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The marine mammal dive response is exercise modulated to maximize aerobic dive duration

Randall W. Davis · Terrie M. Williams

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Abstract When aquatically adapted mammals and birds swim submerged, they exhibit a dive response in which breathing ceases, heart rate slows, and blood flow to peripheral tissues and organs is reduced. The most intense dive response occurs during forced submersion which conserves blood oxygen for the brain and heart, thereby preventing asphyxiation. In free-diving animals, the dive response is less profound, and energy metabolism remains aerobic. However, even this relatively moderate bradycardia seems diametrically opposed to the normal cardiovascular response (i.e., tachycardia and peripheral vasodilation) during physical exertion. As a result, there has been a long-standing paradox regarding how aquatic mammals and birds exercise while submerged. We hypothesized based on cardiovascular modeling that heart rate must increase to ensure adequate oxygen delivery to active muscles. Here, we show that heart rate (HR) does indeed increase with flipper or fluke stroke frequency (SF) during voluntary, aerobic dives in Weddell seals ($HR = 1.48SF - 8.87$) and bottlenose dolphins ($HR = 0.99SF + 2.46$), respectively, two marine mammal species with different evolutionary lineages. These results support our hypothesis that marine mammals maintain aerobic muscle metabolism while swimming submerged by combining elements of both dive and exercise responses, with one or the other predominating depending on the level of exertion.

R. W. Davis (✉)
Department of Marine Biology, Texas A&M University, OCSB, 200 Seawolf Parkway, Galveston, TX 77553, USA
e-mail: davisr@tamug.edu

T. M. Williams
Center for Ocean Health, University of California, 100 Shaffer Road, Santa Cruz, CA 95060, USA

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Abbreviations

ECG	Electrocardiogram
Hb	Hemoglobin
HR	Heart rate (min^{-1})
kPa	Kilopascal
Mb	Myoglobin
O_2	Oxygen
ODBA	Overall dynamic body acceleration
P_{50}	Oxygen partial pressure (mm Hg or kPa) at 50 % saturation
RMSD	Root-mean-square difference
SF	Flipper or fluke stroke frequency (min^{-1})
VHF	Very high frequency
$\dot{V}_{O_2}$	Rate of oxygen consumption (ml O_2 min^{-1} kg^{-1})
$\dot{V}_{MO_2}$	Multiple of resting skeletal muscle oxygen consumption

Introduction

There has been a long-standing paradox regarding how aquatic mammals and birds are able to support aerobic muscle metabolism during voluntary dives (Castellini et al. 1985; Butler 1988). The contradiction arises from the pulmonary and cardiovascular changes associated with the dive response, which include cessation of breathing (apnea), a decrease in heart rate (bradycardia), a concurrent reduction in cardiac output, and peripheral vasoconstriction that maintains central arterial blood pressure while maintaining blood flow primarily to the heart, lungs, and brain

(Scholander 1940; Butler and Jones 1997). These pulmonary and cardiovascular changes are contrary to the normal response (i.e., hyperventilation, tachycardia, an increase in cardiac output, and peripheral vasodilation to active muscles) exhibited by terrestrial mammals and birds during exercise.

During forced submersion and very long voluntary dives, bradycardia can be profound (i.e., sometimes <10 % of resting heart rate) and organ perfusion is reduced to negligible levels except for the brain, heart, lungs and adrenal glands (Kerem and Elsner 1973; Zapol et al. 1979; Blix and Folkow 1983; Hill et al. 1987). The major effect of these cardiovascular changes is to decrease convective oxygen transport to most tissues to conserve oxygen for the brain and heart, two organs that are sensitive to oxygen deficiency and essential for survival when faced with the threat of asphyxia (Elsner and Gooden 1983). A similar asphyxial response occurs in all vertebrates, including fish when they experience hypoxic water or are removed from water and unable to oxygenate their blood (Leivestad et al. 1957). Thus, these cardiovascular adjustments appear to be a primitive response designed to prolong life when breathing is interrupted and were probably involved in the early stages of the evolution of terrestrial life in the vertebrates (Gordon et al. 1969). Indeed, Scholander (1964) called this striking cardiovascular defense against asphyxiation in vertebrate animals the "Master Switch of Life". Although it can prolong life, it causes peripheral tissues to switch to anaerobic glycolysis with a simultaneous increase in lactic acid, reduces or stops abdominal organ (e.g., liver and kidney) function, and disrupts physiological homeostasis (Scholander 1940; Butler and Jones 1997; Davis et al. 1983). Hence, these circulatory and metabolic adjustments cannot be sustained indefinitely and are stressful to the animal (Soini et al. 1992).

