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Effect of muscle temperature on rate of oxygen uptake during exercise in humans at different contraction frequencies

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Summary

The effect of elevated human muscle temperature on energy turnover was investigated during cycling exercise (at 85 % of $\dot{V}_{O_2max}$) at a contraction frequency of 60 revs min^{-1}. Muscle temperature was passively elevated prior to exercise by immersion of the legs in a hot water bath (42 °C). During exercise at this low pedalling rate, total energy turnover was higher (P<0.05) when muscle temperature was elevated compared with normal temperature (70.4±3.7 *versus* 66.9±2.4 kJ min^{-1}, respectively). Estimated net mechanical efficiency was found to be lower when muscle temperature was elevated. A second experiment was conducted in which the effect of elevated human muscle temperature on energy turnover was investigated during cycling exercise (at 85 % of $\dot{V}_{O_2max}$) at a contraction frequency of 120 revs min^{-1}. Under the conditions of a high pedalling frequency, an elevated muscle temperature resulted in a lower energy turnover (P<0.05) compared with the normal muscle temperature (64.9±3.7 *versus* 69.0±4.7 kJ min^{-1}, respectively). The estimated net mechanical efficiency was therefore higher when muscle temperature was elevated. We propose that, in these experiments, prior heating results in an inappropriately fast rate of cross-bridge cycling when exercising at 60 revs min^{-1}, leading to an increased energy turnover and decreased efficiency. However, at the faster pedalling rate, the effect of heating the muscle shifts the efficiency/velocity relationship to the right so that cross-bridge detachment is more appropriately matched to the contraction velocity and, hence, energy turnover is reduced.

Key words: contraction velocity, temperature, cycling exercise, human, efficiency/velocity relationship.

Introduction

Temperature is widely recognised as an important determinant of skeletal muscle function (for reviews, see Bennett, 1984; Rall and Woledge, 1990; Ranatunga, 1998). One effect of heating muscle is to alter the force/velocity relationship both in humans (Binkhorst et al., 1977; De Ruiter and De Haan, 2000; He et al., 2000) and in other mammals (Ranatunga, 1982, 1984). Maximum contraction velocity (V_{max}) and the curvature of the force/velocity relationship (a/P_0) are most sensitive to changes in temperature, such that V_{max} increases and a/P_0 decreases at higher temperatures, which is a consequence of the temperature-dependence of myofibrillar ATPase (mATPase) activity (e.g. Stienen et al., 1996; He et al., 2000). Since it has been observed that specific types of muscle fibre with different force/velocity characteristics have distinct efficiency/velocity relationships (Reggiani et al., 1997; He et al., 2000), it might be inferred that an effect of muscle temperature on the force/velocity characteristics, and mATPase activity, would necessarily change the efficiency/velocity relationship.

Surprisingly, the effect of a change in muscle temperature on the efficiency of muscle contraction in humans *in vivo* has received little attention. This is despite the fact that the *in vivo* temperature of human skeletal muscles can vary over a wide range depending on environmental conditions and the metabolic heat liberated in the muscle itself (e.g. Asmussen and Bøje, 1945; Saltin et al., 1968). Indeed, it is common practice for athletes to perform warming-up exercises prior to training or competition. However, the effect of muscle temperature on the mechanical efficiency of exercise in humans may be velocity-specific since it would imply a shift in the efficiency/velocity relationship analogous to that previously reported for the power/velocity relationship for human exercise (for a review, see Sargeant, 1999).

The purpose of the present study was therefore to

determine whether an increase in muscle temperature affected energy turnover and estimates of net mechanical efficiency during sustained dynamic exercise in humans. It was hypothesised that the effect of increasing muscle temperature on energy turnover would be dependent on contraction frequency. It has previously been speculated that the optimum pedalling frequency for efficiency of the type I fibres during cycling might be approximately 60 revs min^{-1} (see Sargeant, 1999). Any temperature-related shift in the efficiency/velocity relationship towards the right would mean that exercise at a low pedalling frequency of approximately 60 revs min^{-1} would be on the ascending limb of the relationship. Thus, energy turnover would be greater for the same mechanical output. Conversely, a fast pedalling rate would be on the descending limb of the efficiency/velocity relationship, and any temperature-related rightwards shift would lead to an increase in efficiency and, hence, energy turnover for a given mechanical output would be expected to decrease.

