

Field Study

Neurological Diving Accidents in Japanese Breath-Hold Divers: A Preliminary Report

Kiyotaka KOHSHI^{1,4}, Takahiko KATO³, Haruhiko ABE² and Toshio OKUDERA⁵

¹Department of Neurosurgery and ²Internal Medicine, School of Medicine, ³Department of Health Information Science, School of Health Sciences, University of Occupational and Environmental Health, ⁴Division of Hyperbaric Medicine, University Hospital of Occupational and Environmental Health, and ⁵Department of Radiology, Akita Research Institute of Brain and Blood Vessels

Abstract: Neurological Diving Accidents in Japanese Breath-Hold Divers: A Preliminary Report: Kiyotaka KOHSHI, et al. Department of Neurosurgery and Division of Hyperbaric Medicine, University of Occupational and Environmental Health—There have been no reports of diving accidents involving Korean or Japanese professional breath-hold (ama) divers except for our 2 recent cases. We investigated a direct interview survey on their village to determine whether other ama divers had experienced any accidents during or after dives. Interview responses were retrospectively obtained from 16 partially assisted male ama divers belonging to the Utsu Union on Mishima Island, Yamaguchi Prefecture in Japan. We questioned the divers about diving history (diving depth, length of the diving shift, number of dives and diving accidents), medical history (hypertension, cardiac arrhythmia, diabetes mellitus, and cerebrovascular diseases) and health habits (smoking and alcohol consumption). Nine of 16 ama divers previously had stroke-like neurological accidents, in which the most common symptom were unilateral motor weakness [7 cases] and sensory abnormalities [4 cases]. All of these neurological problems had manifested transiently. In addition, 13 of 16 divers had occasional episodes of dizziness, nausea and/or euphoria during such dives. Repeated deep breath-hold dives may be harmful to the divers and cause brain involvement.

(J Occup Health 2001; 43: 56–60)

Key words: Diving accident, Stroke, Cerebral infarction, Breath-hold diving, Japanese male divers

Dysbaric diving accidents, such as barotrauma and decompression sickness occur in compressed air divers or workers. Barotrauma involves tissue damage resulting from a change in the volume of entrapped gas during decompression, and pulmonary barotrauma leads to cerebral arterial gas embolism. Decompression sickness occurs due to the formation of inert gas bubbles, and the symptoms vary according to local damage and intravascular blockage. Since the 1960s, however, when Cross reported “Taravana” in Polynesian pearl divers¹, whether or not breath-hold dives cause diving accidents has been a controversial issue^{2–4}.

Professional breath-hold divers of Korea and Japan are known in the scientific literature as “ama”^{2,5}. Korean ama divers are all women, and in Japan where many ama divers are men, female divers nevertheless are predominant. Although diving accidents have not been reported in ama divers, we recently encountered 2 Japanese ama divers with a history of such accidents⁶. Dysbaric diving accidents are theoretically possible after repeated deep breath-hold dives but the occurrence of such accidents is debatable because the symptoms are unknown in other populations of ama divers in Korea and Japan⁴. It is important to estimate the diving risk for breath-hold divers, and we therefore conducted an interview survey in an island diving village, and herein report on an interview questionnaire survey to determine whether any diving accidents had occurred among Japanese breath-hold divers during or after dives.

Subjects and Methods

Although there were a small number of female ama divers on Mishima Island, Yamaguchi Prefecture in Japan a few decades ago, they are no longer to be found. There are 2 types of ama divers in Utsu village on this island: unassisted ama divers (called *cachido* in Japanese) and partially assisted divers (*funado*). Cachido divers

Received July 21, 2000; Accepted Sept 30, 2000

Corresponding to: K. Kohshi, Department of Neurosurgery and Division of Hyperbaric Medicine, University of Occupational and Environmental Health, 1–1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan

generally dive to depths of 3–6 meters in seawater (msw). In contrast, the diving depths of funado divers are very deep - occasionally over 30 msw. Twenty-six male ama divers were engaged in daily diving work during harvesting season (from January to October). Nineteen were funado divers, and the subjects of this study were selected from among these funado divers.

In 1998 a standard interview was carried out by one neurosurgeon. The ama divers were questioned about their diving patterns including depth, length of diving shift, and number of dives. From each diver we obtained a history of diving-related physical and neurological events such as skin rash, joint pain, dizziness, nausea, euphoria, motor and sensory involvement, unconsciousness and other clinical manifestations. Diving accidents were defined as events with onset during or less than 48 hours after the last dive⁷. A separate questionnaire for the divers included questions on their

smoking habit, alcohol consumption, and the presence of hypertension, cardiac arrhythmia, diabetes mellitus and cerebrovascular diseases.

