Loss of Fiberoptic Laser Tip

To the Editor:

The Nd:YAG laser has become accepted therapy for the relief of tracheobronchial obstruction resulting from both benign and non-removable malignant lesions. Reported complications include hemorrhage, mucosal edema, hypoxemia, pneumothorax, perforation, myocardial infarction, necrotizing pneumonia, and ignition of the fiberoptic bronchoscope sheath and the endotracheal tube. Recently, we experienced an apparently unreported complication: loss of the tip of the laser fiber.

An 82-year-old female was referred to the Cleveland Clinic Foundation because of stridor. Her past history was one of severe restrictive and obstructive lung disease (marked kyphoscoliosis and cigarette smoking) and respiratory failure which had required temporary tracheostomy, mechanical ventilation, and a protracted period of weaning. She presented in moderate respiratory distress with inspiratory stridor, wheezing, and increased use of the accessory muscles of respiration. Fiberoptic bronchoscopic examination showed granulation tissue obstructing 90 percent of the tracheal lumen 2.5 cm below the vocal cords. Nd:YAG laser photoablation was performed with a fiberoptic bronchoscope. Local anesthesia was used due to the location of the lesion and the severity of the underlying lung disease. The obstruction was successfully reduced to 20 percent of the tracheal lumen. An FiO₂ of 1.0 was administered by face mask, and decreased to 0.4 when the laser beam was in use. A total of 11,061 J was delivered to the tissue in 481 pulses. Initially, energy levels of 40 W for 0.4 sec duration were used. Later, in an attempt to vaporize portions of the lesion, 60 W for 0.7 sec were used and a carbon particle on the tip of the laser fiber ignited. As the fiber casing dilated from the heat, the fiber tip was lost. Its location in the left endobronchial tree was confirmed by chest x-ray examination (Fig 1). Multiple attempts at endoscopic retrieval were unsuccessful. The patient suffered no adverse effects from the brief ignition or loss of the fiber tip.

This complication can be avoided by frequent, meticulous cleaning of the laser fiber tip during the procedure. In addition, placing the fiber tip at least 0.5 cm beyond the end of the bronchoscope and at least 0.5 cm from the target, using the lowest FiO₂ compatible with satisfactory oxygenation (preferably 0.5 or less), limiting the power to 45 W and pulse duration to 0.5 sec should lessen the likelihood of both losing the laser fiber tip and igniting the bronchoscope sheath and endotracheal tube.²

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REFERENCES


Hyperventilation and Cerebral Protection

To the Editor:

The review article by Dr. McGillicuddy (Chest 1985; 87:85-93) concerning cerebral protection and treatment of increased intracranial pressure is helpful and relevant to practicing physicians. However, I would like to make the following points.

I think greater clarity is achieved if we consider cerebral flow to be controlled by the cerebral tissue pH balance as affected by changes in arterial CO₂ levels, rather than looking at the CO₂ level alone.² This avoids certain clinical problems. For example, if we follow Dr. McGillicuddy's admonition to hyperventilate a patient to a PCO₂ level of 20 to 25, then we perform a disservice to the chronic obstructive pulmonary disease patient with compensated respiratory acidosis and a near normal pH (arterial as well as central nervous system) at a PCO₂ level of 55 or 60, compensated by an elevated bicarbonate as a buffering modality. Hyperventilation to a PCO₂ level of 20 or 25 will produce marked cerebral alkalosis with cerebral vasocostriction and cerebral hypoxemia in this patient. The opposite end of this clinical spectrum is represented by patients with persistent metabolic acidosis, as encountered in chronic renal disease. If such a patient is hyperventilated with the existing reduced bicarbonate store, the results at a PCO₂ level of 20 to 25 will not be the same as in a patient with a normal bicarbonate store. Hyperventilation has to be administered within the context of the patient's acid base status, rather than trying to achieve a standard PCO₂ level of 20 to 25.

When hyperventilating a patient, consideration must be given to alterations in oxygen transport due to cerebral vasocostriction and the Bohr shift. Both phenomena could cause cerebral hypoxemia.² Some effort should be made to monitor internal jugular venous effluent. Although clinically difficult to monitor on a continuous basis, random sampling would guarantee adequate oxygen transport. In lieu of this variable, I agree with Dr. McGillicuddy's suggestion to maintain arterial oxygen tension at higher physiologic values.
values.

