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RESPIRATORY AND CARDIOVASCULAR RESPONSES OF THE EXERCISING CHICKEN TO SPINAL CORD COOLING AT DIFFERENT AMBIENT TEMPERATURES

I. CARDIOVASCULAR RESPONSES AND BLOOD GASES

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SUMMARY

1. We measured oxygen consumption ($\dot{V}_{O_2}$), heart rate (HR), stroke volume (SV), cardiac output (CO) and mean arterial blood pressure (MBPa) of chickens during 15 min treadmill exercise at $0{\cdot}5\,m\,s^{-1}$ and $0{\cdot}8\,m\,s^{-1}$ at thermoneutral (23°C), low (9°C) and high (34°C) ambient temperature (T_a); the vertebral canal was cooled to 34°C during the middle 5 min of each exercise period. P_{O_2}, P_{CO_2}, pH and oxygen content (C_{O_2}) of the arterial and mixed venous blood were also measured.

2. $\dot{V}_{O_2}$ during exercise was not significantly affected by T_a. Spinal cord cooling produced definite increases in $\dot{V}_{O_2}$, CO and SV during $0{\cdot}5\,m\,s^{-1}$ exercise at 9°C; otherwise, effects of spinal cord cooling were not significant. HR, SV and CO were all linearly related to $\dot{V}_{O_2}$; these relationships were unaffected by spinal cord cooling or T_a.

3. Blood pressure did not increase during exercise.

4. Pa_{CO_2} and $P\bar{v}_{CO_2}$ did not increase significantly during exercise. The arterial-venous C_{O_2} difference was increased by exercise only at 34°C. The chickens generally hyperventilated at 34°C T_a compared to the other T_a values. No consistent effect on blood gases or on pH and C_{O_2} of the blood could be attributed to spinal cord cooling.

INTRODUCTION

Recent studies on cardiovascular responses to bipedal exercise in the duck (Kiley, Kuhlmann & Fedde, 1979, 1982; Bech & Nomoto, 1982) and in the duck and the pigeon (Grubb, 1982), have shown that blood pressure increases during exercise, although the increases in the pigeon were not significant. The cardiac output (CO) was linearly related to oxygen consumption ($\dot{V}_{O_2}$) both in the duck and pigeon (Grubb, 1982), but the relationships between heart rate (HR) or stroke volume (SV) and $\dot{V}_{O_2}$ were more obscure. Kiley *et al.* (1979) and Bech & Nomoto (1982)

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found decreases in Pa_{CO_2} and $P\bar{v}_{O_2}$ during exercise in the ducks, but only the former reported significant changes in Pa_{O_2} (increases) and $P\bar{v}_{CO_2}$ (decreases).

$\dot{V}_{O_2}$ during exercise at different T_a and running speeds has been quantified in the quail (Nomoto, Rautenberg & Iriki, 1983): steady-state $\dot{V}_{O_2}$ was independent of T_a. Kiley *et al.* (1982) have measured blood gases, blood pressure and HR in ducks during exercise a low (−5 °C) T_a but, otherwise, the dependencies of exercising cardiovascular responses on T_a in birds are unknown.

That thermal stimulation of the spinal cord is capable of evoking thermoregulatory responses is well established in birds and mammals (see Simon, 1974 for review). For instance, spinal cord cooling in the pigeon causes immediate shivering and increases in $\dot{V}_{O_2}$ (Rautenberg, Necker & May, 1972) which are greater if T_a is low (Rautenberg, 1971). Increases in $\dot{V}_{O_2}$ caused by spinal cord cooling in the penguin are small except at low (−18 to −22 °C) ambient temperatures (Hammel *et al.* 1976). On the other hand, spinal cord cooling immediately inhibits panting caused by increased T_a in pigeons (Rautenberg *et al.* 1972), chickens (Richards & Avery, 1978) and ducks (Bech & Nomoto, 1982).

