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Did Anesthetic Mismanagement Contribute to Intraoperative Death? I.

To the Editor:—The information provided concerning the dose of epinephrine contained in gingival retraction cord provided by Hilley *et al.*[1] is important to recognize, but I believe the quality of anesthetic care was substandard.

To allow spontaneous ventilation during approximately 2 MAC inhaled anesthetic (1% halothane + 70% nitrous oxide) for almost 3 h is probably not a wise idea. The degree of hypoventilation with consequent hypoxemia, hypercarbia, combined acidosis, and organ damage can only be surmised. To not have recorded, or possibly not monitored, blood pressure is inexcusable.

Note is made of the fact that "heart rate and respiratory rate remained at 80 bpm and 20 breaths · min^{-1}, respectively, during the first two hours of the procedure." What were the values between the first 2 h and the incident? How was heart rate determined? If by reading it off of the oscilloscope, the patient may have been dead long before the ventricular ectopy was noted.

How long after the arrest was the analysis of arterial blood gases obtained? If early, it supports my feeling that this patient had significant myocardial depression, systemic hypoperfusion, hypoxemia, and tissue acidosis (as evidenced by the pH_a of 7.19 after two ampules of bicarbonate and hyperventilation with $Fi_{O_2} = 1.0$).

Although the induction medications were given iv, was there an iv running throughout the procedure? I am led to believe that the patient did not have an iv line running because mention of the drugs administered for resuscitation follows "percutaneous cannulation of the subclavian vein via the external jugular approach was accomplished," which occurred after the arrest. If this be true, how can you justify a lengthy intraoral procedure under general anesthesia without an iv?

The authors state in their second paragraph "We describe a fatality that resulted from the combined use of halothane and gingival retraction cord impregnated with 8% racemic epinephrine." I do not feel that they have proven their case. The fatality appears to have been due to anesthetic mismanagement, with the ventricular fibrillation following application of an overdose of epinephrine as only the final chapter in the tragic death of this healthy ASA I patient.

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REFERENCE

1. Hilley MD, Milam SB, Giescke AH Jr, Giovannitti JA: Fatality associated with combined use of halothane and gingival retraction cord. 60:587–588, 1984

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Did Anesthetic Mismanagement Contribute to Intraoperative Death? II.

To the Editor:—In a recent issue Dr. Hilley *et al.* report a fatality associated with combined use of halothane and gingival retraction cord.[1] While alerting readers to the presence of l-epinephrine in the gingival retraction cord and reaffirming the arrhythmogenic potential of exogenous epinephrine in the presence of halothane anesthesia, the authors fail to note that the case illustrates the importance of meticulous intraoperative monitoring and record keeping.

Since blood pressures were not charted, the reader must assume that this basic and essential monitoring modality was not carried out during the case. In addition, the report does not document changes in respiratory or cardiac rate and rhythm prior to the onset of cyanosis, labored breathing, and ventricular fibrillation. While the combination of exogenous epinephrine and halothane anesthesia probably contributed to this patient's demise, the exclusive cause cannot be determined without detailed knowledge of the patient's status during the 10-min interval from the placement of gingival retraction cord to the onset of ventricular fibrillation.

It is our belief that even though there may be an unawareness of potentially dangerous drug interactions, *i.e.*, exogenous epinephrine in the presence of halothane anesthesia, or unfamiliarity with certain drug preparations, *i.e.*, l-epinephrine in gingival retraction cord, early recognition of changes in a patient's vital signs