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Outcome of sleep endoscopy in obstructive sleep apnoea: the Antwerp experience

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Abstract. *Outcome of sleep endoscopy in obstructive sleep apnoea: the Antwerp experience.*
Objectives: Snoring and obstructive sleep apnoea (OSA) result from upper airway (UA) collapse during sleep. Sleep endoscopy is a dynamic evaluation of the UA that can be used to determine the site(s) of collapse during respiratory events. This study evaluates the feasibility and outcome of sleep endoscopy in patients with OSA, compares the findings with the literature, and reviews the therapeutic advice given to patients.
Methodology: A retrospective analysis was conducted of the data for 70 OSA patients in whom UA surgery was considered. Sleep endoscopy was performed after IV administration of midazolam and propofol. The UA was visualised and assessed for the location of UA flutter, narrowing or collapse. Feasibility and safety were evaluated retrospectively. Outcome data were described as type and pattern of flutter and/or collapse. Treatment advice given to the patients was reviewed.
Results: Sleep endoscopy showed monolevel palatal collapse in 31.9%, monolevel tongue/hypopharyngeal collapse in 27.8% and multilevel collapse in 31.9% of patients. In 5.6% of patients, no collapse was found. In all patients except 2, reliable assessment proved possible of the site(s) of obstruction. No side effects were reported.
Conclusion: Sleep endoscopy is feasible and safe in daily practice when sedation is performed by an anaesthesiologist and can be used to locate the site of collapse in the UA. Sleep endoscopy findings in our study sample, as well as in the literature, differ according to the content of the study sample and the method of sedation. Treatment advice may differ from sleep endoscopy findings since other factors such as age and patient preferences need to be considered.

Introduction

Obstructive sleep apnoea (OSA) is a sleep-related breathing disorder caused by repeated partial or complete collapse of the upper airway (UA). These moments of collapse, which are known as "respiratory events", cause apnoea, hypopnoea, increased upper airway resistance and oxygen desaturation, or a combination of these symptoms.

OSA affects up to 2% of middle-aged women and 4% of middle-aged men.[1]

According to the American Academy of Sleep Medicine, OSA is defined as 5 or more respiratory events per hour (apnoea-hypopnoea-index AHI) accompanied by daytime symptoms.[2] These events lead to recurrent arousals, which in turn result in daytime symptoms like hypersomnolence and fatigue, concentration impairment and an increased incidence of traffic and occupational accidents.[3]

Continuous positive airway pressure (CPAP) is the standard treatment for patients with moderate and severe OSAS (AHI > 15). However, UA surgery may be considered in subjects with mild disease (AHI < 15) and those who do not tolerate or do not comply with CPAP.[4] In Belgium, CPAP is reimbursed by the health insurance system for patients with an AHI ≥ 20 and an arousal index ≥ 30.

Uvulopalatopharyngoplasty (UPPP) is probably the most widely performed procedure for patients with snoring and OSA. In a meta-analysis by Sher *et al.*[5], the overall success rate for this procedure was 41%. This percentage was even lower in patients with multilevel collapse. It is likely that an important reason for this disappointing success rate is inadequate patient selection, and the low success rate suggests that good diagnostic topical work-up to localise the site(s) of obstruction is mandatory to improve treatment results.[6]

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Many OSA patients were found to have UA collapse at several sites and a combination of more or less distinct anatomical abnormalities referred to as "disproportionate anatomy". A thorough UA evaluation should therefore precede therapeutical decision-making for these patients, especially when UA surgery or treatment with a mandibular advancement device is being considered.[7]

Various methods are available to determine the site of upper airway obstruction during sleep in OSA patients. These methods include both static and dynamic methods when the patient is awake or asleep. They all have their specific limitations and advantages.[8]

A routine ENT examination of the patient, awake in a sitting position, including the Müller manoeuvre,[9] supplies limited information about upper airway behaviour during sleep. The dimensions of the upper airway change during sleep because of muscle relaxation.

The examination of the upper airway can therefore be complemented with a dynamic evaluation to determine the site and pattern of upper airway collapse.

Croft and Pringle[10] pioneered sleep endoscopy in the early nineties. They proposed a grading system for upper airway collapse in patients with OSA. This grading system defines the type and site of upper airway collapse in a specific patient, and may improve patient selection for site-specific treatments.[11]

Sleep endoscopy is currently an integrated part of upper airway evaluation in OSA patients who are being considered for surgery according to the guidelines of the Dutch Society of Pulmonology (www.cbo.nl)).

