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Original Article

Sleep-disordered breathing and pulmonary function in obese children and adolescents

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ABSTRACT

Objective: Obese children have an increased risk of developing obstructive sleep apnea syndrome (OSAS) compared to normal-weight children. In obese children, OSAS is more frequently associated with oxygen desaturations, which might be caused by pulmonary function abnormalities. Our goal was to investigate the association between OSAS and pulmonary function in obese children and adolescents.
Methods: There were 185 children included and distributed in groups based on their obstructive apnea–hypopnea index (151 controls, 20 mild OSAS, and 14 moderate-to-severe OSAS). All subjects underwent polysomnography and pulmonary function testing.
Results: Several differences in pulmonary function were observed between groups. Vital capacity (VC) and forced expired volume in 1 s (FEV_1) were significantly decreased in patients with moderate-to-severe OSAS, as were expiratory reserve volume (ERV), total lung capacity, and functional residual capacity (FRC). Correlations between FEV_1, FRC, and ERV with OSAS severity remained significant independent of the degree of adiposity. Correlations between FEV_1/VC and sleep-related respiratory parameters did not persist after correction for adiposity.
Conclusion: An association between awake pulmonary function and sleep-related respiratory parameters could be observed in our population of obese children. These results suggest that OSAS severity is correlated with a diminished lung function. However, the level of obesity remains an important confounding factor in both OSAS severity and pulmonary function.

1. Introduction

The prevalence of childhood obesity is increasing worldwide and has been recognized as a major health problem associated with several physical, psychosocial, and social consequences [1]. Restrictive pulmonary function abnormalities are a well-reported complication of obesity in an adult population, with reductions in lung volumes and expiratory flow rates being most frequently reported [2]. However, similar studies in children show conflicting data and no strong evidence has been found for a correlation between the degree of obesity and pulmonary function abnormalities in children [3].

Sleep-disordered breathing (SDB) is a well-documented complication in obese adults and children. SDB includes primary snoring, upper airway resistance syndrome, and obstructive sleep apnea syndrome (OSAS) [4]. OSAS is defined by intermittent cycles of upper airway collapse associated with hypoxia and arousals during sleep. The prevalence of OSAS has been reported to be between 13% and 59% in obese children, compared to 2–3% in normal-weight children [5,6]. Multiple factors may be responsible for the increased risk of OSAS in obese children and adolescents, including structural changes in the upper airway, adenotonsillar hypertrophy, and excess fat deposition around the pharynx [7–9]. Other possible underlying mechanisms may be pulmonary function abnormalities. Obesity may result in central adiposity and an excess mechanical load on the chest wall, which in turn can result in reduced functional residual capacity (FRC) and tidal volumes [10]. A reduction in FRC can augment the development of severe hypoxia during OSAS. This could explain why OSAS in obese

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children is associated with more frequent oxygen desaturations. Several studies have examined whether pulmonary function tests (PFTs) are abnormal in adults with SBD [11–13]. However, these results have been inconsistent, possibly due to confounding factors such as chest wall abnormalities in obesity. Limited data are available concerning the association between obesity, SDB, and PFT in children. Therefore, our goal was to investigate the association between SDB and pulmonary function in obese children and adolescents, and the following hypotheses were examined: (i) diminished lung volumes are associated with an increased risk for obstructive events and (ii) OSAS is associated with a more obstructive breathing pattern, as measured by PFT.

2. Methods

2.1. Study population

In this prospective study, consecutive overweight and obese children were recruited between November 2006 and December 2012 at the Pediatric Obesity Clinic of the Antwerp University Hospital. Children were excluded in case of infection, chronic medical condition or genetic, neuromuscular, or craniofacial syndromes. The ethics committee of the Antwerp University Hospital approved this study and informed consent was obtained from the patients and their parents.

2.2. Anthropometry

All measurements were performed in the morning, after an overnight fast with patients undressed. Height, weight, waist circumference, and waist-to-hip ratio (WHR) were measured using standardized techniques by skilled personnel. Fat mass was measured with bioelectrical impedance analysis, using the Deurenberg formula for children [14]. Body mass index (BMI) was calculated as weight (kg) divided by height (m^2) and was further analyzed as *z*-scores, using the Flemish growth study as a reference population [15]. Overweight and obesity were defined according to the International Obesity Task Force criteria [16].

