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TARAVANA REVISITED DECOMPRESSION ILLNESS AFTER BREATH-HOLD DIVING

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Key Words
Breathhold diving, decompression illness.

Introduction

Decompression Illness (DCI) following breath-hold (BH) diving is extremely rare. In the past there were numerous BH divers around the world, such as Ama and katsugi divers of Japan, hae-nyo divers of Korea and sponge divers of Greece and Turkey, but now this mode of diving is much less common. These divers do not normally suffer from DCI.

Notwithstanding the rarity of DCI from BH diving, it does occur following extremes of BH diving. In 1958 E R Cross reported a condition known as "Taravana" among pearl divers of the Tuamotu Archipelago near Tahiti.[1] These divers did repetitive BH dives and they suffered from what appeared to be symptoms of DCI. Seven years later Paulev, a naval medical officer, described his personal experience of DCI from BH diving.[2]

Due to the rarity of this condition, it is likely that most medical practitioners are unaware of its existence. This paper reviews the condition and reports two Australian cases of DCI from BH diving.

Taravana

E R Cross described a diving syndrome, called Taravana, in Tuamotu Islander divers working in the Takatopo Lagoon.[1] Taravana is a Paumotan name meaning to fall crazily (tara = to fall; vana = crazily). The report listed 35 male divers. Twelve of them suffered from vertigo and one died. The ages ranged from 19 to 62, and the greatest depth dived was 25 "brasses". A brass is the distance one can reach with outstretched arms and

therefore varies from diver to diver. A brass was the local equivalent of a fathom (6 ft). If this conversion is accurate, the greatest depth dived was some 45 m (150 ft). Cross also mentioned 8 female divers, only one of whom suffered from vertigo. Their ages ranged from 22 to 59 and the depths ranged from 5 to 24 brasses (9 to 43 m or 30 to 144 ft).

Typically the islanders dived from a canoe or an outrigger. They descended using a weight of about 4 to 6 kg (8 to 12 lb) attached to a line and wore goggles or a face mask. Divers at Hikueru Lagoon would hyperventilate for 3 to 10 minutes before diving. The technique was a deep inhalation, followed by deep expiration accompanied by a long drawn "whoooeee" sound. Before the dive, the diver lowered himself into the water and continued to hyperventilate at a faster rate. Just before he dived, he raised himself out of water to the waist, took a deep breath and descended feet first holding the weight. Time on the bottom ranged from 30 to 60 seconds. Ascent time was no more than 20 seconds from depths of 30 to 39 m (100 to 130 ft). As soon as he reached the surface, the diver hyperventilated again for 3 to 10 minutes and then dived. The maximum duration of a dive, from surface to surface, as observed by Cross in Hikueru was 2 minutes and 35 seconds. The average however, was 1 minute 30 seconds for a series of timed dives.

In Hikueru Lagoon, there were an estimated 235 divers. At the end of a 6 hour working day, 47 divers were affected by various symptoms of Taravana. 34 suffered vertigo, nausea and "mental anguish". Six were paralysed, partially or completely. Three suffered temporary unconsciousness and two were mentally affected. Two divers had died.

Cross related further that, in 1958, he spent a morning in the canoe of Turoa Hutihuti at Hikueru Atoll.[3] .."prior to his first dive of the day and during rest periods in and alongside the canoe, he hyperventilated for periods of three to ten minutes. During hyperventilation, the *tete*, or helper, prepared the weighted descending line and the rope and bag for the shell. Whilst continuing to hyperventilate, Turoa went over the side of the canoe and placed the weight between his feet. His rate of hyperventilation increased for about one minute just prior to descent. He then dropped rapidly to the bottom. The diver's time on the bottom and the duration of hyperventilation seemed to depend more on the abundance and quantity of shell in the harvest area than on physiological needs. Bottom time varied from 1 to 2 minutes. In the afternoon, he took me to all parts of the lagoon to observe other divers at work and to talk to divers who had been afflicted by various symptoms of Taravana. My diver told me that when the shell was abundant and of good quality, some divers hyperventilated only briefly but extended their bottom time to the break point of breath holding. Their time on the surface became very short as they dove to harvest the abundant shell they had found. It was then that the diver suffered the syndrome of Taravana".

The most common symptoms of Taravana were vertigo, nausea and, less commonly, mental anguish. Occasionally, vertigo was the only symptom

It has been stated that divers from Mangareva never experienced Taravana. The likely reason was that these divers had longer surface intervals, being 12 to 15 minutes instead of 4 to 10 minutes, and these divers used a less forceful but longer period of hyperventilation.

