Annu. Rev. Physiol. 1998. 60:19–32

THE PHYSIOLOGICAL BASIS OF DIVING TO DEPTH: Birds and Mammals

G. L. Kooyman and P. J. Ponganis
Scripps Institution of Oceanography, University of California, San Diego, La Jolla, California 92093-0204; e-mail: gkooyman@ucsd.edu

KEY WORDS: Aptenodytes, Leptonychotes, Mirounga, oxygen stores, pressure

ABSTRACT

There is wide diversity in the animals that dive to depth and in the distribution of their body oxygen stores. A hallmark of animals diving to depth is a substantial elevation of muscle myoglobin concentration. In deep divers, more than 80% of the oxygen store is in the blood and muscles. How these oxygen stores are managed, particularly within muscle, is unclear. The aerobic endurance of four species has now been measured. These measurements provide a standard for other species in which the limits cannot be measured. Diving to depth requires several adaptations to the effects of pressure. In mammals, one adaptation is lung collapse at shallow depths, which limits absorption of nitrogen. Blood N_2 levels remain below the threshold for decompression sickness. No such adaptive model is known for birds. There appear to be two diving strategies used by animals that dive to depth. Seals, for example, seldom rely on anaerobic metabolism. Birds, on the other hand, frequently rely on anaerobic metabolism to exploit prey-rich depths otherwise unavailable to them.

INTRODUCTION

In the past 10 years, there has been an explosion of information about the diving traits of some aquatic animals, especially penguins, seals, and sea lions. We now know that some seals will dive to depths of nearly 1600 m (1) and that emperor penguins, *Aptenodytes forsteri*, occasionally reach depths of nearly 550 m (2). The deepest dives are not necessarily the longest, and the record 22 min submersion for the emperor penguin (G Robertson, unpublished observations) and

0066-4278/98/0315-0019$08.00

120 min for the elephant seal, *Mirounga leonina* (3), were both well below their record depths. These rare observations represent the results of tens of thousands of recorded dives. The reasons and physiology of such diving extremes are not understood.

Equally important as the extremes are what these animals routinely do. The best examples for comparative purposes are elephant seals, Weddell seals (*Leptonychotes weddellii*), and emperor penguins. Weddell seals, although capable of a maximum dive of at least 82 min (4), usually dive for 10 to 20 min when hunting at depths of 50 to 600 m (5). Elephant seal diving durations last about 20 to 30 min while feeding at depths between 200 and 800 m (3). Emperor penguins hunt for about 2 to 10 min between depths of 50 and 500 m (2).

In this paper, we address the magnitude and distribution of oxygen stores of different divers, the utilization of those stores, and the effects of pressure. Recently, Butler & Jones (6) gave an excellent, extensive review of the physiological responses to breath-holding. However, little was mentioned about the responses of diving to depth and the possible physiological and anatomical adaptations to pressure. Yet, pressure is perhaps as powerful a variable affecting the dive as the submersion and the prerequisite breath-hold. Little is known about pressure because it is such a difficult variable to incorporate into experiments.

Oxygen Stores

An increased total body O_2 store has long been considered an essential factor in the breath-hold capacity of diving birds and mammals. The distribution of O_2 among the respiratory, blood, and muscle compartments is dependent on diving lung volume, blood volume, hemoglobin concentration, myoglobin concentration, and muscle mass. These parameters and calculated O_2 stores are reviewed for both shallow- and deep-diving penguins, pinnipeds, and cetaceans in Tables 1 and 2. Few diving lung volumes and end-of-dive O_2 contents have been measured in species that dive to depth (7–9). It is also debated whether cetaceans, especially large whales, dive at full or partial lung capacity (10). Nonetheless, available data reveal that the respiratory O_2 compartment decreases in magnitude in deep divers. Presumably, this decreases the amount of nitrogen as well as oxygen absorption at depth, and minimizes the associated risks of decompression sickness and nitrogen narcosis. Eighty to 90% of O_2 stores are concentrated in the blood and muscle of these deep divers (Table 2). This is a result of significant increases in blood volume, cell volume, and hemoglobin concentration, as well as myoglobin content. In the emperor penguin, it is primarily a result of an increased myoglobin concentration in the swimming muscles. In fact, the swimming muscles alone contain 19% more O_2 than the entire blood volume of the emperor penguin.

Table 1 Deep versus shallow divers oxygen store determinants

Species	DLV (ml kg^{-1})	BV (ml kg^{-1})	Hb (G 100 ml^{-1})	Muscle mass (% BM)	Mb (g 100 g^{-1})
Adelie penguin	165[a,8]	87[70]	16[70]	40[b]	3.0[c,71]
Emperor penguin	69[a,d]	100[15]	18[15]	38[15]	6.4[c,15]
California sea lion	35[62]	96[31]	18[31]	37[31]	2.7[31]
Weddell seal	27[7]	210[25]	26[25]	35[72]	5.4[25]
Atlantic bottlenose dolphin	81[64]	71[73]	14[73]	30[b]	3.3[74]
Sperm whale	54[e,58]	200[75]	22[10]	34[76]	5.4[63]

[a]No correction for air in feathers.
[b]Assumed.
[c]Pectoral muscle concentration.
[d]PJ Ponganis, unpublished observations.
[e]Lung volume estimates for sperm whales, *Physeter catodon*, are based on measurements from fin whales, *Balaenoptera physalus*. Abbreviations: DLV, diving lung volume; BV, blood volume; Hb, hemoglobin; BM, body mass; mb, myoglobin. Superscript numbers refer to references.

