Asystole From Direct Laryngoscopy: A Case Report and Literature Review

Andrew J. Redmann, MD,* Gregory D. White, DDS,† Benu Makkad, MD,‡ and Rebecca Howell, MD*

*Department of Otolaryngology–Head and Neck Surgery, University of Cincinnati Medical Center, Cincinnati, Ohio, †Department of Surgery, Division of Oral and Maxillofacial Surgery, University of Cincinnati Medical Center, Cincinnati, Ohio, and ‡Department of Anesthesia, Division of Cardiac Anesthesia, University of Cincinnati Medical Center, Cincinnati, Ohio

The rare and potentially fatal complication of asystole during direct laryngoscopy is linked to direct vagal stimulation. This case describes asystole in an 85-year-old female who underwent suspension microlaryngoscopy with tracheal dilation for subglottic stenosis. Quick recognition of this rare event with immediate cessation of laryngoscopy resulted in the return of normal sinus rhythm. This incident emphasizes the implications of continued vigilance during laryngoscopy and the importance of communication between the anesthesia and surgical staff to identify and treat this rare complication. The case was successfully concluded by premedication with an anticholinergic and by increasing the depth of anesthesia.

Key Words: Asystole; Laryngoscopy; Anesthesia.

Direct laryngoscopy is routinely performed by both anesthesiologists and otolaryngologists and is commonly associated with dental injury, tongue paresis, and/or bleeding gums. However, despite its routine nature, more serious complications, such as airway injury, esophageal injury, or bradycardia due to the stimulation of the vagal reflex, can occur.¹ In extreme situations, it can present as asystole. Asystole, especially intraoperatively, is cardiogenic until proven otherwise. Described herein is a case of direct laryngoscopy resulting in asystole attributed to an extreme vagal response.

CASE REPORT

An 85-year-old female with a history of coronary artery disease and a cardiac stent placement, hypertension,

gastroesophageal reflux disease, and hypothyroidism was referred for the surgical management of subglottic stenosis. The patient had a remote history of intubation and intensive care unit admission for 3 days in 2003. Over the past several months, she had noticed a decrease in her exercise tolerance. Recently, she developed increased stridor with daily exercise. On examination, the patient elicited soft stridor with prolonged inspiration but talked comfortably throughout the visit. She underwent a chest computerized tomography scan, which confirmed the presence of grade 3 subglottic narrowing with right lateral shelving and posterior tracheal narrowing. Examination findings by both rigid stroboscopy and flexible laryngoscopy confirmed the diagnosis (Figure 1). She was scheduled for elective surgery to undergo a suspension microlaryngoscopy with endoscopic dilation, a possible steroid injection, and a possible carbon dioxide laser excision of the stenosis.

The patient underwent a preoperative anesthesia consultation given her cardiac history. She had a good exercise tolerance and was still able to walk 2 miles a day 5 days a week on the treadmill. However, the peak

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Address correspondence to Dr Rebecca Howell, Department of Otolaryngology–Head and Neck Surgery, Division of Laryngology, Medical Science Building Room 6212, University of Cincinnati Medical Center, Cincinnati, OH 45239; howellrb@ucmail.uc.edu.

Figure 1. Preoperative stroboscopy findings of grade 3 stenosis of the subglottis.

activity level had progressively declined over the last several months with the associated stridor. She had an echocardiogram that showed normal systolic function. She was also well optimized on medications including beta blocker, statin, aspirin, and clopidogrel.

The patient was taken to the operating room and an intravenous induction was performed in routine manner with propofol and remifentanyl infusions (100 mcg/kg/min and 0.08 mcg/kg/min respectively) preceded by remifentanyl bolus (25 mcg). After paralysis with succinylcholine (30 mg) and a small bolus administration of propofol (20 mg), the patient was easy to mask ventilate, and the decision was made to proceed with direct laryngoscopy. No priming dose of succinylcholine was used in this case. Upon insertion of the Dedo laryngoscope (Karl Storz, El Segundo, Calif; Figure 2), severe bradycardia followed by asystole and an undetectable pulse oximetry signal was noticed on the monitor (Figure 3). The laryngoscope was immediately removed and there was spontaneous return of normal sinus rhythm and the pulse oximetry signal. The equipment was checked for malfunction or disconnection. A second laryngoscopy was attempted with radial pulse palpation after additional bolus administration of remifentanyl (25 mcg) and propofol (20 mg), resulting in a similar episode coupled with the disappearance of the radial pulse during asystole and the return of the pulse upon removal of the laryngoscope. The airway was easily maintained with mask ventilation. There was a discussion between the anesthesiologist and the surgeon about the need to proceed or abort the procedure. The patient had a stable cardiac disease and good exercise tolerance, so an underlying cardiac disease was considered unlikely. In the presence of extensive airway stenosis, the decision to proceed with the surgery was made with the clinical suspicion of the event being a vagally mediated response as opposed to an actual cardiac event. There was a change in the surgical plans to use a sickle knife and telescope instead of laser and

Figure 2. Dedo laryngoscope.