The reader is referred to the original article by ER Cross for further information.[1] Some of the symptoms of Taravana could be due to hypoxia, hypercarbia and drowning, but certainly other symptoms are highly suggestive of DCI, particularly the neurological symptoms.

It is of interest that the divers hyperventilated before diving. Hyperventilation lowers $PaCO_2$ but, although this prolongs breath-hold time, the diver risks loss of consciousness from hypoxia due to a low PaO_2 before $PaCO_2$ rises enough to provide the stimulus for another breath. Alveolar PCO_2 rises as a BH dive proceeds and normally hypercapnia provides the stimulus for surfacing. With hyperventilation, PCO_2 can be lowered so much that a dangerously low level of PO_2 (around 25–30 mm Hg), which can cause unconsciousness which may lead to drowning, can be reached before the PCO_2 reaches a level (50 mm Hg) that stimulates breathing.[4] Although oxygen concentration falls during descent this provides no hypoxic stimulation for breathing because the PO_2 remains relatively high at depth because of the increased pressure. Alveolar PO_2 increases with depth as the alveolar gas is compressed. During ascent, alveolar PO_2 decreases, partly due to oxygen consumption, but mainly due to decreasing pressure. There is a danger of hypoxia, just before reaching the surface, causing unconsciousness. This has been called "shallow water blackout" although it is more accurately hypoxia of ascent. When the alveolar PO_2 falls below the mixed venous PO_2, there is a transfer of oxygen from mixed venous blood to alveolar gas which raises the arterial PO_2. Although this is a transient effect, it may be an important factor in preventing loss of consciousness in the final stages of ascent. Yet this did not appear to occur often with the Tuamotu divers, perhaps because they descend without effort and do not swim up but are pulled up. Nevertheless 5 out of 235 divers went unconscious or died in the water, which is a 2% incidence, during a 6 hour working day.

DCI following BH diving in SETT

Breath-hold diving causing DCI was again reported by Paulev, a Medical Officer in the Royal Danish Navy.[2] The incident occurred in a Submarine Escape Training Tank (SETT) in the Norwegian Naval Base of Haakonsvaern at Bergen, when Paulev spent 8 minutes at 20 m as an attendant in a recompression chamber. After this he performed a number of BH dives in the tank to 20 m as an

instructor supervising escapees. Each dive took 20-25 secs to reach the bottom, where he would sit or walk until he felt the need to breathe (about 2 minutes), then he ascended to the surface, which took 10 – 15 seconds. Surface intervals were between a few seconds and 2 minutes. He was in the water about 5 hours. During the last 2 hours, he experienced nausea, dizziness and eructation (belching). However, during the last 30 minutes, he developed pain in his left hip. The right knee started to hurt and eventually, the right leg felt tired and the right arm was weak. Two hours after leaving the water he had chest pain, abdominal pain, paraesthesia and anaesthesia in the ulnar side of the right hand and blurring of vision. He was compressed to 6 ATA (50 m)and treated with USN Table 3. Treatment was successful although a residual weakness of the right hand persisted.

Three other cases have also been treated in the Norwegian SETT. Each man had been compressed in the hyperbaric chamber before BH diving. All experienced neurological symptoms and were successfully treated, which supported the diagnosis of DCS from BH diving.

In his discussion, Paulev suggested that for breath-hold dives to 20 m the diver should not suffer from DCI if the surface interval was equal or greater than the time at depth. However, if the surface interval was only half the bottom time, the risks DCI was higher. For deeper BH dives, to 36.6 m (120 ft), a surface interval of 3 times the bottom time was needed to avoid DCS. A 10 minute surface interval may permit an indefinite series of dives to a depth of 42.7 m (140 ft). Perhaps this longer surface interval explains why the divers from Mangareva did not suffer from Taravana.

The symptoms these divers all shared were nausea and dizziness. The surface intervals had all been short, less than 10 minutes.

Australian cases

Case 1

ABC is a fit 40 year old Sales Representative (186 cm tall, weight 74 Kg, BMI 21.39) whose hobby is spear-fishing. He has been breath-hold diving since the age of 14 and dives regularly, at least once or twice a week. ABC considers that he has never experienced any problems while diving. However he commented that he had been close to suffering from “shallow water blackout” about 6 times. He is married with 3 children and is in good general health.

The symptoms which led to his presentation for treatment came on after he performed repetitive breath-hold diving for over 6 hours hunting crayfish and fish. In this time he did some 40 to 50 dives.

He and his friend started at 0800, dived to depths between 12 and 13.7 m (40 to 45 ft) for about two and a half hours. Each dive lasted two to two and a half minutes. The surface intervals were about 1 minute each.

