the toxin resulted in very short-lived reduction in movements only and in no change in pain. It is possible that nerve blocks may have little prognostic value for intramuscular botulinus toxin injection, similar to the poor correlation reported for nerve blocks and the long-term outcome of dorsal rhizotomy. We had blocked the dorsal scapular nerve on three occasions, and each time pain decreased proportional to rhomboid paresis. Nevertheless, prolonged weakness of these muscles from botulinus toxin did not result in a similar reduction in pain.

References


Anesthesiology
78:365–366, 1993
© 1993 American Society of Anesthesiologists, Inc.
J. B. Lippincott Company, Philadelphia

False Desaturation Due to Intradermal Patent Blue Dye

Robert C. Morell, M.D.,* Theodore Heyneker, M.D.,* Hillel I. Kasplan, M.D.,* Charlie Ruttepe, CRNA†

THE intravenous administration of dye has been reported to falsely decrease oxygen saturation as determined by pulse oximetry.1,2 We now report the first case of spurious desaturation due to a small intradermal injection of patent blue dye. The recent publication of a new surgical technique for intraoperative lymphatic mapping of early stage melanoma describes the intradermal injection of small aliquots of patent blue dye. This allows mapping of the lymphatic drainage of a malignant melanoma and the identification of a "sentinel node" indicating lymphatic spread of the disease. Morton et al. reported this technique and successfully utilized it to identify a sentinel node in 194 of 237 patients.3 The first use of this technique at our institution resulted in a delayed and marked decrease in oxygen saturation as determined by pulse oximetry.

Case Report

A 54-yr-old, 80.8-kg, 157.5-cm tall woman was scheduled for excision of a malignant melanoma from her right arm. Lymphatic mapping and identification of a sentinel node also was planned. Her medical history included a subtotal thyroidectomy, knee surgery, and mild hypertension. She was allergic to penicillin. Her only medication was levotiroxine, 0.125 mg/day. Physical examination revealed a mildly obese female with a Mallampati class I airway. Laboratory studies were unremarkable. Monitoring included electrocardiogram (leads II and V4), automated blood pressure cuff, precordial stethoscope, pulse oximetry, capnography, and end-tidal gas analysis.

A Nellcor N-100 pulse oximeter (Hayward, CA) with an Oxisensor D-25 oxygen transducer was placed on the index finger of the hand contralateral to the surgical side. Her room air hemoglobin oxygen saturation (Sao2) prior to the induction of anesthesia was 100%. Following premedication with 2 mg intravenous midazolam, anesthesia was induced with 250 mg sodium pentothal and 250 μg fentanyl. Neuromuscular relaxation was obtained with 10 mg vecuronium. Tracheal intubation was accomplished without difficulty following the administration of 150 mg 4% laryngotraceal lidocaine.

Following intubation, anesthesia was maintained with isoflurane, 50% N2O, 50% oxygen, with supplemental fentanyl administered incrementally to a total of 450 μg. The lungs were ventilated with an 800-ml tidal volume at a frequency of 6 breaths/min. Prior to administration of the patent blue dye, the Svo2 was 99–100%. Intraoperatively, 1.5 ml 10% patent blue dye (compounded by our hos-

*Assistant Professor of Anesthesia.
†Certified Registered Nurse Anesthetist.

Received from the Department of Anesthesia, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina. Accepted for publication October 13, 1992.

Address reprint requests to Dr. Morell: Department of Anesthesia, Wake Forest University Medical Center, Medical Center Boulevard, Winston-Salem, North Carolina 27157-1009.

Key words: Measurement technique: Pulse oximetry. Patient blue dye.
pial pharmacy from 50% patent blue dye powder (Aldrich) was administered intradermally into the melanoma. Twenty minutes later a second intradermal injection of 1.0 ml patent blue dye was administered.

Approximately 23 min after the first injection, the \( \text{SPO}_2 \) gradually decreased to 92%. Arterial blood gases drawn from the left radial artery on an \( \text{PiO}_2 \) of 0.5, during an \( \text{SPO}_2 \) reading of 92%, revealed \( \text{pH} 7.45, \text{PaCO}_2 35, \) and \( \text{PiO}_2 225. \) Despite our increasing the \( \text{PiO}_2 \) to 1.0 for 10 min, apparent desaturation continued to a nadir of 89%. Vital signs were unchanged and no cyanosis or abnormal skin color was visible. Arterial co-oximeter obtained 1 h after the initial dye injection revealed \( \text{Hgb} 13.3, \text{O}_2 \text{Hgb} 97.6\%, \) and \( \text{COHgb} 1.4\% \) (Instrumentation Laboratory, Model 282 Co-Oximeter, calibrated 2 h prior to sampling).

A second arterial blood gas obtained 2 h after the decreased dye injection, on an \( \text{PiO}_2 \) of 0.5, demonstrated \( \text{pH} 7.45, \text{PCO}_2 35, \) and \( \text{PO}_2 222. \) At this time the patient's skin had a noticeable blue-green tinge. At the conclusion of surgery, no more patent blue dye had been administered, and the patient's skin color was obviously blue-green. Emergence and recovery were uneventful.

Discussion

Several intravenously administered dyes including methylene blue, indigo carmine, and indocyanine green have been reported to interfere with pulse oximetry.1,2 The intradermal administration of dye has not been reported to be a problem. Gabrielleczky and Buist reported that the intraoperative administration of intravenous patent blue dye did not affect the accuracy of postoperative pulse oximetry.3 The discrepancy between their observations and ours may be due to the unspecified interval between intraoperative dye administration and their postoperative pulse oximetry measurements. The difference between intraarterial and intradermal routes also would influence the pharmacokinetics of dye distribution. The interference of patent blue dye with pulse oximetry is predictable because the absorption spectra of patent blue dye peaks at 635 nm, which easily could overlap with the standard 660-nm wavelength utilized by the Nellcor N-100.

The delayed onset and long duration of the decreased \( \text{SPO}_2 \) is different, however, from the brief effect seen with intravenously administered dyes.4,5 Since a 23-min latency occurred, tissue absorption could be significant and might alter the DC component signal (absorption due to tissue, capillary, venous, and nonpulsatile arterial flow).5 The AC component of the signal is due to pulsatile arterial absorption alone. Since hemodynamics remained unchanged and the patient con-

continued to have good capillary refill and full peripheral pulses, it is unlikely that the dye induced any hemodynamic aberration that would have altered the AC component and caused the spurious desaturation. Co-oximetry demonstrated normal levels of oxyhemoglobin and eliminated the possibility of methemoglobinemia as an etiology for the decreased \( \text{SPO}_2. \)

The recent publication of this new surgical technique for the mapping of the lymphatic drainage of early malignant melanoma will increase the likelihood that anesthesiologists will encounter patients receiving intradermal injections of patent blue dye. Eroneous pulse oximeter readings should be expected to occur.

In this case, approximately 20 min elapsed between the intradermal injection and the beginning of desaturation. The decreased \( \text{SPO}_2 \) was insidious and lasted into the recovery period several hours later. In contrast, the apparent desaturation that occurs with intravenous administration of methylene blue has a latency of 35–80 s, a duration of 50–115 s, and a \( \text{SPO}_2 \) nadir ranging from 1% to 91%.1 The slow onset and long duration of the apparent desaturation seen with intradermal patent blue dye may make pulse oximetry data interpretation inaccurate for an undefined period of time. Clinical judgment and laboratory analysis of arterial blood samples are necessary to differentiate real arterial hemoglobin desaturation from spurious values.

References


Anesthesiology, V 78, No 2, Feb 1993