The O_2 storage capacity of the high myoglobin concentrations in seals was demonstrated by Scholander and co-workers in 1942 (11). Since that time, the myoglobin concentration in the muscles of numerous types of divers has been measured. It is now clear that one of the most consistent hallmarks of oxygen storage in all birds and mammals that dive to depth is an elevated myoglobin concentration (6, 12). This trait is more characteristic of deep divers than any changes in blood volume, hemoglobin concentration, or respiratory volumes. Muscle myoglobin concentrations increase proportionately with diving capacities and are highest in penguins, pinnipeds, and cetaceans. Intermediate concentrations are found in shallow-diving, short-duration divers such as manatees,

Table 2 Magnitude and distribution of oxygen stores in penguins, seals, and cetaceans

Species	Body mass (kg)	Total body oxygen (ml O_2 kg^{-1})	Respiratory system %	Blood %	Muscle %
Adelie penguin	5	55	45	29	26
Emperor penguin	25	53	19	34	47
California sea lion	35	39	21	45	34
Weddell seal	400	87	5	66	29
Atlantic bottlenose dolphin	200	36	34	27	39
Sperm whale	10000	77	10	58	34

Based on data in Table 1 and oxygen saturation and extraction assumptions as in Reference 25. Assumed uniform myoglobin concentration except in emperor penguin [swim muscles: 25% body mass, myoglobin 6.4 g 100 g^{-1}; other muscle: 13% body mass, myoglobin 2.1 g 100 g^{-1} (15)]. Body masses for sea lion and sperm whale were those of the animals in blood volume studies (31, 75).

muskrats, beavers, and dabbling ducks. It is notable that myoglobin concentrations are usually higher in primary locomotory muscles (13–15).

Management of Oxygen Stores

Oxygen store calculations are based on assumptions that require further evaluation. The magnitude of the blood O_2 store is dependent on the arterial/venous blood volume distribution and on the degree of hemoglobin desaturation during diving. The distribution of blood between the large spleens (16–18) and equally capacious hepatic sinuses and abdominal vasculature (19–21) of phocid seals potentially affects both of these parameters. Although the spleen of Weddell seals expands rapidly after isolated dives (17), it does not change fast enough to significantly affect hematocrit during repetitive, foraging dives when surface intervals are 2–4 min (17, 22, 23). In addition, the O_2 saturation status of the splenic effluent is not known. Even if the spleen concentrates fully saturated red cells during each surface period, under current assumptions regarding arterial extraction and venous O_2 depletion during diving, there is not a significant increase in the calculated blood O_2 store (18).

There have also been few studies of the degree of blood O_2 store depletion during diving. A single arterial P_{O_2} sample prior to the end of a 27-min dive of a Weddell seal corresponded to a 28% hemoglobin saturation (23, 24). End-tidal P_{O_2} values during the first post-dive breaths of a free-diving Weddell seal were 3.06 kPa (23 torr) to 1.7 kPa (13 torr) after dives of 21 to 32 min (25). These correspond to a hemoglobin saturation of no less than 45 to 20%, respectively, assuming a normal pH. Actual arterial P_{O_2} is probably lower because of the usual difference in arterial-alveolar oxygen tensions. For seals, these data justify the use of the 20% end-arterial saturation used in O_2 store calculations. A similar assumption for sea lions, cetaceans, and birds is less certain.

The diving durations during which O_2 stores allow metabolism to be primarily aerobic have been assessed by the measurement of post-dive blood lactate concentrations (Figure 1). This is based on Scholander et al's findings (11) that significant muscle lactate accumulation does not occur until the muscle O_2 store is depleted. The aerobic dive limit (ADL), defined as the diving duration beyond which blood lactate concentration increases above resting levels (26), was first measured in adult and juvenile Weddell seals (27, 28). It is the diving duration beyond which there is a net increase in lactate production. ADLs have now also been measured in Weddell seal pups; Baikal seals, *Phoca sibirica*; California sea lions, *Zalophus californianus*; and emperor penguins (29–32). We consider this post-dive lactate accumulation secondary to localized O_2 store depletion and glycolysis in some tissue in the body, most likely in the propulsive muscles. It is important to emphasize that the ADL does not imply exhaustion of all O_2 stores for that dive duration. The rates and magnitude of O_2 store

