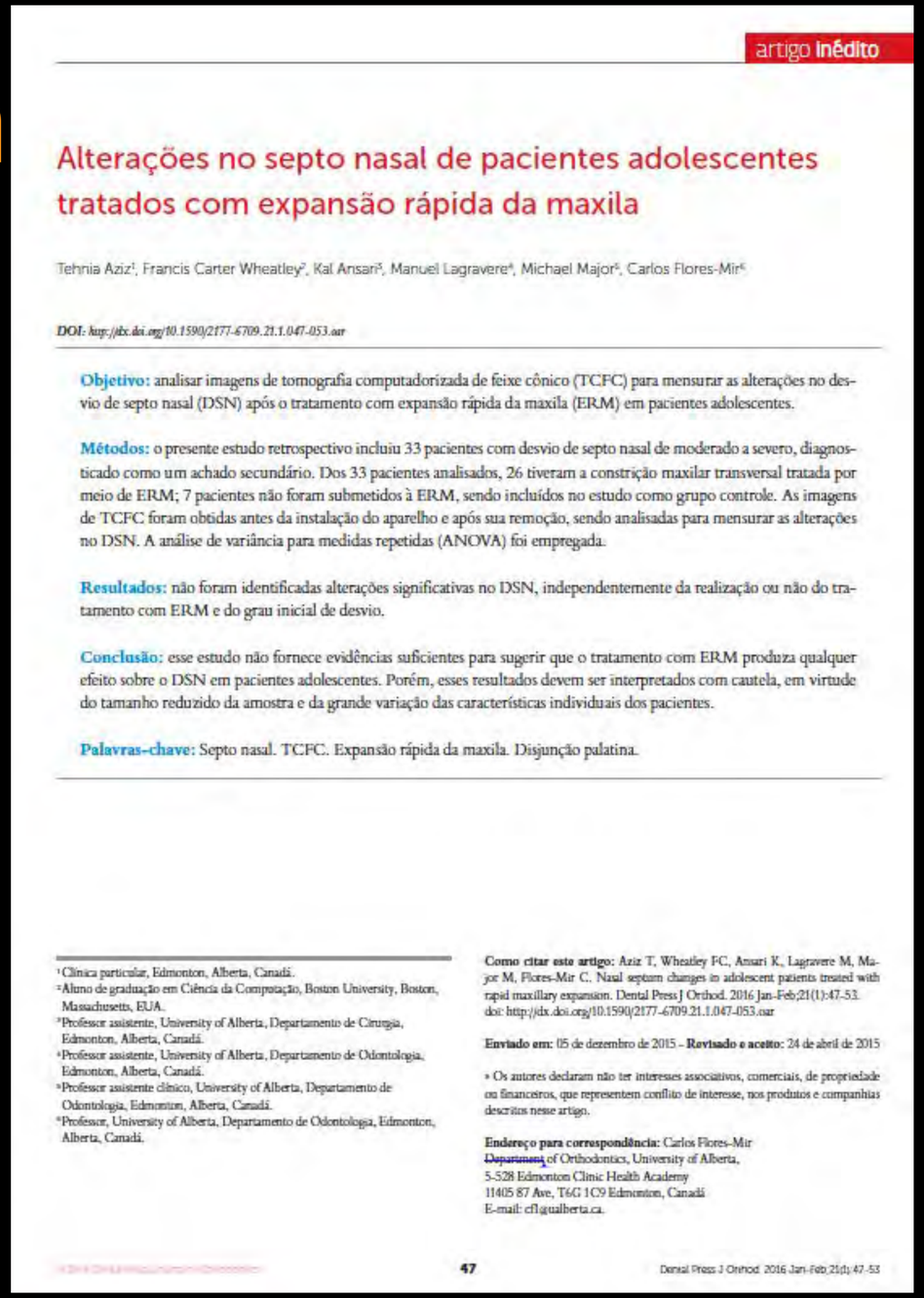
Deviated Nasal Septum

Table 2 Characteristics of included studies

Study	Baseline characteristics of treatment group	Baseline characteristics of control group	RME protocol	Measurement of the nasal septum	Results
Farronato et al. [15]	*N* = 100 Ages 5–9 years (mean = 7.62 years, SD = 0.7) Nasal septal deviation (NSD) of more than 1 mm as seen on PA radiographs (amplitude of deviation)	*N* = 40 Ages 5–9 years (mean = 7.62, SD = 0.7) Not treated with RME Not clear if they presented with NSD	Hyrax expander1 turn (0.25 mm) twice a day for 15 days	Amplitude of NSD measured on frontal/PA cephalograms as millimeter distance between midline axis of symmetry and deviated nasal septum. Measurements taken before appliance insertion (T0), at appliance removal (T1) and 6 months after appliance removal (T2)	94 % reduction in amplitude of NSD from RME in the middle and lower third of the nasal cavity from T0 to T2
Altug-Atac et al. [21]	*N* = 10 Ages 13–17 years (mean = 15 years) Nasal septal angle (from midsagittal plane = 1.05° (SD = 0.91))	*N* = 10 Ages 13–17 years (mean = 15 years) Not treated with RME Nasal septal angle 0.78 (SD = 1.23)	Occlusal coverage, Hyrax type expander with 2 turns a day for 2–3 weeks	Measured in degrees as angle between the nasal septum midsagittal plane on frontal/PA cephalograms. Measurements taken prior to appliance insertion and after 12 weeks active expansion	No significant positional change in nasal septum from RME

Deviated Nasal Septum

Aziz T, Ansari KL, Lagravere MO, Major MP, Flores-Mir C. Nasal Septal Changes in Adolescent Patients treated with Rapid Maxillary Expansion as assessed through Cone Beam Computed Tomography. Dental Press J Orthod. 2016 Jan-Feb;21(1):47-53.



artigo inédito

Alterações no septo nasal de pacientes adolescentes tratados com expansão rápida da maxila

Tehnia Aziz[1], Francis Carter Wheatley[2], Kal Ansari[3], Manuel Lagravere[4], Michael Major[5], Carlos Flores-Mir[6]

DOI: http://dx.doi.org/10.1590/2177-6709.21.1.047-053.oar

Objetivo: analisar imagens de tomografia computadorizada de feixe cônico (TCFC) para mensurar as alterações no desvio de septo nasal (DSN) após o tratamento com expansão rápida da maxila (ERM) em pacientes adolescentes.

