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DIVING PHYSIOLOGY

GERALD L. KOOYMAN AND PAUL J. PONGANIS

I. Introduction

At present the human record for an unassisted dive is 122 m during a breath hold which lasted a little less than 4.5 min. Many marine mammals exceed that depth within the first few months of life.

Cuvier's beaked whales (*Ziphius cavirostris*) regularly dive to depths beyond a kilometer, while other premier divers, such as elephant seals (*Mirounga* spp.) and sperm whales (*Physeter macrocephalus*), also occasionally dive to such depths (Ponganis, 2015; Schorr et al., 2014). The spectacular abilities of marine birds and mammals to dive deep and for long periods of time are a source of interest and curiosity for marine scientists and amateurs alike.

II. Adaptations to Hypoxia

A. Oxygen Stores and Their Distribution

An increased total body O_2 store, located in the lungs, blood, and muscle, is considered an essential factor in the breath-hold capacity of diving mammals. The respiratory oxygen store is dependent on lung volume and the concentration of oxygen in the lung at the start of a breath hold. The blood and muscle oxygen stores are dependent on blood volume and muscle mass, and the concentration of the oxygen-binding proteins of hemoglobin in blood, and myoglobin in muscle. From the measurements of myoglobin concentration in the muscles of many species of divers, it is clear that one of the most consistent hallmarks of oxygen storage in all marine mammals that dive to depth is an elevated myoglobin concentration (Kooyman and Ponganis, 1998; Ponganis, 2015). This trait is more characteristic of deep divers than any changes in blood volume, hemoglobin concentration, or respiratory volumes. However, increased blood volume and hemoglobin concentration often contribute significantly to elevated oxygen storage.

As the distribution of oxygen stores vary among species, so do the ranges of the total oxygen store (Table 1). In humans the total store is 20 mL O_2/kg body mass, which is about a fifth of the nearly 100 mL O_2/kg body mass in elephant seals (*Mirounga* spp.). Using the seal as our basic model it is noted that most of its oxygen is in blood and muscle. The large amount relative to terrestrial animals, using the human average as a standard, is a result of a blood volume 3 times, a hemoglobin concentration 1.5 times, and a myoglobin concentration approximately 10 times the human value. In seals, the lung comprises less than 5% of the total oxygen, in part because seals exhale to 50% of their total lung capacity just before diving.

TABLE 1   Common Dive Depths, Common Dive Durations, and the Oxygen Stores of Several Marine Mammal Species.

Species	Depths (m)	Durations (min)	Total oxygen (mL O_2 kg^{-1})	Lung O_2 %	Blood O_2 %	Muscle O_2 %
California sea lion *Zalophus californianus*	<100	<5	55	13	39	48
Harbor seal *Phoca vitulina*	<100	<10	62	7	57	36
Weddell seal *Leptonychotes weddellii*	150–400	10–15	89	4	66	30
Northern elephant seal *Mirounga angustirostris*	200–600	15–30	94	3	71	26
Bottlenose dolphin *Tursiops truncatus*	<50	<2	34	27	33	40
Beluga whale *Delphinapterus leucas*	50–350	9–16	51	17	51	32
Sperm whale *Physeter macrocephalus*	400–900	40–60	81	5	64	30

Adapted from Ponganis (2015). "Diving Physiology of Marine Mammals and Seabirds," Cambridge University Press, Cornwall.

In most other marine mammals, the respiratory oxygen stores may range up to about 25% of the total (Ponganis, 2015).

B. Cardiovascular Responses

The cardiovascular response to breath holding falls into at least two categories of whether the dive is extended or of routine duration for that species. Measurements of cardiovascular and metabolic responses under these circumstances are very limited for any species and most measurements are from seals. Diving mammals are arrhythmic breathers with pauses between each series of breaths. The resting maintenance heart rate is probably most closely reflected in the rate during the respiratory pause or apnea. Using the heart rate during apnea as a basis of comparison for heart rates during a routine dive, the heart rates during many dives are lower than the rate of a resting apneusis, and this occurs despite the fact that the mammal is swimming (for review, see Ponganis, 2015). When an extended dive is performed, the heart rate is even lower than that during routine diving. Because no measurements of blood flow distribution have been directly measured during dives of marine mammals, it is by extrapolation from indirect measures of other organ functions that allude to what may be occurring (Davis, 2014; Davis et al., 1983; Guppy et al., 1986). During routine dives it is likely that gastric, renal, and hepatic functions are reduced by a small amount, but no more than what can be compensated for by higher than normal performance during the short, breathing intervals at the surface. Muscle may utilize a small part of the circulating blood oxygen, but it probably relies on its internal store of oxygen bound to myoglobin for much of aerobic metabolic needs (Davis, 2014; Davis and Kanatous, 1999; Guyton et al., 1995).

