Review articles

The risk of decompression illness in breath-hold divers: a systematic review

S Lesley Blogg[1], Frauke Tillmans[2], Peter Lindholm[3]

[1] *SLB Consulting, Newbiggin-on-Lune, Cumbria, UK*
[2] *Divers Alert Network, Durham, USA*
[3] *Department of Emergency Medicine, University of California, San Diego, CA, USA*

Corresponding author: *Professor Peter Lindholm, 200 W Arbor Drive, MC8676, San Diego, CA 92103, USA*
plindholm@health.ucsd.edu

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Abstract
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Introduction: Breath-hold (BH) diving has known risks, for example drowning, pulmonary oedema of immersion and barotrauma. There is also the risk of decompression illness (DCI) from decompression sickness (DCS) and/or arterial gas embolism (AGE). The first report on DCS in repetitive freediving was published in 1958 and from then there have been multiple case reports and a few studies but no prior systematic review or meta-analysis.
Methods: We undertook a systematic literature review to identify articles available from PubMed and Google Scholar concerning breath-hold diving and DCI up to August 2021.
Results: The present study identified 17 articles (14 case reports, three experimental studies) covering 44 incidences of DCI following BH diving.
Conclusions: This review found that the literature supports both DCS and AGE as potential mechanisms for DCI in BH divers; both should be considered a risk for this cohort of divers, just as for those breathing compressed gas while underwater.

Introduction

Breath-hold (BH) diving is distinguished by the combination of immersion of the diver's face and voluntary breath-holding or 'apnoea'. It is practiced for several reasons, including the collection of food or resources (e.g., shellfish, pearls), spearfishing, recreational diving, and competitive apnoea diving. Most of these BH dives are made in relatively shallow water, although spearfishing divers and competition divers do make deeper excursions, with the world record standing at 214 metres for a 'no limits' freediving competition.[1] The Guinness world record for static apnea (breath holding only) stands at 11:54 minutes.[2] By comparison, traditional food gatherers such as the Japanese Ama tend to make many dives in a day but to a maximal depth of only ~20 msw and for around one minute at a time.[3]

The risks of BH diving of all types include barotrauma, drowning, hypothermia, marine animal injury, hypoxia/shallow water blackout, and decompression illness (DCI) from arterial gas embolism (AGE) and/or decompression sickness (DCS). Given the short BH periods and depths involved for traditional BH diving, it was long held that DCS was unlikely to occur in these divers although this attitude has now changed, particularly as longer, deeper repetitive dives are now made more commonly.[4]

Decompression sickness arises when inert gas (for example nitrogen) is accumulated at depth and becomes supersaturated in the tissues, then forming bubbles upon ascent from the dive. Historically, it was believed that BH dives such as those made by food gatherers, were not deep or of long enough duration for inert gas tissue pressures to become raised to levels capable of causing DCS.[4] However, as early as 1958, Taravana syndrome, which is a collection of neurological symptoms such as vertigo, nausea, paralysis and unconsciousness, was noted in pearl dives who made up to 60 dives a day in French Polynesia.[5] At this time, there was some acceptance that BH dives to extreme depths, with greater frequency and duration than commonly used while food gathering could prove problematic; this acceptance is particularly pertinent for certain classes of competitive BH diving with the aim of diving as deep or for as long as possible.[4,6,7] Although Lanphier was one of those sceptical of BH diving causing DCS, in 1965 he made calculations on probable nitrogen tissue tensions for a series of BH dives using the United States Navy (USN) decompression tables. These calculations determined that the ratio between surface

interval (S) (to wash out nitrogen) and time at depth (D) regulates nitrogen accumulation.[4,6] When the ratio of these parameters (S/D) is 1, he calculated that no limits need be imposed and the dive was safe, but at a ratio of 0.5 then there was a risk of DCS in < 3 h; values below 1 implied an increasing level of risk with decreasing value. Other workers have also calculated the theoretical risk of breath-hold DCS, with one study concluding that symptomatic nitrogen supersaturation can be reached during BH diving, again with surface interval playing an integral role.[8]

Fitz-Clarke developed a model for BH DCS using documented human BH dives to 100 m or greater, which determined that predicted DCS risk is negligible in a single dive up to around 100 msw.[9] All of these findings indicate that there is more risk to a diver making multiple BH dives, but DCS has also been seen in single, deep dives at depths usually over 100 m.[9] Decompression sickness has been identified in most groups of BH divers, and it is now generally accepted that BH diving can cause neurological DCS following deep and repetitive dives, with most cases affecting the brain.[10] Certainly, competitive apnoea divers and organisations such as International Association for Development of Apnea [AIDA] or the World Underwater Federation [CMAS] are aware of the risk of DCS in their sport and offer oxygen breathing at depth (~5 m) post BH diving in competitions, in an effort to mitigate risk.

