and the severity and frequency of the URI. Those who
must be anesthetized should be given supplemental ox-
gen and have their oxygen saturation monitored in the
recovery room and during transport.

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Pulse Oximeter Desaturation Due to Methemoglobinemia

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Pulse oximetry is widely used to provide a continuous
estimate of arterial oxygen saturation (SpO2). Pulse ox-
imeters are inferential monitors. They measure the
light absorbance of perfused tissues at two wavelengths
(660 and 940 nm) and, from the ratio of the pulse-
absorbed absorbance signals at these two wavelengths,
use an algorithm to estimate arterial oxygen saturation.1,2
Erroneous SpO2 readings have been reported with the
use of dyes3,4 and in the presence of carboxyhemoglo-
bin.2 A case is reported in which a low SpO2 reading
alerted us to the presence of unsuspected methemoglo-
binemia.

REPORT OF A CASE

A 20-yr-old woman was scheduled to undergo a total proctoco-
ectomy. Six years prior to this admission, she had bloody diarrhea and
abdominal pains and was diagnosed as having ulcerative colitis. She
was treated with prednisone and asulfanide. Subsequently, she de-
veloped pyoderma of her left hand, which was treated by surgical drainage
under uneventful general anesthesia 2 yr prior to this admission.
Two months prior to admission, she developed pyoderma gangreno-
sus of the anterior tibial region bilaterally, and therapy with dapsone
was begun. There was nothing else of note in her past medical history.
At the time of admission, her medications included only prednisone 20
mg b.i.d. and dapsone 150 mg q.i.d.; the latter drug was discontinued
upon admission. On physical examination, the patient was in no appar-
ent distress, was obese (72.72 kg, 160 cm), and had pyoderma of both
shins. Arterial blood pressure was 128/70 mmHg, heart rate 100 bpm
and regular, and respiratory rate noted as 22 breaths/min. Physical
examination was otherwise unremarkable, and, in particular, there
was no evidence of cyanosis. Preoperative laboratory values were
within normal limits (hemoglobin 10.9 g/dl, hematocrit 31.7%, blood
CO2 26 mEq/l). Chest radiograph was reported as normal.

On the third day of hospitalization, the patient was premedicated
with morphine sulfate, 10 mg i.m. and hydrocortisone, 100 mg i.v. Upon
arrival to the OR, monitoring with an EKG, automated blood
pressure monitoring device (Dinamap™), and a finger sensor con-
nected to an Ohmeda Biot 3700 pulse oximeter (Ohmeda, Boulder,
CO). With the patient awake, supine on the operating table, and
breathing room air, the pulse oximeter read an SpO2 of 89%. The
sensor was checked and placed on a finger of the opposite hand,
but the SpO2 reading remained 89–90%. The sensor was placed on the
author’s finger, and the SpO2 increased to 97%. The sensor was re-
placed on the patient’s finger, and she was given 100% O2 to breathe
from the anesthesia circle system via a facemask. The pulse oximeter
reading increased to 95%, and remained steady at an SpO2 of 92–95%.
Analysis of arterial blood gases with a Fio2 of 1.0 showed pH 7.46,
PaCO2 39 mmHg, PaO2 587 mmHg, calculated saturation (by nomo-
gram) 99.9%, oxyhemoglobin percentage of total hemoglobin
(%) by an IL 282 laboratory co-oximeter (Instrumentation Lab-
atory, Lexington, MA) 92.8%, hemoglobin 10.5 g/dl and hematoc-
rit 34%. A second arterial sample was analyzed for the presence of
dyshemoglobins. Methemoglobin was detected at a percentage
concentration of 5%, and carboxyhemoglobin 2.0%, of total hemoglobin.
Anesthesia was induced with thiaramyl sodium 300 mg iv, and paralysis
was provided by succinylcholine 100 mg iv followed by iv increments
of vecuronium. Anesthesia was maintained with N2O/O2 (4/1), iso-
flurane, and fentanyl. The anesthetic and surgical courses were
uneventful. The trachea was extubated at the end of the procedure and
the patient was transferred to the recovery room. While breathing an
Fio2 of 0.4 from a face mask, SpO2 read 94%, pH 7.46, PaCO2 33
mmHg, PaO2 129 mmHg, base excess −1.0 mEq/l calculated satu-
ration 98.9%, measured %HBO2 (by IL 252) 98.4%, methemoglobin
2.9%, and carboxyhemoglobin 0.5%. A blood sample sent for hemoglo-
nin electrophoresis showed a normal pattern. The patient made an
uneventful recovery from her surgery and was discharged home 2
weeks later.

