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Dramatic impact of morbid obesity on child lung development

Short title: Impact of morbid obesity on child lung development

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ABSTRACT

Objective: To assess the respiratory function and sleep characteristics of obese adults and children.

Methods: All patients with non-syndromic, severe obesity (BMI ≥ 3 z-scores for children and ≥ 40.00 kg/m^2 for adults), referred for pulmonary function tests at Lille University Hospital, were retrospectively included.

Results: A total of 69 children (mean ±SD BMI 36.8 ±6.7 and mean BMI z-score 4.7 ±1.0) and 70 adults were included (mean BMI 45.7 ±6.2). Metabolic syndrome was diagnosed in 13 children (26%) and 40 adults (80%). Reduced lung volumes were observed in 34 children

(50.0%) and 16 adults (24.0%) and both the mean functional residual capacity (FRC) and the mean residual volume (RV) were lower in children than in adults (FRC: -1.7 ±2.1 z-score in children vs. -1.0 ±1.1 in adults, p=0.026; and RV: -0.8 ±1.2 z-score in children vs. -0.1 ±1.1 in adults, p=0.002). The prevalence of severe obstructive sleep apnea syndrome was greater in adults (40.7% vs. 18.8%, p=0.007). Children had a higher average oxygen saturation (median of 96.0% [91.0–98.0] vs. 93.0% [76.0–97.0] in adults, p<0.0001).

Conclusion: Obesity has consequences for lung volumes in children; however, a longitudinal study is needed to determine the impact on pulmonary expansion and growth.

Keywords: obesity; lung function; polysomnography; metabolic syndrome; sleep disorders breathings

1. Introduction

The World Health Organization (WHO) defines obesity as an excess in fat mass great enough to increase the risk of morbidity, to alter physical, psychological, or social well-being, and/or mortality, and is described as “the most-ignored public health problem” [1]. The prevalence of childhood obesity has increased substantially and rapidly worldwide in less than a generation and childhood obesity is associated with an increased rate of premature death from endogenous causes [2,3]. Indeed, in addition to the known complications of obesity including cardiovascular and metabolic diseases (high blood pressure, dyslipidemia, and impaired glycemia), obesity has also been linked to a number of respiratory disorders [4–7]. Today, sleep-related breathing disorders as complications of obesity are well documented in adults but remain poorly documented in children [8]. To the best of our knowledge, no study to date has investigated nocturnal and diurnal respiratory function, comparing morbidly obese children and adults. The aim of this study was to assess the respiratory function and sleep characteristics of obese adults and children.

2. Methods

2.1 Inclusion criteria

We retrospectively included all patients with non-syndromic, severe obesity, referred to our hospital for pulmonary function tests from December 2010 to July 2014. Obesity in children and in adolescents was defined as body mass index (BMI) ≥ 3 z-scores and severe obesity as BMI ≥ 4 z-scores [9]. In adults, we used the WHO International Classification of adult overweight and included obese class III, those having a BMI ≥ 40.00 kg/m^2 [1].

2.2 Demographic data

The demographic data collected were gender, age, obesity onset (in children), systolic and diastolic blood pressure, waist circumference, weight, height, and BMI. BMI values were converted into z- scores.

2.3 Pulmonary function tests

Spirometry and lung volume measurements were performed following the American Thoracic Society and European Respiratory Society (ATS/ERS) recommendations [10,11] by body plethysmography (Masterscreen, Jaeger, Carefusion, San Diego, CA). Pulmonary function data were collected including slow vital capacity (SVC), plethysmographic functional residual capacity (FRC), residual volume (RV), total lung capacity (TLC), RV/TLC ratio, forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), FEV_1/FVC ratio, and forced expiratory flow between 25 and 75% of FVC (FEF_{25-75}). Reference values for spirometry [12] and lung volume in children [13] and adults [14] were previously described and abnormal values defined as z- score < -1.645.

2.4 Polysomnography

In our obesity management center, polysomnography is performed systematically even in the absence of any clinical symptoms. Polysomnographic studies were performed overnight in a quiet and dark room using Brainnet for adults (Medatec-Medical Data, Brussels, Belgium) and the Embletta sleep system for children (Embla, Inc., Broomfield, CO). Data were recorded on a digital acquisition system (Embla, ResMed, Inc., San Diego, CA) for adults and Medatec (Medatec-Medical Data, Brussels, Belgium) for children. This acquisition system

continuously measured and recorded data, including: electroencephalography (C4-A1 and C3-A2), electro-oculography, chest and abdominal wall motion, oronasal air flow, electrocardiography, microphone sounds at the suprasternal notch, chin and anterior tibial muscles electromyography. Pulse oximetry was recorded using a Nonin 8000 oxymeter.

