Research in Sports Medicine, 14: 163–178, 2006

ISSN 1543-8627 print / 1543-8635 online
DOI: 10.1080/15438620600854710

Taylor & Francis
Taylor & Francis Group

DECOMPRESSION SICKNESS FOLLOWING BREATH-HOLD DIVING

J.D. Schipke

Research Group Experimental Surgery, University Hospital Duesseldorf, Germany

E. Gams

Clinic of Thoracic and Cardiovascular Surgery, Dept. of Surgery I, University Hospital Duesseldorf, Germany

Oliver Kallweit

Research Group Experimental Surgery, University Hospital Duesseldorf, Germany

Despite convincing evidence of a relationship between breath-hold diving and decompression sickness (DCS), the causal connection is only slowly being accepted. Only the more recent textbooks have acknowledged the risks of repetitive breath-hold diving. We compare four groups of breath-hold divers: (1) Japanese and Korean amas and other divers from the Pacific area, (2) instructors at naval training facilities, (3) spear fishers, and (4) free-dive athletes. While the number of amas is likely decreasing, and Scandinavian Navy training facilities recorded only a few accidents, the number of spear fishers suffering accidents is on the rise, in particular

Received 28 December 2005; accepted 20 May 2006.

Reading and correcting of the manuscript by Christian Sherwood (CA) and Prof. Dr. Sinclair Cleveland is greatly appreciated. I thank Mrs. R. Rummel for her valuable secretarial help.

Address correspondence to Prof. Dr. J.D. Schipke, FESC, Forschungsgruppe Experimentelle Chirurgie, Zentrum Operative Medizin I, Universitätsklinikum Düsseldorf, Moorenstr. 5, D 40225 Düsseldorf, Germany. E-mail: schipke@med.uni-duesseldorf.de

during championships or using scooters. Finally, national and international associations (e.g., International Association of Free Drives [IAFD] or Association Internationale pour Le Developpment De L'Apnee [AIDA]) promote free-diving championships including deep diving categories such as constant weight, variable weight, and no limit. A number of free-diving athletes, training for or participating in competitions, are increasingly accident prone as the world record is presently set at a depth of 171 m. This review presents data found after searching Medline and ISI Web of Science and using appropriate Internet search engines (e.g., Google). We report some 90 cases in which DCS occurred after repetitive breath-hold dives. Even today, the risk of suffering from DCS after repetitive breath-hold diving is often not acknowledged. We strongly suggest that breath-hold divers and their advisors and physicians be made aware of the possibility of DCS and of the appropriate therapeutic measures to be taken when DCS is suspected. Because the risk of suffering from DCS increases depending on depth, bottom time, rate of ascent, and duration of surface intervals, some approaches to assess the risks are presented. Regrettably, none of these approaches is widely accepted. We propose therefore the development of easily manageable algorithms for the prevention of those avoidable accidents.

Keywords: nitrogen, saturation, breath-hold diving, DCS

INTRODUCTION

The notion is already 50 years old that breath-hold divers can avoid accumulating dangerous amounts of nitrogen and thus avert decompression sickness (DCS), because they curtail the time spent at appropriate depths (Craig 1961; Lanphier 1956). Only more recently have review articles (Wong 1999, 2000) and textbooks (Edmonds 2002; Ferrigno 2004; Ferrigno and Lundgren 2003) begun to reflect the present state of knowledge.

Consider the following recent example: Benjamin Franz, who holds several world records in breath-hold diving, felt somewhat tired and exhausted in the morning but dived to 137 m, 2 m deeper than the day before. In spite of this good result, he performed 3 to 4 additional dives to 100 m during the afternoon. After the last dive, he suffered from right hemiplegia (Franz 2002). Although stroke was diagnosed initially, diving physicians subsequently discussed also DCS as a possible cause.

The present review (1) collates currently available cases and studies on DCS after repetitive breath-hold diving and (2) presents approaches to assess the risks of this activity. Because both DCS and decompression illness (DCI) are used in the literature, it is remembered that DCI as the generic term also comprises arterial gas embolism (AGE).

DCS AFTER REPETITIVE BREATH-HOLD DIVING

At least four groups of accident-prone breath-hold divers exist: (1) Japanese and Korean amas and other divers from the Pacific area, (2) instructors at naval training facilities, (3) spear fishers, and (4) free-dive athletes.

Amas and Alike

Generations of Japanese and Korean amas have been diving for food and pearls. Some of their diving characteristics were already described more than 70 years ago (Keruoka 1932).

After more than 3 h of repetitively diving to depths between 15 and 25 m, neurological symptoms had developed in two Japanese amas. Magnetic resonance imaging (MRI) showed multiple cerebral infarctions that were in good accord with neurological deficits (Kohshi, Katoh, Abe and Okudera 2001; Kohshi, Kinoshita, Abe, and Okudera 1998). In consequence, conventional therapy in the hyperbaric chamber was recommended, as in DCS cases after SCUBA diving.

Supported amas (=funados) use weights to descend and ropes to quickly ascend. Thus, they usually operate in greater depths than (=cachidos) (Mohri et al. 1995) and dive up to 60 times a shift to depths of 30 m, a single dive lasting approximately 2.5 min.

