ORIGINAL PAPERS

BREATH-HOLD DIVING CAN CAUSE DECOMPRESSION ILLNESS

Robert Wong

Key Words

Breathhold diving, decompression illness, history, risk.

Summary

The widely held belief that breath-hold divers do not develop decompression illness is incorrect. There is historical and modern (medical) evidence to the contrary.

With the introduction of submarine scooters BH divers can now do deeper repetitive dives that have led to DCI.

While DCI is uncommon with BH diving, cases occur. Breath-hold divers should consult their medical practitioners if they experience symptoms after diving.

With the exception of the aggressive mode of diving, associated with Taravana, of some of the pearl divers from the Tuamotu Archipelago in French Polynesia, most professional BH divers do not experience decompression illness (DCI). This is almost certainly due to the fact that they generally limit their dive times and use longer surface intervals.

Introduction

For centuries, humans have performed breath-hold (BH) diving as a profession. However, since the introduction of compressed air diving, the number of professional BH divers has declined. In northern Australian waters, in the middle of the nineteenth century, mother-of-pearl shell harvesting, for the production of buttons and furniture inlays, was initially done by BH divers. With the advent of compressed air diving in the later 1800s BH divers were replaced by hard hat divers, usually Japanese.[1] At about the same time, in the Aegean Sea, compressed air diving also displaced the traditional BH diving for sponges. In both areas the increased underwater achievements were paid for by horrific incidences of decompression sickness (DCS) when the men worked in deeper water.[2]

In the Tuamotu Archipelago, in French Polynesia, BH diving for pearls was still being carried out in 1957 with a number of divers becoming paralysed or dying after BH dives.[3] More recently the introduction of cultured pearls and the availability of scuba gear has eliminated the need for BH diving in this area. But again at the cost of DCS.

Nevertheless, BH diving for a living still exists in a few places, most notably Japan and Korea. Professional BH divers in Korea are predominantly women (known as hae-nyo for sea women), whereas, in Japan, men outnumber the women (ama for sea women and katsugi for sea men). For convenience they have all been referred to as Amas in most publications.

It is a general misconception amongst most recreational divers, that decompression illness (DCI) does not occur with breath-hold diving, unlike compressed air diving where most divers are vaguely aware of the risk of DCI.

Taravana

In 1958, in the Tuamotu Archipelago, pearl divers used a weight (4-6 kg) to assist their descents to depths of from 9 to over 40 m. The dive time was 30-60 seconds and surface intervals were 3-10 minutes. The working day was some 6 hours. In the Hikueru Lagoon, in one day of diving, 47 (20%) of the approximately 235 divers developed symptoms of “Taravana”. Symptoms described included vertigo, nausea and mental anguish. Some divers became paralysed, either partially or completely. Two fatalities occurred, one underwater after about 18 to 20 dives to 39 m (130 ft). This death may have been due to hypoxia during his ascent.[4] However the other victim was pulled into the boat semi-conscious and died two hours later, which does not fit in the clinical picture of ascent hypoxia. Mangareva pearl divers using the same diving technique but with 12-15 minutes on the surface did not develop Taravana.[3]

Compressed air divers reporting similar symptoms after a dive in Australia would be considered as, at least, equivocal cases of DCI.

The Ama

For centuries, the Japanese and Korean breath-hold divers, generally known as Amas, have been diving without apparent incidence of DCI. This may be because both the depths and the number of dives have been inadequate to produce sufficiently high nitrogen partial pressures to cause DCS. An alternative explanation is that such diving accidents are regarded as part of the job and are not reported due to a general lack of awareness of the problems.

Teruoka published the first scientific study of BH diving in 1932.[5] He described the diving patterns of assisted (Funado) and unassisted (Cachido) Ama. Using a

counterweight, which was hauled up by the boatman when dropped, one Funado descended to a depth of 20-25 m at an average speed of 1.2-1.5 m/sec, but occasionally as fast as 1.8 m/sec. After about 28 seconds on the bottom, the counterweight was dropped and Funado ascended at 1.6 m/sec, being pulled up to the boat. The total time underwater was less than 1 minute. Also described were two Cachido who descended under their own power to 5 -11 m. Both ascent and descent were at about 0.6 m/sec. Travel time took an average of 17.4 seconds for the 5 m dive and 38 seconds for the 11 m dive. Total dive time was 30 seconds for 5 m dive and 45 sec for 11 m dive. Observations on a large group of Ama indicated that the typical dive was for a total of 30 seconds to 5 m and was repeated 60 times in an hour. Exceptional Cachido Ama were able to perform sustained repetitive dives for around 60 seconds each. The Funado Amas managed to perform repetitive dives for 60 second duration to 15-20 m with 60 seconds between dives. Before wetsuits were introduced Amas dived either in minimal briefs or in cotton shirts and trousers.[6]

