ORIGINAL ARTICLE

Sleep-disordered breathing in overweight and obese children and adolescents: prevalence, characteristics and the role of fat distribution

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Aims: To determine the prevalence of sleep-disordered breathing (SDB) in a clinical sample of overweight and obese children and adolescents, and to examine the contribution of fat distribution.
Methods: Consecutive subjects without chronic lung disease, neuromuscular disease, laryngomalacia, or any genetic or craniofacial syndrome were recruited. All underwent measurements of neck and waist circumference, waist-to-hip ratio, % fat mass and polysomnography. Obstructive apnoea index ⩾1 or obstructive apnoea–hypopnoea index (OAHI) ⩾2, further classified as mild (2⩽OAHI<5) or moderate-to-severe (OAHI⩾5), were used as diagnostic criteria for obstructive sleep apnoea (OSA). Central sleep apnoea was diagnosed when central apnoeas/hypopnoeas ⩾10 s were present accompanied by >1 age-specific bradytachycardia and/or >1 desaturation <89%. Subjects with desaturation ⩽85% after central events of any duration were also diagnosed with central sleep apnoea. Primary snoring was diagnosed when: snoring was detected by microphone and normal obstructive indices and saturation.
Results: 27 overweight and 64 obese subjects were included (40 boys; mean (standard deviation (SD)) age 11.2 (2.6) years). Among the obese children, 53% were normal, 11% had primary snoring, 11% had mild OSA, 8% had moderate-to-severe OSA and 17% had central sleep apnoea. Half of the patients with central sleep apnoea had desaturation <85%. Only enlarged tonsils were predictive of moderate-to-severe OSA. On the other hand, higher levels of abdominal obesity and fat mass were associated with central sleep apnoea.
Conclusion: SDB is very common in this clinical sample of overweight children. OSA is not associated with abdominal obesity. On the contrary, higher levels of abdominal obesity and fat mass are associated with central sleep apnoea.

Obese children and adolescents are at risk of sleep-disordered breathing (SDB). Several studies, using polysomnography, have documented the prevalence of obstructive sleep apnoea (OSA) in this group, ranging from 13% to 66%.[1–5] This wide range is probably due to factors such as ethnic predisposition, different inclusion criteria and diagnostic criteria for both obesity and OSA. Marcus *et al*[4] also reported on the occurrence of central apnoeas of abnormal duration or followed by desaturation in 4 of 22 children studied. More objective data on the prevalence of these pathological central apnoeas are still lacking.

In adults, studies have shown a strong correlation between central adiposity and OSA.[6–10] This association has not yet been studied in childhood obesity. We therefore determined the prevalence and characteristics of OSA and central sleep apnoea in a clinical sample of overweight and obese children and adolescents, and examined the association with fat distribution.

METHODS

Patients

We recruited consecutive subjects aged 6–16 years between February 2002 and July 2005 at our Pediatric Obesity Clinic (University Hospital of Antwerp, Wilrijk, Belgium). Most of the subjects were referrals from primary care physicians. All overweight or obese patients presenting at the clinic were eligible, except for those with chronic lung disease, neuromuscular disease, laryngomalacia, and any genetic or craniofacial syndrome. A history of adenotonsillectomy was recorded. Tonsillar size was rated as normal/enlarged, using the Brodsky scale.[11] Because puberty affects body composition, patients were classified as prepubertal or pubertal using Tanner stage (stage 1 was prepubertal), bone age (bone age ⩽10 years for girls and ⩽11.5 years for boys was prepubertal), and/or testosterone levels for boys (⩽1.2 nmol/l was prepubertal) and oestradiol levels for girls (⩽15 pg/ml was prepubertal). All subjects underwent anthropometry and polysomnography as a part of their routine clinical evaluation.