We have tested this hypothesis (i) by estimating the energy turnover during cycling at 60 revs min^{-1} at normal muscle temperature and following passive heating and (ii) by estimating the energy turnover at normal and elevated muscle temperature when cycling at 120 revs min^{-1}, this latter pedalling rate being chosen as the fastest pedalling rate that could be reliably sustained.

Materials and methods

Subjects

Six healthy male subjects (age 23±3 years, height 180.5±6.8 cm, mass 72.3±6.9 kg; means ± S.E.M.) volunteered for the study. All subjects participated in some form of recreational physical exercise but none was specifically trained. The subjects were fully informed of the purposes and associated risks of the study and gave their written, informed consent. The study was approved by the Manchester Metropolitan University Ethics Committee.

Pre-experimental procedures

Each subject performed two tests to determine the maximum rate of oxygen uptake ($\dot{V}_{O_2max}$), one at 60 and the other at 120 revs min^{-1}, using a multi-stage protocol on a friction-braked cycle ergometer (Monark 814, Varberg, Sweden). Each stage lasted for 3 min, and expired air was analysed during the last minute of each stage. From the relationship between power output and $\dot{V}_{O_2}$, an external power output equivalent to 85 % of $\dot{V}_{O_2max}$ was calculated for each contraction frequency. All experiments were performed at this intensity, which was chosen to enable exercise to be sustained while eliciting a high $\dot{V}_{O_2}$ so that small changes in $\dot{V}_{O_2}$ would be measurable. In addition, it should be noted that this intensity is also typical of that during prolonged endurance events when small changes in efficiency may critically affect performance. Prior to the experimental trials, each subject performed at least one habituation trial to familiarise himself with the experimental protocol.

Experimental protocol

The subjects arrived at the laboratory in the morning following an overnight fast. In the normal temperature condition, subjects rested for 30 min at normal room temperature (20–22 °C; quadriceps muscle temperature approximately 36 °C at 3 cm depth) (Sargeant, 1987). In the heated temperature condition, muscle temperature was increased by immersing the legs, up to the gluteal fold, in a water bath at 42 °C for 30 min. On these occasions, the subjects exited the water bath, briefly towelled dry and put on their shoes before mounting the cycle ergometer (this typically took less than 2 min). In a parallel study using the same heating protocol, muscle temperature, measured by a needle thermistor (Ellab, Copenhagen, Denmark), was found to be elevated by 2.4±0.2 °C immediately prior to the commencement of exercise compared with normal temperature conditions. In parallel experiments, it was also shown that the effect of exercise was to increase muscle temperature by a further 0.5 °C in the 'heated' conditions and by 3.5 °C in control conditions. This indicates a convergence of muscle temperature towards the end of the exercise period, although there was still a significantly higher temperature in the pre-heated trials ($P<0.05$).

Immediately after temperature manipulation or the 30 min rest period, the exercise trial began. This consisted of a 3 min rest period whilst seated on the cycle ergometer, after which subjects performed 3 min of unloaded cycling followed immediately by a 6 min period of cycling at the predetermined power output equivalent to 85 % of $\dot{V}_{O_2max}$ at 60 or 120 revs min^{-1}. After the exercise bout, there was a final 5 min period of rest seated on the ergometer. Pulmonary gas exchange was continuously measured throughout the exercise trial. Blood samples for the determination of blood lactate concentration were collected prior to leg warming, immediately before exercise and at 0.5, 1.5, 3 and 5 min post-exercise.

The experiments at normal and heated muscle temperatures and at 60 and 120 revs min^{-1} were performed, in randomised order, on separate occasions with at least 4 days between trials. The pulmonary gas exchange data were averaged between two repeated trials. Blood samples were obtained on only one occasion for each condition.

Metabolic measurements

Expired air was sampled continuously for percentage CO_2 and O_2 content and volume on a breath-by-breath gas-analysis system (2900 Metabolic Measurement Cart, Sensormedics, Netherlands). This was calibrated with gases of known concentration and a 3 l syringe immediately prior to each testing session. Breath-by-breath $\dot{V}_{O_2}$ data were computed and then averaged over each minute of sampling for each trial. The coefficient of variation for repeated $\dot{V}_{O_2}$ measurements was 3.4 %.

