

REVIEW ARTICLE

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Recent Developments in Pulse Oximetry

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CONTENTS

Introduction
Methodological Developments
Uses of Pulse oximetry
The Detection of Hypoxia
Monitoring Circulation
Role in Preventing Retinopathy of Prematurity
Investigative Uses
Limitations of Pulse Oximetry
Incidence of Failure
Low Signal-to-noise Ratio
Probe Position
Vasoconstrictors
Low-perfusion Limits
Motion Artifact
Abnormal Pulses
Ventilator-induced Pulse Interference
Response Times
Ambient Light
Electrocautery
Interference of Magnetic Resonance Imaging
Alternative Sites
Reflectance Operation
Adults
Fetal
Skin Pigments, Dyes, Nail Polish
Carboxyhemoglobin and Methemoglobin
Potential Dangers
False Alarms and False Nonalarms
Accuracy
Effect of Anemia on Errors
In Vitro Methods of Testing the Accuracy of Pulse Oximeters
Evaluations of Pulse Oximeter Performance and Features
Does Pulse Oximetry Increase Patient Safety?
Effect of Pulse Oximetry on Other Monitoring Methods
Regulation, Insurance, Standards, and Cost
Possible Effects of Oximetry on Anesthesiologists

Pulse oximeters now occupy most critical care arenas and virtually every operating room in the United States. They are manufactured by more than 35 firms, with 1989 annual world wide sales estimated at 65,000 units valued at \$200 million.

In January 1989, two comprehensive reviews of pulse oximetry were published. One gave relative emphasis to theory of operation and other technical aspects,¹ while the other focused primarily on clinical issues.² Since the reviews of 1989 were completed, more than 500 additional publications have described methods, uses, problems, progress, and effects of pulse oximetry—135 of them in the 6-month period prior to the end date of this review (October 1, 1991). The past 3 yr have seen a variety of other reviews concerning some recent developments³⁻¹² as well as the history of pulse oximetry.^{13,14}

The purpose of this article is to summarize the literature on pulse oximetry that has appeared since the major reviews of early 1989. Expressions such as "before 1988" and "since 1988," unless otherwise indicated, refer herein to the mid-1988 cutoff date for the references appearing in those reviews.

Methodological Developments

There is relatively little to report as to methodological advance in pulse oximetry since 1988. A potential exception is surface reflectance ("surface") oximetry, which has received significant recent experimental attention but does not appear ready for widespread clinical use. In 1988, a review of pulse oximetry² could dismiss the topic of reflectance oximetry by observing that it was "one desirable possibility for the future of pulse oximetry . . . in which measurements of reflected light would allow monitoring at nontransilluminable sites, such as the fetal presenting part during labor." With the possible exception of its use in labor, reflectance oximetry is still largely investigational. Some models now couple the oximeter to the ECG (see "Limitations of Pulse Oximetry: Motion

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Artifact"). Probe design has been evolving toward reduced size and greater comfort. Two recent reviews discussed the technology of pulse oximetry,^{15,16} although it must be noted that by and large this technology has not changed since 1988.

Equipment is now available to permit central nursing stations to monitor several patients remotely (Novamatrix Inc). Pan and James¹⁷ reported trials of a telemetric pulse oximetry network for use in monitoring of patients on a ward (after caesarean section). Their study was concerned primarily with false alarms (see "Limitations of Pulse Oximetry: False Alarms and False Nonalarms").

Uses of Pulse Oximetry

Clinical uses of pulse oximetry may be divided into the oximetric and the plethysmographic.² The oximetric applications are largely concerned with detecting and quantitating hypoxemia in various settings, but considerable attention and controversy have surrounded oximetry's use in avoiding neonatal hyperoxia as well.

In 1988, the detection of hypoxemia in perioperative and critical-care settings was an established use for the pulse oximeter; its utility as the sole monitor for neonatal hyperoxia was controversial at best; and a large number of plethysmographic uses had been suggested anecdotally,

TABLE 1. Uses of Pulse Oximeters: Topical Analysis by Frequency of Publication from May 1988 to October 1991

Subject	Number of Papers
Endoscopy	26
Postoperative recovery	22
Neonatal intensive care unit	21
Oral surgery and dentistry	14
Airway management	13
Sleep studies	13
Hypotension, poor perfusion	12
Premedication	11
Pediatric anesthesia	10
Transport	10
Emergency	9
Chronic obstructive pulmonary disease, lung disease	8
Adequacy of circulatory tests	8
Anesthesia, adult	8

Topics reported in three to seven papers include croup; infection; fetal monitoring; magnetic resonance imaging; exercise; epidural morphine; altitude studies; mechanical ventilation; caesarean section; intensive care units; hypothermia; embolism; one-lung anesthesia; positioning problems; heart surgery; and anemia. Other uses discussed in one or two papers include home monitoring of sudden infant death syndrome patients; weaning from a ventilator; hypoxemia during labor; seizures; asthma; induction of anesthesia with jet ventilation; obesity and apnea; hemodialysis; pneumothorax; pulmonary edema; aspiration; malignant hyperthermia; efficacy of cardiopulmonary resuscitation; effect of sickle cell disease; application in ear, nose, and throat practice; and home O₂ therapy control.

TABLE 2. Performance and Limitations of Pulse Oximetry: Topical Analysis by Frequency of Publication from May 1988 to October 1991

Subject	Number of Papers
Effects of methemoglobin, carboxyhemoglobin	19
Tests of accuracy	18
Sites for probes	13
Failure with hypoperfusion	12
Standards and legal issues	7
Motion artifact	6
Skin pigments	6
Uses in animals	6
Models for <i>in vitro</i> testing	5
Palmar circulation (Allen's test)	5

Other publications described oximetry in burn patients; comparisons with transcutaneous P_{O₂}; use in tissue revascularization and transplantation; effect on use of arterial blood gas analysis; reports of finger burns by probes; blood pressure measurement; and cost-benefit ratio estimations.

relatively few of which had been submitted to more rigorous examination.^{1,2} All three of these generalizations still hold, despite worthwhile developments in each area.

The time-honored, if not ideally rigorous, technique of "citation analysis" (used memorably in Keats's 1983 Rovenstine Lecture,¹⁸ among other publications) helps to illustrate where current clinical interest in pulse oximetry is directed. It is clear from table 1 that pulse oximetry, having entrenched itself in the bank of operating-room monitors, is now under active scrutiny for the recovery room and for outpatient settings such as the endoscopy suite, the dentist's office, and the sleep laboratory. Table 2 lists recent active topics of investigation into performance or limitations of pulse oximetry, independent of clinical setting.

THE DETECTION OF HYPOXEMIA

The high prevalence of clinically unsuspected hypoxemia is perhaps the most famous disclosure that pulse oximetry has made. The incidence of hypoxemia was studied intraoperatively in a single-blind study of 296 adult anesthetics by Moller *et al.*¹⁹ In 53%, mild hypoxemia (86–90%) was seen. Severe hypoxemia, with hemoglobin (Hb) oxygen saturation measured by pulse oximetry (SpO₂) as < 81%, was recorded in 20% of the patients; 70% of these severe episodes were not detected by the anesthetist. McKay and Noble²⁰ found that 6% of a series of nearly 5,000 anesthetics involved critical incidents, of which 29 involved SpO₂ readings < 75%. Coté *et al.*,²¹ in a single-blind study of 402 pediatric cases, separately examined the effect of withholding oximeter and/or capnograph data from the anesthesia team. They identified 59 major

desaturation events ($\text{SpO}_2 < 85\%$ for > 30 s) in 43 patients and 130 minor desaturations ($< 95\%$ for > 60 s). Of the major events, 41 ($\approx 70\%$) were first diagnosed by oximetry, 13 by the anesthesiologist and 5 by the capnograph. Blinding the oximeter data increased the number of patients experiencing major desaturation events from 12 to 31 ($P = 0.003$). Infants ≤ 6 months of age had the highest incidence of major desaturation events. Blinding the capnograph data altered neither the frequency of desaturation events nor the incidence of major capnograph events but did increase the number of patients with minor capnograph events ($P = 0.0026$). The authors concluded: "1) The pulse oximeter is far superior to either the capnograph or clinical judgment in providing the earliest warning of desaturation events. 2) Capnography can provide an early warning to potentially life-threatening problems, but such problems often result in desaturation" ²¹

In a less-controlled study of 91 pediatric anesthesia cases, Schulz *et al.* ²² found hypoxic episodes ($\text{SpO}_2 < 95\%$) in 52% and major desaturations ($\text{SpO}_2 < 85\%$) in 54% of newborn infants. They reported that "desaturation was more likely to occur in intubated children than in those with a mask ($p < 0.05$)" (reflecting problems with the processes of intubation and extubation).

These last 3 yr have seen the investigative focus turn from the operating room and intensive care unit, where unanticipated hypoxemia is relatively rare, to the post-anesthesia recovery period, clearly demonstrating the high incidence of hypoxemia due to the effects of anesthetic, sedative, relaxant, and especially opioid drugs. ²³⁻³⁶ In Copenhagen, a single-blind study ³² of 202 adults in the recovery room following elective operations, found decreases to $\text{SpO}_2 < 91\%$ in 55% of all patients at some time before discharge, despite administration of nasal oxygen in more than half of these hypoxic episodes, and to $\text{SpO}_2 < 80\%$ in 13%. Patients who had undergone regional anesthesia had a lower risk of hypoxemia. A randomized study by Lampe *et al.* ³¹ of 141 patients for carotid endarterectomy or hip replacement noted postoperative $\text{SpO}_2 < 90\%$ in 63% and $< 86\%$ in 21%; they monitored that portion of the postoperative period included in "a 24-hour period beginning just after induction of anesthesia." Use of oxygen during the first postoperative night reduced the incidence of hypoxemia from 29% to 0%.

