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THE EFFECTS OF WALKING ON HEART RATE, VENTILATION RATE AND ACID–BASE STATUS IN THE LOBSTER *HOMARUS AMERICANUS*

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Summary

American lobsters *Homarus americanus* were exercised on an underwater treadmill at speeds from 1.7 to 8 m min^{-1} to determine the effects of exercise on heart rate, ventilation rate and acid–base status. Heart and ventilation rates showed almost instantaneous increases at the start of exercise, but the magnitude of the increase was not related to speed. Maximum heart rate was approximately 80–90 beats min^{-1} and maximum ventilation rate was 175–180 beats min^{-1} at all speeds tested.

Exercise at all speeds caused a decrease in haemolymph pH, with the acidosis after exercise at 8 m min^{-1} being significantly greater than at the other three speeds. Concomitant with this acidosis was a large increase in partial pressure of carbon dioxide, with the largest increase occurring after exercise at 8 m min^{-1}. The concentration of lactate in the haemolymph increased to similar levels at all speeds of walking. Davenport analysis indicates that the acidosis was predominantly respiratory in nature.

Although it was anticipated that heart and ventilation rates would show increases proportional to the speed of exercise, this was not the case. Rather, the responses were fixed regardless of walking speed. The reason for this phenomenon remains unexplained.

Key words: lobster, *Homarus americanus*, locomotion, acid–base status, heart, scaphognathite.

Introduction

If the aerobic demands of the tissues of a lobster are to be met, the heart and scaphognathites (SGs) must be able to coordinate their activities to maintain an effective supply of oxygen (Wilkens, 1976, 1995). The SGs and heart must maintain an adequate flow of water and haemolymph, respectively, to maintain the diffusion gradient across the gills into the haemolymph, and the heart must distribute the oxygenated haemolymph to the rest of the body. Indeed, the heart and SGs display parallel alterations in activity in response to many stimuli (Wilkens *et al.* 1985).

Exercise is a stress that will produce an increased oxygen demand by the tissues that must be met in order to sustain aerobic metabolism. One important way to increase the supply of oxygen to the tissues is to elevate the heart and ventilation rates during periods of increased activity. For the heart, the frequency of contraction is determined by the bursting pattern of the nine cells of the cardiac ganglion. This rate is modulated by the cardioregulatory nerves of the central nervous system that reach the heart in the paired dorsal nerves, which travel in the connective tissue above the posterior-dorsal alary ligaments before entering the heart itself. Each nerve contains two accelerator axons and one inhibitory axon (Alexandrowicz, 1932). Stimulation of the cardioaccelerator nerves will increase heart rate, while stimulation of the cardioinhibitory nerves will slow or stop the heart (Florey, 1960; Field and Larimer, 1975; Wilkens and Walker, 1992). Heart rate is also modulated by the hormones of the pericardial organ (Wilkens and McMahon, 1992).

At the onset of exercise, the heart rate of crabs and lobsters jumps from its pre-exercise resting value to a new elevated value (Hamilton and Houlihan, 1992; Guirguis and Wilkens, 1995). This increase in heart rate occurs in less than 1 min, and in lobsters once the new level is reached there is little to no fluctuation during a 30 min walk.

The scaphognathites (SGs), located at the anterior ends of the branchial chambers, are also important in maintaining an oxygen supply to the tissues (Pasztor, 1968; Wilkens and McMahon, 1972). Their function is to pump water over the gills so that oxygen can be extracted and carried to the tissues in the haemolymph. The rhythmic pattern of movement is generated by paired oscillatory networks located in each half of the suboesophageal ganglion (Mendelson, 1971). In spite of this separate innervation, the two SGs behave as though they receive a common input (Wilkens and Young, 1975). In addition, interneurones originating from the central nervous system provide coordinating control of both the heart and the SGs (Wilkens *et al.* 1974).

If sufficient oxygen is not delivered, anaerobic metabolism must be utilized. Past studies have demonstrated that many crustaceans respond to exercise with a reduction in

haemolymph pH that is due both to increased CO_2 production and to a build up of lactic acid. Thus, crustaceans typically show both respiratory and metabolic acidosis (McDonald *et al.* 1979; Wood and Randall, 1981*a*,*b*; Full and Herreid II, 1984; Full, 1987; Morris and Greenaway, 1989; Hamilton and Houlihan, 1992). By measuring the acid–base status of *H. americanus* at rest, and during exercise and recovery at different walking speeds, the ability of these animals to meet their metabolic requirements, either aerobically or anaerobically, can be assessed.

