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Accuracy of Response of Six Pulse Oximeters to Profound Hypoxia

John W. Severinghaus, M.D., Karen H. Naifeh, Ph.D.†*

Oxygen saturation, Sp_{O_2}%, was recorded during rapidly induced 42.5 ± 7.2-s plateaus of profound hypoxia at 40–70% saturation by 1 or 2 pulse oximeters from each of six manufacturers (NE = Nellcor N100®, OH = Ohmeda 3700®, NO = Novametrix 500® versions 2.2 and 3.3 (revised instrumentation), CR = Criticare CSI 501+® version .27 and version .28 in 501 & 502 (revised instrumentation), PC = PhysioControl Lifestat 1600®, and MQ = Marquest/Minolta PulseOx 7®). Usually, one probe of each pair was mounted on the ear, the other on a finger. Semi-recumbent, healthy, normotensive, non-smoking caucasian or asian volunteers (age range 18–64 yr) performed the test six to seven times each. After insertion of a radial artery catheter, subjects hyperventilated 3% CO_2, 0–5% O_2, balance N_2. Saturation Sc_{O_2}, computed on-line from mass spectrometer end-tidal P_{O_2} and P_{CO_2}, was used to manually adjust FI_{O_2} breath by breath to obtain a rapid fall to a hypoxic plateau lasting 30–45s, followed by rapid resaturation. Arterial Hb_{O_2}% (Radiometer OSM-3®) sampled near the end of the plateau averaged 55.5 ± 7.5%. Sc_{O_2}% (from the mass spectrometer) and Sa_{O_2}% (from *p*H and P_{O_2}, by Corning 178®) differed from Hb_{O_2}% by + 0.2 ± 3.6% and 0.4 ± 2.8%, respectively. The mean and SD errors of pulse oximeters (*vs.* Hb_{O_2}%) were:

Manufacturer	Version	N Ear, Finger	%Sp_{O_2} − %Hb_{O_2} Ear	Finger
NE	N100	60, 60	−0.4 ± 11.7	−6.6 ± 10.8
OH	3700	60, 60	2.4 ± 8.8	−9.0 ± 10.4
NO	2.2	60, 60	4.8 ± 13.9	13.1 ± 12.7
NO	3.3	0, 120	—	1.1 ± 5.4
CR	.27	55, 60	12.0 ± 13.3	−8.1 ± 16.0
CR	.28	36, 60	0.0 ± 3.4	−1.9 ± 4.8
PC	1600	54, 73	−4.3 ± 4.3	−7.9 ± 5.1
MQ	7	0, 36	—	−2.9 ± 5.2

The plateaus were always long enough to permit instruments to demonstrate a plateau with ear probes, but finger probes sometimes failed to provide plateaus in subjects with peripheral vasoconstriction. Nonetheless, Sp_{O_2} read significantly too low with finger probes at 55% mean Sa_{O_2}. The mean error with ear probes was not significant. Several instruments occasionally defaulted to zero saturation during rapid desaturation. Precision was independent of probe location, but differed widely between instruments. The studies provided data with which manufacturers could improve function, as illustrated by subsequent series with CR and NO. The authors conclude that square-waves of hypoxia can assess both the transient and the steady-state profound hypoxic responses of pulse oximeters, disclosing a variety of problems, and facilitating their resolution. An addendum follows the article. (Key words: Blood algorithm for O_2 saturation; oxygen dissociation curve; oxygen saturation; transient hypoxia. Equipment: oximeter. Monitoring: pulse oximetry.)

PULSE OXIMETRY DETERMINES arterial blood O_2 saturation by comparing the pulsatile changes in light transmission through a finger, ear, or other tissue at two wavelengths, one about 660 nM (red), the other at about 940 nM (infrared). This method was invented in 1972–3 by Aoyagi *et al.*‡ Its use was first reported in patients in 1975 by Nakajima *et al.*.§ This device and another Japanese pulse oximeter were tested by various groups of Japanese surgeons and anesthesiologists between 1976 and 1980.[1–4] Tests of pulse oximetry were first reported in the USA in 1980 by Sarnquist *et al.*¶ These devices have now been widely used and undergone accuracy testing.[5–12] At present, there are at least 15 manufacturers engaged in manufacture and sale of pulse oximeters. Although each instrument has been or will be assessed in a group of normals and/or patients before receiving FDA approval in the USA, such approval is granted on the basis of substantial equivalence to previously approved instrumentation.

