Correspondence

Severe hypercarbia with a Bain breathing circuit during malignant hyperthermia reaction

To the Editor:

With better understanding of the rebreathing characteristics of T-piece systems in the last decade,[1-5] these circuits have been found useful for controlled ventilation to obtain a desired arterial $PaCO_2$ by means of controlled rebreathing. Arterial $PaCO_2$ becomes fresh gas flow-dependent when the minute ventilation to fresh gas flow ratio exceeds 1.5 during controlled ventilation with T-piece systems.[5] Indeed, factors determining alveolar CO_2 tension (and hence arterial CO_2 tension) can be expressed by the following equation:

$$F_ACO_2 = F_ICO_2 + \dot{V}CO_2/\dot{V}_A$$

where F_ACO_2 is the alveolar carbon dioxide tension; F_ICO_2 is the inspired carbon dioxide tension; $\dot{V}CO_2$ is CO_2 production in $ml \cdot min^{-1}$; $\dot{V}_A$ is minute alveolar ventilation; f = 0.7 (fractional utilization of fresh gas) when $\dot{V}_E/FGF > 1.5$.

Recently in our institution, a malignant hyperthermia reaction in a patient ventilated with a Bain circuit, resulted in severe hypercarbia, due to the unrecognized increase in CO_2 production ($\dot{V}CO_2$), aggravated by rebreathing due to the breathing circuit.

The patient, a 45 kg, 21-year-old female, was scheduled for elective left frontal parietal craniotomy for tumour. Past medical history included mild intermittent asthma and a seizure disorder related to her brain tumour. Previous anaesthetics for diagnostic laparoscopy and dilatation and curettage were unremarkable. Preoperative medications consisted of diphenylhydantoin and dexamethasone. No preoperative sedation was given. Upon arrival in the operating room intravenous, intra-arterial and central venous pressure catheters were placed under local anaesthesia. After preoxygenation, induction of anaesthesia was carried out with fentanyl 100 μg, tubocurarine 2 mg, thiopentone 250 mg and succinylcholine 100 mg IV. The patient was intubated and anaesthesia was maintained with 60 per cent nitrous oxide/40 per cent oxygen, 1–1.5 per cent isoflurane, pancuronium and fentanyl.

An Ohio Modulus anaesthetic machine equipped with an Ohio 7000 ventilator was used. The patient was ventilated with a Bain circuit with a fresh gas flow of 4.5 $L \cdot min^{-1}$ and minute ventilation of 8.4 $L \cdot min^{-1}$. Proper Bain circuit function was verified by pressurizing the circuit and performing the Pethick test.[6] The nitrous oxide and oxygen flow meters allowed maximum flows of 12 $L \cdot min^{-1}$ of each. Arterial blood gas analysis 45 minutes after induction revealed pH 7.35, $PaCO_2$ 38, PaO_2 203 ($PaCO_2$ was slightly elevated above predicted levels). Fresh gas flow was increased to 5 $L \cdot min^{-1}$ and minute ventilation to 11 $L \cdot min^{-1}$. In the next 45 minutes the patient had a persistent heart rate of 115 bpm and a gradual rise in axillary temperature from 36 to 38° C. During the subsequent 15-minute interval a second anaesthetist's opinion was requested while other causes for the hyperthermia were discussed with the surgeon. The temperature was then noted to be 39° C. Diagnosis of a probable malignant hyperthermia (MH) reaction was made, because no other cause for the hyperthermia was evident. Blood gas analysis at this time revealed pH 6.91, $PaCO_2$ 121, PaO_2 72. Our therapy for the suspected MH reaction included topical cooling, dantrolene 100 mg IV, sodium bicarbonate 100 mg intravenously and attempted hyperventilation with a fresh gas flow of 12 $L \cdot min^{-1}$ of oxygen (maximum for this machine) with minute ventilation of 18 $L \cdot min^{-1}$ using the existing Bain circuit.

A calibrated capnograph was then added to the system and an end-tidal PCO_2 of 75 mmHg was measured from the proximal end of the endotracheal tube. Inspiratory PCO_2 was elevated but the number not recorded. After substitution with a circle system with fresh soda lime, the end-tidal PCO_2 immediately fell to 35 mmHg at a minute ventilation of 15 $L \cdot min^{-1}$ and fresh gas flow of 10 $L \cdot min^{-1}$ oxygen. The Bain circuit was not visibly damaged but was not retested. The patient's temperature peaked at 39.8° C but fell rapidly to 37.3° C over 45 minutes. Nasal and rectal temperature probes confirmed the hyperthermia and were used to follow the course of the suspected MH reaction. Postoperative recovery was subsequently uncomplicated and serum CPK levels revealed a moderate rise only.

We feel this case emphasizes several points. In usual circumstances normocapnia can be achieved with use of a T-piece system by controlled rebreathing.[5] However, hypercarbia may result when $\dot{V}CO_2$ is markedly increased.

According to the above formula, F_ACO_2 increases as a result of both increased $\dot{V}CO_2$ and F_ICO_2 in a T-piece

system. In a non-rebreathing system $\dot{V}CO_2$ is the only variable affected and the hypercapnia which occurs is not aggravated by rebreathing.

Secondly, the use of a Bain circuit during a hypermetabolic state such as an MH reaction is limited by the maximum oxygen flow rate available from the anaesthetic machine. Even with a fresh gas flow of 12 $L \cdot min^{-1}$ we did not achieve normocapnia as we discovered when we inserted the capnograph into our circuit. Of interest, prior to modifying this machine, the oxygen flowmeter allowed a flow of only 6 $L \cdot min^{-1}$.

Finally, we feel that protocols for anaesthetic management of malignant hyperthermia should emphasize the problem of excessive CO_2 production during an MH reaction and gas-free machines should avoid T-piece circuits. The present protocol of the Anaesthesia Section of the Ontario Medical Association[7] suggests "all new tubing, plus fresh soda lime where being used." We feel that a circle system plus fresh soda lime should be recommended as the circuit of choice and that a T-piece system (such as a Bain circuit) or a circuit incorporating only a non-rebreathing valve should not be used unless very high flows of oxygen can be provided.

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Increased resistance to breathing: potentially lethal hazard across a coaxial circuit-connector coupling

To the Editor:

We wish to acknowledge Drs. Branson and Lam's[1] recent findings related to the use of "high shoulder" ET tube connectors and the CPRAM™ coaxial breathing circuit.

The design of the CPRAM™ circuit was intended to forego the use of this type of connector, also known as "low dead space," with its extended tip design. It has been recognized that these connectors in combination with any fresh gas adapter which has an extended tip design, could cause increased resistance to breathing.

In a US Food and Drug Administrated Safety Alert, dated September 2, 1983, this potential hazard was addressed.[2] A mailing to various hospital departments as well as publication in several journals was done.

In support of this, we have always included a "Warning" in our product labelling regarding this matter. This warning reads as follows: *Do not use with endotracheal tube, tracheostomy tube or airway connectors/adapters (including gas sampling) which have small internal orifices or "low dead space." Occlusion of the fresh gas flow may result.*

As the sole manufacturer of the CPRAM™ circuit, we feel a strong obligation to the clinician to inform them of any potential hazard.

We again, appreciate the concern of Drs. Branson and Lam.

Paul E. Dryden
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1 *Branson R, Lam AM.* Increased resistance to breathing: a potentially lethal hazard across a coaxial circuit-connector coupling. Can Anaesth Soc J. 1987; 34: S90–S91.
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