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*EDITORIAL*

# Is the role of UPPP in nonapnoeic snorers underestimated?

W. De Backer*, P. Van de Heyning**

As recently reported, the overall estimated prevalence of sleep related breathing disorders defined by an apnoea-hypopnea score of 5 or higher, between the age of 30 to 60 yrs is 9% for women and 24% for men [1]. These frequencies are higher than generally estimated previously. We also know, from the study of He *et al*. [2], that patients with the most severe forms of the disease (apnoea index >20) have increased mortality rates. The same authors also showed that treatment with nasal continuous positive airway pressure (CPAP) significantly reduces this mortality whereas other treatment modalities such as uvulopalatopharyngoplasty (UPPP) do not influence survival [2]. Despite extensive evaluation of UPPP during the last 10 years, data remain conflicting but more recent studies invite us to re-evaluate its role in the treatment of sleep related breathing disorders (SRBD).

There is almost general agreement that, in the majority of patients, UPPP reduces the intensity of snoring [3]. Beside the (subjective) improvement in snoring, the outcome of UPPP has traditionally also been expressed by the resulting reduction in apnoea index (AI), patients with a 50% reduction in AI 6 weeks after surgery being considered as responders. In most studies about 50% of the patients can be classified as responders [4–7]. In severe sleep apnoea, it was, however, also demonstrated that UPPP has virtually no place. In 11 patients with severe sleep apnoea syndrome (apnoea index >35 or minimum oxygen saturation <75%) only 1 patient improved after UPPP whereas the AI in the others was unchanged and 4 of the latter developed cardiac failure [8].

Many attempts have been made to predict the outcome of the surgical procedure. Some techniques explore the upper airway in the awake patient. One of these is the Müller manoeuvre, where the patient is asked to make an inspiratory effort against a closed mouth and nose while the pharynx is being visualised through a fiber-optic endoscope. The interpretation of the results is difficult, because the degree of inspiratory effort is unknown and the contribution of pharyngeal muscle activation cannot be evaluated. Therefore direct techniques for assessing upper airway collapsibility during sleep were designed using direct monitoring of pressures in the posterior nasopharnyx, oropharynx, hypopharynx and oesophagus [9]. With these methods it became possible to determine critical collapsing pressures (Pcrit) and the site of the collapse. In almost half of the patients collapse was confined to the uvelopharyngeal or retropalatal segment [10]. However, response to UPPP is determined by the fall in Pcrit after surgery rather than by the initial pre-operative level of Pcrit [11]. By using a novel endoscopic method it was demonstrated that narrowing of the passive airway often occurs at several sites. Only patients with exclusively nasopharyngeal collapse improve after UPPP [12].

Measurements of the upper airway dimensions using computed tomography (CT) have also been used in order to predict outcome of UPPP. Patients with a minimal cross-sectional area of the upper airway, less than 1.0 $cm^2$ located 20 mm below the hard palate, were most likely to obtain a favourable effect [13].

The large scatter in the improvement in sleep disordered breathing after UPPP, the difficulties in predicting this result and the absence of any effect on survival have led to a general scepticism regarding the use of UPPP in obstructive sleep apnoea (OSA) patients [14]. Recently, however, some new data about survival after UPPP have been published. In patients treated either with CPAP or UPPP and followed for 6 years, there was no difference in the long-term survival between the two treatment groups [15]. As assessed by the polysomnographic data, the severity of the apnoea syndrome before treatment was even more pronounced in the patients treated with UPPP.

Another important point is the often mentioned dissociation between subjective and objective improvement. This was already recognised in the early studies [4], but has also been stressed by many others [16–18].

In this issue of the Journal, Janson *et al*. [19] demonstrate, in a large series of nonapnoeic snorers, that treatment with UPPP significantly improves many symptoms, confirming previous data obtained in a much more limited number of patients [20]. Patients treated with UPPP, compared to those treated with conservative therapy only, had significantly less snoring, less awakenings because of trouble breathing, less morning headache and increased daytime alertness 3 or 12 months postoperatively. However, this study has some limitations: the diagnosis of nonapnoeic snoring was only based on oxygen desaturation at night; there were no objective measurements during follow-up; there could have been a bias in the selection of the patients for operation. Nevertheless, there was a convincing change in most subjective assessments using a validated multiple-choice questionnaire. In a sense their results confirm previous observations in OSA patients, since in both apnoeic and nonapnoeic snorers a substantial

*Department of Respiratory Medicine and **ENT, University of Antwerp (UIA), Belgium. Correspondence: W. De Backer, Department of Respiratory Medicine, University of Antwerp (UIA), Universiteitsplein 1, 2610 Wilrijk-Antwerpen. Belgium.

improvement in subjective well-being was obtained after UPPP without change in the apnoea indices.

How does one explain the discrepancy between objective and subjective results? One of the possibilities lies in the underestimation of the recently described increased upper airway syndrome during polysomnography. Patients studied were snoring for more than 10% of their total sleep time and presented with snoring leading to a peak end inspiratory pressure which was more negative than the mean±1 SD baseline, awake supine, oesophageal pressure. It was shown that their sleep was disrupted and fragmented. This fragmentation was mainly due to alpha electroencephalogram (EEG) arousals. The latter were defined as sudden changes in EEG frequency to alpha range, lasting between 3 and 14 s. Sometimes, but not always, these alpha EEG arousals were accompanied by increase in muscle tone, eye movements and heart rate [21]. Apparently this upper airway resistance syndrome (UARS) could not be recognised in the study by Janson *et al*, but was probably also overlooked in previous studies where only apnoeas were detected. Therefore, it seems likely that changes in non-recognised increases in upper airway resistance or (obstructive) hypopnoeas can at least partially account for the subjective improvement after UPPP in the absence of improvement in commonly used, so called objective parameters, the apnoea indices.