The calibration of pulse oximeters is built in by manufacturers, and not adjustable by users. The data for calibration are obtained by inducing hypoxia in normal subjects, or, in some instances, by collecting data from patients. The majority of testing of pulse oximeters is done when saturation is higher than 70%, the lower limit of tolerable steady-state hypoxia. The lower 70% of the dissociation curve is, for the most part, not calibrated, but estimated by extrapolation. The relationship between the saturation and the optical signals at the two wavelengths used is not linear or logarithmic (as it would be in clear solutions according to the Lambert-Beer relationship), but is empiric, such that extrapolation from 70% downward is speculative. The instruments detect very small optical signals, and must reject a variety of artifacts, which requires the averaging of data over several seconds at least, and this procedure can affect the response to rapid changes of saturation.

Calibrations done during steady states do not assess the responses to transients. Pulse oximeters use computer programs which average, compute, and weigh the

* Professor of Anesthesia.

† Assistant Professor of Psychiatry.

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Address reprint requests to Dr. Severinghaus: Anesthesia Research Center, 1386 HSE, Box 0542, University of California, San Francisco, San Francisco, California 94143-0542.

‡ Aoyagi T, Kishi M, Yamaguchi K, Watanabe S: Improvement of an earpiece oximeter. Abstracts of the 13th annual meeting of the Japanese Society for Medical Electronics and Biological Engineering, pp. 90–91, 1974.

§ Nakajima S, Hirai Y, Takase H, Kuse A, Aoyagi S, Kishe M, Yamaguchi K: Performance of new pulse wave earpiece oximeter. Respir Circ 23:41–45, 1975.

¶ Sarnquist F, Todd C, Whitcher C: Accuracy of a new non-invasive oxygen saturation monitor. (Abstract) ANESTHESIOLOGY 53:S163, 1980.

TABLE 1. Terminology

NE: Nellcor N100®
OH: Ohmeda (Biox®) 3700
NO: Novametrix 500®, version 2.2 in series 1 and 2, and 3.3 in series 4.
CR: Criticare® CSI 501+, version .27 in series 1 and version .28 (501+ and 502) in series 3
PC: PhysioControl Lifestat 1600®
MQ: Marquest/Minolta PulseOx 7®
Hb_{O_2}%: $100Hb_{O_2}/$(Total Hb), arterial oxyhemoglobin percentage (Radiometer OSM-3®)
Sa_{O_2}%: $100Hb_{O_2}/(Hb_{O_2}$ + Hb), % arterial oxygen saturation herein calculated from *p*H and P_{O_2} (Corning 178®). [In the absence of Hb_{CO} and HbMet (carboxyhemoglobin and methemoglobin) $Hb_{O_2}\% = Sa_{O_2}\%$.]
Sc_{O_2}: % arterial oxygen saturation calculated from mass spectrometer end-tidal P_{CO_2} and P_{O_2}.
Sp_{O_2}: % pulse oximeter saturation.

input and attempt to exclude artifact. Response to transients can best be determined using accurately known profiles which include both transients and plateaus.

Materials and Methods

We began to study pulse oximetry inducing transient desaturation with a few breaths of N_2, or by rebreathing into a 5-l bag filled with N_2. These methods failed to adequately assess accuracy, both because the actual desaturation could not be measured, and because transient responses could not distinguish between plateau errors and errors due to varying methods of responding to transients between instruments. See table 1 for terminology.

We chose to induce a 30–60-s step hypoxic plateau between 40 and 70% saturation in normal volunteers, long enough to record the steady-state responses of the oximeters and to withdraw an arterial sample, but brief enough to avoid loss of consciousness or other effects of profound hypoxia. In order to obtain constant desaturation, the inspired O_2 concentration must be adjusted upward breath by breath after initial N_2 inhalation. This requires a method for immediate determination of saturation. The key to the method was on-line computation of arterial saturation by breath-to-breath analysis of end-tidal P_{O_2} and P_{CO_2}, with display for the operator who adjusts the fresh gas mixture in response to the data from each breath.

These studies were approved by the U.C.S.F. Committee on Human Research. Normotensive healthy volunteers of both sexes aged 29.1 ± 8.6 yr (range 18–64 yr) were positioned on an operating room table with their trunk and head elevated 30°. A radial arterial 20- or 22-gauge catheter was inserted and an ECG monitor was attached. The ears were massaged before placing probes. Subject performed the test 6–7 times with 2–5-min recovery periods after each test. Four sequential series of studies consisted of 10, 9, 6, and 10 subjects as various manufacturers provided new or revised instruments. These instruments and the abbreviations used herein for them were: NE = Nellcor N100®, OH = Ohmeda 3700®, NO = Novametrix 500®, CR = Criticare CSI 501+® and also 502® in series (3), PC = PhysioControl Lifestat 1600®, and MQ = Marquest/Minolta PulseOx 7®. Ohmeda, Criticare, and PhysioControl provided both ear and finger probes. The Nellcor disposable adult finger probes were used both on finger and ear-lobe. The Novametrix finger probe was used both on finger and ear in series 1, because, during these tests, the manufacturer was unable to supply ear probes. Only finger responses were evaluated in series 4. Each instrument was used with its default averaging time (5–8 s), except NO, which was set to 8 s. The studies were done in a laboratory with fluorescent lights. In most tests, opaque barriers were introduced between adjacent probes to test for interference, but none could be demonstrated. There were no other special efforts to optically shield ears and fingers.