The typical 4-h shift of male Japanese partially assisted funados comprises both actual diving time and time spent on the boat. On average, only 23 dives are performed that lead to a depth of about 10m. Each dive lasts around 70 s of which about 45 s are bottom time (Hong et al. 1991).

Cachidos of Hegura Island (JP) undertake 90 to 120 dives per day. A single dive reaches a depth between 13 and 22 m, each lasting about 1 min (Mohri et al. 1995). They stay approximately 4.5 h in the water, spending nearly 2 h actually under water. With such diving profiles, these cachidos must reach higher nitrogen concentrations compared with the above cachidos.

During the 3.5-h early shift and the 1.5-h late shift, funados of Hegura Island perform 150 dives to depths between 10 and 30 m. No DCS symptoms were reported in an earlier study on a relatively small cohort (4 cachidos and 4 funados; Mohri et al. 1995).

As the diving habits of the Hegura amas differ from other Japanese amas, 44 amas were scrutinized more closely. Nine had suffered from a panic-like disorder called *chiyamai*; 3 of them for up to 26 years. In one typical case of chiyamai, the ama described a reversible hemiplegia and dysarthria. These symptoms were diagnosed as DCS and treated in a hyperbaric chamber. Because panic-like attacks began in this ama 2 months after this accident, chiyamai might be a late sequel of a neurological DCS (Tochimoto et al. 1998).

Moreover, stroke-like neurological accidents were reported in 9 of 16 Japanese funados of Mishima Island (Kohshi, Katoh, Abe, and Okudera 2001). These funados dive for 3.5 h in the morning and for an additional 1.5 h in the afternoon 20 to 40 times to depths between 10 and 30 m. The most common symptoms were dysarthria, nausea, unconsciousness, and paresis. All neurological disorders disappeared after 10 min to 4 weeks. Thirteen of 16 amas complained about occasional fatigue, euphoria, and vertigo that developed after more than 3 h of repetitive breath-hold diving. Interestingly, these diving accidents were limited to central nervous system disturbances, as none of these amas complained about skin rashes or joint pain (Kohshi et al. 2001).

Another example comes from the Tuamotu archipelago (French Polynesia), where male pearl divers dive 40 to 60 times per day to depths of 30m or more. One dive lasts on average 2 min and the surface interval lasts between 3 and 4 min. The disease that develops with these diving habits has been given the name *Taravana*, meaning "to fall crazily." Mild cases of Taravana are characterised by vertigo and impaired vision. Unconsciousness and partial or complete, acute or chronic paresis of the extremities are the symptoms in severe cases; deaths also have been reported (Cross 1962, 1965).

It is worth noting that on the neighbouring islands of Mangareva, dives with the same profiles are performed, but with surface intervals of 12 to 15 min. This longer surface interval allows requisite amounts of nitrogen to be released from the tissue and, thus, Taravana is here almost unknown (Cross 1962).

Instructors

In the early sixties, three Norwegian Navy instructors developed episodes of DCS after exercises that included repetitive breath-hold dives (Haavelsrud 1963, 1964). After spending 10 min at maximum 3bar in a pressure chamber, they undertook about 20 breath-hold dives to 20 m. Neurological disorders were among the ensuing symptoms, and their rapid disappearance after the onset of recompression is evidence for a prior DCS.

At about the same time, a Danish Navy instructor (Paulev) reported a very similar accident (Paulev 1965). This instructor sat together with a group of beginners in a hyperbaric chamber at maximum 3 bar for 8 min. He then performed 60 dives over a 5-h period to 20 m. Each dive lasted on average 3 min with a surface interval ranging between a few seconds and 2 min. The instructor's symptoms began to occur already after 3 h of training: dizziness, vertigo, and emesis. The symptoms intensified over the following 2 h of training. Two hours after the end of diving, visual

disturbances, paresis in the right arm, and severe thoracic pain had developed. During the 25-min recompression treatment at 6 bar, the symptoms almost entirely disappeared. After 20-h recompression (U.S. Navy table 3; U.S. Navy Diving Manual), the original physical condition was reestablished.

Spear Fishers

Hobby Spear Fishers

In the following, four cases from the 1990 thesis of Heran are presented (Heran 1990). A male Cuban (19 ys; a later deep-dive world champion) had practised spear fishing to 40 to 45 m during 5 h over 5 days with diving times of 2 min and surface intervals of about 4 min. He then suffered from intense skin pain. The pain in joints of the right arm was followed by paralysis. Both disappeared after treatment in the hyperbaric chamber.

A French high-level spear fisher (31 ys), after intensively training over 2 ys and diving on 3 days per week for 7 to 8 h to 30 m, suffered from lethargy, insomnia, and abdominal pain. Some days later, sensory deficits developed in the face, right hand, and thigh. Decompression Sickness was diagnosed in the hospital and all sensory deficits disappeared after 4 sessions in the hyperbaric chamber, while the digestion disorders persisted (Heran 1990).

The third case particularly underlines the need for better education. A French high-level spear fisher (49 ys) suffered within 10 months at least five times from DCS. The last event is described here in more detail. He was diving 10 times per hour to 30 to 40 m. After 2.5 h of spear fishing, he felt a prickle in the right frontal sinus, left fingers, and left leg. Moreover, his trachea felt dry, his vision was impaired, and he suffered from lethargy. All these symptoms spontaneously disappeared after 10 min except for an “instability” and a sense of a “heavy neck” due to later diagnosed upper trapezius muscle contraction. In the following days, DCS was diagnosed after intense examinations (angiography, ophthalmology, audiometry, spirometry, echocardiography, MRI showed small ischemic lesions).