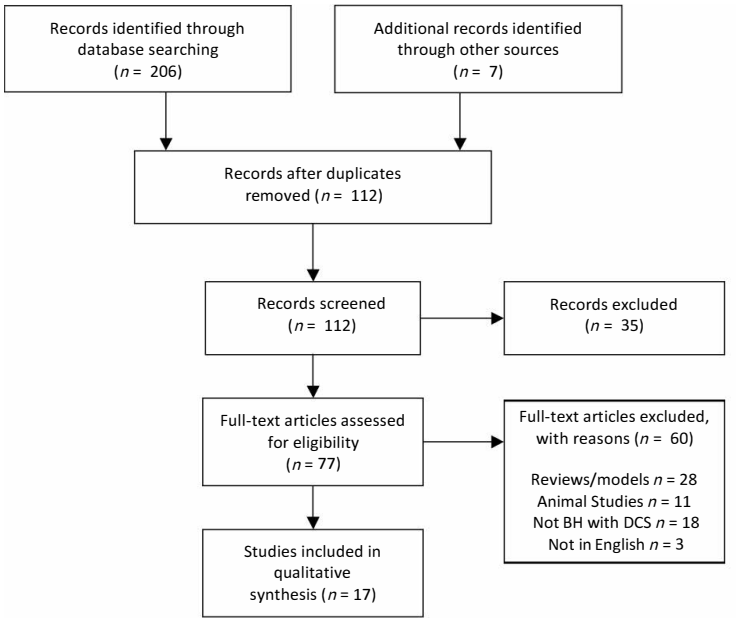
The Divers Alert Network (DAN) collects data on diving incidents across the world.[11–14] In their most recent report (2020, in press), several BH diving fatalities were reported but none of these involved DCS. However, a BH diver in the Caribbean was treated recently with hyperbaric oxygen after suffering post-dive neurological symptoms. Initially the diver was denied treatment, as attending medics did not acknowledge DCS in freedivers but after contact with hyperbaric physicians via DAN, treatment was administered (personal communication with Dr James Chimiak, DAN USA). This shows that awareness of the possibility of DCS/DCI occurring following a BH dive still needs to be improved.

Reviews on this subject have been written in the past, but to date there are no systematic reviews or meta-analysis of BH DCI cases and studies in the literature. To enable future studies elucidating the risk of AGE vs DCS in breath-hold diving and potential guidelines for safer diving we aimed to consolidate the relevant literature with this review.

Methods

A preliminary literature search was carried out (search date 23 March 2021) in PubMed to identify all terms and keywords. A systematic literature search following PRISMA Guidelines was then performed (23 August 2021). Searches were carried out on PubMed and Google Scholar. Search terms included "*breath-hold*", "*breath-hold AND diving*", "*breath-hold AND DCS*", "*apnea/apnoea AND DCS*", "*breath-hold AND diving AND DCI*", "*Taravana*", "*Taravana AND DCS*", "*breath-holding AND DCS*", "*breath-hold AND diving AND DCI*", which yielded 206 results (Figure 1). Seven additional records (excluding duplicates) were found via Google Scholar or were known personally (conference abstracts, references from references). The final selection had all duplicates removed and was limited to studies or abstracts in English. All reviews and animal studies were excluded. In total, 17 studies were found to be of relevance (Figure 1), that is they included reports of cases of breath-

Figure 1
Flow chart of the study selection process; BH – breath-hold; DCS – decompression sickness



hold diving with DCS, or experimental work investigating breath-holding and DCS.

DATA EXTRACTION

Data extracted (if present) from case reports or experimental studies identified in the search that either reported DCI in BH divers or carried out experimental trials to investigate the likelihood of DCS in BH dives included: the number of cases, the number of individuals affected, bubble grades recorded, sex and age of the diver, location, symptoms, diagnostic tools used, diagnosis (type of DCI - DCS, AGE, symptoms), treatment, dive time (period over which dives were carried out) number of dives made, maximum depth dived, maximum individual breath-hold period, and type of BH dive – if competitive BH then which category were the divers taking part in.