Divers who had had any neurological symptoms were interviewed about the courses of these symptoms. In addition, a standard neurological examination was performed by the same neurosurgeon according to a previously defined protocol. The findings were classified as either normal or abnormal. Muscle stretch reflexes were graded as symmetric, asymmetric, or increased. Abdominal muscle reflexes were graded as normal, asymmetric, or absent. Planter reflexes were graded as symmetric, asymmetric or inverted⁸.

Results (Table 1)

We interviewed 16 of 19 funado divers. The mean subject age was 46 yr (range 39 to 63). All ama divers started their profession at the age of 15–16 yr and had

Table 1. Diving patterns and neurological dysfunction in 16 Japanese male ama divers

Case / age	Diving depth (msw)	Number of dives (/h)	Diving time (h) (morning, afternoon)	Neurological symptoms of diving accidents
1/46	10–15	30	5 (3, 2)	left hemiparesis 10 min after 4 h diving (35)*; dizziness
2/39	15–23	20	4.5 (3.5, 2)	crossed sensory numbness in the right body and the left face 5 min after 4 h diving (29); euphoria, dizziness and/or nausea
3/43	15–22	20–30	5.5 (3.5, 2)	right hemiparesis and hemisensory numbness 10 min after 4 h diving, and loss of consciousness 2 h 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 h diving (41); dizziness and/or nausea
5/49	10–20	30	5 (4, 1)	right hemiparesis during 5 h diving (37); nausea
6/41	10–20	20	4.5 (3, 1.5)	left hemiparesis 5 min after 3.5 h diving (25); left lower limb monoparesis during 4 h diving (30); euphoria, dizziness and/or nausea
7/44	10–15	30	5 (3.5, 1.5)	left hemiparesis during 3.5 h diving (25); dizziness
8/41	10–15	30	4.5 (3, 1.5)	right hemisensory numbness during 3.5 h diving (25); dizziness and/or nausea
9/39	15–25	25	5 (3.5, 1.5)	right hemiparesis with facial involvement during 3.5 h 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.

continued working for more than 20 yr. They wore wet suits and fins and carried a weight belt equivalent to neutral buoyancy (4–8 kg). The divers descended passively with a 15–25 kg weight to the bottom, and then swam to the surface without assistance. They worked 4–5 h in the sea in two shifts a day, taking a lunch break of ~30 min. The duration of the morning shift was 3–4 h and that of the afternoon shift was 1–2 h. Diving depths and numbers were 10–30 msw and 20–40 times/h, respectively. Nowadays depth is measured mechanically with fish finders. These diving patterns were almost the same as those of our previous subjects⁶. Many of the divers explained that more than 3 h of diving tended to prolong both diving depth and time without breathing difficulty.

Thirteen of 16 divers had occasionally felt such symptoms as dizziness, nausea, and/or euphoria after more than 3 h of repetitive dives. Moreover, 9 of the 16 divers had experienced stroke-like neurological accidents such as motor weakness, numbness, dysarthria, and/or unconsciousness. These accidents happened during or after more than 3.5 h of dives, but they did not always follow the above symptoms. Motor weakness, the most common neurological symptom, had occurred in unilateral limbs in 7 divers. The second most common symptom was numbness in 4 divers, and 1 diver had experienced a crossed sensory disturbance characterized by numbness on opposite sides of the face and body. The most serious accident involved unconsciousness in 1 diver. However, these neurological disorders were transient and improved completely between 10 min and 4 wk after onset. None of the divers had experienced skin rash or joint pain. Although all ama divers had the habit of alcohol consumption, the prevalence of smoking was higher in divers reporting stroke-like diving accidents, but it was not significant (Fisher's exact test, $P=0.106$). There were no divers with hypertension, diabetes mellitus, or arrhythmia.

Discussion

We found that 13 of 16 ama divers had a history of diving accidents and that 9 of them had experienced stroke-like neurological symptoms after breath-hold dives. All divers had neither vascular diseases nor risk factors for stroke, and the events had happened during and/or after repetitive deep dives. There is therefore a strong possibility that the breath-hold dives induced brain damage in the Japanese ama divers.

