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CARDIOVASCULAR CHANGES ASSOCIATED WITH TREADMILL RUNNING IN THE PEKIN DUCK

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SUMMARY

Six adult white Pekin ducks were trained to run on a treadmill for 10 min at a speed of 1·43 km h^{-1}. O_2 consumption, CO_2 production, breathing rate, blood gas tension and pH, sciatic and carotid blood flows and colonic and skin temperatures were measured in the experiments.

A 2·6 times increase in oxygen consumption was observed when going from resting on the treadmill to exercise. The increased oxygen delivery was achieved by a 61% increase in cardiac output and a 51% increase in the arterio-venous oxygen content difference. Probably the reported resting oxygen consumption was higher than a true resting level causing the increment in oxygen consumption to be more correctly 3·6 times the resting level.

Sciatic and carotid blood flow increased by 3·7 times and 2·3 times, respectively, during exercise. At cessation of exercise, the carotid blood flow decreased very rapidly, while the sciatic blood flow decreased more slowly. An increased web temperature above the exercise level was seen in the post-exercise period.

The results suggest that the ducks prefer to use non-evaporative heat loss in the post-exercise period, respiratory evaporative cooling being of minor importance. This strategy may be correlated to the small internal heat load induced by the running (body temperature increased by only 0·3 °C).

INTRODUCTION

Physical activity increases the aerobic energy requirement several-fold in vertebrates. An increased muscular activity will require a greatly increased muscle blood flow to support the elevated metabolism and for removal of metabolically produced heat from the active muscles and transport it to heat dissipating surfaces. Exercise will consequently require both an increase in cardiac output and marked alteration in regional blood flow distribution.

When in a positive heat load, the skin, particularly of the naked legs and poorly insulated portions of the body in birds, receive a greatly increased blood flow, mainly

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through arteriio-venous anastomoses. The upper respiratory tract, as the site for evaporative cooling during panting and gular fluttering, is also subjected to marked increase in blood flow during hyperthermia. Bech & Johansen (1980*b*) recorded an up to five-fold increase in carotid blood flow during panting in the duck, and Wolfenson *et al.* (1978) reported markedly elevated flow to the upper respiratory tract in hyperthermic chickens assessed by injections of radioactive microspheres.

Exercise, like heat stress, also raises an immediate need for heat dissipation. However, the transport of heat away from the muscles does not always balance muscle heat production during exercise, causing a transient heat storage which reduces the immediate requirement for heat dissipation.

In the present study direct and simultaneous recordings of blood flow in the sciatic and carotid arteries have been employed to estimate altered perfusion requirements associated with treadmill exercise in ducks.

Also, calculations of cardiac output and other circulatory parameters allowed us to evaluate the relative importance of the factors contributing to the elevated tissue oxygen supply during exercise.

MATERIALS AND METHODS

Six adult white Pekin ducks (*Anas platyrhynchos domesticus*) of mean body weight 2·13 kg were used in the study. They were housed indoor at an ambient temperature of 22–25 °C. The ducks were equipped with polyethylene catheters (PE 90) in the brachial artery and vein, the latter advanced to the right atrium. Electromagnetic blood flow probes (Statham, 2·0 mm) were placed around the internal carotid artery and the sciatic artery. The left carotid artery was exposed at the lower part of the neck, and the probe was kept in place by a 2 cm plastic bar glued to the probe head and anchored to the surrounding muscles. The leads from the probe extended out through the skin incision and were secured to strong feathers at the back. The sciatic artery was exposed by dissecting through *M. iliotibialis*, and the probe was similarly anchored in position. Blood flows were measured using Statham flowmeters (type SP 2202). The lead connexions from the bird extended through portholes at the top of the chamber surrounding the treadmill belt. Calibration of the flow probes was done by volumetric perfusion *in situ* (Bech & Johansen, 1980*b*). All operations were done under general anaesthesia (Equithesin, 2·5 ml kg^{-1} given intramuscularly).

After 2 days of recovery the ducks were equipped with a light weight (20 g) face mask made of leather and a soft plastic film before being placed on a motor-driven treadmill. An airpump (Reciprotor, Copenhagen, type 506 R) sucked air through the mask at a rate of about 14 l min^{-1}. The air was dried by passing over silica-gel and its flow rate measured using a calibrated flowmeter (Fischer & Porter, Göttingen, FRG). Aliquots of the air were guided past a Taylor Servomex paramagnetic O_2 analyser (type OA 272) and a Beckman LB 2 infrared CO_2 analyser for measurements of the fractional O_2 and CO_2 concentrations. Prior to the experiments the gas analysers were calibrated with gases accurately mixed by Wösthoff (Bochum, FRG) gas-mixing pumps fed with pure stock gases. The gas concentrations were co

nuously recorded on Servograph recorders. By combined measurements of air flow rates and gas concentrations, the O_2 uptake and CO_2 output were calculated and converted to STPD conditions using the appropriate formulas (Withers, 1977). In some experiments CO_2 production was not measured.

The arterial and venous catheters were extended through holes in the walking chamber placed over the treadmill belt. In this way blood samples could be drawn without disturbing the ducks. Blood samples were taken anaerobically into 1 ml heparinized syringes and immediately stored in iced water. Analyses were performed within a few minutes from sampling. Arterial (a) and venous ($\bar{v}$) blood was analysed for pH, O_2 and CO_2 tensions (P) using Radiometer equipment (BMS 2 and PHM 72 K). The temperature was set to 41 °C, the approximate core temperature of the ducks. Blood O_2 content (C) and capacity were determined using the method of Tucker (1967).

