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What Difference Does Pulse Oximetry Make?

SEVERE HYPOXEMIA can kill a patient, or leave its victim with devastating neurological handicaps. Readers of this journal know that arterial hypoxemia can occur at any time during an anesthetic with astonishing suddenness. Yet, until recently, devices to measure arterial oxygen levels non-invasively and continually during anesthesia have not been available.

Over 40 years ago, Comroe and Botelho published their classic study of the accuracy with which clinicians of varying experience (from medical students to professors) assess degrees of cyanosis and arterial oxygen desaturation.[1] They disturbingly concluded that "Visual impressions of cyanosis are unreliable" and clinical experience made no difference in the accuracy of one's assessment. Still, without reliable continual *in vivo* oxygen measurement, anesthesiologists and other clinicians were forced to detect hypoxemia by skin and blood color or by late occurring hemodynamic deterioration.

Measurement of the arterial oxygen tension in whole blood was difficult until Clark reported in 1956 the development of a polarographic oxygen electrode coated with a semipermeable membrane that made these measurements clinically available.[2] Subsequently, Huch *et al.*[3] modified the Clark electrode for use on the skin of a neonate. With hyperemia caused by topical vasodilators, the oxygen electrode provided values nearly equal to the arterial oxygen tension. In 1972, both Huch and Eberhard[4,5] found that similar data could be obtained in the neonate with a heated electrode.

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Following these developments, monitoring of cutaneous oxygen tension became common in neonatal intensive care units. However, poor correlation with the arterial oxygen tension has diminished enthusiasm for cutaneous oxygen monitoring in older children and adults. Burns, the need for sensor calibration, slow response time, and the variation of data as a result of sensor position all contribute to lack of enthusiasm for this technique to estimate oxygen levels in blood. Furthermore, with hemodynamic instability, cutaneous oxygen tension becomes blood flow dependent and less accurately reflects arterial oxygen tension.[6,7] Under these circumstances, its primary value may be as a monitor of tissue oxygenation.

Pulse oximetry has its origins in the work of Nicolai,[8,9] who, in 1931, applied the Beer-Lambert Law to the transmission of light through the hand to study the dynamics of tissue oxygenation. He demonstrated that occlusion of the circulation produced an exponential fall in oxyhemoglobin and rise in deoxyhemoglobin. Kramer[10] reported, in 1935, the continuous recording of oxygen saturation of blood flowing through unopened vessels using a spectrophometric method. At the same time, Matthes[11] constructed the first device to measure oxygen saturation *in vivo* by transilluminating the ear.

Interest in aviators' oxygenation during World War II stimulated further development. Squire[12] and, later, Goldie[13] developed oximeters that set their zero value on tissue that had been compressed to squeeze out the blood, marking the beginning of modern pulse oximeters. Millikan, in 1942, developed a lightweight, practical aviation ear oxygen meter which he called an oximeter.[14] Based on this work, Wood and Gracie, at the Mayo Clinic in 1949, built an oximeter with improved optics and an inflatable balloon that could make the ear bloodless for a reference setting.[15] This device was

manufactured by the Waters Company, and was extensively used in research, including evaluation of hypoxia during anesthesia.[16] More recently, Shaw, reported by Merrick and Hayes,* developed a self-calibrating ear oximeter using eight wavelengths of light that was commercially produced in 1976 and has become the standard against which other oximeters are judged.

In 1974, Aoyagi, as reported by Nakajima *et al.*,[17] developed an oximeter that used the variations in volume which occurs with pulsatile arterial flow to obtain a signal representing oxygen saturation. Because of sensitivity to motion and because of its large size, the device was not widely used. Yoshiya *et al.*,[18] in 1980, incorporated plethysmography and oximetry in an instrument that needed no calibration or heat to determine oxygen saturation. Using plethysmography to identify a volume change resulting from the arterial impulse, the device compared the light absorbance in the absence of a pulse —its zero—and in the presence of the arterial pulsation to determine arterial oxygen saturation. With this integration of plethysmography and oximetry and subsequent application of solid-state electronics, the pulse oximeter determines values within 2% of those measured *in vitro,* bringing us close to accurate, practical noninvasive assessment of arterial oxygen saturation.[19] Motion artifact and difficulties with low perfusion states continue to limit performance of currently available devices.