With the last of Heran’s spear fisher cases, an additional instrument to increase the risk of a DCS was introduced: underwater scooters. Ten minutes after the end of 2 h spear fishing using a scooter to 27 to 29 m (3 min bottom and 1.5 min surface), the subject reported impaired vision (e.g., hands seem disproportionately long). In addition, the spear fisher could not remove his gloves and could not stand upright. The coordination problems persisted for 2 h, and he suffered from asthenia in the following 3 days. A cervico-dorso-lumbal curvature, a “heavy” back of the head, and a disturbed equilibrium (tumbled repeatedly) persisted for 4 weeks (Heran 1990).

Another French spear fisher performed about 40 dives to maximum 40 m within 3 h (Fanton, Grandjean, and Sobrepere 1994). Both dives and surface intervals lasted 2 min. After his last dive, the spear fisher lost consciousness and fell into a coma and regained consciousness after oxygen administration, but was temporally and spatially disoriented. He later reported on paraesthesia in both his arms and legs after previous similar dive profiles.

The next case is characterized by its presumably conventional diving profile. After repetitively diving for 1 h to 8 m (each dive ≤ 1min), a diver complained about visual impairment in the left eye. The subsequent examination reported occlusion of a branch of the central retinal vein. The authors concluded that this was a result of DCS (Merle et al. 1997).

Two cases are reported from Australia. A male spear fisher used to dive during 5 to 6 h each day with less traditional profiles. During a given hour, he was under water for about 50 min; that is, the surface intervals were just long enough to take the next breath to dive to maximum 30 m. During one presentation at a general practitioner 3 days later, he complained about dizziness, staggering, and nausea. Apparently, he had been suffering such symptoms for some 5 years after breath-hold diving; a former practitioner had diagnosed a "possible middle ear problem." From extensive discussions with that practitioner, the most likely diagnosis appeared to be DCI (Wong 1999).

Another male spear fisher performed 40 to 50 dives within 6 h (Wong 1999). Within the first 2.5 h, he dove to 12 to 14 m with the dives lasting 2.0 to 2.5 min and the surfaces lasting 1min. After a pause of 30 min, the depth was increased from 27 to 29 m. During 3.5 h, the dives lasted 2 to 3 min and the surface intervals 2 min. Three days later, the diver visited a physician because he continuously felt tired and in fact fell asleep in the waiting room. He complained about nausea and blurred vision and was unable to keep his balance. The physician additionally diagnosed both impaired cognitive function and short-term memory. The symptoms disappeared after several treatments in the hyperbaric chamber (U.S. Navy tables 6 and 5); U.S. Navy Diving Manual.

Around the Balearic Islands (ES-Espagne), 35 cases of DCS in spear fishers were collected after breath-hold diving between 1995 and 2002 (Batle 2002). "Shallow" dives lead to depths of 20 m with breath-hold times of 2 min and surface intervals of 0.5 min. This pattern lasted typically 2.5 h and caused neurological symptoms.

As mentioned, the risks increase considerably for divers using scooters for their underwater activities. With this specific equipment a "deep" dive would consist of a 30-s descent to 30 m, a bottom time of 50 s, and a 30-s ascent. With a surface interval of 2 min, 12 to 15 dives were possible within 1 h. This diving pattern lasted between 3 h and 9 h, leading to neurological symptoms.

During "extreme" dives, depths of up to 63m were reached. Both the dive and the surface interval lasted 2 min, such that 15 to 20 dives were possible within 1h. The accidents happened after total in-water times between 3 h and 8 h, and severe neurological symptoms disappeared after treatment in the hyperbaric chamber (Batle 2002).

Competitive Spear Fishers

The list with spear fisher accidents becomes considerably longer once competition is involved. On day 2 of the European Championships in 1987 (Balearic Islands, ES), one member of the Spanish team diving the same profiles up to 30 m suffered from paraesthesia and weakness of the right arm, while another suffered from aphasia and a right-sided facial paralysis. The symptoms' topography and their disappearance after treatment in a hyperbaric chamber are suggestive of DCS (Heran 1990).

On day 1 of three consecutive days of a national competition, another Spanish professional athlete inspected the area of the competition over 1.5 h with the help of a scooter and performed 20 dives of on average 2 min to 25 to 35 m (Oblare and Pascual 1995). The competition on day 2 lasted for 6 h. Ten dives per hour were performed to depths of 35 m. Each single dive lasted 1.5 min, followed by a surface interval of 3 min. The accident happened on day 3 after 4 h of competition with the same diving pattern as the day before, except that the depth was 32 m. The athlete presented neurological CT-documented disorders that disappeared after recompression in a hyperbaric chamber.

During the World Championship in 2000 on Tahiti, the world champion in breath-hold spear fishing (A.M.) only came in second because he needed to be treated in a hyperbaric chamber owing to DCI-related problems (Volpe 2001a). It was the fifth time that A.M. experienced such problems including visual problems, hemiplegia, and dysarthria, all of which were resolved in the hyperbaric chamber (Malpieri 2002).

