ORIGINAL ARTICLE

A controlled study of sleep related disordered breathing in obese children

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Background: Unlike the adult sleep related disordered breathing (SDB) patients who are typically obese, the relation between obesity and childhood SDB is not clear.
Aims: To investigate whether obese children are more at risk of obstructive SDB when compared to normal population, and whether this risk is potentiated by the presence of pharyngeal lymphoid tissue.
Methods: Forty six obese children (age 10.8 (SD 2.3) years; BMI 27.4 (SD 5.1)), and 44 sex and age matched normal weight children (age 11.7 (SD 2.1) years; BMI 18 (SD 1.8)) were studied. All children underwent a set of physical examinations (including the upper airways) and sleep studies.
Results: The obese children were different from the normal weight children in terms of type (predominantly obstructive), frequency, and severity of respiratory disturbances. Depending on the criteria used, 26% or 32.6% of obese children had SDB; 2.3% of normal controls had OAI ⩾1 and 4.5% had RDI ⩾5. Presence of SDB was related to presence of tonsils (size >2; range 0–4) (OR 12.67, 95% CI 2.14 to 75.17) and BMI (OR 1.20, 95% CI 1.08 to 1.33).
Conclusions: Results suggest that obese children are at increased risk of obstructive SDB; the presence of any pharyngeal lymphoid tissue enlargement in obese children should therefore be aggressively managed.

First identified two decades ago, sleep related disordered breathing (SDB) in children is increasingly being recognised. In contrast to the adult population, the epidemiology, risk factors, complications, and outcome prognosis of childhood SDB were less well defined.[1–5]

Unlike the adult SDB patients who are typically obese, the relation between obesity and SDB in children is not clear.[1 6] In light of the increasing prevalence of childhood obesity around the world, it is particularly important to determine whether obesity would predispose to SDB in children.[7–12] In retrospective case series of SDB children, obesity was not found to be associated with SDB, although the incidence of obesity in one series of children was more than twice that of the general paediatric population.[13 14] A recent study in 3671 obese Singaporean children of multi-ethnic origins reported a relatively low prevalence rate (5.7%) of SDB.[15] This study was, however, limited by a high refusal rate, low sensitivity of the questionnaire, lack of normal controls, and sleep examination in the asymptomatic group. On the contrary, based on the clinical series of obese children, one third to nearly two thirds of them were found to be suffering from SDB.[6 16 17] Another large scale genetic-epidemiological study with 399 children in the United States identified the importance of upper and lower respiratory problems, obesity, and ethnicity as independent risk factors for SDB.[2] Nonetheless, this study only used limited channels of unattended ambulatory sleep monitoring devices that might affect the detection of subtle changes in breathing patterns.[2 18] In addition, most studies were limited by the lack of any comparable normal weight, sex, and age matched control subjects. The inclusion of control subjects is particularly important in childhood SDB as the definition of sleep related breathing abnormality is often controversial.[19 20] We therefore carried out a case-control study to determine whether obese children have an increased risk of obstructive SDB. We hypothesised that obese children are more at risk of obstructive SDB when compared to the normal population, and that this risk is potentiated by the presence of pharyngeal lymphoid tissue.

METHODS

Subjects

Forty six children (mean age 10.8 (SD 2.3) years, range 7–15 years) were consecutively recruited from the paediatric obesity clinic at a university hospital. They were all referrals from primary care physicians. Forty four normal weight, sex and age matched controls were randomly selected from the local schools. Children with known clinical conditions, such as Down's syndrome, Prader-Willi syndrome, neuromuscular disease, laryngomalacia, or upper airway surgery were excluded. Obese children were defined as those with actual weight ⩾120% of the ideal weight for height (IBW), whereas normal controls had IBW of 80–120%.[8] The university ethics committee approved the study. All subjects and their parents gave written informed consent.

