

# Snoring in a cohort of obese children: association with palate position and nocturnal desaturations

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## Abstract

**Purpose of the study:** Frequency of habitual snoring is significantly higher in obese than in normal-weight subjects. Obesity and adeno-tonsillar size are risk factors of snoring. Other factors, such as fat distribution and upper airway collapsibility, could explain the relationship between obesity, snoring and obstructive sleep apneas. The aim of the study was to investigate clinical and instrumental significance of snoring in exogenous obese children referred to our department.

**Methodology:** This preliminary study takes part of a larger prospective respiratory sleep study. In 36 consecutive obese children (18 males), body mass index (BMI), BMI Z score and neck circumference were calculated according to age and sex. Nasal patency, tonsil size, palate position scoring were also recorded. An overnight polygraphy was performed using a portable ambulatory device. Statistical analysis was done using SPSS® Statistics 19.0 software for Windows®.

**Main findings:** Snoring, objectively measured by polygraphy, was associated with palate position and with oxygen desaturation index (ODI). The correlation between snoring and ODI completely disappeared when adjusting for palate position scoring.

**Key conclusions:** Low palate position can be identified as an adjunctive, although not unique, factor that can contribute to making snoring and increased desaturation events possibly related to increased risk of upper airway collapsibility during sleep in obese children.

## Keywords

Snoring, obesity, children, palate position, polygraphy.

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## Introduction

The clinical spectrum of sleep-disordered breathing (SDB) includes obstructive sleep apnea syndrome (OSAS), upper airway resistant syndrome and primary or habitual snoring. Approximately 10% of children snore regularly and about 2-4% of the pediatric population has OSAS. Adenotonsillar hypertrophy and reduced neuromuscular tone are the main causes of SDB in children [1]. Finally, studies indicate that mild SDB or snoring may cause many of the same problems as OSAS in children [2].

The gold standard for diagnosis of SDB is overnight polysomnography (PSG) performed in a sleep lab. A polygraphic device is reported to be an acceptable [3] and cost-effective [4-5] alternative.

Brunetti et al. showed that the frequency of habitual snoring was significantly higher in obese (12.5%) than in normal-weight subjects (4.6%), whereas OSAS was 3.1% and 1.6% ( $p = \text{NS}$ ), respectively [6]. Marcus et al. confirmed that obesity is an independent risk factor for snoring [7]. However, other factors besides increased body fatness lead to snoring of high power frequency; one of these factors is adeno-tonsillar size [8].

The aim of the present study was to investigate the clinical and instrumental significance of snoring in an ambulatory group of exogenous obese children.

## Subjects and methods

This study takes part of a larger prospective respiratory sleep study we carried out from January 2014 to August 2015. This research, performed in

36 consecutive exogenous obese children (17 males) aged (mean  $\pm$  SE)  $12.1 \pm 0.45$  years (range 6.5-17 years) referred to our department specializing in endocrine disorders, is aimed to explore the snoring in childhood obesity. They had no declared other conditions than obesity. Caregivers signed an informed consent document prior to enrollment in the study. The protocol was approved by the Institutional Ethics Committee of Verona.

Body mass index (BMI) and BMI Z-scores (<http://nccd.cdc.gov/dnpabmi/Calculator.aspx>) and neck circumference [9] were calculated according to age and sex. Nasal patency (right plus left nostril obstruction: not bilaterally obstructed 0 – totally bilaterally obstructed 8) [10], tonsils size (1 to 4) [11] and palate position (1 to 4) [12] were graded according to previously published roles.

An overnight polygraphic study was performed using a portable ambulatory device (SOMNOscreen™ PSG, SOMNOmedics GmbH, Randersacker, Germany) with continuous monitoring of nasal airflow, chest and abdominal respiratory movements (thoracic and abdominal belts), arterial oxygen saturation ( $\text{SaO}_2$ ; digital pulse oxymetry), heart rate (finger probe), ECG, body position (mercury sensor) and tracheal sounds (microphone). The device was applied between 06:00 PM and 08:00 AM and the recording was for the entire night in a quiet, specifically prepared sleep room. Analysis of the entire recording was done both manually and automatically (DOMINO software, SOMNOmedics v.2.6.0). Estimated total sleep time (eTST) was calculated according to published criteria, and movement periods were excluded [13]. Respiratory events were scored according to the American Academy of Sleep Medicine guidelines [3, 14]. The number of obstructive apneas (OA) plus mixed apneas plus central apneas and hypopneas (H) was divided by hours of eTST (n/h) and expressed as an index (apnea-hypopnea index, AHI). Desaturation was considered if there was a drop  $\geq 3\%$  oxygen. All  $\text{O}_2$  desaturations (n/h) from the baseline, mean  $\text{SaO}_2$  (%) and  $\text{SaO}_2$  minimum (%) were quantified. The oxygen desaturation index (ODI) was calculated as the total number of desaturations divided by the eTST (n/h). Snoring (% of eTST) was also recorded.

Statistical analysis was done using SPSS® Statistics 19.0 software for Windows®.

## Results

Enrolled children had BMI of  $29.4 \pm 0.8$  (mean  $\pm$  SE)  $\text{kg}/\text{cm}^2$ , and BMI (Z-score) of  $2.14 \pm 0.05$ .

