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Can obstructive sleep apnoea be a complication of uvulopalatopharyngoplasty?

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Abstract

Polysomnography is currently used for diagnosis, evaluation and selection of therapy in patients with obstructive sleep apnoea (OSA), but clinically successful uvulopalatopharyngoplasty (UPPP) is not necessarily reflected by post-operative improvement of polysomnographic recordings. Post-operative polysomnography may suggest deterioration of pre-existing OSA or, in snorers, *de-novo* precipitation of OSA. Thus, if polysomnography is a reliable indicator of OSA, then OSA may be a post-operative risk of UPPP. The aims of our study were: (i) to assess the possible deleterious effect of UPPP on sleep patterns; (ii) to further define the role of cardioisotope scanning in the evaluation of OSA; (iii) to assess the reliability of polysomnography given the clinical and cardioisotope scan findings. Symptoms, polysomnography and radionuclide ventriculography were prospectively compared pre- and post-operatively in 41 patients undergoing UPPP. In 12 patients (29 per cent), there were disparate results between pre- or post-operative polysomnography and the clinical and/or radionuclide ventriculography, as follows: In four of 16 patients with abnormal pre-operative ventricular performance, there was pre-operative symptomatology of severe OSA and a bedmate's reports of apnoeic episodes. This was in contrast to normal or near normal sleep apnoea recordings. In eight patients, post-operative improvement of symptoms was reported, despite deterioration of post-operative polysomnographic recordings. In these patients the post-operative improvement of symptoms was also reflected by improved ventricular performance. Worsening of ventricular performance was not demonstrated in any patient. In conclusion, UPPP does not induce OSA. Polysomnography may underestimate or even misdiagnose cases of OSA. The diagnostic importance of patient symptomatology should be stressed particularly in those patients with only mildly abnormal or even completely normal sleep studies. A combination of polysomnographic and cardiovascular evaluation in patients with symptomatology consistent with OSA is recommended.

Key words: Sleep apnoea syndromes; Snoring; Polysomnography; Radionuclide imaging; Uvula, surgery

Introduction

Although pretreatment polysomnography has been used to confirm a clinical suspicion of OSA and although post-operative polysomnography has also been recommended for evaluation of patient response (Aubert-Tulkins *et al.*, 1989), questions have been raised regarding this test's reliability. Reports of UPPP which did not result in an improvement of the Apnoea Index (AI), but which did result in a dramatic improvement of symptoms have been published (Zorick *et al.*, 1983; Simmons *et al.*, 1984; Rice, 1986; Dickson and Blokmanis, 1987; Rey *et al.*, 1989). Furthermore, the cost effectiveness of sleep studies in the evaluation of OSA has been questioned as well (Rice and Persky, 1986; Pelausa and Tarshis, 1989).

While snorers without OSA symptoms may be managed medically (Pelausa and Tarshis, 1989), other reports suggest that uvulopalatopharyngoplasty (UPPP) is the treatment of choice for heavy snorers (Fairbanks, 1984; Rice, 1986). Our preliminary observations revealed occasional post-operative deterioration of AI and oxygen saturation levels in patients who were diagnosed pre-operatively as simple snorers (i.e. without OSA). This indicates deterioration of pre-existing, undiagnosed, mild OSA or even *de-novo* appearance of OSA which suggests that UPPP may have a paradoxical effect on respiratory dynamics during sleep.

Can OSA be a complication of UPPP? The apparent failure of UPPP in these cases suggests that the airway obstruction is in a different anatomical plane than the resected velum. In the event of two localized constrictions, one at the palatal plane and the other at the plane of the base of the tongue (Pedro-Botet and Montaneri, 1989), pressure gradients probably decrease gradually at both levels. Following UPPP in these cases, a single constrict-

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TABLE I
GRADING OF PATIENTS' SYMPTOMS

Grade	Description of sleep disturbances
0	No snoring
1	Occasional mild snoring or snoring on back only
2	Habitual snoring in all positions
3	Habitual snoring associated with some episodes of apnoea and disturbed sleep pattern
4	Habitual snoring associated with episodes of apnoea and some morning and daytime sleepiness
5	Habitual snoring associated with episodes of apnoea and severe daytime sleepiness

ion at the base of the tongue would persist. Thus in this unique constricted segment, the base of the tongue might be exposed to a higher pressure gradient, leading, according to Bernoully's law to increased collapse and subsequent aggravation of OSA. This could explain how UPPP might aggravate or even cause OSA. Thus this 'procedure of choice' for snoring could transform simple snoring, a social disturbance, to OSA, a pathologic condition.