Métodos: o presente estudo retrospectivo incluiu 33 pacientes com desvio de septo nasal de moderado a severo, diagnosticado como um achado secundário. Dos 33 pacientes analisados, 26 tiveram a constrição maxilar transversal tratada por meio de ERM; 7 pacientes não foram submetidos à ERM, sendo incluídos no estudo como grupo controle. As imagens de TCFC foram obtidas antes da instalação do aparelho e após sua remoção, sendo analisadas para mensurar as alterações no DSN. A análise de variância para medidas repetidas (ANOVA) foi empregada.

Resultados: não foram identificadas alterações significativas no DSN, independentemente da realização ou não do tratamento com ERM e do grau inicial de desvio.

Conclusão: esse estudo não fornece evidências suficientes para sugerir que o tratamento com ERM produza qualquer efeito sobre o DSN em pacientes adolescentes. Porém, esses resultados devem ser interpretados com cautela, em virtude do tamanho reduzido da amostra e da grande variação das características individuais dos pacientes.

Palavras-chave: Septo nasal. TCFC. Expansão rápida da maxila. Disjunção palatina.

[1] Clínica particular, Edmonton, Alberta, Canadá.
[2] Aluno de graduação em Ciência da Computação, Boston University, Boston, Massachusetts, EUA.
[3] Professor assistente, University of Alberta, Departamento de Cirurgia, Edmonton, Alberta, Canadá.
[4] Professor assistente, University of Alberta, Departamento de Odontologia, Edmonton, Alberta, Canadá.
[5] Professor assistente clínico, University of Alberta, Departamento de Odontologia, Edmonton, Alberta, Canadá.
[6] Professor, University of Alberta, Departamento de Odontologia, Edmonton, Alberta, Canadá.

Como citar este artigo: Aziz T, Wheatley FC, Ansari K, Lagravere M, Major M, Flores-Mir C. Nasal septum changes in adolescent patients treated with rapid maxillary expansion. Dental Press J Orthod. 2016 Jan-Feb;21(1):47-53. doi: http://dx.doi.org/10.1590/2177-6709.21.1.047-053.oar

Enviado em: 05 de dezembro de 2015 - **Revisado e aceito:** 24 de abril de 2015

» Os autores declaram não ter interesses associativos, comerciais, de propriedade ou financeiros, que representem conflito de interesse, nos produtos e companhias descritos nesse artigo.

Endereço para correspondência: Carlos Flores-Mir
Department of Orthodontics, University of Alberta,
5-528 Edmonton Clinic Health Academy
11405 87 Ave, T6G 1C9 Edmonton, Canadá
E-mail: cfl@ualberta.ca.

Deviated Nasal Septum

Retrospective study 26 patients presenting with mild to severe septal deviation as an incidental finding and treated for transverse maxillary constriction with RME.

A control group of 7 patients was also included without RME treatment and presenting with mild to severe baseline deviation.

CBCT images were taken at T1 (before appliance insertion) and T3 (after appliance removal) and were analyzed to measure changes in nasal septal deviation.

Repeated measures ANOVA resulted in no significant changes in NSD with or without RME treatment and irrespective of baseline deviation.

Deviated Nasal Septum

Ballast F et al. Is there a correlation between nasal septum deviation and maxillary transversal deficiency? A retrospective study on prepubertal subjects.
Int J Ped Otorhinolaryng. 2016; 83: 109-12.

International Journal of Pediatric Otorhinolaryngology 83 (2016) 109–112

Contents lists available at ScienceDirect

International Journal of Pediatric Otorhinolaryngology

journal homepage: www.elsevier.com/locate/ijporl

Is there a correlation between nasal septum deviation and maxillary transversal deficiency? A retrospective study on prepubertal subjects

Fabiana Ballanti, Alberto Baldini *, Salvatore Ranieri, Alessandro Nota, Paola Cozza

Department of Orthodontics, University of Rome Tor Vergata, Viale Oxford, 81, 00133 Rome, Italy

ARTICLE INFO

Article history:
Received 24 November 2015
Received in revised form 26 January 2016
Accepted 31 January 2016
Available online 6 February 2016

Keywords:
Nasal septum
Maxillary transverse deficiency
Nasal cavities
Cephalometry

ABSTRACT

Introduction: Deviated nasal septum may cause a reduction of the nasal airflow, thus, during the craniofacial development, a reduced nasal airflow could originate a chronic mouth-breathing pattern, related with moderate to severe maxillary constriction. The aim of this retrospective study is to analyze the correlation between maxillary transverse deficiency and nasal septum deviation.
Methods: Frontal cephalograms were performed on 66 posterior–anterior radiographs of subjects (34M, 32F; mean age 9.95 ± 2.50 years) with maxillary transverse deficiency and on a control group of 31 posterior–anterior radiographs of subjects (13M, 18F; 9.29 ± 2.08 years). Angular parameters of the nasal cavities were recorded and compared between the two groups using a Student's *t*-test.
Results: Generally all the parameters are very similar between the two groups except for the ASY angle that differs for about the 27%; anyway the Student's *t*-test showed no statistically significant differences between the two groups (mostly $p > 0.20$).
Conclusions: This study failed to show an association between transverse maxillary deficiencies and nasal septum deviations. Moreover, no significant differences were found between the mean nasal cavities dimensions in subjects with transverse maxillary deficiency and the control group.

