Asystole During Laryngoscopy of a Patient With Pleural and Pericardial Effusions: A Case Report

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A 53-year-old woman presented to the operating room for surgical correction of pericardial and pleural effusions. Her history included stage IV breast cancer, well-controlled hypertension, and diverticulitis. Although her baseline blood pressure, heart rate, and respirations were normal, she was short of breath with diminished breath sounds on the left side of the lungs and required oxygen, 2 L/min via nasal cannula.

The nurse anesthesia student, under the direction of the Certified Registered Nurse Anesthetist (CRNA) and anesthesiologist, induced general anesthesia with etomidate, fentanyl, lidocaine, and succinylcholine. During placement of a double-lumen endotracheal tube, the patient became asystolic. The nurse anesthesia student immediately withdrew the laryngoscope, and the

patient returned to normal sinus rhythm. A second attempt at laryngoscopy produced asystole as well. Again, the laryngoscope was withdrawn, and the patient returned to normal sinus rhythm. After resuming ventilation with 100% oxygen and administering atropine, 0.4 mg, the next intubation was successful, producing no untoward effects. Reintubation at the end of the case with a single lumen endotracheal tube was uneventful. The patient was transported to the intensive care unit and mechanically ventilated overnight. The next morning, she was extubated with no further anesthetic complications.

Key words: Asystole, cardiac effusion, laryngoscopy, pleural effusion.

Asystole during laryngoscopy is an uncommon occurrence. It has been attributed to vagal reflexes, inadequate depth of anesthesia, and the use of vagotonic drugs. Our patient had concurrent pericardial and pleural effusion, and we questioned whether this combination of disease processes attributed to the asystole during laryngoscopy.

A review of the literature showed that few cases of asystole during laryngoscopy of the adult have been documented. Wells[1] reported the cases of 3 patients with severe ischemic heart disease who had had profound bradycardia or asystole with laryngoscopy. The cases of asystole and bradycardia were attributed to inadequate depth of general anesthesia.[1] In another case study, Cheong et al[2] reported the case of a healthy male patient who experienced asystole during laryngoscopy. The asystole was attributed to the activation of vagal reflexes during manipulation of the airway and use of vagotonic drugs during induction of general anesthesia.[2] Mizuno et al[3] reported the case of a 43-year-old woman with Plummer disease (toxic nodular goiter) who had a sinus arrest during laryngoscopy. Her sinus arrest was attributed to a vasovagal reflex. The patient received a total of 0.75 mg of atropine and was intubated uneventfully after laryngoscopy.[3]

In 1994, Sutera and Smith[4] reported a case of asystole during direct laryngoscopy and tracheal intubation. Their patient was a healthy 17-year-old boy with a history of re-

current bilateral spontaneous pneumothoraces who was scheduled to have a thoracoscopy to resect apical blebs.[4] During the laryngoscopy, the patient's heart rate decreased to 45/min and when the trachea was intubated, the patient became asystolic. Atropine, 0.5 mg, was used to treat the asystole and the heart rate climbed to 85/min.[4] The case was otherwise uneventful. This same patient returned to the operating room 4 months later for a thoracotomy to resect blebs. The patient received atropine, 0.8 mg, before the laryngoscopy and still had a decrease in his heart rate to 40/min. The laryngoscope was removed, an additional 0.4 mg of atropine was given, and the next laryngoscopy was uneventful.[4] The bradycardia and asystole were attributed to the activation of the parasympathetic nerve fibers during direct laryngoscopy.[4]

Pericardial effusion, an increased amount of pericardial fluid surrounding the heart, is a common feature in neoplastic diseases but occurs less frequently than pleural effusions.[5,6] Signs and symptoms of pericardial effusion include chest pain or pressure, dyspnea, and nausea. Physical examination may reveal muffled heart tones, jugular venous distension, pulsus paradoxus, and pericardial rub.[6] Hemodynamic changes include hypotension, tachycardia, tachypnea, and weak peripheral pulses. Pericardial effusions may lead to cardiac tamponade, which is a compression of the heart by the accumulation of fluid in the pericardial sac. Tachycardia, hypotension, and changing levels of consciousness are symptoms of cardiac tamponade.

Malignant pleural effusion is a common life-threatening problem in patients with advanced malignancies including breast cancer.[7] Recurrent malignant pleural effusions can decrease quality of life and cause a rapid demise in the patient if not treated.[8] Symptoms of pleural effusion range from shortness of breath, chest pain, cough, rapid breathing, and abdominal pain to no symptoms at all.[9] Pleural effusion can be surgically treated by draining the fluid out of the chest cavity followed by talc or chemotherapeutic agent pleurodesis. It also may be treated with an indwelling pleural catheter in which medication is instilled to cause pleural symphysis.[8] Once the pleural effusion is treated, dyspnea usually resolves.