We have recently reported the cardiovascular and blood gas responses of pigeons (Barnas, Nomoto & Rautenberg, 1984*b*) and chickens (Barnas, Gleeson & Rautenberg, 1984*a*) to spinal cord cooling at different T_a. Responses in the pigeon were greater; however, in both birds, we found increases in $\dot{V}_{O_2}$, HR and CO caused by spinal cord cooling at thermoneutral T_a and further increases during spinal cord cooling at low T_a. In all cases, CO was linearly related to $\dot{V}_{O_2}$. At high T_a, CO decreased slightly during spinal cord cooling in the chicken (Barnas *et al.* 1984*b*) and pigeon (unpublished data).

In the present study, we combined the two types of stimulations of $\dot{V}_{O_2}$, exercise and cooling (both internal and external), to test whether (1) thermoregulatory stimuli could evoke significant responses during an already elevated $\dot{V}_{O_2}$, and (2) the relationship between $\dot{V}_{O_2}$ and CO would remain intact.

METHODS

Animals and experimental training

Six white Leghorn cockerels (*Gallus domesticus*), 16–19 weeks of age and weighing 1·3 ± 0·1 kg were used. The cockerels were raised outdoors in the institute's animal facilities and trained to run on a treadmill about three times per week, for 3 months, at speeds of 0·5 m s^{-1} and 0·8 m s^{-1}; training was conducted more often the week before the experiment. The treadmill was 1 m long and 14 cm wide; over it was built a 37 cm high, 55 cm long and 18 cm wide cage in which the cockerel ran. The room in which the treadmill was housed was an environmental controlled chamber. Five days prior to the first experiment with each bird, a 13 cm long loop of nylon tubing (o.d. = 0·63; i.d. = 0·50 mm) was implanted, under halothane anaesthesia, extradural and dorsal to the spinal cord into the 3rd thoracic through 4th cervical segments of the vertebral canal as described previously by Rautenberg (1969). The ends of the loop were later connected to a hot water pump in order to circulate water at desired temperatures within the loop. Additionally, a 5 cm length of nylon tubing, closed at the proximal end, was installed between the

two arms of the loop for subsequent placement of the thermocouple. The day before, or the morning of, the first experiment with each bird, cannulae were implanted under local anaesthesia (1 % xylocaine, Astra Labs) into the brachial artery and the cutaneous ulnar vein. The position of the tip of the latter in the right atrium was checked by measurement of pressure changes or autopsy after the experiment.

Experimental measurements

Temperatures of the rectum (T_{re}) and vertebral canal (T_{vc}) were measured with copper constantan thermocouples (Fine Wire, California, OUS) placed either into the rectum or inserted into the previously placed cannula in the vertebral canal. These temperatures, as well as that of the ambient air near the treadmill, were recorded on a multipoint recorder (Phillips, PM8235).

Blood pressure was measured from the brachial arterial cannula and recorded on a penwriter (Gould, Brush 220); heart rate (HR) and mean arterial blood pressure (MBPa) were calculated from these recordings. Arterial and mixed venous blood samples were measured for pH, P_{O_2} and P_{CO_2} with a Radiometer Blood Micro System (BMS3 MK2) controlled at 41 °C; all measurements were adjusted to the T_{re} recorded at the time the blood sample was taken (Severinghaus, 1965; Kiley *et al.* 1979). The oxygen contents of arterial and mixed venous blood, Ca_{O_2} and $C\bar{v}_{O_2}$ respectively, were measured with a LexO_2Con apparatus (Lexington Instrument Corp.). Haematocrit was routinely measured (average of all those samples measured was about 25 %) and did not decrease during an experiment; samples taken during the final experiment with each chicken tended to be slightly less (by about 3–4 %) than those from the initial experiment.

During the experiments, respiration was measured using a plastic helmet suspended from the top of the cage. This helmet, and respiratory measurements during exercise, will be discussed elsewhere. Gas was drawn from the helmet at a constant, measured flow rate and dried; P_{O_2} of the gas was then measured with an O_2 analyser (Beckman, OM–11).

