
PULSE OXIMETRY

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ABSTRACT. The pulse oximeter, a widely used noninvasive monitor of arterial oxygen saturation, has numerous applications in anesthesiology and critical care. Although pulse oximetry is considered sufficiently accurate for many clinical purposes, there are significant limitations on the accuracy and availability of pulse oximetry data. This article reviews both the clinical uses of the pulse oximeter and the limitations on its performance. The pulse oximeter is generally acknowledged to be one of the most important advances in the history of clinical monitoring.

KEY WORDS. Equipment: pulse oximeters. Measurement techniques: pulse oximetry. Monitoring: oxygen. Blood: oxy-hemoglobin saturation; oxygen saturation, arterial.

That primordial noninvasive indicator of tissue oxygenation, skin color, is an insensitive monitor at best, as Comroe and Botelho [1] demonstrated in their classic study. At worst it is useless, as in the presence of heavy pigmentation or severe anemia. Frank cyanosis develops in the presence of 5 g/dl reduced hemoglobin [2]. This translates into a dangerously low arterial oxygen saturation (SaO_2) of 67%, and then only if the patient has a healthy 15 g/dl of hemoglobin.

In this decade, the noninvasive assessment of SaO_2 has been made possible and simple by pulse oximetry, "arguably the most significant technological advance ever made in monitoring the well-being and safety of patients during anesthesia, recovery, and critical care" [3]. Indeed, pulse oximetry rapidly seems to be approaching "standard of care" in American operating rooms.

Invented in the early 1970s, pulse oximetry remained a research tool for several years before entering the clinical arena in the early 1980s. It represents not so much a basic physiologic discovery as an electronic one. The technologic principles behind it went uncombined for decades preceding its development and microprocessor-based realization.

After 5 years of increasingly widespread clinical use, pulse oximetry has found a number of well-established applications. An active ferment of investigation into further possibilities is underway. Equally vigorous are current efforts to understand the limits on the applicability of data obtained from pulse oximeters under various clinical conditions. Reviews of the principles, applications, and limitations of pulse oximetry have recently been published [4-6a].

This review surveys the clinical literature on pulse oximetry—much of which has appeared in the past 2 years—in somewhat more detail than have previous ac-

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counts. It is hoped that the clinician may thereby gain an overview of the uses and limitations of this monitor.

In this review, the term SaO_2 is used either in the abstract or as calculated or measured by an in vitro blood gas analyzer or oximeter. The term SpO_2 refers specifically to the SaO_2 value displayed by a pulse oximeter.

HISTORY

Like most other inventions, the pulse oximeter embodies many antecedent ideas and discoveries. The discovery that enabled all others to come together in a single instrument was the combination of transmission oximetry and photoplethysmography in 1974 by Aoyagi et al [7], who developed the first pulse oximeter. Space does not permit mention of the countless others whose work led to the discovery of Aoyagi et al, but this incomplete list of people and ideas is itemized more fully in several good articles [8,9], notably the superb historical work by Severinghaus and Astrup [3,10].

In 1864 Stokes [11] reported his discovery that the colored substance in blood is also its carrier of oxygen. Around the same time, Hoppe-Seyler [12], who first crystallized this substance and coined for it the term hemoglobin, showed that its pattern of absorption of light from the solar spectrum changed when it was shaken with air. The hemoglobin-oxygen dissociation curve was elucidated, and, in subsequent decades, Hufner [13] studied the optical spectra of hemoglobin and oxyhemoglobin.

The first device to use these spectra to measure oxygen saturation in human blood by transilluminating it with colored light was built in 1935 by Matthes [14], who based his invention on studies by Nicolai [15]. It was incapable of distinguishing between arterial and venous or capillary blood. In 1941, Millikan [16] refined this device into a lightweight instrument for the British war effort specifically to address the problem of loss of consciousness by aviators during dogfights. Its use was first reported in connection with surgical anesthesia in 1948 [17].

The problem of excluding venous and capillary blood to measure only SaO_2 was addressed in the early 1940s by Goldie [18] and Squire [19], who zeroed an oximeter by taking a "bloodless" reading from an ear lobe compressed between two fingers, and by Millikan [20], who "arterialized" the blood in the ear lobe by heating it. Until the advent of pulse oximetry, heat-induced vasodilation was used to approximate arterialization by devices as sophisticated as the eight-wavelength Hewlett-Packard ear oximeter. This principle is still used by transcutaneous oxygen tension (PO_2) monitors, which

measure PO_2 polarographically with the Clark oxygen electrode.

Meanwhile, an entirely separate phototechnology had been awaiting application to this problem for years. The recording of a pulse wave in the finger was first reported in 1911 [21], and the technique of photoplethysmography was born in the mid-1930s when changes in the intensity of transmitted light were used to describe this waveform [22]. It had been used mainly to assess adequacy of perfusion in extremities.

Aoyagi et al [7] originated the idea of using photoplethysmography to synchronize spectrophotometric measurements of hemoglobin saturation with the peak and trough of the pulse waveform, thereby isolating and characterizing a purely pulsatile component. They discovered it in the early 1970s while trying to accomplish something else altogether: the noninvasive photometric determination of cardiac output by dye dilution. Annoying pulsatile variations plagued their dye-dilution curves, and they initially used pulse waveform detection as a way of *eliminating* this pulsatile "noise." But their subtraction algorithm gave results more erratic than expected—and they were familiar enough with the history and technology of oximetry to realize that these discrepancies were due to changes in oxygen saturation. (Aoyagi and colleagues were using 630-nm light to detect dye, and the difference in absorption between oxidized and reduced hemoglobin at 630 nm is considerable.)

Aoyagi and colleagues then set about developing the pulse oximeter—an instrument that would explicitly measure SaO_2 by measuring absorption data at both the peak and the trough of a detected pulse wave, and cancelling out what was common to both. What remained was the pulsatile, and presumably the arterial, component.

The utility of pulse oximetry in clinical settings went unappreciated for almost a decade. Aoyagi and associates did market their device commercially in 1975, but only briefly. Soon afterward, the Biox Company (Boulder, CO) patented technologic advances on the pulse oximeter in the United States, targeting respiratory care and research markets. The anesthesiologist William New, a cofounder of Nellcor, Inc (Hayward CA), helped catalyze the explosion of pulse oximetry into the clinical arena in the early 1980s [23]. As outlined in this review, the clinical scope of this instrument is still expanding.

TECHNOLOGY

The technology of pulse oximetry is well reviewed [24–28], notably in a highly readable account by Polge [29]. Some basic principles are outlined here.

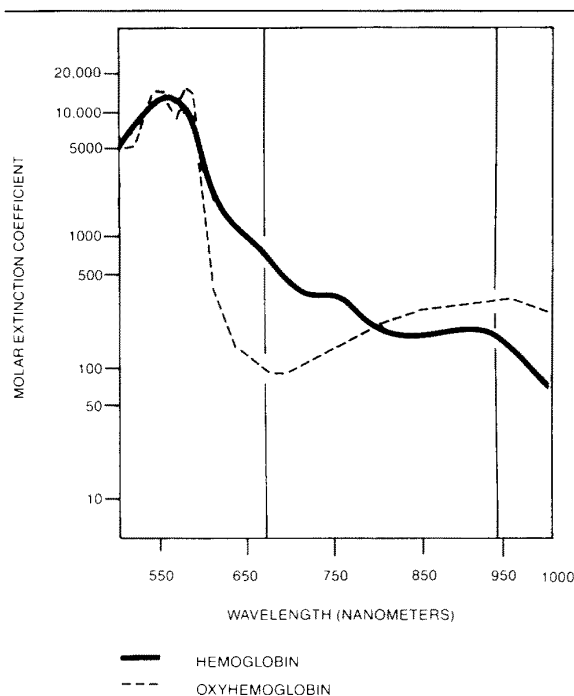


Fig 1. Logarithm of molecular extinction coefficients, representing degrees of light absorption, for hemoglobin (solid line) and oxyhemoglobin (broken line). Typical wavelengths used in pulse oximetry (660 and 940 nm) are shown by vertical lines. Note that the two species of hemoglobin differ widely from each other at each of the wavelengths shown, and differ widely from themselves across the two wavelengths; both differences are important for optimum accuracy in pulse oximetry. From Mackenzie [9]. Used with permission.)

SaO₂ is determined by the relative proportions of oxygenated hemoglobin and reduced hemoglobin in arterial blood. The pulse oximeter estimates SaO₂ by sensing differences in the absorption spectra of these two forms of hemoglobin. Two frequency bands are particularly relevant. Reduced hemoglobin absorbs more light in the red band, 600 to 750 nm, than does oxyhemoglobin—that is, oxidized hemoglobin looks redder than reduced hemoglobin does. The other difference is not visible: oxyhemoglobin absorbs more light in the infrared band, say 850 to 1000 nm, than does reduced hemoglobin (Fig 1).

The oximeter probe is placed on some appendage rich in cutaneous vasculature; the finger tip and the ear lobe are two prime sites. The probe contains two light-emitting diodes (LEDs)—diodes that emit light at specific wavelengths, one in the red band and one in the infrared. For example, the Ohmeda Biox 3700 uses 660 and 940 nm; the Nellcor N-100 uses 660 and 910 nm.

These LEDs are capable of emitting a very bright light—“sufficient power to barrel through a darkly pigmented, edematous infant foot” [29]. The amount of light transmitted through the intervening finger tip or ear lobe is measured several hundred times per second at both wavelengths. This rapid sampling rate allows precise recognition of the times of the peak and trough of each pulse waveform, as determined by changes in light transmission caused by expansion and relaxation of the tissue’s vascular bed. The pulse amplitude accounts for about 1 to 5% of the total signal [30]. Absorbance data from both peak and trough are saved and used in calculations.

At the trough, the finger tip contains arterial, capillary, and venous blood, as well as intervening tissue. At the peak, it contains all this plus a further quantity of purely arterial blood. The presence of this additional arterial blood changes the amount of transmitted light in both red and infrared bands. These changes can be associated algorithmically with SaO₂ values.

If measurements were undertaken on samples of pure oxyhemoglobin and reduced hemoglobin in optical cuvettes, a straightforward application of the Lambert-Beer law would yield the percent hemoglobin saturation. This law governs the total absorbance of a system of absorbers. Unfortunately, the Lambert-Beer law is inapplicable in practice for a variety of reasons, notably the scattering of light by erythrocyte, skin, and other cell membranes [31]. The early pulse oximeters, which used this law, grossly overestimated SaO₂ readings under 90%. For example, one investigation of these devices found SpO₂ to be 70% when SaO₂ was 50% [32].

Since then, theoretical algorithms have been abandoned in favor of empirical ones. Most pulse oximeters currently in use base their calculations on “calibration curves” derived from studies in healthy volunteers (Nellcor is said to have used 5 Olympic athletes [33]), from whom light absorption data were associated with simultaneous *in vitro* measurements of SaO₂. This has adverse implications for the accuracy of pulse oximeters at very low saturations, since healthy volunteers do not attain these depths (see the Accuracy section under Performance and Limitations).

The isolation of the pulsatile component neatly skirts potential problems with the wide interindividual variations in pigment, quantity of intervening tissue, and so on. Those factors do affect absorbance and are all part of the baseline tissue absorbance data, but the algorithm isolates only those changes caused by the pulse of arterial blood, above and beyond the baseline. Thus the pulse oximeter probe can start giving meaningful measurements as soon as it is clipped onto anyone with a pulse, without individual calibration.

