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., SpO₂ < 85% for 30 s or longer) in approximately 17% of cases. 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, hyperventilation, 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 (SpO₂ < 85%), the heart rate, blood pressure, and respiration hardly changed. 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., PETO₂ 40–45 mmHg) are severely impaired or abolished by commonly used halogenated anesthetics. Clearly, cardiorespiratory monitoring, including the ECG, has little or no value as an indicator of moderate hypoxemia during anesthesia with these agents. 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 SpO₂ values as low as 70%. This suggests that, during inhalational anesthesia, cyanosis is a less reliable clinical sign, because cyanosis is nearly always evident at an SaO₂ of 72% in awake humans. 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.

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"
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REFERENCES


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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 anesthesiologists 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 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. Further-