# An Unconscious Diver with Pulmonary Abnormalities: Problems Associated with Closed Circuit Underwater Breathing Apparatus

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## Abstract

Closed circuit underwater breathing apparatus (UBA) have gained popularity in recreational diving. Closed circuit UBAs carry a unique set of risks to the diver. We present the case of a diver who lost consciousness while diving and had pulmonary abnormalities. The case is illustrative of the diving related problems associated with closed circuit UBA that a physician may be faced with.

**Learning Objectives**

1. Review the history and use of a closed circuit diving apparatus in civilian and military medicine.
2. Learn the major complications of diving on a closed circuit breathing apparatus with a high fraction of inspired oxygen.
3. Review the case study of a diving casualty sustained while diving on a closed circuit dive apparatus.

### Introduction

Closed circuit underwater breathing apparatus (UBA) (re-breathers) were first tested in 1879 and have been widely used since World War II.[1] Though primarily used in military applications, UBAs have gained popularity in civilian diving.[2] There are several re-breather units available, most modeled after the Lambertson Amphibious Respiratory Unit (LARU), named for its inventor, Doctor Christopher Lamberston.[3]

Though re-breathers may accommodate a variety of gas mixtures, the simplest closed circuit UBA is an oxygen ($O_2$) re-breather that utilizes 100% $O_2$ as its supply gas. All re-breathers have in common a closed circuit breathing loop, a small high pressured cylinder gas supply, and the ability to remove carbon dioxide ($CO_2$) via chemical scrubbing.[4] The absence of bubbles resulting from the re-breathing system minimizes enemy detection and disruption of surrounding aquatic life, making it popular with operational Special Forces as well as recreational divers. Its light-weight design is another popular feature, and the fact that no inert gas is taken up by the diver minimizes the risk of decompression sickness.[1]

Re-breathers are used in less than 1% of recreational dives but account for 4.5% of diving related deaths in that community.[2] Along with the benefits, re-breather systems carry a set of unique risks for the diver mostly from high partial pressures of $O_2$, potential for increased levels of $CO_2$, and the presence of a caustic $CO_2$ removal substance. We present the case of a military diver who suffered a dive injury while on a closed circuit re-breather that illustrates the unique problems that may be encountered in closed circuit UBA.

### Case Presentation

A 25 year old Navy SEAL (Sea Air and Land) presented after loss of consciousness and witnessed convulsions during a training dive with a Draeger LAR V re-breather apparatus utilizing 100% $O_2$ as the supply gas. The dive profile was part of an extensive five hour training evolution utilizing Navy combat swimmer multi dive tables (CSMD) and this dive consisted of an approximate 30 minute bottom time with two excursions below 40 feet of seawater (fsw). During the second excursion he developed euphoria, facial twitching, and trismus. He made an assisted ascent while attempting to draw from his regulator with an involuntarily clenched mandible. At the surface he was determined to be unconscious by his dive partner and his facemask was removed. The on scene Corpsman witnessed convulsive activity, and on initial evaluation the patient was pulseless and unresponsive. Resuscitation was started immediately with an immediate return of pulse and breathing and expectorated pink, frothy sputum was noted. Concerns for arterial gas embolism prompted an evacuation to a hyperbaric chamber and recompression therapy with a Treatment Table Six from the Navy Dive Manual. The patient had a gradual

improvement of his mental status from the time of his initial resuscitation, but during recompression treatment complained of continued significant substernal chest pain and dyspnea. Subsequently, he was evacuated via air ambulance maintaining an altitude of < 500 feet to an ICU. On presentation to the ICU he was somnolent but arousable.

