Diving Physiology and Decompression Sickness: Considerations from Humans and Marine Animals

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ABSTRACT. The objective of scientific divers using scuba as a research tool is to be able to effectively work under pressure without incurring acute or chronic health effects. Chapters in this volume address underwater science results achieved through the use of scuba. This particular discussion considers physiological and decompression sickness parameters and effects that all humans and marine animals are subject to on a dive. We know that decompression sickness is triggered by a rapid reduction of ambient pressure that allows dissolved inert gas in the body to come out of solution in gas form (i.e., as bubbles.) Dive computers have been accepted as effective tools in assisting divers with the real-time monitoring of their decompression status. Oxygen-enriched air mixtures allow extensions of bottom times in certain depth ranges and optimize decompression on ascent. However, we continue to search for better tools and methods to take our science deeper. Since the publication of Haldane’s “Prevention of Compressed-Air Illness” over 100 years ago, a variety of new research directions have attempted to further explain and reduce the occurrence of the decompression sickness syndrome. Humans have ventured under water for at least 2,000 years without the use of an external compressed air source. However, observations of physiological adaptations in breath-hold diving marine mammals and birds eclipse the capabilities of human breath-hold divers. Nitrogen narcosis and neurological decompression sickness on a single breath-hold dive can occur because of the depths and durations involved. The consummate divers, marine mammals and diving birds, have evolved numerous physiological and morphological adaptations that contribute to such remarkable diving capacity and obviate the two fundamental concerns of diving physiology: oxygen store management and the effects of pressure at depth. A more thorough understanding of the physiology underlying these phenomenal divers may also assist in preventing and treating diving-related pathologies in humans.

INTRODUCTION

Scuba diving conducted by scientists is an invaluable research tool. Since the advent of scuba in the 1950s, placing the trained scientific eye under water on compressed gas has provided research value and flexibility that unmanned systems often could not. One metric substantiating this value is provided by peer-reviewed scientific publications in high-impact journals of research that could not have been performed without the use of scientific diving techniques (Lang, 2007). For example, a recent computer search for the keyword “scuba” returned 671 articles from all 397 volumes published since 1967 of the Journal of Experimental Marine Biology and Ecology (JEMBE, Elsevier).

The purpose of a scientific diving project is the advancement of science requiring divers to use scientific expertise in studying the underwater environment. More often...
than not this research is conducted in challenging and remote environments such as under polar ice, from research vessels at sea, or at atolls far removed from immediate medical assistance. Yet exposure statistics document that these research activities are performed to a remarkable degree of safety and scientific productivity (Lang, 2009; Sayer et al., 2007).

Access to the underwater research site is provided by scuba, and we are in continuous pursuit of technology to expand our operational window within acceptable safety limits. Safety concerns, however, have gradually eroded our depth limit to what has become a 60 m compressed air scuba window for scientific diving in the United States with impacts on how and where research is conducted with diving. Our understanding of the ocean ecosystem in toto is therefore potentially impaired. For example, shallow-water coral reefs are well understood, as are the horizontal linkages between adjacent mangroves and sea grass beds and their contributions to the reef ecosystem. But diving limitations make vertical linkages and deep systems problematic to investigate. Akin to limiting a tropical rainforest biologist from climbing higher than 10 m (thereby missing the majority of biodiversity that resides in the canopy), a scientific diver cannot effectively study the biodiversity and contributions of the deep reef to the shallow-reef system because of current technology and training limitations. The U.S. Department of Labor’s Occupational Safety and Health Administration does not restrict the scientific diving community with regard to technology, leaving the operational flexibility to utilize mixed gases, rebreathers, and saturation habitats in research methodology to meet the nation’s marine science needs.

Physiological considerations and the probability of decompression sickness affect humans and marine animals on every dive. We know that decompression sickness is triggered by a rapid reduction of ambient pressure that allows dissolved inert gas in the body to come out of solution in gas form (i.e., as bubbles). Since the publication of Haldane’s (1908) “Prevention of Compressed-Air Illness,” a variety of new research directions have attempted to further explain and reduce the occurrence of the decompression sickness syndrome. Humans have ventured under water for at least two millennia without the use of an external compressed air source. However, observations of physiological adaptations in breath-hold diving marine mammals and birds eclipse the capabilities of human breath-hold divers. Even the current world-class human depth record of 214 m and breath-hold static apnea time of 11 minutes 35 seconds are orders of magnitude less than recorded marine mammal dives. Nitrogen narcosis and neurological decompression sickness on a single breath-hold dive can occur because of the depths and durations involved. The consummate divers, marine mammals and diving birds, have evolved numerous physiological and morphological adaptations that contribute to such remarkable diving capacity and obviate the two fundamental issues of diving physiology: oxygen store management and the effects of pressure at depth. A more thorough understanding of the physiology underlying these phenomenal divers may also assist in preventing and treating diving-related pathologies in humans.

Waking up the aquatic reflexes we harbor as infants is perhaps one of the most important elements of freediving, a 2,000-year-old diving technique still practiced today by the Ama of Korea and Japan. In 1967, Robert Croft was the first to practice ‘lung packing,’ the glossopharyngeal breathing technique. Today, extreme (No Limits) freediving prompts the question as to whether there is a finite limit to the depths a human can dive. Some contributing factors involve training and fitness regimen, mental strength, state of relaxation, chest (muscles/ribcage) elasticity, partial lung collapse to a residual volume of <0.5 L, possible pulmonary edema, glossopharyngeal insufflation that can increase pulmonary gas stores to >12 L, alternate equalization modes of sinuses and middle ears, dive times of about 4½ minutes, and a freediving decompression stop. Physiological phenomena such as nitrogen narcosis and neurological decompression sickness can affect freedivers on a single breath-hold dive due to the speed of descent, depths, and durations involved.
demonstrate proper buoyancy, weighting, and a controlled ascent, including a “hovering” stop. Ascent rates are controlled at a maximum of 10 m/min from 20 m and are not to exceed 20 m/min from depth, at the rate specified for the make and model of dive computer or table being used. Scientific diving programs and many dive computers usually require a stop in the 5 m depth range for 3–5 minutes on every dive. Scientific divers receive practical training in drysuits, which must have a hands-free exhaust valve. A buoyancy compensator, capable of horizontal deflation, is required with drysuit use for emergency flotation but should not be used under water to avoid uncontrolled buoyancy problems. In the case of any emergency ascent, breathing 100% oxygen above water is preferred to in-water air procedures for omitted decompression. The effects of multiday, repetitive diving on diver physiology were evaluated by Lang and Vann (1992), who estimated decompression sickness (DCS) incident rates in the USA to be 1 per 1,000 dives in the commercial diving sector, 2 per 10,000 dives for the recreational scuba community, and 1 per 100,000 dives in the scientific diving universe.

