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The effect of O_2 and CO_2 on the dive behavior and heart rate of lesser scaup ducks (*Aythya affinis*): quantification of the critical Pa_{O_2} that initiates a diving bradycardia

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Abstract

Lesser scaup ducks were trained to dive for short and long durations following exposure to various gas concentrations to determine the influence of oxygen (O_2) and carbon dioxide (CO_2) on diving behavior and heart rate. Compared with normoxia, hyperoxia (50% O_2) significantly increased the duration of long dives, whereas severe hypoxia (9% O_2) significantly decreased the duration of both short and long dives. Hypercapnia (5% CO_2) had no effect on dive duration. Surface intervals were not significantly altered by the oxygen treatments, but significantly increased following CO_2 exposure. Heart rate during diving was unaffected by hyperoxia and hypercapnia, but gradually declined in long dives after severe hypoxia. Thus, our results suggest that during the majority of dives, O_2 and CO_2 levels in lesser scaup ducks are managed through changes in diving behavior without any major cardiovascular adjustments, but below a threshold Pa_{O_2}, a bradycardia is evoked to conserve the remaining oxygen for hypoxia sensitive tissues. A model of oxygen store utilization during voluntary diving was developed to estimate the critical Pa_{O_2} below which bradycardia is initiated (≈26 mmHg) and predicted that this critical Pa_{O_2} would be reached 19 s into a dive after exposure to severe hypoxia, which corresponded exactly with the time of initiation of bradycardia in the severe hypoxia trials.

Keywords: Dive behaviour; Heart rate; *Aythya affinis*; Peripheral chemoreceptors; Oxygen, Carbondioxide

1. Introduction

Birds that forage underwater are subject to various physiological constraints imposed by the environment that ultimately shape their diving behavior. Most diving birds forage in bouts, performing a series of dives in quick succession interspersed with brief periods of time at the surface, and the majority of these dives are assumed to be metabolically aerobic (Butler and Jones, 1997). The aerobic dive limit is the duration that an animal can sustain aerobic metabolism without resorting to anaerobiosis (Kooyman et al., 1983) and can be

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crudely calculated from the amount of oxygen stored in the body and the rate at which the store is utilized. This is a crude calculation because anaerobiosis, of course, begins long before all oxygen stores are depleted. Aerobic dives are favorable because lengthy surface intervals spent removing the by-products of anaerobic metabolism can be avoided. This allows the animal to maintain a high dive:pause (D:P) ratio such that a series of aerobic dives can occur in rapid succession, maximizing underwater time.

Oxygen is, therefore, crucial during aerobic diving yet its role in determining the time allocation during a dive cycle (time spent underwater traveling and foraging plus the subsequent surface interval) remains unclear. Increasing the concentration of oxygen in the inspired air increased dive duration of redhead ducks (Furilla and Jones, 1986), but had no effect on tufted ducks (Butler and Stephenson, 1988). However, dive duration decreased in both of these species when exposed to hypoxic gas mixtures. The effect of oxygen on surface interval duration was equally variable. Halsey et al. (2003a) found that upon surfacing, tufted ducks fully reloaded their oxygen stores before commencing another dive, indicating that oxygen reloading influenced surface interval duration. If exposed to hyperoxic gas mixtures, surface interval would be expected to decrease. This was the case in double-crested cormorants (Enstipp et al., 2001), but tufted ducks showed no change in surface interval duration when exposed to hyperoxia. Moreover, when exposed to hypoxic gases, surface interval increased in double-crested cormorants while it was reduced in tufted ducks. While the conflicting findings of these studies question the role of oxygen alone in shaping dive behavior, theoretical models assume that the oxygen level within the body is the proximate controller of dive behavior (Kramer, 1988; Houston and Carbone, 1992).

Throughout a dive CO_2 will accumulate in the blood, lung and tissues potentially affecting dive behavior. In response to hypercapnia, dive duration and surface interval duration decreased in tufted ducks while double-crested cormorants showed no change in dive duration, but surface interval increased (Butler and Stephenson, 1988; Enstipp et al., 2001). Boutilier et al. (2001) found in gray seals that oxygen stores were fully readjusted after three or four breaths at the surface, but three to four more breaths were required to eliminate CO_2 from the body before the next dive commenced. On the other hand, Halsey et al. (2003a) found that accumulated CO_2 was removed by the time O_2 stores were fully replenished. Nonetheless, Halsey et al. (2003a) suggested that CO_2 was more important in terminating dive duration than oxygen since tufted ducks exchanged more CO_2 than oxygen in the last few breaths before the first dive in a bout. Hence, breathing high CO_2 before a dive would be expected to significantly reduce dive duration, yet it does not in cormorants (Enstipp et al., 2001). These studies indicate that although both oxygen and CO_2 affect dive behavior, there is no universal pattern to the role each plays in determining the behavioral components of a dive cycle.

Changes in oxygen and CO_2 levels in arterial blood are monitored by peripheral chemoreceptors and the discharge frequency of the receptors increases as Pa_{O_2} falls and Pa_{CO_2} increases (Bamford and Jones, 1974). During forced diving, an increased rate of chemoreceptor firing stimulates the parasympathetic innervation of the heart, inducing a decline in heart rate. Diving bradycardia is associated with a redistribution of blood flow in the body, presumably reducing blood flow to tissues that are inactive or that can rely on anaerobic metabolism so that oxygen can be conserved for the tissues that cannot withstand anoxia. Cardiovascular studies on freely diving tufted ducks indicate that during dives of short duration, there is no bradycardia and that heart rate remains above resting levels (Butler and Woakes, 1979). This indicates that oxygen stores in the body are likely sufficient to maintain aerobic metabolism throughout the dive without the need for oxygen-conserving cardiovascular adjustments. However, there are situations in the wild where animals may have to remain submerged for longer periods, such as to avoid predators or if trapped under ice. In these types of situations, a profound bradycardia can be evoked as shown by Stephenson et al. (1986) when tufted ducks were trained to dive for prolonged periods or prevented from surfacing towards the end of a dive.

Most of the physiological studies on diving ducks conducted in a laboratory setting consist of short duration dives, similar to those seen in the wild when the animals are diving vertically for their food (Butler and Woakes, 1979; Parkes et al., 2002; Halsey et al., 2003a, 2003b). The energetic requirements during a vertical dive are greatest during the descent phase to overcome drag and buoyancy and decrease when the animal forages at the bottom (Stephenson, 1994). At

the end of the foraging period, the animal passively floats to the surface. Consequently, the metabolic costs of diving decrease as oxygen stores are being drawn down. Pushing animals to their physiological limits not only by increasing the energetic demands of the entire dive, but also by increasing dive duration may lead to insights into the physiological mechanisms underlying diving behavior.

The present study was designed to determine the relative influence of oxygen and CO_2 on the behavioral and cardiovascular adjustments during voluntary dives in lesser scaup ducks. In particular, we were interested in how these animals allocate time during the dive cycle in response to various levels of inspired oxygen and CO_2 during short and long horizontal dives that are potentially more energetically costly than vertical dives. Heart rate, measured by telemetry, was used as an indication of the role of peripheral chemoreceptors in regulating the cardiovascular system during short and long dives as well as in dives extended in duration by temporarily trapping the animals underwater. Finally, we developed a simple model of oxygen store utilization throughout a voluntary dive to estimate the Pa_{O_2} at which a bradycardia is evoked.

2. Materials and methods

Three adult female and three adult male lesser scaup ducks, *Aythya affinis*, with a mean mass of 660 ± 90 g (mean ± S.D.; range, 582–738 g) were used for all experiments. The animals were raised from eggs at the South Campus Animal Care Facility at the University of British Columbia (UBC) and subsequently housed there on a shallow diving tank for the duration of the study. All procedures in these experiments were approved by the UBC Animal Care Committee.

