Neurological manifestations in Japanese Ama divers.

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Kohshi K, Wong RM, Abe H, Katoh T, Okedera T, Mano Y. Neurological manifestations in Japanese AMA divers. Undersea Hyperb Med 2005; 32(1):11-20. Repetitive breath-hold (BH) diving can lead to accumulation of nitrogen (N_2) in blood and tissues, which may give rise to decompression illness (DCI). An unusual condition is "Taravana", the diving syndrome reported by Cross in the 1960s. That report generated wide discussion as to whether BH diving can cause DCI. Paulev was the first person to suggest the link between DCI and BH diving. He, a submarine medical officer developed symptoms of DCI after a series of BH dives, having proceeded the dives by spending time in a hyperbaric chamber at 20 meters for 8 minutes. Recently four professional Japanese BH divers (Ama) with histories of diving accidents were reported. Magnetic resonance imaging of these divers detected cerebral infarcts localized in the watershed areas of the brain. A survey conducted on their island revealed that many Ama divers had experienced stroke-like events. A clinical feature of DCI in BH diving is that the damage is limited to the brain. Although the mechanisms of brain damage in BH diving are unclear, N_2 bubbles passing through the lungs or the heart so as to become arterialized are most likely to be the etiological factor.

INTRODUCTION

It is widely thought that decompression illness (DCI) is extremely rare in breath-hold (BH) diving. Until recently, there has been no report of DCI in Ama divers, who are professional BH divers in Japan and Korea (1,2). Although Ama divers are often female, male divers are common in Japan (1). In a survey conducted on Mishima Island in Japan many Ama divers were found to have experienced "stroke-like events" during or after repetitive BH dives (3). Moreover, several similar cases have been reported in professional and amateur BH divers (4-6). Based on the reported, clinical symptoms and neuro-radiological findings of DCI in BH divers, brain lesions are particularly prominent.

CLINICAL SYMPTOMS

I. Professional breath-hold diving

BH diving accidents include "Taravana" diving syndrome observed in the Tuamotu Archipelago in the South Pacific (7). This syndrome was described by Cross in a survey of BH pearl divers (7,8). 235 male and female divers and their diving technique were studied. The divers hyperventilated for 3-10 minutes before diving. They reached the seabed within 30-50 seconds using weights (4-6 kg), worked for 30-60 seconds, and returned to the surface within 20 seconds. The depth was 40 meters, and they dived repeatedly and continuously for six hours. In one day of diving, 47 divers (20%) developed symptoms of "Taravana", which affected mainly the central nervous system (CNS). Of these, 34 divers reported dizziness, nausea or mental anguish; partial or complete hemiplegia occurred in six; transient disturbance of consciousness occurred in three; mental disorder and death occurred in two. In addition, Cross stated that pearl divers on Mongareva, a nearby lagoon, using the same diving technique but with longer surface intervals of 12-15 minutes never developed "Taravana" diving syndrome. However, investigators disagreed as to whether diving accidents with these symptoms constitute DCI, and no consensus has been reached..

Table-1 Diving patterns and neurologic dysfunction in 16 Japanese male Ama divers

Case/ age	Diving depth (msw)	Number of dives (/hour)	Diving time (hours) (morning, afternoon)	Neurological symptoms of diving accidents
1/46	10-15	30	5 (3, 2)	left hemiparesis 10 minutes after 4 hours' diving (35)*; dizziness
2/39	15-23	20	4.5 (3.5, 1)	crossed sensory numbness in the right body and the left face 5 minutes after 4 hours' of diving (29); euphoria, dizziness and/or nausea
3/43	15-22	20-30	5.5 (3.5, 2)	right hemiparesis and hemisensory numbness 10 minutes after 4 hours' diving; and loss of consciousness 2 hours after the last dive (34); dizziness and/or nausea
4/49	15-30	20	5.5 (4, 1.5)	hemiparesis and numbness in the right side and dysarthria during 3.5 hours' diving (41); dizziness and/or nausea
5/49	10-20	30	5 (4, 1)	right hemiparesis during 5 hours' diving (37); nausea
6/41	10-20	20	4.5 (3, 1.5)	left hemiparesis 5 minutes after 3.5 hours' diving (25); left lower limb monoparesis during 4 hours' diving (30); euphoria, dizziness and/or nausea
7/44	10-15	30	5 (3.5, 1.5)	left hemiparesis during 3.5 hours' diving (25); dizziness
8/41	10-15	30	4.5 (3, 1.5)	right hemisensory numbness during 3.5 hours' diving (25); dizziness and/or nausea
9/39	15-25	25	5 (3.5, 1.5)	right hemiparesis with facial involvement during 3.5 hours" diving (33); dizziness
10/40	7-12	25	4.5 (3.5, 1)	euphoria
11/41	10	30	4 (3, 1)	euphoria
12/45	10	20	5 (3, 2)	dizziness
13/48	20-30	30	5 (3.5, 1.5)	dizziness
14/59	10-15	20	5 (3.5, 1.5)	none
15/63	8-15	20	5.5 (4, 1.5)	none
16/44	8-12	40	4.5 (3.5, 1)	none

