

seen during surgery with local anesthesia and in the recovery room after general and local anesthesia. Akathisia would generally appear as agitation, not an uncommon event in those settings and could be misdiagnosed as anxiety or, if consciousness is reduced, as confusion. Questions regarding the nature of the agitation, *e.g.*, asking whether it is experienced as physically based or not, would be necessary to detect akathisia. More confirmatory symptoms, such as repeated leg crossing, swinging of one leg, lateral knee movements, sliding of the feet, and rapid walking would have to await the termination of anesthesia.¹¹ Studies in psychiatric populations suggest that the highest risk groups for akathisia are in the 20–40 yr age group, with women being particularly at risk.¹²

To date, clinical studies suggest that lipophilic beta adrenergic blockers, such as propranolol (10–15 mg), are most effective for the treatment of akathisia.^{8,9} Anticholinergic agents have been shown to be effective but with lower efficacy than beta blockers. Finally, benzodiazepines have been reported to be effective, but it is unclear whether this stems from a general sedative effect or a specific effect on akathisia.^{8,9,13} Lorazepam has been reported to be less effective than propranolol.¹³

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The Penumbra Effect: Vasomotion-Dependent Pulse Oximeter Artifact due to Probe Malposition

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Pulse oximetry is a widely used noninvasive method for measuring arterial hemoglobin oxygen saturation. Its principles, clinical applications, performance, and limitations have been extensively reviewed.^{1–3} Despite careful attempts by manufacturers to safeguard against the display of erroneous data, the value displayed by a pulse oximeter (SpO_2) does not always agree with the fractional arterial

hemoglobin oxygen saturation as determined by an *in vitro* oximeter (SO_2). Recognized causes of pulse oximeter artifact include dyshemoglobins, such as carboxyhemoglobin and methemoglobin,^{4–6} vital dyes, such as methylene blue,^{7,8} ambient light,^{9–11} motion,¹² electrocautery,¹³ venous congestion,¹⁴ and nail polish.¹⁵

We present a case which suggests a further cause for pulse oximeter artifact not previously described in the literature.

CASE REPORT

A 54-yr-old, 74-kg white man was scheduled for left total knee arthroplasty (TKA). The history was otherwise unremarkable except for a 35 pack-year history of smoking. Physical examination was otherwise unremarkable.

The patient received 2 mg midazolam iv, 50 μ g fentanyl iv, and breathed 100% oxygen for 3 min after which anesthesia was induced

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with 350 mg thiopental iv, 50 μ g fentanyl iv, and maintained with nitrous oxide and oxygen (3:3) and isoflurane 1.5%. Vecuronium 7 mg iv was also given at induction. The trachea was intubated with an 8.0-mm cuffed endotracheal tube, and vital signs remained stable throughout. Mechanical ventilation of the lungs was begun at 9 breaths/min and tidal volume 800 ml.

The intraoperative course over the next 45 min was uneventful. Delivered gas flows throughout this period were nitrous oxide 1 l/min and oxygen 1 l/min with 1% isoflurane. The oxygen analyzer consistently read between 48% and 50% $F_{I_{O_2}}$. The pulse oximeter (Nellcor N-100[®]), the nondisposable finger probe (Nellcor DS-100A[®]) of which had been affixed to the right great toe prior to induction, consistently displayed an Sp_{O_2} of 99% or 100%. The right lower extremity and pulse oximeter probe were covered by a surgical drape. The operating room temperature was 67° F.

Between 45 and 47 min after induction, Sp_{O_2} gradually declined from 99% to 94%. During this decline the blood pressure remained at 110/70, the heart rate remained at 68 beats/min, and the nasopharyngeal temperature remained at 97.1° F. Ventilator settings (rate 10/min, tidal volume 700 ml) and end-tidal P_{CO_2} (31 mmHg) were likewise unchanged. The endotracheal tube position had not apparently changed (23 cm at the lips); bilateral breath sounds were present; the pilot balloon was still inflated and there was no audible cuff leak; all circuit connections were tight; the oxygen analyzer read 48%; flowmeters were unchanged and freely movable; and wall O_2 pressure remained at 55 psi.

