

## CASE REPORTS

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## Intraarterial Vasodilator Administration to Restore Pulse Oximeter Function

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PULSE oximetry measurement of peripheral arterial hemoglobin oxygen saturation is considered standard care during administration of anesthesia. A recent review discussed the scientific and clinical issues surrounding this technology.<sup>1</sup> Several circumstances may make pulse oximetry readings inaccurate or unobtainable, including conditions related to the patient (carboxy- and methemoglobinemia, peripheral vasoconstriction, hypotension), use of dyes (methylene blue), and interference from operating room equipment (electrocautery).<sup>1</sup> One common reason for poor or absent pulse oximeter function is peripheral vasoconstriction. When this situation occurs, common strategies to maintain pulse oximeter function include placing the pulse oximeter probe on sites other than the fingertip (ears, nose, toes), and wrapping the hand to conserve heat. Enhancing blood flow to a finger with a digital block using local anesthesia recently was reported to restore pulse oximeter function.<sup>2</sup> Here we report intraarterial administration of vasodilators (ni-

troglycerin and hydralazine) through an ipsilateral indwelling radial artery catheter as a simple alternative to reverse local vasoconstriction and restore pulse oximeter function.

### Case Report

A 54-yr-old woman, American Society of Anesthesiologists physical status 3, was admitted with vascular insufficiency and infection of the distal right leg. History included hypertension, non-insulin-dependent diabetes mellitus, hemodialysis dependent renal failure, left hemisphere cerebrovascular accident 18 yr ago, and 30 pack/yr cigarette use. Medications were insulin, vancomycin, ceftazidime, and metronidazole. Physical examination showed blood pressure 128/72 mmHg in the right arm, heart rate 88 beats/min, and weight 56 kg. There was a left carotid bruit. Lungs were clear to auscultation and percussion, cardiac sounds revealed an S<sub>4</sub> and systolic ejection murmur, and no edema was present. A native vessel arteriovenous fistula was present in the left forearm. No pulse was palpable in the right leg, and the right second toe was dark and painful. Hematocrit was 21.5%. After arteriography was performed, a right leg femoral-anterior tibial bypass procedure was planned.

In the operating room, a 20-G right radial arterial catheter and a lumbar epidural catheter were placed. Bupivacaine (0.5%) was given through the lumbar epidural catheter to maintain a T8 level of anesthesia. Axillary temperature was 35.1° C. Both hands were wrapped in plastic bags, and the room temperature was maintained at 24° C. Twenty minutes after incision, the pulse oximeter stopped functioning, being unable to detect a digital pulse. Both hands were cool to touch. No pulse oximetry signal could be generated from any of the fingers, from either ear, or the nose. Blood pressure was stable throughout this period, and axillary temperature was 35.2° C.

The pulse oximeter dysfunction was thought to be due to peripheral vasoconstriction. To provide local vasodilation, 10 µg intraarterial nitroglycerin in a 2-ml volume was injected over 1-2 min through the radial artery catheter with the pulse oximeter probe placed on the right second fingertip. Pulse oximeter function was restored within 1 min of completing the injection and maintained for 12 min; repeat doses of 10 µg nitroglycerin were given and maintained pulse

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oximeter function for 9, 5, and 7 min on successive doses. In an attempt to obtain a similar result with a longer-acting agent, intraarterial hydralazine diluted to 1 mg in 5 ml normal saline was infused over 1 min in two 0.5-mg doses for a total 1 mg *via* the indwelling arterial catheter. There was no change in systemic blood pressure associated with injection. There was a delay of about 6 min before pulse oximeter function was restored after the hydralazine injection. A noticeable difference in skin temperature developed between the right and left hand, likely reflecting increased blood flow to the right hand from local vasodilation. Beginning at about 2 h, 15 min after intraarterial hydralazine injection, this temperature difference was quantitated using a skin surface temperature probe placed on the middle finger of each hand. When the probe was first applied, the right hand skin surface temperature was 8.7° C warmer than the left hand skin surface temperature. The relative warmth of the right hand gradually declined. The pulse oximeter stopped working 4 h and 41 min after the initial intraarterial hydralazine injection. At this time the left hand temperature was 23.1° C and the right hand temperature was 25.8° C. A repeat injection of 0.5 mg hydralazine was given, and pulse oximetry function was restored within 30 s after slow injection. There was a gradual increase in right hand skin surface temperature, peaking at 2 h after injection. Pulse oximeter function was maintained for 3 h, 6 min. After the pulse oximeter again stopped functioning, 10 µg nitroglycerin intraarterial again was tested to assess the length of its effect. Pulse oximeter function was restored for 16 min. A final dose of 0.2 mg hydralazine intraarterial was then given. Pulse oximeter function was restored until the end of the procedure, 1 h, 17 min later.

### Discussion

Pulse oximetry is a standard intraoperative monitor. The incidence of failure to obtain a pulse oximetry reading, defined as inability to obtain any reading for a period of 30 min or longer despite elimination of mechanical problems and use of all possible sites (other digits, ears, nose) was determined to be 1.1% in a prospective study of 11,046 anesthetics.<sup>3</sup> The incidence was greater in a group of Veterans Administration hospital patients (4.2%) with presumably a greater incidence of multisystem and cardiovascular disease compared to a group of pediatric patients, in whom the incidence was less (0.6%).<sup>3</sup> Once mechanical factors are eliminated, a common reason for pulse oximeter dysfunction is peripheral vasoconstriction.