Results

OVERVIEW OF STUDIES

Of the 17 articles included in the final selection, 14 were case studies and three were experimental studies; two of the case studies were performed by the same group[15,16] and used the same data for analysis and thus, were treated in our analysis as a single study. The articles were published between 1965–2020, of which 15 (88%) were published in or after 2000. The data extracted from these 17 articles noted 44 cases of DCI following BH diving, 43 of these were observed in men (two studies[15,16] do not specify sex), and one case in a woman. Of the clinical case studies, 13 of the 15 detailed that they used imaging (magnetic resonance imaging [MRI] or computed tomography [CT] scans) to investigate the illness. Table 1 shows the characteristics of the studies, including subject information, dive profile and type and diagnosis made, while Table 2 lists the symptoms noted in the studies.

CASE REPORTS (SEE TABLES 1 AND 2)

Accurso et al. 2018[17] – maximum depth 40 metres of sea water (msw)

This paper describes two cases of taravana syndrome (defined in the manuscript as neurological DCS) in the same male diver, age 38 years. The diver had no existing illnesses, did not smoke, and had been spearfishing for 10 years. Further details of his dives are shown in Table 1. In both cases, CT imaging found hypodense areas, in the right internal capsule following dive 1, and in the nucleus of the right thalamus associated with oedema following dive 2. Amongst other laboratory tests, thrombophilic screening was carried out; a high level of homocysteine (28.59 $\mu mol.L^{-1}$) was found. The diver was treated with three recompression sessions on US Navy Table 6 and acetylsalicylate acid 100 mg daily was prescribed as a prophylactic treatment. He was discharged after ten days and dived after four months with no further incidents reported. The authors concluded that their findings suggested that a thrombophilic state due to hyperhomocysteinaemia might be associated with taravana syndrome in BH divers and should be investigated further.

Alaimo et al. 2010[18] – maximum depth 24 msw

This study reports the case of a 41-year-old diver who also suffered from taravana syndrome (Table 1), presenting with various neurological symptoms including (Table 2): vertigo, ringing in the left ear, confusion, paraesthesia of the right arm, and right hemiparesis. A CT scan showed four cerebral bubbles, three in the left hemisphere and one in the right at the level of the internal carotid and ophthalmic arteries. The diver was rehydrated, administered parnaparin and hyperbaric oxygen treatment (HBOT); thereafter the symptoms resolved, and a new CT scan confirmed the disappearance of the bubbles. The authors claimed that this was the first report of the documented presence of bubbles in a patient with taravana syndrome, and this supported the hypothesis that nitrogen bubbles were responsible for symptoms in these cases.

Batle 2000 a and b[15,16] – maximum depth unknown

These abstracts outline a study on 28 patients who suffered from BH diving accidents and were treated at the MEDISUB Hyperbaric Research Institute in Mallorca, Spain, between 1995–1999. The authors reported an increase in the number of incidents over that period. The divers all had neurological symptoms immediately on surfacing and once at the unit, all were given a physical examination and full clinical history taken. All were given a CT scan of the brain, the treated with USN Tables 5 and 6 until symptoms were resolved with another CT scan given several days later to confirm resolution of changes. The authors suggested that following BH diving, any resulting gas emboli would be apparent at the level of the brain. The author also went on to examine the surface intervals made by the divers in an attempt to calculate how long an interval should last in a BH diver in order to avoid the evolution of arterial bubbles but no data or results on minimum surface interval thresholds were provided.

Cortegiani et al. 2013[19] – maximum depth 30–35 msw

This report focused on a male BH diver who was 57-years-old and an experienced underwater spearfishing champion. He presented with neurological disorders including dizziness, sensory numbness, blurred vision, and left frontoparietal pain, after several dives to 30–35 msw with short surface intervals (Table 1). His symptoms spontaneously resolved but the following morning the symptoms reoccurred, so the diver returned to the hospital where he suffered a tonic-clonic seizure and lost consciousness. Magnetic resonance imaging revealed a cortical T1-weighted hypointense area in the temporal region corresponding to infarction with partial haemorrhage. He was then treated by HBOT, and the symptoms resolved swiftly. The authors concluded that

Table 1

Details of studies included in the qualitative synthesis; [*]accumulated time spent underwater across all dives made; [a]same patients as previous Batle reference; [b]mean value; [c]eight repetitive dives; [d]long BH sessions to recreate spear fishing sessions; [e]dives made over 3 days; [f]median value; [g]ratio surface to underwater time. # refers to diver number, where data for more than one diver is reported; CAGE – cerebral arterial embolism; DCI – decompression illness; DCS – decompression sickness; F – female; M – male; NS – not specified; Ref – reference; Rep – repetitive