Physical and ENT examination

All children and their parents completed the sleep questionnaires. The height and weight of each child were measured by the standard standiometer and the digital floor scale, Detector model 6029, respectively. The sizes of tonsils, adenoids, velopharyngeal isthmus, and nasal turbinates were examined by an ENT surgeon (WMP) with both post-nasal mirror and trans-nasal fibreoptic pharyngoscope prior to the sleep assessments and blinded to the sleep problems of the children. The sizes of various upper airway measures were graded from 0 to 4.[13 21] The size of adenoid was graded with reference to the post-nasal space (PNS): grade 0 = no adenoid, 1 = 1–25% of PNS, 2 = 26–50%, 3 = 51–75%, and

Abbreviations: AI, apnoea index; BMI, body mass index; CA, central apnoea; ECG, electrocardiogram; EEG, electroencephalogram; EMG, electromyogram; EOG, electro-oculogram; ETCO2, end tidal CO2; IBW, ideal weight for height; MSLT, multiple sleep latency test; OA, obstructive apnoea; OAI, obstructive apnoea index; ODI, oxygen desaturation index; PSG, polysomnographic studies; RDI, respiratory disturbance index; SDB, sleep disordered breathing

4 = 76–100% PNS. Similarly, the turbinates and tonsils were measured with reference to the nasal and oral cavity from 0 to 4 respectively. The velopharyngeal isthmus was defined as the space behind the uvula and graded according to the degree of obstruction: grade 0 = 100% obstruction, 1 = 99–75%, 2 = 74–50%, 3 = 49–25%, and 4 = 24–0% of obstruction.

Polysomnography

Two consecutive overnight polysomnographic studies (PSG) to be followed by a daytime multiple sleep latency test (MSLT) at the third day was performed for each child using CNS 1000P polygraph. The PSG include the recording of: electro-oculogram (EOG, left and right eyes), electroencephalogram (EEG, left central and right central), submental electromyogram (EMG), intercostal EMG, leg EMG (cross referenced over the anterior tibialis muscle of both legs), electrocardiogram (ECG), end tidal CO_2 ($ETCO_2$, using BCI Capnocheck Plus and measured via a nasal cannula), snoring sound (miniature microphone taped in place on the neck near the larynx), respiratory airflow (thermistor signals), respiratory effort (thoracic movement, abdominal movement, and their sum using pneumatic belts), body position, and arterial oxygen saturation (SaO_2, using Ohmeda 3700 pulse oximeter and measured by finger sensor). The MSLT was performed according to the standard recommendation of five 20-minute naps scheduled every two hours.[22] All sleep data were further edited by experienced polysomnographic technicians and reviewed by the principal investigator (YKW).

Terminology

Obstructive apnoea (OA) was defined as absence of airflow with persistent respiratory effort lasting longer than two baseline breaths (5 seconds in this study), irrespective of SaO_2 changes. Obstructive apnoea index (OAI) was defined as the number of OA per hour of sleep. Central apnoea (CA) was defined as absence of respiratory effort associated with absence of airflow. Those of greater than 20 seconds with or without oxygen desaturation, and those of any duration but associated with oxygen desaturation of at least 4% were quantified. Apnoea index (AI) was defined as the total number of obstructive and central apnoeas per hour of sleep. Hypopnoea was defined as a reduction of 50% or more in the amplitude of the airflow signal. Criteria for scoring obstructive and central apnoea were used to score obstructive and central hypopnoea respectively. Respiratory disturbance index (RDI) was defined as the total number of apnoea and hypopnoea per hour of total sleep time. Oxygen desaturation index (ODI) was defined as the total number of dips in arterial oxygen saturation greater than 4% per hour of sleep. Arousal was defined as an abrupt shift in EEG frequency during sleep, which may include theta, alpha, and/or frequencies greater than 16 Hz but not spindles, with 3–15 seconds in duration. In REM sleep, arousals are scored only when accompanied by concurrent increases in submental EMG amplitude.

Statistical analysis

The χ^2 test, independent *t* test, and stepwise logistic regression were used in the analysis. All tests were considered significant when $p<0.05$.