The subject breathed through a mouthpiece and wore a nose clip. A three-way stopcock switched the mouthpiece from room air to a 5-liter open-ended 6-cm diameter tubing reservoir with fresh gas inlet near the mouthpiece. Gas mixtures were prepared using flow meters, supplying about 3% CO_2 in N_2 at 30 l/min. A Perkin-Elmer® 1100 mass spectrometer sampled gas from the mouthpiece for O_2 and CO_2 determination.

The subject was instructed to exhale deeply, at which point the stopcock was turned into the reservoir tubing and he/she was asked to take five rapid and deep breaths, and then continue to breathe rapidly. The operator watched the saturation, computed breath by breath and displayed every 2 s, and began to add air to the mixture without changing N_2 and CO_2 flows, to maintain a plateau of desaturation. After a plateau of at least 15 s had been observed, an arterial blood sample was drawn into a 2-ml heparinized syringe and the stopcock was opened to room air. Blood oxygen saturation was immediately determined in a Radiometer OSM-3® multi-wavelength bench oximeter, and blood gas analysis was then done using a Corning 178® blood gas analyzer.

Pulse oximeter outputs and O_2 and CO_2 from the mass spectrometer were recorded through a 16 channel A/D converter in a DEC PDP11/44® computer, together with the breath-by-breath computation of saturation for later analysis using a Tektronix 4105® plotting CRT and a Nicolet Zeta 8® pen plotter. Arterial saturation values were entered into the computer plotting program.

The on-line program for saturation calculation used a peak detector program to identify end-tidal CO_2 and

the simultaneous O_2. Arterial pH was estimated from $P_{ET_{CO_2}}$.

$$p\text{H} = 7.42 + 0.66 \times \log (40/P_{ET_{CO_2}}),$$

assuming a 0.02 rise in pH due to 40% reduction of oxygen saturation at 12 g Hb, which increases base by 1.5 mM. 0.66 is the *in vitro* buffer slope of blood, $\Delta p\text{H}/\Delta P_{CO_2}$. Although the program permits entry of abnormal P50, body temperature and base excess, default values of 26.6, 37° C and 0 were used in this study. P_{O_2} was corrected to pH = 7.4 by the modified Bohr correction:[13]

$$Pa_{O_2} = P_{ET_{O_2}} \times e^{\{1.1(p\text{H}-7.4)\}}$$

A standard O_2 dissociation curve equation[13] was then used to compute arterial oxygen saturation, Sc_{O_2}, from Pa_{O_2}.

$$Sc_{O_2} = \frac{100}{1 + \frac{23400}{(Pa_{O_2})^3 + (150\ Pa_{O_2})}}$$

This saturation value was displayed numerically every 2 s on a terminal in front of the operator (always KHN), who added air as needed to hold a constant saturation. Figure 1 illustrates the continuously computed Sc_{O_2} (heavy line) and the measured blood values, together with three ear and four finger probe responses in a typical test.

We assumed that blood transit time from lung to radial artery was 6 s. Because it is generally not possible to maintain a perfectly constant plateau of saturation, any change of the computed saturation Sc_{O_2} from 6 s before the time of blood sampling to the last 6 s of the Sc_{O_2} plateau was added to the measured Hb_{O_2}% before it was compared with each pulse oximeter reading. This plateau drift correction averaged +0.89 ± 2.93 % SD saturation (n = 113). On each recorded response, marks were placed at three times: at 50% of the downward transient, at the end of the plateau, and at 50% of the upward transient. These time marks permitted computation of both the time lags for desaturation and resaturation, and of the average reading of the calculated saturation and of each pulse oximeter during the last 6 s of each plateau by both a Tektronix 4105 with a screen cursor, and by a computer program. The difference or lag thus includes both lung-to-tissue transit time and instrumental response time.

Each of the responses was also plotted in order to observe the shape of the response, and to visualize the differences between the calculated and observed responses of each instrument. Statistical analysis was performed using Minitab®. Student's t test probability of $P < .05$ was assumed significant.

