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RESPIRATORY, CIRCULATORY AND METABOLIC ADJUSTMENTS DURING SWIMMING IN THE TUFTED DUCK, *AYTHYA FULIGULA*

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SUMMARY

Respiratory, and some cardiovascular and metabolic, variables were measured in tufted ducks swimming at different velocities. There were no substantial changes in any of the measured variables up to a swimming speed of $0{\cdot}5\ m\ s^{-1}$. Above this speed there were progressive increases in heart rate, oxygen uptake and respiratory minute volume. At the middle of the maximum speed range ($0{\cdot}8\ m\ s^{-1}$) these variables were 1·7 times, 2·7 times, and 3·4 times their resting values respectively. There was, therefore, an excessive increase in ventilation (hyperventilation), compared with the extra demand for oxygen, and this was evident as a significant decrease in oxygen extraction and as a significant fall in P_{CO_2} in arterial blood.

The possible causes of the hyperventilation are not obvious as there was no hyperthermia and no change in pH of the arterial blood; a 2·3 times increase in lactic acid was balanced by the reduction in P_{CO_2}. There was some evidence of locomotor-respiratory coupling at the highest swimming speeds (leg beat frequency: respiratory frequency, 6:1), which appeared to constrain any further rise in respiratory frequency. At the highest swimming speed tidal volume, for the first time, increased above the resting value and the level of hyperventilation was increased. Hyperventilation may, therefore, serve to maintain arterial pH in the face of a metabolic acidosis.

Arterial blood pressure, P_{O_2} and haematocrit did not change during swimming. There was a doubling in the level of plasma adrenalin with little change in noradrenalin. The possible effects of these increases are discussed.

INTRODUCTION

Birds are supreme athletes, capable of producing high levels of metabolic power during exercise. The physiological adaptations required to support this power output are of particular interest, but the study of exercise in birds, especially under natural conditions, is technically difficult.

Although flight is the predominant form of exercise in birds there have been few comprehensive reports of the respiratory and cardiovascular adjustments to flight, despite the usefulness and availability of windtunnels for studying this form of avian

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activity. Respiratory variables have been obtained from the fish crow (Bernstein, 1976) and the white-necked raven (Hudson & Bernstein, 1981), while cardiovascular changes were monitored in pigeons (Butler, West & Jones, 1977). With the exception of a study on the cardiovascular system in the emu (Grubb, Jorgensen & Conner, 1983), all other investigations on the respiratory and cardiovascular adjustments to exercise in birds have been performed on running in the domesticated duck, chicken and turkey (e.g. Kiley, Kuhlmann & Fedde, 1979; Brackenbury, Gleeson & Avery, 1982; Baudinette *et al.* 1982) and in the pigeon (Grubb, 1982), birds that would not normally run for long durations. There is, therefore, a paucity of data from wild types of birds engaged in a normal form of exercise.

Swimming is a form of moderate exercise that is natural to a wide range of birds. It was decided, therefore, to study the respiratory, and some cardiovascular and metabolic, adjustments to swimming in the tufted duck, a bird that can fly, walk, swim and dive.

MATERIALS AND METHODS

Ten adult tufted ducks, *Aythya fuligula*, of either sex and of mass between 0·54 and 0·68 kg, were raised from eggs hatched within the Department. For details of raising and holding conditions, see Woakes & Butler (1983). A pulse interval modulated radiotransmitter (Butler & Woakes, 1982) was implanted into the abdominal cavity of a bird (for details, see Butler & Woakes, 1979) at least 2 weeks before the animal was used. The transmitter enables ECG and deep body temperature to be monitored and has a life of 4–5 months. The birds were trained to swim on a variable-speed water channel (Armfield Engineering Limited, Ringwood, Hants) which is situated in the room where they were normally housed. The test section of the channel is 0·5 m square with 0·4 m depth of water. Water velocity could be varied between 0 and 1·0 $m\,s^{-1}$ and was measured by a Braystoke BFM 002 current flow meter. Training lasted for at least 2 weeks, by which time the bird was fully fit, could hold station accurately in the test section, and could swim continuously for at least 20 min at any speed from approximately 0·2 to 0·8 $m\,s^{-1}$.

During the second week of training a mask and pneumotachograph tube were fitted to the bird (cf. Glass, Wood & Johansen, 1978). The mask was composed of two sections. A semi-flexible base, moulded and machined from epoxy putty, fitted snugly over the upper surface of the bill, enclosing the nares. This was held tightly in place by a closed-ended rubber sleeve which also gave an airtight seal against the head of the duck. The pneumotachograph tube, constructed from stainless steel and packed with nylon tubing, fitted into a collar mounted in the epoxy base. Although the tube was unheated, no condensation occurred on the nylon-tubing resistive elements. All the gas inspired and expired by the duck passed through this pneumotachograph tube, which had a dead space of 1·85 ml, was linear up to at least a flow rate of 5 l min^{-1}, and had a resistance of 14·4 Pa $min\,l^{-1}$. Ports at either end of the tube were connected to a differential Hewlett-Packard 270 pressure transducer by flexible tubing. This allowed the measurement of respiratory air flow from the

swimming duck with only light restraint. Respiratory gas concentrations were continuously monitored by a Centronics MGA 007 mass spectrometer connected to a third port positioned at the base of the pneumotachograph tube.

