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DIVING MEDICINE

THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY (UHMS) AND THE DIVING SCIENCE FOR DIVERS SERIES

DOES DECOMPRESSION ILLNESS OCCUR IN BREATH-HOLD DIVERS? — Dr H. Alan Wyatt, M.D., Ph.D

From time to time the question arises: can decompression illness occur in breath-hold divers? This topic has been addressed a number of times in the diving medicine literature, and here, we continue the series of Undersea and Hyperbaric Medicine Society "Medical Literature in Plain Language" articles with a discussion written by Dr H. Alan Wyatt, M.D., Ph.D., from the Department of Hyperbaric Medicine, LSU Health Sciences Center, Marrero, LA, in the USA. He has reviewed a paper published in Undersea and Hyperbaric Medicine in 2005.

Is there good evidence that neurological decompression illness occurs in breath-hold divers?

Paper title:
Neurological manifestations in Japanese Ama divers.

Authors:
K. Koshi, R.M. Wong, H. Abe, T. Katoh, T. Okudera, Y. Mano

Reference:
Undersea and Hyperbaric Medicine 2005; 32(1):11-20

The purpose and aims of this study:
It has long been held that breath-hold divers (divers making descents without the benefit of breathing apparatus) rarely, if ever, experience decompression illness (DCI). However, there have been reports in the literature of individuals and groups suffering DCI after repetitive breath-hold dives. This includes the "Taravana" syndrome previously described in the pearl divers of the Tuamotu Archipelago in the South Pacific. Over the course of a diving day, in which the divers made repeated breath-hold dives to 40 meters, with bottom times around two minutes and ascent rates of around 30m/min, approximately 20% of the divers developed Taravana syndrome. The syndrome is comprised mainly of central nervous system symptoms, such as nausea, dizziness, and altered mental state, but may also lead to hemiplegia or death.

A similar population of breath-hold divers exists in Japan. The so-called Ama divers are a traditional group, often female, found in Japan and Korea. This paper summarizes the results of several studies of Japanese Ama divers over the last five years by Dr Koshi and his colleagues, and reviews some of the other literature reports of DCI associated with breath-hold diving. The authors then review some possible mechanisms for the development of DCI in breath-hold diving.

The methods:
The authors conducted a survey of 16 male Ama divers on Japan's Mishima Island. Their diving patterns were analyzed with respect to depth of dive, frequency of dives (per hour), total hours spent diving (sub-divided into morning and afternoon hours), and neurological symptoms associated with diving. Additionally, brain MRIs were obtained in four of the cases. The authors then review their findings and compare them to those obtained with compressed air divers and other reported instances of DCI in breath-hold divers.

The results:
The survey of Ama divers revealed that many were developing stroke-like symptoms while they were diving, or shortly thereafter. The 16 Ama divers in question had made from 20-40 dives per hour to depths from 7-30 meters anywhere from 4-5.5 hours a day. Of this group, 11 divers experienced frank neurological symptoms, ranging from dizziness alone in two, through loss of consciousness and hemiparesis. Examinations of four of the Ama divers using MRI showed that the divers had lesions identical to those previously reported for compressed-gas divers with neurological DCI. Specifically, they had multiple areas of infarct (tissue death from disruption of blood flow) that occurred primarily in those parts of the brain with the poorest blood supply.

Review of the literature reveals a number of other cases of DCI in breath-hold divers in various scenarios, in addition to the Ama and Tuamotu divers mentioned above. The first of these reports involve active breath-hold divers making almost as many dives as the professional divers, with the addition of submarine scooters to increase their depth range. These divers reported profiles remarkably similar to the Ama divers, and they developed much the same neurological symptoms.

Another highly relevant variation was seen in earlier reports from the Danish and Norwegian Navy's submarine escape training facilities. These reports describe 4 divers who had been in the decompression chamber at 20 m breathing compressed gas for a single short period, and who subsequently performed multiple breath-hold dives to 20m in the escape tank resulting in neurological DCI. All were recompressed successfully, but these are cases that may bear considerable relevance to those divers who like to breath-hold dive during the surface interval between compressed gas dives.

