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Acute Airway Obstruction after Uvulopalatopharyngoplasty for Obstructive Sleep Apnea Syndrome

M. R. GABRIELCZYK, M.B., F.F.A.R.C.S.*

The obstructive sleep apnea syndrome (OSAS) is characterized by the frequent cessation of breathing during sleep, which occurs as the result of intermittent mechanical occlusion of the airway and is often accompanied by a disorder of respiratory drive.[1] The preferred surgical treatment for OSAS has been tracheostomy, which allows bypass of the obstruction in the upper airway and usually leads to the alleviation of symptoms of the disorder.[2] Recent evidence indicates that uvulopalatopharyngoplasty (UPPP, *i.e.*, the surgical removal of redundant oropharyngeal tissues) in patients with OSAS reduces the frequency and duration of apneas, improves nocturnal oxygenation, and considerably relieves symptoms.[3,4] The features of obstructive sleep apnea and the anesthetic management of patients with OSAS for tracheostomy have been reviewed elsewhere,[5,6] but there is little information in the literature concerning the anesthetic implications of the relatively new procedure of UPPP. A major dissimilarity from tracheostomy is that difficulties of airway control as the result of mechanical obstruction may occur in the postoperative period after UPPP despite the resection of appreciable amounts of oropharyngeal tissue during the procedure.

This report describes the perioperative course of a patient with OSAS who developed life-threatening respiratory obstruction and central apnea following a stormy emergence from general anesthesia after UPPP.

REPORT OF A CASE

A 29-yr-old man was admitted for UPPP. A diagnosis of OSAS had been made on a prior admission when he had presented with a 12-month history of progressive weight gain, excessive daytime somnolence, and extremely loud snoring at night. Confirmation of the diagnosis by polysomnography ("sleep studies") revealed 380 episodes of apnea of over 10 s duration during seven hours of sleep. These were predominantly obstructive, although some mixed apneas (30%) and a few central apneas (2%) also occurred. At the preoperative visit the classic history of excessive sleepiness and loud snoring was confirmed. In addition, the patient's wife had noticed profound alterations in her husband's sleep behavior including somnambulism at night, and confusion and severe agitation on awakening from sleep. On occasions the patient had even prepared and eaten meals in his sleep with no subsequent recollection. There was no other relevant medical or surgical history. Examination revealed an anxious, thick-set muscular man. His weight was 98 kg and height 1.8 m, although he was not obese. Arterial blood pressure was 150/90 mmHg with a heart rate of 86 beats/min. Examination of the airway revealed good mouth opening with a full view of a low hanging soft palate, and a partially visible uvula. The neck, although short and thick, had a full range of movement. The remainder of the physical examination revealed no abnormality, and laboratory investigations (full blood count, serum electrolytes, chest radiographs, and electrocardiogram) were within normal limits.

The patient was premedicated with 10 mg diazepam orally two hours prior to surgery and observed in a preoperative holding area where he was said to be calm and alert. Anesthesia was induced with 500 mg of thiopental and 100 mg of succinylcholine iv. Control of the airway and ventilation by mask was easy, and orotracheal intubation was accomplished without difficulty, although only the arytenoid cartilages were visualized at laryngoscopy. Anesthesia was maintained with 0.5–1% halothane and 66% nitrous oxide in oxygen, with atracurium 70 mg iv in incremental doses and one single iv dose of 250 μg of fentanyl prior to incision. Surgery and anesthesia proceeded uneventfully for 150 min. Halothane was discontinued 15 min prior to the termination of the procedure at which time residual muscle relaxation was reversed and restitution of neuromuscular function verified with a peripheral nerve stimulator. A soft, red rubber nasopharyngeal airway was inserted at this time. The patient commenced spontaneous respiration with a rate of 8 breaths/min, opened his eyes upon hearing his name, and began responding appropriately to simple commands. However, after approximately a dozen shallow breaths he suddenly and dramatically became violently agitated, sitting upright on the operating table, throwing his limbs around, clawing at his endotracheal tube, and disconnecting most of the monitoring equipment in the process. Physical restraint proved impossible, and the decision was made to extubate the trachea. This immediately caused his agitation to disappear, and while 100% oxygen was administered by face mask, the monitoring equipment was reconnected. It was immediately noted that although still apparently awake and responding, his respiratory rate was now 2–3 breaths/min, and he was making only partially effective, stertorous respiratory efforts with rocking motions of his chest and abdomen. No stridor was evident. Attempted assisted ventilation *via* mask proved ineffective in overcoming the obstruction, and arterial oxygen saturation (Sa_{O_2}) rapidly fell to 82% over a period of approximately two minutes. Further bouts of violent movement of limbs and trunk and increased masseter tone made manipulation of the airway impossible. It was not considered appropriate to administer a neuromuscular blocker at this time because the integrity of iv access was doubtful, and the only gas exchange that was occurring was being accomplished by the patient's own spontaneous efforts. These were nevertheless insufficient to prevent a continuing fall in arterial oxygen saturation to 67% at which time cricothyroid puncture with a 14-G cannula was performed