Physical examination showed petechiae on his upper eyelids, dried peri-oral blood, tachypnea, decreased breath sounds at the lung bases, and mild expiratory wheezing. Neurological exam showed no focal deficits. Lab values were notable for an ABG (pH 7.24, $PCO_2$ 53 mmHg, and $PO_2$ 70mmHg) and a leukocytosis of $9.4x10^3/mm^3$ with 61% bands. Basic chemistry analysis, cardiac enzymes, and liver function tests were normal. Initial chest radiograph (Figure 1) and CT scan demonstrated diffuse alveolar infiltrates with dense bilateral lower lobe consolidation. An echocardiogram with bubble study revealed a patent foramen ovale (PFO); an MRI and EEG were both normal. Antibiotics and diuretics were started with improvement in his dyspnea and chest radiograph over the next three days. Outpatient follow-up 10 days after presentation revealed resolution of shortness of breath, normal vital signs, normal detailed neurological exam and a normal chest radiograph (Figure 2). Pulmonary function testing revealed a mild obstructive pattern with a positive bronchodilator response and normal diffusion capacity for carbon monoxide.

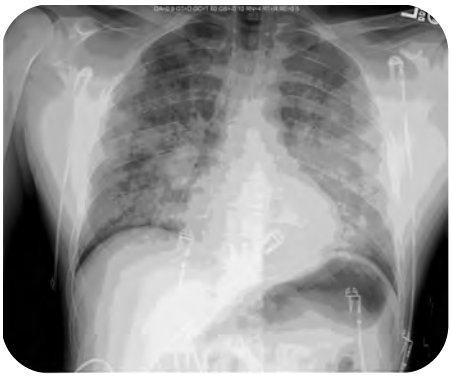

Figure 1

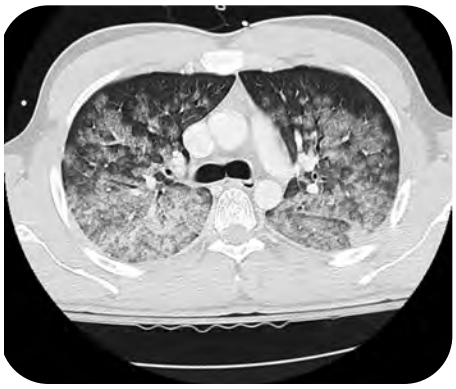

Figure 2

**Discussion - Re-breathers**

There are essentially two forms of self contained underwater breathing apparatus (SCUBA). Open circuit apparatus utilize compressed gas from a cylinder that is then exhaled into the surrounding environment. Closed circuit UBAs (re-breathers) use a closed breathing circuit that allows exhaled air to be "scrubbed" free of $CO_2$ by absorbent materials (soda-lime). This $CO_2$ free air is then returned to the diver to meet their metabolic demands. The most common gas used for re-breathing circuits is $O_2$. Generally a small high-pressure gas cylinder inflates a breathing bag (counter lung) that is in circuit with the scrubbing system. As the $O_2$ is metabolically consumed the counter lung deflates and a pressure regulated demand valve opens a high pressure bottle of $O_2$, filling the bag. Through purging techniques, a fraction of inspired $O_2$ > 74% can be achieved.[4] Re-breathers offer the diver a lightweight design that improves stealth and, by limiting inert gas uptake, decrease the risk of decompression sickness. However re-breathers add complexity and unique risks to diving. This case illustrates some of the potential injuries that may be associated with re-breather systems.

**Hyperbaric Oxygen related Seizures**

In $O_2$ re-breather systems the partial pressure of $O_2$ is increased as the diver descends in the water column. For every 33 fsw, atmospheric pressure is doubled. At a depth of 33 fsw the partial pressure of 100% $O_2$ will be 1520mmHg. High partial pressures of $O_2$ are associated with seizures and diving limits for 100% $O_2$ have been established through extensive work by Butler largely based on seizure risk.[5,6] It has been demonstrated that there is wide inter- and intra-individual variability in central nervous system (CNS) oxygen toxicity and that one's risk can change with exertion and environmental factors.[6] Symptoms of CNS hyperbaric oxygen (HBO) toxicity range from tunnel vision, tinnitus, nausea, irritability, and dizziness to frank convulsions. Though many divers will have symptoms prior to convulsions, convulsions may be the first symptom manifested. The seizure itself is generally self-limited and is not considered to be harmful, but in the underwater environment it can be extremely hazardous.[7] Treatment for in-water seizures is to reduce the partial pressure of $O_2$ by a slow ascent rate once the seizure has stopped. If an HBO seizure occurs in a hyperbaric chamber the diver or patient is removed from the high $O_2$ fraction atmosphere (i.e., switch the breathing gas to air) and the ambient pressure is decreased appropriately.

