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In Reply:

Because the letters by Drs. Abouleish and Cole touch on peer review and the mission of ANESTHESIOLOGY, I will also respond. Dr. Abouleish is familiar with the rigor of peer review at this journal, because he recently served on our Associate Editorial Board. Manuscripts are reviewed by content experts, often including one Editor or Associate Editor, and each review is rated for quality by the Handling Editor. Detailed instructions for reviewers are available on our Web-based system for peer review, and reviewers are asked to specifically rate the following five factors of quality and importance in the manuscript: clinical impact, scientific impact, interest to the specialty, novelty, and definitive interpretation. As Editor-in-Chief, I am responsible for all decisions, whether I handle the manuscript personally or review the recommended decision from the Handling Editor.

The mission of this journal is, "Promoting scientific discovery and knowledge in perioperative, critical care, and pain medicine to advance patient care." Our goal is to provide the highest quality research, rated according to the criteria above, in order to better understand the foundations of our specialty and to affirm or revise practice. I believe that the correspondence concerning the original article¹ has helped clarify its contribution in this regard, and I thank all the authors of the manuscript and these letters for this discussion.

James C. Eisenach, M.D., Editor-in-Chief, ANESTHESIOLOGY, Wake Forest University School of Medicine, Winston-Salem, North Carolina. editor-in-chief@anesthesiology.org

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Understanding the Mechanics of Laryngospasm Is Crucial for Proper Treatment

To the Editor:

In their case scenario, "Perianesthetic management of laryngospasm in children," Orlianguet *et al.*¹ presented a 10-month-old boy who developed an inspiratory stridor after sevoflurane induction, which was initially managed by a jaw thrust and positive pressure ventilation. When the stridor recurred, manual ventilation became difficult with increased resistance to insufflation. Despite a jaw thrust, positive pressure ventilation with $FiO_2 = 1$, and propofol, the obstruction was not relieved and severe hypoxemia (oxygen saturation measured by pulse oximetry, or SpO_2 , = 52%) ensued, requiring the administration of succinylcholine and tracheal intubation.

A basic understanding of the mechanics of laryngospasm is crucial for proper treatment.² In his classic article, Fink described three types of laryngospasm: expiratory stridor, inspiratory stridor, and ball-valve obstruction.³ The stridor is controlled by the intrinsic laryngeal muscles, whereas the ball-valve closure is controlled by both the intrinsic and extrinsic laryngeal muscles. The expiratory stridor occurs as a result of active adduction of the vocal cords. The inspiratory stridor is produced passively as a result of the loss of tone of the abductor muscles. Because the velocity is greater where the passage is most narrow, airway pressure at the subglottic area becomes less than atmospheric during inspiratory efforts, and the passage of gases through the glottis generates a force that approximates the vocal cords together resulting in inspiratory stridor. Positive airway pressure can stent the airway and correct both expiratory and inspiratory stridor.³

In ball-valve obstruction, laryngeal closure occurs at three levels: the true vocal cords, the false cords, and the redundant supraglottic tissue.^{3,4} The approximation of the vocal cords (and false cords) is swiftly followed by contraction of the extrinsic laryngeal muscles, shortening of the thyrohyoid distance resulting in complete closure, and cessation of airflow.^{3,4} In this situation, applying positive pressure can worsen the obstruction,³ as evidenced in the current case. By distending both pyriform fossae, the aryepiglottic folds are pressed more firmly against each other, which reinforces the closure.³ In contrast, the jaw thrust (also referred to as maximum mandibular advancement)⁵ can be effective in correcting ball-valve closure. The forward mandibular movement is transmitted through the geniohyoid muscles to the hyoid bone and the hyoepiglottic ligament. Consequently, the epiglottis and the redundant supraglottic tissue are pulled away from the false cords, and the laryngeal passage is reopened.³ How-

This letter was sent to the author of the original article, who felt that a reply was not necessary.—James C. Eisenach, M.D., Editor-in-Chief.

