Oxygen, Carbon Dioxide, and Internal Transport *at Work:* Diving by Marine Mammals

CHAPTER 26

The Weddell seal (*Leptonychotes weddellii*) captures all its food under water, yet depends entirely on the atmosphere for oxygen. It seems, when first considered, to be one of the most physiologically improbable creatures one could imagine. Not only is it incapable of breathing where it hunts, it is large (400–500 kg), homeothermic, entirely predatory, and confronted by cold throughout its life. A Weddell seal lives through all seasons in the frigid Antarctic, either hauled out on ice sheets or diving in seawater at –2°C to capture fish, crustaceans, and squids. Despite the seeming improbability of such animals, they are ecologically successful. Early Antarctic explorers, such as the Scottish sea captain James Weddell, brought home news of stupendous populations of diving mammals. Weddell seals today number near 1 million.

Worldwide, there are more than 80 species of whales and dolphins, plus more than 30 species of seals and sea lions. They are descended from terrestrial ancestors, probably lured to the sea by a bounty of food. As they evolved methods of feeding on the ocean's riches, however, marine mammals did not evolve ways of gleaning O_2 from the sea, and thus they remain tied to their ancestors' mode of breathing.

Modern technology is enabling us to realize more fully than ever the enormous role of diving in the lives of these animals. **Figure 26.1**, for example, shows the dives of three seal species along a relatively short stretch of coastline in Antarctica. Each vertical yellow trace represents a dive. The seals dive often as they travel horizontally from place to place.

Diving by marine mammals first came under serious study by physiologists around 1935. From the beginning, physiological studies have focused on two basic questions: First, how do animals that are strictly dependent on the atmosphere for O_2 meet their metabolic energy demands during long periods under water? Second, how do diving mammals cope with the high pressures they encounter at depth? This chapter focuses on the first question, but considers the second briefly at the end. The individual development of diving competence, as animals mature from weaning to full adulthood, is a critical topic in the study of diving: As a youngster matures, it must acquire sufficient food at each stage of its life even though its development of full diving competence may require many months. We discussed an example of the development of diving in Chapter 4. Here our focus will be on adults.

Weddell seals These large seals are found only in Antarctica, where they collect all their food by diving. Weddell seals (*Leptonychotes weddellii*) often cut and maintain holes through ice sheets so as to be able to access their dual habitats: water for feeding and air for breathing.

Diving Feats and Behavior

The diving feats of seals and whales have been appreciated in a limited way for centuries. During the heroic era of whaling (chronicled most famously in Herman Melville's *Moby-Dick*), for example, the whalers were amazed by the depths to which wounded whales could dive. Sometimes a sperm whale had to be cut loose after "sounding" so deep as to draw out two lengths of harpoon line, each more than 370 m long. When physiological research on animal diving began, few techniques existed for the study of free-ranging animals in the wild, and thus the first investigations were mostly carried out in bathtubs

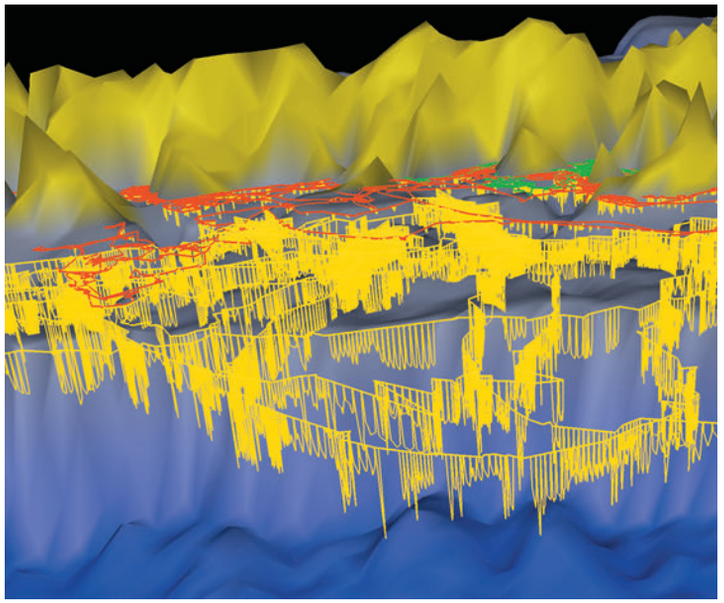


FIGURE 26.1 Natural dives of three seal species in the wild The image is a three-dimensional representation of diving and surface movement by seals in Antarctica. Each dive (regardless of species) is represented by a vertical yellow path, the length of which represents dive depth. The lines at the water surface are the movement tracks of individual seals, color-coded to designate species: yellow, southern elephant seal (*Mirounga leonina*); green, Weddell seal (*Leptonychotes weddellii*); red, crabeater seal (*Lobodon carcinophagus*). Insight into depth is provided by recognizing that the average depth of elephant seal dives was 360 m. Because crabeater seals predominantly eat krill swarming in the upper 100 m of water, they do not dive to particularly great depths in comparison with Weddell and elephant seals, which seek fish and squid. (Courtesy of Daniel Costa.)

and swimming pools. By 1970, however, innovative scientists were starting to take advantage of the revolution in technology to monitor the diving behavior of free-living animals. They discovered that fact is indeed sometimes stranger than fiction. There are mammals that sometimes electively stay under water—holding their breath—for 2 h, and there are ones that go so deep that they leave the atmosphere—their source of O_2—more than a mile behind.

The Weddell seal proved to be a perfect subject for early studies of diving behavior in the wild because the only devices initially available to obtain data on the durations and depths of dives were innovative but primitive instruments that—after being attached to an animal for a recording period—had to be reclaimed for the data to be acquired. Weddell seals living on ice sheets gain access to the water for feeding by cutting and maintaining holes through the ice. After a seal dives through an ice hole, it must ultimately return to that or another hole to breathe. Gerald Kooyman and his colleagues capitalized on this trait. They would attach a data recorder to a wild Weddell seal and then permit the seal to live in its ordinary way for several days, whereupon they would find the seal at one of its breathing holes to remove the device. Using this approach, they were able—for the first time with any species—to describe the durations and depths of thousands of voluntary dives.

The most fantastic revelation of the early studies was that Weddell seals sometimes stay submerged voluntarily for more than 1 h (Figure 26.2); we know today that their dives occasionally last as long as 80 min! Equally important, however, was the revelation that the vast

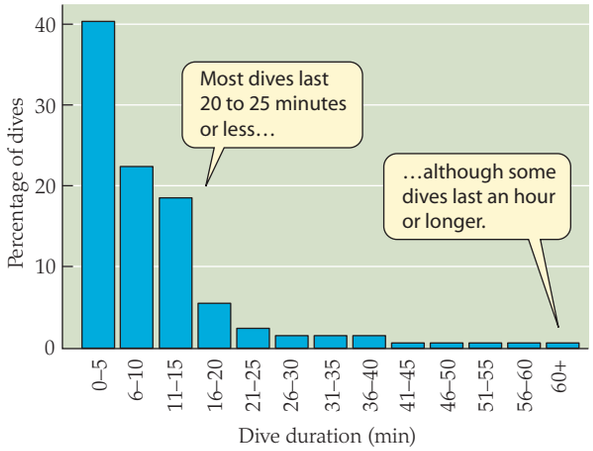


FIGURE 26.2 Durations of dives by wild Weddell seals These data represent more than 1000 dives by six free-living seals. Each vertical bar shows the percentage of dives falling within a particular duration category. Most dives are far shorter than the maximum duration displayed by the species. (After Kooyman et al. 1980.)

majority of dives by Weddell seals are considerably shorter than the maximum durations observed; most dives last 20–25 min or less. The pattern of diving depths resembles that of diving durations in that, during most dives, seals go to depths that are considerably shallower than the maximum the species is capable of reaching (Figure 26.3). On rare occasions, a Weddell seal descends to nearly 600 m (0.37 mile), but few dives are deeper than 400 m. While diving, a seal is sometimes subject to stupendous physical pressures. A useful rule of thumb is that water pressure increases by about 1 atmosphere (atm) (101 kPa) for every 10 m of depth. Thus a seal diving to 400 m voluntarily subjects itself to about 40 atm of hydrostatic pressure.

Today, technology has advanced to the point that data can be radioed from free-living marine mammals and picked up on a global scale by satellite receivers. One dramatic study using this

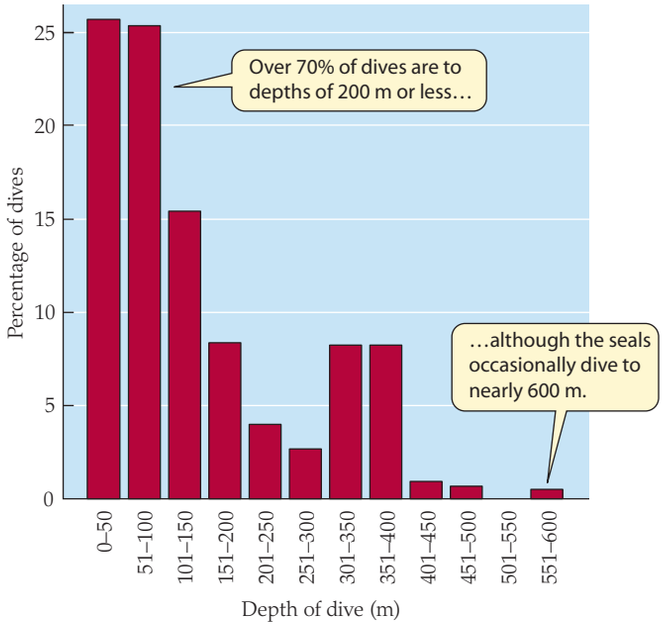


FIGURE 26.3 Depths of dives by wild Weddell seals These data represent more than 380 dives by 27 free-living individuals. Each vertical bar shows the percentage of dives falling within a particular depth category. Most dives are substantially shallower than the maximum depth displayed by the species. (After Kooyman 1966.)

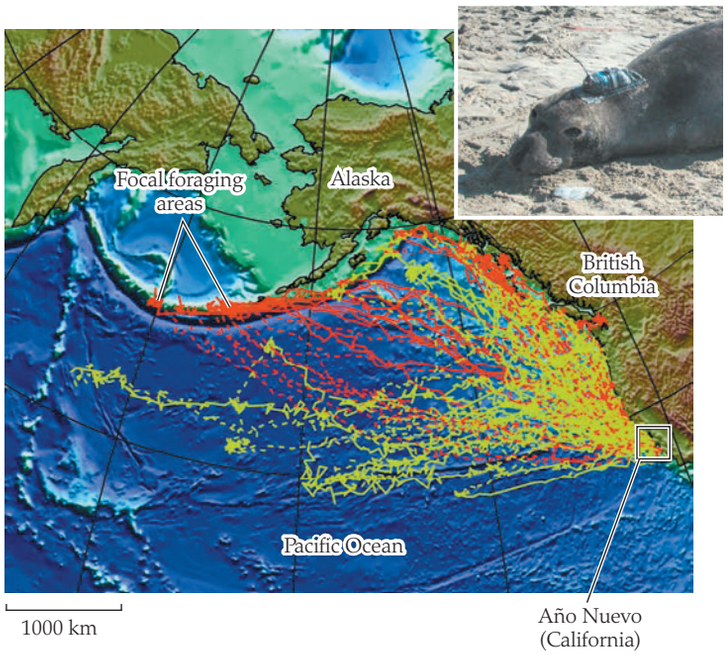


FIGURE 26.4 Migrations of northern elephant seals The elephant seals (*Mirounga angustirostris*) that breed at Año Nuevo in California get their food at distant places in the Pacific Ocean. Shown here are the migration routes of more than 30 animals from the Año Nuevo rookery. They were equipped with radio transmitters that signaled their location (see inset) and followed by satellite. The "focal foraging area" of an animal, which usually looks like a tight cluster of data in these records, is the part of the ocean where the seal lingered for many weeks of diving and feeding after its outward journey. When animals leave Año Nuevo, they tend to travel without pause to their focal foraging areas. During successive migratory trips away from Año Nuevo, individuals often return to their specific focal foraging areas using much the same outbound and inbound travel routes on each trip. Red represents males; yellow-green, females. (Courtesy of Dan Costa; after Le Boeuf et al. 2000.)

technology was carried out on the northern elephant seals (*Mirounga angustirostris*) that breed in a famous colony at Año Nuevo in California, south of San Francisco. Both sexes spend most of their lives at sea. They visit land on two occasions each year to mate, molt, and (in the case of females) rear young. Between these visits to land, the seals undertake long oceanic migrations, often across half the breadth of the Pacific Ocean, during which they feed intensively. The longitudes and latitudes of individual animals can be followed throughout their migrations using radio and satellite technology (Figure 26.4). For monitoring diving depths and durations, instruments that log the data rather than transmit them are still sometimes used. Modern digital data loggers, however, have prodigious capabilities compared with the original data loggers. Therefore, a data logger attached to a seal at the start of its migration can record all the animal's dives throughout its entire migratory trip, and the diving data can later be correlated with the seal's satellite-recorded longitudes and latitudes. Figure 26.5 shows the depths of all the dives undertaken by a male northern elephant seal called Moo while in transit between Año Nuevo and his principal foraging area near the Aleutian Islands. Moo's deepest dive was to about 750 m (0.47 mile). Another northern elephant seal was once observed to dive to almost 1600 m (essentially 1 mile). Most dives by northern elephant seals, however, are to depths far less than these maxima, as exemplified by Moo's diving record (see Figure 26.5).

