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Laryngeal and hypopharyngeal obstruction in sleep disordered breathing patients, evaluated by sleep endoscopy

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Abstract The objectives of our study were to demonstrate the patterns and sites of the upper airway (UA) collapse in obstructive sleep apnea/hypopnea syndrome (OSAHS) patients, utilizing the sleep endoscopy technique, and to describe the technique and summarize our experience in a large series of patients. UA findings during sleep endoscopy with midazolam were examined prospectively in 55 surgical candidates with OSAHS. The uvulopalantine was the most common site of obstruction (89%), followed by the tongue base, hypopharynx and larynx (33% each), and nose (21%); 72% of the patients had multiple obstructions. There was a significant correlation between the number of obstructions and the respiratory distress index (RDI). Laryngeal obstruction was typically supraglottic. Hypopharyngeal obstruction involved concentric UA narrowing. Our findings emphasize the considerable rate of laryngeal and hypopharyngeal obstructions in patients with OSAHS and suggest that their misdiagnosis may explain at least part of the high surgical failure rate of UPPP for OSAHS patients. The number of obstruction sites correlates with respiratory distress index. Sleep endoscopy is safe and simple to perform.

Keywords Sleep apnea · Snoring · Sleep disordered breathing · Sleep endoscopy · Surgery · Midazolam

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Introduction

Sleep disordered breathing (SDB) is a broad term that encompasses snoring, upper airway resistance (UAR) syndrome, obstructive sleep apnea (OSA), and obstructive sleep hypopnea.

Snoring affects about 20% of the adult population. The incidence rises to up to 50% in men aged over 60 years [1]. The underlying mechanism involves sleep-induced hypotonus that causes vibration of the soft tissues in the upper airway, a drop in intrapharyngeal pressure, and narrowing of the air column during inspiration [2]. The disturbance may originate at different anatomic levels, and it may be intermittent or continuous.

The exact incidence of UAR syndrome is unknown. The disorder is manifested by repetitive transient arousals, increased snoring just before arousals, and an increase in inspiratory time and decrease in expiratory time [3]. The repetitive arousals may lead to major sleep fragmentation and excessive daytime sleepiness. Hypopnea is defined as 30% or greater reduction in flow associated with a 4% drop in oxygen saturation. OSA is defined as a cessation of airflow for 10 s or more during sleep despite persistent ventilatory efforts, as demonstrated by contraction of the respiratory muscles. The reported incidence is approximately 4% in men and 2% in women [4].

SDB is associated with increased mortality due to cardiopulmonary and cerebrovascular complications [5].

Accordingly, snoring is now recognized to be related to hypertension, angina, myocardial infarction and cardiovascular changes during sleep [6]. Therefore, early diagnosis and treatment are important.

Polysomnography is the gold-standard diagnostic tool for SDB and is essential for the evaluation of the severity of OSAHS. However, it cannot localize the sites of the upper airway (UA) obstruction. Routine physical examination, Muller maneuver, and head cephalometry are limited, because they are performed in the awaken state when the increased muscle tone can confound the results [7]. Of the real-time imaging techniques, with or without anesthesia, that are currently available, both fluoroscopy and computed tomography require the use of radiation [8]. Magnetic resonance imaging, which provides excellent resolution of the intra- and extra-pharyngeal lumen, is often inaccessible and relatively expensive. Furthermore, imaging techniques can be performed either in the awaken state, when they hardly represent anatomic changes during sleep, or in sleeping patients, which requires a much more complicated setting.

Attempts to overcome these pitfalls with the use of a fiberoptic nasoscope in a sleep laboratory met with limited success because the technique was time-consuming and depended on the patient falling and remaining asleep [9]. In 1991, Croft and Pringle [10] introduced the technique of sleep endoscopy wherein the UA was examined during midazolam-induced sleep. Obstructive episodes could be visualized in real time, even during the day [11], and documented on video or captured on stills. However, although sleep endoscopy was validated in several studies [11–13] and was found to be simple, safe, and cost-effective, data on its potential advantages in terms of patient selection and detection of different levels of obstruction remain sparse.

The purpose of the present study was to investigate the use of sleep endoscopy for detecting the pattern and sites of UA collapse in a large series of OSAHS patients.

