UNCONSCIOUS DIVER ON THE SURFACE: DIAGNOSIS AND TREATMENT DILEMMA IN A US NAVY CLOSED-CIRCUIT, MIXED GAS UBA DIVER

Muller, MS1, Ervin, R1, Stansberry, A1, Davis, LL1
1US Navy EOD Mobile Unit SIX, Charleston, SC
2Roper Hospital, Dept of Hyperbaric and Undersea Medicine, Charleston, SC

Introduction: Loss of consciousness upon surfacing in a diver is a presentation nearly synonymous with arterial gas embolism (AGE) and the need for rapid, hyperbaric oxygen (HBO) recompression treatment. The US Navy Diving Manual (Rev 5) states, “As a basic rule, any diver who has obtained a breath of compressed gas from any source at depth, ... who surfaces unconscious, loses consciousness, or has any obvious neurological symptoms within 15 minutes of reaching the surface, must be assumed to be suffering from arterial gas embolism. Recompression treatment shall be started immediately.”

However, in diving operations utilizing a closed-circuit underwater breathing apparatus (UBA), global cerebral oxygen deficiency may also cause loss of consciousness with little to no warning. When this hypoxia-ischemia insult is extended, neurological signs may persist well past initial recovery. Determining the etiology of diver impairment and making a diagnosis to treat with recompression may be difficult. Recent investigational work on the neuroprotective role of hyperbaric oxygen following hypoxia-ischemia insult further complicates the treatment decision.

Case Report: An US Navy diver presented with loss of consciousness upon surfacing following a 42-foot, 16-minute recompression dive utilizing the MK-16 MOD 1 closed-circuit, mixed gas UBA. The MK-16 MOD 1 is designed to maintain a constant partial pressure of oxygen (pO2), and transitions on ascent from 0.7 to 1.3 ata at 33 feet. Due to operational tasking, the diver was diving alone, tethered to a surface witness buoy (“single marked diver”).

Dive #1 – LS 10:28 / RS 10:35 / BT :08 / TDT :02 / TTD : 10 / Depth 42 / T/S - 56/27 RS A
Dive #2 – LS 10:49 / RS 11:07 / BT : 16 / TDT :02 / TTD : 18 / Depth 42

Upon reaching the surface at the end of dive #2, the diver appeared to be conscious, but immediately re-submerged. The dive boat motored to his witness buoy, where line pull signals on the diver’s marking line were not returned. Recovery of the diver was initiated, with an estimated 45% of line recovered prior to diver retrieval.

On surface, the unconscious diver was reportedly apneic, cyanotic, with blood coming from nose and mouth. Spontaneous breathing returned following two rescue breaths, and consciousness was regained in less than two minutes. Significant agitation, disorientation, and visual impairment improved gradually throughout a 30-min transit. At the treatment facility, the diver presented alert and oriented, with evidence of sinus and middle-ear barotrauma and residual gross neurologic deficits consisting of impaired visual acuity, cerebellar dysfunction and short-term memory impairment. Laboratory, chest radiograph and head CT were normal with the exception of blood within middle ear and paranasal sinuses. During an on-scene debrief with the dive supervisor, it was discovered that pO2 values displayed on the UBA appeared abnormal on diver/UBA recovery. A diagnosis of hypoxic/anoxic brain injury was considered more likely than AGE. The diver was admitted for 24-hour observation and remained on surface oxygen, but was not treated with recompression therapy. Following discharge, subsequent MRI and neurologic evaluations were without abnormality and he was returned to duty following resolution of barotrauma injuries. Evaluation of the UBA involved in this incident determined a malfunction of the oxygen add valve likely resulting from water intrusion into a cable connection with the primary electronics assembly.

Closed-Circuit Diving Risks: CNS oxygen toxicity, hypercapnia and hypoxia are all well-known risks in closed-circuit, mixed-gas diving operations, particularly during descent and ascent phases. To aid the diver in monitoring his air supply, the MK-16 MOD 1 has a warning light mounted in the diver’s field of vision and a umbilical-mounted, digital secondary display. Diver’s are trained to monitor both primary and secondary display frequently throughout the dive, with particular emphasis placed on transition phases. Unfortunately, the insidious nature of hypoxia may lead to confusion, task fixation and visual field loss that undermines diver situational awareness and can lead to poor judgment and decision-making.

In this incident, the diver later recalled seeing an abnormal secondary display pO2 value (0.20, or approx 9% O2 surface equiv) while on the bottom. He manually added oxygen to nominal levels and returned to his task. Upon completion, he again discovered an abnormal secondary display value, manually added oxygen, and aborted the dive. On ascent, the diver called his buddy marker line, and thus, it is unlikely he monitored his secondary display or added oxygen on ascent.

He recalls approaching the surface, but little else until immediately prior to arrival at the treatment facility.

Diagnosis of Hypoxic/Anoxic Brain Injury. While it is taught that hypoxic casualties will quickly regain consciousness when exposed to fresh air, this incident underscores that significant neurologic impairment may persist for an extended period. Familiarity with global cerebral hypoxia symptoms improves differentiation from AGE. Areas of the brain particularly vulnerable to lack of oxygen include the Purkinje’s fibers of the cerebellum and the parieto-occipital cortex, which play a large role in coordination and movement, and the hippocampus, which is one of the major structures responsible for memory consolidation. Typical presentation of hypoxic/anoxic brain injury includes lack of coordination, visual problems, and cognitive deficits such as short-term memory loss and poor reasoning or judgment. Laternalizing or focal extremity signs common in decompression sickness and AGE would be unexpected following global cerebral hypoxia insult.

Treatment with HBO. Hypoxia is well-established as a primary factor inducing neuronal cell injury and death following hypoxic-ischemic injury, acting via disabled energy metabolism and activation of several pathogenic cascades. The great number of hypoxic-ischemic injuries worldwide has led to the development of a number of treatment strategies, all achieving limited clinical success. However, there is increasing experimental evidence documenting the neuroprotective mechanisms of HBO following hypoxic-ischemic injury, including improvement of brain metabolism, reduction of blood-brain barrier permeability and brain edema, decreasing intracranial pressure and inflammatory response, and prevention of apoptotic cell death. Importantly, as with thrombolytic therapy, critical factors determining the effectiveness of HBO in hypoxic-ischemic injury appear to be the therapeutic window for treatment and dosing regimen. While promising, there are insufficient clinical trials, and a further understanding of the neuroprotective mechanisms is needed before HBO can be considered an established treatment following either global hypoxia-anoxia or focal hypoxic-ischemic injury.

Conclusion. Hypoxic brain injury is a risk in closed-circuit UBA diving and can complicate diagnosis and management of diving casualties. The diving medical officer must understand the potential benefits and harm that accompany the use of HBO recompression therapy with this injury.

“As a basic rule, any diver who has obtained a breath of compressed gas from any source at depth, ..., who surfaces unconscious, loses consciousness, or has any obvious neurological symptoms within 10 minutes of reaching the surface, must be assumed to be suffering from arterial gas embolism. Recompression treatment shall be started immediately”
Hypoxic Brain Injury
Diving Mishap Summary

- Closed-circuit, mixed-gas UBA diver deployed as “single, marked” diver
- Loses consciousness upon surfacing, re-submerges, recovered via marking line (~40’)
- Rapid return of spontaneous breathing, consciousness w/ rescue breaths
- Slowly improving neurologic symptoms (hours)
- Differential diagnosis
- Role of HBO in hypoxic-anoxic brain injury