The marine mammals are far from uniform in their diving capabilities. The major groups—the true seals, the fur seals, and the whales—have different phylogenetic histories, and within each group, species have diversified. Weddell seals, elephant seals, and ribbon seals—all true (phocid) seals—are among the most proficient divers. One of the longest voluntary dives ever recorded, 2 h, was observed in a southern elephant seal. Elephant seals also attain astounding depths; a male southern elephant seal was recorded recently to dive to over 2000 m (1.2 miles) on more than 160 different occasions, reaching a maximum depth of 2150 m (1.33 miles)—probably the deepest dive ever observed. Sperm whales and certain of the beaked whales also rank with the most proficient divers. Dives to 1900–2000 m have been recorded in sperm whales (*Physeter macrocephalus*) and Cuvier's beaked whales (*Ziphius cavirostris*). Sperm whales dive routinely for 40–50 min and occasionally dive for longer than 2 h. The opposite end of the spectrum of diving proficiency is exemplified by certain of the fur seals, such as northern fur seals (*Callorhinus ursinus*).[1] Based on records of more than 3000 dives, northern fur seals do not dive longer than 8 min or deeper than 260 m. Despite wide variation in the extreme dives of which species are capable, all species of diving mammals (and birds) seem to adhere to the two important generalizations that we have already illustrated: (1) The durations of most dives are substantially shorter than the maximum duration of which each species is capable, and (2) most dives are to depths substantially more shallow than the species-specific maximum depth. Dives of record duration and

[1]The fur seals and sea lions—which compose the group known as otariid seals—tend as a group to be less proficient divers than the true (phocid) seals.

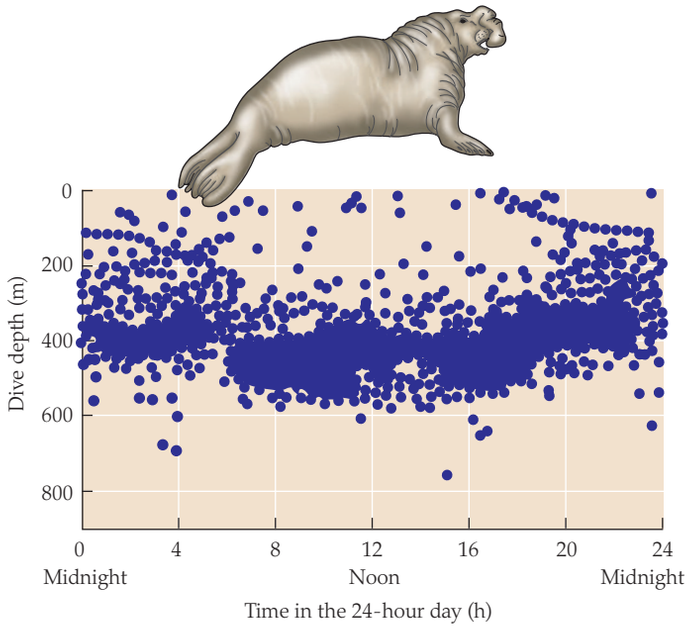


FIGURE 26.5 Diving by Moo, a male northern elephant seal, during his migration The maximum depth reached by Moo during each of his dives is plotted against the time of day of the dive. Each symbol represents a single dive. This plot includes all dives that Moo undertook during several weeks while he was in transit between Año Nuevo and his focal foraging area in Alaskan waters. Even though Moo had to pay the metabolic cost of swimming 6500 km (round trip) during this migration, he increased his body weight from 1270 to 1650 kg by diving for food. Moo's dives were recorded by a time–depth logger with prodigious memory that was attached to him before his departure from Año Nuevo and recovered after his return. (After Le Boeuf et al. 2000.)

depth, although they are awe inspiring and require physiological explanation, are uncommon.

To put the performances of marine mammals into perspective, it is instructive to look at the capabilities of a representative terrestrial mammal, *Homo sapiens*. Trained human breath-hold divers are generally limited to about 3 min of submergence when at rest and about 90 s when swimming. The *ama* of Korea and Japan earn their living by diving for shellfish and edible seaweeds. Their abilities are typical of those exhibited by other people (e.g., pearl and sponge divers) who employ breath-hold diving as a career. The ama who dive in deep waters receive assistance during descent and ascent so that their time at the bottom can be maximized. They carry weights to aid descent and are pulled back to the water's surface on a rope by an assistant in a boat. They routinely remain submerged for 60–80 s and reach depths of 15–25 m. They rest only about 1 min between dives and thus average about 30 dives per hour. The extremes recorded for human performance during breath-hold diving are attained by competitive divers and are difficult to summarize because several distinct types of competitive diving exist and have their own rules. When assistance can be employed in both descent and ascent, depths of about 210 m in dives lasting more than 4 min are achieved. Dives of this depth and duration can be carried out only by exceptional people and are dangerous even for them. However, such dives seem trivial and commonplace for average individuals of many species of marine mammals.

Types of Dives and the Importance of Method

An important theme throughout science—dramatized in the study of diving physiology—is that the methods employed in experiments often affect the results and, therefore, our perception of reality. During the first decades of the modern study of diving physiology (roughly 1935–1970), scientists believed that they should "bring diving into the laboratory" where they could establish experimental controls and employ sophisticated instrumentation. In the lab studies that were carried out, animals were often strapped to a movable platform and lowered under water whenever an investigator wished to elicit diving responses. By the 1960s, research of this sort had produced such a seemingly complete picture of diving physiology that most biologists believed that diving was a "solved problem." Then some radical investigators began to study animals such as Weddell seals in the wild. The methods they had available to use were initially primitive compared with those available in the lab. Nonetheless, their early studies showed that the physiology of *voluntary* diving in the wild often differs from that of *forced* diving in the lab. Perceptions thus changed. The initial view had been that diving physiology shows little variation from one dive to another because it is controlled by highly stereotyped physiological reflexes. The newer view—now abundantly confirmed—was that marine mammals use a spectrum of physiological strategies during different sorts of dives. Today, even as advances in technology have greatly expanded the range of physiological mechanisms that can be studied in the wild, lab studies continue to play a role because certain mechanisms can be studied only in the lab even now.

Recognizing that lab and field studies of diving may produce different results, one of the first questions to ask about any set of data is whether it was obtained by forcing animals under water or by studying free-living animals diving voluntarily. Another important question is whether the dives studied were long or short *relative to the species-specific maximum dive length*, because we know now that qualitatively different suites of responses are often marshaled in long and in short dives. Still another key question is whether the studied animals were active or quiet during diving. These, then, are some of the important organizing distinctions among dives in the study of diving physiology:

- Forced or voluntary?
- Short or long (relative to the maximum length for the species)?
- Quiet or active?

Physiology: The Big Picture

When diving mammals break contact with the atmosphere, they carry O_2 with them in three major internal stores: O_2 bound to blood hemoglobin, O_2 bound to muscle myoglobin, and O_2 contained in air in their lungs. These stores permit aerobic catabolism to continue to some extent during a dive. The internal stores of O_2 are adequate in principle to permit all the tissues of a diver's body to function aerobically throughout a relatively short dive. When we look at the physiology of actual dives, we find, in fact, that voluntary dives of relatively short duration are mostly or completely aerobic in a variety of diving species, according to available evidence.

However, internal O_2 stores are utterly inadequate to permit fully aerobic function throughout a diver's body during protracted dives. How, then, are some species able to survive for 30 min, 60 min, or even longer without breathing? Our basic concept of how energy needs are met during protracted dives was first proposed by Laurence Irving (1895–1979) in 1934. He recognized that certain tissues—notably the central nervous system and heart—are predominantly or exclusively dependent on aerobic catabolism for production of ATP; they need O_2 on a steady basis and are quickly damaged by O_2 insufficiency. However, other tissues—such as skeletal muscle—have a well-developed ability to meet their ATP demands anaerobically and thus are relatively tolerant of O_2 deprivation. Irving then reasoned that dives can be prolonged if animals "reserve" a portion of their O_2 supplies for the tissues that are O_2-dependent. During a dive, if all tissues have equal access to an animal's entire O_2 store, then the concentration of O_2 throughout the body will fall quickly to such a low level that the O_2-dependent tissues are impaired. Under such circumstances, a seal or whale will need to surface, even though many of its tissues could continue to function—anaerobically—for a longer time. However, if some O_2 is reserved for use by the O_2-dependent tissues during a dive, then those tissues can continue to have adequate O_2 even while other parts of the body exhaust their O_2 supplies and turn to anaerobic catabolism, thereby extending the time the animal can remain submerged.

The preferential delivery of some O_2 to the O_2-dependent tissues is achieved, as Irving predicted, by adjustments of circulatory function. These adjustments were first elucidated by Irving, working with Per Scholander (1905–1980), in lab experiments on seals forced under water. During forced dives, blood flow is curtailed to many body regions—such as the skeletal muscles of the appendages and

trunk, the skin, the gut, and the kidneys—by vasoconstriction of the arterial vessels that supply those regions. The tissues that are deprived of active blood flow can make use of the hemoglobin-bound O_2 in the small volume of blood that passes through their capillaries, and they can use their myoglobin-bound O_2. However, as those limited and local O_2 stores are depleted, the circulation-deprived tissues turn to anaerobic catabolism, and lactic acid accumulates in them. *With the circulation to many parts of the body curtailed, the heart pumps blood primarily between itself and the lungs and head*. The O_2 stores of the circulating blood are thereby reserved primarily for the O_2-dependent tissues—the brain and heart—and whatever O_2 is extracted from the air in the lungs is likewise delivered preferentially to those tissues. Consequently, adequate O_2 partial pressures can be maintained in the O_2-dependent tissues for a long period. Lactic acid produced by the skeletal muscles and other circulation-deprived tissues tends to remain sequestered in those tissues during a dive, precisely because the tissues receive little or no blood flow. When the animal surfaces, however, circulation to such tissues is restored, and there is a sudden rise of lactic acid in the circulating blood. *The observation that circulating lactic acid increases principally* after *a forced dive was one of the earliest pieces of evidence that circulatory function is sometimes radically altered during diving.*

When animals are forcibly submerged, the adjustments in the pattern of blood flow just described tend to occur rapidly, consistently, and to a profound extent. Accordingly, during the era prior to 1970, when diving was studied mainly in labs, these responses were labeled a *diving reflex*. Today, however, based on studies of voluntarily diving animals, physiologists recognize that the responses of the circulatory system are not nearly as inflexible and stereotyped as once thought. Indeed, as suggested earlier, relatively little redistribution of blood flow is believed now to occur during voluntary dives that are short enough for all energy needs to be met by aerobic catabolism using an animal's O_2 stores. Seals and whales in the wild undergo a profound redistribution of blood flow when they dive voluntarily for long periods. Animals that are forced under water probably exhibit stereotypic and "reflexive" circulatory responses because they sense that they have no control over the length of time they will be submerged, and thus they consistently marshal the responses they employ for prolonged diving in the wild.

From this overview, you can see that the O_2 stores, circulatory physiology, and metabolic physiology of marine mammals all play critical roles in their diving physiology. The next three sections discuss these three elements in more detail.

The Oxygen Stores of Divers

The size of a diving mammal's total O_2 store is obviously a key determinant of how long the animal can stay submerged. Among other things, a dive can last only as long as the brain is supplied with O_2, and the size of the O_2 store helps determine how long the supply of O_2 to the brain can be sustained.

The blood O_2 store tends to be large in diving mammals

The amount of O_2 stored in the blood depends on three features: (1) the oxygen-carrying capacity of the blood, (2) the total volume of blood, and (3) the degree to which the blood is fully loaded (saturated) with O_2 at the time of submergence.

Regarding the oxygen-carrying capacity, although some species of diving mammals have values that are well within the ordinary range for nondiving, terrestrial mammals, some other diving species have exceptionally high values. Bottlenose dolphins, northern fur seals, and Steller (northern) sea lions illustrate the first group; they have oxygen-carrying capacities of 17–22 vol %[2]—quite ordinary for mammals. In contrast, species of diving mammals known to have especially high oxygen-carrying capacities include the harbor seal (26–29 vol %), sperm whale (31 vol %), Weddell seal (29–36 vol %), and ribbon seal (34 vol %). Among the seals as a group, there is a trend for species that undergo long dives—such as the three true seals just mentioned—to have higher oxygen-carrying capacities than species that perform shorter dives.

Blood volumes in some representative terrestrial species—humans, dogs, horses, and rabbits—average 60–110 mL per kilogram of body weight. Although some diving species have blood volumes in the same range, some of the accomplished divers such as harbor and ribbon seals have blood volumes of 130–140 mL/kg, and the blood volumes of Weddell seals, elephant seals, and sperm whales are 200–250 mL/kg—two to three times as high as is typical of terrestrial mammals.