Scientific diving programs provide continuous training, recertification, and dive site supervision, which helps maintain established safe diving protocols. Making repetitive dives over multiple days may result in a higher risk of DCS. Increasing knowledge regarding the incidence of DCS indicates that the ability to predict the onset of DCS on multilevel, multiday diving is even less sensitive than the ability to predict DCS on single, square-wave-profile dives. There appears to be good evidence that there are many variables that can affect the probability of the occurrence of DCS symptoms. The ability to mitigate these variables through education, supervision, and training appears to be possible by promoting good levels of hydration, fitness, rate of ascent, and fatigue management. There is adequate technical support for the use of oxygen-enriched air (nitrox) and surface-oxygen breathing in scientific diving where higher gas loadings are anticipated in multilevel, multiday dives. Decompression sickness is generally recognized as a probabilistic event, which tends to steer the scientific diving community toward a more conservative approach to occupational diving.

The order of dive profiles was investigated by Lang and Lehner (2000), in part because of the difficulty for scientific divers to adhere to the “dive progressively shallower” rule while on projects investigating coral reefs at varying transect depths. More importantly, the genesis and physiological validity of the “dive deep first” rule was in need of examination. Historically, neither the U.S. Navy nor the commercial sector has prohibited reverse dive profiles. Reverse-dive profiles are acknowledged as being performed in recreational, scientific, commercial, and military diving. The prohibition of reverse-dive profiles by recreational training organizations cannot be traced to any definite diving experience that indicates an increased risk of DCS. There is no convincing evidence that reverse-dive profiles within the no-decompression limits lead to a measurable increase in the risk of DCS. This means that there may be no reason for the diving communities to prohibit reverse-dive profiles for no-decompression dives less than 40 m and with depth differentials less than 12 m.

Oxygen-enriched air (nitrox) has been used in the scientific diving community since the early 1970s. Lang (2001, 2006) reported that for entry-level, open-circuit nitrox diving there is no evidence that shows an increased risk of DCS with the use of oxygen-enriched air (nitrox) compared with compressed air. A maximum PO2 of 1.6 atm is generally accepted based on the history of nitrox use and scientific studies. Routine CO2 retention screening is not necessary for open-circuit nitrox divers. Oxygen analyzers should use a controlled flow-sampling device for accurate mix analysis, which should be performed by the blender and/or dispenser and verified by the end user. It is important to ensure that equipment used with oxygen or mixtures containing over 40% oxygen by volume are designed, dedicated, and maintained for oxygen service.

Operational Diving Procedures

Operational guidelines for remote scientific diving operations were promulgated on a consensual basis by the senior practicing scientific divers for blue-water diving by Heine (1986) and for polar diving by Lang and Stewart (1992) and Lang and Sayer (2007). A phased approach toward the expansion of the scientific diving operational window from 60 m to 90 m was published by Lang and Smith (2006), resulting in the evaluation of commercial and military diving methods for the science community through surface-supplied diving, rebreathers, mixed gas, and saturation techniques. The most immediately transferable method for access to 90 m is surface-supplied diving with mixed gas. This would allow for the dive profile management to occur topside, and for the scientist at the end of the hose to be able to focus on the scientific data collection. There are some disadvantages, such as limitations on horizontal mobility, that would make this method not applicable to all sites of scientific interest. However, surface-supplied training is not overly complicated and would allow for a quick transition from scuba to helmet-and-hose diving given the appropriate commercial diving equipment and topside management (Lang and Robbins, 2009). Deep-air diving for short-bottom durations under ideal conditions remains a possibility for very experienced divers who are cognizant of gas management, nitrogen narcosis, decompression strategies, and maximum operating depth limits of partial pressure of oxygen. Advanced tools such as rebreathers are not new technology, but with their surge in popularity in the technical diving community we are hopeful that with engineering solutions to support their increased reliability and reduce maintenance efforts they will evolve into a mainstream tool as well.

The U.S. scientific diving community has long adhered to a proven experience-accumulation schedule. Depth certifications provide a mechanism to gather diving experience incrementally, and scientific dives are planned around the competency of the least experienced diver. Diving with compressed air in scientific diving operations is not permitted beyond a depth of 58 m. The
100-hour scientific diver training course consists of theoretical training, practical skills training in confined water, and completion of 12 supervised open-water dives in a variety of dive sites for a minimum cumulative bottom time of 6 hours. Additional training is provided for diving specialties such as decompression diving, surface-supplied diving, mixed gas or oxygen-enriched air (nitrox) diving, semi- or closed-circuit rebreather diving, saturation diving, blue-water diving, drysuit diving, overhead environment (ice, cave, or wreck) diving, and altitude diving.

All scientific diving is planned and executed in such a manner as to ensure that every diver maintains constant, effective communication with at least one other comparably equipped, certified scientific diver in the water. This buddy system is based upon mutual assistance, especially in the case of an emergency. If loss of effective communication occurs within a buddy team, all divers surface and reestablish contact. A dive flag is displayed prominently whenever diving is conducted. Scientific diving is not conducted unless procedures have been established for emergency evacuation of the divers to a hyperbaric chamber or appropriate medical facility and these procedures have been approved by the Diving Officer. Diving first aid training is a requisite for scientific diver certification and emergency oxygen kits are present at the dive location. Hyperbaric chambers, as a rule, are not required to be on site. In the case of an asymptomatic diver diving within the dive computer no-decompression limits during the previous 48 hours, there should be a minimum 12-hour delay period with no diving prior to flying. The longer the diver delays an ascent to altitude, the lower the probability of onset of decompression sickness symptoms.

Diving Medical Surveillance

Scientific divers who are exposed to hyperbaric conditions must possess a current diving medical certification. In passing that examination the diver will have been declared by the physician to be medically fit to engage in diving activities as may be limited or restricted in the scientific diver medical certification. All medical evaluations are performed by, or under the direction of, a licensed physician of the applicant diver’s choice, but preferably one trained in diving/undersea medicine. The diver must be free of any acute or chronic disabling disease or conditions contained in the list of conditions by Bove (1998) for which restriction from diving may be recommended. There are currently no fitness standards per se for scientific divers other than during the initial scientific diver training course, which includes in-water time and distance challenges for swimming. A stress tolerance test can be prescribed by a physician based on preliminary screening that indicates the potential of a higher than normal risk of coronary artery disease. Cardiac events are the proximate cause of more than 30% of diving fatalities in the recreational diving community (Vann and Lang, 2011). Medical evaluations are completed before a diver may begin diving and thereafter at five-year intervals up to age 40, three-year intervals after the age of 40, and two year-intervals after age 60. Any major injury or illness or any condition requiring hospital care requires diving medical clearance. If the injury or illness is pressure-related, then the clearance to return to diving must be performed by a physician trained in diving medicine.