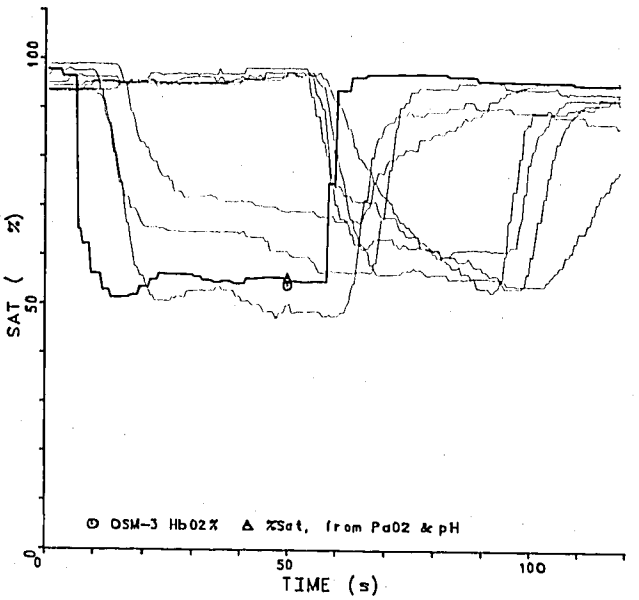

FIG. 1. A typical step hypoxia test. Calculated saturation is the heavy line, Sc_{O_2}. The symbols mark the measured blood Hb_{O_2}% (circle) and Sa_{O_2}% calculated from pH and P_{O_2} (triangle). Simultaneous responses of three ear and four finger probes are shown. Finger response lag in this subject was about 50 s, while ear lag was about 10 s.

Results

Each subject underwent six to seven sequential hypoxic tests after at least a 2-min recovery period breathing air. None of the subjects was distressed by the tests, none showed ECG abnormalities, and none felt that they had lost consciousness. Several subjects noted tingling and numbness of fingers or toes during hypoxia. In several subjects, we noted fluttering eyelids at the end of the hypoxic period. No post-test headaches were reported, but several subjects felt dizzy for a few minutes.

Data are included from four series of tests. Series 1 (60 tests, 10 subjects) evaluated NE, OH, NO, and CR. Series 2 (54 tests in 9 subjects) examined experimental revisions of NE, CR, and OH, the unmodified NO, plus

TABLE 2. End-plateau Blood and Calculated Oxygen Saturation. Values Obtained in Subsequent Series Were Not Significantly Different from Those of Series 1. Number in Parentheses Indicates Series

	N	Mean	SD	SE	Max	Min
(1) Hb_{O_2}%	59	55.5	7.5	1.0	83.3	43.6
(2) Hb_{O_2}%	54	56.4	7.5	1.0	81.0	43.7
(3) Hb_{O_2}%	36	55.2	4.6	0.8	63.8	46.9
(4) Hb_{O_2}%	60	55.3	4.7	0.6	64.2	45.2
(1) Sc_{O_2}%-Hb_{O_2}%	58	0.2	3.6	0.5	8.9	−8.0 n.s.
(1) Sa_{O_2}%-Hb_{O_2}%	58	0.4	2.8	0.4	4.8	−6.3 n.s.
(1) Pa_{CO_2}, mmHg	58	30.3	2.6	0.3	36.0	25.1
(1) BE @ 55% Sa_{O_2}	58	2.5	1.3	0.2	5.1	−0.8

