



Learning From Others:

Anesthesia
Quality Institute
ANESTHESIA INCIDENT
REPORTING SYSTEM (AIRS)

A Case Report From the Anesthesia Incident Reporting System

Review of unusual patient care experiences is a cornerstone of medical education. Each month, the AQI-AIRS Steering Committee abstracts a patient history submitted to the Anesthesia Incident Reporting System (AIRS) and authors a discussion of the safety and human factors challenges involved. Real-life case histories often include multiple clinical decisions, only some of which can be discussed in the space available. Absence of commentary should not be construed as agreement with the clinical decisions described. Feedback regarding this article can be sent by email to airs@asahq.org. Report incidents or download the AIRS mobile app at www.aqiairs.org.

Case

Nasal polyp surgery was completed on a 15-year-old, ASA Physical Status I male. A nasal endoscopy was performed at the end of the procedure and hyperplastic adenoids were found. These were not resected, and the finding was not communicated to the anesthesiologist. The patient was extubated after responding to commands but became obstructed soon after. Vigorous suctioning revealed copious blood in the airway. A nasal airway was placed. The patient developed laryngospasm, O₂ saturation decreased to 58 percent, and 3 minutes of positive pressure CPAP was used to break the laryngospasm. After stabilization, the patient was transported to PACU. One hour later the patient became tachypneic and O₂ saturation decreased to 85 percent. Chest X-ray revealed pulmonary edema affecting the left lung more than the right. A diagnosis of negative pressure pulmonary edema was made. After 24-hours of appropriate treatment, the patient made a full recovery and was discharged home.

Discussion

Pulmonary complications are a major cause of postoperative morbidity and mortality. Reported incidence of postoperative pulmonary complications range from approximately 11.5 percent to 33.4 percent, depending upon definition and criteria.^{1,2} The most common postoperative pulmonary complications are prolonged oxygen therapy by nasal cannula and atelectasis.^{1,2} Negative pressure pulmonary edema is less common and occurs following 0.05-0.1 percent of procedures performed under general anesthesia with endotracheal intubation.³ Importantly, patients experiencing one or more postoperative pulmonary complication face increased mortality, higher incidence of ICU admission and greater ICU and hospital lengths of stay.² Here, we review recognition, diagnosis, and management of laryngospasm and negative pressure pulmonary edema within the context of this interesting submission.

Laryngospasm is a sustained adduction of the vocal cords, with or without the false vocal cords and supraglottic soft tissues, that may result in partial or complete upper-airway obstruction.⁴ Incidence rates vary, but reportedly affect between 0.87 percent and 1.6 percent of patients, with higher rates among children and patients undergoing ENT procedures.^{4,5} Laryngospasm results in significant hypoxemia in 61 percent of patients and causes bradycardia in 6 percent patients, with higher rates (23 percent) among infants less than 1 year old.⁴ Known laryngospasm risk factors include light anesthesia during a time of airway stimulation, irritating volatile anesthetics such as desflurane and isoflurane, blood or secretions within the airway, and reactive airways due to asthma or recent upper-respiratory infections.^{4,6,7}

Prompt recognition and management are crucial for reestablishing patient oxygenation and ventilation. Help should be requested urgently if needed, and the offending stimulus should be removed if possible. This may involve careful suctioning of the airway with the recognition that this may in fact worsen the laryngospasm. Management then follows a progressive process of applying continuous positive airway pressure with 100 percent oxygen, deepening the anesthetic, administering a muscle relaxant and, if necessary, obtaining a definitive airway.⁸ If the laryngospasm does not break with positive airway pressure, excessive pressure should be avoided as this will lead to gastric insufflation. Instead, the level of anesthesia should be deepened, preferably with an I.V. bolus of propofol in 0.5 mg/kg increments.⁴ If propofol fails to release the laryngospasm, then I.V. succinylcholine is the drug of choice. Reported timing and dosage of succinylcholine vary, but suggested dosages range from 0.1 mg/kg to 2 mg/kg.⁴ It is also reasonable to administer succinylcholine intramuscularly with a dose of 4 mg/kg if I.V. access is not available.⁴ I.V. lidocaine may also reduce laryngospasm.⁹ Finally, if orotracheal intubation is not possible to open the patient's mouth, a nasopharyngeal airway may be necessary.

In this report, several factors increased the patient's risk for complications or laryngospasm. Communication is always essential for perioperative patient safety. In this example, nasal endoscopy revealed enlarged adenoids, which put the patient at increased risk for bleeding and airway irritation. Had this finding been effectively communicated, the anesthesiologist might have approached extubation differently and laryngospasm may have been prevented or mitigated. Specifically, knowledge of hypertrophied adenoids would have prompted an examination of the airway, control of bleeding and a slow, cautious approach to extubation, perhaps in the PACU. A gentle wake-up and a low threshold to deepen and re-paralyze with laryngospasm may benefit patients like this. Additionally, though the laryngospasm was successfully relieved with positive airway pressure, the patient experienced three minutes of laryngospasm with an oxygen desaturation to 58 percent. It is impossible to know definitively from this report whether measures were taken to deepen the anesthetic or paralyze the patient. In cases like this, the addition of propofol or succinylcholine may help reduced the duration of laryngospasm. In severe cases a surgical airway may be necessary.

In this case, laryngospasm was an important cause of postoperative negative pressure pulmonary edema (NPPE). NPPE is caused by strong respiratory efforts against a closed glottis or obstructed airway, which leads to strong hydrostatic pressures affecting the pulmonary vasculature, causing a transudative pulmonary edema.¹⁰ One study reported greater incidence of NPPE among men, young patients, and patients classified as ASA Physical Status I or II.^{11,12} These patients can generate intrathoracic pressures against a closed airway as strong as -140 cm H₂O.^{11,12} Patients experiencing NPPE typically present with dyspnea, pink frothy sputum and bilateral infiltrates on their chest radiograph immediately after resolution of an upper-airway obstruction. Less commonly, NPPE may be delayed, as was the case with this patient who developed NPPE after recovering in PACU for one hour.¹³ This supports longer or more conservative periods of observation for patients experiencing severe laryngospasm during extubation.

NPPE management is largely supportive. Some patients require endotracheal intubation or tracheostomy to bypass the obstructed airway. All patients require supplemental oxygen and positive pressure ventilation, which may be invasive or non-invasive depending upon patient stability and severity of NPPE. Though it has not been specifically studied in NPPE, the lung-protective, low-tidal-volume ventilation used in ARDS has been recommended for these patients.¹⁰ Similarly, bronchodilators and gentle diuresis in stable, normovolemic patients has been recommended to hasten NPPE recovery, though more research is needed to support these recommendations.¹⁰ It is unclear which methods were used in this case. However, most patients recover fully within 24-48 hours and experience no long-term sequelae.

In conclusion, we recommend good communication between all team members and taking steps to anticipate and prevent obstruction and laryngospasm. If laryngospasm occurs, the team should aggressively treat laryngospasm to avoid pulmonary edema.

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