This participant felt "unhealthy" after 2 h of competition following the 6 h of exercise the day before. The victim later suggested that the duration of competition but not the depth be limited, since a former world champion (P.C.) also needed treatment in a hyperbaric chamber after repetitive breath-hold dives not deeper than 22 m (Volpe 2001a).

In 2001, the Tahiti experience was almost repeated in Italy. A participant (M.B.) needed hyperbaric chamber treatment in Latina (IT) after breath-hold diving during 2 h to a depth of about 20 m with a profile of 2 min 15 s with surface intervals not longer than 40 to 50 s (Volpe 2001b). The director of the chamber reported on four additional cases (S.P., T.P., F.S., and G.S.) that needed treatment in a hyperbaric chamber after extensive spear fishing. The clinical signs found in these patients were dysarthria, aphasia, and in 3 out of 4 cases hemiplegia or paraesthesia or both. One

other competitive spear fisher (R.C., a member of the Croatian National Team) complained about tunnel vision, hemiplegia, and dysarthria that had self-resolved 3 h after manifestation (Malpieri 2002).

Because of the numerous accidents among breath-holding spear fishers reported in this review, some details of their diving characteristics are summarized. They typically hyperventilate for 2 to 4 min before the first dive. Common, rapidly reached depths vary between 25 and 45 m. Bottom times range between 50 and 90 s followed by a more or less rapid ascent. The surface interval is reported to be shorter than the breath-hold time (Malpieri 2002). If we assume a typical depth of 30 m for 2 min and further assume the ascent plus the descent to last 60 to 90 s, the surface interval will last no longer than 3 min. In case this diving pattern is maintained for 5 h, more than 50 dives (300 min : 6 min) can be performed. Protagonists of spear fishing with such a diving history very likely encounter the risk of suffering DCS. The situation becomes even worse during a spear fishing competition, because the participants are exposed to considerably increased psychic and physical stress. Hence, it is astonishing that more cases of DCS are not reported during a competition (Rolland 1995). The symptoms reported were acute or chronic visual disturbances, dyslalia and aphasia, and paresis and hemiparesis. In severe cases without proper treatment, even death can occur (Malpieri 2002).

Freediving

Freediving was made popular by the 1988 French film *The Big Blue*. Currently, there are more than 20,000 freedivers worldwide; the numbers are increasing (Lindholm and Gennser 2004; McCrory 2003). National and international associations (e.g., IAFD or AIDA) promote freediving championships including several deep-diving categories. An increasing number of freediving athletes, working out for competitions or participating in competitions, are more and more accident prone, as the world record is currently set at 171 m.

At a scientific meeting in Boston, six incidents of neurological problems involving breath-hold divers were reported (Magno, Lundgren, and Ferrigno 1999). Four divers developed neurological problems such as impaired coordination of motion, diplopia, and colour blindness after breath-hold dives. In one of these divers, hemiplegia arose after a single 120-m dive, but likely not owing to DCS, but to an arterial gas embolism that occurred during the descent. The other cases, however, were interpreted as being the result of DCS after repetitive breath-hold dives. On the other hand, the authors admit that some neurological deficits could be the result of rupturing of pulmonary tissue that might have happened via maximum inspiration (lung packing) before the descent.

During his deep dive training, a breath-hold diver performed 10 dives to 30 to 70 m on two consecutive days. During the 90-min surface pause between the two last dives (both ≥70 m), he participated in strenuous work whilst exposed to intense sun irradiation. Five minutes after the last dive, first symptoms developed in his right shoulder. Ten minutes later, the entire right arm was paralysed. In-water recompression was terminated after 10min due to lack of success. The administration of oxygen during transport to hospital provided complete symptomatic relief, and the subsequent analysis of x-ray images and laboratory data exhibited no obvious injuries. It is mentioned that the diving physician on duty refused any DCS treatment arguing that this accident would very unlikely be owing to repetitive breath-hold dives (Welslau and Prohaska 2002).

End of June 2005, a new breath-hold record was set at nearly 210 m (P.M. needed 3 min 28 s). About 20 min after that dive, the diver felt extremely tired and was transported to the hospital, where he was treated for in a hyperbaric chamber. It is worth mentioning that P.M. had lost 8.5 kg body weight during the last 24 days of intensive training due to a severe bacterial intestinal infection (Musimu 2005).

However, breath-hold divers need not reach great depths to develop DCS. A diver experienced headache, dizziness, blurred vision, numbness, and weakness of all four limbs after repetitively diving to only 8 m over 3.5 h. Another diver complained of tiredness, headache, joint pain, and paraesthesia in his upper limbs after repeated dives for about 4 h to only 6 to 8 m. After treatment in a hyperbaric chamber, all symptoms resolved (personal communication in Wong 2000).

DISCUSSION

There is agreement that DCS can occur after repetitive breath-hold diving. This is based on an overwhelming body of evidence, appropriate techniques for DCS diagnosis, and successful therapy of DCS using hyperbaric oxygen treatment.

