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CONVECTIVE OXYGEN TRANSPORT AND TISSUE OXYGEN CONSUMPTION IN WEDDELL SEALS DURING AEROBIC DIVES

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Summary

Unlike their terrestrial counterparts, marine mammals stop breathing and reduce their convective oxygen transport while performing activities (e.g. foraging, courtship, aggressive interactions, predator avoidance and migration) that require sustained power output during submergence. Since most voluntary dives are believed to remain aerobic, the goal of this study was to examine the potential importance of the dive response in optimizing the use of blood and muscle oxygen stores during dives involving different levels of muscular exertion. To accomplish this, we designed a numerical model based on Fick's principle that integrated cardiac output ($\dot{V}$b), regional blood flow, convective oxygen transport ($\dot{Q}_{O_2}$), muscle oxymyoglobin desaturation and regional rates of oxygen consumption ($\dot{V}_{O_2}$). The model quantified how the optimal matching or mismatching of $\dot{Q}_{O_2}$ to $\dot{V}_{O_2}$ affected the aerobic dive limit (ADL). We chose an adult Weddell seal *Leptonycotes weddellii* on which to base our model because of available data on the diving physiology and metabolism of this species. The results show that the use of blood and muscle oxygen stores must be completed at the same time to maximize the ADL for each level of $\dot{V}_{O_2}$. This is achieved by adjusting $\dot{V}$b (range 19–94 % of resting levels) and muscle $\dot{Q}_{O_2}$ according to the rate of muscle oxygen consumption ($\dot{V}_{MO_2}$). At higher values of $\dot{V}_{MO_2}$, $\dot{V}$b and muscle perfusion must increase to maintain an appropriate $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio so that available blood and muscle oxygen stores are depleted at the same time. Although the dive response does not sequester blood oxygen exclusively for brain and heart metabolism during aerobic dives, as it does during forced submersion, a reduction in $\dot{V}$b and muscle perfusion below resting levels is necessary to maximize the ADL over the range of diving $\dot{V}_{O_2}$ (approximately 2–9 ml O_2 min^{-1} kg^{-1}). Despite the reduction in $\dot{V}$b, convective oxygen transport is adequate to maintain aerobic metabolism and normal function in the splanchnic organs, kidneys and other peripheral tissues. As a result, physiological homeostasis is maintained throughout the dive. The model shows that the cardiovascular adjustments known as the dive response enable the diving seal to balance the conflicting metabolic demands of (1) optimizing the distribution and use of blood and muscle oxygen stores to maximize the ADL over the normal range of diving $\dot{V}_{O_2}$ and (2) ensuring that active muscle receives adequate oxygen as $\dot{V}_{MO_2}$ increases.

Key words: Weddell seal, marine mammal, *Leptonycotes weddellii*, dive response, aerobic dive limit, convective oxygen transport.

Introduction

When marine mammals submerge, they exhibit a dive response that involves apnea, bradycardia and peripheral vasoconstriction (for a review, see Butler and Jones, 1997). Apnea interrupts respiratory gas exchange and forces the diving animal to use lung, blood and tissue oxygen stores to sustain aerobic metabolism or to switch to anaerobic energy production. The bradycardia decreases cardiac output and causes reflex peripheral vasoconstriction that maintains normal arterial blood pressure (Irving et al., 1942; Scholander et al., 1942; Elsner and Scholander, 1965; Elsner et al., 1966; Murdaugh et al., 1966). During forced submersion and very long voluntary dives, the bradycardia is profound (i.e. less than 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 in order 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). Peripheral organs and tissues receiving reduced blood flow become anaerobic and may lower their metabolic rate (Valtin, 1973; Nelson et al., 1988; Adams and Cain, 1983) or, in the case of skeletal muscle, continue to support aerobic metabolism with endogenous oxygen stores in the form of oxymyoglobin. When these oxygen stores are depleted, peak force development in skeletal muscle decreases (Hogan et al., 1994, 1998) and ATP

production switches to anaerobic glycolysis, resulting in the rapid formation of lactic acid (Hochachka et al., 1977). The profound dive response that occurs during forced submergence and long voluntary dives is designed to maximize breath-hold duration to prevent asphyxia (Kooyman, 1975). However, normal organ function and physiological homeostasis are disrupted (Murdaugh et al., 1966; Kooyman et al., 1980). The circulatory and metabolic adjustments that occur during these dives are stressful, and only severe hemorrhagic shock ranks as a comparable threat to homeostasis (Rowell, 1986; Grega and Adamski, 1987; Soini et al., 1992).

During most voluntary dives, the dive response of marine mammals is less profound than during forced submersion (Kooyman and Campbell, 1972; Stone et al., 1973; Ridgway and Howard, 1979; Pasche and Krog, 1980; Hill et al., 1987; Butler and Jones, 1997), and energy metabolism is believed to remain aerobic (Kooyman et al., 1980, 1983; Qvist et al., 1986; Kooyman, 1989). These dives are defined as being within an animal's aerobic dive limit (ADL), which is the longest dive that a marine mammal can make while relying principally on oxygen stored in the lungs, blood and muscles to maintain aerobic metabolism. Historically, the ADL has been determined by measuring the maximum voluntary dive duration occurring without a post-dive increase in blood lactate concentration (Kooyman et al., 1980, 1983; Ponganis et al., 1997a) or estimated by dividing total body oxygen stores by the rate of whole-body oxygen consumption (Ponganis et al., 1993a). Although the relationship between convective oxygen transport and tissue oxygen consumption during aerobic dives is poorly understood because of the difficulty of making *in vivo* measurements, most organs and tissues receive sufficient oxygen from the circulation or endogenous oxymyoglobin to maintain aerobic energy production (Kooyman, 1989; Guyton et al., 1995). Measurements of blood chemistry, hepatic perfusion, renal glomerular filtration rate (GFR), muscle nitrogen tension, muscle temperature and oxymyoglobin desaturation in skeletal muscle indicate that some degree of peripheral blood flow is maintained (Ridgway and Howard, 1979; Davis et al., 1983; Castellini et al., 1988; Kooyman, 1989; Ponganis et al., 1993b; Guyton et al., 1995). Organs such as the liver, kidneys and gastrointestinal tract continue to function (Davis et al., 1983), and physiological homeostasis is maintained during the dive (Kooyman et al., 1980, 1983; Castellini et al., 1988). An additional advantage of making short aerobic dives rather than long dives that rely heavily on anaerobic energy production is the shorter recovery time at the surface. The time necessary to metabolize high concentrations of lactic acid and to restore normal blood and tissue pH can be several hours after dives that exceed the ADL (Kooyman et al., 1980). In contrast, only blood and tissue oxygen stores need to be replenished after an aerobic dive, a process that can be accomplished in several minutes. Hence, an animal can spend more time submerged (e.g. foraging) by making many short, aerobic dives rather than a single long dive that produces high concentrations of lactic acid.

If blood oxygen is not sequestered for brain and heart metabolism during aerobic dives as it is during forced submersion and long voluntary dives, then what is the purpose of the dive response during aerobic dives? Why reduce tissue perfusion at all, especially during vigorous swimming when muscle metabolic rate is elevated? The normal mammalian response to exercise is to increase cardiac output and muscle perfusion, not to reduce it (Rowell, 1986; Wagner, 1991). Does the dive response create a conflict between convective oxygen transport and aerobic tissue metabolism, especially in active skeletal muscles? The goal of the present study was to examine the potential importance of the dive response in optimizing the use of blood and muscle oxygen stores during aerobic dives that involve different levels of muscular exertion. To accomplish this, we designed a numerical model that integrated cardiac output, regional blood flow, convective oxygen transport, muscle oxymyoglobin desaturation and regional metabolic rate. The model quantified how the optimal matching or mismatching of convective oxygen transport to tissue oxygen consumption would affect the ADL. We chose an adult Weddell seal (*Leptonycotes weddellii*) on which to base our model because of available data on the diving physiology and metabolism of this species. However, the model can be used with other marine mammals by substituting the appropriate physiological and metabolic variables. Given the difficulty of measuring the contributions of blood and muscle oxygen stores to whole-body oxygen consumption rates at different levels of tissue perfusion and muscular exertion *in vivo*, this model is a logical approach to understanding the physiological basis for the ADL. In addition, it provides a useful conceptual framework for developing testable hypotheses for future research and more sophisticated models.

Materials and methods

Theoretical basis for the model: Fick's principle

A numerical integration technique was used to model the relationship between regional convective oxygen transport ($\dot{Q}_{O_2}$) and the rate of oxygen consumption ($\dot{V}_{O_2}$) in a hypothetical Weddell seal during aerobic dives. The numerical process iteratively determined arterial blood oxygen concentration (C_{aO_2}) and venous blood oxygen concentration C_{vO_2} for various tissues and organs based on equation 1, which describes Fick's principle, and the circulatory diagram shown in Fig. 1:

$$\dot{V}_{O_2} = \dot{Q}(C_{aO_2} - C_{vO_2}), \quad (1)$$

where $\dot{Q}$ is blood flow rate. The six largest regional circulations (i.e. cerebral, coronary, skeletal muscle, splanchnic, renal and cutaneous) were incorporated in the model, although the last three were modeled as a single unit, which included all other organs and tissues (e.g. bone and fat) not covered by the cerebral, coronary and skeletal muscle circulations. The average temporal resolution (i.e. the period between consecutive computations) was 0.25 s.

This model considers only dives that are within the seal's ADL (Kooyman et al., 1980; Ponganis et al., 1993a). The rate

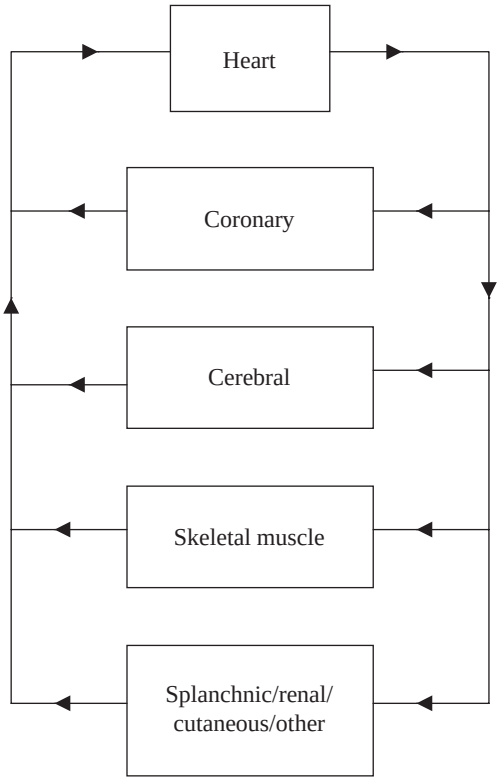

Fig. 1. Simplified circulatory system used in the model. The cardiovascular system was divided into four regional circulations: coronary, cerebral, skeletal muscle and a combined category that included the splanchnic, renal, cutaneous and other circulatory beds.

of oxygen consumption in the tissues is maintained until convective oxygen delivery falls below a critical level and endogenous oxygen stores (skeletal muscle only) are depleted as a result of a combination of ischemia and hypoxic hypoxia. When an organ or tissue no longer has sufficient oxygen to support aerobic metabolism, then the ADL has been reached and the model terminates the dive. We did not consider the use of muscle creatine phosphate in delaying the onset of anaerobiosis and extending the ADL once oxymyoglobin stores in skeletal muscle had been depleted (Butler and Jones, 1997), although this variable could be added to future models.

Assumptions and equations

Organ and tissue masses were based on published values for a 450 kg adult Weddell seal *Leptonycotes weddellii* (Table 1) (Zapol et al., 1979; Fujise et al., 1985). The resting $\dot{V}_{O_2}$ for Weddell seal organs and tissues were estimated from the metabolic-mass-adjusted $\dot{V}_{O_2}$ for the equivalent organs of a human or rat (Field et al., 1939; Kety, 1957; Diem and Lentner, 1970) (Table 1). The basal, whole-body $\dot{V}_{O_2}$ (897 ml O_2 min^{-1} or 2.0 ml O_2 min^{-1} kg^{-1}) was calculated by summing individual organ and tissue metabolic rates. The calculated basal metabolic rate was similar to the minimum metabolic rate measured for adult Weddell seals during sleep (Castellini et al., 1992; Ponganis et al., 1993a) and lay between the allometric prediction (990 ml O_2 min^{-1} or 2.2 ml O_2 min^{-1} kg^{-1}) for terrestrial mammals generally (Kleiber, 1975) and for fissiped carnivores (715 ml O_2 min^{-1} or 1.6 ml O_2 min^{-1} kg^{-1}) specifically (McNab, 1989). This is in agreement with Lavigne et al. (1986) (see also Gallivan and Ronald, 1979; Fedak, 1986), who showed that the basal metabolic rates of seals are indistinguishable from those predicted for other mammals.

Resting heart rate (f_H) (51.5 beats min^{-1}), cardiac output ($\dot{V}$b) (42.72 l min^{-1}) and stroke volume (V_s) (0.83 l) were based on measured values for Weddell seals (Table 2) (Zapol et al., 1979). Resting f_H was nearly identical to the allometric prediction (52.3 beats min^{-1}) for a 450 kg mammal, but the predicted $\dot{V}$b (26.4 l min^{-1}) and V_s (0.51 l) were 38 % less than the measured values used in this model (Calder, 1984). Comparable allometric differences in $\dot{V}$b and V_s have been observed for harbor seals (*Phoca vitulina*) (Ponganis et al., 1990).

During a simulated dive, the reduction in $\dot{V}$b was varied from 19 to 94 % of resting levels (Table 2). For brevity, we hereafter refer to these percentages of resting, predive $\dot{V}$b as

Table 1. *Mass and estimated resting $\dot{V}_{O_2}$ for the organs and tissues of a 450 kg adult Weddell seal*

Organ or tissue	Mass (kg)	$\dot{V}_{O_2}$ (ml O_2 min^{-1} kg^{-1})	$\dot{V}_{O_2}$ (ml O_2 min^{-1})
Brain	0.6	22.1[1]	13.3
Heart	1.9	59.2[1]	112.5
Muscle	157.5	1.4[1]	216.0
Splanchnic			
Liver	8.8	27.7[1]	243.8
Alimentary canal	8.1	10.1[1]	81.8
Kidneys	1.6	38.4[1]	61.4
Spleen	5.9	4.7[2]	27.7
Lungs	5.1	4.5[2]	23.0
Testis	0.1	2.4[2]	0.2
Bone	14.4	0.4[2]	5.8
Skin	25.7	1.6[2]	41.1
Adipose tissue	122.0	0.5[2]	66.8
Other organs (e.g. tongue, bladder)	2.3	1.4[3]	3.2
Blood	96.0		
Total	450.0	2.0	896.6

Values are from Zapol et al. (1979) and Fujise et al. (1985).
$\dot{V}_{O_2}$, rate of oxygen consumption.
[1]Estimated for the organs of a 450 kg Weddell seal on the basis of the metabolic-mass-adjusted $\dot{V}_{O_2}$ for the equivalent organs of a 70 kg human (Kety, 1957; Diem and Lentner, 1970) using the equation:

$$\dot{V}_{O_2\text{seal organ}} = \dot{V}_{O_2\text{human organ}}(450^{-0.25}/70^{-0.25}) .$$

[2]Estimated for the organs of a 450 kg Weddell seal on the basis of the metabolic-mass-adjusted $\dot{V}_{O_2}$ for the equivalent organs of a 0.15 kg rat (Field et al., 1939):

$$\dot{V}_{O_2\text{seal organ}} = \dot{V}_{O_2\text{ rat organ}}(450^{-0.25}/0.15^{-0.25}) .$$

[3]Estimated for a 450 kg Weddell seal assuming a metabolic rate equivalent to the metabolic-mass-adjusted $\dot{V}_{O_2}$ for skeletal muscle of a 70 kg human (Diem and Lentner, 1970).