An animal's maximum possible blood store of O_2 is calculated by multiplying the oxygen-carrying capacity of its blood by its blood volume. This figure, while only indirectly relevant to normal physiology (because the entire volume of blood is never fully oxygenated), is useful for comparing species. The maximum possible blood O_2 store of humans and horses is about 14–15 mL O_2 per kilogram of body weight. In contrast, far higher O_2 storage capacities are found in the species of diving mammals that combine the advantages of both a high oxygen-carrying capacity and a high blood volume. The maximum blood O_2 store in Weddell seals, elephant seals, and sperm whales is 60–85 mL O_2/kg—four to six times higher than the stores of humans and horses.

Diving mammals have high myoglobin concentrations and large myoglobin-bound O_2 stores

The amount of O_2 stored as oxymyoglobin at the time of submergence depends on how much myoglobin is present in each unit of muscle tissue. *One of the most consistent features of diving species of mammals is that, relative to terrestrial species, they have dramatically high myoglobin concentrations in their skeletal muscles*. The skeletal muscles of some proficient divers are so rich in myoglobin that they are almost black. In humans and horses, skeletal muscles contain about 4–9 mg of myoglobin per gram of wet weight. By contrast, sperm whales and harbor, Weddell, elephant, and ribbon seals have 55–70 mg of myoglobin per gram of wet weight!

Oxymyoglobin represents an essentially private store of O_2 for the muscles. As discussed in Chapter 24 (see page 636), myoglobin has such a high affinity for O_2 that it typically draws O_2 from blood hemoglobin rather than donating O_2 to the blood. Thus, even if muscles receive blood circulation during a dive, oxymyoglobin within the muscles does not yield much O_2 to the blood for use elsewhere in the body. Instead, the O_2 remains bound to the myoglobin until the O_2 partial pressure in the muscles falls to a low level; then it is donated to the muscle mitochondria to permit continued aerobic

[2] As applied to blood (see Chapter 24), O_2 vol % is the amount of O_2—expressed as volume in mL at standard conditions of temperature and pressure—that is present per 100 mL of blood (including O_2 present both in combination with hemoglobin and in solution).

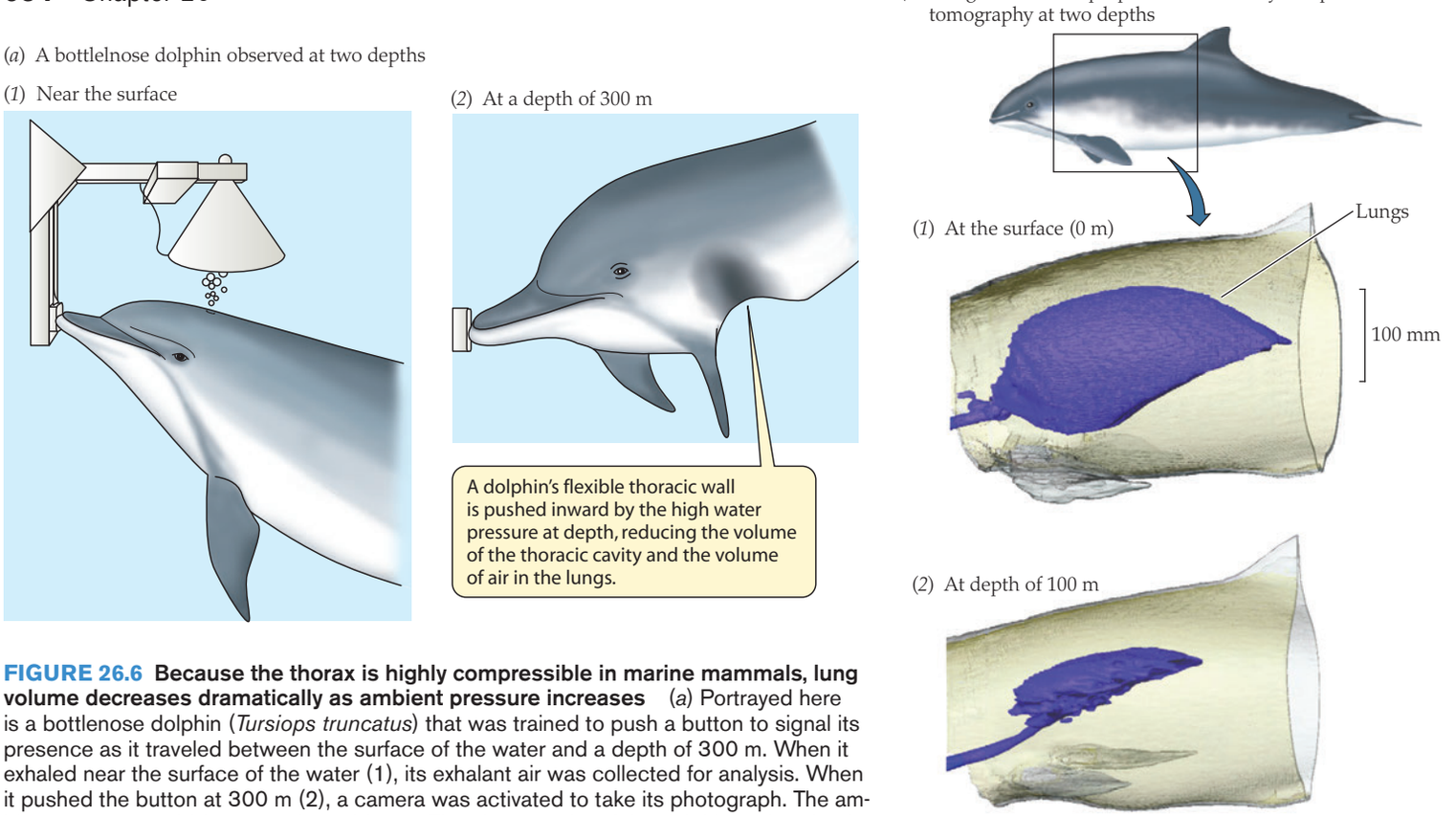


FIGURE 26.6 Because the thorax is highly compressible in marine mammals, lung volume decreases dramatically as ambient pressure increases (*a*) Portrayed here is a bottlenose dolphin (*Tursiops truncatus*) that was trained to push a button to signal its presence as it traveled between the surface of the water and a depth of 300 m. When it exhaled near the surface of the water (1), its exhalant air was collected for analysis. When it pushed the button at 300 m (2), a camera was activated to take its photograph. The ambient pressure at 300 m was about 30 atm. The photographs of the dolphin at that depth revealed that its thoracic wall was pushed far inward by the elevated ambient pressure. (*b*) The lungs inside the thorax of a harbor porpoise (*Phocoena phocoena*) at two different ambient pressures. Although the porpoise had died of an accident, it was freshly dead and undamaged. As the ambient water pressure was raised to simulate depths of 0 and 100 m, the porpoise's lungs were visualized using a CT (computed tomography) scanner, with scaling identical in both images. Note that *both* the thorax and lungs decreased in volume as ambient pressure increased. (*a* after Ridgway et al. 1969; *b* from Moore et al. 2011.)

ATP production in the muscles. An interesting point is that the O_2 partial pressure in a muscle *must* be low for the muscle's myoglobin to unload. One role of the severe reduction of blood flow to the skeletal muscles during prolonged dives is probably to ensure that the muscle O_2 partial pressure falls promptly to a level that will permit myoglobin to be tapped for O_2.

Diving mammals vary in their use of the lungs as an O_2 store

To understand lung function in diving mammals, one must first recognize the unusual structural flexibility of the thorax in these animals. In the deep-diving marine mammals that have been studied, the thorax is structured in ways that permit the thoracic walls to be shoved freely inward as the outside water pressure increases (**Figure 26.6*a***). Thus, over extensive ranges of depth and pressure, the *thoracic cavity and lungs are freely compressible* (**Figure 26.6*b***). If the pressure applied to the thorax is increased tenfold, for example, the volume of air in the lungs is reduced to roughly one-tenth of what it previously was.[3] Humans and other terrestrial mammals are different in that their thoracic walls are structured less flexibly and resist compression.

One might at first think that a large air store in the lungs would be of unquestionable advantage for a diving mammal. Three considerations, however, argue against this conclusion. First, a large amount of air in the lungs can strongly buoy a diving animal upward, forcing it to work hard to remain submerged. Second, the alveoli are believed to be typically the first parts of the lungs to collapse as the lung air compresses at depth. Hence, at depth, the lung air comes to be contained mostly in the *conducting* airways—the trachea, bronchi, and nonrespiratory bronchioles—where the O_2 in the air becomes unavailable because little O_2 transfer to the blood can occur in the conducting airways. Thus, whereas diving mammals can make effective use of their pulmonary O_2 stores during shallow diving, they are not necessarily able to do so at depth. A final consideration is that a large pulmonary air store means not only a large store of O_2 in the lungs, but also a large store of N_2 (air is 78% N_2). A large N_2 store can increase the likelihood of decompression sickness, as discussed later in this chapter.

The size of the air store in a marine mammal's lungs at the start of a dive depends on two factors: the *volumetric capacity* of the lungs (the amount of air they can hold when fully inflated) and the degree to which the animal inflates its lungs before diving. Regarding the first factor, marine mammals do not as a rule have exceptionally large lungs; their volumetric capacities per unit of body weight are generally similar to those of terrestrial mammals, or just modestly larger. Moreover, many of the deep-diving seals and whales have volumetric capacities that are relatively low by comparison with

[3]Too few actual measurements are available to determine if lung volume responds to changes of outside pressure identically as would be predicted from a simple application of the universal gas law (see Equation 22.1).

those of other species of seals or whales of similar size that dive more shallowly. Regarding the second factor—the degree of lung inflation—some marine mammals dive after a vigorous inhalation and thus may carry an amount of lung air that approaches their volumetric capacity. This appears to be true, for example, of whales (including dolphins). By contrast, many deep-diving species of true (phocid) seals dive following *exhalation*; their lungs are filled to just 20–60% of their volumetric capacity when they submerge. All things considered, little premium is placed on having an exceptionally large pulmonary air (and O_2) store in marine mammals.

Total O_2 stores never permit dives of maximum duration to be fully aerobic

Three major points emerge from the study of the O_2 stores of marine mammals:

1. Some species of diving mammals have much greater total O_2 stores per unit of body weight than terrestrial mammals because they have high blood oxygen-carrying capacities and high blood volumes—giving them high blood O_2 stores—and they have high concentrations of myoglobin in their muscles—giving them high myoglobin-bound O_2 stores (**Figure 26.7**).
2. Among diving mammals, species such as fur seals and sea lions that dive for relatively short periods tend to have smaller O_2 stores per unit of body weight than species such as harbor and ribbon seals that are more proficient as divers and dive for longer periods (see Figure 26.7).
3. The O_2 stores of marine mammals of all kinds are utterly inadequate to sustain a rate of O_2 consumption during long dives that is equivalent to the rate of O_2 consumption of these animals while they are at rest and breathing air.

The third point is sufficiently important to deserve illustration. Paul Ponganis and his colleagues estimated the total available O_2 store of a 450-kg Weddell seal to be 38.8 L.[4] Such a seal has a resting rate of O_2 consumption, when it is breathing air, of 1.9–2.3 L O_2/min. Thus the seal could sustain its resting, aerial rate of O_2 consumption for 17–20 min during diving if it completely used its available O_2 store. Actually, however, Weddell seals sometimes dive for 60–80 min (see Figure 26.2). This sort of result is typical of diving species; dives of maximum length always last from two to several times longer than would be predicted if an animal were to function aerobically at the rate seen during rest in air. Another way to see that O_2 stores are inadequate to account fully for diving performance is to recognize that the diving capabilities of long-duration divers are disproportionate to their O_2 stores. For example, consider harbor seals and humans, two species of roughly the same body size. Although the weight-specific O_2 stores of harbor seals are 2–2.5 times higher than those of humans (see Figure 26.7), an average seal can remain submerged for more than 12 times as long as an average human when diving under comparable conditions. Regardless of how large the O_2 stores of diving species may be, these stores do not in themselves explain the dive durations of which the animals are capable.

[4]To calculate this amount from Figure 26.7, multiply the size of the O_2 store per kilogram, 86 mL O_2/kg, by the seal's body weight, 450 kg.

