Review article

Diving-related disorders in commercial breath-hold divers (Ama) of Japan

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Abstract

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Decompression illness (DCI) is well known in compressed-air diving but has been considered anecdotal in breath-hold divers. Nonetheless, reported cases and field studies of the Japanese Ama, commercial or professional breath-hold divers, support DCI as a clinical entity. Clinical characteristics of DCI in Ama divers mainly suggest neurological involvement, especially stroke-like cerebral events with sparing of the spinal cord. Female Ama divers achieving deep depths have rarely experienced a panic-like neurosis from anxiety disorders. Neuroradiological studies of Ama divers have shown symptomatic and/or asymptomatic ischaemic lesions situated in the basal ganglia, brainstem, and deep and superficial cerebral white matter, suggesting arterial insufficiency. The underlying mechanism(s) of brain damage in breath-hold diving remain to be elucidated; one of the plausible mechanisms is arterialization of venous nitrogen bubbles passing through right to left shunts in the heart or lungs. Although the treatment for DCI in Ama divers has not been specifically established, oxygen breathing should be given as soon as possible for injured divers. The strategy for prevention of diving-related disorders includes reducing extreme diving schedules, prolonging surface intervals and avoiding long periods of repetitive diving. This review discusses the clinical manifestations of diving-related disorders in Ama divers and the controversial mechanisms.

Introduction

Decompression illness (DCI), a collective term for the dysbaric diseases decompression sickness (DCS) (caused by bubbles formed from dissolved gas) and arterial gas embolism (AGE) (caused by bubbles introduced to the arterial circulation by pulmonary barotrauma), is well known in compressed-air divers or caisson workers; however, the existence of this condition among breath-hold divers has been disputed by scientists and medical professionals. Since the 1960s, when Cross published articles on 'taravana', a diving syndrome of breath-hold pearl divers of the French Polynesian archipelago of Tuamotu,[1] the occurrence of serious neurological disorders after repetitive breath-hold diving has been widely debated. Nonetheless, a few cases of Japanese commercial or professional breath-hold (Ama) divers afflicted with neurological diving accidents were seen in the 1990s.[2,3] Moreover, a survey conducted in a village of Japan showed that more than half of the Ama divers had experienced neurological events related to diving work.[4] Since Wong summarised the clinical symptoms associated with breath-hold diving,[5] this condition has become more widely recognised.[6,7] Reviews of the symptoms of DCI in breath-hold and compressed gas diving show many manifestations in common,[5,8,9] though some differences in typical presentations may be identified (see Diving-induced disorders). Our aim here is to provide a review of clinical characteristics of diving-related disorders in Ama divers based on our previous case series and field studies, discuss uncertainty about the mechanisms, and propose strategies that could help to treat and protect these divers.

Ama divers

The commercial or professional breath-hold divers of Japan and Korea have been in existence for more than 2,000 years. These divers are scientifically and collectively called Ama (sea women and men) since their diving work was first published by a Japanese scientist in 1932.[10] The origin of the Ama diving practice is not well known; one theory contends that this diving tradition originated from male Polynesian pearl divers. The breath-hold diving profession was essentially for men in warm areas and it was introduced to cold waters of Japan and Korea where the ocean floor is rich in shellfish.[11] Women in these areas may have started to engage diving activities because their physiques are better suited to overcome cold stress.[11] The number of male Ama divers increased because of the advent of wetsuits for thermal protection in 1960s.[12]

In Japan, Ama divers start their profession at the age of 15–16 years and continue working for more than 20 years.[11,12] Divers older than 60 years are not rare; most male divers are between 30 and 50 years, and females between 40 and 60 years old.[13] The Korean Ama divers included men as well as women up to the 17th century; however, nowadays all the divers are female.[11] About 11,000 Ama divers dwell in Japan according to a questionnaire survey compiled in 1986, and 80% of them were male.[13] These Ama divers harvest the ocean floor by gathering seaweed, abalone, and sea urchins daily.

There are two types of diving methods for Ama divers: Cachido divers dive unassisted without any aids; and Funado divers use weights for descending.[11] Funado divers are either pulled up by assistants (completely assisted) or swim up without assistance (partially assisted). Cachido divers generally dive to depths of 3–10 metres of seawater (msw), and the diving depths of Funado divers are deeper; occasionally over 30 msw. In general, Ama divers began to work as Cachido in shallow water, and then graduated to become Funado. One reason for this is a need to develop more rapid middle ear pressure equalisation during fast descending in Funado divers. Most Funado divers are male, while almost all female divers are Cachido.