Table 2. *Circulatory variables used in the model for different levels of cardiac output during a dive response*

		Cardiac output (% resting level)				
Variable	Resting	94	75	56	37	19
f_H (beats min^{-1})	51.5	48.8	41.6	32.9	23.2	12.5
V_S (l)	0.83	0.82	0.77	0.73	0.69	0.64
$\dot{V}b$ (l min^{-1})	42.72	40.00	32.00	24.00	16.00	8.00
$\dot{Q}_B$ (l min^{-1})	0.36	0.36	0.36	0.36	0.36	0.36
$\dot{Q}_H$ (l min^{-1})	1.84	1.72	1.38	1.03	0.69	0.35
$\dot{Q}_M$ (l min^{-1})	7.90	7.40	5.92	4.44	2.96	1.48
$\dot{Q}_{SRC}$ (l min^{-1})	32.63[1]	30.62	24.42	18.23	12.03	5.84

Resting values are based on data for a 450 kg Weddell seal from Zapol et al. (1979).

[1]Individual organ and tissue blood flow: splanchnic organs, 14.53 l min^{-1}; kidneys, 12.39 l min^{-1}; skin, 2.14 l min^{-1}; fat, 1.01 l min^{-1}; bone, 2.56 l min^{-1}.

f_H, heart rate; V_S, stroke volume; $\dot{V}b$, cardiac output; $\dot{Q}_B$, $\dot{Q}_H$, $\dot{Q}_M$, $\dot{Q}_{SRC}$, blood flow in the brain, heart, skeletal muscle and splanchnic, renal cutaneous and other peripheral tissues.

percent $\dot{V}b$ (e.g. 19 % $\dot{V}b$). Most of the reduction in $\dot{V}b$ was the result of a decrease in f_H. However, on the basis of studies of seals during forced submergence and diving voluntarily (Sinnet et al., 1978; Zapol et al., 1979; Kjekshus et al., 1982; Blix and Folkow, 1983; Ponganis et al., 1990), V_S was also reduced as f_H declined. The maximum reduction in V_S in the model was 25 % of the resting value and was proportional to the reduction in f_H. The reduction in cardiac output (i.e. the severity of the dive response) was immediate and remained constant throughout a dive. An 'anticipatory' increase in $\dot{V}b$ towards the end of a dive was not included in the model.

Except for the brain, where circulation was always maintained at resting levels, we assumed that blood flow to the rest of the body decreased proportionately with $\dot{V}b$ during a dive (Elsner et al., 1964; Blix et al., 1976). This assumption 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 (White et al., 1973; Rowell, 1986; Grega and Adamski, 1987).

Body oxygen stores were confined to the blood and skeletal muscle in this model, since no oxygen storage capability exists in the splanchnic organs (Dodd et al., 1987) and the heart represents less than 2 % of the total muscle mass. We assumed that lung oxygen was not available during a dive because of the complete functional pulmonary shunt that occurs in Weddell seals at pressures greater than 3–5 atmospheres (2280–3800 mmHg; 1 mmHg=0.133 kPa; approximately 30–50 m deep) (Falke et al., 1985; Reed et al., 1994). The increased ambient pressure during a dive causes the respiratory bronchioles and alveoli to collapse, forcing air into the bronchi and preventing gas exchange between the lungs and blood. Since adult Weddell seals spend much of their time at depths greater than 50 m (Kooyman et al., 1971; Castellini et al., 1992), this assumption is generally valid, especially for foraging dives. Although the functional pulmonary shunt limits access to lung oxygen during a dive, it also prevents exposure of the blood and tissues to high partial pressures of nitrogen that cause decompression sickness. Even if lung oxygen were available during a dive, it represents only 5 % of the total body oxygen store in Weddell seals (Kooyman and Ponganis, 1998).

To calculate total oxygen stores in the blood, we assumed that the blood volume for a 450 kg Weddell seal was 96 l (Ponganis et al., 1993a) and that 33 % of this volume was arterial blood and 67 % was venous blood (i.e. venules, small and large veins, hepatic sinus and spleen) (Rowell, 1986; Hurford et al., 1996). The blood hemoglobin (Hb) concentration (assuming complete splenic contraction) was 260 g l^{-1}, and the oxygen-binding capacity of Hb was 1.34 ml O_2 g^{-1} Hb (Kooyman et al., 1980; Qvist et al., 1986; Ponganis et al., 1993a). This gave a capacitance coefficient of oxygen in blood (β_{BO_2}) of 348 ml O_2 l^{-1} (260 g Hb l^{-1} blood × 1.34 ml O_2 g^{-1} Hb). At the beginning of a dive, we assumed that the arterial blood was 100 % saturated with oxygen as a result of predive hyperventilation (Kooyman et al., 1980; Qvist et al., 1986; Ponganis et al., 1993a). Mixed venous blood was calculated from equation 2 to be 86 % saturated at the beginning of a dive assuming an oxygen content that was 5 % by volume less (Ponganis et al., 1993a) than an initial Ca_{O_2} of 348 ml O_2 l^{-1} blood.

$$S\bar{v}_{O_2} = [(348 - 50)/348] \times 100 = 86\,\%, \qquad (2)$$

where $S\bar{v}_{O_2}$ is the oxygen saturation of mixed venous blood. We assumed that 35 % of the seal's body mass was skeletal muscle with a myoglobin concentration of 54 g kg^{-1} muscle (Ponganis et al., 1993a), an oxygen-binding capacity of 1.34 ml O_2 g^{-1} myoglobin and complete saturation at the beginning of a dive (Gayeski et al., 1987; Schenkman et al.,

1997). Arterial, venous and muscle oxygen stores were calculated as:

$$\text{Arterial blood oxygen (ml)} = 96 \times 0.33 \times 348 = 11\,025\,, \quad (3)$$

$$\text{Venous blood oxygen (ml)} = 96 \times 0.67 \times 348 \times 0.86 = 19\,250\,, \quad (4)$$

$$\text{Skeletal muscle oxygen (ml)} = 450 \times 0.35 \times 54 \times 1.34 = 11\,397\,. \quad (5)$$

A Weddell seal therefore has a total oxygen store of 41 672 ml O_2 (92.6 ml O_2 kg^{-1}): 27 % in the arterial blood, 46 % in the venous blood and 27 % in skeletal muscle. This is the maximum amount of oxygen that the seal can store in its blood and muscle. As we shall see below, not all of this is available during a dive.

As blood circulates through the four vascular beds (Fig. 1), the organs and tissues extract oxygen from the blood to meet their respective $\dot{V}_{O_2}$ requirements. Cv_{O_2} was calculated for each circulatory bed according to Fick's principle:

$$C_{BV_{O_2}} = Ca_{O_2} - (\dot{V}_{B_{O_2}}/\dot{Q}_B)\,, \quad (6)$$

$$C_{HV_{O_2}} = Ca_{O_2} - (\dot{V}_{H_{O_2}}/\dot{Q}_H)\,, \quad (7)$$

$$C_{MV_{O_2}} = Ca_{O_2} - (\dot{V}_{M_{O_2}}/\dot{Q}_M)\,, \quad (8)$$

$$C_{SRCV_{O_2}} = Ca_{O_2} - (\dot{V}_{SRC_{O_2}}/\dot{Q}_{SRC})\,, \quad (9)$$

where the letters B, H, M and SRC indicate brain, heart, skeletal muscle and splanchnic organs, kidneys, skin and other peripheral tissues, respectively, and $\dot{Q}$ is blood flow rate. However, the extraction coefficient of oxygen from the blood ($E_{B_{O_2}}$), where $E_{B_{O_2}}=(Ca_{O_2}-Cv_{O_2})/Ca_{O_2}$, could never exceed 0.8 (i.e. maximum $E_{B_{O_2}}$ at critical oxygen delivery) during a single pass of the blood through an organ or tissue. This is similar to the maximum $E_{B_{O_2}}$ measured in mammals during hypoxic hypoxia with and without ischemia (Lautt and Graham, 1977; Cain, 1978; Cole, 1983; King et al., 1987; Nelson et al., 1988; Samsel and Schumacker, 1994; Torrance and Wittnich, 1994; Noldge-Schomburg et al., 1996). The mixed venous blood oxygen concentration ($C\bar{v}_{O_2}$) was calculated for the four vascular beds as the difference between Ca_{O_2} and the total oxygen extracted per milliliter of blood:

$$C\bar{v}_{O_2} = Ca_{O_2} - [(\dot{V}_{B_{O_2}} + \dot{V}_{H_{O_2}} + \dot{V}_{M_{O_2}} + \dot{V}_{SRC_{O_2}})/(\dot{Q}_B + \dot{Q}_H + \dot{Q}_M + \dot{Q}_{SRC})]\,. \quad (10)$$

The arterial blood oxygen saturation (Sa_{O_2}) and venous blood oxygen saturation (Sv_{O_2}) were calculated for the blood of each vascular bed as the quotient of their respective oxygen concentrations (equations 6–10) and a $\beta_{B_{O_2}}$ of 348 ml O_2 l^{-1} blood. The arterial (Pa_{O_2}) and venous (Pv_{O_2}) blood oxygen partial pressures were calculated from their respective Sa_{O_2} and Sv_{O_2} using two polynomial equations fitted to the oxyhemoglobin dissociation curve (P_{50}=26.9 mmHg; 1 mmHg=0.133 kPa) for adult Weddell seals (Qvist et al., 1981). We assumed that the oxyhemoglobin dissociation curve did not change (i.e. that there was no right shift) during aerobic dives because blood pH remained at a normal value of 7.4 (Qvist et al., 1986).

Evidence obtained during the forced submergence of harbor seals and Weddell seals indicates that $\dot{Q}_B$ is generally maintained and $\dot{V}_{B_{O_2}}$ does not decline (Kerem and Elsner, 1973; Zapol et al., 1979; Blix and Folkow, 1983). In this model, we assumed that $\dot{Q}_B$ and $\dot{V}_{B_{O_2}}$ remained at resting levels during a dive and were independent of $\dot{V}$b (Tables 2, 3). We also assumed that the minimum Pa_{O_2} and mixed venous blood oxygen partial pressure ($P\bar{v}_{O_2}$) for normal cerebral metabolism

Table 3. *Rates of oxygen consumption of the four vascular beds at different levels of muscle metabolism during aerobic dives*

$\dot{V}_{M_{O_2}}$ (as a multiple of resting $\dot{V}_{M_{O_2}}$)	$\dot{V}$b (% resting $\dot{V}$b)	ADL (min)	$\dot{V}_{B_{O_2}}$ (ml O_2 min^{-1})	$\dot{V}_{H_{O_2}}$ (ml O_2 min^{-1})	$\dot{V}_{M_{O_2}}$ (ml O_2 min^{-1})	$\dot{V}_{SRC_{O_2}}$ (ml O_2 min^{-1})	Whole-body $\dot{V}_{O_2}$ (ml O_2 min^{-1})	Whole-body $\dot{V}_{O_2}$ (ml O_2 min^{-1} kg^{-1})
1	19	28.0	13.3 (1.7%)	21.2 (3%)	216 (27%)	555 (69%)	805	1.8
2	19	24.0	13.3 (1.3%)	21.2 (2%)	442 (43%)	555 (54%)	1032	2.3
3	19	24.0	13.3 (1.1%)	21.2 (2%)	658 (53%)	555 (44%)	1248	2.8
4	19	20.3	13.3 (0.9%)	21.2 (1%)	874 (60%)	555 (38%)	1464	3.3
5	37	18.0	13.3 (0.8%)	42.0 (2%)	1089 (64%)	555 (33%)	1700	3.8
6	37	15.8	13.3 (0.7%)	42.0 (2%)	1305 (68%)	555 (29%)	1916	4.3
7	56	13.3	13.3 (0.6%)	63.3 (3%)	1513 (71%)	555 (26%)	2144	4.8
8	56	12.7	13.3 (0.6%)	63.3 (3%)	1729 (73%)	555 (24%)	2360	5.2
9	75	12.0	13.3 (0.5%)	84.4 (3%)	1954 (75%)	555 (21%)	2607	5.8
10	75	10.8	13.3 (0.5%)	84.4 (3%)	2170 (77%)	555 (20%)	2823	6.3
11	94	9.6	13.3 (0.4%)	105.5 (3%)	2385 (78%)	555 (18%)	3058	6.8
12	94	9.2	13.3 (0.4%)	105.5 (3%)	2601 (79%)	555 (17%)	3274	7.3
13	94	8.2	13.3 (0.4%)	105.5 (3%)	2817 (81%)	555 (16%)	3490	7.8
14	94	7.4	13.3 (0.4%)	105.5 (3%)	3033 (82%)	555 (15%)	3706	8.2
15	94	6.6	13.3 (0.3%)	105.5 (3%)	3249 (83%)	555 (14%)	3922	8.7
16	94	6.0	13.3 (0.3%)	105.5 (3%)	3465 (84%)	555 (13%)	4138	9.2

Values are for the cardiac output that gave the maximum aerobic dive limit for each level of $\dot{V}_{M_{O_2}}$.

Values in parentheses are the percentages of whole-body $\dot{V}_{O_2}$.

ADL, aerobic dive limit; $\dot{V}$b, cardiac output; $\dot{V}_{B_{O_2}}$, $\dot{V}_{H_{O_2}}$, $\dot{V}_{M_{O_2}}$ and $\dot{V}_{SRC_{O_2}}$, rates of oxygen uptake of the brain, heart, skeletal muscle and splanchnic organs, kidneys, skin and other peripheral tissues.

Table 4. *Total oxygen consumed by the muscle from blood and muscle oxygen stores for different levels of muscle metabolic rate during aerobic dives with a cardiac output that gave the maximum aerobic dive limit*

$\dot{V}_{MO_2}$ (as a multiple of resting $\dot{V}_{MO_2}$)	$\dot{V}$b (% resting $\dot{V}$b)	ADL (min)	Blood O_2 consumed by muscle during dive[1] (ml)	Myoglobin O_2 consumed by muscle during dive[2] (ml)	Total O_2 consumed by muscle during dive (ml)	% of $\dot{V}_{MO_2}$ that came from oxymyoglobin (%)
1	19	28.0	6079 (20%)	212 (2%)	6290	3
2	19	24.0	7229 (24%)	3390 (30%)	10619	32
3	19	24.0	7229 (24%)	8574 (75%)	15803	54
4	19	20.3	6429 (21%)	11279 (99%)	17708	64
5	37	18.0	9978 (33%)	9627 (84%)	19606	49
6	37	15.8	9202 (30%)	11355 (100%)	20557	55
7	56	13.3	11167 (37%)	9003 (79%)	20170	45
8	56	12.7	10817 (36%)	11081 (97%)	21898	51
9	75	12.0	12723 (42%)	10729 (94%)	23451	46
10	75	10.8	11942 (39%)	11389 (100%)	23331	49
11	94	9.6	12998 (43%)	9894 (87%)	22892	43
12	94	9.2	12664 (42%)	11262 (99%)	23925	47
13	94	8.2	11828 (39%)	11268 (99%)	23096	49
14	94	7.4	11049 (36%)	11392 (100%)	22441	51
15	94	6.6	10187 (34%)	11254 (99%)	21441	52
16	94	6.0	9529 (31%)	11259 (99%)	20787	54

$\dot{V}_{MO_2}$, rate of skeletal muscle oxygen uptake; $\dot{V}$b, cardiac output; ADL, aerobic dive limit.
[1]Values in parentheses are the percentages of total blood oxygen (i.e. 30 275 ml O_2) consumed by the muscle for the entire dive.
[2]Values in parentheses are the percentages of total oxymyoglobin (i.e. 11 397 ml O_2) consumed by the muscle for the entire dive.

and function were 22 mmHg (Sa_{O_2}=38 %) and 18 mmHg ($S\bar{v}_{O_2}$=27 %), respectively. This is comparable to the mean Pa_{O_2} (24.5±2.86 mmHg; mean ± S.D., N=7) in Weddell seals 2 min before surfacing and to the end-tidal P_{O_2} (24 mmHg) of the first exhalation (assuming that this approximates arterial P_{O_2}) after 17 min aerobic dives (Qvist et al., 1986; Ponganis et al., 1993a). These minimum levels of Pa_{O_2} and $P\bar{v}_{O_2}$ are greater than the 10–15 mmHg that produces abnormal electroencephalographic (EEG) slow waves in Weddell seals and harbor seals during experimental asphyxia or forced submersion (Elsner et al., 1970; Kerem and Elsner, 1973). EEG slow waves indicate incipient brain metabolic impairment and deterioration of cerebral function. As a result, the model terminated a dive if Pa_{O_2} decreased below 22 mmHg. However, the ADL was reached for most (14 out of 16) combinations of $\dot{V}$b and $\dot{V}_{MO_2}$ before Pa_{O_2} decreased to this level (see Table 5). As a result, the Pa_{O_2} of blood perfusing the brain was generally not a consideration in determining ADL.

