CHAPTER

7

Diving physiology in dolphins and human

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Abstract

During diving, the body is exposed to a number of environmental stressors that result in physiological responses. Many of these responses are common across both humans and dolphins. One of the best known is the dive response, historically referred to as the "master switch of life," a defense against asphyxia. This is enacted during submersion and consists of a reduction in heart rate and a redistribution of blood to high-priority organs. Increasing hydrostatic pressure during descent compresses all air-filled spaces, resulting in structural changes, and alters blood gas tensions and hemodynamics. In this chapter, we will briefly outline a number of physiological changes associated with diving and compare and contrast these responses between humans and dolphins.

Introduction to diving physiology

A dolphin perspective

Breath-hold diving exposes the mammalian body to multiple extreme environmental stresses. Some of the most challenging are the separation from ambient air and the increasing atmospheric pressure with depth. Submersion in water also affects heat transfer due to the greater thermal conductivity of water (i.e., heat transfer rates increase 24-fold, compared to air temperatures, see Chapter 3: Thermoregulation). For dolphins, diving is a "full-time" activity, and these challenging states constitute "normal" conditions. As such, their physiological adaptations to these challenges have evolved since their ancestral re-entry into the aquatic environment and have been optimized for a lifestyle where almost all of life's activities (feeding, communicating, traveling, breeding, and sleeping) are conducted while submerged in seawater. To paraphrase Fedak and Thompson's (1993) reflection: *dolphins are not surface-dwellers which occasionally leave the surface to forage, but rather are visitors to the surface which live at depth and only periodically return to the surface to breathe*. In this chapter, we hope to explain how this is more than just a semantic distinction.

A human perspective

For humans, breath-hold diving is a "part-time" activity (in other words, humans must breathe and do not live in the water indefinitely) for recreation, competitive sport, military strategy, and even sustenance. It can take a variety of forms, such as underwater rugby/hockey in a pool, spearfishing (performing repetitive shallow dives over many hours), and free-diving. The latter encompasses different disciplines ranging from long static (i.e., stationary) breath-holds, breath-holding while swimming horizontally in a pool (dynamic), and/or diving deep on a static dive line. Since the 1940s, records of performance have shown divers progressively diving deeper (Fitz-Clarke, 2018)—with current depth records using a monofin to achieve 114 and 130 m for female and males, respectively (Fig. 1A). Sustenance diving is typically characteristic of indigenous diving populations, such as the Ama in Japan and Haenyeo in

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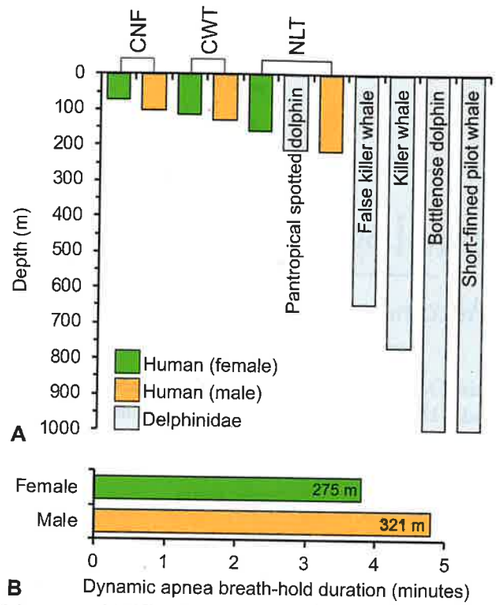

FIG. 1 Comparison of depth records for human and Delphinidae species presented in ascending order of depth (Panel A). Horizontal underwater breath-hold (i.e., dynamic apnea with monofin, DYN) swimming distance and duration records for humans (Panel B). CNF, constant weight without fins; CWT, constant weight with monofin; NLT, no limit discipline. Human depth data from AIDA (2022) and distance and duration data from the top male and female finisher at the 2022 CMAS World Freediving Indoor Championships. Delphinidae maximum dive depths are provided for the pantropical spotted dolphin (*Stenella attenuata*), false killer whale (*Pseudorca crassidens*), bottlenose dolphin (*Tursiops truncatus*) and short-finned pilot whale (*Globicephala macrorhynchus*) based on data from Ropert-Coudert et al. (2018), Fahlman et al. (2023).

Korea (Teruoka, 1932; Rahn and Yokoyama, 1965; Hong and Rahn, 1967; Vaneechoutte et al., 2011), and the Bajau in Indonesia (Abrahamsson and Schagatay, 2014; Ilardo et al., 2018). Despite societal acculturation, these populations still practice breath-hold diving as their primary means of harvesting food. This form of diving has also been performed in coastal countries, such as Croatia, where restrictions on fishing with nets and SCUBA (self-contained underwater breathing apparatus) equipment have established breath-hold diving as a primary fishing modality. Irrespective of the motivations to breath-hold dive, humans, as a species, are less adapted for diving compared to marine species. Indeed, our time in the sea is physiologically challenging, and many of our responses to the challenges of submersion, and our need for equipment, are geared toward simply surviving. Humans are most certainly surface-dwellers, which occasionally leave the surface to forage or to partake in recreational or professional endeavors.

Just as some motivations (i.e., food) for diving in dolphins and humans may overlap, the physical and anaerobic challenges associated with immersion always exist—for example, how is oxygen delivery to vital tissues such as the brain maintained? How do air-filled cavities cope with elevated and rapid hydrostatic pressure changes? How is thermal homeostasis maintained? In this chapter, we will briefly outline a number of physiological changes associated with diving and compare and contrast their responses in humans and dolphins. We will demonstrate how common physiological responses, which are shared among mammals to protect the body, have been accentuated in dolphins to allow them to exploit marine resources without experiencing pathologies often manifested in the human diver.