Working practice

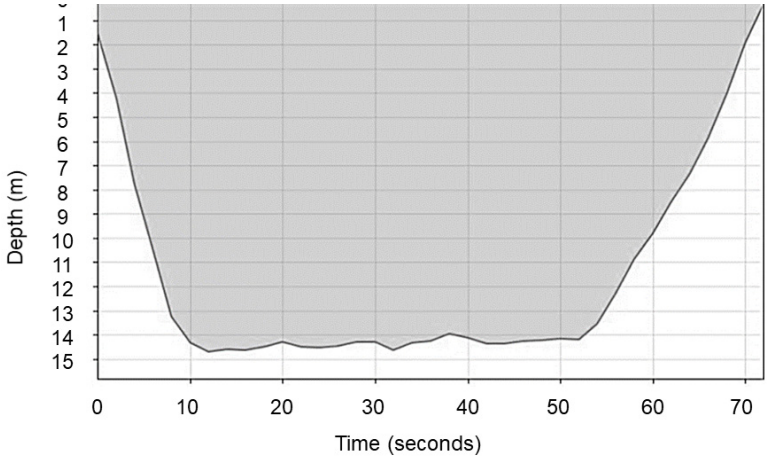
Traditionally, Ama divers were not equipped with any diving devices, except for their facemasks.[11] They wore traditional working clothing such as cotton bathing suits and loincloths. Ama divers make their dives using only light cotton suits mainly in the warm season, while Korean divers worked using only light cotton suits even in wintertime. Since wearing wetsuits has become popular among the fishery divers in Japan for more than half a century, longer-lasting and deep diving is possible even in winter. They wear wet suits and fins and carry weight belts to achieve neutral buoyancy (4–8 kg).

Cachido divers walk into the sea from the shore and swim to the diving grounds holding wooden tubs, and they swim unassisted up to 10 msw in depth from drifting tubs. In Cachido, swimming down and up requires significant energy. Funado dive from boats using iron weights (15–25 kg), and swim to the surface with or without assistance (Figure 1).

Figure 1

A male Funado dive photographed during breath-hold diving. His boat was equipped with a rolling machine to pull up his weight and a basket for seafoods (A). He descended to the floor using an iron weight (B), and swam to the surface without assistance (C)

Figure 2
Representative dive logger record showing the depth-time profiles of a single breath-hold dive



Their partners on the boats pull the weights and/or the Ama divers to the surface after each dive. Funado divers dive up to 30 msw and stay down longer owing to less exertion. Ama divers hyperventilate briefly during each surface interval and emit a pursed-lip whistle before descending. The divers take a couple of deep breaths, dive to the desired depth and stay 15–45 seconds at the bottom for harvesting (Figure 2). After the 30–120 second period of each dive, Ama make their next dive, typically after 30–60 seconds of surface rest, but occasionally up to 10 minutes or longer. Ama divers work 2–6 hours in the sea in one or two shifts per diving day; the duration of the morning shift is 2–4 hours and the afternoon shift is 1–2 hours, taking a lunch break of half an hour. Nowadays, diving depth is measured sonically using fish finders.

Ama divers use small cabins in their boats for frequent warming using propane stoves. After arriving at the diving locations, they stay in the cabin until they are warm enough to perspire. Another group of Ama divers who dive at shallow depths make fires to warm their bodies before and after each shift of dives, either on the beach or in cabins on the seashore.

There are marked differences in the harvesting time and days, and diving patterns and apparel in accordance with union rules. Local Japanese union rules in some areas do not allow the use of wetsuits in order to protect their natural resources from over-harvesting. Moreover, the divers are only allowed to work in specific harvesting seasons in all areas.

