Could Breath-Hold Diving after SCUBA Cause Decompression Sickness?

by Petar Denoble

Recreational divers sometimes practice scuba and breath-hold diving on the same day. Some are concerned that breath-hold diving after scuba may increase their risk of decompression sickness (DCS). Their worry is that repeated descents and ascents might change the ultimate destination of venous gas emboli (VGE) — or bubbles — possibly present in their blood after scuba diving and make them more likely to suffer DCS. Another reason for their concern is the possibility that breath-hold dives build up dissolved inert gas in addition to what remained in the body after scuba and thus may create conditions for DCS. But is DCS feasible in breath-hold diving at all?

Modeling the risk of DCS after freediving in humans provided a hypothetical scenario in which DCS could occur after a single extreme dive, but the mechanisms that cause DCS seem unlikely in breath-hold diving. Cumulative effects of many repeated dives with short surface intervals could theoretically produce conditions for the occurrence of VGE, but so far there have been very few reports of bubbles observed in breath-hold divers. Some argue that DCS in breath-hold diving may be different than in scuba diving and that it may occur independent of VGE.

DCS-like symptoms in breath-hold divers have been reported. Symptoms of brain affliction have been observed in extreme diving done by harvesters, spearfishermen using scooters and in freedivers repeatedly doing very deep dives. DCS is one possible cause of these symptoms, but other causes include oxygen depletion, brain bleeding (due to extreme changes in blood pressure observed in breath-hold diving), arterial gas emboli caused by lung damage at depth, repeated micro-injuries to the brain and other factors. Pre-existing medical conditions like small vessel disease could also be involved.

Regardless of whether breath-hold diving produces VGE by itself, the concern that breath-hold diving after scuba may cause the redistribution of VGE seems rational, but the risk remains very small. Despite uncertainties about their causes, neurological accidents in extreme breath-hold diving have been observed and cannot be dismissed.

Is the occurrence of DCS in breath-hold diving a real risk?

Wong: DCS seems to be a real risk but only in extreme breath-hold diving. Clinical signs and symptoms have been observed in competitive divers, commercial sea harvesters of Japan (ama divers), recreational spear-fishermen of Australia and Spain, and in the pearl divers of the Tuamotu Archipelago. Symptoms never appear on the first day of the diving week for the ama divers, and symptoms occur only after diving for at least 3.5 to 4 hours to depths in excess of 66 feet when the surface interval is shorter than the dive time, suggesting nitrogen accumulation as a contributing factor.

Venous gas bubbles have been detected in Japanese ama divers (K. Kolshi 2010, pers. comm.) as well as in a breath-hold diver who performed a series of dives to depths of 154 feet despite breathing oxygen for decompression (K. Huggins 2006, pers. comm.). Obviously, detection of bubbles per se does not imply occurrence of DCS, but the possibility exists. A patent foramen ovale (PFO) may have been a contributing factor in the case of a diver who made between 10 and 12 dives to depths of 33 to 60 feet, each lasting 60 to 120 seconds with surface intervals of five to six minutes. Two hours after the last dive, he experienced dizziness, visual disturbance, chest tightness and numbness in the right side of his face. It appears that the dives he made were sufficient to produce VGE, which then may have caused symptoms because of the PFO.1

Pollock: There are anecdotal and retrospective data in the literature that are consistent with a diagnosis of DCS. A recent attempt to model the risk found that it was negligible for dives to depths of less than 330 feet, then rising as a function of exposure depth until the depth was sufficient for airway collapse to limit gas uptake from the lung, possibly in the range of 755 feet.2The magnitude of the hazard is unclear, but the absolute risk is probably very low for most freedivers, particularly when conservative surface times between dives are maintained.

Does breath-hold diving after scuba diving increase the risk of DCS?

Wong: Breath-hold diving after scuba may increase the risk of DCS, but the evidence is scarce. The classic case was reported by Paulev, who experienced nausea, dizziness, belching, hip and knee pain, weakness, paresthesia and blurred vision after performing repetitive breath-hold dives to 66 feet for five hours. His breath-hold dives were preceded by a hyperbaric exposure as a chamber attendant for eight minutes at 66 feet.3 Three similar cases of DCS have been reported after divers were exposed to pressure in a hyperbaric chamber prior to breath-hold diving.

