ORIGINAL ARTICLE

Evidence for persistence of upper airway narrowing during sleep, 12 years after adenotonsillectomy

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Aims: To establish whether subjects with previous evidence of sleep apnoea prior to adenotonsillectomy continue to have evidence of narrower upper airways during sleep, 12 years later.
Methods: Twenty subjects (median age 16 years) underwent repeat sleep studies at home, 12 years after such studies had shown significant sleep apnoea in many of them prior to an adenotonsillectomy. Twenty control subjects, also studied 12 years ago, underwent repeat home sleep studies as well. The sleep studies provided information on snoring, hypoxia, and inspiratory effort (from measures of pulse transit time). A questionnaire was also administered, the subjects were weighed, and their heights measured.
Results: There was more reported snoring in the previous adenotonsillectomy group (50% versus 20%) and also during the sleep study (80 versus 31 snores per hour). The measure of inspiratory effort overnight was higher in the previous adenotonsillectomy group (15.6 versus 12.3 ms). Allowance for potentially confounding variables (obesity and nasal congestion) partially reduced the statistical significance of the difference in snoring, but not that of the measure of inspiratory effort.
Conclusion: Results suggest that a narrower upper airway during sleep, to the point of snoring, persists 12 years after adenotonsillectomy, and may partly account for the occurrence earlier of preoperative sleep apnoea while adenotonsillar hypertrophy was present. It is not known if this narrowing is one of the risk factors for later development of adult sleep apnoea.

The exact prevalence of significant obstructive sleep apnoea (OSA) in children is not known. In an unselected population the estimates vary between 0.5 and 3%[1 2]; the exact figure depends on the definition used. The commonest reason for the upper airway narrowing during sleep in young children is adenotonsillar hypertrophy. However, not all children with adenotonsillar hypertrophy have snoring or OSA, and the assumption is that some other factor contributes to its development. There is some evidence that this may be subtle differences in craniofacial anatomy,[3] which might also be expected to predispose to sleep apnoea in adulthood.[4 5]

Eleven years ago we published sleep study data before and after adenotonsillectomy in a group of children who also snored, compared to normal control children.[6] Following adenotonsillectomy there was good resolution of hypoxic dipping and sleep disturbance during the night, but not all children achieved the low levels found in the control group.

We have restudied some of these children 12 years later to see whether they have evidence of increased upper airway narrowing during sleep, and compared them to some of the original control children.

METHODS

Subjects

In our original study, published in 1990, during one year we selected 61 children aged between 2 and 14 years who had been placed on the waiting list for adenotonsillectomy, after referral to the ear, nose, and throat department by their general practitioners for recurrent tonsillitis, and whose parents said that they snored.[6] We received 90 notifications of such children, out of a total of 324 listed for adenotonsillectomy over the same year. Sixty one of these children had preoperative, and six months postoperative, sleep studies and formed the study group. The other 29 children either refused to be studied, or one of their two sleep studies was technically unsatisfactory. Although listed for adenotonsillectomy, in the event, seven underwent tonsillectomy alone, eight adenoidectomy alone, and 46 both. Only in one child had there been preoperative concern over OSA.

Thirty six healthy children were recruited via health visitor clinics, children of colleagues, and healthy siblings of the patients. They were selected to provide a similar age and sex distribution to the patients. Thirty one of these had sleep studies on both occasions, six months apart, and formed the control group. In the other five children, one of their two sleep studies was technically unsatisfactory.

Of the original 61 children in the adenotonsillectomy group, we were able to contact 20 who were still living within a reasonable distance of Oxford for home visiting, all of whom agreed to a repeat sleep study. Of these 20, two had had tonsillectomy alone, three adenoidectomy, and 15 both. Of the original 31 children from the control group, we were able to contact 21, 20 of whom agreed to a repeat sleep study.

Techniques

Questionnaire, anthropometry, and blood pressure

A simple questionnaire, including the Epworth sleepiness score (ESS)[7] was administered on the evening of the study. This included questions on snoring, alcohol consumption, cigarette usage, nasal congestion, previous tonsillectomy, other medical problems, medications, socioeconomic status,[8] and school or work absences caused by illness. Measurements were also made of height, weight, neck circumference, and overjet (distance between upper and lower incisors with teeth clenched in normal bite). Blood pressure was measured three times using a validated, automated device.[9] Patients were taught to measure their own blood pressure (BP) and did so

Abbreviations: BP, blood pressure; EEG, electroencephalogram; ESS, Epworth sleepiness score; OSA, obstructive sleep apnoea; PTT, pulse transit time

three times on the evening of study, and again the following morning, on awakening.

