Non-linear relationship between O_2 uptake and power output at high intensities of exercise in humans

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1. A slow component to pulmonary oxygen uptake ($\dot{V}_{O_2}$) is reported during prolonged high power exercise performed at constant power output at, or above, approximately 60% of the maximal oxygen uptake. The magnitude of the slow component is reported to be associated with the intensity of exercise and to be largely accounted for by an increased $\dot{V}_{O_2}$ across the exercising legs.

2. On the assumption that the control mechanism responsible for the increased $\dot{V}_{O_2}$ is intensity dependent we hypothesized that it should also be apparent in multi-stage incremental exercise tests with the result that the $\dot{V}_{O_2}$–power output relationship would be curvilinear.

3. We further hypothesized that the change in the $\dot{V}_{O_2}$–power output relationship could be related to the hierarchical recruitment of different muscle fibre types with a lower mechanical efficiency.

4. Six subjects each performed five incremental exercise tests, at pedalling rates of 40, 60, 80, 100 and 120 rev min^{-1}, over which range we expected to vary the proportional contribution of different fibre types to the power output. Pulmonary $\dot{V}_{O_2}$ was determined continuously and arterialized capillary blood was sampled and analysed for blood lactate concentration ($[lactate]_b$).

5. Below the level at which a sustained increase in $[lactate]_b$ was observed pulmonary $\dot{V}_{O_2}$ showed a linear relationship with power output; at high power outputs, however, there was an additional increase in $\dot{V}_{O_2}$ above that expected from the extrapolation of that linear relationship, leading to a positive curvilinear $\dot{V}_{O_2}$–power output relationship.

6. No systematic effect on the magnitude or onset of the 'extra' $\dot{V}_{O_2}$ was found in relation to pedalling rate, which suggests that it is not related to the pattern of motor unit recruitment in any simple way.

Whipp & Wasserman (1972) reported that during intensive exercise at constant power output at which blood lactate concentration starts to rise above resting level, there was a slow continuous increase in pulmonary oxygen uptake. This finding has been confirmed in subsequent investigations (Hagberg, Mullin & Nagle, 1978; Poole, Ward, Gardner & Whipp, 1988; Henson, Poole & Whipp, 1989). A number of factors have been proposed to explain this phenomenon including increased concentration of lactate (Casaburi, Storer, Ben-dov & Wasserman, 1987), hydrogen ions (Capelli, Antonutto, Zamparo, Girardis & di Prampero, 1992) and catecholamines (Cath, 1971), the recruitment of less efficient type II muscle fibres (Coyle, Sidossis, Horowitz & Beltz, 1992) and increased muscle temperature (Hagberg *et al.* 1978). In addition it has been suggested that the increase in pulmonary oxygen uptake could simply reflect an increase in the cost of ventilation, cardiac output (Hagberg *et al.* 1978) and posture relative to the external power delivered (Hagberg, Mullin, Giese & Spitznagel, 1981). In recent experiments, however, Poole *et al.* (1991) examined this latter possibility by simultaneously measuring oxygen uptake ($\dot{V}_{O_2}$) across the exercising leg and the pulmonary $\dot{V}_{O_2}$ during supine cycling. They were able to show that the slow increase in pulmonary $\dot{V}_{O_2}$ during severe prolonged exercise at constant power output reflected a similar increase in $\dot{V}_{O_2}$ across the exercising leg. This finding seems to exclude disproportionate increases in e.g. ventilatory cost as a major factor and may indicate a difference in the relationship between $\dot{V}_{O_2}$ and power output which is intensity dependent and intrinsic to the active muscle.

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Based on the above it is difficult to understand why a constant and linear relationship should be expected between $\dot{V}_{O_2}$ and power output in exercise testing where the power output is progressively increased. Nevertheless a linear $\dot{V}_{O_2}$–power output relationship is often reported (e.g. Medbø, Mohn, Tabata, Bahr, Vaage & Sejersted, 1988) and stated by authoritative texts (Åstrand & Rodahl, 1986; Powers & Howley, 1990; Wilmore & Costill, 1994) as evidence of a constant mechanical efficiency, independent of exercise intensity. Based on this it has also been claimed that there can therefore be no difference in the mechanical efficiency of different muscle fibre types since the proportional contribution to mechanical output of these will change as exercise intensity increases (Medbø *et al.* 1988; Medbø, 1990). Furthermore, the assumed linear $\dot{V}_{O_2}$–power output relationship in progressive exercise is often used to predict maximal $\dot{V}_{O_2}$ and if the assumption is incorrect or purely fortuitous depending on the precise protocol used, then it may contribute systematically to the error of predictive techniques.