The dive response

Overview

Immersion and separation from ambient air is perhaps the most immediate limiting factor for a submerged mammal. Without access to air to breathe, aerobic metabolism continuously depletes blood oxygen (O_2) and raises carbon

dioxide (CO_2) levels, causing hypoxia (low O_2), hypercapnia (high CO_2), and tissue acidosis (higher acidity). With the cessation of breathing during submersion, all mammals share the same "dive response." This has been referred to as the master switch of life (Scholander, 1963), as it optimizes the use of O_2 and delays the onset of asphyxia. This may even constitute an ancestral response that is "hard-wired" to protect the body from hypoxia and asphyxia rather than an evolutionary adaptation for diving per se (Mottishaw et al., 1999). While this response is autonomic (i.e., involuntary) in humans, marine mammals (e.g., bottlenose dolphin (*Tursiops truncatus*), the harbor porpoise (*Phocoena phocoena*), and the California sea lion (*Zalophus californianus*)) appear to have the ability to regulate heart rate (and blood flow) depending on their planned dive (Elsner et al., 1966; Ridgway et al., 1975; Elmegaard et al., 2016; Kaczmarek et al., 2018; Fahlman et al., 2020b).

The dive response consists of a suite of cardiovascular changes, which allow the diving body to conserve the available O_2 for central organs such as the heart and brain, via a temporary "metabolic retreat." This involves a reduction of the heart rate below resting levels, called bradycardia, and selective distribution of the available O_2 in the blood to tissues that are unable to sustain any prolonged period of hypoxia (i.e., hypoxia-intolerant) or that do not have endogenous O_2 stores. This reduced metabolic state demonstrates some similarities to resting activities such as sleeping and even meditation, although, during diving, the body is not resting. Instead, the reduction in the rate of O_2 consumption, through reduced cardiac work and organ performance, functions to extend dive time and protect the body's tissues from hypoxia, hypercapnia, and blood acidosis. Together with the significant preferential redistribution of blood flow away from tissues which are nonessential to diving (such as the skin and adipose tissue), these responses maintain blood flow to more vital organs (such as the brain) and help reduce the utilization of available O_2, thus extending the dive duration. Redistribution of blood flow, through peripheral vasoconstriction, is a phenomenon demonstrated in both seals (Zapol et al., 1979) and humans (Brick, 1966). This redistribution of blood flow results in hypoxia in perfusion-poor tissues, which in turn result in a decrease in the respiratory rate of the cells and a reduction in the local rate of O_2 consumption (and CO_2 production) (Scholander, 1940; Wheaton and Chandel, 2011). This "master switch of life" is vital in protecting the body's most hypoxia-intolerant tissues and organs, sustaining consciousness, and conserving O_2 required to conduct longer breath-hold durations (see Chapter 5: Cardiovascular physiology in dolphins and other cetaceans).

Facial receptors and cardiovascular responses

In humans, submersion of the face (together with the inhibition of breathing) stimulates receptors around the nose and in the skin of the face (Schuitema and Holm, 1988), which sends afferent signals to the brain. These afferents initiate (1) a vagally mediated increase in parasympathetic nerve activity that reduces the heart rate (Olsen et al., 1962; Craig, 1963) and (2) sympathetically mediated vasoconstriction of peripheral vascular beds, which reduces blood flow to organs that can endure reduced O_2 availability, such as the kidney and skeletal muscles (Fagius and Sundlöf, 1986; Heusser et al., 2009; Kyhl et al., 2016). These combined effects provide the reduction in O_2 consumption during diving, prolonging breath-hold duration and aim to preserve consciousness.

The magnitude of cardiovascular depression in response to diving is variable, augmented by various extrinsic and intrinsic factors. For instance, in humans, facial immersion in cooler water temperatures accentuates the bradycardia (Hurwitz and Furedy, 1986; Schuitema and Holm, 1988; Schagatay and Holm, 1996). Heart rates of 20–30 beats per minute are recorded during dives while immersed in a hyperbaric chamber or during dry breath-holds in the laboratory (Ferrigno et al., 1997; Ferretti, 2001; Bain et al., 2018b), but heart rates as low as 10 beats per minute have been recorded in facial immersion experiments in water at 1°C (Arnold, 1985). Additionally, air temperature appears to interact with the effect of water temperature to influence the magnitude of the human dive response. Immersion experiments showed greater bradycardia with a greater difference between water and air temperature (Schagatay and Holm, 1996).

Depth (i.e., vertical) diving in humans has an additive effect on the magnitude of the dive response. Submersion causes an initial bradycardia, but continued descent further decreases heart rate (especially when descent becomes passive due to negative buoyancy), until minimum heart rate is reached at, or close to, the bottom of the dive (Lemaître et al., 2013). After reaching the deepest part of the dive, the diver must actively swim to ascend. While heart rate remains bradycardic compared to predive values, the increase in metabolic activity and exertion can bring about small increases in heart rate (Lemaître et al., 2013; Mulder et al., 2021). This change in heart rate of human divers is similar to the cardiac variability during ascent in dolphins (Williams et al., 1999, 2015).

Blood pressure

In humans performing dry or facial immersion static apnea, the combined influence of (1) sympathetic excitation and chemoreflex engagement from combined inputs of hypoxia, hypercapnia, and lack of ventilatory rhythm (Heusser et al., 2009; Steinback et al., 2010) and (2) peripheral vasoconstriction likely drive the increases in mean arterial pressure (Fig. 2). Normal mean arterial blood pressure at rest is around 90 mmHg in a healthy young adult, but has been seen to increase up to ~150 mmHg during dry static apneas (Fig. 2, Bain et al., 2016; Bain et al., 2017), during apnea and exercise with face immersion (Bjertnaes et al., 1984), and fully immersed apneas (Ferrigno et al., 1997; Perini et al., 2010). However, blood pressure measurements during immersion are logistically difficult and data acquired during underwater diving are scarce. In an innovative study by Ferrigno et al. (1997), two participants with radial artery catheterization performed breath-holds in a hyperbaric chamber to simulate the hydrostatic pressure of a dive to 50 m. While there were substantial elevations in mean arterial pressure during descent (with occasional systolic peaks in one diver reaching 345 mmHg), mean arterial pressure subsided (remaining higher and lower than baseline in divers one and two, respectively) during the ascent phase of the dive (Ferrigno et al., 1997). While there does appear to be different blood pressure profile trajectories between static/surface apneas (for which blood pressure progressively increases) and deep diving (for which blood pressure inversely mirrors dive profile), differences in subject apnea experience, methodological designs, and acquisition tools make comparisons difficult. Unfortunately, with respect to dolphins, available blood pressure data are lacking to explore their adaptational advantages.

