

FIG. 1. Fractured inner 11 mm ring.

and fentanyl 100 μ g iv, anesthesia was induced with thiopental 250 mg iv. While establishing comfortable mask fit to the patient's face, all breathing circuit connectors were turned (without undue force or difficulty) to a workable configuration. In doing so, a cracking noise was heard, and the inner 11 mm ring of the breathing circuit Y-piece was found to have fractured and broken out (fig. 1). This made connection of the circuit to the elbow connector impossible; lacking a spare breathing circuit in the room, the anesthesiologist commenced "mouth-to-mask" ventilation, which was easily accomplished. Continuous pulse oximetry suggested moderate arterial desaturation (from 99% to 91%), which then stabilized. A new circuit was obtained, the old replaced, and the case proceeded uneventfully, with a smooth intraoperative course and emergence, followed by complete recovery.

This case illustrates a type of breathing-system mishap for which many anesthetizing locations are poorly prepared. In retrospect, the use of the portable oxygen cylinder (stationed outside the room) might have provided an acceptable alternative circuit. Alternatively, the breathing circuit Y-piece might have been connected to the mask directly, without swiveling connectors, which is as simple as it is unfamiliar and awkward. However, this case suggests that a spare breathing circuit should be at hand within each anesthetizing location, rather than in a central supply area as is common in many institutions. Far worse consequences might have resulted if, for example, this had occurred during an anesthetic in a radiology suite.

We are unaware of other reports of this type of occurrence, and we are unqualified to judge whether this is a single "fluke" episode, or evidence of a design or manufacturing defect that has broader implications. The need for preparedness for either of these possibilities, however, remains indisputable.

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More on Black and White Granules in the Closed Circuit

To the Editor:— Delayed washout of volatile anesthetics and increased absorption of carbon dioxide by soda lime are specific features of closed circuit anesthesia systems. The latter requires frequent exchange of the carbon dioxide absorbent, the first can be dealt with by adding an activated charcoal filter to the circuit. Control of both is

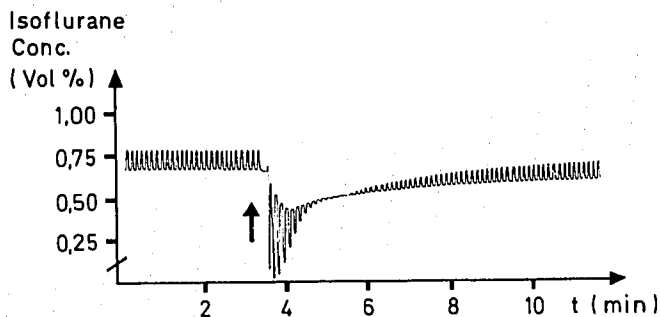


FIG. 1. Isoflurane concentration during minimal-flow anesthesia (FGF 0.5 l·min⁻¹). Arrow indicates exchange of soda lime canisters.

facilitated by anestheticography (on-line monitoring and recording of anesthetic vapor concentration¹).

Soda lime exchange during minimal-flow or closed circuit anesthesia results in a long-lasting reduction of vapor concentration (fig. 1). The immediate decrease is due to dilution, as one set of canisters—besides soda lime—contains 1.5 l of air. The delayed return to the initial concentration is caused by vapor adsorption onto fresh soda lime.² Anesthetic gas monitoring provides the information needed to rapidly restore the desired vapor concentration.¹

Maintaining a closed circuit throughout emergence requires vapor elimination by activated charcoal. A number of devices for this purpose have recently been described.^{1,3,4} The principle of volatile anesthetic agent's adsorption onto charcoal was discussed by Epstein in 1944⁵ and clinically applied by Bushman in 1977.⁶ Such a filter was, however, employed for the elimination of ether vapor from anesthesia systems as early as 1934.*

No mention has yet been made as to the position such a filter should occupy within the circuit. A position upstream from the soda lime

* Jantzen JP, Kleemann PP: Clinical application of anaesthetic gas monitoring. *Fortschr Anaesth* 2:59-63, 1987