Arterialised venous blood samples (Forster et al., 1972) were taken *via* an indwelling butterfly needle (21G) inserted into a superficial vein on the dorsal surface of the hand following immersion in hot water (42 °C) for a minimum of 10 min. The

needle was kept patent by regular flushing with heparinised sterile saline. Blood samples (2.5 ml) were mixed thoroughly with EDTA (3 mg ml^{-1}). From these samples, duplicate aliquots (100 µl) of whole blood were immediately deproteinised in 1 ml of ice-cold perchloric acid (2.5 %) and stored at –20 °C for later analysis. The concentration of blood lactate was determined fluorimetrically using the supernatant from the deproteinised blood (Maughan, 1982). The coefficient of variation for duplicate samples was 3.9 %.

Calculations

Aerobic energy turnover (kJ min^{-1}) was calculated using the respiratory exchange ratio (RER) and rate of oxygen uptake. Net $\dot{V}_{O_2}$ was calculated by subtracting resting from exercise $\dot{V}_{O_2}$, with the exercise $\dot{V}_{O_2}$ averaged over the final 3 min of exercise. Anaerobic energy turnover (kJ min^{-1}) was calculated on the assumption that 1 mmol l^{-1} of post-exercise blood lactate accumulation yields the equivalent of 3.3 ml O_2 kg^{-1} (for a review, see Di Prampero and Ferretti, 1999). Net blood lactate accumulation was calculated as the difference between the peak post-exercise concentration and the corresponding resting lactate concentration. Total energy turnover (kJ min^{-1}) was calculated as the sum of aerobic energy turnover and anaerobic energy turnover. Total mechanical power output (W) was calculated as the sum of the external power delivered to the cycle ergometer plus the estimated 'internal' power output. Internal power output (W kg^{-1}) was estimated as 0.153(frequency)^3, where frequency is in Hz (Minetti et al., 2001). Net mechanical efficiency (%) was defined as the ratio between total mechanical power output (W converted to kJ min^{-1}) and the total rate of energy turnover.

Statistical analyses

Data were analysed by either paired *t*-tests or two-way (temperature and time) analysis of variance (ANOVA) with repeated measures, where appropriate. When a significant effect was detected, differences were located with *post-hoc* paired *t*-tests. Significance was accepted at $P<0.05$. Data are presented as means ± S.E.M. (N=6).

Results

$\dot{V}_{O_2max}$ did not differ between 60 and 120 revs min^{-1} (3.86±0.19 *versus* 3.88±0.18 l min^{-1}, respectively). Thus, 85 % of $\dot{V}_{O_2max}$ under both conditions was calculated to be 3.28±0.20 l min^{-1} for 60 revs min^{-1} and 3.30±0.15 l min^{-1} for 120 revs min^{-1}. Despite $\dot{V}_{O_2max}$ being the same, there was an upward shift in the $\dot{V}_{O_2}$/power output relationship when cycling at 120 revs min^{-1} at submaximal workloads (Fig. 1). Thus, at 85 % of $\dot{V}_{O_2max}$, the external power set on the ergometer was 236±9 W at 60 revs min^{-1} and 170±12 W at 120 revs min^{-1} ($P<0.05$). The $\dot{V}_{O_2}$/power relationship output shown in Fig. 1 indicates a marked difference in the *y*-intercepts extrapolated to zero power. The greater *y*-intercept at 120 revs min^{-1} reflects the added cost of internal work due to the increase in the number of leg movements per minute in addition to the energy cost required to overcome frictional losses in the ergometer transmission.

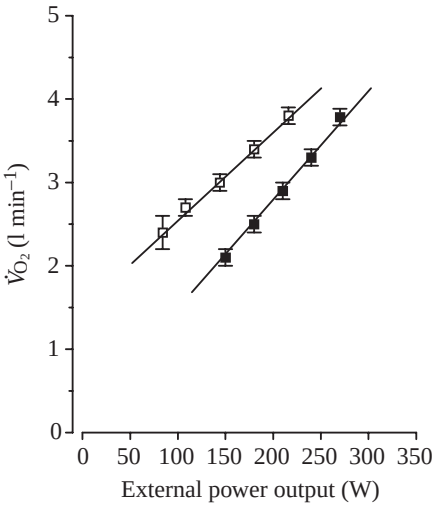

Fig. 1. $\dot{V}_{O_2}$/power output relationship during the incremental multi-stage exercise test at contraction frequencies of 60 revs min^{-1} (filled squares) and 120 revs min^{-1} (open squares), respectively. Regression coefficients were 0.995 in both instances. Values are means ± S.E.M. (N=6).

