

In Reply:

Although it is possible to produce or induce differential inspiratory and expiratory obstruction at the level of the soft palate, this was not relevant to the circumstances of our study. Our purpose was to define evolution of vulnerability to upper airway collapse along the pathway from wakefulness through conscious sedation and unconscious sedation to anesthesia. Our methods are standard tools for determining upper airway collapsibility. The pharyngeal critical pressure, which is associated with occlusion (inspiratory and expiratory—assessed over a sequence of five consecutive inspiratory efforts), quantifies this.¹ The intramuscular genioglossus electromyogram not only provides direct information regarding activation of the major upper airway dilator muscle but also reflects activation of the other upper airway muscles.² It is evident from our findings that this evolution is an alinear process with a disproportionate increase in collapsibility occurring proximate to loss of consciousness and coincident with a precipitate decrease in the upper airway muscle activation in response to application of negative pressure to the upper airway.³ These findings hold independent of the precise site of upper airway obstruction, presence of a catheter in the upper airway, or whether or not the mouth is taped.

Nevertheless, the velopharynx is the most common primary site of upper airway obstruction during both anesthesia^{4,5} and sleep;^{6,7} hence, Dr. Kempen is on firm ground with his suggestion that the soft palate might be important! Indeed, as has been our previous experience in anesthetized subjects with and without sleep apnea,^{5,8–10} the primary site of collapse was velopharyngeal in a substantial majority of subjects during our recent study. Seven of our subjects collapsed at this site. Retrolingual collapse was observed in two (subjects 2 and 5). As Dr. Kempen surmises, the site of collapse was determined manometrically according to methods previously described.⁵

Commonly, it is velopharyngeal obstruction that causes hypopneas and accompanying arousals during sleep. Similarly, velopharyngeal collapse is a common site of obstruction during anesthesia and postanesthetic recovery, accounting for the usefulness of the nasopharyngeal airway in these situations. Unlike infants or other mammals, adults are not obligate nasal breathers, so that oral breathing represents an alternative route when the velopharynx is occluded. However, as Dr. Kempen points out in citing Drummond,¹¹ recourse to oral breathing during anesthesia may well indicate impending airway difficulty, regardless of phase of respiration.

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Venous Air Embolism during Total Laparoscopic Hysterectomy

To the Editor:

We read with great interest the article by Kim *et al.*¹ in the July 2009 issue of *ANESTHESIOLOGY*. In this study, the authors warn us of the high frequency of venous gas embolism that can occur during laparoscopic hysterectomy as opposed to total abdominal hysterectomy. Their study calls for several comments.

The authors found approximately 25% of patent foramen ovale (PFO) among their patients, which is consistent with what we know from autopsic series.² However, the detection of a PFO could have been enhanced by an end-inspiratory occlusion maneuver or by the application of positive end-expiratory pressure during transesophageal echocardiography.^{3–5}

No indication on the filling of the left heart by bubbles, especially for patients presenting with a PFO, was reported. This would have informed us of the risk of systemic air embolism, which is ultimately the most daunting complication.