We assumed that $\dot{Q}_H$ and $\dot{V}_{HO_2}$ decreased proportionately with the reduction in $\dot{V}$b (Blix et al., 1976; Kjekshus et al., 1982; Blix and Folkow, 1983) (Tables 2, 3). Although convective oxygen transport to the myocardium decreased during a dive, it was proportional to the reduction in heart work, and the myocardium always received sufficient blood oxygen to maintain aerobic metabolism.

$\dot{Q}_M$ was also assumed to decrease proportionately with a decrease in $\dot{V}$b (Table 2). Oxygen transported to the muscles in the blood was always used (up to a maximum E_{BO_2} of 0.8) before oxygen bound to myoglobin because of the lower affinity of Hb for oxygen (Schenkman et al., 1997). Oxygen not provided by the blood was obtained from oxymyoglobin stores to meet $\dot{V}_{MO_2}$ requirements. $\dot{V}_{MO_2}$ was assumed to be independent of $\dot{Q}_M$ as long as the combination of convective oxygen transport and oxymyoglobin stores was sufficient to meet metabolic demand.

We assumed that $\dot{Q}_{SRC}$ decreased proportionately with $\dot{V}$b (Table 2). However, $\dot{V}_{SRCO_2}$ remained at resting, predive levels as long as (1) convective oxygen transport was sufficient to support oxygen demand, (2) E_{BO_2} did not exceed 0.8 and (3) Pa_{O_2} was greater than 22 mmHg (Kvietys and Granger, 1982; Schlichtig et al., 1992). Our assumption of sustained splanchnic and renal $\dot{V}_{O_2}$ is consistent with data on hepatic, renal and gastrointestinal function in Weddell seals during aerobic dives (Davis et al., 1983). In addition, splanchnic and whole-body $\dot{V}_{O_2}$ are maintained during progressive arterial hypoxemia in intact pigs down to a Pa_{O_2} of 23 mmHg (Noldge-Schomburg et al., 1996), a value similar to the minimum Pa_{O_2} in this model.

The muscle oxygen diffusive conductance (D_{MO_2}) was estimated from equation 11 describing Fick's law of diffusion:

$$D_{MO_2} = \dot{V}_{MO_2}/(P\bar{c}_{O_2} - Pmito_{O_2}), \quad (11)$$

where $P\bar{c}_{O_2}$ is mean capillary oxygen partial pressure and $Pmito_{O_2}$ is mitochondrial oxygen partial pressure. We assumed (1) that $Pmito_{O_2}$ was close enough to zero to be neglected (Kurdak et al., 1996), (2) that muscle diffusive conductance was constant at each point along the capillary so that $D_{MO_2}/\dot{Q}_M$ was uniform throughout the muscle (Kurdak et al., 1996), and (3) that all residual oxygen in the muscle venous blood was due to diffusion limitation of oxygen transport or to blood flow

heterogeneity (Hogan et al., 1993; Kurdak et al., 1996), with a maximum $E_{B\dot{O}_2}$ set at 0.8. $P\bar{c}_{\dot{O}_2}$ was calculated as the mean of $Pa_{\dot{O}_2}$ and the skeletal muscle blood oxygen partial pressure ($P_{M\bar{v}\dot{O}_2}$), which were computed from their respective $Ca_{\dot{O}_2}$ and $C_{M\bar{v}\dot{O}_2}$ (equation 8) using the oxyhemoglobin dissociation curve for Weddell seals (Qvist et al., 1981). Since $Pa_{\dot{O}_2}$ and $P_{M\bar{v}\dot{O}_2}$ decreased throughout the dive, $P\bar{c}_{\dot{O}_2}$ was recomputed for each pass of the blood through the muscle and never fell below 15 mmHg (mean minimum $P\bar{c}_{\dot{O}_2}$ 16 mmHg; range 15–19 mmHg). Calculating $P\bar{c}_{\dot{O}_2}$ as the mean of $Pa_{\dot{O}_2}$ and $P_{M\bar{v}\dot{O}_2}$ instead of using a numerical integration technique underestimates $D_{M\dot{O}_2}$ by 15–20 % (Kurdak et al., 1996). However, $D_{M\dot{O}_2}$ for a diving Weddell seal was so low compared with that of an exercising terrestrial mammal that this source of error was insignificant.

Computations

The model was run on a standard spreadsheet program (Quattro Pro for Windows Version 6.0, Novell Applications Group, Orem, Utah, USA) for five levels of $\dot{V}$b and 16 levels of $\dot{V}_{M\dot{O}_2}$ up to a maximum, whole-body $\dot{V}_{\dot{O}_2}$ of 9.2 ml O_2 min^{-1} kg^{-1}. This produced 80 combinations of $\dot{V}$b and $\dot{V}_{M\dot{O}_2}$. The general procedure was to set the $\dot{V}$b at a particular level (e.g. 37 % of the resting level) and then to vary $\dot{V}_{M\dot{O}_2}$ from 1 to 16 times the resting level. $\dot{V}_{\dot{O}_2}$ for the four vascular beds and the entire body were calculated for each combination. The ADL was reached and the dive terminated when any organ or tissue did not receive sufficient oxygen through convective oxygen transport or from oxymyoglobin (skeletal muscle only) to maintain aerobic metabolism or when $Pa_{\dot{O}_2}$ fell below 22 mmHg.

Results

The role of $\dot{V}$b in optimizing the ADL at different levels of muscle metabolism

The ADL decreased in a non-linear fashion with increasing $\dot{V}_{M\dot{O}_2}$ and whole-body $\dot{V}_{\dot{O}_2}$ for $\dot{V}$b ranging from 19 to 94 % of resting levels (Fig. 2). Among the five $\dot{V}$b curves, there was a maximum and a minimum ADL for each level of $\dot{V}_{M\dot{O}_2}$ and $\dot{V}_{\dot{O}_2}$. For example, the maximum ADL at five times resting $\dot{V}_{M\dot{O}_2}$ (whole-body $\dot{V}_{\dot{O}_2}$=3.8 ml O_2 min^{-1} kg^{-1}) was 18 min for a $\dot{V}$b of 37 %. In contrast, the minimum ADL for the same $\dot{V}_{M\dot{O}_2}$ was 10.4 min for a $\dot{V}$b of 94 %. Between the maximum and minimum values of ADL, there were intermediate values for each $\dot{V}_{M\dot{O}_2}$ and $\dot{V}_{\dot{O}_2}$. This relationship is more apparent if we plot the maximum and minimum ADL among the five curves for each level of $\dot{V}_{M\dot{O}_2}$ and $\dot{V}_{\dot{O}_2}$ (Fig. 3). The maximum ADL (i.e. ADL with the optimum $\dot{V}$b) and minimum ADL (i.e. ADL with the least-optimum $\dot{V}$b) decreased in parallel as muscle and whole-body $\dot{V}_{\dot{O}_2}$ increased. The average ratio of the maximum to minimum ADL for each level of $\dot{V}_{M\dot{O}_2}$ and $\dot{V}_{\dot{O}_2}$ was 1.5 (range 1.4–1.8); that is, the optimum $\dot{V}$b increased the ADL by an average of 50 %.

Table 5. *Arterial, mixed venous and muscle venous blood oxygen saturation and partial pressure at the beginning and end of aerobic dives for different levels of muscle metabolism*

$\dot{V}_{M\dot{O}_2}$ (as a multiple of resting $\dot{V}_{M\dot{O}_2}$)	$\dot{V}$b (% resting $\dot{V}$b)	ADL (min)	Initial $Sa_{\dot{O}_2}$ and $Pa_{\dot{O}_2}$ % (mmHg)	Final $Sa_{\dot{O}_2}$ and $Pa_{\dot{O}_2}$ % (mmHg)	Arterial desaturation rate (% min^{-1})	Initial $S\bar{v}_{\dot{O}_2}$ and $P\bar{v}_{\dot{O}_2}$ % (mmHg)	Final $S\bar{v}_{\dot{O}_2}$ and $P\bar{v}_{\dot{O}_2}$ % (mmHg)	Initial $S_{M\bar{v}\dot{O}_2}$ and $P_{M\bar{v}\dot{O}_2}$ % (mmHg)	Final $S_{M\bar{v}\dot{O}_2}$ and $P_{M\bar{v}\dot{O}_2}$ % (mmHg)
1	19	28.0	100 (119)	42 (23)	2.1	86 (55)	14 (12)	56 (29)	8 (8)
2	19	24.0	100 (119)	52 (27)	2.0	86 (55)	23 (16)	20 (15)	11 (10)
3	19	24.0	100 (119)	52 (27)	2.0	86 (55)	23 (16)	20 (15)	10 (9)
4	19	20.3	100 (119)	52 (27)	2.3	86 (55)	23 (16)	20 (15)	10 (9)
5	37	18.0	100 (119)	42 (23)	3.2	86 (55)	25 (17)	20 (15)	8 (8)
6	37	15.8	100 (119)	42 (23)	3.7	86 (55)	25 (17)	20 (15)	8 (8)
7	56	13.3	100 (119)	43 (23)	4.3	86 (55)	29 (19)	20 (15)	8 (8)
8	56	12.7	100 (119)	43 (23)	4.5	86 (55)	29 (19)	20 (15)	8 (8)
9	75	12.0	100 (119)	38 (22)	5.2	86 (55)	27 (18)	20 (15)	8 (8)
10	75	10.8	100 (119)	38 (22)	5.8	86 (55)	27 (18)	20 (15)	8 (8)
11	94	9.6	100 (119)	41 (23)	6.2	86 (55)	30 (19)	20 (15)	8 (8)
12	94	9.2	100 (119)	41 (23)	6.5	86 (55)	30 (19)	20 (15)	8 (8)
13	94	8.2	100 (119)	41 (23)	7.3	86 (55)	30 (19)	20 (15)	8 (8)
14	94	7.4	100 (119)	50 (26)	6.8	86 (55)	37 (22)	20 (15)	10 (9)
15	94	6.6	100 (119)	53 (28)	7.1	86 (55)	41 (23)	20 (15)	11 (10)
16	94	6.0	100 (119)	53 (28)	7.8	86 (55)	41 (23)	20 (15)	11 (10)
Mean			100 (119)	45 (24)		86 (55)	28 (18)	22 (16)	9 (9)

The ADL was reached and the dive terminated if an organ or tissue became anaerobic or if the $Sa_{\dot{O}_2}$ decreased below 38 %.

$\dot{V}_{M\dot{O}_2}$, rate of skeletal muscle oxygen consumption; $\dot{V}$b, cardiac output; ADL, aerobic dive limit; $Sa_{\dot{O}_2}$, $S\bar{v}_{\dot{O}_2}$, $S_{M\bar{v}\dot{O}_2}$, arterial, mixed venous and muscle venous blood oxygen saturation; $Pa_{\dot{O}_2}$, $P\bar{v}_{\dot{O}_2}$ and $P_{M\bar{v}\dot{O}_2}$, arterial, mixed venous and muscle venous blood oxygen partial pressure.

1 mmHg=0.133 kPa.

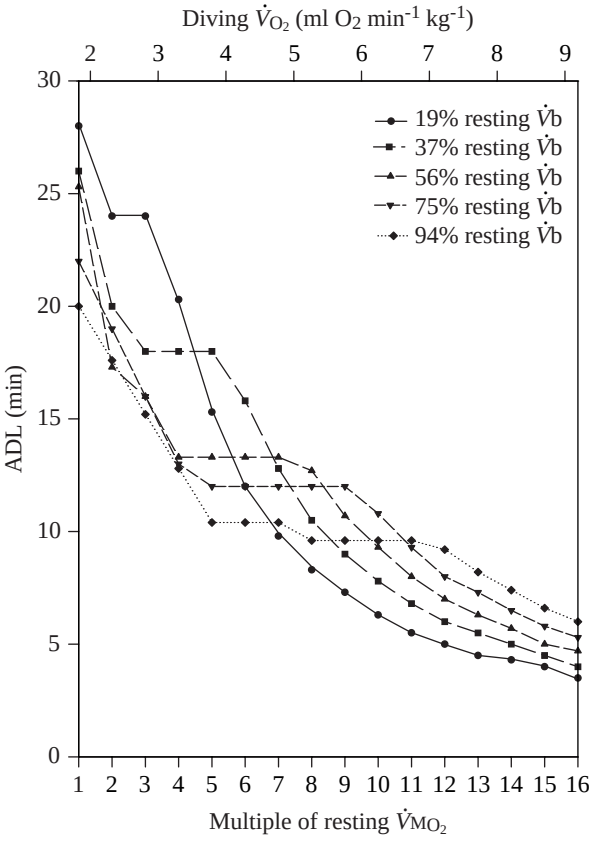

Fig. 2. Calculated aerobic dive limit (ADL) for five levels of cardiac output ($\dot{V}$b) as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) and whole-body oxygen consumption rate ($\dot{V}_{O_2}$). Resting $\dot{V}_{MO_2}$ was 216 ml O_2 min^{-1} or 1.4 ml O_2 min^{-1} kg^{-1} muscle.

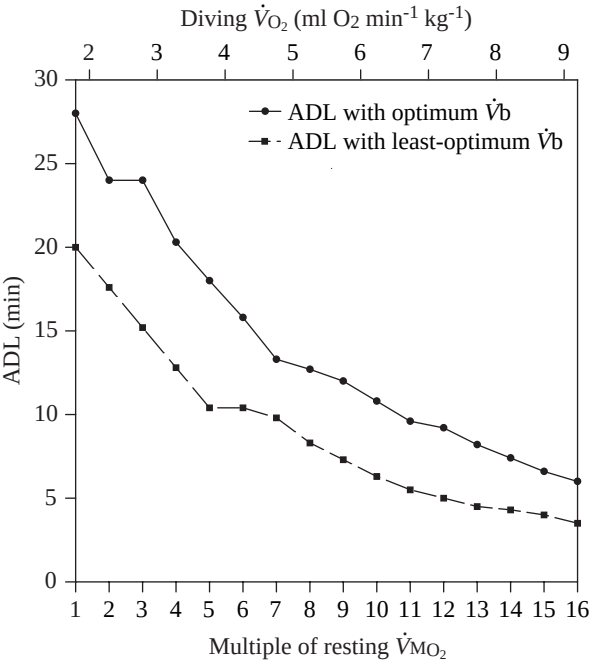

Fig. 3. Maximum and minimum aerobic dive limit (ADL) as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) and whole-body oxygen consumption rate ($\dot{V}_{O_2}$). All values are from Fig. 2. Optimum and least-optimum cardiac outputs ($\dot{V}$b) refer to the $\dot{V}$b value for a particular $\dot{V}_{MO_2}$ value that gives the maximum and minimum ADL, respectively.

If we plot the values of $\dot{V}$b and f_H that give the maximum ADL as a function of $\dot{V}_{MO_2}$ and whole-body $\dot{V}_{O_2}$, we obtain the relationship shown in Fig. 4. These results show that the dive response should be less pronounced as the level of muscular exertion increases. However, the model predicts that the optimum f_H and $\dot{V}$b are greater than resting levels (i.e. tachycardia) for $\dot{V}_{MO_2}$ values greater than 12 times the resting value (a whole-body $\dot{V}_{O_2}$ of 7.3 ml O_2 min^{-1} kg^{-1}). In the model, we assumed that some degree of dive response was obligatory so that the maximum f_H and $\dot{V}$b were 94 % of the resting values, even though this did not result in the maximum use of blood oxygen stores at $\dot{V}_{MO_2}$ values greater than 12 times the resting value. We also ran the model at 9 % resting $\dot{V}$b (f_H=6.5 beats min^{-1}) to determine whether the ADL was further enhanced at 1–4 times resting $\dot{V}_{MO_2}$ (a whole-body $\dot{V}_{O_2}$ of 1.8–3.3 ml O_2 min^{-1} kg^{-1}). However, 19 % $\dot{V}$b gave a higher ADL because convective oxygen transport to the splanchnic organs, kidneys and other tissues (excluding the brain and heart) at 9 % $\dot{V}$b was insufficient to support aerobic metabolism at an Sa_{O_2} value less than 88 %. Hence, the convective oxygen requirements of organs and tissues other than skeletal muscle established the lower limit of optimum $\dot{V}$b at low levels (1–4 times the resting value) of muscular exertion.

