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Pulse Oximeter Failure Thresholds in Hypotension and Vasoconstriction

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The degree of systolic hypotension causing failure and recovery were tested simultaneously with three oximeters (CSI 504US, Nellcor N-200, and Ohmeda 3740) in nine normal male volunteers. Perfusion of the right hand was slowly reduced and restored by 1) elevation of the hand plus systemic hypotension with nitroprusside if needed (EL); 2) clamp compression of the brachial artery (CL); 3) brachial cuff inflation (CU); and 4) intraarterial norepinephrine (NE). With EL, pulse pressure was normal whereas right radial arterial systolic pressure (SP) was 25.3 ± 12.4 mmHg at failure and 34.1 ± 13.3 at recovery (mean of three oximeters, n = 189). With CL, pulse pressure fell more than did mean pressure, and failure occurred at 37.3 ± 9.8 and recovery at 46.8 ± 17.6 mmHg, n = 84. With CL, threshold of function, defined as the average of failure SP and recovery SP, was 47.1 ± 13.5, n = 41 for Nellcor, higher than for either CSI (38.7 ± 14.5, n = 17) or Ohmeda (36.0 ± 3.4, n = 26) ($P < 0.05$). With EL, no difference among instruments was found (mean 29.7 ± 12.8, n = 189). Threshold was 58.2 ± 8.4, n = 17 with CU if cuff inflation was slow (filling veins), but recovery was similar to EL after rapid cuff occlusion. With NE, SP threshold was increased to 58.3 ± 21.0 with CL but only to 41.0 ± 13.8 with EL. Hypoxia to $Sa_{O_2} \simeq 70\%$ reduced SP thresholds with CSI to 25.1 ± 12.0 and with Ohmeda to 21.0 ± 7.6 with EL, but had no effect on the Nellcor, nor on any oximeter with CL. With systolic pressure just above failure, flow measured by finger plethysmograph was usually zero and detection of an induced hypoxic transient was delayed in one test as much as 6 min. In normoxic subjects, Sp_{O_2} decreased at threshold to 88.8 ± 11.5%, n = 210, suggesting that arterial O_2 was lost to tissue by pulsatile movement in and out of capillaries, or by transarterial diffusion. This suggests that failure of pulse oximeters on fingers occurs at pressures too low for adequate tissue perfusion except when vasoconstriction is present. There are minor threshold differences between instruments, but efforts to increase sensitivity may not be justified in view of the local arterial desaturation evident at current thresholds during normoxia. (Key Words: Brachial arterial occlusion. Blood pressure: hypotension, vasoconstriction. Equipment, oximeter. Measurement techniques, pulse oximetry. Measurement error monitoring: noninvasive blood pressure.)

THE MOST IMPORTANT limitation of pulse oximetry has been failure to detect pulse and saturation (Sp_{O_2}) when blood pressure or pulse pressure is low or when peripheral vasoconstriction occurs. Direct tests have suggested that pulse oximeters are surprisingly sensitive at detecting pulse signals in experimental arterial occlusion. Lawson *et al.*[1] noted that pulse oximeter signals disappear when an arm cuff was inflated to approximately 96% of systolic pressure and reappeared during deflation at 93%. In their study, cutaneous flow by a laser Doppler device was reduced to 8% of control at failure, and was 4% of control at recovery. They concluded that the presence of pulse oximeter signals should not be used to indicate adequate circulation. Because they did not measure actual arterial pressure distal to the cuff, they made no attempt to interpret their results in terms of hypotensive limits of pulse oximeters. In Lawson and co-workers' study, because inflation occurred slowly, the distending pressure in the microcirculation was increased, whereas in shock, hypotension, and vasoconstriction, the vascular bed collapses.

Pulse oximeters contain computers with preprogrammed lower limits of pulsatile signal at which they cease to report saturation. These limits are based on the ratio of the pulsatile and steady components of the detected light. They differ among manufacturers, but are not specified in the user's manuals. In the current study, the roles of systolic pressure, pulse pressure, and flow in determining the functional thresholds of three pulse oximeters were tested.