Decompression Sickness

Breathing and Inert Gas

The increased pressure gradient between inspired gas and the dissolved gas tension in the body at depth results in an equilibration of the pressure differential leading to saturation. The composition of the air we breathe consists of nitrogen (79%), an inert gas that is absorbed and dissolved in the bloodstream and tissues. The nitrogen partial pressure (PN₂) at sea level in the lungs and surrounding tissues is in equilibrium. Ambient pressure increase (on descent) causes denser air in the lungs to be driven into the tissues to maintain this equilibrium, a process termed ongassing. Ambient pressure decrease (on ascent) causes the increased PN₂ in the tissues to be driven into the lungs, called offgassing.

It takes time for nitrogen to enter and to leave the body. Upon ascent the body begins to eliminate N₂. If too much N₂ is still present after surfacing, the excess nitrogen forms bubbles in the body, creating microscopic clots that impair circulation and can damage endothelial linings of vessels. Decompression sickness symptoms range from skin rash, extreme fatigue, coughing, and painful joints to paralysis and unconsciousness. Commonly accepted prevention is to stay within dive computer no-decompression limits, maintain slow ascent rates (<10 m/min), perform safety stops, and not run out of breathing gas.

Haldane and Prevention of “The Bends”

Our fundamental knowledge of decompression was provided by Boycott, Damant, and Haldane (1908) in their paper “The Prevention of Compressed Air Illness.” Haldane initially used goats and later divers in his decompression experiments to validate his dive tables and make some important observations and findings: no diver had the bends after rapid decompression from 12.8 m (42 ft) to the surface; the general principle that a 2:1 pressure difference could be tolerated; the concept of staged decompression; a model using six compartments with different half times; and deep compressed-air test dives to 64 m. The tables describing uptake and elimination of nitrogen were developed by Haldane’s son Jack, aged 13 at the time, who with Ronald Fisher and Sewall Wright later became the founder of population genetics (Lang and Brubakk, 2009). Validation of Haldane’s tables was followed by the Royal and U.S. navies’ adoption of their use in 1908 and 1912, respectively. They were revised in 1957 and became the diving guide for military, commercial, scientific, and recreational diving for decades. Subsequently, decompression physiologists and modelers modified surfacing ratios, ascent rules, Workman’s M-values, Thalmann’s algorithm, probabilistic models, bubble
models, and deep stops. Despite these efforts, gas content models that are direct descendants of Haldane’s model remain the most prevalent approach to decompression (Doolittle, 2009).

Decompression Sickness and Bubbles

Decompression sickness is a syndrome caused by a reduction of absolute pressure and a separation of gas in body tissues due to inadequate decompression leading to an excessive degree of gas supersaturation. In 1879, Paul Bert (1978) demonstrated that decompression of animals after a hyperbaric exposure produced bubbles in the blood. The signs and symptoms of DCS and treatment with oxygen and recompression are well described. However, other aspects of DCS are poorly understood, such as the relationship between gas phase separation and DCS injury and the large variation in individual susceptibility to DCS.

Bubbles within the blood and tissues cause vascular obstruction leading to hypoxic tissue damage. Bubbles activate various plasma proteins including the coagulation cascade, complement, and kinins (Ward et al., 1987), and are associated with aggregation of platelets. Vascular endothelium is a monolayer of cells lining blood vessels. The endothelium senses stimuli and triggers release of vasoactive substances including nitric oxide (NO), which can inhibit adhesion of platelets and leukocytes. In response to inflammatory signals initiated by bubbles, endothelial cells become activated, generating endothelial microparticles (EMP), which in turn may reduce endothelial function. Vascular bubbles can injure endothelium and reduce the vasoactive effects of other compounds such as substance P and acetylcholine (Nossum et al., 1999). There is growing evidence that the endothelium may play a key role in the development of DCS.

Venous gas bubbles (VGB) are found in the vasculature and right heart after recreational and professional dives and can be monitored and documented with the use of ultrasound by either Doppler or echocardiography (Eftedal and Brubakk, 1997). There is a statistical relationship between detectable bubbles and the risk of DCS (Eftedal et al., 2007). Doppler-detected VGB is a useful method of measuring decompression stress and the absence of VGB is a good indicator of decompression safety.

Exercise has long been thought to be a risk factor for the development of DCS. However, more recent studies have revealed that pre-dive exercise and exercise performed during a decompression stop may significantly reduce bubble formation. Wisloff and Brubakk (2001) serendipitously found that aerobic endurance exercise training reduced bubble formation in rats exposed to hyperbaric pressures. In follow-up studies, an acute bout of exercise produced the same protective effect against DCS. Rats that were exercised 20 hours prior to a 60 m dive for 45 minutes had protection from bubble formation and improved survival (Wisloff et al., 2004). Survival time was lengthened even with exercise at 48 hours prior to diving. Dujic et al. (2004) found that a single bout of high-intensity exercise in humans 24 hours prior to a dive significantly reduced the number of bubbles in the right heart. In humans, the optimum time to exercise appears to be shorter; exercise just 2 hours prior to a dive provides a protective effect (Blatteau et al., 2005).

Mild exercise during decompression decreases the number of bubbles, most likely due to increased gas elimination from increased alveolar (lung air sacs) ventilation (Dujic et al., 2005). In follow-up studies, post-dive exercise caused an eightfold reduction in gas bubbles, presumably due to increased blood flow causing a depletion of bubble nuclei at the blood vessel lining surface (Dujic, 2009). Hence, exercise performed 24 hours prior to decompression, during decompression stops, and after a dive is not harmful as previously thought, but appears to protect from DCS. The mechanism responsible for these interactions may be related to nitric oxide (NO).

There is growing evidence that the endothelium may play a role in the development of DCS. Endothelial nitric oxide (NO) is an important vasodilator and can attenuate bubble formation and incidence of DCS. Rats that are given an NO blocker exhibit far greater bubble formation and minimal survival following a dive compared to controls (Wisloff et al., 2003, 2004). Moller-løkke et al. (2006) demonstrated similar findings in a pig model, as did Dujic et al. (2006) in humans; pre-dive administration of nitroglycerin to humans results in a reduction of bubble formation after diving. Thus, administration of an NO donor (such as nitroglycerine) may be a reasonable alternative to exercise and may protect against DCS.