Experimental procedure

No chicken was used in more than one experiment in a day, and complete sets of experiments with each chicken lasted about 5 days. Before each experiment, the chicken was left to sit quietly for at least 1 h on the treadmill at the experimental T_a. After positioning of the thermistors, the circulation to the vertebral thermode and the helmet, the chicken was left again to sit quietly. When steady-state measurements were observed for respiration and oxygen consumption (about 30 min later), blood was sampled simultaneously from the artery and vein. We then made resting measurements of responses to spinal cord cooling; these responses are discussed in detail elsewhere (Barnas *et al.* 1984*a*). Control measurements, excluding blood samples, were taken again immediately before exercise – about 10 min after initial control samples – and did not significantly differ from the initial controls except for minor increases in $\dot{V}_{O_2}$ at 23 °C.

The cockerels ran for 15 min at 0·5 m s^{-1}, during the middle 5 min of which the

spinal cord was cooled to 34 ± 0·8°C by opening the circulation of water through the vertebral thermode. Blood was sampled at 4, 9 and 14 min of the exercise. Another set of control samples were taken 20 min after the end of the exercise. Shortly afterwards, a second exercise period, this time at 0·8 m s^{-1}, was attempted during experiments at 23 and 9°C. However, not all cockerels completed the second 15 min exercise period.

Four birds successfully completed at least the first exercise period at T_a of 9, 23 and 34°C. One bird ran only at the high and low T_a, another bird only at 23 and 34°C. No attempt was made either to systematize or randomize the order of T_a in which a chicken ran.

Data analysis

Oxygen consumption ($\dot{V}_{O_2}$) was calculated from the corrected volume of gas (STDP) flow and P_{O_2} of the gas leaving the helmet. The difference between arterial and mixed venous C_{O_2} (ΔC_{O_2}) was used to calculate cardiac output (CO) by the direct Fick method; CO was divided by HR to give stroke volume (SV). Paired *t*-tests were performed on the differences between each measured variable caused by spinal cord cooling, exercise and changes in T_a. We also performed linear regressions of CO, SV, HR and ΔC_{O_2} on $\dot{V}_{O_2}$ at each T_a and tested (student's *t*-test, $P < 0{\cdot}05$) whether the slopes and zero-intercepts of the regressions were affected by T_a or by the inclusion of values during spinal cord cooling.

RESULTS

The responses of $\dot{V}_{O_2}$, HR, SV, CO and BP to spinal cord cooling during two levels of 15-min treadmill exercise at three different T_a are depicted in Fig. 1. The following points should be noted.

(1) Although $\dot{V}_{O_2}$ appears slightly higher during thermoneutral than low T_a at both running speeds, these differences were not significant when values from the same cockerels were compared using a paired *t*-test.

(2) $\dot{V}_{O_2}$ responses to spinal cord cooling were small compared with the responses to exercise. It is difficult to be certain that increases in some variables during the spinal cord cooling periods are the result of spinal cord cooling alone or whether they result partly from the general increases with the continuation of exercise. For example, CO during the first exercise at 23°C and $\dot{V}_{O_2}$, HR, SV and CO during exercise at high T_a increase during spinal cord cooling but remain elevated after

Fig. 1. Oxygen consumption ($\dot{V}_{O_2}$), heart rate (HR), mean arterial blood pressure (MBPa), stroke volume (SV) and cardiac output (CO) of chickens at two levels of 15 min treadmill exercise at 9°C and 23°C T_a and one level at 34°C. The spinal cord was cooled to 34°C during the middle 5 min of each exercise period (shaded areas). Open circles represent resting values. $N = 5$ at 9°C and 23°C except for the last and next to last points at 0·8 m s^{-1} where $N = 3$. $N = 6$ at 34°C T_a except for the second resting value where $N = 5$. Values for all variables, except MBPa, during each exercise period were significantly different ($P < 0{\cdot}05$, paired *t*-test) from the preceding resting value. No significant changes in MBPa were observed except that resting MBPa at 9°C was high. Other significant differences ($P < 0{\cdot}05$, paired *t*-test) are indicated by letters adjacent to S.E. bars: *b*, different from corresponding value at 23°C; *c*, different from corresponding value at 9°C; *d*, different from 4th min of exercise period; *e*, different from 14th min of exercise period.