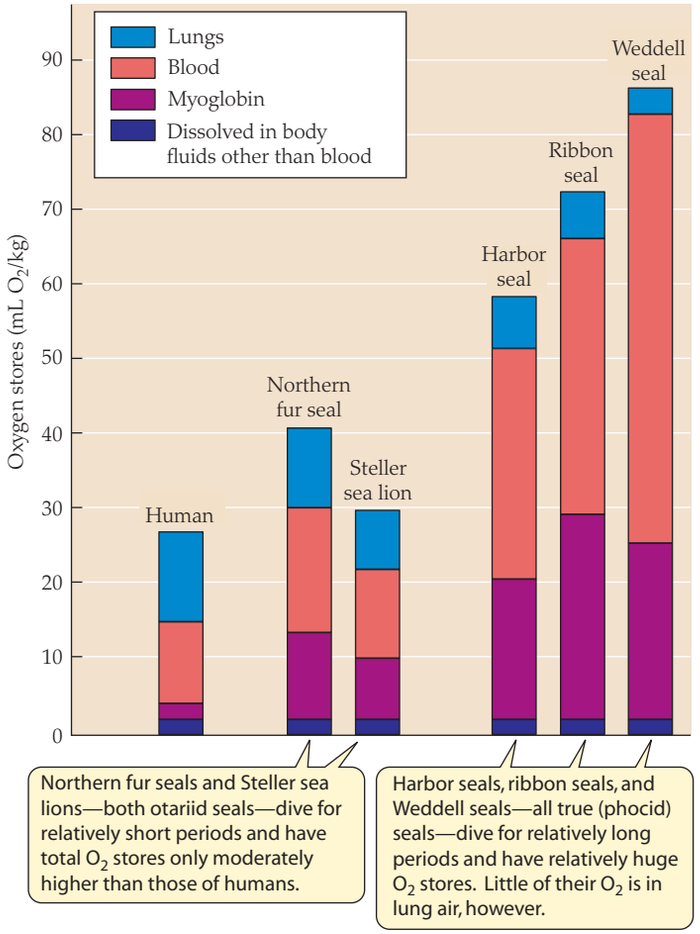


FIGURE 26.7 A comparison of the total O_2 stores of five species of marine mammals and humans The total O_2 available per kilogram of body weight for each species is estimated by adding the O_2 dissolved in body fluids other than blood throughout the body, the O_2 bound to myoglobin in muscles, the O_2 in blood (mostly bound to hemoglobin), and the O_2 in lung air. The lungs of humans are assumed to be fully inflated, but those of the other species are assumed to be only half inflated because many seals dive after exhaling. The data for the fur seal, sea lion, harbor seal, and ribbon seal were calculated by one research team using a standardized set of procedures and assumptions (Lenfant et al. 1970); the data for the Weddell seal (Ponganis et al. 1993) and human, although comparable, are from other sources.

Circulatory Adjustments during Dives

The circulation holds a special place in the chronicles of diving physiology because the very first physiological observations on diving were measures of heart rates. Starting in 1870, the French physiologist Paul Bert (1833–1886) studied ducks and found that their heart rates decreased from 100 to 14 beats/min when he forced them under water. A decrease in heart rate during diving is called **diving bradycardia** (*brady*, "slow"). Bert and others soon demonstrated that the phenomenon is a consistent feature of forced submergence in diving mammals and birds. The universality of diving bradycardia quickly persuaded physiologists that the slowing of the heart is important in permitting animals to stay submerged for extended periods. But how is it important?

Irving and Scholander took the next crucial step. In the 1930s, they postulated that in addition to the heart, other parts of the

cardiovascular system also respond during diving. One of their earliest tests of their idea was to study bleeding from the paw skin of seals. Small cuts that bled freely when the seals were breathing air stopped bleeding suddenly and completely when the seals were pushed under water! From simple observations like this, the modern revolution in diving physiology began, and after seven decades of knowing of the existence of diving bradycardia, scientists began to understand its true significance.

Regional vasoconstriction: Much of a diving mammal's body is cut off from blood flow during forced or protracted dives

After physiologists realized that a marine mammal's pattern of blood flow might change during dives, they employed several techniques to examine how the vascular system functions during diving. One such technique involves the use of a *contrast fluid* that is impenetrable by X-rays. When a contrast fluid is injected into an animal's bloodstream, its flow can be observed on X-ray images. In one of the watershed studies in the history of diving physiology, a group of diagnostic radiologists headed by Klaus Bron injected a contrast fluid into the aorta of a harbor seal, then took X-ray images to observe subsequent blood flow in the seal's visceral arterial system while the animal was breathing air and while it was forcibly submerged (**Figure 26.8**).

When the seal was breathing air, an X-ray image taken 0.5 s after the injection of the contrast fluid showed that blood flowed vigorously from the aorta into abdominal arteries that branch off from the aorta, such as the coeliac and renal arteries (see Figure 26.8*a*). Within 2 s after injection, the left kidney was illuminated in the X-ray image, showing that the elaborate arterial system within the kidney had filled with blood that contained the contrast fluid (see Figure 26.8*b*). After only 6 s, the coeliac and renal arteries had already faded from view (see Figure 26.8*c*), demonstrating that the contrast fluid had flowed through them and exited.

The sequence of X-ray images taken while the seal was submerged revealed a strikingly different circulatory pattern. At 1 s after injection, the X-ray image showed that the blood containing the contrast fluid had penetrated only a short distance from the aorta into the coeliac and renal arteries and stopped, leaving the branches of those arteries unfilled (see Figure 26.8*d*; compare with Figure 26.8*a*). Even after 4 s, the left kidney remained dark, indicating that most of its arterial vessels had not received blood containing contrast fluid (see Figure 26.8*e*). As long as 14 s after injection, the bases of the coeliac and renal arteries remained filled with contrast fluid (see Figure 26.8*f*), indicating that the blood in those arteries had stagnated and was unable to flow through the vessels.

Bron's pioneering radiological study confirmed that blood flow to major parts of a marine mammal's body is profoundly curtailed during a forced dive. The coeliac and renal arteries supply blood to the stomach, spleen, liver, pancreas, and kidneys. During a dive, blood is unable to flow freely (if at all) into any of those visceral organs.

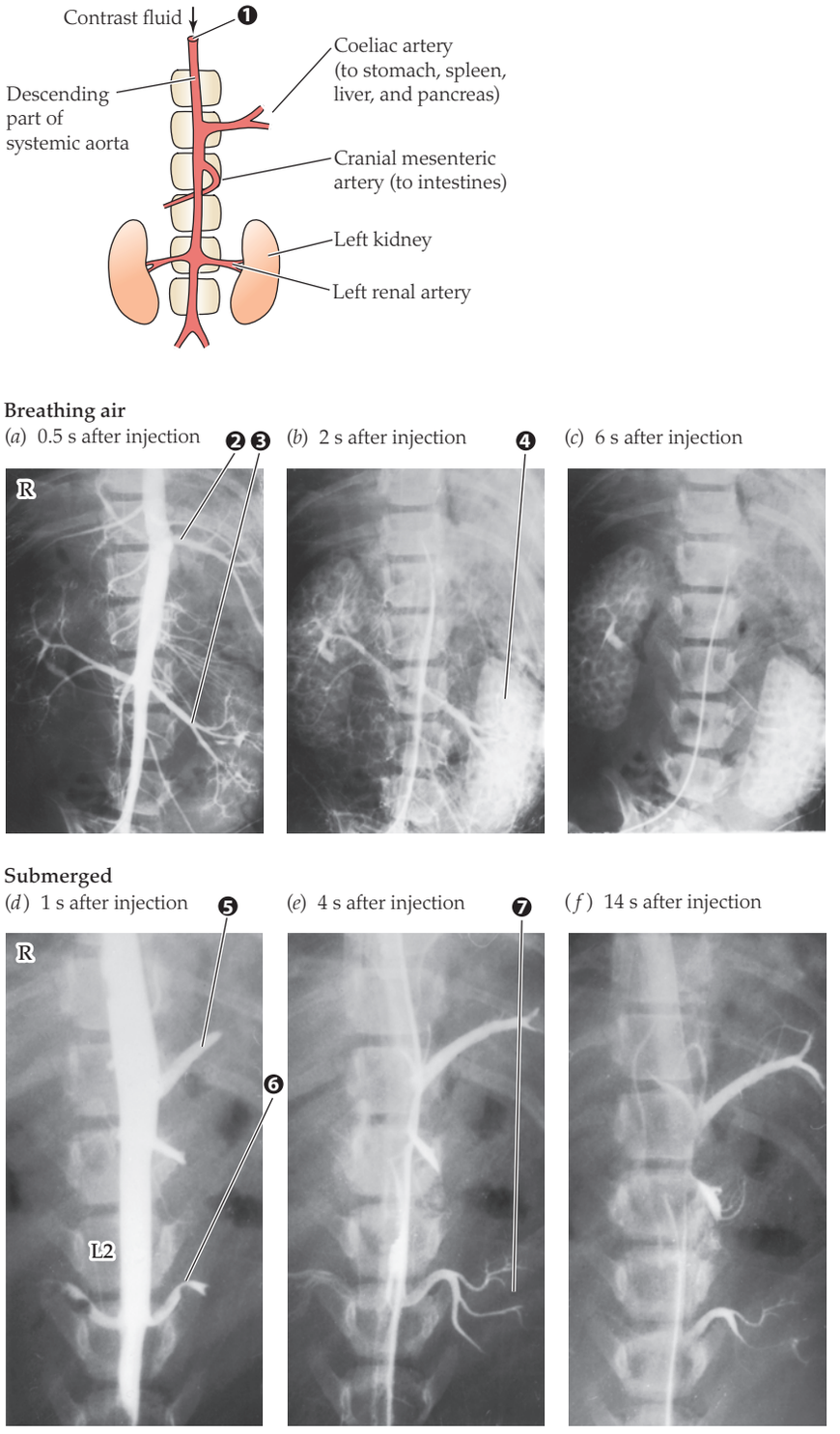


FIGURE 26.8 Circulatory patterns are radically changed during forced or prolonged submergence X-ray images of the visceral cavity of a harbor seal (*Phoca vitulina*), viewed from its ventral side, after injection of contrast fluid during air breathing (*a*–*c*) and during forced submergence (*d*–*f*). The images show that although the blood labeled with contrast fluid flowed into all major branches of the aorta within seconds during air breathing, the blood was unable to flow freely out of the aorta when the seal was submerged. ❶ Contrast fluid was injected into the descending portion of the systemic aorta. Within 0.5 s after the contrast fluid was injected when the seal was breathing air, the coeliac artery ❷ and left renal artery ❸ were filled with blood containing contrast fluid; and within 2 s after injection, the entire left kidney ❹ was filled with blood containing contrast fluid. When the seal was submerged, however, the coeliac artery ❺ and renal artery ❻ did not readily fill with blood containing contrast fluid, and after 4 s, the kidney ❼ remained dark—showing that it had not received blood with contrast fluid. L2 = second lumbar vertebra; R = right side of seal. (Photographs courtesy of Klaus Bron; from Bron et al. 1966.)

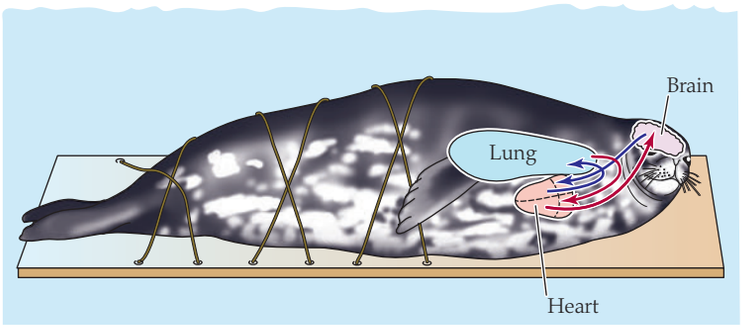


FIGURE 26.9 The forcibly submerged seal as a "heart-lung-brain machine" With blood flow to much of the body curtailed by vasoconstriction, active circulation is principally from the right heart to the lungs, the lungs to the left heart, the left heart to the brain, and the brain back to the right heart.

Vasoconstriction—under control of the sympathetic nervous system—is responsible for cutting off the blood flow. Strikingly, although arterioles are the typical sites of vasomotor control in mammals (see page 659), pronounced vasoconstriction occurs in *sizable arteries* in at least some diving species. Arteries constrict shut, and the organs they supply are denied blood flow (see Figure 26.8*d–f*). The parts of a diving mammal's body that receive little or no blood flow during forced dives commonly include the animal's limbs, the skeletal muscles of the trunk of its body, its pectoral muscles, its skin and body wall, and many visceral organs as already discussed.

Simultaneously, because vasoconstriction occurs selectively, blood flows freely—or relatively freely—to a diving animal's brain, lungs, and myocardium. A pundit once said that a seal becomes a "heart-lung-brain machine" during forced dives (**Figure 26.9**). Blood is pumped by the right heart to the lungs, travels from the lungs to the left heart, then is pumped by the left heart to the head, and finally returns to the right heart to be pumped again to the lungs. The rate of blood flow to the brain during diving tends to remain similar to the rate during air breathing. Meanwhile, as the blood travels round and round between heart and head, flow to the parts of the body posterior to the heart is cut off or severely restricted.

Diving bradycardia matches cardiac output to the circulatory task

After experiments had revealed that blood flow to many regions of a diving mammal's body is curtailed during forced or protracted dives, diving bradycardia could at last be understood for what it is: a single part of an integrated, body-wide reorganization of cardiovascular function. During a forced or protracted dive, vasoconstriction greatly reduces the dimensions of the active circulatory system. Accordingly, less output of blood from the heart is required. An analogy is provided by a faucet that sends a flow of water to six hoses; if five of the hoses are pinched off, the faucet can maintain unaltered flow to the sixth hose with only a sixth of its preexisting output. Bradycardia is a mechanism that matches the heart's output (the cardiac output) to the dimensions of the vascular system being perfused. In marine mammals, the stroke volume of the heart changes to only a modest extent, if at all, during diving, according to studies of several species. Thus cardiac output declines during a dive roughly in proportion to the decline in heart rate (see Equation 25.1).

Studies of blood pressure demonstrate in a particularly graphic way that the drop in cardiac output during a dive is matched to the reduction in the dimensions of the active circulatory system. As discussed in Chapter 25 (see Equation 25.3), the systemic arterial blood pressure depends on two factors: the rate of cardiac pumping and the resistance to blood flow posed by the vascular system. A change in either factor without a compensatory adjustment in the other can severely disturb blood pressure. The vasoconstriction that occurs during a dive increases the overall resistance to blood flow posed by the vascular system. However, the blood pressure in the great systemic arteries remains unaltered or changes only modestly. These two facts demonstrate that cardiac output is reduced during dives in a highly integrated way that closely matches the increase in peripheral vascular resistance.

