Use of heliox for intraoperative bronchospasm: A case report

Stacey L. Linck, CRNA, MA
St. Louis, Missouri

Bronchospasm is an anesthetic emergency that can lead to disastrous outcomes if treatment is irresolvable. An anesthesia provider must immediately initiate treatment if bronchospasm is suspected in order to avoid negative sequelae. The following is a case report of a 32-year-old man who experienced refractory bronchospasm upon emergence from general anesthesia.

This article discusses the initial treatment attempted at resolving the bronchospasm, as well as the use of heliox in the ultimate resolution of the bronchospasm. Although heliox has been used for years to treat patients with various respiratory complications, it is not currently a common treatment instituted by anesthesia practitioners for the treatment of bronchospasm. Consideration of the use of heliox may provide another option for the treatment of a patient suffering from refractory bronchospasm.

Key words: **bronchospasm, density, emergence, heliox, turbulence.**

Bronchospasm can be a serious event that can lead to critical hypoxemia with ultimate brain damage or death.[1-3] Although there is an increased incidence of bronchospasm in patients with preexisting respiratory conditions, such as asthma or chronic obstructive pulmonary disease, it is essential to remember that severe bronchospasm can occur in any patient regardless of medical history.[1-2] It is imperative that the anesthesia provider recognize the signs and symptoms of bronchospasm. The clinical features of bronchospasm may resemble endobronchial intubation, obstruction of the endotracheal tube secondary to increased secretions or blood, pulmonary aspiration, pneumothorax, a kinked endotracheal tube, or pulmonary edema.[2]

Once bronchospasm has been identified, it is essential for the anesthesia provider to initiate treatment to prevent any adverse complications related to hypoxemia. Current therapies for the management of bronchospasm include, but are not limited to, increasing inspired oxygen concentrations (Fio_2), alterations of ventilatory settings, eliminating suspected causative antigens, deepening the general anesthetic, bronchodilator therapy or other pharmacological therapy to decrease parasympathetic reflexes, or deep extubation.[2] An anesthesia provider must quickly consider alternatives to refractory bronchospasm. This case report presents the use of heliox as another alternative for the management of refractory bronchospasm.

Case summary

A 32-year-old man presented for an emergency appendectomy. A preoperative anesthesia evaluation was completed in the emergency department revealing a 1-pack-per-month smoking history with no other significant medical history. The patient denied any regular medications, allergies, or history of any previous surgery. He was unaware of any familial history of anesthetic complications. Assessment of the airway revealed a class I Mallampati score, with greater than 3 fingerbreadths thyromental distance and adequate mouth opening. Native dentition was intact and he demonstrated full cervical range of motion. Laboratory studies were within normal limits. The patient's height was 61 inches (155 cm), and he weighed 187 pounds (84 kg). Preoperative vital signs were as follows: blood pressure, 140/65 mm Hg; heart rate, 65 beats per minute; tympanic membrane temperature, 37°C; and room air oxygen saturation (Spo_2), 100%. Upon auscultation, breath sounds were clear bilaterally. A physical status II was assigned, and the patient was given midazolam intravenously for preoperative sedation and sodium citrate/citric acid by mouth as he was wheeled to the operating room. Preoperative antibiotics started in the emergency department included ceftriaxone and metronidazole.

Upon arrival in the operating room, standard monitors were applied. The patient was preoxygenated for 5 minutes with 100% Fio_2 per breathing circuit face mask. A rapid sequence induction with cricoid pressure was performed using fentanyl, lidocaine, sodium thiopental, and succinylcholine. An atraumatic laryngoscopy was performed revealing grade I visualization of the vocal cords. A 7.5-mm cuffed endotracheal tube was inserted in the trachea on the first attempt, and the tube was secured at 21 cm at the lip after confirmation of tube placement via detection of end-tidal carbon dioxide and auscultation of clear bilateral breath sounds.

Respirations were controlled with mechanical ventilation, and general anesthesia was maintained with 100% Fi_{O_2} and isoflurane 1% to 1.5% end-tidal concentration. Vecuronium, 4 mg, was given during the procedure for muscle relaxation with an uneventful anesthetic. Approximately 30 minutes before the end of surgery, nitrous oxide was initiated at a 50% inspired concentration. Upon closure of the skin, neuromuscular blockade was antagonized with neostigmine, 3 mg, and glycopyrolate, 0.5 mg, and the response to double-burst stimulation exhibited 2 strong contractions without fade. Nitrous oxide and isoflurane were terminated and 100% Fi_{O_2} was reinitiated. As end-tidal isoflurane concentrations decreased to approximately 0.24%, the patient began to cough. Oxygen saturation quickly dropped to 60%, and circumoral cyanosis was present. Tube placement was confirmed with auscultation of the lungs revealing severely diminished bilateral breath sounds. Positive pressure manual ventilation delivered tidal volumes of 200 mL with peak airway pressures greater than 50 cm H_2O pressure. Severe bronchospasm was suspected. Twelve puffs of albuterol (90 µg/puff) were given via the endotracheal tube. Isoflurane was reinstituted, lidocaine, 50 mg, was given intravenously, and subcutaneous epinephrine, 100 µg, was administered. Hemoglobin saturation with oxygen increased to 94% within approximately 1 minute after deepening of the volatile agent and peak airway pressures decreased to 39 cm H_2O pressure. A second emergence was attempted, and once again bronchospasm ensued after the patient coughed. Saturation decreased briefly to 60%.