Materials and Methods

The subjects were nine volunteer males of average age 24.3 ± 3.1 yr, all healthy nonsmokers taking no medications. Approval by the University of California, San Francisco Committee on Human Research was obtained, and each subject provided informed consent. In order to achieve gravitational hypotensive failure by elevation of the hand and arm, tall subjects (with long arms) were chosen (all were over 175 cm). Nondisposable probes of Criticare Systems, Inc. (CSI) 504US, Ohmeda 3740, and Nellcor N-200 oximeters were rotated between right hand digits 2, 4, and 5. The three pulse oximeters were set to their default averaging times (5–8 s). The CSI and Nellcor units were coupled to the subject's ECG. A CSI 501+ was mounted on the left (control) hand index finger. After confirmation of ulnar artery patency by Allen's test, the right radial artery was cannulated with a 1.5-inch 22-G

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wire-guided Insyte-A® catheter coupled to a strain gauge mounted at the level of the hand. Radial arterial systolic pressure at failure and recovery was read with a plotting terminal.

Blood flow into the distal two phalanges of the right third digit was estimated by the plethysmographic method[2] from the initial rate of finger volume increase after sudden venous outflow occlusion. The barrel of a 20-ml syringe was sealed with grease to the proximal interphalangeal joint and connected through a 270-ml glass reservoir to a Validyne DP45 pressure transducer and CD15 carrier demodulator. A 2-cm wide Dynamap® infant blood pressure cuff mounted on the proximal phalanx was inflated every 20 s to 30 mmHg within 0.2 s for 10 s by an electrically timed three-way solenoid valve. During cuff deflation, another solenoid opened the plethysmograph to ambient through a high-resistance (26-G needle, time constant 17 s) to compensate for drift. The finger tissue volume within the plethysmograph was approximately 10 ml, making the total plethysmograph gas volume = 280 ml. The signal was calibrated to read distal finger volume change ΔV by injection of 100 μl air into the closed system (a pressure increase of 0.27 mmHg). Blood flow was subsequently measured as initial slope, $\Delta V/\Delta t$, after each sudden occlusion.

Four methods of inducing oximeter failure were used: arterial compression (to obtain low systolic pressure and low pulse pressure); hand elevation (to lower systolic pressure while sustaining pulse pressure); cuff occlusion (for comparison with the study by Lawson *et al.*[1]); and intraarterial norepinephrine (to induce vasoconstriction). With each method and with each oximeter, systolic blood pressures determined at failure and recovery points were averaged to compute a functional threshold.

1) *Controlled gradual compression of brachial artery (CL):* A 7.6-cm (3-inch) gap carpenter's screw clamp fitted with smooth, rounded 3.8-cm-diameter pressure disks was mounted over the brachial artery just above the antecubital fossa to permit graded stable occlusion. The back of the clamp was centered over the lower humerus. Distal pressure at failure and recovery of each oximeter were determined three times, with rotation of the probes to average interdigit differences.
2) *Elevation (EL):* The right arm and hand were supported by 2-in-wide cloth adhesive tape strapped lengthwise on the radial and ulnar sides of the forearm. These tapes permitted passive elevation of the hand and arm with the subject in left lateral recumbent position. By using only subjects over 175 cm height we were able to reduce pressure in the fingers by 58–64 mmHg (77–86 cm of arm length from midline to fingers). In three subjects, in order to reduce hand arterial pressure to the point of pulse oximeter failure, it also was necessary to reduce systemic systolic arterial pressure to 75–85 mmHg systolic with an iv infusion of sodium nitroprusside at 100–400 $\mu g \cdot min^{-1}$. Failure and recovery pressures were measured three times to permit each probe to be tested on each finger. The height of the hand was continuously recorded with a strain gauge mounted at body midline level connected to a water-filled catheter with an open end attached to the hand.

Tests with the two methods above were repeated in eight subjects during stable hypoxia. With subjects breathing through a mouthpiece, arterial oxygen saturation was rapidly reduced to 70 ± 5% and held constant by adjusting inspired O_2. This was facilitated by an online display of breath by breath saturation Sc_{O_2} computed from end-tidal P_{O_2} and P_{CO_2}.[3,4] The resulting Sp_{O_2} was recorded with an additional pulse oximeter (CSI 501+) on the left index finger.