Anthropometry

Height, weight, neck and waist circumference and waist-to-hip ratio were measured by standardised techniques.[12 13] Fat mass was measured by bioelectrical impedance analysis, using the Deurenberg formula for children.[14 15] Body mass index (BMI) was calculated as weight (kg)/height^2 (m^2), and was further analysed as z scores.[16] Overweight and obesity were defined according to the International Obesity Task Force criteria.[17]

Polysomnography

Each patient underwent nocturnal polysomnography for at least 6 h. The following variables were continuously measured and recorded by a computerised polysomnograph (Oxford Medilog Sac, Oxford Instruments, Oxford, UK): electroencephalography (C4/Al and C3/A2); electro-oculography; electromyography of anterior tibialis and chin muscles; and

Abbreviations: BMI, body mass index; OAHI, obstructive apnoea–hypopnoea index; OSA, obstructive sleep apnoea; SDB, sleep-disordered breathing

electrocardiography. Respiratory effort was measured by thoracoabdominal strain gauges and oxygen saturation by a finger probe connected to a pulse oximeter (Palco Laboratories, Santa Cruz, California, USA). Oronasal air flow was measured by means of a thermistor, and snoring was detected by means of a microphone at suprasternal notch. Children were also monitored on audio/videotape using an infrared camera.

Obstructive apnoea, central apnoea and hypopnoea were defined according to standard criteria.[18] Obstructive apnoea index was defined as the number of obstructive apnoeas lasting ⩾2 respiratory cycles per hour of sleep. The central apnoea index was defined as the number of central apnoeas lasting for ⩾10 s or of any duration, but associated with >4% desaturation per hour of sleep. Central apnoeas occurring immediately after sigh or movement were not counted. The obstructive apnoea–hypopnea index (OAHI) was defined as the number of obstructive apnoeas and hypopnoeas per hour of sleep. The central apnoea–hypopnea index was defined as the number of central apnoeas and hypopneas associated with >4% desaturation per hour of sleep. All drops in oxygen saturation >4% compared with the baseline were quantified (oxygen desaturation index). Measurements associated with poor pulse tracings or following movement were excluded.[18]

Diagnostic criteria for SDB

Obstructive apnoea index ⩾1 or OAHI ⩾2, further classified as mild (2⩽ OAHI <5) or moderate-to-severe (OAHI ⩾5), were used as diagnostic criteria for OSA.[19–21] Central sleep apnoea was diagnosed when central apnoeas/hypopneas ⩾10 s were present[22] and accompanied by a >1 age-specific bradytachycardia event, [18] or >1 event of desaturation <89%.[21 23] Subjects with desaturation ⩽85% after central events of any duration were also diagnosed with central sleep apnoea. Primary snoring was diagnosed when snoring was detected by a microphone and all of the following: (1) obstructive apnoea index ⩽1, (2) OAHI ⩽2 and (3) ⩽1 desaturation between 85% and 89%. A patient without snoring but with all of the mentioned criteria was diagnosed as normal.

Statistical analysis

The statistical analysis was performed using Statistica 7.0 (StatSoft). A Kolmogorov–Smirnov test was used to test normality. Variables were compared between groups using one-way analysis of variance or with the Kruskal–Wallis test when appropriate. A Bonferroni test was used for post hoc analysis after one-way analysis of variance or with the multiple comparisons of mean ranks method after the Kruskal–Wallis test. Multiple logistic regression (stepwise forward) was performed with a diagnostic group as a dependent variable and the anthropometric measurements as covariates. All data are presented as mean (standard deviation (SD)). For all analyses, $p<0.05$ was considered to be statistically significant.

RESULTS

In all, 40 boys (28 prepubertal) and 51 girls (24 prepubertal) were examined; mean age was 11.2 (SD 2.6, range 6.3–16.7) and BMI z score was 2.3 (SD 0.5, range 1.3–3.8). Twenty seven children were overweight and 64 were obese.

Table 1 shows the prevalence of SDB. Table 2 shows the polysomnographic variables for each diagnostic group. Four patients with OSA had desaturation <85%: after an obstructive event during stage 2 in two patients, and after a central apnoea during REM in the other two. Six subjects with central sleep apnoea had desaturation <85%; in five patients this occurred during REM, and all after a central event.