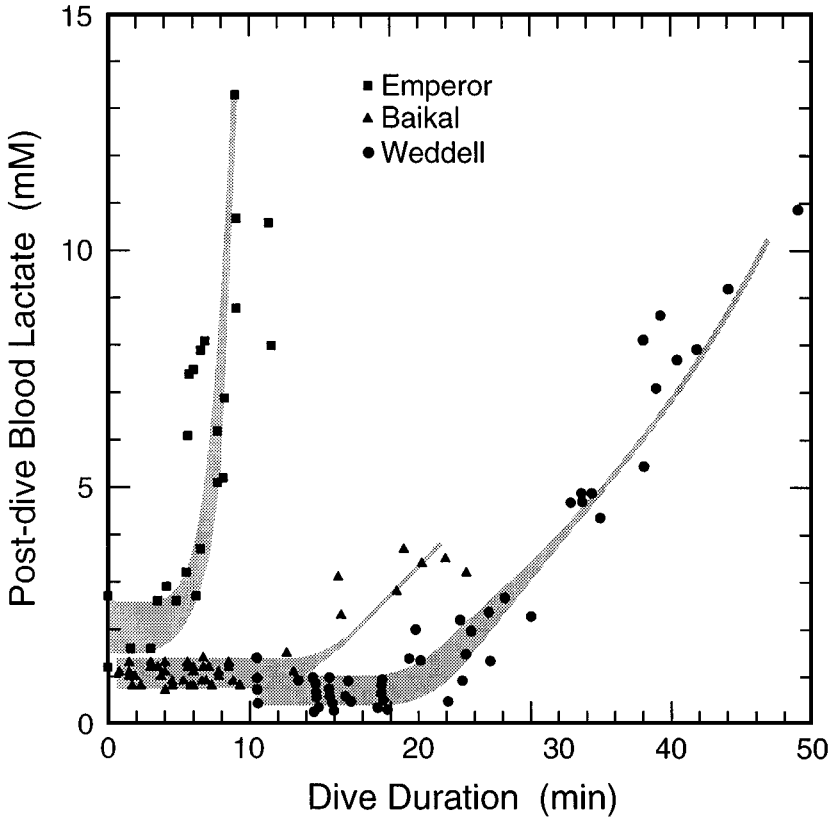

Figure 1 Post-dive blood lactate concentrations and diving durations of the emperor penguin, Baikal seal, and Weddell seal. Lactate concentrations are from whole blood of Weddell seals and emperor penguins (27, 32) and from plasma of Baikal seals (30).

depletion and lactate production at known tissue site(s) require further research in free-diving animals.

In adult Weddell seals and in Baikal seals, the measured ADL (Figure 1) can be predicted by dividing the body O_2 stores by the average diving metabolic rate (25, 27, 32). This metabolic rate is the so-called event metabolic rate, i.e. total O_2 consumed during the surface interval (surface interval duration + dive duration) (4). A calculated ADL has been applied to many species in the analysis of diving durations at sea (6, 29, 33). Although this is useful to the ecologist or behaviorist in the evaluation of foraging theory, there are inaccuracies because only estimates of diving metabolic rate and O_2 stores are available in many

species. We also question whether doubly-labeled water-determined metabolic rates of entire foraging trips to sea represent accurate estimations of diving metabolic rate. Such estimations have led to a recent calculation of a 10-min ADL in southern elephant seals (33). We consider this value highly improbable, as did the authors who were testing a null hypothesis that calculated ADLs can result in close approximations of the measured value. Although they rejected the hypothesis, it may have been for unwarranted reasons. The oxygen consumption rate used was four times resting, which is almost equivalent, on a relative basis, to that of a Tour de France cyclist (34). It is near the upper limit of sustained metabolic scope (34). It is also at least twice the measured diving oxygen consumption of a Weddell seal (4, 25). Underwater observations of seals, sea lions, and penguins suggest to us that diving animals do not work at high oxygen consumption rates. It is also necessary to emphasize that although this ADL calculation may predict the onset of increased post-dive lactate concentration, it does not imply that all O_2 stores are depleted at that time, nor that the event metabolic rate is the actual O_2 store depletion rate during the diving period. Moreover, event metabolic rates vary within a species. Those measured in Weddell seals correlate inversely with dive duration, are often highest during very short, possibly recovery dives after long dives, and are higher in foraging dives than in exploratory dives (4, 25).

Two especially important factors controlling the duration of a dive are the oxygen store depletion rates and the lowest tolerable level of the blood oxygen store. Only a few arterial O_2 depletion rates are available from isolated dives of Weddell seals (24). Venous O_2 store depletion and myoglobin desaturation in the primary locomotory muscles have not been examined. The kidneys, splanchnic organs, and heart account for about 40% of resting oxygen consumption. Metabolic rate of the heart is directly related to heart rate, and renal and hepatic O_2 consumption are perfusion dependent. Therefore, primary determinants of the rate of blood O_2 store depletion should be changes in heart rate and accompanying changes in renal and splanchnic blood flow. Recent advances in technology have allowed examination of some of these factors. The bradycardia during dives to depth is variable, may be as extreme as bradycardia during forced submersions, and is probably dependent on the nature of the dive (31, 35–42). During repetitive foraging dives of Weddell seals, hepatic and renal clearances are maintained (36), whereas in prolonged or stress dives, they are reduced (36, 43).

The degree of muscle perfusion during diving also strongly affects both the blood and muscle O_2 stores. The desaturation rate of myoglobin is dependent on the level of muscle perfusion during diving and on the energy demands of muscle, which vary between locomotory and nonlocomotory muscle. These

factors make both the measurement and interpretation of such data complex (44). Metabolic rate in the primary locomotory muscles is indirectly dependent on swim speed and directly dependent on flipper or fluke stroke effort: stroke frequency and amplitude. Even though swim velocity remains constant, stroke effort may decrease significantly during descent in deep dives secondary to changes in buoyancy with depth. Measurement of blood flow in exercising muscles during shallow submersions of tufted ducks showed an elevation in perfusion over resting levels (45). However, similar measurements in animals diving to depth have been an elusive goal not yet attained.

