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# Reactivation of Phantom Limb Pain after Combined Interscalene Brachial Plexus Block and General Anesthesia: Successful Treatment with Intravenous Lidocaine

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IT is well known that spinal or epidural anesthesia can lead to a recurrence or exacerbation of phantom pain.[1–7] Painless phantom phenomena of the upper extremity after brachial plexus anesthesia are not uncommon and usually are described as perception of the arm in an unusual position.[8]

We describe the reactivation of severe upper extremity phantom pain in a patient after combined interscalene brachial plexus block and general anesthesia, which, to our knowledge, has not been reported previously. This responded to a single dose of intravenous lidocaine.

## Case Report

A 62-yr-old, 106-kg woman with peripheral vascular disease was scheduled for resection of necrotic tissue and bone from her right elbow stump. The patient's medical history was significant for an atherosclerotic lesion of the right innominate artery, which had required brachial embolectomy on two occasions—both with general anesthesia. Most recently, gangrene of the right hand and forearm required above-elbow amputation, which was performed under general anesthesia. Immediately after the amputation, severe phantom pain developed, which the patient localized to the elbow and forearm (fig. 1). She received intravenous morphine for 3 days, which resulted in complete resolution of the phantom pain.

In addition to antibiotics, the patient was receiving the following medications: albuterol inhaler, aspirin, clonidine, diphenhydramine, dipyridamole, hydrochlorothiazide, lisinopril, ranitidine, acetaminophen, and phenytoin for a seizure disorder. The patient was not receiving opioids. Laboratory investigations revealed a hematocrit of 28%, blood urea nitrogen of 22 mg/dl, creatinine of 1.6 mg/dl, and normal electrolyte values.

The patient agreed to an interscalene brachial plexus block but requested a general anesthetic in addition, because of apprehension about being awake during surgery. A right interscalene brachial plexus block was performed, and paresthesias were produced in the patient's "phantom" fingers. Thirty milliliters of a mixture of 0.33% bupivicaine and 0.5% lidocaine with 5 μg/ml epinephrine was injected. General anesthesia was induced 15 min later, at which time a partial sensory and motor block had developed in the stump. Anesthesia was induced with thiopental and maintained with nitrous oxide, oxygen, and 0.2–0.6% isoflurane. The patient also received vecuronium for neuromuscular blockade and a total of 200 μg fentanyl. The op-

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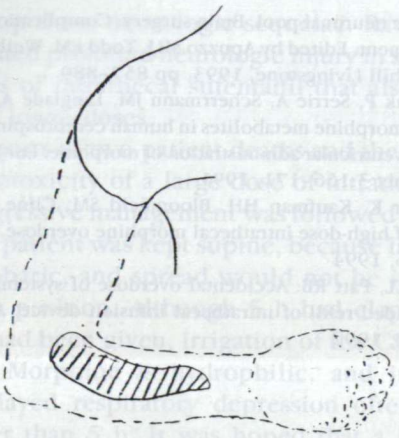

**Fig. 1. Patient drawing of phantom pain (shaded area) experienced 3 days after initial amputation and during phantom pain reactivation.**

eration lasted 90 min. After tracheal extubation, the patient was monitored in the postanesthesia recovery room. Intravenous morphine was ordered for postoperative pain.

Three and one-half hours after the block, the anesthesiologist was informed by the postanesthesia recovery room nurse that the patient was complaining of excruciating arm pain, despite having received a total of 8 mg morphine. The patient was assessed and was noted to be screaming with pain but had complete motor and sensory block of the upper extremity. The pain was localized to the same area in which she previously experienced the phantom pain, *i.e.*, right elbow and forearm (fig. 1). It was described as a severe cramping sensation. We elected to avoid further morphine administration because the patient had experienced transient hemoglobin oxygen desaturation, which appeared related to the morphine. The patient was given 70 mg intravenous lidocaine, which resulted in complete pain relief of the phantom pain within 5 min. The precise duration of the brachial plexus block was not noted. However, an assessment 5 h after the block revealed that the block was resolving. Postoperatively, she did not experience any further episodes of phantom pain. After resolution of the infection, the stump healed, and the patient made a good recovery and was discharged home.