1. Introduction

The septal cartilage is a perpendicular plate with a quadrangular shape that extends from the nasal bone to the bony septum. Two triangular or trapezoidal shaped cartilages flank its upper half fusing with the dorsal septum in the midline.

The nasal septum is made up of a vertical lamina that includes two bony parts and a cartilage that is generally slightly deflected respect to its axis. The primitive nasal cavities and the primitive nasal septum are in communication with the oral cavity until the 8th week with the formation of the posterior palate [1].

For these reasons the maxillary bone and the nasal structures have an important anatomical connection starting from their initial growth phases. Moreover, the septal cartilage is rather a center than an area of growth; it deviation in the first years of life, can determine a distortion of the maxillary bone with a dislocation of the septum toward the deviated site [2,3]. Septal deviations could be related to childhood traumas or to a disharmonic development of the near structures (ethmoid bone, nasal bone, vomer bone, maxillary bone). This disharmonic growth could be related with an excessive compression of the nasal and maxillary structures given by an incorrect position of the fetus. This compression is often asymmetric and this can determine in the maxillary bone, palatal deformities or modification of one of the nasal cavities floor.

Maxillary transverse constriction is one of the most frequent skeletal deformities in the craniofacial region [4]. This deformity usually causes unilateral or bilateral posterior crossbites, dental crowding, high palatal vault, elevation of the nasal floor and mouth breathing [5]. Some studies correlate the maxillary transverse deficiency with small nasal cross sectional areas [6,7]. It was published that skeletally expanding the maxillary bone brings to an improvement of the breathing pattern [8,9] and a reduction of the nasal airway resistance increasing significantly the lower nasal volume, nasal floor width and nasal lateral width [10–12]. Some studies showed that subjects with cleft palate have a nasal septum deviation toward the cleft side [13,14]. In the scientific literature it

* Corresponding author at: Centro Medico Polispecialistico Baldini Srl, Via S. Orsola, n. 5, Bergamo 24122, Italy. Tel.: +39 035 271935; fax: +39 035 694280.
E-mail addresses: fabianaballanti@gmail.com (F. Ballanti), studiomedicobaldini@gmail.com (A. Baldini), salvatore.ranieri87@gmail.com (S. Ranieri), dr.alessandro.nota@gmail.com (A. Nota), paolacozza@alice.it (P. Cozza).

http://dx.doi.org/10.1016/j.ijporl.2015.01.036
0165-5876/

Deviated Nasal Septum

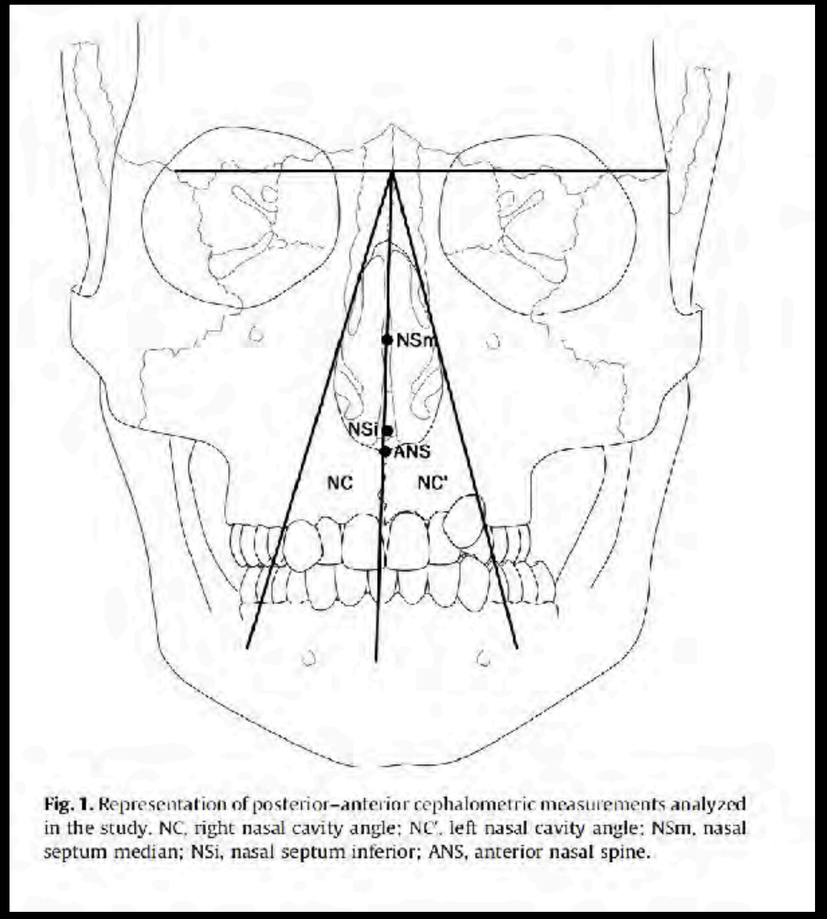


Fig. 1. Representation of posterior–anterior cephalometric measurements analyzed in the study. NC, right nasal cavity angle; NC', left nasal cavity angle; NSm, nasal septum median; NSi, nasal septum inferior; ANS, anterior nasal spine.

