Eur J Appl Physiol (2002) 88: 100–106
DOI 10.1007/s00421-002-0690-5

ORIGINAL ARTICLE

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Oxygen uptake and heart rate kinetics during heavy exercise: a comparison between arm cranking and leg cycling

Accepted: 21 June 2002 / Published online: 18 September 2002

Abstract This study examined the oxygen uptake ($\dot{V}O_2$) and heart rate (HR) kinetics during arm cranking and leg cycling at work rates above the anaerobic threshold (AT). Ten untrained male subjects [21.6 (1.3) years] completed two 7 min 15 s constant-load arm cranking and two leg cycling tests at a power output halfway between the mode-specific AT and peak $\dot{V}O_2$. The time constants for phase II $\dot{V}O_2$ (τ) and HR (τ) kinetics were determined by fitting a monoexponential curve from the end of phase I until 3 min of exercise. $\dot{V}O_2$ τ and HR τ values were significantly ($P<0.001$) slower in arm cranking [$\dot{V}O_2$ τ = 66.4 (3.0) s; HR τ = 74.7 (4.4) s] than in leg cycling [$\dot{V}O_2$ τ = 42.0 (1.9) s; HR τ = 55.6 (3.5) s]. The $\dot{V}O_2$ slow component ($\dot{V}O_{2SC}$) accounted for a significantly ($P<0.001$) greater percentage of the total exercise response during arm cranking [23.8 (1.6)%] than during leg cycling [14.2 (1.5)%]. The greater relative $\dot{V}O_{2SC}$ and the slower $\dot{V}O_2$ τ with arm exercise are consistent with a greater recruitment of metabolically inefficient type II muscle fibres during arm cranking than during leg cycling.

Keywords $\dot{V}O_2$ slow component · Arm and leg exercise · Anaerobic threshold

Introduction

At the onset of moderate intensity exercise (below the anaerobic threshold, AT), oxygen uptake ($\dot{V}O_2$) increases exponentially from baseline and reaches a new steady-state value in about 2–3 min (Whipp and Wasserman 1972; Xu and Rhodes 1999). When exercise intensity is above the AT, the attainment of a $\dot{V}O_2$ steady state is delayed or may not occur and a slow component of increasing $\dot{V}O_2$ ($\dot{V}O_{2SC}$) is observed (Whipp 1994). As a result, $\dot{V}O_2$ may continue to rise to maximal values despite the work rate being classified as submaximal. While the physiological mechanisms causing the $\dot{V}O_2$ slow component (SC) are not clearly understood, identifying those factors which cause $\dot{V}O_2$ to increase continuously during heavy exercise would contribute significantly to our understanding of the limitations to exercise tolerance for work rates above the AT.

The possible mechanisms responsible for the $\dot{V}O_{2SC}$ include increased muscle and blood lactate, elevated plasma adrenaline concentrations, increased ventilatory and cardiac work, and the Q_{10} effect of increased muscle and core temperatures (Poole et al. 1992; Whipp 1994). It has recently been proposed that recruitment of fast-twitch (type II) muscle fibres might be in large part responsible for the $\dot{V}O_{2SC}$ (Barstow et al. 1996; Coyle et al. 1992; Poole et al. 1992; Xu and Rhodes 1999). Research indicates that type II fibres have a higher O_2 cost and a longer time constant ($\dot{V}O_2$ τ) than slow-twitch (type I) fibres (Crow and Kushmeric 1982; Kushmeric et al. 1992). If proportionally more type II fibres are recruited when motor units fatigue during heavy constant-load exercise, then the O_2 cost to maintain a given power output will increase over time.