This perception was not yet accepted in 1965 when Paulev met with his severe accident. According to the prevailing doctrine, DCS could be excluded. Thus, the experts discussed a pulmonary embolism as a consequence of overinflated alveoli (Lanphier 1965b). Typically, such an air embolism would occur only if the breath-hold diver had access to an air supply in the depths. In such a case, however, the injury would manifest itself much more quickly and dramatically than in the cases presented above. A pulmonary barotrauma would, on the other hand, require a relatively unlikely chain of adverse events during the traditional breath-hold diving. Overinflation of pulmonary tissue would be expected only if particular alveoli would contain more air during the ascent than during the

descent. If so, air must then have been relocated during the descent and must have been trapped in other compartments during the ascent. Such air trapping actually can exist in preinjured lungs (Dahlback and Lundgren 1972).

Paulev, being a physician himself, analyzed a hypothetical stay in a decompression chamber lasting 8 min at 3 bar and 13 subsequent breath-hold dives to 20 m using the then-available models. Each dive might last 2 min and be separated by surface intervals of 1min. Under these conditions, an average rapid tissue (= 5-min tissue) would saturate more and more during each individual dive than it would desaturate during surfacing and the following surface period. After 10 such dives, nitrogen partial pressures (pN_2) with a high risk to induce DCS would have been reached. The simulations showed that pN_2 would have exceeded 1500 mmHg in a 5-min tissue. Dives to 30m would have led to an unacceptably high value of ≥2000 mmHg (see Figure 1; Paulev 1965). Even if breath-hold divers do not stay in hyperbaric chambers before their dives—as the Scandinavian

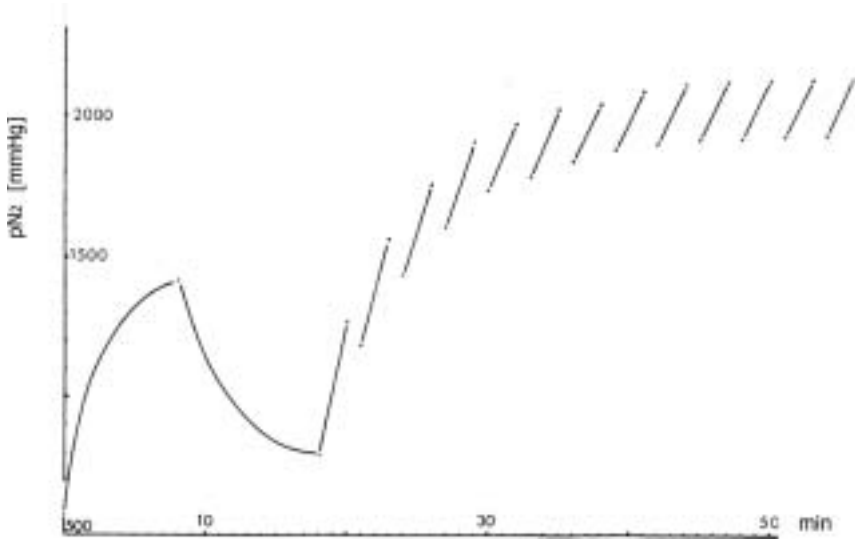

Figure 1. Initial increase in the nitrogen partial pressure (pN_2) during an 8-min stay at 3 bar in a hyperbaric chamber. After a 10-min decompression, 13 breath-hold dives are performed to maximum 33 m (2-min breath-hold and 1-min surface interval). This simulation shows that a maximum nitrogen partial pressure of 2000 mmHg is reached during the final dives (normal nitrogen partial pressures average 600 mmHg). (From Paulev, in *J Appl Physiol*, 20, 1028–1031, 1965, by permission of the Amer. Physiological Society.)

instructors did—a dangerously high pN_2 can build up (Spencer and Okino 1972). (see Figure 2 (Olszowska and Rahn 1987)).

In an early study, the available literature was analysed in order to perform probability calculations of DCS development during repetitive breath-hold dives. The results convincingly support the hypothesis that—dependent on the dive profiles— critical pN_2 values can be both reached and exceeded (Moretti 1968).

In a later study, the concentration of nitrogen dissolved in the cachidos' blood was calculated. The results also demonstrate that nitrogen would easily have been elevated to such levels that DCS would occur, if the duration of the surface intervals had not been long enough (Radermacher et al. 1992).

