of coronary-subclavian steal following use of the IMA have
been reported, other patients who developed significant
perioperative morbidity and mortality following CAGB
may have been casualties of undiagnosed subclavian artery
stenosis.

Perhaps the routine preoperative checklist for patients
scheduled for CAGB with IMA should include a verifica-
tion of whether bilateral blood pressure assessment was
performed, which is similar to what is done to verify the
results of the “routine” preoperative chest x-ray. Al-
though it is clearly not the responsibility of anesthesi-
ologists to decide whether the IMA graft should be em-
ployed and under which circumstances, they can, how-
ever, help to improve the preoperative evaluation of these
patients, thus contributing to a safer operation.

In conclusion, this report describes a case involving the
perioperative diagnosis of subclavian artery stenosis in a
patient scheduled for CAGB with the use of an IMA. The
potentially lethal complication of coronary-subclavian steal
was avoided in the immediate preoperative phase because of
the finding of a 50-mmHg difference between upper
extremity blood pressures. Coronary-subclavian steal
syndrome should be suspected when myocardial ischemia
is present in the postoperative course of patients who re-
ceive IMA-coronary artery bypass grafts.

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Epidural Opioids as a Cause of Vertical Nystagmus

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There are a variety of complications associated with
intrathecal and epidural opioid administration. The most
frequent include pruritus, respiratory depression, urinary
retention, and nausea. Neurologic side effects are rare
and usually are limited to dysphoria and sedation.1 We
recently cared for a patient whose primary complaint was
visual difficulties after epidural administration of mor-
phine.

CASE REPORT

A 64-yr-old white woman was scheduled for exploratory laparotomy
and probable bowel resection. Her medical history was devoid of any
medical problems. She had no history of adverse reactions to medi-
cations and was currently receiving none. Her physical exam was nor-
mal. Laboratory studies were remarkable only for a mild anemia.

The patient received midazolam 3 mg and morphine 2 mg, 1 hr prior
to surgery. A lumbar epidural catheter was inserted preoperatively.
After induction of anesthesia with thiopental and succinylcholine,
anesthesia was maintained with isoflurane, oxygen, and vecuronium.
Prior to the conclusion of the surgical procedure, 8 mg preservative-

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free morphine was injected through the epidural catheter. Estimated blood loss was 350 ml, and 5800 ml crystalloid solution was administered.

In the postanesthesia recovery room, the patient was awake, had stable vital signs, and had no complaints of pain. She was returned to her room, and 10 h later complained of nausea, which was treated with prochlorperazine. Her first request for analgesia was 16 h postoperatively (19.5 h after epidural injection), and as per our routine, she was given 2 mg morphine subcutaneously. A second dose was requested 5 h later.

Five milligrams morphine was given epidurally approximately 1 h after the last parenteral injection. Two hours later the patient complained of visual difficulties. She claimed to be unable to read her wall clock and was dizzy when her eyes were open. Her vital signs were unremarkable. Neurologic examination showed normal motor, sensory, cerebellar, and mental status functions. Her pupils reacted normally, and extraocular muscles were intact. However, vertical nystagmus was present with the fast component upward. The patient was given 40 μg naloxone intravenously. The amplitude and frequency of the nystagmus decreased. Five minutes later a second dose of naloxone, 40 μg, was administered, and resolution of the nystagmus followed. The patient no longer felt lightheaded when her eyes were open and could read the time on the wall clock. The symptoms returned approximately 2 h later and were successfully treated with additional doses of naloxone (total 80 μg). The patient required several more doses (80-μg) 4 h later. She had no complaints after the last dose.

The next day, with the epidural catheter removed, the patient received morphine 2 mg subcutaneously every 3 h. This provided adequate analgesia and did not precipitate any visual problems. She was discharged on the 7th hospital day with no further ocular difficulties. In 5 months of follow-up visits, she has not reported any visual problems.

**DISCUSSION**

Nystagmus is a term that describes a variety of ocular disorders characterized by repetitive eye movements. The rhythm usually is regular and has slow and fast components that typically alternate. The slow phase appears to move the eye away from its intended fixation point, while the fast component is the correction movement, quickly returning the gaze to its proper location.

Nystagmus can be induced in patients with various maneuvers, including body rotation, thermal (caloric) stimulation of the ear, optokinetic stimulation (in which the subject follows a pattern of moving lines while keeping the head still—much as if watching a moving train without moving the head), postural changes, rebound from prolonged extreme gaze, and flicker induction. All involve stimulation of the vestibular-ocular reflex and can be found in normal subjects.

Disorders of the nervous system can elicit nystagmus. Lesions or inflammation of the labyrinth, and lesions of the vestibular nerve, brainstem, and cerebellum are recognized as causes. The direction of the nystagmus varies depending on the location of the lesion.

Nystagmus can be induced with medications. Alcohol, barbiturates, sedative-hypnotics, and phenytoin have been implicated. This nystagmus is typically horizontal, however.

Vertical nystagmus refers to movement in the vertical plane, and can be further divided into upbeating and downbeating types. It usually indicates disease in the pontomedullary or pontodiencephalonic area. Upbeating nystagmus can also be caused by drugs (anti-epileptics), degenerating, and Wernicke disease. Downbeating nystagmus is found in Wernicke disease, syringobulbia, Chiarl malformation, cerebellar ectopia, cerebellar degeneration, lithium carbonate use, and magnesium deficiency. Regardless of the etiology of the vertical nystagmus, patients will complain of visual disturbances and blurred or vertical oscillopsia (described as a loss of vertical hold).

We could find no reports of opioids as causes of vertical nystagmus. Meperidine has been used to treat vertigo with nystagmus. Ketamine has been reported to cause postoperative nystagmus.

Epidural administration of morphine was likely the cause of the downbeating nystagmus in the case of this patient. Confirmation of this cause was found in the reversibility of the phenomena with the opiate antagonist naloxone and in the temporal relationship to the epidural catheter injection. We feel that the nystagmus was not due to the high blood levels of the opioid that may have occurred when the epidural was injected in close temporal proximity to the subcutaneous injection. The evidence for this is the reoccurrence of symptoms 7 h after the last subcutaneous administration of morphine.

Parenteral administration of morphine did not induce nystagmus in this patient. Perhaps the CSF or blood concentrations did not exceed the concentration necessary to induce the problem.

In summary, this case demonstrates the possible relationship between opioid administered epidurally and vertical nystagmus, and the results of treatment directed at opiate antagonism.

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