Several significant observations on muscle energy metabolism have been made in a magnetic resonance study of Pekin ducks, *Anas platyrhynchos* (46). These findings included (*a*) no reduction in resting muscle ATP turnover rate during forced submersion, (*b*) partial maintenance of muscle ATP turnover rate by a reduction in creatine phosphate concentration, and (*c*) an increase in lactate concentration sufficient to maintain ATP turnover rates. Although ion channel arrest and a reduction in glycolytic rate (reversed Pasteur effect) have been postulated as mechanisms of tissue hypometabolism in diving animals (43,47), and occur to some degree in anoxic turtle brain and liver preparations (48,49), these processes are not apparent in muscle of the Pekin duck.

The contribution of creatine phosphate to muscle energy metabolism is relevant to the original arguments for tissue hypometabolism made by Scholander et al (11). In their examination of muscle oxygen consumption and lactate production during forced submersions of harbor seals, *Phoca vitulina*, hypometabolism appeared to occur during the second 5-min interval of the submersion. In this 5-min period when the oxygen store was depleted and significant lactate production began, there was almost a 50% decrease in metabolic rate (based on measured oxygen depletion and lactate accumulation). In terms of ATP turnover, it was 1.3 mmol ATP kg^{-1} muscle min^{-1} or about 6.5 mmol kg^{-1} muscle over 5 min. However, if creatine phosphate breakdown contributes to ATP synthesis during that interval, there need be no shortfall in ATP turnover rate. Because creatine phosphate concentration is 15–20 mmol kg^{-1} in mammals, the concentration of creatine phosphate need only decrease by one third in order to maintain a constant metabolic rate. Thus there appears to be no biochemical evidence for muscle hypometabolism during forced submersions of either birds or mammals. It should be noted that those authors were aware of creatine phosphate's potential contribution; however, they did not consider it in their calculations because of a lack of available data at that time (50).

Body temperature may also affect oxygen store utilization. Metabolic rate reduction may be either a result or a cause of changes in temperature. Decreased

body temperatures during forced submersions of harbor seals were originally attributed to about a 50% reduction in resting metabolic rate (50). However, in their calculation, it was assumed that 200-ml O_2 min^{-1} = 10 kcal min^{-1}, but 200-ml O_2 min^{-1} = 0.96 kcal min^{-1} (51), not 10 kcal min^{-1}. Correction of this factor results in a required 400-ml O_2 min^{-1} metabolic rate reduction in a seal with a 200-ml O_2 min^{-1} resting/presubmersion metabolic rate. Although severe bradycardia and organ hypoperfusion may decrease metabolic rate during the forced submersion, such a large change is unlikely, if not impossible. Therefore, hypometabolism alone cannot account for such large and rapid temperature declines. Other mechanisms must be sought. Body temperatures are a complex result of heat production, heat conduction, and peripheral heat loss. Vascular adaptations may play a significant role in thermoregulation (52). The recording of body temperature changes during diving is just now beginning. The role of metabolic rate, cold prey ingestion, insulation, alterations in peripheral heat loss mechanisms, and high convective losses during swimming will require thorough investigation before the mechanisms and effects of such temperature changes are understood.

Effects of Pressure

Much has been learned about the behavioral traits of divers in the past 10 years. However, little new information has been reported about the effects of pressure on diving birds and mammals during this same period of time. The physiological state of animals at depth will remain a mystery until new kinds of instrumentation become available to the field physiologist.

There are several possible adverse effects of diving to depth. Four of the most notable are (*a*) mechanical distortion or compression of tissues, especially gas-filled spaces; (*b*) higher gas tensions in the lung that result in increased gas absorption into tissues; (*c*) dissolved gas tensions that may be higher than ambient pressure during ascent, which results in the potential for gas bubble formation in blood and tissues; and (*d*) at depth the diver is a long way from its most precious resource, oxygen.

Distortion of the chest requires flexibility in the bones and tissue of the chest to prevent pressure differentials with the resulting rupturing of membranes. Concomitantly with the compression of the air space, the lung gas tensions rise and are more readily absorbed into the blood and into the blood-perfused tissues. The inability to compensate for these effects may result in nitrogen narcosis if the blood and tissue nitrogen tensions become too high. The mechanical compression of nervous tissue can cause a condition called high-pressure nervous syndrome that is manifested in humans and other non-aquatic animals at depth equivalents of about 150 m (53–55) if the rate of descent is about 100 m min^{-1}, the equivalent rate of a marine diver. This is not of much concern for

many species of divers that do not descend to such great depths. However, for those species that commonly dive into this region, we might surmise that the structure of the nervous system has modifications adapted for a life exposed to such high pressure.

Lungs are a liability for deep divers because, in contrast to the muscle and blood discussed above, they are a better nitrogen store than oxygen store. Perhaps this is one of the reasons the lungs of marine mammals are the most structurally modified of any mammalian group (56, 57). Volume pressure curves of the chest wall and lung of the ribbon seal, *Phoca fasciata*, show that both are nearly limitless in the degree of compression collapse they can tolerate (58). This must be so for other diving mammals as well, but there are few observations to verify this. Compressibility of the chest and lungs has been qualitatively or quantitatively shown also for dolphins (58–60) and for other seals and sea lions (9, 61, 62).

The toothed whales, especially small dolphins, show the most extreme modifications within the lung among marine mammals, or any other mammal. The most notable examples are the reinforcement of peripheral airways, the loss of respiratory bronchioles, and the presence of a series of bronchial sphincters (56). Sea lions also have robust cartilaginous airway reinforcement extending to the alveolar sac, but there are no bronchial sphincters. In seals, the terminal airway is fortified by connective tissue and smooth muscle but not cartilage (56).