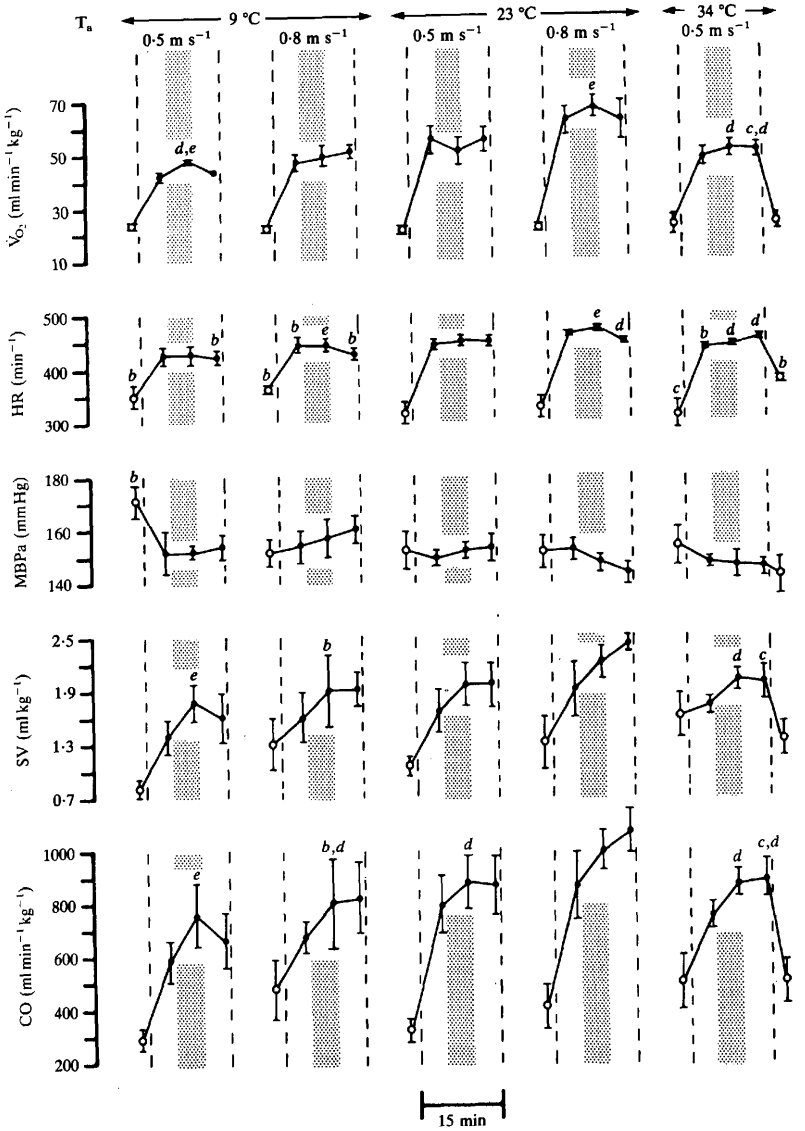
T_a
9 °C
23 °C
34 °C
0·5 m s^{-1}
0·8 m s^{-1}
0·5 m s^{-1}
0·8 m s^{-1}
0·5 m s^{-1}
$\dot{V}_{O_2}$ (ml min^{-1} kg^{-1})
70
50
30
10
HR (min^{-1})
500
400
300
MBPa (mmHg)
180
160
140
SV (ml kg^{-1})
2·5
1·9
1·3
0·7
CO (ml min^{-1} kg^{-1})
1000
800
600
400
200
15 min