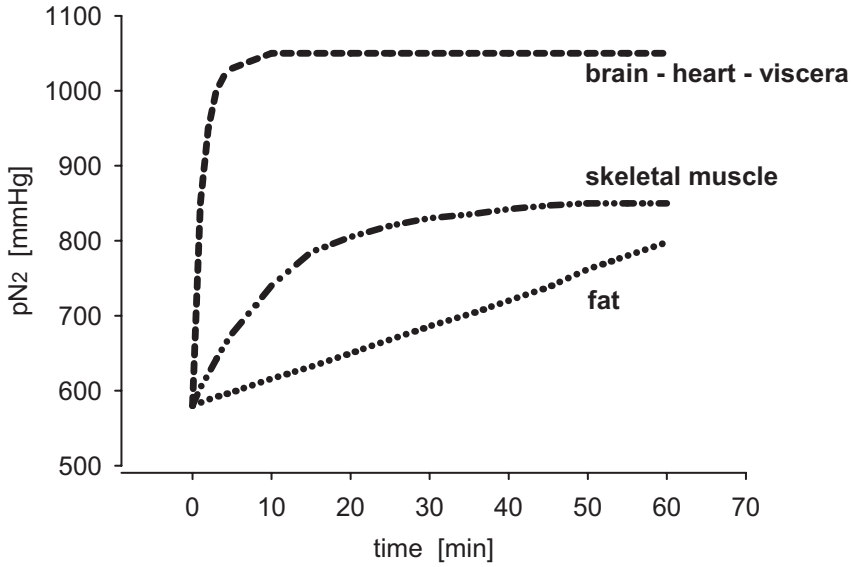

Figure 2. Changes in pN_2 values in different tissues. The simulated diving pattern consisted of 30 dives within 60 min to 20 m. Breath-hold time and surface time both lasted 1min. This diving pattern is typical for Japanese amas. Brain, heart, and viscera are saturated after the first few dives. In contrast, saturation in fat rises continuously. Mean values between immersion and surface are displayed. Actual values in brain, heart, and viscera during immersion are increased by about 110 mmHg and are decreased by 110 mmHg at the end of the surface interval. (From Olszowska et al., in *Man in Stressful Environments*, by permission of Charles C Thomas, Springfield, IL, 1987.)

The notion that DCS can develop from repetitive breath-hold dives is, however, not unanimously supported. In a study involving 10 breath-hold divers, no circulating bubbles were detectable in the right heart even after the divers had performed repetitive dives to 35 m between 2 and 6 h (Boussuges et al. 1997). This result is somewhat surprising and may be explained in part by the relatively few participants. In addition, the ascent velocity seems to play a major role as well as the length of the surface interval (Lanphier 1965b). If, for example, the ratio between the lengths of the surface interval and the dive time equals 1, theoretically, such a dive profile would equal that of a continuous dive at half the depth of the actual repetitive apnea dives (Figure 3). These facts illustrate that divers could indeed very well suffer from DCS after repetitively diving to depths between 30 and 50 m (Bove 2000).

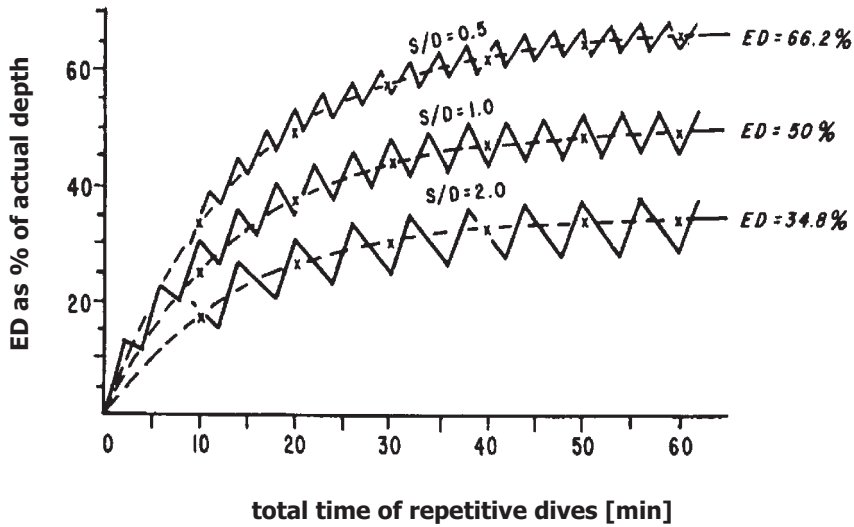

Figure 3. Computed nitrogen pressure over total elapsed time during a series of breath-hold dives in a theoretical tissue with a 10-min half-time. The three saw-tooth lines represent diving profiles with respect to time on the surface (S) and time at the depth (D). From the ratio S/D, a depth can be calculated that would be equivalent to a steady exposure to air while SCUBA diving. If S/D = 1, the equivalent depth (ED) would be 50% of the actual maximum depth, for example, for an actual depth of 30 m, the ED would be equal to 15 m. After transformation to the ED, traditional diving tables become applicable. (From Lanphier, in *Physiology of Breath Hold* Diving and the Ama of Japan. Publication #1341, pp. 227–236, 1965, by permission of National Academy of Science, Washington, DC.)

A straightforward investigation of DCS after repetitive breath-hold dives is difficult. This is in part due to the fact that venous gas emboli are not tightly coupled with DCS. A study on multiday, repetitive, multilevel SCUBA divers (n = 67) reported on venous gas emboli in 91% with none of them developing DCS (Dunford et al. 2002). This observation does not at all mean, however, that such silent bubbles can be disregarded. Two examples from animal experiments demonstrate that bubbles might cause damage other than DCS: (a) in the rabbit, small amounts of venous gas embolism caused delayed impairment of endothelial function (Nossum, Hjelde, and Brubakk 2002); (b) although mammalian lungs are competent filters for microbubbles ≥20 μm, gross blood–brain barrier dysfunction had developed 1 to 2 h after injecting smaller bubbles into the carotid artery of anesthetised guinea pigs, demonstrating that microbubbles impair the integrity of the blood–brain barrier to protein, causing focal oedema (Hills and James 1991).

