

## Intravenous Lidocaine as Adjunctive Therapy in the Treatment of Decompression Illness

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### Abstract

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Two cases of severe decompression illness for which IV lidocaine was used as adjunctive therapy to recompression and **hyperbaric oxygen** therapy are described. The first patient demonstrated improvement only after lidocaine was added to her treatment; the second had essentially complete recovery after only a single treatment despite severe symptoms and a significant delay in presentation. These cases support the need for a controlled clinical trial of lidocaine as an adjunct to hyperbaric therapy in decompression illness.

[Cogar WB: Intravenous lidocaine as adjunctive therapy in the treatment of decompression illness. *Ann Emerg Med* February 1997;29:284-286.]

### Introduction

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The mainstay of treatment for decompression illness is recompression and **hyperbaric oxygen** therapy (HBOT). Patients who fail to respond to this treatment are frequently the most severely injured and sustain significant neurologic sequelae.<sup>1</sup> To date, no effective adjunctive therapies have been identified, although some studies have supported the use of lidocaine in neurologic injuries.<sup>2-5</sup> The cases described here suggest a beneficial effect of IV lidocaine when used as an adjunct to HBOT in severe decompression illness.

### Case Report

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**Patient 1** In January 1991, a 31-year-old woman presented on an emergency basis to the recompression chamber of the United States Navy (USN) Ship Repair Facility, Guam, after a

rapid, poorly controlled ascent during a recreational scuba dive. She complained of severe headache, vomiting, and back pain immediately on surfacing, and she appeared disoriented and anxious. The dive profile could not be accurately assessed because of a malfunctioning depth gauge but reportedly exceeded 60 m, with a bottom time of about 25 minutes followed by a rapid ascent after expiration of air supply. Past medical history was unremarkable with no repetitive dives, recent travel, ethanol use, or prior decompression illness. The patient was transported directly to the chamber from the dive site, arriving within 20 minutes of symptom onset. Therapy en route included administration of oxygen through a nasal cannula and placement of the patient in the Trendelenburg position. The preliminary diagnosis was an arterial gas embolism, and the patient underwent immediate recompression to 165 feet seawater (FSW) in accordance with USN Treatment Table 6A.<sup>6</sup> All presenting symptoms except for slight residual low back pain, resolved during descent, and initial neurologic examination at depth revealed no focal deficits.

On beginning chamber ascent from 140 to 120 FSW, the patient experienced recurrence of symptoms, including “stocking-glove” paresthesias of both legs, left leg weakness, and increased low back pain. She was returned to 165 FSW without relief and continued to note worsening symptoms. Neurologic examination revealed decreased sensation from the mid thigh distally, 4/5 motor strength, and 3/4 deep tendon reflexes (DTRs) on the left leg. USN Treatment Table 4<sup>6</sup> was initiated for symptoms not relieved after 30 minutes at 165 FSW. After 2.5 hours at 165 FSW, clinical symptoms continued to progress, and chamber ascent to 60 FSW was made to institute oxygen therapy.

The patient began standard hyperbaric treatment, consisting of 25 minutes on 100% oxygen, followed by a 5-minute air break. Additional measures included IV hydration with lactated Ringer's solution at 125 mL/hour and 650 mg acetylsalicylic acid every 4 hours as needed for headache. A Foley catheter was placed after the patient complained of “fullness” and was unable to void spontaneously. Despite this treatment, her clinical condition continued to deteriorate, with worsening neurologic deficits. Because of the severity of the deficits and failure to note any improvement, treatment was continued on USN Treatment Table 7.<sup>6</sup> After 12 oxygen-breathing periods at 60 FSW, neurologic examination revealed severe deficits: bilateral lower extremities had 2/5 motor strength, 3/4 DTRs with positive clonus (left > right), neurogenic bladder, decreased rectal sphincter tone, and sensory deficits from T8 distally. Standard oxygen therapy was continued for an additional 20 hours without effect.

The Navy Experimental Diving Unit was consulted, and therapeutic alternatives were discussed, including the use of lidocaine, on the basis of animal studies. This experimental approach was explained to the patient, and informed consent was obtained. Lidocaine therapy commenced with a 1 mg/kg bolus followed by continuous infusion at 2 mg/minute. Approximately 5 minutes after the bolus was infused, the patient complained of a “burning sensation” bilaterally. Within 30 minutes, strength improved in both legs (3/5), although with rapid fatigue. Oxygen-breathing treatments continued with 100% oxygen but were reduced in frequency and duration because of mild pulmonary oxygen toxicity and patient intolerance. The patient gradually improved as chamber ascent continued over the next 28 hours. The lidocaine infusion was continued for 24 hours; the patient then displayed apprehension, nervousness, and paranoid ideation, believing she was worsening and that we had returned to 165 FSW. These symptoms were consistent with lidocaine toxicity and rapidly resolved with discontinuation of the infusion.