Heart rate and mean blood pressure were monitored by connecting the arterial catheter to a Statham pressure transducer (Pb 23 d). The output was recorded on a Gould Brush recorder (220).

Body temperature (T_B) was measured using a thermistor placed approximately 5 cm into the colon. Skin temperatures of beak (T_{beak}) and web (T_{web}) were continuously recorded using small thermistors (about 2 mm in diameter and flat) taped tightly to the skin. A Beckman temperature recording system was used to monitor all temperatures.

Respiratory rate was recorded by placing a thermocouple in the nasal opening and recording the temperature changes associated with breathing.

A minimum adjustment of 1 h to the experimental situation was used to let the ducks recover from handling. During this period they rested on the treadmill belt. The ducks were then exercised at a speed of 1·43 km h^{-1} for 10 min, after which they were allowed 1 h for recovery before a new exercise period was started. All experiments were performed at an ambient temperature of 22 °C.

Prior to actual experiments, all ducks had been trained to run on the treadmill and to wear the mask. This training period last 6–8 weeks.

RESULTS

Oxygen uptake ($\dot{M}_{O_2}$), CO_2 production ($\dot{M}_{CO_2}$), sciatic blood flow (SBF), carotid blood flow (CBF), heart rate (f_H), mean arterial blood pressure and temperature were read at 1 min intervals starting 10 min before exercise, lasting until 10 min after cessation of exercise. Subsequently, readings were made every 5 min until 30 min after termination of exercise. Blood samples were taken immediately before the running period (from 1 to 4 min before) and from minute 6 to min 9 of the 10 min steady-exercise period. In each 3 min period mixed venous blood was sampled first. The changes in most cardio-respiratory parameters (from 12 exercise periods) are summarized in Table 1. Data given are mean values for the 3 min periods. O_2 capacity was measured in eight experiments, and the values for O_2 saturation given in Table 1 are from parallel measurements of O_2 capacity and O_2 content.

P_{a,CO_2} showed a significant decrease from 27·8 to 25·7 torr during running. The simul-

Table 1. *Cardiorespiratory values in awake Pekin ducks studied before and at the end of a 10 minutes exercise period at a speed of 1.43 km h^{-1} (means ± 2 S.E.M., n = number of experiments)*

	n	Rest	Exercise	*P*
W (kg)	6	2·13 ± 0·11	—	—
$\dot{M}_{O_2}$ (ml min^{-1} kg^{-1})	12	16·62 ± 1·80	43·39 ± 2·31	< 0·01
$\dot{M}_{CO_2}$ (ml min^{-1} kg^{-1})	6	11·65 ± 1·13	33·44 ± 3·37	< 0·01
R_E	6	0·73 ± 0·03	0·81 ± 0·03	< 0·01
Acid-base status				
P_{a,CO_2} (torr)	12	27·8 ± 1·5	25·7 ± 2·3	< 0·05
pH_a	12	7·476 ± 0·010	7·488 ± 0·024	NS
$P_{\bar{v},CO_2}$ (torr)	11	30·9 ± 1·5	30·3 ± 2·4	NS
$pH_{\bar{v}}$	12	7·453 ± 0·019	7·443 ± 0·029	NS
Blood O_2 transport				
O_2 capacity (vol %)	8	13·62 ± 3·44	13·44 ± 3·20	NS
P_{a,O_2} (torr)	12	84·8 ± 2·4	82·0 ± 3·2	NS
C_{a,O_2} (ml 100 ml^{-1})	12	12·63 ± 1·42	12·51 ± 1·49	NS
O_2 saturation$_{art}$ (%)	8	93·2 ± 3·0	90·6 ± 3·8	NS
$P_{\bar{v},O_2}$ (torr)	12	51·9 ± 5·6	40·6 ± 4·0	< 0·01
$C_{\bar{v},O_2}$ (ml 100 ml^{-1})	12	8·99 ± 1·80	7·02 ± 1·62	< 0·01
O_2 saturation$_{\bar{v}}$ (%)	8	59·6 ± 16·2	44·2 ± 16·3	< 0·01
$\dot{V}_b$(ml min^{-1} kg^{-1})	12	539·1 ± 143·5	870·0 ± 186·9	< 0·01
$\dot{V}_b/\dot{M}_{O_2}$ (ml ml^{-1})	12	32·4 ± 8·1	20·1 ± 4·2	< 0·01
E_{b,O_2}	12	0·31 ± 0·1	0·46 ± 0·1	< 0·01
f_H (min^{-1})	12	174·2 ± 16·2	327·6 ± 44·1	< 0·01
V_s (ml kg^{-1})	12	3·13 ± 0·80	2·77 ± 0·75	NS
Mean art. pressure (mmHg)	12	128·2 ± 7·3	150·1 ± 6·6	< 0·01
Total periph. resist. (PRU)	12	0·287 ± 0·082	0·194 ± 0·040	< 0·01

Statistical evaluation: the Wilcoxon matched-pairs signed-ranks test (Siegel, 1956).
NS = not significant.

taneous change in pH_a (from 7·476 to 7·488) is, however, not significant. Only small and insignificant changes are found in mixed venous P_{CO_2} and pH. Arterial O_2 tension and O_2 content remained stable during the exercise period, whereas the $P_{\bar{v},O_2}$ and $C_{\bar{v},O_2}$ were markedly reduced (Table 1), causing the $C_{a,O_2} - C_{\bar{v},O_2}$ difference ($\Delta C_{a-\bar{v}}$) to increase from 3·64 to 5·49 vol. %. The change in mixed venous oxygen content caused the blood O_2 utilization (calculated as $C_{a,O_2} - C_{\bar{v},O_2}/C_{a,O_2}$) to increase by nearly 50% (from 0·31 to 0·46 – Table 1).