Sleep study

Sleep studies were performed in the subjects' own homes using RM50 portable monitors (DeVilbiss, London, UK). These devices record for eight hours and can be programmed to turn on and off at particular times (usually 2300 and 0700 respectively). The RM50 records oxygen saturation from a finger probe, snoring via a throat microphone, body position via a sensor in a chest box (held on by one chest band), chest movements, heart rate (from 3 ECG electrodes), and pulse transit time (PTT). The latter is the time delay between the ECG R wave and the arrival of the pulse wave at the finger, detected from the transmittance signal of the oximeter probe. This value is typically about 250 ms and varies inversely with BP. As BP falls, tension in the arterial wall falls, and the PTT increases; and vice versa. Thus beat to beat measures of PTT provide an estimate of beat to beat changes in blood pressure[10 11]; by chance a change of 1 ms is about equivalent to a change of 1 mm Hg in BP. The snoring detector was a small electret microphone, sealed into a small chamber stuck onto the skin 3–4 cm lateral to the thyroid cartilage prominence. There was a vented air gap between the skin and microphone, exactly similar to the arrangement used in the MESAM sleep monitoring system.[12] The threshold used to define a snore by the throat microphone was set to be approximately equivalent to a snore volume of 55 dB, measured 1 metre in front of the mouth; this was determined by experimentation. The snoring detectors on each RM50 were calibrated regularly, using a specially constructed rig to ensure constancy throughout the study; changes in this calibration were only found to occur when microphones were replaced following breakage.

Analysis of these signals for this study provided the following derivatives: the number of >4% dips in oxygen saturation per hour of study,[13] minimum and mean oxygen saturation overnight, numbers of snores per hour of study (where one inspiration accompanied by snoring equals one snore), number of rises in BP per hour of study,[14 15] number of heart rate rises per hour of study,[15] and the average fall in BP caused by each inspiration (pulsus paradoxus) across the whole study.[16 17]

In order to extract BP rises, the raw PTT tracing was automatically processed by the RM50 software to first remove artefact. The difference between adjacent PTT values was computed; if it exceeded 50 ms (physiologically very unlikely), then the start of an artefact was noted. The software continued to record an artefact until three consecutive PTT differences were <50 ms. Missing data were filled in by linear interpolation for a maximum of 20 seconds, after which a gap was left in the record. Data were next passed through a 17 sample (3.4 second) moving window average to remove most of the breath by breath oscillations. Finally, to identify arousal related BP rises, sequential peaks and troughs in the data were detected if they conformed to the following criteria: a fall in PTT of >15 ms, lasting for >5 and <45 seconds. This particular threshold and time window derive from previous work in normal subjects.[15 18] These "BP arousals" correlate well with electroencephalogram (EEG) based definitions of arousal, and also predict daytime sleepiness as effectively as EEG arousals.[14 19 20]

In order to extract measures of inspiratory effort, the same artefact removal process was applied to the raw PTT data as above, except that only 1 second of missing data caused by artefact was filled in by linear interpolation, before a gap was left in the record. Data were then passed through a 0.6 second moving window average. Finally, sequential peaks and troughs in the data were detected to identify inspiratory BP falls (rise in PTT) lasting >0.7 and <4.5 seconds. This definition of inspiratory effort was adapted from our earlier work.[21] The program then counted the number of breaths detected, and calculated the mean of all these breath by breath PTT changes for the whole night's record. Oscillations in BP and pulse transit time over this time course of 0.7 to 4.5 seconds reflect quantitatively the inspiratory effort,[16 17] and have also been shown by other workers to be useful in differentiating central from obstructive apnoeas, for example.[22 23] Thus the units for this index of inspiratory effort are in milliseconds, and the overnight average could represent up to 7000 or more breaths.

Previous work from this unit to assess the reproducibility of these two PTT derivatives compared two separate overnight recordings in 40 adult patients with a wide range of severity of sleep disordered breathing. Both "BP arousals" and mean all night inspiratory effort showed good reproducibility; correlation coefficients between night 1 and night 2 were 0.81 and 0.87 respectively.[20] This non-invasive approach to measuring arousals and inspiratory effort has also been validated recently in children.[24]

Statistics

Data were analysed using the SPSS statistical package (version 7.5.1, Chicago, USA). The statistical significance of any differences between the groups was assessed using the non-parametric Mann–Whitney test, as some of the data are not normally distributed. Categorical data were compared using the χ^2 test. The possibility of confounding variables was explored using multiple logistic modelling. Given the number of comparisons, statistical significance values between 0.05 and 0.01 should be interpreted with caution.