What does all this mean?
While it is widely believed that a breath-hold diver should not suffer from DCI, consideration of the physics involved shows that not only is it possible, but in some circumstances it might be expected. The development of DCI involves the formation of nitrogen bubbles in tissue or blood. This in turn is governed by the amount of nitrogen dissolved in the blood or tissue and the rate of decompression. Henry's law states that the amount of gas dissolved in solution is proportional to the partial pressure of that gas exposed to the solvent and the solubility of the gas in that solvent. Air at sea-level has a total pressure of 760 mmHg. In the airways and lung of a diver, 47 mmHg is water vapor, and of the remaining 713mmHg, approximately 563mmHg is nitrogen (79% of air). At 10m (33fsw, 2ATA) the nitrogen pressure is now 1126mmHg, thus providing a driving force for nitrogen absorption. It should be noted that this is regardless of whether the diver is breathing compressed air or is breath-hold diving. The difference is that the compressed air diver has a much greater supply of gas and remains at depth for longer, where the breath-hold diver only has the amount of nitrogen in one breath, and stays for short periods. However, given enough dives of sufficient depth and duration, a breath-hold diver can accumulate a significant nitrogen load. Add to this the rapid ascents made by the breath-hold divers, and it can be seen that in the course of a working day, the active breath-hold diver may be at a higher risk of DCI than previously imagined.

The authors suggest that the most likely cause of DCI in breath-hold divers may be venous bubbles which pass into the arterial circulation. Venous bubbles are known to form to some extent following most compressed air dives. Using ultrasound, researchers have also shown venous bubbles in a number of breath-hold divers. For venous bubbles to reach the arteries there must be some kind of "shunt" that bypasses the lungs, as most venous bubbles are usually filtered out in

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the lungs. It has been speculated in recent years that communication between the two upper chambers of the heart known as a patent foramen ovale (PFO), which is found in about 30% of the population, might be responsible. The relevance of this in breath-hold divers remains unclear, and studies in Ama divers showed that more had neurological symptoms than had detectable PFOs.

Another mechanism that may be responsible is the passage of tiny bubbles across the lung "filter". These bubbles would have to be fairly small, but they may "clump together" once past the lung, leading to the formation of bubbles large enough to cause trouble, or they may trigger harmful changes in blood like clot formation.

A final theory advanced by the authors is that breath-hold diving may cause pulmonary barotrauma. Not from the heavily emphasised expansion of compressed air inhaled at depth that all scuba divers are taught and understand, but from other less frequently described mechanisms such as simple over-distension from taking a maximal breath, or uneven distribution of gas in the lung with consequent differing rates of expansion of adjacent areas on ascent. Pulmonary barotrauma can allow direct passage of gas in the lungs into the blood.

None of these theories are definitively proven.

The bottom line

Many recreational divers are under the impression that breath-hold diving cannot cause DCI. This paper, and some of the studies discussed therein, show that diverse groups of working breath-hold divers do suffer neurological DCI. However, these divers tend to make many deep dives with very short surface intervals for five or more hours per day, and the relevance to the "average" breath-hold diver is uncertain. Nevertheless, it is sobering to consider that breath-hold diving can lead to DCI, perhaps more so with the apparent sensitizing effect of prior compressed air breathing.

While there is some cause for concern, it is unlikely that the average recreational diver will be making repetitive breath-hold dives to deep depths for hours on end. Arguably the most relevant aspect to those reading this article is the anecdotal evidence for possible danger when breath-hold diving between scuba dives. Perhaps that practice should be reconsidered. At the very least, divers should remember that breath-hold diving in association with scuba diving may render dive planning tools for subsequent scuba dives less accurate and almost certainly less conservative. Finally, remember that any signs and symptoms of DCI should be taken seriously, even if it was several hours since the last compressed air dive, and especially if there was subsequent breath-hold diving.