Evaluation of Patients With Sleep Apnea After Tracheotomy

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Objective: To determine the effect of tracheotomy on polysomnographic and arterial blood gas data in patients with obstructive sleep apnea (OSA).

Design: A retrospective study of all patients who underwent tracheotomy and were studied polysomnographically at the Johns Hopkins Sleep Disorders Center, Baltimore, Md, since 1981.

Setting: A regional sleep disorders center.

Patients: Twenty-eight patients (8 women and 20 men), aged 22 through 77 years. Patients were categorized into 2 groups on the basis of whether they had already undergone tracheotomy before polysomnography. Group 1 patients all had a polysomnographic diagnosis of OSA before tracheotomy. They were further subdivided on the basis of whether cardiopulmonary decompensation had been absent (group 1a, n = 10) or present (group 1b, n = 13). Group 2 patients (n = 5) had undergone tracheotomy to treat upper airway obstruction that developed after non–apnea-related upper aerodigestive tract surgeries.

Intervention: Tracheotomy.

Main Outcome Measures: Nocturnal non–rapid eye movement, apnea-hypopnea index, percentage oxyhemoglobin saturation, and arterial blood gas data.

Results: Patients with OSA underwent tracheotomy as definitive treatment for the apnea (n = 15), to prevent postoperative upper airway compromise after uvulopalatopharyngoplasty (n = 7), and to treat upper airway compromise after non–apnea-related upper aerodigestive tract surgeries (n = 6). Tracheotomy alleviated apnea in all 10 patients with uncomplicated sleep apnea (group 1a). For patients with OSA complicated by cardiopulmonary decompensation (group 1b), tracheotomy improved but did not eliminate sleep apnea in 7 of the 13 patients, despite overall improvement in arterial blood gas values. For patients whose sleep apnea had not been diagnosed polysomnographically before tracheotomy (group 2), tracheotomy was still required to treat OSA that had previously not been recognized.

Conclusions: Tracheotomy effectively treated patients with uncomplicated OSA, but was much less effective in treating patients with OSA and cardiopulmonary decompensation. In patients who underwent tracheotomy in conjunction with other upper aerodigestive tract surgeries, concomitant obstructive sleep apnea often required continued use of a tracheotomy to maintain upper airway patency.

way obstruction. Since it was first used in 1969, tracheotomy has been shown in several large studies to alleviate OSA and the symptoms of this disorder. In recent years, the use of tracheotomy has declined as upper airway surgeries and less invasive methods with nasal continuous positive airway pressure (CPAP) became available to alleviate upper airway obstruction. Nevertheless, tracheotomy is still used in selected patients for whom other treatment approaches are either ineffective or unacceptable. Its present role in the treatment of OSA, however, is not clear. In this study, we sought to characterize the patients undergoing tracheotomy according to presence or absence of cardiopulmonary and upper aerodigestive dysfunction and the response of their sleep apnea to this procedure.

PATIENTS AND METHODS

PATIENT SELECTION

Records of patients who underwent tracheotomy and were studied at the Johns Hopkins Sleep Disorders Center, Baltimore, Md, were reviewed. All patients who had undergone tracheotomy and pretracheotomy or posttracheotomy sleep studies were included.

STUDY DESIGN

All patients who underwent tracheotomy and had nocturnal sleep studies at the Johns Hopkins Sleep Disorders Center were included. Each patient’s clinical chart was reviewed, and polysomnographic and arterial blood gas data were extracted. Patients were initially divided into 2 groups, based on whether they had already undergone tracheotomy before polysomnography. Patients who had not had tracheotomy before initial polysomnography were all found to have OSA as defined by a non–rapid eye movement (NREM) apnea-hypopnea index of greater than 5 episodes per hour. These group 1 patients were further subdivided into those with (group 1b) and without (group 1a) evidence of cardiopulmonary decompensation as defined by an initial PaCO\(_2\) greater than 45 mm Hg, PaO\(_2\) less than 60 mm Hg, or clinical evidence of heart failure. All group 1 patients with OSA had their OSA diagnosed before tracheotomy and had undergone tracheotomy after conventional therapy (weight loss, nasal CPAP) failed or before undergoing uvulopalatopharyngoplasty (UPPP) surgery to prevent upper airway compromise. In contrast, group 2 patients had OSA that had not been diagnosed polysomnographically before tracheotomy. Rather, these patients had tracheotomies placed to prevent airway compromise during upper aerodigestive tract surgery. They had undergone polysomnography only after concern about sleep-related upper airway obstruction had arisen as plans were made to close their tracheotomies.