Fig. 1

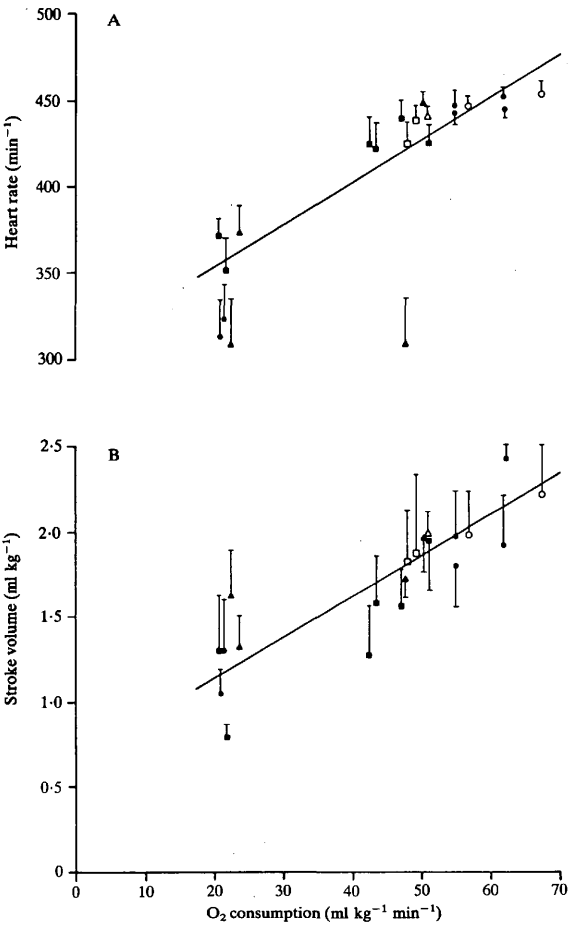

Fig. 2. The relationships of average heart rate (A) and stroke volume (B) to oxygen consumption of chickens at rest (oxygen consumption values 18–24 ml kg^{-1} min^{-1}), during exercise (closed symbols) and during spinal cord cooling during exercise (open symbols). Circles, 23°C; squares, 9°C; and triangles, 34°C. Vertical bars indicate S.E. of stroke volume and heart rate. S.E. of oxygen consumption can be seen as horizontal bars in Fig. 3. Linear regressions of the individual points are indicated by solid lines; see text for formulae.

the cooling. However, increases in $\dot{V}_{O_2}$ during the cooling were significant at the first running period at 9°C and were reversed upon termination of cooling; there were corresponding increases and decreases in CO and SV. Such changes were also observed during rest (Barnas *et al.* 1984*a*) and are most likely to represent a response to spinal cord cooling.

(3) Blood pressure was not consistently changed by exercise, spinal cord cooling or T_a, although control values at low T_a were high.

The relationships between HR and $\dot{V}_{O_2}$ and between SV and $\dot{V}_{O_2}$ are shown in Fig. 2 while that between CO and $\dot{V}_{O_2}$ is shown in Fig. 3. Points represent averages for control, exercise and spinal cord cooling at each T_a. The slopes of the linear regressions calculated for the individual data at each T_a separately were not significantly different from each other; likewise, the intercepts did not significantly change with T_a. Including the values obtained during spinal cord cooling did not significantly alter the slope or intercept of any linear regression. The linear regressions of the individual points from all T_a combined ($N = 102$) were:

$$CO = 13{\cdot}8\dot{V}_{O_2} + 100{\cdot}0;\ r = 0{\cdot}80,$$
$$HR = 2{\cdot}5\dot{V}_{O_2} + 303{\cdot}1;\ r = 0{\cdot}73,$$
$$SV = 0{\cdot}02\dot{V}_{O_2} + 0{\cdot}65;\ r = 0{\cdot}66,$$

where the units for CO and $\dot{V}_{O_2}$ were ml kg^{-1} min^{-1} and those for HR and SV were min^{-1} and ml kg^{-1}, respectively.

P_{CO_2}, P_{O_2}, pH and C_{O_2} of the arterial and mixed venous blood, and the arterial-venous C_{O_2} difference during two levels of 15 min treadmill exercise and three different T_a are shown in Fig. 4. The following points should be noted.

(1) At all temperatures, there is a general tendency for Pa_{CO_2} to decrease from control values as exercise progresses, and for $P\bar{v}_{CO_2}$ to increase slightly at the beginning of exercise and then decrease.

