

OBSTRUCTIVE SLEEP APNEA IN CHILDREN

By Maya Capua

What is Obstructive Sleep Apnea?

Obstructive sleep apnea (OSA) is a temporary suspension of breathing during sleep characterized by repeated episodes of airway obstructions resulting in pauses in breathing. OSA is measured

during a polysomnography and quantified using the Apnea Hypopnea Index (AHI) – the number of times per hour these apneic events occur. In children, an AHI of 1 can be clinically significant, while in adults OSA is characterised by an AHI greater than 5. This is a condition that has been given much attention in the adult population. However, the effects on children are more elusive and understudied.

What is Adenotonsillar Hypertrophy?

Adenotonsillar hypertrophy, also known as

“enlarged tonsils and/or adenoids”, is one of the main causes of OSA in children and may even occur in infants (under 18 months old). Tonsils are small oral masses of lymphoid tissue embedded in the lateral walls of the opening between the mouth and the pharynx and although their exact function is uncertain, they are believed to play a role in the immune system. Adenoids are masses of lymphoid tissue situated at the very back of the nose, in the roof of the nasopharynx where the nose blends into the mouth. The adenoids play a role in the immune system.

Who gets OSA?

The profile of children presenting with OSA is changing from underweight children with adenotonsillar hypertrophy to overweight children. This is consistent with a recent article which noted that increased tonsil size in obese children

suggested a soft tissue change and fat deposition in the upper airway which could possibly play a role in the global differences in adenotonsillar size among obese and non-obese children with OSA. This suggests that the “poster child” for OSA can be any child, any size, any age, making the diagnosis even more challenging. Further, several studies have found that boys are more likely to develop OSA than females. This is consistent with the tendency for overweight adult males to have OSA.

What is the relationship between OSA and Enlarged Tonsils?

Firstly, tonsils and adenoids represent the most common area of hypertrophy that contributes to airway obstruction in children. Further, one study found that tonsil size was directly linked to a higher AHI. Another study found that once children who had upper airway obstructions underwent adenotonsillectomies, almost all of their symptoms were alleviated, except for bed-wetting. The authors concluded the article by strongly suggesting that all children with enlarged tonsils be considered for sleep studies and possibly adenotonsillectomies. In a recent study done in our lab we found that children with enlarged tonsils were over 40 times more likely to develop OSA when compared to a control population. However, we found that there was no correlation between tonsil size and OSA severity.

What are the signs and symptoms of OSA?

Symptoms of OSA include snoring, pauses in breathing while asleep, restless sleep, bizarre sleeping positions, paradoxical chest movements, cyanosis, bedwetting, hyperactivity, stunted growth, and disruptive behaviour in school. In adults, OSA is linked to hypertension, decreased job performance, and increased rate of automotive accidents. In the recent study done in our lab, we found no relationship between typical subjective measures of sleepiness and OSA. The implications of this finding are that in children we cannot rely on typical questionnaires assessing daytime sleepiness and fatigue as we do in adults, because in children these symptoms may be masked or unrecognizable.



Program Director: Colin Shapiro
Medical Director: Joseph Barbera
Lab Manager: Dragana Jovanovic

**Youthdale Child
and Adolescent
Sleep Centre**

227 Victoria St.
Lower Level 2
Toronto, Ontario
M5B 1T8
Phone: 416-703-0505
Fax: 416-703-0507
www.sleepontario.com
or youthdalesleep.com



Overview of Obstructive Sleep Apnea in Children: Exploring the Role of Dentists in Diagnosis and Treatment

Maya Capua, BSc; Negar Ahmadi, BSc; Colin Shapiro, MBChB, PhD, MRC Psych, FRCPC

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Abstract

Among the many factors important in children's development is sleep. Sleep disorders can impair children's sleep and lead to negative consequences. Obstructive sleep apnea (OSA), which involves blockage of the airway during sleep, can affect development and behaviour; thus, OSA in children should be diagnosed and treated at an early stage. One of the main causes of childhood OSA is enlargement of the tonsil tissues and, in most cases, their removal serves as an ultimate treatment of OSA. However, it remains unclear what proportion of children with enlarged tonsil tissue suffer from OSA. Dentists are becoming increasingly aware of the issue of OSA as they are sometimes involved in treatment of this condition using oral appliances. Moreover, as dentists often look into children's mouths, they can play an active role in identifying those with enlarged tonsils and referring them for sleep assessment.