Six of these birds had been used in a previous study in which oxygen uptake was measured by way of an open circuit respirometer (see Woakes & Butler, 1983) when the birds had no external attachments. It was possible, therefore, to see under what conditions and how long it took for the heart rate of the birds with the mask in position to reach the resting value that had been recorded in the previous study. It was found that the lowest value of heart rate was recorded when the motor of the water channel was switched on and there was a very low residual water flow ($< 0{\cdot}1\ m\ s^{-1}$) in order to orientate the bird in the flume. Thus, all other variables measured under these conditions were taken as resting.

During the experiments ECG, instantaneous heart rate, respiratory airflow, tidal volume, respiratory oxygen and carbon dioxide concentrations were recorded on an Ormed six-channel thermal pen recorder with rectilinear coordinates. After fitting the mask and placing the bird into the water channel, a period of 2–3 h was allowed for the animal to settle at a low swimming speed ($<0{\cdot}4\ m\ s^{-1}$) before recordings were made. The experimental procedure commenced with randomly setting the velocity of the water flume and then swimming the duck for at least 15 min or until all the measured variables were stable, whichever was longer. At the end of this period, the variables were recorded for 2–3 min and deep body and water temperatures were noted. The water velocity was then altered arbitrarily and the procedure repeated. Both the pneumotachograph and the mass spectrometer were calibrated before and after each session of experiments and only one session of approximately 2·5 h was performed in any one day. The birds showed no indication of tiring over this period.

During the analysis of these data, a period representing approximately 20 respiratory cycles was selected from each recording. By using a BBC model B microcomputer and a GTCO digitizing pad (Digipad 5) the traces for respiratory air flow and respiratory gas concentrations were digitized, thus enabling the following to be calculated: respiratory frequency, tidal volume (V_T), respiratory minute volume ($\dot{V}_I$), oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide production ($\dot{V}_{CO_2}$), respiratory exchange ratio (RE), percentage extraction of oxygen from the inspired air (% Extr.), peak expiratory gas concentrations and peak inspiratory air flow. Values for tidal volume, respiratory minute volume and peak inspiratory air flow have been corrected to BTPS, whereas those for oxygen uptake and carbon dioxide production have been corrected to STPD.

To obtain physiological measurements that are representative of a particular swimming speed, all measurements taken within $\pm\ 0{\cdot}05\ m\ s^{-1}$ of that chosen velocity were combined and are given as mean ± S.E. with the number of observations in parentheses. A similar procedure was used to determine the value of any variable at a given value of oxygen uptake; measurements within $\pm\ 0{\cdot}025\ ml\ STPD\ s^{-1}$ of the chosen value of oxygen uptake were grouped together.

After these experiments were completed the right brachial artery was cannulated under local anaesthesia (2% w/v Xylocaine with adrenalin 1:80 000) in six of the

ducks before they were placed on the water channel. The cannula was connected to a Bell & Howell pressure transducer (type 4-327-L221) *via* a three-way tap so that arterial blood samples could be taken for the measurement of various factors. Partial pressures of oxygen (Pa_{O_2}) and carbon dioxide (Pa_{CO_2}) together with pH (pHa) were measured using a Radiometer BMS3 Mk II blood micro system and a PHM 71 acid-base analyser with the appropriate Radiometer electrodes. The system was thermostatically controlled at the deep body temperature recorded in the bird and the electrodes were calibrated before each set of measurements. Haematocrit (Hct) was measured with a Hawksley micro-haematocrit centrifuge. Oxygen content of the blood (Ca_{O_2}) was measured by a Lex-O_2-Con analyser. Lactic acid and the catecholamines adrenalin and noradrenalin were also measured. Lactic acid was assayed enzymatically using a Sigma standard kit and a Beckman 25 spectrophotometer, while the catecholamines were measured using a Bioanalytical HPLC system with an Altex pump.

In order to keep the amount of blood removed from these animals to a minimum, they were swum at three velocities only: approximately $0{\cdot}3\,m\,s^{-1}$, $0{\cdot}5\,m\,s^{-1}$ and $0{\cdot}7\,m\,s^{-1}$. The data obtained at $0{\cdot}3\,m\,s^{-1}$ are taken to represent the resting values. This was thought to be acceptable as neither heart rate nor oxygen uptake increase substantially above the resting values at swimming speeds below approximately $0{\cdot}4\,m\,s^{-1}$ (Woakes & Butler, 1983).

The *t*- or *d*-tests or, where appropriate, simultaneous multiple comparison procedures (Wallenstein, Zucker & Fleiss, 1980) were used to test the significance of any difference between two mean values, and the word 'significant' in the present report means at the 95 % confidence limit ($P < 0{\cdot}05$).

RESULTS

Respiratory variables

Mean values (±S.E.) for all the variables, measured and calculated for the 10 ducks with the face mask, at rest and swimming at different velocities, are given in Table 1A, and sample traces which show many of the respiratory variables at rest and at two swimming speeds are shown in Fig. 1. Oxygen uptake ($\dot{V}_{O_2}$) and heart rate varied, in response to increased swimming velocity, in a similar fashion to that reported in a previous study (Woakes & Butler, 1983), when six of the ducks were merely enclosed within a respirometer box and had no attached measuring equipment. Both variables increased little above their resting values up to a swimming velocity of $0{\cdot}5\,m\,s^{-1}$, when there were large increases in both. At an average velocity of $0{\cdot}4\,m\,s^{-1}$, neither oxygen uptake nor heart rate were significantly different from the resting value. At $0{\cdot}8\,m\,s^{-1}$, which was the middle of the highest speed range, oxygen uptake was 2·7 times the resting value and heart rate was 1·7 times resting. The rate of CO_2 production was substantially less than oxygen uptake, giving an RE of 0·76 in the resting birds. This ratio did not change substantially up to a swimming speed of $0{\cdot}6\,m\,s^{-1}$. At $0{\cdot}7\,m\,s^{-1}$, RE was significantly above the resting value.