In examining the $\dot{V}O_{2SC}$, previous research has focused almost exclusively on leg exercise (Barstow et al. 1996; Gaesser et al. 1992, 1994; Poole et al. 1992). Furthermore, few studies have examined the $\dot{V}O_{2SC}$ or phase II $\dot{V}O_2$ kinetics (τ) during arm-cranking exercise performed at an intensity above the AT (Casaburi et al. 1992; Cerretelli et al. 1977; Koga et al. 1996). The metabolic and physiological responses to arm cranking differ markedly to those of leg cycling (Eston and Brodie 1986; Kang et al. 1997; Sawka 1986). Mechanical efficiency is significantly lower in arm cranking than leg cycling and researchers have attributed some of this difference to an increased recruitment of type II muscle fibres during arm-cranking exercise (Kang et al. 1997; Sawka 1986; Xu and Rhodes 1999). Moreover, it has

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been reported that the arm has a greater proportion of type II muscle fibres than the leg in the same subjects (Johnson et al. 1973; Turner et al. 1997). If the $\dot{V}O_{2SC}$ is related to the recruitment of type II fibres, then we would expect to find a slower $\dot{V}O_2$ τ and a greater $\dot{V}O_{2SC}$ in the mode of exercise which results in a greater recruitment of type II muscle fibres.

The purpose of the present study was to determine if differences exist between arm cranking and leg cycling in the $\dot{V}O_{2SC}$ or in phase II $\dot{V}O_2$ kinetics in untrained male subjects. We hypothesised that the $\dot{V}O_{2SC}$ would be greater and the $\dot{V}O_2$ τ would be slower in arm cranking than in leg cycling. Heart rate (HR) kinetics were also examined to provide insight into mechanisms affecting $\dot{V}O_2$ kinetics during exercise performed above the AT.

Methods

Subjects

Ten untrained male subjects volunteered to participate in this study. The mean (SEM) values for age, body mass, and height were 21.6 (1.3) years, 80.7 (3.1) kg and 180.2 (1.2) cm, respectively. Subjects were not currently involved in a training program for sports that predominantly used either the lower body (e.g. cycling) or upper body (e.g. swimming) musculature. Following familiarisation with all testing equipment and experimental procedures, written informed consent was obtained from each subject. The Griffith University Human Research Ethics Committee approved this study.

Experimental protocol

Each subject attended seven testing sessions on separate days within a 3-week period. Subjects were instructed not to engage in vigorous physical activity for 48 h prior to each exercise testing session. The first session was used to familiarise subjects with the testing equipment and procedures, and to obtain informed consent. Once the subject had successfully completed the preliminary screening and familiarisation session, they performed two incremental exercise tests (one arm cranking and one leg cycling) to exhaustion on separate days in random order. These two incremental exercise tests were used to determine peak oxygen uptake ($\dot{V}O_{2peak}$) and AT values for leg cycling and arm cranking. Following the determination of mode-specific $\dot{V}O_{2peak}$ and AT, each subject performed a total of four submaximal constant-load exercise tests to examine $\dot{V}O_2$ and HR kinetics and to quantify the $\dot{V}O_{2SC}$. Each subject performed two constant-load exercise tests for leg cycling and two for arm cranking at a power output halfway between the mode-specific AT and peak $\dot{V}O_2$ (Δ50%). The four constant-load tests were performed in alternate order on 4 separate days.

Determination of $\dot{V}O_{2peak}$ and AT for leg cycling and arm cranking

Each subject performed two incremental exercise tests to volitional fatigue on separate days using a mechanically braked cycle ergometer (Monark 818 E, Varberg, Sweden). For arm cranking, the ergometer was securely mounted on a table. The ergometer was fitted with enlarged handles to provide a secure handgrip during arm cranking. Following 10 min of seated rest, the subject commenced unloaded leg cycling or arm cranking at 60 rev·min^{-1}. After 3 min of unloaded exercise, the power output was increased by 30 W·min^{-1} for leg cycling and by 15 W·min^{-1} for arm cranking. During both incremental exercise tests, $\dot{V}O_2$,$\dot{V}CO_2$ and $\dot{V}_E$ were measured breath-by-breath and averaged over 20-s intervals using a metabolic measuring system (MedGraphics Cardiopulmonary Diagnostic Systems, St. Paul, Minn., USA). The O_2 and CO_2 analysers and the pneumotachograph were calibrated before and after each test using precision reference gases and a syringe of known volume (3 l), respectively. Heart rate and rhythm were monitored continuously throughout the test using an electrocardiograph (Lohemeier M 607, Munich, Germany) with the ECG signal transferred into the gas analysis system for HR storage. $\dot{V}O_{2peak}$ was reported as the average of the three highest 20-s values obtained during the exercise test.