Splenic contraction

An additional component of the dive response seen in both humans and seals, and presumed (although not yet confirmed) to occur in dolphins, is the contraction of the spleen and ejection of stored red blood cells (Schagatay et al., 2001; Thornton et al., 2001; Stewart and McKenzie, 2002). For almost a century, the spleen has been considered to act as a dynamic reservoir, capable of expelling blood into the circulation, resulting in an elevated hematocrit concentration (Barcroft and Poole, 1927). Spleen contraction has been reported in human divers, who, while breath-

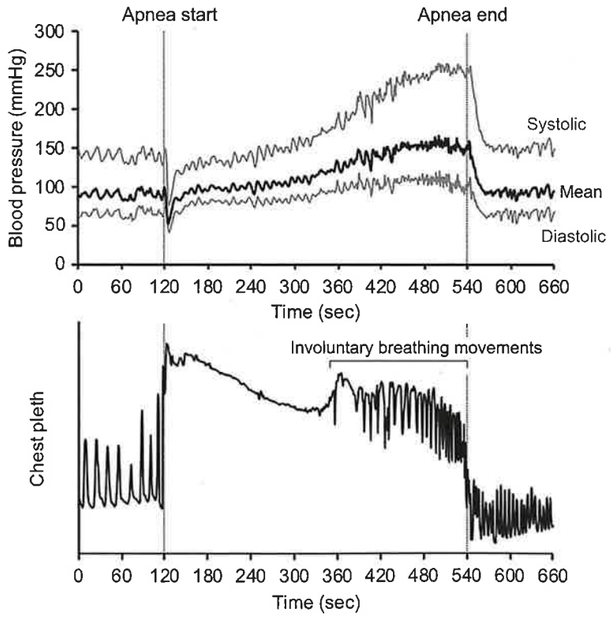

FIG. 2 A 7-min static apnea in a human diver. Continuous blood pressure (via finometer; top panel) and chest plethysmograph (to identify involuntary breathing movements; IBMs; bottom panel). An apnea is divided into an easy-going phase (no IBMs) and struggle phases (with IBMs). Note IBMs progressively intensify with breath-hold duration.

holding, demonstrate a reduction in spleen volume by 18%–35% and an increase in hematocrit of 2%–6% (Schagatay et al., 2001, 2012; Stewart and McKenzie, 2002; Baković et al., 2003). Hypothetically, if there was a 5% increase in hemoglobin concentration from the spleen, this would increase arterial O_2 content by ~5% (assuming all else remains equal; Fig. 4). Larger spleen volumes have been associated with improved apneic performance in competitive divers (Schagatay et al., 2012), and evidence of anatomical and genetic changes in indigenous diving populations (Ama in Japan/Korea and Bajau in Indonesia) also supports the importance of the spleen for apneic diving (Hurford et al., 1990; Ilardo et al., 2018). For example, the Ama have demonstrated a 10.5% increase in hematocrit, following 1 h of repetitive diving to 5–7 m (Hurford et al., 1990). Likewise, the Bajau exhibit larger spleen volumes than demographically matched controls, which have been linked to single nucleoid polymorphisms of the PDE10A gene (Ilardo et al., 2018). Bajau also exhibited single nucleoid polymorphisms to the BDKRB2 gene, which, interestingly, is one of the genes that have been linked to stronger peripheral vasoconstriction during breath-holding (Baranova et al., 2017). With respect to cetaceans, while they generally do not have spleen volumes that are large relative to body mass, cetaceans do have a high prevalence (60%) of one (or more) accessory spleens with similar characteristics to the primary spleen (Tanaka, 1994; Carvalho et al., 2014; Rommel et al., 2018).

Comparative perspectives of the diving response

Just as in the human, the dive response is without a doubt vital to the dolphin's survival while diving. However, for the dolphin spending a considerable time underwater and only intermittently surfacing to breathe, gas management is not just about ensuring there is enough O_2 available to maintain consciousness. Instead, dolphins must use the dive response to both manage the depletion of O_2 and accumulation of metabolic by-products such as CO_2, while also limiting the buildup of nitrogen (N_2). Specifically, dolphins must ensure that there is sufficient available O_2 in the body to support aerobic metabolism where possible. Maintaining aerobic metabolism ensures that dolphins can continue to dive repetitively. Reducing the time spent at the surface is important to maximize the time spent underwater, as required to travel, search for, and catch prey items. Diving can certainly be performed while utilizing anaerobic metabolism in some tissues, but prolonged reliance on anaerobic metabolism eventually results in cellular dysfunction as metabolic end-products accumulate and the supporting glycogen resources deplete. This metabolic *option* is inefficient, as anaerobic metabolism only results in three molecules of adenosine triphosphate (ATP) produced per molecule of sugar, while aerobic metabolism produces 39 ATP. In addition, prolonged anaerobic metabolism eventually forces the animal to stop diving to remove the metabolic by-products (i.e., oxidize lactate) produced. For a wild animal that must breath-hold dive to carry out most of life's activities, this option is not evolutionarily beneficial. Therefore, most diving animals preferentially rely on aerobic metabolism while submerged and stay underwater for durations less than their "aerobic dive limit" (ADL) (Kooyman et al., 1980). ADL is the dive duration associated with the onset of postdive blood lactate accumulation, caused by the necessity for anaerobic metabolism in some body tissues. When diving within the ADL, dolphins can perform repeated dives with short surface intervals. It is likely they only occasionally exceed their aerobic capacity and use anaerobic metabolism sparingly, for instance, to prolong a dive after encountering a high-density aggregation of prey. On these longer dives, anaerobic metabolism should occur in many nonessential tissues, allowing O_2 to be saved for vital tissues that are unable to survive on anaerobic metabolism, such as the brain.