Conclusions: This study failed to show an association between transverse maxillary deficiencies and nasal septum deviations. Moreover, no significant differences were found between the mean nasal cavities dimensions in subjects with transverse maxillary deficiency and the control group.

Deviated Nasal Septum

Effects of Orthopedic Rapid Maxillary Expansion on Nasal Septum Deviation

Evren Öztaş, Gamze Öney*

Istanbul University, Faculty of Dentistry, Department of Orthodontics, Istanbul, Turkiye

STOCKHOLM - 11-16 JUNE 2016
92nd CONGRESS

AIM: The aim of this study was to investigate and evaluate the effects of orthopedic rapid maxillary expansion on nasomaxillary complex and nasal septum deviation.

SUBJECTS AND METHOD: Nine adolescent patients with skeletal maxillary transverse constriction, presenting a septal deviation more than 1 mm were included in the study. All patients were treated with bonded type rapid maxillary expansion device for a period of 15 days. The protocol of activation consisted by activation of the transverse screw one-quarter turn twice a day. Then the appliance was left in place for five months for passive retention. Postero anterior cephalometric radiographs taken before the treatment (T1) and after retention period (T2) were analyzed. A total of 13 transversal cephalometric variables including maxillo-mandibular dentoalveolar structures, skeletal bases and nasal structures were examined.

Table 1. Cephalometric Definitions

X	Crossing point between the perpendicular plate of the ethmoid and the projection of the floor of the anterior cranial fossa floor
SNM	Middle nasal septum
SNI	Inferior point of nasal septum
Mx	Crossing point of the maxillary tuber and the zygomatic arch
CVM	Most prominent point of the vestibular mesial cuspid -upper left and right
Axis of reference	Axis that passes through Ict and IcR
Axis of symmetry	Perpendicular of the axis of reference that passes through point x
X-SNM	The distance between the points x and snm
SNM-SNAC	The distance between snm and snac
SNM-mid //SNI-mid	The distance between snm and sni from the axis of symmetry
Mx l-r	Basal maxillary width
pir l-r	The width of piriform hiatus
CVM l-r	Most prominent point of the vestibular mesial cuspid between left and right 3. molar
U midline -axis	The distance of the upper midline point to the axis of symmetry
L midline -axis	The distance of the lower midline point to the axis of symmetry

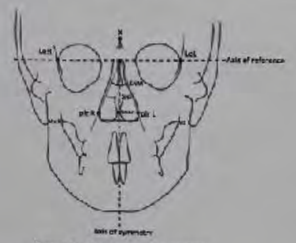
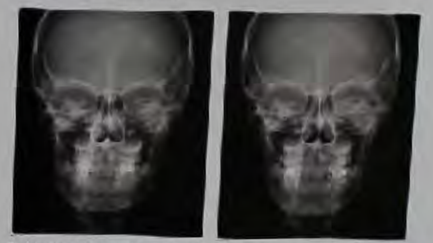


Figure 1. Cephalometric points

Table 2. Skeletal and dentoalveolar changes before (T1) and after (T2) RME

	T1		T2		P	
	mean	S.D.	mean	S.D.	T1	T2
X-SNM	24,78	±4,09	20,74	±10,97	0,916	
SNM-SNAC	29,99	±10,96	30,31	±11,70		0,233
SNM-mid	1,58	±,83	1,39	±,75		0,059
SNI-mid	1,06	±,95	0,81	±,98		0,066
Mx l-r	67	±3,27	68,5	±3,32	,008**	
CVM sl-r	56,89	±3,50	58,78	±2,73	,007**	
CVM il-r	51,11	±2,92	52,5	±2,78	,021*	
Pir l-r	33,44	±2,70	34,83	±2,83	,011*	
CVM sl-r-axis	0,58	±,31	0,48	±,17	0,206	
CVM il-r-axis	0,54	1,57	0,28	±,11	0,066	
Mx l-r-axis	0,72	±,30	0,43	±,30	0,063	
U midline -axis	1,44	±1,05	1,11	±1,15	0,161	
L midline-axis	0,96	±,95	1,49	±1,15	0,343	

Wilcoxon test (*p<0,05 ; **p<0,01)



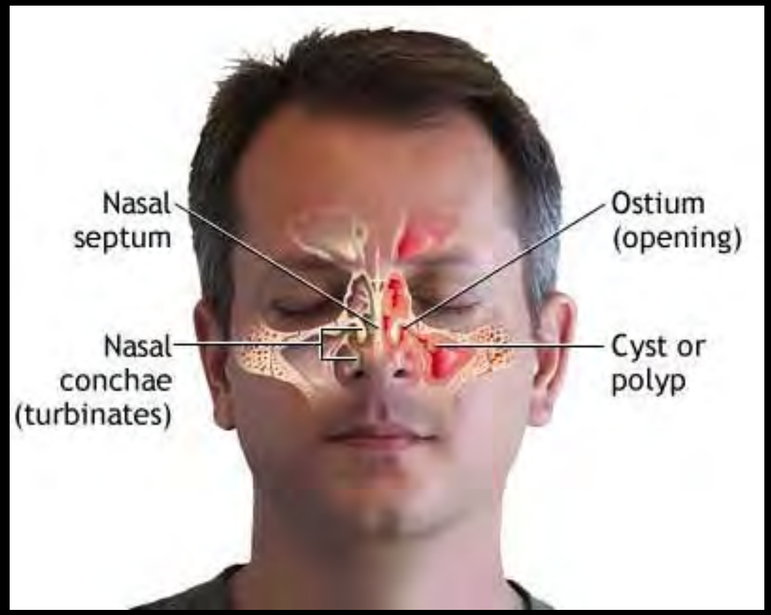
Figures 2 and 3. Cephalometric radiographs before (T1) and after (T2) RME