The patient was interviewed 1 yr later, at which time she stated that she had not experienced any phantom pain since her last operation. She continued to perceive painless phantom sensation of the right upper extremity, which she was able to draw with her left hand (fig. 2). The elbow was flexed at 90° across her chest; she was unable to extend her fingers, and the phantom had telescoped somewhat into the stump.

## Discussion

There are many reports of phantom limb pain being reactivated or exacerbated after spinal or epidural anesthesia.[1–7] It can occur during the onset or regression of a block, is often severe in intensity, and usually is unresponsive to parenteral opioids. Although painless phantom limb phenomena after brachial plexus block are common,[8] to our knowledge, phantom pain occurring after brachial plexus block has not been reported. A possible reason is that amputations of the upper extremity are performed less frequently than those of the lower extremity.

In this case, it is not possible to be certain that phantom pain resulted solely from the brachial block, as the patient also received a general anesthetic. However, it is almost certain that the interscalene block played some role (either alone or by interacting with general anesthesia), because this patient had undergone previous general anesthetics without reactivation of phantom pain.

The neurologic mechanisms regarding the generation of phantom pain generally are divided into peripheral or central.[9] De Jong and Cullen suggested that phantom pain occurring during spinal anesthesia may be mediated by nociceptive impulses carried in sympathetic fibers that gain access to the spinal cord above the level of the block.[10] This hypothesis is supported by the observation that, in some patients, pain relief has resulted by extending the level of the spinal block or, in some cases, selective sympathetic block.[10]

Melzack and Loeser studied phantom phenomena in patients with high spinal cord injuries.[11] These patients experienced sensations and pain in the denervated parts of their bodies. They postulated that central neurons produce "pattern generating mechanisms" to produce these phantom images. However, to explain reactiva-

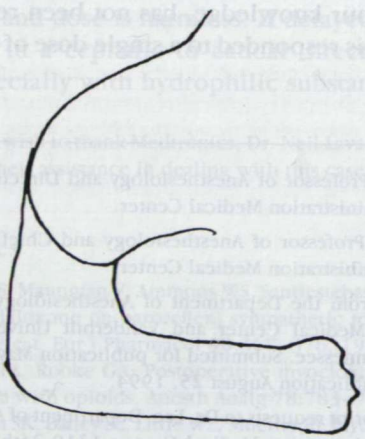

**Fig. 2. Drawing of painless phantom limb sensation experienced by patient 1 yr after amputation. The elbow was flexed at 90°, and she was unable to extend her fingers from a fist position.**

tion of phantom pain after neuraxial or somatic block, the role of afferent input must be considered.

Acute postoperative reactivation of phantom pain differs from chronic phantom pain in that it is typically severe in intensity and unresponsive to opioids. Deafferentation, or more specifically, sudden deafferentation, appears to be a prerequisite for the development of both types of phantom pain. Physical amputation is not essential for the occurrence of phantom pain. Patients who suffer brachial plexus avulsion (sudden deafferentation) without injury to the arm often report an extremely painful phantom, which is referred to as the "third arm."[12] On the other hand, patients who suffer amputations as a result of leprosy (slow deafferentation) have not been reported to experience phantom pain but may experience painless phantom sensations.[13] It is possible that the mechanism of phantom pain reactivation after spinal block is a result of rapid and complete deafferentation in a patient who already has a neuromatrix[14] predisposed to phantom pain.