3) *Arm cuff inflation (CU):* In five subjects a brachial arm cuff was rapidly inflated to occlusion. It then was slowly deflated to determine the radial arterial pressure at oximeter recovery. The cuff then was held at approximately diastolic pressure, allowing veins to fill for several minutes; the pressures at failure and recovery were then redetermined by slow cuff inflation and deflation.
4) *Intra-arterial norepinephrine (NE):* Finger ischemia was transiently induced by injection into the radial artery of 0.1–0.8 μg 1:1,000,000 dilution of norepinephrine in saline followed by approximately 1 ml saline flush with temporary occlusion of the ulnar artery. The radial systolic pressure was then reduced by either brachial arterial compression or elevation, as noted.

A 2-cm-wide Dynamap infant blood pressure cuff was mounted on the base of the index finger proximal to the oximeter from which the pulse waveform was recorded (CSI). As an estimate of arterial systolic pressure in that finger, the cuff was slowly inflated with a constant air flow until disappearance of the pulse waveform, as illustrated in figure 1. This test was done under each of the four ischemia conditions in each subject in order to compare cuff pressure with radial systolic pressure as part of another study to be reported separately.

The data were stored on a DEC PDP11/44 minicomputer and analyzed with a Tektronix 4105 plotting oscilloscope with direct transfer of cursor position to VAX files. The level of significance, assessed by analysis of variance (ANOVA), was considered to be $P < 0.05$.

Results

A radial arterial catheter might partially block distal flow, and result in an incorrect estimation of the lower limits of pressure referred to the radial arterial cannula. The pressure in the 2-cm-wide finger cuff at the time of disappearance of the pulse waveform was 9.9 ± 12.4

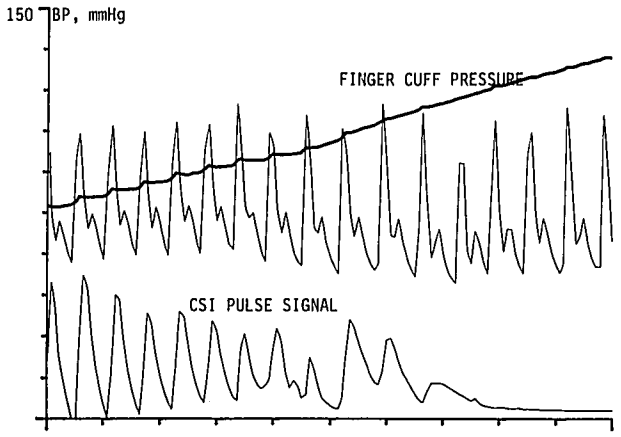

FIG. 1. Pressure rising slowly in a finger cuff occludes the distal oximeter waveform when cuff pressure exceeds radial arterial systolic pressure. This provides an indirect method of estimating finger arterial systolic pressure.

mmHg above the simultaneously recorded radial arterial systolic pressure (n = 149). This bias was independent of the level of pressure: P(ox) = 10.6 + 0.99 (BPsys), $S_{y.x}$ = 12.4, r = 0.918, suggesting that finger circulation was not impaired by either the radial catheter or the mechanical arm supports.

Table 1 indicates systolic pressures at failure and recovery. The pressure at failure was lower than that at recovery with EL and CL by an average of 7.8 mmHg, n = 405 (CSI = 7.0 ± 12.6; Nellcor = 10.6 ± 12.8; Ohmeda = 8.2 ± 9.7), because oximeters are programmed to delay in both failing and recovering. Paradoxically, after venous engorgement by slow brachial cuff inflation, pressure averaged 8.5 mmHg higher at failure than at recovery. The average of the systolic pressures at failure and recovery is defined as threshold.

Table 2 presents the mean and standard deviation of threshold pressure of the three oximeters when perfusion was reduced by the four methods. Differences in thresholds between fingers were not significant. The following conclusions from tables 1 and 2 were significant ($P < 0.05$ by ANOVA).

1) Threshold was lower with elevation (high pulse pressure) than with clamping (low pulse pressure).
2) The threshold with arterial compression by clamp was higher for Nellcor than for CSI or Ohmeda.
3) Norepinephrine vasoconstriction increased threshold except during hypoxia.
4) With elevation, hypoxia lowered thresholds with and without vasoconstriction (except for Nellcor without norepinephrine).
5) Threshold was elevated when occlusion was produced by slow brachial cuff inflation.

