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Intraoperative Bronchospasm Induced by Stimulation of the Vagus Nerve

Maywin Liu, M.D., Andrea G. Schellenberg, M.D.,† Terry Patterson, Ph.D.,‡ Douglas C. Bigelow, M.D.,§ Mark M. Stecker, M.D., Ph.D.‡*

INCREASED peak inspiratory pressure and bronchospasm during general anesthesia can have many etiologies, including the patient's intrinsic disease and mechanical, chemical, or neurogenic causes. We present a case of bronchospasm induced by direct stimulation of the vagus nerve.

Case Report

A 56-yr-old man underwent a left glomus vagal tumor resection during general anesthesia. Preoperative medical history was significant for restrictive lung disease resulting from asbestosis. In addition there was a long smoking history, obesity (5′6″; 260 lbs), and significant cervical stenosis at C5–C6. Previous pulmonary evaluation showed mild asbestosis, requiring no treatment. No pulmonary function tests were obtained preoperatively. The patient denied any history of wheezing or use of any medications.

Because of the severe cervical stenosis, the patient was orally intubated awake with a fiberoptic scope. Placement of a 7.0-mm ID endotracheal tube (ETT) was attempted initially, although the ETT could not be passed through the cords. A 6.0-mm ID ETT was subsequently placed without difficulty. The patient was induced using thiopental and fentanyl, and the lungs were mechanically ventilated. Wheezing was noted immediately after intubation and induction and was treated by deepening the anesthetic depth. Anesthesia was maintained with 70% N_2O/30% O_2 and isoflurane (end tidal concentration stable at 0.9%). After intubation, hookwire electrodes were placed in the vocal cords during direct laryngoscopy. Despite pretreatment with 0.2 mg of glycopyrrolate, the patient continued to produce copious amounts of secretions during and after intubation. No muscle relaxant was used so that a nerve stimulation monitor could be used to identify nerves during the dissection and tumor removal. The patient was positioned with the head 180° away from the anesthesia machine with the head turned to the right. The patient was ventilated with 600 cc tidal volume with peak inspiratory pressures (PIPs) ranging from 55 to 60 mmHg.

The case progressed uneventfully with periodic stimulation of the hypoglossal, vagus, and spinal accessory nerves during dissection until approximately 10 h after incision when the patient developed

* Assistant Professor, Department of Anesthesia.

† Resident, Department of Anesthesia.

‡ Assistant Professor, Department of Neurology.

§ Assistant Professor, Department of Otorhinolaryngology: Head and Neck Surgery.

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Address reprint requests to Dr. Liu: Department of Anesthesia, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania, 19104. Address electronic mail to: mliu@mail.med.upenn.edu

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bradycardia to a low of 34 with manipulation of the carotid body. This was treated with 1 mg of atropine, and the heart rate increased to and remained at 90–100. Approximately 90 min later, the surgeons electrically stimulated the distal vagus nerve segment with an approximately 20-s train of stimuli at a current level of 8.8 milliamperes (ma). This current level was required to obtain an adequate compound motor action potential from the hookwire electrode placed in the vocal cords. Immediately after stimulation of the vagus nerve, the PIPs increased suddenly from 55–60 mmHg to 80–90 mmHg with decreased tidal volumes. Mechanical ventilation was discontinued, and the patient was ventilated by hand. Ventilation continued to be difficult because of extremely high resistance. End-tidal isoflurane concentration remained at 0.9%. The surgical field was inspected. No compression of the ETT or patient's face or trachea was found. Because of the patient's copious production of secretions and to ensure patency of the ETT, a suction catheter was passed into the ETT after ventilating with 100% O_2. A small amount of thick secretion was removed. No change in resistance or PIPs was noted after suctioning, and the O_2 saturation, which had been 96% at the end of suctioning, decreased precipitously to 77%. Auscultation of the lungs revealed bilateral loud wheezes throughout. The patient was administered 10 μg of epinephrine intravenously and four puffs of albuterol *via* the ETT. The O_2 saturation increased to 97% within approximately 30 s and to 100% a few minutes later on 100% O_2, accompanied by markedly decreased resistance to ventilation. On placing the patient back on the ventilator, the PIPs had decreased to the patient's baseline 50–60 mmHg. Auscultation of the chest revealed clear breath sounds throughout.

No significant change in the patient's hemodynamics was noted with stimulation of the vagus or with the occurrence of the bronchospasm. The heart rate remained in the high 80s to mid-90s range, and mean arterial pressure (MAP) was essentially unchanged (decreased from 93 to 83 mmHg). After administration of the epinephrine, an increase in blood pressure to systolic 170 from 100 mmHg (change in MAP from 83 to 117 mmHg) was noted, but no change in heart rate was seen. The remainder of the case progressed uneventfully with no further episodes of bronchospasm. No further stimulation of the vagus nerve was surgically required.

The patient received a planned elective tracheostomy at the end of the case and was transported to the surgical intensive care unit paralyzed, sedated, and hand-ventilated. The patient was allowed to recover from the effects of the muscle relaxant gradually. No further episodes of bronchospasm were noted during his postoperative course.

