Decompression sickness in breath-hold divers: A review

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Abstract
Although it has been generally assumed that the risk of decompression sickness is virtually zero during a single breath-hold dive in humans, repeated dives may result in a cumulative increase in the tissue and blood nitrogen tension. Many species of marine mammals perform extensive foraging bouts with deep and long dives interspersed by a short surface interval, and some human divers regularly perform repeated dives to 30–40 m or a single dive to more than 200 m, all of which may result in nitrogen concentrations that elicit symptoms of decompression sickness. Neurological problems have been reported in humans after single or repeated dives and recent necropsy reports in stranded marine mammals were suggestive of decompression sickness-like symptoms. Modelling attempts have suggested that marine mammals may live permanently with elevated nitrogen concentrations and may be at risk when altering their dive behaviour. In humans, non-pathogenic bubbles have been recorded and symptoms of decompression sickness have been reported after repeated dives to modest depths. The mechanisms implicated in these accidents indicate that repeated breath-hold dives with short surface intervals are factors that predispose to decompression sickness. During deep diving, the effect of pulmonary shunts and/or lung collapse may play a major role in reducing the incidence of decompression sickness in humans and marine mammals.

Keywords: *Bubbles, mammal, human, N_2, diving*

Introduction

Snorkelling and breath-hold diving are enjoyed by millions of people around the world. Although most people dive to shallow depths for short durations, spear-fishermen and competitive breath-hold divers routinely perform repeated dives to depths greater than 30 m for more than a minute (Schipke, Gams, & Kallweit, 2006). While human breath-hold performances are well below those of marine mammals, depth and dive durations have been increased markedly in the last few years. The current depth record is 214 m and breath-hold durations of more than 10 min in static, shallow dives have been recorded. Elite breath-hold divers commonly perform dives lasting 3–3.5 min, with very rapid descent and ascent rates. These factors raise questions about the health risks involved, as such dive practices may cause loss of consciousness from hypoxia during ascent, barotraumas, and decompression sickness (McCrory, Matser, Cantu, & Ferrigno, 2004).

Decompression sickness arises mainly from gas phase separation in body tissues resulting in bubble formation. The bubbles can cause a variety of pathological signs and symptoms depending on the where they form. Arterial gas embolism occurs when lung tissue ruptures during ascent, allowing gas bubbles to enter the arterial circulation, and forming emboli that generally target the brain (Francis & Mitchell, 2002). While decompression sickness is to a large extent a function of the amount of inert gas taken up by tissues during a dive, arterial gas embolism is not necessarily associated with increased gas loading. The term "decompression illness", therefore, refers to any disease that occurs during decompression and includes decompression sickness, arterial gas embolism, and other gas-related forms of barotrauma of ascent.

Despite performing bouts of repeated long and deep dives interspersed with short surface intervals, marine mammals have not been reported to experience decompression sickness during natural dives

(Kooyman, 1973; Scholander, 1940), and it is believed that physiological adaptations (e.g. lung collapse, dive response) help reduce N_2 concentrations and risk of decompression sickness (Fahlman, Hooker, Olszowka, Bostrom, & Jones, 2008; Fahlman & Kayar, 2006; Fahlman, Schmidt, Jones, Bostrom, & Handrich, 2007). However, recent mass stranding events in deep diving whales are suggestive of decompression sickness-like symptoms (Jepson et al., 2003), indicating that dive behaviour may be important to reduce risk.

In human breath-hold divers, the issue of whether breath-hold dives cause decompression sickness has been debated since the 1960s (Hong, Rahn, Kang, Song, & Kang, 1963; Paulev, 1965a; Wong, 1999, 2000), with a growing number of cases reported with symptoms resembling those described in scuba divers (Schipke et al., 2006). In 1965, Lanphier stated that ''decompression sickness is virtually impossible for the skin diver because he cannot submerge deep enough or remain long enough to take up a troublesome amount of nitrogen''. Although this statement has long been accepted as true for single or occasional breath-hold dives, it does not account for dives that are made repeatedly, to great depth, and at very short intervals. Some discrepancies thus emerge from the different dive characteristics observed in different categories of breath-hold diver. Indeed, breath-hold dives can be characterized as either single deep dives or shallow and/or repeated dives. During shallow repeated dives, dives are repeated over several hours to 30–40 m, as in spear-fishing. Single deep dives refer to a single dive of greater than 50–70 m, where the diver descends with the help of fins and/or a weight and ascends with a gas-filled balloon. Elite breath-hold divers have reached depths greater than 200 m during single deep dive record attempts. We assumed that these two divergent dive behaviours may result in similar symptoms through different mechanisms. This review (1) describes and discusses the possible mechanisms and evidence for the occurrence of decompression illness in mammals and humans following breath-hold dives and (2) presents approaches for assessing the risks of this activity.

Decompression sickness and marine mammals

Nitrogen accumulation

Henry's Law states that the amount of a gas dissolved in a fluid will be proportional to the partial pressure of the gas in contact with the fluid. Therefore, pressure and time determine the amount of gas dissolved in the tissues and body fluids. Because N_2 is not metabolized in the body, it remains dissolved until the N_2 pressure in the lungs decreases (Doolette & Mitchell, 2001). As the diver ascends, the amount of N_2 that can be held in solution decreases and when tissue or blood P_{N2} is greater than the ambient N_2 partial pressure, the diver is said to be supersaturated and bubbles may form. It is believed that these bubbles cause the signs and symptoms of decompression sickness. The bubbles can be intra- or extra-vascular. The former may originate from pulmonary barotrauma or from the release of excess dissolved gas into the circulatory system. Extra-vascular bubbles are thought to originate from the release of excess dissolved gas and the severity of decompression sickness symptoms is related to the inert gas load. If present in sufficient quantity, the bubbles can act as emboli causing ischaemic damage. They can injure the tissues in which they appear and act as foreign bodies that damage vascular endothelium, disrupt the blood–brain barrier, and initiate patho-physiological processes such as the complement cascade (Francis & Mitchell, 2002).

Can marine mammals avoid decompression sickness?

In 1940, Per Scholander published his seminal work on diving mammals. In one section of this opus, he noted that marine mammals appeared to have a very compliant rib cage and stiffened upper airways. He suggested that the increasing pressure with depth would compress the chest and push all the air into the upper airways. This would prevent gas exchange at depth and thereby reduce N_2 uptake during breath-hold dives. Using measured volumes of the upper and lower respiratory system in whales and seals (*Hyperoodon ampullatus*, *Cystophora cristata*, *Halichoerus grypus*, *Balaenoptera physalus*, *Phocaena communis*), alveolar collapse and termination of gas exchange were estimated to occur at depths ranging from 30 m to 210 m depending on the initial diving lung volume (Scholander, 1940). Biological tissues have limited scope to resist pressure differences, and negative trans-thoracic pressures exceeding 100 kPa will damage tissue (Brown & Butler, 2000). It was therefore concluded that the chest must compress and eventually collapse to prevent damage. Both direct and indirect evidence of chest collapse has been reported in the diving dolphin (Ridgway & Howard, 1979; Ridgway, Scronce, & Kanwisher, 1969). However, compression of the rib cage does not prove that the alveoli have collapsed and that gas exchange has stopped. Depth of collapse has been estimated by assuming a rigid trachea and highly compliant lung (Denison & Kooyman, 1973; Stephenson, 2005). This assumption is questionable, as the trachea in Weddell and elephant seals showed