Heat stress is a nonpharmacological preconditioning strategy that can lead to protection against various types of insults such as ischemia, hypoxia, inflammation, and bubble-induced injury. Activation of the heat shock protein HSP70 by mild hyperthermia allows cells to resist subsequent insults that would otherwise result in death. This response is referred to as preconditioning. Blatteau et al. (2009) showed that a single pre-dive sauna session significantly increased HSP70 and decreased circulating bubbles after a chamber dive. Previous work by the same group showed that moderate dehydration and hypovolemia induced by pre-dive exercise could decrease VGB in divers (Blatteau et al., 2007). They hypothesize that heat-exposure-induced dehydration and NO pathway could be involved in this protective effect, but further investigation is needed to understand the heat-exposure-induced reduction in bubble formation. The role of HSP may be more related to the attenuation of tissue reaction to vascular bubbles than to direct reduction of bubble formation.

Diving in cold water tends to increase the risk for DCS. Previous studies in the 1960s showed that reduced blood flow caused by vasoconstriction and the resultant reduction in inert gas washout from tissues caused symptoms of DCS. However, more recent work by the U.S. Navy (Ruterbusch et al., 2004, 2005) showed that the risk of DCS could be lowered by keeping the diver warm during decompression. It may be beneficial for the diver to be cold during the bottom phase of the dive but not during decompression. Vasooconstriction can hinder the uptake of gas during the dive while increased peripheral tissue perfusion from being warm during decompression may result in a greater elimination of inert gas (Mueller, 2007).
In a recent article, Møllerlokken and Eftedal (2009) suggested that genetic and epigenetic makeup of individual divers may play a role in the variable susceptibility to DCS. Could there be genetic links between endothelial dysfunction and DCS? Genes involved in NO homeostasis display individual differences in activity. Since NO is an important mechanism in bubble formation, genetic links between endothelial dysfunction and DCS may be possible. Hence, genetic variations in humans may explain the variable susceptibility to DCS. Møllerlokken and Eftedal (2009) proposed to study alterations in genetic expression profiles of RNA in vascular endothelium following decompression using rat models, which may provide further knowledge of genetic variability in the physical and biochemical changes experienced in diving. By understanding the genetic basis of individual responses to diving, we may be better able to predict individual risk of developing DCS with the potential to prevent or relieve disease by preconditioning or pharmacological interventions.

FREEDIVING

DECOMPRESSION SICKNESS IN BREATH-HOLD DIVERS

Breath-hold diving (freediving) is also a method utilized in underwater research and can also subject scientists to decompression sickness. Unlike scuba divers, breath-hold divers do not breathe compressed gas; hence the nitrogen that remains in the lungs after the last breath before diving is the only inert gas that could accumulate in tissues. During a breath-hold dive, compression of the chest increases the nitrogen partial pressure in the alveoli, which causes nitrogen to be taken up by the blood. It was previously assumed that a breath-hold diver could not accumulate enough nitrogen concentration in the tissues to cause the supersaturation that results in decompression sickness. However, reports from the 1960s from the Ama divers in Japan describe symptoms of decompression sickness in these pearl divers, including partial or complete paralysis, vertigo, and loss of consciousness. Cross (1965) described a syndrome in pearl divers called “Taravana,” which means to “fall crazily” and most likely represented decompression sickness. These divers performed frequent dives to over 30 msw (100 fsw) with bottom times of 30–60 seconds. They repeated these dives with short surface intervals for 6 hours per day. Cross (1965) also reported another group of divers who had surface intervals twice as long as the Tuamotu divers and did not get DCS. Other reports have described decompression sickness in breath-hold divers. Schipke et al. (2006) reported 90 cases of decompression sickness after repetitive breath-hold dives. In a recent review of the literature and breath-hold diving, Lemaitre et al. (2009) reported 141 cases of DCS in 447 divers.

Lanphier (1965) proposed that repeated deep breath-hold dives separated by short intervals at the surface could lead to progressive accumulation of enough nitrogen to cause decompression sickness. Short surface intervals do not allow tissue nitrogen to be eliminated. Hence, nitrogen can accumulate in the tissues during repeated breath-hold dives equal to the amount found in scuba divers. As in scuba diving, repetitive breath-hold diving has been reported to produce venous gas emboli detectable with ultrasound Doppler (Spencer and Okino, 1972). A single deep breath-hold dive is much less likely to lead to decompression sickness; however, two reports of neurologic symptoms occurring after a single deep breath-hold dive may represent DCS (Magno et al., 1999; Desola et al., 2000).

Repeated breath-hold dives and short surface intervals are factors that predispose to decompression sickness. Fahlman and Bostrom (2006) have suggested that increasing the surface interval to at least twice the duration of the dive may help reduce accumulated tissue nitrogen and reduce the incidence of DCS in breath-hold divers. Understanding how marine mammals avoid excessive nitrogen tissue concentrations could help reduce decompression sickness in human breath-hold divers and scuba divers.

FREEDIVING HISTORY

For the first nine months of their lives, humans exist in an aquatic environment very similar to sea water. If an infant is submerged, it instinctively holds its breath for up to 40 seconds while swimming breast strokes. It appears that we seem to lose this innate diving ability as soon as we commence walking. Waking up these reflexes is one of the most important elements of freediving, thus giving humans better abilities to be protected at greater depths. Relevant adaptations for freedivers would include reflex bradycardia, blood shifts, vasoconstriction, and splenic contraction.

The Ama of Japan and Korea are female pearl divers who still use a diving technique at least 2,000 years old. Women older than 17 years of age use rocks to descend to the bottom where they pick up shells and sea weeds; they dive naked 8–10 hours per day in water barely over 10°C. Rahn and Yokoyama (1965) reviewed the diving physiology of the Ama, which remains the baseline of our knowledge of breath-hold diving physiology.

Chatzistathis, a leading sponge diver from Symi, Greece, was 1.70 m tall and weighed 65 kg. He suffered from remarkable lung emphysema, smoked tobacco extensively, and was part deaf from a life of diving without proper equalization. In 1913, at age 35, Chatzistathis salvaged an anchor from estimated 88 m depth, freediving up to three minutes at a time. He was carried down by a heavy stone, a primitive diving technique as old as the Greek civilization itself. In 1962, Enzo Maiorca was the first to reach the fateful 50 m barrier unassisted, despite predictions from scientists that beyond 50 m the human lungs would collapse from the pressure. Jacques Mayol was introduced in 1966 and revolutionized freediving with his use of Eastern yoga and meditation traditions, rather than the previous norm of heavy hyperventilation.