The "pulse search" process that occurs when a probe is initially applied (or dislodged) includes sequential trials of various intensities of light in an effort to find one strong enough to transmit through the tissue, but not so strong that the device's front-end detection/amplification system is saturated. Once a pulse is found, there is generally a delay of a few more seconds while SaO_2 values for several pulses are averaged for display. Some models allow this averaging period to be adjusted.

An increasing number of pulse oximeter manufacturers offer trend data storage of pulse and SpO_2 measurements over a period as long as 8 hours. This feature is especially useful in evaluating episodic hypoxemia during sleep. Trend data can be transferred to a personal computer and processed further, as has been described for the Ohmeda Biox 3700 and IBM PC [34].

Current pulse oximetry technology uses "transmission mode," that is, it measures light transmitted through transilluminated tissue. One desirable possibility for the future of pulse oximetry would involve the use of "backscatter" or "reflectance mode," in which measurements of reflected light would allow monitoring at nontransilluminable sites, such as the fetal presenting part during labor [27,30]. Another possibility is to assess deep tissue (especially vital organ) oxygenation via lasers. These may study not only hemoglobin but other biomolecules subject to oxidation-reduction, such as cytochrome *aa_3* [35].

CLINICAL USES: OXIMETRIC

Because desaturation is the final common pathway of many classes of medical mishap, the intraoperative and critical-care applications of a noninvasive, sensitive, easily applied continuous SaO_2 monitor are obvious. The relative merits of pulse oximetry and other oxygenation monitors are well recognized [24,36–38].

Pulse oximetry, although not currently included in the American Society of Anesthesiologists (ASA) Standards for Basic Intra-Operative Monitoring [39], is rapidly approaching unofficial "standard-of-care" status among anesthesiologists in the United States [6,40–43a], Great Britain [44,45], and Australia and New Zealand [46]. Indeed, there is lively controversy over whether pulse oximetry has rendered other time-honored methods, such as the precordial/esophageal stethoscope, obsolete [47,48]. Nevertheless, the dictum of Holland [49] that "there is only one absolutely indispensable monitor, and that's a trained, experienced person in the operating room" has a hallowed place in the collective psyche of anesthesiology.

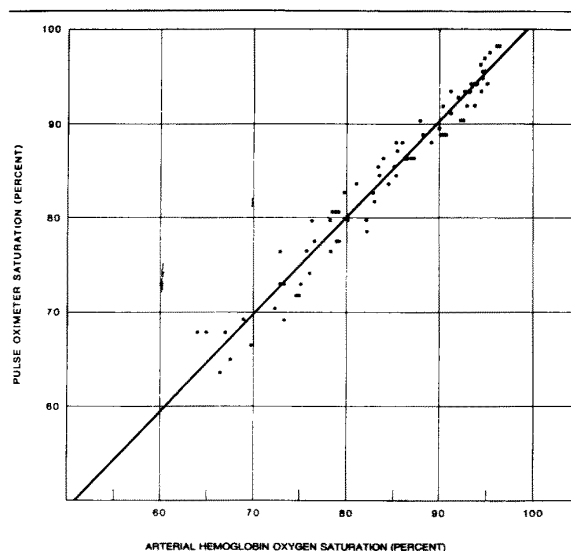


Fig 2. The original demonstration by Yelderman and New that a pulse oximeter (the Nellcor N-100) correlated well with an *in vitro* oximeter ($r = 0.98$). (From Yelderman and New [25]. Used with permission.)

Intraoperative/Critical Care

In the literature on pulse oximetry, the article most often cited is the landmark demonstration by Yelderman and New [25] that a pulse oximeter (the Nellcor N-100) correlates well ($r = 0.98$) with a recognized standard for *in vitro* oximetry, the IL282 CO-Oximeter (Instrumentation Laboratory, Lexington, MA). The 1983 publication of that article (Fig 2) coincides with the onset of large-scale routine clinical use for this monitor in operating rooms and intensive care units.

Since then, the monitor's utility and accuracy have been verified repeatedly in various surgical and intensive care unit subpopulations. Many of these studies simply reperform the correlation experiment between pulse oximeter and SaO_2 standard in some specific clinical setting. When the subpopulation has the same hemoglobin (human hemoglobin A) as the population in the study of Yelderman and New, as it often does, agreement with the original conclusion is generally unsurprising. But other investigations go further, either by illuminating unusual clinical conditions, by examining predispositions to desaturation, or by testing new uses for the pulse oximeter.

Pulse oximetry has been shown to decrease the incidence and duration of desaturation episodes both intraoperatively and after termination of anesthesia. In a study by Coté et al [50], only 10 of 24 hypoxemic epi-

sodes detected by a pulse oximeter were also detected by anesthesia care personnel blinded to the pulse oximeter output. In all 10 detected incidents, the SpO_2 was under 73%. Yet 9 more incidents under 73% went undetected clinically. The lower percentage of patients experiencing major desaturations with pulse oximetry (14%, versus 32% without) suggests that this monitor not only enables prompt recognition and treatment of hypoxemia, but can help prevent it as well.

SaO_2 readings offer some advantages over arterial oxygen tension (PaO_2) readings in the evaluation of hypoxemia, given the possibility of shifts in the hemoglobin-oxygen dissociation curve. On the other hand, SaO_2 is less useful than Po_2 in the assessment of hyperoxemia, where large changes in Po_2 produce little or no change in SaO_2 . For example, pulse oximetry has limitations in detecting *early* desaturation at a *high* inspired oxygen fraction (FiO_2) (e.g., with one-lung ventilation or accidental endobronchial intubation after preoxygenation) [51,52]. Of course, neither SaO_2 nor PaO_2 is useful in hypoxemia due to anemia or dyshemoglobins. When SaO_2 measurements are desired, however, the pulse oximeter offers clear advantages over blood gas analysis and *in vitro* oximetry. It provides SaO_2 readings continuously, noninvasively, and relatively immediately.

The pulse oximeter shows promise for systematically and conveniently studying the risk factors for rapid desaturation. For example, obesity has been suggested as such a risk factor in some [53,54], but not all [55], investigations. The survey by Raemer et al [53] of patients undergoing ambulatory gynecologic surgery found hypoxemia to be associated with age, manual ventilation, the lithotomy position, and postoperative arousal (presumably due to hypoventilation). Tyler et al [54] and others have examined this by measuring continuous SaO_2 on the way to the recovery room. It is interesting that Raemer and colleagues saw no tendency to hypoxemia in asthmatics requiring medication, while Tyler and colleagues saw postoperative desaturations in all 3 of their patients whose asthma was inactive.

A recent cost-benefit analysis showed that pulse oximetry is worthwhile if only 1 in 40,000 intraoperative hypoxemic episodes (SaO_2 of 90% or less) is associated with mortality [53]. Estimates of the per-case cost of pulse oximetry range from \$1.35 [53] to \$2.40 [55]. One institution estimated that this monitor rendered unnecessary 16% of the arterial blood gas analyses done for diagnostic purposes and 48% of those done to assess therapy [56]. A third estimated a savings of \$146,000 just in assessing supplemental oxygen therapy on the wards [57].

A lengthy, elaborately designed study by Cooper et al [58] showed that significantly fewer patients experienced "recovery room impact events" (such as hypotension and dysrhythmia) after pulse oximetry was introduced into the operating room. The study fell short of establishing a cause-and-effect relationship, however. The authors suggest that this may have been due to insufficiently sensitive operational definitions of impact events.

Transport to the Recovery Room

Patients not receiving supplemental oxygen may be vulnerable to desaturation while being transported from operating room to recovery room. Residual anesthetics and narcotics may depress ventilation; muscle relaxants may only recently have been antagonized; preoxygenation for transport may not have been effective (e.g., if there is a large alveolar-arterial oxygen gradient). Five studies addressing this question have found at least a mild potential for desaturation during transport.

Of 95 ASA Status I or II patients studied by Tyler et al [54], 35% became hypoxemic (SaO_2 less than 90%), and 12% severely so (SaO_2 less than 85%), on the way to the recovery room. Two factors predisposed to hypoxemia: obesity and a history of asthma (currently inactive). No significant association was seen with age, anesthetic agent, duration of anesthesia, or level of consciousness during transport.

Elling and Hanning [59] found a minimum SpO_2 of 90% or less in 65% of patients making the journey, most of whom had been preoxygenated. Curiously, Blair et al [60] found this figure to be only 29% in nonpreoxygenated patients, and postulated that the presence of an anesthesiologist during transport may have made the difference. An average SpO_2 of 89% was found by Graham et al [61] in 18 patients 2 minutes after transport was begun.

In pediatric patients, Kataria et al [62] showed that younger patients have lower SpO_2 values during transport; in infants under 6 months old SpO_2 drops to a mean minimum of 88.1% despite preoxygenation. Vijayakumar et al [63] found transient hypoxia (SpO_2 less than 90%) in 10 of 52 pediatric patients preoxygenated with 100% oxygen and in 18 of 49 preoxygenated with 50% oxygen. The data suggested associations with obesity, previous prematurity, and lower airway disease.

All of these studies show that the pulse oximeter can be useful in detecting hypoxemia during transport to the recovery room.

Recovery Room

Hypoxemia is more prevalent at the time of discharge from the recovery room (9%) than it is preoperatively (2%) or at 5 or 30 minutes after admission to the recovery room (4% and 6%, respectively), according to a study of 149 inpatients by Morris et al [64]. Only the 9% prevalence at discharge was a statistically significant difference from baseline. In all, 21 (14%) of these patients exhibited transient hypoxemia; the percentage might have been higher had SpO₂ been measured continuously. Roughly half of the episodes were without apparent explanation. A similar study in 97 children disclosed a 43% incidence of transient desaturation in the recovery room [65]. In both studies, an unknown proportion of patients received supplemental oxygen (predominantly at the onset in the work of Morris et al), complicating the interpretation of these results.

Endobronchial Intubation

Pulse oximetry is *not* reliable in detecting endobronchial intubation in dogs at an FiO₂ of 50% or higher [52], and does not do so with certainty even at an FiO₂ of 30% [66]. At an FiO₂ of 50%, endobronchially intubated dogs maintained an SpO₂ of roughly 100% for the 20 minutes studied, while transcutaneous and intraarterial monitors documented a fall in PO₂ to the low hundreds in the first few minutes.

Viitanen et al [67] found the response time of pulse oximetry superior to that of transcutaneous PO₂ measurement during one-lung ventilation at an FiO₂ of 50%, but they analyzed only data for subjects who became hypoxemic during one-lung ventilation and did not comment on the relative sensitivities of the two monitors in all subjects. Brodsky et al [68] found good correlation between the Nellcor N-100 and in vitro oximetry during one-lung ventilation in the SaO₂ range of 80 to 100%.

Preparation for Cardiac Surgery

In 9 of 12 patients who had received premedication for cardiac surgery (0.1 mg/kg morphine sulfate intramuscularly and 0.4 mg scopolamine intramuscularly, with up to 5 mg diazepam intravenously and/or 100 µg fentanyl intravenously as needed), SpO₂ dropped below 90% while invasive monitors were placed preoperatively [69]. Possible contributing factors included pre-anesthetic medication, drapes over the face, and the Trendelenburg position. This observation could help to explain the 18% incidence of new ischemia found by Slogoff and Keats [70] in patients arriving in the op-

erating room for myocardial revascularization. These authors do not comment, however, on the timing of invasive monitor placement relative to arrival in the operating room.

Cardiac Catheterization

Eleven of 29 patients undergoing cardiac catheterization were found by Dodson et al [71] to have transient desaturations below SpO₂ values of 90% (mean duration, 53 seconds) during cardiac catheterization. The 11 patients had a mean number of 16 desaturations per patient. Baseline SpO₂, duration of procedure, and left ventricular end-diastolic volume were all associated with desaturation.