significant compression at a depth of only 54 m (Ridgway, 1968). Direct measurement of inert gas exchange during diving suggested that alveolar collapse, and a concomitant cessation of gas exchange, occurs at 30 m in the Weddell seal (Falke et al., 1985) and 70 m in the dolphin (Ridgway & Howard, 1979). These depths are relatively shallow and could possibly prevent significant N_2 uptake and minimize risk of decompression sickness during deep diving (Falke et al., 1985; Ridgway & Howard, 1979; Scholander, 1940). In addition, recent modelling attempts have provided an alternative explanation for the uptake and removal of N_2 in the dolphin and Weddell seal (Bostrom, Fahlman, & Jones, 2008; Fahlman, Olszowka, Bostrom, & Jones, 2006; Fahlman et al., 2008): alveolar compression results in an increasing pulmonary shunt.

While the studies on dolphins and Weddell seals implicitly assumed that termination of gas exchange occurred instantaneously, other researchers have shown experimental and theoretical evidence that compression results in a shunt that increases with pressure (Bostrom et al., 2008; Scholander, 1940). In one study, the measurement of pulmonary shunt in California sea lions and harbour seals at pressures equivalent to depths of 70 m and 90 m, respectively, indicated a reduction in gas exchange that correlated with depth (Kooyman & Sinnett, 1982). At a depth of 90 m, the shunt exceeded 70% in the harbour seal and complete alveolar collapse and termination of gas exchange was estimated to occur between 160 m and 170 m (Kooyman & Sinnett, 1982). The species used for this study (California sea lion and harbour seal) were chosen as they show the most divergent airway structure from those measured in pinnipeds (Denison & Kooyman, 1973). Despite this, the compression shunts at pressures below 70 m were not remarkably different from each other (between dolphins and California sea lions, for example) (Kooyman & Sinnett, 1982). A recent mathematical model, describing compression of the upper and lower respiratory tract (Bostrom et al., 2008), showed that graded alveolar collapse and its effect on gas exchange produced results that agreed with the observed data in the dolphin (Ridgway & Howard, 1979), California sea lion (Kooyman & Sinnett, 1982), Weddell seal (Bostrom et al., 2008; Scholander, 1940), and harbour seal (Kooyman & Sinnett, 1982). In addition, the model also predicted compression of the upper respiratory tract that agreed well with the measured data in the Weddell seal. Furthermore, it was predicted that complete collapse would not occur above depths of 150 m (Bostrom et al., 2008; Scholander, 1940).

The antagonistic effect of compression on diffusion rate is an alternative explanation that explains the data in the Weddell seal and bottlenose dolphin (Bostrom et al., 2008; Scholander, 1940). Compression of the respiratory system will on the one hand tend to increase the alveolar-capillary partial pressure gradient, and thereby increase the diffusion rate. On the other hand, further compression will reduce the gas exchange surface area and increase the diffusion distance. Thus compression will initially promote diffusion and inert gas uptake, but as the depth of the dive increases the developing pulmonary shunt will reduce uptake (Bostrom et al., 2008; Fahlman et al., 2008; Scholander, 1940).

If gas exchange does not cease at shallow depths, is it possible that some species of mammals live with elevated N_2 concentrations that could cause bubble formation with alterations in dive behaviour? If so, how do they avoid decompression sickness when foraging for food? In addition, could anthropogenic causes, such as climate change or overfishing, impose behavioural changes that increase risk?

Other mechanisms

Although little is known about how the respiratory system in marine mammals compresses during breath-hold diving and how this affects gas exchange, termination of gas exchange is routinely cited in animal physiology textbooks as the primary adaptation that protects marine mammals from elevated N_2 concentrations and decompression sickness. Indeed, Scholander (1940) suggested that cessation of gas exchange could protect against decompression sickness but indicated that the diving lung volume would determine the depth at which this occurred; there is now theoretical (Bostrom et al., 2008; Fahlman et al., 2008) and experimental evidence (Kooyman & Sinnett, 1982) to support this idea. It is less well known that Scholander also reported two possible cases of decompression sickness in a fin whale and hooded seal respectively during a single dive (Scholander, 1940). If marine mammals adapted for prolonged deep diving can experience decompression sickness during a single dive, decompression sickness may also occur in humans. The structural properties of the human respiratory system, with a more compliant trachea, would delay lung collapse and make humans more susceptible to decompression sickness (Figure 1).

While marine mammals perform single deep and long dives without apparent decompression sickness symptoms, more remarkable still are the extensive foraging bouts of many diving mammals and birds. Such dive behaviour should result in tissue accumulation of N_2, increasing the risk of decompression sickness. Scholander (1940) concluded that ''by repeated dives, conditions as regards diving disease would certainly tend to be worse on account of an accumulation of invaded N_2. There is every reason to
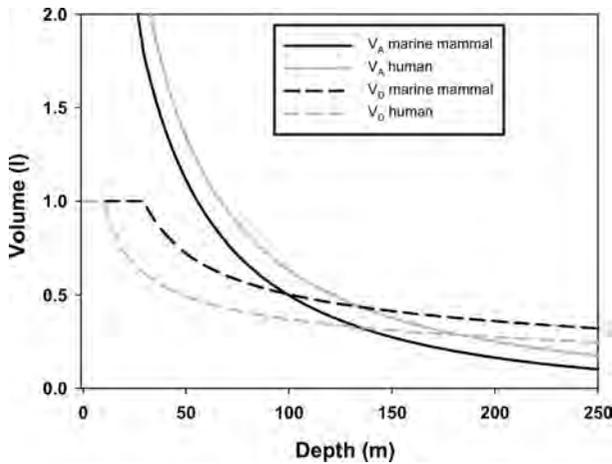
Figure 1. Depth (m) and estimated alveolar (V_A) and dead space (V_D) volumes in a human or marine mammal diving with an initial $V_A = 10$ litres and $V_D = 1$ litre. Alveolar collapse is assumed to occur when total lung volume (V_L) $<1\%$ of maximum, i.e. in this case 0.11 litre. In the marine mammal, alveolar collapse would occur at 252 m and in human 358 m. Model reproduced with permission from Bostrom et al. (2008).

believe that this risk exists unless there is sufficient ventilation between dives" (p. 112). In fact, detailed investigation of sperm whale carcasses revealed evidence of osteonecrosis (Moore & Early, 2004). Dysbaric osteonecrosis is a pathology found in commercial divers who experience repeated decompressions and asymptomatic bubbles. The bubbles reduce blood flow to the bones, eventually resulting in necrotic lesions. In addition, necropsy results in stranded beaked whales and dolphins (Jepson et al., 2003) were suggestive of decompression sickness-like symptoms. These mass stranding events correlated with naval exercises using high-frequency sonar. It was suggested that the sonar activity may have led to disturbances in the natural dive behaviour, resulting in dive profiles that caused bubble formation.