In 1967, Robert Croft of the U.S. Navy was the first to freedive beyond 70 m and his achievements were important in establishing most modern scientific conclusions about freediving, among them the mammalian diving reflex and the blood shift phenomenon. He was the first record breaker to use “lung packing,”
the glossopharyngeal breathing technique first described in polio patients by Dail (1951). Loring et al. (2007) measured results of glossopharyngeal insufflations and found maximal lung volume was increased by 0.13–2.84 L, resulting in volumes 1.5–7.9 SD above predicted values. The increased circumference of the thorax and a downward shift of the diaphragm enable a larger filling of the lungs through chest expansion. The amount of gas in the lungs after packing increased by 0.59–4.16 L, largely due to elevated intrapulmonary pressures of 52–109 cmH₂O that compress gas in the lungs to 100 cmH₂O (=10 kPa or 10% more air compared to 0 kPa).

By 1999, Francisco Rodriguez (aka Pipin Ferreras) and Umberto Pelizzari pushed each other competitively to 150 m depth records in No-Limits. By 2003, Tanya Streeter mirrored Angela Bandini’s 1989 feat by breaking the intergender No-Limits world record, reaching 160 m depth.

Germontpré et al. (2010) described a technique by Patrick Musimu who, by training, was capable of allowing passive flooding of the sinuses and middle ear with sea water during descent by suppressing protective reflexes during this process. Musimu attempted to breach 200 m in No-Limits, but outside the supervision of any diving federation. On one last training attempt he reached 209 m depth with his sled and successfully returned to the surface. Minutes after surfacing, he suffered symptoms of decompression sickness and received hyperbaric treatment, which canceled the public attempt scheduled a few days later.

MODERN-DAY FREEDIVING LIMITS

Herbert Nitsch currently dominates deep freediving and aimed to surpass Musimu’s unofficial but widely acknowledged 209 m dive in this increasingly challenging discipline. On his 4½ minute 214 m record deep dive, Nitsch used deep-water breath-hold decompression by ascending very slowly and making a stop to avoid decompression sickness (Figure 1). Table 1 lists current

FIGURE 1. Profile of Herbert Nitsch’s record no-limit freedive to 214 m in 4½ min, Spetses, Greece, 2007 (from Lindholm, 2009, courtesy AIDA International, with permission).
world men and women records in eight competitive disciplines. Streeter (2006) described her training, work-up dives, narco-
sis symptoms, operational logistics, and actual freedive for the
2002 No-Limits record to 160 m. She utilized a weighted sled
for the descent and an inflated lift bag for the ascent. Approxim-
ate travel speeds were 1.5–2 m s⁻¹ round trip with a total dive
time of 3 min, 32 s. Competition freedivers are athletes first with
performance being the main objective, but they obviously wish
to perform safely.

We stand to improve freediving performances and learn
from clinical research on the physiological aspects of breath-hold
diving and from marine mammal and bird studies.

**Considerations**

The nitrogen that is available during diving is compressed
in the lungs and would potentially be the cause of narcosis. This
amount of nitrogen is not likely to be evenly distributed to the
body tissues in the short period of freedive time. The body’s div-
ing response vasoconstricts the periphery, forcing the nitrogen to
go mostly to the brain, which potentially results in an elevated
brain PN₂. It may not be possible to absorb a significant amount
of nitrogen from a lung that is severely compressed. The lung
tissue follows the compression of the air, so that the exchange
surface area (normally about 70 m² between the lung blood and
the gas space) is reduced to an extremely small area. That could
support the high-pressure nervous syndrome (HPNS) theory. In
freedives, nitrogen is being taken up on the way down, but very
rapidly through a severely shrinking exchange area. It is also pos-
sible that nitrogen narcosis may be limited due to reduction in
gas uptake through atelectasis (lung collapse).

Nitrogen is soluble, allowing pressures to build up substanc-
tially, and it is possible that a level of hypoxia exists. The loss
of lung volume at depth could allow for mixing venous oxygen
levels. The sensations of nitrogen narcosis and reduced oxygen
saturations can be quite similar, with increased susceptibility po-
tentially aided by higher CO₂ levels. Comparative physiological
knowledge seems to indicate that elephant seals’ pre-dive exhal-
ation strategy occurs, in part, to reduce susceptibility to decom-
pression sickness, shallow-water blackout, and nitrogen narcosis.
Observations of many more dives to these depth ranges would
be needed to form a more conclusive opinion on nitrogen nar-
cosis and decompression sickness in extreme freedivers. Diving
physiology in the twilight zone is complicated. There may also
be a transient hypovolemic (decreased blood volume) effect from
significant blood pooling, which may interfere with adequate
 perfusion in the tissues for a short period.

**Physiology of the Consummate Divers: Marine Mammals
and Diving Birds**

The freediving depth records described above are impressive,
yet even these extreme human performances hardly compare to the
capabilities of the true consummate divers, marine mammals and
diving birds. These animals routinely plummet to great depths for
extended durations (Table 2), yet emerge unscathed by the myriad
physiological conditions with which human divers must contend.
Historical studies on diving animals revealed basic physiologi-
cal and morphological adaptations (e.g., “armored” airways, air
sinus modifications, thermoregulatory features, enhanced oxygen
stores) that contributed toward this aptitude, and recent studies
have shed further light on the remarkable capacity and mecha-
nisms underlying this behavior. Advances in technology, remote
monitoring, and advanced modeling have provided significant in-
sight into the two fundamental issues of diving physiology: oxygen
store management and the effects of pressure at depth.