In 1965 Teruoka's study became available in English when it was reproduced in *Physiology of Breath-hold Diving and the Ama of Japan* by Rahn and Yokoyama.[6] The diving pattern of the Amas was governed by the need for the divers to get out of the water and rewarm at intervals. By the 1980s, when wearing wetsuits had become common among the Ama, more time was being spent underwater as rewarming was not needed so often.[7]

Since the introduction of wet suits and fins, the pattern of diving has remained similar to the that of the cotton suit era. The ascent, however, is about 60% faster with the use of fins. The bottom time is 16.5 seconds for the 5 m dive and 12 sec for the 10 m dive. The use of wet suits has lengthened the working time substantially. By 1985 Amas were diving for 3 hours in summer as opposed to an hour in a cotton suit. In winter they dived for 2 hours a day instead of 30 minutes.[7]

In 1955, well before the introduction of doppler ultrasound, Schaefer observed foam in venous and arterial blood drawn immediately after breath-hold divers surfaced from single dives, lasting approximately 1.5 minutes, to 27 m (90 ft).[8] Subsequent samples 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 breath-hold dives.

Lanphier, using theoretical calculations, suggested that enough N_2 could be taken up by the body to cause DCS if deep repetitive dives were separated by short surface intervals.[9]

Doppler studies have demonstrated the presence of venous gas emboli after repeated breath-hold dives in Ama divers.[10,11] However, no cases of DCS had been reported until recently.

In 1992 nitrogen accumulation in venous blood was demonstrated in repetitive breath-hold diving in Korean female divers, however the level of N_2 accumulation was thought insufficient to cause DCS.[12]

In 1998 two cases of multiple cerebral infarctions in Ama divers, who developed neurological symptoms after more than 3 hours of repeated BH diving to 15-25 m, were reported.[13] These divers used a 15 kg weight to assist their descents but ascended unassisted. The dives lasted about 1 minute with surface intervals of 1-3 minutes. Symptoms included euphoria, disturbed conscious state, dizziness, diplopia and nausea.

The incident dive of the first diver, which led to the investigation, was performed in 1990 when, after a series of dives in the morning, the diver complained of diplopia and nausea. About 10 minutes later, he was unable to speak and had weakness and numbness over the right side of the body. His speech recovered after some 20 minutes, but the hemiparesis and paraesthesia persisted for about 1 and 4 weeks respectively. Two weeks after the incident, he was transferred to a hospital for investigation. The delay was because the diver lived a long distance away from any hospitals. CT scan showed no abnormalities but MRI demonstrated 2 small lesions, one in the left internal capsule and the other in the left frontal lobe.

The second diver performed similar dives and had done so for 20 years. His incident dive occurred in 1986. After diving for 4 hours continuously, he took a break for lunch, but within 10 minutes, he developed hemiparesis and sensory disturbances over his left side. Within 2 hours he was unable to walk and within 3 hours he lost consciousness but recovered gradually over several hours. Four years later, he reported to hospital for examination. Physical examination revealed no abnormalities, but MRI showed lesions in the subcortical and subependymal areas.

While the only evidence of their previous problems are the MRI lesions, the clinical histories of rapid onset neurological problems soon after a series of deep immersions and the slow but complete recovery suggest that DCI was the likely diagnosis. There are numerous such case histories to be found in Paul Bert's *Barometric Pressure*.[14] Although these 2 divers developed DCI in 1986 and 1990, their problems only recently came to the attention of knowledgable medical practitioners.

Perhaps, DCI is far more common in BH diving than many doctors believe, but has not been reported because it is accepted as part of the job. In 1974 a study of 301 out of 400 Japanese compressed air, shell-fish divers, living in an isolated village, was published.[15] Between 1966 and 1968 "each year three to five men died from accidents or from decompression sickness. 'The bends' were very common; they almost ignored them, thinking them unavoidable and treating them with baths and by drinking alcohol". If these

compressed air divers phlegmatically accepted paralysis as a frequent, unavoidable occurrence in their lives, as they did, it seems likely that BH divers might accept the occasional occurrence of symptoms after a day's diving in the same way.