During recovery from anesthesia studied in Sydney, Australia ³⁴ with routine use of nasal oxygen as judged appropriate by nurses, 80% of patients' saturations decreased to $< 90\%$ SpO_2 and 26% decreased to $< 80\%$ SpO_2 during the first 15 min of recovery. Sixty-four percent decreased to $< 90\%$ even while the patients received nasal oxygen. On the other hand, in Boston, Morris *et al.* ²³ showed that only 14% of inpatients developed at least transient hypoxemia during recovery, and only 1% of outpatients became hypoxic in the recovery room. The

amounts of premedication and injected sedatives and opioids were not significantly different between inpatients and outpatients.

Canet *et al.* ³⁶ not only demonstrated postoperative hypoxemia in 44% of more than 200 patients and showed that 35% oxygen prevented hypoxemia, but also reviewed other evidence that supported the routine administration of oxygen and the concomitant use of pulse oximetry.

The incidence of hypoxemia has been investigated in various outpatient or "office" procedures accompanied by sedation without professional anesthesia assistance. The greatest attention has centered on bronchoscopy and endoscopy. Schnapf ³⁷ demonstrated $> 5\%$ desaturation in 80% of 36 children aged 6–142 months during fiberoptic bronchoscopy in a pediatric special care unit or pulmonary laboratory, with higher incidence in the youngest patients.

Pulse oximetry has shown potentially dangerous desaturation to be present in 45% of patients undergoing endoscopy under sedation. ³⁸ A prospective study by Moore *et al.* ³⁹ in surgical intensive care disclosed hypoxic episodes in 21% of patients in the surgical intensive care unit, mostly during mechanical ventilation. In a recent review with 63 references, Bell ⁴⁰ suggests that during gastrointestinal endoscopy, hypoxic problems are common, with 60% of deaths during endoscopy attributed to cardiopulmonary complications. Al-Hadeedi and Leaper ⁴¹ observe that the 1:5,000 mortality during upper gastrointestinal endoscopy—itsself "several fold" lower than that for endoscopic retrograde cholangiopancreatography—still "does not compare favorably with the much lower perioperative mortality directly attributed to anesthesia." In 132 patients undergoing endoscopic retrograde cholangiopancreatography under sedation, they found that SpO_2 decreased from $95.7 \pm 2.4\%$ (mean $\pm$ standard deviation) to $88.9 \pm 6.4\%$. The largest decreases occurred after positioning of the endoscope (rather than after administration of the sedative or completion of the procedure). In 57 patients between the ages of 6 weeks and 36 months undergoing 60 flexible upper intestinal endoscopies with parenteral sedation only, 7 patients showed oxygen desaturation to $< 90\%$ after sedation but before insertion of the endoscope without overt clinical evidence of complications. ⁴² The British Society of Gastroenterology recommended and approved guidelines for use of pulse oximetry as a standard during all procedures and also suggested that oxygen be administered. ⁴³

Sedation and anesthesia are commonly used without professional anesthesia assistance in oral surgery and dentistry. In view of the potentially high incidence of hypoxia, pulse oximetry has been strongly recommended, ⁴⁴ although Wilson ⁴⁵ suggested that 87–90% of desaturation episodes in his study of 22 sedated children were due to motion artifacts.

Marjot and Valentine⁴⁶ found an 80% incidence of hypoxemia ("persistent desaturation to S_aO_2 less than 90%") following premedication with lorazepam, morphine, and droperidol for cardiac surgery, accompanied in 33% by ECG changes, previously unsuspected and easily corrected by oxygen administration. Hypoxemia ($S_aO_2 < 90\%$) was documented in 10 of 15 women in labor, 7 of whom had received opioids.⁴⁷

Oximetry has also been used to demonstrate that periodic breathing in premature and newborn infants is unrelated to the occurrence of apneic spells.⁴⁸ Oximetry may reduce the need for intensive care in low-risk groups. In 30 patients during emergency field rescue operations, SpO_2 was compared with arterial blood oxyhemoglobin (O_2Hb) and found to correlate with $r = 0.898$ and bias ($SpO_2 - O_2Hb$) of -0.3 ± 2.4 .⁴⁹

Airway management problems aided by oximetry include evaluation of the performance of experimental rescue devices such as the "glossopalatal tube,"⁵⁰ detection of obstructed⁵¹ or misplaced⁵²⁻⁵⁴ endotracheal tubes, assessment of readiness for removal of tracheostomy,⁵⁵ foreign body removal,⁵⁶ and effectiveness of use of helium.⁵⁷ Oximetry is especially useful in managing one-lung anesthesia.⁵⁸

Use of oximetry and end-tidal P_{CO_2} instead of arterial blood gas in assessing a patient's ability to be separated from mechanical ventilation was studied in 60 patients by Withington *et al.*⁵⁹ After assessing noninvasive method reliability in 20 subjects, they demonstrated the success of the method in the next 40. Pulse oximetry has been used in optimizing continuous positive airway pressure and positive end-expiratory pressure⁶⁰ and, for quantitative purposes, pulse oximetry has been a documented help in a variety of procedures, such as determination of the needed oxygen flow rate or concentration in ventilator-dependent patients.⁶¹

When epidural morphine came into general use for pain relief, there was concern about possible unobserved apnea on wards. Choi *et al.*⁶² demonstrated in 20 post-caesarean section women that half of the patients experience desaturation to $< 85\%$ over the 15-h monitoring period, whether they were given morphine parenterally or epidurally, but the desaturation occurred within 3 h with parenteral morphine and at 13.7 ± 5.9 h with epidural morphine.

In a study in general hospital (non-critical-care) units, Bowton *et al.*⁶³ found that 75% of patients monitored for 36 h had at least one episode of desaturation to $< 90\%$ and 58% had at least one episode of desaturation to $< 85\%$. Few of these episodes were documented, and even the availability of monitoring had little effect on care.

The use of oximetry in emergency care situations was reviewed.⁶⁴ Patients transported by ambulance, helicopter, and aircraft are increasingly monitored by pulse ox-

imetry because the vibration makes most other methods of ventilatory and circulatory monitoring ineffective.⁶⁵ Short *et al.* compared seven oximeters during helicopter transport, and reported three to be subject to vibration interference or otherwise unacceptable.⁶⁶

Pulse oximetry is used (in connection with periodic arterial blood gas determination) to help adjust the flow rates and periods of use of home oxygen therapy of patients with chronic obstructive pulmonary disease, *e.g.* in response to increased needs with exercise and rapid-eye-movement sleep. A review of this field by Tiep (110 references) suggests that the involvement of physician and oxygen supplier may have increased in the effort to tailor oxygen supply to demand and to reduce possible excessive oxygenation (and respiratory depression).⁶⁷

In summary, pulse oximetry is now well-established—both inside and outside the operating room and intensive care unit—as a useful and sensitive detector of hypoxemia. Its acceptance as a supplement to clinical detection for hypoxia is clearly apparent. Equally apparent, however, is the role of motion artifact in limiting its specificity, especially in certain populations (see "Limitations of Pulse Oximetry: Motion Artifact"). Furthermore, outcome studies are generally lacking for the above-mentioned applications, leaving open the question of whether pulse oximetry reduces morbidity and mortality (see "Does Pulse Oximetry Increase Patient Safety?").

MONITORING CIRCULATION

The pulse oximeter's plethysmographic capability has been proposed as a monitor of circulatory adequacy. By 1988, a number of anecdotal applications of this type had been reported.² This list is now longer (below), but an earlier caution² still holds true: the pulsatile perfusion required to generate a pulse signal on a given pulse oximeter is not *a priori* either necessary or sufficient to guarantee adequacy of circulation for a given application. Machine characteristics, intersubject variability, and intrasubject variability all are involved. Only controlled studies can resolve these issues; by and large, these have not been done for plethysmographic applications of the pulse oximeter.

The best-studied use of this type is Allen's test. In 1988 there was no consensus among those who had investigated pulse oximeter plethysmography as a measure of palmar collateral circulation.² Several others have now tried and recommended it without controlled testing.^{68,69} Levinsohn *et al.*,⁷⁰ taking digital cuff sphygmomanometry as the "gold standard," showed laser Doppler to be equally accurate and easier to use, and found the classic ("subjective") Allen's test to be sensitive but not very specific ("a good screening test"). But they found pulse oximetry to indicate false adequacy of collateral circulation in two of three hands with abnormal digital systolic pressure.

In contrast, Pillow and Herrick⁷¹ found that laser Doppler agreed with pulse oximetry in 109 of 109 patients with respect to both ulnar collateral flow (100 sufficient, 9 insufficient) and radial collateral flow (108 sufficient, 1 insufficient). They were unable to account for the contrary results of Glavin and Jones,⁷² who rejected pulse oximetry for this test 2 yr earlier, in 1989, except to speculate that the thumb may be a more sensitive site than the index finger for monitoring ulnar collateral flow. Despite the further study, this issue appears no more settled than it was three yr ago.