Materials and methods

The study group consisted of 55 patients referred for sleep endoscopy prior to surgical intervention. All had complaints of daytime somnolence and a diagnosis of OSAHS on overnight polysomnography (Median Respiratory distress index = 28, Average = 33, ranging from 5 to 72 event per hour).

It is our departmental policy to initially refer all patients with OSAHS for treatment with continuous positive airway pressure (CPAP); only those who fail are recommended for surgery. Consequently, all the patients in the study were CPAP failures. The reason was poor compliance in 50 patients (94%) and lack of effectiveness in 3 (6%).

Patients with a body mass index (BMI) over 40 were excluded.

All patients signed an informed consent form after receiving a detailed explanation of the purpose and nature of the procedure. Endoscopy was performed after a 6 h fast. The medical history was taken and a complete physical examination performed. Thereafter, patients were asked to lie supine in a dark, quiet room throughout the procedure. Heart rate, blood pressure, and pulse oximetry were monitored. The nasal cavity was lubricated with 5% Esracaine gel in 20% of our patients. Sleep was induced by increased doses of intravenous midazolam, titrating according to effect. The midazolam dose was administered in increments of 2 mg every 3–5 min; the total dosage ranged from 2 to 14 mg (average, 6 mg). The airway was visualized before onset of midazolam administration and again after every dose increment. A flexible nasopharyngoscope was gently introduced through the nasal cavity until it reached the larynx and was then moved carefully for better inspection. Complete obstruction was defined clinically as complete blockage of the airway passage for at least 10 s, and partial obstruction, as narrowing or intermittent collapse of the airway passage. Selected events were recorded on video and stills. Since the obstructive events during sleep apnea/hypopnea usually occur during inspirium, the images were generally taken during that phase. Monitoring was continued throughout the procedure and thereafter, until the patient was fully awake. The average examination time was 20 min with a range of 15–55 min, patients were prohibited from leaving the hospital without an escort and were advised to rest for the remainder of the day.

Results

Sleep endoscopy data were obtained in 53 of the 55 OSAHS patients previously diagnosed by polysomnography. In two patients, 14 mg midazolam were insufficient for sedation, either because of the patient's severe anxiety or intolerance of the endoscope insertion.

The final study group consisted of 43 men (81%) and ten women of mean age 47 ± 13.4 years. Mean weight was 82.8 kg (range 50–122 kg) and mean height, 172.9 cm (range 156–192 cm); mean BMI was 27.6 ± 4.

Complete or partial obstructions were detected on level of the uvulopalatal plane (including the tonsils) in 89% of patients, base of the tongue, larynx, and hypopharynx in about 33% of patients, and the nose in 21% (Fig. 1). Thirty-eight patients (72%) had multiple obstructions. There was a significant correlation between the number of levels of obstruction and the respiratory distress index (RDI) ($R = 0.44$, $P = 0.001$). However, some patients with mild OSAHS exhibited two or three levels of obstruction,

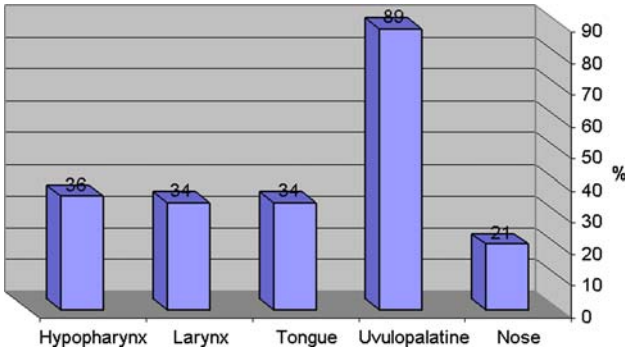

Fig. 1 Distribution of obstructions according to site

whereas others with severe OSAHS had an isolated obstruction. Airway obstruction was more dominant at the tongue base and hypopharynx than at the other sites. Neither age nor weight significantly correlated with RDI or number of levels of obstruction.

The nasal obstructions were due to nasal polyps or a severe deviated septum with compensatory hypertrophy of the contralateral inferior turbinate mucosa.A typical uvulopalatine obstruction is presented in Fig. 2.