This supposition is supported by Kohshi et al.'s interviews with the breath-hold divers from the same village. Eight of 15 divers had previously experienced some neurological disorders during or after a dive.[13] The divers did not consider their symptoms as abnormal and had not sought medical treatment, which was, in any case, not available in their remote villages.

A year later Kohshi et al. reported two more BH divers who experienced neurological symptoms.[16] Again both dived with a 15 kg weight but with unassisted ascents. The dives were to 15-25 m for 1-1.5 minutes with 1 min surface intervals. They usually dived for 5 to 6 hours. One diver, who experienced dizziness and blurred vision, had a MRI performed 4 days later which showed 2 cerebral infarcts. The 2nd patient, 39 years old, was admitted to hospital, after similar dives, with a mild hemiparesis and numbness on the right side. He had also experienced left hemiparesis after breath-hold diving at the ages of 17, 25 and 27.[16] Three cerebral infarcts were demonstrated on MRI. The fact that this diver had experienced and recovered from 3 incidents of hemiparesis since the age of 17 suggests that he had accepted this paralysis as a "normal" consequence of diving and so had not sought medical treatment.

Again in Japan, Kohshi et al. interviewed 15 divers on the island of Mishima, Yamaguchi Prefecture.[17] Seven admitted to having experienced previous diving accidents, with unilateral weakness and sensory disturbances. Eleven divers experienced dizziness, nausea and/or euphoria. None experienced the joint pains commonly seen after compressed air dives. One diver had lost consciousness, which is most likely to have been the result of hypoxia of ascent. Most of the divers spent 4 -5.5 hours in the water. Depths were to between 10-15 m, with some to 20-30 m. They averaged 20 to 30 dives per hour, but did up to 40 dives per hour in shallower water (8 to 12 m). The onset of symptoms was always immediately on reaching the surface.

The above reports are reminiscent of the symptoms reported by Cross.[3] Compressed air divers reporting similar symptoms after a dive in Australia would be considered as, at least, equivocal cases of DCI.

Recreational divers

There are many breath-hold recreational divers who spear-fish and do repetitive dives. Medical attention has been focussed on those who hyperventilate before the dive and die from hypoxic unconsciousness, followed by drowning, on their way to the surface.[4] Other recreational divers attempt BH dive records, although they do not do repetitive dives. Nonetheless, great depths have been achieved. In October 1999 Umberto Pelizziri set a world record for sled-assisted free diving by reaching 150 m.[18] These deep dives last around 3 to 3.5 minutes. These durations expose the divers to the hazards of hypoxia on ascent.

Two BH divers who experienced nausea, vertigo and headache, neither of whom complained of joint pain, were diagnosed as DCI by Wong.[19] One was recompressed with complete resolution of symptoms. The other recovered over time without treatment.

There is a new sport in the Mediterranean Sea off the Balearic Islands. Here there are many BH divers who regularly make use of submarine scooters to achieve quick descents and ascents. Some have dived to 63 m (210 ft) and back in a matter of 2 or so minutes, with surface intervals of 2 minutes or less. They do between 15 to 20 dives per hour for between 3 to 8 hours. Batle reported 25 cases of neurological DCI in such divers whose symptoms appeared immediately on surfacing.[20] They were all treated using either USN Table 5 or 6 with complete resolution of symptoms. The depths of such dives using submarine scooters and the rate of ascent would suggest that supersaturation would occur and produce bubbles on ascent.

Magno et al. described 6 separate incidents of neurological problems which occurred in four BH divers.[21] The dives included a single deep weight and buoyancy assisted dive to 120 m (400 ft), 3 assisted dives to depths ranging from 35-90 m (115-300 ft) and multiple unassisted dives to 25-30 m (89-100 ft) over a period of 2-4 hours. Symptoms and signs appeared within minutes of surfacing and included hemiplegia, ataxia, dysarthria, diplopia and colour blindness. Some were recompressed whereas others received no therapy, nonetheless, all made complete recovery.

Fanton et al. described a spear fisherman who did a series of dives to 40 m consisting of some 14 dives per hour for 3 hours.[22] He was unconscious on surfacing. With resuscitation and oxygen administration he woke up but exhibited slow mentation and disorientation. This is unusual with anoxic unconsciousness of ascent in the absence of near drowning. Subsequent EEG and MRI showed focal cerebral injury. The diver admitted that he had experienced several episodes of weakness in the arms during earlier diving activities. These cannot be explained by ascent hypoxia.