The cardiac output ($\dot{V}_b$) measured by the direct Fick method ($\dot{V}_b = 100\,\dot{M}_{O_2}/(C_{a,O_2} - C_{\bar{v},O_2})$) increased by about 60% (from 539·1 to 870·0 ml min^{-1} kg^{-1}). The changes in blood oxygen content and the cardiac output during treadmill running are depicted in Fig. 1. 45% of the increase in O_2 uptake (from resting on the treadmill to running at 1·43 km h^{-1}) is supported by an increase in the arterio-venous O_2 content difference, while the rest (55%) is due to the increased cardiac output.

The change in cardiac output can in turn be divided into changes in heart rate and stroke volume (V_s). Stroke volume was calculated in each experiment by dividing the calculated $\dot{V}_b$ by the heart rate. The heart rate increased by about 88% (from 174 to 328 min^{-1}), whereas stroke volume showed a small and insignificant decrease from 3·13 to 2·77 ml kg^{-1} (a 12% decrease). In combination, these changes in f_H and v_S make up the approximate 60% increase in cardiac output. A more detailed picture of the changes in heart rate as well as the changes in mean blood pressure is giv

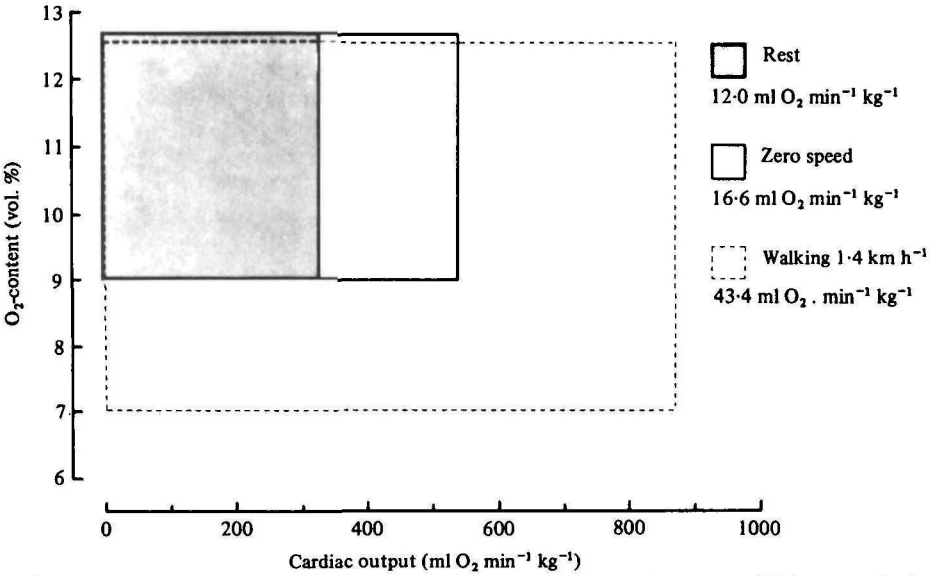

Fig. 1. Relationship between cardiac output and oxygen content in the arterial (upper value) and venous (lower value) blood at different conditions. The arterial and venous oxygen content during resting conditions has been stipulated to be the same as during resting on the treadmill ('zero speed'). The cardiac output used during rest is a mean value obtained from the literature (see text).

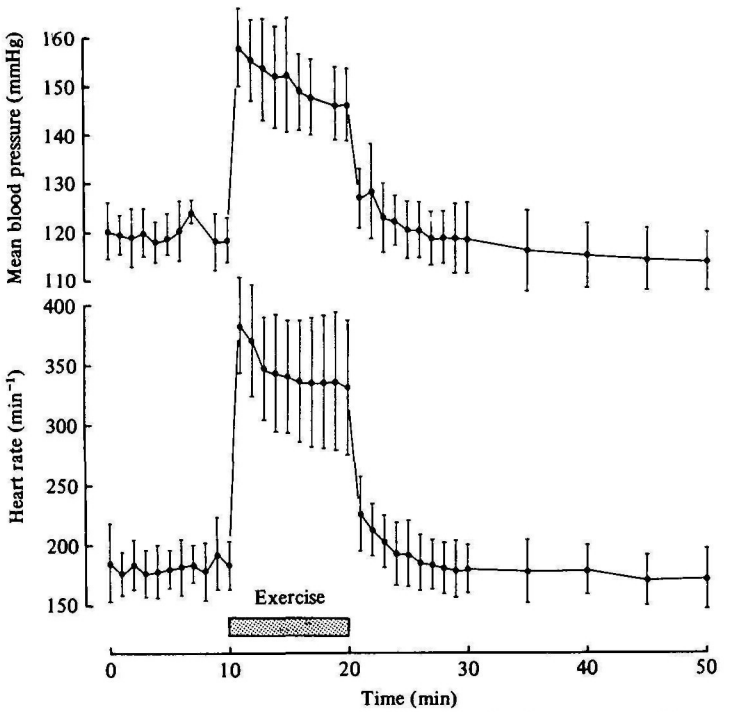

Fig. 2. Effect of exercise on mean arterial blood pressure and heart rate. Mean values ± 2 S.E.M.