Systolic blood pressure may be accurately determined by reappearance of the pulsatile waveform during cuff deflation in instruments that display the waveform (tested with Ohmeda 3700)^{73,74} or, perhaps more accurately, by waveform disappearance during slow cuff inflation.⁷⁵⁻⁷⁷ Comparison of pulse amplitudes on the finger and the toe demonstrated toe vasodilation due to sympathetic blockade with spinal anesthesia.⁷⁸ The waveform response to a Valsalva maneuver can detect patients with autonomic dysfunction or blockade.⁷⁹

Other new reported uses include determining ductus arteriosus patency,⁸⁰ assessing the level of ischemia in peripheral vascular disease (in which it is claimed to be more sensitive than either transcutaneous P_{O_2} [tcP_{O_2}] or Doppler flowmetry⁸¹), assuring patency of major arterial grafts,⁸² testing viability of the bowel,^{83,84} indicating artery compression in shoulder arthroscopy⁸⁵ or fracture manipulation,⁸⁶ determining limb vascularity,⁸⁷ assessing circulatory adequacy of the arm when an unconscious patient is placed in the lateral or prone position or with arms elevated or hyperabducted for surgery,⁸⁸ and monitoring circulation of reimplanted digits or grafts.⁸⁹ In general, these are anecdotal uses, not verified by controlled studies.

ROLE IN PREVENTING RETINOPATHY OF PREMATURITY

In premature infants, the administration of supplemental oxygen is associated with retinopathy of prematurity (ROP).^{90,91} The duration, concentration, and pattern of oxygen toxicity all are implicated—as are gestational age, hypercapnia (possibly *via* a vasodilatory increase in oxygen delivery to the retinal vasculature⁹²), the state of the ductus arteriosus (open⁹³ or closed⁹⁴), light,⁹⁵ and literally dozens of other factors, which may or may not be confounding.⁹¹ Indeed, premature infants can develop ROP despite persistent hypoxemia.⁹⁶

Empirical recommendations for arterial oxygen saturation (Sa_{O_2}) in infants at risk for ROP generally fall into the 90%–95% range.^{2,97} The pulse oximeter has obvious application here, but its main use is in preventing hypoxemia, not hyperoxia. It is generally agreed that pulse oximetry is not adequate for evaluating hyperoxia in this

setting—not only because its inherent inaccuracy of 2–3% looms so large in the region of the Hb-oxygen dissociation curve above Sa_{O_2} 90%, but also, and more fundamentally, because there is no consensus on an acceptable safe upper limit for Sa_{O_2} .

A recent “consensus report” on neonatal pulse oximetry,⁹⁸ definitive enough in its other recommendations, was able to recommend “further research” only after “long and complex” discussion of a safe upper limit. The current uncertainty about this issue is illustrated by the various definitions of hyperoxia used in recent investigations into neonatal pulse oximetry; they include $P_{O_2} > 80$ mmHg,⁹⁹ $P_{O_2} > 90$ mmHg,¹⁰⁰ and $P_{O_2} > 100$ mmHg.⁹⁸

Hay *et al.*¹⁰¹ noted that when an Ohmeda 3800 displayed $Sp_{O_2} = 92 \pm 3\%$ in neonates, the Pa_{O_2} was between 40 and 100 mmHg 100% of the time—but they added that, while keeping $Sp_{O_2} = 92 \pm 3\%$ “seems prudent and safe, . . . this conclusion is arbitrary and not tested.”

Bucher *et al.*¹⁰⁰ recently concluded that the Nellcor N-100 and the Ohmeda Biox 3700 are highly (95%) sensitive for hyperoxic episodes ($P_{O_2} > 90$ mmHg) but have only mediocre specificity (38% and 52%, respectively); this 95% sensitivity was achieved by choosing an Sp_{O_2} upper limit of 96% for the Nellcor N-100 but of only 89% for the Ohmeda Biox 3700. Findings like these suggest that caution is required in interpreting blanket recommendations on safe upper limits, such as Hay’s “consensus” of Sp_{O_2} 94–95%,⁹⁸ that do not make reference to a specific pulse oximeter brand and model.

On the other hand, Reynolds and Yu,⁹⁹ also using the Ohmeda Biox 3700 and studying 175 readings in 12 neonates with pulmonary compromise, recommended an Sp_{O_2} upper limit of 90% to avoid $P_{O_2} > 80$ mmHg. If nothing else, the incongruity between this result and that of Bucher *et al.*¹⁰⁰ underscores that the prevention of ROP may involve more than just control of Sa_{O_2} or Pa_{O_2} . Indeed, the recent slowdown in published investigations into a safe upper limit of Sa_{O_2} for neonates—in contrast to a veritable spate of such studies *circa* 1987—may signal a conceptual retrenching as the multifactorial nature of ROP is acknowledged in practice.

What is certain is that pulse oximetry cannot currently be recommended as the sole monitor of oxygenation for the neonate at risk for ROP. The prevention of ROP is probably best accomplished by either intermittent arterial blood gas analysis or continuous tcP_{O_2} monitoring, with pulse oximetry as a supplementary modality.

As Sa_{O_2} decreases, because of the steepening dissociation curve, oximetry becomes progressively more dependable than tcP_{O_2} measurement in assessing, controlling, and maintaining some defined level of oxygenation. In an infant with mostly fetal Hb, a 5-mmHg change in P_{O_2} at 70 mmHg ($\approx 97\% Sa_{O_2}$) is equivalent to only about

0.6% change of SaO_2 , but the same 5 mmHg change at 40 mmHg ($\approx 87\% \text{SaO}_2$) accompanies a 5% change in SaO_2 . Fetal Hb is not distinguishable from adult Hb by pulse oximeters.¹⁰² For this reason, at $\text{SaO}_2 < 90\%$, some investigators^{99,103,104} believe oximetry to be more reliable than transcutaneous monitoring, but not even they seem to believe that it can supplant other forms of monitoring. The relative merits of tcPO_2 monitoring and pulse oximetry have been reviewed.¹⁰⁵⁻¹⁰⁷

INVESTIGATIVE USES

Cardiopulmonary resting and exercise tests,^{108,109} hypoxic ventilatory response studies,¹¹⁰ and sleep abnormality studies¹¹¹⁻¹¹⁶ are now more easily and accurately done with oximetry. The rate of decrease of SpO_2 during apnea after 5 min of preoxygenation was shown by Jense *et al.*¹¹⁷ to be twice as fast in morbidly obese patients, with SpO_2 decreasing to 90% in 163 ± 15 s in the obese subjects.

Using oximetry, Brodsky *et al.* reassessed diffusion hypoxia after discontinuation of nitrous oxide.¹¹⁸ In ASA physical status I and II patients breathing unspecified concentrations of isoflurane in 3:2 nitrous oxide/oxygen (at $5 \text{ l} \cdot \text{min}^{-1}$), a transient SpO_2 decrease of 4% was seen 3 min after discontinuation of nitrous oxide, whereas no corresponding drop was seen in patients breathing isoflurane in 100% oxygen.

Oximetry has been used to test exposure to and acclimatization at high altitude¹¹⁹⁻¹²³ and to study breath-holding in the diving women of Korea and Japan.¹²⁴ Pulse oximeters have been successfully used in laboratory animals such as the dog,¹²⁵ sheep,^{126,127} pig,¹²⁸ horse,¹²⁹ rabbit,¹³⁰ and rat.¹³¹ The sites used have been the ear, cheek, tongue, and tail.

Limitations of Pulse Oximetry

INCIDENCE OF FAILURE

In a prospective intraoperative study at the University of Washington, Freund *et al.* reported a 1.12% failure rate in 11,046 anesthetics.¹³² Failure was defined conservatively as "the inability to obtain any pulse oximetry reading for a cumulative period of 30 minutes or greater after all mechanical problems . . . had been eliminated and all possible sites had been tried . . ." (*e.g.*, other digits, ear, nose). Curiously, the failure rates differed by hospital: they were 0.78% at the University Hospital, 0.56% at the Harborview Hospital, and 0.56% at the Children's Hospital, but 4.24% at the Veterans' hospital. When a failure occurred, it persisted for 32% of total anesthesia time.

In a retrospective study of 1,403 patients in a postanesthesia care unit at the University of Washington Hospital, Gillies *et al.*¹³³ found a comparable failure rate of 1.1%

(two or more 15-min periods when no values were noted). About 90% of failures were at the beginning of the stay in the postanesthesia care unit.

LOW SIGNAL-TO-NOISE RATIO

Probably a majority of the factors that interfere with pulse oximetry can be explained as either too little signal (low-perfusion state, improper probe placement) or too much "noise" (motion, ambient light, electrocautery, venous pressure waves). When arterial pulsation decreases below some fraction of total transmitted light (about 0.2% for the Ohmeda units), the signal is judged unacceptable by the computer program, which may display a "low quality signal" message, display a blank, or set the display to zero. This lower limit for default varies between manufacturers. Instruments that continue to display SpO_2 at lower pulse pressures may be more prone to providing erroneous data at low signal levels.^{7,134-136} If artificial "pulsations" are present (due to some interfering process such as mechanical movement or ventilation), the processor may either default to zero or display an incorrect SpO_2 with an erroneous heart rate. The inappropriate heart rate can often alert the user that a problem exists.