Cardiopulmonary Bypass

Pulse oximeters are generally thought to be of little use during the low and nonpulsatile flow conditions of cardiopulmonary bypass. However, one study [72] claims good results when an Ohmeda Biox III was checked against in vitro oximetry during cardiopulmonary bypass, despite a "low signal output" message throughout. The Nellcor N-100 did not detect flow under these conditions.

Late Pregnancy and Labor

The only published report on maternal SaO₂ during labor found an association between labor pain and desaturation; both of these were more pronounced with nitrous oxide-meperidine anesthesia than with peridural anesthesia. In primiparas receiving the former, desaturation to $88.8 \pm 3.9\%$ occurred with contractions [73].

In a small number of patients meeting criteria for supine hypotensive syndrome, Calvin et al [74] studied the effects of the supine position on SpO₂. Forty-two female subjects in the third trimester of pregnancy were studied; of these, 6 met criteria for supine hypotensive syndrome, and 3 of the 6 had a drop in SpO₂ of 3 to 5% when supine. No fetal heart rate decelerations were noted. The clinical significance of these findings is uncertain.

Amniotic Fluid Embolism

An excellent example of the early-warning capabilities of pulse oximetry is provided in a case report by Quance [75]. A 28-year-old gravida 1, para 0 woman underwent cesarean section for failure to progress. Within 1 minute of delivery, her SpO₂ fell from 100% on 4 L/min oxygen to 71% on room air and then 83 to 92% on 4 L/min

oxygen, with a PaO_2 of 49 mm Hg. There were no symptoms and no untoward changes in blood pressure or heart rate. Rales and jugular venous distention were appreciated 45 minutes after delivery, and pulmonary artery catheterization 150 minutes after delivery showed a pulmonary artery pressure of 75/34 mm Hg. Three hours after delivery, external uterine massage was followed immediately by convulsions and asystole, which did not respond to prolonged resuscitative efforts. Post-mortem examination was consistent with a massive amniotic fluid embolism. The initial prolonged desaturation was likely due to amniotic fluid embolism as well, although interpretation was complicated by subsequent uterine massage. The pulse oximeter provided the only indication of abnormality for the first several minutes of this episode.

Ward Epidural Narcotic Analgesia

The pulse oximeter has been suggested as a monitor for respiratory depression in ward patients receiving epidural narcotics. There was no difference in the incidence of desaturation between parenteral and epidural morphine in the small ($n = 20$) population examined [76].

Oxygen Therapy and Respiratory Care

The pulse oximeter assists in the adjustment of oxygen therapy in a variety of settings—on the ward [57], during weaning from a ventilator [77], and at home. One study of pulse oximetry and home oxygen found it especially useful during exercise, and discovered that 4 of the 36 patients under study did not need home oxygen after all [78]. During respiratory care, an emergency-room study noted transient desaturations with tracheal suctioning (mean drop in SpO_2 of 5.3%), intubation (5.2%), vomiting (3.8%), coughing (3.1%), and lying supine (2.6%) [79].

The combination of pulse oximeter and pulmonary artery oximeter has been used to titrate continuous positive airway pressure by estimating venous admixture and total body oxygen utilization coefficient, two variables essential in the prescription of continuous positive airway pressure. The estimates thus obtained were found useful in the rapid and accurate titration of continuous positive airway pressure in most patients with acute respiratory failure [80,81].

Kinetic Bed Therapy

In the presence of unilateral lung disease, SaO_2 may drop when the healthy lung is dependent. Brouwers [82] noted that this may make a single arterial blood gas

value misleading if a patient is on a kinetic bed that periodically rolls from side to side to improve pulmonary toilet. He suggests that continuous SaO_2 monitoring by pulse oximetry is a better guide to therapy in these patients.

CLINICAL USES: PLETHYSMOGRAPHIC

Another group of applications for the pulse oximeter ignores saturation data altogether and uses the device as a plethysmograph. Pulse oximetry has been used to verify adequacy of chest compressions in cardiopulmonary resuscitation [83], to test for brachial plexus compression during shoulder arthroscopy [84], to perform Allen's test [85–89], to locate the femoral artery for cannulation when obesity prevented appreciation of a femoral pulse [90], to assess vascular integrity after extremity replantation surgery [91], and to measure systolic blood pressure [92,93]. Several of these proposed applications are open to criticism, however. A pulsation large enough to trigger a pulse signal on an oximeter may or may not be sufficient for the purpose at hand. The objection is especially telling if one accepts the findings of Lawson et al [94] that flows as low as 4 to 8.6% of baseline (as measured by laser Doppler) can trigger a pulse signal on the Nellcor N-100.

Systolic Blood Pressure Measurement

The combination of a sphygmomanometer cuff and pulse oximeter correlated quite well with Doppler determinations of systolic blood pressure in a study by Korbon et al [92]. They inflated the cuff slowly and noted the blood pressure at the point where pulse signal was lost. Readings were generally within 2 mm Hg of Doppler measurements. Far less accurate were attempts to determine systolic blood pressure by inflating the cuff well past the systolic blood pressure and looking for the onset of a pulse signal as the cuff was slowly deflated. With this technique, the oximeter underestimated systolic blood pressure by about 20 mm Hg, at least partly because several beats must be detected before the oximeter displays an initial value.

A similar study by Wallace et al [93] (with the use of cuff inflation rather than deflation) also claimed good correlation between the Nellcor N-100 and Doppler. In patients under 1 year of age the pulse oximeter averaged 2.4 mm Hg less, while a 4.9 mm Hg shortfall was seen in patients aged 1 year or older.

Properly used, a pulse oximeter seems quite suitable for clinical sphygmomanometry, especially when there is limited space for noninvasive monitoring (as in pediatric cases or extensive burn cases).

Assessing Adequacy of Chest Compressions

In a case reported by Narang [83], the presence of a pulse signal on a pulse oximeter helped confirm adequacy of chest compressions during cardiopulmonary resuscitation in an 11-month-old infant who had a cardiac arrest 5 minutes after induction of general anesthesia. However, it is unknown whether pulsations that trigger the signal are sufficient for successful cardiopulmonary resuscitation, or, indeed, whether absence of such pulsations indicates anything about vital organ flow during circulatory arrest [94]. It is not clear whether pulse oximetry adds anything to the evaluation of chest compressions in cardiopulmonary resuscitation.

Detecting Brachial Artery Compression

Herschman et al [84] used a pulse oximeter to detect inadvertent brachial artery compression during shoulder arthroscopy, with loss of signal taken to imply compromised brachial arterial flow. However, Gibbs et al [95] noted that an adequate pulse signal was consistent with brachial plexus compression, citing the experiences of three volunteers who had brachial paresthesias while maintaining good pulse oximeter waveforms in the affected extremity during traction. This proposed use of the pulse oximeter, therefore, cannot be recommended.

Allen's Test

Nowak et al [85] suggested using the pulse oximeter in Allen's test to assess adequacy of ulnar collateral flow in the hand. They suggested placing the probe on the index finger or thumb and waiting 15 seconds for a pulse signal while the radial artery is occluded. Hovagim et al [86] found a high correlation ($r = 0.94$) between pulse oximetry and visual assessment of palmar reperfusion. With pulse oximeters that have a plethysmographic waveform display, qualitative changes in this waveform with manual radial artery occlusion may aid in the evaluation.

However, other results in this area have been mixed and of uncertain significance. Raju [89] tried Allen's test with and without pulse oximetry and, in his small sample of 20 patients, actually found that the conventional Allen's test is more sensitive. Two of his subjects passed the conventional Allen's test but showed no pulse signal with the oximeter; none showed a good pulse signal and failed the conventional test. On the other hand, Lauer et al [88] found pulse oximetry more sensitive than Allen's test in their 31 patients. None of these studies asks whether the degree of pulsation that triggers a pulse oximeter signal is sufficient to permit radial artery can-

nulation. Kurki et al [96] have shown that radial artery cannulation can interfere with pulse oximetry under some circumstances. At this point, there is no consensus on the use of pulse oximetry compared with Allen's test in assessing ulnar collateral flow.

Locating Vessels for Cannulation

In a 5-year-old patient whose obesity prohibited palpation of the femoral artery for cannulation, Introna and Silverstein [90] attached an oximeter probe to the toe and then probed the groin area manually until the pulse signal was lost. They were able to cannulate the femoral artery "without difficulty" with this method.

Monitoring Vascular Integrity after Replantation

A pulse oximeter probe attached to a replanted (reattached) digit is of some use in monitoring the postoperative vascular integrity of the digit, according to Graham et al [91]. Five of 16 patients had SpO_2 values under 85%, and all 5 were found to have venous occlusion of the graft. The other 11 patients never had SpO_2 readings less than 95%, and all 11 of these had successful replantations.

Assessment of Intestinal Perfusion

A sterile pulse oximeter probe applied directly to the antimesenteric border of a bowel segment was judged by Ferrara et al [97] to be superior to Doppler ultrasound and standard clinical criteria in the assessment of intestinal perfusion in canines. Pulse oximetry was seen as especially attractive due to its low incidence of false-negative readings compared with the other methods, minimizing the morbid consequences of leaving unperfused intestine in situ. The most accurate method of all, intravenous fluorescein [98], has limitations that can be partially overcome by adjunctive pulse oximetry.

Early Detection of Hypovolemia

Cyclical variation in systolic blood pressure has been shown to correlate with hypovolemia and to disappear with volume replacement [99]. Partridge [100] attached a Nellcor N-100 pulse oximeter to a SARAcap end-tidal carbon dioxide monitor to obtain a graphic display of pulse waveform. He showed that the resulting display of systolic pressure variation was more sensitive than central venous pressure as an indicator of volume status, even when a pulse oximeter was used instead of an intraarterial line or impedance plethysmograph (Fig 3). The same sort of measurement is possible with pulse

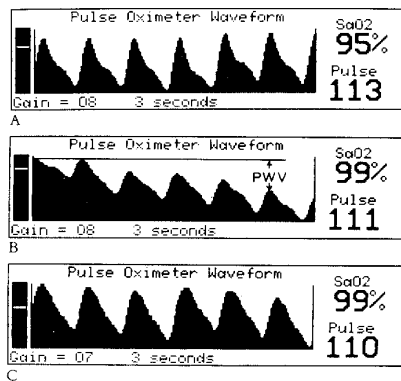


Fig 3. Nellcor/SARAcap display showing evidence of changes in volume status. Top panel: Arrival in operating room; central venous pressure (CVP) 8. Middle panel: Increased pulse waveform variation (PWV) after blood loss and third-spacing; CVP 4–5. Bottom panel: After fluid resuscitation; CVP 8. (From Partridge [100]. Used with permission.)

oximeters with built-in waveform displays, unless the device automatically normalizes the waveform amplitude to the height of the display area.

CLINICAL USES: PEDIATRICS

Studies in critically ill children [101,102]; very-low-birth-weight infants [103]; preterm neonates [104]; neonates with acute cardiopulmonary disease [105–107]; children with croup, epiglottitis, and other upper airway disease [108]; and patients with cyanotic and other congenital heart disease [109–112] have confirmed the acceptability of pulse oximetry in many pediatric settings. Still, pediatrics presents its own set of monitoring requirements and problems [113]. In children under 2 years of age desaturation occurs more often intraoperatively than in other patients [50]. The neonate, in particular, is smaller, carries his or her own brand of hemoglobin, desaturates faster, may present with congenital heart defects, an alternate circulation or both, and is at risk for a unique complication of hyperoxemia.

