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Airway Collapse or Closure *via* the Soft Palate as Mechanism of Obstruction in Sedated Patients?

To the Editor:

The article by Hillman *et al.*, demonstrating airway collapsibility during incrementally increasing propofol sedation-anesthesia as an inferred mechanism of respiratory impairment (measured *via* a nasal mask) relied heavily on a technique using nonphysiologic negative airway pressure to induce "collapse."[1] This method of collapse was strangely documented also in awake patients subjected to sudden high negative pressures (undisclosed) and also occurring abruptly at lower negative pressures just before unconsciousness. Genioglossus electromyogram activity was recorded as a benchmark for muscular activity combating airway collapse and was yet found to increase significantly in a number of patients before collapse at lower pressures under these conditions. Nine subjects were recruited irrespective of "vulnerability to upper airway collapse," and all mouths were taped shut, with pressure measurements only in esophagus and external airway. Negative airway pressures were induced as expiration started and returned to continuous positive airway pressure on a "posttest" inspiratory effort, with airflow cessation deemed the determinant of "airway collapse."

I would suggest that soft palate obstruction be considered as impairing expiration (and airflow measurements) here for the following reasons:

1. It is readily possible to prohibit airflow through the nose even during maximal ventilation attempts by active occlusion of the nasopharyx at the soft palate to allow suction (drinking through a straw) or positive pressure (blowing a balloon).
2. Benumoff's text agrees that reduced genioglossus electromyogram activity of the genioglossus muscles may cause pharyngeal collapse but convincingly warns further that this is controversial and "the site of airway obstruction occurs either at the level of the palate or . . . the hypopharynx," that is, *via* the epiglottis in the latter instance, rather than *via* the tongue.[2]
3. Benumoff's text singularly states "Some 20% of patients occlude the nasopharynx with the soft palate during exhalation, when the muscles are relaxed."[3] Thus, opening of the mouth becomes imperative during mask ventilation to preclude breath "stacking," autopeep, impaired ventilation, or gastric insufflation after stacking—something relatively unknown to even many experienced anesthetists, in my experience, and thus worth mention here, as 20% is quite common.

Sudden introduction of significant negative pressure during the early phase of high flow exhalation past the soft palate may greatly facilitate closure of the airway here, rather than collapse *per se*, as laminar flow and venturi effects approximate tissues. Distention may have actually occurred in the oropharynx after the palate closure occurred and exhalation progressed. Negative esophageal pressure fluctuations against the closed palate valve could still indicate "collapse" under the negative applied nasopharyngeal airway pressures holding the valve closed, until a posttest inspiratory effort (near end-inspiratory volumes with continuous positive airway pressure!) reopens the airway with the aid of reintroduced continuous positive airway pressure in this study. It is interesting that only two subjects exhibited critical closing airway pressures more than 0 Torr (+1 to +3 Torr), pressures which would have eliminated external closure pressure.

I would be interested to know whether measurement of the oropharynx *versus* nasopharyngeal pressures ever occurred in this study? This might well provide clarity on this issue, especially as a transducer was passed into the esophagus, and a thin, multiple site transducer could similarly monitor these sites of interest. However, multiple tubes passing the soft palate might also cause impairment of this "one-way valve." Pressure measurement within an oral lumen (created *via* insertion of an endoscopic bite block) with the taped shut mouth and transtracheal catheter pressure monitoring may prove more adequate in avoiding artifacts, providing documentation of this soft palate valve, often implicated in obstructive sleep apnea and object of treatment *via* uvulopalatopharyngeal resections, while allowing separate site measurements in this study. Taping the mouth shut may itself be a significant alteration in airway dynamics in sedated patients: "I find that a useful feature of impending airway difficulty is the 'poof' sign that comes from expiration through a lax mouth, indicating diversion from the nasopharyngeal route, which is normal in conscious subjects."[4]

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References

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