It has been proposed that the pinniped and cetacean 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 is blunted (7, 63). Radiographic evidence in Weddell seals during forced submersion and compression to 300 m supports this hypothesis (9). Other functions are also possible. The exceptional shortening and thickness of peripheral airways in sea lions and dolphins may meet the more pedestrian need of moving air out of the lung at high rates so that the animal may catch a breath within the short interval when it passes through the air/water interface at high speed (59, 64).

In the Weddell seal and the California sea lion, the measured total lung capacity is about twice the diving lung volume (62, 65). From Table 1 it is inferred that some, if not all, pinnipeds dive with a lung volume well below capacity. Nevertheless, because of the depth and duration of dives, blood and tissue PN_2 could rise substantially if no airway structural adaptations existed. How low PN_2 actually remains during a deep dive was shown in several experiments.

The most extreme case was the forcible submersion in a compression chamber of elephant seals and a Weddell seal (7). Because N_2 tension in blood and tissue is dependent upon the distribution of circulating blood, in addition to depth and duration of the dive, the restricted blood flow during forced submersion would

concentrate the N_2 absorbed in the lungs and raise the PaN_2 to the highest possible level. Under these conditions, the PaN_2 of elephant seals peaked at 300 kPa and equilibrated to 200 kPa, where it was approximately the same as venous PN_2 (7). This was independent of the ambient pressure from 37 to 138 m (1480 kPa). Similar values were obtained for Weddell seals diving voluntarily to depths as great as 230 m (2400 kPa) (61). The PaN_2 never exceeded 323 kPa. These modest increases in PaN_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, whereas it limits N_2 absorption during the dive. Furthermore, the muscle tissue PN_2 measurements in bottlenose dolphins, *Tursiops truncatus*, diving to depths of 100 m never rose above 213 kPa. It was concluded that lung collapse probably occurred at about 70 m (66). All these reported N_2 values are below the minimum PN_2 of 330 kPa found to be necessary for bubble formation in cats (67).

Adelie penguins, *Pygoscelis adeliae*, and gentoo penguins, *P. papua*, dive with a large gas store that in relation to body mass is five to six times that of a diving seal (8). During simulated dives to 68 m (8), blood N_2 tensions in Adelie penguins were slightly higher than those measured in elephant seals. These levels are not dangerously high, and natural diving durations are shorter than were those experimental dives. Therefore, the shortness and shallowness of dives in these species would seem to prevent high blood PN_2 (8).

However, during routine deep dives of emperor and king penguins, ambient pressures may reach 3000 to 5000 kPa. During such a deep dive to 450 m by an emperor penguin, for example, the total time below 100 m is about 8.5 min. This weakens the premise, at least for king and emperor penguins, that the main protection of diving birds is short, shallow dives (8). Furthermore, the ascent rates from deep dives may exceed 100 m/min. This is probably too fast to allow for adequate N_2 elimination if much had been absorbed earlier in the dive.

If tissue nitrogen uptake is simply the result of the differential between ambient and tissue partial pressures, it is possible to show by traditional decompression theory that tissue nitrogen pressures could become a serious liability during serial dives to depth (68). Although the long recoveries after extended deep dives of emperor penguins are more likely driven by a metabolic acidosis resulting from a substantial O_2 debt, an additional value is that the bird remains at or near the surface. During that time absorbed nitrogen might wash out through the lungs without injuring the bird. Also, any shallow dives within the interval before the next deep dive might act as decompression stops and reduce the decompression risk as long as the tissue N_2 remains in solution (68, 69). Like the mysteries of high flying birds, those of deep divers need further study in order to develop suitable gas exchange models.

Concluding Remarks

Efficacy of a marine diver is based on the dual adaptations to pressure and hypoxia. This is different from freshwater divers, most of which are basically surface breath-holders. The adaptations manifested for diving to depth range from anatomical modifications of the respiratory system to elevation in muscle oxygen stores. Only recently have models been developed that might explain some of the peculiar anatomical and physiological properties of these animals. No doubt many more are to follow.

We speculate that there are at least two types of deep divers: Those that rely on aerobic metabolism the great majority of the time, and those that often push this limit and make frequent use of anaerobic glycolysis. Weddell and elephant seals fall into the former category. By virtue of their large oxygen stores and size-related low metabolic rate, they are able to conduct most of their daily diving routines while relying very little on anaerobic glycolysis. On occasional dives, however, intense bradycardia and peripheral vasoconstriction will conserve the large blood oxygen store for metabolism by the central nervous system. Anaerobic conditions will eventually prevail in muscle and other peripheral tissues, resulting in lactate accumulation. Sea lions and dolphins, which rely more on speed in their underwater forays, will have higher muscle metabolic demands and therefore shorter time until muscle exhaustion. Consequently, blood oxygen stores have not been enhanced to the level of seals. Furthermore, the oxygen requirements of the large brain of dolphins are an additional handicap that limit the breath-hold tolerance of this group. Finally, penguins with their small brains and massive propulsive muscles have opted for large oxygen stores in the muscle to help maintain rapid swimming while they often push the limits of those stores. By virtue of their mass-related high metabolic rate, they must frequently rely on anaerobic metabolism in the muscle as the oxygen store depletes when they exploit depths that would otherwise be unavailable to them. Emperor and king penguins are the best examples of this strategy.

