A survey of neurological decompression illness in commercial breath-hold divers (Ama) of Japan

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ABSTRACT

A survey was conducted in the northern district of Yamaguchi, Japan to determine the relationship between neurological diving accidents and risk factors among commercial breath-hold divers (Ama). A questionnaire was distributed to 381 Ama divers who are members of the Ama diving union. We sought information on their dive practices (depth of single dive, single dive time, surface interval, length of dive shifts, lunch break) and the presence or absence of medical problems, such as hypertension, cardiac arrhythmia, diabetic mellitus and other issues. Of the 381 Ama divers, 173 responded (45%): 29 were *Funado* (assisted–descent using weights) and 144 *Cachido* (unassisted) divers. Twelve had experienced strokelike symptoms during or after repetitive breath-hold diving; 11 were assisted and one unassisted (*Funado* vs. *Cachido*). Only two of 12 divers with neurological diving accidents had musculoskeletal symptoms. Neurological events were significantly correlated with dive depth, dive time, and surface interval; however, they were not related to medical history. Neurological diving accidents are more likely to happen among assisted Ama divers than unassisted ones. Repetitive breath-hold diving with a deep dive depth, long dive time, and short surface interval predisposes divers to decompression illness, which characteristically manifests as cerebral stroke.

INTRODUCTION

Commercial or occupational breath-hold harvest divers who reside along the coast and islands of Japan and Korea are collectively called Ama divers (1,2). There are two types of diving techniques: one is unassisted Ama, known as *Cachido*: these Ama dive without any aids. The other group is known as *Funado*, who use weights for descents. Moreover, *Funado* divers are either pulled up by assistants (completely assisted) or swim up without assistance (partially assisted).

In Korea Ama divers are female, whereas male Ama divers are mainly found in Japan. The number of Ama divers in Japan increased from 13,000 (65% male) in 1977 to 16,500 (80% male) in 1986. During this period, wetsuits were introduced for thermal protection (3,4). Although a current survey has not been performed, it is a perception that the numbers of Ama, especially female divers, has dwindled over the past two decades. The occupation of male Ama divers has become popular in Japan due to the high financial reward and the high market price of their marine invertebrate harvest.

Although decompression illness (DCI) is well known in compressed-air divers or caisson workers, the existence of this condition among breath-hold divers has been disputed by scientists and medical professionals. Since the 1960s, when E.R. Cross published articles on "Taravana," a diving syndrome of breath-hold pearl divers of the French Polynesian archipelago of Tuamotu (5,6), the occurrence of serious neurological disorders after repetitive breath-hold diving has been widely debated. Nonetheless, a few cases of Japanese Ama divers afflicted with neurological diving accidents were seen in the 1990s (7,8). A survey conducted in a

village in Yamaguchi Prefecture, Japan, showed that more than half of the *Funado* were found to have had neurological events related to diving (9). Furthermore, several cases of DCI have also been reported in professional and amateur breath-hold divers, some using submarine scooters to achieve great depths (10-12); therefore, DCI following breath-hold diving does indeed appear to exist as a clinical entity (13). We are not aware of any study addressing the issue of how often neurological diving accidents occur among Japanese Ama divers, and whether any risk factors predispose them to developing this illness.

METHODS

Participants

The participants in the present study were 381 Ama divers who belong to the diving union of Abu-Hagi district in the northern area of Yamaguchi Prefecture, Japan (34°29‑47''N and 131°7‑24''E). The union includes 14 villages of Ama divers on two archipelagos and on the coast of the main island. There were 346 male and 35 female divers (91% male), whose age ranged from their teens to 80s. Thirty-nine participants were partially assisted; they were all male. In contrast, all female Ama divers were unassisted.

All 381 divers engaged in daily diving work during the harvesting season (from December 21 to October 20 each year); their working week was from Monday to Thursday. In accordance with their regulations, they began diving at 09:00 and ceased at 15:00. They all wore wetsuits and fins and carried a weight belt to achieve neutral buoyancy. They worked two shifts a day; there is one shift in the morning and two in the afternoon, with a lunch break in between.

All assisted Ama divers descended passively with a weight, and swam to the surface without assistance. Dive depths were previously estimated using the length of outstretched arms; these days however, depths are measured using echo sounders.

Procedures

In April 2009, a questionnaire was distributed to all 381 Ama divers with a letter explaining the purpose of the present study and asking that the divers complete the questionnaire voluntarily. To ensure confidentiality, no names of any Ama divers were on the questionnaires. Moreover, the purpose of this study was explained to the chairperson in charge of the survey at the diving union, who also distributed and collected the anonymous answers to the questionnaires. The study protocol was approved by the human research ethics committee of Kurume University School of Medicine, Japan.