Cardiovascular responses are graded in freely diving animals

Cardiovascular function in free-living marine mammals undergoing voluntary dives has proved particularly challenging to study but is gradually becoming understood. Today, for example, methods exist to monitor the electrocardiogram continuously in unfettered animals at sea. From use of such methods, we know that the heart rates of freely diving mammals typically decrease in a *graded* manner as the animals increase the durations of their dives (**Figure 26.10**). During forced dives, heart rate responses are more of an "on–off" sort; the heart rate of a forcibly submerged harbor seal, for instance, drops to less than 10% of its pre-dive level within 10 s every time the animal is submerged. An important distinction between voluntary and forced dives, therefore, is the graded versus stereotyped nature of the heart rate response during the two sorts of dives.

Ideal methods still do not exist for studying vasoconstriction in freely diving animals. For two major reasons, however, scientists believe that the vasoconstrictor response is also graded during voluntary dives. First, as we have discussed, heart rate and vasoconstriction are believed to be integrated in a way that matches

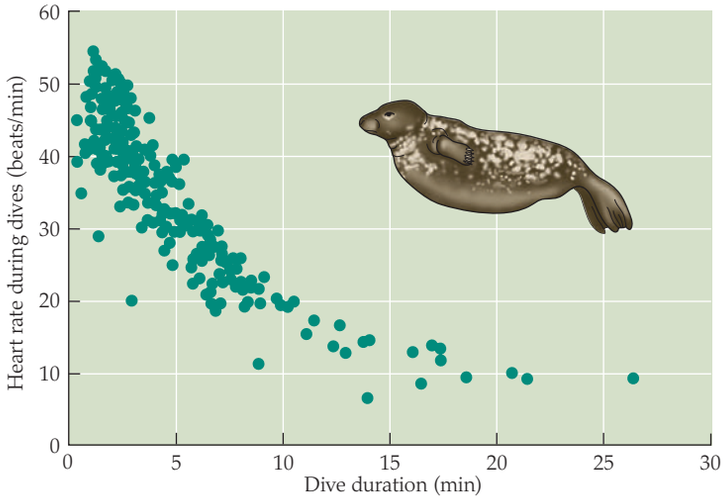


FIGURE 26.10 Diving heart rate varies with dive duration in a graded manner in freely diving seals Grey seals (*Halichoerus grypus*) living freely in the ocean near Scotland were monitored using radio and sonic transmitters. When not diving, their average heart rate was 119 beats/min. During their voluntary dives, the seals always exhibited bradycardia, but they adjusted the degree of bradycardia depending on how long they spent under water. This graded bradycardia is in contrast to the stereotyped bradycardia of forcibly submerged seals, which typically exhibit a maximum or near-maximum drop in heart rate whenever forced under water. (After Thompson and Fedak 1993.)

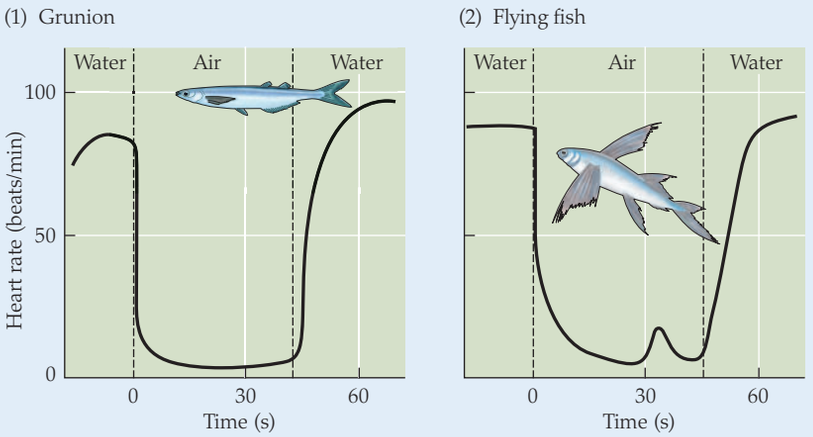
cardiac output to the dimensions of the vascular system requiring blood flow. If this integrated response occurs consistently, then little vasoconstriction would occur in animals that exhibit just a small reduction of heart rate, and progressively greater vasoconstriction would develop as bradycardia becomes more profound. A second line of evidence that corroborates this view is provided by studies of systemic organ function. Although studies of organ function do not tell a completely consistent story, they often indicate that vasoconstriction is graded in voluntary dives. Urine formation by the kidneys, for instance, requires blood flow. Thus one can learn about blood flow to the kidneys during dives by use of chemical markers that permit measurement of urine formation. In free-living Weddell seals, urine formation seems to continue during relatively short dives (indicating continued blood flow) but seems to stop during long dives (indicating cessation of blood flow).

The current working hypothesis of most physiologists who study diving is that the entire suite of cardiovascular responses to voluntary diving occurs in a graded manner. During protracted voluntary dives, free-living animals probably function much like forcibly submerged ones: They undergo profound vasoconstriction accompanied by a profound drop in heart rate, and large parts of the body are cut off from active blood flow. During relatively short voluntary dives, however, vasoconstriction is probably modest, so that only a small reduction in cardiac output is warranted, and most (or all) parts of the body continue to receive blood flow. When all parts of the body receive blood flow, all can share the blood O_2. This means that a dive cannot be of extreme length, but it also means that the animal avoids the stresses of anaerobic catabolism (discussed in Chapter 8 and later in this chapter).

One must wonder how the elaborate cardiovascular responses seen in diving mammals evolved. These responses in fact seem to be specializations of phylogenetically ancient responses to asphyxic conditions, responses that occur widely in vertebrates (**Box 26.1**).

BOX 26.1 THE EVOLUTION OF VERTEBRATE CARDIAC AND VASCULAR RESPONSES TO ASPHYXIA

In the decades following Scholander and Irving's seminal discoveries regarding the diving physiology of marine mammals, Scholander and others sought to learn when the diving responses had originated in the evolution of vertebrates. They were intrigued to find similar responses in fish taken out of water! Two sorts of fish that they studied were flying fish and grunion, both of which breathe with gills but occasionally emerge into air voluntarily. Flying fish sometimes spend tens of seconds out of water as they skitter across the sea surface, and grunion sometimes spend minutes high on beaches where they slither out of the water to mate and lay eggs. The researchers found that both species exhibit profound bradycardia (as the figure shows), plus evidence of peripheral vasoconstriction, when they are out of water. The common denominator between diving marine mammals and these fish is that both are asphyxic (unable to breathe) when they undergo bradycardia and peripheral vasoconstriction. Scholander thus argued that bradycardia and peripheral vasoconstriction first evolved as defenses against asphyxia in the ancestors of modern fish. By now there is a large body of evidence indicating that bradycardia is a typical response of both cartilaginous and bony fish when they are exposed to O_2-poor (hypoxic) water, reinforcing the view that bradycardia is an ancient response of vertebrates to O_2 insufficiency.



The heart rates of fish removed from water The graphs show Scholander's original data for (1) grunion and (2) flying fish. (After Scholander 1964.)

If, in fact, mammals inherited the rudiments of their cardiovascular responses to asphyxia from piscine ancestors, one would guess that those responses would be observed in many kinds of mammals, not just diving ones. In fact, the species of mammals that habitually dive are not alone in undergoing bradycardia and peripheral vasoconstriction when they are unable to obtain O_2 by breathing. Scholander himself reported evidence for bradycardia and peripheral vasoconstriction in neonatal humans and other mammals when they pass through the birth canal, and he observed bradycardia in human pearl divers. Abundant evidence exists today that adult humans and adults of at least some other terrestrial mammals routinely display bradycardia and redistribution of blood flow (e.g., restriction of blood flow to skeletal muscles) when their whole bodies are submerged, or even if they simply immerse their faces in a bowl of water. Humans, however, are not "just like" marine mammals: Human physiological responses to immersion are not as profound as those seen in marine mammals and are not coordinated in the same way (e.g., arterial blood pressure often soars in humans during long breath-hold dives because the output of blood from the heart and the resistance to blood flow through the vascular system are mismatched).

The marine mammals—viewed from the perspectives discussed here—seem in a sense to have "perfected" responses that all or most mammals share and that the mammals may well have inherited from fish. An important objective for future research is to understand better what exactly occurred during this "perfecting" process in the course of evolution and how exactly the control mechanisms in marine mammals differ from those in terrestrial ones.

Red blood cells are removed from the blood between dive sequences in some seals

Species of seals with large blood O_2 stores, such as Weddell seals and ribbon seals (see Figure 26.7), typically have exceptionally large concentrations of red blood cells in their blood *when they are diving*. Although an elevated red blood cell concentration enables the blood to store a large amount of O_2, it has a downside: It increases the viscosity of the blood, forcing the heart to work harder to pump blood. In at least some of the true (phocid) seals, red blood cells are partly removed from the circulating blood and stored in the spleen when the animals are resting at the water's surface or on land. Then the cells are returned to the blood during diving.[5] The blood of a Weddell seal, for instance, although 38% red blood cells (by volume) when the animal is resting in air, may become enriched to 52% red blood cells during diving. The removal of red blood cells from the blood when a seal is not diving means that during such rest periods the heart does not have to work exceptionally hard to pump blood. Red blood cells require at least 10–20 min to move between the spleen and blood. Thus the cells do not move in and out of the blood with each dive, but enter the blood during sequences of dives and are withdrawn during extended periods of rest.

Metabolism during Dives

The stage is now set to discuss metabolism during dives. Is it aerobic or anaerobic, or is it both? In terrestrial mammals that are resting or just modestly active, blood flows freely throughout the body—making O_2 available to all tissues—and all tissues make ATP aerobically. A similar picture seems to exist during short, voluntary dives by many marine mammals, even though they are not breathing; the circulatory system remains open to most or all body regions, and the tissues receive enough O_2 from the body's O_2 stores to function aerobically. A dramatically different picture develops during forced or prolonged dives.

The body becomes metabolically subdivided during forced or protracted dives

The intense peripheral vasoconstrictor response that occurs during forced or protracted dives effectively divides a diving animal's body into two metabolically distinct parts. This subdivision is illustrated in **Figure 26.11** by the classic data that Scholander and Irving gathered on forcibly submerged harbor seals. The tissues that are denied blood flow during submergence, such as the skeletal muscles of the body trunk, initially continue to metabolize aerobically, using local O_2 stores such as O_2 bound to myoglobin. However, as shown by the muscle data in Figure 26.11, the tissues denied blood flow reduce their O_2 supplies to nearly zero long before a dive is over (see Figure 26.11*a*), and simultaneously they start to accumulate lactic acid (see Figure 26.11*b*) as they turn to anaerobic glycolysis to synthesize ATP. The O_2 concentration of the circulating arterial blood in a submerged seal falls much more slowly than the O_2 concentration of the skeletal muscles (see Figure 26.11*a*); therefore, long after the skeletal muscles have exhausted their O_2, the brain, myocardium, and other perfused tissues receive substantial O_2 supplies from the blood. The perfused tissues accordingly remain aerobic and do not produce lactic acid for most of the duration of a dive, as shown by the fact that little lactic acid accumulates in the circulating arterial blood during submergence (see Figure 26.11*b*). All things considered, you can see that well before a long dive is over, a seal's body becomes divided into two regions, one of which remains aerobic while the other becomes O_2-depleted and dependent on anaerobic ATP production.

An important point to note is that lactic acid remains sequestered in the skeletal muscles and other vasoconstricted tissues while a dive is in progress, rather than entering the circulating blood (see Figure 26.11*b*). However, when a dive ends and the diving animal starts to breathe again, blood flow is promptly restored to the vasoconstricted tissues, and the lactic acid accumulated in them is washed out. The washout results in a relatively rapid rise in the lactic acid concentration of the circulating blood, followed

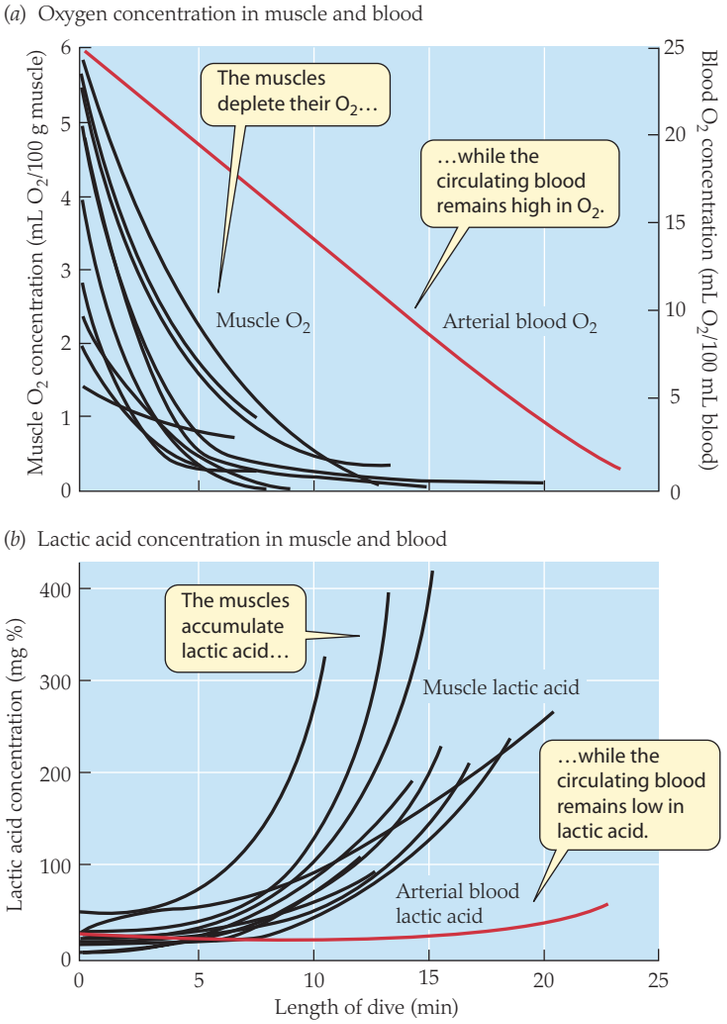


FIGURE 26.11 Metabolic subdivision of the body in seals during forced submergence Levels of O_2 and lactic acid in the dorsal trunk muscles and circulating arterial blood are shown for forcibly submerged harbor seals (*Phoca vitulina*) as functions of their time under water. (*a*) Muscle O_2 concentration in each of ten seals and the average O_2 concentration in circulating arterial blood. (*b*) Muscle lactic acid concentration in each of ten seals and the average lactic acid concentration in circulating arterial blood. (From Scholander et al. 1942.)