PROBE POSITION

Kelleher and Ruff documented that finger probes, when withdrawn partially beyond the fingertip, exhibit what they called a "penumbra effect,"¹³⁷ often causing a false low reading before failure occurs. (The authors speculated that this might also cause a false high reading if SaO_2 is $< 85\%$.) The mechanism probably involves light shunt either edgewise through superficial finger tissue or through air, lowering the signal-to-noise ratio. The erroneous values are more common with warm than with cold fingers.

VASOCONSTRICTORS

Cold vasoconstriction combined with a low pulse pressure (*e.g.*, after cardiac bypass) or an increase in venous pressure often precludes detection of SpO_2 from fingers or at least delays detection of hypoxemia.¹³⁸ In ten pediatric patients deliberately surface-cooled to 25°C , the arterial oxygen saturation was overestimated by the pulse oximeter between 30° and 36°C and was underestimated below 30°C when the initial saturation was low.¹³⁹ Vasoconstriction in shock or cold may essentially stop flow through fingers without eliminating pulsatility in arterioles. This may result in gradual desaturation of the arterial blood remaining in the fingertips, either by diffusion through arterial walls or by movement in and out of sinusoids or capillaries. An example was noted in a cachectic

70-yr-old woman during hemorrhagic shock, initially treated with ephedrine.⁷⁷ Sp_{O_2} decreased from 98% to 45% while the heart rate detected by the oximeter continued to be correct at radial arterial $P_{O_2} = 550$ mmHg. Local finger desaturation has been confirmed during experimental hypotension and vasoconstriction.⁷⁷

Finger vasoconstriction can prevent detection of the changes clearly shown with an ear probe.¹⁴⁰ Anecdotal reports suggest that a finger block with 0.25 ml 1% lidocaine on each side at the base of the finger may restore circulation and oximeter detection in the face of vasoconstriction induced by cold or hypotension. Grayson and Bourke¹⁴¹ were able to restore oximetry signals for 1.5–2 h in five of five patients in whom finger signal had disappeared using 2 ml 2% lidocaine. Topical nitroglycerin paste on the finger has been used to reverse vasoconstriction, but with variable results. Spinal anesthesia strongly increased the pulse amplitude detected in the toe.^{78,140}

LOW-PERFUSION LIMITS

The low-perfusion limits of pulse oximetry were relatively unexplored in 1988. Since then, the low-pressure and -flow limits at which oximeters fail have been extensively investigated.^{7,77,134–136,142} In volunteers, Severinghaus and Spellman⁷⁷ induced hand hypotension or vasoconstriction by several methods such as arm elevation in the lateral decubitus position (a nitroprusside infusion was required in several instances to induce signal detection failure), application of a carpenter's C-clamp slowly over the antecubital brachial artery, inflation of a brachial cuff, and intraarterial injection of norepinephrine. The three pulse oximeters tested were the Nellcor N200, the Ohmeda 3740, and the Criticare 504US. The mean systolic pressure at the threshold of function (mean of failure with decreasing pressure and recovery with increasing pressure) was 29.7 ± 12.8 mmHg with elevation, a test in which pulse pressure remains normal. No difference was found among the three oximeters. However, with partial clamping of the brachial artery, the Nellcor threshold was 47.1 ± 13.5 mmHg, significantly higher than those of the other two (38.7 ± 14.5 with Criticare, 36.0 ± 3.4 with Ohmeda). Norepinephrine injection further increased the thresholds. A potentially significant observation was that when pressure was just above threshold, detection of an induced hypoxic transient was delayed, in one case by 6 min. Also noted was a decrease of Sp_{O_2} despite normoxia in the awake volunteers, reaching on average $88.8 \pm 11.5\%$ Sp_{O_2} just before failure. The authors suggested that pulsation may continue to be detected in the absence of flow, such that oxygen may diffuse from either arteriole or capillary to finger tissue, causing an actual decrease in the pulsating blood oxygen saturation. These failure thresholds were considerably lower than those often seen clinically, suggesting that

clinical failure is more often due to finger vasoconstriction than to hypotension.

The brachial arterial C-clamp method was also used by Falconer and Robinson¹³⁴ to compare failure and error rates in 13 pulse oximeters in volunteers at reduced pulse pressures during steady mild hypoxemia ($80 < Sa_{O_2} < 88\%$). With pulse pressures between 11 and 20 mmHg, only the Physio-Control 1600 failed significantly more than the other instruments. At < 11 mmHg pulse pressure, the authors were able to rank instruments, reporting that "the Datex Satlite® was found to be more accurate than the average pulse oximeter, and, conversely, the Nellcor N100 and N200 without ECG link were found to be less accurate." They reported that the Nellcor N100, Nellcor N200 without ECG link, and Physio-Control 1600 defaulted to zero significantly more often than expected. The addition of an ECG link to the Nellcor N200 and Criticare 504US significantly increased the frequency of correct function at pulse pressures < 11 mmHg.

Clayton *et al.*¹³⁶ evaluated the performance of 20 pulse oximeters with finger probes by comparison of their readings with directly measured arterial blood oxygen saturations in patients who had low peripheral perfusion after cardiac surgery under hypothermic cardiopulmonary bypass. An overall ranking of performance of each pulse oximeter was calculated using five criteria (accuracy, precision, number of readings within 3% of standard, percentage of readings given within 3% of standard, and expected overread limit in 95% of cases). Two pulse oximeter models, the Datex Satlite and the Criticare CSI503, achieved a combination of accuracy and precision such that 95% of measurements would be expected to be within 4% of the laboratory oximeter value; these two also had the lowest dropout rate.

Morris *et al.*⁷ compared the response of a group of oximeters to inflation of a brachial arm cuff to raise venous pressure, thereby lowering perfusion pressure. Some but not all appeared to show decreasing Sp_{O_2} as perfusion pressure decreased.

Oximetry during and after open heart surgery was investigated by Pälve and Vuori.¹³⁵ In 33 open-heart surgery patients immediately after open-heart surgery and in the intensive care unit after the operation, neither low cardiac index ($\leq 2.2 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$) nor low peripheral temperature ($\leq 28^\circ \text{C}$) was found to affect the reliability of three different pulse oximeter models. Kurki *et al.*¹⁴³ found that an Ohmeda Biox III continued to function during nonpulsatile bypass, although with somewhat degraded accuracy, while a Nellcor N-100 ceased functioning.

MOTION ARTIFACT

This subject has also been explored more systematically, both in investigation and by anecdotal reports since 1988.

When subjects are awake, movement is the most common cause of failure and false alarm.⁴⁵ In 63 dental visits, 87–90% of the 235 desaturation episodes were due to patient movement. Similarly, in children with a diagnosis of croup there was poor correlation of clinical status and respiratory rate with low SpO_2 .¹⁴⁴ Frequent dips in SpO_2 were caused by technical problems such as movement artifact.

Probe motion may cause either absent or incorrect readings—the latter particularly if the motion contains frequencies of 0.5–4 Hz (heart rate range). The impression is widespread that shivering interferes. In an effort to simulate recovery shivering and transport, Langton and Hanning¹⁴⁵ vibrated the hand at 4–8 Hz. They noted artifactual decreases of SpO_2 in some instruments (Ohmeda 3700 and Simed S100 but less in Nellcor N-200 with ECG link and Datex Satlite) and extra delay in the response to sudden inhalational hypoxia. Tonic-clonic seizures did not prevent the use of pulse oximeters to document and treat severe desaturation.¹⁴⁶ Vibrational motion during helicopter transport appears not to be a problem.^{65,66}

In a study of eight pulse oximeters during moderate exercise on a cycloergometer, Barthelemy *et al.*¹⁰⁸ were able to distinguish two statistically different subgroups in which Datex, Physiocontrol, and Radiometer oximeters were accurate to within $\pm 1.9\%$ of SaO_2 while Nellcor, Ohmeda, Novamatrix, Kontron, and Hellige varied by ± 2.8 – 5.0% from SaO_2 .

During cardiopulmonary resuscitation, the body motion induced by rescuers can produce oximeter readings of about 85–90% SpO_2 without any actual circulation,¹⁴⁷ in keeping with earlier observations that SpO_2 displays that are generated by motion artifact often tend toward 85%, the SpO_2 at which the alternating current/direct current (AC/DC) ratio of ratios is 1.0.^{148,149}

While monitoring 23 infants with sleep apnea problems (sudden infant death syndrome) at home, Poets *et al.*¹⁵⁰ reported the pulse oximeter to fail in 7 of 69 apneic episodes because of signal loss from movement artifact, whereas they saw no failure of alarm with tCP_{O_2} monitoring, underscoring the apparent advantage of tCP_{O_2} monitoring in this population. (See "Effect of Pulse Oximetry on Other Monitoring Methods").

