Naloxone Reversal of Nystagmus Associated with Intrathecal Morphine Administration

To the Editor—Complications associated with epidural or intrathecal opioid administration include nausea, vomiting, pruritus, and respiratory depression. Neurologic complications are rare, although vertical nystagmus after epidural opioids was reported by Fish and Rosen. We have used naloxone successfully to treat a patient in whom nystagmus was detected after intrathecal morphine administration.

A 43-yr-old, 70-kg woman was scheduled for cesarean section under spinal anesthesia. Except for previous cesarean section for severe toxemia, her medical history was unremarkable. Spinal puncture was performed with a 25-G spinal needle at the L3–L4 intervertebral space. Tetracaine HCl 10 mg and preservative-free morphine HCl 100 μg in 2.5 ml 10% glucose were injected intrathecally. The sensory block extended to T4 on both sides. Although the operation was uneventful, 3.5 h after the intrathecal injection, the patient complained of rotary vertigo. Neurologic examination showed only horizontal nystagmus. Naloxone 0.1 mg was administered intravenously without any improvement; however, after a second dose of 0.1 mg, her nystagmus disappeared. Subsequent neurologic examination revealed no abnormality.

Although many drugs can induce nystagmus, morphine was most likely the cause in our case. Although in general drug-induced nystagmus is observed with higher doses, there have been some reports of small doses of epidural opioids causing vestibular dysfunction. The nystagmus of our patient, therefore, could have been induced by the 100 μg of morphine administered intrathecally. Furthermore, the symptom was completely reversed by naloxone.

In summary, intrathecal morphine induced nystagmus that was effectively antagonized by intravenous naloxone.

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REFERENCES

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Pulse Oximeters and Onychomycosis

To the Editor—Factors affecting the accuracy of pulse oximeters include those that prevent consistent transmission of light (e.g., nail polish) through the tissue to the photodetector of the system. We present here the first description of the effect of onychomycosis on pulse oximeter readings.

A healthy young man was noticed during thyroid surgery to have a hemoglobin oxygen saturation (SpO2) of 75–77% while breathing 35% oxygen. A good pulse signal indicated that the probe (Hewlett-Packard model 78352 A) was correctly positioned on the second finger of the left hand. Clinically, the patient appeared well oxygenated, and no cause for hypoxia was evident. The probe was inspected and found to be correctly placed. However, when the probe was removed, we found a yellowish superficial layer of onychomycosis coating the nail surface. Arterial blood gases showed a normal hemoglobin oxygen saturation (SaO2) of 98%. When the probe was positioned on another finger not affected by the fungus, the SpO2 was normal (97%).

To study this further, we measured SpO2 in five volunteers with onychomycosis. With the probe on the affected finger, SpO2 was 71–84%. When the probe was placed on fingers without onychomycosis, SpO2 increased to 95–98% (SaO2 97%). Table 1 shows SpO2 values in the five volunteers on both the diseased and nondiseased fingers.

We assume that because the pulse oximeter functions by examining the difference in absorbance of two wavelengths, any factor that increases the difference in absorbance between 660 and 940 nm will cause the oximeter to falsely indicate desaturation. The yellowish gray color of the onychomycosis suggests increased absorbance at 660 nm compared to 940 nm and thus may alter the accuracy of pulse oximetry readings and indicate desaturation. In addition, onychomycosis the entire nail becomes brittle and separated from its bed, which condition also may "trick" the sensor.

Thus, nail onychomycosis may be another cause for a falsely low SpO2 reading on a pulse oximeter.

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