Effect of Fetal Hemoglobin

Hemoglobin F constitutes 50 to 85% of the total hemoglobin at birth, less than 15% at 1 year, and somewhat less than 2% into childhood and later [114], barring the odd case of hereditary persistence of hemoglobin F into adulthood (approximately 20%) [115]. Because pulse oximeters are calibrated on adults, the question arises as to whether pulse oximetry is accurate on neonates.

The IL-282 in vitro oximeter is known to report

falsely elevated carboxyhemoglobin levels in the presence of hemoglobin F [116], requiring an empirical “Cornelissen correction,” widely used in the literature [117]. Although three of the four wavelengths used in in vitro oximetry are below 600 nm and hence outside the range of interest, this example does raise the possibility that pulse oximetry may be affected by hemoglobin F as well.

This issue is conceptually distinct from the well-known difference in oxygen affinity between hemoglobins F and A. The pulse oximeter can be expected to perform just as accurately when hemoglobin’s oxygen affinity is altered due to hypercapnia, acidosis, changes in 2,3-diphosphoglycerate concentration, or temperature changes. The question is whether hemoglobin F absorbs light, specifically 660- and 940-nm light, sufficiently differently from hemoglobin A to be misinterpreted by the pulse oximeter as oxidized hemoglobin A, reduced hemoglobin A, or some combination of the two.

Most of the theoretical and experimental evidence suggests that there is no clinically significant effect of hemoglobin F on pulse oximetry. The prosthetic group of heme, ferroprotoporphyrin IX, is invariant across all vertebrate species and is identical in hemoglobins A and F [118]. Inspection of the small differences between the absorption spectra of hemoglobins A and F in the range of interest (630 to 960 nm) suggests that they are identical for practical purposes [119]. In a recent trial, a pulse oximeter algorithm was given hemoglobin F saturation data rather than those of hemoglobin A. The resulting SpO₂ values differed generally by less than 1%, and at most by 1.12%, from the corresponding results for hemoglobin A [120]. Some clinical investigations in neonates likewise show no significant effect of hemoglobin F on pulse oximetry [103,121].

Jennis and Peabody [37] found that the higher the proportion of hemoglobin F in the blood, the greater the difference between pulse oximetry (Nellcor N-100) and in vitro oximetry (IL-282). For example, in neonates with 0 to 24% hemoglobin F, the difference between SaO₂ and SpO₂ was $0.3\% \pm 1.9\%$; with 75 to 100% hemoglobin F, it was $3.6\% \pm 2.3\%$. Possibly confounding these results, however, was a tendency to sample at higher saturations in the subjects with higher percentages of hemoglobin F. It is also possible that the Cornelissen correction is inadequate at the high percentages of hemoglobin F seen here [122].

Ramanathan et al [103] claim that hemoglobin F has no effect on pulse oximeter accuracy, but they examined only the degrees of correlation between pulse (Nellcor N-100) and in vitro oximetry (0.87 for hemoglobin F greater than 50%; 0.89 for hemoglobin F less than

50%). The mean difference between SaO_2 and SpO_2 , with standard deviation, would have been useful information in this study.

Pending further studies on hemoglobin F and patients containing it, the question of whether hemoglobin F causes inaccuracies in pulse oximetry remains open. It is likely that the error, if any, is small—only a few percentage points at most. For the detection of dangerous desaturation trends in neonates, such inaccuracy would be almost irrelevant clinically (see also the Performance and Limitations section). It may have some bearing, however, on the prevention of hyperoxemic retrolental fibroplasia.

Reducing Risk of Retrolental Fibroplasia

In premature neonates, the administration of supplemental oxygen is associated with retrolental fibroplasia [123]. The duration, concentration, and pattern of oxygen administration are all implicated, as is gestational age. Because any FiO_2 above that of room air is associated with risk for retrolental fibroplasia, and because the cause of the disorder is multifactorial [124], there is no consensus on the ideal management of neonatal oxygenation to avoid this problem [125], but recommendations for SaO_2 generally fall into the range of 90 to 95% [37,102,106,126–130]. Some authors argue for a somewhat lower range [125].

The pulse oximeter has obvious application here, as FiO_2 is titrated to observe this guideline. However, its main utility is in preventing hypoxia, not hyperoxia, during these adjustments. In the steep portion of the hemoglobin-oxygen dissociation curve below an SaO_2 of 90%, saturation is more sensitive than PO_2 in assessing oxygenation. But above an SaO_2 of 90%, the curve flattens out, and an error of a few percent SaO_2 could represent a large error in PO_2 , especially at the higher values.

It is generally agreed that pulse oximetry is not adequate to evaluate hyperoxia in this setting. Baeckert et al [131] found that the optimum combination of sensitivity and specificity for hyperoxia in neonates (PO_2 greater than 90 mm Hg) was 70% and 62%, respectively; this was seen with a cutoff point for SaO_2 at 92%. Bossi et al [132], using a cutoff SaO_2 of 93% in neonates, found excellent sensitivity to hyperoxemia (PO_2 greater than 100 mm Hg), but a predictive value of only 25%. Both studies used the Ohmeda Biox 3700. Hodgson et al [133] have demonstrated that the 95% prediction interval for an SpO_2 of 90% is an SaO_2 range of 85 to 98%, the upper end of which exceeds all known recommendations for SaO_2 in infants at risk for retrolental fibroplasia.

Retrolental fibroplasia is probably best prevented by using direct measurement of PO_2 . This can be done either periodically via blood gas analysis or continuously, if clinical conditions are conducive, via transcutaneous PO_2 monitoring. The relative merits of transcutaneous PO_2 monitoring and pulse oximetry have been well reviewed [24,36–38,126,134,135].

Pediatric Intraoperative Peculiarities

The tendency in infants and children for desaturation to occur precipitously during airway management at induction [136], the threat of inadvertent return to a fetal circulation (via reopening of the ductus arteriosus), and the occasional presence of congenital heart defects contribute to the indications for intraoperative pulse oximetry in pediatric anesthesia. Representative case reports documenting the benefit include a Blalock-Taussig shunt for pulmonary atresia, where desaturation was anticipated while a pulmonary artery was clamped [137]; repair of a ventricular septal defect during which SaO_2 was maintained in the recommended range of 87 to 92% during pulmonary artery banding [137]; and repair of a congenital diaphragmatic hernia, where SpO_2 was maintained above 60% as ventilatory variables were adjusted successfully despite decreased functional reserve capacity (after replacement of abdominal contents and wound closure) [138]. Numerous other examples can be cited [139,139a].

Neonatal Asphyxia

Marked changes in SaO_2 are common in the infant immediately after delivery [140], and the pulse oximeter can help assess these [141]. The average SpO_2 at 1 minute after delivery was 59% in a study by House et al [142] of 100 infants (62 delivered by cesarean section). Twenty-five percent of the infants observed were transiently below an SpO_2 of 50%, and in 10% of them SpO_2 dipped below 30%. SpO_2 correlated well with the Apgar scores.

Sudden Infant Death Syndrome

The pulse oximeter has been suggested as a noninvasive adjunct to home apnea and cardiac monitors for children known to be at risk for sudden infant death syndrome [27], but false alarms due to motion artifact are a potential problem [143,144], as is expense.

Pediatric Radiation Therapy

Pediatric patients who require general anesthesia for immobilization during radiation therapy need to be ob-

served and monitored from outside the room while the radiation dose is administered. The pulse oximeter has been described as a useful supplement to observation of chest wall and bag movement, which is often difficult to discern [145].

PERFORMANCE AND LIMITATIONS

Investigations into the performance and limitations of pulse oximetry are being pursued as energetically and in as many directions as are studies of its realm of application. The review of New [36] of the limitations of pulse oximetry is perhaps the best brief one.

How accurate is pulse oximetry? In some respects this question was settled in the relatively early days of its clinical use—say, 1983 [25]. In other ways the question remains a matter for active investigation, as in the realm of profound arterial desaturation (where the Byzantine machinations necessary for data collection may cast some doubt on the clinical pertinence of the findings), or in the narrower areas of dyshemoglobin and vital dye effects on pulse oximetry.

Given the degree of accuracy we may expect from pulse oximetry, what kinds of conditions threaten this accuracy? These concerns range from the profound (low-perfusion states) to the mundane (nail polish), and the full spectrum has been addressed in the literature.

Accuracy

Because pulse oximeters are calibrated empirically by using observations taken from healthy volunteers, it is not surprising that Yelderman and New [25] and others after them have generally found pulse oximeters to be accurate to within 5% (± 2 SD) of in vitro oximetry in the SaO_2 range of 70 to 100%, as long as none of the limitations listed below apply [146]. This is in sharp contrast to the results at an SaO_2 less than 70%, where no empirical correlation exists.

Although this degree of accuracy is generally believed to be acceptable for many applications, such as the assessment of intraoperative oxygenation trends or adequacy of oxygen therapy, it is not adequate for other applications, such as the prevention of retrolental fibroplasia or the calculation of right-to-left shunt in cyanotic heart disease. The acceptability of SpO_2 results in a given setting is generally a matter of clinical judgment.

The Table summarizes the results from selected investigations into the accuracy of pulse oximetry. In general, these results demonstrate the relative acceptability of pulse oximetry readings in the SaO_2 range of 70 to

100% (for many clinical purposes), while an SpO_2 below this range is relatively and sometimes drastically inaccurate. In studies comparing ear and finger probes, readings from the ear are often slightly more accurate; the possible roles of circulation time, probe specifications, dependency of the probe location, variations in cutaneous vasculature, and other factors that may explain this phenomenon remain to be determined.

Although variations in performance between manufacturers are sometimes seen, it is more common to find no significant difference within a given set of makes and models; moreover, when such a difference is found, the degree of reproducibility of the result is not always clear. Assessments of pulse oximeter performance based on investigational reports are further complicated by the tendency of manufacturers to revise software frequently for a given model, sometimes in response to the report itself [154,155,163]. For example, the Ohmeda company modified its Biox 3700 pulse oximeter, upgrading its Version J software to Version XJ1, at least partly in response to published investigational data [154,166]. However, most investigators do not specify which software versions they are evaluating, nor can manufacturers always be relied on to announce software upgrades. In view of this, the disclaimer of Nickerson et al [163], cautioning readers to use their data “to understand the validity of methods for measuring oxygen saturation, and not to decide which oximeter is best,” deserves wide application in this area.

The choice of an SaO_2 standard may also affect outcome. The most popular standard, and also the one most widely accepted as the “gold standard” for pulse oximeter assessment, is the IL282 CO-oximeter. This is a four-wavelength in vitro oximeter capable of discriminating between hemoglobin, oxyhemoglobin, carboxyhemoglobin, and methemoglobin. The Radiometer OSM-3 multiwavelength bench oximeter and the Corning C-2500 CO-oximeter, among others, are also used. These instruments measure SaO_2 directly.

On the other hand, blood gas analyzers, which measure Po_2 directly and then use pH and temperature data to calculate SaO_2 , are subject to potentially significant inaccuracies due to shifts in the oxyhemoglobin dissociation curve. The one study that simultaneously compared pulse oximeters, a blood gas analyzer, and a CO-oximeter found a 1 to 2% difference in accuracy and a precision of 1 to 3% between the latter two instruments at physiologic SaO_2 in healthy volunteers [163]. (Unless special precautions are taken, pulse oximeters are more accurate than blood gas analyzers when the oxygen consumption rate of a blood gas sample is accelerated [“pseudohypoxemia”], as in some cases of leukemia and thrombocytosis [167].)

