Review articles

Decompression sickness in breath-hold diving

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Key words
Breath-hold diving, decompression sickness, review article

Abstract

(Wong RM. Decompression sickness in breath-hold diving. *Diving and Hyperbaric Medicine*. 2006; 36: 139-144.)
Decompression sickness (DCS) in breath-hold (BH) diving has long been disputed as a distinct clinical entity. However, there has been a flurry of case reports describing various symptoms arising in BH divers. Since the 1950s, *Taravana* has been described in French Polynesia, where divers suffered from paralysis, vertigo and nausea. Prior hyperbaric exposure followed by BH dives also led to symptoms and Paulev, a submarine medical officer, described his personal experience of DCS after a series of BH dives. Three similar cases have been described. The Ama divers of Japan and Korea have long been studied, but in the early days no confirmed cases of DCS had been recorded, although Doppler ultrasound had detected circulating bubbles and cerebral injuries have been documented in them. Other BH divers have also been studied, including recreational sports divers and diver fishermen. In all these groups symptoms have been recorded after repetitive deep dives. Theoretical calculations by various authors have concluded that repetitive deep BH dives could give rise to symptoms of DCS. The clinical entity of DCS in BH divers is distinctive in that it tends to affect mainly the brain. Symptoms, even without treatment, tend to subside over time, in many cases without sequelae. Although the aetiological factors causing DCS in BH dives have not been elucidated, there is no doubt that such a clinical entity does exist. Hyperbaric oxygen therapy should be offered to divers with symptoms. Preventive measures are suggested.

Introduction

Breath-hold (BH) diving is common among recreational divers, seafood harvesters, hitherto pearl and sponge divers and, nowadays, competitive freedivers. The risks of breath-hold diving such as barotraumas, salt-water aspiration, near drowning and drowning syndrome, hypothermia, cardiac arrhythmias, marine animal injuries, and ascent hypoxia, 'shallow water blackout', are well known. However, neurological problems caused by decompression sickness (DCS) in BH diving are seldom acknowledged. Does DCS exist as a clinical entity in BH divers?

It has long been held that bubble formation causing DCS from BH diving is most unlikely. Nevertheless, in 1931, Professor Gito Teruoka asserted in his article *Die Ama und ihre Arbeit* that he was aware of the increased nitrogen uptake with increased ambient pressure, but could not find any confirmed cases of DCS among the Ama divers.[1] He indicated then that further research was necessary.

Lanphier believed that bubble formation causing DCS was most unlikely from breath-hold diving, but conceded that this view did not take into account the extremes of depth, frequency and repetition to which breath-hold diving can be carried.[2,3] Using the USN decompression tables, he calculated the probable tissue nitrogen levels for a given series of BH dives and equated them to a specific air-supplied dive for which a decompression schedule or exposure limit could be specified. In his risk assessment, he made the assumptions that the time at depth was spent at maximum depth, ascent and descent were instantaneous, gas uptake and elimination were exponential and the surface/depth (S/D) time ratio was considered.[3] He proposed that no limit of total time needs to be imposed if time at surface is at least equal to time at depth (S/D = 1.0), but that, if the S/D = 0.5, there was a risk of DCS in less than three hours of diving.

Fahlman and Bostrom used a mathematical model to predict nitrogen tensions (PN_2) during hypothetical dives.[4] The model predicted maximum venous PN_2 during a dive to 80 metres of sea water (msw) to be 3 ATA (304 kPa), or 310% higher than the surface equilibrium value (0.74 ATA, 75 kPa). Maximum venous PN_2 during repeated dives to 30 msw were predicted to be 1.44 ATA (145 kPa) and 1.69 ATA (171 kPa) for a surface interval (SI) of 300 and 90 seconds respectively. Predicted venous PN_2 during the first and sixth dive reached 88% and 97% of the maximum estimated venous PN_2 during the entire series respectively. They concluded that the results suggest symptomatic N_2 levels can be reached during BH diving. Furthermore, when diving repeatedly, a SI twice as long as the dive time could help to reduce excessive PN_2 levels.

Nitrogen accumulation measured in brachial venous blood has also been demonstrated in repetitive BH diving in Korean Ama divers, but it was thought that the level of nitrogen accumulation was insufficient to cause DCS.[5] These dives lasted only three hours and to depths of 4 msw.

