

Repetitive breath-hold diving causes serious brain injury

HIDEKI TAMAKI^{1,2,5}, KIYOTAKA KOHSHI⁶, SHUICHI SAJIMA², JUNICHIRO TAKEYAMA³, TAKASHI NAKAMURA⁴, HIDEO ANDO⁵, TATSUYA ISHITAKE⁵

Divisions of ¹Emergency Medicine, ²Surgery and ³Internal Medicine, Tamaki Hospital, Hagi, Japan; ⁴Department of Surgery, Hagi Civil Hospital, Hagi, Japan; ⁵Department of Environmental Medicine, Kurume University School of Medicine, Kurume, Japan; ⁶Clinical Research Team, The Baromedical Research Foundation, Columbia South Carolina, USA

ABSTRACT

We report on a Japanese male professional breath-hold diver (Ama) who developed neurological disorders during repetitive dives to 22 meters of sea water. Each diving duration and surface interval were 40-80 seconds and 20-30 seconds, respectively. He suffered from sensory numbness of the right cheek, hand and foot, and double vision after more than two hours of consecutive dives. Magnetic resonance images of his brain showed multiple cerebral infarcts, and one of the lesions was situated in the brainstem. There is a possibility that repetitive deep breath-hold dives with short surface intervals can induce fatal accidents for divers.

INTRODUCTION

Decompression illness (DCI) following repetitive breath-hold (BH) dives has been disputed and debated in the medical fields. We've experienced some professional Japanese breath-hold divers (Ama) with DCI after dives (1,2). Moreover, our interview survey in a district on Mishima Island demonstrated the high prevalence of DCI in Ama divers (3). Since several similar cases have been reported from some areas, DCI in BH diving nowadays has been gradually known (4-6).

The diving events were mainly strokelike neurological disorders, in which the most common symptoms were unilateral motor weakness and sensory numbness. Because many of the symptoms resolve spontaneously – even without treatments – we initially considered DCI in BH diving as not so serious (4).

We report here a new young Ama diver suffering neurological events during repetitive BH dives with short surface intervals. This case had multiple infarcts of the brain, and one of the lesions was situated in the brainstem. The brainstem infarction can cause fatal cardiorespiratory disturbances (7), and we call divers' attention to the danger of repetitive deep BH dives.

CASE REPORT

A 34-year-old, right-handed man started unassisted BH diving at the age of 27; his diving depth was generally below 6 meters of sea water (msw). In 2007, he changed this diving type to partially assisted BH diving to gather a larger amount of expensive abalones. He wore a full wetsuit and carried a weight belt equivalent to neutral buoyancy (7-10 kg).

He descended passively from his boat to 10-25 msw using a 22-kg weight, and actively ascended without assistance. Another person on the boat had decided their diving grounds, the depth of which was measured by a fish finder. The diver engaged in daily diving work during the harvesting season, from December to October of the next year, in accordance with union rules. Moreover, the rule limited harvesting to Monday to Thursday, and the diving time from 8:00 a.m. to 3:00 p.m.

During that time, he repeatedly made 40- to 80-second dives with 20- to 30-second surface intervals between dives. He generally worked two shifts a day, one in the morning and the other in the afternoon, taking a 30- to 40-minute lunch break. He would normally spend five to six hours in the sea.