Other examples additionally show the loose coupling between venous bubble formation and DCS. In one ama, venous bubbles were detected within 1 h after her last dive. This ama had performed a "traditional" diving profile with 30 dives to 15 m within 51 min, each single dive lasting on average 51 s with an average surface interval of 51 s (Spencer and Okino 1972). Very similar results were reported for Australian pearl divers. These divers developed detectable bubbles 1h after their last dive, and some of the divers with strenuous diving profiles even reached a maximum bubble production only 2 h after their last dives (Wong 1996). This time delay in bubble formation may help explain the results of the above negative study (Boussuges et al. 1997). In a different case study, however, these authors do describe the development of intra-alveolar haemorrhage and other risks that can develop during breath-hold diving (Boussuges, Blanc, and Habib 1995).

WHAT CAN WE LEARN?

Breath-hold diving will increase the pN_2 in body tissue. Decompression sickness can develop depending on the number of dives, the depth, the velocity during the ascent, the duration of the surface interval, and the physical activity during these intervals.

The consequences of this information firstly apply to spear fishers with their relatively complex diving profiles. SCUBA divers also can be involved. Should they perform breath-hold dives between extensive compressed air dives, residual nitrogen cannot leave the tissue in substantial quantities. It is in fact possible and even probable that additional nitrogen would saturate body tissue even further. Neither possibility is contained or projected in a decompression table, nor in a decompression computer.

Competitive breath-hold divers and spear fishers seem to be the most affected. With today's depths, high frequency of dives during the training period, and with dive times up to 5min, critical pN_2 values can be reached easily. Using present knowledge and simulation capabilities, accidents could be retrospectively analysed as soon as they become known to the diverse apnoeic dive associations. Once a dive profile is known, serious accidents could be forecast and simulated to clarify their causal background.

CONCLUSION

Despite some continuing scepticism, we have found and summarized 90 cases of probable DCS following breath-hold diving. Analysing such accidents could modify the training habits of breath-hold divers in such a manner that surface intervals of sufficient length become recommended or even obligatory. Perhaps, decompression tables based on almost 40-year-old antecedents (Lanphier 1965a) may become available for breath-hold divers with their various breath-hold dive profiles. Until such a time, however, breath-hold divers, their medical advisors, and decompression facilities should be aware of DCS as a possible danger.

REFERENCES

Batle JM (2002) *The arterial gas embolism for decompression sickness in free diving hunters. The signs and symptoms. A retrospective study on 35 cases*. (Abstract #56). XIV International Congress on Hyperbaric Medicine 2002.

Boussuges A, Abdellaouil S, Gardette B, Sainty JM (1997) Circulating bubbles and breath-hold underwater fishing divers: A two-dimensional echocardiography and continuous wave Doppler study. *Undersea Hyperb Med* 24(4): 309–314.

Boussuges A, Blanc P, Habib G (1995) [Neurologic accident of decompression: A new indication of transesophageal echocardiography]. *Presse Med* 24(18): 853–854.

Bove F (2000) *Decompression sickness from free diving.* Retrieved 3/8/2003, from http://www.scubamed.com/divess.htm.

Craig AB, Jr. (1961) Underwater swimming and loss of consciousness. *J Am Med Assoc* 176: 255–256.

Cross ER (1962) Taravana, *Skin Diver Magazine*. 11: 42–45.

Cross ER (1965) Taravana—Diving syndrome in the Tuamotu diver. In *Physiology of Breath-hold Diving and the Ama of Japan*. (National Research Council. Publication 1341) Washington, DC: National Academy of Science. 207–219.

Dahlback GO, Lundgren CE (1972) Pulmonary air-trapping induced by water immersion. *Aerosp Med* 43 (7): 768–774.

Dunford RG, Vann RD, Gerth WA, Pieper CF, Huggins K, Wacholtz C, Bennett PB (2002) The incidence of venous gas emboli in recreational diving. *Undersea Hyperb Med* 29(4): 247–259.

Edmonds C (2002) Diving and subaquatic medicine (4 ed). In C Edmonds et al., eds. *Diving and subaquatic medicine* (4 ed). New York: Arnold. 615–618.

Fanton Y, Grandjean B, Sobrepere G (1994) Decompression accident under apnea. *Presse Med* 23(23): 1094–1097.

Ferrigno M (2004) Breath-hold diving. In A.A Bove, ed. *Diving medicine* (4 ed). Philadelphia: W.B. Saunders Co, 77–90.

Ferrigno M, Lundgren CE (2003) Breath-hold diving. In PB Bennett, DH Elliot, eds. *Physiology and Medicine of Diving* (5 ed). Philadelphia: W.B. Saunders. 153–180.

Franz B (2002) *Stellungnahme: War es ein Fehler?* Retrieved 3/8/2003, from http://www.benjamin-franz.de/vorwort/benjamin/benjamin.html

Haavelsrud OI (1963) *Reports to the Norwegian Naval Authorities* (November 1963 and January 1964). Bergen, Norway.

Haavelsrud OI (1964) *Reports to the Norwegian Naval Authorities* (March 1964). Bergen, NO:

Heran N (1990) *Les plongeurs en apnee peuvent-ils presenter une maladie de la decompression?* Thesis. Université de Montpellier, Faculté de Médicine.

