Muscle Relaxation and Lower Airway Characteristics May Affect Tidal Volume during Pressure-controlled Ventilation

To the Editor:

With great interest, we read the article by Sato *et al.*,[1] which reported the effect of sleep-disordered breathing on tidal volume with mask ventilation during anesthetic induction.

Additional details might better clarify this study and affect the interpretation of the results.

The exact location of the intravenous catheter (upper or lower extremities), which would affect the onset of rocuronium after injection, should be mentioned. Calibration of TOF-Watch is recommended before neuromuscular function monitoring during clinical research[2]; however, it was not adopted in this study. Although statistically insignificant, the uncalibrated train-of-four ratio was higher in the sleep-disordered breathing group. More information about the muscle paralysis in each group should have been provided.

The investigation used a ventilator with pressure-controlled ventilation (peak inspiratory pressure, 15 cm H_2O). The resistance and compliance were not reported in this article, which could be important, especially in obese subjects. Both the airway and lungs participate in the process of ventilation, not only the pharyngeal airways. The details of lower respiratory airway and lungs are important to interpret the results. Pressure-controlled rather than volume-controlled ventilation was used. The pressure needed for each subject might vary, and could not be preset individually, especially for obese subjects. If volume-controlled ventilation was chosen, however, the tidal volume could be preset conveniently according to ideal body weight. The inspired volume was the specific aim of mask ventilation. Perhaps the peak inspiratory pressure during volume-controlled ventilation could be a better parameter to describe difficult mask ventilation. And the amount of increased peak pressure to achieve the preset tidal volume among patients with sleep-disordered breathing could be explained by a narrowed pharyngeal airway.

To understand better the dynamic changes of ventilation during mask ventilation, and difficult one-hand mask ventilation in patients with obesity and severe sleep-disordered breathing, particularly when expiratory flow limitation occurs during mask ventilation, additional details would be useful.

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Competing Interests

The authors declare no competing interests.

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Rocuronium Onset May Affect Mask Ventilation

To the Editor:

With great interest, we read the article by Sato *et al.*,[1] which reports the influences of obstructive sleep apnea on mask ventilation during induction of general anesthesia by comparing patients with or without sleep-disordered breathing.In addition to the limitations described in the discussion, we note other issues that may have influenced their findings, and the determination of how much of the difference of mask ventilation was caused by obstructive sleep apnea. Specifically, factors influencing the onset time of rocuronium could affect mask ventilation, and these factors were not clearly described.

First, the exact peripheral intravenous sites of rocuronium administration were not described. The onset time of succinylcholine is closely related to drug transit time from the peripheral to the central circulation,[2] and rocuronium has a similar onset time to succinylcholine.[3] For vecuronium, administration into the pulmonary artery shortened the onset time by approximately 40 s compared with a dorsal vein of the hand.[4] Different intravenous sites of rocuronium may produce different onset times. Therefore, the intravenous sites should be similar in the two groups in order to avoid potential bias.

Second, pressure-controlled ventilation was started 20 s after rocuronium injection,[1] but it was not specified whether that was the time from injection start or finish. There is a 5 s difference between start and finish for a 50-kg patient (1 mg/kg) and 1 ml/s injection (5 ml), while the outcomes of mask ventilation were obtained in the 60 s in the study. Also, it was not specified whether all doses were diluted to equal volume and injected with equal speed.

Third, the manuscript did not report parameters relevant to cardiac output, which can affect rocuronium onset.[5] Cardiac output may be lower in patients with obstructive sleep apnea.[6] Heart rate and mean arterial blood pressure might be

used to speculate on the indirect influence of cardiac output on mask ventilation.

We believe that addressing the above issues could further increase the value of this study.

Competing Interests

The authors declare no competing interests.

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"Loose Lips Are Actually Good"

To the Editor:

We compliment Sato *et al.*[1] on their recent article concerning mask ventilation during the induction of general anesthesia. A prominent feature of this study is the occurrence of *expiratory* flow limitation during positive pressure ventilation in 29% of subjects. This phenomenon is not widely appreciated as a cause of difficult mask ventilation. Sato *et al.*'s results confirm and extend observations that our group has previously reported.[2] Our audit strongly supports a "floppy" soft palate as the site of expiratory obstruction and demonstrates that opening the mouth during exhalation improves ventilation by allowing gas to exit the lungs.

Sato *et al.*'s article is lacking details of how the anesthesia providers managed mask ventilation other than to indicate that no oral or nasal airway was used. The provider was told to "perform his/her best airway opening technique during the anesthesia induction." A leak from the mouth during exhalation may have produced the "partial expiratory flow limitation" waveform pattern denoted by the authors as V2 (fig. 2) based on low expiratory flows and a blunted carbon dioxide trace. The study used an anesthesia mask that covered both the nose and mouth, so it is not possible to identify the source of the limited expiratory flow.

This methodologic issue is important because it has the potential to obviate two of the authors' conclusions: first, that partial expiratory obstruction exists, and second, that switching from one- to two-handed mask ventilation is the key move in normalizing ventilation. We did not observe partial expiratory obstruction in our study; it appeared to be an all-or-none phenomenon. In addition, chin lift and head tilt, maneuvers commonly employed in two-hand ventilation, did not relieve the obstruction. What did work was to allow the mouth to open between positive pressure breaths. An oral airway would provide similar benefit. These issues remain open, however, because we used simple observation rather than quantitative methods to determine the presence or absence of obstruction. We join with Sato *et al.* in calling for more detailed studies of the soft palate and surrounding pharynx in patients with expiratory obstruction.

Competing Interests

The authors declare no competing interests.

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In Reply:

We thank Drs. Zhou and Wang for their thoughtful comments on our study.[1] Certainly, many factors may influence the tidal volume during anesthesia induction. We previously reported that progressive muscle paralysis induced by rocuronium injection did not change the tidal volume during facemask ventilation without airway maneuvers in adult subjects with normal upper airway anatomy.[2] In contrast to Ikeda *et al.*'s study,[2] the tidal volume progressively improved in both non–sleep disordered breathing and sleep disordered breathing groups in Sato *et al.*'s study.[1] We believe there are three major differences between Ikeda *et al.*'s and Sato *et al.*'s study designs: anesthesia depth, initial airway patency, and airway maneuvers by the anesthetists. It is our opinion that anesthesia depth contributes little to time dependence of the tidal volume, given that pharyngeal collapsibility increases only slightly by increasing anesthesia depth with propofol, however, the pharyngeal