Initial treatment ended after 62 hours. Neurologic examination demonstrated 4/5 strength and 4/5 upward Babinski bilaterally, paresthesias from T8 distally, decreased DTRs in the left leg, and resolution of clonus. The patient was transferred to Guam Memorial Hospital for continued inpatient care. She subsequently underwent 10 treatments over a 14-day course in accordance with USN Treatment Table 6. At that time, she was ambulatory, with mild ataxia and weakness of hip flexors, mild paresthesias from T10 distally, and occasional neurogenic bladder. Treatments were discontinued after the last three retreatments failed to produce improvement. At 3-month follow-up, the patient had essentially recovered, complaining only of episodic mild ataxia

associated with rapid positional changes. She subsequently moved home to the United States and was lost to further follow-up.

**Patient 2** In March 1993, a Japanese man in his early twenties was evacuated from Tinian (an island near Guam) 36 hours after sustaining neurologic deficits in an 80-m scuba dive. Exact history and dive profile were unobtainable because the patient spoke only Japanese and interpreters were not available. He had undergone three “in-water” recompression attempts by his diving partner, the first a return to 80 m, a second dive to 60 m, and finally a 40-m dive with safety stops. Despite these efforts, the patient’s condition had deteriorated, with decreased strength in the legs, and the patient was transported to Guam. Treatment en route consisted of oxygen administered through a nonrebreather mask.

On arrival, the patient had 0/5 motor strength bilaterally in the legs, sensory deficits from about T10 distally, and neurogenic bladder. Arm strength and sensation were intact. Vital signs were stable. IV administration of lactated Ringer’s solution was begun at 125 mL/hour, and the patient was recompressed to 60 FSW in accordance with USN Treatment Table 4.<sup>6</sup> The diving partner was asymptomatic but underwent recompression with the patient as a precautionary measure and to assist with attempts at communication.

On the basis of experience with the first patient and prior experience with poor outcomes after delayed presentations, we began adjunctive lidocaine therapy with recompression, using a 1-mg/kg bolus followed by a 2-mg/minute infusion over the first 24 hours. The patient began to demonstrate clinical improvement shortly after the treatment began, rapidly regaining some degree of motor function but retaining paresthesias. He continued to make slow but gradual improvement, and, on the basis of the severity of his presenting symptoms, was switched to a USN Treatment Table 7 protocol.<sup>6</sup> Treatment was continued for a total of 53 hours. At the end of initial treatment, the patient had essentially regained normal motor function and was able to walk from the chamber without assistance. He was admitted to Guam Memorial Hospital for 24 hours’ observation, after which he was returned to Japan for further care and was lost to follow-up.

## Discussion

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The original rationale for recompression and HBOT in decompression illness was to directly relieve the formation of nitrogen bubbles. Although the exact pathophysiology of decompression illness remains unclear, current studies support activation of the coagulation and complement systems, with promotion of interstitial edema and microvascular sludging with progressive local ischemia as contributing mechanisms.<sup>7,8</sup>

Lidocaine has been shown to reverse many of these effects<sup>2-5,9</sup>, to reduce intracranial hypertension associated with arterial gas embolism<sup>3</sup>, to increase spinal cord blood flow<sup>9</sup>, and to preserve nerve conduction in isolated nerves.<sup>10</sup> Conflicting results have been obtained in studies evaluating recovery of somatosensory evoked potentials after experimental air embolism.<sup>2,11</sup> Lidocaine is also known to have a membrane-stabilizing effect, blocking sodium channels and inhibiting cation leakage across cell membranes.

In a previous case report, Drewry and Gorman<sup>12</sup> described complete recovery after administration of IV lidocaine in a patient who failed to improve with HBOT alone. A pilot study by the same authors used lidocaine infusion for patients who demonstrated no recovery in two consecutive HBOT sessions.<sup>13</sup> Complete recovery was achieved in 5 (29%) of 17 patients and improvement in 8 (47%). No prospective, randomized study has been published.

These cases provide further empiric evidence of the efficacy of IV lidocaine when used in conjunction with conventional HBOT. Despite standard recompression and **hyperbaric oxygen**

therapy, certain patients fail to recover completely. In a retrospective study, Ball<sup>1</sup> demonstrated a strong correlation between initial severity and posttreatment morbidity, and an extensive literature review revealed complete recovery rates between 0% and 17% in severe decompression illness. Both of the cases reported here qualified as severe by severity scale index.<sup>1</sup> Patient 1 demonstrated improvement only after lidocaine was added. For patient 2, the severity of symptoms and the delay before presentation make the remarkable recovery noted with only one HBOT session highly unlikely. A prospective, randomized study comparing standard HBOT with HBOT plus lidocaine should be conducted to better evaluate the therapeutic benefit of lidocaine in decompression illness.

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## Articles with References to this Article

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**This article is referenced by these articles:**

### **Cerebral arterial gas embolism by helium: An unusual case successfully treated with hyperbaric oxygen and lidocaine**

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ABSTRACT

FULL TEXT