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## Technique of Avoiding Esophageal Burns

*To the Editor:*—Over the years, we have been cognizant that any electrical apparatus can act as a ground and be the source of electrical burns. Despite the fact that we have not recognized an esophageal burn in 2,000 open-heart procedures, we admire the candor of Dr. Edson O. Parker<sup>1</sup> in publishing this interesting and important report of esophageal burns.

Besides proper grounding, we have a simple solution to this problem. The esophageal temperature probe is inserted into the esophagus via a #6 endotracheal tube. (Our department has been doing this for years for ease of positioning of the probe.) The length of the tube is marked on the probe with tape. When temperature monitoring is needed, the probe is inserted a centimeter or two beyond the edge of the tube. This insures proper positioning of the temperature probe. The probe may be pulled back into the tube during periods of maximal

electrical activity, *i.e.*, opening and closing the chest, and repositioned when temperatures values are needed.

We feel that the benefit of temperature monitoring outweighs the risks if this is done.

F. ROBERT WEIS, JR., M.D.  
*Associate Professor*

ROGER E. KAISER, M.D.  
*Assistant Professor*

*Department of Anesthesiology  
State University of New York at Buffalo  
462 Grider Street  
Buffalo, New York 14215*

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## Delayed Onset of Laryngospasm-induced Pulmonary Edema in an Adult Outpatient

*To the Editor:*—We would like to report another case of laryngospasm resulting in pulmonary edema in an adult.

A 30-year-old, 60-kg G<sub>6</sub>P<sub>4</sub>Ab<sub>1</sub> woman with a 13-week intrauterine pregnancy presented to the ambulatory surgery unit for an elective dilatation and evacuation (D&E). Her medical history included a previous D&E under general anesthesia. She denied cardiovascular and respiratory system disease. She had nothing by mouth for 16 h. Initial blood pressure was 116/74 mmHg, heart rate 68 beats/min, temperature 37.1° C, and respirations 16/min. Hemoglobin concentration was 11.0 g/dl.

Atropine 0.3 mg iv and fentanyl 100 µg iv were administered. Oxygen (FI<sub>O<sub>2</sub></sub> = 1.0) was administered by face mask for 1 min, followed by sodium thiopental 275 mg iv, resulting in a smooth loss of consciousness. N<sub>2</sub>O 3 l/min, O<sub>2</sub> 2 l/min, and isoflurane 1% were administered by mask for maintenance. The patient tolerated positioning and examination under anesthesia, but dilatation of the cervix resulted in laryngospasm. Despite immediate administration of 100% O<sub>2</sub> and positive pressure, the patient could not be ventilated for 45 s.

The patient's heart rate decreased from 100 to 35 beats/min. Atropine 0.5 mg iv was administered and a succinylcholine infusion was started. The patient was intubated without difficulty. Suctioning revealed no evidence of regurgitant material in the pharynx or via the endotracheal tube. Breath sounds were clear bilaterally. The patient's heart rate returned to 100 beats/min, and the surgical procedure continued. The procedure was completed 20 min after intubation. With full return of muscle strength, the patient was extubated and brought to the recovery room.

One hour after arrival in recovery room the patient was fully awake and had no complaints. She had a respiratory rate of 20/min and her lungs were clear to auscultation. Total intravenous fluids were 1,100 ml of 5% dextrose in half normal saline. One hour and 25 min after arrival in the recovery room, she complained of dyspnea. She was tachypneic and coughing up pink frothy material. Rales were present over two-thirds of both lung fields. Breathing room air, her pH was 7.34, Pa<sub>O<sub>2</sub></sub> was 47 mmHg, and Pa<sub>CO<sub>2</sub></sub> was 40 mmHg. A chest radiograph revealed moderate pulmonary edema bilaterally. She was treated with furosemide 5 mg iv and

morphine sulfate 2 mg iv and 40% oxygen by mask, which resulted in a PaO<sub>2</sub> of 70 mmHg. She received an additional 20 mg furosemide and was admitted to the intensive care unit. Her pulmonary edema gradually cleared over 36 h. Upon discharge her chest radiograph showed complete clearing of the pulmonary edema, and her PaO<sub>2</sub> on room air was 98 mmHg.

In the two cases reported by Lee and Downes<sup>1</sup> and the cases detailed by Jenkins<sup>2</sup> and Cozantis *et al.*<sup>3</sup> laryngospasm occurred during emergence from anesthesia, and development of pulmonary edema was immediate. The case reported by Jackson *et al.*<sup>4</sup> occurred following inability to accomplish intubation during anesthesia induction, and signs of pulmonary edema developed within 20 min.

The case reported by Oswalt *et al.*<sup>5</sup> also occurred secondary to airway obstruction during anesthesia induction, necessitating an emergency tracheostomy. Signs of pulmonary edema were not manifested for 2 h, but this diagnosis was clouded by subsequent development of lobar pneumococcal pneumonia.

The mechanism for the development of postlaryngospasm pulmonary edema is said to be the large subatmospheric transpulmonary pressure gradients caused by attempts to ventilate against a closed glottis and/or hypoxia. These have been discussed in the previous case reports.

This case illustrates some important additional points. Laryngospasm can occur at any time during general anesthesia, not just during induction or emergence. Although this patient appeared well anesthetized for the pelvic examination, her depth of anesthesia was not appropriate for the strong stimulus engendered by cervical dilatation.

Although the patient subsequently was intubated for 20 min, there was no immediate development of pink frothy secretions and the lungs were clear to auscultation. Her clinical status did not deteriorate until 85 min after

arrival in the recovery room. Many patients undergoing short procedures with general anesthesia are discharged to home after 60–90 min in the recovery room. Because of the previous reports of pulmonary edema following laryngospasm, we had planned to monitor this patient's postoperative respiratory status for several hours, and this vigilance proved to be necessary.

A third point concerns her hospital course. She was given intravenous diuretics and supplemental oxygen, but reintubation was deemed unnecessary. Improvement was steady, with return of her cardiopulmonary parameters to preanesthetic values during the next 36 h. The "lesion" at the pulmonary alveolar–capillary interface needs time to resolve.

STEVEN A. GLASSER, M.D.  
*Assistant Professor of Anesthesiology*

JANET N. SILER, M.D.  
*Assistant Professor of Anesthesiology*

*Department of Anesthesiology  
Pennsylvania Hospital  
8th and Spruce Streets  
Philadelphia, Pennsylvania 19107*

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### Narcotic Analgesia—Ceiling Effect

*To the Editor:*—The letter from Eisele and Steffey<sup>1</sup> on the ceiling effect of narcotic analgesia is timely but difficult to resolve in the absence of any model for measurement of a very high intensity of analgesia in the human. The only apt relevant "experiment" is the use of narcotic analgesics as anesthetics for major surgery.

With partial agonist opioids, investigation is easier, as

the ceiling effect is lower, at a level of analgesia appropriate for treatment of severe postoperative pain. We believe that we have evidence for a ceiling effect for analgesia using nalbuphine, at a dose comparable with the ceiling effect for respiratory depression, in patients given access to doses up to 200 mg/h "on-demand."<sup>2</sup> This evidence is reinforced by our experience with the