2.1. Dive tank setup and training protocol

Experiments were conducted using a shallow, flow-through freshwater tank (9 m long, 0.60 m wide, 0.55 m high; Fig. 1) located in a sheltered outdoor room where the animals were exposed to daily and seasonal fluctuations in daylight and ambient temperature. The surface of the tank was covered with wire mesh so that the birds were restricted to surfacing into an open-water area at one end of the tank. The open-water section and adjacent dry land were enclosed by a wire cage forming a holding area (1.9 m length, 0.70 m wide, 0.63 m high). To encourage diving activity, a constant quantity of grain was added to discrete areas in the tank on a daily basis. Consequently, the birds were required to dive horizontally to and from food. A shallow tank was used in order to eliminate any affect that depth may have on heart rate during diving (Enstipp et al., 2001). To obtain dive durations similar to those seen in the wild ('short dives', approximately 18–20 s in duration), the food was positioned about 3.6 m from the holding area (position F_S in Fig. 1). Following completion of the short dive trials, the birds were trained to extend their dive durations by gradually moving the food until they were diving to the end of the tank, 9 m from the holding area ('long dives', approximately 30 s in duration; position F_L in Fig. 1).

Trap dives consisted of temporarily denying access back to the surface during the return journey of a voluntary dive. For these trials, a weighted piece of plywood was suspended above the first corner of the tank (Fig. 1). The behavior of the animals was monitored on a TV screen and the trap door could be lowered and raised remotely to block the passageway back to the dive enclosure.

2.2. Instrumentation

Heart rate data were collected by radio telemetry. Each animal was implanted with a small FM radio transmitter (T4D Medical Telemetry Implant, Konigsberg Instruments Inc., Pasadena, CA, USA) while under isoflurane anesthesia. A small incision was made in the lower abdomen below the rib cage, and the body of the transmitter was positioned subcutaneously and sutured in place and the two electrocardiogram (ECG) leads were tunneled under the ribcage for placement on either side of the heart. Baytril antibiotic (2.5–5 mg/kg) was administered intramuscularly twice daily for three days and by that time the birds were behaving and eating normally and were, therefore, determined to have recovered from surgery. Heart rate recordings were not made until one week post-surgery.

Each implanted transmitter was factory set to a specific radio frequency. The FM ECG signal from the birds was picked up by an antenna connected to a

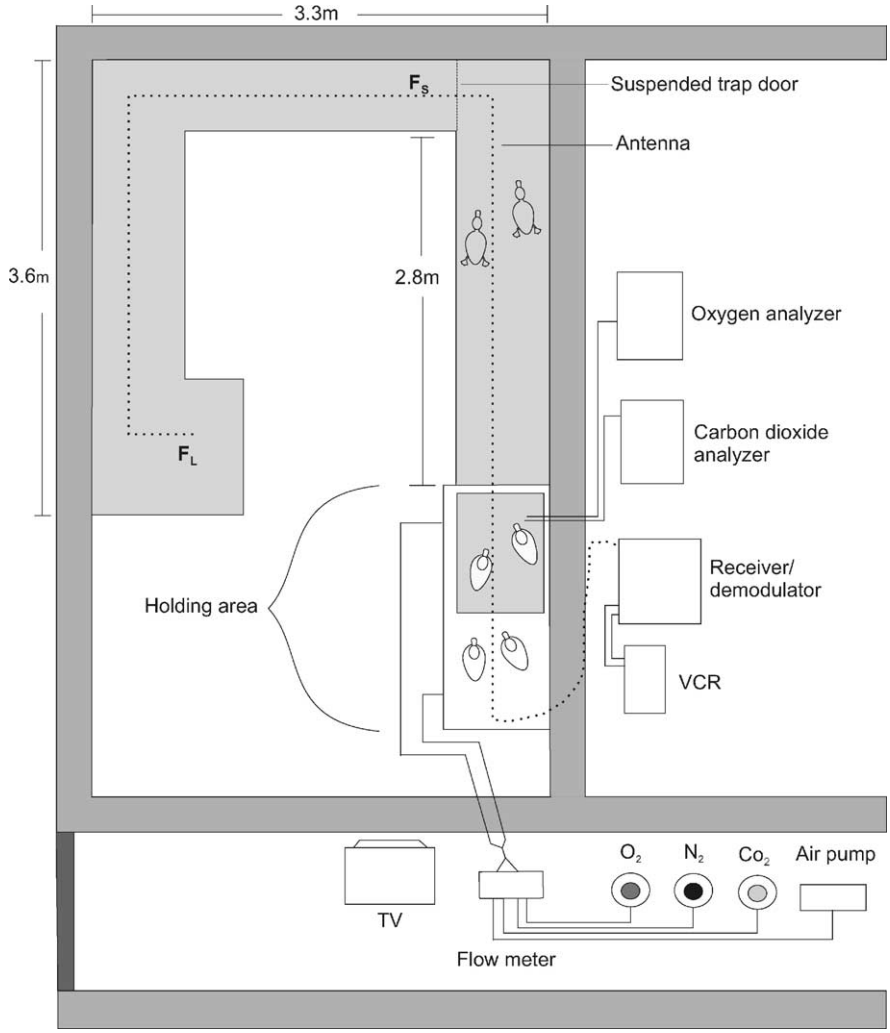

Fig. 1. Aerial view and dimensions of dive tank and apparatus. The shaded region in the holding area represents open water where the animals were diving and surfacing. The entire surface of the tank was covered with a wire mesh cover, which is not illustrated. F_S indicates location of food for short dives and F_L indicates position of food for long dives.

two-channel telemetry receiver and demodulator (TR8-2-2/TD13, Konigsberg Instruments Inc., Pasadena, CA, USA) that allowed simultaneous tuning to two separate frequencies. The ECG signal was recorded on the audio portion of VHS tapes while dive behavior was recorded on the video portion of the tape so that diving behavior could be matched with heart rate. All of the equipment was situated in a room adjacent to the tank to avoid disturbances to the animals during trials.

2.3. Experiments

Heart rate was recorded during short, long, and trap dives with the animals breathing various concentrations of gases before and after a dive. Experiments were run from November 2001 through to May 2003 during non-molting periods and water temperature was kept between 8 and 12 °C.

Transparent vapor-proof polyethylene plastic was used to seal the holding area and medical grade gases

(100% N_2, O_2, or CO_2) were introduced at opposite ends of the box until the desired gas concentration was achieved. Gas samples from the enclosure were passed through an O_2 analyzer (S-3A/1, Applied Electrochemistry, Ametek Inc., Pittsburgh, PA, USA) and a CO_2 analyzer (CD-3A, Applied Electrochemistry, Ametek Inc., Pittsburgh, PA, USA). Two fans located at opposite ends of the holding area ran continuously ensuring mixing and even distribution of gases. Throughout the duration of a trial, gases were sampled directly above where the birds were diving and surfacing to ensure that the gas breathed was of the desired concentration. Compressed air was continuously passed into the holding area when trials with other gases were not being run serving as a control and allowing the birds to acclimate to any noise generated by the gas flow.

All birds received the following gas treatments on separate days, in randomized order: (i) normoxia (20.90% O_2, 0.03% CO_2); (ii) hyperoxia (>50% O_2, 0.03% CO_2); (iii) moderate hypoxia (long dives only; 13% O_2, 0.03% CO_2); (iv) severe hypoxia (9% O_2, 0.03% CO_2) and (v) hypercapnia (20.90% O_2, 5% CO_2). These levels of gases were selected because they have been shown to affect diving heart rate in tufted ducks (Butler and Stephenson, 1988). At least 5 min were allowed before recordings were initiated to ensure that the animals' blood gases had equilibrated with the gases in the dive box. Experiments lasted between 4 and 6 h and were conducted during daylight hours.

Trap dives were performed during the normoxia and hyperoxia gas trials. When a bird swam past the trap to the foraging site, the trap door was lowered and kept in place for 6–8 s and subsequently lifted to allow the bird to return to the enclosure to surface. Because the response to trap dives is subject to habituation (Stephenson, personal communication) each bird was trapped only twice for both short and long dives.