Guirguis and Wilkens (1995) examined the effect of a single speed of walking (1.7 m min^{-1}) on heart rate. The goal of the present study was to determine the effect of walking at different speeds on heart and ventilation rates and on metabolic processes. It was anticipated that heart and ventilation rates would be correlated with the intensity of exercise and that as exercise became more strenuous anaerobic metabolism would become more important.

Materials and methods

American lobsters *Homarus americanus* (Milne-Edwards), weighing between 550 and 650 g, were obtained from a local supplier and held in a tank of artificial aerated sea water at 14 °C. The animals were fed twice a week.

Exercise protocol

Specimens were cooled in ice and prepared for experimentation by implantation of impedance electrodes around the heart and scaphognathites through holes drilled in the carapace. Electrodes were held in place using dental latex and cyanoacrylate adhesive. Another hole was drilled over the pericardial sinus lateral to the heart for the purpose of sampling post-branchial haemolymph. Pre-branchial haemolymph samples were drawn from the joint of one of the walking legs. Following surgery, all animals were allowed to recover for 48 h prior to any experimentation. This was enough time to allow heart and ventilation rates to be completely stable at resting levels.

The animals were exercised on a variable-speed underwater treadmill consisting of a sanding belt passed around a motor-driven tensioning drum. The treadmill was surrounded by aerated sea water at 14 °C, and the sides of the apparatus were covered with black plastic to minimize visual stimuli.

The lobsters were left in the treadmill overnight to allow them to become settled. The resting heart and ventilation rates were measured prior to any other testing, and blood samples (pre-branchial from the joint of a walking leg and post-branchial from the pericardial sinus) were drawn to determine pH, the partial pressure of oxygen (P_{O_2}), the concentration of carbon dioxide and the concentration of lactate before exercise. The partial pressure of carbon dioxide (P_{CO_2}) was calculated using the Henderson–Hasselbach equation and the measured values of pH and the concentration of carbon dioxide.

Once resting measurements had been completed, the treadmill was turned on at a predetermined speed (1.7, 2.4, 4.4 or 8 m min^{-1}) and the animal was exercised for 30 min. To maintain position in the treadmill, animals were tethered *via* the rostrum. The tether was anchored outside the treadmill. In no way did it interfere with the animal's ability to walk nor did it suspend the animal in the water in any way. The tether forced the animal to walk and prevented it from drifting to the back of the treadmill, much like walking a dog on a leash. Heart and ventilation rates were monitored throughout the exercise period and during 30 min of recovery. Blood samples were taken at the end of the 30 min exercise period and at the end of 30 min of recovery.

The effects of haemolymph sampling were tested by taking samples at the same intervals outlined above in animals that remained at rest.

Determination of haemolymph pH, P_{O_2} and concentration of CO_2

Pre-branchial and post-branchial haemolymph samples were drawn using iced, gas-tight, 500 µl glass syringes and analyzed immediately. Sample volume ranged from 400 to 450 µl.

The partial pressure of oxygen was measured using a Radiometer oxygen electrode held at 14 °C in a thermostatted water jacket. The electrode was calibrated to zero with a solution of sodium sulphite and to 19 kPa with air-saturated sea water.

Haemolymph pH was measured using a Radiometer BMS 3MK2 blood micro system at 14 °C.

The concentration of carbon dioxide (mmol l^{-1}) was determined either using the method of Cameron (1971) or with a Capni-Con model 5 total CO_2 analyzer. The results obtained using these two methods were not statistically different (P>0.05).

The partial pressure of CO_2 (in mmHg; 1 mmHg= 133.332 Pa) was calculated using the Henderson–Hasselbach equation as follows:

$$P_{CO_2} = \frac{T_{CO_2}}{\alpha(1 + 10^{pH-pK})} ,$$

where total CO_2 (T_{CO_2}) is in mmol l^{-1}, α is the solubility coefficient of CO_2 (0.051 mmol l^{-1} $mmHg^{-1}$) and pK=6.04.

