mmHg.) In addition, dissolved carbon dioxide accounts for only about five per cent of the total carbon dioxide in the venous blood.[5] Therefore it seems unlikely that any carbon dioxide bubbles would remain in the circulation more than two hours after the embolism occurred, the time when the patient described underwent HBO.

On balance I feel that the hazards associated with the transport of a critically ill patient outweigh the questionable benefits of HBO following carbon dioxide gas embolism.

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REPLY

We appreciate Dr. Etches' comments regarding our case of carbon dioxide embolism. We agree with his comments regarding the differences in solubility between nitrogen and carbon dioxide, but not with his conclusion that carbon dioxide is unlikely to be present in the circulation more than two hours after embolization. While there are no prospective studies examining this issue, we believe that a sufficiently large volume of embolized carbon dioxide can persist in the vascular system despite its high solubility. The report by Root et al. *which we referred to in our article describes a patient who sustained a cardiac arrest 35 min after instillation of carbon dioxide.[1] According to their report, resuscitative efforts were continued for at least 45 min after the arrest, a time lapse of 75 min from the time of gas insufflation. Despite the interval from carbon dioxide instillation to death, a large quantity of gas was found in the heart, pulmonary arteries, cerebral vessels, peritoneal cavity and portal venous system at post mortem examination. Trapping of gas in the portal venous system was the proposed reason for the delay in onset of cardiac arrest. The report of Vourc'h* et al. *describes a case of dramatic reversal of coma and myocardial ischaemia by hyperbaric oxygen therapy initiated two hours after carbon dioxide embolism.[2] They attributed this response to hyperbaric oxygen's effect on residual gas in the body. While we cannot prove that our patient's improvement was due to hyperbaric therapy, we believe that it has a role in cases of massive carbon dioxide embolism.*

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Airway obstruction during anaesthesia in a child with a mediastinal mass

To the Editor:

A 12-year-old 30 kg girl was admitted with a cervicomediastinal tumour. On admission, she was not dyspnoeic or cyanosed. Multiple bilateral cervical nodes were palpable. Cardiac examination and ECG were normal. Breath sounds were coarse in both lung fields. Chest *x*-ray showed a right lobular mass and tracheal deviation to the left. A right supraclavicular lymph node biopsy was planned to make a definitive diagnosis. Diazepam 10 mg and atropine 0.25 mg were given intramuscularly one hour before induction. After preoxygenation general anaesthesia was induced with fentanyl 50 μg, thiopentone 250 mg and vecuronium 3 mg. The trachea was intubated with a 5.5 mm cuffed tube and ventilation was mechanically controlled (VT = 10 ml · kg^{-1}, Freq: 14). Anaesthesia was maintained with enflurane 0.8 per cent and nitrous oxide 50 per cent in oxygen. After 20 minutes, the biopsy had been performed and the child was turned to the left lateral position to allow a right iliac bone crest marrow biopsy. Inspiratory pressures increased from 30 to 40 cm H_2O. When the biopsy was performed it was decided to allow her to breathe spontaneously but she developed severe respiratory distress with laboured inspirations. Breath sounds were inaudible in both lung fields. It was possible to maintain ventilation only with very high inflation pressures and manual ventilation. Superficial neck veins appeared distended. Chest *x*-ray showed atelectasis of the right upper lobe and widening of the mediastinum. Treatment with hydrocortisone hemisuccinate 100 mg and erythromycin was started. A fibreoptic tracheoscopy through the tracheal tube revealed a