The response during most voluntary dives by marine mammals is less profound than during forced submersion (Butler and Jones 1997; Hill et al. 1987; Kooyman and Campbell 1972). Organs continue to function, energy metabolism remains aerobic and, consequently, physiological homeostasis is maintained (Davis et al. 1983; Kooyman et al. 1980; Qvist et al. 1986). However, as Castellini et al. (1985) pointed out, even the relatively moderate bradycardia and associated reduction in cardiac output observed during the majority of voluntary dives seem diametrically opposed to the normal cardiovascular response to exercise.

This apparent physiological conflict led us to ask the question: If blood oxygen is not conserved for brain and heart metabolism during normal diving, then what is the purpose of the dive response? We previously attempted to answer this question by modeling convective oxygen transport and tissue oxygen consumption in Weddell seals during aerobic dives (Davis and Kanatous 1999). Based on model results, we concluded that the primary role of the dive response is to regulate the degree of hypoxia in skeletal muscle so that blood and muscle oxygen (oxy-hemoglobin and oxy-myoglobin, respectively) stores can be effectively used at different levels of exercise to maximize aerobic dive duration. Theoretically, this is achieved by adjusting cardiac output and convective oxygen transport to muscle according to the rate of muscle oxygen consumption. At higher levels of muscle oxygen consumption during exercise, heart rate, cardiac output, and muscle perfusion must increase to allow the coordinated depletion of available blood and muscle oxygen stores. Kooyman (1985) suggested a similar increase in convective oxygen transport to active muscle, but did not attempt to quantify or model blood flow.

Although this may sound similar to a normal exercise response in terrestrial vertebrates, there are important differences in the diving animal. On the one hand, a dive response with bradycardia is still necessary to reduce convective oxygen transport and lower the partial pressure of oxygen in muscle. This promotes the release of myoglobin-bound oxygen and maximizes aerobic dive duration. On the other hand, heart rate must increase (i.e., bradycardia is less intense) with the level of physical exertion to ensure that the active muscle receives additional blood oxygen (ca. 50 % of the muscle's oxygen comes from the blood) to supplement the simultaneous use of myoglobin-bound oxygen to maintain aerobic metabolism (Davis and Kanatous 1999).

The ability to test this hypothesis by simultaneously measuring heart rate (HR) and instantaneous physical exertion (flipper or fluke stroke frequency, SF) during voluntary dives has only recently become possible for marine mammals. In earlier studies, researchers measured HR and either swimming speed or total oxygen consumption (at the surface and submerged) in captive harbor and gray seals (*Phoca vitulina and Halichoerus grypus*, respectively) and California sea lions (*Zalophus californianus*) swimming in a water flume (Fedak 1986; Williams et al. 1991; Butler et al. 1992). HR oscillated between surface tachycardia and submerged bradycardia, and the interval between breathing bouts often decreased with increasing swim speed until the animal was swimming at the surface, a behavior that made flume studies difficult to interpret. Although average HR over complete dive cycles (i.e., surface and submerged swimming) increased linearly with oxygen uptake, there was no clear relationship between *submerged* HR and work load. Studies of diving HR in ocean-trained California sea lions, Weddell seals (*Leptonychotes weddellii*) making voluntary dives, and free-ranging elephant seals (*Mirounga angustirostris*) were also inconclusive about the effects of physical exertion

because, as with the flume studies, SF was not measured (Hill et al. 1987; Andrews et al. 1997; Ponganis et al. 1997). Hindle et al. (2010) measured HR rate and overall dynamic body acceleration (ODBA) in Steller sea lions (*Eumetopias jubatus*) trained to make shallow dives and then swim horizontally at depth. Their results showed that HR and ODBA, which includes SF as one component, were correlated, but only under certain circumstances. However, their method of recording HR without a complete electrocardiogram (ECG) was susceptible to artifacts from muscle noise associated with movements of the animal, and the authors suggested that this may have influenced their results. In addition, the trained dives were relatively short and shallow, and surface effects (i.e., ventilatory tachycardia between dives and release of the dive response just prior to surfacing) may have affected the analysis of HR and ODBA. Overall, the inability to record SF, which is an indicator of instantaneous effort in working muscles, and a high-resolution, unambiguous HR based on a simultaneous ECG have hampered the efforts to understand the relationship between HR and physical exertion, especially during voluntary, aerobic dives.