2.3. Pulmonary function testing

All patients underwent full lung function evaluation. Lung function tests included spirometry and helium dilution (Jaeger MS-PFT analyzer unit, Jaeger, Würzburg, Germany), and full body plethysmography (Jaeger Masterscreen box, Jaeger, Würzburg, Germany). Spirometry yielded the following data: vital capacity (VC) and forced expired volume in 1 s (FEV_1). FRC, total lung capacity (TLC), and residual volume (RV) were measured via body plethysmography and helium dilution. Expiratory reserve volume (ERV) was calculated by means of FRC and RV measured by body plethysmography. Specific airway resistance (sRaw) was measured through body plethysmography. PFT parameters are expressed as a percentage of the predicted value.

2.4. Polysomnography

All children underwent nocturnal polysomnography for $\geqslant$6 h on the day of admission. The following variables were continuously measured and recorded by a computerized polysomnograph (Brain RT, OSG, Rumst, Belgium): electroencephalography (C4–Al and C3–A2); electrooculography; electromyography of anterior tibial and chin muscles; and electrocardiography. Respiratory effort was measured by respiratory inductance plethysmography and oxygen saturation by a finger probe connected to a pulse oximeter. Airflow was measured by means of a nasal pressure cannula and thermistor, and snoring was detected by means of a microphone at the suprasternal notch. All patients were monitored on audio/videotape using an infrared camera. Respiratory events were scored according to the American Academy of Sleep Medicine guidelines [17].

The obstructive apnea–hypopnea index (oAHI) was defined as the average number of obstructive apneas and hypopneas per hour of sleep. Mild OSAS was diagnosed by the presence of an oAHI between 2 and 5 and moderate-to-severe OSAS was defined by an oAHI $\geqslant$ 5. The respiratory disturbance index (RDI) was calculated as the sum of the recorded apneas and hypopneas divided by the total sleep time. All desaturations of $\geqslant$4% from the baseline oxygen saturation were quantified and the oxygen desaturation index (ODI) was calculated as the total number of desaturations divided by the total sleep time.

2.5. Statistical analysis

All statistical analysis was performed using SPSS 20.0 (SPSS, Chicago, IL, USA). A previous study showed a significant difference in FEV_1 between subjects with and without OSAS [18]. Based on that study, a sample size of 27 subjects with OSAS would be needed to achieve adequate statistical power (type I error rate of 5% and a power goal of 90%). Normality was tested by the Kolmogorov–Smirnov test. Normally distributed data are presented as mean ± standard deviation. Skewed data are reported as median ± interquartile range. Patients were distributed in groups based on their oAHI. Groups were compared by means of χ^2, one-way analysis of variance, or Kruskal–Wallis test, as appropriate. The differences between groups were further analyzed by means of the post hoc Tukey test or Jonckheere–Terpstra trend test as appropriate. Correlations between PFT and sleep parameters were calculated using Pearson's or Spearman's correlation analysis as appropriate. Linear regression analysis was performed in the case of a significant correlation to determine whether the correlation persisted after controlling for the degree of adiposity. Because the different measures of adiposity are highly intercorrelated, linear regression was done by the inclusion of one measure of adiposity with the highest univariate correlation coefficient for the respective outcome (BMI *z*-score, waist circumference, WHR, fat mass). For all analyses, $P < 0.05$ was considered statistically significant.

3. Results

3.1. Subjects' characteristics

A total of 185 overweight and obese children were included in this study with an average BMI of 30.4 kg/m^2 (range: 19.7–48.1 kg/m^2), which corresponds to a mean *z*-score of 2.4 (range: 1.5–3.6). Mean age was 12 years (range: 5–17 years) and 40% of subjects were male. OSAS was diagnosed in 34 children (18.3%), 20 subjects had mild OSAS (10.8%), and 14 moderate-to-severe OSAS (7.5%). Patient characteristics between the three groups (oAHI < 2; 2 < oAHI < 5; oAHI $\geqslant$ 5) are compared in Table 1. No significant difference in patient characteristics between groups was found except for WHR.

3.2. Respiratory parameters

Sleep-related respiratory parameters were significantly different between groups as expected. VC and FEV_1 were significantly lower in patients with OSAS, as were TLC and FRC measured by helium dilution. ERV was also significantly lower in patients with OSAS. Post-hoc testing showed a significant decrease for all lung