A non-linear relationship between $\dot{V}_{O_2}$ and power output in incremental tests has been documented previously (Whipp, 1986; Hansen, Casaburi, Cooper & Wasserman, 1988) but the significance and underlying mechanisms of these observations has not been widely recognized or systematically investigated. Furthermore, in neither of these earlier reports was the onset of the extra $\dot{V}_{O_2}$ per watt of heavy work examined in relation to the onset of blood lactate accumulation. Therefore, in these experiments, we have examined the $\dot{V}_{O_2}$–power output relationship during incremental tests using a breath-by-breath measurement system together with blood lactate determination and in contrast to many earlier studies we have used a protocol designed to elicit at least nine steps in power output of 3 min duration each, with minimally three and usually five steps above that at which there was a significant and sustained increase in blood concentration of lactate ($[\text{lactate}]_b$).

To explore the possibility that the hierarchical recruitment of different muscle fibre type populations with lower mechanical efficiencies may contribute to any 'extra' increase in $\dot{V}_{O_2}$ subjects performed five separate tests at different pedalling rates (40, 60, 80, 100 and 120 rev min^{-1}). It has been proposed that over this range of pedalling rates there should be significant variations in the proportional contribution to total power output from the different fibre type populations (Sargeant, 1994).

METHODS

Six healthy, physically active males, not specifically trained (means ± S.D.; age, 25·8 ± 3·8 years; weight, 80·3 ± 6·5 kg; height, 187 ± 8·9 cm) were studied after giving informed consent to the experimental procedure. All of them were experienced in laboratory tests and in cycling at the different pedalling rates used in this study.

Incremental tests

All subjects performed five incremental tests on the same cycle ergometer. Each test was performed at a different pedalling rate (40, 60, 80, 100 and 120 rev min^{-1}) in random order. After a 6 min resting period sitting on the cycle ergometer, the subjects began to cycle at a power output of 50 W and the power output was then increased by 30 W every 3 min. Subjects were encouraged to continue the tests until exhaustion. The tests were stopped when the subjects could no longer maintain the required pedalling rate or power output. All experiments were performed on a modified electromagnetically braked cycle ergometer (Lode Standard, Lode, Groningen, The Netherlands) which is based on the Lanooy principle (Lanooy & Bonjer, 1956). The modified ergometer is equipped with strain gauges mounted inside the pedals to measure continuously pedal forces horizontal and vertical to the pedal surface during each revolution. From these forces, together with the crank and pedal angles and the crank speed, power is calculated. (For a detailed description of the system see Beelen, Sargeant & Wijkhuizen, 1994.) Comparison of this calculated power was at all pedal frequencies studied in very close agreement with the power set on the ergometer and there was no systematic difference related to intensity or pedal frequency. Experiments were performed at constant room temperature and humidity (18 °C and 55%, respectively).

Gas exchange variables

Throughout the test gas exchange variables were measured continuously breath by breath (Oxycon Gamma, Mijnhardt, Bunnik, The Netherlands). Before and after finishing each test the gas analysers were calibrated with certificated calibration gases spanning the range of measured concentrations and room air. The first 20 s of this calibration was used to flush the analysers, the last 10 s was used to measure the concentration of the sample gas. The mouth volume sensor was calibrated with a certificated 3 l syringe. After at least six complete strokes the average values over the last five strokes is used to calculate in- and expiratory values. The measured values had to fall within 2% of the reference value. Both calibration procedures were always performed twice. No significant changes were found in any of the test periods confirming the stability of the breath-by-breath system over the duration of the test period.

Prior to the present experiments the breath-by-breath system was validated for use at high power outputs (450 W) and high minute ventilation (180 l min^{-1}) by comparison of the data with those calculated from simultaneous Douglas bag collections. In more than fifty such duplicate determinations the difference never exceeded ± 5% and there was no systematic bias.

Blood lactate

Blood lactate concentration was determined in samples of arterialized blood taken from the finger tip at the end of each 3 min step and analysed enzymatically (model 23L; YSI, Yellow Springs, OH, USA). For descriptive purposes we identified the stage at which a sustained increase in blood lactate concentration occurred as the percentage $\dot{V}_{O_2,max}$ and as the power output step which elicited an increase of at least 0·5 mmol l^{-1} in $[\text{lactate}]_b$ and above which the same or greater increases were observed during subsequent steps.