The attraction of sleep endoscopy is its potential to provide a dynamic visualisation of the anatomical areas responsible for the generation of noise (snoring) or obstruction under conditions that mimic natural sleep. It has been criticised for not being a true reflection of normal physiological sleep and, in some studies, even non-snoring patients started snoring during drug-induced sleep.[12] On the other hand, sleep endoscopy is a simple and non-invasive way of investigating the upper airway during sleep. Sleep endoscopy using target controlled infusion (TCI) of propofol has been validated and it distinguishes between symptomatic and non-symptomatic subjects.[13]

The primary purpose of this retrospective study is to evaluate the feasibility of drug-induced sleep endoscopy in patients with snoring and obstructive sleep apnoea.

The second purpose is to evaluate the outcome of sleep endoscopy and to compare the findings from our study sample with other samples from the literature.

The third purpose is to compare the sleep endoscopy findings and the therapeutic advice given to the patients.

Materials and methods

Between March 2005 and August 2006, 70 patients who were being considered for UA surgery underwent sleep endoscopy in order to determine the site(s) of collapse. All of these patients presented with a history of snoring and/or excessive daytime sleepiness.

The data of these patients were retrospectively analysed. Table 1 lists the patient characteristics, polysomnographic data and subjective scoring of snoring and daytime sleepiness.

At the first visit and after a routine ENT examination, all the patients with a history of snoring and/or excessive daytime sleepiness were scheduled for polysomnography. In all these patients, the severity of snoring was assessed with a 10-point, bed-partner-evaluated, visual analogue scale (VAS) ranging from 0 (no snoring) to 10 (extreme snoring where bed partner has to leave the room). Daytime sleepiness was assessed using the Epworth Sleepiness Score (ESS). It ranges from 0-24, and abnormal somnolence is considered to be represented by a value greater than 10.[14]

At the second visit, polysomnographic data and therapeutical

Table 1

Subject Demographics

	n	mean (standard deviation)	range
BMI (kg/m²)	70	26.4 (3.28)	19.7-33.6
AHI	70	18.5 (12.6)	0-73
AHI supine	47	40.5 (27.5)	1-98
AHI side	47	10.7 (12.3)	0-62
VAS snoring (/10)	64	7.5 (2.8)	0-10
ESS	68	9.9 (5.4)	1-21
Age (years)	70	49.3 (8.4)	26-78

Table 2

Inclusion and exclusion criteria for sleep endoscopy[16]

1. Inclusion criteria for sleep endoscopy:
   - AHI < 40 or AI < 30
   - ASA classification I and II
   - Invasive intervention or surgery is considered
   - CPAP intolerance
2. Absolute exclusion criteria for sleep endoscopy:
   - AHI > 40 or AI > 30
   - ASA classification > II
   - Conservative management is preferable (weight loss, positional therapy, NCPAP,...)
3. Relative exclusion criteria for sleep endoscopy:
   - Severe obesity
   - Alcohol abuse

options were discussed with the patient. Only those patients with mild OSA (5 < AHI < 20), who did not meet the criteria for CPAP reimbursement in Belgium, were scheduled for sleep endoscopy to locate the site(s) of obstruction. Patients with moderate to severe OSA (AHI > 20) were scheduled for CPAP titration. Patients with non-apnoeic snoring did not undergo sleep endoscopy since palatal flutter is the most likely cause in these patients.[15] Local palatal treatment was proposed as the first-step treatment. Patients with CPAP intolerance or poor compliance were also scheduled for sleep endoscopy, regardless of the AHI.

Patients scheduled for sleep endoscopy were evaluated for risk factors by the anaesthesiologist.[16] Table 2 shows the inclusion and exclusion criteria. These criteria apply to patients who undergo sleep endoscopy without the involvement of an anaesthesiologist. In those cases where sedation is performed by an anaesthesiologist, patients with AHI > 40 may be included.

