Anesthesiology
2000; 92:1209-10

Acute Pulmonary Edema Caused by Impaired Switching from Nasal to Oral Breathing in the Emergence from Anesthesia

To the Editor:—Pulmonary edema occurs because of excessive negative intrathoracic pressure caused by acute airway obstruction (*e.g.*, laryngospasm, vocal cord paralysis) in spontaneously breathing patients.[1-4] During anesthesia and sedation, airway obstruction can occur at the pharynx and larynx levels.[5,6] Even in patients who are awake, alteration in the ability to change the breathing route from nasal to oral may affect breathing against the airway obstruction, thereby causing this catastrophic event.[7] We experienced a case in which acute pulmonary edema occurred because of acute airway obstruction triggered by the patient's inability to switch the breathing route from nasal to oral during emergence from propofol anesthesia.

A 27-yr-old man (170 cm, 60 kg) with chronic tonsillitis scheduled for bilateral tonsillectomy was medicated with hydroxyzine and atropine 30 min before anesthesia. Air passage through the nose was normal. After placement of the electrocardiograph, pulse oxymeter (oxygen saturation measured by pulse oximetry, Sp_{O_2}), and blood pressure cuff, general anesthesia was induced with 2 mg/kg intravenous propofol and was maintained with continuous intravenous propofol, nitrous oxide, and oxygen supplemented with fentanyl. The patient's trachea was intubated with a 7.5-mm endotracheal tube with the aid of vecronium for muscle relaxation; the lungs were mechanically ventilated. A bilateral tonsillectomy was performed uneventfully, using 13 ml local-infiltration epinephrine 0.0005%. Because bleeding persisted, the surgeon replaced a mouth gag several times for hemostasis; thus, the procedure was accomplished in 180 min, during which time 2 $mg \cdot kg^{-1} \cdot h^{-1}$ propofol supplemented with 0.2% isoflurane was maintained.

The patient began controlled mechanical ventilation, and his vital signs were stable until tracheal extubation after full recovery from neuromuscle blockade. He could raise his head, and he responded well to our commands. The patient then fell asleep, and his airway was obstructed. When the patient awoke to our verbal commands, he was able to breathe orally without difficulty; however, when he slept, he could only breathe through the nasal route. The nasal breathing was accompanied by signs of complete airway obstruction, such as sternal retraction and use of the accessory muscles. With each awakening, he breathed through the oral route, and the obstructive breathing disappeared. After several such episodes Sp_{O_2} decreased to less than 90%, and finally to 84%, despite oxygen supplement through a face mask. A chest roentgenogram showed diffuse interstitial edema in his lungs. The patient had a continuous positive airway pressure (CPAP) mask with oxygen on overnight. The next morning, his lungs became clear, and there was neither a significant decrease in Sp_{O_2} associated with spontaneous breathing nor an episode of upper airway obstruction during sleep. The patient's recovery was uneventful.

Pulmonary edema in the postoperative period often is caused by excessive negative intrathoracic pressure generated during inspiratory efforts against acute upper airway obstruction,[1-3] mainly caused by laryngospasm in healthy male patients.[4] However, laryngospasm did not occur in this patient, although, in patients anesthetized with propofol, vigorous airway reflexes could be elicited by laryngeal stimulation.[8] Similarly, closure or paradoxic movements of the vocal cord could be excluded as a factor because the obstruction was above the vocal cords.