Case Summary

A 53-year-old woman was scheduled for surgical treatment of a pericardial and pleural effusion. She was diagnosed with breast cancer in 2001, for which she received chemotherapy. The patient was unsure which chemotherapeutic agent was used in her therapy and it was not documented in her medical record. The patient thought her last dose of chemotherapy was more than 2 years before this hospital admission. Previously, she had been surgically treated for pericardial and pleural effusions without incident. She had hypertension, which was well-controlled with atenolol. Her symptoms included dyspnea, for which she was on 2 L/min of oxygen per nasal cannula, and chest pressure. Her blood pressure was 102/66 mm Hg, her pulse was 83/min, and her respirations were 20/min. Her oxygen saturation on 2 L/min of oxygen was 98%. She was taken to the operating room and sedated with 2 mg of midazolam in 2 divided doses while femoral central and arterial lines were inserted by the surgeon. Once the patient was preoxygenated with 100% oxygen for 5 minutes, the nurse anesthesia student induced her with 500 μg of fentanyl in divided doses; lidocaine, 100 mg; etomidate, 8 mg; and succinylcholine, 120 mg. The patient was easily ventilated with 100% oxygen.

When the nurse anesthesia student attempted to place a double-lumen endobronchial tube, the patient became asystolic and her central venous pressure and arterial-line waveforms flattened. After removal of the laryngoscope, the patient returned to a normal sinus rhythm and normal blood pressure. The nurse anesthesia student attempted a second laryngoscopy but met with the same results. Again, the laryngoscope was withdrawn, and the patient's heart rhythm and vital signs returned to normal. It is important to note that the patient's oxygen saturation reading never fell below 97%. The patient was again ventilated easily with 100% oxygen and given atropine, 0.4 mg, intravenously. When her heart rate began to increase, she was intubated without difficulty or incident. A pericardial window and video-assisted thoracoscopy with talc pleurodesis were performed, and the patient's vital signs

remained stable. At the end of the case, the anesthesia team and surgeon decided to ventilate the patient overnight in the intensive care unit. The endobronchial tube was exchanged for a single-lumen endotracheal tube without incident. The patient was extubated the next day without further complications.

Discussion

Several causative factors for asystole in this surgical patient can be considered. Bradycardia and asystole have been reported and are known risks of laryngoscopy, related to vagal stimulation. Inadequate levels of anesthesia during oral airway procedures may cause bradycardia or asystole. Other considerations include a history of chemotherapy, hypertension treated with a beta-blocking agent, and the physical examination, which was positive for pulmonary and pleural effusions.

A discussion with Michael Levitzky, PhD (written communication, May 2, 2006), pulmonary physiologist and author of *Pulmonary Physiology*, revealed that neither the pulmonary effusion nor the pericardial effusion was likely to cause the asystole with laryngoscopy. Levitzky stated that the asystole was probably caused by a major vagotonic reaction to the laryngoscopy.

Stan Hall, MD, PhD (oral communication, May 25, 2006), anesthesiologist and professor of physiology, arrived at the same conclusion as Levitzky and suggested the possibility that cardiotoxic effects from chemotherapeutic agents caused the asystole with laryngoscopy.

Certain chemotherapeutic agents used in the treatment of breast cancer are cardiotoxic. Injury to the heart may include arrhythmias (ventricular ectopy), conduction disturbances (prolonged Q-T interval, bradycardia, heart block), and congestive heart failure-like symptoms.[10] The patient had a normal electrocardiogram and did not exhibit any of these symptoms. The patient thought her last chemotherapy treatment was more than 2 years before this surgery.

This patient was receiving atenolol for her chronic hypertension. Atenolol is a beta-adrenergic blocking agent that blunts the heart's response to sympathetic stimulation. This patient also received a moderate dose of fentanyl for induction. Fentanyl can decrease sympathetic outflow. With a combination of beta-adrenergic blockade (atenolol) and decreased sympathetic outflow (fentanyl), the patient may have been rendered highly vagotonic, predisposing her to bradycardia and asystole during laryngoscopy. Once the laryngoscope was withdrawn from the oropharynx, the patient's vital signs and heart rhythm returned to normal, so this incident was considered a major vagotonic event.

Conclusion

When anesthetizing patients with malignant neoplasms and pericardial and/or pleural effusions, close monitoring

of the cardiac rhythm is vital. These patients appear to have highly vagotonic functioning. When fentanyl or sympatholytic agents are administered, the patient may be at increased risk for bradyarrhythmias and asystole during laryngoscopy and vagal stimulating maneuvers. While pretreatment with vagolytic agents may prevent the development of bradyarrhythmias during vagal stimulating maneuvers, nothing replaces vigilance and early intervention should such an episode occur.

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