Table 1A. *Means ± S.E. of measured variables from tufted ducks while at rest and swimming at different velocities*

	Rest	Swimming velocity ($m\,s^{-1}$) 0·3	0·4	0·5	0·6	0·7	0·8
	(13)	(9)	(42)	(54)	(68)	(38)	(13)
Oxygen uptake (ml STPD s^{-1})	0·203 ± 0·018	0·237 ± 0·020	0·227 ± 0·010	0·243 ± 0·009	0·323 ± 0·010	0·405 ± 0·013	0·544 ± 0·022
CO_2 production (ml STPD s^{-1})	0·156 ± 0·015	0·187 ± 0·016	0·181 ± 0·010	0·186 ± 0·008	0·256 ± 0·008	0·335 ± 0·011	0·467 ± 0·026
Respiratory exchange ratio	0·76 ± 0·02	0·79 ± 0·02	0·79 ± 0·01	0·77 ± 0·01	0·79 ± 0·01	0·83 ± 0·01	0·86 ± 0·02
Respiratory frequency (breaths min^{-1})	13·5 ± 1·1	16·7 ± 1·5	14·8 ± 0·6	15·9 ± 0·5	22·9 ± 0·7	30·7 ± 1·8	36·0 ± 2·5
Tidal volume (ml BTPS)	21·4 ± 1·3	22·3 ± 2·3	20·5 ± 0·8	19·9 ± 0·6	19·7 ± 0·5	21·7 ± 0·7	26·7 ± 1·4
Respiratory minute volume (ml BTPS min^{-1})	267 ± 29	333 ± 47	309 ± 18	322 ± 14	459 ± 16	638 ± 29	912 ± 54
Percentage extraction of oxygen	27·7 ± 1·8	27·6 ± 1·4	26·8 ± 0·9	27·9 ± 1·2	25·5 ± 0·6	23·5 ± 0·8	21·7 ± 1·2
End expired O_2 (%)	13·00 ± 0·17	13·36 ± 0·12	12·92 ± 0·12	12·61 ± 0·09	12·80 ± 0·09	13·43 ± 0·14	14·05 ± 0·31
Peak inspiratory air flow (l BTPS min^{-1})	1·76 ± 0·09	1·91 ± 0·12	1·94 ± 0·05	1·90 ± 0·04	2·23 ± 0·05	2·61 ± 0·07	3·36 ± 0·16
Deep body temperature (°C)	39·9 ± 0·4	41·2 ± 0·2	40·7 ± 0·1	40·7 ± 0·1	40·8 ± 0·1	40·7 ± 0·1	40·4 ± 0·2
Heart rate (beats min^{-1})	149 ± 9	170 ± 15	155 ± 5	160 ± 4	182 ± 4	189 ± 7	257 ± 19

Mean values of respiratory variables and heart rate from 10 animals (mean mass 613 ± 5 g). Numbers in parenthesis at the top of each column are the numbers of observations. Water temperature was 17·7 ± 0·2°C.

Table 1B. *Means ± S.E. of measured variables from tufted ducks while at rest and swimming at different velocities*

	Swimming velocity ($m\,s^{-1}$)		
	0·3	0·5	0·7
Partial pressure of oxygen in arterial blood (kPa)	11·0 ± 0·3	11·1 ± 0·2	11·0 ± 0·3
Oxygen content of arterial blood (vol %)	20·8 ± 0·6	20·1 ± 0·4	19·8 ± 0·3
Partial pressure of CO_2 in arterial blood (kPa)	4·7 ± 0·1	4·7 ± 0·2	4·5 ± 0·1
pH in arterial blood	7·481 ± 0·006	7·481 ± 0·005	7·477 ± 0·009
Haematocrit (%)	45·9 ± 0·97	45·0 ± 0·45	44·2 ± 0·31
Lactic acid concentration ($mmol\,l^{-1}$)	0·83 ± 0·2 (4)	0·94 ± 0·2 (4)	1·9 ± 0·2 (4)
Plasma adrenalin concentration ($nmol\,l^{-1}$)	13·1 ± 2·3 (4)	—	27·4 ± 4·5 (4)
Plasma noradrenalin concentration ($nmol\,l^{-1}$)	12·5 ± 1·2 (4)	—	16·6 ± 1·1 (4)
Oxygen uptake ($ml\,STPD\,s^{-1}$)	0·207 ± 0·022	0·269 ± 0·012	0·439 ± 0·027
Deep body temperature (°C)	41·0 ± 0·2	—	40·7 ± 0·7
Arterial systolic pressure (kPa)	25·3 ± 0·9	25·6 ± 0·9	26·7 ± 1·1
Arterial diastolic pressure (kPa)	19·9 ± 0·5	19·9 ± 0·6	21·5 ± 0·7
Heart rate ($beats\,min^{-1}$)	193 ± 10	209 ± 6	301 ± 9

Mean values of oxygen uptake, heart rate, blood gases, catecholamines, lactate and arterial blood pressure from 6 animals (mean mass 592 ± 10 g). Number of observations is 6 in all cases except where indicated. Water temperature was 19·5 ± 1·1°C.