Apnea was defined as absence of airflow at the nose for >10 s in adults and two breaths during baseline breathing in children. Hypopnea was defined as a 10-s or longer event (two breaths during baseline breathing in children) characterized by either a clear decrease (>50%) of oronasal airflow, or a clear amplitude reduction (<50%) associated with ≥3% oxygen desaturation or an arousal [15]. Respiratory effort was measured by respiratory inductance plethysmography and oxygen saturation by a finger probe connected to a pulse oxymeter. A snoring event was defined as a breathing sound with a sound pressure level greater than 60 dB. Sleep efficiency was defined as the total sleep time divided by the total recording time. Respiratory events were scored according to the American Academy of Sleep Medicine guidelines [15].

The following data were collected: Epworth score [16], presence of snoring, sleep efficiency, apnea hypopnea index (AHI), average oxygen saturation, minimum oxygen saturation, percentage of total sleep time with an oxygen saturation < 90%, and, in adults, awakening partial arterial pressures of carbon dioxide (P_aCO_2) or, in children, awakening partial venous pressures of carbon dioxide (P_vCO_2). Abnormal values were defined as an Epworth score ≥11, presence of snoring, an average oxygen saturation ≤95%, any time spent with SpO_2 ≤90% and a PCO_2 >50 mmHg. Severe obstructive sleep apnea syndrome (OSAS) was defined as an AHI >10 in children and >30 in adults [15-18].

The data from patients were retrospectively collected. These tests are part of the clinical management of severely obese patients and in this context, and according to our institutional guidelines, no institutional review board approval was required for our study.

2.5 Laboratory data and metabolic syndrome

The following laboratory data were collected: fasting insulinemia, glycemia, triglycerides, high-density lipoprotein (HDL) cholesterol, and total cholesterol.

In children, abdominal obesity was defined as a waist circumference of >90th percentile (or adult cut-off if lower). If BMI was >30 kg/m^2, a positive waist circumference criterion was assumed. In adults, abdominal obesity was defined as a waist circumference of ≥94 cm for men and ≥80 cm for women [19,20].

Abnormal values were elevated triglycerides >1.7 mmol/L (≥150 mg/dL), low HDL cholesterol <1.03 mmol/L in males and children (<40 mg/dL) or <1.29 (50 mg/dL) in females or specific treatment for these lipid abnormalities, increased plasma glucose ≥5.6 mmol/L (100 mg/dL) or previously diagnosed type 2 diabetes, high systolic (>130 mmHg) or diastolic (>85 mmHg) blood pressure or treatment of previously diagnosed hypertension [19,20].

Metabolic syndrome was defined as abdominal obesity and the presence of two or more other clinical features (i.e., elevated triglycerides, low HDL cholesterol, high systolic or diastolic blood pressure, and increased plasma glucose) [20].

2.6 Statistical analysis

Qualitative variables are expressed as frequency (percentage) and quantitative variables as mean ±standard deviation (SD), or median (range) in the case of non-Gaussian distribution.

Normality of distribution was checked graphically and by using the Shapiro–Wilk test. Comparisons between the two groups were made using the chi-square test (or Fisher's exact test when expected cell frequency was <5) for qualitative variables and Student's *t* test or the Mann–Whitney U test according to normality for quantitative variables. Correlations between the two quantitative variables were assessed using Spearman's coefficient correlation. Statistical testing was done at the two-tailed α level of 0.05. Data were analyzed using the SAS software package, release 9.4 (SAS Institute, Cary, NC). Given the exploratory nature of the present study, we did not adjust for multiple comparisons.

3. Results

3.1 Demographic characteristics of study participants

We included 69 children (32 boys [46.4%], age range 7.2–17.9 years). Among them, 18 (26.0%) presented with mild obesity and 51 (74.0%) with severe obesity. Mean BMI was 36.8 ±6.7 kg/m^2. In our population of children, obesity started at a median age of 4 years (range 0.08–9.0), median obesity duration was 10.2 years (range 4.91–15.39), 27 children (39.1%) presented with asthma, 13 (18.8%) had undergone adenoidectomy and 19 (27.5%) tonsillectomy **(Table 1)**.

