Influence of uvulopalatopharyngoplasty on α -EEG arousals in nonapnoeic snorers

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ABSTRACT: Arousals are more numerous in heavy snorers than in nonsnorers and might be a cause of excessive daytime sleepiness (EDS) in these patients. The present study investigated whether treatment of snoring by uvulopalatopharyngoplasty (UPPP) had an influence on sleep microstructure in nonapnoeic snorers.

The polysomnographic records of 10 nonapnoeic snorers were reviewed retrospectively and arousals scored according to the American Sleep Disorders Association (ASDA) 3 s definition. Scores for snoring, EDS and polysomnographic data were compared before and after UPPP (mean (±sD) time interval 249±183 days).

UPPP resulted in a subjective improvement of snoring and a significant decrease in the arousal index (mean 14.6, 95% confidence interval (95% CI) 8.5–20.8 vs mean 9.1, 95% CI 6.6–11.5) (p=0.01). EDS and the amount of slow-wave sleep remained unchanged.

Uvulopalatopharyngoplasty resulted in an improvement of subjective snoring and a significant decrease of arousals in nonapnoeic snorers. Although these data do not provide any insight into whether the improvement observed can be maintained on a long-term basis, uvulopalatopharyngoplasty can be considered as a useful treatment modality to reduce sleep fragmentation and snoring in nonapnoeic snorers.

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At present, it is generally agreed that uvulopalatopharyngoplasty (UPPP) is an efficient treatment modality for snoring, at least when short-term results are considered. Fujita et al. [1] introduced UPPP as a procedure to correct obstructive sleep apnoea, and reported a marked relief or elimination of snoring in 94% of their patients. A long-term longitudinal study on the success of UPPP to control snoring was recently published by LEVIN et al. [2]. In 87% of the 69 patients studied, snoring was initially significantly reduced or totally eliminated. However, after 13 months, the success rate dropped to 46%. Excessive daytime sleepiness (EDS) and daytime dysfunction are major characteristics of sleep apnoea syndrome and are also often observed in snorers. However, at present, no firm conclusion can be drawn about snoring as a cause of EDS in nonapnoeic snorers [3]. There is no conclusive evidence that snoring is linked to nocturnal hypoxaemia, which might be a cause of daytime dysfunction [3].

Data published by Guilleminault and co-workers [4] support the hypothesis that arousals, related to an increased upper airway resistance (UAR), play a role in the pathogenesis of daytime sleepiness in nonapnoeic snorers. The upper airway resistance syndrome (UARS), as defined by Guilleminault and co-workers [4], is characterized by an increased UAR during sleep as a cause of arousals and excessive daytime sleepiness. Snoring is associated with an increased UAR but the reverse is

not always true: approximately 30% of the patients with UARS described by Guilleminault and co-workers [4], were not habitual snorers.

Jansson et al. [5] reported a significant subjective improvement of excessive daytime sleepiness and fatigue in nonapnoeic snorers after UPPP. However, their study had some limitations. The diagnosis of nonapnoeic snoring was based only on oxygen desaturation at night and there were no objective measurements at follow-up. Therefore, the study by Jansson et al. [5] did not provide an answer to the question of whether the improvement of excessive daytime sleepiness is related to an improvement in sleep microstructure. In an attempt to investigate the influence of UPPP on sleep microstructure, 10 nonapnoeic snorers were studied retrospectively. Once polysomnographic data were obtained for all patients, the sleep records were reviewed and arousals scored. The short-term results of UPPP with respect to snoring and sleep microstructure will be discussed.

Materials and methods

Subjects

For this study, the questionnaires and polysomnographic data of 10 consecutive patients, in whom UPPP was performed as a treatment for heavy, socially-disturbing

snoring, were retrospectively reviewed. Patients with concomitant lung disease were excluded.

Study design

All patients underwent a complete polysomnographic evaluation at the Sleep Disorders Unit of the Antwerp University Hospital, before surgery. A follow-up polysomnography was carried out at least 3 months after surgery. On both occasions, a standardized questionnaire for the assessment of snoring and EDS was completed by a physician during the intake [6]. Spirometric evaluation and arterial blood gas analysis during wakefulness were performed at baseline and follow-up. The sleep records were reviewed retrospectively and arousals scored for baseline and follow-up. Data obtained before and after surgery were compared for significant changes.