Lactate measurements

Two different methods were used to determine haemolymph lactate concentrations. The first method was an enzymatic analysis of haemolymph samples using the Sigma diagnostics lactate kit no. 826-uv. This involved mixing the haemolymph sample with lactate dehydrogenase and a glycine buffer (modified for lobster haemolymph by adding enough EDTA to make a 10 mmol l^{-1} solution and adjusting to pH 9 using hydrochloric acid). The samples were incubated in a water bath at 37 °C for 15 min and then analyzed spectrophotometrically (at 340 nm) on a Bausch and Lomb spectrophotometer.

Lactate was also analyzed using a YSI model 27 lactate analyzer. This machine was calibrated with 5 mmol l^{-1} and 15 mmol l^{-1} lactate standard solutions (YSI) and blood samples

were injected into the machine using a YSI no. 2361 syringepet. Results were consistent between the two methods of analysis.

Buffering capacity of haemolymph

To determine the buffering capacity of haemolymph, a 4–5 ml haemolymph sample was drawn from the pericardial sinus and centrifuged at 13 000 ***g*** for 10 min to remove the clot. The supernatant was transferred to a round-bottomed tonometer mounted on a wrist-action shaker and held in a water bath (at 15 °C). Haemolymph was equilibrated to different partial pressures of CO_2 ranging from approximately 0.5 to 5.5 mmHg (1 mmHg=133.332 Pa) using a Wösthoff gas-mixing pump. T_{CO_2} (mmol l^{-1}) and pH were measured following 30 min of equilibration at each P_{CO_2} tested. These data were used to create a Davenport diagram. The measured values of T_{CO_2} and pH were plotted against each other, with the slope of this line being equal to the buffering capacity of the haemolymph in mmol CO_2 l^{-1} pH $unit^{-1}$ (slykes).

Statistics

Statistical analysis was carried out using SigmaStat, a statistical software package. A combination of *t*-tests, paired *t*-tests and Mann–Whitney rank sum tests were utilized. In all instances, a 5 % level of significance was employed.

Results

Walking behaviour

Lobsters were forced to walk at specified speeds because of the tether. As mentioned above, the tether did not affect the animals except to prevent them from drifting to the back of the treadmill and not walking. At the lower speeds (1.7 m min^{-1}, 2.4 m min^{-1}), there was a greater likelihood that the animals would attempt to crawl out of the treadmill apparatus. Therefore, not all the walking legs were necessarily used for walking at all times. At speeds of 4.4 m min^{-1} and 8 m min^{-1}, the animals were more likely to use all their walking legs during the exercise period. At all speeds, the chelae were held up off the surface, the tail was extended and the swimmerets were beating.

Table 1. *Data showing the effects of treadmill speed on frequency of leg movements in* Homarus americanus

Treadmill speed (m min^{-1})	Frequency of leg movements (cycles min^{-1})
1.7	19.2±2.4
2.4	31.8±2.0
4.4	43.5±3.1
8	59.8±3.2

Values are means ± S.E.M. of legs from both sides of the body.
Treadmill speed and the frequency of leg movements were significantly correlated; r^2=0.94, P<0.05; N=4.

To confirm that increasing the treadmill speed actually caused the animals to walk faster, animals were video-taped and the frequency of leg movements was determined for each speed. The average frequencies of leg movements from both sides of the body are presented in Table 1. There was a significant correlation between treadmill speed and the frequency of leg movement (r^2=0.94; P<0.05).

Although the treadmill is able to operate at speeds greater than 8 m min^{-1}, the lobsters would not exceed this speed: increasing the treadmill speed beyond this level caused the animals to drag and attempt to tail-flip. They were more inclined to let themselves drag on the treadmill than try to walk at these speeds. The lobsters did not reach the point of exhaustion (the point at which the animal is not able to walk any longer) in a 30 min walk at any of the speeds tested up to 8 m min^{-1}.