msw: meters of sea water, * parenthesis indicates the age at the time of diving accidents

Among the Ama divers Kohshi et al. surveyed, many experienced stroke-like events (3). These diving accidents were particularly liable to occur when the Ama divers prolonged their repetitive deep dive sessions longer than 3 hours. The first and second most common symptoms were hemiparesis and hemi-sensory disturbance, respectively (Table 1). Some Ama divers had experienced sensory numbness in the unilateral facial and half body, dysarthria, or disturbance of consciousness. Characteristically, these neurological disorders were transient and resolved completely within a short time. In addition to the obvious neurological events, Ama divers frequently presented with neurological symptoms such as euphoria, dizziness, and/or nausea. DCI was discovered in the Ama divers in the village surveyed, as well as other districts (9). Unlike

compressed air diving, the DCI caused by BH diving is limited to brain involvement with sparing of the spinal cord. Another characteristic is that most Ama divers exhibited transient neurological deficits lasting only several hours, and at the very most, one month (3,9,10). Such events frequently developed when diving depths exceeded 20 meters and diving sessions continued for longer than three hours. Although the dive profiles in the village surveyed are similar to those of another district, DCI in Ama divers has not been reported (11). One of the reasons that DCI in BH diving is not well recognized by physicians is because of the transient nature of this disorder (3). Another is that Ama divers visit medical facilities infrequently due to the inherent secrecy of Japanese diving communities.

These diving accidents are similar or identical to those of "Taravana" diving syndrome, suggesting that the syndrome is very likely to be DCI caused by BH diving (12).

II. Breath-hold diving after compressed air exposure

BH diving causing DCI was reported by Paulev, a medical officer in the Royal Danish Navy (13). The incident occurred in a submarine escape training tank (SETT) in the Norwegian Naval base of Haakonsvaern at Bergen, when Paulev spent 8 minutes at 20 meters as an attendant in a recompression chamber. After this, he performed a number of BH dives in the tank to 20 meters as an instructor supervising escapes. Each dive took 20-25 seconds to reach the bottom, where he would sit or walk until he felt the need to breathe (about 2 minutes), and then ascend to the surface, which took 10-15 seconds. Surface intervals were between a few seconds and 2 minutes. He was in the water for about 5 hours. During the last 2 hours, he experienced nausea, dizziness and eructation. However, during the last 30 minutes, he developed pain in his left hip, then the right knee and eventually, the right leg felt tired and the right arm was weak. Two hours after leaving the water he had severe chest pain, which was not influenced by expiration or change of position and was accompanied by a feeling of inspiratory distress and a sense of imminent collapse. Paresthesia developed in the right hand together with blurring of vision. Three hours after the diving a colleague found him markedly pale, exhausted as in impending shock. Examination confirmed the visual disturbances and a partial paresis of the right arm. He also had abdominal pains and anesthesia of the ulnar region of the right hand. He was compressed to 6 ATA (50 meters) where he had immediate relief of symptoms of dizziness and nausea, followed by the disappearance of the paresthesia and anesthesia, with only a mild prickly sensation remained in the right hand. After a careful examination, Paulev was treated with the US Navy treatment Table 3. His treatment was successful with immediate relief of all symptoms and signs.

Three other cases have also been treated in the Norwegian SETT (14). Each man had been compressed in the hyperbaric chamber to 20 meters before BH diving. All experienced neurological symptoms and were successfully treated, which supported the diagnosis of DCI from BH diving.