Figs 3 and 4 show that there is not one but a suite of ADLs that depend on $\dot{V}_{O_2}$ and that, within limits, the optimum $\dot{V}$b and f_H increase with $\dot{V}_{MO_2}$ and $\dot{V}_{O_2}$. Since the ADL is inversely proportional to $\dot{V}_{O_2}$ (assuming a constant level of blood and muscle oxygen depletion), the optimum $\dot{V}$b and f_H decrease as ADL increases (Fig. 5). However, for an ADL less than 9.6 min and greater than 20.3 min, optimum $\dot{V}$b and f_H are maximally limited to resting levels at the surface and minimally limited (i.e. 19 % $\dot{V}$b) by convective oxygen transport sufficient to maintain aerobic metabolism in all organs and tissues, respectively.

Organ and tissue metabolism

$\dot{V}_{BO_2}$, which was independent of $\dot{V}$b in this model, represented a very small part (0.3–1.7 %) of whole-body $\dot{V}_{O_2}$ (Table 3). As $\dot{V}$b increased with muscular exertion, $\dot{V}_{HO_2}$ increased in direct proportion to heart work. However, it represented only 1–3 % of whole-body $\dot{V}_{O_2}$, even at the highest levels of $\dot{V}$b. $\dot{V}_{SCRO_2}$ was also independent of blood flow (i.e. convective oxygen transport was always sufficient to maintain aerobic metabolism), but represented a decreasing percentage (range 69–13 %) of whole-body $\dot{V}_{O_2}$ as muscle metabolic rate increased. In contrast, $\dot{V}_{MO_2}$ represented an increasingly greater percentage (range 27–84 %) of whole-body $\dot{V}_{O_2}$ as muscular exertion increased up to 16 times resting levels. Together, $\dot{V}_{MO_2}$ and $\dot{V}_{SCRO_2}$ accounted for 96–98 % of whole-body metabolic rate at all levels of muscular exertion.

Muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio

To maximize ADL, the increase in $\dot{V}$b with $\dot{V}_{MO_2}$ can be explained by the relationship between muscle $\dot{Q}_{O_2}$ and $\dot{V}_{O_2}$. Fig. 6 shows the muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio at the beginning of a dive as a function of $\dot{V}_{MO_2}$ for the five levels of $\dot{V}$b. The bold line connecting the five curves is the $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio for each level of $\dot{V}$b that gives the maximum ADL for $\dot{V}_{MO_2}$ ranging from 3 to 16 times resting levels. As mentioned above, $\dot{V}$b could not be optimized for 1–2 times resting $\dot{V}_{MO_2}$. The average optimum muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio at the beginning of a dive was 0.88 (range 0.59–1.08). For a $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio below this range, convective oxygen transport to the muscles was too low. As a result, muscle oxymyoglobin stores were depleted before blood oxygen, and the ADL decreased for all levels of $\dot{V}_{MO_2}$. For a $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio above this range, convective oxygen transport to the muscles was too high. In this case, blood oxygen was depleted before muscle oxymyoglobin stores were desaturated, and again the ADL decreased for each level of $\dot{V}_{MO_2}$. In both cases, an inappropriate $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio resulted in a decrease in the ADL because the blood and muscle oxygen stores were not completely used. To maximize the use of body oxygen stores and the ADL for each level of exertion, the full utilization of blood (Sa_{O_2} not less than 38 %) and muscle oxygen stores must be completed at the same time. This is achieved by adjusting $\dot{V}$b according to the level of $\dot{V}_{MO_2}$. As $\dot{V}_{MO_2}$ increases, $\dot{V}$b and muscle perfusion must also increase (up to a maximum of resting, predive $\dot{V}$b and $\dot{Q}_M$) so that the $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio stays within the range 0.59–1.08 at the beginning of a dive.

Although the reduction in $\dot{V}$b was kept constant once a dive began, the muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio declined because Ca_{O_2} decreased continuously as blood oxygen was consumed (Fig. 7). The higher the muscle metabolic rate, the faster the ratio declined. The difference between convective oxygen transport and muscle oxygen requirements was provided by oxymyoglobin. The percentage of $\dot{V}_{MO_2}$ provided by the blood decreased and that provided by endogenous oxymyoglobin stores increased throughout the dive (Fig. 8). At the beginning of dives with a $\dot{V}_{MO_2}$ of 1–2 times resting, (Fig. 8A,B), blood oxygen provided 100 and 93 % of the muscle oxygen consumed, respectively. By the end of these dives, the percentage of $\dot{V}_{MO_2}$ provided by the blood had decreased to 76 and 48 %, respectively, with the remainder provided by oxymyoglobin. Because $\dot{V}$b and $\dot{Q}_M$ (i.e. 19 %) were greater than optimum levels for $\dot{V}_{MO_2}$ of 1–2 times resting level, only 3 and 32 %, respectively, of the total oxygen consumed by the muscle was provided by oxymyoglobin and the remainder by the blood (Table 4). As mentioned above, optimizing muscle $\dot{Q}_{O_2}$ by decreasing $\dot{V}$b below 19 % decreased the ADL because the splanchnic organs and kidneys became anaerobic much sooner at such a low level of perfusion. As a result, an optimum $\dot{V}$b and $\dot{Q}_M$ resulting in the full use of muscle oxygen stores could not be achieved for dives with these low levels of $\dot{V}_{MO_2}$ even though they still had the highest ADL.

For dives with a $\dot{V}_{MO_2}$ of 3–16 times resting levels (Fig. 8C–P), the average initial percentage of oxygen provided by the blood was 71 % (range 60–86 %), and this decreased to 31 % (range 27–35 %) by the end of the dive. Conversely, the average initial percentage of oxygen provided by muscle oxymyoglobin was 29 % (range 14–40 %), and this increased to 69 % (range 65–73 %) by the end of the dive. In most cases, the point at which the blood and muscle oxygen each supported approximately half of $\dot{V}_{MO_2}$ occurred approximately midway

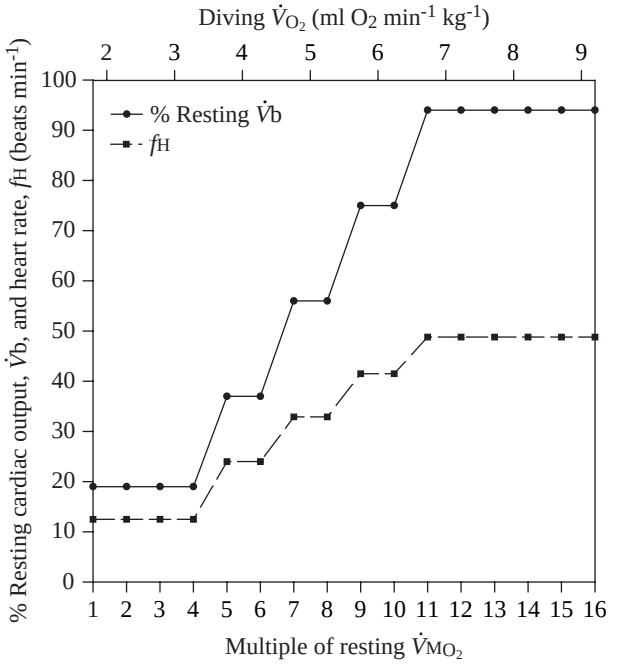

Fig. 4. Optimum cardiac output ($\dot{V}$b) and heart rate (f_H) as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) and whole-body oxygen consumption rate ($\dot{V}_{O_2}$). Optimum refers to values that give the maximum aerobic dive limit.

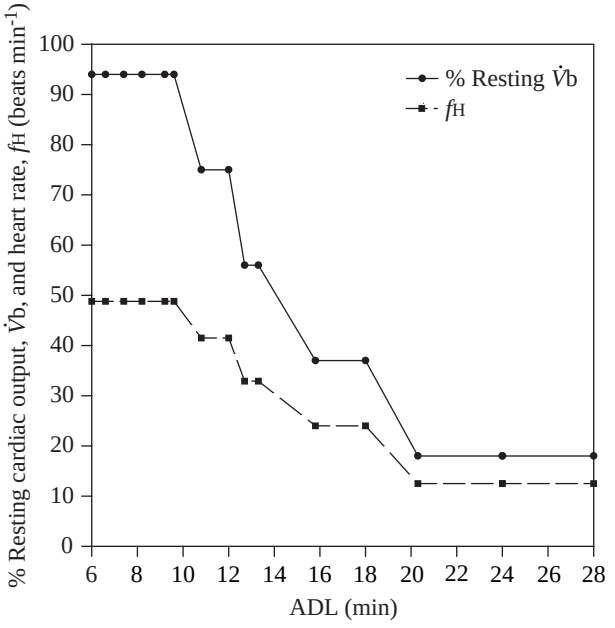

Fig. 5. Optimum cardiac output ($\dot{V}$b) and heart rate (f_H) as a function of aerobic dive limit (ADL).

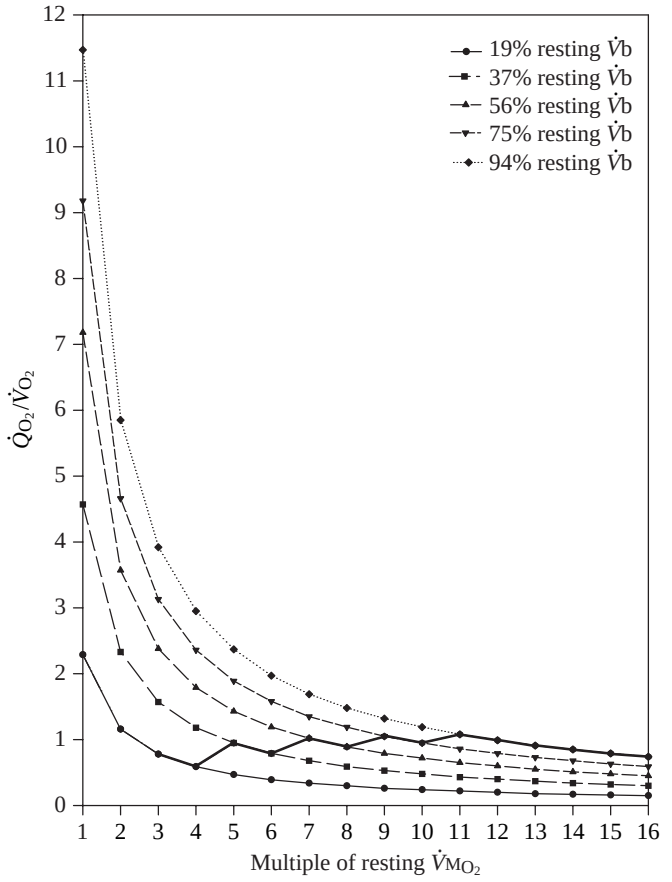

Fig. 6. Muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio at the beginning of dives for five levels of cardiac output ($\dot{V}$b) as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{M_{O_2}}$). The bold line connects the optimum $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratios that give the maximum aerobic dive limit (ADL) at each $\dot{V}_{M_{O_2}}$. $\dot{Q}_{O_2}$, convective oxygen transfer rate in the blood; $\dot{V}_{O_2}$, rate of oxygen consumption.

through the dives. Convective oxygen transport contributed to muscle metabolism throughout the dive ($\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio never fell below 0.30), although this contribution diminished as Ca_{O_2} decreased. Approximately 51 % (range 43–64 %) of the total oxygen consumed by the muscle came from oxymyoglobin and the remainder from blood oxygen (Table 4).

Blood oxygen desaturation and E_{BO_2}

Sa_{O_2} decreased from 100 % (Pa_{O_2}=119 mmHg) at the beginning of dives to an average of 45 % (Pa_{O_2}=24 mmHg) by the end of dives for each of the 16 levels of $\dot{V}_{M_{O_2}}$ (Table 5). The rate of arterial oxygen desaturation increased with $\dot{V}_{M_{O_2}}$ and ranged from 2.1 to 7.8 % min^{-1}. $S\bar{v}_{O_2}$ decreased from 86 % ($P\bar{v}_{O_2}$=55 mmHg) to an average of 28 % ($P\bar{v}_{O_2}$=18 mmHg). In contrast, $S_{MV_{O_2}}$ decreased from an average of 22 % ($P_{MV_{O_2}}$=16 mmHg) to 9 % ($P_{MV_{O_2}}$=9 mmHg), reflecting the much higher oxygen requirements and E_{BO_2} of active skeletal muscle. On average, 55 % of the arterial oxygen and 67 % of the venous oxygen (63 % of the total blood oxygen store; equations 3, 4) were used during aerobic dives. In most cases, the muscle could no longer sustain aerobic metabolism, and the dive was terminated before Sa_{O_2} fell below 38 % (Pa_{O_2}=22 mmHg). On average, 4961 ml of arterial oxygen and 6267 ml of venous oxygen remained in the circulation at the end of aerobic dives.

As Sa_{O_2} decreased during a dive, E_{BO_2} increased for the brain, heart, splanchnic organs, kidneys and other tissues (Fig. 9A,B,D). For the brain and heart, E_{BO_2} never rose above 0.28 and 0.46, respectively. With their low oxygen requirements, convective oxygen transport was more than sufficient to sustain the aerobic metabolism of these two organs at all levels of $\dot{V}$b and Sa_{O_2}. Similarly, E_{BO_2} of the splanchnic organs, kidneys and other tissues remained below 0.32 except for the lowest $\dot{V}_{M_{O_2}}$ and $\dot{V}$b values (i.e. $\dot{V}_{M_{O_2}}$=1–4 times resting levels and $\dot{V}$b=19 %), for which E_{BO_2} increased to a maximum of 0.66. In contrast, E_{BO_2} of skeletal muscle was generally maximized at 0.8 throughout dives at all levels of $\dot{V}_{M_{O_2}}$ and $\dot{V}$b because the product of the optimum muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio (Fig. 7) and the maximum E_{BO_2} was less than 1. The exception was for resting $\dot{V}_{M_{O_2}}$ and 19 % $\dot{V}$b, for which E_{BO_2} increased from 0.44 to 0.8 during the dive.

Muscle oxygen diffusive conductance

Average $D_{M_{O_2}}$ was directly proportional to the percentage of resting $\dot{Q}_M$ and was best described by the equation $D_{M_{O_2}}=(3.75\times10^{-4})\dot{Q}_M-5.73\times10^{-4}$ (r^2=0.999, P=0.05) (Fig. 10). Overall, $D_{M_{O_2}}$ was dependent on muscle $\dot{Q}_{O_2}$ because E_{BO_2} was maximized (i.e. 0.8) throughout dives with a $\dot{V}_{M_{O_2}}$ of 2–16 times the resting level.

Oxymyoglobin desaturation

The desaturation of oxymyoglobin began immediately upon submergence and increased throughout dives for all $\dot{V}_{M_{O_2}}$ values greater than the resting value (Fig. 11). The immediate reliance on oxymyoglobin to support muscle metabolism in all but one case (i.e. resting $\dot{V}_{M_{O_2}}$) resulted from an optimum $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio that was less than 1.25 (Figs 6, 7) and a maximum E_{BO_2} of 0.8. As a result, convective oxygen transport was insufficient [i.e. $(\dot{Q}_{O_2}/\dot{V}_{O_2})\times0.8<1$] to support muscle metabolism, and oxymyoglobin was desaturated throughout the dives. On average, oxymyoglobin was 94 % desaturated (range 75–100 %) during dives with a $\dot{V}_{M_{O_2}}$ ranging from 3 to 16 times the resting value (Table 4). The average rate of oxymyoglobin desaturation increased linearly with $\dot{V}_{M_{O_2}}$ and was best described by the equation y=1.99x–2.99 (r^2=0.97, P=0.05) (Fig. 12).

Discussion

Why optimize the use of blood and muscle oxygen stores?

Unlike their terrestrial counterparts, marine mammals stop breathing and reduce convective oxygen transport while performing activities (e.g. foraging, courtship, aggressive interactions, predator avoidance and migration) that require sustained power output during submergence. This means that the ability of the pulmonary and cardiovascular systems to deliver oxygen to the muscles and other tissues is compromised

compared with terrestrial mammals with unlimited access to atmospheric oxygen. Since most voluntary dives are believed to remain aerobic (Butler and Jones, 1997), it is important that blood and muscle oxygen stores be used optimally to maximize the ADL. Prior to the model presented here, the redistribution of blood flow during aerobic dives has not been examined in terms of optimizing delivery of oxygen to peripheral tissues to sustain ATP synthesis.