RESULTS

Table 1 presents the demographic characteristics of the subjects. In our study, there were more boys (68.9%) than girls (31.1%). As expected, the obese group was heavier than the control group as measured by weight, body mass index (BMI), and IBW. While 34.8% of the obese children reported habitual snoring of more than four nights per week for the past year, only 15.9% of the control subjects had similar complaints (χ^2 test, $p<0.05$). More obese children (50%) than control subjects (22.7%) reported nocturnal mouth breathing (χ^2 test, $p<0.05$).

Table 1 Demographic data of obese and control groups

	Obese group	Control group	p value
Total no. of subjects	46	44	
Age (y)	10.8 (2.3)	11.7 (2.1)	NS
Sex (boys/girls)	33/13	29/15	NS
Body weight (kg)	62.7 (21.2)	41.2 (9.4)	<0.001
Body height (m)	1.50 (0.1)	1.50 (0.1)	NS
BMI (kg/m^2)	27.4 (5.1)	18.0 (1.8)	<0.001
Ideal body weight for height (%)	157.0 (26.2)	102.4 (8.5)	<0.001

SD in parentheses.
BMI, body mass index = body weight/body height.^{2}
Ideal body weight for height (%) = (actual body weight/ideal body weight) × 100%.
NS, not significant, $p>0.05$.

All except three subjects underwent two consecutive sleep assessments and the nocturnal sleep variables were calculated as the mean data of two nights. Two obese children were found to have very severe SDB (one had RDI = 62.6 and the other child had RDI = 106.1) on the first night sleep assessment. In view of their severe SDB, immediate treatment with CPAP were started on the following night. One of them also underwent MSLT on the day following the first night. The third one (control subject) had a signal problem in second night recording. There were no statistical differences in the sleep architecture and MSLT between the obese and control groups (table 2). The sleep architecture and stages between subjects with SDB and those without SDB were similar, except that SDB subjects had higher nocturnal arousal index than non-SDB (11.3 (SD 8.9) *v* 4.3 (SD 2.0), $p<0.01$). The mean sleep latency time in the SDB group (14.3 (SD 3.5) min) also did not differ from non-SDB (15.3 (SD 3.9)) in the MSLT.

Table 3 compares respiratory indices between obese and normal children. In particular, obese children (3.4, SD 10.7) had a higher obstructive apnoea index (OAI) than the control group (0.3, SD 0.8) ($p<0.05$). Obese children (9.3, SD 18.7) had a higher RDI than the control subjects (2.0, SD 1.5) ($p<0.05$). ODI was also significantly higher in obese (9.8, SD 21.4) than control subjects (1.2, SD 1.1) ($p<0.05$). The peak $ETCO_2$ values did not differ between the normal control and obese children.

One of the main problems in diagnosing SDB in children is the lack of unanimously agreed criteria.[4 18] Table 4 summarizes the results of the current study by using different diagnostic criteria. While mild respiratory disturbances, especially of the central type, were commonly seen in both groups, obese children differed from the control subjects by having more obstructive sleep apnoea and hypopnoea. If we used RDI $\geq$5 as the diagnostic cut off, 32.6% (n = 15) of obese children and 4.5% (n = 2) of normal controls satisfied the criterion. If we used OAI $\geq$1 as the diagnostic cut off, 26.1% (n = 12) of obese subjects and only one of the normal weight controls (2.3%) fulfilled the criterion.[18]

There was a clear predominance of boys over girls in suffering from SDB. Although mild sleep related respiratory disturbances were commonly seen in girls, those who fulfilled the diagnostic criteria (OAI $\geq$1 or RDI $\geq$5) of childhood SDB were all boys (male to female ratio: 17.8% *v* 0%, $p<0.05$). Although the girls were similar in age and height to the boys, the obese girls were much lighter than the obese boys in BMI (24.95, SD 3.95 *v* 28.41, SD 5.28, $p<0.05$) and IBW for height (142.1, SD 13.06 *v* 162.8, SD 27.79, $p<0.05$).