Few alternative explanations have been proposed to explain how marine mammals avoid elevated inert gas uptake during breath-hold diving. Kooyman (1973) summarized most of these in a review on the respiratory adaptations in marine mammals. Possible physiological adaptations include (1) increased tissue and blood N_2 solubility, (2) a special N_2 absorbing tissue, and (3) changes in cardiac output and varying blood flow distribution, all of which may help prevent excessive inert gas uptake in addition to pulmonary shunt and alveolar collapse.

We are unaware of any studies that have measured the solubility of N_2 in tissues of diving mammals or birds, but the solubility in blood is similar in the seal and human (Kooyman, 1973). The foam normally found in the upper respiratory tract of marine mammals has been suggested to be a potential N_2-absorbing agent, but there is no experimental support for this supposition (Kooyman, 1973).

Animal research has shown that inert gas removal can be accelerated by intestinal microbes that metabolize a small portion of the inert gas burden (Fahlman & Kayar, 2003; Kayar, Aukhert, Axley, Homer, & Harabin, 1997). For example, a 5% reduction in the inert gas burden reduced the incidence of decompression sickness by as much as 50% (Fahlman, Tikuisis, Himm, Weathersby, & Kayar, 2001). Nitrogen-fixing microbes are found in the gut of animals and if present in diving mammals they would provide an additional avenue for inert gas removal. Interestingly, a similar suggestion was made by Scholander (1940), who observed that N_2 from blood *in vitro* disappeared in the presence of O_2. It was suggested that this is caused by N_2 fixation by a microbe called organism-X (Scholander, 1940), but this hypothesis was dismissed by others.

Diving mammals perform extended dive bouts consisting of repeated dives interspersed by surface intervals that commonly are shorter than each dive. The tissue N_2 tension (P_{N2}) of each tissue throughout a dive bout depends on the specific time of tissue gas uptake, often measured as time to 50% tissue completion ($\tau_{tiss_{1/2}}$). As this variable is governed by local blood flow, $\tau_{tiss_{1/2}}$ is different between tissues. Most diving mammals have large amounts of subcutaneous fat that reduces heat loss and acts as an energy reservoir during extended periods without food. The five-fold higher N_2 solubility in fat compared with lean tissue, combined with the reduction in cardiac output and re-distribution of blood flow that represents the dive response, results in a long $\tau_{tiss_{1/2}}$ for adipose tissue (Fahlman et al., 2006). These properties have led researchers to suggest that adipose tissues could act as an N_2 absorbent and reduce bubble formation during deep and short dives (Behnke, Thomson, & Shaw, 1935; Fahlman et al., 2007). During the first few dives of a dive bout, tissues with a short $\tau_{tiss_{1/2}}$ (central nervous system and muscle) experience high P_{N2} during the dive, but much of the accumulated N_2 is removed during the ascent and only low levels remain as the animal surfaces (Fahlman et al., 2007). The long $\tau_{tiss_{1/2}}$ of subcutaneous fat, on the other hand, leads to a slow but continuous increase in P_{N2} (Behnke et al., 1935; Fahlman et al., 2007). During the ascent, the pre-surface tachycardia reported in both diving mammals and birds (Andrews et al., 1997; Elsner, 1965; Froget et al., 2004) and the increased perfusion to adipose tissue allow a portion of the N_2 in the fast tissues to be taken up by the fat without any dramatic increase in P_{N2}. This could help reduce overall mixed venous P_{N2} and thereby decrease the likelihood of bubble formation (Fahlman et al., 2007; Kooyman et al., 1972). Thus, fat P_{N2} is negligible at the beginning of the bout but slowly increases even during most of the ascent. This continuous increase

in P_{N2} could eventually result in elevated adipose P_{N2} that could force the animal to undertake a long surface interval (Fahlman et al., 2007). Consequently, adipose tissue could help buffer P_{N2} at the beginning of a dive bout but be a liability after a long bout (Fahlman et al., 2007).

The dive response has been suggested as a useful physiological mechanism to reduce inert gas uptake (Fahlman et al., 2007; Ponganis, Kooyman, van Dam, & LeMaho, 1999; Scholander, 1940). This makes intuitive sense and Fahlman et al. (2006) showed that mixed venous P_{N2} could be reduced by as much as 45% when an animal exhibited diving bradycardia during the descent and bottom phase, with a reduced ascent rate and a pre-surface tachycardia. However, Fahlman et al. (2006) only analysed a 1-h dive bout consisting of 23 dives. A more recent theoretical study, estimating tissue and blood P_{N2} levels in deep-diving king penguins during a foraging trip, showed that an increase in blood flow during diving led to increased P_{N2} at the end of an extended dive bout in some tissues, but a decrease in P_{N2} in other tissues (Fahlman et al., 2007). For example, diving bradycardia caused a substantial reduction in brain and central circulation P_{N2}, but an increase in muscle and fat P_{N2}. These surprising results suggest that the diving-related reduction in blood flow does not always reduce N_2 concentrations during repeated diving. Interestingly, each tissue had a specific blood flow rate that resulted in maximum end-bout P_{N2}. A $\tau_{tiss_{1/2}}$ was computed for each tissue and it was shown that the $\tau_{tiss_{1/2}}$ resulting in maximum end-bout P_{N2} was the same for the different tissues and similar to the average dive duration of 1–1.5 min (Fahlman et al., 2007). It will be interesting to determine whether the $\tau_{tiss_{1/2}}$ that results in maximum end-bout P_{N2} corresponds to average dive duration in different species; if so, this could be an inherent property of inert gas flux in diving animals. If that is the case, one would predict that diving animals avoid tissue perfusion rates that result in tissue $\tau_{tiss_{1/2}}$ close to the average dive duration. However, as the circulatory system is also responsible for removing CO_2 and supplying O_2, blood flow distribution among the tissues is a trade-off between the need to exchange metabolic gases and the need to reduce the risks of decompression sickness. Thus, the blood P_{N2} at the end of a dive or an extended bout is a complex function of the need to supply O_2 to, and remove CO_2 from, central organs while simultaneously reducing uptake of N_2. The question is to what extent changes in blood flow are used as a means to reduce extreme P_{N2} without ischaemic injury.

Diving mammals and birds may also use behavioural means coupled with physiology to reduce the inert gas burden. It has been shown in some species

that when approaching the surface, tachycardia (Andrews et al., 1997; Froget et al., 2004) and a reduction in ascent rate (Banish & Gilmartin, 1992; Hooker & Baird, 1999; Sato, Charrassin, Bost, & Naito, 2004; Tyack, Johnson, Soto, Sturlese, & Madsen, 2006) may reduce the inert gas burden by up to 45% before surfacing. (Fahlman et al., 2006). The short and shallow surface dives that are observed between deep dives or at the end of extended dive bouts could be a behavioural phenomenon that helps reduce supersaturation and bubble formation while gas exchange and inert gas removal continue (king penguins) (Fahlman et al., 2007). It must be pointed out that to be protective, these decompression dives have to be to a depth that allows removal of N_2 and therefore not deeper than the current tissue and mixed venous P_{N2}. In king penguins, these decompression dives are deepest at the end of a dive bout and subsequently become more shallow (Fahlman et al., 2007) (Figure 2). Finally, it has long been suggested that some seals exhale before diving to reduce the depth at which the lungs collapse and gas exchange ceases. It was shown that fur seals exhale during the ascent, possibly to sustain the pulmonary shunt and prevent gas exchange and shallow water blackout (Hooker, Miller, Johnson, Cox, & Boyd, 2005), and Bostrom et al. (2008) used a mathematical model to show that this is an efficient behavioural strategy to reduce the depth at which the lungs collapse.