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Continued from page 1

What are the potential consequences of untreated OSA?

The consequences of OSA include failure to thrive, enuresis, attention deficit disorder, inattention, impaired visual perception, impaired working memory, behaviour problems, moodiness, aggressiveness, decreased academic performance, daytime sleepiness, and cardiopulmonary disease. It was also shown in one study that children with sleep disordered breathing were found to have significantly lower mean scores on IQ-like tests. In addition, children with sleep disordered breathing scored significantly lower than the controls on a test of phonological processing (the distribution and patterning of speech sounds in a language and of the rules governing pronunciation). This test measures phonological awareness, a skill that is very important for literacy. Lastly, this study found that the total arousal index was negatively correlated with neurocognitive abilities, suggesting a role for sleep fragmentation in Sleep Disordered Breathing-induced cognitive dysfunction in children.

Impact of orthodontic appliances on sleep quality

Akbar Rawji, Larry Parker, Prativa Deb, Donald Woodside, Bryan Tompson, and Colin M. Shapiro

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Abstract

Introduction: Many young patients are asked to wear headgear or functional appliances during sleep as part of their orthodontic treatment. The objective of this clinical study was to assess the impact of these appliances on sleep quality.

Methods: Twenty-two subjects (8 boys, 14 girls) between 10 and 15 years of age wearing either a headgear or a removable appliance for 3 to 12 months were enrolled in a sleep study. Each subject participated in 2 overnight sleep studies with and without the appliance. There was a 1-week interval after an adaptation night. The studies were performed at the Sleep Research Laboratory at Toronto Western Hospital in Canada. Each subject served as his or her own control. Twelve subjects had an extraoral headgear appliance, and 10 had intraoral functional (5 Fränkel, 5 Twin-block) appliances. No subject reported any sleep or medical disorder. The primary outcome variable was sleep efficiency.

Results: Sleep efficiencies were 90.7% (SD, 7.9) and 91.6% (SD, 4.3) with and without the appliances, respectively. When sleep quality was compared with and without appliances, analysis of variance (ANOVA) showed no significant difference in sleep efficiency or other sleep variables such as sleep onset latency, rapid eye movement onset latency, rapid eye movement percentage, slow-wave sleep percentage, or respiratory disturbance index. Sex and body mass index were not confounding variables. In addition, the use of extraoral or intraoral appliances did not affect sleep quality.

Conclusions: In young orthodontic patients, there appears to be no difference in sleep quality with or without the overnight use of these appliances after they have been worn for a minimum of 3 months.

DR. AKBAR RAWJI



Dr. Akbar Rawji is a staff orthodontist at Bloorview Kids Rehab and an active member of the Craniofacial and Cleft Palate team. He is a member of the American, Canadian and Ontario Association of Orthodontists and the American Cleft Palate-Craniofacial Association. Dr. Rawji's research interest is in orthodontic appliances and their effect on sleep quality. He has presented at several meetings and recently published his research in the American Journal of Orthodontics and Dentofacial Orthopaedics. He also enjoys teaching dental residents at Bloorview and provides occasional seminars in the Graduate Orthodontic Program at the University of Toronto. Dr. Rawji maintains a private practice in Bowmanville.

Dr. Rawji completed specialty training including defense of his Masters Thesis and Fellowship examination with the Royal College of Dentists of Canada in 2003. Following graduation, he completed a one-year Fellowship in the Craniofacial and Cleft Lip and Palate Program at Sick Kids Hospital in Toronto.