To optimize reliance on aerobic metabolism, despite living mostly without access to ambient air, dolphins are required to balance their short- to medium-term needs efficiently. Maintaining aerobic metabolism can be achieved by limiting dive durations to short dives. During such dives, the dive response is modulated so that circulation to most organs is maintained and O_2 supply is sustained. Therefore, both behavior and physiology, i.e., the dive response, are important to the dolphin to match cardiovascular adjustments to the anticipated behavioral challenge of the next dive (or perhaps sequence of dives)—i.e., "how long will the next dive be and how large a dive response should I have?"

Cognitive control over bradycardia

Dolphins, and other marine mammals, appear to have a conditioned ability to influence the magnitude and rate of change of the dive response—which is not possible for humans. The magnitude of the dive response in dolphins is proportional to the expected length of the dive (Elsner et al., 1966; Fahlman et al., 2019b, 2020b). Similar results have also been reported in other cetaceans and pinnipeds (Jones et al., 1973; Ridgway et al., 1975; Thompson and Fedak, 1993; Jobsis et al., 2001; Elmegaard et al., 2016). Interestingly, when dolphins are able to determine the duration of the apnea, it appears that the reduction in heart rate is minimal (Fahlman et al., 2020b). This suggests that cognition

plays a role in the control of the dive response—an exceptional difference between humans and dolphins. Where the dive response in humans is a reflex response, in dolphins, it is, at least partly, cognitively controlled. This ability to vary the dive response is vital to optimize aerobic metabolism as discussed above, ensuring efficient and effective diving patterns, minimizing the time spent at the surface, and maximizing time spent underwater.

Hypoxia and acid-base disturbance

Blood gases in humans

The separation from ambient air is perhaps the most immediate limiting factor for a submerged mammal, and despite the effect of the dive response, hypoxia and tissue acid-base disturbances are an inevitable part of diving. Due to the influence of hydrostatic pressure, the partial pressure of O_2 and CO_2 in the blood (i.e., PO_2 and PCO_2, respectively) is expected to increase in accordance with Boyle's and Henry's law—where the gas is compressed resulting in higher pressure and higher gas solubility. The increasing alveolar PO_2 and PCO_2 result in an elevated diffusion gradient from the lung into the blood, a phenomenon expected to affect the human and the dolphin equally. In two field studies on human divers, the arterial blood gases were measured at 40 m [$n = 6$, deep pool (Bosco et al., 2018)] and 60 m [$n = 1$, open sea (Scott et al., 2021)]. Both studies showed that arterial blood became hyperoxic (i.e., increased PO_2), during descent. In the pool, arterial PO_2 in 4 (out of 6) divers increased from 94 ± 6 mmHg at the surface to 263 ± 32 mmHg at 40 m, and in open ocean, arterial PO_2 increased from 89 mmHg at the surface to 321 mmHg at 60 m. Unexpectedly, two of the pool divers did not experience hyperoxia at depth (PaO_2 at 40 m was 68 ± 10 mmHg), and the authors postulated that atelectasis (i.e., alveolar collapse) resulted in a right-left intrapulmonary shunt (i.e., blood by-passing the lung), which resulted in their low arterial blood O_2 tensions (Bosco et al., 2018). Interestingly, despite the high solubility of CO_2 in blood and predicted exponential increase in arterial PCO_2 due to hydrostatic loading, arterial PCO_2 across both studies did not exceed 50 mmHg. Even after maximal effort 7.5-min dry static apneas, arterial PCO_2 only increased to 55 mmHg (Fig. 3, Bain et al., 2018b). Whether the solubility of CO_2 buffers the alveolar to arterial PO_2 gradient (Fahlman et al., 2020c), alveolar compression augments pulmonary shunt that reduces gas exchange (Kooyman and Sinnett, 1982; Bostrom et al., 2008; Fahlman et al., 2009), or there exists a buffering capacity to restrain acidosis, these data suggest that the body can mediate the rising arterial CO_2 content.

Blood gases in dolphins

Acquisition of blood samples at depth, from which blood gases can be assessed, has technological and logistical challenges even in controlled human studies. Therefore, blood gas tension and chemistry data for the dolphin have not been regularly measured. Even in seals, which have more accessible blood vessels and have been a more common animal model in physio-logging research, there is limited blood gas data (beyond arterial and venous O_2 tension in the northern elephant seal (*Mirounga angustirostris*) and California sea lion). As such, a comparison of blood gas dynamics in the human and the dolphin is challenging. Nevertheless, a small number of pre- and postdiving measures of blood gas in vertical diving, and cross-sectional measures in static dives do exist for the dolphin (Ridgway et al., 1969; Noren

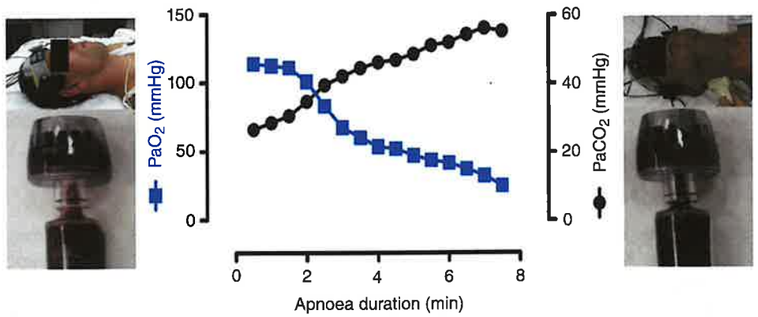

FIG. 3 Arterial blood gases during ~8 min breath-hold in a human. PaO2, arterial partial pressure of oxygen; PaCO2, arterial partial pressure of carbon dioxide. *Reproduced with permission from Bain et al. (2018b).*

et al., 2012; Fahlman et al., 2019a). In the recent study by Fahlman and colleagues, end-expiratory PO_2 following a 4.75-min (284 s) static apnea in a bottlenose dolphin reached 41 mmHg (Fahlman et al., 2019a). In contrast, humans performing static apneas of comparable duration (282 ± 19 s) reported end-expiratory PO_2 of 23.0 ± 0.5 mmHg (Lindholm and Lundgren, 2006).