While the above cases affected divers who performed deep repetitive dives, Bayne and Wurzbacher described a BH diver who twice attempted to swim across a 25 m (25 yard) pool at about 1.8 m (6 ft) deep.[23] After the second

attempt, he surfaced complaining of headache, dizziness and tingling all over. He also complained that "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 (the left heart was not opened at autopsy). Bruch has also reported 2 divers who developed mediastinal emphysema during BH diving to 4.5 m (15 ft).[24] Such cases are seldom mentioned in diving medicine textbooks.

Personal communications, as yet unpublished, include a diver who experienced headache, dizziness, blurred vision, numbness and weakness of all four limbs after repetitive BH dives over a three and half hour period to a depth of only 8 m and a BH diver who did repeated dives for some 4 hours to depths of 6-8 m and complained of tiredness, headache, joint pain and paraesthesia in his upper limbs. This diver was treated in a recompression chamber with resolution of all symptoms.

Breath-hold diving after compressed air diving

Paulev, a submarine medical officer, described his personal experience.[25] After spending 8 minutes at 20 m as an attendant in a recompression chamber, he performed a number of repetitive BH dives in the submarine escape training tank to depths of 20 m for about 5 hours. He suffered from nausea, dizziness and belching. Later, he also developed pain in his left hip, the right knee, the right arm was weak and the right arm felt tired. There was also paraesthesia and blurring of vision. He was recompressed and made a full recovery. Three similar cases of BH diving following previous hyperbaric exposure also presented with symptoms of DCI and were similarly treated with complete resolution.

Discussion

It is a general misconception, and a myth, that DCI does not occur with BH diving. It is probable that symptoms are often ignored or misinterpreted, both by the divers and by any doctors who were consulted, because of the general lack of appreciation of the pathophysiology of this condition.

However, the cases mentioned above show that, under certain circumstances, DCI is possible after BH diving. Hemiplegia has occurred after a single breath-hold dive.[26]

Given the right circumstances (repetitive dives for prolonged periods and short surface intervals, perhaps with excessive ascent rates), it is possible for the body to absorb sufficient inert gas to cause supersaturation and bubble formation on ascent. Short surface intervals also do not allow time for the body to eliminate much inert gas between dives.

In the cases above the following factors were present

1 repetitive dives in excess of some 3 to 4 hours;
2 depths of dives were generally in excess of 15-25 m;
3 surface intervals were generally short and
4 ascent rates were usually rapid.

In the cases of CAGE, one would have to assume that during the dive, air was trapped in a closed off part of the lungs before ascent. It is hypothesised that air trapping during diving can cause local distension of the lung and lung rupture on ascent producing air embolism.[27]

It has also been suggested that forceful inhalation to total lung capacity (TLC) might cause lung rupture by over-distension of a weak area in the lung without pressure changes.[28]

Conclusions

BH diving is not as benign as one would like to think.

Decompression illness, both DCS and CAGE, can occur with BH diving. The incidence is considered to be low. In most cases DCS is due to a combination of repetitive deep diving, short surface intervals and fast ascents. Since it is reputed that 30% of the population might have a Patent Foramen Ovale, it could be considered surprising that few BH divers present with symptoms after repetitive dives. The low rate of symptoms is probably because few breath-hold divers seek medical attention for symptoms which commonly disappear spontaneously.

Deep repetitive BH diving immediately after compressed air diving can cause DCI.[24] Breath-hold diving, to recover an anchor, after completing an appropriate decompression for a compressed air dive has led to sudden death on more than one occasion.[28]

It is highly recommended that any diver who experiences unusual symptoms after BH diving should seek medical advice, as recompression can speed their recovery if the symptoms are due to DCI.

References

1 Edmonds C. Pearl diving: the Australian story. *SPUMS J* 1996; 26 (Suppl): 4-15
2 Blick G. Notes on diver's paralysis. *Brit Med J* 1909; ii (25 December): 1796-1798
3 Cross ER. Taravana: diving syndrome in the Tuamoto diver. In *Physiology of Breath-hold Diving and the Ama of Japan. Publication 1341*. Washington, DC: National Academy of Sciences, 1965; 207-219.