Pulmonary $\dot{V}_{O_2}$

The effects of heating the legs in the water bath was to increase the $\dot{V}_{O_2}$ of exercise by 0.15 l min^{-1} when pedalling at 60 revs min^{-1} compared with the control ($P<0.05$; Figs 2, 3; Table 1). In contrast, when pedalling at 120 revs min^{-1}, the

Table 1. *Respiratory and blood lactate data at rest and at the end of cycling exercise at 60 and 120 revs min^{-1} under conditions of normal and elevated muscle temperature*

	60 revs min^{-1}		120 revs min^{-1}	
	Normal	Elevated	Normal	Elevated
Rest				
$\dot{V}_{O_2}$ (l min^{-1})	0.34±0.04	0.37±0.02	0.33±0.04	0.35±0.08
$\dot{V}_{CO_2}$ (l min^{-1})	0.26±0.04	0.30±0.02	0.25±0.02	0.28±0.01
RER	0.78±0.02	0.82±0.03	0.76±0.02	0.80±0.03
[Lactate] (mmol l^{-1})	0.63±0.04	0.82±0.08	0.52±0.06	0.54±0.04
Exercise				
$\dot{V}_{O_2}$ (l min^{-1})	3.25±0.14	3.40±0.18*	3.35±0.20	3.22±0.16*
$\dot{V}_{CO_2}$ (l min^{-1})	3.39±0.14	3.51±0.20	3.44±0.21	3.31±0.18*
RER	1.04±0.01	1.03±0.02	1.02±0.01	1.02±0.01
[Lactate] (mmol l^{-1})	6.40±0.49	8.08±0.56*	6.08±0.90	4.88±0.48

$\dot{V}_{O_2}$, rate of oxygen uptake; $\dot{V}_{CO_2}$, rate of carbon dioxide release; RER, respiratory exchange ratio ($\dot{V}_{CO_2}/\dot{V}_{O_2}$); [Lactate], peak concentration of blood lactate.

Significant differences ($P<0.05$) between normal and elevated muscle temperature are indicated by an asterisk.

Values are means ± S.E.M., N=6.

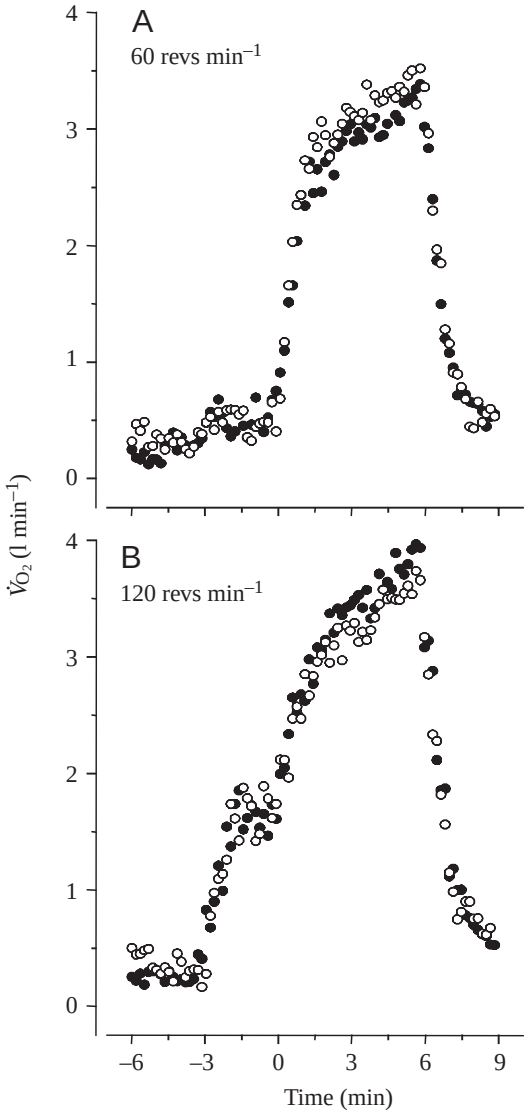

Fig. 2. Continuous measurement of pulmonary $\dot{V}_{O_2}$ in one subject during exercise at 60 (A) and 120 revs min^{-1} (B) under conditions of normal (filled circles) and increased (open circles) muscle temperature. Data points are breath-by-breath values averaged over 10 s.