How is oxygen distributed to the various organs and tissues to maximize the ADL? On the basis of the results from this model, the answer lies in adjusting $\dot{V}$b and peripheral vasoconstriction to match the level of muscular exertion. Blood oxygen is available to tissues throughout the body. However, endogenous oxygen stores in the muscle are not available to other tissues because myoglobin has a much higher affinity (P_{50}=2–3 mmHg; Rossi-Fanelli and Antonini, 1958; Gayeski et al., 1987; Schenkman et al., 1997) for oxygen than hemoglobin (P_{50}=27 mmHg; Qvist et al., 1981). To mobilize the muscle's endogenous oxygen supply and maximize ADL, the product of the muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio and maximum E_{BO_2} should be less than 1 (i.e. below the level of critical oxygen delivery) at the beginning of a dive (Fig. 6). As the dive progresses, muscle metabolism relies increasingly on its endogenous oxymyoglobin stores and less on convective oxygen transport as Ca_{O_2} and the $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio decrease (Fig. 7). Nevertheless, endogenous oxygen stores alone are

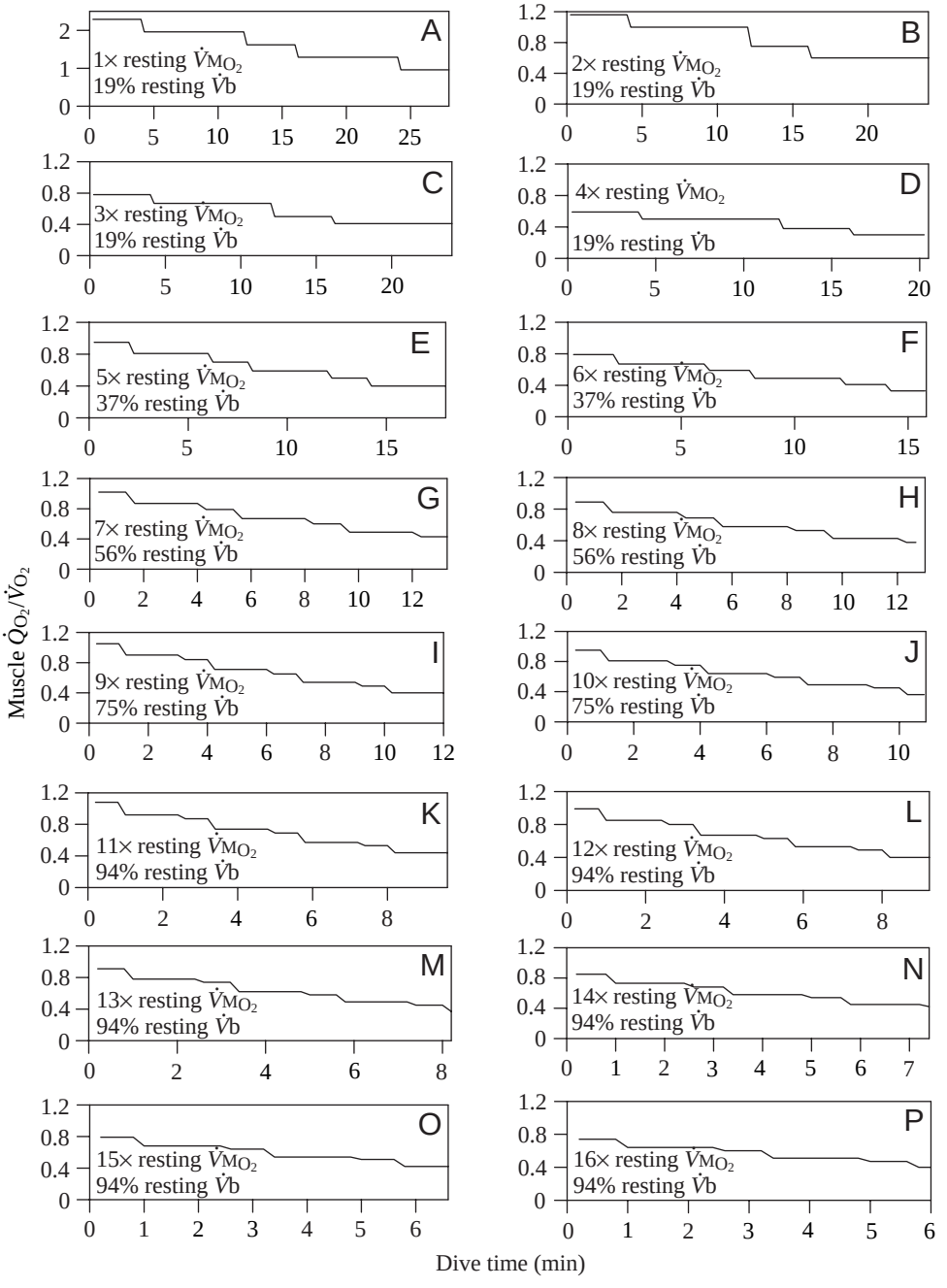

Fig. 7. Optimum muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratios as a function of dive time for skeletal muscle oxygen consumption rates ($\dot{V}_{MO_2}$) ranging from 1 to 16 times the resting value. The abscissa of each plot is scaled to its respective aerobic dive limit (ADL) to give maximum resolution. $\dot{Q}_{O_2}$, convective oxygen transfer rate in the blood; $\dot{V}_{O_2}$, rate of oxygen consumption; $\dot{V}$b, cardiac output.

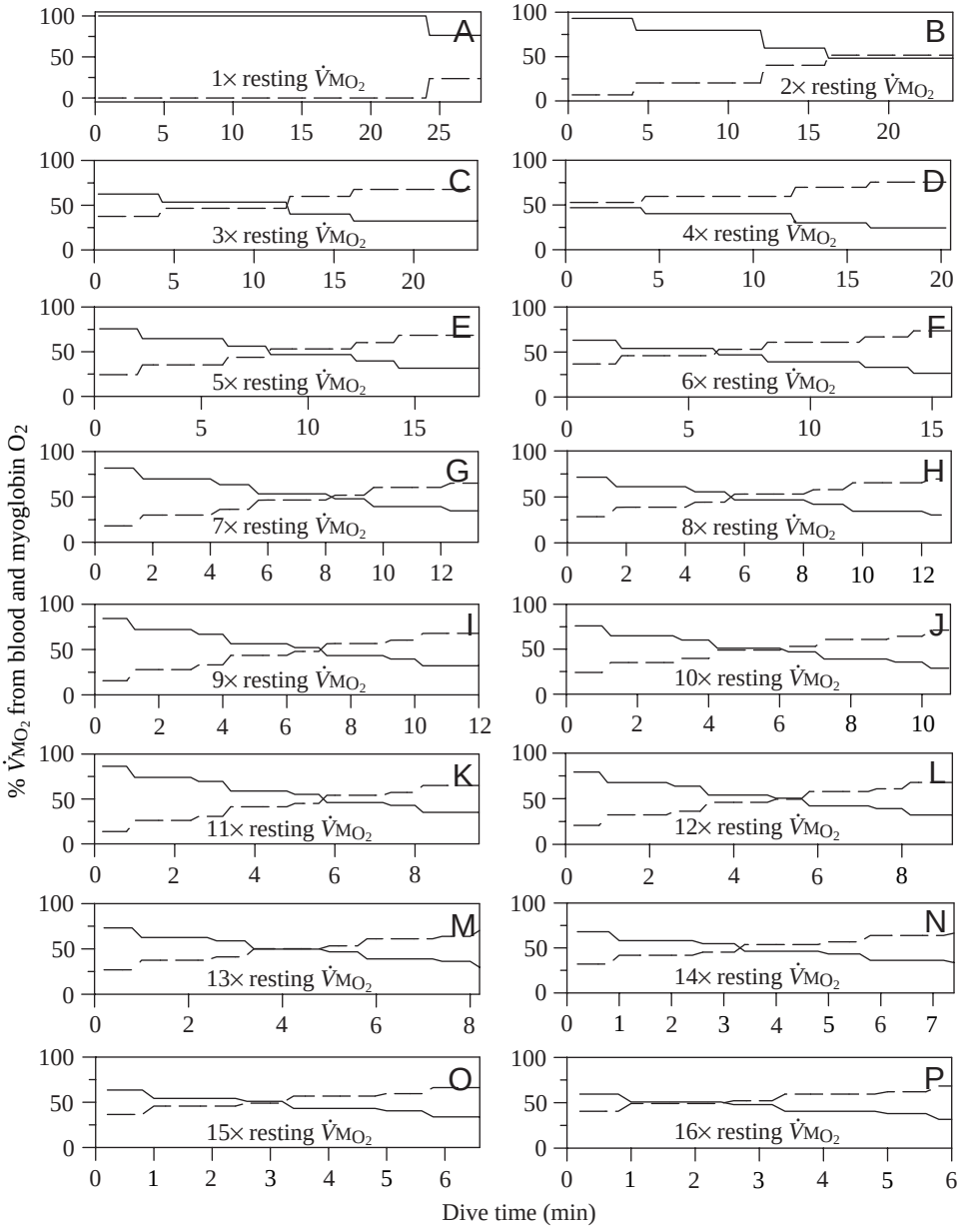

Fig. 8. Percentage of the skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) supported by oxygen from the blood (solid line) and from oxymyoglobin (dashed line) as a function of dive time for $\dot{V}_{MO_2}$ values ranging from 1 to 16 times the resting value. The abscissa of each plot is scaled to its respective aerobic dive limit (ADL) to give maximum resolution.

insufficient to support $\dot{V}_{MO_2}$ and, at the same time, maximize the ADL. Even towards the end of a dive, the muscle continues to receive over 24 % of its oxygen from the blood while simultaneously desaturating oxymyoglobin throughout the dive. This implies a subtle degree of control over $\dot{V}$b and muscle perfusion that is geared to the level of exercise. At higher levels of $\dot{V}_{MO_2}$, $\dot{V}$b and muscle perfusion must increase to maintain an appropriate $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio so that available blood and muscle stores are depleted at the same time.

Previous researchers have commented on the metabolic conflict created by the reduction in convective oxygen transport to active muscle during submerged swimming (Castellini et al., 1985; Hochachka, 1986, 1992; Fedak et al., 1988; Williams et al., 1991). Inevitably, any constraints on $\dot{Q}_{O_2}$ will limit maximum $\dot{V}_{O_2}$ ($\dot{V}_{O_2max}$). Assuming that some degree of dive response is obligatory (Kooyman, 1989; Hill et al., 1987; Fedak et al., 1988; Ponganis et al., 1997a; Butler and Jones, 1997), this conflict becomes apparent at a $\dot{V}_{MO_2}$ greater than 12 times the resting level and a maximum $\dot{V}$b of 94 % (Fig. 4). At this level of exertion, the model predicts an optimum f_H and $\dot{V}$b that are greater than resting levels, an indication of the emerging metabolic conflict between the dive response and muscle oxygen requirements. This conflict is manifest as a small but growing inefficiency in the use of blood oxygen stores (i.e. increasing end-of-dive Sa_{O_2} and $S\bar{v}_{O_2}$) which is detectable at a $\dot{V}_{MO_2}$ of 14 times the resting value and a $\dot{V}_{O_2}$ of 8.2 ml O_2 min^{-1} kg^{-1} (Fig. 13B,C). However, this is near the measured maximum $\dot{V}_{O_2}$ of 9 ml O_2 min^{-1} kg^{-1} (Castellini et al., 1992) for freely diving Weddell seals. As a result, Weddell seals may avoid (or be limited by) the conflict between the dive response and convective oxygen transport to active muscle by swimming at modest levels (i.e. $\dot{V}_{O_2} \leqslant 9$ ml O_2 min^{-1} kg^{-1}) of exertion during

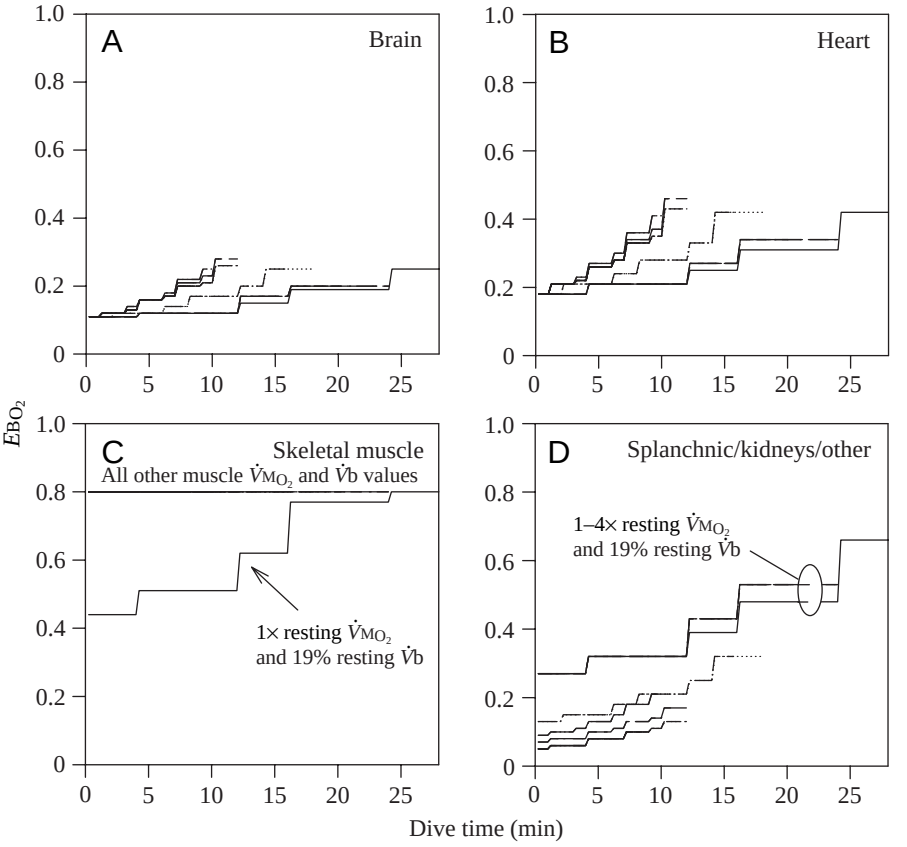

Fig. 9. Extraction coefficient of oxygen from the blood (E_{BO_2}) as a function of dive time for organs and tissues supplied by the four regional circulations indicated in Fig. 1. E_{BO_2} for the 16 levels of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) are plotted for each circulation, but in many cases the lines overlap. Each plot of E_{BO_2} ends at its respective aerobic dive limit (ADL). The objective of this graph is not to show the individual lines, but to show the trend and the range of maximum E_{BO_2} for each circulation. For skeletal muscle, the E_{BO_2} for $\dot{V}_{MO_2}$ ranging from 2 to 16 times the resting value were maximized at 0.8 throughout aerobic dives and were not individually discernible. For resting $\dot{V}_{MO_2}$, E_{BO_2} for skeletal muscle started at 0.44 and increased to 0.8 towards the end of the dive. The average maximum value of E_{BO_2} for each circulation was: brain, 0.24±0.002; heart, 0.40±0.003; skeletal muscle, 0.8±0; splanchnic/kidneys/other organs, 0.27±0.012 (means ± S.E.M., N=16). $\dot{V}$b, cardiac output.

diving. Under these conditions, blood and muscle oxygen stores can be used optimally during the progressive hypoxic hypoxia with ischemia that occurs during breath-hold diving by matching $\dot{V}$b to the level of $\dot{V}_{MO_2}$. These cardiovascular adjustments enable the diving animal to balance the conflicting metabolic demands of (1) optimizing the distribution and use of blood and muscle oxygen stores by all organs and tissues to maximize the ADL and (2) ensuring that active muscle receives adequate oxygen as $\dot{V}_{MO_2}$ increases during submerged swimming.