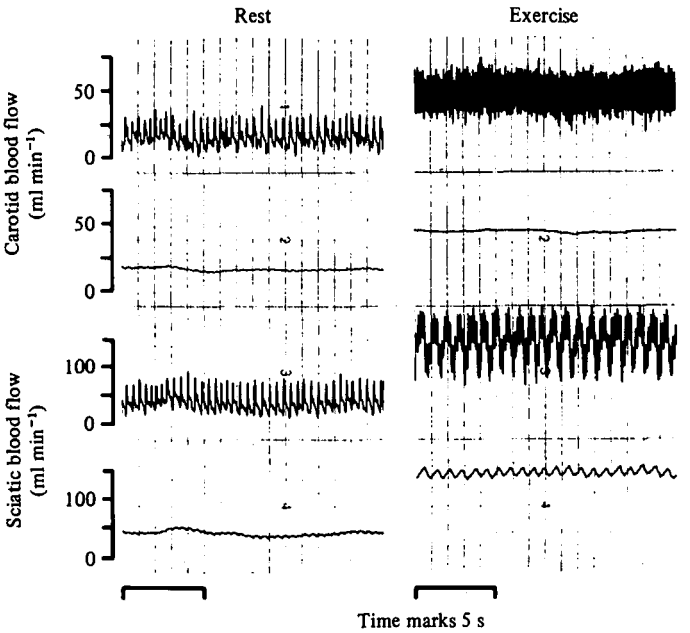

Fig. 3. Original tracings showing pulsatile and mean blood flow two minutes before exercise (rest) and in the last minute of the exercise period (exercise). The oscillations on the sciatic blood flow during exercise reflect the stride frequency.

in Fig. 2. Both parameters increased sharply at the beginning of the exercise but fell again as exercise continued and reached stable values during the last part of the exercise period.

The change in O_2 uptake during exercise was much larger than the changes in cardiac output (Table 1), causing the blood convection requirement ($\dot{V}_b/\dot{M}_{O_2}$) to decrease from 32·4 to 20·1 ml ml^{-1}. This reflects the increased O_2 utilization.

Total peripheral resistance, calculated as mean arterial pressure divided by cardiac output, showed a significant fall during exercise from $0{\cdot}89 \times 10^4$ to $0{\cdot}64 \times 10^4$ dyn s/cm^5. Often the peripheral resistance is expressed in PRU, where 1 PRU = mean arterial blood pressure/cardiac output (in ml min^{-1} kg^{-1}) (Jones & Johansen, 1972). In these units the resistance dropped from 0·29 to 0·19 PRU.

Fig. 3 shows original tracings of the blood flows obtained 2 min before start of exercise and after 9 min of exercise. Note the oscillations in the sciatic blood flow reflecting the stride frequency during running. A more detailed presentation of the changes in sciatic and carotid blood flows is given in Fig. 4. The mean values of unilateral sciatic and carotid blood flows during the 10 min pre-exercise period were 35·0 and 18·1 ml min^{-1}, respectively. At onset of exercise there was a sharp increase in both SBF and CBF reaching levels of about 120 and 40 ml min^{-1} within the first minute. The sciatic blood flow showed a tendency to increase further as exercise continued. Mean SBF for the last minute of the 10 min exercise period was hence 131·2 ml min^{-1}. The corresponding figure for CBF was 44·3 ml min^{-1}. At termination of exercise, the carotid blood flow returned to pre-exercise level within 2 min whereas the sciatic blood flow fell much slower and reached the pre-exercise lev

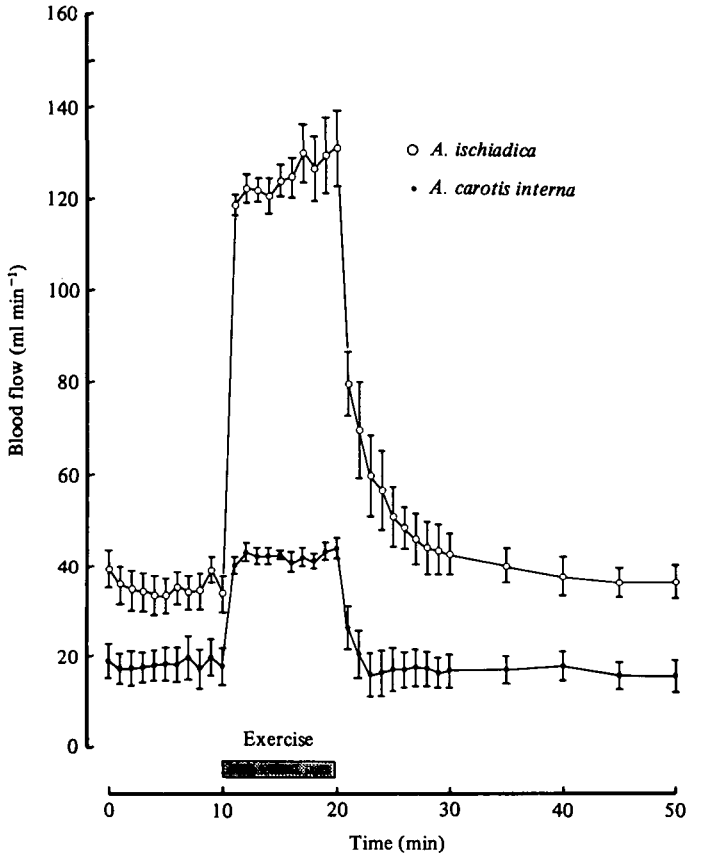

Fig. 4. Effect of exercise on unilateral sciatic (○) and carotid (●) blood flow. Mean values ± 1 S.E.M.

about 20 min after termination of exercise (Fig. 4). The mean values of blood flow in the time intervals, for which cardiac output was calculated, increased from 68·7 to 254·3 ml min^{-1} for total sciatic blood flow and from 36·5 to 82·8 ml min^{-1} for total carotid blood flow. Expressed in percentage of the cardiac output (Table 1) the SBF increased from 6·0 to 13·7% and CBF from 3·2 to 4·5% of the cardiac output.