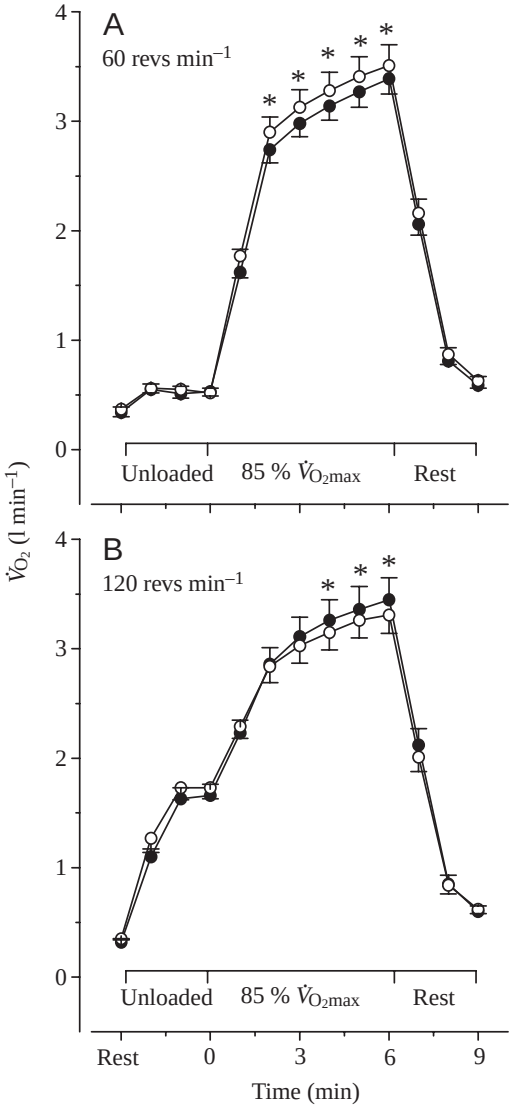

Fig. 3. Pulmonary $\dot{V}_{O_2}$ during exercise at 60 (A) and 120 revs min^{-1} (B) under conditions of normal (filled circles) and increased (open circles) muscle temperature. Data points are breath-by-breath values averaged over 1 min. Values are means ± S.E.M. (N=6). Difference (P<0.05) between normal and elevated muscle temperature are indicated by an asterisk.

effect of the same prior hot water immersion protocol was to decrease the exercise $\dot{V}_{O_2}$ by 0.13 l min^{-1}, compared with the control (P<0.05; Figs 2, 3; Table 1).

Blood lactate levels

Consistent with the changes in pulmonary $\dot{V}_{O_2}$, the effect of heating the legs was to increase (at 60 revs min^{-1}) or decrease (at 120 revs min^{-1}) the concentration of blood lactate by less than 2 mmol l^{-1} (P<0.05; Fig. 4).

Energy turnover and mechanical efficiency

The estimated total rate of energy turnover (aerobic+anaerobic; see Materials and methods) at 60 revs min^{-1} was 5.2 % higher (P<0.05) when the legs were heated compared with the normal condition. In contrast, at 120 revs min^{-1}, the converse was observed, with a 5.9 % decrease (P<0.05) in energy turnover (Table 2). At 60 revs min^{-1}, the total rate of energy turnover (aerobic+anaerobic; see Materials and methods) was higher when the legs were heated compared with the control (P<0.05). Since the total mechanical power (internal and external components) remained the same at 247 W, the estimated net mechanical efficiency decreased by 1 % (P<0.05) following passive heating. Thus, a 1 % decrease in absolute terms would represent a relative decrease in the apparent net mechanical efficiency of 5 %. At 120 revs min^{-1}, the total rate of energy turnover (aerobic+anaerobic; see Materials and methods) was lower when the legs were heated compared with the control