There are currently few data to support the hypotheses in the previous paragraph, but as we move into the next century, one of the goals of diving physiology should be to determine if there are distinctive oxygen store management strategies among deep divers. Those strategies might be (*a*) large brain/large circulating stores/large muscle stores for infrequently exceeding their ADL (seals); (*b*) very large brain/normal circulating stores/moderate muscle stores for seldom exceeding their ADL (dolphins); (*c*) large brain/normal circulating stores/moderately elevated muscle stores for seldom exceeding their ADL (sea lions, fur seals); and (*d*) small brain/normal circulating stores/large muscle stores for frequently exceeding their ADL (penguins).

ACKNOWLEDGMENTS

Preparation of this review was supported by the National Science Foundation, Office of Polar Programs grant 9219872. We thank R Howard for reviewing the manuscript and his many helpful suggestions.

Literature Cited

1. Stewart BS, DeLong RL. 1993. Postbreeding foraging migrations of northern elephant seals. In *Elephant Seals: Population, Ecology, Behavior and Physiology*, ed. BJ Le Boeuf, RM Laws, pp. 290–309. Berkeley: Univ. Calif. Press
2. Kooyman GL, Kooyman TG. 1995. Diving behavior of emperor penguins nurturing chicks at Coulman Island, Antarctica. *Condor* 97:536–49
3. Hindell MA, Slip DJ, Burton HR, Bryden MM. 1991. Physiological implications of continuous, prolonged, and deep dives of southern elephant seals. *Can. J. Zool.* 70:370–79
4. Castellini MA, Kooyman GL, Ponganis PJ. 1992. Metabolic rates of freely diving Weddell seals: correlations with oxygen stores, swim velocity, and diving duration. *J. Exp. Biol.*165:181–94
5. Castellini MA, Davis RW, Kooyman GL. 1992. *Diving Behavior of the Weddell Seal: Annual Cycles,* Vol. 28. Berkeley: Univ. Calif. Press. 54 pp.
6. Butler PW, Jones DR. 1997. The physiology of diving of birds and mammals. *Physiol. Rev.* 77:837–99
7. Kooyman GL, Schroeder JP, Denison DM, Hammond DD, Wright JJ, Bergman WD. 1972. Blood N_2 tensions of seals during simulated deep dives. *Am. J. Physiol.* 223:1016–20
8. Kooyman GL, Schroeder JP, Greene DG, Smith VA. 1973. Gas exchange in penguins during simulated dives to 30 and 68 m. *Am. J. Physiol.* 225:1467–71
9. Kooyman GL, Hammond DD, Schroeder JP. 1970. Bronchograms and tracheograms of seals under pressure. *Science* 169:82–84
10. Ridgway SH. 1986. Diving by cetaceans. In *Diving in Animals and Man*, ed. AO Brubakk, JW Kanwisher, G Sundnes, pp. 33–62. Trondheim: Royal Nor. Soc. Sci. Lett.
11. Scholander PF, Irving L, Grinnell SW. 1942. Aerobic and anaerobic changes in seal muscle during diving. *J. Biol. Chem.* 142:431–40
12. Kooyman GL, Ponganis PJ, Howard RS. 1997. *Diving Animals*. New York: Dekker. In press
13. MacArthur RA. 1990. Seasonal changes in the oxygen storage capacity and aerobic dive limits of the muskrat (*Ondatra zibethicus*). *J. Comp. Physiol. B* 160:593–99
14. Petrov EA, Shoshenko KA. 1987. Total store of oxygen and duration of diving of the Nerpa. In *Morphology and Ecology of Fish*, ed. GI Galazii, pp. 110–28. Novbosibirsk: Acad. Sci. Russia, Siberian Div.
15. Ponganis PJ, Costello ML, Starke LN, Mathieu-Costello O, Kooyman GL. 1997. Structural and biochemical characteristics of locomotory muscles of emperor penguins, *Aptenodytes forsteri*. *Respir. Physiol*. In press
16. Bryden MM, Lim GHK. 1969. Blood parameters of the southern elephant seal (*Mirounga leonina*) in relation to diving. *Comp. Biochem. Physiol.* 28:139–48
17. Hurford WE, Hochachka PW, Schneider RC, Guyton GP, Stanek KS, et al. 1996. Splenic contraction, catecholamine release, and blood volume redistribution during diving in the Weddell seal. *J. Appl. Physiol.* 80:298–306
18. Ponganis PJ, Kooyman GL, Sartoris D, Jobsis PF. 1992. Pinniped splenic volumes. *Am. J. Physiol.* 262:R322–25
19. Elsner RW, Scholander P F, Craig AB, Dimond EG, Irving L, et al. 1964. A venous blood oxygen reservoir in the diving elephant seal. *Physiologist* 7:124
20. Harrison RJ, Tomlinson JDW. 1956. Observations on the venous system in certain Pinnipedia and Cetacea. *Proc. Zool. Soc. London* 126:205–33
21. Ronald K, McCarter R, Selley LJ. 1977. Venous circulation of the Harp seal (*Pagophilus groenlandicus*). In *Functional Anatomy of Marine Mammals*, ed. RJ