Extended dives, those that are 3–5 times the routine dives, are uncommon. They are most likely to occur because of some urgent needs such as a Weddell seal (*Leptonychotes weddellii*) searching for a new hole under sea ice, or an elephant seal hiding at depth to escape notice from a passing pod of killer whales (*Orcinus orca*) near the surface. The cardiovascular response in these extreme cases is a dramatic decrease in heart rate and redistribution of blood flow to obligate aerobic tissues, the most conspicuous of which is the brain (Elsner et al., 1966). There is also a lesser need for transport of oxygen to the heart because of a reduced workload (the slower heart rate) and small store of internal oxygen. In contrast, muscle blood flow can be reduced to a trickle as muscle can draw energy from the large myoglobin-bound oxygen store and its internal anaerobic energy stores, glycogen and creatine phosphate. A reduced blood flow to muscle allows for decreased heart rate, cardiac output, and, hence, the work of the heart and its oxygen consumption. Thus, the degree of muscle blood flow reduction during long and short dives is key to understanding the management of oxygen stores.

C. Metabolic Responses

A decrease in metabolic function in the splanchnic organs can reduce metabolic rate substantially since the function of these organs accounts for nearly 50% of the total resting metabolism of the animal. Similarly, intermittent stroking and prolonged gliding decrease the workload and metabolic demands of locomotory muscle (Williams et al., 2000). In the cold environment at depth, some tissues may also be cooling which results in an additional saving in energy consumption (Butler and Jones, 1997). The final result is a low diving metabolic rate that can be significantly reduced to below the resting levels in extreme dives.

D. Anaerobic Metabolism

Dominating the many factors that affect how long an animal may breath hold is the amount of oxygen available and its rate of utilization. Through oxygen-supported metabolic pathways, 18 times more high energy adenosine triphosphate (ATP) is produced from glucose than through anaerobic processes. Furthermore, carbon dioxide and water, the end products of oxygen-supported metabolism, are less polluting to the cells and circulation than those of anaerobic metabolism. Therefore, the duration of time an animal may breath hold is most strongly affected by the availability of oxygen, with subsidiary support from anaerobic glycolysis and creatine phosphate catabolism. Although an animal may extend its dive considerably by relying on anaerobic glycolysis, the subsequent recovery is in turn extensive because of the time required to process lactic acid and restore the acid base balance of the cells and circulatory system (Kooyman, 1989). For routine dives that occur in sequence over many hours, aerobic metabolism is the only practical option. Oxygen-supported metabolic pathways are also the only means of producing ATP that is derived from catabolism of fat and protein.

It is notable that anaerobic metabolism may play a significant role in some organs. Glycogen concentrations are elevated in both the heart and brain of seals (Czech-Damal et al., 2014; Kerem et al., 1973). Furthermore, in seals, neuroglobin and other markers of oxidative metabolism appear localized in glial cells, but not neurons (Mitz et al., 2009; Schneuer et al., 2012). The mechanisms of the remarkable hypoxemic/ischemic tolerance of seal tissues remain to be fully elucidated (Halasz et al., 1974; Ramirez et al., 2011).

E. Aerobic Diving Limit

The diving mammal in which there has been the most detailed correlation of the diving duration and the postdive blood lactate concentration is the Weddell seal (Fig. 1) (Kooyman et al., 1980; Ponganis et al., 1993; Williams et al., 2004). The source of the
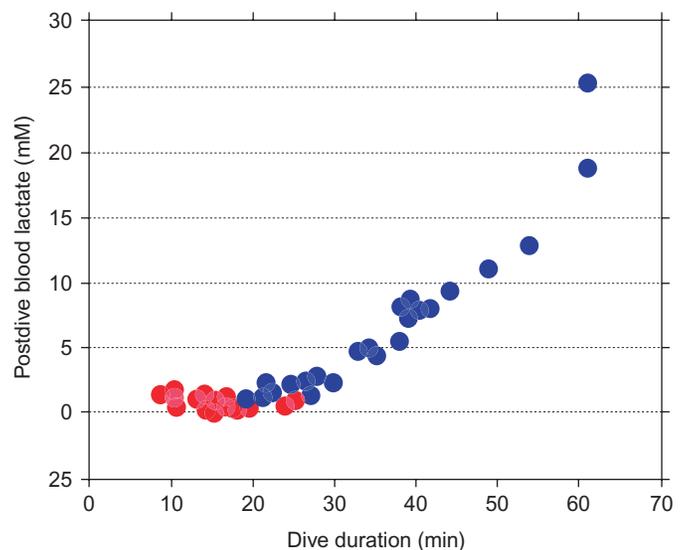