[5] Certain terrestrial mammals also vary the red blood cell concentration of their blood by sequestering cells in the spleen, as noted on page 636.

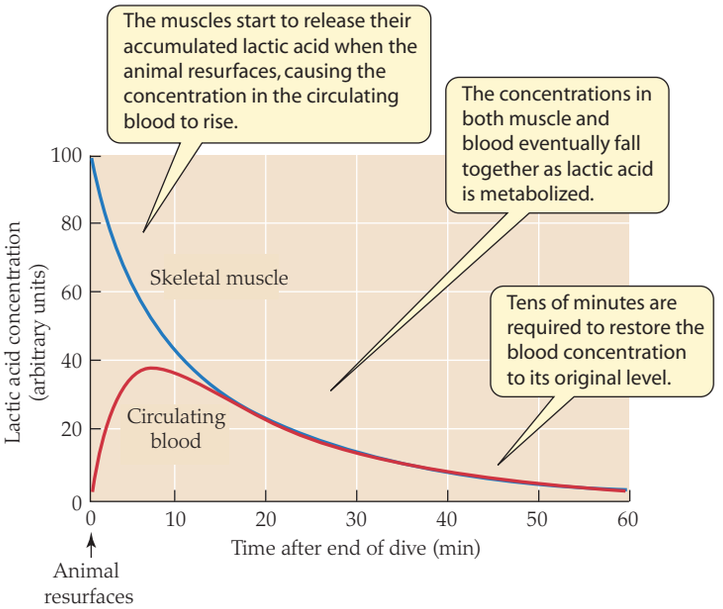


FIGURE 26.12 The aftermath of a prolonged dive: Lactic acid in muscles and blood The graph shows concentrations of lactic acid in skeletal muscles (blue line) and in circulating blood (red line) in the hour following a prolonged dive (a 43-min dive by an adult Weddell seal is assumed). Lactic acid concentrations are expressed relative to the concentration in muscle at dive's end, arbitrarily set equal to 100 units. Blood values are empirical, whereas muscle values are based on the assumption of exponential kinetics. (After Butler and Jones 1997.)

by a gradual decline. Figure 26.12 shows the prevailing view of the processes at work after breathing is resumed. The concentration of lactic acid in the skeletal muscles falls exponentially. After blood lactic acid has risen for several minutes, it also starts to fall because the metabolic processes that clear lactic acid from the blood (see Figure 8.6) eventually outpace the entry of lactic acid into the blood from the muscles. The time scale is highly significant. Because the metabolism of lactic acid is slow (as emphasized in Chapter 8; see page 194), the concentration of lactic acid in muscles and blood may not return to baseline levels *for many tens of minutes after a dive*.

Metabolic limits on dive duration are determined by O_2 supplies, by rates of metabolic O_2 use and lactic acid production, and by tissue tolerances

A diving mammal usually elects to end a dive before its metabolic limits are reached. However, a highly protracted dive could be terminated by exhaustion of O_2 in the part of the body receiving active circulation, by excessive accumulation of lactic acid in anaerobic tissues, or by other metabolic limitations.

Three factors determine the limits of endurance of the O_2-dependent tissues that receive active blood circulation during dives: (1) the magnitude of the O_2 store available to those parts of the body, (2) the rate of use of the O_2 store, and (3) the extent to which the partial pressure of O_2 can fall before impairing function. A scattering of interesting insights are available regarding each of these considerations. For example, even though the myocardium is believed to have a continuous requirement for O_2, it may nonetheless start to employ anaerobic glycolysis to some extent after several minutes of diving. Both this partial recourse to anaerobic catabolism and the drop in cardiac work associated with diving bradycardia reduce the heart's O_2 needs and thus help postpone the time when O_2 supplies become inadequate to sustain myocardial function.

The brain, in contrast to the heart, is believed to be entirely aerobic in diving mammals, just as it is in terrestrial mammals. However, several types of evidence indicate that the brain remains functional at lower O_2 partial pressures in at least some seals than in terrestrial mammals. Recent studies, for example, have been done on the membrane potentials of individual neurons in brain slices taken from a highly defined brain region of hooded seals (*Cystophora cristata*) and lab mice. As discussed in Chapter 12, a relatively normal membrane potential is required for neurons to produce action potentials (impulses), which are essential for brain function. Seal neurons are dramatically more resistant to loss of membrane potential when placed in low-O_2 conditions than are mouse neurons (Figure 26.13). This probably helps explain observations that indicate that seals—of at least some species—are able to remain conscious when blood O_2 has fallen to levels that would cause blackout in most mammals. Neuronal mechanisms that permit the brain cells to maintain conscious function at very low O_2 levels permit the seals to make almost complete use of their O_2 stores and thus dive especially long.

METABOLIC RATE DURING DIVING An animal's metabolic rate during diving is one of the most important factors in determining metabolic limits on dive duration. A low metabolic rate could slow both the rate of O_2 depletion and the rate of lactic acid accumulation, thereby lengthening a dive regardless of whether O_2 or lactic acid sets the limits. Physiologists have sought evidence for depressed metabolism in diving mammals from the dawn of the modern study of diving. Rigorous measurements of metabolic rates during diving are not easy, however, and knowledge of this key subject remains incomplete.

The preponderance of available evidence indicates that submergence commonly brings about a depression of metabolism. Good insight is provided, for example, by studies of free-ranging Weddell seals that were trained to breathe consistently from a monitored source

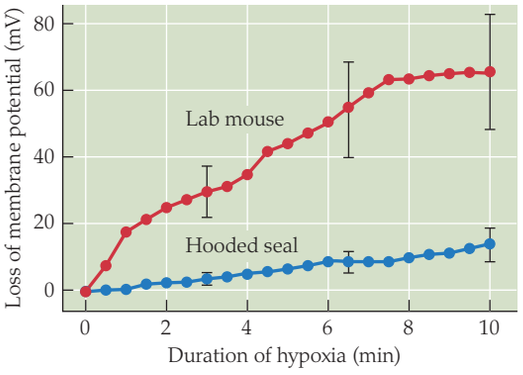


FIGURE 26.13 Loss of normal membrane potential in brain neurons of hooded seals and lab mice during exposure to tissue hypoxia The normal membrane potential in neurons of both species is about 64 mV (inside negative). The *y* axis shows how much of this potential is lost as a function of time after neurons are exposed, starting at time 0, to severe hypoxia. A value of 40 mV on the *y* axis, for example, indicates that membrane potential has fallen by 40 mV, from 64 mV to 24 mV. Error bars show ± 1 standard error. (After Folkow et al. 2008.)

of air for hours at a time as they engaged in repeated voluntary dives (most of which were short enough to be fully aerobic). Remarkably, the average rate of O_2 consumption of these *active* animals was equal to or lower than that of *resting* but nondiving seals!

Depression of metabolism during diving seems paradoxical because swimming ought to increase an animal's metabolic rate. This paradox is far from fully resolved. Hypothermia seems to be a common energy-sparing mechanism among seals: They often let their tissues cool during diving. We will return soon to specific mechanisms by which the diving metabolic rate can be kept low.

ADAPTATIONS TO THE ACCUMULATION OF METABOLIC END PRODUCTS Both the aerobic and anaerobic catabolic pathways produce end products that accumulate in a diving mammal: CO_2 and lactic acid, respectively. The ways in which a diver responds to the buildup of these compounds are important determinants of maximum dive duration.

Acidification is one concern. Both CO_2 and lactic acid tend to cause the pH of tissues and body fluids to decline. How is the pH kept from falling so low during a dive that it forces the dive to end? Part of the answer is that diving species are noted for having particularly high blood buffering capacities.

Stimulation of the urge to breathe is another concern. Anyone who has ever held his or her breath as long as possible under water knows that eventually the urge to breathe becomes impossible to resist. The underlying causes of this phenomenon are the buildup of blood CO_2 and the drop of blood pH: In terrestrial mammals, both factors are potent stimuli for pulmonary ventilation (see page 601). Diving species exhibit blunted (i.e., reduced) ventilatory sensitivity to changes in blood CO_2 and pH compared with terrestrial species. An example is provided by free-ranging harbor seals that were exposed to elevated concentrations of CO_2 in their breathing air. As the CO_2 concentration was raised, the seals increased their ventilation rates, but only about half as much as humans would. A blunted drive to breathe helps a diving mammal stay submerged for long periods.

The Aerobic Dive Limit: One of Physiology's Key Benchmarks for Understanding Diving Behavior

In protracted dives, the accumulation of lactic acid has major behavioral consequences, for three reasons. First, ridding the body of lactic acid requires a lot of time (see Figure 26.12). Second, O_2 is required for the process (see page 191), meaning that a diving mammal typically must stay at the water's surface where it can breathe, or return often to the surface, while it metabolizes lactic acid. Third, if a diving mammal has a lactic acid burden to metabolize following a protracted dive, it cannot engage immediately in a second highly protracted dive. This is so because the lactic acid of a second dive simply adds to the preexisting lactic acid, and there is a cap on the total lactic acid that can be accumulated and tolerated (see Chapter 8).

The lengths of time required to rid the body of lactic acid are impressively long, as already stressed. Table 26.1, for example, lists the lengths of time Weddell seals must stay at the water's surface to fully metabolize various accumulations of lactic acid—a requirement to regain their full range of behavioral options.

TABLE 26.1 Average surface times required for adult Weddell seals to dissipate accumulations of lactic acid

Lactic acid accumulation (mg lactic acid/100 mL blood)[a]	Time required to return to resting level[b] (min)
20	11
40	27
80	70
120	105
145	120

Source: After Kooyman et al. 1980.

[a]Lactic acid accumulation refers to the peak blood concentration measured during dive recovery. This concentration provides a reasonable estimate of the total-body accumulation.

[b]The times listed are for seals that remain continuously at the water's surface until the blood concentration of lactic acid is restored to resting. The resting level of lactic acid is about 5 mg/100 mL blood.

Accumulation of lactic acid can be avoided if dives are kept short. This is illustrated in Figure 26.14, which shows one of the most famous and important sets of results ever reported in the study

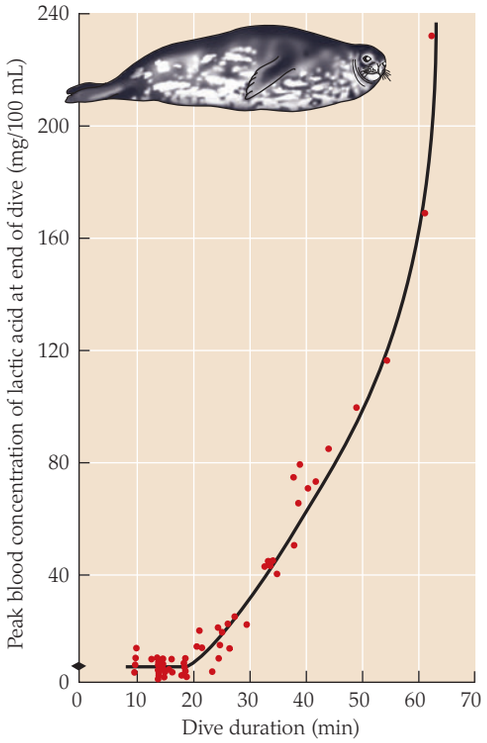


FIGURE 26.14 Peak concentration of lactic acid in arterial blood of freely diving, adult Weddell seals following dives of various durations Because the circulatory system is fully open after a dive, lactic acid produced anywhere in the body during a dive, even if temporarily sequestered while a seal was submerged, appears in the arterial blood after a dive. The peak blood concentration after a dive therefore provides an estimate of the total-body net production of lactic acid during the dive. The diamond on the *y* axis marks the average resting concentration of lactic acid in arterial blood. (After Kooyman et al. 1980.)

of diving physiology. To gather the data shown, Gerald Kooyman and his colleagues drew blood from wild Weddell seals when the seals returned to ice holes to breathe following voluntary dives. Recall that although lactic acid may be sequestered within skeletal muscles *during* a dive, it is released into the general blood circulation immediately *after* a dive (see Figure 26.12). The investigators simply waited long enough after each dive for the release process to drive the blood concentration to its peak level before they drew a seal's blood. Then they could estimate the seal's total-body accumulation of lactic acid from the blood concentration. Put another way, the total-body accumulation and the peak blood concentration are highly correlated. Figure 26.14 shows how the peak blood concentration of lactic acid varies with dive duration. Weddell seals accumulate lactic acid when they dive for 25 min or longer. In fact, they accumulate very high levels during lengthy dives. However, they do not accumulate lactic acid above the resting, baseline level if they dive for 20 min or less.