Diving-induced disorders

Our preliminary interview survey of 16 Funado divers showed that 13 of them had a history of diving accidents and that nine of them had experienced stroke-like neurological symptoms during or after more than 3 hours of repetitive dives.[4] In another study of Japanese Ama divers, 12 of 173 divers (11 of 29 Funado and only one of 144 Cachido, all male) had experienced stroke-like neurological events during or immediately after repetitive breath-hold diving (Table 1).[14] The most common symptoms were numbness in eight cases, dizziness in eight cases and motor weakness on one side (hemiplegia) in six cases. Other symptoms were speech disturbance, limb pain and visual disturbance. Dizziness was particularly common after continuous long-lasting dives in assisted Ama divers. Two of these 12 divers with neurological events also had severe musculoskeletal pain in the knee and limbs, but none of the divers had a skin rash. Many of the neurological disorders were transient and resolved completely in 10 divers. The other two had unresolved symptoms: one with a residual partial visual deficit and the other with sensory numbness of his hand. None of the divers experienced spinal cord disorders, which frequently occurs in compressed-air divers or workers.

Cross reported that out of 235 observed breath-hold pearl divers, 47 (20%) exhibited neurological symptoms known as 'taravana' diving syndrome at the end of 6 hours of diving per day during a three-week period in 1958 (Table 1).[1] The taravana syndrome included partial or complete paralysis in six divers, transient unconsciousness in three cases, mental disturbance and death in two cases, respectively. One of the divers undertaking 18 to 20 breath-hold dives to a depth of 40 msw in less than 2 hours, showed no life-threatening signs after diving work and during the return trip to the village. Another was pulled into the canoe in a semi-comatose state and died 2 hours later. Our case series have shown that Ama divers have experienced serious neurological manifestations which include unconsciousness, seizure, and/or brain stem involvement.[2,4,15,16] These serious neurological DCI events in Ama divers are consistent with the symptoms seen in taravana syndrome in Polynesian pearl divers.

Stroke-like brain involvement in DCI is common in Ama divers,[4,14] while spinal cord involvement, unlike in compressed-air divers and workers, is extremely rare. Typically, cerebral DCI manifests with unilateral sensory numbness or hemiplegia, disturbed speech, and/or visual deficit after repetitive dives exceeding 20 msw in depth executed over shifts longer than three hours.[2,3,15,16] Post-

Table 1
Diving events in Japanese Ama and Polynesian divers. Numbers in parentheses are numbers of cases

Diving group	Ama divers (11/29 assisted, 1/144 non-assisted) in 2009 [14]	Polynesian divers (47/235 assisted) in 1958 [1]
Symptoms (*n* cases)	Dizziness/vertigo/nausea (8) Sensory numbness (8) Hemiplegia (6) Speech disturbance (3) Limb pain (2) Visual disturbance (1)	Vertigo/nausea/mental anguish (34) Paralysis (6) Unconsciousness (3) Mentally affected (2) Death (2)
Dive profiles Mean (SD) or range	Dive depth (msw): 15.0 (3.3) Dive time (sec): 63.0 (16.4) Surface interval (sec): 26.0 (13.7) Length of diving shifts (hours): 5.5 (0.7) Rest time between shifts (min): 36.3 (15.5)	Dive depth (msw): 30–40 Dive time (sec): 90 Surface interval (min): 3–10 Length of diving shift (hours): 6

dive neurological events in Ama divers have often not been considered serious because symptoms resolved spontaneously within several hours or disappeared rapidly after hyperbaric therapy. However, in rare cases post-dive neurological events presented with altered level of consciousness or death.[1,4] One study has classified diving-related symptoms in breath-hold divers into two types; one is benign and quickly reversible, characterised by dizziness, vertigo, nausea, anxiety and fatigue, and the other is serious disease presenting with neurological and persistent disorders.[6] Ama divers experiencing neurological symptoms after repetitive dives require early diagnosis and treatment.

Another controversy is whether psychiatric disorders appear in breath-hold diving. Male Ama divers have reported no psychiatric disorders following diving work, although they may occasionally complain of anxiety during deep and long-lasting dives. In contrast, female Ama divers have suffered specific psychiatric disorders called 'Chiyamai'.[17] A survey of 44 female Ama divers noted that nine had mental disturbances related to anxiety attacks. On the particular island involved, the diving depths and durations were deeper and longer than in other areas,[18] and the diving patterns were similar to those of male Ama divers with diving accidents.[4,14] Although the clinical features of these diving related psychiatric episodes closely resemble those of some types of panic disorders, female divers did not have depersonalisation or de-realisation. The clinical symptoms included palpitation, dizziness or unsteady feelings, dyspnoea, nausea and/or hot flushes; palpitations being the most frequent among them. Several Ama divers who had experienced the illness could not dive and had to stop their diving work. Others who had recovered from the illness were unable to dive at great depths and always had to take anti-anxiety medicine prior to diving.