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Obstructive Sleep Apnea in Children: Relative Contributions of Body Mass Index and Adenotonsillar Hypertrophy

Dayyat E, Kheirandish-Gozal L, Sans Capdevila O, Maarafeya MM, Gozal D.

From the Division of Pediatric Sleep Medicine (Drs. Dayyat, Kheirandish-Gozal, Capdevila, and Gozal), Department of Pediatrics, University of Louisville, Louisville, KY; and the Department of Pediatrics (Dr. Maarafeya), Division of Pediatric Pulmonology, Hamad Medical Corporation, Doha, Qatar.

Large Tonsils
and
Sleep Apnea



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www.sleepontario.com

Introduction The obesity epidemic has prompted remarkable changes in the proportion of obese children referred for habitual snoring. However, the contribution of obesity to adenotonsillar hypertrophy remains undefined. **Methods** 206 nonobese habitually snoring children with polysomnographically diagnosed OSA were matched for age, gender, ethnicity, and AHI to 206 obese children. Size estimate of tonsils, adenoids, and Mallampati class scores were obtained and allowed for assessment of potential relationships between anatomical factors and obesity in pediatric OSA. **Results** The mean obstructive AHI for the 2 groups was approximately 10.0 /hrTST. There was a modest association between adenotonsillar size and AHI in nonobese ($r:0.22$; $p < 0.001$), but not in obese children. Adenotonsillar size was larger in nonobese children (3.85 ± 0.16 vs 3.01 ± 0.14 ; $p < 0.0001$), and conversely Mallampati class scores were significantly higher in obese children ($p < 0.0001$). **Conclusions** The magnitude of adenotonsillar hypertrophy required for any given magnitude of AHI is more likely to be smaller in obese children compared to nonobese children. Increased Mallampati scores in obese children suggest that soft tissue changes and potentially fat deposition in the upper airway may play a significant role in the global differences in tonsillar and adenoidal size among obese and nonobese children with OSA.

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The effect of mode of breathing on craniofacial growth-revisited

Peltomäki T.

Clinic for Orthodontics and Pediatric Dentistry, Center for Dental and Oral Medicine, University of Zurich, Zurich, Switzerland. timo.peltomaki@zmk.uzh.ch

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Dental arch morphology in children with sleep-disordered breathing

Pirilä-Parkkinen K, Pirttiniemi P, Nieminen P, Tolonen U, Pelttari U, Löppönen H.

Department of Oral and Maxillofacial, Oulu University Hospital, Oys, Finland. kirsi.pirila-parkkinen@oulu.fi

The aim of the present study was to examine the effects of nocturnal breathing disorders such as OSA and snoring on developing dental arches. The study group comprised 41 children with diagnosed OSA. Age- and gender-matched groups of 41 snoring and 41 non-obstructed control children were selected. Orthodontic examination was carried out and dental impressions were taken. The differences between the dental arch measurements of the OSA, snoring, and control groups were studied. Children with diagnosed OSA had a significantly increased overjet, a reduced overbite, and narrower upper and shorter lower dental arches when compared with the controls. Snoring children had similar but not as significant differences as OSA children when compared with the controls. There were more children with an anterior open bite (AOB) in the OSA group ($P=0.016$) and with a Class II or asymmetric molar relationship in the groups of OSA ($P=0.013$) and snoring ($P=0.004$) subjects compared with the non-obstructed controls. There were more subjects with mandibular crowding ($P=0.002$) and with an AOB ($P=0.019$) with an increasing obstructive apnoea-hypopnoea index (AHI). These findings are in agreement with previous studies of the effects of increased upper airway resistance on dental arch morphology and can be explained by long-term changes in the position of the head, mandible, and tongue in order to maintain airway adequacy during sleep.