Methods

The answers to the questionnaire were quoted by means of a grading system: ranging from 0 = no snoring to 4 = socially-disturbing snoring (SN score); and 0 = no EDS to 3 = EDS that disables the patient whenperforming daily activities (EDS score). Answers to the questionnaire were available at baseline and follow-up for all patients (SN score) and for nine patients (EDS score). Polysomnographic registration included the following parameters (SAC recording, Oxford): electroencephalogram (EEG) (C3/A2 and C4/A1); right and left electro-oculogram (EOG); submental and anterior tibial muscle electromyogram (EMG). Chest wall and abdominal movements were measured by strain gauges. Oronasal airflow was detected using thermocouples and oxygen saturation was recorded using pulse-oximetry. Qualitative recording of snoring was performed using a microphone placed at the suprasternal notch. Before the recording was started, the patients were asked to make a snoring sound and the signals from the microphone were amplified in order to obtain a clear snoring signal

Sleep stages were scored according to the criteria of RECHTSCHAFFEN and KALES [7]. Sleep efficiency refers to the ratio of total sleep time (TST) to time in bed (TIB). An apnoea was defined as an interruption of oronasal airflow for at least 10 s. Three subtypes of apnoea were recognized, as defined by standard criteria: central, mixed and obstructive [8]. A hypopnoea was defined as a >50% reduction of oronasal airflow followed by a ≥4% drop in arterial oxygen saturation (Sa,O2) from the ongoing value and/or arousal. Apnoea + hypopnoea index (AHI) = number of apnoeas + hypopnoeas \times 60/TIB (min), and apnoea index (AI) = number of apnoeas \times 60/ TIB (min). In agreement with the study of Hoffstein et al. [9], nonapnoeic snorers were defined as snorers with AHI ≤10. Alpha-EEG arousals were scored according to the American Sleep Disorders Association (ASDA) 3 s definition as any shift in EEG frequency to alpha or theta for at least 3 s but not longer than 15 s [10]. Patients having other disorders that could be a reason for alpha-EEG arousals, such as periodic leg movement disorder, were excluded. The arousal index (ArI) is the total number of α -EEG arousals \times 60/TST (min).

UPPP was performed as described by SIMMONS *et al.* [11], and was the only type of surgery aimed to relieve snoring performed in these patients.

Analysis

Statistical analysis was performed with the Stat Soft Inc. 1993 software package. Values are presented as mean and 95% confidence interval (95% CI). Because of the small sample size, nonparametric statistics were used and data were compared by Wilcoxon matched pairs test. Statistical significance was assumed at a p-value less than 0.05.

Results

The study population included 6 males and 4 females. Patient characteristics at the time of diagnosis are presented in table 1. The patients were not overweight, and arterial blood gas values and lung function parameters were within normal limits. The values for snoring and EDS scores, before and after UPPP are presented in table 2. The mean (±sD) time elapsed between the diagnostic polysomnography and the follow-up examination was 249±183 days. None of the patients had long-term complications after UPPP. UPPP improved the subjective estimate of snoring. As shown in table 2, all patients reported major improvement in snoring, with one exception where a minor improvement occurred. Three patients reported no snoring and six patients reported intermittent snoring when lying on the back. Only two patients (case Nos. 8 and 9) reported an improvement of EDS (table 2). There was no change in arterial blood gas values, lung function or body mass index (BMI) after UPPP.

Table 1. - Patients characteristics at baseline

Age yrs	44 (36–51)
BMI Kg·m ⁻²	25.3 (23.2–27.5)
FEV ₁ % pred	100 (95–106)
FEV ₁ /VC %	106 (99–113)
Pa,O ₂ kPa	11.7 (10.7–12.7)
mmHg	87.8 (80.6–95.1)
Pa,CO ₂ kPa	4.8 (4.4–5.3)
mmHg	36.3 (32.7–39.9)

Values are presented as mean, and 95% confidence interval in parenthesis. BMI: body mass index; FEV1: forced expiratory volume in one second; VC: vital capacity; % pred: percentage of predicted value; P_{a,O_2} : arterial oxygen tension; P_{a,CO_2} : arterial carbon dioxide tension.

Table 2. – Scores for subjective estimate of snoring and excessive daytime sleepiness (EDS)

Case No.	Snoring baseline	Snoring follow-up	EDS baseline	EDS follow-up
1	4	1	0	0
2	4	1	0	0
3	4	0		
4	4	1	1	1
5	4	1	0	0
6	4	1	0	0
7	4	0	0	0
8	3	1	2	1
9	4	3	2	0
10	4	0	0	0

	Baseline	Follow-up	p-value
Stage I–II NREM %	48 (36–60)	56 (46–65)	0.3
Stage III–IV NREM %	6.2 (1.6–10.7)	4.6 (0.5–8.7)	0.3
REM %	13.4 (9.4–17.4)	12.6 (9.1–16.1)	0.8
Sleep efficiency %	68 (54–82)	73 (66–79)	0.6
Lowest Sa,O ₂ %	86 (86–87)	86 (83–89)	0.8
AI n×60/TIB	2.7 (0.4–5.0)	0.6 (0.2–1.0)	0.02*
AHI n×60/TIB	3.2 (1.0–5.4)	4.3 (1.5–7.1)	0.4
ArI n×60/TST	14.6 (8.5–20.8)	9.1 (6.6–11.5)	0.02*
α-EEG arousals	71.6 (55.9–87.2)	52.4 (36.9–67.9)	0.02*

Table 3. - Polysomnographic data at baseline and follow-up

Values are presented as mean, and 95% confidence interval in parenthesis. NREM: non-rapid eye movement sleep; REM: rapid eye movement sleep; sleep efficiency: TST/TIB ratio; S_{a,O_2} : arterial oxygen saturation; AI: apnoea index; TIB: time in bed (in min); AHI: apnoea/hypopnoea index; ArI: arousal index; TST: total sleep time (in min); EEG: electroencephalogram. *: p<0.05.