Evidence of intravascular gas in BH divers

Before the advent of the ultrasound Doppler detector, Schaefer had observed foam in venous and arterial blood drawn immediately after a BH diver surfaced from a single dive to 27 msw lasting one-and-a-half minutes.[6] Subsequent samples drawn ten seconds after surfacing did not show any bubbles, indicating their transient nature. Some ten years later, both Spencer's group and Nashimoto detected venous gas emboli following repeated BH dives in Ama divers.[7,8] Further attempts to identify bubbles in BH divers using continuous Doppler and 2-D echocardiography did not find evidence of circulating air bubbles in BH divers who dived to depths of 24–40 msw over a two- to six-hour period.[9] The tests were performed within an average of 30 minutes of the last dive of the day (range 3–75 minutes). However, bubbles, if present, may have been missed because of their transient nature. Huggins demonstrated Grade I bubbles in a breath-hold diver after a series of repetitive dives in the 46–67 msw range, although in-water oxygen breathing for two periods was given (personal communication, Huggins, 2006). Doppler detection was performed some 18 minutes after surfacing from the last dive.

History

In 1956, Cross alerted the diving community to the existence of *Taravana*, a condition known in Tuamoto Archipelago in French Polynesia where the BH pearl divers suffered symptoms such as vertigo, paralysis, mental anguish and unconsciousness (Table 1).[10] They dived to depths of between 13 and 43 msw with BH times of up to two-and-a-half minutes and surface intervals of between four and ten minutes, usually working a six-hour day. Cross stated that "*Taravana is most frequent toward the end of the day of diving when divers working in a rich lagoon under ideal conditions go crazy with greed and dive until the shell is gone or Taravana strikes*". In Hikueru Lagoon, there were estimated to be 235 divers, who hyperventilated for three to ten minutes, and then descended with the assistance of a lead weight of about 3.5–5.5 kg. At the end of one six-hour day, a total of 47 divers had been affected by various taravana symptoms. However, in nearby Mangareva Lagoon, divers who had 12- to 15-minute surface intervals never suffered from *Taravana*.

In 1957, in Takapoto Lagoon, 43 divers (35 males and 8 females) were observed. There were 13 cases of *Taravana* in a three-week period (one died subsequently) and 12 cases of vertigo and nausea. One was paralysed (paralysis was always accompanied by vertigo, nausea and a general feeling of anguish). Depths of dives were to 15–25 brasses (one brass is approximately one fathom, the length of outstretched arms, a common form of measurement of indigenous divers – about 6 feet or 1.83 metres). In 1958, 34 cases of vertigo, nausea and mental anguish were reported. There were also six cases of partial or complete paralysis, three of temporary unconsciousness with no other symptoms, two were mentally affected and two died. Of the mentally affected, one was unable to recognise his family or home. He was restless, irritable and lacking in understanding of his surroundings. The other was unable to speak coherently even though he seemed normal in other respects.

Symptoms of *Taravana* could last from a few hours to a lifetime. One male Paumotan diver, Tahauri Hutihuti, who was 71 years of age in 1958, claimed he had never had *Taravana*, but he was slow mentally and often missed what he was reaching for. Another diver, Turoa Hutihuti, who was 48 years old in 1958, made frequent dives to 140 fsw for two minutes. He suffered *Taravana* a few times. Once he had paralysis of the right side, and had vertigo and nausea several times; the paralysis lasted three months. He also had a slight visual defect and it was thought this was related to *Taravana*. Paralysis is a common form of *Taravana* and Dr Truc, the French physician, believed more than 95% of it to be temporary, and that the divers would recover completely in a matter of hours or occasionally days.[10]

Cross considered *Taravana* to be a form of DCS, but since no one believed DCS occurred in BH diving, he then considered "anoxia" as a likely explanation. Craig, Lanphier and Rahn had emphasised the danger of hypoxia in BH diving, especially with prior hyperventilation, although they questioned anoxia as an explanation, and suggested that no consequences other than loss of consciousness (LOC) and drowning could occur.[11,12] Craig thought air embolism or DCS could be a likely explanation for *Taravana*.