However, if diving animals use behavioural and/or physiological means to reduce the inert gas burden, how do they know that they are at risk? Can they sense low levels of bubbles and does this affect physiology and dive behaviour? To better understand how diving mammals avoid elevated N_2 concentra-
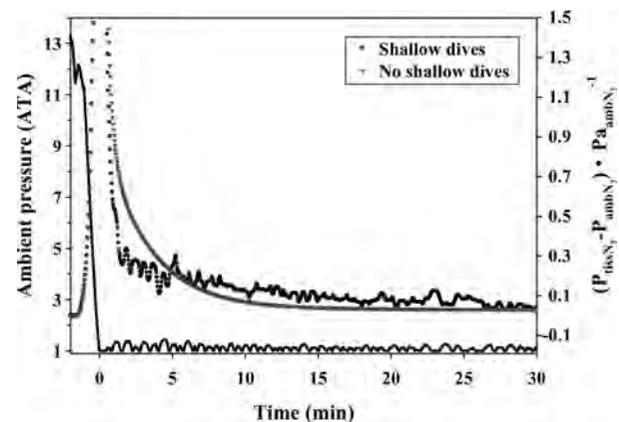

Figure 2. Ambient pressure (P_{amb}, atmospheres absolute, ATA) and estimated mixed venous supersaturation ($\{P_{N2venous} - P_{N2ambient}\} \cdot P_{N2ambient}^{-1}$) for a king penguin performing short and shallow dives (solid dots) or resting at the surface (grey dots) during an inter-bout interval. Modified from (Fahlman et al., 2007).

tions, research efforts must improve our understanding of gas exchange during breath-hold diving. This is not only an interesting physiological problem but also an important question in clinical pulmonary medicine, because recruiting a collapsed human lung may represent a severe clinical problem. Thus, clinical medicine would greatly benefit from a better understanding of how marine mammals are able to repeatedly collapse and recruit their alveoli during each deep dive. In addition, if marine mammals live with elevated blood and tissue N_2 levels, do they have any specialized adaptations that reduce decompression sickness risk? Such information may improve our knowledge of risk of decompression sickness in humans.

Human breath-hold diving and decompression sickness

Modelling decompression sickness risks and the effect of dive behaviour

Human breath-hold divers do not breathe pressurized gas and the only inert gas added is the N_2 that remains in the lungs from the last breath before immersion. The decompression sickness reported in pearl divers and Ama has been attributed to this progressive N_2 accumulation (supersaturation) (Bagnis, 1968; Cross, 1965). Paulev (1965b) estimated P_{N2} in his tissues after shallow repeated dives, and his calculation suggested that the short surface intervals did not allow tissue N_2 to be eliminated. Therefore, the tissue P_{N2} was equivalent to that resulting from a continuous dive.

Further studies by Lanphier (1965) indicated that the ratio of surface time to dive duration (S/D) and the rate of ascent were important factors in the development of decompression sickness from breath-hold diving. Lanphier calculated that an S/D ratio of 1 gave a depth exposure equivalent to about 50% of the actual depth of the dive. Thus, a dive to 30 m with a 90-s dive and a 90-s surface interval would be equivalent to a continuous dive to about 15 m. If the ascent rate was rapid, the equivalent depth was about 65% of the actual depth (22 m). These relationships can explain why a breath-hold diver performing many shallow repeated dives in the range 30–40 m may eventually develop symptoms of decompression sickness. Divers who perform breath-hold dives for 3–5 h will greatly exceed the no-decompression times for their equivalent depths and would be expected to develop severe neurological decompression sickness. By increasing the S/D ratio to 2 (e.g. 90-s dive, 180-s surface interval), the equivalent depth would be about 10 m during a breath-hold dive to 30 m, thus reducing the potential risk of decompression sickness. Repeated dives to

depths shallower than 20 m for several hours with short recovery periods can lead to an accumulation of dissolved N_2 in fat tissues equal to the amounts found for scuba divers (Lanphier, 1965; Paulev, 1965a).

Olszowka and Rahn (1987) estimated the changes in gas stores during shallow repeated dives. A mathematical model was used that estimated tissue and blood P_{N2} for the observed diving pattern of the Japanese Funado. The Funado perform approximately 30 dives to a depth of 20 m during a regular work shift of 1 h (descent and ascent rate of $1.33 \text{ m} \cdot \text{s}^{-1}$; bottom time of 30 s; dive duration of 1 min; surface interval of 1 min). Model output suggested that the brain–heart–viscera component quickly reached a steady-state value after five dives (P_{N2}: 1.31 atmospheres absolute), the muscle component after about 40 min, while the fat component showed a continuous and linear increase throughout the 60-min period. Recently, Fahlman and Bostrom (2006) predicted P_{N2} in mixed venous blood and four tissues (central circulation with heart, liver, and kidney; muscle, brain, and fat) during repeated breath-hold dives in humans (Figure 3). After 30 repeated dives to 30 m with a dive duration of 150 s and a surface interval ranging between 90 s and 300 s, the maximum estimated venous P_{N2} ranged between 1.44 atmospheres absolute and 1.69 atmospheres absolute. Venous P_{N2} during the first
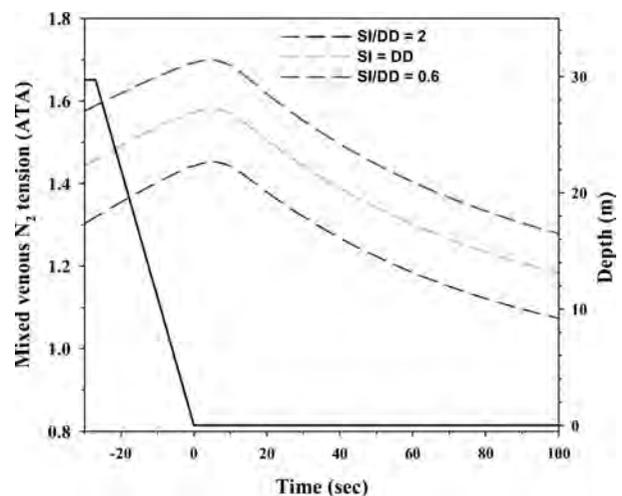

Figure 3. Estimated mixed venous N_2 tension (P_{N2}, atmospheres absolute, ATA) at the end of 30 repeated dives to 30 m (4 ATA) and with a dive duration (DD) of 150 s and varying surface interval durations (SI, 90 s, 150 s, and 300 s) using the model of Fahlman et al. (2006) for a 70-kg human with a cardiac output (Q) of 3 litres $\cdot \text{min}^{-1}$ during diving. Central circulation received 50% of Q_{tot}, muscle 33%, brain 15%, and fat 2%. The percentage of body mass for each compartment was 74% for muscle, 3% heart, 2% for brain, 15% for fat, and 6% for blood. The effect of lung compression on gas exchange (Bostrom et al., 2008) was not accounted for and no diffusion limitation was assumed during the dive. Therefore, these values are overestimated but represent the worst case.

and sixth dives reached 88% and 97%, respectively, of the maximum estimated venous P_{N2} during the entire series (data not shown). Fahlman and Bostrom (2006) suggested that a surface interval of at least twice the dive duration could help to reduce excessive P_{N2}.

