Questioning Conventional Wisdom

New Technology Applied to Investigating an Old Problem

The study by Mathru et al., uses the increasingly recognized power of magnetic resonance imaging to support recent challenges to the conventional wisdom of the past four decades that posterior displacement of the tongue, resulting in apposition with the posterior pharyngeal wall, is the etiology of upper airway obstruction induced by general anesthesia. The authors conclude that conventional wisdom may be wrong and that obstruction during propofol-induced general anesthesia predominantly occurs at the level of the soft palate, rather than between the tongue and posterior pharynx, and that the marked narrowing can be reversed with continuous positive pressure applied to the nose alone without need of a mask applying gas flow at both the nose and mouth.

In recent decades, little work was carried out subsequent to the original studies to confirm the conventional wisdom in the anesthesia literature or to elucidate the pathophysiology of upper airway obstruction, because an anesthesiologist’s fundamental skills include an armamentarium of maneuvers and devices that, one way or another, eliminate the “problem” and establish “an airway.” Hence the presence of effective solutions reduced the interest of investigators and funding agencies to devote resources to the pathophysiology of the upper airway obstruction in general anesthesia.

During the last 5 yr, however, an appreciation has developed that disorders of sleep associated with upper airway obstruction could pose a substantial risk to the physiologic and economic health of substantial portions of the adult male and female populations. This is because, of a 50% association of obstructive sleep apnea with hypertension, much weaker associations with both right and left heart dysfunction, and diminished work productivity, in addition to an increased incidence of job-related and motor vehicle accidents resulting from hypersonomelence. In these populations of children and adults who experience upper airway obstruction during sleep, many of the resulting disturbances can be abrogated by “establishing an airway” by any means, including tracheostomy, uvulopalatoplasty, or continuous positive airway pressure. The size of the populations at risk and the national economic implications have produced a resurgence of interest in understanding at precisely what site(s) in the upper airway the obstruction occurs, what anatomic or neuromuscular conditions contribute to the obstruction, and what simple, practical, and economic interventions can be used to prevent the upper airway obstruction during sleep.

As others have, Mathru et al. recognized that the clinical and neuromuscular events associated with upper airway obstruction during sleep and during general anesthesia have too many factors in common to be dismissed as unrelated. Importantly, both states exhibit diminished phasic and tonic activity of the upper airway muscles controlling airway patency, including the genioglossus muscle. The increasing use of agents, which can produce general anesthesia, to induce “sedation” in nonoperating room sites, at times without the presence of persons skilled in airway management, further heightens the importance of this issue. The imaging techniques including conventional x-ray, computed tomography, acoustical reflectance, and magnetic resonance imaging, which have been applied to define anatomic sites of obstruction during sleep and other conditions, can be logically applied to the airway during anesthesia. Similarly, it is hoped that correlational physiologic studies of anatomic site-specific electromyographic and mechanical characteristics of the upper airway (e.g., neural activation, compliance, resistance, and critical closing pressure), which are being applied to define dynamic characteristics of the upper airway during awake and sleep states, will be applied to the airway during anesthesia. Mathru et al. have provided an important stimulus in that process. They have defined the location of the reversible anterior-posterior obstruction as between the soft palate and posterior pharyngeal wall as is found in sleep, although they are not able to separately define differences in inspiratory versus expiratory events. In addition, as

Accepted for publication December 6, 1995.

in obstructive sleep apnea, the increase in upper airway volume with positive airway pressure is substantially related to lateral opening of the collapsed airway. Because the distance between the posterior tongue and posterior pharyngeal wall did not change after propofol administration, they suggest that posterior displacement of the tongue does not occur, and the diminished genioglossus activity presumptively present with propofol is not related to the airway obstruction.

However, three factors should be considered before accepting this premise: (1) recent studies suggesting that electrical stimulation of the genioglossus during inspiration can alleviate obstructive sleep apnea in humans; (2) the reasonable presumption that, because the tongue is mechanically contiguous with the soft palate, it will contribute to the transmural forces determining airway collapse or patency at the level of the soft palate; and (3) the experience of many that it is dangerous to disregard conventional wisdom. All of these lead one to consider whether the tongue still plays an important role but not in the way we previously thought. The study of Mathru et al. does not exclude the tongue as substantially contributing to airway obstruction. What it defines is that the anatomic level at which obstruction occurs with propofol is not consistent with the conventional wisdom of anesthesiologists but rather with more recent studies during general anesthesia or sleep.

Why is this relevant to our clinical practice? Where and how the upper airway size diminishes to produce obstruction could affect an optimal approach to reestablishing the airway with minimal intervention and minimal risks of airway trauma or gastric distention and possible regurgitation. For example, it is unclear whether nasal positive pressure alone might be superior to combined oral and nasal mask application of positive pressure in reexpanding the airway at the level of the soft palate. From a mechanical analysis, it can be argued that applying positive pressure from the nose only will push the soft palate forward, while a simultaneous and equal force applied from the nose and the mouth should result in a net movement of the soft palate and hence be less efficacious. If the dominant factor is lateral expansion of the pharyngeal volume, delivery of a gas flow by any means sufficient to create a pressure from either or both orifices should be efficacious. Precisely how our present oral and nasal airways/stents function to maintain patency of the upper airway during general anesthesia may deserve further thought.

The authors are to be congratulated for obtaining the time and local institutional support to challenge conventional “wisdom.” They force us to consider whether other anesthetic agents produce the same effects and whether optimal strategies to relieve upper airway obstruction with general anesthesia during induction, maintenance, and emergence remain to be defined. Mathru et al. have validated the need for further studies.

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References