Here, we tested the hypothesis that HR increases with the level of physical exertion during voluntary dives by using novel, animal-borne instruments that simultaneously record HR using a continuous ECG, SF using two- or three-axis accelerometers, and dive depth in two marine mammal species. We chose the Weddell seal (Order: Carnivora) and the bottlenose dolphin (*Tursiops truncatus*; Order: Cetacea) to compare diving mammals representing two independent evolutionary lineages. SF was chosen as a metric for physical exertion because it is correlated with locomotory performance and oxygen consumption for a wide range of mammals including terrestrial mammals (Taylor et al. 1980), semi-aquatic runners and swimmers (Williams 1983), as well as diving Weddell seals (Williams et al. 2004) and bottlenose dolphins (T.M. Williams, unpubl. data).

Materials and methods

Animals

We studied two adult, male Weddell seals (mean mass = 268 kg) in McMurdo Sound (77.729 S lat., 166.596 E long.), Antarctica, from September to October 2009. The four adult, male bottlenose dolphins (mean mass = 205 kg) were housed and trained at Disney's EPCOT Living Seas exhibit, Orlando, Florida.

Heart rate monitor

To avoid the problems caused by motion-induced artifacts such as muscle noise when measuring HR, we used custom-built instruments that recorded a continuous ECG at high resolution (Fig. 1a). For both Weddell seals and bottlenose dolphins, the small (16-cm long, 3-cm diameter, 250 g), water-proof HR recorder (UUB/4-EIAP ECG/IBI/Acceleration/Pressure Recording System, UFI, Morro Bay, CA, USA) was attached to the animal, and the ECG was recorded at 50 Hz by attaching two, waterproof electrodes to the skin (see details below). The HR recorder also contained a pressure (depth) transducer and either a two- or three-axis accelerometer that recorded high-resolution lateral (Weddell seal) or dorsal–ventral oscillations (bottlenose dolphin) of the flippers or flukes, respectively, during swimming. This enabled the simultaneous determination of high-resolution HR and SF (Fig. 1a) throughout the dive (Fig. 1b).

Experimental protocol

The method for capturing, attaching animal-borne instruments, and recovering the instruments on free-ranging Weddell seals has been described (Davis et al. 2003). Briefly, Weddell seals were captured with a purse-string net on the sea ice along the southwestern shore of Ross Island and transported in a sled to the sea ice research camp. Newly captured seals were sedated with an intramuscular injection of ketamine hydrochloride (2 mg kg^{-1}, Fort Dodge Laboratories) and diazepam (0.1 mg kg^{-1}, Steris), weighed, and placed in a climate-controlled research hut located on sea ice that was 3 m thick and over water that was ca. 250 m deep. After cleaning the fur with acetone, a piece of neoprene rubber (0.5-cm thick, 16 cm × 6 cm) was glued to the fur along the dorsal midline behind the shoulders with neoprene rubber cement. The HR recorder was attached to the neoprene rubber with hose clamps. Two electrodes were glued to small patches of shaved skin in the lateral and ventral axillary area. Satellite transmitters (Wildlife Computers, Redmond, WA) and VHF radios (Advanced Telemetry Systems, Isanti, MN) were also glued to the fur to enable relocation when the seals hauled out on the ice. The satellite transmitters provided the seal's location to within a one-half km radius, and VHF radio transmitters enabled us to determine the seal's exact location using a receiver and directional antenna. Seals were allowed 12–18 h to recover from sedation before release through a nearby, man-made hole (1.3 m diameter) in the sea ice. The field camp was located <3 km from Ross Island, and the seals departed the artificial hole within an hour after release. They ranged throughout eastern McMurdo Sound until recaptured on the sea ice 1–2 weeks later. A vinyl bag with breathing holes was placed over their head to calm them while the instruments were removed, a process that took about 20 min.

For the bottlenose dolphins, the HR recorder was attached to a custom-sized neoprene rubber vest that was