III. Recreational breath-hold diving

Batle reported 25 BH divers who used submarine scooters to achieve great depths, some to 63 meters in approximately 2 minutes, and with surface intervals of 2 minutes or less (4). All these recreational BH divers suffered from neurological DCI. Each session would take between 3 to 8 hours and the number of dives per hour was between 15 and 20. Magno et al described four BH divers who suffered from neurological signs such as hemiplegia, ataxia, dysarthria, diplopia and color blindness (5). The dives varied from a single weighted and buoyancy assisted dive to 120 meters, to three assisted dives to 35-90 meters, and to multiple unassisted dives to 25-30 meters over 2-4 hours. All these cases made total recovery, albeit that only some received treatments and others had none. Wong described two BH divers

who had nausea, vertigo and headache, neither of whom complained of joint pain. They were diagnosed as DCI caused by BH diving, and one was recompressed with complete resolution of symptoms and the other recovered over time without treatment (6).

IMAGING DIAGNOSIS

I. Cerebral lesions

In imaging studies, Kizer initially reported the computed tomographic findings of brain lesions caused by compressed gas diving (15). The lesions have been clearly demonstrated using magnetic resonance imaging (MRI) technique (16-18). However, brain images of DCI in BH divers have been obtained from only four of the reported cases (9,10). The MRI findings in these Ama divers showed multiple cerebral infarcts in areas corresponding to the symptoms and elicited signs. The brain lesions are localized in the basal ganglia, internal capsule, and deep and subcortical white matters (Figs. 1 & 2). The type of brain lesions found in Ama divers are identical to those caused by compressed gas diving.

These MRI findings in Ama divers suggest features of circulatory disturbance of the cerebral arteries, and not the veins. The ischemic lesions in the basal ganglia were situated in the terminal zone, and the lesions involving deep or superficial white matter corresponded to border zone or watershed regions (19,20). They are the so-called low-flow cerebral infarctions as a result of the low perfusion pressure in the terminal supply areas. In two Ama divers treated in the acute stage, cerebral vessels were examined by MR angiography, but no obvious abnormality was detected in the cerebral arteries corresponding to the infarcts (9). These MRI findings suggest transient circulatory disturbances due to air embolism.

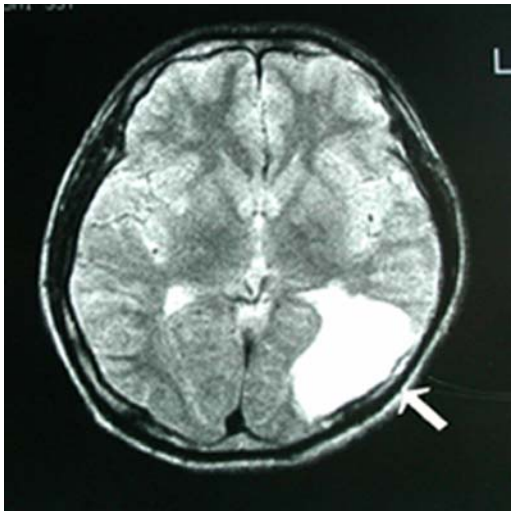

Fig 1-A

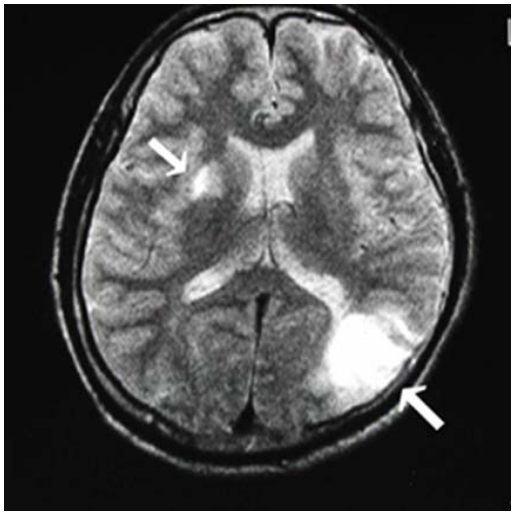

Fig.1-B

Fig. 1. A 33-year-old Japanese male Ama diver began diving at 9:20 A.M. and continued repetitive dives to 22 msw until noon. He repeatedly made 1~1.5 minute dive with 1-minute surfacing intervals between dives. After 20-min lunch , he began and continued such dives. He noticed dizziness and blurred vision in the right field at 2:10 P.M. His MRI on the 4th day after the accident showed two lesions in the left occipital lobe and the right basal ganglia (arrows). His visual disturbance regressed within 3 weeks but he had residual right lower quadrantanopsia. (Reprinted from J of Neurol Sci, 178, Kohshi K, et al: Neurological accidents caused by repetitive breath-hold dives: two case reports, 66-69, Copyright (2000), permission from Elsevier Science)

