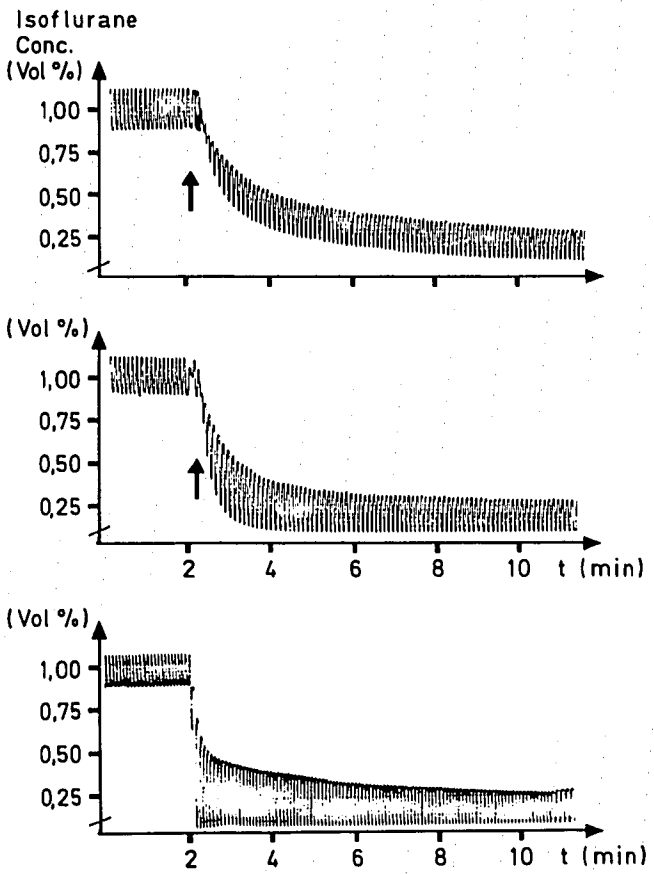

FIG. 2. Isoflurane washout. Arrow indicates commencement of elimination by closing the vaporizer and increasing FGF to 6.0 l^{-1} (top), or maintaining minimal-flow and integrating a charcoal filter either upstream from the soda lime canister (middle) or downstream from it (bottom).

canister[1] grants protection against inadvertently carrying charcoal particles into the patients airways. A position downstream from the CO_2 absorbers, close to the Y-piece,[5] 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 $l \cdot min^{-1}$ with no filter. In the middle curve, wash-out is faster with a FGF of 0.5 $l \cdot min^{-1}$, 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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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.*, $SpO_2 < 85\%$ 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 second ary 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 ($SpO_2 < 85\%$), 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 responses 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"

hypoxic events. Our work had shown that ear oximetry accurately tracks progressive reductions in SaO_2 above the threshold of clinical signs during both halogenated hydrocarbon anesthesia alone and anesthesia with surgery.[8] Thus, Coté's work confirms both the limitations of clinical signs and the usefulness of oximetry that we had previously described.

A clinical sign of hypoxemia during anesthesia that may be more sensitive than others, and which Dr. Coté's group may have had an opportunity to observe, is the color of the blood in the surgical field. During maintenance of anesthesia and (presumably) on-going surgery, they report several episodic desaturations to SpO_2 values of 85% or less.[1] In those circumstances, one would anticipate that any fresh blood at the site of surgery would be perceptibly dark.[5] Did they observe any change in blood color and, if so, at what level of SaO_2? Do the authors believe this to be an early clinical sign? To my knowledge, there are no published data on these questions.

Hypoxemic injury during anesthesia usually represents a failure of *both* primary and secondary prevention. While research has explored questions of primary prevention (*e.g.*, rate of failure, critical incidents, and their determinants), less attention has been given to secondary prevention—in particular, the real difficulties that may be encountered in recognizing hypoxemia during anesthesia. (This is somewhat ironic, since failure of primary prevention is tolerable, whereas failure of secondary prevention is clearly not.) Modern pulse oximeters undoubtedly facilitate secondary prevention, but they are not yet a panacea, being frequently inaccurate at lower saturations[9] (as corroborated by the single hypoxemic Pa_{O_2}/SpO_2 comparison reported by Coté *et al.*[1]), and susceptible to both extraneous artifacts and patient variables other than SaO_2.[8] We must still seek scientific knowledge about the sensitivity and specificity of clinical signs[5] and the reliability of oxygen monitors[9] and, in practice, make use of all clues of hypoxemia available to us.

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In Reply:—We agree with Dr. Knill that more research must be done to improve our primary methods of diagnosing hypoxemia; however, very significant desaturation must occur before a patient is visibly cyanotic and, as Dr. Knill has pointed out, many factors may influence the ability to diagnose cyanosis. Our study was designed to examine the incidence of hypoxic events and correlate these events with changes in vital signs and presence or absence of cyanosis. We clearly demonstrated that pulse oximeters are able to diagnose borderline desaturation well before any individual would be capable of this diagnosis, even if he/she knew that the patient was about to become desaturated. Dr. Knill's research on the cardiovascular effects of volatile anesthetic agents may well explain in part why we did not observe changes in vital signs with brief episodes of desaturation. Dr. Knill points out that, in his experience, cyanosis was more difficult to diagnose during halogenated hydrocarbon anesthesia, perhaps due to a "relative hyperperfusion of the skin and mucous membranes;" interestingly, all 14 patients (17 events) in our study who had major hypoxic events diagnosed by the oximeter and not by the anesthesiologist were receiving halothane anesthesia. Bear in mind, however, that nearly all pediatric patients in this institution are anesthetized with halothane.

Dr. Knill poses another interesting question, *i.e.*, can one observe the changes in the color of blood in the surgical field and relate this to oxygen saturation? Because we did not study this, we cannot provide scientific data; however, it is our clinical impression that only severe desaturation manifests as dark blood on the surgical field. Furthermore, we have had many occasions when the surgeon thought that the blood "looked dark" while the oximeter read 100% saturation. Since both surgeons and anesthesiologists are frequently accustomed to observing hyperoxic blood, any subtle change in color would probably go unnoticed.

We still agree, however, that observing the surgical field is extremely valuable and provides another piece of important data. In addition, we concur that more emphasis should be placed on the sensitivity and specificity of clinical signs and that we must not rely solely on monitors to diagnose desaturation; the oximeter provides an early warning, but it is up to clinicians to integrate the data from the monitors and their own clinical judgment and then take appropriate action.

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