Recent attention has focused on procedures less commonly associated with air embolism, such as liver transplantation and even surgical manipulation of a bone cyst. Two cases of air embolism occurring during transurethral resection of the prostate have been reported in the urologic literature, but none has previously been reported in the anesthesia literature. Although the cause and time of the entry of air into the circulation in this patient is unclear, we believe that the air entered at the end of the procedure when the Foley catheter was inserted. At this time, fluid could easily be instilled but not withdrawn. Multiple attempts at flushing what was believed to be a plugged catheter most likely introduced large amounts of air and fluid. The patient exhibited no manifestations of air embolism until the procedure had been completed and the operating room team prepared to move him from the cystoscope table. This delay in signs of embolism occurred probably because the patient was in lithotomy position, such that the air most likely accumulated in his femoral vessels until his legs were lowered.

This report represents an extreme case of air embolism. Less spectacular air emboli may occur more commonly. Smaller emboli would result in increased pulmonary vascular resistance and heart failure with associated hypoxemia, hypotension, and mental confusion. Under such circumstances, air may be shunted to the systemic circulation via a probe-patent atrial septal defect and result in a postoperative myocardial infarction or cerebral vascular accident. These symptoms can be attributed to the well-reported complications of prostatectomy, such as hypotension, volume overload, and glycine absorption. In the age group and population that usually undergoes prostatectomy, these complications are commonly attributable to underlying disease in combination with minor hypotensive episodes or volume abnormalities under anesthesia.

For this reason, we believe that the possibility of air embolism may be more common than anesthesiologists realize. We suggest that it be considered when cardiac problems arise during or after completion of this type of surgery.

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Intravenous Calcitonin Alleviates Spinal Anesthesia-induced Phantom Limb Pain

DANIEL S. FIDDLER, M.D.,* BRADLEY J. HINDMAN, M.D.†

Spinal or epidural anesthesia can temporarily exacerbate phantom limb pain (PLP) in patients with a prior lower extremity amputation.1–7 Induction of PLP with regional anesthesia has been reported in patients who had been symptom-free as long as 40 yr8 and may occur during either onset1–6 or regression7 of the block. Benzodiazepines,2 barbiturates,6 and intravenous opioids1,2,4 have been tried with variable success in the treatment of acute anesthesia-induced PLP. This report describes a patient in whom PLP, induced by spinal anesthesia, ceased abruptly with a single dose of intravenous calcitonin.

* Chief Resident in Anesthesia.
† Assistant Professor of Anesthesia.

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Address reprint requests to Dr. Fiddler: Department of Anesthesia, University of Iowa Hospitals and Clinics, Iowa City, Iowa 52242.

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removal of the needle. The needle was repositioned via the L2–3 interspace without difficulty, and 15 mg 0.5% isobaric bupivacaine was administered intrathecally. Two minutes later, the patient reported sharp intermittent pain localized to his anecotomically absent “right foot.” Over the next 3–5 min the pain resolved without treatment. Analgesia to pin prick was attained at the T4 level, and surgery proceeded without complication. The patient was admitted to the surgical intensive care unit (ICU) (per protocol for laser angioplasty procedures) 90 min after induction of spinal anesthesia. At this time the patient’s sensory level was T7, with partial return of motor function in his left leg.

Sixty minutes later, 2.5 h after induction of spinal anesthesia, the patient began to complain of paroxysms of pain localized to the stump of the right lower extremity. Over the next 2 h the pain became sharp and excruciating and was described as “coming from the right foot.” Intravenous morphine (4 mg over 2 h) allowed the patient to sleep between bursts of pain but did not change the frequency or intensity of his PLP. By 6 h after his arrival in the ICU, the patient had no detectable motor or sensory block and no discomfort in his left groin (operative site) or left foot. He described an agonizing pain, “like a lightning bolt,” in his “right heel” which occurred every few minutes. He had received no morphine over the prior 2 h.

The authors, having had a prior patient with spinal anesthesia-induced PLP, had reviewed the literature and were aware of reports describing the analgesic properties of intravenous calcitonin (see Discussion). After discussion with the patient, 100 IU salmon calcitonin (Calcimar®, Rorer Consumer Pharmaceuticals, Ft. Washington, PA) was administered intravenously over 5 min. Within 5 min of completion of the injection, the “heel pain” ceased entirely and remained absent for 12 h; over the next 12 h only two fleeting episodes occurred, neither of which was treated. Mild nausea, the only apparent complication of therapy, was treated effectively with prochlorperazine. Several days later, the patient reported an occasional twinge of PLP but of much less severity. Follow-up with the patient several months later revealed a return to baseline phantom limb status.