Respiratory frequency and respiratory minute volume ($\dot{V}_I$) also changed little up to a swimming speed of $0{\cdot}5\,m\,s^{-1}$ (neither was significantly above the resting value at $0{\cdot}4\,m\,s^{-1}$). Both then increased more rapidly with increased swimming speed. At $0{\cdot}8\,m\,s^{-1}$ they were 2·7 times resting and 3·4 times resting, respectively. Tidal volume was significantly different from the resting value at the highest swimming speed, when it was 1·25 times resting. If respiratory frequency and tidal volume are plotted against oxygen uptake (Fig. 2), it can be seen that there was a steady increase in respiratory frequency as oxygen uptake rose to 0·4–0·5 ml O_2 STPD s^{-1} but then it levelled off. On the other hand, tidal volume did not increase above the resting value until oxygen uptake was above 0·5 ml O_2 STPD s^{-1}. At the highest level of oxygen uptake (i.e. at the highest swimming speed) tidal volume was significantly above the resting value.

Despite the fact that tidal volume did not increase above resting, except at the highest swimming speed, peak inspiratory air flow did show a substantial increase at swimming velocities above $0{\cdot}4\,m\,s^{-1}$, and at $0{\cdot}8\,m\,s^{-1}$ it was 1·9 times the resting value. This relates to the reduction in the duration of the respiratory cycle (rise in respiratory frequency) at the higher swimming speeds. As the birds swam above $0{\cdot}6\,m\,s^{-1}$ there was a noticeable increase in end expired oxygen concentration so that at $0{\cdot}8\,m\,s^{-1}$ it was 1% higher than the resting value. This increased level of

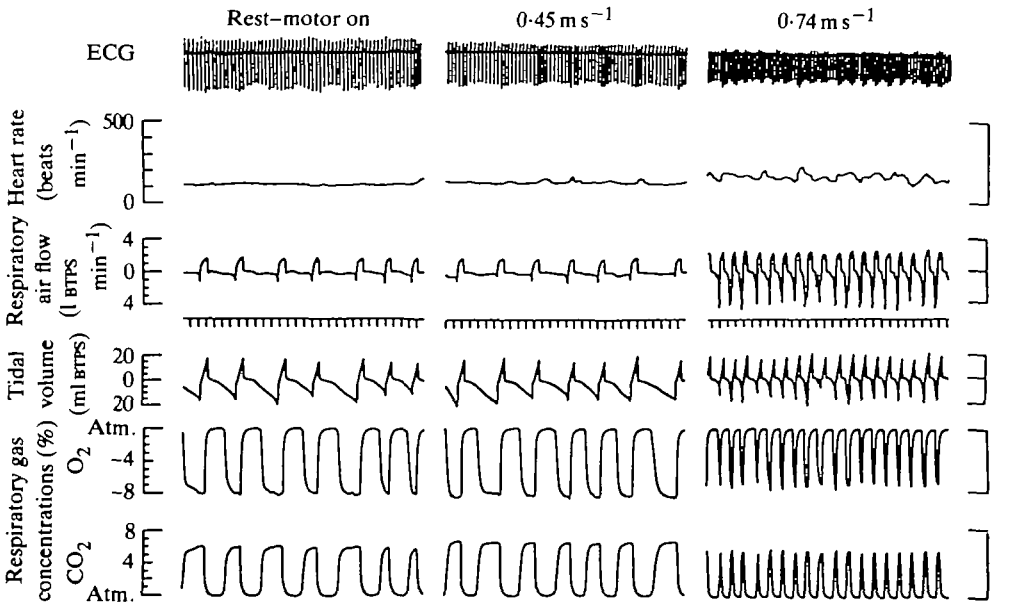

Fig. 1. Traces of respiratory and cardiovascular variables recorded from a tufted duck at rest and while swimming at 0·45 and $0{\cdot}74\,m\,s^{-1}$. For respiratory air flow and tidal volume, up on trace indicates inspiration. Time marker in seconds.

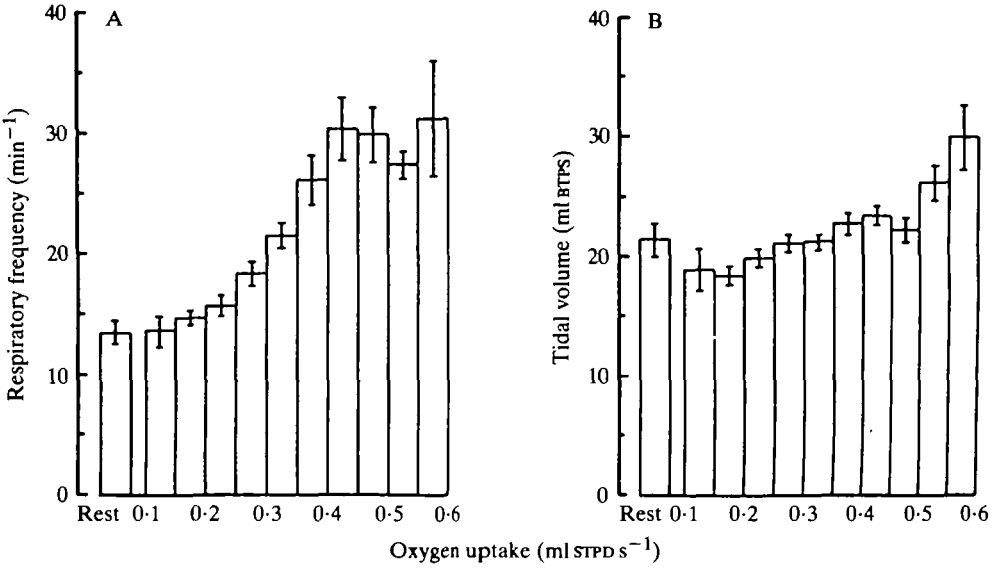

Fig. 2. Respiratory frequency (A) and tidal volume (B) at different values of oxygen uptake in tufted ducks swimming on a water channel. Mean values from 10 birds ± S.E. of mean.