While there is a paucity of empirical data on blood gas changes during diving in dolphins, it is possible to *hypothesize* that these changes might mirror patterns observed in other marine mammals. Dolphins, like humans and sea lions, inhale before diving. In both the human, as mentioned above, and the sea lion (McDonald and Ponganis, 2013), oxygenation remains high or elevated at depth and mostly shows decline during ascent from depth. In contrast, phocid seals exhale prior to diving (Kooyman and Sinnett, 1982) and their arterial blood O_2 tension declines during the entire duration of the dive. Therefore, as inhalation divers, one could assume that at depth, dolphins are both hyperoxic and hypercapnic, like humans and sea lions.

Involuntary breathing movements

The inevitable changes in blood gas tensions during breath-hold diving are followed by physiological phenomena aimed at removing the body from an asphyxiated state. One such phenomenon in humans is known as involuntary breathing movement (IBMs; Fig. 2). Indeed, if you were to hold your breath for as long as you can endure, you may experience this phenomenon. These IBMs are diaphragmatic contractions, which signal the "physiologic breaking-point," and comprise the struggle phase of an apnea (Schagatay, 2009; Bain et al., 2018b). The intensity of the IBMs increase as the drive to breathe increases, hypoxemia and hypercapnia become more severe and a lack of respiratory rhythm persists. Interestingly, IBMs have been shown to improve cerebral oxygenation (Dujic et al., 2009), especially at higher lung volumes (Palada et al., 2008), which may be critical to slow cerebral hypoxemia.

It is perhaps not surprising that there are no reports of IBMs in dolphins, as apnea is the "normal" respiratory state for dolphins, and this species is not thought to suffer from a physiological break-point and ensuing IBMs. Dolphins have been suggested to have voluntary respiration (Lilly, 1966), but later work proposed that their respiration is both automatic and can be brought under voluntary control just as in other mammals (McCormick, 1969). Nonetheless, the apneustic respiratory mechanism of the dolphin appears to respond differently from the apneustic center in the pons in humans, which may act to terminate IBMs if there is a physiological break-point (Comroe, 1966). Additionally, based on end-expired PO_2 and PCO_2, dolphins live with lower arterial PO_2 and higher arterial PCO_2 and a greater tolerance to elevated CO_2, which in humans triggers the desire to breathe (Ridgway et al., 1969; Fahlman et al., 2015, 2020a; West and Luks, 2016). This blunted hypercapnic ventilatory response may be evidence of a higher chemoreceptor threshold, which results in a higher CO_2 tolerance in the dolphin. In addition, the greater blood CO_2 buffering capacity, resulting from a higher concentration of bicarbonate as compared with terrestrial mammals (Lenfant et al., 1970), means that respiratory acidosis and increasing blood CO_2 tension will be slower to develop.

Loss of consciousness: Hypoxic syncope or shallow-water blackout

If the inevitable changes in blood gases associated with diving exceed extreme thresholds, the arterial O_2 tension may become so low that there can be a loss of consciousness, termed hypoxic syncope. The theoretical lower arterial blood O_2 tensions for consciousness in humans is around 20 mmHg (Ernsting, 1963), which has been supported by data from human breath-hold divers. Lindholm and Lundgren (2006) showed that postapnea end-tidal O_2 partial pressure of 20.3 mmHg (range: 19.6–21.0 mmHg) coincided with a loss of motor control, whereas end-tidal O_2 partial pressure values of only ~3 mmHg higher (mean 23.0 mmHg; range 22.4–23.6 mmHg) did not. In elite apneists under "dry" laboratory conditions, radial artery (Willie et al., 2015) and jugular venous catheterizations (Bain et al., 2016, 2017, 2018a) have demonstrated arterial and central venous PO_2 of 29.6 ± 6.6 mmHg and 25 ± 6 mmHg, respectively, at the end of a breath-hold without syncope (Fig. 3). In an extreme example, and still without syncope, an end-apnea PO_2 tension of 23 mmHg was attained in a diver performing a 435 s breath-hold (Bailey et al., 2017). Fig. 4, which illustrates a hypothetical dive to 100 m, highlights the impact of hydrostatic pressure (particularly on the last 10 m of a dive) on arterial hypoxia and the closeness of such deep diving to the theoretic limit of consciousness.

Dolphins are not reported to suffer mortalities as a result of hypoxic syncope. As "full-time" divers, with a long evolutionary provenance in exploiting an apneic lifestyle, it is perhaps not surprising. For dolphins, a blackout would most likely result in death—and, evolutionarily speaking, this is a definite selection pressure and its avoidance a prerequisite for successful adaptation to an entirely aquatic existence. Nevertheless, how dolphins might avoid hypoxic syncope is interesting. It could be that dolphins avoid exposure to such potentially deleteriously low levels of arterial

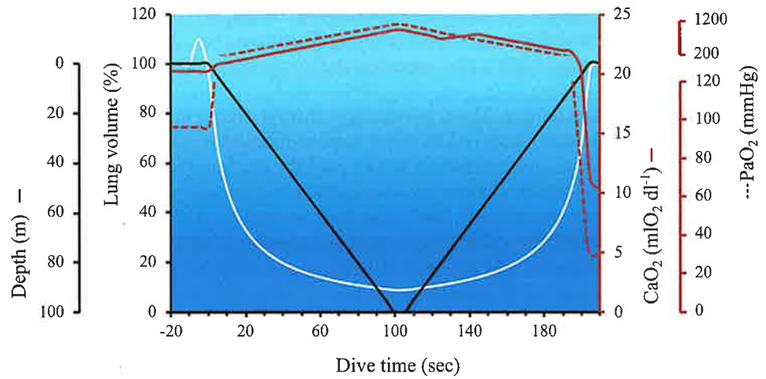

FIG. 4 The impact of hydrostatic pressure (*black*; i.e., depth) on lung volume (*white*) and arterial hypoxia [arterial oxygen content (CaO_2; *dashed red*) and partial pressure of arterial oxygen (PaO_2; *solid red*)] in a human during a simulated dive to 100 m. The increase in lung volume immediately before the start of the dive coincides with lung packing—a maneuver commonly employed by human breath-hold divers to increase the volume of oxygen in the lungs. *Reproduced with permission from Patrician et al. (2021b).*