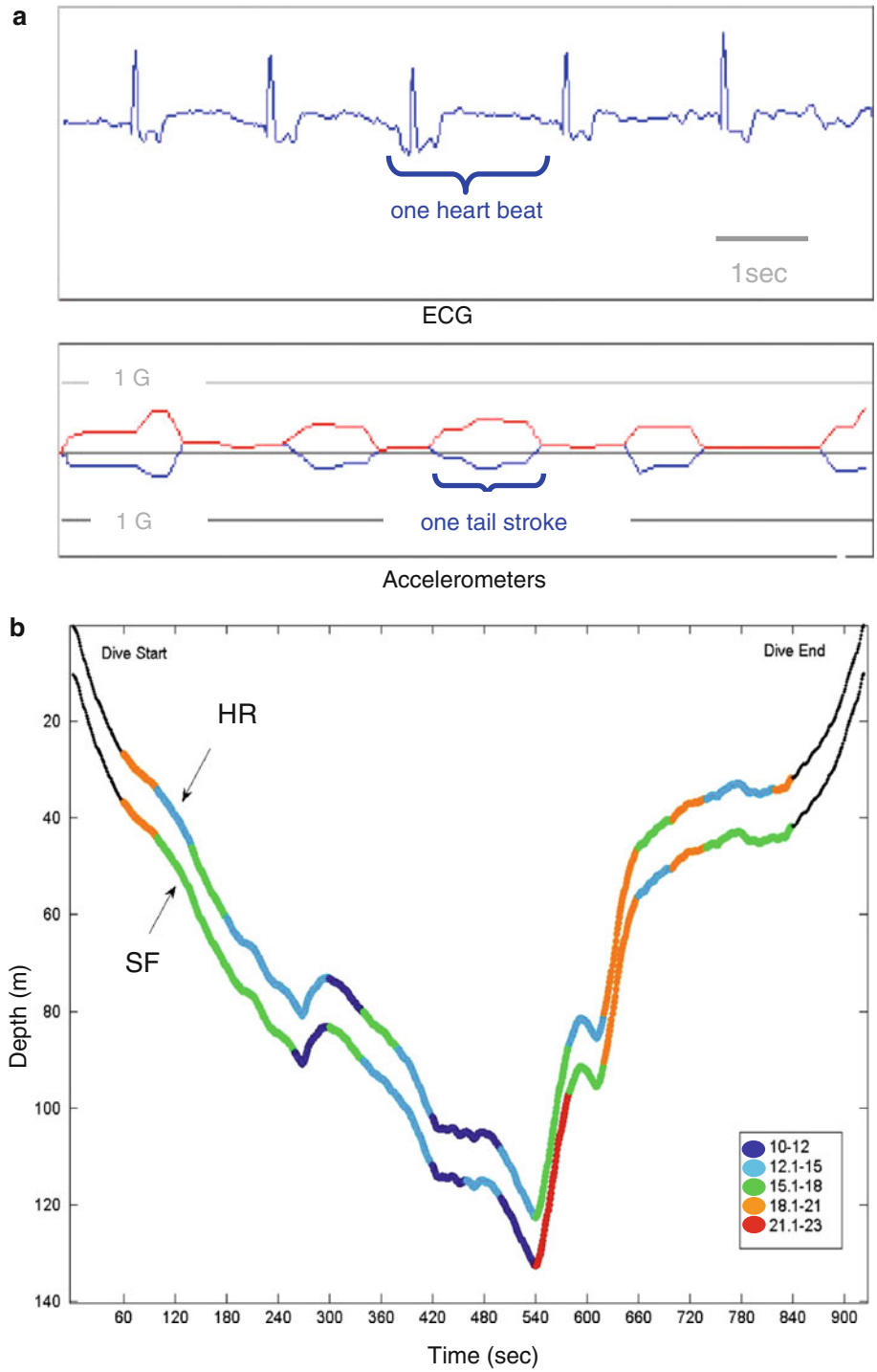

Fig. 1 The correlation between heart rate and stroke frequency in marine mammals. **a** Representative, high-resolution electrocardiogram (ECG) showing the waveform and individual heart beats and the simultaneous two-axis accelerometer traces of individual tail strokes for a dolphin. **b** The correspondence between flipper stroke frequency (SF, min^{-1}) and heart rate (HR, min^{-1}) is demonstrated by superimposing both parameters on the time–depth profile for a single dive by a Weddell seal. Data above a depth of 30 m during initial descent or final ascent were excluded from the analysis to avoid surface effects (e.g., ventilatory tachycardia between dives and release of the dive response just prior to surfacing). *Colors* correspond to specific ranges for each variable

fitted around the chest of the animal, and the electrodes were attached to the skin with suction cups. Resting measurements were made while the animals stationed in front of a window at a depth of 10 m. Exercise tests consisted of horizontal, steady-state swimming though a series of submerged hoops positioned on the bottom of the seawater pool.

Data analysis

Software from UFI enabled the ECG, two- or three-axes of acceleration and pressure (depth) to be displayed simultaneously at different levels of temporal resolution (Fig. 1a). The number of heart beats (R–R intervals) and corresponding flipper or fluke strokes were manually counted

(to avoid potential artifacts from muscle noise) at 40-s intervals for Weddell seals and 5-s intervals for dolphins. The relationship between SF and HR was determined by Type 2 linear regression for all data in which SF was greater than zero, and the root mean square difference (RMSD) was calculated as a measure of vertical variance. Type 2 linear regression is used when there is potential error in both the x and y variables. In practice, it provides a more accurate slope and is calculated as the slope of the conventional Type 1 linear regression divided by the square root of R^2. To compare our results for Weddell seals with those from of our previous model (Davis and Kanatous 1999), we estimated diving metabolism at different rates of stroking using the method of Williams et al. (2004) for the cost per stroke and an assumed resting metabolic rate of 2 ml O_2 min^{-1} kg^{-1} (Davis and Kanatous 1999). Lastly, we calculated muscle metabolic rate (as a multiple of the resting level) at different rates of stroking as the product of the cost per stroke and stroke rate divided by the muscle mass and the oxygen consumption of resting muscle.