Consequences of not optimizing $\dot{V}$b

Using the least-optimum $\dot{V}$b (i.e. the $\dot{V}$b that gives the minimum ADL for a given and $\dot{V}_{O_2}$; Fig. 3) over the range 19–94% reduces blood and muscle oxygen desaturation (Fig. 13). This reduction in desaturation decreases the percentage of total body oxygen stores that are used during aerobic dives and, therefore, decreases the ADL for a given $\dot{V}_{O_2}$. However, the under-utilization of blood and muscle oxygen stores is not the same for all levels of $\dot{V}_{MO_2}$. For $\dot{V}_{MO_2}$ values of 1–6 times resting, the least-optimum $\dot{V}$b (i.e. 94%) results in an over-perfusion of the muscle, a $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio greater than 0.88 (range 2.0–11.5) and a negligible rate of muscle oxymyoglobin desaturation (range 0–0.2% min^{-1}). For $\dot{V}_{MO_2}$ values of 7–16 times resting, the least-optimum $\dot{V}$b (i.e. 19%) results in an under-perfusion of the muscle, a very low $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio (range 0.2–0.3) and a very rapid desaturation of muscle oxymyoglobin (range 10.1–26.8% min^{-1}). In the former case, the blood is desaturated and the ADL is reached, triggered by the oxygen requirements of the splanchnic organs, kidneys and peripheral tissues other than muscle, while muscle oxymyoglobin is still saturated. In the latter case, the muscle is rapidly depleted of oxygen and becomes anaerobic while there are still high levels of oxygen saturation in the arterial (86–100%) and venous (51–64%) blood. In either case, the mismatch between muscle $\dot{Q}_{O_2}$ and $\dot{V}_{O_2}$ results in very low or high rates, respectively, of oxymyoglobin desaturation that are outside the normal range of values observed for Weddell seals during aerobic dives (Guyton et al., 1995). In addition, the mismatch results in an end-of-dive Sa_{O_2} for $\dot{V}_{MO_2}$ of 7–16 times resting that is unusually high (i.e. 86–100%) for aerobic dives (Qvist et al., 1986). With the least-optimum $\dot{V}$b, an average of 45% (range 37–49%) of total body oxygen stores is used before the ADL is reached (Fig. 13A). In contrast, an average of 69% (range 54–76%) of total body oxygen stores is used with an optimum $\dot{V}$b, and this increases the average ADL by a factor of 1.5. We hypothesize that Weddell seals are adapted to make the most efficient use of oxygen stores for a given level of diving $\dot{V}_{O_2}$. This means that cardiovascular adjustments are established at the beginning of a dive to maximize the ADL, even if the dive is terminated for behavioral reasons before body oxygen stores are fully used.

Model predictions during aerobic dives with different levels of $\dot{V}_{O_2}$

Many aerobic dive limits

The model shows that there is not one but a suite of ADLs, the durations of which depend on the level of muscular

exertion during a dive. Although this is not a surprising statement, previous studies generally refer to a single ADL that is (1) based on the observed maximum dive duration without a post-dive increase in blood lactic acid level (Kooyman et al., 1980, 1983; Ponganis et al., 1997a,b) or (2) calculated as the quotient of the estimated body oxygen stores (in ml O_2) and an assumed diving $\dot{V}_{O_2}$ (in ml O_2 min^{-1}) (Ponganis et al., 1993a; Butler and Jones, 1997). However, the ADL is inversely proportional to $\dot{V}_{O_2}$ assuming that the level of oxygen depletion in the blood and skeletal muscle is constant during aerobic dives. As a result, each level of $\dot{V}_{O_2}$ has its own ADL, and the maximum ADL occurs when an animal is resting or sleeping submerged, although grey seals (*Halichoerus grypus*) may use a wait-and-ambush foraging strategy that results in a low foraging $\dot{V}_{O_2}$ (Fedak et al., 1988; Thompson et al., 1991). For free-ranging Weddell seals, maximum aerobic dives are approximately 25 min long (Kooyman et al., 1980), which is similar to the maximum ADL of 28 min estimated from the model. Aerobic dives that are shorter than 25 min may have been terminated before the oxygen stores had been completely exhausted, or the seal may have exercised resulting in a shorter ADL.

Average dive $\dot{V}_{O_2}$

How do the model's predictions compare with what is known about the diving metabolism and physiology of Weddell seals? The measured $\dot{V}_{O_2}$ over a dive/surface cycle for adult Weddell seals (average mass 355±59 kg; mean ± S.D., N=5), which is the current best estimate of diving $\dot{V}_{O_2}$, ranges from 2 to 9 ml O_2 min^{-1} kg^{-1}, with a significant negative correlation between dive duration and metabolic rate (Castellini et al., 1992). For an average metabolic rate of 5 ml O_2 min^{-1} kg^{-1}, maximum recorded dive duration is approximately 14 min (Castellini et al., 1992; see also Kooyman et al., 1983). Average f_H for 10–15 min aerobic dives is 39 beats min^{-1} (range 35–45 beats min^{-1}; Hill et al., 1987), and the average rate of muscle O_2 desaturation is 5.1 % min^{-1} (Guyton et al., 1995). The Sa_{O_2} and Pa_{O_2} at the end of aerobic dives average 44 % and 25 mmHg, respectively (Qvist et al., 1986). For the same diving $\dot{V}_{O_2}$ of 5 ml O_2 min^{-1} kg^{-1} ($\dot{V}_{M_{O_2}}$=7.5 times resting) and an optimum $\dot{V}$b of 56 %, our model predicts an ADL of 13 min, an optimum f_H of 33 beats min^{-1} and a muscle O_2 desaturation rate of 6.8 % min^{-1}. The predicted Sa_{O_2} and Pa_{O_2} at the end of the dive are 42 % and 23 mmHg, respectively. Using the optimum $\dot{V}$b, the model's predictions for ADL, f_H, muscle O_2 desaturation rate and end-of-dive Sa_{O_2} and Pa_{O_2} agree well with previous measurements and provide a unified explanation for the distribution and use of blood and muscle oxygen stores. By comparison, if we use the least-optimum $\dot{V}$b (i.e. 19 % $\dot{V}$b, f_H=12.5 beats min^{-1}; Fig. 2; Table 2) resulting in an inappropriately low $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio, the predicted ADL for a $\dot{V}_{O_2}$ of 5 ml O_2 min^{-1} kg^{-1} decreases to 9.1 min (a 30 % reduction), muscle O_2 desaturation rate increases to 11.0 % min^{-1} and the predicted Sa_{O_2} and Pa_{O_2} at the end of the dive increase to 86 % and 54 mmHg, respectively (Fig. 13). These latter values for f_H, ADL, Sa_{O_2} and Pa_{O_2} are inconsistent with previous measurements from Weddell seals during aerobic dives (Qvist et al., 1986; Hill et al., 1987; Castellini et al., 1992) and support the hypothesis that $\dot{V}$b is matched to the level of muscular exertion.

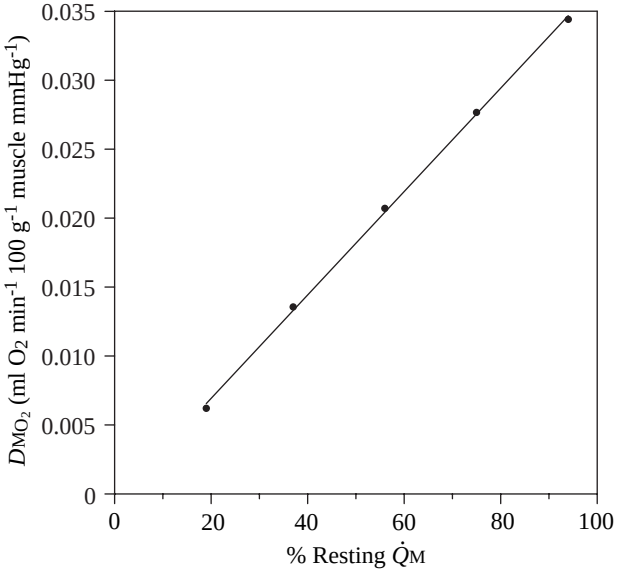

Fig. 10. Average muscle oxygen diffusive conductance ($D_{M_{O_2}}$) as a function of percentage resting skeletal muscle blood flow rate ($\dot{Q}_M$). Linear regression of the data was best described by the equation $D_{M_{O_2}}$=0.000375$\dot{Q}_M$–0.000573 (r^2=0.999, P=0.05).

Minimum dive $\dot{V}_{O_2}$

The minimum estimated $\dot{V}_{O_2}$ over an aerobic dive/surface cycle for Weddell seals is 2 ml O_2 min^{-1} kg^{-1} (Castellini et al., 1992; Ponganis et al., 1993a), which is identical to the basal metabolic rate estimated in this model (Table 1) and within the range predicted for mammals of similar size (Kleiber, 1975; McNab, 1989). This minimum estimate of diving $\dot{V}_{O_2}$ presumably includes any reduction in metabolic rate due to the 1–2 °C decrease in core body temperature that often occurs during dives within the ADL (Hill et al., 1987). Our model predicts a minimum metabolic rate of 1.8 ml O_2 min^{-1} kg^{-1} during submerged resting, which is slightly lower than basal value because $\dot{V}_{H_{O_2}}$ is only 19 % of the resting level (Table 3). At this minimum level of $\dot{V}_{O_2}$, the model predicts an ADL of 28 min, an f_H of 12.5 beats min^{-1}, a muscle O_2 desaturation rate of 0.07 % min^{-1} and end-of-dive Sa_{O_2} and Pa_{O_2} values of 42 % and 23 mmHg, respectively (Table 5; Fig. 12).

Although the predicted ADL under these conditions is long (28 min), muscle oxygen stores are barely used (only 3 % desaturation) because muscle $\dot{Q}_{O_2}$ is too high (Table 4; Fig. 8). To optimize the use of muscle oxygen, $\dot{V}$b would have to be reduced to less than 19 %. However, when we tested a $\dot{V}$b of 9 %, $\dot{Q}_{O_2}$ to the splanchnic organs, kidneys and other tissues (other than brain and heart) was inadequate to maintain aerobic metabolism at an Sa_{O_2} of less than 88 %, and the ADL was less than 28 min. The apparent conflict between the oxygen

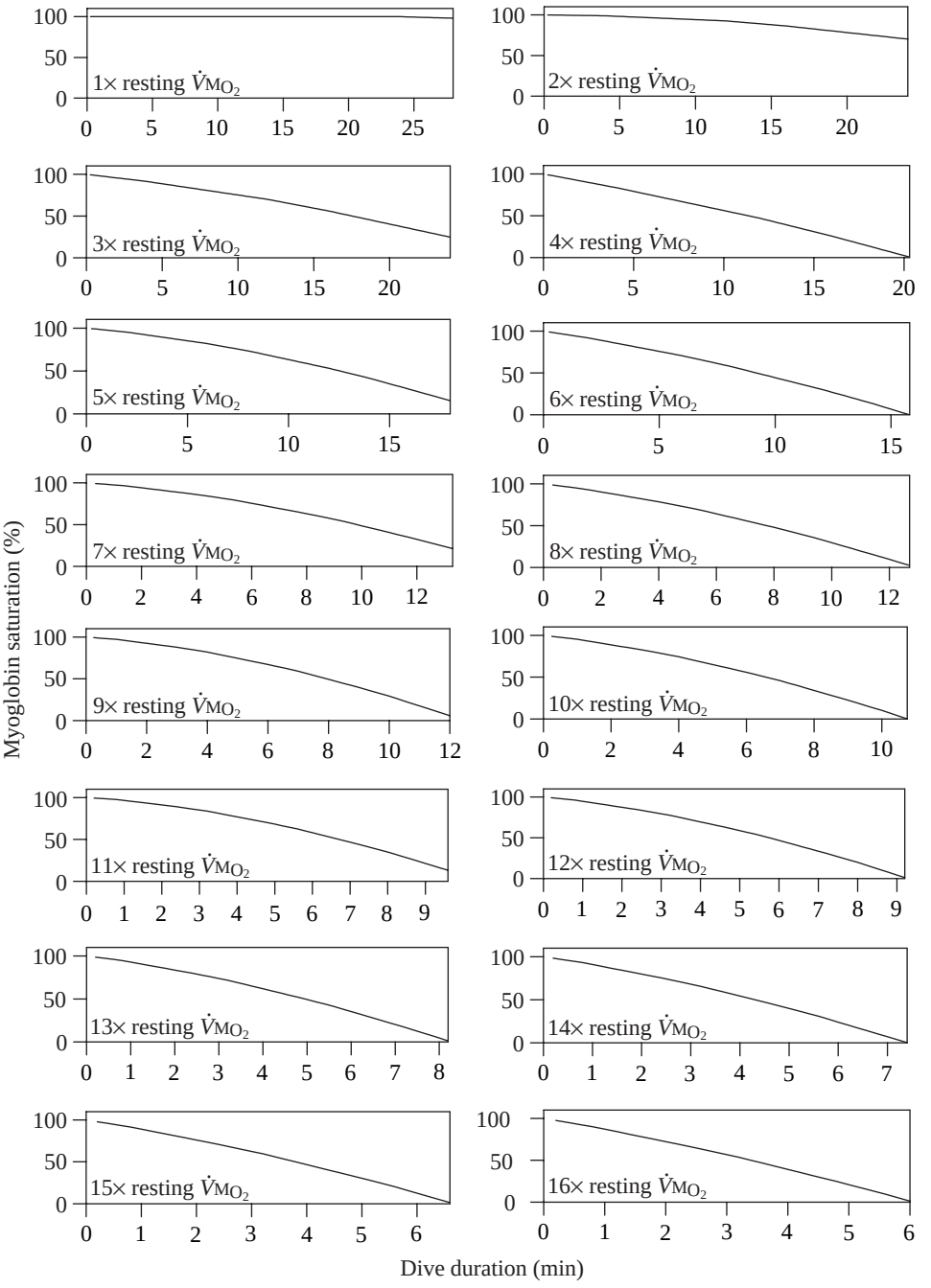

Fig. 11. Percentage oxymyoglobin saturation as a function of dive time for skeletal muscle oxygen consumption rates ($\dot{V}_{MO_2}$) ranging from 1 to 16 times the resting value. The abscissa of each plot is scaled to its respective aerobic dive limit (ADL) to give maximum resolution.

requirements of the splanchnic organs and the skeletal muscle created a lower limit for $\dot{V}$b during aerobic dives. The only way to resolve this conflict in the model is to reduce $\dot{V}_{SCRO_2}$, which would allow a $\dot{V}$b and $\dot{Q}_M$ of less than 19 %. Renal $\dot{V}_{O_2}$ in laboratory animals decreases down to a basal level of approximately 20 % with a reduction in blood flow and renal glomerular filtration rate (GFR) (Valtin, 1973). A graded reduction in renal $\dot{V}_{O_2}$ with decreasing $\dot{V}$b was not included in our model because available data indicate that renal GFR is maintained at resting, predive levels during most aerobic dives (Davis et al., 1983), even though some reduction in renal blood flow probably occurs. However, even if renal metabolic rate declined to basal levels, it would save only 49 ml O_2 min^{-1} or 5 % of resting, whole-body $\dot{V}_{O_2}$ (Table 1). In contrast to renal $\dot{V}_{O_2}$, there is little evidence of an adaptive decrease in oxygen demand (i.e. 'O_2 conformity') in the splanchnic organs and tissues of non-hibernating mammals due to a reduction in convective oxygen transport (Lautt and Graham, 1977; Grum et al., 1984; Schlichtig et al., 1992; Schlichtig and Bowles, 1994). Instead, the available data indicate that critical whole-organ oxygen delivery represents the threshold for ischemic dysoxia and anaerobiosis. A $\dot{V}$b of less than 19 % (f_H less than 12.5 beats min^{-1}) may represent the transition from aerobic to anaerobic dives because $\dot{Q}_{O_2}$ is insufficient to meet the oxygen requirements of peripheral organs and tissues other than skeletal muscle (Kooyman and Campbell, 1972; Zapol et al.,

1979). In addition, the splanchnic organs and kidneys may be insufficiently perfused at such a low $\dot{V}$b to maintain plasma metabolite homeostasis, renal GFR and digestion, a condition that does not occur during aerobic dives (Davis et al., 1983; Castellini et al., 1988). As a result, $\dot{V}$b may not decrease below a minimum level (approximately 19 %) during aerobic dives. Nevertheless, if it were possible to use the entire muscle oxygen store without the splanchnic organs, kidneys and other tissues becoming anaerobic, the ADL would increase to an impressive 39 min assuming average end-of-dive values of Sa_{O_2} and $S\bar{v}_{O_2}$ (Table 5).

