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The Esophageal Detector Device Is Unreliable When the Stomach Has Been Ventilated

Alexander H. Andres, M.D.,* Holger Langenstein, M.D.†

RAPID and accurate diagnosis of the misplacement of an endotracheal tube in the esophagus remains one of the most important problems in anesthesia and intensive care. Clinical signs observed through auscultation, inspection of thoracic excursions, refilling of the breathing bag, or water vapor condensing at the endotracheal tube may be misleading in as many as 30% of patients.¹ Although end-tidal carbon dioxide measurement and bronchoscopy remain the gold standards for evaluating the endotracheal tube position, the esophageal detector device (EDD) recently has assumed an important clinical role, with sensitivity and specificity rates for identifying the endotracheal tube position in adults of nearly 100%.²⁻⁸ An EDD may even indicate correct endotracheal tube position in situations in which the end-tidal carbon dioxide concentration fails, such as cardiopulmonary resuscitation or severe bronchospasm.⁸ Most importantly, misdiagnosis of tubes placed in the esophagus using an EDD in adults is rare.^{6,9} Of more than 1,000 reported applications, only two cases have been described in which air could be aspirated from a tube placed in the esophagus: one in a morbidly obese patient⁷ and one in a patient with gastric distension after vigorous attempts to apply mask ventilation.⁸ Unreliable results also were found in infants younger than 1 yr¹⁰ or

when technically unsuited EDDs were used in adults (*i.e.*, those with a 20-ml bulb).

We report the misdiagnosis of an esophageally placed endotracheal tube with a 60-ml syringe-type EDD in an adult with unexpected laryngospasm after reinsertion of an intubating laryngeal mask airway (ILMA),¹¹ in whom gastric distension of the stomach had occurred as a result of attempted positive-pressure ventilation.

Case Report

A 44-yr-old man (weight, 90 kg) was scheduled for reconstruction of the mandible 1 yr after radical resection of a tonsillar carcinoma with suprahyoidal and neck dissection bilaterally and radionecrosis of the mandible thereafter. A percutaneous gastrostomy had been performed because the patient could not swallow. Examination of the airway showed a slightly reduced thyromental distance (5.5 cm), a moderately reduced mouth opening (3.2 cm awake, 4.5 cm during anesthesia), a fixed retrognathia, and a 1.5-cm right-sided displacement of the thyroid cartilage by scar tissue. General anesthesia was started with 4 mg/kg thiopentone; 0.1 mg fentanyl; and enflurane, 2.5 vol%, in 100% oxygen. Ventilation by face mask was achieved readily, and 0.5 mg/kg atracurium was given. Modest insufflation of the stomach was apparent during mask ventilation and also during ventilation with a correctly inserted ILMA at normal airway pressures (15 cm water) well below the leak pressure of the ILMA (40 cm water).

This patient was a participant in a study to compare the success of blinded intubation through an ILMA *versus* a standard laryngeal mask airway. His trachea was intubated successfully using an ILMA, but a tube could not be placed through a standard laryngeal mask airway. Therefore, we decided to use blinded intubation through the ILMA, again followed by a tube exchange maneuver for definite nasal intubation.¹² We reinserted the ILMA a second time without difficulty 30 min after induction. Laryngospasm occurred but was not detected immediately by the ventilating anesthetist, who was a novice user of the ILMA. Gastric ventilation occurred with the ILMA but was difficult to detect because of the patient's obesity. Thoracic excursions with ventilation could be seen in the xiphoid region; they were slightly more pronounced in the left hemithorax with no movement in the infraclavicular parts. No expired carbon dioxide was detected. Blinded tracheal intubation through the ILMA was attempted using a 7.5-mm ID endotracheal tube without the application of force to an approximate depth of 24 cm. The position of the endotracheal tube was identified using a 60-ml syringe-type EDD. One hundred twenty milliliters of air was aspirated readily, followed by two breaths that did not deliver

* Resident.

† Consultant.