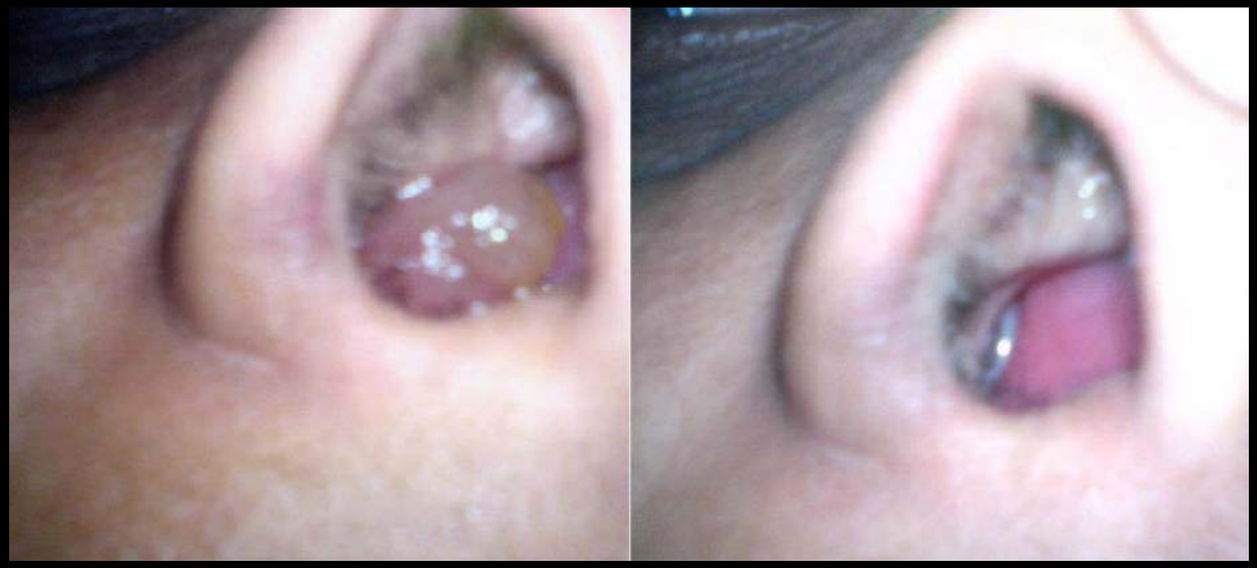
RESULTS: Significant transverse measurement increases were found at skeletal base of the maxilla, maxillary and mandibular dentoalveolar structures and at the base of the nose between T1-T2 period. The nasal septum deviation showed no significant changes even the base of noise has enlarged significantly.

DISCUSSION AND CONCLUSION: Nasal breathing is required for normal growth and development of the craniofacial complex and nasal airway obstructions such as nasal septum deviation can affect craniofacial development. Within the limitations of this study it can be concluded that even the bonded type rapid maxillary expansion device created expansion at the skeletal base of the maxilla and the nose, no positional changes of the nasal septum were found.

No changes in NSD.