Bronchospasm resolved with the deepening of the volatile anesthetic and administration of albuterol per endotracheal tube and subcutaneous epinephrine. Saturation improved to 94% as isoflurane end-tidal concentration increased to 0.48%. The third emergence was unsuccessful, resulting in a saturation of 70% accompanying bronchospasm. Again, isoflurane concentration was increased, 12 puffs of albuterol were administered via the endotracheal tube, and 100 µg subcutaneous epinephrine was given, with saturation improving to 95%.

A request was made to the Respiratory Therapy Department to obtain a heliox tank. Isoflurane was discontinued, and a 70%/30% helium-oxygen mixture was administered via a Jackson-Reese circuit with assisted ventilation. Tidal volumes rapidly increased from 200 mL to 400 mL. The patient initiated a regular respiratory pattern with a rate of 24 breaths per minute. Saturation was maintained between 94% and 97%. The patient opened his eyes and began to cough, and the trachea was extubated after endotracheal cuff deflation. The patient maintained his airway and 100% Fi_{O_2} was delivered via simple face mask. Auscultated breath sounds revealed mild expiratory wheezes. Saturation remained at 98%. The patient was taken to the recovery room where an albuterol nebulizer was immediately instituted. The patient experienced no further respiratory complications and was discharged home on postoperative day 2 with no respiratory sequelae.

Discussion

Bronchospasm is a relatively infrequent occurrence during anesthesia. According to Olsson,[3] who in 1986 completed a restrospective study of 136,929 surgical patients, the incidence of bronchospasm during anesthesia is 1.6 per 1,000 anesthetics. Bronchospasm is a result of extreme contraction of the bronchial smooth muscle that leads to narrowing and increased resistance of the airways. The autonomic nervous system is responsible for controlling the muscle tone by balancing the levels of 3′5′-cyclic adenosine monophospate (cAMP) and 3′5′-cyclic guanine monophosphate (cGMP). The sympathetic nervous system is responsible for increasing cAMP levels through epinephrine-induced β_2 adrenergic receptor stimulation resulting in bronchodilation.[2] The parasympathetic nervous system, through stimulation of muscarinic receptors or vagus nerve activity, is responsible for bronchoconstriction. Some biochemical mediators (prostaglandins, leukotrienes), pharmacological therapies (cholinergic agonists, histaminics), and other noxious stimuli (mechanical stimulation, secretions) also are probable antagonists of bronchial lumen patency. Bronchospasm can be difficult to detect under general anesthesia,[2] and it may manifest in several ways. Coughing, increased peak airway pressures without change in plateau pressures, or a decreased phase II slope on the capnogram are all possible signs. Wheezing is a common sign; however, airway resistance may be so great that wheezing may not occur indicating extremely low gas exchange.[2]

Airway resistance increases dramatically as the bronchial lumen narrows, as derived from Poiseuille's law: $R = 8\eta l/\pi r^4$ (R indicates resistance, η indicates viscosity, l indicates length of tube, and r indicates radius of the tube).[4] Therefore, bronchoconstriction that reduces the airway diameter by half is capable of producing a 16-fold increase in resistance. Turbulent flow in the airways also contributes to the increased airway resistance subsequent to bronchospasm. Turbulent flow is indicated by a Reynold's number (Re) greater

than 2000.[4] Reynold's number is defined as: $Re=2rvd/\eta$ (d indicates density, v indicates average velocity, r indicates radius, and η indicates viscosity).[4] Thus, decreasing the density of a gas lowers Reynold's number achieving a more laminar flow in the airway.

Helium, a biologically inert gas, has a density of one eighth that of nitrogen and oxygen.[5] The density of air is approximately 1.20 kg/m^3. The density of 100% oxygen is approximately 1.33 kg/m^3. The density of a nitrous/oxygen mixture is approximately 1.41 kg/m^3. The density of a 70%/30% heliox mixture is approximately 0.52 kg/m^3. Heliox, the combination of helium and oxygen, therefore has a reduced density compared to air.[6] An 80%/20% helium-oxygen mixture has a density one third that of air-oxygen mixture.[7] This significantly reduced density is capable of decreasing airway resistance by promoting laminar flow. The use of helium-oxygen mixtures for various respiratory complications began in 1934.[5] To date, heliox is not routinely used as primary treatment for any respiratory ailment perhaps because of the lack of research studies confirming its effectiveness. Throughout the years since its discovery, it has been used to aid in treating conditions such as asthma, chronic obstructive pulmonary disease, and upper airway obstruction.[5] Studies are lacking for the use of heliox in bronchospasm experienced during anesthesia.