A total of 70 adults (21 men [30.0%], age range 18.2–76.1 years) were enrolled and 59 (84.3%) underwent polysomnography. Mean BMI was 45.7±6.2 kg/m^2. Among the adults, nine (12.8%) presented with asthma and one (1.4%) presented with chronic obstructive pulmonary disease.

3.2 Pulmonary function tests in the two groups

Abnormally low FRC (observed in 50% [34/68] of children) and RV (in 22.1% [15/68]) was more prevalent in children (p=0.02 and p=0.03, respectively, Table 2) whereas abnormally low FEV_1 and FVC were more prevalent in adults (p=0.007 and p<0.0001, respectively). In agreement with this, children had lower mean z-scores for FRC and RV than adults but higher mean z-scores for FEV_1 and FVC (Table 3). An obstructive ventilatory defect (FEV_1/FVC ratio < lower limit of normal [LLN]) was observed in 11 adults and 11 children (six children suffering from asthma). An isolated decrease in FVC<LLN together with an FEV_1/FVC ratio ≥ LLN suggestive of a restrictive ventilatory defect was observed in 10 adults (a reduced TLC indicating true restriction was observed in one adult) and four children (a reduced TLC indicating true restriction was observed in three children).

In adults, a reduction in FEV_1/FVC was observed in 17.6% of smokers (n=6) and 15.2% of nonsmokers (n=5; p=0.78). In children, a reduction in FEV_1/FVC was observed in 23.1% (n=6) of patients with asthma and 11.6% (n=5) of patients without asthma (p=0.31).

BMI z-scores were not correlated with FRC, neither in children (r= −0.17; p=0.16) nor in adults (r= −0.05, p=0.68).

The prevalence of abnormal FRC was not different between children with and without metabolic syndrome (46.2% vs. 47.2%, respectively, p=0.95) or in adults with and without metabolic syndrome (30.6% vs. 10.0%, respectively, p=0.25).

3.3 Sleep disorders in the two groups

In comparison with adults (Table 2), sleep-related respiratory disorders were observed less frequently in children. Snoring was more common in adults (37/42 [88.1%] vs. 14/47 [29.8%], p<0.0001). The prevalence of severe OSAS was higher in adults: 24/59 adults (40.7%) vs. 13/69 children (18.8%) (p=0.007). Mean average saturation was higher in

children than in adults (96.0% [91.0–98.0] vs. 93.0% [76.0–97.0], $p<0.0001$) (Table 3). Mean Epworth score was higher in adults (9.7±5.1 vs. 6.0±4.6, $p=0.002$). The median oxygen desaturation index was 4.5 in children (0.2–40.1). None of the children (0/55) had SpO_2 <90% vs. 16/54 adults (29.6%) ($p<0.0001$).

BMI z-scores were not correlated with AHI, neither in children (r =0.12, $p=0.33$) nor in adults (r =0.08, $p=0.52$). Abnormal average SpO_2 prevalence was similar between children with and without metabolic syndrome (observed in 2 [15.4%] vs. 10 [27.8%], respectively, $p=0.47$). AHI was not associated with the presence of metabolic syndrome in children ($p=0.63$).

3.4 Metabolic syndrome

Metabolic syndrome was diagnosed in 13 (26%) children with available data compared with 40 adults (80.0%) (Table 2). The prevalence of abdominal obesity and abnormal laboratory and clinical features is presented in Table 2.

Mean (±SD) BMI z-score was not significantly higher in individuals with metabolic syndrome as compared with those without metabolic syndrome, neither in children (4.77 ±1.30 vs. 4.58 ±0.74, respectively, $p=0.63$) nor in adults (4.32 ±0.67 vs. 4.17 ±0.95, respectively, $p=0.56$).

4. Discussion

In our cohort, we found significant differences in pulmonary function tests and polysomnography indices between children and adults. Abnormal lung volumes were more

frequent in morbidly obese children than in morbidly obese adults, whereas OSAS was less frequent in children than in adults.

Childhood-onset obesity frequently persists into adulthood and is associated with increased long-term morbidity and mortality. In 2010, Franck et al. studied a large cohort of 4857 Indian American children without diabetes (mean age 11.3 years) and showed, at the end of follow-up, that 559 of the 4857 participants (11.5%) died before they reached 55 years of age [2]. The rate of death from endogenous causes in the highest quartile of childhood BMI was more than double that in the lowest quartile [2].