Based on the data shown in Figure 26.14, Kooyman and his colleagues defined the concept of the **aerobic dive limit** (**ADL**), which is the longest dive that can be undertaken without a net accumulation of lactic acid above the resting level. In adult Weddell seals, the ADL is 20–25 min. Unfortunately, measuring lactic acid levels in freely diving animals is a challenge, and plots like the one in Figure 26.14 are available for few species. Investigators therefore sought alternative methods to estimate the ADL. The most popular alternative is to assume that a dive will not cause accumulation of lactic acid if the O_2 cost to meet all the metabolic demands of the dive is less than the total available O_2 store, and to then calculate the ADL from the O_2 store and diving metabolic rate. Estimates based on the two methods are not always similar. To distinguish them, investigators usually use the expressions *ADL* and *cADL*; in this system, *ADL* symbolizes a value based on direct measures of lactic acid, whereas *cADL* symbolizes a value calculated from O_2 stores.[6] Here, for simplicity, we will simply use *ADL* to refer to the length of the longest dive possible without net accumulation of lactic acid, regardless of how it has been estimated.

Just as species of marine mammals vary widely in their diving competence, they also vary widely in their ADLs. Compared with the ADL of Weddell seals, the ADLs of fur seals and California sea lions (both otariids), for example, are far shorter, about 4–6 min. In contrast, ADLs in some species are far longer. Some fairly crude estimates indicate that sperm whales and male southern elephant seals have ADLs of 40–50 min!

Why does the ADL matter? *A central hypothesis of modern diving physiology is that it is adaptive for diving mammals to keep most of their dives shorter than their species-specific ADL.* By keeping their dives fully aerobic, they avoid lengthy recovery times to metabolize lactic acid. Accordingly, they can minimize their time at the surface and maximize their time under water.

How exactly do fully aerobic diving patterns translate into increased underwater time? When a dive is fully aerobic, replenishing O_2 stores is the only task an animal must carry out to recover. Vigorous breathing can replenish body O_2 stores rapidly. In fact, based on observations on several species, marine mammals require only 1–4 min to recover at the water surface—between one dive and the next—*when their dives are shorter than the ADL*. Short surface intervals, in turn, provide lots of underwater time for foraging. Consider, for instance, an adult Weddell seal that undertakes six 15-min dives. Because these dives are shorter than its ADL and fully aerobic, the seal will need to be at the surface for only about 4 min between dives. Thus, counting dive time and recovery time, the six dives will take 114 min, of which 90 min will be spent under water. The seal will therefore be able to spend 80% of its time under water, foraging and feeding. Now let's look, in contrast, at the seal's use of time if it dives just once during 114 min for as long as it can while still being fully recovered when the total time is over. A 44-min dive will cause accumulation of lactic acid: specifically about 80 mg per 100 mL of blood (see Figure 26.14). This accumulation of lactic acid, to be metabolized, will require about 70 min of recovery at the surface (see Table 26.1). A 44-min dive and recovery will therefore take the entire 114 min. This total time is the same as required for the six 15-min dives. However, the seal diving for 44 min will spend 40%—not 80%—of its time under water! In brief, because of the long recovery times necessitated by anaerobic metabolism, a diver can typically spend a greater fraction of its time foraging and feeding if it makes many fully aerobic dives, each shorter than its ADL, than if it makes just a few lengthy dives.

Dives shorter than the ADL are also postulated to be adaptive because they permit homeostasis to be maintained throughout the body with little or no interruption. As we discussed earlier, investigators think that most or all organs receive a continuing blood flow and O_2 supply during dives that are relatively short.[7] Short dives, therefore, permit most or all organs to continue functioning in an approximately normal way. Protracted dives, by contrast, can force many organs away from homeostasis; for instance, in a protracted dive, the kidneys may be forced to stop urine production, and enzymes throughout the body may be forced to function at highly altered pH.

If dives shorter than the ADL are, in fact, adaptive as hypothesized, then diving mammals are expected to elect short dive lengths during their natural diving behavior. With this thought, we come full circle. We stressed at the start of this chapter that dives of maximum duration, although important and amazing, tend to be uncommon; most dives are far shorter than the species-specific maximum. Now we can ask more specifically whether most dives are shorter than the species-specific ADL, as expected. Often (although not always), the answer is yes. For example, about 95% of the voluntary dives of wild, adult Weddell seals are shorter than the 20- to 25-min ADL of adult Weddell seals. Adult grey seals have a shorter ADL, about 10 min, and more than 90% of their dives are shorter than 10 min. The ADL of adult bottlenose dolphins is about 4 min, and more than 90% of their dives are shorter than 4 min.

The aerobic dive limit—a *physiological* feature of marine mammals—is therefore an important *behavioral* benchmark. Animals may dive for far longer than their ADL when faced with extraordinary behavioral challenges, such as avoiding danger or searching for new foraging areas. However, dives that are longer than the ADL

[6]There is now a push to distinguish the two methods more emphatically, by using a new expression, the *diving lactate threshold* (*DLT*), to refer to estimates from lactic acid.

[7]This is not to say that blood flow is unaltered. Flow is likely to be redistributed during short dives much as it is redistributed in terrestrial animals during exercise, with some organs receiving relatively more of the total flow than they do during rest and some receiving less. Nonetheless, the current working hypothesis is that blood flow (at one rate or another) is maintained to most or all organs during short dives.

require long recovery times and force many organs away from homeostasis. Therefore, dives are usually kept shorter than the ADL. The significance of large body O_2 stores becomes clearer in this light: Large O_2 stores give a species a high ADL, thereby permitting a greater range of diving options while animals adhere to the "rule" that the ADL is the upper length limit for the majority of dives.

Marine mammals exploit multiple means of reducing their metabolic costs while under water

Intense study of ADL in recent years has led to a renewed focus on the metabolic rates of diving mammals while they are submerged. This is true because the underwater metabolic rate is one of the principal determinants of the ADL. To illustrate, consider a seal that has a usable O_2 store of 3000 mL. If its metabolism is fully aerobic and its metabolic rate during diving is 300 mL O_2/min, its ADL is 10 min. If its metabolic rate could be halved, its ADL would be 20 min.

A lot of contemporary research on the diving metabolic rate is focused on mechanisms by which animals might reduce their metabolic rates—and therefore their rates of O_2 consumption—while they are under water. How, in other words, might they reduce their metabolic costs while diving? Three potential costs are those of keeping warm in cold water, processing food captured during diving, and swimming. Here we mention some results of recent studies on all three costs.

- *Keeping warm in cold water*. Earlier we noted that seals often let their body temperatures decline during diving. In hooded seals (*Cystophora cristata*), at least, investigators have recently shown that tissue cooling is actually promoted during diving because shivering is inhibited. Faced with a particular cold stress, the seals shiver when on land to keep warm, but they do not shiver under water. In this way, they avoid a metabolic cost that would consume their O_2 stores while diving and shorten their ADL.
- *Processing food*. A provocative recent report indicates that grey seals, when capturing prey under water, postpone processing their prey until they are breathing air. In this way, they do not need to meet the O_2 costs of specific dynamic action (see Figure 7.5) while they are diving.
- *Swimming*. Swimming costs are currently the best understood of the topics discussed here. By employing miniature video cameras and other high-technology devices, investigators have established in the last 15 years that seals and dolphins often limit their costs of underwater travel by employing gliding (**Figure 26.15a**) and other high-efficiency modes of locomotion. A principle taught to all scuba divers is that as soon as a diver becomes negatively buoyant, sinking becomes self-reinforcing; a little sinking compresses air cavities in the diving gear, making a diver less buoyant and even more prone to sinking. The lungs of seals, positioned within a flexible thorax, behave as compressible air cavities, and seals seem often to employ the self-reinforcing nature of negative buoyancy to sink, holding their bodies almost motionless as they glide downward. Animals that glide to depth (see Figure 26.15a)

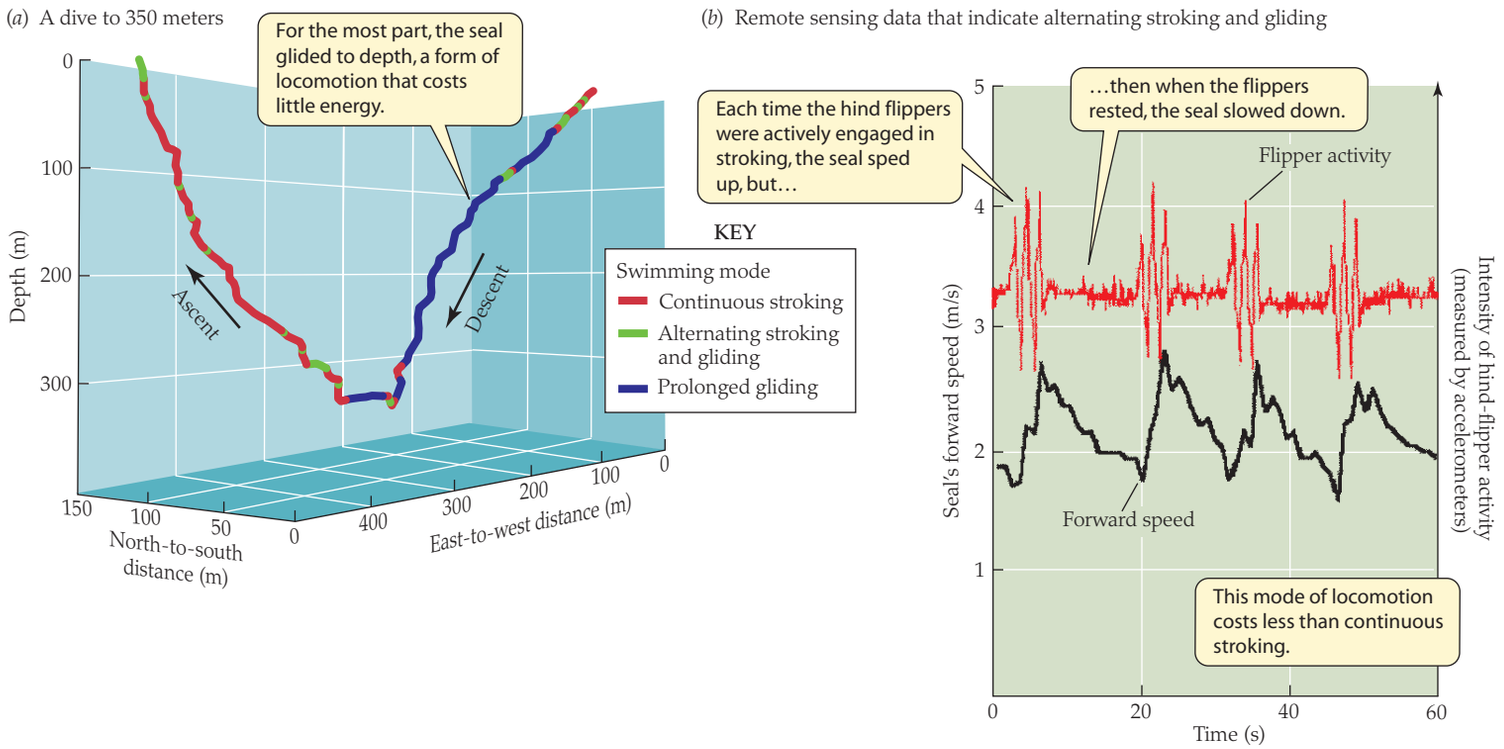


FIGURE 26.15 Energy-sparing behaviors of freely diving Weddell seals Behaviors that spare energy also spare O_2. (*a*) A three-dimensional record of a dive by a Weddell seal, color coded to record the type of locomotion at each stage. Data were gathered by use of video cameras, accelerometers, and other remote-sensing devices attached to each seal that was studied. (*b*) Simultaneous records from remote detectors of forward speed and hind-flipper activity, showing the type of evidence that is interpreted as indicating stroke-and-glide locomotion. (Both parts after data of Randall Davis, Lee Fuiman, Markus Horning, and Terrie Williams, with gratitude.)

avoid the metabolic costs (e.g., O_2 costs) of using muscle power to descend. Research has also shown that the cost of swimming is lower if seals alternately propel themselves and glide, rather than propelling themselves by muscle power continually. Telemetry data indicate that seals in fact often swim by the energy-sparing mechanism of alternately stroking with their flippers and gliding (**Figure 26.15*b***).