The original study in 1990 was approved by the Central Oxford Research Ethics Committee (no. 1359), as was this study (no. 99.194), which was funded from charitable donations.

RESULTS

Tables 1 and 2 show the data from each of the two groups. There were significant differences between the groups for social class, questionnaire defined snoring, presence of nasal congestion, history of other medical conditions, days off work or school because of illness, weight, and body mass index. There were two differences between the groups in the sleep studies: the number of snores registered (almost three times as many in the prior adenotonsillectomy group), and the measure of inspiratory effort (PTT, more than 25% higher in the prior adenotonsillectomy group).

These measures of snoring and inspiratory effort were also analysed using means, rather than medians, in order to provide the average differences and their 95% confidence intervals (CI). In the control and adenotonsillectomy groups respectively, the snoring levels (number per hour) were 31 (SEM 7) and 80 (SEM 15) (difference 49, 95% CI: 14 to 84); and the PTT estimates of inspiratory effort (ms) were 12.3 (SEM 0.7) and 15.6 (SEM 0.8) (difference 3.3, 95% CI: 1.2 to 5.4). There were no differences between the groups in any of the recorded BP values, or the changes in BP from evening to morning.

Multiple logistic regression analysis, with "control" or "adenotonsillectomy" group as the dependent variable, showed that none of the higher values of the PTT estimate of inspiratory effort in the adenotonsillectomy group could be explained by any of the potentially confounding variables of obesity, neck circumference, or a history of nasal congestion (singly or in combination). The statistical significance of the higher level of snoring in the adenotonsillectomy group was, however, reduced by allowing for obesity and nasal congestion first, but did remain statistically significant ($p = 0.05$).

If both groups were analysed together, snoring overall was best predicted by the neck circumference, as it is in adults.[25 26] There were no significant predictors of daytime sleepiness (ESS).

Table 1 Basic data on prior tonsillectomy group (n = 20) and control group (n = 20)

	Control group, medians	5th/95th centiles	Prior adenotonsillectomy group, medians	5th/95th centiles	p value of comparison* where <0.05
Age at 1st study (y)	4.0	3.0/9.8	4.0	2.0/6.0	
Age at 2nd study (y)	16.0	13.0/20.8	16.0	14.0/18.0	
Males:females	9:11		11:9		
Social class†	7:9:0:1:0:0		1:6:3:7:0:1		0.009
ESS	5.5	0.0/12.0	6.5	0.0/12.0	
Snoring (yes:no)	4:16		10:10		0.047
Alcohol (units/day)	0.0	0.0/1.9	0.0	0.0/5.5	
Cigarettes (no./day)	0.0	0.0/14.5	0.0	0.0/19.8	
Nasal congestion (yes:no)	7:13		14:6		0.027
Adenoidectomy, tonsillectomy, or both (yes:no)	0:20		20:0		<0.001
Other medical problems (yes:no)	1:19		7:13		0.018
Current medications (yes:no)	2:18		7:13		(0.058)
Absences (days/y)	3.0	0.0/23.3	9.0	3.0/15.0	<0.001
Weight (kg)	59.0	37.0/69.3	66.5	48.2/106. 7	0.039
Height (m)	1.68	1.48/1.84	1.69	1.58/1.88	
BMI (kg/m^2)	21.0	15.0/24.1	23.0	17.1/37.0	0.027
Neck circumference (cm)	33.5	28.1/38.0	34.5	31.0/41.9	(0.054)
Overjet (mm)	3.0	2.0/4.0	3.0	2.0/4.0	

*Statistical significance of any differences using Mann–Whitney test (continuous variables) or χ^2 (discontinuous data).
†Social class by father's occupation[8]: I, II, IIIn (non-manual), IIIm (manual), IV, and V.