Reported cases

Paulev experienced symptoms of DCS such as nausea, dizziness and belching, followed by onset of pain in his hip and knee, a weak left arm and tired right arm, as well as paraesthesia and blurring of vision after performing repetitive BH dives to 20 msw for five hours.[13] However, his dives were preceded by a hyperbaric exposure as a chamber attendant for eight minutes at 20 msw. Three similar cases of DCS in BH divers were reported, after they were exposed to pressure in a hyperbaric chamber prior to BH diving.[14]

Bayne and Wurzbacher, as well as Bruch have described cases of pulmonary barotraumas (PBTs) in BH divers who were at depths of 1.8 msw and 4.5 msw respectively.[15,16] Fanton et al reported the case of a spear fisherman who performed 14 repetitive dives per hour for three hours to depths of up to 131 fsw (40 msw).[17] He lost consciousness on surfacing without any evidence of near drowning. Investigation showed abnormal EEG and MRI consistent with focal neurological damage.

Kohshi et al reported multiple cerebral infarctions in two Japanese Ama divers who dived repeatedly between 15 and 25 msw for five hours.[18] Subsequent publications discussed 16 Ama divers, 13 of whom had neurological dysfunction (Table 1).[19,20] In earlier days, Ama divers dived in reasonably shallow waters and also they used to wear cotton suits which precluded them from prolonged stays underwater due to

Table 1
Symptoms experienced amongst three groups of divers (rec. – recreational)

Symptoms	Taravana	Japanese Ama	Spanish rec.
Dizziness/vertigo	34/47	9/16	5/30
Nausea	34/47	6/16	1/30
Motor weakness	6/47	6/16	11/30
Sensory changes		3/16	17/30
Altered consciousness	3/47	1/16	13/30
Headache			13/30
Mental disturbance	36/47	4/16	
Visual disturbance			10/30
Speech disturbance		1/16	7/30
Fatigue			7/30
Motor incoordination			5/30
Memory loss			4/30
Convulsion			1/30
Sphincter relaxation			1/30
Auditory disturbance			1/30
Localised pain			1/30
Cardiorespiratory arrest			1/30
Death	2/47		

hypothermia. These days, however, they wear neoprene suits and make weight-assisted dives, typically with 15 kg weights, and descend to between 15 and 25 msw. Dive times would be between one and one-and-a-half minutes with surface intervals of up to three minutes. They generally work two shifts a day (five-hour working day and five days per week.).

One case described was that of a 33-year-old Ama diver who dived to 22 msw, with a bottom time of one to one-and-a-half minutes with a one-minute surface interval. He commenced diving at 0920 and had 20 minutes for lunch. At 1410 he noted symptoms, which included dizziness and blurred vision in the right visual field. MRI performed four days later showed an infarction in the occipital region. However, his disturbed vision regressed within three weeks. It is interesting to note that in the Ama divers there were no spinal or musculoskeletal symptoms reported. Most symptoms were transient. Some paresis improved within ten minutes. Sensory symptoms, however, took longer to recover, sometimes as long as two to four weeks. Symptoms never appeared on the first day of the diving week, and on the day of injury the Ama diver had dived for at least three-and-a-half to four hours to depths of 20 msw.

Neurological problems have also been reported in competitive sports diving: in multiple constant-weight dives to 25–30 msw; three variable-weight dives to 35–90 msw; and a single no-limits dive to 120 msw.[21] * These divers suffered from such symptoms as hemiplegia, ataxia, dysarthria, diplopia and colour blindness. The diver who made a single dive to 120 msw had reached that depth several times previously without problems. On this occasion, however, he used a new assisted-ascent technique (at 4 msw.sec^{-1}). Shortly after surfacing, he experienced paraesthesia in the right leg, followed within minutes by a right-sided hemiplegia that responded to recompression treatment within 30 minutes using a US Navy (USN) Treatment Table.[22]

A large number of Spanish spear fishermen using submarine scooters for BH diving have suffered neurological symptoms. They have managed to achieve depths of 25 to 46 msw, with BH times of up to four minutes (90 to 240 sec, mean 133 sec; Table 1).[23] A depth of 63 msw has also been reported, the number of dives varied between 15 and 20 dives per hour over a period of three to eight hours. The surface interval was usually two minutes or less. Symptoms were immediate on surfacing and were all neurological (Table 1).[24]

In Australia, spear fishing is popular among BH divers. They usually perform multi-day diving spending 5–6 hours in the water each day. Depths of dives range from 13–20 msw to 27–30 msw. Dive times are usually in the vicinity of two to three minutes with a one-minute surface interval; sometimes with deeper dives, a two-minute SI would be common. A preliminary unpublished survey by the author indicated that very few of those who suffer symptoms consult their medical practitioners. This is due mainly to the lack of appreciation that DCS could occur in BH diving and also the fact that such symptoms are usually attributed by the divers to other causes such as viral illness. Furthermore, symptoms are normally of short duration, and mostly no sequelae are experienced.