Sampling effects

Tests were performed to evaluate the effect of haemolymph sampling on otherwise undisturbed animals. Typically, heart rate and ventilation rate showed small increases when

Table 2. *Mean heart rate, ventilation rate and acid–base response following 30 min of exercise in the lobster* Homarus americanus

		Exercise			
	Rest	1.7 m min^{-1}	2.4 m min^{-1}	4.4 m min^{-1}	8 m min^{-1}
Heart rate (beats min^{-1})	60.0±2.4	80.4±4.9*	87.0±1.7*	79.2±3.5*	85.5±2.9*
Ventilation rate (beats min^{-1})	100±12.0	174±7.1*	181.5±3.8*	174±6.1*	177±3.9*
pH	7.78±0.03	7.63±0.04*	7.64±0.03*	7.66±0.04*	7.42±0.01*,[a]
P_{O_2} (kPa)	6.20±0.87	8.53±0.73*	9.01±0.65*	9.44±0.61*	9.50±0.47*
T_{CO_2} (mmol l^{-1})	4.80±0.44	–	5.51±0.53*	–	5.74±0.59*
P_{CO_2} (Pa)	268.0±41.0	–	507.2±50.2*	–	603.5±84*,[a]
Lactate concentration (mmol l^{-1})	0.20±0.02	1.67±0.26*	1.55±0.19*	1.49±0.33*	1.29±0.26*

An asterisk indicates values that are significantly different from rest.
[a] indicates that the value at 8 m min^{-1} is significantly different from the corresponding values at the slower speeds.
Blood chemistry values are from post-branchial haemolymph.
Values are means ± S.E.M. (N=5–7).