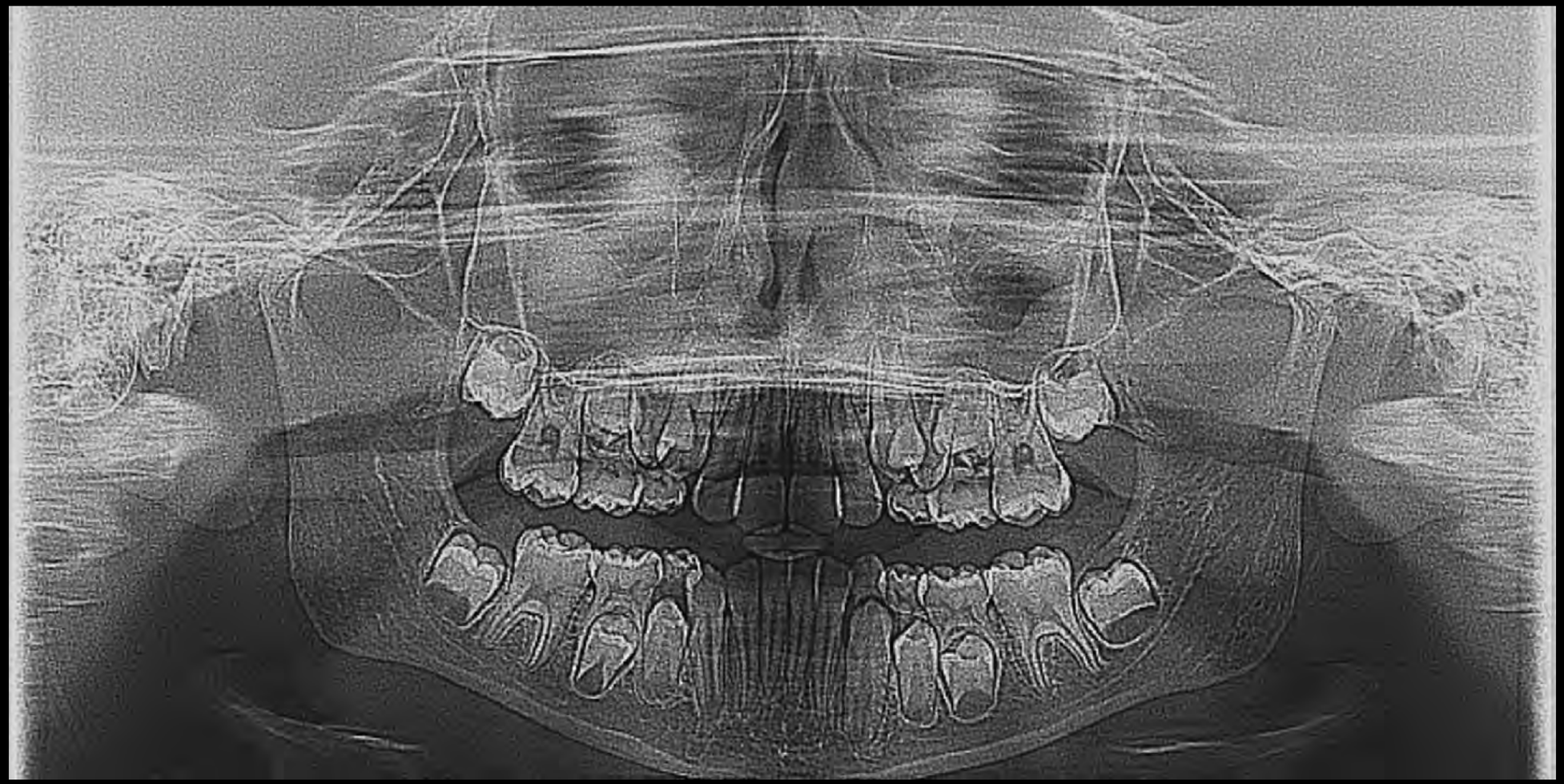
Enlarged Nasal Polyp



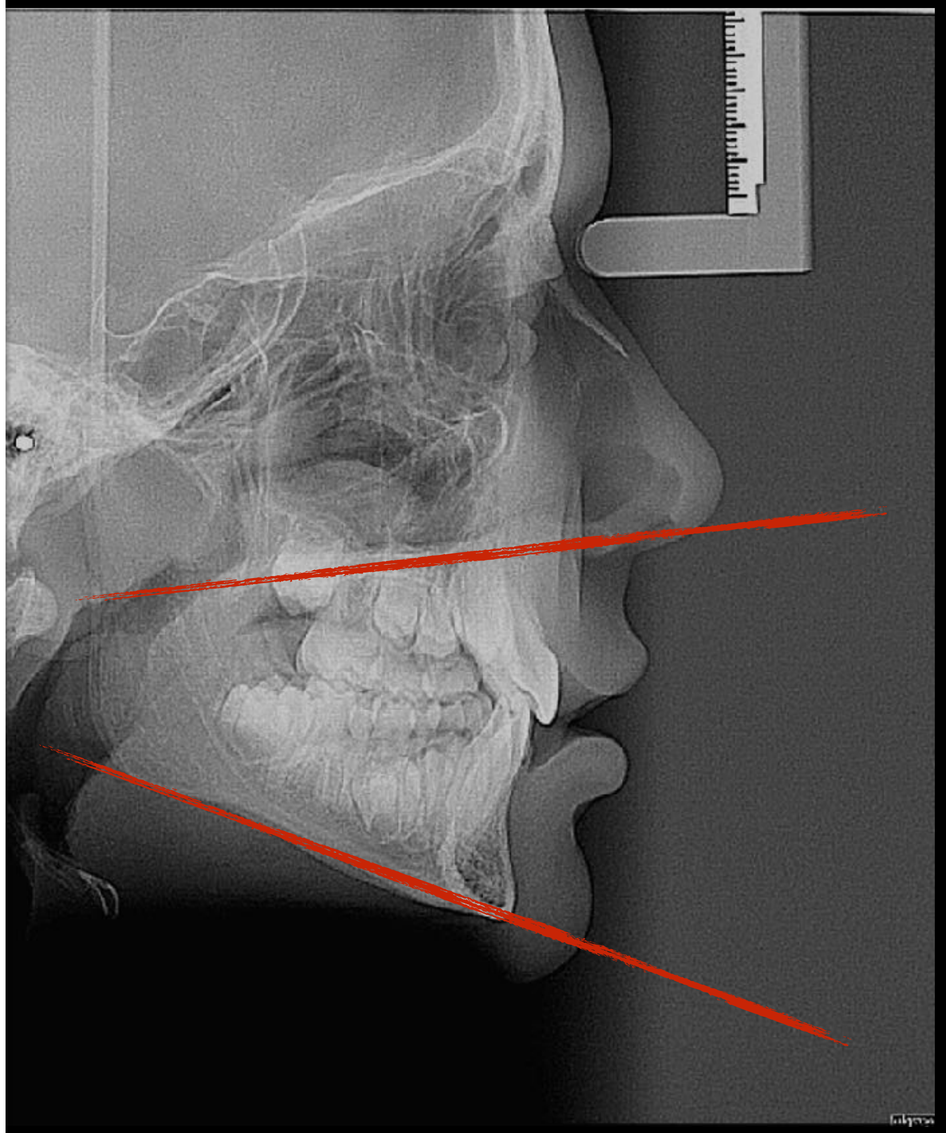


Paediatric Sleep Disorder Breathing

Case #6



FH - SN (∫)	12.4	6.0	4.0	1.6	*
SNA (∫)	74.2	82.0	3.5	-2.2	**
SNB (∫)	72.4	80.9	3.4	-2.5	**
ANB (∫)	1.7	1.6	1.5	0.1	
Mx Unit Length (Co-ANS)	75.1	90.0	5.0	-3.0	***
Md Unit Length (Co-Pog)	95.1	113.0	8.0	-2.2	**
Harvold (CoPog)-(CoANS)	20.0	20.0	3.0	-0.0	
Maxillary Skeletal (A-Na Perp) (mm)	-3.3	0.0	3.1	-1.1	*
Mand. Skeletal (Pg-Na Perp) (mm)	-7.0	-4.0	5.3	-0.6	
MP - SN (∫)	37.5	33.0	6.0	0.8	
U1 - Palatal Plane (∫)	100.9	110.0	5.0	-1.8	*
U1 - SN (∫)	92.4	102.3	5.5	-1.8	*
U1 - ANS (mm)	28.9	30.0	5.0	-0.2	
U6 - PP (UPDH) (mm)	19.5	19.0	2.0	0.3	
L1 - MP (LADH) (mm)	32.1	38.0	2.0	-3.0	***
U1-Me (mm)	4.4	33.0	2.0	-14.3	******
L6 - MP (LPDH) (mm)	22.3	29.0	2.0	-3.3	***
N-Me (mm)	105.8	110.0	6.0	-0.7	
UFH (Na-ANS) (%)	45.1	46.0	5.0	-0.2	
LFH (ANS-Me || FH) (%)	54.9	54.0	5.0	0.2	
Face Ht Ratio (N-A/A-Gn) (%)	114.2	N/A	N/A	N/A	



Pediatric Sleep Questionnaire

(Screening)

Name of the child:

Person completing t

Date that you are co

Instructions: Please answer the questions about how your child **IN THE PAST MONTH**. Circle the correct response or *print* your answers in the space provided. "Y" means "yes," "N" means "no," and "DK" means "don't know." For this questionnaire, the word "usually" means "more than half the time" or "on more than half the nights."