Fig. 5 illustrates the changes in colonic, web and beak surface temperatures. T_B shows a mean increase of 0·3 °C during exercise (from 42·0 to 42·3 °C). Mean values of T_{web} and T_{beak} during the 10 min rest period were 33·1 and 28·1 °C, respectively, and increased to nearly the same value during the exercise (36·9 °C for T_{web} and 37·5 °C for T_{beak}). During the post-exercise period, there was a marked difference in the course of web and beak temperatures. Whereas beak temperature declined rather steeply at termination of exercise, falling from a mean value of 37·5 to 28·3 °C in 20 min, the web skin temperature consistently rose at termination of exercise. This transient rise from a mean temperature of 36·9 to 38·6 °C falling slowly, but remaining higher than maximum web temperature during exercise for about 6–7 min into the recovery period, correlated with a significant fall in colonic

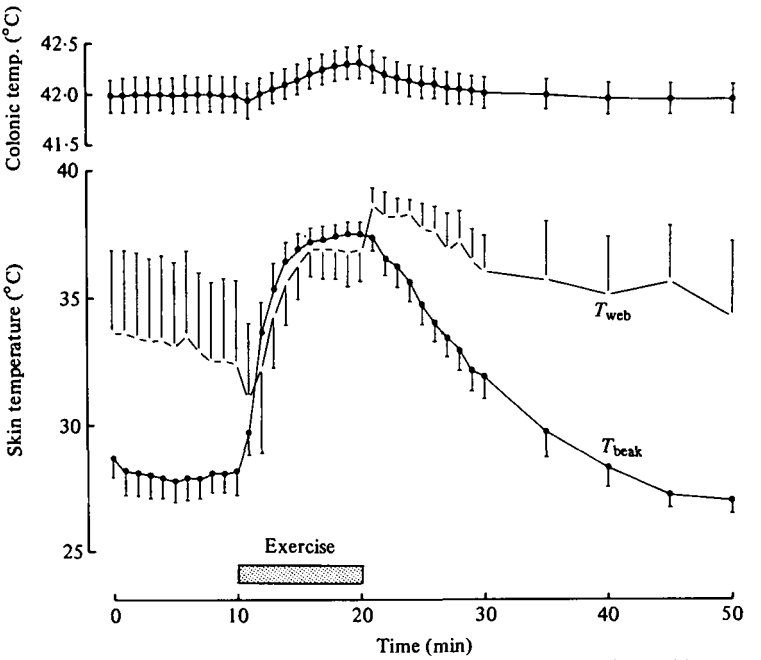

Fig. 5. Effect of exercise on colonic temperature and web and beak skin temperatures. Mean values ± 1 S.E.M.

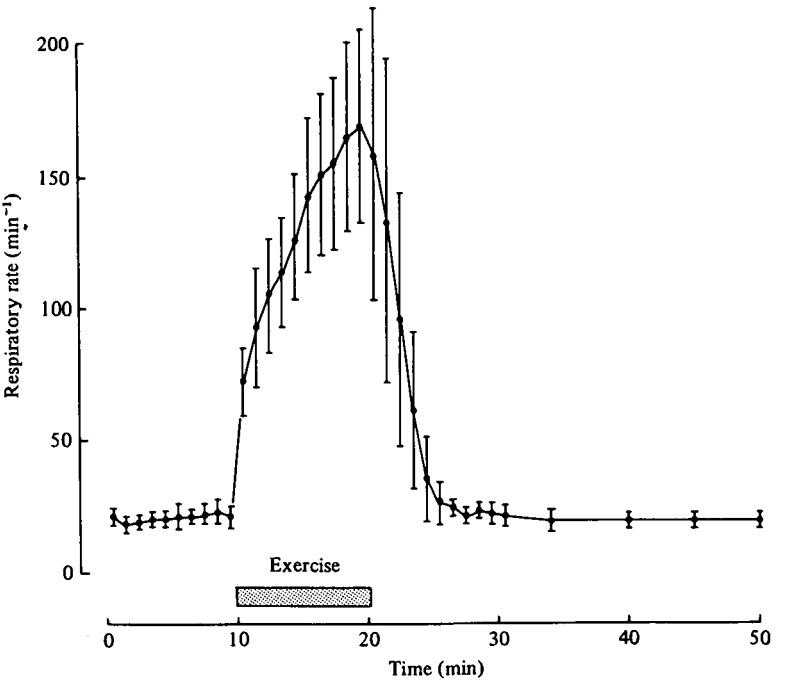

Fig. 6. Effect of exercise on the respiratory rate. Mean values ± 2 S.E.M.