oxygen in the expired gas was reflected in the value of percentage extraction of oxygen (% Extr.), which was obtained as follows:

$$[\dot{V}_{O_2}\,(\text{ml STPD min}^{-1})/\dot{V}\text{I}\,(\text{ml STPD min}^{-1})\times 0{\cdot}2095]\times 100.$$

Oxygen extraction is another variable which remained relatively unchanged (at approximately 27·5 %) up to a swimming speed of 0·5 m s^{-1} but then declined. At 0·7 m s^{-1} it was significantly below the resting value. Another way of presenting this is to relate oxygen uptake to respiratory minute volume: $\dot{V}$I (ml BTPS min^{-1})/$\dot{V}_{O_2}$ (ml STPD min^{-1}). This is called the ventilatory requirement and, taking the mean values of $\dot{V}$I and $\dot{V}_{O_2}$ from Table 1A, it is increased from 21·9 ml (ml O_2)$^{-1}$ (0·491 mmol^{-1}) at rest to 27·9 ml (ml O_2)$^{-1}$ (0·621 mmol^{-1}) at a swimming speed of 0·8 m s^{-1}.

After an initial increase of more than 1 °C, when going from rest to a swimming speed of 0·3 m s^{-1}, followed by a decrease of 0·5 °C at 0·4 m s^{-1}, deep body temperature did not change at increased swimming velocities.

Blood gases and cardiovascular variables

There were no significant changes in arterial blood pressure, haematocrit, the partial pressure of oxygen in arterial blood (Pa_{O_2}), oxygen content of arterial blood (Ca_{O_2}) or pH of arterial blood (pHa) in response to increased swimming speed (Table 1B). There was, however, a slight but statistically significant reduction in the partial pressure of carbon dioxide of arterial blood (Pa_{CO_2}) at a swimming speed of 0·7 m s^{-1} compared with that of 0·3 m s^{-1}. There was a 2·3 times increase in the

concentration of lactic acid in the blood, a doubling in the concentration of plasma adrenalin and a 1·3 times increase in plasma noradrenalin.

DISCUSSION

Assessment of techniques

In a previous series of experiments (Woakes & Butler, 1983) oxygen uptake and heart rate were recorded from tufted ducks, with no attachments, in a respirometer, so it is possible to compare the data from that study with those from the present investigation in order to determine the effects of the recording techniques on the measured variables.

Thus, at swimming speeds of 0·3, 0·4 and 0·5 $m\,s^{-1}$ oxygen uptake in the animals with the mask, but not cannulated, is significantly higher than that recorded from the animals in the respirometer, whereas at 0·7 $m\,s^{-1}$ it is significantly lower. At rest and at the other two swimming speeds there is no significant difference between the two sets of data. Oxygen uptake in those animals that also had an artery cannulated is not significantly different from that in the ducks with the mask alone (Fig. 3A). We feel it is safe to conclude that the technique for determining oxygen uptake (and, by inference, carbon dioxide production) is accurate and that neither the mask alone nor the mask with cannulation of an artery had any consistent effect on oxygen uptake in the birds. It is assumed, therefore, that all of the measured respiratory variables that were used in the computation of oxygen uptake are also accurate and not affected by the small dead space and added resistance of the mask and pneumotachograph screen. Having said that, it is apparent from Fig. 3A that there is more variability in the values of oxygen uptake in the masked birds compared with those in the respirometer, particularly at rest and at the two lowest swimming speeds. This probably explains why oxygen uptake at 0·4 $m\,s^{-1}$ was not significantly greater than at rest in the masked birds, whereas it was in those in the open circuit respirometer (see Woakes & Butler, 1983).

Tidal volume at rest compares favourably with that measured by tracheal cannulation in other birds (Butler & Taylor, 1973, 1974). Respiratory frequency at rest is slightly greater than that recorded from inactive, free-range tufted ducks (Woakes, 1980), but it is somewhat lower than that for free-range pochard ducks drifting on water (Butler & Woakes, 1979). The derived value of % Extr. and ventilatory requirement at rest are similar to those for the Pekin duck at 20 °C (Bech, Johansen, Brent & Nicol, 1984).

One variable that does seem to be affected by the recording techniques is heart rate. Although the values at rest and at swimming speeds of 0·7 and 0·8 $m\,s^{-1}$ are not significantly different between the ducks in the respirometer box and those with a mask, but without a cannula, there is an overall higher heart rate in the latter group compared with those in the respirometer. This is evident in the regression equations for heart rate against oxygen uptake under the two conditions. For those with the mask the relationship is $y = 98{\cdot}3+(253\pm17{\cdot}8)x$ and for those in the respirometer it is $y = 65{\cdot}3+(288\pm10{\cdot}8)x$, where y is heart rate (beats min^{-1}) and x is oxygen uptake

(ml STPD s^{-1}). There is no significant difference between the slopes of these two lines, whereas the intercept is significantly higher for the ducks with the mask than for the ducks in the respirometer. Heart rate is even higher in the ducks after cannulation of an artery. The high value of heart rate at a swimming speed of 0·3 m s^{-1} in the masked birds seems to hide this tendency. However, heart rate at rest and at a swimming speed of 0·4 m s^{-1} is significantly less in these birds than heart rate at 0·3 m s^{-1} in the cannulated birds. It is probably safe to say, therefore, that at *all* swimming speeds heart rate is significantly higher in the cannulated birds than in the other two groups (Fig. 3B).