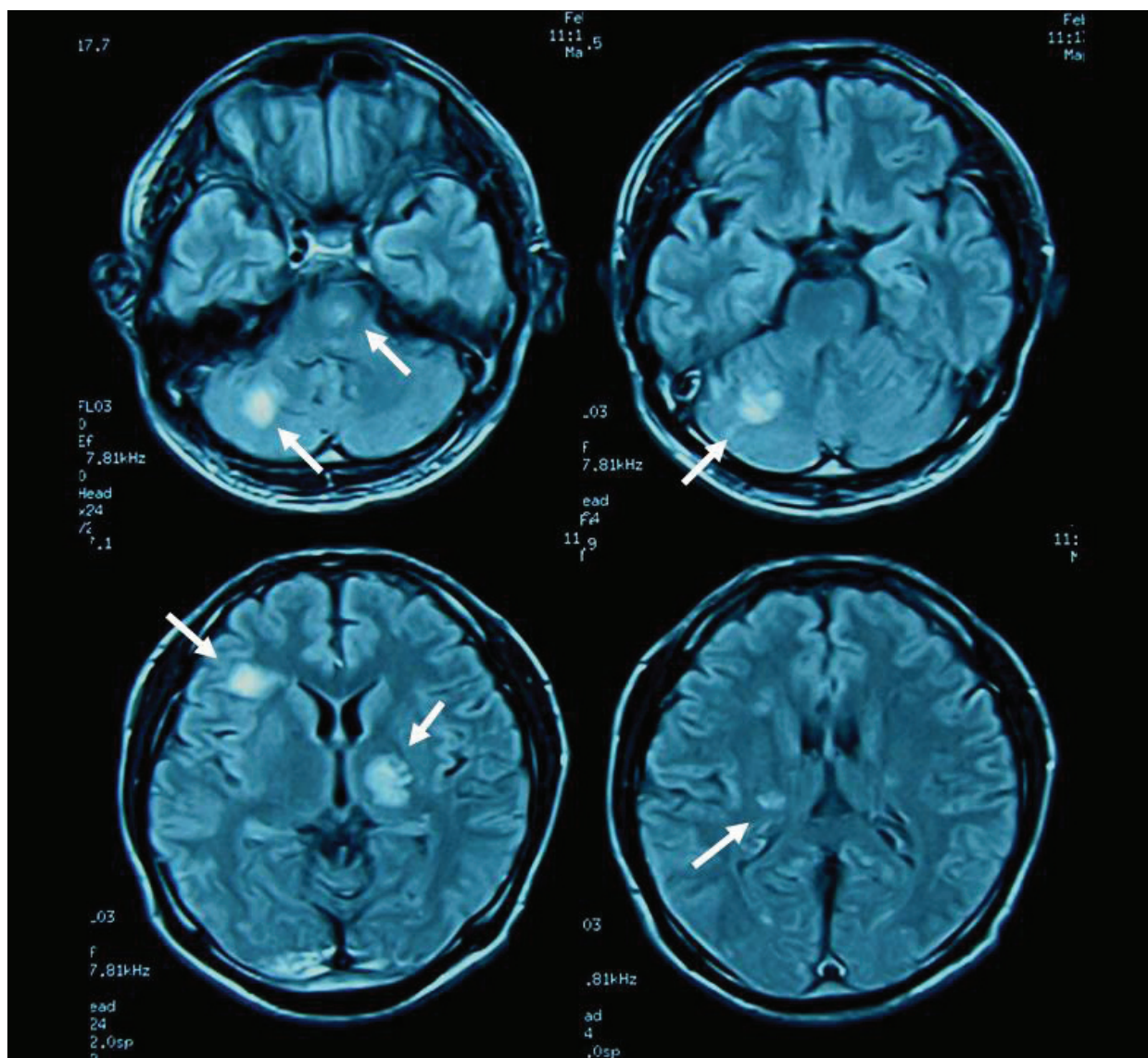


FIGURE 1. T2-weighted MR images (TR/TE: 8000/99) show hyper-intensity areas in the bilateral basal ganglia, right frontal lobe, cerebellar hemisphere and brainstem.

On February 27, 2007 he started diving at 8:00 a.m.; his diving depth was 22 msw, as usual. He stated that he did not work harder than usual, but he noticed paresthesia in the distal area of his right hand at around 10:00 a.m. After taking a 10- to 15-minute break, he continued diving until noon, but his paresthesia did not worsen.

He worked until 3:00 p.m. He reported feeling nauseous after diving and suffered serious dizziness and double vision. He could not walk steadily. After arriving home, he noticed paresthesia in his cheek and toe on the right side.

Since his symptoms continued without progression the next day, he visited a local hospital. A full blood count, standard biological examinations, chest radiograph and 12-lead electrocardiography were normal. Magnetic resonance (MR) imaging showed five hyperintense lesions in the left and right basal ganglia, right frontal lobe, pons and right cerebellar hemisphere (*Figure 1*, above). However, MR angiography showed no vascular abnormalities in the cerebral and cervical arteries.