2.4. Data analysis and statistics

2.4.1. Dive behavior

Video tapes of diving behavior were reviewed for each gas treatment and all dives to the foraging site were analyzed for the following behavioral components: total dive time (t_d), travel time to the food (t_{tt}), foraging time (t_f), and travel time from the food (t_{tf}). A total of 930 short dives and 476 long dives were analyzed. The behavioral dive components were averaged for each individual animal and these values were compiled to generate a grand mean ± S.E.M. (N = 6). One animal did not complete any dives to the food in the long dive protocol during 9% hypoxia exposure, and therefore, all behavioral components in this treatment involve five animals.

Surface intervals (t_s) were analyzed by determining the time spent at the surface between two consecutive foraging dives for all gas treatments. All surface intervals were compiled for each animal in each gas treatment and bout-ending analysis (Slater and Lester, 1982) was used to determine the cutoff point for a dive bout for each individual animal. Surface intervals greater than this value were excluded from analysis while shorter intervals were averaged to generate individual surface interval means. These animal means were then compiled to generate grand means ± S.E.M. (N = 6) for all short and long dives with all gas treatments.

Dive:pause (D:P) ratios were calculated by dividing individual dive duration by subsequent surface interval duration for each bird in all treatments. Proportion of the dive cycle spent foraging (P_{forage}) was calculated for individual birds using the equation:

$$P_{forage} = \frac{t_f}{t_d + t_s}$$

Swim speed was calculated by dividing dive distance (3.6 m for short dives versus 9 m for long dives) by travel time when swimming to and from the food.

Dive duration and surface interval duration means for individual animals were plotted to illustrate trends between the two variables. Data for normoxia treatments were normally distributed whereas data for the remaining three treatments were not. Therefore, statistical analysis was not performed on the latter data, but trend-lines were added for illustrative purposes.

One-way repeated measures analysis of variance (ANOVA) was used to test between gas treatments within each behavioral component (dive time, travel time, etc.) for short and long dives, with Student–Newman–Keuls multiple comparison as a post-hoc test. When single comparisons were made, as in differences between short and long dives within a gas treatment, a Student's paired t-test was used. Significance was accepted at $P < 0.05$.

2.4.2. Heart rate

Video tapes of dive behavior and heart rate were reviewed and a total of six foraging dives were selected for each animal during exposure to each gas treatment during short and long dives. Dives were only selected form the third, fourth or fifth dives within a diving bout as pre-dive heart rate is most stable during this phase (Furilla and Jones, 1987). The electrocardiogram (ECG) from the selected dive was extracted from the video by running the audio portion of the video into Labtech Notebook Software (Version 9.0, Laboratory Technologies Corporation, Wilmington, MA, USA). Instantaneous heart rates were computed by calculating the R–R intervals of the ECG using Acknowledge Software (Version 3.01, BIOPAC Systems Inc., Santa Barbara, CA, USA), which were converted into beats min^{-1}.

Heart rate profiles were generated for short and long dives by analyzing the heart rate 10 s before the dive (pre-dive), throughout the dive, and 12 s following the dive (post-dive). Instantaneous heart rate was then averaged over 2 s time bins. Short dives between 20 and 22 s were selected and standardized to 20 s by removing the penultimate time bin of the longer dives. The same procedure was used to standardize the 30–32 s long dives to 30 s. Long dive durations were shorter during both hypoxia treatments so they were standardized to a mean dive time of 26 s for mild hypoxia (13%) and 24 s for severe hypoxia (9%). For each bird, a total of six dives per gas treatment were analyzed to generate an average response for that animal. The animal means were then compiled to generate grand means ± S.E.M. ($N = 6$).

During trap dives, markers were made on the ECG recording when the animal apparently became aware of the trap door (indicated by a change in swimming behavior) and when the trap door was lifted, because previous experiments have shown that these behaviors coincide with a change in heart rate (Stephenson et al., 1986). Heart rate profiles were then generated for trap dives. Short dives ($N = 6$) were standardized to 20 s, including a 6 s trap and long dives ($N = 4$) were standardized to 36 s, including an 8 s trap. Each animal was trapped twice in normoxia and twice in hyperoxia.

To generate heart rate averages for the various dive components, R–R intervals were averaged over the duration of the particular dive component. For trap dives, heart rate was also averaged over the trapping period, which started at the point the animal was aware of the trap door to the point when the trap door was lifted. Change in heart rate during the trapping period was determined by calculating heart rate during the last 2 s before the animal was aware of the trap door and subtracting the heart rate during the last 2 s before the trap door was lifted.

To obtain resting heart rate, video tapes were reviewed for periods, at least 5 min before or after a diving bout, when the animals were quietly floating on the water surface. Heart rate was averaged over the resting period for each animal in each gas treatment and then compiled as grand means ± S.E.M. ($N = 6$).

Comparisons between gas treatments of individual 2 s means of heart rate profiles (short and long dives) and of the dive component averages (pre-dive, dive and post-dive) were made using one-way repeated measures analysis of variance (ANOVA) with Student–Newman–Keuls multiple comparison tests. Comparison of heart rate averages between short and long dives within each gas treatment and heart rate between normoxia and hyperoxia for traps after short and long dives were made using a Student's paired *t*-test. Significance was accepted at $P < 0.05$.

2.5. Model of oxygen store utilization during a voluntary dive

A model of oxygen store utilization from the lung and blood during a voluntary dive was developed to determine the Pa_{O_2} level that corresponds with the onset of diving bradycardia. The starting values for the model were generated as follows: the estimated oxygen storage capacity in the lung (CL_{O_2}, in ml O_2 kg^{-1}) of dive trained lesser scaup ducks was taken from Stephenson et al. (1989) and the corresponding partial pressure of oxygen in the lung (PL_{O_2}) was set at 105 mmHg to account for hyperventilation before the dive. Oxygen in the lung was assumed to be accessible for gas exchange during the dive (Boggs, 2002). Arterial P_{O_2} (Pa_{O_2}) values were calculated as PL_{O_2}-5 mmHg to allow for limitations of gas exchange. The saturation of hemoglobin at each Pa_{O_2} was obtained from the oxygen–hemoglobin dissociation curve (Scheipers et al., 1975) and this value was multiplied by the oxygen carrying capacity of the blood (20.8 ml O_2 100 ml^{-1}

blood; Stephenson et al., 1989) to yield the oxygen content in arterial blood (Ca_{O_2}). To calculate the arterial oxygen store, Ca_{O_2} was multiplied by arterial blood volume (Stephenson et al., 1989). Venous oxygen content (Cv_{O_2}) was calculated as 70% of Ca_{O_2} under all conditions (Stephenson et al., 1989). This value (Cv_{O_2}) was multiplied by venous blood volume to generate the venous oxygen store. The small oxygen store in the muscle (5% of total oxygen store in normoxia) was not included in the model.

To simulate oxygen utilization during a dive, an iterative process was performed whereby a fixed volume of oxygen was removed from the lung store per unit time (t), corresponding to one of the diving metabolic rates reported in the literature for lesser scaup ducks ($\dot{V}_{O_2}$ = 0.53, 0.93, or 1.46 ml kg^{-1} s^{-1}; taken from Bevan et al. (1992), Woakes and Butler (1983), Stephenson (1994), respectively). The resulting changes in PL_{O_2}, Pa_{O_2}, Ca_{O_2} and Cv_{O_2} were calculated. PL_{O_2} was obtained by multiplying the starting PL_{O_2} by the quotient of CL_{O_2} divided by the starting CL_{O_2}.

Cardiac output ($\dot{Q}$), was calculated using the Fick equation:

$$\dot{Q} = \frac{\dot{V}_{O_2}}{Ca_{O_2} - Cv_{O_2}}$$

where ($Ca_{O_2} - Cv_{O_2}$) and $\dot{V}_{O_2}$ are values from the literature, as explained above, and were held constant through the dive.

