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 any 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 72%. 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”
hypoexcic events. Our work had shown that ear oximetry accurately
tracks progressive reductions in SaO₂ above the threshold of clinical
signs during both halogenated hydrocarbon anesthesia alone and anes-
thetia with surgery. 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 SpO₂ values of 85% or
less. In those circumstances, one would anticipate that any fresh blood
at the site of surgery would be perceptibly dark. Did they observe any
change in blood color and, if so, at what level of SaO₂? 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 accidents,
and their determinants), less attention has been given to secondary
prevention—in particular, the real difficulties that may be encoun-
tered 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 un-
doubtedly facilitate secondary prevention, but they are not yet a pan-
acea, being frequently inaccurate at lower saturations as (corrobo-
rated by the single hypoxic PaO₂/SaO₂ comparison reported by
Coté et al.1), and susceptible to both extraneous artifacts and patient
variables other than SaO₂. We must still seek scientific knowledge
about the sensitivity and specificity of clinical signs and the reliability
of oxygen monitors and, in practice, make use of all clues of hypox-
emia available to us.

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 desat-
urated. 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 diag-
nose during halogenated hydrocarbon anesthesia, perhaps due to a
“relative hyperperfusion of the skin and mucous membranes,” interest-
ingly, 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 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. Further-
more, 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 ob-
serving hypoxic blood, any subtle change in color would probably go
unnoticed.

We still agree, however, that observing the surgical field is ex-
tremely valuable and provides another piece of important data. In
addition, we concur that more emphasis should be placed on the sensi-
tivity 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 moni-
tors and their own clinical judgment and then take appropriate action.

References

1. Coté C, Goldstein E, Coté M, Hoaglin DC, Ryan J: A single blind
study of pulse oximetry in children. Anesthesiology 68:184-188, 1988
2. Manninen PH, Knill RL: Cardiovascular signs of acute hypoxea-
emia and hypercarbia during enflurane and halothane anes-
3. Knill RL, Gelb AW: Ventilatory responses to hypoxia and hyper-
capnia during halothane sedation and anesthesia in man. Anes-
thesiology 40:244-251, 1978
4. Knill RL, Kierszszewicz HT, Dodson BG: Chemical regulation of
6. Comroe JH Jr, Botelho S: The unreliability of cyanosis in the
7. Medd WE, French EB, Wylie VM: Cyanosis as a guide to arterial
oxygen desaturation. Thorax 14:247-250, 1959
of two noninvasive monitors of arterial oxygeenation in
oximeters to profound hypoxia. Anesthesiology 67:351–
358, 1987

(Accepted for publication June 2, 1988.)

Charles J. Coté, M.D.
Associate Professor of Anesthesia
Harvard Medical School;
Associate Anesthetist
Massachusetts General Hospital
Department of Anesthesia
Massachusetts General Hospital
Boston, Massachusetts 02114

(Accepted for publication June 2, 1988.)