Ref	*n*	Cases	Sex	Age (y)	Location	Clinical or exp.	Diagnosis	Total dive time (h:min)*	Number of dives	Max depth (m)	Max BH time (min)	Surface interval	Activity
17	2	2	M	38	Italy	Clinical	Both taravana (neurological DCS)	#1: 7 #2: 4	#1: 100–120 #2: 60	40	1.4	–	Spearfishing
18	1	1	M	41	Italy	Clinical	Taravana (neurological DCS)	5	60–80	24	–	–	–
29	11	0	M	–	Croatia	Exp.	–	Rep[c] 1, Deep[d] 6	Rep 8, Deep – unlimited	35 & 40	–	Rep – 3 min, Deep – not restricted	Spearfishing (simulated)
15	28	28	–	–	Spain	Clinical	Neurological DCS	–	–	–	–	–	–
16	28	28[a]	–	–	Spain	Clinical	Neurological DCS	3–8	~15·h^{-1}	63	2	–	Spearfishing, often with scooters
30	10	0	M	29[b]	Spain	Exp.	–	4:03[b] (2–6)	–	35[b] (24–40)	–	–	Deep harpoon fishing event
32	1	0	M	53	Italy	Exp.	–	–	–	40.2[f] (6.2–41.7)	140 s (± 42)	9.8 min (± 9) 3:4	Spearfishing
19	1	1	M	57	Italy	Clinical	Taravana (neurological DCS)	2:30	19	30–35	2:50	1–1:30 min	Spearfishing
20	1	1	M	21	France	Clinical	Neurological DCS	1–1:30	10–12	18	2	5–6 min	BH prep for ships diving course
21	1	1	M	39	Portugal	Clinical	Neurological DCS? Left frontal white matter lesion	5	–	30	2	“Short”	Sports?
22	2	2	M	33, 39	Japan	Clinical	Neurological DCS	~5–6	–	#1: 22, #2: 23	#1: ~1–1:30, #2:	Both ~ 1 min	Ama – food gathering, weight assisted

Table 1 continued

23	1	1	M	65	Japan	Clinical	CAGE (lung overexpansion) leading to cerebral DCI	~3	–	20	–	–	Ama – food gathering, weight assisted
24	2	2	M	-	Japan	Clinical	Cerebral DCS	#1: rep dives #2: 6	–	#1: 20, #2: 30	–	–	Free divers
25	1	1	M	-	Norway	Clinical	Limb/chest/abdominal pain and neurological DCS (paraesthesia)	5	60	20	2	Few seconds to 1–2 min	Submarine escape training – 'bottom drop'
26	1	1	M	34	Japan	Clinical	Neurological DCS	–	–	25	1:20	20–30 s	Ama – food gathering, weight assisted
27	1	1	M	31	Germany	Clinical	CAGE/stroke	–	3	100	4	15 min	Competitive free diver
28	1	0	F	74	Japan	Clinical	DCS (endogenous cerebral ischaemia)	3	–	5	–	–	Ama diver

the diver had suffered from taravana syndrome, although the reappearance of symptoms after the initial resolution was atypical.

Gempp et al. 2006[20] – maximum depth 18 msw

This case reported a 21-year-old man who complained of various neurological symptoms two hours after a series of BH dives made over approximately one hour. The diver was a sailor in the French navy, was healthy, did not smoke, and had been practicing BH training for the previous three months. Symptoms included: dizziness, visual disturbance, tightness of the chest accompanied by dyspnoea, flushed face, and numbness of all limbs involving the right side of the face. Before transferal to a HBOT unit, the diver was administered normobaric oxygen and on arrival the symptoms had resolved. The patient was treated for neurological DCI using HBOT, and given intravenous rehydration (Ringer lactate, 1 L), aspirin (250 mg), and buflomedil (400 mg) orally. Contrast transcranial Doppler ultrasound revealed appearance of bubbles, suggesting that the patient had a large right-to-left shunt most likely due to a patent foramen ovale, although a pulmonary shunt remained a possible cause. No lesions were detected in the brain upon MRI. The patient was advised to stop scuba diving and to avoid repetitive deep dives with short surface intervals. The authors advised that anyone who experiences unusual symptoms after BH diving should seek immediate medical attention due to the risk of DCI.