The arterial oxygen store was adjusted to account for the volume of blood passing through the tissues per unit time (t) by multiplying Ca_{O_2} by $\dot{Q}(t)$. The volume of blood not passing through the tissues per unit time was, therefore, calculated as total arterial blood volume $-\dot{Q}(t)$. This value was then multiplied by the Ca_{O_2} at the end of the previous cycle of the iteration to give the arterial oxygen store not passing the tissues. These two values were added together to determine the total arterial oxygen store. The same procedure was used to adjust the venous oxygen store. The volumes of oxygen in the lung, arterial and venous stores were added to yield the corrected total oxygen store for each step in the iteration.

The amount of oxygen consumed at each step was calculated by subtracting total oxygen stores used in the current iteration from that at the end of the previous iteration. Dividing the amount of oxygen consumed by $\dot{V}_{O_2}$ gave the time period for that step of the iteration. These time values were summed to give dive time.

The above sequence of calculations was repeated until the venous oxygen content dropped to zero. At this point, the arterial oxygen content was insufficient to supply the metabolic needs of the tissues without some form of cardiovascular adjustment. There are two possible responses to this situation, either an elevation in $\dot{Q}$ or a switch to the oxygen-conserving response. In this study, the latter response was observed. Therefore, the calculated Pa_{O_2} at this point was used as a measure of the critical Pa_{O_2}; the Pa_{O_2} that necessitates the oxygen sparing diving response.

The model was run at three different metabolic rates and starting values for oxygen stores were determined for each level of oxygen used in these experiments (9%, 13%, 21%, and 50% oxygen). The total oxygen stores were plotted against dive time at each $\dot{V}_{O_2}$ to predict when the critical Pa_{O_2}, and thus the diving bradycardia, would be initiated during the dive. Only the model results obtained in normoxia and severe hypoxia using a $\dot{V}_{O_2}$ of 0.93 ml kg^{-1} s^{-1} are presented graphically as the time at which the critical Pa_{O_2} was reached under these conditions corresponded with the time in the dive that bradycardia occurred in our hypoxia treatments.

3. Results

3.1. The effect of breathing various gas mixtures on dive behavior

The relationship between dive time and surface interval during exposure to the various gas treatments is shown in Fig. 2. For each treatment, an increase in dive time resulted in an increase in surface interval with more dramatic increases occurring during hypercapnia and hypoxia (9%) exposure.

Compared with control (normoxia) conditions, the only gas treatment that had a significant effect on short dive duration was hypoxia (Table 1). The decreased dive duration during hypoxic exposures was due to a decrease in time spent swimming to and from the food source (Table 1). The animals also increased swimming speeds on the outgoing journey to food during hypercapnia treatments (Table 2), but this did not cause a reduction in overall dive duration. Although the animals adjusted their swim speed during

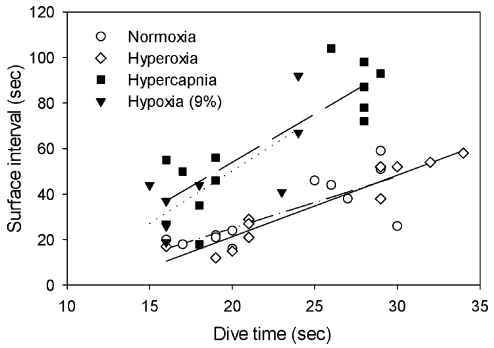

Fig. 2. Relationship between dive duration and surface interval duration during exposure to various gas mixtures. Trendlines are for normoxia (solid line) hyperoxia (dash and dot) hypercapnia (long dash line) and hypoxia (dotted line) treatments. Data points represent individual animal means during short and long dives. $N = 6$ for all gas treatments with the exception of long hypoxic dives where $N = 3$.

hypoxic and hypercapnic exposures, time spent at the foraging site was similar to that found in control animals in short dives. Swim speeds were higher on the return journey from the food during normoxia and hypoxia, but not during hyperoxia and hypercapnia (Table 2).

During short dives, hypercapnic exposure had the largest effect on surface interval duration, causing a 115% increase above control values. This increase in surface interval reduced the D:P ratio by 56%. Surface intervals during hypoxic exposures were longer than those in normoxia, although the differences were not significant. Together with the decreased dive duration, this caused a 56% reduction in the D:P ratio compared to the value found in control animals. The proportion of the dive cycle spent foraging (P_{forage}) was similar in all treatments.

Oxygen treatments had a significant effect on long dive duration whereas hypercapnia did not (Table 1). Breathing hyperoxic gas mixtures significantly increased long dive duration due to an increase in travel time as well as increased time spent at the foraging site. Hypoxia treatments significantly decreased dive duration, solely due to decreased foraging time. Foraging time during the mild hypoxia treatment (13%) was intermediate between the normoxia and severe hypoxia (9%) treatments. Swim speeds remained constant in all dives with the exception of those during hyperoxic exposure (Table 2).

Although hypercapnia had no significant effect on the behavioral components of long dives, surface

Table 1
Time allocation (s) of lesser scaup ducks during short and long dives following exposure to various gas mixtures

Behavioral component	Normoxia (21% O_2)	Hyperoxia (>50% O_2)	Hypoxia (13% O_2)	Hypoxia (9% O_2)	Hypercapnia (5% CO_2)
Short dives					
Dive duration	18.5 ± 0.7	19.7 ± 0.8	–	16.2 ± 0.4[a]	17.8 ± 0.5
Travel to food	7.5 ± 0.2	7.8 ± 0.4	–	6.7 ± 0.3[a]	6.7 ± 0.2[a]
Travel from food	5.8 ± 0.3	6.2 ± 0.3	–	5.0 ± 0.0[a]	6.0 ± 0.3
Foraging duration	5.2 ± 0.5	5.3 ± 0.7	–	4.3 ± 0.2	6.5 ± 0.5
Surface interval	20.2 ± 1.2	20.2 ± 2.8	–	32.8 ± 4.2	43.3 ± 5.9[a]
Dive:pause ratio	0.9 ± 0.1	1.1 ± 0.1	–	0.5 ± 0.1[a]	0.5 ± 0.1[a]
P_{forage}	0.13 ± 0.01	0.13 ± 0.02		0.09 ± 0.02	0.11 ± 0.04
Long dives					
Dive duration	27.7 ± 0.8[b]	30.7 ± 0.8[a,b]	26.0 ± 0.3[a]	24.2 ± 0.4[a,b]	27.8 ± 0.4[b]
Travel to food	11.7 ± 0.3[b]	12.7 ± 0.3[a,b]	12.0 ± 0.3	11.4 ± 0.2[b]	11.8 ± 0.3[b]
Travel from food	10.7 ± 0.2[b]	12.0 ± 0.5[a,b]	10.3 ± 0.2	10.4 ± 0.4[b]	11.0 ± 0.0[b]
Foraging duration	5.3 ± 0.3	6.3 ± 0.2[a]	3.8 ± 0.2[a]	2.2 ± 0.4[a,b]	5.0 ± 0.5
Surface interval	44.0 ± 4.6[b]	56.5 ± 6.3[b]	44.7 ± 6.7	66.7 ± 14.7	88.7 ± 5.0[a,b]
Dive:pause ratio	0.7 ± 0.1	0.6 ± 0.1[b]	0.6 ± 0.1	0.4 ± 0.1	0.3 ± 0.1[a]
P_{forage}	0.075 ± 0.005[b]	0.074 ± 0.004[b]	0.056 ± 0.005[a]	0.030 ± 0.002[a,b]	0.044 ± 0.005[a,b]

Values are grand means (s) ± S.E.M. ($N = 6$).

[a] Significantly different from normoxia value within corresponding short or long dive behavioral component (one-way RM ANOVA; $P < 0.05$).

[b] Significantly different from short dive value of the same gas mixture within corresponding behavioral component (paired t-test; $P < 0.05$).