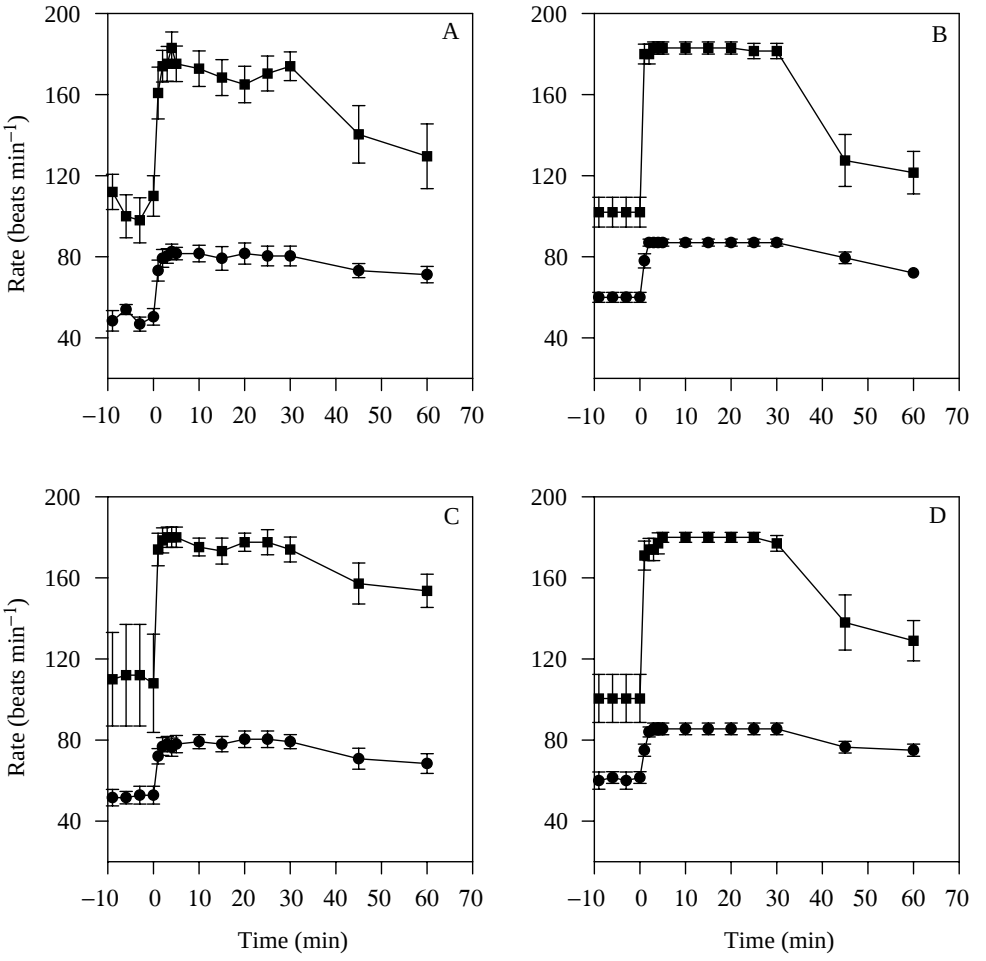

Fig. 1. The effects of walking on heart rate (circles) and ventilation rate (squares) in *Homarus americanus*. A, B, C and D represent treadmill speeds of 1.7, 2.4, 4.4 and 8 m min^{-1} respectively. The treadmill was started at 0 min and stopped at 30 min. All values after 0 min are significantly different from resting values. Ventilation rates are the mean value for the two scaphognathites, which always beat at approximately the same rate. Values are means ± S.E.M. (N=5).

haemolymph was taken, but these increases lasted less than 5 min and were not statistically significant. Blood chemistry variables were also quite stable and were unaffected by the process of removing haemolymph. There were no significant differences in pH, P_{O_2}, T_{CO_2}, P_{CO_2} or lactate levels in comparison with resting animals.

Heart and ventilation rate responses

The responses of heart rate and ventilation rate were virtually identical at all walking speeds. Heart rate increased from approximately 60 beats min^{-1} to approximately 80–90 beats min^{-1}, and ventilation rate increased from approximately 100 beats min^{-1} to 175–180 beats min^{-1} (Table 2; Fig. 1). The maximum values for heart rate and ventilation rate did not differ significantly among the speeds tested. After 30 min of recovery, heart and ventilation rates remained significantly elevated relative to rest at all walking speeds (Table 3; Fig. 1).

The increases in heart rate and ventilation rate were almost instantaneous with the onset of exercise (Fig. 2). Rates always stabilized within 1–2 min of the start of exercise.

Acid–base response

Changes in pH and lactate levels were similar at the three lower speeds (Table 2). There was a tendency for P_{O_2} to increase as walking speed was increased; however, the differences were not statistically significant. The acidosis that resulted from exercise at 8 m min^{-1} was significantly greater

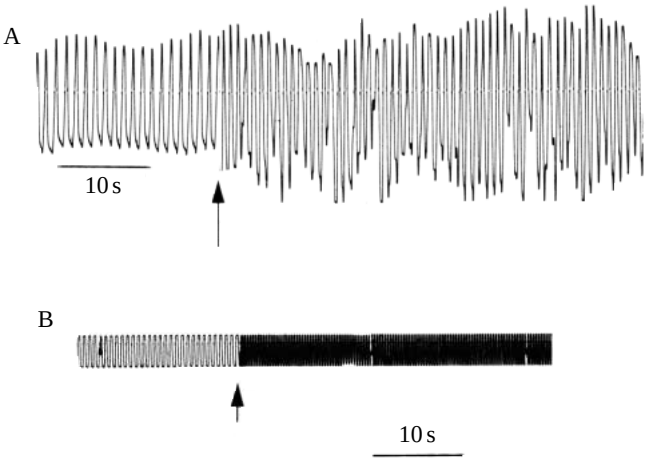

Fig. 2. Sample traces of heart rate (A) and ventilation rate (B) in *Homarus americanus*. Arrows indicate the start of exercise. Note the rapidity with which the rates increase at the start of exercise.

Table 3. *Mean heart rate, ventilation rate and acid–base response following 30 min of recovery from exercise in the lobster* Homarus americanus

		Exercise			
	Rest	1.7 m min^{-1}	2.4 m min^{-1}	4.4 m min^{-1}	8 m min^{-1}
Heart rate (beats min^{-1})	61.5±2.9	71.2±4.0*	72.0±0*	68.4±4.9*	75.0±3.0*
Ventilation rate (beats min^{-1})	100.0±12	129.6±15.9*	121.5±10.5*	153.6±8.2*	129.1±9.9*
pH	7.78±0.03	7.71±0.04	7.73±0.02	7.74±0.04	7.54±0.02*,[a]
P_{O_2} (kPa)	6.20±0.87	6.91±0.6	5.70±0.3	5.92±0.6	9.36±1.4
T_{CO_2} (mmol l^{-1})	4.80±0.44	–	4.26±0.23	–	4.71±0.4
P_{CO_2} (Pa)	268.0±41.0	–	278.1±10.2	–	373.2±39.9
Lactate concentration (mmol l^{-1})	0.20±0.02	1.49±0.18*	0.73±0.15*	1.54±0.43*	1.01±0.26*

An asterisk indicates values significantly different from rest.
[a] indicates that the value at 8 m min^{-1} is significantly different from the corresponding values at the slower speeds.
Blood chemistry data are from post-branchial haemolymph samples.
Values are means ± S.E.M. (N=5–7).

than at the lower speeds. The greatest P_{CO_2} increase occurred after exercise at 8 m min^{-1}, while lactate was 1.29 mmol l^{-1} at this speed.