The laryngeal obstructions were typically supraglottic. We identified two types. The first was characterized by concentric supraglottic soft tissue sucked into the glottic inlet (Fig. 3), and the second, by anterior to posterior collapse of the epiglottis at the glottic opening during inspirium. In some cases, the base of the tongue retracted backwards, worsening the epiglottic obstruction (Fig. 4). One patient, with right vocal cord palsy after hemithyroidectomy, showed right arytenoid collapse into the laryngeal inlet,

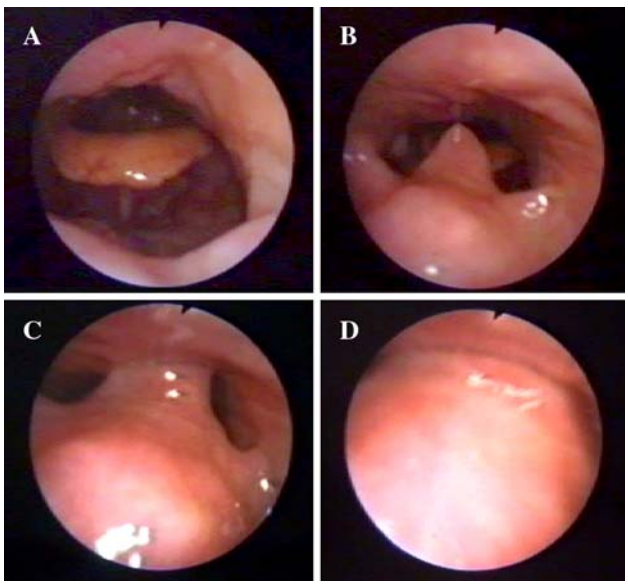

Fig. 2 Typical uvulopalatine obstruction demonstrated on sleep endoscopy (patient 24)

which periodically caused complete obstruction during inspirium.

The hypopharyngeal obstructions were caused by concentric narrowing of the airway due to posterior and lateral hypopharyngeal wall collapse. This pattern was usually documented towards the conclusion of the examination, at the end of an episode of apnea when the breathing effort was maximal, causing substantial subatmospheric pressure in the hypopharynx (Fig. 5). Hypopharyngeal obstruction tended to occur mainly in obese patients (BMI > 30), and it was always concomitant with obstruction of additional UA sites.

Discussion

The main finding of the present descriptive study was the pronounced obstruction at the supraglottis and hypopharyngeal levels. The majority of patients (72%) had multiple obstructions, and there was a significant correlation between the RDI and the number of UA obstructions. The procedure was successfully completed in most of the patients (96%), with no complications. The aim of this study was not to evaluate the surgical outcomes of OSAHS patients, but to describe our results and suggest that sleep endoscopy might be of clinical value in this setting.

The first step in the evaluation of patients with OSAHS is to identify the site or sites of obstruction. Unlike the oral cavity and oropharynx, which can be examined directly, obstructions in the laryngeal and hypopharyngeal areas may be overlooked in the wake patient. Indeed, the low success rate of uvulopalatopharyngoplasty (UPPP) for SDB (50–70%) [14], which is essentially a single-level solution, may be attributable at least in part to the lack of accurate preoperative detection of airway collapse. Although previous reports have found the uvupalatal complex to be frequently affected in patients with SDB (50–85%) [14], in agreement with the present study, most patients have obstructions at other sites as well, all of which need to be treated to achieve surgical success. We suggest that sleep endoscopy may be advantageous in these patients in detecting the levels of obstruction. By accurately detecting site of obstruction, the method enables to choose between the palate and the tongue as the initial target of treatment. The procedure may help by suggesting that more than one location should be addressed at the initial treatment. In some cases, we have shown that sleep endoscopy revealed the obstruction to be at a level that would not have been detected otherwise. Our surgical experience is not presented in the current study, due to the short postoperative follow-up period. However, preliminary results are encouraging. Camilleri et al. [12] demonstrated the predictive power of preoperative sleep endoscopy for the success of UPPP, and