Thorsen and colleagues (Thorsen, Zubieta-Calleja, & Paulev, 2007) tested the diving tables based on the empirical models of Haldane, the US Navy, and Buhlmann to prevent decompression sickness in breath-hold divers. These calculations showed that neither deep dives nor long total diving times are necessary to exceed the maximum-values of the two classical diving tables. For a given ambient pressure, a maximum-value is defined as being the maximum pressure reduction that a tissue can support without presenting decompression sickness symptoms. The maximum-values from the US Navy are exceeded by diving 50 times to 24 m with a total dive duration of 2.5 min and a surface interval of 255 s. Although these calculations indicate that N_2 accumulation during repeated breath-hold dives may eventually put the diver at risk, they need to be experimentally validated in humans, as they are currently speculative.

History of decompression sickness and detection of bubbles

Until 1960, it was generally assumed that tissue and blood P_{N2} concentrations in breath-hold divers could not reach levels that resulted in decompression sickness. However, independent reports around the world suggest that repeated breath-hold dives may result in symptoms that resemble decompression sickness and an on-line search resulted in a total of 141 cases of decompression sickness in more than 447 subjects (Tables I and II).

In 1965, Schaeffer was the first to describe non-pathogenic (silent) bubbles during submarine evacuation training where the divers repeated dives to 30 m with a short surface interval between dives. Paulev (1965a) reported decompression sickness-like symptoms after repeated breath-hold dives to 20 m, and after recompression treatment the symptoms resolved. Similar symptoms were also reported by Norwegian marines who had initially been compressed to 20 m for 8 min and then performed repeated breath-hold dives (Haavelsrud, 1963, 1964).

Several incidents of decompression sickness have been reported after shallow repeated dives, notably in people that use breath-hold diving as a means to hunt for fish or to collect pearls. For example, reports of accidents in pearl divers from the Tuamotu Islands presented the classic signs of decompression sickness. The reports include the ''Taravana''

syndrome, first described in pearl divers by Cross in 1962 and Bagnis in 1968 as a diving syndrome seen in working Tuamotu Island natives diving in the Takatopo Lagoon. ''Taravana'' has been translated as ''to fall crazily'' and is assumed to correspond to decompression sickness in these divers.

Schipke et al. (2006) reported some 90 cases in which decompression sickness occurred after repetitive breath-hold dives. The true number of cases of decompression sickness is probably considerably higher, as breath-hold divers may be reluctant to report symptoms and only consult medical advice when complications persist.

Bubbles are considered to be one of the key factors in the aetiology of decompression sickness. The amount of bubbles detectable in the venous system draining the tissue can be an indicator of the total amount of released gas (Nishi, Brubakk, & Eftedal, 2002). The presence of bubbles is usually not, in itself, sufficient to cause overt decompression sickness. To our knowledge, only two ''older'' studies described the detection of venous gas emboli in breath-hold divers; the description was incomplete in Ama divers following shallow repeated dives (Nashimoto, 1976; Spencer & Okino, 1972). Spencer's study (an abstract text) described detection of bubbles, without symptoms, after shallow repeated dives, but the number of subjects, their anthropometric characteristics, and their exact diving pattern were not reported. Recently, Huggins and Stepanek (2006) reported a grade I Doppler score (Spencer, 1976) after four dives where the descent and ascent were assisted using a scooter (depth range of 30–70 m; surface intervals of 15–20 min; dive durations of 2–3 min). Bubbles are classified on a scale from 0 to IV based on the number of bubble signals per cardiac cycle and the number of cardiac cycles containing bubbles (Spencer, 1976). Grade 0 reflects a complete lack of bubble signals and grade IV the maximum detectable signal overriding the amplitude of the normal cardiac signals. Since this report, several studies have failed to detect venous bubbles after shallow repeated dives when the diver swims (Boussuges et al., 1998; Radermacher et al., 1992) and these divergent results may highlight the difficulty in using Doppler scores to assess risk of decompression sickness.

Diving accidents and decompression sickness with typical diving patterns in human breath-hold divers

Accidents after single deep dives are less well documented than those after shallow repeated dives. Very few studies have reported accidents with decompression sickness symptoms. The diving profile of single deep dive is generally characterized by a rapid descent and ascent. The average ascent rate is

Table I. Diving patterns and decompression sickness-like symptoms in breath-hold divers.

Study	Participants (n)	Depth (m)	Diving duration (min)	Surface intervals (min)	Diving session (h)	Number of dives per hour	Cases of decompression illness	Symptoms
Domard (1957)	43	20–50	1.8–2.5	2–6	6	10	13	Vertigo, nausea, paralysis
Cross (1962)	4	40	2	4	8	13	4	Vertigo, nausea
Cross (1965)	235	30	1.8–2.5	3–4	6	10	47	Vertigo, nausea, mental anguish, partial or complete paralysis, temporary unconsciousness, mental affection, death
Paulev (1965b)	1	20	3	2	5	40	1	Dizziness, vertigo, emesis, visual disturbances, paresis in the right arm and severe thoracic pain
Spencer & Okino (1972)	1	15	1	1	1	30	0	But bubbles detected
Héran (1990)	1	30	2	2	6	15	1	Paraesthesia and weakness
Héran (1991)	6	40–45	2	4	5–8	10	4	Lethargy, insomnia, sensory deficits, skin pain, paralysis, asthenia, "heavy neck"
Radermacher et al. (1992)	9	3–6	3	7	1		0	No DCS, PN_2 low
Fanton et al. (1994)	1	40	2	2	3	13	1	Unconsciousness, coma
Oblare & Pascfual (1995)	1	25–35	1.5	3	6	10	1	Neurological CT disorders
Mohri et al. (1995)	8	7–17	0.5–1	2	8	10	0	No DCS symptoms
Boussuges et al. (1997)	10	24–40	3–4	2.5	2–6	12	0	No bubbles or signs detected (grade 0 Kisman-Masurel)
Merle et al. (1997)	1	8	1	1	1	20	1	Visual impairment attributed to DCS
Kohshi et al. (1998)	2	15–25	1	1–3	4.5–5	20	2	Cerebral infarctions, hemiparesis, sensory deficit, loss of consciousness
Kohshi et al. (1998)	15	25	1	1	4	20	8	Multiple cerebral infarctions
Tochimoto et al. (1998)	44						9	Chiyamai: panic-like disorder, reversible hemiplegia, and dysarthria
Batle (1999)	35	40–63	2	2	3–8	15–20	25	Neurological symptoms
Mango et al. (1999)	4	25–30	2	2	2–4	10	4	Hemiplegia, ataxia, dysarthria, diplopia, colour blindness
Wong (1999)	2	27–29	2–3	2	6	8	2	Dizziness, cerebellar signs, vertigo, nausea, blurred vision
Kohshi et al. (2000)	2	15–25	1–1.5	1	6	20	2	Cerebellar infarcts (hemianopsia, hemiparesis, sensory deficit)
Kohshi et al. (2001)	16	8–30	2	1	5	26	9	Hemiparesis, dizziness, euphoria, nausea, sensory deficits, hemianopsia, loss of consciousness
Volpe (2001)	4	20	2	0.8	2	25	4	Visual problems, hemiplegia, dysarthria, aphasia
Harms et al. (2006)	2	37 or 70					2	Hemiparesis with Broca's aphasia or central vestibular syndrome
Gempp & Blatteau (2006)	1	10–18	2	5–6	2	10–12	1	Dizziness, visual disturbance, tightness of the chest accompanied by dyspnoea, flushed face