On the other hand, post-operative aggravation of polysomnographic data could be explained by failure of pre-operative polysomnography to reliably reflect the patient's true condition. The reliability of polysomnography may best be evaluated by comparison with another objective method. Previous studies have shown (Tal *et al.*, 1988; Zohar *et al.*, 1992) that radionuclide right ventriculography is a useful adjunct for the evaluation of OSA. In the present study, pre- and post-operative clinical, polysomnographic and resting cardioisotope scanning studies in patients undergoing UPPP were investigated. The aims of our study were: (i) to assess the possible deleterious effect of UPPP on sleep patterns; (ii) to further define the role of cardioisotope scanning in the evaluation of OSA; (iii) to assess the reliability of polysomnography given the clinical and cardioisotope scan findings.

Patients and methods

During a three-year period, 41 surgical candidates for UPPP consented to be included in the study. The study group consisted of 37 men and four women, ranging in age from 26 to 65 with a mean of 49 years. None had evidence of coronary artery disease, valvular heart disease or primary myocardial disease. Well-controlled hypertension was present in six patients. All the patients were evaluated as follows: (a) severity of symptoms was evaluated by a questionnaire. Both patients and bedmates responded to questions regarding day time somnolence, chronic fatigue, headaches and heavy snoring. Bedmate assessment of apnoeic episodes was recorded as well. The severity of symptoms was rated from 0–5 (Table I); (b) severity of OSA was defined by AI i.e. the average number of apnoeas per hour of sleep. Mild OSA was defined as an AI between 11 and 20; moderate, 21–40, and severe, with an AI greater than 40 (Table II). Oxygen saturation under 80 per cent was regarded as abnormal. Maximal snoring intensity was also measured; (c) resting cardioisotope scanning was performed with 16 mCi technetium-99 m. Analysis of the data was done independently by two cardiologists, both blinded to the pre-operative results. Ventricular performance was regarded as normal if the right ventricular ejection fraction (RVEF) was greater than 40 per cent and left ventricular ejection fraction (LVEF) was greater than 50 per cent in the presence of normal ventricular wall size and motion.

UPPP was performed in all patients using a similar surgical technique. The soft palate was resected at the pre-operatively marked palatal dimple (Lusk, 1986). All patients underwent a complete re-evaluation four to 26 (mean = 8.9, median = 7) months post-operatively. All polysomnographic examinations were performed in the same sleep laboratory. The pre-operative and post-operative data regarding patients' symptomatology, bedmate reports, polysomnography, and cardiovascular evaluation were compared.

Post-operative symptomatic improvement of one grade was regarded as mild while improvement of two or more grades was regarded as marked. Polysomnographic improvement was defined as a reduction of at least 50 per cent in AI, or of 30 per cent to 49 per cent in AI in those patients who demonstrated abnormal pre-operative oxygen desaturation which normalized post-operatively. A reduction of less than 50 per cent in AI with persistent desaturation was regarded as no change. An absolute change in left and right ventricular ejection fraction of at least five per cent was regarded as significant (Berger *et al.*, 1979a; Berger *et al.*, 1979b; Mathur,1981).

Statistical analysis

Statistical analysis was performed using the Pearson Correlation, One Way Analysis of Variance non-parametric tests and with the parametric *t*-test and paired *t*-test. A probability of less than 0.05 was considered significant.