It has been maintained that because of large adenoids, nasal breathing is obstructed leading to mouth breathing and an 'adenoid face', characterized by an incompetent lip seal, a narrow upper dental arch, increased anterior face height, a steep mandibular plane angle, and a retrognathic mandible. After adenoidectomy and change in head and tongue position, accelerated mandibular growth and closure of the mandibular plane angle have been reported. Children with obstructive sleep apnoea (OSA) have similar craniofacial characteristics as those with large adenoids and tonsils, and the first treatment of choice of OSA children is removal of adenoids and tonsils. These children also have abnormal nocturnal growth hormone (GH) secretion and somatic growth impairment, which is normalized following adenotonsillectomy. It is hypothesized that decreased mandibular growth in adenoid face children is due to abnormal secretion of GH and its mediators. After normalization of hormonal status, ramus growth is enhanced by more intensive endochondral bone formation in the condylar cartilage and/or by appositional bone growth in the lower border of the mandible. This would, in part, explain the noted acceleration in the growth of the mandible and alteration in its growth direction, following the change in the mode of breathing after adenotonsillectomy.

The Snoring Bedtime

John V. Kelleher
(16th Century-Irish)

You thunder at my side,
Lad of ceaseless hum;
There's not a saint would chide
My prayer that you were dumb.

The dead start from the tomb
With each blare from you nose.
I suffer, with less room,
Under these bedclothes.
With could I better bide
Since my head's already broke –
Your pipe-drone at my side,
Woodpecker's drill on oak?

Brass scraped with knicky knives,
A cowbell's tinny clank,
Or the yells of tinkers' wives
Giving birth behind a bank?

A drunken, braying clown
Slapping cards down on a board
Were less easy to disown
Than the softest snore you've snored.

Sweeter the grunts of swine
Than yours that win release.
Sweeter, bedtime mine,
The screech of grieving geese.

A sick calf's moan for aid,
A broken mill's mad clatter,
The snarl of flood cascade...
Christ! Now what's the matter?

That was a ghastly growl!
What signified that twist? –
An old wolf's famished howl,
Wave-boom at some cliff's breast?

Storm screaming round a crag,
Bellow of raging bull,
Hoarse bell or rutting stag.
Compared with this were hull!

Ah, now a gentler fall –
Bark of a crazy hound?
Brats squabbling for a ball?
Ducks squawking on a pond?

No, rough weather's back again.
Some great ships' about to sink
And roaring bursts the main
Over the bulwark's brink!

Farewell, tonight, to sleep.
Avery gust across the bed
Makes hair rise and poor flesh creep.
Would that one of us were dead!

By Deena Sherman

Degas

What makes French nineteenth century artist, Edgar Degas' "Women Ironing" unusual, is that it includes a great big yawn. The 1884 painting was part of a series of works which depict everyday Parisians trying to make a living, and the drudgery that this often involved. But it is not only the drudgery and



Women Ironing, 1884

hard work that is making one of the women in "Women Ironing" yawn, for in her hand she holds what must be a chief contributor to her somnolence: a bottle of wine.

Degas was born in 1834 in Paris, the first of five siblings born to a somewhat wealthy banker, Augustin De Gas and his wife, Célestine Musson De Gas who died while still relatively young, in 1847. Degas' interest in art began early and by his mid-twenties he was a fairly successful artist. While fighting in the Franco-Prussian war (Degas had enlisted in the French army in 1870 in spite of already being in his late thirties), it was discovered that Degas' sight was compromised. It would deteriorate throughout his life leading to eventual blindness in 1889. Degas died in 1917.

Degas has been described as a founder of impressionism – an irony because he wrestled with his association with impressionistic artists. The artist had become disillusioned with the Parisian art establishment, and so found himself exhibiting with the impressionist artists for a number of years in the 1880's. Degas had important differences from impressionist artists and derided some of their methods, most particularly their painting out in the open, thereby getting an immediate, first "impression" of the scenery. What tied him to the movement, aside from exhibiting with them, was his daring use of colors and his depiction of everyday scenes such as "Women Ironing".

Sources

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