RESULTS

The anthropometric data are summarized in Table 1 for patients with OSA whose apnea was either uncomplicated (group 1a) or complicated (group 1b) by cardiopulmonary decompensation, as well as for patients with sleep apnea that had not been diagnosed before their tracheotomies (group 2). In group 1a, tracheotomy was indicated for definitive treatment of sleep apnea (n = 5) after nasal CPAP failure or intolerance, or to prevent post-UPPP airway obstruction (n = 5). In group 1b, 3 patients underwent tracheotomy with UPPP or thyroidectomy, and in the remaining 10, other treatment failed. In group 2, 1 patient underwent tracheotomy after resection of a neck mass to prevent airway compromise from postoperative swelling. The other patients in group 2 (n = 4) underwent tracheotomy either with vocal cord surgery or secondary to laryngeal abnormality (eg, vocal cord polyps, paralysis). Patients in group 1b were generally heavier than those in groups 1a and 2.

CLINICAL TESTING

Sleep studies were performed in the sleep laboratory by means of standard techniques as previously described. In brief, airflow at the nose and mouth was measured with a thermodisc placed at the level of the upper lip in the midline position. Thoracoabdominal efforts were assessed with mercury strain gauges placed at the second intercostal space and at the level of the umbilicus. Surface electroencephalographic electrodes placed at C3-A2 and C3-O1, a submental electrode, and right and left oculoculograms were used to stage sleep. An ear oximeter recorded oxyhemoglobin saturation. A continuous electrocardiogram recorded heart rate and rhythm. A polygraph ran continuously at 10 mm/s to simultaneously record all physiological data throughout the night.

Polysomnograms were scored for disordered breathing episodes (either apneas or hypopneas) and for changes in oxyhemoglobin saturation, as previously described. In each patient, the apnea-hypopnea index (AHI) was calculated as the number of obstructive apneas or hypopneas per hour of NREM and rapid eye movement (REM) sleep. The mean ratio of NREM apnea time to disordered breathing time was defined by the duration of complete absence of airflow (apnea) during disordered breathing episodes. The baseline and nadir oxyhemoglobin saturation were measured for NREM and REM disordered breathing episodes to calculate the average baseline and average low oxyhemoglobin saturation.

In group 1b, arterial blood gases were obtained with the patients awake and resting comfortably. All blood gases were obtained with the patient breathing room air, with the exception of 1 patient who was breathing oxygen, 2 L/min via nasal cannula.

STATISTICAL ANALYSIS

Anthropometric, polysomnographic, and blood gas measures were compared between and within groups by means of 2-sample and paired t tests (Minitab Inc, State College, Pa), respectively. Statistical significance was inferred when P < .05.

PRETRACHEOTOMY POLYSOMNOGRAPHIC DATA (DIAGNOSED OSA, GROUPS 1A AND 1B)

The pretracheotomy polysomnographic data for patients in groups 1a and 1b are reported in Table 2. The mean NREM AHI for each group exceeded 60 episodes per hour, indicating that all patients had severe OSA. No significant difference in AHI between groups was ob-
served (P = .20). All of the initial polysomnographic studies were performed with the patient breathing room air with the exception of 2 patients, patients 19 and 20, who were breathing oxygen, 4 and 8 L/min, respectively, during their sleep studies.

POSTTRACHEOTOMY POLYSOMNOGRAPHIC DATA

In Table 3 and Figure 1, the posttracheotomy polysomnographic data are reported for all patient groups.

Polysomnograms
With Closed Tracheotomy

After tracheotomy placement, sleep studies were performed with the tracheotomy closed and confirmed that OSA persisted, with mean NREM AHI5 exceeding 20 episodes per hour in all patient groups. Three notable exceptions can be attributed to intervention with weight loss (group 1b patients 18 and 16, who lost 27 and 63 kg, respectively) or UPPP (group 1a patient 6). Therefore, resolution of OSA was observed only in selected patients after UPPP or weight loss, with the majority of patients still requiring tracheotomy for treatment of OSA.
tion through an open tracheotomy rather than upper airway obstruction.

In group 2, polysomnograms demonstrated marked OSA when the tracheotomy was closed that was eliminated when the tracheotomy was opened. This finding suggests that tracheotomy was still required to treat sleep apnea in previously undiagnosed patients who had undergone tracheotomy after upper aerodigestive tract surgery.

**ARTERIAL BLOOD GASES**

The pretracheotomy and posttracheotomy arterial blood gas results for patients in group 1b are reported in Figure 2. They show that tracheotomy was associated with an improvement in pH and decrease in PaCO2 levels in these patients with pickwickian syndrome, but no significant change in PaO2.