Note: DCS = decompression sickness.

above $1.5 \text{ m} \cdot \text{s}^{-1}$, which increases the risk of de-compression sickness (Lemaître, 2007). In the study by Mango and associates (Mango, Lundgren, & Ferrigno, 1999), a diver performed 10 dives to between 30 and 70 m on two consecutive days with 90 min surface interval time between the final two dives. He developed decompression sickness-like symptoms, suggesting that it was an acute effect of the breath-holding. It is difficult to explain this type of accident, which occurs after a single, very deep dive as compared with shallow repeated dives. During an attempt to break a record, another breath-hold diver experienced decompression sick-ness-like symptoms (Lemaître, 2007). After a single deep dive to 209 m with dive duration of 3 min 28 s and an ascent and descent rate of more than $1 \text{ m} \cdot \text{s}^{-1}$, the diver felt extremely tired and was treated in a hyperbaric chamber until he recovered completely.

Diving accidents have also been reported among pearl divers in the Tuamotu (Cross, 1962, 1965), Korean and Japanese Ama (Cross, 1962, 1965; Kohshi, Katoh, Abe, & Okudera, 2000, 2001; Kohshi, Kinoshita, Abe, & Okudera, 1998; Kohshi et al., 2005), and spear-fishing (Boussuges, Abdellaoui, Gardette, & Sainty, 1997) after shallow repeated dives (Table I). In the present review, the average diving pattern of the breath-hold divers who experienced decompression sickness was character-ized by dives to 31 m for 2 min, separated by 2.5 min surface intervals over 5 h with 16 dives per hour (Table II). This average diving pattern is similar to that of other studies (Holm et al., 1998; Kita, 1965; Mohri et al., 1995; Nukada, 1965; Park et al., 1983; Rahn, 1965). Such a pattern may lead to decompression sickness and is especially likely to occur when Ama divers prolong their shallow repeated dives beyond 3 h (Kohshi et al., 2005). In addition, Cross (1965) reported that pearl divers in Mongareva, a nearby lagoon, used the same techni-ques but with longer surface intervals of 12–15 min and never developed "Taravana" syndrome. In the same way, the greater the depth, number, and frequency of repeated dives, and the shorter the surface intervals, the greater the risk of decompres-sion sickness (Table I).

Classification of clinical symptoms

In divers breathing compressed gas, the symptoms of decompression sickness are classified as type I or type II based on the severity of the disease (type I being "simple" and type II "serious"). No formal classification has been developed for symptoms observed after breath-hold diving. The symptoms encountered in the studies of the pearl divers on the Tuamotu Island were mainly neurological manifes-tations or type II symptoms (Table III). These included hemi-paresis, hemi-sensory disturbance, dysarthria, vertigo, nausea, dizziness, headache, visual changes, hearing loss, disturbances in con-sciousness and speech, euphoria, an inability to concentrate, and even sudden death after ascent (Tables I and III). Thus, a similar classification to that used for divers breathing compressed gas may also be useful for breath-hold divers, as it helps to orient treatment, prognosis, and overall manage-ment. We classify decompression sickness-like symp-toms for breath-hold divers into two types: accidents that are somewhat benign and quickly reversible (type Ia, where "a" stands for apnoea), characterized by dizziness, nausea, anguish, dizziness, etc., and serious accidents (type IIa) where the symptoms are neurological and persistent (Table IV).

A comparison of decompression sickness accidents shows that the prevalence of the neurological type is highest in both scuba divers (40.4%) and breath-hold divers (56%) (Francis & Mitchell, 2002) (Table III). In Ama, type IIa accidents with multiple cerebral infarctions have been observed after shallow repeated dives (Kohshi et al., 1998, 2000, 2005). These brain lesions localized in the basal ganglia, internal

Table II. Diving characteristics of breath-hold divers.

	Mean $\pm s$	Minimum	Maximum
Age	35.0 ± 8.8	21.0	48.0
Maximal depth (m)	31.4 ± 15.7	6.0	70.0
Maximal diving duration (min)	2.0 ± 0.6	1.0	3.0
Interval between dives (min)	2.5 ± 1.4	0.8	6.0
SD ratio	1.2 ± 0.6	0.4	3.0
Duration of dive session (h)	4.8 ± 2.1	1.0	8.0
Dives per hour	16.0 ± 7.7	8.0	40.0

Table III. Symptoms encountered in the pearl divers of Tuamotu Island.

Manifestation	Prevalence breath-holding (%)	Prevalence scuba diving (%)
Vertigo	43.3	7.2
Nausea	39.7	4.7
Paresis	36.2	8.1
Dizziness	19.1	7.2
Loss of consciousness	12.8	1.9
Visual disturbance	7.1	1.6
Fatigue	7.1	6.8
Cerebral infarction	3.5	/
Mentally affected	2.1	0.3
Death	1.4	
Total cases	447	11471
Total manifestations	141	3495

Table IV. Clinical manifestations of breath-holding "decompression" accidents.

Type	Scuba diving	Breath-hold diving
I	Limb pain (musculoskeletal symptoms) Skin bends (cutaneous symptoms) Lymphatic bends (lymph node swelling and pain)	*Simple*: nausea, vertigo, dizziness, anguish; symptoms disappear quickly
II	Neurological Pulmonary (chokes) Systemic (hypovolaemic shock) Inner ear/vestibular	*Neurological*: all sensory troubles, motor or psychological; serious and persistent

Based on the data of Golding and colleagues (Golding, Griffiths, & Hempleman, 1960).