Guerreiro et al. 2018[21] – maximum depth 30 msw

In this case, a 39-year-old male who had performed 30 extreme BH dives (two minutes in duration to approximately 30 metres over five hours with short surface intervals) developed transient expressive aphasia and headache. On examination with MRI, he exhibited a left frontal white matter lesion. He was given three sessions of HBOT and at one month follow-up, the patient was asymptomatic, and MRI was clear. The authors noted that accumulation of nitrogen in blood and tissues after repetitive BH diving has been suggested to cause endothelial dysfunction and disruption of the blood-brain barrier, with subsequent hyperpermeability in microvasculature and vasogenic oedema.

Kohshi et al. 2000[22] – maximum depth 22 and 23 msw

This paper reports on two cases, both professional (Ama) male divers of 33 and 39-years-old who presented with neurological symptoms following repetitive BH dives (Tables 1 and 2). Both were healthy but smoked. Diver One had a sudden onset of dizziness and blurred vision; MRI of the brain on the 4th day after the onset showed two hyperintense cerebral lesions in the left occipital lobe and right basal ganglia. He was treated with dexamethasone (12 mg i.v. daily for three days), followed by drug tapering of 4 mg daily over three days. His vision improved over

Table 2

Symptoms and findings reported following breath-hold dives in the 18 studies included in the review; CT – computed tomography scan; EB – Eftedal-Brubakk bubble grade; MRI –magnetic resonance imaging scan; NA – not available

Symptoms	Cases	Reference(s)	Time of symptom onset (by reference number)
Neurological symptoms (not specified)	28	15	15, NA
Vertigo/dizziness	9	18–20; 22; 24–26	18 NA; 19 while diving; 20, two hours after diving; 22, while diving; 24, after diving; 25, while diving; 26, immediately after diving
Limb paraesthesia	6	17; 18; 20; 23; 25; 26	17, immediately after diving; 18 NA; 20, 2 h after diving; 23, immediately after diving; 25, 2 h after diving; 26, while diving
Visual disturbances	6	19; 20; 22; 24–26	19, while diving; 20, two hours after diving; 22, while diving; 24, after diving; 25, 2 h after diving; 26, immediately after diving
Facial paraesthesia	5	17; 20; 26.	17, immediately after diving; 20, 2 h after diving; 26, NA
Hemiparesis/hemiplegia	5	18; 22; 27; 28	18 NA; 22 NA; 27, 3 h after surfacing; 28, while surfacing
Non-specified paraesthesia/sensory disturbance	4	19; 22; 24; 27	19, while diving; 22, while diving; 24, after diving; 27, 3 h after diving
Gait disturbances	4	23; 24; 26	23, 1–2 h after diving; 24, after diving; 26, immediately after diving
Asthenia (weakness)	4	17; 22; 25; 27	17, immediately after diving; 22, while diving; 29, within 30 mins of diving; 27, upon surfacing
Nausea	3	24–26	24, after diving; 25, while diving; 26, immediately after diving
Dyspnoea/ chest tightness or pain	3	20; 24; 25	20, 2 h after diving; 24, after diving; 25, 2 h after diving
Speech impairments/aphasia	2	23; 27	23, immediately after diving; 27, upon surfacing
Buccal rhyme deviation	2	17	17, 24 h after diving
Ringing in ears	1	18	18 NA
Confusion	1	18	18 NA
Headache	1	19	19, while diving
Tingling	1	17	17, immediately after diving
Limb pain	1	25	25, 30 mins after diving
Seizure	1	19	19, day after diving
Cerebral palsy	1	27	27, 3 h after surfacing

Diagnostic/laboratory test results			Imaging		
Abnormal heart rate	1	19		Cases	Reference(s)
Abnormal blood count workup	1	17	MRI – lesions in the brain	10	17; 19; 21–24; 26; 27
Abnormal Glasgow coma scale	1	19	CT – brain, hypodense areas	4	17–19; 27
Abnormal oxygen saturation	1	19	CT – pulmonary, ground glass pattern	1	19
Abnormal blood gases	1	19	Echocardiogram (cardiac bubbles)	1 (EB grade 4 max)	32

the next three weeks, but he still had residual right lower quadrantanopsia. At four weeks, his MRI was normal. Diver Two was admitted with moderate right hemiparesis and a hemisensory disturbance; upon MRI examination on the third day after onset three hyperintense cerebral lesions were observed. This patient received daily HBOT for four days with signs of improvement, and MRI made two weeks later showed a reduction of the hyperintensities in the left parietal and basal ganglia but not the lesion in the right frontal lobe. Upon discharge, he retained some numbness in his right upper arm. The authors concluded that diving accidents such as those reported here had not occurred in Japan before this date (2000) and this may be due to transience of symptoms in many cases or the possibility that the Japanese Ama communities keep such accidents secret.