The mechanism of HBO seizures is not fully known but is likely related to $O_2$ generated free radicals and endothelially derived nitric oxide synthase (eNOS).[7,8] When superoxide is generated ($\cdot O_2$-), nitric oxide (NO) is inactivated to hydrogen peroxide ($H_2O_2$) and peroxynitrite (ONOO-). This causes a decrease in available NO and de-

creases cerebral blood flow (CBF).[9] With prolonged HBO exposure, eNOS is upregulated and there is a resultant increase in CBF that precedes the onset of seizure.[7,9,10] This case is consistent with oxygen toxicity seizure in both patient presentation and history.

### Arterial Gas Embolism

An additional concern in the unconscious diver is barotrauma and arterial gas embolism (AGE). Boyle's law states that as pressure is reduced, volume will increase proportionally. Ascent from 33 feet of sea water to the surface would be associated with a theoretical doubling of gas volume. If a diver is unable to exhale during ascent or if there are structural lung abnormalities that impede the normal air flow, pulmonary over-inflation and barotrauma may result.[11] This could manifest as pneumothorax, pneumomedastinum, subcutaneous emphysema or the escape of gas into the arterial system resulting in AGE. The large bubbles produced obstruct blood flow to vital organs, including the CNS. The most common presenting symptoms are changes in cognition and unconsciousness, but seizures and focal deficits may be present. AGE demands rapid assessment and treatment. The definitive treatment for AGE is recompression and HBO. Recompression therapy for AGE in divers has been proven to be successful when performed expeditiously.[12] The patient in this case was unconscious on the surface and appropriately received immediate recompression therapy; he also complained of chest pain that may be consistent with pneumomediastinum, though none was evident on subsequent radiographic imaging.

### Hypoxemia and Hypercarbia

Re-breathers also present a risk for hypoxemia and hypercarbia. Though on $O_2$ re-breather systems the diver is delivered 100% $O_2$, it is possible for the $O_2$ source to be metabolically used on long dives. As the diver is at depth it is unlikely that a low fraction of inspired $O_2$ will become symptomatic, but as the diver ascends and the partial pressure of $O_2$ is decreased there is a potential for symptomatic hypoxia.[4] The clinical presentation expected would be a diver who loses consciousness or suffers a seizure while surfacing. This presentation should be distinguished from unconsciousness at depth related to seizure or hypercarbia, but may be difficult to separate from AGE.

In re-breathers, $CO_2$ is removed from the breathing circuit by a canister containing a $CO_2$ absorbent material, most commonly sodalime, and there are several potential failures in this system. Sodalime's effectiveness may be expended on long dives, compromised in the presence of moisture, reduced through the formation of channels within a canister decreasing the effective surface area, or when a diver's workload exertion creates a high respiratory rate and a "blow by effect" where the exhaled air does not have enough dwell time to allow effective $CO_2$ removal. In addition, the combination of hyperoxia and exercise predispose some divers to $CO_2$ retention that is largely variable and is commonly not recognized.[13] The symptoms of hypercarbia are progressive, and as the concentration of $CO_2$ increases the onset of symptoms is gradual. The first symptom is usually an increase in the rate and volume of breathing as the respiratory system attempts to compensate for the buildup of $CO_2$ in the blood. As hypercarbia worsens hyperventilation evolves to frank dyspnea, and a severe throbbing headache may ensue in a portion of victims. Finally, further $CO_2$ accumulation leads to altered mental status and unconsciousness.[14]

The history and onset of symptoms in this case were rapid and devastating. In hypercarbia the initial hyperventilation is often masked by the increased workload inherent in a strenuous dive. It is not typical, however, for the diver to remain asymptomatic until a loss of consciousness. It should be noted that an increase in the partial pressure of $CO_2$ has been shown to increase susceptibility to an HBO seizure, but there is no evidence to support this in this case.[7] The primary neurologic insult of HBO seizure with trismus and unconsciousness at depth predisposed the diver to have pulmonary complications. These complications have a differential to include near drowning, aspiration of a caustic substance, pulmonary edema of immersion, and negative pressure pulmonary edema.