Table 2
Calculated swim speeds ($m\,s^{-1}$) of lesser scaup ducks during short and long dives following exposure to various gas mixtures

Behavioral component	Normoxia (21% O_2)	Hyperoxia (>50% O_2)	Hypoxia (13% O_2)	Hypoxia (9% O_2)	Hypercapnia (5% CO_2)
Short dives					
Travel to food	0.48 ± 0.01	0.46 ± 0.02	–	0.55 ± 0.03^{a}	0.54 ± 0.02^{a}
Travel from food	0.63 ± 0.03^{c}	0.59 ± 0.03	–	$0.72 \pm 0.00^{a,c}$	0.61 ± 0.03
Long dives					
Travel to food	0.78 ± 0.02^{b}	$0.71 \pm 0.02^{a,b}$	0.75 ± 0.02	0.79 ± 0.02^{b}	0.76 ± 0.02^{b}
Travel from food	$0.85 \pm 0.02^{b,c}$	$0.76 \pm 0.03^{a,b}$	0.87 ± 0.02^{c}	$0.87 \pm 0.03^{b,c}$	$0.82 \pm 0.00^{b,c}$

Values are means ± S.E.M. ($N = 6$), measured in $m\,s^{-1}$.
[a] Significantly different from normoxia values within corresponding behavioral component (one-way RM ANOVA; $P < 0.05$).
[b] Significantly different from corresponding short behavioural component (paired t-test; $P < 0.05$).
[c] Significantly different from travel to food within the short or long behavioural component (paired t-test; $P < 0.05$).

intervals were twice as long under these conditions as they were under control conditions, causing a significant decrease in the D:P ratio. Surface intervals also tended to be higher during 9% hypoxia exposure, compared to control values, but this difference was not significant due to the large variation in these surface intervals. The proportion of the dive cycle spent foraging was significantly lower than under control conditions during hypercapnic and hypoxic exposures.

During all gas treatments, excluding hyperoxia, the animals swam faster on the return journey from the food compared with travel speed to the food and these speeds were similar in each treatment group (Table 2).

3.2. The effect of dive distance on dive behavior

The 2.5-fold increase in distance to the long dive foraging site induced marked changes in dive behavior (Table 1). Dive durations did not increase in proportion with dive distance because the animals swam faster during long dives (Table 2). Surface intervals within each gas treatment were significantly longer than for the corresponding short dive. The exception was during hypoxia treatments, where the values were higher, but not significantly so, due to large variations in long dive surface intervals.

Long dive foraging times during normoxic, hyperoxic and hypercapnic treatments were similar to the corresponding short dive values. However, when animals were exposed to low oxygen levels, long dive foraging time significantly decreased compared to the corresponding short dive foraging time.

3.3. Cardiac responses during short and long dives

Comparisons of short and long dive heart rate profiles are illustrated in Fig. 3A and the corresponding heart rate averages for the dive components are given in Table 3.

Under control conditions, before the onset of a short dive, heart rate gradually increased reaching peak values of 353.5 ± 18.8 beats min^{-1} in the last 2 s before the dive (Fig. 3A). Upon submergence, heart rate dropped to an initial low of 177.5 ± 8.0 beats min^{-1} in the first 2 s of the dive, and increased to stable levels through the mid-portion of the dive. In the last 6 s before surfacing, there was a slight anticipatory increase in heart rate, which corresponded to the behavioral component of traveling from the food back to the surface. Upon surfacing, heart rate reached a peak of 374.9 ± 0.750 beats min^{-1} within the first 4 s post-dive and subsequently decreased with time spent at the surface.

Heart rate profiles during long dives reveal similar pre- and post-dive heart rate patterns as were seen during short dives. However, during long dives, heart rate progressively declined and dropped below short dive values by 14 s into the dive (Fig. 3A). The average diving heart rate during long dives was significantly lower than in short dives although neither of these dropped below resting levels (Table 3). As

Table 3
Average heart rate (beats min^{-1}) of lesser scaup ducks during short and long dives following exposure to various gas mixtures

Dive component	Normoxia	Hyperoxia (>50%)	Hypoxia (13%)	Hypoxia (9%)	Hypercapnia (5%)
Rest	129.7 ± 7.7	157.8 ± 9.4	138.2 ± 17.3	157.7 ± 17.8	164.3 ± 14.6
Short dives					
Pre-dive	323.9 ± 17.0	237.2 ± 17.7	–	297.6 ± 30.4	295.3 ± 34.5
Dive	211.8 ± 6.9	201.8 ± 9.8	–	197.8 ± 17.1	184.1 ± 12.7
Post-dive	330.2 ± 13.3	247.7 ± 15.9[a]	–	288.4 ± 26.2	266.1 ± 26.9[a]
Long dives					
Pre-dive	289.6 ± 17.6	258.8 ± 14.0	294.5 ± 30.5	300.8 ± 36.6	308.4 ± 23.8
Dive	179.6 ± 5.0[b]	185.6 ± 7.4	181.6 ± 3.3	157.2 ± 17.5	178.8 ± 7.4
Post-dive	288.1 ± 16.9[b]	240.2 ± 6.1[a]	293.3 ± 9.7	250.2 ± 9.7	273.5 ± 11.2

Values are grand means (beats min^{-1}) ± S.E.M. (N = 6).
[a] Significantly different from normoxia values within corresponding dive component (one-way RM ANOVA; $P < 0.05$).
[b] Significantly different from short dive values of corresponding dive component (paired t-test; $P < 0.05$).

during short dives, heart rate at the end of long dives slightly increased before surfacing. Post-dive recovery heart rates were significantly lower than in short dives.

3.4. Cardiac responses during exposure to various gas mixtures

During short dives, exposure to various gas mixtures did not have large effects on pre-dive or dive heart rates (Fig. 3B; Table 3). During hyperoxic exposure, pre-dive heart rates were lower than during all other gas treatments, but the difference was not significant (Table 3). Diving heart rate was similar between all treatments although values were highest for control animals and hypercapnic animals had the lowest diving heart rate values. After the dive, heart rates during all gas treatments were lower than those found under control conditions with those for hyperoxic and hypercapnic animals being significantly lower (Table 3). Heart rate profiles show that these differences in post-dive heart rate occurred within the first 2–6 s after surfacing (Fig. 3B).

Although there were no significant differences in average diving heart rates during long dives (Table 3), the heart rate profiles indicate that during hypoxic exposure (9%), there was a gradual decline in heart rate throughout the dive with heart rate eventually falling below resting values (Fig. 3C). Heart rate dropped significantly below control levels by 18 s into the dive reaching a minimum of 107.9 ± 7.0 beats min^{-1} by the end of the dive. It is interesting to note that the intermediate hypoxia treatment (13%) did not induce any cardiovascular changes. After surfacing from 9% hypoxia dives, the peak in post-dive heart rate was delayed by 4–6 s compared to what was normally seen in control conditions.

3.5. Cardiac responses during trap dives

During both short and long dives, when the birds became aware that they were trapped underwater, heart rate rapidly decreased below resting levels (Fig. 4A). When the trap was lifted, heart rate increased slightly during the swim back to the surface and after surfacing, heart rate reached maximum post-dive values within 4–8 s.

When trapped during short dives, heart rate dropped from average dive values by 59%, reaching 117.1 ± 14.8 beats min^{-1} during the first 2 s after being trapped (Fig. 4A). Heart rate progressively dropped throughout the duration of the trap, reaching minimum levels of 93.7 ± 12.3 beats min^{-1}. Trapping during long dives induced similar heart rate patterns as during short dives, although average heart rate was slightly lower at the start of the trap and it reached minimum values of 68.3 ± 4.1 beats min^{-1} at the end of the trap (Fig. 4A). Although heart rate fell further during trapping associated with long dives, the change in heart rate during the trap was the same during short and long trap dives, both decreasing by approximately 100 beats min^{-1} (Table 4).