Figure 1 *Peak concentrations of lactate in arterial blood after dives of different duration in adult Weddell seals. The inflection represents the transition from completely aerobic dives, and is considered the aerobic dive limit (ADL) or diving lactate threshold (DLT). Red circles reflect blood values of dives in which there is no net production of lactate, and blue circles are those in which there was a net production.*

lactate is from muscle, in which it accumulates rapidly as muscle oxygen is depleted (Scholander et al., 1942; Williams et al., 2011). After the seal surfaces, there is increased blood flow to muscle and much of the lactate is flushed into the circulation and gradually disappears over several minutes. If the seal should dive again before all of the blood lactate is processed, it will continue to decline over the course of the dive unless that dive exceeds what has been termed the aerobic diving limit (ADL). The ADL is defined as the diving duration beyond which there is a net increase in lactate production, and has been determined by a rise in postdive blood lactate concentration (Kooyman, 1985). From those measurements, it was also shown that with some reasonable accuracy the ADL could be obtained from the quotient of the O_2 store divided by metabolic rate, the calculated ADL (cADL). It has also been proposed that the limit determined by blood lactate measurements be called the diving lactate threshold (DLT) to avoid confusion about the numerous ways that an ADL has been derived since the first measurements were made on Weddell seals (Butler and Jones, 1997).

III. Adaptations to Pressure

Increased hydrostatic pressure is one of the most imposing physical variables to which vertebrates must adjust (Kooyman, 1989). We become especially sensitive to pressure during the most modest dive to depth because our airspaces such as the middle ear and facial sinuses make us acutely aware of any difference between the ambient pressure and our internal pressure. More subtle is the effect of pressure on the lung. For humans the lung is an important oxygen store, but in deep-diving mammals the lung is not an important oxygen store. Over a long period of evolution the main function of the vertebrate lung became the exchange of gases between blood and air. During the descent to depth, this function is diminished in deep-diving marine mammals. As the transfer of gases between the lung and blood slackens or ceases, the rise in nitrogen partial pressure within the lung is not matched in the blood. The lack of gas exchange also results in the avoidance of nitrogen narcosis and oxygen toxicity. Even with this adaptation there is still the pure physical effect of hydrostatic pressure on the nervous system. In terrestrial mammals, pressure causes overstimulation or uncoordinated nerve conduction and dysfunction called high-pressure nervous syndrome (HPNS). How do marine mammals manage to avoid these problems that are manifested in their terrestrial relatives?

The pressure within all airspaces must closely match that of the ambient pressure or suffer damage to the membranes and blood vessels lining the space and a breakdown in normal function. There are at least two major types of airspaces within most mammals that are liabilities for diving (Ponganis, 2015). First are the facial sinuses and middle ear. These rigid cavities have little or no compressibility. A lack of facial sinuses minimizes this problem in seals. Importantly, however, a pressure differential in the middle ears of marine mammals and in facial sinuses of cetaceans is prevented by a complex vascular sinus lining the walls of these airspaces. These vascular structures can become engorged at depth, decreasing the space of the cavity and minimizing pressure differentials.

Second, the largest airspace of all, and potentially the most problematic is the lung. Volume pressure curves of the chest wall and lung of the ribbon seal, *Histriophoca fasciata*, show that both the chest and the lung are nearly limitless in the degree of compression collapse that they can tolerate (Kooyman et al., 1999). This must be so for other diving mammals as well, and in less detailed studies it has been shown that there is exceptional compressibility in other seals, sea lions, and dolphins. Dolphins and other toothed whales show the most extreme modifications within the lung among marine mammals, or any other mammal. Most notable is the reinforcement of peripheral airways, the loss of respiratory bronchioles, and the presence of a series of bronchial sphincters. Sea lions also have robust cartilaginous airway reinforcement extending to the alveolar sac, but there are no bronchial sphincters. In seals there is no cartilage in the terminal airway, but the walls are thickened by connective tissue and smooth muscle, which reduces their compliance to less than that of the alveoli (Denison and Kooyman, 1973; Kooyman, 1973). Hence, during compression the more compliant alveoli collapse first and the gases within these alveoli are squeezed into the upper airway spaces.