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Key words: Blood; methemoglobin. Measurement techniques: pulse
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oxygenated hemoglobin percentage† (amount of oxygenated hemoglobin expressed as a percentage of the amount of total hemoglobin) using an empirical algorithm built into the oximeter software. This algorithm was created by measuring the absorbances of light at 660 and 940 nm in the fingers and earlobes of healthy adult volunteers breathing various gas mixtures. At these sites, the light absorbance is constant except for the absorbance by the arterial pulse-added blood volume due to arterial pulsations. The varying pulse-added absorbances are converted into plethysmographic waveforms at the 660 and 940 nm wavelengths. The ratio of the amplitude of the waveform at 660 nm to that at 940 nm is directly related to oxygenated hemoglobin percentage (fig. 1) under the conditions in which the calibration algorithm was produced. Thus, the ratios of the absorbances at 660 to those at 940 nm were correlated with actual arterial oxygenated hemoglobin percentages (%HbO2) as determined by simultaneous arterial blood sampling and measurement using a co-oximeter (fig. 2). In the case of the Ohmeda Biox 3700, the adult volunteers upon whom the oximeter calibration is based had, on the average, carboxyhemoglobin levels of 1.7% and methemoglobin levels of 0.4% (J. A. Pologue, personal communication). Under these conditions, %HbO2 is almost identical with the oxygen saturation of available hemoglobin† (total amount of hemoglobin available to bind oxygen, or SaO2).

The effect of methemoglobin on the SpO2 reading is explained by reference to the algorithm (figs. 1, 2) and the spectrophotometric absorption characteristics of the hemoglobins present (fig. 3). In our patient (PaO2 = 587 mmHg), and in the absence of dyshemoglobins, only oxygenated hemoglobin should be present, the ratio of absorbances 660/940 should be 0.43 (fig. 1), and the SpO2 should read 100%. The presence of 5% methemoglobin, which, at 660 nm, has an absorbance similar to that of reduced hemoglobin (fig. 3), causes an increase in the absorbance amplitude at 660 nm. Methemoglobin has a qualitatively similar but quantitatively smaller effect on the absorbance amplitude at 940 nm (fig. 3), so that, overall, the ratio of absorbances at 660/940 nm is increased. An increase in the ratio is interpreted as a decrease in %HbO2, and SpO2 reads less than 100%.

If the PaO2 were such that both reduced and oxygenated hemoglobin existed, the 660/940 nm ratio of absorbances would be greater than 0.43 (fig. 2). The additional presence of methemoglobin in this situation would tend to decrease the ratio towards 0.43 because, at 940 nm, methemoglobin has an absorbance greater than either reduced or oxygenated hemoglobins (fig. 3). The effect


FIG. 1. Relative plethysmographic (pulse-added) signal amplitudes, assuming the transmission intensities are similar.

FIG. 2. Algorithm relating %HbO2 as ordinate to the ratio of the plethysmographic signal amplitudes R/IR (or 660/940 nm), as abscissa.
of decreasing the ratio is to cause an increase in the
SpO₂ reading (fig. 2). As the percentage of methemo-
globin increases, the 660/940 nm ratio of absorbances
tends toward unity, and the SpO₂ approaches 80–85%
regardless of the reduced or oxyhemoglobin percent-
ages present.

The low SpO₂ readings obtained in our patient were
accurate in the presence of 2.9% methemoglobin (SpO₂
= 94%, %HbO₂ = 93.4%) and 5% methemoglobin
(SpO₂ = 92–93%, %HbO₂ = 92.8%), and well within
the accuracy (one standard deviation) of 1.5% reported
for this device in the range of %HbO₂ from 90–100%.‡
However, SpO₂ did not read SaO₂ accurately in the
presence of these levels of methemoglobin (SpO₂
= 94%, SaO₂ = 96.5%; SpO₂ = 92–93%, SaO₂
= 100%, at 2.9% and 5% methemoglobin, respectively).

Normal red cells contain less than 1% methemoglobin,
which is formed when the heme iron in hemoglobin
is oxidized from the ferrous to the ferric state. The
condition of methemoglobinemia has been defined as
existing when more than 1% of total hemoglobin is me-
themoglobin.⁷ Etiologies may be inherited or acquired.
In this patient, the most likely cause was the dapsone
therapy received by the patient prior to her admission.⁸
Other drugs and chemicals causing methemoglobin-
emia include nitrates, nitrites, chlorates, nitrobenzenes,
antimalarials, amyl nitrate, nitroglycerine, sodium ni-
troprusside, prilocaine, benzocaine, and lidocaine.⁹

This patient had a methemoglobin percentage con-
centration of 5%, actual concentration of 0.525 g/dl
(Hb 10.5 g/dl × 5%), and was not clinically cyanosed. It
has been stated⁶ that levels of methemoglobin that are
sufficient to cause cyanosis (1.5 g/dl) should not affect
the pulse oximeter readings, although no supporting
data were provided. While a study quantifying the ef-
ects of increasing percentage concentrations of me-
themoglobin on SpO₂ remains to be reported, the theoreti-
cal considerations described indicate that increasing
levels of methemoglobin will tend to cause an increase
in the SpO₂ reading at low %HbO₂ (J. A. Pologe, personal
communication).