(2) There are significant decreases in $P\bar{v}_{O_2}$ and $C\bar{v}_{O_2}$ with exercise at 23 and 34°C; at 9°C, the tendencies are the same, though not significant.

(3) The cockerels seemed to hyperventilate slightly at 34°C compared with their responses to the lower T_a: control Pa_{CO_2} and $P\bar{v}_{O_2}$ during exercise were less than at the other T_a, while Pa_{O_2} and pH_a were generally higher.

(4) Ca_{O_2}, on the other hand, was decreased at 34°C T_a; ΔC_{O_2} at rest was also less. Only at 34°C T_a were changes (increases) in ΔC_{O_2} significant; unlike at the other T_a, ΔC_{O_2} (vol %) was significantly related to $\dot{V}_{O_2}$ at 34°C ($r = 0{\cdot}58$, $N = 29$):

$$\Delta C_{O_2} = 0{\cdot}04\dot{V}_{O_2} + 4{\cdot}17.$$

(5) No consistent effect could be attributed to spinal cord cooling.

DISCUSSION

Our linear regressions for the increases in CO with increasing $\dot{V}_{O_2}$ are steeper (larger slope, lower intercept) than those found by Grubb (1982) in either the duck or pigeon during treadmill exercise. She also found, in contrast to our findings, only

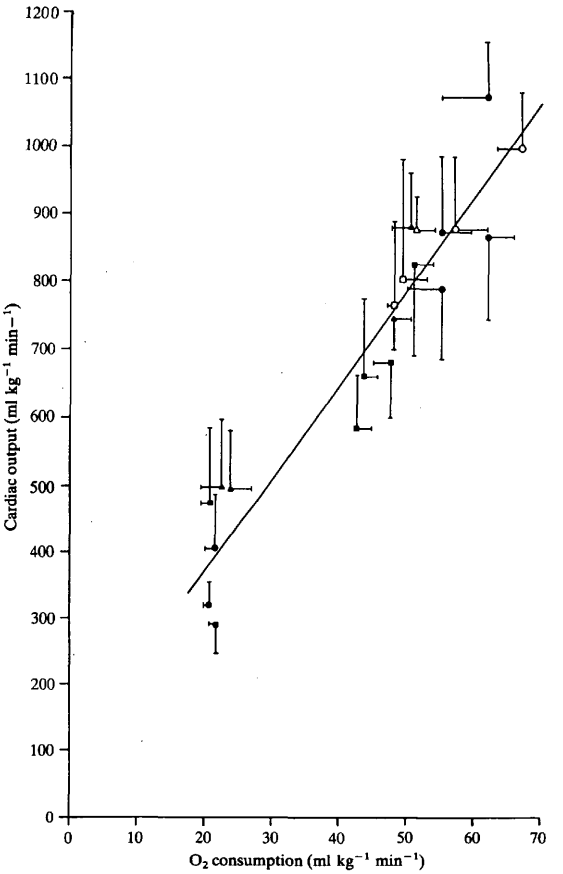

Fig. 3. The relationship between cardiac output and oxygen consumption of chickens during rest, exercise and spinal cord cooling during exercise. See Fig. 1 for explanation of symbols and N. S.E. of oxygen consumption and cardiac output are indicated by horizontal and vertical bars, respectively. Linear regression of the individual points is indicated by a solid line; see text for formula.

Fig. 4. pH, P_{CO_2}, P_{O_2} and C_{O_2} in the arterial (a) and mixed venous ($\bar{v}$) blood of chickens at two levels of 15 min treadmill exercise at T_a of 9°C and 23°C and one level at 34°C. The spinal cord was cooled to 34°C during the middle 5 min of each exercise period. See Fig. 1 for explanation of symbols and N; in addition, here a = significant difference from preceding control ($P < 0{\cdot}05$, paired t-test).