Table 1. Observed and expected oxygen uptake and power output

Pedalling rate (rev min^{-1})	PO_{max} (W)	Obs $\dot{V}_{O_2}$ at PO_{max} (l min^{-1})	Exp $\dot{V}_{O_2}$ at PO_{max} (l min^{-1})	ΔObs/Exp (%)	% PO_{max} $[lactate]_b$
40	335 ± 31*	5·21 ± 0·40*	4·52 ± 0·61	16	67 ± 10
60	365 ± 46	5·32 ± 0·49*	4·58 ± 0·55	17	66 ± 5
80	379 ± 41	5·36 ± 0·29*	4·74 ± 0·49	14	61 ± 8
100	372 ± 52	5·42 ± 0·48*	4·72 ± 0·55	15	63 ± 9
120	308 ± 51*	5·36 ± 0·63*	4·62 ± 0·38	16	59 ± 8

Means ± S.D. for: the final power output achieved (PO_{max}); the observed $\dot{V}_{O_2}$ at final power output; the expected $\dot{V}_{O_2}$ at final power output – predicted from the linear relationship of $\dot{V}_{O_2}$–power output below the stage of sustained increase in $[lactate]_b$; and the percentage of final power output beyond which there was a sustained increase in $[lactate]_b$ (see Methods). *$P < 0·01$, PO_{max} at 40 and 120 *vs.* PO_{max} at 60, 80 and 100 rev min^{-1}; observed $\dot{V}_{O_2}$ *vs.* expected $\dot{V}_{O_2}$.

Statistics

Values represent means ± S.D. Statistical significance was tested by analysis of variance for repeated measures.

RESULTS

The subjects studied were able to accomplish at least nine stages of increasing power output in all tests, and they attained final power outputs which elicited a $\dot{V}_{O_2}$ of ~5·3 l min^{-1}. There was no significant difference between the final $\dot{V}_{O_2}$ attained in exercise tests at different pedalling rates, but there was a significantly lower maximum power output attained in the exercise tests performed at 40 and 120 rev min^{-1} compared with other pedalling rates ($P < 0·01$; Table 1).

As expected blood lactate concentration increased in an exponential fashion during all exercise tests (Fig. 1). There was a sustained increase in blood lactate concentration at approximately 60% of maximum power output and this was not significantly different between pedalling rates. All subjects completed at least three and up to six stages above this stage in all tests (Table 1). $[Lactate]_b$ was, however, significantly higher at all stages of the tests conducted at 120 rev min^{-1} compared with all other pedalling rates (Fig. 1; $P < 0·01$). When $[lactate]_b$ was plotted against $\dot{V}_{O_2}$ for the different pedalling rates $[lactate]_b$ accumulation occurred at the same percentage $\dot{V}_{O_2,max}$ independent of pedalling rate but ranging from 52 to 70% for the different subjects.

As shown by the individual example of the breath-by-breath records, $\dot{V}_{O_2}$ shows an initial linear increase with time and hence power output at all pedalling rates (Figs 2 and 3). In the later stages of the tests, that is at high power outputs, there was a disproportionate increase in $\dot{V}_{O_2}$ above that expected from the initial linear relationship (Fig. 3).

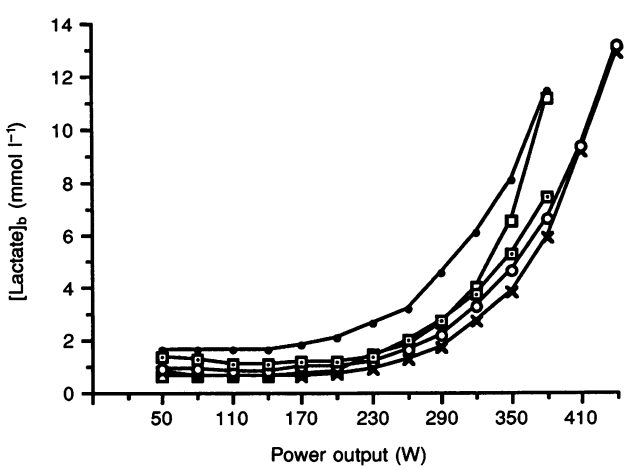

Figure 1. Blood lactate concentration and power output

$[Lactate]_b$ in relation to power output in tests at pedalling rates of 40 (□), 60 (×), 80 (○), 100 (⊡) and 120 (●) rev min^{-1}.