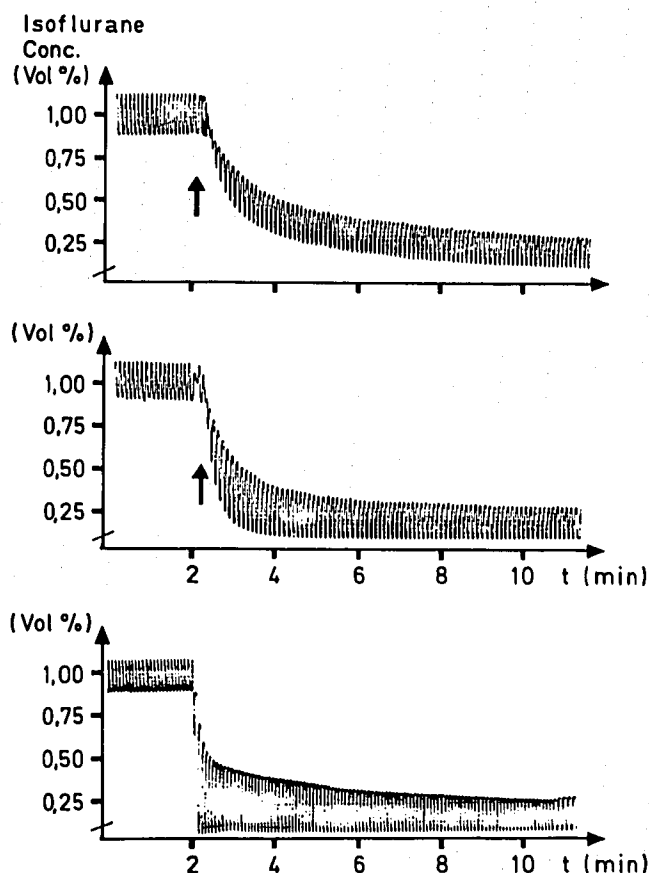


FIG. 2. Isoflurane washout. Arrow indicates commencement of elimination by closing the vaporizer and increasing FGF to 6.0 l^{-1} (top), or maintaining minimal-flow and integrating a charcoal filter either upstream from the soda lime canister (middle) or downstream from it (bottom).

canister¹ grants protection against inadvertently carrying charcoal particles into the patients airways. A position downstream from the CO_2 absorbers, close to the Y-piece,³ may be less safe from this aspect. It is, however, more effective in reducing the unfiltered apparatus dead space. This hitherto unreported position dependence of charcoal filter efficacy is shown in figure 2. The upper curve shows isoflurane wash-out, utilizing a fresh gas flow of $6.0 \text{ l} \cdot \text{min}^{-1}$ with no filter. In the middle curve, wash-out is faster with a FGF of $0.5 \text{ l} \cdot \text{min}^{-1}$, when an activated charcoal filter is positioned upstream from the soda lime canister. The buffer-volume of the soda lime canisters, however, retards the rate of decrease of inspired isoflurane concentration. The bottom curve shows an extremely rapid wash-out. Here the charcoal filter is positioned downstream from the soda lime canister and eliminates isoflurane quantitatively, reducing inspired isoflurane concentration to zero within one ventilatory cycle.

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REFERENCES

1. Jantzen JPAH, Kleemann PP, Erdmann K, Hein HAT, Wallenfäng T: Anestheticography: On line monitoring and documentation of inhalational anesthesia. *Int J Clin Monit Comput* 5:71-78, 1988
2. Grodin WK, Epstein MAF, Epstein RA: Soda lime adsorption of isoflurane and enflurane. *ANESTHESIOLOGY* 62:60-64, 1985
3. Baumgarten RK: Simple charcoal filter for closed circuit anesthesia. *ANESTHESIOLOGY* 63:125, 1985
4. Ernst EA: Use of charcoal to rapidly decrease depth of anesthesia while maintaining a closed circuit. *ANESTHESIOLOGY* 57:343, 1982
5. Epstein HG, Berlin DP: Removal of ether vapour during anaesthesia. *Lancet* 1:114-116, 1944
6. Bushman JA, Enderby DH, Al-Abtrak MH, Askill S: Closed circuit anaesthesia: A new approach. *Br J Anaesth* 49:575-587, 1977

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Secondary Prevention of Hypoxemia

To the Editor:—In a recent clinical study of arterial oxygenation as assessed by oximetry in anesthetized children, Dr. Coté *et al.* detected episodes of hypoxemia (i.e., $\text{SpO}_2 < 85\%$ for 30 s or longer) in approximately 17% of cases.¹ This finding illustrates that the usual measures taken to prevent hypoxemia often fail—i.e., measures to prevent and/or immediately detect and correct airway obstruction, hypoventilation, atelectasis, etc. Failure of primary prevention of hypoxemia is an important problem of anesthetic practice that emphasizes the need for detailed attention to so-called secondary preventive techniques, i.e., ways of recognizing hypoxemia after it develops but before hypoxic injury occurs. Coté's report provides information on secondary prevention which, when interpreted in conjunction with the results of other studies, has important implications for anesthetic practice.

Coté *et al.* noted that, during clinical episodes of hypoxemia ($\text{SpO}_2 < 85\%$), the heart rate, blood pressure, and respiration hardly changed.¹ This observation is explained by the results of our studies of volunteers that showed that both cardiovascular and ventilatory re-

sponses to induced moderate hypoxemia (i.e., PETO_2 40-45 mmHg) are severely impaired or abolished by commonly used halogenated anesthetics.²⁻⁴ Clearly, cardiorespiratory monitoring, including the ECG, has little or no value as an indicator of moderate hypoxemia during anesthesia with these agents.⁵ Coté *et al.* also observed that cyanosis, which has often been regarded as the most characteristic clinical sign of hypoxemia, was not detected consistently in their patients, even at SpO_2 values as low as 72%.¹ This suggests that, during inhalational anesthesia, cyanosis is a less reliable clinical sign, because cyanosis is nearly always evident at an SaO_2 of 72% in awake humans.^{6,7} We previously noted greater difficulty in detecting cyanosis during halogenated hydrocarbon anesthesia and proposed that this may be related to relative hyperperfusion of the skin and mucous membranes.⁵

Coté *et al.* further report that use of an oximeter during anesthesia made possible earlier detection of hypoxemia (defined by reduced oximeter readings) and thereby reduced the incidence of "major"