blood O_2 tensions via sensors, which detect arterial blood O_2 tensions, combined with strict behavioral limits on dive depths and durations. However, considering that some dolphins, such as those in Bermuda, have been recorded to dive to 1000 m (with a total dive time of 13 min, Fahlman et al., 2023), it is likely some animals experience low arterial blood O_2 levels. Cetacean brains have been shown to have higher levels of neuroglobin (Williams et al., 2008; Schneuer et al., 2012), which would help to scavenge reactive O_2 species formed during repeated bouts of ischemia-reperfusion (i.e., an anatomical adaptation to hypoxic exposure). Elevated cerebral glycogen levels along with the astrocyte-neuron lactate shuttle hypothesis (which proposes that the astrocytes increase their rate of glucose uptake, glycolysis, and release of lactate into the extracellular space in response to increased neuron activity), flexible switching between aerobic and anaerobic energy production, and greater tolerance to high lactate concentrations are potential contributing factors (Mason, 2017). Still, the limited data available do not fully explain the dolphin's capacity to maintain neurological performance at low PaO_2.

Pressure effects

Descent into the marine environment comes with the gradual increase in ambient (i.e., hydrostatic) pressure. At the sea surface, all life is exposed to 1 ATA (atmospheres absolute). With increasing depth, the hydrostatic pressure increases by 1 ATA every 10 m (see Chapter 6: Respiratory physiology in the dolphin and other whales) such that at 10 m and 20 m, the hydrostatic pressure is 2 ATA and 3 ATA, respectively. Therefore, descending into the ocean brings with it physiological challenges. However, because humans and dolphins, like all mammals, need to return to the surface to breathe, they are exposed to repeated bouts of elevated pressure, and susceptible to some of the same diving pathologies.

Scholander (1940) proposed that the unusual anatomy of the respiratory system in marine mammals, with a compliant alveolar space and thorax, and stiff conducting airways, might function to reduce pressure-related disease (Scholander, 1940). This balloon-pipe model of the respiratory system has been the main hypothesis as to how dolphins and marine mammals reduce uptake of N_2 and avoid the risks of barotrauma (lung squeeze), N_2 narcosis, and decompression sickness.

Risk of pulmonary barotrauma

Humans have a delicate pulmonary capillary interface (West et al., 1991), and therefore, it is perhaps not surprising that lung injury can occur, due to the cumulative forces of hydrostatic-induced compression and decompression of both the lungs and thoracic cage, centralization of blood volume, hypertension, exertion, and hypercapnic hypoxia. In humans, one such injury, commonly referred to as "lung squeeze," is a form of pulmonary barotrauma that has been extensively reviewed (Ferrigno and Lundgren, 2003; Lindholm and Lundgren, 2009; Dujic and Breskovic, 2012; Mijacika and Dujic, 2016; Moon et al., 2016; Kumar and Thompson, 2019; Schipke et al., 2019). Lung squeeze manifests shortly after surfacing, is characterized by pulmonary edema and hemoptysis (blood originating from the bronchi or lungs) (Boussuges et al., 1999; Lindholm et al., 2008; Patrician et al., 2021a), and is often associated with (1) productive cough, dyspnea, and chest tightness (Cialoni et al., 2012), (2) decrements in lung function and reduced O_2 saturation (Linér and Johan, 2008), and (3) an impairment in pulmonary gas exchange efficiency (Patrician et al., 2021a). Modeling work suggests that human lungs will begin to compress during a dive to 18 m, and the alveoli will be completely collapsed at 235 m (Fitz-Clarke, 2007). During ascent, the reduction in pressure results in lung air expansion and alveolar recruitment/opening (see Chapter 6). As the alveoli are opening, there exists potential for the disproportionate coordination between transpulmonary pressure and regional airway surface tensions that can lead to abrupt compensatory airway equalization (i.e., airway/alveolar "popping"). Beyond pulmonary trauma, but still engendered by hydrostatic compression and decompression, is barotrauma of the paranasal sinuses. As depth and therefore hydrostatic pressure increases, sinuses must be equalized or a relative vacuum in the sinus cavity forms. Failure to equalize pressure during descent elicits mucosal congestion, edema, hemorrhagic bullas, and free blood—manifesting in sharp, intense pain of the maxillary, ethmoid, and/or frontal sinuses. This risk increases under various conditions interfering with equilibration of gas pressure in the body's air-containing cavities (e.g., sinus congestion, Eustachian tube blockage, structural anomaly, inflammation, infection).

The anatomical observations made by Scholander (1940) have helped explain how marine mammals avoid pulmonary barotrauma, with either a highly compliant thorax (as in seals), or with a chest with extensive elastic recoil inwards such that a relaxed state results in almost complete emptying of the lung (see Chapter 6). Consequently, without a stiff chest or residual volume, pressure can push the chest inwards until complete atelectasis (alveolar collapse), which helps prevent transthoracic pressure differences and lung squeeze. Initial evidence for chest collapse was presented by Ridgway et al. (1969) in a seminal study where photos was taken of the chest of Tuffy, a bottlenose dolphin, during a dive to 300 m, with visible compression of the thoracic cavity (see Fig. 14 in Chapter 6).

Measurements of the thorax in dolphin cadavers (Fahlman et al., 2018) and in live pinnipeds (Leith, 1976; Fahlman et al., 2014) have confirmed that chest compression leaves the alveolar space almost empty of air. The highly compliant alveolar space in the dolphin and other marine mammals (Fahlman et al., 2017b) allows alveolar atelectasis (i.e., airway collapse). It has also been reported that some species of dolphins have submucosal venous lacunae in the airways (Cozzi et al., 2005), a tissue that could fill with blood in the tracheal space, which both reduces the tracheal volume and helps stiffen the wall. This further helps to prevent negative pressure and tissue trauma of the upper airways as the pressure increases during a dive.