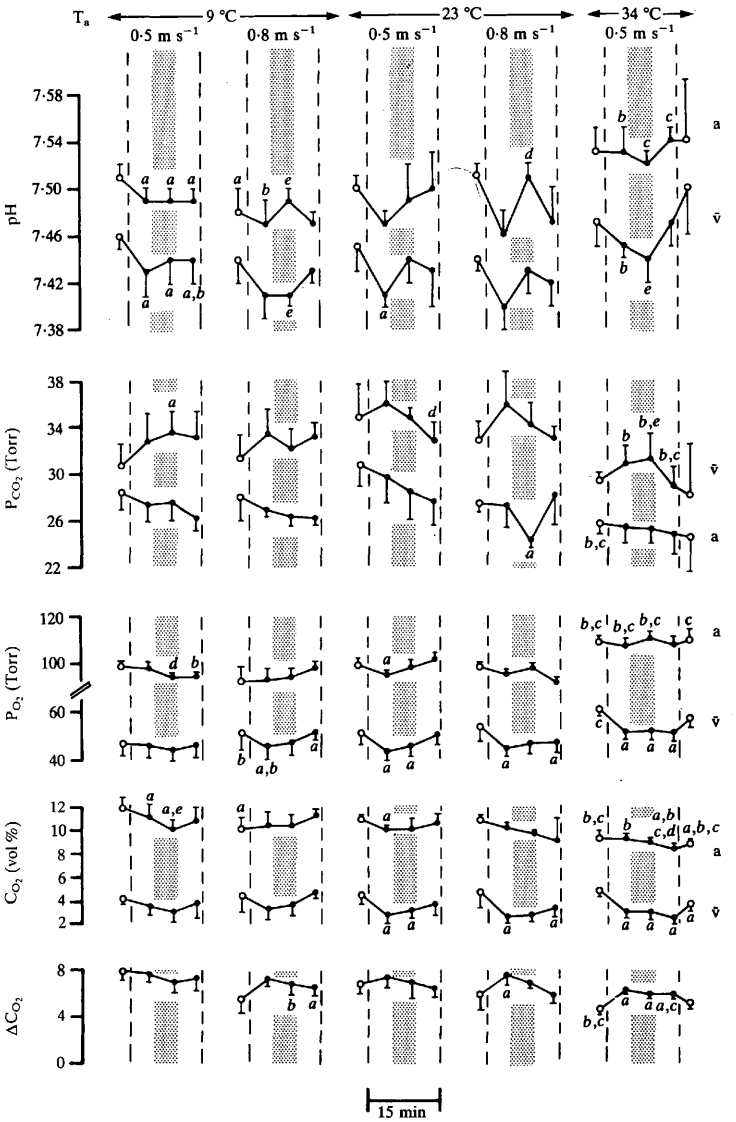
T_a
9 °C
23 °C
34 °C
0·5 m s^{-1}
0·8 m s^{-1}
0·5 m s^{-1}
0·8 m s^{-1}
0·5 m s^{-1}
pH
7·58
7·54
7·50
7·46
7·42
7·38
P_{CO_2} (Torr)
38
34
30
26
22
P_{O_2} (Torr)
120
100
60
40
C_{O_2} (vol %)
12
10
8
6
4
2
ΔC_{O_2}
8
4
0
a
v̄
15 min

Fig. 4

weak correlations between HR and $\dot{V}_{O_2}$. These may indicate some species differences. However, our regressions include more points, especially at the higher levels of $\dot{V}_{O_2}$, which would increase the accuracy of regression analysis. Thus, the differences in the calculated regressions may result from the use of a different protocol rather than a different species.

The response of SV to treadmill exercise in the duck is different from that of chickens and pigeons. Grubb (1982) found, as we did in the chicken, a significant correlation between SV and $\dot{V}_{O_2}$ in the exercising pigeon; no such correlation was observed in the duck. Bech & Nomoto (1982) report, in fact, slight decreases in SV with exercise in the duck. One possible cause for these differences may be the influence of training on cardiovascular adjustments to exercise, which is unknown in the bird. The 'domestic racing pigeons' used by Grubb (1982) were accustomed to exercise during their lifetimes. The ducks from the experiments of Grubb (1982) trained for short periods on 4–5 consecutive days before the experiment; those of Bech & Nomoto (1982) were trained for 6–8 weeks. Otherwise, it can be assumed that the ducks had no rigorous running training. Furthermore, since swimming is the most natural mode of locomotion for the species, cardiovascular performance can be expected to be poor during treadmill exercise in the duck. It may be that the intensive training undergone from a young age by the chickens in our study and the flying experience in the racing pigeons facilitated increases in stroke volume at any level of exercise, as does training in man (Blomqvist, 1983).