Concerning metabolic syndrome, Cook et al. reported an overall prevalence of 4.2% among a general population of 2430 adolescents aged 12–19 years, whereas in obese adolescents of their cohort, metabolic syndrome was present in 28.7% – a prevalence that is similar to our results (26% in our obese children) [21].

It is well known that obesity increases the mechanical work needed for breathing. Indeed, abdominal and thoracic fat accumulation is likely to have effects on the downward movement of the diaphragm and could decrease the functional residual capacity and total lung capacity by limiting the space for lung expansion or inflation [6]. Abnormalities in lung volume (decrease in FRC and RV) were more frequent in morbidly obese children than in morbidly obese adults and we can hypothesize that early onset of obesity may impair lung growth. Indeed, in those children experiencing early-onset obesity (at a median age of 4 years) and long obesity duration (median obesity duration 10.2 years), a decrease in physical activity and impairment in lung volume development may have occurred. This may be true if the obesity in adult patients appeared strictly in adulthood. A prospective longitudinal study is needed to confirm this hypothesis.

In children, we found that the decrease in functional residual capacity was the most common pulmonary abnormality (observed in half of children), whereas the decrease in forced expiratory volume in one second (FEV_1) was the most frequent abnormality among adults (30%) and associated with a decrease in forced vital capacity (FVC) (21%). Obstructive ventilator defect was observed in 16% of adults and may be related to the high rate of smoking in the adult population (47%). On the other hand, asthma was present in more children than adults. We can hypothesize that the children's asthma was well controlled, or that the reduction in FEV_1 in adults was due to the reduction in FVC as seen in advanced pulmonary diseases [22]. However, there was no significant difference regarding obstructive ventilator defect between smokers and nonsmokers, and no difference between children with asthma and children without asthma. This result could be due to the small effect in each group.

Reduction in plethysmographic functional residual capacity (FRC) is the most consistently reported effect of obesity on lung function, particularly in morbidly obese children [4,23]. Correlations between BMI z-score and reduction of FRC are not unanimously consistent in the literature. In our study, FRC was not correlated with BMI z-score, whereas Davidson et al. reported a negative correlation between BMI z-score and FRC [4,5]. In their study of 327 children from a general population, the range of BMI z-score values was larger while in the children of our study, BMI z-scores were homogeneously high (≥3 z-scores), which could partly explain the discrepancy between our studies. Another explanation could be differences in the assessment and definition of obesity between studies. Indeed, in a study by Li et al., BMI was not correlated with FRC whereas they found a significant, negative correlation between the degree of reduction in FRC and adiposity, as measured by dual-energy X-ray absorptiometry [5].

In a recent meta-analysis, Forno et al. demonstrated that reductions in the FEV_1/FVC ratio were more pronounced in obese children than in adults. This result was not replicated in our study, but may be due to our relatively small population [24].

Different definitions for OSAS are used between children and adults [17]. Indeed, in adults, OSAS is diagnosed when an AHI is ≥5, whereas in children, an AHI ≥1 together with another polysomnography index abnormality are required for a diagnosis [25]. The lower prevalence of severe OSAS in children vs. adults could be partly due to the previous adeno-tonsillectomy in nearly half of the pediatric patients. However, given the high proportion of morbidly obese children with severe obstructive sleep apnea in our study (AHI >10 in 18.8% of children), polysomnography should be performed for all morbidly obese children.

While the major strength of our study is the novelty of comparing the impact of morbid obesity on pulmonary function indices and polysomnography indices in children with those in adults, our study has some inherent limitations. These include: the retrospective nature of the study, without a control group; the cross-sectional assessment of lung function at a single time point; the fact that reference values for lung volume indices were different in adults and children, requiring PFT indices to be expressed as z-score values to enable comparison. Another limitation is that chronic obstructive bronchopneumopathy in adults was not assessed and that OSAS in adults is defined based on the respiratory events index rather than the AHI.

5. Conclusion

These results highlight that obesity has consequences on lung volume in children and stress the importance of further developing the detection of lung function abnormalities in obese

children; however, a longitudinal study is needing to determine its impact on pulmonary expansion and growth.

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REFERENCES

1. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 2000; 894:1-253.

2. Franks PW, Hanson RL, Knowler WC, et al. Childhood obesity, other cardiovascular risk factors, and premature death. N Engl J Med 2010;362:485-93.