Results

Simultaneous monitoring of HR and SF demonstrated that diving bradycardia was modulated by exercise in both Weddell seals and bottlenose dolphins (Fig. 1). The free-ranging Weddell seals made dives ($N = 13$ representing ca. 131 min of submerged recording) with an average duration of 10.52 ± 4.71 min s.d. and to a mean maximum depth of 156 ± 60 m s.d. HR and SF varied concurrently during the different phases of the dive (Fig. 1b). Thus, the seal actively stroked during initial descent to a depth of 50 m, and both SF and HR averaged 18.1–21 min^{-1}. However, data above a depth of 30 m during initial descent or final ascent (about 5 % of the total HR and SF data) were excluded from the analysis to avoid surface effects (i.e., ventilatory tachycardia between dives and release of the dive response just prior to surfacing). Below 50 m, the seals began to use a stroke-and-glide mode of locomotion with both SF and HR decreasing in parallel. When the seal began to ascend, SF and HR increased immediately and in parallel depending on the ascent angle. The seals also exhibited periods at depth where they stopped swimming and drifted in the water column, thereby enabling us to measure resting HR while submerged. Based on our previous research, all of the dives were within the seal's aerobic dive limit and appeared to be exploratory (Davis et al. 2003). Average submerged resting HR was 11 ± 4.5 min^{-1} s.d., and it increased linearly with SF (HR = 1.48SF − 8.87) to a maximum of 38 min^{-1} with a coefficient of determination (R^2) of 0.53 and an RMSD of 5.1 min^{-1} [($N = 127$); Fig. 2a].

For the dolphins, there was nearly a direct correlation between each R–R interval of the ECG trace and an individual stroke during steady-state submerged swimming (Fig. 1a). Submersion time ranged from 3 to 4 min for both resting and swimming tests ($N = 21$ dives comprised of 11 swimming trials and 10 submerged resting trials). The average submerged resting HR was 31 ± 5.9 min^{-1} s.d., and it increased linearly with SF (HR = 0.99SF + 2.46) to a maximum of 72 min^{-1} with a coefficient of determination (R^2) of 0.72 and an RMSD of 5.7 min^{-1} [($N = 88$); Fig. 2b].

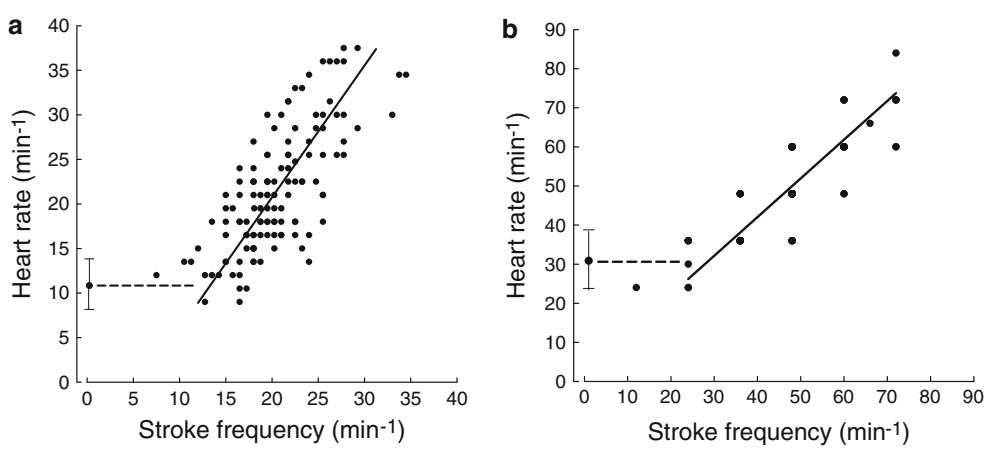

Fig. 2 Heart rates for adult Weddell seals (**a**) and bottlenose dolphins (**b**) as a function of stroke frequency. The heart rates while resting submerged (i.e., 0 stroke frequency) are shown as the average ± s.d. and the *dashed line*. Heart rate increased linearly with stroke frequency for both species, although the range of values differed (regression statistics provided in text). Note the difference in the scales for both plots, with slower swimming seals (**a**) showing lower stroke frequencies and submerged heart rates than the faster swimming dolphins (**b**). Each *point* denotes the average heart rate over 40- and 5-s intervals for seals ($N = 127$) and dolphins ($N = 88$), respectively

Discussion

Although our earlier model underestimated the increase in HR with physical exertion in Weddell seals (Fig. 3), the results from the present study are consistent with model predictions and form the bases for three important conclusions: (1) a dive response is needed to efficiently use blood and muscle oxygen stores to maximize aerobic dive duration; (2) the intensity of the dive response varies inversely with the level of muscle metabolism; (3) there is a minimum HR and concurrent convective oxygen transport needed to maintain aerobic metabolism in organs and tissues other than muscle in diving mammals.