* Associate in Anesthesiology.

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Address reprint requests to Dr. Gabrielczyk: Department of Anesthesiology, Duke University Medical Center, Box 3094, Durham, North Carolina 27710.

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and transtracheal ventilation attempted by manual compression of the reservoir bag. This proved unsuccessful in delivering adequate tidal volumes, and the Sa_{O_2} dropped precipitously reaching a nadir of 37%. An emergency tracheostomy was therefore immediately performed and adequate ventilation reestablished. Vital signs at this time were heart rate 126 beats/min, arterial blood pressure 180/100 mmHg, and end-tidal CO_2 57 mmHg. ECG showed tachycardia. Approximately five to six minutes had elapsed from the time of extubation until control of the airway was regained. Anesthesia was now maintained with 0.5% halothane and 50% nitrous oxide in oxygen and 20 mg atracurium iv while hemostasis was assured, the tracheostomy tube secured, and a larygoscopy performed. This revealed a moderately edematous palate and oropharynx but no other abnormality. On completion of this procedure 220 min after initial induction of anesthesia, halothane and nitrous oxide were again discontinued and residual neuromuscular blockade reversed. A stormy emergence identical to that which had occurred previously took place, with periods of violent agitation and movement of limbs, alternating with episodes of hypopnea and apnea. On this occasion, oxygenation was maintained by assisting ventilation *via* the tracheostomy tube. Subsequently, over a period of five to ten minutes the patient became cooperative with adequate spontaneous ventilation, and when stable was returned to the recovery room for overnight observation. Forty percent oxygen by tracheostomy mask was continued for 24 hours. His remaining postoperative course was uneventful. At six-month follow-up the tracheostomy track had healed and the patient's daytime somnolence and nocturnal snoring had ceased. He had no recollection of the events that had taken place between the times of induction of anesthesia and his arrival in the recovery room.

Discussion

The majority of cases of OSAS respond to medical therapy. Protriptyline and medroxyprogesterone are the two most commonly prescribed therapeutic agents, although drug treatment alone is often disappointing. In many centers nonsurgical treatment of OSAS therefore includes the use of mechanical methods that increase intrapharyngeal pressure and thus splint the airway open. Continuous positive airway pressure (CPAP) applied by nasal mask is probably the most effective such technique, although a variety of other devices have been used successfully. In the case reported here the degree of clinically evident redundant oropharyngeal tissue and the severity of symptoms had led to the decision to perform UPPP without a prior trial of conservative treatment. First described in 1964 for the treatment of snoring,[7] UPPP has only recently been popularized for the treatment of OSAS. Although detailed differences exist in the exact procedure performed depending upon the center, UPPP usually consists of tonsillectomy and excision of the soft palate and removal of tissue from the posterior tonsillar pillar, which is then sutured to the anterior tonsillar pillar. If the severity of airway obstruction is considered to be life-threatening, a tracheostomy is performed together with UPPP, but in many cases of mild to moderate OSAS unresponsive to medical therapy, UPPP alone is said to be the surgical treatment of choice.[3] Previous publications concerning the anesthetic management of patients with OSAS[5,6] have emphasized the hazards of preoperative sedative medication and airway difficulties on induction of anesthesia, but with the increasing popularity of UPPP without concomitant tracheostomy, the resulting potential for postoperative problems of airway control has become greatly increased. This patient exhibited classic features of moderately severe obstructive sleep apnea and exemplifies the type of case commonly presenting for UPPP. Such individuals are known to often have subtle anatomic narrowing of the oropharynx[8] and reduced oropharyngeal muscle tone,[9] which together result in periods of total airway obstruction during sleep. These apneas are longest and most frequent during rapid eye movement (REM) sleep as a result of the muscle hypotonia and depressed ventilatory responses to hypoxemia and hypocarbia, which are most marked during this phase of sleep.[10] A variable number of central and mixed (*i.e.*, combined central and obstructive) apneas will often also occur as the result of an associated disorder of central respiratory drive, the nature of which remains poorly understood. Arousal, which is characterized by abrupt muscular activity, usually occurs when asphyxic stimuli are sufficient to cause an increase in oropharyngeal muscle tone of adequate degree to reopen the airway.[11]