Results

The duration of pre-operative snoring ranged from one

TABLE II
PRE-OPERATIVE PATIENTS' CLASSIFICATION ACCORDING TO AI CORRELATED WITH THEIR SYMPTOMS

Pre-operative polysomnography	No. of patients	Pre-operative AI range/mean	Pre-operative symptomatology					
			0	1	2	3	4	5
Normal	2	0					1	1
Snoring	7	4–10/6			1	1	2	3
Mild OSA	11	11–20/17			1	1	5	4
Moderate OSA	10	21–40/29				2	3	5
Severe OSA	11	41–71/60				1	1	9
Total	41	0–71/29	0	0	2	5	12	22
					5%	12%	29%	54%
						Of the patients		

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TABLE III
CORRELATION BETWEEN PRE-OPERATIVE POLYSOMNOGRAPHIC OSA SEVERITY AND ABNORMALITY IN RADIONUCLIDE VENTRICULOGRAPHY

Pre-operative polysomnography	Pre-operative, right, abnormal ventricular performance: RVEF ≤40 (%)	RVEF ≤40 (%) & RVHK	RVHK	No. of patients	Pre-operative Abnormal LVEF
Normal	–	33,33	–	2/2 (100%)	41
Snoring	35	39	–	2/7 (28.6%)	
Mild OSA	–	40,40	–	2/11 (36.4%)	
Moderate OSA	40	40,40	–	3/10 (20%)	
Severe OSA	35	38,40	45, 50, 50, 58	7/11 (63.6%)	45,48,48
Mean	36.7	38.1	50.7		45.5
No. of patients	3	9	4	16/41	4/41
	7.3%	22%	9.7%	34.1%	9.7%

RVEF = right ventricular ejection fraction; RVHK = right ventricular hypokinesia.

to 33 years with a mean of 15 years. The severity of patient symptoms ranged from grades two to five with a mean of 4.3 (Table II). The patients' body mass index ranged from 20.5 and 36.2 with mean of 26.3. AI ranged between 0 and 71 with a mean of 29. Patient classification according to severity of pre-operative sleep apnoea recordings is presented in Table II. Pre-operative snoring intensity was 55.4 ± 14 dB. By using the Pearson correlation test, a statistically significant correlation was found between AI and oxygen desaturation ($R = 0.65$, $p<0.005$) and borderline significance between the AI and the BMI ($R = 0.41$, $p = 0.07$). No statistically significant correlation could be demonstrated between AI and age, symptoms, and duration or intensity of snoring.

Pre-operative right ventricular performance was abnormal in 16 patients (Table III). Abnormal RVEF was the only finding in four patients. Abnormal RVEF with localized or diffuse right ventricular hypokinesia (RVHK) was found in nine patients and hypokinesia only was found in four patients. LVEF was also abnormal in four of the patients with abnormal right ventricular performance (Table III).

No statistically significant correlation was found between the snoring period and the duration of symptoms or between AI and abnormal radionuclide ventriculography (Table III).

Of nine patients whose bedmates reported occurrences of apnoeic episodes and who had symptomatology of severe OSA (grade 4 or 5), the polysomnographic study was completely normal in two. In the other seven patients snoring was accompanied by only a few apnoeic episodes (Table II). The two patients with normal polysomnography and two snorers with a few apnoeas had abnormal ventricular performance (Table III).

Post-operative subjective improvement was reported by 35 patients (85.4 per cent) as shown in Table IV. Eight of these patients (19.5 per cent) reported mild, and 27 (65.9 per cent) marked improvement. No change was reported by six patients (14.6 per cent). There was no post-operative worsening of symptoms.

Post-operative polysomnography demonstrated improvement in 24 patients (58.5 per cent), no change in nine (22 per cent) and deterioration in eight patients (19.5 per cent) as presented in Table V. Post-operative evaluation demonstrated OSA in the two patients whose pre-operative polysomnographic study was normal and in three patients whose pre-operative polysomnography showed only snoring.

Using One-Way Analysis of Variance, a statistically significant difference was found in pre-operative AI severity between the group of patients showing improvement in AI and those who did not ($p = 0.0005$). The patients whose polysomnography showed improvement had a mean pre-operative AI of 38.9 ± 22.2. While those with no change in their sleep apnoea recordings had a mean pre-operative AI of 17.4 ± 8.1. The patients who's polysomnography showed deterioration had a mean pre-operative AI of 10.5 ± 10.6. No statistically significant correlation was found between improvement of patient symptoms and improvement as recorded by polysomnography.