At this point the drape over the pulse oximeter probe was uncovered, and the probe was found to have become partially dislodged from the right great toe, so that the light-emitting diodes (LED) in the probe were positioned only 1–2 mm from the tip of the toe. The probe was replaced so that the LED were as proximal as possible (11 mm from the tip of the toe), and the Sp_{O_2} returned to 99% within 10 s. *In vitro* oximetry performed on a contemporaneous radial arterial blood sample (IL-282 CO-oximeter, Instrumentation Laboratory, Lexington, Massachusetts), obtained while Sp_{O_2} was 99% and processed within 1 min, yielded an Sa_{O_2} of 98.0%. The remainder of the intraoperative course was uneventful, as was recovery.

MATERIALS AND METHODS

In view of this case, we investigated probe placement as an influence on the Sp_{O_2} value displayed by a pulse oximeter. The study was done in two parts. To rule out the possibility that artifact is due to a single idiosyncratic device, Part 1 examined the effect of probe placement on Sp_{O_2} in a single subject using multiple probes and machines. Part 2 then examined probe placement in 20 anesthetized subjects.

Part 1. Five Nellcor N-100 pulse oximeters (Nellcor, Hayward, California), each with its own nondisposable finger probe, were employed in the study. For each individual machine, two sets of measurements were taken: one in an operating room dedicated primarily to ophthalmologic surgery, where the temperature was 21° C, and one in an operating room dedicated primarily to cardiac surgery, where the temperature was 13° C. The only ambient light in either room was from fluorescent lights of identical make and intensity. No surgical lamps were in use. The left index finger of one investigator (J.F.K.) was used for all measurements.

Before a given set of measurements was recorded, the

subject sat in the designated room and breathed oxygen at 6 l/min by mask for at least 10 min. An ECG monitor was attached. The subject's left arm was positioned horizontally on a table, elbow flexed, at right atrium level. The pulse oximeter was turned on, and the subject's left index finger was inserted into the probe receptacle as far as possible, so that the fingertip touched the blind end of the tunnel formed by the receptacle. This maneuver placed the probe LED 11 mm proximal to the fingertip (position +11 mm). No set of measurements was begun until the baseline value had been continuously displayed for at least 60 s.

A set of measurements consisted of successive Sp_{O_2} determinations as the probe was progressively displaced along the long axis of the finger as follows. A baseline Sp_{O_2} was recorded as above at +11 mm. The probe was then removed and replaced at position +10 mm (LED 10 mm proximal to fingertip), and Sp_{O_2} was again recorded. Sp_{O_2} values were similarly obtained at subsequent 1-mm intervals (+9 mm, +8 mm, etc.) up to and including position –9 mm. Because removal and replacement of the probe perturbs Sp_{O_2} transiently, no Sp_{O_2} value was recorded until it had been displayed continuously for 30 s along with a heart rate within 10% of that recorded simultaneously by ECG. The subject breathed 6 l/min oxygen by mask throughout. Testing of each machine was completed in both rooms before the next machine's tests were begun.

Part 2. Twenty subjects, undergoing a variety of surgical procedures under various general and regional anesthetic techniques, were studied. None of the subjects had clinical or laboratory evidence of pulmonary or peripheral vascular disease.

Two Nellcor DS-100A probes, each attached to a Nellcor N-100 pulse oximeter, were applied, one to each index finger. To estimate cutaneous vasomotor tone, two Mon-a-Therm[®] skin temperature sensors (Mallinckrodt, St. Louis, Missouri) were affixed, one to the tip of the thumb opposite the nail bed and one to the radial side of the ipsilateral arm midway between wrist and elbow away from superficial veins. Forearm-to-digit skin temperature gradients are accepted indicators of cutaneous vasomotor tone.¹⁶ A temperature gradient (dT) was obtained by subtracting thumb temperature from forearm temperature.