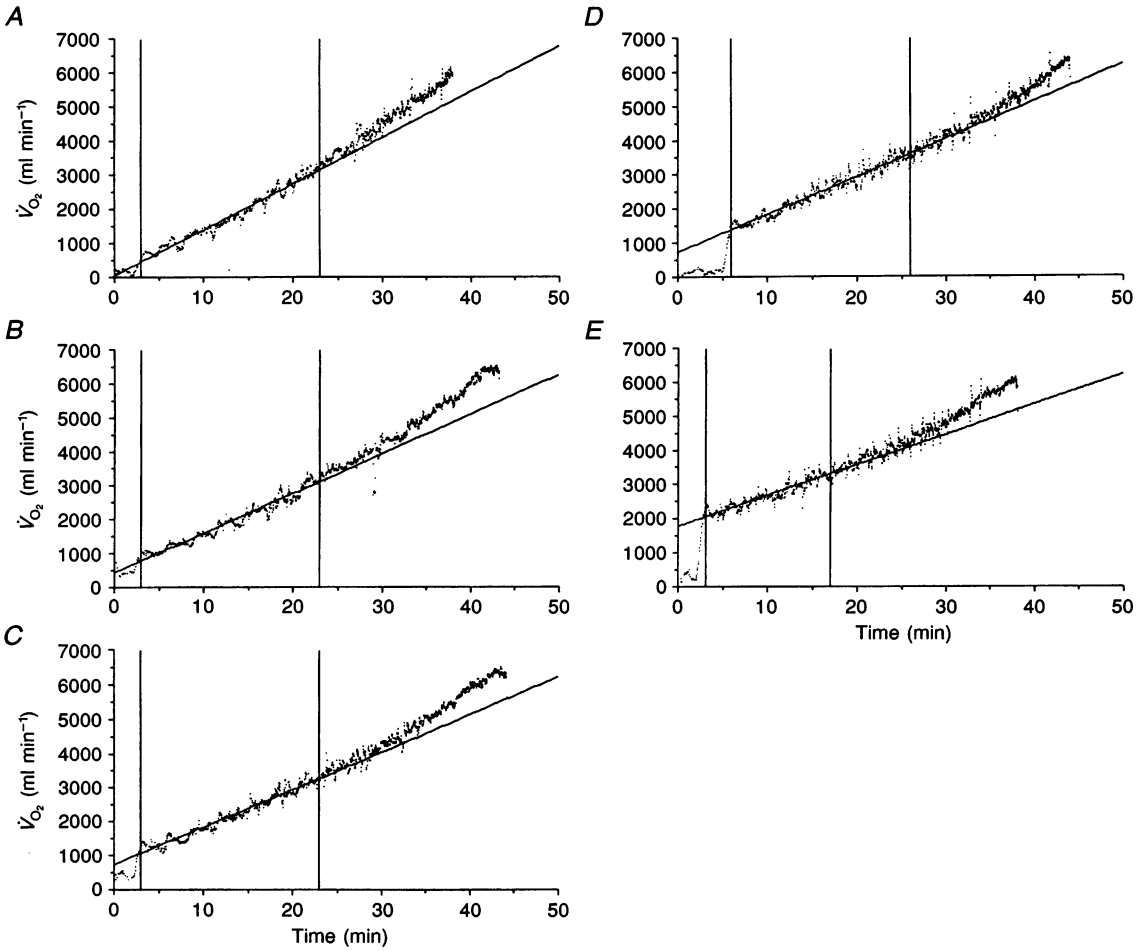

Figure 2. Breath-by-breath determination of oxygen uptake

Original records of breath-by-breath oxygen uptake *vs.* time during incremental tests at pedalling rates of 40 (*A*), 60 (*B*), 80 (*C*), 100 (*D*) and 120 (*E*) rev min^{-1}. The inital power output is 50 W and is increased by 30 W every 3 min in all tests. The stage at which $[lactate]_b$ showed a sustained increase of $> 0{\cdot}5$ mmol l^{-1} step^{-1} is shown by the vertical line: the linear regression for $\dot{V}_{O_2}$ is calculated from the data below this stage.