Postoperative airway obstruction immediately following UPPP has not previously been reported, although it may occur as a late complication.[12] Whereas preoperative airway control and ventilation by mask had been easy, only small inadequate tidal volumes could be delivered after extubation, at which time it was evident that significant airway obstruction was present in the oropharynx despite the excision of palatal and pharyngeal tissues during the surgical procedure. Although laryngospasm may produce acute postextubation airway obstruction, the absence of laryngeal stridor during the periods of stertorous, obstructed respiration following extubation makes this possibility unlikely. The amount of oropharyngeal edema present by the end of surgery was probably sufficient to cause this airway obstruction despite the presence of the soft rubber oropharyngeal airway, which might itself conceivably have been compressed and even occluded by palatal swelling. Certainly, laryngoscopy had confirmed a greater degree of oropharyngeal and palatal edema than had been expected. The effects of airway obstruction were compounded by the occurrence of apnea after extubation. Indeed, on both occasions of recovery from anesthesia, these periods of central apnea occurred despite apparent wakefulness and appropriate response to name and simple command. The etiology of these apneas is unclear. Although diazepam and fentanyl can cause depression both of central respiratory drive and of upper airway muscle tone of sufficient degree to precipitate airway obstruction,[13,14] it is improbable that these depressant effects could have persisted for sufficiently long to have contributed to either airway obstruction or apnea in this instance,

particularly in view of the patient's apparently maintained level of consciousness. Halothane, however, decreases the ventilatory response to hypoxia in subanesthetic concentrations as low as 0.1 MAC,[15] and in higher dosage causes central apneas in individuals with obstructive sleep apnea.[16] It also reduces oropharyngeal muscle tone in a dose-related manner in animals[17] and may therefore exacerbate any residual tendency to airway obstruction. In addition, a recent report has suggested that central apneas may become unmasked after surgery to relieve the obstructive component of OSAS.[5]

The striking similarity in this patient between the wild agitation seen on emergence from anesthesia and that described by his wife as occurring on arousal from sleep is another notable feature of the case. Although, in the author's experience, this "emergence delirium" is an uncommon phenomenon, the number of anecdotal accounts of similar occurrences in patients with sleep apnea obtained from colleagues suggests that it may not in fact be rare. Indeed, postoperative agitation of sufficient severity to interfere with ventilation has been previously described in a sleep apnea patient after elective tracheostomy,[18] although in that particular instance bronchospasm also contributed to the difficulty. Although there is no evidence at present to link stormy emergence from anesthesia with agitation on arousal from sleep, if viewed as a disorder of waking, the two phenomena would appear to be closely related. Their strong similarity alone suggests the possibility of an association, but further study is required to establish whether the preoperative elicitation of a history of agitated arousal from sleep is of value in the prediction of a stormy emergence from general anesthesia. Lastly, it is interesting to speculate on the possibility that just as this patient was able to execute complex tasks in his sleep during periods of somnambulism in the absence of full consciousness, so he was also able to obey simple commands in the initial phase of anesthetic recovery, leading to the mistaken impression that he was more awake than was really the case.

Whereas publications concerning the anesthetic management of patients with OSAS for tracheostomy have emphasized the hazards of the preoperative and induction periods, with the increasing popularity of UPPP postoperative problems of airway control are likely to become of equal concern to the anesthesiologist. This report draws attention to the potentially dangerous combination of postoperative oropharyngeal edema and residual effects of light general anesthesia after UPPP in the patient with obstructive sleep apnea syndrome, and suggests that in some of these patients appropriate responses to command may be an unreliable criterion for extubation following general anesthesia. In conclusion, loss of airway control in the patient with sleep apnea undergoing UPPP is as great a hazard of anesthetic emergence as of induction, and that extubation of the trachea should be performed only when there is absolute certainty of a full return to consciousness, and when airway patency is assured. Furthermore, surgical personnel and instruments should routinely remain present in the operating room until after successful extubation has been accomplished in case emergency tracheostomy is required.

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