Symptoms arising from breath-hold diving

The symptoms encountered among different groups of BH divers are diverse and vary in frequency (Table 1). The most common include dizziness, ataxia, nausea, hemiparesis, paraesthesia, visual and speech disturbances and altered states of consciousness. Symptoms common in scuba divers such as musculoskeletal pains and spinal cord involvement are uncommon in BH divers unless there has been previous hyperbaric exposure prior to BH diving. Musculoskeletal pain has been reported in only two cases: one in an Australian BH diver (two days after the event) and the other in a Spanish recreational diver.[13] In eight Australian spear fishermen, a similar range of symptoms to those listed in Table 1 were noted, headache, nausea and dizziness being the most common.

Lesions, particularly in the Japanese Ama divers, tend to be centrally located with sparing of the spinal cord or musculoskeletal systems. Some of the symptoms described by BH divers could have been due to nitrogen narcosis (such as euphoria), hypoxia (altered level of consciousness,

* Footnote: For definitions see Mckie N. Freediving in cyberspace. *SPUMS J.* 2004; 34: 101-3.

muscular weakness and incoordination, loss of motor control, visual disturbance), carbon dioxide retention (headache, dizziness, confusion and amnesia) or even middle/inner ear barotraumas or alternobaric vertigo (vertigo, nausea, disorientation, visual disturbance) rather than to DCS.

As already stated, even without treatment most of the symptoms in BH divers subside spontaneously. The Japanese Ama divers recorded 10 minutes to four weeks, the Spanish divers took 30 minutes to 72 hours and the Australian spear fishermen took 12 to 36 hours. In the *Taravana* cases, in whom paralysis was the most common symptom, more than 95% completely recovered in hours, the rest in days.

Common factors causing symptoms

Factors causing neurological symptoms include:
- dives in excess of 20 msw
- repetitive dives of three hours or more
- a rapid rate of ascent by necessity
- surface intervals of short duration, shorter than the depth time.

However, it has been reported that a BH diver who performed repetitive dives for three-and-a-half hours to depths of only 8 msw developed severe headache, dizziness, blurred vision, vertigo, numbness and weakness of all four limbs on surfacing and had to be rescued.[25] No clinical evidence of pulmonary barotrauma was detected. Although the presumptive diagnosis was CAGE, it could also have been hypoxia leading to loss of motor control (LMC).

Lindholm and Lundgren have shown that 11% of competitive BH divers performing static apnoea had symptoms of hypoxia such as LOC or LMC.[26] Two subjects who had LMC had P_AO_2 of 19.6 and 21 mmHg (2.6 and 2.8 kPa) respectively.

Mechanism

The aetiological factors of DCS in BH diving have not been elucidated. As noted previously, the Ama divers of Mishimi Island of Japan never complain of symptoms on the first day of the diving week. Their symptoms when manifested appear only after at least three-and-a-half to four hours of repetitive diving to depths of 20 msw and when the surface interval is less than the depth time, suggesting nitrogen accumulation could be a contributing factor. We do not know how long it takes for the body to totally eliminate excess nitrogen from repetitive diving. In compressed-air diving, the USN decompression tables assume a 12-hour surface interval to be clear of residual nitrogen time, whereas the DCIEM tables assume an interval of 18 hours. It is feasible that it takes much longer than 18 hours. After repetitive dives to 3 ATA, sufficient nitrogen is absorbed to cause supersaturation. Fahlman and Bostrom have concluded that symptomatic N_2 levels could be reached during repetitive BH dives using their mathematical model to calculate hypothetical dives.[4] Based on computer modelling, Olszowka and Rahn calculated that N_2 accumulation occurs in fat tissue increases throughout repetitive BH dives, but brain PN_2 does not increase with repetitive BH dives, preventing clinically significant autochthonous bubble formation in the brain.[27]

It is assumed that bubble formation occurs in the venous circulation based on Doppler evidence.[7,8] The lungs have such an efficient filter that the bulk of the bubbles are prevented from reaching the left side of the circulation. However, on repetitive dives, some of the bubbles could reasonably bypass the lung filters and reach the arterial circulation.[28,29] Buoyancy then assists the bubbles to reach the cerebral circulation.