The criteria used for the non-invasive determination of the AT was a systematic increase in the ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$) without an increase in the ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$). The AT calculated by this method was verified using the modified V-slope method described by Schneider et al. (1993).

Oxygen uptake and HR kinetics

Following 10 min of seated rest, the subject began 3 min of unloaded exercise at 60 rev·min^{-1} using a basket-loading ergometer (Monark 824 E, Varberg, Sweden). At the end of the unloaded exercise period, the load was applied instantaneously without prior warning given to the subject. The subject completed a 7 min 15 s square-wave test at a power output equal to Δ50%. Blood samples for the determination of lactate concentrations were obtained from an earlobe prior to and immediately following exercise. The difference between the end-exercise lactate and the resting lactate concentration was expressed as a delta value (Δ[La]). Blood lactate concentrations were determined using an automated blood lactate analyser (Model 2700 SELECT, Yellow Springs Instruments, Ohio, USA). All blood samples were collected and analysed in duplicate.

Gas exchange was measured breath-by-breath (MedGraphics Cardiopulmonary Diagnostic Systems) and HR was recorded beat-by-beat during all constant-load exercise tests. In the present study, the end of phase I could not be reliably determined from the respiratory exchange ratio values obtained during arm cranking. Therefore, the end of phase I was set to 20 s after the onset of constant-load exercise for both modes of exercise (Koga et al. 1996; Lamara et al. 1987). For the determination of the phase II $\dot{V}O_2$ (τ) kinetics, a monoexponential curve was fitted between the end of phase I and the 3rd min of exercise. The breath-by-breath data from the two trials for each mode of exercise were time-aligned and added together to create a single data set for each subject. To enhance the underlying physiological response pattern of $\dot{V}O_2$, a moving average of five consecutive breaths was used in the analysis of the $\dot{V}O_2$ response during the exercise transition. Nonlinear regression techniques (Tablecurve 2D, Jandel Scientific) were used to fit the $\dot{V}O_2$ and HR data with a monoexponential function. Tau (τ) represents the time (in seconds) to achieve 63% of the change in $\dot{V}O_2$ or HR from the end of the phase I value to the 3rd min value. A time of 3 min was chosen as the practical frame of reference for the end of phase II as it has been shown that during leg cycling and arm cranking at sub-AT workloads, phase II is complete within 3 min (Koga et al. 1996; Whipp 1994; Whipp and Wasserman 1972).

The HR kinetics were analysed in the same manner as $\dot{V}O_2$ kinetics. HR τ was determined by fitting a monoexponential curve to the HR data between the end of the $\dot{V}O_2$ phase I and the 3rd min of exercise. The purpose of analysing the HR kinetics with respect to phase I and II of the $\dot{V}O_2$ kinetics was to examine the relationship between HR and $\dot{V}O_2$ kinetics during both modes of exercise.

Determination of the slow component

The $\dot{V}O_{2SC}$ was determined as the increase in $\dot{V}O_2$ between the 3rd min of exercise and the end of constant-load exercise (7 min 15 s). The $\dot{V}O_2$ value at each minute was calculated as the average $\dot{V}O_2$ for the preceding and subsequent 15 s of exercise (3 min $\dot{V}O_2$ = $\dot{V}O_2$ between 2 min 45 s and 3 min 15 s; 7 min $\dot{V}O_2$ = $\dot{V}O_2$

between 6 min 45 s and 7 min 15 s). The increase in $\dot{V}O_2$ between 3 and 7 min of exercise was also expressed relative to the net increase in $\dot{V}O_2$ above that measured during unloaded exercise (A_{tot}). In addition, the percentage increase in $\dot{V}O_2$ above the 3-min value was determined at minutes 4, 5, 6, and 7 of constant-load exercise (see Fig. 2).

