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.

Richard C. Etches MD FRCPC
Department of Anaesthesia
University of Alberta Hospital
Edmonton

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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.*

Brian McGrath MD
Department of Anesthesiology
The George Washington University
Washington, D.C.

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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

constriction 2 cm above the carina. The trachea was extubated and reintubated with a longer tube (Portex no. 5) which passed beyond the constriction, improving ventilation. Histological examination of the cervical node biopsy confirmed the clinical diagnosis of Hodgkin's disease. Seven hours after the event cytotoxic chemotherapy was begun and continued for seven days with vincristine 1.8 mg and chlormethine 7 mg. The next day procarbazine (250 mg/m^2) was given. Forty-eight hours later, chest *x*-ray revealed no further atelectasis and a marked decrease in the size of the mediastinal tumor. The trachea was extubated successfully.

This case illustrates the potential hazard of general anaesthesia in children with an anterior mediastinal mass leading to airway obstruction. Dynamic pulmonary function should be assessed preoperatively with standing and supine flow volume loops.[1,2] If the risk of obstruction is high, general anaesthesia should be avoided; biopsy under local anaesthesia would be preferable. If general anaesthesia is employed, certain approaches are recommended:[3] maintenance of spontaneous ventilation, induction with head-up position, avoidance of muscle relaxants, readiness to change the patient's position rapidly to a lateral or prone position, availability of a rigid bronchoscope and other airway support equipment.

F. Montange MD
J. Truffa-Bachi MD
E. Pichard MD
Department of Anesthesiology
Institut Gustave-Roussy
39, rue Camille Desmoulins
94805 Villejuif Cedex France

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Sporadic PCA pump failure accompanied by activation of a "fail safe" mechanism

To the Editor:

In the management of postoperative pain following total knee arthroplasty with patient-controlled analgesia (PCA), pump failure has occurred in six of 27 consecutive patients enrolled in a research study (22.2 per cent). Treatment was started in the Post Anaesthesia Care Unit (PACU), where the Bard Harvard PCA pump* mounted on a dedicated mobile stand and equipped with a printer interface was programmed to deliver morphine 1.0 mg with a delay of six minutes, into an intravenous infusion. During transfer to the ward the pump was powered from its internal battery. Pump failure occurred three times following the patient's transfer to the surgical ward immediately the external power cable was plugged into the 110V electrical outlet, and sporadically during treatment of the other three patients.

Investigation of the problem revealed that the pump did not respond to the patient control button, the display panel contained the self check messages "RAM-OK CTC-OK INT-OK RTC-OK ROM-OK," and the keyboard was frozen. Using the printer interface, it was possible to obtain a print-out of the pump's performance prior to its failure, but the same information could not be obtained from the keyboard. The pump was reactivated by switching the power switch off, then on again, and then reprogramming. The memory of the interface was erased by switching the power off. The problem has been experienced by other members of the acute pain management service and has occurred with each of the six Bard Harvard PCA pumps being used.

In the light of this experience it is now my practice to obtain a record of the pump's performance immediately before the patient's transfer from the PACU. On arrival at the ward, immediately after the external power cable is reconnected the pump's performance is checked, and reprogrammed if necessary by the PACU nurse who is trained and certified to do so.

A.C. Webster BSc MB ChB FFARCS FRCPC
Department of Anaesthesia
St. Joseph's Health Centre
London, Ontario

*Bard Canada Inc., 2345 Stanfield Road, Mississauga, Ontario, L4Y 3Y3