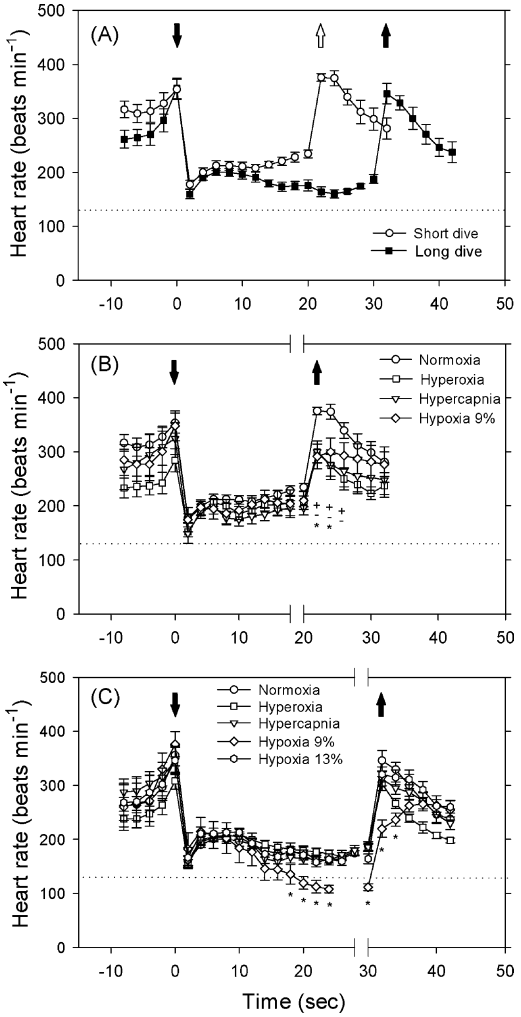

Fig. 3. (A) Heart rate profiles during short and long voluntary dives. (B) Heart rate profiles during short dives and (C) long dives following exposure to various gas mixtures. Heart rate profile values are mean ± S.E.M. averaged over 2 s time intervals from six dives per bird. Bold arrows indicate point of submergence and surfacing, respectively. Horizontal dotted line represents resting heart rate in air. (+) Hyperoxia; (−) hypercapnia; (★) hypoxia values significantly different from control (normoxia).

Pre-dive heart rate was lower than that found under control conditions in hyperoxic animals although this difference was not significant (Fig. 4B). During the dive, heart rate remained similar to that seen in control animals although post-dive heart rate was significantly lower than that found in control animals due to a decrease in the last 10–12 s after surfacing.

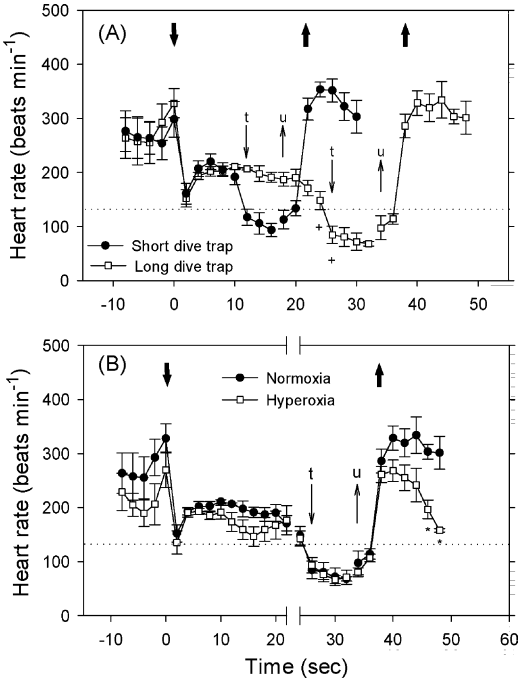

Fig. 4. Heart rate profiles during short and long trap dives (A) and of long trap dives following exposure to normoxia and hyperoxia (B). Bold arrows denote beginning and end of the respective dives. Arrow marked 't' indicates point where animals were aware of the trap door, 'u' indicates when the trap door was lifted. Heart rate profile values are mean ± S.E.M. averaged over 2 s time intervals from two dives per bird for both short and long trap dives. N = 6 birds for short trap dives and N = 4 birds for long trap dives. Dotted horizontal line indicates resting heart rate. (+) Significantly different than corresponding trap dive heart rate during short dive. (★) Significantly different from control (normoxia) values.

4. Discussion

4.1. The effect of breathing elevated or reduced levels of oxygen and carbon dioxide on diving behavior

When the animals were exposed to elevated oxygen levels, there was no change in short dive behaviour, although dive time was prolonged in long dives. Increasing the oxygen concentration of the inspired air to 50% increases the total body oxygen stores by nearly 80%. Using an average diving metabolic rate for tufted ducks of 0.9 ml O_2 s^{-1} kg^{-1} (estimated from values reported by Woakes and Butler, 1983 and Halsey et al., 2003b) and the oxygen storage value reported by Stephenson

Table 4
Average heart rate (beats min^{-1}) of lesser scaup ducks during short and long trap dives following exposure to normoxia and hyperoxia

Dive component	Trap dive normoxia	Trap dive hyperoxia (>50%)
Short dives		
Pre-dive	271.5 ± 32.1	265.5 ± 31.8
Dive	197.0 ± 14.1	197.1 ± 18.2
Trap	105.6 ± 14.9	118.9 ± 17.8
Post-dive	323.7 ± 22.1	319.2 ± 15.4
Min. trap	93.7 ± 12.3	90.3 ± 20.4
Change in trap HR	98.0 ± 10.8	101.1 ± 16.2
Long dives		
Pre-dive	285.0 ± 35.0	220.3 ± 28.6
Dive	188.0 ± 6.4	167.9 ± 13.1
Trap	76.6 ± 12.5	77.0 ± 7.6
Post-dive	312.4 ± 6.5	230.4 ± 14.0[a]
Min. trap	68.3 ± 4.1	71.3 ± 12.9
Change in trap HR	99.3 ± 11.8	89.8 ± 10.5

Values are grand means (beats min^{-1}) ± S.E.M. (N = 6 for short dives and N = 4 for long dives).

[a] Significantly different from normoxia value within corresponding dive component (paired t-test; $P < 0.05$).

et al. (1989), the calculated aerobic dive limit (cADL) during hyperoxic exposure should be 84 s. Therefore, the potential existed to extend aerobic dive duration well beyond the 30 s that long dives actually lasted. A disconnect between the size of the oxygen store and dive time during hyperoxic exposures has also been observed in tufted ducks as well as in double-crested cormorants (Butler and Stephenson, 1988; Enstipp et al., 2001, respectively).

Butler and Stephenson (1988) suggested that the shorter dive durations they observed during hyperoxic exposure were due to a gradual increase in Pa_{CO_2} throughout the dive. This contention seemed to be supported by the fact that dive duration decreased during hypercapnic exposure when the animals were performing shallow dives. They suggested that the increased Pa_{CO_2} during a dive is a strong stimulus to ventilate and is the signal that terminates a dive. However, our results indicate that dive duration was not altered during hypercania exposure during short or long dives and is unlikely to be involved in terminating dives of these lengths.

In contrast to hyperoxic exposure, decreasing oxygen had significant effects on dive behavior in both short and long dives. Long dive durations during exposure to moderate hypoxia (13%) were intermediate between those recorded under control and severe hypoxia treatments. The cADL during moderate hypoxia is 37 s and the animals surfaced well before this time limit. Reducing the inspired oxygen concentration to 9% reduced total body oxygen stores by 41% and the cADL to 28 s, which was well above short but close to long dive duration. One must keep in mind, however, that the cADL assumes that all available oxygen has been utilized while anaerobiosis begins long before all oxygen stores are depleted. Thus, not surprisingly, the birds responded by reducing durations of both short and long dives. There were indications in the long dives that severe limitations were being approached as on occasion birds were observed to switch from leg to wing propulsion, suggesting that the leg muscles were possibly anoxic and had lost their capacity for force generation (Jones et al., 1988).