The changes in acid–base status at all four speeds were significantly different from those at rest. The animals that were walked at 8 m min^{-1} had previously been walked at 2.4 m min^{-1} several days earlier. In these individuals, pH and P_{CO_2} showed significantly greater changes at 8 m min^{-1} compared with 2.4 m min^{-1}. The changes in P_{O_2} and lactate concentration were not significantly different between the two speeds.

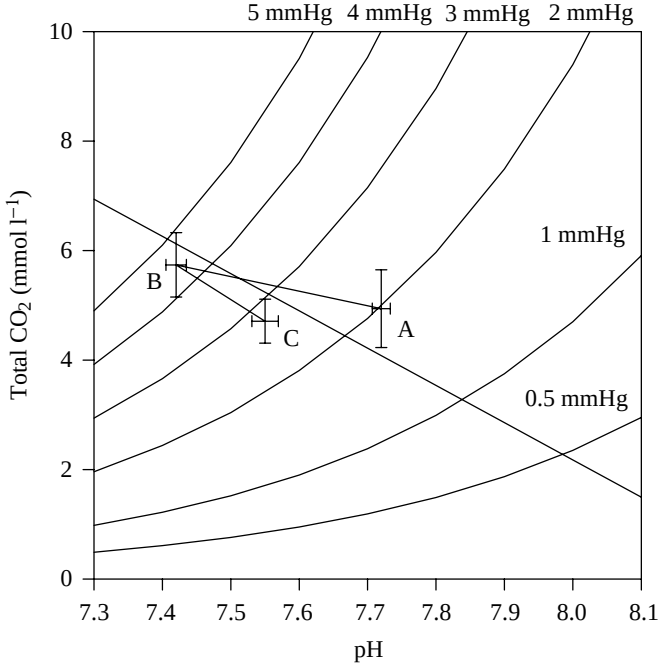

Fig. 3. Davenport diagram for *Homarus americanus* haemolymph. Curved lines are P_{CO_2} isopleths. The straight line is the haemolymph buffering capacity (−6.8 mmol CO_2 l^{-1} pH unit^{-1}). A, rest; B, after 30 min of exercise at 8 m min^{-1}; C, after 30 min of recovery from exercise at 8 m min^{-1}. See text for further details. 1 mmHg=133.332 Pa. Values are means ± S.E.M. (N=5).

Data from pre-branchial haemolymph samples are not shown. Post-branchial oxygen tension was approximately 30 % higher than pre-branchial P_{O_2} throughout rest, exercise and recovery. There were no significant differences between pre-branchial and post-branchial values for pH, T_{CO_2}, P_{CO_2} and lactate levels.

The acid–base status following 30 min of recovery from exercise is shown in Table 3. Haemolymph lactate concentrations were significantly greater than pre-exercise values following 30 min of recovery from exercise at all speeds. Haemolymph pH remained significantly depressed relative to the resting value only after exercise at 8 m min^{-1}. The other blood chemistry variables were not significantly different from rest after 30 min of recovery.

Fig. 3 is a Davenport diagram (Davenport, 1974) used to relate changes in T_{CO_2} (mmol l^{-1}) to changes in pH at different partial pressures of carbon dioxide. The curved lines are the P_{CO_2} isopleths calculated from the Henderson–Hasselbach equation (α=0.051 mmol CO_2 l^{-1} mmHg^{-1}; pK=6.04; T=15 °C). The straight line represents the buffering value of the haemolymph (−6.8 mmol CO_2 l^{-1} pH unit^{-1}). The fact that the data points basically follow the buffering line indicates that the acidosis produced from walking is due predominantly to changes in CO_2 levels in the blood.