Kohshi et al. 2020[23] – maximum depth 20 msw

This report focused on a 65-year-old male Ama fisherman who was in good health and had started diving at the age of 30. At the end of his morning dive shift, the diver experienced slurred speech and right-handed paraesthesia, and he was unable to walk properly. The diver received an MRI within two hours of the accident; hyperintense areas on fluid-attenuated inversion recovery (FLAIR) and diffusion weighted imaging were shown in the pons and right-sided parietal lobe. Diagnosis was given as hyperacute pontine ischaemia caused by AGE and a subacute ischaemic lesion in the parietal white matter. The diver was given HBOT in a monoplace chamber and received intravenous rehydration (Ringer's lactate, 1,000 mL). His gait disturbance resolved the next day and HBOT was continued over the next seven days; he was discharged with some residual numbness in his hand, which had resolved on follow-up at five months. A follow-up MRI showed a reduction of the pontine hyperintensity area but no dramatic change in the right parietal lesion. The authors concluded that repetitive BH dives tend to induce stroke-like neurological disorders which are occasionally serious.

Matuso et al. 2014[24] – maximum depth 20 and 30 msw

This paper reported two cases of BH divers presenting with cerebral DCS, who were then investigated with MRI to demonstrate distinctive characteristics of this condition. The first diver presented with right hemiparesthesia, diplopia, and gait disturbance after BH diving to a depth of 20 msw. Neurological exam revealed: left abducens nerve palsy, right-sided sensory disturbance, dysmetria, and ataxic gait. Upon MRI, multiple hyperintense lesions in the right frontal lobe, bilateral thalamus, pons, and right cerebellar hemisphere were observed and the diver was treated with HBOT. Two weeks later, all symptoms had improved, and the MRI findings were attenuated. The second patient had made BH dives to 25–30 msw over a period of six hours. He presented with left quadrant hemianopia and an unstable gait. Upon MRI, hyperintense areas in the bilateral occipito-parietal lobes were seen. This patient also received HBOT, and after three weeks his neurological symptoms disappeared and multiple hyperintense lesions on MRI were attenuated. The authors concluded that in these cases, vasogenic oedema had caused cerebral DCS and that MRI is often more useful compared with other imaging modalities for the examination of patients with DCI. In particular, they noted that AGE can be differentiated from DCS with the use of diffusion weighted imaging and apparent diffusion coefficient mapping.

Paulev 1965[25] – maximum depth 20 metres of fresh water (mfw)

In this paper, Poul-Erik Paulev reported on his own experience following repeated BH dives made to 15–20 m in the Norwegian Navy escape-training tank. Paulev made BH dives over a total period of about five hours; during the last two hours of diving, he experienced some nausea, dizziness, and eructation. Thirty-minutes after the last dive he presented with pain in the hip, right knee, and general fatigue and weakness in the whole right side of his body. Two hours after surfacing he also experienced severe chest pain, paraesthesia of the right hand and blurred vision; one hour later a colleague found him overtly pale and weak, and he was recompressed on US Navy Table 3. Following this treatment, all symptoms resolved bar some residual weakness in his right hand. Paulev reported that there were three similar cases experienced by the training tank training staff within a year prior to his accident. In each, neurological symptoms were present and were relieved by HBOT. Paulev concluded that there was little reason to question that these cases were anything other than DCS.

Tamaki et al. 2010[26] – maximum depth 25 msw

This case study reports on a 34-year-old Japanese Ama diver who had developed neurological symptoms during repetitive dives to 22 msw. His diving pattern generally involved dives of 40–80 s duration with 20–30 s surface intervals over a period of around six hours. After around two hours in the water, he noticed paraesthesia in his right hand, which did not worsen. At the end of his shift, he reported symptoms of nausea, dizziness, and double vision, with some disturbances in gait. Arriving home, he had paraesthesia in his cheek and toe on the right side of his body. With no abatement of symptoms, the next morning he went to hospital. Upon MRI, five hyperintense lesions were found in the right and left basal ganglia, right frontal lobe, pons, and right cerebellar hemispheres. He was treated with HBOT on US Navy Table 6 with no resolution of symptoms; he was treated daily thereafter for ten days. His symptoms gradually resolved, though the residual numbness in his hand persisted for nearly a year. The authors recommended that as the Ama divers tend to harvest the deep ocean floor, longer surface intervals should be taken to prevent DCS; they noted that in this case the diver tended to perform shorter surface intervals

than most Ama divers (20–30 s), although his dive depth and duration were similar.