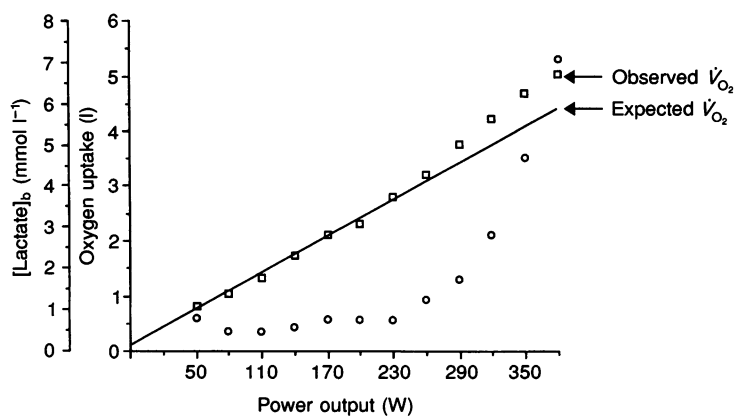

Figure 3. Oxygen uptake and blood lactate in relation to power output

$\dot{V}_{O_2}$ (□) and $[lactate]_b$ (○) in relation to power output. Subject A.K. pedalling at 100 rev min^{-1}. $\dot{V}_{O_2}$ values are the means of the last minute of each increment in power output. Blood for [lactate] analysis was sampled at the end (3 min) of each increment. The linear regression was calculated from the $\dot{V}_{O_2}$–power output data below the stage at which a sustained increase in $[lactate]_b$ was observed ($r^2 = 0{\cdot}992$).

The magnitude of this effect can be characterized by comparing the observed $\dot{V}_{O_2}$ elicited at the final power output achieved with the 'expected' $\dot{V}_{O_2}$ as predicted from extrapolation of the initial $\dot{V}_{O_2}$–power output relationship calculated separately for each test using only the data points below the stage at which a sustained increase in $[lactate]_b$ was seen. This comparison indicates that the true oxygen cost of the final power output was 14–17% higher than expected from the initial linear relationship (Table 1; $P < 0.01$). Analysis of variance experiments performed at the different pedalling rates revealed no systematic effect on either the magnitude or the point of onset of the 'extra' increase in $\dot{V}_{O_2}$ when expressed in terms of percentage of the maximum power output achieved.

DISCUSSION

The results of our experiments support the phenomenon noted by Whipp (1986) that during incremental cycle ergometer tests performed until exhaustion, the $\dot{V}_{O_2}$–power output relationship is not linear over the whole range of power outputs from rest to maximum. In the initial part of the test there was a linear increase in $\dot{V}_{O_2}$ with increasing power output (Figs 2 and 3), with a similar slope at 60 rev min^{-1} (10·4 ml min^{-1} W^{-1}) as reported by others. However, at higher power outputs there was an additional increase in $\dot{V}_{O_2}$ above that expected from the power output increase alone. The actual values of $\dot{V}_{O_2}$ at the final power output attained were approximately 15% higher than the $\dot{V}_{O_2}$ predicted from the observed linear $\dot{V}_{O_2}$–power output relationship derived from the incremental data below the onset of sustained increases in $[lactate]_b$ (Table 1, Fig. 3).

The present findings are consistent with a previous report of Hansen *et al.* (1988) who reported a steeper $\dot{V}_{O_2}$–power output relationship in the second compared with the first half of tests in which power increased by 0·25 and 0·5 W every 0·5 s. Nevertheless our findings, the earlier comments of Whipp (1986) and the findings of Hansen *et al.* (1988) are in contradiction to the still widely held belief, derived from authoritative texts, that the $\dot{V}_{O_2}$–power output relationship is linear up to $\dot{V}_{O_2,max}$ (e.g. Åstrand & Rodahl, 1986; Powers & Howley, 1990; Wilmore & Costill, 1994). Nevertheless our observations are also consistent with a number of reports of a 'slow component' of $\dot{V}_{O_2}$ or 'O_2 drift' during prolonged exercise at intensities above the level at which significant increases in blood lactate occur (Roston, Whipp, Davis, Cunningham, Effros & Wasserman, 1987; Poole *et al.* 1988, 1991). These latter reports clearly show that the oxygen cost for power output does not remain constant over time and that the causal factor for the observed increase is associated with the intensity of the exercise itself and is largely intrinsic to the exercising legs.

