Episodic Complete Airway Obstruction in Children with Undiagnosed Obstructive Sleep Apnea

STUART WEINBERG, M.D.,* RICHARD KRAVATH, M.D.,† LYDIA PHILLIPS, M.D.,§ HERMAN MENDEZ, M.D.; GERALD L. WOLF, M.D.$

Unrecognized and unanticipated episodes of acute upper airway obstruction associated with obstructive sleep apnea can result in death. The diagnosis should be considered in children with acute airway obstruction and in patients scheduled for tonsillectomy and adenoidectomy. We describe two patients with obstructive sleep apnea syndrome and acute upper airway obstruction.

REPORTS OF TWO CASES

Case 1. An 11-year-old girl arrived at another hospital with extreme respiratory distress. The patient had a three-day history of headache, sore throat, and fever and had been given penicillin orally. On the night of admission the parents were aroused by noisy breathing during sleep and found the child cyanotic and unresponsive. In the emergency room, the clinical diagnosis of epiglottitis was made. The trachea was intubated in the operating room at which time a "red swollen" epiglottis was observed.

Prior history disclosed restless sleep with loud snoring and frequent awakenings. She also was obese and had a history of asthma. The patient had frequent bouts of tonsillitis and chronic tonsillar hypertrophy. Tonsillectomy had been recommended on several occasions over the prior two years but was not done because of repeated respiratory tract infections. The remainder of the physical examination was within normal limits.

The patient was transferred to the pediatric intensive care unit of our hospital. Intravenously administered fluids, endotracheal tube care, antibiotics iv, steroids iv, and sedation were provided. The patient's condition improved, and her trachea was extubated 24 hours after admission. Throat and blood cultures were negative.

Although she had no respiratory obstruction while awake, she had noisy respiration, loud snoring, dyspnea, restlessness, diaphoresis, perioral cyanosis, and absent airflow through nose and mouth for brief periods while asleep followed by arousal and re-established airflow. Fiberoptic examination of the naso-opharynx revealed a normal epiglottis with no evidence of residual hyperemia, edema, or resolving infection. Tonsils and adenoids were enlarged and obstructing the airway. Tonsillectomy was again advised but she was first managed and monitored in the pediatric intensive care unit while the acute infection subsided. Episodes of respiratory obstruction occurred during sleep over the next 10 days as attempts to keep a nasopharyngeal airway in place were unsuccessful. Twelve days following admission, a tonsillectomy and adenoidectomy were planned.

Atropine, 0.4 mg, was given im prior to induction of anesthesia with inhalation of increasing inspired halothane concentrations. Airway obstruction was relieved by a jaw thrust maneuver. Following several minutes of ventilation with 100% O₂ and high concentrations of halothane, her trachea was intubated without difficulty while she was breathing spontaneously. Following an uncomplicated resection of tonsils and adenoids, bilateral nasopharyngeal airways were inserted and the endotracheal tube removed once the patient regained full consciousness.

Following arrival in the recovery room, the child removed the bilateral nasopharyngeal tubes and subsequently developed airway obstruction that was relieved with the jaw thrust maneuver. She continued to have episodes of airway obstruction which subsided over the next 24 hours. No further episodes of airway obstruction have been noted.

Case 2. A five-year-old boy was scheduled for a routine tonsillectomy and adenoidectomy. On the morning of surgery he was asleep in the holding area of the operating room with moderate to severe respiratory obstruction and with episodes of loud snoring interrupted by periods of absent air flow and associated with marked intercostal and suprasternal retraction along with nasal flaring. Review of the child's history revealed loud snoring during sleep and history of repeated bouts of tonsillitis. Prior to the transfer to the operating room area, he received im atropine, 0.02 mg/kg, meperidine, 1 mg/kg, and pentobarbital, 2 mg/kg. In the operating room, a transcutaneous PO₂ monitor was used to monitor respiratory status while breathing room air prior to induction of anesthesia. Transcutaneous PO₂ during obstructed sleep ranged from 60 to 65 mmHg. When the child was aroused and prevented from resuming sleep, the transcutaneous oxygen tension rose to 95 mmHg and an arterial sample obtained at that time had a pH of 7.38, Paco₂ 44 mmHg, and a Po₂ 113 mmHg.