Table 2 Sleep architectures of obese and control groups

	Obese group (n = 46)		Control group (n = 44)	
Total sleep time, TST (min)	542.9	(15.8)	542.3	(15.3)
Actual sleep time, AST (min)	459.1	(51.1)	464.3	(46.0)
Sleep efficiency, SE (%)	84.6	(9.1)	85.6	(8.0)
Sleep onset latency, SOL (min)	23.3	(22.1)	22.3	(15.4)
REM onset latency, ROL (min)	132.1	(46.7)	117.2	(32.3)
Stage 1 (Tis%)	5.4	(2.1)	5.7	(2.6)
Stage 2 (Tis%)	48.2	(6.3)	48.6	(7.9)
Stage 3 (Tis%)	7.4	(2.7)	6.7	(2.6)
Stage 4 (Tis%)	19.5	(6.9)	18.7	(7.3)
REM sleep (Tis%)	19.5	(4.3)	20.2	(3.8)
MSLT (min)	15.5	(3.4)	14.7	(4.2)

All data were mean values of two nights except for three subjects (see text).
SD in parentheses.
Tis%, % of total time in sleep.
MSLT, all except one obese subject.

The obese children had more adenoid enlargements and narrower velopharyngeal space than the control subjects (table 5). The obese children also had more tonsillar enlargements, but the difference did not reach statistical significance. By using stepwise logistic regression with the factors (sex, age, body weight, height, BMI, IBW, size of adenoid, tonsil, nasal turbinate, and velopharyngeal isthmus) as the independent variables and RDI ≥ 5 as the dependent variable, SDB was significantly related to tonsillar size of greater than 2 (range 0–4) (OR 12.67, 95% CI 2.14 to 75.17, $p = 0.005$) and BMI (OR 1.20, 95% CI 1.08 to 1.33, $p = 0.001$). BMI and tonsil size did not show any interaction ($p = 0.647$) in the logistic regression. If we replicated the regression with the use of diagnostic criterion of OAI ≥ 1, the same risk factors, BMI and tonsillar enlargement, were identified. RDI and OAI were highly correlated (Pearson correlation coefficient = 0.942, $p<0.005$). Thirteen subjects were identified as cases by both RDI and OAI, and four subjects were identified as cases by RDI only. Using tonsil size greater than 2 as a screening test for SDB in the obese children group, positive predictive value (PPV) for predicting SDB was 83.3%, negative predictive value (NPV) was 78.9%, sensitivity (SN) was 38.5%, and specificity (SP) was 96.8%.

DISCUSSION

Both normal and obese children slept well in the sleep laboratory. Both groups slept for about nine hours, with satisfactory sleep efficiency of 85%. Obese children had similar sleep architecture to the normal weight controls, although they had more respiratory disturbances. When the SDB group was compared with the non-SDB group, both had similar sleep architecture, except for higher arousals in the SDB group. This observation of relatively normal PSG data in the presence of SDB supports previous findings that the sleep architecture of children was more robust to any fragmentation and arousal induced by respiratory events.[23–25] Alternatively, it could be suggested that the degree of SDB in children is generally mild when compared with that of adult SDB. The sleep fragmentation and/or oxygen desaturation was not of a sufficient degree to contribute to daytime sleepiness. Thus, these two factors together may help to explain the normal MSLT in our study and the lower prevalence of daytime somnolence in childhood SDB in general.

Mild sleep related respiratory disturbances were also commonly seen in the normal weight controls; 16% reported habitual snoring. This was similar to most studies that reported about 10–20% of white children snored regularly.[26 27] The current study further replicated the common finding of frequent occurrences of mild respiratory disturbances during sleep, mainly in the form of central apnoeas and hypopnoeas in normal young children.[18] Depending on the criteria used, 4.5% (RDI ≥ 5) or 2.3% (OAI ≥ 1) of normal subjects would be suffering from SDB. This was comparable to the previously reported prevalence rate of SDB in 1–3% of white children in the community.[4 27] Although ethnicity, especially African-Americans in the United States and Indian descendants in the United Kingdom, has been suggested to be the risk factor for childhood SDB and general sleep problems respectively,[2 28] our current study found a similar prevalence rate of SDB in Chinese normal-weight subjects. Further epidemiological studies of Chinese population will be needed to clarify the ethnicity issue in childhood SDB.