To reduce motion artifact, several manufacturers couple the ECG signal to the oximeter to synchronize detection to heart rate (Nellcor N-200, Criticare US). To our knowledge, ECG coupling has not been studied in connection with shivering, although Langton and Hanning¹⁴⁵ found it to be useful when motion was artificially produced via an "industrial vibration facility." In a neonatal intensive care unit, Barrington *et al.*¹⁰⁴ reported a nearly 50% reduction (from an incidence of 4.1–2.1%) in motion artifact when ECG coupling was added in the neonatal intensive care unit. They found pulse oximeters to be unreliable for 11.9–25% of events when used in the short

averaging mode (display representing average of < 6 s of data), and from 13.8% to 29% in the longer averaging mode. ECG coupling has also been claimed to be of use in experimental hypotension.¹³⁶

ABNORMAL PULSES

Some oximeters detect a large dicrotic notch as a separate heartbeat, giving about twice the actual rate but (usually) a correct SpO_2 . Venous pulsations due to tricuspid insufficiency¹⁵¹ or ischemic cardiomyopathy have caused low SpO_2 ,¹⁵² and venous pulses have been blamed for low or absent readings on the forehead in supine patients.¹³⁶

VENTILATOR-INDUCED PULSE INTERFERENCE

With positive pressure ventilation, cycling venous and arterial pressure may cause continuous searching for an optimal signal in some instruments (*e.g.*, Nellcor).¹⁵³ One letter writer¹⁵⁴ makes a virtue of necessity by using ventilator-induced changes in oximeter pulse amplitude as an indicator of developing hypovolemia.

RESPONSE TIMES

Slow finger circulation due to cold vasoconstriction may delay responses by more than 1 min after pulmonary oxygen change at normal blood pressure and by much longer periods during severe hypotension.⁷⁷ Similar delays have not been reported when probes are used on the ear, forehead, nose, or lip.

AMBIENT LIGHT

If ambient light is very strong, or is flickering at frequencies similar to harmonics of the light-emitting diode (LED) pulse rate, it may interfere with the rate and saturation measurements. (Manufacturers either synchronize LED pulse rates with harmonics of line power [Ohmeda] or choose rates with no harmonic overlap.) Some lights (fluorescent and especially xenon operating room lights) have caused both falsely normal and high readings even without a subject connected. In one rare instance,¹⁵⁵ a Nellcor N-100 probe continued to indicate 100% SpO_2 in response to ambient light while the patient became hypoxic because of unrecognized extubation. A 15-W handheld mercury vapor fluorescent light caused Nellcor readings to decrease dramatically in infants with disposable finger probes.¹⁵⁶ Although anecdotal reports have suggested interference by phototherapy lights and heaters, Zubrow *et al.*¹⁵⁷ were unable to find such effects in a newborn nursery.

Opaque covering of the probe is helpful in minimizing the effects of ambient light.

ELECTROCAUTERY

Many early pulse oximeters failed in the presence of cautery. With design changes such as careful shielding and filtering, most oximeters are now somewhat better protected. Separation of the probe from the site of surgery and the ground pad may help if problems are seen. In a first-generation Kontron 7840, using an unshielded probe, the memory (random-access memory) was permanently "corrupted," presumably by electrocautery, causing it to read $\text{SpO}_2 = 95\%$ and heart rate = 139 beats/min at all times.¹⁵⁸

INTERFERENCE OF MAGNETIC RESONANCE IMAGING

Ferromagnetic materials within or near the scanner can distort the magnetic field, and monitoring wires act as antennae and may cause granularity in magnetic resonance imaging (MRI).¹⁵⁹ In our own experience, there are remarkable differences between different oximeters, without obvious reason. Unfortunately, no one has reported a comparative study of a variety of pulse oximeters, so the user is left to testing and choosing for his or her own environment. MRI sometimes interferes with pulse oximetry, apparently because of radiofrequency signal detection; an abrupt change in SpO_2 as imaging begins may indicate such interference. Grounding and filtering of radiofrequency have been shown to reduce or eliminate these problems,¹⁶⁰ but this may require special probe extender/couplers, with grounding of the oximeter cable shield to the nuclear magnetic resonance magnet case. In a recently described novel approach, fiber optics replaced wires between the patient and device, eliminating both MRI interference and possible burns.‡

ALTERNATIVE SITES

In general, the pulsatile or AC fraction of total light is smaller from the ears than from the fingers because the DC component is greater (thin ear transmits more light), so most pulse oximeters use the finger, except when peripheral vasoconstriction or hypotension limits finger perfusion. Initially, the Biox and Ohmeda ear probes were heated to 37° C to induce local vasodilation, but this practice has been discontinued. In infants, flexible probes work through the palm, foot, penis,¹⁶¹ or even arm.¹⁶² The bridge of the nose and nasal septum have been used, but for unknown reasons the Nellcor nasal (bridge) probe

read $4.7 \pm 1.4\%$ higher than SaO_2 in ten postoperative or nonoperated patients with mean SaO_2 of 91% (range about 85–94%)^{163,164} and $2.8 \pm 3.9\%$ too high in 15 post-cardiac surgery patients. Three other groups reported similar high-reading errors.^{138,165,166} Probes have been mounted successfully across the tongue,^{167,168} although they are difficult to maintain in place.¹⁶⁹ The cheek may be used by placing either a light source or a detector inside the mouth,¹⁷⁰ to be held in place by the malleable metal from the bridge of an oxygen mask so that the buccal mucosa and skin are transilluminated. The wing of the nostril provides a useful site.

It has often been noted that the SpO_2 reading differs from site to site. The initial comparison of ear and finger sites by Severinghaus and Naifeh¹⁷¹—showing both faster response and greater accuracy at very low saturation for the ear—has been supplemented by Clayton *et al.*,¹³⁶ who studied the performances of ten pulse oximeters using finger probes and compared them to the same pulse oximeters using alternative probes (eight finger probes, two nose probes, and a forehead probe) in poorly perfused patients. Nose and forehead probes performed poorly. Some ear probes performed well compared to some finger probes, but the overall performance of probes in other sites compared to finger probes was worse ($P = 0.05$). Two of eight ear probes and no nose or forehead probes were reliable within 4% of the reference value in 95% of readings. Putative causes included 1) low blood flow causing actual desaturation of the pulsing blood due to local tissue oxygen consumption, 2) light leakage around instead of through the tissue, 3) venous pulsation, associated with venous congestion, and 4) variations of tissue thickness.¹⁶⁶ At low saturation, the ear read closer to SaO_2 than the finger in most tested oximeters.¹⁷¹

REFLECTANCE OPERATION

Adults

Cui *et al.*¹⁷² reported the reflectance spectrum of blood in tissue. The peak absorption is in the green region (as with blood *in vitro*), suggesting that the largest pulse signal would be in that region, although that color is not used in present devices. Mendelson *et al.*^{173,174} obtained satisfactory monitoring with a forehead reflectance probe of their own design. Cheng *et al.*¹⁷⁵ reported that a Criticare forehead probe performed well in healthy subjects but was less satisfactory in critically ill patients. Although this site might be expected to have some advantages in restless patients, reflectance from skin surfaces seems little used and has not yet been widely tested for accuracy. On the forehead at about 55% SaO_2 , the bias and precision were found to average $-0.7 \pm 4.0\%$ with Criticare, -0.2 ± 6.5

‡ Shellock FG, Kimble K, Myers S: Monitoring heart rate and oxygen saturation during MRI with a fiber-optic pulse oximeter (abstract). 10th Annual Meeting, Society of Magnetic Resonance Medicine 2: 974, 1991. Device: Nonin Inc. (Plymouth, MN).

with Datex, and -1.6 ± 5.5 with Kontron oximeters,¹⁷⁶ accuracies that matched or surpassed finger data with the same instruments. In each case the probes were experimental. A Simed forehead probe tested by Decker *et al.*¹⁷⁷ was less stable, more subject to motion artifact, and less accurate. Failure to detect signal is more common on the forehead than on the finger. Infrared light penetrates more deeply than red light,¹⁷⁸ so the paths of the two wavelengths traverse different tissue and thus detect different degrees of pulsatility. To obtain accurate tracking at low saturation, it was found that the infrared LED needed to be located slightly closer than the red LED to the detector and that the spacing between the infrared LED and detector should be at least 1 cm.¹⁷⁶

At least one manufacturer is now actively marketing reflectance oximetry (Ciba-Corning). Barker *et al.*¹⁷⁹ found an unacceptable 59% signal failure rate for this instrument used on the forehead and a 27% rate for the same on the finger, compared to signal failure rates of 1.3–5.8% for various transmission oximetry finger probes.

Mendelson and McGinn¹⁸⁰ found reasonable agreement (within 2–3%) between a standard finger probe and reflectance sensors on the forearm and calf, but it is noteworthy that the reflectance sites required heating of the skin to 40° C to produce a detectable signal.

Fetal

Reflectance oximetry in the fetal presenting part has been investigated with mixed results. Johnson and associates reported reasonable data obtained by placing a reflectance probe on the fetal scalp *via* the vagina.¹⁸¹ The authors restricted their study to labors involving vertex presentations without palpable caput or meconium staining of amniotic fluid. Continuous fetal monitoring was achieved in all 86 of the labors thus selected. Amniochorionic membranes did not appear to interfere with measurements either *in vitro* (by absorption spectrum analysis) or *in vivo*. The same group also monitored fetuses during caesarean section.¹⁸¹ Another group¹⁸² attempted application of pulse oximetry to intrapartum monitoring in 105 women. No adequate reading could be obtained in 44 cases. Two major sources of artifact, related to probe apposition and signal processing, were identified and excluded. The average SpO₂ from the fetal scalp was 82% (standard deviation 6%), which is higher than has been inferred from PO₂ levels. Major problems were attachment of the probe and stagnant blood when palpable caput was present. Johnson *et al.*¹⁸³ focused on this issue by studying 20 neonates with significant caput succedaneum. The scalp average SpO₂ was 69% when SaO₂ was 84%, and in one extreme case at 90% SaO₂ the caput sensor read 40%. These studies suggest the need for a probe designed to slip inside the cervix beyond the caput, when it is present.

