



ELSEVIER

Contents lists available at ScienceDirect

Air Medical Journal

journal homepage: <http://www.airmedicaljournal.com/>

Case Report

Troubleshooting Hypoxemia After Placement of an Extraglottic Airway

J. Britton Hopkins, MD^{1,2}, Matthew A. Roginski, MD^{3,4}, Darren A. Braude, MD^{5,6,7}, Andrew D. Cathers, MD^{1,2}, Trevor Johnson, RN¹, Michael T. Steuerwald, MD^{1,2,*}

¹ UW Med Flight, Madison, WI

² Department of Emergency Medicine, University of Wisconsin–Madison, Madison, WI

³ DHART, Lebanon, NH

⁴ Department of Medicine, Geisel School of Medicine, Hanover, NH

⁵ Lifeguard Air Emergency Services, Albuquerque, NM

⁶ Department of Emergency Medicine, University of New Mexico, Albuquerque, NM

⁷ Department of Anesthesiology, University of New Mexico, Albuquerque, NM



A B S T R A C T

The case presented here highlights the feasibility of using an extraglottic airway device as a conduit for delivering high levels of lifesaving positive end expiratory pressure (PEEP), as well as other means of combating recalcitrant hypoxia. The case also highlights the merit of an approach to the hypoxic patient with an in-situ extraglottic airway device based not only on deciding if the device is functioning to maintain a patent airway, but also, simultaneously considering the patient's physiology. A 71 year old male suffered an out-of-hospital cardiac arrest. Part of his resuscitation included placement of a dual-balloon extraglottic airway device by EMS. He was hypoxic, but the device seemed to be providing for a patent airway without an air leak. There was also a favorable end-tidal carbon dioxide waveform. The flight team chose to leave the device in place. PEEP was up-titrated to 17 cmH₂O without issue. Sigh breaths, as well as breath holds, were also able to be delivered. The patient's hypoxia improved over the course of the patient's transport, and he ultimately did well.

© 2019 Air Medical Journal Associates. Published by Elsevier Inc. All rights reserved.

Extraglottic airway devices (EGDs) are becoming much more common in prehospital care such that critical care transport teams are much more likely to encounter or place such a device than they were 10 years ago.^{1,2} These devices may be placed primarily, either in the setting of cardiac arrest or rapid sequence airway,³ or secondarily after 1 or more missed attempts at endotracheal intubation. These changes have been made in light of a growing pool of research that has shown an attempt to place an EGD is both faster and more reliable than an attempt at endotracheal intubation in the prehospital setting.^{4,5}

Although EGDs have been proven effective at providing adequate oxygenation and ventilation in many circumstances, they can fail for a variety of reasons. In the event of failure, the provider is forced to make a rapid and oftentimes difficult decision regarding the patient's

airway management. Airway experts have varying opinions on the appropriate next steps to establish a definitive airway in this scenario.

We report a case in which an EGD, specifically a King LTS-D (Ambu, Ballerup, Denmark), had been placed before flight team arrival; however, the patient remained hypoxic, necessitating rapid troubleshooting.

Case Report

Rural community emergency medical services and police responded to the call of a 71-year-old gentleman with a history of atrial fibrillation, congestive heart failure, and coronary artery disease who collapsed at his home. Police arrived within 2 to 3 minutes and found the patient to be in cardiac arrest; they immediately started cardiopulmonary resuscitation and delivered 1 shock with an automated external defibrillator, which resulted in return of spontaneous circulation. Upon emergency medical services arrival, the patient was found to be in a perfusing rhythm but still unconscious; thus, he

* Address for correspondence: Michael T. Steuerwald, MD, Department of Emergency Medicine, University of Wisconsin–Madison, 800 University Bay Drive, Suite 310, Mail Code 9123, Madison, WI 53705

E-mail address: mtsteuer@medicine.wisc.edu (M.T. Steuerwald).

underwent rapid sequence airway with a King LTS-D.³ His electrocardiogram was pertinent for a ventricular-paced rhythm with nonspecific ischemic changes. At this point, a hospital-based, physician-staffed helicopter emergency medical service team was contacted for transport.

Upon arrival of the air medical crew, the patient's airway, breathing, and hemodynamics were rapidly assessed. The King LTS-D was in a suitable position with ventilations being provided by a bag valve self-inflating device connected to high-flow oxygen. He was noted to have favorable hemodynamics with a heart rate of 70 to 80 beats/min and systolic blood pressure of approximately 140 mm Hg; however, the patient's oxygen saturations ranged from 70% to 85%. On examination, bilateral chest rise, equal respirations, normal end-tidal capnography waveform without evidence of bronchospasm or other pathology, and no audible air leak were noted. Pneumothorax was considered but was felt to be less likely; close attention was paid to equal chest rise and fall, as well as airway pressure indicators, throughout the transport as a strategy to evaluate for developing complications. Pulmonary embolism was considered; however, because of stable hemodynamics, no findings of overt right ventricular strain on the electrocardiogram, and other diagnoses felt to be more likely, the pretest probability was thought to be low. Collectively, the findings indicated that the issue was with the patient's physiology rather than the EGD, so the focus became rapidly maximizing interventions to affect the physiology rather than removing the device.