Cetaceans have lost the air-filled, bony-walled, noncompliant paranasal sinus system found in humans and other terrestrial mammals. However, cetaceans do have gas-filled sacs akin to sinuses (in which the gas is derived from respiratory air but may vary in composition) that are similar to those of the paranasal system. The cetacean accessory sinus system is unique (Fraser and Purvevs, 1960) in that the unpigmented mucosa-lined structures, which are located on the ventral aspect of the skull, are typically associated with hearing and acoustic isolation of the ears (Houser et al., 2004). The accessory sinus system, which is not completely encased in robust, rigid bony compartments, is presumed to be exposed to the effects of changing barometric pressures encountered during diving. In order to avoid sinus barotrauma, it is assumed that there exists a significant amount of structural flexibility within those structures to compensate for the compression and decompression of the air within these spaces—unlike the much less compliant sinuses of the human. In addition, Fraser and Purves (1960) hypothesized that the elaborate venous plexus in the accessory sinus system could help compensate for lost volume of air during compression, by replacing it with blood to avoid dysbaric trauma to the tissues, as well as facilitating hydrostatic equilibrium of the ears, which are surrounded by the accessory sinus system (Costidis and Rommel, 2012).

Risk of nitrogen narcosis

In his book "Silent World," Jacque Yves Costeau coined the synonym for nitrogen narcosis, "L'ivresse des grandes profondeurs," or "rapture of depths" (Cousteau and Frédéric Dumas, 1953). The narcotic effects of high gas tensions are pronounced in the human diver, and the anesthetic effect of nitrogen greatly affects brain function. Depending on depth, time at depth, and inspired lung volume, the alveolar N_2 partial pressure will be elevated and will facilitate diffusion of N_2 into the blood. Nitrogen narcosis may be relatively common in human during breath-hold dives exceeding ~70–90 m, but there are only a few published examples in the literature (Lindholm and Lundgren, 2009; Fitz-Clarke, 2018; Patrician et al., 2021a). Prevalence of reporting is likely impacted due to amnesia (Ferrigno and Lundgren, 2003)—for example, in a recent report by Patrician et al. (2021a) one diver spent 42 s beyond 90 m (with

maximum depth of 117 m). Upon surfacing, the diver did not recall the entire dive, and only slowly began remembering the details of the dive over the subsequent minutes.

Dolphins, and other marine mammals, are not thought to suffer from N_2 narcosis, and Kooyman (1989) suggested that it is unlikely that N_2 levels reach narcotic levels. Still, arterial blood N_2 tensions in a harbor seal (*Phoca vitulina*) exceeded 5 ATA (Kooyman et al., 1972)—levels that would cause severe narcosis in humans. Interestingly, it has been shown that marine mammals appear to have different NMDA (N-methyl-D-aspartate) receptors, which have been suggested to provide resistance toward high-pressure nervous syndrome and possibly also N_2 narcosis (Bliznyuk et al., 2018).

Risk of decompression sickness

In 1908, Haldane first published his seminal work on diving physiology during pressure exposures in a dry pressure chamber, and the first tables to avoid decompression sickness (DCS) were developed (Boycott et al., 1908). Today, it is commonly accepted that DCS, also called "bends," or "caisson disease" is caused by the formation of vascular gas bubbles due to supersaturation in body fluids and tissues when a diver is returning to the surface after a dive (Vann et al., 2011; Pollock and Buteau, 2017). The vascular bubbles do not always result in DCS symptoms, but the probability of symptoms increases with increasing amount of bubbles detected (Sawatzky and Nishi, 1991). Thus, alternative theories were also proposed due to the fact that most SCUBA divers show asymptomatic vascular gas bubbles postdive, whereas DCS is a very rare clinical condition (Ljubkovic et al., 2010, 2011). DCS can also occur in breath-hold divers (commonly referred to as "Taravana," which stems from DCS reports in the indigenous diving populations in Polynesia)—especially those diving repetitively (e.g., spearfishing, safety divers, and/or using underwater scooters) and those performing extreme depths (Cross, 1965; Paulev, 1965; Rahn and Yokoyama, 1965; Schipke et al., 2006; Fitz-Clarke, 2009; Lemaitre et al., 2009; Moon and Gray, 2010; Dujic and Breskovic, 2012). The pathology of DCS, including its manifestation and risk factors, has been extensively reviewed elsewhere (Brubakk and Neuman, 2003); however, in breath-hold divers, symptoms can range from dizziness, nausea, thoracic/skin/joint pain, hemiplegia, paresis, dysarthria, vertigo, and unconsciousness, with short- to long-term prognoses (Cross, 1965; Schipke et al., 2006; Cortegiani et al., 2013; Tetzlaff et al., 2017; Blogg et al., 2023).

Until recently, it was thought that the anatomy of the respiratory system in marine mammals, with stiff airways and compliant alveoli proposed by Scholander (1940), would be sufficient to explain how all marine mammals avoid gas emboli and DCS. However, following naval associated mass-strandings of deep-diving cetaceans (Jepson et al., 2003; Fernández et al., 2005), with symptoms resembling DCS in human divers, a number of studies have shown that marine mammals experience elevated blood and tissue gas tensions that can result in gas emboli, and at times, trauma (Fig. 5, Moore et al., 2009; Van Bonn et al., 2013; Fernández et al., 2017). Theoretical modeling suggested that despite the unusual respiratory anatomy, the repeated dive behavior seen in dolphins would result in blood and tissue N_2 levels that would likely result in gas emboli and a high probability of severe symptoms of DCS (see Figure 3 in Fahlman et al., 2021). Furthermore, work on the cardiorespiratory physiology of cetaceans has attempted to elucidate the mechanism by which cetaceans avoid gas emboli, resulting in the *selective gas exchange* hypothesis (García-Párraga et al., 2018; Fahlman et al., 2021). In short, the lung architecture in the dolphin results in two distinctly different pulmonary regions: one that is open for gas exchange and the other which is collapsed. The conditioned ability to vary heart rate (Fahlman et al., 2020b), and likely blood flow, may enable marine mammals to direct blood either to the region that is open or collapsed and allows selective exchange of gases with different gas solubilities (West, 1962; Farhi and Yokoyama, 1967). This would provide the ability to exchange O_2 and CO_2 while minimizing N_2 exchange (García-Párraga et al., 2018; Fahlman et al., 2021). This hypothesis also explains how stress, such as exposure to sonar, might result in failure of this mechanism causing uptake of N_2 and increasing the risk of gas emboli formation. This provides avenues for new areas of research, offering an alternative explanation for how marine vertebrates can avoid diving-related problems observed in human divers, and how failure of this adaptation may result in diving-related trauma (Frantzis, 1998; Fernández et al., 2005, 2017; García-Párraga et al., 2014; Fahlman et al., 2017a).