Decompression Sickness

Many "diver's diseases" have been described in the annals of human diving, and physiologists have long been concerned about whether and how these diseases are avoided by marine mammals. Although nitrogen narcosis (altered cognitive function attributed to high N_2 partial pressure) is starting to receive attention in studies of marine mammals, the disease that has been studied the most is *decompression sickness*, also called the *bends*. An informative starting point is to examine the etiology of this disease in human divers.

Human decompression sickness is usually caused by N_2 absorption from a compressed-air source

Unambiguous cases of decompression sickness occur in humans when they are diving with a source of compressed air (such as a scuba tank) that steadily resupplies the lungs with air. During a dive, the compressed-air source maintains the air pressure in the lungs at a level high enough to equal the ambient water pressure at all depths. This arrangement prevents the lungs from collapsing under the force of the ambient pressure and allows continued breathing. It also, however, means that an unusually elevated N_2 partial pressure is maintained continuously in the lung air. Assuming that a diver is breathing pressurized ordinary air, 78% of the air is N_2. Thus, if the total pressure at depth is 3 atmospheres (atm), the partial pressure of N_2 is 2.3 atm; and if the total pressure is 5 atm, the partial pressure of N_2 is almost 4 atm. Because these elevated N_2 partial pressures are maintained continuously in the lungs during dives with a compressed-air source, all the body tissues gradually come to equilibrium with them by dissolving more N_2 than ordinary. If a dive is long enough, all tissues will in fact dissolve enough N_2 to have a N_2 partial pressure equal to that in the lung air.

Decompression sickness may then occur if the person suddenly surfaces ("decompresses"). Under such circumstances, as explained in Chapter 22 (see page 573), outgassing of N_2 from solution can cause macroscopic bubbles to form within the blood and other tissues, much as bubbles form within a bottle of soda water when the cap is removed and the contents are decompressed. Bubbles formed in this way are generally believed to be the primary agents of decompression sickness. The most common symptom is throbbing pain in the joints and muscles of the arms and legs (the "bends"). In addition, an afflicted person may have neurological symptoms, such as paralysis, and severe breathing problems (the "chokes"). Exactly how the bubbles cause these symptoms remains a topic of ongoing research. Bubbles can block blood flow, press on nerve endings, and even disturb the structures of proteins because of electrical phenomena at gas–water interfaces.

The factors that determine whether *clinical* decompression sickness occurs are not completely known. One consideration is that, after a person surfaces, the N_2 partial pressure in his or her lung air is restored to its ordinary value, and the N_2-charged blood and other tissues start to lose N_2 into the lung air. In this way, excess dissolved gas is steadily eliminated across the lungs. If the N_2 overload is not too great, this elimination may lower the N_2 partial pressure in the blood and other tissues rapidly enough that even if macroscopic bubbles start to form, their growth and proliferation are halted before clinical symptoms occur. As a very rough rule of thumb, humans can surface immediately without fear of the bends if their blood and tissue N_2 partial pressure is less than 2 atm. Otherwise they must alter their behavior and surface gradually to avoid illness (which in severe cases can be fatal).

Breath-hold dives must be repeated many times to cause decompression sickness in humans

When we consider humans undergoing *breath-hold* diving, a crucial difference from diving with compressed air is immediately apparent. A breath-hold diver descends with only the limited amount of extra N_2 contained within his or her lung air upon submergence; his or her pulmonary N_2 supply is not steadily renewed. During descent to depth, the lungs of a breath-hold diver are compressed under the force of the increasing ambient pressure, and the N_2 partial pressure in the lungs increases initially to high levels, just as in diving with compressed air. This process creates a partial-pressure gradient favoring the transfer of N_2 from the lungs to the blood and other tissues. However, the *quantity* of N_2 in the lungs is limited, and thus only a limited quantity can be transferred. In humans, the amount of N_2 that is transferred to the blood and other tissues during a single breath-hold dive is far too small to cause decompression sickness.

What happens, however, if a person undergoes *many repeated* breath-hold dives? If the time between successive dives is insufficient for the tissues to release accumulated dissolved N_2 after each dive, the tissue N_2 partial pressure can conceivably be elevated *in a series of upward steps* to a threatening level. This possibility seems to be far from theoretical. Several reports exist of people who have developed symptoms of decompression sickness after sequences of many breath-hold dives. In a classic instance described by Poul-Erik Paulev (1935–), for example, an individual complained of decompression symptoms after diving about 60 times to depths of 15–20 m over a period of 5 h.

Marine mammals have been thought—perhaps erroneously—to avoid decompression sickness during deep dives by alveolar collapse

Marine mammals are breath-hold divers. We must wonder, then, if they dive in ways such that N_2 transfer to their tissues by breath-hold dives might additively become great enough to cause decompression sickness. Calculations and experiments indicate that if N_2 is presumed to be able to move freely from the lungs into the rest of the body, N_2 partial pressures in the blood and other tissues of a diving mammal may often rise to threatening levels following many repeated dives.

Evidence indicates, however, that during deep dives, decompression sickness is in fact generally prevented in marine mammals. How? For 50 years, *alveolar collapse* has been considered the primary mechanism. Because of the compressibility of the thorax in marine mammals, the volume of the air in their lungs decreases as the animals descend during diving (see Figure 26.6). Moreover,

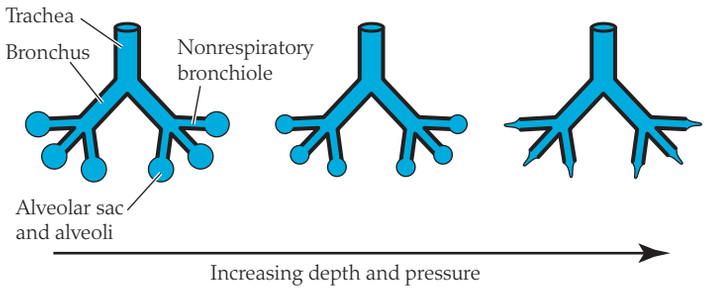


FIGURE 26.16 The hypothesis of preferential collapse of the alveoli and alveolar sacs at depth As the lung air is compressed to a smaller volume at depth, the alveoli and alveolar sacs collapse preferentially, and all air moves into the trachea, bronchi, and nonrespiratory bronchioles—the conducting airways (anatomical dead space)—where gas exchange with the blood is negligible.

because of distinctive patterns of structural reinforcement in their lungs, the *respiratory* parts of the lungs (see Figure 23.18) are far more prone to collapsing shut than any of the other lung parts. These factors taken together have been hypothesized to mean that as the animals descend to great depths, their lung air comes to be contained entirely in the *conducting* (nonrespiratory) airways of their lungs (**Figure 26.16**). In this way, below a certain depth, N_2 transfer from the lungs into the blood and other tissues cannot occur, because the N_2 in the lung air is safely sequestered in the parts of the lungs where gas exchange between the lung air and blood is not possible. Oxygen, as we have already stressed, is also sequestered within the conducting airways. The sequestration of O_2 might simply be a price an animal must pay to avoid decompression sickness, or possibly—as discussed in the next section of this chapter—it may hold advantages of its own.

Without doubt, alveolar collapse helps prevent N_2 in the lungs from dissolving in the blood and other body fluids by sequestering the N_2 in the lungs. But how complete is this sequestration? Until a decade ago, physiologists believed that alveolar collapse occurs at relatively low ambient pressures and thus occurs quickly as an animal descends into deep waters (e.g., by the time 50 m is reached).

From that viewpoint, only shallow dives could present a problem. Certain marine mammals, (e.g., some dolphins, fur seals, and sea lions) dive repeatedly to depths that, although sufficient to elevate the pulmonary N_2 pressure to levels of questionable safety, are clearly too shallow to induce full alveolar collapse. During repeated shallow dives of this sort, N_2 might invade the blood and other tissues sufficiently to pose a risk of decompression sickness. Experiments on trained bottlenose dolphins undergoing repeated dives to modest depths suggest that this worry is realistic. After an hour of shallow diving, the animals developed muscle N_2 partial pressures—1.7 to 2.1 atm—that in a human would be only marginally safe.

Deep dives are now a new concern. Under the postulated scenario in Figure 26.16, deep dives would not be a worry: The alveoli would collapse shut early in each dive, sequestering N_2 in the lungs. However, recent empirical evidence indicates that full collapse may not reliably occur at as shallow depths as previously thought during deep dives. Ultrasound scans for bubbles, for example, reveal that some deep-diving species have bubbles in their tissues when they have returned to the sea surface after deep dive sequences.

If a purely *physical* mechanism (alveolar collapse) turns out not to be fully adequate to prevent decompression sickness, *behavioral* prevention may be required. Physiologists have started to wonder if diving marine mammals need to manage the threat of decompression sickness behaviorally just as human divers must. If a person accumulates excess dissolved N_2, he or she slows ascent so N_2 can outgas from the tissues and body fluids without formation of clinically consequential bubbles. Perhaps that is what diving mammals do.

Decompression sickness is an unresolved phenomenon

Decompression sickness in marine mammals is clearly one of the phenomena that remain far from fully understood. It is in the spotlight now because, among other things, certain beaked whales seem to develop widespread bubble formation in their tissues after deep diving while being exposed to high-intensity sonar signals of human origin. Such bubble formation strongly suggests that the whales accumulate excess dissolved N_2 in their tissues during diving. The sonar could conceivably destabilize the dissolved state. Alternatively, it might disorient the animals so much that their normal behaviors are disrupted and they ascend too rapidly. Efforts to understand these phenomena have accentuated how little is confidently known about diving diseases in marine mammals.

A Possible Advantage for Pulmonary O_2 Sequestration in Deep Dives

A recent hypothesis regarding the relation between alveolar collapse and lung O_2 provides a thought-provoking note on which to end this chapter. From the beginning of the scientific study of marine mammals, investigators have tended to perceive the lungs as analogous to scuba tanks. Only a defective scuba tank would fail to deliver all its O_2. Thus, when lungs fail to deliver all their O_2, that failure is perceived as a flaw. Alveolar collapse in deep-diving marine mammals, in this view, is simultaneously advantageous and flawed. It is advantageous because even if not perfect, it helps prevent much of the lung N_2 from dissolving in the blood. It is flawed because it denies an animal the use of some of its O_2 store. Translated into evolutionary terms, this view presupposes that alveolar collapse was favored by natural selection as a means of preventing decompression sickness, and that the sequestration of O_2 is simply a negative side effect. The new hypothesis postulates that in deep divers, O_2 sequestration by alveolar collapse is itself an advantage.

As a diving mammal ascends to the water's surface at the end of a deep dive, the total pressure in its lungs declines as its lungs expand. The partial pressure of O_2 in the lung air accordingly decreases precipitously as the mammal ascends. The *amount* of O_2 in the lung air needs to be great enough that the O_2 partial pressure does not fall to a dangerously low level during this process. To illustrate, suppose that during a dive to 400 m, a seal starts with 0.1 mol of O_2 in its lungs but uses 95% of it, leaving only 5%. At 400 m, the O_2 partial pressure in its lung air will still be higher than the normal value at the sea surface because of the compression of the lung air at depth. As the seal nears the surface at the end of its dive, however, the O_2 partial pressure in the expanding lung air will fall to be only 5% of normal, a level low enough to cause blackout (unconsciousness), even in some seals. Moreover, with the lung air so dilute in O_2, O_2 might easily diffuse *from the blood into the lung air*!

Thus, even if the blood is at a high enough O_2 partial pressure to prevent blackout when ascent begins, the blood O_2 partial pressure might fall to blackout-inducing levels before ascent is completed. These worries suggest that it is important for the lungs to retain a sizable portion of their O_2 throughout a deep dive. Thus, in addition to impeding transfer of N_2 to the seal's tissues and body fluids, alveolar collapse may in fact be a mechanism for preventing the risk of blackout during ascent by ensuring that enough O_2 remains in the lungs to keep the O_2 partial pressure acceptably high in the ever-expanding pulmonary air at the dive's end.

One reason to end on this novel note is that these thoughts emphasize the highly *interactive* nature of the challenges faced by the lungs, blood, and other tissues during all stages of diving. A second reason is to stress the importance of striving to see physiological challenges from the point of view of the animals studied. Lungs cease to look like mere scuba tanks when viewed from the perspective of a deep-diving marine mammal.

Study Questions

1. Comparative physiology is sometimes defined as being the identification and use of "ideal" species for the study of each phenomenon of interest. The Weddell seal is the best known of all diving mammals. In what ways has it been the "ideal" species for the study of voluntary diving in the wild?
2. Outline the pros and cons of carrying lots of lung air during a dive.
3. Based on the study of O_2 needs and stores, the aerobic dive limit (ADL) for young Weddell seals weighing 140 kg is calculated to be 10 min, whereas that for fully grown 400-kg Weddell seals is calculated to be about 20 min. Why might small individuals in general be expected to have shorter ADLs than large individuals? To carry out an empirical study of the changes in ADL during postnatal development in a species, what experiments and measurements would you plan?
4. There is evidence that marine mammals practice unihemispheric sleep: sleep that occurs in only one brain hemisphere at a time, so that while one hemisphere sleeps, the other is awake. Such sleep is essentially unheard of in terrestrial mammals (although common in birds). What might be the advantages of unihemispheric sleep for a marine mammal?

Go to **sites.sinauer.com/animalphys3e** for box extensions, quizzes, flashcards, and other resources.

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