Data analysis

All data were analysed using computerised statistical packages (SigmaStat v2.0, Jandel Corporation and SAS v6.12, SAS Institute). Group data are reported as means (SEM). Paired *t*-tests were used to determine whether significant differences existed between arm cranking and leg cycling in maximal exercise values and in constant-load exercise responses. Inferential statistical analysis to determine time-course differences in $\dot{V}O_2$ and HR between arm cranking and leg cycling included the use of a two-factor (mode of exercise and time) repeated measures analysis of variance (ANOVA). Post hoc comparisons to determine where significant differences existed were performed using Scheffé's confidence interval test. Pearson product-moment coefficients (*r*) were used to assess the significance of the relationship between variables. The alpha level was set to 0.05 for statistical significance.

Results

Peak exercise and AT values

Table 1 presents the peak values obtained during incremental arm and leg exercises. $\dot{V}O_{2peak}$ values were significantly higher for leg cycling than for arm cranking. The peak power achieved during incremental arm cranking was only 46% of that achieved during leg cycling. The peak exercise HR obtained for leg cycling was significantly higher than for arm cranking. $\dot{V}O_2$ measured at the gas-exchange AT was significantly higher for leg cycling [1.55 (0.08) l·min^{-1}] than for arm cranking [1.06 (0.05) l·min^{-1}]. However, the AT as a percentage of the mode-specific $\dot{V}O_{2peak}$ was not significantly different between leg cycling [50.2 (1.3)%] and arm cranking [51.3 (1.0)%].

$\dot{V}O_2$kinetics and $\dot{V}O_{2peak}$

The $\dot{V}O_2$ response to constant-load exercise is presented in Table 2. During the final 30 s of unloaded exercise prior to the workload transition, $\dot{V}O_2$ was similar for both modes of exercise. However, the subjects commenced constant-load exercise at a significantly higher percentage of $\dot{V}O_{2peak}$ during arm cranking than during leg cycling (Fig. 1). Phase II $\dot{V}O_2$ kinetics ($\dot{V}O_2$ τ) were significantly slower for arm cranking than for leg cycling, such that despite starting at a higher percentage of $\dot{V}O_{2peak}$ during unloaded arm cranking, both modes of exercise were at a similar percentage of $\dot{V}O_{2peak}$ at 3 min of exercise (Fig. 1). The absolute $\dot{V}O_{2SC}$ ($\Delta\dot{V}O_2$ 7–3 min) was not different between arm cranking and leg cycling. However, the $\dot{V}O_{2SC}$ accounted for a significantly greater percentage of the total increase in $\dot{V}O_2$ above baseline (%A_{tot}) during arm cranking than during leg cycling ($P < 0.001$). Moreover, the percentage increase in $\dot{V}O_2$ above the 3-min value was significantly greater for arm cranking than for leg cycling at 4, 5, 6 and 7 min of exercise (Fig. 2).

The Δ[La] value was 6.4 (0.4) mM for leg cycling and 5.8 (0.4) mM for arm cranking. The small difference between the two modes of exercise in Δ[La] was not significant. The magnitude of the $\dot{V}O_{2SC}$ was significantly correlated with the increase in blood lactate concentration in both leg cycling ($r = 0.70$, $P < 0.05$) and arm cranking ($r = 0.69$, $P < 0.05$).

Table 1. Peak exercise values obtained during incremental arm cranking and leg cycling. Values presented are means (SEM). $\dot{V}O_{2peak}$ Peak oxygen uptake; HR_{max} maximal exercise heart rate

	Leg cycling	Arm cranking
$\dot{V}O_{2peak}$ (l·min^{-1})	3.10 (0.14)	2.08 (0.11) **
$\dot{V}O_{2peak}$ (ml·kg^{-1}·min^{-1})	39.0 (2.2)	25.9 (1.6) **
Peak power (W)	280.2 (14.9)	128.6 (6.9)**
HR_{max} (beats·min^{-1})	192.5 (2.0)	180.8 (5.2)*

*Significant difference between arm and leg exercise, $P < 0.05$
**Significant difference between arm and leg exercise, $P < 0.001$