Table 3 illustrates that a decrease of oxygen saturation occurred at the hypotensive threshold, both at failure and recovery. The difference between failure and recovery saturations was significant only for slow brachial cuff inflation ($P < 0.002$, paired t-test). The differences between oximeters were not significant (CSI 87.7 ± 11.6; Nellcor 91.4 ± 14.0; Ohmeda 86.5 ± 6.5).

Figure 2 illustrates flow measurement from three plethysmograms. At zero flow, an initial volume displacement artifact was visible. At high flow, the veins filled to capacity in one or two pulses.

Systolic and diastolic hypotension with undiminished pulse pressure resulted from elevation, as illustrated in figure 3. Two oximeters continued to display pulses with the correct heart rate while plethysmographic finger blood flow was zero. Note the gradual fall of Sp_{O_2} (the subject was normoxic) suggesting that O_2 was lost from the stagnant but pulsing blood in arteries to tissue. With brachial artery clamping, pulse pressure was reduced proportionally more than systolic pressure, as shown in figure 4. Two of the oximeters default to zero at the right end of figure 4.

In several subjects, complete clamp occlusion of the brachial artery did not terminate pulse oximeter signal

TABLE 1. Systolic Pressures at Oximeter Failure, Recovery, and Their Average* as Threshold

Mode	O_2	N	Failure		Recovery		Threshold	
			BP_{77}	SD	BP_{77}	SD	BP_{77}	SD
EL	Ox	189	25.3	12.4	34.1	13.3	29.7	12.8
EL	Hy	86	21.3	11.7	28.7	10.5	25.0	11.1
CL	Ox	84	37.3	9.8	46.8	17.6	42.1	13.7
CL	Hy	46	37.7	9.4	46.0	14.7	41.8	12.0
CU_{fast}	Ox	19			31.9	13.9		
CU_{slow}	Ox	17	62.5	9.7	54.0	7.2	58.2	8.4
NE_{CL}	Ox	20	55.1	20.3	61.5	21.7	58.3	21.0
NE_{EL}	Ox	15	36.6	12.7	45.4	14.9	41.0	13.8
NE_{EL}	Hy	16	27.7	6.1	33.1	8.6	30.4	7.3

* Average calculated as mean of three oximeters.
EL = elevation; CL = brachial clamp; CU = brachial cuff; NE = ia norepinephrine; cuff inflation = fast (veins empty, recovery only); slow (veins full); Ox = normoxia; Hy = Hypoxia, Sa_{O_2} = 65–75%.

TABLE 2. Hypotensive Thresholds of Three Pulse Oximeters

Mode	O_2	CSI			Nellcor			Ohmeda		
		BP	SD	N	BP	SD	N	BP	SD	N
EL	Ox	31.0	15.3	70	29.8	11.4	49	28.4	11.8	70
EL	Hy	25.1†	12.0	31	28.1	11.4	26	21.0†	7.6	28
CL	Ox	38.7	14.5	17	47.1*	13.5	41	36.0	3.4	26
CL	Hy	38.9	11.3	12	46.7*	11.1	23	34.8	7.6	11
CU_{fast}	Ox	25.7	4.9	7	40.0	23.1	5	32.3	11.1	9
CU_{slow}	Ox	59.8	7.8	14	60.3	7.4	10	53.8	5.0	10
NE_{CL}	Ox	58.7	23.0	16	59.2	19.1	16	55.7	24.9	8
NE_{EL}	Ox	36.0	3.7	10	47.5*	11.2	8	40.5	15.1	12
NE_{EL}	Hy	30.5†	7.2	16	34.1†	3.8	6	27.9†	7.9	10

* Significant difference from other oximeters.

† Significant difference from normoxia.

detection or cause a decrease in saturation, demonstrating the presence of arterial collaterals. In one case a 6-min delay was recorded before a 4-min induced step of hypoxia was detected in a hypotensive elevated hand (fig. 5).

Discussion

The lower observed pressures at failure than at recovery depended upon the speed and direction of pressure change, because oximeters are programmed to delay defaulting to zero when no pulse is present, and must accumulate several cycles of data before reporting during recovery. The average of these values is not a true threshold value because the rate of change of pressure could not be kept constant; because delay in some instruments depends on an integral number of pulses and on pulse amplitudes; and because delays are not equal in failure and recovery.