3. Lobstein T, Jackson-Leach R, Moodie ML, et al. Child and adolescent obesity: part of a bigger picture. Lancet Lond Engl. 2015;385:2510-20.

4. Davidson WJ, Mackenzie-Rife KA, Witmans MB, et al. Obesity negatively impacts lung function in children and adolescents. Pediatr Pulmonol 2014;49:1003-10.

5. Li AM, Chan D, Wong E, et al. The effects of obesity on pulmonary function. Arch Dis Child 2003;88:361-3.

6. Salome CM, King GG, Berend N, et al. Physiology of obesity and effects on lung function. J Appl Physiol Bethesda Md 1985 2010;108:206-11.

7. Littleton SW. Impact of obesity on respiratory function. Respirol Carlton Vic 2012;17:43-9.

8. Watson SE, Li Z, Tu W, et al. Obstructive sleep apnoea in obese adolescents and cardiometabolic risk markers. Pediatr Obes 2014;9:471-7.

9. Rolland-Cachera MF, Cole TJ, Sempé M, et al. A Body Mass Index variations: centiles from birth to 87 years. Eur J Clin Nutr 1991;45:13-21.

10. Miller MR, Hankinson J, Brusasco, et al. Standardisation of spirometry. Eur Respir J 2005;26:319-38.

11. Wanger J, Clausen JL, Coates A, et al. Standardisation of the measurement of lung volumes. Eur Respir J 2005;26:511-22.

12. Quanjer PH, Stanojevic S, Cole TJ, et al. Multi-ethnic reference values for spirometry for the 3-95-yr age range: the global lung function 2012 equations. Eur Respir J 2012;40:1324-43.

13. Zapletal A, Paul T, Samánek M. Significance of contemporary methods of lung function testing for the detection of airway obstruction in children and adolescents. Z Erkr Atmungsorgane 1977;149:343-71.

14. Quanjer PH, Tammeling GJ, Cotes JE, et al. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl 1993;16:5-40.

15. Iber C, Ancoli-Israel S, Chesson AL Jr, et al. for the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. 1st ed. Westchester, IL: American Academy of Sleep Medicine; 2007

16. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540-5.

17. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med 2012;8:597-619.

18. Sateia MJ. International classification of sleep disorders-third edition: highlights and modifications. Chest 2014;146:1387-94.

19. Alberti KGMM, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The metabolic syndrome--a new worldwide definition. Lancet Lond Engl 2005;366:1059-62.

20. Zimmet P, Alberti G, Kaufman F, et al. The metabolic syndrome in children and adolescents. Lancet Lond Engl 2007;369:2059-61.

21. Cook S, Weitzman M, Auinger P, et al. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc 2003;157:821-7.

22. Rubinstein I, Zamel N, DuBarry L, et al. Airflow limitation in morbidly obese, nonsmoking men. Ann Intern Med 1990 ;112:828-32.

23. Dubern B, Tounian P, Medjadhi N, et al. Pulmonary function and sleep-related breathing disorders in severely obese children. Clin Nutr Edinb Scotl 2006;25:803-9.

24. Forno E, Han Y, Mullen J, et al. Overweight, Obesity, and Lung Function in Children and Adults-A Meta-analysis. J Allergy Clin Immunol Pract 2018;6:570-81.

25. Kaditis G, Alonso Alvarez M, Boudewyns A. Obstructive sleep disordered breathing in 2- to 18-year-old children: diagnosis and management. Eur Resp J 2016;47:69-94.

Table 1: Population description

	Children (*n*=69)	**Adults (*n*=70)**
Age (years)	13.7 ±2.6	44.9 ±15.5
Males, *n* (%)	32 (46.4%)	21 (30%)
Weight (kg)	95.5 ±24.3	126.6 ±19.9
BMI (kg/m^2)	36.8 ±6.7	45.7 ±6.2
BMI z-score	4.7 ±0.97	4.3 ±0.76
Smoking	2 (2.9%)	33 (47%)
Asthma	27 (39.1%)	9 (12.8%)
Lung disease	0 (0%)	3 (4.3%)

Values are presented as mean ± standard deviation or a percentage.