There could be many factors that cause partial obstruction of the upper airway after tonsillectomy. Edema in the velopharyngeal area and tongue base could have occurred because of the prolonged procedure in this patient, thereby narrowing the nasal breathing. During sleep, upper airway obstruction usually occurs at the velopharyngeal level and leads to nasal airway obstruction; switching the breathing route from the obstructed nasal route to the oral route is crucial for maintaining a patent airway. Because this switching mechanism is believed to be an upper airway reflex, impairment of any part of the reflex arc might have caused the delayed switch from nasal to oral breathing observed in this patient. For example, airway anesthesia of the nasal passage delays the shift to oral breathing in response to acute nasal obstruction.[9] Upper airway edema also might impair airway receptor function in this palatoglossal area, and residual general anesthetics can depress the arousal function. Residual muscular blockade might also impair upper airway muscle function. Moreover, propofol *per se* has been reported to cause velopharyngeal narrowing.[10] Although propofol seems unlikely to affect spontaneous inspiratory and expiratory times,[11] it may affect coordinated movements of respiratory muscles. During sleep, upper airway obstruction at the retropalantal level may be related to an imbalance between the activity of the levator and the tensor palatini. Both muscles are reflex activated by negative pressure in humans.[12] During oral breathing, levator palatini activity is increased significantly compared with nasal breathing, suggesting that the levator palatini lifts the soft palate upward and separates the nasopharynx from the oropharynx, at the same time pulling the palatal arches apart and upward, thus creating a patent oropharynx.[12] If the velopharyngeal occlusion occurs without the change from nasal to oral breathing, complete airway obstruction results. The current patient was able to release the upper airway obstruction exclusively in response to the commands to breathe orally. This suggests that the ability to change the breathing route was impaired, probably because of sedation with propofol.

We are not certain whether patients with enlarged tonsils, who might normally be unable to breathe *via* the nasal route, would have difficulty associated with the impaired ability to change the breathing route after tonsillectomy. However, because airway obstruction of the nasal route frequently occurs during emergence from anesthesia, especially after adenoidectomy or tonsillectomy, and because the switch from nasal to oral breathing is accomplished only when the patient awakens, one should be aware that disability or delay of switching from the nasal to the oral breathing route may provoke pulmonary edema.

Asako Furuhashi-Yonaha, M.D.
Instructor
Shuji Dohi, M.D.
Professor and Chair
shu-dohi@cc.gifu-u.ac.jp
Tsutomu Oshima, M.D.
Assistant Professor
Department of Anesthesiology and Critical Care Medicine
Gifu University School of Medicine
Gifu City
Gifu, Japan
Norio Ueda, M.D.
Vice Director
Department of Anesthesia
Gifu City Hospital
Gifu City
Gifu, Japan

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(Accepted for publication November 4, 1999.)

Anesthesiology
2000; 92:1210-1

An Efficient Technique for Tracheal Intubation Using the StyletScope Alone

To the Editor:—The StyletScope (NihonKoden Corp., Tokyo, Japan) is a new device for tracheal intubation. It is a light-weight stylet with a fiber-optic view, maneuverability of its distal tip, and a built-in light source. Previously, we reported that the StyletScope, in combination with a standard laryngoscope, allows successful intubation in patients with simulated difficult airway.[1] A similar fiber-optic device was reported to function well without a laryngoscope[2]; however, it remains unclear whether the StyletScope can be used for tracheal intubation without the aid of a standard laryngoscope. We prospectively assessed a new procedure for tracheal intubation using the StyletScope alone.

After obtaining approval from the Ethics Committee for Research of our institution and the informed consent of each patient, 11 patients undergoing general surgery participated in this study. Mean age, height, and weight values were 56 yr (range, 25–72 yr), 160 cm (range, 143–176 cm), and 56 kg (range, 38–77 kg), respectively. Using the Mallampati test modified by Samsoon and Young,[3] seven patients were classified as class I, three patients were classified as class II, and one patient was classified as class III.

Patients were premedicated with 0.01 mg/kg atropine and 0.5 mg/kg hydroxyzine. After preoxygenation, general anesthesia was induced with 2.0 μg/kg fentanyl and 1.0 mg/kg propofol, with subsequent infusion at a rate of 10 $mg \cdot kg^{-1} \cdot h^{-1}$ propofol and 0.15 mg/kg vecuronium. With the patient's head and neck in the sniffing position and the lower jaw held upward by an assistant, an endotracheal tube (ETT) with the StyletScope was inserted into the mouth. At this point, the back of epiglottis could be viewed through the eyepiece of the StyletScope (fig. 1). By advancing the tip into the space between the epiglottis and the posterior wall of the pharynx and depressing the lever of the StyletScope gently to bend the tip of the ETT anteriorly, we could obtain the view of laryngeal structure and insert the ETT into the glottic opening during visual control. During the intubation procedure, all views of pharyngolaryngeal structures were obtained through the StyletScope.