Accuracy of Pulse Oximetry^a

Year of Publication: Investigator(s)	Oximeter Model (Software Version)	Probe Location	Study Population [ST]	No. of Data Sets (Subjects)	r	SEE	% Bias ^b	% SD (95% Tolerance)	% Range of SaO ₂ ^c (Standard)
1983									
Yelderman and New [25]	NE N-100	Finger	AV	79 (5)	0.98	1.83			65–100(IL282)
1984									
Kim et al [147]	MI SM32	Finger, ear	ICU	21 (21)	0.71				80–100
Deckardt and Steward [130]	NE N-100	Various	PI	52 (31)					40–100
1985									
Mihm and Halperin [148]	NE N-100	Finger	AI	131 (18)	0.96				56–100(IL282)
Mackenzie [9]	NE N-100	Finger	AV	277 (11)	0.98				65–100(IL282)
Fanconi et al [102]	NE N-100	Various	NPICU	108 (36)	0.95		+1.51	3.49	58–100(IL182)
Fair et al [101]	NE N-100	Various	PICU	192 (15)	0.89				70–100(IL282)
1986									
Tytler and Seeley [149]	NE N-101	Finger	AI	72			+0.4	1.2 (0.7)	70–100(OSM-2)
Dautzenberg et al [150]			AI	200 (200)	0.76		–2.0	3.3	85–100
Miyasaka [136]	BI III	Various	NPICU	100	0.96				40–100(C2500)
Chapman et al [151]	BI II		AV [S]	117 (8)	0.96	2.72			55–100(IL282)
Lynn and Bosenberg [152]	NE N-100	Various	PI (CCHD)	47	0.94	4.0			55–90(OSM-2)
	NE N-100	Various	PI (CCHD)	133 (205)	0.95	3.3			55–100(OSM-2)
1987									
Southall et al [38]	NE N-101	Various	NPICU	92 (43)	0.89		+2.6	2.4	70–100(C2500 or IL282)
Lafeber et al [153]	NE N-101	Various	NPICU	70 (8)	0.80				30–100(OSM-2)
Hay et al [107]	OH 3700	Various	NICU	73 (142)	0.98				70–100(OSM-2)
Fanconi [127] ^d		Various	NPICU	178 (54)	0.95		–2.74	7.69	30–100(C2500 or OSM-2)
Baeckert et al [131] ^e	OH 3700	Various	NICU						70–100(C2500)
Bossi et al [132]	OH 3700	Various	NICU	15	0.94				40–85(IL282)
	OH 3700	Various	NICU	52	0.01				93–100(IL282)
Hodgson et al [133]	NE N-100	Various	NICU	64 (16)			–0.8	1.8 (13.0)	85–100(IL282)
Kagle et al [154]	OH 3700 (J)	Ear	AV [S]	40 (8)	0.98			(±8%) ^f	60–100(C2500)
	OH 3700 (J)	Finger	AV [S]	88 (8)	0.98			(±8%) ^f	60–100(C2500)
	OH 3700 (XJ) ^l	Finger	AV [S]	48 (8)	0.99				60–100(C2500)
	NE N-100	Finger	AV [S]	48 (8)	0.99			(±8%) ^f	60–100(C2500)
	OH 3700 (J)	Ear	AV [T]	24 (8)	0.96				60–100(C2500)
	OH 3700 (J)	Finger	AV [T]	24 (8)	0.78				60–100(C2500)
Viitanen et al [67]	NE N-100	Finger	AI (OLV)	90 (10)	0.93				88–100(IL282)
Ramanathan et al [103]	NE N-100	Various	NICU (RDS)	88 (44)	0.88	1.9	+0.3	2.1	78–100(IL282)
	NE N-100	Various	NICU (BD)	48 (24)	0.90	1.9	–2.7	1.9	79–100(IL282)
Jennis and Peabody [37] ^g	NE N-100	Extremity ^h	NICU	177 (26)	0.90	1.87	+1.7	2.2	70–100(IL282)
	NE N-100	Extremity ^h	NICU (BI)	25 (5)		–2.00	3.0		(IL282)
Lazzell and Jopling [109] (abstract)	NE N-100	Various	PI	26 (9)	0.97	4.1			30–85(IL282)
Severinghaus and Naifeh [155]	NE N-100	Ear	AV [S]	60 (10)			+0.4	11.7	40–70(OSM-3)
	NE N-100	Finger	AV [S]	60 (10)			+6.6	10.8	40–70(OSM-3)
	NE N-200	Ear	AV [S]	58			+2.4	8.7	40–70(OSM-3)
	NE N-200	Finger	AV [S]	58			+4.5	6.2	40–70(OSM-3)
	OH 3700	Ear	AV [S]	60 (10)			–2.4	8.8	40–70(OSM-3)
	OH 3700	Ear	AV [S]	60			–2.4	8.8	40–70(OSM-3)
	OH 3700	Finger	AV [S]	60 (10)			+9.0	10.4	40–70(OSM-3)
	OH 3700	Finger	AV [S]	60			–2.7	5.8	40–70(OSM-3)
	NO 500 (2.2)	Ear	AV [S]	60 (10)			–4.8	13.9	40–70(OSM-3)
	NO 500 (2.2)	Finger	AV [S]	60 (10)			–13.1	12.7	40–70(OSM-3)
	NO 500 (3.3)	Finger	AV [S]	120 (10)			–1.1	5.4	40–70(OSM-3)
	CR 501 + (0.27)	Ear	AV [S]	55 (10)			–12.0	13.3	40–70(OSM-3)
	CR 501 + (0.27)	Finger	AV [S]	60 (10)			+8.1	16.0	40–70(OSM-3)
	CR 501 + (0.28)	Ear	AV [S]	36 (6)			0.0	3.4	40–70(OSM-3)
	CR 501 + (0.28) ⁱ	Ear	AV [S]	60			–4.4	4.4	40–70(OSM-3)
	CR 501 + (0.28) ⁱ	Finger	AV [S]	60 (6)			+1.9	4.8	40–70(OSM-3)
	CR 501 + (0.28) ⁱ	Finger	AV [S]	60			–1.4	5.9	40–70(OSM-3)
	PC 1600	Ear	AV [S]	54 (9)			–4.3	4.3	40–70(OSM-3)
	PC 1600	Ear	AV [S]	57			–2.9	4.3	40–70(OSM-3)
	PC 1600	Finger	AV [S]	73 (9)			+7.9	5.1	40–70(OSM-3)
	PC 1600	Finger	AV [S]	60			0.0	3.5	40–70(OSM-3)
	MQ 7	Finger	AV [S]	36 (6)			+2.9	5.2	40–70(OSM-3)
	DX	Ear	AV [S]	60			+4.9	5.7	40–70(OSM-3)
	DX	Finger	AV [S]	59			+1.6	5.4	40–70(OSM-3)
Sidi et al [156]	OH 3700 (J) ^j	Tongue	Canine	42 (10)	0.93				80–100(IL282)
	OH 3700 (J) ^j	Tongue	Canine	108 (10)	0.71				0–80(IL282)
	NE N-100	Tongue	Canine	58 (10)	0.79				80–100(IL282)
	NE N-100	Tongue	Canine	132 (10)	0.62				0–80(IL282)

Accuracy of Pulse Oximetry^a (continued)

Year of Publication: Investigator(s)	Oximeter Model (Software Version)	Probe Location	Study Population [ST]	No. of Data Sets (Subjects)	<i>r</i>	SEE	% Bias ^b	% SD (95% Tolerance)	% Range of SaO ₂ ^c (Standard)
Boxer et al [112]	NE N-100	Various	PI (CCHD)	46 (32)			-0.56	2.33	80-100(IL282)
	NE N-100	Various	PI (CCHD)	62 (32)			-1.09	4.46	40-80(IL282)
Gussack and Tacchi [108]		Extremity ^h	PICU	30 (18)	0.98	0.63			85-100
Warley et al [157]	OH 3700	Ear	AI	65 (10)			+0.1	3.4	60-100(ABL-2)
	OH 3700	Finger ^k	AI	65 (10)			+0.4	1.8	60-100(ABL-2)
	OH 3700	Finger	AI	65 (10)			+0.5	2.9	60-100(ABL-2)
Walsh et al [158]	NE N-100	Various	NICU	63 (21)	0.93				45-100(OM-II)
1988									
Sendak et al [159]	NE N-100	Tongue	Canine	46 (5)	0.98	3.9	-5.5	4.2	22-100(IL282)
	NE N-100	Tongue	Canine	66 (5)	0.54	8.1	-5.9	8.1	8-22(IL282)
Mendelson et al [160]	DA	Finger	AI [S]	135 (15)	0.99	1.29			65-100(IL282)
Cecil et al [161]	NE N-100	Finger	AI	330 (152)	0.80		-0.59	(12.7)	75-100(IL282)
	OH 3700	Finger	AI	333 (152)	0.83		+0.31	(10.3)	75-100(IL282)
	OH 3700	Finger	AI [BI]	43 (15)	0.81			(13.5)	75-100(IL282)
	NE N-100	Finger	AI [BI]	43 (15)	0.79			(15.1)	75-100(IL282)
Fanconi [162]	NE N-100	Various	NPICU	160 (20)			-4.53	7.86	30-95(OSM-2)
Nickerson et al [163]	OH 3700	Finger	AI	165 (5)			+2.6	2.1	65-100(IL282)
	CR 501	Finger	AI	165 (5)			+1.0	2.8	65-100(IL282)
	NE N-100	Finger	AI	165 (5)			+0.4	1.7	65-100(IL282)
	NO 500	Finger	AI	165 (5)			+1.0	1.6	65-100(IL282)
Ridley [111]	NE N-100	Various	PI	114 (25)			+0.7	3.1	50-100(OSM-2)
	OH 3700	Various	PI	114 (25)			+2.8	3.9	45-100(OSM-2)
Taylor and Whitwam [164]	BR 4400	Finger	AI	~70 (20)	0.98		+2.34		70-100(IL282)
	CR 501	Finger	AI	~70 (20)	0.97		+2.0		70-100(IL282)
	NO 500	Finger	AI	~70 (20)	0.88		+2.8		65-100(IL282)
	OH 3700	Finger	AI	~70 (20)	0.98		+2.4		65-100(IL282)
	NE N-100	Finger	AI	~70 (20)	0.98		+2.0		80-100(IL282)
Gabrielczyk and Buist [165]	NE N-100	Finger	AI [H]	68 (20)			-0.6	1.6	90-100(OSM-2)
Jones et al [79]	OH 3700	Finger	AI	52 (40)	0.95		-1.86	3.1	50-100(IL282)

^aData are from selected publications. [ST] represents steady-state [S] or transient [T] arterial oxygen saturation (SaO₂) values. SEE = standard error of the estimate; SD = standard deviation; 95% tolerance represents the range for pulse oximeter (SpO₂) readings of SaO₂. Oximeters were manufactured by Nellcor (NE), Minolta (MI), Ohmeda Biox (BI), Ohmeda (OH), Novamatrix (NO), Criticare (CR), Physio-Control (PC), Marquest (MQ), Datex (DX), Datascope (DA), and Bird (BR). Study populations comprised adult volunteers (AV), adult intensive care unit patients (ICU), pediatric inpatients (PI), adult inpatients (AI), neonatal and pediatric intensive care unit patients (NPICU), pediatric intensive care unit patients (PICU), and neonatal intensive care unit patients (NICU). CCHD = cyanotic congenital heart disease; OLV = one-lung ventilation; RDS = respiratory distress syndrome; BD = bronchopulmonary dysplasia; BI = black subjects; H = hypothermic subjects. SaO₂ standards were the Radiometer ABL-2 and OM-II blood gas analyzers, OSM-2 hemoximeter, and OSM-3 bench oximeter; the Corning C-2500 CO-oximeter; and the Instrumentation Laboratory IL182 and IL282 CO-oximeters.