For the entire patient group, RVEF increased from a mean of 47.3 ± 8.2 per cent to 51.8 ± 8.5 per cent. Post-operative radionuclide ventriculography showed improvement in 14 (87.5 per cent) of the 16 patients with pre-operative abnormal ventricular performance (Table VI). In two patients, one of whom had focal RVHK, there was no change. In addition to the significant increase in

TABLE IV
CORRELATION BETWEEN PRE-OPERATIVE POLYSOMNOGRAPHIC SEVERITY OF OSA AND POST-OPERATIVE IMPROVEMENT OF SYMPTOMS

Pre-operative Polysomnography	No. of patients	Post-operative symptomatology: No change	Improvement	Deterioration
Normal	2	–	2	–
Snoring	7	2	3+2*	–
Mild OSA	11	1	8+2*	–
Moderate OSA	10	2	6+2*	–
Severe OSA	11	1	8+2*	–
Total	41	6	27+8*	0
		14.6%	65.9%+19.5%	

*Mild improvement.

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TABLE V
CORRELATION BETWEEN SUBGROUP PRE-OPERATIVE AND POST-OPERATIVE POLYSOMNOGRAPHIC SEVERITY OF OSA IMPROVEMENT

Polysomnography	No. of patients	Post-operative polysomnography		
		No change	Improvement	Deterioration
Normal	2	–	–	2
Snoring	7	1	3	3
Mild OSA	11	5	5	1
Moderate OSA	10	3	5	2
Severe OSA	11	1	10	–
Total	41	10 24.4%	23 56%	8 19.6%

RVEF demonstrated in 14 of the patients with abnormal pre-operative ventricular performance (Table VI) there was also an increase in five patients with normal pre-operative RVEF. A significant increase in LVEF (mean difference = 14.15, mean = 56.15) was found in three of the four patients whose pre-operative RVEF and LVEF were both abnormal. Snoring did not correlate in any way with pre- and post-operative indices.

In four patients, improvement in symptoms was associated with post-operative deterioration of polysomnographic recordings or even appearance of OSA. The abnormal pre-operative ventricular performance had normalized in these patients (Table VI).

Discussion

Our data revealed a marked discrepancy between patients' symptoms and polysomnographic analysis. If success of UPPP is defined by symptomatology alone, the procedure was successful in 85.4 per cent of the cases and unsuccessful in 14.6 per cent. On the other hand, if success is measured as a function of post-operative sleep apnoea recordings, UPPP was successful in only 58.5 per cent of the cases and unsuccessful in 22 per cent of cases. Furthermore, by this criteria, the condition of 19.5 per cent of all patients actually deteriorated or even developed OSA *de-novo* as a result of the procedure. It could be concluded that OSA might even paradoxically be a complication of UPPP. It is well known that while post-operative subjective improvement has been found in 64 per cent (Simmons *et al.*, 1984) and up to 100 per cent of patients (Zorick *et al.*, 1983), objective improvements of apnoea is seen only in about 50 per cent of cases (Zorick *et al.*, 1983; Simmons *et al.*, 1984). In our patients, the discrepancy between polysomnography and symptomatology was even greater. Our use of resting radionuclide ventriculography was two-fold. First, to assess the reliability of patient symptoms and bedmate reports *versus* the reliability of polysomnography, and second, to define the potential risk of OSA induced by UPPP.

OSA has been associated with reduced ventricular ejection fractions (Fletcher *et al.*, 1987; Tal *et al*, 1988; Zohar *et al.*, 1992), left and right ventricular hypokinesia, septal hypokinesia and right ventricular hypertrophy (Berman *et al.*, 1991). Factors contributing to the haemodynamic abnormalities in OSA patients include severe oxyhaemoglobin desaturation and respiratory acidosis. Respiratory acidosis in turn has a vasoconstrictive effect on the pulmonary circulation which results in pulmonary hypertension, increased right ventricular afterload and subsequent cor pulmonale. Reversal of pre-operative right-sided haemodynamic abnormalities in our patients suggests that aetiologies other than OSA may be excluded as a cause of the impaired ventricular performance. This emphasizes the effectiveness and importance of UPPP treatment for OSA.