The first conclusion results from the reliance on two types of molecules to transport and store oxygen in the vertebrate body that have very different oxygen affinities. Hemoglobin has a moderate binding affinity for oxygen (an oxygen partial pressure at 50 % saturation or P_{50} of about 27 mm Hg [3.59 kPa]), while myoglobin has a very high binding affinity for oxygen (P_{50} of 3 mm Hg [0.40 kPa]). Unlike terrestrial animals, many aquatically adapted vertebrates rely on a high (up to 10× greater) concentration of muscle oxy-myoglobin to store from one-third to one-half of the total oxygen used during a dive (Kooyman et al. 1999). For myoglobin-bound oxygen to become available for aerobic metabolism, the intracellular partial pressure of oxygen in the muscle must be <10 mm Hg (1.3 kPa); in other words, active muscles must become very hypoxic (but not anaerobic). We know from studies of Weddell seals making voluntary dives that desaturation of myoglobin begins as soon as the seal submerges (Guyton et al. 1995). At the beginning of a dive when the partial pressure of oxygen in arterial blood is around 100 mm Hg (13.33 kPa), the most effective way to make the muscle hypoxic is by ischemia. This is where the dive response plays a major role. Peripheral vasoconstriction reduces convective oxygen transport to the muscle resulting in tissue hypoxia. The partial pressure of oxygen in the muscle becomes low enough for oxygen to dissociate from myoglobin and diffuse into mitochondria. As the dive progresses, hypoxic hypoxia (i.e., a reduction in arterial oxygen partial pressure) also contributes to the level of muscle hypoxia and myoglobin desaturation (Davis and Kanatous 1999).

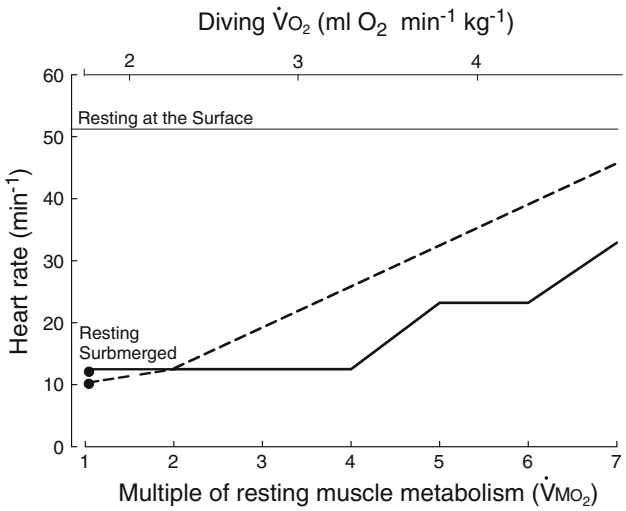

Fig. 3 Heart rate for adult Weddell seals as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) predicted from the model (*solid line*) (Davis and Kanatous 1999) and based on measurements for free-diving seals (*dashed line*) from this study. The heart rate while resting at the surface is from Zapol et al. (1979). Estimated diving $\dot{V}_{O_2}$ is from (Davis and Kanatous 1999)

The explanation for the second conclusion results from the balance between convective oxygen transport and muscle oxygen consumption. To maximize the use of available body oxygen stores and the aerobic dive duration for a broad range of muscle metabolic rates, the full use of the oxygen available in the blood (arterial oxygen saturation not less than ca. 38 %; Qvist et al. 1986; Davis and Kanatous 1999) and total muscle oxygen stores must be completed simultaneously. According to our model, this can be achieved by adjusting cardiac output, most of which results from changes in HR (Ponganis et al. 1990), according to the level of muscular exertion. Thus, as muscle oxygen consumption rises, a concomitant increase in cardiac output and muscle perfusion (i.e., the dive response is less pronounced) will augment muscle oxy-myoglobin (Fig. 4). Based on our model for an exercising Weddell seal, the blood provides about 50 % of the muscle's oxygen consumption over a *broad range* of exercise levels with the remainder coming from endogenous oxy-myoglobin (Davis and Kanatous 1999). As a result, the aerobic dive limit decreases as muscular exertion increases.