Discussion

Heart and ventilatory responses

The heart and ventilation rate responses to exercise were similar at all walking speeds. No significant changes with walking speed were evident in either variable. Furthermore, the changes in heart rate and ventilation rate were highly correlated (r^2=0.94). Wilkens *et al.* (1974) showed that the heart and SGs receive a common input from higher levels of the central nervous system, which might account for the simultaneous increases in heart and ventilation rates.

The absence of a graded response to increased walking speed is not due to a lack of ability to vary the heart and

ventilation rates with exercise. Work in our laboratory has shown that, under conditions of severe cardiac impairment (achieved by cutting the regulatory nerves to the heart and the alary ligaments of the heart), ventilation rate can peak at rates higher than those reported here in intact animals (R. A. Rose, J. L. Wilkens and R. L. Walker, unpublished data). Similarly, heart rate has been reported to exceed those levels recorded here when cardiac function is impaired or when hormones are administered (Guirguis and Wilkens, 1995). Thus, the fact that heart rate and ventilation rate responded similarly at all the speeds tested in this study cannot be attributed to physical limitations of the structures and tissues themselves. Although they are capable of beating at faster rates, they simply do not do so.

It is apparent from Figs 1 and 2 that the heart and SGs respond to exercise immediately and with a fixed, constant response. While the mechanism of such a response in the lobster is not known, it clearly does not include sensory input regarding the intensity of exercise. This new and unexpected result cannot be explained at this time.

Contrary to the 'on' response seen at the onset of exercise, recovery from exercise is a longer process that probably depends on the level of stress caused by the walk. Recovery of both heart and ventilation rates can take as long as 8–12 h in ectothermic animals (McMahon, 1981). The time constants of recovery for heart and ventilation rates in this study also indicate that recovery is an extended process. For heart rate, the time constants are 23 min at 2.4 m min^{-1} and 30 min at 8 m min^{-1}. The values for ventilation rate are 10 min and 12 min, respectively. It is possible that the recovery of heart and ventilation rates is driven by the metabolic consequences of the exercise period.

Metabolic response

Since the heart and ventilation rate responses seem to be fixed, it is possible that increased walking speed would have metabolic consequences. The responses to exercise at 1.7, 2.4 and 4.4 m min^{-1} were not significantly different, suggesting that these speeds were within the aerobic capacity of these animals. This is further indicated by low levels of lactate. Anaerobic metabolism did not seem to play much of a role at these speeds.

The reduction in pH after exercise at 8 m min^{-1} was much more severe than after walking at lower speeds, yet lactate production was still low. This is in stark contrast to past studies on crustacean exercise physiology that have shown far more substantial increases in lactate levels. For example, Wood and Randall (1981*b*) found that lactate concentration peaked at 5.5–7.5 mmol l^{-1} after exhaustive exercise in the land crab *Cardisoma carnifex*. Even submaximal treadmill exercise at 5.8 m min^{-1} in *Carcinus maenas* resulted in accumulation of lactate to levels of almost 4 mmol l^{-1} (Hamilton and Houlihan, 1992). Thus, the levels of lactate measured in this study seem low compared with other studies, even taking into account that we did not exercise the lobsters to the point of exhaustion.

Davenport analysis (Davenport, 1974) shows that the acidosis produced by walking in *H. americanus* is almost entirely respiratory in nature (Fig. 3). This means that anaerobic metabolism is utilized only to a small degree. While many crustaceans show a mixed (respiratory and metabolic) acidosis (McDonald *et al.* 1979; Smatresk *et al.* 1979; Smatresk and Cameron, 1981; Wood and Randall, 1981*a*,*b*; Wheatly *et al.* 1986; Hamilton and Houlihan, 1992), it seems that *H. americanus* shows a predominantly respiratory acidosis. This means that aerobic respiration is the main route of energy production during exercise at these intensities and under these conditions.

Another factor must be considered, however. The best way to determine lactate concentration is by whole-body analysis, because lactate can become compartmentalized in the tissues of the animal and be under-represented in the haemolymph (Full and Herreid II, 1984). However, this procedure was not feasible in this study because it requires the animals to be killed for each determination. Furthermore, measurement of haemolymph lactate levels may not be a completely accurate indicator of anaerobic metabolism since the time course of release of lactate from the muscles is not clear. Booth and McMahon (1985) report a discrepancy between haemolymph lactate and muscle lactate levels in the blue crab *Callinectes sapidus*. At all points throughout exercise and recovery, muscle lactate levels were higher than haemolymph lactate levels. Thus, the contribution of lactate to the acidosis and the portion of metabolism carried out anaerobically may be underestimated in the present study.