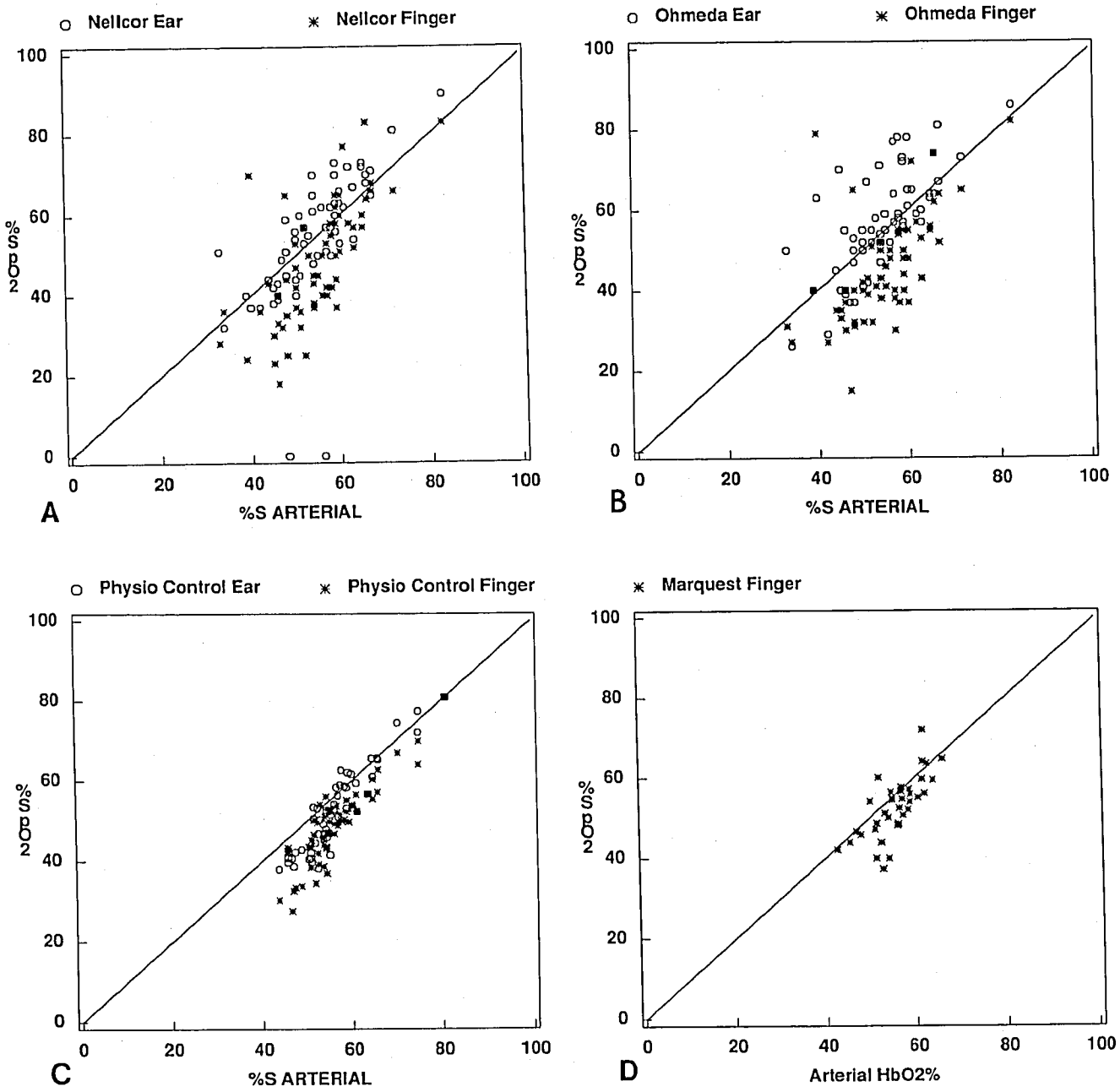

FIG. 2. Relationship of end plateau pulse oximeter saturation values plotted against end plateau arterial blood oxyhemoglobin percentage for six instruments. *A–D* are NE, OH, PC, and MQ. *F* and *H* illustrate the improved performance of CR and NO in series 3 and 4 compared with their series 1 responses shown in *E* and *G*. CSI = Criticare®.

PC. Series 3 (36 tests, 6 subjects) assessed the new commercial versions of CR 501+ and 502 (February, 1987) and MQ. Series 4 (60 tests in 10 subjects) tested experimental versions of OH and NE (not reported here) and the revised NO (as marketed since February 1, 1987), using two finger probes. Series 1 control resting Pa_{CO_2} was 40.5 ± 2.6 mmHg (SD), with a range from 36.4–43.8. HbCO and HbMet were less than 1% in all samples. The mean plateau Hb_{O_2}% was about 55% in each series (table 2). The errors of the Sa_{O_2}% calculated from arterial *p*H and P_{CO_2}, and Sc_{O_2}% calculated from mass spectrometer end-tidal P_{O_2} and P_{CO_2}, were not significant (table 2). Pa_{CO_2} at end-plateau (series 1) was 30.3 ± 2.6 mmHg, and BE was 2.5 ± 1.3 mM. These values were not significantly different in series 2, 3, and 4.

Figure 1 illustrates simultaneous responses of three ear and four finger probes. The heavy line is the