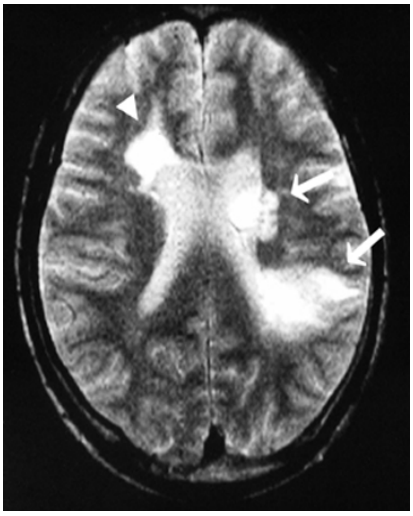

Fig. 2-A

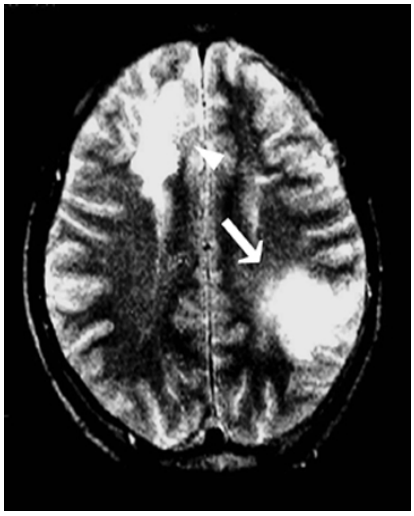

Fig. 2-B

Fig. 2. A 39-year-old Japanese male Ama diver began to dive around 9:00 A.M. and continued diving to 20 to 23 meters of seawater. He repeatedly made 1~1.5 minute dives with 1-minute surfacing intervals between dives. He noticed dizziness before noon and stopped diving, and this symptom disappeared about 30 minutes later. After a 1-hour lunch break, he began diving and noticed hemiparesis and hemi-sensory disturbance in the right side at 2:10 P.M. He has suffered from transient left hemiparesis at the ages of 17, 25 and 27 years. After the last accident his MRI on day 3 showed acute (arrows) and chronic cerebral lesions (arrow head). (Reprinted from Journal of the Neurological Sciences, 178, Kohshi K, et al: Neurological accidents caused by repetitive breath-hold dives: two case reports, 66-69, Copyright (2000), with permission from Elsevier Science)

Recently, multiple asymptomatic brain lesions, suggesting small infarcts, have been an issue in compressed gas diving (16,21). Some investigators have pointed out that the frequency was significantly higher in divers than in matched control healthy subjects (16,22). Similar brain lesions are probably present in Ama divers because our cases had small asymptomatic infarcts in the deep white matter and basal ganglia (9,10).

II. Lesions in spinal cord and vertebrae

Although myelopathy frequently occurs in DCI caused by compressed gas diving (23-25), spinal lesions have not been reported in BH divers (3-10). Since neither Japanese Ama divers nor Tuamotu pearl divers exhibited myelopathic symptoms (3,8), spinal cord lesions may be a rare occurrence in BH diving. However, in compressed gas divers, MRI has detected asymptomatic abnormalities in the vertebral bodies or intervertebral discs (16,18). Whether similar lesions are present in Ama divers remains to be investigated.

DEVELOPMENT MECHANISMS

I. Nitrogen accumulation

In compressed air diving, nitrogen (N_2) dissolves in tissues, particularly in fat tissues, and accumulates in accordance with Henry's Law. Due to possible N_2 accumulation after repetitive deep BH dives (13, 26,27), N_2 bubbles may form in the intra- or extra-vascular space of the brain during and after decompression. Using computer modeling Olszowka and Rahn calculated that N_2 accumulation in fat tissue increases throughout repetitive BH dives (26). With respect to N_2 kinetics, autochthonous N_2 bubbles do not normally form in the brain. Although autochthonous bubble formation has been observed in the brain following decompression in vivo, in typical human pressure exposures, the relatively luxurious cerebral perfusion is widely

considered to limit inert gas supersaturation, thus preventing clinically significant autochthonous bubbling (28). This is supported by the finding of autochthonous bubbles in brain and spinal cord of dogs in which the circulation is stopped before decompression (29). Brain PN_2 does not increase gradually from repetitive BH dives, despite a gradual increase of PN_2 in fat tissue (26). Even if N_2 bubbles form in the brain after decompression, the site may be in the smallest veins. However, brain images of Ama divers do not suggest that the lesions are caused by disturbed venous circulation (9,10).