At about 1030, they moved the boat to deeper water and started diving again at 1100. For three and a half hours, they dived to depths of 90 - 95 feet (27.4–29 msw). Each dive lasted between 2 to 3 minutes. The surface intervals were about 2 minutes after these deeper dives.

On one dive in the deeper depths, he caught a fish after some strenuous swimming. After this he felt exhausted and a little “giddy” and experienced a cramp in his left calf. He surfaced, feeling very tired so he merely floated on his back for a while to rest, then swam towards the boat. Despite feeling unwell, he continued diving.

He subsequently experienced headache (much like a sinus headache from which he suffers); difficulty in focusing with some visual blurring, “giddiness” (which was probably vertigo as there was some spinning), nausea and a feeling that he was going to pass out. He attributed this to his nausea, assuming that he had a viral infection. He also complained of being unable to balance. He could not concentrate and unable to think clearly as well as being lethargic. He denied any symptoms of pain in any joints or any altered sensation anywhere.

He went to work thinking the symptoms would disappear. He found that during the day he achieved nothing at work, even though he spent hours there. At night, he felt tired yet he was unable to sleep. When he got to sleep he woke frequently.

Eventually, 3 days after his dives, he decided to consult a doctor. The doctor thought that he had suffered from a hypoxic episode and would probably benefit from hyperbaric oxygen therapy. He presented the next afternoon for assessment at the Department of Diving and Hyperbaric Medicine.

On examination, he appeared tired. In fact, he fell asleep in the waiting room. He exhibited disturbance of cognitive function and demonstrable cerebellar signs with nystagmus, loss of fluency with limb movement with the finger-nose test and showed dysdiadochokinesis. He had mild ataxia, but there was no intention tremor or hypotonia. His muscle strength was normal with a score of 5/5, he was able to do a “duck walk” without any difficulty. Sensation was normal as were all the reflexes. He was unable to do a Sharpened Romberg Test, falling over in less than 2 to 3 seconds. Even with the Standard Romberg Test he only managed about 10 seconds. His other proprioceptive senses such as vibration and joint position sense were normal. The Mini-mental test score was 21/30, worst affected was his short term memory.

He was treated with a USN Table 6 using the Toronto modification, which has a slow rate of ascent, 60 minutes instead of 30 minutes, from 18 to 9 m (Figure 1). At 9 m oxygen is given for spells of 20 minutes with 5 minute air breaks. Ascent from 9 m takes 30 minutes. He was given 2 litres of IV fluids during treatment. He was dehydrated as he passed no urine in spite of the 2 litres of fluids. There was little improvement after the first oxygen period, although he thought his eyes had cleared, but he still felt quite dazed. After the second period, he felt mentally clearer and his headache and giddiness were gone. By the third period, he performed all the alternating rapid movements perfectly and there was no evidence of dysdiadochokinesis, but was still unable to perform the Sharpened Romberg. At 9 m he felt that he was mentally alert and was back to his "normal" self. However, he still could not manage the Sharpened Romberg, but fared a little better on the Standard Romberg.

On completion of USN 6, he felt he was back to normal. All tests were normal, he managed the Sharpened Romberg without any problems and his Mini-mental Test score was 30/30. The complicating factor was that he developed some retrosternal chest discomfort and a mild cough consistent with mild pulmonary oxygen toxicity. This is unusual, as he did not have any oxygen before treatment and the table was not extended. The unit pulmonary toxicity dose (UPTD) was not excessive.

ABC returned the next day for further treatment with a USN 5. There were no abnormalities before treatment and he remained well subsequently. After the treatment he was free from symptoms of oxygen toxicity. He was symptomless when reviewed 13 weeks later.

Case 2

DEF is a 56 year old ex-pearl diver, who was a champion skin diver in his youth in NSW. He still has a keen interest in breath-hold diving. He is a fit man and is still able to free dive to over 30 m (100 ft). He could hold his breath for about 3 minutes.