In the Tuamotu Archipelago in the South Pacific, professional breath-hold divers make 40–60 dives/d to a depth of 30 msw or more, spend an average of 2 min/dive underwater, and remain at the surface for 3–4 min between dives^{1, 9}. Many of them have suffered "Taravana" diving syndrome, the symptoms and signs of which include vertigo, nausea, partial or complete

paralysis, unconsciousness, and even death⁹. Motor paralysis was the most common neurological sign, but it was transient and disappeared completely in almost all divers. Many diving physiologists suspect that these disorders are compatible with decompression sickness. Our findings of neurological disorders in Japanese ama divers were similar to the observations in "Taravana" diving syndrome. Characteristically, these diving accidents were limited to central nervous system disturbances, but were not accompanied with skin rash or joint pain, which occur frequently in decompression sickness in compressed air divers¹⁰. The diving patterns in this village were very similar to those in another region of Japan⁴, and those Japanese ama divers might also have had diving accidents but it has not been investigated.

The mechanisms of brain damage after repeated breath-hold dives are poorly understood. Paulev³ concluded that the disorder caused by repetitive breath-hold dives was decompression sickness because it was immediately relieved by recompression. Such symptoms as euphoria and dizziness after repeated dives suggest an increase in nitrogen partial pressure in the brain causing neurological dysfunction, i.e., nitrogen narcosis¹¹. Because of possible nitrogen accumulation after repeated deep breath-hold dives^{3, 12, 13}, nitrogen gas bubbles may form in the intra- and extravascular spaces of the brain during or after decompression. But Olszowka and Rahn found that nitrogen accumulation in the fat increases throughout repeated breath-hold dives despite quickly reaching a steady state in the brain¹². Therefore, regarding nitrogen kinetics, diving accidents limited to brain involvement such as *de novo* bubbles generated in the brain and cerebral vessels are difficult to explain.

The cerebral lesions demonstrated by MRI in 1 ama diver, on the other hand, were located in the subcortical white matter and the centrum semiovale (Fig. 1). Our 2 previously reported patients also had multiple cerebral lesions in the same areas as well as in the basal ganglia⁶. These findings were compatible with occlusion of cerebral arteries. Gas bubbles released from the peripheral fatty tissues probably caused the cerebral arterial embolisation. In fact, silent venous gas bubbles were recorded in a Japanese ama diver after a 51-min period of 30 dives to 15 msw¹⁴. However, venous gas bubbles do not induce brain involvement unless they migrate into the arterial circulation via a pulmonary or intracardiac shunt. Because mammalian lungs usually constitute a competent filter for bubbles larger than 21 μm in diameter^{15, 16}, smaller bubbles would be harmless to the brain. Hills and James however, showed that such microbubbles impair the blood-brain barrier transiently¹⁷. Although our results do not show the causes of diving accidents, we propose that in a susceptible individual venous gas emboli may enter the systemic circulation and cause cerebral arterial embolisation through the

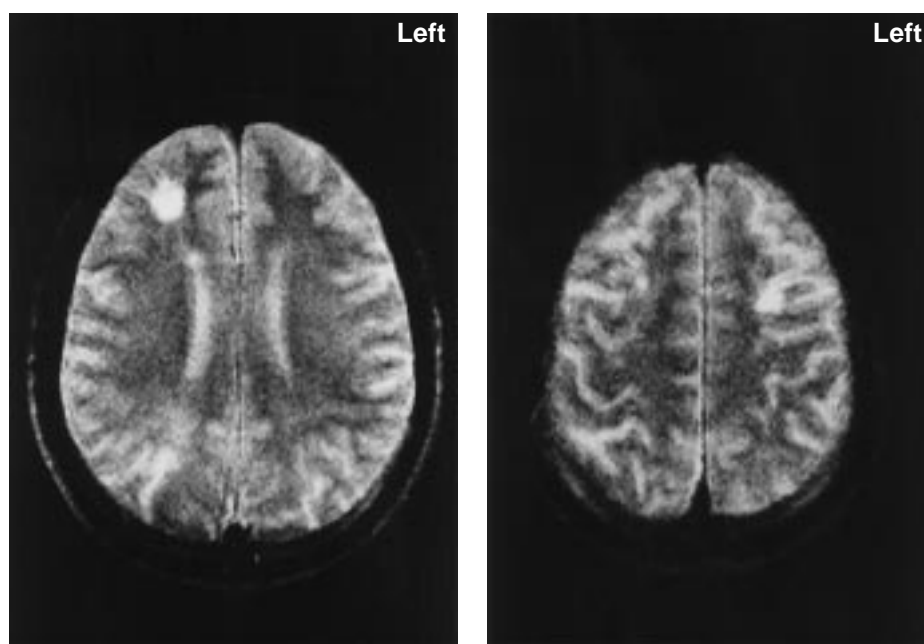


Fig. 1. Axial T2-weighted MR images for Case 3 diver who gave informed consent. He had a history of left-sided motor and sensory involvement and unconsciousness 9 years earlier. Despite the absence of neurological abnormalities, his MRI scans revealed multiple brain lesions.