### Near Drowning and Aspiration

Near drowning (ND) refers to aspiration of a liquid (most commonly water) that does not result in immediate death, where drowning refers to death by suffocation after immersion. Civilian SCUBA diving related drowning accounts for (53%) of diving related deaths but are <1% of the more than 5,600 drowning deaths in the United States alone.[2] At least 85% percent of drowning is associated with aspiration of contents into the lungs, where 3 to 15% may be not be associated with fluid filled lungs (dry drowning).[15] The early (< 4h) pulmonary effects of ND include loss of surfactant, pneumonitis, and decreased compliance leading to shunt physiology and hypoxemia. Bronchospasm and pneumothorax have also been described in the ND victim.[14]

In this case the diver was at high risk for aspirating the $CO_2$ scrubbing medium. In closed circuit breathing a chemical agent is used to eliminate $CO_2$. The most commonly used substance is soda-lime, which is a combination of calcium hydroxide ($Ca(OH)_2$), sodium hydroxide (NaOH), and potassium hydroxide (KOH). Optimally $CO_2$ combines with the hydroxides and creates an anhydride,

water, and heat. Generally, the soda-lime is contained in a canister to prevent particulate matter from entering the breathing loop. However, when the hydroxides comes in contact with water it creates a caustic alkali liquid, sometimes referred to as a "caustic cocktail" which can escape into the breathing loop.[15] Alkalis produce liquefactive necrosis that can cause deeper tissue penetration potentially more significant than thermal burns.[16] The major anticipated respiratory injury is to the upper and lower airways and include laryngeal edema and airflow obstruction.[16] There was no evidence of the phenomenon in this case.

### Pulmonary Edema of Immersion

Pulmonary edema of immersion has been described in surface swimming and in SCUBA diving. When associated with surface swimming it has been referred to as swimming induced pulmonary edema (SIPE). SIPE is a form of exertionally related non-cardiogenic pulmonary edema as a result of pulmonary capillary stress failure.[17,18] In upright immersion external pressure differentials increase intra-thoracic fluid volume that may combine with an increased cardiac after load and exercise with increased in pulmonary artery pressure.[19] These forces then increase the pulmonary trans-capillary pressure leading to stress failure of the thin walled pulmonary capillaries. SIPE is self-limiting and generally resolves within 24 h of presentation. Treatment for SIPE is supportive and may include positive pressure breathing and beta agonists to enhance alveolar clearance of fluid.[20]

### Negative Pressure Pulmonary Edema

Negative pressure pulmonary edema (NPPE) has been reported in a large array of conditions associated with upper airway obstruction.[21] As described by Louis, obstruction of the upper airway is associated with the generation of large negative and positive airway pressure. Large swings in negative intra-thoracic pressure increase venous return and combine with increased after-load and left-ventricular dysfunction to increase capillary pressure. The large positive airway pressure associated with upper airway obstruction prevent alveolar edema formation based on Starling forces. Within minutes after the upper airway obstruction is relieved the increased capillary pressures allow transudation of fluid into the alveolar space, or may be associated with capillary stress failure and leakage of large molecular weight proteins and red blood cells. The treatment for NPPE is largely supportive and may require mechanical ventilation. Generally there is significant clinical and radiographic improvement within 24 hours.

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### Summary

This case illustrates potential dive related injuries associated with oxygen re-breather systems. The patient suffered from hyperbaric oxygen related seizure and likely had negative pressure pulmonary edema and aspiration of seawater. Given the concern for AGE the patient appropriately received immediate recompression therapy and responded to supportive care for his lung injury. On follow-up at one year he had normalization of his lung function and a medical return to dive status.

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