The patients were hospitalised in the surgical day care centre. Sleep endoscopy was performed in a darkened operating room with the patients in a supine position. To mimic the sleep condition of the patient at home, the patient was positioned in a hospital bed instead of on the OR table. Continuous monitoring took place with electrocardioagram and oxygen saturation. An anaesthesiologist induced artificial sleep through the intravenous administration of midazolam and propofol. Midazolam was administered in a bolus injection (1.5 mg) and propofol was titrated by target controlled infusion (TCI). No local anaesthetic was used in the nose in order not to interfere with the effect of local reflexes on breathing.[17] Before the state of unconsciousness was achieved and the patients started snoring, a flexible videolaryngoscope was introduced through the nose to visualise the upper airway and to assess the location of upper airway narrowing or collapse.

After the introduction of the flexible endoscope, it took a few minutes to reach a stage of stable sleep where reliable assessment was possible of the pattern and site of obstruction. Once the endoscope was introduced, the manipulation of the endoscope was kept to a minimum in order not to wake up the patient since this could cause irritation in the nose or throat, resulting in bothersome sneezing.

After sleep endoscopy all patients were moved to an upright position and an oxygen mask was placed on the nose. The patients were monitored in the recovery room for 1 hour. Three hours after sleep endoscopy, patients were discharged. All of them were told to leave the hospital with a companion, and not to drive a car. Patients were given a new appointment to discuss the results of the sleep endoscopy. Discussion of the results directly after the procedure was not recommended because most patients experience retrograde amnesia due to the sedation.

Obstruction was specified as flutter or collapse. Collapse was specified as monolevel palatal collapse (type I), multilevel palatal and tongue-base (type II) or monolevel tongue-base collapse (type III) in accordance with Fujita.[18] The type of collapse was described as circular, postero-anterior or originating from the lateral wall. Flutter was described as present or absent, and the site was noted.

Data relating to the therapeutic advice given after sleep endoscopy were reviewed.

Results

Sleep endoscopy was easily performed in all patients. It took about 20 minutes per patient, including the induction of sedation and transport to the recovery room.

There was no severe O_2 desaturation during the procedure. When saturation dropped below 85%, a chin-lift manoeuvre was performed to open the upper airway.

Table 3

Type of collapse (N = 70)

No obstruction	Monolevel obstruction			Multilevel obstruction
	Palate Type I	Tongue/Hypopharynx Type III	Total	Type II
4 (5,7%)	23 (32.9%)	20 (28.5%)	43 (61.4%)	23 (32.9%)

Table 4

Pattern of collapse according to obstruction site

	Anteroposterior	Laterolateral	Circular
Palatal	9 (19,6%)	10 (21,7%)	27 (58,7%)
Tongue / Hypopharyngeal	14 (32,6%)	9 (20,9%)	20 (46,5%)

Table 5

Generation of noise: site of flutter (N = 70)

Flutter	Absent	Palatal	Tongue/Hypopharyngeal	Multilevel
Total	6	49	13	2

In all cases this manoeuvre resulted in an immediate improvement in oxygen saturation. No intubation (emergency or otherwise) was needed in this group of patients.

The reliability of sleep endoscopy was poor because of bothersome continuous sneezing in 2 cases (2.8%). Visibility was sometimes compromised by abundant saliva but this could be removed in all cases with a small suction probe through the nose, resulting in good visibility. No other side-effects were noted.

Tables 3, 4 and 5 sets out the endoscopic findings.

Table 3 shows the type of collapse. Monolevel obstruction (type I and type III) was observed in 61.4% of patients, while multilevel obstruction (type II) was found in 32.9%. No obstruction was observed in 5.7% of patients. Table 4 specifies the pattern of collapse. Palatal collapse was mainly described as a circular collapse (58.7%). Lateral wall or antero-posterior collapse was of minor importance at the palatal level. The same tendency was seen at the hypopharyngeal level: circular collapse was seen in 46.5%. The generation of a snoring sound (flutter) (Table 5) was mainly observed at the palatal level (69%). Flutter was observed at the hypopharyngeal level in only 20% of cases.

After sleep endoscopy, patients were given treatment advice (Table 6) based on the severity of OSAS, the results of the ENT examination, age, findings based on sleep endoscopy and patient preference. This table mentions two experimental surgical treatments: hyoid-expansion[19] and adjustable tongue advancement.[20] Both surgical procedures address the hypopharyngeal segment of the UA.