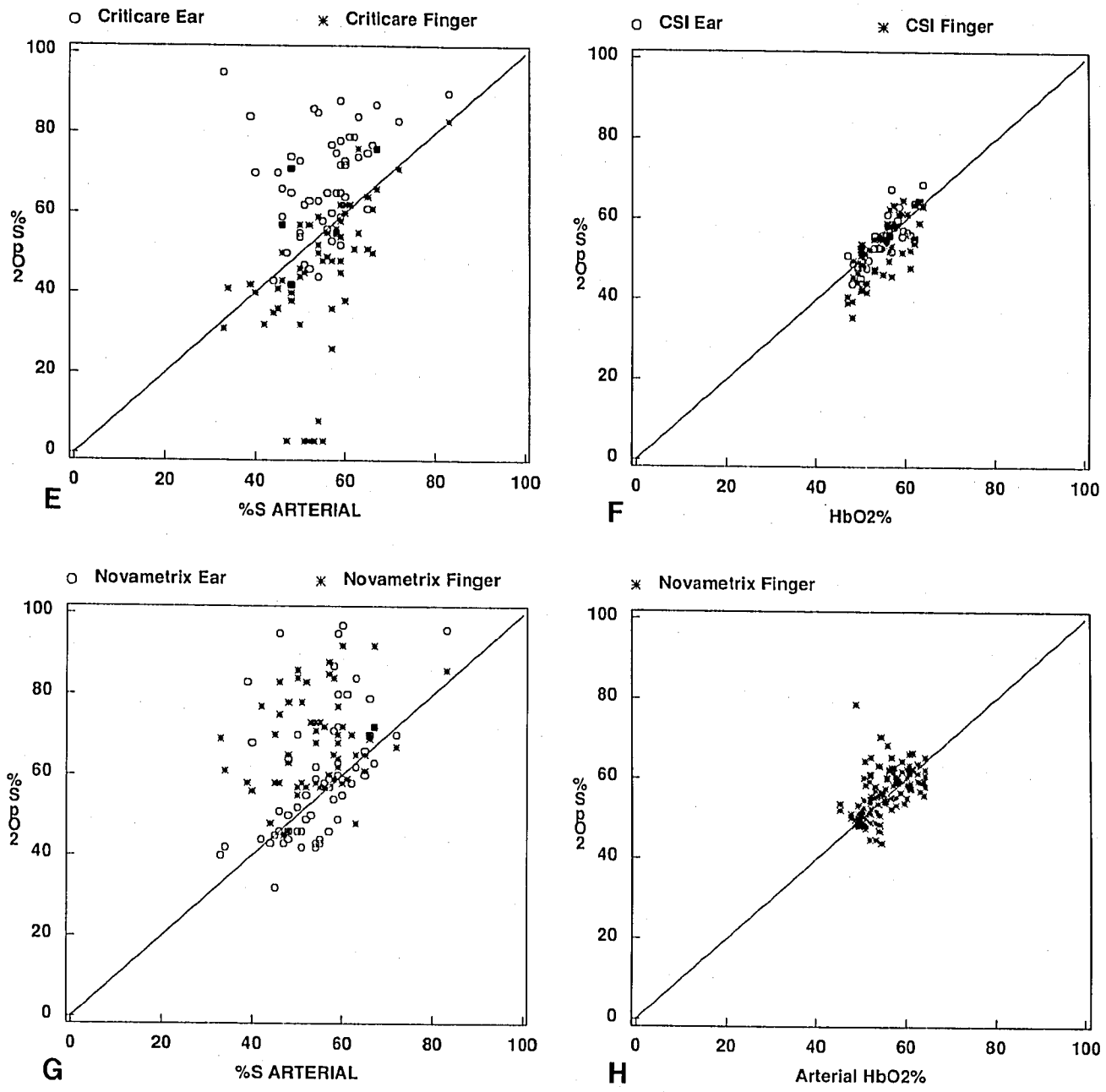

FIG. 2-*Continued.*

breath-by-breath computed saturation. In some subjects, such as this one, we observed a pronounced delay of the desaturation responses from the fingers due to vasoconstriction. The resaturation responses from these fingers were less delayed, suggesting that vasodilation occurred immediately when the hypoxic blood reached the finger. Despite this delay and, occasionally, lack of a plateau, on average, the finger saturation data were lower than ear data.

Figure 2 presents the pulse oximeter end-plateau Sp_{O_2}% readings plotted against the corrected end-plateau Hb_{O_2}% for six instruments. Figure 2F and H illustrate the improvement seen after modifications by CR and NO.

The mean and standard deviation of errors at the end of the hypoxic plateau are presented in table 3. The ear probe responses were generally more accurate than the finger probes. The differences between finger and ear saturation values (table 4) were significant for most oximeter pairs.

Table 5 presents the delays for five instruments (series 1 except CR = series 3) measured from the time

TABLE 3. Pulse Oximeter Error (%Sp_{O_2}-%Hb_{O_2} at End-plateau)

Manufacturer	Version	Series	N	Ear	Finger
NE	N100	1	60, 60	−0.4 ± 11.7	−6.6 ± 10.8
OH	3700	1	60, 60	2.4 ± 8.8	−9.0 ± 10.4
NO	2.2	1	60, 60	4.8 ± 13.9	13.1 ± 12.7
NO	3.3	4	0, 120	—	1.1 ± 5.4
CR	.27	1	55, 60	12.0 ± 13.3	−8.1 ± 16.0
CR	.28	3	36, 60	0.0 ± 3.4	−1.9 ± 4.8
PC	1600	2	54, 73	−4.3 ± 4.3	−7.9 ± 5.8
MQ	7	3	0, 36	—	−2.9 ± 5.2

TABLE 4. Mean of ΔSp_{O_2} (Finger-ear).