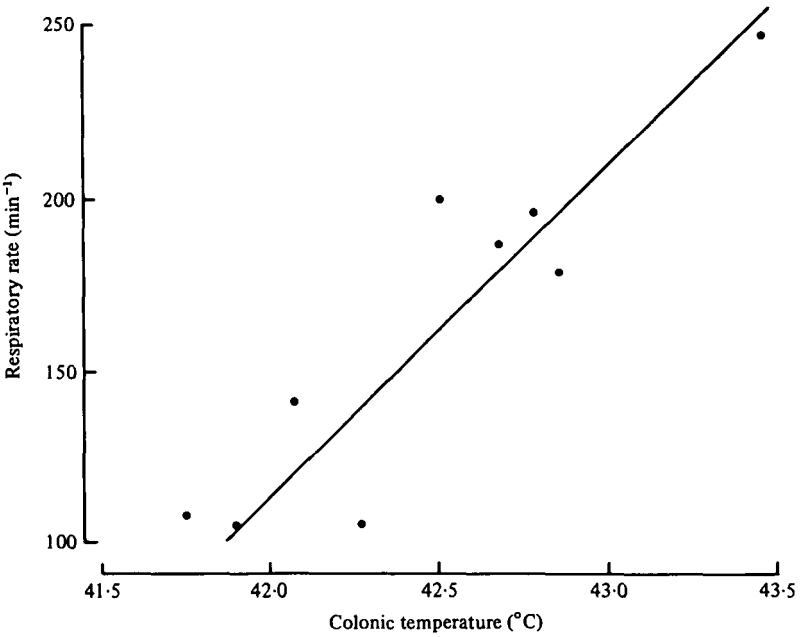

Fig. 7. Relationship between the maximum colonic temperature at the end of exercise and the simultaneous maximum breathing rate. Each point represents one experiment.

temperature from 42·3 to 42·0 °C during the same period. Subsequently, the web temperature fell slowly but remained above 35 °C 25 min into the recovery period.

During exercise the respiratory rate increased gradually throughout the running period from a pre-exercise value of 20·6 min^{-1} to a mean maximum value of 169·1 min^{-1} (Fig. 6). The maximum level reached during exercise was related to the level of colonic temperature. Fig. 7 illustrates the relationship between the maximum respiratory rate (minute 10 of the exercise period) and the colonic temperature reached at the termination of exercise. A clear relationship is evident, the maximum breathing rates correlating with high colonic temperatures. The highest values of T_B at the end of the exercise periods occurred in birds that also had higher colonic temperatures during the pre-exercise period.

DISCUSSION

Circulatory oxygen transport

The oxygen uptake ($\dot{M}_{O_2}$) measured in the Pekin duck standing on the treadmill (16·6 ml O_2 min^{-1} kg^{-1} – Table 1) exceeds the predicted value for a 2·13 kg non-passerine bird (Lasiewski & Calder, 1971) by 82% and is higher than other reported values in resting ducks (Bouverot, Hildwein & Legoff, 1974; Bouverot & Hildwein, 1978; Powell *et al.* 1978). Similar, higher than predicted, values of oxygen uptake were found in the Possum (Baudinette, Seymour & Orbach, 1978) and in the dog (Barger *et al.* 1966) when these species had been resting on a treadmill, and this has been attributed to excitement and anticipation of exercise. Also, Kiley, Kuhlmann

& Fedde (1979) reported the minute ventilation to be far higher than that predicted for Pekin ducks resting on a treadmill. The values of $\dot{M}_{O_2}$ obtained before exercise are hence not strictly comparable with other resting values for ducks, but should be considered as 'zero-speed' values (Fig. 1).

The value for the cardiac output (539 ml min^{-1} kg^{-1}) is also higher than other reported values found in resting ducks, causing the total peripheral resistance to be very low at only 0·29 PRU compared to values between 0·46 and 0·99 PRU found in ducks that were truly resting (Bech & Johansen, 1980*a*). Resting cardiac output in ducks have been reported by several authors, the mean value being 329 ml min^{-1} kg^{-1} (Bech & Johansen, 1980*b*). Using this figure and applying the arterio-venous O_2 content difference of 3·64 vol % reported here, which is probably near the true resting values (Folkow, Nilsson & Yonce, 1967; Bouverot, Doughet & Sebert, 1979), gives a resting O_2 uptake of 12·0 ml O_2 min^{-1} kg^{-1} (Fig. 1). The increased O_2 transport in the ducks standing on the treadmill is thus mainly due to a high cardiac output. The blood convection requirement is high compared with earlier reports on birds (Bech & Johansen, 1980*a*). This reflects a lower $\Delta C_{a-\bar{v}}$ in ducks.

The observed increase in oxygen uptake during exercise (2·6 fold – Table 1) is likely to be much lower than the maximal metabolic scope of ducks. Firstly, the true resting O_2 uptake is, as discussed above, lower than reported here and the resulting change in metabolic rate during treadmill walking at a speed of 1·43 km h^{-1} would more correctly be about 3·6 times the true resting value (Fig. 1). Secondly, we do not know if the present oxygen uptake during walking represents a maximum value. Kiley *et al.* (1979) found that their ducks were unable to walk at a speed higher than 2 km h^{-1}, and the highest speed at which all ducks could walk for 20 min was 1·47 km h^{-1}. The speed used here, 1·43 km h^{-1}, must therefore be close to the maximum.