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**TABLE 1. Current open water and pool world records.**

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<thead>
<tr>
<th>Record type</th>
<th>Male divers</th>
<th>Female divers</th>
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<tr>
<td><strong>Open water</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No limits</td>
<td>214 m: Herbert Nitsch (06/07)</td>
<td>160 m: Tanya Streeter (08/02)</td>
</tr>
<tr>
<td>Variable weight</td>
<td>142 m: Herbert Nitsch (12/09)</td>
<td>122 m: Tanya Streeter (07/03)</td>
</tr>
<tr>
<td>Constant weight</td>
<td>124 m: Herbert Nitsch (04/10)</td>
<td>101 m: Natalia Molchanova (09/09)</td>
</tr>
<tr>
<td>Free immersion</td>
<td>116 m: William Trubridge (04/10)</td>
<td>90 m: Natalia Molchanova (09/09)</td>
</tr>
<tr>
<td>Constant weight without fins</td>
<td>92 m: William Trubridge (04/10)</td>
<td>62 m: Natalia Molchanova (12/09)</td>
</tr>
<tr>
<td><strong>Pool</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Static apnea</td>
<td>11 min, 35 s: Stephane Mifsud (06/09)</td>
<td>08 min, 23 s: Natalia Molchanova (08/09)</td>
</tr>
<tr>
<td>Dynamic apnea</td>
<td>250 m: Alexey Molchanov (10/08)</td>
<td>214 m: Natalia Molchanova (10/08)</td>
</tr>
<tr>
<td>Dynamic without fins</td>
<td>213 m: Tom Sietas (07/08); 213 m: Dave Mullins (08/08)</td>
<td>160 m: Natalia Molchanova (08/09)</td>
</tr>
</tbody>
</table>
TABLE 2. Maximum dive depth and duration records for various diving species (Association Internationale pour le Développement de l’Apnée [AIDA International]; Norris and Harvey, 1972; Watkins et al., 1983; Ridgway, 1986; Le Boeuf et al., 1988; Eckert et al., 1989; Lutcavage et al., 1990, 1992; Thorson and Le Boeuf, 1994; Kooyman and Kooyman, 1995; Mate et al., 1995; Stewart and DeLong, 1995; Ponganis et al., 1997; Kooyman and Ponganis, 1998; Kooyman et al., 1999; Southwood et al., 1999; Noren and Williams, 2000; Ponganis et al., 2003; Hays et al., 2004; Tyack et al., 2006; Ponganis et al., 2007).

<table>
<thead>
<tr>
<th>Species, record type (holder: year)</th>
<th>Depth (m)</th>
<th>Duration (mins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human <em>Homo sapiens</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men: No Limits (Nitsch: 2007)</td>
<td>214</td>
<td>4:30</td>
</tr>
<tr>
<td>Women: No Limits (Streeter: 2002)</td>
<td>160</td>
<td>3:32</td>
</tr>
<tr>
<td>Leatherback turtle <em>Dermochelys coriacea</em></td>
<td>1230</td>
<td>67:18</td>
</tr>
<tr>
<td>Bottlenose dolphin <em>Tursiops truncatus</em></td>
<td>390</td>
<td>8</td>
</tr>
<tr>
<td>Emperor penguin <em>Aptenodytes forsteri</em></td>
<td>564</td>
<td>23:06</td>
</tr>
<tr>
<td>Northern elephant seal <em>Mirounga angustirostris</em></td>
<td>1581</td>
<td>119</td>
</tr>
<tr>
<td>Beaked whale <em>Ziphius cavirostris</em></td>
<td>1888</td>
<td>85</td>
</tr>
<tr>
<td>Sperm whale <em>Physeter macrocephalus</em></td>
<td>2250</td>
<td>138</td>
</tr>
</tbody>
</table>

The diving performance of humans is significantly impacted by the effects of pressure, namely via decompression sickness (DCS), N, narcosis, and high-pressure nervous syndrome (HPNS). Although more commonly associated with breathing compressed air, DCS has been reported in human breath-hold divers, as discussed above. How, then, do the elite divers of the animal kingdom escape the deleterious effects of pressure during their frequent, repetitive, and very deep dives? Several recent reviews detail the morphological and experimental evidence of the adaptations of diving animals to the pressures of diving (Fahlman, 2009; Lemaître et al., 2009; Ponganis, 2011) with modifications such as the absence of air-filled cranial sinuses in seals, middle ear cavities lined with venous plexuses that may engorge with blood at depth, anatomical modifications of the airways and their responses to compression, and enhanced lung surfactants. Scholander’s original model of lung collapse and the cessation of gas exchange at depth has been particularly well supported by both anatomical and experimental evidence in marine mammals; lung collapse undoubtedly plays a role in limiting the accumulation of nitrogen at depth, though the degree and depth of collapse are likely variable and dependent on a variety of factors (Scholander, 1940; Fahlman, 2009; Ponganis, 2011).

Cardiovascular adjustments such as reduced heart rates and decreased cardiac outputs that occur during diving serve not only to conserve oxygen, as discussed below, but also to reduce the accumulation of nitrogen. Theoretical and modeling studies have demonstrated that such responses may play a large role in limiting the accumulation of nitrogen, at least in certain tissues (Fahlman et al., 2006; Lemaître et al., 2009). Dive patterns (depths, durations, and ascent rates) and dive behavior will also dictate the accumulation of nitrogen at depth, though no data exist as to how animals might sense or cognitively influence these types of parameters.

Despite the protective adaptations of diving animals, relatively recent strandings of beaked whales associated with naval sonar exercises and accounts of dysbaric osteonecrosis in sperm whales have heightened interest regarding the effects of pressure on diving animals, as these findings are consistent with, though not diagnostic of, DCS and N, absorption at depth (Jepson et al., 2003; Moore and Early, 2004; Fernandez et al., 2005). If nitrogen loads do reach high levels in diving animals, novel means of dealing with this gas burden may exist in the animal kingdom. Because of the few actual studies of nitrogen pressures in diving animals, more biological data are necessary to further elucidate these adaptations.

Oxygen stores in animals are distributed between the respiratory system, blood, and muscle. It is well documented in the field of diving physiology that accomplished divers have enhanced oxygen stores, mainly attributed to increased blood volumes and increased hemoglobin and myoglobin concentrations. For example, an elephant seal has almost twice the hemoglobin concentration, three times the mass-specific blood volume (Simpson et al., 1970), and 10 to 15 times the myoglobin concentration (Thorson and Le Boeuf, 1994) as compared to a human. Equally as important as these increased oxygen stores, however, are the rate at which oxygen is depleted and hypoxemic tolerance, or lowest level of oxygen that an animal can tolerate. Despite the importance of these latter two parameters, few studies have addressed the rate and magnitude of oxygen depletion while diving.

Another hallmark of diving animals is a redistribution of oxygen stores among the three compartments (Figure 2). Compared to humans, phocid seals have minimized the percentage of oxygen located in the respiratory store, likely an advantage toward the avoidance of nitrogen accumulation and decompression sickness. This fits with the fact that phocids dive upon expiration and undergo lung collapse at depth, as reviewed previously (Fahlman, 2009). Penguins, otariid seals, and cetaceans, however, are thought to dive upon inspiration, and correspondingly have a higher percentage of oxygen stores in the respiratory system (Kooyman and Sinnett, 1982; Ponganis et al., 2010; Skrovan et al., 1999). In line with their increased oxygen stores, diving animals have large percentages of their total oxygen within the blood and muscle (Figure 2).