Please answer the following questions as they pertain to your child in the past month.

		YES	NO	Don't Know
1.	**While sleeping, does your child:**			
	Snore more than half the time?	Y	N	DK
	Always snore?	Y	N	DK
	Snore loudly?	Y	N	DK
	Have "heavy" or loud breathing? Heavy & Loud	Y	N	DK
	Have trouble breathing, or struggle to breath?	Y	N	DK
2.	**Have you ever seen your child stop breathing during the night?**	Y	N	DK
3.	**Does your child:**			
	Tend to breathe through the mouth during the day? Usually	Y	N	DK
	Have a dry mouth on waking up in the morning? Usually	Y	N	DK
	Occasionally wet the bed?	Y	N	DK
4.	**Does your child:**			
	Wake up feeling unrefreshed in the morning?	Y	N	DK
	Have a problem with sleepiness during the day? Usually	Y	N	DK
5.	**Has a teacher or other supervisor commented that your child appears sleepy during the day?**	Y	N	DK
6.	**Is it hard to wake your child up in the morning?**	Y	N	DK
7.	**Does your child wake up with headaches in the morning?**	Y	N	DK
8.	**Did your child stop growing at a normal rate at any time since birth?**	Y	N	DK
9.	**Is your child overweight?**	Y	N	DK
10.	**This child often:**			
	Does not seem to listen when spoken to directly	Y	N	DK
	Has difficulty organizing tasks and activities	Y	N	DK
	Is easily distracted by extraneous stimuli	Y	N	DK
	Fidgets with hands or feet, or squirms in seat	Y	N	DK
	Is "on the go" or often acts as if "driven by a motor"	Y	N	DK
	Interrupts or intrudes on others (eg butts into conversations or games)	Y	N	DK