SKIN PIGMENTS, DYES, NAIL POLISH

In black patients, erroneously high readings (about 3–5%) and a higher incidence of signal detection failure have been reported.^{61,184–186} In black people, the nail bed and fingertips are usually less pigmented than other areas of the skin. This effect has been simulated using an *in vitro* model.¹⁸⁷ Injected methylene blue and indocyanine green are known to produce transient false desaturation.¹⁸⁸ The effects of dyes, dyshemoglobins, and other pigments were recently reviewed by Ralston *et al.*¹² They pointed out that although nail polish reduces total light and may render the signal too small and cause up to 6% underestimation, probes can be mounted from side to side on a finger. (The effect of nail polish at low SaO₂ has not been studied, but it is predicted to cause overestimation of SaO₂.)

For a comprehensive statement about the effect of nail polish on pulse oximetry, the work of Coté *et al.*¹⁸⁹ remains definitive. Their analysis, which considers the relative slopes of the nail polish absorbance curve at the two wavelengths of interest, has implications beyond the effects of nail polish; according to their research, pigment may cause error despite its nonpulsatility. Indirect evidence for this is provided by anecdotes about low SpO₂ readings from fingerprinting ink (which can even be transferred from finger to oximeter probe),¹⁹⁰ from henna, a stain used by some Middle Eastern women on the fingers and toes,¹⁹¹ and from meconium in newborns.¹⁹²

Jaundice appears to have no direct effect on pulse oximetry but may cause confusion because it may be read by multiwavelength laboratory oximeters as increased carboxyhemoglobin (COHb) or methemoglobin (MetHb) or both, reducing the *in vitro* O₂Hb reading.¹⁹³ This laboratory error was found greatest with the IL282, less with the Corning Co-2500, and not at all with the Radiometer OSM3.¹⁹⁴ These authors¹⁹⁴ also caution that jaundiced patients often have truly elevated COHb and MetHb as well, which may reduce O₂Hb (but not SaO₂).

CARBOXYHEMOGLOBIN AND METHEMOGLOBIN

With pulse oximeters, COHb is nearly indistinguishable from O₂Hb.¹⁹⁵ In a clinical comparison of pulse oximeter SpO₂ against values obtained from a laboratory blood oximeter in a patient with carbon monoxide poisoning, SpO₂ was equal to the sum of the O₂Hb and COHb values, with COHb as great as 30%.¹⁹⁶ Multiwavelength laboratory blood oximeters read and report O₂Hb as a percentage, which can underestimate SaO₂ by as much as the sum of the concentrations of COHb and MetHb. Consider, for example, a situation with 15% COHb and 10% HHb. The multiwavelength laboratory analyzer will report O₂Hb as 75%. (Some devices may term this "oxygen

saturation.”) Arterial oxygen saturation will be interpreted as approximately 90% by a pulse oximeter, measured as 90% by gasometric analysis, and computed as about 88% from blood gas analysis, due to the left shift of the dissociation curve caused by COHb. The pulse oximeter Sp_{O_2} of approximately 90% indicates the presence of 10% HHb, desaturated normal Hb, but neither pulse oximetry nor blood gas analysis provides any clue to the presence of COHb.

The effects of MetHb on pulse oximetry has been studied extensively.^{197–204} MetHb absorbs both 660- and 940-nm light. Barker *et al.*¹⁹⁸ found that Sp_{O_2} readings decrease with MetHb, but by only about half of the MetHb percent concentration up to 20%, whereas at higher MetHb concentrations, Sp_{O_2} tends toward 85%. MetHb is commonly caused by use of 20% benzocaine sprays and ointments and may be first detected by pulse oximetry.^{197,198,203,204}

When clinically significant levels of COHb or MetHb are suspected or known, *in vitro* blood gas analysis and oximetry should supplement pulse oximetry, not only because dyshemoglobins interfere with pulse oximetry but also because they decrease the oxygen-carrying capacity of blood.

POTENTIAL DANGERS

Instances of second- and even third-degree skin burns have been reported in connection with pulse oximetry during MRI^{205,206} due to induced skin current beneath looped pulse oximeter cables acting as antennae. Burns also have been caused by using probes with defects or defective designs²⁰⁷ and by plugging a Physio-Control probe into an Ohmeda oximeter.²⁰⁸ There is a possibility of pressure necrosis from some probes left on a single site too long (if a spring compresses the tissue or if tape is applied too tightly²⁰⁹). At high FI_{O_2} , changes such as hypoventilation or endobronchial intubation may cause large changes in Pa_{O_2} without altering Sa_{O_2} significantly and thus may go undetected by pulse oximetry. tcP_{O_2} monitoring, on the other hand, is relatively sensitive to such changes at high FI_{O_2} .⁵⁴ As Tremper and Barker¹ aptly put it, “the pulse oximeter is effectively a sentry standing at the ‘cliff’ of *desaturation*” (*italics ours*)—while tcP_{O_2} monitoring is stationed well up the road.

FALSE ALARMS AND FALSE NONALARMS

Most false alarms are due to motion. Pan and James¹⁷ demonstrated in a study of 43 post-caesarean section patients monitored on the ward that 88% of false alarms could be avoided by incorporating a 60-s wait-period before sounding the alarm. When the lower Sp_{O_2} alarm limit

was set at 90%, with a 60-s wait, the maximum frequency of alarms was reduced from 324 to 4 alarms per 24-h period per patient. It was originally expected¹⁴⁸ that some oximeters without adequate signal might continue to indicate a Sp_{O_2} of 80–90% when saturation is much lower. Direct attempts have failed to confirm this.⁷⁷ An instrument that frequently alarms or drops to zero because of motion or weak signals may be ignored by the physician when real desaturation has occurred. A low but nonzero reading should be treated as a danger signal of hypoxemia until proven otherwise, whereas a zero reading usually means no signal. Unfortunately, alarms may be so distracting when problems arise that users may turn them off or even turn the oximeter off if the alarm cannot be silenced, in order to concentrate on the problems.

Accuracy

The indices used to quantify error in connection with accuracy testing of oximeters have been criticized^{1,210} and changed as investigators search for a single numerical description of accuracy. Regression analysis was soon found inadequate because in most studies no deliberate wide variations of Sa_{O_2} were introduced and because even a good correlation does not imply an accurate measurement, just as a poor correlation does not imply an inaccurate measurement. Bias, which is defined as the mean error ($Sp_{O_2} - Sa_{O_2}$) and its standard deviation are currently the preferred error indices,^{1,2} but a single bias value derived from a large range of Sa_{O_2} (*e.g.*, 40–100%) is less than ideal because bias usually is greater at low Sa_{O_2} .^{148,176} Bias and its standard deviation should therefore be presented as a function of Sa_{O_2} range. The standard deviation of the bias has sometimes been called “precision.” However, precision has also been defined as mean absolute error. A bias of $10 \pm 1\%$ standard deviation would have a precision of 1% according to the former but 10% according to the latter. An additional possible confusing aspect is that a high numerical “precision” reflects a poor performance.

A table summarizing 39 accuracy studies from 1983 to 1988 was included in a previous review.² Subsequent studies of accuracy are likewise numerous.^{7,49,101,211–217} In general, the claim of most manufacturers that errors are $< \pm 3\%$ at $Sa_{O_2} > 70\%$ has been confirmed.^{10,11,171,176,216,218} Such studies continue to appear, sometimes with special groups of patients. In a subset of 17 patients with chronic obstructive pulmonary disease, Hannhart *et al.*²¹⁸ found consistent overestimation of Sa_{O_2} by Sp_{O_2} at low saturation in at least three oximeters (Nellcor, Ohmeda, and Critikon), but the bias was within 4% for all instruments. This group also found overestimation error with the same instruments in steady-state hypoxic tests in normal subjects. Their “gold standard,”

the two wavelength OSM-2 (Radiometer), is less dependable than the newer multiwavelength oximeters. Most other workers have not found similar high readings at low saturation.

Pulse oximeters may be considered more than sufficiently precise for most clinical purposes (except perhaps for detecting neonatal hyperoxia; see above). The above-described limitations on accuracy appear minor in comparison with requirements for effective patient monitoring. The device's ability to detect an unfavorable trend in oxygenation—subject to acknowledged limitations at high FI_{O_2} and in the presence of artifact—can be at least as important as its accuracy. In the clinical setting, pulse oximeters often function appropriately as “desaturation meters,” with a low Sp_{O_2} taken to imply a low Sa_{O_2} until proven otherwise by clinical evaluation.

EFFECT OF ANEMIA ON ERRORS

Retrospective analysis in tests of 43 oximeters of 12 manufacturers²¹⁹ disclosed a negative error inversely proportional to Hb concentration when Sa_{O_2} was < 80%. The mean error when $Sa_{O_2} = 54.5\%$ appeared to be a nearly linear function of Hb concentration, with no error at 14.5 g/dl. In 45 determinations with 13 oximeters at Hb = 8.2 g/dl, the mean bias at 53% Sa_{O_2} was $-15 \pm 9\%$, of which 8% could be attributed to anemia and 7% to oximeter errors at normal Hb concentrations. This was a “fail-safe” error, providing exaggerated warning of hypoxemia in anemia.

Anemia has been studied in dogs by Lee *et al.*²²⁰ The overall Sp_{O_2} bias and precision was $0.2 \pm 7.6\%$ for 178 points. Below 10% hematocrit, the bias and precision worsened to $-5.4 \pm 18.8\%$.