Pulse occlusion with a brachial cuff has been reported as a means of estimating systolic pressure,[5,6] and in the study by Lawson *et al.*[1] occlusion occurred with cuff pressure at approximately 95% of systolic arterial pressure. An incautious interpretation of that might suggest that arterial pressure could decrease to 4–7% of normal before failure occurred. Clearly this is not the case. The true systolic arterial pressure in the fingers was not measured in this study. It might be either higher or lower than radial arterial systolic pressure, higher pressures having been shown to result from the transfer of kinetic to pressure energy as blood slows in terminal vessels, and lower distal pressures to result from resistive pressure drops. Finger cuff occlusion pressure generally was higher than radial pressure because a 2-cm cuff may have been too small to transmit its internal pressure to the finger arteries, and perhaps also because of the transfer of kinetic energy to potential (pressure) energy with deceleration.

TABLE 3. Spo_2 in Normoxic Subjects at the Points of Hypotensive Failure and Recovery

Mode	N	Failure		Recovery		Threshold	
		Spo_2	SD	Spo_2	SD	Spo_2	SD
CSI	55	86.5	11.6	88.9	11.6	87.7	11.6
Nellcor	79	92.6	11.2	91.4	12.6	91.4	14.0
Ohmeda	72	87.0	6.1	86.1	6.9	86.5	6.5
CL	77	92.5	6.7	90.6	10.0	91.5	6.9
EL	133	86.6	12.8	87.8	12.5	87.2	10.8
CL + EL	210	88.8	11.3	88.8	11.7	88.8	11.5
CU_{slow}	17	93.8	4.8	81.6*	8.1	87.8	6.4

Sa_{O_2} is assumed to have been 97.5 ± 1.5 because all subjects were breathing air.

* Significant difference from failure ($P < 0.002$, Student's paired t test).

During brachial clamp arterial occlusion, pulse pressure decreased proportionally more than did mean pressure (fig. 4). With elevation of the hand, diastolic and systolic pressure were reduced equally, with pulse pressure remaining nearly constant (fig. 3). In several subjects, diastolic pressure fell below zero at maximum height because the transducer was mounted and zeroed at the level of the base of the fingers approximately 15 cm above the tip of the radial arterial catheter. This higher pulse pressure is presumed responsible for continued oximeter function at lower systolic pressure with gravitational hypotension.

Arterial pressure distal to an occluding brachial cuff depended on the rate of cuff inflation. The pressure at which failure occurred depended upon the venous filling before occlusion and was approximately 25 mmHg higher with slow inflation than with brachial artery clamping. Brachial cuff inflation is not a satisfactory method of testing the hypotensive thresholds of pulse oximeters, although cuff pressure at failure was shown to be close to *upstream* arterial systolic pressure.[1]

Despite the use of ECG signals to indicate timing and rate in both the Nellcor and CSI oximeters, the Ohmeda appeared able to detect a correct heart rate and display pulse signals at lower perfusion pressures than were the other two instruments. This suggests that in-built low pulse signal cutoff levels in the CSI and Nellcor are set at

