CASE REPORT

Arterial gas embolism in a diver using a closed-circuit oxygen rebreathing diving apparatus

S. CARSTAIRS

1st Marine Expeditionary Force, Camp Pendleton, California

Carstairs S. Arterial gas embolism in a diver using a closed-circuit oxygen rebreathing diving apparatus. Undersea Hyper Med 2001; 28(4):229–231.—A U.S. Marine Corps Reconnaissance diver suffered arterial gas embolism (AGE) while using a closed-circuit oxygen rebreathing scuba apparatus; there are few, if any, reported occurrences of AGE due to gases other than air. The high oxygen fraction of the diver's inspired gas may have contributed to his rapid recovery once recompression therapy was initiated.

gas embolism, pulmonary barotrauma, oxygen rebreather, recompression therapy, underwater breating apparatus

Pulmonary barotrauma (PBT) is a well-known hazard of compressed-air diving, primarily occurring during ascent from depth. PBT is a corollary of Boyle's law, which states that pressure and volume are inversely related; thus, as a diver ascends, intrapulmonary gas expands and can cause lung rupture and extrapulmonary extravasation of the gas. This extrapulmonary gas can manifest itself in a number of ways, including pneumothorax, mediastinal or subcutaneous emphysema, and arterial gas embolism (AGE), the latter being the most potentially serious. While the breathing medium in most cases of PBT is compressed air, it can also potentially be caused by any gas or gas mixture. The presentation and clinical course of a diver suffering from PBT due to gases other than air may, however, be quite different from that due to compressed air. Although there is one recent case report of mediastinal emphysema in a diver using an oxygen rebreather (1), there are few (if any) cases in the literature of gas embolism in a diver breathing a hyperoxic gas mixture.

Presented here is the case of a healthy U.S. Marine Corps diver who suffered cerebral AGE after diving using closed-circuit O_2 rebreathing equipment, and who experienced quite rapid clinical improvement with recompression therapy.

CASE REPORT

A 35-yr-old U.S. Marine Corps Reconnaissance diver was performing a training dive using a U.S. Navy MK-25 (Draeger LAR V) closed-circuit O_2 rebreathing apparatus in a harbor on the southern California coast. The dive profile was a 1,000-m compass dive; maximum depth attained was approximately 7 meters of seawater (msw). The diver had completed about 450 m of the swim when he began to feel "dizzy and nauseated". He completed another 150 m, then surfaced in a controlled manner with his diving partner. Upon reaching the surface, he notified the diving supervisor of his condition and was removed from the water. The diver denied breath-holding or skip-breathing while submerged, but stated that he had been having problems with his buoyancy compensator and had been unable to maintain a constant depth in the water column, varying between approximately 2 and 7 msw. Within several minutes after surfacing, his symptoms began to worsen markedly. Neurologic examination revealed confusion, memory loss (the diver was unable to remember his name, occupation, location, or the date), and marked cerebellar abnormalities (bilateral nystagmus, severe truncal and limb ataxia, positive Romberg sign). Vital signs were normal and the remainder of the neurologic examination was normal, with no evidence of motor weakness or sensory deficit.

A diagnosis of cerebral AGE was made and the diver commenced treatment on a U.S. Navy treatment table 6 (TT6). Complete symptomatic resolution occurred immediately upon arrival at a depth of 18 m; repeat neurologic examination in the chamber revealed normal memory, mentation, and cerebellar function. The treatment table was continued to completion, with one extension at 18 m. The patient remained asymptomatic for the remainder of the treatment. The patient was observed overnight, with no recurrence of symptoms.

DISCUSSION

Arterial gas embolism and other sequelae of PBT are

well-recognized complications of compressed-air breathing. While most cases occur after rapid, often uncontrolled ascents from depth, several cases have been described in the literature of gas emboli occurring after ascents from as shallow as 1 m (2,3). This correlates well with experimental studies which show that transpulmonic pressures as little as 95–110 cm H_2O are sufficient to cause lung tissue rupture (4).

The diver in the case described here suffered gas embolism while performing a dive in relatively shallow water. In addition, the diver reported that, before the onset of symptoms, he was having difficulty maintaining neutral buoyancy in the water column; this fact could have played a major role in the pathogenesis of his injury, particularly if he inadvertently breath-held during these periods.

The most unusual aspect of this case is that the diver suffered from a gas embolism that probably contained a higher-than-average fraction of O_2 relative to nitrogen. Divers using the MK-25 (LAR V) rebreather perform a "purging" procedure before submerging, which effectively increases the inspired O_2 fraction and decreases the inspired fraction of inert (nitrogen) gas. U.S. military divers perform a "Single Fill/Empty Cycle" (SFE) purge procedure, whereby they fill the breathing bag with O_2, then empty the bag by inhaling through the mouth and exhaling through the nose (outside the breathing loop), and finally refill the bag with oxygen. Butler and Thalmann (5) have shown that the SFE technique increases the oxygen fraction within the breathing loop to 74%. The diver in the case discussed here reported having performed an SFE purge before submerging, in accordance with U.S. Navy and Marine Corps diving procedures. This suggests that the emboli causing his symptoms contained approximately 74% oxygen and 26% inert gas (in contrast to an air embolism, which contains 21% oxygen and 79% inert gas). While nitrogen can only be eliminated by perfusion, oxygen within the emboli can be directly metabolized by the body's tissues into carbon dioxide (6). The elevated fraction of inspired oxygen in this case could help to explain the rather rapid resolution of the diver's symp-toms. Other factors may also have played a role, however, including the relatively short time between manifestation of symptoms and definitive treatment (approximately 20 min); this may have blunted the cerebral inflammatory response that is also known to contribute to brain injury from AGE.

The diver underwent chest radiography approximately 12 h after completion of treatment; results in his case were normal, with no evidence of residual pneumothorax or mediastinal emphysema. Additionally, the diver underwent spiral volumetric computed tomography (CT) of the chest 3 days postinjury; no pathologic findings were found. Nevertheless, some authors feel it advisable to search for occult anatomic lesions, such as emphysematous bullae, in cases of PBT, because patients with such anatomic abnormalities have an increased risk of PBT (7) and a high likelihood of recurrence (8). Reuter et al. (9) have suggested that in any case of suspected PBT, spiral CT should be performed, looking for preexisting pathology that might affect future fitness to dive. Others believe that postinjury imaging studies are unnecessary in cases where a clear precipitating factor for the injury can be found (so-called "deserved" gas emboli) (10). The case presented here was a "deserved" AGE in that the diver had problems with buoyancy within the water column, which could have played a significant role in the pathogenesis of his injuries.

Because the diver's symptoms resolved completely with a single treatment, no further recompression therapy was necessary, in accordance with U.S. Navy Diving Manual procedures. Navy and Marine Corps divers who suffer from residual symptoms are recommended to undergo daily TT6 or twice-daily TT5 or TT9 until no further improvement is seen (11).

Since no anatomic abnormalities were found on radiographic studies and the diver suffered from no neurologic sequelae, he has since returned to diving (military divers are restricted from diving for at least 4 wk after treatment for AGE), and has not suffered any further ill effects.

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