Discussion

In our experience, sleep endoscopy proved to be a convenient and safe way of assessing upper airway obstruction in OSA patients. The procedure was well tolerated by all patients. No adverse events were seen, and the level of obstruction and snoring could be located in all but two patients. In 2 cases (2.8%) the reliability of the procedure was poor.

Sleep endoscopy remains controversial. It has been demonstrated that snoring and sleep apnoea varies with sleep position and sleep stage[21]. Information about the site of the obstruction is particularly important when surgical therapy is being considered. Although X-ray cephalometry and computed tomography could be performed during sleep, these are static and non-physiological approaches to UA investigation, and are limited by the side-effects of irradiation.[22] Magnetic resonance imaging is a safe method and can be used to perform dynamic studies of the upper airway, but its potential use is limited by costs and availability.[23] The ideal method is the direct visualisation of the upper airway during natural sleep with a flexible endoscope via the nose.[24] This method depends on the sleep quality of the patient while an endoscope is present in the nose and while sleeping in an unnatural sleep laboratory environment. It also places a heavy burden on the sleep laboratory personnel and the physician performing the examination. The next best option appears to be drug-induced sleep endoscopy. However, it remains controversial for several reasons.

Drug-induced sleep endoscopy comes closer to the natural

Table 6
Endoscopic findings and therapeutic advice (n = 70)

Collapse	Absent	Palatal	Tongue/ Hypopharyngeal	Both sites
Therapeutic advice				
No therapy	0	2	0	0
MAD	1	0	8	9
UPPP	3	13	4	9
RF Palate	0	1	0	0
RF Tongue	0	0	1	1
RF palate and tongue	0	0	1	1
Hyoid expansion	0	6	2	1
Advance	0	1	4	1
CPAP	0	0	0	1
Total	4	23	20	23

physiological state of sleep than all the other methods currently available. However, the procedure suffers from limitations.

It is uncertain whether the short analysis time (15-20 minutes) is representative for all obstructive events during a full night of sleep. Since the procedure is performed only in the supine position, it is not possible to evaluate positional effects.

Midazolam may induce excessive muscle relaxation during sleep, yielding false-positive obstructive events. On the other hand, if this is the case, relaxation affects the entire upper airway and not a specific site. We do not therefore believe that there will be a major effect on the site of obstruction. The use of TCI with propofol, starting at a low target concentration which is slowly increased, provides an objective and reproducible state of sedation, reducing the likelihood of excessive muscle relaxation and consequent false-positive obstructive events, and therefore enhancing the validity of sleep endoscopy.[13]

Sleep endoscopy is a subjective assessment. There is probably inter-observer variability and also variation between anaesthesiologists' sedation methods, for which there are no standardised protocols. Sleep endoscopy should therefore be performed by an experienced physician using a strict protocol for both collecting and reporting the data as described above.

If the inclusion and exclusion criteria are respected, sleep endoscopy is a safe procedure. These inclusion criteria are extremely important in those cases where the sedation is performed by a non-anaesthesiologist.[16] Oxygen desaturation due to obstructive events during sedation might be a risk in patients with severe OSA. We perform sleep endoscopy together with an anaesthesiologist, and we are therefore able to perform the procedure in patients with severe OSA. In the case of severe oxygen desaturation, the anaesthesiologist is able to intervene immediately.

In our experience, sleep endoscopy was useful for counselling the patient about the nature of the disease and the need for further treatment (with possible associated morbidity).

Table 7 compares our sleep endoscopy findings with data in similar studies in the literature.[11,25,26]

In our study, monolevel obstruction was seen in 61.4% of our patients: palatal obstruction in 32.9% and tongue/hypopharyngeal obstruction in 28.5% of cases. Quinn *et al.*[25] and Pringle *et al.*[11] report comparable rates of monolevel collapse: 78% and 60% respectively. Hessel *et al.*[26] report 24.1% for monolevel collapse, which is substantially lower than the rate we found.