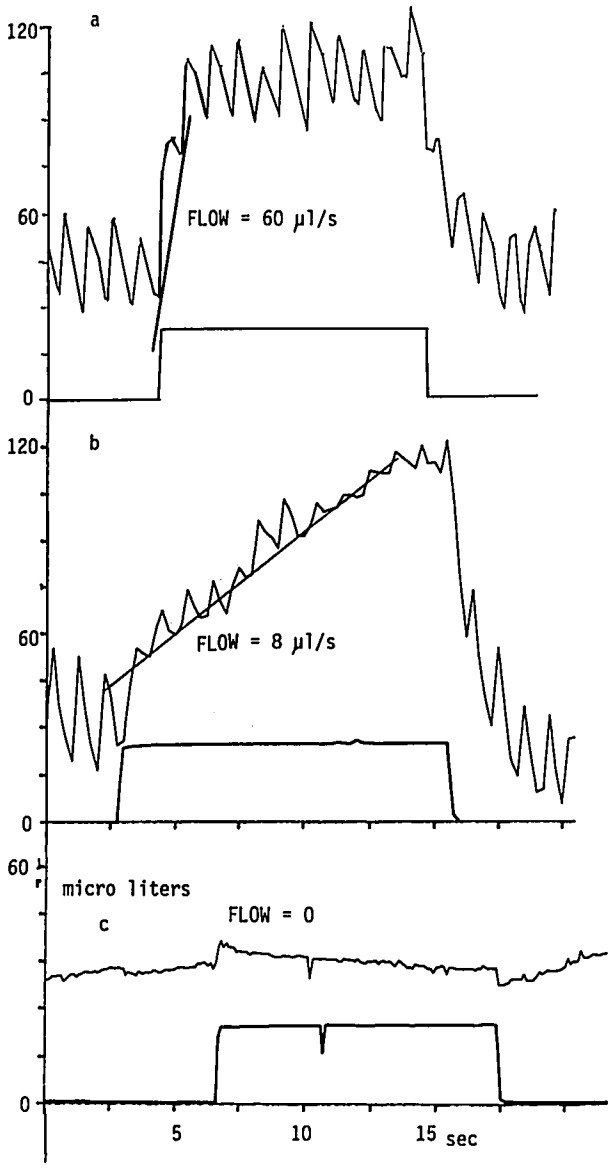

FIG. 2. Three examples of a plethysmographic method of estimating blood flow of a finger. Ordinate is finger volume in µl. When the cuff is suddenly inflated to 30 mmHg, venous outflow ceases while finger volume increases due to continued inflow. (*A*) Flow is so high that the finger distends to cuff pressure within one or two pulses. (*B*) A normal flow. (*C*) The transient artifact of tissue or blood displaced into the distal finger by cuff inflation and made visible when flow is zero. The volume of finger in the plethysmograph was about 10 ml.

higher levels than in the Ohmeda, or that the Ohmeda is more sensitive. However, this difference was not sufficient to yield lower Sp_{O_2} values with Ohmeda at failure and recovery.

Unlike the laser Doppler flowmeter, a finger plethysmograph measures flow deep within the finger in the arteriovenous shunts as well as in skin. With both elevation and clamping, as threshold was approached the blood flow

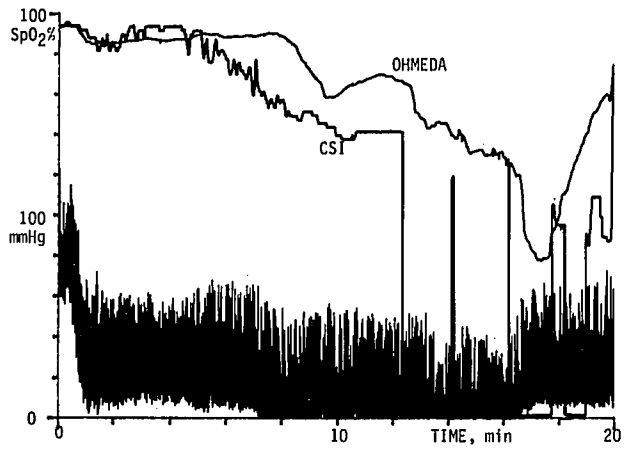

FIG. 3. Hypotension produced by elevation reduced Sp_{O_2} to 70% (CSI, followed by failure) and to 40% (Ohmeda). The gradual progressive fall suggested loss of O_2 from the pulsatile arterial bed to the surrounding tissue when flow is low or absent (or possibly venous pulsation). Note that diastolic pressure reached zero (actually fell to −12 mmHg).

determined with the plethysmographic method decreased to zero while pulse oximeter function continued. At this point, Sp_{O_2} often fell slowly (fig. 3). The gradual and progressive nature of the decrease seen in all oximeters suggests that this is not an artifact of oximetry but is loss of O_2 from the pulsating arterial bed to the surrounding tissue, either by diffusion through arterial walls or by pulsatile movement into and back from capillaries. A gradual Sa_{O_2} decrease was noted also with intraarterial norepinephrine injection. The presence of arterial pulses does not imply that blood is flowing through tissue. Venous pulsation, if present, might also lower Sp_{O_2}, as suggested by Kim *et al.*[7] A decrease in oximeter saturation reading