Pollock: Compressed-gas diving prior to freediving certainly increases the theoretical risk. High tissue concentrations of inert gas after compressed-gas dives could make the impact of the freediving important. While no experimental evidence exists, bubbles produced following the compressed-gas dive could migrate to more sensitive tissue when transiently compressed by the freedive. Similarly, the physiological stress of freediving could enhance pulmonary shunting, potentially increasing the risk or frequency of bubbles entering arterial circulation. The hazard might be greatest in the first part of the freedive when both bubble size and physical effort would be relatively high or at the end of the freedive if augmented shunting continued. Again, though, there is no evidence of these factors causing injury. Studying a relatively rare event like DCS is difficult; studying a second rare event on top of the first is much more difficult.

What is the nature of neurological symptoms reported in breath-hold divers?

Wong: Symptoms after breath-hold dives appear to affect the central nervous system more frequently than symptoms that follow scuba dives. The most common are vertigo, nausea, vomiting, paresthesia, muscular weakness and paralysis. Others include impaired concentration, lethargy, speech disturbance and altered level of consciousness. Musculoskeletal or joint pain appears uncommon.

Pollock: A key feature of the neurological symptoms reported by freedivers is the transient nature. This could be consistent with the lower gas loads associated with freediving exposures and the faster compression and decompression rates freedivers experience. It is tempting to think that we understand decompression hazards based on the wealth of compressed-gas data, but this includes relatively little at the high descent and ascent rates — on the order of 6 feet per second — commonly employed by freedivers.

About the Author

As Senior Research Director of DAN America, **Dr. Petar Denoble,** M.D., D.Sc., helps develop new studies about dive safety, oversees ongoing studies and monitors their progress. He currently leads Project Dive Exploration, the DAN fatality, incident and injury database and the patent foramen ovale (PFO) study.

What is the risk of neurological accidents in breath-hold diving, and how could it be mitigated?

Wong: The common factors causing neurological symptoms include breath-hold dives in excess of 66 feet, repetitive dives over the course of three hours or more and short surface intervals. If time spent at depth is more than double the time on the surface, even a series of repetitive dives lasting less than three hours would risk DCS. To avoid an increased risk of DCS, breath-hold divers should limit the number of repetitive dives and keep the surface interval time greater than the dive time.

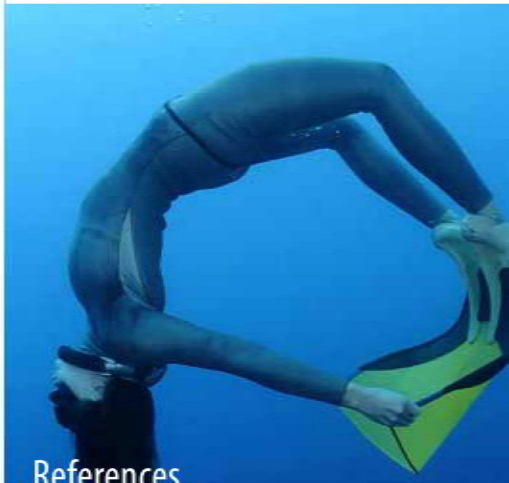
Pollock: Neurological compromise in freediving may result from hypoxic loss of consciousness, decompression-induced insult or other problems. A battery of strategies should be employed to reduce the hazard.4 First, freedivers must understand and limit predive hyperventilation; it works to extend breath-hold time but can completely remove normal protections against loss of consciousness. Freedivers should also employ defensive weighting, establishing empty lung neutral buoyancy at 16 feet, or deeper with deeper dives.

Adequate supervision to address incidents immediately is also critical. Direct supervision by a partner or partners should be maintained throughout a breath-hold dive and for 30 seconds postdive to ensure stable consciousness. The complexity of the support network increases with dive depth and other complications such as low visibility. Automatic surfacing devices have the potential to reduce the life risk under a range of conditions. The risk of DCS is reduced by separating freediving and compressed-gas diving and by maintaining a minimum surface interval between dives. The surface interval should start at twice the duration of the dive time and increase as a function of the exposure depth. ■

Meet the Experts

Neal W. Pollock, Ph.D., is a research director at DAN and a research associate at the Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, in Durham, N.C.

Robert M. Wong, M.D., FANZCA, was an anaesthesiologist at the Royal Perth Hospital and a medical director in the department of diving and hyperbaric medicine at Fremantle Hospital in Australia. He is a diving medicine consultant to the Australian pearling industry.



References

1. Gemp and Blatteau. Aviat Space Environ Med. 2006; 77:971-3.
2. Fitz-Clarke JR. Risk of decompression sickness in extreme human breath-hold diving. Undersea Hyperb Med. 2009; 36(2): 83-91.
3. Paulev P. Decompression sickness following repeated breath-hold dives. J Appl Physiol. 1965; 20(5):1028-31.
4. Pollock NW. Breath-hold diving: physiology and safety. Diving Hyperb Med. 2008; 38(2): 18-25.