References


Anesthesiology

Laser-induced Endotracheal Tube Fire

KATHRYN COZINE, M.D.,* LEE M. ROSENBAUM, M.D.,† JEFFREY ASKANAZI, M.D.,‡ STANLEY H. ROSENBAUM, M.D.§

The energy of a high-intensity monochromatic beam of light as produced by a laser is frequently used as a surgical tool. On contact with the tissues, the light energy is converted to heat which is used to resect tissue. If the laser beam strikes a flammable object such as an endotracheal tube (ETT), a fire may result. Such fires have produced either superficial tracheal burns or no significant injury.1,2 In those cases, direct ignition of the endotracheal tube occurred. Indirect ignition of the endotracheal tube can occur when the tube is ignited by burning pieces of tissue that lie next to the tip of the tube.3,4 In our case described below, a polyvinyl chloride (PVC) ETT wrapped with aluminum foil and wet cotton was used. Contact of the laser with the tube or cotton (which had dried) resulted in a fire and injury to both the trachea and pulmonary parenchyma.

Report of a Case

A 41-year-old man with carcinoma of the larynx was scheduled for a partial "laser" laryngectomy (Cavitron® infrared carbon dioxide laser, model AO 500). His past medical history was remarkable only for a 1 pack/day history of cigarette smoking. After premedication with 0.5 mg atropine, and 75 mg meperidine, im, anesthesia was induced with thiopental, fentanyl, diazepam, and succinylcholine. The trachea was intubated under direct vision with a polyvinyl chloride disposable endotracheal tube (N.C.C. 5 mm 1D) that had been wrapped above the inflatable balloon with aluminum foil. The tube was positioned carefully with the wrapped portion extending several centimeters above and below the cords. A moistened cotton surgical sponge tied to a long tape was placed above the balloon of the ETT to shield the balloon from direct damage by the laser.

Anesthesia was maintained with d-tubocurarine, nitrous oxide, and enflurane. The tube was periodically checked for position and the protective cotton sponge was moistened periodically with saline. One hour following induction of anesthesia, the metal Yankauer suction was noted to be hot. Within seconds thereafter, smoke was noted to be emerging from the patient's mouth. It soon became impossible to ventilate the lungs through the ETT. The procedure was stopped and the endotracheal tube replaced. Examination of the endotracheal tube revealed that the tip was fused shut and the cuff perforated. With replacement of the endotracheal tube, ventilation of the lungs could be reinstituted. There was no clinical evidence that severe bronchospasm had occurred.

During laryngoscopy no remaining fragments of the ETT were found. The remainder of the operation proceeded uneventfully. The tip of the removed endotracheal tube and part of the surgical sponge were charred.

Laryngoscopy and bronchoscopy were performed at the termination of the operation and revealed an absent left cord (surgically removed), a dark swollen right cord, and mild supraglottic edema but no apparent edema below the level of the cord. Breath sounds were clear; respiration was not labored. With a PaO2 of 100 per cent, pH was 7.35, PaCO2 49 torr, and PaO2 387 torr.

Three hours later upper airway obstruction secondary to severe supraglottic edema developed. An emergency tracheotomy was performed rather than endotracheal intubation because it was felt that there was a risk that complete obstruction could develop during laryngoscopy. The findings upon physical examination and chest roentgenographs were consistent with upper airway edema. The respiratory distress resolved following tracheotomy. After a 4-h period of observation in the recovery room, the patient was discharged to routine floor care.

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* Assistant Professor of Clinical Anesthesiology.
† Assistant Professor of Clinical Anesthesiology.
‡ Assistant Professor of Anesthesiology.
§ Assistant Professor of Anesthesiology and Medicine.

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Address reprint requests to Dr. Kathryn Cozine: Department of Anesthesiology, Columbia-Presbyterian Medical Center, New York City, N.Y. 10032.

Twelve hours postoperatively, copious, thick purulent secretions were noted. On physical examination he was comfortable despite diffuse coarse rhonchi auscultated over both lungs and an oral temperature of 38.6°C. A roentgenogram revealed bibasilar infiltrates and right lower lung consolidation with possible atelectasis. A gram stain of his secretions showed many leukocytes and gram positive cocci. The laboratory reported a leukocyte count on a peripheral smear of 8800 cells/mm³ (56 per cent polymorphonuclear cells, 19 per cent bands). Penicillin, 400,000 units, was given intravenously every 4 hours and vigorous chest physical therapy was instituted. On the second postoperative day his leukocyte count dropped, inexplicably, to 3300 cells/mm³ (with a normal differential); his body temperature rose to 38.9°C, with a FiO₂ of 30 percent, pH was 7.39, PaO₂ 34 torr, and PaCO₂ 70 torr. Abundant, thick secretions were being suctioned out of his tracheostomy tube continually. On the third postoperative day his respiratory status acutely deteriorated. Secretions were now so thick that they were difficult to remove via suction, with an FiO₂ of 30 percent, pH was 7.49, PaO₂ 34 torr, and PaCO₂ 57 torr. The patient was transferred to the intensive care unit where fiber-optic bronchoscopy performed via the tracheostomy showed diffuse erythema and edema of the tracheal, carinal and proximal bronchial mucosa, white plaques along the more inferior tracheal wall which were not removable with suctioning, and diffuse thick secretions throughout all the major airways. The chest roentgenogram showed scattered small infiltrates as well as bilateral lower lung densities. The clinical impression was that the patient had a diffuse pulmonary burn. Mechanical ventilation was begun, and the antibiotics were changed to high doses of oxacillin when the initial sputum culture grew out S. Aureus. His leukocyte count was 4000 cells/mm³. Over the next several days he remained febrile to 39.4°C. Vigorous pulmonary toilet was required, as well as therapeutic bronchoscopies on the fourth and sixth postoperative days to reexpand a collapsed right lower lobe. On the seventh postoperative day weaning was begun from the mechanical ventilator using intermittent mandatory ventilation. His white blood cell count rose gradually to 6800 cells/mm³. His chest roentgenogram showed bilateral markings. Additional bronchoscopies were required on the seventh and ninth postoperative days to remove secretions and open atelectatic areas of the lungs. On the tenth postoperative day the secretions began to lessen, and he was weaned completely from the mechanical ventilator.