Maximum dive $\dot{V}_{O_2}$

The maximum $\dot{V}_{O_2}$ measured over a dive/surface cycle for adult Weddell seals is 9 ml O_2 min^{-1} kg^{-1} during a maximum recorded dive duration of approximately 6 min (Castellini et al., 1992). For this level of diving $\dot{V}_{O_2}$ ($\dot{V}_{MO_2}$=15.5 times resting; Table 3) and a $\dot{V}$b of 94 %, our model predicts an ADL of 6.3 min, an f_H of 49 beats min^{-1}, a muscle O_2 desaturation rate of 15.7 % min^{-1} and end-of-dive Sa_{O_2} and Pa_{O_2} values of 53 % and 28 mmHg, respectively (Table 5; Fig. 12). Again, the model's predictions agree well with previous measurements of ADL and are within the normal range for f_H, muscle O_2 desaturation rate and end-of-dive Sa_{O_2} and Pa_{O_2} (Qvist et al., 1986; Hill et al., 1987; Guyton et al., 1995). By comparison, if we use the least-optimum $\dot{V}$b (i.e. 19 % $\dot{V}$b, f_H=12.5 beats min^{-1}; Fig. 2; Table 2), resulting in a very low $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio, the predicted ADL for a $\dot{V}_{O_2}$ of 9 ml O_2 min^{-1} kg^{-1} decreases to 3.8 min (a 40 % reduction). Furthermore, the muscle O_2 desaturation rate becomes very high at 25.9 % min^{-1}, and the end-of-dive Sa_{O_2} and Pa_{O_2} are 100 % and 119 mmHg, respectively (Fig. 13). With such a low $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio, muscle oxygen stores are depleted very quickly, and the ADL is reached before much blood oxygen is consumed. Under these conditions, the predicted values for muscle O_2 desaturation rate and end-of-dive Sa_{O_2} and Pa_{O_2} exceed the normal range for Weddell seals making aerobic dives.

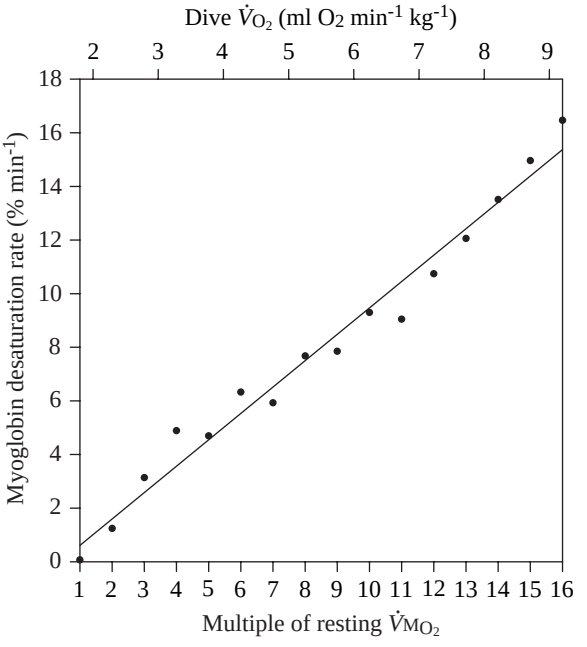

Fig. 12. Average rate of oxymyoglobin desaturation as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$) and whole-body oxygen consumption rate ($\dot{V}_{O_2}$). Linear regression of the data for $\dot{V}_{MO_2}$ was best described by the equation y=1.99x–2.99 (r^2=0.97, P=0.05).

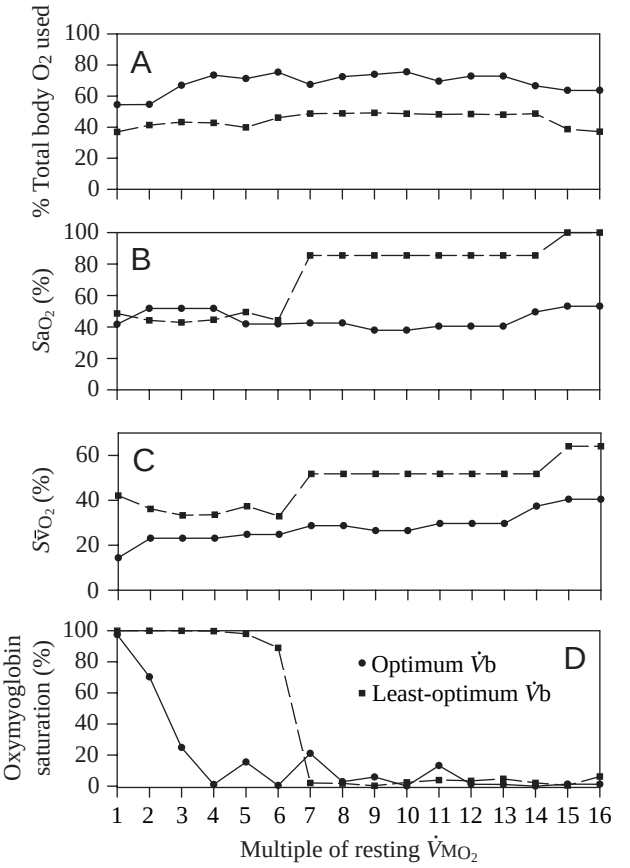

Fig. 13. Comparison of (A) percentage of total body oxygen (blood and muscle) used during a dive, (B) end-of-dive arterial blood oxygen saturation (Sa_{O_2}), (C) end-of-dive mixed venous blood oxygen saturation ($S\bar{v}_{O_2}$) and (D) end-of-dive oxymyoglobin saturation as a function of skeletal muscle oxygen consumption rate ($\dot{V}_{MO_2}$). Results were calculated using the optimum and least-optimum cardiac output ($\dot{V}$b). The least-optimum $\dot{V}$b for $\dot{V}_{MO_2}$ ranging from 1 to 6 times resting levels was 94 %. For $\dot{V}_{MO_2}$ ranging from 7 to 16 times resting, the least-optimum $\dot{V}$b was 19 %.

Although a $\dot{V}$b of 94 % gave the maximum ADL for a $\dot{V}_{O_2}$ of 9.2 ml O_2 min^{-1} kg^{-1}, it was not the optimum $\dot{V}$b. For metabolic rates higher than 7.3 ml O_2 min^{-1} kg^{-1} (12 times resting $\dot{V}_{MO_2}$), the optimum $\dot{V}$b was greater than resting levels; that is, no dive response at all (Fig. 4). For this model, we assumed that the maximum f_H and $\dot{V}$b during a dive were 94 % (i.e. approximately resting levels). However, if we extrapolate the results from Fig. 4 to a $\dot{V}_{O_2}$ of 9.2 ml O_2 min^{-1} kg^{-1} (16 times resting $\dot{V}_{MO_2}$), the optimum f_H and $\dot{V}$b would be 68 beats min^{-1} and 57 l min^{-1}, respectively, which is 32 %

greater than resting levels. Submergence without some degree of dive response has not been documented in marine mammals, and this physiological adjustment to diving appears to be obligatory, although its intensity varies (Kooyman, 1989; Hill et al., 1987; Fedak et al., 1988; Ponganis et al., 1997a; Butler and Jones, 1997). Although there is considerable psychogenic influence on heart rate, bradycardia is reflexly initiated by (1) trigeminal nerve stimulation when the face is wetted, (2) cessation of respiratory movements and lung compression, and (3) stimulation of the carotid bodies by decreasing arterial P_{O_2} (Dykes, 1974; Elsner and Gooden, 1983). If some degree of dive response cannot be avoided during submergence, it may limit the aerobic scope of Weddell seals (and other marine mammals) by placing an upper limit on convective oxygen transport to skeletal muscles.

The metabolic scope of harbor seals is approximately ninefold on the basis of $\dot{V}_{O_2}$ measurements during swimming trials in a water flume (Davis et al., 1991). This is less than the 15- to 30-fold scope for athletic terrestrial mammals of comparable size (Taylor et al., 1987). The maximum diving $\dot{V}_{O_2}$ ($9\,ml\,O_2\,min^{-1}\,kg^{-1}$) measured for Weddell seals (Castellini et al., 1992) appears to be only 31 % of the $\dot{V}_{O_2max}$ predicted for terrestrial mammals of equivalent size (Taylor et al., 1981), even though the mitochondrial volume density and citrate synthase activity of seal skeletal muscle may be comparable with those of animal athletes such as the pony (Kanatous et al., 1999). However, the estimated average P_{aO_2} (24 mmHg) at the end of aerobic dives (Table 5) is equivalent to the hypoxic hypoxia experienced by human climbers at an altitude of 8850 m (the height of Mount Everest), where $\dot{V}_{O_2max}$ is reduced to 25 % of that at sea level (West et al., 1983). Other factors that limit convective oxygen transport, such as a high hematocrit, may also constrain $\dot{V}_{O_2max}$ (Hedrick and Duffield, 1991). We therefore conclude that diving Weddell seals normally exercise at modest levels of $\dot{V}_{O_2}$ ($5\,ml\,O_2\,min^{-1}\,kg^{-1}$ or 15 % of the predicted $\dot{V}_{O_2max}$) to prolong dive duration and that their functional $\dot{V}_{O_2max}$ may be constrained by the inability to increase convective oxygen transport to muscle because of the dive response and progressive hypoxic hypoxia. As with terrestrial mammals, $\dot{V}_{O_2max}$ is probably limited by the availability of oxygen to muscle mitochondria, rather than by the muscle's oxidative capacity (Wagner et al., 1991).

Metabolism of the splanchnic organs, kidneys and other organs and tissues

The model shows that the $\dot{V}_{O_2}$ of the splanchnic organs, kidneys, lungs, reproductive organs, bone, skin and adipose tissue can be maintained at resting, predive levels over a range of ADLs. This is consistent with previous measurements of renal GFR, hepatic and gastrointestinal function (Davis et al., 1983) and blood metabolite concentrations (Castellini et al., 1988) in Weddell seals making repetitive, aerobic dives. Despite a reduction in blood flow and convective oxygen transport, the maximum E_{BO_2} of the splanchnic organs, kidneys and other peripheral tissues never rose above 0.66 (for $\dot{V}$b=19 %) and usually remained below 0.32 (for $\dot{V}$b>19 %) down to an end-of-dive average P_{aO_2} of 24 mmHg (Fig. 9D; Table 5). As with the brain and heart, $\dot{Q}_{O_2}$ did not limit the metabolic rate of these organs and tissues for $\dot{V}$b values ranging from 19 to 94 %. However, when blood flow was reduced to 9 %, E_{BO_2} was maximized at 0.8, and $\dot{Q}_{O_2}$ was insufficient to support aerobic metabolism (i.e. critical oxygen delivery) at an S_{aO_2} value less than 88 %. Below this value of S_{aO_2}, these organs and tissues would begin metabolizing anaerobically, resulting in the production of lactic acid (Schlichtig et al., 1992: Rozenfeld et al., 1996). As mentioned above, renal $\dot{V}_{O_2}$ may decrease with the reduction in blood flow (Kramer and Deetjen, 1964; Valtin, 1973) during diving, although renal GFR appears to be maintained during aerobic dives (Davis et al., 1983). In contrast, splanchnic organs maintain $\dot{V}_{O_2}$ until critical oxygen delivery is reached (Lautt and Graham, 1977; Hughes et al., 1979; Kvietys and Granger, 1982; Grum et al., 1984; Schlichtig et al., 1992; Samsel and Schumacker, 1994; Schlichtig and Bowles, 1994). In the model, this occurs at a $\dot{V}$b below 19 % (f_H less than $12.5\,beats\,min^{-1}$), which represents the transition from aerobic to anaerobic dives (Kooyman and Campbell, 1972; Zapol et al., 1979). However, for aerobic dives with a $\dot{V}$b greater than 19 %, $\dot{Q}_{O_2}$ is sufficient to maintain splanchnic and renal metabolism. This is critical for the maintenance of physiological homeostasis in Weddell seals and other marine mammals that may spend over 90 % of their time at sea submerged (Kooyman et al., 1980; Fedak, 1986; Le Boeuf et al., 1989; Hindell et al., 1992; Thompson et al., 1991; Butler and Jones, 1997).

Blood oxygen stores

In calculating the total body oxygen stores of Weddell seals and other marine mammals, previous investigators, including one of the present authors (R.W.D.), have assumed that approximately 80 % of the arterial oxygen and all the venous oxygen stores could be used to support aerobic metabolism during a dive (Kooyman et al., 1980, 1983; Ponganis et al., 1993a, 1997a,b). In our model, the ADL for a given $\dot{V}_{O_2}$ and optimum $\dot{V}$b was generally reached (i.e. skeletal muscle became anaerobic) before the average S_{aO_2} and $S\bar{v}_{O_2}$ fell below 45 % (P_{aO_2}=24 mmHg) and 28 % ($P\bar{v}_{O_2}$=18 mmHg), respectively (Fig. 13; Table 5). The former value is similar to the S_{aO_2} of 43.5±9.80 % (mean ± S.D., N=7) observed for Weddell seals at the end of voluntary dives lasting less than 17 min (Qvist et al., 1986). As a result, 11.23 l or 37 % of the total blood oxygen store remained unused at the end of a dive.

If the oxygen remaining in the arterial and venous blood were entirely available for metabolism, it would extend the ADL of a dive with an average $\dot{V}_{O_2}$ of approximately $4.8\,ml\,O_2\,min^{-1}\,kg^{-1}$ (Castellini et al., 1992) by 5.2 min (i.e. from 13.3 min, Table 3, to 18.5 min). Of course, arterial and venous blood cannot be entirely depleted of oxygen because diffusion limitations (see equation 11) would interrupt aerobic metabolism in most tissues, especially the brain which is sensitive to oxygen deficiency (Elsner and Gooden, 1983). However, if we extend the duration of a dive with a $\dot{V}_{O_2}$ of

4.8 ml O_2 min^{-1} kg^{-1} and an optimum $\dot{V}b$ of 56 % (ADL=13.3 min, Table 3) to 18.5 min, end-of-dive Pa_{O_2} decreases to 15.5 mmHg ($P\bar{v}_{O_2}$=9.4 mmHg), a dangerously low blood oxygen tension associated with abnormal EEG slow waves and brain metabolic impairment in Weddell seals and harbor seals (Elsner et al., 1970; Kerem and Elsner, 1973). Further reductions in arterial Pa_{O_2} could cause a seal to lose consciousness before reaching the surface. In addition, the skeletal muscle incurs an oxygen deficit of 3.33 l, equivalent to the formation of 839 mmol of lactate (assuming 22.414 ml O_2 $mmol^{-1}$ O_2, 5.65 mmol ATP $mmol^{-1}$ O_2 and 1 mmol ATP $mmol^{-1}$ lactate formed; Davis, 1983). This would produce an average lactate concentration in the skeletal muscle of 7.6 mmol l^{-1} (assuming that the body mass, 450 kg, is 35 % muscle and that muscle is 70 % water) and a peak, post-dive blood lactate concentration of approximately 4.1 mmol l^{-1} (assuming a volume of dilution equivalent to the skeletal muscle water space, see above, plus the blood volume, 96 l, or approximately half the body mass; Rowell et al., 1966). Despite extending the dive to 18.5 min, 4.63 l or 15 % of the total blood oxygen store would still remain. Since Weddell seals do not normally experience such a low Pa_{O_2} and little or no lactate is formed during dives lasting less than 17 min (Kooyman et al., 1980; Qvist et al., 1986; Ponganis et al., 1993a), a significant percentage (approximately 37 %) of total blood oxygen stores will probably be unused during most aerobic dives. However, this residual blood oxygen may serve as a reserve or 'safety margin' if an emergency arises (e.g. predator avoidance).