The unchanged arterial O_2 tension and content during exercise in the Pekin duck means that the increased O_2 utilization is achieved by a lowering of the venous oxygen content alone (Table 1). In contrast, Millard, Johansen & Milson (1973), for penguins, and Kiley *et al.* (1979), for ducks, found that the arterial P_{O_2} was increased during treadmill exercise. Likewise, Butler, West & Jones (1977) reported an increased arterial O_2 tension during windtunnel flight in pigeons. In their study, however, there was a decrease in arterial oxygen content in spite of the increased P_{O_2} due to acidification of the blood. In mammals, the O_2 utilization during exercise is also enhanced, but only by a slight increase in arterial O_2 content and a considerable decrease in venous O_2 content (Ekelund & Holmgren, 1964; Bouverot *et al.* 1981). Gleeson, Mitchell & Bennett (1980) reported an increased arterial oxygen content during treadmill exercise also in lizards. The presently found change in $\Delta C_{a-\bar{v}}$ (from 3·64 to 5·49 vol %) represents a mean value for the whole body, and much larger differences will exist between various tissues and organs.

In order to increase the oxygen delivery during exercise either $\dot{V}_b$, $\Delta C_{a-\bar{v}}$ or both can change. The present study has demonstrated that Pekin ducks running at a speed of 1·43 km h^{-1} elevate their O_2 uptake 3·6 times above a truly resting level (Fig. 1), the increase being effected by a 51 % expansion of the arterio-venous O_2 content difference and by a 164 % increase in the cardiac output. These calculations

re based on the changes from the stipulated true resting values to exercise values. If the values obtained from the ducks when standing on the treadmill are used as resting values, the changes in $\dot{V}_b$ and $\Delta C_{a-\bar{v}}$ are more alike, being a 61% and a 51% increase, respectively.

Gleeson *et al.* (1980), working on circulatory adjustment to exercise in lizards, made a comparison between different animals to assess the employment of $\dot{V}_b$ and $\Delta C_{a-\bar{v}}$ to the increased metabolism associated with activity. In all species discussed (trout, lizard, possum, dog and man) there seemed to be an equal contribution of both parameters to meet the increased oxygen demand during exercise. The resting data used in this comparison, however, are based on values obtained pre-exercise, and hence correspond to the zero speed values in the present study. In this respect the present data for the Pekin duck conform to the scheme of Gleeson *et al.* (1980).

The estimation of a 3·6 times increase in metabolic rate during exercise (Fig. 1) is low compared to maximum metabolic scopes found in flying birds. During flight exercise the O_2 uptake can exceed the resting value by 10–12 times. Butler, West & Jones (1977) have reported on changes in $\dot{V}_b$ and $\Delta C_{a-\bar{v}}$ in flying pigeons, when the oxygen uptake reached values of 12·5 times resting. This conspicuous increase in the O_2 requirement was accompanied by an only 1·8 × increase in the arterio-venous O_2 content difference, the rest being provided by an increased cardiac output. The $\Delta C_{a-\bar{v}}$ can obviously only increase to a certain level due to the finite magnitude of the circulating O_2 pool. A further increase in oxygen delivery beyond 2–3 times must hence depend on an increase in $\dot{V}_b$. It is particularly interesting that birds often have a very high venous oxygen content at rest, and therefore retain a large O_2 potential stored in venous blood (Jones & Johansen, 1972). The mobilization of this O_2 store by a reduction in the mixed venous O_2 saturation may, however, be limited due to a requirement for a minimum diffusion gradient for O_2 from capillaries to cells in certain tissues. The inherent O_2 requirements for certain tissues will therefore correlate with high venous oxygen tensions, requiring that further increase in oxygen delivery occur by augmentation of the cardiac output.

The relative importance of heart rate and stroke volume changes for increasing the cardiac output is not similar among vertebrates. In fish, V_s plays a very important role and may increase considerably during activity (Stevens & Randall, 1967; Kiceniuk & Jones, 1977). Also in man, the stroke volume may increase during exercise (Wang, Marshall & Sheperd, 1961; Rowell, 1974). At the other extreme, V_s decreases during exercise in the Iguana (Gleeson *et al.* 1980) and in the Possum (Baudinette *et al.* 1978). In the dog, stroke volume changes only little during exercise (Barger *et al.* 1956).

Jones & Johansen (1972) reviewed the literature on cardiovascular performance during flight in birds. They argued, primarily based on data from Folkow *et al.* (1967), that the stroke volume proably will increase during vigorous exercise in birds. However, the present study showing a small decrease in stroke volume during treadmill running in ducks, and the study by Butler *et al.* (1977) on wind-tunnel flight in pigeons showing a very small increase in V_s (from 1·44 to 1·58 ml), suggest that an augmentation of stroke volume is of minor importance for increasing cardiac output during exercise in some birds. Heart rate hence becomes the foremost, if

not the sole, factor in increasing the cardiac output. Gleeson *et al.* (1980) similarly concluded that stroke volume is of minor importance in most terrestrial vertebrates (excluding man) for adjusting oxygen delivery during exercise.

Regional blood-flow changes:

The 3·7 times increase in SBF and 2·3 times increase in CBF when going from rest to exercise (Fig. 2), compared to a much smaller simultaneous increase in cardiac output (1·6×), indicates a major redistribution of the regional blood flow during exercise. In mammals (including man) a decreased blood flow to the kidney and splanchnic areas has been recorded during exercise (Rowell, 1974), but no earlier study on this subject exists for birds. The blood flow to the visceral organs (like spleen, kidney and liver) is rather high in the fowl during resting (Wolfenson *et al.* 1978) and may decrease during exercise.