The demographic information collected was on diving techniques and average diving profiles, including depth of a single dive, single dive time, surface interval, length of dive shifts (morning, afternoon and total), and rest time for lunch; also included were requests for divers' current medical history (the presence of hypertension, cardiac arrhythmia, diabetes mellitus and others), and their past medical history (ischemic heart disease, cerebrovascular diseases and others).

An additional questionnaire was distributed and collected concerning diving-related physical and neurological disorders, such as skin rash, joint pain, dizziness, vertigo, nausea, euphoria, motor weakness, sensory numbness, visual field deficits, speech difficulty, convulsive seizure and/or unconsciousness, as well as the time of onset of symptoms. Neurological events were defined as obvious "strokelike" deficits. All accidents related to diving work were defined as events with onset during or less than 24 hours after the last dive (14).

Statistics

Demographic variables were statistically analyzed (using Student's T-test and Welch T-test) to show the correlation between the depth of a single dive, single dive time, surface interval, length of dive shifts, rest time between shifts, and medical history. Multiple logistic regression analysis (step-wise method) was employed to investigate risk factors determining neurological accidents. Statistical analysis was performed using commercially available statistical software (JMP 7.01J). Statistical significance was tested assuming a standard *p*-value of 0.05.

TABLE 1

Age	No.	assisted/unassisted*
~20	1	0/1
~30	8	0/8
~40	4	0/4
~50	31	4/27
~60	58	14/44
~70	42	8/34
~80	27	3/24
~90	2	0/2
Total	173	29/144

* - types of diving techniques.

RESULTS

We received answers from 185 Ama divers; 12 were excluded due to incomplete answers to the questionnaires or very shallow diving depths of ~2 meters; all of these divers were female. The other 173 male Ama divers were analyzed in this study: 29 assisted and 144 unassisted divers. Table 1 *(above)* shows the demographic characteristics of the subjects. Many of them started their profession at 20-30 years of age and continued their commercial diving for 10-40 years.

In general, Ama divers began to work as unassisted divers in shallow water, and then graduated to become assisted divers. All divers carried a weight belt to maintain neutral buoyancy (6-10 kg), and assisted Ama divers descended passively using a 10- to 20-kg weight and swam to the surface without assistance. Ama divers worked for five to six hours in the sea in two shifts a day, taking a rest for lunch. The duration of the morning shift was three to four hours, and the afternoon shift was one to three hours in duration.

Twelve of 173 Ama divers (6.9%) had experienced strokelike neurological events during or immediately after repetitive breath-hold diving; 11 were assisted and only one was unassisted (*Table 2*, below). The most common symptoms were sensory numbness (7 cases) and motor weakness on one side (6 cases). Other symptoms were dizziness, vertigo, nausea (these symptoms, however, could be due to ear barotraumas), and limb pains, which were accompanied with or without neurological disorders. Dizziness was particularly common after continuous long-term diving 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. Characteristically, many of these neurological disorders were transient and resolved

TABLE 2

Case No.	Age (yr)	Diving techniques A/U	Dive depth (msw)	Dive time (sec)	Surface interval (sec)	Length of diving shifts (hr)	Neurological symptoms of diving accidents
1	66	A	15	50	40	6 (4, 2)*	hemiparesis
2	71	A	13	60	15	5 (4, 1)	hemiparesis, dizziness, nausea, joint pain
3	52	A	12	50	50	5 (3, 2)	speech disturbance, sensory numbness, dizziness
4	53	A	10	30	30	6 (4, 2)	sensory numbness, dizziness
5	57	A	15	40	15	5 (3, 2)	hemiparesis, sensory numbness, dizziness
6	59	A	13	40	☐	6 (-, -)	sensory numbness, dizziness
7	62	A	10	60	20	4 (4, 0)	visual disturbance, dizziness
8	45	A	20	90	20	6 (3, 3)	speech disturbance, sensory numbness, vertigo, dizziness
9	53	A	15	90	30	5 (3, 2)	sensory numbness
10	71	A	15	60	25	6 (3, 3)	speech disturbance, hemiparesis, sensory numbness, limb pain
11	46	A	20	70	50	5.5 (3.5, 2)	hemiparesis
12	59	U	12	60	120	6.5 (4, 2.5)	hemiparesis, sensory numbness, dizziness, vertigo, nausea

A/U: assisted/unassisted Ama divers; **msw**: meters of sea water; ***** – parenthesis means dive shifts in morning and afternoon.