In discussing the management of oxygen stores in diving animals, specific examples from the literature highlight three central themes: (1) cardiovascular responses, (2) hypoxemic tolerance and the rate of oxygen depletion, and (3) energy saving mechanisms such as a hydrodynamic shape and locomotive strategies.
Since Scholander’s and Irving’s classic findings during forced submersion studies, it has been suggested that reductions in heart rate and peripheral perfusion are the principal determinants of oxygen depletion (Scholander, 1940; Irving et al., 1941; Scholander et al., 1942). This follows from the fact that the organs accounting for approximately half of O$_2$ consumption at rest are either perfusion dependent or directly related to heart rate (Schmidt-Nielsen, 1983; Butler and Jones, 1997; Davis and Kana tous, 1999). Pre- and postdive tachycardias provide optimal loading of O$_2$ before the dive, and heart rate alterations can result in a reduction and redistribution of blood flow during the dive. Most diving animals experience a decrease in heart rate upon submer- sion, the degree varying with species and dive duration (Scholander, 1940; Irving et al., 1941; Butler and Jones, 1997). This can be thought of in terms of an energy conservation strategy, as a reduced heart rate will result in a slower rate of oxygen depletion, consequently yielding increased aerobic dive duration.

A recent investigation that deployed digital electrocardio- gram (ECG) recorders on diving emperor penguins has revealed a particularly extreme heart rate response in this elite avian diver, documenting the highest and lowest measured heart rates for the emperor penguin (Meir et al., 2008). Heart rate decreased to as low as 3 bpm during diving, with periods of bradycardia near 5–6 bpm sustained for over five-minute intervals (Figure 3).

Despite these extreme reductions in heart rate, this diver remains capable of propulsion for active maneuvering and pursuit of prey. The mean heart rate during diving was significantly lower than the ~70 bpm heart rate of a penguin at rest (heart rates of penguins at rest are in the same range as those of humans at rest, with these extreme decreases occurring routinely) (Meir et al., 2008). In contrast, although other diving birds show decreases in heart rate upon submersion, this is only relative to their pre- dive tachycardic values (i.e., not lower than resting heart rate) (Millard et al., 1973; Butler and Woakes, 1984; Enstipp et al., 2001; Froget et al., 2004). Heart rates as high as 256 bpm, the highest value ever measured in this species, were also measured during pre- and post-dive periods (Meir et al., 2008). These extreme highs and lows show the dramatic range of physiological responses of which this animal is capable, while its heart rate at rest hovers around 70 bpm, not unlike that of our own. In all diving animals, heart rate responses can be quite variable, demon- strating the plasticity of oxygen management strategies during diving (Thompson and Fedak, 1993; Andrews et al., 1997; Meir et al., 2008).

Although heart rate responses are indicative of overall oxygen consumption, a more direct approach to understanding the management of oxygen stores is to measure oxygen directly during the dive. Miniaturized microprocessors, biologging instrumentation, and the novel use of an oxygen electrode have recently allowed researchers to document hypoxemic tolerance (the lowest tolerable level of oxygen) and oxygen depletion in this manner in freely diving emperor penguins and elephant seals. This PO$_2$ (partial pressure of oxygen) electrode has been successfully deployed in air sacs and blood vessels in the emperor penguin (Stockard et al., 2005; Ponganis et al., 2007, 2009), and in blood vessels in the elephant seal (Meir et al., 2009). With the recent characterization of the oxygen–hemoglobin dissociation curve for these species (Meir et al., 2009; Meir and Ponganis, 2009), the PO$_2$ profiles can be converted to hemoglobin (Hb)
saturation profiles, providing values for the amount of oxygen present in the system at the start of, throughout, and after the dive.

These studies have demonstrated exceptional hypoxemic tolerance in these species, particularly in the elephant seal. Arterial PO$_2$ values as low as 12 mmHg (6–8% Hb saturation) at the end of the dive were measured in the elephant seal, the lowest PO$_2$ ever measured in a freely diving seal (Meir et al., 2009). This is well below the limits of most other mammals, including those of mountaineers at the brink of human tolerance at the summit of Mt. Everest (Grocott et al., 2009). This value, obtained in a voluntary diving setting with unrestricted access to the surface in the open ocean, is even nearly equivalent to the critical arterial PO$_2$ of seals in forced submersion studies, as defined by EEG criteria marking the threshold of cerebral dysfunction. Venous saturation values at the end of the dive were routinely as low as 2–10 mmHg (0–4% Hb saturation) in these seals (Figure 4A; Meir et al., 2009), and as low as 2–6 mmHg in emperor penguins (Ponganis et al., 2007). Again, these values are well below the limits of humans or mammals at maximal exercise, and even lower than those of the well-documented hypoxemic extremes of horses performing strenuous exercise (Taylor et al., 1987; Bayly et al., 1989; Roca et al., 1989; Manohar et al., 2001). Both species demonstrated an arterialization of the venous oxygen store (Hb saturations > 90%) (Figure 4A). Combined with the near-complete depletion of the venous oxygen store, this illustrates highly efficient optimization of the venous oxygen reserves.

Adaptation at the biochemical level relevant to oxygen management has also been recently revealed in the emperor penguin. The hemoglobin of the emperor penguin has a higher affinity for oxygen than that of other birds (Meir and Ponganis, 2009), similar to that which has been reported for other penguin species and the high-flying bar-headed goose (Lenfant et al., 1969; Milsom et al., 1973; Petschow et al., 1977; Black and Tenney, 1980). This is particularly advantageous for a diving animal that experiences low levels of oxygen while diving, as it implies that more oxygen is available at any given PO$_2$. It also allows for more complete depletion of the significant respiratory oxygen store in this diving bird.

An analysis of results from these studies further highlights the differences in the distribution of oxygen stores in different species. For example, oxygen profiles of diving emperor penguins illustrate that arterial hemoglobin saturation is maintained near 100% throughout most of the dive, declining only in the final portion of the dive when the bird makes its ascent, when ambient pressure also declines (Meir and Ponganis, 2009) (Figure 5). This keeps oxygen levels high for critical organs like the heart and brain, and is consistent with the large respiratory oxygen store in this species, the high-affinity hemoglobin of this species, and ongoing gas exchange from the lungs to the blood. In contrast, arterial hemoglobin saturation values of elephant seals show a continuous decline after the start of the dive, often to lower final values than those of the emperor penguin at the end of dives (Figure 4B; Meir et al., 2009). This is consistent with the fact that the elephant seal has only about 4% of its total oxygen stores in the respiratory system (Figure 2), undergoes lung collapse, and dives upon expiration.