In long dives, the birds significantly increased swimming speed by 30% over that seen in short dives and in all dives the birds increased swimming speed on the return leg from the food. Lowest swimming speeds on both legs occurred after breathing hyperoxic gas. The swim speeds in the short dives were similar to those of tufted ducks diving vertically on a shallow tank (0.55 m s^{-1}, Woakes and Butler, 1983; 0.68 m s^{-1}, Lovvorn et al., 1991). An increased swimming speed implies a higher rate of oxygen utilization due to the increase in mechanical costs of diving. Drag forces that occur with increases in underwater swim speed do not change substantially up to 0.5 m s^{-1}. Above this speed, drag increases and reaches about 20% of the buoyant force at the highest speeds recorded in the present experiments (Stephenson, 1994). Consequently, there will be an increase in the metabolic costs of diving that seems contraindicated especially after exposure to moderate or severe hypoxia.

Alteration in dive duration during exposure to low oxygen concentrations was due to decreases in foraging time as traveling time remained the same as it was in control animals. Foraging time seems to be relatively unchanged under a given set of conditions only falling when the ducks were pushed to extremes of their endurance (Stephenson et al., 1986). Certainly, endurance limits appeared to be reached during the severe hypoxia trials because the birds were reluctant to dive for their food and had to be fasted for several days to induce diving. Also, the birds did not reach the food during a

large portion of dives and turned back to surface (these dives were not included in the analysis).

Surface intervals following short and long dives during severe hypoxia were longer than in control animals although, due to large variability in surface times, the differences were not significant. Following many 9% hypoxia dives, the birds would not perform another dive for a considerable duration. Since the analyzed surface intervals are between two consecutive dives, an event that occurred infrequently, it is likely that the surface intervals after a dive in severe hypoxia are underestimated. The above notwithstanding, long surface intervals could be indicative of extra time required to correct acid–base disturbances caused by anaerobic end products.

Acid–base disturbances are also probably at the root of the significantly prolonged surface intervals following hypercapnic exposure before short and long dives although other, less speculative, reasons can be invoked in explanation. CO_2 must be mobilized from stores in the tissues and blood that build up during a dive (Boutilier et al., 2001). Thus, the more chemically complex mechanisms involved in CO_2 removal during the surface interval compared with oxygen uptake will increase the time to readjust CO_2 stores back to their pre-diving level. Gray seals remain at the surface until built-up CO_2 levels are readjusted even though oxygen stores are replenished within the first two breaths (Boutilier et al., 2001). On the other hand, CO_2 stores in tufted ducks are readjusted to normal values about the same time as oxygen stores are restocked (Halsey et al., 2003a) suggesting that CO_2 stores in ducks are far more readily accessible than in gray seals. Certainly, it is acid–base imbalance that drives ventilation after forced dives because arterial blood gas levels of CO_2 are restored long before ventilation returns to normal (Milsom et al., 1983).

The prolonged surface intervals after the severe hypoxia and hypercapnia dives are mixed together in the plot of dive against surface time (Fig. 2) and above the intervals for normoxia and hyperoxia treatments. The dive:pause (D:P) ratio is an indication of diving efficiency which is far lower in both short and long dives after exposure to severe hypoxia and hyperoxia. The surface intervals recorded in the 13% hypoxia treatment group were similar to those recorded in control animals and so were the D:P ratios. Due to the non-proportionate relationship between dive and surface time the D:P ratio also declines markedly between short and long dives under all imposed conditions. A reduction in diving efficiency may be the reason why hyperoxic ducks do not unduly extend their dives. If this is indeed the case, it represents a triumph of behavioral over physiological regulation.

4.2. Heart rate during short and long voluntary dives and trapping underwater

Sudden and dramatic changes in heart rate occurred at the start and termination of all dives (Butler and Woakes, 1979). These changes are accentuated by an increase in heart rate of about 50 beats min^{-1} starting some 4 s before the dive. Heart rate after 1–2 s submergence was the lowest in short but was often equaled towards the end of long dives. This produced an obvious difference in heart rate profiles between short and long dives although none of the diving heart rates appeared to be influenced by the gases breathed pre-dive except 9% oxygen. Heart rate in long dives was below that in short dives after 14 s but only in normoxia was average heart rate significantly different between short and long dives (Table 3). In long dives, the decline in heart rate with time was gradual but major and rapid changes in heart rate occurred 18 s into long dives following severe hypoxic exposure and heart rate continued to fall as the dive progressed. Under these conditions, heart rate fell below resting levels; a true diving bradycardia. Hence, it seems unlikely that peripheral chemoreceptors play any significant role in dives unless Pa_{O_2} declines below a threshold level that is obviously not broached in the majority of dives performed by this species.

These results are supported by those of Butler and Woakes (1982) who showed that in short dives by tufted ducks carotid body denervation had little effect on diving heart rate except at the end of the dive when the heart rate of denervated animals was significantly above that of intact birds. This was also the case in longer dives when heart rate fell (between 7.5 and 32.5 s of diving) almost twice as fast in intact and sham-operated birds as in denervates (Butler and Stephenson, 1988). Shams and intact ducks achieved a true bradycardia while heart rate in denervates never fell below resting levels. In contrast, a study of diving cormorants indicated an important role for chemoreceptors in control of heart rate (Enstipp et al., 2001). Mean dive heart

rate was significantly lower during shallow compared with deep diving, probably due to an increase in Pa_{O_2} during the descent phase of the dive (compression hyperoxia). As might be expected, exposure to hyperoxic and hypoxic gases before the dive increased and reduced mean dive heart rate in cormorants (Enstipp et al., 2001). Not all birds appear to be the same when it comes to cardiac control during voluntary diving!

On surfacing, heart rate returns to levels close to those established immediately pre-dive and then declines. Halsey et al. (2003a) showed that after short dives, restoration of oxygen and CO_2 stores was completed more or less simultaneously and usually before the next submergence. Heart rates are high in the post-dive period but even so circulation times will be of the order of 15 s (Bevan and Butler, 1994). Thus, it appears that restoration of stores occurs within one circulation time. Given this, it is not surprising that the impact of slight but significant changes in post-dive heart rate had little influence on surface intervals in the present experiments.

The most rapid and largest declines in heart rate seen in the present experiments were induced by trapping birds under water. Again, a major chemoreceptor contribution to the bradycardia does not appear to be present even though the sudden bradycardia resembles the oxygen-conserving response seen during forced dives (Jones and Purves, 1970; Furilla and Jones, 1986). In the present study, heart rate levels during trapping after short dives was higher than the levels reached after long dives, however, the rate of decline was similar in both (Fig. 4A). Also, the response after breathing hyperoxic gas pre-dive was the same as in ducks diving from normoxia. These observations are supported by those of Butler and Stephenson (1988) who showed that although denervation of carotid bodies significantly slows the evolution of the cardiac response to trapping there is no effect on the initial sudden decline in heart rate in intact, sham-operated and denervated ducks.

4.3. Model of oxygen store utilization during a voluntary dive

Results from our study reveal that there are no major cardiovascular adjustments during the majority of voluntary dives in lesser scaup ducks. However, during long duration dives after severe hypoxia, a bradycardia was induced and suggests that peripheral chemoreceptors exert a cardiovascular influence when oxygen levels drop below a threshold level. To obtain an estimate of the Pa_{O_2} level that stimulates peripheral chemoreceptors to induce a diving bradycardia, a model of oxygen store utilization from the lung and blood during a voluntary dive was generated.

The absolute and relative changes in oxygen stores of the lung and blood during a normoxic voluntary dive are shown in Fig. 5A and B. Initially, the large oxygen store in the lung serves as an oxygen reservoir 'topping up' arterial blood to maintain hemoglobin saturation, and thus, content near pre-dive levels. Therefore, at the beginning of a dive, the lung oxygen store falls quickly whereas the rate of decline in the arterial store is slow (Fig. 5B). When Pa_{O_2} declines to levels that cause large drops in saturation, the arterial store starts to decline more rapidly (about 15 s into the dive; Fig. 5) and by approximately 35 s into the dive, the venous store drops to zero and arterial oxygen content drops to levels that are insufficient to supply aerobic metabolism in the tissues at the given metabolic rate. This is when we expect the peripheral chemoreceptors to induce bradycardia at this given metabolic rate and at this point in the dive, the total oxygen store equals 7.0 ml kg^{-1} and the critical Pa_{O_2} is 26 mmHg (Fig. 5A).