TABLE 3

	DCI (n=12)	non-DCI (n=161)	P value
Age (yr)	57.8 ± 8.6	57.7 ± 12.7	0.971
Diving history (yr)	33.7 ± 9.9	25.4 ± 13.6	0.041
Diving techniques (assisted/unassisted)	11/1	18/143	<0.0001
Diving profiles			
Dive depth (msw)	15.0 ± 3.3	9.6 ± 4.5	0.0006
Dive time (sec)	63.0 ± 16.4	43.2 ± 18.9	0.0016
Surface interval (sec)	26.0 ± 13.7	42.5 ± 54.4	0.034*
Length of diving shifts (hr)	5.5 ± 0.7	5.2 ± 1.1	0.347
Rest time between shifts (min)	36.3 ±15.5	28.6 ±17.6	0.152
Medical history			
Hypertension	9	27	0.468
Diabetes mellitus	2	14	0.359
Cardiac arrhythmia	0	7	0.461

DCI: decompression illness; **msw**: meters in sea water; values are expressed as mean ± SD; * – Welch T-test.

completely in 10 divers. At the time of the present survey, however, two divers had unresolved symptoms, one with a residual partial visual deficit and the other with sensory numbness of his hand.

The results of the analyses on associations between the study variables and neurological DCI are shown in Table 3 *(above)*. The strokelike diving events were significantly correlated with diving history, diving techniques and diving profiles. Of the three variables of diving procedures, dive depth and time were the most relevant relative to surface interval and showed the highest statistical significance. Neurological DCI was not related to a medical history of hypertension, diabetes or cardiac arrhythmia. The medical history in 173 Ama divers also included ischemic heart disease in two, cerebrovascular disease in one, and two divers had cancer; however, they were not related to the neurological disorders.

DISCUSSION

The aim of our study was to determine the risk factors of neurological disorders for Japanese Ama breath-hold divers by examining the prevalence of diving accidents among assisted and unassisted Ama divers. We found that strokelike events occurred exclusively in assisted Ama divers and that they were correlated with dive depth, dive time and surface interval. These results suggest the accumulation of inert gas with time and depth.

While DCI should be possible theoretically in breath-hold divers, the events were considered anecdotal or extremely rare until the early 1990s. Subsequently, diving accidents in commercial and amateur breath-hold divers have been reported with increasing frequency, particularly with some using submarine scooters to achieve great depths in the Mediterranean Sea near the coast of Spain (10-12).

Among 16 assisted Japanese Ama divers surveyed in a village, nine had experienced stroke-like neurological events during or immediately after repetitive breath-hold diving (9). Based on the reports of breath-hold divers with diving accidents, DCI following breath-hold diving indeed appears to exist as a clinical entity (13). Moreover, the present study is the first to show that neurological events are common among assisted Ama divers in Japan, whereas the risk of DCI has been very low in other forms of breath-hold diving.

Clinical characteristics

The most noteworthy characteristics of neurological DCI in Japanese Ama divers is limited to brain involvement (9,15), while sparing the spinal cord, which occurs frequently in compressed-air divers. The most common symptom was sensory numbness or motor weakness on one side. The findings of neurological disorders in the present study were akin to the observations in the "Taravana" diving syndrome in Polynesian pearl divers (6).

Motor weakness was the most common disturbance among their symptoms, including vertigo, nausea, unconsciousness and even sudden death. Moreover, commercial and/or amateur breath-hold divers using submarine scooters – and who achieve great depths – tended to suffer from serious neurological events after repetitive diving for several hours (10-12).

Another characteristic of DCI is that many Ama divers exhibited transient neurological deficits that lasted only several hours, even without any treatments. The clinical courses of neurological disorders in Japanese Ama divers are similar to those in breath-hold divers in other districts (6,9-12). The spontaneous resolution of neurological symptoms in many cases is typical of breath-hold divers with suspected DCI, although only some received treatments. From the Batle's series of 28 breath-hold divers with neurological DCI, four divers received normobaric oxygenation alone, and the others were treated with recompression therapy employing Comex 12, U.S. Navy Treatment Tables 5 and 6, and all recovered completely (10).

However, our current case who received recompression therapy on the second day had residual numbness in the hand over one year (16). These clinical characteristics suggest specific involvement of the brain regions.

While the diving patterns of male Ama divers with neurological DCI in this district were similar to those of female divers on an island in Japan, no field study has described DCI among Ama divers, either from medical records or by rumors (17). However, a report of psychiatric disorders among nine Ama divers on the same island showed that one female Ama was struck by transient left-sided hemiplegia and dysarthria after diving (18). A diagnosis of DCI was made, the diver was treated immediately, and no neurological sequela was reported.