In this issue of ANESTHESIOLOGY, Coté and his coworkers describe the incidence, duration, and severity of arterial oxygen desaturation as detected by pulse oximetry in infants and children.[20] Additionally, in a cleverly designed single-blinded prospective manner, they determined the impact of pulse oximetry information on the anesthesia care team function. Their results, consistent with our own clinical experience with pulse oximetry, indicate the device is a valuable aid to the inspection, palpation, and auscultation used by the anesthesiologist seeking the best care for his patient.

In 152 pediatric patients who ranged in age from 1 day to 15 yr and in whom duration of anesthesia ranged from 4 min to 5 h, Coté *et al.* found the following:

First, the incidence of serious arterial oxygen desaturation detected by pulse oximetry ($SpO_2 < 85\%$) is not significantly different from that observed in adults (17% overall in the children, 20% in adults).[27] However, the incidence is significantly higher in children less than 2 yr of age. Explanations for this apparent vulnerability of younger children include: 1) pulmonary vasoconstriction and reversion to a fetal circulatory pattern in the neonate develops in response to moderate hypoxemia and acidemia; and 2) oxygen consumption per minute per kg body weight of neonates and older infants is approximately double that of the adult, while the functional residual capacity is 10–20% less per kg than in the adult.[22,23] Coté's observations provide a possible explanation for the threefold greater incidence of cardiac arrest during anesthesia in children than in adults recently reported from a large tertiary medical center by Keenan and Boyan.[24]

Second, in the absence of SpO_2 measurement, a significantly increased incidence and duration of desaturation episodes occurred. Without pulse oximetry information, only ten of 24 episodes of hypoxemia were detected by the anesthesia care team; although, in all 24 of these patients, the SpO_2 was less than 73%. Nine patients with SpO_2 under 73% were diagnosed as acyanotic at the time. The clinical experience of the observer bore no relationship to subjective assessment of desaturation confirming Comroe's and Botelho's findings of 40 yr ago.[1]

Third, the decrease in SpO_2 precedes changes in skin color or hemodynamic variables. Indeed, Coté and coworkers confirmed the clinical opinion of many experienced anesthesiologists that changes in heart rate and heart tones or ECG are late signs for the detection of arterial hypoxemia (in only 17% of the major desaturation episodes did hemodynamic variables change).

Fourth, 32% of the patients whose anesthesiologist did not have pulse oximetry information experienced major desaturation ($SpO_2 < 85\%$), whereas only 14% of the patients whose anesthesiologist had continuous oximetry information available had such episodes ($P = 0.02$). Similar differences also were observed between the two groups exhibiting borderline desaturation after termination of anesthesia.

A recent study of adult patients, by Cooper *et al.*, focuses upon an analysis of unanticipated, undesirable events that were possibly related to anesthetic management and required intervention in the recovery room.[25] In this study, the random use of pulse oximetry did not significantly increase the number of reports in the recovery room of hypoxic or hypoxemic events, although the authors note that pulse oximetry may have provided information significantly earlier to the clinician, thus preventing an hypoxemic event and the need for reporting these events to the recovery room staff.

As Coté points out, numerous artifacts can occur with pulse oximeters, especially when fitting the sensor to a small digit or when the patient is moving. These difficulties, along with others, are being addressed by the manufacturers. Another recent study by Severinghaus and Naifeh[26] describes in detail the performance of six commonly available pulse oximeters under conditions of sudden and profound transient arterial desaturation

* Merrick EB, Hayes JJ: Continuous non-invasive measurements of arterial blood oxygen levels. Hewlett-Packard Journal 28:2–9, 1976

in adult volunteers. Similar additional evaluations of the performance of new oximeters will enable users to select the best performing device among available choices.

We believe that pulse oximetry should become part of the routine monitoring of all patients undergoing general anesthesia, especially pediatric patients. Supplementing our senses with devices to assess hemodynamic function, body temperature, inspired oxygen, end-tidal carbon dioxide, and anesthetic concentration, as well as continual arterial SpO_2, will help us to detect potentially harmful conditions earlier and prevent injury.

Further prospective studies must be carried out to confirm the value of the other instrumentation, such as continual measurement of end-tidal CO_2 and inhalation agents, and verify the claims that these devices improve anesthetic care. In our opinion, Coté and co-workers have done that for pulse oximetry in pediatric patients.

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