Table 2. *Energy turnover and estimates of net mechanical efficiency during cycling exercise at 60 and 120 revs min^{-1} under conditions of normal and elevated muscle temperature*

	60 revs min^{-1}		120 revs min^{-1}	
	Normal	Elevated	Normal	Elevated
Aerobic energy turnover (kJ min^{-1})	62.0±2.3	64.4±3.5	64.2±4.0	61.2±3.4*
Anaerobic energy turnover (kJ min^{-1})	4.8±0.4	6.0±0.4*	4.8±0.8	3.7±0.4
Total energy turnover (kJ min^{-1})	66.9±2.4	70.4±3.7*	69.0±4.7	64.9±3.7*
Total power output (W)	247±10	247±10	259±14	259±14
Mechanical efficiency (%)	22.2±0.3	21.2±0.4*	22.7±0.6	24.0±0.5*

See Materials and methods for calculations.

Significant differences (P<0.05) between normal and elevated muscle temperature are indicated by an asterisk.

Values are means ± S.E.M., N=6.

(P<0.05). With the total mechanical power (internal and external components) remaining the same at 259 W, the estimated net mechanical efficiency increased by just over 1 % (P<0.05) following passive heating. In this case, a 1 % increase in absolute terms would represent a relative increase in net mechanical efficiency of just over 5 %.

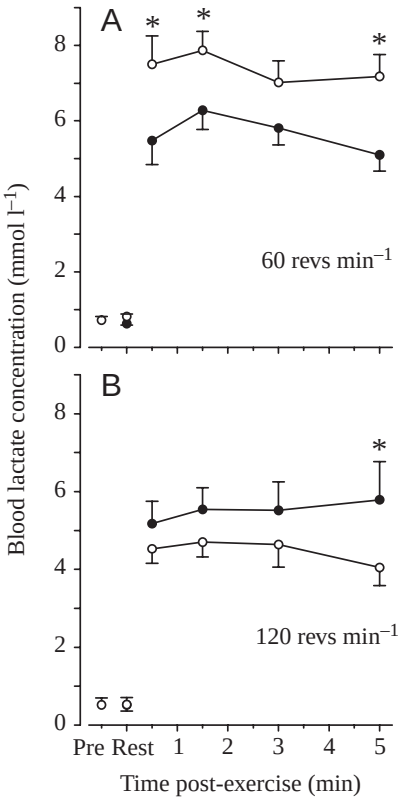

Fig. 4. Concentration of blood lactate before hot water immersion (Pre), immediately before exercise (Rest) and post-exercise at 60 (A) and 120 revs min^{-1} (B) under conditions of normal (filled circles) and increased (open circles) muscle temperature. Values are means ± S.E.M. (N=6). Difference (P<0.05) between normal and elevated muscle temperature are indicated by an asterisk. Note that the data points at Pre and Rest are superimposed.

Discussion

The present investigation demonstrated that passively increasing the temperature of the exercising muscle prior to sustained dynamic exercise significantly alters energy turnover and estimates of net mechanical efficiency during exercise. Furthermore, these alterations are influenced by the contraction frequency at which the exercise is performed.

During exercise at 60 revs min^{-1}, energy turnover was greater when muscle temperature was elevated. This observation is perhaps not surprising since the main determinant of total energy turnover for muscle contraction is the cost of cross-bridge cycling. It is to be expected that the rate of cross-bridge cycling will increase with an elevated muscle temperature since, as with other enzymatic processes, myofibrillar ATPase activity is temperature-dependent (see Stienen et al., 1996; He et al., 2000). Indeed, in isometric contractions in humans, the economy of muscle contraction had previously been reported to decrease, as shown by an elevated ATP utilisation, when muscle temperature was increased from 22.5 to 38.6 ° C (Edwards et al., 1972). Just as increased cross-bridge cycling during isometric contractions will require an increased energy turnover for the same sustained force, at relatively slow contraction velocities, the effect of prior heating could result in cross-bridge cycling that is faster than required by the actin–myosin movement. Thus, we would propose that, in these experiments, prior heating results in an inappropriately fast rate of cross-bridge cycling at 60 revs min^{-1} leading to an increased energy turnover and decreased efficiency.