DCI in Ama divers has not been well recognized by Japanese physicians and scientists. One reason is due to the transient nature of this disorder, which is seldom reported; another is that Ama divers visit medical facilities infrequently; a third is the inherent secrecy of Japanese diving communities (9).

A new finding of the present survey is the discovery of musculoskeletal involvement caused by breath-hold diving. While limb pain is the most prevalent manifestation in compressed-air divers or workers, the occurrence has not been observed previously in breath-hold divers nor has it been recognized in breath-hold divers with serious neurological events (5-12). However, one diver did experience neurological problems with joint pain after repetitive diving to 6-8 meters over three to four hours (19). It should be noted that all breath-hold divers who had serious neurological disorders without limb pain were completely assisted and dived passively (6,10-12). In contrast, Ama divers in the northern district of Yamaguchi, Japan, are partially assisted with their descents but swim to the surface without aid. Musculoskeletal involvement in breath-hold diving appears to be related to the dynamic exercise of limbs due to physical exertion during diving, which increases nitrogen (N_2) uptake and bubbling in tissues (14).

Risk factors for DCI

Susceptibility to decompression illness in breath-hold divers is influenced by a multitude of factors. This multiplicity of operating factors tends to obscure the influence of any one factor. Attempts have been made to correlate this variation in individual susceptibility with dive depth, breath-hold time, surface interval or dive duration (13,20).

Dive depth was examined as a factor in a number of DCI studies conducted with single deep breath-hold diving (20). Our results are also in accordance with those factors for neurological deficits in Ama divers (*Table 4*, next page). This study, however, has a few limitations. We could not clarify causality between risk factors because the study was a questionnaire survey and the diving profiles of Ama divers were not monitored. Another is that the incidence of neurological DCI was not high in our participants, including unassisted Ama divers. In addition, we cannot deny that most divers with neurological events tended to respond to the questionnaire.

Despite these limitations, this study provides useful information that repetitive breath-hold diving induces DCI. Repetitive diving to depths of ~4 meters for four hours did not reach levels of nitrogen partial pressure (PN_2) sufficient to cause the

TABLE 4

Variable	Parameter estimate	Standard error	P-value	Odds ratio (95% CI)
Age (yr) (increase of 10 yr)	0.045	0.054	0.398	1.57 (0.55-4.51)
Diving techniques (assisted/unassisted)	1.922	0.570	0.0007	6.84 (2.24-20.83)
Dive time (sec) (increase of 20 sec.)	0.049	0.023	0.033	2.66 (1.08-6.56)
History of diabetes mellitus (+/-)	0.308	0.531	0.562	1.36 (0.48-3.85)

Multiple logistic regression analysis was performed by step-wise method (forward selection) for 11 variables (age, diving history, diving techniques, dive depth, single dive time, length of dive shifts, surface interval, rest time, history of hypertension, diabetes mellitus and cardiac arrhythmia).

development of intravascular bubble formation and symptoms of DCI (21). The present study suggests that the effective depth of DCI in repetitive breath-hold diving for several hours is more than 15 meters of sea water (msw). Recently, one case report showed a 21-year-old man who performed 10-12 unassisted breath-hold dives to 10-18 msw over 60-90 minutes; his dive duration was one to two minutes each with surface intervals of five to six minutes (22). He presented with symptoms within two hours of the dives, which included dizziness, visual disturbance, chest tightness, dyspnea, flushed face and numbness of all limbs, involving the right side of the face. A patent foramen ovale (PFO) was subsequently discovered. Nonetheless, such a dive profile was sufficient to cause nitrogen retention in breath-hold divers and produce "bubbles" to cause symptoms of DCI, even though he had a PFO.

Another risk factor that has been revealed is the relationship between dive and surface times. A short surface time – in the order of half that of the time at depth – would increase the risk of DCI in less than two hours of repetitive breath-hold diving, particularly to depths of 20 meters or deeper (23). Olszowka and Rahn simulated the diving pattern of Japanese assisted Ama divers performing 30 dives to a depth of 20 meters for one hour and found that N_2 accumulation in the fat increased throughout repetitive breath-hold diving in contrast to N_2 quickly reaching a steady state in the brain (24). Diving to 20 meters repeatedly for five hours would require a surface time to a diving depth ratio of more

than 0.8 in order to prevent the development of DCI (25). In this study, the ratio in Ama divers with neurological disorders appears to be low in comparison with that of divers without events (0.40 ±0.23 vs. 1.29±2.25, *p*=0.008). As shown in the study of the Taravana diving syndrome by Cross, divers in Mongareva Lagoon who spent at least 10 minutes at the surface between dives did not suffer from this condition (6), whereas divers in other lagoons using short surface intervals of three to five minutes experienced Taravana. Short surface intervals with long dive times are serious risks in repetitive breath-hold diving.