A physician at this hospital diagnosed multiple cerebral infarcts and referred him to a hyperbaric

center. Although he received recompression therapy according to U.S. Navy Table 6 on that day, his symptoms did not improve after the treatment. His recompression therapy on the third day was U.S. Navy Table 5, and he received daily hyperbaric oxygen therapy for 10 days.

Paresthesia in his cheek and toe disappeared gradually two and four weeks respectively after the accident, and his double vision resolved within one month. However, residual numbness in his hand continued until late March 2008.

DISCUSSION

This Ama diver is a typical case of DCI following multiple repetitive BH dives. Although his diving depth and duration were similar to those of other Ama divers with DCI (1-3), his surface interval was shorter and the symptoms appeared after two hours of continuous diving sessions.

The short surface interval in BH diving is a possible major risk factor of DCI, including diving depth and duration. Moreover, the other serious problem is that one of the brain lesions was situated in the brainstem. From the standpoint of its location (7), repetitive deep BH diving is not as benign as we would like to think.

The clinical characteristics of DCI in BH diving are as follows:

- 1) main symptoms and signs are based on brain involvement; and
- 2) many resolve spontaneously without recompression therapy (3,4).

In addition, it is extremely rare for DCI in BH diving to be accompanied by musculoskeletal pains and spinal involvement, which occur frequently in compressed-air divers with DCI (5). However, the symptoms and signs of brain involvement are similar or the same in both BH and compressed-air diving (3,8,9).

The MRI findings of DCI in BH divers suggest features of circulatory disturbance of the cerebral arteries. Ischemic lesions in the basal ganglia were situated in the terminal zone, and lesions involving deep or superficial white matter corresponded to border zone or watershed regions (10). These neuro-radiological findings in BH divers with DCI

are in agreement with those in compressed-air divers (4,8,9) and are so-called low-flow cerebral infarctions as a result of low perfusion pressure in terminal supply areas. Although cerebral vessels were examined by MR angiography in this case and other Ama divers treated in the acute stage, no obvious abnormality was detected in the cerebral arteries corresponding to the infarcts and in the cervical and vertebral arteries (2). These results suggest transient circulatory insults.

We consider that cerebral events following diving can be ascribed to multifocal arterial gas embolism. Neurological events have not been considered to be serious in BH divers because the symptoms disappeared rapidly after recompression therapy or tended to resolve spontaneously within a short period (4). In general, the main clinical features of cerebral infarction in the border or terminal zone are transient ischemic attacks or mild, even though their lesions are considerably large on CT or MRI (10). However, as Cross reported, at the end of a six-hour working day, 47 (20%) of 235 BH pearl divers developed symptoms of “Taravana” diving syndrome, which included serious conditions such as transient unconsciousness in three, and mental disturbance and death in two (11). One of two victims was pulled into the boat semi-conscious and died two hours later. In addition, our investigations documented that one of the nine Ama divers with neurological disorders had transient crossed sensory numbness in the right body and left face (3). Their symptoms and courses are compatible with clinical images of brainstem stroke.

The mechanisms of DCI in BH diving are still uncertain. Although a strong correlation between high venous gas emboli levels and the risk of DCI is well known in compressed-air diving (12), cerebral insults are not common compared to spinal cord disorders. In contrast to compressed air diving, the neurological events are due to brain involvement alone despite the difficulty of detecting venous bubbles after BH diving (4,13).

Regarding the discrepancy between cerebral DCI and venous bubbles in BH diving, we have previously considered that the main mechanism of cerebral

lesions is microbubbles passing through the lungs (2-4). However, it is difficult to explain such microbubbles causing cerebral DCI in BH divers, because compressed-air divers have a low prevalence of cerebral DCI despite high levels of venous bubbling, including a large number of microbubbles. Other possible mechanisms, such as cardiopulmonary shunts or pulmonary barotraumas, could be considered (4,5).