Table 2 Sleep study and blood pressure data on the two groups

	Control group, medians	5th/95th centiles	Prior adenotonsillectomy group, medians	5th/95th centiles	p value of comparison* where <0.05
Mean overnight Sa_{O_2} (%)	97.0	96.0/98.0	97.0	96.0/98.0	
>4% Sa_{o_2} dips/h overnight	0.0	0.0/1.0	0.0	0.0/1.0	
Snores/h overnight	25.0	5.0/64.2	73.5	8.2/247	0.02
Heart rate arousals/h overnight	27.0	11.0/59.3	27.8	12.0/104	
PTT arousals/h overnight	47.0	15.0/82.9	36.0	15.0/61.2	
Mean PTT swing (ms)	11.9	8.0/16.8	15.0	10.1/20.23	0.002
Systolic BP am	114.5	86.4/133.5	111.5	99.1/129.9	
Diastolic BP am	66.0	49.0/87.0	68.5	50.2/87.8	
Systolic BP pm	122.0	100.1/148.5	116.0	100.4/132.8	
Diastolic BP pm	71.0	49.2/78.9	63.0	38.0/82.0	
Systolic BP, am–pm difference	–8.5	–36.8/+6.9	–6.0	–17.9/+6.9	
Diastolic BP, am–pm difference	+2.00	–14.9/+15.8	–1.0	–43.8/+13.0	

*Statistical significance of any differences using Mann–Whitney test.

DISCUSSION

This study has shown that there remain differences between the two groups of subjects studied, those who 12 years earlier had been snorers and listed for adenotonsillectomy for recurrent tonsillitis, and a matched group of control children. The original sleep studies on the two groups showed considerably more hypoxic dipping and sleep disturbance in the adenotonsillectomy group, compared to the control group.[6] Although there was considerable resolution of hypoxic dipping and sleep disturbance six months postoperatively, not all children achieved the low levels found in the control group, suggesting possible residual abnormalities of upper airway function during sleep.

The main differences we have found on this occasion support the hypothesis that children in the prior adenotonsillectomy group retain narrower upper airways during sleep. The greater presence of snoring on both the questionnaire, and during the sleep study, in the prior adenotonsillectomy group suggests this, although the statistical significance of this compared to control children was slightly reduced by first allowing for obesity and nasal congestion. However, the higher levels of inspiratory effort, unaffected by first allowing for obesity and nasal congestion, is further compelling evidence. We had thought that the greater number of days off sick from work or school in the prior adenotonsillectomy group might have been a result of consequences of mild sleep apnoea; however, this difference proved to be explicable by the confounding variable of social class. In a previous prevalence study of sleep apnoea in children from this unit,[1] we showed that lower social class was one of the predictors of OSA, as was the co-correlate of maternal smoking. This, and the method we used to recruit control children (which included approaching the children of colleagues), produced a difference in social class distribution; at the time of designing the original study, we were not aware that this might be important. Thus social class matching was unfortunately not added to the other matching criteria of age and sex.

Further support for the absence of any symptomatic consequences from this increase in snoring and inspiratory effort, were the similar levels of nocturnal micro-arousals (from the autonomic indices, heart rate and BP rises) and daytime sleepiness (ESS). In addition, there was no effect of this increased inspiratory effort on evening to morning BP, as has been shown to occur in adults.[27]

Since not all obese adults, and children with enlarged tonsils, get sleep apnoea, it is apparent that other factors must contribute to its development. In adults, cephalometric analysis has clearly shown the importance of changes in craniofacial shape.[3] In its most notable form, these changes consist of increased face height in the lower third of the facial skeleton, posterior buccal cross bite, high palate, steep mandibular plane, and an overjet or class II occlusion.[28–30]

These changes probably reduce the retroglossal space. In children it has been shown that in addition to tonsillar size, a reduced distance between the lateral pharyngeal walls is a contributing factor.[4 5] Such craniofacial shapes may predispose both children and adults to OSA by making them vulnerable to the extra pharyngeal crowding brought about by either adenotonsillar hypertrophy or obesity respectively. A further link between paediatric and adult sleep apnoea comes from the suggestion that adenoidal enlargement and nasal obstruction at a very young age, by provoking mouth breathing, may lead to the very changes in lower facial shape described above that are thought to contribute to airway crowding.[31–33] If nasal patency is improved early enough, then there may be a mandibular growth spurt with a return to a more normal shape.[34 35]

We have no evidence that our findings are in any way related to the future development of obstructive sleep apnoea, symptomatic or otherwise. However, these findings are one further piece of evidence that a predisposition to snoring and OSA in both children and adults may result from the common risk factor of a small upper airway, even though the usual added precipitant is different in each case—adenotonsillar hypertrophy and obesity respectively.

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