	NE	OH	NO	CR	CR (3)	PC
N	60	60	60	55	36	54
Mean	−6.2	−11.3	8.3	−20.6	−1.7	−3.6
SD	14.9	12.7	18.4	18.8	5.1	5.3
SE	1.9	1.6	2.4	2.5	0.8	0.72
Max	40.0	16.0	38.0	10.0	11.9	8.3
Min	−34.0	−47.0	−39.0	−83.0	−7.5	−13.1
P	0.002	0.000	0.001	0.000	0.18	0.000

of half of the step response of the end-tidal Sc_{O_2} to half of the response (whatever its magnitude) of each instrument. These include circulation times and instrumental delay and averaging time.

Several individual instrumental characteristics were observed which were not reflected in the end-plateau statistical data. In about half of the tests NE over-responded to hypoxic steps, such that, early in its response, the finger averaged 10.2% low (n = 24) and the ear averaged 3.4% low (n = 27) in those studies which showed such undershoots. In a few instances, NE fell to less than 15% Sp_{O_2} initially, but usually recovered by the end of the plateau. Occasionally, its recorded Sp_{O_2} fell suddenly to zero during desaturation, or the signal remained unchanged as if not responding for as long as 15 s, but it always recovered promptly upon resaturation.

OH showed a brief reversal during desaturation or "hiccup" in 78 of 120 tests, usually at about 85% saturation, with a typical upward deflection of 2–5% (maximum 15%) usually lasting 4–10 s. In studies where saturation fell as low as 50%, a second hiccup was sometimes seen around 55% Sp_{O_2}.

In series 1, CR exhibited large slow oscillations in 27 of 115 responses during severe hypoxia, both in ear and finger probes and large errors (table 3). The typical oscillation had an 8-s period and a ±5% amplitude, with maximum amplitudes, peak to peak of 20% Sp_{O_2}. In six instances, finger saturation read zero at the end of the plateau, but recovered upon resaturation. The ear probe response severely overestimated the saturation, with a mean error of ±12%, whereas the finger error averaged −8.1%. In response to these results, the instrument was modified by the manufacturer, and retested in series 3. The oscillations were eliminated, providing the excellent responses shown in table 3. Only this modified version has been sold since February, 1987.

NO version 2.2 showed great variability both in accuracy and in time delay (tables 3, 5). In six of 120 studies, the Sp_{O_2} dropped abruptly to zero, usually briefly, during the desaturation period, but, in each case, tracking recovered before the end of the plateau. A revised software program (version 3.3, February, 1987) showed excellent responses (fig. 2H, table 3).

TABLE 5. Half Response Time, Seconds Measured Between the Computed Desaturation Transient Half Point and the Oximeter Half Points

	Ear		Finger	
	Desat	Resat	Desat	Resat
NE	9.6 ± 4.3	8.6 ± 4.4	24.0 ± 14.4	19.4 ± 6.6
OH	13.2 ± 4.3	9.8 ± 3.2	31.3 ± 11.8	19.7 ± 5.3
PC	10.2 ± 3.0	7.1 ± 4.0	34.1 ± 14.6	20.0 ± 6.4
NO	19.8 ± 12.9	12.7 ± 9.4	35.1 ± 9.4	30.0 ± 9.1
CR	10.3 ± 2.8	10.4 ± 4.7	33.9 ± 15.9	25.8 ± 8.6

Discussion

Why should one be concerned about errors at these very low saturations? If one needs only a warning that saturation is falling, the only requirement would be a faithful trend display. Unfortunately, there were many instances in which even the downward transient was not

displayed, or was inadequately represented. Instruments continued to show nearly normal saturation values when real saturation was 40–70%. Some instruments routinely failed to indicate saturation at all during the critical period of falling saturation. In the wide variety of patients where these instruments are used, there may be many with profound hypoxia in whom accuracy is important. Furthermore, in the premature infant, the accuracy of the instrument is important, since it may be used to hold a constant level of desaturation, between 80 and 90%. Since errors at low levels are due in part to lack of suitable calibration data, these tests permit improvements to be made by manufacturers.

In several instruments, these errors were much larger than errors reported at Sa_{O_2} values in the 80–100% range. The brevity of the tests probably contribute to this. In many instances, particularly with finger probes, outputs had not shown an adequate plateau when resaturation occurred, even though the actual plateau had lasted more than 20 s. Vasoconstriction in the arm and hand appears to have caused most of this, and, when the hypoxic blood reached the fingers, blood flow increased. Thus, the lag was greater for downward than for upward transients, sometimes very much more, shortening the plateaus. In view of this, one might expect to find evidence that the finger minima did not fall as far as either the ear or the actual blood values. On the contrary, the mean desaturation seen by the finger probes in all but one instrument was lower than ear or arterial blood values. We conclude that these plateaus were long enough to obtain meaningful steady-state data in all subjects with ear probes, and in most with finger probes. Only one instrument (NE) showed undershooting during rapid desaturation that might, in part, account for its lower average reading on fingers than on ears.