**COMMENT**

We found that our 28 patients who had undergone tracheotomy could be categorized into 2 distinct groups: those whose OSA had been diagnosed before tracheotomy and those in whom the suspicion of OSA arose after upper aerodigestive tract surgery. For patients whose OSA was uncomplicated by cardiopulmonary decompensation (group 1a), tracheotomy was effective in eliminating clinically significant levels of apnea (NREM AHI fell below 20 episodes per hour with
the tracheotomy open). Sleep apnea also improved significantly in patients whose sleep apnea was complicated by cardiopulmonary decompensation, although clinically significant levels of apnea persisted in more than half of these patients (7 of 13) on follow-up sleep studies. In patients who had undergone polysomnography only after concern about upper airway obstruction had arisen after upper aerodigestive tract surgery (group 2), sleep studies disclosed the presence of marked OSA when their tracheotomy had been capped. In fact, apnea remitted uniformly in these patients when the tracheotomy was left open, a finding that hindered decannulation. Overall, our findings suggest that tracheotomy plays an important role in the management of sleep apnea in each of the patient groups. In the following discussion, we consider how clinical and polysomnographic findings can be used to guide clinicians in evaluating possible deannulation in patients who have undergone tracheotomy.

Our patients with diagnosed, uncomplicated sleep apnea (group 1a) suffered from predominantly OSA without daytime hypoventilation, hypoxemia, or cor pulmonale. In this group, tracheotomy represented a highly effective treatment alternative for patients in whom conventional medical therapy, such as weight loss or nasal CPAP, failed. In addition, tracheotomy was used in a substantial number of these patients with uncomplicated sleep apnea undergoing UPPP to prevent perioperative upper airway compromise. In patients undergoing tracheotomy in conjunction with UPPP, therefore, a sleep study should be performed 1 to 2 months postoperatively with the tracheotomy closed to identify patients whose OSA persists (UPPP failures). Thus, tracheotomy should be considered a long-term treatment option for patients with uncomplicated OSA in whom other medical (eg, nasal CPAP, weight loss) or surgical (UPPP) therapy fails.

Our patients with sleep apnea (group 1b) with cardiopulmonary decompensation were those characterized by morbid obesity, hypoventilation and hypoxemia that worsened during sleep, and right-sided heart failure (pickwickian syndrome). As in the previous group (group 1a), tracheotomy was performed to treat patients in whom conventional therapy failed. Our results indicated that tracheotomy was associated with significant improvement of sleep apnea. Although tracheotomy effectively reduced the AHI in these patients, more than half of these patients still demonstrated marked sleep apnea in follow-up sleep studies, even when their tracheotomies remained open. Persistent sleep apnea in these patients can be attributed to external obstruction of the tracheotomy orifice by chin and neck adiposity or to the appearance of central sleep apnea. Regardless of the mechanism for persistent sleep apnea, some of these group 1b patients continued to have alterations in both daytime and nocturnal gas exchange. These abnormalities suggest that tracheotomy may only partially alleviate alterations in ventilatory control, and suggest that other factors may also contribute to observed gas exchange disturbances. These factors may include morbid obesity and concomitant lung or cardiac disease. The failure to eliminate sleep apnea in each of these patients emphasizes the importance of obtaining a follow-up sleep study in any patient with OSA with pickwickian features and cardiopulmonary decompensation to identify those with suboptimal responses. A follow-up arterial blood gas evaluation may also help predict in which patients sleep-disordered breathing will persist after tracheotomy.

For patients whose OSA had not been diagnosed before tracheotomy (group 2), the sleep study served to identify those for whom the presence of OSA placed them at risk for developing upper airway obstruction and respiratory insufficiency after decannulation. Our results suggested that many patients undergoing tracheotomy in conjunction with laryngeal or other upper aerodigestive tract surgeries may have evidence of sleep apnea that could hinder tracheotomy decannulation. As many of our patients had evidence of laryngeal abnormality, it is likely that patients with alterations in laryngeal function may be at increased risk for the development of sleep apnea. Again, a sleep study before decannulation is required to identify patients who might require treatment for sleep apnea even after their tracheotomies are removed.

In conclusion, our study describes 2 groups of apneic patients who exhibited distinct responses to tracheotomy. The role of sleep studies in each patient group has also been outlined. Although our study is limited by its relatively small sample size and retrospective nature, our results suggest that tracheotomy is associated with good success in treating patients with uncomplicated sleep apnea and with variable success in treating sleep apnea complicated by other diseases of the lung, heart, or aerodigestive tract.

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