Anesthesia was induced by inhalation of increasing concentrations of inspired halothane. Early airway obstruction was managed with a jaw thrust maneuver and gentle insertion of a nasopharyngeal airway. Following the iv administration of succinylcholine, the trachea was intubated, and anesthesia maintained with nitrous oxide, oxygen, and halothane. At the conclusion of the tonsillectomy and adenoidectomy, bilateral nasopharyngeal tubes were inserted and the endotracheal tube removed in the operating room when the patient was fully awake. The patient was transferred to the recovery room for several hours and then observed in the pediatric ICU for an additional 24 hours when the nasopharyngeal tubes were removed. No further episodes of obstruction were noted and the patient is doing well at home.

DISCUSSION

The first case illustrates the many problems associated with the diagnosis and management of children with obstructive sleep apnea. This disorder usually presents with a long history of transient episodes of respiratory ob-
struction during sleep. Tonsils and adenoids generally are enlarged and exacerbations of sleep apnea may occur during periods of upper respiratory tract infection.2

Although the pathophysiology of obstructive sleep apnea is not clear, enlarged tonsils and adenoids in late infancy and childhood are a factor since the syndrome generally resolves following adeno-nectomy and tonsillectomy.2,3 Other theories suggest the involvement of central mechanisms with resultant decreases in muscle tone of upper airway structures crucial to the maintenance of airway patency.4 Cinefluoroscopy can demonstrate occlusion of the pharyngeal and hypopharyngeal airway during sleep.5 These studies show that during obstructive sleep apnea, the soft palate falls backward against the posterior pharynx, the tongue moves posteriorly, and the lateral walls of the hypopharynx approximate medially during inspiration. Attempts to overcome the obstruction with gasping are followed by partial arousal with resumption of airflow marked by loud snoring.

Clinical experience and experimental evidence suggests that sedation and anesthesia can exacerbate these mechanisms.4,5,7 Maintenance of muscle tone in the genioglossus and geniohyoid muscles are essential in preventing passive pharyngeal closure during respiratory efforts that may generate up to –100 cmH2O of pressure.4-6 Several radiographic studies have demonstrated posterior displacement of the tongue against the posterior oropharyngeal wall during anesthesia. Brouillette and Thach4 showed that deepening levels of anesthesia caused decreasing tone in genioglossus and geniohyoid muscles of rabbits, and these correlated with EMGs and electromyograms that were depressed to a greater extent than diaphragm and other accessory muscles of respiration.

Arousal following periods of obstructive apnea appear to be crucial in re-establishing airflow. Increasing levels of sedation depress the arousal that accompanies obstructive sleep patterns in unseated sleep. Remmers et al.8 observed that in patients with obstructive apnea, preferential recruitment of the genioglossus muscle was associated with generalized “arousal” pattern on the electroencephalogram. This arousal pattern may be triggered by asphyxial blood-gas tensions. In addition to the arousal mechanism, asphyxial depression of electrical activity in the cerebral cortex initiates “cortical release” mechanisms that produce involuntary cervical extension in children, in addition to augmentation of genioglossus activity.8,10 Cervical extension, a maneuver well known to anesthesia personnel, draws the tongue forward and thereby enlarges the pharyngeal airway.11

The presentation of the child in case 1 was especially confusing since she presented to another hospital initially and was transferred to our institution intubated with a diagnosis of epiglottitis. Several factors, however, suggest that the diagnosis of epiglottitis may have been erroneous. The child’s older age, the chronic history, the lack of stridor, the history of snoring, and the lack of positive cultures argue against a diagnosis of epiglottitis.12 The unconfirmed report of a “swollen red” epiglottis and the history of penicillin use before blood cultures makes the situation confusing. The child may have presented simultaneously with two disorders, epiglottitis and obstructive sleep apnea. A normal fiberoptic or direct laryngoscopy examination on admission could have resolved this diagnostic dilemma.15