There was a predilection of male obese subjects suffering from SDB in our studies. This may be due to the fact that the obese boys were much heavier than the girls. In our study, neither sex nor age were risk factors for childhood SDB.

The obese children were different from their normal weight controls in terms of type (predominantly obstructive), frequency, and severity of respiratory disturbances. About one third of obese children were found to have SDB (RDI

Table 3 Respiratory indices of obese and control groups

	Obese group		Control group		p value
Obstructive apnoea index (OAI)	3.44	(10.65)	0.26	(0.81)	<0.05
Central apnoea index (CAI)	0.39	(0.99)	0.38	(0.45)	NS
Apnoea index (AI)	3.9	(10.88)	0.67	(0.92)	NS
Hypopnoea index (HI)	5.36	(9.11)	1.28	(0.93)	<0.01
Respiratory disturbance index (RDI)	9.3	(18.7)	2.01	(1.5)	<0.02
Oxygen desaturation index (ODI)	9.77	(21.38)	1.20	(1.11)	<0.01
Arousal index	6.55	(6.55)	4.69	(2.26)	NS
Peak end tidal CO_2*	50.99	(9.18)	50.34	(6.27)	NS

All data are mean values of two nights. All indexes are events/hour.
SD in parentheses.
*Not all subjects had the end tidal CO_2 measurement.
NS, not significant, $p>0.05$.

Table 4 Number of subjects satisfying different criteria of SDB in children

	Obese group		Control group		
	Yes (%)	No (%)	Yes (%)	No (%)	p value
AI					
⩾1	14 (30.4)	32 (69.6)	10 (22.7)	34 (77.3)	NS
⩾5	7 (15.2)	39 (84.8)	1 (2.3)	43 (97.7)	NS
O-AI					
⩾1	12 (26.1)	34 (73.9)	1 (2.3)	43 (97.7)	0.002
⩾5	7 (15.2)	39 (84.8)	1 (2.3)	43 (97.7)	NS
RDI					
⩾1	36 (78.3)	10 (21.7)	31 (70.5)	13 (29.5)	NS
⩾5	15 (32.6)	31 (67.4)	2 (4.5)	42 (95.5)	0.001
ODI					
⩾1.4	28 (39.1)	18 (60.9)	12 (27.3)	32 (72.7)	0.002
Peak P_{ETCO2} >53 mm Hg*	9 (26.5)	25 (73.5)	5 (12.5)	35 (87.5)	NS

All data were mean values of two nights. All indexes were events/hour.
AI: apnoea index = total number of obstructive and central apnoea episodes per hour of sleep.
O-AI: obstructive apnoea index = number of obstructive apnoea (OA) episodes per hour of sleep.
RDI: respiratory disturbance index = total number of apnoea and hypopnoea episodes per hour of sleep.
ODI: oxygen desaturation index = total number of dips in SaO_2 greater than 4% per hour of sleep.
Peak P_{ETCO2} >53 mm Hg: peak end tidal CO_2.
NS: not significant by χ^2 test, p>0.05.

⩾5). The current finding was similar to previous uncontrolled western studies that reported at least one third of obese children were suffering from significant SDB.[6] The presence of tonsillar enlargement had a much higher odds ratio than obesity alone in predicting SDB in children. However, both tonsillar hypertrophy and obesity were significant and independent risk factors in predicting SDB in young children. In this study, there is a high specificity but low sensitivity in using tonsillar size in predicting the occurrence of obstructive SDB in obese children. The overall results suggest a modest argument for using ENT evaluation as a screening tool for SDB in obese children. In other words, those obese children with obviously enlarged pharyngeal tissues will have a high chance of suffering from obstructive SDB. As the use of PSG in diagnosing and confirming SDB is costly and requires a long waiting time, more studies in investigating the use of upper airway ENT examination as a screening tool in obese children are indicated.