After repetitive BH dives, N_2 bubbles may be formed in the venous side of tissues and flow into the right atrium. By using the ultrasonic Doppler method Spencer and Okino confirmed such bubbles in a Japanese Ama diver after a 51-min period of 30 dives to 15 meters of seawater (msw) (30). Nashimoto and Gotoh reported grade 1 bubble signals in one out of 33 Ama divers after 3 hours of repetitive dives in the morning and 2 hours of diving after lunch (31). Several possible causes have been identified for DCI in BH divers, and Lanphier reported that venous "silent" bubbles are most likely to induce diving accidents (32).

II. Venous bubbles

Since brain MRI findings of DCI in Ama divers are identical to those in compressed air divers (16-18,21,22), brain lesions may develop by a similar mechanism in both types of diving. Although cerebral disorders following compressed air diving have been diagnosed as CAGE or Type II DCS (23,25), arterialized venous bubbles through cardiac right-to-left shunts have been attracting attention rather than bubbles resulting from pulmonary barotrauma (21,33,34). The presence of a patent foramen ovale (PFO) or atrial septal defect is important in cerebral diving accidents. Knauth et al. reported that the frequency of DCI varied depending on shunt size (21), and Moon et al. suggested that shunting might induce severe DCI in the brain (33). Since the intra-cardiac shunts are present in 10-30% of healthy adults (35,36), bubbles passing through these shunts may induce paradoxical embolisms as a cause of DCI in the brain (21,33,34). It is also possible that in Ama divers, when venous bubbles flow into the pulmonary circulation, gas emboli could lodge in the smallest pulmonary arteries, leading to a rise in pulmonary arterial pressure. In fact, an experimental study has confirmed that increasing the pulmonary arterial pressure is easily caused by a small amount of bubbles and this allows bubbles to pass through the shunts such as a PFO (37). A forceful Valsalva maneuver may transiently increase right atrial pressure, exceeding that of the left and cause post-release central blood shift, which could lead to paradoxical cerebral embolism. However, among Ama divers surveyed, cerebral symtoms were considerably higher than among divers with shunts (3). Using the 2D Doppler echocardiography, Kohshi et al. could not detect the shunt in one Ama diver with DCI (9). Thus shunt per se does not account for all the neurological symptoms.

Although some investigators confirmed venous bubbles in Japanese Ama divers (30,31), the bubble formation caused by BH dives is controversial. This may be due to the transient nature of bubbles. In 1955, well before the introduction of Doppler ultrasound, Schaefer observed foam in venous and arterial blood drawn immediately after BH divers surfaced from single dives, lasting approximately 1.5 minutes to 27 meters (90 ft) (38). Blood drawn 10 seconds after surfacing did not show bubbles. This was a demonstration of a short-lived presence of bubbles, presumably due to supersaturation in the blood after single BH dives. Boussuges et al. noted that venous bubbles were not detected in 10 divers after 2~6-hour period of repetitive BH dives to mean maximum depth 35 msw (39). Their diving patterns are similar to those of Japanese Ama divers with neurological diving accidents (3,9,10). In Japanese Ama divers, bubble formation was detected in only one out of 33 cases (31). Microbubbles smaller than 30~50 μm in diameter

might be difficult to detect using continuous wave Doppler and 2D echocardiography (39).