The first intervention was elevation of the head of bed and placement of a positive end-expiratory pressure (PEEP) valve on the bag. Because the PEEP was initially uptitrated, the patient's oxygen saturation rose to 85% but plateaued there. This degree of hypoxemia was felt to be suboptimal, so exchange for an endotracheal tube (ETT) was considered. However, the patient was noted to have several difficult laryngoscopy and cricothyroidotomy predictors, including a short neck with excessive amount of adipose tissue and the aforementioned hypoxemia. Additionally, the duration of time that the King Airway had been in place could have led to significant airway edema. Overall, there was great concern that if the EGD was removed, intubation would not be possible before the patient experienced critical hypoxemia. An emergent cricothyrotomy without prior intubation attempt was also considered but was not pursued.

Given these factors, the decision was made to initiate air transport with the King Airway left in place. The strategy was to continue to uptitrate PEEP over the course of the flight, something the flight crew felt could be done via the EGD. During the 25-minute flight, the EGD was attached to a transport ventilator, and additional PEEP was applied in a stepwise fashion until 17 cm H₂O was reached. Ventilator settings were otherwise standard assist control (volume) with a tidal volume based on 6 to 8 mL/kg ideal body weight. Peak pressures were approximately 40 cm H₂O. Sigh breaths, as well as occasional breath holds, were also delivered. Upon arrival to the tertiary care center emergency department, the patient's saturations had improved to 95% to 100%. The patient's EGD was exchanged in the emergency department using an intubating bronchoscope and an exchange kit, and the patient was admitted to the cardiac intensive care unit. He survived and was discharged from the hospital several weeks later with a meaningful neurologic outcome.

Discussion

This case shows a holistic EGD evaluation and troubleshooting process and demonstrates that an EGD can be an effective conduit for the delivery of high levels of PEEP. In the case of hypoxia after placement of an EGD, exchange for an ETT will be performed in most cases. However, in a patient with a predicted difficult airway, the team must focus on the pathophysiology of the hypoxia and potentially prescribe an appropriate ventilator strategy using the EGD that is already in place. In this case, the team felt that they could oxygenate

and ventilate via the EGD and, thus, felt there was an unfavorable risk/benefit analysis of placing an ETT or performing a cricothyrotomy. Given that the device seemed to be functioning appropriately, attention was turned to treating intrinsic causes of hypoxia within the patient, exactly like what would be done if the patient had an ETT in place.

When assessing hypoxia after an EGD or ETT placement, we propose that failure can be broadly categorized as falling into 1 of 2 categories: 1) equipment or upper airway failure or 2) physiological failure. In this case, the team assessed for adequate placement of the King LTS-D, functioning oxygen supply, and adequate ventilation with end-tidal capnography. After there was no apparent equipment failure present, attention could then be turned to the pathological causes of hypoxia.

The differential diagnosis of hypoxia has 5 broad categories: 1) decreased partial pressure of oxygen being delivered, 2) hypoventilation, 3) alveolar diffusion abnormality, 4) intrapulmonary shunt, and 5) ventilation/perfusion mismatch.^{6,7} The history, physical examination, and adjunctive tests (such as ultrasound) may help narrow the differential in the prehospital environment. Making care more complex is the fact that patients may have more than 1 cause of their hypoxia at 1 time, and providers are tasked with prescribing a treatment plan in an information- and resource-limited environment. This is of paramount importance because a physiologic approach to the treatment of hypoxia is preferred compared with arbitrary titration of PEEP and fraction of inspired oxygen because one must consider the anticipated benefits as well as harms.

PEEP has multiple physiologic effects, but in this case the discussion will focus on the distribution of lung water, increasing the number of alveoli reached by air, and the cardiogenic effects of increased intrathoracic pressure. Changing the lung water distribution when PEEP increases in cases of pulmonary edema and excess lung water, the increased pressure does not push fluid out of the lungs, but rather it helps redistribute the fluid from the alveoli to the perivascular space, which improves gas exchange.^{6,8,9} PEEP also increases the number of alveoli the tidal volume is reaching. Previously atelectatic sections of dependent lung segments (eg, in a supine, positively ventilated patient, with an obese abdomen) are opened with an increase in PEEP, allowing a greater surface area for gas exchange.¹⁰ The provider must keep in mind that high levels of PEEP may also be detrimental to oxygenation because overdistention of apical lung segments may increase dead space ventilation.^{11,12}

Although PEEP and the increase in intrathoracic pressure have multiple cardiovascular effects, they can be broken down into the effects on preload, right ventricular afterload (pulmonary vascular resistance), and left ventricular afterload. Overall, the effect that PEEP has on preload strongly depends on volume status.^{13,14} In a hypovolemic patient, increasing PEEP will cause a decrease in overall cardiac output, but this relationship is not observed in hypervolemic patients because of a concomitant increase in mean systemic filling pressures.¹⁵ Increased lung volumes and PEEP increase right ventricular afterload by compressing the alveolar vessels.^{10,16} The increase in intrathoracic pressure also affects the left ventricular (LV) afterload. Afterload is a combination of both aortic pressures and LV transmural pressures because they both oppose ventricular contraction. By increasing the intrathoracic pressures, the pressure gradient across the LV is decreased, which means the heart does not have to work as hard to contract.