Table 2. Parameters of the oxygen uptake response to constant-load exercise for arm cranking and leg cycling. Values presented are means (SEM). $\dot{V}O_2$ Oxygen uptake; *BL* baseline unloaded exercise 30 s prior to Δ50% exercise; $\dot{V}O_2$ τ oxygen uptake phase II time constant; $\dot{V}O_2$ *@ 3 min* oxygen uptake at 3 min of exercise; $\Delta\dot{V}O_2$ *7–3 min* increase in oxygen uptake between 3 and 7 min of exercise; A_{tot} = net increase in oxygen uptake at end of exercise above BL

	Leg cycling	Arm cranking
$\dot{V}O_2$ @ BL (ml·min^{-1})	607 (17)	571 (19)
$\dot{V}O_2$ @ BL (%$\dot{V}O_{2peak}$)	20.0 (1.1)	28.9 (1.6) *
$\dot{V}O_2$ τ (s)	42.0 (1.9)	66.4 (3.0) *
$\dot{V}O_2$ @ 3 min (ml·min^{-1})	2278 (98)	1469 (73) *
$\dot{V}O_2$ @ 3 min (%$\dot{V}O_{2peak}$)	73.7 (1.3)	72.0 (0.7)
$\Delta\dot{V}O_2$ 7–3 min (ml·min^{-1})	273 (31)	294 (36)
Relative $\Delta\dot{V}O_2$ 7–3 min (% A_{tot})	14.2 (1.5)	23.8 (1.6) *

*Significant difference between arm and leg exercise, $P < 0.001$

HR kinetics

Table 3 presents the HR response to constant-load work in both modes of exercise. The baseline HR during the final 30 s of unloaded exercise prior to the Δ50% transition was not significantly different between the two modes of exercise. Similarly, when expressed relative to the mode-specific maximal heart rate (HR_{max}), the baseline HR was not statistically different between the two exercise modes. The time taken for the HR to reach 100 beats·min^{-1} was not different between arm and leg exercise. However, HR τ values were significantly slower for arm cranking than for leg cycling, such that despite both modes of exercise starting at a similar percentage of the mode-specific HR_{max}, arm cranking was at a lower percentage of HR_{max} at 3 min of exercise. The correlation between the HR τ and $\dot{V}O_2$ τ for arm cranking was

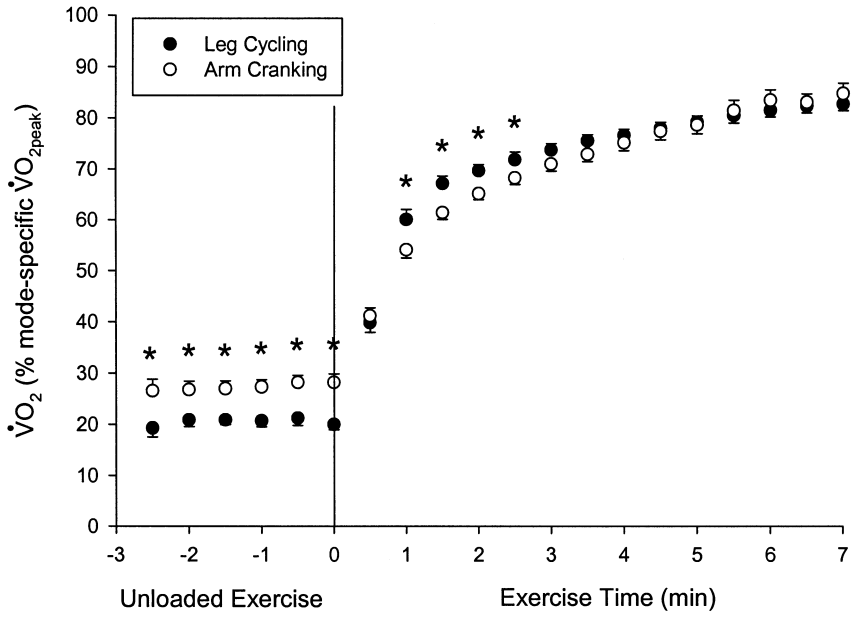

Fig. 1. Oxygen uptake ($\dot{V}O_2$) expressed as a percentage of mode-specific $\dot{V}O_{2peak}$ measured during unloaded exercise and during constant-load arm cranking and leg cycling in untrained subjects. Data are means (SEM) for the group at 30-s intervals. *Significant difference between arm cranking and leg cycling, $P < 0.05$