^bExpressed as (SaO₂ - SpO₂); some studies report (SpO₂ - SaO₂).

^cSaO₂ values generally are weighted toward the high end of the reported SaO₂ range, except in [155] and in the canine studies [156, 159].

^dPooled data [102, 162].

^eReports only statistics related to the regression of bias on SaO₂.

^f99% tolerance (prediction) limits were noted to be within $\pm 8\%$.

^gExamines differential effect of hemoglobin F on SpO₂. (See text.)

^hHand, foot, finger, or toe.

ⁱData from Criticare model 502 also included.

^j"Good-quality" data (i.e., low-quality signal message absent) only.

^kProbe location: finger; probe type: flex probe (versus finger probe).

The typical study assessing pulse oximeter performance compares SpO₂ with an in vitro oximeter, yielding a graph resembling Figure 2. The correlation coefficient and regression line equation are often noted. These are useful, but there are better statistics in a study of this type: *bias*, or error, which is the mean value of (SaO₂ - SpO₂), and *precision*, which is the standard deviation of the bias. These figures, instead of merely assuring the user that the correlation is "good" or "excellent" (whatever these may mean), tell him or her what to expect from a specific SpO₂ reading clinically encountered [154,168-170]. Only in the past year or two have investigators offered these statistics regularly (see the Table).

The data collected in these studies tend to be weighted toward the higher saturation values. Even studies attempting to examine performance at very low saturation values sometimes include a preponderance of data above SaO₂ values of 85 or 90%. Correlation coefficients determined from such samples tend to underestimate the true degree of correlation [6]. In practice, this is rarely a problem, as the claimed correlation coefficients in these experiments are almost always above 0.85. In any case, one does not always know how to translate correlation coefficients into clinical import.

When statistical calculations include data both above and below SaO₂ values of 70%, the methodology must

be regarded carefully. Pulse oximeters are calibrated with empirical observations, generally above an SaO_2 of 70%. Data on either side of this breaking point are best treated separately, lest overall accuracy be over- or underestimated, depending on the mix. It may also be helpful to regress bias ($\text{SaO}_2 - \text{SpO}_2$ or vice versa) on SaO_2 , to determine whether bias worsens with decreasing SaO_2 ; if no particular range of SaO_2 is especially inaccurate, a regression slope of zero would be expected [162].

Regarding accuracy at very high saturation values, it is interesting to note that the most commonly displayed SpO_2 by the Nellcor N-100 when the subject is breathing 50% oxygen is 100%, while the value most commonly displayed by the Novamatrix 500 under similar conditions is 98% and the most common value obtained by a Corning 2500 in vitro CO-oximeter is 96% [51]. These are incidental findings in a study in which degree of pulmonary disease and other relevant factors were not controlled, but they may represent software differences between manufacturers. For example, calibration using fractional rather than functional saturation values (see the Dyshemoglobins section) might account for such differences.

Accuracy at Very Low Saturations

An active area of current research involves the accuracy of pulse oximeters at very low SaO_2 values, that is, below 70%. The algorithms for deriving SaO_2 from absorption data are derived experimentally, not theoretically (see the Technology section). Because humans cannot casually be taken to SaO_2 values below 70%, manufacturers have until recently had scant basis for SpO_2 values when SaO_2 is much below 70%; hence the suspicion that pulse oximeters may be inaccurate at very low SaO_2 values.

Pologe [29] offers an entirely different reason to be concerned about inaccuracy at low SaO_2 values. State-of-the-art LEDs, as used in pulse oximetry, may deviate from the desired wavelength by a few nanometers; an LED designed to transmit 660-nm light may well transmit at 650 nm instead. Such variations could be expected to affect calculations most where the absorption curves are steepest, and reduced hemoglobin happens to have a steeper curve at 660 nm (virtually a universal wavelength for pulse oximetry) than does oxyhemoglobin at 660 nm, or than does either species in the 910- to 940-nm range. Therefore, desaturated arterial blood may be more susceptible to inaccuracy than well-saturated arterial blood; the amount of inaccuracy would vary with each individual pulse oximetry unit.

An occasional article has presented data from hu-

mans who have attained SaO_2 values below 70% in clinical settings. For example, Mihm and Halperin [148] claimed good correlation between Nellcor N-100 and in vitro oximetry for "profound desaturations," although their data included only a few points between SaO_2 values of 60 and 70%, with most above 90%, and without separate statistical treatment for the very low range. Since 70% is roughly the cutoff for availability of empirical data, such treatment would seem to be mandatory for proper evaluation of performance at lower SaO_2 values. Another few data points from an even lower range are reported by Lazzell and Jopling [109], Boxer et al [112], and Lafeber et al [153] from observations in children with cyanotic congenital heart disease and bronchopulmonary dysplasia, with similar conclusions. Fanconi [162] offers 160 data sets (from 20 infants using a Nellcor N-100), nearly half of which are from an SpO_2 less than 60%; he finds an unacceptably high bias and low precision of $-4.53\% \pm 7.86\%$, with error increasing as SpO_2 decreases ($r = 0.69$ for regression of bias on SaO_2).

Others have turned to animals to explore this terra incognita. (The general acceptability of canine results, with proper recalibration, is discussed in the Dyshemoglobins section.) Sidi et al [156] took dogs down to very low saturation values (essentially the 0 to 70% range) and found that both the Nellcor N-100 and the Ohmeda Biox III (software version J) overestimate SaO_2 in that range. Given their regression lines, the two oximeters theoretically would report SpO_2 values of 21% and 33%, respectively, for an SaO_2 of 0% (Fig 4A). On the other hand, Sendak et al [159] found the Nellcor N-100 very accurate in dogs at equally low saturations (Fig 4B).

The most elaborate studies in this area have maintained volunteers at very low steady-state SaO_2 values before taking measurements. The steady state is important because oximeters average the past several seconds' data in calculating SpO_2 , and clinically occurring desaturations often are so transient that this lag can cause overestimation of SpO_2 during early desaturation. Kagle et al [154], the first to report on this technique, concluded that within the 60 to 100% range the SpO_2 displayed by the Ohmeda Biox 3700 is within 8% of the actual SaO_2 with a 99% confidence, but found no increased tendency to inaccuracy at the lower saturation values. However, their analysis does not give separate statistical treatment to the well-calibrated range above 70% and the uncalibrated range below 70%.

The most extensive and ingenious tests on human steady-state very low SaO_2 measurements are those of Severinghaus and Naifeh [155]. By using a feedback loop containing a mass spectrometer for end-tidal car-

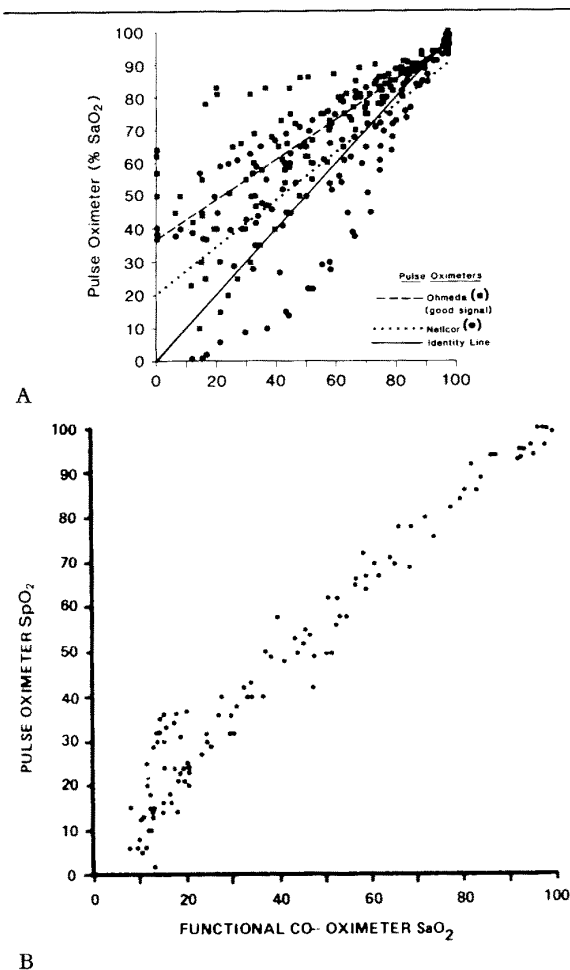


Fig 4. (A) Results of canine study showing overestimation of arterial oxygen saturation (SaO₂) and loss of precision at very low saturation values. (Reprinted from [156] by permission.) (B) Results of canine study showing good correlation between pulse oximeter arterial oxygen saturation (SpO₂) and SaO₂ at all but the lowest saturation values. (From Sendak et al [159]. Used with permission.)

bon dioxide, a computer to calculate pulmonary capillary oxygen saturation, and a human operator to titrate FiO₂ to a constant capillary oxygen saturation, they achieved high-quality steady-state plateaus at low saturations. They rated a wide variety of pulse oximeters, including the Nellcor N-100 and N-200, the Ohmeda Biox 3700, the Novamatrix 500 (versions 2.2 and 2.3), the Datex, the Criticare CSI 501+ and 502, the Physio-Control Lifestat 1600, and the Marquest/Minolta PulseOx 7, usually with both finger and ear probes. Their study is also important for exploring only the range of 40 to 70%, and not higher ranges, where em-

pirical observations are already incorporated into the algorithms of pulse oximeters.

In this study, pulse oximeters generally underestimated SaO₂ values in the 40 to 70% range by roughly 0 to 9%, as opposed to the canine studies of Sidi et al [156], in which SpO₂ overestimated SaO₂. The discrepancy may have been because Severinghaus and Naifeh used steady-state measurements, whereas Sidi et al measured transient values. Severinghaus and Naifeh also found ear probes to be more accurate than finger probes—an effect not explicable by shorter circulation time to the ear, since steady-state measurements were performed. The accuracies of ear and finger probes may depend in part on the type of probe originally used to calibrate the device.

Although noticeable variations were found between certain pulse oximeter models in the study of Severinghaus and Naifeh (see the Table), their significance is uncertain. An appendix to their article presents the results of a second set of experiments, presumably using the same methods, on some of the same oximeters. (The report does not state whether the second set used the same individual instruments as the first, or merely different instruments of the same make and model.) In some cases, the results varied considerably, as with the Ohmeda Biox 3700 and Physio-Control 1600, both with finger probes. These differences suggest that the respective accuracies of various pulse oximeter models at very low SaO₂ values may not yet have been compared in a reproducible way.

The study also uncovered a few idiosyncrasies in particular models. For example, the Nellcor N-100 overresponds to, that is, underestimates, SaO₂ more than do most models in the early stages of profound desaturation (which are the most clinically relevant). The Ohmeda Biox 3700 sometimes shows an unexplained brief reversal ("hiccup") during desaturation as the 85% mark is passed. It is not known whether these responses were observed in one or several instruments.

Interestingly, experimental subjects seem to tolerate brief periods of steady-state hypoxemia well. The subjects described by Kagle et al [154] had no changes in electrocardiogram or mental status and no chest pain at an SaO₂ of 60%. The subjects of Severinghaus and Naifeh [155] went to as low as an SaO₂ of 40% for 30 to 60 seconds; although none were distressed or lost consciousness, some felt dizzy, experienced tingling in the extremities, or had fluttering eyelids at the end of the period of hypoxemia.