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Address reprint requests to Dr. Andres: University of Bochum (Ruhr Universität Bochum am Knappschaftskrankenhaus), Department of Anesthesiology, In der Schornau 23-25, 44892 Bochum, Germany. Address electronic mail to: Alexander.H.Andres@ruhr-uni-bochum.de

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carbon dioxide. Atracurium (15 mg) was given. The stomach was emptied by manual compression and the EDD was used again. After aspirating another 90 ml air, further air aspiration was no longer possible, indicating the esophageal position of the endotracheal tube by the EDD. The endotracheal tube was removed. Ventilation of the lungs *via* the ILMA was possible, as apparent by carbon dioxide detected in the expired air. Blinded oral intubation through the ILMA was performed, and subsequently this tube was exchanged for a nasotracheal tube. The operation was uncomplicated and the patient had no further problems. Oxyhemoglobin saturation by pulse oximetry was greater than 92% during the entire procedure.

Discussion

This case shows that an EDD may not detect esophageal positioning of an endotracheal tube when gastric inflation has occurred inadvertently. This can happen with a face mask, with all types of laryngeal masks, or with an esophageally placed endotracheal tube when positive-pressure ventilation is performed. Repeated gastric inflation also may fill the esophagus with air, which apparently happened in our patient. We initially questioned whether a mispositioned ILMA might have facilitated such distension, but the correct insertion procedure, the position of the ILMA, and correct ventilation after relaxation do not support a malposition of the ILMA after its second insertion. However, users of the ILMA regularly find an esophageal tube position if it is diverted from the laryngeal inlet by laryngospasm.

Studies with an EDD reported a sensitivity rate of 100% for discriminating esophageal from endotracheal intubation in healthy adults classified as American Society of Anesthesiologists grades I and II who were undergoing routine operations,^{1,2-4,13} in children between the ages of 2 and 10 yr,¹⁰ or when the EDD was used by paramedics.¹⁴ Even a nasogastric tube or an endotracheal tube placed in the esophagus did not interfere with the reliability of detecting esophageal intubation.⁶ One consistent finding in most studies was delayed refill of the bulb-type EDD, which occurred in 6–30%,^{3,4,6-8} which may be interpreted falsely as the mispositioning of the endotracheal tube when, in fact, it was correctly placed.

Some studies have directly addressed the problem of EDD failure in the setting of previous air insufflation into the esophagus. In a study that used one pig in which the esophagus was ventilated with a bag *via* a tube for 1 min before the EDD was tested, 30 investigators did not fail to discriminate endotracheal from esophageal intubation.¹ Salem *et al.*⁵ reported that the delivery of three small breaths (tidal volume of 300–350 ml each) through a tube placed in the esophagus to mimic gastric insuffla-

tion did not affect the reliability of the self-inflating bulb, indicating neither instantaneous nor delayed reinflation. This led to the conclusion that false-positive findings with an EDD are “probably nonexistent”⁶ or “only theoretical.”⁹ However, Lang *et al.*⁷ reported a misdiagnosis in 1 of 54 grossly obese patients when the bulb was squeezed through an esophageally placed endotracheal tube before aspiration. A second case of an esophageal intubation undetected by an EDD was reported after vigorous attempts at mask ventilation before testing with a bulb-type EDD.⁸ In addition, the EDD was found to be unreliable in children younger than 1 yr¹⁰ and in adults if the bulb used was too small.

In conclusion, we add one more case in which an EDD failed to show correct endotracheal tube placement in a situation of repeated ventilation of the stomach. If there is any doubt about correct endotracheal tube placement, all achievable indicators of correct endotracheal tube placement should be sought: direct visualization of the transglottic tube passage, auscultation of the epigastrium and the chest, measurement of end-tidal carbon dioxide concentration for a series of breaths, and bronchoscopy.

