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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, there was an 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 disconnect. 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,

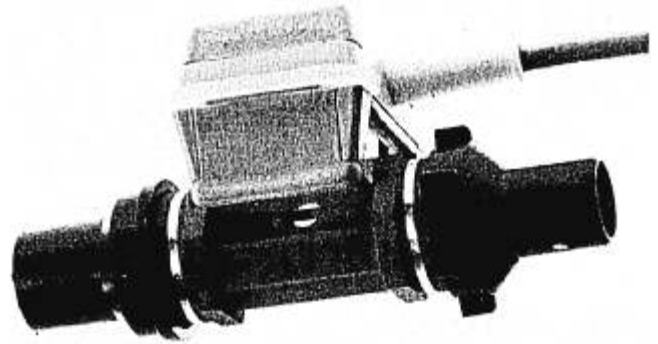


FIG. 1. Incomplete displacement between sensor (above) and sample cell (below) leading to abrupt decrease in measured CO₂.

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.*,¹ 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^{2,3} 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 discontin-

uation 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 Peterson⁴. 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