Discussion

Sudden onset of increased peak inspiratory pressure intraoperatively can have many causes. Patient intrinsic disease; mechanical factors, *e.g.*, kinked ETT, endobronchial intubation, mucous plug, surgeon leaning on the patient's ETT, neck, or chest; chemical factors (aspiration); or drug induction can all cause an increase in PIPs.[1] In this particular case, the cause of the bronchospasm appeared to be neurogenic as a result of direct stimulation of the vagus nerve. The vagus nerve was successfully identified during surgery with the help of nerve stimulation recorded from the vocal cord electrodes. It is possible that the patient imperceptibly coughed, although no change was seen on the end-tidal CO_2 tracing at the onset of the event to indicate a cough. The appearance of the bronchospasm corresponded temporally with stimulation of the vagus nerve. The stimulation used at that point was greater than that used earlier in the case (8.8 ma *vs.* 0.5–1.0 ma, respectively), and the burst delivered was somewhat longer than used previously during the case. The resistance to ventilation remained high and unchanged after suctioning, most likely indicating that the bronchospasm was not initiated by the suctioning. No other etiology could explain the bronchospasm; no mechanical cause was found; no additional drugs had been administered in the previous 90 min, and there was no change in the end-tidal isoflurane concentration at that time.

Bronchoconstriction can result from direct activation of receptors in the pulmonary system or from neurogenic activation.[2] Vagally mediated bronchoconstriction affects the large airways.[3] Although the vagus nerve is known to play a role in bronchoconstriction, production of bronchospasm by direct stimulation of the vagus nerve has never been demonstrated in humans.[4,5] In animal studies, however, bronchospasm could be induced by direct stimulation of the vagus nerve.[3,4,6] This neurogenic bronchospasm could be relieved with the use of intravenous or inhaled atropine. In this patient the atropine given 90 min earlier did not have a protective effect. This is most likely a result of the short duration of action of atropine. In animal studies the bronchodilatory effect of intravenously administered atropine had an effective duration of action of 30 min.[7] The patient had been treated with glycopyrrolate, an anticholinergic drug with a longer duration of action, previously. This may explain why bronchoconstriction caused by direct vagal stimulation was not seen with earlier testing.

The amplitude and length of time of the vagal stimulation may have contributed to the bronchospasm. Lower levels of stimulation used previously did not appear to cause the bronchoconstriction.

During the vagally induced bronchospasm, minimal effects were noted on the hemodynamics (approximately 10% change in MAP). The pulmonary system appears to have a greater sensitivity to vagal stimulation than the cardiac system. In dogs, as in this report, stimulation of the vagus nerve induced bronchospasm without affecting the heart rate. A differential effect was also seen in dogs in response to atropine administration.

Vagally induced bronchospasm could be completely relieved with atropine,and heart rate was essentially unaffected.[7]

Although monitoring of the vagus nerve during surgical procedures is a well-accepted technique for protection of the nerve during resection of glomus tumors,[8] it is possible, as this case demonstrates, that direct stimulation of the vagus nerve in humans can lead to bronchoconstriction. This etiology should be considered in cases with sudden onset of increased PIPs when stimulation and monitoring of the vagus is performed intraoperatively. Patients with a history of bronchospastic disease may be at increased risk for this. This particular patient may have been at higher risk for developing bronchospasm in response to vagal stimulation because of his history of bronchospasm after intubation. To decrease the risk of vagally induced bronchospasm, current levels of the stimulus should be kept at the lowest possible level and shortest duration required to obtain a reproducible response and to prevent stimulus-associated side effects.

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The Cuffed Oropharyngeal Airway, a Novel Adjunct to the Management of Difficult Airways

Shoichi Uezono, M.D., Takahisa Goto, M.D.,* Yoshinori Nakata, M.D., M.B.A.,* Fumito Ichinose, M.D.,* Yosinari Niimi, M.D., Ph.D.,† Shigeho Morita, M.D.‡*

A CUFFED oropharyngeal airway (COPA) is a new airway device, which is fundamentally a regular oropharyngeal airway with a large cuff attached around the distal end. The cuff separates the tongue from the posterior pharyngeal wall to create a patent airway. Proximally it has a standard 15-mm adapter connectable to a breathing circuit. Like a laryngeal mask airway (LMA), it is intended primarily for use in spontaneously breathing patients who are not at risk of aspiration of gastric contents. Consistent with the preliminary reports by others,[1,2] our initial experiences with COPA in more than 100 patients have been favorable.

We report here our experience of using this new device in two patients requiring general anesthesia for whom fiberoptic intubation was performed with the COPA in place.

* Assistant Professor of Anesthesiology.

† Associate Professor of Anesthesiology.

‡ Professor of Anesthesiology and Chair.

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Address reprint requests to Dr. Uezono: Department of Anesthesiology, Teikyo University Ichihara Hospital, 3426-3, Anesaki, Ichihara-shi, Chiba 299-0111, Japan. Address electronic mail to: QZQ07627@niftyserve.orjp

Key words: Equipment; fiberoptic; general anesthesia; intubation; nasal; pharyngeal; tracheal.

Case Reports

Case 1

A 50-yr-old woman with rheumatoid arthritis was scheduled for abdominal hysterectomy during general combined with epidural an-