BMI: body mass index

Table 2: Abnormalities in pulmonary function (z-score < -1.64) and polysomnography indices and of metabolic syndrome in morbidly obese children and adults

Variable	Children (*n*=69)	Adults (*n*=70)	*p*
Pulmonary function tests			
Slow vital capacity	7/68 (10.3%)	5/66 (7.6%)	0.58
Functional residual capacity	34/68 (50.0%)	16/66 (24.2%)	0.002
Residual volume (RV)	15/68 (22.1%)	3/65 (4.6%)	0.003
Total lung capacity (TLC)	8/68 (11.8%)	2/65 (3.1%)	0.097
RV/TLC	2/68 (2.9%)	8/65 (12.3%)	0.051
FEV_1	3/69 (4.4%)	21/70 (30.0%)	<0.0001
FVC	4/69 (5.8%)	15/70 (21.4%)	0.007
FEV_1/FVC ratio	11/69 (15.9%)	11/70 (15.7%)	0.97
FEF_{25-75}	13/69 (18.8%)	16/70 (22.9%)	0.56
Polysomnography			
Epworth score ≥11	4/28 (14.3%)	17/51 (33.3%)	0.067
Presence of snoring	14/47 (29.8%)	37/42 (88.1%)	<0.0001
Average oxygen saturation <95%	14/67 (20.9%)	42/58 (72.4%)	<0.0001
Severe OSAS	13/69 (18.8%)	24/59 (40.7%)	0.007
Time spent with SpO_2 <90%	0/55 (0.0%)	16/54 (29.6%)	<0.0001

Awakening PCO_2 > 50 mmHg	1/51 (2.0%)	3/52 (5.8%)	
Metabolic syndrome	13/50 (26.0%)	40/50 (80.0%)	<0.0001
Abdominal obesity	68/69 (98.5%)	70/70 (100%)	
High blood pressure	25/59 (42.4%)	42/66 (63.6%)	
Elevated triglycerides	5/49 (10.2%)	26/53 (49.1%)	
Low HDL cholesterol	20/49 (40.8%)	40/50 (80.0%)	
Increased plasma glucose	0/59 (0.0%)	19/43 (44.2%)	

Values are presented as number/total number (percentage). *p*-value calculated using a chi-square test or Fisher's exact test. FEV_1: Forced expiratory volume in one second; FVC: forced vital capacity; PCO_2: partial pressures of carbon dioxide; FEF25-75: forced expiratory flow between 25–75% of FVC; AHI: apnea hypopnea index.

Table 3: Comparison of pulmonary function and polysomnography indices between morbidly obese children and adults

Variable	**Children (*n*=69)**	**Adults (*n*=70)**	***p***
Pulmonary function tests (z-score)			
Slow vital capacity	1.2 ±2.6	0.0 ±1.3	0.002
Functional residual capacity	-1.7 ±2.1	-1.0 ±1.1	0.026
Residual volume (RV)	-0.8 ±1.2	-0.1 ±1.1	0.002
Total lung capacity (TLC)	0.0 ±2.1	0.0 ±0.7	0.820
RV/TLC	-0.1 ±1.0	-0.3 ±1.3	0.370
FEV_1	0.0 ±1.3	-1.1 ±1.3	<0.0001
FVC	0.2 ±1.2	-0.8 ±1.3	<0.0001
FEV_1/FVC ratio	-0.3 ±1.1	-0.6 ±1.2	0.230
FEF_{25-75}	-0.3 ±1.2	-0.7 ±1.3	0.069
Polysomnography			
Epworth score	6.0 ±4.6	9.7 ±5.1	0.002
Sleep efficiency (%)[#]	88.0 (52.0 to 96.0)	86.0 (41.0 to 97.0)	0.950[#]
Apnea hypopnea index per hour[#]	4.4 (0.0 to 78.4)	23.6 (0.7 to 111.0)	<0.0001[#]
Average oxygen saturation (%)[#]	96.0 (91.0 to 98.0)	93.0 (76.0 to 97.0)	<0.0001[#]
Minimum oxygen saturation (%)[#]	79.0 (50.0 to 96.0)	79.0 (50.0 to 92.0)	0.61[#]
% of time spent with SpO_2<90%[#]	0.5 (0.0 to 11.0)	6.5 (0.0 to 99.5)	<0.0001[#]
Awakening PCO_2 (mmHg)	42.2 ±3.7	42.0 ±4.9	0.80

Values are mean ± standard deviation or median (range). FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; PCO_2: partial venous pressures of carbon dioxide; FEF_{25-75}: forced expiratory flow between 25–75% of FVC.

[#]Student's *t* test or Mann–Whitney *U* test according to normality for quantitative variables.