It may be questioned why the non-linearity of the $\dot{V}_{O_2}$–power output relationship has not been recognized more often. We believe there are a number of reasons. The most important are related to the type of protocol used in many multi-stage tests; these often give very few points at high intensities, so that it is not easy to identify, let alone test for, non-linearity. Moreover, any isolated data points which deviate from linearity may be dismissed either as experimental noise or the consequence of a disproportionate increase in ventilatory, cardiac or postural costs. Furthermore, the 'near-linearity' seen in some protocols may also be the consequence of 'the fortuitous balance' of different factors as suggested by Hansen *et al.* (1988).

The reason for the 'extra' oxygen cost for power output at high intensity exercise is not clear (Poole *et al.* 1991). Hansen *et al.* (1988) calculated that central factors, such as disproportionate increase in ventilatory, cardiac or postural costs, were unlikely to account for more than a small proportion of the 'extra' $\dot{V}_{O_2}$ in their experiments. Subsequently Poole *et al.* (1991) demonstrated that 86% of the increase in pulmonary $\dot{V}_{O_2}$ seen between 3 and 21 min of constant power high intensity exercise was attributable to the exercising legs. This latter observation would also seem to rule out a major contribution from other processes *outside* the exercising limb such as stimulation of tissue metabolism by catecholamines, potassium or lactate.

Other factors which are intrinsic to the exercising muscles and may play a role in the increased $\dot{V}_{O_2}$ at high exercise intensity include increases in muscle temperature, lactate, H^+, potassium and catecholamines. With increasing exercise intensity the hierarchical recruitment of different fibre types with different efficiencies may also be a candidate for contributing to the increased $\dot{V}_{O_2}$. In the present investigation we hoped to gain some insight into the role of the last of these possibilities by having subjects perform five tests at different pedalling rates from 40 to 120 rev min^{-1}.

On the basis of a previous analysis we hypothesized that as a consequence of the power–velocity relationship for slow and fast muscle fibres we might expect, at pedalling rates above 60 rev min^{-1}, to see an earlier involvement and progressive increase in the proportional contribution of the fast fibre population to the total power delivered (Sargeant, 1994). This latter analysis was based on measurement of the maximum peak power–velocity (pedalling rate) relationship in subjects with known muscle fibre type composition determined from needle biopsies (Sargeant, Hoinville & Young, 1981; Sargeant, 1994) and supported by (a) evidence of selective fatigue of fast fatigue-sensitive fibres following sustained exercise (Beelen & Sargeant, 1991, 1993) and (b) the effect of changes in muscle temperature on the power–velocity relationship in subjects with different muscle fibre composition (Rademaker, Blonk, de Haan & Sargeant, 1994). Indeed, in the present study the consistently higher $[lactate]_b$ at 120 rev min^{-1} at all power output increments could be evidence of an earlier and proportionately greater contribution of faster glycolytic

fibres, as previously suggested by a glycogen depletion study (Beelen, Sargeant, Lind, de Haan, Kernell & van Mechelen, 1993).

In the event, however, we could not detect any systematic effect on either the magnitude or the onset of the 'extra' $\dot{V}_{O_2}$ in relation to pedalling rate. At first sight this may seem to exclude the hierarchical recruitment of fibres with lower efficiency from being a significant factor in the change in the $\dot{V}_{O_2}$–power output relationship. It should be remembered, however, that little is known of the mechanical efficiency–velocity relationship of human muscle fibres (see Sargeant & Beelen, 1993 for a discussion of this point). It is possible that in the tests conducted at higher pedalling rates the effect on energy turnover of an earlier involvement of faster fibres is obscured by the fact that simultaneous with a proportional increase in the power contributed, these fibres may be effectively operating at a higher point on the ascending left side of their mechanical efficiency–velocity relationship; that is they may be operating at a velocity closer to the optimum for maximal efficiency. We conclude therefore that the effect of hierarchical or progressive recruitment of muscle fibres with different mechanical efficiencies may still be a factor and should not be excluded from consideration.

Of the other possibilities, increases in lactate has been found to be most consistently associated with the 'extra' or 'slow component' of $\dot{V}_{O_2}$ in prolonged exercise. The slow component is only found at exercise intensities above which a sustained lactate concentration is produced (Whipp & Wasserman, 1972). The magnitude of the 'extra' $\dot{V}_{O_2}$ seems to follow closely the intensity of exercise and the degree of lactate accumulation and concomitant acidosis (Roston *et al.* 1987) and there seems to be a temporal association between increasing acidosis and an increase in 'extra' $\dot{V}_{O_2}$ (Poole *et al.* 1988). Consistent with these observations we saw no evidence of a deviation from linearity in the $\dot{V}_{O_2}$–power output relationship until $[\text{lactate}]_b$ showed a sustained increase. However, in some cases the 'additional' increase in $\dot{V}_{O_2}$ started to appear with a delay in relation to the beginning of lactate acidosis as we have characterized it. This may suggest that in some cases a significant change in $[\text{lactate}]_b$ or associated $[H^+]$ is required to cause the additional increase in $\dot{V}_{O_2}$ or it may simply reflect the fact that blood concentrations are indirect indicators of the levels prevailing in the muscle.

