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.
Results: There was more reported snoring in the previous adenotonsillectomy group (50% versus 20%) and also during the sleep study (80 versus 31 snorers 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.

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. In the original 31 children from the control group, we were able to contact 21, 20 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), 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, 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. Patients were taught to measure their own blood pressure (BP) and did so weekly.

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 pressure10 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 14 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,14 and the average fall in BP caused by each inspiration (pulsus paradoxus) across the whole night inspiratory effort showed good reproducibility; correlations of comparisons, statistical significance values between 0.05 and 0.01 should be interpreted with caution.

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 seconds of interpolation for a maximum of 20 seconds, after which a gap was left in the record. Data were next passed through a 17

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 χ2 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 no 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).
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. 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, 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.

We showed that lower craniofacial shape.

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.
Upper airway narrowing during sleep after adenotonsillectomy

These changes probably reduce the retroglottal 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. 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 narrowing. If nasal patency is improved early enough, then there may be a mandibular growth spurt with a return to a more normal shape.

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.

References