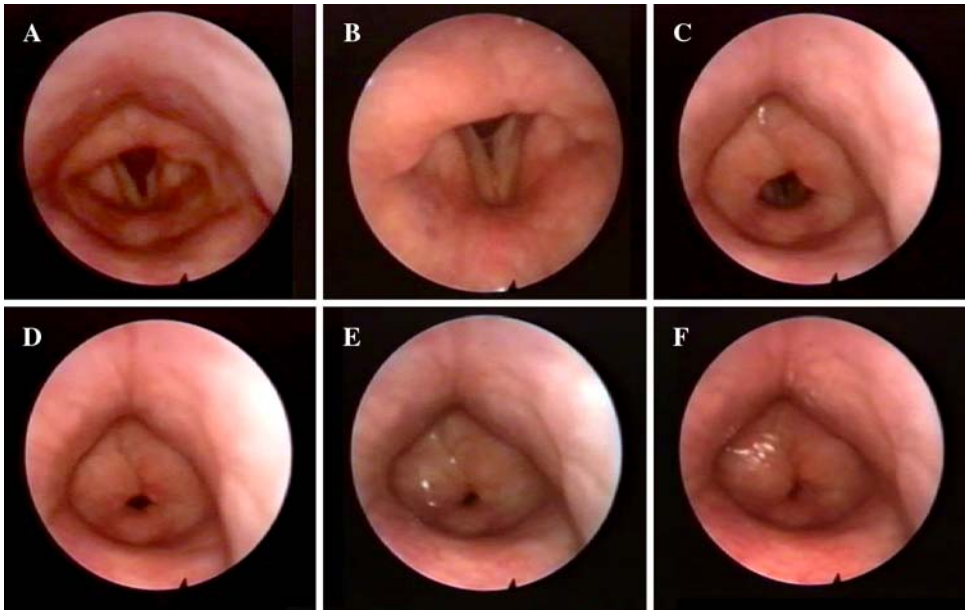

Fig. 3 Endoscopic view of a gradual supraglottic obstruction during midazolam-induced sleep endoscopy (patient 45)

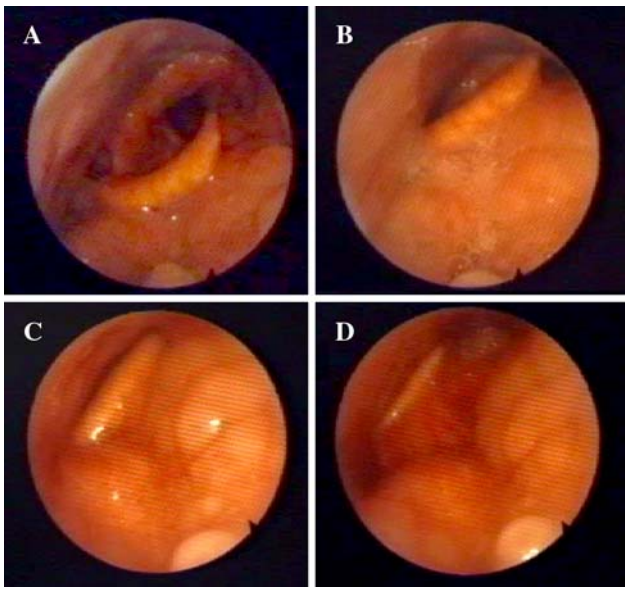

Fig. 4 Endoscopic view of a base of tongue retraction worsening the epiglottic obstruction (patient 5)

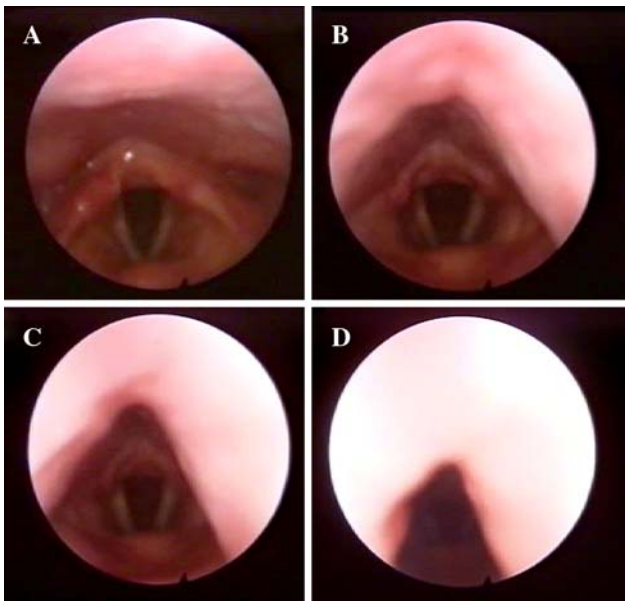

Fig. 5 Hypopharyngeal obstruction during sleep endoscopy. Note the concentric narrowing of the airway due to posterior and lateral hypopharyngeal walls collapse (patient 13)

Lin et al. [15] reported that endoscopic pharyngeal assessment in patients with OSA had clinical value for improving the UPPP outcome.