The on-line estimation of arterial saturation from end-tidal P_{O_2} and P_{CO_2} was found to predict Hb_{O_2}% with an accuracy of -0.7 ± 2.9%, despite the many assumptions, which include: 1) alveolar to arterial P_{O_2} and P_{CO_2} gradients = 0; 2) P50 = 26.6; 3) base excess = 0; 4) body temperature = 37° C; 5) Haldane and Bohr effects to be normal in all subjects; and 6) $Sa_{O_2} = Hb_{O_2}$%. Assumption 1), of no gradient, is appropriate because the sudden reduction of alveolar P_{O_2} to near venous P_{O_2} eliminates the gradient, and hyperventilation under these circumstances equilibrates venous, arterial, and alveolar P_{O_2}. Abnormal P50 values would appear as differences between Hb_{O_2} measured by the OSM-3 oximeter and Sa_{O_2} computed from arterial *pH* and P_{O_2} by the blood gas analyzer. Temperature was determined to be between 36 and 37° C, but no correction was made for these small differences.

In one subject, a 48-yr-old male, the difference between the measured and computed saturations increased from zero to 8%, suggesting an increasing alveolar to arterial P_{O_2} gradient, probably due to small airway closure induced by the forced expiration we requested before each run. At the end of six tests, his air breathing saturation was 90–92%, but rose to 97% with several deep sustained inspirations.

A surprising aspect of the study was the large variability in the Sp_{O_2} responses in many instruments, not only between subjects, but within subjects on sequential tests which produced comparable hypoxic square waves. One might ask whether, in the second and third series, there might have been changes in methods which could have artifactually resulted in the superior performance of PC and the revised CR. For example, the mean hypoxia time was increased from 42.5 ± 7.2 to 54.3 ± 4.6 s to obtain a longer plateau in series 2, 3, and 4. Our experience in adjusting gas mixtures improved with time, providing flatter plateaus. In order to assess the equivalence of the subsequent series, we also recorded outputs from the series 1 instruments together with PC in series 2, and compared PC and CR in series 3, and NE, OH, CR, and NO in series 4. Modifications were made by NE and OH between series, as a result of the series 1 data. CR was partially modified in series 2, and NO made no changes in series 2 but changed for series 4. We found no significant improvement in series 2–4 in performance of the instruments used in series 1. Series 3 also included tests of PC which were not different than series 2. In series 4, NE and OH were not significantly more accurate or precise than in series 1.

Response times were longer for step desaturation than for step resaturation. It is probable that blood flow to both ear and finger increases when hypoxic blood reaches the tissue. The differences of delay time on the ear are assumed to reflect small instrumental data handling differences, where the finger delays have a large transit time component which may be variable.

In conclusion, six pulse oximeters were tested using suddenly induced profound hypoxic plateaus. Four initially showed mean end-plateau errors greater than 6% and standard deviations greater than 10% with finger probes. Responses were more accurate on the ear than on the finger. Two manufacturers modified and significantly improved function of their instrument.

The authors thank Radiometer Company of Copenhagen for providing the new multiwavelength OSM-3® blood oximeter for these studies, and each of the manufacturers for providing the pulse oximeters and for support of the studies. The authors also appreciate the expert assistance of C. A. Richardson and W. G. Young in programming the on-line saturation calculation, G. Moskowitz in data analysis and A. Cordano in manuscript preparation.

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Addendum

The following table provides data describing the response of eight oximeters as of June 1, 1987. Note that, in addition to different results with the Physiocontrol 1600, the Criticare .28®, and the Ohmeda 3700®, two additional oximeters—Datex and Nellcor N-200—have been evaluated.

Manufacturer	Version	N Ear, Finger	$\%Sp_{O_2} - \%Hb_{O_2}$ Ear	Finger
NE	N-100	60, 60	−0.4 ± 11.7	−6.6 ± 10.8
NE	N-200*	58, 60	−2.4 ± 8.7	−4.5 ± 8.2
OH	3700	60, 60	2.4 ± 8.8	2.7 ± 5.8
CR	.28	60, 60	4.4 ± 4.4	1.4 ± 5.9
PC	1600	57, 60	2.9 ± 4.3	0.0 ± 3.5
NO	3.3	0, 120	—	1.1 ± 5.4
MQ	7	0, 36	—	−2.9 ± 5.2
Datex		60, 59	−4.9 ± 5.7	−1.6 ± 5.4

* With ECG synchronization.