4 Craig AB Jr. Underwater swimming and loss of consciousness. *JAMA* 1961; 176 (4): 256-258
5 Teruoka G. Die Ama und ihre Arbeit. *Arbeitsphysiol* 1932; 5: 239-251
6 Rahn H and Yokoyama T. Eds. *Physiology of Breath-hold Diving and the Ama of Japan. Publication 1341.* Washington, DC: National Academy of Sciences, 1965
7 Lin Y-C. Breath-hold diving: Human imitation of aquatic mammals. In *Diving in Animals and Man. The Kongsvoll Symposium*. Brubakk AO, Kanwisher JW and Sundes G. Eds. Trondheim: Tapir Publishers, 1985; 81-111
8 Schaefer KE. The role of carbon dioxide in the physiology of human diving. In *Underwater Physiology Symposium. Publication No 377*. Grolf LG. Ed. Washington, DC: National Academy of Sciences, National Research Council, 1955; 131-141
9 Lanphier EH. Application of decompression tables to repeated breath-hold dives. In *Physiology of Breath-hold Diving and the Ama of Japan. Publication 1341*. Rahn H and Yokoyama T. Eds. Washington DC: National Academy of Sciences/ National Research Council, 1965: 227-236
10 Spencer MP and Okino H. Venous gas emboli following repeated breath-hold dives. *Fed Proc* 1972; 31: 355
11 Nashimoto I. Intravenous bubbles following repeated breath-hold dives. *Jpn J Hyg* 1976; 31: 439
12 Radermacher P, Falk KJ, Park YS, Ahn DW, Hong SK, Qvist J and Zapol WM. Nitrogen tensions in brachial vein blood in Korean Ama divers. *J Appl Physiol* 1992; 73: 2592-2595
13 Kohshi K, Kinoshita Y, Abe H and Okudera T. Multiple cerebral infarction in Japanese breath-hold divers: two case reports. *Mt Sinai J Med* 1998; 65 (4): 280-283
14 Bert P. *Barometric Pressure*. 1878. Translated by Hitchcock MA and Hitchcock FA. Columbus Ohio: College Book Company, 1943. Reprinted by the Undersea Medical Society, 1978
15 Ohta Y and Matsunga H. Bone lesions in divers. *J Bone Joint Surg* 1974; 56 B (1): 3-16
16 Kohshi K, Okudera T, Katoh H and Abe H. Diving accidents during repetitive breath-hold dives: two case reports. In *13th International Congress on Hyperbaric Medicine Program*. Kobe, Japan, 1999; 116
17 Kohshi K, Okudera T, Katoh T, Kinoshita Y and Abe H. Do breath-hold dives cause dysbaric diving accidents? In *13th International Congress on Hyperbaric Medicine Program*. Kobe, Japan, 1999; 117
18 Ecott T. No limits: Pelizzari smashes record. *Diver* 1999; 44 (12): 13
19 Wong RM. Taravana revisited: decompression illness after breath-hold diving. *SPUMS J* 1999; 29 (3): 126-131
20 Batle JM. Decompression sickness caused by breath-hold diving hunting. *13th International Congress on Hyperbaric Medicine Program*. Kobe, Japan, 1999; 87
21 Magno L, Lundgren CEG and Ferringo M. Neurological problems after breath-hold diving. *Undersea Hyper Med* 1999; 26 (Suppl): 28-29
22 Fanton Y, Grandjean B and Sobrepere G. Accident de decompression en apnee. *Presse Med* 1994; 23: 1094
23 Bayne CG and Wurzbacher T. Can pulmonary barotrauma cause cerebral air embolism in a non-diver? *Chest* 1982; 81: 648-650
24 Bruch FR. Pulmonary barotrauma. *Ann Emerg Med* 1986; 15: 1373-1375
25 Paulev P. Decompression sickness following repeated breath-hold dives. *J Appl Physiol* 1965; 20 (5): 1028-1031
26 Ferringo M and Ludgren CEG. Human breath-hold diving. *The Lung at Depth* Lundgren CEG and Miller JN. Eds. New York: Marcel Dekker Inc., 1999; 529-585
27 Dahlback GO and Lundgren CEG. Pulmonary air-trapping induced by water immersion. *Aerospace Med* 1972; 434:768-774.
28 Francis TJR and Denison D. Pulmonary barotrauma. In *The Lung at Depth* Lundgren CEG and Miller JN. Eds. New York: Marcel Dekker Inc., 1999; 295-374
29 Elliott D and Kindwall E. Decompression Sickness. In *Hyperbaric Medicine Practice*. Kindwall ED and Whelan HT. Eds. Flagstaff, Arizona: Best Publishing Company, 1999; 436-437

Dr Robert M Wong, FANZCA, DDHM, is Director of the Diving and Hyperbaric Medicine Unit at Fremantle Hospital, Fremantle Hospital, PO Box 480, Fremantle, Western Australia 6160. Phone +61-(0)8-9431-2233. Fax +61-08-9431-2234.
E-mail <Robert.Wong@health.wa.gov.au>.