Recognition of the phenomena of central nervous system acidosis, which has been described in a variety of comatose states,4 would prompt measurement of cerebral spinal fluid pH level as an additional variable to be monitored to determine the success of hyperventilation.

Finally, the inverse steal phenomenon has to be considered when dealing with increased intracranial pressure due to focal brain damage.5 Areas of injured brain tissue may lose vascular autoregulation. There is then a potential, when hyperventilating this type of patient, to cause vasoconstriction of normal brain tissue with no response from vessels in the injured tissue area. Blood flow would be shifted away from normal tissue with luxury perfusion of damaged cerebral tissue. Such a phenomenon could create ischemia in normal brain tissue. Recognizing this possibility, it might be prudent to choose other therapeutic modalities, before hyperventilation, to reduce intracranial pressure in the patient presenting with focal brain damage.

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To the Editor:

I appreciate Dr. Reid's letter responding to my review article. The letter makes a number of very valid points regarding the action of carbon dioxide and the bicarbonate ion in the regulation of cerebral blood flow (CBF). The possibility of cerebral ischemia certainly exists when Pco2 is decreased dramatically, at least in normal control subjects. In patients with head injury and widespread loss of autoregulation due to cerebral vasodilatation, the situation is less clear. It must also be remembered that a rather large decrease in CBF may be tolerated without developing brain ischemia. The normal flow of approximately 50 ml/100 g/min can be lowered to 20 ml/100 g/min before EEG changes occur.

While the monitoring of jugular venous Pco2 and CSF pH levels add an element of safety to the use of hyperventilation, the present inability to monitor these values continuously limits their usefulness. We have not monitored these parameters at our institution, but employ frequent analysis of arterial gas levels and attempt to maintain a Pco2 level in the high normal range.

The inverse steal phenomenon is real, but in a brain with large areas of autoregulatory loss, and a high likelihood of ischemia therein, the importance of this steal is unknown. There is little evidence of clinical neurological deterioration due specifically to hyperventilation and steal in these patients.

Dr. Reid has pointed out the dangers of hyperventilation and they are to be respected. Nonetheless, these problems are of a lesser severity than the complications due to the use of ventricular drainage or osmotic diuretics. The complications of hypocarbia can be fairly rapidly reversed—a distinct contrast to the difficulties of dealing with hyperosmolality and hypernatremia following the prolonged use of osmotic diuretics.

Despite the dangers clearly outlined by Dr. Reid, hyperventilation is the safest means of reducing increased intracranial pressure and, at least for the present, it is the central modality in the treatment of this serious and frequently lethal problem.

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Reproduction of Original Scientific and Educational Material

To the Editor:

A relatively minor nuisance for those concerned with writing comprehensive review articles, books and book chapters is the inability to freely reuse the fruits of one's own labor, particularly illustrations and tables. This is a proposal to remedy the situation.

When illustrative and other materials appear in print, the copyright traditionally resides in the hands of the publisher of original journal articles and most books. When one wishes to reproduce illustrations and explanatory diagrams—originated and paid for by the authors—explicit permission must be sought from the editor and publisher for each item, with some publishers requiring precise details of where the material will appear. Since it is virtually inconceivable that publishers and editors would reuse an author's material, in spite of owning the rights to it, this seems to be an anomalous situation. Morally, rights to original material should belong to its creators. It is annoying to continually solicit permissions from journal editors and publishers.

Relatively recently, editorial offices have required from authors specific assignment of copyright for every manuscript—including letters to the editor—through a form for each author to sign upon acceptance of the material. (Formerly, transfer of copyright was merely assumed.) I propose that acceptance of a manuscript produce an additional form to be exchanged simultaneously with the assignment of copyright. This form would give blanket permission from the editor and publisher for authors to reproduce their own illustrations and tables with, of course, citation of the original publication.

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Assisted Mechanical Ventilation with the Servo Ventilator

To the Editor:

We were interested to read the observations by Dr. Marini and colleagues (CHEST 1985; 87:612-17) with regard to the Servo ventilator 900C (Siemens-Elema AB, Solna, Sweden). They indicate that, when the ventilator is used to provide assisted ventilation (volume-controlled mode), inappropriately slow gas delivery can occur when the patient's actual minute ventilation exceeds that of pre-set values. Their data, collected in normal volunteers, suggests that this slow gas delivery is deleterious, resulting in a significant increase in the