When exposed to severe hypoxia, total oxygen stores are reduced by 46% with the largest decline in the lung store. Fig. 5C and D illustrates how the oxygen stores are utilized during an hypoxic dive. At the onset of the dive, due to the low oxygen store in the lung, arterial stores start to decline quickly and by 19 s a critical Pa_{O_2} of 21 mmHg is reached, corresponding to total store of 5.5 ml O_2 kg^{-1}. The model is consistent with our experimental results, as during the hypoxia treatments, a bradycardia occurred 18 s into the dive. Therefore, based on our normoxia and hypoxia models, we estimate peripheral chemoreceptors are stimulated to levels that cause a diving bradycardia when Pa_{O_2} reaches between 21 and 26 mmHg.

The critical Pa_{O_2}s derived from the model of 21–26 mmHg are surprisingly low but if the animal switched to an oxygen-conserving mode at this time then the stores may be sufficient to last for 30–60 s. In forced dives of Pekin ducks, Hudson and Jones (1986) showed that the EEG began to show impairment when Pa_{O_2} reached about 30 mmHg, which would apparently set a lower limit to tolerance of submergence. The

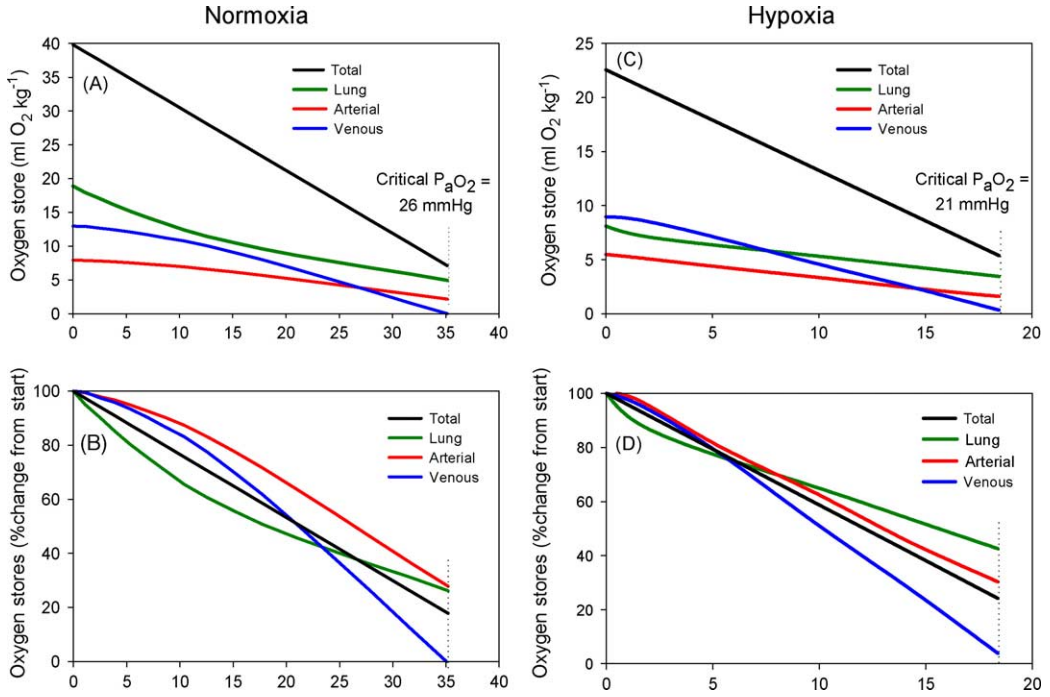

Fig. 5. Model of oxygen store utilization during a voluntary dive in lesser scaup ducks. Absolute (A) and relative (B) changes in oxygen stores in the lung and blood during a normoxic dive. Absolute (C) and relative (D) changes in oxygen stores in the lung and blood during an hypoxic dive. $\dot{V}_{O_2}$ = 0.93 ml O_2 kg^{-1} s^{-1}. Vertical dotted line represents dive time at which the 'critical Pa_{O_2}' was reached and thus bradycardia was expected.

forced dive critical Pa_{O_2} was accompanied by a very high level of CO_2 which would have influenced the relation between Pa_{O_2} and oxygen content. We have not included CO_2 dynamics in the model because the high solubility of CO_2 in the total body water complicates the relation between CO_2 production and its appearance in the blood. Nevertheless, elevated Pa_{CO_2} levels will increase Pa_{O_2} for a given content, narrowing the gap between critical Pa_{O_2}s determined by experiment on the one hand and modeling on the other.

Our model suggests an important role for the lung during voluntary diving in lesser scaup ducks. These animals perform short duration, shallow dives (Custer et al., 1996) and it appears that the large lung store 'tops up' Pa_{O_2} levels during diving such that they do not drop to levels that stimulate peripheral chemoreceptors to induce cardiovascular adjustment. Diving birds that perform long duration dives and ones that rely more heavily on blood and myoglobin stores rather than respiratory stores, such as emperor penguins, king penguins and Georgian Shags (Ponganis and Kooyman, 2000; Bevan et al., 1997), may experience larger drops in Pa_{O_2} during a dive. Therefore, peripheral chemoreceptors may play a larger role in heart rate control which may explain the more drastic cardiovascular adjustments during diving in these animals (Ponganis and Kooyman, 2000; Bevan et al., 1997).

There have not been many measurements of Pa_{O_2} levels in freely diving birds due to difficulties of instrumentation. Furthermore, cannulation of the brachial artery has been shown to cause an elevation in heart rate that would skew diving heart rate measurements (Woakes and Butler, 1986). In forced dives, Pa_{O_2} levels in Pekin ducks reach 40 mmHg near the end of a 120 s dive. However, it is difficult to compare these values with those during voluntary diving as the cardiac physiology is very different in these two situations. One study of Pa_{O_2} measurements in tufted ducks showed that Pa_{O_2} dropped by 18 mmHg by the end an 18 s trained dive (Butler and Jones, 1997). Although

this study gives an estimation of diving Pa_{O_2} values during short duration dives, it does not give an indication of how Pa_{O_2} levels correspond to cardiac physiology throughout a dive. To our knowledge, our model provides the first estimate of the Pa_{O_2} value that corresponds with the initiation of diving bradycardia in freely diving birds.

4.4. Concluding remarks

Altering inspired gas levels before voluntary diving provides valuable insights into mechanisms underlying the diving behavior and physiology of lesser scaup ducks. Our study reveals that during the majority of dives, O_2 and CO_2 levels in the body are managed through changes in diving behavior (changes in swimming speed or foraging time) without any major cardiovascular adjustments occurring throughout the dive. We found that oxygen levels within the body influence dive duration while CO_2 clearance sets surface interval duration. Extreme cardiovascular adjustments to voluntary diving (bradycardia) are only seen towards the end of dives following exposure to severe hypoxia, but not hypercapnia, indicating that chemoreceptors are more sensitive to changes in Pa_{O_2} levels during diving. Our model of oxygen utilization indicates that Pa_{O_2} levels must drop to threshold levels of between 21 and 26 mmHg before bradycardia is evoked but our data indicate that the profound bradycardia evoked during trapping is not the result of chemoreceptor input.

Acknowledgements

This paper recognizes Peter Scheid, a friend and colleague, on the occasion of his 65th birthday. The authors wish to thank Charles Darveau for his help with generating the model as well as Aurthur Vanderhorst and Sam Gopaul for their assistance with the birds. This research was supported by NSERC Discovery Grants to D.R.J. and W.K.M.

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