No errors or problems were detected in the use of pulse oximeters in 27 burned patients.²²¹

IN VITRO METHODS OF TESTING THE ACCURACY OF PULSE OXIMETERS

De Kock and Tarassenko²²² attempted to investigate the effect of a number of physiologic parameters on pulse oximetry accuracy in an *in vitro* model but limited their conclusions to the effects on the pulsatile or AC ratio of red to infrared light. At > 50% saturation, pulse oximeters were not affected by variations in hematocrit, blood flow rate, tissue blood content, or pulse amplitude. A variety of *in vitro* models have been tried in an effort to test oximeter response more simply, especially at very low saturation.^{223–227} Volgyesi *et al.*, using a mechanical *in vitro* finger model with two oximeters,²²⁵ reported a strongly positive (*i.e.*, dangerous) bias error ($Sp_{O_2} - Sa_{O_2}$) with normal Hb (16 g/dl) at low (50%) saturation. The error disappeared with 3-fold dilution of blood.²²⁶ In view of the conflict between these *in vitro* results and the *in vivo*

tests reported above, the validity of these finger models was inadequate as described. Volgyesi has been able to correct this error by use of a thinner (less opaque) model. § Aoyagi and Miyasaka²²⁷ devised a model finger with a layer of blood on one side of an elastic membrane and milk on the other side in a rigid opal cuvette. This model yielded a relationship (of Φ vs. S_{O_2}) “similar to that of the human finger” down to 60% S_{O_2} (the limit of testing). Φ is the ratio of ratios, $(AC_{red}/DC_{red})/(AC_{infrared}/DC_{infrared})$. However, neither this nor any *in vitro* calibration models have been documented *in vivo* as adequate to represent human responses at low saturation.

Evaluations of Pulse Oximeter Performance and Features

New to the literature on pulse oximetry is the *Consumer Reports*-style comparison of multiple features of several devices. Alexander *et al.*,¹⁶ evaluating nine oximeters, compared 21 aspects of each, gave their ranking of overall performance, and judged four to perform significantly better than the other five (the Ohmeda 3700, In-Vivo 4500, Nellcor N-200, and Datascope Accusaf, were better than the Criticare 501+, Physio-Control 1600, SARA, SensorMedics, and Catalyst MiniOx). This ranking differed from that based only on bias and precision data obtained at lower saturations^{171,176} because accuracy (which they evaluated only down to $Sa_{O_2} \approx 85\%$) contributed only a small and undefined part of their determination of performance rank. Morris *et al.*⁷ compared 15 oximeters, listing 41 features, and were able to divide them into “two major groupings with considerable variation in each group.” The reader is referred to these comparisons for further details.

The papers listed and described above (under “Accuracy”) all are partial performance evaluations in that all of the tested instruments worked, providing useful data. The other features evaluated by Alexander *et al.*¹⁶ and Morris *et al.*⁷ should be considered in this context. There appears to be relatively little difference in failure at low perfusion and pressure among tested instruments, while some evidence does support differences in motion rejection as described (see “Limitations of Pulse Oximetry: Motion Artifact”).

Does Pulse Oximetry Increase Patient Safety?

E. C. Pierce, Jr., President of the Anesthesia Patient Safety Foundation, recently suggested¶ that since 1984,

§ Volgyesi GA: Personal communication to Severinghaus JW. December 19, 1991.

¶ Pierce EC Jr: Anesthesia Patient Safety Movement, 1991. ASA Newsletter 55(11):4–8, 1991.

In my view, it is probable that mortality from anesthesia in the healthy patient has declined from the range of 1 to 2 deaths per 10,000 anesthetics to the range of 1 per 100,000 or more anesthetics, at least a 10-fold decrease.

If it is true, why has this change occurred? While technological advances in anesthesiology are considered a strong factor by many, perhaps more important is the way anesthesiologists view their practice today, given the explosion of anesthesia patient safety and risk management endeavors.

Is it possible to dissect from these improvements the contribution of pulse oximetry? Many of the studies reviewed above, like several earlier studies,^{1,2} appear to demonstrate clearly that pulse oximetry is superior to clinical judgment (and to capnography²⁰) in providing early warning of hypoxic events. Although this capability is accepted *prima facie* as impressive and valuable by most anesthesiologists, it is equally impressive that *no published investigation has yet demonstrated that pulse oximetry makes a difference in morbidity and mortality.* The task of making such a determination is daunting in view of the relative safety of anesthesia even before pulse oximetry was available; very large numbers of patients may be required.

In general, the myriad studies documenting clinically undetected hypoxemia aim only at description. They do not answer the questions raised by Fairley²²⁸ and others: "What kind of desaturation is *unacceptable*? Under what circumstances? For how long? In whom?" (italics ours). No answers will ever satisfy all conditions for all observers and patients. At the physiologic level, recent pulse oximetry data do provide some surprises about how tolerable severe hypoxemia is in humans. Saturation cycling repeatedly down to 30–40% has been recorded during sleep without detectable brain or other systemic damage both in subjects with chronic mountain polycythemia²²⁹ and in obese patients with chronic obstructive pulmonary disease and sleep apnea.^{111,230} In addition, the subtle evidence of permanent brain injury reported by Hornbein *et al.*²³¹ in Mount Everest climbers was found in those who had the best hypoxic ventilatory drive, and who therefore were presumed to have been least hypoxic but most hypocapnic during exposure. On the other hand, when anesthesiologists find SpO₂ decreasing from normal to 90% without evident cause, they may well judge this sufficiently unacceptable to warrant further investigation and therapy.

The near-uncritical enthusiasm that greeted the introduction of pulse oximetry has become tempered, especially in the past 3 yr, by commentary regarding the paucity of risk/benefit data. As Keats¹⁸ so trenchantly observed, *prima facie* clinical impressions need not bear any relationship to what might be demonstrable *via* that grail of clinical research, the outcome study: "Without [outcome data on pulse oximetry] I can envision a subpopulation of this country walking around without their front teeth because of urgent intubation when an oximeter read less than 90%."²³²

Attempts to examine outcome with pulse oximetry date back to 1987 when Cooper *et al.*²³³ showed that significantly fewer patients experienced "recovery room impact events" such as hypotension and dysrhythmia after pulse oximetry was introduced into the operating room. Their study fell short of establishing a cause-and-effect relationship, however, perhaps (as the authors suggest) because of insufficiently sensitive definitions of "impact events."

In 1988 Coté *et al.*²³⁴ showed that "major hypoxic events" (SpO₂ < 85% for ≥ 30 s) were significantly less common when pulse oximetry was available to the anesthesia team, and thereby drew editorial praise²³⁵ for demonstrating the device's early-warning capability. Coté *et al.* also found, however, that "no morbidity was documented in any patient [in either group] who suffered an hypoxic event." The 1991 study by Coté *et al.*²¹ makes no mention of differences in morbidity or mortality.

A 20,802-case prospective controlled study in Denmark in which half were monitored by pulse oximetry failed to show an effect of that monitoring on morbidity or mortality, except for a decreased incidence of intraoperative myocardial ischemia.^{236,237} Although the final report has not been published, data in the abstracts suggest that there were 113 deaths in the monitored group and 94 in the group not using oximetry (difference not significant). Four deaths in each group (of 10,000) were believed to be related to anesthesia. (Note: this study included all ASA categories, which suggests that anesthesia-related deaths are more common in patients who are more severely ill.)

Another approach to assessing the benefits of pulse oximetry is retrospective. The Closed Claims Project of the Professional Liability Committee of the ASA²³⁸ reported in 1989 a review of 1,175 claims from 17 insurance companies. The reviewers determined that, of 348 "preventable" injuries or deaths, "pulse oximetry . . . would have been efficacious in preventing injury in 138 cases."

The need for comparable routine use during postoperative transport and recovery is supported by evidence of hypoxic injury after discontinuation of operating room oximetry.**

Eichhorn,²³⁹ in a 1989 analysis of the anesthetics of an estimated 1,001,000 ASA I and II patients in the Harvard hospitals between 1976 and mid-1988, found 11 major anesthesia-related accidents, 7 or 8 related to inadequate ventilation or oxygenation, only 1 of which occurred after pulse oximetry began to be used routinely in 1985. A thoughtful editorial by Orkin²⁴⁰ accompanied the article, pointing out that this was not a statistically significant difference and that neither Eichhorn's nor any other work had yet shown clear evidence of a cost-benefit justification for additional monitoring, including pulse oximetry.

** Cheney FW: The ASA Closed Claims Project after the pulse oximeter: A preliminary look. ASA Newsletter 54:10, 1990.

Caplan *et al.*,²⁴¹ using the Closed Claims Project database, described 14 cardiac arrests in healthy patients during spinal anesthesia between 1978 and 1986. All were monitored by blood pressure cuff and 13 by ECG, but apparently none were monitored by pulse oximetry. In 12 of these patients, intravenous sedation or opioids had been given, although never "inappropriately or carelessly" in the reviewers' judgment. Because "half of the patients were verbalizing at the time of arrest," the authors believed that "respiratory changes produced by sedation may have played an important role in approximately one-half of the arrests." They postulated an adverse cascade of physiologic events, involving hypoxia, hypercapnia, vasodilation, and inability to maintain circulatory homeostasis in the face of sympathetic block. On the basis of this information, the authors recommended pulse oximetry when sedatives are given or the patient is otherwise not communicating well, but they refrained from judgments about whether any of the arrests might have been thereby "preventable."