Other considerations to minimize metabolic rate

Other factors that affect metabolic rate, and therefore impact on diving, include mechanisms to retain heat balance (i.e., thermoregulation, see Chapter 3) and mechanisms to improve the efficiency of swimming (see Chapter 2: Energetic costs of rest and locomotion in dolphins). For humans, prolonged immersion anywhere other than in the tropics requires a wet suit to protect against the fundamentally different thermal properties of water than air. This challenge is

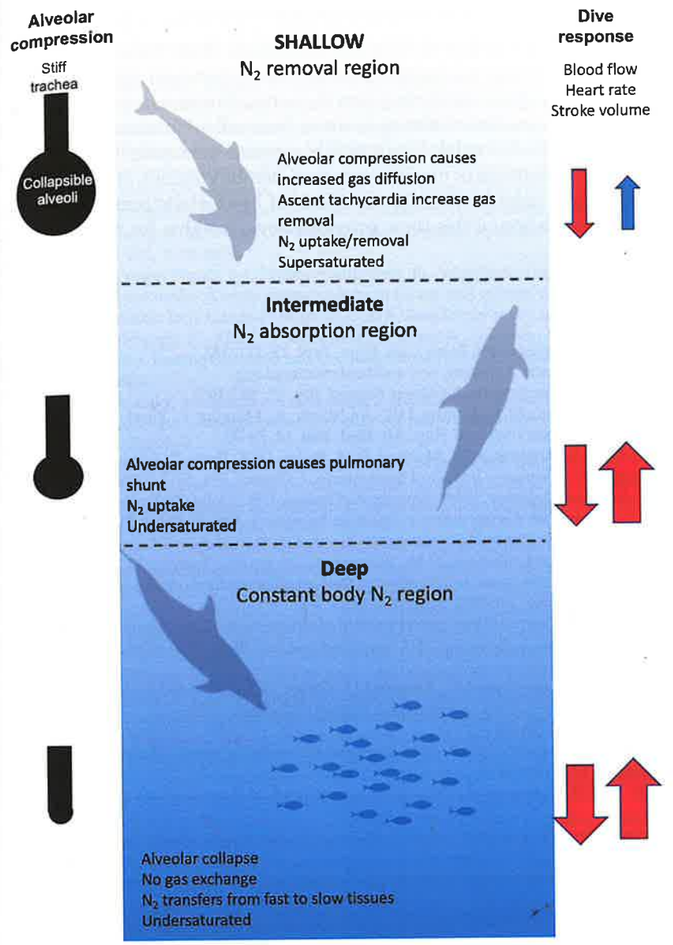

FIG. 5 Risk factors for gas bubble formation, across three generalized regions: shallow (0 to ~30 m), intermediate (~30 to ~200 m), and deep (beyond ~200 m). In the shallow region, tissue and blood PN_2 begins to exceed ambient PN_2, and N_2 is removed. In the intermediate region, ambient PN_2 exceeds tissue and blood PN_2 and N_2 is taken up until the alveoli collapse in the deep region. Following alveolar collapse, N_2 is redistributed from fast tissues to slow tissues. On the left, the balloon/pipe model proposed by Scholander (1940) shows how increasing dive depth causes alveolar compression and collapse. Increasing lung partial pressure initially causes an increase in the gas diffusion rate in the shallow region. In the intermediate region, the compression of the alveoli results in reduction and then cessation of diffusion as the alveoli collapse. On the right, arrows illustrate how diving alters heart rate, stroke volume, and cardiac output (the dive response). During descent (*arrow down*), the heart rate, stroke volume, and cardiac output decreases (*red color*), with a greater reduction with increasing dive duration and depth (*thickness of arrow*). During ascent (*arrow up*), the dive response is maintained until the animal approaches the surface, where the heart rate, stroke volume, and cardiac output increase (*blue colored arrow*) to prediving levels to prepare the animal to rapidly restore the O_2 stores, and remove CO_2 and N_2. *Modified from Fahlman et al. (2021).*

perhaps one of the most difficult faced by mammals making the transition from land to the sea. In retaining the same high body temperatures of terrestrial mammals (including humans), these animals face large thermal gradients and potential heat loss, without adaptations that help minimize this thermal loss.

One of the most notable adaptations recorded in the Ama freedivers was their thermoregulatory adaptation to cold exposure. Historically, the Ama dove year-round in only cotton linens, despite sea temperatures ranging from 27°C in the summer to 10°C in the winter. Early studies concluded that the Ama may have an improved (i.e., reduced) thermal conductance based on findings of improved peripheral vasoconstriction, increased basal metabolic rate in the winter (one of the few clearly recognized and described instances of human metabolic adaptation to regular cold water exposure), and higher shivering threshold (i.e., takes longer to shiver), compared to controls with comparable subcutaneous fat thickness (Rennie et al., 1962; Hong et al., 1986). However, with the introduction of wetsuits (as early as 1977, and more common by 1980), many of these thermoregulatory adaptations against cold in the Ama disappeared and these were no longer evident in the 1980s (Park et al., 1983a,b; Ferretti and Costa, 2003). Unlike the human, where endogenous heat conservation mechanisms are not adequate to resist heat loss in water, dolphins have increased insulation provided by a thick subcutaneous blubber layer (see Chapter 3).

Conclusion

Breath-hold diving exposes all mammals to a number of extreme environmental stresses. Both humans and dolphins show basic mammalian features in their response to diving, but there are stark contrasts between dolphins living permanently at sea, and humans occasionally making forays from the surface into the water. Our understanding of the diving physiology of dolphins nevertheless benefits from studies of diving in more "accessible" human subjects to compare and contrast with the more limited studies available from dolphins and other marine mammals. In this chapter, we have explored similarities and differences between features of the dive response, hemodynamics, and acid-base changes during diving and we have compared strategies used to mediate the effects of hydrostatic pressure on the body. While many differences exist, a comparative exploration of this topic provides novel insights for future work.

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