Calculations based on the increase in oxygen consumption and the colonic temperature during exercise show that 18% of the added heat is stored, leaving 82% to be dissipated in the course of the 10 min exercise. The dissipation of heat occurs partly by non-evaporative routes (legs, general body surface) and partly by respiratory evaporative cooling. The observed increase in carotid blood flow of 2·3 times the resting value serves to direct heat to the evaporative surfaces, a similar response to that found in heat-stressed ducks (Bech & Johansen, 1980*b*). In accord with this an increased expired air temperature was observed during running. The use of respiratory evaporative heat loss during exercise clearly depends on the total heat load of the ducks (Fig. 7). However, there was no clear relationship between the increase in breathing rate and the increase in CBF during exercise.

It was not possible directly to distinguish between muscle and skin blood flow to the legs, but the increase in SBF probably reflects both an increased muscle and skin blood flow. An increase in peripheral blood flow to the webbed feet is also evident from the greatly increased web temperature (Fig. 5).

Immediately after cessation of exercise, the ducks are in a hyperthermic state because of heat stored during running. The dissipation of this excess heat can occur from the respiratory tract by evaporative cooling or from the legs and feet and general body surface as non-evaporative cooling. Any difference in the use of the two avenues for heat dissipiation should turn up in differences in peripheral blood flow distribution. Peripheral vascular resistance calculated for the carotid and sciatic vessels is shown in Fig. 8. Resistance was calculated as the mean arterial blood pressure divided by blood flow. A sudden increase in the carotid vascular resistance is seen to occur in the first 2–3 min after cessation of exercise, after which pre-exercise resistance is reached. The change in sciatic vascular resistance is much slower, the pre-exercise level being reached after 20–30 min (Fig. 8).

These differences suggest that the ducks preferably perfuse the skin of legs and feet emphasizing non-evaporative heat dissipation in the post-exercise period. The decrease of the colonic temperature in the period of a highly elevated web temperature (Fig. 5) also points to a very important role of the legs as sites of heat dissipation. The rise in the web temperature and the simultaneous slow decrease in sciatic blood flow at the termination of exercise suggest that opposite changes take

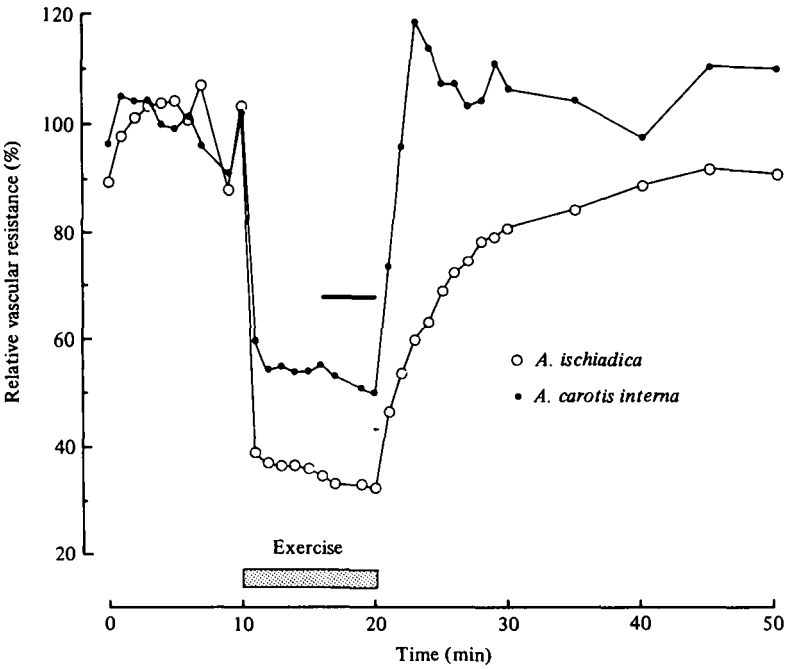

Fig. 8. Effect of exercise on the vascular resistance in the sciatic (○) and carotid (●) artery. The bar represents the value of the total vascular resistance during exercise. The resistance was obtained by dividing the mean arterial blood pressure by the blood flow (in the case of the total resistance the cardiac output was used). For a comparative purpose the mean vascular resistance in the 10 min pre-exercise period has been set to 100%.

place in the vascular beds perfused by the sciatic artery; this is apparent in a decrease in muscle blood flow reflecting a decreased muscle O_2 requirement, while an increased skin blood flow persists to effectuate a need for heat dissipation. Kluger, Gonzalez & Stolwijk (1973) similarly reported that during the post-exercise period of rabbits, the ear skin temperature greatly increased, suggesting a very important role of the ears in dissipating the stored heat.

The rapid increase in carotid resistance (Fig. 8) and decreased carotid blood flow following exercise suggest a decreased blood supply to the evaporative surfaces during the post-exercise period. This is in contrast to the situation when an internal heat load is associated with high ambient temperatures. In this case, the evaporative cooling is the primary heat-dissipating mechanism, resulting in a large increase in carotid blood flow (Bech & Johansen, 1980*b*).

After exhausting exercise, resulting in a large internal heat load and post-exercise panting (Brackenbury & Avery, 1980), there may be a different pattern in SBF and CBF. In this situation an elevated carotid blood flow in the post-exercise period will probably persist in order to direct heat to the evaporative surfaces. The post-exercise decline in carotid blood flow found in the present study is likely to reflect the normally rapid decline in the respiratory frequency (Fig. 6). Obviously, the severity of the internal heat load and the thermal environment influences the relative importance of non-evaporative and evaporative cooling.

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