The pulse oximeter ipsilateral to the skin temperature sensors was manipulated, as described for Part 1, to obtain a set of Sp_{O_2} values at positions +11 mm to –9 mm. The contralateral pulse oximeter served as a control; data collection began only after the control oximeter had displayed a single Sp_{O_2} value, within 2% of the ipsilateral oximeter's Sp_{O_2} value, continuously for 60 s. If control Sp_{O_2} changed at all during data collection, the entire set was rejected.

Statistical analysis was by Student's *t* test.

RESULTS

Part 1. Data are shown in table 1. In each of the ten data sets, a baseline SpO₂ value was easily obtainable when the finger was inserted all the way into the probe (position +11 mm). Although these ten baseline values ranged from 98% to 100%, each particular combination of machine and room had its own specific baseline value, which was readily maintained for the requisite 60 s.

As the finger was progressively withdrawn from the probe receptacle in 1-mm intervals, the baseline value was maintained for a distance that varied between 3 and 9 mm. In some data sets, further withdrawal of the finger caused loss of signal and display of an SpO₂ of zero with "pulse signal" alarm.

In other data sets, however, further withdrawal of the finger yielded nonzero SpO₂ values, which were generally lower than the baseline value and decreased as the finger continued to be withdrawn. This "penumbra" of nonzero SpO₂ values ranged from 1 to 14 mm. Eventually, an SpO₂ of zero was attained in all ten data sets.

At 21° C, a penumbra at least 4 mm long was observed for all five machines (mean ± SD, 9.4 ± 5.1 mm). At 13° C, however, only two of the five machines demonstrated penumbras of 1 and 4 mm, respectively (mean ± SD, 1.0 ± 1.7 mm). The difference in mean penumbra length at the two temperatures was statistically significant (*P* < 0.02).

Part 2. Of the nine subjects whose fingers were colder than their forearms (dT > 0), three (33%) exhibited penumbras; mean penumbra length for all nine subjects was

0.4 ± 0.7 mm. Of the 11 subjects whose fingers were warmer than their forearms (dT < 0), nine (82%) exhibited penumbras; mean penumbra length for these 11 subjects was 5.2 ± 4.3 mm. The difference in penumbra length between the two groups was significant (*P* < 0.005).

In both Parts 1 and 2, machines were noted to be more sensitive to motion artifact as the end of a penumbra (*i.e.*, the onset of loss of pulse signal) was approached. In data sets that did not exhibit penumbras, however, no such increase in motion artifact was noted before pulse signal was lost.

DISCUSSION

SpO₂ can be artifactually altered by an improperly placed pulse oximeter probe. This effect is more likely to occur, and occurs over a wider range of probe positions, with cutaneous vasodilation.

Pulse oximeters use LED to transilluminate tissue with red (660 nm) and infrared (910 nm or 940 nm) light, measuring and processing the resulting absorbance data. By sampling several hundred times per second, they detect pulsatile variations in the data and distinguish constant absorbances (due to skin, bone, muscle, and other absorbers) from pulsatile ones. If arterial blood is the only significant pulsatile absorber, arterial oxygen saturation can be derived from the absorbance characteristics of oxygenated and deoxygenated hemoglobin.¹²

The term "penumbra" denotes, among other things, the partial shadow cast by an orbiting astronomical body and observable during eclipses (Latin *paene*-, almost, *um*-

TABLE 1. SpO₂ Values at Successive Probe Positions at 21° C and 13° C

Position	21° C					13° C				
	Machine No.					Machine No.				
	1	2	3	4	5	1	2	3	4	5
+11	99	98	98	98	100	100	100	100	100	100
+10	99	98	98	98	100	100	100	100	100	100
+9	99	98	98	98	100	100	100	100	100	100
+8	99	98	98	98	100	100	100	100	100	100
+7	98	98	97	98	100	100	100	100	100	100
+6	97	97	97	98	100	100	100	100	100	100
+5	97	97	97	97	100	0	97	100	100	100
+4	99	96	96	97	100	0	0	100	100	100
+3	98	95	96	97	98	0	0	0	100	0
+2	97	0	96	98	98	0	0	0	100	0
+1	95	0	95	97	98	0	0	0	99	0
+0	94	0	92	95	95	0	0	0	97	0
-1	93	0	90	91	0	0	0	0	93	0
-2	92	0	88	90	0	0	0	0	93	0
-3	91	0	87	90	0	0	0	0	0	0
-4	0	0	87	89	0	0	0	0	0	0
-5	0	0	88	90	0	0	0	0	0	0
-6	0	0	86	90	0	0	0	0	0	0
-7	0	0	0	89	0	0	0	0	0	0
-8	0	0	0	89	0	0	0	0	0	0
-9	0	0	0	0	0	0	0	0	0	0