All the children had a neck circumference > 97<sup>th</sup> percentile for age and sex. The distribution of the scoring was assigned for nasal patency (0 = 44.4%, 1 = 16.7%, 2 = 13.9% and 3 & 4: 25%), tonsil enlargement (1 = 61.1%, 2 = 27.8%, 3 = 11.1% and 4 = 0%) and Friedman palate position (1 = 41.7%, 2 = 44.4%, 3 = 8.3% and 4 = 5.6%). The eTST was  $8.2 \pm 0.1$  hours, snoring (% eTST) was  $3.35 \pm 1.33\%$ , OA (n/h), H (n/h) and AHI (n/h) were  $0.18 \pm 0.05/h$ ,  $0.50 \pm 0.15/h$ ,  $0.81 \pm 0.15/h$ , respectively. Basal SpO<sub>2</sub> (%) was  $97.6 \pm 0.11\%$  and ODI (n/h) was  $0.89 \pm 0.17/h$ . Correlation analysis between snoring (% eTST) and clinical variables (age, BMI Z-score, and nasal patency, tonsil patency and palate position scoring) showed that only palate position was included in the model ( $r = 0.427$ ;  $p = 0.009$ ). Correlation analysis (Tab. 1) between snoring (% eTST) and sleep respiratory parameters (OA, H, AHI, ODI and basal SpO<sub>2</sub>) showed that both ODI and basal SpO<sub>2</sub> were included in the model, even after adjustment for nasal patency and tonsil hypertrophy; the correlation disappeared when adjusting for palate position scoring.

## Discussion

The major finding of the present study was that in a group of obese children a simple clinical parameter – snoring – objectively measured by an overnight polygraphy was associated with palate position and nocturnal desaturations (ODI). Thus, we speculate that low palate position, combined with an enlarged neck circumference, can contribute to a higher number of desaturation events possibly related to the propensity to upper airway collapsibility during sleep.

The prevalence of OSAS in obese children is high (37.1%) [15]. The clinical conditions that

increase the risk of OSAS in childhood obesity have been investigated. In particular, enlarged tonsil and adenoid size increases the risk for OSAS in these children [16-18]. A lower minimum oxygen saturation has also been found [19]. Therefore, the prevalence of OSAS in obese snorers has been estimated to be much higher. Other factors, such as fat distribution and upper airway collapsibility, could explain the relationship between obesity, snoring and OSAS [20]. In particular, soft palate size and position, tongue and mandibular individual variations have been involved in giving predisposition to the upper airway collapsibility during sleep [21].

Snoring has been reported as very frequent in children. The prevalence of habitual snoring in children of primary and secondary school age was 5.6%. Increased body fatness, decreased nasal patency and enlarged tonsils have been associated with snoring [22, 23]. In particular, children who had become obese or were persistently obese had a significant risk of becoming snorers than normal-weight counterparts, overall after adjusting for tonsil size [24].

Obese patients with snoring, confirmed apneas, and upper airway obstruction on polysomnograms may have a “prodromal” form of obese hypoventilation syndrome (OHS) characterized by hypoventilation during sleep without awake hypercapnia [25]. Although SDB is not currently part of the basic definition of OHS, these individuals typically show severe OSAS with hypercapnia and obstructive hypoventilation during sleep [26]. There is still the need for a better understanding of which patients with obesity and SDB develop this condition over time. The emergence of an increasing number of very obese individuals will pose management challenges in the near future [27].

**Table 1.** Correlation analysis between snoring and sleep respiratory parameters.

Dependent variable	Independent variables	r	p	r <sub>par</sub> <sup>1</sup>	p <sub>par</sub>	r <sub>par</sub> <sup>2</sup>	p <sub>par</sub>	r <sub>par</sub> <sup>3</sup>	p <sub>par</sub>
Snoring (% eTST)	OA (n/h)	-0.028	0.873	0.054	0.759	-0.027	0.877	0.026	0.881
	H (n/h)	0.259	0.128	0.259	0.134	0.252	0.145	0.242	0.161
	Flux limitation (n/h)	-0.027	0.877	-0.056	0.751	-0.006	0.975	-0.104	0.550
	AHI (n/h)	0.154	0.370	0.200	0.249	0.145	0.406	0.185	0.286
	RDI (n/h)	-0.003	0.988	-0.024	0.892	0.017	0.923	-0.073	0.678
	ODI (n/h)	<b>0.356</b>	<b>0.033</b>	<b>0.356</b>	<b>0.036</b>	<b>0.349</b>	<b>0.040</b>	0.303	0.077
	Basal SpO <sub>2</sub> (%)	<b>-0.397</b>	<b>0.017</b>	<b>-0.361</b>	<b>0.033</b>	<b>-0.400</b>	<b>0.017</b>	-0.333	0.051
Phase angle (degrees)	0.094	0.585	0.084	0.632	0.115	0.510	-0.037	0.831	

eTST: estimated total sleep time; OA: obstructive apneas; H: hypopneas; AHI: apnea-hypopnea index; RDI: respiratory disturbance index; ODI: oxygen desaturation index.

Adjusted for <sup>1</sup>nasal patency, <sup>2</sup>tonsil hypertrophy and <sup>3</sup>Friedman palate position.

## Conclusion

In conclusion, in our pooled group of obese children, low palate position can be considered an adjunctive, although not unique, factor that can contribute to snoring and increased desaturation events possibly related to increased risk of upper airway collapsibility during sleep. Palate position and habitual snoring should be included in the initial evaluation of obese children in an out-patient setting before referring for sleep respiratory investigation.

## Abbreviations

AHI: apnea-hypopnea index

BMI: body mass index

eTST: estimated total sleep time

H: hypopneas

ODI: oxygen desaturation index

OHS: obese hypoventilation syndrome

OSAS: obstructive sleep apnea syndrome

PSG: polysomnography

SDB: sleep-disordered breathing

## Declaration of interest

All Authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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