These airways enable a graded collapse of the lung to occur during a dive to depth. The result is that most of the lung air is forced into the upper airways where gas exchange with the blood ceases. It has been shown that blood PN_2 in seals only rises slightly, no matter how deep the dive. During simulated dives to depth, the P_aN_2 of the elephant seal, *Mirounga angustirostris*, peaked at 300 kPa and equilibrated to 200 kPa where it was approximately the same as venous PN_2 (Kooyman et al., 1973). This was independent of the ambient pressure from 30 to 136 m (1460 kPa). Similar values were obtained for Weddell seals diving voluntarily to depths as great as 230 m (2400 kPa) (Fig. 2) (Falke et al., 1985). These small increases in P_aN_2 indicate that lung collapse in both species occurred between 20 and 50 m. The early occurrence of lung collapse in seals makes the lung almost useless as an O_2 store, but it limits N_2 absorption during the dive. These N_2 values are below the minimum PN_2 of 330 kPa found to be necessary for bubble formation in cats, and it is assumed that a similar threshold for bubble formation prevails in marine mammals. An additional benefit of early lung collapse is that it eliminates the likelihood of nitrogen narcosis. This condition is often experienced by scuba divers descending to depths greater than 30 m. At these depths, tissue nitrogen level is at least 399 kPa, greater than the P_aN_2 measured in seals. A final thought is the intriguing condition of elephant seals at sea when they spend 90% of their time underwater and at depths greater than 100 m. At these times the lung does not do what it was originally evolved to do, i.e., to exchange gas with the blood and with the atmosphere. Instead, it is collapsed to a solid organ, and the alveoli become unavailable for gas exchange. In sea lions, evidence from chamber and free-diving studies indicates that the depth of complete lung collapse is in the 160–250 m range, much deeper than in the elephant seal and Weddell seal (Kooyman and Sinnett, 1982; McDonald and Ponganis, 2012).

Recent strandings of beaked whales in association with naval sonar exercises have raised questions as to magnitude of gas exchange at depth and the risk of decompression sickness in these animals. Postmortem dissections of some of the beaked whales showed the presence of gas bubbles that appeared to have occurred in vivo (Bernaldo de Quirós et al., 2012; Fernandez et al., 2005; Jepson et al., 2003). We know that these species make extreme dives while hunting for prey (Schorr et al., 2014). Is it possible that gas bubbles could have formed during abnormal diving behavior related to the sonar tests, which resulted in decompression sickness (DeRuiter et al., 2013; Hooker et al., 2012)?

Finally, in humans and other nonaquatic animals descending to depths of more than 100 m and at rates of 100 m/min the mechanical compression on nervous tissue can cause HPNS. The symptoms are modest to severe tremors throughout the body that can become so severe as to be incapacitating. The range of depths and rate of descent are modest compared to that of some deep-diving marine mammals (Ponganis, 2015). Surely a well-adapted mammal does not experience
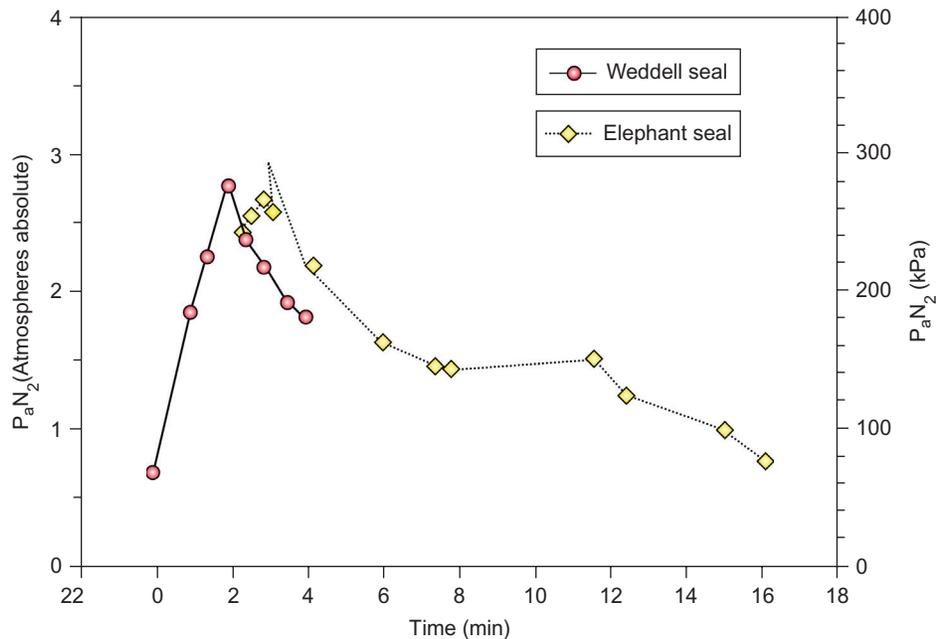
Figure 2 *Arterial N$_2$ tensions in elephant seals and a Weddell seal. The elephant seal submersion (yellow diamonds) was simulated in a water-filled hydraulic pressure chamber to pressures equivalent to a sea water depth of 136 m. Compression began at zero; at 11 min, pressure was released, and, at 14 min, the submersion ended. The Weddell seal (red circles) made a free dive to 89 m under Antarctic sea ice. Maximum depth was reached at 5 min, and the dive ended after 8 min.*