How an increase in muscle lactate or $[H^+]$ might act is not clear although it has been suggested that acidosis may accelerate the rate of mitochondrial respiration in the skeletal muscle by increasing the free creatine (Mahler, 1985) consequent to a shift in the equilibrium of the creatine kinase reaction (Harris, Sahlin & Hultman, 1977). Such a mechanism was recently proposed by Capelli *et al.* (1992) as a possible explanation of the relationship between the progressive upward drift in oxygen uptake and increasing acidosis during prolonged exercise of high intensity. Additionally, increases in $[H^+]$ have been proposed to reduce the free energy yield from ATP hydrolysis at least in single fibres (Cooke, Franks, Luciani & Pate, 1988).

In conclusion our data demonstrate that during incremental tests, performed at different pedalling rates above the onset of a sustained increase in $[\text{lactate}]_b$, there was an additional increase in $\dot{V}_{O_2}$ above that expected from the power output increase alone. We believe that the mechanism(s) of this phenomenon are probably the same as those causing the 'slow component' of $\dot{V}_{O_2}$ when performing a prolonged high power output exercise. Despite the fact that the mechanism(s) responsible for this phenomenon are not clear, this is a substantial effect which needs to be taken into consideration in understanding skeletal muscle energy turnover *in vivo* and which has practical significance for determinations of the physical work capacity and the apparent mechanical efficiency or economy of human exercise.

Åstrand, P. O. & Rodahl, K. (1986). *Textbook of Work Physiology; Physiological Bases of Exercise*, p. 300. McGraw-Hill Company, New York.

Beelen, A. & Sargeant, A. J. (1991). Effect of fatigue on maximal power at different contraction velocities in humans. *Journal of Applied Physiology* **71**, 2332–2337.

Beelen, A. & Sargeant, A. J. (1993). Effect of prior exercise at different pedal frequencies on maximal power in humans. *European Journal of Applied Physiology* **66**, 102–107.

Beelen, A., Sargeant, A. J., Lind, A., de Haan, A., Kernell, D. & van Mechelen, W. (1993). Effect of contraction velocity on the pattern of glycogen depletion in human muscle fibre types. In *Neuromuscular Fatigue*, ed. Sargeant, A. J. & Kernell, D., pp. 93–96. Academy Series, Royal Netherlands Academy of Arts and Sciences-North Holland, Amsterdam.

Beelen, A., Sargeant, A. J. & Wijkhuizen, F. (1994). Measurements of directional force and power during human submaximal and maximal isokinetic exercise. *European Journal of Applied Physiology* **68**, 177–181.

Capelli, C., Antonutto, G., Zamparo, P., Girardis, M. & di Prampero, P. E. (1993). Effects of prolonged cycle ergometer exercise on maximal muscle power and oxygen uptake in humans. *European Journal of Applied Physiology* **66**, 189–195.

Casaburi, R., Storer, T. W., Ben-dov, I. & Wassermen. K. (1987). Effect of endurance training on possible determinants of V_{O_2} during heavy exercise. *Journal of Applied Physiology* **62**, 199–207.

Cath, S. M. (1971). Exercise O_2 debt of dogs at ground level and at altitude with and without β-block. *Journal of Applied Physiology* **30**, 838–843.

COOKE, R., FRANKS, K., LUCIANA, G. B. & PATE, E. (1988). The inhibition of rabbit skeletal muscle contraction by hydrogen ions and phosphate. *Journal of Physiology* **395**, 77–97.

COYLE, E. F., SIDOSSIS, L. S., HOROWITZ, J. F. & BELTZ, J. D. (1992). Cycling efficiency is related to the percentage of type I muscle fibres. *Medicine Science in Sports and Exercise* **24**, 782–788.

HAGBERG, J. M., MULLIN, J. P., GIESE, M. D. & SPITZNAGEL, E. (1981). Effects of pedalling rate on submaximal exercise responses of competetive cyclists. *Journal of Applied Physiology* **51**, 447–451.