capsule, and deep and subcortical white matter (Figure 4a, b) were so-called low-flow cerebral infarctions as a result of the low perfusion pressure in the terminal supply areas (Kohshi et al., 1998). Because no obvious abnormality was detected in the cerebral arteries corresponding to the infarcts (Kohshi et al., 2000), these features suggested circulatory disturbances due to air embolism. Cerebral impairment would probably depend on the location of the cerebral infarction. In addition, a high frequency of multiple asymptomatic brain lesions has been observed in scuba divers (Knauth et al., 1997; Reul, Weis, Jung, Willmes, & Thron, 1995) and in Ama divers (Kohshi et al., 1998, 2000), suggesting a long-term effect of breath-hold diving. However, the long-term consequences of shallow repeated dives on the central nervous system are unknown. Although
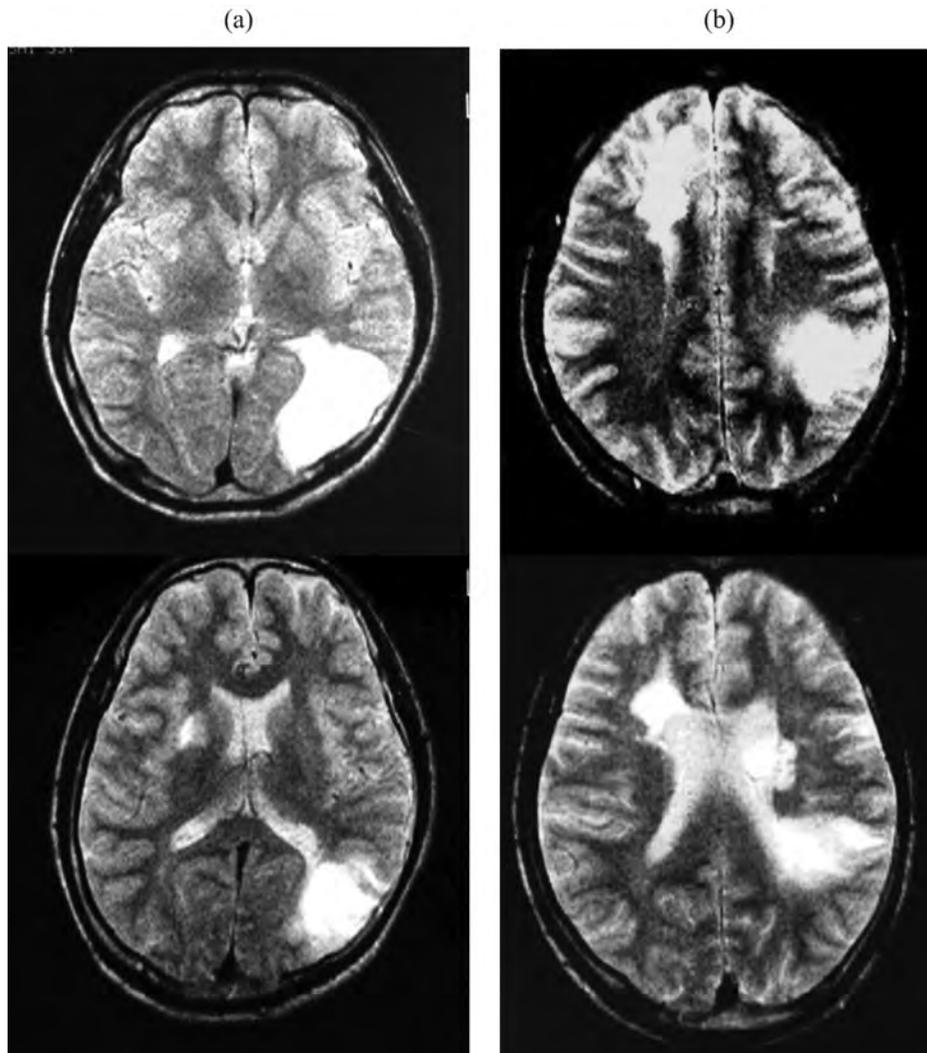

Figure 4. Magnetic resonance imaging of the brain of two cases (a) and b). (a) T2-weighted images [repetition time/echo time (TR/TE): 2000 ms/112 ms] obtained 4 days after the accident show two increased signal intensities in the left occipital lobe and the right basal ganglia. (b) T2-weighted images (TR/TE: 2000/112) obtained 3 days after the accident show three increased signal intensities in the left parietal lobe and basal ganglia and in the right frontal lobe (reproduced with permission from Kohshi et al., 2000). (Image made with a sequence with long TR and TE to show contrast in tissues with varying T2 relaxation times; water gives a strong signal.)

Kohshi et al. noted brain abnormalities in symptomatic breath-hold divers, Potkin and colleagues (Potkin, Cheng, & Siegel, 2007) studied five divers with a history of over 1000 dives to depths of at least 30 m over a period of at least 5 years. No diver had any neurological complaints, but single proton emission computed tomographic brain scans (PET scans) were abnormal in all five divers, revealing large focal and/or diffuse areas of hypoperfusion and hyperperfusion in the frontal and temporal lobes and cerebellar hemispheres. Thus, shallow repeated dives may be associated with asymptomatic brain function abnormalities. Although the occurrence of hypoxic episodes (loss of consciousness or loss of motor control) was not screened in this study, it is interesting that these cerebral areas were chosen: they are most sensitive to hypoxia, suggesting a possible additional role of hypoxia in cerebral after-effects (Kohshi et al., 2001). If symptoms develop following a dive, a diagnosis of decompression sickness must be considered. In scuba diving, decompression sickness that presents very soon after surfacing is likely to be due to arterial gas embolism, especially if the inert gas load is negligible and there are signs or symptoms of pulmonary barotraumas (Melamed, Shupak, & Bitterman, 1992).

Because increased cerebral perfusion is observed during breath-holding (Przybylowski et al., 2003), the inert gas supersaturation in the brain may be limited, thus preventing autochthonous (i.e. spontaneous) bubbling (Elliott & Moon, 1993). Even if bubbles form in the brain after shallow repeated dives, the site may be in the smallest veins. However, Kohshi et al. (2005) suggested that the brain lesions found in Ama are not caused by a disturbed venous circulation. Several possible causes have been identified for decompression sickness in breath-hold divers. Arterial blood gas tensions quite quickly reflect inspired partial pressures, whereas other tissues will equilibrate more slowly; it is therefore possible that bubbles form in arterial but not in venous blood, which would have a lower P_{N2}.

In some breath-hold divers, the symptoms were sudden, occurring as the divers left the water, and for others they appeared 1–2 h later, depending of the dive profile (Table I). The rapid onset of symptoms and their features point to neurological accidents caused by arterial bubbles. All of these cases showed total recovery, although some received treatment and others did not. Some Ama divers suffer from a panic-like disorder called "Chiyamai". Because the panic-like attacks only begin 2 months after the accident, Chiyamai might be a late sequela of a neurological decompression sickness (Tochimoto, Kitamura, Kurata, Nakamura, & Koshino, 1998). Although "Taravana" is likely to be decompression sickness, there are some features that do not fit and other cases

resemble hypoxia (unconsciousness, visual disturbance, muscular weakness, and uncoordinated movement), CO_2 retention (headache, dizziness, confusion), and even middle/inner ear barotrauma (vertigo, nausea, visual disturbance).