Although we did not measure muscle blood flow, previous studies have demonstrated sustained muscle perfusion during aerobic, free-ranging dives in seals. For example, Ponganis et al. (1993) reported that muscle temperature in Weddell seals during dives was constant or decreased, which suggested that the working muscles were not isolated from cooler blood in the circulation. Similarly, Guyton et al. (1995) measured lower than expected muscle deoxygenation rates in free-diving Weddell seals, which suggested some level of perfusion. The relationship between heart rate and blood flow was also reported for trained harbor seals that were habituated to forced submersion (Jobsis et al. 2001). The trained seals showed significantly greater HR (2×) and muscle blood flow (3×) compared to naïve, untrained seals in that study. The increased HR and muscle blood flow during submersion in trained seals was also associated with a reduced rate of muscle deoxygenation and an increased rate of venous

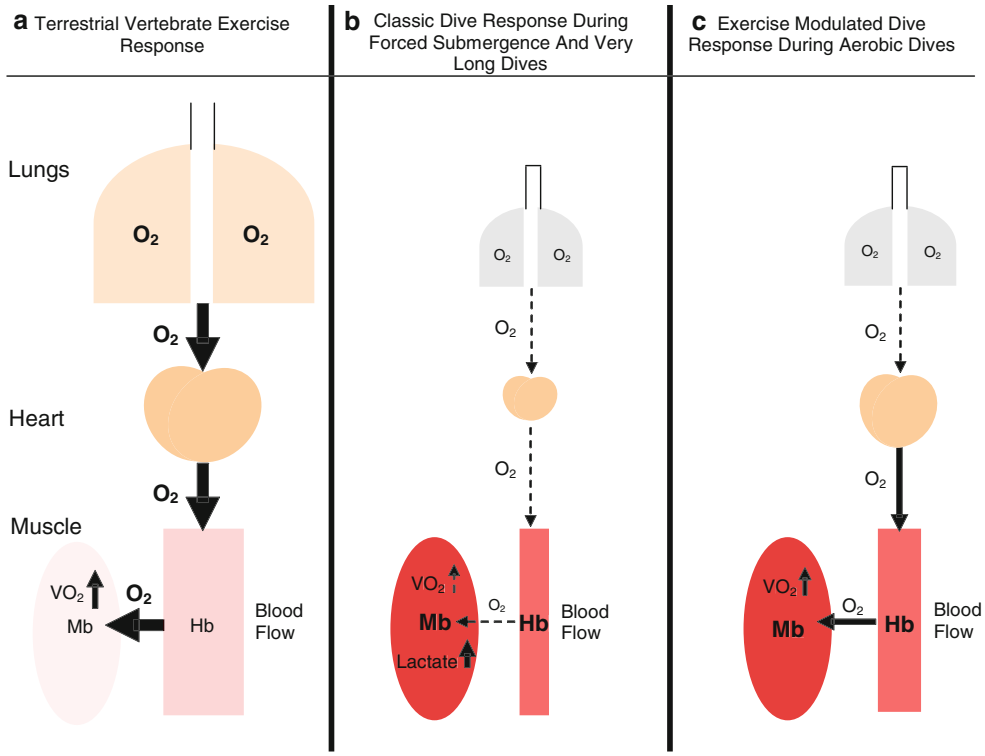

Fig. 4 A comparison of pulmonary and cardiovascular changes and their influence on convective oxygen transport to muscle during exercise in terrestrial mammals (**a**) and in marine mammals during forced submergence (**b**) or aerobic, voluntary dives (**c**). The *symbol*, *font size*, and *arrow thickness* indicate relative magnitude of the variables. The large lungs in **a** indicate hyperventilation and enhanced convective oxygen transport, while ventilation ceases and there is also lung compression at depth with little oxygenation of the blood in **b** and **c**. The *arrow* from the lungs to the heart represents the pulmonary vein. The *size of the heart symbol* indicates heart rate, with a tachycardia in **a**, a profound bradycardia in **b**, and a variable bradycardia depending on the level of physical exertion in **c**. The *size of the arrow from the heart symbol* indicates relative arterial blood flow, with peripheral vasodilation and increased flow in **a**, severe peripheral vasoconstriction and reduced flow in **b**, and variable vasoconstriction and blood flow depending on the level of physical exertion in **c**. The *color* of the blood vessels indicate relative hemoglobin (Hb) concentration and that of the muscle myoglobin (Mb) concentration, which are relatively low in **a** and high in **b** and **c**. Finally, the *arrows* in the muscle indicate the relative increase in oxygen consumption ($\dot{V}_{O_2}$) and lactate concentration