Hessel's study included patients with socially disturbing snoring without excessive daytime sleepiness, but with comparable BMI and age. It does not state the mean AHI for the included patients, but since the study adopted stringent inclusion criteria (Table 2), they probably included patients with milder disease. We included patients with a mean AHI of 18.5 (range 0-73) and a mean ESS of 9.9. Since the role of the hypopharynx increases with the severity of the disease, this could explain why we found more monolevel hypopharyngeal obstruction (28.5%) than in their sample (2.4%). The fact that they report a very high percentage of multilevel collapse in their sample (60%) is probably due to the fact that their assessment of upper airway narrowing was less stringent than ours. We would expect a relative low percentage of multilevel collapse in a group of patients with snoring only and without excessive daytime sleepiness. The fact that they use a higher dose of midazolam (7-12 mg) for sedation might explain a more collapsible airway in comparison with propofol. Quinn's study[25] included only non-apnoeic snoring patients, and did not state the cut-off AHI value. The mean age of their sample was comparable with ours, but they included no

Table 7
Comparison of results of our sample with literature

	No obstruction	Monolevel obstruction			Multilevel obstruction (both sites)
		Palate	Tongue/Hypopharynx	Total	
Present study (n = 70)	4 (5.5%)	23 (31.9%)	20 (27.8%)	43 (59.7%)	23 (31.9%)
Hessel study[26] (n = 340)		74 (21.7%)	8 (2.4%)	82 (24.1%)	205 (60.3%)
Quinn study[25] (n = 50)		35 (70%)	4 (8%)	39 (78%)	11 (22%)
Pringle study[11] (n = 70)		33 (47.1%)	9 (13%)	42 (60%)	28 (40%)

data about BMI. Since OSA was excluded from this group of patients, it is logical that they report a 70% rate of monolevel palatal obstruction. Their observation of 22% multilevel collapse seems high for a sample of non-apnoeic snorers. Their method of sedation (midazolam 12 mg IV and topical nasal anaesthesia) may well explain these findings. The endoscopic findings in Pringle's study[11] are closest to our findings, although that study did not include any demographic or polysomnographic data about their sample. They included both snoring and OSA patients without defining them. It is therefore impossible to comment about the comparability of the findings.

This study makes it clear that snoring noise is mostly generated by palatal flutter (68%). In 19% of cases it is generated by vibrations of the hypopharyngeal lateral wall or the epiglottis. This finding compares well with the fact that UPPP is an efficient treatment for snoring, although not always efficient for OSA.[5]

Treatment advice was given according to the patient complaints, severity of OSA, age and comorbidity, the characteristics of collapse observed during sleep endoscopy and patient preference. In some cases, therefore, advice about treatment could contradict the sleep endoscopy findings. The four patients with no collapse had an AHI in excess of 15, which warranted treatment. Both ENT examination and PSG results were taken into account when deciding to proceed with UPPP in 3 of these patients. The fourth patient opted for a mandibular advancement device himself. Of 23 patients with monolevel palatal collapse, 13 were advised to undergo UPPP. Six patients were advised to undergo hyoid expansion[19] because of the severity of OSA and because of the collapsibility of the hypopharynx seen during the Müller manoeuvre. In 2 cases it was agreed with the patient not to proceed with treatment because there was no medical reason to treat (despite their snoring) and because of the patients' reluctance to undergo surgery. The 20 patients with monolevel hypopharyngeal/tongue-base collapse were treated with a procedure addressing the hypopharynx in all but 4 patients. These 4 patients were advised to proceed with UPPP because their main complaint was snoring without daytime sleepiness. Eleven of the 23 patients with multilevel collapse were advised to undergo treatment addressing both the palatal and tongue-base levels. UPPP was advised in 9 patients because their major complaint was snoring without daytime sleepiness. Radiofrequency ablation of the palate and/or tongue base was performed at the request of the patients to prevent morbidity.

In 41 patients (58.6%), the treatment advice matched the sleep endoscopy findings.

Sleep endoscopy is not the "final diagnostic tool" upon which the treatment decision is based. It should be considered part of a comprehensive diagnostic work-up taking into account both patient characteristics and habits, UA findings, polysomnographic data and the personal experience of the surgeon.

Conclusion

Drug-induced sleep endoscopy is a fast and safe way of evaluating the site(s) of upper airway obstruction. Sleep endoscopy findings help to choose a targeted treatment. A standardised method for the procedure is essential to minimise inter-observer and inter-anaesthesiologist variability, yielding reproducible results. Treatment advice was given taking into account the sleep endoscopy findings in the majority of the patients but additional patient characteristics were considered to be more important in the final decision-making, and so the advice did not always match

the findings. Further analysis of outcome after treatment will assess the value of sleep endoscopy for the selection of surgical techniques and treatments for patients with OSA.

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