Oxygen depletion patterns in both arterial and venous compartments are highly variable, even for dives of the same duration. As discussed in relation to heart responses, these investigations further support the plasticity of oxygen management strategies during diving.
Energy-Saving Mechanisms

In addition to physiological adaptations and responses, the hydrodynamic shape of marine mammals and diving birds (Fish, 1994) coupled with various locomotor strategies also allows for significant energy and oxygen conservation in these animals. Investigations of various species have shown that diving animals routinely employ efficient swimming speeds (consistently around 1–2 m s\(^{-1}\)) and optimal stroke frequencies while maneuvering through their aquatic habitat (Ponganis et al., 1990; Sato et al., 2007). Burst and glide swimming strategies and the exploitation of buoyancy changes can also result in considerable oxygen savings. It has been estimated that gliding alone could reduce the cost of diving by an average of approximately 28% (Williams et al., 2000).

Discussion

Given our current operational framework and knowledge of human physiology and decompression sickness, we would expect the following elements to receive further consideration: a 90 m for 30 min operational scientific diving window within acceptable risk parameters; rebreather development with a goal toward providing engineering solutions to minimize investment of training time and pre- and post-dive maintenance requirements while enhancing reliability through mass production; replacement of passive thermal protection strategies with the advent of electrically heated gloves, socks, and undergarments; portable diving saturation system development, attainable within the constraints of scientific resources, with advantages that outweigh the lower cost and relative simplicity of bell diving/surface-supplied systems; and operational and physiological limits of wet diving using scuba versus one-man atmospheric diving systems.

Further development of dive computers will better approximate inert gas loads in the diver. Most current units have a dive-profile logging function, downloading capabilities for paperless databases, ascent rate monitors, an air-integration mode, and gas programmability. Benefits from advances in consumer electronics technology could bring the next generation of dive computers high resolution color displays, rechargeable batteries, GPS receivers, underwater communication and navigation, and emergency position-indicating radio beacons (EPIRB). Benefits from monitoring technology integrated into the dive computer algorithm could provide heart rate monitoring, skin temperature and oxygen saturation measurements, and possibly even inert gas bubble detection. Dive computers have for all practical purposes replaced dive tables in scientific diving and it would not be unreasonable to state that regardless of the number of algorithm variations incorporated within them, they all appear to fall within an acceptable window of effectiveness based on available databases of pressure-related injuries. It is also clear that neither tables nor dive computers can eliminate all decompression problems, but when utilized conservatively computers have emerged as an important tool for the improvement of scientific diver safety.

In a recent review of DCS in breath-hold diving, Lemaitre et al (2009) proposed that if marine mammals could sense low levels of bubbles, they could possibly use behavioral or physiological means to reduce the inert gas burden. The future of human diving might entail some type of device that allows for measurement of our own tissue supersaturation and bubble formation. Understanding the specialized adaptations that reduce decompression sickness in marine mammals and how they avoid excessive blood and tissue PN\(_2\) and prevent bubble formation may improve our knowledge of reducing the risk of DCS in human breath-hold divers and scuba divers.

How deep can a human go? Avoiding barotrauma of descent and decompression illness and managing nitrogen narcosis are critical in attaining extreme breath-hold depths. Mental strength, fitness, and exercise to increase elasticity of the ribcage, muscles, and diaphragm are important. Trachea and lungs must be able to withstand a collapse to a residual volume of < 0.5 L. Lindholm (2009) concludes that it is possible for (some) humans to hold their breath for more than 10 minutes or to dive to more than 200 m. Special lenses in fluid-filled goggles can be used instead of a mask to reduce the non-collapsible air spaces, and glossopharyngeal insufflations (lung packing) may be used to increase pulmonary gas stores to over 12 L. Water equalization of the sinuses and middle ear, while painful, is a useful adjunct. Pulmonary edema and partial lung collapse will likely occur. Currently, the limits of deep breath-hold diving seem to be pulmonary barotrauma of descent causing pulmonary edema and nitrogen narcosis incapacitating the diver at depth or causing decompression sickness on ascent.

FIGURE 5. Arterial percent hemoglobin (Hb) saturation and dive profile of four dives of an emperor penguin (modified from Meir and Ponganis, 2009).
Recent advances in technology and continued effort in the arena of the diving physiology of marine mammals and birds have been fruitful, documenting extreme responses beyond what has been traditionally hypothesized for these divers. These insights have implications for a wide variety of topics in diving physiology and physiological ecology, with the potential to redefine previous efforts of estimating oxygen utilization, which depend on accurate assessments of oxygen storage capacity and the extent of depletion while diving. Future work remains in further elucidating the mysteries of the management and exchange of gases in marine mammals and diving birds. A complete understanding of the physiology underlying these phenomenal divers and the mechanisms behind these abilities may also assist in preventing and treating diving-related pathologies in humans, as well as human medical situations involving hypoxic damage including heart attack, stroke, organ transplant, and reperfusion energy.

CONCLUSIONS

The U.S. scientific diving regulatory, medical, training, and operational framework has resulted in a remarkably low decompression sickness incidence rate. In this particular segment of the diving community, diving safety research on dive computers, ascent rates, midday repetitive diving, polar diving, reverse-dive profiles, oxygen-enriched air, and advanced scientific diving contributes to the conservative risk-management approach of scientific diving. Advances in diving medicine allow for mitigation of additional risk while pursuing further understanding of the mechanisms of decompression disease. Freediving sheds light on the extreme capabilities of human performance, approximating the outer limit. Physiological considerations, such as morphological adaptations, and processes, such as management of oxygen stores in diving animals, shed light on the remarkable capacity and mechanisms that enhance diving capabilities in diving animals. Investigations of cardiovascular responses, oxygen depletion, and energy-saving mechanisms have found further evidence in support of the phenomenal capabilities of the consummate divers. The importance of scientific diving as a valuable research tool that places the trained scientist’s eye under water is illustrated by the quantity of research projects reported in this volume that could otherwise not have been performed, with results published in the scientific literature. The peer-review publication process validates the viability and effectiveness of scuba as a research methodology.

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REFERENCES