The exact pathophysiology of childhood SDB is not well understood and is likely to have a multifactorial aetiology.[24] The close interaction between genetic factors, central neuromotor and ventilatory control, and upper airway narrowing may culminate into clinical presentation of SDB in children.[24] Thus, obesity could predispose to SDB in children through the mass loading of upper airway and respiratory muscles as well as impairment in ventilatory control.[6 24 29 30] It is apparent that obese children, by having a narrower velopharyngeal airway, will further compromise their airway patency in the presence of soft tissue enlargement.[24 31] In short, the presence of slight pharyngeal lymphoid tissue enlargement in an obese child should raise the high index of suspicion of SDB and suggest an aggressive approach of surgical intervention.

Table 5 The status of adenoid, tonsil, turbinate enlargement, and velopharyngeal isthmus in both obese and control groups

	Obese group* n=44	Control group n=44	p value
Adenoid†			
0–2	35	43	
3–4	9	1	<0.05
Tonsil†			
0–2	38	42	
3–4	6	2	NS
Nasal turbinate†			
0–2	36	35	
3–4	8	9	NS
Velopharyngeal isthmus‡			
0–2	16	7	
3–4	28	37	<0.05

*Two obese subjects were reluctant to undergo ENT examination.
†All the sizes of adenoid, tonsil, and turbinate enlargement were measured from grade 0 (no enlargement) to 4.
‡The smaller the size, the narrower the breathing airway.

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ARCHIVIST

Uveitis

About one in twenty patients with intraocular inflammation (uveitis) is a child. Uveitis in children differs from that in adults because of its association with juvenile idiopathic arthritis (JIA) and because of its insidious onset. The latter feature means that uveitis may often be diagnosed only at screening and ocular complications may be present already. Up to a third of children with uveitis may develop severe visual impairment. Ophthalmologists in Utrecht (J de Boer and colleagues. *British Journal of Ophthalmology* 2003;**87**:879–84) have reported a retrospective study.

The study included 123 consecutive patients (69 girls) who developed active uveitis at age 2–15 years (mean 8 years). One hundred and two patients (83%) had chronic uveitis and in 88 (72%) it was bilateral. Thirty six children (29%) had associated systemic disease. Twenty five of these had JIA (22 ANA positive oligoarticular JIA), and the other 11 had a variety of diagnoses: sarcoidosis (n = 3, only one with raised serum angiotensin converting enzyme concentration), chronic infantile neurological cutaneous and articular/neonatal onset multisystem inflammatory disease (CINCA/NOMID) syndrome (n = 3), tubulointerstitial nephritis and uveitis (TINU) syndrome (n = 2), psoriasis (n = 1), multiple sclerosis (n = 1), and masquerade syndrome (n = 1). Twelve children had toxoplasmosis and four herpesvirus infection. Five had specific ocular disease and in 66 cases the cause of the uveitis was not discovered.

The uveitis was anterior in 44 patients, intermediate in 30, posterior in 23, and throughout the eye (panuveitis) in 26. The uveitis of JIA was anterior (n = 18) or panuveitis (n = 7). Toxoplasmosis caused only posterior uveitis and herpesvirus only anterior. No cause was discovered for intermediate uveitis.

Ninety three patients (76%) had ocular complications including cataract (n = 43), papilloedema (n = 36), glaucoma (n = 23), and cystoid macular oedema (n = 21). Thirty five (28%) needed intraocular surgery and 57 received systemic medication including steroid (n = 39), methotrexate (n = 11), and cyclosporin (n = 4).

Three children became blind in both eyes and 20 in one eye. Eight had unilateral visual impairment (visual acuity 0.3 or less). Of the 25 patients with JIA, one developed bilateral blindness and four unilateral blindness. The most frequent causes of blindness were macular scars (most often from toxoplasma retinochoroiditis) and secondary glaucoma (most commonly with JIA).

Uveitis in childhood often becomes chronic and has a serious ocular prognosis. It may develop years after the onset of JIA and repeated ocular screening is necessary for patients with JIA.