If high percentage concentrations of methemoglobin
are present, patients may become symptomatic due to
the diminished oxygen-carrying capacity of the blood
and greater affinity of the remaining hemoglobin for
oxygen (left-shift of the oxygen-hemoglobin dissociation
curve). In this patient, the methemoglobinemia re-
solved spontaneously with cessation of the dapsone
therapy. The usual therapy for methemoglobinemia, if
indicated, is methylene blue, 1 mg/kg iv.⁷ Methylene

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Transesophageal Atrial Pacing for the Treatment of Dysrhythmias in Pediatric Surgical Patients

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Cardiac dysrhythmias occur frequently in pediatric patients with congenital heart defects during general anesthesia, and may cause hemodynamic instability.1 Urgent treatment of hemodynamically significant dysrhythmias in children during surgery heretofore has required antiarrhythmic therapy where efficacy is unpredictable. Alternatively, emergency transvenous cardiac pacing, which is highly invasive and technically difficult, is possible. We report two cases where transesophageal atrial pacing was successfully performed in pediatric surgical patients in the treatment of hemodynamically significant dysrhythmias during general anesthesia.

REPORT OF TWO CASES

Case 1. A 4-day-old, 3.1-kg neonate with Ebstein's anomaly (right ventricular dysplasia, tricuspid regurgitation, and Wolff-Parkinson-White syndrome) and pulmonary atresia was anesthetized for a right modified Blalock-Taussig (BT) shunt. At the time of the procedure, the patient had severe hypoxemia due to reduced pulmonary blood flow despite prostaglandin E1 therapy. Chest radiograph showed cardiomegaly and echocardiogram demonstrated generalized hypertrophy of both ventricles in addition to the abovementioned structural defects. The electrocardiogram showed right atrial enlargement, biventricular hypertrophy, and pre-excitation syndrome (WPW). Prior to surgery, the patient developed several episodes of a wide QRS tachycardia (heart rates 240-260 bpm associated with hypotension) which was diagnosed as a reentrant supraventricular tachycardia (SVT) with antegrade conduction along the accessory pathway. These episodes were initially treated with cardioversion; the patient was then given a loading dose of procainamide (6 mg/kg iv), and a continuous iv infusion of procainamide (90 μg·kg⁻¹·min⁻¹) was begun. Digoxin was contraindicated because of the tendency to accelerate conduction in the accessory pathway; propranolol was inadvisable because of the presence of cardiomegaly and signs of congestive heart failure; and verapamil was not administered because of the propensity of this drug to produce significant hypotension in infants. Upon achieving therapeutic levels of procainamide, the episodes of SVT were well controlled.

General anesthesia was induced and maintained with sufentanil (10 μg/kg) and pancuronium (0.1 mg/kg) iv with a FiO₂ of 1.0. Ventilation was controlled, and the patient was placed in a left lateral position for the surgical procedure. In addition to standard noninvasive monitoring, a radial artery catheter was inserted for the continuous measurement of arterial blood pressure and monitoring of arterial blood gases. A 12-French esophageal catheter (Portex Inc., Wilmington, MA) was placed in the esophagus to the point of maximal atrial waveform, as previously reported, and used for dysrhythmia detection, as well as auscultation of heart and breath sounds.2 During dissection of the pulmonary artery and surgical manipulation of the heart, sudden onset of a wide QRS tachycardia at 240 bpm associated with hypotension (mean arterial pressure = 30-35 mmHg) was observed (fig. 1A). Immediate cardioversion was indicated, but internal defibrillation required extension of the incision across the midline and external defibrillation required immediate breakdown of the sterile surgical field.

Therefore, an urgent attempt at esophageal pacing was performed. The two esophageal electrode wires at the proximal end of the esophageal catheter were attached to a pacing pulse generator, and atrial pacing with the esophageal catheter was begun at a rate of 320 bpm and a pulse width of 3 ms. The current was increased until capture was achieved at 10 mA. The pacing artifact obscured the QRS, and pacing was confirmed by monitoring the arterial pressure waveform. Atrial overdrive pacing was continued for 5 s, and then the pulse generator was turned off. We observed the immediate return of the rhythm to a sinus mechanism at a rate of 150 bpm on the ECG limb lead and the return of the arterial blood pressure to normal (fig. 1B).

A second, similar intraoperative episode of SVT was observed and successfully treated in the same manner. The remainder of the operative course was uneventful, and the patient was taken to the pediatric intensive care unit in satisfactory condition.

The postoperative course was marked by persistent respiratory failure due to congestive heart failure and right hemi-diaphragm paralysis requiring mechanical ventilation. On the 6th postoperative day, the patient underwent an uneventful fiberoptic bronchoscopy under general anesthesia to determine reversible causes of respiratory failure. At the time of bronchoscopy, the entire esophagus was directly examined through the bronchoscope, and no evidence of esophageal injury was visualized. Because of the paradoxical motion of the paralyzed right diaphragm during spontaneous breathing contributing to ventilatory dependency, the patient was scheduled for a diaphragmatic plication at