bra, shadow). It also finds literal application in pulse oximetry because tissue poorly interposed between LED and sensors may block the path enough to allow transillumination of a pulsatile vascular bed and production of a pulsatile signal, but not enough to prevent a significant degree of direct illumination of sensor by LED (noise). As pulsatile signal amplitude decreases, gain is compensatorily increased; background noise amplitude increases as well. Signal and noise are added at each of two wavelengths, and the ratio of the two sums is associated with an empirically derived Sp_{O_2} value for display. As the contribution of noise to both of these sums is increased, the ratio tends toward unity, which happens to correspond to an Sp_{O_2} of 85.

This phenomenon, termed "optical shunt" by the manufacturer (Pulse Oximetry Technical Note No. 5, Nellcor, Inc., Hayward, California) may be partially or entirely responsible for the penumbra effect. If so, Sp_{O_2} values above 85 (as were reported in this study) would be expected to decrease artifactually toward 85 with progressive probe displacement, while Sp_{O_2} values below 85 would be expected to increase.

At least two mechanisms may be responsible for the influence of cutaneous vasomotor tone on the penumbra effect. The first is optical shunt. Generally, a low signal-to-noise ratio causes display of some notification (pulse search light, probe off finger message) that the data is unacceptable, but pulse oximeters may not be able to reject unacceptable data under all circumstances,¹⁷ especially when the pulsatile signal is relatively strong, as during cutaneous vasodilation.

The second mechanism that may explain the observed influence of vasomotor tone on the penumbra effect involves cutaneous arteriovenous anastomoses, which mediate the vasoconstrictive thermoregulatory response. Using photoelectric plethysmography, Kim *et al.*¹⁴ have suggested that the pulsatile flow detected in pulse oximetry is physically present not only in arteries or arterioles but also, at least partly, in venules or veins that receive pulsatile flow directly from the arterial circulation *via* these anastomoses, which are unique to the cutaneous circulation. At 21° C, these anastomoses may be more patent than at 13° C if thermoregulation is intact.

During systole at 21° C, relatively more (well-oxygenated) blood may be expected to course through these anastomoses, expanding the vascular bed and producing a pulse signal. As it does so, however, it may displace blood from venules, which receive both well-oxygenated blood from the anastomoses and poorly oxygenated blood from capillaries perfusing cutaneous and other tissue. Because resistance is low between anastomosis and venule, this volume of displaced blood may also be pulsatile, contributing a relatively desaturated component to the pulse oximeter signal. (This reasoning was used by Kim *et al.*¹⁴

to explain decreased Sp_{O_2} in dependent extremities, but it is conceivable that cutaneous anastomoses permit venular pulsation even at the level of the right atrium, as in this study. Indeed, the variation we observed in baseline Sp_{O_2} with temperature—usually 98% or 99% at 21° C but always 100% at 13° C—could be taken as evidence for this phenomenon.)

During systole at 13° C, however, the anastomoses are relatively closed, and any desaturated blood draining into venules is more likely to be nonpulsatile. Sp_{O_2} at 13° C is thus more likely to be accurate up until the signal is lost due to progressive probe displacement.

In summary, our data suggest that inaccurate pulse oximeter probe placement can cause spuriously low Sp_{O_2} , especially at warmer ambient temperatures. The mechanism probably involves some combination of optical shunt, a weak pulse signal, and flow through cutaneous arteriovenous anastomoses. Unlike many other sources of pulse oximeter artifact, which depend on interference from the external or internal milieu (*e.g.*, dyes and dyshemoglobins), the penumbra effect is a potential threat to accuracy virtually every time a pulse oximeter is used. Because it is easily detected and remedied, and probably common as well, it should be high in the differential diagnosis whenever a pulse oximeter displays a mild degree of desaturation.