At this time body temperature remained below 37.8°C. The tracheostomy was cuffed intermittently on postoperative days 12 to 23. Laryngoscopy on postoperative day 23 demonstrated a hypertrophic epiglottis but otherwise resolution of the acute problem, and the tracheostomy was removed. One month following the operation, the patient was discharged.

Six months following the first operation, the patient underwent a total laryngeal resection because of extension of his primary carcinoma. The trachea at this time was fibrous and contracted, possibly secondary to the prior burn injury.

**DISCUSSION**

Infrared radiation increases the temperature of absorbent tissue and materials. All flammable objects must be kept out of the line of projection of the laser. Several techniques have been devised for preventing endotracheal tube fires which include use of metal tubes, wrapping tubes with heat resistant tape or wet cotton, and use of venturi (jet) ventilation. In our case, a polyvinyl chloride endotracheal tube was wrapped with aluminum foil. Since wrapping the cuff with foil would prevent its expansion, the cuff was protected with wet cotton. The fire could have resulted from the laser striking a portion of the tube that became unprotected or drying of the cotton leading to ignition by the laser. There are a number of additional problems reported with wrapping of the endotracheal tube with foil. Wrapping of the endotracheal tube with foil may predispose to kinking. The foil may become loose from the tube and lead to airway obstruction. To circumvent these problems a stainless steel endotracheal tube has been developed. Venturi ventilation has also come into use as an alternative.

This case differs from other reports of endotracheal tube fires in that there was extensive injury to both the airway and parenchyma of the lung. Hydrochloride gas is a known product of PVC combustion and a respiratory irritant. The complications described in our case may have in part been due to hydrochloride gas, smoke inhalation or direct heat injury. Smoke inhalation has been reported to result in a decrease in pulmonary compliance due to a decrease in surfactant. Pulmonary edema is not a universal finding immediately following smoke inhalation. Hence the chest roentgenogram of victims of smoke inhalation may be clear initially, as was the case in this report. The clinical course and pathological lesions reported in this present case (diffuse bronchitis with bronchorrhea) are consistent with smoke inhalation, and decrease of surfactant with subsequent pulmonary collapse and infection. Thus, the clear initial chest roentgenogram seen following the events reported should not have been construed as an indication of absence of pulmonary injury. The edema of the upper airway and the whitish plaques along the lower portion of trachea were probably due to direct flame injury. Tracheal stenosis was a late complication of this injury.

**REFERENCES**

The Failure of Nasal Plethysmography to Estimate Cerebral Blood Flow during Carotid Occlusion

ROY F. CUCCHIARA, M.D.,* AND JOSEPH M. MESSICK, M.D.*

There are several ways of monitoring the adequacy of cerebral blood flow during carotid endarterectomy. The electroencephalogram (EEG) is the standard functional monitor of cerebral ischemia. Various computerized EEG signals (e.g., compressed spectral array) and filtered EEG signals (e.g., cerebral function monitor) have been developed. Measurements of cerebral blood flow (CBF) directly with radioactive tracers (e.g., xenon) or by inference via pressure (e.g., stomp pressure measurements) have also been used to detect the occurrence of cerebral ischemia during carotid artery occlusion for endarterectomy. The anterior ethmoidal artery is a distal branch of the internal carotid artery and emerges into the nose high along the septum. By measuring pulsations of the ipsilateral anterior ethmoidal artery during carotid occlusion, CBF to the affected hemisphere can be estimated by whatever collateral circulation might be present. This work examines the effectiveness for the light-emitting-diode nasal plethysmograph† as a method of estimating CBF during carotid artery occlusion.

METHOD

Nineteen patients undergoing carotid endarterectomy with EEG monitoring and CBF determinations by $^{133}$Xe washout were studied. This study was approved by our local Committee on Human Research. The nasal probe was covered with its disposable plastic condom and inserted high along the nasal septum until a clear large pulse wave could be recorded with a Gould® 2400 pen recorder.

Pulse height was continuously recorded before carotid occlusion, during occlusion of the external carotid, during occlusion of both external and internal carotid, and after restoration of flow in both vessels. Pulse height with the external carotid artery occluded was used as a control value, and the pulse height during internal carotid occlusion was used to calculate percent change in pulse height. Simultaneous CBF measurements were made in these two circumstances. Normocarbia was maintained and anesthetic circumstances (enflurane/N₂O/pancuronium) were in a steady state during the measurements.

RESULTS

The change in pulse height is compared to CBF in figure 1. There is no close correlation between the two. Furthermore, the patients who exhibited critically low CBF (<18 ml/100 g/min) and EEG changes during carotid occlusion could not be identified by a characteristic change in the nasal plethysmograph. Thus, the nasal plethysmograph failed to estimate reductions in CBF and failed to help identify patients with critically low CBF producing EEG changes of ischemia.

The only technical problem encountered with the device was the occurrence of epistaxis in five patients probably due to the systemic heparinization associated with this surgical procedure rather than the device or its placement.

DISCUSSION

Otorhinolaryngologists and anatomists have long appreciated the rich anastomotic vasculature of the nasal mucosa. Attempts to reduce the difficulty and increase the success rate for surgical treatment of severe arterial epistaxis have been the subject of many papers. We