Heliox has been shown to be beneficial in the treatment of upper airway obstruction by reducing airway resistance.[8-9] In a study by Kemper et al,[8] postextubation stridor scores were significantly lowered with the use of heliox therapy compared to the use of standard oxygen-enriched air. In this double-blind, randomized, controlled crossover trial, helium was found be safe and effective for the treatment of postextubation respiratory distress.[8] From this study it appears that heliox could be considered before reintubation or other airway manipulation to improve ventilation in certain situations of unresolved partial upper airway obstruction. Another study by Lu et al[9] also demonstrated the successful use of heliox in treating upper airway obstruction. Simulating a case study of a right pneumonectomy, they occluded the right mainstem bronchi of 6 dogs. After the right mainstem bronchus was occluded, they proceeded to partially occlude the left mainstem and initiate heliox therapy in incremental doses. The peak airway pressure and $PaCO_2$ were both reduced with the addition of heliox therapy thus improving ventilation. Heliox may be beneficial for preventing prolonged mechanical ventilation or respiratory failure associated with negative outcomes.

Asthma, a condition marked by recurrent spasmodic constriction of the bronchi, is a disease that can pose significant challenge to the anesthesia provider. The incidence of bronchospasm and other perioperative respiratory events is increased in patients with this condition.[10] A serious bronchospasm can present in a patient with a history of asthma at any time. If severe, sometimes routine pharmacological therapy is insufficient and other treatment is crucial to avoid a negative outcome.[10] Heliox has been shown to be therapeutically effective and beneficial in treating asthmatics in several anecdotal case studies.[5,10-13] In a restrospective case-match control design, Schaeffer et al[11] compared oxygenation in 22 mechanically ventilated patients suffering from status asthmaticus. Eleven patients who received heliox therapy after intitation of mechanical ventilation were compared with 11 patients in a control group that did not receive heliox. They found that a brief period of heliox administration during mechanical ventilation in status asthmaticus significantly improved oxygenation and decreased the alveolar-arterial gradient. Current literature clearly indicates the need for prospective, reliable clinical studies regarding the use of heliox with asthmatics.[5,12-13] Review of the literature demonstrates that heliox can be beneficial in reducing peak inspiratory pressures and decreasing pulsus paradoxus (which is suggested to indicate decreased work of breathing). Heliox also may be capable of promoting the delivery of an inhaled β-agonist to the distal airways thus facilitating the desired action.[5] Controversy exists in the interpretation of these studies, and further research is needed to confirm the efficacy of heliox therapy in asthma.

There is no documented literature about the use of heliox for the treatment of bronchospasm under general anesthesia. In a case report by Polaner,[14] heliox was used in conjunction with a laryngeal mask airway for a child with compression of his carina and left mainstem bronchus due to an anterior mediastinal mass. In this specific case, heliox administration preoperatively increased the patient's SaO_2 from 76% to 96%.[14] Heliox was continued intraoperatively and contributed to the successful outcome of the patient.[14] In the current literature, heliox appears to be safe and effective in appropriately selected patients. Research may be indicated for the use of heliox therapy in general anesthesia.

In the present case study, the use of heliox allowed emergence from general anesthesia with tidal volumes adequate for extubation. The bronchospasm may have been caused by the reversal agent, by the mechanical

stimulus of the endotracheal tube, or by a number of other biochemical mediators or triggers. Clinically, bronchospasm resolved following removal of the endotracheal tube. Theoretically, the heliox promoted laminar flow in the airway, reducing the effect of the bronchospasm. Successful extubation may have removed a potent stimulus allowing resolution of the event. Additional studies are needed to address the possible benefit of heliox administration in management of bronchospasm under general anesthesia.

It is prudent for the anesthesia provider to integrate knowledge of pharmacology and physiology of heliox into current practice. Heliox is relatively inexpensive when used in short increments of time. Heliox is not readily available in all institutions, however. When faced with a severe bronchospasm, detection and treatment must be instituted rapidly. If a bronchospasm is resistant to standard therapy, other alternatives must be initiated to prevent life-threatening sequelae. Heliox therapy may contribute to effective management of brochospasm under general anesthesia in the appropriate patient.

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AUTHOR

Stacey L. Linck, CRNA, MA, is a staff nurse anesthetist at Ballas Anesthesia, St Louis, Mo. At the time this paper was written, she was a student at Truman Medical Center School of Nurse Anesthesia, Kansas City, Mo. Email: staceylinck@hotmail.com.

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