Prepubertal children with central sleep apnoea had a higher waist-to-hip ratio than the normal group (0.93 (SD 0.05) *v* 0.87 (SD 0.04); $p = 0.046$). Pubertal subjects with central sleep apnoea had a higher fat mass than the severe OSA group (39.2 (SD 1.6) *v* 31.4 (SD 4.6); $p = 0.01$) and a higher z score (3.0 (SD 0.1)) than the normal group (2.3 (SD 0.4); $p = 0.04$) and the severe OSA group (2.1 (SD 0.4); $p = 0.04$). None of the anthropometric variables was a significant predictor of the presence of mild OSA. The presence of enlarged tonsils (odds ratio (OR) 8.3; 95% confidence interval (CI) 1.4 to 51.2; $p = 0.02$) was the only significant covariate in the model for moderate-to-severe OSA. Central sleep apnoea was significantly predicted by the BMI z score (OR 8.1; 95% CI 2.0 to 33.3; $p = 0.01$), waist circumference (OR 1.1; 95% CI 1.0 to 1.2; $p = 0.003$), waist-to-hip ratio (OR 1.2; 95% CI 1.0 to 1.5; $p = 0.02$) and % fat mass (OR 1.4; 95% CI1.1 to 1.9; $p = 0.01$).

DISCUSSION

We found a high prevalence of SDB in this clinical sample of overweight and obese subjects. This is the first study to describe pathological central apnoeas, often associated with severe oxygen desaturation, in obese children and adolescents. We also found that OSA was not associated with estimates of abdominal obesity. On the other hand, higher levels of abdominal obesity and fat mass were associated with central sleep apnoea.

We found a prevalence of OSA of 19% in the obese group and of 41% in the overweight group. This agrees with previous findings that overweight children have a higher prevalence of OSA.[1–5]

Central sleep apnoea was diagnosed in 13% of our patients, and was often associated with serious desaturation. Marcus *et al*[4] reported three subjects with central apnoeas associated with desaturation. To the best of our knowledge, there are no other data on central sleep apnoea in obese children. A first limitation of our central sleep apnoea diagnosis is that we measured the breathing effort with strain gauges and not by a more sensitive method such as pressure monitoring.[24] The diagnostic criteria for central sleep apnoea, which were arbitrarily chosen, are a second shortcoming. Nevertheless, they were based on normative data. Central apnoeas lasting ⩾10 s are common in children,[21 23] but are almost never accompanied by serious desaturation, and a desaturation <89% is considered to be abnormal.[23]

In contrast with OSA in adults, our subjects OSA had lower values of BMI and fat mass. Additionally, there was no association between OSA and estimates of adiposity. A possible explanation is that OSA leads to increased nocturnal energy expenditure due to the increased work of breathing.[25] This hypothesis is supported by a recent paper that studied overweight children with OSA after adenotonsillectomy; this

Table 1 Prevalence of sleep-disordered breathing in 27 overweight and 64 obese children and adolescents

	Normal	Primary snoring	Mild obstructive sleep apnoea	Moderate-to-severe obstructive sleep apnoea	Central sleep apnoea
Overweight group (n = 27)	56%	0%	19%	22%	4%
Obese group (n = 64)	53%	11%	11%	8%	17%

Table 2 Sleep parameters and respiratory indices of the described diagnostic groups