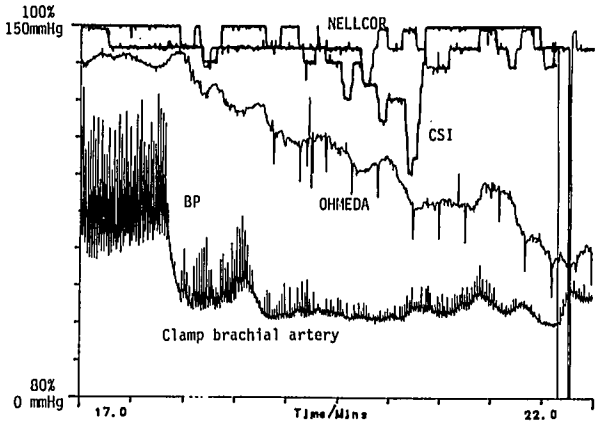

FIG. 4. Pulse pressure falls proportionally more than systolic pressure when the brachial artery is partially occluded by a clamp. Note the slow fall of Sp_{O_2} in one finger, a minor transient fall in a second finger, and no fall in a third.

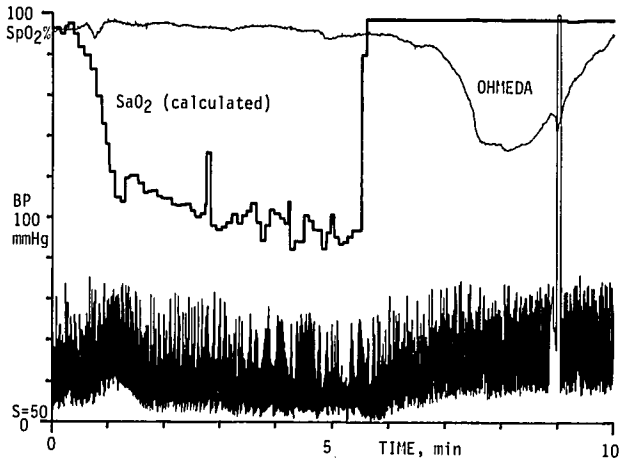

FIG. 5. Example of a delay of 6 min before a 4-min step of hypoxia to 75% is detected by a pulse oximeter during severe gravitational hypotension (55/5 mmHg).

due to vasoconstriction in shock was observed in the following case (reported by JWS):

A 70-yr-old emaciated normotensive female with colonic carcinoma was undergoing bowel and vascular surgery for complications of prior surgery, under opioid and isoflurane–O_2 anesthesia with paralysis and artificial ventilation. Hct was 29%. Esophageal temperature was 34.8° C. A Nellcor N-200 pulse oximeter on an index finger, without ECG link, indicated 97–100% saturation despite severe progressive hypotension to 60 mmHg systolic due to blood loss. Rapid crystalloid administration was begun while awaiting blood, and 10 mg ephedrine was administered iv. While arterial blood pressure slowly rose to 70 mmHg, Sp_{O_2} fell slowly and steadily over the next 5 min to 45%. The oximeter pulse display continued normally with correct heart rate. Ventilation, ECG, and end-tidal P_{CO_2} and isoflurane concentration were unchanged. An arterial blood sample obtained from the radial artery of the hand on which the pulse oximeter was mounted showed Pa_{O_2} = 550 mmHg with P_{CO_2} = 28, pH = 7.38. When blood was administered and the ephedrine induced vasoconstriction wore off, the Sp_{O_2} value returned to >95% over approximately 15 min.

The clinical implications of this study are: 1) pulse oximeters are so sensitive that they may detect pulses when pressure is too low to provide adequate tissue blood flow; b) reduction of Sp_{O_2} may occur due to O_2 consumption by the finger from the pulsing but stagnant arterial blood at low pressure or with vasoconstriction; 3) vasoconstriction significantly increases the hypotensive threshold of oximeters; 4) oximeter failure occurs at higher systolic pressure when pulse pressure is low; and 5) it may be counterproductive to attempt to increase oximeter sensitivity in an effort to obtain saturation data during severe hypotension or vasoconstriction, since the data thus obtained would often severely underestimate arterial oxygen saturation.

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