microscope and to proceed using the intermittent apneic technique. Along with increasing remifentanyl infusion (0.12 mcg/kg/min), the inhalational anesthetic agent sevoflurane was added to increase the depth of anesthesia, and atropine (1.6 mg total; 0.8 mg followed by another 0.8 mg) was given to pretreat the vagal response. After an additional dose of propofol (20 mg) and succinylcholine (30 mg), the Dedo laryngoscope was quickly suspended using the Lewy arm, resulting in a transient bradycardia to the 30s that recovered without an occurrence of asystole. A 0° rigid endoscope was used, which showed 90% narrowing of the trachea, allowing a 5-mm telescope to scarcely fit through the stenosis. The steroid agent Kenalog was then injected circumferentially around the stenosis, and a sickle knife was used to cut the 3- and 6-o'clock positions. Next, tracheal dilation was performed using a 14-mm balloon (Boston Scientific, Marlborough, Mass) inflated to 8 atmospheres pressure for 2 minutes. The balloon was then removed to photo document the residual grade 1 stenosis. The patient was easily intubated using a 5-0 endotracheal tube. She was successfully extubated in the operating room without any subsequent complications.

Upon arrival in the recovery room, an electrocardiogram and troponins were ordered and a cardiology consult was requested. The electrocardiogram and troponins were unremarkable. The patient had an uneventful recovery from the anesthetic. No further recommendations were made by the cardiology service, who confirmed the clinical suspicion of an extreme case of vagal stimulation. She was observed overnight and discharged the following day. One month later, she had a complete resolution of inspiratory stridor and no further cardiac issues.

DISCUSSION

Asystole following direct laryngoscopy is a rare complication, with only 2 other case reports in the literature. It

Figure 3. Asystole at 2:20 PM captured on multiple monitoring devices with spontaneous return of pulse and normal sinus rhythm following laryngoscope removal. Repeat episode of asystole with loss of radial pulse too brief to be captured on documented operating room record at 2:22 PM upon second attempt with laryngoscope. Previously witnessed bradycardia and asystole counteracted on third attempt of laryngoscope placement (2:29 PM) with anticholinergic medication and deepening of anesthetic.

is well known that laryngoscopy in children can precipitate increased vagal tone and bradycardia, but it is less common in adult populations.¹ In adult patients with documented asystole during direct laryngoscopy, the patients had either severe cardiac disease or neurologic trauma with increased intracranial pressures.^{2,3}

There are 2 presumed mechanisms of bradycardia during direct laryngoscopy. First, hypoxia can occur, leading to reflex bradycardia and systemic vasoconstriction and shunting of oxygen to the brain. This was less likely in our case, given normal O₂ saturations and easy mask ventilation during the entire case. Second, direct vagal stimulation of the receptors in the larynx and pharynx may lead to an increased parasympathetic tone and subsequent bradycardia and/or asystole. The laryngeal surface of the epiglottis is innervated by the internal branch of the superior laryngeal nerve (the vagus nerve), and suspension laryngoscopy with a Dedo laryngoscope is particularly stimulating to this area compared to intubation with a Macintosh or Miller laryngoscope blade,⁴ which is positioned at the vallecula. The patient underwent suspension laryngoscopy, which is known to be more vagally stimulating than direct laryngoscopy. In addition, she was given vagotonic agents, which included propofol, remifentanyl, and succinylcholine. Remifentanyl is known to reduce sympathetic activity from the central nervous system,^{5,6} and succinylcholine causes stimulation of the muscarinic receptors in the sinoatrial node to cause bradycardia.⁷ The data are sparse on systematic trials investigating this rare occurrence, but limited data suggest that topical lidocaine and deep anesthesia may decrease the likelihood of asystole after direct laryngoscopy.⁸

It is likely that the patient had elevated vagal tone at baseline given her age and her active lifestyle. She was on a beta blocker, which blunted her heart rate response to sympathetic stimulation. The stimulation of her laryngopharynx, coupled with the use of the vagotonic anesthetic agents remifentanyl, propofol, and succinyl-

choline, increased this tone, leading to an episode of asystole that was resolved after the cessation of stimulation. Other considerations for the differential diagnosis of severe bradycardia/asystole include underlying cardiac conduction abnormalities, myocardial infarction, pulmonary embolism, cerebrovascular accident, and swallowing syncope. According to literature, the latter is elicited by swallowing, manifesting as bradyarrhythmias, asystole, and hypotension attributed to vagal stimulation.^{9,10} Because the resting electrocardiogram was normal, the workup was negative for myocardial infarction, and the patient had normal neurologic examination, cardiac conduction abnormalities, myocardial infarction, and cerebrovascular accident were unlikely diagnoses. The absence of oxygenation difficulty and normal end tidal carbon dioxide (ETCO₂) makes pulmonary embolism less likely, leaving severe vagal reaction the most likely diagnosis.

CONCLUSION

Asystole after direct laryngoscopy is a rare and potentially life-threatening complication. In this case, asystole was most likely due to a paradoxical increase in vagal tone with upper airway stimulation. Clear communication between the anesthesia staff and surgeon can prevent a potential catastrophe, and pretreatment with anticholinergics, such as glycopyrrolate or atropine, may blunt the increased vagal tone and provide stable hemodynamics. Reducing the laryngeal reflexes with aerosolized lidocaine and inhalational anesthetics should also be taken into consideration.

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