Steady-state studies of pulse oximeter performance at very low saturation values may have at best only indirect clinical relevance—as demonstrated by the investigators' need to go to artificial lengths just to create the

steady-state conditions needed to perform them. Few, if any, decisions about the treatment of hypoxemia turn on whether the SaO_2 is 50% or 60% at a given instant. Far more relevant clinically is the pulse oximeter's ability to indicate the presence or absence of a desaturation trend. Subject to the limitations discussed herein, virtually all models of pulse oximeter have this ability.

Response Time

Pulse oximeter response time depends on both inherent machine characteristics and circulation time. Most models average pulse data over the past 5 to 8 seconds before displaying a value. This inherent lag may be overridden in some machines in favor of either a shorter averaging period or an overt beat-by-beat display.

Aside from the obvious dependence on hemodynamic status, circulation time also varies with pulse oximeter probe location. For example, in the study of Severinghaus and Naifeh [155], ear probes for five models (all using their default delays of 5 to 8 seconds) had average desaturation response times ranging from 9.6 to 19.8 seconds; finger probe average response times ranged from 24.0 to 35.1 seconds. Warley et al [157], using an Ohmeda Biox 3700, found ear, finger, and toe response times of 9.8 ± 2.6 , 23.6 ± 4.1 , and 56.8 ± 15.8 seconds, respectively, in 10 subjects, illustrating the significance of probe location.

Pulse oximeters respond to changes in oxygenation considerably faster than do transcutaneous PO_2 monitors [67]. One study found a 50% time constant of 11.2 seconds for the response of the Ohmeda Biox 3700 with finger probe, compared with 57.6 seconds for a transcutaneous PO_2 monitor [171].

Low-Amplitude States

Pulse oximeters cannot report data unless they detect an adequate pulse. Thus, if a peripheral pulse is absent (cardiac arrest or sphygmomanometer cuff inflation proximally), or of low amplitude (hypovolemia, hypotension, hypothermia, vasoconstrictor infusions, or cardiac bypass), readings are intermittent or unavailable. Unfortunately, accurate pulse oximeter readings are especially desirable in just these situations. For example, the monitor generally is unreliable during cardiac arrest, although it has on occasion been used to assess the adequacy of chest compressions in cardiopulmonary resuscitation [83]. Locally applied vasodilating creams sometimes are recommended to enhance the pulse signal in low-amplitude states [172].

Where a finger probe fails, sometimes an ear probe can succeed. A special probe is available for the anterior

nasal septal artery (a branch of the ethmoidal artery), another sensitive location. A photoplethysmographic study of the ear lobe, nail bed, finger pad, and nasal septum has found the ear lobe to be the least vasoactive site, and therefore the least susceptible to pulse signal loss due to vasoconstriction [173]. The tongue, long used in dogs, is currently being developed as a monitoring site in humans when other sites will not do. It is said to be unusually resistant to signal interference from electrocautery as well, and sensor modifications can be made by using easily available equipment [174]. The forehead has also recently been found acceptable as a monitoring site in healthy subjects, but proved less reliable and more difficult to use in critically ill patients [175].

The only published study of pulse oximetry in post-operative hypothermia (32.3 to 35.0°C) found no significant loss of accuracy under these conditions (bias, 0.6%; precision, 1.6%), although signal failure occurred in 2 of the 21 patients. One of these 2, the coldest subject studied (32.3°C), regained a pulse signal when a nitroprusside infusion was increased from 0.5 to 1.0 $\mu\text{g}/\text{kg}/\text{min}$. Eight of the patients were receiving nitroprusside or nitroglycerin infusions [165].

The Ohmeda Biox III has been called more sensitive to pulse under low-amplitude conditions than is the Nellcor N-100 [176], and at least one investigation (during cardiopulmonary bypass) seems to suggest this [72]. The Ohmeda Biox 3700, which displays a "low quality signal" message when perfusion is poor, continues to display accurate SpO_2 values throughout progressive arterial occlusion (by sphygmomanometer cuff) up until the appearance of this message. However, when perfusion is poor enough to cause display of this message, motion may produce an artifactual waveform and cause an erroneous SpO_2 display without the message [157]. Indeed, pulse oximeters are more sensitive to motion artifact under low-amplitude conditions.

Lawson et al [94] have shown that peripheral flows of 4.0 to 8.6% of baseline can trigger a signal on a pulse oximeter. It is not yet clear whether other values would be obtained under types of low-amplitude conditions other than outright arterial occlusion.

Venous Congestion

An imaginative study by Kim et al [177] suggests that the pulse of blood sensed by the pulse oximeter, although arterial in content, is actually physically present in veins, having been shunted there by the systolic pressure wave through the arteriovenous anastomoses that are unique to the cutaneous circulation.

If so, SpO_2 is especially vulnerable to artifact from

venous congestion. If the probe is placed on a dependent extremity, some of the pulse wave may actually contain venous blood admixed with arterialized. Kim et al showed that dependent extremities do indeed have lower SpO_2 values than do elevated ones. They also showed that the finger tip is a better place for the probe than the base of the finger; veins at the finger base receive not only arterial blood through the cutaneous anastomoses, but venous drainage from digital musculature. In an otherwise unrelated study, however, Viitanen et al [67] noted incidentally and anecdotally that simultaneous measurements in dependent and nondependent extremities in the same patient showed no difference in SpO_2 .

Coughing sends pressure waves through the venous system, causing artifactual "pulses." These transiently perturb the displayed SpO_2 , which represents an average of the past several pulses [29].

Anemia

There is some evidence that severe anemia affects pulse oximetry. The Nellcor N-100 slightly underestimated SaO_2 , and suffered in precision as well, in a small ($n = 7$) sample of euvolemic dogs with hematocrits under 10%. No adverse effects on pulse oximetry were seen at hematocrits over 10% [178].

Dyshemoglobins

Modern pulse oximeters are designed to detect the relative amounts of reduced hemoglobin and oxyhemoglobin. They do this by sensing absorbance at two wavelengths, for example, 660 and 940 nm, which are well suited for differentiating these species. But suppose a third hemoglobin species is present in significant amounts. Because the pulse oximeter is ostensibly reporting only on reduced hemoglobin and oxyhemoglobin, a third species can only interfere with the results by causing changes in absorbance in the red and infrared regions, and, depending on the relative perturbations at the two wavelengths of interest (Fig 5), by being treated as some mixture of reduced hemoglobin and oxyhemoglobin.

In vitro oximeters, on the other hand, generally use more than two wavelengths and often can quantify other hemoglobin species directly. When dyshemoglobins such as carboxyhemoglobin and methemoglobin can be measured, it becomes meaningful to distinguish between functional saturation ($100 \times \text{oxyhemoglobin} / [\text{oxyhemoglobin} + \text{reduced hemoglobin}]$) and fractional saturation ($100 \times \text{oxyhemoglobin} / [\text{oxyhemoglobin} + \text{reduced hemoglobin} + \text{carboxyhemoglobin}]$).

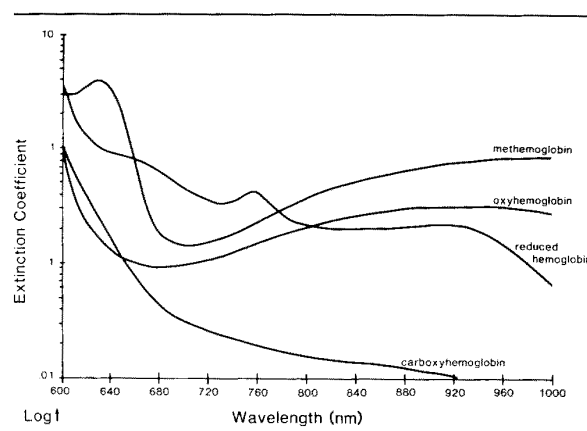


Fig 5. Light absorbance spectra for reduced hemoglobin, oxyhemoglobin, carboxyhemoglobin, and methemoglobin. (Used with permission of Ohmeda, A Division of The BOC Group, Inc, Louisville, CO.)

+ methemoglobin]). The former is commonly known as SaO_2 (or SvO_2) and refers to the oxidized fraction of that subset of hemoglobin molecules available for physiologic oxygen transport. The latter is also known as percent oxyhemoglobin, and refers to the oxidized fraction of all hemoglobin molecules; it is also called the oxyhemoglobin fraction.

Carboxyhemoglobin acts like a mixture of about 90% oxyhemoglobin and 10% reduced hemoglobin on the Nellcor N-100 and the Ohmeda Biox III pulse oximeters [179]. Since heavy smokers can attain carboxyhemoglobin levels as high as 10 to 20% and since carboxyhemoglobin has a half-life of about 8 hours [180], pulse oximetry could significantly overestimate the percent oxyhemoglobin in such patients. The potential for inaccuracy in carbon monoxide overdose is even higher. An extreme example is given in the canine experiments of Barker et al [181], in which blood with 70% carboxyhemoglobin was reported as having an SpO_2 of over 90%.

Methemoglobin causes overestimation or underestimation of percent oxyhemoglobin, depending on the relative amounts of reduced hemoglobin and oxyhemoglobin in the specimen studied. The nonlinearity of the effect depends on the difference in the magnitude of methemoglobin effect at the two wavelengths sensed by the pulse oximeter [182]. As methemoglobin increases, SpO_2 seeks the 80 to 85% range and stays there once methemoglobin is above 40%, a supraclinical level attained only experimentally in canines [183].

In at least one case report [182], dyshemoglobinemia was suspected and proved to be the cause of erroneous pulse oximeter readings. The patient, a 20-year-old

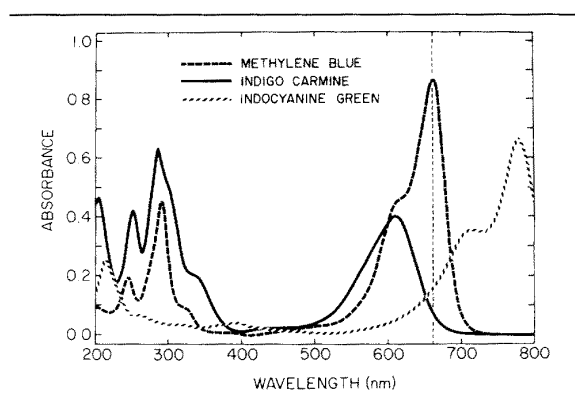


Fig 6. Absorbance spectra of methylene blue, indigo carmine, and indocyanine green dyes. The profound absorbance peak for methylene blue at 668 nm virtually coincides with the universal pulse oximetry wavelength of 660 nm (vertical dashed line). (From Scheller et al [188]. Used with permission.)

woman who was taking dapsone for pyoderma gangrenosum, was breathing an FiO_2 of 1.0 and had a Po_2 of 587 mm Hg, but the SpO_2 was only 92%. She proved to have 5% methemoglobin, probably due to the dapsone.

Fetal hemoglobin has its own special implications and is discussed under Clinical Uses: Pediatrics.

Canine hemoglobin is sufficiently similar to human hemoglobin A at 660 and 910 to 940 nm [159,184] to allow qualitative cross-species acceptance of pulse oximetry studies that probe the extremes of applicability, as above. The IL282 in vitro oximeter, used as the "gold standard" in such studies, can and should be recalibrated for canine hemoglobin [185].

Dyes

Dye interference with pulse oximetry has been actively studied before and since Kessler et al [186] described a 64-year-old patient undergoing transurethral resection of the prostate who received methylene blue and spuriously "desaturated" to 65% SpO_2 for a few minutes on 50% FiO_2 ; similar cases are described elsewhere [187]. Of the four dyes studied (methylene blue, indocyanine green, indigo carmine, and fluorescein), methylene blue drops SpO_2 the furthest and has the most interesting physiologic implications as well.