The optimization of PEEP is critical in resuscitation because there are substantial helpful and potentially harmful effects; a balance must be found. In a patient with bilateral lung disease and concern for acute respiratory distress syndrome, increased PEEP helps to recruit atelectatic lungs and improves oxygenation. In cases of acute cardiogenic pulmonary edema, the redistribution of lung water and the improvement of cardiac output with decreased preload and afterload can profoundly improve the patient's condition.

The incorrect use of PEEP can also cause significant harm. For example, increasing PEEP with unilateral pneumonia can make hypoxia worse. PEEP can distend nondiseased segments of lung and compress alveolar capillaries in compliant lung units. This causes shunting of blood to the noncompliant lung units with alveolar infiltrate and no air reaching the alveoli, thus worsening the shunt. Similarly, if PEEP is increased too high with highly compliant lungs and low systemic venous pressures, the hemodynamic consequences can be profound from the decrease in cardiac preload. For these reasons, it is essential that the resuscitator understands the physiology of respiratory failure and the effects of PEEP.

In this patient, the crew systematically evaluated and treated apnea and hypoxia and continued hypoxemic respiratory failure after return of spontaneous circulation with an EGD. They confirmed the airway placement and functionality. Given the cardiac history, acute pulmonary edema and early bilateral lung infiltrates from an infectious or inflammatory process, such as aspiration, were strongly considered, as well as atelectasis from the patient being in a supine position. All of these processes can improve with increased PEEP. The team was able to therapeutically titrate PEEP to improve oxygenation during the initial resuscitation and transport. The EGD, specifically the King LTS-D, provided an adequate conduit for PEEP titration. This case shows that the EGD is a way to deliver lifesaving ventilator management through the optimization of PEEP without an endotracheal tube in a time-, information-, and resource-limited environment. To our knowledge, this is the first report of this level of PEEP being delivered via a King LTS-D in a successful prehospital case setting.

References

1. McMullan J, Gerecht R, Bonomo J, et al. Airway management and out-of-hospital cardiac arrest outcome in the CARES registry. *Resuscitation*. 2014;85:617–622.
2. Guyette FX, Greenwood MJ, Neubecker D, et al. Alternate airways in the prehospital setting (resource document to NAEMSP position statement). *Prehosp Emerg Care*. 2007;11:56–61.
3. Braude D, Richards M. Rapid sequence airway (RSA)—a novel approach to prehospital airway management. *Prehosp Emerg Care*. 2007;11:250–252.
4. Gahan K, Studnek JR, Vandeventer S. King LT-D use by urban basic life support first responders as the primary airway device for out-of-hospital cardiac arrest. *Resuscitation*. 2011;82:1525–1528.
5. Russi CS, Miller L, Hartley MJ. A comparison of the King-LT to endotracheal intubation and combitube in a simulated difficult airway. *Prehosp Emerg Care*. 2008;12:35–41.
6. Vincent JL, Abraham E, Moore FA, Kochanek PM, Fink MP. *Textbook of Critical Care*. 7th ed. Philadelphia, PA: Elsevier; 2017.
7. West JB, Luks AM. *West's Respiratory Physiology: The Essentials*. 10th ed. Philadelphia, PA: Wolters Kluwer; 2016.
8. Malo J, Ali J, Wood LD. How does positive end-expiratory pressure reduce intrapulmonary shunt in canine pulmonary edema? *J Appl Physiol Respir Environ Exerc Physiol*. 1984;57:1002–1010.
9. Paré PD, Warriner B, Baile EM, Hogg JC. Redistribution of pulmonary extravascular water with positive end-expiratory pressure in canine pulmonary edema. *Am Rev Respir Dis*. 1983;127:590–593.
10. Tobin MJ. *Principles and Practice of Mechanical Ventilation*. 3rd ed. New York, NY: McGraw-Hill Companies; 2013.
11. Beydon L, Uttman L, Rawal R, Jonson B. Effects of positive end-expiratory pressure on dead space and its partitions in acute lung injury. *Intensive Care Med*. 2002;28:1239–1245.
12. Gattinoni L, Caironi P, Cressoni M, et al. Lung recruitment in patients with the acute respiratory distress syndrome. *N Engl J Med*. 2006;354:1775–1786.
13. Pinsky MR. Cardiovascular issues in respiratory care. *Chest*. 2005;128:592S–597S.
14. Schreuder JJ, Jansen JR, Versprille A. Hemodynamic effects of PEEP applied as a ramp in normo-, hyper-, and hypovolemia. *J Appl Physiol*. 1985;59:1178–1184.
15. van den Berg PC, Jansen JR, Pinsky MR. Effect of positive pressure on venous return in volume-loaded cardiac surgical patients. *J Appl Physiol*. 2002;92:1223–1231.
16. Kato T, Suda S, Kasai T. Positive airway pressure therapy for heart failure. *World J Cardiol*. 2014;6:1175–1191.