To detect UARS it is necessary to monitor oesophageal pressures, thus increasing the invasive character of the sleep studies. However, in order to make a good therapeutic decision this information may be needed. There is little doubt that nasal CPAP (nCPAP) will improve this syndrome [22]. The data on UPPP, such as those presented by Janson *et al*, encourage evaluation of the effect of UPPP in the presence of UARS. It seems likely that UPPP will reduce the increase in airway resistance in this syndrome and probably also the related alpha EEG arousals and, therefore, sleep fragmentation. Until we know the results of UPPP for this specific indication in a sufficiently large population, we have to remain open-minded towards its application in the wide (and expanding) range of sleep related breathing disorders.

## References


1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230–1235.
2. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea. *Chest* 1988; 94: 9–14.
3. Van de Heyning P, Claes J, Valcke H, De Roeck J, Koekelkoren E, Bru J. Results of uvulopalatopharyngoplasty: an evaluation emphasizing preoperative selection criteria and tailoring technique of the procedure. *In:* Choward CN, ed. Chronic Ronchopathy. London J Libbey, 1988; pp. 373–378.
4. Fujita S, Conway WA, Zorick FJ, Sicklesteel JM, Roehrs TH, Wittig RM, Roth T. Evaluation of the effectiveness of uvulopalatopharyngoplasty. *Laryngoscope* 1985; 95: 70–74.
5. DeBerry-Borowiecki B, Kukwa AA, Blanks RH. Indications for Palatopharyngoplasty. *Acta Otolaryngol* 1985; 111: 659–663.
6. Wetmore SJ, Scrima L, Snyderman NL, Hiller FC. Postoperative evaluation of sleep apnea after uvulopalatopharyngoplasty. *Laryngoscope* 1986; 96: 738–741.
7. Dickson RI, Blokmanis A. Treatment of obstructive sleep apnea by uvulopalatopharyngoplasty. *Laryngoscope* 1987; 97: 1054–1059.
8. Walker EB, Frith RW, Harding DA, Cant BR. Uvulopalatopharyngoplasty in severe idiopathic obstructive sleep apnoea syndrome. *Thorax* 1989; 44: 205–208.
9. Chaban R, Cole P, Hoffstein V. Site of upper airway obstruction in patients with idiopathic obstructive sleep apnea. *Laryngoscope* 1988; 98: 641–647.
10. Shepard JW, Thawley SE. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1990; 141: 1350–1355.
11. Schwartz AR, Schubert N, Rothman W, *et al*. Effect of uvulopalatopharyngoplasty on upper airway collapsibility in obstructive sleep apnea. *Am Rev Respir Dis* 1992; 145: 527–532.
12. Launois SH, Feroah TR, Campbell WN, *et al*. Site of pharyngeal narrowing predicts outcome of surgery for obstructive sleep apnea. *Am Rev Respir Dis* 1993; 147: 182–189.
13. Shepard JW, Thawley SE. Evaluation of upper airway by computerized tomography in patients undergoing uvulopalatopharyngoplasty for obstructive sleep apnea. *Am Rev Respir Dis* 1989; 140: 711–716.
14. Rodenstein DO. Assessment of uvulopalatopharyngoplasty for the treatment of sleep apnea syndrome. *Sleep* 1992; 15: S56–S62.
15. Keenan SP, Burt H, Ryan CF, Fleetham JA. Long-term survival of patients with obstructive sleep apnea treated by uvulopalatopharyngoplasty or nasal CPAP. *Chest* 1994; 105: 155–159.
16. Guilleminault C, Hayes B, Smith L, Simmons FB. Palatopharyngoplasty and obstructive sleep apnea syndrome. *Bull Europ Physiopath Resp* 1983; 19: 595–599.
17. Blakley BW, Maisel RH, Mahowald M, Ettinger M. Sleep parameters after surgery for obstructive sleep apnea. *Otolaryngol Head Neck Surg* 1986; 95: 23–28.
18. Regestein QR, Ferber R, Johnson TS, Murawski BJ, Strome M. Relief of sleep apnea by revision of the adult upper airway. A review of clinical experience. *Arch Otolaryngol Head Neck Surg* 1988; 114: 1109–1113.
19. Janson C, Hillerdal G, Larsson L, Hultcrantz E, Lindholm C-E, Bengtsson H, Hetta J. Excessive daytime sleepiness and fatigue in nonapnoeic snorers: improvement after UPPP. *Eur Respir J* 1994; 7: 845–849.
20. Haraldsson P-O, Carenfelt C, Persson HE, Sachs C, Tornros J. Simulated long-term driving performance before and after uvulopalatopharyngoplasty. *ORL* 1991; 53: 106–110.
21. Guilleminault C, Stoohs R, Clerk A, Simmons J, Labanowski M. From obstructive sleep apnea syndrome to upper airway resistance syndrome: consistency of daytime sleepiness. *Sleep* 1992; 15: S13–S16.
22. Strollo PJ, Sanders MH. Significance and treatment of nonapnoeic snoring. *Sleep* 1993; 16(5): 403–408.