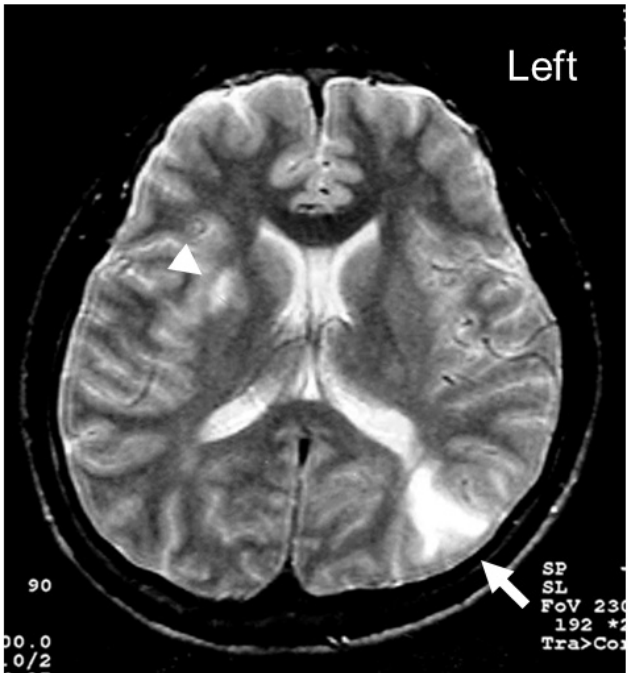
Except for the above specific cases, no diving-related psychiatric disturbances among female or male Ama divers have been reported where diving depths are shallower. However, Polynesian pearl divers frequently experienced mental anxiety as a form of taravana syndrome, and a few cases were affected with symptoms such as restlessness, irritability and altered comprehension.[1] Therefore, there is a possibility that deep and long-lasting repetitive breath-hold dives cause psychological conditions in Ama divers. Further studies are needed to investigate whether breath-hold diving affects psychological or mental functions.[19]

Brain imaging

There was no DCI reported among Ama divers until our group described two cases of cerebral infarction in 1998 occurring after repetitive dives to 15 to 25 msw.[2] There are now many reports of ischaemic brain lesions in breath-hold divers documented using magnetic resonance imaging (MRI). Brain MRIs in Ama divers with a history of DCI showed multiple cerebral infarcts in areas corresponding to the symptoms and elicited signs (Figure 3). The brain lesions are localised in the basal ganglia, internal capsule, and deep and superficial white matter.[20] In another study, we further investigated whether long-term breath-hold divers who exhibited cerebral symptoms and also those who did not would exhibit cerebral damage on MRI.[21] Twelve Japanese Funado divers with an average age of 54.9 (SD 5.1) years and diving experience of 29.8 (7.6) years were examined. Four had histories of transient diving-related neurological symptoms, and eleven demonstrated ischaemic cerebral lesions. These lesions were situated in the cortical and/or subcortical area (nine cases), deep white matter (four cases), the basal ganglia (four cases), and the thalamus (one case). Cerebral hyperintensities observed with T2-MRI are seen in healthy subjects, and the prevalence increases with older age, occurring in 10–20% of people aged around 60 years.[22,23] However, the higher prevalence of cerebral ischaemic lesions in Ama divers cannot be simply explained by aging.

The types of brain lesions in the Ama divers were identical to those seen in compressed-air divers or patients suffering iatrogenic AGE.[24–26] The ischaemic 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. They are so-called

Figure 3
Magnetic resonance imaging (MRI) of the brain in a male Ama diver with right homonymous hemianopsia. T2-weighted MRI obtained on the 5th day after the accident showed two increased signal intensities in the left occipital lobe (arrow) and the right basal ganglia (arrow head)



'low-flow' cerebral infarctions resulting from low perfusion pressure in the terminal supply areas, and may be due to cerebral AGE. The previously mentioned studies in Ama divers demonstrated that long-lasting breath-hold diving may cause damage to the brain, probably through accumulation of repeated transient cerebral arterial ischaemic injury. Moreover, another study suggests possible underestimation of the true damage to the brain.[27] In that study, five elite breath-hold divers with normal neurological examinations and brain MRIs all exhibited diffusely abnormal perfusion images of the brain using single photon emission tomography (SPECT) scans. In our series of 12 Ama divers, frontal lobe atrophy was found in two cases.[21] Recently, a possible impairment of cerebral autoregulation in elite competitive breath-hold divers has been reported.[28] Long-term repetitive breath-hold diving probably affects cerebral perfusion in a fashion that may increase susceptibility to local or diffuse ischaemia caused by AGE, and this is an important issue for future research.