Results of the polysomnographic variables are listed in table 3. The only parameters that significantly improved after UPPP were the number of α -EEG arousals (p=0.02), the arousal index (p=0.01) and the apnoea index (p=0.02). Sleep efficiency and the amount of slow-wave sleep remained unchanged.

Discussion

A significant decrease of the arousal index and a subjective improvement of snoring was found in nonapnoeic snorers after UPPP.

We believe that the observed improvement of snoring is clinically relevant. All but one patient reported a complete disappearance or only intermittent snoring after surgery. The improvement of snoring confirms the statement of SIMMONS *et al.* [11] and many others that UPPP is an efficient operation to treat snoring. However, we did not measure snoring in a quantitative manner. Recently, the discrepancy between subjective estimates of snoring and objective measurements before and after UPPP, was stressed by MILJETEIG *et al.* [12]. In a series of 69 snorers, the overall reduction (or disappearance) of snoring was reported by 78%. However, there was no significant difference in any of the objective measurements of snoring (mean and maximal nocturnal snoring intensity).

There were no major changes in EDS after UPPP in the present study population. This is probably linked to the fact that only three patients suffered from EDS at baseline, two of these reported improvement after surgery. We cannot exclude the possibility that our scoring system for EDS lacks sensitivity and that more than three patients were sleepy to some degree before surgery. It might be that other scales, such as the Epworth Sleepiness Scale [13], are more appropriate to detect subtle differences in EDS. Another drawback is that we did not evaluate EDS in an objective way, for instance by means of multiple sleep latency tests (MSLT) or maintenance of wakefulness tests. The apnoea index decreased significantly and, after surgery, all patients had an AI <5.

There was a significant decrease in the number of α -EEG arousals after UPPP. At present it is rather difficult to determine whether this improvement is also

clinically important, since there is no consensus about the number of α -EEG arousals that can be considered as normal. Furthermore, data regarding the threshold of sleep fragmentation required to produce daytime dysfunction are lacking [13]. Based on a study in normals [14], Guilleminault and co-workers [4] chose an ArI of 10 as their cut-off point. MATHUR [15] studied normal subjects during the first night in the sleep laboratory and scored arousals in three different ways. Applying the 3 s ASDA definition, as in the present paper, the authors found a mean of 21 (95% CI 7–56) arousals per hour of sleep. Exclusion of those patients who reported snoring, daytime sleepiness or witnessed apnoea, did not change the results. In a study of 14 untreated, sociallydisturbing snorers, we found a mean ArI of 10.5 (SD 4.5) in nonsleepy patients, and a mean ArI of 11.6 (sp 5.3) in sleepy patients [16].

Although the number of patients in the present study is limited, some remarks concerning the role of UPPP in the treatment of nonapnoeic snoring can be made. A relationship between snoring, increased UAR and daytime dysfunction is an attractive hypothesis. Guilleminault and co-workers [4] treated their patients with UARS by nasal continuous positive airway pressure (CPAP). This resulted in a significant improvement of the mean arousal index and mean MSLT scores in these patients [4]. That CPAP is effective in eliminating snoring and the associated sleep fragmentation was confirmed by others [17]. Strollo and Sanders [17] recommend that CPAP or bi-level positive airway pressure (BiPAP) titration should be offered to nonapnoeic snorers complaining of EDS and with an increased number of EEG arousals. The question arises, however, whether nonapnoeic snorers will accept this efficient but constraining treatment modality. At this time, data concerning the tolerance and long-term benefit of CPAP for nonapnoeic snorers are scarce. In the above mentioned study by Guilleminault and co-workers [4], none of the nonapnoeic snorers desired CPAP treatment. KRIEGER et al. [18] reported a CPAP acceptance rate of 78% in nonapnoeic snorers.

It is generally assumed that UPPP decreases UAR. We are not aware of any study that has objectively measured UAR during sleep before and after UPPP. In the present study, we did not measure respiratory effort during sleep and so we can make no conclusion as to

whether the decrease of ArI was the result of a decrease in respiratory effort during sleep. De Backer and Van De Heyning [19] hypothesized that the role of UPPP in nonapnoeic snorers might be underestimated. Further studies are necessary to determine whether the decrease of α -EEG arousals, as observed in our nonapnoeic snorers, is due to a decrease of respiratory effort and upper airway resistance during sleep. If this could be confirmed, uvulopalatopharyngoplasty might be a useful treatment modality to reduce sleep fragmentation and excessive daytime sleepiness in nonapnoeic snorers and patients with an upper airway resistance syndrome.

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