Harrison, pp. 235–70. New York: Academic
22. Castellini MA, Davis RW, Kooyman GL. 1988. Blood chemistry regulation during repetitive diving in Weddell seals. *Physiol. Zool*. 61:379–86
23. Qvist J, Hill RD, Schneider RC, Falke KJ, Liggins GC, et al. 1986. Hemoglobin concentrations and blood gas tensions of free-diving Weddell seals. *J. Appl. Physiol*. 61:1560–69
24. Qvist J, Weber RE, Zapol WM. 1981. Oxygen equilibrium properties of blood and hemoglobin of fetal and adult Weddell seals. *J. Appl. Physiol*. 50:999–1005
25. Ponganis PJ, Kooyman GL, Castellini MA. 1993. Determinants of the aerobic dive limit of Weddell seals: analysis of diving metabolic rates, post-dive end tidal Po_2s, and blood and muscle oxygen stores. *Physiol. Zool*. 66:732–49
26. Kooyman GL. 1985. Physiology without restraint in diving mammals. *Mar. Mamm. Sci*. 1:166–78
27. Kooyman GL, Wahrenbrock EA, Castellini MA, Davis RW, Sinnett EE. 1980. Aerobic and anaerobic metabolism during diving in Weddell seals: evidence of preferred pathways from blood chemistry and behavior. *J. Comp. Physiol*. 138:335–46
28. Kooyman GL, Castellini MA, Davis RW, Maue RA. 1983. Aerobic dive limits in immature Weddell seals. *J. Comp. Physiol*. 151:171–74
29. Burns JM, Castellini MA. 1996. Physiological and behavioral determinants of the aerobic dive limit in Weddell seal (*Letonychotes weddellii*) pups. *J. Comp. Physiol. B* 166:473–83
30. Ponganis PJ, Kooyman GL, Baronov EA, Thorson PH, Stewart BS. 1997. The aerobic submersion limit of Baikal seals, *Phoca sibirica*. *Can. J. Zool*. In press
31. Ponganis, PJ, Kooyman GL, Winter LM, Starke LN. 1997. Heart rate and plasma lactate responses during submerged swimming and diving in California seal lions (*Zalophus californianus*). *J. Comp. Physiol. B* 167:9–16
32. Ponganis PJ, Kooyman GL, Starke LN, Kooyman CA, Kooyman TG. 1997. Post-dive blood lactate concentrations in emperor penguins, *Aptenodytes forsteri*. *J. Exp. Biol*. 200:1623–26
33. Boyd IL, Croxall JP. 1996. Dive durations in pinnipeds and seabirds. *Can. J. Zool*. 74:1696–705
34. Peterson CC, Nagy KA, Diamond J. 1990. Sustained metabolic scope. *Proc. Natl. Acad. Sci. USA* 87:2324–28
35. Bevan RM, Boyd IL, Reid K, Woakes AJ, Croxall JP. 1997. Heart rates and abdominal temperatures of free-ranging south Georgian shags, *Phalacrocorax georgianus*. *J. Exp. Biol*. 200:661–75
36. Davis RW, Castellini MA, Kooyman GL, Maue R. 1983. Renal GFR and hepatic blood flow during voluntary diving in Weddell seals. *Am. J. Physiol*. 245:743–48
37. Hill RD, Schneider RC, Liggins GC, Schuette AH, Elliott RL, et al. 1987. Heart rate and body temperature during free diving of Weddell seals. *Am. J. Physiol*. 253:R344–51
38. Fedak MA, Pullen MR, Kanwisher J. 1988. Circulatory responses of seals to periodic breathing: heart rate and breathing during exercise and diving in the laboratory and open sea. *Can. J. Zool*. 66:53–60
39. Fedak MA, Thompson D. 1993. Behavioural and physiological options in diving seals. *Symp. Zool. Soc. London* 66:333–48
40. Kooyman GL, Ponganis PJ, Castellini MA, Ponganis EP, Ponganis KV, et al. 1992. Heart rates and swim speeds of emperor penguins diving under sea ice. *J. Exp. Biol*. 165:161–80
41. Butler PJ. 1994. To what extent can heart rate be used as an indicator of metabolic rate in free-living marine mammals? In *Marine Mammals: Advances in Behavioural and Population Biology*, ed. IL Boyd, 66:317–32. London: Zool. Soc. London
42. Elsner R, Wartzok D, Sonafrank NB, Kelly BP. 1989. Behavioral and physiogical reactions of arctic seals during under-ice pilotage. *Can. J. Zool*. 67:2506–13
43. Guppy M, Hill RD, Liggins GC, Zapol WM, Hochachka PW. 1986. Microcomputer assisted metabolic studies of voluntary diving of Weddell seals. *Am. J. Physiol*. 250:175–87
44. Guyton, GP, Stanek KS, Schneider RC, Hochachka PW, Hurford WE, et al. 1995. Myoglobin-saturation in free-diving Weddell seals. *J. Appl. Physiol*. 79:1148–55
45. Bevan RM, Butler PJ. 1992. Cardiac output and blood flow distribution during swimming and voluntary diving of the tufted duck (*Aythya fuligula*). *J. Exp. Biol*. 1668:199–217
46. Stephenson R, Jones DR. 1992. Metabolic responses to forced dives in Pekin duck measured by indirect calorimetry and ^{31}P-MRS. *Am. J. Physiol*. 262:R1309–17
47. Hochachka PW, Guppy M. 1987. *Metabolic Arrest and the Control of*

