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The Occurrence of Obstructive Sleep Apnea in a Recovery Room Patient

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Sleep apnea is a syndrome characterized by disorders of respiratory drive and/or airway control during sleep.[1,2] When viewed as a pathological consequence of depressed consciousness, sleep apnea may be accentuated by anesthesia. This hypothesis suggests that patients who exhibit a sleep apnea-like syndrome after pharmacologically induced sedation could have an underlying sleep-breathing disorder. If this is true, the perioperative period may be an effective time to screen patients for previously undiagnosed sleep apnea through the preanesthetic history and perianesthetic observation.

We describe a patient in whom the initial suspicion of obstructive sleep apnea was based on postoperative observations made in the recovery room following spinal anesthesia and light sedation.

REPORT OF A CASE

A 70-yr-old man was admitted following trauma sustained while performing yard work. His prior medical history was complex, including multiple episodes of upper gastrointestinal bleeding, a left anterior hemi-block (LAHB) and right bundle branch block (RBBB) with syncopal episodes for which a permanent demand (VVI) pacer was inserted, and presence of mild congestive heart failure with occurrence of dyspnea on exertion. In 1979, he underwent uneventful retropubic prostatectomy for benign prostatic hypertrophy with epidural anesthesia. In 1982, he suffered a cerebral infarction, manifested by right facial weakness and dysarthria. These symptoms resolved within 4 months. At that time, the patient had an elevated hemoglobin (19.4 g/dl). The diagnosis of secondary erythrocytosis of unknown etiology was made. In February 1984, the patient experienced another episode of dysarthria and vertigo. He also had decreased strength in his tongue and masseter muscles. No new lesions were found on brain CT scan.

In September 1984, the patient underwent urgent cholecystectomy. Upon tracheal extubation, he had an episode of respiratory distress described on his chart as laryngospasm lasting 30 min, and requiring reintubation. He developed transient noncardiogenic pulmonary edema (pulmonary capillary wedge pressure 9–12 mmHg, pulmonary artery pressures 35/20/10 mmHg, central venous pressure 9 mmHg, and cardiac output 6.2 L/m). Subsequently, the patient made an uneventful recovery.

In December 1984, the patient sustained fractures of six ribs on the left, without flail, and an ankle fracture when a tree limb fell on him. At the time of admission, arterial blood pressure was 140/100 mmHg and heart rate was 100 beats/min. He was described as being moderately obese, with height of 172 cm and weight of 80 kg. On the seventh day postadmission, the patient underwent open repair of his ankle fracture. Spinal anesthesia was established for this procedure with 10 mg tetracaine 0.5% hyperbaric with 0.3 mg epinephrine; a T-6 sensory level was obtained at 15 min. The patient received 100 mcg of fentanyl iv in incremental doses for sedation. Over the ensuing 5 min, he became somnolent, and developed a respiratory pattern of airway obstruction with "rocking" motions of the chest at a rate of 24/min. These obstructive respiratory efforts were characterized by a progressive increase in apparent diaphragmatic effort as the episode proceeded, and terminated with the patient stirring and clearing his airway, which was accompanied by loud gasping and snorting sounds. These episodes persisted into the recovery room with a duration of up to 50 s each, and a period of three min between onsets of apnea. The pattern could be interrupted by patient arousal, which was easy to accomplish with light tapping or soft speech. Placement of a soft rubber nasopharyngeal airway did not alter the pattern of intermittent obstruction. During arousal, the patient demonstrated normal symmetrical upper extremity strength and sustainable head lift; sensory level remained at T-6.

Overnight, we monitored O_2 saturation *via* pulse oximetry and cardiac rhythm with ECG; narcotic analgesia was not given. In the following 48 h, apnea episodes continued during sleep, and pulse oximetry revealed multiple transient hemoglobin desaturations on room air from 97–80% accompanied by multiple cardiac dysrhythmias. An interview with the patient's wife revealed a lifelong history of snoring during sleep, and a 2-yr history of worsening daytime somnolence. The tentative diagnosis of obstructive sleep apnea was made. Due to the patient's unstable medical condition, formal sleep laboratory testing was deferred; bedside sleep polygraph recordings were technically unfeasible. Therefore, tracheostomy was recommended based upon the patient's overall clinical assessment, in keeping with previously established clinical surgical criteria.[3] An otolaryngologist examined the patient prior to tracheostomy; they reported right facial droop, right ptosis, and dysarthria. The procedure was performed under local anesthesia; post-tracheostomy, he maintained normal (>95%) saturation during sleep. Following hospital discharge, the patient underwent polysomnography with his tracheostomy occluded. The diagnosis of obstructive sleep apnea was confirmed, with an apnea index of 25, and it was recommended that the tracheostomy be maintained.

DISCUSSION

The sleep apnea syndrome was initially described by several investigators[4–6] working with patients with the Pickwickian syndrome. Typically, the patient with sleep apnea presents with daytime somnolence, which may be

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sufficiently severe to cause disorientation and personality changes.[7] The patient himself is frequently unaware of nocturnal symptoms; however, when questioned, the bed partner will usually report loud snoring and gasping sounds and nocturnal breath cessations.[3] The diagnosis is based on the demonstration of 30 or more episodes of total apnea, each of at least 10 s duration, during a 7-h period of sleep. Sleep apnea is diagnosed as being either obstructive, central, or mixed. Obstructive apnea is the most prevalent.[8,9] As the name suggests, obstructive apnea is characterized by hypo-pharyngeal or tongue obstruction of the airway with intact respiratory muscle efforts. It can be distinguished by the persistence of respiratory muscle effort in the absence of air flow at the nose or mouth. Central apnea presents with a diminution in respiratory muscle effort secondary to diminished drive, as in Cheyne-Stokes or Biot breathing.