Various other mechanisms of DCS have been postulated, such as:
- Cardiac shunting via an atrial septal defect (ASD) or patent foramen ovale (PFO). However, evidence from the Ama divers surveyed does not lend support to PFO or ASD as a contributing factor.[18]
- Pulmonary barotraumas. These lead to CAGE and are not common in BH diving.[14,15]
- Bubble formation in the arterial circulation. Although arterial bubbles have been seen in decompressed animals, bubbles are unlikely to form *de novo* in large arteries. Inert gas supersaturation sufficient to provoke bubble formation is improbable in arterial blood since the healthy lung essentially equilibrates alveolar and arterial gas tensions in a single pass. Arterial supersaturation may occur in a very rapid ascent of 6 $msw.sec^{-1}$, but arterial bubbles have proven difficult to demonstrate even under these conditions.[30]
- Microbubbles.[31,32] Bubbles smaller than 21 μm can pass through capillaries in the brain and do not usually cause any lesions.[33] However, Hills and James have shown experimentally that microbubbles can impair the blood-brain barrier transiently.[34] It is possible that with repetitive diving, microbubbles can and do impair the cerebral circulation, as has been seen in the Ama divers who show multiple cerebral infarctions in the terminal or border zone of cerebral arteries visible on MRI.[17,19] Such lesions could be the consequence of microbubbles which are too small to be detected by conventional Doppler technique.

Treatment

Most BH divers do not appreciate that BH diving could cause DCS and consequently when they have symptoms do not seek medical advice. Nonetheless, even without treatment, symptoms tend to subside and generally have no sequelae. No standard protocol of treatment has been agreed upon. BH divers have received surface oxygen and various hyperbaric treatment tables have been employed, which include USN 6, USN 5, Comex 12, Comex 18, HBO 14.[21,23] Adjuvant therapy such as nifedipine, non-steroidal anti-inflammatory drugs, heparin, steroids and diazepam have all been tried.

Divers who present with symptoms after BH diving should be offered hyperbaric oxygen therapy. A delayed presentation is not a contra-indication to treatment. A BH diver presenting four days after his dives with symptoms of tiredness and cognitive dysfunction, and cerebellar signs and failed sharpened Romberg test, as well as poor short-term memory, responded to a USN 6.[35]

Prevention

It would appear that to avoid DCS from BH diving, the following empirical strategy could be adopted:

- dive no deeper than 20 msw (although this might not be practicable)
- limit the number of dives per day or dive less than three hours continuously
- ensure the SI is at least twice as long as the dive time
- if feasible, breathe surface oxygen, at least during the lunch break and at the end of the diving day
- the Ama divers could conceivably breathe oxygen to decompress for 5 to 10 minutes at 6 msw at the end of the diving day.

It has been claimed that oxygen decreases decompression time by 30% to 50% depending on the depths of the dives. Imbert and Bontoux, using the French air decompression tables with in-water oxygen decompression, indicated that oxygen decompression not only saves decompression time but also has the effect of decreasing the incidence of DCS to two to three times lower than air decompression for dives of the same depths and bottom times.[36]

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This article is based on a presentation by Dr Wong at the Symposium on Breath-hold Diving, Orlando, Florida, hosted by the Undersea Hyperbaric Medicine Society and Divers Alert Network, 2006.

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Dates: April 15 - 20
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Guest Speaker
Neal Pollock, PhD

Themes
From mountain high to ocean deep – the physiological challenges of extreme environments
Medical aspects of technical diving

Neal Pollock is a research physiologist at the Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC. He is also heavily involved in DAN International and was one of the editors of the recent guidelines for scuba diving and diabetes. He works regularly in Antarctica and has also been involved in high-altitude physiology studies. He thus brings a wealth of expertise to our meeting and is an excellent speaker.

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