Compensation for exercise

The ventilatory activity of the SGs is oxygen-sensitive in water breathers (Dejours, 1981). It has been suggested that crustaceans possess an internal sensor for monitoring the oxygen tension of post-branchial blood (Ishii *et al.* 1989; Zinebi *et al.* 1990). This was thought to be a means of controlling both heart rate and ventilation rate by feedback inhibition (Larimer, 1964). Crayfish (*Astacus leptodactylus*) possess chemoreceptors whose activity increases with a decrease in oxygen tension (Ishii *et al.* 1989). These receptors are localized in the wall of the branchiocardiac veins carrying post-branchial haemolymph and could therefore monitor the oxygen tension in the blood. Similarly, Zinebi *et al.* (1990) concluded that peripheral oxygen-sensitive chemoreceptors are present in the ventral anterior region of the arterial system near the SGs in the crab *C. maenas*. Another possibility is that there are oxygen sensors on the gills themselves. Such oxygen-sensitive elements have been characterized on the gills of *Limulus polyphenus* (Page, 1973; Crabtree and Page, 1974) and could be important in determining the heart and ventilation rates during recovery from exercise by responding to decreased haemolymph oxygen tension, but their activity would not predict exercise onset.

While some crustaceans (*Cancer magister*, *C. carnifex*) do indeed show reductions in haemolymph P_{O_2} after exercise (McMahon, 1981), this was not the case for *H. americanus* in the present study, which showed an increased oxygen tension

after a 30 min walk (Table 2). Also, the increase in ventilation rate occurs within seconds after exercise begins, while changes in P_{O_2} occur over the order of minutes; therefore, even if sensors for detecting decreasing oxygen tension are present in *H. americanus*, some other mechanism must account for the changes in ventilation rate at the start of the walking period.

While heart rate and ventilation rate respond to the onset of exercise with a sharp increase, there is no correlation between these variables and exercise intensity during the exercise period. However, other mechanisms could meet the increased oxygen demand. McMahon (1995) suggests that changes in the stroke volume of the heart may be more variable than heart rate responses in *H. americanus* and may depend on the level of activity. This makes sense since increasing the heart rate too much would be counterproductive if there were not enough time between beats for adequate filling of the heart.

Increasing the stroke volume of the heart would result in greater delivery of oxygen to the muscles. A similar argument can be made for ventilation volume (the volume of water pumped over the gills). Increasing the ventilation volume would enable more oxygen to be extracted per beat of the scaphognathites. Preliminary work on measuring ventilation volume and oxygen consumption has shown that ventilation volume increases as the speed of walking increases (R. A. Rose, J. L. Wilkens and R. L. Walker, unpublished data). This response has also been demonstrated in other crustaceans (Herreid II *et al.* 1979). Whether ventilation volume is directly correlated with exercise intensity in the lobster remains to be determined.

One other source of compensation could be from the pattern of walking. As mentioned previously, the animals are less likely to utilize all the walking legs at the slower speeds. At these speeds, some of the legs are typically used for 'feeling' the surroundings rather than for locomotion. At $8\,m\,min^{-1}$, however, all the legs usually contribute to the locomotion of the animal. Increasing the number of walking legs being used may divide the work more evenly between the available muscles. However, increasing the number of muscles used may result in an increase in the oxygen demand and thus have a greater metabolic impact on the animal.

In conclusion, the hypothesis that heart rate and ventilation rate would show increases in proportion to increases in exercise must be rejected. Heart and ventilation rates showed fixed responses to exercise regardless of speed. Animals that walked at $8\,m\,min^{-1}$ showed significantly greater decreases in pH that were due predominantly to a respiratory acidosis. P_{CO_2} was significantly greater at the highest exercise intensity ($8\,m\,min^{-1}$). Haemolymph lactate levels showed similar, low-level increases for all exercise speeds tested. The reason why there is no gradation in heart rate and ventilation rate responses to exercise is not clear.

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