Laryngospasm

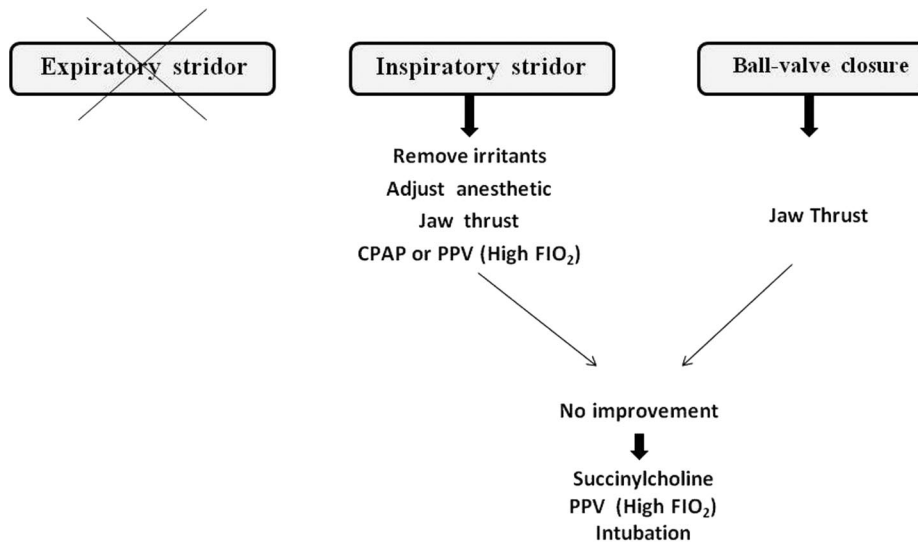


Fig. 1. Algorithm for management of laryngospasm. Ball-valve obstruction is diagnosed by cessation of gas entry in spite of active inspiratory efforts. Succinylcholine is administered intravenously, but can be administered intramuscularly in case an intravenous access is not available. CPAP = continuous positive airway pressure; F_{IO_2} = fraction of inspired oxygen; PPV = positive pressure ventilation.

ever, it is frequently necessary, as in the case presented by Orliaguet *et al.*, to administer succinylcholine to relieve this type of severe obstruction.

It is interesting that the expiratory stridor, which was common in the days of ether anesthesia and was regarded as “a vocal protest by the patient against inadequate anesthesia,” has virtually disappeared in modern anesthesia practice. In our recent quality-improvement study of laryngospasm, expiratory stridor did not occur in any patient. This is probably related to the use of sevoflurane yielding faster induction because of its low blood/gas partition coefficient.

We propose a simple algorithm (fig. 1) for the management of laryngospasm, which can be easily remembered and utilized by clinicians. Based on the mechanics of laryngospasm, this algorithm addresses both inspiratory stridor and ball-valve obstruction, but ignores the expiratory stridor because it is no longer observed in modern anesthesia practice. Positive pressure, which is effective in the management of inspiratory stridor, is avoided in ball-valve obstruction because it can worsen the obstruction. If a jaw thrust fails to correct ball-valve obstruction, succinylcholine is administered, followed by positive pressure ventilation and tracheal intubation.

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Ebenezer Hopkins Frost

To the Editor:

Ebenezer (Eben) Hopkins Frost (1824–1866) was the first patient to receive ether from William T. G. Morton (1819–1868) in the evening of September 30, 1846, in Morton’s dental rooms at 19 Tremont Row, Boston, Massachusetts. While researching the earliest known photographs of anesthesia,¹ the author found that there was little information, some of it contradictory, on Frost. There were no publications on Frost, although there was significantly more information available on Edward Gilbert Abbott (1825–1855), who received ether from Morton on October 16, 1846, at Massachusetts General Hospital, Boston, Massachusetts.²

This biography of Frost will review the recorded information on Frost, and correct some of the errors in the details of his life and career. Frost’s encounters with Morton are also described.