Mechanisms of injury

The mechanisms of DCI incidents after repetitive breath-hold diving are poorly understood. A more recent study has demonstrated that deep repetitive breath-hold dives lead to endothelial dysfunction that may play a role in neurological DCI.[29] It was suggested that intravascular microparticles following breath-hold dives initiate a systemic inflammatory process including neutrophil activation.[30] It is possible that some forms of neurological DCI are a "*reversible cerebral vasoconstriction syndrome*" resulting from a transient segmental constriction of cerebral arteries.[31] However, some Ama divers with neurological DCI showed large multifocal ischaemic cerebral lesions on MRI studies.[20] These findings are more compatible with cerebral infarcts caused by a large gas or thrombotic emboli load.[26] Moreover, serious stroke-like neurological disorders were immediately relieved by recompression.[32] The dramatic and rapid response suggests the presence of bubbles in the cerebral arteries. Nitrogen accumulation in fat tissues increases throughout repetitive breath-hold diving despite quickly reaching a steady state in the brain, heart and viscera.[33]

Given high cerebral blood flow and consequently 'fast' nitrogen kinetics, the development of in situ bubbles in the brain is unlikely. The characteristics of diving accidents in Ama divers are that stroke-like brain involvement is common, and moreover their MRI findings suggest occlusion of cerebral arteries.[20] In one case of an Ama diver who suffered neurological symptom onset, computed tomography showed an air density area in the parietal lobe of the brain 3 hours later.[34] After repetitive breath-hold dives with short surface intervals, venous nitrogen bubbles may arise from the peripheral fatty tissues similar to the mechanisms seen in compressed-air diving. Some investigators have reported venous bubbles following repetitive breath-hold dives in Ama and spearfishing divers.[35–37] However, venous bubbles should be filtered by lung capillaries and would enter the cerebral arterial circulation unless they arterialize across a right-to-left shunt (RLS). Serious neurological events are usually induced in repetitive breath-hold diving, while the detection of venous bubbles is more difficult in breath-hold divers than in compressed-air divers.[29,35,36,38] Why lesions in breath-hold diving mainly involve the brain but not spinal cord is an unresolved question.[39]

Bubbles formed in the venous blood after long-lasting repetitive breath-hold dives, can cross from the venous side to the arterial side of the circulation (arterialization) in the presence of an intracardiac RLS and/or intrapulmonary arterio-venous shunt (AVS). While the proportion of cerebral ischaemic lesions were closely related to the presence of intracardiac RLS in compressed-air divers,[40,41] RLSs have not been detected in Ama and other breath-hold divers with brain involvement.[3,34,42,43] These reported cases suggest that neurological DCI in breath-hold divers cannot be explained only by intracardiac RLS, and alternative mechanisms have been suggested.[44] After repetitive deep breath-hold diving venous bubbles may be retained or trapped in the pulmonary arterioles. Then, when the divers continue with repetitive descents, the trapped bubbles may be compressed and therefore able pass through the pulmonary circulation. Arterialized bubbles might then expand during each ascent and accumulate in the terminal supply areas of the brain, border zones and watershed regions. It has

previously been suggested that this is the most likely hypothesis to explain cerebral involvement in Ama divers.[14]

Arieli has published a new theory of decompression bubbles developing from intravascular gas micronuclei on small distal arterial walls.[45] Elevation of nitrogen tension in the brain and blood results in enhanced nitrogen transfer to these nuclei which become bubbles. However, this hypothesis cannot explain why one or more large ischaemic lesions are not invariably accompanied by multiple small ones in the subcortical areas,[2,3,34] where arterial gas embolism typically involves the small arteries (average diameter, 30 to 60 microns).[46] Hypoxia following breath-holding may affect the opening of intrapulmonary AVSs which exist in normal humans.[47] We consider cerebral DCI in Ama divers is explained by arterialization of venous nitrogen bubbles and cerebral arterial gas embolism as the main mechanism. However, the pathophysiology is not clear and probably multifactorial.