Laryngeal and hypopharyngeal obstructions are not uncommon; in the present series, they were detected in about one-third of subjects with OSAHS. Thus, since sleep endoscopy can demonstrate the entire airway under slight sedation, it is probably an effective tool to investigate changes at any anatomical level. The literature on UA obstruction usually refers to the uvulopalatine or base of the tongue levels; some authors considered the hypopharynx and base of the tongue as a single unit [14]. It is important to differentiate base of the tongue obstruction from laryngeal obstruction (supraglottic or glottic) or lateral hypopharyngeal wall collapse, because hypopharyngeal obstruction is not amenable to surgery. Data on the rate of obstruction at each anatomical level in OSAHS are inconsistent. We could demonstrate a significant correlation between the number of levels of obstruction of our OSAHS patients and their RDI. Others found that the base of the tongue obstruction is the main factor that correlated with higher values of the RDI [16].

As expected, most of our patients also snored prior (at lower midazolam doses) and in between apnea/hypopnea

events. However, the obstructive episodes and the snoring did not necessarily express pathology at the same level of the upper airway. Other studies support this observation [11]. Croft and Pringle [10], using their grading system for OSAHS syndrome, found an almost equal distribution of single- and multi-level obstruction, whereas more recently, Abdullah et al. [17] reported that 87% of 93 patients with OSA had multilevel obstructions, a rate compatible with the 72% noted here. Unlike our findings, however, the latter authors noted no correlation between the number of levels of obstruction and the severity of the OSAHS. Their most common sites of obstruction, with equal frequency, were the soft palate, the lateral pharyngeal wall, and the base of the tongue. These findings were confirmed in our series, where multiple obstructions were noted in 38 patients (72%) and the uvulopalatal complex was the most common site of obstruction (89% of patients).

Previous publications have reported that nasal obstruction is a major mechanism of snoring in OSA patients. In a study of approximately 5,000 patients, Young et al. [18] found that subjects with nasal obstruction were at higher risk for SDB. In our series, the nose and nasopharynx appeared to play a minor role, with only 11 of the 53 (21%) affected patients showing nasal obstruction (severe deviated septum, inferior concha, hypertrophy or hypertrophic adenoids). We speculate that nasal pathology probably increases the subatmospheric pressure in the UA during inspirium, consequently worsening the obstruction. Twenty percent of our patients were administered local anesthesia to their nasal cavity. However, the impact on UA reflex mechanisms, changing the maintenance of UA patency, is difficult to evaluate and beyond the scope of this study. We concluded that the presence of nasal obstruction most likely has an impact on the severity of OSAHS. Identification of nasal obstruction is important in the diagnostic work-up of OSAHS patients.

Sleep endoscopy with midazolam provides additional information to the physical examination and the Muller technique [7] performed in the awake state. However, whether midazolam anesthesia reliably simulates sleep remains controversial. In a unique comparison study [19] of nocturnal polysomnography and diurnal diazepam-induced sleep monitored by polysomnography and nasendoscopy, the author found no significant difference in type of apnea identified by the two modes, in terms of apnea index, desaturation index or mean maximal esophageal pressure. By contrast, others criticized the value of sleep endoscopy on the basis of findings that 45% of normal subjects snored under sedation and that 10% of subjects who snored under normal conditions did not do so under sedation. Sadaoka et al. [19] noted a significantly shorter duration of rapid eye movement (REM) sleep during diurnal drug-induced polysomnography as compared to nocturnal sleep. Moreover, the examination time is short and does to represent all of the changes in sleep stages or the dynamic pattern that occur during sleep.

Additional potential disadvantages of sleep endoscopy include possible anesthesia-induced alterations of the airway, failure of the technique to visualize events outside the pharyngeal airway, and variable patient tolerance to endoscope insertion [17]. Unlike normal sleep, which is characterized by different body positions, sleep endoscopy is performed in the supine only.

In summary, in the present study of 53 patients referred for sleep endoscopy, we found the technique to be safe, reliable and easy to perform. We were able to document obstructive events in real time and to effectively localize the level of airway obstruction. Our findings emphasize the considerable rate of laryngeal and hypopharyngeal obstructions in patients with OSAHS and suggest that their misdiagnosis may explain at least part of the high surgical failure rate of uppp for OSAHS patients. Further investigations are needed in order to evaluate the clinical importance of these findings.

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