CORRESPONDENCE

False-positive Abrupt Decrease in EtCO₂ during Craniotomy in the Sitting Position

To the Editor: Capnography, the measurement of end-tidal carbon dioxide tension (EtCO₂), often is used for the detection of venous air embolization. During posterior fossa craniotomy in a 26-year-old man for resection of an arteriovenous malformation, the was abrupt drop in EtCO₂ from 22 to 16 mmHg, recorded on a Hewlett Packard® 47210A capnometer, despite no change in precordial doppler tone. The surgeons were alerted but could find no site of air entry. Nitrous oxide was discontinued, and a stable plane of anesthesia was maintained with isoflurane in oxygen. EtCO₂ remained constant at this lower level, and the patient exhibited no hemodynamic changes. Visual inspection of the airway CO₂ tracing as well as auscultation with the esophageal stethoscope revealed no airway disconect. When the drapes were removed at the end of the procedure, an incomplete displacement between the capnometer sensor and the sample cell on the airway adapter was noted (fig. 1).

The theoretic basis for the operation of the Hewlett Packard® 47210A capnometer is the measurement of the amount of infrared light emitted from the unit's black-body source that is absorbed by CO₂ within the sample chamber. Any portion of infrared light that reaches the unit's detector without first passing through the CO₂ containing sample will have none of its light absorbed, resulting in a falsely low reported EtCO₂. Such was the situation reported here, whereby a partial displacement of the capnometer sensor from the sample cell resulted in an abrupt fall in apparent EtCO₂, indicating a false-positive air embolus. This technical error may be averted by simply securing these two modules with a rubber strap or plastic tape.

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Methemoglobinemia and Respiratory Failure

To the Editor:—We read with interest the case report by Zurick et al., describing the occurrence of methemoglobinemia and respiratory failure following nitroglycerin infusion. We would like to make two comments. First, as stated by the authors, nitroglycerin-associated methemoglobinemia has been well described and may have played a role in their patient's methemoglobinemia. The authors acknowledge, however, certain reservations: the relatively low dose of nitroglycerin, the short duration of the infusion, and the time lag between its discontinuation and the methemoglobinemia. An alternative explanation would consider a possible contribution of nitroprusside, which the patient had received for 25 h following surgery. Nitroprusside-associated methemoglobinemia has been described by Bower and Peterson. It is not known whether the consecutive use of nitroglycerin and nitroprusside is more likely to be associated with methemoglobinemia than either agent alone.

The second observation concerns the etiology of the respiratory distress of Zurick's patient. We agree with
the authors that the methemoglobinemia may have contributed to the respiratory distress. Alternatively, methemoglobinemia and respiratory failure both may have been due to the same factors. In effect, the oxidation of hemoglobin into methemoglobin is believed to be due to oxygen-derived toxic radicals (hydrogen peroxide, superoxide anion, singlet oxygen and hydroxyl ion). The same activated species of oxygen are also thought to play a key role in the pathogenesis of adult respiratory distress syndrome (ARDS). The simultaneous occurrence of ARDS and methemoglobinemia, to our knowledge, has not been reported. Certain conditions, however, such as paraquat intoxication and oxygen toxicity that are typically associated with ARDS recently have been linked to methemoglobinemia. Furthermore, we recently had a patient with ARDS who, incidentally, was found to have significant methemoglobinemia and evidence of severe tissue hypoxia.

Thus, in addition to impairing oxygen-carrying capacity of the blood, methemoglobinemia either may predispose a patient to acute respiratory failure, as suggested by Zurick et al., or share with it the same pathophysiology, namely the toxic action of the oxygen-derived radicals.

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Another Example of Hypoxic Gas Mixture Delivery

To the Editor:—Delivery of a hypoxic gas mixture due to mechanical failure of the oxygen supply system to an anesthesia machine is a serious problem. Many causes of this type of mishap have been reported. I would like to bring to the attention of practicing anesthesiologists another variation causing this potential disaster.

An anesthesiologist, working in an operating room suite equipped with a central supply of oxygen and a central supply of nitrous oxide commenced administering general anesthesia for an intraabdominal procedure. After an intravenous induction, the intention was to maintain the patient on a 50:50 nitrous oxide:oxygen gas mixture at a total flow rate of 4 l/min. Upon opening the needle valve on the nitrous oxide flow meter, it appeared that a much greater than usual number of turns had to be used to reach a nitrous oxide flow of 2 l/min. The bobbin in the flow meter of nitrous oxide was rotating well, with no suggestion of it sticking in the column. After several minutes, it was noted that the bobbin of the nitrous oxide flow meter suddenly was rising in the column without further adjustment to the valve, and within a few seconds, it indicated a nitrous oxide flow rate of 8 l/min. The oxygen flow meter was still at 2 l/min. The nitrous oxide flow immediately was turned back to 2 l/min and kept under constant observation for the rest of the procedure. A later check with the hospital engineer showed that, by an unfortunate set of circumstances, the central supply of nitrous oxide was nearly exhausted right at the moment that the gas mixture was started. During the procedure, the central supply switched from main to standby operation, and the line pressure shot up accordingly.

In order to prevent this problem from recurring in