HPNS, which leads to the compelling question of what the difference is between the neural makeup of a marine mammal that protects it from experiencing HPNS and that of a terrestrial mammal that is susceptible to HPNS. We must conclude that the structure of the nervous system is modified in a unique way for a life at high pressure.

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DOLPHINS, PORPOISES, AND MONODONTIDS, EVOLUTION

Rachel Racicot

Crown delphinoids (Odontoceti: Delphinoidea) are a disparate and diverse clade of toothed whales comprising the Delphinidae (~36 extant species of oceanic dolphins), Monodontidae (extant members include the narwhal and beluga), and Phocoenidae (~7 extant species of true porpoises). Recent phylogenetic hypotheses reconstructed using genotypic, phenotypic, or combined datasets support a sister relationship of monodontids and phocoenids, which are subsequently sister to delphinids (e.g., McGowen et al., 2009; Geisler et al., 2011). Delphinoids are supported as sister to a lineage including several "river dolphins" (Iniidae, Pontoporiidae, and Lipotidae). The clade including both Delphinoidea and this "river dolphin" clade (Inioidea) is known as the Delphinida (Muizon 1984, Fig. 1). Molecular divergence estimates suggest that delphinoids diverged from their delphinidan relatives by the early Miocene (~18 Ma), and gave rise to the phocoenids, monodontids, and delphinids. The phocoenid and monodontid lineage diverged approximately 15.5 Ma, with modern phocoenids diverging ~8.4 Ma, and monodontids ~6.28 Ma. Delphinids recently radiated around 10 Ma, with most divergences occurring in the Pliocene-Pleistocene.

The Albireonidae are an extinct, rare lineage of medium-sized (~2.5 m in length) odontocetes that lived in the North Pacific Ocean from at least 9 to 2 Ma (late Miocene to late Pliocene; Barnes 2008). They are often reconstructed in phylogenetic analyses as stem delphinoids, but are here considered to be as the sister group to Inioidea (Fig. 1). Albireonid fossils have been found in the southwestern coast of North America (Baja California, Mexico, and California, USA) and central and northern Japan. The skulls of albireonids are similar in overall shape to Dall's porpoises (*Phocoenoides dalli*), and share many similarities to modern delphinoids, including homodont teeth. They can be diagnosed by a number of characters including a large braincase and dorsally upturned rostral extremity (Fig. 2A). Only the second and third cervical vertebrae are fused, suggesting a somewhat mobile neck. They had a relatively dorsoventrally deep thorax and anteroposteriorly wide pectoral flippers.

Extant phocoenids are relatively small in size (1.4–2.5 m in length) and are antitropically distributed. Diagnostic characters of the phocoenids include the premaxilla not extending posteriorly behind the anterior half of the nares, and spatulate-shaped (rather than conical) teeth. Previously phocoenids and delphinids had been found as sister clades, based on independent phenotype- and genotype-based analyses, but more recent analyses using combined phenotype + genotype and genotype-only datasets find an alliance between Monodontidae and Phocoenidae (Monodontoidea), which is subsequently sister to Delphinidae. Among extant species, *Neophocaena* (finless porpoise) is usually reconstructed as sister to the remaining extant porpoise species in most phylogenetic analyses. Recent analyses using phenotype and genotype data find *Phocoena phocoena* (harbor porpoise) and *P. dalli* (Dall's porpoise) are reconstructed as sister taxa, and this clade is sister to a clade including *P. sinus* (vaquita), *P. dioptrica* (spectacled porpoise), and *P. spinipinnis* (Burmeister's porpoise) with equivocal relationships (McGowen et al., 2009). *P. sinus* is endemic to the Gulf of