Patient symptoms indicative of severe OSA supported by bedmate reports of apnoeic episodes were confirmed by radionuclide ventriculography in spite of normal or near normal sleep apnoea recordings. Furthermore, post-operative deterioration of sleep apnoea or even precipitation of OSA documented by polysomnography after UPPP (but denied by patients and their bedmates) was excluded by radionuclide ventriculography. In no patient was the deterioration in polysomnographic studies confirmed by a parallel change in ventriculography. Since post-operative deterioration of polysomnographic recordings could not be confirmed by ventriculography and since sleep apnoea recordings can hardly be false–positive it must be concluded that the pre-operative polysomnographic evaluation does not reflect in some instances the patient's true condition. One-night sleep apnoea recordings may underestimate or misdiagnose OSA (Meyer *et al.*, 1993). This may explain why many patients

TABLE VI
CORRELATION BETWEEN PRE-OPERATIVE POLYSOMNOGRAPHIC SEVERITY OF OSA AND POST-OPERATIVE VENTRICULAR PERFORMANCE

Pre-operative polysomnography	Post-operative right ventricular performance			Post-operative LVEF	
	No change	Improvement	Deterioration	Improvement	No change
Normal	–	2	–	1	–
Snoring	4	3	–	–	–
Mild OSA	3	7	1	–	–
Moderate OSA	6	4	–	–	–
Severe OSA	4	7	–	2	1
Total	17 41.5%	23 56.4%	1 2.5%	3 7.5%	1 2.5%

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report symptoms consistent with severe OSA while they are diagnosed as being 'only' heavy snorers (Hillerdal *et al.*, 1991; Meyer *et al.*, 1993).

But why should the post-operative sleep study be more reliable than the pre-operative one? Sleep study results are dependent upon the quality of sleep. The 'first night effect' has been used to describe a patient's sleep pattern in a new and different environment – such as the sleep laboratory. Patients undergoing their first polysomnography are undoubtedly stressed, having become recently aware of their condition, and of the possibility of impending surgery. In addition, they must sleep while connected to electrodes in new and relatively depressing surroundings. In contrast, in the post-operative examination, the patient returns to a now familiar environment and without the specter of surgery looming ahead. Pedro-Botet and Montaneri (1989) suggested that polysomnography of at least two consecutive nights is indispensable for a correct diagnosis. On the other hand, patients with OSA are often hypersomnolent, suffer from excessive daytime sleepiness and may fall asleep even in unusual settings. Night-to-night variability of breathing and oxygenation in elderly patients without OSA has been studied during two (Bliwise *et al.*, 1983; Aber *et al.*, 1989), three (Mosko *et al.*, 1988), or even more (Lord *et al.*, 1991) consecutive nights. Although in these patients, sleep patterns were consistent from night to night, OSA subjects with an AI greater than 12 are more likely to exhibit a consistent number of apnoeas during two nights than those patients with a lower AI (Witting *et al.*, 1984) and a significant number of patients with OSA can be missed with one polysomnography alone (Meyer *et al.*, 1993). Thus, consideration must also be given to other parameters such as: oesophageal pressure monitoring (Stoohs and Guilleminault, 1991; Guilleminault *et al.*, 1992), mean sleep latency time, duration of respiratory disturbances, oxygen desaturation index (Dickson and Blokmanis, 1987; Rey *et al.*, 1989), sleep position (Cartwrite, 1984; Cartwrite *et al.*, 1985) and nasal resistance (Metes *et al.*, 1991).

In our present study, OSA induction or deterioration caused by UPPP could not be substantiated. The data demonstrates that polysomnography may be inaccurate and may underestimate or even misdiagnose OSA. Cardiovascular evaluation in addition to the polysomnography is a valuable asset in the clinical investigation of patients suspected of having OSA, and should be included in the diagnostic approach especially if surgery is considered.

Conclusion

Uvulopalatopharyngoplasty does not induce OSA. Nocturnal sleep apnoea recordings may underestimate or even misdiagnose cases of OSA. Patient symptoms must be seriously considered even in the presence of relatively benign or even normal sleep studies. The importance of combined polysomnographic and cardiovascular evaluation in patients having symptoms consistent with OSA is emphasized.

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