In 1994 he had used a dive computer to document an aggregate dive time of 50 minutes within a one hour period. His surface intervals are believed to have been very short, just sufficient to unload his catch and to take another big

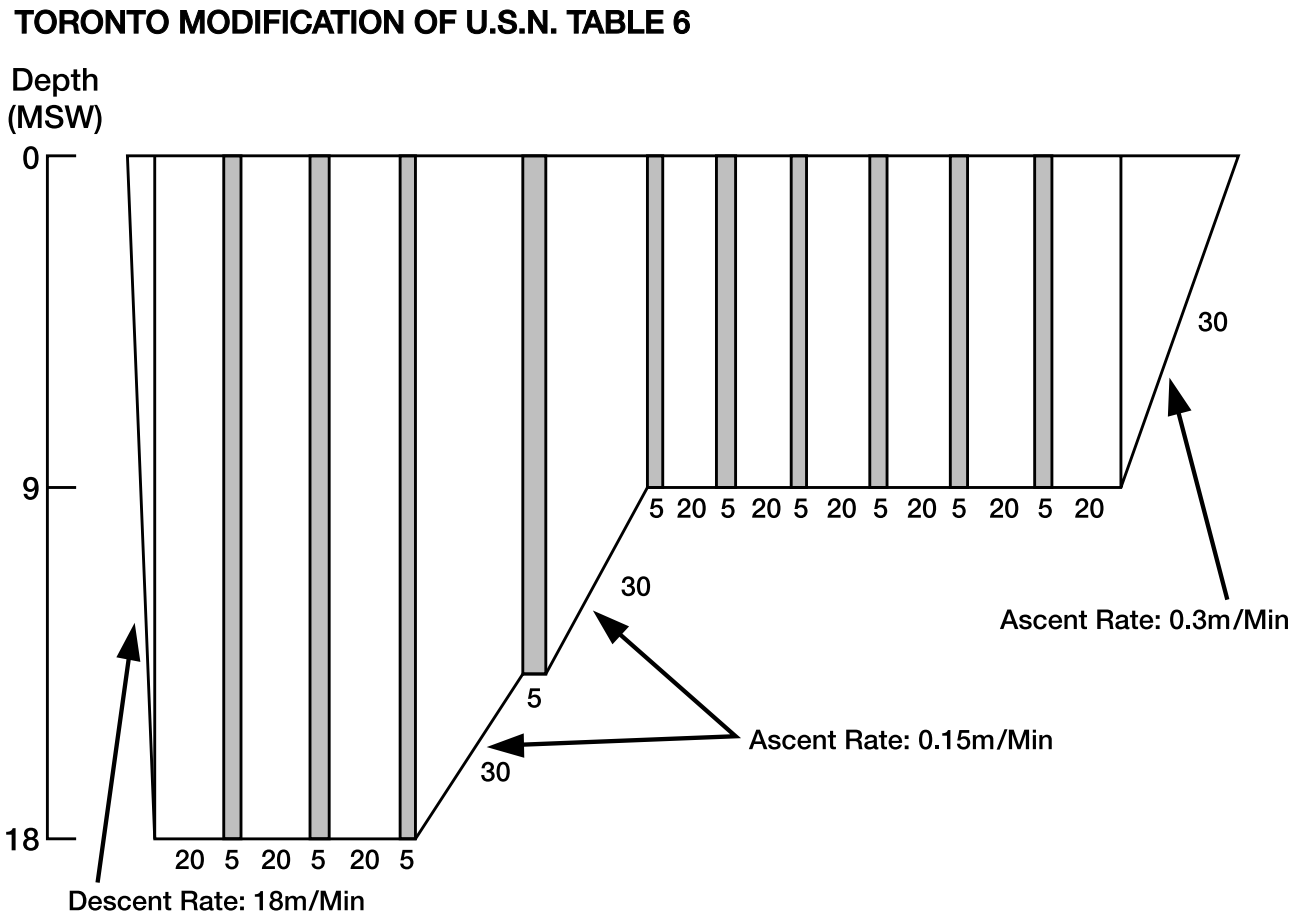

Figure 1. Toronto modification of USN Table 6. The modifications consist of a slower rate of ascent from 18 to 9 m, 0.15 m/minute instead of 0.3 m/minute, and at 9 m 20 minutes oxygen breathing periods separated by 5 minute air breaks. Ascent from 9 m to the surface is at the usual USN rate of 0.3 m/minute (1 ft/minute).

breath. At that time he used to dive for 5 – 6 hours each day.

In 1997 he presented to his local general practitioner with a history of attacks of dizziness, staggering and nausea after BH diving. He had experienced "staggers" previously when pearl diving in the 70s, and he stated that the sensation was similar. He reported that the attacks usually started after diving and were associated with difficulty in walking. On occasions he had been unable to drive home. Generally, these episodes resolved within 12 hours, but the most recent one persisted for 36 hours. Apparently, he had been suffering such symptoms for some 5 years.

He stated that he had consulted another medical practitioner in 1992. The diagnosis then was "a possible middle ear problem" but there was no follow up. It is not clear what the symptoms were that he presented but dizziness and nausea seem likely.