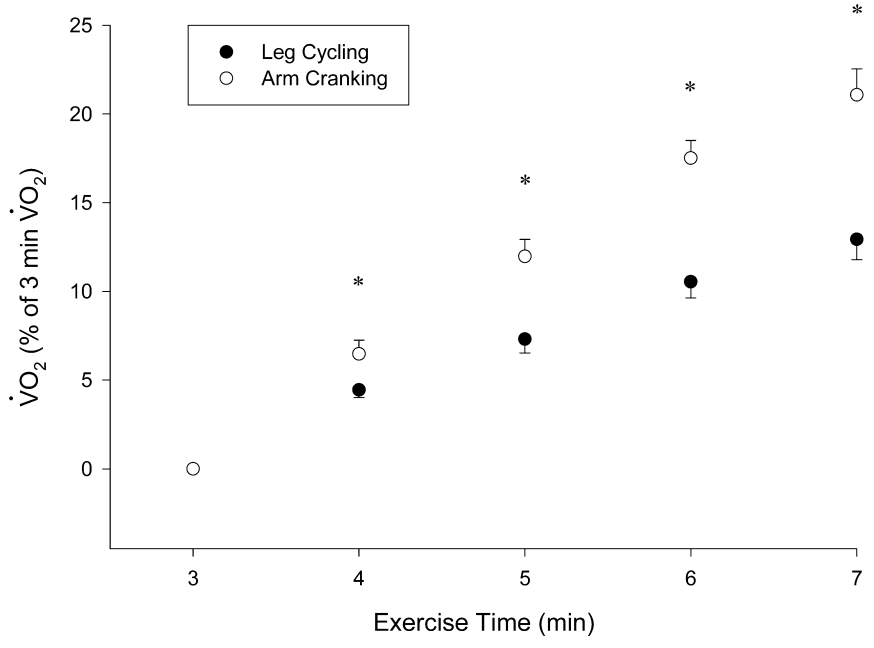

Fig. 2. The increase in oxygen uptake ($\dot{V}O_2$) expressed as a percentage of the $\dot{V}O_2$ measured at 3 min of exercise. Data are means (SEM) for the group. *Arm cranking significantly higher than leg cycling, $P < 0.05$

weak ($r = 0.21$, $P > 0.05$), whereas this relationship was strong during leg cycling ($r = 0.76$, $P < 0.05$).

Discussion

The primary findings of this study were slower $\dot{V}O_2$ τ and HR τ values for arm cranking than for leg cycling, whereas the relative $\dot{V}O_{2SC}$ (expressed as the percentage rise in $\dot{V}O_2$ above the 3-min value) was greater during arm than during leg exercise. The time taken for the HR to reach 100 beats·min^{-1} was not different between the two modes of exercise. This suggests that the rate of vagal withdrawal is not different between arm and leg exercise when performed at the same relative intensity. Therefore, the slower HR τ during arm cranking compared to leg cycling is most likely due to lower sympathetic stimulation of the heart during arm exercise. Plasma noradrenaline (NA) concentration has been used as an index of sympathetic nervous system (SNS) activity during exercise (Mazzeo 1991; Schneider et al. 2000). Lewis et al. (1983) and Savard et al. (1989) reported that NA spillover during exercise is positively related to the amount of active muscle mass. This suggests that there may be greater SNS activity during leg cycling compared to arm cranking due in part to the larger muscle mass used in this mode of exercise. Lower sympathetic activation of the heart during arm cranking

Table 3. The heart rate response to constant-load exercise for arm cranking and leg cycling. Values presented are means (SEM). *HR* Heart rate; *BL* baseline – 30 s unloaded exercise prior to Δ50% exercise; *TTR* time after the onset of exercise to reach 100 beats·min^{-1}; *HR* τ HR phase II time constant

	Leg cycling	Arm cranking
HR @ BL (beats·min^{-1})	86.1 (4.7)	86.6 (3.3)
HR @ BL (%HRmax)	44.7 (2.3)	47.9 (1.3)
TTR 100 beats·min^{-1} (s)	10.1 (2.8)	12.6 (3.1)
HR τ (s)	55.6 (3.5)	74.7 (4.4) *

*Significant difference between arm and leg exercise, $P < 0.001$

could cause slower phase II $\dot{V}O_2$ kinetics by delaying O_2 delivery to the active muscles during arm exercise (Hughson and Imman 1986).