Many different modalities have been described to treat phantom pain reactivation, with varying efficacy. These include thiopental in small doses,[15] fentanyl and diazepam, parenteral opioids,[1-3] calcitonin,[16] spinal opioids,[17] and transcutaneous electrical nerve stimulation.[3] Although calcitonin and thiopental have proved successful in a small number of patients, we avoided these agents because of the possibility of adverse side effects. Our patient already had experienced transient hemoglobin oxygen desaturation secondary to intravenous morphine, and it was believed that thiopental, even in a small dose, might lead to further respiratory depression. Calcitonin has been used with some degree of success to treat both chronic phantom pain[18] and one case of phantom pain reactivation after spinal anesthesia.[16] Its mechanism of action is unknown but may be mediated by enhancement of central serotonergic pathways.[19] We avoided it because of the possibility of side effects, which include nausea, vomiting, and painful dysesthesias.

The use of local anesthetics is a well established treatment modality for a variety of chronic pain conditions.[20] These agents block sodium channels in a frequency-dependent fashion and have become popular in managing neuropathic pain syndromes, such as postherpetic neuralgia,[21] diabetic neuropathy, and chronic phantom pain.[22] Both peripheral and central actions of lidocaine have been proposed depending on the particular type of neuropathic pain being studied and the dose used. Bach *et al.*[23] showed that 5 mg/kg lidocaine administered to patients with diabetic neuropathy had no effect on thermal sensation but raised the response thresholds of nociceptive reflexes. They concluded that lidocaine acted at a spinal or possibly supraspinal level in these patients. Abram and Yaksh[24] studied the effects of varying doses of lidocaine on pain behavior responses to subcutaneous formalin and thermal hyperalgesia in a nerve compression model in the rat. They showed that low-dose lidocaine (serum level 1.0 μg/ml) attenuated thermal hyperalgesia without affecting the formalin test. They concluded that lidocaine in this particular pain model acted on peripheral nerves rather than centrally.

Our patient exhibited a dramatic and sustained response within 5 min to a single small dose of intravenous lidocaine. As phantom pain reactivation episodes are self-limited, it is conceivable that the pain would have disappeared at that time without treatment. Our rationale for administering a small dose was that (1) our patient had a history of seizures and (2) it was difficult to know to what extent lidocaine and bupivicaine had been absorbed from the earlier brachial plexus block.

This case report highlights the importance of carefully assessing patients with postoperative pain, particularly those with amputations. Recovery room personnel initially were under the assumption that this patient's pain was nociceptive in nature. Only later did it become clear that this was not nociceptive pain, after a careful assessment revealed the presence of complete sensory and motor block. Such a block would obviate nociception but not centrally mediated neuropathic pain.

In summary, we report a case in which reactivation of upper extremity phantom limb pain occurred after general anesthesia with interscalene brachial plexus block, which was treated successfully with a small dose of intravenous lidocaine. We conclude that, in patients with a prior history of upper extremity phantom pain, one should be alert to the possibility of phantom pain reactivation if a brachial plexus block is administered. Lidocaine may be useful in treating a variety of neuropathic pain states and is readily available and well tolerated in small doses. Unlike thiopental, calcitonin, or benzodiazepines, lidocaine in a low dose does not cause respiratory depression, sedation, or nausea. Although further study to assess the efficacy of lidocaine for reactivation of phantom pain is indicated, it may be a useful and perhaps safer alternative than currently recommended agents.

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# Complete Electrical Failure during Cardiopulmonary Bypass

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THE consequences of electrical failure can be particularly hazardous for the cardiac surgical patient during cardiopulmonary bypass. In addition to monitoring equipment, electrocautery, operating room lights, air conditioning, and overhead paging, the cardiopulmonary bypass pump and heat exchanger will cease to function. This case report describes the management of a patient undergoing quadruple coronary artery bypass surgery during a 50-min loss of electrical power that occurred when the heart was arrested and the aorta cross-clamped and as the surgeon was performing the first distal coronary anastomosis.

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Key words: Complications: equipment failure; monitoring.

## Case Report

A 66-yr-old, 94-kg, 180-cm man was admitted through the emergency room complaining of chest pain and sustaining an acute inferior