Pathogenesis

Effects of depth/blood shift/haemodynamic changes

During a single dive, only a finite amount of N_2 is available to dissolve in the tissues, and with limited dive duration only a fraction has time to be taken up. Olszowka and Rahn (1987) estimated that an extra 700 ml of N_2 would accumulate in the tissues after a single 220-s dive to 90 m, while Fahlman and Bostrom (2006) estimated that mixed venous P_{N2} could reach as high as 3.0 atmospheres absolute, 310% higher than the surface equilibrium value (0.74 atmospheres absolute). The present record in No-Limit is 214 m in more than 4 min. For these extreme depths, both the maximum depth and descent and ascent rates determine the amount of N_2 taken up and removed during the dive and therefore the risk of decompression sickness. During a rapid ascent, the blood that has shifted into the thoracic cavity can reverse. However, this reversal is probably not as rapid as the pulmonary expansion, resulting in entrapment of bubbles and their passage into the arterial circulation (Vik, Jenssen, Eftedal, & Brubakk, 1993). Further research is needed to determine the qualitative and quantitative factors that render bubbles pathogenic in breath-hold divers.

In mammals, lung collapse is assumed to be a universal mechanism to reduce supersaturation and, therefore, the risk of decompression sickness during breath-hold diving (see above). Alveolar collapse might protect the lungs from excessive vascular stress and limit the risk of decompression sickness by reducing the gas exchange surface area and N_2 uptake to the lungs, as demonstrated in marine mammals (Bostrom et al., 2008; Fahlman et al., 2008; Kooyman & Sinnett, 1982). The squeeze that would result in pulmonary oedema formation was estimated to occur at 34 m (Craig, 1968). At higher pressures, redistribution of blood from peripheral to central compartments would reduce lung volume and prevent extreme intra-thoracic pressures in these marine mammals. Recently, Liner and Andersson (2008) reported that the great depths reached by elite breath-hold divers during an international breath-holding competition are associated with a risk of pulmonary oedema.

The thoracic blood shift is estimated to be about 1 litre at 27 m (Schaefer et al., 1968). However, such vascular engorgement could lead to capillary stress failure and oedema (West, 2000). In humans,

complete alveolar collapse is predicted to occur at about 235 m if the dive is started at total lung capacity (Fitz-Clarke, 2007). The vascular engorgement, which initially protects the lung at depth, could eventually cause a mechanical pulmonary shunt, thereby allowing venous bubbles to pass into the arterial circulation resulting in cerebral arterial gas embolism. The spleen is known to serve as a dynamic red blood cell reservoir in several mammalian species (Qvist et al., 1986). Erythrocyte release from the spleen during diving increases the gas storage capacity and transport function of circulating blood, which may facilitate breath-holding diving in humans (Hurford et al., 1990). If some erythrocytes are released into the circulation, it can reduce the blood fluidity and increase problems related to this blood redistribution, increasing the possible risks of mechanical shunt and cerebral arterial gas embolism. However, because only a few supplementary erythrocytes are released during diving (Schagatay, Andersson, Hallen, & Palsson, 2001), we think that their role in the pathogenesis of decompression sickness is limited.

Buccal pumping

Cases of haemoptysis caused by alveolar haemorrhage during breath-hold diving, as reflected by blood in the lungs, have been described (Boussuges et al., 1999; Fitz-Clarke, 2006; Kiyan, Aktas, & Toklu, 2001). The underlying mechanism (Kiyan et al., 2001) may be capillary stress failure as a consequence of the drop in intra-thoracic pressure, with rupture of pulmonary capillaries due to the wide difference between alveolar and pulmonary capillary pressures. This form of pulmonary barotrauma may lead to arterial gas embolism from vigorous hyperventilation, including forced inhalation manoeuvres (e.g. buccal pumping) or air trapping in the lungs during a dive (McCrory et al., 2004). Buccal pumping is a technique that allows the lungs to be ventilated without the use of the respiratory muscles (Dale & Rahn, 1955). Breath-hold divers perform buccal pumping before deep dives to over-inflate the lungs above total lung capacity and to increase breath-hold performance (Lindholm & Nyren, 2005; Overgaard, Friis, Pedersen, & Lykkeboe, 2006). Nevertheless, no study has established the relationship between buccal pumping and decompression sickness in breath-hold divers.

Trapped and venous bubbles and patent foramen ovale

The lungs usually filter out bubbles, which remain trapped in the pulmonary microvasculature until the gas has diffused out into the alveolar space and is then exhaled. Large or small bubbles are usually trapped in small pulmonary arteries or pulmonary capillaries (Francis & Mitchell, 2002). Some venous bubbles may avoid pulmonary filtration completely by passing through a right–left circulatory shunt such as a patent foramen ovale. This shunting may lead to cerebral arterial gas embolism and decompression sickness in scuba divers (Germonpre et al., 2005; Schwerzmann & Seiler, 2001). The clinical manifestations are similar to thromboembolic stroke syndrome, varying from focal neurological deficits with a rapid onset, producing hemiplegia, confusion, or convulsions, to collapse and death (Strauss & Borer, 2001). Patent foramen ovale is variously reported to be present in 15–30% of the normal population and may explain some cases of decompression sickness (Germonpre et al., 2005; Moon, Camporesi, & Kisslo, 1989). Thus, breath-hold divers with patent foramen ovale may be at increased risk of decompression sickness. But in some studies, Ama with decompression sickness did not have a patent foramen ovale (Kohshi et al., 2000, 2001), indicating that patent foramen ovale is only a supplementary risk factor.

Factors associated with decompression sickness

Many other factors have been suggested to either increase bubble formation or predispose tissues to bubble injury, but most are anecdotal and have not been adequately studied. Obesity, age, excessive physical exertion during the dive, pre-dive physical condition, dehydration, and cold are factors that may possibly predispose an individual diver to decompression sickness (Carturan et al., 1999, 2000, 2002). Older divers have been shown to generate more venous bubbles than their more youthful counterparts after equivalent dives (Eckenhoff, Olstad, & Carrod, 1990). Indeed, susceptibility to decompression sickness has been shown to increase with age and increasing body fat mass (Carturan et al., 1999, 2002). Since N_2 solubility in fat tissue is high, it may initially act to reduce overall tissue P_{N2} but may, after many repeated dives, increase the risk of decompression sickness (Fahlman et al., 2007). Broome and colleagues (Broome, Dutka, & McNamee, 1995) suggested that poor aerobic fitness, associated with obesity or overweight, increased bubble load. Animal studies have shown an increased risk of decompression sickness in dehydrated individuals, while the effect of temperature appears to be more complex (Fahlman & Dromsky, 2006). Indeed, it is physiologically plausible that dehydration could alter inert gas removal by reducing blood flow to poorly perfused tissues, or that it may decrease surface tension and thereby facilitate bubble formation. While there is a clear relationship

between body mass and susceptibility with P_{N2} load in a range of terrestrial animals (Berghage, David, & Dyson, 1979), no data exist that link morphological or physiological factors to risk of decompression sickness in breath-hold divers.

Conclusions

Decompression accidents occur in breath-hold diving humans and may in rare cases happen in marine mammals. The mechanisms implicated in these accidents indicate that repeated breath-hold dives with short surface intervals are factors that predispose to decompression sickness. During deep diving, the effect of pulmonary shunts and/or lung collapse may play a major role in reducing the incidence of decompression sickness. No study to date has investigated the decompression sickness risk in breath-hold diving humans. A better understanding of how marine mammals avoid excessive blood and tissue N_2 concentrations or prevent bubble formation could lead to novel methods to avoid decompression sickness in human breath-hold and scuba divers.

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