III. Microbubbles

In addition to paradoxical gas embolism, microbubbles passing through pulmonary capillaries into the systemic circulation have recently been considered as another cause of DCI in compressed gas divers (24). Generally mammalian lungs usually constitute a complete filter for bubbles larger than 21 μm in diameter (40,41). They would not normally cause detectable brain lesions since bubbles smaller than 21 μm can pass through capillaries of the brain (42). Hills and James however, showed that such microbubbles impair the blood-brain barrier transiently in an experimental study (43). When Ama divers perform repetitive deep BH dives for prolonged periods, it is possible that microbubbles are released continuously from the venous side of tissues and reach the cerebral arteries via the pulmonary circulation to impair the blood-brain barrier. In addition, aggregation of microbubbles could form platelet thrombi and cause cerebral embolism (24). Brain MRI in reported Ama divers detected multiple cerebral infarcts in the terminal or border zone of brain arteries (9,10), suggesting that bubbles were most likely to flow into these areas and cause cerebral arterial embolism. Thus, microbubbles may explain brain involvement following BH diving.

IV. Pulmonary barotrauma

CAGE following pulmonary barotrauma remains a possible cause of DCI in BH divers. This barotrauma is known in situations at 1 ATA in which patients inhale to high lung volumes, and presumably have stretch injury (44). Some investigators noted that pulmonary barotrauma could be induced by BH diving. Bayne and Wurzbacher described a 21-year-old man who twice attempted to swim across a 25-yard pool at about 1.8 meters (6 ft) deep (45). After the second attempt, he surfaced complaining of headache, dizziness and tingling all over. He also complained "his lungs were hurting". Minutes later, he suffered a grand mal fit, became pulseless and apnoeic. Autopsy showed bleeding under the visceral pleura, mediastinal emphysema and large amount of air in the right heart and cerebral vessels. Kiyan et al. described 3 BH divers who were nonsmoking healthy men and had hemoptysis after surfacing (46). Boussuges et al. also showed 3 BH divers with hemoptysis secondary to alveolar hemorrhage who had taken aspirin before diving (47). Bruch has also reported 2 divers who developed mediastinal emphysema during BH diving to 4.5 meters (15 ft) (48). In cases of CAGE secondary to pulmonary barotrauma, one would have to assume that during the dive, air was trapped in a closed off part of the lungs before ascent. It is hypothesized that air trapping during diving can cause local distension of the lung and lung rupture on ascent producing air embolism (49). It has also been suggested that forceful inhalation to total lung capacity (TLC) might cause lung rupture by overdistension of a weak area in the lung without pressure changes (50).

V. Other factors

Smoking is well known to be a risk factor of cerebral infarction, and it has also been implicated to be a risk factor of DCI in compressed gas divers (22,51). Among Ama divers in the survey, diving accidents tended to occur in smokers (3), suggesting that smoking may also be a risk factor of DCI in BH diving.

TREATMENT

DCI is commonly managed by recompression therapy employing US Navy Treatment Tables (23-25). In contrast, there have been few reported cases of treatment of DCI in BH diving. Since two Ama divers had neurological disturbances two or more days after the onset of symptoms, they were not given recompression therapy (9). Nevertheless, using Navy Table 6, Wong treated a patient who developed DCI

following recreational BH diving with complete resolution despite a treatment delay of 4 days. Another, despite experiencing multiple episodes of symptoms, such as staggers, dizziness and nausea, did not seek treatment and symptoms resolved spontaneously usually within 12 hours, although his last episode lasted 36 hours. He had consulted his local physician who referred him to an ENT surgeon. CT scan was normal and no MRI was performed (6). Batle also used Table 5 or 6 for the treatment of neurological accidents in BH divers with complete resolution (4). The patients of Magno et al also made complete recovery, although only some received recompression treatment, some had diazepam and steroids, whilst others had no treatment at all (5). From the reported series above, whilst DCI in BH divers might be treated with Table 6, the most effective treatment is a subject for future investigation.

PREVENTION

As shown by the study on Taravana by Cross, divers on Mongareva Lagoon who made use of a longer surface interval did not suffer from this condition (8), it is therefore recommended that BH divers should adopt the following strategy to avoid developing neurological symptoms:

1. limit the Bottom Time per dive,
2. reduce the number of dives per day,
3. increase the surface interval.

CONCLUSIONS

Ama divers have experienced stroke-like symptoms in some districts in Japan, and brain MRI detected multiple infarctions in the terminal and border zones of the cerebral arteries, where perfusion is poorest. We conclude that repetitive deep BH dives can cause DCI in the brain. Venous bubbles passing through the heart or lungs may be a plausible mechanism. Although no therapeutic strategy has been established, therapy following the conventional DCI treatment patterns is recommended.

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