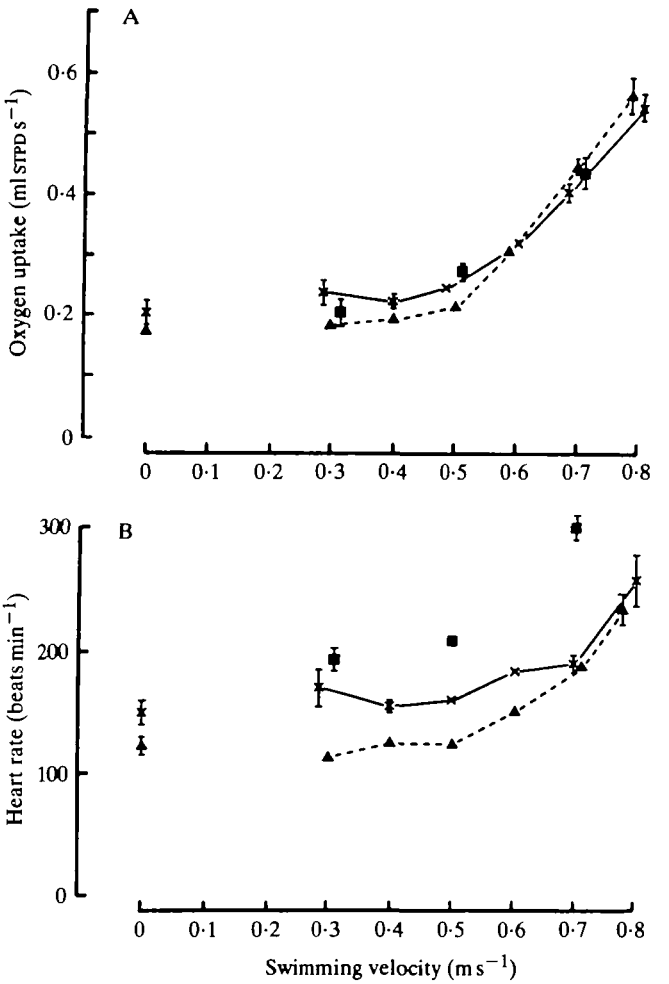

Fig. 3. Mean values, ± S.E., of oxygen uptake (A) and heart rate (B) in tufted ducks at different swimming velocities. Data were obtained from 10 ducks with a mask and pneumotachograph tube attached over the nares (×), from six ducks with a mask, pneumotachograph tube and a cannulated artery (■) and from six birds with no attachments in an open circuit respirometer (▲). The last set of data from Woakes & Butler (1983).

The fact that the large differences in heart rate between the three groups of ducks were not accompanied by equally large differences in oxygen uptake, illustrates the problems of using heart rate as an indicator of metabolic rate in field studies. It appears that the same animals under different conditions can have different relationships between these two variables. Exhaustive studies of these relationships in the laboratory are necessary before the technique can be applied to the field, but then the telemetry of heart rate could be an extremely powerful tool in establishing the energy budgets of animals in their natural environment.

According to a recent study (Grubb, 1983), the allometric formula for heart rate (HR) in birds is $HR = 178{\cdot}5M^{-0{\cdot}282}$ where M is body mass in kg. The value of resting heart rate recorded from the ducks in the respirometer box (Woakes & Butler, 1983) is 59% of the value predicted by this formula, while that of the ducks with the face mask is 72% of the predicted value. Resting heart rate in the cannulated ducks with a face mask is similar to the predicted value. Resting heart rates recorded in instrumented ducks in other studies were 21% greater (Bech & Nomoto, 1982), 50% greater (Grubb, 1982) and 71% greater (Kiley *et al.* 1979) than the values predicted from the allometric formula above. There is no doubt, therefore, that resting heart rate recorded by radiotelemetry in unencumbered birds is substantially lower than values in the literature obtained by more conventional methods (see also Butler & Woakes, 1980). It is also clear that, although encumbrances such as face masks and cannulae in blood vessels do cause an increase in heart rate, some workers may not have allowed their animals sufficient time to settle completely before commencing the investigation.

Arterial blood pressure is similar to that of lightly restrained mallard ducks (Butler & Taylor, 1983), while haematocrit is 6% lower than that measured in lightly restrained tufted ducks (Keijer & Butler, 1982). Arterial P_{O_2} in the tufted ducks swimming slowly is similar to that measured in lightly restrained mallards (Butler & Taylor, 1983) and in unrestrained, undisturbed domestic ducks (Kawashiro & Scheid, 1975). Arterial P_{CO_2} and pH in the slowly swimming tufted ducks lie between the values quoted for the mallards and domestic ducks in the above papers. Blood lactate in the slowly swimming tufted ducks is similar to that measured in resting pigeons (Butler *et al.* 1977) but considerably lower than the value presented by Kiley, Kuhlmann & Fedde (1982) in resting domestic ducks. On the other hand, the levels of plasma adrenalin and noradrenalin in slowly swimming tufted ducks are 2–4 times higher than in resting domestic ducks and chickens (Hudson & Jones, 1982; Rees, Hall & Harvey, 1984). There is no obvious explanation for this unless the low level of activity performed by our 'resting' ducks causes catecholamines to rise above the true resting level.