Diffuse cerebral hypoperfusion or brain atrophy in breath-hold divers suggests that there is endothelial damage of cerebral arteries caused by micro-emboli-like microbubbles and microparticles. While microbubbles smaller than 22 microns in diameter can pass through cerebral capillaries,[48] it has been shown that this damages the blood-brain barrier.[49] In addition, others have suggested that microparticles play an important role in cerebral endothelial dysfunction after breath-hold diving.[29] There is a possibility that such micro-embolic particles induce cerebral hypoperfusion or brain atrophy.

Treatment

Hyperbaric oxygen treatment (HBOT) has a key role in treating bubble disease and appears to be effective for the hyperacute phase of iatrogenic arterial gas embolism,[50,51] although the beneficial effect has not been shown in acute ischaemic stroke not caused by bubbles.[52] Twenty two divers with cerebral symptoms of DCI who received HBOT within 6 hours all completely recovered.[53] In fact, the rate of spontaneous clinical recovery is high in patients with cerebral AGE following decompression although the improvement is not invariably sustained.[54] Another study described early normobaric oxygen breathing completely relieved or improved DCI symptoms in 65% of 1,045 cases.[55] In Ama divers, DCI symptoms typically reflect cerebral involvement (stroke-like symptoms). For the treatment of DCI in breath-hold divers, oxygen breathing should start as early as possible, followed by HBOT within 6 hours after the onset. Permanent brain injury may be prevented by early treatment so it must be emphasised in local diving villages that HBOT should start as soon as possible.

Prevention

Prevention of DCI is important for Ama divers. One somewhat radical preventative strategy previously proposed was to take a breath of oxygen immediately prior to the dive to minimise inert gas uptake.[56] However, this might risk oxygen toxicity, and many Japanese diving fishermen do not have access to oxygen for their diving work, even as a first aid strategy. A more practical approach is to take longer surface intervals. As shown in the study of taravana by Cross,[1] pearl divers in Mongareva Lagoon who spent at least 10 minutes at the surface between dives never developed this condition, whereas many divers in another lagoon using shorter surface intervals of three to five minutes experienced taravana. A short surface time would increase the risk of DCI, particularly when diving to deeper depths.[57]

Based on our surveys of Ama divers with neurological DCI,[4,14] multiple repetitive breath-hold dives to depths shallower than 20 msw for several hours with short recovery periods can lead to nitrogen accumulation in tissues analogous to the amounts found in compressed-air diving. A simulation of the diving pattern of Japanese Ama divers performing 30 dives to a depth of 20 msw over an hour found that nitrogen loading in the fat increased throughout repetitive breath-hold diving despite reaching a steady state value after five dives in the brain, heart and viscera.[33] Diving to 20 msw repeatedly for several hours would require an average surface-to-dive time (S/D) ratio of more than 0.8 to avoid development of DCI.[58] Our survey showed the S/D ratio in Ama divers with neurological disorders tended to be low in comparison to that of divers without events.[14] While dive depth, bottom time and duration of the diving pattern are well known as risk factors for DCI in breath-hold diving, short surface interval is a possible major cause in breath-hold dives to 10–20 msw. The risk of DCI in Ama divers can be decreased by taking a longer surface interval and a shorter diving shift of less than two hours. Twenty years ago, serious manifestations occasionally appeared in Ama divers in a district of Japan, but they became rare after educational meetings for Ama divers were introduced at the union.[59] However, neurological events happened in some divers whose shifts were longer than two hours,[15,16] and the union rule may need to include the time of single diving shift.

Conclusions

Repetitive breath-hold dives can cause decompression disorders characterised by stroke-like brain involvement. Brain MRIs of Ama divers showed symptomatic and/or asymptomatic ischaemic lesions typically situated in the terminal and border zones of cerebral arteries. The prevalent theory of brain involvement is that arterialised venous gas bubbles passing through right to left shunts may be a plausible mechanism. Although no therapeutic strategy has been established specifically for DCI in breath-hold divers, early oxygen breathing is recommended to help mitigate permanent brain ischaemic injuries. It is more important for Ama divers to protect themselves from the diving-related disorders by reducing hard diving schedules of long-lasting repetitive dives and short surface intervals.

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