Hills BA, James PB (1991) Microbubble damage to the blood-brain barrier: Relevance to decompression sickness. *Undersea Biomed Res* 18(2): 111–116.

Hong SK, Henderson J, Olszowska AJ, Hurford WE, Falke KJ, Qvist J, Radermacher P, Shiraki K, Mohri M, Takeuchi H, Zapol WJ, Ahn DW, Choi JK, Park YS (1991) Daily diving pattern of Korean and Japanese breath hold divers (Ama). *Undersea Biomed Res* 18(5–6): 433–443.

Keruoka G (1932) Die Ama und ihre Arbeit. *Arbeitsphysiologie* 5: 239–251.

Kohshi K, Katoh T, Abe H, Okudera T (2001) Neurological accidents in Japanese breath-hold divers: A preliminary report. *J Occup Health* 43: 56–60.

Kohshi K, Kinoshita Y, Abe H, Okudera T (1998) Multiple cerebral infarction in Japanese breath-hold divers: Two case reports. *Mt Sinai J Med* 65(4): 280–283.

Lanphier EH (1956) *Submarine Medicine Practice*. Washington, DC: Bureau of Medicine and Surgery, Dept. of the Navy.

Lanphier EH (1965a) Application of decompression tables to repeated breath hold dives. In *Physiology of Breath Hold Diving and the Ama of Japan* (National Research Council Publication 1341). Washington, DC: National Academy of Science. 227–236.

Lanphier EH (1965b) Overinflation of the lung. In *Handbook of Physiology. Respiration*. Washington, DC: Am Physiol Soc, AP Fishman. 1189–1193.

Lindholm P, Gennser M (2004) [Breath-hold diving—An increasing adventure sport with medical risks]. *Lakartidningen* 101(9): 787–790.

Magno L, Lundgren CEG, Ferrigno M (1999) Neurological problems after breath-hold diving. *Undersea and Hyperbaric Medicine* 26: 28–29.

Malpieri M (2002) *Apnea e PDD: Il Taravana*. Retrieved 3/8/2003, from www.apneamagazine.com/articulo

McCrory P (2003) The big blue. *Br J Sports Med*. 37: 1.

Merle H, Drault JN, Gerard M, Alliot E, Mehdaoui H, Elisabeth L (1997) [Retinal vein occlusion and deep-sea diving]. *J Fr Ophtalmol* 20(6): 456–460.

Mohri M, Torii R, Nagaya K, Shiraki K, Elsner R, Takeuchi H, Park YS, Hong SK (1995) Diving patterns of Ama divers of Hegura Island, Japan. *Undersea and Hyperbaric Medicine* 22: 137–143.

Moretti G (1968) [Decompression sickness as a consequence of repeated diving in apnea]. *Ann Med Nav (Roma)* 73(6): 509–522.

Musimu P (2005) Homepage. www.patrickmusimu.com/

Nossum V, Hjelde A, Brubakk AO (2002) Small amounts of venous gas embolism cause delayed impairment of endothelial function and increase polymorphonuclear neutrophil infiltration. *Eur J Appl Physiol* 86(3): 209–214.

Oblare BL, Pascual FC (1995) Accidente disbarico en pesca submarina. *Med Clin (Barc)* 104: 742–743.

Olszowska AJ, Rahn H (1987) Gas store changes during repetitive breath-hold diving. In K Shiraki, MK Yousef, eds. *Man in Stressfull Environments—Diving, Hyper- and Hypobaric Physiology*. Springfield, IL: Charles Thomas.

Paulev P (1965) Decompression sickness following repeated breath-hold dives. *Journal of Applied Physiology* 20(5): 1028–1031.

Radermacher P, Falke KJ, Park YS, Ahn DW, Hong SK, Qvist J, Zapol WM (1992) Nitrogen tensions in brachial vein blood of Korean Ama divers. *J Appl Physiol* 73(6): 2592–2595.

Rolland JM (1995) *Les accidents de decompression en apnee*. Aresub. Retrieved 3/8/2003, from www.perso.wanadoo.fr/aresub/medecinesubaquatic/taravana.htm

Spencer MP, Okino H (1972) Venous gas emboli following repeated breath hold dives. *Fed Proc* 31: 355–359.

Tochimoto S, Kitamura T, Kurata K, Nakamura I, Koshino Y (1998) "Chiyamai," a panic-like disorder in woman divers from Hegura Island. *Psychiatry Clin Neurosci* 52(4): 425–427.

Volpe G, March, (2001a) Campiopne di pesca in apnea. *Apnea Magazine*. Retrieved 3/8/2003, from www.apneamagazine.com/articolo.php

Volpe G, Bardi M (2001b) Retrieved ___, from www.apneamagazine.com/articolo.php/380

Welslau W, Prohaska R (2002) Dekompressionskrankheit (DCS) beim Apnoetieftauchtraining – Fallberichte und 'anekdotische' Fallsammlung. *Caisson* 17: 42–43.

Wong RM (2000) Breath-hold diving can cause decompression illness, *SPUMS J*, 30: 2–6.

Wong RM (1996) Doppler studies on the dive schedules of the pearl divers of Broome. *SPUMS J* 26: 36–42.

Wong RM (1999) Taravana revisited; Decompression illness after breath-hold diving. *SPUMS J* 29(3): 126–131.