**DISCUSSION**

The mechanisms mediating PLP and its exacerbation by spinal or epidural anesthesia are not understood. With deafferentation, neuronal activity in the affected area of the spinal cord is characterized by spontaneous synchronous volleys of high-frequency burst activity. Melzack and Loeser proposed that neuron pools from these areas can act as pattern-generating mechanisms, but these nuclei normally are inhibited by somatic sensory input and by projections from a “central biasing mechanism” in the reticular formation. Loss of segmental afferent input, due to spinal or epidural anesthesia, might lead to decreased input to brain stem mechanisms normally exerting an inhibitory influence on sensory transmission. The release from descending inhibition is postulated to allow transmission of abnormal bursting activity, such that phantom pain becomes manifest. Although this patient experienced paresthesias during placement of the spinal needle, differences in quality and location between the paresthesias and his PLP, as well as the delayed onset of his PLP, suggest that these two phenomena were not related.

PLP induced by regional anesthesia often is extremely severe and poorly responsive to intravenous opioids. Other therapeutic options previously reported include benzodiazepines, transcutaneous electrical nerve stimulation (TENS) units, subanesthetic doses of thiopental, intrathecal or epidural opioids, and general anesthesia. Of the above alternatives, thiopental in small doses (0.5–1.5 mg/kg) and intrathecal or epidural opioids have given the best results reported to date. In 1984 Mertz reported that eight patients with severe (non–anesthesia-induced) PLP were successfully treated with a single intravenous injection of 100 IU calcitonin. Subsequently, Kessel and Worz compared the analgesic efficacy of calcitonin in ten patients with persistent or recurrent PLP to a group of ten control patients with other (non-PLP) chronic pain. After 100 IU calcitonin iv, nine of the ten patients with PLP responded with a rapid (5–30-min onset) analgesic effect that lasted from 2 h to 3 months. Only one of ten patients with other forms of chronic pain reported any analgesic effect. Side effects included nausea (40% in both groups), vomiting (10% both groups), and dysesthesias (50% in PLP group only).

Although the minimal amount of morphine administered to the patient reported here may not constitute an adequate trial of systemic opioids, the patient was comfortable and sleeping between bursts of pain. Our prior experience, and that reported in the literature, indicated that additional systemic opioids probably would be ineffective. The use of calcitonin for spinal anesthesia-induced PLP had not been previously described, but based on the reports described above, we believed a trial of intravenous calcitonin was appropriate.

Calcitonin, a polypeptide hormone consisting of 32 amino acids secreted by parafollicular cells, or “C cells,” of the thyroid gland, is believed to be involved in the regulation of calcium and phosphate metabolism. Analgesic properties were recognized in animals in 1975. Subsequent animal studies indicated that its analgesic effect is independent of opiate receptors and may be mediated via central serotoninergic pathways. Specific calcitonin receptors are present in the mammalian central nervous system (CNS); the highest concentration is in the hypothalamus. The analgesic effect of electrical stimulation of certain “analgesic centers” in the reticular formation in the rat can be blocked with serotonin antagonists. Thus, calcitonin may act via serotoninergic mechanisms to increase the activity of descending pain-suppressing systems and thereby inhibit transmission of PLP. Despite the large molecular weight of calcitonin, Giusti and co-workers found the onset and character of its analgesic effect in animals to be similar with either intravenous or intracerebroventricular administration, suggesting relatively easy access of calcitonin to the CNS.

Concerns regarding safety and possible neurotoxicity have slowed human research on intrathecal calcitonin, although it has been used in treatment of intractable pain in patients with advanced cancer. Intrathecal or epidural calcitonin produced analgesia with only minor side effects (nausea, vomiting, and decreased appetite), although long-term tolerance did occur. In the only human study to evaluate postoperative analgesia, intrathecal calcitonin (100 IU) given with a lidocaine spinal anesthetic resulted in excellent postoperative analgesia in patients undergoing lower-abdominal, extraabdominal (prostate), and lower-extremity procedures. Nausea and vomiting were reported in 7% of patients and “nervousness” in 30%.

In the operating room, thiopental is more readily available and is perhaps more appropriate as an initial intervention for PLP induced by regional anesthesia. However, when the PLP is resistant to this therapy, or when PLP occurs with the regression of the block (such as in the recovery room), intravenous calcitonin may be a therapeutic alternative. In the case reported here, calcitonin was rapidly effective and had only minor side effects (nausea). Unlike intrathecal or epidural opioids, it does not necessitate another invasive procedure. Unlike barbiturates, benzodiazepines, and systemic opioids, iv calcitonin has not been reported to produce sedation. Because calcitonin is routinely stocked by hospital pharmacies for the treatment of symptomatic Paget’s disease of bone and hypercalcemic emergencies, it is available on short notice. Although calcitonin appeared to be effective in this case of regional anesthesia-induced PLP, it is not approved by the Federal Drug Administration for this indication, and the potential for allergic reactions and hypocalcemia exists. Additional studies concerning the safety, efficacy, and analgesic effect of calcitonin in this and other pain syndromes are warranted.


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