Variable	Normal	Primary snoring	Mild obstructive sleep apnoea	Moderate-to-severe obstructive sleep apnoea	Central sleep apnoea
n	49	7	12	11	12
Total sleep time (min)	449 (42)	456 (51)	444 (67)	464 (54)	475 (50)
Stage 1	3.0 (2.5)	1.6 (2.0)	2.0 (2.2)	1.9 (1.6)	1.8 (1.8)
Stage 2	44.1 (7.2)	37.6 (7.4)	39.9 (6.1)	42.8 (7.7)	41.7 (8.6)
Stage 3	9.8 (4.7)	9.7 (4.6)	10.9 (5.6)	11.6 (3.9)	10.5 (4.3)
Stage 4	22.0 (6.4)	27.0 (7.9)	23.9 (5.2)	21.7 (8.2)	22.4 (6.9)
REM	21.1 (5.1)	24.1 (6.8)	23.2 (4.2)	21.1 (5.9)	23.6 (7.5)
Sleep efficiency (%)	84.7 (6.8)	85.4 (5.7)	84.0 (9.2)	86.1 (7.3)	88.0 (5.6)
REM latency (min)	139.8 (57.3)	126.6 (78.5)	122.0 (53.0)	162.9 (66.7)	124.0 (58.3)
Sleep latency (min)	26.6 (22.4)	23.4 (13.0)	44.0 (36.2)	36.7 (28.1)	21.2 (19.8)
Central apnoea index	0.33 (0.37)	0.83 (0.61)	0.92 (1.13)	1.20 (0.97)	1.57 (2.39)
Obstructive apnoea index	0.07 (0.18)	0.17 (0.31)	0.42 (0.52)	0.85 (1.84)	0.16 (0.25)
OAHI	0.25 (0.41)	0.80 (0.65)	3.13 (1.32)*	10.70 (2.88)*†	0.22 (0.26)‡§
Central apnoea hypopnea index	0.59 (0.53)	1.49 (0.92)	1.74 (1.69)	3.19 (4.54)*	4.32 (6.13)*
SaO_2 nadir (%)	91.5 (2.6)	89.3 (2.0)	88.8 (3.28)	87.8 (6.8)	82.2 (4.76)*§
Mean SaO_2 (%)	96.9 (0.7)	96.3 (0.5)	96.6 (1.6)	96.7 (0.6)	96.4 (1.0)
Oxygen desaturation index	0.4 (0.4)	1.2 (0.9)	2.4 (2.5)*	3.2 (5.5)	5.0 (8.1)*
% of total sleep time with $SaO_2 \geqslant 95\%$	98.7 (1.5)	97.4 (1.9)	89.4 (25.5)	94.6 (7.8)	93.6 (4.8)*

OAHI, Obstructive apnoea hypopnoea index; REM, rapid eye movement; SaO_2, oxygen saturation.
Values are given as mean (SD).
Stage 1– 4 and REM sleep are all presented as a percentage of total sleep time.
*$p<0.05$ with normal group.
†$p<0.05$ with primary snoring.
‡$p<0.05$ with mild obstructive sleep apnoea ($2 \leqslant$ OAHI<5).
§$p<0.05$ with moderate-to-severe obstructive sleep apnoea (OAHI$\geqslant 5$).

resulted in some clinical improvement of obstructive sleep apnoea, but caused an increase in BMI. This increase was associated with a decrease in hyperactivity scores and in sleep, waking and total daily motor activity.[26]

The presence of tonsillar enlargement was significantly associated with predicting moderate-to-severe OSA, which agrees with the study by Wing *et al*.[5] This finding provides a rationale for tonsillectomy as a treatment option for OSA in overweight children.

We also found evidence that obesity and adipose tissue are correlated with central sleep apnoea, reflecting an unstable breathing pattern. Several hypotheses could explain this interaction: reduction of the intrathoracic volume causing lower oxygen reserves,[27] impaired ventilatory responses to hypoxia and hypercapnia, hypoventilation because of leptin resistance,[28] and central apnoea followed by narrowing or collapse of the upper airway.[29–31] More research is necessary to clarify this relationship. Again, we believe that these pathological central apnoeas need to be counted separately from obstructive apnoeas and should not be incorporated in a total apnoea–hypopnoea index if obstructive apnoea is used as a threshold for adenotonsillectomy. We can hypothesise that adenotonsillectomy in these subjects with central sleep apnoea will not completely normalise the breathing pattern. Other treatment modalities of central sleep apnoea can include non-invasive ventilation or drug treatment,[32] which needs to be established by further studies.

In conclusion, SDB is very common in overweight children. In view of the associated neurocognitive and cardiovascular complications,[33–39] overweight children and adolescents should be screened for SDB. We recommend more research into the pathogenesis and treatment of central sleep apnoea in obese children.

What is already known on this topic

- Obesity is a risk factor of obstructive sleep apnoea (OSA) in children and adolescents.
- In adults, OSA is strongly associated with abdominal adiposity.

What this study adds

- A high prevalence of pathological central apnoeas, often associated with severe oxygen desaturation, in obese children and adolescents.
- In obese children and adolescents, OSA was not associated with abdominal obesity. However, higher levels of abdominal obesity and fat mass were associated with central sleep apnoea.

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