Biological Time. Cambridge, MA: Harvard Univ. Press
48. Land SC, Sanger RH, Smith PJS. 1997. O_2 availability modulates transmembrane Ca^{2+} flux via second-messenger pathways in anoxia-tolerant hepatocytes. *J. Appl. Physiol*. 82:776–83
49. Lutz PL, Nilsson GE. 1997. Contrasting strategies for anoxic brain survival—glycolysis up or down. *J. Exp. Biol*. 200: 411–19
50. Scholander PF, Irving L, Grinnell SW. 1942. On the temperature and metabolism of the seal during diving. *J. Cell. Comp. Physiol*. 19:67–78
51. Schmidt-Nielsen K. 1983. *Animal Physiology: Adaptation and Environment*. Cambridge: Cambridge Univ. Press. 619 pp. 3rd ed.
52. Rommell SA, Pabst DA, McLellan WA, Williams TM, Friedl WA. 1994. Temperature regulation of the testes of the bottlenose dolphin (*Tursiops truncatus*): evidence from colonic temperatures. *J. Comp. Physiol. B* 164:130–34
53. Bennett PB. 1975. The high pressure nervous syndrome: man. In *The Physiology and Medicine of Diving and Compressed Air Work*, ed. PB Bennett, DH Elliott, pp. 248–63. Baltimore: Williams & Wilkins
54. Brauer RW. 1975. The high pressure nervous syndrome: animals. In *The Physiology and Medicine of Diving and Compressed Air Work*, ed. PB Bennett, DH Elliott, pp. 231–47. Baltimore: Williams & Wilkins
55. Kooyman GL. 1989. *Diverse Divers: Physiology and Behavior. Zoophysiology Series*, ed. K Johansen, DS Farner, Vol. 23. New York: Springer-Verlag. 216 pp.
56. Belanger LF. 1940. A study of the histological structure of the respiratory portion of the lungs of aquatic mammals. *Am. J. Anat*. 67:437–61
57. Denison DM, Kooyman GL. 1973. The structure and the function of the small airways in pinniped and sea otter lungs. *Respir. Physiol*. 17:1–10
58. Leith D. 1976. Comparative mammalian respiratory mechanics. *Physiologist* 19: 485–510
59. Kooyman GL, Sinnett EE. 1979. Mechanical properties of the harbor porpoise lung. *Respir. Physiol*. 36:287–300
60. Ridgway SH, Scronce BL, Kanwisher J. 1969. Respiration and deep diving in the bottlenose porpoise. *Science* 166:1651–54
61. Falke KJ, Hill RD, Qvist J, Schneider RC, Guppy M, et al. 1985. Seal lungs collapse during free diving: evidence from arterial nitrogen tensions. *Science* 229:556–58
62. Kooyman GL, Sinnett EE. 1982. Pulmonary shunts in harbor seals and sea lions during simulated dives to depth. *Physiol. Zool*. 55:105–11
63. Scholander PF. 1940. Experimental investigations on the respiratory function in diving mammals and birds. *Hvalradets Skrif*. 22:1–131
64. Kooyman GL, Cornell LH. 1981. Flow properties of expiration and inspiration in a trained bottlenose porpoise. *Physiol. Zool*. 54:55–61
65. Kooyman GL, Kerem DH, Campbell W, Wright JJ. 1971. Pulmonary function in free-diving Weddell seals. *Respir. Physiol*. 12:271–83
66. Ridgway SH, Howard R. 1979. Dolphin lung collapse and intramuscular circulation during free diving: evidence from nitrogen washout. *Science* 206:1182–83
67. Harvey EN, McElroy WD, Whitely AH, Warren GH, Pease DC. 1944. Bubble formation in animals. III. An analysis of gas tension and hydrostatic pressure in cats. *J. Cell. Comp. Physiol*. 24:117–32
68. Hills BA. 1968. Relevant phase conditions for predicting occurrence of decompression sickness. *J. Appl. Physiol*. 25:310–15
69. Griffiths HB. 1971. On the role of separated gas in decompression sickness. *Proc. R. Soc. London Ser. B* 178:389–406
70. Lenfant C, Kooyman GL, Elsner R, Drabek CM. 1969. Respiratory function of the blood of the Adelie penguin (*Pygoscelis adeliae*). *Am. J. Physiol*. 216:1598–600
71. Weber RE, Hemmingsen EA, Johansen K. 1974. Functional and biochemical studies of penguin myoglobins. *Comp. Biochem. Physiol*. 49:197–214
72. Fujise Y, Hidaka H, Tatsukawa R, Miyazaki N. 1985. External measurements and organ weights of five Weddell seals (*Leptonychotes weddellii*) caught near Syowa Station. *Antarc. Rec. (Tokyo)* 85:96–101
73. Ridgway SH, Johnston DG. 1966. Blood oxygen and ecology of porpoises of three genera. *Science* 151:456–58
74. Blessing MH. 1972. Studies on the concentration of myoglobin in the sea-cow and porpoise. *Comp. Physiol. Biochem*. 41A:475–80
75. Sleet RB, Sumich JL, Weber LJ. 1981. Estimates of total blood volume and total body weight of sperm whale (*Physeter catodon*). *Can. J. Zool*. 59:567–70
76. Lockyer C. 1976. Body weights of some species of large whales. *J. Const. Int. Explor. Mer.* 36:259–73