In view of the above points, any further reference to heart rate in the present study will apply to the values given in Table 1A; those in Table 1B will be ignored.

Changes during swimming

The absence of exercise hyperthermia in the present study is not surprising as the birds were able to dissipate metabolic heat across the legs and feet to the water. There

was, therefore, no thermal stimulus to ventilation as the ducks increased their swimming from $0{\cdot}3\,m\,s^{-1}$ and yet there is clear evidence of hyperventilation at the two highest swimming speeds. The reduction in % Extr., the slight hypocapnia and possibly the increase in RE are all signs that at $0{\cdot}7\,m\,s^{-1}$ and above, ventilation was higher than was required to match the increased demand for oxygen and the accompanying rise in the production of CO_2. There was no change in arterial pH, presumably because of the increase in blood lactic acid.

Hyperventilation, leading to hypocapnia, is a common phenomenon in exercising birds (Butler *et al.* 1977; Kiley *et al.* 1979; Brackenbury, Gleeson & Avery, 1981) and in a number of mammals (Flandrois, Lacour & Eclache, 1974; Smith *et al.* 1983; Pan *et al.* 1983). In birds, at least part of the excessive ventilation is the result of an increase in body temperature, for prevention of such an increase reduces, but does not abolish, the hypocapnia (Kiley *et al.* 1982; Brackenbury & Gleeson, 1983). The hyperventilation that remains has been attributed to a metabolic acidosis (Gleeson & Brackenbury, 1984), although the present study demonstrates that this need not be the case.

The slight hypocapnia at $0{\cdot}7\,m\,s^{-1}$ (compared with Pa_{CO_2} at $0{\cdot}3\,m\,s^{-1}$) was the result of a small difference in the proportional increases in respiratory minute volume and oxygen uptake between these two velocities (1·9 and 1·7 times, respectively). The causative hyperventilation resulted entirely from an excessive increase in respiratory frequency, as there was no change in tidal volume. At $0{\cdot}8\,m\,s^{-1}$ respiratory minute volume was 2·7 times the value at $0{\cdot}3\,m\,s^{-1}$, whereas oxygen uptake was 2·3 times greater. In other words the hyperventilation increased. This was apparent as a further decrease in % Extr. and, presumably, a more pronounced hypocapnia. This time, however, there was a significant increase in tidal volume as well as a rise in respiratory frequency.

Bearing these changes in mind, it is interesting to see in Fig. 4 that when the ducks were swimming at 0·7 and $0{\cdot}8\,m\,s^{-1}$ there was a fixed relationship between leg beat frequency and respiratory frequency of 6:1, whereas at lower swimming speeds no such correspondence existed. Correspondence between limb beat frequency and respiratory frequency during steady-state exercise has been seen in flying birds (Butler *et al.* 1977; Butler & Woakes, 1980) and in a number of mammals (Bramble & Carrier, 1983). It is assumed that such locomotor-respiratory coupling is an important factor in the efficient operation of both systems. The effect in the tufted ducks was to increase respiratory frequency so that its proportional increase above the resting value was greater than that for oxygen uptake at $0{\cdot}7\,m\,s^{-1}$, but to bring them into line at $0{\cdot}8\,m\,s^{-1}$. It is, therefore, difficult to explain why tidal volume should have increased at the highest swimming speed and actually enhanced the hyperventilation, unless the function of this excessive ventilation was in fact to produce a respiratory alkalosis to counteract the metabolic acidosis and thus maintain arterial pH. For animals exercising on land or in the air, it may also have a thermoregulatory function. In the present experiments, thermoregulation may have been adequately dealt with by the additional ability of the ducks to lose heat across the feet and legs to the water, and the degree of hyperventilation was adequate to

maintain arterial pH in the face of a moderate metabolic acidosis. The absence of a detectable 'error' in these variables does not mean that compensatory respiratory adjustments were not being made to prevent acidosis and/or hyperthermia.

It may seem strange that, with Pa_{O_2} at its normal level and with heart rate well below its maximum (Butler & Woakes, 1985), the level of blood lactate was elevated at a swimming speed of $0{\cdot}7\,m\,s^{-1}$. The explanation of a similar phenomenon in mammals is that lactic acid is always produced, even in normoxic individuals at rest, and the low, stable levels of lactate in the blood merely indicate that it is being removed as rapidly as it is produced. Both production and removal rates increase as aerobic metabolism increases, although rate of removal does not quite keep pace with rate of production so that a slight accumulation of blood lactate occurs as the intensity of sustainable exercise increases (Brooks, 1985). Most of the lactate is removed by oxidation, and in rats during submaximal exercise the contribution of anaerobiosis to total ATP production is insignificant (Brooks, Donovan & White, 1984). These authors propose that most of the lactate produced during steady-state exercise, mainly by fast glycolytic (FG) muscle fibres, is oxidized by fast oxidative glycolytic (FOG) and slow oxidative (SO) muscle fibres as well as by other tissues. Hochachka, Runciman & Baudinette (1985) have further suggested that the function of anaerobic metabolism in FG fibres is less to produce ATP than to generate lactate as a fuel for FOG and SO fibres.