Muscle oxygen stores

To optimize the ADL for each level of $\dot{V}_{MO_2}$, the desaturation of muscle oxymyoglobin had to begin immediately upon submergence, even though some degree of convective oxygen transport was maintained. Using a laser near-infrared spectrophotometer to measure oxymyoglobin saturation on the surface of the non-locomotory latissimus dorsi muscle of freely diving Weddell seals, Guyton et al. (1995) observed that myoglobin desaturation began immediately upon submergence. The average rate of myoglobin desaturation for these dives was 7.3 % min^{-1}, with a range of 2–15 % min^{-1}. These rates of muscle oxymyoglobin desaturation are consistent with the results of our model over the range of $\dot{V}_{MO_2}$ (i.e. 2–16 times resting $\dot{V}_{MO_2}$; Figs 11, 12). For an average diving metabolic rate of 5 ml O_2 min^{-1} kg^{-1} (Castellini et al., 1992), our model predicts a $\dot{V}_{MO_2}$ of 7.5 times resting (10.3 ml O_2 min^{-1} kg^{-1} muscle) and an average muscle desaturation rate of 6.8 % min^{-1}, very similar to the average muscle desaturation rate of 7.3 % min^{-1} observed by Guyton et al. (1995).

Unlike blood oxygen, nearly all (94 %) the muscle oxygen was consumed to optimize the ADL (Table 4; Fig. 8). Guyton et al. (1995) did not observe desaturation of oxymyoglobin greater than 70 % on the surface of the latissimus dorsi muscle of Weddell seals during short dives. Whether this is representative of all the muscles, especially deeper regions of active locomotory muscles, is uncertain. Despite the nearly complete desaturation of oxymyoglobin in our model, it contributed, on average, only 51 % of the total oxygen consumed by the muscle for $\dot{V}_{MO_2}$ values ranging from 3 to 16 times resting; the remainder came from blood oxygen stores. By adjusting the muscle $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio within narrow limits (Figs 6, 7), blood and myoglobin oxygen stores contribute equally to $\dot{V}_{MO_2}$ to maximize ADL.

The results of our model agree with Guyton et al. (1995), who concluded that convective oxygen transport to muscle must be maintained to resolve any discrepancy between its metabolic rate and the measured oxymyoglobin desaturation rate. If half the muscle mass were engaged in swimming, they estimated that muscle blood flow at the beginning of a dive would have to be 41.7 ml min^{-1} kg^{-1} to provide the additional oxygen required by completely aerobic locomotory muscle. Furthermore, they concluded that muscle blood flow would have to double by the end of the dive if the same rate of muscle oxymyoglobin desaturation was to be maintained as Ca_{O_2} decreased. Our model predicts a total muscle blood flow of 28.2 ml min^{-1} kg^{-1} that remains constant throughout the dive. Rather than increasing blood flow, the rate of muscle O_2 desaturation increases gradually so that the contribution of oxymyoglobin to $\dot{V}_{MO_2}$ increases as Ca_{O_2} decreases (Fig. 11). Although not included in this model, seals may experience a gradual increase in $\dot{V}b$ during an anticipatory release of the dive response (i.e. 'anticipatory tachycardia') during ascent (Thompson and Fedak, 1993; Butler and Jones, 1997). Whether these increases in $\dot{V}b$ and muscle $\dot{Q}_{O_2}$ would prevent the gradual increase in muscle O_2 desaturation rate predicted in the model is uncertain.

D*MO2*

D_{MO_2} is as complex parameter that encompasses many variables not incorporated in the model. Some of these variables include red blood cell spacing, O_2 off-loading kinetics from Hb, muscle capillarity, diffusion distances and myoglobin-facilitated diffusion (Hogan et al., 1993; Kurdak et al., 1996). To estimate D_{MO_2}, we assumed that any resistance to oxygen diffusion was accounted for by setting the maximum E_{BO_2} to 0.8, a value that has been measured in mammals during hypoxic hypoxia with and without ischemia (Lautt and Graham, 1977; Cain, 1978; Cole, 1983; King et al., 1987; Samsel and Schumacker, 1994; Torrance and Wittnich, 1994). The maximum D_{MO_2} (0.034 ml O_2 100 g^{-1} muscle min^{-1} $mmHg^{-1}$) in this model occurred at a muscle $\dot{V}_{O_2}$ of 11–16 times resting and a muscle blood flow of 4.7 ml 100 g^{-1} muscle min^{-1} (i.e. 94 % of resting levels). Kurdak et al. (1996) examined the effect of moderate ischemia (i.e. 50 % of normal exercise hyperemia) in the isolated dog gastrocnemius muscle during isometric contractions. D_{MO_2} at peak $\dot{V}_{MO_2}$ was 0.16 ml O_2 100 g^{-1} muscle min^{-1} $mmHg^{-1}$, which was five times the maximum D_{MO_2} calculated in this model. However, we modeled D_{MO_2} at much less than $\dot{V}_{O_2max}$, and $\dot{Q}_M$ was also 11 times higher in the dog muscle preparation. In our analysis, D_{MO_2} decreased in proportion to blood flow without a further

reduction in muscle venous or mean capillary P_{O_2} because the $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio was generally less than 1, and muscle $E_{B O_2}$ was maximized throughout dives at all levels of $\dot{V}$b and $\dot{V}_{M O_2}$. As a result, convective oxygen transport became the major determinant of $D_{M O_2}$.

Variability in f_H *and* $\dot{V}_{O_2}$ *as a function of dive duration*

Previous studies have shown a high degree of variability in f_H and $\dot{V}_{O_2}$ for Weddell seals during voluntary dives lasting less than 25 min (Kooyman and Campbell, 1972; Hill et al., 1987; Castellini et al., 1992). Plots of either f_H or $\dot{V}_{O_2}$ as a function of dive duration show points that are widely scattered below a maximum boundary that decreases curvilinearly with dive duration and above a minimum boundary that is independent of dive duration. For f_H, the maximum boundary intersects the *y*-axis at approximately 50–60 beats min^{-1}, with the minimum boundary at 20–30 beats min^{-1}, although even lower values of f_H sometimes occur (Kooyman and Campbell, 1972; Hill et al., 1987). For $\dot{V}_{O_2}$, the maximum boundary intersects the *y*-axis at approximately 9 ml O_2 min^{-1} kg^{-1}, with the minimum boundary at 2 ml O_2 min^{-1} kg^{-1} (Castellini et al., 1992). Lines representing the maximum and minimum boundaries for either f_H or $\dot{V}_{O_2}$ intersect at a dive duration of approximately 30 min, a value similar to the estimated ADL under resting conditions in our model. Variability in f_H and $\dot{V}_{O_2}$ as a function of dive duration (i.e. the scatter of points between the maximum and minimum boundaries) probably results when, for behavioral reasons, dives are terminated before oxygen stores are depleted.

If we consider only the maximum f_H or $\dot{V}_{O_2}$ (i.e. just those points forming the upper boundary) as a function of dive duration, there is a clear inverse relationship; that is, f_H and $\dot{V}_{O_2}$ decrease with dive duration (Kooyman and Campbell, 1972; Hill et al., 1987; Castellini et al., 1992). Using only values along the maximum boundaries, a plot of f_H as a function of $\dot{V}_{O_2}$ for voluntary dives up to 25 min long would look similar to Fig. 4; that is, there is a positive correlation between f_H and $\dot{V}_{O_2}$. To confirm this, we need simultaneous data for f_H and $\dot{V}_{O_2}$ for dives of different duration. Unfortunately, simultaneous measurements of these two variables have not been made for any marine mammal during voluntary dives in the wild. However, data for f_H and dive duration are available for a variety of birds and mammals (for a review, see Butler and Jones, 1997, their Fig. 24; see also Fedak et al., 1988; Thompson and Fedak, 1993; Andrews et al., 1997; Ponganis et al., 1997a). As with Weddell seals, an inverse relationship between f_H and aerobic dive duration is apparent for northern elephant seals (*Mirounga angustirostris*), grey seals, harbor seals, California sea lions (*Zalophus californianus*), muskrats (*Ondatra zibethicus*), tufted ducks (*Aythya fuligula*) and emperor penguins (*Aptenodytes forsteri*). Assuming that aerobic dive duration is inversely proportional to $\dot{V}_{O_2}$ (Castellini et al., 1992; Reed et al., 1994; Butler and Jones, 1997), then f_H should be positively correlated with $\dot{V}_{O_2}$ for these species. This conclusion is supported by the observation that the lowest f_H in free-ranging grey seals occurs when they rest on the bottom, whereas bradycardia is less pronounced during traveling dives (Fedak and Thompson, 1993). Resting dives also happen to be the longest for grey seals.

Simultaneous measurements of f_H and $\dot{V}_{O_2}$ have been made for harbor seals, grey seals and California sea lions swimming in a water flume. Submerged f_H increases with $\dot{V}_{O_2}$ for California sea lions (Williams et al., 1991; Ponganis et al., 1990, 1991) and, in some cases (Ponganis et al., 1990), harbor seals. However, other studies have found that the f_H of grey seals and harbor seals is poorly correlated with $\dot{V}_{O_2}$ (or speed) during submerged swimming (Williams et al., 1991; Fedak et al., 1988). In both cases, the animals decrease submergence duration at higher work loads so that the weighted average f_H (weighted average of submerged and surface f_H) increases with $\dot{V}_{O_2}$. The weighted average f_H increases much more dramatically than the submerged f_H because the former includes the tachycardia between dives (Williams et al., 1991; Fedak et al., 1988; Butler et al., 1992). However, the weighted average f_H has little to do with maximizing the ADL. Rather than asking why animals perform differently, perhaps we should ask whether the cardiovascular adjustments exhibited by animals during shallow, submerged swimming in a water flume are representative of voluntary dives in the wild. In most cases, dive durations for animals in a water flume are very short (e.g. less than 30 s) and do not approach the animals' ADL. Under these circumstances, there is less need to manage oxygen stores efficiently, and this may alter the relationship between f_H and $\dot{V}_{O_2}$. However, this is not the case for Weddell seals making 15–20 min foraging dives with limited access to breathing holes (Castellini et al., 1988). As a result, Weddell seals and other marine mammals such as elephant seals that make deep, prolonged dives that routinely approach their ADL (Le Boeuf et al., 1988; Hindell et al., 1992) may optimize the use of blood and muscle oxygen stores by adjusting $\dot{V}$b and muscle $\dot{Q}_{O_2}$ according to the level of exertion.

Tolerating anaerobiosis to extend dive duration

We have assumed that Weddell seals make cardiovascular adjustments that maximize aerobic dive duration. Another strategy to extend dive duration is to tolerate limited anaerobic muscle metabolism resulting in the formation of lactic acid. This means that $\dot{V}$b and the muscle's $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio might be less than required to maintain aerobic metabolism throughout the dive. Anaerobiosis will disrupt physiological homeostasis and possibly increase recovery time at the surface or require that subsequent dives be completely aerobic to metabolize the modest lactate load. However, by definition, most dives within the ADL show no post-dive increase in blood lactic acid concentration (Castellini et al., 1988). In addition, most voluntary dives made by Weddell seals show a clear, minimum f_H of 15–25 beats min^{-1} (Kooyman and Campbell, 1972; Hill et al., 1987), which may represent a lower threshold for a $\dot{Q}_{O_2}$ sufficient to maintain aerobic metabolism in all the peripheral tissues including the splanchnic organs and kidneys.

Nevertheless, tolerating limited anaerobiosis may be a physiological strategy that some species use to extend dive duration.

Conclusions

This model describes cardiovascular changes in Weddell seals that could theoretically maximize the ADL for different levels of muscular exertion up to a $\dot{V}_{MO_2}$ of 16 times resting levels (whole-body $\dot{V}_{O_2}$=9.2 ml O_2 min^{-1} kg^{-1}). Like all numerical models of biological systems, many aspects of the complex oxygen transport process have not been included, so that this is a simplified view to aid understanding. Despite its limitations, the model can explain existing data from a variety of experiments and provides new insight into the potential role of the dive response in optimizing convective oxygen transport to peripheral tissues during aerobic dives. This is achieved by adjusting $\dot{V}$b and muscle $\dot{Q}_{O_2}$ according to the level of $\dot{V}_{MO_2}$. At higher levels of $\dot{V}_{MO_2}$, $\dot{V}$b and muscle perfusion must increase to maintain an appropriate $\dot{Q}_{O_2}/\dot{V}_{O_2}$ ratio so that available blood and muscle oxygen stores are depleted at the same time. Although the dive response does not sequester blood oxygen exclusively for brain and heart metabolism during aerobic dives, as it does during forced submersion, a reduction in $\dot{V}$b and muscle perfusion below resting levels is necessary to maximize the ADL over most of the normal range of diving $\dot{V}_{O_2}$ (approximately 2–9 ml O_2 min^{-1} kg^{-1}). Despite the reduction in $\dot{V}$b, convective oxygen transport is adequate to maintain aerobic metabolism and normal function in the brain, heart, splanchnic organs, kidneys and other peripheral tissues. As a result, physiological homeostasis is maintained throughout the dive. Contrary to earlier beliefs, the dive response does not create a conflict between convective oxygen transport and aerobic muscle metabolism in diving marine mammals, although it may limit $\dot{V}_{O_2max}$. On the basis of results from this model, cardiovascular adjustments during the dive response that optimize the use of blood and muscle oxygen stores for a particular level of power output will maximize aerobic dive duration.

List of symbols

C_{aO_2}	arterial blood oxygen concentration (ml O_2 l^{-1} blood)
C_{BvO_2}	cerebral venous blood oxygen concentration (ml O_2 l^{-1} blood)
C_{HvO_2}	coronary venous blood oxygen concentration (ml O_2 l^{-1} blood)
C_{MvO_2}	skeletal muscle venous blood oxygen concentration (ml O_2 l^{-1} blood)
C_{SRCvO_2}	splanchnic, renal, cutaneous and other peripheral tissue venous blood oxygen concentration (ml O_2 l^{-1} blood)
C_{vO_2}	venous blood oxygen concentration (ml O_2 l^{-1} blood)
$C_{\bar{v}O_2}$	mixed venous blood oxygen concentration (ml O_2 l^{-1} blood)
D_{MO_2}	muscle oxygen diffusive conductance (ml O_2 100 g^{-1} muscle min^{-1} $mmHg^{-1}$)
E_{BO_2}	extraction coefficient of oxygen from blood [$(Ca_{O_2}-Cv_{O_2})/Ca_{O_2}$]
f_H	heart frequency (beats min^{-1})
GFR	glomerular filtration rate (ml min^{-1} kg^{-1})
Hb	hemoglobin
Pa_{O_2}	arterial blood oxygen partial pressure (mmHg)
$P\bar{c}_{O_2}$	mean capillary oxygen partial pressure (mmHg)
$Pmito_{O_2}$	mitochondrial oxygen partial pressure (mmHg)
P_{MvO_2}	skeletal muscle venous blood oxygen partial pressure (mmHg)
P_{O_2}	oxygen partial pressure (mmHg)
Pv_{O_2}	venous blood oxygen partial pressure (mmHg)
$P\bar{v}_{O_2}$	mixed venous blood oxygen partial pressure (mmHg)
$\dot{Q}$	blood flow rate (l min^{-1})
$\dot{Q}_B$	brain blood flow (l min^{-1})
$\dot{Q}_H$	heart blood flow (l min^{-1})
$\dot{Q}_M$	skeletal muscle blood flow (l min^{-1})
$\dot{Q}_{O_2}$	convective oxygen transport in the blood (ml O_2 min^{-1})
$\dot{Q}_{SRC}$	splanchnic, renal, cutaneous and other peripheral tissue blood flow (l min^{-1})
Sa_{O_2}	arterial blood oxygen saturation (%)
S_{MvO_2}	skeletal muscle venous blood oxygen saturation (%)
Sv_{O_2}	venous blood oxygen saturation (%)
$S\bar{v}_{O_2}$	mixed venous blood oxygen saturation (%)
$\dot{V}$b	cardiac output (l min^{-1})
$\dot{V}_{BO_2}$	brain oxygen consumption rate (ml O_2 min^{-1})
$\dot{V}_{HO_2}$	heart oxygen consumption rate (ml O_2 min^{-1})
$\dot{V}_{MO_2}$	skeletal muscle oxygen consumption rate (ml O_2 min^{-1})
$\dot{V}_{O_2}$	rate of oxygen consumption (ml O_2 min^{-1})
$\dot{V}_{O_2max}$	maximum rate of oxygen consumption (ml O_2 min^{-1})
V_S	stroke volume (l)
$\dot{V}_{SRCO_2}$	splanchnic, renal, cutaneous and other peripheral tissue oxygen consumption rate (ml O_2 min^{-1})
β_{BO_2}	capacitance coefficient of oxygen in blood (ml O_2 l^{-1} blood)

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