At his most recent consultation DEF had a poor Sharpened Romberg Test, but no other neurological abnormalities were detected. ENT referral was made, but apparently no pathology was found. Tests, including brainstem audiometry and CT scanning were all normal. No perilymph fistula was detected nor was any audio-vestibular pathology demonstrated. He did not seek or receive any treatment after that. From extensive discussion with the medical practitioner involved, the most likely diagnosis appears to be DCI. DEF said that when he used scuba equipment, he experienced no problems.

Discussion

Two earlier reports of DCI following BH diving have been reviewed and two Australian cases of decompression illness which resulted from BH diving have been presented. Since this paper was submitted, a further report of six cases of neurological problems in four breath-hold divers has been published.[5]

DCI from BH diving is extremely uncommon for a number of reasons. Among them are shorter dive times, lesser depths and longer surface intervals. All the cases reported above occurred in warm water and the divers were without thermal protection. Divers working off the coasts of Japan and Korea had to, before they acquired wet suits, come out of the water and huddle over a fire to warm up after a short period in the water. Cold divers have much reduced peripheral circulation and so less tissue exposed to the opportunity for gas uptake.

The Amas do not get symptoms of DCI because they limit the frequency of diving, the depth and the total dive time.[6] The depth might be as much as 25 m, but the total diving time was less than a minute. Nonetheless, Spencer and Okino, who used a precordial ultrasonic bubble detector, studied an ama diver who had done 30 dives to 15 msw in 51 minutes, averaging 53 sec each dive, and found that venous gas emboli were present for as long as one hour after the last dive.[7] It might be that, if such a diver continued to dive for a few more hours, DCI could ensue. In studies with Australian pearl divers, bubbles detected by doppler tended to peak at around 60 minutes, and with more severe profiles, the maximum grades were not reached until 120 minutes after the dive.[8]

Doppler ultrasound techniques have been used extensively to detect moving bubbles in decompression studies. Boussuges et al. used 2D echocardiography and continuous Doppler ultrasound recordings to study 10 breath hold divers who dived to a mean maximum depth of 32 msw (24 - 40 msw) for a mean duration of 3 hours 15 minutes (2- 6 hours). Three divers' dive profiles were recorded on computer. Echo studies were performed in 73% of the cases within half an hour of the last breath hold dive of the day (3-75 mins). They did not find any evidence of circulating air bubbles. Nonetheless, they concluded that the study was insufficient to eliminate the hypothesis of supersaturation in BH diving, and another study is being planned.[9]

Unlike humans, diving animals such as Weddell Seals and other Cetaceans (whales, dolphins etc) do not suffer from DCI or nitrogen narcosis. As they dive, nitrogen uptake is stopped by the collapse of gas-exchanging alveoli. During a dive the major fraction of the Weddell seal's lung gas is compressed into the non-gas exchanging parts of the respiratory tract. At the end of the dive, as pressure is reduced, this gas can expand to open the collapsed alveoli.[10]

Diving medicine texts in the 1970s still contained predictions of lung damage at depths deeper than 30 m.[11] The maximum depth of BH dive for humans was calculated to be about 40 m [Depth = P x (TLC/RV –1) x 10, where P is barometric pressure expressed in atmospheres and 10 represents the sea water equivalent depth in metres of 1 atmosphere].[12] But this calculation fell short of the actual depths reached. In 1951, Felco and Novbelli dived to 35 m. What had been overlooked was the effect of pressure on the rest of the body. The compression of legs and abdomen dislocates a litre or more of blood into the thorax which takes up space occupied by air at the surface. In early 1976, Mayol dived to 86 m. In November 1976, he reached a record depth of 100 m with a single breath in a dive of 3 min 39 secs. Mayol's depth has since been passed by other divers. Recently there has been a resurgence in deep breath-hold diving competitions.[13] The divers, usually wearing wetsuits, use weighted sledges to get down the rope and swim and pull themselves up to the surface. The women's depth record, in April 1999, was 113 m![14]

It is common for spearfishermen to dive regularly to 30 m. Those who are more accomplished can breath-hold dive for 2 to 3 minutes.

Certainly, it appears that repetitive BH dives can give rise to DCI in humans, especially in fit individuals who can breath-hold for prolonged periods and who also have large lung volumes. That DCI is a rarity is probably because most who practise this kind of diving either do not dive to great depths or do not do multiple repetitive dives, with short surface intervals, for 5 to 6 consecutive hours.

It is worth bearing in mind that anyone who has done repetitive BH dives for prolonged periods could suffer from DCI. The most common symptoms appear to be vertigo, nausea and lethargy. In the 2 cases mentioned above, pain was not a symptom. As with DCI from compressed air diving, recompression therapy is highly recommended.

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