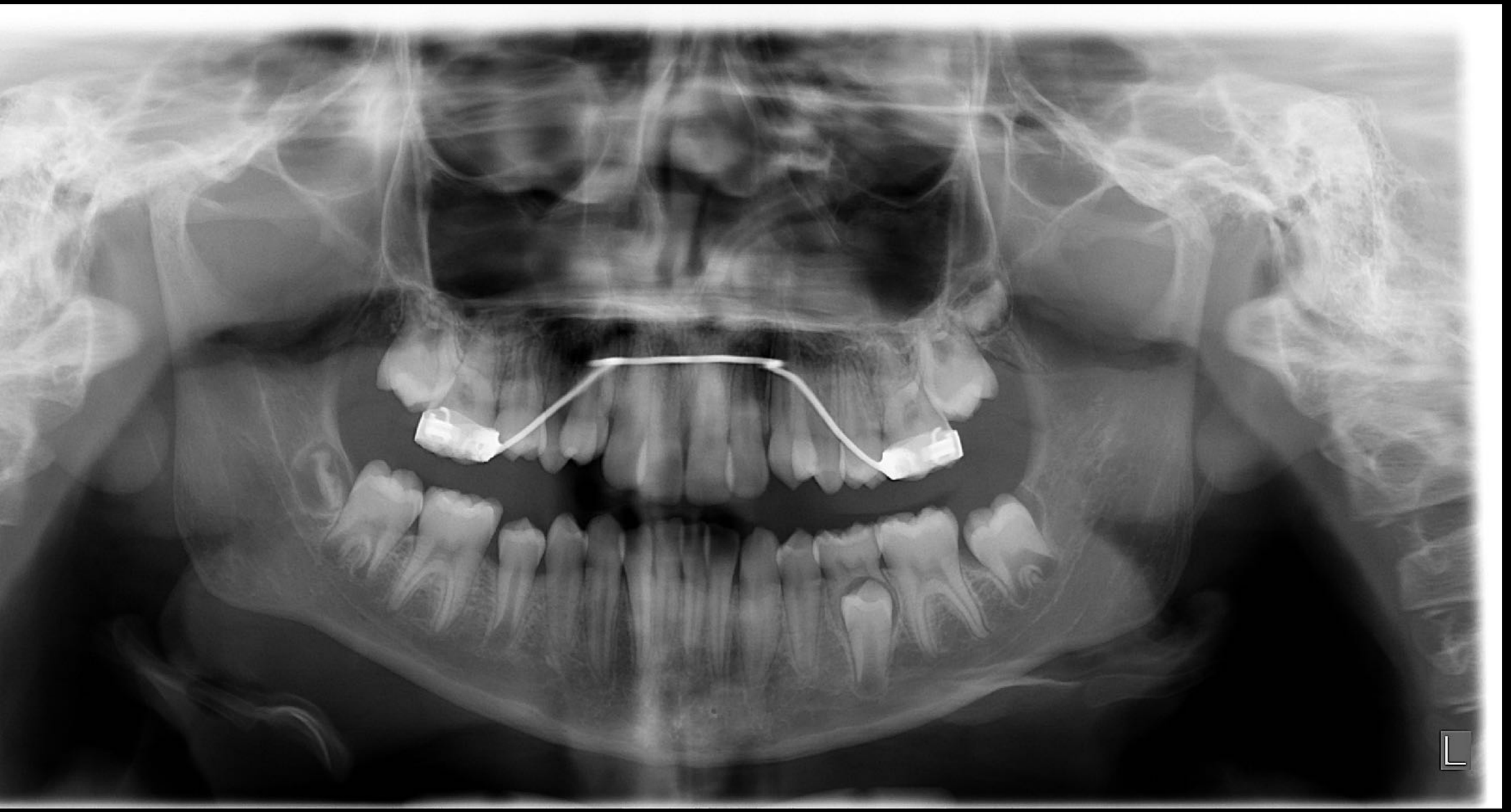
Paediatric Obstructive Sleep Apnea

ENT referral

Adenotonsillectomy planned Jan 15

RME Sep 14

Cancelled AT Jan 15 no more signs and symptoms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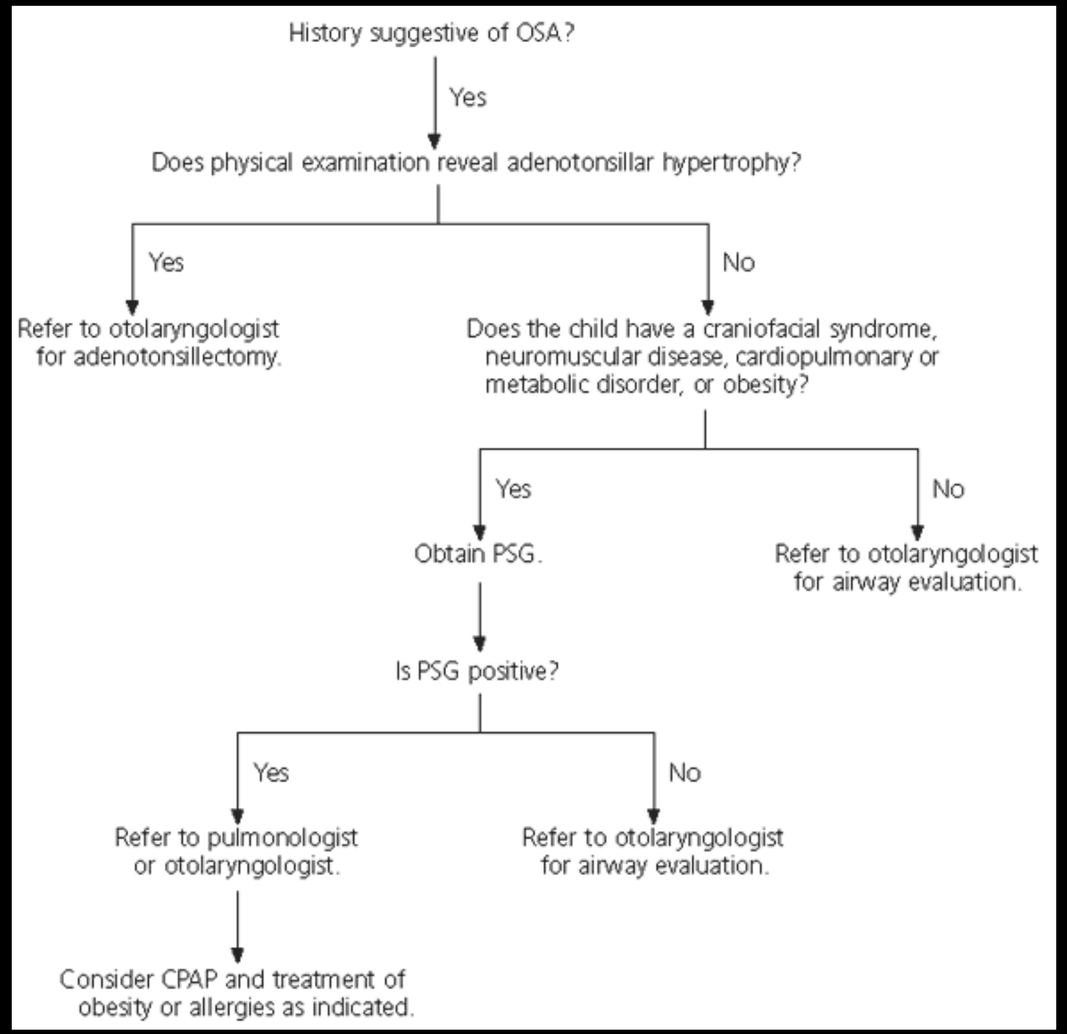
Obstructive Sleep Apnea

Treatment



Take home message

Not all POSA cases are Class II Div 1 cases with open bite.

Not all cases have problems localized in the oropharynx.

Take home message

Medical history is more important than any imaging.

Referral to ENT first, and sometimes then to paediatric sleep medicine is the best dx/tx route

Questions?

carlosflores@ualberta.ca