In our patient, pulse oximeter function was restored using an infusion of vasodilators, nitroglycerin, and hydralazine, administered through an indwelling arterial catheter. The nitroglycerin in a dose of 10 µg would restore pulse oximeter function within 1 min; though in this patient, pulse oximeter function failed again after 5–16 min for each of five nitroglycerin doses.

(Anecdotal experience of the authors with nitroglycerin to restore pulse oximeter function in several other patients with peripheral vasoconstriction sometimes showed an effect for up to several hours). To maintain pulse oximeter function for a longer period of time in this patient, hydralazine, which has a longer active effect than nitroglycerin, was given as a total dose of 1 mg through the arterial catheter. The hydralazine maintained pulse oximeter function for more than 4 h.

Hydralazine is a phthalazine derivative used principally as an antihypertensive agent. Its mechanism of action is a direct vasodilation of vascular smooth muscle, with arterioles effected more than veins.<sup>4,5</sup> The vasodilation is hypothesized to result from the breakdown of hydralazine to nitric oxide.<sup>6</sup> *In vitro*, a suspension of erythrocytes to which hydralazine is added leads to generation of nitric oxide.<sup>6</sup> The effects of intraarterial injection of hydralazine in humans were investigated nearly 30 yr ago by Ablad.<sup>7,8</sup> When administered into the brachial artery in normal volunteers, 0.3–0.55 mg hydralazine evoked a decrease in vascular resistance in the hand and forearm on the side where it was given but no effects on the opposite side. The vasodilation developed gradually and reached its maximum effect 12–20 min after administration. The vasodilation lasted 1–4 h.<sup>7,8</sup> Similar to our patient, no adverse systemic hemodynamic effects were noted.

While the technique described is most easily performed in a patient with an indwelling arterial catheter, we have employed percutaneous arterial injection using a small-gauge (26-G Butterfly) needle. A dramatic, prolonged restoration of pulse oximetry function ensued.

Potential problems with intraarterial drug administration include the possibility of injection of air or particulate matter, but this risk is no greater than that associated with flushing or other manipulations of an intraarterial catheter. Systemic hypotension could result from intraarterial administration of vasodilators, but the doses used are small compared to standard hypotensive doses and are injected slowly. Systemic hypotension has not been a problem in human volunteers<sup>7</sup> or to date in approximately ten patients in whom we have used this technique.† These patients received between 0.5 and 1.0 mg hydralazine diluted in 10 ml of normal saline. We have not attempted to determine a minimum effective dose. The duration of effect seems to be a function of dose, as in the case reported. Our experience indicates intraarterial vasodilator administration

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should be considered among a growing list of techniques for restoring pulse oximeter function.

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## Pacemaker Interactions with Transcutaneous Cardiac Pacing

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BECAUSE of electrical interference, patients with permanent or temporary pacemakers are at increased risk in the operating room and critical care environment.<sup>1</sup> We describe two cases of interaction of noninvasive transcutaneous pacing with an implanted demand (VVI) transvenous pacemaker and temporary epimyocardial pacing, respectively. Whereas in the first case, this interaction could be exploited therapeutically to inhibit ventricular pacing by the implanted transvenous pacemaker, resulting in resumption of sinus rhythm with improved hemodynamics, it evoked asystole by suppression of epimyocardial pacing in the second case.

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## Case 1

An 84-yr-old woman (53 kg body weight, 160 cm) with femoral neck fracture was scheduled for total hip arthroplasty. Past medical

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history included sick sinus syndrome with several episodes of syncope, resulting in right infraclavicular implantation of a ventricular pacemaker (Siemens, Munich, Germany; programmed mode VVI, rate 65 beats/min) 3 yr ago, congestive heart failure (New York Heart Association classification grade 3), hypertension, and diabetes. Except for bilateral tibial edema and discrete rales over both lower dorsal lung fields, preoperative physical examination, vital signs, and laboratory studies were unremarkable. Medications included digitoxin, verapamil, nifedipine, isosorbide dinitrate, furosemide, glyburide, and L-thyroxine. A chest x-ray showed left heart enlargement, Kerley-B lines, and somewhat prominent interstitial markings. A 12-lead electrocardiogram revealed sinus rhythm (85 beats/min) with regular ventricular pacing (65 beats/min) during application of a magnet.

Following placement of electrocardiogram leads (II and V5), radial artery, and basilic vein central venous catheters, anesthesia was induced with divided doses of thiopental (total 100 mg intravenously), fentanyl (total 200 µg), and vecuronium (6 mg), and after tracheal intubation, maintained with enflurane (0.6-1%) in nitrous oxide/oxygen (50%/50%). Arterial pressure decreased from a baseline of 160/90 to 120/60 mmHg, with sinus rhythm at a rate of 80-100 beats/min. However, over the next 20 min, heart rate decreased progressively, and when ventricular pacing ensued at the programmed demand rate of 65 beats/min, arterial pressure precipitously decreased to 70/35 mmHg (mean 46 mmHg).

To improve hemodynamics, the anesthesiologist intended to increase the heart rate using a transcutaneous pacemaker (Pace 500 D, Osypka, Grenzach, Germany) connected to circular electrodes (diameter 13 cm, effective electrode area 50 cm<sup>2</sup>) that had been taped to the precordial and left paravertebral skin to be used in case the implanted pacemaker would malfunction during electrocautery. Transcutaneous stimulation (pulse width 40 ms, rate 80 beats/min) was begun with a current of 10 mA when it was noted that this maneuver evidently inhibited the implanted pacemaker and resulted in recovery of sinus rhythm at a rate of 60 beats/min (fig. 1). Under these conditions, arterial pressure immediately increased to 98/45