HAGBERG, J. M., MULLIN, J. P. & NAGLE, F. J. (1978). Oxygen consumption during constant-load exercise. *Journal of Applied Physiology* **45**, 381–384.

HANSEN, J. E., CASABURI, R., COOPER, D. M. & WASSERMAN, K. (1988). Oxygen uptake as related to work rate increment during cycle ergometer exercise. *European Journal of Applied Physiology* **57**, 140–145.

HARRIS, R. C., SAHLIN, K. & HULTMAN, E. (1977). Phosphagen and lactate content of m. quadriceps femoris of man after exercise. *Journal of Applied Physiology* **43**, 852–857.

HENSON, L. C., POOLE, D. C. & WHIPP, B. J. (1989). Fitness as a determinant of oxygen uptake response to constant-load exercise. *European Journal of Applied Physiology* **59**, 21–28.

LANOOY, C. & BONJER, F. H. (1956). A hyperbolic ergometer for cycling and crancking. *Journal of Applied Physiology* **9**, 499–500.

MAHLER, M. (1985). First-order kinetics of muscle oxygen consumption and an equivalent proportionality between Q_{O_2} and phosphorylcreatine level. *Journal of General Physiology* **86**, 135–165.

MEDBØ, J. I. (1990). Type I and type II fibres work with the same mechanical efficiency during bicycling. In *Muscle and Motility*, ed. MARÉCHAL, G. & CARRARO, U., pp. 303–308. Intercept, Andover, Hampshire, UK.

MEDBØ, J. I., MOHN, A. C., TABATA, I., BAHR, R., VAAGE, O. & SEJERSTED, O. M. (1988). Anaerobic capacity determined by maximal accumulated O_2 deficit. *Journal of Applied Physiology* **64**, 50–60.

POOLE, D. C., SCHAFFARTZIK, W., KNIGHT, D. R., DERION, T., KENNEDY, B., GUY, H. J., PREDILETTO, R. & WAGNER, P. D. (1991). Contribution of exercising legs to the slow component of oxygen uptake kinetics in humans. *Journal of Applied Physiology* **71**, 1245–1253.

POOLE, D. C., WARD, S. A., GARNER, G. W. & WHIPP, B. J. (1988). Metabolic respiratory profile of the upper limit for prolonged exercise in man. *Ergonomics* **9**, 1265–1279.

POWERS, S. K. & HOWLEY, E. T. (1990). *Exercise Physiology: Theory and Application to Fitness and Performance*, p. 54. Wm. C. Brown Publishers, Dubuque, IN, USA.

RADEMAKER, A., BLONK, F., DE HAAN, A. & SARGEANT, A. J. (1994). Muscle fibre type dependent effect of muscle temperature on power output in human locomotion. *European Journal of Applied Physiology* **69**, suppl. S9.

ROSTON, W. L., WHIPP, B. J., DAVIS, J. A., CUNNINGHAM, D. A., EFFROS, R. M. & WASSERMAN, K. (1987). Oxygen uptake kinetics and lactate concentration during exercise in humans. *American Review of Respiratory Diseases* **135**, 1080–1084.

SARGEANT, A. J. (1994). Human power output and muscle fatigue. *International Journal of Sports Medicine* **15**, 116–121.

SARGEANT, A. J. & BEELEN, A. (1993). Human muscle fatigue in dynamic exercise. In *Neuromuscular Fatigue*, ed. SARGEANT, A. J. & KERNELL, D., pp. 81–92. Academy Series, Royal Netherlands Academy of Arts and Sciences-North Holland, Amsterdam.

SARGEANT, A. J., HOINVILE, E. & YOUNG, A. (1981). Maximum leg force and power output during short-term dynamic exercise. *Journal of Applied Physiology* **51**, 1175–1182.

WHIPP, B. J. (1986). Exercise bioenergetics and gas exchange. In *Cardio-Pulmonary Exercise Testing*, ed. LEFF, A. R., pp. 1–21. Grune & Sratton, Orlando, FL, USA.

WHIPP, B. J. & WASSERMAN, K. (1972). Oxygen uptake kinetics for various intensities of constant load work. *Journal of Applied Physiology* **33**, 351–356.

WILMORE, J. H. & COSTILL, D. L. (1994). *Physiology of Sport and Exercise*, p. 110. Human Kinetics, Champaign.

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