This is illustrated by considering the schematic relationship between mechanical efficiency and velocity as shown in Fig. 5. The solid line represents the efficiency/velocity relationship under control conditions. In this schema, we have assumed that 60 revs min^{-1} (point a) is around the optimum velocity for maximal efficiency, i.e. the velocity at which the cross-bridge cycling rate is close to the required rate of actin–myosin movement. Heating the muscle will increase the cross-bridge cycling rate, shifting the efficiency/velocity relationship to the right (as shown by the dashed line in Fig. 5). As a consequence, the mechanical efficiency will be reduced at 60 revs min^{-1} (point b), i.e. energy turnover for a given mechanical output delivered will be increased. This shift in the efficiency/velocity relationship of course reflects the change in the force/velocity relationship of muscle that occurs when temperature is elevated (Ranatunga, 1984). It should also be noted that the shift is analogous to the difference in the mechanical efficiency/

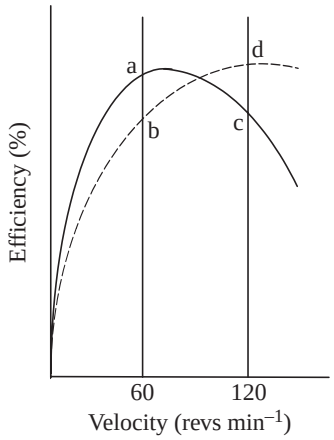

Fig. 5. A schematic representation of the qualitative changes that, on theoretical grounds, might be expected of the efficiency/velocity relationship consequent upon an increase in muscle temperature. In the illustration, the solid line shows the efficiency/velocity relationship for a muscle that has an optimum velocity for maximum efficiency close to 60 revs min^{-1} under normal conditions. The dashed line indicates the expected rightward shift consequent upon an increase in muscle temperature. Thus, increasing muscle temperature at 60 revs min^{-1} results in a decrease in efficiency from point a to b. In contrast, at a higher movement frequency (120 revs min^{-1}), efficiency increases from to c to d as a result of the rightward shift in the relationship.

velocity relationship between slow and fast muscle (Woledge, 1968; Goldspink, 1978; Reggiani et al., 1997; He et al., 2000).

In contrast to the effects seen at 60 revs min^{-1}, our experiments show that during exercise at 120 revs min^{-1} energy turnover was lower after muscle temperature had been elevated. In the schematic illustration shown in Fig. 5, a pedalling rate of 120 revs min^{-1} lies to the right of the optimum velocity for maximum efficiency under normal conditions (point c), i.e. on the descending right limb of the efficiency/velocity relationship. Consequently, the effect of heating the muscle, as shown by the dashed line, is to shift the efficiency/velocity relationship to the right so that optimum velocity occurs at a higher pedalling rate and efficiency at 120 revs min^{-1} (point d) is increased.

In this Discussion, we have been concerned with the effect of local heating on the energy turnover of the active muscles as a whole. Thus, changes in the efficiency/velocity relationship refer to a 'global' relationship of the involved muscles and the recruited fibres of those muscles that have diverse contractile and metabolic properties. It has not been possible to determine the pattern of fibre type recruitment in the present experiments. Nevertheless, at an exercise intensity of 85 % of $\dot{V}_{O_2max}$ at 60 revs min^{-1}, the power required probably represents less than 50 % of the maximal power available at that velocity of contraction (Greig et al., 1986) (see Sargeant and Jones, 1995). It is probable that the majority of the power required in those experiments could be generated by type I muscle fibres acting alone if motor units were recruited purely on a hierarchical size principle without any modulation due to rate coding, i.e. a change in force generated due to the frequency of stimulation (Sargeant, 1999). It is clear, however, that while the hierarchy of motor units is the major determinant of recruitment there is some element of rate coding, as indicated by studies of metabolic intermediates and glycogen depletion (Greig et al., 1986; Ivy et al., 1987). Thus, even during relatively low-intensity exercise, type II muscle fibres will be active, albeit at low firing frequencies and thereby making a minor contribution to force production and metabolic cost.