Other causes or risk factors for DCI in breath-hold diving are long dive duration, PFO and/or arterial gas embolism (AGE) secondary to pulmonary barotrauma. From the kinetics of N_2 in tissues, the prolonged diving period should be reduced when dive depth increases. Thus, the depth-time product should be reduced to avoid the accumulation of inert gas, as the time for total elimination of the absorbed inert gas is unknown. In compressed-air diving, the U.S. Navy assumes that 12 hours will give a clean dive, whereas the Defence and Civil Institute of Environmental Medicine (DCIEM) requires 18 hours.

Moreover, pulmonary barotrauma remain a possible cause of DCI in repetitive breath-hold diving because a single breath-hold dive could induce AGE or mediastinal emphysema (15). Air-trapping during diving can cause local distension of the lungs and lung rupture on ascent, producing

air embolism (26). Alveolar gas volumes in *Funado* carrying weights change more rapidly than those in *Cachido*, which results in a possibility of pulmonary barotraumas in *Funado*.

Development mechanisms

The potential mechanisms of cerebral involvement following breath-hold diving are still unclear. The marked response to recompression treatment cannot be explained readily on any basis other than the presence of bubbles in the brain (23). Nevertheless, regarding N_2 kinetics and the relatively extensive cerebral perfusion, the development of autochthonous bubbles in the brain is difficult to explain. N_2 gas bubbles probably form in the intra- and extravascular spaces of systemic organs after multiple repetitive breath-hold diving. Compression of air in the lungs during diving increases N_2 pressure in the alveoli, inducing N_2 to be taken up by the blood. Because of a smaller gradient in N_2 tension from tissues to blood during ascent, N_2 is not released into the alveoli as rapidly as it is taken up by the blood and tissues during descent. N_2 accumulation in venous blood has been described in unassisted Ama divers (21); hence, multiple deep breath-hold diving with short intervals can theoretically cause DCI.

Radiological findings in Ama divers with neurological DCI show multiple cerebral infarcts in the areas corresponding to the symptoms and elicited signs. Brain lesions are localized in the basal ganglia, internal capsule, brainstem, and deep and subcortical white matter (7-9,16). The types of brain lesions in Ama divers are identical to those caused by compressed-air diving. Ischemic lesions in the basal ganglia are situated in the terminal zone, and lesions involving deep or superficial white matter corresponded to border zone or watershed regions. These are so-called "low-flow" cerebral infarctions are the result of low perfusion pressure in terminal supply areas, and these lesions are considered to be AGE of the brain.

In compressed-air diving, there is a strong correlation between high venous bubble levels and the risk of DCI (27). While a large number of venous bubbles can be found in compressed-air diving, the rate of cerebral involvement is not high because mammalian lungs usually constitute a complete filter for bubbles larger than 21 µm in diameter (28,29). However, serious neurological events are usually induced in breath-hold divers, but the detection of venous bubbles is more difficult in breath-hold divers than in compressed-air divers (30-32).

Why lesions in breath-hold diving mainly involve the brain is an unresolved dilemma. Several possible mechanisms have been identified for DCI in breath-hold divers. Lanphier reported that venous "silent" bubbles are most likely to induce diving accidents (25). After deep, repetitive diving, bubbles are formed on the venous side and travel through the heart to the lungs. Microbubbles, a component of venous bubbles passing through the pulmonary capillaries, are generally harmless to some tissues. Initially, the proposed mechanism to explain DCI in Ama divers purports that microbubbles impair the blood-brain barrier transiently, based on experimental data (33). This hypothesis, however, is inconsistent with MRI findings of the brain in Ama divers with neurological DCI. Large or small venous bubbles after repetitive deep breath-hold diving are retained or trapped in the small pulmonary arteries. When Ama divers descend, the trapped bubbles are compressed and are liable to pass through the pulmonary capillaries (34). Arterialized bubbles expand during each ascent and gather in the terminal supply areas of the brain, border zones and watershed regions. While this is our hypothesis of neurological DCI in Ama divers, the exact mechanisms of this condition are in dispute.

CONCLUSIONS

Neurological diving accidents involving the brain are common in assisted male Japanese Ama divers. Repetitive deep breath-hold diving with a prolonged total dive time and short surface intervals appears to cause neurological DCI, which characteristically manifests as cerebral stroke. The most plausible mechanism of brain involvement is N_2 bubbles retained or trapped in the pulmonary arteries and which are liable to pass through the lungs during breath-hold diving and expand during each ascent.

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