More recent work by Caplan's group,²⁴² using cases from the Closed Claims Project database, has suggested the possibility that knowledge of the severity of outcome could influence a reviewer's judgment of the appropriateness of care, which may prompt a reinterpretation of the Closed Claims Project's findings about the preventability of injury by monitors. Keats,²⁴³ commenting on ongoing Professional Liability Committee work, notes that "cases that only two years ago would have been classified as inadequate ventilation . . . now . . . have oximeters in place and they show no desaturation." In view of this very recent development, clinical intuitions about the benefits of pulse oximetry continue to await rigorous corroboration.

We conclude that pulse oximetry *probably* did contribute to the increasing safety of anesthesia. In one sense, however, this change may have come through the device's educational role in promoting vigilance and awareness of inadequacies in technique. We suspect that the experience anesthesiologists have had in the last decade with oximetry would ensure safer anesthesia even if a functioning oximeter were unobtainable. However, we may never be able to prove it.

Effect of Pulse Oximetry on Other Monitoring Methods

Pulse oximetry and tcPO_2 measurement were invented in the same year (1972). The need to protect premature infants from hyperoxia resulted in rapid application of tcPO_2 in nurseries within 5 yr, whereas pulse oximetry development and application required > 10 yr, as it awaited both technological development and realization

of its potential benefits in all anesthesia and critical care settings.

tcPO_2 closely estimates PaO_2 in hemodynamically stable infants, permitting control of oxygenation to avoid retinal oxygen damage (see "Role in Preventing Retinopathy of Prematurity"). Comparison of tcPO_2 with oximetry in hemodynamically stable infants and children showed no difference in the value of SaO_2 computed from tcPO_2 versus measured SaO_2 or SpO_2 .^{244,245} Poets *et al.*¹⁵⁰ compared oximetry and tcPO_2 as home monitors in 23 patients (age 0.5–40 months) with recurrent cyanotic episodes. Of 69 episodes in which the arterial oxygen saturation was $\leq 80\%$ for ≥ 20 s and/or central cyanosis was present, the tcPO_2 monitor alarm sounded (≤ 20 mmHg or 2.67 kPa) in every episode. The pulse oximeter (Poet®, Criticare) identified hypoxemia in 62 of 69 episodes, failing in seven episodes because of signal loss from movement artifact. In 32 episodes in which SaO_2 decreased to $\leq 60\%$, the tcPO_2 monitor alarm sounded after a median time interval of 16 s (maximum time interval 30 s). Indications for monitoring included apparently life-threatening events or cyanotic episodes ($n = 163$), prematurity and prematurity related disorders ($n = 86$), and sudden unexpected death in one or more siblings ($n = 122$).

In adults and during anesthesia, tcPO_2 is variable, usually averaging 60–80% of PaO_2 .²⁴⁴ However, pulse oximetry has several advantages over tcPO_2 monitoring: permanent calibration; speed; simplicity; extremely low incidence of burns; no drift; and no electrode membrane failures. Perhaps most important, oximetry can be used and maintained by personnel involved in direct patient care rather than by specially trained technical support staff. Accordingly, pulse oximetry has both supplemented and partially replaced tcPO_2 in the care of premature infants.

Because of pulse oximetry, the use of arterial blood gas analysis has decreased in patient care arenas,⁵⁹ but it remains essential for pH and PCO_2 measurement, for high PO_2 analysis in the determination of shunts, for instances when pulse oximetry fails, and for determination of whether a low SpO_2 reading reflects arterial or only finger tissue hypoxia. By 1988, institutional surveys had already suggested that pulse oximetry had reduced the need for arterial blood gas analysis on various hospital services²; this was supplemented in 1991 by a study of 20,120 emergency-room visits in which physician orders for arterial blood gas analysis decreased by 37% without adversely affecting the quality of emergency care.²⁴⁶

Regulation, Insurance, Standards, and Cost

On January 1, 1990, pulse oximetry became an ASA standard for basic intraoperative monitoring, and after January 1, 1992 the same standard applied to the postan-

esthesia care unit,^{††} stated as follows: "During recovery from all anesthetics, a quantitative method of assessing oxygenation such as pulse oximetry shall be employed in the initial phase of recovery." An ASA publication^{‡‡} described the various state governmental, medical, and insurance regulation agency actions relative to the use of pulse oximetry. These efforts began in 1986 in Massachusetts with the establishment of a risk management unit in the Board of Registration in Medicine, set up by the state legislature "to provide technical assistance and quality assurance programs designed to reduce or stabilize the frequency, amount and cost of claims against physicians." The Board was given the authority to promulgate regulations requiring physicians to participate in risk management programs as a condition of licensure. The Medical Malpractice Joint Underwriting Association of Massachusetts was required, subject to the Insurance Commissioner's approval, "to impose a system of discounts for physicians or other insured who participate in risk management activities." By 1987 the Joint Underwriting Association and the Commissioner had approved the use of standards (243 CMR 3.00) that reduced premiums for "anesthesiologists who participate in risk management activities that would require the use of a pulse oximeter, whenever physically possible. . . . The 20 percent premium discount in 1989 translates into a savings of approximately \$4000 per year for each anesthesiologist who holds a policy that provides \$1 million/\$3 million coverage." An updated review of state efforts has been published.^{§§}

According to Pierce,^{¶¶} between 1985 and 1990, the "liability insurance relativity factor," an industry index of relative risk, decreased from 5.0 to 2.5–3.0. In the 1975–1978 period, anesthesia liability claims paid by members of the National Association of Insurance Commissioners were 367% of the average of all specialties' payments, whereas in 1986–1987, the ratio had fallen to equal the mean (103%, St. Paul Insurance Co). It may never be known whether the temporal relationship of reduced insurance costs and payments to the introduction of pulse oximetry was incidental or causal. Concurrent with the introduction of pulse oximetry came far greater emphasis on patient safety, increased use of capnometry, and appreciation, in part due to publication of closed

claims analyses, that there were preventable gaps in anesthesia practice in which hypoxia occurred.

The American Society for Testing and Materials Committee has proposed a specification standard for pulse oximeters.^{¶¶} Similarly, the International Standardization Organization has proposed a draft standard for pulse oximeters.^{***} Neither document is final; the contents are subject to change; and their impact on manufacturers cannot be accurately foreseen.

Possible Effects of Oximetry on Anesthesiologists

Pulse oximetry is remarkable among monitors in that it involves no calibration, negligible time lag, and infrequent false negative data (*i.e.*, infrequent incorrectly high Sp_{O₂}) and requires no routine maintenance, no disposable components, no training, and little interpretation while it continuously and noninvasively displays a vital variable. Oximetry reduces dependence on blood gas analysis, obviates visual dependence on cyanosis to detect hypoxemia, and may make anesthesia safer (and, by implication, easier). Does it thereby threaten to make the anesthesiologist less vigilant, as suggested by Orkin?²⁴⁰ What are the potential effects on anesthesiologists as well as on patients of increasing technological perfection? Can technology and vigilance coexist? Is Hein right in this grook?²⁴⁷

When technology is master
We shall reach disaster faster.

Monitors and associated computers, robots, and printers might replace three human functions: measuring, recording, and responding. An example is servo-control of administration of vapors depending on the patient's end-tidal concentration. Is a machine with alarms vigilant? Does it add to or subtract from supervision and care and attention? What will be the effect on vigilance of automatic generation of the anesthetic record, with which the vital signs can be precisely recorded without having to pass through the brain of the anesthetist?

Snow deplored the separation of intellectual human life into two cultures, sciences and humanities, unable to communicate with each other.²⁴⁸ In anesthesia, the two-culture metaphor translates loosely into technology *versus* vigilance. The contemporary writer Robert Pirsig, in *Zen and the Art of Motorcycle Maintenance*,²⁴⁹ traces Snow's two-

^{††} Standards for postanesthesia care. (Approved by House of Delegates on October 12, 1988 and last amended on October 23, 1990.) ASA Directory of Members, 56th edition, 1991, p 672.

^{‡‡} FYI . . . A commitment to patient safety. ASA Office of Government Affairs, 111 14th Street NW, Suite 501, Washington, DC 20005. January 1990, pp 1–12.

^{§§} Anesthesia Patient Safety Newsletter. Anesthesia Patient Safety Foundation 5(4):40–41, Winter 1990–1991.

^{¶¶} American Society for Testing and Materials document F29.03.10-005. Contact R. I. Godinez, M.D., Ph.D., Children's Hospital of Philadelphia, 34th Street and Civic Center Boulevard, Philadelphia, Pennsylvania 19104.

^{***} ISO/DIS 9919.2. Obtainable from ISO, Case postale 56, CH1221 Geneva 20, Switzerland.

culture dichotomy back to the dialogues of Plato. Pirsig maintains that a catastrophic loss of human cultural development over the past 2,500 yr resulted from alienation between the two cultures, a "Socratic" emphasis on discovery of the facts of nature, and a "Sophistic" preoccupation with merit, virtue, and excellence. The maintenance of the metaphoric motorcycle requires of its user both thorough knowledge of its mechanical workings and limitations and a resolute determination to use one's skills. To combine Socratic science—medical intelligence—with Sophist emphasis on quality in the healing art: that is the goal.

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