The hypoxic and hypercarbic stress of these apnea episodes can result in polycythemia. Pulmonary hypertension and cor pulmonale can be induced by the ongoing hypoxia and acidosis.[10] Bradycardia, atrioventricular blocks, and ventricular tachycardias have been recorded in conjunction with sleep apnea episodes.[11,12]

A hypoplastic jaw* and prior cerebral infarction with bulbar symptoms[13] predisposed this man to obstructive sleep apnea. The laryngospasm encountered during the prior anesthetic was probably hypopharyngeal or tongue obstruction. On inquiry, the patient's wife reported loud snoring by her husband when asleep; in retrospective chart review, mention had been made in nursing notes of loud snoring. The patient's hypoventilatory response differed from usual narcotic respiratory depression in its obstructive character, with maintenance of rapid, but ineffective, respiratory muscle efforts. The alleviation of apneas following tracheostomy is inconsistent with a diagnosis of central sleep apnea. His normal upper extremity strength and sustainable head lift ruled out generalized neuromuscular weakness. In addition, with a clearly defined spinal level of T-6, we deemed it unlikely that local anesthesia of the brainstem could account for the observed apneas.

The anesthetic management for tracheostomy in sleep apnea patients has been discussed.[14,15] Little has been said, however, concerning undiagnosed and unsuspected sleep apnea patients presenting for surgery unrelated to their respiratory disorder. This case report is unique in that the diagnosis of sleep apnea was suggested by the patient's intra- and postoperative airway symptoms following minimal sedation. Extrathoracic airway (pharynx, hypopharynx) patency has been shown to rely on an active neuromuscular mechanism[16] which is blunted by general anesthesia.[17] Likewise, the precipitation of upper airway obstruction in patients with known sleep apnea by minimal sedation is not a new concept.[18–22] There is some evidence of a relationship between the effects of the diminished consciousness of sleep and pharmacologic sedation on upper airway tone and central respiratory drive. Therefore, the presumptive diagnosis of sleep apnea in the post-anesthetic period rests on the observation of upper airway obstruction, out of proportion to the level of sedation. The determination of what constitutes an "inappropriately severe" response for a given level of sedation obviously requires experienced clinical judgement; however, the presence of obstructive apnea during light sedation without motor paralysis should alert the clinician to this potential diagnosis.

Based on this case and other anecdotal examples, a study aimed at assessing this relationship is warranted; such a study might easily be accomplished by the referral of patients with severe intra- and postoperative airway obstruction for sleep evaluation. In addition, the assessment of respiratory function during sleep by eliciting symptoms of loud snoring, gasping respirations and breath cessation during sleep, or daytime somnolence may be a useful addition to the routine preanesthetic evaluation, in that it may be predictive of potential obstructive airway problems in the anesthetized patient whose trachea is not intubated.

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Transient Hypoxemia from a Transient Right-to-Left Shunt in a Child During Emergence from Anesthesia

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Hypoxemia during and after anesthesia may be secondary to a number of changes in the circulatory and respiratory systems.[1] We have observed that some infants develop signs of hypoxemia (cyanosis, decreased transcutaneous PO_2, decreased arterial saturation) during emergence from anesthesia. This decrease in oxygenation occurs despite continued ventilation of the lungs with 100% oxygen. We speculate that straining against a tracheal tube causes increased airway resistance and pulmonary vascular resistance. Consequently, a right-to-left intracardiac shunt at the atrial level could occur in patients with a persistent foramen ovale. The following case report demonstrates such a phenomenon.

REPORT OF A CASE

The patient was a 13-month-old male child who previously had two uneventful general anesthetics. The patient underwent inhalation anesthesia for eye examination and possible lens aspiration. The preanesthetic examination showed a normally developed patient with left microphthalmia, with clear lungs on auscultation and no cardiac murmurs. Following induction of anesthesia with halothane nitrous oxide oxygen, an iv catheter was inserted. Atropine 10 μg/kg, thiopental 4 mg/kg, and succinylcholine 2 mg/kg were administered iv. The trachea was intubated with a 3.5 mm I.D. tracheal tube. In addition to the usual monitors, we used a pulse oximeter (Nellcor® B-100, Nellcor, Inc., Hayward, CA). Following tracheal intubation, the lungs were ventilated with an inspired oxygen concentration of 35%, resulting in an arterial oxygen saturation of 99%. Twenty-five minutes after endotracheal intubation and during the eye examination, the patient began to cough and strain against the tracheal tube. The arterial oxygen saturation rapidly declined to 63%, despite ventilation with 100% oxygen. Arterial saturation increased to 99% with continued ventilation with 100% oxygen. The level of anesthesia was deepened by increasing the inspired halothane concentration, and the procedure was continued. During emergence from anesthesia, the patient again strained and coughed against the tracheal tube. Arterial saturation decreased to 80%. At that time, a contrast echocardiogram with stirred saline revealed a right-to-left shunt at the atrial level (figs. 1 and 2). No shunt was present when the child was not straining. A chest radiograph showed the tracheal tube to be in the mid-trachea, and the lung fields were clear. The arterial saturation again increased to 99% within 1 min by continued ventilation with 100% oxygen. The trachea was extubated when the child was fully awake. The post-anesthesia recovery period was uneventful.

DISCUSSION

An interatrial right-to-left shunting has not been described as a cause of hypoxemia associated with emergence

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