It has previously been suggested that, at a contraction frequency of 60 revs min^{-1} in cycling exercise, human type I fibres might be operating close to their optimum for maximum efficiency (Sargeant and Jones, 1995). Thus, a temperature-induced shift to the right of the efficiency/velocity relationship for the recruited fibre population, as proposed schematically in Fig. 5, would lead to a decrease in efficiency; i.e. energy turnover for a given mechanical power output would have to increase. The increased rate of oxygen uptake and increased blood lactate concentration observed following passive warming when cycling at 60 revs min^{-1} are entirely consistent with this suggestion.

At 85 % of $\dot{V}_{O_2max}$ at a contraction frequency of 120 revs min^{-1}, the type I fibres would have remained fully recruited in accordance with the hierarchical pattern of recruitment (see Beelen et al., 1993; Sargeant and Kernell, 1993). However, at a contraction frequency of 120 revs min^{-1}, the type I fibres may normally operate on the descending right side of the power/velocity relationship and therefore also of the efficiency/velocity relationship (Sargeant, 1999). Following heating and the subsequent shift to the right of the efficiency/velocity relationship, the type I fibres may be closer to their optimum velocity for efficiency, as suggested by point d in Fig. 5. Hence, efficiency will increase and energy turnover for a given mechanical power output will decrease, as we have observed.

In the present investigation, we have examined the effect of prior passive heating of the active muscle fibres on the subsequent energy cost during dynamic exercise. It will be realised that muscle temperature can also be expected to rise during exercise as a result of the liberation of metabolic heat. A number of authors have speculated that the so-called 'slow component' of $\dot{V}_{O_2}$ seen during sustained exercise is a consequence of an increase in muscle temperature (for a review, see Gaesser and Poole, 1996). It has been suggested that this is due to a decrease in mitochondrial efficiency with increasing temperature (e.g. Brooks et al., 1971). The present data do not, however, provide evidence to support this hypothesis. At 60 revs min^{-1}, there was a temperature-related increase in energy turnover; in contrast, while cycling at 120 revs min^{-1} with the same heating protocol, the situation was reversed and energy turnover decreased. Furthermore it is notable that during recovery, i.e. when the mechanical efficiency/velocity relationship is no longer a factor, the $\dot{V}_{O_2}$-off kinetics were not demonstrably different between the heated and control conditions, either at 60 or at 120 revs min^{-1} (Fig. 2).

If muscle temperature affected mitochondrial efficiency, as proposed by earlier authors, it might be expected that an elevated

$\dot{V}_{O_2}$ would be observed during recovery when the muscle was heated. Of course, it is still just possible that there is a temperature-related decrease in mitochondrial efficiency, but that at 120 revs min^{-1} the effect of this on the total energy turnover may be obscured by the magnitude of the proposed shift in the efficiency/velocity relationship as a result of elevated temperature. Recent data, however, have also suggested that elevated muscle temperature does not contribute to the slow component of $\dot{V}_{O_2}$ during heavy exercise (Koga et al., 1997). Notwithstanding the underlying mechanism for the slow component of $\dot{V}_{O_2}$, this will affect the estimated efficiency. In the present experiments where the $\dot{V}_{O_2}$-on kinetics (and off-kinetics) appear similar regardless of temperature conditions, efficiency was calculated in the sixth minute. It should be noted however, that the absolute efficiency values would change if exercise were prolonged as a result of the increased energy turnover characterised as the 'slow component of $\dot{V}_{O_2}$ kinetics'. It should also be realised that heating the legs can be expected to have an effect on core temperature (which has not been measured in this investigation) and may have energetic consequences for pulmonary $\dot{V}_{O_2}$, although the existing evidence is equivocal (e.g. Nielsen et al., 1990; González-Alonso et al., 1998). In our investigation, the impact of heating the legs on core temperature would be the same at both 60 and 120 revs min^{-1}, but the unique observation is that $\dot{V}_{O_2}$ changed in opposite directions depending upon contraction frequency.

In conclusion, we believe that, in these experiments, the effects of heating on energy turnover reflect the dominant contribution of type I muscle fibres to the external power output at both 60 and 120 revs min^{-1}. These observations may also help to explain why athletes adopt relatively fast cadences during sustained high-intensity exercise (Sargeant, 1994). At low cadences, there would be an additional increasing energy cost as a result of an exercise-induced increase in muscle temperature. In contrast, at high cadences, the increase in muscle temperature during exercise will lead to a reduction in energy cost and a greater efficiency of locomotion.

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