Tetzlaff et al. 2016[27] – maximum depth 100 msw

In this case, a healthy, non-smoker, 31-year-old competitive BH diver was treated for motor weakness in his right arm and difficulty speaking following a 100 msw training dive; this was the third of three dives with a BH time of four minutes for each. He was administered normobaric oxygen en-route to the hospital, where right-sided hemiplegia, hypesthesia, cerebral palsy and aphasia were noted. Upon CT imaging, an ischaemic lesion in the left frontotemporal region was observed. Heparin was given along with HBOT on US Navy Table 6, with a second session performed the following day with no noticeable improvement. After repatriation, HBOT continued over seven sessions; MRI findings noted a large ischaemic lesion in the area of the left medial cerebral artery. Following treatment, and rehabilitation over several months, paresis of his right leg and hypesthesia improved but he could not return to his profession as a wood worker. The authors concluded that this case provided evidence that serious neurological injury after deep BH dives may occur more often than previously thought.

Yanagawa et al. 2018[28] – maximum depth 5 msw

This case focused on a 74-year-old Japanese female Ama diver who developed hemiparesis on ascent from a BH dive to 5 msw, following multiple dives made over three hours. She had hypertension that was medicated. On examination with echocardiography one hour after her accident, no bubbles could be observed in her inferior vena cava and she was diagnosed with endogenous cerebral ischaemia, not induced by DCS or AGE. On arrival at hospital various tests including MRI confirmed the initial diagnosis, and she received antiplatelet therapy. The authors comment on the usefulness of ultrasound for on-site differential diagnosis.

EXPERIMENTAL STUDIES (SEE TABLES 1 AND 2)

Barak et al. 2020[29] – maximum depth 35 and 40 msw

This study investigated the multifactorial nature of DCS specifically in BH diving, investigating causes other than bubbles. In protocol one, eleven BH divers performed eight deep (35 msw) dives with surface intervals of three minutes and cumulative BH time of 12 minutes. In protocol two, the same divers participated in a six-hour BH session, diving multiple times throughout to depths between 15–40 msw; surface intervals were not dictated. Endothelium-dependent vasodilation of the brachial artery, via flow-mediated dilation (FMD), and the number of microparticles (MPs) were assessed before and after each protocol. Absolute FMD was reduced following both diving protocols ($P < 0.001$), and there was a difference in the number of MPs produced between protocols ($P = 0.007$), with both increasing post-dive. The authors concluded that both protocols, which represented deep or repeated BH dives, seemed to cause endothelial dysfunction that may play an important role in neurological DCS (in particular, stroke-like symptoms) in addition to bubbles.

Boussuges et al. 1997[30] – mean depth 35 msw

This study had the simple aim to detect any circulating bubbles after BH diving in spearfishermen. Ten BH divers took part while participating at a deep harpoon fishing event in Minorca, Spain, in 1995. Bubbles were monitored with continuous wave Doppler over the left chest immediately after the last dive and around 30 minutes post-dive. Two-dimensional echocardiography was also used, obtaining images from the parasternal view, again immediately after diving. The mean maximum depth achieved by the ten divers was 35 msw (range 24–40 msw) for a mean duration of 4 h 03 min (2–6 h). No evidence of circulating bubbles was found with either technique, despite the relatively 'aggressive dives'. However, the authors noted that their study was limited both by the number of subjects, and that their measurements may not have been comprehensive enough to detect any bubbles that evolved. They noted that dives published in the literature suggested the onset of neurological DCI after BH diving often involved underwater scooters, delivering divers to deeper depths with longer bottom times due to the swift movement up and down of the divers to their target.[31]

Cialoni et al. 2016[32] – median depth 40.25 mfw

This study focused on one 53-year-old male spearfishing BH diver to investigate bubbles loads following BH dives. Transthoracic echocardiography was performed on the diver 15 minutes before diving and at 15-minute intervals for 90 minutes after diving in a 42-meter-deep pool, who determined his own diving pattern. Median depth was 40.25 mfw (range 6.2–41.7 mfw), mean diving time was 140.9 (SD 42.1) s and mean surface interval was 593.8 (SD 540) s, with a surface interval ratio of 3:4. Over the first 45 min post-dive, high bubbles loads were observed in all images at grade 4 Eftedal Brubakk (Scale from 1–5; 1 equates to a very low bubble load in the heart, 5 is a very high load with the heart obscured by bubbles in the field). At 60 mins to 90 mins, the load gradually decreased to Eftedal Brubakk grade 1. The authors concluded that despite the limitation of investigating one diver only, high bubble grades can occur after BH diving and that ordinary methods to predict inert gas supersaturation may not be able to predict taravana syndrome cases.