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Desaturation Noted by Pulmonary Artery Catheter Oximeter after Methylene Blue Injection

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Oxyhemoglobin saturation of mixed venous blood ($S\bar{v}O_2$) reflects a balance between oxygen supply and demand. Many physicians have used this parameter to evaluate the adequacy of oxygen transport in the critically ill and in patients undergoing major surgery.^{1,2} The saturation measured by catheter oximetry correlates well with values obtained from a laboratory co-oximeter, particularly when a three wavelength catheter is used.³

Previous reports⁴⁻⁷ have drawn attention to the association between artifactual decreases in oxyhemoglobin saturation as measured by pulse oximetry (SpO_2) and intravenously administered dyes. The effects of intravenously administered methylene blue on continuous mixed venous oximetry have not yet been described.

We report two cases in which the iv administration of methylene blue caused a significant decrease in the oxyhemoglobin saturation measured by the Opticath® pulmonary artery catheter (SxO_2), (Abbott Critical Care Systems, Mountain View, California):

CASE REPORTS

Case 1. A 54-yr-old man with bladder cancer but otherwise healthy was scheduled to undergo radical cystectomy and formation of a continent colonic reservoir.

Anesthesia was induced with sufentanil and maintained with N_2O , O_2 , isoflurane, and sufentanil. Paralysis was provided by vecuronium. Intraoperative monitoring included electrocardiogram, precordial stethoscope, oral temperature, intraarterial blood pressure, noninvasive automated blood pressure, end-tidal CO_2 (ET_{CO_2}) tension, and finger pulse oximetry (Oximetrix® 3 System). The latter three integrated in the monitoring modules of the Narkomed 3 anesthesia system (North American Dräger, Telford, Pennsylvania).

After surgery started a 7.5-Fr fiberoptic pulmonary artery (PA) catheter (Opticath® model P-7110) was inserted *via* the right internal jugular vein. The fiberoptic PA catheter was connected to a saturation/cardiac output computer (Oximetrix® 3 System). The procedures recommended by the manufacturer for pre-insertion calibration and light intensity calibration were followed. During the initial portion of the operation, the SxO_2 and the SpO_2 ranged 78-85%, and 99-100%, respectively.

After completion of the resection, 100 mg (10 ml) of methylene blue was injected over 10 s *via* a peripheral iv catheter to evaluate urinary patency. Within 30 s the SxO_2 , which was 78% before the injection, decreased to 13% (fig. 1). There were no changes in other monitored parameters obtained at the SxO_2 nadir, including cardiac output, heart rate, pulmonary artery pressure, wedge pressure, and mean arterial blood pressure (table 1). The hematocrit was also unchanged. At the same time, a mixed venous specimen sent for blood gas analysis disclosed a $P\bar{v}O_2$ of 31 mmHg (calculated saturation 68%) compared with 35 mmHg (calculated saturation 78%) prior to the dye administration.

The SpO_2 decreased to 22% at 30 s after the methylene blue administration. The SpO_2 and SxO_2 returned to baseline within 5 and 10 min, respectively. At the end of the otherwise uneventful operation, the patient was transferred to the recovery room.

Case 2. A 67-yr-old man was admitted to the surgical emergency room after a motor vehicle accident. On admission he was hypotensive and noted to have ischemic changes on the electrocardiogram. After resuscitation his workup disclosed multiple bone fractures. The patient was transferred to the surgical intensive care unit to continue fluid resuscitation, monitoring, and respiratory support. Monitoring included electrocardiogram, intraarterial blood pressure, and SpO_2 (Nellcor® N-200). A 7.5-Fr fiberoptic PA catheter (Opticath® model P-7110) was inserted *via* the right subclavian vein and was connected to the saturation/cardiac output computer.

On the second day after admission, the patient underwent fixation of bilateral tibia and fibula fractures. Postoperatively, the patient de-

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