Methylene blue happens to have its major visible spectral absorbance peak at 668 nm, close to where pulse oximeters sense red absorbance (Fig 6). Hence the findings of Scheller et al [188], in which 5 ml of 1% methylene blue reliably caused transient, spurious "desaturations" in 5 patients—1 briefly to 1% SpO_2 . In-

docyanine green caused less dramatic changes (never below 93% in 5 patients). Indigo carmine caused equally minor changes, but, curiously, did so only in the toe, not in the finger. Another study in dogs [189] corroborated these results for the blue and green dyes, and found no effect of fluorescein on SpO_2 .

Methylene blue, in particular, has further and more complex effects on SpO_2 [189]. The dye reacts both to produce and to clear methemoglobin, which has its own misleading effects on SpO_2 . It also transiently increases and then depresses cardiac output, which could cause a real drop in SaO_2 via increases in pulmonary vascular resistance and thereby in pulmonary venous admixture. The relative contributions of absorbance, methemoglobinemia, and hemodynamic changes have not yet been ascertained.

An in vitro oximeter such as the IL282 cannot serve as a standard for pulse oximetry in the presence of methylene blue, since the dye also interferes with in vitro oximeters [185,190,191]. Indeed, with in vitro oximeters, methylene blue is said to falsely depress SaO_2 for up to 48 hours [192], but no figures are available on the magnitude of the effect [185,190].

In pulse oximeters, on the other hand, desaturation is reported only briefly. To explain this effect, both arteriovenous equilibration [192] and a combination of rapid tissue uptake, metabolism, and clearance [186, 192a] have been invoked. No published report has settled this question definitively, but device operation theory suggests that SpO_2 will be affected by any dye that absorbs in the range(s) of interest and that is present in sufficient amounts in the pulsatile signal, whether or not the dye has equilibrated with tissue or venous blood. This is consistent with the observed effect of dyshemoglobins, which are in arteriovenous equilibrium yet alter SpO_2 .

Bilirubin, a naturally occurring pigmented substance, does not affect pulse oximetry [102,121,193]. This is unsurprising in view of the above discussion and the generally subacute onset of hyperbilirubinemia. However, a bilirubin level over 20 mg/dl interferes with nonpulsatile methods of oximetry [194].

Ambient Light

In theory, excessive ambient light should never cause pulse oximeters to display false data, although "probe off finger" messages can be expected as the pulse amplitude grows vanishingly small compared with the baseline level of illumination. In practice, however, all manner of artifact is possible.

A Nellcor D-20 finger probe, connected to an N-100 oximeter and covered with a thin brown plastic bag,

nevertheless displayed an SpO₂ of 100% and a heart rate of 180 to 255 beats/min while irradiated by a xenon arc surgical lamp (Narco-Pilling); meanwhile, the patient was cyanotic and had in fact accidentally been extubated [195]. “Sharp-edged irregular modulations” in the surgical light, too rapid to see, allegedly interacted in pulsatile fashion with the oximeter’s several-hundred-per-second LED sensing cycle to cause this artifact.

The common fluorescent light was blamed for causing a Nellcor D-25 sensor to register a heart rate of 189 beats/min and an SpO₂ of 98% [196]. An infrared light falsely lowered both heart rate and SpO₂ on the Nellcor N-100 and made an Ohmeda Biox III display nonsense (note that oximeters sense infrared light) [197]. The spinning fan blades inside a fiberoptic cystoscope unit combined with light shining through the back vents of the unit to wreak stroboscopic havoc on a Nellcor D-25 sensor, which displayed a heart rate of 250 beats/min and an SpO₂ of 98%; subsequent experimentation showed that this setup could be made to display any desired SpO₂ down to 0% [198]. Direct sunlight has been reported to cause display of an inappropriately high SpO₂ in a pulse oximeter of unidentified make [199].

Finally, “optical cross-talk” is an unusual form of interference that occurs when two probes are placed very close to each other (a rare occurrence outside the laboratory). When the light from one probe is received by the sensor for another, measurements are erratic [200].

Thus, although a pulse oximeter generally rejects data in the presence of excessive ambient light, it cannot be trusted to do so. Remedies recommended in the literature include a surgical towel drape [198,201], an alcohol wipe pack or other foil shield [201,202], or a piece of Webril gauze as used for cushioning plaster casts [203]. This last remedy has the additional advantage of stabilizing the probe.

Skin Pigment

Despite the considerable luminescence of the LEDs used in pulse oximeters, readings cannot be obtained in a few darkly pigmented individuals, due more to failure of red than infrared transmission. The nail bed, a relatively lightly pigmented area, may be tried in these patients [204].

Slight variations in pulse oximeter error have been detected by some investigators in black adults [169] and black infants [106,205] when compared with lightly pigmented patients; other studies find no effect [165]. It remains to be explained whether these differences might be accounted for by differential changes in red and infrared transmission in darkly pigmented subjects. It is

noteworthy that individuals with dark pigmentation have generally not been used by manufacturers to generate pulse oximeter calibration curves and algorithms.

Electrocautery

This bane of most electronic monitors (e.g., electrocardiogram) intersperses false beats, artifactually decreases the SpO₂ reading, and sets off false alarms on pulse oximeters. The cause is wide-spectrum radio frequency emissions picked up directly by the photodiode in the pulse oximeter sensor (not by the cabinet electronics) [206]. Block and Detko [207] offer some suggestions on minimizing interference from electrocautery, oriented specifically toward the Nellcor N-100 but applicable in part to other devices. The relative placement of the electrocautery current return plate, electrosurgical unit, and pulse oximeter probe and cable are all important, as are alarm and various other oximeter settings. Some pulse oximeter models detect and report electromagnetic interference.

Motion Artifact

Any instrument sensitive enough to detect a change in optical path length due to cutaneous vascular pulsations will also be sensitive to motion artifact, as during shivering [29] or transport to the recovery room [54]. A trial of the Ohmeda Biox 3700 in Life Flight helicopter runs, on the other hand, uncovered no flight-related motion artifact [208].

Repetitive and persistent motion artifact of any kind will tend SpO₂ toward a specific value that depends on the device’s algorithm. Such motion tends to introduce signals of roughly similar amplitudes into the red and infrared channels, usually yielding a red-to-infrared absorbance ratio near 1. Because pulse oximeters use empirical “calibration curves” relating absorbance ratios to SpO₂ values, a ratio of 1 is associated with a specific SpO₂ value. For example, the Ohmeda Biox 3700 displays an SpO₂ of 85% under these circumstances [29], and the value is probably similar for other pulse oximeters.

Onychoparaphernalia

A Nellcor N-100 pulse oximeter (with unspecified probe) was reported to function well through a layer of nail polish of unspecified type [209]. This prompted Rubin [210] to assess the Ohmeda Biox 3700’s performance with 31 nail polish colors throughout the visible spectrum, including black. Of these, only “Blue Flame” (Avon Inc, New York, NY) affected SpO₂ (decreasing it from 97% to 87%); other shades of blue or violet had

no effect. The offending shade had an absorption maximum at 688 nm; like methylene blue, this interferes with the universal 660-nm transmission used by pulse oximeters.

On the other hand, a revealing study by Coté et al [211] found significant effects of black, blue, green, and brown red nail polish on pulse oximetry. They also showed that, for any given nail polish color, the degree of SpO₂ artifact correlates best with the *difference* between its absorption at 660 nm and its absorption at 940 nm. This difference has a greater effect on SpO₂ than does the absolute degree of absorbance at either wavelength. Hence, blue polish, with strong absorbance at 660 nm but weak absorbance at 940 nm, decreased mean SpO₂ by 5.92%, whereas black polish, with strong absorbance at both 660 nm and 940 nm, decreased mean SpO₂ by only 3.08%. The apparent disagreement with the results of Rubin may be due to the multiple layers of polish used by Coté et al. This study sheds light on a mechanism for nonpulsatile forms of interference with pulse oximetry and may help to explain past and future findings about skin pigment, dyes, ambient light, and other possible causes of SpO₂ artifact.

Synthetic nails have also been noted to interfere with pulse oximetry [36].

Complications of Monitoring

A Nellcor I-25 probe was reported to blister the underlying skin of a 3-month-old patient after 6 hours of continuous use. The probe temperature, an abnormally high 70°C, was later found to have been due to a faulty oximeter device, not a faulty probe [212]. An Ohmeda Biox III ear probe used for 48 hours on a 4-month-old caused mild skin erosion. The fiberoptic finger probe for the Mochida MET 147 (Minolta) "tanned" (hyperpigmented) the foot of an 8-month-old infant after 5 days of continuous use [213].

In adults, 1 case of ischemic skin necrosis and 3 associated cases of blistered skin have been reported. Ischemic skin necrosis was produced by a Nellcor DS100-A non-disposable probe that was used continuously for several days in a critically ill 74-year-old woman whose ipsilateral radial artery had been cannulated [214]. Intermittent shorting in a single defective Datascope Accusat probe was reported to have blistered the index fingers of 3 patients, resulting in voluntary recall of the devices and redesign of the probes [215].

REUSING PROBES

Pulse oximeter probes are costly, and a whole subterranean lore devoted to the preservation of disposable probes has appeared in the correspondence departments

of anesthesia and monitoring journals. The most imaginative is due to Foltz [216]. He lines a malleable aluminum finger splint with a flexible probe trimmed of its marginal adhesive tape. Others use a finger cot, also after trimming the tape [217].

Addressing another aspect of probe preservation, one author has advocated attaching Velcro to the bulkier part of a nondisposable probe, and its mate to the oximeter case, preventing it from "leaping to the floor" while not in use [218].

With repeated reuse, adhesive may get onto the LED and obscure the sensor's light source; this was recently blamed for falsely lowering SpO₂ and thereby exposing a neonate to risk of iatrogenic retrolental fibroplasia (the old probe gave an SpO₂ of 80%; a new one then showed 100%) [219]. The probe "may just have exceeded its lifespan" [220], a fate that awaits all sensory apparatus, however inventive their users.

SUMMARY

Pulse oximetry, a noninvasive method for evaluating arterial oxygen saturation, has enjoyed widespread use since its invention in the early 1970s and its clinical introduction in the early 1980s. Its intraoperative, postoperative, and critical-care applications are well established, and many observers believe that its use will soon become "standard of care" in operating rooms in the United States. Proposed applications that use the pulse oximeter primarily as a plethysmograph must await further investigation of its performance under low-flow conditions.

In general, pulse oximeters are sufficiently accurate and precise to monitor trends in oxygenation. However, they are not accurate enough for all applications, owing either to inherent machine limitations or to the shape of the oxyhemoglobin dissociation curve (as in the assessment of early desaturation at high FiO₂ or of neonatal hyperoxemia) or both. Many proposed uses, such as the prescription of ward oxygen therapy, occupy a middle ground where clinical judgment regarding the use and utility of pulse oximeter data is paramount. Accuracy may be compromised in the presence of very low SaO₂, dyshemoglobins, and vital dyes; other conditions, such as low flow, ambient light, and motion, may render data collection impossible. Despite these limitations, the pulse oximeter is generally acknowledged to be one of the most significant advances in the history of clinical monitoring.

ADDENDUM

Final approval is pending on regulations mandating the use of "a pulse oximeter or superior equipment" during all general

and regional anesthesia administered in the State of New York—an electronic monitoring requirement that is probably unprecedented in American law (Lees DE. New York State regulations to be implemented: work hours, resident supervision, anesthesia monitors mandated. *Anesthesia Patient Safety Foundation Newsletter* 1988;3(3):17–19).

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