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Difficult Mask Ventilation and Muscle Paralysis

To the Editor:
We read with great interest the study by Ikeda et al.1 and the accompanying editorial.2 It is now increasingly recognized that muscle relaxants are beneficial in overcoming difficult mask ventilation in adults.2–4 However, both current papers are lacking a sufficient discussion of the reasons why muscle relaxation improves difficult mask ventilation. This can primarily be deduced from recent pediatric evidence where functional airway obstructions are the main reason for difficult mask ventilation.

Difficult mask ventilation in otherwise normal children is exceptionally rare and usually caused by anatomical/mechanical or functional airway obstructions.5 Functional airway obstructions (laryngospasm, insufficient depth of anesthesia, opioid-induced muscle rigidity with glottic closure, and bronchospasm) are common in children;6 result in significant morbidity; and require clear concepts and algorithms.7 Early muscle relaxation or even “pre-ventilation” muscle paralysis will overcome all functional airway problems with the exception of severe bronchospasm for which systemic epinephrine should be immediately available.3 This approach will also allow early and less traumatic direct laryngoscopy and tracheal intubation, if required urgently, without provoking coughing and straining or regurgitation and vomiting.

Amazingly, although muscle paralysis has been shown to improve mask ventilation in adults and is increasingly becoming a key role in the difficult mask ventilation in children with normal airways,9 none of current difficult airway algorithms in adults consider functional airway obstructions. However, this view is shifting in adults too, as the recently published NAP4 report recommends muscle paralysis prior to proceeding with an invasive (surgical) airway in the “cannot intubate - cannot ventilate” scenario or when waking the patient up is not an option.10†

Difficult mask ventilation due to functional airway obstruction with increasing hypoxemia requires muscle paralysis. “Cross the Rubicon fast” in patients with a normal airway.

Thomas Engelhardt, M.D., Ph.D.,* Markus Weiss, M.D.
*Royal Aberdeen Children’s Hospital, Aberdeen, United Kingdom. t.engelhardt@nhls.net

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In Reply:
We appreciate the comments of Xue et al., who highlight important aspects of our study design,1 which likely influenced the results. We sought to establish a patent, yet less than fully dilated upper airway, in order to assess whether muscle relaxants dilate or narrow the pharyngeal airways of anesthetized subjects. Had the initial airway been fully optimized by airway maneuvers, we might have failed to observe succinylcholine-induced pharyngeal airway dilation; therefore, we chose to maintain a neutral head and mandible position in anesthetized normal subjects. Furthermore, the tightly fitted facemask and mouthpiece may have further narrowed the airway.2 Although the experimental settings were different from our usual clinical practice of airway management, we aimed to test the research hypothesis successfully while assuring patient safety. Our experimental design limits application of these results to patients with obstructive sleep apnea, an independent risk factor for impossible mask ventilation.3 In the future, direct assessments of behavior of the whole upper airway, including both pharyngeal and laryngeal regions, in this patient population will address the important questions raised by Xue et al.
We thank Dr. Priebe for his insightful comments. We are in agreement that the safety of airway management for the vast majority of patients has become nearly perfect, due to the high prevalence of sufficiently favorable airways, modern airway devices, and our well-honed procedural and decision-making skills. This is a blessing, but does curse us with the challenge of studying this rare but high-stakes problem, eradication of which still eludes us despite decades of research and advances.3–6

While not the main point of our editorial,7 Priebe takes strong issue with our practice of ventilation before paralysis in selected intermediate-risk patients. Our approach and rationale are not unique; it was formally employed in the study protocol that Priebe cites—“To reduce the duration of apnea, succinylcholine (1 mg/kg) was given when ventilation difficulty was graded III or IV.”5 Changing course in response to unexpected challenges during attempts to ventilate and administering an appropriate dose of succinylcholine (we choose 0.6 mg/kg),8 with or without the insertion of a supraglottic airway device, most often improve or do not worsen ventilation. This is not surprising and is recommended based on newer, more granular evidence.6 Additionally, timely muscle relaxation shortens the duration of (or eliminates) the struggle to ventilate before advancing to definitive management interventions (intubation). To be clear, we too have abandoned “the insistence on effective facemask ventilation before administering muscle relaxant,” but we have not substituted adoption of the earliest possible administration of neuromuscular blockade in routine practice for every patient. We agree that neuromuscular blockers are often part of a solution to some airway difficulties. However, we consider the choice of a specific muscle relaxant to be an important decision point when the planned choice (non-depolarizer) is either confirmed or changed in response to what is learned during an attempt to mask ventilate. This is reflected in the research protocol arm of Amathieu et al.5 So, why not administer succinylcholine to all patients, or to all those with three or more risk factors? While we have colleagues who use succinylcholine very liberally, even routinely, we think that its use is often unnecessary and exposes patients to risks unique to that drug. Given the unacceptable false-positive predictive rate around difficult mask ventilation, we disagree with routine succinylcholine administration to all patients with three or more difficult mask ventilation risk factors. Instead, we prefer the approach of administering succinylcholine when indicated.

Priebe contends that the duration of the effect of succinylcholine prevents spontaneous ventilation before the onset of significant hypoxemia. His supporting quote from theoretical work9 omits key underlying assumptions—a succinylcholine dose of 1 mg/kg and complete apnea. Debate over the “wake up” option resurfaced a decade ago.8,10–12 In difficult airway situations (including those following ill-fated administration of long-acting neuromuscular blockade), we rarely see complete inability to exchange gas. Because that gas is oxygen, inadequate ventilation but sufficient oxygenation typically sustains life long enough to resolve the airway crisis, as was observed by Amathieu et al.5 Of 17 patients experiencing difficult mask ventilation and SpO2 less than 80%, all survived without significant complication. How should we respond to other experts’ tenacious contention that the “wake up” option is nonviable in the face of our successful use of it in practice? “Anecdotal” experience is routinely discounted as nonevidence by all but those who own it; yet personal observation, reflection, and judgment play a much greater role in our clinical work and research than we recognize.13 We continue to agree with Kopman’s conclusion that “0.6 mg/kg may be a wise choice under some conditions,”8 at least preserving the option to awaken the patient in an escalating life-threatening difficult airway situation.

We also thank Englehardt and Weiss for their excellent points, particularly the distinction between mechanical and functional obstruction, as occurs not uncommonly in pediatric anesthesia practice. We agree with their assertions and encourage investigation of the variables contributing to functional upper airway obstruction, as well as the decision-making process for timely interventions that include administration of muscle relaxants.

Michael Richardson, M.D., Aya Ikeda, M.D., Shiroh Isono, M.D., Ronald S. Litman, D.O.* *University of Pennsylvania, Philadelphia, Pennsylvania, and The Children’s Hospital of Philadelphia, Philadelphia, Pennsylvania. litmanr@email.chop.edu

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Correspondence

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