pulmonary microcirculation. In addition, other causes or risk factors for diving accidents are smoking, patent foramen ovale, air embolism following pulmonary barotrauma, and repeated severe hypoxemia immediately after diving¹⁸⁻²⁰. Smoking in particular is suspected as a risk factor for dysbaric accidents in compressed air divers²¹ and probably plays an important role as a cofactor in accidents in breath-hold divers. The precise nature of this mechanism is a subject for future study.

In conclusion, our interview results for Japanese ama divers suggest that long-term repeated breath-hold dives appear to be harmful and cause cerebral injuries.

References

- 1) Cross ER. Taravana. *Skin Diver Magazine* 1962; 11: 42-45.
- 2) Hong SK, Rahn H, Kang DH, Song SH, Kang BS. Diving pattern, lung volumes, and alveolar gas of the Korean diving women (ama). *J Appl Physiol* 1963; 18: 457-465.
- 3) Paulev P. Decompression sickness following repeated breath-hold dives. *J Appl Physiol* 1965; 20: 1028-1031.
- 4) Mohri M, Torii R, Nagaya K, et al. Diving patterns of ama divers of Hegura Island, Japan. *Undersea Hyperbaric Med* 1995; 22: 137-143.
- 5) Hong SK, Rahn H. The diving women of Korea and Japan. *Sci Am* 1967; 216: 34-43.
- 6) 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.
- 7) Wilmshurst PT, Byrne JC, Webb-Peploe MM. Relation between interatrial shunts and decompression sickness. *Lancet* 1989; 1: 1302-1306.
- 8) Skre H. Neurological signs in a normal population. *Acta Neurol Scand* 1972; 48: 575-606.
- 9) 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.
- 10) Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. *N Engl J Med* 1992; 326: 30-35.
- 11) Behnke AR, Thomson RM, Motley EP. The psychologic effects from breathing air at 4 atmospheres pressure. *Am J Physiol* 1935; 112: 554-558.
- 12) Olszowka AJ, Rahn H. Gas store changes during repetitive breath-hold diving. In: Shiraki K, Yousef MK, eds. *Man in stressful environments -diving, hyper-, and hypobaric physiology*. Illinois: Charles Thomas, 1987: 41-56.
- 13) Spencer MP, Okino H. Venous gas emboli following repeated breath hold dives. *Fed Proc* 1972; 31: 355.
- 14) Radermacher P, Falke KJ, Park YS, et al. Nitrogen tensions in brachial vein blood of Korean ama divers. *J Appl Physiol* 1992; 73: 2592-2595.
- 15) Butler BD, Hills BA. The lung as a filter for microbubbles. *J Appl Physiol* 1979; 47: 537-543.
- 16) Butler BD, Katz J. Vascular pressures and passage of

- gas emboli through the pulmonary circulation. *Undersea Biomed Res* 1988; 15: 203–209.
- 17) Hills BA, James PB. Microbubble damage to the blood-brain barrier: relevance to decompression sickness. *Undersea Biomed Res* 1991; 18: 111–116.
 - 18) Hickey DD, Lundgren CEG. Breath-hold diving. In: Schilling CW, Carlston CB, Matthias RA, eds. *The physician's guide to diving medicine*. New York: Plenum, 1984: 206–221.
 - 19) Qvist J, Hurford WE, Park YS, et al. Arterial blood gas tensions during breath-hold diving in the Korean ama. *J Appl Physiol* 1993; 75: 285–293.
 - 20) Knauth M, Ries S, Pohmann S, et al. Cohort study of multiple brain lesions in sport divers: Role of a patent foramen ovale. *Br Med J* 1997; 314: 701–705.
 - 21) Wilmshurst P, Davidson, O'Connell G, Byrne C. Role of cardiorespiratory abnormalities, smoking and dive characteristics in the manifestations of neurological decompression illness. *Clin Sci (Colch)* 1994; 86: 297–303.