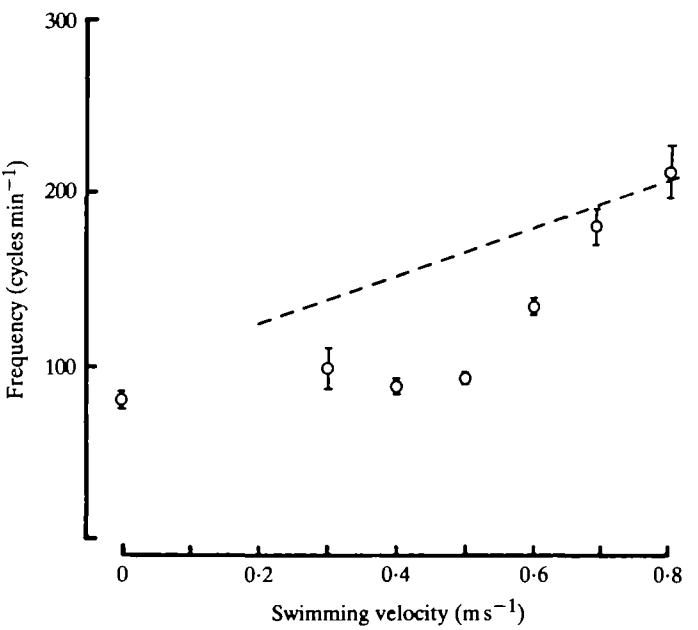

Fig. 4. Mean values, ± S.E., six times respiratory frequency in tufted ducks swimming at different velocities. Dashed line is the mean relationship between leg beat frequency and swimming velocity in tufted ducks from Woakes & Butler (1983).

In birds, $\dot{V}_{O_2}max$ is lower during running and swimming than during flight (Butler, 1982), and yet arterio-venous oxygen difference ($a-\bar{v}O_2$ difference) increases by approximately 50% in running ducks (Grubb, 1982; Bech & Nomoto, 1982), compared with an 80% increase in flying pigeons (Butler *et al.* 1977). As the increase in heart rate was less than the rise in oxygen uptake in the present study, it is assumed that during swimming also, $a-\bar{v}O_2$ difference increases despite the fact that heart rate is not at its maximum (Butler & Woakes, 1985). This would indicate a limitation of blood flow to the active leg muscles. There is no indication of an increase in oxygen-carrying capacity of the blood during exercise in birds. This is unlike the situation in some mammals where the mobilization of erythrocytes from the spleen occurs (Pan *et al.* 1984).

The increase in cardiac output during exercise is matched by a similar proportional decrease in peripheral vascular resistance in flying pigeons (Butler *et al.* 1977) and running emu (Grubb *et al.* 1983), so that there is no significant rise in central arterial blood pressure. A similar situation occurs in swimming tufted ducks. However, when domesticated ducks and turkeys run there is a significant hypertension (Kiley *et al.* 1979; Bech & Nomoto, 1982; Grubb, 1982; Baudinette *et al.* 1982). This difference in response is probably related to the selective breeding and/or to the relatively inactive lifestyle of domesticated birds, and not to the fact that running is not a normal form of exercise in ducks, as there is no hypertension in running pigeons (Grubb, 1982). It would, nonetheless, be interesting to know what happens in the running tufted duck.

The greater increase in plasma adrenalin, compared with noradrenalin, during swimming in the tufted ducks is similar to the situation in running cockerels (Rees *et al.* 1984), but the functional significance of the rise in plasma catecholamines is unknown. They appear not to have a large lipolytic effect in birds (Freeman & Manning, 1974), although adrenalin inhibits insulin secretion and stimulates glucagon release in ducks (Tyler, Kajinuma & Mialhe, 1972). Thus, the increase in plasma glucagon during running in ducks could well be the result of the elevated levels of catecholamines (Harvey *et al.* 1982), and glucagon is the main lipolytic hormone in birds (Cramb & Langslow, 1984). It has, indeed, recently been demonstrated that at 60% of maximum work intensity there is a significant increase in plasma fatty acids in the cockerel (Brackenbury & El-Sayed, 1984).

This is similar to the situation in man, where fat is the main substrate for oxidative metabolism at moderate work loads. Also, in man there is a shift to aerobic carbohydrate metabolism at higher work loads (Gollnick, 1985). Indeed, the swimming tufted ducks show an increase in RE that is consistent with this shift in substrate at high swimming speeds ($> 0{\cdot}7\,m\,s^{-1}$). Hyperventilation will also cause an increase in RE, albeit transient. Until the dynamics of excess CO_2 removal are quantified, it is not possible to conclude that the whole of the change in RE can be attributed to a change in substrate.

In man, the major plasma catecholamine during exercise is noradrenalin and it has been suggested that it is released primarily from sympathetic nerve endings located in visceral blood vessels, its function being to reduce splanchnic blood flow (Davies,

Brotherhood, Few & Zeidifard, 1976). The same authors suggest, therefore, that the adrenal gland does not make a significant contribution to the release of catecholamines during exercise in man, and perhaps other mammals. In contrast, it does seem that both noradrenalin and adrenalin are released from the adrenal glands of cockerels during exercise, and that noradrenalin may be methylated to adrenalin (Rees *et al.* 1984). It is clear that further studies on endocrine function during exercise in birds are required.

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