deoxygenation. Furthermore, the muscle blood flow was continuous and not intermittently pulsatile as we had hypothesized in our earlier model (Davis and Kanatous 1999). Although trained submersion differs from free diving by not mimicking the energy demands associated with swimming or the ability of the seal to determine submersion duration, it does demonstrate that muscle blood flow increases with HR during submersion. Except for the brain, where circulation is always maintained at resting levels, blood flow to the rest of the body changes proportionately with HR and cardiac output during a dive (Elsner et al. 1964; Blix et al. 1976; Jobsis et al. 2001). This is consistent with evidence that the sympathetic nervous system is activated en masse so as to produce a generally uniform peripheral vasoconstriction during forced submersions and trained dives (Stone et al. 1973; Zapol et al. 1979; Kjekshus et al. 1982; Elsner and Gooden 1983; Blix and Folkow 1983). The sites of vasoconstriction include arteries larger than small arterioles (Irving et al. 1942; Blix and Folkow 1983). This allows constriction of peripheral vasculature sites proximal to the competitive influence of tissue metabolites (i.e., autocoids) that are released locally in response to tissue ischemia and are known to have a vasodilator action on the arterioles (Scholander 1940; White et al. 1973; Grega and Adamski 1987). Hence, the regulation of muscle and organ blood flow in seals is largely independent of local tissue metabolite control at the capillary level and is controlled at a higher level in the vascular system. Since our results show that HR increases with flipper or fluke SF during voluntary, aerobic dives, available evidence indicates that muscle blood flow also increases proportionately with HR and muscular exertion.

Finally, our third conclusion is based on the convective oxygen requirements of other tissues and organs to maintain aerobic metabolism. Our model predicted that HR in Weddell seals would not decrease below 19 % of the predive, resting level (i.e., 10 min^{-1}) to maintain aerobic metabolism in tissues and organs other than the muscles.

This is nearly identical to the measured minimum HR of the seals in the present study (Fig. 2a). At this level of bradycardia, the theoretical extraction coefficient of oxygen from the arterial blood (arterial–venous O_2 difference/arterial O_2 content) is 60 % for the splanchnic organs and kidneys, 50 % for the heart, and <30 % for the brain, indicating sufficient convective oxygen delivery to support aerobic metabolism (Davis and Kanatous 1999). This is critical for the maintenance of physiological homeostasis in Weddell seals and other long-duration diving marine mammals that may spend over 90 % of their time at sea submerged (Butler and Jones 1997; Kooyman et al. 1980; Fedak 1986; Le Boeuf et al. 1989; Hindell et al. 1992) and is consistent with normal renal and hepatic function during such dives (Davis et al. 1983).

The need to match an increase in muscle oxygen consumption with an increase in convective oxygen transport explains why long-duration divers such as the Weddell seal, which maintain a low level of metabolism by using cost-efficient modes of locomotion (Williams et al. 2004), have a more pronounced dive response than short-duration divers such as the bottlenose dolphin, which have a higher range of stroke frequencies and swim speeds as well as a lower muscle myoglobin concentration (Noren et al. 2001) (Fig. 2). Despite differences in the level of bradycardia, the dolphins in this study also showed an increase in HR with increased stroking while submerged. Because the evolutionary origins of these species differ, our data suggest that the underlying cardiovascular adjustments to maximize aerobic dive duration at different levels of swimming exertion are a response to similar evolutionary pressures to prevent asphyxia while supporting exercise. In fact, semi-aquatic divers such as the tufted duck (*Aythya fuligula*), which are very energetic, exhibit a bias toward an exercise response that results in an increase in HR and perfusion of the active leg muscles above resting levels during the majority of relatively short duration, voluntary dives (Bevan and Butler 1992). These examples show the influence that muscular exertion has on the optimal level of bradycardia during voluntary dives.

In diving mammals and birds, the concentration of myoglobin in the muscles represents another important adaptation for exercising underwater. Only those species with a comparatively large muscle oxygen store will benefit from a dive response. We speculate that the evolution of high myoglobin concentrations in the muscles of diving mammals and birds as a means of increasing oxygen stores and extending aerobic dive duration was only possible because the dive response already existed to protect the animals against asphyxia.

In summary, our results provide an explanation that is consistent with our previous physiological model for long-duration divers (Davis and Kanatous 1999) and the results of other studies examining the ability of diving mammals and birds to support aerobic muscle metabolism while swimming submerged (Butler 1988; Butler and Jones 1997; Kooyman et al. 1980; Guyton et al. 1995; Bevan and Butler 1992). During completely aerobic, voluntary dives, the cardiovascular adjustments that maximize dive duration combine elements of both a dive response and an exercise response (Fig. 4c), with one or the other predominating depending on the level of physical exertion.

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