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Anesthetic Management of the Parturient with Systemic Lupus Erythematosus, Pulmonary Hypertension, and Pulmonary Edema

Joseph Cuenco, M.D.,* Gary Tzeng, M.D.,† Bernard Wittels, M.D., Ph.D.†

Coexistence of pulmonary hypertension with pulmonary parenchymal and vascular inflammation secondary to systemic lupus erythematosus (SLE) severely limits the cardiac and pulmonary reserves of parturients, predisposing mother and fetus to hypoxemia, and increasing the morbidity and mortality rates associated with anesthetic interventions. We report the successful anesthetic management of a parturient with pulmonary hypertension complicated by SLE pneumonitis and vasculitis, pulmonary edema, and severe orthopnea.

Case Report

Our patient, a 28-yr-old, gravida VII, para I, ab V, with an intrauterine pregnancy at 31 weeks' estimated gestational age, was admitted to the hospital by her pulmonologist. Her chief complaint was worsening dyspnea at rest, associated with paroxysmal nocturnal dyspnea, 3-5 pillow orthopnea, and bilateral lower extremity edema. She claimed to have no drug allergies. The surgical history was significant for a postpartum cholecystectomy 7 yr prior with general anesthesia that required postoperative ventilatory support for 7 days. Her medical records suggested that an acute, multilobe pneumonitis, combined with decreased respiratory effort secondary to upper abdominal post-

surgical pain, resulted in severe hypoxemia and was slow to resolve. A subsequent lung biopsy was diagnostic for SLE, lupus pneumonitis, and vasculitis. Moderately restrictive and severe interstitial lung disease developed and required immunosuppressive therapy from 1995-1997. Medications included home oxygen 2-4 l/min *via* nasal cannula and betamethasone 35 mg intramuscularly each day for 3 days. Antepartum pulmonary function parameters included total lung capacity of 2.67 l, forced expiratory volume in 1 s of 1.62 l, and DLCO 7.7 ml · min⁻¹ mmHg⁻¹, which were 48%, 51%, and 33% of predicted, respectively.

She tolerated minimal exercise with mild dyspnea until 27 weeks estimated gestational age, when she developed severe dyspnea at rest that required home oxygen therapy of 3 l/min during waking hours. Upon admission, she had severe dyspnea, tachypnea, and orthopnea at rest. Her blood pressure was 130/80 mmHg; maternal heart rate 120 beats/min; respiratory rate 40-45 breaths/min; temperature 36.1°C; weight 70 kg; and height 65 inches. Pulmonary examination revealed inspiratory crackles from the base to midthorax bilaterally; cardiac examination showed a regular, tachycardic rhythm with an S₄ gallop. Moderate, dependent, pretibial edema was present bilaterally despite a mild diuresis from furosemide. Arterial blood-gas analysis revealed pH 7.39, pO₂ 178 mmHg, and pCO₂ 52 mmHg while the patient received oxygen at 6 l/min by face mask. Compared with the patient's previous echocardiograms, a transthoracic echocardiogram on admission revealed increased right-atrial enlargement, new right-ventricular dilation, and higher pulmonary arterial pressure, estimated at 40-50/25-35 mmHg. Direct measurements revealed pulmonary arterial pressure of 48/25 with a mean of 32 mmHg. An anteroposterior chest radiogram was obtained prior to delivery and demonstrated bilateral pleural effusions, fluid in the fissure between the right middle and lower lobes, air bronchograms, and patchy infiltrates in all lung fields consistent with severe, diffuse, interstitial pulmonary edema. Despite declining maternal respiratory function, the fetus continued to have frequent spontaneous movements and a reassuring fetal heart rate pattern without decelerations.

After receiving metoclopramide 10 mg intravenously and sodium bicarbonate 30 ml orally, the patient moved onto the operating table in the semisitting position. We applied routine monitors (noninvasive blood-pressure cuff, five-lead electrocardiogram, continuous-pulse oximetry), transduced pressures from *in situ* radial and pulmonary arterial cath-

* Resident.

† Assistant Professor.

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Address reprint requests to Dr. Wittels: 5841 S. Maryland Avenue, MC 4028, Chicago, Illinois 60637. Address electronic mail to: wit7@acs-popmail.uchicago.edu

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