Although O_2 delivery to the active muscle affects the $\dot{V}O_2$ τ (Hughson and Imman 1986), the intrinsic ability of the muscle to utilise O_2 may also limit $\dot{V}O_2$ kinetics during exercise. The poor correlation observed between $\dot{V}O_2$ τ and HR τ during arm cranking ($r = 0.21$) in the present study indicates that the slower $\dot{V}O_2$ time constant for arm cranking is caused by a mechanism other than reduced O_2 delivery. Koga et al. (1996) reported that phase II $\dot{V}O_2$ kinetics were similar for both modes of exercise, whereas cardiac output kinetics were slower for arm cranking than for leg cycling at 50% of mode-specific $\dot{V}O_{2peak}$. This suggests that reduced O_2 delivery does not limit $\dot{V}O_2$ kinetics during arm cranking at workloads below the AT. In addition, the findings of similar muscle capillarisation (Turner et al. 1997), relative blood flow (Pendergast et al. 1979), and oxygenation (Bhambhani et al. 1998; Jensen-Urstad et al. 1993) for arm cranking and leg cycling suggest that the slower $\dot{V}O_2$ τ for arm exercise is caused by an O_2 utilisation limitation rather than by reduced O_2 delivery. Furthermore, fibre-type composition of the active muscle affects O_2 utilisation kinetics within the muscle. Crow and Kushmerick (1982) have shown that type IIb fibres are slower in the rate of O_2 utilisation adjustment in response to a standardised contraction than type I or IIa fibres.

Theoretical reasons suggest that the kinetics of O_2 uptake are slower in type II than in type I fibres. Type II fibres have a much lower oxidative enzyme capacity and greater glycolytic enzyme activity compared to type I fibres (Maughan et al. 1997). Therefore, type II fibres have a faster rate of lactate production under most exercise conditions than type I fibres. Cerretelli and di Prampero (1987) argued that the slowing of O_2 utilisation kinetics is related to the rate of lactate production by the working muscle fibres and not to the lactate concentration in muscle or blood. The rate of $\dot{V}O_2$ kinetics in skeletal muscle is coupled to ATP hydrolysis by a single reaction with apparent first-order kinetics (Cerretelli and di Prampero 1987). At the onset of muscle contraction, changes in [ATP] and [ADP] are prevented by the extramitochondrial creatine phosphokinase (CPK) reaction (ADP phosphorylated to ATP by phosphocreatine, PCr). The resulting increase in [Cr] leads to ADP production within the mitochondrial membrane via intramitochondrial CPK (ATP + Cr yields ADP + PCr). The ADP so produced reaches the mitochondrial matrix to be reconverted to ATP via oxidative phosphorylation. Finally, completing the cycle, ATP produced in the mitochondrion reacts with Cr within the mitochondrial membrane to form PCr and ADP. Thus, the mitochondrion couples O_2 uptake to the phosphorylation of Cr. However, the accumulation of Cr is slowed when there is concomitant lactate production at the onset of exercise. The ATP resynthesised via anaerobic glycolysis bypasses the CPK reaction. Under conditions in which lactate is produced at the beginning of exercise, the accumulation of Cr is slowed and this will result in a slower $\dot{V}O_2$ on-response. This is one possible mechanism that could result in slowed O_2 utilisation kinetics in type II fibres.