Discussion

Upon systematic review of the literature for studies reporting a convergence of breath-hold diving and DCI, only 14 clinical and three experimental papers of relevance were

found. The majority (10) of these reports related to spear fishing and food gathering, which prior to the advent of competitive freediving would naturally make up the bulk of this type of diving. As reports are few, it was interesting that in the case study reported by Kohshi,[22] the authors noted that diving accidents involving taravana syndrome / cerebral DCI had not occurred in Japan before 2000 to their knowledge; they explained this may be due to symptom transience/ spontaneous resolution but they did also acknowledge the possibility that the Japanese Ama communities keep such accidents secret.

It is of note that only one study reported on a freediving accident, given that competitive apnoea and freediving is now more common.[27] However, there does seem to be much in the way of anecdotal data involving freedivers, as evidenced by the database created to investigate the risk of DCS in extreme BH dives by Fitz-Clarke, who gathered information from 'reliable sources', including websites maintained by champion divers. These data identified 192 dives to 100 m or deeper with two cases of DCS recorded.[9,10] Perhaps some impetus should be made to follow competitive freedivers and publish findings if more is to be learned on this subject. It would be helpful if the BH apnoea diving community could be made aware that in gathering data, the safety of their sport would likely improve as researchers would be able to optimise techniques/protocols to aid this. For example, several of the studies reported here discuss the surface intervals employed by the afflicted divers, reporting that they were thought to be inadequate. They go on to recommend that the surface interval be increased to improve their safety in the future. Table 1 reports all surface intervals where available and shows great variation, from 'a few seconds', to 20–30s, to 5–6 mins; all of these for dives of not dissimilar depths (18–25 m), although the task undertaken during the dive might be very different. It is reassuring, however, to note that the deepest dive reported (100 m) does cite the longest surface interval at 15 mins. The importance of an appropriate surface interval between BH dives for the safety of divers should not be underestimated and is an increasingly common safety strategy used by BH divers. If appropriate data were gathered on this parameter from apnoea competitions/training sessions, then we would be able to offer the participants meaningful and precise advice.

Of the 14 case studies included here, eight made a diagnosis of cerebral DCS, and four mentioned DCI or AGE. In the remaining cases, one gave a diagnosis of endogenous cerebral ischaemia, caused by existing hypertension;[28] and the other, although not giving a diagnosis of DCS, suggested that nitrogen in the body after repetitive BH could cause endothelial dysfunction and disruption of the blood–brain barrier, with subsequent hyperpermeability in the microvasculature and vasogenic oedema. However, a recent BH study has suggested that disruption to the blood-brain barrier after static apnoea is transient and minor in nature and found that apnoea did not show any indication of neuronal damage.[21,33]

There is consensus in the scientific literature that BH-divers can suffer neurological symptoms after diving but to what extent this attributable to DCS or AGE (thus a decompression illness [DCI]) is not completely understood.[4,20,22,27,34–38] As mentioned previously, theoretical calculations have been made that suggest over the course of a bout of repetitive diving, nitrogen supersaturation and thus the evolution of bubbles in the circulation and tissues is possible, with any resulting illness identifying DCS as the likely culprit.[6,39] However, a single deep dive could result in AGE, possibly from expansion from a collapsed state of lung compression and pulmonary atelectasis, causing overexpansion of regional lung segments during ascent. If one part of the lung does not open up, other parts will suffer from the gas expanding to volumes beyond the anatomical limits in that region. In an experimental simulation of lung volumes below residual volume MRI with hyperpolarised gas has showed regional bronchial collapse in volunteer elite divers.[40]

The risk of AGE from a single deep dive could potentially be even higher if the diver uses glossopharyngeal insufflation to overfill the lungs prior to diving, as this manoeuvre has been shown to cause transient neurological symptoms suggestive of AGE when performed dry and in water.[41]

Conclusions

In summary, this review finds that the literature supports both DCS and AGE as potential causes of DCI in BH divers; both should be considered a risk for this cohort of divers, just as for those breathing compressed gas while underwater. The review has identified some gaps in the literature for future study, especially the optimisation of surface interval periods, but also the lingering question whether single dive DCI is AGE or DCS. We also suggest using DCI as terminology when the underlying pathology is unclear, as DCI should cover DCS, AGE and the older terminology of Taravana syndrome.

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