Exercise did not consistently affect blood pressure in our cockerels which contrasts with previous reports in ducks (Kiley *et al.* 1979; Grubb, 1982; Bech & Nomoto, 1982). Increases in blood pressure measured during treadmill exercise in the domestic racing pigeon were not statistically significant (Grubb, 1982), and the trained, flying pigeon showed no increases in blood pressure compared to rest (Butler, West & Jones, 1977). Again, either species differences or the effect of training may be involved.

There is a tendency for Pa_{CO_2} to decrease and for $P\bar{v}_{CO_2}$ to stay the same or decrease slightly during treadmill exercise in ducks (Kiley *et al.* 1979, 1982; Bech & Nomoto, 1982). During flying in the pigeon, a decrease in Pa_{CO_2} of over 10 Torr has been measured (Butler *et al.* 1977). Our results are in qualitative agreement with these findings.

The receptors responsible for mediating the large increases in respiration and cardiac output needed to effect such responses are unknown, since central and carotid chemoreceptor stimulation would lessen; moreover, Kiley *et al.* (1982) and Kiley & Fedde (1983) have shown that neither intrapulmonary chemoreceptivity nor changes in body temperature are solely responsible for the hyperpnea during exercise. In mammals, it is also unclear as to which receptors are responsible for effecting cardiovascular and respiratory responses during exercise, although much evidence suggests that increased discharge frequency of group III and IV afferents from the muscles due to metabolic changes may be important (Whipp, 1981). In the present study, temperature stimulation by neither ambient nor spinal cord cooling had independent effects on CO, HR or SV; that is, the increases in these variables depended only on $\dot{V}_{O_2}$. We suggest, therefore, that the cardiovascular changes measured in the present study resulted from stimulation of muscle afferents due to

increased metabolism, although the effects of movement on these afferents cannot be ruled out.

$\dot{V}_{O_2}$ was not significantly different for a given level of exercise at different T_a. This is not surprising since Nomoto *et al.* (1983) found similar results in the quail, and independence of exercising $\dot{V}_{O_2}$ from T_a has often been observed in man (Astrand & Rodahl, 1970; Rowell, 1974). This implies that thermoregulatory responses of $\dot{V}_{O_2}$ to T_a were small compared to the elevated $\dot{V}_{O_2}$ during exercise. However, the exercising chicken is capable of responding to thermal stimulation of the spinal cord at 9°C T_a (Fig. 1). The increment of $\dot{V}_{O_2}$ during the spinal cord cooling probably resulted from shivering, although we were not able to record EMG from the muscles during running. Nomoto *et al.* (1984) have recently given evidence that shivering can be evoked by spinal cord cooling during exercise in the pigeon. Shivering can also occur during exercise at T_a of 10°C in man (Hong & Nadel, 1979); this shivering is suppressed by increased exercise intensity. Whether shivering could be evoked during exercise in birds if T_a and exercise intensity are low enough has not been studied and warrants future investigation.

That the increase in $\dot{V}_{O_2}$ at 23°C when the spinal cord was cooled during exercise was not significant in the present study is not unexpected since the response during rest was also small at 23°C (Barnas *et al.* 1984*a*). The lack of any blood gas changes evoked by spinal cord cooling was also observed at rest.

The chickens generally showed decreases in Pa_{CO_2} and $P\bar{v}_{CO_2}$, and increases in pH_a and Pa_{O_2} at the high T_a compared to the other T_a. This is probably due to hyperventilation (i.e. a decrease in oxygen extraction from the inhaled air) in response to thermoregulatory needs.

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