The similar absolute increase in blood lactate concentration following constant-load arm cranking and leg cycling in the present study suggests that the rate of lactate production per active muscle unit must be greater during arm cranking than leg cycling (Grichko et al. 1999). This is supported by Ahlborg and Jensen-Urstad (1991), who reported that the rate of lactate release per active muscle was significantly greater for arm exercise than for leg exercise when expressed relative to active-limb lean muscle mass. These investigators found that the arm muscles released more lactate per active muscle mass than leg muscles and that a substantial proportion of the lactate was derived from intramuscle glycogenolysis during arm exercise. Both of these findings during arm crank exercise represent metabolic characteristics of type II muscle fibres and suggest a greater recruitment of fast twitch muscle fibres during arm compared to leg exercise. Therefore, the blood lactate results and the slower O_2 utilisation kinetics observed for arm exercise in the present study are consistent with a greater relative recruitment of type II fibres during arm compared to leg exercise (Crow and Kushmeric 1982; Poole et al 1992; Sawka 1986; Xu and Rhodes 1999).

Although the absolute $\dot{V}O_{2SC}$ was similar for arm cranking and leg cycling in the present study, the $\dot{V}O_{2SC}$ accounted for a significantly greater percentage of the total exercise response during arm cranking than during leg cycling (Table 2). Figure 2 illustrates that the increase in $\dot{V}O_2$ expressed as a percentage of the 3-min value was significantly greater for arm cranking than for leg cycling at minutes 4 through 7 of exercise. In the present study, the Δ[La] was not significantly different between the two modes of exercise. However, a significant correlation between Δ[La] and the magnitude of the $\dot{V}O_{2SC}$ was observed in both modes of exercise. Earlier studies (Gaesser et al. 1992,1994; Poole et al. 1994) suggest that the high correlation between the $\dot{V}O_{2SC}$ and Δ[La] is coincidental rather than causal. Elevation of blood lactate either by infusion of adrenaline (Gaesser et al. 1994) or lactate (Poole et al. 1994) has been found to have no effect on exercise $\dot{V}O_2$ or the $\dot{V}O_{2SC}$. These

findings support the notion that increased adrenaline and/or lactate is not the cause of the elevated $\dot{V}O_2$ during heavy constant-load exercise.

Other researchers have shown that arm-cranking exercise results in a significantly greater $\dot{V}O_2$ than that of leg cycling at the same submaximal power output (Eston and Brodie 1986; Powers et al. 1984). The higher $\dot{V}O_2$ during arm cranking compared to leg cycling is reflected by lower gross and net efficiencies for arm cranking (Eston and Brodie 1986). The differences in efficiency between arm cranking and leg cycling seem too large to be explained by leverage considerations, the need for torso stabilisation, or by the oxygen cost of gripping ergometer cranks with the hands (Eston and Brodie 1986). Sawka et al. (1986) and others (Kang et al. 1997; Xu and Rhodes 1999) suggest the lower delta efficiency values reported by Powers et al. (1984) and Kang et al. (1997) during arm cranking than leg cycling indicates a greater participation of type II muscle fibres during upper body exercise. Prior research indicates that type II fibres have a higher O_2 cost during a standard contraction than type I fibres (Crow and Kushmeric 1982; Kushmeric et al. 1992).

In a previous study, we found a significant increase in the mean power frequency (MPF) of the EMG between 3 and 7 min of heavy constant-load exercise in both young and older subjects (unpublished data). Some researchers (Lucia et al. 2000; Scheuermann et al. 2001) have failed to demonstrate changes in muscle EMG activity during heavy constant-load work, whereas several other investigators found a significant increase in the MPF (Borrani et al. 2001; Garland et al. 1999; Saunders et al. 2000) or integrated EMG (iEMG) activity during heavy-intensity exercise (Shinohara and Moritani 1992). These researchers (Borrani et al. 2001; Garland et al. 1999; Saunders et al. 2000; Shinohara and Moritani 1992) suggest that the increase in MPF and/or iEMG is due in part to an increase in the proportion of type II fibres recruited during heavy constant-load exercise. The greater relative $\dot{V}O_{2SC}$ and the slower $\dot{V}O_2$ τ observed for arm exercise in the present study are consistent with a greater recruitment of metabolically inefficient type II muscle fibres during arm cranking than during leg cycling.

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