Now we have a new hypothesis that “trapped bubbles” in the lungs cause cerebral DCI in repetitive BH diving (14). The high prevalence of cerebral accidents among the Ama divers surveyed was considerably higher than the rate of intracardiac shunts in healthy adults (8); and, moreover, a right-to-left shunt could not be detected in an Ama diver with cerebral DCI using 2D Doppler echocardiography (2). Although venous bubbles larger than 21 μm in diameter cannot cross the pulmonary circulation and are trapped in smaller pulmonary arteries (15), their passage through the lungs is possible during dives because the “trapped” bubbles are compressed at the bottom under increasing pulmonary arterial pressure caused by the accumulation or lodging of bubbles (16).

On the surface, arterialized microbubbles could be expanded due to decompression and cause cerebral gas embolism. Multiple and large cerebral infarcts are probably formed by means of repeating the phenomena.

In conclusion, repetitive deep BH dives with short surface intervals tend to induce cerebral DCI, and lesions in the brainstem are serious for divers. Since Ama divers tend to harvest the deeper ocean floor, we recommend longer surface breathing time to prevent DCI. Although standard treatments for DCI in BH diving have not been established, the divers with neurological disorders should receive normobaric or hyperbaric oxygen immediately. ■

References

1. Kohshi K, Kinoshita Y, Abe H, Okudera T. Multiple cerebral infarction in Japanese breath-hold divers: two case reports. *Mt Sinai J Med* 1998; 65: 280-283.
2. Kohshi K, Katoh T, Abe H, Okudera T. Neurological accidents caused by repetitive breath-hold dives: two case reports. *J Neurol Sci* 2000; 178: 66-69.
3. Kohshi K, Katoh T, Abe H, Okudera T. Neurological diving accidents in Japanese breath-hold divers: a preliminary report. *J Occup Health* 2001; 43: 56-60.
4. Kohshi K, Wong RM, Abe H, Katoh T, Okudera T, Mano Y. Neurological manifestations in Japanese Ama divers. *Undersea Hyperb Med* 2005; 32: 11-20.
5. Wong RM. Decompression sickness in breath-hold diving. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving. Proceedings of the Undersea and Hyperbaric Medical Society/ Divers Alert Network 2006 June 20-21 Workshop*, pp119-129.
6. Schipke JD, Gams E, Kallweit O. Decompression sickness following breath-hold diving. *Res Sports Med* 2006; 14: 163-178.
7. Evans DE, Kobrine AI, Weathersby PK, Bradley ME. Cardiovascular effects of cerebral air embolism. *Stroke* 1981; 12: 338-344A.
8. Warren LP Jr, Djang WT, Moon RE, et al. Neuroimaging of scuba diving injuries to the CNS. *AJR Am J Roentgenol* 1988; 151: 1003-1008.
9. Reuter M, Tetzlaff K, Hutzelmann A, et al. MR imaging of the central nervous system in diving-related decompression illness. *Acta Radiol* 1997; 38: 940-944.
10. Mull M, Schwarz M, Thron A. Cerebral hemispheric low-flow infarcts in arterial occlusive disease. Lesion patterns and angiomorphological conditions. *Stroke* 1997; 28: 118-123.
11. Cross ER. Taravana diving syndrome in the Tuamotu diver. In: Rahn E, Yokoyama T, eds. *Physiology of Breath-Hold Diving and the Ama of Japan*. Washington D.C.: Natl. Acad. Sci.-Natl. Res. Council publ. 1341, 1965: 205-219.

12. Gardette B. Correlation between decompression sickness and circulating bubbles in 232 divers. *Undersea Biomed Res* 1979; 6: 99-107.
13. Boussuges A, Abdellaoui S, Gardette B, Sainty JM. Circulating bubbles and breath-hold underwater fishing divers: a two-dimensional echocardiography and continuous wave Doppler study. *Undersea Hyperb Med* 1997; 24: 309-314.
14. Kohshi K, Mano Y, Wong RM. Manifestations of decompression illness in Japanese Ama divers. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving. Proceedings of the Undersea and Hyperbaric Medical Society/ Divers Alert Network 2006 June 20-21 Workshop*, pp119-129.
15. Butler BD, Katz J. Vascular pressures and passage of gas emboli through the pulmonary circulation. *Undersea Biomed Res* 1988; 15: 203-209.
16. Brubakk AO. Transport of bubbles through a PFO. *Aviat Space Environ Med* 2000; 71: 352.

□