The distinction between these two entities is important since the management of these two disorders is different. Children with epiglottitis require temporary management with orotracheal or nasotracheal intubation.12 On the other hand, patients with obstructive sleep apnea do not need tracheal intubation; they generally are in no danger when awake and arousal from sleep terminates the obstruction. Careful monitoring, head tilt, neck extension, and placement of a nasopharyngeal airway is often all that is necessary until tonsillectomy can be performed.9

Obstructive sleep apnea should not be confused with forms of central sleep apnea. Patients with central apnea have no respiratory movement during apnea and consequently, standard apnea monitors effectively alarm during central apneic episodes. Patients with obstructive sleep apnea continue to make respiratory effort and therefore may not trigger these alarms.14 Such patients require the availability of air flow-sensitive monitors or cardiac monitors that alarm with the onset of apnea-associated bradycardia.15

The second case emphasizes the importance of considering the possibility of undiagnosed obstructive sleep apnea in all children presenting to the anesthesia and surgical services for tonsillectomy and adenoidectomy, especially if the parents report a history of loud snoring during sleep.2 Parents of a child with obstructive sleep apnea also may relate the observation of restless disturbed sleep, and blockage of breathing that may be associated with cyanosis, diaphoresis, nightmares, enuresis, and peculiar behavior. Other findings include excessive daytime sleepiness, morning headaches, poor growth and development, dysphagia, mood changes, and declining school performance.2,16,17

Physical examination of the awake patient may reveal mouth breathing and noisy respiration. During sleep, auscultation over the nose, mouth, and chest of the patient and observation for chest wall retractions, cyanosis, and bradycardia can precisely define obstructive apneic episodes. Other complications of chronic airway obstruction and sleep apnea that should be considered include systemic hypertension, cardiac arrhythmias, pulmonary hypertension, cardiac hypertrophy, congestive heart failure, and sudden death.1,2,17,18

Laboratory investigation that may detect deterioration
in ventilatory status and aid in the diagnosis include continuous PaO₂ and PaCO₂ monitoring obtained noninvasively by continuous transcutaneous PO₂ and PCO₂ electrodes. Improvement in ventilatory function with placement of nasopharyngeal tubes in patients with suspected airway obstruction can confirm the diagnosis. Polysomnography can document the frequency and duration of the apneic episodes, and a cephalometric lateral neck radiograph can establish the size of the adenoids in relation to the dimension of the surrounding oropharynx. In patients with chronic hypoventilation, the hemoglobin concentration may be elevated and a compensatory metabolic alkalosis may be detected.

The primary consideration in anesthetic management is the maintenance of a patent airway which may be facilitated by spontaneous respiration during induction. Obstruction can be managed with a jaw thrust maneuver or the insertion of a oral airway. If these maneuvers are inadequate, endotracheal intubation or nasopharyngeal airway insertion may be necessary, although the latter maneuver may produce adenoidal bleeding. These patients continue to be at high risk for obstruction in the immediate postoperative period and consequently, bilateral nasopharyngeal airways inserted under direct visualization intraoperatively by the surgeons and the tonsil position postoperatively are often helpful in maintaining airway patency. Endotracheal tubes generally can be removed in the operating room provided that the patient is fully awake and the nasopharyngeal tubes are functioning well. Postoperative care should include at least 24 hours of observation and monitoring in a recovery room or intensive care unit. The nasopharyngeal tubes may be removed on the morning after the operation; however, following removal the patient’s breathing during sleep must be checked carefully by an experienced observer before discharge, since airway obstruction may persist postoperatively for a variable period of time.

Recognition of the importance of obstructive sleep apnea as a distinct clinical entity along with appropriate skilled high-risk airway management will minimize the morbidity and mortality of this potentially life-threatening disorder.

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